THE EFFECT OF A CORPORATE WELLNESS PROGRAMME IN REDUCING SELECTED MODIFIABLE CORONARY ARTERY DISEASE RISK FACTORS IN MEN

by

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ABSTRACT

Chronic degenerative disease is responsible for a high percentage of deaths in industrialised westernised communities. These findings are not surprising if viewed in the light that most people consider physical activity not to be a priority in their daily activities. During the last decade, it would seem that employers have realised that the health status of an employee can have a direct influence on productivity, efficiency and absenteeism in the working environment. In an attempt to lower health-related costs, reduce absenteeism and improve productivity, some employers have started implementing a variation of total well-being and workers' support programmes as part of their employee assistance programmes. A pressing question is, however, how effective these wellness programmes are.

In order to evaluate the effect of such a programme, the present study used a sample of 76 employees in middle- to top management at a big corporate company.

During 2002, tests were performed on twelve CAD risk factors, after which an intervention programme was introduced. Twelve months later (2003), the first intermediate test was conducted, using the same protocol. The second intermediate test followed in 2004, duplicating the procedures. The post-test and data analysing were conducted four years after the first evaluations. Subjects were monitored for changes in: total cholesterol, HDL-cholesterol, LDL-cholesterol, triglycerides, TC/HDL ratio, LDL/HDL ratio, fasting blood glucose, systolic blood pressure, diastolic blood pressure, body mass index, waist-to-hip-ratio and waist circumference.

The Repeated Measures General Linear Model Test was used to determine significance ($P \leq 0.05$) from pre- to post-test. A novel CAD risk score was also

developed from peer-reviewed literature by considering each of the 12 CAD risk factors measured.

The results demonstrated that the wellness programme decreased CAD risk by 26% amongst the 76 participants in this study. The TC, LDL-C, LDL/HDL-C ratio, TC/HDL-C ratio, fasting blood glucose, resting systolic blood pressure, resting diastolic blood pressure and waist-to-hip ratio improved significantly, while triglycerides showed a non-significant improvement. The three CAD risk factors that deteriorated significantly during the study period were high-density lipoprotein cholesterol, BMI and waist circumference.

The major finding of this investigation thus suggests that a corporate wellness programme has long-term beneficial effects on CAD risk and that the reduction in CAD risk is mainly attributed to the beneficial effects of regular exercise.

Keywords: exercise, coronary artery disease, wellness, risk factors, heart disease.

OPSOMMING

Kroniese degeneratiewe siektes is verantwoordelik vir ’n hoë persentasie van sterftes in geïndustrialiseerde verwesterde gemeenskappe. Hierdie bevindings is nie ’n verrassing nie, veral nie in die lig van die feit dat die meeste mense gladnie fisieke aktiwiteite as prioriteit in hul daaglikse roetine beskou nie. Gedurende die afgelope dekade het dit geblyk dat werkgewers besef het dat die goeie gesondheid van ’n werknemer ’n direkte impak kan hê op produktiwiteit, effektiwiteit en afwesigheid. Dus het sommige werkgewers begin om ’n verskeidenheid van totale welstandsprogramme as deel van hul maatskappy se werknemershulpprogramme aan te bied. Die vraag bly egter steeds of hierdie welstandsprogramme enigsins effektief is.

Ten einde die effek van so ’n program te evalueer, het hierdie studie ’n steekproef van 76 werknemers van die middel- en topbestuur van ’n korporatiewe maatskappy gebruik.

Gedurende 2002 is daar toetse gedoen op 12 risikofaktore vir koronêre bloedvatsiektes, waarna ’n intervensieprogram ingestel is. Twaalf maande later (2003) is die eerste opvolg-toetse uitgevoer deur dieselfde protokol te gebruik. Die tweede opvolg-toetse is in 2004 uitgevoer en die finale toetse is vier jaar na die eerste toets uitgevoer. Proefpersone is gemonitor vir enige veranderinge in totale cholesterol, HDL-cholesterol, LDL-cholesterol, trigliseriedes, TC/HDL-C verhouding, LDL-C/HDL-C verhouding, vastende bloedglukose, sistoliese bloeddruk, diastoliese bloeddruk, liggaamsmassa-indeks, middel-tot-heup verhouding en middelomtrek.

Die “Repeated Measures General Linear Model Test” is gebruik om die betekenisvolheid ($P \leq 0.05$) van die veranderinge te toets. ’n Nuwe koronêre risikotelling vir kroonslagaarsiekte is ook ontwikkel met behulp van die literatuur.

Hierdie resultate het gedemonstreer dat die 76 deelnemers gebaat het by die program, deurdat hul risiko vir koronêre siektes met 26% afgeneem het. Die TC, LDL-C, LDL-C/HDL-C verhouding, vastende bloedglukose, rustende sistoliese bloeddruk, rustende diastoliese bloeddruk en middel-tot-heup verhouding het betekenisvol verbeter tydens die studie, terwyl trigliseriedes nie betekenisvol verbeter het nie. Die drie risikofaktore wat betekenisvol verswak het gedurende die toetse, was HDL-C, LMI en middelomtrek.

Die belangrikste bevindinge van hierdie ondersoek dui dus daarop dat ’n korporatiewe welstandsprogram ’n voordelige langtermynuitwerking het op koronêre risikofaktore, en dat hierdie verbetering hoofsaaklik aan gereelde oefening toegeskryf kan word.

Trefwoorde: oefening, koronêre bloedvatsiektes, welstand, risikofaktore, hartsiektes.

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LIST OF ABBREVATIONS

%	percentage
AACPVR	American Association of Cardiovascular and Pulmonary Rehabilitation
ACSM	American College of Sport Medicine
ACTH	adrenocorticotropic hormone
ADA	American Diabetic Association
AHA	American Heart Association
BMI	body mass index
BP	blood pressure
CAD	coronary artery disease
CETP	cholesterol ester transfer protein
CHD	coronary heart disease
cm	centimetres
CRH	corticotrophin releasing hormone
CRP	C-reactive protein
CVA	Cerebrovascular accident
CVD	cardiovascular disease
DART	Diet and Reinfarction Trial
DASH	Dietary Approach to Stop Hypertension
DBP	diastolic blood pressure
ECG	electrocardiogram
EURODIAB	Diabetes in Europe

FH	homozygous familial hypercholesterolemia
FITT	frequency, intensity, time, type
GI	glycaemic index
GL	glycaemic load
HDL-C	high-density lipoprotein cholesterol
HPA	Health Protection Agency
HT^2	height squared
IDDM	insulin dependent diabetes mellitus
IRS	independent variables to health status
JAMA	Journal of the American Medical Association
JNC	Joint National Committee
Kcal	kilocalorie
kg	kilogram
km	kilometres
km/h	kilometres per hour
LBM	lean body mass
LCAT	lecithin-cholesterol acyltransferase
LDL-C	low-density lipoprotein cholesterol
LDL/HDL-C	Low-density / High-density lipoprotein cholesterol ratio
LPA	low physical activity
LPL	lipoprotein lipase
LTA	London Transport Authority
MI	myocardial infarction

min	minute(s)
mm Hg	millimetres Mercury
mmol/l	millimoles per litre
MRFI	Multiple Risk Factor Intervention Trial
NIDDM	non insuline dépendant diabetes mellites
NHANES	National Health and Nutrition Examination Survey
n3 FA	Omega-3 polyunsaturated fatty acids
PAI	physical activity index
PWC	physical work capacity
RDBP	resting diastolic blood pressure
RSBP	resting systolic blood pressure
SA	South Africa
SANGALA	South African National Games and Leisure Activities
SBP	systolic blood pressure
TC	total cholesterol
TC/HDL-C	Total Cholesterol / High Density Lipoprotein Cholesterol ratio
USA	United States of America
VLDL-C	very low-density lipoprotein cholesterol
VLF	very low fat
VO_2 max	maximum oxygen consumption
WHO	World Health Organization
WHR	waist-to-hip ratio
WT	body weight

GLOSSARY OF TERMS

The following operational definitions and explanations are proposed to clarify and avoid any confusion within the present study:

Aerobic training (AER): Exercise training (i.e. cycling, swimming, and jogging) that takes place at intensity below that of anaerobic/lactic acid turning point where oxygen metabolism is primarily used (i.e. aerobic metabolism utilising oxygen to produce energy).

Arteriosclerosis: The end stage of atherosclerosis. It is characterised by the calcification of the artery wall (death of smooth muscle cells in tunica media and the deterioration of the elastic fibres of the vessel wall).

Basal metabolic rate (BMR): The rate at which energy is expended during rest.

Blood pressure (BP): The force per unit area exerted on the wall of the arterial blood vessel by its contained blood. BP is expressed in millimetres Mercury (mm Hg).

Body fat: Also called adipose tissue. Refers to fat-storing tissue.

Body mass index: Also called Q*uetelet Index.* It is used to assess weight relative to height.

Cardiac output: Amount of blood pumped out of a ventricle in one minute (Q=HR x SV).

Cerebrovascular accident (CVA): A stroke.

Cholesterol: A fatty substance found in all animal fat, but not in plant oils. It belongs to a family of chemicals called steroids. These include sex hormones such as oestrogen, progesterone and testosterone, stress hormones such as cortisone, bile acids, and vitamin D. Cholesterol plays an important role in the structure of all cell membranes and helps maintain cell wall rigidity.

Coronary artery disease (CAD): Can be considered as a collection of diseases affecting the heart and the body's vascular system (i.e. angina pectoris, cardiac arrest and myocardial infarction). CAD is the single most important cause of death and, more importantly, the single most important cause of premature death in modern, industrialised countries.

Coronary artery disease risk factors: When quantifying the risk of developing CAD, these are the statistical techniques used to estimate the strength of the relationship between CAD risk factors, and the likelihood of developing CAD. Risk factors for developing CAD can be divided into modifiable and non-modifiable primary, secondary, and post-traditional risk factors.

C-reactive protein (CRP): CRP is produced by the liver and is considered a sensitive marker of inflammation. It has been demonstrated to increase the prediction of lipid variables and the risk of coronary artery disease (Myocardial Infraction in particular).

Diabetes mellitus (DM): DM is a disease associated with lack of control in blood glucose, resulting in hyperglycaemia. There are two major types of diabetes, namely type 1, or juvenile-onset diabetes, and type 2, or adult-onset diabetes.

Diastolic blood pressure (DBP): DBP is the lowest pressure observed in the arteries and occurs after the aortic semilunar valve closes. It is approximately 70 to 80 mm Hg in healthy adults.

Dyslipidemia: Is generally characterised by hypertriglyceridemia, increased low-density lipoprotein cholesterol (LDL-C) and very low-density lipoprotein cholesterol (VLDL-C) and decreased high-density lipoprotein cholesterol (HDL-C).

Heart rate: The frequency of the heart's contraction per time unit. It is usually measured in beats per minute (bpm).

High-density lipoprotein cholesterol (HDL-C): Considered to be 'good' cholesterol, since high levels reduce an individual's tendency to develop atherosclerosis.

Homocysteine: Homocysteine is a highly reactive sulphur amino acid produced as a metabolite of methionine due to its demethylation, and may damage the vascular endothelial wall via several mechanisms.

Hypercholesterolemia: Implies only elevated cholesterol levels.

Hyperfibrinogenemia: An increased blood fibrinogen level.

Hyperglycaemia: Elevated or excessive blood glucose levels.

Hyperinsulinism: Excessive levels of insulin.

Hyperlipidemia: Excessive levels of lipids.

Hypertension (HTN): High blood pressure. HTN is a condition of increased peripheral vascular resistance and/or altered kidney function (BP > 140/90).

Hypertriglyceridemia: Excessive triglyceride (TG) levels.

Inactivity: Low levels of physical activity (less than 500 to 2000 calories per week).

Insulin: A hormone that enhances the carrier-mediated diffusion of glucose into the tissue cells, thereby lowering blood glucose levels.

Lipoproteins: Lipoproteins are small lipid-protein complexes that transport cholesterol and triglycerides to and from the body tissue.

Low-density lipoprotein cholesterol (LDL-C): Identified as 'bad' cholesterol, since high levels increase an individual's tendency to develop atherosclerosis.

Low-density lipoprotein cholesterol/ High-density lipoprotein cholesterol Ratio (LDL-C/HDL-C): used as an index of coronary artery disease (CAD) risk. Use specifically to determine the ratio between 'bad' and 'good' cholesterol.

Metabolic syndrome: A condition characterised by a combination of obesity, insulin resistance, hyperinsulinemia, hypertension, dyslipidemia and blood clotting abnormalities.

Obesity: Obesity is used to classify individuals who have excessive fat deposits according to their percentage body fat.

Resting blood pressure (RBP): The force per unit area exerted on the wall of arterial blood vessels by its contained blood at rest. Blood pressure is expressed in mm Hg, and consists of systolic and diastolic blood pressure.

Saturated fat: A fatty acid saturated with a hydrogen ion filling each site on a carbon molecule.

Serum lipid factors: Serum lipid factors are individual serum lipoprotein and lipid parameters associated with an increased risk of coronary artery disease.

Smoking: Refer to the habitual smoking of cigarettes, cigars and/or pipe.

Stress: Any circumstance that threaten, or are perceived to threaten, one's wellbeing and which thereby taxes one's coping abilities.

Systolic blood pressure (SBP): The peak pressure exerted by the blood in the arteries as the left ventricle contracts. SBP is approximately 110 to 130 millimetres of Mercury (mm Hg) in healthy adults.

Total cholesterol (TC): The total amount of cholesterol, i.e. high-density lipoprotein cholesterol (HDL-C) and low-density lipoprotein cholesterol (LDL-C) and triglycerides (TG) combined to form a single value.

Total cholesterol/high-density lipoprotein cholesterol ratio (TC/HDL-ratio): used as an index of coronary artery disease (CAD) risk.

Triglycerides (TG): Also called triglycerol. Fats and oils composed of fatty acids and glycerol. Triglycerides are the body's most concentrated source of energy and are also known as neutral fats.

Uric acid: Uric acid is the end product of purine catabolism and is produced mainly by the kidneys when renal function is not impaired. Uric acid is increased in a variety of clinicopathologic entities including gout, haemolytic anaemia, polycythemia, and other coronary artery disease (CAD) risk factors including hypertension, dyslipidemia, diabetes mellitus and obesity.

Very low-density lipoprotein cholesterol (VLDL-C): The cholesterol contained within the VLDL compound.

Waist-to-hip ratio (WHR): The WHR is a simple and inexpensive method for determining body fat pattern, since individuals with more fat on the trunk (especially abdominal fat) are at increased risk for CAD.

CHAPTER ONE: INTRODUCTION AND AIM OF THE STUDY

1.1 Introduction

Chronic degenerative disease is responsible for more than 70% of deaths in industrialised westernised communities (Chapman, 1991). Strydom *et al.* (1998) reported that South African men showed four coronary artery disease (CAD) risk factors: high serum cholesterol, smoking, hypertension and physical inactivity. Dreyer (1996) found elevated values for total cholesterol (TC), LDL-cholesterol (LDL-C), triglycerides and the TC/HDL-cholesterol ratio (TC/HDL-C ratio) in white males. A study done by Van Zyl (1995) showed that 61.8% of employees in middle management had elevated TC levels (> 5.2 mmol/l).

These findings are not surprising if viewed in light of an investigation conducted by Uys and Coetzee (1989), who found that only 12.4% of male managers consider physical activity a priority in their schedule. The results of a study by Dreyer (1991) on 777 male managers and academics revealed that only 2.8% of the respondents were physically active at work, although 63.8% indicated that they participated in regular physical activities or sport in their own time. However, making use of the physical activity index of Sharkey (1984), Dreyer (1991) found that only 14.3% of the group had a physical activity index above 64. In other words, only a few of the respondents participated in adequate physical activity to render significant health benefits.

During the last decade, it would seem that employers have realised that the health status of employees can have a direct influence on their productivity and efficiency in the working environment. Employees in general, and particularly those in a managerial position, are constantly subjected to work-related pressures. The manner in which a manager handles these pressures was shown

to be related to his or her physical health status. It is therefore clear that low physical conditioning can be one of the major contributors of low productivity among employees (Cox, 1982; Shephard, 1986; Falkenberg, 1987). The physiological advantages of regular physical exercise, such as an improvement in cardio-respiratory fitness and improved energy levels, also have some psychological advantages. These include improved morale and a positive feeling towards employers, as well as lower levels of anxiety, higher mood state and lower depression (Shephard, 1986).

Not only does physical conditioning have a positive effect on productivity, it also has a direct influence on employee absenteeism (Pretorius *et al.*, 1989). A comparative study between two life insurance companies in Toronto, Canada, revealed a 20% lower absenteeism level in employees from the experimental company who partook in at least two physical workouts per week over a ten-month period, compared to their more sedentary counterparts (Cox 1982). The results of a classical study done by Anderson and Jose (1987) showed that sedentary employees spent 30% more days in hospital than their active counterparts. Studies by Keeler *et al.* (1989) and Pretorius *et al.* (1989) revealed that persons who partook in physical activities of average to high intensity during their free time, visited general practitioners 12% and 8% less, respectively, than their sedentary colleagues. The latter holds certain advantages for a company, such as lower health care costs (Baun *et al.*, 1986; Pretorius *et al.*, 1989).

In an attempt to lower health-related costs of employees and improve productivity, some employers have started implementing a variation of total well-being and worker-support programmes as part of their employee assistance programmes (Seaward, 1988; Uys & Coetzee, 1989; Schwartz, 1989). This phenomenon also began taking shape in South Africa in the 1980's, when the first wellness programmes were implemented in local companies (Strydom *et al.*, 1985).

The implementation of a wellness programme on the work premises can lead to an environment that will inspire the employee to make healthier lifestyle choices (Weinstein, 1987). This opinion is shared by Shephard (1986), who stated that it would be difficult not to be positively influenced when you are encouraged and motivated from within the working environment to adopt a healthier lifestyle. Cox (1982) is of the opinion that an entire wellness programme could be justified in terms of cost, if only a single premature death of an employee in a managerial position could be prevented.

However, companies have started to question the economic viability of running expensive wellness programmes. Very few studies have measured the long-term impact of corporate wellness programmes.

1.2 Aim of the study

The aim of this research was to determine what effect the company's wellness programme would have on modifiable CAD risk factors of 76 male employees in middle- to top managerial positions at Kumba Resources.

1.3 Delimitations

1.3.1 The research only focused on male employees in middle- to top management employed by the company for the entire study period.

1.3.2 Employees who participated in the first fitness and medical evaluation, but did not participate in the second evaluation, did not form part of the study.

1.3.3 Employees who did not wish their data to be made available were not used in this research.

1.3.4 Employees who participated in the programme, but were transferred, retrenched or left the company during the four-year period, were excluded from the study.

1.3.5 Employees who missed the medical evaluations because of the nature of their work (e.g. travelling) were excluded from the study.

1.4 Limitations

1.4.1 Kumba Resources utilised an external company (Careways) to conduct the medical evaluations on its employees and the researcher was thus dependent upon their database for this study. Certain values (i.e. resting pulse, fat percentage and smoking status) were not included in the analysis, since they were missing from the database.

1.4.2 No record was kept on the level and frequency of exercise participation of the subjects and thus the researcher was unable to divide the groups according to their activity levels (i.e. active vs. inactive).

1.4.3 No psychological evaluation data was available to the researcher and this limited/excluded the levels of stress experienced by the subjects (an important CAD risk factor).

1.4.4 Measurements done at Careways weren't always performed by the same professional, and could have resulted in some irregular measurements.

CHAPTER TWO: LITERATURE REVIEW

2.1 Introduction

Cardiovascular disease (CVD) is the leading killer in Westernised society. Some are classified as coronary heart disease (CHD), because they affect the heart muscle and the blood vessels of the heart. Coronary occlusion (heart attack) is a type of CHD. Atherosclerosis and arteriosclerosis increase the risk of heart attack and are considered to be types of CHD. Angina pectoris (chest or arm pain), which occurs when the oxygen supply to the heart muscle is diminished, is sometimes considered to be a type of CHD, although it is really a symptom of poor circulation. Hypertension (high blood pressure), stroke (brain attack), peripheral vascular disease and congestive heart failure are other forms of CVD. Physical inactivity may relate in some way to each of these types of diseases (Corbin *et al.*, 2006).

Cardiovascular disease accounted for nearly 41% of deaths in the United States of America (USA) during 2000, according to the American Heart Association (AHA, 2003). In other words, one in 2.4 Americans die each year from CVD. More than one in five Americans suffer from disorders related to CVD. Coronary artery disease is still the single largest killer of American men and women (about one in every five). The cost of CVD in 2003 was estimated at $ 351.8 billion. These costs include medical treatment and nursing, hospital and nursing home services, medication and loss of productivity resulting from disability. While costs for treatment are spiralling upward, the death rate for these diseases appears to be declining. Advances in medical treatment, education and healthy lifestyle changes can be credited for the declining death rate. However, CVD is still the number one health concern in the USA. On average somebody dies of CVD every 33 seconds, resulting in more than 2 600 deaths each day (AHA, 2003; Robbins *et al.*, 2005).

In South Africa, one in four women and one in three men will have CHD by the time they reach the age of sixty. A total of 48,000 South Africans suffer heart attacks every year. Of these, only 36,000 survive. The most tragic part of these statistics is the age of the victims. Many CHD sufferers are in their most productive, middle-aged years. The famous Framingham study, an ongoing medical study administered by the National Heart, Lung and Blood Institute (NHLBI, Framingham, Middlesex Massachusetts, USA) was initiated in 1948 to examine associations of various genetic, psychological, and behavioural factors with the incidence of myocardial infarctions and sudden cardiac death. The results of this study underscore the fact that some 5% of all heart attacks occur in people under the age of 40, rising to an astonishing 45% in persons under the age of 65 (Gordon & Gibbons, 1990).

2.2 Coronary artery disease

Coronary artery disease (CAD) describes the presence of angina pectoris, myocardial infarction, and the atherosclerotic processes in coronary blood vessels. Coronary artery disease accounts for more than half of all cases of CVD in both sexes. The heart is a muscle that works consistently and never stops beating. Every day, the average heart beats approximately 100,000 times. Besides providing oxygen and other nutrients to all body tissues, the heart supplies itself with oxygen. The heart has a dedicated circulatory system, which nourishes only the heart muscle. The two coronary arteries (right and left) branch off the left aorta and divide into many small arteries, that lie in the heart muscle and feed the heart. The heart requires a steady supply of oxygen-rich blood to function optimally (Robbins *et al.*, 2005).

2.2.1 Atherosclerosis

Coronary artery heart disease is the most common result of atherosclerosis (Robbins *et al.*, 2005), which, in turn, is the leading cause of heart disease in

most Western populations (Ross, 1986). Atherosclerosis contributes to myocardial infraction, stroke, hypertension, angina pectoris, and peripheral vascular disease. There are a variety of causes for this condition, many of which are related to unhealthy life-style choices, such as excessive dietary cholesterol, a diet rich in saturated fat, reaction to perceived emotional stress and nicotine intake from smoking, which may contribute to hypertension and high cholesterol levels (Sharkey, 1997; Robbins *et al.*, 2005). Viral and bacterial infections also have a role to play. The bacterium Chlamydia pneumonia, known primarily as a cause of sexually transmitted disease, cytomegalovirus, which causes respiratory infections, oral bacteria from infected gums and even Helicobacter pylori, the ulcer–causing bacterium, have all been found in vascular lesions. Indeed, researchers suspect that almost any type of chronic infection – including respiratory, periodontal, and urinary tract problems, could set the stage for atherosclerosis (Marieb, 2004). Atherosclerosis of the coronary arteries is particularly harmful. If these arteries become damaged, the blood supply to the heart muscle is diminished, and angina pectoris may occur. Atherosclerosis increases the risk of heart attack, because a fibrous clot is more likely to obstruct a damaged artery than a healthy one (Corbin *et al.*, 2006).

Research conducted at the University of Washington (Seattle) showed that the process of atherosclerosis is initiated by injury to the inside lining (endothelium) of the coronary arteries. The damage enables cells to penetrate the inner lining and burrow into the inner layer of the intima media. These monocytes release chemicals that not only attract more monocytes to the damaged site, but also cause some of the muscle cells of the middle layer of the coronary artery to proliferate and move into the wall's inner layer. Both the monocytes and muscle cells start to fill up with cholesterol and other fats, which results in fatty streaks (Ross, 1986).

In the presence of CAD, risk factors such as smoking, high cholesterol levels, hypertension, obesity and prolonged stress, injury to the endothelium may persist or even grow worse. Many monocytes and muscle cells may accumulate in the

inner layer of the coronary artery walls. In addition, platelets and lipids may attach themselves to the injured endothelium and worsen an already deteriorating situation. As a result, the formation of atherosclerotic plaque occurs. Atherosclerotic plaque is a hard mass containing monocytes, muscle cells, cholesterol and other fats, fibrous tissue, calcium and certain other elements transported by blood. These plaques are covered by an outer lining of cells and are yellowish in colour (Gordon & Gibbons, 1990).

Atherosclerotic plaque may develop even when the damage to the endothelium is not severe enough to permit the entry of monocytes into the inner layer of the coronary artery (Ross, 1986). This author postulates that, in this instance, it is the injured endothelium itself that releases the chemicals which gradually results in atherosclerotic plaque build-up. Again, this process tends to occur only when CHD risk factors such as smoking, high blood cholesterol, obesity, hypertension and prolonged stress are present. Such a build-up of plaque has two major negative effects. Firstly, the coronary artery wall becomes so rigid that it can no longer effectively regulate blood flow and secondly, the artery lumen (diameter of the inside of the artery) is narrowed, thereby reducing the ability of the artery to supply the myocardium with adequate blood and oxygen (Ross, 1986).

Blood lipids are thought to contribute to the formation of atherosclerotic deposits in the inner walls of the arteries (Corbin *et al.*, 2006). The body manufactures many of these blood lipids, whilst others are ingested in high fat foods, particularly those high in saturated fat. There are several different kinds of blood lipids, including lipoproteins, phospholipids, triglycerides and cholesterol. Cholesterol is the most well-known, but it is not the only culprit contributing to atherosclerosis. LDL-C is considered the major contributing factor to the development of atherosclerosis. It is composed of a core of cholesterol surrounded by protein and another substance that makes it water-soluble. People with elevated levels of TC and LDL-C have been shown to be at a higher than normal risk of contracting heart disease. New evidence indicates that there

are subtypes of LDL-C that pose an even greater risk, but these are difficult to measure and are not included in current routine blood tests (Corbin *et al.*, 2006).

In sharp contrast, the findings of a retrospective study of 194 consecutive autopsies performed by three experienced pathologists between 1985 and 1988 at the Providence Veterans Administration Medical Centre revealed that about two thirds of cases with severe atherosclerosis presented none of the usual risk factors such as elevated serum cholesterol, diabetes mellitus or hypertension (McCully, 1990). During this study, cases were divided into four groups according to the severity of atherosclerosis and the cause of death. In the first group, severe atherosclerosis was the cause of death and included those with CAD who had a myocardial infarction, those with congestive heart failure, those with cerebrovascular disease who had a stroke, those with aneurysms and those with peripheral vascular disease who had contracted gangrene. In this group, more than 50% of the arteries were narrowed and 50% of the surface of the aorta was covered with fatty plaque. The second group comprised similar cases with severe atherosclerosis but the cause of death was different including, cancers, cirrhosis, pneumonia/emphysema, infection, pulmonary embolism, or other diseases. The third group comprised cases with mild to moderate atherosclerosis where less than 50% of the arteries were narrowed and less than 50% of the aortic intima was involved with plaque formation. The fourth group comprised cases with no hardening of the arteries or minimum plaque formation. This group had less than 10% narrowing of the arteries.

Clinical records were recovered for each case and relevant data on serum cholesterol, glucose, urea, creatinine, total lipids, high-density lipoprotein, triglycerides and blood pressure was recorded. The number of cases with severe atherosclerosis (groups 1 and 2) was 122, representing 63% of the total number. The number of cases with significant atherosclerosis (group 1 to 3) was 170 (83% of cases) and only 24 cases (12%) had minimal atherosclerosis (group 4). Surprisingly, of all 170 patients with severe or moderate atherosclerosis (groups 1 to 3), 123 cases (72%) had serum cholesterol levels under the

recommended maximum of 5.2 mmol/l. In patients dying as a result of severe atherosclerosis, only 27% had high cholesterol levels of between 5.2 mmol/l and 6.1 mmol/l, and a minute 7% had cholesterol levels greater than 6.2 mmol/l.

A total of 135 out of 147 cases (92%) had neither diabetes nor hypertension, and were shown to have a serum cholesterol level of less than 6.2 mmol/l. In 109 cases (74%) the serum cholesterol was less than 5.2 mmol/l. In those cases with severe atherosclerosis (group 1 and 2), but without diabetes or hypertension, 80 out of 88 cases (91%) were shown to have serum cholesterol less than 6.2 mmol/l. In the total sample of 122 cases representing groups 1 and 2, severe atherosclerotic plaque developed in 80 cases (66%) without any evidence of diabetes, hypertension, or elevated serum cholesterol. This indicates that the greater majority of patients with atherosclerosis did not have high cholesterol (McCully, 1990). These findings certainly contradict the current knowledge that high cholesterol results in the hardening of the arteries, especially when considering that, among patients dying with severe or moderate atherosclerosis, almost three-quarters had a total serum cholesterol level of less than 5.2 mmol/l (normal), and the incidence of individuals with elevated cholesterol levels (5.2 to 6.1 mmol/l) was fewer than 1 in 10 (Buist, 1995).

2.2.2 Angina pectoris

Dr. William Heberden first used the term ‘angina pectoris’ in a report published in 1772. He chose ‘angina’, derived from the Latin root *angere*, which means, “to struggle,” as he wanted to express a sense of strangling (Rutherford *et al.*, 1988). Coronary atherosclerosis involves a localised accumulation of fibrous tissue and, to a lesser extent, lipids within the coronary artery, progressively narrowing the lumen of the vessel. Ischemia occurs when clinically-significant lesions (70% or more of the vessel’s cross-sectional area) result in a blood flow inadequate to meet myocardial oxygen demands. Consequently, significant ST-segment depression, or angina pectoris, or both, occur. When the former occurs without detectable symptoms, it is referred to as silent ischemia. For a given patient,

stable angina is expected to occur with continuous exercise at approximately the same rate-pressure product. In contrast, unstable angina may be characterised by an abrupt increase in the frequency of angina at rest, or with less activity than stable angina. The acceleration of symptoms may be a messenger of an impending cardiovascular event and serves as an absolute contra-indication to exercise. Such patients generally require immediate medical attention (Franklin *et al.*, 2002; Robbins *et al.*, 2005; Corbin *et al.*, 2006).

Angina symptoms vary greatly and an event can last more than five seconds, but usually less than 20 minutes. It is not brought on or worsened by deep breathing, and is not alleviated by changes in posture. An angina attack usually presents a generalised pain that is not restricted to a small area. It gradually intensifies over a couple of minutes, and then slowly subsides when the activity that precipitated it has stopped. An angina attack that occurs while an individual is at rest (often referred to as variant or rest angina) is frequently the result of a spasm of a coronary artery. It occurs when the muscular wall of a coronary artery goes into spasm and occludes the artery temporarily (Gordon & Gibbons, 1990). This typical form of angina is sometimes called *Prinzmetal's angina*, since Dr. Myron Printzmetal offered the first detailed account of it in the American Journal of Medicine (Prinzmetal *et al.*, 1959).

2.2.3 Myocardial infraction

Damage to the myocardium is referred to as a myocardial infarction (MI), which means "death of heart muscle cells". The chest discomfort experienced by an individual during a myocardial infarction can be very similar to the symptoms of angina, except that the feeling lasts longer and is more intense. During a MI, the discomfort often turns into severe pain that may be accompanied by shortness of breath, nausea, dizziness and pallor. Unlike angina, which does not cause permanent damage to the heart, a MI nearly always does (Gordon & Gibbons, 1990). Ninety percent of MI's are caused by a blood clot superimposed on an underlying atherosclerotic plaque on the coronary artery wall. As the plaque

builds up, it disrupts the normal flow of blood. As the blood flow becomes increasingly turbulent, blood platelets coagulate at the site of the plaque obstruction, resulting in the formation of a blood clot. The latter is a gelatinous clump called a thrombus, from which the synonymous term for an MI, coronary thrombosis, is derived. The clot may remain attached to the plaque, or partially brake off to attach elsewhere in the coronary artery. Myocardial infarction occurs when one or more of the coronary arteries is blocked by a blood clot and stifles the blood supply to the heart muscle. Consequently, the portion of the heart muscle beyond the blockage is deprived of oxygen, resulting in injury or death of the relevant portion. If the blocked artery supplies a major portion of the heart muscle, cardiac output will be compromised and death will occur within minutes (Ross, 1986; Gordon & Gibbons, 1990; Robbins *et al*, 2005; Corbin *et al*, 2006).

There are several causes for MI. A coronary spasm could bring about an attack, as it may cut off the blood supply to the heart muscle completely. Coronary spasm increases an individual's predisposition to thrombus formation. Plaque plays a role here as well, since coronary spasm typically occurs at the site of a plaque build-up. Furthermore, heart attacks often occur in individuals who refuse to admit that they are experiencing chest discomfort, pain or shortness of breath. If such individuals continue engaging in physical activity or whatever form of physical exertion, their heart muscles are usually deprived of oxygen, which may result in an attack (Gordon & Gibbons, 1990).

2.2.4 Cerebrovascular accident (CVA)

A stroke occurs when blood flow to the brain is disrupted either by a blockage (ischemic stroke), or by a burst blood vessel (cerebral haemorrhage). Other causes include compression of brain tissue by haemorrhage, or edema and narrowing of brain vessels by atherosclerosis (Marieb, 2004). The brain needs a continuous supply of oxygen-rich blood to function. When a blood clot interrupts the flow of blood to the brain it lacks its essential nourishment, and brain cells perish. Strokes, in turn, are a prominent cause of serious disability and a major

contributor to dementia later in life. They can result in paralysis of one side of the body, loss of muscle control, the ability to speak or understand the speech of others, memory loss and behavioural changes (Sharkey, 1997; Robbins *et al.*, 2005).

The risk of experiencing a stroke increases when an individual is hypertensive. A hypertensive individual is two to four times more likely to experience a stroke than an individual with normal blood pressure. This is because in a hypertensive individual, it would be more likely that a blood clot formed could be transported to the brain where it may cause a blockage to blood flow and, consequently, result in a stroke. Individuals with CAD are therefore also at greater risk. Gender plays an important role, since women are 60% more likely to suffer a stroke than men. Diabetics have double the risk than do non-diabetics, and for each decade after the age of 55, the risk doubles. Race appears to be a major contributing factor to risk, with African Americans suffering almost double the number of fatal strokes than whites in the USA. In these cases, hypertension and diabetes are suspected to be the primary causes. Other life-style factors that may increase the risk of stroke are diets high in fat and cholesterol, alcohol and drug abuse, smoking, sedentary habits, stress and hostility, lots of salt in the diet and obesity (Robbins *et al.*, 2005).

Whatever the cause, fewer than 35% of patients who survive a stroke are alive three years later. Some patients recover at least part of their lost faculties, because undamaged neurons sprout new branches that spread into the injured area and take over some lost functions. Physical therapy is usually started as soon as possible to prevent muscular contractures (Marieb, 2004). In most studies the risk of stroke decreases as physical activity levels increase. However, with more vigorous activity, and possibly heavy lifting, the trend may reverse (Sharkey, 1997).

2.3 Risk factors for coronary artery disease

2.3.1 Introduction

The risk of various behaviours for disease is determined, in part, through epidemiological research, which involves studying large populations to investigate the cause and control of diseases (Robbins *et al.*, 2005). The famous Framingham Study is a classical example of epidemiological research (Gordon & Gibbons, 1990).

Such studies identified several risk factors, some primary, and others secondary, that may lead to the development of atherosclerosis. The more CAD risk factors an individual possesses, the greater his/her chances are of developing CAD. The five primary CAD risk factors that can be controlled are: inactivity, hypertension, dyslipidemia, cigarette smoking and obesity. The seven secondary risk factors are stress, emotional behaviour (especially negative behaviour such as hostility and anger), increasing age, male gender, race and heredity. Recently, emerging additional risk factors that have been identified are Metabolic Syndrome (Syndrome X), elevated homocysteine and C-reactive protein levels (AACVPR, 1999; Jackson *et al.,* 2004; Robbins *et al.*, 2005).

2.3.2 Primary risk factors

2.3.2.1 Dyslipidemia

2.3.2.1.1 Total serum cholesterol

High levels of TC above 5.2 mmol/l and other blood fats have long been associated with the development of arterial plaque, a major cause of atherosclerosis and CAD (JAMA, 1993a). About 80% of TC is synthesised by the liver, whereas 15% comes from dietary sources, mainly from foods of animal

origin (Sharkey, 1997; Marieb, 2004; Robbins *et al.*, 2005). With fat constituting a large percentage of the nutritional consumption of Americans, they are prone to high TC levels (AACVPR, 1999). Westernised South Africans have similar eating habits to Americans, and thus 20% of South Africans have been reported to have TC levels placing them at high risk for CAD and at least a further 60% are at moderate risk (Gordon & Gibbons, 1990).

In spite of the negative connotations linked with cholesterol as the body's enemy, it is actually vital for human existence. It is a crucial substance for healthy cell membranes, brain cells, digestion and functioning of adrenal glands (Robbins *et al.*, 2005).

Cholesterol levels become dangerously high because the human body manufactures most of what it needs, but the modern westernised diet creates an excess. Ninety-five percent of the lipids in the body are in the form of triglycerides, a true fat that is stored in the fat cells and found in the blood. Both high cholesterol and elevated triglyceride levels are known to increase the risk of developing atherosclerosis (Robbins *et al.*, 2005).

When evaluating the blood lipid profile of an individual for risk of CAD, two factors need to be considered. Firstly, the level of TC and triglycerides in the blood and, secondly, the way triglycerides and cholesterol are transported in the bloodstream. The TC level includes the amount of cholesterol carried by the blood and knowledge of these levels provides the individual with a rough estimate of their CAD risk. A lipoprotein analysis provides a more accurate estimate of this risk than checking the TC on its own. This type of analysis separates TC levels into its compartments, or lipoproteins, of which there are two main types. The first, termed low-density lipoprotein or LDL-C (60-70% of TC) is harmful to the arteries and transports cholesterol away from the liver to the body. The second is high-density lipoprotein (HDL-C, 20–30% of TC), which protects the arteries and carries cholesterol back to the liver to be reused if needed. A fine balance needs to be struck between these. A third subtype of cholesterol is

very low-density lipoproteins (VLDL, 10-15% of TC), and is a forerunner of LDL. Certain forms of VLDL, especially VLDL remnants, seem to be atherogenic, on the evidence available (Buist, 1995; AACVPR 1999; Robbins *et al.*, 2005; ACSM, 2006).

2.3.2.1.2 Low-density lipoprotein cholesterol

The role of the LDL-C in the blood is to transport cholesterol to the peripheral tissue, making it available to the tissue cells for membrane or hormonal synthesis and for storage for later use (Marieb, 2004; Robbins *et al.*, 2005). This type of cholesterol is considered harmful as it transports a large amount of cholesterol. The lower density of this lipoprotein allows easy attachment to the inner wall of the blood vessel, thus stimulating the atherosclerotic process. Cigarette smoking, emotional stressors, and diets high in saturated and trans-fats have been shown to increase LDL-C levels (Robbins *et al.*, 2005). Acceptable levels measure bellow 3.5 mmol/l (JAMA, 1993 a).

An elevated LDL-C concentration higher than 3.5 mmol/l is a risk factor for CAD (Castelli, 1990; Castelli *et al.*, 1992; Laakso, 1996; Lehto *et al.*, 1997; Sharkey, 1997; Robbins *et al.*, 2005; Corbin *et al.*, 2006). It is recommended that LDL-C levels be maintained at levels lower than 3.4 mmol/l. Higher levels point towards an increased risk for developing CAD (JAMA, 1993 a). New evidence suggests that some subtypes of LDL-C (characterised by their small size and high density) present even greater risks for contracting CAD. These subtypes are difficult to measure and do not form part of most routine blood tests, but future research is expected to enhance our understanding and quantification thereof (Corbin *et al.*, 2006). Sixty-four percent of white South African civil servants have been diagnosed with elevated LDL-C levels (Jacobs, 1991). These findings were confirmed by Dreyer (1996), who found that LDL-C levels are among the premier CAD risk indicators in male civil servants. The average LDL-C for this group was 4.17 mmol/l, which suggests a high risk of contracting CAD.

The ground-breaking discovery of LDL-C receptors, which are located primarily in liver cells, won Brown and Goldstein the Nobel Prize for Medicine in 1985. These receptors bind and remove cholesterol from the blood, the efficiency of which is determined by the number of receptors present. This number is, to some extent, genetically determined, but is also influenced by lifestyle factors. A diet high in saturated fat and cholesterol not only saturates the receptors, but also decreases their number, which is disadvantageous to the health (Robbins *et al.*, 2005).

Several medical researchers have compiled convincing evidence linking high LDL-C with CAD (Gordon & Gibbons, 1990; Sharkey, 1997; Robbins *et al.*, 2005). Enough data exists to confidently claim that consistently high LDL-C levels alone can cause CAD, even in the absence of other risk factors. These authors base this statement partially on knowledge of individuals who suffer from homozygous familial hyper-cholesterolemia (FH), a rare physiological disorder characterised by a total absence of cell-surface receptors that normally remove LDL-C from the blood. This disorder results in astonishingly high LDL-C levels (as high as 25 mmol/l) and severe atherosclerosis. Children who have been diagnosed with this condition generally develop CHD in, or even prior to, their teenage years. Moreover, individuals with familial hyper-cholesterolemia, the more common heterozygous form, have a reduced number of LDL-C receptors, instead of none, and generally develop CAD in their early thirties or forties. About one in every 75 Americans, or one in a hundred South Africans, who suffers premature heart attacks has been diagnosed with this disorder (Gordon & Gibbons, 1990; Sharkey, 1997; Robbins *et al.*, 2005).

2.3.2.1.3 High-density lipoprotein cholesterol

As a result of the dense structure of the lipoprotein, HDL-C is considered beneficial (Robbins *et al.*, 2005). The main function of HDL-C, which is particularly rich in phospholipids and cholesterol, is to transport cholesterol from peripheral tissue to the liver, where it is broken down and becomes part of the

bile. The liver synthesises the protein envelopes of the HDL-C particles and then discharges them into the bloodstream in a collapsed form. Once part of the circulatory system, these incomplete HDL particles fill with cholesterol originating from the tissue cells and extracted from the arterial walls (Marieb, 2004; Corbin *et al.*, 2006).

In the Framingham epidemiological study (1948 - 1989) of an entire community, a low HDL-C was identified as the best single indicator of CAD risk (Sharkey, 1997). The average HDL-C amongst male South African civil servants was measured at 0.97 mmol/l, which is beneficial, given the positive effect of HDL-C on CAD (Dreyer, 1996). The female sex hormone estrogen tends to raise HDL-C levels, and may explain why pre-menopausal women are usually less prone to heart disease. Levels above 1.8 mmol/l may guard an individual against heart disease, while levels below 0.9 mmol/l place them at a moderate risk, given the positive effect of HDL-C on CAD (Dreyer, 1996). Levels above 1.55 - 1.8 mmol/l may guard an individual against CAD, while levels below 0.9 mmol/l signals an increased risk for CAD (Robbins *et al.*, 2005).

2.3.2.1.4 Total cholesterol / HDL-cholesterol ratio

HDL-C concentration and the ratio between TC and HDL are considered factors that could reduce the risk for CAD dependent on weight reduction (Robbins *et al.*, 2005). According to Dr. William Castelli, the director of the Framingham Heart Study, the best index of heart attack risk is that of TC to HDL-C ratio (American Heart Association, (1987). The acceptable norm for TC/HDL-C is below 3 mmol/l (Bornow, 1992).

The lower the ratio the better, since it indicates that a higher percentage of the TC in the blood is composed of helpful HDL-C, rather than harmful LDL-C (Gordon & Gibbons, 1990).

In summary, if an individual therefore has a TC reading of 5.1 mmol/l with HDL-C at 2.0 mmol/l and LDL-C at 3.1 mmol/l, then they are considered to be at low risk for CAD. However, even if the TC is less than 5.1 mmol/l, when coupled with an HDL-C level below 0.9 mmol/l, the individual is still considered to be at increased risk for CAD (Robbins *et al.*, 2005).

2.3.2.1.5 Triglycerides

Triglycerides are the chemical form in which most fat exists in food, as well as in the human body (also known as free fatty acids). Triglycerides are found in poultry skin, meat and shellfish, but they are mainly manufactured in the liver, from alcohol, starches and refined sugars. These substances are not classified as fats, but they can be converted into fats by the body and are then disposed of in the bloodstream. Triglycerides store excess fats and are carried in the bloodstream by very low-density lipoproteins (VLDL). In combination with cholesterol, triglycerides stimulate the formation of plaque on the blood vessel walls. It is generally accepted that triglyceride levels should be maintained below 1.6 mmol/l (Robbins *et al.*, 2005).

The connection between serum triglyceride levels and CAD is complex. Elevated triglyceride levels in the blood have been positively correlated to CAD in a uni-variant analysis, but a multivariate analysis controlling for other risk factors (such as blood pressure, inactivity and obesity) points towards a reduced effect of triglycerides. Elevated levels of serum triglycerides have also been linked positively to lower levels of HDL-C and small and dense LDL-C particle size. Both these conditions have been linked to an elevated risk of developing atherosclerosis (JAMA, 1993 a). Elevated triglyceride levels may emphasize the need to evaluate the presence of alcohol abuse, diabetes, steroid use, dietary patterns and obesity. Increased physical activity, together with non-pharmacological therapy (e.g. weight loss and alcohol restriction), is recommended for all persons with elevated triglyceride levels. Drug treatment

should be considered when non-pharmacological approaches fail to lower these levels adequately in persons with CAD or a high CAD risk (Franklin *et al.*, 2002).

2.3.2.2 Hypertension

Both the systolic blood pressure (SBP) and diastolic blood pressure (DBP) values are important. Elevated levels of either could mean greater risk of stroke or heart attack. Normal human blood pressure is less than 120/80 mm Hg. Pre-hypertension is defined as blood pressures between 120/80 mm Hg and 139/89 mm Hg. Until as recent as 2003, these were considered within normal boundaries, but are currently considered unsafe and calls for lifestyle changes and monitoring. Hypertension is evident at a measurement of 140/90 mm Hg or above, and requires medical intervention (Institute for Aerobic Research, 1994; Robbins *et al.*, 2005). About 50 million Americans, aged 6 years and older, have high blood pressure (AHA, 1995). In South Africa there is similar reason for great concern, since hypertension is a significant problem. Black people in particular are apparently inclined towards this disorder. Unfortunately the local situation is not as well researched as in the USA. Public awareness campaigns are therefore largely lacking (Gordon & Gibbons, 1990).

Chronic hypertension, often called the 'silent killer', is a dangerous and prevalent disease that causes increased peripheral resistance to blood flow. It affects approximately 50 million USA citizens and about one billion individuals worldwide (ACSM, 2006). Although hypertension is usually asymptomatic for the first 10 to 20 years, it gradually, but persistently, causes strain on the heart and damages the arteries. Prolonged hypertension is the single major cause of heart failure, vascular disease, renal failure and stroke. Typically, the myocardium enlarges over time, because the heart works harder than normal as it is forced to pump against increased resistance. When finally strained beyond capacity to respond, the heart weakens and its walls become flaccid. Hypertension also destroys blood vessels by creating small lesions in the endothelium, which hasten the progressive hardening of the arteries and, ultimately, cause arteriosclerosis.

Impaired blood flow to body tissues is caused by the blockage of the blood vessels and vascular complications eventually appear in the brain vessels, retinas, heart and kidneys. Although hypertension and atherosclerosis are often linked, it is difficult to link hypertension to any distinct anatomical pathology (Marieb, 2004).

Several factors have been shown to contribute significantly to the development of hypertension in humans (Franklin *et al.*, 2002). The most important of these include unhealthy diet, obesity, hereditary factors, race, gender, stress, smoking, diabetes, elevated TC levels, alcohol abuse, use of oral contraceptives, and sedentary lifestyles (Byrne, 1991; JNC VI, 1997; Franklin *et al.*, 2000; Robbins *et al.*, 2005; Corbin *et al.*, 2006).

Examining some of these factors more closely, the diet of an individual plays an important role in the onset of the condition, especially excessive consumption of sodium, saturated fat and cholesterol. A deficiency in certain metallic ions such as potassium, calcium, and magnesium is also a contributing factor. Obesity also plays a major role in individuals with persistent hypertension, because the total length of an obese person's blood vessels is, relatively, greater than that of individuals with a normal body composition. For each kilogram of fat, 3.2 km of additional blood vessels are required to support the excess tissue. Beyond these two factors, heredity plays a major role. Offspring of a hypertensive parent are twice as likely to develop high blood pressure as those whose parents had normal blood pressure. Studies in the US have indicated that hypertension is more prevalent in African Americans and Hispanics than Asians, American Indians and white Americans. There may also be some gender issues. Up to the age of 55, men have been shown to be at greater risk of hypertension than women are, but after age 74, the situation reverses and women run a greater risk (Byrne, 1991; JNC VI, 1997; Franklin *et al.*, 2002; Robbins *et al.*, 2005; Corbin *et al.*, 2006).

2.3.2.3 Smoking

Despite the governmental health warnings, which by law appear on every cigarette packet, thousands of South Africans still persist with the smoking habit. Nonetheless, the majority of smokers are aware of their increased risk of developing lung cancer. This often terminal disease forms only part of the problem, since many individuals die from heart disease that can be directly related to the smoking habit. A study conducted by the Medical Research Council of South Africa revealed that smoking-related deaths in the population are distributed as follows: Whites (34%), Coloureds (14.5%), Asians (24.5%) and Blacks (3.9%). In addition, smokers are at a greater risk of not surviving a heart attack than non-smokers. Individuals who survive either a heart attack or bypass surgery, but continue to smoke, appreciably increase their chances of dying soon thereafter (Gordon & Gibbons, 1990). Data from numerous studies reveals that quitting smoking is beneficial and results in increased oxygen-carrying capacity of the blood, lower blood pressure, enhanced night vision and improved effectiveness of prescription drugs (Sharkey, 1997).

2.3.2.3.1 Pathophysiology associated with smoking

Smoking reduces the HDL–C concentration and elevates the white blood cell count in the blood stream. The latter phenomenon may, independent of smoking, predict a heart attack. Smoking is also responsible for changes in the blood composition and enhances the tendency to form clots that may bring about a heart attack or stroke. Cigarette smoke contains nicotine and carbon monoxide, two harmful substances that are highly detrimental to a person's health. Nicotine causes an acceleration of the heart rate (by 15 - 20 beats/min) and an increase in SBP (by 10 - 20 mm Hg). Nicotine increases the body's requirement for oxygen by stimulating the release of adrenaline-like substances that, in turn, cause constriction of the blood vessels, and increase the blood pressure.

Furthermore, nicotine increases the heart's susceptibility to ventricular tachycardia, or fibrillation, a life-threatening rhythm disturbance that is especially prevalent in smokers. The carbon monoxide level in the blood of smokers is ten times higher than in non-smokers. This component of tobacco binds to the oxygen-carrying haemoglobin in the blood and consequently diminishes the effectiveness of the oxygen supply to the body tissues by about 20% (Gordon & Gibbons, 1990).

The cholesterol metabolism of ten smokers who consumed at least 20 or more cigarettes per day, was compared with 10 non-smokers who formed a control group. All subjects were healthy 20- to 40-year-old males with no familial history of atherosclerotic vascular disease. The results of blood tests taken after an overnight fast were compared to the control group. The smokers, without exception, all displayed abnormal cholesterol metabolism, as indicated by decreases in apolipoprotein A1 (a major component of HDL), cholesterol transport from the cell membranes to blood plasma, and in cholesterol transfer to LDL and VLDL. These results are consistent with an abnormal cholesterol metabolism, characteristic of patients with atherosclerotic vascular disease (AVD) and possibly accounts for the high incidence of CAD among smokers (Buist, 1995).

2.3.2.3.2 Passive smoking

Passive smoking is defined as smoke inhaled by non-smokers from environmental air. Such 'second-hand' smoke has four major negative effects on non-smokers, which include an increased risk of heart and vascular damage, even though much lower doses of toxins are absorbed. This is because smokers develop compensatory responses to some of the adverse cardiovascular effects of cigarette smoking, but non-smokers do not benefit from these responses. The cardiovascular system is tremendously sensitive to some chemicals present in second-hand smoke, such as carbon monoxide, nicotine and hydrocarbons. The former damages the smooth inner lining of blood vessel walls and consequently

accelerates atherosclerotic build-up of plaque. Low levels of second-hand smoke increase the stickiness of blood platelets in non-smokers, which increases the risk of a clot formation in the narrow arteries that could ultimately result in a heart attack. Prior exposure to passive smoke intensifies the damage caused when a heart attack occurs. Non-smokers living with smokers have a 58% to 91% higher risk of dying from heart disease than those who are not. Moreover, smoking in the workplace affects the non-smoker in the same way as if they were smoking two to three cigarettes per day (Levine & Kidd, 1985; Robbins *et al.*, 2005).

2.3.2.4 Physical inactivity

Physical inactivity is considered an important risk factor for developing hypertension, high levels of TC and obesity. It has been causally linked to atherosclerosis and CAD in several prominent studies conducted in the USA, including the USA Surgeon General's Report on Physical Activity and Health (1996), which reviewed hundreds of studies relating exercise to heart disease (Sharkey, 1997; Corbin *et al.*, 2006). Occupational research relating physical inactivity to heart disease in the USA revealed a high incidence in individuals involved exclusively in sedentary work contributing to 34% of deaths, at an annual medical expenditure of $5.7 billion (Sharkey, 1997). Sedentary living was recently promoted from a secondary to a primary risk factor for CAD, comparable to hypertension, high cholesterol, obesity and cigarette smoking, given that inactivity increases risk in numerous ways and many adults are considered vulnerable (AHA, 2003).

The number of forthcoming epidemiologic studies that support the view that both a physically-active lifestyle and a moderate- to high level of cardio respiratory fitness independently lower the risk for various chronic diseases is consistently rising (Blair, 1993). It is consistently reported in the literature that inverse dose-response relationships for physical activity or cardio-respiratory fitness are most important for all-cause and cardiovascular mortality. However, lower incidence

rates for hypertension, obesity, cancer of the colon, type 2 diabetes and osteoporosis have also been mentioned. The majority of the epidemiological evidence that promotes the health benefits of a more active lifestyle has been founded on studies using single assessments of physical activity or fitness. However, results from recent studies correlating a rise in physical activity in initially sedentary, unfit adults, with subsequent reductions in mortality, support the hypothesis that regular activity increases longevity (Paffenbarger *et al.*, 1994; Blair *et al.*, 1995). Owing to the multifactorial nature of chronic diseases associated with a sedentary lifestyle, it is unlikely that a single minimal amount of physical activity that yields protection from all of these diseases will be determinable (Blair, 1993). Further research is needed to define the minimal threshold required for primary prevention of different chronic diseases.

The United States Department of Health and Human Services Report on Physical Activity and Health (1996), states that significant health benefits can be obtained by including a moderate amount of physical activity (e.g. 30 min of brisk walking, 15 min of running, or 15 min of playing volley ball) on most, if not all, days of the week. Additional health benefits can also be obtained through greater amounts of physical activity. People who can maintain a regular regimen of activity that is of longer duration or of more vigorous intensity are likely to derive greater benefits.

Implicit within this report is the notion that the health and fitness benefits associated with physical activity most likely follow a dose-response relationship. In other words, some activity is better than none, and more activity, up to a point, is better than less. Although the optimal dose of physical activity has yet to be defined, the dose-response relationship between physical activity and health benefits clearly support the need for at least moderate amounts of moderate daily physical activity. Moderate daily physical activities are those that are approximately 3 to 6 metabolic equivalents METS, or the equivalent of walking 4.8 to 8 km/h for most healthy adults (Pate *et al.*, 1995). The public health benefits from higher physical activity within the general population would be

enormous, due to both the high prevalence of sedentary lifestyle and the impact that physical activity has on disease risk (Hahn *et al.*, 1986).

To promote this message, the Centres for Disease Control and Prevention (CDC) and the ACSM (2006) recommended that every adult should partake in 30 minutes or more of moderate-intensity physical activity every day, (or at least on most days). Those who follow the recommendation will experience many of the health-related benefits of physical activity and will eventually be able to achieve higher levels of fitness (Pate *et al.*, 1995).

2.3.3 Secondary risk factors

2.3.3.1 Diabetes mellitus

According to the American Diabetic Association (ADA), 17 million people in the USA have diabetes. Unfortunately, 5.9 million of those are not aware of the fact, and an estimated further 10 million are pre-diabetic (Robbins *et al.*, 2005; Corbin *et al.*, 2006).

Diabetes mellitus is a group of metabolic diseases characterised by hyperglycaemia resulting from defects in insulin secretion, insulin action, or both. The chronic hyperglycaemia of diabetes is associated with long-term damage, dysfunction, and failure of various organs, especially the eyes, kidneys, nerves, heart, and blood vessels (Diabetes Care, 1997). Most cases of diabetes fall into two broad etiopathogenetic categories. In one category (type 1 diabetes), the cause is an absolute deficiency of insulin secretion. In the other, much more prevalent category (type 2 diabetes), the cause is a combination of resistance to insulin action and an inadequate compensatory insulin secretion response (Diabetes Care, 1997; Robbins *et al.*, 2005).

2.3.3.1.1 Pathophysiology of diabetes mellitus

Diabetes mellitus results from hypo-secretion or hypo-activity of insulin. When insulin activity is absent or deficient, the levels of blood sugars remain high after a meal because glucose is unable to enter most tissue cells. Ordinarily, when blood sugar levels rise, hyperglycaemic hormones are not released, but when hyperglycaemia becomes excessive, the person begins to feel nauseous, which precipitates the fight or flight response. This results, inappropriately, in all the reactions that normally occur in the hypoglycaemic (fasting) state to make glucose available: glycogenolosis, lipolysis, and gluconeogenesis. Thus, the already high blood glucose levels rise even higher, and excess glucose is lost in the urine (Marieb, 2004).

When sugar cannot be used as cellular fuel, more fats are metabolised. In severe cases of diabetes mellitus, blood levels of fatty acids and their metabolites (acetoacetic acid, acetone, and others) rise dramatically. The fatty acid metabolites, collectively called ketones or ketone bodies, are strong organic acids. When they accumulate faster than they can be used or extracted, the blood pH drops, resulting in a condition called ketosis. Severe ketosis is life threatening (Marieb, 2004).

The three cardinal signs of diabetes mellitus are polyuria, polydipsia and polyphagia. When there is excessive glucose in the kidney filtrate, water is prevented from being reabsorbed by the kidney tubules, resulting in polyuria, excessive urine output that leads to decreased blood volume and dehydration. Serious electrolyte losses occur because of the need to rid the body of excess ketones. Since ketone bodies are negatively charged, they carry positive ions out with them. As a result, sodium and potassium ions are also depleted from the body. This electrolyte imbalance may cause the person to feel abdominal pains, and they may vomit, spiralling the stress reaction higher. Dehydration stimulates hypothalamic thirst centres, causing polydipsia, or excessive thirst. The final cardinal sign, polyphagia, refers to excessive hunger and food

consumption, a sign that the person is 'starving in the land of plenty'. Although plenty of glucose is available, it cannot be used, and the body starts to utilise its fat and protein stores for energy metabolism. Heredity factors play an important role in many cases of diabetes mellitus (Marieb, 2004).

2.3.3.1.2 Type 1 diabetes mellitus

In insulin-dependent diabetes mellitus (IDDM), also known as type 1 or juvenile onset, the pancreas produces no or little insulin. The diabetic must receive insulin injections every day to stay alive and must carefully watch his or her diet and exercise regularly. Symptoms develop rapidly, usually within a period of months, or even weeks. Approximately one million Americans have type 1 diabetes (Robbins *et al.*, 2005).

Type 1 diabetes is one of the most chronic childhood illnesses, annually affecting 18 - 20 children out of 100 000 in the United Kingdom (Onkamo *et al.*, 1999). Although most of the attention has been focused on the increase of type 2 diabetes, a parallel rise in type 1 diabetes has occurred. Type 1 diabetes has always been known as a disease of childhood, but more recent epidemiological studies have indicated that the incidence is comparable in adults (Molback *et al.*, 1994). The EURODIAB collaborative study, a registry involving 44 countries in Europe, indicates an annual rate of increase in incidence of type 1 diabetes of 3-4%, with a larger increase in some central and eastern European countries (EURODIAB, 2000).

2.3.3.1.2.1 Environmental factors

Two major hypothesis exist that may account for the increase in type 1 diabetes. The first hypothesis is that an environmental agent such as a virus may account for this. Seasonality, increasing incidence and epidemics of type 1 diabetes, as well as many cross-sectional and retrospective studies, suggest that certain viruses and some aspects of early childhood diet may influence the risk for type 1

diabetes. Many associations with various environmental triggers have been found in Type I diabetes, but so far only congenital rubella syndrome has been conclusively associated with the disease (Hyoty & Taylor, 2002).

The second hypothesis, under the rubric of the "hygiene hypothesis" indicates that environmental factors can also inhibit the development of autoimmunity. As an oversimplification, our environment for young infants is far to clean, leading to a deficiency in immunoregulation such that th 2 diseases (for example, asthma) and th 1 diseases (for example type 1 diabetes) are increasing dramatically (Gale, 2002; Bach, 2002).

2.3.3.1.2.2 Pathogenesis of type 1 diabetes mellitus

The hallmark of type 1 diabetes is the selective destruction of insulin-producing cells in the pancreas, or insulitis. Studies measuring the expression of diabetes-related auto-antibodies in young children from birth suggest that the appearance of these markers is a major risk for the future development of type 1 diabetes (Yu *et al.*, 2000). However, the role of auto-antibodies in the actual pathogenesis of type 1 diabetes has not been established in humans. In fact, a recent case report showed the development of type 1 diabetes in a patient with X-linked agammaglobulinaemia, suggesting that auto-antibodies are not needed for either the initiation or the progression of type 1 diabetes (Martin *et al.*, 2001). In general, type 1 diabetes is considered primarily a T-cell mediated disease, and extensive evidence exists in humans and mice to support this. Examination of islet tissue obtained from pancreatic biopsy from patients with recent onset type 1 diabetes confirms insulitis, with the presence of an infiltrated composed CD4 and CD8T lymphocyte, B lymphocyte and macrophages, suggesting that these cells have a role in the destruction of the *B* cells (Imagawa *et al.*, 1999).

2.3.3.1.3 Type 2 diabetes mellitus

The more common form of diabetes is non-insulin-dependent (NIDDM), also known as type 2 diabetes, or adult onset, in which the pancreas produces insulin, but either the amount is released insufficiently, or the body is unable to properly use what is available (Robbins *et al.*, 2005). In some individuals with type 2 diabetes, adequate glycaemic control can be achieved with exercise training and weight control. Oral hypoglycaemic agents are needed to achieve adequate glycaemic control in other individuals with type 2 diabetes who do not require insulin. However, there are individuals with type 2 diabetes who have some residual insulin secretion, but still require exogenous insulin for adequate glycaemic control (Kriska *et al.*, 1994; Blair *et al.*, 1995; Diabetes Care, 1997).

Many people know their blood pressure and cholesterol levels, but few know their glucose levels. A substantially-elevated glucose level is the chief diagnostic sign for diabetes. Unfortunately, far too few people are properly tested. As a result, researchers say that nearly half the estimated 17 million people who have type 2 diabetes in the USA are unaware of the fact (Corbin *et al.*, 2006).

The onset of type 2 diabetes is gradual, which means that the disease may go undetected for years. This period could be as long as 10 to 12 years, during which the disease has attacked the patient's vision, injured their kidneys and nerves and set the stage for heart disease. Diabetes seriously increases the risk of developing cardiovascular disease.

More than 80% of people with diabetes die from some form of heart or blood vessel disease. Part of the reason for this is that diabetes effects cholesterol and triglyceride levels by producing a different kind of LDL-C that is even worse for the arteries than ordinary LDL (Robbins *et al.*, 2005).

One condition shared almost universally by type 2 diabetics is obesity. Not all obese people become diabetic, but 90% of people with type 2 diabetes are

overweight or obese. The surge in youth obesity has paralleled a rise in type 2 obesity. At one stage type 2 diabetes was almost unheard of in children; they almost always had type 1 diabetes. A new advisory from the American Academy of Paediatrics and the American Diabetic Association (ADA) calls for diabetes testing in overweight children with any other two risk factors starting at the age of 10, or at puberty, if it comes earlier (Corbin *et al.*, 2006).

Detecting only a minimally-elevated glucose level has become important too, according to the guidelines issued by the ADA. This is because it signals a metabolic disorder called insulin resistance, which effects up to 30% of adults (Robbins *et al.*, 2005). Insulin resistance is a condition associated with being overweight, hypertensive, low HDL-C levels, and elevated triglycerides and blood glucose (Sharkey, 1997).

According to the ADA's new guidelines, all people should, from the age of 45, have their fasting blood glucose level tested at least every three years. However, they also advise that several groups of people are at greater risk and should be checked for diabetes at least once a year. The testing should start at the age of 35 if they are overweight and with extra belly fat, have a brother, sister or parent with belly fat, are not white, or had a baby weighing more than 9 pounds, or had gestational diabetes (diabetes during pregnancy). Earlier testing is also recommended when the individuals have HDL-C levels of 0.9 mmol/l or less, or a triglyceride level of 2.8 mmol/l or more, have hypertension, or take anti-hypertension drugs, or have had a minimally-elevated glucose level on a previous test (Robbins *et al.*, 2005).

Two readings of 6.9 mmol/l and higher on fasting blood-glucose tests taken on different days means you have diabetes. Less elevated readings between 6.1-6.8 mmol/l indicate impaired fasting glucose, which means the individual is insulin-resistant and faces a sharply-increased risk of diabetes (Robbins *et al.*, 2005).

2.3.3.2 Stress

It is difficult to give a succinct definition for stress, but Selye (1997), the Montreal-based author of the book *Stress Without Distress,* perhaps comes closest when he says that it's "the non-specific response of the body to any demand made upon it". In other words, stress is the body's natural and necessary reaction to challenge. In 1914, Dr. Walter B. Cannon, a Harvard University physiologist, labelled this state of arousal the "fight or flight" response. The phrase stuck, because it aptly describes a living organism's exquisite ability to respond, appropriately and instantaneously, to physical threats (Cannon, 1932).

Robbins *et al.* (2005) refer to Dr. Hans Selye in modern literature, as he describes the manner in which we react to stress as either good (*eustress)* or bad (*distress)*. In both cases, the physiological response is the same. In the case of eustress, during pleasant events, health and performance improve even if the stresses increase. Distress, in contrast, is where, during unpleasant events (failing an exam, or the break-up of a relationship), health and performance start to decline. Optimal stress is a point where eustress and distress are intense enough to motivate and physically prepare an individual to perform optimally, yet not enough to cause the body to overreact or sustain harmful effects. Positive stress gives a feeling of control, whilst negative stress causes the opposite feelings.

2.3.3.2.1 Sources of stress

There are many kinds of stressors. These fall into various categories. Firstly, environmental, which includes heat, noise, and terrain. Secondly, physiological, which includes drugs, caffeine, tobacco, injury, infection or disease, and physical effort. The third variant, emotional stressors are the most frequent and important stressors that affect humans. Sometimes, they are referred to as psychosocial stressors. These include life-changing events, such as a change in work or

working hours, family illness, problems with superiors and increased responsibilities (Corbin *et al.*, 2006).

2.3.3.2.2 Hormonal response to stress

All humans have the same type of physiological system for responding to stress, leading to certain commonalities in experience. The sympathetic response of the autonomic nervous system (ANS) is associated with a fight-or-flight response, which mobilize bodily resources when a stressor is identified.

Sometimes this alarm reaction of the body is essential to survival, but, when evoked inappropriately or excessively, it may be more harmful than the original stressor. After the initial sympathetic response, the parasympathetic response takes over in an attempt to restore homeostasis and conserve resources.

In addition to the ANS response, several hormones are secreted by the hypothalamus, pituitary and adrenal glands. The endocrine system is slower to respond than the ANS, and takes longer to return to baseline functioning after a stressful event. When presented with a stressor, the hypothalamus produces corticotrophin-releasing hormone (CRH), and the pituitary gland secretes adrenocorticotropic hormone (ACTH). The introduction of ACTH leads to the release of cortisol by the adrenal cortex. Cortisol is often referred to as the 'stress hormone'. After exposure to chronic stress, the glucagon system can become deregulated (Sharkey, 1997; Corbin *et al.*, 2006).

2.3.3.2.3 Harmful effects of stress

When stressful events persist or recur frequently, as in chronic stress, it can result in a variety of physical illness symptoms. Excessive stress, over a long period, can cause an individual to lose the ability to "calm down" physiologically.

Chronic stress causes the 'wear and tear' that result in the over-activity or under-activity of the allostatic system. This can also damage the immune system. Chronic stress is known to be linked to the development of insulin resistance (a risk factor for diabetes), as well as hypertension and coronary heart disease, osteoporosis, premature aging, asthma and other disorders. It may even promote cancer. Excessive chronic stress contributes to a number of psychological disorders, including depression, anxiety, phobias and addictions. Behaviour manifestations of excess stress include disordered eating, excessive drinking, irritability, insomnia and compulsive behaviours, such as nail biting. It is believed that the two most important causes of heavy allostatic load are a sense of isolation (lack of social support) and a perceived lack of control (Robbins *et al.*, 2005).

2.3.3.2.4 Stress management

Although "being stressed" is typically equated to "being anxious," it is also associated with a variety of emotional correlates, including anger, alarm, lack of control, vulnerability and depression (Sotile, 1998). Although exercise may help to alleviate feelings of distress and mild depression in some people, it does not consistently improve measures of anxiety and depression after an acute cardiac event, since it is a sole intervention (Blumenthal *et al.*, 1988).

However, training in behavioural modification, stress management and relaxation techniques, with or without concomitant exercise therapy, have been shown to be effective in lowering levels of self-reported emotional stress and in modifying Type-A behaviour (Wenger, 1995).

Stress management techniques aimed at attenuating physiologic responses like heart rate and blood pressure, inducing an improved sense of well-being, modifying behaviour patterns (e.g. responding rather than reacting), enhancing coping mechanisms and promoting positive thinking, have become increasingly popular. Effective programmes to facilitate these objectives may include

methods to avoid stressful situations, adjustment and/or adaptation techniques to cope more appropriately with stressors, and using relaxation or biofeedback to neutralise physiologic reactions to stress (Shwartz, 1987).

How one reacts to stress seems to be the critical factor. The best specific stress-management technique remains elusive, and no single intervention has been universally accepted. Most people can be trained easily in relaxation therapy, but other stress management techniques may require more intensive instruction by a behavioural therapist. Although a variety of techniques are commonly promulgated for relaxation training, including yoga, self-hypothesis, progressive muscular exercises, and biofeedback, there is no convincing evidence that one form of therapy is more effective than another (Sotile, 1996). Group stress-management programmes are offered by many hospitals, universities, health promotion programmes, and private practitioners. However, participants with emotional disorders should be referred for more intensive counselling by a licensed psychiatrist, psychologist, or social worker (Franklin *et al.*, 2002).

2.3.3.3 Family history

A family history of heart disease increases an individual's risk of developing CAD. Tendencies toward high blood pressure, stroke, peripheral blood vessel disease, rheumatic fever, high blood lipid levels, obesity and early heart attack appear to be, to some extent, hereditary (Gordon & Gibbons, 1990; Robbins *et al.*, 2005). It is estimated that human biology or heredity accounts for 16% of all health problems, including early death (Corbin *et al.*, 2006). The Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults stated that myocardial infarction, coronary revascularisation, or sudden death before the age of 55 in a father or other male first-degree relative (i.e. brother), must be seen as a CAD risk factor. The same applies for those conditions before the age of 65 years in a mother or any first-degree female relative (i.e. sister) (JAMA, 1993 a).

2.3.3.3.1 Family lifestyle

There is no doubt that genetic factors are a major factor in heart disease. Indeed, according to Goldstein and Brown (1988), the risk of dying from heart disease is five to seven times greater in an individual with a close relative who died from a heart attack before the age of sixty. Family ties, however, seem to be only partially accountable for the unhealthy personal habits in which some coronary heart disease patients and other individuals engage. Examples of such habits are smoking, inactivity, the excessive use of salt (which exacerbates hypertension) and the consumption of rich foods (which drives up blood lipid levels and may add extra kilograms). While these habits may have been picked up by mimicking relatives and other role models, this is not what is meant when family history is listed as an uncontrollable risk factor for CHD (Gordon & Gibbons, 1990; Robbins *et al.*, 2005; Corbin *et al.*, 2006).

Studies have shown that this statement regarding family history needs clarification. The studies listed above indicate that this alarming increase in risk can be contributed to the influence of genetic factors in the presence of such lifestyle factors as cigarette smoking, high cholesterol, hypertension, obesity and diabetes (Gordon & Gibbons, 1990).

Khaw and Barette-Conner (1986) estimated that 68% of deaths where there is a family history of heart attacks are attributable to the interaction between cigarette smoking and genetic factors. Having considered other modifiable risk factors, they concluded that family history itself has little effect on heart disease risk. They suggested that the CAD risk appears to be profoundly affected by modifiable behaviour and not by an inherited predisposition.

Such behaviour can be seen in the study by Kate *et al.* (1984), which shows that wives of heart disease patients are at greater risk for heart disease than those whose husbands' hearts are healthy. The reason for this is that men and women whose lifestyles resemble each other are more likely to marry each other. In

addition, people who are close to each other are likely to mimic one another's habits, good or bad, during the years of marriage.

2.3.3.3.2 Management of heredity factors

Should an individual learn about their family history, they would be more able to overcome negative predispositions and take advantage of positive ones. On discovering that members of their family have had specific diseases or health problems, the individual can inform their physician and so investigate the issue in their own lives. The individual would also be in a position to then take action to diminish risk factors for which there is a predisposition, for example type 2 diabetes, by partaking of regular exercise, eating well, and by keeping body composition under control (Corbin *et al.*, 2006).

2.3.3.4 Obesity

Obesity may be caused by improper diet, overeating, hormonal imbalances, genetic factors and lack of physical activity (Heyward, 1997). According to the third National Health and Nutrition Examination Survey (NHANES III), which ended in 1994, nearly 100 million US adults were overweight or obese (Kuczmarski *et al.*, 1997). According to this survey, 33.4% of Americans are overweight, representing an increase from 25% found in the earlier 1976-1980 survey (Kuczmarski *et al.*, 1994). According to the AHA (1995), the prevalence of obesity has increased by 36% since the early 1960s and by 61% since 1891 (Mokdad *et al.*, 2001). According to Robbins *et al.* (2005), 65% of Americans are now obese or overweight. Weight control is clearly an important goal for US adults. At any given time in the US, 44% of women and 29% of men reported that they are attempting to lose weight, and 5 million of these adults have used prescription drugs to treat obesity (Serdula *et al.*, 1999 b; Kahn *et al.*, 2001). Despite the public awareness of the health hazards associated with overweight and obesity, recent reports from the Centre for Disease Control and Prevention

indicate that the prevalence of overweight and obesity in the US continues to increase (Mokdad *et al.*, 2001).

The prevalence of overweight among young Americans is also on the rise; more than one in every five children and adolescents are overweight (Troiano *et al.*, 1995). Childhood obesity has nearly tripled in the USA since the 1970's (Robbins *et al.*, 2005). Added together, obesity and physical inactivity account for approximately 9.4% of USA health-care expenditures (Colditz, 1999) and each year, an estimated 300 000 USA adults die from obesity-related causes (Allison *et al.*, 1999; Robbins *et al.*, 2005).

At both individual and population levels, strategies that improve nutrition and increase physical activity are fundamental to the control of the epidemic of overweight and obesity (National Institute of Health, 2000; Liu and Manson, 2001 a). Yet, the long-term efficacy of any specific dietary approach to weight control remains to be determined (World Health Organization, 1998; Willet, 2002; Liu & Manson, 2001b).

In some sectors of the white population (both sexes), and in urban black women, obesity rates are excessively high. It is also high in the Indian and Coloured populations, especially in females (Gordon & Gibbons, 1990).

2.3.3.4.1 Body mass index and obesity

Obesity may be classified as having a Body Mass Index of 30.0 kg/m^2 or more, and it is functionally defined as the percentage of body fat at which disease risk increases (Franklin *et al.*, 2002). The BMI is used to assess weight relative to height and is calculated by dividing body weight in kilograms by height in metres squared (kg/m^2). Obesity-related health problems increase beyond a BMI of 25 for most people. The ACSM (2006) lists a BMI of 25 – 29 kg/m^2 for overweight and a BMI of greater than, or equal to, 30.0 kg/m^2 for obesity.

2.3.3.4.2 Obesity and coronary artery disease

Before 1998, obesity was considered to be a secondary risk factor for heart disease. The reason for this was that the effects of obesity were thought to be mediated by other risk factors such as high blood pressure and lipids (Corbin *et al.*, 2006). Brookes and Fahey (1984) stated that, in the absence of other serious risk factors such as hypertension and diabetes, obesity seems to pose a much lower risk to overall mortality. However, there exists the possibility that obesity may be the cause of hypertension and diabetes and thus be vitally linked with CAD. Researchers found that hypertension is twice as common in overweight people as it is in those with normal weight, and three times more than in those who are underweight. Owing to the mounting evidence, the AHA classifies obesity as a primary risk factor, along with hypertension, high blood lipids, tobacco use and sedentary living (Corbin *et al.*, 2006).

Health hazards related to obesity include some types of cancer and gall bladder disease, high blood pressure, impaired glucose tolerance (the precursor to diabetes), a cholesterol ratio high in the bad LDL-C and low in HDL-C, and elevated triglyceride and VLDL-C levels. Most of these hazards listed here are risk factors for CAD (Gordon *et al.*, 1977; National Institute of Health, 2000; Robbins *et al.*, 2005; Corbin *et al.*, 2006).

Obesity is often found in conjunction with diabetes. There has been an increase in the number of cases of type 2 diabetes, paralleling the increased obesity rates in the USA, where studies show that 90% of people with type 2 diabetes are overweight. This combination of diabetes and obesity can lead to blindness, kidney failure and cardiovascular disease. Similar links have been made between hypertension and obesity. Hypertension is nearly six times higher in overweight people aged 20-44, and high cholesterol levels are twice as frequent in the obese (Robbins *et al.*, 2005).

2.3.3.5 Waist-to-hip-ratio

Body fat located at the core of the body is referred to as central or visceral fat. Visceral fat is located in the abdominal cavity, as opposed to subcutaneous fat, which is located just under the skin (subcutaneous). Although subcutaneous fat can be used to measure body fatness, it is not a good indicator of visceral fatness. A useful indicator of fat distribution is the waist-to hip ratio (WHR). The risk for CAD in men increases at WHR of above 0.95 (ACSM, 2000; ACSM, 2006).

A high waist circumference relative to hip circumference yields a high ratio, and is indicative of elevated levels of visceral fat (Corbin *et al.*, 2006). Such individuals with more fat distributed on the trunk, especially abdominal fat, are at an increased risk of hypertension, type 2 diabetes, hyperlipidemia, CAD and premature death when compared to individuals who are as fat, but whose fat deposits are located on the body extremities (Sharkey, 1997; Bray & Popkin, 1998; Robbins *et al.*, 2005).

2.3.3.6 Waist circumference

The waist circumference (WC) alone can be used as an indicator of health risk, because abdominal obesity is a pertinent issue (Franklin *et al.*, 2000). Increasing evidence shows that abdominal adiposity has a direct influence on health, and that visceral fat correlates with health risks to a greater extent than does adipose tissue in other regions of the body (Wang, 2003). The third National Nutrition and Health Examination survey recently reported that waist circumference is more closely linked to CAD risk than BMI (Zhu *et al.*, 2002). The ideal waist circumference for men is less than 100 cm (ACSM, 2006).

The results presented in an article by Kahn and Valdez (2003) provide the first irrefutable evidence that waist circumference is a reliable risk indicator for the syndrome of lipid over accumulation, as documented by elevated fasting

triglyceride concentration and by accelerated mortality after middle age in a large population with wide age and BMI ranges. The results shown by Kahn and Valdez (2003) serve to validate the potential usefulness of waist circumference in physical examinations for clinical purposes. Therefore, their results should encourage physicians to include waist circumference measurement in the evaluation procedure that they use in their routine clinical practices. Ross and Katzmarzyk (2003) also suggested the inclusion during their involvement in the Canadian Population Survey, where they reported that cardio-respiratory fitness was inversely associated with WC and independent of BMI. Therefore, they emphasised that the measurement of waist circumference would be a reliable, easy and inexpensive indicator by which to identify persons at risk of CAD.

The WHO stated in a recent report that obesity has become a global problem (WHO Technical Report, 2000). Because developing/ under-developed countries may not be able to provide treatment for health-related problems associated with obesity, such as diabetes and heart disease, it is expected that obese persons in these countries probably face even more severe health-related consequences than do their counterparts in the United States and other developed countries. Waist circumference measurement would offer clinicians in these countries a desperately needed, practical and cheap tool for their routine practice (Wang, 2003). This statement is supported by the National Institute of Health (2000), which stated that WC is an easy and inexpensive useful tool for identifying overweight and obesity.

The use of abdominal size and circulating triglycerol concentration to estimate CAD risk is not new. Investigators in Quebec have pointed out that a "hypertriglyceridemic waist" could serve to identify men with hyper-insulinemia, elevated apolipoprotein B, and small, dense LDL-C particles (Lemieux *et al.*, 2000). Without reference to age dependence of abdominal size and triglycerol concentration, they assigned cut-offs for waist circumference (≥90 cm) and triglycerol concentration (≥1.45 mmol/l) that were rounded for simplicity. In cross-sectional studies they found that the hypertriglyceridemic waist was

positively associated with angiographically-assessed coronary artery disease, dyslipidemia, hyperglycaemia and hyperinsulinemia (Lemieux *et al.*, 2000; St-Pierre *et al.*, 2002).

2.3.3.7 Male gender

Although CAD is the leading cause of death for both men and women, males have a higher risk of heart attack, especially earlier in life. Until the age of 55, men also have greater risk for hypertension (high blood pressure) than women do. The incidence of strokes is higher in males than females under the age of 65. This increased male risk is not clearly understood and some researchers attribute it to the male sex hormone, testosterone, which triggers the production of LDL-C. Others are of the opinion that the male's lifestyle plays a major role in this finding. We know that females are protected until menopause. Female hormones signal the liver to produce more 'good' cholesterol (HDL-C), especially during childbearing years. Once women reach menopause (usually in their early to middle 50s), the incidence of heart disease becomes equal to, or surpasses, those of men of the same age (Robbins *et al.*, 2005).

Men can reduce their risk of contracting CAD by modifying other risk factors. These include leading an active lifestyle, maintaining a healthy weight, refraining from smoking, maintaining blood pressure and cholesterol at recommended levels, reducing stress and modifying emotional behaviour (Gordon *et al.*, 1990; Robbins *et al.*, 2005; Corbin *et al.*, 2006).

2.3.3.8 Advanced age

The risk of developing CAD increases with age. However, this does not mean that CAD is a disease of the elderly. Rather the risks accumulate over time, so that by the time an individual has reached an 'advanced age', they have an excess amount of cholesterol in their arteries (Robbins *et al.*, 2005).

Although nothing can stop the aging process, adopting an active lifestyle early in life may add 'years to your life and life to your years' (Robbins *et al.*, 2005). Through an active lifestyle, many factors believed to be associated with age, including heart and lung function, bone density, blood pressure and cholesterol, can be maintained at healthy thresholds. People who choose not to age rapidly by living an active, healthy life, can significantly reduce morbidity and extend their active years (Fries & Crapo, 1981).

2.3.3.8.1 Age and aerobic fitness

In any individual, aerobic fitness declines by 8-10% per decade regardless of the initial fitness level. Those individuals who remain active can cut the decline in half (4-5% per decade), and those who engage in fitness training can cut that rate in half again (2.5% per decade). A considerable body of evidence shows an inverse relationship between aerobic fitness and a number of risk factors. Thus, it is possible at the age of 60 to have the health and performance capabilities of the average 30-year-old. Age does not connote a rapid decline in performance, and when physical performance is important, the physiological age is a more accurate predictor of performance potential than chronological age (Sharkey, 1997).

2.3.3.8.2 Age and muscular fitness

Strength reaches a peak in the early 30's and declines slowly until the age of 60 or above. Thereafter, the rate of decline accelerates, but it does not have to. When strength is used, it hardly declines at all, even when individuals are in their 60's. Physical training conducted before puberty can lead to improvements that are mostly due to changes in the nervous system (neurogenic factors, including reduced inhibition and learning how to exert force). Training after puberty combines nervous system changes with changes in the muscle tissue (myogenic changes). Since testosterone declines in old age, many physiologists believe that senior citizens would be limited to neurogenic changes, should they undergo

a programme of physical training (Sharkey, 1997). However, a study on elderly people (72-98 years) has shown that resistance training leads to increased strength, muscle mass and reduced morbidity (Faitarone *et al.*, 1994).

2.3.3.9 Personality type

There are basically three emotional behavioural patterns: Types A, B, and C. Type A individuals exhibit aggressiveness, competitiveness, impatience and are easily annoyed. Type A individuals demonstrate a high degree of time urgency and a tendency to do two or three things at the same time. Type B individuals are more relaxed, non-competitive, patient and slow to anger. Finally, the type C individual has been identified as a type A who has learned to cope with emotional stress by making use of 'the Five Cs': control, commitment, challenge, choice in lifestyle and connectedness. Such individuals welcome change, and see it as a challenge. They are also committed to goals and gain confidence as a result (Robbins *et al.*, 2005).

Earlier studies identified type A people as being at greater risk of suffering heart attacks. However, more recent research indicates that a serious risk is only apparent when a type A person exhibits hostile and angry behaviour (Buist, 1995). Among the definitive research conducted in this regard is a long-term study of men, who in Law School took a standardised personality test that identified a number of them as hostile. Twenty-five years later it was found that the death rate among the men with the highest hostility scores was 4.2 times greater than that of the men who reflected low hostility scores (Bishop, 1989).

Williams (1987), has conducted research into the biochemical reactions of type A people when non-physical pressure is applied to them. In one experiment, the blood pressure was taken of men grappling with a difficult mental puzzle while being harassed. True to form, the type A men showed a markedly sharper leap in blood pressure than did the calmer type B individuals.

Based on his own research, as well as a comprehensive review of type A literature, Williams (1987) concluded that of the three characteristics time urgency, competitive achievement and hostility, which make up the global type A pattern, only measures of the latter are independently related to a wide variety of CHD end points. The research also suggested that the hostility complex may play a role in increasing the risk of dying from other causes.

2.3.3.10 Homocysteine

Homocysteine is an amino acid in the blood and a natural by-product of protein metabolism. The consumption of meat or vegetable sources (such as soya) starts a series of biochemical reactions that ultimately leads to the production of homocysteine. Normally, homocysteine is converted into non-damaging amino acids by folacin (often called folate or folic acid) and vitamin B_6 and B_{12}. However, in some individuals, these processes are impaired, and homocysteine accumulates in greater quantities than normal (Robbins *et al.*, 2005).

Excessive homocysteine in the blood is related to a higher risk of CAD, stroke, and peripheral vascular disease. Furthermore, it is known that homocysteine has a toxic effect on the cells lining the arteries, causing the blood to be more prone to clotting and promoting oxidation of low-density lipoprotein, resulting in an increased likelihood of cholesterol being deposited as plaque in the blood vessels (Robbins *et al.*, 2005; McCully, 1990). Highly elevated levels of homocysteine were found in the serum taken from ischemic heart disease patients, whereas low levels were found in every one of the healthy control groups (Clarke *et al.*, 1991). The homocysteine theory of atherosclerosis was developed after observing arteriosclerosis in children with different genetic enzyme deficiencies that caused elevated homocysteine levels in their blood, urine and arteriosclerotic lesions (Buist, 1995).

Additional evidence has shown that injecting rabbits with homocysteine derivatives caused elevations in serum cholesterol, LDL-C and triglycerides

(McCully, 1990). It has been suggested that laboratory testing for homocysteine levels can improve the assessment of CAD. It may be particularly useful in people who have a personal or family history of CAD, but in whom the well-established risk factors (inactivity, smoking, hypertension, and obesity) do not exist (Robbins *et al.*, 2005).

Homocysteine levels in the blood are strongly influenced by diet, as well as genetic factors. The dietary components with the greatest positive effects are folic acid and vitamins B_{12} and B_6. Folic acid and the B-vitamins help to decompose homocysteine and lower the blood concentration levels. Low levels of folic acid in the blood are linked with a higher risk of fatal CAD and stroke (McCully, 1990; Robbins *et al.*, 2005). Along with diets high in protein and low in B vitamins, heavy smoking has also been linked with high homocysteine levels. Heavy smokers tested up to 50% higher than did non-smokers (Robbins *et al.*, 2005).

2.3.3.11 C–reactive protein

It is true that primary and secondary risk factors increase the risk for heart disease, but scientists have known for some time that they are not the only culprits. Inflammation of the blood vessels may also trigger heart attacks and strokes by causing fatty deposits (plaque) in the arteries to rupture, creating the blood clot that causes heart attack or stroke. The process begins when the body interprets plaque in the arteries of the heart as an injury to the blood vessel wall. The immune system attacks the plaque, resulting in inflammation. Inflammation can be measured with a blood test that checks for a substance called C-reactive protein (CRP), a maker for this inflammation. CRP is produced by the liver in response to inflammation somewhere in the body and is now recognised as an important factor in heart disease. Elevated levels of CPR are linked to an increased risk for heart attack. A person can have no outward sign of inflammation, but still have subtle inflammation and hence elevated CRP. Doctors do not recommend universal blood testing for CRP, because there is no

treatment for high levels of CRP and because sometimes even something as simple as the common cold can elevate it (Robbins *et al.*, 2005; Corbin *et al.*, 2006).

2.3.3.12 Lipoprotein (a) and fibrinogen

Lipoprotein (a) plays a regulatory role in atherothrombosis. Unlike other lipid molecules which participate in the process of atherosclerosis, lipoprotein (a) is thought to compete with plasminogen in binding to fibrin. This results in a potential direct inhibition of endogenous fibrinolysis (Scanu, 1992). Fibrinogen, a protein produced by the liver, plays a role in the development of atherosclerotic plaque. Fibrinogen can also cause acute blood clot formation that may block a coronary or cerebral artery (Ernst, 1993). The fibrinogen levels of a group of physicians were tracked over a period of several years. In physicians with fibrinogen levels higher than 3.43 mmol/L, heart attack risk doubled compared to physicians with a fibrinogen reading below this number (Ma *et al.*, 1999). Even after adjusting for other risk factors such as BMI, diabetes, hypertension, alcohol consumption and HDL-C, there was an approximate two-fold increase in heart attack risk among physicians with the higher fibrinogen levels. Elevated fibrinogen is not only a powerful predictor of who will develop CAD. A recent study by Coppola *et al.* (2005), showed that high fibrinogen foretells who is likely to die within 42 months of suffering a heart attack. According to them, fibrinogen levels were the only independent predictor of mortality. Danesh *et al.* (2005) analysed 154,211 subjects, and based on age and sex alone the findings showed that for each 1.00 mmol/l increase in fibrinogen over the initial baseline level of 4.23 mmol/l, there was a 2.4-fold greater likelihood of contracting CAD. Deaths from cancers of the digestive tract, smoking-related cancers, and hormone-related cancers were also greater.

2.3.3.13 CAD risk indicators

The Framingham Study is one of the most important studies affecting health promotion, as it was the first to demonstrate correlations between cardiovascular disease and behaviour-related factors such as cigarette smoking, obesity, hypertension and physical inactivity (Dawber, 1980). The Framingham Study continues to provide much of the scientific justification for health promotion and disease prevention programmes. Since the risk factors identified in the study are primarily within the control of individuals (e.g. cigarette smoking, obesity, and physical inactivity), there are numerous opportunities to help persons reduce their risk of premature death (Castelli, 1990; Dawber, 1980).

Another example of such a study is the Multiple Risk Factor Intervention Trial (MRFIT), which is sponsored by the National Heart, Lung, and Blood Institute (NHLBI). The NHLBI falls under the joint control of the Department of Health and Human Services (DHHS), and the National Institutes of Health in the USA. The MRFIT involved 12,866 males, (aged 35-57), selected on the basis of being free from previous coronary disease, but who had elevated risk factors like hypertension, hypercholesterolemia, or cigarette smoking. The overall purpose of the study was to determine if a special intervention programme designed to simultaneously lower serum cholesterol, reduce blood pressure and eliminate cigarette smoking, would result in a significant reduction in mortality from CAD. The special intervention involved advice to limit daily intake of fat to 30-35% of total calories, encouragement and assistance to stop smoking, and use of the stepped-care approach for the treatment of hypertension. Participants in the control group were referred to their personal physicians for their usual care. (Kuller & Neaton, 1980).

There was a 10.5 mm Hg decrease in DBP for the special intervention group, 72 months following collection of baseline data, compared to a 7.3 mm Hg decrease for the control group ($p < 0.01$). Participants in the special intervention group reduced their total plasma cholesterol after six years by 0.3 mmol/L, compared to

a reduction of 0.2 mmol/L for the control group ($p < 0.01$). For men smoking at the start of the study, 50% in the special intervention group and 29% in the control group reported having stopped smoking ($p < 0.01$) after six years. The thiocyanate-adjusted quit rates were 46% for the special intervention group and 29% for the control group ($p < 0.01$). Mortality from CAD was 17.9 deaths per 1000 in the special intervention group and 19.3 per 1000 in the control group. The finding that both groups had lower mortality rates than expected, demonstrated the importance of current trends in society with respect to the general well-being of individuals. People are already making basic lifestyle changes, thus effective health promotion programmes are likely to accelerate the rate of behavioural changes and, as a consequence, produce more dramatic results (Bellingham & Cohen, 1987).

2.4. PHYSICAL ACTIVITY AND CORONARY ARTERY DISEASE

2.4.1 Introduction

Physical activity can be defined as bodily movement that is produced by the contraction of skeletal muscles whilst substantially increasing energy expenditure. Exercise, a subclass of physical activity, is defined as planned, structured and repetitive bodily movement done to improve or maintain one or more components of physical fitness. Physical fitness is defined as a set of attributes that people have, or achieve, that relate to the ability to perform physical activity (Pate, 1995; USA Department of Health and Human Services, 1996).

A study done on 31,000 bus drivers and conductors of the London Transport Authority (LTA) found that the drivers suffered significantly more coronary heart disease than the conductors did. Since the LTA uses a large number of double-deck busses, it is possible to consider the drivers as being sedentary, while the

conductors do considerable walking and stair climbing. It can therefore seem that men in active jobs suffer less from CAD (Morris *et al.*, 1953).

Numerous laboratory studies have quantified the many health and fitness benefits (e.g. physiological, metabolic and psychological) associated with endurance exercise training. In addition, an ever-increasing number of prospective epidemiologic studies support the notion that both a physically-active lifestyle and moderate to high level of cardio-respiratory fitness independently lower the risk of various chronic diseases. Inverse dose-response relationships for physical activity or cardio respiratory fitness are strongest for all-cause and cardiovascular mortality. However, lower incidence rates for hypertension, obesity, cancer of the colon, type 2 diabetes and osteoporosis have also consistently been reported in the literature. The bulk of the epidemiologic evidence supporting the health benefits of a more active lifestyle has been based on studies using single assessments of physical activity, or fitness. However, results from recent studies that correlate increases in physical activity or fitness in sedentary or unfit adults with subsequent reductions in mortality, support the hypothesis that regular activity increases longevity (Paffenbarger *et al.*, 1993; Paffenbarger *et al.*, 1994; Blair *et al.*, 1995).

In a classic epidemiological study, Paffenbarger *et al.* (1986) studied thousands of Harvard alumni to determine the influence of activity and sports on cardiovascular illness and death. In comparison with less active subjects (those who engaged in less than 1000 kcal of activity per week – a risk ratio of 1.0), moderate and high levels of activity yielded mortality risk ratios of 0.71 and 0.54, respectively. Thus, moderate activity yielded a 29% reduction in CAD risk, and high levels of activity yielded a 46% reduction. Those who played low intensity or moderately vigorous sports had mortality ratios of 0.79 and 0.63 compared to those who played no sports. Risk ratios for mortality, as well as first attacks of CAD were inversely related to physical activity, as indicated by kcal of weekly exercise. The mortality ratio approached 0.5, which means a 50% reduction in risk, when energy expenditure exceeded 2500 kcal/week.

In another study, Paffenbarger *et al.* (1990) presented data indicating that moderately vigorous activity and sport were more effective than low levels of activity in reducing the risk of CAD. The risk for low-activity alumni who played no vigorous sport was 2.4 times that of active alumni who engaged in vigorous sport.

In conclusion, Paffenbarger *et al.* (1984) stated that males, who expend more than 2000 kcal/week through exercising, reduce their risk of developing CAD by 39% in comparison to men who expend less. However, they admitted that 2000 kcal/week can be seen as the optimum amount of exercise, because those who use more than 2000 kcal/week showed very little extra protection against CAD.

2.4.2 The effect of physical activity on total cholesterol

Lopez *et al.* (1974), at the Louisiana State University School of Medicine, studied the effects of seven weeks of aerobic training on the serum lipids and lipoproteins in 13 young medical students. As expected, training reduced triglycerides from 1.2 to 0.9 mmol/l; a total reduction of 0.3 mmol/l. Furthermore, the researchers found a marked reduction of beta lipoprotein cholesterol (Cholesterol in LDL and VLDC), a concomitant increase in alpha lipoprotein cholesterol (HDL) and no change in body weight.

Wood (1975) of the Stanford Heart Disease Prevention Programme compared the lipoproteins of sedentary and active middle-aged men (35-59 years old). The active group consisted of runners averaging at least 24 km per week for the preceding year. The triglycerides were strikingly lower for the active group compared to the sedentary group, whereas TC was only modestly reduced. However, analysis of the lipoprotein pattern showed that the runners exhibited a significantly lower level of LDL-C and an elevated level of HDL-C.

Similar studies have been conducted in South Africa. In a community study of 1212 white males from two South African cities, Witbank and Vanderbijlpark, Van der Westhuizen (1997) demonstrated that males with a high fitness level had significantly lower TC levels compared to males with lower fitness levels. In a previous study by Strydom and co-workers (1985), 26 male executives from the South African motor industry were given a 24-week training programme and they managed to reduce their TC levels significantly.

Such studies have not been conducted on men exclusively. Andersen and Haraldsdottir (1995) showed that men who were considered fit (according to a bicycle ergometer test) had an average TC level of 4.62 mmol/l compared to 5.46 mmol/l for an unfit group. Similar results were gathered from a group of females, where fit women had an average TC of 4.60 mmol/l compared to the 5.26 mmol/l from the unfit group.

Kokkinos *et al.* (1991) and Manning *et al.* (1991) have observed that resistance training does not improve lipid profiles in men at risk for CHD and obese women when body weight remains stable. These findings suggest that changes in lipid profiles in response to resistance training may be partially dependent on weight loss and total training volume (Manning *et al.*, 1991).

Diet alone is not the ideal way to effectively reduce body fat and cholesterol levels and need to be combined with exercise. This will lead to an increase in fat metabolism, decrease in protein loss and maintenance of the metabolic rate. By therefore combining diet with exercise, excess caloric intake and inadequate caloric expenditure may be overcome to produce favourable body fat and therefore favourable lipid changes (ACSM, 1997; Joseph *et al.*, 1999b; Prabhakaran *et al.*, 1999). This was shown by Shaw (2004), who demonstrated that a 16-week combination fitness programme of dynamic resistance and aerobic training showed a decrease of 13.5% in the TC of healthy, but sedentary, males aged 20-35.

2.4.3 The effect of physical activity on LDL-cholesterol

A number of studies have demonstrated significant alterations in LDL-C. Two studies, in particular, showed that such alterations could be made with work loads ranging from exercising using stationary cycling for 50 min twice daily (Després *et al.,* 1990), to 30 min of organised gymnasium workouts combined with 30 min of stationary cycling in addition to walking 7 to 10 km per day (Filipovsky *et al.*, 1991). According to Shaw (2004), a combination between aerobic and resistance exercise showed a decrease of 26.3% in LDL-C. A decrease in LDL-C following exercise training may be due to an increased activity of cholesterol ester transfer protein (CETP), lecithin-cholesterol acyltransferase (LCAT), and lipoprotein lipase (LPL), which effectively increases lipoprotein metabolism (ACSM, 1997).

2.4.4 The effect of physical activity on HDL-cholesterol

Exercise can have a positive effect on HDL-C levels. Studies have shown that both regular exercise and weight loss are equally effective in raising HDL-C, but it may take several months to get their full effect (Gordon *et al.*, 1977).

Well-trained runners, of both sexes, posess lipoprotein profiles associated with a low CAD risk. Sedentary controls had LDL-C readings that were 20-30% higher than that of the runners. Even those who were less athletic, but physically-active, showed lipoprotein profiles that were associated with a reduced risk of cardiovascular disease. For example, data from the Lipid Clinics' Prevalence Study showed that men and women who reported some strenuous physical activity had higher HDL-C levels than those who reported none (Haskell, 1991).

Numerous studies have shown that vigorous exercise can raise HDL-C values from 0.5 to 1.0 mmol/l, which translates as a 20-40% reduction in CAD risk. The effect is more pronounced when fat weight is lost. Weight loss alone, from exercise or diet, may also lead to an improvement in HDL-C (Wood *et al.*, 1988).

That said, despite there being no record of changes in body weight, the study at Louisiana State University School of Medicine conducted by Lopez *et al.* (1974), also showed that training lowered triglycerides from 110 to 80 mg (a total reduction of 30 mg, along with the effects mentioned above).

The majority of longitudinal studies have employed rather high intensity exercise, most frequently jogging/running, but evidence is gradually becoming available that more accessible, self-governed exercise regimes may also be effective. An example is previously sedentary middle-aged women who had rather low levels of HDL-C (1.2 mmol/l) at baseline, walked briskly for about 20 km/week over a year, which resulted in a 27% increase in HDL-C (Després & Lamarche, 1990). A similar study examined the effects of walking at different speeds (up to 8 km/h) on a group of women over a 24-week period. Although the fitness of the women in question improved, there was no discernable difference in the level of HDL-C in the different groups walking at different speeds (Duncan *et al.*, 1991).

Despite this latter study, it can be assumed that, generally speaking, HDL-C levels can be increased by exercising regularly, by not smoking, by reducing weight if necessary and maintaining that weight, and by embarking on a high monounsaturated and low saturated diet (Robbins *et al.*, 2005; Corbin *et al.*, 2006).

2.4.5 The effect of physical activity on triglycerides

Franklin *et al.* (2002) conducted a study using 117 consecutive patients with an average age of 66.5 years, of which 68% were men. The patients, programme participants and staff, each filled in a health history questionnaire, which included information on clinical variables, coronary risk factors, submaximal and peak exercise test performance, subject demographics, and psycho-social well-being. Height, weight, resting SBP and DBP, fasting serum lipids and lipoproteins and fasting glucose were measured at programme entry and exit.

To evaluate the effectiveness of the physical training programme, heart rate, SBP and rating of perceived exertion at an individually determined standard were assessed. The same treadmill speed and grade, which initially evoked a heart rate response within the patient's prescribed training zone, were used for both evaluations.

The physical training programme consisted of an aerobic and circuit component. The aerobic circuit training programme included 45 to 60 minute sessions per week for 6 to 8 weeks. Each session included a warm-up period (5 to 10 min), an endurance phase (30 min) and a cool-down period. Training involved two upper-extremity, and/or lower extremity exercise devices, 15 min at each station. Potential training modalities included treadmill walking, rowing, automated stair climbing, recumbent ergometry, and arm, leg, or combined arm-leg ergometry.

Each patient's work rate during the endurance phase was directed at achieving a minimum of 40-50% to a maximum of 75% of their estimated Vo_2 max achieved at baseline exercise testing. When appropriate, the relative training intensity was gradually increased over the duration of the phase-two programme.

In addition to participating in exercise training, patients received education on cardiovascular disease, coronary risk factors and lifestyle modification. Education was provided during cardiac rehabilitation sessions and included the use of written materials, audio compact disc (nutrition, physical activity and exercise, stress management, prevention and health promotion), group education and one-on-one counselling.

The effectiveness of the exercise programme on the triglyceride levels of the patients participating in this study was substantiated by a significant ($p \leq 0.05$) reduction of 0.8 mmol/l.

2.4.6 The effect of physical activity on hypertension

Blood pressure is determined by cardiac output and total peripheral resistance. Consequently, it can be augmented by elevations in either, or both of these variables. The goal in prevention and management of hypertension is to reduce mortality by the least intrusive means possible. This may be accomplished by achieving and maintaining a SBP below 140 mm Hg and DBP less than 90/99 mm Hg and lower if tolerated, while controlling other cardiovascular disease risk factors (JNC VI, 1997).

A recent research project indicted that the effects of physical activity on blood pressure are more dramatic than previously thought and are independent of age, body fatness and other factors. The most plausible reason is a reduction in peripheral resistance to blood flow in the blood vessels, probably resulting from vasodilation in the vessels (Corbin *et al.*, 2006).

This finding is important, as it has been established that inactivity increases the risk of developing hypertension by 35%, and that physically-inactive subjects have a 52% greater risk than do the physically active (Sharkey, 1997; Corbin *et al.*, 2006). Active hypertensive patients have half the risk of death, from all causes, compared with inactive hypertensive patients (Paffenbarger, 1994). This has been demonstrated though research that showed that regular physical activity (i.e. aerobic exercise performed at 40-60% of maximal oxygen uptake, 3 to 5 times a week) reduces systolic and diastolic BP by about 10 mm Hg in hypertensive individuals. Moreover, those reductions in blood pressure in overweight and normal-weight individuals are independent of weight loss (Sharkey, 1997).

In the British Regional Heart Study, Wannamethee and Shaper (1992) found that the age-adjusted rate for stroke showed a steep and significant inverse gradient in the physical activity category in men with or without heart disease or stroke at baseline. In moderately-active subjects, the risks of developing hypertension

and, therefore, of suffering from a stroke, were less than half that reported for inactive men. This would appear to be supported by Paffenbarger and co-workers in their study of Harvard alumni. There it was shown that only 14% of 500 alumni students who were studied, who were physically active and free of hypertension when entering the study, developed hypertension over a period of 14 years (Paffenbarger *et al.*, 1991). Controlled exercise intervention trials have found an average reduction of 3.3 mm Hg (systolic/diastolic) in normotensives, with somewhat greater reductions in borderline hypertensive, i.e. 6/7 mm Hg and 10/8 mm Hg respectively (Bouchard, 1994).

In most studies, the risk of stroke decreases as activity increases. However, this cannot be taken as a rule that applies across the board. Data from the Honolulu Heart Programme showed an association between the risk of stroke and physical inactivity in older middle-aged men (55-68 years), but not in younger men aged between 45 and 54 (Abbot *et al.*, 1994). There is also evidence to suggest that, with activity that is more vigorous and possibly includes heavy lifting, the trend may reverse. Thus, compelling arguments are repeatedly found for moderation (Sharkey, 1997). Resistance training is not recommended as a primary form of exercise training for hypertensive individuals (ACSM, 1993; Gordon, 1997).

2.4.7 The effect of physical activity on type 1 diabetes mellitus

The response to exercise in the diabetic taking insulin depends on a variety of factors, including the adequacy of control by exogenous insulin. If the diabetic is under appropriate control or is only slightly hyperglycaemic without ketosis, exercise decreases blood glucose concentration, and a lower insulin dosage may be required. However, problems can arise during exercise if the diabetic is not under adequate control. A lack of sufficient insulin before exercise may impair glucose transport into the muscle, limiting the availability of glucose as an energy substrate. To compensate, the use of free fatty acids increases and ketone bodies are produced, possibly leading to the development of ketosis. For these reasons, type 1 diabetics must be under adequate control before beginning an

exercise programme. A blood concentration greater than 16.5 mmol/l (or greater than 13.2 mmol/l with urinary ketone bodies) is considered a relative contraindication to exercise participation (Gordon, 1995).

However, because exercise has an insulin-like effect, exercised-induced hypoglycaemia is the most common problem experienced by exercising diabetics who take exogenous insulin (or, to a lesser degree, oral hypoglycaemic agents). Hypoglycaemia may be the result when too much insulin is present, or if there is accelerated absorption of insulin from the injection site, both of which may occur with exercise. Hypoglycaemia occurs not only during exercise, but may occur 4 to 6 hours after a bout of exercise. To counteract the response, the diabetic may need to reduce his or her insulin dosage, or increase carbohydrate intake before or after exercise. In persons taking insulin, consideration should be given to the ingestion of 20-30 grams of additional carbohydrates before exercise when the pre-exercise blood glucose is under 5.5 mmol/l (Berger, 1995).

The risk of hypoglycaemic events may be minimised by measuring blood glucose before, during and after exercise and by avoiding exercise during peak insulin activity. Extra carbohydrates should precede unplanned exercise, e.g., 20-30 g per 30 min of exercise and insulin may have to be decreased after exercise. If exercise is planned, then the insulin dosage must be decreased before and after exercise, according to the exercise intensity and duration, as well as the personal experience of the individual. Insulin dosage reductions may amount to 50 to 90% of insulin requirement. During exercise, easily absorbent carbohydrates may have to be consumed and, after exercise, a carbohydrate snack may be necessary. The individual must be knowledgeable of the signs and symptoms of hypoglycaemia and always exercise with a partner (Berger, 1995).

2.4.8 The effect of physical activity on type 2 diabetes mellitus

Exercise increases insulin sensitivity and the movement of glucose into working muscles. Regular activity has returned to a place of prominence in the treatment

of non-insulin-dependant diabetes (NIDDM), and for some it removes the need for insulin substitutes. In general, regularly-active adults have a 42% lower risk of NIDDM (Sharkey, 1997). Manson *et al.* (1991) reported that women who engaged in vigorous physical activity at least once a week have a reduced risk of developing type 2 diabetes. The reduction in diabetes risk, however, appears to be more associated with the frequency of physical activity. The risk of developing type 2 diabetes decreased 23%, 38% and 42% respectively in male physicians who exercised vigorously once, twice or up to four or five times a week (Manson *et al.*, 1992). Male physicians in the United States, who exercised 'vigorously' at least once per week, experienced only 64% of the risk of developing non-insulin dependent diabetes mellitus, compared to those who exercised less frequently. However, even leisure-time activities can prove to be beneficial in reducing the risk of developing NIDDM. Among male alumni of the University of Pennsylvania, the incidence of non-insulin dependent diabetes mellitus decreased by some 6% for each 500 kcal expended in physical activity per week (Kriska *et al.*, 1994).

2.4.9 The effect of physical activity on obesity

The USA Surgeon General's Report (2000) provides strong support for physical activity in the prevention of obesity. This statement is supported by Anderson and Heraldsdottir (1995) and McArdle *et al.* (1991), who found that physical activity has a significant effect on the reduction of BMI and percentage body fat.

Physical activity increases the resting metabolic rate, as well as the basal metabolic rate, which results in elevated energy consumption (McAdle *et al.*, 1991; Rippe & Hess, 1998). Fat oxidation is enhanced during submaximal exercise, more so in people who are well-trained, and is enhanced for some hours afterwards, even when the post-exercise elevation of metabolic rate has disappeared (Calles-Escandon *et al.*, 1992).

The consensus in the literature is that relatively small increases in physical activity (for example walking 3,2 km per day, three times per week, adding up to 500-600 kcal) are not associated with changes in body fatness over a 3-6 months period. Above this amount of exercise, there tends to be a consistent loss of body fat (0.12 kg/week for men and a little less for women). Thus, total exercise expenditure may be the variable most strongly related to the body mass change (Haskell, 1991). However, the risk of major weight gain (>13 kg) over a 10-year period was twice as high among inactive men and sevenfold higher among inactive women, compared with men and women of a high activity level (Williamson *et al.*, 1993).

Sjodin *et al.* (1992) conducted research on male and female athletes and compared them with a sedentary control group. The study showed that the female athletes had 7% less fat than the females in the sedentary control group. The male athletes had 6% less fat than the male sedentary control group. Not only athletes benefit, however. Ready *et al.* (1996) researched the effect that a walking programme has on the fat percentage of postmenopausal women older than 50 years. After an exercise routine of 5-days per week for 24 days at an intensity of 60% of VO_{2max}, they showed a reduction of 4.2% in body fat, compared to an increase in body fat percentage in the controlled group.

Klem *et al.* (1997) performed a retrospective analysis of exercise habits of subjects in the National Weight Control Registry and found that those persons who successfully maintained long-term weight loss reported an energy expenditure of 2800 kcal/week. Using more objective measurement methods, Schoeller *et al.* (1997) performed a cross-sectional study, in which an energy expenditure of 2400 kcal/week was associated with improved 12-month weight loss maintenance. In an unusual consistency across studies, Jacicic *et al.* (1999) also performed a retrospective study on the amount of physical activity reported by participants in a diet and exercise study. They found significantly better 12 and 18-month weight loss and maintenance among those who reported an energy expenditure of 2200 kcal/week than among those who reported an energy

expenditure of only 1300 kcal/week. However, despite the quantitative agreement among the above, none of them prospectively tested the efficacy of a prescription of the amount of physical activity for long-term weight loss.

Jeffery *et al.* (2003) compared the efficacy of two physical activity prescriptions in a weight-loss treatment that combined diet and exercise. One group of participants was asked to initiate a physical activity programme with a dietary energy expenditure goal of 1000 kcal/week, and the other group was given an energy expenditure goal of 2500 kcal/week. Both groups received a prescription to restrict energy intake to 1000-1500 kcal/day with 20% of energy from fat. After the first six months, there was no difference in weight loss between the groups. However, over the next six months, the high physical active (HPA) maintained their weight (0.5 kg gain in 6-months) whereas the low physical active (LPA) had a 2 kg regain. The difference in rate of regain disappeared during the interval between 12 and 18 months, but the HPA group still had a significantly greater weight loss than did the LPA group (6.7 kg compared with 4.1 kg). Compliance with the physical activity prescription was very good. According to their self-reporting, the LPA group actually exceeded their prescription by 500 kcal/week on average, whereas the HPA group fell short of their goal by 100-200 kcal/week.

As noted Jeffery *et al.*, (2003) found little or no difference in weight loss after six months, although the HPA group averaged a 0.9 kg greater loss. These findings are consistent with findings in a large body of literature comparing short-term weight loss for dietary restriction with and without exercise (Votruba *et al.*, 2000). Meta-analysis reveals that the added physical activity does, indeed, lead to a fat loss that is significantly greater by 1 kg, although weight-loss differences are small or insignificant (Bellor & Phoelman, 1994).

The small size or absence of the short-term effect appears counter-intuitive, but it should be remembered that the various amounts and types of physical activity add only 100-350 kcal/day to the energy expenditure, whereas the energy-restricting diets reduce energy intake by 900 to 1600 kcal/day. The energy deficit

from energy restriction, therefore, far exceeds that from added physical activity, and the small added deficit from physical activity is lost quickly in the variance with respect to individual compliance.

Although it was concluded by Jeffery *et al.* (2003) that an energy expenditure prescription of 2500 kcal/week, or 75 minutes of brisk walking per day is effective for long-term weight loss, the efficacy waned during the period between 12 and 18 months. This finding is almost identical to the findings of Jacicic *et al.* (1999). Inspection of the self-reported energy intake and physical activity amounts reported by Jeffery *et al.* (2003) provides no indication as to why the HPA group began to regain weight. They did not report mean increases in energy intake or reductions in physical activity. Indeed, both values trended in the opposite direction. The failure to identify the component of energy balance that that changed is probably a limitation of self-reporting. Both self-report energy intake and physical activity are subject to misreporting in studies of weight loss (Jacicic, 1999; Racette *et al.*, 1995). This should not detract from the importance of the findings of this study, but should rather provide motivation for further studies using more objective measures of energy balance that can answer the question of which treatment component is subject to the faltering compliance that leads to weight regain.

2.4.10 The effect of physical activity on stress

Aerobic exercise promotes health and energy and is a powerful antidote for anxiety and stress (Labbate *et al.*, 1995; Robbins *et al.*, 2005; Corbin *et al.*, 2006). Exercise allows us to play out the inappropriate fight-or-flight response, Use the muscles that are tense for action, and reduce the adrenaline being pumped into the blood stream. Exercise reduces the intensity of the stress response, shortens the time it takes to recover from stress, and helps ward of illness in people experiencing stress. While stress increases blood pressure and platelet stickiness (the factor that increases clotting), exercise reduces them (Sharkey, 1997; Robbins *et al.*, 2005).

As a result, many physicians recommend exercise to their "stressed" patients instead of tranquilisers (Robbins *et al.*, 2005). A randomised clinical trial into the treatment of major depressive disorders studied three groups. One group took antidepressant medication, another was prescribed aerobic exercise and a third was given a combined antidepressant and exercise programme. Results indicated that the aerobic exercise group fared as well as the other two at the end of treatment. In addition, patients who only exercised were less likely to have a remission to depression after six months. Individuals in the exercise programme possibly felt more responsible for the improvements in their condition and this increase in self-efficacy led to better long-term outcomes (Corbin *et al.*, 2006).

It can be suggested then that physical activity is associated with a physiological response that is similar, in many ways, to the body's response to psychosocial stressors. Individuals who are physically fit have a reduced physiological response to exercise. Presumably, someone who is physically fit would also have a reduced response to psychosocial stressors. However, more research is necessary. Recent studies have attempted to clarify the role of exercise in stress reactivity by more clearly specifying the physiological system activated by exercise-related stress and non-exercise related stress. The body's physiological response consists of both a sympathetic nervous system and endocrine response. The sympathetic nervous system response to exercise is immediate, whereas the endocrine response to exercise is delayed. In contrast, the endocrine response to psychological stressors is, usually, immediate. Such differences in responses suggest that more complex models may be necessary to understand the role of exercise in protecting against psychosocial stress (Corbin *et al.*, 2006).

Brown (1991) found that people who tested physically fit during a cycle ergometer test were less prone to falling ill during stressful life situations than did people with a relatively low fitness level. Individuals who are exposed to stressful

life situations generally experience an increase in cardiovascular reactivity (increase in heart rate, blood pressure, secretion of stress hormones and neuro-system activity). These can lead to myocardial infarction in some individuals (Nieman, 1998). Physical activities also seem to have a counteractive influence on this cardiovascular reactivity (Pretorius *et al.*, 1989; Labbate *et al.*, 1995). Furthermore, individuals with a propensity towards emotional responsitivity during stressful work situations show an increased tendency towards the development of CHD (Melamed, 1996). Stressed individuals, therefore, seem to be predisposed to the development of CHD within and outside of the work environment.

Research shows that the average executive in South Africa find themselves in a work environment of high stress, yet low physical activity (Jacobs, 1991; Van Zyl, 1995). In this context, Dreyer and Strydom (1994) showed that only 3% of South African executives were physically active at work, and that only 14.3% participate in leisure-time physical activities, which would be sufficient to ensure improved health. Strumpfer (1989) indicated that South African executives experience higher levels of stress than did their American and Dutch peers. He ascribes this to the heavier workloads, which have to be handled by fewer available managers. As a result, this can place the high-level employee in a work environment that can be detrimental to their health.

The South African National Games and Leisure Activities (SANGALA, 2000) project gathered some 833 male managers from companies across South Africa. The subjects were all between 21 and 71 years of age. The study set out to determine whether a mutual relationship existed between the physical activity, coronary risk and burnout indices. The study showed that the highly physically-active ($\geq$1000 kcal/week) and moderately-active (151-999 kcal/week) managers showed a significantly lower burnout index than did the physically less-active ($\leq$150 kcal/week) managers. The managers in the physically low-, moderately- and highly-active groups, however, all fell within the lower burnout category.

The latter shows that physical activities can significantly lower the burnout index of respondents, even in a low-burnout category. This corresponds with research that indicates that participation in even moderate activity could offer protection against coronary heart disease (Paffenbarger, 1988; Pate *et al.*, 1995).

From the literature it seems that about 50% of managers in South Africa show a physical activity profile of ≥1000 kcal/week (Van Zyl, 1995). For that group then, this level of activity should contain health-promoting and preserving benefits, not only for the individual, but also for the organisations for which they work (Pestana *et al.*, 1996; Katzmarzyk *et al.*, 2000).

The effect of physical activity on the relationship between burnout and the coronary risk index (CRI) of South African managers within each of the various burnout groups (low-, moderate- and high burn-out) showed that highly active managers had a statistically significant smaller risk for the development of CHD than those who were low-active (SANGALA, 2000).

No statistically-significant variances were found between the moderately physical and low-active managers within the same burnout group with regard to the CRI burnout connection. The reason for this could possibly be that the exercise response of the moderately-active group was too low to bring about any physiological or psychological influence (SANGALA, 2000).

2.5 NUTRITION AND CORONARY ARTERY DISEASE

2.5.1 Introduction

The typical American diet is high in calories, fat (particularly saturated fat), cholesterol and sodium, and low in fibre (Franklin *et al.*, 2002). This dietary profile contributes to a number of chronic diseases, including heart disease, obesity, stroke, diabetes, hypertension, osteoporosis and certain cancers (Eckel

& Klauss, 1998; Corbin *et al.*, 2006). Pollock and Schmidt (1977) showed that the total calorie intake has a direct effect on TC concentration values, as well as on lipoprotein fractions. Diet and being overweight play an important role in elevated cholesterol levels. Well-controlled exercise programmes coinciding with a balanced diet and calorie intake can play a major role in controlling elevated cholesterol. Consumption of too many saturated fatty acids is one of the major causes of CAD. The ideal intake should be a maximum of 10% of total kilojoules, but white and coloured South Africans consume an estimated 13.3% and 11.6% respectively. Relatively high consumption of cholesterol is also likely to be responsible for CAD. White and coloured South Africans consume an estimated 362 mg and 314 mg, respectively, when the ideal is 300 mg, (Gordon & Gibbons, 1990).

The goals of dietary modification are often to reduce or maintain body weight and fat stores, decrease elevated plasma TC and LDL-C, and lower blood pressure (Franklin *et al.*, 2002).

Clearly, the amount of fat that is taken in through the regular diet should be limited. A diet where 30% of the total calories come from fat (of which 20% should come from poly- and mono-unsaturated fat and 10 % from saturated fat) is recommended for weight-loss (National Institute of Health, 2000). That said, there is an ongoing discussion about what the ideal macronutrient profile should be to optimise and maintain weight loss (Freedman *et al.*, 2001). However, an unintended consequence of emphasising this low-fat diet may have been to promote unrestricted carbohydrate intake (Wynber, 2004).

2.5.2 Diets and cardiovascular disease

The USA is facing an increasing public health crisis due to the prevalence of obesity and people being overweight. The increase in incidents of these conditions and their associated risks for chronic diseases like CAD or diabetes are a major cause of concern (Chan *et al.*, 1994; Colditz *et al.*, 1995; Lamon-

Fava *et al.*, 1996; Rexrode *et al.*, 1997; National Institute of Health, 2000; Colditz, 1999).

Due to the increasing prevalence of obesity, despite low-fat recommendations, many new popular diets have emerged. Although some of these new diets may offer health benefits, others may potentially harm cardiovascular or overall health (Parikh *et al.*, 2005).

2.5.2.1 Low-carbohydrate diet

This type of diet has received much attention due to the Dr. Atkins's New Diet Revolution (Atkins, 1998). The Atkins Diet recommends two weeks of extreme carbohydrate restriction, followed by gradually increasing carbohydrates to 35 g/day. The Atkins diet has 68% of total calories from fat, 27% from protein, and 5% from carbohydrates. Low carbohydrate diets recommend limiting complex and simple sugars, causing the body to oxidise fat to meet energy requirements. During the initial carbohydrate restriction, the body resorts to ketosis for energy needs. Ketones are excreted in the urine with fluid. Rapid initial weight loss may be from this diuretic effect (St-Jeor *et al.*, 2001). A drastic reduction in carbohydrates also adds to an overall decrease in caloric intake (Bonow & Eckel, 2003). Even when calories are not actively restricted, low-carbohydrate dieters consume fever calories compared with baseline (Bravata *et al.*, 2003).

Foster *et al.* (2003) conducted a randomised, controlled trial lasting one year. Sixty-three obese patients were assigned either a low-carbohydrate diet, or a low-fat diet. The low-carbohydrate group showed greater weight loss at six months, but the weight loss between the groups was not significant at one year. Lower carbohydrate dieters showed a greater increase in HDL-C and a decrease in triglycerides that was independent of weight loss. A similar study was conducted by Stern *et al.* (2004), where 132 obese patients were followed over the course of a year. The subjects were randomly assigned to either a carbohydrate-restricted or fat-restricted diet. Average caloric intake decreased

by 501 kcal/day in the low-carbohydrate group, but only by 97 kcal/day in the low-fat group. After six months, the low-carbohydrate group showed greater weight loss, increased HDL-C, decreased triglycerides, and increased insulin sensitivity. After one year, there was no difference in weight loss between the two groups, although those on the low-carbohydrate diet continued to have lower levels of triglyceride and higher HDL-C levels.

A low-carbohydrate diet, therefore, may increase HDL-C, decrease triglyceride levels and increase glycaemic control, but there appears to be no significant difference in weight loss when compared to a low-fat diet over one year. However, because the longest trial only extended as far as one year and had relatively few subjects, more studies are required to determine the efficacy of a low-carbohydrate diet on long-term weight loss and cardiovascular outcomes (Parikh *et al.*, 2005).

2.5.2.2 Glycaemic Index diet

The diet of an individual may affect body weight through multiple pathways, including the control of satiety and metabolic efficiency, or through modulation of insulin secretion and action. Total and saturated fats have been the focus of intense scientific scrutiny as potential causes for being overweight and obesity but there is no conclusive evidence directly linking dietary fats to body fat as yet. Whereas there is no doubt that overfeeding, where a large percentage of energy comes from fat, can cause obesity and insulin resistance (Storlein *et al.*, 1991), the magnitude and long-term significance of the effect of a low-fat diet on weight control in humans remains uncertain (Willet *et al.*, 1999; Willet, 2002). In feeding trials, reductions of approximately 10% have been shown from low-fat diets (Jeffery *et al,* 1995; Bray & Popkin, 1998), but much of the weight that was lost was regained within 12-months, and no long-term efficacy has been convincingly shown. Moreover, the effects of changes in dietary composition (percentage of energy from fat rather from carbohydrates) alone on weight loss appeared minimal (Willet, 2002). Reports based on the Third National Health and Nutrition

Examination Survey (NHANES III) and a recent time-trend analysis indicated that intake of both total and saturated fat, in terms of percentage of total caloric intake, have been declining in the USA since the 1960s (Stephen & Wald, 1990). In contrast, during the same period, an increase in the intake of refined carbohydrates in the form of processed grains, soft drinks, sugars and refined flours in the USA food supply has been reported to parallel the increased prevalence of obesity and diabetes (Ludwig *et al.*, 2001).

The glycaemic index diet allows carbohydrate consumption as long as they have a low Glycaemic Index (GI). The GI is a measure of the blood glucose response to intake of a particular carbohydrate (Jenkins *et al.*, 1981). The higher the peak in postprandial blood glucose levels, the higher the GI value. The glycaemic load (GL) is the product of dietary GI and total dietary carbohydrate, providing a useful measure of the total glycaemic effect (Jenkins *et al.*, 2002). A high-GI diet is supposed to increase hunger and elevate free fatty acid levels, leading to an increased risk of obesity, diabetes and CVD (Foster *et al.*, 2002). Several in-vitro experiments indicated that elevated postprandial blood glucose levels cause oxidative stress, leading to endothelia damage and activation of coagulation (Leafebvre & Scheen, 1998).

The longest interventional study related to GI, and conducted with human subjects, was a crossover study lasting twelve weeks (Slabber *et al.*, 1994). Thirty women were randomly assigned either a low-GI or high-GI diet. Those on a high-GI diet lost 7.4 kg, whereas those on a low-GI diet lost 9.4 kg. In 16 women who participated in a 12-week follow-up crossover study, those on a low-GI diet lost a further 7.4 kg compared to 4.5 kg on a high-GI diet. However, the results from other interventional studies, although shorter in duration and with smaller populations, have been inconsistent (Pi-Sunyer, 2002).

A possible association between a high GI and diabetes has been observed. Studies that investigated this relationship include the Nurse's Health Study by Salmeron *et al.* (1997), which followed over 65,000 American women for six

years, and the Health Professionals' Survey by Salmeron *et al.* (1997), which followed 42,750 American men for six years. Both these prospective cohort studies showed an association between diabetes and high GI. High-GI diets may alter HDL-C metabolism. In a survey of 1,420 British adults, Frost *et al.* (1999) evaluated GI through a seven-day diet survey and showed an inverse relationship between GI and HDL-C. The NHANES III, which followed 13,907 subjects older than 20 years old, demonstrated that for every 15-U increase in GI, there was a 0.06 mmol/l decrease in HDL-C (Ford & Lui, 2001).

Results from several short-term feeding trials in humans (a single meal or a single day) suggest that the consumption of wholegrain products with a low glycaemic index might increase satiety and reduce energy consumption and, thus, contribute to weight loss (Roberts & Heyman, 2000). The belief that diets rich in fibre are generally low in saturated fat has led many national authorities to recommend greater consumption of grain products to control weight (USA Department of Agriculture and USA Department of Health & Human Services, 2000). However, most grain products consumed in the US are highly refined (Slavin *et al.*, 1999; Putnam *et al.*, 2002). Several epidemiological studies of dietary fibre also suggest that the intake of whole grains, but not of refined grains, is inversely associated with body weight and fat distribution (Ludwig *et al.*, 1999; Roberts & Heyman, 2000; Liu, 2002).

Whole grains may have beneficial effects on weight control through promoting satiety (Slavin *et al.*, 1999). This may be because refined-grain products have higher starch content, but lower fibre content than do whole grains (Liu, 2002). The intake of whole grains slows starch digestion or absorption, which leads to relatively lower insulin and glucose responses, so favouring the oxidation of fat rather than its storage (Slavin *et al.*, 1999; Liu, 2002).

However, very few trials have directly examined the effects of whole grains, as apposed to those of refined grains, on body weight and weight change. Nor is there any epidemiological study directly linking the intake of whole grains to

changes in weight over time. Of those that have been conducted, results show that the consumption of whole-grain products with a low glycaemic index might increase satiety and reduce energy consumption and thus contribute to weight loss in overweight individuals (Roberts & Heyman, 2000).

To further examine the association between grain intake, body weight and weight changes, Liu *et al.* (2003) analysed prospective data from the Nurses' Health Study (NHS) from 1984 to 1996, using repeated measurements of grain intake and body weight. It was found that women who consumed more whole grains consistently weighed less than did women who consumed less wholegrain food. Over the 12 years of the study, those with the greater increase in dietary fibre gained an average of 1.52 kg less than did those with the smallest increase in intake of dietary fibre. These results are independent of body weight at baseline, age and changes in covariate status. Women in the highest quintile of dietary fibre intake had a 49% lower risk of major weight gain than did women in the lowest quintile. The conclusion of the study was that weight gain was inversely associated with the intake of high-fibre and wholegrain foods, but positively related to the intake of refined-grain foods, which indicates the importance of distinguishing wholegrain products from refined-grain products to aid in weight control.

Despite suggestive evidence, no trials have shown that low-GI diets prevent CVD. Longer studies with more participants are needed before low-GI diets can be definitely recommended (Parikh *et al.*, 2005).

2.5.2.3 Very low fat diet

Very low-fat (VLF) diets allow less than 15% of total calories from fat (with an equal distribution of saturated, mono-unsaturated and poly-unsaturated fats), only 15% from protein and 70% from carbohydrates. The VLF diet includes variations of vegetarian diets that may include eggs and dairy. Although an AHA scientific statement concluded that there was little long-term data to suggest that

a low-fat diet alone will sustain long-term weight loss, there is evidence that this diet can have an impact on CAD (Lichtenstein & van Horn, 1998).

During the Heidelberg trial, Niebauer *et al.* (1995) evaluated 113 patients with stable angina. Each member of the experimental group that reduced their fat intake to less than 20% of their calories, reduced their TC to 5.2 mmol/l (and engaged in exercise of moderate intensity). After 12 months, the intervention group's body weight had decreased by 5%. Their TC had decreased by 10% and triglycerides by 24%. In the intervention group, progression of coronary lesions by angiography had decreased when compared with that of the control group. However, given the confounding effect of exercise, this study makes it difficult to assess the effects of diet alone.

Similarly, during a Lifestyle Heart Trial, Ornish *et al.* (1998) randomised 48 patients with moderate to severe coronary heart disease (CHD), assigning them to either intensive lifestyle changes or usual care. The intensive life-style changes included a vegetarian diet where 7% of caloric intake came from fat, moderate aerobic exercise, stress management training, smoking cessation and group psychological support. A total of 195 coronary artery lesions were analysed angiographically. Overall, 82% of the experimental group had an average change toward lesion regression. After five years there were 2.5 times fewer cardiac events in the intervention group, and the average stenosis showed an 8% decrease in diameter, whereas the control group had a 28% progression. However, once again, the data is difficult to interpret due to the confounding effects of exercise, stress reduction, and an average of an 11 kg weight loss in the intervention group. Although the intervention seems beneficial, the small sample size and intense lifestyle changes raise concerns about the universal sustainability of such a programme.

The VLF diet and intense lifestyle changes have significant results in terms of reducing risk factors and cardiac event rates. However, these studies are relatively small, and the programmes involved may be influenced by selection

bias. The programmes require a motivated group of patients to undergo lifestyle adjustments. The VLF diet may be unnecessary if other lifestyle characteristics like exercise, smoking cessation and stress management are optimised (Parikh *et al.*, 2005).

2.5.2.4 The Mediterranean diet

The Mediterranean diet is characterised by a daily abundance of plant food (fruit, vegetables, breads, cereals, potatoes, beans, nuts and seeds), and foods that are minimally processed, seasonally fresh, and locally grown. It also includes the occasional sweets containing refined sugars and honey; olive oil, which is high in polyunsaturated fat, as the principal source of fat; and daily dairy products, coming mainly from cheese and yogurt, in low- to moderate amounts. It also includes fish and poultry in moderate amounts (but red meat only rarely), up to four eggs weekly, and wine in low- to moderate amounts with meals (Hu, 2003).

Although a Mediterranean-style diet has demonstrated greater weight reduction compared to controlled diets in randomised, controlled trials (Esposito *et al.*, 2004), the most impressive benefits of the diet are related to cardiovascular mortality. No single isolated aspect of the Mediterranean diet can explain these benefits, but much of the attention has been focused on the omega 3-polyunsaturated fatty acids (N3-FA) (Kris-Etherton *et al.*, 2002). Examples of N3-FA include eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) found in fatty fish like salmon, trout and mackerel (Kris-Etherton *et al.,* 2002). A form of N3-FA derived from plants (alpha-linolenic acid) is found in nuts, canola oil, flaxseed oil and soybean oil (Kris-Etherton *et al.*, 2004). Alpha-linolenic acid can be converted to EPH and DHA, which is thought to be cardioprotective (Grundy, 2003).

One mechanism of protection may be related to the anti-arrhythmic effects of Omega-3 polyunsaturated fatty acids (Leaf *et al.*, 2003). Data from various animal, epidemiologic, and metabolic studies, as well as smaller clinical trials,

demonstrates the benefits of in reducing the risk of sudden cardiac death (Kang & Leaf, 2000). Moreover, fish oil supplements can lower triglycerides, inhibit endothelial cell activation, and improve endothelial function in diabetics (Brown & Hu, 2001). They can also reduce platelet aggregation, (Dallongeville *et al.*, 2003) and decrease heart rate (Hu & Willet, 2002).

During the Diet and Reinfarction Trial (DART), Von Shacy (2000) studied 2000 men over two years to study the effects of fish oil on the secondary prevention of MI. The men were randomised into four groups. The first group received advice in accordance with American Heart Association weekly. The third group was told to increase cereal fibre intake to 18 g per day. A fourth group (the control) did not receive any dietary advice. In comparing the four groups, the fish group showed a 29% reduction in mortality compared with the control group. The rate of fatal MI was also less in the fish group.

A similar study, the Lyon Diet Heart Study, conducted by De Lorgeril *et al.* (1999) randomised 605 participants with a previous MI for 46 months, and showed an inverse relationship between ALA intake and the risk of a second MI. The intervention group was advised to eat more fish, fruit, and vegetables and use an ALA-rich margarine. The control group was advised to follow a prudent diet. There was a 68% decrease in primary endpoints (cardiac death and non-fatal MI). Secondary endpoints (periprocedural infarctions, unstable angina, heart failure, stroke and pulmonary or peripheral embolisms) also decreased. It is also worth noting that, at a follow-up after four years, most of the experimental patients were still closely following the recommended diet.

Both from the point of view of basic science and clinical trials, there is consistent evidence for the cardio-protective effects of the Mediterranean Diet. This evidence is particularly clear in secondary prevention of MI. Patients on a Mediterranean diet have been shown to lose weight, have lower levels of CRP, have less insulin resistance, and have lower TC and triglycerides and higher levels of HDL-C and a decreased prevalence of the Metabolic Syndrome

(Esposito *et al.*, 2004). With regard to primary prevention, studies show that a Mediterranean diet may be linked to decreased rates of sudden cardiac death, CHD, and possibly overall mortality. A systematic review by Hu and Willet (2002) of metabolic, epidemiologic and clinical trial evidence indicated that three dietary strategies are effective in preventing CHD: substituting non-hydrogenated unsaturated fats for saturated and trans-fats, increasing consumption of omega-3 fatty acids and consuming more fruits, vegetables, nuts and wholegrains, while avoiding refined grain products.

2.5.2.5 The dietary approach to stop hypertension (DASH)

The dietary approach to stop hypertension (DASH) is similar to a Mediterranean-type diet, emphasising the high intake of fruit, vegetables, low-fat dairy products, whole grains, nuts, fish and poultry, as well as reducing total and saturated fats. Reduced intake of red meat, sweets and sugar-containing beverages is encouraged, which results in a diet high in potassium, calcium, magnesium and fibre. This dietary approach has been shown to lower blood pressure, but little has been published regarding weight loss (Parikh *et al.*, 2005).

The original DASH trials consisted of 459 subjects with SBP that was <160 mm Hg and DBP of between 80 and 95mm Hg. For three weeks all participants were fed a control diet low in fruit and vegetables, and dairy products with a fat content typical of the American diet (37% of daily caloric intake). During the following eight weeks, the participants were randomised to one of three diets: the control diet, a diet rich in fruits and vegetables, or the DASH diet. The DASH diet reduced SBP by 5.5mm Hg and DBP by 3.3mm Hg when compared with the control group.

To investigate the effects of sodium restriction, the DASH sodium trial was conducted (Sacks *et al.*, 2001). A total of 412 subjects were randomised to either the control diet or the DASH diet for 90 days. Within each group, patients were further stratified and randomly assigned to three diets: high (3.5 g/day),

intermediate (2.3 g/day), or low (1.2 g/day) sodium, each for a 30-day period. In the control group, there was a dose response with the greater reductions in sodium intake correlating with greater decreases in blood pressure. For those on the DASH diet, the dose response persisted, although the effects of sodium reduction were smaller. Additionally, there was no significant difference between high and intermediate sodium intake on DBP for those on the DASH diet. The difference was only significant between the high and low sodium groups. Therefore, it can be seen that the DASH diet can reduce SBP by 5.5 mm Hg and the DBP by 3.3 mm Hg. However, the effect of sodium reduction on hypertension remains controversial. Lowering sodium to the levels of 1.2 g/day, as achieved in the lowest sodium intake group of the DASH sodium trial, would nearly be impossible without changes in the food industry, as 75% of sodium intake comes from the processing of food (Whelton & Appel, 2002).

2.5.2.6 Fruit and vegetable consumption

In a cross-sectional study performed by Douse *et al.* (2004), which was conducted on 4466 individuals (2047 men and 2419 women), it was found that the consumption of fruit and vegetables was inversely related to LDL-C concentrations in both sexes, independent of age, Keys scores, smoking status, exercise, educational attainment and use of vitamin supplements. Subjects in the group with the highest fruit and vegetable intake had LDL-C concentrations that were 6-7% lower than those in the group with the lowest fruit and vegetable intake. Although several clinical trials and observational studies have assessed the effects of dietary fat on LDL-C concentrations, limited data is available on the relationship between the consumption of fruit and vegetables and LDL-C concentrations in a community-based population. In the DASH trial, the LDL-C concentrations of 146 subjects assigned to consume a fruit and vegetable diet were not significantly lower than concentrations in the control group after an eight-week intervention period (Obarzanek *et al.*, 2001). In sharp contrast, the Indian Diet Heart Study showed that fruit and vegetable consumption was associated with a 7.3% decrease in LDL-C after 12 weeks of intervention (Singh

et al., 1992). Neither the DASH trial, nor the Indian Diet Heart Study assessed a dose-response effect between fruit and vegetable consumption and LDL-C concentrations. Fornes *et al.* (2000) reported in a cross-sectional study that the intake of fruit and vegetables was inversely correlated with LDL-C concentrations. Singh *et al.* (1996) and Bruce *et al.* (2000) reported beneficial effects of fruit and vegetable intake on LDL-C from baseline after 8 weeks. In a randomised trial, fruit and vegetable intake was associated with a reduction in LDL-cholesterol among patients with acute MI after 12 weeks of intervention (Singh *et al.*, 1992). All this may be possible because fruit and vegetables are rich in dietary fibre, which have been shown to decrease LDL-C concentrations (Ballesteros et al, 2002; Stone; 2001; Hagander *et al.*, 1998).

Douse *et al.* (2004) investigated whether the observed inverse association between fruit and vegetable consumption and LDL-C concentrations could simply be attributed to a lower intake of saturated fat and dietary cholesterol in subjects with higher fruit and vegetable intake. The data collected by Douse and co-workers (2004) showed that the intake of fruit and vegetables was associated with a lower intake of saturated fat, especially in men. This suggested that residual confounding by saturated fats could have biased their estimates. However, dietary cholesterol differs across categories of fruit and vegetable intakes in men and was in the opposite direction in women, because a higher intake of fruit and vegetables was related to higher dietary cholesterol in women. In addition, the inverse relationship between fruit and vegetable consumption and LDL-C concentrations was observed across all subjects whose energy intake from saturated fat was above the 75th percentile of the total population. This finding is not consistent with the fact that subjects with a higher intake of fruit and vegetables were more likely to eat less saturated fat and dietary cholesterol. Thus, the findings of Douse *et al.* (2004) are less likely to be attributable to the effect of substituting saturated fat and dietary cholesterol with fruit and vegetables. In conclusion, the data by Douse *et al.* (2004) shows that the consumption of fruit and vegetables is associated with lower concentrations of LDL-C.

2.6 CORPORATE WELLNESS

2.6.1 Introduction

Most employees in factories and companies have been forced into a sedentary lifestyle due to the technological progress and mechanisation that has changed work situations in industry (Blakeslee & Stailer, 1963). In this respect, Keeler (1980) maintained that the desk and revolving chair have become the biggest threats to executive health in modern time. This physical inactivity leads to a condition of hypokinesis, which can seriously harm human health (Mellerowicz, 1973; Grobler, 1990). This, together with other destructive lifestyle habits like cigarette- and/or tobacco smoke, unhealthy eating habits, alcohol abuse and stress, may have a direct effect on the healthcare costs, productivity and employee turnover of a company (Edington, 1986; Murphy *et al.*, 1987).

Shephard (1992) maintains that lowered productivity can be a result of qualitative and quantitative restrictions of the individual's performance, which manifests itself in absenteeism, staff turnover and illnesses. In most cases, absenteeism cannot be ascribed to specific health problems and, in some companies, is responsible for the loss of up to 220 work days per year. This constant fluctuation, particularly in terms of turnover of employees, is also a real problem at many companies, due to the high costs involved in the training of new employees (Shephard, 1987).

Anderson and Jose (1987) pointed out that the medical expenses of employees who regularly smoke are 18% per year higher than the expenses of non-smokers. Sedentary employees will also spend 30% more days in hospital than those who have maintained sufficient physical activity levels and that obese employees have a 48% bigger chance of incurring medical expenses of more than $5,000 per year, compared to employees who are not obese.

By contrast, there is a lot of research that indicates that participation in regular physical activity programmes can improve work productivity, stress handling, vitality and quality of life (Cox *et al.*, 1981; Pauly, 1981; Berlin, 1983; Johnson, 1983; Tsai *et al.*, 1987; Sarvela *et al.*, 1991; Shephard, 1992; Bouchard, 1994; Shephard & Bouchard, 1994; Pate, 1995; Shephard, 1999). In the long term, improved physical fitness may lead to the individual enjoying greater emotional stability, job satisfaction, improved work productivity, lower healthcare costs and lower absenteeism (Shephard, 1986; Falkenberg, 1987).

Walker *et al.* (1987) indicated that the implementation of fitness programmes can make an important contribution to the prevention of illnesses in general, as well to the improvement of health. Regular physical activity has a health improvement and CAD-protection effect in men (Francis, 1996; Thune *et al.*, 1998; Sesso *et al.*, 2000). Paffenbarger (1988) showed that the smoker and hypertension sufferer who is highly physically active has just about the same risk for CAD than the physically-inactive, non-smoker and normotensive person. This phenomenon is ascribed to the fact that regular physical activity contains an inherent protection mechanism by combating the patho-physiological factors that lead to the development of CAD (Paffenbarger, 1988; Thune *et al.*, 1998).

Both physiologically and psychologically, there exist no real substitutes for regular exercise and physical activity (Paffenbarger, 1987a; Unger, 1995). The development of CAD goes hand in hand with physiological and psychological risk factors respectively, factors that may be influenced significantly by a physically-active lifestyle (Nieman, 1998; Stoney & Hughes, 1999).

From the literature it is therefore clear that companies have to take active steps in caring for their most important commodity, namely their employees. It can be no coincidence, then, that since the mid-1970s, companies in the USA have started to show an increased interest in industrial fitness programmes (Friedman, 1986). The primary objective of such physical conditioning programmes were

mainly to combat certain financial expenses, such as increasing healthcare costs, lower productivity, increase in employee resignations and short-term absenteeism (Edington, 1986; Eakin *et al.*, 1988; Cox *et al.*, 1988). Almost certainly, this financial motivation led to the fact that 90% of companies that were evaluated in the USA were offering at least one health-improving activity to its executives (Messer *et al.*, 2000).

A year-long study at Johnson & Johnson on 884 employees taking part in a fitness programme showed that even the participants who exercised once a week as part of the programme, managed to cut their average number of sick days in half, from more than 10 days in the previous year to less than 5 sick days (Parks & Steelman, 1997). Coca Cola reported a reduction in healthcare claims, with an exercise programme alone, saving $500 per employee per year for the employees (60%) who joined their "Health Works" fitness programme (Wellness Councils of America, 1995). City Bank launched a comprehensive health-management programme that demonstrated that, for every dollar invested in programming activities, $4.56 to $4.73 was saved in reduced health-care costs (Ozminkowski *et al.*, 1999). The Coors Brewing Company in the USA has an onsite wellness centre that has been offering fitness programmes since 1981. According to Stanaland and Gelb (1995), the company has averaged a return on investment of $6.15 for every dollar invested in wellness programming. Financial savings have been in the form of lower medical costs, reduced absenteeism and increased productivity.

In South Africa (SA), company fitness programmes started as far back as 1982, aiming mainly at employees working in middle- to top management. According to data collected by the Biokinetics Association of SA, work situations in South Africa put exceptional pressure on company executives, especially if it is taken into account that the average working week of such executives amounts to more than 52 hours per week. This leaves very little time to participate in organised sport or exercise, a situation that often leads to the executives neglecting themselves physically, personally and otherwise (Uys & Coetzee, 1989).

Research by Strydom and Delport (1986) has shown that a large percentage of executives in SA display CAD factors and are physically inactive and unfit. Dreyer *et al.* (1988) also found that executives are overworked and disgruntled with their working conditions. All of the factors above can cause huge financial expenditures for any company, because they can negatively affect productivity, staff turnover, absenteeism and healthcare costs (Shephard, 1999).

Ozminkowski *et al.* (1999) stated that corporate wellness programmes varied tremendously in their design, comprehensiveness, intensity and impact and that a single model for a successful wellness programme doesn't exist. Ferreira (2006) investigated several wellness models developed over the years and came to the conclusion that each of these programmes encompasses a varying number of components or dimensions. The Ardell Wellness Model (1986) and the John and Janice Fisher Institute Model, cited in De Jager (2002) were analysed in this study. According to Ferreira (2006), the eight dimensions of wellness identified in the literature are spiritual, emotional, intellectual, physical, occupational, social, environmental and cultural. The South African companies surveyed focus on only four of these dimensions. These dimensions were physical, through exercise and fitness; occupational, through occupational safety and ergonomics; social, through smoking sensations and alcohol abuse; and HIV/Aids awareness.

The spiritual, emotional, environmental and cultural dimensions that are lacking are important dimensions of wellness. A holistic wellness model interlinks the aforementioned dimensions with the intellectual, physical, social and occupational wellness. Ferreira (2006) developed a model which has occupational wellness in the centre, surrounded by seven other dimensions of wellness. In addition, financial wellness was added with its focus to ensure that employees manage their finances adequately, that their financial situation is stable and sustainable and that they are adequately remunerated for the amount and complexity of the work they perform.

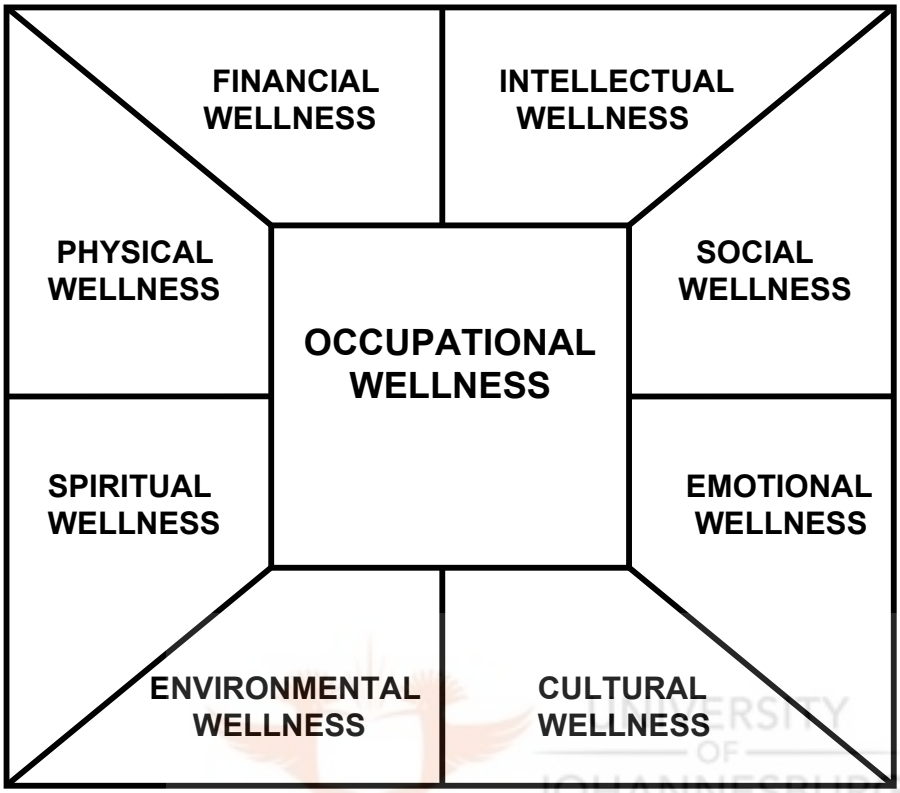

Figure 2.1 Ferreira's Corporate Wellness Model

This model can be applied to wellness programmes implemented and run in the corporate world, with each dimension included as a focal point, varying in intensity as defined by the wellness needs of the employees of the company for whom the wellness programme is designed.

2.6.2 Participation profile and physical work capacity of South African executives

From the literature, it is clear that specific exercise principles exist, which have a direct influence on the long-term outcome of an exercise programme. These principles are governed by the intensity, duration and frequency of participation, as well as the period of exercise and the type of activity in which people

participate (Davies & Convertino, 1975; Bouchard *et al.*, 1980; Pollock *et al.*, 1984; Strydom *et al.*, 1986). When participants do not get the "correct" quantity of exercise, no significant results can be expected. Paffenbarger (1987a) showed that people who maintained an energy consumption of 2000 kcal per/week in physical activity during their leisure time had a 39% lower risk of contracting CAD.

Very little information is available regarding the participation of executives in physical activity in SA. It was because of this, therefore, that Dreyer *et al.* (1988) conducted a qualitative and quantitative study on the participation profile and physical work capacity of executives in a number of South African companies, as well as to establish the motivation for participation or non-participation. One hundred and fifty-seven senior officials from 12 companies of the mining, steel, construction and motor industries, along with academia, were invited to participate. The average age of the respondents in the survey was 44.6 years.

Seventy-one percent of the respondents were non-smokers. Sixty-four percent of the respondents indicated that they regularly participated in sport, while the remaining 36% never participated. The participation figure is relatively high when compared to a group of average South African executives. This average was investigated by Noakes *et al.* (1986), who pointed out that fewer than 26% of the men and 16% of the women above the age of 44 in three rural towns in the Western Cape participated effectively in physical activity.

As previously mentioned, the effectiveness of participation in sport and physical activity may be influenced by specific exercise principles like intensity, duration, frequency, type and period of exercise. Paffenbarger (1987) points out that, as the physical activity increases from 500 kcal/week to 3500 kcal/week, the mortality rate decreases accordingly. Studies of Harvard alumni with active lifestyles have shown that at a physical activity level of 2000 kcal/week, there was a 28% lower risk in comparison to the less active ones. According to Parr (1987), the intensity of 2000 kcal/week can be seen as the most favourable

energy consumption/benefit ratio. This intensity can, in some way, be seen as a threshold value.

As another method of quantifying physical activity participation in order to determine its effectiveness, Sharkey (1979) suggested a physical activity index (PAI). This index is calculated by multiplying the factor values that describe the intensity, duration and frequency of a specific activity with each other in order to obtain a numerical index value (Table 2.1).

Various researchers, like Pollock *et al.* (1978), ACSM (1986) and Snyman (1986), have already indicated that a frequency of 3-5 times per week, an intensity of 60-90% of maximum heart rate and a duration of 20-30 minutes are essential for creating meaningful conditioning effects. When this suggestion is quantified, by using the index suggested by Sharkey (1979), then 36 must be seen as the threshold value for effective participation (i.e. 3 x 3 x 3 = 36).

Table 2.1 Physical Activity Index (PAI) (Kasari, 1976)

Based on your regular daily activity, calculate your activity index by multiplying your score for each category (Score = Intensity x Duration x Frequency)

	Score	Daily activity
Intensity	5	Sustained heavy breathing and perspiration
	4	Intermittent heavy breathing and perspiration – as in tennis, racquetball
	3	Moderately heavy – as in recreational sports and cycling
	2	Moderate – as in volleyball, softball
	1	Light – as in fishing, walking
Duration	4	Over 30 min
	3	20 to 30 min
	2	10 to 20 min
	1	Under 10 min
Frequency	5	Daily or almost daily
	4	3 to 5 times a week
	3	1 to 2 times a month
	2	Few times a month
	1	Less than once a month

Evaluation and Fitness Category

Score	Evaluation	Fitness Category
100	Very active lifestyle	High
80 to 100	Active and healthy	Very good
40 to 60	Acceptable (could be better)	Fair
20 to 40	Not good enough	Poor
Under 20	Sedentary	Very Poor

Index score is highly related to aerobic fitness.

In theses studies, it was found that 40% of those who indicated that they participate in physical activity were not active enough to ensure meaningful physiological adjustments or exercise effects. On the other hand, in 60% of the respondents the participation was of such a standard that said exercise effects could well be obtained (Pollock *et al.*, 1978; ACSM, 1986; Snyman, 1986). This means that 39% of the total group of executives in this study participated "effectively" in physical activity. The participation of those who indicated that they did participate in physical activity, but achieved an index lower than 36 (40%), should actually be seen more as recreational participation. The latter should, however, not be downplayed as worthless, since there are indications in the literature that even low physical activity levels can give some "protection" against ischemic heart disease (Sobolski *et al.*, 1987).

The exercise regime, or activity profile, of persons could be an important indication of the risk of contracting CAD. In earlier literature, hypertension, hypercholesterolemia and smoking were pointed out as the so-called primary risk factors (Pollock *et al.*, 1978). In a study by Collingwood *et al.* (1987), 4 351 men and women were examined regarding 23 CAD risk factors, including blood lipids, blood pressure, family history, smoking habits, body composition, physical fitness, and stress- and exercise regime. From the results, it appeared that the lack of exercise could very well be a meaningful and primary CAD risk factor. Although the scope of the study was not such that the researchers could definitely confirm that lack of exercise was the most important risk factor, there is little doubt that it should be seen as one of the primary coronary risks (Paffenbarger *et al.*, 1986; ACSM, 2006).

Participation in physical activity, and not necessarily physical fitness, could have specific advantages and offer possible "protection" against CAD. This means that people in a profession requiring physical activity are less exposed to CAD than those who practice a sedentary profession. It seems that 49.3% and 36% of the executives in South Africa fall in the sedentary and occasionally-active groups as far as their physical activity at work is concerned. This means,

therefore, that 86.1% in of executives in South Africa are inactive during their day at work (Dreyer *et al.*, 1988).

The lowered activity level of executives in the Republic of South Africa during their work day, as well as after hours, is possibly the most important cause of their apparent diminished physical work capacity. According to Mellerowicz (1972), 3.0 - 3.5 W/kg must be seen as the average physical work capacity for men. Jones and Campbell (1982), on the other hand, accept 2.5 W/kg as an average for men. It would seem that only 11.2% of executives in South Africa show a physical work capacity of more than 3.0 W/kg, and 21.7% a PWC_{170} of more than 2.5 W/kg. This confirms the declared concern of managing directors regarding the poor physical condition of this group of managers.

In order to establish the reason for non-participation in physical activity, the time that the person spends at work was investigated. As mentioned, the total work involvement of a South African executive is approximately 52 hours per week, and if after-hour social events are added, it does indeed seem that the executive has very little time left for participation in physical exercise. People not participating in regular physical activities were asked to list a number of reasons for not exercising. Sixty percent indicated that “too little time” was an important reason for non-participation. A lack of interest (48%) and “too lazy” (40%) were also cited. Thus concerted efforts will have to be made by companies to establish suitable exercise facilities, as well as to inform their managerial corps about, and introduce them to, the benefits of a healthy and physically-active lifestyle (Dreyer *et al.*, 1988).

2.6.3 Benefits of an executive fitness programme

From the research literature, it would appear that physical fitness can offer a certain protection against loss of life as a result of CAD, even in the presence of the primary coronary risk factors like cigarette smoke, hypertension and increased serum cholesterol (Paffenbarger, 1987 b; Barlow *et al.*, 1990). Various

studies also show that an improvement in the employees' physical fitness gives rise to a decline in absenteeism, (Cox *et al.*, 1981; Fielding, 1982), staff turnover, (Tsai *et al.*, 1987), healthcare costs (Baun *et al.*, 1986) and an improved work performance (Bernacki & Baun, 1984).

Strydom *et al.* (1985) found that exercise programmes led by a full-time programme leader had a positive effect on executives. The positive effects were seen in the areas of cardio-vascular fitness improvements and a decrease in CAD risk factors.

In another study, Dreyer and Strydom (1992) investigated whether physical, physiological and personal benefits would result from an exercise programme performed by a large group of executives from twelve different companies, six months after these companies had implemented an on-site fitness programme. Significant ($p \leq 0.05$) improvements occurred in the PWC_{170} (2.2 - 2.6 W/kg), SBP (134 - 129 mm Hg), DBP (90-87 mm Hg), abdominal strength (28 - 35 sit-ups/min), sit-and-reach flexibility (35 - 38.9 cm) and body-fat percentage (13.9% - 12.7%). Non-significant changes occurred in total body mass. Some perceived benefits that can have an influence on the company's productivity and healthcare costs were also experienced by the participants. In this respect, 41.6% indicated that their job performance and their attitude towards their work improved, while 72% and 44.6% felt that they had more stamina and could manage work pressure better. Of the executives in this study, 79.6% and 77.4% indicated that their personal health attitude and consciousness of the importance of a healthy life-style improved. Seventy-nine percent of the executives also felt that such a programme was cost effective for the company. The demands for performance are high, and company executives are willing to make certain sacrifices in order to meet those demands. These executives work a 52-hour week or more, and have rated their priorities as being their job, then their spouses. Families come next, and the list is finished by health, sports, and relaxation (Uys & Coetzee, 1989).

The lack of importance that South African managers give to sport, recreation and health could relate to the large number of respondents who show coronary risk factors (Strydom *et al.*, 1988), the low percentage who participate regularly in physical activity, (Dreyer *et al.*, 1988) and the relatively-low physical ability of the group who were examined during this study. The average PWC_{170} of the respondents was 2.2 W/kg, which, according to Mellerowicz and Meller (1972), as well as Jones and Campbell (1982), can be seen as below-average performance.

In a similar study Dreyer and Strydom (1994) studied the PAI of 777 male South African executives and its relation with some selected CAD risk factors. The PAI was computed as the product of the intensity, frequency and duration of participation in physical activity during leisure time (Sharkey, 1984). Subjects were classified into percentiles low, moderate and high.

Subjects that expended more than 1500 kcal/week were classified as highly active, while those who expended less than 150 kcal/week were placed in the low-active group. Those who expended between 450 kcal/week and 1500 kcal/week were placed in the moderate category. Only 14.3% of the entire survey group expended more than 1500 kcal/week and 61% reported no participation or participation bellow 150 kcal/week. The sedentary existence of the group was reflected in the fact that their average PWC_{170} was 2.3 W/kg, which is lower than the norm for adult males (2.5 W/kg). High-intensity participation (1500 kcal/week) showed significant ($p \leq 0.05$) associations with age, percentage body fat, TC, HDL-C and TC/HDL-C.

Carrol (1980) pointed out that unfit workers are not only less productive, but that they are also prone to injuries and tiredness. Liberty Mutual Insurance noted that about 66% of the back injuries that were reported would not have happened if the workers were physically fit (Thomas, 1983). Shephard (1986) pointed out that an industrial fitness programme could decrease short-term absenteeism due to lower back pain through participation in a physical exercise programme that

included abdominal muscle strengthening and hamstrings and lower back muscle strengthening.

It is clear from the research literature that participation in physical activity can show a definite decline in both SBP and DBP, even in the absence of weight loss (Leon, 1983; Cooper, 1985). It also appears that participation in physical activity and the level of fitness show an inverse relationship with systolic and DBP values (Cooper, 1982; Phelps, 1987). Seen in the light of the fact that 35-45% of all cardiovascular mortality and morbidity could be attributed to hypertension, the possible contribution of a fitness programme to hypertension prevention cannot be disregarded. The Mutual Insurance Company was able to show a saving of $32,000 per year in 1985 through their hypertension intervention programme, which had a declining effect on the incidence of cardiovascular episodes (Smith, 1986).

A study was conducted by Laubscher *et al.* (2003) to examine the possible connection between physical activity, lifestyle and the health status of black, male, middle managers in the public sector in the North West Province of South Africa. The respondents were divided into three groups according to the PAI values of Sharkey (1997), namely inactive (0-16), moderately active (17-44) and highly active ($\geq$45). A PAI of 36 represents more or less the prescriptions of the American College of Sports Medicine (ACSM, 2006) for effective participation in physical activity. The "seven healthy lifestyle habits", according to Belloc and Breslow (1972), were used to categorise the lifestyle of the respondents. The respondents were divided into three groups on the basis of their compliance with healthy lifestyle habits. Respondents who followed 0-3 of these lifestyle habits were classified as having a poor lifestyle, while those who followed 4-5 and 6-7 of these lifestyles were classified as having moderate and healthy lifestyles respectively. The "Seriousness of Illness Rating Scale" by Wyler *et al.* (1968) was used to establish the health status of the respondents. The respondents were divided into three groups on the basis of the illness grading, namely a good ($\leq$134), moderate (135-294) and poor ($\geq$295) health status respectively. The

respondents who followed poor lifestyle habits also displayed a statistically-significant ($p \leq 0.05$) lower physical activity level. The middle manager who has a healthier lifestyle also had a statistically-significant ($p \leq 0.05$) higher quality of participation in physical activity. As far as health status was concerned, respondents who followed fewer than three of the habits had a statistically-significant ($p \leq 0.05$) weaker health status than the other two groups who followed more than four of these habits. It is therefore clear that lifestyle is a very important aspect to which individual middle managers must give attention to in order to maintain, or even improve, their health status.

2.7 SUMMARY

Good health and physical condition is a very important aspect of human life. The benefit of physical activity on primary and secondary CAD risk variables are evident throughout the literature reviewed in this study.

Nutrition also proves to play a vital role in reducing the chances of contracting CAD. Many new popular diets have emerged, and although some of these may offer health benefits, caution must be taken, as others may harm cardiovascular or overall health.

Good health not only has its proven benefits for the individual, but also for the organisation who the individual works for. Some of these benefits include reduction in absenteeism, employee turnover and medical costs. It has also proven to improve employee moral and productivity.

For years corporate wellness programmes have been implemented in the USA with great success, and although relatively new in South Africa, are proven to be just as popular here.

CHAPTER THREE: METHODOLOGY

3.1 Introduction

Significant quantities of time and money are being spent in South Africa, and around the world, in striving to improve the general health and wellness of employees (Bellingham & Cohen, 1987). A pressing question is, however, how effective these wellness programmes are. In this study, the success of such a wellness programme, implemented and managed over a period of four years (2002-2005), was investigated.

3.2 Aim of the research

The aim of this research was to determine whether the wellness programme implemented at Kumba Resources headquarters, South Africa, had a positive effect on twelve modifiable CAD risk factors of employees.

3.3 Research design

The present study used a sample of 76 employees on whom pre-tests were performed on twelve CAD risk variables, after which an intervention programme was introduced. Twelve months after the pre-test (2003), the first intermediate test was conducted using the same protocol. The second intermediate test followed in 2004, duplicating the procedures of the first intermediate test. The post-test was conducted four years after the pre-test and data was analysed in 2005. This can be seen in the diagram below.

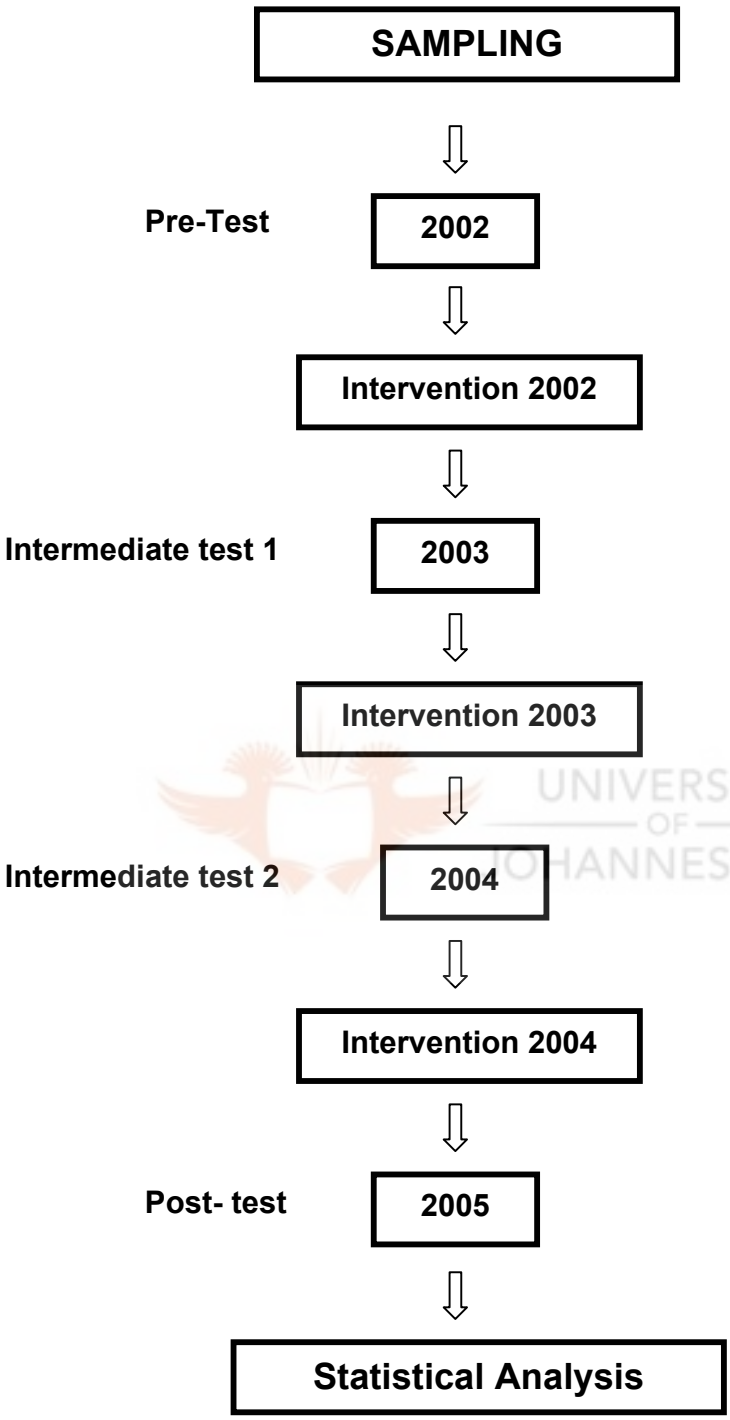

Figure 3.1: Research Design

3.3.1 Medical and fitness evaluations

The employees were required to attend, on a voluntary basis, an annual medical evaluation for the duration of the four year study period (2002 to 2005), conducted by an outside agency called Careways. The twelve modifiable CAD risk factors that formed part of the present investigation were: TC, HDL-cholesterol, LDL-cholesterol, triglycerides, TC/HDL ratio, LDL/HDL ratio, fasting blood glucose, SBP, DBP, BMI, WHR and waist circumference. Data collected in 2002 was used as the pre-test. The data collected during 2003 and 2004 was used as intermediate tests and the data collected during 2005 served as the post-test.

3.3.2 Interventions

Interventions included access to exercise facilities, recreational clubs and various health-related information sessions. In the gymnasium they had access to group training sessions (spinning, aerobics, callisthenics and stretching), a free-weight circuit and super circuit area. Cardiovascular equipment, such as treadmills, stationary bicycles and rowing machines, were also at their disposal.

The opportunity to monitor progress on-site was made possible in the form of regular screening tests. These tests included height, mass, BMI, waist circumference, hip circumference, WHR, double product, SBP, DBP, resting heart rate, % body fat, abdominal, lower- and upper body strength, blood glucose, cardiovascular risk profile and predicted $VO_{2\ max}$. According to their progress, adaptations were then implemented to the individual's current programmes.

Some recreational activities were also at the individual's disposal in the form of running, cycling, scuba diving and soccer clubs, managed by the fitness professional at Kumba headquarters. Employees had the opportunity to participate in these activities, which included various courses and information

sessions being presented in the above-mentioned disciplines. In addition, tours were organised to events like the Comrades Marathon, Two Oceans Marathon, Cape Argus Cycling Tour and many others, which were used to give the individuals a goal to strive for. Participants not necessarily participating in these specific sporting events were accommodated by alternative recreational activities in the form of river rafting and hiking.

Nutritional information sessions were presented to individuals who took part in the intervention programmes, by the fitness professional, on a monthly basis. Various efforts were also made to involve the contracted company who managed the restaurant.

3.3.3 Risk analysis

A novel CAD risk score was developed from peer-reviewed literature by considering each of the 12 CAD risk factors measured. Each risk variable was divided into three risk categories. This was done by awarding numbers 0 (desirable or low risk), 1 (borderline high or average risk) or 2 (high risk) to a specific CAD risk variable, depending on the health risk it posed. This procedure was applied to all twelve variables. The scores were added up, resulting in a "total CAD risk score" for each individual. If all 12 risk variables were taken into consideration and the worst-case scenario was calculated, the "total CAD risk score" would amount to "24" and the best-case scenario would amount to "0". The risk level, norm values and risk scores for each of the twelve CAD variables are provided in Table 3.1, with reference to the relevant literature sources for each.

Table 3.1 Risk scores attributed to different degrees of coronary artery disease risk factors

VARIABLE (unit)	RISK	NORM	RISK Score	SOURCE
1. TC (mmol/l)	Desirable	<5.2	0	JAMA (1993a); ACSM (2006)
	Borderline High	>5.2-6.2	1	
	High	>6.2	2	
2. LDL- Cholesterol (mmol/l)	Desirable	<3.4	0	JAMA (1993a); ACSM (2006)
	Borderline High	3.4-4.1	1	
	High	>4.0	2	
3. HDL-cholesterol (mmol/l)	Low risk	>1.55	0	JAMA (1993a); ACSM (2006)
	Average risk	0.91-1.55	1	
	High risk	<0.9	2	
4. LDL/HDL-cholesterol ratio	Normal risk	<3.5	0	Holford (1997); Williams (2002)
	High risk	>3.5	1	
5. TC/HDL-cholesterol ratio	Normal risk	<3:1	0	Bornow (1992); Powers and Dodd (1983)
	Borderline High risk	3:1-5:1	1	
	High risk	5:1-9:1	2	
	Very High risk	>9:1	3	
6. Triglycerides (mmol/l)	Normal risk	<2.0	0	JAMA (1993a)
	Boderline High risk	2.01-4.51	1	
	High risk	4.52-11.28	2	
	Very High risk	>11.28	3	
7. Fasting Blood Glucose (mmol/l)	Normal risk	3.3-6.1	0	Powers and Howley (1997); AACVPR (1999)
	Pre-diabetes	6.1-6.9	1	
	Diabetes	>6.9	2	
8. Resting SBP (mm Hg)	Normal risk	<120	0	Robbins *et al.* (2005); ACSM (2006)
	Prehypertention	120-139	1	
	Stage 1 Hypertension	140-159 mm Hg	2	
	Stage 2 Hypertension	160-180	3	
	Stage 3 Hypertension	>180	4	

VARIABLE (unit)	RISK	NORM	RISK Score	SOURCE
9. Resting DBP (mm Hg)	Normal risk	<80	0	Robbins *et al.* (2005); ACSM (2006)
	Pre-hypertension	80-89	1	
	Stage 1 Hypertension	90-99	2	
	Stage 2 Hypertension	100-110	3	
	Stage 3 Hypertension	>110	4	
10. BMI (kg/m²)	Normal risk	18.5-24.9	0	ACSM (2006)
	Overweight	25-29.9	1	
	Obesity Class 1	30-34.9	2	
	Obesity Class 2	35-39.9	3	
	Obesity Class 3	>40	4	
11. Waist Circumference (Men)	Normal risk	< 80	0	ACSM (2006)
	Average risk	80-99	1	
	Above Average risk	100-120	2	
	High risk	>120	3	
12. Waist-Hip-Ratio (Men)	Normal risk	<0.95	0	ACSM (2000)
	Average risk	0.96-1.00	1	
	High risk	>1.00	2	

3.4 Subject demographics

The data was collected from top- and middle management male employees at the Kumba Resources headquarters. Seventy-six of the initial 91 employees completed all assessments over the four-year study period. Subjects gave informed consent before each evaluation and have given consent for their data to be used for research purposes.

3.5 Assessment methods

Data collection was conducted by the Careways Medical Group, contracted by Kumba Resources, on an annual basis, spanning from 2002 to 2005. Exactly the same testing protocol was used each year.

3.6 Anthropometry

3.6.1 Height

Height was measured in centimetres (cm), using a wall-mounted steel stadiometer (to the nearest 0.1 cm). Participants were required to stand barefoot with heels together and buttocks and scapulae against the wall, and with their heads in the Frankfort plane (reference).

3.6.2 Weight

Weight was measured to the nearest 0.5 kg on a Detecto 449 digital electronic medical scale, (Webb City Missouri, USA), with the men only wearing a pair of shorts.

3.6.3 Body mass index

Body mass index was calculated using the formula: BMI (kg/m^2) = WT (kg) /HT^2 (m), where WT = body weight measured in kilograms and HT^2 = height squared measured in meters (Heyward, 1997).

3.6.4 Waist circumference

A non-distensible anthropometric tape measure was used to measure waist circumference in centimetres. The zero end of the tape was held in the left hand, positioned below the other part of the tape, which was held in the right hand. The tape was applied snugly around the waist at the level of the narrowest part of the trunk, level of the "natural" waist between the ribs and iliac crest. The measurement was taken at the end of normal expiration (Callaway *et al.*, 1988).

3.6.5 Hip circumference

A non-distensible tape measure was used to measure the hip circumference in centimetres. The zero end of the tape was held in the left hand, positioned below the other part of the tape, which was held in the right hand. The tape was applied snugly around the maximum posterior extension of the buttocks. An assistant was used to position the tape on the opposite side of the body (Callaway *et al.*, 1988).

3.6.6 Waist-to-hip-ratio

The WHR was calculated by dividing waist circumference (cm) by hip circumference (cm) (Bray & Gray, 1998).

3.7 Blood pressure

Blood pressure was measured with a stethoscope and standard Mercury sphygmomanometer. The subject was seated in a quiet room for at least five minutes. His left/right arm rested on a table so that the middle of the arm was at the level of the heart.

The brachial artery pulse was palpated on the anteromedial aspect of the arm below the belly of the bicep brachii and two to three cm above the antecubital fossa. The deflated cuff was firmly wrapped around the upper arm so that the midline of the cuff was covering the brachial artery pulse. The lower edge of the cuff was placed approximately 2.5 cm above the antecubital fossa. The valve was closed and the cuff was inflated quickly but steadily to approximately 150 mm Hg.

The valve was slowly opened, releasing the pressure at a rate of 2 to 3 mm Hg per second. Attention was paid to the first sharp thud caused by the sudden rush

of blood when the artery opened. This is known as the first Korotkoff sound and corresponds with the systolic pressure.

The pressure was further reduced by no more than 2 mm Hg per second, until the metallic tapping sound become muffled (phase 4 diastolic pressure) and finally disappeared (phase 5 diastolic pressure). Phase 5 was used as an index for DBP. The cuff was further deflated for another 10 mm Hg to ensure that no additional sounds were heard. After 30 seconds, a second reading was taken, and the average was used as the measurement (Reeves, 1995).

3.8 Blood chemistry

Blood analysis was conducted by a commercial pathological laboratory. After a fasting period of nine hours, a qualified nurse took a sample of arterial blood. The blood was analysed to determine the TC, LDL-C, HDL-C, triglycerides and fasting blood glucose levels of each individual.

3.9 Statistical analysis

The data collected over the four-year period (2002 - 2005) was analysed by the University of Johannesburg's Statistical Consultation Services (STATCON). The first statistical measure applied to the data was the Repeated Measures General Linear Model Test (Pohlmann *et al.*, 1974). This test was used to determine whether the mean values of the 12 coronary artery disease risk variables (Table 3.1) collected over the four-year study period differed significantly from the pre- and post-tests. Included in the statistical analysis was the utilisation of percentage change to illustrate relative changes observed from the pre- to post-test periods. To calculate the percentage change, the pre- to post-test mean values of the 12 Coronary artery disease risk variables were used. A confidence level of 95% was used to determine statistical significance.

CHAPTER FOUR: RESULTS

4.1 INTRODUCTION

The study investigated the effect of a corporate wellness programme on 12 modifiable Coronary artery disease (CAD) risk factors in a study group constituting 76 male employees of Kumba Resources in Pretoria, South Africa, over a period of four years (2002 to 2005). During this period, these employees experienced instability in the work environment as a result of mergers and restructuring. This chapter will focus on the results emanating from a statistical analysis of each of these risk factors from a statistical perspective. Each risk variable was monitored annually and the outcomes are summarised in tabular and graphic format (Tables 4.3 to 4.26; Figures 4.1 to 4.14). This summarisation reflects the effects of annual medical evaluations, an on-site training facility, a full-time fitness professional and various recreational activities on each of these variables. Values were quantitatively tested for significant differences at the 95% level ($P < 0.05$). The mean subject characteristics (height, weight) and age range of the study group are summarised in Table 2.

Table 4.1 Subject demographics

Characteristic (unit)	Gender	N	Mean
Height (cm)	M	76	180.3 cm
Weight (kg)	M	76	90.6 kg
Age (yr)	M	76	45 yr's

4.2 Blood lipids

4.2.1 Total cholesterol

The TC mean value statistics, summarised in Table 3 and Figure 1, reveal significant change in TC levels during the study period (2002 - 2005). A significant decrease in mean levels of this risk variable was observed during both the pre- and post-test periods. A pre-test value of 5.51 mmol/l was measured in 2002. During the first intermediate test in 2003, a statistically significant decrease of 0.32 mmol/l (5.88%) was measured. Similarly, a 0.48 mmol/l (9.11%) reduction was observed during the post-test (2005). Thus, over the four-year study period, a total reduction of 0.77 mmol/l (13.98%) in mean TC levels was observed.

Table 4.2 Total cholesterol

Lipid Profile TC	**N**	**Mean (mmol/l)**	**SD**	**Min**	**Max**	**Difference (mmol/l)**	**%**	**P value**
2002	76	5.51	±0.923	3.57	8.07			
2003	76	5.19	±0.929	3.38	7.79	- 0.32	5.88	0.000*
2004	76	5.22	±0.954	2.97	7.59	+ 0.03	-0.57	0.722
2005	76	4.74	±0.920	2.60	7.43	- 0.48	9.11	0.000*
2002-2005						- 0.77	13.98	0.000*

* : Statistically significant difference (P < 0.05); *Abbreviations:* mmol/l = milli-mole per litre;
N = number of individuals in study group; SD = standard deviation.

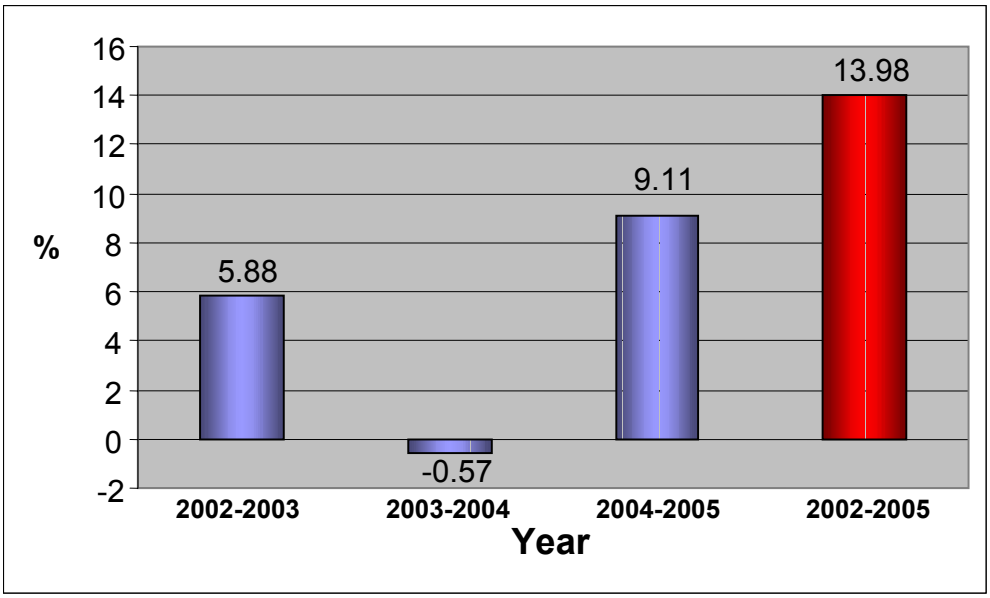

Figure 4.1 Percentage change in total cholesterol

4.2.1.1 Total cholesterol risk

Coronary artery disease risk factors, as outlined in Table 1, were evaluated in light of the three TC risk scores (desirable = 0, borderline high = 1 and high = 2). Table 4, below, summarises the number of individuals in each of these three categories during the first (2003) and second (2004) intermediate test periods, as well as at the time of the final evaluations in 2005. By 2005, a marked increase in the number of participants (29 to 54) in the desirable (i.e. 0) cholesterol risk category was observed, whereas only 5 of 19 initial participants (6.6%) remained in the high-risk category (2).

Table 4.3 Total cholesterol risk

		Risk Score			
Year		**0**	**1**	**2**	**N**
2002	*No. of individuals*	29	28	19	76
	%	38.2	36.8	25	100
2003	*No. of individuals*	41	25	10	76
	%	53.9	32.9	13.2	100
2004	*No. of individuals*	37	27	12	76
	%	48.7	35.5	15.8	100
2005	*No. of individuals*	54	17	5	76
	%	71.0	22.4	6.6	100

4.2.2 LDL-cholesterol

The mean LDL-cholesterol values of the 76 male participants measured over the four-year study period are summarised in Table 5 and Figure 2. Overall, the mean values decreased significantly by 0.65 mmol/l, (17.85%) from the pre- to post-test periods. This constitutes an 8.42% (0.32 mmol/l) reduction from the pre-test (2002) to first intermediate period (2003), and a 9.96% (0.33 mmol/l) reduction from the second intermediate (2004) to post-test period (2005). A slight and non-significant increase was observed in 2004, when compared to the previous year.

Table 4.4 LDL-cholesterol

LDL-Cholesterol	N	Mean (mmol/l)	SD	Min	Max	Difference (mmol/l)	%	P value
2002	76	3.64	±0.837	1.92	5.75			
2003	76	3.33	±0.872	1.77	5.46	- 0.30	8.42	0.000*
2004	76	3.32	±0.855	1.60	5.32	- 0.01	0.37	0.871
2005	76	2.99	±0.817	1.05	5.03	- 0.33	9.96	0.000*
2002-2005						-0.65	17.85	0.000*

* : Statistically-significant difference (P < 0.05); *Abbreviations:* mmol/l = milli-mole per litre; N = number of individuals in study group; SD = standard deviation.

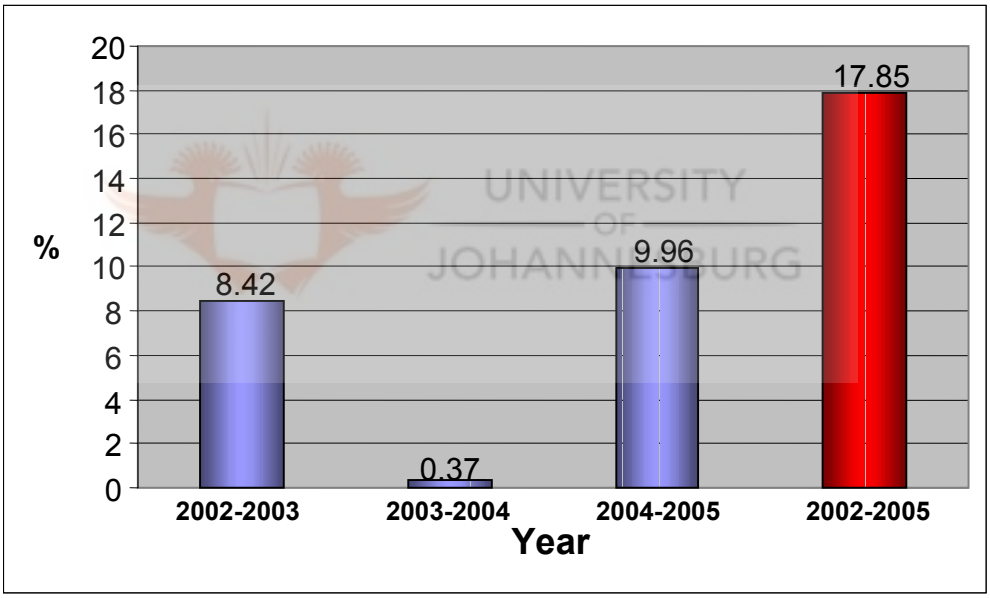

Figure 4.2 Percentage change in LDL-cholesterol

4.2.2.1 LDL-cholesterol risk

Coronary artery disease risk was interpreted in light of the mean LDL-cholesterol values measured across the entire group of male employees from 2002 to 2005 (Table 6). In the 2002 pre-test period, 31 (40.8%) of the participants were shown to have desirable levels of LDL-cholesterol, 25 (32.9%) were borderline high, and

20 (26.3%) had high levels. A steady improvement in LDL-cholesterol levels was noted during the first and second intermediate years, as fewer employees found themselves in the borderline high (1) and high-risk (2) categories. The final evaluations in 2005 saw a marked increase to 72.4% of participants in the desirable category, 19.7% in the borderline high, and only 7.9% in the high-risk category or above.

Table 4.5 LDL-cholesterol risk

		Risk Score			
Year		**0**	**1**	**2**	**Total**
2002	*No. of individuals*	31	25	20	76
	%	40.8	32.9	26.3	100
2003	*No. of individuals*	42	19	15	76
	%	55.3	25.0	19.7	100
2004	*No. of individuals*	37	23	16	76
	%	48.7	30.3	21.1	100
2005	*No. of individuals*	55	15	6	76
	%	72.4	19.7	7.9	100

4.2.3 HDL-cholesterol

The results presented in Table 7 and Figure 3 encapsulate the changes in mean HDL-cholesterol over the relevant study period in view of coronary artery disease risk. A significant overall decline in the mean HDL-cholesterol values measured across the 76 members of the study group was apparent from the pre-test to post-test periods (2002 and 2005). A pre-test value of 1.22 mmol/l was measured for the group in 2002, and was maintained at the same level in the following year. A marginal, non-significant increase was observed in the second intermediate year (2004), but culminated in a significant decrease in the mean value of 0.07 mmol/l (5.69%) at the time of the post-test (2005). An overall

decrease in the mean HDL-cholesterol 0.05 mmol/l (4.20%) was evident for the study group.

Table 4.6 HDL-cholesterol

HDL-C	N	Mean (mmol/l)	SD	Min	Max	Difference (mmol/l)	%	P value
2002	76	1.22	±0.261	0.73	2.09			
2003	76	1.22	±0.274	0.71	2.06	0.00	-0.48	0.794
2004	76	1.24	±0.260	0.83	1.99	+ 0.02	1.09	0.557
2005	76	1.17	±0.247	0.66	1.74	- 0.07	-5.69	0.000*
2002-2005						- 0.05	-4.20	0.018*

* : Statistically-significant difference (P < 0.05); *Abbreviations:* mmol/l = milli-mole per litre; N = number of individuals in study group; SD = standard deviation.

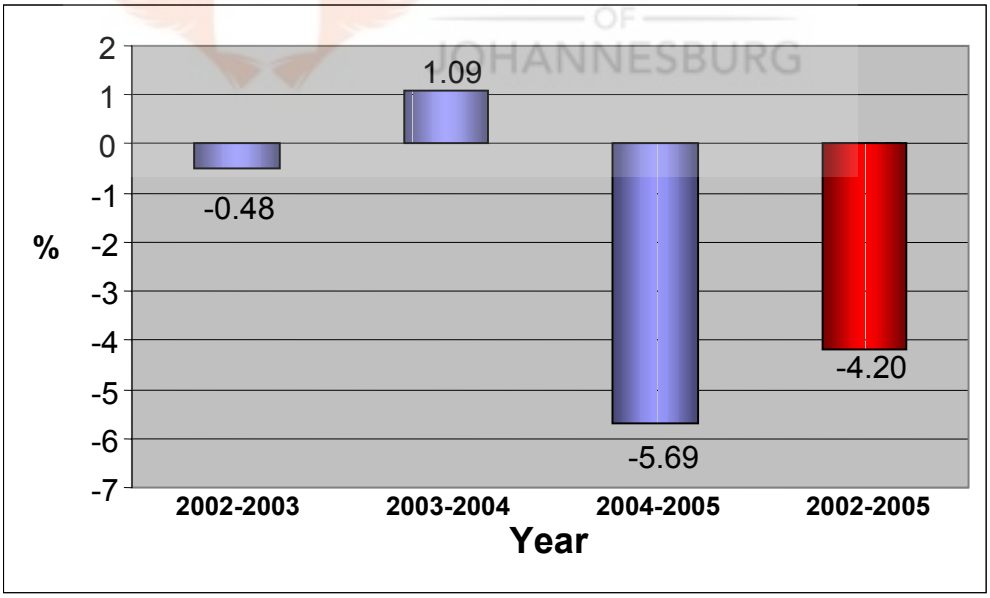

Figure 4.3 Percentage change in HDL-cholesterol

4.2.3.1 HDL-cholesterol risk

Coronary artery disease risk was interpreted from the perspective of the mean HDL-cholesterol values measured among study group members over the four-year period (Table 8).

Table 4.7 HDL-cholesterol risk

		Risk Score			
Year		**0**	**1**	**2**	**Total**
2002	*No. of individuals*	8	62	6	76
	%	10.5	81.6	7.9	100
2003	*No. of individuals*	9	60	7	76
	%	11.8	78.9	9.2	100
2004	*No. of individuals*	8	64	4	76
	%	10.5	84.2	5.3	100
2005	*No. of individuals*	5	61	10	76
	%	6.6	80.3	13.2	100

In the pre-test (2002), 8 of 76 participants (10.5%) scored in the desirable-risk category (0), 62 (81.6%) in the borderline-high category (1) and 6 (7.9%) found themselves in the high-risk category (2) or above. The number of participants in the desirable-risk category remained fairly stable in the first and second intermediate years (2003: 9 and 2004: 8), but decreased to 5 during the final evaluations in 2005. The number of participants in the borderline-high category (1) was relatively stable across consecutive years. An overall decrease in the number of participants in the high-risk category (2) was evident in the two years following the pre-test (2003: 7 or 9.2%) and (2004: 7 or 9.2%). However, at the time of final evaluation the number of employees in this category increased by 10 (13.2%) from the previous year.

4.2.4 LDL/HDL-cholesterol ratio

The results presented in Table 9 and Figure 4 summarise the change in the mean LDL/HDL-cholesterol ratio of the study group over the four-year period. A pre-test ratio of 3.12 was measured across members of the study group in 2002. A significant 0.28 (8.97%) decrease in the mean ratio was observed between the pre-test and first intermediate test, and again between the second intermediate test and the post-test (0.09 or 3.35%). At the time of final evaluation (2005) the mean LDL/HDL-cholesterol ratio measured across the 76 participants was significantly lower than in the pre-test according to the repeated measures GLM-test. Over the four-year period, the mean LDL/HDL-cholesterol ratio for the study group improved significantly by 14.03% (0.44).

Table 4.8: LDL / HDL-cholesterol ratio

LDL/HDL-C	N	Mean	SD	Min	Max	Difference	%	P value
2002	76	3.12	±1.00	1.02	6.52			
2003	76	2.84	±0.94	0.97	5.47	- 0.28	8.97	0.003*
2004	76	2.77	±0.88	1.01	6.10	- 0.07	2.29	0.360
2005	76	2.68	±0.98	0.93	5.94	- 0.09	3.35	0.235
2002-2005						- 0.44	14.03	0.000*

* : Statistically-significant difference (P < 0.05); *Abbreviations*; N = number of individuals in study group; SD = standard deviation.

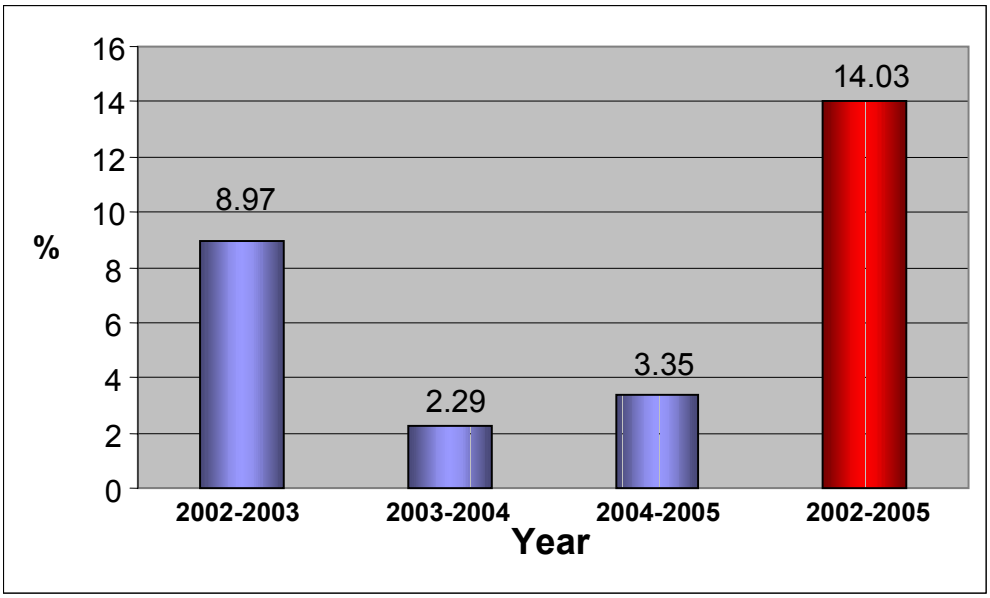

Figure 4.4: Percentage change in LDL/HDL-cholesterol

4.2.4.1 LDL/HDL-cholesterol ratio risk

Coronary artery disease risk as outlined in Table 1 was evaluated in light of the LDL/HDL-cholesterol ratio mean risk values measured across members of the study group from 2002 to 2005 (Table 10). The number of participants in the desirable-risk category (0) increased from 38 (50%) to 54 (71.1%) between the pre-test period and the time of final evaluation in 2005. An exception was in the second intermediate test (2004), when two more individuals tested in this category than in 2003. Simultaneously, a decrease in the number of participants that fell in the borderline-high category was evident each year, except for 2004 (2nd intermediate test), when 3 more participants found themselves in this category than the previous year. Two individuals (2.6%) consistently tested in the high-risk category (2) and above throughout the study, except in 2004 when only a single individual scored in this category.

Table 4.9: LDL / HDL-cholesterol ratio risk

		Risk Score			
Year		**0**	**1**	**2**	**Total**
2002	*No. of individuals*	38	36	2	76
	%	50	47.4	2.6	100
2003	*No. of individuals*	48	26	2	76
	%	63.2	34.2	2.6	100
2004	*No. of individuals*	46	29	1	76
	%	60.5	38.2	1.3	100
2005	*No. of individuals*	54	20	2	76
	%	71.1	26.3	2.6	100

4.2.5 TC/HDL-cholesterol ratio

Examination of the TC/HDL-cholesterol ratio mean values for the study group (Table 11 and Figure 5) revealed that significant changes in this variable took place between the pre- and post-test periods. In 2002, a pre-test mean value of 4.70 mmol/l was measured. A significantly reduced mean value of 0.30 mmol/l (6.39%) was measured in the following year during the first intermediate test. Although the mean ratios were slightly lower at the time of the second intermediate test, as well as the post-test, it was not statistically meaningful. Nonetheless, an overall decrease of 0.48 mmol/l (10.13%) in the mean TC/HDL-cholesterol ratio of the study group was recorded over the four-year period.

Table 4.10: TC/HDL-cholesterol ratio

Ratio TC/HDL	N	Mean	SD	Min	Max	Difference	%	P-value*
2002	76	4.70	1.253	2.11	8.70			
2003	76	4.40	1.155	2.15	7.29	- 0.30	6.39	0.003*
2004	76	4.35	1.079	2.12	7.81	- 0.05	1.26	0.470
2005	76	4.22	1.249	2.30	9.41	- 0.13	2.76	0.220
2002-2005						- 0.48	10.13	0.000*

* : Statistically -significant difference (P < 0.05); *Abbreviations:* N = number of individuals in study group; SD = standard deviation.

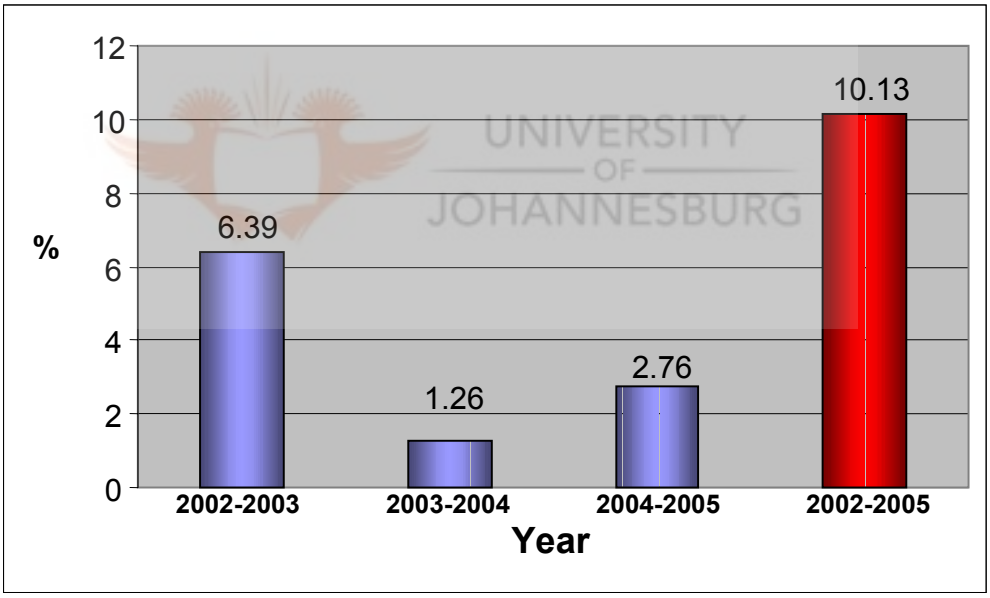

Figure 4.5: Percentage change in TC/HDL-cholesterol

4.2.5.1 TC/HDL-cholesterol risk

Coronary artery disease risk was evaluated in light of the mean TC/HDL-cholesterol ratio for the study group. Table 12 summarises the number of individuals in each of the three risk categories over the four-year period. By 2005,

a marked increase in the number of participants (3 to 15) in the desirable-risk category (0) was evident, and those in the high-risk category (2 or more) decreased from 28 to 19 over the same period. In the first and second intermediate tests, the number of participants in the borderline-high category increased slightly, but eventually decreased to 42 individuals.

Table 4.11: TC/HDL-cholesterol ratio risk

		Risk Score			
Year		**0**	**1**	**2**	**Total**
2002	*No. of individuals*	3	45	28	76
	%	3.9	59.2	36.8	100
2003	*No. of individuals*	7	47	21	76
	%	9.3	62.7	28	100
2004	*No. of individuals*	8	46	22	76
	%	10.5	60.5	28.9	100
2005	*No. of individuals*	15	42	19	76
	%	19.7	55.3	25	100

4.2.6 Triglycerides

From the examination of triglycerides mean values summarised in Table 13 and Figure 6, it is evident that no significant changes took place in this risk variable during the four-year study period.

Table 4.12: Triglycerides

Triglyceride	N	Mean (mmol/l)	Std	Min	Max	Difference (mmol/l)	%	P-value*
2002	76	1.45	0.732	0.42	3.43			
2003	76	1.42	0.697	0.36	3.55	- 0.03	2.32	0.219
2004	76	1.42	0..723	0.49	3.63	0.00	0.33	0.935
2005	76	1.33	0.812	0.34	3.59	- 0.09	6.34	0.337
2002-2005						- 0.12	8.82	0.563

* : Statistically-significant difference (P < 0.05); *Abbreviations:* mmol/l = milli-mole per litre; N = number of individuals in study group; SD = standard deviation.

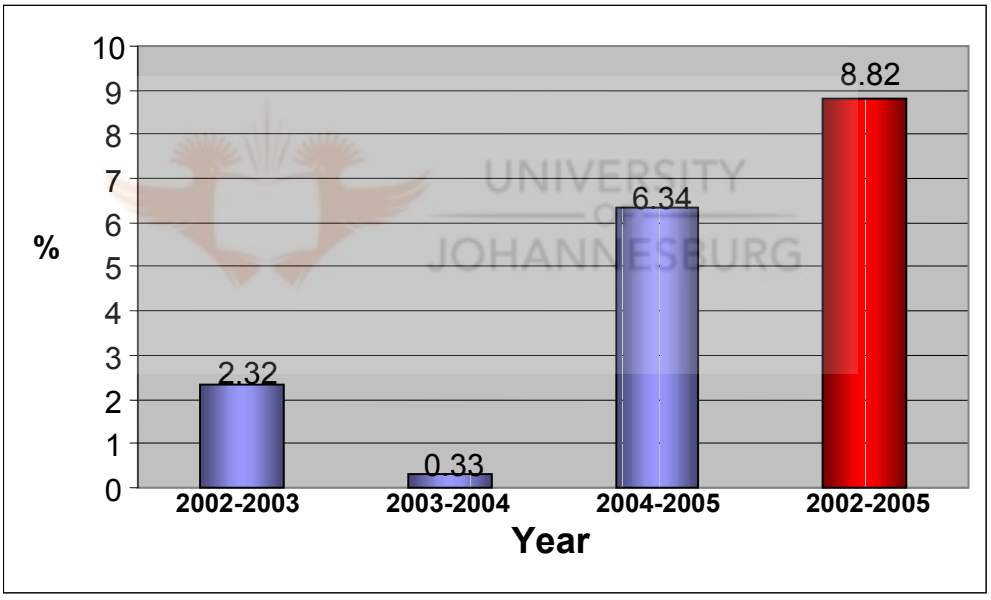

Figure 4.6: Percentage change in triglycerides

4.2.6.1 Triglycerides risk

Coronary artery disease risk factors as outlined in Table 1, were evaluated in light of triglyceride levels in the blood (Table 14). During the pre-test period in 2002, 62 (81.6%) of the 76 participants had desirable triglyceride levels. At the time of the first and second intermediate years, only one more employee scored

in the desirable-risk category (0), whereas one fewer participant fell in the borderline-high category (1). Notwithstanding, the final evaluations in 2005 saw a marked increase to 70 (92.1%) participants in the desirable category, and only 4 participants (5.3%) remained in the borderline-high category. It is noteworthy that in 2005, two of 76 participants scored in the high-risk category, while no individuals found themselves in this category in the previous three years.

Table 4.13: Triglycerides risk

		Risk Score			
Year		**0**	**1**	**2**	**Total**
2002	*No. of individuals*	62	14	0	76
	%	81.6	18.4	0	100
2003	*No. of individuals*	63	13	0	76
	%	82.9	17.1	0	100
2004	*No. of individuals*	63	13	0	76
	%	82.9	17.1	0	100
2005	*No. of individuals*	70	4	2	76
	%	92.1	5.3	2.6	100

4.3 BLOOD GLUCOSE

4.3.1 Fasting blood glucose

The results presented in Table 15 and Figure 7 summarise the change in fasting blood glucose levels across all 76 participants during the study period (2002 - 2005). During 2002, a fasting pre-test blood glucose mean value of 5.01 mmol/l was measured. At the time of the first and second intermediate tests (2003 and 2004) only slight, non-significant changes in these values were noted. However, a significant 0.35 mmol/l reduction in the mean fasting blood glucose levels was evident during the post-test (2005), which largely accounts for the decrease in

mean fasting glucose blood levels of the study group over the four years. An overall improvement of 7.43% resulted from a mean reduction of 0.37 mmol/l of this risk variable across members of the study group.

Table 4.14: Fasting blood glucose

Fasting Blood Glucose	N	Mean (mmol/l)	SD	Min	Max	Difference (mmol/l)	%	P-value
2002	76	5.01	±0.472	3.90	6.50			
2003	76	5.06	±0.609	3.00	7.30	+ 0.05	- 1.05	0.488
2004	76	4.99	±0.672	3.40	7.00	- 0.07	1.35	0.444
2005	76	4.64	±0.591	3.40	7.10	- 0.35	7.14	0.000*
2002-2005						- 0.37	7.43	0.000*

* : Statistically-significant difference (P < 0.05); *Abbreviations:* mmol/l = milli-mole per litre; N = number of individuals in study group; SD = standard deviation.

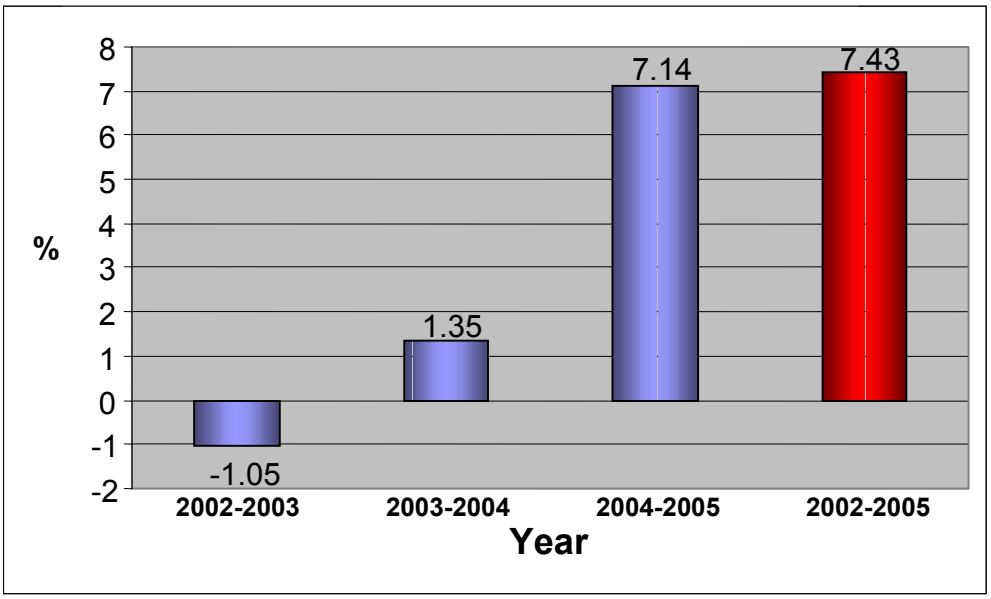

Figure 4.7: Percentage change in fasting blood glucose levels

4.3.1.1 Fasting blood glucose risk

Coronary artery disease risk factors (Table 1) were evaluated in light of fasting blood glucose risk scores. Table 16 summarises the number of individuals in each of these three categories during each year of the study period (2002 - 2005). All but 2 (2.6%) individuals tested in the desirable category for this risk factor in the pre-test. At the time of final evaluation (2005), only a single individual fell in the high-risk category and the remaining 75 in were all in the desirable category. It is noteworthy that 3 individuals showed an increase in fasting blood glucose levels during 2004 (2nd intermediate test).

Table 4.15: Fasting blood glucose risk

		Risk Scores			
Year		**0**	**1**	**2**	**Total**
2002	*No. of individuals*	74	2	0	76
	%	97.4	2.6	0	100
2003	*No. of individuals*	75	0	1	76
	%	98.7	0	1.3	100
2004	*No. of individuals*	73	2	1	76
	%	96.1	2.6	1.3	100
2005	*No. of individuals*	75	0	1	76
	%	98.7	0	1.3	100

4.4 RESTING BOOD PRESSURE

4.4.1 Systolic blood pressure

The mean SBP of the study group (measured as mm Hg), summarised in Table 17 and Figure 8, suggests that significant changes occurred from 2002 to 2005 with respect to this risk factor. Overall, a reduction of 6.34mm Hg (4.85%) was measured in the group as a whole. The mean pre-test value was 130.64 mm Hg in 2002. With the exception of 2004, the mean value decreased significantly in subsequent years (2003: 26 mm Hg, or 2.5%; 2005: 3.01 mm Hg or 2.37%).

Table 4.16: Systolic blood pressure

Systolic Blood Pressure	N	Mean (mm Hg)	SD	Min	Max	Difference (mm Hg)	%	P-value
2002	76	130.64	±11.69	109.0	170.0			
2003	76	127.38	±11.46	103.0	194.0	- 3.26	2.50	0.046*
2004	76	127.31	±11.46	106.0	160.0	- 0.07	0.05	0.966
2005	76	124.30	±10.89	100.0	144.0	- 3.01	2.37	0.039*
2002-2005						- 6.34	4.84	0.022*

* : Statistically-significant difference (P < 0.05); *Abbreviations:* mm Hg = millimeter Mercury;
N = number of individuals in study group; SD = standard deviation.

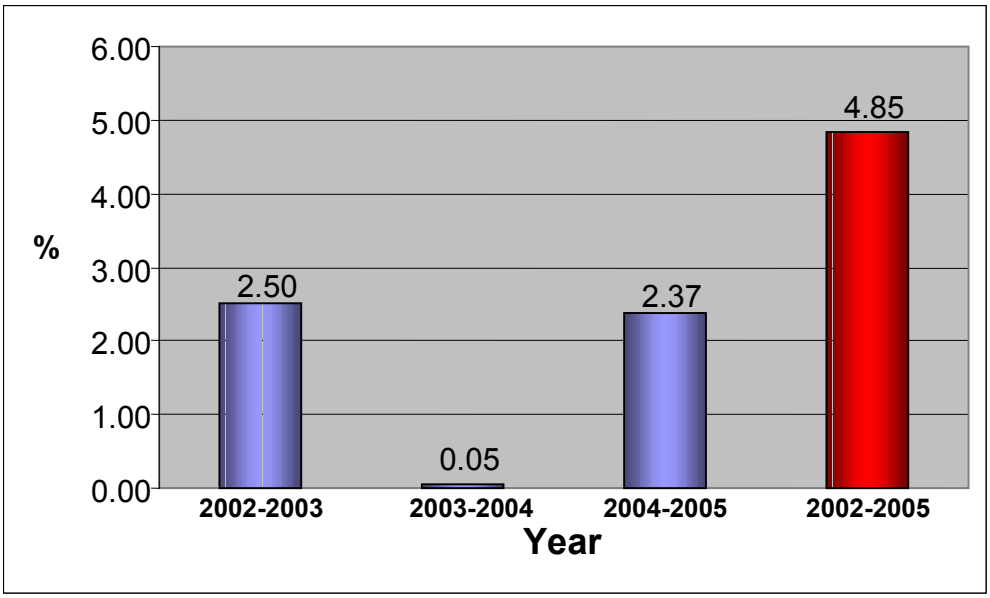

Figure 4.8: Percentage change in systolic blood pressure

4.4.1.1 Systolic blood pressure risk

Coronary artery disease risk was interpreted in light of the mean SBP of the 76 males participating in the study (Table 18). In the pre-test (2002), 12 individuals' (15.8%) SBP fell in the desirable-risk category, 45 (59.2%) had a borderline high-risk profile, while 19 were placed in the high-risk category for contracting coronary artery disease. After the four-year period, the risk profile of all participants had improved at the time of the first intermediate test (2003). In the second intermediate year (2004), two more individuals saw themselves in the high-risk (2) category than the previous year. Nonetheless, at the time of final evaluations (2005), more than 100% more individuals (25) fell in the desirable-risk category than in the pre-test (2002), whereas only four participants (5.3%) remained in the high-risk category.

Table 4.17: Systolic blood pressure risk

		Risk Scores			
Year		**0**	**1**	**2**	**Total**
2002	*No. of individuals*	12	45	19	76
	%	15.8	59.2	25	100
2003	*No. of individuals*	18	50	8	76
	%	23.7	65.8	10.5	100
2004	*No. of individuals*	18	48	10	76
	%	23.7	63.2	13.2	100
2005	*No. of individuals*	25	47	4	76
	%	32.9	61.8	5.3	100

4.4.2 Diastolic blood pressure

The mean DBP of the 76 participants was measured over the four-year study period (Table 19 and Figure 9). It showed a non-significant decrease of 2.82 mmolHg (3.63%) over the period of the study. During 2002, a pre-test mean of 77.82 mm Hg was measured for the group as a whole. A slight, but non-significant, decrease was observed during the first and second intermediate tests when compared to each other and to the pre-test value. However, the mean DBP measured across all participants in 2005 was significantly lower than the previous year (2003).

Table 4.18: Diastolic blood pressure

DBP	N	Mean (mm Hg)	SD	Min	Max	Difference (mm Hg)	%	P value
2002	76	77.82	9.12	58.00	100.00			
2003	76	77.26	6.78	62.00	96.00	- 0.56	0.73	0.531
2004	76	77.38	6.67	65.00	94.00	+ 0.12	-0.15	0.877
2005	76	75.00	6.64	62.00	91.00	- 2.38	3.08	0.006*
2002-2005						- 2.82	3.63	0.020

* : Statistically-significant difference (P < 0.05); *Abbreviations:* mm Hg = millimetre Mercury; N = number of individuals in study group; SD = standard deviation.

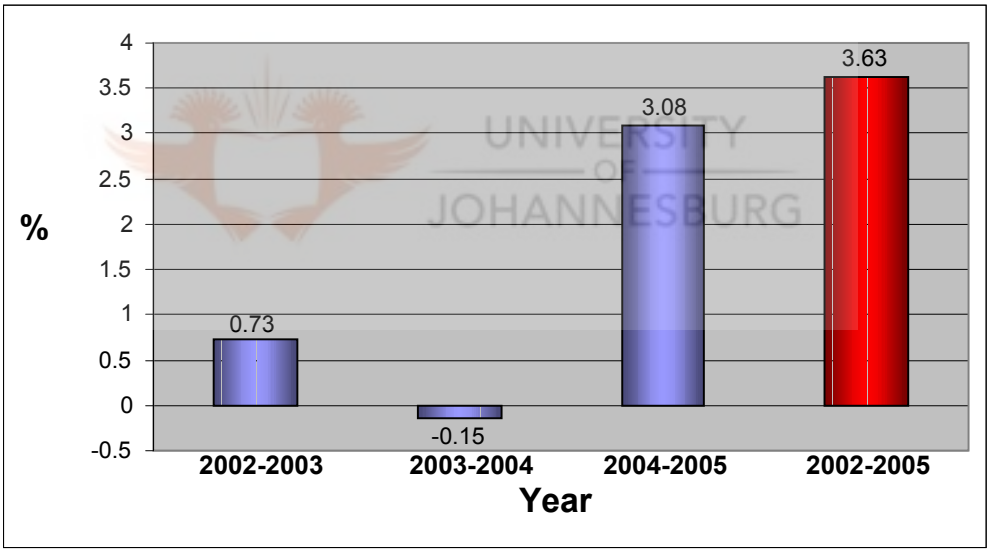

Figure 4.9: Percentage change in diastolic blood pressure

4.4.2.1 Diastolic blood pressure risk

Coronary artery disease risk factors (see Table 1) were evaluated in light of the mean DBP measured across the study group from 2002 to 2005 (Table 20). In the 2002 pre-test period, 46 (60.5%) of the participants were shown to have desirable levels of LDL-cholesterol, 22 (28.9%) had borderline-high-, and eight

(10.5%) high levels. A steady improvement in DBP was noted during the first intermediate year, as fewer employees found themselves in the borderline-high category, and only a single individual remained in the high-risk category. At the time of the second intermediate test (2004), two fewer individuals (59.2%) scored in the desirable category and three more individuals (5.3%) scored in the high-risk category than the previous year. The final evaluations in 2005 saw a substantial increase to 72.4% of participants scoring in the desirable category, 26.3% in the borderline-high and only 1.3% (one individual) remained in the high-risk category.

Table 4.19: Diastolic blood pressure risk

		Risk Score			
Year		**0**	**1**	**2**	**Total**
2002	*No. of individuals*	46	22	8	76
	%	60.5	28.9	10.5	100
2003	*No. of individuals*	47	28	1	76
	%	61.8	36.8	1.3	100
2004	*No. of individuals*	45	27	4	76
	%	59.2	35.5	5.3	100
2005	*No. of individuals*	55	20	1	76
	%	72.4	26.3	1.3	100

4.5 BODY COMPOSITION

4.5.1 Body mass index

An examination of the mean BMI of the study group (Table 21 and Figure 10) revealed a significant improvement in this risk variable from 2002 to 2005. In 2002, a pre-test value of 27.58 kg/m² was measured for the group as a whole.

No significant improvement was observed in 2003 (first intermediate test) and 2005 (post-test), but in 2004 the group's mean BMI was 1.02% lower (0.28kg/m^2) than the previous year. An overall reduction of 1.81% (0.5 kg/m^2) in mean BMI was evident after the four-year period.

Table 4.20: Body mass index

Body Mass Index	N	Mean (kg/m^2)	SD	Min	Max	Difference (kg/m^2)	%	P value
2002	76	27.58	±4.80	19.00	44.70			
2003	76	27.59	±4.76	18.20	46.28	+ 0.01	-0.02	0.961
2004	76	27.87	±4.99	19.10	47.20	+ 0.28	-1.02	0.013*
2005	76	28.08	±4.85	18.80	45.40	+ 0.21	-0.76	0.119
2002-2005						+ 0.50	-1.81	0.012*

* : Statistically-significant difference (P < 0.05); *Abbreviations:* kg/m^2 = kilogram per meter squared; N = number of individuals in study group; SD = standard deviation.

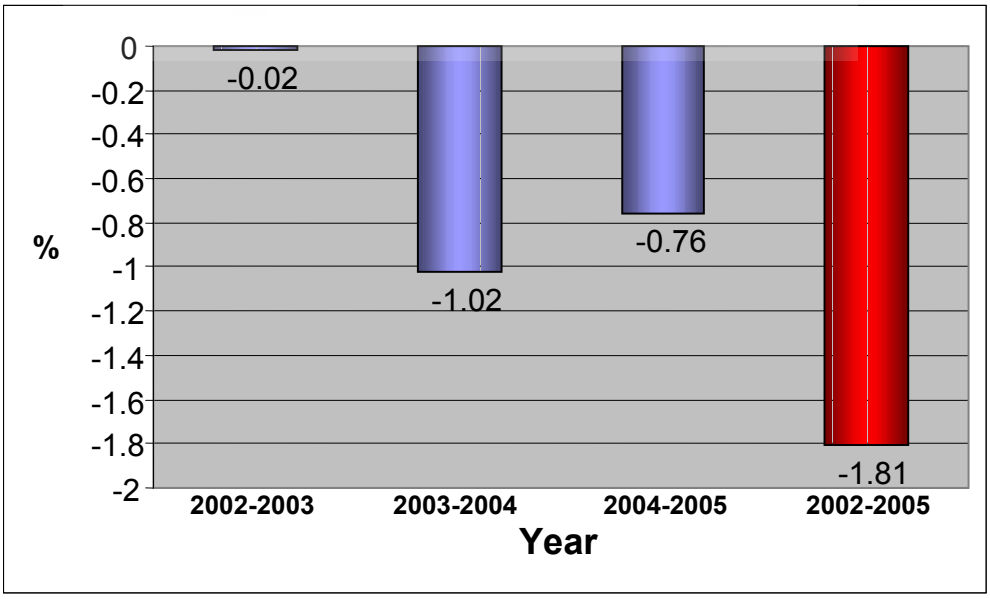

Figure 4.10: Percentage change in BMI

4.5.1.1 Body mass index risk

Coronary artery disease risk was interpreted in light of the mean BMI measured across the study group from 2002 to 2005 (Table 22). In the pre-test (2002), 24 (31.6%) of the participants fell in the desirable-risk category, 37 (48.7%) had a borderline-high BMI risk, and 15 (19.7%) high BMI risk levels. No marked improvement in BMI risk was detected from the pre- to post-test periods. The number of individuals in the desirable category decreased from 24 to 19, those in the borderline-high category was only slightly higher, whereas three more individuals finished in the high-risk category than at the onset of the study.

Table 4.21: Body mass index risk

		Risk Score			
Year		**0**	**1**	**2**	**Total**
2002	*No. of individuals*	24	37	15	76
	%	31.6	48.7	19.7	100
2003	*No. of individuals*	23	36	17	76
	%	30.3	47.4	22.4	100
2004	*No. of individuals*	20	37	18	75
	%	26.7	49.3	24.0	100
2005	*No. of individuals*	19	39	18	76
	%	25.0	51.3	23.7	100

4.5.2 Waist circumference

The results presented in Table 23 and Figure 11 summarise the change in mean waist circumference of the study group over the four-year study period. In 2002, a pre-test value of 96.85 cm was measured for the group as a whole. No significant reduction in waist circumference was obvious from mean values measured during the first and second intermediate tests, according to the

Repeated Measures General Linear Model Test. However, the mean waist circumference of the group increased by 1.75 cm (0.79%) at the time of final evaluations (2005) and by 2.68 cm (2.77%) over the four-year period.

Table 4.22: Waist circumference

Waist Circumference	**N**	**Mean (cm)**	**SD**	**Min**	**Max**	**Difference (cm)**	**%**	**P value**
2002	76	96.85	12.71	72.50	134.00			
2003	76	97.65	12.29	73.00	136.00	+ 0.80	0.26	0.063
2004	76	97.78	16.67	74.00	143.00	+ 0.13	0.30	0.917
2005	76	99.53	12.81	76.50	139.00	+ 1.75	4.11	0.167 *
2002-2005						+ 2.68	4.66	0.000 *

* : Statistically significant difference (P < 0.05); *Abbreviations:* cm = centimetres;
N = number of individuals in study group; SD = standard deviation.

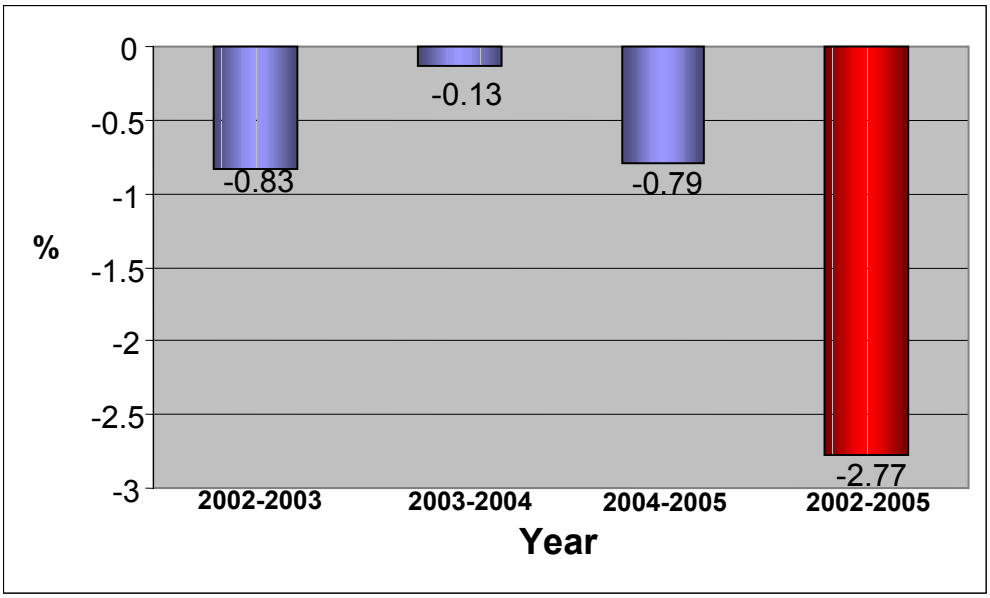

Figure 4.11: Percentage change in waist circumference

4.5.2.1 Waist circumference risk

The results presented in Table 24 and Figure 3 encapsulates the number of participants resorting in each of the three risk categories with respect to their mean waist circumference over the study period. In the pre-test (2002), only three (3.9%) individuals portrayed desirable levels of LDL-cholesterol, 45 (59.2%) borderline-high, and 28 (36.8%) high levels of this risk variable. The final evaluations in 2005 showed very little improvement on the pre-test. Only a single participant acquired desirable waist circumference and individuals resorting in the high-risk category or above escalated by 8%, to include 44.7% of the group as a whole.

Table 4.23: Waist circumference risk

		Risk Score			
Year		**0**	**1**	**2**	**Total**
2002	*No. of individuals*	3	45	28	76
	%	3.9	59.2	36.8	100
2003	*No. of individuals*	4	39	33	76
	%	5.3	51.3	43.4	100
2004	*No. of individuals*	4	38	34	76
	%	5.3	50.0	44.7	100
2005	*No. of individuals*	4	38	34	76
	%	5.3	50	44.7	100

4.5.3 Waist-to-hip-ratio

The WHR statistics for the study group (Table 25 and Figure 12) improved significantly by 4.66% over the four-year study period. In the pre-test (2002), the mean WHR risk factor was 1.00, which was reduced to 0.95 (4.11%) at the time

of the post-test (2005), although the improvements noted in each intermediate year (2003, 2004) were non-significant.

Table 4.24: Waist-to-hip ratio

Waist-Hip-Ratio	N	Mean	SD	Min	Max	Difference	%	P value
2002	76	1.00	±0.057	0.85	1.14			
2003	76	0.99	±0.052	0.86	1.13	- 0.01	0.26	0.635
2004	76	0.99	±0.053	0.82	1.08	- 0.00	0.30	0.529
2005	76	0.95	±0.061	0.79	1.13	- 0.04	4.11	0.000*
2002-2005						- 0.05	4.66	0.000*

* : Statistically-significant difference (P < 0.05); *Abbreviations*; N = number of individuals in study group; SD = standard deviation.

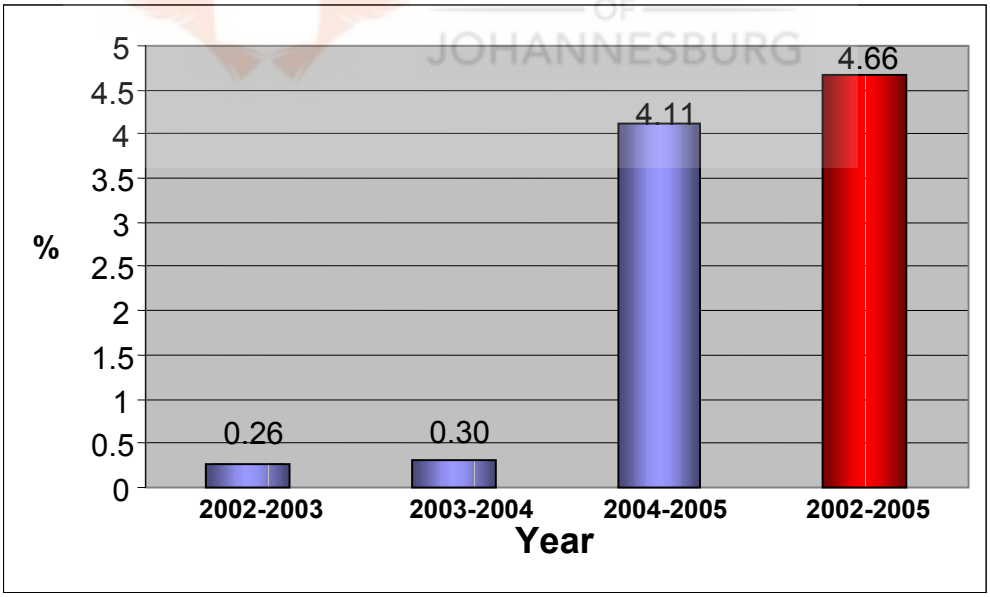

Figure 4.12: Percentage change in WHR

4.5.3.1 Waist-to-hip ratio risk

Coronary artery disease risk was evaluated in relation to the mean WHR of the study group. Table 26 summarises the number of individuals in each of the three risk categories over the four-year period. The majority of change in all three risk categories can be attributed to that which occurred between the second intermediate test (2004) and the post-test (2005). The number of participants finishing in the desirable-risk category increased almost threefold between the pre- and post-test periods, from 13 (17.1%) in 2002 to 35 (46.1%) in 2005. Six more individuals fell in the borderline-high category in the post-test than at the onset in 2002 (comprising 36.8% of the group). The number of participants in the high-risk category was markedly reduced from 41 (53.9%) to a mere 13 (17.1%) at the end of the study period.

Table 4.25: Waist-to-hip ratio risk

		Risk Score			
Year		**0**	**1**	**2**	**Total**
2002	*No. of individuals*	13	22	41	76
	%	17.1	28.9	53.9	100
2003	*No. of individuals*	16	21	39	76
	%	21.1	27.6	51.3	100
2004	*No. of individuals*	17	19	40	76
	%	22.4	25	52.6	100
2005	*No. of individuals*	35	28	13	76
	%	46.1	36.8	17.1	100

4.6 CAD risk

The results of a composite examination of the 12 coronary artery disease risk variables (outlined in Table 1) are presented as a total CAD risk score (Table 27, Figures 13, 14). This score was calculated across the mean values of all 12 variables. Overall, it is evident that the mean CAD risk values decreased and that some significant changes had taken place in 9 of these variables over the relevant study period (2002 - 2005). The change observed from the pre-test (2002) to first intermediate period (2003) constituted a 9.93% (0.08) reduction in the mean values measured in the 76-member study group. A marginal, non-significant increase was subsequently observed in 2004 (second intermediate test), but culminated in a significant decrease of 1.59 (20.93%) at the time of the post-test (2005). Across the variables relevant to CAD risk, a highly-significant reduction of 2.07 or 25.57% was detected for the study group as a whole.

Table 4.26: Changes in CAD risk scores

Total Risk	N	Mean	Std	Min	Max	Difference	%	P value
2002	76	8.08	3.067	1	15			
2003	76	7.28	2.965	1	14	- 0.8	9.93	0.007*
2004	76	7.61	2.738	0	15	+ 0.33	-4.42	0.176
2005	76	6.01	2.490	1	12	- 1.6	20.93	0.000*
2002-2005	76					- 2.07	25.57	0.000*

* : Statistically-significant difference (P < 0.05); *Abbreviations:* N = number of individuals in study group; SD = standard deviation.

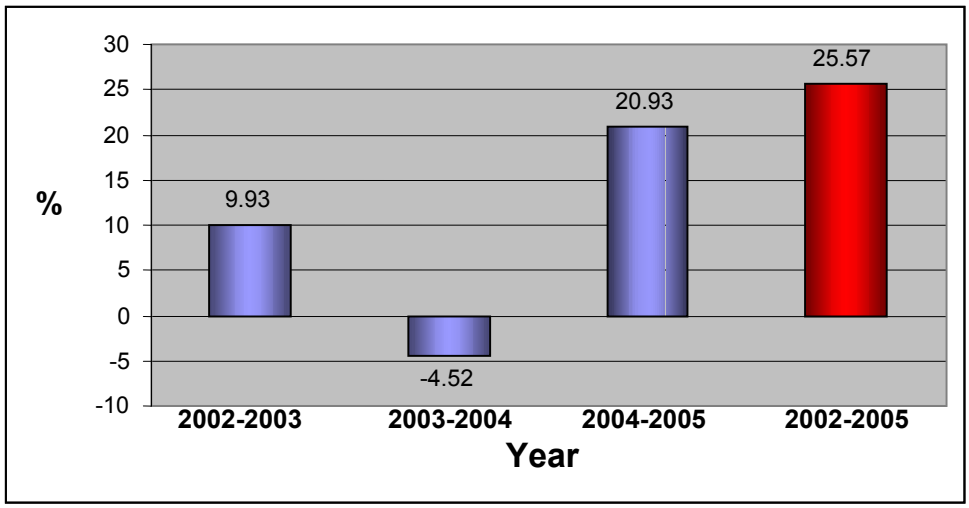

Figure 4.13: Percentage change in CAD risk

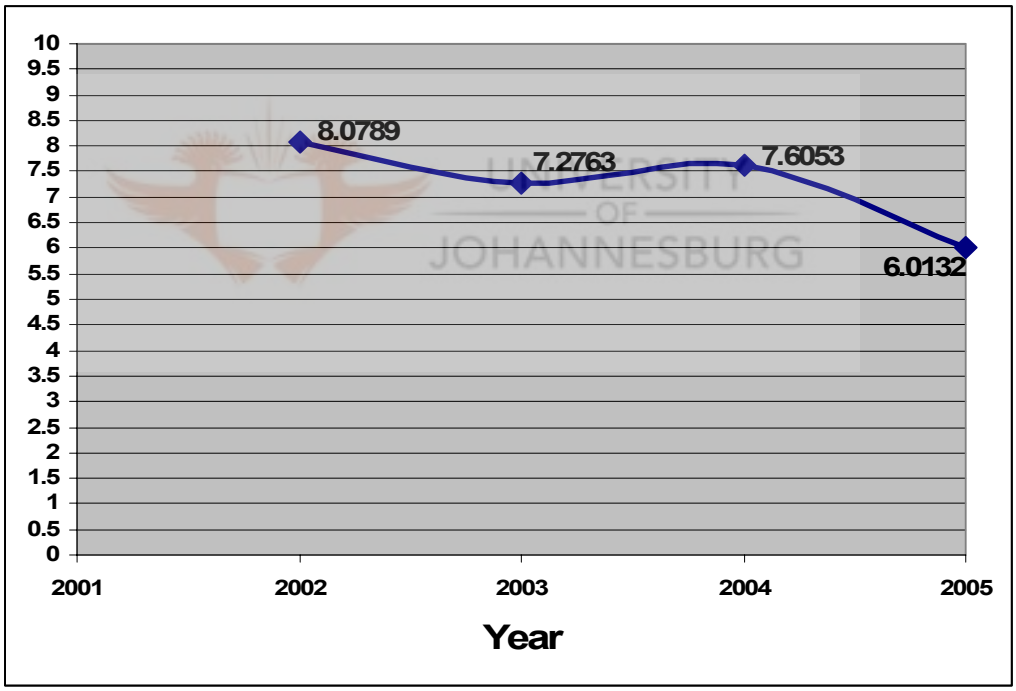

Figure 4.14 Changes in CAD risk

4.7 Summary

From the above results, it is evident that TC; LDL-C, low-density/high-density cholesterol ratio, total/high-density cholesterol ratio, fasting blood glucose, resting SBP, resting DBP, and WHR improved significantly during the study period, while triglycerides showed a non-significant improvement.

Four of the twelve risk variables did not show a significant improvement over the four-year study period. The three that actually deteriorated significantly during the study period were HDL-C, BMI and waist circumference. The fourth, triglycerides, did manage to improve, but the improvement failed to be significant.

The total CAD risk score improved significantly by 25.57% at the end of the four-year period from 2002 to 2005.

CHAPTER FIVE: DISCUSSION

5.1 Introduction

Research conducted on high-level employees in the South African corporate sector indicated that the working environment is often not beneficial to the health of the employees (Grobler, 1990; Boshoff, 2000).

Almost 97% of employees in management positions showed some definite risk factors for the development of CVD (Jacobs, 1991). According to wellness consultants, wellness programmes for executives and employees have become an extremely important factor in many South African companies such as Vodacom, Eskom and Murray and Roberts. This trend is also apparent in international companies such as General Electric, Johnson and Johnson and IBM (Bellingham & Cohan, 1987).

Since the early 1970s, several industrialised countries in the Western world have developed an interest in the influence of regular physical activity on employees. Where previously the emphasis in sport participation was driven by competition, the emphasis has shifted to the health-conservative nature of such programmes and the benefits relevant to a more efficient work force (Strydom *et al.*, 1985). The present study investigated the effect of an in-house wellness programme on twelve CVD risk variables over a four-year study period (2002 to 2005) at Kumba Iron Ore and Exxaro Resources Limited (formerly known as Kumba Resources Limited), a South African company that was subject to restructuring during this time. Although no distinction was made between employees who exercised and those who did not, the results clearly showed that the mere availability of such a programme had a positive effect on the majority of the CVD risk variables in the 76 participants. This chapter focuses on the discussion and interpretation of the findings of the current research in light of some similar research conducted in the past.

5.2 Dislipidimia

Dyslipidemia is associated with the progression of atherosclerosis, as well as endothelial dysfunction (Ansell *et al.*, 1999). Inactive individuals have been linked with elevated blood lipid levels when compared to more active individuals (Kesaniemi *et al.*, 2001).

5.2.1 Total cholesterol

A significant reduction in the TC levels of 13.98% of the participants observed in this study is supported by the results of previous research (Strydom *et al.*, 1985; Dreyer & Strydom, 1994; Andersen & Haraldsdottir, 1995; Van der Westhuizen, 1997). After the four-year study period, the mean TC level of participants in this study was significantly reduced by 0.77 mmol/l, a 14% reduction.

This improvement was adequate to move the variable from the borderline-high risk category (5.51 mmol/l) at the onset of the study, to the desirable risk category (4.74 mmol/l) at the end of the period. Although Strydom *et al.* (1985) failed to demonstrate a significant improvement in TC after a 24-week training programme, 26 male executives in the South African motor industry managed to reduce their TC levels, although not significantly. Dreyer and Strydom (1995) demonstrated a significant difference between the TC values (5.7 mmol/l) of employees considered highly active and expending more than1500 kcal/week during exercise, compared to the levels (6.0 mmol/l) of those considered inactive, expending less than 150 kcal/week. Van der Westhuizen (1997) demonstrated that active males had significantly lower TC levels compared to that of inactive males. Andersen and Haraldsdottir (1995) reported that males considered as being physically fit had lower TC values than those regarded as unfit, which further supports the hypothesis of this study that corporate wellness programmes would have a positive effect on the TC values of employees who actively participate in them.

5.2.2 LDL-cholesterol

The findings of this study revealed a significant decrease of 17.85% (0.33 mmol/l) in LDL-cholesterol during the study period. Once again the participants managed to reduce their values from above to below the normal risk levels of less than 3.5 mmol/l. Similarly, Shaw (2004) showed that a 16-week combination fitness and aerobic programme resulted in a decrease of 26.3% and 21.3% in LDL-cholesterol.

In the same study, an aerobic training programme resulted in a 21.3% decrease, and a dynamic resistance training programme in an 8.3% decrease in this risk variable.

Dreyer and Strydom (1992) also showed that employees who were classified as highly active, exerting more than 1500 kcal/week during exercise, had lower LDL-cholesterol levels (3.89 mmol/l) than their more sedentary colleagues (3.43 mmol/l) expanding less than 150 kcal/week.

The decrease in LDL-cholesterol following exercise training, as documented in the examples above, may be due to an increased activity of Cholesterylester Transfer Protein (CETP), Lecithin Cholesterol acyltransferase (LCAT) and Lipoprotein Lipase (LPL), which effectively increases lipoprotein metabolism (ACSM, 1997). These studies also confirm the findings of the present study in those employees who actively participate in physical and corporate wellness programmes, benefited by reducing their LDL-cholesterol levels and, as a result, their CVD risk profile is improved.

5.2.3 HDL-cholesterol

Contrary to the findings of some earlier studies on the effects of an exercise programme on HDL-cholesterol (Coopoo et al, 1995; Filipovsky *et al.*, 1991), this risk variable for CVD did not respond positively to the wellness programme

employed in the present study. At the end of the four-year study period, the HDL-cholesterol mean values of the study group deteriorated by 0.05 mmol/l (4.20%). However, even though they did not manage to improve their HDL-cholesterol values, the deterioration in this risk variable was not enough to place them in the high-risk category. These results are in agreement with the findings of Franklin *et al.* (2002), who investigated the effectiveness of a contemporary, exercise-based cardiac rehabilitation program on 117 patients who completed pre- and post-phase two evaluations. The exercise training programme involved three 45 to 60 minute sessions per week, at a minimum of 45%, to a maximum of 75% oxygen consumption, for six to eight weeks. The average HDL-cholesterol for their study group as a whole did not show any change, but it is notable that the HDL-cholesterol mean values of patients with abnormal baseline values improved by 2.8 mmol/l.

In sharp contrast, Coopoo *et al.* (1995) and Filipovsky *et al.* (1991) demonstrated significant increases in HDL-cholesterol levels following an exercise programme consisting of walk-jog or jogging aerobically for 30 minutes, in combination with 30 minutes of stationary cycling, or alternatively strength training and stretching in the gymnasium for 30 minutes in combination with 30 minutes of cycling. The majority of longitudinal studies have employed rather high-intensity exercise, most frequently jogging/running, but evidence is gradually becoming available that more accessible, self-governed exercise regimes may also be effective. Previously-sedentary middle-aged women, who walked briskly for about 20 km per week for more than a year, achieved a 27% increase in HDL-cholesterol (Després & Lamarche, 1994).

It has been reported that, for every 1kg/m^2 increase in BMI, there is a corresponding 5% decrease in HDL-cholesterol, leading to an elevated CVD risk (Wilson, 1990). On the contrary, weight loss as a result of exercise or diet may lead to an improvement in HDL-cholesterol (Wood *et al.*, 1988). The BMI of the present study group increased from 27.58 kg/m² to 28.08 kg/m², which, when taken into account with the statements above, may explain why their HDL-

cholesterol did not improve and, consequently, had a negative effect on their CVD risk equation.

5.2.4 LDL/HDL-cholesterol ratio

In the present study, the LDL/HDL-C ratio was significantly reduced by 0.44 units from 3.12 to 2.68, at the end of the four-year period (a 14.03% improvement). [This reduction adjusted their risk profile from borderline high risk (1) to the normal risk category (0)]. Studies by Fleck and Kraemer (1988), Després *et al.* (1990) and Shaw (2004) also demonstrated that aerobic training successfully reduced LDL/HDL-cholesterol ratios. The latter study demonstrated that 16 weeks of exercise led to average reductions of between 15.7% and 43% in LDL/HDL-cholesterol ratio values for three different modes of exercise. These studies support the findings of the present study that employees who actively participated in the corporate wellness programme were likely to reduce their LDL/HDL-cholesterol ratio and, thus, decrease their risk of CAD.

5.2.5 TC/HDL-cholesterol ratio

The results of the present study corroborate previous evidence that individuals who participate in physical activities or corporate wellness programmes have a lower TC/HDL-cholesterol ratio, and thus a reduced CVD than their inactive counterparts (Coopoo *et al.*, 1995; Shaw, 2004). The TC/HDL-C ratio of the participants in this study significantly improved by 10.13% at the end of the four-year study period. This reduction did not, however, reduce the groups' risk for CVD from the borderline high-risk category (1) to the normal risk category (0). This finding may in part be attributed to the fact that HDL-cholesterol values decreased by 5.69% over the four years. Shaw (2004) also reported significant decreases ranging from 9 to 36% in the TC/HDL-C ratio following exercise training.

Although Dreyer and Strydom (1992) found that employees who were considered to be physically active, (exceeding 1500 kcal/day) had a significantly lower TC/HDL-cholesterol ratio (5.1) than their more sedentary counterparts (6.5), a value of 5.1 is still considered “high risk”, indicating that moderate exercise alone may not be effective on its own to decrease the TC/HDL-C ratio, once elevated.

5.2.6 Triglycerides

Judging from the current study and literature reviewed, it would seem that participation in physical activity and a corporate wellness programme has a lowering effect on the triglyceride levels in the blood stream. Although having desirable triglyceride levels of 1.45 mmol/l at the onset of the four-year study period, the triglyceride levels of participants in the present study improved further by 8.82% (0,12 mmol/l). This reduction was however not significant. Similar results were obtained by Dreyer and Strydom (1994), who reported that employees who were considered highly physically active (expending in excess of 1500 kcal/day) had lower triglyceride levels (1.5 mmol/l) when compared to the less active employees (1.8 mmol/l) using less than 150 kcal/day. The study by Franklin *et al.* (2002) on 117 patients whom have participated in a physical training programme for eight weeks, consisting of circuit and endurance exercises, also showed a significant 0.82 mol/l reduction in triglyceride levels.

5.3 BLOOD GLUCOSE

The benefits of exercise in reducing fasting blood glucose levels are well recognised (Strydom *et al.*, 1985; Dreyer & Strydom, 1992; Shaw 2004). The present study confirmed this by showing a significant decrease of 7.43% in the fasting blood glucose levels over the four-year study period, even though the values were already at a desirable level (5.01 mol/l) at its commencement in 2002.

Dreyer and Strydom (1992) reported significant decreases in fasting blood glucose levels following a regular exercise programme. Shaw (2004) reported that a 16-week combination fitness programme, a dynamic resistance programme and an aerobic training programme showed a decrease of between 9.7% and 14.2% in fasting blood glucose levels of participants.

5.4 BLOOD PRESSURE

5.4.1 Resting systolic blood pressure

Strydom *et al.* (1985) indicated that physical exercise can potentially be beneficial in reducing resting SBP levels, although other covariables (age, body mass, fat percentage) may influence the effectiveness of physical activity as an intervention method.

In the present study, SBP decreased significantly by 4.85% at the end of the four-year period, but this was not enough to move the group from the pre-hypertension category (120-139 mm Hg) to the normal category. Although Strydom *et al.* (1985) showed that the resting SBP of a group of hypertensive executives who participated in a 24-week training programme was reduced from 163 mm Hg to 144 mm Hg, and that the SBP for the entire group, constituting of hypertensive and non-hypertensives, decreased from 144 mm Hg to 137 mm Hg), studies by Dreyer and Strydom (1995) failed to reported a statistically-significant relationship between the resting systolic pressure for a highly-active group of individuals (> 1500 kcal/day) having a mean pressure of 131 mm Hg and a less-active group having a mean pressure of 132mm Hg, but who expended less than 150 kcal/day. It is noteworthy that changes in covariables like age, body mass and fat percentage may also influence SBP values (Dreyer & Strydom, 1994).

5.4.2 Resting diastolic blood pressure

Regular exercise and a corporate wellness programme may lower DBP (Norris *et al.*, 1990). This was also the finding of the present study in which the DBP was significantly reduced from an already-favourable 77.82 mm Hg to 75 mm Hg at the end of the study, resulting in a 3.63% improvement.

These findings are supported by the findings of Strydom *et al.* (1985), who reported on the positive effect that a 24-week training programme had on a group of hypertensive executives who managed to improve their DBP significantly from 109 mm Hg to 101 mm Hg. Norris *et al.* (1990) also found that the resting DBP of a study group was reduced after running between 20 and 30 minutes per day for 10 consecutive weeks. The known response to exercise is that blood pressure is reduced, and this appears to be mediated by the decrease in the stroke volume, rather than by peripheral vasodilatation (ACSM, 1997). Long-term exercise attenuates the sympathetic response to stress and lowers serum concentrations of catecholamines (Filipovsky *et al.*, 1991).

Contrary to the findings mentioned above, Dreyer and Strydom (1994) discovered no significant difference between the changes in the mean DBP of highly-active employees (> 1500 kcal/week) and less-active employees using no more than 150 kcal/week through physical activities. Both these groups had a mean resting DBP of 89 mm Hg and covariables like age, body mass and fat percentage could have had an influence. This is supported by another recent meta-analysis using 54 randomised clinical trails that concluded that aerobic exercise training would only elicit average DBP by 3mm Hg to 4 mm Hg (Whelton & Appel, 2002).

5.5 BODY COMPOSITION

5.5.1 Body mass index

One of the three CAD risk variables that did not improve as a result of the corporate wellness programme employed in this study, was the mean BMI (BMI) of the 76 participating employees. During the four-year period (2002-2005) BMI increased by 0.5 kg/m², from 27.58 kg/m² to 28.08 kg/m², and the group remained in the overweight risk category. The same phenomenon was reported by Strydom and co-workers (1985), who discovered that a 24-week training programme, involving a group of executives from the motor industry, had no effect on the participants' BMI, although a non-significant decrease in the sum of their skin folds was evident. Similarly, Shaw (2004) found that the BMI of individuals who were actively involved in dynamic resistance training for 16 consecutive weeks increased by 2.4%, but that individuals who actively participated in a combination and aerobic programme managed to reduce their BMI by between 0.1% and 3.2%.

Ample evidence exists that BMI should not be considered in CVD risk calculations (Sjöström, 1993; Robbins *et al.*, 2005). According to Sjöström (1993), the BMI represents an undefined mixture of risk and body fat indices, because body weight is positively associated with CAD risk, whereas height is negatively associated therewith. A combination of these two variables should, therefore, be avoided when calculating CVD risk. Robbins *et al.* (2005) is of the opinion that one important disadvantage of using BMI for this purpose is that it remains a measure of body mass and height, not fatness (i.e. does not distinguish between body fat and muscle mass). It is therefore not appropriate, for example, for an athlete or body builder with a lot of muscle mass. A decrease in BMI may also not necessarily reflect a positive change. For example, in elderly people who apparently reflect a healthy body mass, a loss of muscle mass may have resulted from the loss of nutritional reserves (Robbins *et al.*, 2005).

It is, thus, clear from the results of the present study, as well as from previously-documented research, that employees who embarked on an exercise programme, especially those that involve resistance training, would not necessarily have a lower mean BMI than individuals who were less active, and the question arises whether BMI should be regarded as a CVD risk indicator or not.

5.5.2 Waist circumference

At the end of the four-year study period, waist circumference increased significantly by 2.77% (2.68 cm), but fortunately not enough to move the employees from the initial average-risk category to the above-average category. Android obesity, which is characterised by more fat on the trunk, are at an increased risk of hypertension, type 2 diabetes, coronary artery disease and premature death when compared to individuals who demonstrates gynoid obesity (Sharkey, 1997; ACSM, 1997; Bray & Gray, 1988; Marieb, 2004; Robbins *et al.*, 2005).

In sharp contrast, Strydom *et al.* (1985) discovered a decrease of 3 cm in the waist circumference of the executives who participated in a 42-week training programme. Similarly, Okura *et al.* (2003) found that Japanese women who followed a healthy eating plan and participated in aerobic activities that consisted of low-impact walking seven days a week for 30 minutes, and high-impact step aerobics three days per week for 45 minutes, significantly reduced their waist circumference. A hypertriglyceridemic waist could serve to identify men with hyper insulinemia, elevated apolipoprotein B, and small dense LDL-cholesterol particles (Lemieux *et al.*, 2000).

Therefore, the fact that participants in the present study failed to reduce their waist circumference might have been because the total caloric cost of their physical exercise might have fallen short of the weekly threshold of 1000 to 1500

exercise calories, as recommended by the American College of Sports Medicine to facilitate weight loss (Franklin *et al.*, 2002). The employees might have to adjust the frequency and duration of their exercise to ensure a higher exercise volume, and ensure that their diet has a positive calorie intake/usage ratio. This was, however, not formally monitored during this investigation.

5.5.3 Waist-to-hip ratio

The WHR can be useful in determining CAD risk, since the ACSM (1997) and Marieb (2004) point out that abdominal body fat is associated with an increased risk for hyper-lipidemia and other lipid abnormalities. This ratio can help distinguish between patterns of fat distribution in the upper- and lower body and is also strongly associated with visceral fat and appears to be an acceptable index of intra-abdominal fat (Seidell *et al.*, 1987).

The employees in the present study significantly reduced their WHR from 1.00 to 0.95 over the four-year period, a reduction of 5%. This moved them from the high-risk to the normal-risk category. These results are supportive of the findings from a 16-week study conducted by Shaw (2004), who found that a fitness programme combining dynamic resistance and aerobic activity resulted in a 0.8% decrease in mean waist to-hip ratio, while an exclusively aerobic programme of 45 min resulted in a 0.9% decrease.

On the contrary, Shaw (2004) found that a 16-week dynamic resistance training programme did not have a decreasing effect on the WHR. It is of interest that the majority of longitudinal studies that have employed dynamic resistance training to examine its effect on WHR, have confirmed Shaw's (2004) finding that a consistent non-existent relationship exists between dynamic resistance training and mean WHR.

The result is, however, surprising if the increase in BMI and waist circumference of the present study is taken into consideration, and poses the question whether

the body composition of the employees possessed similar characteristics of the original validation population, and whether the girth measurements were precise.

5.6 TOTAL CAD RISK

When examining the results of the CAD risk in the present study, it would seem that the 76 employees who participated in the wellness programme implemented at Kumba Resources Limited managed to reduce their CAD risk by 25.57% (8.08 to 6.01) at the end of the four-year period from 2002 to 2005. These findings clearly corroborate existing literature, which states that exercise impacts favourably on hypertension, personality behaviour patterns, cigarette smoking, obesity, diabetes mellitus and psychological stress (Byrne, 1991; Wallace *et al.*, 1997; Prabhakaran *et al.*, 1999).

Moreover, it is well known that scientific and individual training programmes for employees generally result in an improvement in physiological (Edington, 1986; Pauly, 1981) and psychological parameters (Johnson, 1983; Pauly, 1981), as well as a positive change in healthy lifestyle habits of employees (Gordon, 1997; Berlin, 1983). With these changes, a company may eventually finish up with healthier employees and a more efficient workforce, which may lead to greater productivity and a far greater financial gain than the initial investment (Brown *et al.*, 1980).

CHAPTER SIX: SUMMARY AND CONCLUSION

The development of CAD goes hand in hand with physiological, psychological, environmental and other risk factors. Regular physical activity may significantly influence some of these risk factors, and physical inactivity is currently considered as an independent CAD risk factor (Nieman, 1998; Stoney & Hughes, 1999). It is clear from the literature that companies have to take active steps in caring for their most important assets: their employees. In South Africa, wellness programmes have been implemented since 1982, striving to improve the health status of employees in middle- to top management positions. No information is, however, available on the long-term benefits of these corporate wellness programmes in SA.

It can be no coincidence then that, since the mid-1970s, companies in the USA have started to show an increased interest in industrial fitness programmes (Friedman, 1986). The primary objective of such physical conditioning programmes were mainly to combat certain financial expenses, such as increasing healthcare costs, lower productivity, increase in employee resignations and short-term absenteeism (Edington, 1986; Eakin *et al.*, 1988; Cox *et al.*, 1988). This financial motivation almost certainly led to the fact that 90% of large companies that were evaluated in the USA were offering at least one health-improving activity to its executives (Messer *et al.*, 2000).

The present author analysed the results of one such a programme at Kumba Resources Limited and found that it had significant benefits on CAD risk (Table 6.1).

Table 6.1: Summary of the CAD risk changes from pre-test (2002) to post-test (2005).

Variable	Pre-test (2002)	Post-test (2005)	% Change	P-Value
TC (mmol/l)	5.51	4.74	-13.98	0.000*
LDL-C (mmol/l)	3.64	2.99	-17.85	0.000*
HDL-C (mmol/l)	1.22	1.17	-4.20	0.018*
LDL/HDL-C	3.12	2.68	-14.03	0.000*
TC/HDL-C	4.70	4.22	-10.13	0.000*
Triglycerides (mmol/l)	1.45	1.33	-8.82	0.563
Blood Glucose (mmol/l)	5.01	4.64	-7.43	0.000*
SBP (mm Hg)	130.64	124.30	-4.85	0.022*
DBP (mm Hg)	77.82	75.00	-3.63	0.020*
BMI (kg/m²)	27.58	28.08	1.81	0.012*
Waist Circumference (cm)	96.85	99.53	2.77	0.000*
WHR	1.00	0.95	-4.66	0.000*
CAD risk score	8.08	6.01	-25.57	0.000*

* : Statistical-significant difference (P<0.05)

From the above results, it is evident that TC, LDL-C, LDL-C/HDL-C ratio, TC/HDL-C ratio, fasting blood glucose, resting SBP, resting DBP and WHR improved significantly during the study period, while TG showed a non-significant improvement.

Four of the twelve risk variables did not show a significant improvement over the four-year study period. The three that actually deteriorated significantly during the study period were HDL-C, BMI and WC. The fourth, triglyceride, did manage to improve, but the improvement failed to be significant.

Body mass index increased by 1.81%, but it has been documented that an increase in BMI might not necessarily reflect a negative phenomenon. One of the disadvantages of using this variable as an indicator for CAD risk is that it remains a measure only of body weight and height, not fatness (i.e. it does not distinguish between body fat and muscle mass). It may therefore not be appropriate for an athlete or body builder with a lot of muscle mass (Robbins *et al.*, 2005).

On the other hand, an increase in BMI could partially have been responsible for the deterioration in HDL-cholesterol values, because according to Wilson (1990), for every one unit increase in BMI, there is a corresponding 5% decrease in HDL-C, leading to an increased CAD risk. At the end of the four-year study period, the HDL-C mean values of the study group deteriorated by 0.05 mmol/l (4.20%). This deterioration, however, was not enough to move them into a higher risk category.

The third variable that did not improve was waist circumference, which increased by 2.77%. This could be attributed to an inadequate diet, or exercise threshold falling short of the weekly 1000 to 1500 kcal/week that the ACSM recommends (ACSM, 1997; Franklin *et al.*, 2000).

Despite being in the desirable-risk category at the onset of the study, the employees managed to improve their triglyceride levels even further. The fact

that the improvement failed to be significant could be attributed to the already-favourable levels at the onset of the study.

During the third quarter of the present study, a restructuring process was implemented at Kumba Resources Limited, and employees were faced with the harsh reality of losing their jobs. This stressful situation proved to have had a negative effect on the employees' wellness, as 11 of the 12 variables deteriorated, or did not improve significantly during this period. Individuals with a propensity towards emotional responsibility during stressful work situations showed an increased tendency towards the development of CAD (Melamed, 1996). This situation might have contributed to a situation where employees neglected healthy eating habits, and spent less time being physically active.

In future, this research could be extended to focus on the following aspects: determining the effects of a similar corporate wellness programme on white- and blue-collar workers; the effect of a restructuring programme on CAD risk factors of employees; and, lastly, the effect of a similar corporate wellness programme, and its effect on the CAD risk variables amongst women, may also produce some interesting and important results.

When considering the 26% reduction in CAD risk of the present study, it would seem that the corporate wellness programme did have a significant effect in the company. The major finding of this investigation thus suggests that a corporate wellness programme has long-term beneficial effects on CAD risk, and that the reduction in CAD risk is mainly attributed to the beneficial effect of regular exercise.

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ANNEXURE A: HEALTH RISK ANALYSIS

The following survey (Sharkey, 1997) will help analyse health risk in the area of CAD. Scores to be tabled to discover CAD risk.

Coronary heart disease (CHD) risk factors

TC, TC/HDL-C ratio				
under 160 <3 +2	160-200 3-4 +1	200-220 4-5 -1	220-240 5-6 -2	over 240 >6 -4
Blood pressure (systolic/diastolic)				
110 60-80 +1	110-130 60-80 0	130-150 80-90 -1	150-170 90-100 -2	170 >100 -4
Smoking				
never +1	quit 0	smoke cigar or pipe or close family member smoke -1	1 pack cigarettes daily -3	2 or more packs daily -5
Heredity				
no family history of CHD +2	1 close relative over 60 with CHD 0	2 close relatives over 60 with CHD -1	1 close relative under 60 with CHD -2	2 or more close relatives under 60 -4

Body weight (or fat)				
5lb below desirable weight (<10% fat-M; <16% fat-F) +2	5lb below to 4lb above desirable weight (10-15% fat-M; 16-22% fat-F) +1	5-20lb overweight (15-20% fat-M; 22-30% fat-F) 0	20-35lb overweight (20-25% fat-M; 30-35% fat-F) -2	35lb overweight (>25% fat-M; >35% fat-F) -3

Sex				
female under 45yr 0	female over 45yr -1	male -1	stocky male -2	bald, stocky male -4
Stress				
phlegmatic, unhurried, generally happy +1	ambitious but generally relaxed 0	sometimes, hard-driving, time-competitive 0	hard-driving, time-conscious, competitive (Type A) -1	Type A with repressed hostility -3

ANNEXURE B: ESTIMATE OF 10-YR RISK FOR MEN AND WOMEN

Estimate of 10-yr Risk for Men (Framingham Point Scores)

Age (yr)	Points
20-34	-9
35-39	-4
40-44	0
45-49	3
50-54	6
55-59	8
60-64	10
65-69	11
70-74	12
75-79	13

Points

Total cholesterol, mmol/l	Age 20-39	Age 40-49	Age 50-59	Age 60-69	Age 70-79
<4.16	0	0	0	0	0
4.16-5.17	4	3	2	1	0
5.2-6.21	7	5	3	1	0
6.24-7.25	9	6	4	2	1
≥7.28	11	8	5	3	1

Points

	Age 20-39	Age 40-49	Age 50-59	Age 60-69	Age 70-79
Nonsmoker	0	0	0	0	0
Smoker	8	5	3	1	1

HDL, mmol/l	Points
≥1.56	-1
1.3-1.53	0
1.04-1.27	1
<1.04	2

Systolic BP, mm Hg	If untreated	If treated
<120	0	0
120-129	0	1
130-139	1	2
140-159	1	2
≥160	2	3

Estimate of 10-yr Risk for Women (Framingham Point Scores)

Age (yr)	Points
20-34	-7
35-39	-3
40-44	0
45-49	3
50-54	6
55-59	8
60-64	10
65-69	12
70-74	14
75-79	16

Points

Total cholesterol, mmol/l	20-39	40-49	50-59	60-69	70-79
<4.16	0	0	0	0	0
4.16-5.17	4	3	2	1	1
5.2-6.21	8	6	4	2	1
6.24-7.25	11	8	5	3	2
≥7.28	13	10	7	4	2

Points

	Age 20-39	Age 40-49	Age 50-59	Age 60-69	Age 70-79
Nonsmoker	0	0	0	0	0
Smoker	9	7	4	2	1

HDL, mmol/l	Points
≥1.56	-1
1.3-1.53	0
1.04-1.27	1
<1.04	2

Systolic BP, mm Hg	If untreated	If treated
<120	0	0
120-129	1	3
130-139	2	4
140-159	3	5
≥160	4	6

Point total	10yr Risk, %
<0	<1
0	1
1	1
2	1
3	1
4	1
5	2
6	2
7	3
8	4
9	5
10	6
11	8
12	10
13	12
14	16
15	20
16	25
≥17	≥30

Point total	10yr Risk, %
<9	<1
9	1
10	1
11	1
12	1
13	2
14	2
15	3
16	4
17	5
18	6
19	8
20	11
21	14
22	17
23	22
24	27
≥25	≥30

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