THESIS TITLE

The impact of multiple behaviour health intervention strategies on coronary heart disease risk, health-related physical fitness, and health-risk behaviours in first year university students.

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I hereby declare that "The impact of multiple behaviour health intervention strategies on coronary heart disease risk, health-related physical fitness, and health-risk behaviours in first year university students" is my own work, that it has not been submitted before for any other degree in any other university, and that the sources I have used have been indicated and acknowledged as complete references.

Lloyd Llewellyn Leach   November 2011

Signed _____

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Acknowledgements

DEDICATION

To family, immediate and extended, young and not-so-young, and friends, you are all

highly valued and most cherished. Continue to stay close …...

Most especially, to my wife Noleen who endured countless hours of stress and anxiety,

but particularly for her critical insights and editing of the thesis, without whose support

this study would not have met the appropriate standard and quality of academic rigour.

Thank you honey…..

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List of Acronyms

ACSM	=	American College of Sports Medicine
CAD	=	coronary artery disease
CAPHER	=	Canadian Association for Physical, Health Education, and Recreation
CDL	=	chronic diseases of lifestyle
CG	=	control group
CHD	=	coronary heart disease
CMH	=	Cochran-Mantel-Haenszel
CPUT	=	Cape Peninsula University of Technology
CSSS	=	Centre for Student Support Services
CVD	=	cardiovascular disease
DG	=	diet group
EG	=	exercise group
EMS	=	Emergency Medical Services
GG	=	grand group
HBI	=	historically black institution
HBU	=	historically black university
HIG	=	health information group
HLQ	=	healthy lifestyle questionnaire
HRB	=	health-risk behaviours

HRPF	=	health-related physical fitness
HSRC	=	Human Sciences Research Council
HWI	=	historically white institution
IPAQ	=	international physical activity questionnaire
ISAK	=	International Society for the Advancement of Kinanthropometry
MG	=	multiple group
MS	=	Microsoft
PA	=	physical activity
PAR-Q	=	physical activity readiness questionnaire
OTC	=	over-the-counter
RF	=	risk factor
SA	=	South Africa
SAMRC	=	South African Medical Research Council
SAS	=	statistical analysis system
SCT	=	Social Cognitive Theory
SPSS	=	Statistical Package for the Social Sciences
SRCQ	=	stages of readiness to change questionnaire
SRES	=	Sport, Recreation and Exercise Science
SSA	=	Sub-Saharan Africa
STDs	=	sexually transmitted diseases
TEM	=	technical error of measurement
TTM	=	Transtheoretical Model
UWC	=	University of the Western Cape

WHO	=	World Health Organization
WP	=	Western Province

List of Scientific Abbreviations

Apo	=	apolipoprotein
BF	=	body fat
BP_{dias}	=	diastolic blood pressure
BP_{sys}	=	systolic blood pressure
BMI	=	body mass index
BP	=	blood pressure
CHD	=	coronary heart disease
Chol	=	cholesterol
CVD	=	cardiovascular disease
HDL	=	high density lipoprotein
HDLC	=	high density lipoprotein cholesterol
HR	=	heart rate
HR_{max}	=	maximal heart rate
IFG	=	impaired fasting glucose
LBM	=	lean body mass
LDL	=	low density lipoprotein
LDLC	=	low density lipoprotein cholesterol
RHR	=	resting heart rate

Trig	=	triglycerides
$\dot{V}O_2max$	=	maximal volume of oxygen consumed per minute
$\dot{V}O_2peak$	=	highest volume of oxygen obtained for a particular exercise stress test
WHR	=	waist-to-hip ratio
TC	=	total cholesterol

Units of Measurement

bpm	=	beats per minute
cm	=	centimetres
$d \cdot wk^{-1}$	=	days per week
kg	=	kilogram
$kg \cdot m^{-2}$	=	kilograms per square metre
m	=	metre
$MET \cdot min \cdot wk^{-1}$	=	Metabolic equivalent minutes per week
min	=	minutes
$mg \cdot dL^{-1}$	=	milligrams per decilitre
mm	=	millimetres
$mmol \cdot L^{-1}$	=	millimoles per litre
mm Hg	=	millimetres of mercury
$ml \cdot kg^{-1} \cdot min^{-1}$	=	millilitres per kilogram per minute
rpm	=	repetitions per minute
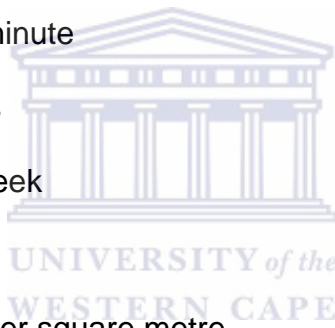
List of Statistical Units

NS   =   non-significant

SD   =   standard deviation

X   =   mean

%   =   percent

α =   alpha

Miscellaneous Notation

cpd   =   cigarettes per day

CD   =   computer diskette

email   =   electronic mail

Abstract

Background: There is compelling body of evidence that coronary heart disease (CHD) risk factors are present in people of all ages. The extent to which the problem exists in university students in South Africa (SA) has not been confirmed in the literature. Furthermore, the effects of physical activity, physical fitness, diet and health behaviours on CHD risk factors has not been studied extensively in SA and needs further investigation.

Aim: The aim of the study was to assess the impact of multiple behaviour health intervention strategies on CHD risk, health-related physical fitness(HRPF) and health-risk behaviours (HRB) in first year students at the University of the Western Cape (UWC). It was hypothesized that exposure to various health behavioural interventions would reduce CHD risk factors in subjects at moderate risk, and improve health-related physical fitness, as well as health-risk behaviours.

Methods and Study Design: An experimental study design was used wherein subjects at moderate risk for CHD were identified and exposed to multiple health behavioural interventions for 16 weeks in order to determine the impact of the various interventions on CHD risk, health-related physical fitness and health-risk behaviours.

Population and Sample: The target population consisted of first year students at UWC aged 18 – 44 years who were screened and a sample of 173 subjects were identified as being at moderate risk for CHD. Next, the subjects were randomly assigned to a control and four treatment groups, namely, health information, diet, exercise, and a multiple group that included all three treatments. The intervention, based upon Prochaska's Transtheoretical Model of behaviour change, continued for a period of 16 weeks and, thereafter, the subjects were retested.

Data Collection Process: Subject information was obtained using self-reported questionnaires, namely, the physical activity readiness questionnaire (PAR-Q), the stages of readiness to change questionnaire (SRCQ), the international physical activity questionnaire (IPAQ), and the healthy lifestyle questionnaire (HLQ), together with physical and hematological (blood) measurements. The measurements taken before and after the intervention programme were the following:

- Coronary heart disease risk factors, namely: family history, cigarette smoking, hypertension, obesity, dyslipidemia, impaired fasting glucose and a sedentary lifestyle;

- Health-related physical fitness, namely: body composition, cardiovascular fitness, muscular strength, muscular endurance, and flexibility; and

- Health-risk behaviours, namely: physical activity, nutrition, managing stress, avoiding destructive habits, practising safe sex, adopting safety habits, knowing first

aid, personal health habits, using medical advice, being an informed consumer, protecting the environment and mental well-being.

Types of interventions: A control group was used in which subjects did not receive any treatment. The health behavioural interventions were arranged into four groups of subjects that received either the health information, diet, exercise or a combination of all three individual treatments.

Statistical analyses of data: In the analyses of the data, the procedure followed was that where the outcome variable was approximately normally distributed, the groups were compared using a two-sample t-test. For outcomes with a highly non-normal distribution or ordinal level data, the nonparametric Wilcoxon Rank Sum test was used for group comparisons. To account for baseline differences, repeated measures analysis of variance was used. In the case where nonparametric methods were appropriate, analysis was done using Cochran-Mantel-Haenszel (CMH) methodology stratifying on the baseline values. For the case of nominal level outcomes, groups were compared by Chi-square tests for homogeneity of proportions. When baseline values needed to be incorporated into the analysis, this was done using CMH methodology.

Main Outcome Measures: The main outcome measures tested in the study related to the three areas of investigation, namely:

- Modifiable CHD risk factors: systolic and diastolic blood pressure, cigarette smoking, total cholesterol (TC) concentration, high-density lipoprotein (HDL) cholesterol concentration, low-density lipoprotein (LDL) cholesterol concentration, triglycerides, fasting glucose, body mass index, waist circumference, waist-hip ratio and physical inactivity;

- Health-related physical fitness: body mass, percent body fat, absolute body fat, percent lean body mass, absolute lean body mass, the multi-stage shuttle run, handgrip strength, repeated sit-ups in a minute, and the sit-and-reach test; and

- Health-risk behaviours: physical activity, nutrition, managing stress, avoiding destructive habits, practising safe sex, adopting safety habits, knowing first aid, personal health habits, using medical advice, being an informed consumer, protecting the environment and mental well-being.

Results: The results showed significant decreases for body mass, waist and hip circumferences, resting heart rate, systolic blood pressure, cigarette smoking and a sedentary lifestyle ($p < .05$) primarily in the multiple group. No significant differences were recorded for blood biochemistry, however, favourable trends were observed in the lipoprotein ratios.

For health-related physical fitness, only the multiple group showed significant ($p < .005$) improvements in predicted maximal oxygen consumption ($\dot{V}O_2max$), body composition, muscular strength and muscular endurance. The exercise group also recorded

significant differences in muscular endurance. In all groups, including the controls, no significant differences were found for stature, waist-hip ratio, and flexibility at pre- and post-test.

Overall, the participants reflected positive health behaviours, especially for managing stress, avoiding destructive habits, practising safe sex, adopting safety habits, personal health habits and mental well-being at pre- and post-test. The intervention programme had a corrective influence on providing the participants with a more realistic perception of their level of physical activity and nutritional habits. The participants scored poorly on being informed consumers and for recycling waste both at pre- and post-test.

A substantial net reduction in CHD risk factors as well as in cumulative risk was achieved with treatment that impacted positively on the re-stratification of participants at moderate risk. In terms of treatment efficacy, the dietary intervention appeared to be the least effective (10.91%), with health information and exercise sharing similar levels of efficacy (32.81% and 33.93%, respectively) and, the combined treatment in the multiple group stood out as the most effective treatment (50.00%), and supported the hypothesis of the study.

Conclusions: The net and cumulative decline in CHD risk factors was substantial with treatment and was directly related to the number of treatments administered. The evidence suggests that such multiple health behaviour interventions when implemented through a university-based setting have substantial benefits on reducing CHD risk and may be of considerable public health benefit.

Key messages

- Despite being a relatively educated population, a substantial number of first year university students are at considerable heart disease risk.
- Physical inactivity constitutes one of the main CHD risk factors amongst first year students and, together with smoking, place many of them at moderate CHD risk.
- The effectiveness of health behavioural strategies designed to modify lifestyle and prevent coronary heart disease is supported by this study.

Keywords: coronary artery disease, cardiovascular disease, coronary heart disease, modifiable risk factors, physical fitness, health-risk behaviours, university students, intervention programme, multiple behaviour health interventions, lifestyle behaviour modification

Chapter One: Statement of the Problem

1.1 Introduction

An epidemic in cardiovascular disease is rapidly evolving globally (WHO, 2002). The burden of disease is steadily shifting towards chronic diseases of lifestyle and, in particular, cardiovascular disease (CVD) (Murray and Lopez 1997a; Murray and Lopez 1997b; Pearson et al., 1998; Yusuf et al., 2001; Yusuf et al., 2001b; WHO, 2002; Rothstein, 2003; Mar et al., 2007; Lopez et al., 2006). CVD causes twice as many deaths in developing than in developed countries (Pearson, 1999; Akinboboye et al., 2003; Gaziano, 2005; Lopez et al., 2006). The main causes of CVD can be attributed to conventional CHD risk factors, such as physical inactivity, cigarette smoking, hypertension and obesity (Thomas et al., 2003). Kruger et al. (2005) found that, in sub-Saharan Africa (SSA), high blood pressure, high cholesterol, tobacco and alcohol use and low vegetable and fruit consumption were the main risk factors for CVD. This appeared to be the case even among university students (Steptoe et al., 2002; Tamim et al., 2003; Tamim et al., 2004; Irazusta et al., 2007).

Coetsee (2003) reported that, in SA, many clients who frequent fitness centres are relatively active and health conscious. However, their health and physical fitness profiles still remained alarmingly poor. This indicated the need for professional service providers, such as biokineticists and personal fitness trainers, to address these health concerns in an attempt to reduce the escalating burden of disease.

1.2   Statement of the Problem

CHD risk factors are early indicators of future heart disease (McGill et al., 2000) and the presence of multiple these risk factors in people is considered to be a reflection of poor community health and wellbeing (Pearson, 1999). Since most of the risk factors for heart disease are modifiable, it would be expedient, if not a health imperative, to identify individuals who are at risk at the earliest possible opportunity, so that appropriate preventive measures can be implemented to avert the risk. Furthermore, strategic efforts that are community-wide, economically cost-effective, and educationally sound that are undertaken by public health agencies and supported by the state certainly have the potential to impact favourably on public health. The potential benefits can be direct, as seen in the reduced morbidity and mortality statistics, or indirect, through improved quality of life and reduced medical costs for the public at large.
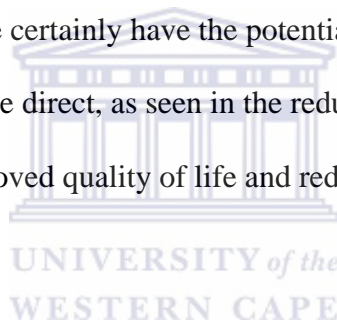
There is a compelling body of evidence which shows that CHD risk factors are present in young people (Coetsee, 2003; Gaziano, 2005; Schmidt et al., 1998; Remsberg et al., 2002; Thomas et al., 2003). However, the extent to which this problem exists in university students in South Africa has not been adequately investigated (Kazi and Coopoo, 2010; Porter et al., 2009). A contributing factor is the fact that various studies that have examined the effects of different behavioural interventions, such as physical activity, physical fitness and diet on CHD risk factors are quite varied in terms of the target population, inconsistent in the methodology, and ambivalent in its findings. This makes it difficult to compare, arduous to repeat, and challenging to understand. Therefore, the purpose of the present study is to address these shortcomings by
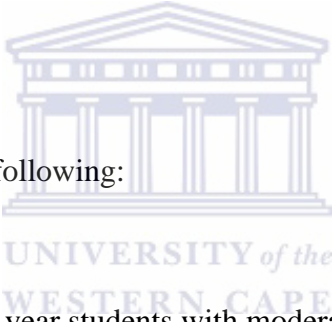
investigating the impact of a multiple health behavioural intervention strategy on CHD risk factors, health-related physical fitness (HRPF) and health-risk behaviours (HRB) in first year university students.

1.3 Aim of the Study

This study aims to assess the impact of a multiple health intervention strategy on CHD risk factors, HRPF, and HRB in students in their first year of study at a university.

1.4 Objectives of the Study

The objectives of the study are the following:

- To identify a sample of first year students with moderate CHD risk;

- To measure the HRPF levels;

- To measure the HRB;

- To assess the impact of multiple health interventions on CHD risk factors;

- To assess the impact of multiple health interventions on HRPF;

- To assess the impact of multiple health interventions on HRB; and

- To assess whether the impact of multiple health interventions are dose-response related.

1.5 Research Questions

In an attempt to meet the objectives of the study, the following research questions were identified:

- What was the frequency of CHD risk factors in subjects with moderate CHD risk?
- What was the HRPF of subjects at moderate CHD risk?
- What were the HRB in subjects at moderate CHD risk?
- What was the effect of multiple health behavioural interventions on CHD risk, HRPF and HRB?
- Is there a dose-response relationship between treatment and CHD risk factors, HRPF, and HRBs?

1.6 Study Hypotheses

The hypotheses of the study are that the effect of multiple health behavioural interventions will:

- Reduce CHD risk factors in subjects at moderate risk;
- Improve HRPF in subjects with moderate CHD risk;
- Reduce HRB in subjects with moderate CHD risk; and
- Reduce CHD risk and improve HRPF and HRB in a dose-response manner.

1.7   Significance of the Study

This study has relevance for two main reasons. Firstly, the prevalence of CHD is a global public health problem and, secondly, scientific studies in developing countries are generally scarce and often questionable in terms of research methodology. The scarcity of research data on CHD risk and physical fitness in the student population of South Africa and the need to identify guidelines for effective lifestyle modification for public health benefit, are of particular concern and need urgent attention. The more marginalized communities in SA, which contribute largely to the student population at UWC, are given low public health priority and are denied access to scarce skills. This further strengthens the demand for research that can help address these social disparities.

Students in higher education form an important population in public and private life of any society, therefore, having an insight into the public health problems that burden this population has public health and economic implications.

This study attempts to address the need for effective and efficient intervention strategies for addressing CHD risk, especially given the extraordinary challenges placed upon the healthcare system in SA. Therefore, using potentially efficacious and cost-effective methods would be opportune and well-received.

The health and physical fitness status of the youth in SA has not been studied extensively, which makes studies such as this one relevant.

Finally, because physical inactivity and poor physical fitness amongst the youth have failed to be adequately addressed by most government ministries in SA, including the departments of Health, Sport and Education, the findings from this study may prove helpful in providing information for the development of policy and help shape the future health of the youth.

1.8   Delimitations of the Study

The delimitations of the study were divided into two categories, namely, inclusion and exclusion criteria. The former related to factors that determined whether subjects would be included in the study, and the latter to factors that excluded subjects from participating in the study.

1.8.1 Inclusion Criteria
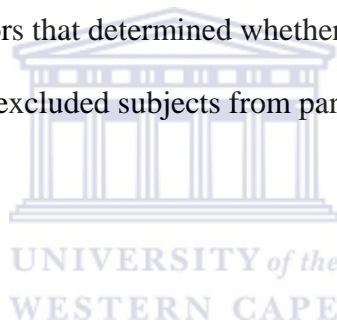
The following inclusion criteria were applied in the study, namely:

- Non-repeating, full-time first year students registered at UWC;
- Students between the ages of 18 and 44 years; and
- Students who did not plan on travelling or leaving the institution for more than two weeks during the 16 week intervention period of the study.

1.8.2 Exclusion Criteria

The following exclusion criteria were used in the study, namely:

- Subjects classified as high-risk for CHD, that is, subjects diagnosed with cardiovascular, pulmonary, metabolic, and/or orthopaedic disease;
- Subjects with chronic musculo-skeletal injuries, illnesses, or ailments, such as recurring ankle sprains, asthma, and contact dermatitis;
- Subjects diagnosed with psychological disorders, such as anxiety, depression, and bulimia;
- Subjects who were athletes or sportspersons;
- Subjects participating in weight loss programmes, such as Weigh-Less;
- Subjects taking prescribed medication;
- Senior and postgraduate students;
- Students registered for non-degree purposes for one semester only and
- Students who failed their first year.

1.9 Limitations of the Study

There were several limitations related to this study, namely:

- Leisure time physical activity, which included a wide range of sports and recreational activities that vary in their energy demands, was not measured directly in the study but

was based on self-reported information provided by the subjects. Self-reported

assessments constitute only a portion of an individual's total daily energy expenditure,

and are imprecise and prone to error. This may have impacted the statistical significance

of some of the findings;

- A relatively small sample of subjects from one university only were selected, therefore,
  caution is advised when generalizing the results;

- Sampling was non-randomized and sought to identify students with multiple risk factors,
  and may not be representative of the general university population demographics or
  health and physical fitness characteristics; and

- Subjects were not isolated during the intervention period and the natural interaction of
  subjects from the different treatment groups may have contaminated the effects of the
  treatments and influenced the overall outcomes of the study.

1.10   Definitions of Terms

Cardiovascular disease: CVD refers to any disease of the heart and blood vessels, with the most

common being heart attacks, heart failure, and strokes (Heart and Stroke Foundation of SA,

2007).

CHD: CHD is synonymous with coronary artery disease (CAD), and is a component of CVD that pertains to pathology in the blood vessels of the heart. It is the progressive build-up of fatty deposits in the lining of the arteries supplying blood to the heart. Due to this process, called atherosclerosis, the narrowed arteries cannot maintain a healthy flow of blood to the heart. As CHD worsens, heart function is adversely affected, resulting in damage to myocardial tissue due to a shortage of oxygen and nutrients. CHD is the most preventable form of CVD (McArdle et al., 2001, p. 893).

Chronic diseases of lifestyle: CDLs are a group of diseases, such as obesity, metabolic syndrome, diabetes, hypertension and hypercholesterolemia that develop gradually over time due to regular exposure to risk factors such as unhealthy diets, smoking, lack of physical activity, and stress. These risks result in various long-term disease processes, culminating in high morbidity and mortality rates attributable to strokes, heart attacks, tobacco- and nutrition-induced cancers, chronic bronchitis, emphysema, renal failure, and many others (Steyn, 2006).

Hypercholesterolemia: Hypercholesterolemia is a high level of cholesterol (>5.2 mM/L) in the blood and is a major risk factor for CHD. Cholesterol is transported in the blood in different forms depending upon the types of lipoproteins. The most important lipoproteins are low-density lipoprotein (LDL), known as the "bad' cholesterol", and the high-density lipoprotein (HDL), which is known as the 'good' cholesterol". A high level of LDL cholesterol increases the risk of heart disease. Therefore, the lower the LDL cholesterol concentration, the lower is the risk of CHD, and vice versa. Alternatively, high HDL levels indicate good health, and give some protection against CHD (Steyn, 2007).

Health-risk behaviour: HRB refers to the conditioned or repetitive action of an individual that places his/her health at increased risk be engaging in negative activities, such as drug or substance abuse, promiscuous sexual activity, violence, a sedentary lifestyle, etc (Steyn, 2006).

Maximal oxygen consumption: Maximal oxygen consumption ($\dot{V}O_2max$) is commonly referred to as aerobic capacity, and is measured during an exercise stress test in which the body's cardiovascular, respiratory and muscular systems are activated to near maximal capacity in an attempt to record the maximal amount of oxygen that can be consumed in a period of one minute. The criterion used to indicate the attainment of a maximal value is the leveling-off or plateau-effect in oxygen consumption with increasing exercise intensity over multiple exercise stress tests (McArdle et al., 2001, p. 233).

Peak oxygen consumption: The term peak oxygen consumption or $\dot{V}O_2peak$ refers to the highest oxygen consumption measured in an isolated graded exercise test, and is used when leveling-off does not occur with increasing exercise intensity or the exercise stress test appears to be limited by local muscular factors rather than central circulatory dynamics (McArdle et al., 2001, p. 233).

Physical activity: Physical activity is a complex, multifactorial behaviour that is commonly defined as any bodily or physical movement that is produced as a result of repetitive muscular (skeletal) contraction of brief or prolonged duration and generally contributes towards overall physical well-being (Caspersen et al., 1985). Similarly, exercise, as a component of physical

activity, is synonymous with physical training or conditioning that is planned, structured and regularly performed in order to achieve the objective of maintaining or improving personal physical fitness (Caspersen et al., 1985).

Physical fitness: Physical fitness is a complex and multidimensional concept that is also elusive to categorize and is in constant flux, making it difficult for most individuals to attain and maintain, especially in later years. However, it is commonly referred to as the manifestation of exercise or physical training that results in the development of a number of physical and/or physiological attributes that may be either health- or skill-related (Caspersen et al., 1985).

HRPF: HRPF are those components of physical fitness associated with maintaining or improving personal health and minimizing the risk of developing hypokinetic (inactivity) diseases, also called chronic disease of lifestyle (CDL), such as hypertension, diabetes, obesity and cardiovascular disease (ACSM, 2006a, p. 3). Physical activity is, therefore, positively correlated with physical fitness to the extent that increased amounts of physical activity, specifically progressively incremental exercise, translate directly into improved levels of physical fitness, and vice versa (Caspersen et al., 1985). However, once attained, physical fitness is not a constant, inert characteristic, but a continually changing state. It therefore requires persistent effort and regular training, especially with advancing age, in order to be maintained optimally.

Whereas most sportspersons require both health-related and skill-related physical fitness for optimal performance, the health enthusiast only requires health-related fitness. The singular

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focus is on optimizing health and functional daily living, while simultaneously minimizing health risk, with little or no regard for skill level or performance outcome.

Risk factor: Risk factors are those characteristics, variables, or hazards that, if present for a given individual, make it more likely that this individual will develop a disorder. In order to qualify as a risk, the factor must antedate the onset of the disorder. Also, risk factors are not static. They can change in relation to age or a new stressor in one's life, and can reside within the individual, family or community. (ACSM, 2006b, p. 95).

Skill-related physical fitness: The concept of skill-related physical fitness (ACSM, 2006a, p. 3) was derived to clarify the dualistic meaning of physical fitness. As expected, skill-related fitness refers to the fitness components required for skillful sports performance, such as agility, speed, balance, coordination, power, and reaction time, amongst others.
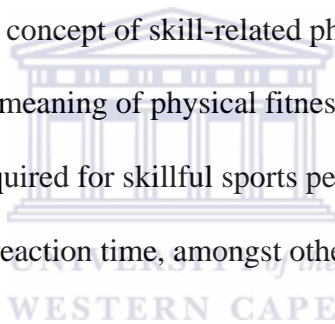
Chapter Two: Review of the Related Literature

2.1   Introduction

The epidemic of CHD is unprecedented, with many people at risk of premature disability and death (Mark et al., 2007). CHD has a relatively prolonged latent period before the signs and symptoms become evident and it is diagnosed clinically (Barker, 1995). Ironically, it is a preventable condition and a wealth of information is available to empower people to act against it (Waldron, et al., 2011). Risk factors, such as physical inactivity, regular cigarette smoking, high blood pressure and hypercholesterolemia are the main causes of CHD (Navas-Nacher, et al., 2001). Hypercholesterolemia is reported to cause more than 4 million premature deaths a year, smoking about 5 million and hypertension almost 7 million (WHO, 2002, p. 8).

Some of the stark conditions endemic to developing countries are, on the one hand, rapidly declining levels of physical activity (Andrews et al., 1985) and poor levels of physical fitness (Gaziano, 2005; Leeder et al., 2004) contrasted, on the other hand, by rapidly rising pathology in HRB and widespread CHD (Pearson, 1999; Reddy, et al., 2003). The poor and indigent are especially vulnerable (Lopez et al., 2006). The problem is compounded the fact that CHD remains insidious for many years due to a prolonged gestation period (McGill et al., 2000). Consequently, the full extent of the problem only becomes evident in later years, at a time when treatment becomes challenging and expensive (Ashen 2010; Pasternak et al., 2003).

The public healthcare system in most developing countries is inequitable, with the vast majority denied access to primary healthcare (Eriksson et al., 2007). Some of the reasons cited as rationale for maintaining the status quo include the fallacy that CDLs, such as CHD, are primarily diseases of affluence, and affect mainly the elderly (Pearson, 1999). Moreover, HRB such as smoking, unhealthy diets and a lack of exercise are independently acquired and do not warrant action on the part of the state or public health agencies. However, the reality of the matter shows quite the opposite. The developing nations are the most vulnerable to CHD, and the poor are often the victims within those countries (Levenson et al., 2002; Yusuf et al., 2001). Future predictions report CHD to manifest at a younger age, causing higher age-specific morbidity and mortality rates (Murray and Lopez, 1997a). CHD is also presumed to become the leading cause of death and disability worldwide by 2020 (Pearson, 1999). Clearly, as the CHD epidemic continues to proliferate, the poor are increasingly becoming the unfortunate victims in all nations (Yusuf et al, 2001).
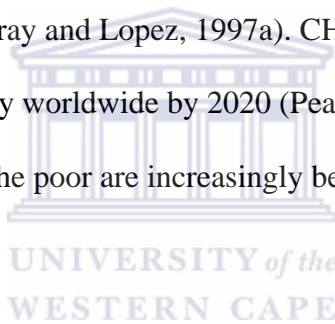
The growing incidence of CVD in developing countries is thought to be primarily the result of CHD (Lopez et al., 2006). Recent statistics reported on low- and middle-income countries show that about 80% of the global deaths, and almost 87% of the disabilities are attributed to CVD (Bloom et al., 2011; Leeder et al., 2004). This is as a result of rapid westernization and growing urbanization. These new lifestyles are characterised, on the one hand, by an excessive consumption of high-energy foods and, on the other hand, by a sharp decline in energy expenditure due to improved mechanization (WHO, 2002). The demise of the traditional family unit and the loss of social support structures have compounded the problem further and have led to increasing rates of obesity, hypertension, hyperglycemia and hypercholesterolemia (Vorster,

2002). These chronic diseases of lifestyle are all associated with higher rates of CHD and strokes (Erikson et al., 2007; Iso, 2011; Sarwar et al., 2010; Steyn et al., 1991, 2004; Steyn and Fourie, 2007).

In developing countries, the combination of limited research on CHD and minimal educational programmes targeting risk reduction and prevention contribute to the lack of a formidable response against the CHD epidemic (Mark et al., 2007). Of particular concern is the lack of response at the tertiary level, especially because university students constitute the future workforce of the economy and the aspirant leadership of the nation (Tamragouri et al., 1986). At most universities, students are generally unaware of the public health challenges that confront society. Many do not demonstrate an integration of that knowledge into a quality of life that resonates well with positive health practices (Frost, 1995). The knowledge required for a comprehensive public health response against CHD is, for the most part, readily available. Unfortunately, the effective application of that knowledge for the benefit of the broader community is sorely lacking.

The negative impact of CVD, like many risk factors, is largely reversible, provided that it is addressed early (Ebrahim et al., 2011). Most of the benefits arising from early intervention become noticeable within a few years, with even modest changes having substantial public health benefits (Anderson et al., 1991). Unfortunately, for many developing countries, instead of developing an integrated, multidisciplinary approach to chronic disease management, the systems in place are outdated and parochial, at the very least, and unimaginative at best. This negates most of the constructive efforts designed to address the problem.

Currently, most public health resources are aimed at curative rather than preventative healthcare, in other words, treating CVD has become more of a priority than preventing it. The fact that CHD continues to occupy a low priority in present times is unconscionable and an indictment against modern healthcare. The challenge, therefore, is to find creative ways and cost-effective resources in order to minimize the burden of CHD.

In an attempt to address the pandemic of CHD, therefore, the present study investigates the impact of various health behavioural intervention strategies on CHD risk factors, HRPF and HRB of university students.
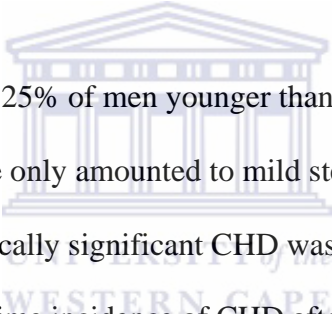
A critical review of the literature on CHD, in connection to HRPF and HRB, particularly in university students is therefore required.

2.2   Understanding Coronary Heart Disease

Historically, CHD is thought of as a man's disease that manifests mainly in old age (American Heart Association, 2003). Similarly, women's health issues were thought to revolve primarily around menopause and breast cancer, with CHD given scant attention. The literature, however, shows CHD to be a leading cause of death in women as well but with women less prone than men (Henderson, 1996). Women are at higher risk because of their lack of knowledge and their tendency to ignore early warning signs (American Heart Association, 2003; Steyl, 2008; Steyn, 2007).

CHD is a complex disease that causes reduced or absent blood flow to one or more arteries (American Heart Association, 2003). Apart from rare congenital anomalies, CHD is generally considered to be a progressive, degenerative disease, uncommon as a clinical problem before the age of 30 years, but common by the age of 60 years (McArdle et al., 2001, p. 714). There is established literature showing that fatty streaks are present in blood vessels in people of all ages (Franklin and Cushman, 2011; McGill et al., 2000), including infants (Barker, 1995), and may be genetically-linked (Herrmann & Paul, 2001). The development of CHD is shown schematically in Figure 2.1.

Autopsy observations revealed that 25% of men younger than 25 years have clinically significant coronary lesions, even though some only amounted to mild stenosis (McArdle et al., 2001, p. 719). However, after 35 years, clinically significant CHD was shown to be present in 75% of men (McGill et al., 2000). The lifetime incidence of CHD after 35 years exceeds 25% of all adults, in other words, one in every four people will have a cardiovascular emergency (Lopez et al., 2006). Of particular concern, however, is the fact that often the first overt sign of CHD is sudden death (American Heart Association, 2003).

2.2.1 Risk Factors for Coronary Heart Disease

Factors that influence the development of CHD are referred to as either risk factors or risk markers. Those factors that show direct causation are called risk factors, such as physical inactivity and cigarette smoking, and those that show an indirect association with CHD, but a

Onset of CHD is due to the presence of one or more risk factors: hypertension, smoking, family history, obesity, sedentary lifestyle, etc.

↓

Resulting chronic injury to endothelium (blood vessel lining) has an accumulative effect related to the number of risk factors present

↓

Progression to LDL-C oxidation, i.e., a process of LDL-C activation, distribution, impregnation, etc.

↓

Apo lipoprotein B stimulates monocyte macrophage infiltration and lipoprotein deposition

↓

Appearance of fatty streaks: initial damage to inner (endothelial) lining of arteries

↓

Plaque formation: initially lipid-filled soft, metabolically-active tissue that later hardens due to calcium impregnation

↓

Lesion formation (damage to smooth muscle walls): distinct bulge forms under the blood vessel endothelial lining due to clustering of lipid-laden macrophage cells

↓

Proliferative fibrous scar tissue changes

↓

Aneurysm formation: outward protrusion into arterial wall or inward bulge into vessel lining

↓

Thrombi formation: blood clots that develop from plaque rupture within blood vessel

↓

Heart muscle Ischemia

↓

Acute coronary event: angina, myocardial infarction or sudden death

Figure 2.1. Flow diagram of the development of coronary heart disease.

cause-and-effect relationship has not yet been proven, are called risk markers. These include high-sensitivity C-reactive protein and tissue plasminogen activator (Pearson et al., 2003; Yusuf et al., 2001).

Two important aspects about risk factors need clarification. Firstly, risk factors function on a scale of progressively diminishing returns with increasing risk rather than on an "all-or-none" principle. In other words, an individual with a risk factor classified as severe is more at risk than someone with a borderline value. For example, an individual with severe hypertension, that is, a systolic blood pressure (SBP) of 300 mm Hg, has three to five times the risk than someone with borderline hypertension, that is, a SBP of 140 mm Hg. Thus, a sliding scale of risk stratification exists called as a "risk pyramid", where the few at the top of the pyramid are at severe risk, while the majority at the base constitutes much lower risk (ACSM, 2006b, p. 95). The focus of most primary prevention strategies is targeted at the latter group comprising large numbers of individuals at lower risk (Ebrahim et al., 2011). Secondly, in the presence of multiple risk factors, the overall impact of each risk factor is combined to exert an exponentially magnified effect on CHD, the so-called "interactive, multiplicative effect" that outweighs the "pyramidal" effect (Raitakari et al., 1995). Thus, an obese individual who also smokes (multiple risks) is more at risk than the individual who is only obese (single risk), even if morbidly obese. Therefore, a complete assessment should be conducted which entails evaluating both the number of risk factors and the severity of each risk factor in order to accurately stratify CHD risk.

Risk factors are used expediently, because of their predictive value and cost-effectiveness (Rothstein, 2003, p. 2). They are safe and can be measured accurately and inexpensively using

basic equipment, thus saving time, effort, and overall cost. An example is body composition that

is based upon measuring height and weight. Height-weight tables were one of the first risk

instruments implemented by life insurance companies. It reflected one of the basic actuarial

principles, namely, to adjust insurance premiums based upon statistical differences in death rates

(Rothstein, 2003, p. 3). Thus, an individual having a desirable weight for height also had a lower

death rate and was given a better insurance premium in accordance with their lower risk.

2.2.2 Classification of Risk Factors for CHD

Risk factors for CHD can be classified as modifiable or non-modifiable, with the modifiable risk

factors further grouped into traditional or conventional and non-traditional, non-conventional or

emerging risk factors, as illustrated in Table 2.1 (Pearson et al., 2003).
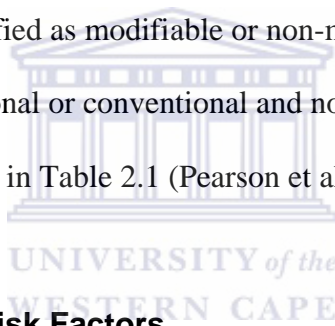
2.2.2.1 Non-Modifiable CHD Risk Factors

Age is associated with an increased risk for CHD in men from 45 years and in women from 55

years (ACSM, 2006b, p. 105). As with age, a family history of heart disease is established at a

younger age in men than women. A family history is considered to be present when clinical

evidence or sudden death occurs in male first-degree relatives younger than 55 years or female

first-degree relatives younger than 65 years (ACSM, 2006b, p. 105).

Table 2.1. Risk Factors for coronary heart disease.

Non-Modifiable	Modifiable		
	Traditional or Conventional	**Non-Traditional, Non-Conventional or Emerging**	
		Biological	**Psycho-Social**
• Age • Gender • Family history	• Dyslipidemia • Hypertension • Cigarette smoking • Sedentary lifestyle • Obesity (Excessive body fat; Central, abdominal or visceral body fat distribution - adiposity) • Impaired fasting glucose	<u>**LIPID Risk Factors**</u> • Lipoprotein (a) • Lipoprotein remnants • Small LDL particles • HDL subspecies • Triglycerides • Apolipoproteins B and A-1 • Total cholesterol-to-HDL cholesterol ratio • Endothelial lipase <u>**Non-Lipid Risk Factors**</u> • Sleep apnea • Anemia • Pre-eclampsia • Left ventricular hypertrophy • Ankle-Brachial BP Index • Renal Disease • Metabolic syndrome • Diabetes Mellitus	• Personality and behaviour patterns • Stress • Hormone replacement therapy

2.2.2.2 Modifiable CHD Risk Factors

2.2.2.2.1   Physical Inactivity

Physical activity that provides health benefits is described as 30 minutes of moderate-intensity activity that is performed either continuously or discontinuously at least 5 days a week (Blair et al., 1992). Research shows a lack of physical activity to be an independent risk factor for CHD (Sebregts et al, 2000; Franklin and Cushman, 2011). Globally, less than 60% of the population achieves the minimum amount of physical activity as recommended by the American College of Sports Medicine (ACSM) and the United States Center for Disease Control and Prevention (Myers, 2003). Physical activity tends to decrease with age and is less prevalent amongst those with disabilities or chronic diseases (Sebregts et al, 2000).
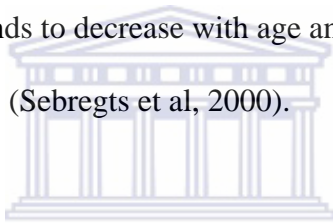
Pioneering studies by J.N Morris on London bus drivers and conductors in the 1950s (Morris and Crawford, 1958), and R.S. Paffenbarger on Harvard alumni in the 1970s (Sesso et al., 2000) showed that exercise protects against heart disease, in other words, it induces a cardioprotective effect. Their work has contributed enormously in promoting physical activity as a primary strategy for reducing CHD (ACSM, 2006a, p. 645). Since then, other studies have yielded similar findings showing that active subjects had lower rates of CHD than inactive ones, across all age and ethnic groups (Blair et al., 1995; Myers, 2003; Shiroma and Lee, 2010).

A systematic review of randomized controlled trials found that aerobic exercise can achieve clinically significant reductions in CHD risk (Ebrahim and Smith, 1997; Ebrahim et al., 2011). Multiple mechanisms are implicated in the process whereby moderate-to-vigorous physical

activity decreases mortality rates associated with CHD, including anti-atherosclerotic, anti-thrombotic, anti-ischemic, anti-arrhythmic, and psychological effects (Franklin and Cushman, 2011).

A variety of different interventions showed that exercise significantly lowers blood pressure and improves lipids and lipoproteins (Baruth et al., 2011; Eriksson et al., 2009; Myers, 2003). Independent of these indirect effects on CHD risk factors, regular exercise also exerts direct physiological effects, such as increased energy expenditure and associated weight loss (Ashen, 2010). Favourable psychological changes also acquired relate to enhanced body image, and elevated mood status, because of the greater amounts of circulating neurochemicals, such as serotonin and endorphins, in the brain (Ortega et al., 2008b).

In patients diagnosed with CHD, regular, brisk aerobic activity increases coronary blood flow through vasodilation, allowing more blood flow through the heart and, thereby, reducing both resting and exercise heart rates (Ashen, 2010) and increasing maximal oxygen consumption (McArdle et al., 2001, p. 320). Over time, prolonged endurance training improves ventricular function and myocardial efficiency, and reduces myocardial oxygen demand. These favourable changes ultimately serve to slow the progression of coronary atherosclerosis (Ashen, 2010). In addition, regular exercise can also stimulate the immune system to inhibit harmful agents, such as cytokines, that facilitate arterial disease and the development of plaque (McArdle et al., 2001, p. 900).

Exercise intensity and duration exert independent physiological effects, in a dose-dependent manner, on modifying CHD risk (Eriksson et al., 2009; Hedblad et al., 1997; Sesso et al., 2000; Tanasescu et al., 2002) in women (Colditz, 1997; Rollini et al., 2009), and in younger and older persons (Lee and Skerrett, 2001; Shiroma and Lee, 2010; Warburton et al., 2006). The evidence shows that exercise duration exerts its greatest effect on HDL cholesterol, while exercise intensity favourably modifies blood pressure and waist girth (McArdle et al., 2001, p. 899).

Adherence to physical activity that yields an energy expenditure equal to or more than 1000 kcal·wk-1 ($\geq$ 4200 kJ·wk^{-1}) or is performed for 150 min/wk at a moderate-intensity or for 75 min/wk at a vigorous-intensity is associated with a significant reduction in all-cause mortality (Sesso et al., 2000; Shiroma and Lee, 2010). Further reductions in risk were observed at higher volumes of energy expenditure, that is, 300 min/wk of moderate- or 150 min/wk of vigorous-intensity physical activity, but with progressively diminishing returns (Lee and Skerrett, 2001; Shiroma and Lee, 2010).

Regular physical activity, unlike other modifiable CHD risk factors, is shown to exert the single most potent effect on CHD, because of its direct positive influence as well as indirectly through other risk factors, such as, obesity, cholesterol, hypertension, diabetes, stress, and mental wellbeing (Arena et al., 2010; Garber et al., 2011). It is considered by many to be the single most important behavioural intervention to reduce CHD (Garber et al., 2011). In recent years, a strong impetus has been created by the ACSM which advocates that exercise be viewed as "medicine", because of its health benefits (Smitherman et al., 2007).

2.2.2.2.2   Cigarette Smoking

Cigarette smoking, whether done actively or passively, provides one of the strongest preventable risks for CHD (Critchley and Capewell, 2003; Naslund et al., 1996; Ward et al., 2003, WHO, 2002, p. 9). Smoking is not only considered atherogenic, but augments any vascular damage that already exists (Ward et al., 2003). It acts in a dose- and duration-dependent manner with the magnitude of risk directly proportional to the number of cigarettes smoked, the depth of inhalation, and the strength of the cigarette (Critchley and Capewell, 2003). Included among the damaging effects of smoking are acute increases in blood pressure, coronary vascular resistance, a reduction in oxygen delivery, impaired dilation of the coronary vessel wall, enhancement of platelet aggregation, increased fibrinogen production, and a decrease in HDL-cholesterol concentration (McArdle et al., 2001, p. 902).
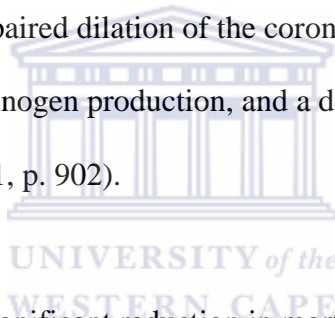
Smoking cessation may lead to a significant reduction in mortality, as well as a reduced rate of hospitalization (Critchley and Capewell, 2003). In coronary patients, the risk of having a coronary event declines soon after smoking cessation and is largely dissipated after 2 to 3 years. The beneficial effects of quitting were found to persist for at least 13 years (Critchley and Capewell, 2003).

Within the next 25 years, cigarette smoking is presumed to be the world's single leading cause of death and disability (McArdle et al., 2001, p. 902). Smoking results in a two- to three-fold risk of dying from CHD, making smoking avoidance and cessation a cornerstone for primary and secondary prevention (Ashen, 2010). The best CHD prevention action that could be taken by

non-smokers, is to continue to abstain, and by smokers, is to quit. Those who cannot stop should

be encouraged to improve their diet and start exercising, if not already active, in order to

counteract the hazards of smoking (Näslund et al., 1996). Research shows that even amongst

smokers, CHD risk is significantly reduced in physically active than inactive smokers (Hedblad

et al., 1997; Ward et al., 2003). Given the diverse health benefits and cost-saving from smoking

cessation, preventive efforts undertaken in this area should be encouraged and expanded,

especially in developing countries where the threat is the greatest, and where tobacco companies

have targeted their marketing for future growth.

2.2.2.2.3   Hypertension
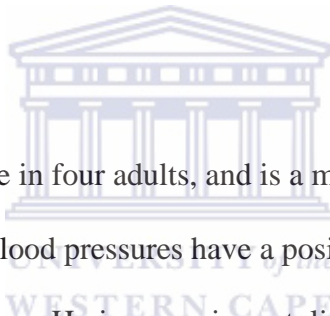
Hypertension is known to affect one in four adults, and is a major risk factor for CHD (Escobar,

2002). Both systolic and diastolic blood pressures have a positive, graded relationship with CHD

risk (Nghiem, 2007). For every 20 mm Hg increase in systolic blood pressure above 115 mm Hg,

there is a 50% increase in cardiovascular mortality (Escobar, 2002).

Hypertension is frequently seen as a precursor to cerebrovascular, renal and heart disease

(Nghiem, 2007). The pathophysiology linking hypertension with CHD starts with endothelial

dysfunction or injury caused by rapid increases in blood flow that exacerbate the atherosclerotic

process by making plaque more unstable (Escobar, 2002). Over time, persistent hypertension

results in left ventricular hypertrophy that decreases cardiac reserve and increases myocardial

oxygen demand, both of which contribute to myocardial ischaemia (Escobar, 2002).

The literature shows that stress and type-A behaviour personality (TABP) also affect blood pressure causing hypertension (Pulkki et al., 2003). Persons exhibiting TABP may habitually react to stress with an exaggerated response of the sympathetic nervous system, leading to chronic increases in catecholamine secretion. This chronic adrenergic activity contributes to hypertension that over time leads to CHD. Psychological stress may potentiate platelet activation and stimulate platelet aggregation, progressively leading to thrombus formation and, subsequently, occlusive activity in vulnerable individuals, such as those with TABP (Pulkki et al., 2003).

High blood pressure is a multi-factorial risk factor for CHD, influenced largely by lifestyle factors, such as body weight, exercise, diet, and alcohol consumption (Seedat et al., 2006). Along with other therapeutic measures, lifestyle modification is an important avenue used to modify blood pressure and minimize CHD risk (Ashen, 2010).
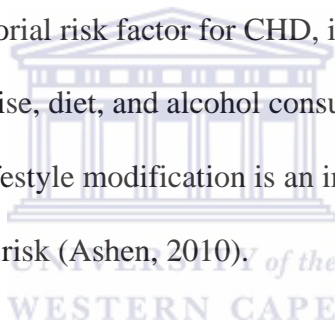
2.2.2.2.4   Obesity

Obesity invariably develops from a positive energy balance, with the risk of CHD directly related to the accumulation of visceral (central or abdominal) adiposity (The Emerging Risk Factor Collaboration, 2011). Obesity is thought to be partly inherited and partly due to environmental and lifestyle factors that create a positive energy balance for fat storage (Day and Bailey, 2011).

Being obese is a major risk factor for CHD, partly because obese individuals are more likely to have high blood pressure, be diabetic and have dyslipidemia, and partly because of visceral adiposity and coronary microvascular dysfunction (Onat et al., 2004). Because of the close proximity of visceral fat to the heart, it provides an adverse metabolic environment that fosters the development of microvascular dysfunction and CHD (Day and Bailey, 2011).

Although the distribution of fat between visceral and subcutaneous compartments varies widely, subcutaneous fat typically accounts for over 80% of body fat (McArdle et al., 2001, p.760). Women have more subcutaneous fat than men, but men appear to be more susceptible to the accumulation of visceral fat (McArdle et al., 2001, p.768). Most studies indicate that excess visceral fat, as opposed to excess subcutaneous fat, carries a much higher risk of cardiovascular disease (Day and Bailey, 2011; Mokdad et al., 2003; Onat et al., 2004). Glucocorticoids also contribute to the development of visceral adiposity by antagonizing the actions of insulin (Onat et al., 2004). They raise plasma lipids, increase hepatic gluconeogensis, impair glucose tolerance and stimulate food intake, all of which serve to increase adipocyte hypertrophy and visceral adiposity (Day and Bailey, 2011). Thus, several mechanisms exist through which increased adiposity causes CHD.

Weight loss is important, because it improves glucose tolerance, insulin resistance, blood pressure, and dyslipidemia and, ultimately, CHD risk (Ashen, 2010).

2.2.2.2.5   Dyslipidemia
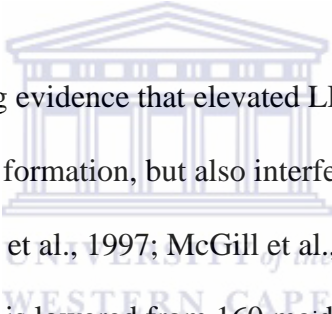
Lipids are transported in blood plasma in combination with a carrier to form lipoproteins that are produced in the liver and composed of a regulatory protein (apolipoprotein – Apo), a phospholipid, and cholesterol. Four types of lipoproteins act as carriers for cholesterol, namely, high-density lipoproteins (HDL), low-density lipoproteins (LDL), very low-density lipoproteins (VLDL), and chylomicrons. Total cholesterol (TC) is the composite of each of the different lipoproteins, that is, HDL, LDL and VLDL. However, the cholesterol distribution among the various lipoproteins is a more powerful predictor of heart disease than is total cholesterol.

Dyslipidemia is determined by three measurements, namely, elevated levels of atherogenic LDL cholesterol, that is, LDL >130 mg$^.$dl^{-1} or 3.4 mmol$^.$L^{-1}, reduced levels of protective (anti-atherogenic) HDL cholesterol, that is, HDL < 40 mg$^.$dl^{-1} or 1.03 mmol$^.$L^{-1} and, elevated levels of total serum cholesterol, that is, TC > 200 mg$^.$dl^{-1} or 5.2 mmol$^.$L^{-1} (ACSM, 2006a, p.22). Elevated fat or triglyceride in the blood is directly linked with raised blood LDL cholesterol levels, and when combined with low HDL cholesterol concentration, constitutes a lethal triad termed atherogenic dyslipidemia. This condition is commonly prevalent in individuals with premature CHD (ACSM, 2006a, p. 101).

The various lipoprotein ratios, that is, TC:HDL and LDL:HDL, as well as the triglyceride level are thought to have reinforcing value to the individual measurements (ACSM, 2006a, p.22). Thus, a TC:HDL ratio greater than 4.5 indicates high heart disease risk, while a value less than or equal to 3.5 represents more normal risk (McArdle et al., 2001, p. 898). The LDL:HDL ratio is

thought to be a marker of carotid artery plaque accumulation, with ideal levels rated as less than 4.4, normal risk between 4.4 and 7.1, moderate risk between 7.1 and 11; and greater than 11 seen as high risk (Gaziano et al., 1997). The ratio of triglycerides to HDL (Trig:HDL) indicates whether LDL is small and dense (bad) or large and fluffy (neutral) (Gaziano et al., 1997). The ideal ratio of Trig:HDL should be less than or equal to 2, with 4 being high, and 6 undesirable. Currently, the Trig:HDL ratio is believed to be a potent predictor of CHD, even stronger than the LDL:HDL ratio (Gaziano et al., 1997). From a treatment perspective, however, it is generally felt that the independent cholesterol measurements, that is, HDL and LDL, are more useful than using any of the ratios.

Previous studies provide convincing evidence that elevated LDL cholesterol levels not only contribute to atherosclerotic plaque formation, but also interfere with normal endothelial control of arterial vasomotor tone (Gaziano et al., 1997; McGill et al., 2000; Stark, 1996). In patients with known CHD, LDL cholesterol is lowered from 160 mg$^.$dl^{-1} to less than 100 mg$^.$dl^{-1}, and sometimes to even less than 70 mg$^.$dl^{-1} because the lower levels reduce the recurrence of heart attacks (Gaziano et al., 1997).

Whereas high LDL cholesterol and apolipoprotein B (Apo B) levels relate causally with an increased CHD risk, elevated HDL cholesterol and apolipoprotein A-I (Apo A-I) levels relate causally with a lower heart disease risk (Van Der Steeg et al., 2007). HDL levels should be kept high, that is, above 40 mg$^.$dl^{-1} or 1 mmol$^.$L^{-1} in men, and 50 mg$^.$dl^{-1} or 1.25 mmol$^.$L^{-1} in women (ACSM, 2006b, 45) because of its cardioprotective effects. HDL cholesterol brings excess cholesterol from the tissues to the liver for processing and excretion (McArdle et al., 2001, p.

716). HDL cholesterol, by acting through the compound Apo A-1, facilitates the removal of cholesterol from peripheral tissues to the liver for bile synthesis and subsequent excretion via the digestive tract (Van Der Steeg et al., 2007). A low level of HDL cholesterol is an important predictor of heart disease in men and women (Ingelsson et al., 2007). This condition may be inherited, or occur in individuals who are chronically inactive and/or obese (Ingelsson et al., 2007).

In a large population-based cohort, the overall performance of Apo B:Apo A-I ratio for predicting CHD was comparable with traditional lipid ratios, such as TC:HDL, and did not offer any additional value (Ingelsson et al., 2007). Consequently, there is little support for measuring Apo B or Apo A-I, when TC and HDL measurements are available.

Dyslipidemia already becomes evident in individuals in their late teens (McGill et al., 2000). The results are now consistently showing that CHD risk factors start to accelerate atherogenesis in the second decade of life already (McGill et al., 2000). Raised fatty streaks (fatty plaque) is the general term used to describe the endothelial blood vessel injury that occurs from the juvenile (flat) fatty streak and the raised atherosclerotic lesion. The endothelial surface area affected by raised fatty streaks increases with age, and is associated with elevated LDL cholesterol and reduced HDL cholesterol concentrations, as well as hypertension, obesity, and impaired glucose tolerance (McGill et al., 2000). Thus, prevention of CHD through lifestyle modification, weight reduction and regular exercise should begin as early as possible.

2.2.2.2.6   Impaired Fasting Glucose

Measuring fasting blood glucose (FBG) concentration is an indicator of steady-state glucose metabolism in the body (ACSM, 2006a, p. 101). One of the main routes though which excess adiposity impedes glucose metabolism is via an increased supply of fatty acids into the circulation (Day and Bailey, 2011). Under conditions of normal insulin sensitivity, insulin suppresses the activity of hormone-sensitive lipase and, thereby, reduces lipolysis. Fatty acids are taken up by the liver and muscle and used together with glucose as energy. This, in turn, limits the supply of fatty acids into the circulation. However, when adipocytes become enlarged they also become less sensitive to the antilipolytic action of insulin, causing an increased release and turnover of fatty acids into the bloodstream. Thus, an imbalance in the glucose–fatty acid cycle develops that increases the availability of fatty acids, and reduces the utilization of glucose. Consequently, fatty acid metabolites are produced that impair insulin sensitivity and further decreases glucose transport into muscle and promote chronic hyperglycaemia. Recent evidence shows that locally acting adipokines can modify insulin sensitivity and contribute to the development and maintenance of insulin resistance in overweight patients with insulin resistance (Day and Bailey, 2011).

Macrovascular disease typically develops as a consequence of poor glycaemic control, but is also strongly affected by dyslipidaemia and hypertension, all of which are associated with insulin resistance and commonly seen in obese individuals (Meigs et al., 2002). The result is the formation of an atheromatous plaque and, when unstable, precipitates thrombus formation that causes the typical diabetic macrovascular scenario. A further complication is that the

microvascular disease of CHD interacts with the macrovascular disease of diabetes, and vice versa (Day and Bailey, 2011). So, for example, the development of a diabetic foot and the increased risk for myocardial infarction reflect a combined failure of both small and large blood vessel function (Day and Bailey, 2011).

The current data from population-based prospective studies indicate that both type 1 and type 2 diabetes mellitus are established risk factors for CHD. Alternatively, a fasting glucose concentration indicating impaired fasting glucose is associated with pre-diabetes, and is only modestly associated with CHD risk (Sawar et al., 2010, Sung et al., 2009).
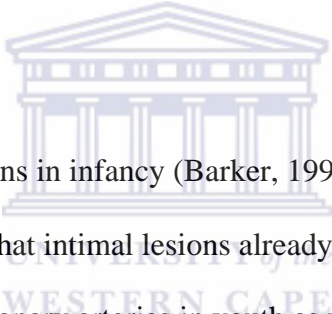
2.2.3 Non-Traditional and Emerging Biomarkers of CHD

Modifiable, traditional risk factors do not explain all of the risk associated with CHD. New and emerging risk factors have been shown to improve risk assessment for CHD (Helfand et al., 2009), such as high-sensitivity C-reactive protein (hs-CRP), a common inflammatory marker found in increased levels in patients at risk for CHD (Pearson et al., 2003). Another is osteoprotegerin which is involved in the regulation of a key inflammatory transcription factor called nuclear factor kappa B (NF-κB) (Venuraju et al., 2010). High-sensitivity C-reactive protein levels are increased in smokers, the obese, the sedentary, diabetics, and users of hormone replacement therapy (HRT), and remains the best new marker in screening for CHD, and the most rigorously studied (Pearson et al., 2003).

Other emerging risk factors thought to reflect an increased risk for CHD are coronary artery calcium (CAC) score, plasminogen activator inhibitor-1 (PAI-1), fibrinogen, homocysteine,

asymmetric dimethylarginine, brain natriuretic peptide (BNP), and carotid intima–media thickness (Helfand et al., 2009; Wang, 2006). Several new risk factors for CHD have been identified, but the available evidence shows the clinical value to be questionable and the predictive value inconclusive (Wang, 2006). Consequently, it appears that the addition of new biomarkers does little to improve the predictive value of the conventional risk factors. Future research on emerging biomarkers remains of limited value, and subject to more robust investigation, with genetic studies viewed as one of the primary avenues of enquiry (Naingglolan, 2011).

2.2.4 Onset of CHD

CHD is a complex disease that begins in infancy (Barker, 1995; Herrmann and Paul, 2001). McGill et al. (2000) demonstrated that intimal lesions already appeared in all the aortas, as well as in more than half of the right coronary arteries in youth as young as 7-9 years. The process of CHD proliferation is often described as a "lag effect" that becomes increasingly pronounced and more damaging with time (Raitakari et al., 1995).

Major risk factors that accelerate its development are a positive family history of CHD, hypertension, hypercholesterolemia, physical inactivity, smoking, obesity, and impaired glucose tolerance (Raitakari et al., 1995). The prevalence of CHD risk increases with age, that is, 64% of men and 63% of women have one or more major risk factors in the age group 45-54 years, while the prevalence of two or more risk factors is highest for men and women aged 65-74 years, that is, 80% and 89%, respectively (MacDonald et al., 1992).
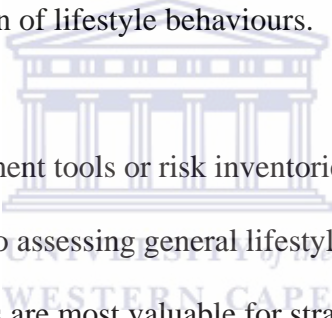
Other health-risk indicators implicated in the future risk of CHD include elevated body mass, particularly body fat, and early and rapid maturation (Lie, 2007; Remsberg et al., 2002; Hills et al., 2007, 20). Future assessments of CHD risk factors should continue to focus on early life measurements, in order to target early intervention and avert disease onset and progression (Remsberg et al., 2002).

2.2.5 CHD Risk Assessment

The measurement of traditional risk factors does not identify all CHD risk (Demyanets et al., 2011). However, the absence of major risk factors does reliably identify those individuals at low risk (Pasternak et al., 2003). Similarly, for high-risk patients, the major risk factors account for between 50% and 80% of subsequent cardiovascular events (Pasternak et al., 2003).

Emerging risk markers, such as the inflammatory markers, retinal artery narrowing, coronary artery calcification, endothelial dysfunction, anaemia and high-sensitivity C-reactive protein have been evaluated and reviewed, but the results are still inconclusive and await further investigation (U.S. Preventive Services Task Force, 2009; Rollini et al., 2009). The ability of these novel biomarkers to accurately predict risk remains limited, especially in individuals with asymptomatic or "subclinical" atherosclerosis (Helfand et al., 2009). A substantial gap, therefore, remains in the detection of asymptomatic individuals who ultimately develop CHD (Pasternak et al., 2003). Also, limitations exist in identifying individuals who should be targeted for therapy (Pasternak et al., 2003).

The frequent occurrence of multiple CHD risk factors signaling the onset and development of atherosclerosis is increasingly seen in young children (McGill et al., 2000). However, because the youth possess a limited knowledge and awareness of CHD risk, they do not perceive themselves as a population at risk for developing CHD (Frost, 1992). Therefore, preventive measures are seldom taken seriously by this group. This emphasizes the need for early assessment (Navas-Nacher et al., 2001) and early intervention to reduce the negative impact of atherosclerosis in later life (McArdle et al., 2001, p. 904). The American Heart Association (2003) suggests that screening for CHD should begin at the age of 20 years in order to facilitate early identification and modification of lifestyle behaviours.

Over the years, various risk assessment tools or risk inventories have been designed to assess susceptibility to CHD, in addition to assessing general lifestyle behaviours (McArdle et al., 2001, p. 720). These risk assessment tools are most valuable for strategizing primary prevention and identifying the level of prevention that ultimately guides treatment, with less usefulness for secondary prevention (Waldron et al., 2011). For example, the Framingham Risk Score (FRS), the most widely used risk assessment tool, allows for subject stratification into low 10-year risk (<10%), intermediate 10-year risk (10 - 20%), and high 10-year risk (>20%) (Anderson et al., 1991). The new modified FRS also determines vascular age ("heart age") that enables individuals to more readily grasp their CHD risk, because it shows how 'heart age' can change with a reduction in risk factors (Wilson et al., 1998). In addition to indentifying subjects at-risk, it can also help improve patient compliance to treatment and reinforce patient outcomes (Ashen, 2010).

2.2.6 CHD Risk Factor Associations and Clustering

When considering the most common associations between CHD risk factors, strong links are shown between smoking and high blood cholesterol (MacDonald et al., 1992), smoking and hypertension in men (Steyn et al., 1990), hypertension and hypercholesterolemia in women (Steyn et al., 1990) and between morbid obesity (BMI $\geq$ 40), hypertension, hypercholesterolemia and poor health (Morkad et al, 2003). The prevalence of high blood pressure and elevated blood cholesterol increases proportionately with increases in BMI and WHR (Morkad et al, 2003). High blood pressure and high blood cholesterol are closely related to the excessive consumption of fatty, sugary and salty foods (Steyn et al., 1990).

There is evidence that total and abdominal adiposity are associated with elevated blood pressures in subjects with low levels of cardiorespiratory fitness, that is, hypertension correlated significantly with higher fatness and lower fitness levels than with normotension (Ortega et al., 2008b). These associations become alarming when clustered with risks such as smoking and excessive alcohol consumption (Groenewald et al., 2007).

CHD risk factors, such as raised blood pressure, dyslipidemia, hyperglycaemia and elevated body mass index track consistently over time from childhood to adulthood (Nghiem, 2007; Lie, 2007; Hills et al., 2007: 20).

Several markers are responsible for the pathogenic risk factor clustering (Pasternak et al, 2003). These include biological markers, such as male sex and aggressiveness, as well as non-biological

markers, such as socioeconomic status, income level and level of education (Pasternak et al, 2003). A low level of education, a meagre income and a low socioeconomic status are inversely related to CHD risk (Hemingway and Marmot, 1999). Research also shows that BMI, cigarette smoking and total cholesterol are all inversely related to the level of education (Hemingway and Marmot, 1999; Kirkland et al., 1999; MacDonald et al., 1992).

Data from the National Health and Nutrition Examination Survey (NHANES) 2005 – 2008, show that low-income youth are more likely to be obese than their higher income counterparts, but the relationship is not consistent across race and ethnicity groups (Ogden et al, 2010a). Also, children and adolescents living in households where the head of household has a university degree are less likely to be obese compared with those living in households where the household head has less education (Ogden et al, 2010).
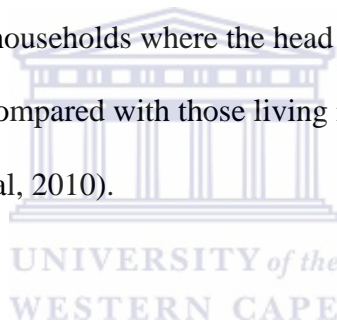
Amongst women, there is a trend that those with university degrees and/or higher salaries are less likely to be obese compared with those who are less educated and/or are lower income earners (Ogden et al., 2010b). MacDonald et al. (1992) reported that about 48% of individuals with a lower level of education were unaware of any major CHD risk factor.

There is some evidence that high levels of impatience and aggressiveness are associated with poor school achievement, downward occupational mobility, long-term unemployment, and a high level of CHD risk (Pulkki, 2003). These pathogenic components of type A behaviour pattern (TABP), have their origin in childhood hyperactivity, and are shown to predict adjustment problems, such as antisocial personality, poor school achievement, and the onset of

substance use (Pulkki et al., 2003). Barker (1995) reported that a mother's low social class might predispose the fetus to an unfavourable growing environment, thus programming the child's health even before birth. The findings of this study call for an approach that stresses collaboration between the different health sectors to reach the population as a whole, and especially those most vulnerable in order to reduce CHD.

Positive health habits, a higher level of education (being a lifelong learner), and a high sense of responsibility appear to be protective against risk factor clustering (Raitakari et al., 1995). Successful primary and secondary CHD prevention, however, requires early risk factor identification, changes in negative behaviours and the adoption of positive behaviours (Pulkki, 2003).

2.2.7 CHD Epidemiology

2.2.7.1 Global Prevalence of CHD

Compelling evidence exists showing CHD as one of the leading preventable causes of death in developed countries (Lopez et al., 2006; Mark et al., 2007; Murray and Lopez, 1997b; Pearson, 1999) as well as developing countries (Henderson, 1996; Akinboboye et al., 2003; WHO, 2002). In developed nations, 30 - 50% of all deaths are due to CHD (Lopez et al., 2006; Mark et al., 2007). One in two men and one in three women in their fifties are reported to develop CHD during their lifetime, with the risk increasing with age and a westernized lifestyle (Beranova & Sykes, 2007). With the exception of sub-Saharan Africa, where HIV/AIDS is most prevalent

(Akinboboye et al., 2003), CHD is expected to be the leading cause of global morbidity and mortality by 2020, (Levenson et al., 2002; Murray and Lopez, 1997a; Pearson, 1999), with the increase largely affecting males (Murray and Lopez, 1997a) at an increasingly younger ages (Barker, 1995; McGill et al., 2000). Between 1990 and 2020, CHD alone is anticipated to increase by 120% for women and 137% for men in developing countries, compared to increases of between 30% and 60% in developed countries (Gaziano, 2005). However, more epidemiological information is needed from developing countries, particularly in Africa, in order to predict future trends in CHD and to plan resources for treatment (Baruth et al., 2011).

In 1990, nearly 90% of the worldwide burden of disease occurred in developing regions, but only 10% of the funds were spent on healthcare (Murray and Lopez, 1997b). In terms of the global burden of disease, sub-Saharan Africa and India had the highest burdens of disease at 21.4% and 20.9%, respectively, but allocated the smallest budgets to healthcare, at 0.7% and 1.0%, respectively (Murray and Lopez, 1997b).

CHD is a serious public health burden in terms of life-years lost, reduced quality of life, reduced productivity and medical costs (Baruth et al., 2011). The direct cost of heart disease is twofold. Firstly, there is the direct cost of increased healthcare attributable to the costs for screening, laboratory tests, drugs, and clinic or doctor's visits. Secondly, there are indirect costs due to an absence from work, lower job productivity and lifestyle costs to support disease-related incapacitation (Baruth et al., 2011). Low-cost interventions that can be implemented successfully on a population-wide basis and result in significant reductions in CHD risk should be a public health priority, especially in this region.

2.2.7.2 Regional Prevalence of CHD

The prevalence of CHD is relatively low in most regions in Africa compared to developed countries (Akinboboye et al., 2003; Lopez et al., 2006; Mark et al., 2007). In most urban and virtually all rural regions of sub-Saharan Africa (SSA), the prevalence of CHD risk factors, particularly amongst blacks, has traditionally been low (Yusuf et al., 2001b). HIV/AIDS has been the major cause for the rise in mortality in SSA, and is attributable to unsafe sex (Lopez et al., 2006). The situation, however, is rapidly changing due to rapid westernization and urbanization and associated changes in lifestyle and technology (Lopez et al., 2006).

The growth in urbanization has brought with it a western lifestyle and an associated increase in CHD, especially amongst the poor (Lopez et al., 2006; Vorster, 2003). The lifestyle changes amongst urban blacks resulted in higher caloric and fat intakes, and lower levels of physical activity (Vorster, 2003). These risks worked synergistically to cause a higher prevalence of obesity and CHD (Kruger et al., 2005). Cigarette smoking in Africa has increased by more than 40% over the past two decades, particularly amongst young black men. This added further to the health problems in the region (Akinboboye et al., 2003).

There still remains a paucity of information on the epidemiology of CHD on a regional and national basis (Mark et al., 2007). Many countries in SSA do not have reliable national mortality statistics for examining CHD trends (Lopez et al., 2006). Also, many of the healthcare systems in place are fragile and characterized by poor infrastructure, a lack of adequately trained staff, and

poor management. Consequently, the resources needed to track an epidemic such as CHD are not readily available, and amplifies the crisis in healthcare in the region.

Research reports from small-scale population studies in Africa indicate that the burden from CHD in 1990 was considerably lower for blacks than whites, and lower than the rates reported in most western countries (Murray and Lopez, 1997a). Updated epidemiological data show that the mortality rate from CVD accounts for 10% of all deaths, and CHD accounts for 3% of all deaths (Yusuf et al., 2001b; Akinboboye et al., 2003). However, less data is available on the pattern of CHD risk factors (Yusuf et al., 2001b). Some limitations still exist around the reporting of reliable data that relate to the varying survey methods used in the region (Yusuf et al., 2001a).

2.2.7.3   National Prevalence of CHD
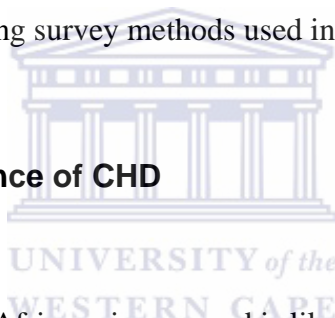
The general health status of South Africans is poor and is likely to become an even greater public health problem in future (Bradshaw et al., 2006; Kahn, 2007 and 2011; Norman et al., 2006). Since independence, public healthcare in SA has been left unprecedentedly burdened and incapacitated (Kahn, 2011). Population morbidity and mortality has worsened in virtually all age groups, driven largely by the HIV/AIDS pandemic (Bradshaw et al., 2006; Kahn, 2011; Norman et al., 2006). Life-expectancy has shortened by 12 for females and 14 years for males, respectively (Kahn et al., 2007).

SA has regressed as a country with a double burden of disease in the 1990s (SADHS, 1998), a triple burden at the turn of the century (Vorster, 2002) to its current position having a quadrupled

burden of disease (Mayosi et al., 2009). These diseases are classified as: (1) poverty-related conditions; (2) emerging chronic diseases; (3) injuries; and (4) HIV/AIDS (Bradshaw et al., 2003; Steyn and Fourie, 2007). HIV/AIDS prevalence is reported to be at 30%, with CVD mortality second at 17% (Bradshaw et al., 2003).

In 1991, spending on CVD in SA was between R4 - 5 billion, with the value of lost earnings reported at approximately R29.7 billion (Pestana et al., 1996). This expenditure reflected 2 to 3% of gross domestic product (GDP) or roughly 25% of all health-care expenditure (Steyn and Fourie, 2007). More than half the deaths caused by chronic diseases, including heart disease, occurred before 65 years. Premature deaths caused by heart and blood vessel diseases (CVD) in people of working age (35-64 years) are expected to increase by 41% between 2000 and 2030 (Kahn, 2011). The negative economic impact of this will be enormous (Steyn and Fourie, 2007).
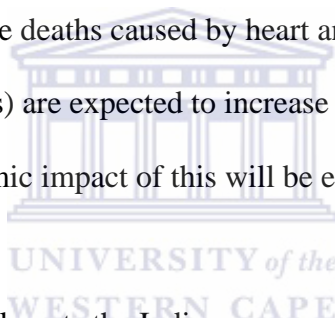
In SA, the CHD demographic profile puts the Indian community at highest risk, followed by people of mixed ethnicity, with whites and blacks least affected (Akinboboye et al., 2003; Norman et al., 2006; Steyn and Fourie, 2007). The rate of heart attacks, however, is steadily increasing for blacks, especially with increasing urbanization and the adoption of a western lifestyle (Vorster, 2002), and in rural areas as well (Alberts et al., 2005).

Amongst the Indian and mixed ethnic groups, a particular biological and psychosocial profile emerges, presumed to place them at risk. These include a high prevalence of diabetes and hypertension, a poor diet, high stress levels, poor access to health care, a possible genetic trait that increases the danger of triglycerides, especially in women, and a decreased production of

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nitric oxide which is critical for increasing blood flow in response to stress (Norman et al., 2007; Seedat et al., 2006; Steyn et al., 1990, 1997 and 2004).

Several behavioural and lifestyle factors are endemic to South Africans that place them at risk. In men, 30 years and older, the highest tobacco-related death rates are found in the population of mixed ancestry followed by Africans and Indians with whites the lowest (Groenewald et al., 2007). For women, 30 years and older, the tobacco-related death rate was much lower than for men. The highest rate was also found in the mixed ethnic population, followed by whites, then blacks and lastly Indians (Groenewald et al., 2007). Of particular concern is the high prevalence of smoking (46%) amongst pregnant women of mixed ethnicity. Not only do these women have babies with low birth-weight, but they also suffer many complications of pregnancy, some being life-threatening (Steyn et al., 2004).
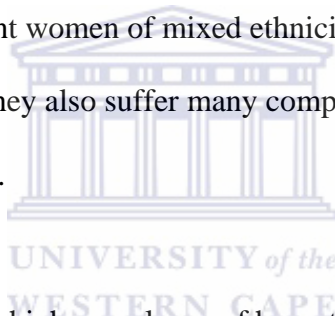
All population groups in SA have a high prevalence of hypertension (Vorster, 2002; Seedat et al., 2006; Steyn et al., 2001). Approximately 5 to 6 million South Africans 15 years and older suffer from hypertension (Norman et al., 2007), millions of whom are not adequately diagnosed, that is, only 26% of men and 51% of women knew they have hypertension, with fewer properly treated (Steyn et al., 2001).

Blood cholesterol levels vary considerably among the different population groups, with whites having the highest levels, followed by persons of mixed ethnicity, then Indians with blacks having the lowest rates (Norman et al., 2007; Oelofse et al., 1996). In blacks, favourable lipid profiles characterized by low total cholesterol and high HDL cholesterol may provide them with

a cardioprotective effect (Steyn et al., 1997). This positive effect together with genetically low homocysteine values has an inverse influence on CHD risk in this population (Akinboboye et al., 2003). However, they still have an increased risk for strokes (Steyn et al., 1991). This is due to the high prevalence of hypertension, obesity, smoking and hyperfibrinogenemia which, together with poor dietary patterns, have negated the cardioprotective benefits (Norman et al., 2007; Steyn et al., 2001) Vorster, 2002).

Obesity is another common problem amongst South Africans, with the highest prevalence in men found amongst the whites, followed by Indians, then persons of mixed ancestry, with blacks the lowest (SADHS, 1998). In women, blacks had the highest rates, followed by persons of mixed ethnicity, then whites and, the lowest in the Indian community (SADHS, 1998).

Based upon population surveys, most South Africans are reported as being physically inactive (Bradshaw et al., 2003 and 2006), that is also inclusive of the youth (Reddy et al., 2003).

As in most developing countries, the pattern of CHD in SA appears to be passing through a stage of transition, one in which differences in heart disease risk are based upon differences in population group (black vs white), geography (urban vs rural), socioeconomic status (rich vs poor), and genetics (disease-resistant vs disease-susceptible) (Kahn et al., 2007).

Therefore, a population that may have experienced rapid social and economic development (such as white South Africans) may experience an early increase in CHD and have a higher level of risk than other sectors (Indians and blacks). However, the decline in CHD in this population may

also occur sooner than in others. This transition of CHD from previously being a disease of the wealthy to one that currently affects the poor has been documented (Yusuf et al., 2001a).

The challenge of CHD for South Africans lies in identifying the risks most relevant to each community, and for action to be taken in order to suppress the increased future risk.In SA, during the past 20 years, CHD risk factors showed a predominantly unfavorable trend that applied not only to the adult population but, more alarmingly, to young people as well. It is important to assess the disease burden resulting from this risk so that the issues that need to be addressed can be identified, and cost-effective health interventions implemented in order to reduce the disease burden (Bradshaw et al., 2003).

2.2.8 CHD Prevention

CHD is particularly suited to prevention because it is a common problem in the population, has a long latency period, is easily modified by human behaviour with tremendous potential for cost-saving and public health benefits.

Historically, prevention is described as being of two types, that is, primary prevention that occurs before evidence of disease appears, and secondary prevention when disease is already present (Gaziano, 1998). Recently, prevention has been expanded to three basic types of intervention, namely, primordial, that is, the prevention of risk factors; primary, that is, the treatment of risk factors; and, secondary, that is, the prevention of recurrent cardiovascular events (Franklin and Cushman, 2011).

Traditionally, much of the focus has been on secondary than primary prevention. As the burden of CHD in developing countries rises, there is a strong view that primary prevention should be the cornerstone of public health intervention programmes (WHO, 2002; Yusuf et al., 2001b).

Two strategies commonly used in primary prevention are the population approach and the high-risk approach (Yusuf at al., 2001b). The population approach uses community-based interventions to modify behaviours and help reduce the number of risk factors. In the high-risk approach, a few high-risk individuals are targeted for intervention. Ultimately, in the population approach, even modest changes in risk produce substantial benefits because of the large numbers involved, whereas in the high-risk approach the benefits are individualized to a vulnerable few, with minimal benefit for the larger community (Yusuf et al., 2001b).
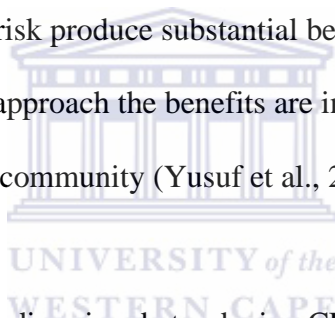
Several randomized intervention studies aimed at reducing CHD risk factors in the population show this approach as extremely challenging and have questioned its suitability when applied universally (Ebrahim et al., 2011; Ebrahim and Smith, 1997; Eriksson et al., 2006). In contrast, the high-risk approach demonstrated convincing proof of efficacy, whether from lifestyle or pharmacological interventions, but only in individuals with high-risk and not moderate CHD risk, and thus failed to address the burden in most of the population (Ebrahim, et al., 2011; Eriksson et al., 2009). The challenge, therefore, lies in blending the merits of the high risk approach with that of the community-based approach in order to comprehensively address all levels of risk that exists in the broader community. Intervention should start early, preferably in

childhood, where CHD starts, and continue across the lifespan, because the disease tracks through with age (Yusuf et al., 2001b).

Since CHD risk factors are invariably linked with lifestyle and human behaviour, prevention should also target behaviour change (Sebregts et al, 2000). Effective prevention requires evidence-based strategies that support vulnerable individuals and encourage most to adopt and maintain healthier lifestyles throughout their lifespan (Ashen, 2010). A proverbial "window of opportunity" still exists to prevent the epidemic from reaching its full potential (Gaziano, 2005). However, for the disease to be contained, affordable resources, cost-effective programmes, and accessible strategies need to be rapidly deployed. Ultimately, such interventions should aim to make healthy human behaviours a social norm for most, if not for all.

Lifestyle programmes used in CHD prevention studies include education together with diet, weight management, exercise, and smoking cessation (Ebrahim et al., 2011; Fernandez et al., 2007; Kemper et al., 2002). Instilling self-efficacy in participants is achieved by targeting changes in knowledge, attitudes and skills. Individuals ultimately better understand how negative behaviours are learnt early in life, and how to change them in future (Smith et al., 1997; Steptoe et al., 1999). Such life skills that significantly reduce disease risk and cut costs, while improving healthcare and quality of life, have also proven to be cardioprotective (Ashen, 2010).

During the last decade, there has been an increase in educational computer-based technology and electronic learning. This increase, in addition to being cost-effective and less time-consuming, translates into improved communication, better health outcomes, enhanced compliance, and

more empowered patient decision-making (Beranova and Sykes, 2007). Computerized educational systems seem to offer an ideal opportunity for comprehensive education, and increasingly appears to represent the way of the future, especially for university students who typify independent, adult learning (Bayne-Smith et al., 2004; Beranova and Sykes, 2007).

In SA, as in many other countries, there is growing enthusiasm for heart health programmes that use counseling and educational methods to encourage people to reduce their risks for developing heart disease (A Report of the Surgeon General; 2011; American Heart Association, 2003; Pestana et al., 1996; Steyn and Fourie, 2007). Counseling, when that is tailored to the individual's stage of readiness to change and directed at improving knowledge and attitudes, is reported to be more effective than conventional approaches to health education, (Lie, 2007; Calfas et al., 1996).

Recent scientific literature indicates that a theoretical cardioprotective polypill has been proposed as a population strategy to combat CHD (Franklin and Cushman, 2011. The pharmacological formulation of such a polypill would include a statin, 3 blood pressure–lowering drugs, folic acid and aspirin. The combined effect is purported to reduce coronary events by 88% and stroke by 80%. However, the polypill is still highly controversial, and recommended as an adjunct to it is lifestyle modification based upon regular moderate-to-vigorous physical activity, a low-fat, low-cholesterol diet, weight management, and the avoidance or cessation of cigarette smoking (Franklin and Cushman, 2011).

Most of the literature on CHD prevention and treatment is derived from studies conducted in developed countries and among select populations. There is, therefore, an urgent need for studies that can contribute to the current body of knowledge on CHD prevention, but are also culturally-sensitive, population-based and evidence-driven (Yusuf et al., 2001b). Such studies, if well-constructed, have the potential to translate into effective and efficient strategies of best practice.

2.3   Understanding Health-Related Physical Fitness

Physical activity and physical fitness represent distinct, yet related, concepts. Physical activity is commonly described as any bodily movement produced by skeletal muscle that results in energy expenditure (Caspersen et al., 1985). Physical fitness represents a set of physical and/or physiological characteristics that is partially inherited and partially achieved through regular physical activity or exercise (Caspersen et al., 1985; Ortega et al., 2008b). The components of physical fitness related to health include body composition, cardiorespiratory fitness, muscular endurance (stamina), muscular strength, and flexibility (Caspersen et al., 1985; Ortega et al., 2011).

Several test batteries exist to assess HRPF in young people, for example, the EUROFIT battery in Europe (Committee of Experts on Sports Research EUROFIT, 1993) and the FITNESSGRAM battery in the USA (Cooper Institute for Aerobics Research, 1999). Most contemporary studies on HRPF follow the guidelines stated in these fitness batteries (Jourkesh et al., 2011; Kemper et al., 2002; Monyeki et al., 2005; Ortega et al., 2005, 2008 and 2011).

For many individuals, particularly those with low levels of physical fitness, increases in physical fitness can be achieved by increasing one's physical activity (Blair et al., 1992; Garber et al., 2011). The results from epidemiological studies and randomized controlled trials indicate that increased levels of vigorous physical activity, rather than light-to-moderate physical activity, are associated with higher levels of cardiorespiratory fitness in children and adolescents (Katzmarzyk et al., 1999; Ortega et al., 2005; Ortega et al., 2011). The genetic component in physical fitness makes it highly variable across individuals (Rankinen et al., 2006). Individuals who are physically active report higher levels of physical fitness (Blair et al., 2001), reduced age-related weight gain (Shiroma and Lee, 2010), and an absence or reduction in cardiovascular risk factors (Wang et al., 2010).

2.3.1 Relationship Between Health-Related Physical Fitness and CHD

A review of observational epidemiological studies indicates that an inverse relationship exists between of physical activity and CHD risk. This also applies to physical fitness and CHD risk (Blair et al., 1992 and 2001; Myers, 2003; Wang et al., 2010). The relationship may be stronger between physical fitness and CHD risk because of the greater precision with which physical fitness is measured (Blair et al., 2001). In contrast, variable techniques are used to quantify physical activity and are often biased by self-reporting (Ainsworth et al., 1993).

Data derived from systematic reviews provides evidence that cardiorespiratory fitness can be achieved by following current physical activity guidelines recommending at least 150 min/wk of moderate-intensity physical activity, such as brisk walking (Garber et al., 2011; Eakin et al.,

2007; Smitherman et al., 2007). In addition, data from the Aerobics Center Longitudinal Study shows that a moderate level cardiorespiratory fitness is associated with a lower rate of premature mortality and CHD risk (Shiroma and Lee, 2010).

When making comparisons of physical fitness results, children perform poorer than adults, women poorer than men, and the obese poorer than individuals with a weight within the normal range. There is no consistent pattern across race or ethnicity, socioeconomic status and level of education (Shiroma and Lee, 2010; Wang et al., 2010).

The current literature supports the clinical value of cardiorespiratory exercise testing in at-risk cohorts, whether individuals have suspected or confirmed CHD (Blair et al., 2001; Katzmarzyk et al., 1999). A substantial body of evidence indicates that a decrease in mortality risk is directly linked with an improvement in the level of physical fitness (Arena et al., 2010; Coetsee, 2003; Grundy et al., 1999; Lee and Skerrett, 2001; Tikkanen et al., 1998). Maximal aerobic capacity has been demonstrated to be a more powerful predictor of CHD risk than other clinical and exercise variables (Ortega et al., 2008b). $\dot{V}O_2$peak was also a significant predictor of future adverse events, with peak $\dot{V}O_2$ values of ≥ 13 ml $O_2 \cdot kg^{-1} \cdot min^{-1}$ associated with a 50% reduction mortality risk compared to those below this threshold (Arena et al., 2010). It has been suggested that adverse cardiac events are related to aerobic capacity thresholds ($\dot{V}O_2$peak values) in the ranges of <5, 5–8 and >8 METs indicating high, intermediate and low levels of risk respectively (Arena et al., 2010). In young girls and boys, a low aerobic capacity, that is, <37.0 ml$\cdot$kg$^{-1}\cdot$min^{-1} and <42.1 ml$\cdot$kg$^{-1}\cdot$min^{-1} in girls and boys respectively, appeared to be a strong predictor of increased cardiovascular risk factors in later life (Ortega et al., 2008b; Arena et al., 2010).

High levels of physical fitness and physical activity are consistently associated with reduced body weight, total adiposity and lower visceral adiposity (Ortega et al., 2008b). Physical activity also causes favourable changes in lipid profiles, that is, lower levels of total cholesterol, LDL cholesterol, and triglycerides, and increased levels of HDL cholesterol (Tikkanen et al., 1998). These changes act to modify CHD risk in ways that are cardioprotective and anti-atherogenic (Myers, 2003).

Data collected from the AVENA study on adolescents showed an inverse relationship between muscular fitness and CHD risk (Ortega et al., 2008b). Muscular fitness, when combined with cardiorespiratory fitness, produces a cumulative effect that acts to significantly improve overall cardiovascular health, especially when started at an early age (Ortega et al., 2008b).

In addition, muscle enzyme activity for lipid metabolism in skeletal muscle was significantly lower in both CHD patients and sedentary or low-fit subjects (Tikkanen et al., 1998). As a consequence, skeletal muscle profiling may be an important determinant of risk, and is recommended in studies that assess CHD risk factors (Tikkanen et al., 1998).

There is a dirth of literature on the physical fitness levels of the South African population, especially young adults (Steyn 2006; Reddy et al., 2003). Andrews et al. (1985) compared the physical fitness levels of South African and American university students aged 18 - 24 years. In this study, students from three universities in the Western Cape (SA) were compared to students from the University of Utah (USA) according to the Canadian Association for Health, Physical

Education and Recreation (CAHPER) test battery of 1970. The results showed that the Utah students demonstrated significantly higher levels of physical fitness than their South African counterparts. However, students from the USA were also heavier, with greater skinfold thicknesses at the abdominal site, indicative of greater visceral adiposity and an increased risk for CHD (Onat et al., 2004). The study also indicated a trend of diminishing returns in fitness levels with age, and highlighted the importance of participation in physical activity and exercise early in life, especially before the pubertal growth spurt for the health benefits to be maximised.

A comparative study by Coopoo et al. (2003) on the fitness levels of South African youth of Indian descent in 1977 and in 1997 showed that most of the subjects were not regularly active, and could not attain even the minimal level of health-related fitness in order to reduce their risk.

Frantz (2004) conducted a cross-sectional study in which learners from four high schools in Belhar (SA) were assessed for daily physical activity patterns through a 24-hour recall. Their HRPF and views on physical activity participation were also investigated. This study showed that 32% of learners were physically inactive, and amongst the inactive learners, 23% were overweight, 15% symptomatic of hypertension, and 50% failed to meet the minimal requirements of HRPF (Garber et al., 2011). Inactive learners were also more likely to engage in HRB, such as smoking, practising unsafe sex and consuming alcohol. This raised concerns as lower fitness levels in adolescents track into adulthood and predispose these learners to future risk (Kvaavik et al, 2001). This evidence identifies physical fitness as an important health index for signalling underlying pathology early in life and reflects the need to promote higher levels of physical activity and physical fitness amongst South African youth.

The Hong Kong Student Obesity Surveillance (HKSOS) project in 2006-2007 found that more boys were overweight or obese than girls, but more girls were underweight. Boys performed significantly better on most HRPF tests, except in flexibility, where the girls excelled. A trend towards increased physical fitness was observed in the boys as they aged, whereas fitness levels stabilized in girls across age.

Similar results were reported for university students (Jourkesh et al., 2011). Performance on the HRPF tests was inversely related to obesity and underweight in both sexes, and was described as an inverted J-shape association, that is, underweight and obese subjects had the poorest results, while those with normal body weight produced the best performances.
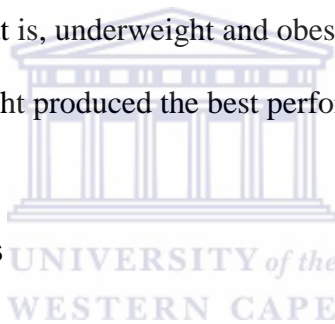
2.4   Health-Risk Behaviours

The World Health Report (2002) presented findings from a global review of risk factors, and identified ten health risks that account for more than a third of all deaths worldwide. The health risks were unsafe sex, alcohol consumption, tobacco consumption, obesity, high blood pressure, high blood cholesterol, iron deficiency, under-nutrition, unsafe water, lack of sanitation and hygiene, and indoor smoke from solid fuels. At least 30% of all disease burdens in developing countries come from less than five of these risks (Lopez et al., 2006). Furthermore, more than three-quarters of CHD results from tobacco use, high blood pressure and high blood cholesterol (Lopez et al., 2006).

Overall, tobacco use causes more than 5 million premature deaths per year (Lopez et al., 2006)

The effect of the massive increase in tobacco consumption on disease mortality, especially in developing countries that started in the 1970s and 1980s, is presumed to cause tobacco-related mortality to increase in excess of 8 million by 2020 (Murray and Lopez, 1997b).

Alcohol was estimated to have caused about 750 000 more deaths worldwide, with more than 80% of these deaths in developing countries (Murray and Lopez, 1997b). Alcohol consumption is associated with a range of high-risk behaviours, including unsafe sex and the use of psychoactive substances. As a result, disorders due to alcohol-use carry a high degree of comorbidity with other substance-use disorders, such as nicotine dependence and sexually transmitted infections. There is an established body of evidence of an association between alcohol-use disorders and HIV/AIDS (WHO, 2002).

Unsafe sex was estimated to account for more than 6% of the regional disease burden in sub-Saharan Africa, while in women aged 15 to 44 years, unsafe sex was estimated to account for 30% (Murray and Lopez, 1997b).

A disparity exists between young adult women and men in terms of the symptoms of ill health. Women are shown to be less likely to have good health and favourable health-related performance compared to men (Eriksson et al., 2007). The risk factors among young adult women are not only related to current chronic disease, but also to HRB, such as a lack of physical activity. Jones et al. (2007) found that female learners were of the opinion that they were most vulnerable to drug experimentation and excessive drug use, and emphasized the need

for future research to focus more explicitly on investigating the ways that females experience their vulnerability to HRB. The acquisition of life skills was viewed as the most important vehicle for engaging females in overcoming their vulnerability and developing a healthier lifestyle by becoming more physically active, controlling the diet and properly managing body weight (Jones et al., 2007). The gender inequality in health and health-related performance stresses the importance of understanding gender-based psychosocial factors when addressing HRB and designing health promotion activities and programmes.

In men, the prevalence of high levels of trait anxiety was associated with the progression from pre-hypertension to hypertension and incident CHD (Vega, 2007). Long-term psychological stress, the bane of most males, was also associated with an increased risk of CHD (Pasternak et al, 2003). Regular physical activity was recommended as a panacea for both trait anxiety and chronic psychological stress (Vega, 2007).
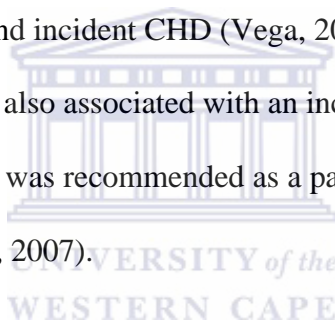
The top five causes of stress among university students were identified as anxiety caused by the start of the first year of university, social integration, a change in lifestyle habits (eating, sleeping and travelling) and managing their academic studies (Gibney et al., 2011; Ross, 1999). The students' anxieties are largely unfounded and overestimated (Gibney et al., 2011). These anxieties nevertheless manifest themselves in students engaging in HRB. Tumusiime (2004), for example, found that 70% of the university students were physically inactive, with females more affected than males. A decline in physical activity was most noticeable in the transition from secondary school to tertiary education. Tamim et al (2004) found that 30% of students were trying to lose weight, with females more preoccupied with weight loss than males. Omoteso

(2006) further found that 63% of students who were in a relationship had sexual intercourse. Condoms were never or rarely used by 35% of those who were sexually active (Omoteso, 2006). In a study conducted by Ma et al. (2006) it was found that pregnancy and induced abortion were experienced by approximately 10% of sexually active females, while 1.5% of sexually active students were diagnosed with STDs. Lee et al. (2007) reported a high rate of fatigue among graduate students as a result of the levels of anxiety experienced.

First year university students have poor nutritional habits, which are associated with increased risk factors for CHD (Irazusta et al., 2007). Shifts in dietary habits and activity patterns reflect higher fat intakes and less physical activity, contributing to a higher prevalence of obesity (Grundy et al., 1999; Westerterp, 1999; Kruger et al., 2005). Few overweight black women view themselves as overweight (Kruger et al., 2005), and some associate thinness with HIV/AIDS (Matoti-Mvalo and Puoane, 2011). Therefore, greater resources need to be allocated to the nutritional education of students at risk and obesity prevention and treatment should be based upon the correct education and the eradication of misconceptions.

Steptoe et al. (1997) assessed the prevalence of exercise, health beliefs, health-related behaviours, and emotional well-being among young adults. The associations between health-related behaviours and emotional well-being suggested that regular physical exercise was a behaviour that was consistently associated with a healthy lifestyle across all cultures. Also, the consistency of the association between physical exercise and health belief, justified focusing attention on addressing attitude change in preventive programmes. However, a lack of

knowledge in young adults concerning the consequences of a sedentary lifestyle remained a cause for concern (Steptoe et al., 1997).

When comparing eastern and western European students, Steptoe and Wardle (2001) showed that the eastern European students led a less healthy lifestyle due to a lack of information about health and behaviour, greater beliefs in uncontrollable influences and diminished emotional well-being. Eastern European students were also less likely to be aware of the relationship between lifestyle factors, such as smoking, fat and salt consumption, and CHD risk. These researchers also reported lower social support for these students and they were more prone to depression. The presence of HRB in a relatively well-educated sector of society emphasized the need for proper education, as well as the importance of developing positive attitudes and lifestyles early in life (Steptoe et al., 2002).
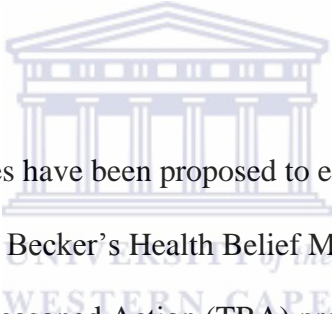
Haase et al. (2004) assessed the prevalence of HRB, health beliefs, and knowledge of the risks of inactivity across 23 developed and developing countries. Leisure-time physical activity for many students was below the recommended level, and was related to cultural factors and the state of national economic development. Participation in leisure-time physical activity was positively associated with the health belief in the benefit of physical activity and with national economic development. The students' knowledge about physical activity and health was poor, with only 40 to 60% aware that physical inactivity was linked to heart disease risk. This study indicates that the relationship between health belief and behaviour may be robust across cultures, but that students' knowledge about health and HRB is alarming, and remains an indictment against current educational and public health practices.

In many ways, healthy lifestyle habits help define individuals and sectors in society who are at low risk for developing CHD. In a Nurses' Health Study report, the relative risk of incident CHD was 82% lower among physically active, non-smoking, non-obese women (Colditz et al., 1997). The nurses also consumed a diet high in cereal fibre, omega-3 fatty acids, and folate, with a high ratio of polyunsaturated to saturated fat and low transfats and glycemic load. They also were reported to have at least half a drink of an alcoholic beverage daily (Colditz et al., 1997).

The high prevalence of HRB amongst students adds an additional burden to the risk factors and signifies increased risk of developing CHD which may, in future, become characteristic of this population (Haase et al., 2004; Lee et al., 2007; Ma et al., 2006; Omoteso, 2006; Steptoe et al., 2002). Despite an increased awareness of the negative consequences of various health risks, there has not been a reciprocal decrease in risk-taking behaviours among students (Choi and Choi, 2007). Furthermore, health behaviours in adolescence and young adulthood generally track through with age and have an impact on disease burden in later life (Von Ah et al., 2004). Thus, the presence of HRB in youth may represent an early warning system for future risk of developing CHD (Remsberg et al., 2002; Thomas et al., 2003; Schmidt et al, 1998). The challenge, therefore, is to identify those who will ultimately develop CHD accurately, and to take pre-emptive action. Clearly, establishing healthy habits early in life confers a low risk for CHD and has the potential to track through with age and confer good health in later life. The youth, therefore, need to be informed of the benefits of healthy habits.

2.5   Theoretical Basis for Health Behaviour Change

Health behaviour change is a complex phenomenon, not easily understood and, certainly, not easily changed (Noar et al., 2008). Understanding the factors that influence behaviour is important when designing intervention programmes, especially those that target the youth (Choi and Choi, 2007). In this study, HRB are defined as those behaviours historically viewed as "risky" behaviours, for example, smoking, not using condoms, and not wearing a seat-belt, as opposed to health behaviours that are protective and promoted in major public health campaigns, such as not smoking or quitting smoking, using a condom, and protecting the environment (Choi and Choi, 2007).

Different health behavioural theories have been proposed to explain why people engage in health promoting behavior. These include Becker's Health Belief Model (HBM) proposed in 1974, Ajzen and Fishbein's Theories of Reasoned Action (TRA) proposed in 1980, Prochaska and DiClemente's Transtheoretical Model (TTM) proposed in1983, stages of readiness to change (1986), Bandura's Social Cognitive Theory (SCT) proposed in 1991 and Ajzen's Theory of Planned Behaviour (TPB) (ACSM, 2006c, pp. 546-547; Noar et al., 2008). In recent years, newer theories have been proposed, such as Fisher and Fisher's Information-Motivation-Behavioural Skills Model (IMB) and Weinstein and Sandman's Precaution Adoption Process Model (PAPM) in 2002 (Noar et al., 2008). These health behaviour theories attempt to explain why individuals take risks (are risk-seekers) or do not take risks (risk averse), and how they go about changing HRB (Noar et al., 2008). The basis for successful health promotion and lifestyle management, as postulated by these models, depends upon sustaining long-term changes that are achieved by

targeting the relevant determinants of health behaviour (ACSM, 2006c, pp. 546-547). Certain determinants, such as personality traits and knowledge, only explain in part why people engage in health behaviours, and fail to take other factors into consideration, such as attitudes, social support, self-efficacy and perceived barriers (Kemper et al, 2002). There is also concern that some health decision-making models, such as the HBM, TRA, and TPB are less successful in predicting young adults' risk-taking behaviours where situational and emotional factors are presumed to be important (Choi and Choi, 2007).

The intervention strategies considered in this study are based upon a theory that emphasizes the importance of cognition as well as behavioural processes in changing behaviour. The structure and content of the various interventions used in this study have been designed based upon the Transtheoretical Model (TTM) of behaviour change. This model focuses explicitly on behaviour change as the progression through a series of discrete stages, therefore, the alternative name, stages of change model, and promotes self-efficacy as a central concept (Prochaska and DiClemente, 1992). With this model, participants tend to have better adherence to intervention programmes when given the choice (cognitive, decision-making ability) of using various intervention strategies, and when perceiving themselves to be in control (self-efficacy) of their behaviours (Epstein, 1998).

In recent times, there has been an educational shift in the methods used to teach youth about health-enhancing lifestyles (Choi and Choi, 2007). Previous programmes were designed simply to instill knowledge and information about health facts (Griffin, 2006, pp. 3-19). Current programmes are more educationally sound and are designed to instill social and behavioural

skills that are required to translate knowledge into practice, and are geared towards social enlightenment and empowerment (Griffin, 2006, pp. 3-19).

The TTM has been increasingly incorporated in many health promotion programmes (Noar et al., 2008). One of the educational aspects of the model is that it lends itself to active interventions (Griffin, 2006, pp. 19-40). The model describes how people adopt risky behaviours, as well as the change that occurs in them (Prochaska and DiClemente, 1992). The theory identifies ten psychological processes describing how people move through the stages. Some processes are mainly important for explaining movement from one particular stage to another. Other components of the TTM comprise decision-making (balancing the pros and cons of inactivity), self-efficacy (that is, having the self-confidence to become active or to remain an active person), and resisting temptations (to remain or become sedentary) (Prochaska and DiClemente, 1992).
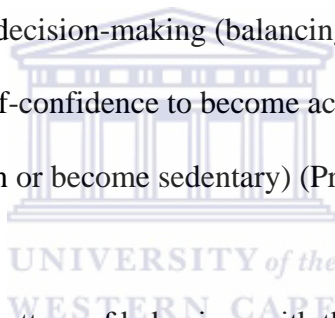
TTM confronts a person's current pattern of behaviour with their intention to either maintain or change that behaviour in future. For example, when applied to physical activity, five stages of behaviour change (motivational readiness) are hypothesized, namely:

- Stage 1: *Precontemplative Stage*, that is, the person is not thinking about becoming physically active in the immediate future;

- Stage 2: *Contemplative Stage*, that is, the person has advanced one step ahead and is now thinking about making a behaviour change and becoming physically active in the near future;

- Stage 3: *Preparation Stage*, that is, the person is currently engaging in physical activity, but does not meet the recommended levels for comprehensive health benefits, according to the ACSM guidelines (ACSM, 2006b. p.22);

- Stage 4: *Action Stage*, that is, the person is currently engaging in physical activity at the ACSM recommended levels for less than 6 months;

- Stage 5: *Maintenance Stage*, that is, the person is engaging in physical activity at the ACSM recommended levels for 6 months or longer; and

- Stage 6: *Relapse Stage*, that is, the person has fallen back into previously unhealthy behaviours of physical inactivity that do not meet the recommended levels for health.
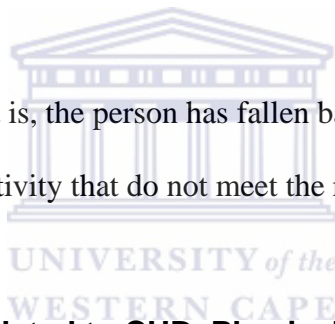
2.6 Intervention Studies Related to CHD, Physical Fitness and Health-Risk Behaviours

There is a compelling body of evidence showing the benefits of health promotion programmes (Pulkki et al., 2003; Saelens et al., 2000; Steptoe et al., 1997; Steptoe et al., 1999; Steptoe et al., 2001; Steptoe et al., 2002). HRB related to diet, exercise, and smoking develop gradually during childhood and early adolescence and, eventually, become habitual during adulthood (Pulkki et al., 2003). One preventive strategy is to provide sound educational programmes designed to promote healthy habits at an early age (Pulkki et al., 2003). Additional supportive influences, related to psychosocial and environmental factors, such as parental support, self-enjoyment, peer

influence, and social access to equipment and facilities should also be incorporated (Trost et al., 1997). These have all been proven to impact significantly on compliance and ultimately upon behaviour change (Trost et al., 1997).

The need to change HRB, especially early in life, should be seen as a public health imperative and as a pivotal preventive strategy that impacts positively on the quality of life. Interventions designed to address CHD risk factors and health risks invariably target a number of health behaviours, specifically diet, physical activity and stress management (Rosenberg et al., 2007). Health behaviours may operate independently, meaning that change in one kind of behaviour might not impact on change in another (Rosenberg et al., 2007). Alternatively, there is also the likelihood that health behaviours are interrelated in a synergistic way, where improvements in one might positively affect others (Rosenberg et al., 2007). Hence, if health behaviours tend to covary in a positive way, then manipulating these behaviours might suggest a more efficient way to target health risk (Rosenberg et al., 2007).

The general approaches that are used to modify health behaviours in interventions are individual-based interventions, group-based interventions, computer/technology-based (interactive session, personal or automated telephone calls) interventions and multicomponent interventions (Artinian et al., 2010).

Individual-focused interventions consist of a health risk appraisal, activity counseling, and/or cognitive behavioural strategies that use in-person, telephonic, electronic and combined

approaches. It allows for tailoring or personalization of healthcare recommendations to the individual's particular health concerns and lifestyle (Artinian et al., 2010).

Typically, group interventions are administered in small, closed group formats, that is, 7 to 10 members per group (Artinian et al., 2010). Group-based interventions are characterized by opportunities for social interaction, and support from others who are experiencing similar challenges in HRB (Artinian et al., 2010).

With the growth of computer technology and the internet, health interventions are increasingly delivered online or with the use of technology. The advantages of internet-based interventions include the ability to reach many people with a single posting, easy storage of large amounts of information, ease of updating information, ability to provide personalized feedback, cost effectiveness and convenience for users (Artinian et al., 2010).

Multicomponent programmes include combinations of technology or media that uses group or individual-based delivery strategies, such as interactive computer-based programmes plus telephonic follow-up and community resource enhancement (Artinian et al., 2010). Additional strategies include computerized assessment and feedback plus videotapes, telephonic follow-up, or individual counseling, physician advice and motivational videotapes, telephone calls, interactive mail, group sessions and individual motivational interviewing, or individual and group sessions (Artinian et al., 2010). In most multicomponent studies, various behavioural strategies were also included, such as goal setting, self-monitoring, feedback, social support, problem solving, or motivational interviewing (Artinian et al., 2010). The results in these studies

have been limited by the duration of the intervention or lack of effective combination of behaviour change strategies (Artinian et al., 2010). The optimal combination of behaviour change strategies in multicomponent interventions has yet to be determined.

In addition to the various approaches designed to facilitate health behaviour change, there has also been a debate over the efficacy of single versus multiple intervention strategies, that is, facilitating lifestyle change by targeting one behaviour at a time or addressing multiple risky behaviours simultaneously. There is a body of evidence that confirms the effectiveness of single-intervention strategies in health behaviour change (Aittasalo et al., 2006; Ackermann et al., 2005; Calfas et al., 1996; Green et al., 2002; Hudon et al., 2008; Kerse et al., 2005). The health behaviour theories are based upon changing a single behaviour at a time, and provide little advice on changing multiple behaviours or the process of multiple behaviour change (Noar et al., 2008).
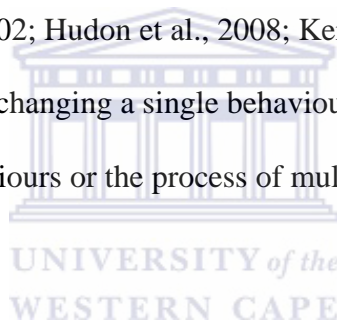
Evidence from several epidemiological and randomized, controlled studies provide strong support for the reduction in multiple risk factors of CHD using multiple intervention strategies (Artinian et al., 2010; Ebrahim and Smith, 1997; Ebrahim et al., 2011; Eriksson et al., 2009; Haskell et al., 1994; Ornish et al., 1998; Werch et al., 2007). Effectiveness in reducing CHD incidence appears to be associated with the degree of risk factor control achieved (Ebrahim et al., 2011). Evidence from quasi-experimental studies, such as the North Karelia project (Puska 1976; Puska 1981) and the Stanford Heart Disease Prevention Programme (Farquhar 1977; Farquhar 1990; Fortmann 1993), indicate that multiple risk factor interventions using counseling and educational methods are both efficacious and cost-effective and should be expanded.

The Lifestyle Heart Trial was the first randomized trial to show that comprehensive lifestyle changes in coronary patients lead to the regression of atherosclerosis (Ornish et al., 1998). It was concluded that comprehensive lifestyle changes can bring about substantial regression of severe coronary atherosclerosis after only one year without the use of lipid-lowering drugs (Ornish et al., 1998). Recent data support the regression in atherosclerosis, even after five years, but only in patients who continue to adhere to the lifestyle changes (Pischke et al., 2008).

Other studies investigated various types of diets, from lipid lowering and low-fat to vegetarian. All these studies reported improvements on CVD status (Sebregts et al, 2000). The improvements were reflected in clinically significant decreases in plasma cholesterol levels, accompanied by significant reductions in LDL-cholesterol and plasma triglycerides, and increased HDL-cholesterol, and slower rates of progression of coronary artery stenoses (Sebregts et al, 2000). These findings support the role of appropriate diets in treating CHD risk factors, even when serum cholesterol concentrations are moderately elevated (Sebregts et al, 2000).

Interventional studies on CHD risk factors expanded and included varied protocols, such as diet, exercise and behaviour therapy, either in isolation or combination. Again, the results showed consistent changes in risk reduction, especially for the combined therapies (McArdle et al., 2001, p. 905). These findings demonstrate that a supervised programme of diet, exercise and behaviour modification is effective in reducing CHD risk factors, especially when the behaviours are combined.

Studies using structured intervention protocols also showed improvements in physical fitness, body mass index, body composition, self-image and personnel well-being (Eriksson et al., 2009; Pillay, 2005; Smith et al., 1997). In addition, physical activity promoted stabilization of body weight at relatively lower than normal levels (Eriksson et al., 2009). The resulting increase physical fitness helped to improve body image, and negated the tendency towards creeping obesity with age (Eriksson et al., 2009).

However, studies dealing with smoking cessation show that exercise training, in isolation, is not sufficient to cause sustainable change (Ebrahim et al., 2011). The appropriate behaviour therapy recommended is incorporating cognitive strategies with behavioural techniques on smoking cessation in order to achieve positive results (Sebregts et al, 2000; Ebrahim et al., 2011).

Behavioural counseling and health education are highly recommended as intervention strategies to stop cigarette smoking, as well as making healthy food choices, losing weight and increasing physical activity (Fernandez et al., 2007). Such strategies of behaviour change, however, are not always easy to accomplish (Ebrahim and Smith, 1997). The effectiveness of the intervention in reducing disease burden, in particular, seemed to be associated with the degree of control achieved by the participants. Not many of the studies were randomized or lasted long enough to permit conclusions to be drawn about the methodology or the effects (Nilsson et al., 2001).In addition, the relative contribution of each component of the lifestyle intervention programme was absent or vague, so that the specific health impact remained obscure (Sebregts et al, 2000).

Nevertheless, even though lifestyle counseling is effective in lifestyle management programmes (Lie, 2007; Saelens et al., 2000), there still exists a need to investigate more thoroughly the use of self-management strategies before adopting them for behaviour modification and lifestyle change (Saelens et al., 2000; Burke et al., 2005). While men appeared to benefit equally from counseling and non-counseling interventions, counseling-based interventions were more effective for women than men (Writing Group for the Activity Counseling Trial [ACT] Research Group, 2001).

In university students, using multiple interventions that ranged from positive goal-imaging (of fitness) to a contractual agreement and/or a consultation strategy, Werch et al. (2007) found these methods positively influenced health behaviours. However, no indication was given about whether the positive changes in health habits resulted from the content of the programme, the frequency of contact, or the compliance monitoring techniques utilized in the study (Werch et al., 2007).

Results of these studies have been variable and have cast some doubt on the effectiveness of multiple risk factor interventions on changing health behaviours and CHD risk (Artinian et al., 2010; Ebrahim and Smith, 1997; Rosal et al., 2004; Rosenberg et al., 2007; Steptoe et al., 2002). Many of the studies aimed at changing HRB and lifestyle, have generally applied the same type of intervention strategy, for example, provision of health information, individual counseling and close monitoring (Artinian et al., 2010). There is a limited body of knowledge on the relative benefits of simultaneous versus sequential delivery of multiple behaviour change interventions in

adults (Ebrahim et al, 2011). The results for the superiority of sequential versus simultaneous intervention strategies are mixed (Artinian et al., 2010; Ebrahim et al., 2011).

A further concern expressed with many of the studies using multiple interventions is the fact that the interventions were not standardized but varied between sites and over time (Ebrahim et al., 2011). Moreover, validation of smoking status using biochemical assay of thiocyanate was only used in one trial, (Ebrahim and Smith, 1997). Subject drop-out and losses at follow-up were a particular problem and changes in risk factors could not be assessed on an intention-to-treat basis (Rosenberg et al., 2007). Furthermore, follow up of participants over the long term showed no consistent pattern in behaviour change between the control and intervention groups, and this indicated that the initial behaviour modification was only transient and not sustainable over time (Rosal et al., 2004). The interventions consisted of different components and it was therefore difficult to determine which interventions accounted for the favorable effects (Ebrahim and Smith, 1997). Recent research in CHD risk factors show the changes associated with multiple risk factor interventions to be of a more modest nature (Fleming and Godwin, 2008; Ebrahim et al., 2011). In addition, the interventions were more effective in populations with particularly adverse risk factor profiles, in other words, subjects most at risk also benefitted the most from the interventions (Ebrahim et al., 2011).

In summary, although there is some evidence of the benefit of multiple risk factor intervention programmes, more research is needed to determine which interventions are most effective and in which combinations (Eriksson 2006 and 2009; Ebrahim et al., 2011; Ebrahim and Smith, 1997). Having reviewed the literature on how multiple risk factor interventions in youth can change

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unhealthy lifestyle behaviours, the present study was undertaken to investigate the effects of multiple health interventions on CHD risk.
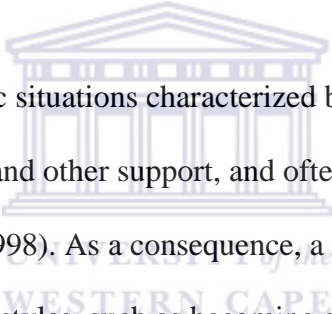
2.7 Intervention Studies Related to CHD, Physical Fitness and Health-Risk Behaviours in University Students

Few randomized intervention studies have been published on CHD risk at the tertiary level (Ebrahim and Smith, 1997; Eriksson et al., 2006 and 2009; Fernandez et al., 2007), and none on the African continent (Ebrahim et al., 2011). Most of the studies have been primarily epidemiological, observational ones with mainly a cross-sectional rather than longitudinal focus, and the dirth of information is evident in tertiary institutions (Frost, 1992; Werch et al., 2007). Some of the areas investigated in studies with university students were health status (Steyn et al., 2000), HRB (Borkon et la., 1983; Ojikutu and Adeleke, 2010) and the level of health risk awareness of students (Adelekan et al., 1992; Steptoe and Waddle, 2000; Romero et al., 2005; Tamragouri et al., 1986).

Previous studies indicate that educational interventions aimed at increasing public awareness about heart disease fail to reach various population groups equally (Collins et al., 2004; Steyl, 2008). A five-year evaluation of a community-wide cardiovascular disease intervention programme demonstrates greater behavioural changes and knowledge in white respondents when compared to black respondents. This phenomenon was also observed in other studies (Collins et al., 2004; Frost, 1992; Tamragouri et al., 1986). Women are less informed about CHD than men, and often fail to identify heart disease as their greatest cause of death or their most important

health concern (Romero et al., 2005; Steyl, 2008; Tamragouri et al., 1986). Many of them continue to believe that breast cancer is a more significant health concern (Steyl, 2008).

Students in most population groups believe that whites are most at risk for developing heart disease (Steyl, 2008). Despite a significant number of Indian and white university students being aware of a family history of CHD, many do not perceive themselves as a population at-risk (Steyl, 2008). This perceived lack of vulnerability translates into an unwillingness to participate in risk-reducing behaviours or lifestyle modification. Consequently, many of them continue to engage in social activities that place them at risk (Steyl, 2008).

Students increasingly face academic situations characterized by high demands, disempowered decision-making, low institutional and other support, and often long hours studying or completing tasks (Hedberg et al., 1998). As a consequence, a trend is developing in which more students are adopting unhealthy lifestyles, such as becoming sedentary, smoking, and overeating in their leisure time (Hedberg et al., 1998; Ojikutu and Adeleke, 2010; Peltzer, 2010; Steptoe et al., 1997; Steptoe and Waddle, 2001).

Many students are aware of the link between smoking and lung cancer but their knowledge of the link between smoking and heart disease is very low (Peltzer, 2001). Most are unable to identify smoking as the single biggest risk for heart disease (Collins et al., 2004). Interventions to control smoking produce poor and disappointing results, with short term improvements in smoking cessation completely lost after a few years (Rosal et al, 2004; Peltzer, 2001). This phenomenon is

disconcerting given that smoking prevalence is on the increase amongst university students (Peltzer 2010) and is the most preventable cause of CHD.

There is a strong association between the socioeconomic background of students and substance use, with religiosity reported as a determinant (Peltzer, 2001 and 2010). Students from low income families are more likely to consume alcohol and smoke, especially young males (Ojikutu and Adeleke, 2010; Peltzer 2002 and 2010). Fewer female students smoke marijuana (Peltzer et al., 2002) but those who do are more likely to drink alcohol (Adelekan et al., 1992). Older black females($\geq$ 24 years) are more likely to be overweight and obese, to smoke heavily and consume alcohol excessively and display significant increases in waist circumference and blood pressure, They are also prone to be symptomatic of anaemia with moderate to severe depression (Steyn et al., 2000). Amongst black women, a thin figure is associated with a person infected with HIV, or who has AIDS (Matoti-Mvalo & Puoane, 2011). Therefore, many prefer to be overweight and at risk for CHD, than thin and stigmatized (Matoti-Mvalo and Puoane, 2011).

These results suggest that educational intervention is necessary to increase the knowledge about CHD, especially among women and educationally disadvantaged groups (Collins et al., 2004; Frost, 1992; Matoti-Mvalo and Puoane, 2011; Tamragouri et al., 1986). This population has frequent access to the media, with 80% listening to the radio every day and 88% watching television at least once a week (Steyn et al., 1991). Therefore, health and lifestyle information could be promoted quite successfully by means of these media. Moreover, future educational interventions should address common misconceptions about which demographic sectors of society are at risk (Matoti-Mvalo and Puoane, 2011). These differences in heart health

knowledge between genders and various population groups begin early in life, and require

culturally relevant health information in already childhood (Jones et al., 2007). This further

supports the wisdom of primary prevention of CHD through early risk identification and

appropriate intervention in childhood or adolescence (Kemper et al., 2002).

2.8   Brief Profile on the University of the Western Cape

UWC focuses its attention on addressing social and political issues and on empowering its

graduates to be aware and accountable to the communities from which they derive their roots.

The student racial demographics typically represent a "rainbow nation." that is multi-ethnic,

multicultural and multilingual in its diversity.
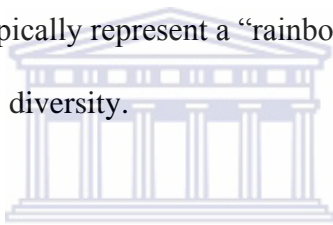
Research conducted by the Human Sciences Research Council (HSRC) on historically black

institutions(HBI) and white institutions (HWI) helps to provide insight into the lives of students

at HBIs (Breier, 2007, p. 3). Reporting on why students drop out of higher education, the

research found that many students, despite their promising academic performance, leave because

their financially impoverished families cannot afford to keep them there (Breier, 2007, p. 40;).

Many of them live under difficult circumstances, with dysfunctional single parent families and

absent fathers, without basic accommodation and adequate food (Holborn and Eddy, 2011). They

are also expected to top up meagre study loans or bursary grants in order to remain at university.

Inevitably, the burden becomes too heavy, so many of them end up leaving university to find

work in order to support families and repay loans. But, many still intend returning in future,

when financially stable, to complete their studies.

From the seven South African tertiary institutions that participated in the study, two were particularly noticeable for students who drop out before graduating, namely, Fort Hare and the University of the Western Cape (UWC) (Breier, 2007, p. 40). The tertiary institution in SA that was categorized as having the highest percentage (75%) of its graduates coming from a background of low socio-economic status, is UWC (Breier, 2007, 78).

In many ways, the institutional difficulties at UWC are associated with limited resources with its concomitant restricted access. It is within this socio-economic context that the present study is conducted. The study therefore aims to address some of the health and wellness issues confronting the students on this campus and to provide a strategy to address them holistically.
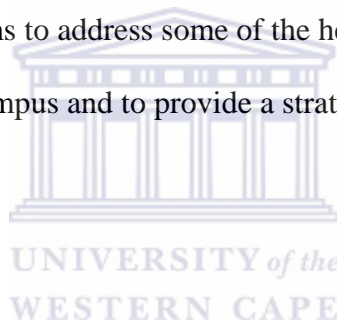
2.9   Summary

CHD is unquestionably a major public health problem affecting all ages. The poor and indigent are especially vulnerable. Studies on CHD risk factor reduction using multiple intervention strategies has been reported but the improvements have been modest.

University students, particularly in SA, are most aware of STDs, HIV/AIDS and psychological disorders, probably because these health behaviours are their immediate concerns and affect them disproportionately. Thus, students either seek out information about these problems or get reinforcement from the public broadcasting media.

The public at large and university students, in particular, lack essential knowledge of the major risk factors that contribute to the development of CHD. This makes health education a priority and heralds the call for professional guidelines on CHD prevention that is applicable to all ages.

The extent of the data on CHD risk factors and HRB is matched by the paucity of data on HRPF of South Africans, especially university students. Consequently, there is a need for basic research to address this shortcoming.
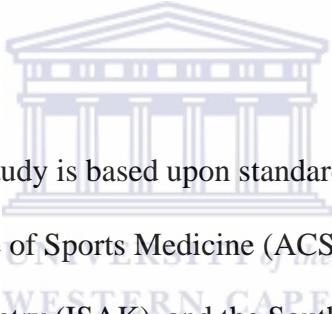
Against this background, this study was undertaken, and a multiple risk factor intervention strategy selected as the preferred mode of treatment to address the aim of the study.

Chapter Three: Research Methods

3.1   Introduction

This chapter discusses how the study was conducted, commencing with the recruitment of first year university students, followed by the identification of subjects at moderate CHD risk, leading to the randomization of subjects into different treatment groups, followed by the implementation of various health behaviour interventions and culminating in the evaluation of its impact on CHD risk, HRPF and HRB. The study design is illustrated by Figure 3.1 This design will be discussed in detail below.

The methodology followed in the study is based upon standardized testing procedures as advocated by the American College of Sports Medicine (ACSM), the International Society for the Advancement of Kinanthropometry (ISAK), and the South African Medical Research Council (SAMRC).

3.2   Study Design

A quantitative, experimentally-controlled design was used through which a selected sample of university students identified as being moderately at risk of CHD underwent multiple health behaviour interventions for a period of 16 weeks. The impact of the various health interventions on CHD risk factors, HRPF, and HRB was then assessed.

Phase 1: Subject recruitment
Step 1: Liaison with staff of the Centre for Student Support Services (CSSS)
Step 2: Liaison with peer facilitators/orientation leaders
Step 3: Liaison with first year students
Step 4: Dissemination of information to first year students
Step 5: Distribution of research posters
Step 6: Distribution of information letters

Phase 2: Subject selection, screening and preparation
Step 1: Obtaining informed consent
Step 2: Assessment of physical activity readiness and stage of readiness to change
Step 3: Administration of pre-test instructions
Step 4: Scheduling of subjects for pre-test (baseline) measurements

Phase 3: Pre-test (Baseline) Measurements
Step 1: CHD risk factors
Step 2: Risk stratification
Step 3: Health-related physical fitness
Step 4: Health-risk behaviours
Step 5: Diet
Step 6: Physical Activity

Phase 4: Randomization of subjects into treatment groups

P E R I O D S

P R E T E S T

I N T E R V E N T I O N

P O S T T E S T

Control Group | Health Information | Diet Group | Exercise Group | Multiple Group

Phase 5: 16 week intervention period

Control Group | Health Information | Diet Group | Exercise Group | Multiple Group

Stage 6: Post-test (follow-up) Measurements: repetition of phase 3

Figure 3.1. Flowchart of the study design.

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Subjects who were selected to participate in the study were randomized into five treatment groups, four experimental and one control group (CG). These groups were tested before and after a 16 week multifactorial intervention programme. The treatment groups comprised a health information group (HIG), a diet group (DG), an exercise group (EG) and a multiple group (MG) that included all three treatments.

3.3.   Study Population and Sample

The study population comprised of first year, full-time students entering UWC for the first time. The population was confined to these students so that neither prolonged exposure to university life nor year of study were influencing factors. The target population was determined by screening students for CHD risk factors. Abscondments following the pre-test and the intervention phase reduced the target population. The sample size for the study was determined in consultation with the Department of Statistics at UWC to ensure the statistical power of the study. The final sample consisted of subjects stratified as being moderately at risk for CHD.

Racial classification in SA is based upon four officially designated population groups according to the population registry, namely, black (historically of African ethnic ancestry) coloured (historically of mixed ethnic ancestry) Indian (historically of oriental ancestry), and white (historically of Caucasian ancestry). Thus, the subjects in the study were also racially classified in accordance with the population registry.

3.4   Pre-Test (Baseline) Period

3.4.1 Phase 1: Subject Recruitment

The period of subject recruitment and data collection extended over two years, from January

2008 to December 2009. Gaining access to the first year students was done through the Centre

for Student Support Services (CSSS) at UWC. Active methods were used during the university

orientation programme to recruit students. This was done through announcements at student

faculty forums, mounting posters and distributing flyers throughout the campus and sending out

information about the study through the university's student email. In addition, information

desks were set up in the university student centre and the library for the dissemination of study

information. The peer facilitators, responsible for the first year students during the orientation

period, were informed about the study and encouraged to inform the students..

The students who were interested in participating in the study were first informed verbally about

the nature and scope of the study. Each received an information letter (Appendix A) and a

consent form (Appendix B). Participation in the study was voluntary and students were informed

that they could withdraw at any stage.

3.4.2 Phase 2: Subject Selection, Preparation and Screening

The subjects selected for the study where only those who granted their written consent. As a

safety precaution, the subjects were initially screened by a physical activity readiness

questionnaire (PAR-Q) (Appendix C). This determined whether the subjects' had any sign or symptom suggesting underlying cardiovascular dysfunction and the likelihood of sustaining a heart attack during exercise testing and/or training. Subjects found to be at risk on completion of the PAR-Q were referred to their family physician or campus doctor for medical clearance.

Once declared suitable, subjects received pretest instructions (Appendix D), at least 24 hours prior to being tested, informing them about the mandatory preparatory requirements for the various batteries of tests – clinical and physical. Testing was scheduled over two or more days in order to keep subjects' levels of motivation high and ensure optimal performance.

3.4.3 Phase 3: Pre-Test (Baseline) Measurements
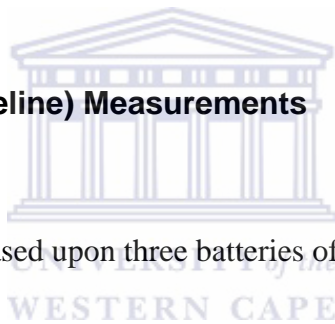
The pre-test measurements were based upon three batteries of assessments, namely:

- CHD risk factors: These measurements consisted of seven risk factors, namely, a family history of heart disease, cigarette smoking, obesity, hypertension, dyslipidemia, impaired fasting glucose, and a sedentary lifestyle. Age, as a risk factor, was one of the exclusion criteria of the study;

- HRPF tests: These comprised of five components, namely, body composition, cardiorespiratory fitness, muscular strength, muscular endurance, and flexibility.

- HRB: This assessment comprised of a healthy lifestyle questionnaire that contained 37 questions based upon the following HRB, namely, physical activity, nutrition, managing stress, avoiding destructive habits, practicing safe sex, adopting safety habits, knowing first aid, personal health habits, using medical advice, being an informed customer, protecting the environment and mental well-being.

3.4.3.1   CHD Risk

The present study measured only the major risk factors of CHD that are reported to account for almost 90% of CHD risk (Nainggolan, 2011).

The clinical measurements taken were the following; resting heart rate, resting systolic and diastolic blood pressures, lipoprotein profile, impaired fasting glucose and physical measurement to determine obesity. All measurements adhered to ACSM (American College of Sports Medicine, 2006a, pp. 39-92), ISAK and SAMRC guidelines.

3.4.3.1.1   Family History, Cigarette Smoking and a Sedentary Lifestyle

The CHD risk assessment form (Appendix E) was used to record the family history of premature CHD, cigarette smoking, and physical activity habits. Additional personal information, such as, gender, marital status, race/ethnicity, previous and current physical injury status, and prescribed medication was recorded on the physical fitness sheet (Appendix F).

3.4.3.1.2   Obesity

Obesity as a CHD risk factor was defined as a body mass index > 30 kg·m^{-2} in both men and women or waist hip ratio ≥ 0.95 and ≥ 0.86 in men and women, respectively, or a waist girth $>$ 102 cm and > 88 cm in men and women, respectively. All measurements were recorded on the CHD risk assessment form.

The anthropometric or physical measurements used to describe obesity are explained below.

3.4.3.1.2.1   Anthropometry (Physical Characteristics)

The anthropometric measurements taken consisted of the following, namely, body mass (weight), stature (height), subcutaneous skinfolds and waist and hip circumferences according to ISAK guidelines (Marfell-Jones et al., 2006, pp 5-88). Subjects were informed in the pre-test instructions about what garments to wear and the duration of testing. Subjects were offered a tester of the same sex, and to be accompanied by a friend or relative, if preferred.

The assessment rooms of the biokinetics clinic were used for taking the anthropometric measurements at a comfortable room temperature and in a quiet atmosphere, with privacy observed at all times. Preference was given for taking measurements in the mornings to minimize bias due to diurnal variations in body mass. The time was recorded for all pre-test measurements and the post-test measurements were repeated at the same time ($\pm$ 1 hour).

3.4.3.1.2.2   Body Mass (Weight)

Subjects were weighed wearing light (minimal) indoor clothing, that is, males in shorts only and females in shorts and a light T-shirt or swim suit. Body weight was measured to the nearest 50g using a beam balance scale (Seca model 700, Gmbh & Co., Germany) with a measurement range from 0 to 220 kg calibrated against standard low and high weights.

The scale was first checked and zeroed. Each subject stood on the centre of the scale, facing away from the balance beam, without support and with their weight evenly distributed across both feet. The tester determined the appropriate weight of the subject using the sliding weights on the beam-balance, and reported it to the recorder to enter onto the data sheet.

The average of two measurements was used as the final measurement, provided that the measurements were within 0.1 kg of each other. If not, additional measurements were taken until the appropriate limits were obtained.

3.4.3.1.2.3   Stature (Height)

Stature (height) was measured without shoes to the nearest 0.1 cm using a stadiometer (Seca model 700, Gmbh & Co., Germany) with a measurement range from 60 to 200 cm and calibrated against an anthropometer (Holtain, UK).

The stretch stature was measured with subjects standing barefoot on a level wooden floor with the heels together and the heels, buttocks and upper back touching the scale. The subject's head was placed in the Frankfort plane, in other words, the tester placed the tips of the thumbs on each orbitale (lower edge of the eye socket) and the tips of the index fingers on each tragion (the notch superior to the tragus of the ear), then horizontally aligned the two. The tester then relocated the thumbs posteriorly towards the subject's ears, and far enough along the line of the jaw to ensure that upward pressure, when applied, is transferred through the mastoid process. The subject was then instructed to inhale deeply and, while keeping the head in the Frankfort plane, the tester applied gentle upward lift through the mastoid process. Next, the recorder placed the sliding head-board firmly down on the vertex (highest point of the skull), compressing the hair as much as possible, but not affecting the stretch height. The recorder finally checked that the subject's heels did not leave the floor, before taking the measurement and before the subject exhaled.
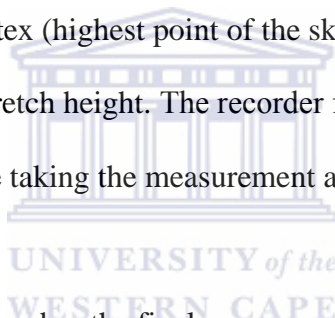
The average of two measures was used as the final measurement provided that the measures were within 5 mm of each other. If it was not, the standard protocol was observed.

3.4.3.1.2.4   Body Mass Index

Body mass index (BMI), or Quetelet index, is used to assess weight relative to height, and was calculated from body mass in kilograms (kg) divided by stature in metres squared (m^{-2}) and was expressed as $kg \cdot m^{-2}$. This value is considered a good indicator of general body composition in population-based studies, and is related to health outcomes (The Emerging Risk Factor

Collaboration, 2011). Significant CHD risk begins at a BMI of 30.0 kg·m^{-2} in both males and females (ACSM, 2006a, p. 58) and is associated with excess body fat.

3.4.3.1.2.5   Waist and Hip Circumferences

A non-extensible, flexible metal tape (Sanny Medical, HK) with a measurement range from 0 to 2 m was used to measure waist and hip circumferences to the nearest 0.3 cm.

The cross-hand technique was used for measuring all girths and the reading was taken from the steel tape measure held at right angles to the body segment where the zero was located more lateral than medial on the subject. In order to position the tape measure correctly, the case was held in the right hand and the stub in the left hand. The stub was passed around and to the back of the body segment and grasped in the right hand, while facing the body segment, thereby freeing the left hand. The left hand was used to manipulate the tape to the correct level and then passed underneath the casing to grasp the stub again. The middle fingers of both hands were used to locate the tape at the precise landmark for measurement and to orientate the tape so that the zero was easily read. The objective was to minimize gaps between the tape and the skin and indentations of the skin wherever possible. The juxtaposition of the segments of the tape ensures that there is contiguity of the two parts from which girth is determined. When taking the reading, the tester's eyes were positioned at the same level as the tape to avoid the error of parallax.

The waist was measured at the level of the umbilicus, in other words, the narrowest part of the abdomen between the lower costal (10th rib) border and top of the iliac crest, perpendicular to the long axis of the trunk.

The hip measurement was taken at the level of the greatest posterior protuberance of the buttocks that usually corresponds anteriorly to about the level of the symphysis pubis, that is, at the widest part of the hips.

When measuring the waist, the tester stood in front of the subject who abducted the arms slightly to allow the tape to pass freely around the abdomen. The measurement was taken at the end of a normal expiration (end-tidal measurement).

Similarly, when measuring the hips, the tester stood in front of the subject who folded the arms across the chest and stood feet together with gluteal muscles relaxed. The tape was passed around the hips and held in a horizontal plane at the target level before the measurement was taken.

The average of two measures was used as the final measurement, provided that the measures were within 5 mm of each other. If not, then the standard protocol was observed.

3.4.3.1.2.6 Waist-Hip Ratio

Waist hip ratio is a simple measure calculated by dividing the waist circumference (cm) by the hip circumference (cm) and is considered an indicator of body fat distribution (central or android versus peripheral or gynocoidal fat patterning). Ratios above 0.95 and 0.86 in men and women respectively, are considered indicative of obesity and significantly increased CHD risk (ACSM, 2006a, p. 59). Because of its association with abdominal obesity, waist circumference alone is also used as an indicator of significant CHD risk when recorded above 102 cm and 88 cm in men and women, respectively (ACSM, 2006a, p. 59).

3.4.3.1.3 Hypertension

Hypertension as a CHD risk factor was defined as a subject on anti-hypertensive medication or systolic blood pressure equal to or above 140 mm Hg and/or diastolic blood pressure equal to or above 90 mm Hg, confirmed by three measurements on two separate occasions.

Subjects presented themselves at the biokinetics clinic and were taken to one of the assessment rooms for BP measurement. Before being tested, the subjects had to confirm that the pre-test instructions were observed. If not, the test was rescheduled for another day.

Resting heart rate (RHR) and blood pressure (SBP and DBP) were measured indirectly. Three measurements were taken (minimum of one minute apart) with a pressure cuff of appropriate size (12 x 35 cm^2) placed around the left upper arm, while using a standard mercury sphygmomanometer (Goodpro International Co., Limited, China), an acoustic Sprague

Rappaport stethoscope (Medical Supplies and Equipment Company, Houston, Texas, USA). The standard auscultatory method of blood pressure measurement was observed.

In preparation for testing, each subject sat quietly for at least 5 minutes, breathing spontaneously and without talking. With the cuff fully deflated, the mercury meniscus was checked for a zero rating. The cuff was quickly inflated to 20-30 mm Hg above the first Korotkoff sound and then the pressure was slowly released at a rate of 2-3 mm Hg per second. Systolic blood pressure (SBP) was noted at the first appearance of the Korotkoff sounds (phase 1) and diastolic blood pressure (DBP) at the disappearance of the sounds (phase 5). All values were corrected to the nearest 2 mm Hg. The first BP reading was discarded and the mean of the second and third measurements was used as the final BP measurement.
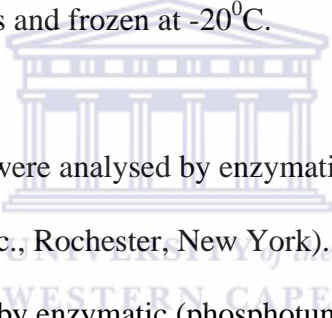
3.4.3.1.4   Dyslipidemia and Impaired Fasting Glucose

Dyslipidemia as a CHD risk factor was defined as low-density lipoprotein (LDL) cholesterol concentration > 3.4 mmol$^.$L^{-1} (1.03 mg$^.$dL^{-1}) or high-density lipoprotein (HDL) cholesterol concentration > 1.03 mmol$^.$L^{-1} (40 mg$^.$dL^{-1}) or total cholesterol (TC) concentration > 5.2 mmol$^.$L^{-1} (200 mg$^.$dL^{-1}) or subjects currently using cholesterol-lowering medication. A lipid profile of the individual lipoprotein fragments was done using invasive procedures and automated enzymatic precipitation methods.

Impaired fasting glucose as a CHD risk factor was defined as post-prandial (fasting) blood glucose concentration level > 5.6 mmol$^.$L^{-1} (100 mg$^.$dL^{-1}). Invasive procedures were also used for glucose determination.

The blood draw was performed at the campus health clinic on weekday mornings between 07h30 and 08h30. Subjects were required to confirm that they had observed the pre-test instructions before proceeding with the blood draw, failing which, the test was re-scheduled.

Separate vacutainer tubes (Becton, Dickinson and Company), one used to collect a blood sample for the lipid profile and another for the glucose concentration, were labeled in advance with the subject's identification code, date and time of the blood draw. A medical doctor drew the blood samples using standard phlebotomy procedures. A blood sample of approximately 10 ml of venous blood was drawn from the antecubital vein after an overnight fast of at least 10 hours. Serum was separated within 2 hours and frozen at -20^0C.

Total cholesterol and triglycerides were analysed by enzymatic colorimetry (slide method, Vitros 250, Ortho-Clinical Diagnostics, Inc., Rochester, New York). High density lipoprotein cholesterol (HDL-C) was analysed by enzymatic (phosphotungstic acid and magnesium chloride) colorimetry (slide method, Vitros 250, Ortho-Clinical Diagnostics, Inc., Rochester, New York). Low-density lipoprotein (LDL) cholesterol was calculated using the Friedewald equation. Serum glucose was analysed by enzymatic (glucose oxidase) colorimetry (slide method, Vitros 250, Ortho-Clinical Diagnostics, Inc., Rochester, New York). All biochemical analyses, instrument calibration and quality control were performed at the Biomedical Science Laboratory at the Cape Peninsula University of Technology (CPUT). The various ratios, namely, TC:HDL, LDL:HDL, and Trig:HDL was calculated manually.

The Vitros 250 Analyzer and biochemical reagents used were supplied by the South African Scientific Group (Cape Town, SA).

3.4.3.2 Subject Risk Stratification

Risk stratification was determined according to the guidelines of the American College of Sports Medicine (ACSM, 2006a, p. 27) as follows:

- *Low risk stratification*: asymptomatic subjects presenting with only one CAD risk factor.

- *Moderate risk stratification*: asymptomatic subjects presenting with two or more CAD risk factors, but without clinical signs or symptoms of disease.

- *High risk stratification*: subjects presenting with clinical (overt) signs and symptoms indicative of cardiovascular, respiratory, and/or metabolic disease.

A total of 610 subjects were screened for CHD risk. A total of 437 subjects were found to be at low risk and excluded, while 173 (28.4%) subjects were found to be at moderate CHD risk, and were retained for the HRPF and HRB assessments.

3.4.3.3 Health-Related Physical Fitness

HRPF was assessed according to the following five components, namely, body composition, cardiorespiratory fitness, muscular strength, muscular endurance, and flexibility. The rating of

the levels of physical fitness of the subjects was done in accordance with the guidelines reported by ACSM (ACSM, 2006a, pp. 55-92). For each of the components of HRPF an appropriate method and specific type of test were selected that best met the purposes of the study, as indicated in the table below.

Fitness Variable	Method	Test
Body composition	Anthropometry	Skinfold measurements
Cardiovascular fitness	Maximal aerobic capacity	Multistage shuttle run
Muscular strength	Isometric strength	Handgrip dynamometry
Muscular endurance	Isotonic strength	Sit-up test
Flexibility	Static stretch	Sit-and-reach test

All HRPF tests were conducted in the biokinetics clinic of the Sport, Recreation and Exercise Science (SRES) department at UWC. Prior to testing, explicit instructions were given to subjects about the order of testing, the recommended procedures for performing each test and the safety precautions to be observed.

The HRPF test order was as follows: 1) body composition, 2) cardiorespiratory fitness, 3) muscular strength, 4) muscular endurance, and 5) flexibility. Adequate rest was given between tests to prevent overuse of particular muscle groups and premature fatigue (negative systematic bias).

Prior to testing, a mandatory warm-up and subject familiarization with each test item was conducted to negate any learning effect (positive systematic bias).

The reasons for conducting the HRPF tests were the following:

- To collect baseline data;

- To stratify cardiovascular risk;

- To identify the physical fitness status of the subjects;

- To establish appropriate fitness goals for the subjects in order to keep those participating in the exercise intervention programme motivated and to facilitate exercise adherence; and

- To prescribe the appropriate training programme;

3.4.3.3.1   Body Composition

Body composition comprises the relative percentage of fat, bone, muscle and other tissues of the body (Corbin et al., 2006, p. 8).

Fat mass was determined by calculating the percentage body fat from the sum of three skinfold ($\sum$SF) measurements, namely, triceps, supra-iliac, thigh in women and thigh, chest, and abdomen in men. The skinfolds were measured to the nearest 5% with a calibrated (Carlyon et al., 1996, pp. 97-118) skinfold caliper (Harpenden, UK), while adhering to the criteria established by the

International Society for the Advancement of Kinanthropometry (Marfell-Jones et al., 2006, pp. 63-75) as well as Norton and Olds (1996, pp. 369 – 410). The caliper had a constant closing compression of 10 $g \cdot mm^{-2}$ throughout the range of measurement.

Each skinfold site was carefully located by using anatomical landmarks that were identified with the thumb and index finger. The site directly over the landmark was marked as a small cross (+) using a washable fine-tipped felt pen for ease of repeating measurements and for minimizing location errors. Each mark was then re-checked to ensure there was no displacement of skin relative to the underlying bone. When landmarking with a tape measure, the tape was held at right angles to the limb axis and the mark made at the top edge of the tape. All landmarks were first identified in a fixed order, that is, triceps, supra-iliac, thigh in women, and thigh, chest, and abdomen in men, before any measurements were made.
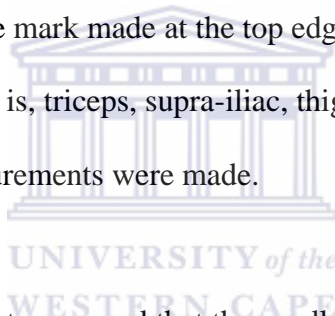
Before commencing the test, the tester ensured that the needle in the caliper dial was on zero. The skinfold was picked up so that a double fold of skin and the underlying subcutaneous adipose tissue was held between the thumb and index finger of the left hand, in line with the marked site. Thereafter, the skinfold caliper was applied 1 cm away from the thumb and finger at a depth of approximately mid-fingernail and held at 90º to the skinfold site at all times. Measurement was recorded 2 seconds after the full pressure of the caliper was applied, whilst still holding the skinfold in the left hand. This was required for the standardization of the test-retest measurements, whilst controlling skinfold compressibility. Measurements were done in a fixed, rotational rather than consecutive order, in other words, scores were recorded for all the skinfold sites in a fixed sequence before repeat measurements were taken. All measurements

were taken on the right side of the body in and in duplicate. If measurements were not within 5% (usually 1-2 mm), they were retaken.

The procedure for locating the various skinfold is as follows:

Triceps: this is a vertical skinfold taken on the posterior midline of the upper arm. The subject stood with the right arm relaxed and shoulder joint externally rotated to the mid-prone position and the elbow fully extended. First, the midpoint of the acromiale and radiale was measured perpendicularly to the long axis of the arm with a large sliding caliper (Holtain, UK) and marked on the lateral border of the arm with a felt pen. The triceps skinfold site was located by projecting mid-acromiale-radiale mark around to the back of the arm and intersecting the projected line with a vertical line in the middle of the arm when viewed from behind.

Supra-iliac: this is a slight diagonal skinfold taken immediately superior to the iliocristale, that is, a point on the iliac crest where a line from the mid-axilla (mid-armpit), on the longitudinal axis of the body, to the ilium. The subject stood relaxed with the right arm folded across the chest, while the tester stabilized the subject's pelvis on the left side with the right hand and palpated the top of the iliac crest with the left hand to locate the iliocristale site. The skinfold was then raised, running slightly downward anteriorly, and marked with a cross (+) in the centre.

Thigh: this is a vertical skinfold on the anterior midline of the thigh, midway between the proximal border of the patellare (knee cap) and inguinal point (hip). The subject sat with the

arms at the sides and the knee of the right leg bent 90º, while the tester landmarked the site using a large sliding caliper.

Chest/Pectoral: this is a diagonal skinfold midway between the anterior axillary line and the nipple. The subject stood with the right arm relaxed.

Abdomen: this is a vertical skinfold taken horizontally to the right of the omphalion (centre of the umbilicus) according to a fixed formula: 5 cm x height/170 cm. For example, if the subject was 155 cm, then the horizontal distance to the right of the omphalion was 5 x 155/170 = 4.5 cm, which worked out to between 4 and 5 cm for most subjects. This distance was calculated to avoid putting either the fingers or caliper inside the navel when measuring. The subject stood relaxed with the arms hanging at the sides, while the site was landmarked with particular care taken to isolate the underlying abdominal musculature, a part of the body that is usually poorly developed and, thereby, easily incorporated into the skinfold.

3.4.3.3.1.1   Percent Body Fat

Body composition when determined from skinfold measurements correlates well (r = 0.7 − 0.9) when compared with hydrodensitometry (ACSM, 2006a, p. 59).

The calculations for determining percent body fat were twofold, first body density was calculated (ACSM, 2006a, p. 63), and then percent body fat determined (ACSM, 2006a, p. 65) as follows:

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In women: using the three skinfold measurements, namely, triceps, supra-iliac, and thigh, body density was calculated as follows:

Body density = 1.099421 – 0.0009929 (sum of 3 skinfolds) + 0.0000023 (sum of 3 skinfolds)^2 – 0.0001392 (age)

In men: using the three skinfold measurements, namely, chest, abdomen, and thigh, body density was calculated as follows:

Body density = 1.10938 – 0.0008267 (sum of 3 skinfolds) + 0.0000016 (sum of 3 skinfolds)^2 – 0.0002574 (age)

Finally, to determine percent body fat, the appropriate gender- and population-specific formulae were applied (ACSM, 2006a, p. 65).

There are no universally accepted norms for body composition, as well as the exact percent body fat associated with health risk. However, health risk is thought to start at a body fat percentage of 22% in men and 32% in women (ACSM, 2006a, p. 66).

3.4.3.3.2   Cardiorespiratory Fitness

Cardiorespiratory or cardiovascular fitness, also called aerobic fitness or stamina, is defined as the ability of the cardiovascular and respiratory systems to supply fuel and oxygen to the tissues of the body, particularly the skeletal muscular system, both at rest and during various intensities

of exercise, and to efficiently remove the end products of cellular metabolism in order to avoid undue fatigue (Corbin et al., 2006, p. 8).

Cardiorespiratory fitness is expressed as maximal oxygen uptake relative to body weight ($\dot{V}O_{2max}$ $mL^{.}kg^{-1.}min^{-1}$). $\dot{V}O_{2max}$ was estimated indirectly as described by Leger (2007), while the subject ran on a level tarmac (cemented) surface following the verbal commands of the 20 m multistage shuttle run test on a computer diskette (CD) being played through an amplified sound system.

In the original test, Leger et al., (1988) reported a correlation of r = 0.78 between estimated and measured $\dot{V}O_{2max}$. Other authors have reported similar correlations within the range r = 0.69–0.95, with results varying by age and gender (Ortega et al., 2008a; Ramsbottom et al., 1988; Stickland et al., 2003).
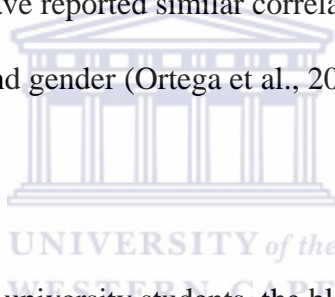
When performed on undergraduate university students, the bleep test was also proven reliable (Chatterjee et al., 2010; Chatterjee et al., 2008; Sproule et al., 1993), irrespective of gender or race (Ortega et al., 2008a), provided that they were not highly trained (Cooper et al., 2005) or sport-specifically trained (Gibson et al., 1998).

The protocol for the 20 m multi-stage fitness test consisted of shuttle running between two parallel lines set 20 m apart, running speed cues being indicated by signals emitted from a commercially available pre-recorded computer diskette (CD) (Sports Coach, The National Coaching Foundation, UK). The CD dictated that the subjects started running at an initial speed of 8.5 $km^{.}h^{-1}$ (2.36 $m^{.}s^{-1}$) with increments in running speed of 0.5 $km^{.}h^{-1}$ (0.14 $m^{.}s^{-1}$) each minute (1 minute equals one stage). The increase in running speed corresponds to a change in test level

that is designed to progressively tax the aerobic energy system, thereby, eliciting a maximal response, hence the link to maximal aerobic capacity. The speed of the diskette player was checked for accuracy in accordance with the manufacturer's instructions before each application.

Standardized instructions were given to participants in every test and all measurements were carried out under controlled conditions. All subjects performed a 10 minute warm-up that included prescribed jogging and stretching. The shuttle run test was conducted outdoors on a non-slip, surface. Subjects ran accompanied by an assistant (pace-setter), if preferred, in order to add an element of competition and to aid them in producing a maximal effort. Subjects were instructed to run in a straight line, to pivot (turn) on one leg on completing a shuttle, and to pace themselves in accordance with the audio signals. All subjects were verbally encouraged to perform maximally during each assessment, with the performances recorded on a standardized score sheet (Appendix G).
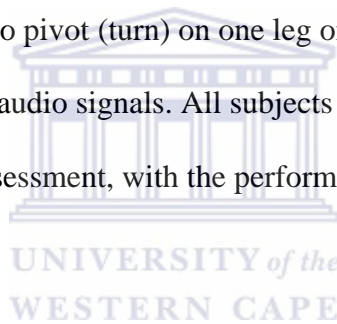
The test finished when the participant failed to reach the end lines concurrent with the audio signals on two consecutive shuttle runs, or, when the subject stopped due to volitional fatigue. After finishing the test, all subjects participated in a 5 minute cool-down session that involved walking and stretching. The test results for each subject were denoted as a predicted maximal aerobic capacity ($\dot{V}O_{2max}$) and expressed in millimetres per kilogram per minute ($mL^{-1}kg^{-1}min^{-1}$). This was obtained by cross-referencing the final level and shuttle number completed by the subject with that of the $\dot{V}O_{2max}$ reference table provided in the instruction booklet. All the tests were performed twice and the best score was retained as the final measurement.

3.4.3.3.3   Muscular Strength

Muscular strength is defined as the ability of the musculo-skeletal system to exert force over a singular muscular contraction of either an isometric (static) or dynamic (isotonic) nature (Corbin et al., 2006, p. 8).

For the handgrip test, a calibrated hand dynamometer (TKK 5401 Grip D; Takei Scientific Instruments, Co., Tokyo, Japan) with an adjustable grip was used, with measurements recorded to the nearest 0.1 kg. With the dynamometer set on zero and the subject standing with the arm held away from the body and elbow bent, the handgrip dynamometer was gradually and continuously squeezed with maximal effort for at least 3 s, performing the test with the right (dominant) and left (non-dominant) hands consecutively, using the optimal grip span. The handgrip span was adjusted according to hand size. The maximum score in kilograms for each hand was recorded. Subjects performed a minimum of two trials for each hand, with at least one minute's rest between trials. The best scores achieved for both hands were used as the final handgrip scores.

3.4.3.3.4   Muscular Endurance

Muscular endurance is the ability of the musculo-skeletal system to exert repetitive force of either an isotonic or isokinetic nature over a prolonged period of time without undue fatigue (Corbin et al., 2006, p. 8).

Muscular endurance of the abdominals and hip-flexors were assessed using the sit-up test. To

assume the starting position, subjects were in the supine position on a gymnasium mat, with the

feet on the floor, knees flexed 90°, and the hands placed on the opposite shoulders. By

contracting the abdominal and hip flexor muscles, the subject curled up to the sitting position.

The feet were held in touch with the floor by research assistants who also timed the performance.

Hand contact with the shoulders was maintained throughout and the chin was kept tucked into

the chest. The sit-ups were completed when the elbows touched the upper leg with the subject

returning to the down position where the lower back made contact with the floor. Only the

number of correctly executed sit-ups performed in 60 seconds was recorded, with the best score

of a minimum of two tests used as the final test measurement.

3.4.3.3.5   Flexibility

Flexibility is the ability of the body or parts of the body to move through the full range of motion

without undue resistance or strain, and is affected by the constitution of the soft and hard tissues

that comprise the joint(s) (Corbin et al., 2006, p. 8).

For assessing lower back and hamstring flexibility, the sit-and-reach test (flexibility assessment)

was performed against a standard reach box 70.1 cm long by 30.5 cm high and 30.5 cm wide

with a calibrated measuring board, and 23 cm marked at the level of the feet against the

stopboard. The subject sat barefoot on the floor, bent forward at the waist, while keeping the

knees straight and the feet against the flexometer foot stop. One hand was placed on top of the other

with fingertips aligned and without bouncing, the subject reached forward as far as possible on the

flexometer by slowly and concentrically contracting the hip flexors and abdominals. The final position was held with an isometric contraction for 2 seconds and the measurement was taken at the furthest point reached by the fingertips on the flexometer to the nearest centimeter (cm). The test was repeated a second time, and if the scores differed by more than 1 cm, it was repeated once again. The best score for the farthest distance reached was used as the final measurement.

3.4.3.4   Health-Risk Behaviours

For assessing the subjects' knowledge of HRB, the modified healthy lifestyle questionnaire (Appendix H) was used (Corbin et al., 2006, p. 15). The questionnaire included questions related to subjects' perceptions about their own health risks and about public health. The standardized self-administered questionnaire consisted of 37 close-ended questions grouped into twelve categories, namely: physical activity, nutrition, managing stress, avoiding destructive habits, practicing safe sex, adopting safety habits, knowing first aid, personal health habits, using medical advice, being an informed consumer, protecting the environment, and mental health and wellbeing.

The first four variables were based on four items or questions (also called 4-item scales), whereas the last eight variables had two items (2-item scales). The scores on the questionnaire were interpreted as follows: scores of 3 or 4 on the 4-item scales were indicative of generally positive lifestyles. For the 2-item scales, a score of 2 indicated the presence of positive lifestyles. An overall score of 26 or more was a good indicator of health lifestyle behaviours.

Caution, however, was advised when interpreting the scores on the HLQ, as explained below:

- Firstly, all lifestyle behaviours do not pose the same risk, for example, smoking cigarettes or abusing drugs has immediate deleterious physiological effects on health and wellness, whereas others, such as knowing first aid or being an informed consumer may only have occasional use;

- Secondly, focusing on the overall score can be particularly deceiving, for example, a subject may score well on one item in a scale, such as physical activity, but not on another, such as practicing safe sex; and

- Lastly, if a subject scores a 3 out of 4 on a 4-item scale, and the one item that the subject "fails" indicates an unhealthy lifestyle in an area that poses a serious health risk, such as failing to use a condom when having sex, the subject's lifestyle may appear to be healthier than it really is.
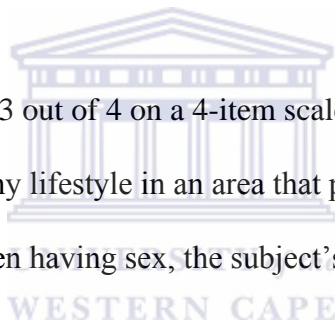
3.4.3.5   Dietary Intake

Dietary intake was assessed by means of three 24-hour recalls (Appendix I) on non-consecutive days, that is, two weekdays (Monday to Friday) and one weekend day (Saturday or Sunday). The dietary recording booklets were supplied by SAMRC. A dietitian explained to the subjects how to complete the 3-day food record. Food quantities and composition were determined using manuals supplied by SAMRC (1991a and 1991b). The dietary data was analyzed using the Foodfinder Version 3 Diet Analyzer software (SAMRC, 2000).

3.4.3.6   Physical Activity

The international physical activity questionnaire (IPAQ) (Appendix J) assessed physical activity undertaken across four domains, namely:

- leisure time physical activity;
- domestic and gardening (yard) activities;
- work-related physical activity; and
- transport-related physical activity.

The IPAQ enquired about specific types of activities undertaken within each of the four domains. The items were structured to provide separate domain specific scores for walking, moderate-intensity and vigorous-intensity activity within each of the work, transportation, domestic chores and gardening (yard) and leisure-time domains. Computation of the total scores required summation of the duration (in minutes) and frequency (days) for all the types of activities in all the domains.

In order to ensure accurate computation of the results, the recommended data cleaning procedures were followed (IPAQ Research Committee, 2005). Results were reported as a continuous measure. One measure of the volume of activity can be computed by allocating a weight to each type of activity based on its energy requirements, defined in METs to yield a score in MET·minutes. METs are multiples of the resting metabolic rate and a MET·minute is

computed by multiplying the MET score of an activity by the minutes performed. MET·minutes·week^{-1} was used as the preferred unit of measurement.
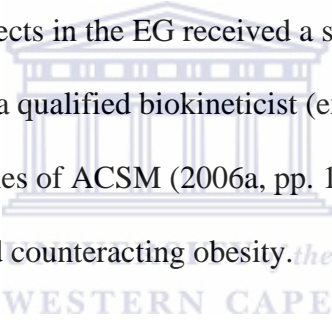
3.5 Intervention Period

3.5.1 Phase 4: Subject Randomization into Treatment Groups

A total of 173 subjects were identified as being moderately at risk for CHD and were randomly assigned to one of five intervention groups. A list of computer-generated random numbers was done by a statistician and the assignment of subjects to a random number was done as the subjects were stratified for CHD risk, in other words, the first subject stratified as moderate risk was allocated the first number, and the second moderate risk subject the second number, and so forth until all 173 subjects were completed. The initial number of subjects stratified as moderate risk totaled 182, so the allocation of random numbers was done for this larger group of subjects, and resulted in the groups being of relatively equal size. However, because nine subjects absconded after baseline testing, this affected the size of the groups.

The five treatment groups were described as follows:

- Control Group (CG): Subjects assigned to this group were asked to maintain their normal lifestyle for the duration of the intervention period;

- Health Information Group (HIG): Subjects in this group received health information designed to educate and empower them with life skills so that they adopt and/or maintain a healthy lifestyle.

- Diet Group (DG): Subjects allocated to this group received weekly dietary sessions facilitated by a dietician that focused on establishing a sound knowledge of nutrition and developing the basic skills to make healthy choices about the type and amount of food consumed daily.

- Exercise Group (EG): Subjects in the EG received a structured exercise programme designed by the researcher, a qualified biokineticist (exercise therapist). The programme was based upon the guidelines of ACSM (2006a, pp. 133-167) for developing cardiorespiratory fitness and counteracting obesity.

- Multiple Group (MG): Subjects in this group received all three treatments, that is, the health information, diet and exercise interventions.

3.5.2 Stages of Readiness to Change and Intervention Strategy

The content and structure of the intervention strategies applied in this study are based upon the Transtheoretical Model (TTM) or stages of readiness to change (Appendix K) developed by Prochaska et al. (1992). This model was used to explain why students succumbed to risk, as well

as how to strategize in order to deal with and overcome behavioural problems effectively (Appendix L).

The nature of the various health interventions was based upon the SMART acronym, that is, S = specific, M = measurable, A = attainable, R = realistic and T = time-bound (Griffin, 2006, p. 34). The benefits of such interventions is based upon the fact that explicit lifestyle change objectives provide practical targets for treatment and their attainment signifies effective risk factor modification and behaviour change.

In this regard, strategic lifestyle outcome targets (SLOT) were set for the various experimental groups to be attained on completion of the intervention period, without being too prescriptive on the proportion of change during the intervention. These targets were as follows:

HIG:

1. To achieve and maintain a desirable body weight;

2. To achieve and maintain a desirable level of HRPF;

3. To quit smoking;

DG:

1. To limit the daily intake of calories;

2. To restrict the intake of simple sugars, saturated fat and salt;

3. To increase the daily consumption of fish, fruits, vegetables, whole grain and fibre-rich products, and complex carbohydrates;

EG:

1. To improve the level of cardiovascular fitness (ACSM, 2006a: 79);

2. To perform moderate intensity physical activity at least four times per week or two or more sessions of vigorous activity per week (ACSM, 2006a: 22);

3. To lose body weight, specifically fat weight, while maintaining or improving lean body mass;

MG:

In this group, the subjects were encouraged to comply with the outcomes for each of the other experimental groups.

3.5.3 Phase 5: Implementation of Multiple Health Behaviour Interventions

All activities took place in the biokinetics clinic at UWC. Subject compliance was monitored either by an attendance register (manually) or via email (electronically). Subjects were also requested to report all physical activities engaged in outside of the intervention programme, such as sports training and competition, attending health and fitness centres and consuming weight loss drugs.

The various health interventions were managed as follows:

- CG: Following the baseline assessment, subjects who were randomized into the control group received standard feedback about their CHD risk factors, HRPF, and HRB.

Thereafter, they were instructed to maintain their normal daily routine, and not participate in any exercise or diet programme for the 16 week intervention period;

- HIG: The principle idea behind this intervention strategy was to provide students with health information and to observe whether they made use of it. Subjects were informed about various health concepts and lifestyle management skills. No reinforcement or professional behavioural therapy was provided, but lifestyle change was expected simply as a result of the subjects being exposed to regular information and through heightened awareness (Appendix M). E-communication, that is, computer-delivered e-mail contact was used to disseminate the health information. Subjects were considered to have adequate literacy skills to read e-based health-related materials, with sufficient access to computers at the various computer laboratories on campus. Through monitoring subjects accessing their emails, compliance to the intervention was recorded. However, the efficacy of the HIG strategy may have been compromised, at least in part, by a lack of sensitivity to e-literacy levels of the subjects and the absence of reinforcement in this group.

- DG: The purpose of this intervention was for subjects to acquire the appropriate knowledge about nutrition as well as nutritional life skills (Appendix N). Subjects received nutritional guidelines in accordance with the dietary recommendations of National Cholesterol Education Programme (National Institutes of Health, National Heart, Lung, and Blood Institute, 2001) and consensus documents of SAMRC (Bradshaw et al., 2003; Senekal et al., 2003) and WHO (Aboderin et al., 2002). Subjects were

encouraged to increase their intake of fish, fruits, vegetables, whole grain and fibre-rich products, complex carbohydrates, to restrict total caloric intake by reducing consumption of simple (processed) sugars and saturated fat and to use low-fat or fat-free milk products, soft margarines and vegetable oils rich in mono- or poly-unsaturated fatty acids. With their attendance recorded in a register for monitoring compliance, subjects met once a week in the Sport, Recreation and Exercise Science (SRES) department for nutritional activities that included skill-building sessions, such as reading food labels, weight loss tips, compiling a healthy grocery shopping list, preparing for grocery shopping, methods for healthy cooking, making healthy choices when eating out, and reducing exposure to unhealthy fast foods or high-calorie/low-nutrient foods. Self-monitoring tools were used to guide eating behaviour, such as graphs of weight change, healthy recipes, and fortnightly weigh-ins for celebrating the "biggest loser."

- EG: The exercise sessions took place in the biokinetics clinic at UWC and were conducted by the research assistants who were biokinetics interns with five years of training in exercise therapy. A structured exercise programme was prescribed in accordance with ACSM guidelines (Garber et al., 2011) that involved supervised aerobic endurance and/or circuit weight training for at least 20 minutes performed individually or in groups at least thrice weekly. Attendance was recorded in the attendance register. The sessions were moderate- intensity exercise performed at least thrice weekly for a minimum of 20 minutes. Opportunities for self-monitoring, enhancing self-efficacy, and relapse prevention, included fitness challenges conducted fortnightly to identify and acknowledge "Ms and Mr. Fitness."

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- MG: subjects randomized to the MG received all three treatments, that is, the health information, diet and exercise interventions with the same protocols observed as in the individual treatments.

3.5.4 Blinding

The nature of lifestyle intervention studies, based upon behavioural modification, does not lend itself to blinding of the treatments. Thus, neither the participants nor the research assistants were blinded in this study.

3.6 Post-Test (Follow-up) Period

The post-test (follow-up) period constituted the last phase of the study during which the measurements taken in the pre-test (baseline) period were repeated.

3.6.1 Phase 6: Post-Test (Follow-up) Measurements

The measurements taken at post-test were a repetition of those in the pre-test and related to CHD risk factors, HRPF, HRB, daily dietary intake and daily physical activity. All test procedures were the same as the pre-test procedures, and the same testers were used as well.

3.7   Quality Control

The main aspects of quality control in this study could be classified into two categories, that is, (1) the research instruments used, and (2) the research assistants (testers) who conducted the tests.

3.7.1 Instrument Validity and Reliability

The research instruments used to gather data in the study were the four questionnaires and the technical equipment for taking physical and physiological measurements.

3.7.1.1   Questionnaires
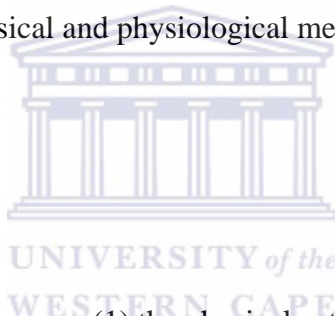
The questionnaires used in the study were (1) the physical activity readiness questionnaire (PAR-Q) for screening purposes, the SRCQ questionnaire for stages of readiness to change, (3) the HLQ questionnaire for healthy lifestyle and the long version IPAQ for daily physical activity.

3.7.1.1.1   Physical Activity Readiness-Questionnaire (PAR-Q)

The PARQ is a screening instrument consisting of seven close-ended (yes – no) questions that is used to identify people aged 15 to 69 years who may be at risk, because they do not exercise regularly and may become symptomatic should they undergo a fitness assessment or become

more physically active. The PAR-Q has previously been validated as a screening instrument prior to participating in moderate-to-vigorous exercise (Cardinal et al., 1996).

3.7.1.1.2   Stages of Readiness to Change Questionnaire (SRCQ)

The SRCQ contains six stages and response categories ranging from precontemplation to relapse that are used to identify and classify the readiness of respondents to change one or more health behaviours. The SRCQ has been shown reliable in a survey of the Perth metropolitan general population aged 16-69 years (Donovan et al., 1998).

3.7.1.1.3   Healthy Lifestyle Questionnaire (HLQ)

No validation studies have been reported for the HLQ. Three health promotion experts with scientific and technical training and published in the public health field reviewed the questionnaire to establish face validity. In addition, the questionnaire was pre-tested on several occasions with first year university students who did not participate in the study.

The reliability of the HLQ was tested using the Cohen's reliability coefficient method. Stability of the instrument was demonstrated by test-retest reliability ($r = 0.52$) and internal consistency by Cronbach alpha (α) (pre-test $r = 0.98$ and post-test $r = 0.89$). Internal reliability refers to the extent to which the questionnaire is consistent within itself, in other words, how consistently the questions within each section measure overall health-risk behaviour. The Cronbach α statistic

indicates the consistency of responses to all items in the questionnaire. Cronbach α values range from 0 to 1, and a score ≥ 0.7 is generally acceptable.

3.7.1.1.4   International Physical Activity Questionnaire (IPAQ)

No standardized method exists for assessing physical activity. The IPAQ has been developed as an instrument for the international assessment and monitoring of physical activity and inactivity in individuals aged 18 to 65 years, and has been validated in 12 countries (Craig et al., 2003).

3.7.1.2   Clinical Equipment

The following clinical equipment was used, namely, a stethoscope, sphygmomanometer, beam balance scale, stadiometer, skinfold calipers, tape measure, enzymatic colorimeter, and dynamometer. All equipment used in the collection of research data was accurately calibrated following approved guidelines, and remained consistent throughout the study.

3.7.2   Inter-Rater (Tester)

All testing personnel used in the study were appropriately trained. Blood pressure measurements were standardized against an experienced clinician, using a double-headed stethoscope with measurements recorded to the nearest 2 mm Hg.

Testers measuring skinfolds were also trained by criterion testers according to the ISAK guidelines (Marfell-Jones et al., 2006), and the technical error of measurement (TEM) for testers was established within acceptable limits for research. In this regard, the requirements of tester accuracy (minimal error per test) and consistency (producing repeatable results from test to test) were standardized across all measurements, and testers not meeting acceptable criteria underwent further training. The accepted anthropometric technical error of measurement (measurement tolerance) for testers was as follows (Pederson and Gore, 1996, pp. 77-95):

- Body mass. 0.1 kg

- Height (stretch), 3 mm

- Skinfolds, 5%

- Girths (trunk), 3 mm

When taking skinfolds, the testers were instructed to measure the sites in succession (rotational rather than consecutive order), in other words, all measurements were taken once, before rotating back and repeating them a second or third time. This was done in order to allow the skin time to regain its normal tension, texture and colour and minimize bias. The right side of the body was used for all anthropometric measurements, unless this was impractical due to injury or disability.

For the HRPF tests, the testers were also trained and required to perform trials on a minimum of 20 subjects before proceeding to test the research subjects (Appendix O). A checklist was developed that stipulated the pre-test procedures that had to be followed (Appendix P)

With regard to subject integrity, the testers were instructed when taking measurements not to compromise the physical and emotional well-being of subjects, for example, struggling to measure subjects with extremely tight skin or extreme subcutaneous adiposity. Similarly, the testers were also sensitized to the cultural practices and traditions of the subjects, and encouraged to be mindful of these throughout the study, in addition to observing basic practices of hygiene keeping their fingernails trimmed and hands washed at all times.

3.7.3 Data Management

During testing, a recorder accompanied each tester, who entered and verified the accuracy of the data. The quality of testing was standardized by carefully preparing both the subject and the tester. To safeguard the privacy of all subject information, all testers participating in the study were required to sign a confidentiality declaration (Appendix O).

All subject data was captured electronically on computer into Microsoft Excel and stored against a private access code. Data entry was performed by an experienced data capturer (Master's student) with duplicate entry and cross-checking by a senior statistician. Queries were sent in batches to the principle researcher for resolution and verification.

3.8 Ethical Considerations

Ethical approval for the study (reference number: 09/6/7) was obtained from the institutional Review Board.

Three publications governed the ethics of the study, namely, "Guidelines on ethics for medical research: general principles" (SAMRC, 2001), "Guidelines on ethics for medical research: use of biohazards and radiation" (SAMRC, 2002), and the "Ethical approaches to gathering information from children and adolescents in international settings: guidelines and resources" (Shenk and Williamson, 2005).

The following ethical considerations were addressed:

- **Confidentiality**: Provision was made for the confidential safekeeping of subject information at all times, while subject anonymity was guaranteed in all publications.

- **Voluntary Written Informed Consent**: Prior to participating in the study, each subject received accurate and comprehensive information on the nature, scope, risks and benefits of the study before voluntarily granting their written consent. Subjects were informed that they could withdraw from the study at any stage. Subjects found to be at high risk for developing heart disease were referred to a physician and if their high risk status was confirmed, they were excluded from the study as a delimiting criterion.

- **Privacy**: Throughout the study, subjects were consulted individually and tested privately at the biokinetics clinic, Department of SRES at UWC.

- **Safety**: Every effort was made to ensure that no harm came to any subject. Universal precautions of safety and standardized emergency procedures (Appendix P) were observed

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when testing subjects. Also, reasonable precautions were taken to ensure that the participants did not endure unnecessary discomfort, while simultaneously adhering to standards of best practice. Equally important was the disposal of biohazardous material, such as the disposable lancets, gloves, cholesterol and glucose test strips and sterilizing materials. Such equipment was disposed of with due regard for the guidelines stipulated by the SAMRC (2002).

- **Inappropriate Inducement**: No inducements, such as monetary rewards were used to coerce subjects to participate in the study. In addition, at no stage were subjects made to feel that inclusion in the study would be beneficial or exclusion detrimental.

The following logistical considerations were observed to ensure that the subjects' academic progress was not compromised by their involvement in the study, namely:

- No exceptions were made for subjects attending lectures, is other words, all research activity commenced outside of the subjects' academic activity; and

- All research activity was suspended during the university "block" week, that is, the week preceding examinations when students had to study.

3.9   Statistical Analyses

All subject data was transferred, in duplicate, into an MS Office Excel 2007 spreadsheet, while maintaining subject anonymity at all times. The data was analyzed using the Statistical Analysis

System (SAS) for Windows (version 9.2, Cary, North Carolina, USA) software. In the analysis, the intention-to-treat principle was not applied, therefore, the impact of the subjects' levels of compliance was taken into consideration, and the results analyzed accordingly. All electronic back-up copies of the data were stored against password access restrictions that were controlled by the researcher.

The study contained multiple datasets, namely, clinical, performance and behavioural measurements. Across all datasets, a significance level $p < .05$ was used. Where the outcome variables were normally distributed, the groups were compared using a two-sample t-test. For outcomes with a non-normal distribution or ordinal level data, the nonparametric Wilcoxon Rank Sum test was used for group comparisons. To account for baseline differences, repeated measures analysis of variance was used. Where nonparametric methods were appropriate, analysis was done using Cochran-Mantel-Haenszel (CMH) methodology, stratifying on the baseline values. For nominal level outcomes, groups were compared by Chi-square tests for homogeneity of proportions. When baseline values needed to be incorporated into the analysis, this was done using CMH methodology.

A simplified model of the procedure used in the statistical analyses of data is presented below with between and within groups comparisons made at pretest and post-test:

	Between Groups	
Within Groups	Pre-test $\longrightarrow$ $\quad$ Pre-test $\downarrow$	
	Post-test $\longrightarrow$ $\quad$ Post-test	
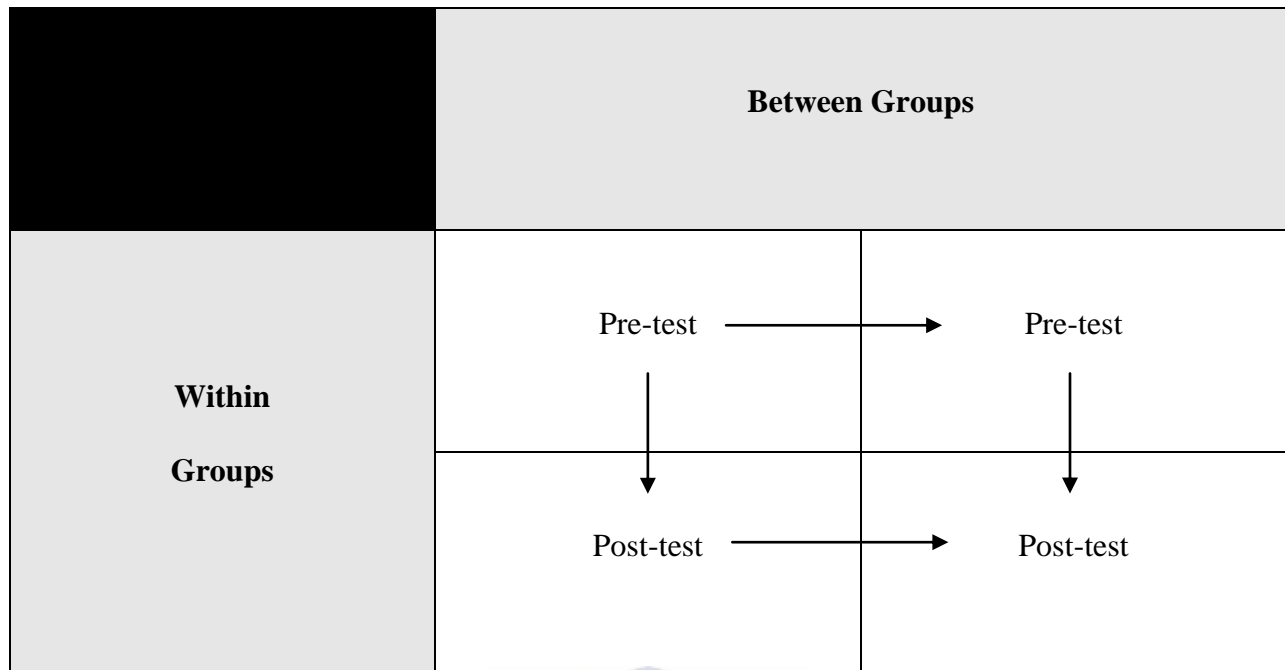
Below is a grid used for interpreting statistical significance of varying levels. The letters "a to d" indicate four of the groups, namely, a = CG, b = HIG, c = DG and d = EG. The numbers 1 to 5 indicate the various levels of significance ranging from $1 = p < .05$ to $5 = p < .0005$. Thus, for example, a rating of 1 means that when compared to the control group there was a significant difference at $p < .05$ and, similarly a rating of b3 indicates significance at $p < .005$ when compared to the HIG.

Groups	Significance Levels				
	1= p < .05	2 = p < .01	3 = p < .005	4 = p < .001	5 = p < .0005
CG = a	a1	a2	a3	a4	a5
HIG = b	b1	b2	b3	b4	b5
DG = c	c1	c2	c3	c4	c5
EG = d	d1	d2	d3	d4	d5

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Chapter Four: Results

4.1   Introduction

This chapter presents the results of the study investigating the impact of multiple health behavioural interventions on CHD risk, HRPF and HRB in first year university students. There were several objectives, namely:

- To identify a sample of first year students at moderate CHD risk;

- To measure the HRPF levels in the at-risk subjects;

- To measure the HRB in the at-risk subjects;

- To assess the impact of multiple health interventions on CHD risk factors;

- To assess the impact of multiple health interventions on HRPF;

- To assess the impact of multiple health interventions on HRB; and

- To assess whether the impact of multiple health interventions are dose-response related.

The results are presented in 3 periods containing a total of 6 phases. The periods are the **Pre-test**, **Intervention** and **Post-test** periods.

The Pre-test period: This period contains Phase 1 (subject recruitment), Phase 2 (subject selection, screening and preparation) and Phase 3 (pre-test measurements of CHD risk factors, HRPF, HRB, diet and physical activity).

The Intervention period: This period contains Phase 4 (subject randomization into treatment groups) and Phase 5 (implementation of the intervention strategy, during which the recording of the attrition of subjects, subject compliance to treatment and adverse events during the implementation of treatments took place).

The Post-test period: This period contains Phase 6 (post-test measurements of CHD risk factors, HRPF, HRB, diet and physical activity)

A variety of applications were used in the analysis of the data, namely, descriptive statistics, such as frequency distributions and measures of central tendency and dispersion, as well as inferential statistics in order to establish whether statistically significant differences between groups at baseline (pre-test) and follow-up (post-test) were present. A comparison of the treatment groups at baseline and after intervention was done by tabulation of the mean ($\pm$ SD). To account for baseline differences, the repeated measures analysis of variance was used and $p < .05$ was accepted as significant. The findings are presented in text as well as visual (tables and graphs) formats.

4.2   Pre-Test (Baseline) Period

4.2.1 Phases 1 and 2: Study Population and Recruitment

The study population consisted of first year university students enrolled at a historically-black

university (HBU) in the Western Province (WP), SA. Subjects were recruited for two

consecutive years, in 2008 and 2009.

For the periods 2008 and 2009, the numbers of first year students enrolled at the university were

4171 and 4 593, respectively, totaling 8 764 students over the two year period. For 2008, the

number of students who volunteered to participate in the study was 368, and in 2009 the number

was 426. Over the two years, the number of subjects recruited totaled 794 and represented 9.06%

of the enrolment figures. Figure 4.1 as a schematic representation of the subject flow through the

initial recruitment phase.
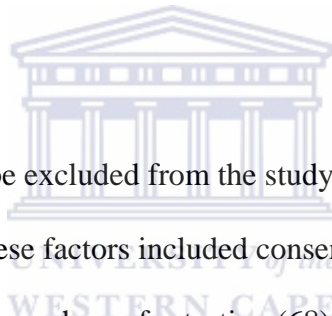
Several factors caused subjects to be excluded from the study and impacted upon the size of the

sample that was tested initially. These factors included consent forms not returned (58),

university drop-out (2), repeats (5), no-shows for testing (68), positive PAR-Q, and excluded

based upon the physician's advice (2), physically injured in a motor vehicle accident (2),

adversely affected by testing (1), declined to have their blood drawn (3), declined to participate

in the HRPF tests (5), incomplete baseline results (34), abscondments after baseline testing (9).
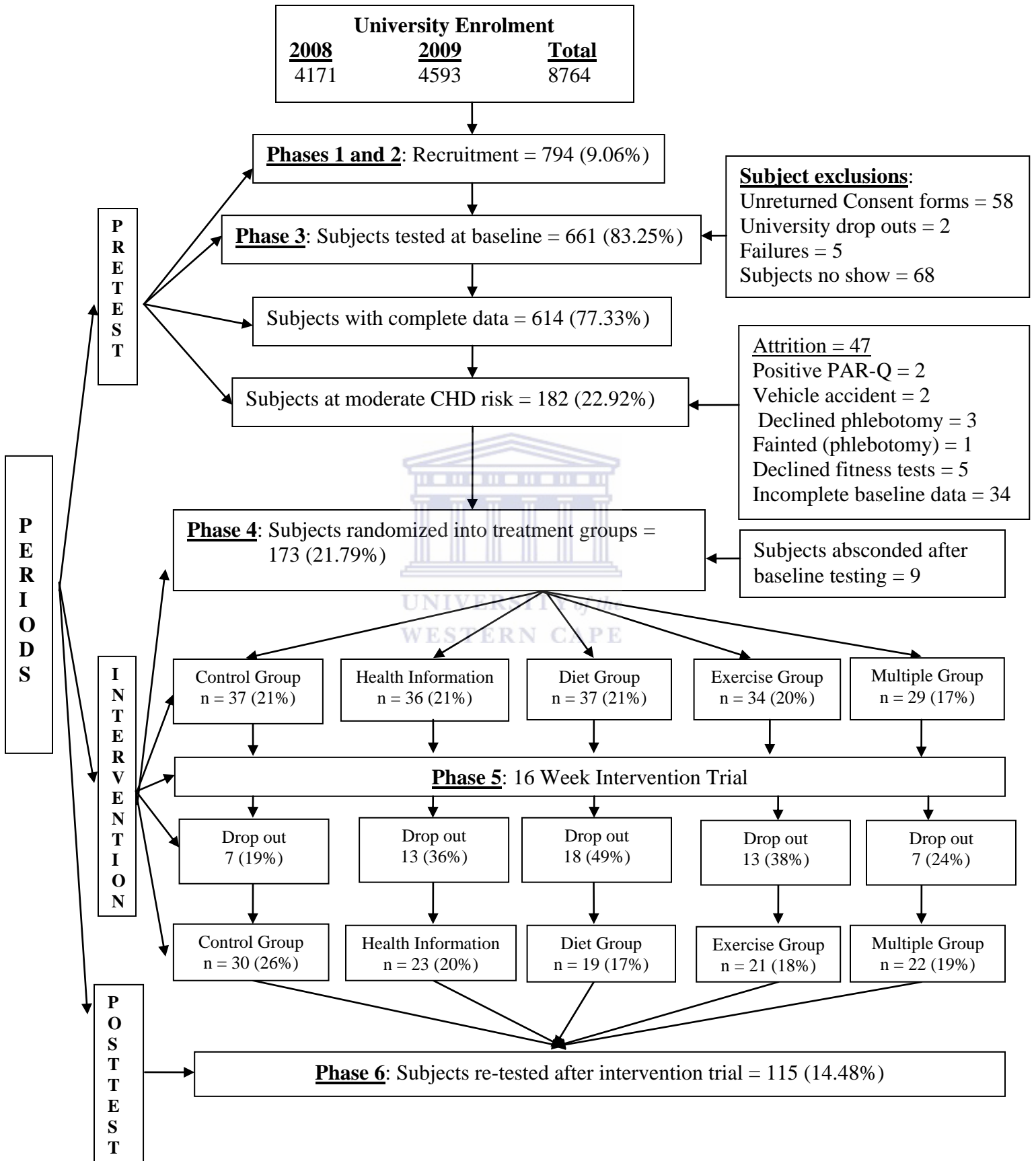
Figure 4.1. Flow chart of subjects' movement through the study.

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From a total of 794 subjects, 614 presented themselves for baseline testing, and 182 were found to be at moderate CHD risk. After receiving their baseline results, 9 subjects did not return and were excluded from the study. In total, 189 (23.80%) subjects were excluded, and 173 (21.79%) were stratified as moderate CHD risk and proceeded with the study.

4.2.2 Phase 3: Pre-Test (Baseline) Results

The pre-test results presented in this section refer to the following components of the study, namely, CHD risk factors, HRPF, HRB, daily dietary intake and daily physical activity. The results for Phase 4, subject randomization into treatment groups, is presented first, as the pre-test data is discussed within the context of each one of the groups.

4.2.2.1 Phase 4: Subject Randomization into Treatment Groups

Table 4.1 contains the results of the randomization of subjects to the different treatment groups, expressed in numbers and percentages. A total of 182 subjects were initially stratified as being moderately at risk for CHD. Following the baseline tests, 9 subjects absconded, that is, 2 from the exercise group (EG) and 7 from the multiple group (MG), which left these groups with 34 and 29 subjects respectively, and caused them to be unbalanced compared to the other groups. Thus, from the 173 subjects who remained in the study, the sizes of each of the groups were as follows: 37 (21.39%) in the control group (CG), (n 36 (20.81%) in the health information group (HIG), 37 (21.39%) in the diet group (DG), 34 (19.65%) in the exercise group (EG) and 29 (16.76%) in the multiple group (MG).

The subjects who absconded from the study came from the two groups that had exercise as a common treatment intervention. This factor might have influenced their decision not to return to the study following baseline testing as these were predominantly sedentary individuals.

Table 4.1. Randomization of subjects to treatment groups.

Groups	Initial Group Size	%	Subject Loss	Final Group Size	%
Control Group (CG)	37	20.33	0	37	21.39
Health Information Group (HIG)	36	19.78	0	36	20.81
Diet Group (DG)	37	20.33	0	37	21.39
Exercise Group (EG)	36	19.78	2	34	19.65
Multiple Group (MG	36	19.78	7	29	16.76
Total	182	100	9	173	100
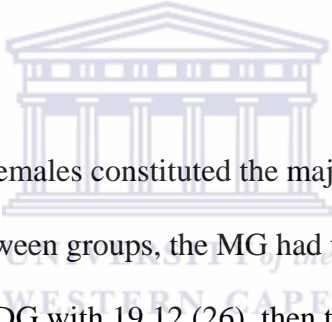
4.2.2.2 Demographic Characteristics of Subjects

Table 4.2 contains the distribution of gender results arranged according to the 5 treatment groups. The size varied between the groups, and ranged from the smallest to largest as follows: the MG with 29 (16.76%) subjects, the EG with 34 (19.65%) subjects, the HIG with 36 (20.81%) subjects, and the DG and CG both with 37 (21.39%) subjects each.

Two types of comparisons were made, namely, between groups (or row %) and within groups (or column %), with the former comparing gender distribution across the 5 groups, and the latter comparing gender distribution within a group.

Between each group, the DG had the highest distribution of males with 29.73% (11) and the MG the lowest with 10.81% (4). The EG had 16.22% (6), and the HIG and CG with 21.62% (8) each. When comparing within groups, the males were in the minority in all the groups, with the lowest distribution in the MG with 13.79% (4), followed by the EG with 17.65% (6), then the CG with 21.62% (8), and the HIG with 22.22% (8). The highest number of males was in the DG with 29.73% (11).

The grand group (GG) shows that females constituted the majority with 78.61% (136) compared to the males with 21.39% (37). Between groups, the MG had the lowest distribution of females with 18.38% (25), followed by the DG with 19.12 (26), then the HIG and EG with 20.59% (28), and the highest being the CG with 21.32% (29). Within groups, the distribution of females varied from the lowest in the DG with 70.27% (26), followed by HIG with 77.78% (28), then the CG with 78.34% (29), and the EG with 82.35% (28), to the highest in the MG with 86.21% (25). Thus, even though the MG had the lowest number of males with 25, they constituted a larger percentage 86.21%, because it was the smallest group (29).

Briefly stated, the DG had the highest distribution of males both between and within groups, whereas for females the CG had the highest distribution between groups and the MG the highest distribution within groups.

Table 4.2. Gender distribution of subjects.

Subjects	Pre-Test Treatment Groups					
	Control (CG)	Health Information (HIG)	Diet (DG)	Exercise (EG)	Multiple (MG)	Grand (GG)
Number of subjects	37	36	37	34	29	173
Row%	21.39	20.81	21.39	19.65	16.76	100
Males	8	8	11	6	4	37
Row%	21.62	21.62	29.73	16.22	10.81	100
Column%	21.62	22.22	29.73	17.65	13.79	21.39
Females	29	28	26	28	25	136
Row%	21.32	20.59	19.12	20.59	18.38	100
Column%	78.34	77.78	70.27	82.35	86.21	78.61

Table 4.3 contains the racial demographics of the subjects distributed across the 5 treatment groups. The GG shows that the majority of the subjects were of mixed ethnic ancestry at 63.01% (109), followed by blacks making up 32.37% (56), with the same distribution for Indians and Whites at 2.31% (4) each. Across the different treatment groups, a similar racial distribution was seen as well. Subjects of mixed ethnic ancestry constituted the largest distribution in each of the groups, and varied from the lowest in the EG with 15.60% (17), followed by the MG with 17.43% (19), then the CG with 20.18% (22), and the DG with 22.94% (25), to the highest in the HIG with 23.85% (26). Blacks were the second largest population in each of the groups, with most being in the CG and EG at 23.21% (13) each, followed by the DG with 21.43% (12), and the least in HIG and MG with 16.07 (9) each. White and Indian subjects were in the minority and constituted only 2.31% (4) each of the GG. Indian subjects were distributed in the CG, HIG and EG, and made up 50.00 (2) in the CG and 25.00% (1) each in the HIG and EG. Similarly, the

white subjects were found in two groups only, the EG and MG, and constituted 75.00% (3) and 25.00% (1) of each group, respectively.

Table 4.3. Racial demographics of the subjects.

Groups		Racial Demographics				
		Black	Mixed Ethnic Ancestry*	Indian Ancestry[#]	White	Total
Grand	Total Frequency	56	109	4	4	173
	Row%	32.37	63.01	2.31	2.31	100
Control	Column Frequency	13	22	2	0	37
	Row%	35.14	59.46	5.41	0.00	100
	Column%	23.21	20.18	50.00	0.00	21.39
Health Information	Column Frequency	9	26	1	0	36
	Row%	25.00	72.22	2.78	0.00	100
	Column%	16.07	23.85	25.00	0.00	20.81
Diet	Column Frequency	12	25	0	0	37
	Row%	32.43	67.57	0.00	0.00	100
	Column%	21.43	22.94	0.00	0.00	21.39
Exercise	Column Frequency	13	17	1	3	34
	Row%	38.24	50.00	2.94	8.82	100
	Column%	23.21	15.60	25.00	75.00	19.65
Multiple	Column Frequency	9	19	0	1	29
	Row%	31.03	65.52	0.00	3.45	100
	Column%	16.07	17.43	0.00	25.00	16.76

* refers to persons of mixed ethnic ancestry or origin based upon the South African population registry
[#] refers to Asian, oriental ancestry or origin based upon the South African population registry

Within each group, subjects of mixed ethnic origin were in the majority with the HIG having the highest proportion at 72.22% (26), followed by the DG with 67.57% (25), then the MG with 65.52% (19), and the CG with 59.46% (22), the lowest in the EG with 50.00% (17). A

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considerable number of blacks were also found within each group, with the largest proportion being in the EG with 38.24% (13), followed by the CG with 35.14 (13), then the DG with 32.43% (12), and the MG with 31.03% (9), and the smallest proportion in the HIG with 25.00% (9). Indian and white subjects were the least in each of the groups, with Indian subjects comprising 5.41% (2), 2.94% (1) and 2.78% (1), in the CG, EG and HIG, respectively, and white subjects comprising 8.82% (3) and 3.45% (1) in the EG and MG, respectively.

In brief, a comparison of the racial distribution of subjects in the different treatment groups showed that between groups, subjects of mixed ethnic ancestry constituted the majority followed by black subjects, with Indian and white subjects constituting the minority. This pattern of racial distribution was consistent for within group comparisons as well.

The marital status is contained in Table 4.4, and shows most of the subjects as unmarried (90.17%), and a small percentage as married (9.25%) or widowed (0.58%).

Table 4.4. Marital status of the subjects.

Subjects	Marital Status			
	Unmarried	Married	Widowed	Total
Number of Subjects	156	16	1	173
%	90.17	9.25	0.58	100

The injury status is presented in Table 4.5, and shows most (81.50%) of the subjects as injury-free at baseline, while some (13.87%) complained of prolonged orthopedic conditions, such as shoulder, knee, back or other joint pain, and a few (4.62%) had chronic medical conditions, such as asthma, sinusitis, allergies and/or hypertension. However, none of these conditions were considered exclusion criteria or debilitating enough to exclude any of these subjects from the study.

Table 4.5. Injury status of the subjects.

Subjects	Injury Status			
	Uninjured	Injured	Chronic Condition	Total
Number of subjects	141	24	8	173
%	81.50	13.87	4.62	100

At the start of the pre-test period, the subjects were assessed for the stage of readiness to change the lifestyle. The purpose of the assessment was not aimed at targeting specific behaviours, such as cigarette smoking or physical inactivity, but rather at getting an overview of the subjects' willingness to change their lifestyle in general.

Table 4.6 presents the results of the subjects' readiness to change their lifestyles at baseline. The majority of the subjects were in the contemplative stage (CS) of change as shown in the GG with 68.21% (118). Subjects in the preparatory stage (PS) followed next with 19.65% (34), with a few

subjects in the maintenance stage (MS) (1.16% or 2) and the relapse stage (RS) (2.31% or 4). A few subjects were in the precontemplative stage (1.73% or 3), and were not considering a lifestyle change. The distribution of subjects in the 6 stages of readiness to change followed a similar pattern as that shown in the GG, with most of the subjects in the CS, followed by the PS, and then the AC. The MG contained the largest number of subjects in the PS, 34.48% (10), which indicated considerable progress towards lifestyle change. Subjects in the MS, RS and PCS were few in number, and varied across the treatment groups.

Table 4.6.   Distribution of subjects for stages of readiness to change at pre-test.

Groups		Stages of Readiness to Change						
		PCS	CS	PS	AC	MS	RS	Total
Grand	Frequency	3	118	34	12	2	4	173
	%	1.73	68.21	19.65	6.94	1.16	2.31	100
Control	Frequency	1	26	5	4	0	1	37
	%	2.70	70.27	13.51	10.81	0.00	2.70	100
Health Information	Frequency	1	25	7	2	0	1	36
	%	2.78	69.44	19.44	5.56	0.00	2.78	100
Diet	Frequency	0	26	6	3	0	2	37
	%	0.00	70.27	16.22	8.11	0.00	5.41	100
Exercise	Frequency	1	25	6	1	1	0	34
	%	2.94	73.53	17.65	2.94	2.94	0.00	100
Multiple	Frequency	0	16	10	2	1	0	29
	%	0.00	55.17	34.48	6.90	3.45	0.00	100

Key: PCS = precontemplation stage; CS = contemplation stage; PS = preparation stage; AC = action stage; MS = maintenance stage; RS = relapse stage

4.2.2.3   CHD Risk Factors

Table 4.7 contains the findings on the physical characteristics and CHD risk factors of the subjects at pre-test expressed as mean±SD. The mean age for the GG was 21.35±6.24 years, and varied from the youngest in the EG at 19.68±3.54 years to the oldest in the CG at 23.92±9.14 years. Except for the HIG, age differed significantly ($p < .05$) in the DG, EG and MG when compared to the CG.

Family history of CHD was relatively high in all groups, with the GG at 40.37±49.22%. Higher values were recorded in the CG and HIG with 47.06±50.66% and 46.88±50.70%, respectively, and reflected the extent to which a family history of CHD was prevalent amongst this group. The CG had a higher mean value for height at 163.91 ±8.39 cm, with similar values in the other groups ranging from 162.15±7.79 cm in the MG to 162.71±8.41 cm in the DG, with none being significantly different.

Body composition, as indicated by body mass, BMI, waist circumference, hip circumference and waist-hip ratio (WHR), was highest in the DG and lowest in the HIG with no significant differences indicated for any of these variables. The mean values for BMI in all the groups fell into the overweight rating, but were low for CHD risk. The mean values for WHR and waist circumference in all the groups also into the low risk rating for CHD.

For the clinical measurements of resting heart rate, systolic and diastolic blood pressures, higher values were found in the DG than in the other groups, with no significant differences

Table 4.7. Physical characteristics and CHD risk factors of the subjects at pre-test.

Physical Characteristics and CHD risk factors	Grand Group (mean±SD) (n = 173)	Control Group (mean±SD) (n = 37)	Health Information Group (mean±SD) (n = 36)	Diet Group (mean±SD) (n = 37)	Exercise Group (mean±SD) (n = 34)	Multiple Group (mean±SD) (n = 29)	p value (Between Groups)
Age (years)	21.35±6.24	23.92±9.14	21.50±6.36	20.57±3.83[a1]	19.68±3.54[a3]	20.86±5.88[a1]	.048
Family history of CHD (%)	40.37±49.22	47.06±50.66	46.88±50.70	37.84±49.17	33.33±47.95	35.71±48.80	.695
Height (cm)	162.73±8.49	163.91±8.39	162.34±8.44	162.71±8.41	162.39±9.65	162.15±7.79	.915
Body Mass (kg)	76.38±19.24	77.80±18.61	71.78±15.69	79.32±21.02	75.89±19.79	77.10±21.19	.532
BMI (kg·m^{-2})	28.92±7.30	29.25±8.15	27.43±6.49	29.74±6.45	28.86±7.27	29.40±8.31	.710
Waist Circumference (cm)	82.21±14.85	83.97±17.05	80.09±11.69	83.72±13.97	82.07±15.99	80.86±15.58	.763
Hip Circumference (cm)	106.15±13.91	106.94±15.07	103.37±11.61	107.92±13.35	105.31±13.23	107.28±16.62	.655
Waist-Hip Ratio	0.77±0.09	0.78±0.10	0.77±0.07	0.77±0.07	0.78±0.12	0.75±0.06	.663
Resting Heart Rate (bpm)	78.42±10.73	77.11±11.11	79.14±7.51	80.46±13.63	78.38±9.88	76.62±10.56	.584
Resting Systolic BP (mmHg)	123.40±12.05	122.70±11.23	122.39±15.13	124.54±10.98	123.47±11.12	124.00±11.77	.942
Resting Diastolic BP(mmHg)	80.45±9.55	81.49±10.28	77.36±9.84	80.87±8.24	81.59±8.27	81.10±10.95	.301
Cigarette Smoking (cpd)	2.72±4.03	2.46±3.00	2.53±3.60	3.38±4.99	2.41±4.19	2.83±4.31	.840
Sedentary (%)	91.95±27.40	89.24±31.50	94.40±23.20	91.90±27.70	94.14±23.97	89.74±31.94	.898

Key: CHD = coronary heart disease; BP = blood pressure; bpm = beats per minute; mm Hg = millimetres mercury; cpd = cigarettes per day
[a1] = $p < .05$ and [a3] = $p < .005$

Table 4.8.   Blood biochemical results of the subjects at pre-test.

Blood Biochemical Parameters	Grand Group (mean±SD) (n = 147)	Control Group (mean±SD) (n = 37)	Health Information Group (mean±SD) (n = 26)	Diet Group (DG) (mean±SD) (n = 36)	Exercise Group (mean±SD) (n = 24)	Multiple Group (mean±SD) (n = 24)	p value (Between Groups)
Total Cholesterol (TC) mmol$^.$L^{-1})	4.28±0.78	4.22±0.87	4.35±1.07	4.28±0.71	4.37±0.65	4.24±0.51	.945
HDL Cholesterol (mmol$^.$L^{-1})	1.20±0.26	1.14±0.26	1.17±0.22	1.22±0.26	1.19±0.27	1.29±0.26	.268
LDL Cholesterol (mmol$^.$L^{-1})	2.61±0.73	2.58±0.78	2.69±0.98	2.60±0.65	2.69±0.58	2.51±0.59	.898
Triglycerides (mmol$^.$L^{-1})	0.82±0.45	0.75±0.33	1.01±0.60	0.82±0.53	0.77±0.24	0.78±0.44	.186
Impaired Fasting Glucose (mmol$^.$L^{-1})	4.42±0.44	4.44±0.50	4.52±0.38	4.28±0.43	4.42±0.26	4.53±0.53	.134
TC/HDL Cholesterol Ratio	3.78±1.31	3.97±1.65	3.91±1.47	3.93±1.32	3.83±0.91	3.40±0.72	.538
LDL/HDL Cholesterol Ratio	2.34±1.04	2.46±1.27	2.45±1.21	2.28±0.96	2.40±0.84	2.04±0.70	.572
Triglyceride/HDL Ratio	0.75±0.55	0.71±0.41	0.94±0.67	0.77±0.78	0.69±0.28	0.61±0.30	.261

Key: LDL = low density lipoprotein; HDL = high density lipoprotein

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present. The mean values for systolic and diastolic blood pressures in all the groups fell into the pre-hypertensive category, but were low for CHD risk.

Cigarette smoking was also highest in the DG at 3.38±4.99 cpd, but the mean number of cigarettes smoked per day was relatively low in all the groups, with the GG at 2.72±4.03 cpd, and is generally classified as occasional (< 5 cpd) than regular smoking.

Similar to the findings for a family history of CHD, a sedentary lifestyle was also particularly high in all groups, with the GG at 91.95±27.40%. Higher values were recorded in the HIG and EG with 94.40±23.30% and 94.14±23.97%, respectively, and reflected the extent to which a sedentary lifestyle was prevalent amongst this group.

The above-mentioned findings show that the results of DG for body composition, heart rate, systolic and diastolic blood pressures, and cigarette smoking, when combined with a family history of CHD and a sedentary lifestyle, predispose this group to CHD. In addition, the clustering of high values for a family history of CHD, cigarette smoking and a sedentary lifestyle in all the groups is also noteworthy. With the exception of age, no significant differences were present for any of the other variables measured at baseline (Table 4.7).

Table 4.8 contains the results for blood biochemistry at baseline, expressed as mean±SD. LDL cholesterol concentration is the recommended measurement for indicating CHD risk, and in its absence, total cholesterol and HDL cholesterol. The mean concentrations for all of these measurements fell within the normal range as shown in the GG with mean concentrations of

4.28±0.78, 1.20±0.26, and 2.61±0.73 mmol·L^{-1}, for TC, HDL and LDL, respectively. The mean triglyceride concentration in the GG was 0.82±0.45 mmol·L^{-1}, and was similar to the values in the other groups as well with mean concentrations varying from 0.75±0.33 mmol·L^{-1} in the CG to 1.01±0.60 mmol·L^{-1} in the HIG, but all concentrations fell within the normal range.

In the HIG, even though most of the values for blood biochemistry appeared higher compared to the other groups, none were noteworthy, and all fell within normal limits. The MG had the highest mean HDL cholesterol concentration with 1.29±0.26 mmol·L^{-1} and the lowest LDL and TC cholesterol concentrations with 2.51 ±0.59 and 4.24±0.51 mmol·L^{-1}, respectively.

The mean concentrations for all the cholesterol ratios, namely, TC:HDL, LDL:HDL and trig:HDL, were low and within normal limits. All cholesterol concentrations and ratios were not significant.
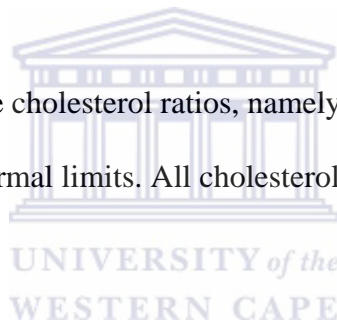
The MG recorded the lowest ratios for TC:HDL, LDL:HDL and trig:HDL with values of 3.40±0.72, 2.04±0.70 and 0.61±0.30 mmol·L^{-1}. The CG, in contrast, recorded the highest ratios for TC:HDL and LDL:HDL with values of 3.97±1.47 and 2.46±1.27 mmol·L^{-1}. The HIG had the highest trig:HDL ratio of 0.94±0.67 mmol·L^{-1}.

In summary, TC, HDL cholesterol, LDL cholesterol and triglycerides, together with the ratios and impaired fasting glucose did not differ significantly at baseline. The inverse relationship between HDL and LDL cholesterol concentration was evident in all the groups and also impacted the LDL:HDL ratio.

4.2.2.3.1 Prevalence of CHD Risk Factors

As shown in Figure 4.2, at baseline, the seven CHD risk factors investigated in this study were ranked according to prevalence or frequency of occurrence. Impaired fasting glucose was least prevalent at 0.34 % (1), whereas a sedentary lifestyle was most prevalent at 31.19% (92). Cigarette smoking, obesity, a family history of CHD, and dyslipidemia all had similar prevalence rates of 17.97% (53), 14.24% (42), and 13.56% (40) for the last two, respectively. Hypertension was present in 9.15% (27) of the subjects.

Series1	Impaired Fasting Glucose	Hyper-tension	Dys-lipidemia	Family History of CHD	Obesity	Smoking	Sedentary Lifestyle
	0.34	9.15	13.56	13.56	14.24	17.97	31.19
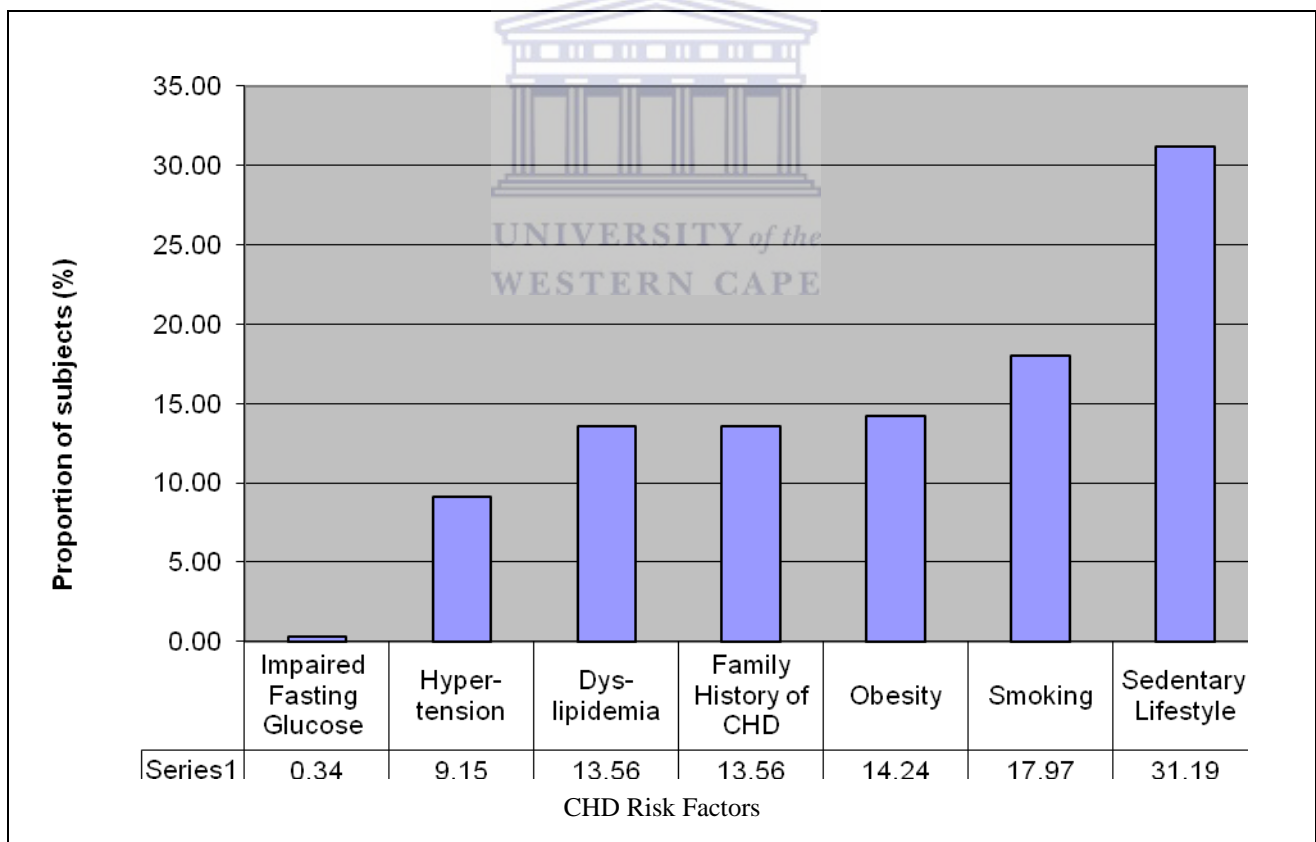
CHD Risk Factors

Figure 4.2.   Prevalence of CHD risk factors in subjects at pre-test.

Figure 4.3 presents findings on the frequencies of the CHD risk factors, inter alia, the number of subjects with a frequency of two CHD risk factors and frequencies of three risk factors. For participants to be eligible for the study, they had to have a minimum of two (2) CHD risk factors present, hence the absence of subjects with zero (0) or one (1) risk factor at baseline. Also, none of the subjects presented with a frequency of all 7 CHD risk factors at baseline. The percentage of subjects with two, three, four, five and six risk factors were 45.66% (79), 30.06% (52), 16.18% (28), 7.51% (13) and 0.58% (1), respectively. At baseline, 54.34% (94) of the subjects had three or more risk factors.

	0 RF	1 RF	2 RF	3 RF	4 RF	5 RF	6 RF	7 RF
Series1	0.00	0.00	45.66	30.06	16.18	7.51	0.58	0.00

Frequencies of CHD Risk Factors
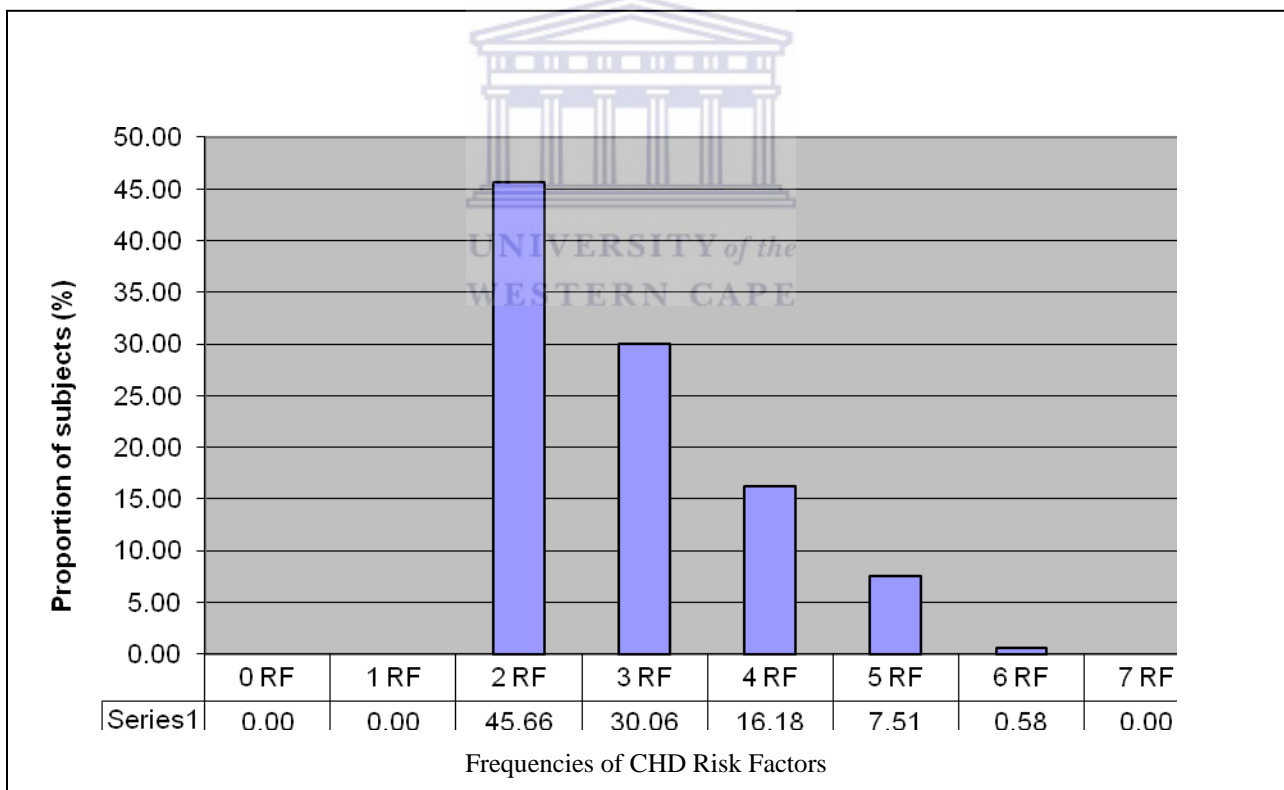
RF = risk factor

Figure 4.3.   Distribution of frequencies of CHD risk in the subjects at pre-test.

Table 4.9 shows the distribution of CHD risk factor frequencies per treatment group. A similar pattern of distribution of CHD risk factor frequencies for the GG was also evident across the various treatment groups. All the groups showed that subjects with a frequency of two CHD risk factors occurred the most, except in the CG where subjects with a frequency of three risk factors occurred the most at 37.84% (14).

The mean frequency of CHD risk factors for the GG was 2.88±0.98, with the highest frequency recorded in the CG at 3.16±1.07 and the lowest in the HIG at 2.75±1.00.

Table 4.9. Distribution of frequencies of CHD risk per treatment group at pre-test.

Groups		Pre-Test Period Frequencies of CHD Risk						
		2 RF	3 RF	4 RF	5 RF	6 RF	Total	Mean±SD
Grand (n = 173)	Total Frequency	79	52	28	13	1	173	2.88±0.98
	Row%	45.66	30.06	16.19	7.51	0.58	100	
Control (n = 37)	Column Frequency	12	14	5	5	1	37	3.16±1.07
	Row%	32.44	37.84	13.51	13.51	2.70	100	
	Column%	15.19	26.92	17.86	38.46	100	21.39	
Health Information (n = 36)	Column Frequency	20	9	4	3	0	36	2.75±1.00
	Row%	55.56	25.00	11.11	8.33	0	100	
	Column%	25.32	17.31	14.29	23.08	0	20.81	
Diet (n = 37)	Column Frequency	15	13	8	1	0	37	2.84±0.87
	Row%	40.54	35.14	21.62	2.70	0	100	
	Column%	18.99	25.00	28.57	7.69	0	21.39	
Exercise (n = 34)	Column Frequency	21	6	4	3	0	34	2.77±1.02
	Row%	61.77	17.65	11.76	8.82	0	100	
	Column%	26.58	11.54	14.29	23.08	0	19.65	
Multiple (n = 29)	Column Frequency	11	10	7	1	0	29	2.86±0.92
	Row%	37.93	34.48	24.14	3.45	0	100	
	Column%	13.92	19.23	25.00	7.69	0	16.76	

Key: RF = risk factors.

4.2.2.4   Health-Related Physical Fitness

The results for HRPF, as depicted in Table 4.10, show that the GG with a mean for predicted maximal aerobic capacity ($\dot{V}O_2$max) recorded at 26.32 ± 7.22 ml$\cdot$kg$^{-1}\cdot$min^{-1}. The range of scores for maximal aerobic capacity in the treatment groups was rather narrow, with the exercise group recording the lowest value at 25.26 ± 6.36 ml$\cdot$kg$^{-1}\cdot$min^{-1} and the HIG the highest with 27.42 ± 6.88 ml$\cdot$kg$^{-1}\cdot$min^{-1}. None of the measurements for maximal aerobic capacity were statistically significant, and all fell within a poor rating for cardiorespiratory fitness, a level synonymous with unfit subjects. The poor rating for cardiorespiratory fitness found in these subjects is understandable given the sedentary lifestyle recorded in over 90% of them, and adds to the health risk already present.
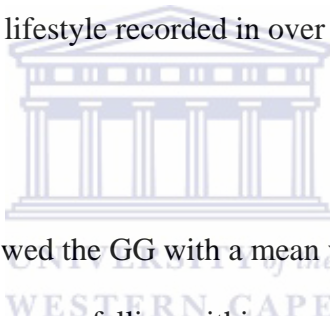
The results for percent body fat showed the GG with a mean value of $27.83\pm9.71\%$, and the range of scores in all the treatment groups falling within narrow limits with the lowest score recorded in the MG with $26.27\pm8.67\%$, followed by the CG with $27.42\pm10.68\%$, then the HIG at $28.13\pm10.81\%$, the DG at $28.02\pm8.97\%$, and the highest score recoded in the EG with $29.11\pm9.3\%$, but none were significant. The EG, recording the highest score for percent body fat, also recorded to lowest maximal aerobic capacity and supported the inverse relationship between maximal aerobic capacity and percent body fat. The findings for percent body fat showed all the groups to be classified as overfat and supported similar findings reported on BMI in the CHD risk assessment of these subjects.

Table 4.10. HRPF measurements of the subjects at pre-test.

Health-related Physical Fitness Measurements	Grand Group (mean±SD) (n = 173)	Control Group (mean±SD) (n = 37)	Health Information Group (mean±SD) (n = 36)	Diet Group (mean±SD) (n = 37)	Exercise Group (mean±SD) (n = 34)	Multiple Group (mean±SD) (n = 29)	p value (Between Groups)
Predicted $\dot{V}O_2$max (ml·kg^{-1}·min^{-1})	26.32±7.22	25.99±8.96	27.42±6.88	26.78±6.69	25.26±6.36	26.06±6.98	.769
Relative Body Fat (%)	27.83±9.71	27.42±10.68	28.13±10.81	28.02±8.97	29.11±9.31	26.27±8.67	.837
Absolute Body Fat (kg)	22.02±10.93	22.42±12.13	20.88±10.49	22.62±9.83	22.87±10.81	21.15±11.87	.921
Relative Lean Body Mass (%)	72.17±9.71	72.59±10.68	71.87±10.81	71.98±8.97	70.89±9.31	73.73±8.67	.837
Absolute Lean Body Mass (kg)	54.36±12.68	55.38±10.95	50.90±9.90	56.70±15.69	53.03±12.41	55.95±13.55	.288
Handgrip Strength (kg)	45.12±20.93	46.04±23.12	43.58±20.22	49.21±24.85	43.69±19.03	42.35±15.14	.495
Sit-Ups (rpm)	19.85±9.69	19.43±10.13	23.17±9.44	18.08±9.90	19.21±7.95	19.28±10.68	.217
Sit-and-Reach (cm)	46.36±9.43	44.82±9.93	45.61±9.54	45.71±10.75	47.38±9.19	48.92±6.72	.113

Key: rpm = repetitions per minute; $\dot{V}O_2$max = maximal aerobic capacity

The results for absolute body fat matched those reported for percent body fat, with the lowest (21.15±11.87 kg) and highest scores (22.87±10.81 kg) recorded in the MG and EG, respectively.

Percent lean body mass (LBM), which is the reciprocal measurement of percent body fat, was lowest in the EG with 70.89±9.31% and highest in the MG with 73.73±8.67%. Absolute LBM, however, was lowest in the EG with 53.03±12.41 kg but highest in the DG with 56.70±15.69 kg, and not the MG. This was probably because of the larger number of males in the DG group compared to the MG.

For muscular strength, the DG also recorded the highest handgrip strength with 49.21±24.85 kg. The highest handgrip strength recording in the DG could be related to the highest absolute LBM also recorded in this group. The lowest handgrip strength was found in the MG with 42.35±15.14 kg. None of the scores for handgrip strength were significant.

The HIG completed the highest number of sit-ups with 23.17±9.44 rpm. This group reported the highest scores for both muscular endurance and cardiorespiratory endurance. The DG completed the least number of sit-ups with 18.08±9.90 rpm. However, none of the scores differed significantly.

For the sit-and-reach test, the MG scored the highest with 48.92±6.72 cm and the CG the lowest with 44.82±9.93 cm, and none were significantly different.

The performances of the treatment groups on muscular fitness, which is a composite of muscular strength, muscular endurance and flexibility, were much better in all treatment groups when compared to the performances for cardiorespiratory fitness and body composition.

4.2.2.5   Health-Risk Behaviours

Table 4.11 contains a summary of the HRB of the participants from the healthy lifestyle questionnaire (HLQ).

Overall the responses of the participants in the different treatment groups are reflected in the results of the GG. Therefore, the results of the HLQ will be reported for the GG rather than the individual scores of the different treatment groups.

In the GG, 82.08% (142) of the participants felt that they were physically active enough to keep healthy. Most of them, 80.35% (139) responded that they were physically active at a moderate intensity for 30 minutes at least thrice per week, while 69.36% (120) were physically active at a vigorous intensity for 20 minutes at least twice a week. However, a minority, 9.25% (16), did a sufficient amount of stretching at least thrice per week. Similarly, conditioning for muscular fitness at least twice a week was done by only 8.67% (15) participants. Nevertheless, 72.25% (125) responded as being physically fit.

Table 4.11. HRB of the subjects at pre-test.

Health Behaviours	Control Group (n = 37)		Health Information Group (n = 36)		Diet Group (n = 37)		Exercise Group (n = 34)		Multiple Group (n = 29)		Grand Group (n = 173)	
Physical Activity	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Physically active enough to keep healthy	27	72.97	32	88.89	31	83.78	30	88.24	22	75.86	142	82.08
Physically active for 30 min. at least 3 d/wk	27	72.97	29	80.56	31	83.78	30	88.24	22	75.86	139	80.35
Vigorously active for 20 min. at least 2 d/wk	21	56.76	24	66.67	30	81.08	28	82.35	17	58.62	120	69.36
Stretch at least 3d/wk	6	16.22	0	0.00	5	13.51	2	5.88	3	10.34	16	9.25
Muscle fitness at least 2 d/wk	5	13.51	3	8.33	4	10.81	1	2.94	2	6.90	15	8.67
Physically fit	22	59.46	27	75.00	29	78.38	29	85.29	18	62.07	125	72.25
Total	108	48.65	115	53.24	130	58.56	120	58.82	84	48.28	557	53.66
Nutrition	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Eat at least 3 meals daily	30	81.08	35	97.22	37	100	31	91.18	27	93.10	160	92.49
Daily diet from 4 basic food groups	31	83.78	33	91.67	31	83.78	31	91.18	27	93.10	153	88.44
Try to cut down on fat in diet	29	78.38	32	88.89	34	91.89	32	94.12	24	82.76	151	87.28
Balance food intake to maintain weight	28	75.68	30	83.33	30	81.08	30	88.24	19	65.52	137	79.19
Think body is overweight	13	35.14	8	22.22	8	21.62	5	14.71	12	41.38	46	26.59
Total	131	70.81	138	76.67	140	75.68	129	75.88	109	75.17	647	74.80
Managing Stress	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Can identify stressful situations in daily life	26	70.27	33	91.67	33	89.19	31	91.18	26	89.66	149	86.13
Take time out daily to relax and recover	30	81.08	28	77.78	35	94.59	30	88.24	25	86.21	148	85.55
Find time for enjoyable activities	31	83.78	30	83.33	36	97.30	32	94.12	26	89.66	155	89.60
Know how to relieve tension	25	67.57	26	72.22	31	83.78	28	82.35	23	79.31	133	76.88
Total	112	75.68	117	81.25	135	91.22	121	88.97	100	86.21	585	84.54

Avoiding Destructive Habits	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Do not smoke or use tobacco products	25	67.57	29	80.56	34	91.89	30	88.24	22	75.86	140	80.92
Do not use alcohol or use to legal limit	30	81.08	30	83.33	34	91.89	292	85.29	25	86.21	148	85.55
Do not abuse drugs	34	91.89	34	94.44	35	94.59	31	91.18	27	93.10	161	93.06
Use OTC drugs as prescribed only	32	86.49	33	91.67	34	91.89	31	91.18	25	86.21	155	89.06
Total	121	81.76	126	87.50	137	92.57	121	88.97	99	85.34	604	87.28
Pratising Safe Sex	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Abstain from or limit sex to a safe partner	35	94.59	35	97.22	35	94.59	32	94.12	27	93.10	164	94.80
Practise safe procedures for avoiding STDs	32	86.49	34	94.44	33	89.19	32	94.12	28	96.55	159	91.91
Total	67	90.54	69	95.83	68	91.89	64	94.12	55	94.83	323	93.35
Adopting Safety Habits	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Use seat belt in a vehicle	32	86.49	32	88.89	35	94.59	31	91.18	26	89.66	156	90.17
Afraid when exceeding speed limit	22	59.46	27	75.00	33	89.19	28	82.35	23	79.31	133	76.88
Total	54	72.97	59	81.94	68	91.89	59	86.76	49	84.48	289	83.53
Knowing First Aid	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Trained in first aid	19	51.35	25	69.44	30	81.08	28	82.35	20	68.97	122	70.52
Can perform first aid in an emergency	20	54.05	25	69.44	30	81.08	28	82.35	19	65.52	122	70.52
Can call medical help in an emergency	35	94.59	35	97.22	36	97.30	31	91.18	27	93.10	164	94.80
Total	74	66.67	85	78.70	96	86.49	87	85.29	66	75.86	408	78.61
Personal Health Habits	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Brush teeth at least twice daily	32	86.49	35	97.22	37	100	34	100	29	100	167	96.53
Get enough sleep daily	25	67.57	30	83.33	34	91.89	28	82.35	21	72.41	138	80.23
Total	57	77.03	65	90.28	71	95.95	62	91.18	50	86.21	305	88.41
Using Medical Advice	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Seek medical advice when necessary	32	86.49	30	83.33	35	94.59	33	97.06	25	86.21	155	89.60
Follow advice or medication as prescribed	34	91.89	34	94.44	37	100	34	100	26	89.66	165	95.38
Total	66	89.19	64	88.89	72	97.30	67	98.53	51	87.93	320	92.49
Being an Informed Consumer	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Read product labels	13	35.14	8	22.22	6	16.22	4	11.76	11	37.93	42	24.28
Use products shown effective by research	22	59.46	26	72.22	33	89.19	30	88.24	25	86.21	136	78.61

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	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Total	35	47.30	34	47.22	39	52.70	34	50.00	36	62.07	178	51.45
Protecting the Environment	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Recycle waste	6	16.22	1	2.78	5	13.51	1	2.94	3	10.34	16	9.25
Conserve energy	29	78.38	31	86.11	34	91.89	33	97.06	26	89.66	153	88.44
Concerned about global warming	30	81.08	31	86.11	37	100	34	100	27	93.10	159	91.91
Total	65	58.56	63	58.33	76	68.47	68	66.67	56	64.37	328	63.20
Mental Well-being	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Have a sense of purpose in life	36	97.30	35	97.22	36	97.30	34	100	28	96.55	169	97.69
Positive about country's future	27	72.97	30	83.33	34	91.89	31	91.18	27	93.10	149	86.13
Total	63	85.14	65	90.28	70	94.59	65	95.59	55	94.83	318	91.91

Key: OTC = over-the counter; STDs = sexually transmitted diseases

Most of the participants, 92.49% (160), had at least three meals daily, and most of them, 88.44% (153), selected foods from the four basic food groups. Many of them, 79.19% (137), found it difficult to balance their food intake to maintain their body weight. Therefore, 87.28% (151) were still trying to reduce the fat and energy intake in their diet. However, less than a third of them, 26.59% (46), thought that they were overweight.

In general, not only could most of the participants, 86.13% (149), identify stressful situations in daily life, but most of them, 76.88% (133), also knew how to relieve tension through either taking time out to relax and recover (85.55% or 148) or by participating in enjoyable activities (89.60% or 155).

Most participants did not smoke or use tobacco products (80.92% or 140), use alcohol above the legal limit (85.55% or 148), abuse drugs (93.06% or 161) or abuse over-the-counter prescription medication (89.06% or 155).

The majority of participants practised safe sex (91.91% or 159) by either abstaining or limiting sex to a safe partner (94.80% or 164). This safety consciousness also extended to driving, where most participants preferred using a seat belt (90.17% or 156) and not exceeding the speed limit (76.88% or 133). Moreover, most were trained in first aid (70.52% or 122) and felt competent to perform first aid in an emergency (70.52% or 122) or at the very least able to call emergency (medical) help (94.80% or 164).

The participants were generally health conscious by brushing their teeth at least twice daily (96.53% or 167), got enough sleep (80.23% or 138), sought medical help when needed (89.60% or 155) and followed the medical advice or medication as prescribed (95.38% or 165).

Even though most of the participants, 78.61% (136), preferred to use products only shown to be effective by research, only 24.28% (42) made the effort to read the labels of products.

The majority of participants, 91.91% (159), expressed concerns about "global warming" and many, 88.44% (153), saw the need to conserve energy, unfortunately, only 9.25% (16) were committed to recycling waste.

Most participants expressed positive sentiments relating to a sense of purpose in life (97.69% or 169), as well as that of the country's future (86.13% or 149), which was rather encouraging.
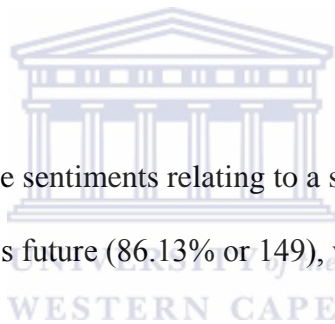
4.2.2.6   Dietary Intake

Measures of dietary habits were collected from data on the intake of energy and nutrients calculated from the food record sheets (food diaries) completed by subjects. At baseline, 79.77% (138) of the participants did not return their food diaries, while at the follow-up assessment 85.22% (98) of the participants did not hand in their food diaries. Consequently, too little dietary information was gathered for the group analysis, hence, no dietary data is reported in the study.

4.2.2.7 Physical Activity

The guidelines for data processing and analysis of the international physical activity questionnaire (IPAQ) developed by the IPAQ research committee were followed for this section of the study (IPAQ, 2005). Only physical activity lasting 10 minutes or more was included in the calculation of summary scores. The scientific rationale for this was that only physical activity lasting for a minimum of 10 minutes produces health benefits. Therefore, responses of less than 10 minutes were excluded from the analysis.

The response rate for the IPAQ questionnaire was 29.48% (51). Table 4.12 contains the results of the daily physical activity. The GG showed a mean weekly energy expenditure of 574.61±461.80 MET·min·wk^{-1}, with the lowest expenditure in the MG with 480.71±370.04 MET·min·wk^{-1}, followed by the DG with 499.55±354.21 MET·min·wk^{-1}, then the EG with 564.38±448.78 MET·min·wk^{-1}, and the CG with 564.77±339.17 MET·min·wk^{-1}, and the highest expenditure in the HIG with 907.50±827.17 MET·min·wk^{-1}. The minimum weekly energy expenditure was 70 MET·min·wk^{-1} in the CG, and the maximum weekly energy expenditure was 2625 MET·min·wk^{-1} in the HIG. Most of the subjects, 70.59% (36) did not reach the recommended 30 minutes of moderate intensity activity (3.0 – 5.9 METs), on 4 or more days per week, and were typical of a sedentary population. The MG was the only group at baseline to show a mean weekly physical activity score of 480.71 MET·min·wk^{-1} that was below the minimum 500 MET·min·wk^{-1} recommended for health benefits. For most of the subjects, 88.25% (45) physical activity usually occurred during their leisure time as opposed to being a part of their academic programme.

Table 4.12. Mean weekly energy expenditure at pre-test.

Groups	N	Mean (MET·min·wk^{-1})	SD	Minimum (MET·min·wk^{-1})	Maximum (MET·min·wk^{-1})
Grand	51	574.61	481.80	70.00	2625.00
Control	11	564.77	339.17	70.00	1260.00
Health Information	7	907.50	827.17	157.50	2625.00
Diet	11	499.55	354.21	140.00	1260.00
Exercise	8	564.38	448.78	210.00	1575.00
Multiple	14	480.71	370.04	210.00	1680.00

4.3   Intervention Period
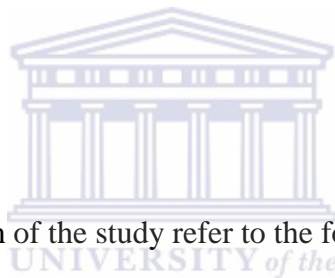
The results presented in this section of the study refer to the following components, namely: attrition or drop out of subjects from the study, subject adherence to or compliance with treatment, and adverse events arising from participation of subjects in one or more treatments.

4.3.1   Phase 5: Implementation of the Intervention

During the implementation of the intervention programme the subjects were reminded of the S.M.A.R.T goals for each treatment group, and support was provided by trained research assistants through the biokinetics clinic.

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All the subjects were encouraged to keep a record or report changes to their daily physical activity and dietary habits, especially changes implemented outside of the study, such as sports practices and competitions over weekends.

Subjects were also encouraged to consult with the research assistants should they experience added stress from the intervention programme or feel that they required professional support.

4.3.2 Attrition from the Study

Fewer subjects returned for the follow-up assessments after four months (Table 4.13). From the original 173 subjects who began the study, there was a loss of 58 (33.33%) subjects during the intervention period, so that only 115 (66.47%) subjects completed the follow-up assessment. The loss of subjects affected all the groups, so that the change in group size from pre- to post-test was as follows: in the CG from 37 to 30 subjects, in the HIG from 36 to 23 subjects, in the DG from 37 to 19 subjects, in the EG from 34 to 21 subjects, and in the MG from 29 to 22 subjects.

Overall, the attrition rate was highest in the DG with the loss of 18 (10.40%) subjects, followed by the EG and the HIG with 13 (7.51%) subjects each, and MG and the CG with 7 (4.05%) subjects each. However, the loss of subjects was felt to a much greater extent within the various treatment groups where the DG experience the loss of almost half its subjects, 49% (18), and dropped from being the largest group at pre-test (37) to being the smallest group at post-test(19). The other groups also experienced substantial subject drop-out with the losses varying from 38.24% (13) in the EG, 36.11% (13) in the HIG, 24.14% (7) in the MG, to 18.92% (7) in the CG.

Several reasons were presented by subjects to account for the variable dropout rate that ranged from heavy academic workloads, insufficient time, non-compliance, a lack of support, poor self-control, to a lack of motivation because of minimal or no visible improvement in the short term. Some of the subjects also "dropped out of university", and accounted for those who did not respond.

Table 4.13. Affect of attrition on group size at pre- and post-test.

Groups	Pre-test		Post-test		Drop out		Attrition Rate
	(n)	%	(n)	%	(n)	%	(%)
Grand	173	100	115	100	58	33.53	33.53
Control	37	21.39	30	26.09	7	18.92	4.05
Health Information	36	20.81	23	20.00	13	36.11	7.51
Diet	37	21.39	19	16.52	18	48.65	10.40
Exercise	34	19.65	21	18.26	13	38.24	7.51
Multiple	29	16.76	22	19.13	7	24.14	4.05

Failure to complete the intervention programme was not related to age, gender, race, obesity or family history of CHD. Participants lost to follow-up were mostly sedentary, more likely to be smokers. Participants who were sedentary and smoked tended to drop out more from the experimental (diet, exercise and health information) than control group.

4.3.3 Compliance

The overall compliance rate in the four experimental groups was highest in the HIG with 46.70%, followed by the DG and the MG at 35.30% and 31.55%, respectively, with the exercise group having the lowest compliance with 9.74% (Figure 4.4). Poor compliance in the experimental groups was greatest during the first four weeks of the study, attributed mainly to subjects not wanting to undergo lifestyle change in addition to their academic commitments, such as, settling into academic life, attending extra academic classes, tutorials, practicals/laboratory sessions, and extensive travelling to and from the university campus. Contributory factors impacting upon subject adherence were ill health, heavy academic workloads (preparation for classes, tests, assignments and other continuous assessments), or stress arising from a lack of suitable accommodation due to relocation from another geographic region (province).

4.3.4 Adverse Events

Except for the occasional delayed onset muscle soreness, no adverse incidents, such as musculo-skeletal injuries, were associated with the exercise testing and training intervention. However, minor disruptions were encountered in the biokinetics clinic when subjects did not adhere to their set appointments and turned up outside of their scheduled exercise times.
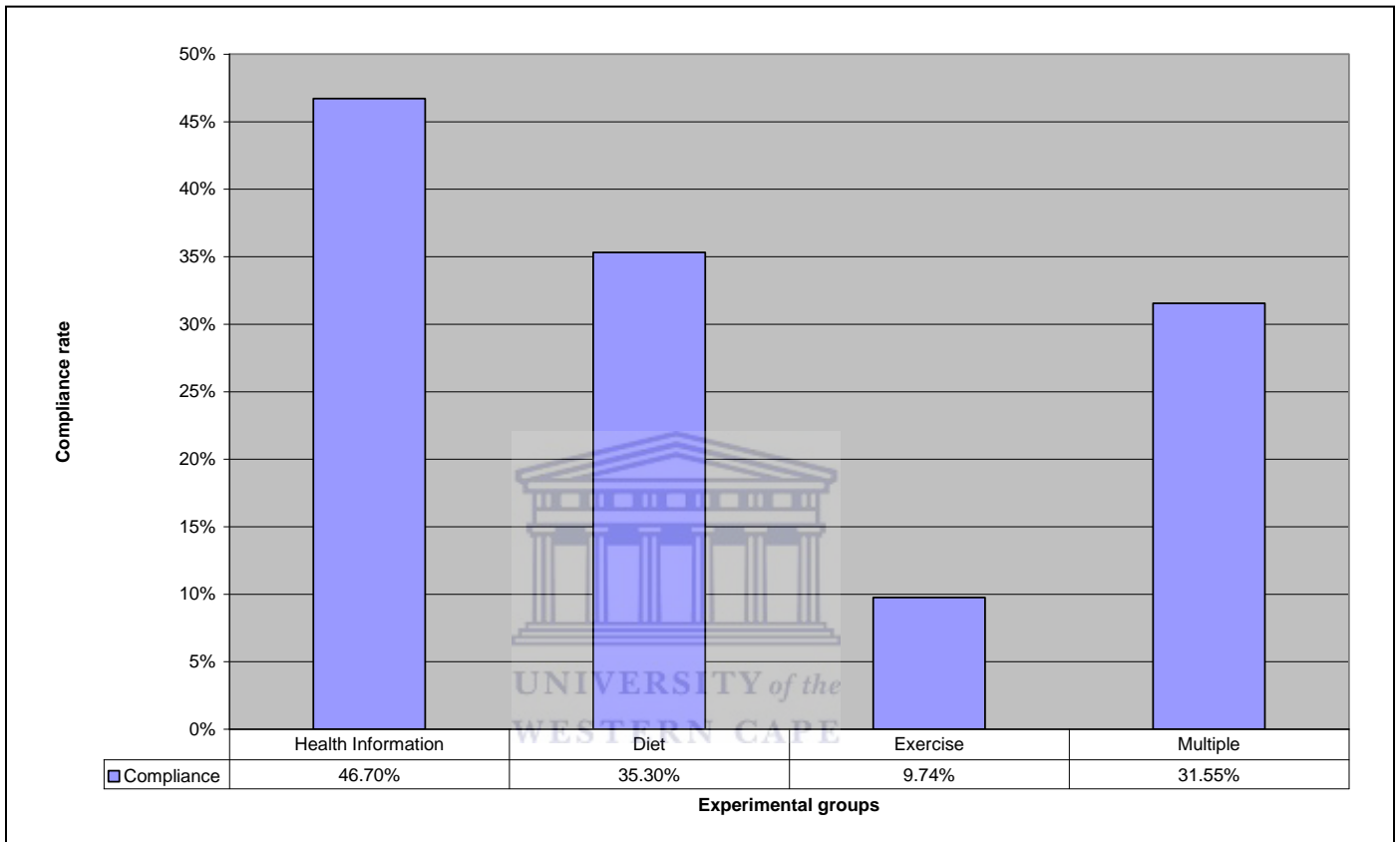
Figure 4.4. Compliance rate of the subjects.

4.4   Post-Test (Follow-up) Period

The gender composition of the study sample changed slightly from the pre- to post-test period
with a marginally higher attrition rate coming from males (40.54%) when compared to the
females (31. 62%). However, the racial distribution of the study sample at follow-up remained
essentially unchanged from the pre-test period.

4.4.1 Phase 6: Post-test Results

The results presented in this section of the study refer to the following factors, namely, CHD risk, HRPF, HRB and physical activity.

The post-intervention changes in non-modifiable CHD risk factors, such as age and family history of CHD, were not significant, and occurred primarily as a result of the attrition of subjects from the study.
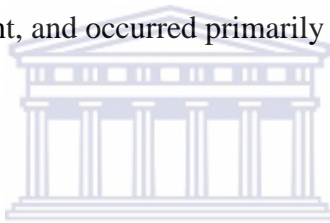
Table 4.14 contains the results for the stages of readiness from pre- to post-test. In the GG, most of the subjects were in the contemplative stage at pre-test, 68.21%, whereas at post-test most of the subjects shifted to the preparatory stage, 41.74%, and the action stage, 25.22%. Changes to the other stages at post-test in this group were not noteworthy and appeared to be largely as a result of subject attrition.

In the treatment groups the changes as post-test tended to follow those in the GG. In the CG, subjects in the preparatory stage increased by 33.16% largely due to subjects coming from the contemplative stage that decreased by 30.37%. Also noteworthy in this group was that 10.63% of the subjects fell into relapse at post-test, unlike the other treatment groups.

In the HIG, the increase in subjects at post-test were in the preparatory and action stages by 15.34% and 16.18%, respectively, came primarily from the decline in subjects in the contemplative stage by 25.96%. In the DG, primarily the preparatory stage increased in subjects

Table 4.14. Comparison of the stages of readiness to change at pre- and post-test.

Groups		PCS	%	CS	%	PS	%	AC	%	MS	%	RS	%	Total
Grand	**Pre**	3	1.73	118	68.21	34	19.65	12	6.94	2	1.16	4	2.31	173
	Post	0	0.00	33	28.70	48	41.74	29	25.22	0	0.00	5	4.35	115
	Pre-Post	3	1.73	85	39.51	-14	-22.09	-17	-18.28	2	1.16	-1	-2.04	58
Control	**Pre**	1	2.70	26	70.27	5	13.51	4	10.81	0	0.00	1	2.70	37
	Post	0	0.00	12	40.00	14	46.67	0	0.00	0	0.00	4	13.33	30
	Pre-Post	1	2.70	14	30.27	-9	-33.16	4	10.81	0	0.00	-3	-10.63	7
Health Information	**Pre**	1	2.78	25	69.44	7	19.44	2	5.56	0	0.00	1	2.78	36
	Post	0	0.00	10	43.48	8	34.78	5	21.74	0	0.00	0	0.00	23
	Pre-Post	1	2.78	15	25.96	-1	-15.34	-3	-16.18	0	0.00	1	2.78	13
Diet	**Pre**	0	0.00	26	70.27	6	16.22	3	8.11	0	0.00	2	5.41	37
	Post	0	0.00	5	26.32	12	63.16	1	5.26	0	0.00	1	5.26	19
	Pre-Post	0	0.00	21	43.95	-6	-46.94	2	2.85	0	0.00	1	0.15	18
Exercise	**Pre**	1	2.94	25	73.53	6	17.65	1	2.94	1	2.94	0	0.00	34
	Post	0	0.00	4	19.05	7	33.33	10	47.62	0	0.00	0	0.00	22
	Pre-Post	1	2.94	21	54.48	-1	-15.68	-9	-44.68	1	2.94	0	0.00	12
Multiple	**Pre**	0	0.00	16	55.17	10	34.48	2	6.90	1	3.45	0	0.00	29
	Post	0	0.00	2	9.09	7	31.82	13	59.09	0	0.00	0	0.00	22
	Pre-Post	0	0.00	14	46.08	3	2.66	-11	-52.19	1	3.45	0	0.00	7

Key: PCS = precontemplation stage; CS = contemplation stage; PS = preparation stage; AC = action stage; MS = maintenance stage; RS = relapse stage

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by 46.94% at post-test, that came mainly from subjects in the contemplative stage that decreased by 43.95%.

In the EG, but especially the MG, the largest change came in the action stage at post-test by 44.68% and 52.19%, respectively, with the subjects coming mainly from the contemplative stage in each group accordingly. The EG also showed an additional improvement in subjects in the preparatory stage by 15.68% at post-test.

4.4.1.1 CHD Risk Factors

As shown in Table 4.15, significant improvements in anthropometric characteristics were observed, specifically for body mass, body mass index, and waist and hip circumferences that were consistent in the MG than in the other experimental groups. Measurements at post-test in the CG, in contrast, remained basically unchanged or deteriorated.

The decrease in body mass was statistically significant (p range = .020 - .0005) in the HIG, DG, and MG when compared to the CG, but not in the EG. The decrease in body mass in the MG remained significant when compared to the DG and EG as well ($p < .05$). Body mass index (BMI), which is closely related to body mass, proved to be statistically significant ($p < .0005$), but only between the CG and MG.

Except for the EG, waist circumference differed significantly (p range = .026 – .001) between the CG and the HIF, DG and MG. For hip circumference, the CG differed significantly (p range =

Table 4.15. Comparison of the physical characteristics and CHD risk factors of the subjects at pre- and post-test.

Physical Characteristics and CHD risk factors		Grand Group (mean±SD) (Pre = 173) (Post =115)	Control Group (mean±SD) (Pre = 37) (Post = 30)	Health Information Group (mean±SD) (Pre n = 36) (Post n = 23)	Diet Group (mean±SD) (Pre n = 37) (Post n = 19)	Exercise Group (mean±SD) (Pre n = 34) (Post n = 21)	Multiple Group (mean±SD) (Pre n = 29) (Post n = 22)	p-value (Between Groups)
Age	Pre	21.35±6.24	23.92±9.14	21.50±6.36	20.57±3.83	19.68±3.54	20.86±5.88	.066
(years)	Post	22.06±6.86	24.70±9.52	22.17±7.00	20.42±3.42	20.38±3.81[a3]	21.36±6.29	
Family History	Pre	40.37±49.22	47.06±50.66	46.88±50.70	37.84±49.17	33.33±47.95	35.71±48.80	.281
of CHD (%)	Post	35.85±48.18	50.00±50.92	31.58±47.76	31.58±47.76	22.22±47.78	36.36±49.24	
Height	Pre	162.73±8.49	163.91±8.39	162.34±8.44	162.71±8.41	162.39±9.65	162.15±7.79	.299
(cm)	Post	161.96±8.08	164.32±8.29	160.63±7.71	161.56±7.84	160.50±8.78	161.86±7.70	
Body Mass	Pre	76.38±19.24	77.80±18.61	71.78±15.69	79.32±21.02	75.89±19.79	77.10±21.19	.0005
(kg)	Post	75.80±18.10	77.44±18.29	71.77±13.07[a2]	78.71±20.83[a1]	76.61±18.78	74.50±19.91[a5]	
BMI	Pre	28.92±7.30	29.25±8.15	27.43±6.49	29.74±6.45	28.86±7.27	29.40±8.31	.0005
(kg·m^{-2})	Post	28.92±6.73	28.83±7.28	27.88±5.14	29.84±5.71	29.85±7.46	28.46±7.79[a5]	
Waist	Pre	82.21±14.85	83.97±17.05	80.09±11.69	83.72±13.97	82.07±15.99	80.86±15.58	.005
Circumference (cm)	Post	81.63±14.05	83.41±15.36	79.86±11.13[a4]	82.96±13.78[a1]	83.10±15.37	78.53±14.35[a3, c1, d1]	
Hip	Pre	106.15±13.91	106.94±15.07	103.37±11.61	107.92±13.35	105.31±13.23	107.28±16.62	.001
Circumference (cm)	Post	105.34±13.67	107.04±14.31	102.97±8.33[a1]	107.38±14.21[a2]	104.42±15.46[a2]	104.64±15.59[a5]	
Waist-Hip	Pre	0.77±0.09	0.78±0.10	0.77±0.07	0.77±0.07	0.78±0.12	0.75±0.06	.044
Ratio	Post	0.77±0.09	0.78±0.08	0.77±0.07[a1]	0.77±0.07	0.80±0.14	0.75±0.05	
Resting	Pre	78.42±10.73	77.11±11.11	79.14±7.51	80.46±13.63	78.38±9.88	76.62±10.56	.0005
Heart Rate (bpm)	Post	77.24±10.81	80.57±11.21	75.30±6.20[a4]	82.00±13.78	76.81±10.06[a2]	71.00±8.93[a5, c2, d1]	

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Systolic	**Pre**	123.40±12.05	122.70±11.23	122.39±15.13	124.54±10.98	123.47±11.12	124.00±11.77	.005
BP (mm Hg)	**Post**	122.85±8.98	123.93±11.23	123.83±7.33	125.26±9.89	121.81±7.64	119.27±6.75[a3]	
Diastolic	**Pre**	80.45±9.55	81.49±10.28	77.36±9.84	80.87±8.24	81.59±8.27	81.10±10.95	.0005
BP (mm Hg)	**Post**	80.45±6.22	82.00±6.64	79.83±5.36	83.05±6.12	79.91±6.62[a1]	77.27±4.96[a5, b4, c1, d4]	
Cigarette Smoking	**Pre**	2.72±4.03	2.46±3.00	2.53±3.60	3.38±4.99	2.41±4.19	2.83±4.31	.001
(cigarettes per day	**Post**	1.97±3.06	3.10±3.37	1.30±1.58[a1]	2.11±3.54[a2]	1.24±2.98[a1]	1.68±3.24[a5]	
Sedentary	**Pre**	91.95±27.40	89.24±31.50	94.40±23.20	91.90±27.70	94.14±23.97	89.74±31.94	.0005
Lifestyle (%)	**Post**	75.71±43.15	96.70±18.30	82.63±38.82[a1]	100.00±0.00	52.47±51.29[a5, c5]	40.95±50.31[a5, b3, c5]	

.20 – .0005) with all the groups, that is, HIG, DG, EG and MG. However, only the HIG produced statistically significant results (p < .05) for waist-hip ratio when compared to the CG.

With the exception of the DG, resting heart rate decreased significantly (p range = .009 – .0005) in the HIG, EG and MG when compared to the CG. Systolic blood pressure was only significantly different (p < .005) between the CG and MG. Diastolic blood pressure decreased significantly (p < .05) in the EG and MG when compared to the CG.

Cigarette smoking decreased significantly (p range = 0.033 – 0.0005) in all the experimental groups compared to the controls. Except for the DG, the HIG, EG and MG all adopted a less sedentary lifestyle, that were all significant (p range = .047 – .0005) when compared to the CG. As shown in Table 4.16, age was the only variable that differed significantly within all the groups between pre- and post-test (p range = 0.005 – 0.001). The EG showed significant within group differences for a sedentary lifestyle only (p = .001), and the HIG for resting heart rate (p = 0.029), and waist circumference (p = 0.015). The MG showed significant within group differences for all nine other variables, namely: body mass (p = .0005), BMI (p = .0005), waist circumference (p = .015), hip circumference (p = .001), resting heart rate (p = .029), resting systolic blood pressure (p = .002), resting diastolic blood pressure (p = .0005) smoking (p = .009), and a sedentary lifestyle (p = .0005)

Table 4. 16.   Within group differences in CHD risk factors at pre- and post-test.

Variables	Group	Mean Difference (Pre - Post) (absolute)	Mean Difference (Pre - Post) (%)	p-value (Within Groups)
Age (years)	Health Information	-0.67	-3.02	.005
	Diet	0.15	0.73	.005
	Exercise	-0.70	-3.44	.001
	Multiple	-0.50	-2.34	.001
Body Mass (kg)	Multiple	2.60	3.37	.0005
BMI (kg·m^{-2})	Multiple	0.94	3.20	.0005
Waist Circumference (cm)	Health Information	0.23	0.29	.015
	Multiple	2.33	2.97	.001
Hip Circumference (cm)	Multiple	2.64	2.46	.001
Resting Heart Rate (bpm)	Health Information	3.84	4.85	.029
	Multiple	5.62	7.33	.0005
Resting SBP (mm Hg)	Multiple	4.73	3.82	.002
Resting DBP (mm Hg)	Multiple	3.83	4.72	.0005
Smoking (cpd)	Multiple	1.15	40.64	.009
Sedentary Lifestyle (%)	Exercise	41.67	44.26	.001
	Multiple	48.79	54.37	.0005

Key: SBP = systolic blood pressure; BMI = body mass index; systolic blood pressure; DBP = diastolic blood pressure; cpd = cigarettes per day

Table 4.17 contains the results of the blood biochemistry between pre- and post-test. For all the

variables on blood biochemistry, there were no significant changes between groups at post-test.

Total cholesterol concentration decreased in the HIG, DG and EG, and increased in the MG and

CG. HDL cholesterol concentration, in contrast, increased in all the groups at post-test,

especially in the EG and MG.

A decrease in LDL cholesterol concentration was observed for the HIG, DG, EG and MG at post-test, whereas the CG had an increase.

Plasma triglyceride levels decreased only in the HIG and DG at post-test, with the EG and MG remaining essentially unchanged, and the CG showing an increase.

For impaired fasting glucose, the concentrations decreased in all the groups at post-test, including the CG.

Overall, the improvement in lipoprotein levels was better demonstrated when these indices were depicted as ratios, namely, TC:HDL, LDL:HDL and trig:HDL. There were noticeable reductions for each of these ratios for all the experimental groups, that is, HIG, DG, EG and MG, whereas the CG, in contrast, showed consistent increases for all these ratios, but none were significant.

The only significant within group differences from pre- to post-test were shown for HDL cholesterol, and in the EG and MG only (p < .01).

Table 4.17. Comparison of the blood biochemical results of the subjects at pre- and post-test.

Blood Biochemical Variables		Grand Group (mean±SD) (Pre = 147) (Post = 86)	Control Group (mean±SD) (Pre = 37) (Post = 24)	Health Information Group (mean±SD) (Pre = 26) (Post = 11)	Diet Group (mean±SD) (Pre = 36) (Post = 16)	Exercise Group (mean±SD) (Pre = 24) (Post = 17)	Multiple Group (mean±SD) (Pre = 24) (Post = 18)	p value (Between Groups)
Total Cholesterol (TC)	Pre	4.28±0.78	4.22±0.87	4.35±1.07	4.28±0.71	4.37±0.65	4.24±0.51	.130
$(mmol^.L^{-1})$	Post	4.24±0.64	4.54±0.72	4.03±0.81	3.90±0.47	4.15±0.58	4.33±0.41	
HDL Cholesterol	Pre	1.20±0.26	1.14±0.26	1.17±0.22	1.22±0.26	1.19±0.27	1.29±0.26	.104
$(mmol^.L^{-1})$	Post	1.32±0.29	1.19±0.26	1.18±0.19	1.26±0.21	1.39±0.24*	1.57±0.34*	
LDL Cholesterol	Pre	2.61±0.73	2.58±0.78	2.69±0.98	2.60±0.65	2.69±0.58	2.51±0.59	.370
$(mmol^.L^{-1})$	Post	2.41±0.69	2.71±0.84	2.12±0.80	2.36±0.42	2.31±0.64	2.35±0.54	
Triglyceride	Pre	0.82±0.45	0.75±0.33	1.01±0.60	0.82±0.53	0.77±0.24	0.78±0.44	.070
$(mmol^.L^{-1})$	Post	0.79±0.30	0.86±0.40	0.78±0.19	0.73±0.19	0.78±0.18	0.78±0.36	
Impaired Fasting	Pre	4.42±0.44	4.44±0.50	4.52±0.38	4.28±0.43	4.42±0.26	4.53±0.53	.277
Glucose $(mmol^.L^{-1})$	Post	4.33±0.44	4.36±0.43	4.47±0.31	4.28±0.41	4.31±0.49	4.26±0.52	
TC/HDL	Pre	3.78±1.31	3.97±1.65	3.91±1.47	3.93±1.32	3.83±0.91	3.40±0.72	.542
Ratio	Post	3.43±1.37	4.20±2.10	3.48±0.84	3.17±0.68	3.05±0.65	2.94±0.92	
LDL/HDL	Pre	2.34±1.04	2.46±1.27	2.45±1.21	2.28±0.96	2.40±0.84	2.04±0.70	.634
Ratio	Post	1.98±1.12	2.56±1.74	1.86±0.86	1.92±0.48	1.70±0.59	1.62±0.73	
Triglyceride/HDL	Pre	0.75±0.55	0.71±0.41	0.94±0.67	0.77±0.78	0.69±0.28	0.61±0.30	.064
Ratio	Post	0.64±0.31	0.78±0.44	0.69±0.23	0.59±0.15	0.59±0.22	0.52±0.25	

Key: LDL = low density lipoprotein; HDL = high density lipoprotein
*indicates significant within group differences (p < .01)

4.4.1.1.1   Prevalence of CHD Risk Factors

Table 4.18 presents the prevalence of CHD risk factors at pre- and post-test. The prevalence of CHD risk factors at post-test followed the same pattern as that at pre-test and ranged from the lowest prevalence for impaired fasting glucose to the highest for a sedentary lifestyle. The difference in prevalence between pre- and post –test showed a percentage decline for all CHD risk factors from impaired fasting glucose to a sedentary lifestyle. The CHD risk factors showing a substantial decrease at post-test were dyslipidemia with 20.37%, hypertension with 16.73%, a sedentary lifestyle with 15.39% and, to a lesser extent, cigarette smoking with 10.57%. The changes for the other CHD risk factors at post-test, though positive, were not noteworthy.

Table 4.18.   Prevalence of CHD risk factors at pre- and post-test.

CHD Risk Factors	Pre-Test			Post-Test			Difference
	Number of subjects	Total number of subjects tested	%	Number of subjects	Total number of subjects tested	%	Pre-Post (%)
Impaired fasting glucose	2	147	1.36	1	86	1.16	0.20
Hypertension	47	173	27.17	12	115	10.44	16.73
Dyslipidemia	59	147	40.14	17	86	19.77	20.37
Family history of CHD	65	161	40.37	38	105	36.16	4.21
Obesity	73	173	42.20	46	115	40.00	2.20
Smoking	92	173	53.18	49	115	42.61	10.57
Sedentary lifestyle	159	173	91.91	88	115	76.52	15.39

Figure 4.5 shows the prevalence of CHD risk factors between pre-and post-test. The prevalence rates were similar for each of the CHD risk factors at pre- and post-test, with the exception being

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lower proportions at post-test. Clearly, a sedentary lifestyle stands out as the most prevalent

CHD risk factor both at pre- and post-test. The prevalence rates for family history of CHD,

obesity and cigarette smoking all remained in excess of 30% at post-test, primarily as a result of

the dropout effect of subjects. Dyslipidemia and hypertension were reduced the most at post-test,

with impaired fasting glucose remaining essentially unchanged from pre- to post-test.
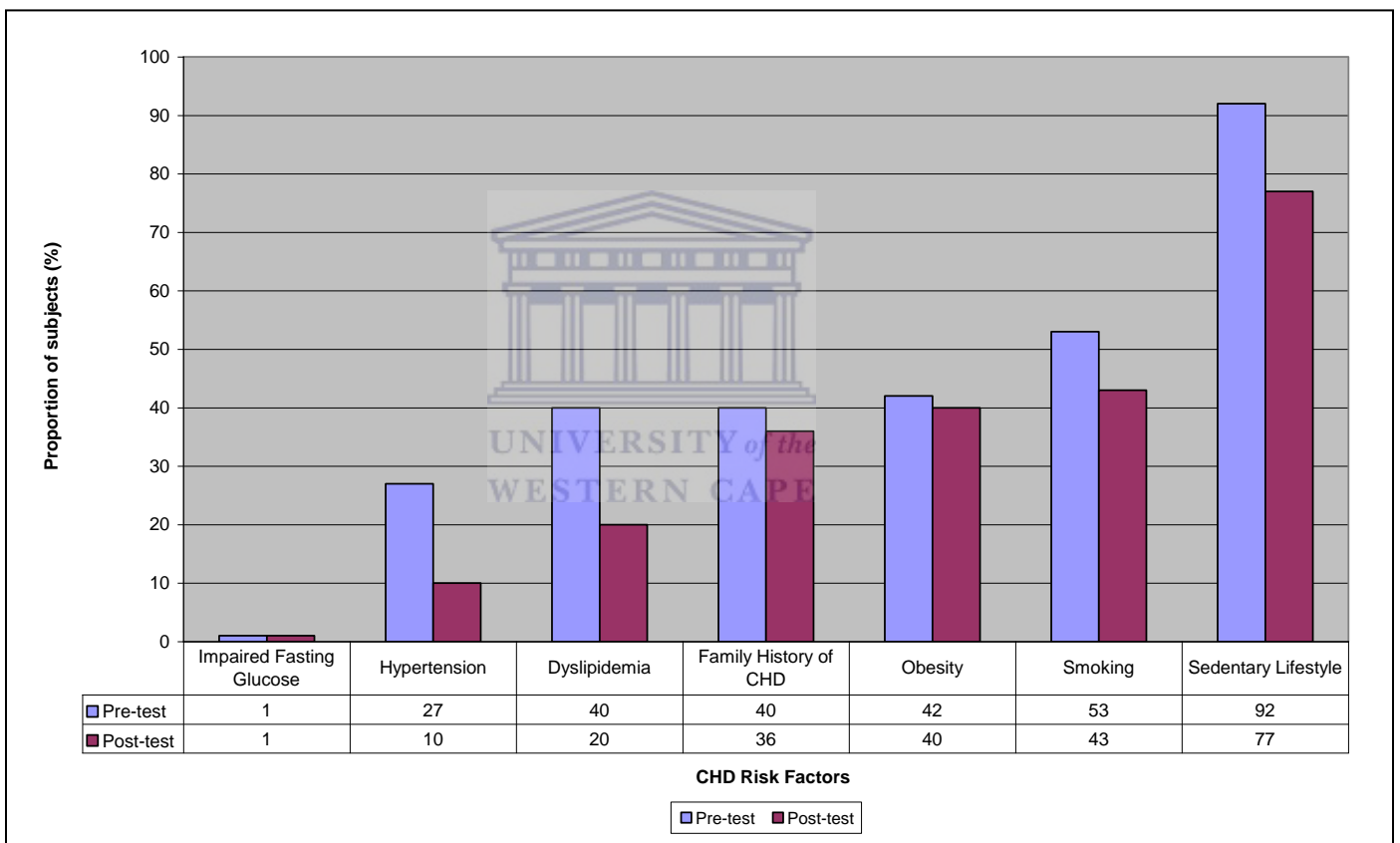
Figure 4.5. Frequency distribution of CHD risk factors at pre- and post-test.

The frequencies of CHD risk factors appearing in the subjects, that is, the number of subjects

having frequencies of two risk factors or frequencies of three risk factors and the like, is

indicated in Table 4.19. One of the noteworthy findings was the general shift in subjects from higher frequencies (≥ 2 CHD risk factors) at pre-test to lower frequencies (≤ 1 CHD risk factors) of risk at post-test. In all the frequencies ranging from two to six CHD risk factors, the proportion of subjects decreased consistently from pre- to post-test ranging from 6.96% for two CHD risk factors, 8.69% for three risk factors, 4.35% for 4 risk factors, 6.96% for five risk factors, and 0.87% for six risk factors. More especially was the shift in subjects to categories of zero and one risk factor by 6.09% and 21.74%, respectively, at post-test. At baseline, all the subjects were stratified as moderate risk having two or more CHD risk factors. However, at post-test, only 72.17% of subjects were stratified as moderate risk, with a re-stratification of subjects as low risk by 27.83%.
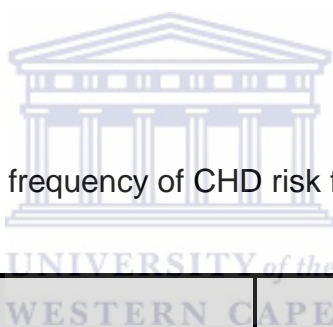
Table 4.19.   Comparison of the frequency of CHD risk factors in the subjects at pre- and post-test.

Frequencies of CHD risk factors	Pre-Test		Post-Test		Difference
	Number of subjects	%	Number of subjects	%	Pre-Post (%)
0	0	0.00	7	6.09	-6.09
1	0	0.00	25	21.74	-21.74
2	50	43.48	42	36.52	6.96
3	36	31.30	26	22.61	8.69
4	18	15.65	13	11.30	4.35
5	10	8.70	2	1.74	6.96
6	1	0.87	0	0.00	0.87
7	0	0.00	0	0.00	0.00
Total	115	100	115	100	0.00

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The frequencies of CHD risk, shown in Figures 4.6 (a-f), produced a noteworthy trend from pre- to post-test, that is, the consistent decrease in number of subjects for each frequency ranging from two to six CHD risk factors from pre- to post-test. Essentially, the subjects experienced a leftward shift from a higher to a lower frequency that is clearly illustrated in Figure 4.6(a) for the GG.

Unlike the other treatment groups that experienced a distinct leftward shift in the frequencies of CHD risk factors, Figure 4.6(b) shows that the CG had a mixed response in the shift of frequencies at post-test, with some subjects moving rightward from three to four frequencies, while others moved leftward from two to a frequency of one risk factor. This mixed response was unique to the CG and was anticipated given the absence of treatment in this group.

In the HIG, the shift in frequencies of CHD risk factors was clearly leftward at post-test with subjects having fewer risk factors at post-test [Figure 4.6(c)], and likewise for the DG [Figure 4.6(d)].

The EG and MG showed the most dramatic leftward shifts in frequencies of CHD risk factors at post-test as depicted in Figures 4.6(e) and (f), respectively. Particularly noteworthy in the MG was that subjects with frequencies of four and five CHD risk factors at pre-test completely disappeared at post-test, shifting to lower frequencies. The same response also occurred in the EG, but only in subjects with a frequency of five CHD risk factors.
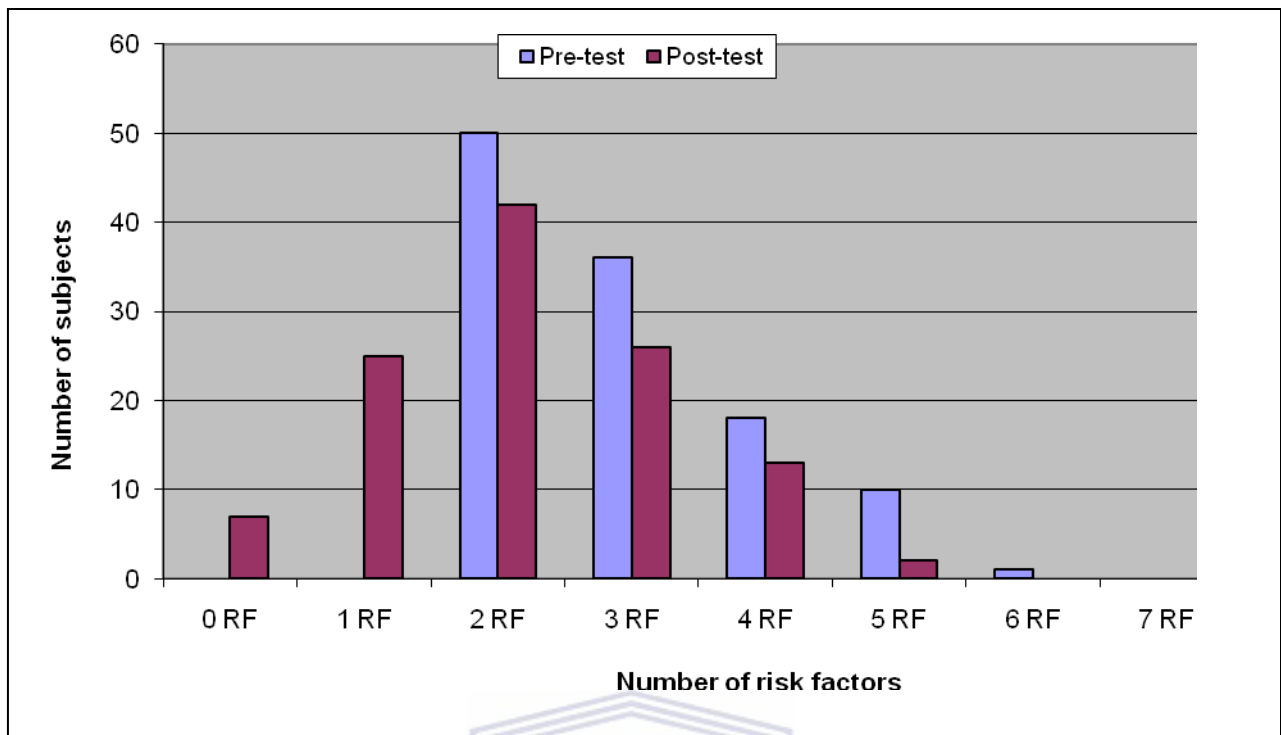
Figure 4.6 (a).   Frequency of CHD risk factors in the subjects at pre- and post-test in the grand group.
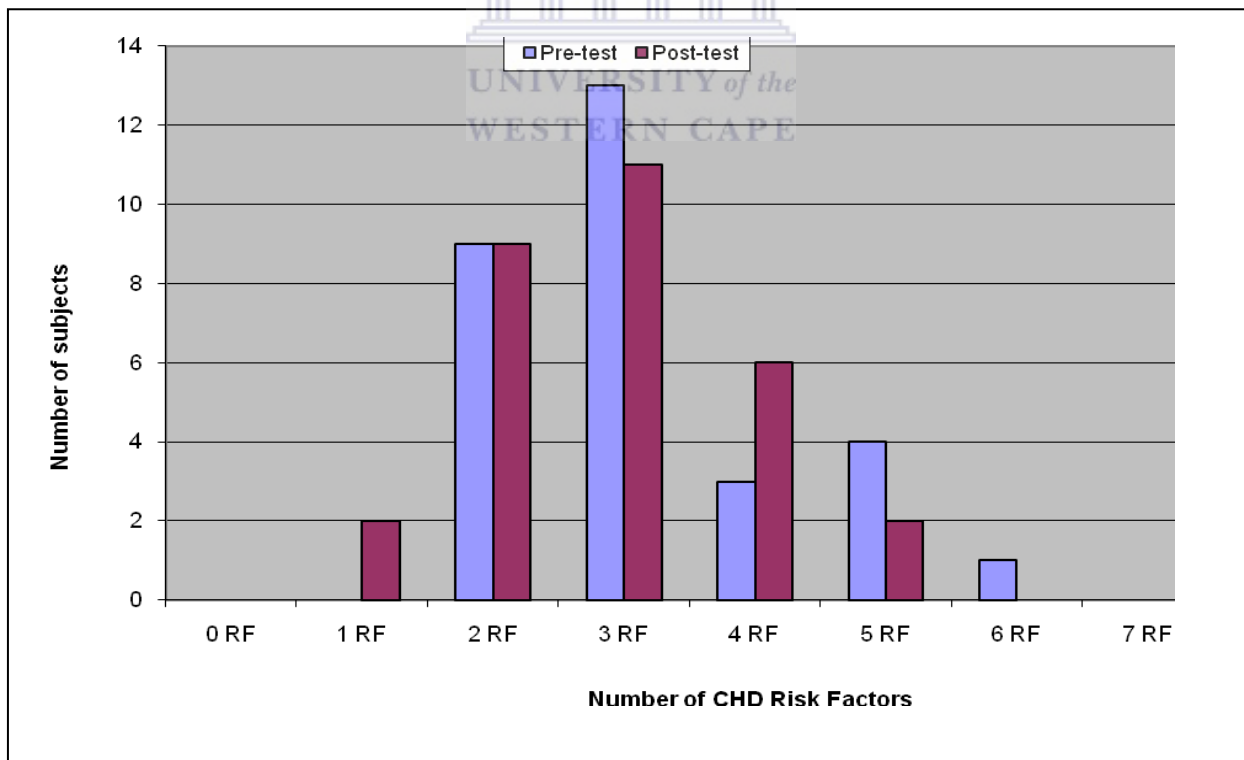
Figure 4.6 (b).   Frequency of CHD risk factors in the subjects at pre- and post-test in the control group.
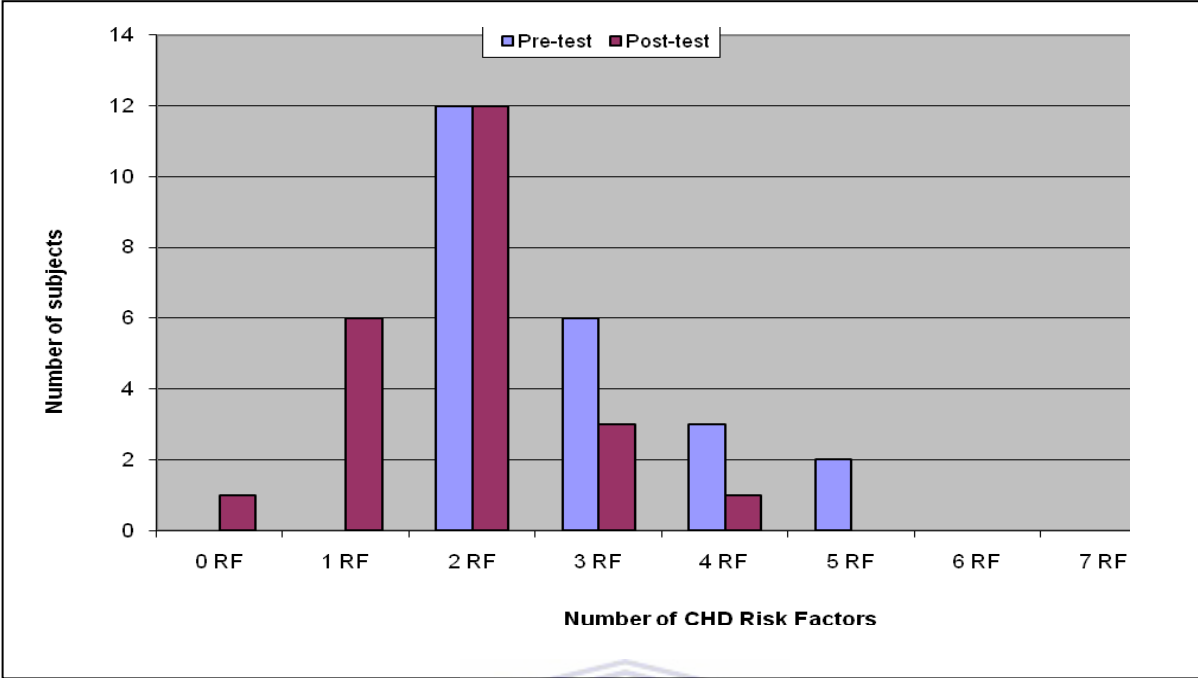
Figure 4.6 (c).   Frequency of CHD risk factors in the subjects at pre- and post-test in the health information group.
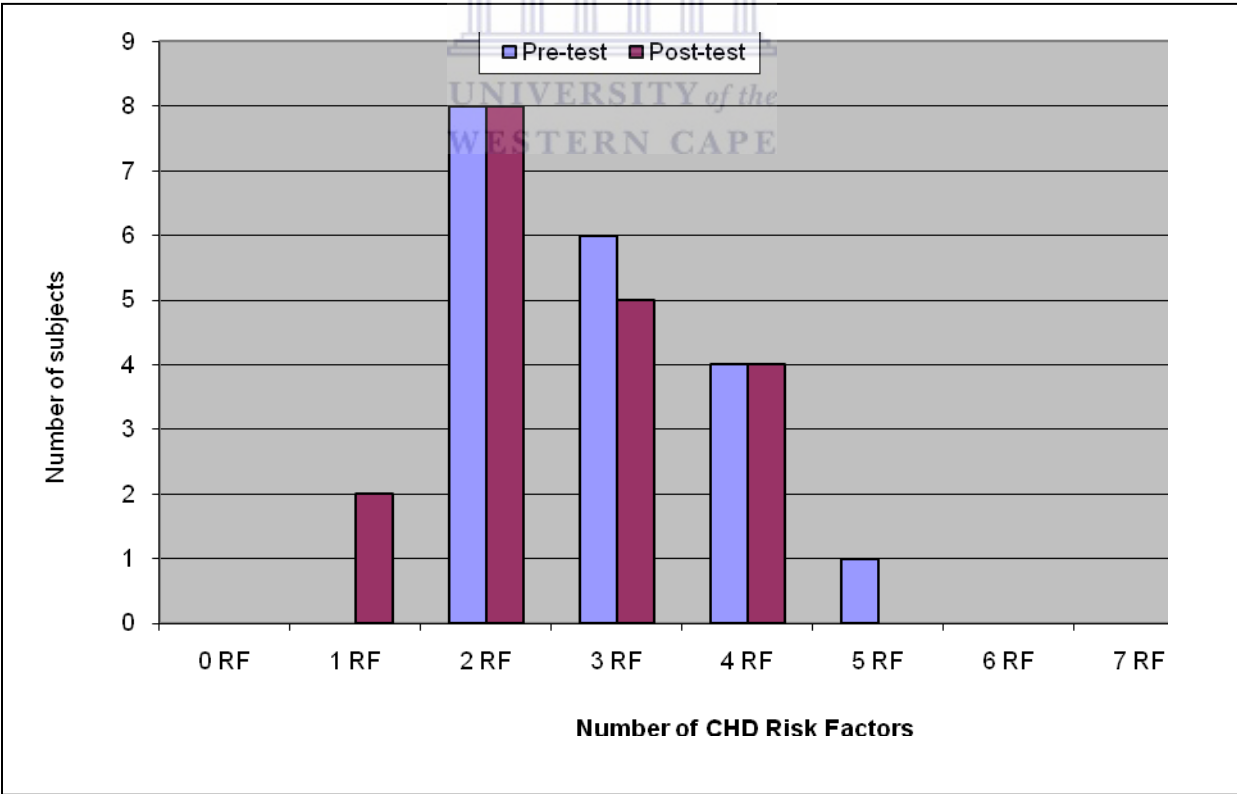
Figure 4.6 (d).   Frequency of CHD risk factors in the subjects at pre- vs post-test in the diet group.
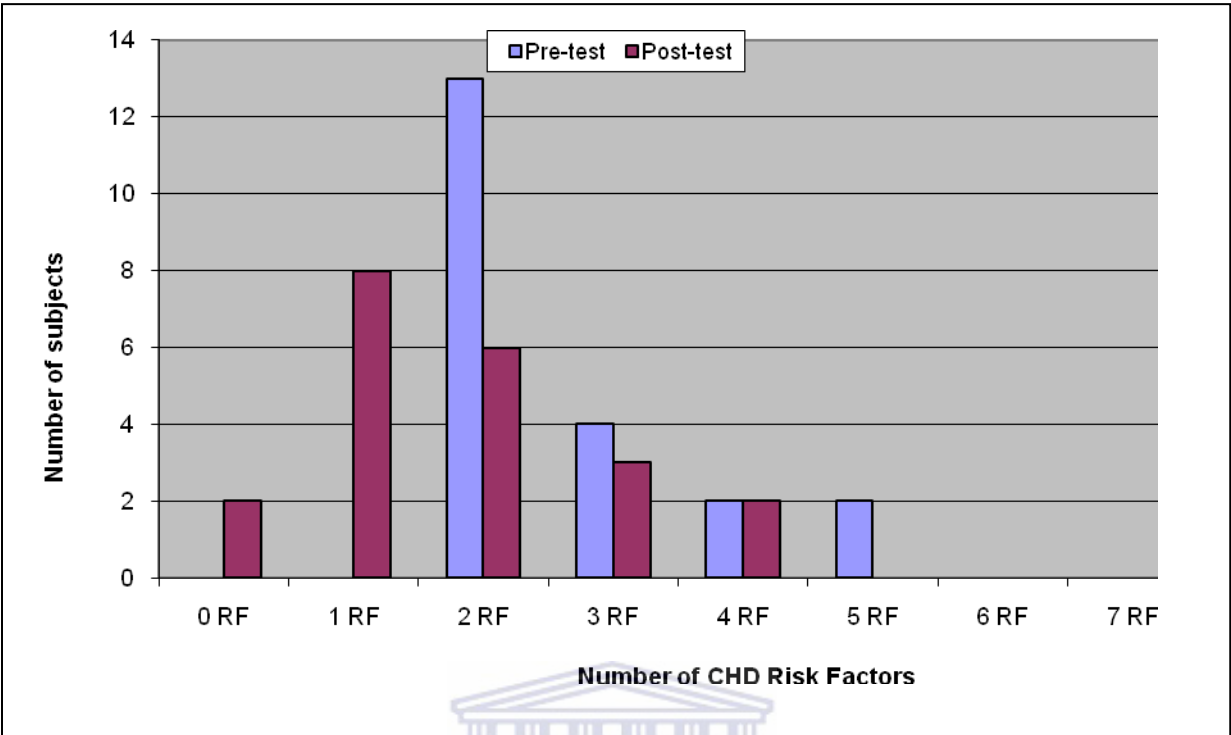
Figure 4.6 (e).   Frequency of CHD risk factors in the subjects at pre- and post-test in the exercise group.
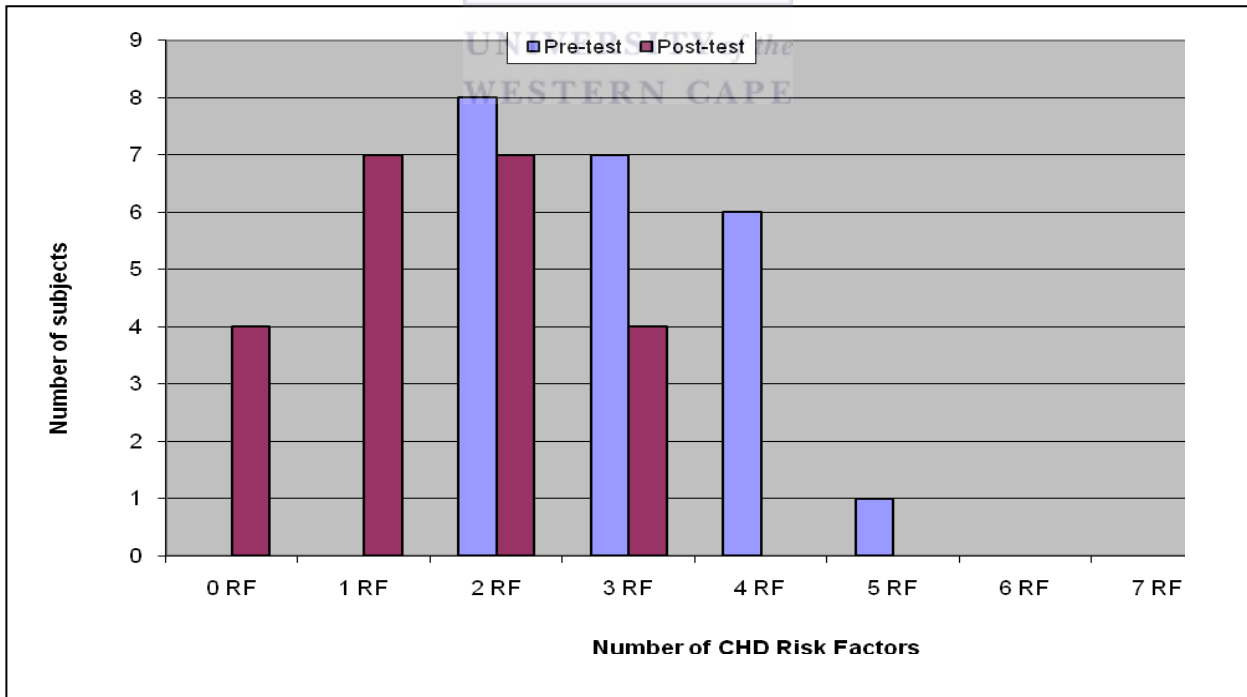
(RF = risk factor)

Figure 4.6 (f).   Frequency of CHD risk factors in the subjects at pre- and post-test in the multiple group.

The improvement in CHD risk at post-test occurred primarily as a result of the decrease in modifiable CHD risk factors, namely, dyslipidemia, hypertension, a sedentary lifestyle and, to a lesser extent, cigarette smoking (Figure 4.5).

In order to adequately address the question of which intervention programme was most effective, the following two calculations had to be performed, that is, firstly, the total number of CHD risk factors in all the subjects or cumulative risk had to quantified and, secondly, the change in CHD risk factors between the different treatments had to be calculated. The procedure followed in performing these calculations is depicted in the Table 4.20 below, and was applied to the GG comprising all the subjects at pre- and post-test.

Table 4.20. Comparison of the cumulative risk of the subjects at pre- and post-test.

Pre-Test			Post-Test			Difference	
Number of risk factors (A)	Number of subjects (B)	Cumulative risk (C) (A x B)	Number of risk factors (D)	Number of subjects (E)	Cumulative risk (F) (D x E)	(Pre – Post) (G) (C – F)	%
0	0	0	0	7	0	0	0.00
1	0	0	1	25	25	-25	0.00
2	50	100	2	42	84	16	3.27
3	36	108	3	26	78	30	7.45
4	18	72	4	13	52	20	4.46
5	10	50	5	2	10	40	10.42
6	1	6	6	0	0	6	0.29
Total	115	336	Total	115	249	87	25.89

With reference to Table 4.20, there was a total reduction of 87 CHD risk factors from pre- to post-test that is depicted as follows:

The difference between pre- and post-test:

- For two (2) risk factors, was eight (8) subjects (50 – 42)   = 16 risk factors……..(2 x 8)

- For three (3) risk factors, was ten (10) subjects (36 – 26)   = 30 risk factors……..(3 x10)

- For four (4) risk factors, was five (5) subjects (18 – 13)   = 20 risk factors……..(4 x 5)

- For five (5) risk factors, was eight (8) subjects (10 – 2)   = 40 risk factors……..(5 x 8)

- For six (6) risk factors, was one (1) subject (1 – 0)   = 6 risk factors……..(6 x 1)

   Total reduction in risk factors (post – pre)   = 112 risk factors
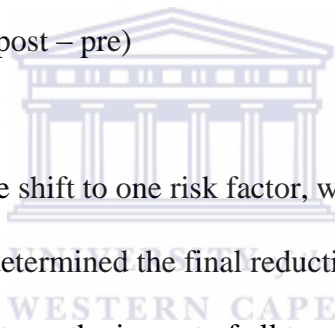
However, 25 subjects had a positive shift to one risk factor, which when subtracted from 112, gave a total of 87 risk factors that determined the final reduction in CHD risk factors in this group. When expressed as a percentage, the impact of all treatments in the intervention period resulted in an overall reduction in CHD risk by 25.89% with the largest reduction in subjects with five risk factors (10.42%), followed by subjects with three risk factors (7.45%), then subjects with four risk factors (4.46%), and subjects with two risk factors (3.27%), with the smallest reduction in the subject with six risk factors (0.29%).

The above-mentioned procedure was then applied across all the treatment groups in order to determine the change or, more specifically, the reduction in risk factors from pre- to post-treatment. Across all the treatment groups, including the CG, there was an overall decrease in CHD risk factors at post test. More specifically, the impact of the different treatments on

reducing CHD risk, expressed as a percentage, was determined as follows, in the CG it was 9.20%, in the HIG it was 24.14%, in the DG it was 6.90%, in the EG it was 21.84% and in the MG it was 37.93%. Thus, the efficacy of the different treatments ranged from the least effective being the DG with 6.90%, to the EG and HIG being equally effective with 21.84 and 24.14%, respectively), to the most effective in the MG with 37.93%.

4.4.1.2   Health-Related Physical Fitness

Table 4.21 contains the results for HRPF at pre-and post-test. Predicted maximal aerobic capacity ($\dot{V}O_2$max) decreased in all the groups from pre- to post-test, except in the MG where it increased. Predicted $\dot{V}O_2$max was significant in the MG when compared with the CG ($p <$ .0005), the HIG ($p <$ .01), the DG ($p <$ .005) and the EG ($p <$ .05). Despite the improvement in $\dot{V}O_2$max in the MG, the results remained low in all the groups at post-test.

The percent body fat followed the same trend as predicted $\dot{V}O_2$max with all the groups showing an increase in percent body fat at post-test decrease, except the MG that had a decrease. The percent body fat was significant in the MG when compared with the CG ($p <$ .0005), the HIG ($p <$ .005), the DG ($p <$ .0005) and the EG ($p <$ .05). At post-test, percent body fat for the CG, DG and EG fell into the overfat category, whereas for the HIG it was rated as obese, and in the MG as average body fat.

Because absolute body fat is the same measurement as percent body fat, but expressed differently, the findings were the same for absolute and percent body fat.

Table 4.21. Comparison of HRPF measurements of the subjects at pre- and post-test.

Health-Related Physical Fitness Measurements		Grand Group (mean±SD) (Pre =173) (Post =114)	Control Group (mean±SD) (Pre = 37) (Post = 29)	Health Information Group (mean±SD) (Pre = 36) (Post = 23)	Diet Group (mean±SD) (Pre = 37) (Post = 19)	Exercise Group (mean±SD) (Pre = 34) (Post = 21)	Multiple Group (mean±SD) (Pre = 29) (Post = 22)	p value (Between Groups)
Predicted $\dot{V}O_2$max	Pre	26.32±7.22	25.99±8.96	27.42±6.88	26.78±6.69	25.26±6.36	26.06±6.98	.000
($ml^{.}kg^{-1.}min^{-1}$)	Post	25.91±6.51	25.53±8.61	25.96±4.86	24.84±5.08	24.70±5.53	28.43±6.65[a5, b2, c3, d1]	
Relative BF	Pre	27.83±9.71	27.42±10.68	28.13±10.81	28.02±8.97	29.11±9.31	26.27±8.67	.000
(%)	Post	28.36±8.94	28.59±10.03	30.34±8.97	29.99±8.02	29.18±8.16	23.77±7.89[a5, b3, c5, d1]	
Absolute BF	Pre	22.02±10.93	22.42±12.13	20.88±10.49	22.62±9.83	22.87±10.81	21.15±11.87	.000
(kg)	Post	22.18±10.21	23.00±11.06	22.29±9.23	23.70±8.52	23.27±10.88	18.60±10.74[a5, b3, c3, d1]	
Relative LBM	Pre	72.17±9.71	72.59±10.68	71.87±10.81	71.98±8.97	70.89±9.31	73.73±8.67	.000
(%)	Post	71.64±8.94	71.41±10.03	69.66±8.97	70.01±8.02	70.82±8.16	76.23±7.89[a5, b3, c5]	
Absolute LBM	Pre	54.36±12.68	55.38±10.95	50.90±9.90	56.70±15.69	53.03±12.41	55.95±13.55	.021
(kg)	Post	53.62±11.73	54.44±11.38	49.48±8.01	55.01±16.31	53.35±10.37[a1, b1, c3]	55.90±11.96[c1]	
Handgrip	Pre	45.12±20.93	46.04±23.12	43.58±20.22	49.21±24.85	43.69±19.03	42.35±15.14	.039
Strength (kg)	Post	43.57±18.92	44.62±20.69	41.80±17.37	43.65±25.18	40.78±15.61	46.60±15.23[a3, c1]	
Sit-Ups	Pre	19.85±9.69	19.43±10.13	23.17±9.44	18.08±9.90	19.21±7.95	19.28±10.68	.000
(rpm)	Post	20.13±8.71	18.03±8.95	22.65±7.20	15.79±6.59	20.86±8.32[a4, c2]	23.32±10.13[a5, b4, c4]	
Sit-and-Reach	Pre	46.36±9.43	44.82±9.93	45.61±9.54	45.71±10.75	47.38±9.19	48.92±6.72	.353
(cm)	Post	46.23±8.83	45.93±9.07	44.52±9.05	44.05±8.77	46.95±9.33	49.59±7.45	

Key: rpm = repetitions per minute; $\dot{V}O_2$max = maximal aerobic capacity; BF = body fat; LBM = lean body mass

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Absolute body fat increased in all the groups from pre- to post-test, except in the MG where it decreased. Absolute body fat was significant in the MG when compared with the CG ($p < .0005$), the HIG ($p < .005$), the DG ($p < .005$) and the EG ($p < .05$).

Since percent body fat is the reciprocal index of percent lean body mass with regard to body composition, the reciprocal outcome to percent body fat was evident for percent LBM. Percent LBM decreased in all the groups from pre- to post-test, except in the MG where it increased. Percent LBM was significant in the MG when compared with the CG ($p < .0005$), the HIG ($p < .005$), and the DG ($p < .0005$).

Absolute LBM decreased in all the groups at post-test, except in the EG were it increased by 0.32 kg. Absolute LBM was significant in the EG, when compared to the CG ($p < .05$), the HIG ($p < .05$) and the DG ($p < .005$). The MG was significantly different to the DG only ($p < .05$).

In terms of muscular fitness, that is, muscular strength, muscular endurance and flexibility, the MG was the only group showing improvement at post-test. All the groups showed a decrease in handgrip strength at post-test, except the MG that had an increase. For handgrip strength, there was a significant difference between CG and MG ($p < .005$), and between DG and MG ($p < .05$).

For muscular endurance (sit-ups), the CG, HIG and DG had a decrease at post-test, and the EG and MG an increase. In the EG, the increase in sit-ups was significant when compared to the CG ($p < .001$) and the DG ($p < .01$), while in the MG significant differences were observed when compared to the CG ($p < .0005$), the HIG ($p < .001$) and the DG ($p < .001$).

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For flexibility, the results for the treatment groups were mixed, with the HIG, DG and EG showing a decrease at post-test, while the CG and MG had an increase, but none of the changes were significant.

Stated briefly, for HRPF, the MG showed significant improvements in four of the five components at post-test, namely, cardiorespiratory fitness, body composition, muscular strength and muscular endurance, but not for flexibility. The EG was the only other group that had a significant improvement at post-test, but specifically for muscular endurance and absolute LBM.

Table 4.22 contains a comparison of the differences within groups for HRPF between pre- and post-test. Significant within group differences were found in the EG for absolute LBM (p = .021) and sit-ups (p = .048). Significant within group differences were consistently present in the MG for predicted $\dot{V}O_2$max (p = .001), relative BF (p = .000), absolute BF (p = .000), relative LBM (p = .000), handgrip strength (p = .005) and sit-ups (p = .000).

Table 4. 22.   Comparison of within group differences in HRPF at pre- and post-test.

Variables	Group	Mean Difference (Pre - Post) (absolute)	Mean Difference (Pre - Post) (%)	p-value (Within Groups)
Pred. $\dot{V}O_2$max	Multiple	2.37	8.34	.001
Relative BF	Multiple	2.50	9.52	.000
Absolute BF	Multiple	2.55	12.06	.000
Relative LBM	Multiple	-2.50	-3.28	.000
Absolute LBM	Exercise	0.32	0.60	.021
HG Strength	Multiple	-4.25	-9.12	.005
Sit-ups	Exercise	-1.65	-7.91	.048
	Multiple	-4.04	-17.32	.000

Key: BF = body fat; LBM = lean body mass; $\dot{V}O_2$max = maximal aerobic capacity; HG = handgrip

4.4.1.3   Health-Risk Behaviours

The HRB that underwent substantial change at post-test were those related to physical activity

and nutrition, while the others remained essentially unchanged from baseline (Table 4.23).

Throughout the questionnaire, the results of the treatment groups were generally similar to that

of the GG, therefore, only the results of the GG were reported.

The number of participants who felt that they were physically active enough to keep healthy

decreased by 36.86% from 82.08% at pre-test to 45.22 % at post-test. The number of subjects

who were moderately physically active for 30 minutes at least 3 days per week decreased by

17.74% from 80.35% at pre-test to 62.61% at post-test. Similarly, there was a 25.01% decrease

in the number of subjects who were vigorously active for 20 minutes at least twice a week from

69.36% to 44.35% at pre- and post-test, respectively. There was minimal improvement in the

numbers of subjects who performed stretching at least thrice a week (4.66%), but a more substantial improvement in those engaged in muscular fitness exercises at least twice a week (29.59%). However, the number of subjects who felt that they were physically fit decreased markedly by 33.99% from 72.25% at pre-test to 38.26% at post-test.

When considering HRB relating to nutrition, the nutritional habits that remained essentially unchanged at post test were that most participants still ate at least three meals a day, a slight increase by 4.90% from baseline. Also, the foods selected were from the four basic food groups, the same as at baseline. Moreover, many participants still wanted to cut down on fat in their diet, similar to the results at baseline. The number of subjects who tried to balance their food intake in order to maintain body weight decreased by 35.71% from 79.19% at pre-test to 43.48% at post-test. This indicated far fewer subjects pre-occupied with dieting at post-test. Many of the participants, however, still felt that they were overweight at post-test (53.91%) as opposed to the number at pre-test (26.59).

The positive results on all the items for managing stress at baseline remained consistently high at post-test. However, the number of subjects who did not smoke or use tobacco products decreased by 16.88 % from 80.92% to 64.04% at pre- and post-test, respectively. This indicated an increase in smoking prevalence in the subjects at post-test. For the other destructive habits, such as alcohol, drug and over-the-counter medication use, the results were maintained high at post-test.

The high scores obtained at pre-test for practicing safe sex, adopting safety habits, knowing first aid, personal health habits and using medical advice were maintained at post-test.

Table 4.23. HRB of the subjects at pre- and post-test.

Health-risk Behaviours		Control Group (Pre = 37) (Post = 30)		Health Information Group (Pre = 36) (Post = 23)		Diet Group (Pre = 37) (Post = 19)		Exercise Group (Pre = 34) (Post = 21)		Multiple Group (Pre = 29) (Post = 22)		Grand Group (Pre = 173) (Post = 115)	
Physical Activity		No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Physically active enough	Pre	27	72.97	32	88.89	31	83.78	30	88.24	22	75.86	142	82.08
to keep healthy	Post	14	46.67	6	26.09	8	42.11	11	52.38	13	59.09	52	45.22
Physically active for 30 min.	Pre	27	72.97	29	80.56	31	83.78	30	88.24	22	75.86	139	80.35
at least 3d/wk	Post	17	56.67	12	52.17	14	73.68	13	61.90	16	72.73	72	62.61
Vigorously active for 20 min.	Pre	21	56.76	24	66.67	30	81.08	28	82.35	17	58.62	120	69.36
at least 2d/wk	Post	12	40.00	5	21.74	10	52.63	12	57.14	12	54.55	51	44.35
Stretch at least 3d/wk	Pre	6	16.22	0	0.00	5	13.51	2	5.88	3	10.34	16	9.25
	Post	6	20.00	1	4.35	1	5.26	2	9.52	6	27.27	16	13.91
Muscle fitness at least 2d/wk	Pre	5	13.51	3	8.33	4	10.81	1	2.94	2	6.90	15	8.67
	Post	5	16.67	1	4.35	2	10.53	5	23.81	8	36.36	21	38.26
Physically fit	Pre	22	59.46	27	75.00	29	78.38	29	85.29	18	62.07	125	72.25
	Post	11	36.67	5	21.74	6	31.58	12	57.14	10	45.45	44	38.26
Total	Pre	108	48.65	115	53.24	130	58.56	120	58.82	84	48.28	557	53.66
	Post	65	36.11	30	21.74	41	35.96	55	43.65	65	49.24	256	37.10
Nutrition		No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Eat at least 3 meals daily	Pre	30	81.08	35	97.22	37	100	31	91.18	27	93.10	160	92.49
	Post	29	96.67	22	95.65	19	100	20	95.24	22	100	112	97.39
Daily diet from 4 food groups	Pre	31	83.78	33	91.67	31	83.78	31	91.18	27	93.10	153	88.44
at least 3d/wk	Post	28	93.33	20	86.96	17	89.47	18	85.71	19	86.36	102	88.70

		No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Try to cut down on fat in diet	Pre	29	78.38	32	88.89	34	91.89	32	94.12	24	82.76	151	87.28
	Post	25	83.33	21	91.30	19	100	20	92.24	17	77.27	102	89.47
Balance food intake to	Pre	28	75.68	30	83.33	30	81.08	30	88.24	19	65.52	137	79.19
maintain weight	Post	14	46.67	8	34.78	7	36.84	13	61.90	8	36.36	50	43.48
Think body is overweight	Pre	13	35.14	8	22.22	8	21.62	5	14.71	12	41.38	46	26.59
	Post	15	50.00	12	52.17	13	68.42	8	38.10	14	63.64	62	53.91
Total	Pre	131	70.81	138	76.67	140	75.68	129	75.88	109	75.17	647	74.80
	Post	111	74.00	83	72.17	75	78.95	79	75.24	80	72.73	428	74.56
Managing Stress		No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Can identify stressful	Pre	26	70.27	33	91.67	33	89.19	31	91.18	26	89.66	149	86.13
situations in daily life	Post	23	76.67	21	91.30	18	94.74	19	90.48	21	95.45	102	88.70
Take time out daily to	Pre	30	81.08	28	77.78	35	94.59	30	88.24	25	86.21	148	85.55
relax and recover	Post	22	73.33	20	86.96	17	89.47	18	85.71	20	90.91	97	84.35
Find time for enjoyable	Pre	31	83.78	30	83.33	36	97.30	32	94.12	26	89.66	155	89.60
activities	Post	26	86.67	19	82.61	17	89.47	18	85.71	20	90.91	100	86.96
Know how to relieve	Pre	25	67.57	26	72.22	31	83.78	28	82.35	23	79.31	133	76.88
Tension Post		23	76.67	20	86.96	16	84.21	18	85.71	17	77.27	94	81.74
Total	Pre	112	75.68	117	81.25	135	91.22	121	88.97	100	86.21	585	84.54
	Post	94	78.33	80	86.96	68	89.47	73	86.90	78	88.64	393	85.43
Avoiding Destructive Habits		No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Do not smoke or use	Pre	25	67.57	29	80.56	34	91.89	30	88.24	22	75.86	140	80.92
Tobacco products	Post	20	66.67	12	52.17	13	68.42	15	71.43	13	59.09	73	64.04
Do not use alcohol or use	Pre	30	81.08	30	83.33	34	91.89	292	85.29	25	86.21	148	85.55
to legal limit	Post	26	86.67	16	69.57	14	73.68	18	85.71	16	72.73	90	78.26
Do not abuse drugs	Pre	34	91.89	34	94.44	35	94.59	31	91.18	27	93.10	161	93.06
	Post	28	93.33	22	95.65	18	94.74	19	90.48	21	95.45	108	93.91
Use OTC drugs as	Pre	32	86.49	33	91.67	34	91.89	31	91.18	25	86.21	155	89.06
prescribed only	Post	28	93.33	21	91.30	18	94.74	19	90.48	19	86.36	105	91.30

		No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Total	Pre	121	81.76	126	87.50	137	92.57	121	88.97	99	85.34	604	87.28
	Post	102	85.00	71	77.17	63	82.89	71	84.52	69	78.41	376	81.92
Pratising Safe Sex		No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Abstain from or limit sex	Pre	35	94.59	35	97.22	35	94.59	32	94.12	27	93.10	164	94.80
to a safe partner	Post	29	96.67	22	95.65	18	94.74	19	90.48	19	86.36	107	93.04
Practise safe procedures	Pre	32	86.49	34	94.44	33	89.19	32	94.12	28	96.55	159	91.91
for avoiding STDs	Post	25	83.33	22	95.65	17	89.47	21	100	20	90.91	105	91.30
Total	Pre	67	90.54	69	95.83	68	91.89	64	94.12	55	94.83	323	93.35
	Post	54	90.00	44	95.65	35	92.11	40	95.24	39	88.64	212	92.17
Adopting Safety Habits		No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Use seat belt in a vehicle	Pre	32	86.49	32	88.89	35	94.59	31	91.18	26	89.66	156	90.17
	Post	26	86.67	21	91.30	16	84.21	19	90.48	21	95.45	103	90.35
Afraid when exceeding	Pre	22	59.46	27	75.00	33	89.19	28	82.35	23	79.31	133	76.88
speed limit	Post	21	70.00	19	82.61	16	84.21	18	85.71	18	81.82	92	80.00
Total	Pre	54	72.97	59	81.94	68	91.89	59	86.76	49	84.48	289	83.53
	Post	47	78.33	40	86.96	32	84.21	37	88.10	39	88.64	195	85.15
Knowing First Aid		No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Trained in first aid	Pre	19	51.35	25	69.44	30	81.08	28	82.35	20	68.97	122	70.52
	Post	19	63.33	19	82.61	14	73.68	18	85.71	15	68.18	85	73.91
Can perform first aid	Pre	20	54.05	25	69.44	30	81.08	28	82.35	19	65.52	122	70.52
in an emergency	Post	20	66.67	18	78.26	16	84.21	18	85.71	16	72.73	88	76.52
Can call medical help	Pre	35	94.59	35	97.22	36	97.30	31	91.18	27	93.10	164	94.80
in an emergency	Post	28	93.33	22	95.65	17	89.47	20	95.24	21	95.45	108	93.91
Total	Pre	74	66.67	85	78.70	96	86.49	87	85.29	66	75.86	408	78.61
	Post	67	74.44	59	85.51	47	82.46	56	88.89	52	78.79	281	81.45
Personal Health Habits		No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Brush teeth at least	Pre	32	86.49	35	97.22	37	100	34	100	29	100	167	96.53
twice daily	Post	28	93.33	21	91.30	19	100	19	90.48	22	100	109	94.78
Get enough sleep daily	Pre	25	67.57	30	83.33	34	91.89	28	82.35	21	72.41	138	80.23

		No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
	Post	20	66.67	19	82.61	15	78.95	19	90.48	17	77.27	90	78.26
Total	Pre	57	77.03	65	90.28	71	95.95	62	91.18	50	86.21	305	88.41
	Post	48	80.00	40	86.96	34	89.47	38	90.48	39	88.64	199	86.52
Using Medical Advice		**No.**	**%**	**No.**	**%**	**No.**	**%**	**No.**	**%**	**No.**	**%**	**No.**	**%**
Seek medical advice	Pre	32	86.49	30	83.33	35	94.59	33	97.06	25	86.21	155	89.60
when necessary	Post	25	83.33	20	86.96	18	94.74	19	90.48	17	77.27	99	86.09
Follow advice or medication	Pre	34	91.89	34	94.44	37	100	34	100	26	89.66	165	95.38
as prescribed	Post	24	80.00	18	78.26	15	78.95	19	90.48	15	68.18	91	79.13
Total	Pre	66	89.19	64	88.89	72	97.30	67	98.53	51	87.93	320	92.49
	Post	49	81.67	38	82.61	33	86.84	38	90.48	32	72.73	190	82.61
Being an Informed Consumer		**No.**	**%**	**No.**	**%**	**No.**	**%**	**No.**	**%**	**No.**	**%**	**No.**	**%**
Read product labels	Pre	13	35.14	8	22.22	6	16.22	4	11.76	11	37.93	42	24.28
	Post	10	33.33	6	26.09	3	15.79	2	9.52	3	13.64	24	20.87
Use products shown	Pre	22	59.46	26	72.22	33	89.19	30	88.24	25	86.21	136	78.61
Effective by research	Post	26	86.67	20	86.96	17	89.47	18	85.71	19	86.36	100	86.96
Total	Pre	35	47.30	34	47.22	39	52.70	34	50.00	36	62.07	178	51.45
	Post	14	23.33	9	19.57	5	13.16	5	11.90	6	13.64	39	16.96
Protecting the Environment		**No.**	**%**	**No.**	**%**	**No.**	**%**	**No.**	**%**	**No.**	**%**	**No.**	**%**
Recycle waste	Pre	6	16.22	1	2.78	5	13.51	1	2.94	3	10.34	16	9.25
	Post	6	20.00	1	4.35	1	5.26	2	9.52	2	9.09	12	10.43
Conserve energy	Pre	29	78.38	31	86.11	34	91.89	33	97.06	26	89.66	153	88.44
	Post	26	86.67	20	86.96	19	100	20	95.24	22	100	107	93.04
Concerned about	Pre	30	81.08	31	86.11	37	100	34	100	27	93.10	159	91.91
global warming	Post	29	96.67	23	100	19	100	21	100	22	100	114	99.13
Total	Pre	65	58.56	63	58.33	76	68.47	68	66.67	56	64.37	328	63.20
	Post	58	64.44	41	59.42	38	66.67	41	65.08	44	66.67	222	64.35
Mental Well-being		**No.**	**%**	**No.**	**%**	**No.**	**%**	**No.**	**%**	**No.**	**%**	**No.**	**%**
Have a sense of purpose	Pre	36	97.30	35	97.22	36	97.30	34	100	28	96.55	169	97.69
in life	Post	29	96.69	23	100	19	100	21	100	22	100	115	99.13

Positive about country's future	Pre	27	72.97	30	83.33	34	91.89	31	91.18	27	93.10	149	86.13
	Post	25	83.33	19	82.61	17	89.47	19	90.48	19	86.36	99	86.09
Total	Pre	63	85.14	65	90.28	70	94.59	65	95.59	55	94.83	318	91.91
	Post	54	90.00	42	91.30	36	94.74	40	95.24	41	93.18	213	92.61

Key: OTC = over-the counter; STDs = sexually transmitted diseases.

The subjects failed to act as informed consumers, since only 20.87 % read product labels at post-test, similar to the 24.28% at pre-test. However, most of them continued to score high on only using products shown effective by research at post-test (86.96%), much like that at pre-test (78.61%).

Subjects continued to score high on conserving energy (93.04%), and were particularly concerned about global warming (99.13%). Nevertheless, they continued to score low on recycling waste at post-test (10.43%).

Subjects continued to score high at post-test for having a sense of purpose in life (99.13%) and being positive about the country's future (86.09%).
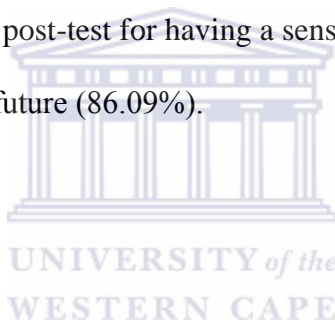
4.4.1.4. Physical Activity

The response rate for the IPAQ questionnaire at post-test was 43.48% (50). Table 4.24 contains a comparison of the subjects' results for physical activity between pre- and post-test. Overall, weekly energy expenditure decreased from pre- to post test, with the GG showing a decrease of 6.93% from pre-test at 574.61 to post-test at 534.80 MET·min·wk^{-1}. The CG, HIG and DG all showed similar decreases in weekly energy expenditure of 19.44%, 26.45% and 9.15%, respectively. The EG and MG, in contrast, showed an improvement in weekly energy expenditure of 6.23% and 13.86%, respectively.

The minimum amount of physical activity remained relatively unchanged from pre- to post-test with the results ranging from 70 to 210 MET·min·wk^{-1} at pre-test to scores ranging from 70 to 315 MET·min·wk^{-1} at post-test. However, the results for the maximum amount of physical activity decreased considerably from pre- to post-test. The results for the maximum amount of physical activity at pre-test ranged from 1260 to 2625 MET·min·wk^{-1}, whereas the results at post-test ranged from 840 to 1260 MET·min·wk^{-1}. This represented a decline in the maximum amount of physical activity in the range of 15 to 52%. The maximum amount of physical activity declined in all the groups at post-test, with the results ranging from 16.67% in the DG, 25% in the MG, 33.67 % in the CG and EG each, to 52% in the HIG.

Most of the subjects, 56.52% (28), did not reach the recommended 30 minutes of moderate intensity activity (3.0 – 5.9 METs) on 4 or more days per week and were typical of a sedentary population. At post-test, the CG and DG showed a decrease in mean weekly physical activity to scores of 455.00 and 453.86 MET·min·wk^{-1} respectively. This is below the minimum 500 MET·min·wk^{-1} recommended for health benefits. The MG, in contrast, experienced an improvement in mean weekly physical activity from 480.71 MET·min·wk^{-1} at pre-test to 558.08 MET·min·wk^{-1} at post-test, meeting the minimum requirements for health benefits.

In general, the results for physical activity showed that, most of the subjects continued to have low levels of weekly physical activity at post–test, despite participating in an intervention programme.

Table 4.24. Mean weekly energy expenditure of the subjects at pre-test and post-test.

Groups		N	Mean (MET·min·wk^{-1})	SD	Minimum (MET·min·wk^{-1})	Maximum (MET·min·wk^{-1})
Grand	Pre	51	574.61	461.80	70.00	2625.00
	Post	50	534.80	310.03	70.00	1260.00
Control	Pre	11	564.77	339.17	70.00	1260.00
	Post	11	455.00	248.97	70.00	840.00
Health Information	Pre	7	907.50	827.17	157.50	2625.00
	Post	7	667.50	439.76	87.50	1260
Diet	Pre	11	499.55	354.21	140.00	1260.00
	Post	11	453.86	290.98	80.00	1050.00
Exercise	Pre	8	564.38	448.78	210.00	1575.00
	Post	8	601.88	292.36	315.00	1050.00
Multiple	Pre	14	480.71	370.04	210.00	1680.00
	Post	13	558.08	313.12	210.00	1260
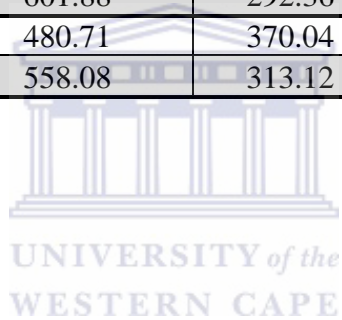
Chapter Five: Discussion, Conclusion and Recommendations

5.1   Introduction

This study sought to investigate the impact of multiple health behaviour interventions over a 16 week trial on CHD risk factors, HRPF, and HRB in first year university study stratified as being moderately at risk for CHD. The study has as its premise that the impact of different behavioural interventions would bring about the following changes, namely:

- Reduction in CHD risk factors;

- Improvement of HRPF;

- Reduction of HRB; and

- Reduction of CHD risk factors in a dose-response relationship, that is, the decrease in CHD risk factors would be proportionate to the number of treatments applied, so that subjects exposed to more treatments (higher dose) would show more benefits (better response), with the effect being cumulative in the MG.

Invariably it was contended that subjects presenting with a higher number of CHD risk factors ($\geq$ 3 risk factors) would benefit more from treatment as opposed to subjects presenting with two risk factors only.

This chapter will therefore address the following aspects:

- Demographic characteristics of subject

- Research Measurements: -

    - CHD risk factors

    - HRPF

    - HRB

    - Physical activity

- Shortcomings of the study

- Strengths of the study

- Recommendations

- Conclusion

5.2   Demographic Characteristics of the Subjects

The subjects in the study represented a diverse ethnic group similar to that of the university's

pluralistic racial demographic. The gender and racial distribution of the subjects in the study

showed that females and persons of mixed ethnic ancestry constituted the majority of the sample.

The results in a survey by the Human Sciences Research Council (HSRC) reported the regional

gender and racial demographics as 39% male, and 61% female with 38% of mixed ethnic

ancestry, 26% black, 22% Indian, 10% white, and 4% other (Breier, 2007). Also reported in the

HSRC survey was the gender and racial profile of the university student population at the first

year level with 43% males and 57% females of which 49% were persons of mixed ethnicity,

39% black, 9% Indian, 3% white (Breier, 2007). However, the university student demographics

were quite different to that of the sample group which had 21% male and 79% female, and comprising 63% subjects of mixed ethnic ancestry, 32% black, 2% Indian, and 2% white.

5.3   Stages of Readiness to Change

The intervention strategies applied in this study were based upon the Transtheoretical Model (TTM) or stages of readiness to change (Prochaska et al., 1992), because it offered a promising theoretical framework for multiple health behaviour interventions (Johnson et al., 2008). Being stage-dependent, the interventions were designed to progressively move subjects from one (lower) stage to the next (higher) stage as treatment progressed (Griffin, 2006, pp. 3-40). Two behavioural processes, derived from the TTM, explained how subjects progressed through the different stages. Cognitive processes were used to mobilize subjects in the precontemplative and contemplative stages, whereas for subjects in the other stages (preparation through to maintenance), behavioural processes were applied (Pinto et al., 2002). Subjects in precontemplation, for example, were provided information on the benefits of physical activity and on overcoming barriers to physical activity. Subjects in the action stage were informed how to make physical activity enjoyable and how to prevent injuries, that is, combining cognitive and psychological approaches to behaviour change in order to foster self-efficacy through empowerment and self-actualization.

At baseline, most subjects were in the contemplative stage of behaviour change. Identifying situations that counteracted healthy behaviours, such as holidays or heavy workloads, and strategies to handle these situations were discussed.

The general focus of the interventions was on individualizing treatment to the needs of the subject by, firstly, creating a general sense awareness of the potentially harmful effects of the various CHD risk factors; secondly, getting subjects to identify the CHD risk factor(s) that placed them at risk in order of priority of management, thirdly, by changing the subjects' perceptions towards the risk factors through examining how they effectively managed other risk factors in the past, and, finally, by consciously modifying the subjects' lifestyles and changing their behaviours, such as making a conscious decision to start exercising or to stop smoking.

Following the intervention period, the majority of the subjects had progressed from the precontemplative stage to the preparation and action stages, which indicated that the subjects were beginning to overcome their behavioural problems, particularly related to nutrition and exercise and were either preparing or taking action to adopt healthier lifestyles. Also, the impact of the multiple behaviour intervention was greater than that of the single behaviour interventions. A similar finding was reported by Johnson et al. (2008) where they found that the impact of the multiple behaviour interventions was three times more effective than that of the single behaviour interventions. The present study was successful in its primary aim of showing that a multiple health behaviour intervention programme, based upon the systematic application of Prochaska's Transtheoretical Model was efficacious in stimulating lifestyle modification.

Part of the strategy of changing the subjects' behaviours was also to provide them with the resources for long-term sustainability, such as the professional support from student counselors at the Centre for Student Support Services (CSSS), exercise therapists at the biokinetics clinic,

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and intern dietitians at the dietetics helpdesk on campus. Additional resources for reinforcement of lifestyle change were having access to social support from family, friends, peers, significant others, and the community. It was reiterated those with minimal or absent coping skills were more prone to relapse and succumb to previous risky behaviours.

5.4 CHD Risk Factors

5.4.1 Physical Characteristics

In this study, age was not a risk factor because the subjects were all young with a mean age of 21.35±6.34 years, mostly unmarried, relatively injury-free and, although classified as moderately at risk for CHD, were otherwise normal, healthy individuals. Some subjects complained of recalcitrant orthopedic conditions, such as shoulder, knee, back or other joint pain, and a few had chronic medical conditions, such as asthma, sinusitis, allergies and/or hypertension, but none of these conditions were too debilitating to affect their participation in the study.

At baseline, although the mean BMI for the subjects placed them in the overweight category, it was rated low for CHD risk. This finding was further supported by a low CHD risk rating for WHR and waist circumference.

Waist circumference is a significant predictor of CHD risk (ACSM, 2006b, p.59). Findings from the present study suggest that participation in either health information or diet programme or a combination of the two together with exercise can significantly reduce waist circumference.

Abdominal adipocytes are apparently more responsive to exercise training than femoral adipocytes, suggesting that exercise training may result in the selective loss of abdominal fat as opposed to femoral and gluteal fat and, thereby, reduce the waist-hip ratio (Wood and Stefanick, 1990, p. 420). In this study, exercise did not produce the same beneficial effects as the other treatments on waist circumference but was effective when administered in combination with diet and health information. Reductions in waist circumference observed in the present study together with reductions in body weight, were consistent with the results from other exercise intervention studies (Eriksson et al., 2009). Furthermore, findings by Baruth et al. (2011) suggest that it was primarily the exercise, when combined with health information and diet that resulted in significant amounts of weight loss. These findings are also consistent with other investigations that studied the impact of health behaviour interventions on CHD risk factors in individuals at risk (Fernandez et al., 2007).

Grundy et al., 1999 and Monyeki et al., 2005 reported that participation in an exercise programme preferentially helped to preserve lean tissue mass, similar to the findings of this study. The findings in these study demonstrated that through simultaneous dieting and exercise, subjects were able to lose a large amount of body weight, specifically fat weight, while maintaining a relatively large proportion of fat-free mass. In the absence of exercise, a down-regulation in resting energy expenditure (metabolism) has been reported with dieting, and is linked with difficulties in weight management, especially with advancing age (McArdle et al., 2001, p. 827). Collectively, these findings suggest that resting energy metabolism and body

weight can be maintained by participating in regular exercise, coupled with a modest diet programme and relevant health information.

5.4.2 Blood Biochemistry

The relationship between exercise, fitness and lipoprotein cholesterol has been characterized quite extensively in the literature (Wood and Stefanick, 1990, p. 410 - 421). In a meta-analysis of forty-nine randomized controlled trials, aerobic exercise reduced total cholesterol and triglycerides and increased HDL cholesterol in men 18 years or older (Kelley and Kelley, 2006). The authors reported statistically significant improvements for total cholesterol, HDL cholesterol and triglycerides, with a trend for decreased LDL cholesterol concentration. Changes were equivalent to improvements of 2% for total cholesterol and HDL cholesterol, 3% of LDL cholesterol, and 9% for triglycerides.
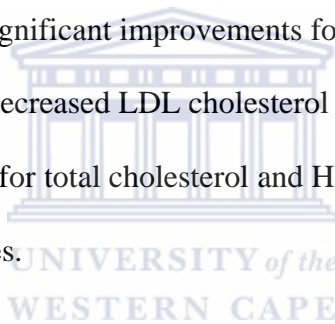
In this study, the reduction in total serum cholesterol concentration in the HIG, DG and EG, but not in the MG, was larger than anticipated considering that pre-test values in all groups were within normal limits. It is possible that even though the subjects in this study had in fact adopted a healthier lifestyle following the intervention, this behavioural change had not (yet) translated into a sizeable and significant effect on serum cholesterol concentration, an observation also reported by other investigators (Blair et al., 1983; Kemper et al., 2002; Wood et al., 1983). The results from this study are in agreement with those of Wood et al. (1983) who showed that plasma total cholesterol in the general population does not vary by much with physical activity, that is, more active individuals usually do not show significantly lower total cholesterol

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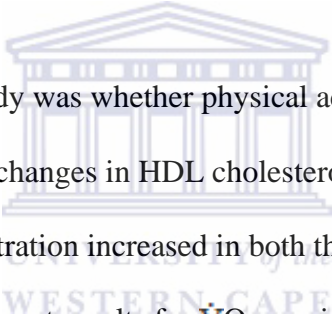
concentrations than less active individuals. A significant reduction in plasma cholesterol concentration is more typical of dedicated, habitual exercisers and shown only in long-term trials (Wood and Stefanick, 1990, p. 416). Very likely, for subjects at risk, a more extended intervention programme in excess of four months is required to translate the favourable changes in total cholesterol into measurable reductions in risk.

Wood and Stefanick (1990, p. 417) showed in several uncontrolled studies that increases in HDL cholesterol concentration occurred as part of regular training and was inversely related to CHD risk. This result, however, was not the case in a large, controlled study over one year by LaRosa et al. (1982). In contrast, unlike HDL cholesterol, raised LDL cholesterol concentration is precipitous in causing increased CHD risk. Clearly, the progression or regression of cardiovascular disease, in most part, appears to depend upon the interplay of LDL (ingress system) and HDL (egress system) cholesterol concentrations, with the former system acting as a donor depositing cholesterol in body tissues, with the latter system acting as a cholesterol acceptor in the reverse cholesterol transport system that removes it from the tissues for elimination in bile.

In this study, the decrease in LDL cholesterol concentration following intervention, though positive and promising, was not significant, and was similar to the findings reported in a study on exercise training over a limited period of time by Wood et al. (1983).

In contrast to LDL cholesterol concentration, an increase in HDL cholesterol occurred with exercise training in the present study but the results were not significant. These results, however,

support other research data showing that exercise training invokes a positive alteration in lipoprotein levels, particularly HDL, in a moderate risk, but otherwise healthy population (Kelley and Kelley, 2006). In general, most exercise studies lasting longer than 12 weeks reported an increase in mean HDL cholesterol concentration averaging about 5 mg$^.$dl^{-1}, although not all were significant (Wood and Stefanick, 1990, pp. 416-7). As indicated in a longitudinal training study by Wood et al. (1983), there is apparently a minimum training threshold of 15 km per week of running that must be exceeded before significant changes in HDL cholesterol concentration become manifest as significant. No such training threshold was considered or recorded in this study to monitor changes in HDL cholesterol concentration.

A question raised in the present study was whether physical activity or physical fitness as defined by $\dot{V}O_2$max best predicted changes in HDL cholesterol concentration and associated CHD risk. HDL cholesterol concentration increased in both the EG and MG with exercise training. However, there were divergent results for $\dot{V}O_2$max in these two groups, with the MG showing a significant increase in $\dot{V}O_2$max post-test, while the EG, in contrast, had a decrease in $\dot{V}O_2$max. It is conceivable that the EG, even though more physically active subsequent to treatment, failed to exercise at an intensity exceeding the minimum threshold, a critical level for providing the stimulus to precipitate change in physical fitness or $\dot{V}O_2$max. The divergent changes in $\dot{V}O_2$max in the EG and MG reflected differences in physical fitness rather than differences in physical activity. Therefore, it is argued in the present study that physical fitness could quite reasonably be excluded as an influencing factor on HDL cholesterol changes produced at post-test. Since physical activity increased consistently in both these groups at post-

test, it remained an influencing factor and possibly accounted for the favourable rise in HDL cholesterol concentration in these two groups.

Plasma triglyceride levels correlate closely with VLDL cholesterol concentration and, like total cholesterol concentration, tended to be markedly lower in physically active individuals (Wood et al., 1983). This adaptation is explained on the basis of trained muscles utilizing fat more efficiently for energy, leading to considerable amounts of triglycerides being burned for fuel during exercise (Wood and Stefanick, 1990, p. 414).

In this study, subsequent to the intervention programme, plasma triglyceride level decreased in all the groups, except the controls, but was not significant. A plausible explanation for the lack of a significant outcome in triglyceride levels could be that at baseline triglyceride levels were also within normal limits. Furthermore, the impact of treatment, although positive in helping to shift triglyceride levels in a favourable direction, was not long enough to produce a significant change. Similar to precipitating change in total cholesterol concentration, apparently a high volume of intensive physical activity is also required to produce markedly lower triglyceride levels in subjects with initially normal values (Blair et al., 1983).

The ratio of LDL to HDL cholesterol concentration appears to be another important predictor of risk for CHD with low ratios directly associated to attenuated risk and increased ratios with protective benefits (Wood and Stefanick, 1990, p. 412). The inverse relationship between HDL cholesterol concentration and LDL cholesterol concentration, shown in the present study,

particularly with multiple behaviour interventions, is also reported in the literature (Leon and Bronas, 2009).

The cholesterol distribution among the various lipoproteins is a powerful predictor of heart disease risk (McArdle et al., 2001, p. 898). Overall, the improvement in lipoprotein levels was better demonstrated when these indices were depicted as ratios, namely, TC:HDL, LDL:HDL and trig:HDL (Table 4.13). There were noticeable reductions for each of these ratios across all the experimental groups when compared to the controls. The control group reflected consistent increases for all ratios at post-test indicative of a progressively worsening cholesterol status.

5.4.3 Clinical Indices

At baseline, the elevated levels for resting heart rate, and systolic and diastolic blood pressures appeared to be linked with cigarette smoking. The beneficial impact of the intervention programme was evident primarily with multiple treatments, where it reduced resting heart rate significantly, and changed systolic and diastolic blood pressures from pre-hypertensive values at baseline to normotensive values at follow-up. Ashen (2010) reported lifestyle modification through multiple behavioural interventions as an important avenue to modify blood pressure and minimize CHD risk.

Smoking is associated with a number of physiological changes which increase CHD risk, and is identified as the single most preventable cause of morbidity and mortality in SA (Norman et al.,

2006; Steyn and Fourie, 2007). Smokers are more likely to have a clustering of risk factors such as inactivity, dyslipidemia and hypertension (Ward et al., 2003). Compared with non-smokers, smokers have raised serum concentrations of cholesterol, serum triglycerides, and VLDL triglycerides, and attenuated HDL cholesterol (Näslund et al., 1996; Wood and Stefanick, 1990, pp. 418-19).

In the present study, a reduction in cigarette smoking was evidenced in all the experimental groups, but was significant for the multiple interventions only. These findings provide some evidence confirming the benefit of brief structured interventions involving health information, diet and exercise for reducing cigarette smoking, and are supported by similar findings by Fernandez et al. (2007). Cigarette smoking appears to decrease as exercise levels increase, so the adoption of an exercise programme is likely to result in attenuated smoking levels (Wood and Stefanick, 1990, p. 419). In this study, the reduction in cigarette smoking could be accounted for, at least in part, by the change in physical activity status of the subjects. Obviously, the reduction in smoking was beneficial and served not only to lower the estimated CHD risk but, possibly, also reduced the impact of risk mechanisms, such as improving hypertension and HDL levels (Kelley and Kelley, 2006; Wood and Stefanick, 1990, p. 419) and reducing oxidative damage (Näslund et al., 1996).

5.4.4 CHD Risk Stratification

Having an optimal risk-factor status (≤ 1 CHD risk factor) confers a low risk for CHD, and is an important concept to inculcate, especially in youth. One of the main outcomes in the present study was the positive impact of the intervention programme on the risk stratification of subjects.

Bearing in mind that all subjects were stratified as moderate risk at baseline, Table 5.1 shows the re-stratification of the subjects at post-test with over a quarter of them re-stratified as low risk. In the GG, 27.83% of subjects were re-stratified as low risk ($\leq$ 1 CHD risk factor) at post-test. Furthermore, 6.09% of the subjects stratified as low risk also had zero risk factors. CHD risk re-stratification affected all the groups, including the controls, with the number of subjects re-stratified as low risk at post-test ranging in order of percentage from 6.67%, 10.50%, 26.09%, 38.10% to 49.90% in the CG, DG, HIG, EG and MG, respectively. With the exception of the DG, all the other experimental groups showed subjects with zero risk ranging from 4.35% and 9.52% in the HIG and EG, respectively, to an impressive 18.90% in the MG. The CG showed no subjects with zero CHD risk at post-test, and reflected some evidence of a lack of a treatment effect for this group.
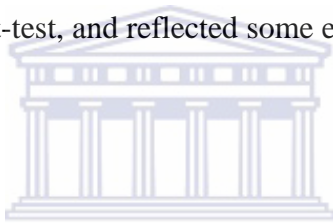
Table 5.1 Stratification of CHD risk at post-test.

Groups	Risk Stratification		Zero CHD Risk (%)
	Moderate CHD Risk (%)	Low CHD Risk (%)	
Grand	72.17	27.83	6.09
Control	93.38	6.67	0.00
Diet	89.50	10.50	0.00
Health information	73.91	26.09	4.35
Exercise	61.90	38.10	9.52
Multiple	50.10	49.90	18.90

Except for age, seven CHD risk factors were assessed in this study, namely, a family history of CHD, cigarette smoking, hypertension, dyslipidemia, impaired fasting glucose, obesity, and a sedentary lifestyle. For stratifying subjects as moderate CHD risk, they needed to have two or more CHD risk factors present. For the purposes of this study, the CHD risk factors were grouped as frequencies ranging from two to six risk factors, since none of the subjects had all seven risk factors either at pre- or post-test. When considering the number of subjects with various frequencies of risk factors, it was apparent that there was a substantial shift in subjects from higher frequencies (≥ 2 risk factors) at pre-test to lower frequencies (≤ 1 risk factor) at post-test (Table 4.19). The presence of subjects with zero and one risk factor at post-test, unlike baseline, is clear evidence of the beneficial impact of the intervention programme. Thus, the positive changes could be considered important on two levels. Firstly when considering the shift in the number of subjects who dropped not only to one but to zero risk. Secondly, for all frequencies of risk (≥ 2 risk factors), there was a substantial decrease in the number of subjects. Favourable changes, however, also occurred in the controls, but these were much smaller, and could be ascribed to the spillover effects of the intervention programme.

When the frequencies of CHD risk factors were viewed across the various treatment groups, considerable changes occurred subsequent to the intervention (Figure 5.1). In the GG, throughout all the frequencies of risk at post-test ranging from two, three, four, five and six risk factors, there were consistent percentage decreases in the number of subjects to the values of 9.14%, 7.45%, 4.88%, 5.78% and 0.58%, respectively [Figure 5.1(a)]. Furthermore, subjects showing one and zero risk amounted to 21.74%, and 6.09%, respectively.

In the CG, the frequencies of three, five and six CHD risk factors decreased by 6.67%, 6.67% and 3.33%, respectively, but this change was offset by a 10.00% increase in subjects with four risk factors, and a corresponding improvement by 6.67% in subjects with one CHD risk factor [Figure 5.1(b)]. Thus, despite these modest improvements since some subjects did move from higher to lower frequencies, however, most of them still remained at moderate risk with minimal re-stratification to low risk, and none to zero risk, at post-test.

With regard to the change in frequencies of risk factors in the experimental groups subsequent to the intervention programme, the results were positive and additive relative to the type of treatment administered, and supports the hypothesis of the study. The groups ranging from least to most responsive were the DG and MG, respectively, with the HIG and EG producing variable results between these two groups. In the DG, there were modest decreases in the number of subjects with frequencies of three and five risk factors by 5.26% for each frequency, and an increase of 10.53% in subjects with one risk factor, but none moved to zero risk at post-test [Figure 5.1(d)].

In the HIG, the frequencies with three, four and five CHD risk factors decreased by 13.04%, 8.70 and 8.70%, respectively, with 26.09% of subjects shifting to one risk factor, and 4.35% shifting to zero risk [Figure 5.1(c)]. Similarly, in the EG, there were substantial decreases for the frequencies with two, three, and five CHD risk factors by 33.33%, 4.76% and 9.52%, respectively [Figure 5.1(e)]. The proportions of subjects who shifted to one and zero risk were 38.10% and 9.52%, respectively.
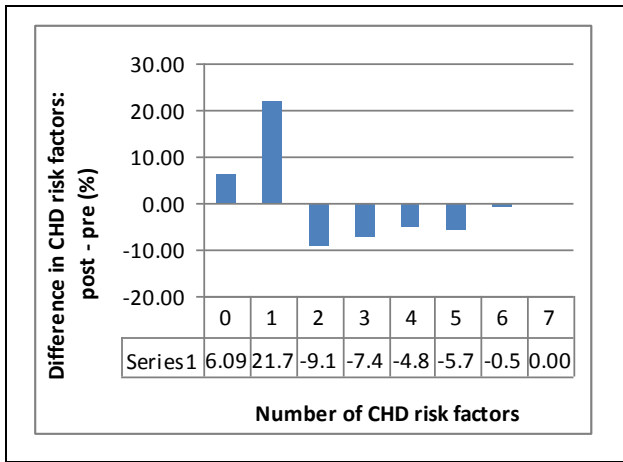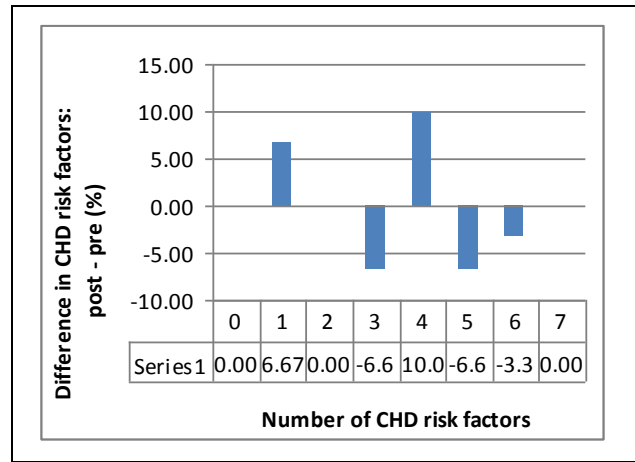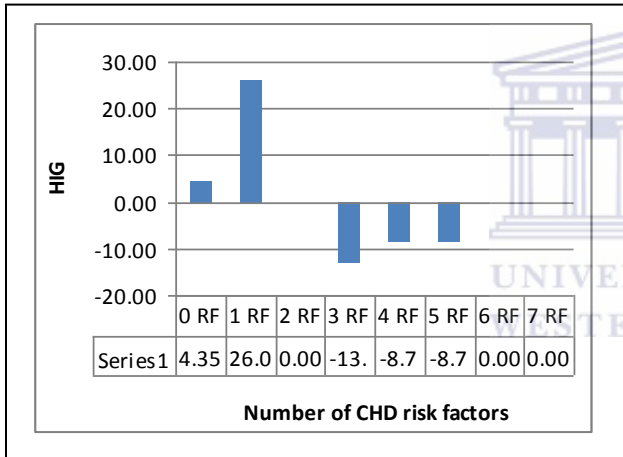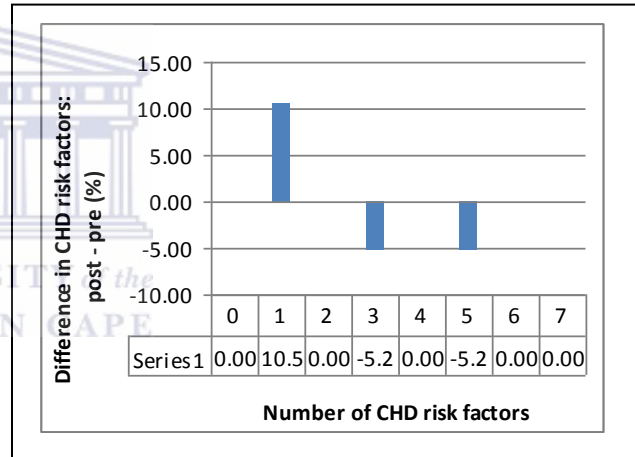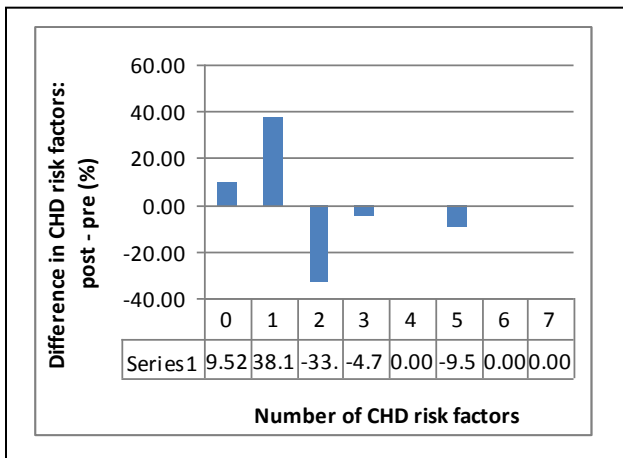
	0	1	2	3	4	5	6	7
Series1	6.09	21.7	-9.1	-7.4	-4.8	-5.7	-0.5	0.00

Number of CHD risk factors

(a)   Grand group.

	0	1	2	3	4	5	6	7
Series1	0.00	6.67	0.00	-6.6	10.0	-6.6	-3.3	0.00

Number of CHD risk factors

(b)   Control group.

	0 RF	1 RF	2 RF	3 RF	4 RF	5 RF	6 RF	7 RF
Series1	4.35	26.0	0.00	-13.	-8.7	-8.7	0.00	0.00

Number of CHD risk factors

(c)   Health information group.

	0	1	2	3	4	5	6	7
Series1	0.00	10.5	0.00	-5.2	0.00	-5.2	0.00	0.00

Number of CHD risk factors

(d)   Diet group.

	0	1	2	3	4	5	6	7
Series1	9.52	38.1	-33.	-4.7	0.00	-9.5	0.00	0.00

Number of CHD risk factors

(e)   Exercise group.

	0	1	2	3	4	5	6	7
Series1	18.1	31.8	-4.5	-13.	-27.	-4.5	0.00	0.00
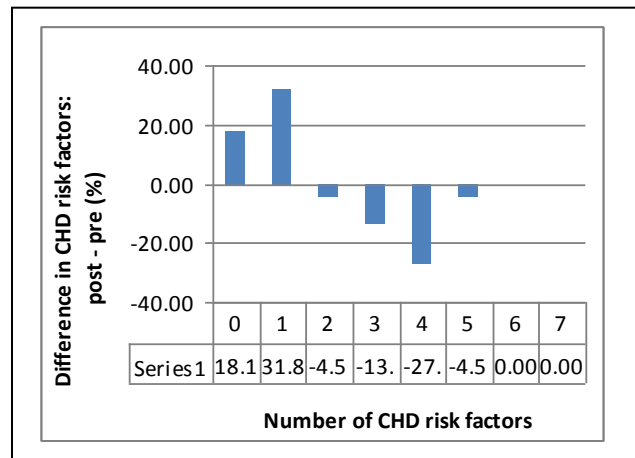
Number of CHD risk factors

(f)   Multiple group.
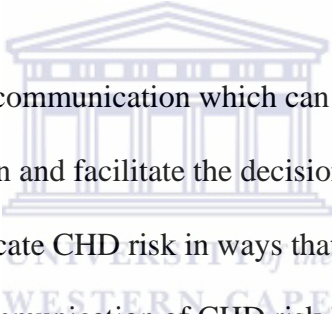
Subjects in the MG were affected most favourably. In this group, participants throughout all the frequencies of risk from two, three, four and five CHD risk factors showed a decrease by 4.53%, 13.64%, 27.27% and 4.55%, respectively [Figure 5.1(f)]. More impressive was the shift in subjects to one and zero risk that corresponded to 31.82% and 18.18%, respectively. The beneficial change in CHD risk for most subjects in this group represented a pattern of CHD prevalence synonymous with optimal health and more normal risk for the general population.

Also noteworthy, was the finding that in the MG the largest improvement was for subjects having very high risk frequencies (≥ 4 risk factors), unlike in the other experimental groups that evidenced a decrease in the number of subjects at lower frequencies ($2 - 3$ risk factors). Presumably, a multi-faceted behavioural intervention programme is more of a pre-requisite than a recommendation to comprehensively attenuate risk in subjects with higher frequencies of risk (≥ 4 risk factors), unlike those with lower risk frequencies that may be more responsive to single, individualized interventions. Nonetheless, the improvements in CHD risk across all the frequencies of risk for all the experimental groups was of particular significance in this study and testimony of the efficacy of the intervention programme. The positive change in frequencies of risk based upon a multipronged intervention that used exercise, diet and health information are consistent with the findings shown by Becque et al. (1988). Their results show that there is indeed an improvement in CHD risk factors with various single-based treatments but, by far, the best results occurred in subjects exposed to all three treatments (diet, exercise and behaviour therapy).

Pate et al. (1997) stated that, in order to be effective, the health education intervention must be given at different levels, partly to individuals, but also to public health agencies, health professionals, communities, educators, and families that all interact with at-risk individuals. Thus, a reason for only partial intervention effects from health information in the present study may be due to the fact that the information was given solely to the subjects and not to additional support structures. Moreover, the health information was only aimed at information dissemination and knowledge acquisition, and not at other relevant determinants, such as attitudes and social support that also appear to be significant influences (Kemper et al., 2002).

Of considerable importance is risk communication which can influence individual awareness, change inappropriate risk perception and facilitate the decision to reduce risk. Little is known about how to portray and communicate CHD risk in ways that motivate people to reduce their risk (Waldron et al., 2011). The communication of CHD risk is particularly complex for a number of reasons. Firstly, there is a plethora of risk factors, ranging from non-modifiable ones such as age, family history and gender, to the modifiable risks such as hypertension, dyslipidemia, cigarette smoking, lack of physical activity and obesity, with each strongly influencing personal risk either in isolation or in combination (Waldron et al., 2011). Secondly, heart disease is a subtle, insidious disorder and reducing its prevalence has to start early in life and be carried out over many decades. Most CHD risk inventories are done much later in life and are based upon methods that, almost always, assume that the issue of risk is preferentially addressed in later life (Waldron et al., 2011). Finally, most individuals find cardiovascular

disease an abstract concept, and have difficulty interpreting personal risk, especially at a young age (Waldron et al., 2011).

The public's perception of what constitutes CHD risk is generally poor and insufficient (Steyl, 2008, Crouch, 2008, p. 57), even more so when that risk is presented in prediction tools. In essence, therefore, appropriate cardiovascular risk communication strategies, firstly, need to help individuals better understand the multiplicity of risk factors and the contribution of each to future risk; secondly, to be able to properly and accurately promote perceptions of risk that are realistic and unambiguous; and, lastly, enable informed decisions to be made regarding cardiovascular health in order to motivate personal behaviour change and promote self-efficacy in lifestyle management. Making individuals aware of their risk, especially if introduced at an early age, can encourage action for risk reduction and, hopefully, translate not only into a better quality of life, but also a longer life.
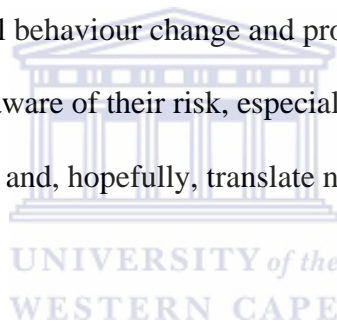
The benefit of expressing CHD in risk frequencies as in the present study is that the numerical presentation of risk as opposed to absolute risk factors leads to a more accurate perception of risk, and influences treatment decisions. Furthermore, an individual's perception of risk is more readily comparable to the average person when using the concept of "heart age equivalent" (Waldron et al., 2011). So, for example, an individual with one or more CHD risk factors has a "heart age" equivalent to someone much older in "heart years". Moreover, an interactive multiplicity effect was found where the higher the numbers of risk factors present in subjects, the more severe was the risk profile, and the resulting effect was that cardiovascular age increased exponentially. This served to raise the subjects' perceptions of personal risk to levels that

invoked heightened emotions causing them to address the risk immediately and more

conscientiously (Waldron et al., 2011). However appealing this approach may sound, it did not

form the basis for encouraging behaviour change in the present study, and provides an intriguing

avenue for motivating behaviour modification in further studies.

One of the novel outcomes of the present study was the determination of the total number of risk

factors, that is, cumulative risk. These results are shown for the GG at pre- and post-test in Table

4.20. Thus, by controlling for the same number of subjects at pre- and post-test (n = 115), the

results showed a substantial drop in cumulative risk by 87 risk factors at post-test.

With reference to Table 5.2, the cumulative risk was calculated for each treatment group. The

cumulative risk at pre-test ranged from 95, 64, 55, 56 to 66 risk factors for the CG, HIG, DG, EG

and MG, respectively. When the same calculation was applied at post-test it produced 87, 43, 49,

37 and 33 risk factors in these same groups.

As shown in Table 5.2, when comparing the differences in cumulative risk between groups, post

hoc analysis showed that, except for the DG, the changes in the HIG, EG and MG were all

significantly different (p range = .003 - .0005) from the CG. In addition, the MG also showed

Table 5.2 Comparison of the cumulative risk at pre- and post-test.

CHD Risk Factors	Grand Group	Control Group	Health Information Group	Diet Group	Exercise Group	Multiple Group	p value
	(Pre = 115) (Post =115)	(Pre = 30) (Post =30)	(Pre = 23) (Post = 23)	(Pre = 19) (Post =19)	(Pre=21) (Post=21)	(Pre = 22) (Post =22)	
Cumulative Risk Pre	336	95	64	55	56	66	---
Cumulative Risk Post	249	87	43*	49	37*	33*	---
Difference (Pre – Post)	87	8	21^{a3}	6	19^{a3}	33$^{a5, b3, c5}$	.0005
Difference (%)	25.89	8.42	32.81	10.91	33.93	50.00	---
MRC per subject (ratio)	0.76	0.27	0.91	0.32	0.91	1.50	---
MRC per subject (%)	76	27	91	32	91	150	---

Key: MRC = mean rate of change

*post-test significantly different from pre-test (p < .0005)

[a3] post-treatment significantly different from control (p < .005)

[a5] post-treatment significantly different from control (p < .0005)

[b3] post-treatment significantly different from health information (p < .005)

[c5] post-treatment significantly different from diet (p < .0005)

significant differences when compared with the HIG (p < .005) and the DG (p < .000), indicative of the profound impact of this intervention strategy on CHD risk amelioration.

Also indicated in Table 5.2, the percentage difference in cumulative risk for each group was calculated and used to indicate the impact or efficacy of the different treatment regimens on CHD. In the GG, the percentage difference in cumulative risk was 25.89%, which indicated a measurable drop in the number of risk factors at post-test, once again indicative of the positive impact of the overall intervention strategy.

Within the various treatment groups, the percentage difference in cumulative risk from pre- to post-test varied quite markedly from 8.42% in the CG, 10.91% in the DG, 32.81% in the HIG, 33.93% in the EG to 50% in the MG. Indeed, the dietary intervention appeared to be the least effective (10.91%), with health information and exercise sharing similar levels of efficacy (32.81% and 33.93%, respectively). By far, the combined treatment in the multiple group stood out from the others as the most effective (50.00%) and supported the hypothesis of the study.

The mean rate of change per subject was calculated from the difference in cumulative risk divided by the total number of subjects in the group, e.g., in the GG, the mean rate of change was 0.76 (87 ÷ 115) risk factors per subject, and when expressed as a percentage, it equaled 76%. The mean rate of change is an indication of the rate at which change or more appropriately reduction in CHD risk factors occurred per subject with a specified treatment. Thus, the mean rate of change in the GG was 76%, as opposed to the CG that had the slowest mean rate of change (27%). Similar to the CG, the DG had a mean rate of change of 32%. The HIG and EG

showed more substantial changes and were tied equally on 91% for mean rate of change. However, most profound was the mean rate of change in MG at 150%. This change in cumulative risk together with the mean rate of change per subject indicated that all interventions produced improvement in CHD risk factors, and the change in CHD risk was dose- response related, in other words, the reduction in CHD risk was inversely related to the number of treatments administered.

As a consequence, positive conclusions can be drawn from this study. These results show that single treatments, when administered over a limited period of 16 weeks, have beneficial effects in reducing CHD risk factors in subjects at moderate risk. However, these treatments when applied in combination produce an additive effect and proportionately reduce the prevalence of CHD risk. Most importantly, behavioural modification through various structured lifestyle interventions acted rehabilitatively in reducing CHD risk, and may add considerably to improving the existing knowledge on best practice for CHD prevention and treatment.

An important observation was that radical lifestyle change was not necessary to facilitate a reduction in CHD risk. Indeed, less comprehensive behavioural changes might have less substantial, but still, positive effects, and may be considered more palatable for many individuals. Certainly, the effects of isolated, single treatments, such as diet, exercise and health information are strong and convincing but, when combined in a multiple treatment strategy, the individualized effects are additive and accelerated. Considerable attention should, therefore, be paid to capitalizing on these treatment effects, if not from an educational perspective then certainly from a public health perspective. Obviously, individuals presenting with higher

frequencies of CHD risk stand to benefit the most from such strategic interventions, and they should be targeted for treatment early.

One important question raised in this study was whether participants with the best adherence showed the most regression in CHD risk? This may provide interesting information on the potential impact of the various treatments. More especially, it also opens up the possibility for considering a threshold of efficacy in treatment where participants' outcomes are based upon a reciprocal dose-response relationship. Stated differently, it is plausible that subjects who complied with treatment up to a certain threshold or dose also responded most favourably in decreasing CHD risk. Thus, beyond a certain threshold of treatment a positive linear relationship may develop with regard to reduction in CHD risk. However, this hypothesis fell outside the scope of this study.
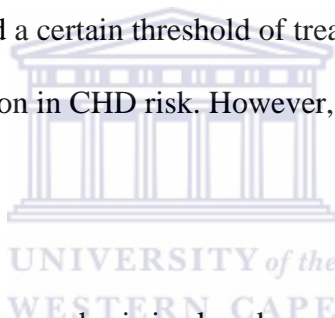
For the purpose of the present study, emphasis is placed upon the favourable impact of the multiple health behavioural intervention strategy on reducing CHD risk, and the correct communication of that information as an important public health initiative in order to improve risk perceptions, increase individual intention to initiate change, and develop self-efficacy in sustaining lifelong behavioural change.

5.5   Health-Related Physical Fitness

Several components of physical fitness are associated with health benefits, including a high level of cardiorespiratory fitness, maintaining a desirable body weight and participating in regular

physical activity (Katzmarzyk et al., 1999; Ortega et al., 2008b; 2011; Tikkanen et al., 1998; Wang et al., 2010). In a predominantly sedentary male Belgian population, good physical fitness was an independent protective factor against CHD (Sobolski et al., 1987). Alternatively, low cardiorespiratory fitness and obesity are independent risk factors for CVD morbidity and mortality (Ortega et al., 2005; Wei et al., 1999).

In the present study, an analysis of the measures of HRPF at baseline showed the participants to have poor results for cardiorespiratory fitness, body composition and muscular strength (ACSM, 2006b, pp. 55 – 90). Significant improvements in HRPF were found primarily with multiple interventions. The EG was the only other experimental group to show a significant improvement in muscular fitness, and this for muscular endurance only (p < .001). Similar finding were also reported by Eriksson et al. (2009) who employed comparable intervention regimens.

According to McArdle et al. (1991, p. 212) and Saltin (1990, p. 187), the mean value for maximal oxygen consumption ($\dot{V}O_2$max) for ages 15 to 20 years in both males and females is generally between 40 to 55 ml$^.$kg$^.$min^{-1}, and remains quite stable for most of young adulthood until about 30 years before starting a steady, progressive decline. Laukkanen et al. (2004) showed $\dot{V}O_2$max values for unfit healthy subjects with two or more risk factors to be less than 27.6 ml$^.$kg$^.$min^{-1}, and for most fit subjects to be equal to or more than 37.1 ml$^.$kg$^.$min^{-1}. For most individuals, males and females, the upper critical threshold for maximal oxygen consumption is about 55 and 60 ml$^.$kg$^.$min^{-1} that is genetically determined regardless of the training regimen (McArdle et al., 1991, p. 212). Only about 20% of the population has the genetic endowment to exceed these limits (Saltin, 1990, p. 188).

In the present study the mean $\dot{V}O_2max$ values of 26.32 ± 7.22 and 25.91 ± 6.51 ml·kg·min^{-1} were obtained at pre- and post test, and fell far below normal values. In spite of the significant improvement in $\dot{V}O_2max$ in the MG by approximately 8%, it still fell short of the standard considered acceptable for optimal health. Coopoo et al. (2003) also reported poor fitness levels in South African youth of Indian descent, in which most of the subjects were sedentary, and could not even attain the minimal level of health-related fitness in order to reduce their risk.

Possible reasons accounting for the poor physical fitness performance of the subjects in the present study may be related to the overall decreased opportunities for physical activity. This stems, in part, from the decreased hours for physical education in the school curricula since 1994 that have naturally lead to decreased levels of physical activity in young adults (Marshall and Hardman, 2000). In addition, the popularity of sport has declined over the years with the result that the youth are leading more sedentary lifestyles and spending more time watching television and playing computer games (Tammelin et al., 2003). Saltin (1990, p. 187) reported that prolonged physical inactivity might cause maximal oxygen consumption levels to become reduced to values as low as 25 ml·kg·min^{-1}, similar to the values found for subjects in the present study.

Physical fitness training for three to four months can increase $\dot{V}O_2max$ between 15 to 20% and produce changes synonymous with normal values of $40 - 55$ ml·kg·min^{-1} (Saltin, 1990, p. 189). Certainly, the subjects in the present study could benefit from physical fitness training, and even low-intensity exercise training (e.g., exercise at less than 45% of maximum aerobic power) has

also been associated with improvements in health status (Warburton et al., 2006). Physical exercise is reported to improve cardiorespiratory capacity and act as a protective mechanism against cardiovascular disease (Steyn et al., 1997). From a public health perspective, Blair et al. (2001) have argued that it is preferable to encourage people to become more physically active rather than to become physically fit, since, as they stated, sedentary people will likely achieve the latter if they do the former.

Apparently among unhealthy subjects, a threshold exists at an exercise intensity of 21.2 ml$^.$kg$^.$min^{-1} in $\dot{V}O_2$peak that, when exceeded, acts as a strong predictor of risk reduction for cardiovascular disease (Laukkanen et al., 2004). This provides concrete evidence that subjects with very low exercise capacities, in the presence of other risk factors, are a target population for further evaluation, and stand to benefit substantially from exercise intervention. Unfortunately, the determination of a threshold for CVD risk reduction fell outside the scope of the present study but provides a theme for further investigation in young, at-risk individuals.

5.6   Health-Risk Behaviours

An additional area of importance relative to CHD risk was that of HRB. It is clear that negative behaviour or lifestyle is closely related to poor health, and the pathogenesis of cardiovascular disease (Blair et al. 1990, p. 385). Behaviours associated with major health problems include malnutrition (under- and over-nutrition), physical inactivity, inappropriate use of alcohol, stress overload, cigarette smoking, drug abuse, unsafe sexual practices, and risk-taking practices, such as driving under the influence of alcohol, exceeding the speed limit, and the like. Equally

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alarming is the fact that many HRB in young adults track relatively well with advancing age, and impact on disease in later life (Von Ah et al., 2004).

Often poor health habits exist in unison so, for example, smokers find it difficult to perform exercise, especially vigorous exercise and, generally, also have poor diets due to the loss of taste sensitivity caused by chronic smoking (Ward et al., 2003). With advancing age, negative influences upon lifestyle tend to accumulate, such as stress and inappropriate drug use. These can operate synergistically to cause chronic diseases of lifestyle that adversely impact personal health (Groenewald et al., 2007; Tamin et al., 2003).

In the present study, most of the subjects perceived themselves to be physically fit at baseline, and considered themselves physically active enough to keep healthy. At follow-up, only 38.26% reported themselves as being physically fit. These results, even though self-reported, were in agreement with the physical fitness measurements obtained for these subjects when actually tested. Consequently, the subjects experienced a substantial change in fitness perceptions at post-test that was, presumably, largely due to the effects of the intervention programme.

$\dot{V}O_2max$ is generally considered an objective measure of physical fitness and a more reliable index than self-reported data. Viewed comprehensively, the results obtained for the subjects on HRB related to physical activity and nutrition, showed that subsequent to the intervention programme one of the learning experiences of the subjects was that they developed an accurate and more realistic perception of their actual physical activity habits and physical fitness status.

This was congruent with their actual measurements for $\dot{V}O_2max$, despite their levels of physical activity being alarmingly low.
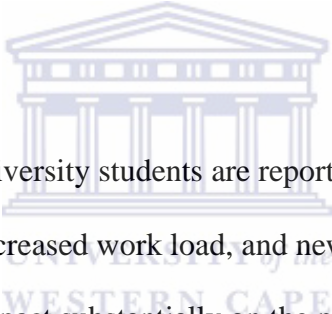
On an equally positive note, the improvement in the subjects scoring on muscular fitness at post-test reflected a better understanding of the concept of physical fitness, and the ability to translate that conceptual knowledge into quantifiable lifestyle habits.

With regard to nutrition, most participants indicated an adequate and relatively controlled diet daily. Many, nonetheless, still expressed the desire to cut down on fat intake, thus reflecting a situation of poor food choices related either to excessive caloric (energy) intake and/or improper food composition, that is, poor food preparation. Poor nutritional habits were also reported by others investigating first year university students (Irazusta et al., 2007), where shifts in dietary intakes and activity patterns were reported to reflect higher fat intakes and less physical activity, contributing to a higher prevalence of obesity (Grundy et al., 1999; Westerterp, 1999; Kruger et al., 2005).

Peculiar to the southern African region is that few black women generally view themselves as overweight (Kruger et al., 2005), and some associate thinness with HIV/AIDS (Matoti-Mvalo and Puoane, 2011). Many participants in this study, though, thought that they were overweight, particularly at post-test. This may be accounted for by the fact that many of them were more conscious and knowledgeable about body composition after having been tested for physical fitness. They are therefore better informed about the concept of body composition through the study. However, there still exists a need for more resources to be allocated to the nutritional

education of this at-risk group, not only to correct culturally-based misconceptions about overweight and obesity, but also to improve the current knowledge of risk awareness and to circumvent health problems in later life.

Equally positive were the results showing fewer participants attempting to balance food intake in order to maintain body weight at post-test. Similar results were reported by Tamim et al., (2004) who found in their study that a minimal number of participants were trying to lose weight. In the present study, it is possible that the subjects became more knowledgeable about acceptable weight control techniques and practices through the intervention programme, other than simply dieting.

The top sources of stress among university students are reported to be a change in sleeping habits, a change in eating habits, increased work load, and new responsibilities (Ross, 1999). These stresses, however, did not impact substantially on the participants in this study, since many reported favourable results for identifying and managing stress.

A review of the literature showed that many young people, especially from impoverished circumstances, are living precariously (Eriksson et al., 2007; Jones et al., 2007; Lee et al., 2007; Omoteso, 2006; Ross, 1999). Chronic exposure to numerous negative influences and stresses manifest themselves in students engaging in HRB (Omoteso, 2006; Ross, 1999). For example, 63% of students who are in a relationship were shown to have sexual intercourse (Omoteso, 2006). Condoms are never or rarely used by 35% of sexually active students. Pregnancy and

induced abortion are experienced by about 10% of sexually active females, while 1.5% of sexually active students are diagnosed with STDs (Ma et al., 2006).

In the present study, positive results were obtained for avoiding destructive habits related to smoking, alcohol and drug use. These results are very encouraging, especially when considering that sedentary behaviour among South African youth is highly associated with alcohol, tobacco, and drug use (Peltzer, 2010). Equally promising, were the high scores obtained at pre- and post-test for practising safe sex, especially given the high prevalence of HIV/AIDS in the SSA region and the devastating impact of this pandemic on public health.

Regarding personal safety, participants overwhelmingly favoured positive behaviours, such as using seat belts when driving, and not exceeding the speed limit. Here again, the positive results are laudable and most encouraging, especially considering the inordinate accident and death toll on South African roads.

However, the subjects failed to inform themselves as consumers by reading product labels and they have shown their lack of commitment to protecting the environment by continuing to refrain from recycling waste. This provides some cause for concern, especially since these subjects constitute a well-educated cohort. Steptoe et al. (2002) reported that some of the health behaviours in a relatively well-educated sector of young adult Europeans was also disappointing, and emphasized the importance of enhancing positive attitudes and lifestyles early in life.

The results for mental well-being showed that most participants had a sense of purpose in life, and were positive about the country's future.

In general, the results of the study were positive and showed that the participants displayed highly positive health behaviours from the outset that were either maintained or improved at follow-up. However, the positive health behaviours did not seem to translate into preventive action on CHD risk. Studies have shown that greater knowledge did not always result in improved lifestyle or reduced risk (Crouch, 2008, p.57). Nevertheless, except for the shortcomings in the participants' knowledge about physical activity, nutrition, and environmental protection, the results were most encouraging related to stress management and personal safety, especially considering destructive behaviours such as alcohol and drug abuse and promiscuous sexual practices (Reddy et al., 2003). Furthermore, HRB related to physical inactivity, poor physical fitness and inappropriate nutrition that present during early adulthood, may represent an early warning system for future risk and provide a window of opportunity for early identification and intervention.

5.7   Physical Activity

It is commonly assumed that a low level of habitual physical activity is directly associated with low levels of physical fitness (Blair et al., 2001). Furthermore, a low level of physical activity, like a low level of physical fitness, is considered an independent risk factor for cardiovascular morbidity and mortality (Blair et al., 1992 and 2001). However, the association between physical activity and physical fitness is not always consistent and direct, especially in children and

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adolescents (Huang and Malina (2002). The original large cohort studies of Morris et al (1958) in the 1950's and Paffenbarger et al. (1978) in the 1970's are consistent in showing that a high level of regular physical activity was inversely associated with and causally related to cardiovascular disease, even after adjustment for other major risk factors (Wood and Stefanick, 1990, p. 410).

In the present study, most of the subjects were sedentary at baseline, with a wide range of physical activity participation patterns. Following the intervention programme, the overall amount of physical activity in this group decreased even further, especially for the maximal amount of weekly physical activity. Of grave concern is the finding that most subjects did not reach the recommended 30 minutes of moderate intensity physical activity (3.0 – 5.9 METs), on four or more days in the week, and were typical of a sedentary population. Across both genders, very few subjects practiced sports or showed a total weekly physical activity score superior to the minimum of 500 MET·min·wk^{-1}, a critical threshold for health benefits (Wang et al., 2010). For all subjects, physical activity usually occurred during leisure time as opposed to being part of their formal academic programme, that is, studying sport, recreation and exercise science.

In the present study, exercise intervention in the EG and MG had a critical effect on changing physical activity status. The EG and MG were the only groups with positive changes in physical activity, and both met the minimum requirements for health benefits at post-test. This exercise effect was independent of a training (fitness) effect, as reflected in a change in $\dot{V}O_2$max. The absence of a training effect on $\dot{V}O_2$max, despite subjects being exposed to a regular exercise programme, is not without precedent and has been observed by Becque et al. (1988).

The change in physical activity in the EG and MG appeared also to have positively impacted HDL cholesterol concentration, a finding that is in agreement with that of Blair et al. (1983) who also showed a positive association between the amount of exercise performed and HDL cholesterol change, and provided strong evidence of a dose-response relationship. In the present study, however, physical activity, not physical fitness, appeared to be an independent predictor of HDL cholesterol concentration.

Despite participating in the intervention programme, most of the subjects had unfavourable levels of physical activity that pose potentially serious health risks. An innovative programme launched by the American College of Sports Medicine (ACSM) and the American Medical Association (AMA), called Exercise is Medicine, is encouraging healthcare professionals, to include regular physical activity in treatment plans for patients. This initiative recognizes the importance of regular physical activity and calls on healthcare providers to treat physical activity as a "vital sign", which will result in the assessment and recording of physical activity at every clinic visit. The results from the present study provide support for such health initiatives, particularly amongst at-risk individuals.

5.8   Shortcomings of the Study

The non-randomized method of subject recruitment, the use of self-reported results on HRB and physical activity, and the loss of statistical power due to the attrition of subjects, have all impacted on the outcomes of the present study and, thereby, the generalizability of the findings.

The present study is further weakened by insufficient dietary data that impacted the potential effects of dietary intervention on CHD risk. This did not allow for quantification of either caloric/energy intake or nutrient composition as originally intended, and prevented any comparisons being made across related studies.

In a typical quasi-experimental design, the results in one or more of the treatment groups are compared to that of the control group. One concern with this design is that repeated testing may introduce an intervention effect that acts as a motivational influence to change behaviour unexpectedly and, thereby, detract from maintaining a true control group, the so-called Hawthorne effect. Furthermore, the spillover effects due to subject interaction across treatment groups are difficult to determine and quantify and may have also impacted the final outcomes of the study.
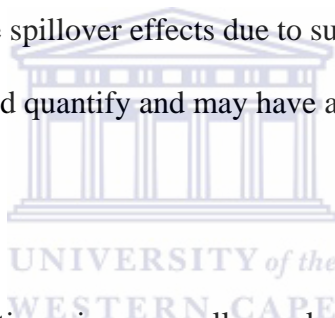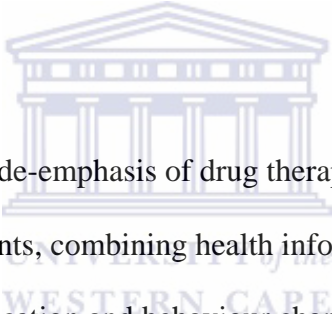
Given the use of self-reported questionnaires, a small sample size and a relatively high attrition rate, the quality of the study has, nevertheless, been maintained at a high standard, and has provided unique insights into the effects of multiple health interventions on CHD risk reduction.

5.9   Strengths of the Study

One of the noteworthy strengths of this study is the rigorous randomized control design, with all research measurements administered by trained and competent assistants.

Interventions applied in a university setting that can also be transferred to population-based settings are needed. This study provides evidence that such interventions can be integrated within the existing South African health care infrastructure. In the widely disparate and under-resourced healthcare service sector of SA, an intervention such as this one has been shown to be parsimonious, but efficient, and much needed.

Clearly shown in this study is that a multiple health intervention programme beneficially impacts CHD risk. These findings provide some empirical evidence that affordable programmes of lifestyle intervention, if more widely prescribed, can effectively reduce risk factors for CHD in moderate-risk individuals.

Studies, such as this one, support a de-emphasis of drug therapies in favour of safer, more holistic, and more effective treatments, combining health information and education, diet and exercise therapy for lifestyle modification and behaviour change. Most notably, CHD diminishes quality of life and imposes considerable economic burdens on health care systems. Thus, lifestyle modification interventions that beneficially impact CHD risk in moderate risk individuals are likely to improve personal well-being, may be more cost-effective than other primary care strategies, and certainly safer and less expensive than drug therapy.

One of the more promising outcomes of this study was a critical examination of the cumulative risk and frequencies of CHD risk factors. This not only provided a unique insight into the quantification of CHD risk change with different types of health interventions, but also presented a novel approach in communicating CHD risk that is both feasible and effective. Hopefully, this

will provide grounds for further investigation and refinement with time that can translate into unique opportunities for addressing CHD risk assessment and communication.

5.10   Recommendations

Clearly, there is a dearth of information on CHD risk and levels of physical fitness of university students. This community is not immune to the ravages of heart disease and, like others, is a population at risk that needs to be investigated further. More especially because they constitute the future leadership of the country with the potential to profoundly impact the lives of everyone.

Despite there being significant associations between indicators of physical activity and physical fitness, a large fraction of variability still remains in indicators of HRPF not accounted for by physical activity, age and gender. This suggests that other factors related in part to biology, behaviour or the environment is likely involved. Therefore, the relationship between physical activity and HRPF across the lifespan needs further study, especially in different cultural contexts with large samples of African origin, that is, a bio-cultural approach. Such research is feasible, and likely to inform best practice.

Apparently, most individuals seem to attend to risk information based upon the magnitude of personal risk and their risk in comparison to others (Steptoe et al., 2001). The former appears to be independent and additive, whereas comparing individual risk with others or the 'average' may be less important when ones' personal risk is high (Peltzer et al., 2002). Correct communication of coronary risk information can improve accuracy of risk perceptions and increase intention to

initiate prevention strategies, especially when that risk is communicated through novel and creative means, such as "cardiovascular age equivalents". Such an approach is considered clear, impressionable and may be an 'eye-opener' or 'wake-up call' for many who are at "silent" risk. It also has the potential of being an effective motivator in reducing risk, and instinctively holds much promise for further investigation. Invariably, individuals are poorly attuned to how risk accumulates over time. Thus, when exposed to risk, communication that depicts that one's 'heart age' will be higher than one's chronological age is likely to be refreshing and educational, and encourage behaviour change. This hypothesis is unproven at present, but provides intriguing grounds for future study.
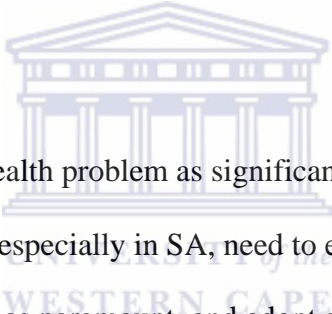
Many gaps exist in our current knowledge about promoting lifestyle change. Optimal follow-up strategies to bring about sustainable lifestyle change needs to be addressed in future studies. There is a lack of knowledge about the specific design features that determine which interventions are most effective for whom, that is, young versus old, male versus female, high versus low socioeconomic status, and black versus white, and incumbent cost implications. More knowledge about treatment efficacy that is sensitive to sex, race, ethnicity, socioeconomic status, and disability is needed.

There is inconclusive evidence from previous trials of a benefit in the use of brief interventions for risk factor modification in subjects with CHD (Fernandez et al., 2007). This study, however, supports those trials showing that brief structured interventions for participants with CHD has beneficial effects on risk factor modification and, consequently, on the progression of CHD. However, additional trials using randomized, controlled samples of large size are recommended

in order to demonstrate whether these findings are consistent and conclusive. Bearing in mind that health promotion advice and campaigns have not produced substantial changes in CVD prevalence, behaviour modification through multiple intervention strategies in subjects at increased risk offers considerable benefits, and holds much promise for further investigation. This study should be viewed as an initial step toward examining the longitudinal effects of multiple behaviour interventions on CHD risk and health-related quality of life of university students. Multiple behaviour interventions for individuals are rare, but hold much promise to impact public health cost-effectively.

5.11   Conclusion

CHD will continue to be a public health problem as significant numbers invariably succumb to this condition. Healthcare systems, especially in SA, need to establish health priorities that target primary prevention of heart disease as paramount, and adopt a community-centred approach that shifts current paradigms to emphasize people of all ages and healthy lifestyles in all communities. Such an approach that focuses on early detection, effective communication and early intervention is likely to be more receptive and impactful. This study, unequivocally, supports the use of multiple interventions in subjects with CHD.

In conclusion, the present study shows that a number of first year university students are at risk for CHD. More importantly, a multiple health behaviour intervention strategy was effective in reducing CHD risk in students stratified as moderate risk. Furthermore, the impact of the interventions were dose-response related, in which the single treatments produced modest

improvements in CHD risk, but when combined were additive and significantly reduced CHD risk. The impact of multiple interventions on HRPF, though positive for combined treatments, is inconclusive and awaits further investigation. No noteworthy effects were shown for multiple interventions on HRB and physical activity.

Due to the limited duration of the present study, the long-term effects of multiple interventions were not shown, but the short-term changes are most promising and augur well for future research in this area.

For many, heart disease is as abstract a concept as it is as insidious a process, therefore reducing its risk is work that invariably needs to be carried out over many years. The optimum time, needs to start early, preferably in childhood, and continue throughout life. CHD is not an intractable, permanent condition but rather one that is amenable to change, especially when targeted in a collaborative, multidisciplinary approach that is cost-effective, population-based, and sustainable. As a consequence there is an obligation to take pre-emptive action from all sectors of society, inclusive of education, health, and sport, failing which present profiles of heart disease risk are destined to persist and probably worsen in future, and serve as a legacy, if not an indictment, against current leadership.

Bibliography

A Report of the Surgeon General. (2011). *Overview of prevention*. Retrieved May 16, 2011 from http://www.surgeongeneral.gov/library/mentalhealth/chapter2/sec5.html

Aboderin, I., Kalache, A., Ben-Shlomo, Y., Lynch, J. W., Yajnik, C. S., Kuh, D. and Yach, D. (2002) *Life Course Perspectives on Coronary Heart Disease, Stroke and Diabetes: Key Issues and Implications for Policy and Research*. Geneva, World Health Organization.

Ackermann, R. T., Deyo, R. A. and Logerfo, J. P. (2005). Prompting primary providers to increase community exercise referrals for older adults: a randomized trial. *Journal of the American Geriatric Society, 53,* 283-289.

Adelekan, M. L., Abiodun, O. A., Obayan, A. O., Oni, G. and Ogunremi, O. O. (1992)**.** The prevalence and pattern of substance use among undergraduates in a Nigerian University. *Drug and Alcohol Dependence, 29*(3), 255-261.

Ainsworth, B. E., Jacobs, Jr., D. R. and Leon, A. S. (1993). Validity and reliability of self-reported physical activity status: the lipid research clinics questionnaire. *Medicine and Science in Sports and Exercise, 25(*1), 92-98.

Aittasalo, M., Miilumpalo, S., Kukkonen-Harjula, K. and Pasanen, M. (2006). A randomized intervention of physical activity promotion and patient self-monitoring in primary health care. *Preventive medicine, 42,* 40-46.

Akinboboye, O., Idris, O., Akinboboye, O. and Akinkugbe, O. (2003). Trends in coronary artery disease and associated risk factors in sub-Saharan Africans. *Journal of Human Hypertension, 17*, 381-387.

Alberts, M., Urdal, P., Steyn, K., Stensvold, I., Tverdal, A., Nel, J. H. and Steyn, N. P. (2005). Prevalence of cardiovascular diseases and associated risk factors in a rural black population of South Africa. *European Journal of Cardiovascular Prevention and Rehabilitation, 12*(4), 347-354.

American College of Sports Medicine. (2006a). *ACSM's Advanced Exercise Physiology*. Lippincott, Williams & Wilkins, Philadelphia.

American College of Sports Medicine. (2006b). *ACSM's Guidelines for Exercise Testing and Prescription*. Seventh edition. Lippincott, Williams & Wilkins, Philadelphia.

American College of Sports Medicine. (2006c). *ACSM's Resource Manual for Guidelines for Exercise Testing and Prescription*. 5th edition. Lippincott, Williams and Wilkins Publishers, Phildelphia.

American Heart Association. (2003). *Heart attack, stroke, and cardiac arrest warning signs*. Retrieved May 10, 2008, from http://www.americanheart.org/presenter.jhtml?identifier=3053

Anderson, K. M., Wilson, P. W., Odell, P. M. and Kannel, W. B. (1991). An updated coronary risk profile. A statement for health professionals. *Circulation, 83,* 356-362.

Andrews, B.C., Ruhling, R.O., Burrell, E., Basset, S. & Sands, B. (1985). Physical fitness of South African and American young adults. *South African Journal for Research in Sport, Physical Education and Recreation, 8*(2), 23-33.

Arena, R., Myers, J. and Guazzi, M. (2010). The future of aerobic exercise testing in clinical practice: Is it the ultimate vital sign? *Future Cardiology, 6*(3), 325-342.

Artinian, N. T., Fletcher, G. F., Mozaffarian, D., Kris-Etherton, P., Van Horn, L., Lichtenstein, A. H., Kumanyika, S., Kraus, W. E., Fleg, J. L., Redeker, N. S., Meininger, J. C., Banks, J., Stuart-Shor, E. M., Fletcher, B. J., Miller, T. D., Hughes, S., Braun, L. T., Kopin, L. A., Berra, K., Hayman, L. L., Ewing, L. J., Ades, P. A., Durstine, J. L., Houston-Miller, N. and Burke, L. E. (2010). Interventions to promote physical activity and dietary lifestyle changes for cardiovascular risk factor reduction in adults: a scientific statement from the American Heart Association. *Circulation*, *122*, 406-441.

Ashen, D. (2010). Cost-effective prevention of coronary heart disease. *Journal for Nurse Practitioners, 6*(10), 754-764.

Australian Sports Commission (2000). *Physiological Tests for Elite Athletes*. Human Kinetics Publishers, Champaign, Illinois.

Barker, D. J. P. (1995). Fetal origins of coronary heart disease. *British Medical Journal, 311*, 171-74.

Bayne-Smith, M., Fardy, P. S., Azzollini, A., Magel, J., Schmitz, K. H. and Agin, D. (2004). Improvements in heart health behaviours and reduction in coronary artery disease risk factors in urban teenage girls through a school-based intervention: The PATH program. *American Journal of Public Health, 94* (9), 1538-1543.

Beranova, E. and Sykes, C. (2007). Review. A systematic review of computer-based softwares for educating patients with coronary heart disease. *Patient Education and Counseling, 66*, 21-28.

Blair, S.N., and Bouchard, C. (1999) Introductory comments for the consensus on physical activity and obesity. *Medicine and Science in Sports and Exercise, 31* (11), S498-S501.

Blair, S. N., Cheng, Y. and Holder, J. S. (2001). Is physical activity or physical fitness more important in defining health benefits? *Medicine and Science in Sports and Exercise, 33*(6), S379-S399.

Blair, S. N., Kohl, H. W., Gordon, N. F. and Paffenbarger, Jr., R. S. (1992). How much physical activity is good for health? *Annual Review of Public Health, 13*, 99-126.

Bloom, D. E., Cafiero, E. T., Jané-Llopis, E., Abrahams-Gessel, S., Bloom, L. R., Fathima, S., Feigl, A. B., Gaziano, T., Mowafi, M., Pandya, A., Prettner, K., Rosenberg, L., Seligman, B., Stein, A., and Weinstein, C. (2011). *The Global Economic Burden of Non-communicable Diseases*. Retrieved on November 15, 2011 from http://www.weforum.org/EconomicsOfNCD

Borkon, L., Baird, D. M. and Siff, M. (1983). Tobacco smoking among students at the University of the Witwatersrand, Johannesburg. *South African Medical Journal, 64*(21), 809-812.

Bradshaw, D., Groenewald, P., Laubscher, R., Nannan, N., Nojilana, B., Norman, R., Pieterse, D. and Schneider, M. (2003). Initial estimates from the South African National Burden of Disease Study, 2000. *Medical Research Council Policy Brief No. 1*.

Bradshaw, D., Schneider, M., Norman, R. and Bourne, D. (2006). Mortality patterns of chronic disease of lifestyle in South Africa. In: Steyn K, Fourie J, Temple N (eds). *Chronic Diseases of Lifestyle in South Africa: 1995-2005*, Technical Report, Cape Town: South African Medical Research Council. [On line] Available:http://www.mrc.ac.za/chronic/cdl1995-2005.pdf.

Breier, M. (2007). *Pathways through higher education to the labour market: factors affecting student retention, graduation, and destination. A case study report.* University of the Western Cape. Human Sciences Research Council (HSRC).

Burke, S., Carron, A. and Eys, M. (2005). Physical activity context: Preferences of university students. *Psychology of Sport and Exercise, 7*, 1-13.

Calfas, K. J., Long, B. J., Sallis, J. F., Wooten, W. J., Pratt, M. and Patrick, K. (1996). A controlled trial of physician counseling to promote the adoption of physical activity. *Preventive Medicine, 25*, 225-233.

Cardinal, B J., Esters, J. and Cardinal, M. K. (1996). Evaluation of the revised physical activity readiness questionnaire in older adults. *Medicine and Science in Sports and Exercise, 28*(4), 468-72.

Carlyon, R. G., Gore, C. J., Woolford, S. M. and Bryant, R. W. (1996). Calibrating Harpenden skinfold calipers. In: K. I. Norton & T. Olds (Eds.). *Anthropometrica*. Sydney, University of New South Wales Press.

Caspersen, C. J., Powell, K. E. and Christenson, G. M. (1985). Physical activity, exercise, and physical fitness: definitions and distinctions for health-related research. *Public Health Reports, 100*, 126-131.

Chatterjee, P., Banerjee, A. K., Das, P. and Debnath, P. (2010). A regression equation for the estimation of VO2max in Nepalese male adults. *Journal of Human Sport and Exercise, 5*(2), 127-133.

Chatterjee, P., Banerjee, A. K., Das, P., Debnath, P. and Chatterjee, P. (2008). Validity of 20 meter multi-stage shuttle run test for prediction of maximum oxygen uptake in Indian female university students. *Kathmandu University Medical Journal, 6*(2), 176-180.

Choi, Y. and Choi, Ja. (2007). Their Side of the Story: Understanding Health Risk Behaviours from the Participants' Perspective. *Journal of School Health*, *75*, 229-231.

Coetsee, M.F. (2003). An assessment of the physical fitness and coronary heart disease risk factors of white females in the Zululand area that attended health and fitness clubs. *South African Journal for Research in Sport, Physical Education and Recreation, 25*(1), 13-21.

Collins, K., Dantico, M., Shearer, N. and Mossman, K. (2004). Heart disease awareness among college students. *Journal of Community Health, 29*(5), 405-420.

Colditz, G. A., Manson, J. E. and Hankinson, S. E. (1997). The nurses' health study: 20-year contribution to the understanding of health among women. *Journal of Women's Health, 6*(1), 49-62.

Committee of Experts on Sports Research EUROFIT. (1993). *Handbook for the EUROFIT Tests of Physical Fitness*. Council of Europe, Strasburg, Germany.

Cooper Institute for Aerobics Research. (1999). *FITNESSGRAM test administration manual*. Human Kinetics, Champaign.

Cooper, S-M., Baker, J. S., Tong, R. J., Roberts, E. and Hanford, M. (2005). The repeatability and criterion related validity of the 20 m multistage fitness test as a predictor of maximal oxygen uptake in active young men. *British Journal of Sports Medicine, 39*(4), 19-26.

Coopoo, Y. and Naidoo, K. (2003). Fitness levels of South African youth of Indian descent - 1977 and 1997. *South African Journal of Sports Medicine, 5*(1), 4-8.

Corbin, C. B., Welk, G. J., Corbin, W. R. and Welk, K. A. (2006). *Concepts of Fitness and Wellnes. A comprehensive lifestyle approach*. 6th Edition. McGraw-Hill Companies, Boston.

Craig, C. L., Marshall, A. L., Sjöström, M., Bauman, A. E., Booth, M. L., Anisworth, B. E., Pratt, M., Ekelund, U., Yngve, A., Sallis, J. F. and Oja, P. (2003). International physical activity questionnaire: 12-country reliability and validity. Medicine and Science in Sports and Exercise, 35(8), 1381-1395.

Critchley, J. and Capewell, S. (2003). Mortality risk reduction associated with smoking cessation in patients with coronary heart disease. *Journal of the American Medical Association, 290*, 86-97.

Day, C. and Bailey, C. J. (2011). Obesity in the pathogenesis of type 2 diabetes. *British Journal of Diabetes and Vascular Disease, 11*(2), 55-61.

Demyanets, S., Huber, K. and Wojta, J. (2011). Inflammation and the cardiovascular system. *European Surgery, 43*(2), 78-89.

Dipietro, L. (1999). Physical activity in the prevention of obesity: current evidence and research issues. *Medicine and Science in Sports and Exercise, 31*(11), S542-S546.

Donovan, R. J., Jones, S., D'Arcy, C., Holman, J. and Corti, B. (1998). Assessing the reliability of a stage of change scale. *Health Education Research, 13*(2), 285-291.

Dunn, A. L., Marcus, B. H., Kampert, J. B., Garcia, M. E., Kohl III, H. W. and Blair, S. N. (1997). Reduction in cardiovascular disease risk factors: 6-month results from project active. *Preventive Medicine, 26*, 883-892.

Du Pré, R.H. (1996) *Historical positioning*. In: Thomas, Cornelis C. (1997) *Wakker wakker en aan the brand; Waarneminge van a studente-aktivis, UWK 1976*. Mayibuye History and Literature Series, No.75.

Eakin, E. G., Lawler, S. P., Vandelanotte, C. and Owen, N. (2007). Telephone interventions for physical activity and dietary behaviour change: A systematic review. *American Journal of Preventive Medicine, 32*(5), 419-434.

Ebrahim, S. and Smith, G. (1997). Systematic review of randomized controlled trials of multiple risk factor interventions for preventing coronary heart disease. *British Medical Journal, 314,* 1666-1691.

Ebrahim, S., Taylor, F., Ward. K., Beswick, A., Burke, M. and Davey-Smith, G. (2011). *Multiple risk factor interventions for primary prevention of coronary heart disease (Review).* Retrieved on March 28, 2011 from http://www.thecochranelibrary.com

Epstein, L. (1998). Integrating theoretical approaches to promote physical activity. *American Journal of Preventive Medicine, 15*(4), 257-265.

Eriksson, .K. M., Westborg, C. J., and Eliasson, M. C. (2006). A randomized trial of lifestyle intervention in primary healthcare for the modification of cardiovascular risk factors. *Scandinavian Journal of Public Health, 34*(5), 453-61.

Eriksson, J., Dellve, L., Eklök, M., and Hagsberg, M. (2007). Early inequalities in excellent health and performance among young adult women and men in Sweden. *Gender Medicine, 4* (2), 172-182.

Eriksson, K. M., Franks, P. W. and Eliasson, M. (2009) A 3-year randomized trial of lifestyle intervention for cardiovascular risk reduction in the primary care setting: The Swedish Björknäs study. PLoS ONE 4(4): e5195. doi:10.1371/journal.pone.0005195.

Escobar, H. (2002). Hypertension and coronary heart disease. *Journal of Human Hypertension, 16*(1), S61-S63.

Fernandez, R., Griffiths, R., Everett, B., Davidson, P., Salamonson, Y. and Andrew, S. (2007). Effectiveness of brief structured interventions on risk factor modification for patients with coronary heart disease: a systematic review. *International Journal of Evidence-Based Healthcare, 5*(4), 370-405.

Fleming, P. and Godwin, M. (2008). Lifestyle interventions in primary care: Systematic review of randomized controlled trial. *Canadian Family Physician, 54*, 1706-1713.

Franklin, B. A. and Cushman, M. (2011). Recent advances in preventive cardiology and lifestyle medicine: A themed series. *Circulation*, *123,* 2274-2283.

Frantz, J. M. (2004). *Physical inactivity among high school learners in Belhar- A public concern*. Unpublished Doctor of Philosophy thesis. University of the Western Cape.

Frost, R. (1992). Cardiovascular risk modification in the college student: Knowledge, attitudes, and behaviours. *Journal of General Internal Medicine, 7*, 317-320.

Garber, C. E., Blissmer, B., Deschenes, M. R., Franklin, B. A., Lamonte, M. J., Lee, I-Min., Nieman, D. C. and Swain, D. P. (2011). Quantity and quality of exercise for developing and maintaining cardiorespiratory, musculoskeletal, and neuromotor fitness in apparently healthy adults: guidance for prescribing exercise. *Medicine and Science in Sports and Exercise, 43*(7), 1334-1359.

Gaziano, T. (2005). Cardiovascular disease in the developing world and its cost-effective management. *Circulation, 112*, 3547-3553.

Gaziano, J. M. (1998). Primary and secondary prevention of coronary heart disease. *Cardiology Rounds, 2*(8), 1-8.

Gaziano, J. M., Hennekens, C. H., O'Donnell, C. J., Breslow, J. L. and Buring, J. E. (1997). Fasting triglycerides, high-density lipoprotein, and risk of myocardial infarction. *Circulation, 96,* 2520-2525.

Gibney, A., Moore, N., Murphy, F. and O'Sullivan, S. (2011). The first semester of university life; 'will I be able to manage it at all?' *Higher Education, 62*, 351-366.

Gibson, A., Broomhead, S., Lambert, M. I. and Hawley, J. A. (1998). Prediction of maximal oxygen uptake from a 20-m shuttle run as measured directly in runners and squash players. *Journal of Sports Sciences, 16*(4), 331-335.

Green, B. B., McAfee, T., Hindmarsh, M., Madsen, L., Caplow, M. and Buist, D. (2002). Effectiveness of telephone support in increasing physical activity levels in primary care patients. *American Journal of Preventive Medicine, 22,* 177-183.

Griffin, J. C. (2006). *Client-Centred Exercise Prescription*. Second edition. Human Kinetics Publishers, Champaign, Illinois.

Groenewald, P., Vos, T., Norman, R., Laubscher, R., Van Walbeek, C., Saloojee, Y., Sitas, F. and Bradshaw, D. (2007). Estimating the burden of disease attributable to smoking in South Africa in 2000. *South African Medical Journal, 97*, 674-681.

Grundy, S. M., Blackburn, G., Higgins, M., Lauer, R., Perri, M. G. and Ryan, D. (1999). Physical activity in the prevention and treatment of obesity and its comobidities. *Medicine and Science in Sports and Exercise, 31*(11), S502-S508.

Gyurcsik, N. C., Spink, K. S., Bray, S. R., Chad, K. and Kwan, M. (2006). An ecologically-based examination of barriers to physical activity in students from grade seven through first-year university. *Journal of Adolescent Health, 38*, 704-711.

Haase, A., Steptoe, A., Sallis, J. F. and Wardle, J. (2004). Leisure-time physical activity in university students from 23 countries: associates with health beliefs, risk awareness, and national economic development. *Preventive Medicine, 39*, 182-190.

Haskell, W. L., Alderman, E. L., Fair, J. M., Maron, D. J., Mackey, S. F., Superko, H. R., Williams, P. T., Johnstone, I. M., Champagne, M. A. and Krauss, R. M. (1994). Effects of intensive multiple risk factor reduction on coronary atherosclerosis and clinical cardiac events in men and women with coronary artery disease. The Stanford coronary risk intervention project (SCRIP) *Circulation*, *89*, 975-990.

Hedberg, G. E., Wikström-Frisén, L. and Janlert, U. (1998). Comparison between two programmes for reducing the levels of risk indicators of heart diseases among male professional drivers. *Occupational and Environmental Medicine, 55*, 554-561.

Hedblad, B., Ogren, M., Isacsson, S. and Janzon, L. (1997). Reduced cardiovascular mortality risk in male smokers who are physically active. *Archives of Internal Medicine, 157*(8), 893-899.

Helfand, M., Buckley, D. I., Freeman, M., Fu, R., Rogers, K., Fleming, C. and Humphrey, L. L. (2009). Emerging risk factors for coronary heart disease: A summary of systematic reviews conducted for the U.S. Preventive Services Task Force. *Annals of Internal Medicine, 151*, 496-507.

Hemingway, H. and Marmot, M. (1999). Psychosocial factors in the aetiology and prognosis of coronary heart disease: systematic review of prospective cohort studies. *British Medical Journal, 318*, 1460-1467.

Henderson, A. (1996). Coronary heart disease: overview. *Lancet, 348*, S1-S4.

Herrmann, S. and Paul, M. (2001). The genetics of coronary heart disease. *European Journal of Internal Medicine, 12,* 2-10.

Hills, A. P., King, N. A. and Byrne, N. M. (2007). *Children, obesity, and exercise: prevention, treatment, and management of childhood and adolescent obesity*. International Studies in Physical Education and Youth Sport Series. Routledge, Taylor, and Francis Group, London.

Holborn, L. and Eddy, G. (2011).*First steps to healing the South African family*. A paper produced by the South African Institute of Race Relations.

Howley, E. T. and Franks, B. D. (1997). *Health Fitness Instructor's Handbook*. Third edition. Human Kinetics Publishers, Champaign, Illinois.

Hudon, C., Fortin, M. and Soubhi, H. (2008). Single risk factor interventions to promote physical activity among patients with chronic diseases. A Systematic review. *Canadian Family Physician, 54,* 1130-1137.

IPAQ. (2005). Retrieved Ocotber 12, 2007 from www.ipaq.ki.se

Ingelsson, E., Schaefer, F. J., Coutois, J. H., McNamara, J. R., Sullivan, L., Keyes, M. J., Pencina, M. J., Schoonmaker, C., Wilson, P. W., D'Agostino, R. B. and Vasan, R. S. (2007). Clinical utility of different lipid measures for prediction of coronary heart disease in men and women. *Journal of American Medical Association, 298*(7), 776-785.

Irazusta, A., Hoyos, I., Irazusta, J., Ruiz, F., Diaz, E. and Gil, J. (2007). Increased cardiovascular risk associated with poor nutritional habits in first-year university students. *Nutrition Research, 27*, 387-394.

Iso, H. (2011). Lifestyle and cardiovascular disease in Japan. *Journal of Atherosclerosis and Thrombosis, 18*(2), 83–88.

Johnson, S. S., Paiva, A. L., Cummins, C. O., Johnson, J. L., Dyment, S . J., Wright, J. A., Prochaska, J. O., Prochaska, J. and Sherman, K. (2008). Transtheoretical Model-based multiple behaviour intervention for weight management: Effectiveness on a population basis. *Preventive Medicine, 46*, 238-246.

Jones, D. E. M., Bester, P., Ghebrehmedhin-Asihel, S. and Humphreys, A. (2007). We know what makes us vulnerable: female learners, their health and its relationship to physical education and school sports. *African Journal for Physical, Health Education, Recreation and Dance, 13*(2), 173-183.

Jourkesh, M., Sadri, I., Ojagi, A. and Sharanavard, A. (2011). Comparison of Physical fitness level among the students of IAU, Shabestar Branch. *Annals of Biological Research, 2*(2), 460-467.

Kahn, K. (2011). Population health in South Africa: Dynamics over the past two decades. *Journal of Public Health Policy, 32*(1), S30-S36.

Kahn, K., Garenne, M. L., Collinson, M. A. and Tollman, S. M. (2007). Mortality trends in a new South Africa: hard to make a fresh start. *Scandinavian Journal of Public Health, 35*(69), 26-34.

Katzmarzyk, P. T., Malina, R. M. and Bouchard, C. (1999). Physical activity, physical fitness, and coronary heart disease risk factors in youth: The Quebec family study. *Preventive Medicine, 29***,** 555-562.

Kazi, H .A. and Coopoo, Y. (2010). Physical activity, alcohol use, smoking and dietary profiles of a cohort of university students. *African Journal for Physical, Health Education, Recreation and Dance, 16*(4), 104-118.

Kemper, H. C. G., Koppes, L. L. J., de Vente, W., van Lenthe, F. J., van Mechelen, W., Twisk, J. W. R. and Post, G. B. (2002). Effects of health information in youth and young adulthood on risk Factors for chronic diseases - 20-year study results from the Amsterdam growth and health longitudinal study. *Preventive Medicine, 35*, 533-539.

Kerse, N., Elley, C. R., Robinson, E. and Arroll, B. (2005). Is physical activity counseling effective for older people? A cluster, randomized control trial in primary care. *Journal of the American Geriatric Society, 53*, 1951-1956.

King, A. C., Haskell, W. L., Taylor, C. B., Kraemer, H. C. and Debusk, R. F. (1991). Group- vs home-based exercise training in healthy older men and women: A community-based clinical trial. *Journal of American Medical Association, 266*, 1535-1542.

Kinosian, B., Glick, H. and Garland, G. (1994). Cholesterol and coronary heart disease: predicting risks by levels and ratios. *Annals of Internal Medicine, 121*, 641-647.

Kirkland, S. A., MacLean, D. R., Langille, D. B., Joffres, M. R., MacPherson, K. M., and Andreou, P. (1999). Knowledge and awareness of risk factors for cardiovascular disease among Canadians 55 to 74 years of age: results from the Canadian heart health surveys, 1986 -1992. *Canadian Medical Association Journal, 161*(8), S10-6.

Kruger, H. S., Puoane, T., Senekal, M. and van der Merwe, T. (2005). Obesity in South-Africa: challenges for government and health professionals. *Public Health Nutrition, 8*(5), 491-500. Kvaavik, E., Klepp, K-I., Tell, G., Meyer, H. E. and Batty, G. D. (2001). Physical fitness and physical activity at age 13 years as predictors of cardiovascular disease risk factors at ages 15, 25, 33, and 40 years: extended follow-up of the Oslo youth study. *Pediatrics, 123*(1), e80-e86.

Léger, L. A., Mercier, D., Gadoury, C. and Lambert, J. (1988). The multistage 20-metre shuttle run test for aerobic fitness. *Journal of Sports Science, 6*(2), 93-101.

Leeder, S., Raymond, S. and Greenberg, H. (2004). *A Race against time. The challenge of cardiovascular disease in developing economies.* Trustees of Columbia University, New York.

Lee, I. and Skerrett, P. J. (2001). Physical activity and all-cause mortality: what is the dose-response relation? *Medicine and Science in Sports and Exercise, 33*(6), supplement, S459-S471.

Lee, Y., Chien, K. and Chen, H. (2007) Lifestyle risk factors associated with fatigue in graduate students. *Journal of the Formosan Medical Assoction, 106*, 565-572.

Leon, A. S. and Bronas, U. G. (2009). Dyslipidemia and risk of coronary heart disease: role of lifestyle approaches for its management. *American Journal of Lifestyle Medicine, 3*: 257-273.

Levenson, J. W., Skerrett, P. J. and Gaziano, M. (2002*).* Reducing the global burden of cardiovascular disease: the role of risk factors. *Preventive Cardiology, 5*, 188-199.

Lie, D. (2007). *Lifestyle counseling program may help reduce risk of developing type 2 diabetes.* Retrieved October 6, 2007 from http://www.mediscape.com/viewarticle/563637?/src=mp

Little, P., Dorward, M. and Gralton, S. (2004). A randomized controlled trial of three pragmatic approaches to initiate increased physical activity in sedentary patients with risk factors for cardiovascular disease. *British Journal of General Practice, 54*, 189-195.

Lopez, A. D., Mathers, C. D., Ezzati, M., Jamison, D. T. and Murray, C. J. L. (2006). Global and regional burden of disease and risk factors, 2001: systematic analysis of population health data. *Lancet, 367*, 1747-57.

Ma, Q., Ono-Kihara, M., Cong, L., Xu, G., Zamani, S., Ravari, S.M. and Kihara, M. (2006). *Sexual behaviour and awareness of Chinese university students in transition with implied risk of sexually transmitted diseases and HIV infection: A cross-sectional study*. Retrieved October 6, 2007 from http://www.biomedcentral.com/1471-2458/6/232

MacDonald, S., Joffres, M. R., Stachenko, S., Horlick, L. and Fodor, G. (1992). Multiple cardiovascular disease risk factors in Canadian adults. *Canadian Medical Association Journal, 146*(11), 2021-2029.

Mak, K., Ho, S., Lo, W., Thomas, G. N., McManus, A. M., Day, J. R. and Lam, T. (2010). Health-related physical fitness and weight status in Hong Kong adolescents. *Biomedical Central Public Health, 10*, 88-93.

Marfell-Jones, M., Olds, T., Stewart, A., and Carter, J.E.L. (2006). *International standards for anthropometric assessment*. International Society for the Advancement of Kinanthropometry Publishers, Australia.

Mark, D. B., Van de Werf, F. J., Simes, R. J., White, H. D., Wallentin, L. C., Califf, R. M. and Armstrong, P. W. (2007). Cardiovascular disease on a global scale: defining the path forward for research and practice. *European Heart Journal, 28*, 2678-2684.

Marshall, J. and Hardman, K. (2000). The State and Status of Physical Education in Schools in International Context. *European Physical Education Review, 6*(3), 203-229.
Matoti-Mvalo, T. and Puoane, T. (2011). Perceptions of body size and its association with HIV/AIDS. *South African Journal of Clinical Nutrition, 24*(1), 40-45.

Mayosi, B. M., Flisher, A. J., Lalloo, U. G., Sitas, F., Tollman, S. M. and Bradshaw, D. (2009). The burden of non-communicable diseases in South Africa. *The Lancet, 374 (9693*), 934-947.

McArdle, W. D., Katch, F. I. and Katch, V. L. (2001). *Exercise Physiology: Energy, Nutrition, and Human Performance.* Fifth edition. Lippincott, Williams & Wilkins, Philadelphia.

McGill, H. C., McMahan, C. A. and Zieske, A. W. (2000). Associations of coronary heart disease risk factors with the intermediate lesion of atherosclerosis in youth. The pathobiological determinants of atherosclerosis in youth (PDAY) research group. *Arteriosclerosis, Thrombosis and Vascular Biology, 20*(8), 1998-2004.

Meigs, J. B., Nathan, D. M., D'Agostino SR., R. B. and Wilson, P. W. F. (2002). Fasting and post-challenge glycemia and cardiovascular disease risk - The Framingham offspring study. *Diabetes Care, 25*, 1845-1850.

Mokdad, A. H., Ford, E. S., Bowman, B. A., Dietz, W. H., Vinicor, F., Bales, V. S. and Marks, J. S. (2003). Prevalence of obesity, diabetes, and obesity-related health risk factors, 2001. *Journal of the American Medical Association, 289*, 76-9.

Mollentze, W. F., Moore, A. J., Steyn, A. F. Joubert, G., Steyn, K., Oosthuizen, G. M. and Weich, D. J. V. (1995). Coronary heart disease risk factors in a rural and urban Orange Free State black Population. *South African Medical Journal, 85,* 90-96.

Monyeki, M. A., Koppes, L. L., Kemper, H. C., Monyeki, K. D., Toriola, A. L., Pienaar, A. E. and Twisk, J. W. (2005). Body composition and physical fitness of undernourished South African rural primary school children. *European Journal of Clinical Nutrition, 59*, 877-883.

Morris, J. N. and Crawford, M. D. (1958). Coronary heart disease and physical activity of work. *British Medical Journal, 2*, 1485-1496.

Mozaffarian, D., Hao, T., Rimm, E. B., Willett, W. C. and Hu, F. B. (2011). Changes in diet and lifestyle and long-term weight gain in women and men. *New England Journal of Medicine, 364*(25), 2392-404.

Murray, C. J. L. and Lopez, A. D. (1997a). Alternative projections of mortality and disability by cause 1990–2020: Global Burden of Disease Study. *Lancet, 349*, 1498-1504.

Murray, C. J. L. and Lopez, A. D. (1997b). Global mortality, disability, and the contribution of risk factors: Global Burden of Disease Study. *Lancet, 349*, 1436-42.

Myers, J. (2003). Exercise and cardiovascular health. *Circulation, 107*, e2-e5.

Nainggolan, L. (2011). *CRP, other biomarkers, not clinically useful to predict cardiovascular disease risk*. Retrieved on July 30, 2011 from http://www.medscape.com/viewarticle/705124

Naslund,G. K., Fredrikson, M., Hellenius, M-L. and de Faire, U. (1996). Effect of diet and physical exercise intervention programmes on coronary heart disease risk in smoking and non-smoking men in Sweden. *Journal of Epidemiology and Community Health, 50*, 131-136.

National Institutes of Health, National Heart, Lung, and Blood Institute. (2001). ATP *III Guidelines At-A-Glance Quick Desk Reference*. National Cholesterol Education Program. NIH Publication No. 01-3305.

Navas-Nacher, E. L., Colangelo, L., Beam, C., and Greenland, P. (2001). Risk factors for coronary heart disease in men 18 to 39 years of age. *Annals of Internal Medicine, 134*, 433-439.

Nghiem, H.T. (2007). *Raised blood pressure and BMI in midlife predicts heart failure in later life*. Retrieved on October 6, 2007 from http://www.medscape.com/viewarticle/563680?src=mp

Nilsson, P. M., Klasson, E-B., Nyberg, P. (2001). Life-style intervention at the worksite — reduction of cardiovascular risk factors in a randomized study. *Scandinavian Journal of Work and Environmental Health, 27*(1), 57-62.

Noar, S. N., Chabot, M. and Zimmerman, R. S. (2008). Applying health behaviour theory to multiple behaviour change: considerations and approaches. *Preventive Medicine, 46*, 275-280.

Norman, R., Bradshaw, D., Schneider, M., Pieterse, D. and Groenewald, P. (2006). *Revised burden of disease estimate for the comparative risk factor assessment, South Africa 2000*. Cape Town: Medical Research Council (MRC).

Norman, R., Bradshaw, D., Steyn, K., Gaziano, T. and the South African Comparative Risk Assessment Collaborating Group. (2007). Estimating the burden of disease attributable to High Cholesterol in South Africa in 2000. *South African Medical Journal, 97*, 708-715.

Norton, K., and Olds, T. (eds.) (1996). *Anthropometrica*. University of New South Wales Press, Sydney.

Oelofse, A., Jooste, P. L., Steyn, K., Badenhorst, C. J., Lombard, C., Bourne, L. and Fourie, J. (1996). The lipid and lipoprotein profile of the urban black South African population of the Cape Peninsula – the BRISK study. *South African Medical Journal, 86*(2), 162-166.

Ogden, C. L., Lamb, M. M., Carroll, M. D. and Flegal, K. M. (2010a). Obesity and Socioeconomic Status in Children and Adolescents: United States, 2005-2008. *National Center for Health Statisitics (NCHS) Data Brief, No. 51*, 1-8.
Ogden, C. L., Lamb, M. M., Carroll, M. D. and Flegal, K. M. (2010b). Obesity and Socioeconomic Status in Adults: United States, 2005-2008. *National Center for Health Statisitics (NCHS) Data Brief, No. 50*, 1-8.

Ojikutu, R. K. and Adeleke, I. A. (2010). Tracing the path of substance use among students of tertiary institutions in Lagos State, Nigeria. *International Journal of Academic Research, 2*(1), 216-223.

Omar-Fauzee, M. S., Ahmad, R., Rashid, S., Din, A. and Hod, H. (2010). Sport science students' fitness level at university of Malaysia Sabah. *European Journal of Social Sicences, 12*(4), 538-544.

Omoteso, B.A. (2006). A Study of the sexual behaviour of university undergarduate students in southwestern Nigeria. *Journal of Social Sciences, 12*(2), 129-133.

Onat, A., Avci, G. S., Barlan, M. M., Uyarel, H., Uzunlar, B., and Sansoy, V. (2004). Measures of abdominal obesity assessed for visceral adiposity and relation to coronary risk. *International Journal of Obesity and Related Metabolic Disorders, 28*(8), 1018-25.

Ornish, D., Scherwitz, L. W., Billings, J. H., Gould, K. L., Merritt, T. A., Sparler, S., Armstrong, W. T., Ports, T. A. Kirkeeide, R. L., Hogeboom, C., and Brand, R. J. (1998). Intensive lifestyle changes for reversal of coronary heart disease. *Journal of American Medical Association, 280*, 2001-2007.

Ortega, F. B., Artero, E. G., Ruiz, J. R., Vicente-Rodriguez, G., Bergman, P., Hagströmer, M., Ottevaere, C., Nagy, E., Konsta, O., Rey-Lopez, J. P., Polito, A., Dietrich, S., Plada, M., Béghin, L., Manios, Y., Sjoström, M., and Castillo, M. J. on behalf of the HELENA Study Group. (2008a). Reliability of health-related physical fitness tests in European adolescents. The HELENA Study. *International Journal of Obesity, 32*, S49-S57.

Ortega, F. B., Artero, E. G., Ruiz, J. R., España-Romero, V., Jiménez-Pavón, D., Vicente-Rodriguez, G., Moreno, L. A., Manios, Y., Béghin, L ., Ottevaere, C., Ciarapica, D., Sarri, K., Dietrich, S., Blair, S. N., Kersting, M., Molnar, D., González-Gross, M., Gutiérrez, Á., Sjoström, M. and Castillo, M. J. on behalf of the HELENA study group. (2011). Physical fitness levels among European adolescents: the HELENA study. *British Journal of Sports Medicine, 45*, 20-29.

Ortega, F. B., Ruiz, J. R., Castillo, M. J., Moreno, L. A., Gonzalez-Gross, M., Warnberg, J., Gutierrez, A. and the AVENA Group. (2005). Low level of physical fitness in Spanish adolescents. Relevance for future cardiovascular health. (AVENA study). *Spanish Journal of Cardiology, 58*, 898-909.

Ortega, F. B., Ruiz, J. R., Castillo, M. J. and Sjoström, M. (2008b). Physical fitness in childhood and adolescence: a powerful marker of health. *International Journal of Obesity, 32*, 1-11.

Pasternak, R. C., Abrams, J., Greenland, P., Smaha, L. A., Wilson, P. W. F. and Houston-Miller, N. (2003). Identification of coronary heart disease risk: is there a detection gap? *Journal of the American College of Cardiology, 41*(11), 1863-1874.

Pearlman, J. (2011). *Imaging in Coronary artery disease.* Retrieved June 12, 2011 from http://emedicine.medscape.com/article/349040-overview.

Pearson, T. A. (1999). Cardiovascular disease in developing countries: myths, realities, and opportunities. *Cardiovascular Drugs and Therapy, 13*, 95-104.

Pearson, T. A., Mensah, G. A., Alexander, R. W., Anderson, J. L., Cannon III, R. O., Criqui, Fadl, Y. Y., Fortmann, S. P., Hong, Y., Myers, G. L., Rifai, N., Smith, Jr, S. C., Taubert, K., Tracy, R. P. and Vinicor, F. (2003). Markers of inflammation and cardiovascular disease. Application to clinical and public health practice. A statement for healthcare professionals from the Centers for Disease Control and Prevention and the American Heart Association. *Circulation, 107*, 499-511.

Pearson, T. A., Smith, Jr., S. C. and Poole-Wilson, P. (1998). Cardiovascular specialty societies and the emerging global burden of cardiovascular disease: a call to action. *Circulation*, *97*, 602-604.

Pederson, D. and Gore, C. (1996). Anthropometry Measurement Error (Chapter 3). In K. Norton and T. Olds (eds.). *Anthropometrica*. University of New South Wales Press, Sydney.

Peltzer, K. (2001). Tobacco use among black South African university students: attitudes, risk awareness and health locus of control. *Curationis, 24*(2), 4-8.

Peltzer, K. (2010). Leisure Time Physical Activity and Sedentary Behaviour and Substance Use Among In-School Adolescents in Eight African Countries. *International Journal of Behavioural Medicine, 17,* 271-278.

Peltzer, K., Malaka, D.W. and Phaswana, N. (2002). Sociodemographic factors, religiosity, academic performance, and substance use among first-year university students in South Africa. *Psychological Reports, 91*(1), 105-113.

Pestana, J. A. X., Steyn, K., Leiman, A., Hartzenberg, G. M. (1996). The direct and indirect costs of cardiovascular disease in South Africa in 1991. *South African Medical Journal, 86*, 679-684.

Pillay, T. (2005). Determining the effects of a short-term physical activity intervention programme on body mass index, blood pressure, pulse rate and percentage body fat among high school learners. Unpublished Master's thesis. University of the Western Cape.

Pinto, B.M., Friedman, R., Marcus, B.H., Kelley, H., Tennstedt, S., and Gillman, M.W. (2002). Effects of a computer-based, telephone-counseling system of physical activity. American journal of preventive medicine, 23(2): 113 – 120.

Pischke, C. R., Scherwitz, L., Weidner, G. and Ornish, D. (2008). Long-term effects of lifestyle changes on well-being and cardiac variables among coronary heart disease patients. *Health Psychology, 27*, 584-592.

Porter, K., Johnson, P. H and Petrillo, J. (2009). Priority health behaviours among South African undergraduate students. *International Electronic Journal of Health Education, 12*, 222-243.

Prochaska, J. O., DiClemente, C. C. and Norcross, J. C. (1992). In search of how people change - applications to addictive behaviours. *American Psychologist, 47*(9), 1102-1114.

Pulkki, L., Kivimäki, M., Keltikangas-Järvinen, L., Elovainio, M., Leino, M. and Viikari, J. (2003). Contribution of adolescent and early adult personality to the inverse association between education and cardiovascular risk behaviours: prospective population-based cohort study. *International Journal of Epidemiology, 32*, 968-975.

Puoane, T. R., Steyn, K., Bradshaw, D., Laubscher, R., Fourie, J. M., Lambert, V., Mbananga, N. (2002). Obesity in South Africa: the South African demographic and health survey. *Obesity Research, 10*(10), 1038-1048.

Puska, P., Tuomilehto, J., Salonen, J., Neittaanmaki, L., Maki, J., Virtamo, J., Nissinen, A., Koskela, K. and Takalo, T. (1979). Changes in coronary risk factors during comprehensive five-year community programme to control cardiovascular diseases (North Karelia project). *British Medical Journal, 2*, 1173-1178.

Raitakari, O. T., Leino, M., Raikkonen, K., Porkka, K. V. K., Taimela, S. and Rasanen, L. (1995). Clustering of risk habits in young adults: the cardiovascular risk in young Finns study. *American Journal of Epidemiology, 142*(1), 36-44.

Ramsbottom, R., Brewer, J. and Williams, C. (1988). A progressive shuttle run test to estimate maximal oxygen uptake. *British Journal of Sports Medicine, 22*(4), 141-144.

Rankinen, T., Bray, M. S., Hagberg, J. M., Perusse, L., Roth, S. M., Wolfarth, B. and Bouchard, C. (2006). The human gene map for performance and health-related fitness phenotypes: The 2005 update. *Medicine and Science in Sports and Exercise, 38*(11), 1863-1888.

Reddy, S. P., Panday, S., Swart, D., Jinabhai, C. C., Amosun, S. L. and James, S. (2003). *Umthenthe Uhlaba Usamila – The South African Youth Risk Behaviour Survey 2002.* Cape Town: South African Medical Research Council. Retrieved August 16, 2009 from http://www.mrc.ac.za/healthpromotion/reports.htm.

Remsberg, K. E. and Siervogel, R. M. (2003). A life span approach to cardiovascular disease risk and aging: The Fels longitudinal study. *Mechanisms of Aging and Development, 124*, 249-257.

Rollini, F., Mfeukeu, L. and Modena, M.G. (2009). Assessing coronary heart disease in women. *Maturitas, 62*, 243-247.

Romero, D., McMahan, S. and Cathorall, M. (2005). Cardiovascular disease risk: should we target college women? *Californian Journal of Health Promotion, 3*(1), 117-129.

Ross, S. E., Niebling, B. C., and Heckert, T. M. (1999). Sources of stress among college students. *College Student Journal, 33*, 1999-2001.

Rosal, M. C., Ockene, J. K., Luckmann, R., Zapka, J., Goins, K. V., Saperia, G., Mason, T. and Donnelly, G. (2004). Coronary heart disease multiple risk factor reduction: providers' perspectives. *American Journal of Preventive Medicine, 27*(2), 54-60.

Rosenberg, D. E., Norman, G. J., Sallis, J. F., Calfas, K. J. and Patrick, K. (2007). Covariation of adolescent physical activity and dietary behaviours over 12 months. *Journal of adolescent health, 41*, 472-478.

Rothstein, W. G. (2003) *Public health and the risk factor: a history of an uneven medical revolution.* University of Rochester Press, Manchester.

Saelens, B. E., Gehrman, C. A., Sallis, J. F., Calfas, K. J., Sarkin, J. A. and Caparosa, S. (2000). Use of self-management strategies in a 2-year cognitive-behavioural intervention to promote physical activity. *Behaviour Therapy, 31*, 365-379.

Sarkin, J. A., Marshall, S. J., Larson, K. A., Calfas, K. J. and Sallis, J. F. (1998). A comparison of methods of recruitment to a health promotion program for university seniors. *Preventive Medicine, 27*, 562-571.

Sarwar, N., Aspelund, T., Eiriksdottir, G., Gobin, R., Seshasai, S. R. K., Forouhi, N. G., Sigurdsson, G., Danesh, J. and Gudnason, V. (2010) Markers of dysglycaemia and risk of coronary heart disease in people without diabetes: Reykjavik prospective study and systematic review. *Public Library of Science (PLoS) Medicine, 7*(5), e1000278.

Schmidt, G. J., Walkuski, J. J., and Stensel, D. J. (1998). The Singapore youth coronary risk and physical activity study. *Medicine and Science in Sports and Exercise, 30*(1), 105-113.

Sebregts, E. H. W. J., Falger, P. R. J. and Bär, F. W. H. M. (2000). A Review. Risk factor modification through nonpharmacological interventions in patients with coronary heart disease. *Journal of Psychosomatic Research, 48*, 425-441.

Seedat, Y. K., Croasdale. M. A., Milne, F. J., Opie, L. H., Pinkney-Atkinson, V. J., Rayner, B. L., Veriava, Y. and Joint National Hypertension Guideline Working Group. (2006). South African Hypertension Guidelines. *South African Medical Journal, 96*, 337-362.

Selig, S., Levinger, I. and Broadbent, S. (2011). Exercise as therapy in cardiovascular disease. In M. Cameron and S. Selig. *Clinical Exercise: A case-based approach*. Hemphill, Churchill Livingstone, Elsevier. Sydney.

Senekal, M., Steyn, N. P. and Nel, J. H. (2003). Factors associated with overweight/obesity in economically active South African populations. *Ethnicity and Disease, 13*, 109-116.

Sesso, H. D., Paffenbarger Jr, R. S. and Lee, I. (2000). Physical activity and coronary heart disease in men: the Harvard alumni health study. *Circulation, 102*, 975-980.

Shenk, K. and Williamson, J. (2005). *Ethical approaches to gathering information from children and adolescents in international settings: guidelines and resources*. The Population Council Inc, Washington, DC, USA.

Shiroma, E. J. and Lee, I. (2010). Physical activity and cardiovascular health: lessons learned from epidemiological studies across age, gender, and race/ethnicity. *Circulation*, *122*,743-752.

Smith, D. C., Robinson, C. S. and Myers, E. F. (1997). Incorporating a quality life and well-being instrument as an additional outcome measure of a comprehensive weight management program. *Journal of American Dietetic Association, 97*(9), 108.

Smitherman, T. A., Kendzor, D. E., Grothe, K. B. and Dubbert, P. M. (2007). State of the art review: promoting physical activity in primary care settings. A review of cognitive and behavioural strategies. *American Journal of Lifestyle Medicine, 1*(5), 397-409.

South African Medical Research Council. (1991a). MRC Food Quantities Manual. Second Edition. South African Medical Research Council, Parow Valley.

South African Medical Research Council. (1991b). MRC Food Composition Tables. Third Edition. South African Medical Research Council, Parow Valley.

South African Medical Research Council. (2001). *Guidelines on ethics for medical research: general principles*. South African Medical Research Council, Parow Valley.

South African Medical Research Council. (2002). *Guidelines on ethics for medical research: use of biohazards and radiation*. South African Medical Research Council, Parow Valley.

Sproule, J., Kunalan, C., McNeill, M. and Wright, H. (1993). Validity of 20-MST for predicting VO2max of adult Singaporean athletes. *British Journal of Sports Medicine, 27*, 202-204.

Stark, R. M. (1996). Review of the major intervention trials of lowering coronay artery disease risk through cholesterol reduction. *American Journal of Cardiology, 78*(supplement 6A), 13-19.

Steptoe, A., Doherty, S., Rink, E., Kerry, S., Kendrick, T. and Hilton, S. (1999). Behavioural counseling in general practice for the promotion of healthy behaviour among adults at increased risk of coronary heart disease: randomized trial. *British Medical Journal, 319*, 943-8.

Steptoe, A. and Wardle, J. (2001). Health behaviour, risk awareness and emotional well-being in students from eastern Europe and western Europe. *Social Science and Medicine, 53*, 1621-1630.

Steptoe, A., Wardle, J., Cui, W., Bellisle, F., Zotti, A., Baranyi, R. and Sanderman, R. (2002). Trends in smoking, diet, physical exercise, and attitudes toward health in European university students from 13 countries, 1999-2002. *Preventive Medicine, 35*, 97-104.

Steptoe, A., Wardle, J., Fuller, R., Holte, A., Justo, J., Sanderman, R. and WichstrØm, L. (1997). Leisure-time physical exercise: prevalence, attitudinal correlates, and behavioural correlates among young Europeans from 21 countries. *Preventative Medicine, 26*, 845-854.

Steyl, T. (2008). Knowledge and perception of cardiovascular disease risks of female university students. *Journal of Community and Health Sciences, 3*(1), 21-29.

Steyn, N. (2006). Nutrition and chronic diseases of lifestyle in South Africa. In: Steyn, K., Fourie, J., & Temple, N. (eds). *Chronic Diseases of Lifestyle in South Africa: 1995-2005, Technical Report*. Cape Town: South African Medical Research Council.

Steyn, N. P., Senekal, M., Brits, S., Alberts, M., Mashego, T. and Nel, J. H. (2000). Weight and health status of black female students. *South African Medical Journal, 90*(2), 146-52.

Steyn, K., Everett, K. and Odendaal, H. (2004). Smoking during pregnancy. *Medical Research Council Policy brief; No. 3.*

Steyn, K. and Fourie, J. M. (2007). *Heart disease in South Africa: media data document.* Cape Town: South African Medical Research Council. Retrieved April 11, 2009 from http://www.heartfoundation.co.za/docs/heartmonth/HeartDiseaseinSA.pdf

Steyn, K., Gaziano, T. A., Bradshaw, D., Laubscher, R. and Fourie, J. M. (2001). Hypertension in South African adults: results from the demographic and health survey, 1998. *Journal of Hypertension, 19*(9), 1717-1725.

Steyn, K., Jooste, P. L., Bourne, L., Fourie, J., Badenhorst, C. J., Bourne, D. E., Langenhoven, M. L., Lombard, C. J., Truter, H., Katzenellenbogen, J., Marais, M. and Oelofse, A. (1991). Risk factors for coronary heart disease in the black population of the Cape Peninsula. The BRISK study. *South African Medical Journal, 79*(8), 480-485.

Steyn, K., Levitt, N. S., Hoffman, M., Marais, A. D., Fourie, J. M., Lambert, E. V., Gaziano, T. A., Kepe, L., Lombard, C. J. (2004). The global cardiovascular diseases risk pattern in a peri-urban working-class community in South Africa. The Mamre study. *Ethnicity and Disease, 14*(2), 233-242**.**

Steyn, K., Rossouw, J. E. and Joubert, G. (1990). The coexistence of major coronary heart disease risk factors in the coloured population of the Cape Peninsula (CRISIC study). *South African Medical Journal, 78*(2), 61-3.

Steyn, K., Rossouw, J. E., Weight, M. J., Fourie, J. M., Benade, A. J., Jooste, P. L., Chalton, D. O. and Wolmanrans, P. (1996). Apolipoprotein B levels and related factors in a rural white South African community – the CORIS study. *South African Medical Journal, 86*(4), 359-364.

Steyn, K., Steyn, M., Swanepoel, A., Jordaan, P., Jooste, P., Fourie, J., and Rossouw, J. (1997). Twelve year results of the coronary risk factor study. *International Journal of Epidemiology, 26*(5), 964-971

Stickland, M. K., Petersen, S. R. and Bouffard, M. (2003). Prediction of maximal aerobic power from the 20-m multi-stage shuttle run test. *Canadian Journal of Applied Physiology, 28*(2), 272-82.

Sung, J., Song, Y., Ebrahim, S. and Lawlor, D. A. (2009). Fasting blood glucose and the risk of stroke and myocardial infarction. *Circulation, 119*, 812-819.

Tamin, H., Dumit, N., Terro, A., Al-Hourany, R., Sinno, D., Seif, F., Steiteh, S. and Musharrafieh, U. (2004). Weight control measures among university students in a developing country: a cultural association or a risk behaviour. *Journal of the American College of Nutrition, 23*(5), 391-396.

Tamin, H., Terro, A., Kassem, H., Ghazi, A., Khamis, T. A., Hay, M. M. A. and Mushaarrafieh, U. (2003). Tobacco use by university students Lebanon, 2001. *Addiction, 98*, 933-939.

Tammelin, T., Nayha, S., Hills, A. P. and Jarvelin, M. (2003). Adolescent participation in sports and adult physical activity. *American Journal of Preventive Medicine, 24*(1), 22-28.

Tamragouri, R. N., Martin, R. W., Cleavenger, R. L. and Sieber, Jr., W. K. (1986). Cardiovascular risk factors and health knowledge among freshman college students with a family history of cardiovascular disease. *Journal of American College Health, 34*, 267-270.

Tanasescu, M., Leitzmann, M. F., Rimm, E. B., Willett, W. C., Stampfer, M. J. and Hu, F. B. (2002). Exercise Type and Intensity in Relation to Coronary Heart Disease in Men. *Journal of the American Medical Association, 288*, 1994-2000.

The Emerging Risk Factor Collaboration. (2011). Separate and combined associations of body-mass index and abdominal adiposity with cardiovascular disease: collaborative analysis of 58 prospective studies. *Lancet, 377*, 1085-95.

Thomas, N. E., Baker, J. S. and Davies, B. (2003). Established and recently identified coronary heart disease risk factors in young people: the influence of physical activity and physical fitness. *Sports Medicine, 33*(9), 633-650.

Tikkanen, H. O., Hamalainen, E., Sarna, S., Adlercreutz, H. and Harkonen, M. (1998). Associations between skeletal muscle properties, physical fitness, physical activity and coronary heart disease risk factors in men. *Atherosclerosis, 137*, 377-389.

Trost, S. G., Pate, R. R., Saunders, R., Ward, D. S., Dowda, M. and Felton, G. (1997). A prospective study of the determinants of physical activity in rural fifth-grade children. *Preventive Medicine, 26*, 257-263.

Tumusiime, D. K. (2004). Perceived benefits of, barriers and helpful cues to physical activity among tertiary institution students in Rwanda. Unpublished Master's thesis. University of the Western Cape.

U.S. Preventive Services Task Force. (2009). Using nontraditional risk factors in coronary heart disease risk assessment: U.S. Preventive Services Task Force recommendation statement. *Annals of Internal Medicine, 151*, 474-482.

Van Der Steeg, W. A., Boekholdt, S. M., Stein, E. A., El-Harchaoui, K., Stroes, E. S., Sandhu, M. S., Wareham, N. J., Jukema, J. W., Luben, R., Zwinderman, A. H., Kastelein, J. J. and Khaw, K. T. (2007). Role of the apolipoprotein B-apolipoprotein A-I ratio in cardiovascular risk assessment: a case-control analysis in EPIC-Norfolk. *Annals of Internal Medicine, 146*(9), 640-648.

Vega, C. (2007). Psychosocial factors predict hypertension and coronary heart disease. Retrieved on March 12, 2010 from http://www.mediscape.com/veiwarticle/563642?src=mp

Venuraju, S. M., Yerramasu, A., Corder, R. and Lahiri, A. (2010). Osteoprotegerin as a predictor of coronary artery disease and cardiovascular mortality and morbidity. *Journal of the American College of Cardiology, 55*(19), 2049-61.

Von Ah, D. (2004). Predictors of health behaviours in college students. *Journal of Advanced Nursing, 48*(5), 463-474.

Vorster, H. H. (2002). The emergence of cardiovascular disease during urbanisation of Africans. *Public Health Nutrition, 5*(1A), 239-243.

Waldron, C. A., Van der Weijden, T., Ludt , S., Gallacher, J. and Elwyn, G. (2011). What are effective strategies to communicate cardiovascular risk information to patients? A systematic review. *Patient Education and Counselling, 82*, 169-181.

Wang, C., Haskell, W. L., Farrell, S. W., LaMonte, M. J., Blair, S. N., Curtin, L. R., Hughes, J. P. and Burt, V. L. (2010). Cardiorespiratory fitness levels among US adults 20–49 years of age: findings from the 1999–2004 national health and nutrition examination survey. *American Journal of Epidemiology, 171*(4), 426-435.

Wang. T. J., Gona, P., Larson, M. G., Tofler, G. H., Levy, D., Newton-Cheh, C., Jacques, P. F., Rifai, N., Selhub, J., Robins, S. J., Benjamin, E. J., D'Agostino, R. B. and Vasan, R. S. (2006). Multiple biomarkers for the prediction of first major cardiovascular events and death. *New England Journal of Medicine, 355*(25), 2631-2639.

Warburton, D. E. R., Nicol, C. W. and Bredin, S. S. D. (2006). Health benefits of physical activity: the evidence. *Canadian Medical Association Journal, 174*(6), 801-809.

Ward, K. D., Vander Weg, M. W., Klesges, R. C., Kovach, K. W., Elrod, M. C., DeBon, M., Haddock, C. K., Talcott, G. W. and Lando, H. A. (2003). Characteristics of highly physically active smokers in a population of young adult military recruits. *Addictive Behaviours, 28*, 1405-1418.

Werch, C. E., Bian, H., Moore, M. J., Ames S., DiClemente, C. C. and Weiler, R. M. (2007). Brief multiple behaviour interventions in a college student health care clinic. *Journal of Adolescent Health*, *39*, 31-39.

Westerterp, K.R. (1999). Assessment of physical activity level in relation to obesity: current evidence and research issues. *Medicine and Science in Sports and Exercise*, *l3*(11), S522 - S525.

Wilson, P., D'Agostino, R., Levy, D., Belanger, A., Silbershatz, H. and Kannel, W. (1998). Prediction of Coronary Heart Disease Using Risk Factor Categories. *Circulation, 97,* 1837-1847.

World Health Organisation. *World health report 2002*. Geneva: World Health Organisation, 2002.

Writing Group for the Activity Counseling Trial (ACT) Research Group. (2001). Effects of physical activity counseling in primary care. *Journal of the American medical association*, *286*, 677-687.

Yusuf, S., Reddy, S., Ôunpuu, S. and Anand, S. (2001a). Global burden of cardiovascular diseases: Part I. General considerations, the epidemiologic transition, risk factors, and impact of urbanization. *Circulation*, *104*, 2746-2753.

Yusuf, S., Reddy, S., Ôunpuu, S. and Anand, S. (2001b). Global burden of cardiovascular diseases: Part II: variations in cardiovascular disease by specific ethnic groups and geographic regions and prevention strategies. *Circulation*, *104*, 2855-2864.

Appendix A: Information Sheet

UNIVERSITY OF THE WESTERN CAPE

Private Bag X 17, Bellville 7535, South Africa
Tel: +27 21-959 2350, Fax: 27 21-959 3688
E-mail: vkensley@uwc.ac.za

INFORMATION SHEET

Title of Research Project: The impact of multiple behaviour health intervention strategies on coronary artery disease risk, health-related physical fitness, and health-risk behaviours of university students.

What is the study about?

This is a research project being conducted by Lloyd Leach at the University of the Western Cape. You are invited to participate in this research project because you are a first year student studying at UWC and who may have one or more risk factors for heart disease, deficits in health-related physical fitness, and/or lifestyle behaviours that may place you at health risk. The purpose of this research project is to determine the impact of a variety of health interventions, such as health awareness, diet and exercise management, on the following health indicators: coronary artery (heart) disease risk, health-related physical fitness, and health-risk behaviours. The intervention period of the study will run over a period of sixteen (16) weeks, which excludes preliminary (baseline) and follow-up (post-intervention) measurements.

What will I be asked to do if I agree to participate?

You will undergo initial screening at the Biokinetics clinic in the Department of Sport, Recreation and Exercise Science (UWC) in order to establish their health risk status. These tests will last about ninety (90) minutes and be conducted on three (3) separate days, as follows:

Day 1: Filling in two (2) questionnaires, namely, a physical activity readiness questionnaire (PAR-Q) and a healthy lifestyle questionnaire (HLQ). Both these questionnaires will take about 30 minutes in total;

Day 2: Non-invasive measurements will be taken of selected coronary artery disease risk factors (about 20 minutes), as well as undergoing a physical fitness test to measure aspects of fitness related to health, such as cardiovascular fitness, muscular strength and endurance, and flexibility (about 30 minutes); and

Day 3: In the fasting state, a micro-capillary blood sample will be taken by the campus medical doctor at the Student Health Centre for determining blood glucose and cholesterol concentration. This will take about 5 minutes.

All these tests will be repeated again at the end of the study, after the intervention period of sixteen (16) weeks.

What are the exercise tests of this research?

Participants' will perform a battery of physical fitness tests in the UWC Biokinetics Clinic, at the Department of Sport, Recreation, and Exercise Science. The tests will be performed using various pieces of exercise equipment for evaluating cardiovascular endurance, muscular strength, muscular endurance, and flexibility.

The exercise test for measuring cardiovascular endurance is a progressive incremental test, which usually begins at a slow speed but will advance in stages until you are no longer able to continue at the required speed of the test. You may also stop the test at any time, because of signs of distress.

Will my identity and information be kept confidential?

All participants are tested anonymously in a private setting, and all information obtained is kept confidential, except for the purposes of research. You will be assigned a randomly generated identification code (research number) in order to maintain your anonymity, and all your information will be secured either in a locked filing cabinet or password-protected computer files accessible by the researcher only. In the instance of a report or article being written about this research project, your identity will be protected to the maximum extent possible.

What are the risks and discomforts of this research?

The possibility exists that certain abnormal changes can occur during the tests. These include abnormal blood pressure, fainting, disordered heart beat, and in rare instances, heart attack, stroke, or death. Every effort will be made to minimize these risks by evaluating preliminary information related to your health and fitness and by observations during testing. Trained personnel will be available who can deal with emergency situations that may arise.

In addition, your muscles may feel quite sore in the days following these tests, because of the strenuousness of the tests. This is normal for most individuals, and the soreness will soon disappear with time as the muscles heal.

What are the expected benefits of this research?

The results obtained from the various tests will be used exclusively for the purposes of research. Additionally, it may assist us in identifying your health risk. If you are found to be at high risk you will be advised to consult your family doctor in order to have a proper medical examination as soon as possible. Based upon your medical condition, your doctor will then determine whether or not you should participate further in this research project.

Following the period of health intervention, should any of the multiple interventions prove beneficial to health and wellbeing, all participants not subjected to such interventions during the

project will be accommodated at the conclusion of the study, and given access to the health intervention(s) subsequently.

What will you be expected to do?

Information you possess about your health status or previous experiences of unusual feelings with physical effort may affect the safety and value of your fitness test. Your prompt reporting of how you feel during the exercise test is also important. You are responsible for fully disclosing such information when requested to do so by the testing staff.

How long will the study last?

The initial measurements will be done over a period of three (3) weeks. Thereafter, the intervention period will follow for another sixteen (16) weeks. Finally, the measurements will be repeated over a period of about two (2) weeks when the research project ends. In total, therefore, the study will last 21 weeks or five-and-a half months.

Do I have to be in this research and may I stop participating at any time?

Your participation in this research is completely voluntary. You may choose not to participate at all. If you decide to participate in this research, you may stop any test at any point or even withdraw from the study as a whole without being penalized or losing any benefits to which you otherwise qualify. However, should your medical doctor confirm that your medical condition places you at high risk for heart disease, then your participation may be terminated. Subsequent assistance may be provided to you depending upon the recommendations of your doctor.

What if I have any questions

This research is being conducted by Lloyd Leach at the Department of Sport, Recreation and Exercise Science at the University of the Western Cape. If you have any questions about the research project itself, please contact:

Lloyd Leach
Tel: 021-959 3653
Cell: 082 200 6987
Email: lleach@uwc.ac.za
Address: see departmental address above

We encourage you to ask any questions about either the tests or the procedures used in the research project. If you have doubts or questions regarding this study and your rights as a research participant or if you wish to report any problems you have experienced related to the study, please contact:

Head of Department: Dr Sue Bassett
Dean of the Faculty of Community and Health Sciences: Prof Rati Mpofu
University of the Western Cape
Private Bag X17

Bellville
7535

N.B. This research study has been approved by the University of the Western Cape's Senate Research Committee and Ethics Committee.

Questions asked by the participant:

1. _____

Response: _____

2. _____

Response: _____

3. _____

Response: _____

THANK YOU

Appendix B: Consent Form

UNIVERSITY OF THE WESTERN CAPE

Private Bag X 17, Bellville 7535, South Africa

Tel: +27 21-959 2350, Fax: 27 21-959 3688

E-mail: vkensley@uwc.ac.za

CONSENT FORM

Title of Research Project: The impact of multiple behaviour health intervention strategies on coronary artery disease risk, health-related physical fitness, and health-risk behaviours of university students.

Because of the nature of the study, you will be expected to give a blood sample (drawn by a medical doctor: Dr Jo-Anne Kirby) at the start and at the end of the project that will be used to determine coronary heart disease risk only.

The study has been described to me in language that I understand and I freely and voluntarily agree to participate. My questions about the study have been answered. I understand that my identity will not be disclosed and that I may withdraw from the study without giving a reason at any time and this will not negatively affect me in any way.

Participant's name: …………………………………………………………………………..

Participant's signature: ...………………………………………………………………………..

Witnesses Name: …………………………………………………………………………………...

Date: …………………………………………………………………………………………………..

Should you have any questions regarding this study or wish to report any problems you have experienced related to the study, please contact the study coordinator:

Study Coordinator's Name: Lloyd Leach

University of the Western Cape, Private Bag X17, Bellville, 7535.

+27-21-959-2653   *+27-21-959 3688*   *082-200-6987*   **lleach@uwc.ac.za**

Appendix C: Physical Activity Readiness Questionnaire (PAR-Q)

(Mark with an X in the appropriate space)

		Yes	No

1. Has your doctor ever said that you have a heart condition <u>and</u> that you should only do physical activity recommended by a doctor   ____   ____

2. Do you feel pain in your chest when you do physical activity   ____   ____

3. In the past month, have you had chest pain when you were not doing physical activity   ____   ____

4. Do you lose your balance because of dizziness or do you ever lose consciousness   ____   ____

5. Do you have a bone or joint problem that could be made worse by a change in your physical activity   ____   ____

6. Is your doctor currently prescribing drugs (for example, water pills) for your blood pressure or heart condition   ____   ____

7. Do you know of any other reason why you should not do physical activity   ____   ____

I, _____ do hereby declare that, to the best of

my knowledge, I am currently free of any physical injuries, medical condition, mental

condition or any other complaint that would preclude me from undertaking any of the

physical, physiological and / or anthropometric tests.

Signature of Participant: _____ Date: _____

Signature of Witness: _____ Date: _____

THANK YOU

Appendix D: Pre-Test Instructions

In order to increase the validity and accuracy of the physical fitness test data, the pre-test instructions should be adhered to strictly.

<u>Before the test:</u>

1. Participants should refrain from ingesting food, alcohol, or caffeine or using tobacco products within 3 hours of testing.

2. Participants should be rested for the assessment, avoiding significant exercise or exercise on the day of the assessment.

3. Clothing worn for the assessment should be light, loose fitting to permit freedom of movement, and include shorts and running shoes. Females should bring a loose-fitting, short-sleeved blouse (top), and shorts, tights or swim suit (two piece), and should avoid restrictive undergarments.

4. If you are currently on medication (for example, asthma pump), please make sure that you have it available when you report for testing, as the tests may affect you adversely. Also report to the researcher the last actual dose taken.

5. Participant's with any injuries or illness on the day of testing must report them to the researcher immediately and, if possible, schedule another appointment so as not to be unduly penalized before or during testing.

6. Drink plenty of fluids over the 24-hour period preceding the test to ensure normal hydration prior to testing.

7. Get an adequate amount of sleep (6 to 8 hours) the night before the test.

8. Participants must be aware that the physical fitness evaluations may be fatiguing, so you may wish to have someone accompany you to the assessment in order to drive or accompany you home afterward.

N.B: Please observe the above instructions strictly when preparing for testing, since failing to do so can have a negative impact on the outcome of your evaluations

<u>After the Test:</u>

At the end of the test, participants may use the shower facilities to wash and change before leaving. Because the testing is relatively time-consuming (2 – 3 hours), participants may wish to bring some refreshments to be consumed only after all the tests have been completed.

THANK YOU

Appendix E: Coronary Heart Disease (CHD) Risk Assessment Form
(Mark with an X in the appropriate space, if applicable)

Study ID No.: _____   **Date**: __/__/20__   **Time**: __H__

- **Age**: ____ years   Date of Birth: _____ **Subject ID No**: _____

  [Office use only ………………………. Risk: No ☐   Yes ☐]

- **Family history of heart disease:** Don't Know ☐   No ☐   Yes ☐
  (Myocardial infarction, coronary revascularization, or sudden death before 55 years in father or other male first-degree relative (that is, brother or son) or before 65 years in mother or other female first-degree relative (that is, sister or daughter)

  [Office use only ………………………. Risk: No ☐   Yes ☐]

- **Cigarette smoking:** No ☐   Yes ☐
  (current cigarette smoker or quit within the previous 6 months)
  If yes, indicate number of cigarettes smoked per day: _____

  [Office use only …………………………. Risk: No ☐   Yes ☐]

- **Hypertension:**
  1. HR: _____ bpm   **BP$_{SYS}$** _____ mm Hg   **BP$_{DIAS}$** ____ mm Hg
  2. HR: _____ bpm   **BP$_{SYS}$** _____ mm Hg   **BP$_{DIAS}$** ____ mm Hg
  3. HR: _____ bpm   **BP$_{SYS}$** _____ mm Hg   **BP$_{DIAS}$** ____ mm Hg

  [Office use only …………………………. Risk: No ☐   Yes ☐]

- **Dyslipidemia:**   **Total serum cholesterol:**   _____ $mmol^.L^{-1}$
  LDL cholesterol: _____ $mmol^.L^{-1}$

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HDL cholesterol: _____ mmol·L^{-1}

Triglycerides: _____ mmol·L^{-1}

[Office use only …………………. **Risk**: No ☐   Yes ☐]

- **Impaired Fasting Glucose**:   **Blood glucose:** _____ mmol·L^{-1}

  [Office use only …………………. **Risk**: No ☐   Yes ☐]

- **Obesity:**

1. **Ht:** ____ cm   **Wt:** ____ kg   **BMI:** ____ kg·m^{-2}
2. **Ht:** ____ cm   **Wt:** ____ kg   **BMI:** ____ kg·m^{-2}
3. **Ht:** ____ cm   **Wt:** ____ kg   **BMI:** ____ kg·m^{-2}

1. **Waist girth:** ____ cm   **Hip girth:** ____ cm   **WHR:** ____
2. **Waist girth:** ____ cm   **Hip girth:** ____ cm   **WHR:** ____
3. **Waist girth:** ____ cm   **Hip girth:** ____ cm   **WHR:** ____

[Office use only …………………. **Risk**: No ☐   Yes ☐]

- **Sedentary Lifestyle: Physical activity**

  Current Status: Active ☐   Inactive ☐

  If active, ………….. **Mode:** Aerobic ☐   Anaerobic ☐   Mixed ☐

  Frequency: 1 ☐   **2** ☐   **3** ☐   **4** ☐   **>4** ☐ times/week

  Intensity: Low ☐   Moderate ☐   Vigorous ☐

  Duration: <10 ☐   **10-30** ☐   **>30** ☐ min/session

  Office use only …………………. Risk: No _____   Yes _____]

THANK YOU

Appendix F: Physical Fitness Data Recording Sheet

Personal Information

(Mark with an X in the appropriate space, if applicable)

Subject ID No.: _____ Date: _____ Time: _____

Student Number: _____ Degree Name: _____

Address: _____

Tel: (   ) _____ (h)   (   ) _____ (cell)

Gender: male (♂) ☐   female (♀) ☐

Racial Group: Black ☐   Mixed Ethnicity ☐   Indian ☐   White ☐

Marital status: Single ☐   Married ☐   Divorced ☐   Widowed ☐

Current injuries or illnesses: _____

Anthropometry

Skinfolds:

Females (♀):

1. a) Triceps: ____ mm   b) Supra-iliac : ____ mm   c) Thigh: ____ mm
2. a) Triceps: ____ mm   b) Supra-iliac : ____ mm   c) Thigh: ____ mm
3. a) Triceps: ____ mm   b) Supra-iliac : ____ mm   c) Thigh: ____ mm

RATING: _____

Males (♂):

1. a) Pectoral: ____ mm   b) Abdominal: ____ mm   c) Thigh: ____ mm
2. a) Pectoral: ____ mm   b) Abdominal: ____ mm   c) Thigh: ____ mm
3. a) Pectoral: ____ mm   b) Abdominal: ____ mm   c) Thigh: ____ mm

RATING: _____

Cardiovascular Endurance
1. Multistage Shuttle Run (Bleep) Test: Level: _____   Shuttle: _____
2. Multistage Shuttle Run (Bleep) Test: Level: _____   Shuttle: _____

RATING: _____

Muscular Strength
Static Muscular Strength:
1. a) Right Handgrip Strength: _____ kg   b) Left Handgrip Strength: _____ kg
2. a) Right Handgrip Strength: _____ kg   b) Left Handgrip Strength: _____ kg
3. a) Right Handgrip Strength: _____ kg   b) Left Handgrip Strength: _____ kg

RATING: _____
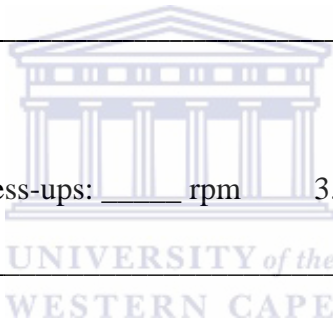
Muscular Endurance
1. Press-ups: _____ rpm   2. Press-ups: _____ rpm   3. Press-ups: _____ rpm

RATING: _____

Flexibility
1. Sit-and-Reach: _____ cm   2. Sit-and-Reach: _____ cm   3. Sit-and-Reach: _____ cm

RATING**: _____**

THANK YOU

Appendix G: Multi-Stage Shuttle Run (Bleep Test) Recording Sheet

topend sports.com Beep Test Recording Sheet

Date: _____ Time: _____ Conditions: _____

Level 1   1 2 3 4 5 6 7

Level 2   1 2 3 4 5 6 7 8

Level 3   1 2 3 4 5 6 7 8

Level 4   1 2 3 4 5 6 7 8 9

Level 5   1 2 3 4 5 6 7 8 9

Level 6   1 2 3 4 5 6 7 8 9 10

Level 7   1 2 3 4 5 6 7 8 9 10

Level 8   1 2 3 4 5 6 7 8 9 10 11

Level 9   1 2 3 4 5 6 7 8 9 10 11

Level 10   1 2 3 4 5 6 7 8 9 10 11

Level 11   1 2 3 4 5 6 7 8 9 10 11 12

Level 12   1 2 3 4 5 6 7 8 9 10 11 12

Level 13   1 2 3 4 5 6 7 8 9 10 11 12 13

Level 14   1 2 3 4 5 6 7 8 9 10 11 12 13

Level 15   1 2 3 4 5 6 7 8 9 10 11 12 13

Level 16   1 2 3 4 5 6 7 8 9 10 11 12 13 14

Level 17   1 2 3 4 5 6 7 8 9 10 11 12 13 14

Level 18   1 2 3 4 5 6 7 8 9 10 11 12 13 14 15

Level 19   1 2 3 4 5 6 7 8 9 10 11 12 13 14 15

Level 20   1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16

Level 21   1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16
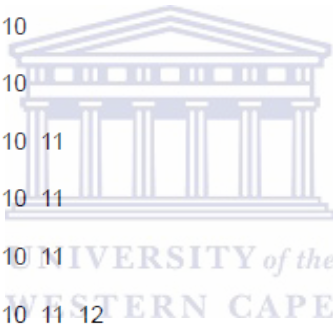
* circle the level reached for each participant, and write their name next to that line.

Appendix H: The Healthy Lifestyle Questionnaire

The purpose of this questionnaire is to help you analyze your lifestyle behaviours and to help you make decisions concerning good health and wellness for the future. Answer each question as honestly as possible. The questions refer to your lifestyle in general.

Directions: Place an "X" over the "box" to answer yes. **If you answer "no", leave the box blank**.

Initials only: ☐☐☐☐ First 4 numbers of your ID number: ☐☐☐☐ Any four digit number: ☐☐☐☐

(This will ensure your identity is kept confidential, and you can check your results according to this information**.**)

Feedback on the results of the questionnaire

☐ 1. In general, I'm physically active enough to keep healthy.

☐ 2. I am physically active for 30 minutes on at least 4 days of the week (e.g., by brisk walking, jogging, playing social sport, going to gym, etc).

☐ 3. I do hard (vigorous) physical activity for 20 minutes on at least 3 days of the week (e.g., running, playing competitive sport, training hard, aerobics, skipping, etc).

☐ 4. I do stretching exercises at least 3 days a week.

☐ 5. I do exercises for muscle fitness at least 2 days a week (e.g., sit-ups, push-ups, weight lifting, skipping, etc).

☐ 6. In general, I am physically fit.

☐ 7. In general, I eat at least three meals each day.

☐ 8. My daily diet consists of food from the four food groups: 1) grains and cereals; 2) fruits and vegetables; 3) meat and meat products; and 4) fats and oils.

☐ 9. I try to cut down on the amount of fat in my diet.

☐ 10. I eat only as much food as I need each day, so my weight is kept relatively constant.

☐ 11. I think my body is overweight.

☐ 12. I am able to identify situations in daily life that cause stress.

☐ 13. I take time out during the day to relax and recover from stress.

☐ 14. I find time for things I enjoy doing.

☐ 15. I know how to relieve tension.

☐ 16. I do not smoke or use any tobacco products.

☐ 17. I do not use alcohol or limit alcohol use to the legal limit.

☐ 18. I do not abuse drugs (prescription or illegal).

☐ 19. I use over-the-counter drugs only when needed, and use them according to prescription only.

☐ 20. I abstain from sex or limit sexual activity to a safe partner.

☐ 21. I practise safe procedures for avoiding sexually transmitted diseases (STDs).

☐ 22. I fasten my seat belt when in a vehicle.

☐ 23. I'm afraid when drivers exceed the speed limit.

☐ 24. I have been trained in first aid.

☐ 25. I can perform first aid, if called on in an emergency.

☐ 26. In an emergency, I know who to call for medical help.

☐ 27. I brush my teeth at least two times a day.

☐ 28. I get enough sleep each day.

☐ 29. I seek medical advice when signs and symptoms of sickness, disease and/or infection are present.

☐ 30. When I receive advice and/or medication from a doctor, I follow the advice and take the medication as prescribed.

☐ 31. Whenever I buy or use a new product, I read the product's label for information (e.g., food labels, weight loss products, etc)

☐ 32. I only use products that research shows to be effective (e.g., exercise equipment, weight loss products, etc).

☐ 33. I recycle waste, such as paper, glass, plastic, etc.

☐ 34. I conserve energy (e.g., using electricity and water sparingly/carefully).

☐ 35. I'm concerned about "global warming".

☐ 36. I have a sense of purpose in my life.

☐ 37. I'm positive about the future of this country.

THANK YOU.

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Appendix I: Daily Dietary Recording Sheet

Part of this study involves recording the food you have consumed, so that we can assess your average daily intake.

Please complete the GENERAL QUESTIONNAIRE and keep a 3-DAY FOOD RECORD. (This must include 2 weekdays: Monday to Friday; and 1 day over the weekend: Saturday or Sunday.)

INSTRUCTIONS

1. **GENERAL QUESTIONNAIRE**

   Complete all the questions. Comments may be written on the form.

2. **3-DAY FOOD RECORD**

   Please keep a record of all that you eat and drink for 3 days (2 weekdays and 1 weekend day). This should be done on the sheets provided in the following way:

   - Use a new page for each day.
   - Write down the DAY, DATE and your NAME on each page.
   - State what type of meal it is in the final column. Please use the following codes to define what type of meal it is:
     MM = main meal
     LM = light meal
     S = snack
     UM = unstructured meal

   - In the second column, record the TIME at which the food item was consumed.
   - In the third column record the FOOD ITEM.
   - A very detailed DESCRIPTION of the food item consumed must be recorded in the fourth column.
   - In the fifth column, the amount of food consumed must be recorded.

PLEASE PAY VERY SPECIAL ATTENTION TO THE FOLLOWING:

- the type of milk consumed
  e.g. full cream, low fat (2%), skim, powdered (including the brand name) or fresh.

- the type of cheese consumed
  e.g. Cheddar, feta, Creamed cottage or low fat cottage cheese.
  Low fat Edam.

- the type of margarine used
  e.g. hard, soft or medium spread and whether it was spread thickly, medium or thinly.

- Whether the meat eaten was:
LEAN	-	no visible fat
MEDIUM FAT	-	prepared with visible fat removed
FATTY	-	prepared and eaten with visible fat.

- whether fish eaten was canned in water or oil.

- additions to foods: cream, sugar, etc.
  e.g. 70g spinach with cream added
  1 cup tea with 2 heaped teaspoons of brown sugar.

- record commercial names where possible.
  e.g. TRIM mayonnaise, CREMORA coffee creamer.

GENERAL DIETARY QUESTIONNAIRE

1. Which of the following do you use in your coffee and tea?

	Coffee	Amount Per Cup	Tea	Amount Per Cup
Full cream milk				
Low fat (2%) milk				
Skimmed Milk				
Milk Blend				
Coffee Creamer				
Evaporated Milk				
Condensed Milk				
White Sugar				
Brown Sugar				
Other				

2. Which of the following do you use with coked porridge and cereals?

	Cooked Porridge	Amount Per Bowl	Cereal	Amount Per Bowl
Full cream milk				
Low fat (2%) milk				
Skimmed milk				
Milk Blend				
Coffee Creamer				
Evaporated Milk				
White Sugar				
Brown Sugar				
Other				

3. Do you ever drink a glass of milk?   YES / NO

Full Cream	
2% Milk	
Skimmed Milk	
Blended Milk	

4. Do you use HARD CHEESE?   YES / NO

   If yes, what type do you usually use? _____

5. Do you use COTTAGE CHEESE?   YES / NO

   If yes, what type do you usually use? _____

Creamed Cottage Cheese	
Low Fat Cottage Cheese	
Fat Free Cottage Cheese	

6. Do you eat YOGHURT?   YES / NO

   If yes, what type do you usually eat? _____

Fruit Flavoured, Low Fat	
Fruit Flavoured, Full Fat	
Plain, Low Fat	
Plain, Full Fat	

7.   What type of FRUIT-JUICE do you drink?

Sweetened	
Unsweetened	

8.   What type of bread do you usually use?

White	
Brown	
Wholewheat	
Rye	

9. Do you usually buy SLICED BREAD?   YES / NO

If not, then how thick is the slice of bread you normally cut? _____

10. Which of the following do you normally use?

Spreads	On Bread	In Food Preparation
Butter		
Hard Margarine		
Soft Margarine (50% Polyunsaturated)		
Soft Margarine (medium fat e.g. Floro Lite)		
Oil		

11. How thickly do you usually spread margarine/butter on your bread?

Thin	
Medium	
Thick	

12. Do you eat LEAN or FATTY meat?

Lean	
Fatty	

If there is fat on the meat do you:

Eat the fat	
Remove the fat before eating	
Trim the fat after cooking	
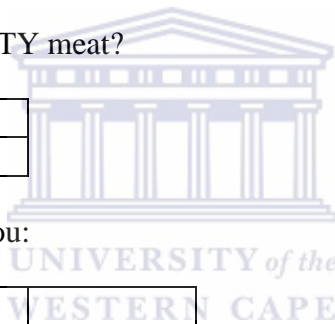
13. Do you eat the skin of poultry?   YES / NO

PLEASE NOTE IN DETAIL THE QUANTITY OF FOOD CONSUMED

The amount of food consumed may be indicated in any one of the following ways:

- WEIGHT, in ounces or grams. Please state whether the amount is the cooked or raw weight.
  e.g. chicken fillet (skinless) 100g (raw weight)

- VOLUME, e.g. 300ml Coke

- HOUSEHOLD MEASURES
  e.g. 1 mug, 1 teaspoon, 2 tablespoons

- DIMENSIONS, please describe the size
  e.g boerewors 10 cm; 1 small pizza 8 cm diameter

- If you are at a loss for words, DRAW the size of the food item on the back of the page.

METHOD OF PREPERATION

Please describe the method of preparation of the food.

e.g.   1 egg fried in butter
70g roast mutton, visible fat removed
1 baked apple (medium)

SUPPLEMENTS

Please give the description of any dietary supplements taken. State the commercial name, the manufacturer's name, the dosage and the amount consumed each day.

e.g.   10g LECITHIN POWDER, VITAL products
1 tablet ASCORBIC ACID, 100mg VITAL products
60g ENSURE POWDER, ABBOTT Laboratories.
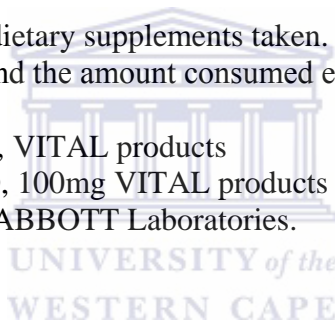
REMEMBER

- Eat as you usually do. Please try not to let the fact that you are keeping a food diary, interfere with your usual eating habits.

- The information is to be used for research purposes only, no-one will criticize your eating habits.

THANK-YOU

DAILY FOOD RECORD – DAY 1

DAY: _____ DATE: _____ NAME: _____

Meal Type	Time	Food Item	Description or Method of Preparation	Amount	Office Use

COMPLEMENTS: Optional, e.g., Nutritional supplements, etc.

NAME AND DESCRIPTION	QUANTITY

Appendix J: International Physical Activity Questionnaire (IPAQ)

FOR USE WITH YOUNG AND MIDDLE-AGED ADULTS (15-69 years)
The purpose of the questionnaires is to provide common instruments that can be used to obtain internationally comparable data on health–related physical activity.

We are interested in finding out about the kinds of physical activities that people do as part of their everyday lives. The questions will ask you about the time you spent being physically active in the **last 7 days**. Please answer each question even if you do not consider yourself to be an active person. Please think about the activities you do at work, as part of your house and yard work, to get from place to place, and in your spare time for recreation, exercise or sport. Think about all the **vigorous** and **moderate** activities that you did in the **last 7 days**. **Vigorous** physical activities refer to activities that take hard physical effort and make you breathe much harder than normal. **Moderate** activities refer to activities that take moderate physical effort and make you breathe somewhat harder than normal.

PART 1: JOB-RELATED PHYSICAL ACTIVITY

The first questions are about your work. This includes paid jobs, farming, volunteer work, course work and any other unpaid work that you did outside your home. Do not include unpaid work you might do around your home, like housework, yard work, general maintenance, and caring for your family. I will ask you about these later.

1.   Do you currently have a job or do any unpaid work outside your home?
      [WORK; Yes=1, No=0; 8, 9]
      _____   Yes
      _____   No *[Skip to PART 2]*
      8.   Don't Know/Not Sure *[Skip to PART 2]*
      9.   Refused *[Skip to PART 2]*

The following questions are about all the physical activity you did as part of your paid or unpaid work. This does not include traveling to and from work.

First, think about all the *vigorous* activities which take *hard physical effort* that you did as part of your work. Vigorous activities make you breathe much harder than normal. These may include things like heavy lifting, digging, heavy construction work, or climbing up stairs. Think about only those vigorous physical activities that you did for at least 10 minutes at a time.

2.   During the **last 7 days**, on how many days did you do **vigorous** physical activities **as part of your work**? [OVDAY; Range 0-7, 8, 9]
      _____   Days per week *[If respondent answers 0, skip to Question 4]*
      8.   Don't Know/Not Sure *[Skip to Question 4]*
      9.   Refused *[Skip to Question 4]*

      [Clarification: Think about only those physical activities that you did for at least 10 minutes at a time.]

[Clarification: Work includes paid and unpaid work as well as course work. Include all jobs and volunteer work.]

3.   How much time did you usually spend on one of those days doing **vigorous** physical activities as part of your work?

   ___ ___   Hours per day [OVDHRS; Range 0-16]

   ___ ___ ___   Minutes per day [OVDMIN; Range 0-960, 998, 999]

   998.   Don't Know/Not Sure

   999.   Refused

   [**Clarification**: Think about only those physical activities you did for at least 10 minutes at a time.]

   [**Clarification**: An average time per day is being sought. If you can't answer because the pattern of time spent varies widely from day to day, or includes time spent doing a variety of paid and unpaid work, ask: "What is the total amount of time you spent **over the last 7 days** doing vigorous physical activities as part of your work?"]

Now think about activities which take *moderate* physical effort that you did as part of you work. Moderate physical activities make you breathe somewhat harder than normal and may include activities like carrying light loads. Do not include walking. Again, think about only those moderate physical activities that you did for at least 10 minutes at a time.

4.   During the **last 7 days**, on how many days did you do **moderate** physical activities **as part of your work**? [OMDAY; Range 0-7, 8, 9]

   _____   Days per week *[If respondent answers 0, skip to Question 6]*

   8.   Don't Know/Not Sure *[Skip to Question 6]*

   9.   Refused *[Skip to Question 6]*

   [**Clarification**: Think about only those physical activities that you did for at least 10 minutes at a time.]

   [**Clarification**: Work includes paid and unpaid work as well as course work. Include all jobs.]

5.   How much time did you usually spend on one of those days doing **moderate** physical activities as part of your work?

   ___ ___   Hours per day [OMDHRS; Range 0-16]

   ___ ___ ___   Minutes per day [OMDMIN; Range 0-960, 998, 999]

   998.   Don't Know/Not Sure

   999.   Refused

   [**Clarification**: Think about only those physical activities you did for at least 10 minutes at a time.]

[**Clarification**: An average time per day is being sought. If you can't answer because the pattern of time spent varies widely from day to day, or includes time spent doing a variety of paid and unpaid work, ask: "What is the total amount of time you spent **over the last 7 days** doing moderate physical activities as part of your work?"]

Now think about the time you spend *walking* for at least 10 minutes at a time as part of your work. Please do not count any walking you did to travel to or from work.

6. **During the** last 7 days**, on how many days did you** walk as part of your work**?**
   [OWDAY; Range 0-7, 8, 9]

   _____ Days per week *[If respondent answers 0, skip to PART 2]*
   8. Don't Know/Not Sure *[Skip to PART 2]*
   9. Refused *[Skip to PART 2]*

   [**Clarification**: Think about only the walking that you did for at least 10 minutes at a time.]

   [**Clarification**: Include all jobs.]

7. How much time did you usually spend on one of those days **walking** as part of your work?
   ___ ___ Hours per day [OWDHRS; Range 0-16]
   ___ ___ ___ Minutes per day [OWDMIN; Range 0-960, 998, 999]
   998. Don't Know/Not Sure
   999. Refused

   [**Clarification**: Think about only the walking you did for at least 10 minutes at a time.]

   [**Clarification**: An average time per day is being sought. If you can't answer because the pattern of time spent varies widely from day to day, or includes time spent doing a variety of paid and unpaid work, ask: "What is the total amount of time you spent walking **over the last 7 days** as part of your work?"]

PART 2: TRANSPORTATION PHYSICAL ACTIVITY

Now, think about how you traveled from place to place, including to places like work, stores, movies and so on.

8. During the **last 7 days**, on how many days did you **travel in a motor vehicle** like a train, bus, car or tram? [TMDAY; Range 0-7, 8, 9]
   _____ Days per week *[If respondent answer 0, skip to Question 10]*
   8. Don't Know/Not Sure *[Skip to Question 10]*
   9. Refused *[Skip to Question 10]*

9. How much time did you usually spend on one of those days **traveling** in a car, bus, train or other kind of motor vehicle?

   ___ ___ Hours per day [TMDHRS; Range 0-16]

   ___ ___ ___ Minutes per day [TMDMIN; Range 0-960, 998, 999]

998. Don't Know/Not Sure

999. Refused

[**Clarification**: An average time per day is being sought. If you can't answer because the pattern of time spent varies widely from day to day, ask: "What is the total amount of time you spent **over the last 7 days** traveling in a motor vehicle?"]

Now think only about the *bicycling* you did to travel to and from work, to do errands, or to go from place to place. Only include bicycling that you did for at least 10 minutes at a time.

10. During the **last 7 days**, on how many days did you **bicycle** to go from place to place?
[TBDAY; Range 0-7, 8, 9]

   _____ Days per week *[If respondent answers 0, skip to Question 12]*

8. Don't Know/Not Sure *[Skip to Question 12]*

9. Refused *[Skip to Question 12]*

[**Clarification**: Think only about the bicycling that you did for at least 10 minutes at a time.]

11. How much time did you usually spend on one of those days **to bicycle** from place to place?

   ___ ___ Hours per day [TBDHRS; Range 0-16]

   ___ ___ ___ Minutes per day [TBDMIN; Range 0-960, 998, 999]

998. Don't Know/Not Sure

999. Refused

[**Clarification**: Think about only the bicycling that you did for at least 10 minutes at a time.]

[**Clarification**: An average time per day is being sought. If you can't answer because the pattern of time spent varies widely from day to day, ask: "What is the total amount of time you spent bicycling **over the last 7 days** to travel from place to place?"]

Now think only about the *walking* you did to travel to and from work, to do errands or to go from place to place. Only include walking that you did for at least 10 minutes at a time.

12. During the **last 7 days**, on how many days did you **walk to go from place to place**?
[TWDAY; Range 0-7, 8, 9]

   _____ Days per week *[If respondent answers 0, skip to PART 3]*

8. Don't Know/Not Sure *[Skip to PART 3]*

9. Refused *[Skip to PART 3]*

[**Clarification**: Think only about the walking that you did for at least 10 minutes at a time.]

13.   How much time did you usually spend on one of those days **walking from place to place**?

   ___ ___   Hours per day [TWDHRS; Range 0-16]
   ___ ___ ___   Minutes per day [TWDMIN; Range 0-960, 998, 999]
   998.   Don't Know/Not Sure
   999.   Refused

[**Clarification**: Think about only the walking that you did for at least 10 minutes at a time.]

[**Clarification**: An average time per day is being sought. If you can't answer because the pattern of time spent varies widely from day to day, ask: "What is the total amount of time you spent **over the last 7 days** walking from place to place?"]

PART 3: HOUSEWORK, HOUSE MAINTENANCE AND CARING FOR FAMILY

Now think about the physical activities you have done in the last 7 days in and around your home, like housework, gardening, yard work, general maintenance work, and caring for your family.

First think about *vigorous* activities which take hard physical effort that you did in the garden or yard. Vigorous activities make you breathe much harder than normal and may include heavy lifting, chopping wood, shoveling snow, or digging. Again, think about only those vigorous physical activities that you did for at least 10 minutes at a time.

14.   During the **last 7 days**, on how many days did you do **vigorous** physical activities **in the garden or yard**? [GVDAY; Range 0-7, 8, 9]

   _____   Days per week *[If respondent answers 0, skip to Question 16]*
   8.   Don't Know/Not Sure *[Skip to Question 16]*
   9.   Refused *[Skip to Question 16]*

[**Clarification**: Think about only those physical activities that you did for at least 10 minutes at a time.]

15.   How much time did you usually spend on one of those days doing **vigorous** physical activities in the garden or yard?

   ___ ___   Hours per day [GVDHRS; Range 0-16]
   ___ ___ ___   Minutes per day [GVDMIN; Range 0-960, 998, 999]
   998.   Don't Know/Not Sure
   999.   Refused

[**Clarification**: Think about only those physical activities that you did for at least 10 minutes at a time.]

[**Clarification**: An average time per day is being sought. If you can't answer because the pattern of time spent varies widely from day to day, ask: "What is the total amount of time you spent **over the last 7 days** doing vigorous physical activities in the garden or yard?"]

Now think about activities which take *moderate* physical effort that you did in the garden or yard. Moderate physical activities make you breathe somewhat harder than normal and may include carrying light loads, sweeping, washing windows, and raking. Again, include only those moderate physical activities that you did for at least 10 minutes at a time.

16. During the **last 7 days**, on how many days did you do **moderate** activities **in the garden or yard**? [GMDAY; Range 0-7, 8, 9]

     _____ Days per week *[If respondent answers 0, skip to Question 18]*
     8. Don't Know/Not Sure *[Skip to Question 18]*
     9. Refused *[Skip to Question 18]*

     [**Clarification**: Think about only those physical activities that you did for at least 10 minutes at a time.]

17. How much time did you usually spend on one of those days doing **moderate** physical activities in the garden or yard?

     ___ ___   Hours per day [GMDHRS; Range 0-16]
     ___ ___ ___   Minutes per day [GMDMIN; Range 0-960, 998, 999]
     998. Don't Know/Not Sure
     999. Refused

     [**Clarification**: Think about only those physical activities that you did for at least 10 minutes at a time.]

     [**Clarification**: An average time per day is being sought. If you can't answer because the pattern of time spent varies widely from day to day, ask: "What is the total amount of time you spent **over the last 7 days** doing moderate physical activities in the garden or yard?"]

Now think about activities which take at least *moderate* physical effort that you did *inside your home*. Examples include carrying light loads, washing windows, scrubbing floors, and sweeping. Include only those moderate physical activities that you did for at least 10 minutes at a time.

     [**Clarification**: Moderate activities make you breathe somewhat harder than normal.]

18. During the **last 7 days**, on how many days did you do **moderate** activities **inside your home**? [HMDAY; Range 0-7, 8, 9]

     _____ Days per week *[If respondent answers 0, skip to PART 4]*

     8. Don't Know/Not Sure *[Skip to PART 4]*

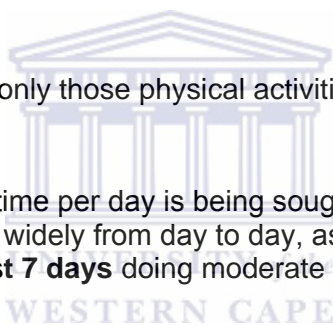   9. Refused *[Skip to PART 4]*

[**Clarification**: Think about only those physical activities that you did for at least 10 minutes at a time.]

[**Clarification**: During the last 7 days, on how many days did you do activities that take **at least moderate** effort inside your home?]

19. How much time did you usually spend on one of those days doing **moderate** physical activities inside your home?

     ___ ___ Hours per day [HMDHRS; Range 0-16]

     ___ ___ ___ Minutes per day [HMDMIN; Range 0-960, 998, 999]

     998. Don't Know/Not Sure

     999. Refused

[**Clarification**: Think about only those physical activities that you did for at least 10 minutes at a time.]

[**Clarification**: An average time per day is being sought. If you can't answer because the pattern of time spent varies widely from day to day, ask: "What is the total amount of time you spent **over the last 7 days** doing moderate physical activities inside your home?"]

PART 4: RECREATION, SPORT, AND LEISURE-TIME PHYSICAL ACTIVIITY

Now, think about all the physical activities that you did in the last 7 days solely for recreation, sport, exercise or leisure. Please do not include any activities you have already mentioned.

20. Not counting any walking you have already mentioned, during the **last 7 days**, on how many days did you **walk** for at least 10 minutes at a time **in your leisure time**? [LWDAY; Range 0-7, 8, 9]

     _____ Days per week *[If respondent answers 0, skip to Question 22]*

     8. Don't Know/Not Sure *[Skip to Question 22]*

     9. Refused *[Skip to Question 22]*

[**Clarification**: Think about only the walking that you did for at least 10 minutes at a time.]

21. How much time did you usually spend on one of those days **walking** in your leisure time?

     ___ ___ Hours per day [LWDHRS; Range 0-16]

___ ___ ___   Minutes per day [LWDMIN; Range 0-960, 998, 999]

998.   Don't Know/Not Sure

999.   Refused

22.   During the **last 7 days**, on how many days did you do **vigorous** physical activities like aerobics, running, fast swimming, or fast bicycling **in your leisure time**? [LVDAY; Range 0-7, 8, 9]

_____   Days per week *[If respondent answers 0, skip to Question 24]*

8.   Don't Know/Not Sure *[Skip to Question 24]*

9.   Refused *[Skip to Question 24]*

23.   How much time did you usually spend on one of those days doing **vigorous** physical activities in your leisure time?

___ ___   Hours per day [LVDHRS; Range 0-16]

___ ___ ___   Minutes per day [LVDMIN; Range 0-960, 998, 999]

998.   Don't Know/Not Sure

999.   Refused

[**Clarification**: Think about only those physical activities that you did for at least 10 minutes at a time.]

[**Clarification**: An average time per day is being sought. If you can't answer because the pattern of time spent varies widely from day to day, ask: "What is the total amount of time you spent **over the last 7 days** doing vigorous physical activities in your leisure time?"]
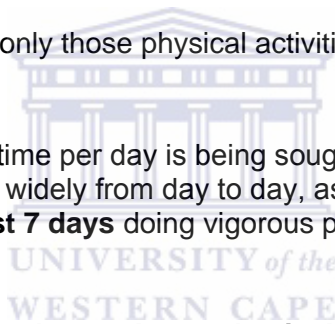
Now think about activities which take *moderate* physical effort that you did in your leisure time. Examples include bicycling at a regular pace, swimming at a regular pace, and doubles tennis. Again, include only those moderate activities that you did for at least 10 minutes at a time.

[**Clarification**: Moderate physical activities make you breathe somewhat harder than normal.]

24.   Again, think about only those physical activities that you did for at least 10 minutes at a time. During the **last 7 days**, on how many days did you do **moderate** physical activities like bicycling at a regular pace, swimming at a regular pace, and doubles tennis **in your leisure time**? [**LMDAY; Range 0-7, 8, 9**]

_____   Days per week *[If respondent answers 0, skip to PART 5]*

8.   Don't Know/Not Sure *[Skip to PART 5]*

9.   Refused *[Skip to PART 5]*

25.   How much time did you usually spend on one of those days doing **moderate** physical activities **in your leisure time**? [**LMDAY; Range 0-7, 8, 9**]

___ ___   Hours per week [LMWHRS; Range 0-112]

___ ___ ___ ___   Minutes per week [LMWMIN; Range 0-6720, 9998, 9999]

9998.   Don't Know/Not Sure

9999.   Refused

[**Clarification**: Think about only those physical activities that you did for at least 10 minutes at a time.]

[**Clarification**: An average time per day is being sought. If you can't answer because the pattern of time spent varies widely from day to day, ask: "What is the total amount of time you spent **over the last 7 days** doing moderate physical activities in your leisure time?"]

PART 5: TIME SPENT SITTING

The last questions are about the time that you spent *sitting* during the last 7 days. Include time at work, at home, while doing course work and during leisure time. This may include time spent sitting at a desk, visiting friends, reading or sitting or lying down to watch television. Do not include any time spent sitting in a motor vehicle that you have already told me about.

26.   During the **last 7 days**, how much time did you usually spend **sitting** on a **weekday**?

___ ___   Hours per day [SDHRS; Range 0-16]

___ ___ ___   Minutes per day [SDMIN; Range 0-960, 998, 999]

998.   Don't Know/Not Sure

999.   Refused

[**Clarification**: Include time spent lying down (awake) as well as sitting.]

[**Clarification**: An average time per day is being sought. If you can't answer because the pattern of time spent sitting varies widely from day to day, ask: "How much time in total did you spend sitting on a **weekend day**?"]

27.   During the last 7 days, how much time did you usually spend **sitting** on a **weekend day**?

___ ___   Hours per day [SEHRS; Range 0-16]

___ ___ ___   Minutes per day [SEMIN; Range 0-960, 998, 999]

998.   Don't Know/Not Sure

999.   Refused

[**Clarification**: Include time spent lying down (awake) as well as sitting.]

[**Clarification**: An average time per day is being sought. If you can't answer because the pattern of time spent sitting varies widely from day to day, ask: "How much time in total did you spend sitting on **Saturday**?"]

Appendix K: Stages of "Readiness to Change" Questionnaire

(Tick the appropriate box)

STAGE

1. PRECONTEMPLATIVE: At the present time, I am NOT INTERESTED in making a change in my health-related behaviour. ☐

2. CONTEMPLATIVE: At the present time, I am THINKING ABOUT in making a desired change in my health-related behaviour. ☐

3. PREPARATORY: At the present time, I AM DOING SOME PHYSICAL ACTIVITY, but not meeting the recommended criteria for health-related benefits, that is, ☐

HEALTH-RELATED CRITERIA FOR PHYSICAL ACTIVITY		
Intensity	Moderate-to-Intense Physical activity	Vigorous Physical Activity
Duration	30 min per session	$\geq$ 20 min per session
Frequency	$\geq$ 5 days per week	3 – 5 days per week

4. ACTIVE: At the present time, I AM DOING REGULAR PHYSICAL ACTIVITY, and meet the above-refernced (preparation) criteria on a consistent basis, but I HAVE NOT MAINTAINED the behaviour for 6 months. ☐

5. MAINTENANCE: At the present time, I AM DOING REGULAR PHYSICAL ACTIVITY FOR 6 MONTHS OR MORE, AND MEETING the recommended criteria for health-related benefits. ☐

6. RELAPSE: At present, I have not MAINTAINED REGULAR PHYSICAL ACTIVITY FOR 6 MONTHS. ☐

Appendix L: Determinants of Behaviour and Strategies Used to Overcome and Change Negative Behaviours

At part of the intervention strategy, it was explained to students that unusual emotional responses associated with withdrawal from previous risky behaviour was normal and expected as part of the 'journey of healing'. Moreover, if there was a history of failed attempts at behaviour change, the resulting fear of failure in future could be a critical emotional hurdle to overcome. The greater the extent to which a student was able to identify with certain risky behaviour(s) in the past as acceptable and tolerable, the greater would be the emotional trauma experienced in relinquishing such behaviour(s). Bearing this in mind, the process of behaviour change started with students identifying moments wherein they derived significant self-esteem and personal satisfaction from overcoming certain CHD risk factors, and using this as a foundation to strategize a plan of action for managing the positive risk factors.

Many factors influence an individual's psychological response to conditioned behaviour, and may also influence their ability to comply with an intervention programme. Some of the major considerations in this regard relate to the following:

- Behaviour influences: cause, onset and seriousness of the behaviour, and potential impact on health
- Personal influences: age, gender, personality, maturity and level of education of the individual; previous history of dealing with the behaviour, pain tolerance and expression; past influences of family and/or intimate others, past experiences with professional support;
- Social influences: social support of friends, peers and community, predisposing environmental conditions and life experiences, ethnic, cultural and religious background.

Research assistants were prepared to anticipate a wide range of psychological responses to behaviour change in students. Normal responses included the following:
- Anger, frustration, sadness and a strong desire to relapse into previous risky behaviour
- Some denial and minimization of the risky behaviour
- Concern about emotional trauma, even pain
- Feeling discouraged about how long the process is taking
- Concern about the loss of control and long-term adherence

The critical issue for the research assistant was being able to distinguish between students who were just 'blowing off steam' and those who were showing signs and symptoms of poor adjustment to behaviour change. Students in the latter category were referred to professional student health services: medical, counseling, exercise and nutritional services.

Research assistants were also instructed to be vigilant when adverse psychological responses tended to be progressive and did not resolve with time or adequate rest. The

research assistants were in the ideal position to identify students with adjustment difficulties, encouraged to establish a rapport with them to show empathy and enabling them to share more of their concerns and challenges. Some of the warning signs and symptoms of students with maladjustment behaviours included the following:

- Denial, reflected in remarks such as "the risk is no big deal" and the student making an extraordinary effort to convince others that he/she was in control and that the risky behaviour did not matter
- Loss of enthusiasm and motivation
- Demonstration of anger, depression, confusion, anxiety or apathy with the appearance of little or no progress in behaviour change
- a history of relapse
- Constant whining and dwelling on minor aches and pains, which was a possible indication that the student was overly anxious of a relapse.
- Unusual dependence on the research assistant or intervention or spending too much time in treatment
- Withdrawal/isolation/alienation from family, friends, peers and social activities
- Burnout and feelings of chronic fatigue
- Sudden mood swings without apparent provocation
- Statements that indicate helplessness to affect change such as "I can't seem to kick the habit…" and lack of confidence, "It's just too hard…" or procrastination "I don't have the time right now, but I'll do it later when I can be more focused…"

These warning signs and symptoms were not indicative of 'impending doom', but were suggestive of something going wrong with the student that warranted closer individualized attention.

Focus of the Intervention

Do's for research assistants:

- To let students know what to expect on their journey of behaviour change
- To educate students about their risk-taking behaviours
- To explain the relevance of each of the stages in the intervention programme to reinforce compliance and establish credibility
- To focus more on the plan of behaviour change and future outcomes than on present transient difficulties
- Explain the reality of behaviour change: total commitment and effort all the time was difficult and some relapse was inevitable
- Establish a rapport to create a shared partnership
- Be realistic about change, but to remain committed and positive over the long-term
- To progressively empower students to personally take responsibility for their progress through joint decision-making initially, and be held accountable for the outcomes

- To reinforce daily programme compliance and improvement no matter how small, emphasizing that long-term change starts with taking 'baby steps' now
- Using emails and SMS's to supplement verbal information and show support
- To listen attentively and empathetically
- Assess every students likelihood of adhering to the intervention and respond accordingly
- Monitor progress
- Be firm and set sound rules so that students knew what to expect and which boundaries to observe
- To involve others into the student's support network: peers, family, significant others, friends, etc.
- To tailor the intervention to meet the students individual needs and circumstances
- To recognize personality and attitudinal differences in students, so as not to trivialize or minimize their experiences
- Set SMART short-and long-term goals
- To share difficulties and success stories with others
- Allow free expression for students emotional and traumatic experiences, within acceptable boundaries
- Base effective behaviour change on specific criteria to clarify expected outcomes
- To ensure early successful experiences in the programme, and continued success throughout
- Demonstrate confidence and belief in the students' ability to change

Don'ts for research assistants:

- Overload the students with information
- Allow students to admit defeat and give up on the programme
- Be cornered into overly serious discussions by difficult students who tended to have a history of resistance or intolerance towards behaviour change
- Use technical terminology and complex concepts when educating students
- Use threats and scare tactics as motivational tools
- be inflexible and dogmatic about the change process
- allow students to stay away from or miss daily programme commitments

Several psychological skills were used in the behavioural change process, such as communication, goal-setting, positive self-talk, and relaxation techniques as helpful skills for participants to use both during and even after the intervention period. The purpose of the psychological intervention with the at-risk participants was to facilitate the behavioural change process, maintain participants emotional stability, mobilize coping resources and enhance mental readiness for maintenance of the changed behaviour (stage 5 in the model of 'stages of readiness to change' process) and promote self-efficacy.

When introducing the psychological intervention strategies, research assistants were informed to be careful to present the concepts to participants in a non-threatening manner. People, in general, are accustomed to having a 'sense of control' over their environment. Once involved in risk-taking behaviour, the student is placed in a position of 'dependency'. The subsequent feelings of helplessness can be uncomfortable for students who were accustomed to being in control. During the change process, assistants were informed to make sure that the students had a vital role to play in the intervention programme. Helping the student regain and maintain a sense of control would facilitate both physical and psychological recovery.

Psychological Skills

Communication

Communication as a tool for education is a simple but essential component of the intervention programme. The first step in establishing good communication with the participant was to establish rapport or dialogue in order to foster trust if the research assistant is to be valued and respected. An essential element in this communication process is listening, not just to the spoken word but especially to the manner in which it was said, and the associated posturing or body language. Suggestions to promote better communication were the following:

- Understanding the problem before trying to fix it: listening carefully to what the student was saying about their risky behaviour and paraphrasing or summarizing what was said to uncover the emotional meaning, e.g., "You seem to be anxious about an approaching test…."
- Valuing the student's input: change is a collaborative process, so it is important to seek out the student's input in a manner that communicated respect.
- Checking perceptions and getting specifics: just because students don't ask questions about the intervention does not mean that they understood the process and goals set. Assistants were instructed to periodically ask questions about the exercise prescription or dietary guidelines in order to assess how students were coping.
- Listen for the "but": part of being a good listener is paying attention to the content and structure of what is being said and then intervene according to the student's needs not wants, e.g., "During the week the programme is kinda like great, but over weekends I struggle a bit." Clearly, such an individual wants added support over weekends, but needed to change his/her lifestyle in order to manage the risky behaviour.

Too often research assistants had a tendency to focus on the physical aspects of behaviour change, but the psychosocial needs of the student should be seen as equally important. Initially, assistants provided health information about the modifiable risk factors, the proposed intervention strategy, and expectations for long-term maintenance in words the students could understand. Also to inform the students how they as research assistants would facilitate the process of behaviour change from start to finish.

Finally included was an explanation of the possibility of plateaus, setbacks or relapses and their importance in the change process.

Self-Communication

Positive self-communication, an intrapersonal form of self-talk, was encouraged amongst students to counteract negative thoughts through a systematic sequence of steps:

- Awareness: students becoming more consciously aware about dysfunctional or destructive thoughts that could be detrimental to successful change. This often related to the possible negative side-effects of or withdrawal from risky behaviour.
- Thought stoppage: deliberately using a trigger word such as "stop" to halt negative, destructive thoughts from becoming overwhelming.
- Replacement: replacing negative thoughts with positive, constructive ones.

Students were encouraged to anticipate when these negative thoughts occurred, to recognize them as anxiety-inducing stressors, and to keep a self-talk diary (for their personal use only) as part of the intervention strategy to gauge the frequency and severity of the occurrence, and how the situation was effectively managed.

Collaborative Goal-setting

It was expected that the students would be particularly comfortable and familiar with this strategy, because of their immediate educational achievements in passing matric and being admitted into higher education. This was used to break down the change process into manageable steps for continued progress and avoiding 'pitfalls', to provide motivation and commitment, to stick with the intervention programme (adherence), to divert the students' attention away from their risky behaviours towards controllable factors, to help build self-confidence, to regain control of the risky behaviour, and to be held accountable for their actions, good or bad. The S.M.A.R.T acronym was used for goal-setting.

Research assistants were informed not to assume that the students would all be comfortable with the treatment groups they were placed in or the techniques used during the intervention period. In addition, students might also feel isolated from family and or friends who previously indulged their risky behaviour, but now could not identify with the changed behaviour, especially if significant others had also succumbed to the risky behaviour. Most participants would need support during this vulnerable time and, therefore, a social support network was seen as necessary to make the participants feel that they did not have to deal with the change alone. Interacting with others who had experienced or were experiencing the change process by sharing of experiences was seen as particularly helpful and supportive and, therefore, formed a vital part of the weekly intervention strategy.

Research assistants were also asked to use a comfortable, non-threatening environment when communicating with students.

Finally, research assistants who found it difficult to deal with serious psychological issues from students, such as depression, undue stress, disordered eating behaviours, substance abuse, academic difficulties, etc. were instructed to refer the students to the professional counseling services at the Centre for Student Support Services on campus.

Appendix M: Titles of Weekly Articles Used in the Health Information Intervention Programme

WEEKS   **THEME**

Article 1:   Lifestyle Changes Begin with Personal Motivation

Article 2:   Success Through Goalsetting

Article 3:   Future Fitness Predictions

Article 4:   The Role of Nutrition in Good Health

Article 5:   Night-time Eating (Eat Late, Put on Weight)

Article 6:   Don't Skip Breakfast to Cut Calories

Article 7:   Walkers should aim for 100 steps per minute

Article 8:   Tips on Sticking with Exercise

Article 9:   Transforming Bad Habits

Article 10:   3 Things You're Aren't Doing for Fat Loss

Article 11:   How to Stick with any Diet or Fitness Programme

Article 12:   Barriers to Fitness

Article 13:   The 2 Pounds per Week Rule

Article 14:   Reduce Calories by Cutting Calorie Density

Article 15:   Listen to Maintainers not to Losers

Article 16:   Staying Motivated – Living without Limits

Appendix N: Titles of Weekly Articles Used in the Dietary Intervention Programme

<u>**WEEKS**</u>   <u>**THEME**</u>

Article 1:   Lifestyle Changes Begin with Personal Motivation

Article 2:   Success Through Goal-setting

Article 3:   Future Fitness Predictions

Article 4:   The Role of Nutrition in Good Health

Article 5:   Night-time Eating (Eat Late, Put on Weight)

Article 6:   Don't Skip Breakfast to Cut Calories

Article 7:   Down with Dumb Dieting

Article 8:   Top 10 Nutritional Mistakes Made by Active People

Article 9:   Transforming Bad Habits

Article 10:   3 Things You're Aren't Doing for Fat Loss

Article 11:   How to Stick with any Diet or Fitness Programme

Article 12:   5 Everyday Activities that can Burn Calories

Article 13:   The 2 Pounds per Week Rule

Article 14:   Reduce Calories by Cutting Calorie Density

Article 15:   Listen to Maintainers not to Losers

Article 16:   Staying Motivated – Living without Limits

Appendix O: Tester Reliability Form

Anthropometric Tester Reliability Form		
Tester Information	Surname	
	First name	
	Sex (Male=1, female=2)	
	Date of Birth	
	Ethnicity (B, C, I, W)	

Subject Information	Surname	
	First Name	
	Country	
	Ethnicity (B, C, I, W)	
	Sex (Male=1, female=2)	
	Sport	
	Date of Birth	

FIRST- TEST	Date of Measurement:				
	Time of Measurement:	**Measure**	**Measure**	**Measure**	**Final**
	Measurements	1	2	3	**Measures**
	Body mass (kg)				
	Stretch stature (cm)				
	Triceps sf (females) (mm)				
	Supra-iliac sf (females) (mm)				
	Front Thigh sf (females & males) (mm)				
	Chest/Pectoral sf (males) (mm)				
	Abdominal sf (males) (mm)				
	Waist girth (min.) (cm)				
	Gluteal girth (max.) (cm)				
	Systolic Blood Pressure (mm Hg)				
	Diastolic Blood Pressure (mm Hg)				

RETEST	Date of Measurement:				
	Time of Measurement:	**Measure**	**Measure**	**Measure**	**Final**
	Measurements	1	2	3	**Measures**
	Body mass (kg)				
	Stretch stature (cm)				
	Triceps sf (female) (mm)				
	Supra-iliac sf (female) (mm)				
	Front Thigh sf (female & male) (mm)				
	Chest/Pectoral sf (male) (mm)				
	Abdominal sf (male) (mm)				
	Waist girth (min.) (cm)				
	Gluteal girth (max.) (cm)				
	Systolic Blood Pressure (mm Hg)				
	Diastolic Blood Pressure (mm Hg)				

Appendix P: Testing Preparation Checklist*

Subject confirmation	Yes	No

The subject has:

	Yes	No
1. read and understood the test procedures	☐	☐
2. signed the consent form	☐	☐
3. been familiarized with the test(s) and is comfortable with it (them)	☐	☐
4. understood the starting and stopping procedures	☐	☐
5. understood the expectations before, during and after testing	☐	☐
6. complied with all pre-test instructions concerning: rest, food and drink, smoking, clothing and shoes.	☐	☐
7. no illnesses or injuries	☐	☐
8. not taken any medication	☐	☐
9. warmed-up properly, if required	☐	☐
10. confirmed being physically and psychologically ready for testing	☐	☐

Tester confirmation

The tester has:

	Yes	No
1. determined the tests to be administered	☐	☐
2. checked the equipment (and calibration, if required)	☐	☐
3. checked the recording sheets and other supplies, such as stationery, etc.	☐	☐
4. checked the testing area	☐	☐
5. understood the responsibilities clearly	☐	☐
6. confirmed who will be the recorder or assistant	☐	☐
7. understood the testing sequence	☐	☐
8. understood and rehearsed the emergency or safety procedures	☐	☐
9. understood the procedures before during and after testing	☐	☐
10. controlled the testing environment to ensure an atmosphere of privacy, safety and calmness	☐	☐

Comments: _____

THANK YOU

* (Checklist modified from Howley and Franks, 1997, p. 114)

Appendix Q: Confidentiality Declaration by Research Assistants

I, _____, have been informed and understand that the subject
    (Research assistant's name and surname)
information, study documentation, and related research materials obtained by me or other

research staff linked to the research project entitled, "The impact of multiple behaviour health

intervention strategies on coronary artery disease risk, health-related physical fitness, and health-

risk behaviours of university students" will be treated as priviledged and confidential and will,

consequently, not be released or revealed to any person, without exception, without the

expressed written permission of the principle researcher.

Date: _____

Researcher's Signature: _____

Principle Researcher's Signature: _____

Appendix R: Emergency Procedures

In the event that an emergency should occur and **no medical personnel** are present, the following guidelines should be followed:

1. The researcher or research assistant identifies him/herself as a professional rescuer trained in emergency care. This helps to reassure the victim and bystanders. If the victim is conscious, legally one must ask permission to assist the victim. (The law assumes that an unconscious person would give consent.) The researcher or assistant should stay with the individual at all times. He/she should attempt to reassure the person and protect the individual from personal bodily harm. A senior research person will assume control of the situation and issue further orders as needed.

2. A second research member will call telephone extension: **2508**, Mr. Gonsalves of campus EMS, and the state EMS telephone: **10177**, and give the following information:

❖ Phone number and location (building name, address, specific suite or room number)
❖ Site-specific entrance instructions for ambulance driver
❖ Brief description of the problem. If it is a definite cardiac event, that is, respiratory arrest and CPR is in progress, an Advanced Life Support unit will be sent. If it is non-life threatening, that is, seizures, a Basic Life Support unit will be sent.
❖ After 2508 has been called, a research member will notify Campus Protection Services (telephone extension: 2564), and wait in the lobby to meet the ambulance at the clinic entrance to escort them to the emergency.

3. The individual should be monitored at all times. This will include:

❖ Checking a heart rate, noting the regularity and strength of each heart beat
❖ Monitoring and recording blood pressure
❖ Observing skin colour and breathing pattern
❖ Maintaining open airway
❖ Establishing unresponsiveness and initiating CPR when appropriate
❖ Before the individual is transported (if unconscious), give the Ambulance Assistant as much information as possible regarding individuals: n*ame, age, medical considerations, and home phone emergency numbers. (The attending physician and the hospital will normally make the call to the family).*

4. Once the individual is transported, the senior staff person in charge should:

❖ Notify the individual's work place so that the employer can decide how to handle the family.
❖ Assume responsibility for personnel belongings, and valuables. Please remember that it is important to respect the individual's privacy. Be as brief as possible, when disclosing the information pertinent to the event.

❖ Fill out an injury/accident report, and file one copy in the member's folder and one copy with the researcher.