The effect of a herbal formulation on Body Mass Index and abdominal girth measurements in overweight and obese individuals

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DECLARATION

I declare that this research dissertation is my own, unaided work. It is being submitted for the degree of Master's of Technology: Homoeopathy at the University of Johannesburg. It has not been submitted before for any degree or examination in any other Technikon or University.

_____

Robert Durrheim

This _____ day of _____ 2012

AFFIDAVIT

ABSTRACT

In South Africa, approximately 61% of the population is believed to be overweight, obese or morbidly obese (Smith, 2010). Risk factors to developing obesity include a sedentary lifestyle, unhealthy diet and poor eating habits, smoking, age, medications such as corticosteroids and other illnesses such as polycystic ovarian syndrome, hypothyroidism and Cushing's syndrome (Polsdorfer, 2011). Obesity is fast becoming a major problem in all communities in South Africa, not only in regard to the health of individuals but as it continues to increase the costs of health care in the country (Goedecke *et al.,* 2005).

The aim of this study is to determine the effect of a herbal formulation consisting of Caffeine*, Coffea canephora* bean*, Coleus forskholii, Camellia sinensis,* Evodiamine*, Ilex paraguariensis* and *Phaseolus vulgaris* on Body Mass Index and abdominal girth measurements in overweight and obese individuals.

Previous studies conducted on the herbal formulation have shown positive results with regard to weight loss, however, the need for a longer trial period was indicated in order to establish long term results as in this study (Baillie, 2011a).

The study was a twelve week, double-blind, placebo-controlled study. The participants were males and females between the ages of 18 and 45 years with a BMI between 25 and 35kg/$m^{2.}$ Sixty participants were recruited from the University of Johannesburg, as well as from the public sector, in response to posters posted at the university, local gymnasiums and fitness clubs and given to other Homoeopathic practitioners. Of the sample of sixty participants, thirty participants were placed in the experimental group and thirty in the placebo group according to matched pairing of gender and BMI. The groups received either two capsules of the herbal formulation consisting of Caffeine*, Coffea canephora* bean*, Coleus forskholii, Camellia sinensis,* Evodiamine*, Ilex paraguariensis* and *Phaseolus vulgaris* or two capsules of a placebo composed of pharmaceutical starch, from Monday to Friday.

At the initial consultation, a detailed case history and the vital signs (including blood pressure, respiratory rate, heart rate and temperature) of the participants were taken. Their height and weight was determined and from these measurements, their BMI was calculated. Their abdominal girth was measured three times during each consultation, each time using a standardized method and the average measurement was obtained. The participants then returned for follow-up evaluations in the second, sixth and twelfth week of the study. At each follow-up

consultation, the participants' vital signs and abdominal girth measurements were taken again and their BMI calculated by measuring their weight.

The data collected during the study was analysed using statistical techniques including the Shapiro-Wilk test, Friedman Analysis of Variance test, Mann-Whitney test and Wilcoxon Signed-Rank test.

From the statistical analysis it was determined that the herbal formulation containing Caffeine, *Coffea canephora* bean, *Coleus forskholii, Camellia sinensis,* Evodiamine, *Ilex paraguariensis* and *Phaseolus vulgaris* was ineffective compared to placebo in decreasing weight and BMI in overweight and obese individuals over a twelve week period.

DEDICATION

This research is dedicated to my family and friends, for all their love and support for me in following my dream.

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Robert	Grandfather: For always believing in me and supporting me. I've finally reached my goal.
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CHAPTER 1

INTRODUCTION

1.1 Problem statement

Obesity has been established as a chronic disease and one of the most prevalent contributors to other chronic conditions (Goedecke *et al.,* 2005). In South Africa, approximately 61% of the population is believed to be overweight, obese or morbidly obese (Smith, 2010). Risk factors to developing obesity include a sedentary lifestyle, unhealthy diet and poor eating habits, smoking, age, medications such as corticosteroids and other illnesses such as polycystic ovarian syndrome, hypothyroidism and Cushing's syndrome (Polsdorfer, 2011).

Body Mass Index (BMI) is an indicator used by practitioners to determine if an individual is overweight or obese by dividing their weight in kilograms by the square of their height in metres. BMI has been linked to an increased risk of early death in both males and females. It was found that for every 5 unit increase in BMI for men and women, their risk of death increased by 31% (National Institute of Health, 2010). An increase in the abdominal girth and abdominal obesity has been found to be a major precursor for coronary heart disease (Allison *et al.,* 2004). Increased abdominal girth has also been linked as being a possible marker for early diagnosis of metabolic syndrome and may be an indicator of a dysmetabolic state and *in vivo* insulin resistance (Després and Lemieux, 2006).

Some complications of obesity that may eventually lead to death include cerebrovascular accidents, hypercholesterolaemia, non insulin dependant diabetes mellitus (NIDDM), hypertension, coronary heart disease, gall bladder disease, certain types of cancer and non-alcoholic fatty liver disease (Centers for Disease Control and Prevention, 2004).

Obesity is fast becoming a major problem in all communities in South Africa, not only in regard to the health of individuals but as it continues to increase the costs of health care in the country (Goedecke *et al.,* 2005).

1.2 Aim of the study

The aim of this study is to determine the effect of a herbal formulation consisting of Caffeine*, Coffea canephora* bean*, Coleus forskholii, Camellia sinensis,* Evodiamine*, Ilex paraguariensis* and *Phaseolus vulgaris* on Body Mass Index and abdominal girth measurements in overweight and obese individuals.

1.3 Hypothesis

It is anticipated that the herbal formulation consisting of Caffeine, *Coffea canephora* bean, *Coleus forskholii, Camellia sinensis,* Evodiamine, *Ilex paraguariensis* and *Phaseolus vulgaris* will cause a decrease in weight and BMI, as well as reduce abdominal girth measurements compared to a placebo.

1.4 Null-hypothesis

The herbal formulation consisting of Caffeine, *Coffea canephora* bean, *Coleus forskholii, Camellia sinensis,* Evodiamine, *Ilex paraguariensis* and *Phaseolus vulgaris* will not cause a decrease in weight and BMI, nor will it reduce abdominal girth measurements compared to a placebo.

1.5 Objective and expected outcome

The objective of this study is to determine the effect of a herbal formulation consisting of Caffeine, *Coffea canephora* bean, *Coleus forskholii, Camellia sinensis,* Evodiamine, *Ilex paraguariensis* and *Phaseolus vulgaris* on BMI and abdominal girth measurements in overweight and obese individuals.

The expected outcome of this study is that the herbal formulation will decrease the BMI and abdominal girth measurements of participants. This will thereby assist in weight loss as well as reduce further health risks associated with overweight and obesity.

CHAPTER 2

LITERATURE REVIEW

2.1. Overweight and Obesity

Obesity as defined by the World Health Organization (2000) is, "abnormal or excessive fat accumulation that presents a risk to health". It has been labelled as an epidemic and is associated with many other diseases that are the result of obesity and cause a high mortality rate (Knobler and Malnick, 2006).

There are two major storage sites for fat in the body, the liver and adipose tissue; while muscles may store a small amount. When fat is deposited in adipose tissue around the abdomen, it is called central or abdominal obesity. Abdominal obesity is common amongst males and is associated with an increased risk of developing Type 2 diabetes mellitus. Females usually have an increase in fat deposition around the hips and buttocks, which is associated with an increased risk of developing osteoarthtitis (Escott-Stump, 2011).

Abdominal obesity is a serious health concern as it exposes people to other serious health risks regardless of their BMI. This is why it is important to monitor both BMI as well as abdominal girth in individuals who are overweight or obese (Centers for Disease Control and Prevention, 2004).

Some complications of overweight and obesity that may eventually lead to death include cerebrovascular accidents, hypercholesterolaemia, non insulin dependant diabetes mellitus (NIDDM), hypertension, coronary heart disease, gall bladder disease, certain types of cancer and non-alcoholic fatty liver disease (Centers for Disease Control and Prevention, 2004).

Risk factors to developing obesity include a sedentary lifestyle, unhealthy diet and poor eating habits, smoking, age, medications such as corticosteroids and other illnesses such as polycystic ovarian syndrome and Cushing's syndrome (Polsdorfer, 2011).

2.1.1 Physiology of fat deposition

Obesity occurs when there is an increase in the deposition of fat in adipocytes, or fat cells, around the body. The amount of adipocytes in the body is determined within the first few weeks of an infant's life. Under normal circumstances the amount of adipocytes will remain the same for an individual's lifetime, with only the size of the adipocytes increasing (Martini *et al.*, 2006).

However, in cases where there is a constant, long term elevation of circulating triglyceride levels, mesenchymal cells may divide and differentiate into adipocytes (Coppack *et al.,* 2003).

Fats are broken down into triglycerides in the gut and after they are absorbed from the gastrointestinal tract, they are transported by the lymphatic system into the blood stream in the form of chylomicrons. If the triglycerides are not needed to produce energy immediately, they enter adipocytes. The membranes of adipose cells contain lipoprotein lipase, an enzyme which hydrolyses or splits the triglycerides in the chylomicrons into glycerol and fatty acids. These fatty acids then diffuse across the cell membrane and into the adipocyte. Here, glycerol synthesized within the adipocyte and the fatty acids which entered the cell recombine to form triglycerides once again. These triglycerides are stored until they are needed to provide energy elsewhere in the body. Carbohydrates are usually broken down into simple sugars and either used for energy production or they are stored as glycogen. If glycogen stores are full, carbohydrates are converted into triglycerides and stored in adipocytes (Guyton and Hall, 1997).

When an individual's caloric intake exceeds their caloric expenditure, the body will store the excess nutrients. Therefore the amount of triglycerides stored is a direct reflection of the imbalance between energy intake and energy expenditure (Coppack *et al.,* 2003).

2.2 Body Mass Index (BMI)

BMI is used as an indicator or screening method for the amount of adipose tissue an individual has. It is used by health care practitioners as a good indication of whether a patient is underweight, average weight, overweight or obese compared to the general population (Beers *et al.,* 2006). This is done by using a standardized calculation and then reading this value off a graph to determine which category a participant is in. BMI is also used to assess the risk of a patient for other health problems such as metabolic syndrome and cardiovascular disease (Centers for Disease Control and Prevention, 2004).

The Center for Disease Control and Prevention and the World Health Organization has defined a normal BMI range as 18.5 to 24.9 kg/m^2; overweight is defined as a BMI of 25.0 to 29.9 kg/m^2; obesity is defined as a BMI over 30.0 kg/m^2 and morbid obesity is defined as a BMI 35 kg/m^2 or higher (National Institute of Health, 2010).

When measuring an individual's BMI certain factors such as athleticism and muscle build/mass are taken into consideration. This is because athletes may be more muscular than average individuals and therefore on the BMI chart, they may be classified as overweight. It is important to distinguish these factors in order to properly evaluate an individual's health risk, as a person

with abdominal obesity may have the same BMI as a muscular athlete but their health risks will be vastly different (Beers *et al.*, 2006).

2.3 Abdominal girth

Abdominal girth is the most widely used assessment of abdominal obesity and the monitoring of changes in abdominal fat by practitioners. Abdominal fat can be divided into two categories, subcutaneous fat and intra-abdominal fat. Subcutaneous fat is the fat which is deposited just below the skin and intra-abdominal or visceral fat is the fat which exists inside the abdominal cavity between the organs or viscera (International Chair on Cardiometabolic Risk, 2008).

Abdominal girth has been shown to be reduced in sedentary, overweight postmenopausal women who underwent exercise regimes of different intensity. In this study there was no weight loss evident, concluding that weight loss and abdominal girth do not always coincide with one another but should be evaluated separately and independently (Blair *et al.,* 2009). Abdominal girth measurements are an important independent indicator for determining potential risks associated with an increased amount of abdominal fat (Allison *et al.,* 2002b), as well as for the monitoring of abdominal fat loss with weight loss regimes (Hudson *et al.,* 1996).

An increase in abdominal girth of over 93cm in men and 79cm in women is a risk factor for obesity and the complications thereof (Beers *et al.,* 2006). It has been indicated that general obesity has many health hazards associated with it, however abdominal obesity has been shown to be a better predictor of cardiovascular disease and diabetes mellitus than general obesity (Lamarche, 1998). Increased abdominal girth measurements were also shown to increase the likelihood of an individual developing hypertension, diabetes, dyslipidaemia and metabolic syndrome irrespective of their BMI (Janssen *et al.,* 2002).

2.4 Aetiology of overweight and obesity

2.4.1 Diet

Body weight is determined by the amount of energy consumed compared to the amount of energy expended. Any imbalance between energy consumption and energy expenditure will result in either weight loss or weight gain. The amount of energy a person needs in order to perform a certain task is called metabolic efficiency. This metabolic efficiency differs from person to person and is said to be the reason why some people are able to lose weight and keep that weight off, while others struggle not to regain lost weight (Dokken and Tsao, 2007).

Large portion sizes, eating convenience food as well as the consuming foods high in energy, and therefore high in calories, all result in high energy intake. This high intake of energy, if not used efficiently, has the ability to cause weight gain (Hensrud, 2004).

Increased urbanization within South Africa has caused the general population to shift to a more westernized diet. This means that people are ingesting a higher content of fat and less complex carbohydrates and fibre in their diets (Goedecke *et al.,* 2005).

Low carbohydrate diets have shown to decrease weight as well as reduce the risk of heart disease by increasing the level of high density lipoproteins and decreasing the level of low density lipoproteins over a six month period (Brill *et al.,* 2003). However, high fat diets have been shown to promote the accumulation of adipose tissue considerably more than a diet high in carbohydrates (Goedecke *et al.,* 2005).

In order to reduce weight gain, a lower total energy intake is needed. This means an eating plan low in calories, saturated fat and refined carbohydrates and high in fruits, vegetables and whole grains should be adopted for an extended period of time. This ensures a loss of weight as well as a prolonged maintenance of this weight loss (Hensrud, 2004).

A study conducted in 1995 found that when participants were made to gain 10% of their body weight, their non-resting energy expenditure increased. This suggested that muscle uses more energy post weight gain in order for the body to inhibit further weight gain. After a 10% body weight loss in the same experiment it was noticed that energy expenditure decreased in order to decrease further weight loss. This study may explain why some people who partake in dieting struggle to lose the last amount of weight they want to and why it is sometimes effortless to gain the weight which was recently lost. It can, however, be said with certainty that as long as energy expenditure exceeds energy intake, weight loss will occur (Dokken and Tsao, 2007).

2.4.2 Physical activity

A low amount of physical activity is a large contributing factor to a sedentary lifestyle and therefore to the development of weight gain and obesity (Ahrens *et al.,* 2012). A study conducted in the North West province of South Africa showed that a decrease in physical activity was a major contributor to an increase in BMI in black women and as a result this increased the risk for other metabolic illnesses to be established (Kruger *et al.,* 2002).

Approximately one third of the amount of energy used by a person each day is done so through the process of muscular contraction. Therefore the relationship between food intake and physical

activity is important and it shows that reduced physical activity will contribute to overweight and obesity (Guyton and Hall, 1997).

Exercising increases energy use, thermogenesis and basal metabolic rate. Muscle tissue uses more calories at rest than fat tissue. This means that by doing strengthening exercises such as muscle building, a lasting, high resting metabolic rate can be maintained. Several other benefits are gained from physical activity such as regulation in appetite, increased sensitivity to insulin, improvement in ratio of plasma lipids, decreased blood pressure, increase in bone density and psychological wellness (Beers *et al.,* 2006).

2.4.3 Genetics

Obesity is determined by many factors and as the growing body of evidence shows, genetics is one of these factors (Dokken and Tsao, 2007). An individual's BMI has been said to be one third heritable. These genetic factors regulate processes of thermogenesis, basal metabolic rate as well as energy expenditure (Beers *et al.,* 2006).

Recent studies conducted on monozygotic twins show that the responsiveness of these individuals to an increased or decreased caloric diet are very similar with regards to the changes in bodyweight and composition (Loos and Rankinen, 2005).

Genetic predispositions to obesity have been described as mutations in genes causing phenotypic changes. These genes are usually those needed for the production of certain proteins in the body as well as the regulation of certain metabolic processes (Dokken and Tsao, 2007). These mutations cause a disruption in the hypothalamic pathways which are responsible for the regulation of satiety and food intake and the suppression of appetite. This evidence suggests that obesity may have an underlying cause apart from environmental factors and that it may not only be a metabolic disease but a neuro-behavioural disease with metabolic changes (Farooqi and O'Rahilly, 2006).

Genetics can cause abnormal feeding in several ways. The neurogenic feeding centres in the hypothalamus may be programmed for high or low amounts of energy storage, thereby increasing or decreasing weight gain. Genetics may also cause the person to eat food as a mechanism of "release" in certain situations such as stress (Guyton and Hall, 1997).

There are over 50 loci which have been linked to obesity, making it a very complicated phenotype. Genetics therefore may play a larger role in obesity than previously anticipated, and together with certain environmental conditions, may predispose certain people to eventually develop obesity (Goedecke *et al.,* 2005).

2.4.4 Gender

An individual may be predisposed to a certain percentage of body fat due to their gender. The reason for this is because of the dominance of certain sex hormones in the male and female body. Males secrete testosterone, which is an anabolic hormone, that promotes protein deposition around the body. Females secrete larger quantities of oestrogen; which promotes fat deposition, especially in the breasts, hips and subcutaneous tissue (Guyton and Hall, 1997).

In males between the ages of 50 and 60 years, the levels of testosterone begin to decline. This decline causes a decrease in muscle and bone growth and an increase in fat deposition and therefore weight gain (Martini *et al.*, 2006).

Menopause in females usually occurs between the ages of 45 and 55 years but the decline in oestrogen levels usually precedes menopause by several years, disrupting the ovarian and uterine cycles. During these years the primordial follicles respond less and less to follicle stimulating hormone (FSH) and therefore levels of oestrogen decline (Martini *et al.*, 2006). On average, a woman may gain up to eight kilograms in around the time of menopause. There are two theroies as to why this occurs. Firstly, fat cells are able to produce oestrogen if the oestrogen levels in the body are low. The decline in oestrogen production from the ovaries during menopause may cause an increase in fat deposition in order for the fat cells to produce the amount of oestrogen needed. Another theory is that there is a decrease in thyroid function during menopause and therefore a decrease in thyroid hormone production. This will lead to a decrease in metabolic rate and therefore weight gain (Moskowitz, 2008).

A decrease in growth hormone production with ageing in both sexes will also contribute to a decline in muscle mass and therefore in metabolic rate causing weight gain (Neighmond, 2010).

2.4.5 Age

Ageing causes a decrease in the rate of energy consumption and an alteration in normal hormone levels. These factors together with an increase in sedentary lifestyle, poor eating habits and a decrease in physical activity with age all contribute to weight gain (Martini, 2006).

Age related weight gain may occur due to a decrease in physical activity as an individual ages (Williams and Wood, 2005). Decreasing physical activity may contribute to muscle loss as ageing occurs, but other contributing factors may play a role. Stem cells within the muscles may not respond to growth or repair as efficiently as they did at a younger age, leading to a decrease in muscle mass (Neighmond, 2010). This decrease in muscle mass will cause an increase in adipose deposition as a decrease in muscle may cause a decrease in the resting metabolic rate (Guyton and Hall, 1997).

2.4.6 Hypothyroidism

Hypothyroidism is classified as an endocrine disorder whereby there is a deficiency in thyroid hormone (Beers *et al.,* 2006). Thyroid hormone is made by the follicle cells in the thyroid gland and is involved in the production of adenosine triphosphate (ATP) or energy by the mitochondria. The overall result is an increase in metabolic activities of all cells in the body as well as an increase in the basal metabolic rate (Guyton and Hall, 1997).

Due to the physiological function of thyroid hormone and the lack of it in hypothyroid individuals, weight gain is usually present. This weight gain is the result of both fluid retention and a decreased metabolism (Beers *et al.,* 2006). Thyroid hormone plays a large role in fat metabolism by greatly increasing fat oxidation and lipid mobilization from the fat stores. In hypothyroidism, however, this hormone is diminished thereby decreasing fat mobilization and increasing body weight (Guyton and Hall, 1997). Other symptoms of hypothyroidism include intolerance to cold, constipation, forgetfulness, voice hoarseness, facial puffiness, decreased appetite and dry skin (Beers *et al.,* 2006).

2.4.7 Cushing's syndrome

Weight gain is one of the most common symptoms in people who develop Cushing's syndrome. Fat is usually deposited on the posterior cervical area and is known as a "buffalo hump". Fat deposition also often occurs on the trunk, face and abdomen more often than the limbs where muscle wasting is often seen instead. The reason for this fat deposition is the excess levels of cortisol or hydrocortisone in the body (Nieman, 2011).

Excess secretion of glucocorticoids such as cortisol from the adrenal glands may be caused by an increased secretion of Adrenocorticotrophic Hormone (ACTH) from the pituitary gland or external administration of corticosteroid medication. The high levels of cortisol in the body promote gluconeogenesis and the mobilization of free fatty acids. These factors coupled with the fact that there is also a decrease in protein synthesis and an increase in protein catabolism are the reasons why there is an increase in fat deposition and an increase in muscle wasting (Guyton and Hall, 1997).

Cushing's syndrome may either be ACTH-dependent or ACTH-independent hyper-function of the adrenal glands. ACTH-dependent hyper-function occurs when there is an increased secretion of ACTH by the pituitary gland or ACTH secreting tumour or by the administration of exogenous ACTH. ACTH-independent hyper-function usually occurs due to the administration of corticosteroid medications. However, either mechanism will cause the same symptom picture (Beers *et al.,* 2006).

2.4.8 Psychological disorders

- **Depressive disorders**

  Depressive disorders are characterized by sadness, severe and persistent enough to interfere with an individual's day to day functioning, and a decreased interest or pleasure in normally pleasurable activities (Beers *et al.,* 2006).

  Major depression was found to be a significant predictor in adolescents for the development of an elevated BMI in adulthood compared to adolescents who did not suffer from depressive episodes (Allison *et al.,* 2002a).

  Depression may either be the cause for obesity or the result of obesity. The physical, social and emotional stresses of being obese or overweight greatly increase the risk of people developing depression (Cooke and Wardle, 2007). Obesity may not be the cause of depression but the physical and emotional impact of being obese may lead to the development of depression as people are less able to cope with their situation (Bailey, 2009).

  However, emotional eating and binge eating disorders have also been found to be associated with disorders such as depression and other stress related disorders (Beers *et al.,* 2006).

- **Anxiety disorders**

  Anxiety is a pervasive fear that may be known or unknown and which dominates a person's life or is only experienced in certain situations (Martin, 2007).

  Positive associations between obesity and anxiety disorders have been found (Christensen *et al.,* 2007; Gariepy *et al.,* 2009; McGee, *et al.,* 2007). The relationship between anxiety and obesity is still not fully understood and it is unclear whether obesity causes anxiety or whether anxiety causes obesity due to excessive eating. However, there is a growing body of evidence that the two are related (Bailey, 2009).

- **Stress**

  Stress is defined as any factor which threatens the health of the body or has an adverse effect on its functioning (Martin, 2007).

  A 2004 study showed that Danish nurses had an increase in weigh gain when subjected to intense psychological workload and certain job stressors over a six year period (Gamborg *et al.,* 2004). A group of full time Australian employees were analyzed over a four year

period and showed that certain occupational factors such as extensive or inflexible working hours significantly predicted an increase in BMI (Caputi *et al.,* 2010).

A possible reason or theory suggested by Akana *et al,* is that the chronic secretion of glucocorticoids enhances compulsive activity thereby leading to an increase in the ingestion of "comfort food", usually involving sucrose and fat. This compulsive activity is said to be a reaction by a stressed or depressed person in order to try and decrease the activity of the chronic stress response (2003).

The chronic stress response, also known as the general adaption syndrome (GAS) is a hormonal response to both acute and chronic physical or emotional stress. The ultimate aim of this response is to maintain homeostasis of the body by activating certain hormonal responses. The effects of long term GAS are to mobilize protein and lipid reserves, conserve glucose for neural tissue use, elevate blood glucose concentrations and conserve salts and water (Martini et al., 2006).

Therefore, in order to elevate glucose levels and use lipids for primary body functions, the individual will begin to eat "comfort food" (Akana *et al.,* 2003).

- **Binge eating disorders**

Binge eating disorder is described as the consumption of excessive amounts of food in a short amount of time which is not followed by actions such as self induced vomiting or the abuse of laxatives (Beers *et al.,* 2006).

Approximately 30% of individuals who investigate treatment for obesity have binge eating disorder. The prevalence of this disorder has been found to be 2-5% of the population (de Zwaan, 2001).

Binge eating disorder is usually very distressing for the individual as many of these people are already obese and trying to lose weight. This disorder usually goes hand in hand with psychological illnesses or strong emotional states such as depression, stress, anger, worry and boredom which may then cause further overeating. It is not known whether people with depression develop binge eating disorder or whether binge eating disorder causes depression, however the two have been shown to be related (National Institute of Health, 2008).

It is therefore important to treat both the psychopathology or eating disorder and the obesity of the patient to get optimal results (de Zwaan, 2001).

2.4.9 Medication linked to weight gain and obesity

- **Corticosteroids**

  Corticosteroids are drugs which are used for a number of different illnesses such as adrenal gland disease, rashes and inflammatory disorders such as arthritis (Chilnick *et al.,* 2008).

  Corticosteroids cause an increase in gluconeogenesis, elevating the levels of glucose in the blood and therefore increasing insulin levels. This may result in diabetes with long term use. Gluconeogenesis requires the breakdown of amino acids as well as lipolysis to be used as energy and building blocks of glucose (Champe and Harvey, 2009).

  Adverse effects of excess intake of corticosteroids include an increase in appetite as well as weigh gain. The characteristic pattern of fat deposition in Cushing's syndrome results in a puffy face and trunkal weight gain. This is due to the inability of the body to absorb glucose effectively thereby promoting fat deposition (Cheskin, 2007).

- **Diabetic medication**

  Common oral hyperglycaemic drugs include Thiazolidinediones and Sulfonylureas, both of which have been shown to cause weight gain (Champe and Harvey, 2009).

  Thiazolidinedione is a drug which is used to treat diabetic patients by increasing peripheral tissue insulin sensitivity. They may cause weight gain through increased subcutaneous fat deposition or because of an increase in fluid retention caused by the drug (Champe and Harvey, 2009).

  Sulfonylureas promote the secretion of insulin from the pancreas and increase peripheral tissue sensitivity to insulin, thereby lowering plasma glucose levels. However, these drugs have been shown to cause weight gain, hyperinsulinaemia and hypoglycaemia (Champe and Harvey, 2009).

- **Antidepressants**

  Approximately 25% of people who are administered antidepressant medication gain weight of up to 5 kilograms. Selective serotonin reuptake inhibitors (SSRI's), Monoamine oxidase inhibitors (MAO's) and Tricyclic antidepressants (TCA's) have all been shown to induce weight gain in both long and short term use (Bouchez, 2007).

The side effect of weight gain is a major problem with regards to patient compliance as patients who do gain weight while taking the medication may be reluctant to continue treatment (Deshmukh and Franco, 2003).

Weight gain occurs due to either an impairment or decrease in metabolism, or due to an increase in appetite and therefore increase in consumption of food. This weight gain may cause further depression for patients if they prematurely stop their medication. The suggested management of this weight gain includes education of the patient on the possible side effects; the introduction of a calorie restricted diet; increased physical activity and possibly the administration of an alternative drug that may not cause weight gain (Deshmukh and Franco, 2003).

2.5 Harmful effects of overweight and obesity

2.5.1 Cerebrovascular accidents

A cerebrovascular accident or a stroke is a disorder characterized by the sudden interruption of blood flow to a specific area in the brain. A stroke can either be ischaemic (80%) or haemorrhagic (20%) depending on the cause (Beers *et al.*, 2006).

A study conducted in 2002 showed that abdominal obesity is a very strong potential risk factor for the development of an ischaemic stroke (Boden-Albala *et al.,* 2003).

Obesity was shown to be a risk factor for strokes in adult men who were monitored over a 12.5 year period. This study showed that there was a significant increase in the risk of a stroke for every unit increase in BMI (Berger *et al.*, 2002).

2.5.2 Hyperlipidaemia

Hyperlipidaemia occurs when there is an elevation of plasma cholesterol and triglyceride levels. Elevated levels of cholesterol and triglycerides may predispose an individual to the development of atherosclerosis (Beers *et al.*, 2006).

In the year 2000 it was estimated that approximately five million South African adults were suffering from hyperlipidaemia (Steyn, 2007). It was determined by a study conducted in 2007 that 59% of ischaemic heart disease is caused by hypercholesterolemia in adults over 30 years of age and that 4.6% of all South African deaths are caused by high cholesterol levels (Bradshaw *et al.,* 2007).

Adipose tissue not only stores fat but also acts as an endocrine organ, releasing certain factors and bioactive mediators in the body, which alter lipid metabolism resulting in dyslipidaemia.

This may then result in further complications such as hyperlipidaemia, atherosclerosis and cardiovascular disease (De Bloc *et al.,* 2006).

2.5.3 Type 2 Diabetes mellitus

Type 2 diabetes mellitus or NIDDM has been described as an epidemic and is a risk factor for developing heart disease, strokes, blindness, kidney disease and nerve disorders (Ahima *et al.*, 2001).

Diabetes mellitus occurs when there is an impaired insulin secretion and peripheral insulin resistance, which leads to hyperglycaemia. Type 2 diabetes mellitus occurs when insulin secretion can no longer compensate for peripheral tissue insulin resistance. High levels of insulin are discovered early in the disease but the increase in gluconeogenesis by the liver and decrease in peripheral tissue uptake of glucose eventually causes the very high insulin levels to become insufficient. The insulin level then decreases over time, causing hyperglycaemia later in the disease process (Beers *et al.*, 2006).

Obesity, lifestyle, diet and exercise all have some effect on glucose metabolism. Adipocytes have been shown to release certain factors which ultimately adversely affect the glucose metabolism (Beers *et al.*, 2006). Resistin is a signaling molecule produced by adipocytes. The function of this hormone is to reduce insulin sensitivity throughout the body so that glucose is not absorbed (Martini et al., 2006). Resistin has shown to be a possible link between obesity and Type 2 diabetes mellitus (Ahima *et al.,* 2001).

The risk of developing Type 2 diabetes mellitus in overweight and obese adults is the same for all ages, sexes and races and is associated with other major health risks (Bales *et al.,* 2003). However the risk of developing Type 2 diabetes mellitus increases with the advancing gain in weight and severity of central obesity, as well as with the length of time an individual is overweight. It has been shown that the risk of developing Type 2 diabetes mellitus increases 40 fold when the BMI of an individual increases from 22 to 35 kg/m^2 (Goedecke *et al.,* 2005).

2.5.4 Hypertension

Hypertension occurs when there is a constant elevation of the arterial blood pressure. The resting systolic value is 140mmHg or more and the resting diastolic value is 90mmHg or more, in hypertensive individuals (Beers *et al.*, 2006).

Many mechanisms linking obesity to the development of hypertension have been identified. These mechanisms include the activation of the renin-angiotensin-aldosterone system, endothelial dysfunction and renal impairment (Allyn *et al.,* 2004).

Obesity is associated with an increased risk of developing hypertension by approximately 3.5 times (Goedecke *et al.,* 2005).

2.5.5 Coronary heart disease

Coronary heart disease occurs when there is an impairment of blood flow through the coronary arteries of the heart. The most common cause of cardiac ischaemia is the development of atheroma's within the blood vessel walls. Ischaemia of the myocardium leads to symptoms of angina pectoris and may cause a myocardial infarction (Beers *et al.*, 2006).

Obesity has been associated with an increased risk for developing coronary heart disease, increasing to 2.8 times for men and 3.4 times for women (Goedecke *et al.,* 2005). Further studies have shown the relationship between obesity being a risk factor for coronary heart disease and Type 2 diabetes (Abbasi *et al.,* 2002).

Between 1997 and 2004 it was estimated that approximately 195 people in South Africa died per day because of some form of heart or blood vessel disease. Premature deaths caused by cardiovascular disease are expected to rise by 41% in people between the ages of 35 and 64 years by the year 2030 and will have a major socioeconomic impact on the country (Steyn, 2007).

2.5.6 Gall bladder disease

Gall bladder disease has many environmental causes. Obesity is one of the most important risk factors for the development of gall stones or cholelithiasis (Chávez-Tapia *et al.,* 2004).

More than 85% of all gallstones are made of cholesterol. Cholesterol stones form when there is a supersaturation of bile with cholesterol due to an increase in cholesterol secretion or due to a decrease in bile salt secretion (Beers *et al.*, 2006).

During periods of weight loss where 24% or more of the body weight is lost or more than 1.5 kg in weight is lost per week, the risk of cholelithiasis formation is also increased. This is due to the increased amount of cholesterol circulating through the portal system and therefore a supersaturation of bile (Erlinger, 2000).

The prevalence of gallstone formation is highest in obese individuals, especially in those in the highest categories of BMI (Erlinger, 2000).

2.5.7 Non-alcoholic fatty liver disease (NAFLD)

NAFLD occurs when there is an excessive accumulation of triglycerides in hepatocytes which later cause liver damage. Once fatty infiltration has occurred, inflammation and later fibrosis of the liver can occur (Beers *et al.*, 2006).

The pathogenesis of NAFLD has been linked to obesity, especially if the individual has central abdominal obesity (MacDonald, 2010).

Obesity increases the risk of developing NAFLD by a factor of 4.6 times compared to individuals of a normal BMI. Steatosis, or abnormal retention of lipids, is found in more than 66% of obese people and in more than 90% of morbidly obese people (Angulo, 2002).

If NAFLD advances it can cause liver cirrhosis and portal hypertension causing further health complications (Beers *et al.*, 2006).

2.6 Management

The overall goal of managing obesity is to reduce and maintain a lower body weight over a long term period and therefore prevent weight gain in the future. A healthy amount of weight to lose is estimated to be 0.5kg per week (Escott-Stump, 2011).

2.6.1 Diet

A recommended dietary approach to weigh loss includes several different aspects. A diet with moderate amounts of protein and carbohydrates is optimal, with six to eight small meals being scheduled at frequent intervals. Foods high in fibre content should also be consumed as they take longer to eat, increase satiety and are low in calories (Escott-Stump, 2011). Protein consumption has been shown to be very important as it may increase the metabolic rate by up to 30% and by stimulating pancreatic hormone secretion, help to balance serum glucose levels (Balch, 2006).

Daily calorie intake for women and inactive men should be approximately 2000 calories in order to maintain weight. For men and very active women 2500 calories is suggested to maintain a healthy weight. In order to lose weight in a healthy manner, it is suggested that 300 – 500 fewer calories be eaten daily (Balch, 2006).

In a study conducted in 2004, the effects of a low-carbohydrate diet compared to a low-fat diet on obesity and hyperlipidaemia showed that a low-carbohydrate diet allowed a greater loss of weight and also decreased triglyceride levels (Bakst *et al.,* 2004).

High protein consumption with low-carbohydrate diets have shown to be effective in causing weight loss. The reason for this is that an increase of protein intake of up to 30% has an anorexic effect on the body by increasing satiety, thereby decreasing caloric intake and aiding in weight loss (Breen *et al.,* 2005).

However, there are many different diets available for use by the public and some people may not benefit from certain diets whereas others may. It is therefore very important for an overweight or

obese individual to consult a dietician who can alter a diet for their specific needs in order to receive the beneficial effects (Escott-Stump, 2011).

2.6.2 Physical activity

Exercise, especially aerobic exercise has many positive effects on the body and many benefits which assist in weight loss. Physical activity increases the basal metabolic rate as well as thermogenesis and energy consumption (Beers *et al.*, 2006).

Approximately one third of the amount of energy expended per day is done so through muscular contraction. Exercise is therefore vital for energy expenditure and obesity is said to be caused from a high ratio of calorie intake compared to that of daily exercise (Guyton and Hall, 1997).

Exercising increases muscle mass and assists in weight loss. Muscle tissue also expends more energy at rest than adipose tissue. This is very important as this will raise the basal metabolic rate and keep it higher for a longer period of time, thereby assisting in prolonged maintenance of body weight (Beers *et al.*, 2006).

Exercises which may assist in weight loss include brisk walking or running, cycling and swimming. Other exercises that may help to increase strength and flexibility include weight lifting, yoga and stretching exercises. A combination of dieting and regular exercise has shown to be more effective than either of these factors alone (Balch, 2006).

It is important for an individual to maintain an interest in the physical activity that they pursue as this will promote long term compliance (Beers *et al.*, 2006).

2.6.3 Medication

Medication is usually administered when an individual's BMI is more than 30 kg/m^2 or if it is more than 27 kg/m^2 and there are other associated health complications (Beers *et al.*, 2006).

2.6.3.1 Allopathic medication

- **Sibutramine**

  Sibutramine is an appetite suppressant. It works by increasing the levels of norepinephrine, serotonin and dopamine in the brain and preventing their re-uptake. By doing this it stimulates parts of the brain associated with appetite control such as the hypothalamus, thereby reducing appetite (Chilnick *et al.,* 2008). Side effects include hypertension, headache, dry mouth, constipation and insomnia (Beers *et al.*, 2006); however, long term use for up to two years is considered to be safe (Champe and Harvey, 2009).

- **Orlistat**

  Orlistat works by inhibiting intestinal lipase secretion which decreases the hydrolysis of ingested fats into glycerol and fatty acids. This in turn causes a decreased absorption of fats into the cells of the body (Beers *et al.*, 2006). Approximately 30% of ingested fat is eliminated due to the function of Orlistat. Side effects include steatorrhoea (oily stools), flatus, increased urgency for stool, diarrhoea and headaches (Chilnick *et al.,* 2008). Albeit side effects, this drug has been shown to be safe to use for up to four years (Champe and Harvey, 2009).

2.6.3.2 Supplementation

- **L-carnitine**

  L- carnitine is a substance which helps to transport fatty acids to mitochondria in the cells of the body so that they can be used for energy. This process therefore increases the use of fat by the body, thereby reducing fatty build up in the body (Balch, 2006). There is no evidence to confirm the effectiveness of L-carnitine in weight loss however, some studies suggest that it can reduce fat and increase muscle mass as well as reduce fatigue (Ehrlich, 2009b).

- **Chromium**

  Chromium is used to stabilize blood glucose levels and is mainly used for people with diabetes or hypoglycaemia. It is used in the treatment of weight loss to stabilize blood glucose levels and reduce sugar cravings (Friedensohn *et al.,* 2004).

- **Lecithin**

  Lecithin is a type of lipid which is a constituent of many parts of the body such as cell membranes and myelin sheaths. Lecithin allows cholesterol and other lipids to be released from body stores and either used or eliminated from the body. This therefore aids in weight loss and decreases the risk of developing other metabolic illnesses (Balch, 2006).

2.6.4 Surgery

Long term treatment for morbid obesity is bariatric surgery, also known as gastric bypass surgery. The reason for this surgery is not only done in order to decrease the capacity the stomach can hold or to decrease the surface area for absorption, but it is also used to decrease the release of ghrelin which is an appetite stimulator released from the gastric fundus (Boon *et al.,* 2006).

Surgery is only considered in the morbidly obese when other modalities of treatment such as diet, exercise, behavioural therapy and medication are not successful or when the person has serious complications associated with obesity (Beers *et al.*, 2006).

Complications of bariatric surgery include anastomotic leak, thromboembolism, stricture formation, internal hernia, ulcer formation, cholelithiasis, haemorrhage, nutritional and metabolic derangements (Bloomberg and Herron, 2006).

2.7 Homoeopathy

2.7.1 Homoeopathic scope of practice in South Africa

According to the Allied Health Professions Act 63, a homoeopathic practitioner may conduct an investigation and physical examination, whereby physical symptoms and their modalities are investigated and taken into account, in order for the practitioner to diagnose an illness, defect or deficiency and treat the illness, defect or deficiency, according to homeopathic principles with the use of remedies, dietary advice and supplementation (1982).

2.7.2 Homoeopathic treatment of obesity

In homoeopathy there exists the belief that all levels of the body; mental, emotional and physical are inter-related and that an imbalance in one level may cause an imbalance in another. This is the holistic view of homoeopathy (Bhatia, 2007). The vital force is described as an innate inner force or energy of the body, which when diseased or out of balance, causes the physical, mental or emotional manifestation of symptoms (Boericke, 1997). Therefore homeopaths will treat a patient according to the totality of their mental, emotional and physical symptoms and attempt to bring the vital force back into balance or homeostasis (Bhatia, 2007).

The vital force works by bringing back the balance in an individual's body by regulating internal bodily functions (Griffith, 2010). In homoeopathy, obesity will be treated by taking into account all three levels, mental, emotional and physical. By doing this, the individual is treated holistically and obesity is not targeted alone, but the underlying cause of the obesity will be addressed and treated, restoring normal body regulation and overall well being to the individual (Jain, 2006).

2.8 Herbal medication

Herbal products are fast becoming very popular, with approximately 80% of people worldwide using them today (Ehrlich, 2009a). Some commonly used herbal weight loss products currently available over the counter in South Africa include Leanor Herbal Slimming Concentrate™,

Thinz Diet Pills™, Herbex Herbal Diet Drops™, G.I Lean Fat Burn Herbal Drops™, CLA Hoodia™ and Phenadrine Xtra™.

Herbal products are shown to have fewer, albeit possible, side effects compared to most other medications (Ehrlich, 2009a). Even though herbal medications are considered safer by many people, scientific studies have not been conducted over long enough periods of time in order to determine long term side effects (Mattsson and Nilsson, 2002). Side effects of herbal medications may include allergic reactions, gastrointestinal upsets, insomnia and fatigue. Herbal medications should therefore be used with caution and under medical supervision (Ehrlich, 2009a).

Homoeopaths use herbal medications to re-establish equilibrium in the body when there is a physiological imbalance. Herbal tinctures are used to support specific organ systems, acting as a supportive tonic for an organ or body system (Kramer, 2010).

2.8.1 Herbal formulation and toxicity

The herbal formulation in this study contains the following ingredients: Caffeine (100mg), *Camellia sinensis* (200mg), *Ilex paraguariensis* (240mg), *Phaseolus vulgaris* (60mg), *Coleus forskohlii* (125mg), *Coffea canephora* bean (100mg) and Evodiamine (26mg) (Appendix G). The dosages of all active ingredients included in the herbal formulation used in this study are below accepted levels of toxicity and are safe for short term usage. There are no anticipated side effects at the administered dosage (Baillie, 2011a).

Caffeine, *Camellia sinensis* and *Coffea canephora* bean are safe to use orally if 400mg or less of each is consumed per day. An excess of this dosage may cause symptoms such as heart palpitations and changes in behaviour such as anxiety (Eastwood *et al.,* 2003). Over a twelve week period, the use of 500mg of *Coleus forskohlii* per day showed no clinically significant side effects (Henderson, 2005). Evodiamine has been shown to be safe in herbal form when used in an eight week trial (Kim *et al.,* 2008). *Ilex paraguariensis* was used over a twelve month period and was shown to be safe if less than 1000mg are consumed per day (Andersen and Fogh, 2001). *Phaseolus vulgaris* has been shown to be safe to use over short periods of two to three months, provided the dosage does not exceed 3000mg per day (Birketvedt *et al.,* 2002).

2.8.1.1 Caffeine

Caffeine is able to decrease body weight and adipose tissue (Blanchard *et al.,* 2002). Caffeine affects the nervous system by blocking adenosine, which causes an overall increase in metabolic activity, thereby assisting in weight loss (Buttars, 2011).

Caffeine has been shown to be both effective and safe to use orally over a twenty week period for the treatment of obesity, with negligible side effects when compared to a placebo group (Erhardt *et al*., 2000).

2.8.1.2 Coffea canephora bean

Coffea canephora bean or green coffee bean extract contains the active ingredients caffeine and chlorogenic acid, which inhibit fat absorption and reduce the production of hepatic triglycerides by increasing fat metabolism by the liver (Aitani *et al.*, 2006).

Chlorogenic acid was found to be essential in the inhibition of gluconeogenesis, as well as the absorption of glucose from the gut, resulting in an overall weight loss effect (Thom, 2007).

Coffea canephora bean was also effective in preventing weight gain and fat accumulation when administered to mice (Aitani *et al.,* 2006).

2.8.1.3 Coleus forskohlii

Coleus forskohlii contains an active ingredient known as forskolin; a product which has been directly linked with cyclic-AMP (c-AMP), a chemical messenger within the human body that acts within the cells to assist in increasing the metabolic rate (Clear Springs Press, 2009).

Coleus forskohlii has also shown to be able to cause the breakdown of stored fat cells. In a study done in 2005 it was concluded that it may diminish weight gain in overweight females (Henderson, 2005). It was also shown to decrease body fat percentage and significantly increase lean muscle mass, bone density and testosterone levels in obese men (Godard *et al.,* 2005).

2.8.1.4 Camellia sinensis

Camellia sinensis or green tea extract decreases BMI, body fat ratio, body fat mass, waist circumference and subcutaneous fat area (Hase *et al.,* 2007).

It contains active polyphenol substances called catechins. These catechins, together with caffeine in the green tea extract, have had marked results in weight loss. The weight loss effect imposed by *Camellia sinensis* is achieved by thermogenesis which effectively increases lipid oxidation, promoting weight loss (Chantre *et al.,* 1999).

A study concluded that the ingestion of an extract such as *Camellia sinensis* may contribute to a decrease in obesity and cardiovascular disease (Hase *et al.,* 2007).

2.8.1.5 Evodiamine

Evodiamine has the potential to prevent diet-induced obesity and decrease adipose deposition (Wang and Wang, 2008).

Evodiamine is the alkaloid in Evodia fruits of the plant *Evodia rutaecarpa* (Hoshikuma *et al.,* 2001). Evodiamine increases the release of catecholamines thereby suppressing the appetite, increasing energy levels and allowing greater caloric breakdown, thus assisting in weight loss (Wang and Wang, 2008).

It is an alkaloid which has cholesterol lowering and thermogenic effects, increasing the production of body heat by inducing a greater rate of lipolysis (Hoshikuma *et al.,* 2001).

2.8.1.6 Ilex paraguariensis

Ilex paraguariensis is able to effectively decrease the level of triglycerides, low density lipoproteins and weight in mice (Bastos *et al.,* 2009).

A study conducted on various herbs used for weight loss in Brazil and Porto Alegre showed that of approximately 23 species of plants tested, *Ilex paraguarensis* was the only herb which showed conclusive evidence of its ability to effectively contribute to the treatment of obesity (Dickel *et al.,* 2006).

It increases the amount of fat which is excreted via faeces, thereby improving the overall lipid profile and decreasing lipids in the body (Birketvedt *et al.,* 2002).

2.8.1.7 Phaseolus vulgaris

Phaseolus vulgaris has an inhibitory effect on the activity of the digestive enzyme alpha amylase, reducing the absorption of carbohydrates and their conversion into fat, thereby assisting in weight loss (Hardy *et al.,* 2004).

It has been shown to significantly reduce body weight, BMI and weight circumference in overweight men and women over a sixty day study, while also showing a long term weight loss effect (Preuss *et al,* 2010).

CHAPTER 3

METHODOLOGY

3.1 Research sample

Sixty participants were recruited from the University of Johannesburg, as well as from the public sector, in response to posters that were posted at the university and given to other Homoeopathic practitioners. Posters were also posted in local gymnasiums and fitness clubs with granted permission (Appendix A).

The participants were placed in either a placebo group or an experimental group. The placebo group included thirty participants and the experimental group included thirty participants. The selection of these two groups was done according to matched pairing of gender and BMI. This grouping was done by an independent person and was not influenced by the researcher. Participants were required to maintain their normal lifestyle, with no major changes in stress levels, eating and exercise habits for the duration of the study.

3.1.1 Inclusion criteria

The participants were males and females between the ages of 18 and 45 years with a BMI between 25 and 35kg/m^2.

3.1.2 Exclusion criteria

Individuals with any chronic illnesses, who were on any chronic allopathic, herbal or homoeopathic medication; who were on a weight loss programme; who had known allergies or sensitivities to any substances in the herbal formulation; or were pregnant or breastfeeding were excluded from the research study.

3.2 Research design and procedure

This was a twelve week, double-blind, placebo-controlled study. The prospective participants were asked to sign a Participant Information and Consent Form which informed them of the procedures, risks and benefits of the study (Appendix B). All of the consultations took place at the University of Johannesburg Health Centre at the Doornfontein campus. This research study, assessing BMI and abdominal girth, was conducted in conjunction with two other researchers. This allowed for a larger sample group to be tested with numerous variables being researched. One researcher measured the resting metabolic rate and body composition of the participants over the twelve week period (Withers *et al*., 2012), and the other evaluated their general well being and the possible side effects of the medication over this time (Lord *et al*., 2012).

At the initial consultation, a detailed case history and the vital signs (including blood pressure, respiratory rate, heart rate and temperature) of the participant was taken. Their height was measured and their weight determined on a scale calibrated to 0.1kg. From these measurements, their BMI was calculated using the standardised formula (Appendix D). They were then categorized into the overweight or obese categories, depending on the result of the calculation (Appendix E). Their abdominal girth was measured three times during each consultation, each time using a standardized method (Appendix F), and the average measurement was obtained. This data was recorded on a consultation form (Appendix C). At the end of the initial consultation participants were administered their medication, which they were asked to take from Monday to Friday, over the twelve week period, with a rest period for two days over the weekend.

The participants then returned for follow-up evaluations in the second, sixth and twelfth week of the study. At each follow-up consultation, the participants' vital signs and abdominal girth measurements were taken again and their BMI calculated by measuring their weight.

3.3 Medication administration

Each dose of the herbal formulation (2 capsules per day) is 851mg. Together these capsules are composed of Caffeine (100mg), *Camellia sinensis* (200mg), *Ilex paraguariensis* (240mg), *Phaseolus vulgaris* (60mg), *Coleus forskohlii* (125mg), *Coffea canephora* bean (100mg) and Evodiamine (26mg) (Appendix G).

Each dose of the placebo (2 capsules per day) was 600mg. It was composed of pharmaceutical starch enclosed in silica capsules. These capsules were identical to the capsules which contain the herbal formulation so that it allowed for a double blind study.

Each participant was asked to take two capsules of their medication per day, in the morning, on an empty stomach, from Monday to Friday, whether they were in the placebo or the experimental group. A two day period of no medication administration took place on Saturdays and Sundays to avoid the development of a tolerance to the herbal formulation. If any adverse effects such as nausea were experienced, the participants were requested to take their medication with their meals (Baillie, 2011a).

3.4 Reliability and validity measures

The placebo and the herbal formulation were not distinguishable from one another allowing the medication administration to remain anonymous to both the participants and the researcher. The placebo and the herbal formulation were supplied by Lunar Pharmaceuticals which ensured that

the composition and the quality of both of these products remained the same. The placebo was composed of pharmaceutical starch and has no known effects on weight gain or loss, in the dosages used in this study (Baillie, 2011a).

The weight scale on which the participants were weighed each time was the same scale, allowing for no variability in the calibration, and therefore measurement ability, of the scale. The abdominal girth measurements were taken three times over the same landmarks at each consultation. These measurements were then added together and the average circumference was established which allowed for the most accurate measurement possible.

The most commonly used and accepted method for diagnosing overweight and obese individuals is by the measurement of BMI and abdominal girth, an elevation of these two factors is enough evidence to lead to the diagnosis (Allison *et al.,* 2008).

3.5 Data collection and analysis

All the data was coalesced, analysed and compared in order to provide the overall results of the study. A number of tests were used to analyse the data. The Shapiro Wilk test tested the normality of the results. For inter-group comparisons of the results, between the placebo and experimental groups, the Mann-Whitney test was used. For comparisons within the placebo and the experimental groups on the measurements of the BMI and the abdominal girth, taken during the four consultations, the Friedman test and the Wilcoxon signed ranks test were used (Smith, 2011).

3.6 Ethics

All participants involved in the study were participating under voluntary informed consent and were free to withdraw from the research study at any point in time, if they wished to do so. The procedures that took place during the study were explained in detail to the participants and they were informed of their rights. Total anonymity of the participants was ensured throughout the duration of this study as well as in the results. The names of the participants were not disclosed to anyone other than the researchers during the study or in the results of the study. All information supplied by the participants was kept in a secure location at the University of Johannesburg, only being accessible to the researchers, thereby remaining confidential.

The dosage of all active ingredients included in the herbal formulation are all below accepted individual levels of toxicity and have also been shown to be safe when used together over a 4 week period (Baillie, 2011a). There were no anticipated side-effects at the administered dosage and therefore there were no anticipated risks involved. If it became necessary for any participants to take medication during the course of the study, their results were excluded. If any participants

experienced negative side effects during the course of the study, they were advised to contact the researcher and would have been taken off the medication and referred to their relevant healthcare provider if necessary. After the study had been concluded the results were made available to the participants on request.

CHAPTER 4

RESULTS

4.1 Introduction to results

This chapter presents all of the results obtained during the twelve week study on the effect of a herbal formulation consisting of Caffeine, *Coffea canephora* bean, *Coleus forskholii, Camellia sinensis,* Evodiamine, *Ilex paraguariensis* and *Phaseolus vulgaris* on Body Mass Index and abdominal girth measurements in overweight and obese individuals.

All the data obtained during the study were recorded on the consultation form (Appendix C). This information was then analysed using several statistical methods in order to determine the differences between the placebo and the treatment groups.

The statistical techniques used to compare the results from the two groups included the Shapiro-Wilk test, Friedman Analysis of Variance test, Mann-Whitney U test and Wilcoxon Signed-Rank test (Smith, 2011).

The hypothesis of the study was that the herbal formulation consisting of Caffeine, *Coffea canephora* bean, *Coleus forskholii, Camellia sinensis,* Evodiamine, *Ilex paraguariensis* and *Phaseolus vulgaris* will cause a decrease in weight and therefore in BMI as well as reduce abdominal girth measurements compared to a placebo. According to the results, this hypothesis was rejected. The null hypothesis which anticipated that the herbal formulation consisting of Caffeine, *Coffea canephora* bean, *Coleus forskholii, Camellia sinensis,* Evodiamine, *Ilex paraguariensis* and *Phaseolus vulgaris* will not cause a decrease in weight and BMI nor will it reduce abdominal girth measurements compared to a placebo was accepted in this study.

4.2 Initial description

The following abbreviations are used in the description of the results of the study (Smith, 2011):

- N - represents the number of participants within each group of the study.
- Mean - is the average result for a specific measurement.
- Median - is the number that occurs directly in the middle of a series of values.
- Mode - is the number which occurs most often in a series of values.
- Standard deviation – is the measurement of how much a value differs from the mean of the result.

- P-value – indicates the statistical significance of a value representative of the population. If the value is less than 0.05 then there are significant differences between the treatment and placebo groups or within each group. If the value is more than 0.05 then there are not significant differences between the treatment and placebo groups or within each group.

4.2.1 Group frequency

Sixty participants were recruited and enrolled to take part in the study; thirty were placed into the placebo group and thirty were placed into the treatment group. Groups were selected according to matched pairs of BMI and gender as stipulated in 3.1. One person in the placebo group did not complete the study and two people in the treatment group did not complete the study. Their results were excluded from the results for statistical purposes.

As shown in Table 4.1, there were a total of 57 participants who completed the twelve week study, 29 in the placebo group and 28 in the treatment group.

Table 4.1 Group frequency and percentage

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Placebo	29	50.9	50.9	50.9
	Treatment	28	49.1	49.1	100.0
	Total	57	100.0	100.0	

4.2.2 Gender frequency

Of all 57 participants who completed the twelve week study, 26 were male and 31 were female as shown in Table 4.2.

Table 4.2 Gender frequency and percentage

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Male	26	45.6	45.6	45.6
	Female	31	54.4	54.4	100.0
	Total	57	100.0	100.0	

Table 4.3 illustrates that within the placebo group there were 12 male participants and 17 female participants. In the treatment group there were 14 male and 14 female participants.

Table 4.3 Gender per group

			Gender		
			Male	Female	Total
Group	Placebo	Count	12	17	29
		% within Group	41.4%	58.6%	100.0%
	Treatment	Count	14	14	28
		% within Group	50.0%	50.0%	100.0%
Total		Count	26	31	57
		% within Group	45.6%	54.4%	100.0%

4.2.3 Age frequency

All participants selected were between the ages of 18 and 45 as stated in 3.1.1. The mean age for the participants was 28.75 years with a median age of 25 years. The standard deviation from the mean was 7.259 years, as represented in Table 4.4. The most common age of the participants as represented by the mode, was 24 years of age.

Table 4.4 Age statistics

	N							
	Valid	Missing	Mean	Median	Mode	Std. Deviation	Minimum	Maximum
Age	57	0	28.75	25.00	24.00	7.259	18.00	45.00

Table 4.5 shows a p-value (Sig) of less than 0.05 for the placebo and the treatment groups, showing that the age distribution was not normal and that there was a large variation in age within each group. Table 4.5 also represents the number of people (df) within each group.

Table 4.5 Age normality statistics

		Shapiro-Wilk		
Group		Statistic	df	Sig.
Age	Placebo	.910	29	.017
	Treatment	.876	28	.003

4.2.4 Height frequency

The average height (mean) of each individual in the study was 1.70m. Table 4.6 shows the median height as well as the standard deviation from the mean. The median height was 1.68m and the most common height (mode) was 1.65m.

Table 4.6 Height statistics

	N		Mean	Median	Mode	Std. Deviation	Minimum	Maximum
	Valid	Missing						
Height	57	0	1.70	1.68	1.65	.096	1.53	1.98

Table 4.7 shows that there was a normal distribution of height in the placebo and the treatment groups because the p-value (Sig) is more than 0.05.

Table 4.7 Height normality statistics

		Shapiro-Wilk		
Group		Statistic	df	Sig.
Height	Placebo	.933	29	.067
	Treatment	.948	28	.172

4.2.5 Weight

Table 4.8 represents the distribution of weight within the placebo and the treatment groups over the twelve week study period. There was a normal distribution of weight within the placebo and the treatment groups throughout the twelve week period as the p-value (Sig.) was more than 0.05 (Refer to Appendix H).

Table 4.8 Weight distribution

		Shapiro-Wilk		
Group		Statistic	df	Sig.
Weight_w0	Placebo	.959	29	.314
	Treatment	.962	28	.382
Weight_w2	Placebo	.961	29	.340
	Treatment	.965	28	.444
Weight_w6	Placebo	.965	29	.440
	Treatment	.961	28	.378
Weight_w12	Placebo	.953	29	.224
	Treatment	.950	28	.194

Table 4.9 shows the mean weight of participants in the treatment group (week 0 mean weight = 82.97 kg) and the placebo group (week 0 mean weight =81.28 kg). It also shows, by inter-group analysis, that there was no significant difference in weight between the placebo and treatment groups during the twelve week study, as the p-value (Asymp. Sig. (2-tailed)) is more than 0.05 (week 0 = 0.672; week 12 = 0.829) (Refer to Appendix I).

Table 4.9 Weight inter-group comparison

Group		N	Mean	Std. Deviation	Std. Error Mean	Mean Rank	Sum of Ranks	Mann-Whitney U	Z	Asymp. Sig. (2-tailed)
Weight_w0	Placebo	29	81.28	13.610	2.527	28.09	814.50	379.500	-.423	.672
	Treatment	28	82.97	12.201	2.306	29.95	838.50			
Weight_w2	Placebo	29	81.11	13.729	2.549	27.93	810.00	375.000	-.495	.621
	Treatment	28	82.69	12.374	2.339	30.11	843.00			
Weight_w6	Placebo	29	81.67	13.635	2.532	28.53	827.50	392.500	-.215	.829
	Treatment	28	82.78	12.460	2.355	29.48	825.50			
Weight_w12	Placebo	29	82.36	14.220	2.641	28.53	827.50	392.500	-.216	.829
	Treatment	28	83.61	12.592	2.380	29.48	825.50			

Figure 4.1 shows the average (mean) weight for each group over the twelve week period of the study. The treatment group (week 12 mean weight = 83.61 kg) and the placebo group (week 12 mean weight = 82.36 kg) had a net gain in weight of 0.64 kg and 1.08 kg respectively during the study.

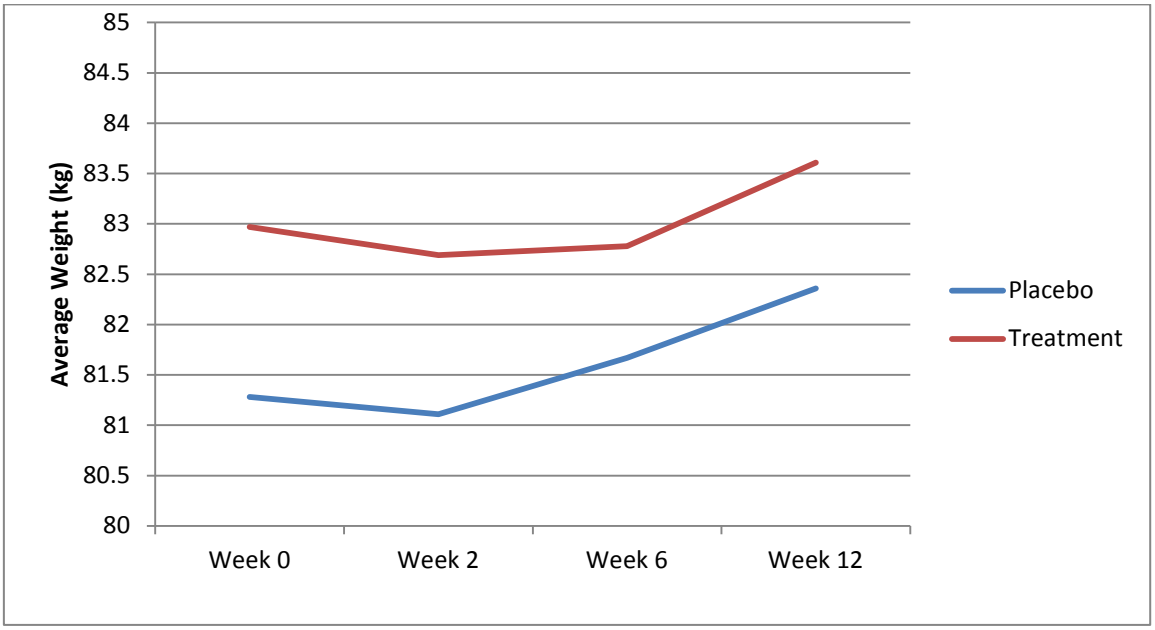

Figure 4.1: Average weight over the 12 week study

Table 4.10 represents data gathered for weight change within each of the groups. Both of the groups showed a change in weight over the twelve weeks. The p-value (Asymp. Sig) was 0.31 for the placebo group and 0.003 for the treatment group, which is less than 0.05 (Refer to Appendices J and K).

Table 4.10 Weight intra-group analysis

Placebo	N	29
	Chi-Square	8.880
	df	3
	Asymp. Sig.	.031
Treatment	N	28
	Chi-Square	13.787
	df	3
	Asymp. Sig.	.003

Table 4.11 represents the changes in weight in the placebo and treatment groups at specific points in time. Neither the placebo nor the treatment groups show statistically significant changes over the twelve week period using the more specific Wilcoxon signed ranks test which has a stricter p-value. The treatment group showed a p-value (Asymp. Sig) of more than 0.05 (week 0 to week 2 = 0.148; week 0 to week 6 = 0.318 and week 0 to week 12 = 0.85) and the placebo group also showed a p-value (Asymp. Sig) of more than 0.05 (week 0 to week 2 = 0.368; week 0 to week 6 = 0.183 and week 0 to week 12 = 0.67) (Refer to Appendices L and M).

Table 4.11 Weight change over time

	Group			
	Placebo		Treatment	
	Z	Asymp. Sig. (2-tailed)	Z	Asymp. Sig. (2-tailed)
Weight_w2 - Weight_w0	-.900	.368	-1.447	.148
Weight_w6 - Weight_w0	-1.333	.183	-.999	.318
Weight_w12 - Weight_w0	-1.834	.067	-1.720	.085

4.2.6 BMI

BMI was not normally distributed within the placebo or the treatment groups during the twelve week study. This is shown in Table 4.12 where the p-value (Sig.) is less than 0.05 throughout the study in both groups (Refer to Appendix H).

Table 4.12 BMI distribution

		Shapiro-Wilk		
Group		Statistic	df	Sig.
BMI_w0	Placebo	.889	29	.005
	Treatment	.840	28	.001
BMI_w2	Placebo	.919	29	.028
	Treatment	.865	28	.002
BMI_w6	Placebo	.909	29	.016
	Treatment	.885	28	.005
BMI_w12	Placebo	.896	29	.008
	Treatment	.927	28	.051

Table 4.13 shows that there was no significant change in BMI between the treatment (week 0 = 28.14 kg/m^2; week 12 = 28.31 kg/m^2) and placebo (week 0 = 28.77 kg/m^2; week 12 = 29.16 kg/m^2) groups over the twelve week period. The p-values (Asymp. Sig. (2-tailed)) were more than 0.05 (week 0 =0.502; week 12 = 0.439) over the twelve week period (Refer to Appendix I).

Table 4.13 BMI inter-group comparison

Group		N	Mean	Std. Deviation	Std. Error Mean	Mean Rank	Sum of Ranks	Mann-Whitney U	Z	Asymp. Sig. (2-tailed)
BMI_w0	Placebo	29	28.77	3.188	.592	30.45	883.00	364.000	-.671	.502
	Treatment	28	28.14	3.247	.614	27.50	770.00			
BMI_w2	Placebo	29	28.72	3.292	.611	30.55	886.00	361.000	-.719	.472
	Treatment	28	28.03	3.221	.609	27.39	767.00			
BMI_w6	Placebo	29	28.93	3.211	.596	31.45	912.00	335.000	-1.134	.257
	Treatment	28	28.08	3.414	.645	26.46	741.00			
BMI_w12	Placebo	29	29.16	3.407	.633	30.67	889.50	357.500	-.774	.439
	Treatment	28	28.31	3.397	.642	27.27	763.50			

Figure 4.2 shows the average (mean) BMI for each group over the twelve week period of the study. The treatment (week 12 = 28.31kg/m^2) and the placebo (week 12 = 29.16 kg/m^2) groups showed a net gain in BMI of 0.17 kg/m^2 and 0.39 kg/m^2 respectively.

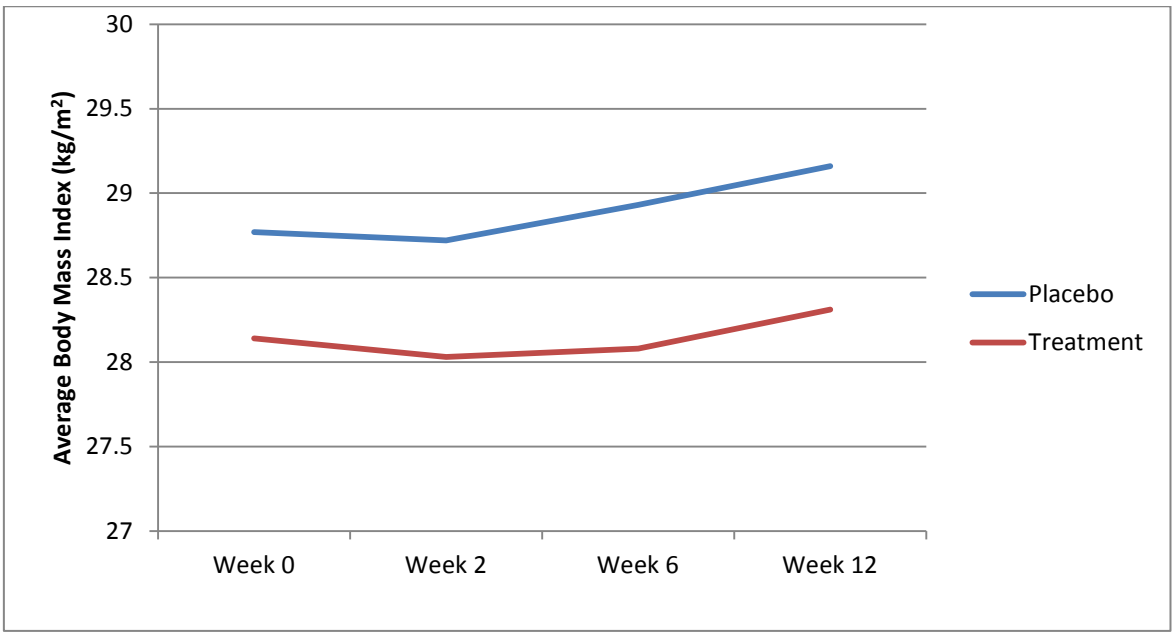

Figure 4.2: Average Body Mass Index over the 12 week study

Table 4.14 represents the changes in BMI within the placebo group and the treatment group. The data shows a significant change within the treatment group (p = 0.019) and the placebo group (p = 0.014) as the p-value (Asymp. Sig) of both groups is less than 0.05 (Refer to Appendices J and K).

Table 4.14 BMI intra-group analysis

Placebo	N	29
	Chi-Square	10.588
	df	3
	Asymp. Sig.	.014
Treatment	N	28
	Chi-Square	10.004
	df	3
	Asymp. Sig.	.019

BMI changes at specific intervals over the twelve week study are shown in Table 4.15. It shows that the p-value (Asymp. Sig. (2-tailed)) of the placebo group (week 0 to week 2 = 0.523; week 0 to week 6 = 0.120) was more than 0.05 at certain intervals. However, during week 0 to 12, the p-value (Asymp. Sig. (2-tailed)) of the placebo group was 0.040, which shows a statistically significant change in BMI in this group. There were no statistically significant changes shown in the average BMI of the treatment group during the study.

The treatment group showed no statistical change during the study (week 0 to week 2 = 0.214; week 0 to week 6 = 0.477 and week 0 to week 12 = 0.127) as the p-value (Asymp. Sig. (2-tailed)) was more than 0,05 (Refer to Appendices L and M).

Table 4.15 BMI change over time

	Group			
	Placebo		Treatment	
	Z	Asymp. Sig. (2-tailed)	Z	Asymp. Sig. (2-tailed)
BMI_w2 - BMI_w0	-.639	.523	-1.243	.214
BMI_w6 - BMI_w0	-1.556	.120	-.710	.477
BMI_w12 - BMI_w0	-2.051	.040	-1.528	.127

4.2.7 Abdominal girth

Table 4.16 shows that there was a normal distribution of abdominal girth within the placebo and the treatment groups throughout the twelve week study, as the p-value (Sig.) is more than 0.05 (Refer to Appendix H).

Table 4.16 Abdominal girth distribution

		Shapiro-Wilk		
Group		Statistic	df	Sig.
AG_w0	Placebo	.970	29	.559
	Treatment	.942	28	.127
AG_w2	Placebo	.982	29	.892
	Treatment	.947	28	.166
AG_w6	Placebo	.977	29	.766
	Treatment	.931	28	.067
AG_w12	Placebo	.969	29	.534
	Treatment	.929	28	.059

The mean abdominal girth, standard deviation from the abdominal girth and the p-value are shown in Table 4.17. There were no significant changes in abdominal girth between the placebo (week 0 = 91.04 cm; week 12 = 91.74cm) and the treatment group (week 0 = 92.36 cm; week 12 = 91.36cm) throughout the twelve week study, as the p-value (Asymp. Sig. (2-tailed)) was more than 0.05 (Refer to Appendix I).

Table 4.17 Abdominal girth inter-group comparison

Group		N	Mean	Std. Deviation	Std. Error Mean	Mean Rank	Sum of Ranks	Mann-Whitney U	Z	Asymp. Sig. (2-tailed)
AG_w0	Placebo	29	91.04	9.307	1.728	27.67	802.50	367.500	-.615	.539
	Treatment	28	92.36	8.911	1.684	30.38	850.50			
AG_w2	Placebo	29	91.57	9.546	1.773	28.78	834.50	399.500	-.104	.917
	Treatment	28	92.10	9.002	1.701	29.23	818.50			
AG_w6	Placebo	29	90.92	9.998	1.857	29.12	844.50	402.500	-.056	.955
	Treatment	28	90.74	8.432	1.594	28.88	808.50			
AG_w12	Placebo	29	91.74	9.298	1.727	29.64	859.50	387.500	-.295	.768
	Treatment	28	91.36	8.859	1.674	28.34	793.50			

Figure 4.3 represents the average (mean) abdominal girth for the placebo group (week 12 = 91.74 cm) and the treatment group (week 12 = 91.36 cm) over the twelve week period of the study. The treatment group showed a net loss in abdominal girth measurements of 1cm, whereas the placebo group showed a net gain of 0.7cm in abdominal girth over the twelve week study.

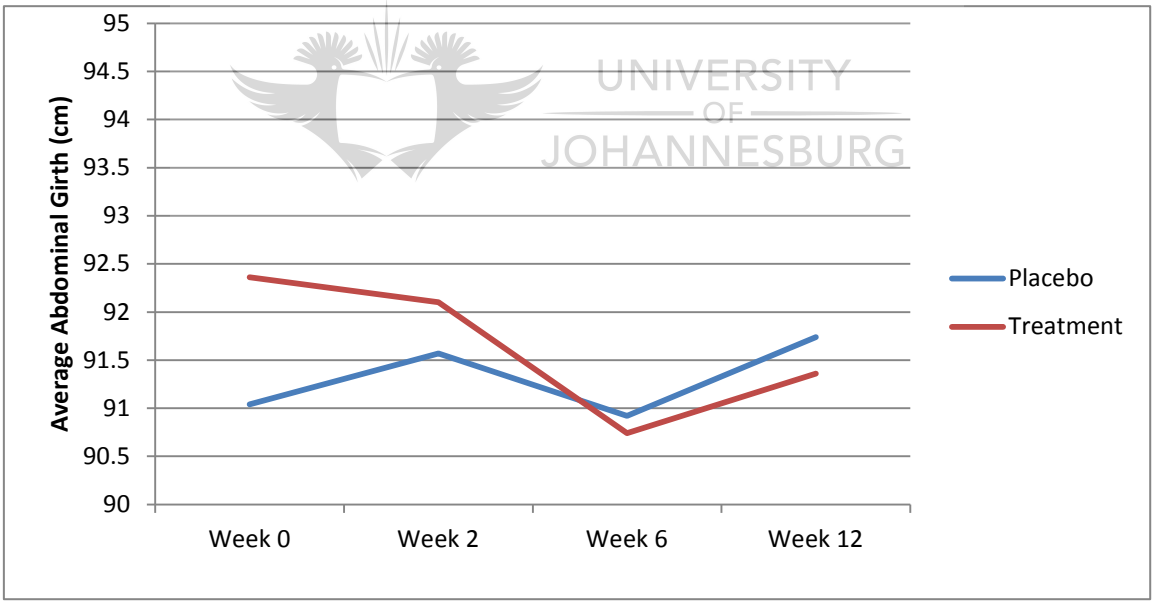

Figure 4.3: Average abdominal girth over the 12 week study

Table 4.18 represents the changes in abdominal girth within each of the groups. The p-value (Asymp. Sig) in the placebo group was 0.075, showing that there was no statistically significant change in abdominal girth. The treatment group showed a p-value (Asymp. Sig) of 0.002, illustrating a statistically significant change in abdominal girth measurements within the group (Refer to Appendices J and K).

Table 4.18 Abdominal girth intra-group analysis

Placebo	N	29
	Chi-Square	6.939
	df	3
	Asymp. Sig.	.074
Treatment	N	28
	Chi-Square	15.121
	df	3
	Asymp. Sig.	.002

Abdominal girth measurement changes at specific points in time are recorded in Table 4.19. There were no changes in abdominal girth in the placebo group over the twelve week study and therefore the Wilcoxon Signed ranks test showed no data, as represented in Table 4.19. The treatment group showed a statistically significant change in abdominal girth between week 0 and week 6 (week 0 to week 2= 0.431; week 0 to week 6 = 0.000; week 0 to week 12 = 0.78) as the p-value (Asymp. Sig. (2-tailed)) is less than 0.05 for this period of time (Refer to Appendices L and M).

Table 4.19 Abdominal girth change over time

	Group			
	Placebo		Treatment	
	Z	Asymp. Sig. (2-tailed)	Z	Asymp. Sig. (2-tailed)
AG_w2 - AG_w0			-.788	.431
AG_w6 - AG_w0			-3.509	.000
AG_w12 - AG_w0			-1.765	.078

CHAPTER 5

DISCUSSION OF RESULTS

5.1 Group frequency

Sixty participants were enrolled and recruited for the study and randomly placed into two groups. Thirty participants were in the placebo group and thirty were in the treatment group. However, three of them did not participate for the full duration of the study and their results were excluded for statistical purposes. One of the participants could not attend the follow up appointments as she works for the University of Johannesburg but was relocated to another campus. The other two participants could not attend the follow up appointments due to other work commitments which they were engaged in. As a result, 29 (50.9%) participants were in the placebo group and 28 (49.1%) were in the treatment group.

5.2 Gender frequency

Both male and female participants were included in the study as represented in 3.1.1. Of the 57 total participants, 26 were male and 31 were female. The placebo group had 12 male and 17 female participants and the treatment group had 14 males and 14 females. The percentage of male to female participants was 45.6% to 54.4% respectively but the difference between the two was not anticipated to affect the results.

5.3 Age frequency

Participants between the ages of 18 and 45 were included in the study. The results show that there was not a normal distribution of the age of the participants as the placebo group and treatment group showed a p-value (Sig.) of 0.017 and 0.003 respectively. The mean age for all the participants was 28.75 years. It is estimated that the average BMI for men and women between the ages of 15 and 29 years is 22.95kg/m^2, which is considered a normal weight. The estimated BMI for individuals between the age of 30 and 44 years is 26.35 kg/m^2, which is considered to be overweight (Goedecke *et al.,* 2005) The mean age of 28.75 years, seen in this study, is therefore significant as the age groups of 29 years and below moving into 30 years and over, are shown to move from a normal body weight to being overweight. Therefore the mean age in this study is near this transitional phase of moving from normal weight to overweight, which is relevant for use in a weight loss study. Because the age of the participants was not

normally distributed amongst the participants, we were able to see the effects that the herbal formulation had on people of different age groups.

5.4 Height frequency

There was no height criteria needed for the selection of participants in the study, however, Table 4.7 shows that there was a normal distribution of height amongst all of the participants.

5.5 Weight statistics

Participants were selected according to their BMI value. If it was determined that they were severely obese, they were excluded from the study. The placebo group had a mean weight of 81.28 kg at the beginning of the study while the treatment group had a mean weight of 82.97 kg.

The results show that the placebo group had an end mean weight measurement of 82.36 kg and the treatment group had an end mean weight measurement of 83.61 kg. This shows that the placebo group had a mean weight gain of 1.08 kg and the treatment group had a mean weight gain of 0.64 kg over the twelve week study.

The results do show that there was a statistically significant change within both the treatment and the placebo groups using the Friedman analysis of variance test. The placebo group and the treatment group showed a p-value (Asymp. Sig) of 0.031 and 0.003 respectively, as shown in Table 4.10. The results therefore indicate that there was a statistically significant increase in weight as the placebo group and the treatment group gained 1.08kg and 0.64kg respectively.

This may be due to the fact that the participants were not placed on a regular eating plan or exercise regime, and were told to maintain their normal lifestyle. Diet plays a very important role in weight loss as body weight is determined by the amount of energy ingested and the amount of energy expended. An imbalance in this ratio may cause weight gain, if energy intake exceeds expenditure (Dokken and Tsao, 2007). Portion sizes and the frequency of meals also play an integral part in weight gain as large portion sizes mean that large amounts of energy are being taken in at one sitting, and if not used effectively, will result in weight gain (Hensrud, 2004). The composition of a diet may also affect weight gain or loss. High carbohydrate diets as well as high fat diets have been shown to have a great impact on weight, causing weight gain (Goedecke, *te al.,* 2005). An eating plan low in calories, saturated fat and refined carbohydrate with high amounts of fruits, vegetables and whole grains is more suitable for weight loss (Hensrud, 2004).

The participants in this study were not given a diet or eating guidelines, meaning that they were not prevented from eating high amounts of saturated fats, refined carbohydrates and therefore having high caloric intakes. In this way the lack of an eating plan may have had an effect on the results of the study.

Exercise is another important factor contributing to weight loss. Up to one third of an individuals energy intake per day is used by muscular activity. Therefore, a lack of exercise will cause an excess accumulation of unused energy in the form of fat deposition (Guyton and Hall, 1997). The overall lack of an exercise regime may have therefore affected the weight loss results of the study.

Another possible reason as to why some of the participants did not lose weight compared to others, is that the participants were not treated holistically and they were only treated on the basis of them being overweight or obese. In homoeopathy, obesity will be treated by taking into account, all three levels of the body; mental, emotional and physical. By doing this, the individual is treated holistically and obesity is not targeted alone but the underlying cause of the obesity, being physical or emotional, is addressed and treated, restoring normal body regulation and overall well being to the individual (Jain, 2006).

The Wilcoxon Signed Ranks test however, shows that there was no statistically significant change of weight at any point in time within each of the groups. The values in the placebo group (week 0 to week 2= 0.368; week 0 to week 6= 0.183; week 0 to week 12 = 0.67) were all more than 0.05, showing no statistical significant change. In the treatment group, the values (week 0 to week 2= 0.148; week 0 to week 6= 0.318; week 0 to week 12 = 0.085) were all more than 0.05, indicating that there was no statistically significant change in the results. The reason for this is because the Wilcoxon Signed Ranks test has a stricter p-value, leaving less than a 5% chance of false results, while other tests may wrongfully reject the null hypothesis. Another reason may be that there were only sixty participants who took part in the study, which is classified as a small sample size (Smith, 2011).

The overall results showed that there were no significant differences in weight between the placebo and the treatment groups although the treatment group did not gain as much weight as the placebo group. This suggests that the herbal formulation containing Caffeine*, Coffea canephora* bean*, Coleus forskholii, Camellia sinensis,* Evodiamine*, Ilex paraguariensis* and *Phaseolus vulgaris* may be able to reduce weight gain.

5.6 BMI statistics

Participants were selected if their BMI was between 25 and 35 kg/m^2. The placebo group began the study with a mean BMI of 28.77 kg/m^2 and the treatment group began with a mean BMI of 28.14 kg/m^2.

After the twelve week period, the placebo group ended the study with a mean BMI of 29.16 kg/m^2 and the treatment group ended with a mean BMI of 28.31 kg/m^2. The results acquired show that the average BMI increased for the placebo and treatment group. The placebo group had an average increase in BMI of 0.39kg/m^2 and the treatment group had an average increase in BMI of 0.17 kg/m^2 over the twelve week period.

As shown in Table 14.4, there was a statistically significant change in BMI over the twelve week study, with each p-value (Asymp. Sig) being less that 0.05. The placebo group showed a p-value (Asymp. Sig) of 0.014 and the treatment group showed a p-value (Asymp. Sig) of 0.019.

Using the Wilcoxon Signed Ranks test, using stricter parameters, it was shown that there was no statistically significant change in BMI in the treatment group which had a p-value (Asymp. Sig. (2-tailed)) of 0.127 from week 0 to week 12. The placebo group showed a statistically significant gain in BMI of 0.39 kg/m^2 over the twelve week study, with a p-value (Asymp. Sig. (2-tailed)) of 0.04 from week 0 to week 12.

Because there was a significant increase in BMI within the placebo group compared to the treatment group which showed no statistically significant change, it is suggested that the herbal formulation may have prevented significant weight gain and increase in BMI in this study. As stated from 2.8.1.1 to 2.8.1.7, the herbal formulation containing Caffeine*, Coffea canephora* bean*, Coleus forskholii, Camellia sinensis,* Evodiamine*, Ilex paraguariensis* and *Phaseolus vulgaris* has many different effects on the body which may have helped to reduce weight gain.

Caffeine, *Coleus forskholii*, *Camellia sinensis* and Evodiamine have all been shown to increase the body's metabolic activity, thereby enhancing energy use and aiding in weight loss and weight stabilization (Clear Springs Press, 2009; Chantre *et al.,* 1999; Hoshikuma *et al.,* 2001). *Coffea canephora* bean, *Coleus forskholii,* Evodiamine *and Ilex paraguariensis* have all shown to have an effect on fat metabolism (Aitani *et al.*, 2006; Godard *et al.,* 2005; Hoshikuma *et al.,* 2001; Birketvedt *et al.,* 2002). *Coffea canephora* bean decreases the absorption of fat and, along with *Coleus forskholii,* increases the metabolism of fat (Aitani *et al.*, 2006; Henderson, 2005). Evodiamine has been shown to reduce fat deposition in the body (Wang and Wang, 2008) and *Ilex paraguarensis* has been found to increase fat excretion from the body (Birketvedt *et al.,*

2002). The combined effect of these products suggests that the herbal formulation may prevent weight gain by preventing fat deposition and absorption, increasing fat metabolism and excretion and increasing metabolic activity.

Certain products incorporated in the herbal formulation, such as *Phaseolus vulgaris*, have shown to significantly reduce body weight, BMI and weight circumference in overweight men and women (Preuss *et al.,* 2010). This suggests that certain herbs need to be taken over longer periods of time in order to have successful results. Therefore another reason that the treatment group did not lose weight may be that the duration of the study was not long enough, indicating that a longer study period may be needed.

Dietary factors discussed in 5.5 may have also affected the results of the BMI measurements as the participants overall quality and quantity of food was not specified or monitored.

This may show that although there were no statistically significant changes in BMI between the placebo and the treatment groups, the herbal formulation containing Caffeine*, Coffea canephora* bean*, Coleus forskholii, Camellia sinensis,* Evodiamine*, Ilex paraguariensis* and *Phaseolus vulgaris* may reduce weight gain and BMI increase.

5.7 Abdominal girth statistics

Abdominal girth measurements were taken four times over the twelve week study. The placebo group began the study with a mean abdominal girth of 91.04cm and the treatment group began with a mean abdominal girth of 92.36cm.

The placebo group ended the twelve week study at an average abdominal girth of 91.74cm and the treatment group ended at 91.36cm. The placebo group had an average gain in abdominal girth measurements of 0.7cm, while the treatment group showed a 1cm loss in abdominal girth over the twelve week study.

There was a no statistically significant change in abdominal girth over time in the placebo group, which had a p-value (Asymp. Sig) of 0.074. The treatment group showed a statistically significant change in abdominal girth with a p-value (Asymp. Sig) of 0,002. The statistically significant change was shown to be between week 0 and week 6 of the study using the Wilcoxon Signed Ranks test. The treatment group, at week 0, had a mean abdominal girth of 92.36cm and at week 6, was at 90.74cm. The average loss in abdominal girth was 1.62cm during this time. The placebo group had an average loss of only 0.12cm over the same period of time, which was not shown to be statistically significant.

Over the last six weeks of the study (week 6= 90.74cm; week 12= 91.36cm), the treatment group showed an increase of 0.62cm in abdominal girth. The placebo group also showed an increase in abdominal girth of 0.82cm over the last six weeks (week 6= 90.92cm; week 12= 91.74cm).

The reason that the treatment group may have had an increase in abdominal girth over the last six weeks of the study after having lost abdominal girth size in the first six weeks may have several contributing factors.

Firstly, diet plays a major role in weight loss and may be the single most important factor affecting it. Because the participants were not on a specific eating plan or exercise regime, as mentioned in 5.5, they may have eaten more and exercised less over the final six weeks of the study, therefore influencing their abdominal girth measurements.

Secondly, there were no guidelines on how much exercise each individual should be doing, as they were asked to maintain their normal lifestyle. Abdominal obesity and abdominal girth measurement were shown to be reduced in overweight, sedentary individuals when they underwent certain exercise regimes, even though no weight loss was evident. This shows evidence that even though weight loss may not occur, abdominal girth can be reduced and weight and abdominal girth should be monitored independently of one another (Blair *et al.,* 2009).

Thirdly, the last six weeks of the study were conducted over the festive season from the middle of November until the beginning of January for all the participants. Generally people have been known to over indulge in food and alcohol over this time. A study was conducted on weight gain over the same period of time (from Thanksgiving to New Year's Day) in the United States. It was estimated that an average individual would gain 0.2 to 0.8 kg over a one year period. The study showed that over this holiday period (mid-November to January) the participants gained between 0.37 kg and 1.52 kg, with an average gain of 0.48 kg which was not lost thereafter (Nguyen *et al.,* 2000).

This suggests that the average abdominal girth loss in the first six weeks and overall gain in the subsequent weeks may be due to the fact that week 6 to week 12 was conducted over December which has been shown to be a period of weight gain.

Although there was no significant difference in abdominal girth measurements between the treatment and placebo groups, the results show that the herbal formulation containing Caffeine*, Coffea canephora* bean*, Coleus forskholii, Camellia sinensis,* Evodiamine*, Ilex paraguariensis*

and *Phaseolus vulgaris* may be able to reduce abdominal girth measurements in overweight and obese individuals.

5.8 Summary of results

There was no significant difference between the placebo and treatment groups for weight, BMI or abdominal girth however some changes were noted within each of these groups.

The reason that the herbal formulation did not have any significant effect on weight loss may be due to several factors. Patient compliance may have been poor with regard to taking the medication as it was indicated. The participants of the study were also asked to maintain their normal lifestyle, thereby allowing them to eat food which may have been weight enhancing. There was also no exercise regime specified throughout the study, which could account for poor weight loss.

The study was also conducted over the December festive season which is a period in the year known for overindulgence in food and alcohol as well as a more sedentary period of time, which could have negatively affected the results of the study.

However, the placebo group did have a greater net gain in weight and BMI over the twelve week period which may indicate that the herbal formulation used may be able to reduce weight gain or increasing an individual's metabolic rate.

The treatment group had a significant loss in abdominal girth measurement over the first six weeks compared to the placebo group. Over the second six weeks, the average abdominal girth in the treatment group did increase slightly, which could be attributed to the holiday season from mid-November to early January. However, the results in the first six weeks of the study show that the herbal formulation may be able to reduce abdominal girth measurements.

CHAPTER 6

CONCLUSION AND RECOMMENDATIONS

6.1 Conclusion

The results of the study show that the herbal formulation containing Caffeine, *Coffea canephora* bean, *Coleus forskholii, Camellia sinensis,* Evodiamine, *Ilex paraguariensis* and *Phaseolus vulgaris* was ineffective in decreasing weight and BMI in overweight and obese individuals over a twelve week period.

The overall results showed that there were no significant differences in weight between the placebo and the treatment groups, but the treatment group did not gain as much weight as the placebo group. This suggests that the herbal formulation containing Caffeine, *Coffea canephora* bean, *Coleus forskholii, Camellia sinensis,* Evodiamine, *Ilex paraguariensis* and *Phaseolus vulgaris* may be able to reduce weight gain in overweight and obese individuals.

The placebo group showed a significant increase in BMI between week 0 and week 12. The treatment group showed no significant increase or decrease in BMI at any point of the study. Although the differences in BMI between each group were not significant, the inter-group analysis shows that the placebo group did have a greater increase in BMI compared to the treatment group. Therefore, the herbal formulation containing Caffeine, *Coffea canephora* bean, *Coleus forskholii, Camellia sinensis,* Evodiamine, *Ilex paraguariensis* and *Phaseolus vulgaris* may reduce weight gain and therefore prevent an increase in BMI in overweight and obese individuals.

There were no statistically significant changes in abdominal girth measurements between the placebo and treatment groups. However, there was a significant decrease in abdominal girth measurements for the treatment group which occurred between week 0 and week 6 of the study. The results showed that the treatment group had significant change in abdominal girth measurements, meaning that the herbal formulation containing Caffeine, *Coffea canephora* bean, *Coleus forskholii, Camellia sinensis,* Evodiamine, *Ilex paraguariensis* and *Phaseolus vulgaris* may reduce abdominal girth measurements in overweigh and obese individuals.

6.2 Recommendations for further research

Future research which uses the same herbal formulation used in this study should consider the following recommendations:

- Using a calorie restricting diet in the placebo and the treatment groups to limit other variables, such as unhealthy eating, affecting the study.
- Provide an exercise regime for each of the participants to follow to limit the variable effect that excessive or insufficient exercise may have on the study.
- Avoid conducting the study over periods of overindulgence, such as holidays, in order to prevent such periods affecting the study.
- Reduce the age group used to a smaller sample group in order to see the effects of the herbal formulation on a specific age group that may be more prone to obesity.
- Use overweight or obese males only, as female hormones such as oestrogen and progesterone may affect weight gain or loss.
- The duration of the study could be extended as some of the herbs in the herbal formulation need to be used over extended periods of time.
- A homoeopathic similimum which corresponds to any underlying causes of overweight and obesity could be used in conjunction with the herbal formulation.

RERERENCES

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APPENDIX A

Advertisement

Overweight?

Are you between the ages of 18 – 45?

You are invited to take part in a weight loss study conducted by the Department of Homoeopathy.

Consultations and medication are FREE OF CHARGE

DURATION: 12 weeks

WHERE: University of Johannesburg Campus Health Clinic

CONTACT PERSON: Katherine Withers 0836963717

Robert Durrheim 0823436492

Nancy Lord 0824603251

Ethical clearance number: AEC 63/01-2011, AEC63/02-2011, AEC64/02-2011

APPENDIX B

Participant information and consent form

Dear Prospective Participant

My name is Robert Durrheim and I am currently studying Homoeopathy at the University of Johannesburg. I am in my fifth and final year and I am currently conducting my research study in order to qualify for an M-Tech Homoeopathy degree. The aim of this study is to determine the effect of a herbal formulation consisting of Caffeine*, Coffea canephora* bean*, Coleus forskholii, Camellia sinensis,* Evodiamine*, Ilex paraguariensis* and *Phaseolus vulgaris* on Body Mass Index and abdominal girth measurements in overweight and obese individuals.

Outline of the study

The research study will be conducted over a twelve week period at the University of Johannesburg Health Centre. You will be asked to come into the clinic four times over the twelve week period and all treatment will be free of charge.

If you are between the ages of 18 and 45 years with a Body Mass Index (BMI) between 25 and 35kg/m^2 on the initial consultation, you are invited to take part in this study. During this study you are requested to maintain your normal lifestyle, with no major changes in stress levels, eating and exercise habits as much as is possible and in your control.

If you have any chronic illnesses, are on any chronic allopathic, herbal or homoeopathic medication, are on a weight loss programme, have known allergies or sensitivities to any substances in the herbal formulation or are female participants that are pregnant or breastfeeding, you will be excluded from the research study.

During the initial consultation a case taking will be performed and your vital signs (blood pressure, respiratory rate, pulse rate and temperature) will be checked. Your abdominal girth measurements will be taken and your height and weight measured in order to determine your Body Mass Index. At this consultation you will be administered medication which will either be the active herbal formulation or a placebo. This process is randomised and neither you nor the researcher will know which one you are given. The placebo group is one which receives an inactive substance in capsule form for the duration of the study and it may have no effect on weight loss. However once the study is completed the placebo group will receive the active herbal formulation as well as an eating plan in order to encourage weight loss. After the initial consultation you will be asked to return for evaluations in the second, sixth and twelfth week of

the study. During these follow up consultations, the same measurements will be taken in order to determine any weight loss and you will be given your next prescription of medication.

During the twelve week period, you will be asked to take two capsules of the administered medication per day, in the morning, on an empty stomach from Monday to Friday with a two day rest period on Saturday and Sunday. If any adverse effects such as nausea are experienced, you are requested to take your medication with your meals.

As a participant of this study, you are assured anonymity, confidentiality and rights to privacy. Participation is voluntary and you may withdraw from the research study at any point in time if you wish to do so. The results of the research will be made available to you once it has been concluded. There are no anticipated side effects of the medication, however if you experience any worrying symptoms during the course of the study stop the medication immediately. If necessary please contact your health care provider and the researcher, Robert Durrheim on 0823436492. If there are any questions pertaining to the study during its duration, please feel free to contact the researcher.

Participant Declaration and Consent

I, the participant am fully aware of and understand all the risks and benefits of this research procedure. I am aware that my participation in this study is voluntary and that I am free to withdraw my participation at any time.

The research procedure has been fully explained to me and I give my permission to the researcher that he may include me as a participant in this study.

I have been informed that if I have any queries or experience any side effects during the duration of the study, that I am free to contact Robert Durrheim on 0823436492

_____ _____

Participant's Signature Date

Researcher Declaration

I, the researcher, have fully explained the research procedure as well as its risks and benefits to the participant. I will have his/her best interests at heart throughout the study in order to assure

his/her safety. Any questions the participant may have will be answered honestly and to the best of my medical knowledge.

_____	_____
Researcher	Date

APPENDIX C

Consultation form

Name: _____

Date: _____

Week of study: _____

Consultation number: _____

Vital Signs

Heart rate: _____

Respiratory rate: _____

Blood pressure: _____

Temperature: _____

BMI

Height: _____

Weight: _____

BMI: _____

Abdominal Girth Measurement

Abdominal girth measurement 1 (cm): _____

Abdominal girth measurement 2 (cm): _____

Abdominal girth measurement 3 (cm): _____

Mean Abdominal girth measurement (cm): _____

APPENDIX D

BMI calculation (Beers *et al.,* 2006)

$$\text{BMI} = \frac{\text{Weight (kg)}}{\text{Height (m}^2\text{)}}$$

APPENDIX E

BMI categorization graph

WEIGHT lbs	100	105	110	115	120	125	130	135	140	145	150	155	160	165	170	175	180	185	190	195	200	205	210	215
kgs	45.5	47.7	50.0	52.3	54.5	56.8	59.1	61.4	63.6	65.9	68.2	70.5	72.7	75.0	77.3	79.5	81.8	84.1	86.4	88.6	90.9	93.2	95.5	97.7
HEIGHT in/cm	Underweight					Healthy					Overweight					Obese				Extremely obese				
5'0" - 152.4	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38	39	40	41	42
5'1" - 154.9	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	36	37	38	39	40
5'2" - 157.4	18	19	20	21	22	22	23	24	25	26	27	28	29	30	31	32	33	33	34	35	36	37	38	39
5'3" - 160.0	17	18	19	20	21	22	23	24	24	25	26	27	28	29	30	31	32	32	33	34	35	36	37	38
5'4" - 162.5	17	18	18	19	20	21	22	23	24	24	25	26	27	28	29	30	31	31	32	33	34	35	36	37
5'5" - 165.1	16	17	18	19	20	20	21	22	23	24	25	25	26	27	28	29	30	30	31	32	33	34	35	35
5'6" - 167.6	16	17	17	18	19	20	21	21	22	23	24	25	25	26	27	28	29	29	30	31	32	33	34	34
5'7" - 170.1	15	16	17	18	18	19	20	21	22	22	23	24	25	25	26	27	28	29	29	30	31	32	33	33
5'8" - 172.7	15	16	16	17	18	19	19	20	21	22	22	23	24	25	25	26	27	28	28	29	30	31	32	32
5'9" - 175.2	14	15	16	17	17	18	19	20	20	21	22	22	23	24	25	25	26	27	28	28	29	30	31	31
5'10" - 177.8	14	15	15	16	17	18	18	19	20	20	21	22	23	23	24	25	25	26	27	28	28	29	30	30
5'11" - 180.3	14	14	15	16	16	17	18	18	19	20	21	21	22	23	23	24	25	25	26	27	28	28	29	30
6'0" - 182.8	13	14	14	15	16	17	17	18	19	19	20	21	21	22	23	23	24	25	25	26	27	27	28	29
6'1" - 185.4	13	13	14	15	15	16	17	17	18	19	19	20	21	21	22	23	23	24	25	25	26	27	27	28
6'2" - 187.9	12	13	14	14	15	16	16	17	18	18	19	19	20	21	21	22	23	23	24	25	25	26	27	27
6'3" - 190.5	12	13	13	14	15	15	16	16	17	18	18	19	20	20	21	21	22	23	23	24	25	25	26	26
6'4" - 193.0	12	12	13	14	14	15	15	16	17	17	18	18	19	20	20	21	22	22	23	23	24	25	25	26

(Healthinformatics, 2009)

APPENDIX F

Abdominal girth measurement (International Chair on Cardiometabolic Risk, 2011)

- Abdominal girth is measured by locating the iliac crests on both of the hip bones with the person facing forward with both arms at their sides.
- The tape measure is then wrapped around the waist, midway between the iliac crest and the lowest rib on each side ensuring that it is flat and horizontal.
- Once the tape measure is secured but not compressing or indenting the skin and after a normal expiration by the person, the reading is taken.
- Three measurements are taken in this area and the measurements are then added together and divided by three to get an average reading.
- The calculation will provide the mean abdominal girth of the person.

FNC-001-V04

PRODUCT MASTER FILE

THIN-K HERBAL BLISTER (10)

FARMASEUTIES BK
PHARMACEUTICALS CC

Client: Lunar Pharmaceuticals (Pt

Product code: TFR100 Rev no: 001 Product category: CAP

Date: 22/07/2011

Lunar Pharmaceuticals (Pt hereby instructs Sastel Pharmaceuticals CC to manufacture this product on the basis as set out herein below. Lunar Pharmaceuticals (Pt confirms that they familiarised themselfs with the formula and accepts responsibility that it complies to the BP, Martindale and the Laws and Regulations of the Replublic of South Africa. Lunar Pharmaceuticals (Pt hereby indemnifies Sastel Pharmaceuticals CC from any claim regarding to the composition of the specified formulation of the desired mixture, any pre-blended mixture supplied by Lunar Pharmaceuticals (Pt, and any packaging and labelling supplied by Lunar Pharmaceuticals (Pt.

Sastel Pharmaceuticals CC hereby accepts the responsibility for the accuracy and quality of the composition of the formula and that same is manufactured in accordance with the specified parameters as issued by Lunar Pharmaceuticals (Pt, as well as the accuracy and quality required of all labeling and / or packaging of product by Sastel Pharmaceuticals CC for Lunar Pharmaceuticals (Pt. Details of all responsibilities and requirements are reflected in the TECHNICAL AGREEMENT between the parties

Signature of Responsible Pharmacist from Lunar Pharmaceuticals (Pt

Date

Signature of Responsible Pharmacist of Sastel Pharmaceuticals CC

Date

		Unit	No	Description	Supl code	Ref.	Comments
Raw material	62.50 mg	0.000625 kg	1	Coleus Forskholi extract 40% from Lunar Pharmaceuticals			Fine dark brown powder
	100.00 mg	0.001000 kg	2	Green Tea (40% Polyphenols)			Fine light brown powder
	50.00 mg	0.000500 kg	3	Green Coffee Bean extract 50% from Lunar Pharmaceuticals			Fine light brown/light yellow powder
	50.00 mg	0.000500 kg	4	Caffeine anhydrous			Fine white powder
	120.00 mg	0.001200 kg	5	Finamate/Yerbamate/Green Mate leaf powder from Lunar			Fine light brown/dark yellow powder
	13.00 mg	0.000130 kg	6	Evodiamine 98% from Lunar Pharmaceuticals			
	30.00 mg	0.000300 kg	7	White Kidney Bean extract 20:1 from Lunar Pharmaceuticals			Fine light creamy powder
	425.50 mg			**Total raw material**			
Filling material	3.00 mg	0.000030 kg	40	Perkasil SM660			Fine white puffy powder
	10.00 mg	0.000100 kg	41	Magnesium Stearate			White fine, static, powdery powder
	13.00 mg			**Total filling material**			
	438.50 mg			**Total capsule weight**			
Packing material		10.000 each	50	Capsules white/white size 0			
		0.001000 kg	51	Blister foil unprinted 78mm			
		0.003000 kg	52	Blister clear PVC 78mm x 93mm diam, 250um			
		1.000 each	54	Silica gel sachet 0,5g	SILICA GEL 001		
		0.010000 each	56	Box standard size 4 - 30x20x30	SWB04		
Production		10.000 each	80	Capsule filling size 0		PDS-003	
		1.000 each	81	Blistering of 10 x size 0 caps 93 x 78 x 8mm		PDW-00	10 caps per blister

End of document

APPENDIX H

Shapiro-Wilk: Test of normality

		Kolmogorov-Smirnov[a]			Shapiro-Wilk		
Group		Statistic	df	Sig.	Statistic	df	Sig.
Age	Placebo	.213	29	.002	.910	29	.017
	Treatment	.218	28	.002	.876	28	.003
Height	Placebo	.149	29	.098	.933	29	.067
	Treatment	.095	28	.200*	.948	28	.172
Weight_w0	Placebo	.122	29	.200*	.959	29	.314
	Treatment	.152	28	.098	.962	28	.382
Weight_w2	Placebo	.101	29	.200*	.961	29	.340
	Treatment	.128	28	.200*	.965	28	.444
Weight_w6	Placebo	.122	29	.200*	.965	29	.440
	Treatment	.126	28	.200*	.961	28	.378
Weight_w12	Placebo	.145	29	.123	.953	29	.224
	Treatment	.155	28	.084	.950	28	.194
BMI_w0	Placebo	.178	29	.019	.889	29	.005
	Treatment	.204	28	.004	.840	28	.001
BMI_w2	Placebo	.154	29	.077	.919	29	.028
	Treatment	.185	28	.015	.865	28	.002
BMI_w6	Placebo	.154	29	.075	.909	29	.016
	Treatment	.186	28	.015	.885	28	.005
BMI_w12	Placebo	.176	29	.023	.896	29	.008
	Treatment	.141	28	.161	.927	28	.051
AG_w0	Placebo	.133	29	.200*	.970	29	.559
	Treatment	.176	28	.026	.942	28	.127
AG_w2	Placebo	.130	29	.200*	.982	29	.892
	Treatment	.142	28	.156	.947	28	.166
AG_w6	Placebo	.078	29	.200*	.977	29	.766
	Treatment	.178	28	.024	.931	28	.067
AG_w12	Placebo	.166	29	.040	.969	29	.534
	Treatment	.141	28	.166	.929	28	.059

APPENDIX I

Non-parametric tests between groups over time: Mann-Whitney test

I-1: Mann-Whitney test: Group statistics

Group		N	Mean	Std. Deviation	Std. Error Mean
Age	Placebo	29	29.07	7.392	1.373
	Treatment	28	28.43	7.239	1.368
Height	Placebo	29	1.68	.101	.019
	Treatment	28	1.72	.088	.017
Weight_w0	Placebo	29	81.28	13.610	2.527
	Treatment	28	82.97	12.201	2.306
Weight_w2	Placebo	29	81.11	13.729	2.549
	Treatment	28	82.69	12.374	2.339
Weight_w6	Placebo	29	81.67	13.635	2.532
	Treatment	28	82.78	12.460	2.355
Weight_w12	Placebo	29	82.36	14.220	2.641
	Treatment	28	83.61	12.592	2.380
BMI_w0	Placebo	29	28.77	3.188	.592
	Treatment	28	28.14	3.247	.614
BMI_w2	Placebo	29	28.72	3.292	.611
	Treatment	28	28.03	3.221	.609
BMI_w6	Placebo	29	28.93	3.211	.596
	Treatment	28	28.08	3.414	.645
BMI_w12	Placebo	29	29.16	3.407	.633
	Treatment	28	28.31	3.397	.642
AG_w0	Placebo	29	91.04	9.307	1.728
	Treatment	28	92.36	8.911	1.684
AG_w2	Placebo	29	91.57	9.546	1.773
	Treatment	28	92.10	9.002	1.701
AG_w6	Placebo	29	90.92	9.998	1.857
	Treatment	28	90.74	8.432	1.594
AG_w12	Placebo	29	91.74	9.298	1.727
	Treatment	28	91.36	8.859	1.674

I-2: Mann-Whitney test: Ranks

Group		N	Mean Rank	Sum of Ranks
Age	Placebo	29	29.86	866.00
	Treatment	28	28.11	787.00
	Total	57		
Height	Placebo	29	25.88	750.50
	Treatment	28	32.23	902.50
	Total	57		
Weight_w0	Placebo	29	28.09	814.50
	Treatment	28	29.95	838.50
	Total	57		
Weight_w2	Placebo	29	27.93	810.00
	Treatment	28	30.11	843.00
	Total	57		
Weight_w6	Placebo	29	28.53	827.50
	Treatment	28	29.48	825.50
	Total	57		
Weight_w12	Placebo	29	28.53	827.50
	Treatment	28	29.48	825.50
	Total	57		
BMI_w0	Placebo	29	30.45	883.00
	Treatment	28	27.50	770.00
	Total	57		
BMI_w2	Placebo	29	30.55	886.00
	Treatment	28	27.39	767.00
	Total	57		
BMI_w6	Placebo	29	31.45	912.00
	Treatment	28	26.46	741.00
	Total	57		
BMI_w12	Placebo	29	30.67	889.50
	Treatment	28	27.27	763.50
	Total	57		
AG_w0	Placebo	29	27.67	802.50
	Treatment	28	30.38	850.50
	Total	57		
AG_w2	Placebo	29	28.78	834.50
	Treatment	28	29.23	818.50
	Total	57		
AG_w6	Placebo	29	29.12	844.50
	Treatment	28	28.88	808.50

	Total	57		
AG_w12	Placebo	29	29.64	859.50
	Treatment	28	28.34	793.50
	Total	57		

I-3: Mann-Whitney test: Test statistics

	Mann-Whitney U	Wilcoxon W	Z	Asymp. Sig. (2-tailed)
Age	381.000	787.000	-.400	.689
Height	315.500	750.500	-1.448	.148
Weight_w0	379.500	814.500	-.423	.672
Weight_w2	375.000	810.000	-.495	.621
Weight_w6	392.500	827.500	-.215	.829
Weight_w12	392.500	827.500	-.216	.829
BMI_w0	364.000	770.000	-.671	.502
BMI_w2	361.000	767.000	-.719	.472
BMI_w6	335.000	741.000	-1.134	.257
BMI_w12	357.500	763.500	-.774	.439
AG_w0	367.500	802.500	-.615	.539
AG_w2	399.500	834.500	-.104	.917
AG_w6	402.500	808.500	-.056	.955
AG_w12	387.500	793.500	-.295	.768

APPENDIX J

Friedman test statistics: Non-parametric tests within groups over time

J-1: Weight descriptive statistics

Group		N	Mean	Std. Deviation	Minimum	Maximum
Placebo	Weight_w0	29	81.28	13.610	60	114
	Weight_w2	29	81.11	13.729	60	114
	Weight_w6	29	81.67	13.635	60	114
	Weight_w12	29	82.36	14.220	61	114
Treatment	Weight_w0	28	82.97	12.201	60	110
	Weight_w2	28	82.69	12.374	61	111
	Weight_w6	28	82.78	12.460	60	110
	Weight_w12	28	83.61	12.592	61	114

J-2: BMI descriptive statistics

Group		N	Mean	Std. Deviation	Minimum	Maximum
Placebo	BMI_w0	29	28.77	3.188	25	35
	BMI_w2	29	28.72	3.292	24	35
	BMI_w6	29	28.93	3.211	25	35
	BMI_w12	29	29.16	3.407	25	35
Treatment	BMI_w0	28	28.14	3.247	25	36
	BMI_w2	28	28.03	3.221	25	35
	BMI_w6	28	28.08	3.414	24	36
	BMI_w12	28	28.31	3.397	23	35

J-3: Abdominal girth descriptive statistics

Group		N	Mean	Std. Deviation	Minimum	Maximum
Placebo	AG_w0	29	91.04	9.307	72	113
	AG_w2	29	91.57	9.546	72	113
	AG_w6	29	90.92	9.998	70	111
	AG_w12	29	91.74	9.298	74	113
Treatment	AG_w0	28	92.36	8.911	80	113
	AG_w2	28	92.10	9.002	80	113
	AG_w6	28	90.74	8.432	80	109
	AG_w12	28	91.36	8.859	79	110

APPENDIX K

Friedman ranks test

K-1: Weight ranks

Group		Mean Rank
Placebo	Weight_w0	2.34
	Weight_w2	2.05
	Weight_w6	2.60
	Weight_w12	3.00
Treatment	Weight_w0	2.55
	Weight_w2	2.07
	Weight_w6	2.16
	Weight_w12	3.21

K-2: BMI ranks

Group		Mean Rank
Placebo	BMI_w0	2.33
	BMI_w2	2.03
	BMI_w6	2.57
	BMI_w12	3.07
Treatment	BMI_w0	2.52
	BMI_w2	2.13
	BMI_w6	2.25
	BMI_w12	3.11

K-3: Abdominal girth ranks

Group		Mean Rank
Placebo	AG_w0	2.33
	AG_w2	2.57
	AG_w6	2.14
	AG_w12	2.97
Treatment	AG_w0	3.05
	AG_w2	2.79
	AG_w6	1.82
	AG_w12	2.34

APPENDIX L

Wicoxon signed ranks test: Ranks

Group				N	Mean Rank	Sum of Ranks
Placebo	Weight_w2 - Weight_w0		Negative Ranks	16[a]	15.16	242.50
			Positive Ranks	12[b]	13.63	163.50
			Ties	1[c]		
			Total	29		
	Weight_w6 - Weight_w0		Negative Ranks	9[d]	12.56	113.00
			Positive Ranks	16[e]	13.25	212.00
			Ties	4[f]		
			Total	29		
	Weight_w12 - Weight_w0		Negative Ranks	11[g]	11.14	122.50
			Positive Ranks	17[h]	16.68	283.50
			Ties	1[i]		
			Total	29		
	BMI_w2 - BMI_w0		Negative Ranks	15[ed]	14.37	215.50
			Positive Ranks	12[ce]	13.54	162.50
			Ties	2[cf]		
			Total	29		
	BMI_w6 - BMI_w0		Negative Ranks	8[cg]	10.88	87.00
			Positive Ranks	15[ch]	12.60	189.00
			Ties	6[ci]		
			Total	29		
	BMI_w12 - BMI_w0		Negative Ranks	11[cj]	10.27	113.00
			Positive Ranks	17[ck]	17.24	293.00
			Ties	1[cl]		
			Total	29		
	AG_w2 - AG_w0		Negative Ranks	10[cm]	10.80	108.00
			Positive Ranks	13[cn]	12.92	168.00

		Ties	6[co]		
		Total	29		
	AG_w6 - AG_w0	Negative Ranks	16[cp]	14.91	238.50
		Positive Ranks	13[cq]	15.12	196.50
		Ties	0[cr]		
		Total	29		
	AG_w12 - AG_w0	Negative Ranks	9[cs]	15.33	138.00
		Positive Ranks	19[ct]	14.11	268.00
		Ties	1[cu]		
		Total	29		
Treatment	Weight_w2 - Weight_w0	Negative Ranks	17[a]	15.68	266.50
		Positive Ranks	11[b]	12.68	139.50
		Ties	0[c]		
		Total	28		
	Weight_w6 - Weight_w0	Negative Ranks	18[d]	12.81	230.50
		Positive Ranks	9[e]	16.39	147.50
		Ties	1[f]		
		Total	28		
	Weight_w12 - Weight_w0	Negative Ranks	8[g]	15.94	127.50
		Positive Ranks	20[h]	13.93	278.50
		Ties	0[i]		
		Total	28		
	BMI_w2 - BMI_w0	Negative Ranks	15[cd]	13.90	208.50
		Positive Ranks	10[ce]	11.65	116.50
		Ties	3[cf]		
		Total	28		
	BMI_w6 - BMI_w0	Negative Ranks	17[cg]	12.85	218.50
		Positive Ranks	10[ch]	15.95	159.50
		Ties	1[ci]		
		Total	28		
	BMI_w12 - BMI_w0	Negative Ranks	8[cj]	15.69	125.50

	Positive Ranks	19[ck]	13.29	252.50
	Ties	1[cl]		
	Total	28		
AG_w2 - AG_w0	Negative Ranks	16[cm]	12.91	206.50
	Positive Ranks	10[cn]	14.45	144.50
	Ties	2[co]		
	Total	28		
AG_w6 - AG_w0	Negative Ranks	22[cp]	15.23	335.00
	Positive Ranks	5[cq]	8.60	43.00
	Ties	1[cr]		
	Total	28		
AG_w12 - AG_w0	Negative Ranks	18[cs]	15.58	280.50
	Positive Ranks	10[ct]	12.55	125.50
	Ties	0[cu]		
	Total	28		

APPENDIX M

Wilcoxon signed ranks test: Paired sample statistics

Group			Mean	N	Std. Deviation	Std. Error Mean
Placebo	Pair 1	Weight_w0	81.28	29	13.610	2.527
		Weight_w2	81.11	29	13.729	2.549
	Pair 2	Weight_w0	81.28	29	13.610	2.527
		Weight_w6	81.67	29	13.635	2.532
	Pair 3	Weight_w0	81.28	29	13.610	2.527
		Weight_w12	82.36	29	14.220	2.641
	Pair 4	BMI_w0	28.77	29	3.188	.592
		BMI_w2	28.72	29	3.292	.611
	Pair 5	BMI_w0	28.77	29	3.188	.592
		BMI_w6	28.93	29	3.211	.596
	Pair 6	BMI_w0	28.77	29	3.188	.592
		BMI_w12	29.16	29	3.407	.633
	Pair 7	AG_w0	91.04	29	9.307	1.728
		AG_w2	91.57	29	9.546	1.773
	Pair 8	AG_w0	91.04	29	9.307	1.728
		AG_w6	90.92	29	9.998	1.857
	Pair 9	AG_w0	91.04	29	9.307	1.728
		AG_w12	91.74	29	9.298	1.727
Treatment	Pair 1	Weight_w0	82.97	28	12.201	2.306
		Weight_w2	82.69	28	12.374	2.339
	Pair 2	Weight_w0	82.97	28	12.201	2.306
		Weight_w6	82.78	28	12.460	2.355
	Pair 3	Weight_w0	82.97	28	12.201	2.306
		Weight_w12	83.61	28	12.592	2.380
	Pair 4	BMI_w0	28.14	28	3.247	.614
		BMI_w2	28.03	28	3.221	.609
	Pair 5	BMI_w0	28.14	28	3.247	.614
		BMI_w6	28.08	28	3.414	.645
	Pair 6	BMI_w0	28.14	28	3.247	.614
		BMI_w12	28.31	28	3.397	.642

	Pair 7	AG_w0	92.36	28	8.911	1.684
		AG_w2	92.10	28	9.002	1.701
	Pair 8	AG_w0	92.36	28	8.911	1.684
		AG_w6	90.74	28	8.432	1.594
	Pair 9	AG_w0	92.36	28	8.911	1.684
		AG_w12	91.36	28	8.859	1.674