EFFICACY OF ELECTRICAL AND THERMOGENIC STIMULATION ON WEIGHT REDUCTION AMONG OBESE FEMALES

by

NICK MENTZ

Submitted in partial fulfillment of the requirements for the degree

Doctor Philosophiae

in the

DEPARTMENT BIOKINETICS, SPORT AND LEISURE SCIENCES

FACULTY OF ARTS

UNIVERSITY OF PRETORIA

PRETORIA
MAY 2003

The epidemic of obesity and inactivity is just as deadly –
if not more so – than any virus, but it receives less
attention because it acts slowly and because we
have adjusted to its presence among us.

Like infectious disease epidemics, this epidemic can
be stopped in its tracks – not with a vaccine,
but with a formula of healthier eating and
more activity that is well within our reach.
(Koplan, 2000)

ACKNOWLEDGEMENTS

I wish to express my thanks and gratitude to the following persons and institutions for their guidance and assistance, in the completion of this study:

DR H.J. VAN HEERDEN: (Department of Biokinetics, Sport and Leisure Sciences, University of Pretoria). For the valuable time afforded to me as promotor of this study, and his unstinted guidance, support and advice at all times.

PROF G.J. VAN WYK: (Head of the Department of Biokinetics, Sport and Leisure Sciences, University of Pretoria). For his interest and motivation.

PROF P.E. KRÜGER: (Director, Institute of Sport Research, University of Pretoria). For the use of the laboratory in conducting the study and financial support.

DR ZANET OSCHMAN: For conducting the ultra-sound sonography measurements.

SUBJECTS THAT PARTICIPATED IN THE PROJECT: Their involvement and willingness made this study possible.

CHRISTINE SMIT: For the assistance in the statistical analysis of the data.

TILLA BOSHOFF: For her diligent typing and editing of this thesis.

HEINRICH NOLTE, BYRON MALGA AND STEVEN BALL: For their assistance in compiling and editing the tabular and graphic presentation of data.

FAMILY AND FRIENDS: For their prayers encouragement and support throughout.

MY WIFE, RIANA: For her encouragement and loving support.

THE ALMIGHTY: In Him everything is possible.

SYNOPSIS

TITLE	:	Efficacy of Electrical and Thermogenic Stimulation on Weight Reduction among Obese Females
CANDIDATE	:	N. W. Mentz
PROMOTOR	:	Dr H.J. van Heerden
DEGREE	:	D.Phil

The primary aim of this study was to evaluate the effect of an eight-week programme of electrical muscle stimulation (EMS) performed on Slimline Slimming Machines in conjunction with (Group EST), and without (Group ESP), a thermogenic agent (Thermo Lean) and following a standardized diet (Group TS). In order to achieve this goal a pre-test-post test experimental groups design, with three levels of the independent variable, was adopted for the study. A group of 69 females between the ages of 25 - 40 years (mean age = 35.26 ± 6.02 years), who were recruited through newspaper advertisements, served as subjects. To be included in the study, subjects were required to be physically suitable for the intervention programmes; pre-menopausal; obese (BMI > 30); sedentary; and amenable to being assigned to any of three study groups. The following categories of dependent variables were measured: Anthropometry; Morphology; Ultrasound Sonography; Respiratory Quotient; Pulmonary Function; Haematology; Cardiovascular Responses; and Musculoskeletal Function.

There was a statistically significant difference between groups ($p \leq 0,05$) in the reduction of abdominal body girths measured at three different body sites viz. abdominal (level of greatest anterior protrusion); abdominal AB-1 (midway between the xyphoid process and the umbilicus); and abdominal AB-2 (level of the umbilicus). Group EST (6.02%) had the greatest reduction in girth at the abdominal body site. This reduction was significantly ($p \leq 0,05$) better than the reduction found in group ESP (4.79%) and group TS (4.69%). The same tendency was found at the abdominal AB-1 body site. Group EST (6.42%) had the greatest reduction in girth which was significantly ($p \leq 0,05$) better than the reduction found in group TS (4.35%) and group ESP (4.28%). Group ESP had the greatest reduction in girth at

the umbilicus level (7.39%). This reduction was significantly ($p \leq 0,05$) better than the reduction found in group TS (4.85%).

The greatest reduction of skinfold measurements was found at the tricep skinfold. Group EST had the greatest reduction (12.75%). This reduction was significantly ($p \leq 0,05$) better than the reduction found in both groups TS (9.27%) and ESP (6.63%). The second greatest reduction in skinfolds was found at the abdominal skinfold. Group EST had the greatest reduction (12.14%). This reduction was significantly ($p \leq 0,05$) better than the reduction found in both groups TS (11.80%) and ESP (10.36%). The third greatest skinfold reduction was found at the subscapular skinfold. Group EST had the greatest reduction (9.70%). This reduction was significantly ($p \leq 0,05$) better than the reduction found in both groups TS (8.64%) and ESP (3.93%). The observed significantly ($p \leq 0,05$) greater reduction in skinfold measurement at the abdominal site in group EST corresponded with the same significantly ($p \leq 0,05$) greater reduction in girth measurements at the abdominal body sites in the same group.

Whith respect to saggital height measurements, at the umbilicus body site (saggital umbi), group ESP (11.48%) had the greatest reduction. This reduction was similar to the reduction found in group EST (11.02%). At the saggital ½ umbi body site, group EST (13.52%) had the greatest reduction in saggital height. This reduction was significantly ($p \leq 0,05$) greater than that found in both groups ESP (10.61%) and TS (10.60%). This significantly ($p \leq 0,05$) greater reduction in saggital height at the saggital ½ umbi body site in group EST, corresponds with the significant ($p \leq 0,05$) decreases found in body girths and skinfolds in the same group.

A significantly reduced ($p \leq 0,05$) waist-to-hip ratio (WHR) was observed within two of the three experimental groups. The greatest reduction was found in group EST (2.53%) and this reduction was significantly ($p \leq 0,05$) better than the reduction found in group TS (1.27%) and group ESP (1.27%). The largest (3.03%) reduction in body surface area (BSA) was seen in group EST and this reduction was significantly greater ($p \leq 0,05$) than in group ESP (1.96%).

The ultrasound sonographic subcutaneous fat layer in group EST (21.22%) showed the greatest reduction. This reduction was significantly ($p \leq 0,05$) greater than the reduction in subcutaneous fat found in both groups TS (18.04%) and ESP (12.11%). The visceral fat layer

in group EST (27.74%) also showed the greatest reduction. This reduction was significantly ($p \leq 0,05$) greater than that found in both groups ESP (22.82%) and TS (21.87%). This significantly ($p \leq 0,05$) greater reduction in subcutaneous and visceral fat found in group EST, corresponds with the significant ($p \leq 0,05$) decreases found in body girths, skinfolds and saggital height in the abdominal area in the same group.

In conclusion, obese females participating in a program of dietary restriction, thermogenic or electrical muscle stimulation with the aim of achieving weight-loss should note that: diet with or without electrical muscle stimulation (EMS) proved effective, but these modalities in conjunction with thermogenic stimulation proved the most effective intervention program after eight weeks.

KEY WORDS: ELECTRICAL MUSCLE STIMULATION; THERMOGENIC STIMULATION; CALORIE RESTRICTION; OBESE FEMALES; ABDOMINAL; SUBCUTANEOUS; VISCERAL; WEIGHT-LOSS.

SINOPSIS

TITEL	:	Effektiwiteit van Elektriese en Termogenetiese Stimulasie op Gewigsverlies by Obese Dames
KANDIDAAT	:	N. W. Mentz
PROMOTOR	:	Dr H.J. van Heerden
GRAAD	:	D.Phil

Die primêre doel van hierdie studie was om die effek te evalueer van ‘n agt-weke program van elektriese spierstimulasie (ESS), uitgevoer op Slimline Verslankingsapparate, tesame met (Groep EST), en sonder (Groep ESP), ‘n termogenetiese middel (Thermo Lean) asook ‘n gestandaardiseerde dieet (Groep TS). ‘n Voortoets- natoets eksperimentele groepsontwerp, met drie vlakke van die onafhaniklike veranderlike, is gebruik vir die studie. ‘n Totaal van 69 vroulike proefpersone tussen die ouderdom van 25 – 40 jaar (gemiddelde ouderdom 35.26 ± 6.02 jaar), wie deur koerantadvertensies gewerf is, het as proefpersone gedien. Insluitingskriteria vir die studie het vereis dat proefpersone fisies geskik was vir die intervensieprogramme, en premenoposaal; obees (LMI > 30); sedentêr en bereid moes wees om by enige van die drie studiegroepe ingedeel te word. Die volgende afhanklike veranderlikes is gemeet: Antropometrie; Morfologie; Ultraklank Sonografie; Respiratoriese Kwosiënt; Pulmonêre Funksie; Hematologie; Kardiovaskulêre Respons; en Muskuloskeletale Funksie.

Daar was ‘n statisties beduidende verskil tussen groepe ($p \leq 0,05$) met die afname in abdominale liggaamsomtrekke by drie verskillende anatomiese liggings naamlik; abdominaal (vlak van grootste anterior uitsetting; abdominaal AB-1 (halfpad tussen die xiphoid proses en die umbilicus); en abdominaal AB-2 (vlak van die umbilicus). Groep EST (6.02%) het die grootste afname getoon by die abdominale ligging. Hierdie afname was beduidend ($p \leq 0,05$) beter as die afname in groep ESP (4.79%) en groep TS (4.69%). Dieselfde tendens is gevind by die abdominale AB-1 ligging. Groep EST (6.42%) het die grootste afname in omtrekke getoon wat beduidend ($p \leq 0,05$) beter was as die afnames in groep TS (4.35%) en groep ESP

(4.28%). Groep ESP het by die umbilicus die grootste afname in omtrekke getoon (7.39%). Hierdie afname was beduidend ($p \leq 0,05$) beter as die afname in groep TS (4.85%).

Die grootste afname in velvoumetings is gevind by die trisepvelvou. Groep EST het die grootste afname getoon (12.75%). Hierdie afname was beduidend ($p \leq 0,05$) beter as die afnames in beide groep TS (9.27%) en groep ESP (6.63%). Die tweede grootste afname is gevind by die abdominale-velvou. Groep EST het die grootste afname getoon (12.14%). Hierdie afname was beduidend ($p \leq 0,05$) beter as die afnames in beide groepe TS (11.80%) en ESP (10.36%). Die derde grootste velvou afname was by die subscapula-velvou. Groep EST het die grootste afname getoon (9.70%). Hierdie afname was beduidend ($p \leq 0,05$) beter as die afnames in beide groepe TS (8.64%) en ESP (3.93%). Die waargenome beduidend ($p \leq 0,05$) groter afname in velvoumetinge by die abdominale ligging in groep EST stem ooreen met dienooreenkomstige beduidend ($p \leq 0,05$) groter afnames in omtrekmetinge by die abdominale liggings in dieselfde groep.

Met betrekking tot saggitalehoogte metinge, by die umbilicus ligging (saggitaal umbi), het groep ESP (11.48%) die grootste afname getoon. Hierdie afname was soortgelyk aan die afnames gevind in groep EST (11.02%). By die saggitaal ½ umbi ligging het groep EST (13.52%) die grootste afname in saggitale hoogte getoon. Hierdie afname was beduidend ($p \leq 0,05$) beter as in beide groepe ESP (10.61%) en TS (10.60%). Die beduidend ($p \leq 0,05$) groter afname in saggitale hoogte by die saggitaal ½ umbi ligging in groep EST, stem ooreen met die beduidende ($p \leq 0,05$) afnames gevind in liggaamsomtrekke en velvouemetinge in dieselfde groep.

Beduidende afnames ($p \leq 0,05$) in middel-tot-heup omtrekverhouding (MHV) is waargeneem in twee van die drie eksperimentele groepe. Die grootste afname is gevind in groep EST (2.53%) en hierdie afname was beduidend ($p \leq 0,05$) beter as die afnames in groep TS (1.27%) en groep ESP (1.27%). Die grootste (3.03%) afname in liggaamsoppervlakte meting (LOM) is waargeneem in groep EST en hierdie afname was beduidend beter ($p \leq 0,05$) as groep ESP (1.96%).

Die ultraklank sonografiese onderhuidse vetlaagmeting in groep EST (21.22%) het die grootste afname getoon. Hierdie afname was beduidend ($p \leq 0,05$) beter as die afnames in onderhuidse vet in beide groepe TS (18.04%) en ESP (12.11%). Die viserale vetlaag in groep EST (27.74%) het ook die grootste verlaging getoon. Hierdie verlaging was beduidend ($p \leq 0,05$) beter as in beide groepe ESP (22.82%) en TS (21.87%). Hierdie beduidende ($p \leq 0,05$) groter afname in onderhuidse en viserale vet in groep EST, stem ooreen met die beduidende ($p \leq 0,05$) afnames gevind in liggaamsomtrekke, velvoue en saggitale hoogte in die abdominale gebied binne dieselfde groep.

Ter afsluiting, obese dames wat deelneem aan ‘n program van kalorie-inperking, termogenetiese of elektriese spierstimulasie met die oog op gewingsverlies moet kennis dra dat: dieet met of sonder elektriese spierstimulasie (ESS) effektief is, maar dat hierdie modaliteite in samewerking met termogenetiese stimulasie bewys is as die mees effektiewe intervensieprogram na agt-weke.

SLEUTELWOORDE: ELEKTRIESE SPIERSTIMULASIE; TERMOGENETIESE STIMULASIE; KALORIEBEPERKING; OBESE DAMES; ABDOMINALE-; ONDERHUIDSE-; VISERALE-; GEWIGSVERLIES.

TABLE OF CONTENTS

Page No.

CHAPTER 3 : METHODS AND PROCEDURES

CHAPTER 4 : RESULTS AND DISCUSSION

CHAPTER 5 : SUMMARY, CONCLUSION AND RECOMMENDATIONS

Page No.

APPENDICES

Page No.

LIST OF TABLES

Page No.

Page No.

LIST OF FIGURES

Page No.

Page No.

CHAPTER 1

THE PROBLEM

1.1 INTRODUCTION

Weight-loss and obesity is a major problem in South Africa as well as in the rest of the world. Weight-loss is associated with improvements in obesity-related complications, but patients and practitioners are frequently disappointed by the long-term results of weight control efforts. Recent research has yielded new findings concerning the causes of obesity, as well as new goals for obesity treatment. Traditionally, the goal of therapy has been to attain ideal weight. Several scientific bodies, now recommend a modest 5% tot 15% reduction in initial weight (Jakicic et al., 2001; Klein, 2000; Anderson & Wadden, 1999). Regardless of how much weight a person would like to lose, modest goals and a slow course will maximize the probability of losing and maintaining weight. It should be recognized that for most people, achieving a body weight or figure like those often depicted by the media is neither reasonable, appropriate, nor achievable. Failure to achieve this "look" does not imply a weakness of will power or character. Numerous methods of weight-loss exist where the objective is short-term, rapid or unsupervised weight-loss, which 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.

1.2 OBESITY DEFINED

Obesity is defined in terms of excess body fat. Because precise assessment of body fat is cumbersome and expensive, body weight is often used as an estimate of obesity. The term overweight has traditionally referred to weight in excess of some ideal, usually stipulated by actuarial height and weight tables. Unfortunately, the definition of "ideal" weight varies over time and across cultures, thus making it difficult for example, to compare the prevalence of obesity in two nations. In recent years, investigators have begun to use the Body Mass Index (BMI) as a measure of

overweight because it does not rely on comparison with an ideal weight. The World Health Organization has defined overweight as a BMI of 25.0 to 29.9, while obesity is a BMI of 30 or greater (World Health Organization, 1997). South Africa is on its way to overtaking America as the world's fattest nation. Almost half of South Africans over the age of 15 are overweight or obese, and medical researchers warn that the government may soon have to step in to manage the epidemic (Health 24, 2002).

Obesity has long been thought to be a behavioural disorder that resulted from simply eating too much and or exercising too little. There is no question that these factors are associated with weight gain. Changes in our national lifestyle, including the increased consumption of high fat foods, as well as our increasingly sedentary work and leisure habits, undoubtedly contributed to the marked rise in obesity. Recent studies, however, have suggested that body weight is under substantial genetic control, accounting for approximately one third of the variation in BMI (Bouchard, 1997). Genetic influences appear to contribute to differences among individuals in resting metabolic rate (Rice et al., 1996), as well as body fat distribution (Bouchard et al., 1998) and weight gain in response to overfeeding (Bouchard et al., 1990). Some people appear to come into the world with a predisposition to obesity, which is readily nourished by our high-fat, low-activity lifestyle.

Society is unforgiving of overweight individuals. Stunkard (1995) have called the disparagement of obese individuals "the last socially acceptable form of prejudice". Historically, the public has believed that weight-loss is a matter of willpower. Obese individuals have been considered weak-willed and unmotivated, a view that is compounded by the claims of easy weight-loss promised by many books on diet. Unfortunately, many obese persons seem to have accepted this view of themselves. Practitioners are not immune to these beliefs. In one study obese patients were described in such negative terms as "weak-willed", "ugly" and "awkward" (Maddox & Leiderman, 1969). Obese women also have been found to delay or avoid medical care because of weight concerns (Olson et al., 1994). Attitudes, towards obesity appear to be changing with the recognition that obesity is a complex, multidetermined disorder with a genetic component (Bouchard, 1997). Findings suggest that physiological and genetic factors may limit the amount of weight that an individual can lose and maintain (Keesey, 1996). These findings have led to new empathy for

overweight individuals, as well as to a change in the goals of obesity treatment.

It seems that although obese individuals may have different therapeutic objectives e.g. to reduce disease risk, to ameliorate disease symptomology, to build self-esteem and to increase functional capacity, the immediate measurable outcome variable of body weight becomes the focus intervention. There are many options for treating obese individuals. In the past, obesity has been treated as an acute disorder. Many patients still appear to believe that 10 to 20 weeks of treatment should be enough to "cure" obesity or at least control it for several years. This view of obesity is often encouraged by the commercial diet industry, which promises miraculous results with little or no effort. The results of such an approach indicate clearly that if treated as an acute disorder, obesity will return. Guy-Grand (1992) noted that all obesity treatments to date are palliative, not curative. Practitioners cannot cure obesity any more than they can diabetes or hypertension. Practitioners need to help patients recognize that obesity is a chronic disorder that requires long-term care. Acceptance of this fact should help improve the results of most treatment modalities. Aggressive therapy should be used with persons who are more obese and who have greater health complications. The five recognised treatment modalities available are diet modification, exercise, behaviour modification, medication therapy and surgery. Diet and exercise are the most frequently cited methods for both men and woman attempting to lose weight. Many forms of therapy are used and promoted including countless fad diets, herbal remedies, acupuncture, acupressure, appetite suppressing "aroma sticks", medication, surgery, electrical muscle stimulation and many more.

1.3 ELECTRICAL MUSCLE STIMULATION DEFINED

Electrical muscle stimulation (EMS) make use off electrical pulse generators which produce repetitive muscular contractions in affected areas (Bailey, 1976). In essence, these apparatus are automatically cycling, multiple-output, faradic muscle stimulators, which produce trains of pulses with variable pulse repetition and frequency. The individual pulses are of short duration and of low energy, but at appropriate gain levels the pulse trains produce rhythmic and powerful muscular contractions when they are fed to the muscle by skin contact electrodes placed over or near the motor points. It is claimed that repeated application of such pulse stimulation produce

breakdown of adipose tissue by local passive exercise of the muscle unit, and so afford a generalized reduction in both size and weight (Bailey, 1976).

The use of electrical muscle stimulation has rapidly increased in the last few years and Slimline slimming machines are a popular modality among the public for the treatment of weight control in South Africa. The usual treatment session last 30 to 40 minutes, and is repeated two or three times per week. The conductive rubber electrode pads are place over those parts of the body (on or near motor points) where muscular contractions are desired, and the gain levels are adjusted so as to produce maximum muscle contractions without discomfort. During the treatment period of eight weeks the patient is given some simple dietary rules, but generally no drastic caloric restriction are imposed. Electrical muscle stimulation is a useful tool in training the obese patient, although it may not stimulate muscle hypertrophy, it has positive effect on muscle firming and further re-educates and develops the nerve muscle interaction.

1.4 THERMOGENIC STIMULATION DEFINED

Thermogenesis is a term referring to the body's production of heat. Heat production is a normal part of metabolic processes. At certain times, such as when exposed to cold temperatures, the body may bring about an additional form of thermogenesis-shivering. Shivering is an attempt by the body to create needed supplemental body heat by increasing muscular activity. Certain nutritional substances can also stimulate thermogenesis. Thermogenesis, when not simply needed for routine food digestion and metabolism, is both a source of heat and when stimulated through appropriate dietary supplementation serves as a mechanism to increase metabolic rate. Fuel for this increased metabolic rate can be provided by stored body fat, if released and available for use.

Nutritional stimulation of the body's β (beta) receptor pathway, can induce the breakdown and release of stored body fat, and thereby allow stored fats to be turned into energy. The β-agonists path is a signalling pathway in the body for a number of related areas. Many cell surfaces, especially muscle and fat cell surfaces, contain β-receptors. When a β-agonist (or antagonist) binds to a β-receptor site, a sequence of processes are activated that can both induce lipolysis (the breakdown and use of stored

fat for energy production) and increase muscle metabolism. This increased muscle metabolism may facilitate muscle cell growth.

Common β-agonists already available in the body include the neurotransmitter catecholamines, epinephrine and norephinephrine. These agonists are especially released during times of stress. Other materials which can interfere with the sequence of steps in the β-agonist process include dietary materials such as caffeine. Obese individuals interested in nutritionally supporting the body's natural fat-release and thermogenic mechanisms could consider the use of thermogenic agents as part of their dietary and weight management program. Thermo Lean is one such product on the South African market and comprises a unique formulation of special extracts and herbs to nutritionally support the release and burning of stored body fat.

1.5 STATEMENT OF THE PROBLEM

Women may have unrealistic expectations of attainable weight and body shape associated with the Western emphasis on thinness. Substantial costs are icurred in the attempt to alter body weight and shape. Federal surveys indicate that among US adults trying to lose weight, over a two year period women make an average of 2.5 weight-loss attempts, each lasting an average of 6.4 months, and men make an average of two attempts, each lasting an average of 5.8 months. As a result, in the United States over $30 billion dollars are spent annually on weight-loss efforts (Technology Assessment Conference Panel, 1992). As with any goal, there are costs and consequences associated with the quest for the ideal body. If taken to an extreme, the ultimate cost may be life itself.

Some of the support for a healthy weight management paradigm, rather than the traditional weight-loss management paradigm, comes from exercise scientists. The American College of Sports Medicine acknowledges that obese individuals could reap health benefits from exercise without demanding that the exercise meet the traditional intensity requirements suggested for weight-loss (American College of Sports Medicine, 1990). The Surgeon General's Report on Physical Activity and Health declared that physical activity need not be vigorous to improve health (US Department of Health and Human Services, 1996).

Research reflects a paucity of knowledge with respect to electrical muscle stimulation (EMS) and thermogenic stimulation (TS). 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. Despite the meager data base to support claims that electrical muscle stimulation, thermogenic stimulation, diet or a combination of these three modalities are effective in long-term weight control, this should not deter the pursuance of health intervention strategies for the obese. In cognisance of the foregoing, the question comes to mind whether or not, and to what extent, the advent of electrical muscle stimulation (EMS) and thermogenic stimulation (TS) can make a significant contribution to help the obese.

1.6 MOTIVATION FOR THE STUDY

- The treatment of obesity and weight-loss strategies has become a commercial enterprise.

- Unless the efficacy of a potential therapeutic modality, whether it is exercise, behavioural, dietary, pharmacological or electrical, has been scientifically proven, its application can be considered irresponsible.

- Electrical muscle stimulation and thermogenic stimulation has great appeal to many sedentary and obese subjects because it promises some of the benefits of vigorous exercise without strenuous effort.

1.7 PURPOSE AND AIM OF THE STUDY

The aim of the study was to conduct a randomized placebo-controlled trial to evaluate the effect of electrical muscle stimulation (EMS) performed on *Slimline Slimming Machines,* in conjunction with and without a thermogenic agent *(Thermo Lean)* and dietary control, on various physiological parameters among obese females.

1.8 HYPOTHESES

In accordance with the stated purpose of this study the following hypothesis was formulated:

A program of electrical muscle stimulation (EMS) performed on *Slimline Slimming Machines*, in conjunction with a thermogenic agent *(Thermo Lean)* and dietary control would have a beneficial effect on various physiological parameters among obese females.

1.9 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 electrical stimulation in conjunction with a thermogenic agent and dietary control serving as an assessment of the efficacy of both interventions as a rehabilitative 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).

- 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).

- Accumulation of fat beyond that considered normal for one's age, gender and body type (Sharkey, 1984).

- 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 (Buskirk, 1974).

- Obesity is simply an accumulation of an excessive amount of fat in the adipose tissues (Williams, 1995).

- Obesity is having an excessive amount of body fat that is associated with an increased risk of medical illnesses (Klein, 2000).

Although any single definition of obesity is apt to be incomplete, obesity is probably best defined by a combination of above-mentioned definitions.

Obesity is an excessive accumulation of energy (weight) in the form of body fat beyond that considered normal for one's age, gender and body type, which impairs health. The degree of health impairment is determined by three factors:

1. the amount of fat;
2. the distribution of fat; and
3. the presence of other risk factors.

Obesity = BMI > 30 kg/m^2 (BMI = Body Mass Index) (Klein, 2000).

2.2 EPIDEMIOLOGY OF OBESITY

From an anthropological view of time, the current "epidemic" of overweight and obesity is very recent. For 99% of history, the exclusive productive economic pattern of human culture was one of hunting and gathering. Today, this original human lifestyle is rare, but a few such groups have been the subjects of intensive cultural and biological investigation. An important finding has been that there are no reported cases of obesity among people following a traditional hunting and gathering way of life. Food foragers do not store surplus food and, in general, demonstrate an egalitarian distribution of food brought into camp (Brown, 1991). Obesity was essentially nonexistent until after the invention of farming some 10 000 years ago, and more specifically until after the Industrial Revolution. Throughout the developed world the incidence of obesity has been climbing at an alarming rate. In the United States, there has been roughly a thirty percent increase in obesity prevalence in the decade of the Nineties alone and this large increase has brought a great deal of public health attention (Brown, 1998).

Obesity is a serious and widespread health problem encountered only in certain kind of societies - ones characterized by economic modernization, some affluence, food surplus, and social stratification. Numerous studies of traditional societies undergoing the process of economic modernization demonstrate rapid increases in the prevalence of obesity (West, 1978; Zimmet, 1979; Phillips & Kubisch, 1985). Trowell and Burkitt's (1981) volume of 15 case studies of epidemiological changes in modernizing societies concludes that obesity is the first of the "diseases of civilization" to appear.

The highest reported prevalence of obesity is on the Micronesian island of Nauru with the age standardized prevalence of adult obesity being 84,7% for males and 92.8% for females (Collins & Dowse, 1990). The inhabitants of Nauru are extremely wealthy because of valuable mineral deposits on the island (Baba & Zimmet, 1990). In Europe, there are higher prevalence's of obesity in southern European countries than northern ones, and within those countries the risk of obesity is higher in rural than urban areas (Kluthe & Schubert, 1985). Within the United States, a country with a high overall prevalence of overweight and obesity the behavioural risk factor surveys coordinated by the Centres for Disease Control (CDC) indicate that the South-East and Central region had the highest obesity rates (21.2%) and that West Virgina had the highest single state prevalence (23.9%) (Gurney & Gornstein, 1988).

The most important epidemiological question that can be asked is what social groupings are most at risk for obesity. There are three facts about this social distribution that are particularly cogent:

1. a gender difference in the total percent and site distribution of body fat, as well as the prevalence of obesity;

2. the concentration of obesity in certain ethnic groups; and

3. a powerful and complex relationship between social class and obesity.

The greatest degree of sexual dimorphism in humans is in the site of fat tissue distribution. Women have much more peripheral body fat in the legs and hips than men. This gender dimorphism has evolutionary roots, since energy storage in peripheral fat has an adaptive advantage in maintaining pregnancy and lactation during periods when diet cannot be supplemented. This pattern of gender differences appears to be universal since it is found in contemporary hunting and gathering groups (Brown, 1991). When obesity is measured in terms of BMI rather than alternative measures of fat deposition like waist-to-hip ratio or waist circumference, the epidemiological significance of central body fat characteristic of males can be underestimated. Peripheral body fat appears to be epidemiologically benign, so the

real health concern should be concentrated on the phenomenon of central body fatness (Bjorntorp, 1988).

The relationship between the risk of obesity and social class has received substantial research attention. Social class is a powerful predictor of the prevalence of obesity in both modernizing and affluent societies, although the direction of the association is different. This relationship was the subject of a comprehensive review by Stunkard (1996) that reviewed more than one hundred separate studies. In developing countries, there was a strong and consistent positive association of social class and obesity for men and women, and there was an inverse correlation between social class and protein-calorie malnutrition (Arteaga & Dos Santos, 1982). When examining health indicators within households in developing countries, there is a common finding of undernourished children living in the same household with obese adults (Goodman & Leatherman, 1998). Historically, the positive correlation between social class and fatness is a logical and expected pattern in the socially dominant groups. With better access to resources they should however have better nutrition and better health. On the other hand, the contemporary processes of modernization are unfolding such that there is a relationship between poverty and obesity. This is increasingly the case in developing societies, as well as affluent societies, as a recent collection of studies from Latin America indicates (Pena & Bacallao, 2000).

In heterogeneous and affluent societies like the U.S., there is a strong inverse correlation between social class and obesity for females (Sobal, 1991). The association of obesity and social class among women in affluent societies is not constant through the life cycle. Garn and Clark (1976) have demonstrated a pattern of reversal in which economically advantaged girls are initially fatter than their poor counterparts, but as adults they show less overweight and obesity. The inverse correlation of obesity and social class for females in affluent societies is extremely strong and carries with it important socially symbolic associations. The high prevalence of obesity in ethnic groups probably reflects the interaction of genes, social class, and culture. It is a difficult task to disentangle the relative effects of these factors. The role of the economy and the availability of resources for the purchase of foods with high fat content should not be minimized. Drewnowski and Popkin's (1997) review of the historical "nutrition transition" indicates that with improved

economic purchasing power there seems to be a limit on the per capita demand for protein and carbohydrates, but no such limit in the demand for fat. This desirability of fat, both on an individual and social level, probably has strong evolutionary roots and is related to the neurophysiology of human preferences for the taste of sweetness (Dobbing, 1987). These biological drives to prefer sweet and fat foods are exploited by the food industry to increase profits.

2.2.1 Obesity, economics and the industrial food system

In contemporary North American culture, food production is based on a highly mechanized agricultural system that is highly dependent on petroleum as an energy source. Bodley (1985) argues that this industrial food system is so inefficient from an energy standpoint that it would be better if Americans could discover a way to consume oil directly. Industrial food production not only means that people eat at a higher trophic level on the food chain, primarily by eating meat, but also that we have a wide variety of energy enhanced and calorie rich food available to us. Our society's complex post-industrial economy, allows members access to high calorie diets without requiring physical energy expenditures in food production. To a large extent, it is the power of the economy and our society's position within the global political economic system, that provides the material infrastructure for our current epidemic of obesity (Drewnowski & Popkin, 1997).

The industrial food production system also involves the processing of foods for storage, distribution, and purchase by consumers in a competitive market. The transformation of food through industrial processing often involves the addition of fat, sugar, salt or other preservatives to appeal to consumer taste preferences. The transformation of potatoes in potato chips is a good example. The potato serves primarily as a physical matrix for carrying fat and salt, so that they might be more honestly labelled "fat and salt chips". Butter fat and sugar are industrially transformed into ice cream, a food that is rich in both symbolism and calories. One of the most important aspects of this food processing system is the fact that fat-added or value-added product provides a much higher margin of profit for food manufacturers. The food industry spends a remarkable amount on advertising, particularly on television, for high-calorie, high-profit products. This industry exploits the human evolutionary

heritage that accounts for the consumer's "sweet tooth" and "fat tooth". Ironically, the industrial food industry also processes a wide variety of diet foods, most often containing expensive substitutes for fat and sugar that also have a high profit margin. The food industry's goal is to produce increased revenues and profit for capitalist owners, not to provide an adequate or healthy diet to society. This profit motive shapes both the availability of certain foods (it is hard to find fresh fruits and vegetables in a super market), as well as the manipulative use of advertising to create consumer desire for certain food products. The industrial food system therefore changes culture (Bodley, 1985; Drewnowski & Popkin, 1997).

2.2.2 Fast food as a fat delivery system

It was a breakthrough in Public Health when some key members of the tobacco industry admitted that cigarettes function primarily as a "nicotine-delivery system" providing addicts access to a drug upon which they depend. The negative health consequences of cigarette smoking, therefore, are a by-product of a political economic system. In a very similar way the fast-food industry, part of the industrial food complex, serves as a "fat delivery system" for millions. The appeal of fast food, restaurant food and convenience food to the middle class South African caught in the "time squeeze" of frantic daily schedules, is an important part of culture change.

The number of restaurants in the United States is at an all-time high, as consumers have become increasingly dependent on them as a source of meals and snacks (Census, 1994). Eating establishments in the United States increased by 75% between 1977 and 1991 and Americans are getting an increased proportion of daily energy from establishments outside the home. In 1977 about 16% of all meals and snacks in the U.S. were eaten away from home, accounting for 31% of daily energy intake (Mc Crory et al., 1999). Throughout the 1990s, the fast food industry has expanded at a rate of about 5% per year, and one important area of expansion is "home replacement meals" (HRM) that are cooked industrially and eaten at home. An increased proportion of household food income is spent on meals consumed outside the home, and an increasing consumption of fast food within that category, may be related to the rising prevalence of obesity (Mc Crory et al., 1999).

Several studies have shown that the nutritional quality of foods consumed outside the home are inferior because they contain much more total fat, saturated fat, cholesterol, and sodium per unit energy (Lin & Frazao, 1997). The frequency of eating at fast food restaurants was positively associated with total energy intake and percentage of energy from fat, as well as BMI in women (Jeffery & French, 1998). People who ate more frequently at fast food restaurants also have lower dietary fiber intakes. Mc Crory and colleagues (1999) demonstrate that the consumption of fried chicken and hamburgers, were both correlated with body fatness. They suggest four factors contributing to the increased energy derived from restaurant meals:

1. restaurant meals tend to be higher in fat and energy density;
2. restaurants usually serve large portions;
3. they serve food that is highly palatable; and
4. most fast food and restaurants provide the consumer with dietary variety.

Studies have shown that individuals who consume an excess amount of energy from fat, as apposed to carbohydrates or protein, have a propensity to store body fat at higher rates (Horton & Drougas, 1995). These studies imply that individual dietary restraint and conscious control over food intake are a necessary protective mechanism against a high-fat, high-energy density diet. The environment of our material culture and political economy is "obeso-genic", and individuals must consciously and constantly fight against this cultural current to either lose weight or maintain a proper weight. Currently nearly 40% of the average American dietary intake is derived from fat. A study of meals purchased at Mc Donalds, Kentucky Fried Chicken, and Pizza Hut, demonstrated that typical meals from these menus would provide the consumer with an excess intake of 1000 kcal per day over the recommended levels (Malouf & Colaguiri, 1995).

There has been a marked trend in recent decades for restaurant meals to increase portion size as a strategy both to attract consumers and increase profitability. This is noticeable in the drive-thru at local fast food restaurants when, after having placed an order the consumer is asked, "Would you like to "super-size" that order for an additional two rand?" Hill and Peters (1998) suggest that underlying these larger portion sizes is a capitalist value of "getting the most for your buck". The purchase of

a meal in a restaurant is based upon a market principle of maximizing consumer value (Wansink, 1996). This is a very different sociocultural context from a meal eaten with one's family at home, where sharing a meal rather than getting a good deal or matching individual palates are central to the cognitive cultural model of food consumption. Increased portion sizes and increased calorie density of restaurant food increases profitability for the food industry. Drewnowski (1997) observed that higher fat content is synonymous with the price of the food product, often seen with steak, chocolate, and ice cream. Because of the desirability and palatability of energy dense foods, people eat larger portions per sitting (Schiffman & Graham, 1998). It is possible that preferences for or associations with "fat texture" since there is no basic "fat taste" like a "sweet taste", would be evolutionarily adaptive because of the energy-dense quality of fat (Birch, 1992).

2.2.3 Automobile dependence and inactivity

South-Africans generally live in dispersed settlements, largely in unattached houses, in suburbs. One of the largest rooms in suburban houses is the garage. Generally South-Africans have distinct and segregated geographical areas for work or shopping, and large paved areas for parking that surround these buildings. From a historical standpoint, communities tend to spread farther apart at remarkable rates. Communities are linked by a complex system of wide and expensive roads that have been designed primarily for automobile traffic. The settlement pattern includes very few public spaces like parks or plazas for physical activities like walking, running and cycling.

All the above mentioned actively discourage non-motorized transportation. The dependence on the automobile for nearly all transportation needs is a significant factor in physical inactivity and therefore the current epidemic of overweight and obesity. Street and community design features of suburbs privilege automobile safety and have shown to discourage walking and bicycle riding activities, both for daily errands and recreation (Frank & Engelke, 2000). A consequence of our settlement pattern is that physical activity is not a normal part of daily life. Instead of being related to daily work, physical activity in the form of workouts becomes another thing that has to be squeezed into an already over-scheduled life. Long commuting time for many

individuals, adds to the difficulty of finding time for physical activity. It is ironic that many people feel constrained, for reasons of convenience, custom, and safety, to drive to the gym in order to walk on a treadmill or cycle on a static exercise bike (Frank & Engelke, 2000).

2.2.4 Behaviour patterns, television and obesity

In the past half-century, the introduction and expansion of new technologies, as elements of material culture, have radically changed patterns of social behaviour. Behaviour changes, in turn, influence the prevalence of obesity. A major influence in the South-African culture is the television, a technological device present in nearly all homes and the centre of our most common recreation activity. Putman (2002) states: "Nothing else in the twentieth century so rapidly and profoundly affected our leisure".

There has been much research on the relationship between television viewing, physical activity, and body composition of adults (Jeffery & French, 1998; Mokdad et al., 1999; Buchowski & Sun, 1996). The central question in these studies is the role that television viewing plays in the etiology of obesity, although the problem of the validity of self-reported measures of both physical activity and television viewing has been a persistent problem (Dietz & Gortmaker, 1985). The correlations are strong enough that dose-response effects between times spent watching television and obesity prevalence has been suggested (Dietz & Gortmaker, 1985). The exact mechanisms that explain the correlation between television viewing time and obesity risk is not completely understood, but it obviously involves physical activity, energy expenditure, dietary intake, or a combination of these (Buchowski & Sun 1996). Two primary mechanisms have been suggested: reduced energy expenditure from displacement of physical activity, and increased intake either during viewing or in response to food advertising (Robinson, 1998). The effect of television watching on inactivity and obesity has also been demonstrated in developing societies like India (Gupta & Saini, 1994). Worldwide the average per capita viewing has continued to increase steadily by 7-8% per decade since the 1960's (Dietz & Gortmaker, 1985).

Television is also linked to changes in diet. People snack while they view hundreds of advertisements about energy-dense industrially produced foods per hour of television viewing. Food is the most heavily advertised product on television programs. Roughly 40% of all advertisements concern food, with breakfast cereals, cold drinks and snack foods being the most common products (Barcus & Mc Laughlin, 1978). There is a major inconsistency between nutritional messages contained in commercials and the official nutritional guidelines. Content analysis of television advertisements and the eating behaviour of television characters indicate that eating what is seen on TV will result in energy imbalance and obesity (Wilson & Quigley, 1999). There is clear evidence that people change their eating behaviour to consume what they see on TV (Taras & Sallis, 1989). Obviously, this is why advertisements are made, because eating these products are good for the profits of the food companies, even if they are not necessarily good for our health.

Excessive television watching is correlated with feelings of loneliness, depression and other affective disorders. There is some evidence that people who suffer seasonal affective disorder also experience significant seasonal weight gain during winter months, this may be a case of individuals "self medicating" their dysphoria through eating (Yanovski & Yanovski, 2000).

2.2.5 The ideology of fat versus thin

Television is a major source of cultural beliefs and attitudes about a wide variety of topics, besides food, like beauty and body image (Nichter, 2000). Culture encompasses cultural symbols, beliefs and values. Aspects of cultural ideology relevant to the etiology of obesity include the symbolic meaning of fatness, eating, ideal body types and perceived risks of food shortages.

Fatness is symbolically linked to psychological dimensions such as "self-worth", sensuality and fertility in many societies of the world, but the nature of that symbolic association is not constant. In mainstream first world culture, obesity is socially stigmatised, but for most third world cultures of the world, fatness is viewed as a welcome sign of health and prosperity (Brown, 1991). Given the rarity of obesity in preindustrial societies, it is not surprising that they lack ethno-medical terms for

obesity. There is much more attention placed on thinness as a symptom of starvation, as in societies of contemporary Africa where thinness is a sign of acquired immunodeficiency syndrome (AIDS). In the context of the AIDS epidemic, as well as the general ubiquity of infectious disease, plumpness is indeed a marker of health. In many traditional peasant societies as Teti (1995) describes for southern Italy, plumpness was a symbolic marker of prosperity and power whereas thinness was a symbol of poverty, misery and evil.

Studies about weight gain across the life cycle demonstrate that there are periods of fattening that can be culturally acceptable. The most obvious example of this is the seclusion of adolescent girls from elite families in parts of Africa in preparation for their marriage (Brink, 1995). In developed countries, weight gain has been identified as being typical during certain times, including holidays, the first year at university and following marriage (Yanovski & Yanovski, 2000; Kahn & Williamson, 1990).

For women, fatness may also be a symbol of maternity and nurturance. In traditional societies where women attain status only through motherhood, this symbolic association increases the cultural acceptability of obesity (Powdermaker, 1961). A fat woman symbolically, is well taken care of, and she in turn takes good care of her children. Fellahin Arabs in Egypt describe the proper woman as fat because she has more room to bear a child, lactates abundantly and gives warmth to her children (Amnar, 1954). The cultural ideal of thinness in developed societies, in contrast, is found in societies where motherhood is not the sole or even primary means of status attainment for women. In terms of pediatric obesity, it is important to note that the idea that fat babies and children are healthy children is very widespread. Food can be treated as a symbol of love and nurturance. In some cultures it may be impolite for a guest to refuse some offered food, but it is taboo to refuse food from one's mother.

Obese individuals in industrialized nations suffer significant prejudice and discrimination. Daily, when looking at television programs and magazines, they are reminded that "thin is in" and "fat is not where it's at". Contemptuous attitudes are expressed in jokes heard on the street and on late night talk shows, as well as in the nation's respected news weeklies. The author of a "My Turn" column in Newsweek wrote, "This information (about genetic determinants of obesity) should be withheld

from the fat maltitudes because the obese will latch onto any excuse for failing to lose weight. Face it Chubbo, when was the last time you were force-fed" (Hecht, 1990). Such prejudice has been observed in children as young as six years of age, who described silhouettes of an overweight child as "lazy, dirty, stupid, ugly, cheats, and liars" (Staffieri, 1967). When shown black and white line drawings of an obese child and children with various handicaps, including missing hands and facial disfigurement, both children and adults rated the obese child as the one they least wished to play with (Goodman et al., 1963). Regrettably, overweight individuals display this same prejudice. In a similar study, university students were asked to rate various categories of persons as to their suitability as a marriage partner. Embezzlers, cocaine users, shoplifters, and blind persons were all rated as more suitable partners than were the obese (Venes et al., 1982).

2.3 PREVALENCE OF OBESITY

The prevalence of obesity is rising to epidemic proportions at an alarming rate in both developed "Westernised" and less developed countries around the world (Brown, 1998). Obesity represents a major threat to health and quality of life and is becoming increasingly common. It is now recognised as a major public health problem in South Africa (South African Society for the Study of Obesity, 1999). South Africa is on its way to overtaking America as the world's fattest nation. Almost half of South Africans over the age of 15 are overweight or obese, and medical researchers warn that the government may soon have to step in to manage the epidemic (Van der Merwe, 2002; 2003).

Approximately 45% of South Africans are overweight, compared to 65% Americans, a recent study by the Medical Research Council found. The study reports that 25 percent of South Africans fell into the overweight category, with a Body Mass Index (BMI) in excess of 25, while 20 percent fell into the obese category, with a BMI of more than 30. Black women had the highest rate of obesity at 30%, followed closely by white women at 26% and coloured women at 25%. Twenty one percent of Indian women were classified as obese (Van der Merwe, 2002; 2003). Van der Merwe (2003), speaking on behalf of the South African Society for the Study of Obesity

(SASSO), claims that about 40 percent of the country's women suffered from abdominal obesity.

Most alarming, is the fact that 20% of children under the age of six were overweight. Studies indicate overweight children are highly likely to grow into over-weight adults. (A shocking 35% of them will be overweight by the time they reach 18). Most of these overweight children will grow up with a number of potentially life-threatening problems in tow, including increased risk of heart disease, diabetes, hypertension, kidney disease and even some forms of cancer. Some children are experiencing such problems even before they reach adulthood. Doctors report seeing Type II diabetes (usually in adults) in overweight children (Lambert, 2002).

South Africans are facing an explosion in obesity because such a large segment of our population is rapidly moving to the cities and adopting Western eating habits (transition from traditional high-fibre, low-animal fat diets to high fat diets). Cultural practices and advertising play an important role in making people regard foods as desirable even if they are "fatal" for weight gain. One is often amazed at the subtle connotations the advertisers use to seduce the public to buy all kinds of foods that are laden with fat and kilojoules (Van Heerden, 2002).

Despite the high prevalence of obesity and the many advances in our understanding of how it develops, present management strategies have persistently failed to achieve long-term success (Brown, 1998).

2.4 CONSEQUENCES OF OBESITY

Obesity is a major health problem in many countries because of its increasing prevalence, association with serious medical diseases, psychosocial aspects, and considerable economic impact.

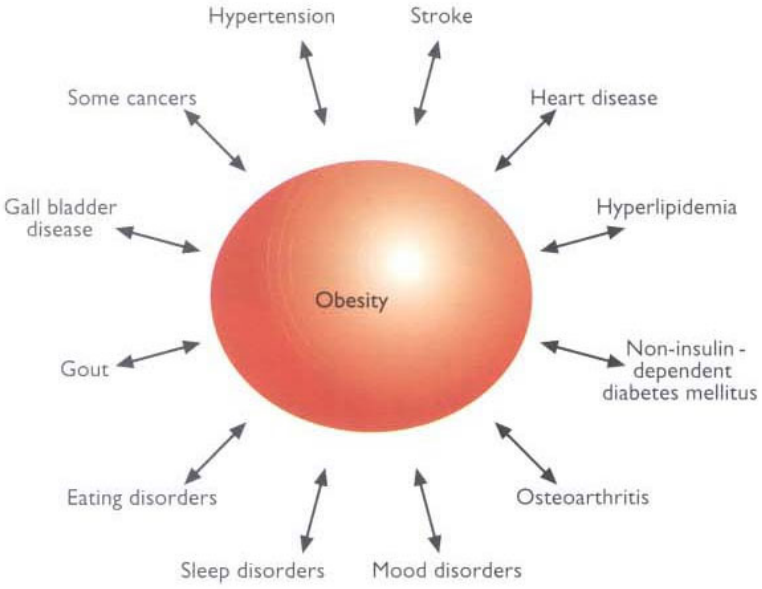


FIGURE 2.1: CONDITIONS ASSOCIATED WITH OBESITY
(Bray, 2003)

2.4.1 Psychosocial aspects of overweight and obesity

Research relating obesity to psychological disorders and emotional distress is based on community studies and clinical studies of patients seeking treatment. Community based studies in the United States have not found significant differences in psychological status between the obese and non-obese (Wadden & Stunkard, 1993). Several European studies in general populations do suggest a relationship between obesity and emotional problems (Lapidus et al., 1989; Lissau & Sorensen, 1994). It may be premature to state that there is no association between obesity and psychopathology or emotional distress in the general population. According to the National Institutes of Health (NIH) more focused, hypothesis-driven and long-term studies are needed (NIH, 1998).

2.4.1.1 Psychopathology and obesity

Overweight people seeking weight-loss treatment may, in clinical settings, show emotional disturbances (Fitzgibbon et al., 1993). In a review of dieting and depression there was a high incidence of emotional illness symptoms in outpatients treated for obesity (Stunkard & Rush, 1974). Several factors influenced these emotional

responses, including childhood onset versus adult onset of obesity (those with childhood onset obesity appear to be vulnerable). Another study that compared different eating disorder groups found that obese seeking treatment showed considerable psychopathology, most prominently mild to severe depression (Prather & Williamson, 1988).

Sixty-two percent of the obese group seeking treatment showed clinically significant elevations on the depression subscale of the Minnesota Multiphasic Personality Inventory, and 37 percent of this same group showed a score of 20 or higher (indicating clinical depression) on the Beck Depression Inventory (NIH, 1998). Another study compared obese people who had not sought treatment to an obese group that had sought treatment in a professional, hospital-based program, and to a normal weight control group (Fitzgibbon et al., 1993). Again, obese individuals seeking treatment reported more psychopathology and binge eating compared to the other groups. Both obese groups reported more symptoms of distress than did normal weight controls. The authors suggest that the obese population is not a homogeneous group, and may not respond in the same way to stand and treatment programs. Obese individuals seeking treatment in clinic settings are more likely to report more psychopathology and binge eating than obese individuals not seeking treatment (NIH, 1998).

2.4.1.2 Binge eating disorder

Binge eating disorder is characterized by eating larger amounts of food than most people would eat in a discrete time period (e.g. 2 hours) with a sense of lack of control during these episodes (American Psychiatric Association, 1994). It is estimated to occur in 20 to 50 percent of individuals who seek specialized obesity treatment (Marcus et al., 1995). In community based samples, the prevalence is estimated to be approximately two percent (Spitzer et al., 1992). Comparisons have been made between binge eating disorder and bulimia nervosa, an eating disorder characterized by recurrent and persistent binge eating, accompanied by the regular vomiting, fasting, or using laxatives.

Studies comparing normal weight individuals who have bulimia nervosa with obese binge eating disorder individuals have found that obese binge eaters are likely to demonstrate dietary restraint and show few, if any, adverse reactions to moderate or severe dieting. Most obese binge eaters do not engage in inappropriate compensatory behaviours such as purging (Yanovski et al., 1994). Compared with bulimia nervosa, the demographic distribution of binge eating disorder is broader with respect to age, gender, and race while data suggest that binge eating disorder is as common in African-American women as in white women (Marcus et al., 1995; Spitzer et al., 1992; Spitzer et al., 1993). The difference between binge eating disorder and bulimia nervosa is dramatic regarding gender. Very few men have bulimia nervosa, whereas the distribution is close to equal in binge eating disorder (Striegel-Moore et al., 1998).

Compared to obese nonbingers, obese individuals with binge eating disorder tend to be heavier, report greater psychological distress, and are more likely to have experienced a psychiatric illness (especially affective disorder) (Yanovski, 1993). They also report an earlier onset of obesity and a greater percentage of their lifetime on a diet (Kuehnel & Wadden, 1994). These individuals are also more likely than non-binging obese people to drop out of behavioural weight-loss programs and to regain weight more quickly (Yanovski et al., 1994; Marcus et al., 1990).

Critics of behavioural treatment of obesity have argued that caloric restriction may cause or contribute to the episodes of binge eating (Garner & Wooley, 1991). Three studies have tested this hypothesis. Neither moderate nor severe caloric restriction exacerbated binge eating. All three studies showed that weight control treatment featuring caloric restriction significantly reduced the frequency of binge eating in these patients (Marcus et al., 1995; Telch & Agras, 1993; Yanovski & Sebing, 1994).

2.4.1.3 Body image

Body image is defined as the perception of one's own body size and appearance and the emotional response to this perception (O'Neil & Jarrell, 1992). Inaccurate perception of body size or proportion and negative emotional reaction to size perceptions contribute to poor body image. Obese individuals especially women, tend to overestimate their body size (Collins, 1987).

People at greater risk for a poor body image are binge eaters, women, those who were obese during adolescence or with early onset of obesity and those with emotional disturbances (Mussell et al., 1996). These individuals are more dissatisfied and preoccupied with their physical appearance, and avoid more social situations due to their appearance (Tiggemann & Rothblum, 1988). Body image dissatisfaction and the desire to improve physical appearance often drive individuals to seek weight-loss. Obese persons seeking weight reduction must come to terms with real limits in their biological and behavioural capacities to lose weight. Weight-loss attempts may only intensify the sense of failure and struggle that is already present among many obese individuals. Psychosocial interventions, which incorporate strategies to improve body image, may be helpful for those who want to lose weight and are very concerned about their physical appearance (NIH, 1998).

Body image perception of individuals in various ethnic and racial groups may be different from those of the mainstream culture. There may be a similar range of attitudes but on a different scale. It may take a much greater degree of overweight to elicit negative reactions. In general, black girls and black women report less social pressure to be slim, fewer incidences of weight-related discrimination, less weight and body dissatisfaction and greater acceptance of overweight than their white counterparts (Kumanyika et al., 1993; Powell & Kahn, 1995; Kemper et al., 1994). University-age black women report less concern and fear about fatness, less drive to be thin, and less concern about dieting than do university-age white women (Rucker & Cash, 1992). Black women may ascribe some positive qualities to being large, such as having stamina, strength, and solidity and are less likely to link body size to health than white women. Black elementary school and high school girls were more likely to be trying to gain weight and less likely to be trying to lose weight as compared to white girls (Schreiber et al., 1996; Rosen & Gross, 1987). It is possible that weight control initiatives may elicit different reactions from black and white women. Less is known about the relationship between obesity and body image disturbance in other racial and ethnic groups (Gittelsohn et al., 1996).

2.4.1.4 Social stigmatisation

In many Westernised societies there are powerful messages that people, especially women, should be thin, and that to be fat is sign of poor self-control (Brownell & Fairburn, 1995; Wadden & Stunkard, 1993). Negative attitudes about the obese have been reported in children and adults. People's negative attitudes toward the obese often translate into discrimination in employment opportunities, university acceptance, less financial aid from their parents in paying for university, job earnings, rental availabilities, and opportunities for marriage (Larkin & Pines, 1979; Pingitore et al., 1994; Canning & Mayer, 1966).

Much of the research on the social stigma of obesity has suffered from methodological limitations. A number of the early studies relied on line drawings rather than more lifelike representations of obese people and on checklists that forced one to make categorical YES or NO choices (NIH, 1998).

There has been a lack of research looking at the impact of obesity in the context of other variables, such as physical attractiveness, the situational context, and the degree of obesity (Herman et al., 1986). Social stigma toward the obese has primarily been assessed among white individuals. There is some evidence that members of other racial and ethnic groups are less harsh in their evaluation of obese persons. One study assessed 213 Puerto Rican immigrants to the United States, and found a wide range of acceptable weights among them (Massara & Stunkard, 1979). Crandall found that Mexican students were significantly less concerned about their own weight and were more accepting of other obese people than were US students (Crandall & Martinez, 1996). The degree of acceptance of obesity among people of lower education and income has not been well studied. Data with respect to racial and ethnic groups other than whites is incomplete (NIH, 1998).

2.4.2 Health risks associated with being overweight or obese

Above an initial BMI of 20 kg/m^2, morbidity for a number of health conditions increases as the BMI increases.

	Obesity class	BMI (kg/m^2)	Risk
Underweight		<18.5	Increased
Normal		18.5 – 24.9	Normal
Overweight		25.0 – 29.9	Increased
Obesity	I	30.0 – 34.9	High
	II	35.0 – 39.9	Very high
Extreme obesity	III	≥40.0	Extremely high

BMI, body mass index.

FIGURE 2.2: BODY WEIGHT-ASSOCIATED DISEASE RISK
(Klein, 2000)

Higher morbidity in association with overweight and obesity has been observed for hypertension, type II diabetes, coronary heart disease (CHD), stroke, gallbladder disease, osteoarthritis, sleep apnea and respiratory problems and some types of cancer (endometrial, breast, prostate, colon) National Institute of Health (NIH, 1998).

Obesity is also associated with complications of pregnancy, menstrual irregularities, hirsutism (presence of excess body and facial hair), stress incontinence (urine leakage caused by weak pelvic floor muscles), psychological disorders (depression) and increased surgical risks (NIH, 1998).

The nature of obesity-related health risks is similar in all populations, although the specific level of risk associated with a given level of overweight or obesity may vary with race/ethnicity, and also with age, gender, and societal conditions. The absolute risk of morbidity in chronic conditions such as CHD is highest in the aged population, while the relative risk of having CHD in obese versus non-obese individuals is highest in the middle adult years (Feinlieb, 1985).

A high prevalence of diabetes mellitus in association with obesity is observed consistently across races/ethnicities, while the relative prevalence of hypertension and CHD in obese versus non-obese populations varies between groups (Du Plessis, 2000).

2.4.2.1 Hypertension

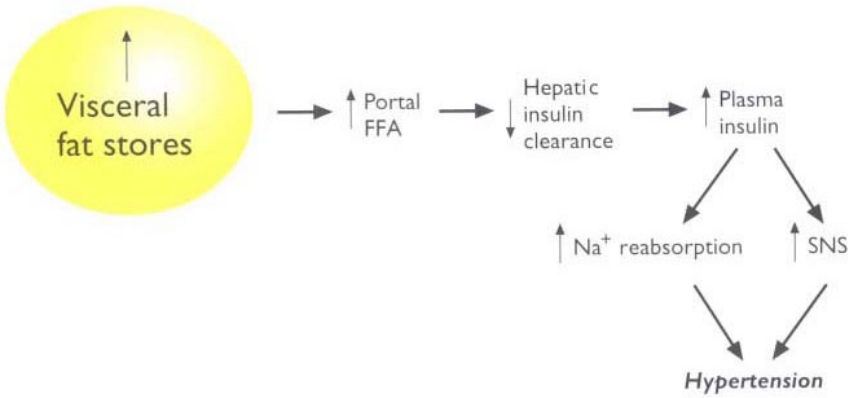


FIGURE 2.3: PATHOPHYSIOLOGICAL MODEL FOR THE RISK OF DEVELOPING HYPENTENSION (Bray, 2003)

High blood pressure is defined as mean systolic blood pressure ≥140 mm Hg, or mean diastolic blood pressure of ≥90 mm Hg, or currently taking antihypertensive medication. The age adjusted prevalence of high blood pressure increases progressively with higher levels of BMI and increases in waist-to-hip ratio. In severely obese people with a ratio greater than 0.81, almost 70% are hypertensive (Berchtold et al., 1981). The prevalence of high blood pressure in adults with BMI 30 is 38.4 percent for men and 32.2 percent for women, respectively, compared with 18.2 percent for men and 16.5 percent for women with BMI < 25, translating a relative risk of 2.1 and 1.9 for men and women, respectively (Brown et al., 1998). The direct and independent association between blood pressure and BMI or weight has been shown in numerous cross-sectional studies (Stamler et al., 1978), including the large international study of salt (INTERSALT) carried out in more than 10,000 men and women (Dyer & Elliott, 1989). INTERSALT reported that a 10 kg higher body weight is associated with 3.0 mm Hg higher systolic and 2.3 mm Hg diastolic blood pressure. These differences in blood pressure translate into an estimated 12 percent increased risk for CHD and 24 percent increased risk for stroke (Dyer & Elliott, 1989).

Obesity and hypertension are co-morbid risk factors for the development of cardiovascular disease. The pathophysiology underlying the development of hypertension associated with obesity may be increased by arterial peripheral

resistance, sodium retention, and increased blood volume and cardiac output. Peripheral resistance may rise in obesity because of the expanded tissue volume and relatively smaller capillary density. High dietary sodium intake causes increased blood volume, due to expansion of erythrocyte mass and greater plasma volume. Cardiovascular abnormalities associated with obesity are believed to be related to a combination of increased sodium retention, increased sympathetic nervous system activity, alterations of the renin-angiotensin system and insulin resistance (Reisin et al., 1983; Tuck et al., 1981).

The precise mechanism whereby weight-loss results in a decrease in blood pressure is unknown. It is known that weight-loss is associated with a reduction in vascular resistance, total blood volume and cardiac output, an improvement in insulin resistance, a reduction in sympathetic nervous system activity, and suppression of the renin angiotensin aldosterone system (Reisin et al., 1983; Tuck et al., 1981; Landsberg & Krieger, 1989; Jacobs & Sowers, 1993).

2.4.2.2 Coronary heart disease (CHD)

Observational studies have shown that overweight, obesity, and excess abdominal fat are directly related to cardiovascular risk factors, including high levels of total cholesterol, LDL-cholesterol, triglycerides, blood pressure, fibrinogen and insulin, and low levels of HDL-cholesterol (National Institutes of Health Consensus Development Conference Statement, 1985). Plasminogen activator inhibitor-I causing impaired fibrinolytic activity is elevated in persons with abdominal obesity (Landin et al., 1990). Overweight, obesity, and abdominal fat are also associated with increased morbidity and mortality from CHD (NIH, 1998).

Studies have shown that the risks of non-fatal myocardial infarction and CHD death, increase with increasing levels of BMI. Risks are lowest in men and women with BMI's of 22 or less and increase with even modest elevations of BMI. In the Nurses' Health Study, which controlled for age, smoking, parental history of CHD, menopausal status and hormone use, relative risks for CHD were twice as high at BMI's of 25 to 28.9 and more that three times as high at BMI's of 29 or greater, compared with BMI's of less than 21 (Willett et al., 1995). Weight gain of 5 to 8 kg

increased CHD risk (non-fatal myocardial infarction and CHD death) by 25 percent, and weight gains of 20 kg or more increased risk more than 25 times in comparison with women whose weight was stable within a range of 5 kg (Willett et al., 1995). A relationship between obesity and CHD has not always been found. Two reasons may account for this: the first is an inappropriate controlling for cholesterol, blood pressure, diabetes, and other risk factors in statistical analysis; and the second is that there was not an adequate control for the confounding effect of cigarette smoking on weight. People who smoke often have a lower body weight but more CHD (Lundgren et al., 1989).

2.4.2.3 Congestive heart failure (CHF)

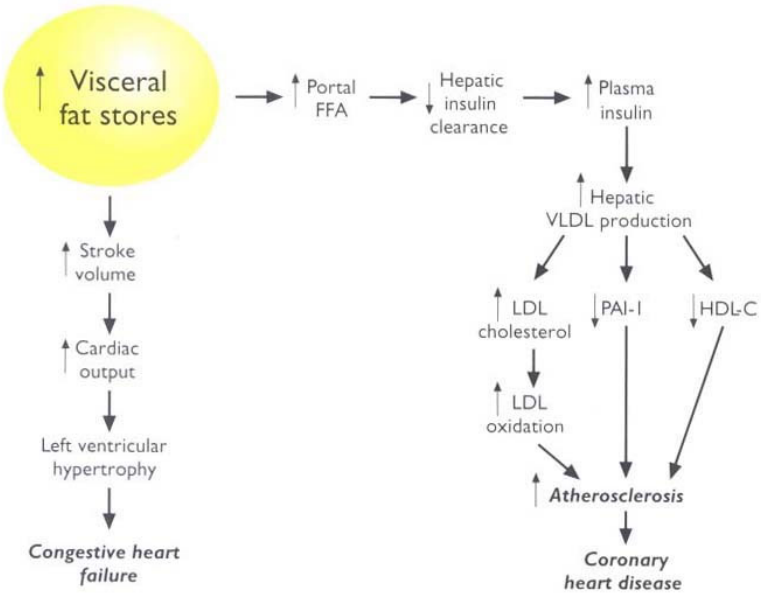


FIGURE 2.4: PATHOPHISIOLOGICAL MODEL FOR THE RISK OF DEVELOPING CONGESTIVE HEART FAILURE AND CORONARY HEART DISEASE (BRAY, 2003)

Overweight and obesity have been identified as important and independent risk factors for congestive heart failure (CHF) in a number of studies (Alpert & Hashimi, 1993), including the Framingham Heart Study (Hubert et al., 1983). CHF is a frequent complication of severe obesity and a major cause of death. The duration of obesity is a strong predictor of CHF (Alpert & Hashimi, 1993). Since hypertension and type II diabetes are positively associated with increasing weight, the coexistence of these

conditions facilitates the development of CHF (Urbina et al., 1995). Data from the Bogalusa Heart Study demonstrate that excess weight may lead to acquisition of left ventricular mass beyond that expected from normal growth (Urbina et al., 1995). Obesity can result in alterations in cardiac structure and function even in the absence of systemic hypertension or underlying heart disease (NIH, 1998). Ventricular dilatation and eccentric hypertrophy may result from elevated total blood volume and high cardiac output. Diastolic dysfunction from eccentric hypertrophy and systolic dysfunction from excessive wall stress results in so-called "obesity cardiomyopathy" (Alpert & Hashimi, 1993; Garavaglia et al., 1988).

2.4.2.4 Stroke

The relationship of cerebrovascular disease to obesity and overweight has not been as well studied as the relationship to CHD. A report from the Framingham Heart Study suggested that overweight might contribute to the risk of stroke, independent of the known association of hypertension and diabetes with stroke (Hubert et al., 1983). More recently published reports are based on larger samples and delineate the importance of stroke subtypes in assessing these relationships (Rexrode et al., 1997; Walker et al., 1997). They also attempt to capture all stroke events, whether fatal or non-fatal. These studies suggest distinct risk factors for ischemic stroke as compared to hemorrhagic stroke, and found overweight to be associated with the former, but not the latter. This may explain why studies that use only fatal stroke outcomes (and thus over represent hemorrhagic strokes) show only weak relationships between overweight and stroke (NIH, 1998). These prospective studies demonstrate that the risk of stroke shows a graded increase as BMI rises. Ischemic stroke risk is 75 percent higher in women with BMI >27 and 137 percent higher in women with a BMI >32, compared with woman having a BMI <21.

2.4.2.5 Sleep apnea

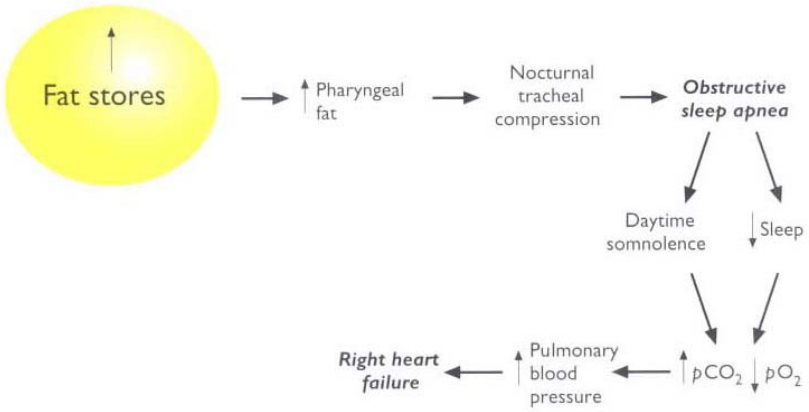


FIGURE 2.5: PATHOPHYSIOLOGICAL MODEL FOR THE DEVELOPMENT OF SLEEP APNEA (Bray, 2003)

Obesity, particularly upper body obesity, is a risk factor for sleep apnea and has been shown to be related to its severity (Young et al., 1993). The major pathophysiologic consequences of severe sleep apnea include arterial hypoxemia, recurrent arousals from sleep, increased sympathetic tone, pulmonary and systemic hypertension, and cardiac arrhythmias (Young et al., 1993). Most people with sleep apnea have a BMI > 30 (Loube et al., 1994). Large neck girth in both men and women who snore is highly predictive of sleep apnea. In general, men whose neck circumference is 43 cm or greater and women whose neck circumference is 41 cm or greater are at higher risk for sleep apnea (Loube et al., 1994).

The sleep apnea-obesity hyperventilation syndrome occurs in five percent of severely obese individuals and is potentially life threatening. Extreme hypoxemia induced by obstructive sleep apnea syndrome may result in heart failure in the absence of cardiac dysfunction (Loube et al., 1994).

2.4.2.6 Dyslipidemia

High total cholesterol

At each BMI level, the prevalence of high blood cholesterol is greater in women than in men. Higher body weight is associated with higher levels of total serum cholesterol

in woman at levels of BMI > 25 (Denke et al., 1994). Several large longitudinal studies also provide evidence that overweight, obesity and weight gain are associated with increased cholesterol levels (Ashley & Kannel, 1974; Shekelle et al., 1981). In addition, the pattern of fat distribution appears to affect cholesterol levels independently of total weight. In women, the incidence of hyper-cholesterolemia also increases with increasing BMI (Manson et al., 1990). Total cholesterol levels are usually higher in persons with predominant abdominal obesity, defined as a waist-to-hip circumference ratio of ≥ 0.8 for women and ≥ 1.0 for men (Reeder et al., 1992).

Low levels of high-density lipoprotein cholesterol

High-density lipoprotein (HDL)-cholesterol levels at all ages and weights are lower in men than in women (NIH, 1998). There is a strong negative correlation between plasma HDL and relative weight (proximity to ideal weight). HDL increases during weight reduction in the obese. Exercise also increases HDL, which reinforces the importance of physical activity in weight reduction. A 10% decrease in weight typically decreases cholesterol by 11 mg·100mℓ^{-1} (National Cholesterol Education Program, 1994).

Normal to elevated low-density lipoprotein cholesterol

The link between total serum cholesterol and CHD is largely due to low-density lipoprotein (LDL). A high-risk LDL-cholesterol is defined as a serum concentration of ≥ 3.4 mmol/ℓ. This lipoprotein is the predominant atherogenic lipoprotein and is therefore the primary target of cholesterol-lowering therapy (NIH, 1998). According to extensive epidemiological data, a 10 mg·100 mℓ^{-1} rise in LDL-cholesterol corresponds to approximately a 10 percent increase in CHD risk over a period of 5 to 10 years (Law et al., 1994).

Small, dense low-density lipoprotein particles

Very little large-scale epidemiological data are available on small, dense low-density lipoprotein (LDL) particles (Rydker et al., 1993). Clinical studies have shown that small, dense LDL particles are particularly atherogenic and tend to be present in

greater proportion in hypertriglyceridemic patients with insulin resistance syndrome associated with abdominal obesity (NIH, 1998).

High triglycerides

In obese people with hyperlipidemia, three-quarters have high triglycerides, one-quarter have high cholesterol and half have a combination of both. The strong association of triglyceride levels with BMI has been shown in both cross-sectional and longitudinal studies, for both sexes and all age groups (Denke et al., 1994; Ashley & Kannel, 1974). In three adult age groups, namely 20 to 44 years, 45 to 59 years and 60 to 74 years, higher levels of BMI, ranging from 21 or less to more than 30, have been associated with increasing triglyceride levels. The difference in triglycerides ranged from 0.68 to 0.74 mmol/ℓ in women (Denke et al., 1994).

2.4.2.7 Diabetes mellitus

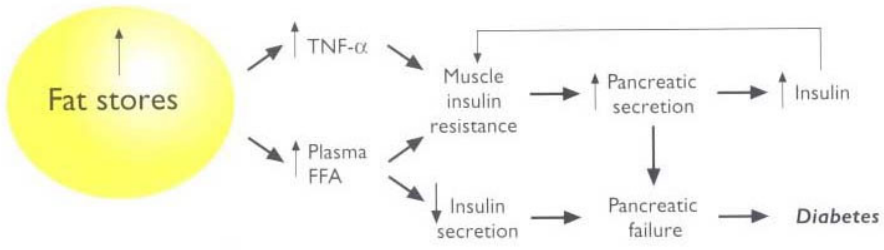


FIGURE 2.6: PATHOPHYSIOLOGICAL MODEL FOR THE DEVELOPMENT OF DIABETES AND INSULIN RESISTANCE (Bray, 2003)

The increased risk of diabetes as weight increases, has been shown by prospective studies in Norway, Sweden, Israel and the United States (Lew & Garfinkel, 1979; Larsson et al., 1981). The Nurses' Health Study, using data on self-reported weights, found that the risk of developing type II diabetes increases as BMI increases from as low as 22 (Colditz et al., 1995). Since women in particular tend to underreport weight, the actual BMI values associated with these risks are likely to be higher than the Nurses' Health Study would suggest (Colditz et al., 1995). An association

between type two diabetes and increasing relative weight is also observed in populations at high risk for obesity and diabetes, such as in American Indians (Lee et al., 1995). The development of type II diabetes has been found to be associated with weight gain after age 18 in women (Colditz et al., 1995). The relative risk of diabetes increases by approximately 25 percent for each additional unit of BMI over 22 kg/m^2. In a prospective study representative of the US population, it was estimated that 27 percent of new cases of diabetes was attributable to weight gain in adulthood of 5 kg or more (Ford et al., 1997). Both cross-sectional and longitudinal studies show that abdominal obesity is a major risk factor for type II diabetes (Despres et al., 1989).

2.4.2.8 Gall-bladder disease and hypercholesterolemia

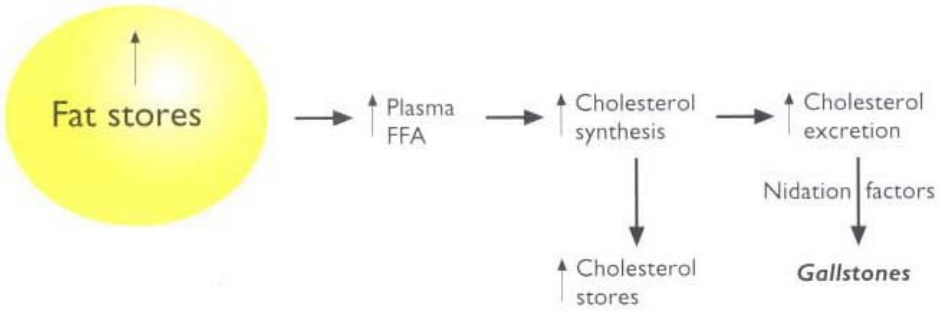


FIGURE 2.7: PATOPHYSIOLOGICAL MODEL FOR THE METABOLISM OF CHOLESTEROL IN THE DEVEOPMENT OF GALL-BLADDER DISEASE (Bray, 2003)

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·100 ml^{-1} 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).

2.4.2.9 Gallstones

The risk of gallstones increases with adult weight. Risk of either gallstones or cholecystectomy is as high as 20 per 1 000 women per year when BMI is above 40, compared with three per 1 000 among women with BMI < 24 (Stampfer et al., 1992). The prevalence of gallstones among women increased from 9.4 percent in the first quartile of BMI to 25.5 percent in the fourth quartile of BMI (NIH, 1998).

2.4.2.10 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 (sleepiness) 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).

2.4.2.11 Osteoarthritis

Individuals who are overweight or obese increase their risk for the development of osteoarthritis (NIH, 1998). The association between increased weight and the risk for development of knee osteoarthritis is stronger in women than in men (Felson et al., 1992). In a study of twin middle-aged women, it was estimated that for every kilogram increase of weight, the risk of developing osteoarthritis increases by 9 to 13 percent. The twins with knee osteoarthritis were generally three to five kg heavier

than the co-twin with no disease (Cicuttini et al., 1996). An increase in weight is significantly associated with increased pain in weight-bearing joints. There is no evidence that the development of osteoarthritis leads to the subsequent onset of obesity (Felson et al., 1992). A decrease in BMI of two units or more during a 10-year period decreased the odds for developing knee osteoarthritis by more than 50 percent and weight gain was associated with a slight increase in risk (Felson et al., 1992).

A randomised controlled trial of six months duration examined the effect of weight-loss on clinical improvement in patients with osteoarthritis (Williams & Foulsham, 1981). Patients taking phenteramine (anorectic drug) had an average weight-loss of 12.6 percent after six months while the control group had an average weight-loss of 9.2 percent. There was improvement in pain free range of motion and a decrease in analgesic use in association with weight-loss. Improvement of joint pain was observed in individuals who had undergone gastric stapling, resulting in an average weight-loss of 45 kg (Mc Goey et al., 1990).

2.4.2.12 Cancer

Colon Cancer

Studies have found a positive relation between obesity and colon cancer in men but a weaker association in women (Lew and Garfinkel., 1979; Bostick et al., 1994). Data from the Nurses' Health Study suggest that the relationship between obesity and colon cancer in women may be similar to that seen in men. Twice as many women with a BMI of > 29 kg/m^2 had distal colon cancer as compared to women with a BMI < 21 kg/m^2 (Giovannucci et al., 1996). Other data from the Nurses' Health Study show a substantially stronger relationship between waist-to-hip ratio and the prevalence of colon polyps on sigmoidoscopy (Giovannucci, 1995). Even among leaner women, a high waist-to-hip ratio is also associated with significantly increased risk of colon polyps (Giovannucci et al., 1996).

Endometrial Cancer

Obesity increases the risk of endometrial cancer. The risk is three times higher among obese women (BMI > 30 kg/m^2) compared to normal weight women (Schottenfeld & Fraumeni, 1996). The absolute risk of this condition is low when compared to breast cancer, heart disease, and diabetes. Adult weight gain is also related to increased risk (Schottenfeld & Fraumeni, 1996).

Breast Cancer

Epidemiologic studies consistently show that obesity is directly related to mortality from breast cancer, predominantly in postmenopausal women, but inversely related to the incidence of premenopausal breast cancer (Lew & Garfinkel, 1979; Rosenberg et al., 1990). Ten or more years after menopause, the premenopausal "benefit" of obesity has dissipated (Schottenfeld & Traumeni, 1996). Among postmenopausal women, peripheral fat is the primary source of estrogens, the major modifiable risk factor for postmenopausal breast cancer. This crossover in the relationship of obesity with breast cancer, pre- and postmenopausal, complicates prevention messages from this common female cancer. Data from the Nurses' Health Study, show that adult weight gain is positively related to risk of postmenopausal breast cancer (Giovannucci et al., 1996). This relation is seen most clearly among women who do not use postmenopausal hormone replacement. Even modest weight gains are positively related to risk of postmenopausal cancer (Huang et al., 1997).

Gallbladder Cancer

Obesity is related to the risk of gallbladder cancer, particularly among women (Garfinkel, 1986). Using a weight index of 100 as the average weight with a corresponding mortality ratio of 1.0 for the cohort, mortality ratios were 1.16 at a weight index of 120 to 129, 1.22 at 130 to 139 and 1.53 at ≥ 140 (NIH, 1998).

2.4.2.13 Musculoskeletal injury

Obesity increases the risk of many musculoskeletal disorders, particularly osteoarthritis and backache. Increased body mass places chronic stress on joints, eventually leading to arthritic changes (Hartz et al., 1986). A sagging abdomen increases the lumbar lordosis (low back curve). An increased low back curve increases the risk of back pain. Generally, weak and inflexible abdominal, spinal and leg muscles compound the problem (Garrow, 1988).

2.4.2.14 Increased surgical risk

Severely obese patients present the surgeon with problems concerning the closure of abdominal wounds. In obese patients the incisions has to be bigger than normal to give the surgeon access to the abdominal contents. Closing the incision is made difficult by the weight of tissue tending to pull the wound open, and by the difficulty of preventing blood oozing from the fat layer, which will not hold sutures (Kozol et al., 1986).

2.4.2.15 Menstrual irregularities and infertility

Obesity in premenopausal women is associated with menstrual irregularity and amenorrhea (Hartz et al., 1979). As part of the Nurses' Health Study, a case control study suggested that the greater the BMI at age 18 years, even at levels lower than those considered obese, the greater the risk of subsequent ovulatory infertility (Rich-Edwards et al., 1994). The most prominent condition associated with abdominal obesity is polycystic ovarian syndrome, a combination of infertility, menstrual disturbances, hirsutism, abdominal hyperandrogenism, and anovulation (Dunaif, 1992). This syndrome is strongly associated with hyperinsulinemia and insulin resistance (Garbaciak et al., 1985).

2.4.2.16 Pregnancy complications

Pregnancy can result in excessive weight gain and retention. The retained weight gain associated with pregnancy was corroborated by the study of Coronary Artery Risk

Development in Young Adults (CARDIA). As a result of their first pregnancy, both black and white young women had a sustained weight gain of two to three kg of body weight (Smith et al., 1994). Another study on a national cohort of women followed for 10 years reported that weight gain associated with childbearing ranged from 1.7 kg for those having one live birth during the study to 2.2 kg for those having three (Williamson et al., 1994). Higher prepregnancy weights have been shown to increase the risk of late fetal deaths (Cnattingius et al., 1998).

Obesity during pregnancy is associated with increased morbidity for both the mother and the child. A tenfold increase in the prevalence of hypertension and a 10 percent incidence of gestational diabetes have been reported in obese pregnant women. Obesity is also associated with difficulties in managing labour and delivery, leading to a higher rate of induction and primary Caesarean section. Risks associated with anaesthesia are higher in obese women, as there is greater tendency toward hypoxemia and greater technical difficulty in administering local or general anaesthesia. Obesity during pregnancy is associated with an increased risk of congenital malformations, particularly of neural tube defects (Prentice & Goldberg, 1996).

A certain amount of weight gain during pregnancy is desirable. The fetus itself, expanded blood volume, uterine enlargement, breast tissue growth, and other products of conception generate an estimated 2.27 – 4.0 kg of extra weight. Weight gain beyond this point is predominantly maternal adipose tissue. It is this fat tissue that, in large measure, accounts for the postpartum retention of weight gained during pregnancy. This retention reflects a postpartum energy balance that does not lead to catabolism of the gained adipose tissue. This may reflect reduced energy expenditure through decreased physical activity, even while caring for young children, but it may also reflect retention of the pattern of increased caloric intake acquired during pregnancy (Shils et al., 1994).

The 1990 Institute of Medicine report made recommendations concerning maternal weight gain (Institute of Medicine, 1990). It recommended that each woman have her BMI measured and recorded at the time of entry into prenatal care. For women with a BMI of less than 20, the target weight gain should be 0.5 kg of weight gain per week during the second and third trimester. For a woman whose BMI is greater than 26, the

weight gain target is 0.3 kg per week during the last two trimesters. Women who are overweight or obese at the onset of pregnancy are advised to gain less total weight during the pregnancy (Institute of Medicine, 1990).

2.4.3 Mortality and obesity

It has been known since Greco-Roman times that manifest obesity can be hazardous to health, however the strength of the association between overweight and increased morbidity and mortality has been found to vary from one epidemiologic investigation to another (Manson et al., 1987). Factors believed to explain such variability include differences in the duration of the period of observation and in the size and nature of the subject population (Van Itallie & Lew, 1990). Cohort studies that have been unable to demonstrate a relationship between body weight and mortality have (a) involved a relatively small population (fewer than 7,000 subjects); (b) had a follow-up period of insufficient duration (less than 10 years); (c) failed to control for the fact that cigarette smoking is more frequent among the nonobese than the obese; (d) failed to adjust for early mortality among subjects in whom clinical or subclinical illness was present at the outset of the study; (e) selected unsuitable subjects [as in the Seven Countries Study (Keys, 1980), which focused on working populations, which tend to be much fitter by virtue of the "healthy worker effect"]; and (f) improperly controlled for "inter-mediate" risk attributes like hypertension and hyperlipidemia that are, at least in part, caused by obesity (Stunkard & Wadden, 1993).

Studies of sufficient size and heterogeneity (with 20,000 or more participants) and longer duration (10 to 30 years) have all shown a positive relationship between overweight and increased relative mortality (Stunkard & Wadden, 1993). In the majority of epidemiologic studies, mortality begins to increase with BMI's above 25 kg/m^2 (Van Itallie & Lew, 1990). The increase in mortality generally tends to be modest until a BMI of 30 kg/m^2 is reached (Van Itallie & Lew, 1990; Van Itallie, 1985; Manson et al., 1987; Troiano et al., 1996). For persons with a BMI of 30 kg/m^2 or above, mortality rates from all causes, and especially from cardiovascular disease, are generally increased by 50 to 100 percent above that of persons with BMI's in the range of 20 to 25 kg/m^2 (Manson et al., 1987; Troiano et al., 1996).

2.4.3.1 Association of body mass index with mortality

Many of the observational epidemiologic studies of BMI and mortality have reported a "U"- or "J-shaped" relationship between BMI and mortality (Allison et al., 1997). Mortality rates are elevated in persons with low BMI (usually below 20) as well as in persons with high BMI (Manson et al., 1987; Troiano et al., 1996). In some studies, adjustment for factors that potentially confound the relationship between BMI and mortality, such as smoking status and pre-existing illness, tends to reduce the upturn in mortality rate at low BMI (Manson et al., 1987), but in a meta-analysis the higher mortality at low BMI's was not eliminated after adjustment for confounding factors (Seidell, 1995). It is unclear whether the elevated mortality observed at low BMI is due to an artefact of incomplete control for confounding factors, inadequate body fat and/or inadequate body protein stores that result from unintentional weight-loss, or individual genetic factors. Currently, there is no evidence that intentional weight gain in persons with low BMIs will lead to a reduction mortality rates (NIH, 1998).

Many of the observational epidemiologic studies suggest that the relationship between BMI and mortality weakens with increasing age (Stevens et al., 1998; Diehr et al., 1998). Older adults are more likely than younger adults to have diseases that both increase mortality and cause weight-loss leading to lower body weight (Baumgartner et al., 1995; Losonczy et al., 1995; Fried et al., 1998). In addition, as people age, they tend to have larger waist circumferences that increase their risk of mortality even at lower BMI's. Weight gain in middle age is positively related to risk of mortality in old age. The impact of smoking on body weight and mortality is likely to be much stronger in older adults because of the cumulative health effects of smoking (Losonczy et al., 1995; Willett et al., 1991).

2.4.3.2 Weight-loss and mortality

A number of studies of "generic weight-loss" (cause of weight-loss unknown), weight cycling (cycles of weight-loss followed by weight regain), and mortality have been published (Andres et al., 1993; Williamson & Pamuk, 1993). In most, but not all of these studies, generic weight-loss and weight cycling are associated with increases in mortality (Andres et al., 1993; Williamson & Pamuk, 1993; Williamson, 1996).

None of these studies differentiated between intentional and unintentional weight-loss (Williamson, 1996). Very little is currently known about factors related to intentional and unintentional weight-loss in the general population or about the relationship between weight-loss intention and mortality (Williamson, 1997).

2.5 ETIOLOGY OF OBESITY

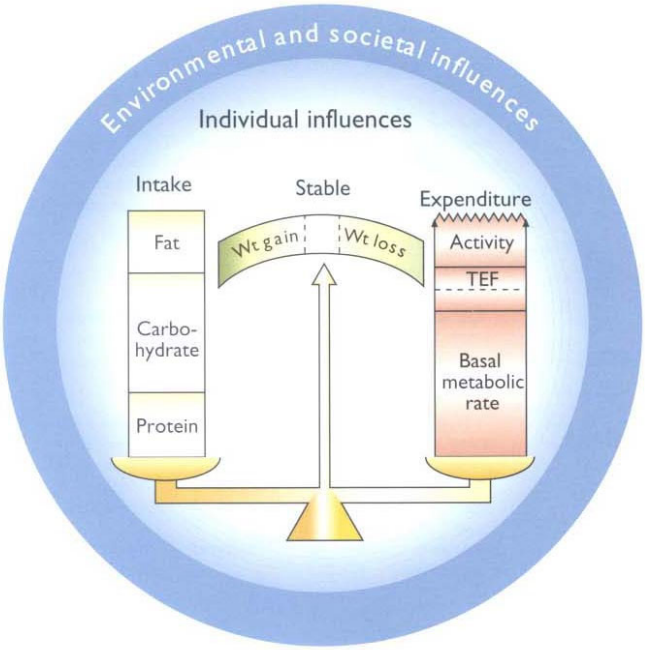


FIGURE 2.8: RELATIONSHIP OF VARIOUS FACTORS ASSOCIATED WITH THE CONTROL OF OBESITY (Bray, 2003)

TEF = Thermic Effect of Food

Wt = Weight

When caloric intake exceeds energy expenditure the excess calories are stored in adipose tissue. Obesity results if this net positive balance is prolonged. Although it is often assumed that obesity results simply from overeating or a sedentary life-style, it 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 caloric intake and energy expenditure resulting from a complex interaction of genetic,

environmental, nutritional, physiological, psychological, social and cultural factors (Rippe et al., 1998).

FIGURE 2.9: ILLUSTRATED GENOTYPE – ETIOLOGICAL BASIS OF OBESITY

Evolutionary biology may explain some of the story. Over a third of a century ago Neel at the University of Michigan proposed that the pressure of natural selection endowed our ancestors with a "thrifty" genotype, which boosted the ability to store fat from each feast in order to sustain people through the next famine (Neel, 1962). Unlike our hunter-gatherer ancestors whose food supply was scarce and sporadic, our food supply in western societies is abundant and constant. As a consequence, the ability to store fat efficiently has actually become a liability with obesity as the result (Oeser, 1997).

2.5.1 Genetic factors

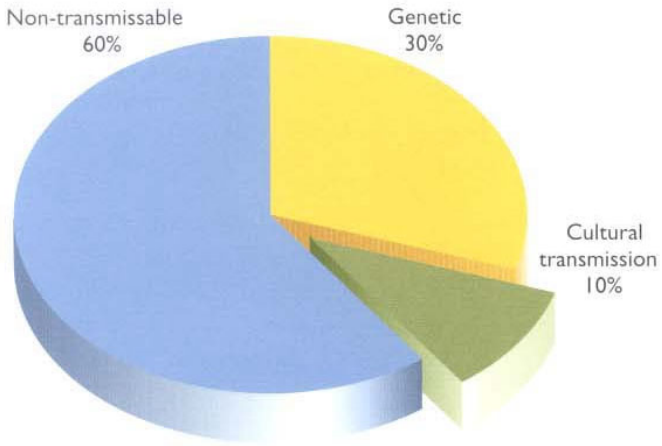


FIGURE 2.10: GENETIC FACTORS INVOLVED IN THE DEVOLOPMENT OF OBESITY (BRAY, 2003)

It has been long known that the tendency to gain weight runs in families. Early studies estimated that hereditary influence accounted for up to 80% of the tendency to gain weight, but 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). 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; Reed et al., 1996). Persons with mutations in the gene that encodes for adipose beta-3 receptors appear prone to weight gain (Clement 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).

A large number of studies have shown that obesity is both under genetic control and influenced by several environmental factors, including energy expenditure and intake. Studies using twin-pair designs and families with adopted children have shown that BMI and other measures of fatness are under strong genetic control (Bouchard, 1994).

There is no doubt that environmental factors, such as food intake, smoking status, and physical activity level, also influence the development of obesity (Bouchard, 1994). Several studies indicate that both food intake and spontaneous physical activity levels are influenced partly by genetic factors, with heritability (i.e. the ratio of genetic variance to total phenotypic variance of the trait) up to 33% (Bouchard, 1994).

It has been hypothesized that development of obesity is, in part, due to differential effects of environmental influences for those who are genetically predisposed compared with those who are not (Bouchard, 1994; Price et al., 1991). In this context, a few studies in animals and humans have shown that food intake seems to play a specific role for obesity development in association with a predisposition to obesity (West et al., 1992; Astrup, 1993; Heitmann et al., 1997). For instance, obesity-prone mice have been found to gain weight at a much faster rate than wild-type mice fed the same high-fat diets (West et al., 1992), suggesting a gene-environment interaction between the high-fat diet and subsequent weight gain. Compared with non-obese control subjects, an impaired ability to increase the ratio of fat to carbohydrate oxidation in response to a high-fat diet has been shown in post-obese women (Astrup, 1993), suggesting that this obesity-prone group is particularly susceptible to weight gain when consuming such a diet. With a high-fat diet, obese women with a familial history of obesity had a risk of major weight gain almost 10 times that of woman without such a predisposition (Heitmann et al., 1997). It may be hypothesized that a genetic susceptibility to obesity modifies the response to the other aspect of energy balance, namely physical inactivity (Heitmann et al., 1997). The genetic basis of obesity is complex with the probability of a number of interacting genes being involved (polygenic inheritance). Each of the main components of energy balance relationship (energy intake, body mass ratio, the thermic effect of food and physical activity) and even body fat distribution has a distinct genetic basis (Kulkarni & Kaur, 1999).

2.5.2 Environmental factors

The environment is a major determinant of overweight and obesity. Environmental influences on overweight and obesity are primarily related to food intake and physical activity behaviours (James, 1995). There is an overall abundance of palatable, calorie-

dense food. Aggressive and sophisticated food marketing in the mass media, supermarkets, and restaurants, and the large portions of food served outside the home, promote high calorie consumption. Sociocultural traditions promote overeating and the preferential consumption of high calorie foods. Even when caloric intake is not above the recommended level, the number of calories expended in physical activity is insufficient to offset consumption. Mechanization limits the necessity of physical activity required to function in society. Many people are entrenched in sedentary daily routines consisting of sitting at work, sitting in traffic, and sitting in front of a television or a computer monitor for most of their waking hours (NIH, 1998).

In this obesity-promoting environment, individual attitudes and behaviours are critical in weight management. Individuals may need extended treatment in clinical or community settings to enable them to cope with the complexities of long-term weight management, especially if there is a history of unsuccessful attempts at self treatment (Thomas, 1995). When the typical daily routine is so strongly biased towards promoting and perpetuating overweight and obesity, very high levels of knowledge, motivation, personal behavioural management skill, and lifestyle flexibility are required for an overweight or obesity-prone individual to avoid becoming over-weight, or progressing to moderate or severe obesity (NIH, 1998).

There are undoubtedly some inter- and intrapopulation variations in the genetic predisposition to become overweight or obese. Several lines of evidence suggest that genetic factors alone cannot explain the demographic and ethnic variations in overweight and obesity prevalence. There is a difference in obesity prevalence among low- and high-income white women in industrialized societies (Sobal & Stunkard, 1989; Kumanyika, 1994; Ravussin et al., 1994). Studies of populations, including migration studies, have shown an increase in average body weight in those who move from a traditional to a Westernized environment (Ravussin et al., 1994; Kawate et al., 1979).

Culturally determined attitudes about food, physical activity, and factors that vary with income, education, and occupation may increase the level of difficulty in weight management. Body image concerns and other motivations for avoiding obesity or controlling weight within given limits also vary with ethnic background, age, sosio-

economic status, and gender. The competence of practitioners in working with diverse sociocultural perspectives can be a critical factor in the success of obesity treatment (Kumanyika & Morssink, 1997).

2.5.3 Nutritional factors

Numerous metabolic studies have shown that high fat diets may lead to a high-energy intake and hyperphagia. The reason may be that fat has a weaker effect on the satiety centre and on heat production (diet-induced thermogenesis) and it possesses a higher energy density compared to carbohydrates. Fats are highly palatable and heighten the flavour of food stuffs which leads to their passive over-consumption. This ultimately increases fat deposits and causes obesity and related problems (Poppitt, 1995).

2.5.4 Physiological factors

These involve the impairment of the central mechanism regulating appetite and food intake, which is thought to be regulated by a complex interplay of neurotransmitters in the hypothalamic region of the brain. Approximately 1-2% of obesity can be ascribed to lesions in hypothalamic regulatory centres. Such lesions may be due to trauma, tumours, inflammatory processes, or carotid artery aneurysms (Zhang et al., 1994).

2.5.5 Psychological factors

The psychogenic theory of obesity long held that obesity resulted from an emotional disorder in which food intake relieved the anxiety and depression to which obese persons are usually susceptible. Stress associated with traumatic emotional events has been held responsible to certain cases of obesity and has been implicated in the pathogenesis of eating disorders such as night-eating syndrome and bulimia (Bray & York, 1979).

2.5.6 Cultural, economic and social factors

Cultural and economic factors play critical roles in the etiology of obesity and the increase in obesity prevalence. Culture refers to the learned patterns of behaviour and

thought characteristics of a social group; culture forms the context of people's lives, and to a large extent that context is beyond an individual's control. Culture includes material aspects, such as diet and activity patterns that are directly causal to fat deposition and the causation of obesity. Culture also has social and cognitive components, including social pressures and ideals of beauty that are more indirectly related to obesity (Ritenbaugh, 1982; Ross & Mirowsky, 1983). Cultural context includes many unconscious and taken-for-granted circumstances that greatly limit individual choice and behaviour. Some things are simply considered "normal" and unquestioned parts of life including driving cars instead of walking, eating calorie-dense industrially produced foods, and watching television for hours per day. The cultural and economic contexts, historically shaped by powerful socio-economic forces like corporations, constrain individual choices in habitual behaviours. At the same time, a consumer-oriented capitalist economy establishes an illusion of personal choice about work, diet and activity patterns. It is difficult for individuals to swim against the current of cultural forces that lead towards fatness; it is a culturally constructed "obesogenic" environment (Shore, 1996; Sobal, 1991; Sobal & Stunkard, 1989).

It is remarkably easy to become fat and a large percentage of people do so. The incidence of obesity is rising at a striking rate, so that many Public Health leaders are calling it an epidemic (Mokdad et al., 1999). In this context, thinness can be a marker of social distinction for women. In a capitalist economy, increased physical activity has become a commodity to be purchased (exercise equipment, health club membership) and lower calorie intake often involves the purchase of special commodity (diet foods) (Sobal, 1991). Thinness has been invested in its historical symbolic association to poverty and manual labour to become a marker of excess leisure time and economic prosperity. Obesity and overweight, which are positively evaluated in many societies, have therefore become issues of social stigma and ridicule. The difficulty of losing weight or maintaining weight-loss in a cultural context cannot be minimized. The cultural and economic contexts predispose individuals to failure (Shore, 1996).

2.6 PATHOPHYSIOLOGICAL FACTORS UNDERLYING OBESITY

In order to develop new treatments for obesity, it is important to understand the physiological pathways that regulate the energy balance (both intake and expenditure) and to identify factors causing obesity.

2.6.1 Energy balance equation

Energy intake = energy expenditure + energy storage
Adipose tissue (Triacylglycerol)

As illustrated above energy intake and energy expenditure play a pivotal role in controlling body fat stores (Arner, 1998). The energy intake depends on appetite, which is mainly controlled by glucostatic and lipostatic feedback systems. The energy expenditure depends on heat production (diet induced thermogenesis, DIT, 10-12%), metabolism (basal metabolic rate, BMR, 65%) and physical exercise (variable). Any imbalance between these is translated into a change in fat stores and obesity. This balancing act also involves various neural and endocrine signals acting peripherally as well as centrally which cause disturbance and derangement of various other physiological functions as well (Bray, 1992).

2.6.2 Energy intake regulation in obesity

The nerve centre for the regulation of energy balance is the hypothalamus, which integrates neural, hormonal, nutrient messages from elsewhere in the body and sends signals to the higher centres leading to feeling of hunger or satiety. The hypothalamus also controls energy expenditure via the autonomic nervous system and pituitary hormones (Kalkarni & Kaur, 1999).

The neuroendocrine axis involved in energy balance regulation is the hypothalamic links with the adrenals, gonads and the sympathetic nervous system. The hypothalamic arousal by physical and mental stressors result in fight or flight reaction, which leads to changes in these links (Frayn, 1995). The fight reaction occurs via the

sympathetic pathways to help gain control by increasing the readiness of circulatory factors and the mobilise substrates needed to meet the challenges.

When the individual loses control and ends up in a defeated, submissive, helpless situation, there is hyperactivity of the cortisol axis, hypercortisolism and decreased secretion of sex hormones. This type of reaction has been shown to be followed by abdominal fat accumulation and metabolic aberrations, including signs of insulin resistance (Polkow, 1988). The reaction patterns may also shift from one type to another due to perceived stimulus, varying between and among individuals. It has been shown that long-term stress, in addition to being responsible for over-eating through neural mechanisms, results in elevated plasma levels of glucocorticoids. Glucocorticoid excess results in increased hepatic gluconeogenesis and diminished arterial glucose transport and utilisation (Bjorntorp, 1991).

A large number of hypothalamic neurotransmitters have also been implicated in the control of energy balance. Those that increase food intake generally suppress sympathetic nervous system activity and thus thermogenesis, whereas the reverse is true for neurotransmitters that decrease the appetite (Amatruda et al., 1985).

2.6.2.1 Neuropeptide Y

Of the few neurotransmitters that stimulate feeding, neuropeptide Y (NPY) has attracted the most interest. It is a linear 36 amino acid peptide, which was first isolated from the porcine brain. It is a member of the pancreatic polypeptide (PP) family of regulatory peptides that includes the endocrine peptides, peptide YY (PYY) and PP. It has been shown to have powerful and complex effects on feeding, anxiety, circadian rhythms, reproduction, pituitary-adrenocortical axis function, memory retention, seizures, thermoregulation, and cardiovascular and gastro-intestinal functions (Kalkarni & Kaur, 1999). One of the most striking actions of NPY is the induction of feeding. NPY activates a heterogeneous population of at least six receptor subtypes, Y1-Y6, of which Y1-Y5 receptors are required for appetite regulation by NPY (Inui, 1999).

Neuropeptide Y is the most powerful appetite stimulant known in rodents. It has been reported to increase the triglyceride content in white fat and after five days of intracerebral administration it doubled the fat stores in both lean and ob/ob mice. Its expression is also reported to be increased in ARC, PVN and DMN in the ob/ob mouse and Zucker rat. Genetic studies have not shown any association with the genes for neuropeptide Y1 or Y5 receptors in human obesity (Kulkarni & Kaur, 1999).

The prototype NPY5 antagonists have been shown to inhibit feeding in genetically obese rats and in food deprivation (Bray, 1992). Their effects in rodents with diet-induced obesity (the nearest model to human obesity) have not yet been reported. It is speculated that NPY5 antagonists could be effective anti-obesity drugs.

2.6.2.2 Melanin concentrating hormone

Melanin concentrating hormone (MCH) is a cyclic 19 amino-acid neuropeptide identified in the intermediate lobe of the teleost fish pituitary from which it is released into circulation and causes aggregation of melanophores. MCH receptors are found exclusively in the zona incerta and the lateral hypothalamus. It is reported to stimulate food intake after central administration, and expression of gene encoding MCH is unregulated in ob/ob mice and through fasting in wild type of mice (Wilding, 1997). Leptin decreases expression of this gene in the hypothalamus via co-existing leptin receptors. The exact mechanism of action of MCH in the hypothalamus is not known (Kulkarni & Kaur, 1999).

2.6.2.3 Serotonin

The neurotransmitter serotonin (5-hydroxy-tryptamine, 5-HT) has an established role in decreasing appetite. Widely prescribed anorectic drugs also cause hypophagia by increasing brain extracellular 5-HT levels (Miller, 1993). The main antagonistic system for this is noradrenergic. Besides regulating appetite, serotonin is also responsible for the selection of foods of major constituents. The high local levels of serotonin result in preference for proteins and low levels the opposite. The high carbohydrate and low protein diet promotes uptake of serum tryptophan in the brain and promote its conversion to serotonin with decreases in feeding. This forms a self-

regulatory mechanism for the serotonergic system. In patients with decreased insulin sensitivity, this system may be disturbed, causing over-consumption of carbohydrates resulting in clinical picture of "carbohydrate craving obesity" (Kulkarni & Kaur, 1999).

2.6.2.4 Lipoprotein lipase

Human fat cells do not synthesise lipids but derive most of their lipids for storage from circulating triglycerides (TGs). Lipoprotein lipase (LPL) provides these lipids by hydrolyzing TGs and acts as "metabolic gate-keeper". Lipoprotein lipase is synthesized in heart and skeletal muscle, adipose tissue, and several other extrahepatic tissues. After secretion, the enzyme is transported to its functional site, which is at vascular endothelium bound to heparin sulphate. Fatty acids are liberated through the hydrolytic action of LPL on TG in circulating very low-density lipoproteins (VLDL) and chylomicrons. The changes in LPL have been reported in metabolic disorders associated with ageing like insulin resistance, obesity and impaired hormonal balance (Kulkarni & Kaur, 1999). Adipose LPL is also reported to be elevated in obesity. When obese subjects lose weight, LPL is elevated further, suggesting attempts to maintain lipid stores during fasting and to replenish lipid stores during refeeding. The muscle LPL is regulated in response to feeding and fasting. Feeding results in an increase in adipose enzyme and a decrease in muscle LPL. An increased adipose/muscle LPL ratio would partition dietary lipid into adipose tissue and explains some of the variability in weight gain when humans are exposed to excess calories (Bergö et al., 1997).

2.6.2.5 Leptin

Leptin (from Greek leptos meaning thin) is a plasma protein exclusively produced in white adipose tissue. It is the product of ob gene that is defective in ob/ob mice (Kern, 1997). It plays an important role in control of body weight, energy expenditure, reproduction and neuroendocrine signalling (Zhang et al., 1994). It is secreted into circulation and then transported to the brain through a modified area of blood brain barrier near the arcuate nucleus or via the choroid plexus, where short-form leptin receptors are sited which transfer leptin to the hypothalamus. Once within the central

nervous sistem (CNS) leptin acts on long-term receptors to stimulate satiety. It also mediates a negative feedback control on white adipose tissue. Through action in the hypothalamus, possibly mediated by inhibition of neuropeptide Y (NPY) secretion and stimulation of corticotrophin releasing hormone (CRH) secretion, leptin decreases food intake, stimulates sympathetically (SNS)-mediated brown adipose tissue (BAT) thermogenesis, and reduces the parasympathetically (PNS)-mediated insulin secretion. The increased thermogenesis and decreased energy intake leads to the energy deficit that is satisfied by mobilisation of fat stores. Leptin may enhance the glucose uptake into nonadipose tissue, such as muscle, to improve glucose disposal and insulin sensitivity. Leptin levels show diurnal rhythm in both sexes, with the highest leptin levels found between midnight and early morning hours (Auwerx & Staels, 1998). Production in white adipose tissue (WAT) is enhanced by body fat, feeding, fever, insulin, glucocorticoids, cytokines and its production is decreased by fasting, cold, exercise, sympathetic activity acting on β_3 adrenoceptors, testosterone, thyroid hormone and thiazolidinediones (Bray, 1992).

The ob/ob mice with a mutation in the ob gene don't secrete leptin and exhibit obesity, hyperphagia, hyperglycaemia, insulin resistance and increased expression of NPY in the hypothalamus. Injection of small quantities of leptin into ob/ob mice reverses hyperinsulinemia, decreases NPY level and normalises food intake and energy expenditure (Sinha et al., 1996).

Plasma leptin levels are closely correlated with body fat; they are decreased in states of severe malnutrition such as anorexia nervosa and elevated in obese humans suggesting resistance to the central effects of leptin in obesity. The importance of leptin in humans was recently established with the description of two cousins with morbid obesity of early onset who had a mutation in the leptin gene (Wang et al., 1997). Leptin receptor polymorphism has also been linked to obesity in three sisters (Montague et al., 1997). Although low leptin production or mutations of the leptin gene may not be a direct or common cause of human obesity, understanding of its action and regulation may yet help in the development of new therapeutic strategies (Clement, 1998).

2.6.2.6 Ghrelin-growth hormone releasing peptide

Growth hormone (GH) is produced and secreted from the anterior pituitary gland and extends its action by binding to GH receptor. GH does not directly stimulate growth, but induces insulin-like growth factor-I (IGF-I) production in liver. Increased IGF-I stimulates postnatal growth. GH also directly activates several tissues to control metabolism, to regulate water and electrolyte balances, and to control cell growth and differentiation. GH is released from the pituitary gland in a pulsatile manner, regulated by episodic changes in two hypothalamic hormones, growth hormone releasing hormone (GHRH) and somatostatin. GH secretion is stimulated by GHRH and inhibited by somatostatin. Many efforts have been made to develop and improve potential synthetic growth hormone secretagogues (GHS's). GHS's are divided into two classes: peptidergic and non-peptidergic. These GHS's act on the pituitary and hypothalamus to release GH, not through the GHRH receptor but through an orphan receptor, the growth hormone secretagogue receptor (GHS-R). These facts indicate that an unknown endogenous ligand for GHS-R may exist. As the endogenous ligand for GHS-R has not been identified, the regulatory mechanism of this growth hormone secretagogue system remains elusive. Progress in molecular and cellular biology has made it possible to use orphan receptors as assay systems to purify unknown ligands. This technique uses cells expressing the orphan receptor to monitor intracellular changes of second messengers, such as CAMP (adenosine 3',5'-cyclic phosphate) production, calcium concentration and arachidonic acid release. Using this technique, scientists succeeded in the purification of an endogenous ligand for GHS-R from the stomach and named it ghrelin (Kojima et al., 1999; Nakazato et al., 2001).

The loss of appetite in bypass patients may be linked to the recent discovery of the gastric hormone ghrelin. Ghrelin may turn out to be one reason people feel hungry in the first place and why it's so hard for dieters to keep weight off. Understanding how ghrelin works could even lead to effective weight-loss drugs or drugs to promote weight gain in anorexics.

- Ghrelin levels rise in the bloodstream significantly before meals and drop afterwards.

- Ghrelin injection just before a meal causes people to eat more than they normally would.

- Ghrelin levels are higher on average in people who have lost weight from dieting.

- Bypass patients have only a quarter as much ghrelin as people of normal weight.

Ghrelin is produced mostly by cells in the stomach. If large parts of that organ are cut off from the rest of the digestive system, it may inhibit the production of the hormone (Lemonick, 2002; Kojima et al., 1999; Nakazato, 2001).

2.6.3 Energy expenditure regulation in obesity

Energy expenditure has three components – resting metabolic rate, physical activity and thermogenesis. Among these, thermogenesis plays an important role in regulating the body's fat stores. By definition, thermogenesis is a process which is used to generate heat from food energy and also in response to stress. It represents about 10% of energy expenditure. It mainly occurs in brown adipose tissue (BAT). Catecholamines (adrenaline and noradrenaline) stimulate thermogenesis in BAT, as well as lipolysis in white adipose tissue (WAT). Any drug which could increase thermogenesis might, therefore, be useful for the treatment of obesity (Mason, 1997).

2.7 BIO-ENERGETICS OF METABOLISM

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 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).

2.7.1 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-thoxacic 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 x 7 x 7) different combinations possible (Péronnet et al., 1987). Some common fatty acids are oleic acid, palmitic 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 these 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).

2.7.1.1 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).

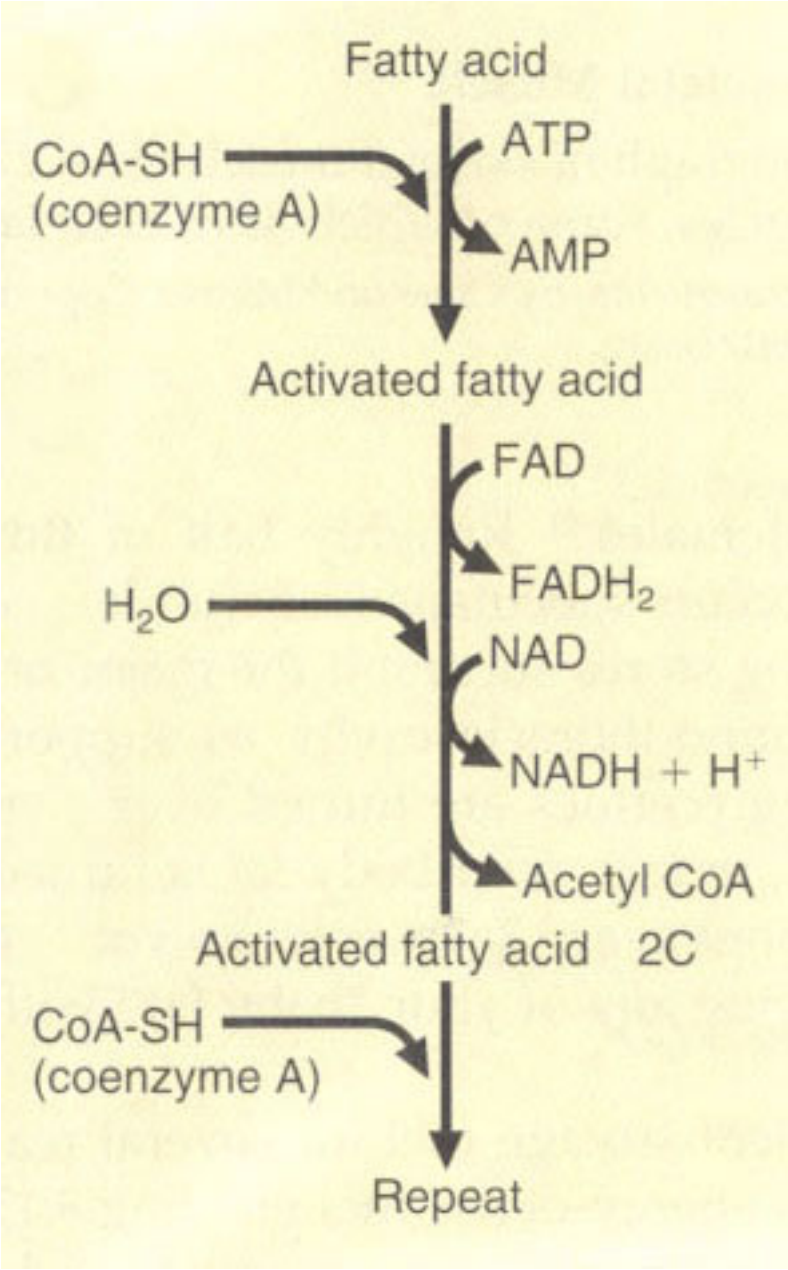


FIGURE 2.11: BETA OXIDATION (PLOWMAN & SMITH, 1997)

SUMMARY OF BETA OXIDATION

- Does not directly utilise O_2, 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 $FADH_2$ + 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).

2.7.1.2 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 x 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 triglycericle molecule catabolized for energy. This quantity represents 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).

2.7.1.3 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).

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).

2.7.1.4 Respiratory quotient and low rates of fat oxidation

If the composition of nutrient intake is an important factor in the genesis of obesity, one might expect that the composition of nutrient oxidation would play a role. The nonprotein respiratory quotient, which is the ratio of carbohydrate to fat oxidation, ranges from a value of about 0.80 after an overnight fast in which fat is the main oxidative substrate (McNeil et al., 1988) to values close to, and beyond 1.00 after a large carbohydrate meal in which glucose is the major substrate (Felber et al., 1987). Under unusual conditions of massive carbohydrate loading, respiratory quotient values can go beyond 1.00, indicating de novo lipogenesis (Just et al., 1990; Acheson et al., 1987). Apart from these effects of diet composition, the respiratory quotient is also influenced by recent energy balance (negative balance causing more fat oxidation), gender (females tending toward carbohydrate oxidation and fat storage), adiposity (higher fat mass means higher fat oxidation), and family membership, suggesting genetic determinants. All of these factors only explain about 40% of the variance in the 24-hour respiratory quotient as measured in a respiratory chamber (Zurlo et al., 1990). In a longitudinal study in Pima Indians, it was found that even after adjusting for these known effects, the 24-hour respiratory quotient showed considerable inter-individual variation and predicted weight gain (Zurlo et al., 1990). Those in the 90th percentile for respiratory quotient ("carbohydrate oxidizers") had a 2.5 times greater risk of gaining 5 kg or more body weight than those in the 10th percentile. This effect was independent of a "low" or "high" 24-hour metabolic rate. Thus "carbohydrate oxidizers" tend to conserve fat and over time gain weight faster than "fat oxidizers"; conversely, a low respiratory quotient ("exaggerated" fat oxidation) is associated with a slower weight gain (Zurlo et al., 1990).

2.7.1.5 De novo lipogenesis

Lipogenic enzyme activities are detectable in the liver of humans, but are 4-to-70 fold lower than in rat or bird livers. In vitro lipogenesis can also be demonstrated in

human adipose tissue (Chascione et al., 1987). Whole body net de novo lipogenesis can be readily demonstrated (as indicated by a respiratory quotient greater than 1.00) during parenteral infusions or peritoneal dialysis (Just et al., 1990; Acheson et al., 1987). Large amounts of oral carbohydrate are needed before small amounts are converted into triglycerides. Acheson et al. (1987) found that the acute ingestion of 2,000 kcal of simple sugar (500g of dextrin maltose) resulted in only a few grams of lipid production, and even massive carbohydrate overfeeding (about 5,000 kcal, 85% carbohydrate) for several days after saturation of glycogen stores resulted in about 150g/day of lipid synthesis. None of the situations in which de novo lipogenesis has been demonstrated could be considered physiological, and indeed the respiratory quotient does not exceed 1.00 under normal conditions. Studies of isocaloric diets rich in carbohydrates (especially simple carbohydrates) have shown an increase in triglyceride concentrations and very low-density lipoprotein (VLDL) ApoB pool size, but this is not equivalent to lipogenesis (Coulston et al., 1987; Reaven, 1965). The increase in VLDL is largely due to a decrease in the conversion of VLDL to intermediate-density lipoprotein (IDL) and low-density lipoprotein (LDL), although those individuals who are susceptible to carbohydrate-induced hypertriglyceridemia usually also demonstrate an increased VLDL production rate as well. De novo lipogenesis in humans should be regarded as negligible under the dietary conditions of industrialized countries (Reavan et al., 1965; Quarfordt et al., 1970).

2.8 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 sqeezed 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 remains 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 non-obese people (Mc Ardle et al., 1996). Thus once adipocyte hypertrophy has ended, cell number becomes the key factor determining any further extent of obesity.

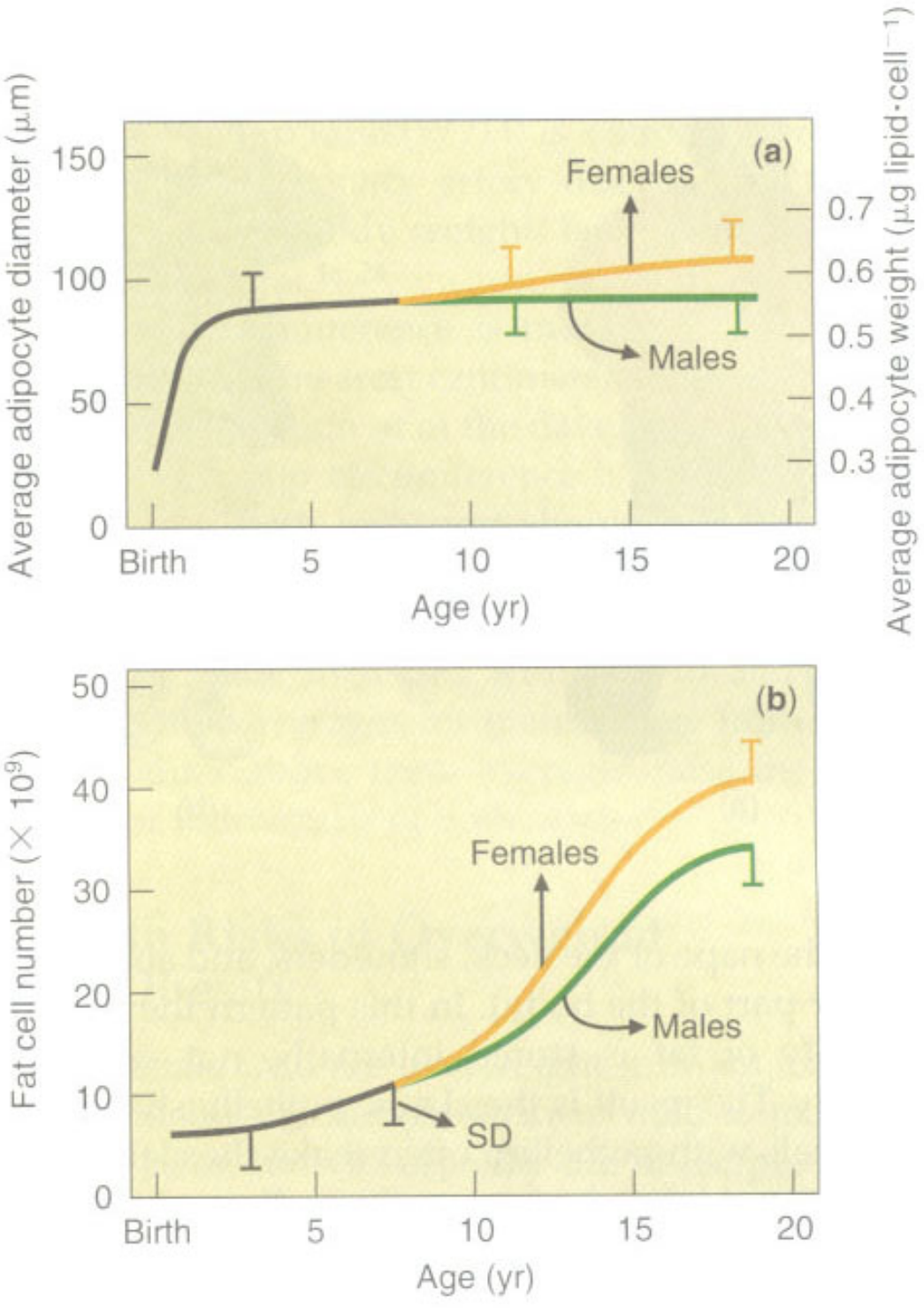


FIGURE 2.12: CHANGES IN ADIPOSE CELL SIZE AND NUMBER WITH GROWTH (PLOWMAN & SMITH, 1997)

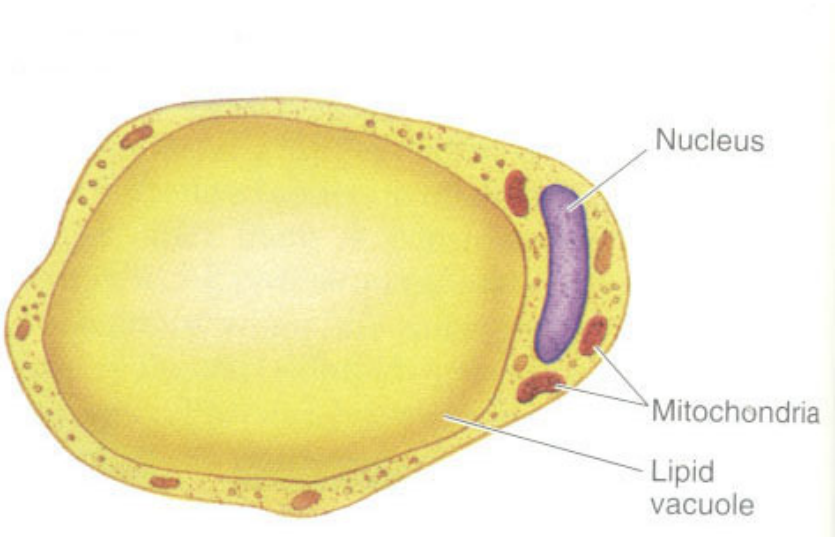


FIGURE 2.13: WHITE ADIPOSE CELL (PLOWMAN & SMITH, 1997)

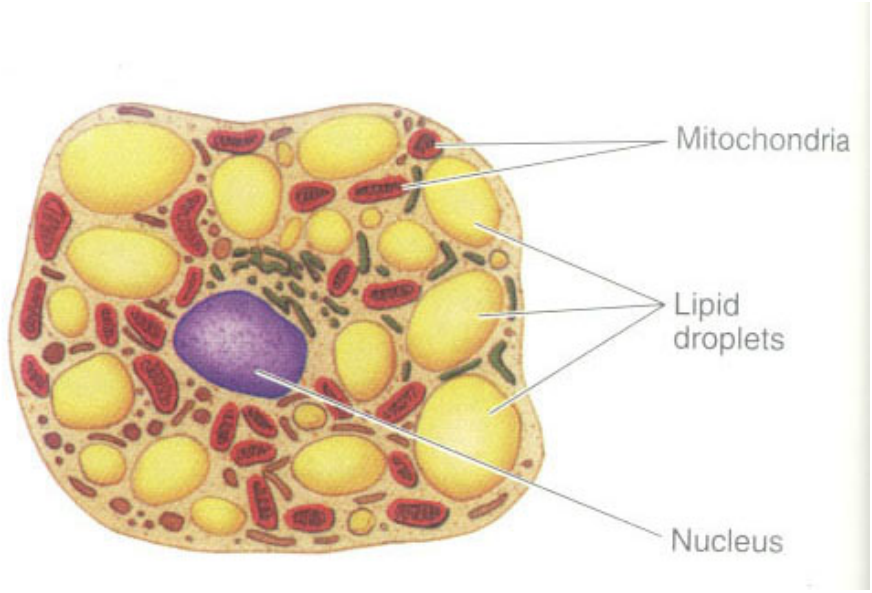


FIGURE 2.14: BROWN ADIPOSE CELL (PLOWMAN & SMITH, 1997)

2.9 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 ºC) and neutral ambient temperature (27 - 29 ºC) 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 $kcal.m^2$ (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).

2.9.1 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., 1971; 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).

2.9.2 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 $mL.kg^{-1}$ min. of oxygen or 1 $kcal.kg^{-1}.hr^{-1}$. 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; 1970). Recently the terms 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.:

- Resynthesis of ATP and CP;
- Resynthesis of lactate to glycogen (Cori cycle);
- Oxidation of lactate in energy metabolism

- Restoration of oxygen to blood;

- Thermogenic effects of elevated core temperature;

- Thermogenic effects of hormones, particularly the catecholamines-epinephrine and norephinephrine; and

- 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).

2.9.3 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).

2.10 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).

2.10.1 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 a 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).

2.10.2 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).

2.10.3 Physical activity

Reduced physical activity, as a cause of obesity is an obvious and attractive hypothesis. The energy expended in physical activity is quite variable, and the secular increase in obesity parallels the increase in sedentary lifestyles. Until the introduction of the doubly labelled water method to measure energy expenditure in free-living conditions, there has been no satisfactory method by which to assess the impact of physical activity on daily energy expenditure (Schoeller & Webb, 1984). Under the artificial conditions of a respiratory chamber, large differences in energy expenditure between individuals (100 to 800 kcal/day) could be attributed to differences in spontaneous physical activity (Ravussin et al., 1986). These differences may be much larger in free-living conditions in which voluntary physical activity varies widely among individuals.

The energy cost of a given activity is proportional to body weight and therefore is higher in obese individuals, although obesity is generally associated with lower activity levels (Bullen et al., 1964; Chirico & Stunkard, 1960). Ferraro et al., (1991) found a negative relationship between the energy expenditure of activity and the degree of obesity, implying that the higher cost of activity was more than offset by the lower activity level among obese subjects. Studies in a large number of subjects are needed to assess physical activity and its impact on the development of obesity. Data on more than 50 Pima Indians suggest that the level of physical activity decreases with both increasing age and increasing adiposity (Rising et al., 1991; Ravussin et al., 1991). Whether a low level of physical activity is the cause or the consequence of obesity cannot be derived from such cross-sectional studies.

2.10.4 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).

2.10.5 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 (Elliot, 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).

2.11 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).

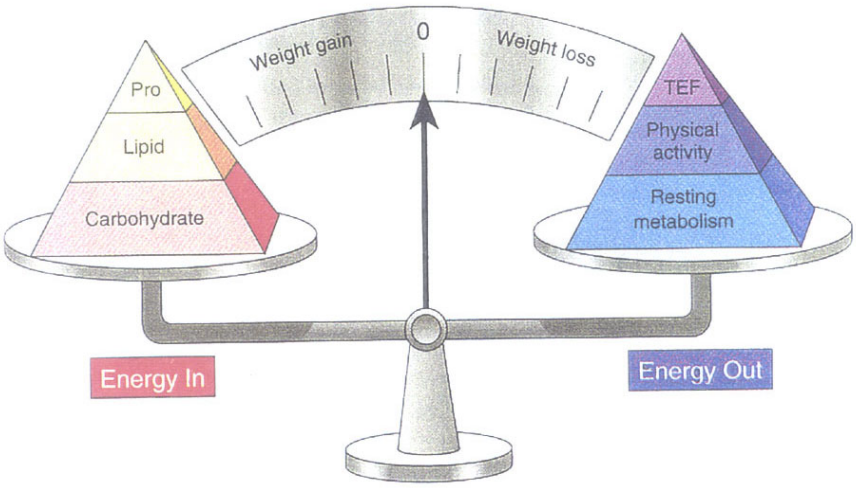


FIGURE 2.15: THE ENERGY BALANCE EQUATION (TEF REFERS TO THE THERMIC EFFECT OF FOOD) (MC AARDLE ET AL., 1996)

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:

Caloric balance	=	+	food ingested (kcal)
		-	basal or resting metabolic rate (kcal)
		-	thermogenesis (kcal)
		-	work or exercise metabolism (kcal)
		-	energy excreted in waste products (kcal)
			(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).

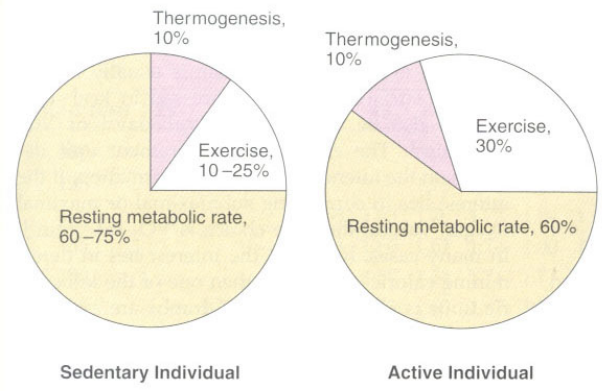


FIGURE 2.16: ENERGY EXPENDITURE (PLOWMAN & SMITH, 1997)

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 diets 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 et al., 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).

2.12 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.

2.12.1 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 melano-cortin receptor as key component of a 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 decreasing 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).

2.12.2 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).

2.13 METABOLIC ADAPTATION

Common experience suggests that obese individuals are very resistant to the weight-loss effects of a hypocaloric diet, but are very susceptible to regaining the weight while eating an apparently normal diet. Does adaptation explain why an individual of normal weight does not gain weight on a hypercaloric diet as quickly as predicted, but then loses it with ease? Do differences in metabolic responses explain the great variation among individuals in response to the same change in energy balance?

In their work on the biology of semi starvation, Keys et al. (1950) stated: "It might seem entirely reasonable that the energetic processes of the body diminish in intensity as the exogenous food supply is reduced. It is reasonable, in the sense that a wise man reduces his expenditure when his income is cut. Adaptation is defined as " a useful adjustment to altered circumstances". A definition of adaptation has been proposed by the 1985 Food and Agriculture Organization report as "a process by which a new or different steady state is reached in response to a change or difference in the intake of food and nutrients". In this context, the adaptation can be genetic, metabolic, social, or behavioural.

A more quantifiable method is needed for assessing the presence and extent of adaptation. The gap between observed and predicted weight change could be used provided that the prediction is based on the dynamic energy balance equation. A method is to compare the changes predicted from cross-sectional data with those observed in longitudinal studies. If the fall in resting metabolic rate associated with a 20 kg weight-loss is greater than would be expected for that loss of fat-free mass and fat mass then adaptation can be implied. The validity of this method is probably improved by comparing stable states (i.e. measurements made during weight stability before and after weight-loss), by correcting for all important determinants (e.g. correcting metabolic rates for changes in fat-free mass and fat mass), and by appropriate normalization (e.g. a simple division of metabolic rate by fat-free mass ignores the significant intercept between the two and can give erroneous results) (Ravussin & Bogardus, 1989).

2.13.1 Metabolic adaptation to overfeeding

In 1902, Neumann (1902), in his pioneering work on himself, observed that the changes in his body weight and nitrogen balance were lower than predicted in conditions of positive energy balance. He proposed that in response to energy excess, the body can adapt and waste part of the excess energy intake and he termed this mechanism "Luxuskonsumption" (Stunkard & Wadden, 1993). Twenty years later, Gulick (1922) performed a similar long-term experiment on himself. In the late sixties and early seventies, the studies of Miller and Mumford (1967), on the effects of overfeeding in humans upon body weight and the energy cost of exercise, as well as the classical studies of experimental obesity on the Vermont prisoners (Sims, 1976), served to revive the interest in this area. In his 1978 review of overfeeding studies, Garrow (1978) found that in 11 of 16 studies, energy expenditure increased in response to overfeeding, whereas five other studies showed no significant thermogenesis. Garrow (1978) suggested that a possible metabolic adaptation and detectable thermogenesis occur, only when the excess energy intake is over 20 000 kcal.

Most of these pioneering studies have been difficult to interpret since only partial measurements of energy expenditure were performed, and some of the studies were poorly controlled for food intake. In 1980, Norgan and Durnin (1980) extended their previous overfeeding studies (Norgan & Durnin, 1980) and overfed six subjects for 42 days with a total energy excess of 62 000 kcal. Their metabolic rate for standard test tasks increased 10% after overfeeding, but not in relation to total body weight. The discrepancy between the weight gain and energy intake was attributed to probable errors in calculations based on the measured variables, and to the fact that the degree of physical activity was not measured. Ravussin et al. (1985) conducted an overfeeding study for 9 days after a 13-day period of dietary equilibration. Twenty-four-hour energy expenditure was measured at the end of the baseline period and on the second and ninth day of over-feeding. Body weight increased by 3.2 kg, 56% being fat and the remainder fat-free mass. After nine days of overfeeding, only 25% of the energy excess was dissipated as increased energy expenditure (490 kcal/day). One third of this increase in energy expenditure was accounted for by an increase in resting metabolic rate, mostly related to the increase in fat-free mass. Another third

was accounted for by the increase in the thermic effect of food in response to the increased energy intake and the remainder by an increased energy cost of physical activity at the heavier body weight. All of the excess energy expenditure could be accounted for without the need to invoke a "facultative" or "adaptive" component.

The use of the doubly labelled water technique has shed some light on the effect of overfeeding on total energy expenditure (Roberts et al., 1990). Roberts et al. (1990) overfed seven young lean men for three weeks with approximately 1,000 kcal/day excess energy intake. In response to overfeeding, 85 to 90% of the excess energy intake was deposited mostly as body fat, whereas the energy expenditure for physical activity or thermoregulation was unchanged. Only the 24-hour resting metabolic rate increased (RMR + thermic effect of food) and accounted for 15% of the excess metabolizable energy. All the calories were accounted for by increased energy stores and a small increase in energy expenditure. Roberts et al (1990) concluded that regulation of food intake rather than energy expenditure is the primary determinant of energy balance.

Whether obese individuals gain weight more easily than nonobese individuals remain an open question. In his review of the literature, Forbes (1990) tends to imply that, in response to similar excess energy intake, obese individuals gain less weight than thin individuals because they tend to gain a larger proportion of fat. The 100-day overfeeding study of 12 pairs of monozygotic twins has highlighted the importance of genetic background to the weight gain in response to overfeeding (Bouchard et al., 1990). The large interpair variability in weight gain was surprising, as was the small intrapair variability in weight gain. These results point out that human subjects have different genetic susceptibilities to environmental changes, which would lead to variable positive energy balances and variable weight gains. Under free-living conditions, both the genetic susceptibility and the extent to which the environment favours weight gain (such as the availability of fatty foods and labour saving devices) seem to determine the magnitude of weight gain in response spontaneous overfeeding (Stunkard & Wadden, 1993).

2.13.2 Metabolic adaptation to underfeeding

Under the conditions of a large deficit in energy intake, does the body adapt its metabolic rate (over and beyond the predictable effects just described) to minimize the impact of negative energy imbalance on body weight? The concept of metabolic adaptation implies a drop in the metabolic rate that is dependent upon the recent history of negative energy balance and leads to a smaller reduction in body weight than would be predicted from the reduction in energy intake. An appropriate definition of adaptation to a low-calorie diet would be any decrease in energy expenditure that is more than would be expected from the decrease in fat-free mass, fat mass, and energy intake (Stunkard & Wadden, 1993).

Three studies have indirectly assessed 24-hour energy expenditure using accurate energy intake measurements in weight-stable subjects. Liebel and Hirsch (1984) studied 26 subjects in the obese state and in their reduced state. In the obese state, subjects needed 9% more energy per surface area for weight maintenance than controls, whereas, in the reduced state, they required 24% fewer calories while still being 38 kg heavier than the controls. Weigle et al. (1988) found that the energy expenditure in the reduced-obese fell below the regression line between 24-hour energy expenditure and fat free mass, which was established in their baseline, obese state. This was related mostly to the predictable decreased energy cost of physical activity. Studies that have used indirect calorimetry in the steady obese state and non-steady weight-losing state have found a fall in 24-hour energy expenditure (Apfelbaum et al., 1971; De Groot et al., 1990; Bessard et al., 1983). These studies did not all agree on the presence or the absence of adaptation to hypocaloric diets.

Ravussin and Swinburn (1992) reviewed the impact of hypocaloric diets on total energy expenditure and its major components. There is general agreement among the studies that weight-loss in obese subjects on hypocaloric diets causes a fall in 24-hour energy expenditure corrected for fat-free mass, the major but not the sole determinant of energy expenditure. This fall is mainly related to the non-resting metabolic rate. Possible adaptive changes to weight-loss include a decrease in activity levels with weight-loss, a decrease in the thermic effect of food (beyond that expected from the reduction in calories) and, under certain circumstances, a decrease in resting metabolic

rate (beyond that expected for the decreased body size). Quantitative estimates of the contribution of these adaptive changes to the overall lower metabolic rate are clearly not possible, but based on the review of the data, it seems that in obese individuals the adaptive changes are minor compared with the predictable changes (Ravussin & Swinburn, 1992). Low-calorie diets in lean subjects seem to evoke significant adaptive changes in resting metabolic rate and probably in the energy cost of physical activity (Keys et al., 1950; Ravussin & Swinburn, 1992). This response is not surprising, since the threat that a hypocaloric diet poses to survival is greater in leaner subjects.

2.14 REGIONAL FAT DISTRIBUTION

The concept of fat distribution (android and gynoid) was first introduced in obese subjects with diabetes mellitus or cardiovascular disease by Vague (1947). Later, Kiessebah & Krakower (1994) proposed a classification of obesity: upper body segment and lower body segment using waist-to-hip circumference ratio.

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)

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 2.17: PATTERNS OF FAT DISTRIBUTION (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 (android 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 glueteo-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 (LDL's). 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).

2.15 PREVENTION OF OVERWEIGHT AND OBESITY

Prevention of overweight and obesity is as important as treatment. The prevention of obesity is a topic that must be considered given the major increases both in the prevalence of obesity and in the mean body weights of people worldwide over the past decade. Despite the appeal of prevention as an ideal, it appears that the world has been unable to prevent obesity. These facts led a recent review to conclude that "we have not been able to prevent obesity in the past and we do not have the tools to do better in the future" (Stunkard, 1995).

It has been proposed that genetic vulnerability may lie at the root of the epidemic of obesity and the problem of controlling, let alone preventing, obesity (Bouchard, 1994). There has been no real change in the gene pool during this period of increasing obesity. The root of the problem must lie in the powerful social and cultural forces that promote an energy-rich diet and a sedentary lifestyle. If social and cultural forces can promote obesity, these same forces should be able to control it. Therein lies the still unrealised potential for preventing obesity (Thomas, 1995).

There is some ambiguity of terminology in the prevention literature. The verb prevent implies taking an action or interposing an impediment to stop or keep something from happening. Different ideas about what it is that should be stopped or kept from happening have been suggested in terms of obesity prevention. Is it the incidence of obesity itself? Is it preventing weight gain among those treated for obesity to prevent progression from a moderate to more severe levels? Does the success of prevention efforts depend upon the effect of comorbid medical disabilities? Is what should be stopped or kept from happening an underlying risk condition or predisposition factor for obesity development (Thomas, 1995)?

An Institute of Medicine (IOM) report recommends an approach to clarifying definitions of prevention that, although developed in relation to mental disorders, apply to obesity (IOM, 1994). This IOM report reviews existing classification systems for preventive interventions for physical illness. The familiar public health classification system designates three types of prevention: primary, secondary and tertiary. The goal of primary prevention is to decrease the number of new cases (incidence) of a disorder. In secondary prevention, the goal is to lower the rate of established cases of the disorder in the population (prevalence). Tertiary prevention seeks to stabilize or decrease the amount of disability associated with an existing disorder. For obesity, tertiary prevention could refer to decreasing the progression to more severe obesity or decreasing the likelihood of associated musculoskeletal, metabolic, or vascular disorders (Thomas, 1995; National Task Force on Prevention and Treatment of Obesity, 1994).

When this prevention classification system was introduced more than 25 years ago, the implicit disease model was one of an acute condition with a specific unifactorial cause. It was assumed that mechanisms linking the cause of a specific disease to its subsequent occurrence could be identified. In the intervening years, many chronic diseases prevalent have been recognised as having multi-factorial etiologies. Research on these diseases has advanced our knowledge about the complicated relations that exist between risk factors and protective factors for disease and the outcomes of preventive interventions. This knowledge can breed the pessimistic view that prevention efforts will be futile until the etiologies of diseases are better understood (IOM, 1994).

According to this analysis, the concept of risk reduction is critical to prevention programs and research. Addressing the degrees of risk for a condition supplants the more simplistic concept of prevention in which a disease is simply present or absent. Risk factors refer to those characteristics that, if present for a particular individual, make it more likely that this person (compared to someone selected from the general population) will develop a disorder (Werner & Smith, 1992). Both risk and protective factors are included here. Research also shows that many at-risk individuals have factors in their background or environment that protect against the development of a disorder (Garmezy, 1983). In furthering the establishment of successful preventive intervention programmes, the IOM (1994) report recommends instituting a "preventive intervention research cycle". This research cycle consists of, first, description of the interplay between risk and protective factors, next, identification of causal risk factors that may be alterable through interventions, finally, systematic, empirical, and rigorous testing of these interventions, most often in preventive intervention trials. At the current stage of research into preventing obesity, work is still in the first two phases of this research cycle: identifying high-risk and protective factors for the development of obesity, and determining which factors are malleable and can be altered by preventive interventions (Thomas, 1995).

The IOM (1994) report also recommended an alternative terminology for physical disease prevention, proposed by Gordon (1987). This terminology identifies three types of prevention: universal, selective, and indicated prevention. Each category represents a population group, rather than a disorder or disease state, to whom preventive interventions are directed. Universal preventive measures or interventions are designed for everyone in the eligible population. Selective preventive measures are directed toward a subgroup of the population whose risk of developing the disorder is above average or high. Indicated preventive interventions are targeted to high-risk individuals identified as having minimal but detectable signs or symptoms that foreshadow the disorder, or exhibiting biological markers indicating predisposition, who do not meet the full diagnostic criteria for the disorder itself. The earlier IOM (1992) report reserves the term prevention for those interventions that occur before the onset of a diagnosed disorder. What was previously known as tertiary prevention is redefined as maintenance intervention, whose aim is to reduce

the disability associated with an ongoing disorder. Maintenance interventions, which can be supportive, educational, and/or pharmacological, are provided on a long-term basis to reduce relapse and recurrence.

The primary aim of obesity prevention is to reduce the number of new cases of obesity. This can be accomplished by means of a risk-reduction model. Even if the obesity outcomes are in the distant future, the decrease in risk factors and increase in protective factors for obesity can be identified. An important secondary aim is to delay the onset of obesity. The goals of indicated prevention programs are harder to define than those of universal and selective prevention programs. They might be framed in terms of reducing the length of time initial weight gain persists beyond certain pre-obese limits and halting its progression before diagnostic criteria for obesity are met. Even if the individual does eventually develop obesity, the prior preventive intervention may still have had an effect by reducing the duration or severity of the disorder (Thomas, 1995).

Many approaches to prevent obesity appear promising, though few studies are available to document long-term positive outcomes. Since success in prevention programs is often equated with the absence of future problems, the impact of universal prevention programs that target education and behaviour change (e.g. in diet or exercise patterns) is difficult to evaluate except through longitudinal population studies (IOM, 1994). The recent literature in prevention has focused more on working with groups or individuals who are known to be at risk for a particular disorder. The emphasis on working with high-risk individuals with interventions that are matched or targeted to specific risk factors (as in selective and indicated prevention strategies) appears to have considerable merit. Only a few studies of this type have been conducted in obesity prevention. Future research on the development of prevention programs targeted to those at high risk for obesity is necessary before any conclusions can be drawn concerning this promising new approach (Thomas, 1995).

2.15.1 Additional research needs in obesity prevention

- Studies are needed to assess the usefulness of medication(s), including combination of drugs, in the prevention of both further weight gain and regain

of weight following treatment. Long-term use of medication, refuting the belief that tolerance develops to its effects, clear the way for more aggressive use of this therapy (Thomas, 1995).

- Studies should be designed to assess the applicability of a "harm - reduction" approach to prevention. Developed originally as an approach to substance abuse prevention and treatment, harm reduction is based on a continuum model, with incremental changes encouraged (e.g. encouragement of proximal goals of gradual weight-loss over time in contrast to traditional programs with their emphasis on distal goals of ideal weight-loss) (Marlatt & Tapert, 1993; Marlatt et al., 1993). The harm-reduction model is consistent with a stepped-care approach that begins with the least intensive intervention before proceeding to more intensive and expensive steps (Thomas, 1995).

- Studies are needed that compare the impact of prevention programs designed for specific weight-loss goals with others designed to (1) affect personal beliefs in ideal body image; (2) decrease reliance on exaggerated dietary behaviours; and (3) treat associated eating disorders (e.g., binge or purge cycling) (Thomas, 1995).

- Studies should be conducted to investigate the effects of prevention programs representing either (1) individual clinic-based intervention programs designed to modify personal behaviour; (2) community-based public health programs, including public policy and regulatory policy change; or (3) a combination of both clinical and public health approaches (Thomas, 1995).

- Research on unaided weight management is necessary, for example, natural-history studies of formerly obese people who learned to manage their weight successfully by themselves and investigations of people who are at risk for obesity but who nonetheless maintain healthy weights (Thomas, 1995).

2.16 OBESITY TREATMENT STRATEGIES

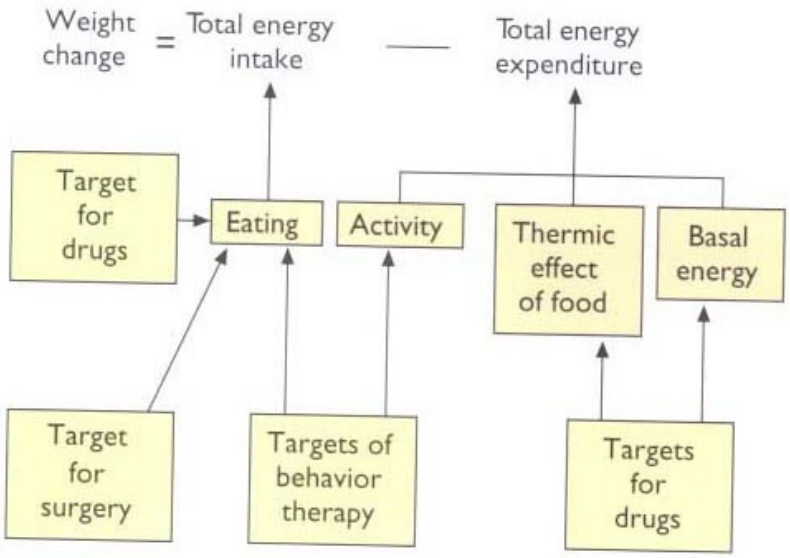


FIGURE 2.18: ALGORITHMIC APPROACH FOR theropy selection (BRAY, 2003)

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 suppression, "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).

2.16.1 Pharmacotherapy

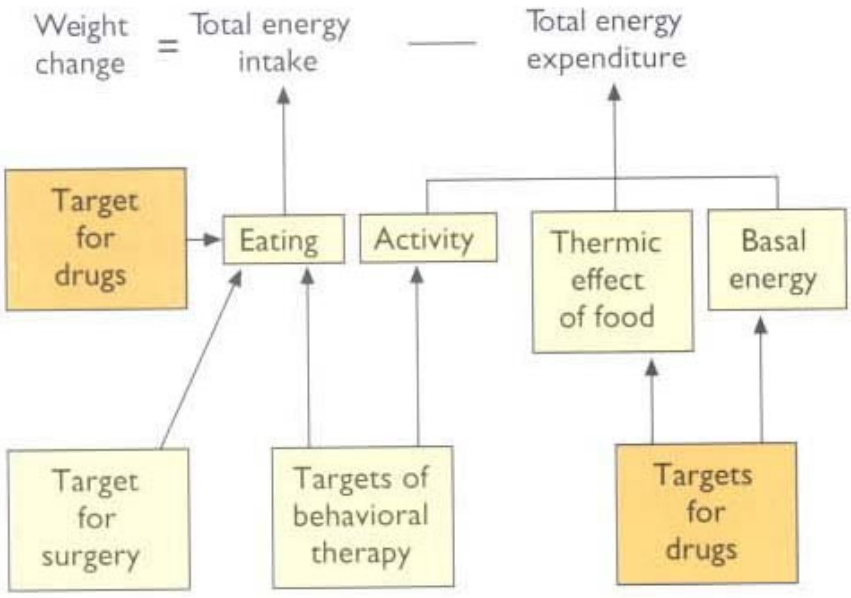


FIGURE 2.19: THE FIRST LAW OF THERMODYNAMICS CAN BE USED TO IDENTIFY THE PLACE WHERE DRUG TREATMENT CAN BE EFFECTIVE (BRAY, 2003)

Like hypertension or diabetes mellitus, obesity is a chronic disease that requires continued treatment. Short-term (weeks or months) treatment with medication is not warranted nor is it probably appropriate. Treatment with medication is likely to be necessary for years, and perhaps for a lifetime in order to sustain weight-loss and improve health. To date, there have been few published studies where patients have received anorexiants for more than one year. In addition, data on the long-term safety and efficacy of anorectic medication combinations is also very limited. The lack of long-term safety and efficacy data is disappointing, given that most of these anorexiants have been available for more than 20 years. (Oeser, 1997). The National Task Force on the Prevention and Treatment of Obesity (1996) does not recommend pharmacotherapy for the routine treatment of obesity.

Although routine use is not justified, some carefully selected patients may benefit from pharmacotherapy. Most authorities recommend a BMI of 27 as the minimum indication for medication therapy in the absence of obesity-related co-morbidities who

have failed diet, exercise, and behaviour modification (National Task Force on the Preventing and Treatment of Obesity, 1996). If medication therapy is warranted, it should only be used in conjunction with a comprehensive program that includes nutrition education, exercise if appropriate, and behaviour modification. Therapy should only be continued if the patient has an initial response (National Task Force on the Preventing and Treatment of Obesity, 1996). Unfortunately, it is not yet possible to predict who will respond to pharmacotherapy. There is some evidence that clinically significant weight-loss within the first several weeks of treatment with a given medication is predictive of further responsiveness to the same medication. An initial trial period of several weeks with a medication or medication combination may help determine their efficacy. If the patient fails to respond to a given medication or combination of medication with a reasonable degree of weight-loss such as 0.45 kg per week after a 4-week trial period, therapy should be reassessed. Reassessment should include an evaluation of compliance to the medication regimen, as well as any adjunctive therapies, and assessment of the need for a dosage adjustment. If the patient continues to be unresponsive, the medication should be discontinued.

A wide variety of pharmacological substances have been used for the treatment of obesity. Current pharmacotherapy for obesity includes medication that decrease caloric intake by suppressing appetite and thermogenic medication that increase metabolism. More recent approaches are to involve medication that block gastrointestinal absorption of nutrients, as well as more specific agents that alter the neurochemical or hormonal signals that affect fat stores, food intake and energy expenditure. Obesity may also lead itself to novel therapeutic approaches involving gene therapy. Studies evaluating the effect of medication for the treatment of obesity invariably use weight as the chief endpoint and have not included major morbidity endpoints or mortality.

2.16.1.1 History of pharmacotherapy

The fascinating and sometimes disturbing history of medication therapy for obesity dates back to 1893 with the introduction of the thyroid hormone (Bray, 1993). Thyroid extract was believed to be therapeutic because obese persons were thought to have a low metabolic rate. This treatment was in vogue well into the 1970's but was

abandoned because of the risk for electrolyte disturbances, metabolic imbalances, and cardiac dysrhythmias. Excessive thyroid hormone supplementation increased bone resorption leading to decreased bone density and osteoporosis. In addition, the hormone may have a detrimental effect on lean body mass. Thyroid hormone preparations are therefore not indicated for the treatment of obesity unless the obese patient is hypothyroid.

Dinitrophenol (2,4-DNP) was introduced in 1933 for the treatment of obesity and soon found its way into numerous "anti-fat patent medicines" (Tainter et al., 1933). The use of these compounds was abandoned in 1937 because of reports of severe intoxications and deaths. Dinitrophenol is used currently as a wood preservative and insecticide.

The use of cardiac glycosides, alone or as an ingredient in bizarre weight-loss concoctions was popular during the first half of this century. Digitalis pushed to the point of toxicity to induce nausea and therefore anorexia, was advocated in 1940 and was in vogue until the late 1960's when reports of cardiac dysrhythmias surfaced. (Kattus et al., 1968). Digitalis was typically added to weight control regimens for its anorexic action and to counteract the tachycardia produced by the supraphysiologic doses of thyroid hormone. The cardiac glycoside strophanthin was actually combined with thyroxin in a commercial product known by the brand name of Neo-Barine, which was advertised as a "safe, dependable anti-adipoexic Weight-reducing agent" (Neo-Barine, 1964). Neo-Barine was commonly used for weight reduction but was cardiotoxic, prompting the June 19, 1964 Medical Letter on Drugs and Therapeutics to brand it as a dangerous drug and recommend its removal from the market (Neo-Barine, 1964).

A 1969 report in the Journal of the American Medical Association highlighted the problems with polypharmacy in the treatment of obesity. The report describes a 19-year-old male who died from a weight reduction program that included the combination of thyroid extract, digitalis, amphetamines, and diuretics (Jelliffe et al., 1969).

In the 1970's, ephedrine-containing products used in Europe for the treatment of asthma were noted to cause anorexia and weight-loss in asthmatic patients. In 1972 a Danish physician by the name of Dr Eriksen observed loss of appetite and weight in asthmatic patients for whom he had prescribed a mixture containing ephedrine, caffeine, and phenobarbital. Rumour spread and when sales culminated in 1977, more than 70 000 Danes were taking what become known as the "Elsinore pill". One provincial pharmacy alone was manufacturing a million tablets weekly (Malchow-Moller et al., 1981).

Another asthma product containing ephedrine known as the Do-Do pill, which was available over the counter in England, was studied (Miller, 1985). These observations and further research revealed how methylxanthines (caffeine and theophyllini) potentiate the thermogenic effect of ephedrine – which stimulated interest in the combination (Dullo & Miller, 1985).

Dextroamphetamine was observed to cause weight-loss after it was introduced in the 1930's for the treatment of narcolepsy (Bray, 1993). The amphetamines were subsequently shown to induce weight-loss by suppressing appetite but enthusiasm faded as the potential for abuse became apparent. Problems with amphetamine abuse dampened enthusiasm for medication therapy resulting in a 23 year hiatus from 1973 when the FDA approved fenfluramine to 1996 when dexfenfluramine was approved. The introduction of the fenfluramines, medication that resemble the amphetamines chemically but not pharmacologically, vastly increased the understanding of the role of serotonin (5-hydroxy-tryptamine) in food intake and ushered in the current era of pharmacotherapy of obesity (Oeser, 1997).

2.16.1.2 Herbal preparations

Although many useful medicines have been derived from herbs and other plants, claims of therapeutic benefit, especially with regard to obesity, often overstate the existing scientific evidence (Oeser, 1997). Kelp in the form of powder or tablets is often advocated for the control of obesity because the iodine in kelp is thought to stimulate thyroid hormone production. Such stimulation would only occur in persons with iodine deficiency and iodine deficiency is very rare in this age of iodised salt

(Tyler, 1993). Many herbal products contains multiple ingredients and are promoted based on anecdotal claims of weight-loss. Reports of serious adverse effects associated with the use of herbal remedies for weight-loss are increasingly common (Larrey et al., 1992; Capwell, 1995; Nadir et al., 1996).

Because these products are considered "natural" and typically marketed as food supplements, consumers may assume incorrectly that they are safe and without side effects. Interpretation of reports of adverse effects is fraught with difficulty because herbal preparations are occasionally mislabelled or, in some cases adulterated, and contain ingredients or quantities not listed in the labelling (Huxtable, 1992). The Texas Department of Health, during an investigation of adverse events related to ephedrine-containing products, purchased a product labelled "no side effects" that listed wild Chinese ginseng as the only ingredient. Upon laboratory analysis, a single tablet was found to contain 45 mg of ephedrine and 20 mg of caffeine. The label on the product recommended users take five tablets, representing a total ephedrine dosage of about 11 times the usual recommended dosage in bronchodilators, which contains 12.5 to 24 mg ephedrine per dose (Capwell, 1995).

Ma-huang is an herbal product derived from plants of the Ephedra species that has been used in China for thousands of years and is promoted as a weight-loss aid (Tyler, 1993). The herb is sold as a dietary aid and is therefore exempt from the rigorous testing for safety and efficacy that drugs must undergo. The active constituent is ephedrine. Although ephedrine is capable of inducing modest weight-loss by virtue of its anorectic and thermogenic properties its use is associated with substantial risk for serious side effects including liver injury and psychiatric disturbances (Capwell, 1995; Nadir et al., 1996).

The blossoms of wall germander (Teucrium chamaedrys) have long been used in folk medicine for obesity. Preparations include herbal teas, a medicinal liquor of germander admixed with other herbs, and capsules containing powdered germander alone or mixed with green tea. Although the herb was assumed to be harmless, hepatotoxicity has been reported (Larrey et al., 1992).

The aforementioned reports underscore the fact that just because a product derives from a natural source does not necessarily mean it is safe. It is likely that many natural products possessing clinically useful anoresciant activity await discovery. Data regarding safety and efficacy for many naturopathic remedies is lacking (Oeser, 1997).

2.16.1.3 Thermogenic agents

Ephedrine, the active constituent of various Ephedra plant species, is an alkaloid that was originally isolated by the Japanese chemist N Nagai in 1887 (Oeser, 1997). Ephedrine has long been used as a nasal decongestant, and as a treatment for asthma (Tyler, 1993). Interest in the anorectic properties stemmed from the observation in the 1970's that asthmatics tended to lose their appetite and weight when treated with ephedrine-containing products (Malchow-Moller et al., 1981).

The way in which the combination of ephedrine and caffeine induce weight-loss is not completely understood. Because of its noradrenergic properties, ephedrine is considered to suppress food intake via noradrenergic pathways in the hypothalamus and related areas. Ephedrine also appears to possess a thermogenic effect and therefore increases energy expenditure (Oeser, 1997). The addition of a methylxanthine, such as caffeine, appears to potentate both the anorectic and thermogenic effects of ephedrine (Astrup et al., 1992). The degree of weight-loss attributed to the combination of ephedrine and caffeine is moderate and, when used as an adjunct to caloric restriction, has been reported to average 3.4 kg over six months. Although the degree of weight-loss attributable to ephedrine and caffeine has been reported to be similar to that induced by dexfenfluramine, side effects are common with the ephedrine/caffeine combination (Breum et al., 1994). The combination of ephedrine and caffeine increases both systolic and diastolic blood pressure. It may also increase heart rate and may cause palpitations as well as nervousness, headache, insomnia and dizziness. Its use in persons suffering from heart conditions, hypertension, and diabetes is therefore not recommended (Oeser, 1997).

Dietary supplements containing ephedrine and associated alkaloids (pseudo-ephedrine, norephedrine, N-methyl ephedrine) have been implicated in numerous adverse events

and appear to pose a significant health risk. Many of these products are advertised as "natural" or promoted as food supplements, consumers may assume incorrectly that they are safe and devoid of side effects. Some of these products appear to have been purposely misbranded and even adulterated and contain inappropriately high dosages of ephedrine (Oeser, 1997). Adverse effects from ephedrine may be variable and are not always dose-related (Astrup et al., 1995). Serious adverse effects from ephedrine, such as acute cardiovascular and central nervous system stimulation, can occur even with low doses. Side effects associated with ephedrine include palpitations, tachycardia, hypertension, coronary artery vasospasm, psychosis, convulsions, respiratory depression, coma and death. The combination of ephedrine with caffeine or phenylpropanolamine has also been associated with hemorrhage stroke, seizures, mania and psychosis (Oeser, 1997).

Beta 3-adrenergic receptors, which are found in brown adipose tissue, appear to be involved in promoting lipolysis and heat generation in fat. Activation of these receptors increases lipolysis and thermogensis and thereby increases energy expenditure (Levin & Routh, 1996). Several experimental compounds with beta-3-stimulating properties have been shown to possess thermogenic activity in humans and to induce weight-loss when given with a caloric restricted diet (Astrup et al., 1992). Beta-adrenoceptor agonist (BRL 26830A) has shown to stimulate thermogenesis in the brown adipose tissue and skeletal muscle of rats and increases the resting metabolic rate in normal human volunteers (Connacher et al., 1992). Two clinical studies reported improved weight-loss compared to placebo when used in conjunction with caloric restriction (Zed et al., 1985; Connacher et al., 1988).

Another study failed to detect any difference with the use of BRL 26830A, but this was attributed to poor dietary compliance and the refractory nature of the patient population (Chapman et al., 1988). Tachycardia, tremor and shaky hands have been the only routinely reported side effects which suggests some activity at beta-3 receptors (Astrup et al., 1992; Connacher et al., 1992).

2.16.1.4 Lipase inhibitors

Caloric intake can be modulated by decreasing the absorption of nutrients in the gastrointestinal tract. Orlistat (marketed under the name Xenical by Hoffman-La Roche) a synthetic derivative of lipostatin, which is the natural product of streptomyces toxytricini, an inhibitor of intestinal lipases (Hadvary et al., 1988). Lipase is an enzyme that breaks down fat so that it can be absorbed in the gastrointestinal tract. Inhibition of lipase causes some of the fat that is eaten to pass undigested through the body.

Fifty milligrams of Orlistat three times daily produced significantly greater weight-loss (3.4 kg) than placebo (2.8 kg) at 12 weeks in a preliminary study involving 39 healthy obese persons. Treatment with Orlistat was associated with more gastrointestinal side effects including abdominal pain, fecal incontinence, liquid or oily stools, nausea and vomiting. Orlistat also decreased blood levels of vitamin E in some subjects (Drent & Van der Veen, 1993). In a larger study, comparing 30, 180, and 360 mg/day of Orlistat with placebo, only the 360 mg dose of Orlistat produced significantly greater weight-loss at 12 weeks. Gastrointestinal side effects were reported frequently and about twice as many subjects receiving Orlistat dropped out of the study compared to those taking the placebo. A decrease in blood levels for vitamins A, E and D was observed in the subjects receiving Orlistat, but was not considered clinically significant by the investigators (Drent et al., 1995).

2.16.1.5 Noradrenergic agents

The extreme central nervous system excitation caused by the amphetamines led to the development of noradrenergic or norepinephrine-like agents which promote weight-loss by suppressing appetite and may also increase energy expenditure. They act by promoting the release of norepinephrine and dopamine from nerve terminals in the central nervous system and by blocking the subsequent reuptake (Samanin & Garattini, 1993).

Phenteramine, phendimetrazine, phenmetrazine, diethylpropion, benzphetamine, and mazindol are the prescription nor-adrenergics currently available. Phenteramine

provides the noradrenergic activity when combined with fenfluramine in the combination popularly referred to as "Fen-Phen". Amphetamines are rarely prescribed for the treatment of obesity because of the potential for abuse and because safer alternatives are available. The prescribing of amphetamines for the treatment of obesity is severely restricted by medical and pharmacy laws (National Task Force on the Prevention and Treatment of Obesity, 1996).

In most randomised controlled studies, ranging from 6 to 64 weeks in duration, the noradrenergic anorexiants promoted significantly greater weight-loss than placebo. The rate of weight-loss attributed to these drugs averages 0.32 kg per week. Weight-loss continues as long as the drugs are taken but tends to be regained when treatment stops (Bray, 1993). Common side-effects associated with this class of drugs include heart palpitations, tachycardia, nervousness, restlessness, insomnia, anxiety, tremors and dry mouth.

2.16.1.6 Serotonergic agents

The two most commonly prescribed serotonergic anorexiants, fenfluramine (Pondimin) and dexfenfluramine antidepressants, such as Prozac, that selectively inhibit the reuptake of serotonin have also been studied. Fenfluramine and dexfenfluramine induce the release and inhibit the reuptake of serotonin (5 hydroxytryptamine, 5-HT) in the brain (Mc Tavish & Heel, 1992).

Elevated brain serotonin levels are associated with early satiety and appetite suppression (Samanin & Garrattini, 1993). Although weight-loss from serotonergic agents has traditionally been ascribed to their anorectic effects, there is evidence suggesting that these drugs are also capable of increasing energy expenditure (Bross & Hoffer, 1995). Fenfluramine is a racemate of the dextro- and levo-rotating isomers of flenfluramine and is commonly used with phenteramine in the "Fen-Phen" combination.

There have been several well-controlled studies of single drug therapy (Goldstein et al., 1993). Only one long-term controlled study of combination therapy has been published. In studies conducted for six months or longer, both single-drug and

combination therapy induced a net weight-loss ranging from 2 to 10 kg (Weintraub et al., 1992).

The rationale for the aforementioned combination of fenfluramine and phenteramine is that, by combining drugs with different mechanisms of action, a lower dose of each drug can be used thereby maintaining efficacy and minimizing toxicity. Weintraub and co-workers showed that the combination of low doses of fenfluramine and phenteramine resulted in weight-loss equal to that achieved with either agent alone (Weintraub et al., 1984). Patients who took the combination reported fewer cardiovascular and central nervous system side effects than patients who took phenteramine alone. This landmark study is widely cited as justification for the routine use of "Fen-Phen" and is partly responsible for the recent resurgence in the use of diet pills for weight-loss (Oeser, 1997). The combined treatment with fenfluramine and phenteramine produced a modest degree of weight-loss above and beyond that resulting from behaviour modification alone, and some effects were sustained for more than three years in the few patients who continued to receive active drug therapy. Weight-loss tended to reach a plateau after about six months, and some weight regain occurred between years two and three despite continued treatment. In most studies weight begins returning to baseline when drug therapy is stopped (Weintraub, 1992).

Adverse effects associated with dexfenfluramine include tiredness, drowsiness, diarrhea, polyuria and dry mouth. Dry mouth is also a common complaint of patients taking the combination of fenfluramine and phenteramine. The abuse of fenfluramine or dexfenfluramine appears to be rare, although some cases have been reported with fenfluramine (Dare & Goldney, 1976). These agents should be used with caution in patients with a history of substance abuse. Both fenfluramine and dexfenfluramine are associated with a discontinuation phenomenon, therefore it may be best to gradually taper the patients off these agents over several days when discontinuing therapy (Steel & Briggs, 1972).

The National Task Force on the Prevention and Treatment of Obesity (1996), recently stated that "evidence of neurotoxin effects in humans has not been reported with fenfluramine or dexfenfluramine" but went on to recommend further studies

"evaluating the possibility of subtle neuropsychological changes, particularly with prolonged administration".

The recommended dosage of dexfenfluramine is 15 mg twice daily with meals. Patients should be cautioned to avoid beverages containing alcohol (since alcohol may exacerbate drowsiness) and warned to report any symptoms of pulmonary hypertension dyspnea, edema, angina, or syncope (Redux prescribing information, 1996). The recommended dose of fenfluramine is 30 mg twice daily (Pondimin prescribing information, 1994).

2.16.1.7 Selective serotonin reuptake inhibitors

Fluoxetine (Prozac) inhibits the reuptake and increases the availability of serotonin in the central nervous system. The mechanism by which the drug induces weight-loss is not known but serotonin is likely involved in the regulation of satiety (Abramowicz, 1994).

During studies evaluating the antidepressant efficacy of fluoxetine, it was noted that patients tended to lose small amounts of weight. Based on this observation some of the largest and best-designed studies of drug therapy for the treatment of obsity were conducted (Oeser, 1997). The greatest treatment effect with fluoxetine was observed in the centres that provided behavioural or nutritional counselling (Goldstein et al., 1994). A pattern of initial weight-loss followed by weight regain despite continued treatment has been reported in studies (Goldstein et al., 1993).

Side-effects associated with high-dose fluoxetine therapy include asthenia (weakness), somnolence, insomnia, nausea, diarrhea, sweating, nervousness, tremor, dyspepsia, sexual dysfunction, particularly delayed or absent orgasm. Some patients treated with fluoxetine for depression have had an increase in appetite and some have gained weight. Fluoxetine can help some patients lose weight for 5 or 6 months, but its continued effectiveness remains to be established, troublesome side-effects can occur, and it is very expensive at the doses required (Abramowicz, 1994).

2.16.1.8 Other agents

- **Sibutramine**

  Sibutramine is a promising new agent that boosts brain levels of both norepinephrine and serotonin by blocking their reuptake (Ryan et al., 1995). In a 12 week study doses ranging from 10 to 30 mg daily were shown to produce significantly greater weight-loss than placebo in a recent 24-week double-blind trial (Bray et al., 1996). Weintraub and colleagues compared two different daily doses of sibutramine with placebo and showed a dose-related effect on weight-loss (Weintraub et al., 1991). Patients who took 20 mg daily lost an average of 5.0 kg, compared to 2.9 kg with 5 mg daily, and 1.4 kg with placebo. Side effects associated with sibutramine treatment included difficulty sleeping, irritability, unusual impatience, and excitation.

- **Nultrexone**

  Endogenous opioids (synthetic narcotic that resemble opiates in action but are not derived from opium) have been postulated to be involved in the control of food intake (De Szaan & Mitchell, 1992).

- **Pectin**

  Pectin is a purified carbohydrate product obtained from the dilute acid extract of the inner portion of the rind of citrus fruits or apple pomace and consists mainly of partially methoxylated polygabacturonic acids. Pectin is an absorbent and bulk-forming agent often present in multi-ingredient preparations for the management of diarrhea, constipation, and obesity. Pectin has also been used to decrease the rate of carbohydrate absorption in the dumping syndrome (Martindale, 1997).

  The results of a preliminary study indicate that pectin may serve as a useful adjuvant in the treatment of obesity by virtue of its ability to induce satiety via delayed gastric emptying. Fifteen grams of pectin administered with a meal slowed gastric emptying, induced satiety and prolonged the time to the next meal in nine obese subjects. Additional research is needed

to define the effect of pectin on weight-loss and to determine the adverse effect profile of long-term pectin ingestion (Di Lorenzo et al., 1988).

- **Bromocriptine**

  Bromocriptine is a dopamine agonist that has been shown to decrease body fat stores in experimental animals with little or no reduction in body weight or food intake (Cincotta & Meier, 1989). Meier and colleagues treated 33 obese postmenopausal woman and 15 men with poorly controlled Type II diabetes mellitus with bromocriptine. The subjects received 1.25 mg or 2.5 mg daily for 10 weeks with no other interventions. Body fat, as estimated by skin fold measurement, reduced by 11.7% in the non-diabetic subjects. Nausea was the only side-effect reported (Meier et al., 1992). Treatment with bromocriptine was also associated with a greater reduction in body fat and improved glucose tolerance (Cincotta & Meier, 1996).

  The manner in which bromocriptine decreases fat stores and body weight and improves glucose tolerance is not known, but appetite suppression or a reduction of lipogenesis has been proposed (Meier et al., 1992; Cincotta & Meier, 1996). Although these data are promising, the use of bromocriptine cannot be recommended until more information about long-term safety and efficacy is available.

- **Human chorionic gonadotropin (hcG)**

  Injections of hcG have been tried as an adjunct for weight-loss. Touted benefits include weight-loss beyond that resulting from dieting, a more attractive or normal distribution of fat, decreased hunger and discomfort associated with dieting. Although an early study suggested a benefit (Asher & Harper, 1973), subsequent randomised and controlled studies have demonstrated absolutely no benefit from hcG (Young et al., 1976). The effectiveness of hcG-diet programs has been attributed to the caloric restriction, frequent physician contact, and the placebo effect (Bennett, 1995).

- **Dehydroepiandrosterone (DHEA)**

  Several steroidal drugs have been evaluated. Dehydroepiandrosterone (DHEA), a naturally occurring steroid precursor of both androgens and estrogens is being sold at health food stores and some pharmacies as a "food supplement". It is being promoted to offset the effects of aging even though it is not approved for any indication by the US Food and Drug Administration (Abramowicz, 1996). DHEA has been reported to have beneficial effects on obesity, diabetes mellitus, and serum lipids in animals. A randomised controlled study has shown no benefit in obese humans (Vogiatzi et al., 1996).

- **Chromium picolinate**

  Chromium picolinate is the latest rage and is sold in health food outlets and pharmacies as an ingredient in "fat burner" products. Chromium is an essential nutrient that appears to serve as a cofactor with insulin in the maintenance of normal metabolism. Chromium deficiency can lead to insulin resistance and dysfunctions in carbohydrate, protein, and fat metabolism (Mertz, 1993).

  Subsequent studies have failed to demonstrate a significant effect from chromium picolinate on body fat (Hasten et al., 1992; Clancy et al., 1994). In the most recent of these, 95 active duty obese navy personnel were randomly assigned to receive 400 micrograms of chromium picolinate or placebo. This very well-controlled study found absolutely no difference in the magnitude of weight-loss, percent body fat, or lean body mass between those who took chromium picolinate and those who took placebo after 16 weeks of treatment (Clancy et al., 1994).

- **Pyruvate and dihydroxyacetone**

  Pyruvate is another supplement that has recently gained attention. Marketers of nutritional supplements are promoting the product as a way to burn fat without reducing muscle mass. Studies in experimental animals suggest that the combination of pyruvate and dihydroxyacetone may prevent fat accumulation, without a deleterious effect on body protein stores (Stanko & Adibi, 1986; Cortex et al., 1991).

In a series of studies involving a small number of obese women, pyruvate with or without dihydroxyacetone appeared to enhance body fat and weight-loss during caloric restriction and to inhibit weight regain after weight-loss (Stanko et al., 1992). Subjects receiving the combination of pyruvate and dihydroxyacetone during severe caloric restriction lost an average of 1 kg (body weight) more than those receiving placebo during severe caloric restriction (Stanko et al., 1992). Subjects, who received pyruvate alone during moderate caloric restriction, lost an average of 1.6 kg more than subjects taking placebo during caloric restriction (Stanko et al., 1992). In obese women who had lost weight during caloric restriction, the combination (15 grams of pyruvate and 75 grams of dihydroxyacetone per day as a portion of daily carbohydrate energy intake) appeared to inhibit regain of body weight and the reaccumulation of body fat during subsequent refeeding with a high caloric diet (Stanko & Arch, 1996).

Side-effects attributable to pyruvate and dihydroxyacetone supplementation included diarrhea and borlorygmus (rumbling in the bowels produced by gas). The mechanism by which pyruvate and dihydroxyacetone induce these changes in body composition has not been established, but may involve increased energy expenditure or promotion of fat oxidation. The effects of simply adding these 3-carbon compounds to the diet like a vitamin supplement are not known. These studies are encouraging, but further research is needed to determine the effect of these supplements on long-term weight-loss and to define fully the adverse effect profile.

- **Growth hormone (Somatotropin)**

  Growth hormone is secreted by the anterior pituitary and, in addition to promoting growth in children, possesses anabolic, lipolytic and diabetogenic actions. Growth hormone also affects energy balance by stimulating energy expenditure. The shrinkage of lean body mass and the expansion of adipose tissue mass associated with aging appears to be related to a decline in the production of growth hormone. Growth hormone secretion is also suppressed in obese persons. The widespread availability of synthetic human growth

hormone has allowed the evaluation of the effects of the hormone in a variety of conditions including obesity.

In an effort to avert the loss of lean body mass that accompanies weight reduction programs for obesity, growth hormone appears to reduce body fat and preserve lean body mass (Drent et al., 1995). In 12 obese women, randomly assigned to receive growth hormone (0.08 mg/kg intramuscularly three times weekly) or placebo, treatment with growth hormone was associated with a reduction in body fat and increased lean body mass (Skaggs & Crist, 1991).

These results are promising, but growth hormone excess is exemplified by acromegaly and adverse carbohydrate metabolism (inducing hyperinsulinemia, glucose intolerance, and diabetes mellitus), it also affects the musculoskeletal system (causing arthritis and arthralgia), and the cardiovascular system (producing hypertension, edema, and congestive heart failure) (Vance, 1990). Longer-term studies are needed to define fully the beneficial and toxic effects of growth hormone before treatment of the obese person can be recommended.

- **Anabolic and adrogenic steroids**

  Central fat distribution (android obesity) is an important risk factor for metabolic and cardiovascular disease in woman (Pi-Sunyer, 1993). These health risks appear to be mostly mediated by increased visceral intra-abdominal fat, rather than subcutaneously distributed abdominal fat. Factors influencing abdominal fat distribution are age, smoking, alcohol consumption, and hormones. Visceral fat accumulation appears to be associated with lower levels of androgens in woman (Björntorp, 1993). Anabolic steroids have been tried in obese post-menopausal women. Treatment with nandrolone deconoate decreased total body fat and increased lean body mass in obese postmenopausal women but was associated with increased visceral fat, unfavourable changes in serum lipids, and undesirable side-effects. These effects included darkening of facial and body hair, acne, rash, changes in sleep habits, breakthrough bleeding, and headache (Lovejoy et al., 1996).

2.17 PHYSICAL ACTIVITY AND THE OBESITY EPIDEMIC

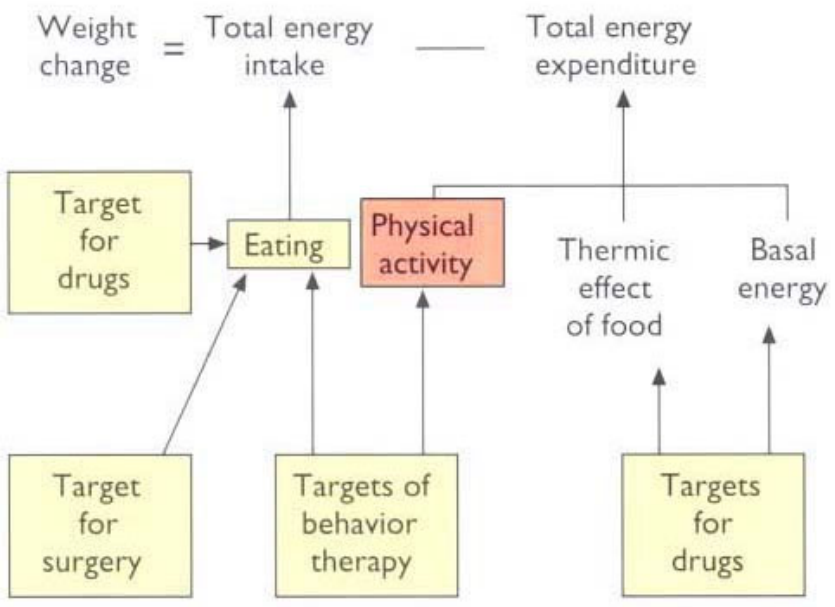


FIGURE 2.20: THE RELATION OF PHYSICAL ACTIVITY TO THE ENERGY BALANCE EQUATION (BRAY 2003)

Body weight is a function of energy balance over an extended period of time. Positive energy balance over weeks and months results in weight gain, whereas negative energy balance has the opposite effect. The increase in the prevalence of overweight and obesity cases worldwide is occurring against a background of a progressive reduction in the energy expended for work and occupational activities as well as for the accomplishment of personal chores and daily necessities (Haskell, 1996; Prentice & Jebb, 1995). The reduction in energy expenditure associated with physical activity brought about by automation and changing job and professional environmental circumstances has been nothing but dramatic in the second half of this century. The energy expenditure of leisure time physical activity may have increased slightly but not enough to keep pace with the changes brought about by urbanization and automation (Bouchard & Blair, 1999).

The availability of relatively inexpensive and highly palatable foods in almost unlimited abundance is undoubtedly contributing to the epidemic as some of the affected individuals eat numerous times a day and consume large portions (Foreyt &

Goodrick, 1995). The proportion of calories derived from fats is also potentially involved (Bray & Popkin, 1998), particularly in those who consume a high-fat diet while living a sedentary life (Stubbs et al., 1995) although the exact contribution of a high-fat diet to the current obesity epidemic remains controversial (Willett, 1998).

The increase of the last decade in the prevalence of overweight and obesity is thought to result from the following circumstances:

a) a large proportion of the population is consuming more calories than individuals of past generations with no changes in habitual daily energy expenditure;

b) for a large number of people, there is an abnormally low daily energy expenditure for a normal caloric intake; or

c) caloric intake per capita is actually lower than expected in comparison with previous generations but daily energy expenditure is, on the average, even lower. In all three scenarios, energy expenditure of physical activity is a major determinant (Bouchard & Blair, 1999).

One can hypothesize that the contribution of a diminished energy expenditure to the current overweight and obesity epidemic is determined by the decrease in the level of habitual physical activity associated with work and chores of daily living and by the growing amount of time spent in a very sedentary mode, such as watching TV, working on the computer, playing video games, etc. It is not associated with decreases in resting metabolic rate or in dietary-induced thermogenesis. There is absolutely no indication that there is a downward secular trend for these two components of daily energy expenditure (Bouchard & Blair, 1999).

2.17.1 Justification for inclusion of exercise for weight-loss

It is increasingly clear that people who maintain a physically active lifestyle or who become involved in endurance exercise programs maintain a desirable level of body composition. Evidence is accumulating to support the contention that exercise may be more effective than dieting for long-term weight control (Miller & Sims, 1981).

Scientific evidence suggest that the combination of dietary modification and exercise is the most effective behavioural approach for weight-loss (NIH, 1998), and the maintenance of exercise may be one of the best predictors of long-term weight maintenance (Miller et al., 1997; Pronk & Wing, 1994). Despite the importance of exercise, there is little evidence suggesting that exercise alone produces magnitudes of weight-loss that are similar to what can be achieved with dietary modification (NIH, 1998).

In a review by Ross et al. (2000) it was shown that when the energy deficit is held constant and other factors that affect energy balance are controlled, exercise can induce significant weight-loss. They have shown that a daily 700 kcal energy deficit produced solely through exercise, with energy intake remaining constant resulted in a weight-loss of 7.6 kg over a 3-moth period. The weight-loss resulting from a similar energy deficit achieved through changes in energy intake was 7.4 kg. It is important to note that the energy deficit in this study was achieved through strictly supervised exercise for a duration of approximately 60 min daily, and there were strong recommendations for participants not to change their dietary intake across this 12-week program (Ross et al., 2000). The failure of exercise to produce a magnitude of weight-loss similar to diet in studies of free-living individuals may be a result of individuals compensating by increasing energy intake and/or failing to achieve adequate levels of energy expenditure (Jakicic et al., 2001).

When examining the effect of exercise on body weight, it has been suggested that there may be “responders” and “non-responders” to the same exercise intervention (Jakicic, 2001). Bouchard et al. (1994) examined the effect of exercise on weight-loss both within and between pairs of identical twins. Results of this study showed that the variance for changes in body weight was 6.8 times greater between pairs than within pairs, suggesting that the effect of exercise may be influenced by genetic differences between individuals. The effectiveness of exercise for weight management may also be influenced by gender, with Wood et al. (1991) reporting that exercise resulted in greater weight-loss in men compared to women. These factors should be considered when examining the effectiveness of exercise for managing body weight across individuals.

2.17.2 Exercise prescription considerations for weight-loss

It is important to consider the amount, intensity and type of exercise that should be recommended for weight-loss. To allow adequate time for individuals to progressively increase their exercise, the recommended level of exercise for sedentary adults during the initial phases of weight-loss should be differentiated from the amount of exercise that can be achieved at later stages in the weight-loss process. The amount of exercise necessary to improve fitness may be different than the amount of exercise necessary for successful long-term weight-loss.

2.17.3 Exercise duration and weight-loss

A direct dose-response relationship has been demonstrated between weight-loss and the time spent in exercise (Gwinup, 1971). An overweight person who starts out at a light exercise intensity with slow walking can accrue a considerable caloric expenditure simply by extending the duration of the exercise. This effect of duration offsets the inability of the previously sedentary, obese person beginning a program at high exercise intensities. The energy cost of weight bearing exercise such as walking is proportional to body mass, thus an overweight person expends considerably more calories to perform the same task than someone of normal body mass.

The current public health recommendation for physical activity is for individuals to participate in at least 30 min of moderate intensity physical activity on most, preferably all, days of the week (Pate et al., 1995). This recommendation has typically been interpreted as a minimum of 150 min of physical activity per week (5d, 30 min. d^{-1}) and is based primarily on the effects of exercise on cardiovascular disease and other chronic conditions such as diabetes mellitus. Close examination of the scientific evidence suggests that levels of exercise greater than this minimum recommended amount might be important for maintaining long-term weight-loss.

Results from a randomised trial of overweight women in a weight-loss program that included dietary modification showed that individuals adopting and maintaining an average of >280 min of exercise per week maintained a weight-loss of 13 kg over an 18-month intervention (Jakicic et al., 1999). This amount of weight-loss was

significantly greater than the 6.5 kg and 3.5 kg weight-losses after 18 months shown with 150-200 and <150 min of exercise per week, respectively. Individuals averaging approximately 280 min of exercise per week showed no weight regain from 6 to 18 months treatment, whereas individuals exercising <200 min-wk^{1} showed significant weight regain during this period. Individuals reporting >200 min of exercise per week also reported >2000 kcal-wk^{-1} of leisure time physical activity measured by the questionnaire developed by Paffenbarger and colleagues (Paffenbarger et al., 1978). The recommendation for levels of exercise that are greater than the minimal public health recommendation is supported by Schoeller et al. (1997), with their results showing that the equivalent of 65 min-d^{-1} of moderate-intensity activity was associated with improvements in the maintenance of long-term weight-loss.

The recommendation to progress overweight adults to 200-300 min of exercise per week or >2000 kcal-wk^{-1} may present a significant challenge for interventionists and public health professionals. This recommendation should be viewed in the context of the exercise level that most individuals are willing to adopt and maintain. In response to this concern, it is recommended that individuals be progressed to these higher levels of exercise gradually over time and that a variety of behavioural strategies be used to facilitate the adoption of this level of exercise. Even in the absence of weight-loss and attainment of these higher levels of exercise, overweight individuals can realize significant improvements in health and improve their level of cardiorespiratory fitness (Lee et al., 1998; Wei et al., 1999).

2.17.4 Exercise intensity and weight-loss

The initial stage of an exercise program for a previously sedentary, overweight person should be developmental in nature and should not include a high total energy output. The individual should be urged to adopt long-term goals, personal discipline, and a restructuring of both eating and exercise behaviours (Mc Ardle & Toner, 1988). It is often counterproductive to include unduly rapid training progressions because many obese women initially show psychological resistance to physical training.

There have been few studies that have adequately examined the impact of various intensities of exercise on weight-loss. Duncan et al. (1991) attempted to maintain total

volume of exercise while manipulating intensity in a 24-week study of overweight women. The results of this study showed that the intensity of exercise affected the magnitude of change in cardiorespiratory fitness, with greater increases demonstrated with higher intensities of exercise, but the intensity of exercise did not result in differential effects on body weight or body composition after 24 weeks of treatment. Data from the National Weight Control Registry suggest that long-term maintenance of weight-loss may be enhanced with at least 26% of exercise being vigorous in intensity (Klem et al., 1997); however these data are from an uncontrolled observational study, which limits the ability to draw any meaningful conclusions related to causality. There are long-term clinical interventions being conducted that focus primarily on the impact of exercise intensity on long-term weight-loss and the prevention of weight regain. It appears that a sufficient amount of moderate-intensity (55-69% of maximal heart rate) exercise can be beneficial for management of body weight, with limited published scientific evidence from randomised trials to support the necessity of more vigorous (≥70% maximal heart rate) forms of exercise for management of body weight in the long-term.

2.17.5 Lifestyle activity and weight-loss

Lifestyle activity may be an effective option for increasing fitness and modifying body weight in overweight adults. Anderson et al. (1999) reported that when combined with a dietary intervention, lifestyle activity resulted in weight-loss that was comparable to aerobic forms of exercise both 16 and 68 weeks of treatment. Dunn et al. (1999) have reported that lifestyle activity is as effective as structured exercise at improving cardio-respiratory fitness across a 24-month intervention. Based on these results, lifestyle activity appears to be a promising alternative to structured forms of exercise.

Although these studies have documented the type of activity that was prescribed, these studies have not provided data with regard to the type of activity that was performed that constituted lifestyle activity. Overweight adults should be encouraged to engage in activities that are at least moderate in intensity as part of a physically active lifestyle. Research is necessary to examine the effectiveness of specific forms of

lifestyle physical activity to change body weight, cardiorespiratory fitness, and risk factors that may be common in overweight and obese women.

2.17.6 Intermittent exercise and weight-loss

There have been a few studies that have examined the effectiveness of intermittent exercise in weight-loss programs (Donnelly et al., 2000; Jakicic et al., 1995; Jakicic et al., 1999). Intermittent exercise has typically been defined as accumulation of 30-40 min of exercise per day through participation in multiple 10- tot 15- min exercise sessions daily (Debusk et al., 1990; Jakicic et al., 1999). There has been interest in this form of exercise because early studies showed that intermittent exercise effectively increased cardio-respiratory fitness and favourably impacts coronary heart disease risk factors (Debusk et al., 1990). This resulted in the Centres for Disease Control and Prevention and the American College of Sports Medicine recommending the "accumulation" of at least 30 min of moderate intensity exercise per day (Pate et al., 1995). Jakicic et al. *(*1995) showed that this strategy was effective for increasing the adoption of exercise in overweight women in a 20-week behavioural weight-loss program and there was a trend that this could potentially improve weight-loss. Jakicic et al., (1999) showed again that this strategy can be effective for initial adoption of exercise; however, there was no added weight-loss benefit when compared with continuous exercise across an 18-month behavioural weight-loss program that also included a dietary intervention. Donnelly et al. (2000) compared continuous and intermittent exercise, with no dietary intervention, over a period of 18 months and showed no change in body weight following treatment. The use of intermittent exercise may be advantageous for individuals that dislike continuous exercise or perceive barriers to continuous exercise. These factors should be considered when prescribing exercise to overweight adults seeking weight-loss treatment (Jakicic et al., 2001).

2.17.7 Resistance exercise and weight-loss

Most research studies have examined the effect of endurance exercise on weight-loss, but the inclusion of resistance training in weight-loss programs has clear advantages. Resistance training is a potent stimulus to increase fat-free mass (FFM), muscular

strength, and power and may be an important component of a successful weight-loss program by helping to preserve FFM while maximizing fat loss (Kraemer et al., 1999; Marks et al., 1995). When resistance exercise is combined with dietary energy restriction, there appears to be little benefit in terms of absolute weight-loss (Kraemer et al., 1997). These results have been consistent across studies with energy intakes as low as <800 kcal-d^{-1} or as high as approximately 1300 kcal-d^{-1}. In a long-term study (40 weeks), Wadden et al. (1997) have shown that resistance exercise alone or in combination with endurance exercise did not enhance weight-loss compared with endurance exercise alone in a behavioural weight-loss program with all groups prescribed a diet ranging from 900 to 1250 kcal-d^{-1}. Leibel et al. (1995) showed that reduction in body weight and fat free mass (FFM) resulted in reduction in resting energy expenditure (REE), whereas increases in body weight resulted in increases in REE. These data may suggest that preserving FFM will prevent declines in REE that are often observed with weight-loss. Intervention studies do not support this belief, with the majority of studies showing that resistance training does not prevent the decline in REE that occurs with diet-induced weight-loss (Geliebter et al., 1997; Kraemer et al., 1999). Kraemer et al. (1999) combined a periodized resistance-training program with an endurance exercise component and dietary modification. The periodized resistance exercise program consisted of 3d-wk^{-1} that alternated heavy and moderate training days. Approximately 11 different exercises were performed, and subjects progressed to three sets of each exercise throughout the duration of the 12-week study. On heavy days, 5-7 repetitions per set were performed to fatigue, with moderate days including 8-10 repetitions per set to fatigue. Endurance exercise consisted of 3 d-wk^{-1} with duration progressing from 30-50 min-d^{-1}, at an intensity of 70-80% of functional capacity. Energy intake was approximately 1500 kcal-d^{-1}. The addition of resistance exercise did not improve weight-loss or blunt decreases in REE compared with diet combined with endurance exercise or the diet only condition. The addition of resistance exercise to endurance exercise and diet modification did not minimize the loss of FFM compared with endurance exercise combined with dietary modification or dietary modification alone. Although resistance exercise may improve muscular strength in overweight and obese adults, there is no scientific evidence to suggest that resistance exercise is superior to more commonly used forms of endurance exercise for weight-loss (Jakicic et al., 2001).

The ability of resistance exercise to improve muscular strength and endurance may be especially beneficial because of the impact on functional tasks (e.g. getting out of a chair, lifting one's own body weight), which may facilitate the adoption of a more active lifestyle in sedentary overweight and obese individuals (Jakicic et al., 2001).

2.7.18 Effectiveness of exercise in weight control

Although exercise prescriptions for weight control vary, the literature reveals that exercise effects on body weight are rather small, but significant (Jakicic et al., 2001). Weight-loss of about 2.0 kg has been reported for various exercise programs depending on duration, intensity and prescription considerations (King & Tribble, 1991). A meta-analytical review reported that exercise causes body weight to decrease at a rate of about 0.2 kg-wk^{-1} and that people do not lose as much weight as would be expected from the prescribed exercise (Miller et al., 1997). The data indicate that the effectiveness of exercise for weight-loss is directly related to the initial degree of adiposity and the total of kilocalories (kcal) expended.

The long-term effects of exercise in weight control seem to be the most promising, but the follow-up data for exercise intervention are scant for the first couple of years post intervention and non-existent after five years. One of these long-term exercise studies that is often cited in the literature compared body weight changes of police officers participating in an 8-week-diet or diet-plus-exercise program consisting of 35-60 min aerobic activity, callisthenics and relaxation techniques 3d-wk^{-1} (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 19 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-year of exercise research also suggests that exercise is critical to weight-loss maintenance (Miller et al., 1997). Weight-loss during the average 21-week exercise program reviewed in this meta-analysis was only 2.9 ± 0.4 kg, but at one year follow up the net weight-loss had increased to 6.1 ± 2.1 kg. On the other hand, weight-loss in the average 13-week diet-plus-exercise program amounted to 11.0 ± 0.6 kg with a 22% regain in weight after the first year (Miller et al., 1997). Any universal

exercise intervention strategy for the obese should be one that would be beneficial for the whole population. For example, people of all sizes can benefit from an exercise program, not just the obese. With regard to obesity intervention per se, the intervention should be one that focuses on developing healthful behaviours and/or physiologic parameters, not body weight, as outcome variables to evaluate the effectiveness of intervention. Formal exercise as well as increased activities of daily living should be encouraged. The exercise prescription should be individualized with a focus on helping the client become more active through frequent, regular activity that is enjoyable.

2.18 BEHAVIOUR MODIFICATION FOR WEIGHT-LOSS

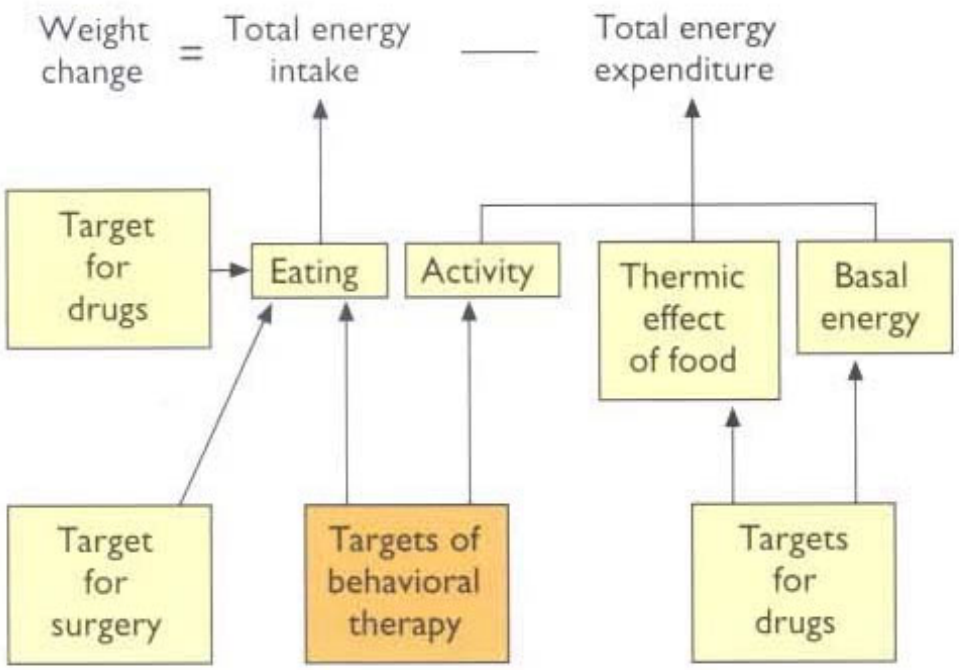


FIGURE 2.21: TARGETS OF BEHAVIORAL THERAPY IN THE ENERGY BALANCE DIAGRAM (BRAY, 2003)

The application of behaviour modification therapy to obesity began in 1967 with the publication of a small paper on the behavioural control of eating (Stuart, 1967). Behaviour modification methodologies are aimed at helping individuals identify the idiosyncratic problems and barriers interfering with their weight-loss management (Institute of Medicine, 1995). No obesity-treatment program can afford to ignore this treatment approach. The principles used in behaviour modification typically include self-monitoring, stimulus control, contingency management, stress management, cognitive behavioural strategies, and social support.

- Self monitoring consists of two steps: self-observation and self-recording of those observations. Food and exercise diaries are used to assess the client's eating habits and activity levels.

- Stimulus control involves identifying the environmental views associated with unhealthy eating and under-exercising. Modifying the views often involves strategies such as limiting eating to specific times and places, buying food when not hungry, and laying out exercise clothing to encourage a regular habit of physical activity.

- Contingency management includes the use of rewards for appropriate behaviour changes, such as reducing grams of fat in the diet and increasing minutes of daily exercise.

- Stress management involves the use of problem-solving strategies to reduce or cope with stressful events. Meditation, relaxation procedures, and regular exercise are examples of stress-reducing techniques.

- Cognitive-behavioural strategies are used to help change client's attitudes and beliefs about unrealistic expectations, appropriate goals, and body image. Examples include the use of affirmations (positive self-statements) and visual imagery (seeing oneself eating and exercising appropriately). The principles and techniques are tailored to each person's specific problems.

- Social support, usually from the family or a group, is used to maintain motivation and provide reinforcement for appropriate behaviour changes. All behavioural principles are used to help individuals adhere to a healthy diet and exercise program.
  (Institute of Medicine, 1995).

There is evidence that including behavioural principles within a weight-loss program improves long-term outcomes. For example, obesity is a chronic disease and should be treated with a chronic disease model to improve overall success (Perri & Nezuam, 1993). It is assumed that it is important to maintain treatment focusing on healthful eating and exercise behaviours to maintain weight-loss and prevent weight regain long-term. In a summary of behavioural weight-loss programs, Wadden (1993) has shown that the duration of treatment programs has gradually increased from the early 1970s to the mid 1990s. This may be important because Perri and colleagues (1987) have shown that maintaining contact with participants long-term improves long-term weight-loss outcome, and this is considered an important component of behavioural weight-loss programs.

2.18.1 What behaviour therapy can do

Fifteen years of research and clinical experience have made it clear what behaviour therapy can, and cannot, contribute to the treatment of obesity.

- Considerable progress has been made in decreasing attrition from treatment. Whereas attrition from traditional outpatient treatment of obesity has been as high as 25 to 75%, most behavioural programmes report rates of 15% or less.

- Side-effects of weight reduction regimens have been greatly reduced.

- Great variability in weight changes during treatment and even greater variability following treatment.

- Prediction of the outcome of behavioural (as of other) treatments for obesity has not been successful. This failure leads to inefficient use of scarce treatment resources.

- By the most important measure of treatment efficacy, weight-loss, behaviour therapy of obesity is only moderately effective.

- Weight-losses achieved during behavioural treatments for obesity are better maintained than are weight-losses achieved by either pharmacotherapy or diet alone.
  (Bender et al., 1987)

The extensive experience of the past few years suggests that the limits of the effectiveness of current behaviour therapy for obesity have been reached. Two kinds of efforts are now being made to extend these limits:

1. making treatment more intensive by combining it with other modalities, particularly, with low fat, low caloric diets; and

2. making treatment more extensive by applying it to large populations through the agencies of weight-loss groups (Wadden, 1993).

The use of portion control diets may also improve weight-loss outcomes by minimizing choice and providing specific guidance to overweight and obese adults that precipitates weight-loss. Structured meal plans which specifically outline types and amounts of food to be consumed resulted in greater weight-loss than recommending a specific energy and fat intake goal with no structure (Wing et al., 1996). In addition, the use of pre-packaged meals with predetermined portion sizes may also be an effective strategy for inducing decreases in energy intake that result in improved weight-loss (Ditschuneit et al., 1999).

It is also important to identify strategies that may facilitate the adoption and maintenance of exercise behaviours in previously sedentary overweight or obese adults. For example, home-based exercise or non-supervised exercise may improve

participation compared with requiring individuals to attend supervised exercise sessions (King et al., 1995). Despite the advantage to supervised exercise for research purposes, there may be advantages to not requiring supervised exercise within clinical weight-loss programs. It may be important to provide exercise options, such as the use of intermittent (Jakicic et al., 1999) or lifestyle (Dunn et al., 1999) approaches to exercise, which may improve the adoption and facilitate the maintenance of a physically active lifestyle.

Regardless of the option selected, patients should strive to develop the skills that have been reported by successful weight-loss maintainers. Such individuals (1) exercise regularly (at least three times weekly), (2) monitor their weight frequently (at least once a week), (3) eat a low-fat diet, (4) record their food intake (at least occasionally) and (5) develop effective problem-solving skills (Wadden, 1995).

2.19 DIETING AS A WEIGHT-LOSS STRATEGY

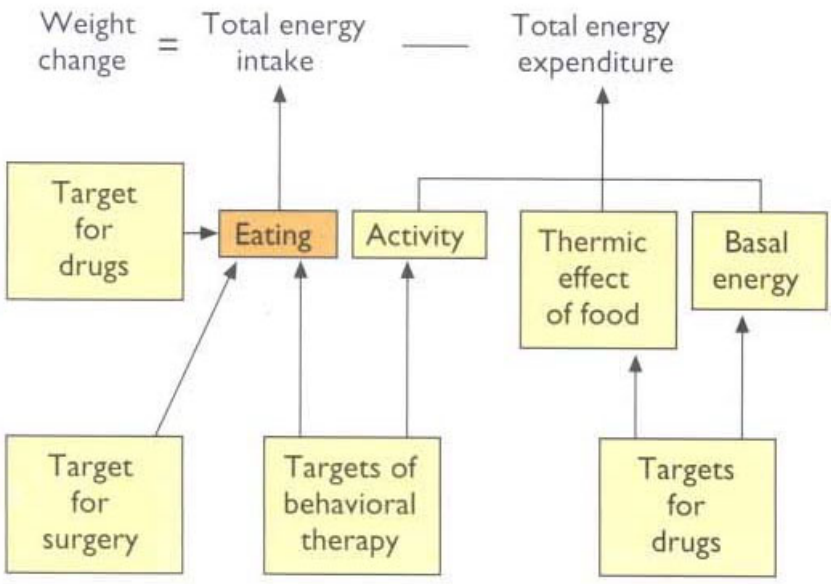


FIGURE 2.22: IDENTIFICATION OF THE SITE AT WHICH DIET WORKS TO INFLUENCE ENERGY BALANCE (BRAY, 2003)

Health professionals recognize that success in long-term weight management is most likely to occur when a program includes long-term changes in lifestyle such as eating in moderation, a physically active daily life that involves exercise, and behaviour modification. The public remains convinced that there must be quicker and easier ways to lose weight without addressing all of these issues. Sporadic dieting attempts designed to cut calories and lose weight, are common. Data on the long-term efficacy of various calorie reducing diets, diet foods and commercial and non-profit weight-loss programs are unavailable to most consumers. Lay people are vulnerable to advertisements and ill-conceived programs in books, magazines, and elsewhere in spite of scientific evidence that many of the claims of effectiveness are exaggerated or outright false. When overweight patients turn to health professionals for help they frequently ask questions about these heavily advertised and promoted diets and weight-loss schemes. It is important for health professionals to know how weight-loss is best achieved and to have the information to answer the patients' questions (Dwyer & Lu, 1993).

The means by which body fatness can be reduced are straightforward. Either energy intake is decreased, energy output is increased, or both are altered simultaneously to produce a net energy deficit. If the energy deficit persists for long enough and other nutrients are provided in adequate amounts, body fat and weight are lost in a linear manner. The slope of the decline is determined by the size of the energy deficit. The loss of fat is directly proportional to the size and duration of the energy deficit. Rate of weight-loss depends also on the extent to which the individual adheres to the regime (Dwyer, 1980; Van Itallie, 1980).

Over the short-term, such as a few weeks, deviation from this linear relationship between caloric deficit and weight-loss often occurs. Weight-loss also reflects shifts in water balance, especially in the first few weeks. Such alterations are most pronounced early in weight-loss, but may occur at other points as well, depending on adherence. The size of the caloric deficit and diet composition can alter the mix of metabolic fuels that are burned for energy, the type of tissue lost, body water balance, and weight-loss (Dwyer & Lu, 1993). Ultimate success in keeping a leaner physique depends on sustaining somewhat lower levels of energy intake and higher levels of energy output, as compared with those prior to weight reduction.

Essential criteria of healthy weight control plans: the seven c's:

- Calories;
- Composition;
- Costs;
- Consumer friendliness;
- Coping with existing health problems;
- Components of sound weight management: healthful hypocaloric diet; physical activity and exercise; behavioural modification;
- Continuation provision for long-term maintenance: plan for maintaining weight at reduced levels after healthier weights achieved by slightly decreased food intakes and increased energy outputs compared with before dieting. (Dwyer & Lu, 1993)

2.19.1 Popular diets for weight-loss

Total fasting was used to reduce body weight quickly in massively obese patients during the late 1950's and early 1960's. Weight-loss at a rate of about 1.0 kg-d^{-1} occurred during the first month of fasting with this leveling off to about 0.5 kg-d^{-1} thereafter (Drenick et al., 1964). 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 wane in popularity (Atkinson, 1986). Fasting is a drastic weight reduction method that has profound metabolic effects. It is diuretic, kaliuretic and saliuretic, and leads to insufficient nutrients unless supplemented. Fasting is self-defeating, since the large losses in lean body mass (LBM) are accompanied by declines in resting metabolic rate (RMR). Energy outputs at rest, which are reflected in the RMR, result from

catabolism in actively metabolizing cells in the vital organs and muscles that constitute the LBM. As LBM falls, so too does RMR. Lethargy is common, voluntary physical activity tends to fall of dramatically, and exercise tolerance is greatly diminished. Energy outputs from physical activity also tend to fall. The metabolic changes associated with starvation, on very hypocaloric diets, and during refeeding are well described in the literature (Aoki & Finley, 1986; Dulloo & Girardier, 1990).

By the late 1960's and early 1970's the focus had shifted to high-protein, low-carbohydrate diets. Popular diets of the time were the Atkins (Atkins, 1972) and Stillman (Stillman & Baker, 1978) diets. 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 refeeding edema (Zeman, 1991).

In the mid 1970's, very low-calorie liquid diets became available. These diets were also known as protein-sparing-modified fasts or liquid-protein diets. Their extremely low energy content (300-400 kcal-d^{-1}) caused rapid weight-loss. In spite of medical supervision, high quality protein, and potassium supplementation, these diets were called the "last chance diet" due to deaths caused by ventricular arrhythmia (Sours et al., 1981). The 58 deaths that were reported in 1977 and 1978 caused the Food and Drug Administration and the US Centres for Disease Control to terminate the use of very low-caloric weight reduction regimens until further studies could assure their safety (Sours et al., 1981).

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. Clients were first only offered 450 to 500 kcal programs, but later 800 kcal programs became available for men and women who wanted to be more active. 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 were gallbladder disease and cardiac problems (Fisler, 1992; Gallagher & Heymsfield, 1994; Wadden et al., 1990).

The 1980's brought about pre-packaged low-caloric diets. Composition of these prepackaged diets, by energy value was 20% protein, 20% fat and 60% carbohydrate. There is no sharp line of demarcation, in terms of their biological effects, between VLCD and more moderate low-caloric diets (LCD) providing 800 to 1 200 kcal-d^{-1}. A rule of thumb is that the less severe the caloric restriction, the less likely are the risks of metabolic complications and other side effects (Dwyer & Lu, 1993). Diets of 800-1200 kcal-d^{-1} are still below RMR for most adults, so they too have noticeable effects on metabolism as well as a loss of adipose tissue. Diets that are this hypocaloric should not be embarked upon without physician approval. They are most suited for individuals who have significant medical reasons for losing weight, such as non insulin-dependent diabetes mellitus, hyperlipidemia and hypertension (Fisler, 1992).

When 800 to 1 200 kcal-d^{-1} LCDs are based on regular foods rather than on specially formulated or fortified products, they usually require vitamin and mineral supplementation to be nutritionally adequate. Multivitamin-multimineral supplements are usually recommended. For women iron and calcium supplementation needs special attention (Dwyer & Lu, 1993). Some of the more reasonable LCD commercial weight-loss programs include Weight Watchers, Diet Workshop, Jenny Craig Nutri-System, Weigh-Less and Sure Slim. Years ago, most of the diet meal replacements were milk-based formula products such as Metrecal, which were sold over the counter. Today, frozen microwavable meals, dried products, and canned meals are available.

For the past two decades balanced deficit diets (1 200 or more kcal-d^{-1}) have become increasingly popular. Diets that provide 1 200 or more kcal-d^{-1} are often referred to as balanced deficit diets (BDD), since deficits in calories and the distribution of energy providing nutrients are better balanced than they are in the high-protein, low-fat, low carbohydrate VLCD's. With wise menu choices from ordinary foods, moderately low caloric BDD's provide most individuals with adequate nutrient intakes. Their higher caloric level makes adherence easier, and minimizes undesirable physiological effects

(Wing, 1989). Some diets permit the dieter to choose from a variety of regular foods, from a variety of frozen entrees, or from foods sold through the weight-loss program. Several low-fat diet books have become bestsellers Pritikin; T Factor diet; Fit for Life; East More Weigh Less; Set point diet (Pritikin, 1982; Katahn, 1989; Diamond & Diamond, 1987; Ornish, 1993; Leveille, 1985).

The Ornish diet itself was originally designed as a rehabilitation diet for cardiovascular disease (Ornish, 1992) but has resurfaced as a weight-loss diet (Ornish, 1993). Only about 10-15% of the 1 200-1 700 kcal in these diets typically comes from fat (Diamond & Diamond, 1987; Fisher & Lachance, 1985; Katahn, 1989; Ornish, 1993; Pritikin, 1982; Leveille, 1985). The singular focus on 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 increase (Centres for Disease Control and Prevention, 1994). Miller et al. (1993) have shown that obese 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 fiber than the lean (Miller et al, 1994).

2.19.2 Cost and consumer appeal of diet programs

The costs of various diets and other obesity therapies range from modest to exorbitant. Obesity is big business and billions of rands are spent each year on its treatment. The good news for dieters is that there is no association between cost and effectiveness. The bad news is that standardized disclosures of the effectiveness of various treatments for obesity are simply unavailable (Dwyer & Lu, 1993). Medically supervised programs, particularly those that supply food or extensive interventions such as special exercise programs, are among the more costly programs. Few medically supervised programs provide monitored exercise components. Some programs demand that the dieter pay all costs, regardless of the services actually used or the weight-loss that is achieved. Other programs provide rebates to those who are able to keep their weight off over the long-term (Dwyer & Lu, 1993).

Ethical marketing and business practices are mandatory in obesity treatment programs, whether they be for profit or non-profit. There is a need for greater documentation of the various effects, both positive and negative, of existing weight control services. One and five year cure rates should be included in description of the programs (Fisher, & Lachance, 1985). Many high-quality programs and regimens exist, but professional and operational standards for consumer safety and protection in the weight-loss industry are insufficient and need to be improved. Weight-loss involves a health-related human service and the ethical standards that apply to it should incorporate those involved in all forms of health care (Dwyer & Lu, 1993).

2.19.3 Effectiveness of dieting in weight control

Despite the immense popularity of dieting over the past 40 years, the effectiveness of these programs must be questioned. A review of the VLCD programs suggests 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 claims, and it is difficult to interpret the results because dropout rates can be as high as 80% in some VLCD programs (NIH Technology Assessment Conference Panel, 1993). The initial weight-loss success seen with VLCD is followed by gradual weight regain to the point that VLCD programs show no more success long-term than other forms of therapy (National Task Force On the Prevention And Treatment Of Obesity, 1993). Results from programs with more moderate dietary restrictions seem less promising than those from the VLCD. The conventional 1 200 kcal diet will produce a weight-loss of 8.5 kg in 20 weeks and 66% of this can be maintained at one year (Wadden, 1993). As time progresses, weight is regained until pre-diet weights are reached within five years (Brownell & Jeffery, 1987; Kramer et al., 1989).

Nutrition education and behaviour modification have been touted as having self-empowering capabilities. During the average 18-wk behaviour modification program, one can expect to lose about 10 kg, but 33% of this is regained during the first year post-diet with a 95% relapse after two years (Kramer et al., 1989). Similar relapses have been seen with community education programs, worksite interventions, and home correspondence courses (Jeffery, 1993). Initial weight-loss in these programs is marginal and maintenance after 1-3 years is negligible (Jeffery, 1993). 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; Stevens et al., 1989). 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).

Data to support the weight-loss effectiveness claims of the commercial weight-loss industry were requested by the National Institutes of Health (NIH) and US Food and Drug Administration at the 1992 NIH Technology Assessment conference on methods for voluntary weight-loss and control (Hyman et al., 1993; NIH Technology Assessment Conference Panel, 1993). Material was received from only five companies, three representing non-physician-directed programs, and two representing physician-directed programs. For the non-physician-directed programs, one company submitted a study showing reduced cardiovascular risk with short-term use of their program along with several abstracts that were judged as being scientifically inadequate because of poor study design, high dropout rates, small sample sizes, and inadequate follow up. The second company submitted four studies for review but later withdrew them. The third company submitted information that was judged as being inadequate (Hyman et al., 1993).

For the physician-directed programs, one company submitted 55 publications that were well designed and well controlled. The studies showed that the major program benefits were not weight-loss but better control of diabetes and reduced cardiovascular risk. The second company submitted three published articles showing beneficial metabolic and weight responses to the program, but further data were deemed necessary to draw conclusions. The paucity of weight-loss effectiveness data received led to the industry being judged to be inadequate, questionable and inconclusive (Hyman et al., 1993).

Schachter (1982) hypothesized that the poor weight-loss results seen in clinical research studies and commercial programs is moulded largely by a self-selected group of people who, unable or unwilling to help themselves, go to therapists for help, thereby becoming the only easily available subjects for studies of recidivism and

addiction. Regardless of whether it is true that only the most difficult obesity cases come to professionals for help, the argument remains that no commercial program, clinical program, or research model has been able to demonstrate significant long-term weight-loss for more than a small fraction of the participants. Given the potential dangers of weight cycling and repeated failure, it is unscientific and unethical to support the continued use of dieting as the only intervention for obesity (American Dietetic Association, 1997; Berg, 1995; Garner, 1991).

2.20 SURGERY IN WEIGHT CONTROL

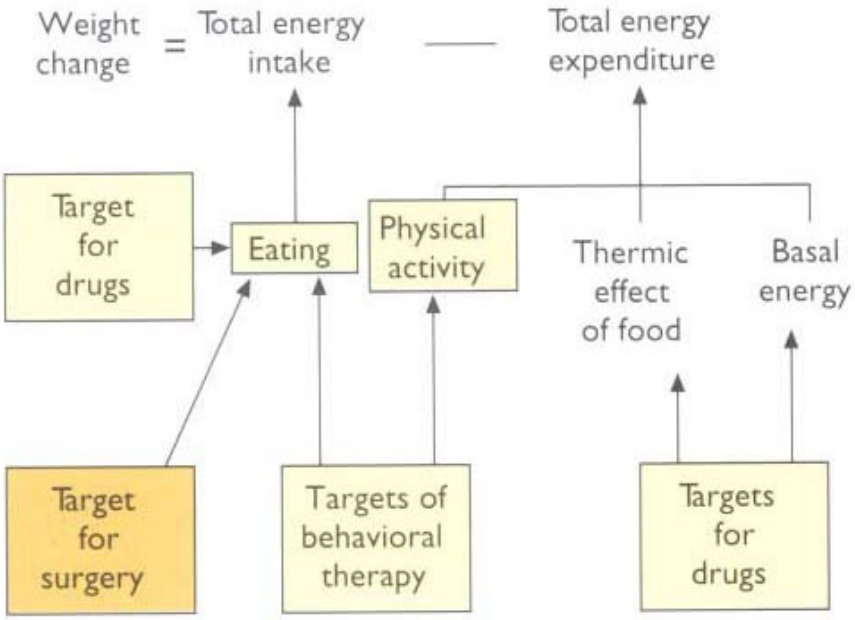


FIGURE 2.23: ENERGY BALANCE DIAGRAM SHOWING WHERE SURGICAL TREATMENT HAS ITS INFLUENCE (BRAY, 2003)

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).

2.20.1 Gastric surgery

Two proven surgical procedures exist for the treatment of severe and very severe obesity: vertical banded gastroplasty and Roux-en-Y gastric bypass. Vertical banded gastroplasty consists of constructing a small pouch with a restricted outlet along the lesser curvature of the stomach. Roux-en-Y gastric bypass involves constructing a proximal gastric pouch whose outlet is a Y-shaped limb of small bowel of varying lengths (National Institutes of Health, 1992). Vertical banded gastroplasty is less complex to perform and has fewer preoperative complications than gastric bypass, but produces less long-term weight-loss (Sugerman et al., 1989). On the other hand, a higher risk of nutritional deficiencies exists following gastric bypass (National Institutes of Health, 1992).

Biliopancreatic bypass is a less proven surgical procedure for the treatment of severe and very severe obesity. Biliopancreatic diversion is an operation designed to bypass much of the intestine without introducing the complications from overgrowth of bacteria in the bypassed bowel. Biliopancreatic diversion has the configuration of the Roux-en-Y gastric bypass, but the limbs are much longer and much of the stomach is removed to decrease the risk of stomach ulcer. Biliopancreatic bypass was introduced by Scopinaro et al., (1980) to answer the objective of reducing weight to a normal level. Biliopancreatic bypass requires intensive follow-up, with a considerable array of laboratory work. Since any operation for treatment of obesity must be effective for a lifetime, the use of a complex operation that requires ingestion of large amounts of protein and indefinite and expensive follow-up should not be used unless the patient can understand and afford these requirements and be willing to have the operation reversed later in life if the requirements cannot be met (Scopinaro et al., 1980).

Gastric banding is another less proven surgical procedure for the treatment of severe and very severe obesity. Gastric banding involves the placement of a plastic collar around the entire circumference of the stomach with a small portion of the stomach left above the band, as the meal-sizing reservoir. Since there is no stapling there is no risk of breakdown of a staple line (Duzmak, 1991). The problem with gastric banding has been related to the use of pouches that were too large (often unmeasured as well) and the difficulty in calibration of the passage between the upper and lower stomach.

Reoperations were frequently required to remove or reduce the tension of a band, that was too tight. Enlargement of a pouch that was too large to begin with also resulted in obstruction, which was another reason for removal of the band (Duzmak, 1991).

As a result of above mentioned surgical procedures substantial weight-loss generally occurs within 12 months of the operation, with some of the weight being regained within two to five years. With weight-loss comes improvement in the co morbid conditions that often accompany obesity. The risks associated with the surgical treatment of obesity include postoperative complications, micronutrient deficiencies and late postoperative depression (National Institutes of Health, 1992).

There is compelling evidence that comorbidities are reduced in severely obese patients who have lost weight as a result of gastric surgery. It is puzzling that this treatment is not more widely used for severely obese individuals at very high risk for obesity-related morbidity and mortality. It is possible that health-care providers and individuals alike fail to fully understand the severity and costs of obesity in terms of both increased morbidity and mortality and its impact on the quality of life. There is also an intrinsic fear of the dangers of surgery due, in part, to a lack of knowledge. Mortality associated with gastric surgery for obesity is less than one percent (Kral, 1992). It has been proposed that most of the complications associated with this type of surgery, unlike most other surgery, are modifiable by behaviour. Kral (1994) notes that the vomiting seen in approximately ten percent of patients after surgery is due more to eating behaviour than to stenosis or stricture of the gastroplasty stoma. Another reason for the limited use of gastric surgery for obesity is that it is not always reimbursable (Chase, 1994). In the Swedish Obesity Study, patients in the surgical intervention group reported marked improvements in health, mood, and obesity-specific problems compared to controls (Näslund, 1994). This same study estimated that seven percent of the costs to the work force of lost productivity due to sick leave and disability pension are related to obesity. Obesity surgery would profit from cost-benefit analyses that include the social and psychological benefits that may experience from the procedure. Weight-loss surgery clearly involves hospital care. Any surgical program should be supported by appropriate nutritional, medical, and psychological counselling for the long-term management of the patients enrolled, although some

programs of this kind in fact have no such support systems (National Institutes of Health, 1992).

A major contribution of the surgical treatment of obesity has been the light that it has thrown on the origins of the psychological problems that afflict severely obese persons. For many years it had been believed that psychological problems were root causes of obesity: the corollary of this belief was that the more severe the obesity the more severe the psychological problems. It is true that severely obese persons often have severe psychological problems, but experience with surgical treatment of severe obesity has made it clear that these psychological problems are largely secondary to the obesity (Rand & Macgregor, 1991).

Rand and Macgregor (1991) studied the perceived liability of morbid obesity in patients three years after successful obesity surgery and found that the perception of these patients was quite different from that of patients with other medical problems. The formerly obese would choose a major handicap such as deafness, dyslexia, diabetes, legal blindness, very bad acne, heart disease, or amputation of one leg, in preference to resumption of severe obesity. These answers are in marked contrast to those people with other physical disabilities. People with these disabilities almost always find the familiar condition to be preferable to some other condition with which they have had no experience. All patients expressed a preference for being of normal weight and of modest means as compared with the imagined choice of being a morbidly obese multimillionaire.

Rand and Macgregor (1991) have also investigated the conditions that have given rise to the derogatory views obese people have of themselves, beginning with Gluttony, one of the seven deadly sins listed by the early Christian church. Even today severely obese persons suffer from prejudice and discrimination. They are frequently and erroneously considered to be lacking in will power, lazy, and emotionally unstable. Many physicians share this attitude toward the severely obese.

After operation and weight-loss these patients perceived little or no prejudice in their daily lives. The only exceptions were members of the medical profession and insurance companies. Self-insured formerly obese persons often cannot obtain

insurance at the rates charged to normal weight adults even though they have a normal weight following a gastric reduction operation (Rand & McGregor, 1991). It might be expected that an operation that markedly reduced weight would have an effect on marriage. Neill et al. (1978) thought there was a destabilizing effect. Rand et al. (1982) cast a far more favourable light on the marital consequences of gastric restriction surgery. They found a relatively high frequency of divorce, but only among couples in conflicted marriages, in which divorce might be viewed as a positive step. In uncomplicated marriages there was increased marital satisfaction. All studies agree that sexual satisfaction is improved. Hafner and Rogers (1990), studying the adjustment of husbands to their wives weight-loss, found that patients became more assertive and their husbands less assertive, and more dissatisfied.

There is today no basis for requiring routine psychological testing of patients before they are selected for treatment for obesity with an operation. However, psychological testing might influence the decision to operate upon a patient whose weight was of borderline severity. It might be of greatest use to identify patients for whom special care could be provided in an effort to reduce the risk of medical and psychological complications and to ensure satisfaction with the operation (Stunkard & Wadden, 1993).

When do patients choose to have an operation for severe obesity? They are usually at their highest lifetime weight and it might be concluded that they reach a degree of stress from the severity of obesity that causes them to elect this drastic action. Most of them have tried and failed at diet and other non-surgical means. Rand et al. (1983) found that there were significantly more stressful life events during the 1- and 3-year period before gastric bypass for obesity than occurred in a control group of adults. These events included a major illness of a family member, major personal illness, major financial difficulties, a move to another city, or a new person in the household.

Rand et al. (1983) suggested that clinicians consider whether a patient was seeking a operation because of added life-stressful events, when in less stressful times the patient might seek other, safer means of weight control. An operation might be temporarily deferred, but there is no more effective, safer means of weight control.

The safest course for most severely overweight patients is still an effective weight-reducing operation (Stunkard & Wadden, 1993).

2.20.2 Plastic surgery

In 1992 more than 1.5 million people in the United States underwent plastic surgery to alter appearance. In 1996, this figure rose to over 1.9 million. Women comprise 89% of clients who undergo plastic surgery (American Society of Plastic and Reconstructive Surgeons, 1997).

Liposuction is a popular method of plastic surgery treating the obese patient, accounting for 5.6% of all plastic surgeries. In 1996 more than 109 000 people elected to have liposuction alter their appearance. The satisfaction index for lipectomy is equivocal (American Society of Plastic and Reconstructive Surgeons, 1997). In a survey of 1339 patients who underwent lipectomy, Dillerud and Häheim (1993) reported a 76% satisfaction rate with the results. The authors observed that many of the patients assumed the procedure would permanently remove fat and prevent its regain. Thirty percent of patients complained that too little fat was removed. Half the patients reported weight gain after surgery, and 29% claimed that their fat returned to the site of the liposuction. Dissatisfaction and disillusion with the results of liposuction may lead to lawsuits (Dillerud & Häheim, 1993).

2.21 ALTERNATIVE TREATMENTS FOR WEIGHT-LOSS

Safe and effective alternative treatments for weight-loss would be very desirable given that obesity is an increasingly prevalent condition with serious adverse health outcomes.

The majority of alternative treatments for weight-loss have not been clearly demonstrated to be safe or effective (Allison et al., 2001). Alternative treatments are defined by exclusion. Allison et al. (2001) define any currently available treatment intended to produce weight-loss or reduction as alternative if it is not one of the following:

- A pharmaceutical agent currently approved by the US Food and Drug Administration (FDA) or a drug under development by a pharmaceutical company;

- Surgery; and

- The application of cognitive-behavioural techniques to promote weight-reducing changes in diet and exercise behaviours.

Some specific treatments selected for further analyses are reviewed below.

2.21.1 Acupuncture and acupressure

Acupuncture (APC) is the insertion of small needles into the body at defined anatomic locations for therapeutic purposes. Acupressure (APR) is the application of pressure without puncture to those same points. The exact mechanisms by which APC or APR achieve effects in situations where they are efficacious are not clear (Chan, 1984). It is claimed that acupuncture stimulates the auricular branch of the vagal nerve and raises serotonin levels, which increases tone in the smooth muscle of the stomach, thus suppressing appetite. Numerous reports have been published examining the effects of APC or APR (Liu et al., 1993; Soong, 1975; Sun & Xu, 1993; Zhang, 1990). The majority of these report very beneficial effects. The majority have also been uncontrolled case studies. Better controlled trials that directly studied body weight, observed no significant effects of APC or APR (Ernst, 1997).

Richards and Marley (1998) randomly divided 60 overweight subjects into either a treatment (i.e., transcutaneous electrical nerve stimulation of specific auricular acupuncture points twice daily using a "Acuslim" device) or control group, and found that 95% of those in the treatment group reported suppression of appetite, whereas none of the control group noticed such a change. The mean weight-loss in the treatment group (± 1.35 kg) was significantly greater than that found in the control group (+ 0.25 kg). The use of electrical muscle stimulation in this study places it outside the category of traditional acupuncture and in a category of its own, and

replication by independent investigators with close attention to blinding and placebo effects would be desirable (Allison et al., 2001).

2.21.2 Aromatherapy

Aromatherapy is a term that "encompasses everything from a marketing adjective for cosmetic products to a serious branch of complementary medicine, usually involving massage with essential oils" (King, 1994). Cole and Bond (1983) define aromatherapy as exposure to particular olfactory stimuli with the intent of producing weight-loss through mechanisms other than aversive conditioning. At least two uncontrolled studies suggested efficacy of aromatic inhalants in producing weight-loss (Hirsch, 1993). The statistically significant reductions in weight were limited to subgroups defined post-hoc.

2.21.3 Hypnosis

Hypnosis has been used as a treatment or as a component of treatment for obesity since at least the 1950's (Winkelstein, 1959). The majority of qualitative reviews have concluded that hypnosis has either no effect or a very trivial effect on weight reduction or the maintenance of treatment-induced weight reduction (Wadden & Flaxman, 1981). The vast majority of the literature pertaining to hypnosis has not been subjected to meta-analysis because of methodological problems inherent in their design (i.e., the absence of a non-hypnosis control group, multi-component protocols that make it impossible to isolate the effect of hypnosis, small sample sizes, the preponderance of case studies, the absence of intent-to-treat analyses). Recent meta-analytic reviews have yielded, at best, equivocal findings with respect to the role of hypnosis as an adjunct to cognitive-behavioural treatment of obesity (Cochrane, 1992). Allison & Faith (1996) compared the efficacy of two programs of hypnosis among 172 overweight adult women. One program involved imagery, diet, behaviour management, support, and overt aversion (i.e., electric shock, disgusting tastes, and smells), and the other had identical components but without the overt aversion. Both groups lost comparable amounts of weight.

2.21.4 Electro-muscular stimulation

In recent years a number of devices have been promulgated for the localized, non-surgical reduction of adipose tissue. These range from mechanical heating devices and occlusive plastic wraps and garments to electrical pulse generators which produce repetitive muscular contractions in the affected areas (Bailey, 1976).

In essence, such apparatus are automatically cycling, multiple-output, faradic muscle stimulators, which produce trains of pulses with variable pulse repetition frequency. The individual pulses are of short duration and of low energy, but at appropriate gain levels the pulse trains produce rhythmic and powerful muscular contractions when they are fed to the muscle by skin contact electrodes placed over or near the motor points. It is claimed that repeated application of such pulse stimulation produce breakdown of adipose tissue by local passive exercise of the muscle unit, and so afford a generalized reduction in both size and weight (Bailey, 1976).

The usual treatment session lasts 30 to 40 minutes, and is repeated two or three times per week The conductive rubber electrode pads are place over those parts of the body (on or near motor points) where muscular contractions are desired, and the gain levels are adjusted so as to produce maximum muscle contractions without discomfort. During the treatment period the patient is given some simple dietary rules, but generally no drastic caloric restrictions are imposed.

There is a paucity of literature published specifically regarding the efficacy of electrical muscle stimulation (EMS). In this regard only two articles directly linked to this study, and a few indirectly related articles could be located, each publishing varing results.

On an early study, focussing on an obese population, Bailey (1976) conducted an in-depth study of the Hawkins Electrokinetic Body Activating (EBA) Machine to evaluate the safety and effectiveness of the device as a method of localized adipose tissue (fat) reduction and general weight-loss modality. In this trial a group of 40 moderately obese female patients with a mean age of 34-35 years, was selected to

serve as subjects. The pre-posttesting battery included measurements of body weight, height, body surface area and appearance (via photographs).

Subjects were given a course of six weeks treatment (3 x one-hour sessions per week) followed by a four-weeks break with no specific instruction. They were then required to attend for a second six-weeks treatment. No attempt was made to regulate the patients diet or exercise during the intermediate four-week period, which was included as a balancing device to lessen any psychological halo effect.

Of the 30 subjects completing the trial, all lost weight in significant amounts (significant at 1% level). Weight losses ranged from 2.2 kg to 21.5 kg with a mean weight-loss of 7.83 kg. Twenty subjects (50%) lost less than 7.83 kg, but even in this group significant losses were noted in specific tissue regions, such as the "upper arms", the abdomen and particularly the thighs. All patients showed a reduction in body surface area and all showed localized reduction in fat depot areas. There were no reported side effects of treatment, and all subjects expressed satisfaction with the weight and tissue losses achieved.

Abstinence from exercise often results in gradual deterioration in exercise capacity and muscle strength. Electrical muscle stimulation (EMS) is seen as a method of augmenting muscle performance. It has been shown to improve oxidative potential of the muscle (Bigard et al., 1993). Clinical trials in humans have shown that electrical muscle stimulation improve muscle strength and performance in patients with major knee ligament injuries (Wigerstad-Lossing et al., 1988), those who are immobile after surgery (Morrissey et al., 1985), promotes muscle growth in paraplegic patients (Buckley et al., 1987) and improves the performance of ischaemic muscles in patients with peripheral vascular disease (Tsang et al., 1994).

Bourjeily-Habr et al. (2002) noted that although exercise training improves exercise tolerance in most patients with obstructive pulmonary disease, some severely affected patients may be unable to tolerate it because of incapacitating breathlessness. This led the investigators to test whether electrical muscle stimulation of the lower extremities could improve muscle strength and exercise tolerance in patients with moderate to severe chronic obstructive pulmonary disease. The investigator performed electrical

muscle stimulation of the lower extremities for 20 minutes three times a week for six weeks in 18 medically stable patients aged 60 ± 1.5 years. The patients were divided into two equal groups who received either genuine or sham treatment. Quadriceps and hamstring muscle strength, exercise capacity and peak oxygen uptake were measured at baseline and at the end of the six weeks of stimulation. It was found that the muscle stimulation improved quadriceps function by 39.0 ± 20.4 percent in the treated patients compared with 9.0 ± 8.1 percent in the sham-treated group. The investigators also found that the muscle stimulation improved hamstring muscle strength by 33.9 ± 13.0 percent in the treated patients compared with 2.9 ± 4.7 percent in the controls. There were no significant changes in lung function, peak workload or peak oxygen consumption in either group.

Poor exercise capacity is also a common manifestation among the obese. The American College of Sports Medicine acknowledges that obese individuals could reap health benefits from exercise without demanding that the exercise meet the traditional intensity requirements suggested for weight-loss (American College of Sports Medicine, 1990). The Surgeon General's Report on Physical Activity and Health declared that physical activity need not be vigorous to improve health (US Department of Health and Human Services, 1996).

In cognisance of the foregoing, this study thus set out to determine whether or not, and to what extent, the advent of electrical muscle stimulation (EMS) could make a significant contribution to help the obese. In a more recent study related to this investigation, Porcari et al. (2002) recruited 27 college-aged volunteers to test the effectiveness of electrical muscle stimulation devices (EMS) on muscle strength, muscle tone, body weight, and body fat in healthy individuals. Volunteers were assigned to either an EMS (n = 16) or control group (n =11). The EMS group underwent electrical muscle stimulation 3 times per week following the manufacturer's recommendations, whereas the control group underwent sham stimulation session. The pre- and posttesting battery included measurements of body weight, body fat (via skinfolds), girths, isometric and isokinetic strength and appearance (via photographs from the anterior, side and posterior) EMS had no significant effect on any of the measured parameters. Claims relative to the

effectiveness of EMS for the apparently healthy individual are not supported by the findings of this study.

2.22 RECOMMENDATIONS FOR WEIGHT-LOSS TREATMENTS

The American College of Sports Medicine recommends that individuals seeking weight-loss treatment and prevention of weight regain select programs that meet the following guidelines:

1. It is recommended that individuals with a BMI >25 kg $\cdot m^2$ consider reducing their body weight, especially if this level of body weight is accompanied by an increase in abdominal adiposity. Individuals with a BMI ≥30 kg $\cdot m^2$ are encouraged to seek weight-loss treatment. Although it is recognized that BMI may misclassify the health risk of very active and/or lean individuals, its use provides a meaningful clinical assessment of health risk. Moreover, although it is also recognized that more sophisticated measures of body composition are available, there is no consensus on the absolute amount of body fatness at which health risk increases.

2. It is recommended that overweight and obese individuals target reducing their body weight by a minimum of 5-10% and maintain this weight-loss in the long-term. This amount of weight-loss is consistent with what is attainable with standard weight-loss programs that focus on modifying eating and exercise behaviours, and this amount of weight-loss has been shown to be associated with improvements in risk factors and a reduced likelihood of chronic diseases including coronary heart disease, type II diabetes, hypertension, and hyperlipidemia.

3. It is recommended that individuals strive for long-term weight maintenance and the prevention of weight regain over the long-term, especially when weight-loss is not desired, or when attainment of ideal body weight is not achievable. Prevention of weight gain or weight regain has been defined as maintaining a body weight that is within 2.3 kg of one's current weight.

4. It is recommended that weight-loss programs target changing both eating and exercise behaviours, as sustained changes in both behaviours have been shown to result in significant long-term weight-loss. Moreover, it is important for programs targeting modifications in these behaviours to incorporate strong behavioural modification strategies to facilitate the adoption and maintenance of the desired changes in behaviour.

5. It is recommended that overweight and obese individuals reduce their current level of energy intake by 500-1000 kcal.d^{-1} to achieve weight-loss and that this be combined with a reduction in dietary fat to <30% of total energy intake. It is also recommended that an individualized level of energy intake be established that prevents weight regain after initial weight-loss, while maintaining a low-fat diet (≤30% of total energy intake). Additional research is needed with regard to changes in other macronutrients and long-term weight-loss.

6. It is recommended that overweight and obese individuals progressively increase to a minimum of 150 min. of moderate intensity physical activity per week, as this level of exercise may have a positive impact on health in over-weight and obese adults. However, for long-term weight-loss, overweight and obese adults should eventually progress to higher amounts of exercise (e.g., 200-300 min.wk^{-1} or ≥2000 kcal .wk^{-1} of leisure-time physical activity).

7. It is recommended that resistance exercise supplement the endurance exercise program in overweight and obese adults that are undertaking modest reductions in energy intake to lose weight. Resistance exercise should focus on improving muscular strength and endurance in this population.

8. It is recommended that pharmacotherapy for weight-loss only be used in individuals with a BMI ≥30 kg ·m^2 in the presence of additional comorbidities. In addition, it is recommended that weight-loss medications only be used in combination with a strong behavioural intervention that focuses on modifying eating and exercise behaviours, and be used under the supervision of a physician. Jakicic et al., 2001 (American College of Sports Medicine Position Stand).

CHAPTER 3

METHODS AND PROCEDURES

3.1 SUBJECTS

A group of 69 females between the ages of 25 - 40 years (mean age = 35.26 ± 6.02 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 electrical muscle stimulation (EMS) performed on Slimline Slimming Machines in conjunction with, and without, a thermogenic agent (Thermo Lean) and following a specific diet; pre-menopausal; obese (BMI > 30); 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 written informed consent (Appendix A) prior to participating and took cognisance of the compliant requirement of not engaging in any exercise in addition to that required over the duration of the study. During the course of the investigation seven subjects withdrew - three because of medical and four 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 electrical muscle stimulation (EMS) performed on the Slimline Slimming Machines in conjunction with, and without, a thermogenic agent (Thermo Lean) and following a specific diet. In order to achieve this goal a pretest-post test placebo-controlled experimental groups design, with three levels of the independent variable, was adopted for the study (Appendix F). Subjects were randomly assigned to one of the following three groups:

- Group TS (N = 23) - Thermogenic Stimulation and following a standardized diet.
- Group EST (N = 23) - Electrical Muscle Stimulation and Thermogenic Stimulation combined and following the standardized diet.
- Group ESP (N = 23) - Electrical Muscle Stimulation and a Thermogenic placebo combine and following the standardized diet.

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

Table 3.1: Subject Characteristics

GROUPS		TS (N = 23)		EST (N = 23)		ESP (N = 23)	
VARIABLE	UNITS	PRE (Mean)	Std. Dev.	PRE (Mean)	Std. Dev.	PRE (Mean)	Std. Dev.
Age	years	33.50	± 6.65	37.80	± 3.49	34.55	± 6.69
Stature	cm	166.57	± 6.40	166.01	± 6.71	164.87	± 6.52
Body Mass	kg	98.53	± 22.13	99.99	± 17.00	100.12	± 24.08
Body Mass Index	kg/m^2	35.49	± 7.51	32.53	± 5.13	36.32	± 7.02
Lean Body Mass	kg	51.35	± 6.48	48.86	± 6.88	51.27	± 8.19
Body Surface Area	m^2	2.06	± 0.19	1.92	± 0.18	2.04	± 0.21
Body Fat	%	46.91	± 4.82	45.26	± 2.77	48.05	± 4.43
Waist-to-Hip Ratio		0.78	± 0.06	0.79	± 0.06	0.79	± 0.06

TS = Thermogenic Stimulation and following a standardized diet.

EST = Electrical Muscle Stimulation and Thermogenic Stimulation following a standardized diet.

ESP = Electrical Muscle Stimulation and following a specific standardized diet (Placebo controlled).

Table I summarises the subject characteristics of the respective experimental groups at the onset of the study. 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 DEPENDENT VARIABLES (MEASUREMENTS)

The following categories of dependent variables were measured during the pre- and post-tests:

- Anthropometry
- Morphology
- Ultrasound Sonography
- Respiratory Quotient (RQ)
- Pulmonary Function

- Haematology
- Cardiovascular Responses
- Musculoskeletal Function

3.3.1 Anthropometry

All variables, unless stated otherwise, were measured according to the procedures of the Anthropometric Standardization Reference Manual of Lohman et al. (1988).

3.3.1.1 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 stadiometer. The subject stood barefoot, feet together and heels, buttocks and upper part of the back touching the gauge 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 alligned 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.

3.3.1.2 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.

3.3.1.3 Skeletal Widths

Skeletal width measurements are 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; Carter & Heath, 1990). 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.

3.3.1.4 Saggital Height

Saggital height or abdominal depth is the vertical distance from the small of the back to the front of the abdomen when the subject is lying supine and is used as an indication of visceral fat (Sjöstrom, 1991). Apparently an increased amount of visceral fat would maintain the depth of the abdomen in a saggital direction, while subcutaneous abdominal fat would have an apposite effect due to the force of gravity (Van der Kooy & Seidell, 1993). Although saggital height was mostly derived from computed tomography or magnetic resonance images in the past, it was measured anthropometrically in this study. A small scale anthropmeter which measures to the closest 0.1 mm, was used to determined saggital height. Two measurements were made:

1) Saggital ½ umbi:

for the first measurement the spirit level of the anthropometer was placed on the abdomen halfway, between the xyphoid process and the umbilicus;

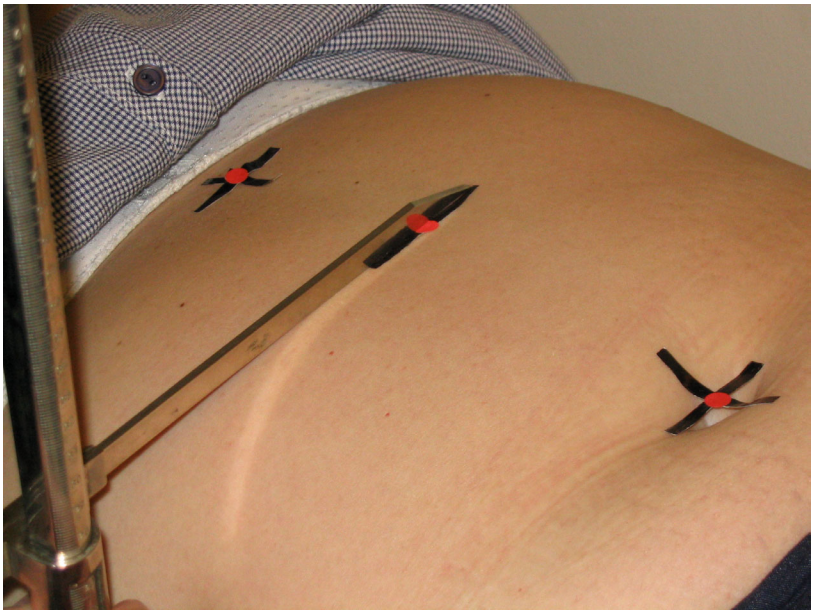


FIGURE 3.2: SAGGITAL HEIGHT ½ UMBI

2) Saggittal umbi:

and for the second, the spirit level of the anthropometer was placed on the umbilicus (Zamboni et al., 1998).

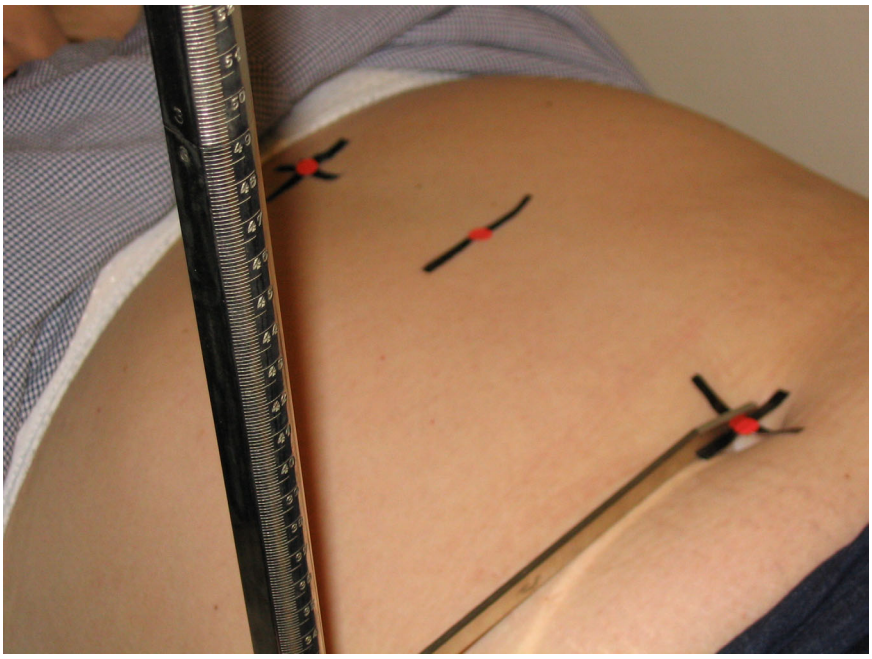


FIGURE 3.2:SAGGITAL HEIGHT UMBI

Measurements were made at the end of normal expiration with subjects lying on their backs on the plinth with knees bent up and the small of the back pushed down on the plinth to counteract the effect of large buttocks.

3.3.1.5 Skinfolds

Skinfolds were taken using a John Bull skinfold calliper exerting a uniform pressure of 10 g per mm^2 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, medial-calf, abdominal, and mid-thigh.

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.

Abdominal: Skinfold was taken 3 cm lateral and 1 cm inferior to the centre of the umbilicus.

Mid-thigh: Skinfold was taken on the anterior aspect of the thigh midway between the inguinal crease and the proximal border of the patella.

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.

3.3.1.6 Girth Measures

A Rabone-Chesterman calibrated steel tape and the cross hand technique was used for measuring all 10 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 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 ten sites measured were:

1. Calf - at the point of maximum circumference.

2. Mid-thigh - midway between the distance from the superior margin of the patella to the anterior superior iliac spine.

3. 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.

4. Contracted upper-arm - at the point of maximum circumference.

5. Forearm - at the point of maximum circumference.

6. Hip – at maximum posterior extension of buttocks.

7. 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.

8. 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.

9. Abdominal (AB^1) – abdominal circumference anteriorly midway between the xyphoid process of the sternum and the umbilicus and laterally between the lower end of the rib cage and iliac crests.

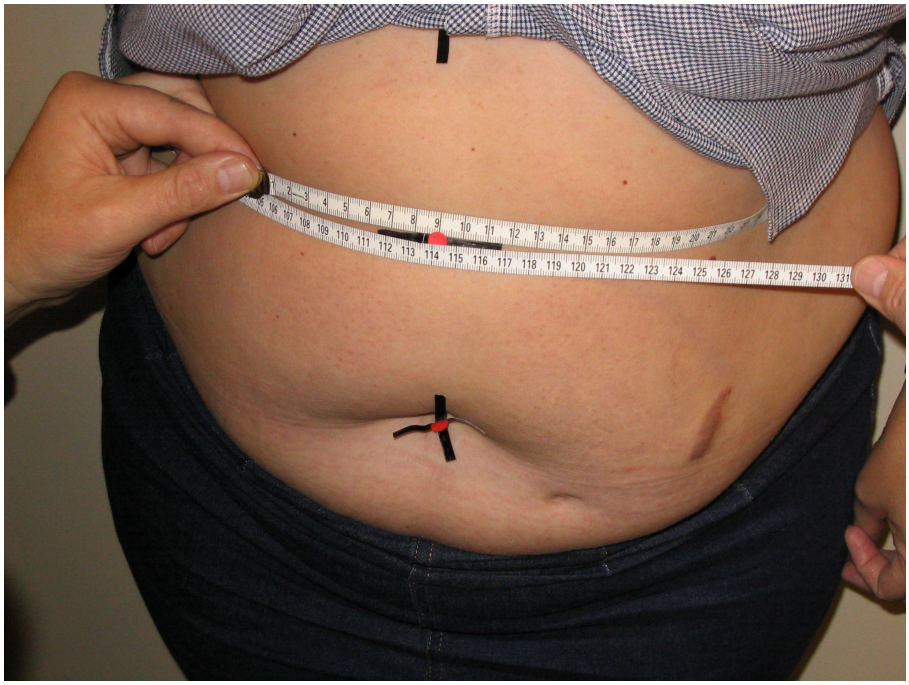


FIGURE 3.3: ABDOMINAL GIRTH AB^1

10. Abdominal (AB^2) – abdominal circumference at the umbilicus level

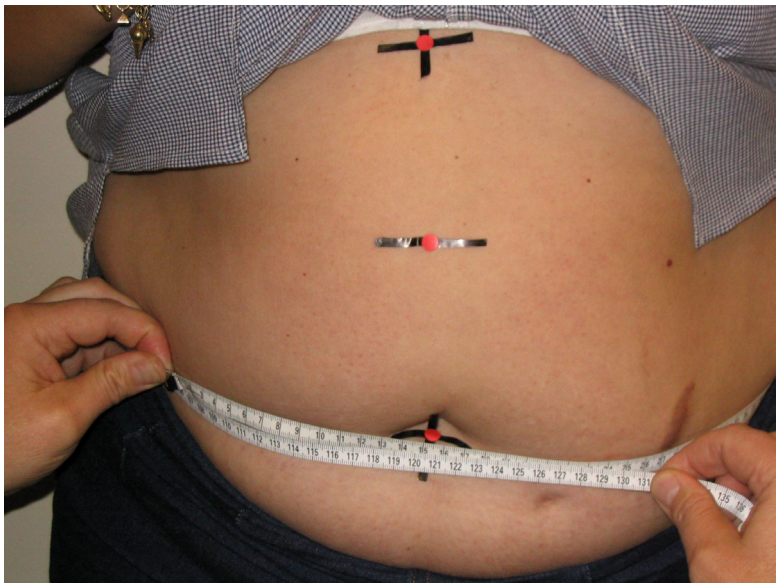


FIGURE 3.4: ABDOMINAL GIRTH AB^2

3.3.2 Morphology

3.3.2.1 Percentage Body Fat (BF)

Weltman's obesity-specific anthropometric equations for women aged 20 - 60 years using circumference measures rather than skinfolds, was employed to estimate percentage body fat (% BF). The advantage of using this method was that circumferenced could easily be measured, regardless of the subject's level of body fatness.

The equation is:

% BF = 0.11077 (ABC) – 0.17666 (HT) + 0.14354 (BW) + 51.03301, where
BW = body weight (kg); ABC (cm): average abdominal circumference = [(AB_1 + AB_2)/2], where AB_1 (cm) = abdominal circumference anteriorly midway between the xyphoid process of the sternum and the umbilicus and laterally between the lower end of the rib cage and iliac crest, and, AB_2 (cm) = abdominal circumference at the umbilicus level (Weltman et al., 1988).

3.3.2.2 Lean Body Mass (LBM)

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

$$\textbf{LBM = BM - ABF} \quad \textbf{and} \quad \textbf{ABF} = \frac{\textbf{RBF X BM}}{\textbf{100}}$$

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

3.3.2.3 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:

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

3.3.2.4 Body Surface Area

As originally developed by Du Bois & Du Bois (1916) the nomogram for calculating body surface area (BSA) in square meters (m^2) from height measured in centimetres (cm) and for calculating body weight, measured in kilograms is given in appendix G. The nomogram was used by placing one end of a ruler on the body weight and the other end on the body height. Where the ruler intersects the middle scale is the body surface area (Fox et al., 1993).

3.3.2.5 Waist-to-Hip Ratio

Waist-to-hip ratio (WHR) is strongly associated with visceral fat (Ashwell et al., 1985; Seidell et al., 1987) and appears to be an acceptable index of intra-abdominal fat (Jakicic, 1993).

The Anthropometric Standardization Reference Manual (Callaway et al., 1988) recommends measuring the waist circumference at the narrowest part of the torso and hip circumference at the level of the maximum extension of the buttocks. The WHR was established using the standardized measurement procedures described in the Anthropometric Standardization Reference Manual.

The WHR was simply calculated by dividing waist circumference (measured in cm) by hip circumference (measured in cm) (Heyward & Stolarczyk, 1996).

3.3.2.6 Somatotype

Heath and Carter have contributed extensively to the field of somatotyping for both men and women (Heath-Carter Anthropometric Somatotype) (Carter & Heath, 1990).

Ten variables were measured to calculate the anthropometric somatotype rating:

- Stature
- Body Mass

- Skinfolds
  - Triceps
  - Subscapular
  - Suprailiac
  - Medial Mid-Calf

- Bone Widths
  - Biepicondylar Humerus
  - Bicondylar Femur

- Limb Girths
  - Flexed Upper Arm
  - Calf (Carter & Heath, 1990).

Somatotyping describes the body type or physical classification of the human body using endomorphy, mesomorphy, and ectomorphy ratings, respectively.

Endomorphy

The first somatotype component is endomorphy and is characterized by roundness and softness of the body. In essence, endomorphy is the "fatness" component of the body. There is a smoothness of contours with no muscle relief.

Mesomorphy

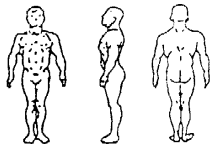
The second component is mesomorphy and is characterized by a square body with hard, rugged and prominent musculation. In essence, mesomorphy is the "muscle" component of the body.

Ectomorphy

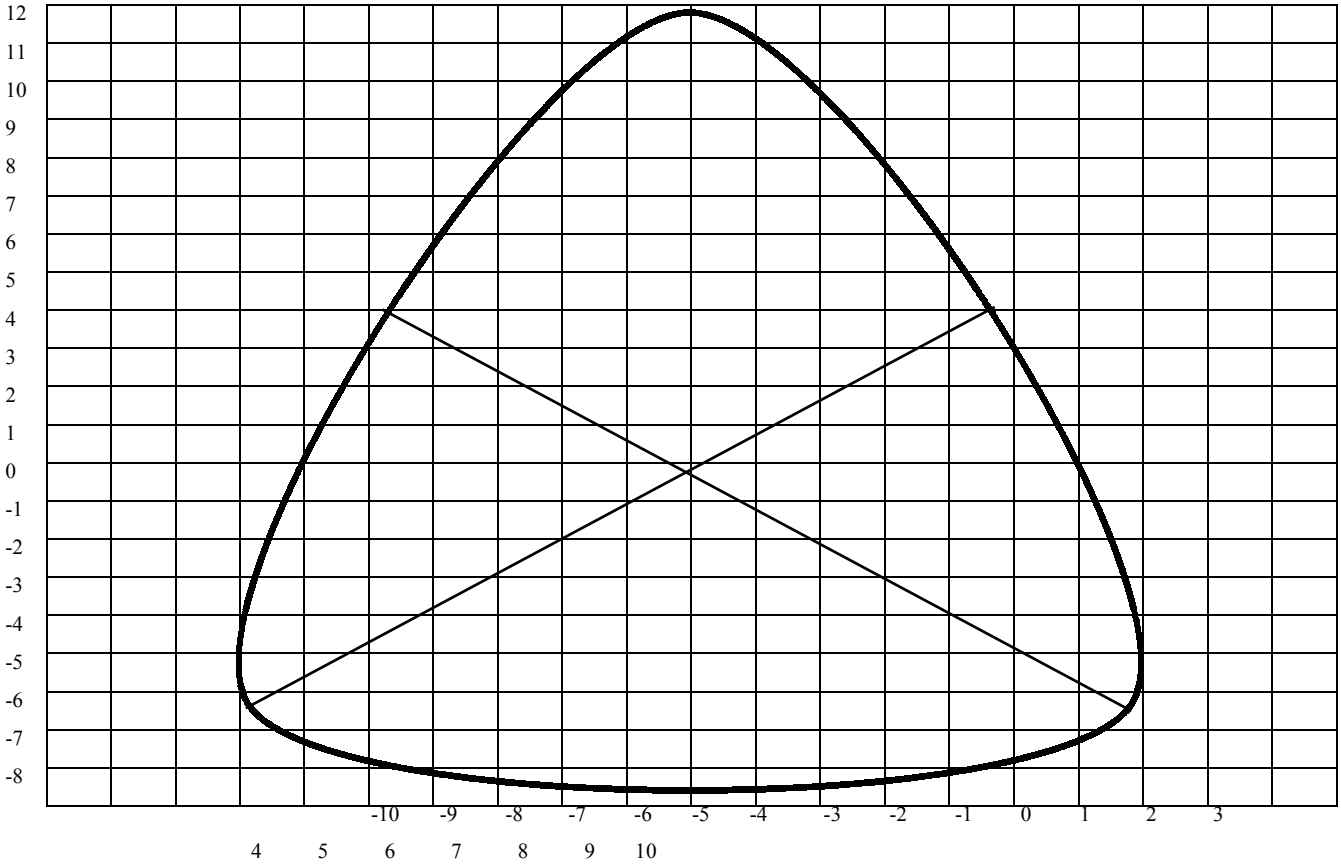
The third component, ectomorphy, includes as predominant characteristics linearity, fragility, and delicacy of body. This is the "leanness" component. The bones are small and the muscles thin. The limbs are relatively long and the trunk short; however, this does not necessarily mean that the individual is tall.

3.3.2.7 Somatogram

A somatogram is an anthropometric profile that graphically depicts subjects pattern of muscle and fat distribution (Carter, 1992). Somatograms may be especially useful for charting changes (pre-and post-test profiles) and monitoring progress of subject's involved in weight management (diet and exercise) programs (Heyward & Stolarczyk, 1996).

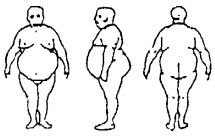


MESOMORPHY



ENDOMORPHY

ECTOMORPHY



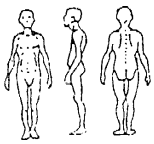


FIGURE 3.5: SOMATOGRAM

3.3.3 Ultrasound Sonography

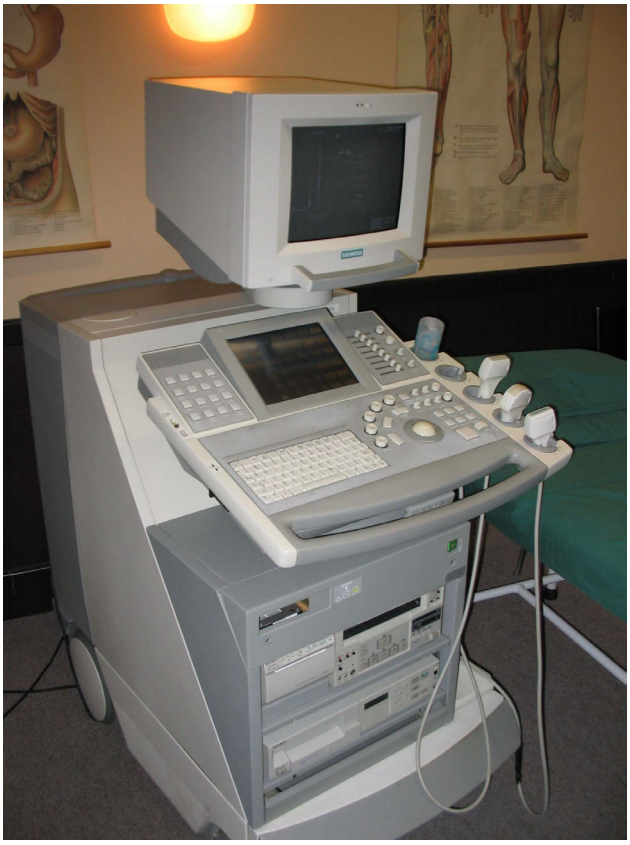


FIGURE 3.6: SIEMENS (SONOLINE ELLEGRA) SONOGRAPH

The sonographic measurements were conducted by a practicing specialist at the Jakaranda Hospital, Pretoria using a 3.5 MHz Siemens (Sonoline Ellegra) sonograph. Sonars were taken at a level 10 cm inferior to the xipho sternum on the xipho-umbilical line while the subjects were in a supine position with heels, buttocks and shoulders in contact with the examination table. The transducer was placed on the skin with minimal pressure and measurements were taken after normal expiration. Thicknesses were measured by using electronic callipers placed from leading edge to leading edge. The subcutaneous fat layer was measured from the skin to the M. rectus abdominus and the visceral fat layer (intra-abdominal fat) from the M. rectus abdominus to the anterior wall of the aorta.

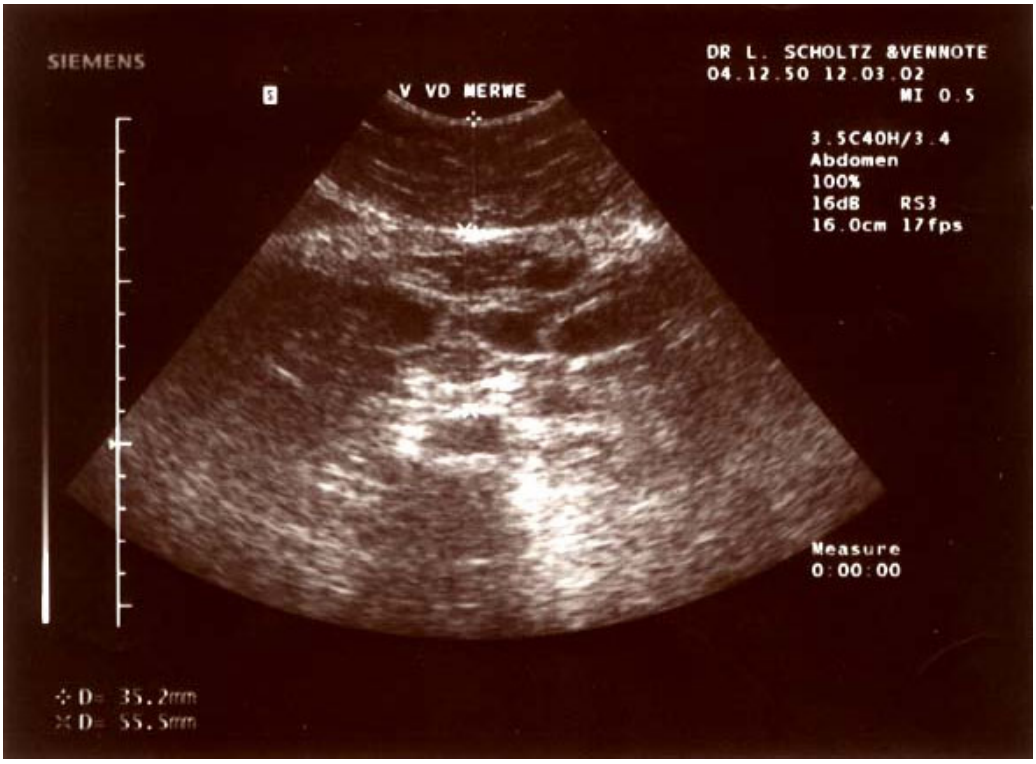


FIGURE 3.7: SONOGRAPHIC MEASUREMENT OF SUBCUTANEOUS AND INTRA-ABDOMINAL FAT

3.3.4 Respiratory Quotient (RQ)

Respiratory quotient was determined by an ambulatory metabolic measurement system (Aerosport KB1-C) with cognisance of the following variables: ambient temperature, barometric pressure, the subject's age, stature, body mass and gender. Subjects lay supine on plinth with the facemask covering nose and mouth. Subjects were asked to breathe in a normal relaxed manner. Respiratory quotient was measured after four minutes while subject was in a steady-state.

As respiratory gas was exhaled through the pneumotach (flow head) a micro sample, proportional to the expired flow, was drawn off through the centre (sample) line of the pneumotach into the base unit. A fixed rate of this proportional sample (known as a pulse) was drawn into a mixing system. For each pulse drawn in, a pulse of identical volume from the mixing system was emitted to the oxygen and carbon dioxide detectors. Over a fixed time period, electronic variable sampling (EVS), allows the

pulse trains to be reduced to a constant volume, resulting in similar equilibration times at varying expired flow rates. Following gas analysis and flow integration, the gas was exported out of the exhaust of the system to ambient air. The whole system was under microprocessor control. RQ was calculated according to standard procedures.

$$RQ = \frac{VCO_2}{VO_2}$$ (Cooper & Storer, 2001)

An important distinction must be made between the respiratory exchange ratio (R) which is a non-steady-state measure derived from instantaneous values of VCO_2 and VO_2 and respiratory quotient (RQ), which is normally derived from steady-state measures of VCO_2 and VO_2. If the metabolic substrate is purely carbohydrate, then the RQ value is 1.0. When the metabolic substrate is predominantly fat, the RQ approaches 0.7. Whole-body RQ represents the summation of many different organ system RQ values. Since VCO_2 and VO_2 both have units of liters per minute RQ has no units (Cooper & Storer, 2001).

3.3.5 Pulmonary Function

Lung volume and lung function was determined by a Schiller CS-200 Ergo-spirometer 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 trials were taken and the best result was recorded.

The following parameters were used:

FVC - Forced vital capacity indicating lung volume expressed in litres.

FEV_1 - Forced expiratory volume during the 1st second of FVC.

FEV_1 % - FEV_1/FVC * 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.

3.3.6 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
Triglycerides	:	0.8 – 1.5 mmol/ℓ
		(Tietz, 1994).

3.3.7 Cardiovascular Responses

3.3.7.1 Heart Rate

Heart Rate was measured with a Polar Accurex Plus coded transmitter and wrist receiver. The elastic strip was adjusted to fit the subject comfortably. The strap was secured around the subject's chest, with the transmitter on the xyphoid sternum. It

was checked that the moist electrode area was secured firmly against the subject's skin and the Polar logo on the transmitter was in a central upright position. After subjects had been lying in a supine position for five minutes on a plinth in a quiet room, heart rate readings were taken from the wrist receiver.

3.3.7.2 Blood Pressure

Blood pressure was measured after the subjects had been lying in a supine position, on a plinth, for five minutes in a quiet room. Measurements were taken with a Tycos sphygmomanometer and Littmann lightweight stethoscope. Blood pressure was taken at the brachial artery by the auscultatory method. Great care was taken that there was no tension in the arm muscles and the forearm was supported with the cubital fossa at heart level. Subjects used a normal adult cuff size. The sphygmomanometer was inflated until its pressure exceeded the systolic pressure within the artery. Blood flow was occluded and a brachial pulse (at the elbow fossa) could not be felt (palpated) or heard (auscultated). The pressure within the cuff was reduced by small increments and the examiner listened until korotkoff sounds were audible. The systolic pressure was the pressure exerted on the walls of the artery when the first soft tic sounds occurred. Diastolic pressure was referred to as the pressure in the artery when the korotkoff sounds were greatly muffled or had disappeared.

3.3.8 Musculoskeletal Function

3.3.8.1 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).

3.3.8.2 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 was expected 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 INDEPENDENT VARIABLES (INTERVENTION PROGRAMME)

3.3.1 Electrical Muscle Stimulation

FIGURE 3.8: SLIMLINE ELECTRICAL MUSCLE STIMULATION (EMS) MACHINE

Electrical muscle stimulation (EMS) was performed using a Slimline EMS machine. According to the suppliers, Slimline is a unique type of electro-medical apparatus, which ensures that, a perfectly balanced exchange of electrons are constantly flowing between each set of electro pads during treatment. A "Set of pads" consists of one (+) pad and one (-) pad both of which are plugged into one of the eight channels of the Slimline machine, thus providing a total of 16 pads. A greater sensation of contraction is experienced in the region of the (+) pad or positive electrode. This is considered normal and, necessary if treatment is to be effective (Slimline Promotional Literature, 2001).

Slimline slimming machines have three different electrokinetic modulations which perform their functions at varying exercise levels and muscle depths, working in gradual stages, from the deepest or basal muscles to the middle layers and finally to the surface muscle layers. The entire treatment session is controlled by Slimline's computer, which varies all the modulations, depth of therapy and exercise levels automatically. Pad placements are done according to pad placement charts, provided by the manufacturer. The complete pad placement chart is included in Appendix D.

Each subject used the machine for eight weeks, twice per week for a duration of 45 minutes per session. The training frequency was selected according to the manufacturer's recommendation of at least two training sessions per week for maximum results.

3.4.2 Thermogenic Stimulation

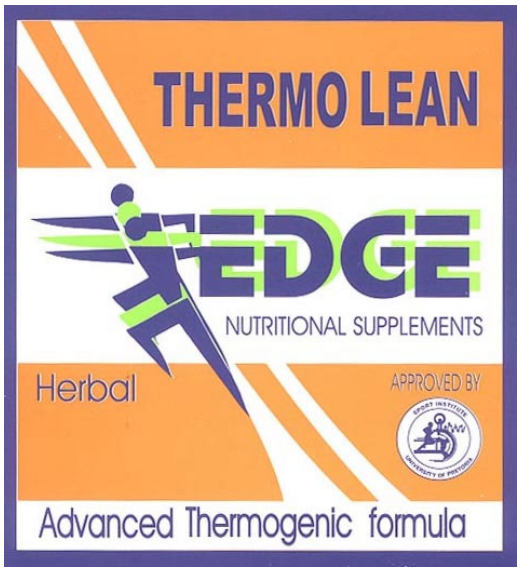


FIGURE 3.9: THERMO LEAN LABEL

Thermo Lean is an advanced thermogenic formula which was specifically prepared and capsulated for the study by a pharmaceutical laboratory (Biomox Pharmaceuticals Pty Ltd). It is a unique formulation of special extracts and herbs to nutritionally support the natural release and burning of stored body fat. Thermo Lean aids in a temporary, natural increase in body temperature (thermogenesis). Thermogenesis, when not simply needed for routine food digestion and metabolism, is both a source of heat and, when stimulated through appropriate dietary supplementation, a mechanism to increase metabolic rate. Thermo Lean (2001) is purported to accelerate caloric burning, enhances fat fuel utilization and increases metabolic rate.

Subjects received 200 light brown, odourless, tasteless capsules in a sealed white securitainer. Subjects ingested Thermo Lean capsules for 5> of the 7^{-} days per week with an initial dosage of four Thermo Lean capsules per day. The dosage was increased to six capsules per day in the 5^{th} week of the study. A total of 200 Thermo Lean capsules were consumed by each subject over 8 weeks with no contra-indications resulting. Subjects were instructed to maintain an adequate state of hydration while taking Thermo Lean capsules and not to take Thermo Lean capsules after 16:00 to counteract potential insomnia. Instructions, dosage and frequency of use were the same for the placebo capsules.

Composition per serving			
Thermo Lean		**Placebo**	
Carcinia Cambogia Extract	2000 mg	Cellulose	3415 mg
Gaurana Extract (Paullinia Cupana 22%)	910 mg	Collour Agent	250 mg
White Willow Bark	200 mg		
Yohambine 3% Extract	160 mg		
Citrus Aurantium (Citrus 6% Extract)	125 mg		
Acetyl L-Carnitine	100 mg		
L-Tyrosine	80 mg		
Ginger Root (Zingiber Officinale)	50 mg		
Calcium D Pantothenate	40 mg		
Chromium Polynicotinate	200 mcg		
Total	**3665.2 mg**		**3665 mg**

FIGURE 3.10: COMPOSITION OF THERMOGENIC AGENT AND PLACEBO

3.4.3 Standardized Diet Program

The standardized dietary intervention program, labelled 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 Metabolism Diet is included in Appendix E.

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.

  Subjects alternated between the low calorie (two weeks) and the booster phase (one-week). This was done in order to prevent potential plateaus in the subject's diet (slowing the metabolic rate).

- Re-entry phase

  When subjects got to within 2 to 3 kg from their target goal-weight, they switched to the re-entry phase. Subjects thus gradually increased their caloric consumption to reduce 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 viz.: 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.

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, and 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

An independent statistician was consulted and utilized for all statistical analyses. Standard descriptive statistics for central tendency (mean) and spread (standard deviation) were applied to all variables measured. Differences between pre- and post-test scores within the three experimental groups were determined by the Wilcoxon signed rank test. The Kruskal-Wallis test for three or more independent groups was adopted as the appropriate statistical technique for the between group inferential analysis of the data. This test is a non-parametric equivalent to a one-way analysis of variance (ANOVA) (Howel, 1992).

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 ($p \leq 0,05$), multiple comparisons were used to detect differences between two groups (TS vs. EST, EST vs. ESP etc.) using Scheffe and LSD (least sign difference) methods (Smit, 2002). All computations 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 electrical muscle stimulation (EMS) performed on Slimline Slimming Machines in conjunction with, and without, a thermogenic agent (Thermo Lean) and a standardized diet.

The results of the study are displayed in tabular (Tables 4.1a – 4.8b) and graphic form (Figures 4.1a - 4.8) and are reported in the following categories of dependent variables:

- Anthropometry
- Morphology
- Ultrasound Sonography
- Respiratory Quotient (RQ)
- Pulmonary Function
- Haematology
- Cardiovascular response
- Musculoskeletal Function

Henceforth each variable is presented with respect to responses within and between experimental groups (Tables 4.1a – 4.8b) with the latter also being depicted graphically, as relative differences in Figures 4.1a – 4.8. The variables are further discussed within the context of the relevant literature. It should be emphasized however, that there is a paucity of literature published specifically regarding the efficacy of electrical muscle stimulation (EMS). In this regard only two articles directly linked to this study, and a few indirectly related articles could be located, each publishing varing results.

In an early study, focussing on an obese population, Bailey (1976) conducted an in-depth study of the Hawkins Electrokinetic Body Activating (EBA) Machine to evaluate the safety and effectiveness of the device as a method of localized adipose tissue (fat) reduction and general weight-loss modality. In this trial a group of 40 moderately obese female patients with a mean age of 34-35 years, was selected to serve as subjects. The pre-posttesting battery included measurements of body weight, height, body surface area and appearance (via photographs).

Subjects were given a course of six weeks treatment (3 x one-hour sessions per week) followed by a four-weeks break with no specific instruction. They were then required to attend for a second six-weeks treatment. No attempt was made to regulate the patients diet or exercise during the intermediate four-week period, which was included as a balancing device to lessen any psychological halo effect.

Of the 30 subjects completing the trial, all lost weight in significant amounts (significant at 1% level). Weight-loss ranged from 2.2 kg to 21.5 kg with a mean weight-loss of 7.83 kg. Twenty subjects (50%) lost less than 7.83 kg, but even in this group significant losses were noted in specific tissue regions, such as the "upper arms", the abdomen and particularly the thighs. All patients showed a reduction in body surface area and all showed localized reduction in fat depot areas. There were no reported side effects of treatment, and all subjects expressed satisfaction with the weight and tissue losses achieved.

Abstinence from exercise often results in gradual deterioration in exercise capacity and muscle strength. Electrical muscle stimulation (EMS) is seen as a method of augmenting muscle performance. It has been shown to improve oxidative potential of the muscle (Bigard et al., 1993). Clinical trials in humans have shown that electrical muscle stimulation improve muscle strength and performance in patients with major knee ligament injuries (Wigerstad-Lossing et al., 1988), those who are immobile after surgery (Morrissey et al., 1985), promotes muscle growth in paraplegic patients (Buckley et al., 1987) and improves the performance of ischaemic muscles in patients with peripheral vascular disease (Tsang et al., 1994).

Bourjeily-Habr et al. (2002) noted that although exercise training improves exercise tolerance in most patients with obstructive pulmonary disease, some severely affected patients may be unable to tolerate it because of incapacitating breathlessness. This led the investigators to test whether electrical muscle stimulation of the lower extremities could improve muscle strength

and exercise tolerance in patients with moderate to severe chronic obstructive pulmonary disease. The investigator performed electrical muscle stimulation of the lower extremities for 20 minutes three times a week for six weeks in 18 medically stable patients aged 60 ± 1.5 years. The patients were divided into two equal groups who received either genuine or sham treatment. Quadriceps and hamstring muscle strength, exercise capacity and peak oxygen uptake were measured at baseline and at the end of the six weeks of stimulation. It was found that the muscle stimulation improved quadriceps function by 39.0 ± 20.4 percent in the treated patients compared with 9.0 ± 8.1 percent in the sham-treated group. The investigators also found that the muscle stimulation improved hamstring muscle strength by 33.9 ± 13.0 percent in the treated patients compared with 2.9 ± 4.7 percent in the controls. There were no significant changes in lung function, peak workload or peak oxygen consumption in either group.

Poor exercise capacity is also a common manifestation among the obese. The American College of Sports Medicine acknowledges that obese individuals could reap health benefits from exercise without demanding that the exercise meet the traditional intensity requirements suggested for weight-loss (American College of Sports Medicine, 1990). The Surgeon General's Report on Physical Activity and Health declared that physical activity need not be vigorous to improve health (US Department of Health and Human Services, 1996).

In cognisance of the foregoing, this study thus set out to determine whether or not, and to what extent, the advent of electrical muscle stimulation (EMS) could make a significant contribution to help the obese. In a more recent study related to this investigation, Porcari et al. (2002) recruited 27 college-aged volunteers to test the effectiveness of electrical muscle stimulation devices (EMS) on muscle strength, muscle tone, body weight, and body fat in healthy individuals. Volunteers were assigned to either an EMS (n = 16) or control group (n =11). The EMS group underwent electrical muscle stimulation 3 times per week following the manufacturer's recommendations, whereas the control group underwent sham stimulation sessions. The pre- and posttesting battery included measurements of body weight, body fat (via skinfolds), girths, isometric and isokinetic strength and appearance (via photographs from the anterior, side and posterior) EMS had no significant effect on any of the measured parameters. Claims relative to the effectiveness of EMS for the apparently healthy individual are not supported by the findings of this study.

4.1 ANTHROPOMETRY

The results indicating the response of anthropometric variables within and among the experimental groups are reflected in Tables 4.1a – 4.1h and Figures 4.1a – 4.1d.

4.1.1 Body girths

A significant decrease ($p \leq 0,05$) in all body girths was observed within all three groups. A total of ten girth measurements were taken, viz.

	Group EST	Group ESP	Group TS
Forearm	*		
Relaxed arm	*		
Contracted arm		*	
Chest	*		
Mid thigh		*	
Calf	*		
Hip		*	
Abdominal	*		
AB-1 ½ Umbi	*		
AB-2 Umbi		*	

(*Greatest reductions)

Six out of ten girth measurements showed the greatest reduction in group EST (thermogenic and electrical muscle stimulation following a standardized diet). The remaining girth measures had the greatest reduction in group ESP (electrical muscle stimulation, following a standardized diet and placebo controlled). There were no girth measures where group TS (thermogenic stimulation following a standardized diet) showed the greatest reduction.

If girths at all ten body sites were summed, group EST (thermogenic and electrical muscle stimulation following a standardized diet) had the greatest reduction (5.11%), followed by group ESP (electrical muscle stimulation, following a standardized diet and placebo controlled) with a reduction of 4.74% and then group TS (thermogenic stimulation following a standardized diet) with a 4.05% reduction. The greatest

decrease in the sum of body girths was observed in group EST but this decrease was not significantly greater ($p>0,05$) than decreases found in group ESP or group TS (Figure 4.16 and Table 4.1c & d). The same tendency that was found in the reduction in the sum of body girths was found in the reduction of body mass, body mass index, waist-to-hip ratio and body surface area between the three experimental groups. In all of the above mentioned variables group EST (thermogenic and electrical stimulation following a standardized diet) had the greatest reductions followed by group ESP (electrical muscle stimulation following a standardized diet and placebo controlled) and group TS (thermogenic stimulation following a standardized diet). The combination of diet, thermogenic and electrical muscle stimulation was the more successful intervention program because of the negative caloric balance created by the diet, enhanced metabolic rate as a result of the thermogenic agent and a combination of a negative caloric balance and enhanced metabolic rate induced by the electrical muscle stimulation.

There was a statistically significant difference between groups ($p\leq0,05$) in the reduction of body girths at three different body sites viz. abdominal, abdominal (AB-1), abdominal AB-2).

Coincidentally these three body sites included sites utilized for determing the relative body fat according to the technique of Weltman et al. (1988). Weight-loss is associated with a significant reduction in abdominal subcutaneous and visceral fat and the reduction in visceral fat is related to corresponding reduction in metabolic risk factors. The greater the weight-loss, the greater the reduction in abdominal fat (Ross, 1997). Accordingly reductions in body girths of the abdominal region were of great importance for this study. A reduction in abdominal body girths meant a reduction in percent body fat and this may relate to a reduction in metabolic risk factors.

Group EST (6.02%) had the greatest reduction in girth at the abdominal body site. This reduction was significantly ($p\leq0,05$) better than the reduction found in group ESP (4.79%) and group TS (4.69%).

Table 4.1a: Anthropometry : Body Girth Responses. Intra-Group Comparisons (NS =p > 0,05; ∗ p ≤ 0,05)

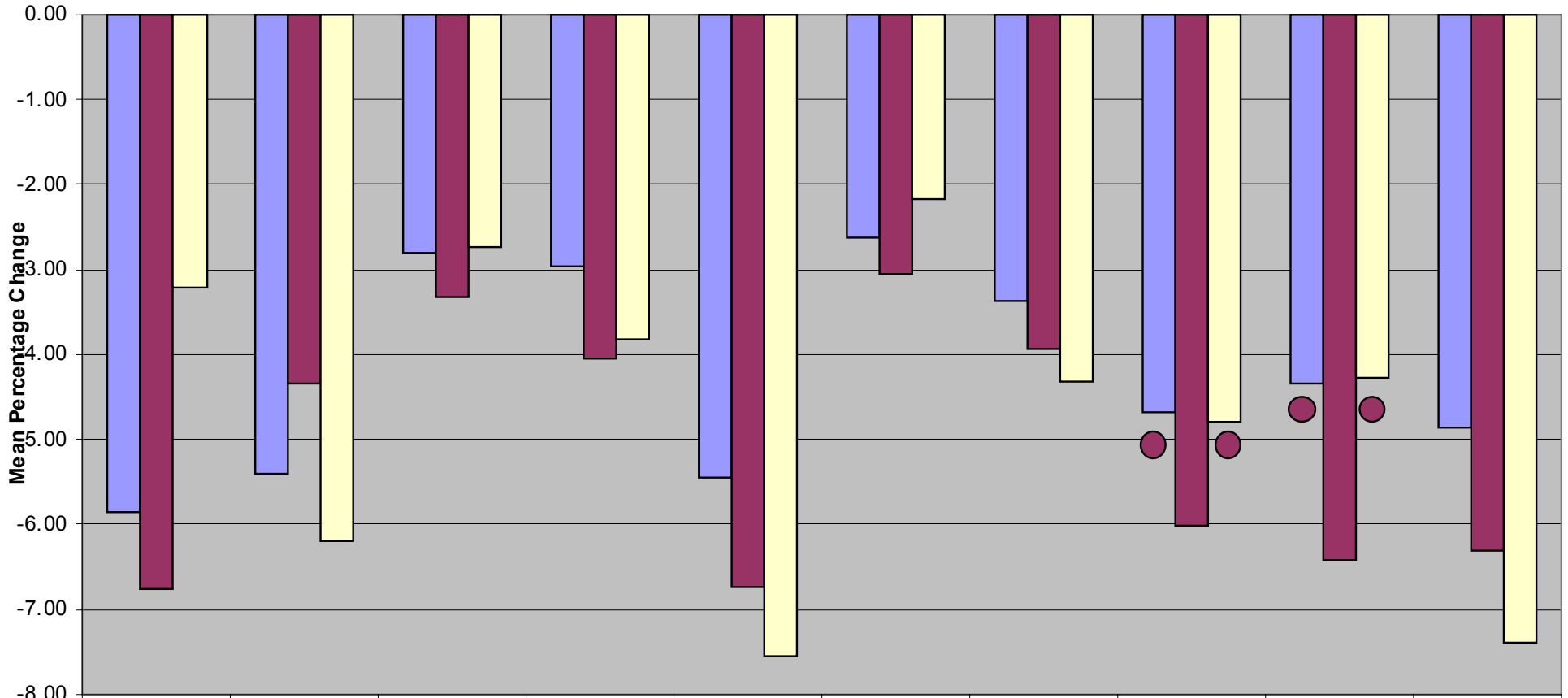
Groups		TS (n=20)						EST (n=20)						ESP (n=22)					
VARIABLES	UNITS	PRE Mean	Std. Dev.	POST Mean	Std. Dev.	Δ	p	PRE Mean	Std. Dev.	POST Mean	Std. Dev.	Δ	p	PRE Mean	Std. Dev.	POST Mean	Std. Dev.	Δ	p
Relaxed Arm	cm	35.48	6.12	33.40	5.69	-2.08	∗	33.37	4.61	31.11	3.66	-2.26	∗	34.56	5.31	33.45	4.99	-1.11	∗
Contracted Arm	cm	38.37	6.27	36.30	5.75	-2.07	∗	35.04	5.17	33.52	4.08	-1.52	∗	37.83	5.37	35.49	4.97	-2.34	∗
Forearm	cm	28.90	3.05	28.09	2.92	-0.81	∗	27.44	2.48	26.53	2.28	-0.91	∗	28.79	2.51	28.00	2.39	-0.79	∗
Chest	cm	112.63	13.48	109.29	13.84	-3.34	∗	108.68	8.88	104.27	8.15	-4.41	∗	115.09	13.18	110.70	11.54	-4.39	∗
Mid Thigh	cm	70.89	7.88	67.03	8.04	-3.86	∗	65.85	8.37	61.41	6.29	-4.44	∗	69.90	10.67	64.35	10.11	-5.25	∗
Calf	cm	42.53	4.35	41.41	4.07	-1.12	∗	40.13	4.54	38.90	4.03	-1.23	∗	42.73	5.17	41.80	5.15	-0.93	∗
Hip	cm	122.80	15.30	118.65	15.25	-4.15	∗	116.33	10.95	111.74	8.64	-4.59	∗	126.39	15.80	120.94	15.07	-5.45	∗
Abdominal	cm	97.03	14.35	92.48	14.21	-4.55	∗	91.50	10.35	85.99	8.02	-5.51	∗	99.63	15.39	94.86	13.71	-4.77	∗
AB–1 ½Umbi	cm	96.48	14.57	92.28	13.99	-4.20	∗	82.52	9.82	86.58	8.01	-5.94	∗	101.74	16.34	97.36	15.46	-4.35	∗
AB-2 Umbi	cm	106.95	20.89	101.76	20.59	-5.19	∗	101.32	13.81	94.93	10.35	-6.39	∗	114.08	18.64	105.65	14.94	-8.43	∗

Group TS ---Thermogenic stimulation and following a standardized diet.
Group EST ---Electrical muscle stimulation and thermogenic stimulation following a standardized diet.
Group ESP ---Electrical muscle stimulation and following a standardized diet (Placebo controlled).

Table 4.1b: Anthropometry : Body Girth Responses . Inter-Group Comparisons (NS =p > 0,05; ● p ≤ 0,05)

Groups		TS	EST	ESP	Significance		
VARIABLES		%Δ	%Δ	%Δ	TS vs EST	TS vs ESP	EST vs ESP
Relaxed Arm	cm	-5.68	-6.77	-3.21	NS	NS	NS
Contracted Arm	cm	-5.39	-4.34	-6.19	NS	NS	NS
Forearm	cm	-2.80	-3.32	-2.74	NS	NS	NS
Chest	cm	-2.97	-4.06	-3.81	NS	NS	NS
Mid Thigh	cm	-5.45	-6.74	-7.54	NS	NS	NS
Calf	cm	-2.63	-3.07	-2.18	NS	NS	NS
Hip	cm	-3.38	-3.95	-4.31	NS	NS	NS
Abdominal	cm	-4.69	-6.02	-4.79	●	NS	●
AB–1 ½Umbi	cm	-4.35	-6.42	-4.28	●	NS	●
AB-2 Umbi	cm	-4.85	-6.31	-7.39	NS	NS	NS

Group TS---Thermogenic stimulation and following a standardized diet.
Group EST ---Electrical muscle stimulation and thermogenic stimulation following a standardized diet.
Group ESP ---Electrical muscle stimulation and following a standardized diet (Placebo controlled).



	Relaxed Arm	Contracted Arm	Forearm	Chest	Mid Thigh	Calf	Hip	Abdominal	AB-1 ½Umbi	AB-2 Umbi
TS	-5.86	-5.39	-2.80	-2.97	-5.45	-2.63	-3.38	-4.69	-4.35	-4.85
EST	-6.77	-4.34	-3.32	-4.06	-6.74	-3.07	-3.95	-6.02	-6.42	-6.31
ESP	-3.21	-6.19	-2.74	-3.81	-7.54	-2.18	-4.31	-4.79	-4.28	-7.39

Figure 4.1a: Anthropometry : Body Girth Responses between Groups

Table 4.1c: Anthropometry : Sum of Body Girths Response. Intra-Group Comparisons (NS =p > 0.05; ∗ p ≤ 0.05)

Groups		TS (n=20)						EST (n=20)						ESP (n=22)					
VARIABLES	UNITS	PRE Mean	Std. Dev.	POST Mean	Std. Dev.	Δ	p	PRE Mean	Std. Dev.	POST Mean	Std. Dev.	Δ	p	PRE Mean	Std. Dev.	POST Mean	Std. Dev.	Δ	p
Girths	cm	794.63	99.91	762.41	98.45	-32.22	∗	754.11	69.79	715.54	53.11	-38.57	∗	813.17	100.25	774.63	90.30	-38.54	∗

Table 4.1d: Anthropometry : Sum of Body Girths Response. Inter-Group Comparisons (NS =p > 0,05; ● p ≤ 0,05)

Groups		TS	EST	ESP	Significance		
VARIABLES		%Δ	%Δ	%Δ	TS vs EST	TS vs ESP	EST vs ESP
Girths	cm	-4.05	-5.11	-4.74	NS	NS	NS

Group TS ---Thermogenic stimulation and following a standardized diet.
Group EST ---Electrical muscle stimulation and thermogenic stimulation following a standardized diet.
Group ESP ---Electrical muscle stimulation and following a standardized diet (Placebo controlled).

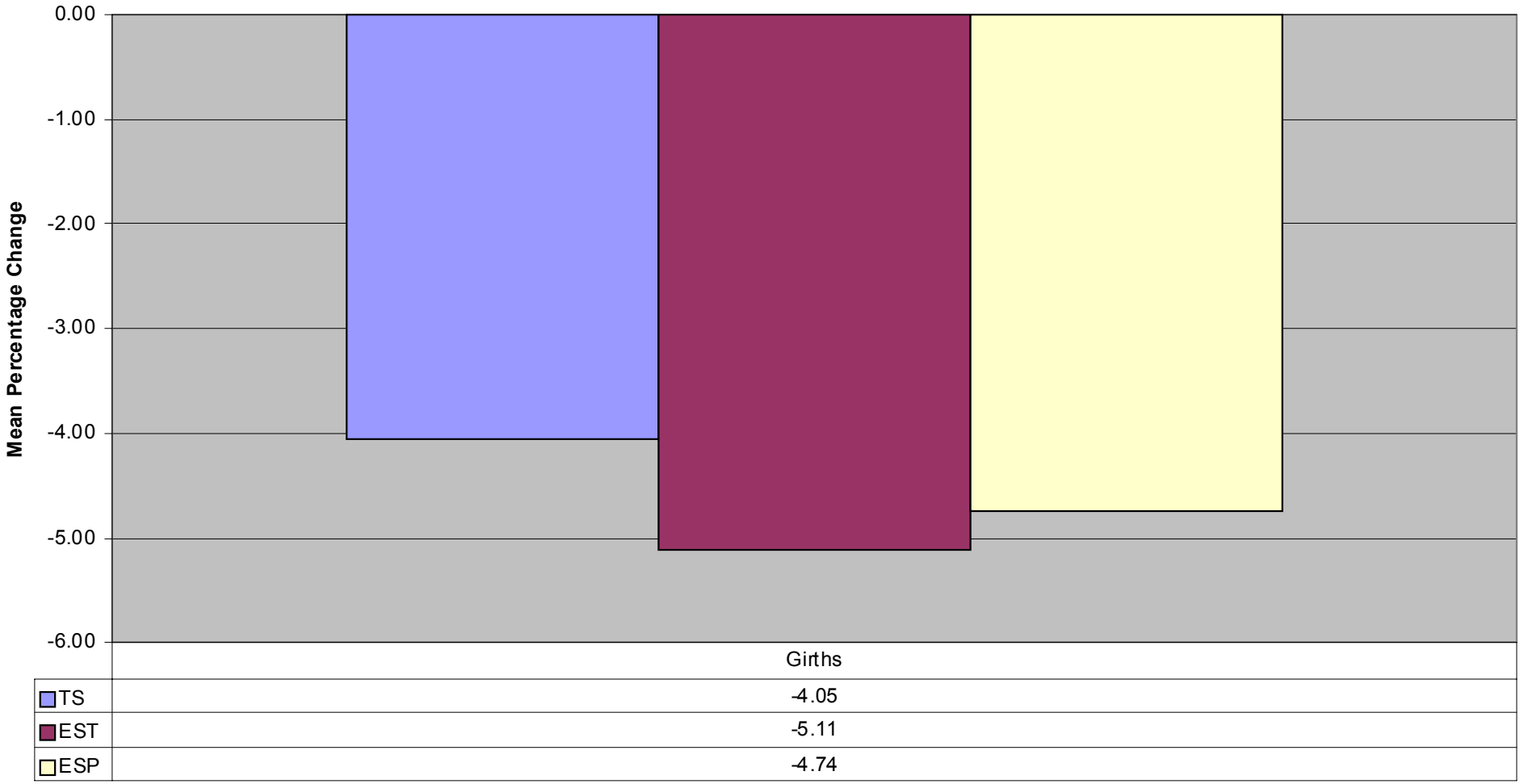


Figure 4.1b: Anthropometry : Sum of Body Girths Response between Groups

The same tendency was found at the abdominal AB-1 body site. Group EST (6.42%) had the greatest reduction in girth at the abdominal AB-1 body site. This reduction was significantly ($p \leq 0,05$) better than the reduction found in group TS (4.35%) and group ESP (4.28%).

Group ESP had the greatest reduction in girth at the umbilicus level (7.39%). This reduction was significantly ($p \leq 0,05$) better than the reduction found in group TS (4.85%) but not significantly ($p > 0,05$) better than the reduction found in group EST (6.31%). Interventions with EMS (electrical muscle stimulation) did better at the abdominal body sites than those without.

The biggest reduction in specific body girth measurements was found at the mid-thigh body site. Group ESP (7.54%) had the greatest reduction, but this was not significantly ($p > 0,05$) better than reductions found in group EST (6.74%) or group TS (5.45%), at the same site.

4.1.2 Skinfolds

A significant ($p \leq 0,05$) reduction in all skinfold measurements were observed within the three experimental groups. A total of seven skinfold measurements were taken, viz.

	Group EST	**Group ESP**	**Group TS**
Triceps	*		
Subscapular	*		
Suprailiac			*
Biceps	*		
Calf			
Abdominal			
Mid-thigh			*

(*Greatest reductions)

Table 4.1e: Anthropometry : Skinfold Responses. Intra-Group Comparisons (NS =p > 0,05; ∗ p ≤ 0,05)

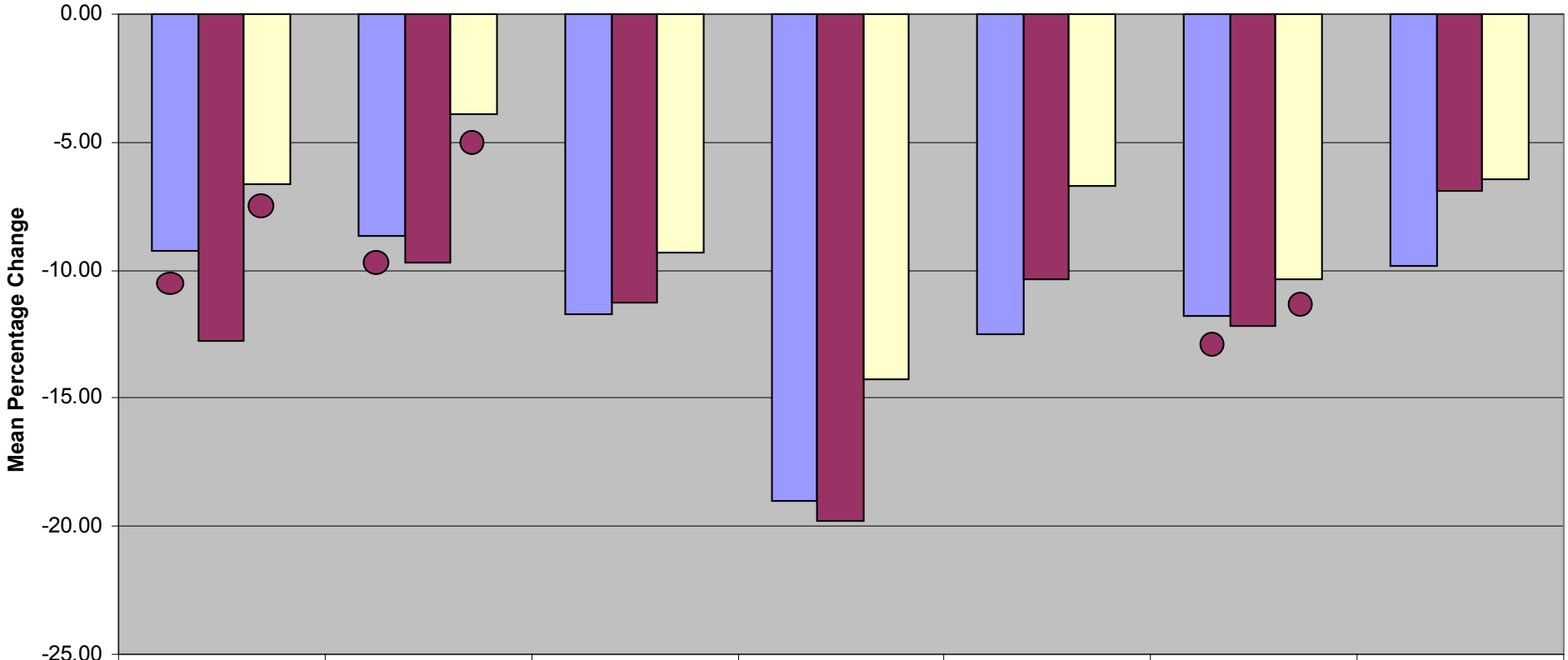
Groups		TS (n=20)						EST (n=20)						ESP (n=22)					
VARIABLES	UNITS	PRE Mean	Std. Dev.	POST Mean	Std. Dev.	Δ	p	PRE Mean	Std. Dev.	POST Mean	Std. Dev.	Δ	p	PRE Mean	Std. Dev.	POST Mean	Std. Dev.	Δ	p
Triceps	mm	34.29	9.06	31.11	8.75	-3.18	∗	30.35	11.59	26.48	7.46	-3.87	∗	37.12	11.28	34.66	10.59	-2.46	∗
Subscapular	mm	37.95	11.51	34.67	11.40	-3.28	∗	32.07	7.22	28.96	5.94	-3.11	∗	37.65	12.08	36.17	12.23	-1.48	∗
Suprailiac	mm	38.99	10.80	34.43	10.88	-4.56	∗	33.33	8.78	29.57	7.75	-3.76	∗	39.43	10.57	35.77	10.23	-3.66	∗
Biceps	mm	24.60	9.00	19.93	9.03	-4.67	∗	23.78	6.56	19.07	5.35	-4.71	∗	23.97	7.89	20.56	7.56	-3.41	∗
Calf	mm	34.00	12.16	29.75	11.83	-4.25	∗	32.83	9.22	29.43	7.93	-3.40	∗	39.25	10.93	36.61	12.37	-2.64	∗
Abdominal	mm	43.14	8.69	38.05	10.11	-5.09	∗	40.27	10.34	35.38	8.96	-4.89	∗	47.41	11.04	42.50	11.08	-4.91	∗
Mid-Thigh	mm	50.19	8.88	45.26	9.86	-4.93	∗	47.42	12.48	44.14	9.73	-3.28	∗	49.44	9.59	46.25	10.67	-3.19	∗

Group TS ---Thermogenic stimulation and following a standardized diet.
Group EST ---Electrical muscle stimulation and thermogenic stimulation following a standardized diet.
Group ESP ---Electrical muscle stimulation and following a standardized diet (Placebo controlled).

Table 4.1f: Anthropometry : Skinfold Responses. Inter-Group Comparisons (NS =p > 0,05; ● p ≤ 0,05)

Groups		TS	EST	ESP	Significance		
VARIABLES		%Δ	%Δ	%Δ	TS vs EST	TS vs ESP	EST vs ESP
Triceps	mm	-9.27	-12.75	-6.63	●	NS	●
Subscapular	mm	-8.64	-9.70	-3.93	●	NS	●
Suprailiac	mm	-11.70	-11.28	-9.28	NS	NS	NS
Biceps	mm	-18.98	-19.81	-14.23	NS	NS	NS
Calf	mm	-12.50	-10.36	-6.73	NS	NS	NS
Abdominal	mm	-11.80	-12.14	-10.36	●	NS	●
Mid-Thigh	mm	-9.82	-6.92	-6.45	NS	NS	NS

Group TS---Thermogenic stimulation and following a standardized diet.
Group EST ---Electrical muscle stimulation and thermogenic stimulation following a standardized diet.
Group ESP ---Electrical muscle stimulation and following a standardized diet (Placebo controlled).



	Triceps	Subscapular	Suprailiac	Biceps	Calf	Abdominal	Mid-thigh
TS	-9.27	-8.64	-11.70	-18.98	-12.50	-11.80	-9.82
EST	-12.75	-9.70	-11.28	-19.81	-10.36	-12.14	-6.92
ESP	-6.63	-3.93	-9.28	-14.23	-6.73	-10.36	-6.45

Figure 4.1c: Anthropometry : Skinfold Responses between Groups

In four out of seven skinfold measurements the greatest reduction was found in group EST (thermogenic and electrical muscle stimulation following a standardized diet). At the remaining three body sites group TS (thermogenic stimulation following a standardized diet) showed the greatest reduction. There were no body sites where group ESP (electrical muscle stimulation, following a standardized diet and placebo controlled) had the greatest reduction. The combination of diet, thermogenic and electrical muscle stimulation was the more successful intervention program when considering the skinfold response among groups.

There was a significant difference ($p \leq 0,05$) between groups in the reduction in skinfolds at three sites viz. triceps, sub-scapula, abdominal.

The greatest statistically significant difference ($p \leq 0,05$) in the reduction of skinfold measurements between groups was found at the tricep body site. Group EST had the greatest reduction (12.75%). This reduction was significantly ($p \leq 0,05$) better than the reduction found in both groups TS (9.27%) and ESP (6.63%).

The second greatest statistically significant difference ($p \leq 0,05$) in the reduction of skinfold measurementsbetween groups was found at the abdominal body site. Once again group EST had the greatest reduction (12.14%). This reduction was significantly ($p \leq 0,05$) better than the reduction found in both groups TS (11.80%) and ESP (10.36%).

The third greatest statistically significant difference ($p \leq 0,05$) in the reduction of skinfold measurements between groups was found at the subscapular body site. Again group EST had the greatest reduction (9.70%). This reduction was significantly ($p \leq 0,05$) better than the reduction found in both groups TS (8.64%) and ESP (3.93%).

The greatest reduction in all seven skinfolds that were measured was at the bicep site. Group EST had the greatest reduction (19.81%) followed by group TS (18.98%) and then group ESP (14.23%). None of these decreases between groups were significant ($p > 0,05$) however.

The observed significantly ($p \leq 0,05$) greater reduction in skinfold measurement at the abdominal body site in group EST is in accordance with a significantly ($p \leq 0,05$) greater reduction in girth measurements at the abdominal body sites in the same group. One of the most profound problem areas in all obese woman is the abdomen. The combination of diet, thermogenic and electrical muscle stimulation had the greatest effect in the reduction in skinfolds as well as girths in this problematic body area.

A number of investigators have tested the accuracy of population-specific (Durnin & Womersley, 1974) and generalized (Jackson & Pollock, 1978; Jackson et al., 1980) skinfold equations in overweight and obese samples. Overall, it appears that the SKF method have limited applicability to obese individuals.

The applicability of skinfolds methods in obese individuals is limited for the following reasons:

1. Site selection and palpation of bony landmarks are more difficult in obese individuals (Bray & Gray, 1988).

2. The skinfold thickness may be larger than the jaw aperture of most callipers, and it may not be possible to lift the skinfold from the underlying tissue in some obese clients (Gray et al., 1990).

3. There is greater variation in the depth at which the calliper tips can be placed on the SKF, and the calliper tips may slide on larger skinfolds (Bray & Gray, 1988).

4. Variability in adipose tissue composition may affect skinfolds compressibility in obese clients (Clarys et al., 1987).

5. There is greater variability among testers when measuring larger skinfold thickness (Bray & Gray, 1988).

4.1.3 Saggital height

Two saggital height measurements were made:

i) Saggital ½ umbi: The spirit level of the anthropometer was placed on the abdomen halfway between the xyphoid process and the umbilicus.

ii) Saggital umbi: The spirit level was placed on the umbilicus.

A significant ($p \leq 0,05$) decrease in saggital height measurements was observed within all three experimental groups at both body sites (saggital ½ umbi and saggital umbi). At the umbilicus body site (saggital umbi) group ESP (11.48%) had the greatest reduction. This reduction was similar to the reduction found in group EST (11.02%) but did not differ significantly ($p > 0,05$) from the reduction found in group TS (8.59%). At the saggital ½ umbi body site, group EST (13.52%) had the greatest reduction in saggital height. This reduction was significantly ($p \leq 0,05$) greater than that found in both groups ESP (10.61%) and TS (10.60%).

This significantly ($p \leq 0,05$) greater reduction in saggital height at the saggital ½ umbi body site in group EST, corresponds with the significant ($p \leq 0,05$) decreases found in body girths and skinfolds in the same group. Diet, thermogenic and electrical muscle stimulation (Group EST) thus had the greatest effect on the reduction in saggital height, skinfolds and girths at these problematic abdominal body sites.

The impact of regional fat distribution on health is related to the amount of visceral fat located within the abdominal cavity. The proliferation of research on body fat distribution and its relationship to disease has expanded at an exponential rate over the past 10 years, providing clear evidence that a link exists between increased abdominal fat and increased morbidity and mortality (Després, 1991). This research demonstrate strong associations between abdominal fat and diseases such as coronary artery disease (Donahue et al., 1987), diabetes (Björntorp, 1988), hypertension (Blair et al., 1984; White et al., 1986) and hyperlipidemia (Blair et al., 1984; Després et al., 1987).

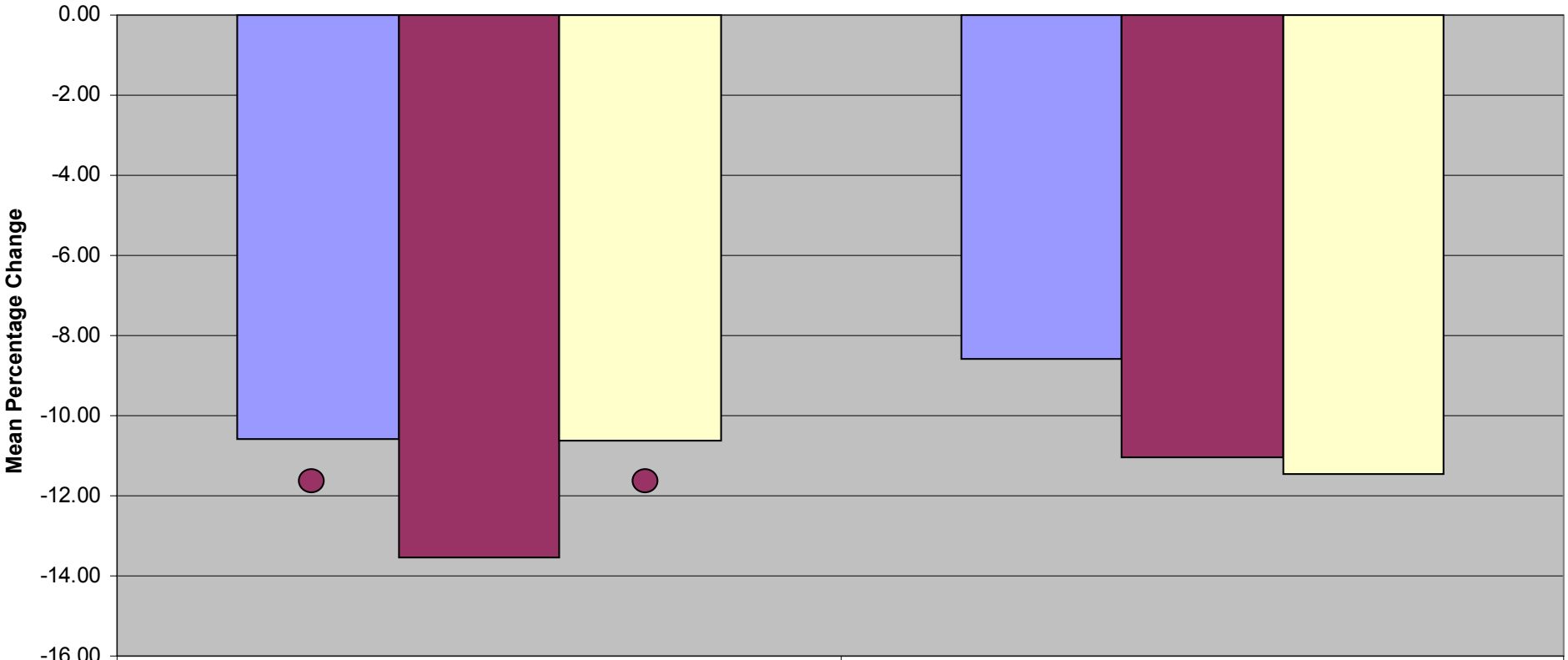
Table 4.1g: Anthropometry : Saggital Height Responses. Intra-Group Comparisons (NS =p > 0.05; ∗ p ≤ 0.05)

Groups		TS (n=20)						EST (n=20)						ESP (n=22)					
VARIABLES	UNITS	PRE Mean	Std. Dev.	POST Mean	Std. Dev.	Δ	p	PRE Mean	Std. Dev.	POST Mean	Std. Dev.	Δ	p	PRE Mean	Std. Dev.	POST Mean	Std. Dev.	Δ	p
Saggital ½Umbi	cm	24.34	4.54	21.76	4.57	-2.58	∗	22.63	2.88	19.57	1.62	-3.06	∗	24.50	3.89	21.90	3.69	-2.60	∗
Saggital Umbi	cm	22.46	4.29	20.53	3.76	-1.93	∗	21.59	3.26	19.2	1.88	-2.38	∗	24.05	4.06	21.29	3.79	-2.76	∗

Table 4.1h: Anthropometry : Saggital Height Responses. Inter-Group Comparisons (NS =p > 0,05; ● p ≤ 0,05)

Groups		TS	EST	ESP	Significance		
VARIABLES		%Δ	%Δ	%Δ	TS vs EST	TS vs ESP	EST vs ESP
Saggital ½Umbi	cm	-10.60	-13.50	-10.61	●	NS	●
Saggital Umbi	cm	-8.59	-11.00	-11.48	NS	NS	NS

Group TS ---Thermogenic stimulation and following a standardized diet.
Group EST ---Electrical muscle stimulation and thermogenic stimulation following a standardized diet.
Group ESP ---Electrical muscle stimulation and following a standardized diet (Placebo controlled).



	Saggital ½Umbi	Saggital Umbi
TS	-10.60	-8.59
EST	-13.52	-11.02
ESP	-10.61	-11.48

Figure 4.1d: Anthropometry : Saggital Height Responses between Groups

Anthropmetric indices can be used to classify individuals according to their type of obesity, such as upper body or abdominal obesity (high-risk) or lower-body obesity (low-risk). Numerous anthropometric measures are significantly related to intra-abdominal fat, viz. skinfold measure of the trunk (umbilicus, suprailiac, subscapular), waist and hip circumferences, the ratio of the waist-to-hip circumference (WHR), and body mass index (BMI). There are only two anthropometric indixes, BMI and WHR that are widely recognized for their ability to predict disease risk. These two indices have existing norms and standards for classifying individuals into high- or low-risk categories (Björntorp, 1993; Ciba Foundation, 1996).

If a positive correlation can be established between supine saggital abdominal height and visceral sonography (absolute measurement of intra-abdominal fat) then a link between increased saggital diameters and increased morbidity and mortality could be inferred among obese females.

4.2 MORPHOLOGY

The results indicating the response of morphological variables among the experimental groups are reflected in Tables 4.2a – 4.2f and Figures 4.2a – 4.2c.

4.2.1 Body mass

A significant ($p \leq 0,05$) decrease in body mass was observed within all three groups. The largest (6.43%) reduction in body mass was seen in those subjects on both thermogenic and electrical muscle stimulation following a standardized diet (Group EST) but this was not significantly greater ($p > 0,05$) than in those on thermogenic stimulation and following a standardized diet (Group TS = 5.72%) or those on electrical muscle stimulation and following a standardized diet (placebo controlled) (Group ESP = 5.10%).

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).

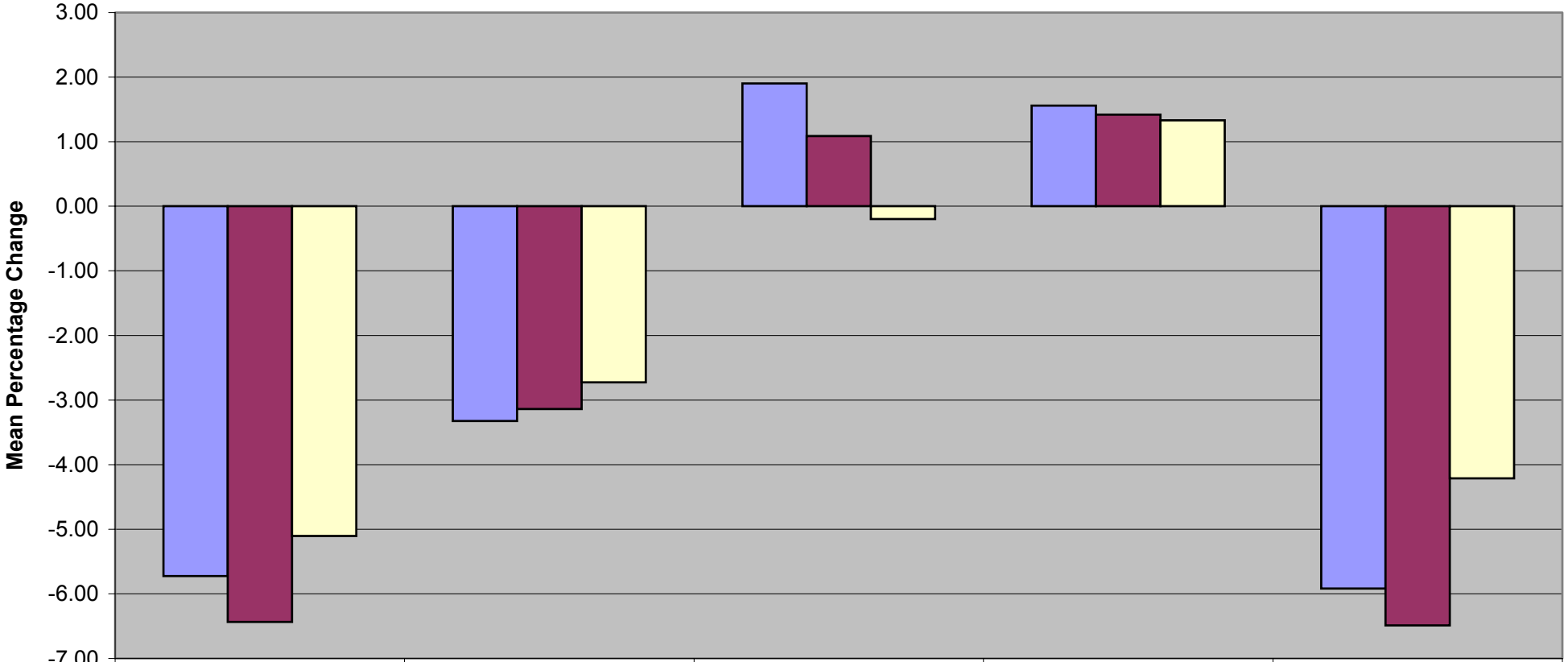
Table 4.2a: Morphological Responses. Intra-Group Comparisons (NS =p > 0,05; ∗ p ≤ 0,05)

Groups		TS (n=20)						EST (n=20)						ESP (n=22)					
VARIABLES	UNITS	PRE Mean	Std. Dev.	POST Mean	Std. Dev.	Δ	p	PRE Mean	Std. Dev.	POST Mean	Std. Dev.	Δ	p	PRE Mean	Std. Dev.	POST Mean	Std. Dev.	Δ	p
Body Mass	kg	98.53	22.13	92.89	20.41	-5.64	∗	89.99	17.00	84.20	13.94	-5.79	∗	100.12	24.08	95.01	23.16	-5.11	∗
Fat %	%	46.91	4.82	45.35	4.49	-1.56	∗	45.26	2.77	43.84	2.27	-1.42	∗	48.04	4.43	46.73	4.41	-1.31	∗
Muscle %	%	32.11	3.17	32.72	3.72	0.61	NS	32.24	2.87	32.59	2.65	0.35	NS	30.76	2.96	30.70	3.84	-0.06	NS
Lean Body Mass	%	53.09	4.82	45.65	4.49	1.56	∗	54.74	2.77	56.15	2.27	1.42	∗	51.68	4.55	53.01	4.55	1.33	∗
Body Mass Index	kg/m²	35.49	7.51	33.39	7.49	-2.10	∗	32.53	5.13	30.42	3.89	-2.11	∗	36.32	7.02	34.79	7.54	-1.53	∗

Table 4.2b: Morphological Responses. Inter-Group Comparisons (NS =p > 0,05; • p ≤ 0,05)

Groups		TS	EST	ESP	Significance		
VARIABLES		%Δ	%Δ	%Δ	TS vs EST	TS vs ESP	EST vs ESP
Body Mass	kg	-5.72	-6.43	-5.10	NS	NS	NS
Fat %	%	-3.33	-3.14	-2.73	NS	NS	NS
Muscle %	%	1.90	1.09	-0.20	NS	NS	NS
Lean Body Mass	%	1.56	1.42	1.33	NS	NS	NS
Body Mass Index	kg/m²	-5.92	-6.49	-4.21	NS	NS	NS

Group TS ---Thermogenic stimulation and following a standardized diet.
Group EST ---Electrical muscle stimulation and thermogenic stimulation following a standardized diet.
Group ESP ---Electrical muscle stimulation and following a standardized diet (Placebo controlled).



	Body Mass	Fat %	Muscle %	Lean Body Mass	Body Mass Index
TS	-5.72	-3.33	1.90	1.56	-5.92
EST	-6.43	-3.14	1.09	1.42	-6.49
ESP	-5.10	-2.73	-0.20	1.33	-4.21

Figure 4.2a: Morphological Responses between Groups

The negative caloric balance created through diet plus thermogenic stimulation (Group TS), diet plus thermogenic and electrical muscle stimulation (Group EST) or diet plus electrical muscle stimulation, placebo controlled (Group) all significantly contributed to a reduction in body mass.

The combination of diet, thermogenic and electrical muscle stimulation (Group EST) tended to be the more successful intervention program because of the negative caloric balance induced by the diet, and the enhanced metabolic rate as a result of the thermogenic agent and electrical muscle stimulation combined.

4.2.2 Percentage body fat (BF)

A significant ($p \leq 0,05$) decrease in percentage body fat was observed within all three groups. Group TS (3.33%) and group EST's (3.14%) reduction in percentage body fat were the highest but not significantly ($p > 0,05$) greater than the other modality, ESP (2.73%). The previously observed reduction in absolute body mass in groups TS, EST and ESP can thus be ascribed to the reduction of body fat in these groups.

The negative caloric balance created through diet plus a thermogenic agent (Group TS), diet plus thermogenic and electrical muscle stimulation (Group EST) or diet plus electrical muscle stimulation, placebo controlled (Group ESP) significantly contributed to the reduction in percentage body fat.

4.2.3 Percentage muscle

No significant ($p > 0,05$) changes were observed within groups. The greatest increase in percentage muscle was found in group TS (1.90%) followed by group EST (1.09%). None of these two increases were significantly greater ($p > 0,05$) than the reduction found in group ESP (-0.20%).

There were an inverted correlation between percentage muscle gained and percentage fat loss in all three groups. Group TS had the highest percentage fat loss (3.33%) and the greatest gain in muscle percentage (1.90%) followed by group EST with a 3.14% fat loss and a 1.09% gain in muscle percentage. Group ESP had the lowest percentage of fat loss (2.73%) and a loss in muscle mass (-0.20%).

4.2.4 Lean body mass

An increase in lean body mass (LBM) was observed in all three groups. The greatest (1.56%) increase in lean body mass, was seen in those subjects on thermogenic stimulation following a standardized diet (Group TS) but this was not significantly greater ($p>0,05$) than those on both thermogenic and electrical muscle stimulation following a standardized diet (Group EST = 1.42%) or those on electrical muscle stimulation following a standardized diet, placebo controlled (Group ESP = 1.33%)

Lean body mass was calculated as an anthropometric variable of body composition. The previously observed reduction in absolute body mass and fat percentage in groups TS, EST and ESP can thus be ascribed to the increase in lean body mass in all three groups. The negative caloric balanced created through diet plus a thermogenic agent (Group TS), diet plus thermogenic and electrical muscle stimulation (Group EST) or diet plus electrical muscle stimulation, placebo controlled (Group ESP) significantly contributed to the increase in lean body mass.

4.2.5 Body mass index

A significantly reduced ($p\leq0,05$) BMI was observed within all groups. As in the case of the related decrease in body mass the greatest reduction in BMI was in group EST (6.49%) but this reduction was not significantly ($p>0,05$) better than in group TS (5.92%) or group ESP (4.21%). The negative caloric balance created through diet plus the increase in metabolism created by the thermogenic agent in conjunction with the electrical muscle stimulation (exercise) had the greatest effect on the reduction of BMI (Group EST).

4.2.6 Waist-to-hip ratio

A significantly reduced ($p\leq0,05$) waist-to-hip ratio (WHR) was observed within two of the three experimental groups. The greatest reduction was found in group EST (2.53%) and this reduction was significantly ($p\leq0,05$) better than the reduction found in group TS (1.27%) and group ESP (1.27%).

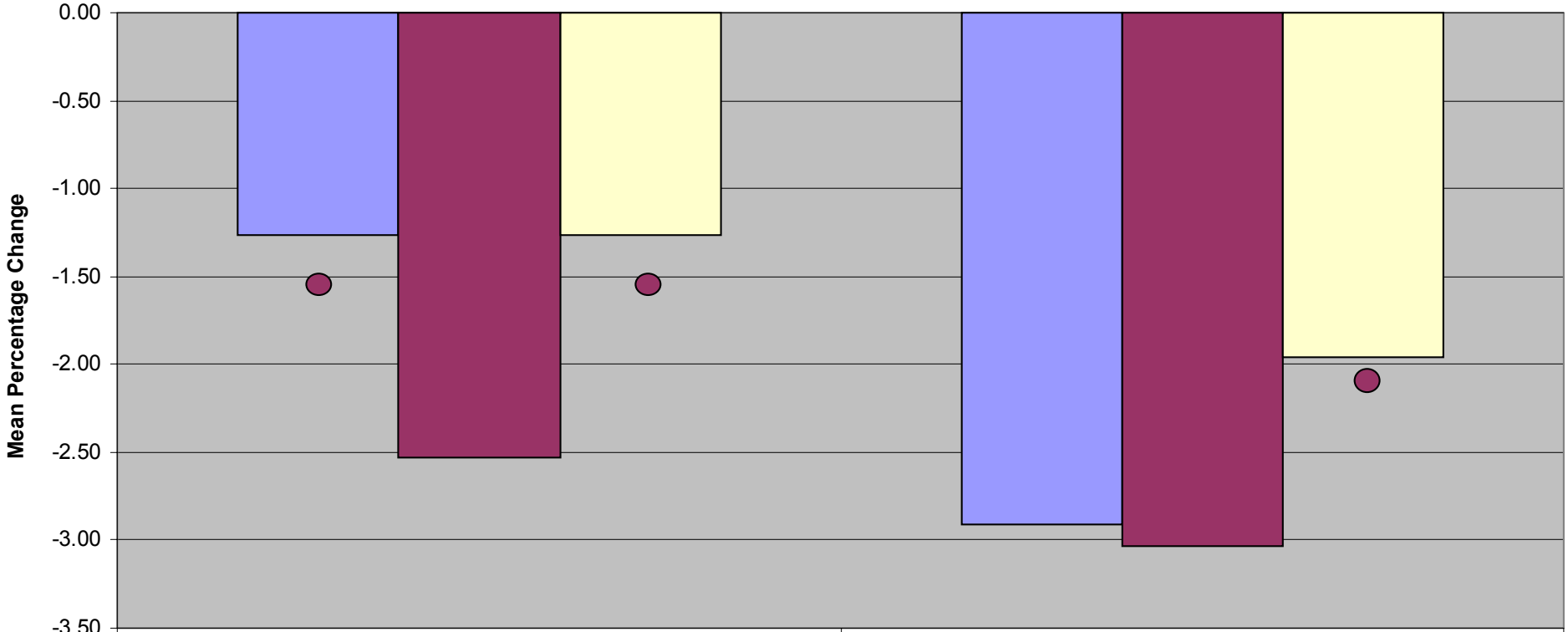
Table 4.2c: Waist-to-Hip Ratio and Body Surface Area Responses. Intra-Group Comparisons (NS =p > 0.05; ∗ p ≤ 0.05)

Groups		TS (n=20)						EST (n=20)						ESP (n=22)					
VARIABLES	UNITS	PRE Mean	Std. Dev.	POST Mean	Std. Dev.	Δ	p	PRE Mean	Std. Dev.	POST Mean	Std. Dev.	Δ	p	PRE Mean	Std. Dev.	POST Mean	Std. Dev.	Δ	p
Waist-to-Hip Ratio	N/A	0.79	0.06	0.78	0.06	-0.01	∗	0.79	0.06	0.77	0.06	-0.02	∗	0.79	0.07	0.78	0.06	-0.01	NS
Body Surface Area	m²	2.06	0.19	2.00	0.19	-0.06	∗	1.98	0.19	1.92	0.18	-0.06	∗	2.04	0.22	2.00	0.24	-0.04	∗

Table 4.2d: Waist-to-Hip Ratio and Body Surface Area Responses. Inter-Group Comparisons (NS =p > 0,05; ● p ≤ 0,05)

Groups		TS	EST	ESP	Significance		
VARIABLES		%Δ	%Δ	%Δ	TS vs EST	TS vs ESP	EST vs ESP
Waist-to-Hip Ratio	N/A	-1.27	-2.53	-1.27	●	NS	●
Body Surface Area	m²	-2.91	-3.03	-1.96	NS	NS	●

Group TS ---Thermogenic stimulation and following a standardized diet.
Group EST ---Electrical muscle stimulation and thermogenic stimulation following a standardized diet.
Group ESP ---Electrical muscle stimulation and following a standardized diet (Placebo controlled).



	Waist-to-Hip Ratio	Body Surface Area
TS	-1.27	-2.91
EST	-2.53	-3.03
ESP	-1.27	-1.96

Figure 4.2b: Waist-to-Hip Ratio and Body Surface Area Responses between Groups

The statistically significant reduction ($p \leq 0,05$) found in group EST (2.53%) indicates that a combination of diet, electrical muscle stimulation and a thermogenic agent had the greatest effect on WHR. WHR is strongly associated with visceral fat (Ashwell et al., 1985; Seidell et al., 1987) and appears to be an acceptable index of intra-abdominal fat (Jakicic, 1993). Certain predictions about health-related comorbidities can be made by using WHR (Van Itallie, 1985). Although an abdominal fat distribution was proposed as a health hazard for coronary heart disease in the 1940's for the first time, it took a long time before it was confirmed (Després et al., 1995). Confirmation only came when various studies showed that a simple anthropometric measurement such as the waist-to-hip ratio (WHR) correlated with insulin resistance, hyperinsulinaemia, dyslipidaemia, hypertension and CHD (Després et al., 1990; Gillum, 1987; Lapidus et al., 1984). A study on females undergoing coronary angiography, showed that the pattern of their body fat distribution not only correlated with metabolic and hormonal cardiovascular risk factors, but that those with major coronary stenoses in fact had a significantly higher WHR than a healthy control group (Hauner et al., 1994). These observations suggest that strategies designed to reduce obesity would be enhanced if abdominal obesity, in particular visceral fat, was substantially reduced. It is well established that diet-induced weight-loss is associated with a significant reduction in abdominal subcutaneous and visceral fat and that reduction in visceral fat is related to a corresponding reduction in metabolic risk factors. The greater the diet-induced weight-loss, the greater the reduction in abdominal fat, with a 10% weight-loss corresponding to a 30 to 35% reduction in visceral fat (Ross, 1997).

Few studies have examined whether exercise-induced weight-loss is associated with concomitant reduction in abdominal subcutaneous and/or visceral fat. Using waist circumference as a surrogate measure of abdominal obesity, it is reported that minor reductions (2 cm) in waist circumference are observed in response to exercise-induced weight-loss in the order of 2 to 3 kg (Ross et al., 2000).

No other studies have examined the influence of a combination of diet, electrical muscle stimulation and thermogenic stimulation on weight-loss and especially abdominal fat loss. In this study it is reasonable to conclude that a combination of diet, electrical muscle stimulation and thermogenic stimulation led to a decrease in waist-to-hip ratio and could have positive effects on certain health aspects.

4.2.7 Body surface area

A significant decrease ($p \leq 0,05$) in body surface area (BSA) was observed within all three groups. The largest (3.03%) reduction in BSA was seen in those subjects on both thermogenic and electrical muscle stimulation following a standardized diet (Group EST) and this reduction was significantly greater ($p \leq 0,05$) than the group on electrical muscle stimulation following a standardized diet, placebo controlled (Group ESP = 1.96%). There was no significant difference between group EST (3.03%) and group TS (2.91%). The combination of a standardized diet and the thermogenic agent (Group TS = 2.91%) had a far greater effect albeit statistically insignificant ($p > 0,05$) on body surface area reduction than a standardized diet and electrical muscle stimulation (Group ESP = 1.96%).

4.2.8 Somatotype

Somatotype deals with the body type or physical classification of the human body. The terms endomorph, mesomorph, and ectomorph are used to describe a person in terms of his or her somatotype.

The results indicating somatotype responses among the experimental groups are reflected in Table 4.2e & f and Figure 4.2c.

4.2.8.1 Endomorphy (Somatotype I)

The first component (somatotype I) is endomorphy and is characterized by roundness and softness of the body. In ordinary language, endomorphy is the "fatness" component of the body. A reduction in this component was of importance in this study. Significant decreases ($p \leq 0,05$) were observed in all three groups. The largest (6.98%) reduction in the endomorph component was seen in those subjects on both thermogenic and electrical muscle stimulation following a standardized diet (Group EST) but this was not significantly greater ($p > 0,05$) than those on a thermogenic stimulation and following a standardized diet (Group TS = 6.94%) or those on electrical muscle stimulation and following a standardized diet, placebo controlled (Group ESP = 3.97%). Group EST (6.98%) and group TS (6.94%) had a similar percentage reduction in somatotype I (endomorphy). This shows the superiority of

interventions with a thermogenic agent following a standardized diet (Groups EST and TS) over group ESP with only electrical muscle stimulation and following a standardized diet. This reduction found in somatotype I (fatness component) correlates with the reduction found earlier with body mass and percentage body fat in all three study groups.

All of the subjects included in this study were of endomorphic build. Features of this build are a predominance of abdomen over thorax, high square shoulders, and short neck. There is a smoothness of contours throughout, with no muscle relief.

4.2.8.2 Mesomorphy (Somatotype II)

The second component is mesomorphy (somatotype II) and is characterized by a square body with hard, rugged and prominent musculation. The bones are large and covered with thick muscles. Legs, trunks, and arms are usually massive in bone and heavily muscled throughout. Outstanding characteristics of this type are forearm thickness and heavy wrists, hands and fingers. The thorax is large and the waist is relatively slender.

In ordinary language mesomorphy is the "muscle" component of the body. It was suspected that a decrease in the endomorphic component could have led to a decrease in the mesomorph component as well. A decrease in skinfold and increase in girth measurements usually results in the increase in somatotype II (mesomorph).

A significant decrease ($p \leq 0,05$) in somatotype II (mesomorphy) was observed within all three groups. The reduction in somatotype II (mesomorphy) was similar in all three the groups with group EST (6.97%) followed by group ESP (6.53%) and then group TS (6.14%). The electrical muscle stimulation (Group EST and ESP) had no increased effect on somatotype II (mesomorphy) and this raises the question if it can be used as and substitute for active training.

4.2.8.3 Ectomorphy (Somatotype III)

The third component ectomorphy (somatotype III) includes as predominant characteristics linearity, fragility, and delicacy of body. The bones are small and th

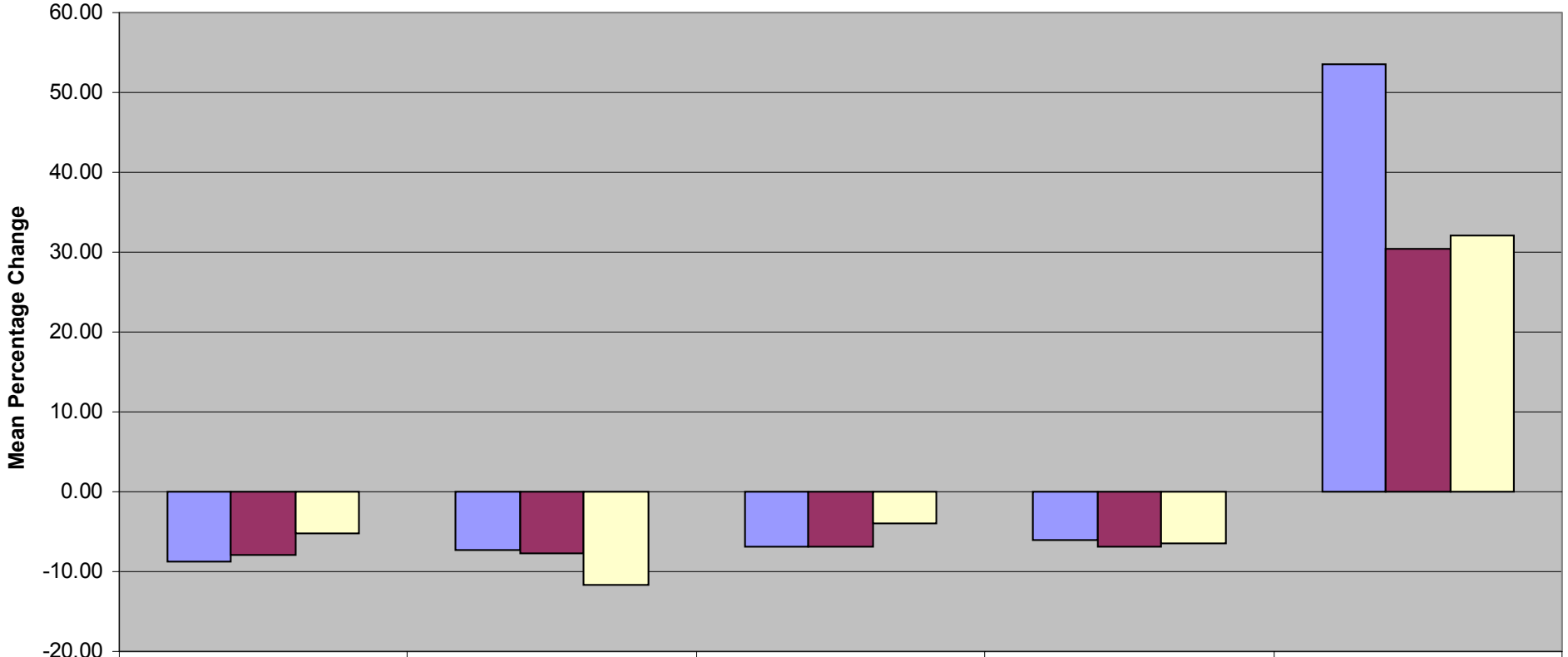
Table 4.2e: Somatotype Responses. Intra-Group Comparisons (NS =p > 0,05; ∗ p ≤ 0,05)

Groups		TS (n=20)						EST (n=20)						ESP (n=22)					
VARIABLES	UNITS	PRE Mean	Std. Dev.	POST Mean	Std. Dev.	Δ	p	PRE Mean	Std. Dev.	POST Mean	Std. Dev.	Δ	p	PRE Mean	Std. Dev.	POST Mean	Std. Dev.	Δ	p
Somatotype X	N/A	-8.53	1.49	-7.79	2.06	-0.74	∗	-7.80	1.44	-7.19	1.21	-0.61	∗	-8.66	1.75	-8.2	2.09	-0.45	∗
Somatotype Y	N/A	6.87	4.58	6.37	4.35	0.50	∗	5.17	2.80	4.77	2.79	-0.40	∗	6.17	3.69	5.45	3.56	-0.72	∗
Endomorph	N/A	8.93	1.24	8.31	1.52	-0.62	∗	8.17	1.24	7.60	1.02	-0.57	∗	9.07	1.49	8.71	1.65	-0.36	∗
Mesomorph	N/A	7.98	2.58	7.49	2.46	-0.49	∗	6.74	1.65	6.27	1.47	-0.47	∗	7.66	2.24	7.16	2.15	-0.50	∗
Ectomorph	N/A	0.28	0.45	0.43	0.72	0.15	∗	0.23	0.24	0.30	0.28	0.07	∗	0.25	0.38	0.33	0.57	0.08	∗

Table 4.2f: Somatotype Responses. Inter-Group Comparisons (NS =p > 0,05; • p ≤ 0,05)

Groups		TS	EST	ESP	Significance		
VARIABLES		%Δ	%Δ	%Δ	TS vs EST	TS vs ESP	EST vs ESP
Somatotype X	N/A	-8.68	-7.82	-5.2	NS	NS	NS
Somatotype Y	N/A	-7.28	-7.14	-6.98	NS	NS	NS
Endomorph	N/A	-6.94	-6.98	-3.97	NS	NS	NS
Mesomorph	N/A	-6.14	-6.97	-6.53	NS	NS	NS
Ectomorph	N/A	53.57	30.43	32.00	NS	NS	NS

Group TS ---Thermogenic stimulation and following a standardized diet.
Group EST ---Electrical muscle stimulation and thermogenic stimulation following a standardized diet.
Group ESP ---Electrical muscle stimulation and following a standardized diet (Placebo controlled).



	Somatotype X	Somatotype Y	Endomorph	Mesomorph	Ectomorph
TS	-8.68	-7.28	-6.94	-6.14	53.57
EST	-7.82	-7.74	-6.98	-6.97	30.43
ESP	-5.20	-11.67	-3.97	-6.53	32.00

Figure 4.2c: Somatotype Responses between Groups

muscles thin. This is the "leanness" component. All subjects used in this study (BMI ≥ 30) were directly placed in the extreme opposite of this somatotype III (ectomorphy). A reduction in the "fatness" component would have meant an increase in the "leanness" component.

A significant increase ($p \leq 0,05$) in somatotype III (ectomorphy) was observed in all experimental groups. The greatest increase in somatotype III (ectomorphy) was found in group TS (53.57%) followed by groups ESP and EST with similar percentage increases (Group ESP = 32.00% and Group EST = 30.43%). Although the greatest increase was observed in group TS (thermogenic stimulation following a standardized diet) this increase was not significantly greater ($p > 0,05$) than increases found in group ESP (electrical muscle stimulation following a standardized diet, placebo controlled) or group EST (thermogenic and electrical muscle stimulation following a standardized diet). This increase in somatotype III (ectomorphy) correlates with the percentage increase in muscle mass found in group TS. The largest increase in percentage muscle (Group TS = 1.90%) and the greatest decrease in percentage fat (Group TS = 3.33%) lead to the biggest difference in somatotype III (Group TS = 53.57%).

4.2.8.4 Somatogram

A somatogram was used as an anthropometric profile that graphically depicts the subjects pattern of muscle and fat distribution. Somatograms were especially useful for charting changes (pre- and post-test profiles) and monitoring progress of subjects involved in this weight-loss study. Somatotyping has also been used to describe the type of physique that was most susceptible to various diseases. The Heath-Carter anthropometric somatogram comprising of an x and y-axis was used to graphically depict subjects in each experimental group. The pre-test (x) and post-test (✔) positions of each group are displayed on the somatogram (Appendix C).

4.2.8.4a Somatogram (x-axis)

All three experimental groups had significantly decreased ($p \leq 0,05$) x-axis values. The largest reduction on the x-axis value was found in group TS (8.68%) followed by

group EST (7.82%) and group ESP (5.20%). None of these decreases between groups were significant (p>0,05).

4.2.8.4b Somatogram (y-axis)

A significantly decrease (p≤0,05) value on the y-axis was observed within all experimental groups. The largest reduction (11.67%) in value on the y-axis was seen in those subjects on electrical muscle stimulation following a standardized diet (placebo controlled) in group ESP, but this was not significantly greater (p>0,05) than those on both thermogenic and electrical muscle stimulation following a standardized diet (Group EST = 7.74%) or those on thermogenic stimulation following a standardized diet (Group TS = 7.28%).

4.3 ULTRASOUND SONOGRAPHY

The results indicating the response of ultrasound sonographic variables among the experimental groups are reflected in Table 4.3a & b and Figure 4.3.

Two sonographic measurements were taken:

i) Subcutaneous fat layer: Measured from the skin surface to the M. rectus abdominus; and

ii) Visceral fat layer: Measured from the M. rectus abdominus to the anterior wall of the aorta.

A significant (p≤0,05) decrease in sonographic measurements was found within all three experimental groups at both subcutaneous as well as visceral fat layers. The subcutaneous fat layer in group EST (21.22%) showed the greatest reduction. This reduction was significantly (p≤0,05) greater than the reduction in subcutaneous fat found in both groups TS (18.04%) and ESP (12.11%).

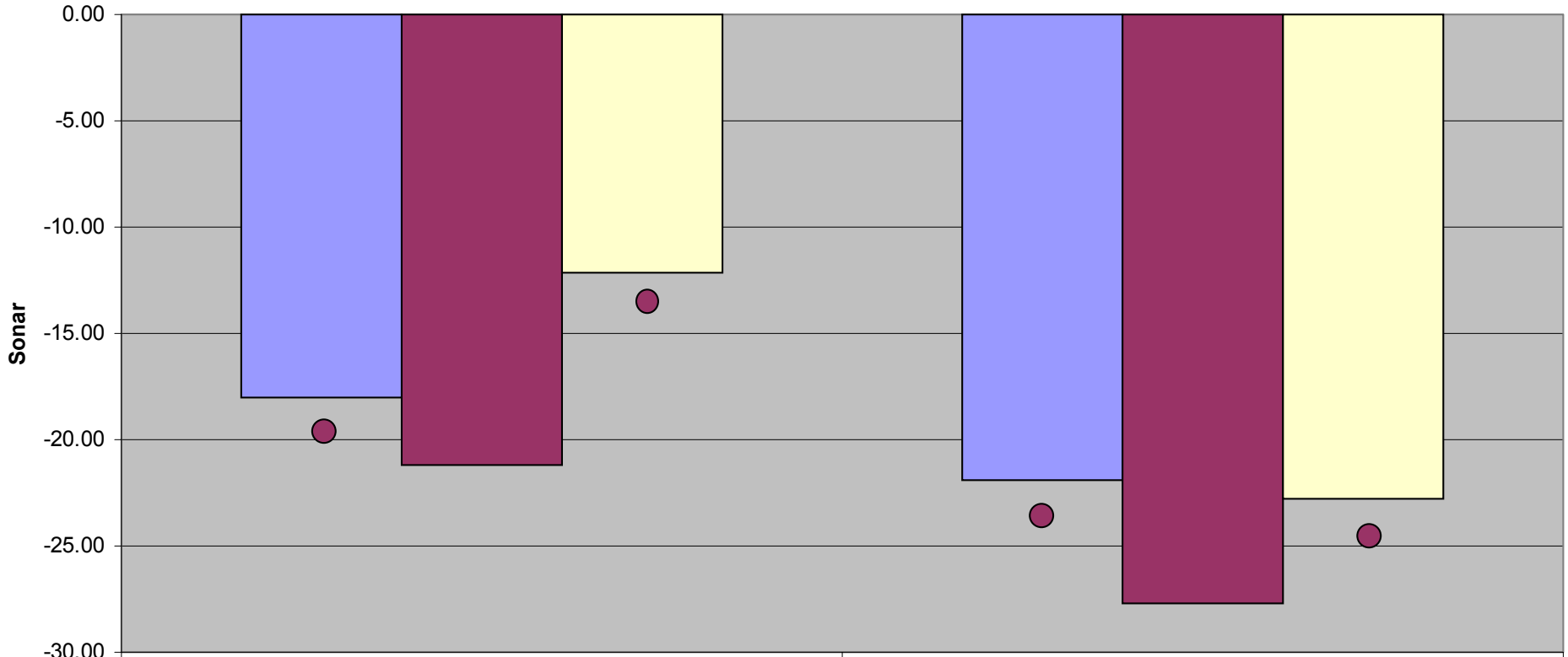
Table 4.3a: Ultrasound Sonography Responses. Intra-Group Comparisons (NS =p > 0.05; ∗ p ≤ 0.05)

Groups		TS (n=20)						EST (n=20)						ESP (n=22)					
VARIABLES	UNITS	PRE Mean	Std. Dev.	POST Mean	Std. Dev.	Δ	p	PRE Mean	Std. Dev.	POST Mean	Std. Dev.	Δ	p	PRE Mean	Std. Dev.	POST Mean	Std. Dev.	Δ	p
Subcutaneous	mm	32.31	8.58	26.48	9.04	-5.83	∗	27.24	9.61	21.46	7.09	-5.78	∗	33.02	9.64	29.02	10.72	-4.00	∗
Visceral	mm	58.86	25.14	45.99	23.13	-12.87	∗	59.92	22.27	43.30	15.14	-16.62	∗	62.19	14.13	48.00	73.00	-14.19	∗

Table 4.3b: Ultrasound Sonography Responses. Inter-Group Comparisons (NS =p > 0,05; ● p ≤ 0,05)

Groups		TS	EST	ESP	Significance		
VARIABLES		%Δ	%Δ	%Δ	TS vs EST	TS vs ESP	EST vs ESP
Subcutaneous	mm	-18.04	-21.22	-12.11	●	NS	●
Visceral	mm	-21.87	-27.74	-22.82	●	NS	●

Group TS ---Thermogenic stimulation and following a standardized diet.
Group EST ---Electrical muscle stimulation and thermogenic stimulation following a standardized diet.
Group ESP ---Electrical muscle stimulation and following a standardized diet (Placebo controlled).



	Subcutaneous	Visceral
TS	-18.04	-21.87
EST	-21.22	-27.74
ESP	-12.11	-22.82

Figure 4.3: Ultrasound Sonography Responses between Groups

The visceral fat layer in group EST (27.74%) also showed the greatest reduction among the three experimental groups. This reduction was significantly ($p \leq 0,05$) greater than that found in both groups ESP (22.82%) and TS (21.87%).

This significantly ($p \leq 0,05$) greater reduction in subcutaneous and visceral fat found in group EST, corresponds with the significant ($p \leq 0,05$) decreases found in body girths, skinfolds and saggital height at the abdominal body site in the same group. Thus for the best reduction in subcutaneous and visceral fat in the abdominal area a combination of diet, thermogenic and electrical muscle stimulation (Group EST) was the most effective.

Group TS (thermogenic stimulation and following a standardized diet) had the second largest reduction in subcutaneous fat (18.04%). This reduction was however not significantly ($p > 0,05$) greater than the reduction found in group ESP (electrical muscle stimulation following a standardized diet and placebo controlled) with a 12.11% reduction. The combination of diet and thermogenic stimulation (Group TS) however did show a tangible effect on the reduction in subcutaneous fat in the abdominal region and may be a usefull modality for obese females.

Visceral abdominal obesity is damaging to health. It has a strong link to "syndrome X" the deadly quartet of high insulin, high sugar, high cholesterol, and high blood pressure (Després, 1991). Even in people who don't have these problems, abdominal obesity is clearly associated with high levels of detrimental LDL cholesterol and low levels of protective HDL cholesterol. Abdominal (visceral) obesity is strongly linked to an increased risk of heart disease and stroke and is far more hazardous to health than subcutaneous obesity. Gender is the most powerful influence on the distribution of body fat, but it's not the only factor. Genetics is responsible for up to 70 percent of an individual's tendency to accumulate extra weight in the midsection (subcutaneous and visceral fat). Age, however, is most responsible for abdominal obesity (Després, 1991).

Abdominal obesity frequently coexist with general overweight or obesity but it could be present in the absence of general onverweight. The presence of abdominal obesity aggravates the deleterious effects arising from general overweight/obesity alone.

The gold standard to determine the presence and extent of intra-abdominal fat (visceral) is with imaging procedures (sonographic measurements). However, as it is so costly to measure intra-abdominal obesity, until now very little data has existed on the impact of exercise, electrical muscle stimulation, thermogenic stimulation and diet on this dangerous, hidden health risk. The advantage of thermogenic and electrical muscle stimulation following a standardized diet (Group EST) as a method to reduce total and intra-abdominal fat, and therefore chronic disease, is that it can be done by most women at affordable cost and with minor risk of side effects.

4.4 RESPIRATORY QUOTIENT

The results indicating the response of respiratory quotient (RQ) when testing in the supine position among the experimental groups are reflected in Table 4.4a & b and Figure 4.4.

A significant ($p \leq 0,05$) decrease in RQ was observed within all groups. The greatest decrease in RQ was observed in group ESP (14.04%). This decrease in RQ in group ESP (electrical muscle stimulation following a standardized diet, placebo controlled) was not significantly ($p > 0,05$) greater than the decreases found in group TS (thermogenic stimulation following a standardized diet = 11.61%) or group EST (thermogenic and electrical muscle stimulation following a standardized diet = 9.35%).

In all intra-group comparisons the significant ($p \leq 0.05$) reduction in pre- post test RQ values indicated a shift from the metabolic substrate carbohydrate to the metabolic substrate of fat. This could be ascribed to all three experimental groups following the same diet. When the metabolic substrate is purely carbohydrate, then the RQ value is 1.0+. When the metabolic substrate is predominantly fat, the RQ approaches 0.7 (Plowman & Smith, 1997).

4.5 PULMONARY FUNCTION

The results indicating the pulmonary function variables among the experimental groups are reflected in Table 4.5a & b and Figure 4.5. An improvement in lung function was observed to a smaller or larger extent within all groups.

Table 4.4a: Respiratory Quotient Response. Intra-Group Comparisons (NS =p > 0.05; ∗ p ≤ 0.05)

Groups		TS (n=20)						EST (n=20)						ESP (n=22)					
VARIABLES	UNITS	PRE Mean	Std. Dev.	POST Mean	Std. Dev.	Δ	p	PRE Mean	Std. Dev.	POST Mean	Std. Dev.	Δ	p	PRE Mean	Std. Dev.	POST Mean	Std. Dev.	Δ	p
RQ	N/A	1.12	0.22	0.99	2.13	-0.13	∗	1.07	0.15	0.97	0.13	-0.10	∗	1.14	0.29	0.98	0.19	-0.16	∗

Table 4.4b: Respiratory Quotient Response. Inter-Group Comparisons (NS =p > 0,05; ● p ≤ 0,05)

Groups		TS	EST	ESP	Significance		
VARIABLES		%Δ	%Δ	%Δ	TS vs EST	TS vs ESP	EST vs ESP
RQ	N/A	-11.61	-9.35	-14.04	NS	NS	NS

Group TS ---Thermogenic stimulation and following a standardized diet.
Group EST ---Electrical muscle stimulation and thermogenic stimulation following a standardized diet.
Group ESP ---Electrical muscle stimulation and following a standardized diet (Placebo controlled).

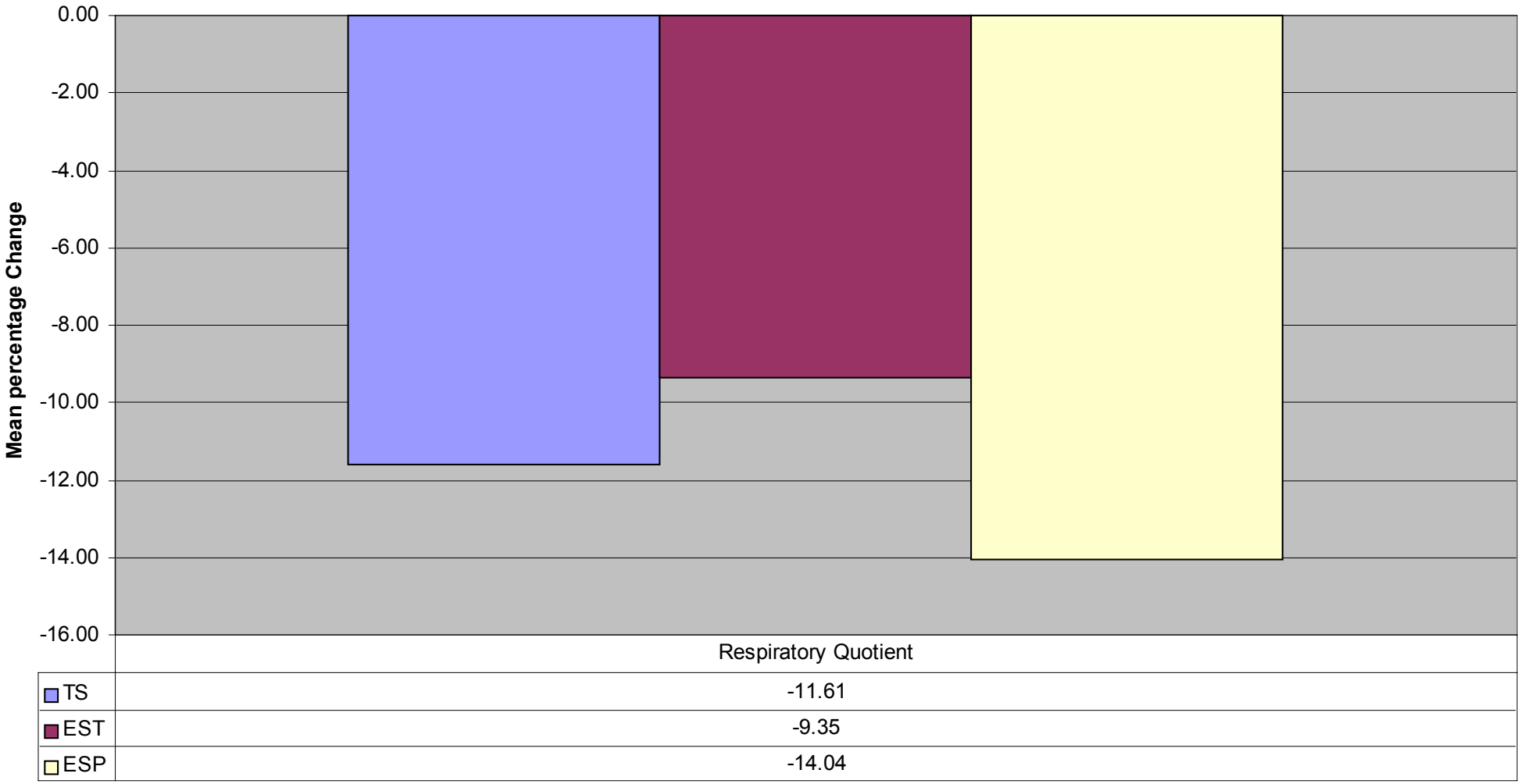


Figure 4.4: Respiratory Quotient Response between Groups

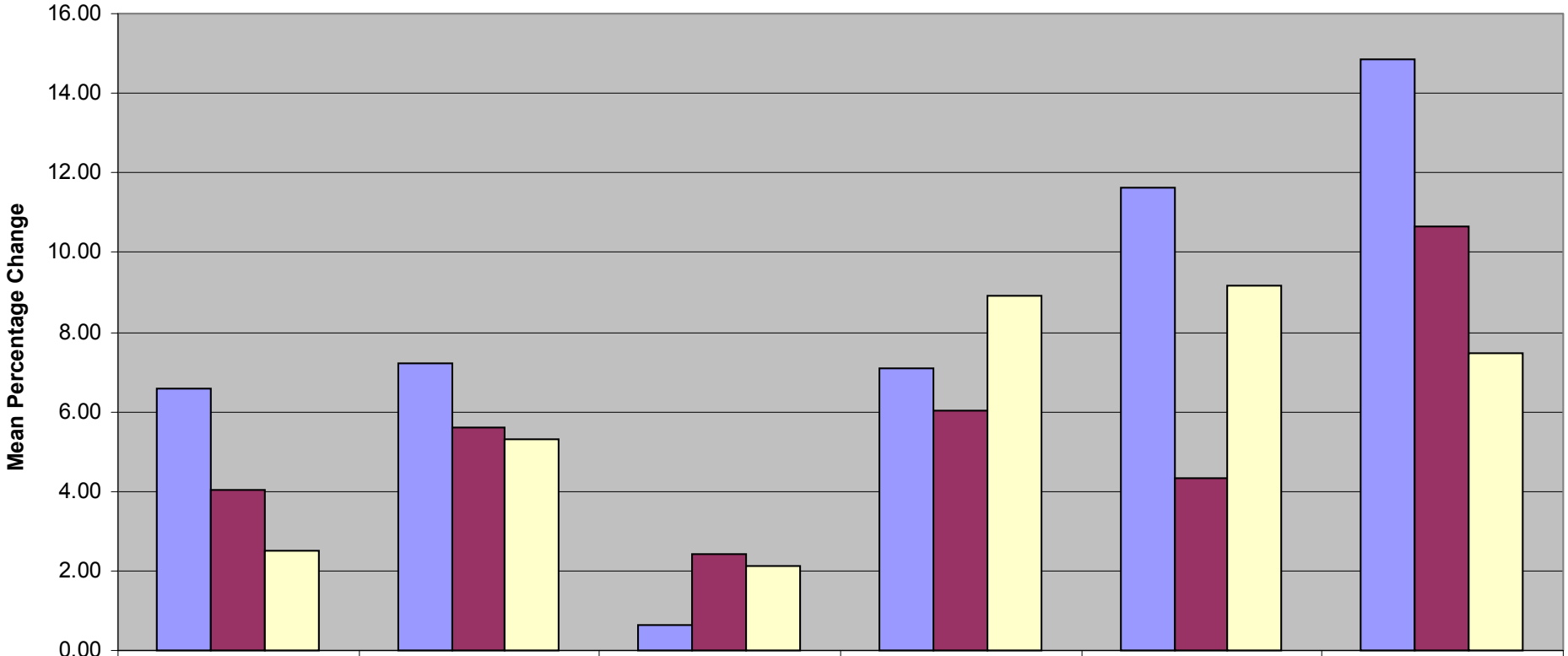
Table 4.5a: Pulmonary Function Responses. Intra-Group Comparisons (NS =p > 0,05; ∗ p ≤ 0,05)

Groups		TS (n=20)						EST (n=20)						ESP (n=22)					
VARIABLES	UNITS	PRE Mean	Std. Dev.	POST Mean	Std. Dev.	Δ	p	PRE Mean	Std. Dev.	POST Mean	Std. Dev.	Δ	p	PRE Mean	Std. Dev.	POST Mean	Std. Dev.	Δ	P
FVC	L	3.64	0.46	3.88	0.64	0.24	∗	3.46	0.51	3.60	0.50	0.14	∗	3.61	0.75	3.70	0.77	0.09	NS
FEV 1.0	L	3.05	0.40	3.27	0.60	0.22	∗	2.86	0.43	3.02	0.40	0.16	∗	3.01	0.55	3.17	0.62	0.16	∗
FEV 1%	%	83.90	4.62	84.45	4.91	0.55	NS	82.70	5.07	84.70	4.30	2.00	∗	83.86	4.91	85.64	4.10	1.78	∗
PEF	L/sec	6.34	0.95	6.79	1.11	0.45	∗	6.13	0.98	6.50	1.03	0.37	NS	6.06	1.06	6.60	1.33	0.54	∗
MEF 50%	L/sec	3.78	0.75	4.22	1.23	0.44	∗	3.70	0.94	3.86	0.94	0.16	NS	3.71	0.70	4.05	0.82	0.34	∗
MEF 25%	L/sec	1.55	0.00	1.78	0.56	0.23	∗	1.14	0.46	1.56	0.35	0.15	∗	1.61	0.50	1.73	0.47	0.12	∗

Table 4.5b: Pulmonary Function Responses. Inter-Group Comparisons (NS =p > 0,05; ● p ≤ 0,05)

Groups		TS	EST	ESP	Significance		
VARIABLES		%Δ	%Δ	%Δ	TS vs EST	TS vs ESP	EST vs ESP
FVC	L	6.59	4.05	2.49	NS	NS	NS
FEV 1.0	L	7.21	5.59	5.32	NS	NS	NS
FEV 1%	%	0.66	2.42	2.12	NS	NS	NS
PEF	L/sec	7.10	6.04	8.91	NS	NS	NS
MEF 50%	L/sec	11.64	4.32	9.16	NS	NS	NS
MEF 25%	L/sec	14.84	10.64	7.45	NS	NS	NS

Group TS---Thermogenic stimulation and following a standardized diet.
Group EST ---Electrical muscle stimulation and thermogenic stimulation following a standardized diet.
Group ESP ---Electrical muscle stimulation and following a standardized diet (Placebo controlled).



	FVC	FEV 1.0	FEV 1%	PEF	MEF 50%	MEF 25%
TS	6.59	7.21	0.66	7.10	11.64	14.84
EST	4.05	5.59	2.42	6.04	4.32	10.64
ESP	2.49	5.32	2.12	8.91	9.16	7.45

Figure 4.5: Pulmonary Function Responses between Groups

The greatest and significant (p≤0,05) increase in forced vital capacity (FVC) was observed in group TS (6.59%). This increase in FVC in group TS (thermogenic stimulation following a standardized diet) was however not significant (p>0,05) greater than the significant (p≤0,05) increase found in group EST (thermogenic and electrical muscle stimulation following a standardized diet = 4.05%) or the non-significant (p>0,05) increase within group ESP (electrical muscle stimulation following a standardized diet, and placebo controlled = 2.49%).

The greatest and significant (p≤0,05) increase in forced expiratory volume during the 1st second of forced vital capacity (FEV_1) was also found in group TS. This increase in FEV_1 in group TS (7.21%) was, however not significantly (p>0,05) greater than the equally significant (p≤0,05) increases found within groups EST (5.59%) and group ESP (5.32%).

The greatest and significant (p≤0,05) increase in FEV_1%, thus indicating breathing efficiency, was observed in group EST (2.42%). This increase in FEV_1% observed in group EST was not significantly (p>0,05) greater than the significant (p≤0,05) increase found within group ESP (2.12%) or the non-significant (p>0,05) increase within group TS (0.66%).

An increased amount of fat in the chest wall and diaphragm leads to an alteration of respiratory excursion during inspiration and expiration. The increase 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). If the increase amount of fat leads to the above mentioned, the decrease of fat should lead to an increase in FEV_1% (breathing efficiency).

The greatest reduction in body mass, lean body mass, body mass index, waist-to-hip ratio, body surface area, sum of body girths, sum of seven skinfolds, saggital height and abdominal sonography measurements were found in group EST and thus corresponds positively with the increase found in FEV_1% (breathing efficiency).

The greatest and significant (p≤0,05) increase in peak expiratory flow (PEF) was observed within group ESP (8.91%). This increase in PEF in group ESP was not significantly (p>0,05) different from the significant (p≤0,05) increase found in group TS (7.10%) or the insignificant (p>0,05) increases within group EST (6.04%). 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 strength (Babb, 1999). If abdominal exercise improves expiration force, the potential influence of electrical muscle stimulation (EMS) was observed in group ESP (8.91%) and group EST (6.04%) both with an increase in PEF values.

The greatest and significant (p≤0,05) increase in mid-flow (MEF 50%) was observed in group TS (11.64%). This increase in MEF 50% in group TS did not differ significantly (p>0,05) from the equally significant (p≤0,05) increases found in group ESP (9.16%) and group EST (4.32%). The same tendency was observed in end-flow (MEF 25%) with the greatest and significant (p≤0,05) increase observed in group TS (14.84%). This increase in MEF 25% in group TS did not differ significantly (p>0,05) from the equally significant (p≤0,05)increases found in group EST (10.64%) and group ESP (7.45%).

In four out of the six pulmonary function parameters that were tested, group TS (thermogenic stimulation following a standardized diet) showed the greatest increase. Subjects in all three experimental groups showed signs of restricted pulmonary ventilation, especially in the bronchial tubes (MEF 25%). Ventilation-perfusion is the most common abnormality of gas exchange found in extreme obesity (Kopelman, 1984). Although there were significant (p≤0,05) increases in MEF 25% values in the pre-post test values within each group, there were no significant differences (p>0,05) between groups after the eight weeks on the respective intervention programs. These results do, however, support findings from other studies (Babb et al., 1989) that weight-loss results in a mechanical improvement in respiration among obese people.

Investigators at Yale University in the United States, noted that although exercise training improves exercise tolerance in most patients with chronic obstructive pulmonary disease, some severely affected patients may be unable to tolerate it because of incapacitating breathlessness (Bourjeily-Habr et al., 2002). There were no significant changes in lung function, peak workload or peak oxygen consumption in either group (Bourjeily-Habr et al., 2002).

4.6 HAEMATOLOGY

Obesity is associated with metabolic abnormalities such as abnormal cholesterol and triglyceride levels, impaired glucose and insulin metabolism and increased blood pressure. The results indicating the response of haematological variables among the experimental groups are reflected in Tables 4.6a & b and Figure 4.6.

4.6.1 Total cholesterol

The only reduction in total cholesterol was seen in group TS (0.58%). The reduction found in group TS (thermogenic stimulation following an standardized diet) did not differ significantly ($p>0.05$) from the increases found in group EST (4.48%) and group ESP (3.61%). Thermogenic stimulation following a standardized diet (Group TS) was thus no more effective in alternating total cholesterol than was thermogenic and electrical muscle stimulation following a standardized diet (Group EST) or electrical muscle stimulation and following a standardized diet, placebo controlled (Group ESP). An increased lipid level in the blood is termed hyperlipidemia. Cholesterol and triglycerides are the two most common lipids associated with coronary heart disease (CHD) risk. These fats do not circulate freely in the blood plasma, but rather are transported in combination with a carrier protein to form a lipoprotein. Serum cholesterol represents a composite of the total cholesterol contained in the different lipoproteins. The distribution of cholesterol among the various types of lipoproteins may be a more powerful predictor of heart disease than simply the total quantity of plasma lipids. The quantity of LDL and HDL, as well as the specific ratio of these plasma lipoproteins to each other and to total cholesterol, may provide a more meaningful signal than cholesterol per se in predicting the probability of contracting coronary heart disease. The ratio is improved with a low-calorie, low-saturated fat

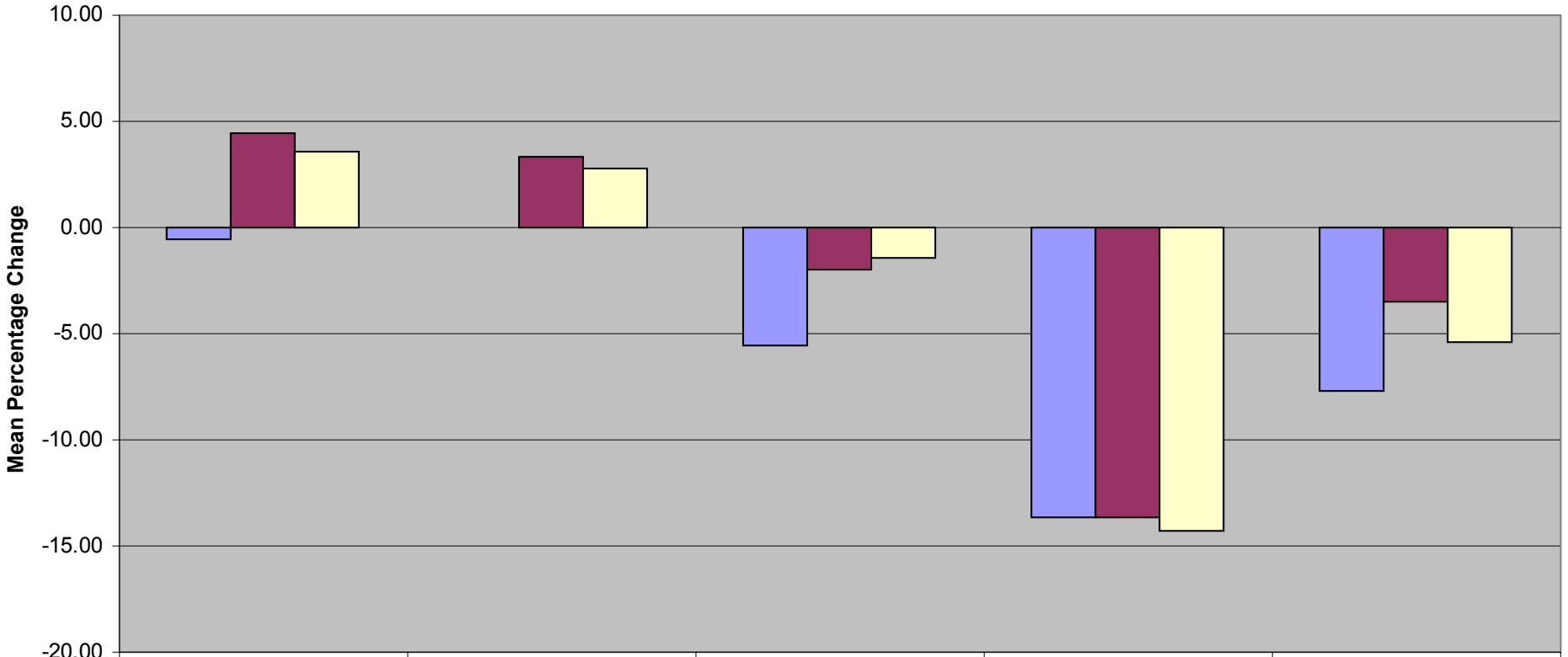
Table 4.6a: Heamatological Responses. Intra-Group Comparisons (NS =p > 0,05; ∗ p ≤ 0,05)

Groups		TS (n=20)						EST (n=20)						ESP (n=22)					
VARIABLES	UNITS	PRE Mean	Std. Dev.	POST Mean	Std. Dev.	Δ	p	PRE Mean	Std. Dev.	POST Mean	Std. Dev.	Δ	p	PRE Mean	Std. Dev.	POST Mean	Std. Dev.	Δ	p
Cholesterol	mmol/L	5.13	1.03	5.10	1.01	-0.03	NS	5.13	0.68	5.36	0.62	0.23	NS	4.99	0.90	5.17	1.21	0.18	NS
Cholesterol HDL	mmol/L	1.12	0.25	1.12	0.31	0.00	NS	1.21	0.27	1.25	0.29	0.04	NS	1.09	0.22	1.12	0.26	0.03	NS
Cholesterol LDL	mmol/L	3.60	0.98	1.01	0.44	-0.20	∗	3.47	0.60	3.40	0.71	-0.07	NS	3.49	0.85	3.44	0.99	-0.05	NS
Triglycerides	mmol/L	1.17	0.78	1.01	0.44	-0.13	NS	1.17	0.88	1.01	0.48	-0.16	NS	0.98	0.54	0.84	0.37	-0.14	∗
Glucose	mmol/L	4.95	0.42	4.57	0.43	-0.38	∗	4.90	0.66	4.73	0.61	-0.17	NS	4.97	0.83	4.70	0.73	-0.27	∗

Table 4.6b: Heamatological Responses. Inter-Group Comparisons (NS =p > 0,05; • p ≤ 0,05)

Groups		TS	EST	ESP	Significance		
VARIABLES		%Δ	%Δ	%Δ	TS vs EST	TS vs ESP	EST vs ESP
Total Cholesterol	mmol/L	-0.58	4.48	3.61	NS	NS	NS
HDL-Cholesterol	mmol/L	0.00	3.31	2.75	NS	NS	NS
LDL-Cholesterol	mmol/L	-5.56	-2.02	-1.43	NS	NS	NS
Triglycerides	mmol/L	-13.68	-13.70	-14.29	NS	NS	NS
Glucose	mmol/L	-7.68	-3.47	-5.43	NS	NS	NS

Group TS---Thermogenic stimulation and following a standardized diet.
Group EST ---Electrical muscle stimulation and thermogenic stimulation following a standardized diet.
Group ESP ---Electrical muscle stimulation and following a standardized diet (Placebo controlled).



	Total Cholesterol	HDL-Cholesterol	LDL-Cholesterol	Triglycerides	Glucose
TS	-0.58	0.00	-5.56	-13.68	-7.68
EST	4.48	3.31	-2.02	-13.68	-3.47
ESP	3.61	2.75	-1.43	-14.29	-5.43

Figure 4.6: Haematological Responses between Groups

diet. Regular and moderate levels of aerobic exercise may also increase the HDL level and favourably affect the LDL/HDL ratio (Manson et al., 1990).

4.6.2 LDL-Cholesterol

A lowering in LDL cholesterol level was observed within all three experimental groups. The greatest (5.56%) and only significant ($p \leq 0,05$) reduction in LDL cholesterol level was seen in those subjects on thermogenic stimulation following a standardized diet (Group TS) but this reduction did not differ significantly ($p > 0.05$) from those subjects on thermogenic and electrical muscle stimulation following a standardized diet (Group EST = 2.02%) or those subjects on electrical muscle stimulation following a standardized diet, placebo controlled (Group ESP = 1.43%).

Low density lipoproteins (LDL) contain the greatest fat and least protein components. The LDLs, which normally carry 60 to 80% of the total cholesterol, have the greatest affinity for the arterial wall. They help to carry cholesterol into the arterial tissue to become chemically modified and ultimately cause a proliferation of underlying smooth muscle cells and further changes that damage and narrow the artery in the process of coronary heart disease. The relation between high LDL cholesterol and death from coronary artery disease is not related to some threshold level but, instead, is continuous and graded so that any lowering of this blood lipid may offer cardio-protection. Numerous animal studies indicate that diets high in cholesterol and saturated fat raise LDL cholesterol in "susceptible" animals and eventually produce a degenerative process characterized by the formation of cholesterol rich plaque deposits on the inner lining of the medium and larger arteries. This process of arteriosclerosis leads to a narrowing and eventual closure of these vessels. In humans, a reduced saturated fat and cholesterol intake generally has a lowering effect on LDL cholesterol, although for most people the effect is modest.

4.6.3 HDL-Cholesterol

The greatest increase in HDL cholesterol was observed in group EST (3.31%). This increase in HDL cholesterol was closely followed by the increase found in group ESP (2.75%). There was no change in HDL cholesterol level found in group TS (0.00%). Although increases within groups were observed in group EST and group ESP none of

these changes were statistically significant (p>0,05) nor differ significantly nor did they differ significantly (p>0,05) between groups.

HDL cholesterol may operate to protect against heart disease in two ways:

1. to carry cholesterol away from the arterial wall for degradation to bile in the liver and subsequently excreted by the intestines; and

2. to compete with the LDL cholesterol fragments for entrance into the cells of the arterial wall. Regular and moderate levels of aerobic exercise may also increase the HDL cholesterol level and favourably affect the LDL/HDL ratio (Manson et al., 1990). The above mentioned might be the reason for the increases in HDL cholesterol level found in those groups on electrical muscle stimulation (Group EST and Group ESP).

4.6.4 Triglycerides

Triglycerides, the most plentiful fat in the body, constitute the major storage form of fat (more than 95% of the body fat is in the form of triglycerides). Fatty acids released from triglycerides in the fat storage sites and delivered in the circulation to muscle tissue as free fatty acids (FFA) bound to blood albumin, as well as the triglycerides stored in the muscle cell itself, contribute considerably to the energy requirements of exercise.

Of all haematological responses tested the greatest reduction was found in triglyceride levels. The greatest reduction of all three experimental groups were found in group ESP (electrical muscle stimulation, following a standardized diet and placebo controlled) with a 14.29% reduction in triglyceride level. Both group EST (thermogenic stimulation following a standardized diet) and group TS (thermogenic and electrical muscle stimulation following a standardized diet) had the same reduction (13.68%) in triglyceride levels. None of these reductions in triglyceride levels within groups or between groups were statistically significant (p>0,05). All three experimental groups were on the same diet thus the reason for the same reduction in triglyceride levels. Only if exercise continues for an hour or more and carbohydrates become depleted then there is a gradual increase in the quantity of fat

(triglycerides) utilized for energy (Plowman & Smith, 1997). Subjects in group EST and ESP were on electrical muscle stimulation for a duration of 45 minutes per session thus the influence of moderate exercise could not have had an influence on triglyceride levels.

4.6.5 Glucose

More than 200 monosaccharides have been found in nature. The most common of the monosaccharides are glucose, fructose and galactose. Glucose, also referred to as dextrose or blood sugar, is formed as a natural sugar in food or is produced in the body as a result of digestion of more complex carbohydrates or via the process of gluconeogenesis, whereby it is synthesized from the carbon skeletons of other compounds. After absorption by the small intestines, glucose can be used directly by the cell for energy, stored as glycogen in the muscles and liver, or converted to fats for energy storage. Fructose, or fruit sugar, is present in large amounts in natural form in fruits and honey and is the sweetest of the simple sugars. Although some fructose is absorbed directly into the blood from the digestive tract, it is all slowly converted to glucose in the liver. In the body, galactose is also converted to glucose for energy metabolism (Mc Ardle et al., 1996).

A significant ($p \leq 0,05$) decrease in glucose levels was observed within two of the experimental groups (TS and ESP). The largest decrease (7.68%) was seen in those subjects on thermogenic stimulation and following a standardized diet(Group TS) but this did not differ significantly ($p > 0,05$) from those on electrical muscle stimulation following a standardized diet, placebo controlled (Group ESP = 5.43%) or those on both thermogenic and electrical muscle stimulation following a standardized diet (Group EST = 3.47%). The decrease in glucose levels in all three experimental groups may be ascribed to the subjects being on the same diet for the duration of the study.

4.7 CARDIOVASCULAR RESPONSES

The results indicating cardiovascular response variables among the experimental groups are reflected in Table 4.7a & b and Figure 4.7.

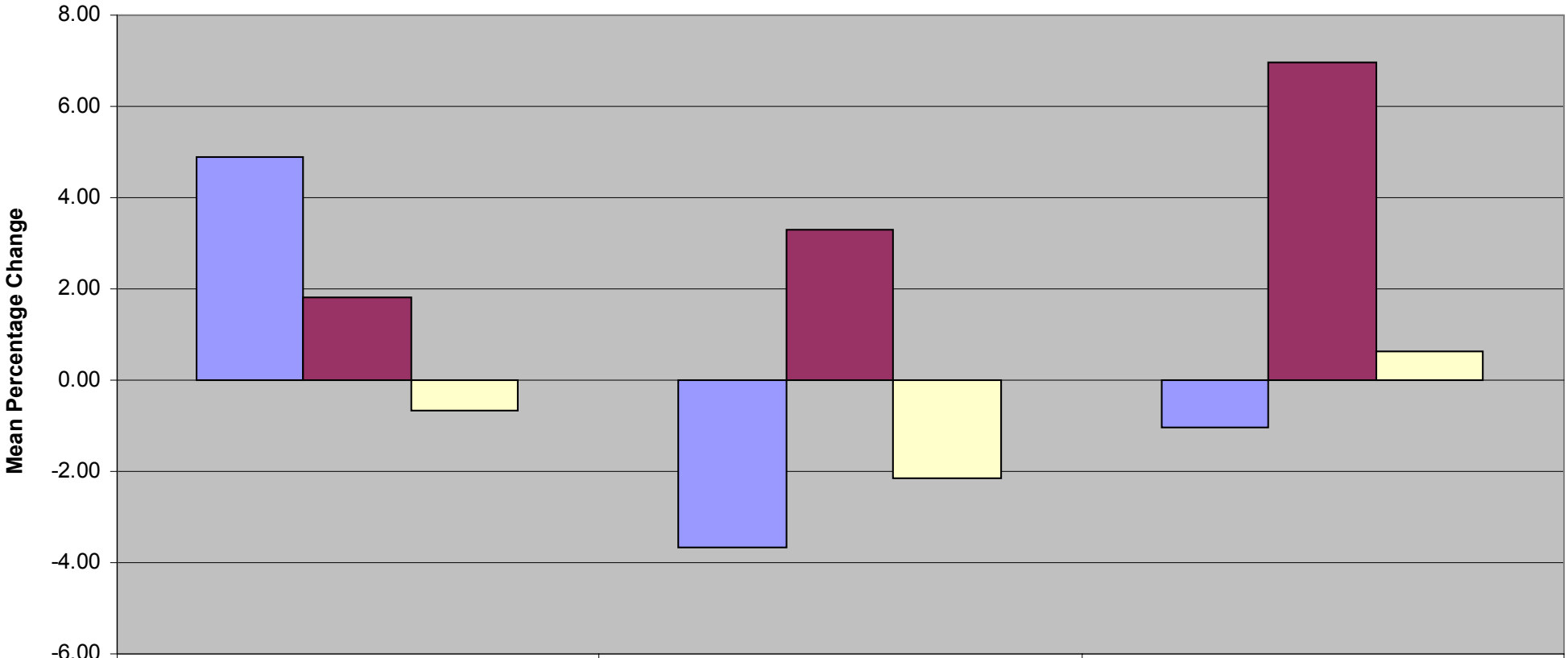
Table 4.7a: Cardiovascular Responses. Intra-Group Comparisons (NS =p > 0,05; ∗ p ≤ 0,05)

Groups		TS (n=20)						EST (n=20)						ESP (n=22)					
VARIABLES	UNITS	PRE Mean	Std. Dev.	POST Mean	Std. Dev.	Δ	p	PRE Mean	Std. Dev.	POST Mean	Std. Dev.	Δ	p	PRE Mean	Std. Dev.	POST Mean	Std. Dev.	Δ	p
Heart Rate	bpm	74.60	9.69	78.25	13.58	3.65	NS	68.80	10.83	70.05	10.47	1.25	NS	75.55	13.93	75.05	10.41	-0.50	NS
Systolic BP	mmHg	122.25	25.47	117.75	13.81	-4.50	NS	113.25	11.27	117.00	13.42	3.75	NS	117.05	12.97	114.55	11.54	-2.50	NS
Diastolic BP	mmHg	73.00	14.90	72.25	9.79	-0.75	NS	68.25	8.12	73.00	9.79	4.75	NS	70.91	9.71	71.36	9.41	0.45	NS

Table 4.7b: Cardiovascular Responses. Inter-Group Comparisons (NS =p > 0,05; • p ≤ 0,05)

Groups		TS	EST	ESP	Significance		
VARIABLES		%Δ	%Δ	%Δ	TS vs EST	TS vs ESP	EST vs ESP
Heart Rate	bpm	4.89	1.82	-0.66	NS	NS	NS
Systolic BP	mmHg	-3.68	3.31	-2.14	NS	NS	NS
Diastolic BP	mmHg	-1.03	6.96	0.63	NS	NS	NS

Group TS ---Thermogenic stimulation and following a standardized diet.
Group EST ---Electrical muscle stimulation and thermogenic stimulation following a standardized diet.
Group ESP ---Electrical muscle stimulation and following a standardized diet (Placebo controlled).



	Heart Rate	Systolic BP	Diastolic BP
TS	4.89	-3.68	-1.03
EST	1.82	3.31	6.96
ESP	-0.66	-2.14	0.63

Figure 4.7: Cardiovascular Responses between Groups

4.7.1 Heart rate

The only reduction in resting heart rate was noted in group ESP (0.66%). This reduction in heart rate in group ESP (electrical muscle stimulation following a standardized diet placebo controlled) did not differ significantly (p>0,05) from the increases found in group EST (thermogenic and electrical muscle stimulation following a standardized diet = 1.82%) and group TS (thermogenic stimulation following a standardized diet = 4.89%). Heart rate fluctuations in all three-study groups were ever so slight, and may be ascribed to anxiety levels of the subjects during the post-intervention testing.

4.7.2 Blood pressure

Systolic blood pressure

Both group TS (3.68%) and group ESP (2.14%) had reductions in systolic blood pressure levels. These reductions in systolic blood pressure levels did not differ significantly (p>0,05) from the increase found in group EST (3.31%).

Diastolic blood pressure

Only group TS showed a reduction (1.03%) in diastolic blood pressure. This reduction in diastolic blood pressure did not differ significantly (p>0,05) from the increases found in group ESP (0.73%) and group EST (6.96%). It is difficult to explain the slight increases found in diastolic blood pressure in groups ESP and EST. Group ESP was on a placebo and thus the influence of a thermogenic agent can be negated. The only group with a reduction in diastolic blood pressure was group TS (thermogenic stimulation following a standardized diet). The influence of a thermogenic agent (Group TS) on the lowering of diastolic blood pressure was thus positive. Groups ESP (electrical muscle stimulation, following a standardized diet and placebo controlled) and group EST (electrical muscle stimulation following a standardized diet) had increases in diastolic blood pressure. Both groups ESP and EST used electrical muscle stimulation in their intervention program. Although not statistically significant (p>0,05) the influence of electrical stimulation (Groups ESP and EST) on the lowering of diastolic blood pressure appears to be inversely related.

4.8 MUSCULOSKELETAL FUNCTION

The results indicating the response of musculoskeletal function variables among the experimental groups are reflected in Table 4.8a & b and Figure 4.8.

4.8.1 Flexibility

The sit-and-reach test was used to determine hip flexion (flexibility of the hamstrings and lower back). A positive percentage score is interpreted as an increase in hip flexion. There was a significant ($p \leq 0,05$) 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 EST (21.22%) and group TS (18.04%) improved their hip flexibility more in relation to group ESP (12.11%).

People who are active tend to be more flexible than those who are not. The reason for this is that flexibility is motion dependent (Mentz, 2000). 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 (subcutaneous and visceral) usually limits flexibility. Fat deposits act as a wedge between moving parts of the body restricting movement (Hockey, 1993).

The increase in flexibility in all three experimental groups could be due to the reduction in body mass. An inverse relationship was evident when comparing the increase in flexibility with the decrease in subcutaneous fat in all three groups. The same tendency was found when flexibility was compared to visceral (intra-abdominal fat). The assumption can be made that the lower the fat % the higher the flexibility will be in obese females thus confirming the statement that excessive body fat limits flexibility and range of motion at certain joints. The results also indicate that light to moderate muscle action of gradually increasing intensity might be more appropriate for increasing flexibility than stretching itself.

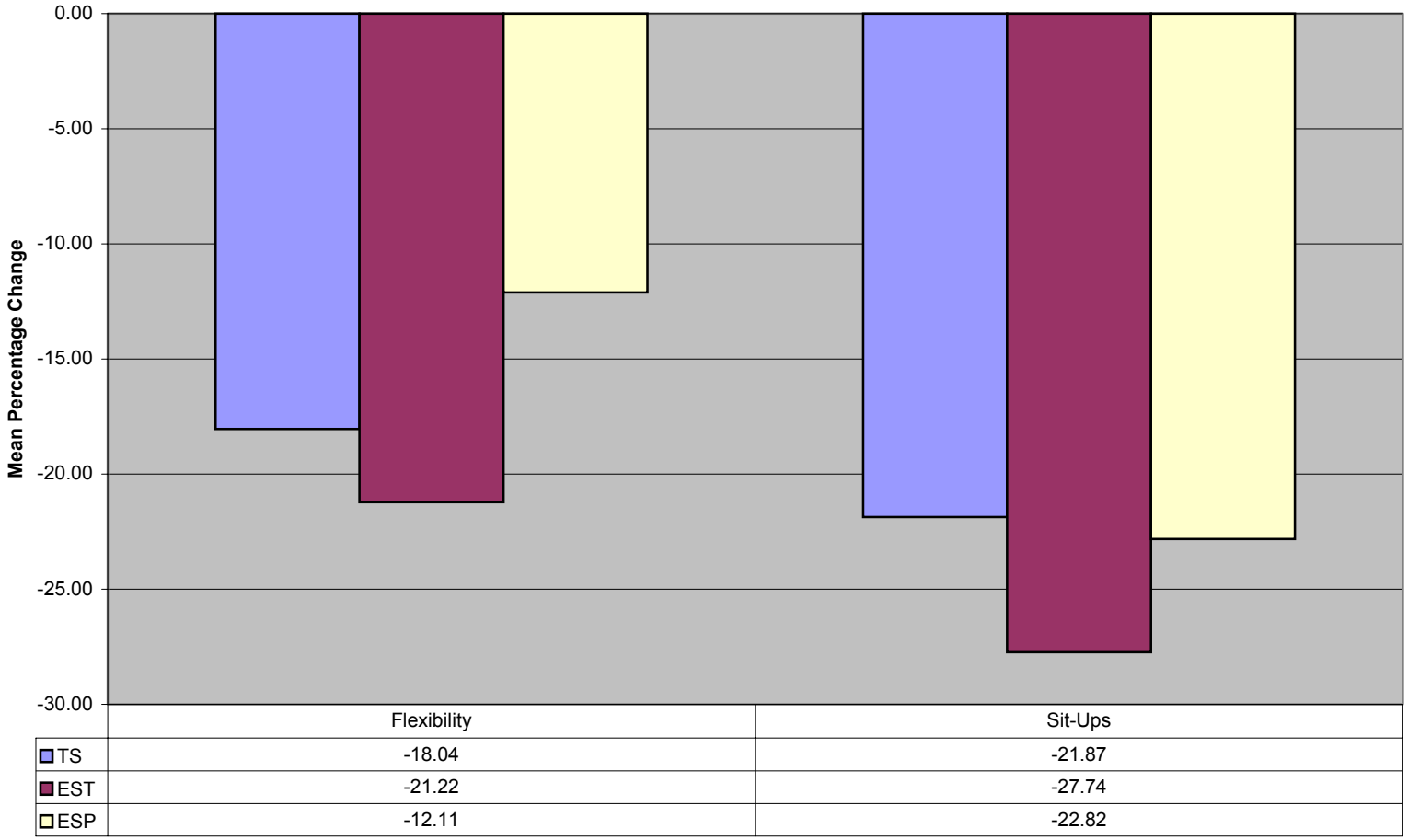
Table 4.8a: Musculoskeletal Function Responses. Intra-Group Comparisons (NS =p > 0.05; ∗ p ≤ 0.05)

Groups		TS (n=20)						EST (n=20)						ESP (n=22)					
VARIABLES	UNITS	PRE Mean	Std. Dev.	POST Mean	Std. Dev.	Δ	p	PRE Mean	Std. Dev.	POST Mean	Std. Dev.	Δ	p	PRE Mean	Std. Dev.	POST Mean	Std. Dev.	Δ	p
Flexibility	cm	30.03	9.85	32.47	9.83	2.44	∗	32.25	6.71	35.10	6.17	2.85	∗	27.36	8.71	30.62	8.47	3.26	∗
Sit-Ups	no.	12.95	8.25	16.75	8.54	3.80	∗	16.45	9.08	20.75	9.93	4.30	∗	13.32	9.18	16.32	9.73	3.00	∗

Table 4.8b: Musculoskeletal Function Responses. Inter-Group Comparisons (NS =p > 0,05; ● p ≤ 0,05)

Groups		TS	EST	ESP	Significance		
VARIABLES		%Δ	%Δ	%Δ	TS vs EST	TS vs ESP	EST vs ESP
Flexibility	cm	18.04	21.22	12.11	NS	NS	NS
Sit-Ups	no.	21.87	27.74	22.82	NS	NS	NS

Group TS ---Thermogenic stimulation and following a standardized diet.
Group EST ---Electrical muscle stimulation and thermogenic stimulation following a standardized diet.
Group ESP ---Electrical muscle stimulation and following a standardized diet (Placebo controlled).



	Flexibility	Sit-Ups
TS	-18.04	-21.87
EST	-21.22	-27.74
ESP	-12.11	-22.82

Figure 4.8: Musculoskeletal Function Responses between Groups

4.8.2 Abdominal muscle endurance

Abdominal muscle endurance was evaluated with the maximum number of sit-ups performed in one-minute. Abdominal muscle endurance improved significantly ($p \leq 0,05$)in all three experimental groups. The greatest improvement was in group EST (27.74%) but this was not significantly ($p > 0,05$) better than the other two modalities, ESP (22.82%) and TS (21.87%).

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 (Mentz, 2000). Thermogenic and electrical muscle stimulation following a standardized diet(Group EST) had the best effect on abdominal endurance, although not significantly more so than electrical muscle stimulation following a standardized diet, (placebo controlled, Group ESP) or thermogenic stimulation following a standardized diet (Group TS). Further significant strength toning and power gains could have been achieved through an increase in isotonic muscle involvement. There is no single best exercise to train abdominal muscles. A routine of low resistance and high repetitions favours abdominal muscle toning and endurance. Thus the impressive results found in both groups with electrical muscle stimulation (Group EST and ESP).

Fat deposits act as a wedge between moving parts of the body, restricting movements (Hockey, 1993). With a decrease found in both subcutaneous and visceral body fat in all three the experimental groups (EST, TS, ESP), the increase in the maximal number of sit-ups and thus abdominal muscle endurance, appears logical.

CHAPTER 5

SUMMARY, CONCLUSION AND RECOMMENDATIONS

5.1 GENERAL CONSIDERATIONS REGARDING WEIGHT

5.1.1 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 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 in any of the experimental groups (TS; EST; ESP) experienced any adverse medical, psychological or psychological events;

- Participants adhered to the program in that only 7 of 69 subjects withdrew from the study in eight weeks; and

- Subjects who were part of the intervention involving dietary restriction, thermogenic stimulation and electrical muscle stimulation, showed greater benefit than those subjects on dietary restriction, electrical muscle stimulation and a placebo instead of a thermogenic agent.

5.1.2 Recommendations for weight-loss programs

- Thermogenic agents, diet, exercise and behaviour modification should be combined.

- Exercise, nutritional, psychological and medical expertise should be consulted.

- The amount and type of counselling should be individualized and/or limited to small groups rather than large audiences/groups.

- The weight maintenance phase is more stable and prolonged when the weight-loss program includes a diet in combination with exercise.

- Individuals with a body mass index (BMI) > 25 $kg \cdot m^{-2}$ should consider engaging in weight-loss efforts to reduce their body weight.

- Individuals undertaking non-medically supervised weight-loss initiatives should reduce their energy intake by 500-1000 $kcal \cdot d^{-1}$ to elicit a weight-loss of approximately 0.5-0.9 $kg \cdot wk^{-1}$. It is recommended that dietary fat intake should be reduced to <30% of total energy intake.

- Individuals seeking weight-loss should include exercise as a key component to their weight-loss program.

- Individuals considering using dietary supplements or weight-loss enhancing agents (thermogenic stimulation) should do so only under the guidance of their personal physician or other trained health care providers (biokineticist).

5.1.3 Pro-active steps for weight-loss

- Health care providers should counsel obese patients (currently a relatively rare event).

- Workplaces should offer healthier food choices in their cafeterias and opportunities for physical activity.

- Schools should offer not only more physical activity, but also the types of activities that help children form the habit of daily lifelong activity.

- Urban planners and residents should motivate for more sidewalks, like paths, and other safe alternatives to cars.

- Parents can encourage their children to get away from screens and monitors and out into yards and parks.

(Koplan, 2000)

5.2 SPECIFIC WEIGHT-LOSS CONSIDERATIONS BASED ON THIS STUDY

The primary aim of this study was to evaluate the effect of an eight-week programme of electrical muscle stimulation (EMS) performed on Slimline Slimming Machines in conjunction with, and without, a thermogenic agent (Thermo Lean) and following a specific diet. In order to achieve this goal a pretest-post test experimental groups 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 TS (N = 23) - Thermogenic Stimulation and following a standardized diet.

- Group EST (N = 23) - Electrical Muscle Stimulation and Thermogenic stimulation combined following the standardized specific diet.

- Group ESP (N = 23) - Electrical Muscle Stimulation and a thermogenic placebo combined following the standardized diet

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

5.2.1 Relative efficacy of the interventions

TABLE 5.1: RELATIVE EFFICACY OF INTERVENTIONS

Composite rating of results after an eight-week intervention programme

5 : Greatest effect on specific physiological parameters ($p \leq 0,05$)

3 : Greatest effect on specific physiological parameters ($p > 0,05$)

2 : Intermediate effect on specific physiological parameters

1 : Smallest effect on specific physiological parameters

Variable	TS	EST	ESP	(NVT)
Anthropometry	30	32	13	(12)
Morphology	34	61	34	(19)
Ultrasound sonography	3	10	3	(2)
Respiratory quotient	2	1	3	(1)
Pulmonary function	15	11	10	(6)
Haematology	11	9	10	(5)
Cardiovascular response	7	4	7	(3)
Musculoskeletal function	3	5	4	(2)
Total Score	**105**	**133**	**84**	**(50)**
Max Score (*)	**250**	**250**	**250**	
Relative Score (%)	**42.0**	**53.2**	**33.6**	

NVT = Number of variables tested

* = 250 is the maximum score attainable for 50 variables tested achieving a rating of 5 (Greatest effect on specific physiological parameters $p \leq 0,05$).

TS = Thermogenic stimulation and following a standardized diet.

EST = Electrical muscle stimulation and thermogenic stimulation following a standardized diet.

ESP = Electrical muscle stimulation and following a standardized diet (Placebo controlled).

Obesity develops when a chronic quantitive imbalance exist between energy intake and energy expenditure. To unbalance the energy equation in the direction of weight-loss, requires decreased food energy intake (diet – Group EST, TS, ESP) increased physical activity (electrical muscle stimulation – Group EST, ESP) increased energy expenditure pharmacologically (thermogenic stimulation – Group EST, TS) or altering all three intervention strategies simultaneously (diet, thermogenic and electrical muscle stimulation – Group EST).

In this study great efforts were made to manipulate the diet in all three experimental groups (metabolism diet) and in two of the experimental groups (EST, TS) efforts were made to increase energy expenditure pharmacologically as a tool to enhance the weight-loss after the eight-week intervention program. There is accumulating evidence to support the hypothesis that a low-energy output phenotype predisposes individuals to weight gain and obesity, whether the low energy output is caused by a low resting metabolic rate (RMR), physical inactivity or both (Plowman & Smith, 1997). Increased energy metabolism is therefore an attractive target because it may allow people to maintain food intake at socially more acceptable levels. There is evidence to support the view that any increase in energy expenditure is not fully counteracted by a similar increase in appetite and energy intake, irrespective of whether the increased energy output was achieved through exercise or pharmacologically (Oomura et al., 1999). Even a slight increase of 2 to 3% in daily energy expenditure through thermogenic stimulation may therefore have clinical relevance, particularly in preventing the decline in resting metabolic rate with weight-loss, but also in decreasing the risk of weight regain following weight-loss. Both groups with a pharmacologic intervention (Groups EST, TS) as part of their intervention program, had the greatest influence on physiological parameters tested thus confirming the above-mentioned hypothesis.

To date, the emphasis in the treatment of obesity has been on restricting the energy intake (dietary) side of the energy balance equation. There are at least two reasons why decreasing energy intake has been favoured over increasing energy expenditure through physical activity. The first is economic. In recent years, the cost of burning extra calories has surged as work has become more sedentary. In the past people were paid to engage in manual labour. Today one pays to exercise, not so much in money, but in foregone leisure time (Postrel, 2001).

Secondly, it has been difficult to demonstrate the efficacy of exercise as a treatment strategy for obesity. Although a considerable number of studies have been done to investigate whether exercise contributes to weight-loss, the results have been mixed (Saris, 1996). McArdle et al. (1996) cite evidence to support the contention that an increased level of regular physical activity may be more effective than dieting for long-term weight control. In a meta-analysis study conducted to assess the effects of exercise on changes in body mass, Ballor and Keesey (1991) found that increases in physical activity results in body mass reductions. One possible clue to the variability of results in this area (impact of exercise on weight-loss) is offered in a recent review (Saris, 1996), which found that only a few of the studies to-date were well-controlled.

In our study physical exercise was substituted by electrical muscle stimulation (EMS) done on the Slimline Platinum 16 pad machine. In essence, this apparatus was an automatically cycling multiple output, faradic muscle stimulator which produced trains of pulses with variable pulse repetition frequency. The individual pulses were of short duration (0-5 millisecond) and of low energy, but at appropriate gain levels the pulse trains produced rhythmic and powerful muscular contractions when they were fed to the muscle by skin contact electrodes placed over or near the specific motor points. Pre study claims were made that repeated application of such pulse stimulation would produce breakdown of adipose tissue by localized passive exercise of the muscle unit, and so afford a generalized reduction in both size and weight.

As shown in Table 5.1 the most effective overall intervention modality when comparing groups was EST. EST had the greatest effect on anthropometry, morphology, ultrasound sonography and musculoskeletal function. Group TS had the greatest effect on pulmonary function and haematology and ESP had the greatest effect on respiratory quotient and musculoskeletal function. TS and ESP had the same effect on cardiovascular responses. When comparing total scores for number of variables tested both groups with thermogenic interventions (Groups EST and TS) had similar results. Electrical muscle stimulation, following a standardized diet and placebo controlled (Group ESP) also had an impact on physiological parameters but not as significant as group EST and TS. A more critical picture emerged, however, when only those variables showing significant differences in improvements between groups are considered (Table 5.2) to determine the greatest efficacy among the experimental groups.

TABLE 5.2: VARIABLES SHOWING DIFFERENCES* BETWEEN GROUPS

GIRTHS			
- Abdominal	EST = 6.02	>	TS = 4.69 ESP = 4.79
- AB-1 ½ Umbi	EST = 6.42	>	TS = 4.35 ESP = 4.28
SKINFOLDS			
- Triceps	EST = 12.75	>	TS = 9.27 ESP = 6.63
- Subscapular	EST = 9.70	>	TS = 8.64 ESP = 3.93
- Abdominal	EST = 12.14	>	TS = 11.80 ESP = 10.36
INDICES			
- Waist-to-Hip	EST = 2.53	>	TS = 1.27 ESP = 1.27
- Body Surface Area	EST = 3.03	>	ESP = 1.96
SONOGRAPHY			
- Subcutaneous	EST = 21.22	>	TS = 18.04 ESP = 12.11
- Visceral	EST = 27.74	>	TS = 21.87 ESP = 22.82
SAGGITAL HEIGHT			
- Saggital ½ Umbi	EST = 13.50	>	TS = 10.60 ESP = 10.61

*$p \leq 0,05$

Although the magnitude of improvements within groups tended to be greater in the TS group than in the ESP group, neither of these groups showed a statistically greater performance. The use of thermogenic stimulation (Group TS) and a standardized diet was thus as efficacious as the use of electrical muscle stimulation (Group ESP) and a standardized diet without thermogenic stimulation (placebo controlled) and vice-versa.

On the other hand group EST performed better than group TS (9 of 11 variables) and group ESP (10 of 11 variables). The use of electrical muscle stimulation in conjunction with thermogenic stimulation and a standardized diet thus showed greater

efficacy than the use of thermogenic stimulation only in conjunction with a standardized diet or electrical muscle stimulation only in conjunction with a standardized diet. A combination of the two primary modalities thus proved to be the most successful tools for weight-loss among obese woman.

Solving the problem of obesity within the context of this study thus appears to be three fold when considering changes within groups:.

- Energy expenditure through thermogenic stimulation in conjunction with calorie restriction (diet) has clinical and weight-loss relevance for the obese (Group TS).

- Electrical muscle stimulation in conjunction with calorie restriction (diet) has relevance for weight-loss in obese females (Group ESP).

- Electrical muscle stimulation in conjunction with thermogenic stimulation and calorie restriction (diet) has relevance for weight-loss in obese females (Group EST) and has been proven the most effective in this study.

5.2.2 Implication for weight-loss practice

Based on the results of this study obese individuals participating on a program of dietary restriction, thermogenic and electrical muscle stimulation with the aim of achieving weight-loss should note that:

- Diet in combination with electric muscle stimulation (EMS) is effective for weight-loss.

- Diet in combination with thermogenic stimulation, with/or without electrical muscle stimulation (EMS) proved the most effective intervention program after eight weeks.

5.2.3 Limitations of the study

Potentially limiting factors in this study which could be addressed in future studies are:

- Focus on a wider gender, cultural, ethnic and socio-economic diverse population;
- Identification of characteristics in individuals who are successful in weight reduction;
- Identification of appropriate and successful intervention content;
  - Magnitude of weight-loss goals
  - Goals for the rate of weight-loss
- Integration of behavioural methods with thermogenic and electrical muscle stimulation; and
- Extending the duration of the study for long-term observation.

5.3 FUTURE RESEARCH DIRECTIONS

Obesity is a heterogeneous chronic disorder that has many causes, although the fundamental basis is an imbalance between energy intake and energy expenditure. Future research needs to examine the most effective ways to treat and prevent obesity, the causes of obesity and their mechanisms, the influence of fat distribution on health risk, and the development of better methods for assessing energy intake and energy expenditure (NIH, 1998).

5.3.1 Assessment methods

Much of the current research is hampered by the lack of good methods to accurately, objectively, and economically assesses energy intake and expenditure, including physical activity, body composition and fat distribution, and behavioural and

psychological variables. More research is therefore needed to focus on measures to assess intake of fat and other dietary components, levels of physical activity, energy metabolism, and body fat and visceral obesity. In addition, better methods for assessment of psychological, behavioural, and psychosocial variables that may be related to behavioural risk factors for obesity (such as poor diet and inactive lifestyle) are needed, and particularly so for special population segments based on race, ethnicity, and socio-economic status. Methods for assessing culture, social integration, and psychological stress should also be developed (NIH, 1998).

5.3.2 Intervention approaches

Considerable research is needed on intervention approaches to treat and prevent obesity. Increased research on behavioural theory specifically addressing obesity treatment and prevention for all individuals, including children and adolescents, needs to be conducted. More research is needed on behavioural intervention methods conducted in various settings, particularly the primary care setting. Effective programs to treat or prevent obesity in culturally, ethnically, and socioeconomically diverse populations need to be developed and tested. Simple screening tools should be tested for their predictive value in achieving lifestyle modifications that lead to weight-loss or weight control practices. Research is needed on identifying appropriate and successful intervention content; for example, magnitude of weight-loss goals (smaller changes versus larger changes), and goals for the rate of weight-loss 0.5 kg versus 1.0 kg per week; initial weight-loss goal of 5 percent of body weight and, subsequently, an additional 5 percent versus a single initial goal of 10 percent at the outset). Of particular importance is research on the nature and optimal dose of physical activity to promote weight-loss, the maintenance of weight-loss and the prevention of obesity. Also important, are strategies which preserve muscle and bone in the face of weight-loss. More research is needed on identifying the characteristics of individuals who have successfully maintained their weight-loss over the long term. Research on pharmacologic interventions for weight-loss should include evaluating changes in fat distribution, cardiorespiratory fitness, obesity-related comorbidities, and the degree of success of long-term weight-loss maintenance. Better methods for integrating behavioural methods, along with pharmacologic treatment, should also be investigated. Finally, research is needed on environmental and population-based intervention methods, including community- and school-based interventions, to

augment public health approaches toward promoting weight maintenance and preventing obesity in the general population (NIH, 1998).

5.3.3 Causes and mechanisms of overweight and obesity

The regulation of energy balance needs to be explored, including the neuroendocrine factors that control energy intake, energy expenditure, and the differentiation of adipose tissue resulting from excess calories. The genes that are important in human obesity need to be identified. These include those that alter eating and physical activity behaviours, those that effect thermogenesis, and those associated with the comorbidities of obesity. The roles of environmental and behavioural influences on metabolic factors important in obesity, as well as gene-environment interactions, need to be studied. Predictive factors should be examined to identify who is most at risk of developing obesity, and whether there are critical periods of life when these factors are most operative (NIH, 1998).

5.3.4 Abdominal fat, body weight and disease risk

The influence of abdominal fat independent of total body fat on health risk needs to be further defined. More information is needed on the relationship between differential body fat compartments and increased risk, the distribution of body fat compartments among various racial group populations, and the relationship between abdominal fat and disease risk in racial groups. Weight-loss studies should include measurements of abdominal fat, as well as cardiorespiratory fitness, to better assess health improvement. Intentional weight-loss treatments need to be examined in terms of their acute and chronic effect on the development and progression of diabetes, heart disease, and overall mortality. Large prospective studies are needed to examine the relationship of body mass index and body fat distribution to overall mortality (NIH, 1998).

REFERENCES

ABRAMOWICZ M (1994).
Fluoxetine (Prozac) and other drugs for treatment of obesity. **The Medical Letter on Drugs and Therapeutics**. 36: 107-108.

ABRAMOWICZ M (1996).
Dehydroepiandrosterone (DHEA). **Medical Letter on Drugs and Therapeutics**. 38: 91-92.

ACHESON K J ; SCHUTZ Y; BESSARD T; FLATT J P; JÉQUIER E (1987).
Carbohydrate metabolism and de novo lipogenesis in human obesity. **American Journal of Clinical Nutrition**. 45: 78-85.

ALLISON D B; FAITH M S (1996).
Hypnosis as an adjunct to cognitive-behavioural psychotherapy for obesity: a meta-analytic reappraisal. **Journal of Consulting Clinical Psychology**. 64: 513-516.

ALLISON D B; FAITH M S; HEO M; KOTLER D P (1997).
Hypothesis concerning the U-shaped relation between body mass index and mortality. **American Journal of Epidemiology**. 146: 339-349.

ALLISON D B; FONTAINE KR; HESHKA 5; MENTORE J L (2001).
Alternative treatments for weight loss: a critical review. **Critical Reviews in Food Science and Nutrition**. 41: 2-28.

ALPERT M A; HASHIMI M W (1993).
Obesity and the heart. **American Journal of Medical Science**. 306: 117-123.

AMATRUDA J M; LIVINGSTONE J N; LOCKWOOD D N (1985).
Cellular mechanism in selected states of insulin resistance: human obesity, glucocorticoid excess and chronic renal failure. **Diabetes Metabolic Review**. 3: 293-317.

AMERICAN COLLEGE OF SPORTS MEDICINE (1990).
The recommended quantity and quality of exercise for developing and maintaining cardio respiratory and muscular fitness in healthy adults. **Medicine and Science in Sport and Exercise;** 22: 265-274.

AMERICAN DIETETIC ASSOCIATION (1997).
Position of the American Dietetic Association: weight management. **Journal of American Dietetic Association**. 97: 71-74.

AMERICAN PSYCHIATRIC ASSOCIATION (1994).
Task Forse on DSM-IV, Diagnostic and Statistical Manuel of Mental Disorders: DSM-IV. 4th ed. Washingtong, DC. The Association.

AMERICAN SOCIETY OF PLASTIC AND RECONSTRUCTIVE SURGEONS (1997).
1996 Average surgeon fees. **American Society of Plastic and Reconstructive Surgeons**. 1: 1-4.

AMNAR H (1954).
Growing up in an Egyptian Village. London. Routledge and Paul.

ANDERSON DA; WADDEN T A (1999).
Treating the obese patient. **Journal of the American Medical Association**. 8: 156-167.

ANDERSON R E; WADDEN T A; BARTLETT S J; ZEMEL B; VERDE T J
FRANCKOWIAK S C (1999).
Effects of lifestyle activity vs. structured aerobic exercise in obese women: a randomised trial. **Journal of American Medical Association**. 281: 335-340.

ANDRES R; MULLER D C; SORKIN J D (1993).
Long term effects of change in body weight on all-cause mortality. A review. **Annals of Internal Medicine.** 119: 737-743

AOKI T T; FINLEY R J (1986).
The metabolic response to fasting. In. **Parenteral Nutrition**. Rombeau J, Caldwell M. (eds.). Philadelphia. W B Saunders.

APFELBAUM M; BOSTSARRON J; LACATIS D (1971).
Effect of caloric restriction and excessive caloric intake on energy expenditure. **American Journal of Clinical Nutrition**. 24: 1404-1409.

ARNER P (1998).
Not all fat is alike. **Lancet**. 351: 1301-1302

ARTEAGA P; DOS SANTOS J E (1982).
Obesity among schoolchildren of different socioeconomic levels in a developing country. **International Journal of Obesity**. 6: 291-297.

ASHER W L; HARPER H W (1973).
Effect of human chorionic gonadotropin on weight loss, hunger, and feeling of well-being. **American Journal of Clinical Nutrition**. 26: 211-218.

ASHLEY F W; KANNEL W B (1974).
Relation of weight change to changes in atherogenic traits: the Framingham Study. **Journal of Chronic Diseases**. 27: 103-114.

ASHWELL M; McCALL S A; COLE T J; DIXON A K (1985).
Fat distribution and its metabolic complications: Interpretations. In: **Human body composition and fat distribution**. Norgan NG. Ed. Waginegen, Netherlands. Euronut.

ASTRUP A (1993).
Dietary composition, substrate balances and body fat in subjects with a predisposition to obesity. **International Journal of Obesity**. 17: 32-36.

ASTRUP A; BREUM L; TOUBRO S (1995).
Pharmacological and clinical studies of ephedrine and other thermogenic agonist. **Obesity Research**. 3: 537-540.

ASTRUP A; TOUBRO S; CHRISTENSEN N J; QUAADE F (1992).
Pharmacology of thermogenic drugs. **American Journal of Clinical Nutrition**. 55: 246-248.

ATKINS R C (1972).
Dr Atkins Diet Revolution. New York. Bantam Books.

ATKINSON R L (1986).
Issues and opinions in nutrition. **Journal of Nutrition**. 116: 918-919.

AUWERX J; STAELS B (1998).
Leptin. **Lancet**. 351: 737-742.

BABA S; ZIMMET P (1990).
World Data Book on Obesity. New York. Elsevier Science Publishers.

BABB T G (1999).
Mechanical ventilatory constraints in ageing, lung disease, and obesity: perspectives and brief review. **Medicine and Science in Sports and Exercise.** 31(1): 12-22.

BABB T G; BUSKIRK E R; HODGSON J L (1989).
Exercise end-expiratory lung volumes in lean and moderately obese women. **International Journal of Obesity**; 13: 11-19.

BAHR R. (1992).
Excess postexercise oxygen consumption. Magnitude, mechanisms and practical implications. **Acta Physiologica Scandinavia.** 605: 9-70.

BAILEY H R (1976).
Localized tissue reduction. **Medical Journal of Australia**. 1: 780-781.

BALLOR D L; KEESEY R E (1991).
A meta-analysis of the factor affecting exercise-induced changes in body mass, fat mass, and fat-free mass in males and females. **International Journal of Obesity**; 15: 717-726.

BARCUS E; MC LAUGHLIN L (1978).
Food Advertising on Children's Television: An Analysis of Appeals and Nutritional Content. Newtonville, MA. Action for Children's Television.

BAUMGARTNER R N; HEYMSFIELD S B; ROCHE A F (1995).
Human body composition and the epidemiology of chronic disease. **Obesity Research**. 3: 73-95.

BEESON V; RAY C; COXON R A; KREITZMAN S (1989).
The myth of the yo-yo: Consistent rate of weight loss with successive dieting by very low carbonhydrate diet. (VLCD). **International Journal of Obesity**; 13: 135-139.

BEGLEY C E (1991).
Government should strengthen regulation in the weight loss industry. **Journal of American Dietetic Association.** 91: 1255.

BELKO A Z; BARBIER T F; WONG E C (1986).
Effect of energy and protein intake and exercise intensity on the thermic effect of food. **American Journal of Clinical Nutrition.** 43: 863-868.

BENDER I; ARNOLD E; BROOKES L J (1987).
Body Weight Control: the Physiology, Clinical Treatment and Prevention of Obesity. Great Britain. Bath Press.

BENNETT W I(1995).
Beyond overeating. **New England Journal of Medicine**. 332: 673-674.

BENOTTI P N; FORSE R A (1995).
The role of gastric surgery in the multidisciplinary management of severe obesity. **American Journal of Surgery**. 169: 361-367.

BERCHTOLD P; JORGENS V; FINKE C; BERGER M (1981).
Epidemiology of obesity and hypertension. **International Journal of Obesity**. 5: 1-7.

BERG F M (1995).
Health risks of weight loss. **Healthy Weight Journal**. 3: 27-33.

BERGÖ M; OLIVECRONA G; OLIVECRONA T (1997).
Regulation of adipose tissue lipoprotein lipase in young and old rats. **International Journal of Obesity**. 21: 980-986.

BESSARD T; SCHUTZ Y; JÉQUIER E (1983).
Energy expenditure and postprandial thermogenesis in obese woman before and after weight loss. **American Journal of Clinical Nutrition**. 38: 680-693.

BIGARD A X; LIENHARD F; MERINO D (1993).
Effects of surface electrostimulation on the structure and metabolic properties in monkey skeletal muscle. **Medicine and Science in Sport and Exercise**. 25: 355-362.

BINGHAM S A; GOLDBERG G R; COWARD W A; PRENTICE A M; CUMMINGS J H (1989).
The effect of exercise and improved physical fitness on basal metabolic rate. **British Journal of Nutrition.** 61: 155-173.

BIRCH L (1992).
Children's preferences for high-fat foods. **Nutrition Review**. 50: 249-255.

BJÖRNTORP P (1988).
The association between obesity, adipose tissue distribution and disease. **Acta Medica Scandinavica**. 723: 121-134.

BJÖRNTORP P (1988).
Abdominal obesity and the development of non-insulin diabetes mellitus. **Diabetes and Metabolism Reviews**. 4: 615-622.

BJÖRNTORP P (1989).
Sex differences in the regulation of energy balance with exercise. **American Journal of Clinical Nutrition.** 49: 958-961.

BJÖRNTORP P (1991).
Metabolic implications of body fat distribution. **Diabetes Care**. 14: 1132-1143.

BJÖRNTORP P (1993).
Visceral obesity: A "civilization syndrome". **Obesity Research**. 1: 206-222.

BLACKBURN G L; WILSON G T; KANDERS B S; STEIN L J; LAVIN P T; ADLER J; BROWNELL K D (1989).
Weight cycling: The experience of human dieters. **American Journal of Clinical Nutrition**; 49: 1105-1109.

BLAIR D; HABRICHT J P; SIMS E A; SYLWESTER D; ABRAHAM S (1984).
Evidence for an increased risk for hypertension with centrally located body fat, and the effect of race and sex on this risk. **American Journal of Epidemiology**. 119: 526-540.

BLANCHARD, M.S. (1982).
Thermogenesis and its relationship to obesity and exercise. **Quest**; 34(2): 143-153.

BLUMENKRANTZ M (1998).
Obesity and Health. Website. (http://www.quantum.hcp.com/obesity.htm).

BODLEY J H (1985).
Anthropology and Contemporary Human Problems. Mountain View. Mayfield.

BOGERT L J; BRIGGS G M; CALLOUWAY D H (1973).
Nutrition and Physical Fitness (9th ed.). Philadelphia: Saunders.

BOSTICK R M; POTTER J D; KUSHI L H (1994).
Sugar, meat, and fat intake, and nondietary risk factors for colon cancer incidence in women (United States). **Cancer Causes Control**. 5: 38-52.

BOUCHARD C (1991).
Heredity and the path to overweight and obesity. **Medicine and Science in Sports and Exercise**; 23(3): 285-291.

BOUCHARD C (1994).
The Genetics of Obesity. Boca Raton, FL. CRC Press.
BOUCHARD C (1997).
Human variation in body mass: evidence for a role of the genes. **Nutritional Review.** 55: 21-30.

BOUCHARD C; BLAIR S N (1999).
Introductory comments for the consensus on physical activity and obesity. **Medicine and Science in Sport and Exercise**. 31: 498-501.

BOUCHARD C; PÉRUSSE L; RICE T; RAU D C (1998).
The genetics of human o besity. In: Bray G A; Bouchard C; James W P T (eds.). **Handbook of Obesity**. New York: Marcel Dekker.

BOUCHARD C; TREMBLAY A; DESPRÉS J P (1990).
The response to long-term overfeeding in identical twins. **New England Journal of Medicine.** 322: 1477-1482.

BOUCHARD C; TREMBLAY A; DESPRÉS J P (1994).
The response to exercise with constant energy intake in identical twins. **Obesity Research**. 5: 400-410.

BOURJEILY-HABR G; ROCHESTER C L; PALERMO F; SNYDER P; MOHSENIN V (2002).
Randomised controlled trial of transcutaneous electrical muscle stimulation of the lower extremities in patients with chronic obstructive pulmonary disease. **Thorax**. 57(12): 1045-1049.

BRAY G A (1969).
Effects of caloric restriction on energy expenditure in obese patients. **Lancet**; 2: 397-398.

BRAY G A (1987).
Overweight is risking fate: Definition, classification, prevalence, and risks. **New York Academy of Science.** 499: 14-28.

BRAY G A (1992).
Genetic, hypothalamic and endocrine features of clinical and experimental obesity. In. **Progress in Brain Research**. Swaab DF, Hofman MA, Mirmiran M (eds.). Amsterdam, The Netherlands. Elsevier Science Publishers.

BRAY G A (1993).
Use and abuse of appetite-suppressant drugs in the treatment of obesity. **Annals of Internal Medicine**. 119: 707-713.

BRAY G A (2003).
An Atlas of Obesity and Weight Control. New York. Parthenon Publishing Group.

BRAY G A; GRAY D S (1988).
Anthropometric measurements in the obese. In. **Anthropmetric standardization reference manuel**. Lohman TG, Roche AF, Martorell R (eds.). Champaign, IL. Human Kinetics.

BRAY G A; POPKIN B M (1998).
Dietary fat intake does affect obesity! **American Journal of Clinical Nutrition**. 68: 1157-1173.

BRAY G A; RYAN D H; GORDON D; HEIDINGSFELDER S(1996).
A double-blind randomised placebo-controlled trial of sibutramine. **Obesity Research**. 4: 263-270.

BRAY G A; YORK D A (1979).
Hypothalamic and genetic obesity in experimental animals: an autocrine and endocrine hypothesis. **Physiology Review**. 59: 719-809.

BREUM L; PEDERSEN J K; AHLSTROM F; FRIMODT-MOLLER J (1994).
Comparison of an ephedrine/caffeine combination and dexfenfluramine in the treatment of obesity: A double blind, multi-centre trial in general practice. **Journal of Obesity and Related Metabolic Disorders**. 18: 99-103.

BRINK P (1995).
Fertility and Fat: The Annany Fattening Room. In. **Social Aspects of Obesity**. Garine ID, Pollack NJ. (eds.). Amsterdam. Gordon and Breach Publishers.

BROOKS G A; FAHEY T D (1984).
Exercise Physiology: Human Bioenergetics and its Application. New York: John Wiley.

BROSS R; HOFFER L J (1995).
Fluoxetine increases resting energy expenditure and basal body temperature in humans. **American Journal of Clinical Nutrition**. 61:1020-1025.

BROUNS F (1993).
Nutritional Needs of Athletes. West Sussex, England: John Wiley & Sons.

BROWN C D; DONATO K A; OBARZANEK E (1998).
Body mass index and prevalence of risk factors for cardiovascular disease. **Obesity Research**. 4: 57-63.

BROWN D B (1998).
About Obesity. International Obesity Task Force (IOTF).
http://www.obesity.chair.alaval.ca/IOTF.htm

BROWN P J (1991).
Culture and the evolution of obesity. **Human Nature**. 2: 31-57.

BROWNELL K D; FAIRBURN C G (1995).
Psychosocial consequences of obesity. In: Stunkard AJ, Sobal JG (eds.). **Eating Disorders and Obesity: a Comprehensive Handbook**. New York. Guilford Press

BROWNELL K D; JEFFERY R W (1987).
Improving long-term weight loss: pushing the limits of treatment. **Behavioural Therapy**. 18: 353-374.

BROWNELL K D; STEEN S N; WILMORE J H (1987).
Weight regulation practices in athletes: Analysis of metabolic and health effects. **Medicine and Science in Sport and Exercise.** 19(6): 546-556.

BUCHOWSKI M; SUN M (1996).
Energy expenditure, television viewing and obesity. **International Journal of Obesity Related Metabolic Disorders**. 20: 236-244.

BUCKLEY D C; KUDSK K A; ROSE B (1987).
Transuitaneous muscle stimulation promotes muscle growth in immobilised patients. **Journal of Parenteral and Enteral Nutrition**. 11: 547-551.

BULLEN B A; REED R B; MAYER J (1964).
Physical activity of obese and non-obese adolescent girls, appraised by motion pictures sampling. **American Journal of Clinical Nutrition**. 14: 211-223.

BURSZTEIN S E; ELUGN D H; ASKANAZI J; KINNEY J M (1989).
Energy Metabolism, Indirect Calorimetry and Nutrition. Baltimore: Williams & Wilkins.

BUSKIRK E R (1974).
Obesity: a brief overview with emphasis on exercise. **Medicine and Science in Sport and Exercise**. 33(8):1948 – 1951.

CALLAWAY C W; CHUMLEA W C; BOUCHARD C; HIMES J H; LOHMAN T G; MARTIN A D; MITCHELL C D; MUELLER W H; ROCHE A F; SEEVELDT V D (1988).
Circumferences. In. Lohman TG, Roche AF, Martorel Rl (eds.). **Anthropometric standardization reference manual**. Champaign. Human Kinetics.

CAMPAIGNE B N (1990).
Body fat distribution in females: Metabolic consequences and implication for weight loss. **Medicine and Science in Sport and Exercise.** 22(3): 291-297.

CANNING H; MAYER J (1966).
Obesity: its possible effects on college admissions. **New England Journal of Medicicine**. 275: 1172-1174.

CAPWELL R R (1995).
Ephedrine-induced mania from an herbal diet supplement. **American Journal of Psychiatry**. 152: 647.

CARTER J E L (1992).
The Heath-Carter anthropometric somatotype instruction manual. San Diego. J E L Carter (Publisher).

CARTER J E L; HEATH B H (1990)
Somatotyping: development and applications. Cambridge. Cambridge University Press.

CENSUS, U S BUREAU (1994).
Statistical Abstract of the United States: Washington, DC. U S Bureau of the Census: 114th Edition.

CENTERS FOR DISEASE CONTROL AND PREVENTION (1994).
Daily dietary fat and total food-energy intakes – NHANES III, Phase I, 1988-1991. **Journal of American Medical Association**. 271:1309.

CHAN S H (1984).
What is being stimulated in acupuncture: evaluation of the existence of a specific substrate. **Neuroscience Biobehavioural Review**. 8: 25-33.

CHAPMAN B J; FARQUAHAR D L; GALLOWAY S M C L (1988).
The effects of a new beta-adrenoceptor agonist, BRL 26830A in refractory obesity. **International Journal of Obesity**. 12: 119-123.

CHARNEY E; GOODMAN H C; MC BRIDE M; LYON B; PRATT R (1975).
Childhood antecedents of adult obesity. **New England Journal of Medicine.** 295(1): 6-9.

CHASCIONE C; ELWYN D H; DAVILA M; GIL K M; ASKANAZI J; KINNEY J M (1987).
Effect of carbohydrate intake on de novo lipogenesis in human adipose tissue. **American Journal of Physiology**. 95: 469-472.

CHASE M (1994).
Oregon's new health rationing means more care for some but less for other. **Wall Street Journal**. January 28. 1-2.

CHIRICO A M; STUNKARD A J (1960).
Physical activity and human obesity. **New England Journal of Medicine**. 263: 935-940.

CIBA FOUNDATION (1996).
The Origins and Consequences of Obesity. New York: John Wiley.

CICUTTINI F M; BAKER J R; SPECTOR T D (1996).
The association of obesity with osteoarthritis of the hand and knee in women: a twin study. **Journal of Rheumatology**. 23: 1221-1226.

CINCOTTA A H; MEIER A H (1989).
Reduction of body fat stores and total plasma cholesterol and triglyceride concentrations in several species by bromocriptine treatment. **Life Science**. 45: 2247-2254.

CINCOTTA A H; MEIER A H (1996).
Bromocriptine (Ergoset) reduces body weight and improves glucose tolerance in obese subject. **Diabetes Care**. 19: 667-670.

CLANCY S P; CLARKSON P M ; DE CHEKE M E; NOSAKA K (1994).
Effects of chromium picolinate supplementation on body composition, strength and urinary chromium loss in football players. **International Journal of Sport Nutrition**. 4:142-153.

CLARYS J P; MARTIN A D; DRINKWATER D T; MARFELL-JONES M J (1987).
The skinfold: Myth and reality. **Journal of Sports Sciences**. 5: 3-33.

CLEMENT K (1998).
A mutation in the human leptin receptor gene causes obesity and putuitary dysfunction. **Nature**. 392: 398-404.

CLEMENT K; GARNER C; HAGER J; PHILIPPI A (1996).
Indication for linkage of the human ob gene region with extreme obesity. **Diabetes.** 45: 687-690.

CLEMENT K; PHILIPPI A; JURY C; PIVDAL R (1996).
Candidate gene approach of familial morbid obesity-linkage analysis of the glucocorticoid receptor gene. **International Journal of Obesity.** 20: 507-512.

CNATTINGIUS S; BERGSTROM R; LIPWORTH L; KRAMER M S (1998).
Prepregnancy weight and the risk of adverse pregnancy outcomes. **New England Journal of Medicine**. 338: 147-152.

COCHRANE G (1992).
Hypnosis and weight reduction: which is the cart and which is the horse? **American Journal of Clinical Hypnosis**. 35: 108-118.

COLDITZ G A; WILLETT W C; ROTNITZKY A; MANSON J E (1995).
Weight gain as a risk factor for clinical diabetes mellitus in women. **Annals of Internal Medicine**. 122: 481-486.

COLE AD; BOND NW (1983).
Olfactory aversion conditioning and overeating: a review and some data. **Perceptual and Motor Skills**. 57: 667-668.

COLEMAN D L (1973).
Effects of parabiosis of obese with diabetes and normal mice. **Diabetologica.** 9: 294-298.

COLLINS J K (1987).
Methodology for the objective measurement of body image. **International Journal of Eating Disorders**. 6: 393-399.

COLLINS V; DOWSE G (1990).
Prevalence of obesity in Pacific and Indian Ocean populations. In. **World Data Book on Obesity**. Baba S, Zimmet P.(eds.). New York. Elsevier Science Publishers.

CONNACHER A A; BENNETT W M; JUNG R T (1992).
Clinical studies with the beta-adrenoreceptor agonist BRL 26830A. **American Journal of Clinical Nutrition**. 55: 258-261.

CONNACHER A A; JUNG R T; MITCHELL P E G (1988).
Weight loss in obese subjects on a restricted diet given BRL 26830A, a new atypical beta adrenoceptor agonist. **British Medical Journal**. 296: 1217-1220

COOPER C B; STORER T W (2001).
Exercise Testing and Interpretation: a Practical Guide. Cambridge University Press. United Kingdom.

CORTEX M Y; TORGAN C E; BROZINICK J T; MILLER R H; IVY J L (1991).
Effects of pyruvate and dihydroxyacetone consumption on the growth and metabolic state of obese zucker rats. **American Journal of Clinical Nutrition**. 53: 847-853.

COULSTON A M; HOLLENBECK C B; SWISLOCKI A L M; CHEN Y D I; REAVAN G M (1987).
Deleterious effects of high-carbohydrate, sucrose-containing diets in patients with non-insulin dependent diabetes mellitus. **American Journal of Medicine**. 82: 213-220.

CRANDALL C S; MARTINEZ R (1996).
Culture, ideology, and antifat attitudes. **Personality, Social Psycholgy Bulletin**. 22: 1165-1176.

DALLASSO H M; JAMES W P (1984).
Whole-body calorimetry studies in adult men. The interaction of exercise and over-feeding on the thermic effect of a meal. **British Journal of Nutrition**; 52: 65-72.

DARE G L; GOLDNEY R D (1976).
Fenfluramine abuse. **Medical Journal of Australia**. 2: 537-540.

DEBUSK R F; STENESTRAND U; SHEEHAM N; HASKELL W L (1990).
Training effects of long versus short bouts of exercise in healthy subjects. **American Journal of Cardiology**. 65: 1010-1013.

DE GROOT L C G M; VAN ES A J H; VAN RAAJI J M A; VOGT J E; HAUTVAST J G A J (1990).
Energy metabolism of overweight women 1 mo. and 1 y. after an 8-wk slimming period. **American Journal of Clinical Nutrition**. 51: 578-583.

DENKE M A; SEMPOS C T; GRUNDY S M (1994).
Excess body weight. An underrecognized contributor to dyslipidemia in white American women. **Archives of Internal Medicine**. 154: 401-410.

DESPRÉS J P (1991).
Lipoprotein metabolism in visceral obesity. **International Journal of Obesity**. 15: 45-52.

DESPRÉS J P; FONG B S; JULIEN P; JIMENEZ J; ANGEL A (1987).
Regional variation in HDL metabolism in human fat cells. Effect of fat cell size. **American Journal of Physiology**. 252: 654-659.

DESPRÉS J P; LEMIEUX S; LAMARCHE B; PRUDHOMME D; MOORJANI S; BRUN L D; GAGNÉ C; LUPIEN P J (1995).
The insulin resistance-dyslipidemic syndrome: contribution of visceral obesity and therapeutic implications. **International Journal of Obesity**. 19: 76-86.

DESPRÉS J P; MOORJANI S; LUPIEN P J; TREMBLAY A; NADEAU A; BOUCHARD C (1990).
Regional distribution of body fat, plasma lipoproteins, and cardiovascular disease. **Atherosclerosis**. 10: 497-511.

DESPRÉS J P; NADEAU A; TREMBLAY A (1989).
Role of deep abdominal fat in the association between regional adipose tissue distribution and glucose tolerance in obese women. **Diabetes**. 38: 304-309.

DE ZWAAN M; MITCHELL J E (1992).
Opiate antagonists and eating behavior in humans: A review. **Journal of Clinical Pharmacology**. 32: 1060-1072

DIAMOND H; DIAMOND M (1987).
Fit for Life. New York. Warner Books.

DIEHR P; BILD D E; HARRIS T B; DUXBURY A; SISCOVICK D; ROSSI M (1998).
Body mass index and mortality in non-smoking older adults: The cardiovascular health study. **American Journal of Public Health**. 88: 623-629.

DIETZ W H (1987).
Childhood obesity. **New York Academy of Science**; 499: 47-54

DIETZ W H; GORTMAKER S L (1985).
Do we fatten our children at the television set? Obesity and television viewing in children and adolescents. **Pediatrics**. 75(5): 807-812.

DILLERUD E; H ÁHEIM L L (1993).
Long-term results of blunt suction lipectomy assessed by patient questionnaire survey. **Plastic and Reconstructive surgery**. 92: 35-38.

DI LORENZO C; WILLIAMS C M; HAJNAL F; VALENZUELA J E (1988).
Pectin delays gastric emptying and increase satiety in obese subjects. **Gastroenterology**. 95: 1211-1215.

DITSCHUNEIT H H; FLECHTNER-MORS M; JOHNSON T D; ADLER G (1999).
Metabolic and weight-loss effects of a long-term dietary intervention in obese patients. **American Journal of Clinical Nutrition**. 69: 198-204.

DOBBING J (1987).
Sweetness. London. Springer-Verlag.

DONAHUE R P; ABBOT R D; BLOOM E; REED D M; YANO K (1987).
Central fat distribution in middle-aged men and the risk of coronary heart disease in men. **Lancet**. 1: 822-824.

DONNELLY J E; JACOBSEN D J; SNYDER HEELAN K; SELP R; SMITH S (2000).
The effects of 18 months of intermittent vs. continuous exercise on aerobic capacity, body weight and composition, and metabolic fitness in previously sedentary, moderately obese females. **International Journal of Obesity**. 24: 566-572.

DRENICK E J; SWENDSEID M E; BLAHD W H (1964).
Prolonged starvation as treatment for servere obesity. **Journal of American Medical Association**. 187: 100-106.

DRENT M L; LARSSON I; WILLIAM-OLSSON T W; QUAADE F (1995).
Orlistat (RO 18-0647), a lipase inhibitor, in the treatment of human obesity: A multiple dose study. **International Journal of Obesity**. 19: 221-226.

DRENT M L; VAN DER VEEN E A (1993). Lipase inhibition. A novel concept in the treatment of obesity. **International Journal of Obesity**. 17: 241-244.

DRENT M L; WEVER L D; ADER H J; VAN DER VEEN E A (1995).
Growth hormone administration in addition to a very low calorie diet and an exercise program in obese subjects. **Europian Journal of Endocrinology**. 132: 565-572.

DREWNOWSKI A (1997).
Why do we like fat? **Journal of American Dietetic Association**. 97: 58-62.

DREWNOWSKI A; POPKIN B (1997).
The nutrition transition: new trends in the global diet. **Nutrition Review**. 55(2): 31-43.

DU BOIS D; DU BOIS E F (1916).
Clinical calorimetry: A formula to estimate the approximate surface area if height and weight be known. **Archives of Internal Medicine**. 17: 863-871.

DULLO A G; GIRARDIER L (1990).
Adaptive changes in energy expenditure during refeeding following low calorie intake: evidence for a specific metabolic component favouring fat storage. **American Journal of Clinical Nutrition**. 52: 415-420.

DULLO A G; MILLER D S (1985).
The Do-Do pill: Potentiation of the thermogenic effects of ephedrine by methylxanthines. **International Journal of Obesity**. 10: 160-165.

DUNAIF A (1992).
Polycystic Ovary Syndrome. Boston. Blackwell Scientific Publications.

DUNCAN J J; GORDON N F; SCOTT C B (1991).
Women walking for health and fitness: how much is enough? **Journal of American Medical Association**. 266: 3295-3299.

DUNN A L; MARCUS B H; KAMPERT J B; GARCIA H W; KOHL II; BLAIR S N (1999).
Comparison of lifestyle and structured interventions to increase physical activity and cardio respiratory fitness. **Journal of American Medical Association**. 281: 327-334.

DU PLESSIS L A S (2000).
Lifestyle, body fat distribution and insulin-related coronary heart disease risk factors in hypertensive females. Unpublished D Phil thesis. Pretoria. University of Pretoria.

DURNIN J V; NORGAN N G (1969).
Overfeeding. **Journal of Physiology**. 202: 106.

DURNIN J V; WOMERSLEY J (1974).
Body fat assessed from body density and its estimation from skinfold thickness: Measurements on 481 men and women aged from 16 to 72 years. **British Journal of Nutrition**. 32: 77-97.

DUZMAK L I(1991).
Stoma adjustable silicone gastric banding. **Surgical Rounds**. 14:19-28.

DWYER J T (1980).
Sixteen popular diets: brief nutritional analyses. In. **Obesity**. Stunkard AJ (ed.). Philadephia. W B Saunders.

DWYER J T; LU D (1993).
Popular Diets for Weight loss. From Nutritionally Hazardous to Healthful. In. **Obesity: Theory and Therapy**. 2nd ed. Stunkard AJ, Wadeen TA (eds.). New York. Raven Press.

DYER A R; ELLIOTT P (1989).
The INTERSALT study: relations of body mass index to blood pressure. INTERSALT co-operative Research Group. **Journal of Human Hypertension**. 3: 299-308.

ELLIOT D L (1989).
Sustained depression of the resting metabolic rate after massive weight loss. **American Journal of Clinical Nutrition**; 49: 93.

ERICKSON J C; HOLLOPETER G; PALMITER R D (1996).
Attenuation of the obesity syndrome of ob/ob mice by the loss of neuropeptide Y. **Science.** 274: 1704-1707.

ERNST E (1997).
Acupuncture/acupressure for weight reduction? A systematic review. **Wiener Klinisch Wochenschrift**. 109: 60-62.

FEINLEIB M (1985).
Epidemiology of obesity in relation to health hazards. **Annals of Internal Medicine**. 103: 1019-1024.

FELBER J P; FERRANNINI E; GOLAY A (1987).
Pole of lipid oxidation in pathogenesis of insulin resistance of obesity and type II diabetes. **Diabetes**. 36: 1341-1350.

FELSON D T; ZHANG Y; ANTHONY J M; NAIMARK A; ANDERSON J J (1992).
Weight loss reduces the risk for symptomatic knee osteoarthritis in women. The Framingham Study. **Annals of Internal Medicine**. 116: 535-539.

FERRARO R; BOYCE V L; SWINBURN B; DE GREGORIO M; RAVUSSIN E (1991).
Energy cost of physical activity on a metabolic ward in relationship to obesity. **American Journal of Clinical Nutrition**. 53: 1368-1371.

FISHER M C; LACHANCE P A (1985).
Nutrition evaluation of published weight-reduction diets. **Journal of American Dietetic Association**. 85: 450-454.

FISLER J S (1992).
Cardiac effects in starvation and semistarvation diets. **American Journal of Clinical Nutrition**. 56: 230-234.

FITZGIBBON M I; STOLLEY M R; KIRSCHENBAUM D B (1993).
Obese people who seek treatment have different characteristics than those who do not seek treatment. **Health Psychology**. 12: 342-345.

FOLSOM A R; KAYE S A; SELLERS T A; HONG C P; CERHAN J R; POTTER J D PRINEAS R J (1986).
Body fat distribution and 5-year risk of death in older woman. **Journal of the American Medical Association.** 269(4): 483-487.

FOOD AND AGRICULTURE ORGANIZATION/WORLD HEALTH ORGANIZATION/ UNITED NATIONS UNIVERSITY (1985).
Energy and protein requirements. WHO Technical Report Series No. 724. Geneva: WHO.

FORBES G B (1990).
Do obese individuals gain weight more easily than non-obese individuals? **American Journal of Clinical Nutrition**. 52: 224-227.

FORD E S; WILLIAMSON D F; LIU S (1997).
Weight change and diabetes incidence: findings from a national cohort of US adults. **American Journal of Epidemiology**. 146: 214-222.

FOREYT J; GOODRICK K (1995).
The ultimate triumph of obesity. **Lancet**. 346: 134-135.

FOSS M L; KETEYIAN S J (1998).
Fox's Physiological Basis for Exercise and Sport (6th ed.). New York: McGraw-Hill.

FOX E L; BOWERS R W; FOSS M L (1993).
The Physiological Basis of Physical Education and Athletics. (4th ed.). Iowa Wm. C. Brown.

FOX E L; BOWERS R W; FOSS M L (1993).
The Physiological Basis for Exercise and Sport. (5th ed.). Iowa. Wm. C. Brown.

FRANK L D; ENGELKE P (2000).
How land use and transportation systems impact public health. Atlanta, GA Centers for Disease Control. Georgia Institute of Technology.

FRAYN K N (1995).
Physiological regulation of macronutrient balance. **International Journal of Obesity**. 19: 4-10.

FRIED L P; KRONMAL R A; NEWMAN A B (1998).
Risk factors for 5-year mortality in older adults (for the cardiovascular Health Study Collaborative Research Group. **Journal of American Medical Association**. 279: 585-592.

FRIEDMAN J M (1996).
The alphabet of weight control. **Nature.** 385: 119-120.

GALLAGHER D; HEYMSFIELD S B (1994).
Obesity is bad for the heart, but is weight loss always good? **Obesity Research**. 2:160-163.

GARAVAGLIA G E; MESSERLI F H; NUNEZ B D; SCHMIEDER R E; GROSSMAN E (1988).
Myocardial contractility and left ventricular function in obese patients with essential hypertension. **American Journal of Cardiology**. 62: 594-597.

GARBACIAK J A; RICHTER M; MILLER S; BARTON J J (1985).
Maternal weight and pregnancy complications. **American Journal of Obstetrics and Gynecology**. 152: 238-245.

GARFINKEL L (1986).
Overweight and mortality. **Cancer**. 58: 1826-1829

GARMEZY N (1983).
Stressors of childhood. In. **Stress, Coping, and Development in Children**. Garmezy N, Rutter M. (eds.). New York. McGraw Hill.

GARN S M; CLARK D C (1976).
Trends in fatness and the origin of obesity. **Pediatrics**. 57: 443-456.

GARNER D M (1991).
Obesity treatment: more harm than good. **Journal of American Dietetic Association**. 91: 152.

GARNER D M; WOOLEY S C (1991).
Confronting the failure of behavioral and dietary treatments for obesity. **Clinical Psychology Review**. 11: 729-780.

GARROW J S (1978).
The regulation of energy expenditure in man. In. **Recent Advances in Obesity Research**. Bray GA (ed.). Vol 2. London. Newman.

GARROW J S (1988).
Obesity and Related Diseases. (2nd ed). New York. Churchill Livingstone.

GELIEBTER A; MAHER M M; GERACE L; GUTIN B; HEYMSFIELD S B; HASHIM S A (1997).
Effects of strength or aerobic training on body composition, resting metabolic rate, and peak oxygen consumption in obese dieting subjects. **American Journal of Clinical Nutrition**. 66: 557-563.

GILLUM R F (1987).
The association of body fat distribution with hypertension, hypertensive heart disease, coronary heart disease, diabetes, and cardiovascular risk factors in men and women aged 18-79 years. **Journal of Chronic Disease**. 40: 421-428.

GIOVANNUCCI E; COLDITZ G A; STAMPFER M J; WILLETT W C (1996).
Physical activity, obesity, and risk of colorectal adenoma in women (United States). **Cancer Causes Control**. 7: 253-263.

GIOVANNUCCI E (1995).
Insulin and colon cancer. **Cancer Causes Control**. 6: 167-179.

GITTELSOHN J; HARRIS S B; THORNE-LYMAN A L; HANLEY A J; BARNIE A; ZINMAN B (1996).
Body image conceps differ by age and sex in an Ojibway-Cree community in Canada. **Journal of Nutrition**. 126: 2990-3000.

GLICKMAN N M; MITCHELL H H; LAMBERT E H; KEETON W (1948).
The total specific dynamic action of high-protein and high-carbohydrate diets of human subjects. **Journal of Nutrition.** 36: 41-57.

GOLDBLATT P B; MOORE M E; STUNKARD A J (1965).
Social factors in obesity. **Journal of American Medical Association**. 192: 1039-1044.

GOLDSTEIN D J; RAMPEY A H; DORNSEIF B E; LEVINE L R (1993).
Fluoxetine: A randomised clinical trial in the maintenance of weight loss. **Obesity Research**. 2: 92-98.

GOLDSTEIN D J; RAMPEY A H; ENAS G G; POTVIN J H (1994).
Fluoxetine: A randomised clinical trial in the treatment of obesity. **International Journal of Obesity**. 18: 129-135.

GOODMAN A H; LEATHERMAN T L (1998).
Building a New Biocultural Synthesis: Politic-Economic Perspective on Human Biology. Ann Arbor. University of Michigan Press.

GOODMAN N; DORNBUSCH S M; RICHARDSON S A; HASTORF A H (1963).
Variant reactions to physical disabilities. **American Sosiology Review**. 28: 4299-435.

GORDON R (1987).
An operational classification of disease prevention. In. **Preventing Mental Disorders: A Research Perspective. U.S. Department of Health and Human Services, Public Health Service**. Steinberg JA, Silverman MM (eds.). Washington, D.C. Government Printing Office.

GRAY D S; BRAY G A; BAUER M; KAPLAN K; GEMAYEL N; WOOD R; GREENWAY R; KIRK S (1990).
Skinfold thickness measurements in obese subjects. **American Journal of Clinical Nutrition**. 51: 571-577.

GULICK A (1922).
A study of weight regulation in the adult human body during over-nutrition. **American Journal of Physiology**. 60: 371-395.

GUPTA R K; SAINI D P (1994).
Impact of television on children. **Indian Journal of Pediatrics**. 61: 153-159.

GURNEY M; GORNSTEIN J (1988).
The global prevalence of obesity: an initial overview of available data. **Word Health Statistics Quarterly** (Rapport Trimestriel de Statistiques Sanitaires Mondales). 41: 251-254.

GUY-GRAND B (1992).
Log-term pharmacological treatment of obesity. In. **Treatment of the Seriously Obese Patient**. Wadden TA, Vanltalie TB (eds.). New York: Guilford Press. (478-495).

GWINUP G (1971).
Thickness of subcutaneous fat and activity of underlying muscles. **Annals of Internal Medicine**. 74: 408.

HADVARY P; LENGSFELD H; WALFER H (1988).
Inhibition of pancreatic lipase in vitro by the covalent inhibitor tetrahydrolipstatin. **Biochemistry Journal**. 256: 357-361.

HAFNER R J; ROGERS J (1990).
Husbands adjustment to wives weight loss after gastric restriction for morbid obesity. **International Journal of Obesity**. 14:1069-1078.

HARTZ A J; BARBORIAK P N; WONG A; RIMM AA (1979).
The association of obesity with infertility and related menstrual abnormalities in women. **International Journal of Obesity**. 3: 57-73.

HARTZ A J; FISCHER M E; BRIL G; KELBER S; RUPLEY D; OKEN B; RIMM A A (1986).
The association of obesity with joint pain and osteoarthritis omn the HANES data. **Journal of Chronic Diseases**. 39: 311-319.

HASKELL W L (1996).
Physical activity, sport and health: toward the next century. **Research Quarterly for Exercise and Sport**. 67: 37-47.

HASTEN D L; ROME E P; FRANKS B D; HEGSTED M (1992).
Effects of chromium picolinate on beginning weight training students. **International Journal of Sport Nutrition**. 2: 343-349.

HAUNER H; BOGNAR E; BLUM A (1994).
Body fat distribution and its association with metabolic and hormonal risk factors in women with angiographically assessed coronary artery disease. Evidence for the presence of a metabolic syndrome. **Atherosclerosis**. 105: 209-216.

HAWLEY J A (1998).
Fat burning during exercise. **The Physician and Sports Medicine.** 26 (9): 56-62.

HEALTH 24 (2004).
Fat, fatter, South African? Obesity tops health risk list. **Health 24.co.za**

HECHT K (1990).
Oh, come on fatties. **Newsweek**. Sept 3:8.

HEITMANN B L; KAPRIO J; HARRIS J R; RISSANEN A; KORKEILA M; KOSKENVUO M (1997).
Are genetic determinants of weight gain modified by leisure-time physical activity? A prospective study of Finnish twins. **American Journal of Clinical Nutrition**. 66: 672-678.

HERMAN C P; ZANNA M P; HIGGINS E T (1986).
Physical Appearance, Stigma and Social Behavior. Hillsdale, N J. L Erlbaum.

HERVEY G R (1952).
The effects of lesions in the hypothalmus in parabiotic rats. **Journal of Physiology.** 145: 336-352.

HEYWARD V H; STOLARCZYK L M (1996).
Applied Body Composition Assessment. Champaign. Human Kinetics.

HILL J O; PETERS J C (1998).
Environmental contribution to the obesity epidemic. **Science**. 280: 1371-1374.

HIMMS-HAGEN J (1984).
Thermogenesis in brown adipose tissue as an energy buffer. **New England Journal of Medicine.** 311(24): 1549-1558.

HIRSCH A R (1993).
Inhalation of 2-acelylpyridine for weight reduction. **Chemical Senses**. 18: 570.

HIRSCH J; LEIBEL R L (1970).
New light on obesity. **New England Journal of Medicine.** 318: 509.

HOCKEY R V (1993).
Physical Fitness: The Pathway to Healthful Living. (7th ed.) Missouri Mosby.

HORTON E S (1985).
Metabolic aspects of exercise and weight reduction. **Medicine and Science in Sport and Exercise**; 18(1): 10-18.

HORTON T J; DROUGAS H (1995).
Fat and carbohydrate overfeeding in humans: different effects on energy storage. **American Journal of Clinical Nutrition**. 62: 19-29.

HOVELL M F (1988).
Long term weight loss maintenance: assessment of behavioral and supplemental fasting regimen. **American Journal of Public Health**; 78: 663.

HOWEL D C (1992)
Statistical Methods for Psychology. (3rd ed.). California. Duxbury Press.

HUANG Z; HANKINSON S E; COLDITZ G A (1997).
Dual effects of weight and weight gain on breast cancer risk. **Journal of American Medical Association**. 278: 1407-1411.

HUBERT H B; FEINLEIB M; MC NAMARA P M; CASTELLI W P (1983).
Obesity as an independent risk factor for cardiovascular disease: a 26-year follow-up of participants in the Framingham Heart Study. **Circulation**. 67: 968-977.

HUXTABLE R J (1992).
The myth of beneficient nature: The risks of herbal preparations. **Annals of Internal Medicine**. 117: 165-166.

HYMAN F N; SEMPOS E; SALTSMAN J; GLINSMANN W H (1993).
Evidence for success of caloric restriction in weight loss and control: summary of data from industry. **Annals of Internal Medicine**. 119: 719-721.

INSEL P A (1996).
Adrenergic receptors - evolving concepts and clinical implications. **New England Journal of Medicine**; 334: 580-585.

INSTITUTE OF MEDICINE (IOM) (1990).
Committee on Nutritional Status During Pregnancy and Lactation. Nutrient Supplements. Washington, D C. National Academic Press.

INSTITUTE OF MEDICINE (IOM) (1992).
Eat for Life: The Food and Nutrition Board's Guide to Reducing your Risk of Chronic Disease. Woteki C E & Thomas P R (eds.). Washington, D.C. National Academy Press.

INSTITUTE OF MEDICINE (IOM) (1994).
Reducing Risks for Mental Disorders: Frontiers for Preventive Intervention Research. Report of the Committee on Prevention of Mental Disorders, Division of Biobehavioral Sciences and Mental Disorders. Washington, D.C. National Academy Press.

INSTITUTE OF MEDICINE (IOM) (1995).
Weighing The Options: Criteria for Evaluating Weight-Management Programs. Washington, DC. National Academy Press.

INUI A (1999).
Neuropeptide Y feeding receptors: are multiple subtypes involved? **TINS**. 20: 43-46.

JACKSON A S; POLLOCK M L (1978).
Generalized equations for predicting body density of men. **British Journal of Nutrition**. 40: 497-504.

JACKSON A S; POLLOCK M L; WARD A (1980).
Generalized equations for predicting body density of women. **Medicine and Science in Sports and Exercise**. 12: 175-182.

JACOBS D B; SOWERS J R (1993).
Effects of weight reduction on cellular metabolism and vascular resistance. **Hypertension**. 21: 308-314.

JAKICIC J M; CLARK K; COLEMAN E; DONNELLY J E; FOREYT J; MELANSON E; VOLEK J; VOLPE S L (2001).
Appropriate intervention strategies for weight loss and prevention of weight regain for adults. **American College of Sports Medicine**. 33: 2145-2156.

JAKICIC J M; DONNELLY J E; JAWAD A F; JACOBSEN D J; GUNDERSON S C; PASCALE R (1993).
Association between blood lipids and different measures of body fat distribution: Effects of BMI and age. **International Journal of Obesity**. 17: 131-137.

JAKICIC J M; WING R R; BUTLER B A; ROBERTSON R J (1995).
Prescribing exercise in multiple short bouts versus one continuous bout: effects on adherence, cardiorespiratory fitness, and weight loss in overweight women. **International Journal of Obesity**. 19: 893-901.

JAKICIC J M; WINTERS C; LANG W; WING R R (1999).
Effects of intermittent exercise and use of home exercise equipment on adherence, weight loss and fitness in overweight woman: a randomised trial. **Journal of American Medical Association**. 282: 1554-1560.

JAMES W P (1995).
A public health approach to the problem of obesity. **International Journal of Obese Related Metabolic Disorders**. 19: 37-45.

JEFFERY R W (1993).
Minnesota studies on community-based approaches to weight loss and control. **Annals of Internal Medicine**. 119: 719-721.

JEFFERY R W; FRENCH S A (1998).
Epidemic obesity in the United States: are fast foods and television viewing contributin? **American Journal of Public Health**. 88: 277-280.

JELLIFFE R; HILL D; TATTER D; LEWIS E (1969).
Death from weight-control pills: A case report with objective post-mortem confirmation. **Journal of American Medical Association**. 208: 1843-1847.

JEQUIER E (1987).
Energy, obesity, and body weight standards. **American Journal of Clinical Nutrition**; 45: 1035-1047.

JUST B; MESSING B; DARMAUN B; RONGIER M; CARMILLO E (1990).
Comparison of substrate utilization by indirect calorimetry during cyclic and continuous total parenteral nutrition. **American Journal of Clinical Nutrition**. 51: 107-111.

KAHN H S; WILLIAMSON D F (1990).
The contributions of income, education and changing marital status to weight change among US men. **International Journal of Obesity**. 14: 1057-1068.

KATAHN M (1989).
The T-Factor Diet. New York. Bantam Books.

KATTUS A A; BISCOE B W; DASHE A M; DAVIS J H (1968).
Spurious heart disease induced by digitalis containing reducing pills. **Archives of Internal Medicine**. 122: 298-304.

KAWATE R; YAMAKIDO M; NISHIMOTO Y (1979).
Diabetes mellitus and its vascular complications in Japanese migrants on the Island of Hawaii. **Diabetes Care**. 29: 161-170.

KEESEY R (1996).
Physiologigal regulation of body energy: implication for obesity. In: Stunkard A J, Wadden T A (eds.). **Obesity: Theory and Therapy**. 2nd ed. Philadelpia: Lippincotl-Raven.

KEMPER K A; SARGENT R G; DRANE J W; VALOIS R F; HUSSEY J W (1994).
Black and white females perceptions of ideal body size and social norms. **Obesity Research**. 2: 117-126.

KERN P A (1997).
Potential role of TNF-α-and lipoprotein lipase as candidate genes for obesity. **Journal of Nutrition**. 127: 1917-1922.

KESSEY R E (1986).
A set-point theory of obesity. In. **Handbook of Eating Disorders**. Brownell K.D. and Foreyt J.P. (eds.). New York. Basic Books.

KEYS A (1980).
Seven Countries. **A Multivariate Analysis of Health and Coronary Heart Disease**. Cambridge, MA. Harvard University Press.

KEYS A; BROZEK J; HENSCHEL A; MICKELSON O; TAYLOR H L (1950).
The biology of human starvation. Vol 1. Minneapolis. University of Minnesota Press.

KIENS B; ESSEN-GUSTAVSSON B; CHRISTENSEN N J; SALTIN B (1993).
Skeletal muscle substrate utilization during submaximal exercise in man: Effects of endurance training. **Journal of Physiology.** 469: 459-478.

KIESSEBAH A H; KRAKOWER G R (1994).
Regional adiposity and morbidity. **Physiology Review**. 74: 775-811.

KING A C; TRIBBLE D L (1991).
The role of exercise in weight reduction in non-athletes. **Sports Medicine**. 11: 331-349.

KING A C; HASKELL W L; YOUNG D R; OKA R K; STEFANICK M L (1995).
Long-term effects of varying intensities and formats of physical activity on participation rates, fitness and lipoproteins in men and woman aged 50 to 65 years. **Circulation**. 91: 2596-2604.

KING J R (1994).
Scientific status of aromatherapy. **Perspective of Biological Medicine**. 37: 409-415.

KLEIN S (2000).
Medical treatment of obesity. **Problems in General Surgery**. 17(2): 1-6.

KLEM M L; WING R R; MC GUIRE M T; SEAGLE H M; HILL J 0(1997).
A descriptive study of individuals successful at long-term maintenance of substantial weight loss. **American Journal of Clinical Nutrition**. 66: 239-246.

KLUTE R; SCHUBERT A (1985).
Obesity in Europe. **Animals of Internal Medicine**. 103: 1037-1042.

KOJIMA M; HOSODA H; DATE Y; NAKAZATO M; MATSUO H; KANGAWA K (1999).
Ghrelin is a growth-hormone-releasing acylated peptide from stomach. **Nature**. 402: 656-660.

KOPELMAN P G (1984).
Clinical complications of obesity. **Clinical Endocrinology and Metabolism**. 13(3): 613-635.

KOPLAN J P (2000).
The obesity epidemic: trends and solutions. **Sports Medicine Bulletin**. 35(3):8.

KOZOL R A; FROMM D; ACKERMANN N B; CHUNG R (1986).
Wound closure in obese patients. **Surgery, Gynecology and Obstetrics**. 162: 442-444.

KRAEMER W J; VOLEK J S; CLARK K L (1997)
Physiological adaptations to a weight-loss dietary regimen and exercise programs in women. **Journal of Applied Physiology**. 83: 270-279.

KRAEMER W J; VOLEK J S; CLARK K L (1999).
Influence of exercise training on physiological and performance changes with weight loss in men. **Medicine and Science in Sport and Exercise**. 31: 1320-1329.

KRAL J G (1992).
Surgical treatment of obesity. In. **Treatment of the Seriously Obese patient**. Wadden TA, Vanltallie TB (eds.). New York. Guilford Press.

KRAL J G (1994).
Side-effects, complications and problems in anti-obesity surgery. **International Journal of Obesity**. 18: 86.

KRAMER F M; JEFFERY R W; FORSTER J L; SNELL M K (1989).
Long-term follow-up of behavioural treatment for obesity: patterns of weight regain in men and women. **International Journal of Obesity**. 13: 123-126.

KUEHNEL R H; WADDEN T A (1994).
Binge eating disorder, weight cycling, and psychopathology. **International Journal of Eating Disorders**. 15: 321-329.

KULKARNI S K; KAUR G (1999).
Obesity: an insight into its neurochemical basis and treatment. **Indian Journal of Pharmacology**. 31: 388-403.

KUMANYIKA S (1994).
Obesity in minority populations: An epidemiologic assessment. **Obesity Research**. 2: 166-182.

KUMANYIKA S; MORSSINK J (1997).
Cultural appropriateness of weight management programs. In. **Overweight and Weight Management: The Health Professional's Guide to Understanding and Practice**. Dalton S, (ed.). Gaithersburg, MD. Aspen Publishers.

KUMANYIKA S; WILSON J F; GUILFORD-DAVENPORT M (1993).
Weight-related attitudes and behaviors of black women. **Journal of American Dietetic Association**. 93: 416-422.

LAMBERT V (2002).
Slothful lifestyle fattens up SA teens. **Sunday Times**. Sunday 10 March 2002.

LANDIN K; STIGENDAL L; ERIKSSON E (1990).
Abdominal obesity is associated with an impaired fibrinolytic activity and elevated plasminogen activator inhibitor-I. **Metabolism**. 39: 1044-1048.

LANDSBERG L; KIEGER D R (1989).
Obesity, metabolism and the sympathetic nervous system. **American Journal of Hypertension**. 2: 125-132.

LAPIDUS L; BENGTSSON C; HALLSTROM T; BJÖRNTORP P (1989).
Obesity, adipose tissue distribution and health in women – results from a population study in Gothenburg, Sweden. **Appetite**. 12: 25-35.

LAPIDUS L; BENGTSSON C; LARSSON-PENNERT K; RYBO E; SJÖSTRÖM L (1984).
Distribution of adipose tissue and risk of cardiovascular disease and death: a 12 year follow-up of participants in the population study of women in Gothenburg, Sweden. **British Medical Journal**. 289: 1261-1263.

LARKIN J C; PINES H A (1979).
No fat persons need apply: experimental studies of the overweight stereotype and hiring preference. **Sociology of Work and Occupations**. 6: 312-327.

LARREY D; VIAL T; PAUWELS A; CASTOT A (1992).
Hepatitis after germander (Teucrium chamaedrys) administration. Another instance of herbal medicine hepatotoxicity. **Annals of Internal Medicine**. 117:129-132

LARSSON B; BJÖRNTORP P; TIBBLIN G (1981).
The health consequences of moderate obesity. **International Journal of Obesity**. 5:97-116.

LAW M R; WALD N J; THOMPSON S G (1994).
By how much and how quickly does reduction in serum cholesterol concentration lower risk of ischaemic heart disease? **British Medical Journal**. 308: 367-773.

LEE C D; JACKSON A S; BLAIR S N (1998).
US weight guidelines: is it also important to consider cardio respiratory fitness? **International Journal of Obesity**. 22: 2-7.

LEE E T; HOWARD B V; SAVAGE P J (1995).
Diabetes and impaired glucose tolerance in three American Indian populations aged 45-74 years. The Strong Heart Study. **Diabetes Care**. 18:599-610.

LEE I M; MANSON J E; HENNEKENS C H; PAFFENBARGER R S (1998).
Body weight and mortality. A 27-year follow-up of middle-aged men. **Journal of American Medical Association**. 270: 2823-2828.

LEIBEL R L; HIRSCH J (1984).
Diminished energy requirements in reduced-obese patients. **Metabolism**. 33: 164-170.

LEIBEL R L; ROSENBAUM M; HIRSCH J (1995).
Changes in energy expenditure resulting from altered body weight. **New England Journal of Medicine**. 332: 621-628.

LEMONICK M D (2002).
Lean and hungrier. A digestive-system hormone called ghrelin plays a bigger role in weight control than anyone thought. **Time**. June: 54.

LEVEILLE G (1985).
Set Point Diet. New York. Ballantine Books.

LEVIN B E; ROUTH V H (1996).
Role of the brain in energy balance and obesity. **American Journal of Physical Anthropology.**. 271:491-500.

LEW E A; GARFINKEL L (1979).
Variations in mortality by weight among 750,000 men and women. **Journal of Chronic Diseases**. 32: 563-576.

LIN B H; FRAZAO E (1997).
Nutritional quality of foods at and away from home. **Food Review**. 2: 33-40.

LINDPAINTER K (1995).
Finding an obesity gene - a tale of mice and man. **New England Journal of Medicine**. 332. 679-680.

LISSAU I; SORENSEN T I (1994).
Parental neglect during childhood and increased risk of obesity in young adulthood. **Lancet**. 343: 324-327.

LIU *Z;* SUN F; LI J; WANG Y; HU K (1993).
Effect of acupuncture on weight loss evaluated by adrenal function. **Journal of Traditional Chinese Medicine**. 13: 169-173.

LOHMAN T G (1989).
Assessment of body composition in children. **Pediatric Exercise Science**; 1: 19-30.

LOHMAN T G; ROCHE A F; MARTORELL R (1988).
Anthropometric Standardization Reference Manual. Champaign, IL. Human Kinetics.

LOSONCZY K G; HARRIS T B; CORNONI-HUNTLEY J (1995).
Does weight loss from middle age to old age explain the inverse weight mortality relation in old age? **American Journal of Epidemiology**. 141: 312-321.

LOUBE D I; LOUBE A A; MITLER M M (1994).
Weight loss for obstructive sleep apnea: the optimal therapy for obese patients. **Journal of American Dietetic Association**. 94: 1291-1295.

LOVEJOY J C; BRAY G A; BOURGEOIS M 0; MACCHIAVELLI R (1996).
Exogenous androgens influence body composition and regional body fat distribution in obese post-menopausal women. A clinical research center study. **Journal of Clinical Endocrinology Metabolism**. 81: 2198-2203.

LUNDGREN H; BENGTSSON C; BLOHME G; LAPIDUS L; SJÖSTRÖM L (1989).
Adiposity and adipose tissue distribution in relation to incidence of diabetes in women: results from a prospective population study in Gothenburg, Sweden. **International Journal of Obesity**. 13: 413-423.

MADDOX G L; LIEDERMAN V (1969).
Overweight as a social disability with medical implications. **Journal of Medical Education.** 44: 214-220.

MALCHOW-MOLLER A; LARSEN S; HEY H; STOKHOLM K H (1981).
Ephedrine as an anorectic: The story of the "Elsinore pill". **International Journal of Obesity**. 5:183-187.

MALOUF N; COLAYUIRI S (1995).
The effects of McDonalds, Kentucky Fried Chicken and Pizza Hut meals on recommended diets. **Asia Pacific Journal of Clinical Nutrition**. 4: 265-269.

MALINA R M; BOUCHARD C (1991).
Growth, Maturation and Physical Activity. Champaign. IL: Human Kinetics.

MANSON J E; COLDITZ G A; STAMPFER M J (1990).
A prospective study of obesity and risk of coronary heart disease in women. **New England Journal of Medicine**. 322: 882-889.

MANSON J E; STAMPFER M J; HENNEKENS C H; WILLETT W C (1987).
Body weight and longevity. A reassessment. **Journal of American Medical Association**. 257: 353-358.

MARCUS M D; WING R R; EWING L; KERN E; GOODING W; MC DERMOTT M (1990).
Psychiatric disorders among obese binge eaters. **International Journal of Eating Disorders**.

MARCUS M D; WING R R; FAIRBURN C G (1995).
Cognitive treatment of binge eating versus behavioral weight control in the treatment of binge eating disorder. **Annals of Behavioral Medicine**. 17: 90-91.

MARIEB E N (1992).
Human Anatomy and Physiology (2nd ed.). Redwood City, CA. Benjamin Cummings.

MARKS B L; WARD A; MORRIS D H; CASTELLANI J; RIPPE J M (1995).
Fat-free mass is maintained in women following a moderate diet and exercise program. **Medicine and Science in Sport and Exercise**. 27: 1243-125l.

MARLATT G A; LARIMER M E; BAER J S; QUIGLEY L A (1993).
Harm reduction for alcohol problems: Moving beyond the controlled drinking controversy. **Behavioral Therapy**. 24: 461-503.

MARLATT G A; TAPERT S F (1993).
Harm reduction: Reducing the risks of addictive behaviors. In. **Addictive Behaviors Across the Lifespan**. Baer JS, Marlatt GA, McMahan R. (eds.). Newbury Park, CA. Sage Publications.

MARROW J R; JACKSON A W; DISCH J G; MOOD D P (1995).
Measurement and Evaluation in Human Performance. Champaign, IL. Human Kinetics.

MARTINDALE: THE EXTRA PHARMACOPOEIA (1997).
The Royal Pharmaceutical Society of Great Britain.

MARTINI F H (1995).
Fundamentals of Anatomy and Physiology. (3rd ed.). New Jersey. Prentice Hall.

MASON P (1997).
Obesity – new insights into a growing problem. **Pharmaceutical Journal**. 258: 800-802.

MASSARA E B; STUNKARD A J (1979).
A method of quantifying cultural ideals of beauty and the obese. **International Journal of Obesity**. 3: 149-152.

MC ARDLE W D; KATCH F I; KATCH V L (1996).
Exercise Physiology: Energy Nutrition and Human Performance. (4th ed.). Baltimore, Maryland. Williams & Wilkins.

MC ARDLE W D; TONER M M (1988).
Application of exercise for weight control: the exercise prescription. In **Obesity and Weight Control**. Frankle RT, Yong MU (eds.). Rockville, Aspen.

MC CRORY M A; HAYS N P; VINKEN A G; GREENBERG A S; ROBERTS S B (1999).
Overeating in America: Association between restaurant food consumption and body fatness in healthy adult men and women ages 19 to 80. **Obesity Research**. 7: 564-571.

MC GOEY B V; DEITEL M; SAPLYS R J; KLIMAN M E (1990).
Effect of weight loss on musculoskeletal pain in the morbidly obese. **Journal of Bone and Joint Surgery**. 72: 322-323.

MC NEIL G; BRUCE A C; RALPH A; JAMES W P T (1988).
Interindividual differences in fasting nutrient oxidation and the influence of diet composition. **International Journal of Obesity**. 12: 445-463.

MC TAVISH D; HEEL R C (1992).
A review of its pharmacological properties and therapeutic potential in obesity. **Drugs**. 43: 713-733.

MELBY C; SCHOLL C; EDWARDS G; BULLUGH R (1993).
Effect of acute resistance exercise on postexercise energy expenditure and resting metabolic rate. **Journal of Applied Physiology**; 75(4): 1847-1853.

MEIER A H; CINCOTTA A H; LOVELL W C (1992).
Timed bromocriptine administration reduces body fat stores in obese subject and hyperglycemia in type II diabetes. **Experientia**. 48: 248-253.

MENTZ N W (2000).
Effect of Continuous Assistive-Passive Exercise on Physiological Parameters among Obese Females. Unpublished MA(HMS) dissertation. Pretoria. University of Pretoria.

MERTZ W (1993).
Chromium in human nutrition. A review. **Journal of Nutrition**. 123: 625-633.

MILLER C L (1993).
Hruby VI, α-MSH and MCH are functional antagonists in a central nervous system auditory gating paradigm. **Annals of New York Academic Science**. 680: 571-574.

MILLER D S (1985).
A controlled trial using ephedrine in the treatment of obesity. **International Journal of Obesity**. 10: 159-160.

MILLER D S; MUMFORD P (1967).
Gluttony. An Experimental study of overeating low- or high-protein diets. **American Journal of Clinical Nutrition**. 20: 1212-1222.

MILLER P M; SIMS K L (1981).
Evaluation and component analysis of a comprehensive weight control program. **International Journal of Obesity**. 5: 57.

MILLER W C; EGGERT K E; WALLACE J P; LINDEMAN A K; JASTREMSKI C (1993).
Successful weight loss in a self-taught, self-administered program. **International Journal of Sports Medicine**. 14: 401-405.

MILLER W C; KOCEJA D M; HAMILTON E J (1997).
A meta-analysis of the past 25 years of weight loss research using diet, exercise, or diet plus exercise intervention. **International Journal of Obesity**. 21: 941-947.

MILLER W C; LINDEMAN A K (1997).
The role of diet and exercise in weight management. In **Overweight and Weight Management: The Health Professional's Guide to Understanding and Practice**. Gaithersburg, M.D. (ed.). Ohio. Aspen Publishers.

MILLER W C; NIEDERPRUEM M G; WALLACE J P; LINDEMAN A K (1994).
Dietary fat, sugar, and fiber predict body fat content. **Journal of American Dietetic Association**. 94: 612-615.

MILLER W C; WALLACE J P; EGGERT K E; LINDEMAN A K (1993).
Cardiovascular risk reduction in a self-taught, self-administered weight-loss program called the nondiet diet. **Medicine, Exercise, Nutrition and Health**. 2: 218-223.

MOKDAD A H; DIETZ W H; BOWMAN B A; MARKS J S; KOPLAN J P (1999).
The spread of the obesity epidemic in the United States. **Journal of American Medical Association**. 282: 1519-1522.

MONTAGUE C T; FAROOQUI I S; WHITEHEAD J P; SOOS M A; RAU H; WAREHAM N J (1997).
Congenital leptin deficiency is associated with severe early-onset obesity in humans. **Nature**. 387: 903-908.

MORRISSEY M C; BREWSTER C E; SHIELDS C L (1985).
The effects of electrical stimulation on the quadriceps during postoperative knee immobilization. **American Journal of Sports Medicine**. 13: 40-45.

MUSSELL M P; PETERSON C B; WELLER C L; CROSBY R D; DE ZWAAN M; MITCHELL J E (1996).
Differences in body image and depression among obese women with and without binge eating disorder. **Obesity Research**. 4: 431-439.

NADIR A; AGRAWAL 5; KING P D; MARSHALL J B (1996).
Acute hepatitis associated with the use of a Chinese herbal product, ma-huang. **American Journal of Gastroenterology**. 91: 1436-1438.

NAIR K S; HALLIDAY D; GARROW J S (1983).
Thermic response to isoenergetic protein, carbohidrate or fat meals in lean and obese subjects. **Clinical Science**; 66:307-312.

NAKAZATO M; MURAKAMI N; DATE Y; KOJIMA M; MATSUO H; KANYAWA K; MATSUKURA S (2001).
A role for ghrelin in the central regulation of feeding. **Nature**. 409: 194-198.

NASLUND 1(1994).
Effects of weight reduction on the somatic psychological and social complications of morbid obesity. **International Journal of Obesity**. 18: 86-87.

NATIONAL CHOLESTEROL EDUCATION PROGRAM (1994).
Second Report of the Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel II). **Circulation**. 89: 1333-1445.

NATIONAL INSTITUTES OF HEALTH (NIH) 1992.
Gastrointestinal surgery for severe obesity: National Institutes of Health Consensus Development Conference Statement 1991 March 25-27. **American Journal of Clinical Nutrition**. 55: 615-619.

NATIONAL INSTITUTES OF HEALTH (NIH) 1998.
Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults: the evidence report. **National Institutes of Health Publication**. 98: 51-210.

NATIONAL INSTITUTES OF HEALTH CONSENSUS DEVELOPMENT CONFERENCE STATEMENT (1985).
Health implications of obesity. **Annals of Internal Medicine**. 103: 1073-1077.

NATIONAL TASK FORCE ON THE PREVENTION AND TREATMENT OF OBESITY (1993).
Very low-caloric diets. **Journal of American Medical Association**. 270: 967-976.

NATIONAL TASK FORCE ON THE PREVENTION AND TREATMENT OF OBESITY (1994).
Towards Prevention of Obesity: Research Directions. **Obesity Research**. 2: 571-584.

NATIONAL TASK FORCE ON THE PREVENTION AND TREATMENT OF OBESITY (1996).
Long-term pharmacotherapy in the management of obesity. **Journal of American Medical Association**. 276: 1907-1915.

NEEL J V (1962).
Diabetes mellitus: A "thrifty" genotype rendered detrimental by "progress". **American Journal of Human Genetics.** 14: 353-362.

NEILL J R; MARSHALL J R; YALE C E (1978).
Marital changes after intestinal bypass surgery. **Journal of American Medical Association**. 240: 447-450.

NEO-BARINE (1964).
Medical Letter on Drugs and Therapeutics. 6: 50-51.

NEUMANN R O (1902).
Experimentelle beitraege zur lehre von dem taeglichen nahrungsbedarf des menschen unter besonderer beruecksichtigung der notwendigen eiweissmenge. **Archiv fur Hugiene und Bakteriologie**. XLV: 1-87.

NEWSHOLME E A (1980).
A possible metabolic basis for the control of body weight. **New England Journal of Medicine**; 302(7): 400-405.

NEWSHOLME E A; LEECH A R (1983).
Biochemistry for the Medical Sciences. New York: John Wiley.

NICHTER M (2000).
Fat Talk: What Girls and Their Parents Say About Dieting. Cambridge. Harvard University Press.

NIH TECHNOLOGY ASSESSMENT CONFERENCE PANEL (1993).
Methods for voluntary weight loss and control. **Annals of Internal Medicine**. 119: 764-770.

NORGAN N G; DURNIN J V (1980).
The effect of 6 weeks of overfeeding on the body weight, body composition, and energy metabolism of young men. **American Journal of Clinical Nutrition**. 33: 978-988.

OESER D (1997).
Obesity Part 1: Epidemiology, Etiology and Pathophysiology and Nonpharmacotherapeutic Treatments. **Internet Journal of Academic Physician Assistants.** 1(2).

OESER D (1997).
Obesity Part 2: Pharmacotherapy. **Internet Journal of Academic Physician Assistants**. 1(2).

OLEFSKY J M (1994).
Obesity. In. **Harrison's Principles of Internal Medicine**. Isselbacher K.J.; Braunwald E.; Wilson J.D.; Martin J.B.; Fauci D.L.; Kasper (eds.). (13th ed.). New York. McGraw-Hill.

OLSON C L; SCHUMAKER H D; YAWN B P (1994).
Overweight women delay medical care. **Archive of Family Medicine**; 10: 888-892.

O'NEIL P M; JARRELL M P (1992).
Psychological aspects of obesity and dieting. In. **Treatment of The Seriously Obese Patient**. Wadden TA; Van Itallie TB (eds.). New York. Guilford Press.

OOMURA Y; INOUE S; SHIMAZA T (1999).
Progress in Obesity Research. London. John Libbey.

ORNISH D (1992).
Dr Dean Ornish's Program for Reversing Heart Disease: The Only System Scientifically Proven to Reverse Heart Disease Without Drugs or Surgery. New York. Ballantine Books.

ORNISH D (1993).
Eat More, Weight Less. New York. Harper Collins.

PAFFENBARGER R S; WING A L; HYDE R T (1978).
Physical activity as an index of heart attack risk in college alumni. **American Journal of Epidemiology**. 108: 161-175.

PATE R R; PRATT M; BLAIR S N (1995).
Physical activity and public health: a recommendation from the Centers for Disease Control and Prevention and the American College of Sports Medicine. **Journal of American Medical Association**. 273: 402-407.

PAVLOU K N; KREY S; STEFFEE W P (1989).
Exercise as an adjunct to weight loss and maintenance in moderately obese subjects. **American Journal of Clinical Nutrition**. 49: 1115-1123.

PENA M; BACALLAO J (2000).
Obesity and Poverty. Pan American Health Organization. Scientific Publication. Washington, DC. PAHO.

PÉRONNET F G; THIBAULT M; LEDOUX M & BRISSON G (1987).
Performance in Endurance Events: Energy Balance, Nutrition, and Temperature Regulation in Distance Running. London, Spodym.

PERRI M G; McADOO W G; McALLISTER (1987).
Effects of peer support and therapist contact on long-term weight loss. **Journal of Clinical Psychology**. *55:* 615-617.

PERRI M G; NEZUAM (1993).
Preventing relapse following treatment for obesity. In. **Obesity: Theory and Therapy.** (2nd ed.)**.** Stunkard AJ, Wadden TA (eds.). New York. Raven Press.

PHILLIPS M; KUBISCH D (1985).
Lifestyle diseases of Aborigines. **Medical Journal of Australia**. 143: 218.

PILLET P H; GYGAZ P H; JEQUIER E (1974).
Thermic effect of glucose and amino acids in man studied by direct and indirect calorimetry. **British Journal of Nutrition**; 31: 343-349.

PINGITORE R; DUGONI B L; TINDALE R S; SPRING B (1994).
Bias against overweight job applicants in a simulated employment interview. **Journal of Applied Psychology**. 79: 909-917.

PI-SUNYER F X (1993).
Medical hazards of obesity. **Annals of Internal Medicine**. 119: 655-660.

PLOWMAN S A; SMITH D L (1997).
Exercise Physiology for Health, Fitness and Performance. Boston. Allan and Bacon.

POEHLMAN E T (1989).
A Review: Exercise and its influence on resting energy metabolism in man. **Medicine and Science in Sports and Exercise**; 21(5): 515-525.

POLKOW B (1988).
Stress: hypothalamic functions and neuroendocrine consequences. **Acta Phisiologica Scandinavia**. 723: 61-70.

PONDIMIN PRESCRIBING INFORMATION (1994).
A H Robins Company, Richmond.

POPPITT S D (1995).
Energy density of diets and obesity. **International Journal of Obesity**. 5: 20-26.

PORCARI J P; FOSTER C; CRENSHAW B; SWENSON C (2002).
Effects of electrical muscle stimulation on body composition, muscle strength, and physical appearance. **The Journal of Strength and Conditioning Research**. 16(2): 165-172.

POSTREL V (2001)
Americans' waistlines have become the victims of economic progress. **The New York Times**. March. 22.

POWDERMAKER H (1961).
An anthropological approach to the problem of obesity. **Bulletin of the New York Academy of Science**. 36: 286-295

POWELL A D; KAHN A S (1995).
Racial differences in women's desires to be thin. **International Journal of Eating Disorders**. 17: 191-195.

PRATHER R C; WILLIAMSON D A (1988).
Phychopathology associated with bulimia, binge eating, and obesity. **International Journal of Eating Disorders**. 7: 177-184.

PRENTICE A M; GOLDBERG G (1996).
Maternal obesity increases congenital malformations. **Nutrition Review**. 54: 146-152.

PRENTICE A M; JEBB S A (1995).
Obesity in Britain: gluttony or sloth? **British Medical Journal**. 311: 437-439.

PRICE R A; NESS R; SORENSEN T I A (1991).
Change in commingled body mass index distribution associated with secular trends in overweight among Danish young men. **American Journal of Epidemiology**. 133: 501-510.

PRITIKIN N (1982).
The Pritikin Permanent Weight Loss Manual. New York. Bantam Books.

PRONK N P; WING R R (1994).
Physical activity and long-term maintenance of weight loss. **Obesity Research**. 2: 587-599.

PUTMAN R (2002).
Bowling Alone: The Collapse and Revival of American Community. New York. Simon and Schuster.

QUARFORDT S H; FRANK A; SHAMES D M; BERMAN M; STEINBERG D (1970).
Very low density lipoprotein triglyceride transport in type IV hyperlipoproteinemia and the effect of carbohydrate rich diets. **Journal of Clinical Investigation**. 44: 1826-1833.

RAND C S W; KULDAU J M; ROBBINS L (1982).
Surgery for obesity and marriage equality. **Journal of American Medical Association**. 247: 1419-1422.

RAND C S W; MACGREGOR A M C (1991).
Morbidly obese patients perceptions of social discrimination before and after surgery for obesity. **Southern Medical Journal**. 83: 1390-1395.

RAND C S W; MACGREGOR A M C (1991).
Successful weight loss following obesity surgery and the perceived liability of morbid obesity. **International Journal of Obesity**. 15: 70-75.

RAND C S W; ROBBINS L; KULDAU J M (1983).
Stressful life events and the decision for surgery for obesity. **Psychosomatics**. 24: 534-539.

RAVUSSIN E; BOGARDUS C (1989).
Relationship of genetics, age, physical fitness to daily energy expenditure and fuel utilization. **American Journal of Clinical Nutrition**. 49: 968-975.

RAVUSSIN E; HARPER I; RISING R; FERRARO R (1991).
Human obesity is associated with lower levels of physical activity: results from doubly labeled water and gas exchanges. **FASEB Journal**. 5: A554.

RAVUSSIN E; LILLIOJA S; ANDERSON T E; CHRISTIN L; BOGARDUS C (1986).
Determinants of 24-hour energy expenditure in man: methods and results using a respiratory chamber. **Journal of Clinical Investigation**. 78: 1568-1579.

RAVUSSIN E; SCHUTZ Y; ACHESON K J; DUSMET M; BOURQUIN L; JÉQUIER E (1985).
Short-term, mixed diet overfeeding in man: no evidence for "Luxuskon sumption". **American Journal of Physiology**. 249: 470-477.

RAVUSSIN E; SWINBURN B A (1992).
Effect of calorie restriction and weight loss on energy expenditure. In. **Treatment of the seriously obese patient**. Wadden TA, Van Itallie TB (eds.). New York. Guilford Press.

RAVUSSIN E; VALENCIA M E; ESPARZA J; BENNETT P H; SCHULTZ L O (1994).
Effects of a traditional lifestyle on obesity in Pima Indians. **Diabetes Care**. 17: 1067-1074.

REAVEN G M; HILL D B; GROSS R C; FARQHUAR J W (1965).
Kinetics of triglyceride turnover of very low density lipoproteins of human plasma. **Journal of Clinical Investigation**. 44: 1826-1833.

REDUX PRESCRIBING INFORMATION (1996).
Wyeth Laboratories, Inc., Philadelphia.

REED P; DING Y; ZU W; CATHER C (1996).
Extreme obesity may be linked to markers flanking the human ob gene. **Diabetes**. 45: 691-694.

REEDER B A; ANGEL A; LEDOUX M; RABKIN S W; YOUNG T K; SWEET L E (1992).
Obesity and its relation to cardiovascular disease risk factors in Canadian adults. **Canadian Heart Health Surveys Research Group**. **Canadian Medical Association Journal**. 146: 2009-2019.

REISIN E; FROHLICH E D; MESSERLI F H; DRESLINSKI G R (1983).
Cardiovascular changes after weight reduction in obesity hypertension. **Annals of Internal Medicine**. 98: 315-319.

REXRODE K M; HENNEKENS C H; WILLET W C (1997).
A prospective study of body mass index, weight change and risk of stroke in women. **Journal of American Medical Association**. 277: 1539-1545.

RICE T; TREMBLAY A; DERIAZ 0; PERUSSE L; RAO D C; BOUCHARD C (1996).
A major gene for resting metabolic rate unassociated with body composition: results from the Québec Family Study. **Obesity Research**; 4: 441-449.

RICHARDS D; MARLEY J (1998).
Stimulation of auricular acupuncture points in weight loss. **Australian Family Physician**. 2: 73-77.

RICH-EDWARDS J W; GOLDMAN M B; WILLETT W C (1994).
Adolescent body mass index and infertility caused by ovulatory disorder. **American Journal of Obstetrics and Gynecology**. 171: 171-177.

RIPPE J M; CROSSLEY S; RINGER R (1998).
Obesity as a chronic disease: Modern medical and lifestyle management. **Journal of American Dietetic Association**. 98: 9-5
.
RISING R; HARPER I; FERRARO R; FONTVIEILLE A M; RAVUSSIN E (1991).
Effect of age on free-living energy expenditure in Pima Indians. **FASEB Journal**. 5: A550.

RITENBAUGH C (1982).
Obesity as a culture-bound syndrome. **Culture, Medicine and Psychiatry**. 6: 347-361.

ROBERTS S B; YOUNG V R; FUSS P (1990).
Energy expenditure and subsequent nutrient intakes in overfed young men. **American Journal of Physiology**. 259: 461-469.

ROBINSON T (1998).
Does television cause childhood obesity. **Journal of American Medical Association**. 279: 959-960.

ROMIJN J A; COYLE E F; SIDOSSIS L S; GASTALDELLI A; HOROWITZ J F; ENDERT E; WOLFE R R (1993).
Regulation of endogenous fat and carbohydrate metabolism in relation to exercise intensity and duration. **American Journal of Physical Anthropology**; 265: 380-391.

ROSEN J C; GROSS J (1987).
Prevalence of weight reducing and weight gaining in adolescent girls and boys. **Health Psychology**. 6: 131-147.

ROSENBERG L; PALMER J R; MILLER D R; CLARKE E A; SHAPIRO S (1990).
A case-control study of alcoholic beverage consumption and breast cancer. **American Journal of Epidemiology**. 131: 6-14.

ROSS C E; MIROWSKY J (1983).
The social epidemiology of overweight: a substantive and methodological investigation. **Journal of Health and Social Behaviour**. 24: 288-298.

ROSS R (1997).
Effects of diet and exercise-induced weight loss on visceral adipose tissue in men and woman. **Sports Medicine**. 24: 55-64.

ROSS R; DAGNONE D; JONES P J (2000).
Reduction in obesity and related co-morbid conditions after diet-induced weight loss or exercise-induced weight loss in men: a randomised, controlled trial. **Annals of Internal Medicine**. 133: 92-103.

ROSS R; FREEMAN J A; JANSSEN I (2000).
Exercise alone is an effective strategy for reducing obesity and related co morbidities. **Exercise and Sport Science Review**. 28: 165-170.

RUCKER C E; CASH T F (1992).
Body images, body size perception, and eating behaviors among African-American and white college woman. **International Journal of Eating Disorders**. 12: 291-300.

RYAN D H; KAISER P; BRAY G A (1995).
Sibutramine: A novel new agent for obesity treatment. **Obesity Research**. 3: 553-559.

RYDKER P M; HENNEKENS C H; STAMPFER M J (1993).
A prospective study of lipoprotein (a) and the risk for myocardial infarction. **Journal of American Medical Association**. 270: 2195-2199.

SAMANIN R; GARATTINI S (1993).
Neurochemical mechanism of action of anorectic drugs. **Pharmacology and Toxicology**. 73: 63-68.

SARIS W H M (1996).
Physical activity and body weight regulation. In: **Regulation of Body Weight: Biological and Behavioral Mechanisms**. Bouchard C, Brag G A (eds.). New York . Wiley.

SCHACHTER S (1982).
Recidivism and self-cure of smoking and obesity. **American Journal of Psychology**. 37: 436-444.

SCHELKUN P H (1991).
The risks of riding the weight-loss roller coaster. **The Physician and Sportsmedicine**; 19(6): 149-156.

SCHIFFMAN S; GRAHAM B G (1998).
Orosensory perception of dietary fat. **Current Directions in Psychological Science**. 7: 137-143.

SCHOELLER D A; SHAY K; KUSHNER R F (1997).
How much physical activity is needed to minimize weight gain in previously obese woman? **American Journal of Clinical Nutrition**. 66: 551-556.

SCHOELLER D A; WEBB P (1984).
Five day comparison of the doubly labeled water method with respiratory gas exchange. **American Journal of Clinical Nutrition**. 40: 143-158.

SCHOTTENFELD D; FRAUMENI J F (1996).
Cancer Epidemiology and Prevention. New York. Oxford University Press.

SCHREIBER G G; ROBINS M; STRIEGEL-MOORE R; OBARZANEK E; MORRISON J A; WRIGHT D J (1996).
Weight modification efforts reported by black and white preadolescent girls: National Heart, Lung, and Blood Institute Growth and Health Study. **Pediatrics**. 98: 63-70.

SCHWARTZ M W; SEELEY R J (1997).
The new biology of body weight regulation. **Journal of American Dietetic Association.** 97: 54-58.

SCHWARTZ R S; HALTER J B; BIERMAN E L (1983).
Reduced thermic effect of feeding in obesity: Role of nor-epinephrine. **Metabolism**; 32: 114-117.

SCOPINARO N; GIANETTA E; CIVALLERI D; BONALUMI U; BACHI V (1980).
Two years experience with biliopancreatic bypass for obesity. **American Journal of Clinical Nutrition**. 33: 506-514.

SEIDELL J C (1995).
The impact of obesity on health status: some implications for health care costs. **International Journal of Obese Related Metabolic Disorders**. 19: 13-16.

SEIDELL J C; OOSTERLEE A; THIYSSEN M; BUREMA J; DEURENBERG P; HAUTVAST J; RUIJS J (1987).
Assessment of intra-abdominal and subcutaneous abdominal fat: Relation between anthropometry and computed tomography. **American Journal of Clinical Nutrition**. 45: 7-13.

SHARKEY B J (1984).
Physiology of Fitness. Champaign IL. Human Kinetics.

SHEKELLE R B; SKRYOCK A M; PAUL O (1981).
Diet, serum cholesterol, and death from coronary heart disease. The Western Electric study. **New England Journal of Medicine**. 304: 65-70.

SHILS M E; OLSON J A; SHIKE M (1994).
Modern Nutrition in Health and Disease. Philadelphia. Lea & Febiger.

SHORE B (1996).
Culture in Mind. New York. Oxford University Press.

SIMS E A H (1976).
Experimental obesity, dietary induced thermogenesis and their clinical implications. **Clinical Endocrinology**. 5: 377-395.

SIMS E A H; DANFORTH E (1987).
Expenditure and storage of energy in man (perspective). **Journal of Clinical Investigation**; 79: 1019.

SINHA M K; OHANNESIAN J P; HEIMAN M L (1996).
Nocturnal rise in leptin in lean, obese and NIDDM subjects. **Journal of Clinical Investigation**. 97: 1344-1347.

SJÖSTRÖM L (1991).
A computer-tomography based multi-compartment body composition technique and anthropometric predictions of lean body mass, total and subcutaneous adipose tissue. **International Journal of Obesity**. 15: 19-30.

SJÖSTRÖM L; BJÖRNTORP P A (1974).
Body composition and adipose tissue cellularity in human obesity. **Acta Medica Scandinavica**; 195: 201-211.

SKAGGS S R; CRIST D M (1991).
Exogenous human growth hormone reduces body fat in obese women. **Hormone Research**.
35: 19-24.

SLIMLINE (2001).
Slimline Promational Literature. http://www.slimline.co.za

SMIT C (2002).
Afristat Researchers. Personal communication

SMITH D E; LEWIS C E; CAVENY J L; PERKINS L L; BURKE G L; BILD D E (1994).
Longitudinal changes in adiposity associated with pregnancy. The CARDIA Study. Coronary Artery Risk Development in Young Adults. **Journal of American Medical Association**. 271: 1747-1751.

SOBAL J (1991).
Obesity and socioeconomic status: a framework for examining relationships between physical and social variables. **Medical Anthropology**. 13: 231-248.

SOBAL J; STUNKARD A J (1989).
Socioeconomic status and obesity: A review of the literature. **Psychological Bulletin**. 105: 260-275.

SOONG Y S (1975).
The treatment of exogenous obesity employing auricular acupuncture. **American Journal of Chinese Medicine**. 3: 285-287.

SOURS H E; FRATTALI U P; BRAND C D (1981).
Sudden death associated with very low caloric weight reduction regimens. **American Journal of Clinical Nutrition**. 34: 453-461.

SOUTH AFRICAN SOCIETY FOR THE STUDY OF OBESITY (1999).
Obesity and health update. **Official Newsletter**. Vol. 3.

SPIEGELMAN B M; FLIER J S (1996).
Adipogenesis and obesity: Rounding out the big picture. **American Journal of Medicine.** 87: 377-389.

SPITZER R L; DEVLIN M; WALSH B T (1992).
Binge eating disorder: a multi site field trial of the diagnostic criteria. **International Journal of Eating Disorders**. 11: 191-203.

SPITZER R L; YANOVSKI S; WADDEN T (1993).
Binge eating disorder: its further validation in a multi site study. **International Journal of Eating Disorders**. 13: 137-153

STAFFIERI J R (1967).
A Study of social stereotype of body image in children. **Journal of Social Psychology**. 7: 101-104.

STAMFORD B (1991).
Apples and pears: Where you "wear" your fat can effect your health. **The Physician and Sportsmedicine**; 19(1). 123-124.

STAMLER R; STAMLER J; RIEDLINGER W E; ALGERA G; ROBERTS R H (1978).
Weight and blood pressure. Findings in hypertension screening of 1 million Americans. **Journal of American Medical Association**. 240: 1607-1610.

STAMPFER M J; MACLURE R M; COLDITZ G A; MANSON J E; WILLETT W C (1992).
Risk of symptomatic gallstones in women with severe obesity. **American Journal of Clinical Nutrition**. 55: 652-658.

STANKO R T; ADIBI S A (1986).
Inhibition of lipid accumulation and enhancement of energy expenditure by the addition of pyruvate and dihydroxyacetone to a rat diet. **Metabolism**. 35: 182-186.

STANKO R T; ARCH J E (1996).
Inhibition of regain in body weight and fat with addition of 3-carbon compounds to the diet with hyper-energetic re-feeding after weight reduction. **International Journal of Obesity and Related Metabolic Disorders**. 20: 925-930.

STANKO R T; TEITZE D L; ARCH J E (1992)
Body composition, energy utilization and nitrogen metabolism with a severely restricted diet supplemented with dihydroxyacetone and pyruvate. **American Journal of Clinical Nutrition**. 55: 771-775.

STATISTICAL PACKAGE FOR SOCIAL SCIENCE (SPSS) (1999).
Seattle. USA. Microsoft.

STEEL J M; BRIGGS M (1972).
Withdrawal depression in obese patients after fenfluramine treatment. **British Medical Journal**. 3: 67-69.

STEVENS J; CAI J; PAMUK E R; WILLIAMSON D F; THUN M J; WOOD J L (1998).
The effect of age on the association between body-mass index and mortality. **New England Journal of Medicine**. 338: 1-7.

STEVENS V J; ROSSNER J; HYG M S (1989).
Freedom from fat: a contemporary multi-component weight loss program for the general population of obese adults. **Journal of American Dietetic Association**. 89: 1254-1258.

STILLMAN J M; BAKER S S (1978).
The Doctor's Quick Weight Loss Diet. New York. Dell Publishing Co.

STRIEGEL-MOORE R H; WILSON G T; WILFLEY D E; ELDER K A; BROWNELL K D (1998).
Binge eating in an obese community sample. **International Journal of Eating Disorders**. 13: 137-153.

STUART RB (1967).
Behavioural control of overeating. B**ehaviour Research and Therapy**. 5: 357-365.

STUBBS R J; RITZ W A; COWARD W A; PRENTICE A M (1995).
Covert manipulation of the ratio of dietary fat to carbohydrate and energy density: effect on food intake and energy balance in free-living men eating ad libitum. **American Journal of Clinical Nutrition.** 62: 330-337.

STUNKARD A J (1995).
Prevention of obesity. In. **Eating Disorders and Obesity: A Comprehensive Handbook**. Brownell KD, Fairburn CG (eds.). New York. Guilford Press.

STUNKARD A J (1996).
Current views on obesity. **American Journal of Medicine**. 100: 230-236.

STUNKARD A J; RUSH J (1974).
Dieting and depression reexamined: a critical review of reports of untoward responses during weight reduction for obesity. **Annals of Internal Medicine**. 81: 526-533.

STUNKARD A J; WADDEN T A (1993).
Obesity. Theory and Therapy. (2nd ed.). New York. Raven Press.

SUGERMAN H J; LANDREY G L; KELLUM J M; WOLF L; LISZKA T; ENGLE K M; BIRKENIIAUER R; STARKEY J V (1989).
Weight loss with vertical banded gastroplasty and roux-y gastric bypass for morbid obesity with selective versus random assignment. **American Journal** of Surgery. 157: 93-102.

SUN Q; XU Y (1993).
Simple obesity hyperlipemia treated with otoacupoint pellet pressure and body acupuncture. **Journal of Traditional Chinese Medicine**. 13: 22-26.

SWAMINATHAN R K; KING R F; HALMFIELD J; SIWEK R A; BAKER M; WALES J K (1985). Thermic effect of feeding carbohydrate, fat, protein, and mixed meat in lean and obese subjects. **American Journal of Clinical Nutrition**; 42: 177-181.

SWEENEY M E; HILL J O; HILLER P A; BANEY K; DIGIROLAMO M (1993).
Severe vs: moderate energy restriction with and without exercise in the treatment of obesity: Efficiency of weight loss. **American Journal of Clinical Nutrition**; 57: 127-134.

TAINTER M L; STOCKTON A B; CUTTING W C (1933).
Use of dinitrophenol in obesity and related conditions. **Journal of American Medical Association.** 101:1472-1475.

TARAS H L; SALLIS J F (1989).
Television's influence on children's diet and physical activity. **Journal of Developmental and Behavioral Pediatrics**. 10(4): 176-180.

TECHNOLOGY ASSESSMENT CONFERENCE PANEL (1992).
Methods for voluntary weight loss and control: Technology Assessment Conference Statement. **Annals of Internal Medicine; 116: 942-949.**

TECHNOLOGY ASSESSMENT CONFERENCE PANEL (1993).
Methods for voluntary weight loss and control: Technology Assessment Conference Statement. **Annals of Internal Medicine**; 115: 956-961.

TELCH C F; AGRAS W S (1993).
The effects of a very low-calorie diet on binge eating. **Behavioral Therapy**. 24: 177-194.

TETI V (1995).
Food and Fatness in Calabria. In. **Social Aspects of Obesity**. Garine ID, Pollack NJ. (eds.). Amsterdam. Gordon and Breach Publishers.

THERMO LEAN (2001).
Thermo Lean Promotional Literature. E-mail: nickmentz(@sport.up.ac.za

THOMAS P R (1995).
Weighing the Options. Criteria for Evaluating Weight-Management Programs. Washington, D.C. National Academy Press.

TIETZ N W (1994).
Specimen Collection and Processing, Sources of Biological Variation. Textbook of Clinical Chemistry. (2nd ed.). Philadelphia. W.B. Saunders.

TITCHENAL C A (1988).
Exercise and food intake: What is the relationship? **Sports Medicine**; 6: 135-145.

TIGGEMANN M; ROTHBLUM E D (1988).
Gender differences in social consequences of perceived overweight in the United States and Australia. **Sex Roles**. 18: 75-86.

TOMASZYK A; SIMPSON C; WILLIAMS G (1996).
Neuropeptide Y, the hypothalmus and the regulation of energy homeostasis. **Hormone Research.** 46: 53-58.

TROIANO R P; FRONGILLO E A JR; SOBAL J; LEVITSKY D A (1996).
The relationship between body weight and mortality: a quantitative analysis of combined information from existing studies. **International Journal of Obese Related Metabolic Disorders**. 20: 63-75.

TROWELL H C; BURKITT D P (1981).
Western Diseases: Their Emergence and Prevention. Cambridge, M A. Harvard University Press.

TSANG G M; GREEN M A; CROW A J (1994).
Chronic muscle stimulation improves ischaemic muscle performance in patients with peripheral vascular disease. **European Journal of Vascular Surgery**. 8: 419-422.

TUCK M I; SOWERS J; DORNFIELD L (1981).
The effect of weight reduction on blood pressure plasma renin activity and plasma aldosterone level in obese patients. **New England Journal of Medicine**. 304: 930-933.

TYLER V E (1993).
The Honest Herbal: A Sensible Guide to the Use of Herbs and Related Remedies.
New York. Pharmaceutical Products Press.

URBINA E M; GIDDING S S; BAO W; PICKOFF A S; BERDUSIS K; BERENSON G S (1995).
Effect of body size, ponderosity, and blood pressure on left ventricular growth in children and young adults in the Bogalusa Heart Study. **Circulation**. 91: 2400-2406.

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES (1996).
Physical Activity and Health: A Report of the Surgeon General. Atlanta Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion; 9-14.

VAGUE J (1947).
La differenciation sexuelle facteur determinant des formes de l'obésité. **Presse Medical**. 55: 339-340.

VANCE M L (1990).
Growth hormone for the elderly? New **England Journal of Medicine.** 323: 52-54.

VAN DALE O; SARIS W HM (1989).
Repetitive weight loss and weight regain: Effect on weight reduction, resting metabolic rate and lipolytic activity before and after exercise and/or diet treatment. **American Journal of Clinical Nutrition**; 49: 409-416.

VANDER A J; SHERMAN J H; LUCIANO D S (1994).
Human Physiology: The Mechanisms of Body Function (6th ed.). New York: McGraw-Hill, Inc.

VAN DER KOOY K; SEIDELL J C (1993).
Techniques for the measurement of visceral fat: a practical guide. **International Journal of Obesity**. 17: 187-196.

VAN DER MERWE T (2002)
SA Woman can match US fatties.**dispatch.co.za/2002/08/14**

VAN DER MERWE T (2003)
SA Woman just as fat as Americans. **health.iafrica.com/healthnews**

VAN HEERDEN H J (1996).
Preparticipation Evaluation and Identification of Aetiological risk facts in the Epidemiology of Sport Injuries among Youths. Unpublished D.Phil thesis. Pretoria: University of Pretoria.

VAN HEERDEN I (2002).
Fat, fatter, South African? Obesity tops health risk list. **Health 24.co.za**.

VAN ITALLIE T B (1980).
Dietary approaches to the treatment of obesity. In. **Obesity**. Stunkard AJ (ed.). Philadelphia. W B Saunders.

VAN ITALLIE T B (1985).
Health implications of overweight and obesity in the United States. **Annals of Internal Medicine.** 103: 983-988.

VAN ITALLIE T B; ABRAHAM S (1985).
Some hazards of obesity and its treatment. In. **Recent Advances in Obesity Research.** Hirsch J., Van Itallie T.B.(eds.). London. John Libbey.

VAN ITALLIE T B; LEW E A (1990).
Overweight and underweight. In. **Medical Risks 1987: Mortality Trends by Age and Time Elapsed.** Lew EA, Gajewski J (eds.). New York. Praeger.

VENES A M; KRUPKA L R; GERARD R J (1982).
Overweight/obese patients: an overview. **Practitioner**. 226: 1102-1109.

VOGIATZI M G; BOECH M A; VLACHO-PAPADOPOULOU E; EL-RASHID R; NEW M I (1996).
Dehydroepiandrosterone in morbidly obese adolescents. Effect on weight, body composition, lipids, and insulin resistance. **Metabolism: Clinical & Experimental**. 45: 1011-1015.

WADDEN T A (1993).
Treatment of obesity by moderate and severe caloric restriction: results of clinical research trials. **Annals of Internal Medicine. 119: 688-693.**

WADDEN T A (1993).
The treatment of obesity: an overview. In. **Obesity: Theory and Therapy.** (2nd ed.). Stunkard AJ, Wadden TA (eds.). New York. Raven Press.

WADDEN T A (1995).
Obesity Treatment: Establishing Goals, Improving Outcomes, and Reviewing the Research Agenda. New York. Plenum Publishing Corp.

WADDEN T A; BARTLETT S; LETIZIA K A (1992).
Relationship of dieting history to resting metabolic rate, body composition, eating behavior and subsequent weight loss. **American Journal of Clinical Nutrition**; 56: 206-211.

WADDEN T A; FLAXMAN J (1981).
The clinical use of hypnosis. **Psychology Bulletin**. 91: 215-243.

WADDEN T A; STUNKARD A J (1993).
The psychological and social complications of obesity. **Annals of Internal Medicine**. 103: 1062-1067.

WADDEN T A; STUNKARD A J (1993).
Psychosocial consequences of obesity and dieting research and clinical findings. In. **Obesity Theory and Therapy.** Stunkard AJ, Wadden TA (eds.). New York. Raven Press.

WADDEN T A; VAN ITALIE T B; BLACKBURN G L (1990).
Responsible and irresponsible use of very low-caloric diets in the treatment of obesity. **Journal of American Medical Association.** 263: 83-85.

WADDEN T A; VOGT R A; ANDERSEN R E (1997).
Exercise in the treatment of obesity: effects of four interventions on body composition, resting energy expenditure, appetite and mood. **Journal of Clinical Psychology**. 65: 269-277.

WADDEN T A (1995).
Compassionate treatment of the obese individual. In. **Eating Disorders and Obesity: A Comprehensive Handbook.** Brownell KD, Fairburn C (eds.). New York. Guilford Press.

WALKER S P; RIMM E B; ASCHERIO A; KAWACHI I; STAMPFER M J; WILLET W C (1997).
Body size and fat distribution as predictors of stroke among US men. **American Journal of Epidemiology**. 144: 1143-1150.

WALTER S D; HART L E (1990).
Application of epidemiological methodology to sports and exercise science: **Exercise and Sport Sciences Reviews. 18: 417-448.**

WANG Q; BING C; AL-BARAZANJI K; MOSSAKOWASKA D E; WANG X; MC BAY D (1997).
Interaction between leptin and hypothalamic neuropeptide Y neurons in the control of food intake and energy homeostasis in the rat. **Diabetes**. 46: 335-341
.
WANSINK B (1996).
Can package size accelerate usage volume? **Journal of Marketing**. 60: 1-14.

WEBSTER (1988).
Webster's New World Dictionary of the American Language, Third College Edition. New York. Prentice Hall.

WEI M; KAMPERT J B; BARLOW C E (1999).
Relationship between low cardiorespiratory fitness and mortality in normal weight, overweight, and obese men. **Journal of American Medical Association.** 282: 1547-1553.

WEIGLE D S; SANDE K J; IVERIUS P H; MONSEN E R; BRUNZELL J D (1988).
Weight loss leads to a marked decrease in nonresting energy expenditure in ambulatory human subjects. **Metabolism**. 37: 930-936.

WEINTRAUB M (1992).
Long-term weight control: The National Heart, Lung and Blood Institute funded multi-modal intervention study. **Clinical Pharmacological Therapy**. 51: 581-585.

WEINTRAUB M; HASDAY J D; MUSHLIN A I; LOCKWOOD D H (1984).
A double-blind clinical trial in weight control. Use of fenfluramine and phenteramine alone and in combination. **Archive of Internal Medicine. 144: 1143-1148.**

WEINTRAUB M; RUBIO A; GOLIK A; BRYNE L; SCHEINBAUM M L (1991).
Sibutramine in weight control: A dose-ranging, efficacy study. **Clinical Pharmacological Therapy**. 50: 330-337.

WEINTRAUB M; SUNDARESAN P R; SCHUSTER B (1992).
Long-term weight control study II (weeks 34-104): An open-label study of continuous fenfluramine plus phenteramine versus targeted intermittent medication as adjuncts to behaviour modification, caloric restriction, and exercise. **Clinical Pharmacological Therapy**. 51: 586-594.

WELLE S (1984).
Metabolic response to a meal during rest and low intensity exercise. **American Journal of Clinical Nutrition**; 40: 990-994.

WELLE S L (1986).
Some metabolic effects of overeating in man. **American Journal of Clinical Nutrition.** 44: 718.

WELTMAN A; LEVINE S; SEIP R L; TRAN Z V (1988).
Accurate assessment of body composition in obese females. **American Journal of Clinical Nutrition**. 48: 1179-1183.

WERNER E E; SMITH R S (1992).
Overcoming the Odds: High Children from Birth to Adulthood. Ithacu, NY. Cornell University Press.

WEST D; BOOZER C N; MOODY D L; ATKINSON R (1992).
Dietary obesity in nine inbred mouse strains. **American Journal of Physiology**. 262: 1025-1032.

WEST K (1978).
Diabetes in American Indians. **Advances in Metabolic Disorders**. 9: 29-48.

WHITE F; PERCERA L; GARNER J B (1986).
Associations of body mass index and waist-hip ratio with hypertension. **Canadian Medical Association Journal**. 135: 313-320.

WIGERSTAD-LOSSING I; GRIMBY G; JONSSON T; MORELLI B; PETERSON L; RENSTRÖM P (1988).
Effects of electrical muscle stimulation combined with voluntary contractions after knee ligament surgery. **Medicine and Science in Sport and Exercise**. 20: 93-80.

WILDING J (1997).
Obesity treatment. **British Medical Journal**. 315: 997-1000.

WILLETT W C (1998).
Is dietary fat a major determinant of body fat. **American Journal of Clinical Nutrition**. 67: 556-562.

WILLETT W C; MANSON J E; STAMPFER M J (1995).
Weight, weight change, and coronary heart disease in women. Risk within the "normal" weight range. **Journal of American Medical Association**. 273: 461-465.

WILLETT W C; STAMPFER M; MANSON J; VAN ITALLIE T B (1991).
New Weight guidelines for Americans: justified or injudicious? **American Journal of Clinical Nutrition**. 53: 1102-1103.

WILLIAMS M H (1995).
Nutrition for Fitness & Sport. (4th ed.). Dubuque. USA. Brown and Benihmark.

WILLIAMS R A; FOULSHAM B M (1981).
Weight reduction in osteoarthritis using phentermine. **Practitioner**. 225: 231-232.

WILLIAMSON D F (1996).
"Weight cycling" and mortality: how do the epidemiologists explain the role of intentional weight loss? **Journal of the American College of Nutrition**. 15: 6-13.

WILLIAMSON D F (1997).
Intentional weight loss: patterns in the general population and its association with morbidity and mortality. **International Journal of Obese Related Metabolic Disorders**. 21: 14-19.

WILLIAMSON D F; MADANS J; PAMUK E; FLEGEL K M; KENDRICK J S; SERDULA M K(1994).
A prospective study of childbearing and 10-year weight gain in U.S. white women 25 to 45 years of age. **International Journal of Obese Related Metabolic Disorders**. 18: 561-569.

WILLIAMSON D F; PAMUK E R (1993).
The association between weight loss and increased longevity. A review of the evidence. **Annals of Internal Medicine**. 119: 731-736.

WILMORE J H (1983).
Appetite and body composition consequent to physical activity. **Research Quarterly for Exercise and Sport**; 54(4): 415-425.

WILSON N; QUIGLEY R (1999).
Food ads on TV: a health hazard for children? **Australian Journal of Public Health**. 23: 647-650.

WING R R (1989).
Behavioural strategies for weight reduction in obese type II diabetics. **Diabetes Care**. 12:139-144.

WING R R; JEFFERY R W; BURTON L R; THORSON C; NISSINOFF K S; BAXTER J E (1996).
Food provision vs. structured meal plans in the behavioural treatment of obesity. **International Journal of Obesity.** 20: 56-62.

WINKELSTEIN L B (1959).
Hypnosis, diet and weight reduction. **New York State Journal of Medicine.** 59: 661-665.

WOOD P D; STEFANICK M L; WILLIAMS P T; HASKELL W L (1991).
The effects on plasma lipoproteins of a prudent weight-reducing diet, with or without exercise, in overweight men and women. **New England Journal of Medicine.** 325: 461-466.

WORLD HEALTH ORGANIZATION (WHO) (1997).
Preventing and Managing the Global Epidemic. Report of a WHO Consultation of Obesity. Geneva, 3-5 June.

YANOVSKI J A; YANOVSKI S Z (2000).
A prospective study of holiday weight. **New England Journal of Medicine**. 342: 861-867

YANOVSKI S Z (1993).
Binge eating disorder: Current knowledge and future directions. **Obesity Research**. 1: 306-324.

YANOVSKI S Z; GORMALLY J F; LESER M B; GWIRTSMAN H E; YANOVSKI J A (1994).
Binge eating disorder affects outcome of comprehensive very-low-calorie diet treatment. **Obesity Research**. 2: 205-212.

YANOVSKI S Z; SEBING N G (1994).
Recorded food intake of obese women with binge eating disorder before and after weight loss. **International Journal of Eating Disorders**. 15: 135-150.

YOUNG R L; FUCHS R J; WOLTJEN M J (1976).
Chorionic gonadotropin in weight control. A double blind crossover study. **American Medical Association.** 236: 2495-2497.

YOUNG T; PALTA M; DEMPSEY J; SKATRUD J; WEBER S; BADR S (1993).
The occurrence of sleepdisordered breathing among middle-aged adults. **New England Journal of Medicine**. 328: 1230-1235.

ZAHORSKA-MARKIEWICZ B (1980).
Thermic effect of food and exercise in obesity. **European Journal of Applied Physiology**; 44: 231-235.

ZAMBONI M; MARKIEWICZ B (1998).
Thermic effect of food and exercise in obesity. **European Journal of Applied Physiology**; 44: 231-235.

ZAMBONI M; TURCATO E; ARMELLINI F; KAHN H S; ZIVELONGHI A; SANTANA H; BERGAMO-ANDREIS I A; BOSELLO O (1998).
Sagittal abdominal diameter as a practical predictor of visceral fat. **International Journal of Obesity**. 22: 655- 660.

ZED C A; HARRIS G A; HARRISON P J (1985).
Anti-obesity activity of a novel beta-adrenoceptor agonist (BRI 26830A) in diet-restricted obese subjects. **International Journal of Obesity.** 9:231.

ZEMAN F (1991).
Clinical Nutrition and Dietetics. New York. Macmillan Publishing Co.

ZHANG Y; PROENCA R; MUFFEI M; BARONE M; LEOPOLD L; FREIDMAN J M (1994).
Positional cloning of the mouse obese gene and its human hormologue. **Nature**. 372: 425-432.

ZHANG Z (1990).
Weight reduction by auriculo-acupuncture: a report of 110 cases. **Journal of Traditional Chinese Medicine.** 10: 17-18.

ZIMMET P (1979).
Epidemiology of diabetes and its macro-vascular manifestations in pacific populations. The medical effects of social progress. **Diabetes Care**. 2: 144-153.

ZURLO F; LILLIOJA S; ESPOSITO-DEL PUENTE A (1990).
Low ratio of fat to carbohydrate oxidation a predictor of weight gain: study of 24-h RQ. **American Journal of Physiology**. 259: 650-657.

APPENDIX A

INFORMED CONSENT

I _____
(full name of prospective participant)

have been informed of the procedures and requirements to participate in a research project dealing with the effect of Electrical and Thermogenic Stimulation on Weight Reduction and various physiological parameters, to be conducted at the Institute of Sport Research of the University of Pretoria.

I am amenable to being assigned to any of the experimental groups and thus willingly participate in the said research project at my own risk.

I declare hereby that no information has been withheld that could exclude me from participating in an exercise programme, and am aware that I am entitled to withdraw from the study at any time if I should wish.

I hereby also grant the researcher permission to use my results for publication and/or presentation purposes, with my anonymity being ensured.

_____ _____
Signature of prospective participant. Date

Tel: _____(h) _____(w)

Witness

1. _____

2. _____

APPENDIX B

Efficacy of Electrical and Thermogenic Stimulation on Weight Reduction among Obese Females

1. RESULT SHEET

NAME:_____
(surname and initials)

SEX: E DATE:_____

STATURE:_____cm AGE:_____yrs MASS:_____kg

SKINFOLDS (mm)				
Triceps				
Subscapular				
Suprailiac				
Biceps				
Calf				
Abdominal				
Mid-thigh				

DIAMETER (cm)				
Biacromial				
Trans chest				
Ant-Post chest				
Bi-iliac				
Humerus				
Femur				
Sagital ½ umbi				
Sagital umbi				

GIRTHS (cm)				
Relaxed arm				
Contracted arm				
Forearm				
Wrist				
Chest				
Mid-thigh				
Calf				
Ankle				
Hip				
Abdominal				
AB-1 ½ umbi				
AB-2 umbi				

Fat %	%	kg
Residual %		kg
Bone %		kg
Somatotype	I	
X =	II	
Y =	III	
Muscle mass	%	Kg
Middle/Hip Ratio		
Body Surface Area		m^2
LBM		%
Body Mass Index		Kg/m2
Fat Mass (W)		Kg

BLOOD PRESSURE _____ PULSE _____

GAS ANALYSIS (RQ) _____

FLEXIBILITY (Hip Flexion) _____ (1)

_____(2) Difference _____

SIT UPS (1 minute) _____

LUNG FUNCTION

FVC _____ l

FEV1 _____ l

FEV1 % _____ %

PEF _____ l/s

MEF 50% _____l/s

MEF 25% _____ l/s

HAEMATOLOGICAL RESPONSES

Cholesterol _____ m.mol/L

HDL Cholesterol _____ m.mol/L

LDL Cholesterol _____ m.mol/L

Triglycerides _____ m.mol/L

Glucose _____ m.mol/L

ULTRASOUND MEASUREMENTS (Sonar)

O1 _____mm

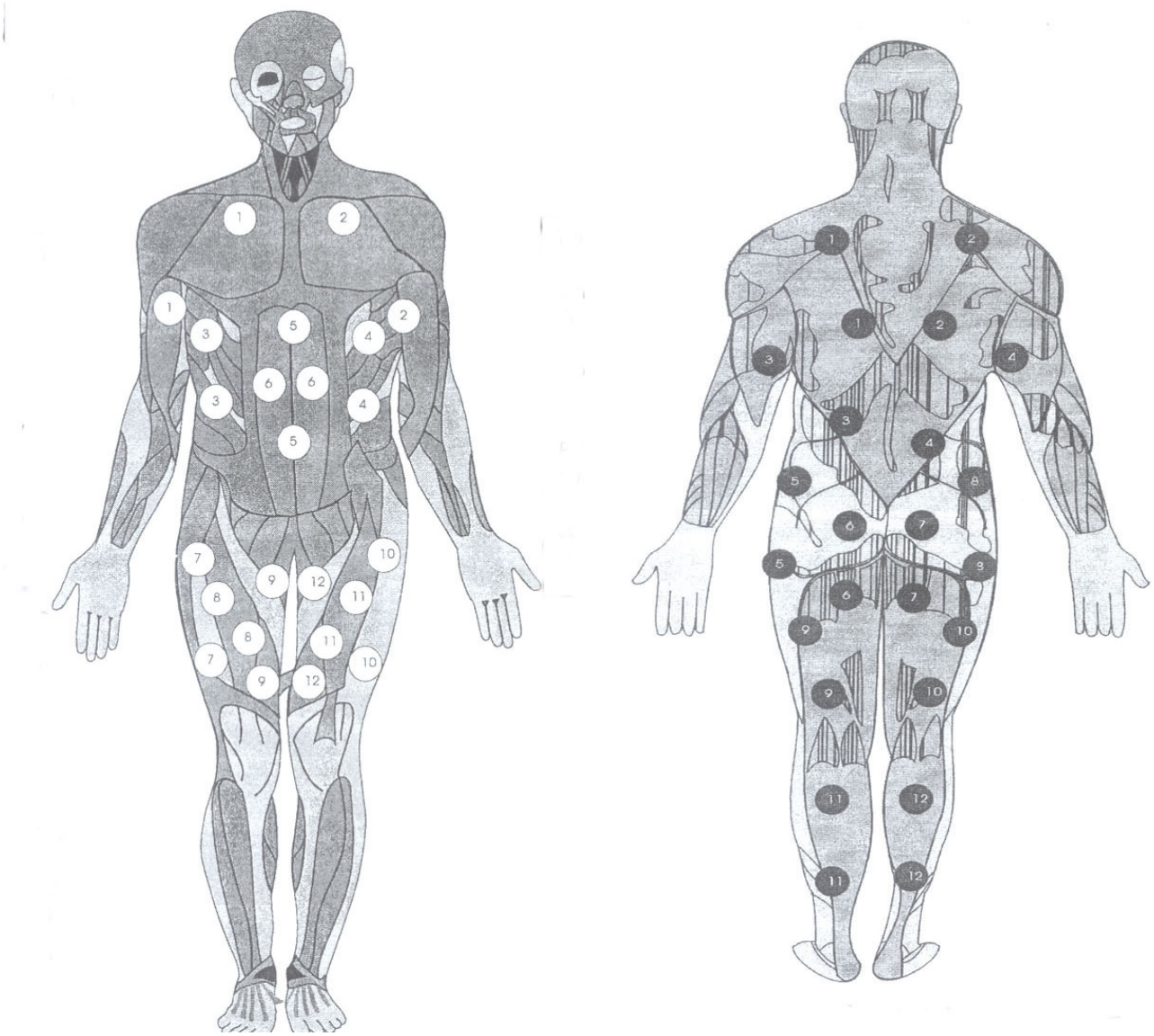
O2 _____mm

APPENDIX C

(nothing available)

APPENDIX D

EMS PAD PLACEMENT CHART



APPENDIX E

METABOLISM DIET

In general you consume normal everyday food, eating different amounts of food (calories) in three phases.

- Low calorie phase

  At 1000 calories per day, this phase is designed for maximum weight loss, while you consume a nutritionally balanced diet. This menu must be followed for two weeks.

- Booster phase

  After two weeks on the low calorie phase you must switch to the booster menu plan with 300 more calories. This phase is designed to boost the metabolic rate. The added calories during the booster phase are made up by carbohydrates.

- Re-entry phase

  When you get within two to three kg from your target goal-weight, you must switch to the re-entry phase. Unless you gradually increase your calories you have the risk of gaining weight. This pre-maintenance period serve to get your metabolism ready for normal eating.

LOW CALORIE PHASE

Breakfast every day

Cereal (¾ cup)
Choose that are low in sugar content, such as:
- Special K
- 40% Bran
- Shredded wheat
- Wheaties
- Raisin Bran
- Oatmeal
- Puffed Wheat

Milk (½ cup)
Low-fat (2%) or skim milk

Fruit (½ piece)
Choice of
- Orange
- Pear
- Apple
- Grapefruit
- Peach

Coffee or Tea
Sugar substitute and/or skim milk may be added if desired.

Lunch: 1

Fruit plate
- Strawberries (½ cup)
- Honeydew melon (¼ cup) or substitute fruit in season
- Sweet melon (¼ cup)
- Cottage Cheese (¼ cup low-fat)
- Lettuce (a few leaves under the fruit)

Lunch: 2
- Egg (1 whole egg)
- Whole wheat bread (1 slice toasted)
- Grapefruit (½)

Lunch: 3
- Tuna fish (90g water packed)
- Lettuce (¼ small head)
- Tomato (½ medium)
- Cucumber slices (10 slices)
- Diet salad dressing (2 table spoons)

Lunch: 4
- Tossed salad (large salad bowl with lettuce, tomato, cucumber, radish mix)
- 2 tablespoons diet dressing
- Roll (1 small hard or Kaiser roll)
- 1 tablespoon diet margarine

Lunch: 5
Whole tomato stuffed with chicken salad ($^{2}/_{3}$ cup)

Lunch: 6
Fruit salad
- Cottage cheese (¼ cup of low fat 50g)
- Lettuce (¼ head)
- Orange (½ 100g)
- Apple (½ 100g)
- Grapes (¾ cup)

Lunch: 7
- Omelette: egg whites (3 eggs)
- Cottage cheese (¼ cup low fat 50g)
- Onion ($^{1}/_{8}$ small 20g)
- Green pepper (¼ cup chopped 50g)
- Mushroom (¼ cup chopped 50g)

NOTE: Fry egg whites lightly in frying pan, using vegetable cooking spray. As egg whites set, add cottage cheese, onion, green pepper and fold over omelette to cover filling.

Dinner: 1
- Baked chicken (150g or 2 breasts
- Baked potato (1 medium, no butter
- Vegetable: Choice of green beans, broccoli, asparagus (½ cup), strawberries (½ cup 80g), vanilla yoghurt (1 tablespoon over fruit)

Dinner: 2
- Baked or broiled fish (190g of any type, no butter
- Rice (½ cup)
- Vegetable: Choice of two – broccoli, carrots, green beans, cauliflower, asparagus, spinach (½ cup each)
- Orange slices (½ cup with dash of vanilla yoghurt if desired 100g)

Dinner: 3
- Broiled lean hamburger (180g)
- Egg noodles (½ cup, cooked)
- Vegetable: Choice of green beans , broccoli, asparagus (½ cup)

Dinner: 4
- Baked chicken (150g or 2 breasts
- New potatoes (½ cup)
- Vegetable: Choice of spinach, green beans, broccoli (½ cup)
- Fruit: Choice of pineapple (slice) or strawberries (½ cup)

Dinner: 5
- Broiled fish or shrimp (180g)
- Corn on the cob (1 medium)
- Small tossed salad (with 2 tablespoons diet dressing)
- Peach (1 whole) or substitute fruit in season

Dinner: 6
- Broiled steak (150g visible fat removed)
- Baked potato (medium with 2 tablespoons of diet margarine
- Vegetables: choice of broccoli, asparagus (½ cup)

Dinner: 7
- Spaghetti (½ cup)
- Meatless sauce (commercially prepared sauce
- Parmesan cheese (sprinkled lightly over spaghetti 5-10g)
- Tossed salads (small bowl of lettuce, tomato, cucumber, radish mix with 2 tablespoons diet dressing)
- Roll or bread (2 medium slices of Italian or French bread, with not butter or garlic

Late-snack: 1
Cinnamon toast (2 slices of in-sliced wheat bread sprinkled lightly with cinnamon and artificial sweetner and toast under the broiler)

Late-snack: 2
- Small tossed salad (2 tablespoons diet dressing
- Apple (½)

Late-snack: 3
- Cereal (½ cup; choice of shredded whet or 40% bran)
- Milk (½ cup low-fat or skim)
- Banana (½ 50g)

Late-snack: 4
- Cottage cheese (¼ cup low-fat 150g)
- Apple (½)

Late-snack: 5
Raw vegetable plate (mixture of 6 each of raw carrot sticks, celery, radishes and cauliflower with diet dip – if desired)

Late-snack: 6
Sliced banana (1 whole, lightly sprinkled with cinnamon artificial sweetner mixture and toasted under broiler

Late-snack: 7
Fruit plate
- Apple (½ 100g)
- Banana (½ 50g)
- Raisins (1 tablespoon 10g)

NOTE: Slice and mix fruit together.

BOOSTER PHASE

Breakfast every day

Cereal (¾ cup)
Choose that are low in sugar content, such as:
- Special K
- 40% Bran
- Shredded wheat
- Wheaties
- Raisin Bran
- Oatmeal
- Puffed Wheat

Milk (½ cup)
Low-fat (2%) or skim milk

Fruit (½ piece)
Choice of
- Orange
- Pear
- Apple
- Grapefruit
- Peach

Coffee or Tea
Sugar substitute and/or skim milk may be added if desired.

Lunch: 1
- Tuna fish sandwich: Whole wheat bread (2 slices) tuna (80g) water-packed canned tuna, mixed with 1 teaspoon mayonnaise, lettuce
- Peach (½ small 100g)

Lunch: 2
- Chicken salad (90g chicken with teaspoon mayonnaise and 1 teaspoon chopped celery)
- Lettuce (few leaves under salad)
- Tomato (½ sliced)

Lunch: 3
- Cottage cheese ($^1/_3$ cup low-fat 45g)
- Sweet melon (¼ cut in wedges)
- Strawberries (½ cup sliced)
- Lettuce (few leaves under fruit)

Lunch: 4
- Eggs (2 any style, if fried use vegetable cooking spray)
- Whole wheat bread, toasted (1 slice)
- Grapefruit (½)

Lunch: 5
- Tossed salad (large salad bowl with lettuce, tomato, cucumber, radish, green pepper and carrot, mixed together with 3 tablespoons diet dressing)
- Fruit: Choice of apple of pear (120g)

Lunch: 6
Open-faced grilled cheese and tomato sandwich (2 slices of white or whole wheat bread, each topped with slices of tomato and low-fat (diet) cheese and grilled lightly under broiler)

Lunch: 7
- Egg salad sandwich: Rye or whole wheat bread (2 slices)
- Egg salad: (1 whole egg, chopped with 1 tablespoon mayonnaise)
- Strawberries (½ cup substitute fruit in season)

Dinner: 1
- Cornish hen (app 150g of meat)
- New potatoes (½ cup 100g)
- Vegetables: Choice of carrots, green beans, broccoli, asparagus (½ cup 125ml)
- Strawberries (½ cup, topped with a tablespoon vanilla yoghurt) or fruit in season

Dinner: 2
- Baked or broiled fish (180g no butter)
- Baked potato (1 medium, no butter)
- Vegetable: Choice of two – broccoli, carrots, green beans, cauliflower, asparagus, spinach (½ cup each 250 ml)
- Apple-raisin mix (½ apple, diced mixed with 1 tablespoon raisins 10g)

Dinner: 3
- Roast lamb or veal (150g fat removed)
- Baked potato (1 medium 100g with 1 teaspoon diet margarine)
- Vegetable: Choice of green beans, broccoli, asparagus (½ cup)

Dinner: 4
- Spaghetti (1 cup cooked noodles)
- 250 ml meatless sauce (5g commercially prepared sauce
- Parmesan cheese (10 ml) sprinkled lightly over spaghetti
- Bread (2 medium slices of Italian or French bread, with 1 tablespoon diet margarine)

Dinner: 5
- Broiled fish or shrimp – 200g
- Rice (1 cup – 250 ml)
- Vegetables: Choice of two – broccoli, carrots, green beans, cauliflower, asparagus, spinach (½ cup each)
- Orange slices (½ cup with one tablespoon vanilla or lemon yoghurt)

Dinner: 6
- Roast beef (150g)
- Baked potato (1 medium, no butter
- Vegetables: Choice of broccoli, baby marrow or asparagus (½ cup 125 ml)
- Tossed salad (small bowl of lettuce, onion, cucumber and radishes mixed with 2 table-spoons diet dressing)

Dinner: 7
- Turkey or chicken (150g)
- Rice (½ cup 125 ml)
- Vegetable: Choice of two: spinach, green beans, broccoli, carrots, as-paragus, cauliflower (½ cup each)
- Fruit: Apple, orange, peach (½ - 100g)

Late-snack: 1
English muffin (1 whole with 1 tablespoon diet margarine)

Late-snack: 2
Popcorn (4 cups of popped popcorn with 1 tablespoon diet margarine, no salt

Late-snack: 3
- Raw vegetable plate (mix raw carrot sticks, celery, cauliflower, radishes)
- Cream Crackers (6 unsalted)

Late-snack: 4
Fruit: Choice of two: banana, orange, pear, apple peach or grapefruit

Late-snack: 5
Whole wheat bread roll with ½ teaspoon cream cheese

Late-snack: 6
- Cereal: Choice of shredded wheat or 40% bran (½ cup 125 ml)
- Milk (½ cup low-fat or skim 125 ml)
- Banana (1 whole 100g)

Late-snack: 7
Repeat your favourite meal above

RE-ENTRY PHASE

Breakfast every day

Fruit juice (½ cup)
choice of unsweetened or fresh grapefruit, orange, apricot or prune juice

Cereal (¾ cup)
Choose 40% Bran Albran, Raisin Bran or other high fibre cereal

Milk (½ cup)
Low-fat (2%) or skim milk

Fruit (1 whole
Choice of
Orange
Pear
Banana
Apple
Grapefruit
Peach

Lunch: 1
- Chicken salad sandwich: Chicken salad (90g diced chicken mixed with 1 table-spoon mayonnaise)
- Whole wheat bread (2 pieces)
- Lettuce (1 or 3 leaves on sandwich)
- Pear (½ 120g)

Lunch: 2
- Hamburger: Hamburger roll (1 whole), beef patty (90g of broiled lean beef) with ½ tablespoon mayonnaise or 2 tablespoons tomato sauce
- Lettuce (1 or 2 leaves)
- Tomato (120g)

Lunch: 3
- Cottage cheese ($^{1}/_{3}$ cup, low-fat 45g)
- Sweet melon or fruit in season (120g)
- Strawberries (½ cup sliced 120g)
- Lettuce (2 or 3 leaves)

Lunch: 4
- Omelette (2egg omelette filled with tomatoes, peppers, onions and cheddar cheese)
- Bread or roll (2pieces with 2 tablespoons diet margarine)

Lunch: 5
- Tuna fish sandwich: tuna (60g with 1 tablespoon mayonnaise)
- Bran bread (2 slices)
- Lettuce (1 or 2 leaves)
- Tomato (2slices)
- Peach (1 whole 120g)

Lunch: 6
Large salad: (large salad bowl) with lettuce, tomato, onion, carrots, pepper, radishes, diced chicken or turkey and croutons, topped with 2 tablespoons salad dressing

Lunch: 7
- Open-faced grilled cheese and tomato sandwich (2 slices of whole wheat or rye bread, each topped with slices of tomato and diet cheese, grilled lightly under broiler)

Dinner: 1
- Baked or broiled fish, shrimp, crab or lobster meat (180g)
- Tossed salad (small bowl of lettuce, tomato, onion, radish mixture with 2 tablespoons salad dressing
- Sweet potato (1 whole, ½ teaspoon diet margarine)
- Vegetable: choice of carrots, green beans broccoli, spinach, asparagus (½ cup)

Dinner: 2
Macaroni and cheese (1 cup 250 ml) asparagus spears (4), tomato (1 whole grilled under broiler with tarragon 120g)

Dinner: 3
- Steak (150g, broiled, visible fat removed)
- Baked potato (1 whole with 1 tablespoon diet margarine)
- Vegetable: choice of corn, peas, beans spinach (½ cup), salad (small bowl of raw spinach, onions, radishes, cucumber and carrots with 2 tablespoons diet dressing)

Dinner: 4
- Baked chicken (150g or 2 breasts)
- New potatoes (½ cup 100g)
- Vegetable: choice of carrots, spinach, asparagus, green beans (1 cup)
- Baked apple (1 whole, cut in half, topped with cinnamon and sugar substitute, baked for 2-30 minutes at 180c

Dinner: 5
- Baked or broiled fish (180g)
- Rice (1 cup, tablespoon diet margarine)
- Vegetables: corn, peas, beans (1 cup)
- Orange slices (1 cup 120g)

Dinner: 6
- Roast beef, lamb or veal (150g visible fat removed)
- Baked potato (1 with 2 tablespoons sour cream)
- Vegetables: choice of asparagus, green beans, carrots, broccoli, spinach (1 cup)
- Bread or roll (2 pieces, no margarine)

Dinner: 7
- Spaghetti (2 cups cooked noodles)
- Meatless sauce (180 ml commercially prepared sauce)
- Parmesan cheese sprinkled lightly over spaghetti 30g
- Bread (2 medium slices of Italian or French bread, 1 tablespoon diet margarine)

Late-snack: 1
- English muffin (1 whole with 1 tables. diet margarine, 1 tables. jam or jelly))
- Grapes (1 cup) or seasonal fruit

Late-snack: 2
- Cream crackers (6)
- Milk (1 cup, low-fat)

Late-snack: 3
- Whole wheat bread roll (1 whole with 2 tbles. Cream cheese)
- Fruit: choice of orange, apple, pear (1 whole 120g)

Late-snack: 4
- Raw vegetable plate (mix raw carrot sticks, celery radishes and cauliflower
- Melba toast (4 pieces with 2 tbles. Diet margarine)
- Juice: choice of orange, apple, grapefruit or prune

Late-snack: 5
Yoghurt (250 ml of any fruit-flavoured yoghurt)

Late-snack: 6
- Popcorn (4 cups, 1 tbles. Diet margarine, no salt)
- Fruit: choice of apple, orange, pear, peach, melon (1 whole 120g)

Late-snack: 7
Repeat your favourite meal above

APPENDIX F

RANDOMIZED TRIAL SYNOPSIS

***Design** : Pretest-posttest, double-blind placebo-controlled trial.

***Randomisation** : Subjects assigned to three groups using a table of random numbers.

***Participant flow:**

- Enrollment : n = 76
- Allocation : Group TS = 26
  Group EST = 25
  Group ESP = 25

- Follow-up : Withdrawals: n = 7
  Medical reasons: n = 3
  Personal reasons: n = 4

- Analysis : n = 69
  Group TS = 23
  Group EST = 23
  Groups ESP = 23

  - Group TS : Thermogenic Stimulation and following a standardized diet.

  - Group EST : Electrical Muscle Stimulation and Thermogenic Stimulation combined and following the standardized diet.

  - Group ESP : Electrical Muscle Stimulation and Thermogenic Placebo combined and following the standardized diet.

***Allocation Concealment** : Product (Thermo Lean) and placebo housed in securitainers of same color and size. Distinction between product and placebo made with a letter of the alphabet e.g. A; B or C.

***Blinding** : Double blinding used with the study leader keeping record of subject group allocation. Group allocation revealed post data analysis.

APPENDIX G

NOMOGRAPHIC CHART FOR COMPUTING BODY SURFACE AREA (BSA)

(nothing available)