Whole body vibration as training modality in selected physical, physiological, haemodynamic, and biochemical parameters

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Foreword

"For of Him and through Him and to Him are all things..." Rom. 11:36

Probably the only people who will be reading this thesis will be the people determining whether or not I get a degree! How short minded would it be to spend four years of your life on a book that will only reach the eyes of few. It was only when I realized that this project should serve the majority of One, that the planning became purposeful and fun.

The project was inspired by:

- Our receptionist, who wishes to study at tertiary level, but is unable to do so for a number of reasons.
- Oom Japie Badenhorst who gyms at the age of 91, proclaiming to us each morning that he knew not a better time.
- My two beautiful sisters, Anéll Groenewald and Louise Dreyer, who know no limits and have mastered the art of true friendship.

I was humbled by:

- My parents who, once again, supported me financially and emotionally. "Mom and Dad, I know I cannot thank or repay you in the measure that you deserve, but may God give me the opportunity to pay it forward. Your kindness will not be in vain."

I was impressed by:

- God's timing! His timing is simply exquisite. In everything He provided exactly enough at exactly the right time for exactly the right purpose.
- The man whose spouse I will shortly become – Ben Foulds, your value is inexpressible.

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- The participants of the study. I hope you gained as much health benefits as I have gained knowledge.

I planned to serve, but was served in return.

The Author
December 2011

The testing team

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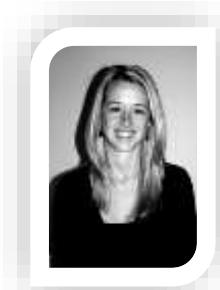
<tr><td>  </td><td>  </td><td>  </td></tr>
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Plagiarism statement

I, Berna de Kock (student nu. 2008008762) am registered for a Ph.D-degree in the Department of Exercise- and Sport Sciences, in the Faculty Humanities at the University of the Free State, Bloemfontein campus.

Title of the project:

Whole-body vibration as training modality on selected physical, physiological, haemodynamic, and biological parameters

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The author Date

Abstract

Problem statement and aim: In the Biokinetics practice the safety and effectiveness of whole-body vibration training is frequently queried. Literature supports whole-body vibration exercise and training in terms of improvement in body composition, muscle strength, flexibility, posture, and pain. A scarcity of research, however, addresses the training effect of whole-body vibration in variables that influence cardiovascular disease risk. For this reason, the study aimed to investigate the effect of WBVT on body composition, cardiovascular function, blood lipids, blood glucose, and metabolism.

Methods and procedures: Baseline testing was performed on two groups, namely an exercise group (N=23) and control group (N=17). Testing included measurement of body composition, cardiovascular function, blood lipids, blood glucose, and metabolism. The exercise group was submitted to a 12-week progressive whole-body vibration exercise intervention program (f=30-40 Hz; A=2-6 mm; t=30-60 s) during which time the control group remained sedentary. After the 12 weeks, baseline tests were repeated and differences determined.

Results: Findings that can be attributed to whole-body vibration training comprised improvements in body composition, systolic and diastolic blood pressure, double product, end-diastolic and end-systolic volumes, ejection fraction and left-ventricular myocardial cell velocities, total cholesterol and LDL, oxygen uptake and fat oxidation.

Conclusion: Whole-body vibration training over a period of 12 weeks beneficially influences body composition, the cardiovascular system, the blood lipid profile and metabolism of apparently healthy sedentary men.

Keywords: whole body vibration, body composition, body fat percentage, blood pressure, haemodynamics, echocardiography, blood lipids, blood glucose, metabolism, oxygen consumption, substrate oxidation

Opsomming

Probleem- en doelstelling: In die Biokinetika praktyk word al hoe meer navrae rakende die veiligheid en effektiwiteit van totale liggaamsvibrasieoefening aan oefenkundiges gerig. Literatuur ondersteun totale liggaamsvibrasieoefening in terme van die verbetering van liggaamsamestelling, spiertonus en spierkrag, soepelheid, postuur en pyn. Daar is egter ‘n tekort aan inligting rakende die inoefeningseffek van totale liggaamsvibrasie op veranderlikes wat kardiovaskulêre siekterisiko raak. Hierdie studie het gevolglik ten doel om die inoefeningseffek van totale liggaamsvibrasie op liggaamsamestelling, kardiovaskulêre veranderlikes, bloedlipiede, bloedglukose en metabolisme te ondersoek.

Metode van ondersoek: Twee groepe, nl. ‘n oefengroep (N=23) en kontrolegroep (N=17) het basislyntoetsing ondergaan vir liggaamsamestelling, kardiovaskulêre funksie, bloedlipiede, bloedglukose en metabolisme. Die oefengroep is aan ‘n 12-week progressiewe totale liggaamsvibrasie oefenprogram onderwerp (*f*=30-40 Hz; *A*=2-6 mm; *t*=30-60 s). Gedurende hierdie tyd het die kontrolegroep sedentêr gebly. Na afloop van die 12 weke is die basislyntoetse herhaal en verskille is bepaal.

Resultate: Bevindinge wat aan die totale liggaamsvibrasie oefeningsintervensie toegeskryf kan word sluit verbetering in liggaamsamestelling, sistoliese en diastoliese bloeddruk, dubbelproduk, eind-diastoliese en eind-sistoliese volumes, ejeksiefraksie en linker ventrikulêre spierkontraksiesnelhede, totale cholesterol en laedigtheidslipoproteïen-cholesterol, asook suurstofverbruik en vetverbranding in.

Gevolgtrekking: Totale liggaamsvibrasieoefening oor ‘n tydperk van 12 weke het ‘n positiewe invloed op die liggaamsamestelling, kardiovaskulêre sisteem, bloedlipiedbeeld en metabolisme van sedentêre, oënskynlik gesonde mans.

Sleutelwoorde: Totale liggaamsvibrasie, liggaamsamestelling, liggaamsvetpersentasie, bloeddruk, haemodinamika, sonografie, bloedlipiede, bloedglukose, metabolisme, suurstofverbruik, substraat oksidasie.

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List of abbreviations

%BF	Percentage body fat
A	Amplitude
A	Trans mitral flow velocity during active ventricular filling
beats.min^{-1}	Beats per minute
BMI	Body-mass Index
BP	Blood Pressure
Cardio Chek PA	Cardio Chek portable analyzer
CHO	Carbohydrates
Cir. Waist	Circumference of the waist
cm	Centimeter
CR	Cardio Respiratory
CVD	Cardiovascular disease
DBP	Diastolic blood pressure
E	Trans mitral flow velocity during passive ventricular filling
f	Frequency
FFM	Fat free mass
FM	Fat mass
g	Acceleration
g.mm^{-2}	Grams per square millimeter
HbA_{1c}	Glycosylated hemoglobin
HDL	High density lipoprotein
HR	Heart rate
HRR	Heart rate reserve
I/R	Ischemia/Reperfusion
IGF	Insulin growth factor
IGT	Impaired glucose tolerance
ISO	International organization for standardization
kg	Kilogram
kg.m^{-2}	Kilogram per square meter
LDL	Low density lipoprotein
m	Meter
m.s^{-1}	Meters per second
m^2	Square meter
mg.dL^{-1}	Milligrams per deciliters
MHR	Maximum heart rate
min.	Minute
mL·kg^{-1}·min^{-1}	Milliliters per kilogram per minute
mm	Millimeter
mm.s^{-1}	Millimeters per second
mmol.L^{-1}	Millimoles per liter
N	Newton
n.a.	Not applicable
nl.	Naamlik
PCr	Phosphocreatine
p-p	Peak-to-peak
PVC	Premature Ventricular Complex
RPE	Rate of perceived exertion

RQ	Respiratory quotient
SBP	Systolic blood pressure
SV	Side-alternating vertical sinusoidal vibration
t	Time
TC	Total Cholesterol
TDI	Tissue Doppler imaging
TVI	Time velocity integral
UFS	University of the Free State
$\dot{V}O_2$	Peak oxygen uptake
$\dot{V}O_{2\text{-max}}$	Maximum oxygen uptake
VV	Synchronous sinusoidal vibration
WBV	Whole body vibration
WBVT	Whole body vibration training
WC	Waist circumference
WHR	Waist-to-hip ratio

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Chapter 3: Methods and procedures

Chapter 4: Results and discussion

1

Problem statement and aim of the study

1.1 Introduction

Over the past two decades, whole-body vibration (WBV) has become an attractive exercise modality to diseased populations, the elderly and the general public. Not only due to its time effectiveness, but also because of its beneficial treatment possibilities and cosmetic effects (Signorile, 2008:20). Health-related effects of whole-body vibration training (WBVT), however, remain inconclusive, as literature argues both, benefits and risks.

Initial research into vibration exposure was primarily aimed at safety in working environments (Griffin, 1990:174). Research then addressed health-related implications of vibration exposure to a passive human body such as in airplane pilots and drivers of heavy-duty vehicles. Harmful consequences of vibration were noted at frequencies lower than 26 Hz and higher than 100 Hz (Griffin, 1990:174). They included damage to peripheral nerves, blood vessels, joints and perceptual function, as well as chest pain, internal bleeding, menstrual disorders, and abnormal child birth in severe cases (Gratsianskaya *et al.*, 1974:9; Roman, 1958:102).

1.2 Problem statement

In the past two decades whole-body vibration has evolved into a modality of exercise training. Instead of transmission of vibration through a passive body, vibration is now absorbed by the activated muscles through stretch-reflex actions that result in rapid muscle contractions (Van der Meer *et al.,* 2007:24). Benefits of exposure to active vibration have been recorded in the areas of strength and power (Becerra Motta & Becker, 2001:33; Bosco *et al.*, 1999:309), flexibility (Cochrane & Stannard, 2005:860), metabolic rate and energy expenditure (Zange *et al*., 2008:268; Van den Tillaar, 2006:192; Rittweger *et al.*, 2001:169) bone mineral density (Verschueren *et al.*, 2004:352; Rittweger *et al.,* 1999:134), posture (Polonyova & Hlavacka, 2001:408; Kavounoudias *et al.*, 2001:869), the treatment of pain (Weerakkody *et al.*, 2003:209) and skin and muscle perfusion (Lohman *et al.,* 2007:71). Few adverse effects have been reported in participants who maintained isometric muscle contractions during vibration training in a functional body position. Mester *et al*. (2006:1062) mentions headaches, lower-leg erythema, vertigo, and nausia, when extremely strong vibrations are applied.

Because all systems in the body, including the neuromuscular and cardiovascular systems, react to whole-body vibration (Yue & Mester, 2007:96), it is difficult to reduce research findings to a single system or a part thereof. Whether whole-body vibration influences the human body in a positive or negative way will depend on the type, time and intensity of vibration applied, as well as the properties of the body undergoing vibration (Mester *et al*., 2006:1059; Griffin, 1990:173).

In any exercise regime for patients with cardiovascular disease risk, or established cardiovascular disease, safety is of great concern. In comparison to conventional aerobic and resistance training, WBVT is researched to a far lesser extent and is also less frequently prescribed for diseased populations.

In order to minimize injury risk, Power-plate International (2008), a tri-directional vibration plate manufacturer and distributer, identified conditions where WBVT is contra-indicated. These include open wounds, recent surgery, cancer, and heavy migraine

(Power-plate International, 2008). They also advise individuals with established cardiovascular disease (CVD) or those at risk for developing CVD against unsupervised use of vibration platforms as training modality. This cautionary statement seems to be for the sake of prevention, as limited research addresses the influence of WBVT on cardiovascular disease and CVD risk (Bogaerts *et al.*, 2007:630; Mester *et al.*, 2006:1059). Consequently little support is offered to these individuals and WBVT-program prescription remains unclear. The scarcity of research especially on the topic of cardiac function strengthens the necessity for research in the area of WBVT in populations with CVD risk and CVD occurrence.

To address these essential spaces in the literature, this study will investigate the effects of progressive WBVT on selected variables associated with CVD; namely, physiological, haemodynamic and biochemical variables (Table 1.1). It is not known whether this type of intervention would be detrimental or beneficial to high-risk populations. In light of this, a low-risk population has been selected for this study.

In Biokinetics practices, patients with CVD or those carrying risk for developing CVD are frequently treated in the form of endurance and resistance training. These types of training have shown to effectively lower an individual's risk for developing CVD (ACSM, 2010:9). However, the literature lacks sufficient evidence for whole-body vibration as training modality aimed at lowering the risk of an individual developing CVD.

1.3 Research questions

Research questions that will be addressed in this study are the following:

- What are the effects of WBVT on variables associated with CVD risk?
- Are the effects beneficial or detrimental?

1.4 Objectives

In light of the research questions, the aim of the study was to investigate the effect(s) that WBVT might have on variables associated with CVD risk (Table 1.1). Cardiac function and metabolism (especially substrate oxidation) were new areas of research in the continuum of whole-body vibration training.

The aim of the study was split into six sub-divisions, namely:

1.4.1 To investigate the effect of WBVT on body composition variables i.e. percentage body fat (%BF), body mass index (BMI), waist circumference (WC), waist-to-hip ratio (WHR), and fat-free mass (FFM).

1.4.2 To explore the effect of WBVT on systolic and diastolic blood pressure.

1.4.3 To investigate the effect of WBVT on the blood lipid profile, which included high-density lipoprotein (HDL) cholesterol, low density lipoprotein (LDL) cholesterol, total serum cholesterol (TC), and triglycerides.

1.4.4 To investigate the effect of WBVT on blood glucose.

1.4.5 To explore the effect of WBVT on cardiac function (e.g. heart rate, filling volumes and myocardial velocities).

1.4.6 To determine the effect of WBVT on metabolism as measured by oxygen uptake ($\dot{V}O_2$), substrate oxidation (carbohydrates and fat), the respiratory exchange ratio (RER), and heart rate at rest, as well as during activity.

1.5 Hypotheses

The following outcomes were expected as result of WBVT over a period of 12 weeks:

1.5.1 Body composition will improve significantly.

1.5.2 Systolic and diastolic blood pressure will improve significantly.

1.5.3 Blood lipids will improve significantly.

1.5.4 Blood glucose will improve significantly.

1.5.5 Cardiac function will improve significantly.

1.5.6 Metabolism will react as follows:

1.5.6.1 Oxygen uptake ($\dot{V}O_2$) will remain unchanged during rest (pre-test vs. post-test) and decrease during activity (pre-test vs. post-test).

1.5.6.2 Fat oxidation will decrease during rest (pre-test vs. post-test) and activity (pre-test vs. post-test).

1.5.6.3 Carbohydrate oxidation will increase during rest (pre-test vs. post-test) and activity (pre-test vs. post-test).

1.5.6.4 The respiratory exchange ratio will remain constant during rest (pre-test vs. post-test) and decrease during activity (pre-test vs. post-test).

1.5.6.5 Heart rate will decrease during rest (pre-test vs. post-test) as well as during activity (pre-test vs. post-test).

Table 1.1: Variables investigated in sedentary young men who submitted to whole-body vibration training for an intervention period of 12 weeks

	Variable category	Variable	Risk factor	Risk term
Variables associated with cardiovascular disease				
1	Body composition	• BMI • %BF • WC • WHR • FFM	• ≥ 25.0 $kg.m^{-2}$ • ≥ 20% • ≥ 102 cm • ≥ 0.95 • n.a	Obesity (ACSM, 2010:63-72)
2	Blood pressure (resting)	• SBP • DBP	• ≥140 mmHg • ≥90 mmHg	Hypertension (ACSM, 2010:47)
3	Blood lipids (fasting)	• TC • HDL, • LDL, • Triglycerides	• ≥ 5.18 $mmol.L^{-1}$ • ≤ 1.04 $mmol.L^{-1}$ • ≥ 3.37 $mmol.L^{-1}$ • ≥ 1.7 $mmol.L^{-1}$	Dislipidaemia (ACSM, 2010:28)
4	Blood glucose (fasting)	• Blood glucose	• ≥ 5.5 $mmol.L^{-1}$ and ≤6.93 $mmol.L^{-1}$	Impaired glucose tolerance (ACSM, 2010:28)
5	Cardiac function (resting)	• Heart rate • Double product (RPP) • End-diastolic volume • End-systolic volume • Stroke volume (SV) • Ejection fraction (EF) • Shortening fraction (SF) • Time velocity integral (TVI) • Deceleration time (DT) • Passive filling velocity (E) • Active filling velocity (A) • Myocardial velocities at septal and lateral regions during active and passive filling	n.a	n.a
6	Metabolism (during rest and activity)	• $\dot{V}O_2$ • Fat oxidation • CHO oxidation	n.a	n.a

BMI = Body mass index; %BF = percentage body fat; WC = waist circumference; WHR = waist-to-hip ratio; FFM = fat-free mass; SBP = systolic blood pressure; DBP = diastolic blood pressure; TC = total cholesterol; LDL = low density lipoprotein; HDL = high-density lipoprotein; $\dot{V}O_2$ = oxygen uptake; CHO = carbohydrate; n.a = not applicable

1.6 Structure of the thesis

Four chapters will follow the current introduction (Figure 1.1). They comprise a review of the applicable current literature (Chapter 2), a chapter on testing procedures and the intervention strategy (Chapter 3), one containing the results and discussion of results (Chapter 4), and a summary of the main findings (Chapter 5). Each chapter will include its own list of references. Referencing will adhere to the regulations and conventions of the Department of Exercise and Sport Sciences at the University of the Free State, which uses the Harvard referencing method (Van der Walt, 2006:1).

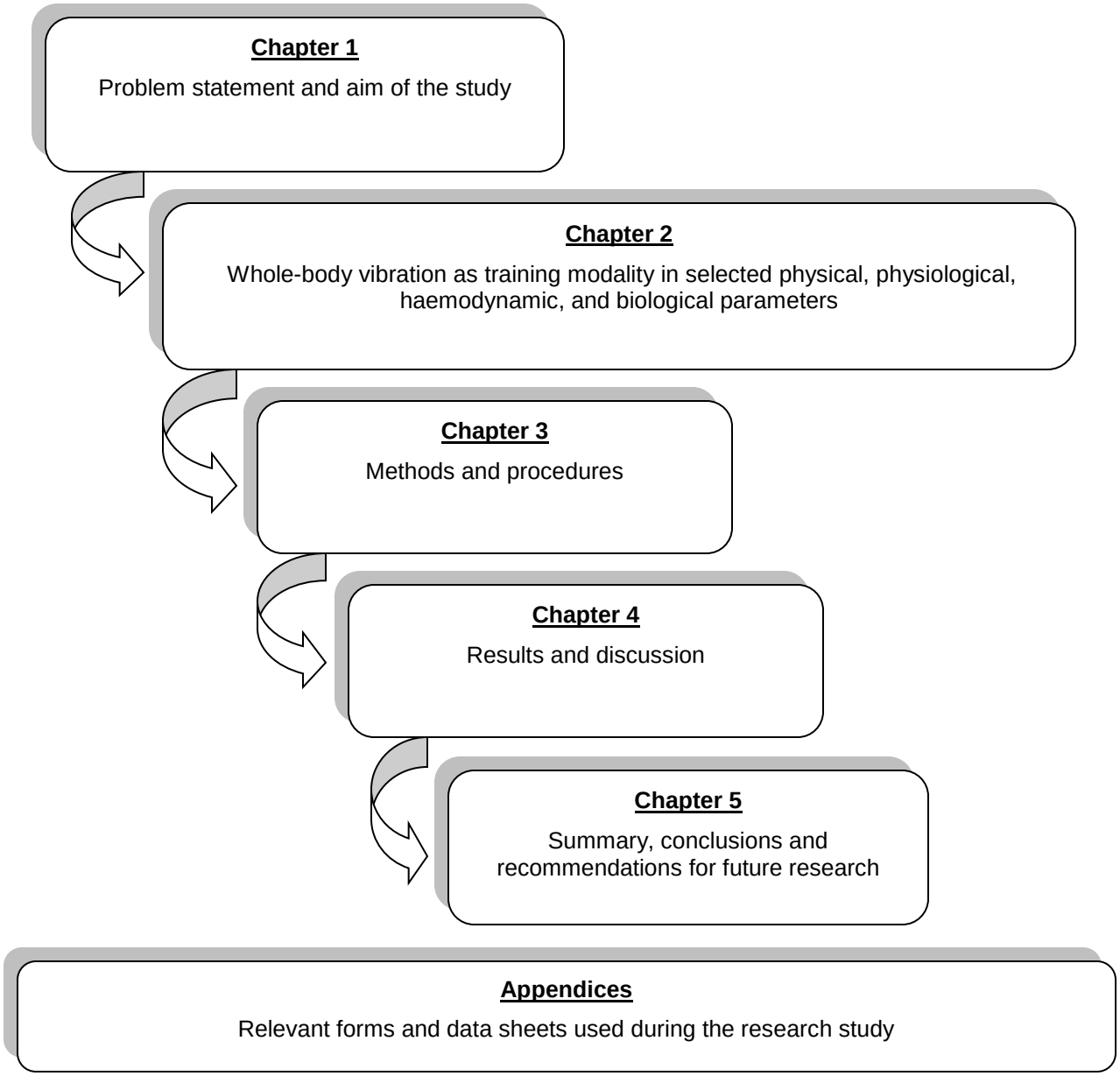

Figure 1.1: Structure of the thesis

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2

Whole body vibration as training modality in selected physical, physiological, haemodynamic, and biochemical parameters

2.1 Introduction

Regular physical activity forms an integral part of preventative health and plays a major role in both the prevention and treatment of diseased populations (ACSM, 2010:7). Sedentary behaviour in the Western world is escalating annually, resulting in increased numbers of all-cause mortality, cardiovascular and coronary heart disease, hypertension, obesity, diabetes mellitus, cancer and depression (Blair, 2009:1). Reasons for insufficient daily physical activity include lack of time, boredom with exercise routines, poor visible results, and confusion as to which exercises are suitable for your age and physical condition (Wahener, 2010). For such individuals, whole-body vibration as mode of exercise seems an attractive option as it is propagated to be quick, interesting, safe and effective (Signorile, 2008:20).

Even though whole-body vibration training escalates in popularity, much seems still unknown by scientists. Currently, literature supports whole-body vibration exercise and training to be beneficial with regards to strength and power (Delecluse *et al.,* 2003:1039; Becerra Motta & Becker, 2001:33; Bosco *et al.*, 1999:309), flexibility (Cochrane & Stannard, 2005:860), metabolic rate and energy expenditure (Zange *et al*., 2008:268; Van den Tillaar, 2006:192; Rittweger *et al.*, 2001:169) bone mineral density (Verschueren *et al.*, 2004:352; Rittweger *et al.,* 1999:134), posture (Polonyova & Hlavacka, 2001:408) and skin and muscle perfusion (Lohman *et al.,* 2007:71).

By contrast, evidence exists that WBV could be harmful to peripheral nerves, blood vessels, joints and perceptual function (Griffin, 1990:174). In severe cases it could cause chest pain and internal bleeding as well as disorders of menstruation and abnormal child birth (Gratsianskaya *et al.*, 1974:38). Contact areas, transmission of the vibration, and whether the vibratory stimulus was applied actively or passively has not been reported sufficiently. Also, the transmission of pressure and touch via the autonomic nervous system is masked during WBV (Ribot-Ciscar *et al.*, 1989:130).

Disparities in testing protocol composition and exercise program design as well as inconsistencies in the use of terminology are attributed to opposing and inconclusive research results (Lorenzo *et al*., 2009:676).

The human body is a complex system with many structures and functions. Interference in certain bodily structures and functions may either improve health or increase the risk of an individual developing chronic disease. For example, interference such as exercise training in sedentary populations may improve resting metabolism and consequently influence body composition beneficially (Horowitz & Klein, 2000:5585). On the contrary, detrimental interference such as chronic cigarette smoking may cause cancer (Joshu *et al.*, 2011:835).

The ACSM (2010:28) identified 8 risk factors that help predict an individual's risk of developing chronic disease. Of those 8 factors, 5 can be improved by regular physical activity. They are a sedentary lifestyle, obesity, hypertension, dyslipidaemia and prediabetes. These 5 factors have been selected for investigation and discussion as they have not been explored extensively in the area of whole-body vibration. Additionally, metabolism and ventricular function are explored as new areas of investigation. Variables discussed in this chapter therefore include body composition, blood pressure, blood lipids, blood glucose, metabolism and cardiac function with specific reference to left ventricular diastolic function.

In order to obtain an understanding of the unique type of exercise that whole-body vibration is, mechanical effects of WBV on the human body in general will be discussed. Published literature on the selected variables in the area of WBV will then follow. Relevant research was scarce and is supplemented by studies on conventional exercise training so as to evaluate the position of WBVT in this specific milieu.

2.2 Types of vibration

Vibration is an oscillatory motion that occurs in a variety of forms and is not necessarily constant (Mester *et al.*, 1999:211). Whole-body vibration occurs in a number of instances in the human environment, e.g. motorized vehicles, marine ships, aircrafts, buildings, and industrial equipment. The characteristics of vibrations differ considerably between sources, as does the sensation of an individual in the event of exposure (Jordan *et al.*, 2005:460).

In order to understand the phenomenon of vibration it is necessary to know the different types of oscillatory motion that are generally encountered. Categories of vibration and different wave forms are set out in Figure 2.1 and Table 2.1. Vibration platforms, for exercise purposes, are designed to produce sinusoidal vibrations as these types of vibration are controllable and adjustable. Sinusoidal vibrations are oscillatory, deterministic and periodic, as classified in Figure 2.1. A graphic example of sinusoidal vibration is presented in Table 2.1 (the first wave form).

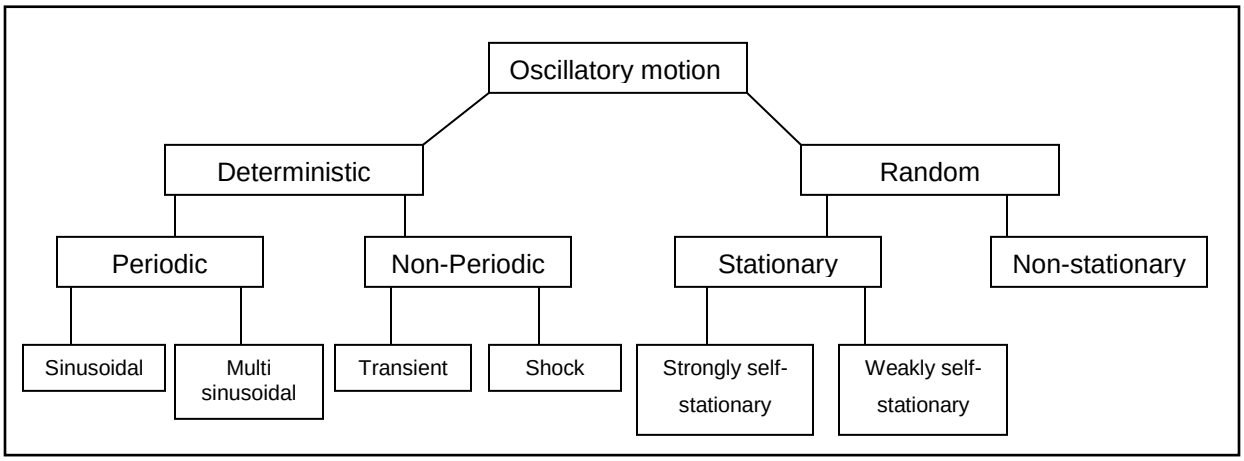
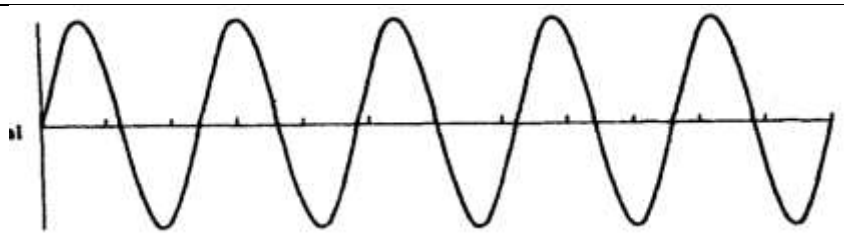
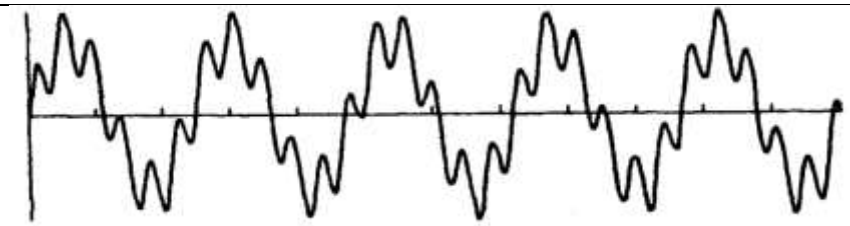
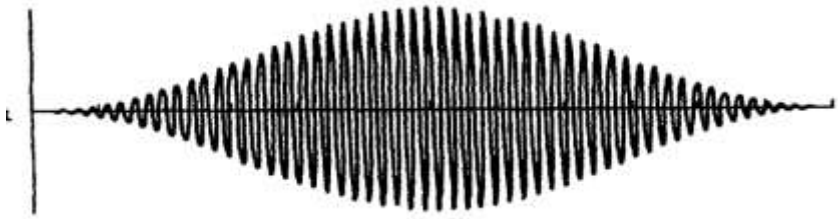
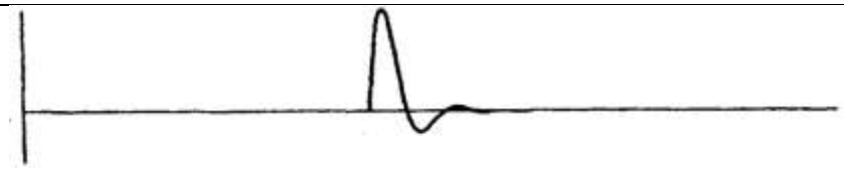
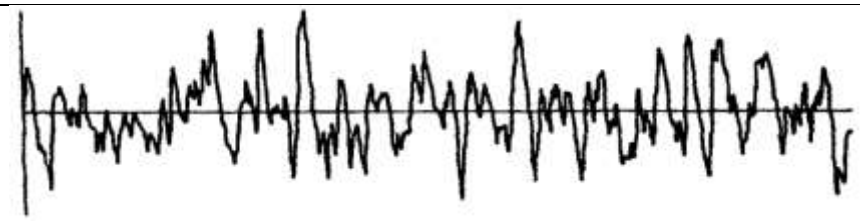
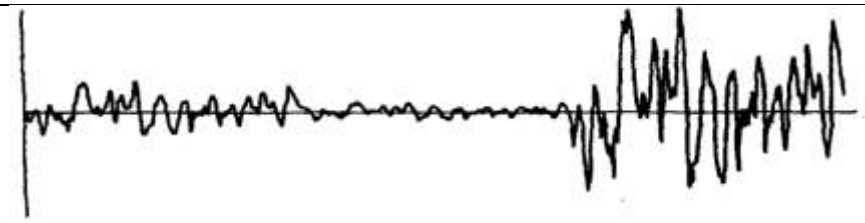


Figure 2.1: Categorization of types of oscillatory motion (Griffin, 1990:4)

Table 2.1: Different oscillatory vibration waveforms (Griffin, 1990:6)

Type	Wave form
Sinusoidal	
Multi-sinusoidal	
Transient	
Shock	
Stationary random	
Non-stationary random	

2.3 Development of vibration into a training modality

The use of single-directional vibration with the aim of improving human performance dates back as far as ancient Greece. According to Van der Meer *et al.* (2007:16) saws were covered in cotton and used to transfer vibrations to certain parts of the body that needed therapy. Only in the 19th century multi-directional vibration has been introduced. Physicians used sagital and circular movements to treat atrophy, constipation and neuralgia (Van der Meer *et al.,* 2007:16), whereas Nazarov and Spivak (1985:445) applied vibration as a training modality on athletes.

By the end of the 19th century Gustav Zander became popular with his clever conversion of steam-powered vibratory devices into massage machines. It was, however, Professor Biermann who studied "cycloid vibrations" and the "Rhythmic Neuromuscular Stimulator" who founded the principles of acceleration training as we know them today. His methods were first researched and applied to astronauts in the Soviet Union during the 1960's (Biermann, 1960:219).

Previously, astronauts suffered from severe loss of strength and bone-mineral density during space flights. It was demonstrated that, through the use of acceleration training, these men could withstand the negative effects of microgravity (Van Loon *et al.*, 1996:297). Later, Nazarov as well as the Israeli scientist Issurin broadened the research on vibration during the 1960's and 1970's as they studied these effects on athletes. The studies yielded favourable results on the areas of muscular strength and power, flexibility, bone density and blood circulation (Hinman, 2010).

After some delay the Western world was introduced to this breakthrough in exercise technology (Van der Meer *et. al.*, 2007:17; Rittweger *et al*., 2001:169; Kerschan-Schindl *et al.*, 2001:377; Bosco *et al.*, 1989:157) and from here on contributed to the scientific development of vibration as a training modality as we know it today.

2.4 Terminology dilemma

During the development of vibration as a training modality, inconsistencies in the use of terminology have surfaced. In the past researchers have used the term "Whole-body Vibration (WBV)" to refer to (a) Acceleration training (Cardinale & Bosco, 2003:5) (b) Whole-body vibration in passive mode (Maikala & Bhambhani, 2008:775), and (c) Whole-body vibration in active (isometric) and/or dynamic (moving) mode (Bogaerts *et al.*, 2007:634; Roelants *et al.*, 2004:3).

The same tendency is prevalent when reporting the magnitude of vibration. Inconsistencies exist regarding frequency, amplitude, displacement, peak-to-peak displacement, acceleration and gravitational acceleration of the WBV platform (Lorenzo *et al.,* 2009:676). This creates confusion when reproducing research methods and results. The abovementioned terms are clarified in Table 2.2.

Table 2.2: Definitions of terminology describing magnitude of vibration

Term	Definition	Author
Acceleration (*a*)	The rate of change (or derivative with respect to time) of velocity, a vector quantity with dimension length.time^{-2}.	Van der Meer *et al.* (2007:163)
Amplitude (*A*)	The nonnegative scalar measure of a wave's magnitude of oscillation, that is, magnitude of the maximum disturbance in the medium during one wave cycle.	Van der Meer *et al.* (2007:163)
Frequency (*f*)	Measurement of how often a recurring event such as a wave occurs in a measured amount of time. One completion of the repeating pattern is called a cycle.	Van der Meer *et al.* (2007:171)
G-Force (Gravitational acceleration) (*g*)	The nominal acceleration due to gravity at sea level on the earth's surface, also known as standard gravity.	Van der Meer *et al.* (2007:171)
Peak-to-peak displacement (p-p)	The total vibration excursion of a point between its positive and negative extremes.	Lorenzo *et al.* (2009:677)

Contact areas of the subject with the vibration plate have also been poorly reported. Contact areas include upper extremities, lower extremities (Otsuki *et al.*, 2008:189) and/or the trunk (Maikala & Bhambani, 2008:775). Lorenzo *et al.* (2009:677) suggest that "frequency, maximal acceleration and peak-to-peak displacement at a clearly defined location of the platform should be the minimum parameters reported by all studies". These parameters will be subsequently reported according to the definitions in Table 2.2.

2.5 Vibration axes

A system for expressing the magnitudes of vibration occurring in different directions have been defined by the International Organization for Standardization (ISO): International Standard 2631-5 (2004). The notation used to refer to axes of vibration with respect to the body is outlined in Table 2.3.

Table 2.3: Notation used to refer to axes of vibration with respect to the body (Griffin, 1990:35)

Notation	Description	Other terms in use	For normally seated persons
Translation vibration			
x-axis	Back to front	Fore-and-after Surge Shunt (Transverse) (Longitudinal)	Fore-and-after
y-axis	Right to left	Side-to-side Sway (Transverse) (Shoulder to shoulder) (Left-to-right)	Lateral
z-axis	Foot to head	Heave (Longitudinal) (Vertical) (Up-and-down)	Vertical
Rotational vibration			
r_x	Rotation about *x-axis*	Roll	Rotational motion always produces some translation at all points other than the centre of rotation. In a rigid body the rotation is the same at all points.
r_y	Rotation about *y-axis*	Pitch	
r_z	Rotation about *z-axis*	Yaw	

2.6 Physical principles

2.6.1 G-force and acceleration

The force of attraction between masses in the universe and the earth, called *gravity,* causes nominal acceleration on an object, called *g-force*. Acceleration is proportional to the force applied and is expressed as $m.s^{-2}$ or as multiples of terrestrial gravitation in *g* (where $1g=9.81\ m.s^{-2}$), (Cochrane, 2011:80). The human body is adapted to the gravitational field of the earth, which equals one g-force. One such adaptation in the body is its ability to oppose acceleration through skeletal muscle strength, keeping the body in a desired functional position (Van der Meer *et al.,* 2007:24).

Newton's second law states the definition of force:

Force = mass * acceleration F = m*a

Instead of increasing mass, vibration exercise has the unique ability of producing force because of acceleration. This implies that, when on a vibration platform, force can be increased during the execution of a movement without the adding of extra load (Van der Meer *et al.,* 2007:24).

A few studies have investigated acceleration by means of accelerometers that directly measure acceleration of the vibration platforms (Di Giminiani *et al.*, 2009:169; Lythgo *et al.*, 2009:53; Pel *et al*., 2009:937; Bazett-Jones *et al.*, 2008:144; Roelants *et al.,* 2004:1). Other researchers have measured acceleration transmission at various joints by placing accelerometers on body landmarks (Abercromby *et al.*, 2007:1794; Crewther *et al.*, 2004:37). Greater acceleration was observed proximal to the vibration platform compared to distal locations.

2.6.2 The actuator: Vibration exercise device

The actuator is the mechanical device transmitting vibration to the object in contact with the device. In vibration-training studies the actuator is the vibration platform.

Vibration training is mostly practised whilst standing on an oscillating platform. Among the different platforms, two distinctly different types can be distinguished. One type operates in a side-alternating way so that one foot is highest when the other is lowest, and vice versa. The other type transfers vibration to both feet synchronously. Rittweger *et al*. (2001:169) argues that side-alternating oscillation induces rotational forces around the hip and lumbo-sacral joints. Abercromby *et al.* (2007:1798) adds that, compared to synchronous WBV, additional degrees of freedom allow for smaller whole-body mechanical impedance.

A third type of vibration exercise device inducing random horizontal and vertical planes has been attempted, but only tested at frequencies well below 10 Hz due to technical difficulties. Furthermore, vibrating dumbbells have been developed for upper-body exercise where the physiological responses closely resemble those of platform devices if controlled for force, velocity, and energy (Cardinale & Rittweger, 2006:12).

Vibration devices also differ in terms of energy generation. Some operate by mechanical transmission (e.g. Gelileo®), some by electromagnetic transmission, and most operate by oscillating mass-spring systems (e.g. Power-Plate® or FitVibe®). In this particular study the oscillating mass-spring system (Power-Plate®) was applicable.

2.6.3 The resonator: Body stiffness, posture and vibration absorption

The resonator is the recipient of the vibration caused by the actuator. In vibration training, the individual performing exercises on the vibration platform is the resonator.

Assuming a rigid body is attached to a vibration device, the rigid body will follow the sinusoidal trajectory imposed by the actuator. The force applied is then determined by the acceleration of the mass of the body. On WBV-platforms, however, there is no firm attachment and the only downward force acting on the body is gravity (Yue & Mester,

2002:639). Consequently, a rigid body would lose contact with the platform and become air-bound when the acceleration of the platform is smaller than -1*g*. As the plate tilts to the counter side, collision with the platform will occur towards the end of the air-borne phase, which leads to the generation of impact forces (Rittweger, 2010:879).

Muscles and tendons, on the other hand, act as spring-like elements that store and release mechanical energy. During the vibration up stroke and down stroke, compression and expansion occur respectively. Consequently displacement in the body's centre of mass is smaller than at the platform level. Furthermore, mass-spring resonator systems can accumulate mechanical energy that can lead to a situation where the vibration amplitude in the resonator is greater than in the actuator. The amplification of vibrations increases internal forces which could lead to the body's destruction. This phenomenon is termed the "resonance catastrophe" and can only occur when the generated forces exceed the resonator's structural strength.

Resonance can occur within the trunk at vibration frequencies around 5 Hz (Pope *et al.*, 1990:135) and in the lower extremities below 20 Hz (Rubin *et al.*, 2003:2621). It seems therefore safe to train at vibration frequencies higher than 20 Hz, provided that the contact area is in the lower extremities. Applied vibration higher than 20 Hz will exceed the resonance of the body's organs and consequently reduce injury risk. No studies with regard to transmissions of vibration with contact areas in the upper body could be found.

Wakeling *et al.* (2002:1098) found that muscles have damping properties which will lead to the absorption of energy and thus generate heat. Furthermore, in the human body, vibration will be transmitted from one segment to the next, i.e. from the foot to the lower leg, from the lower leg to the thigh and so forth. Musculo-skeletal stiffness and damping determines the amount of vibration energy transmitted. Axial body stiffness increases with straightened limbs and may result in higher resonance frequencies (Lafortune *et al.*, 1996:1535). Appropriate posture is therefore the prerequisite for avoiding dangerous vibration transmission to the trunk and head.

2.7 Musculo-skeletal and neuro-physiological responses to vibration

Vibration causes rapid cyclic transition between eccentric and concentric muscle contractions (Cochrane *et al.*, 2009:420). Applying vibration to the muscle belly or tendon elicits a phase-oriented discharge from primary (Roll & Vedel, 1982:177; Burke *et al.*, 1976:695) and secondary spindle endings (Burke *et al*., 1976:695; McGrath & Matthews, 1973:371) with primary endings being more responsive than secondary endings (Brown *et al.*, 1967:773). Spindle discharge depends on the pre-stretch of the muscle and increases with muscle length or stretch, as well as isometric voluntary contraction (Burke *et al*., 1976:697).

Golgi tendon organs, like the muscle spindles, become more responsive to vibration when the muscle is contracting or elongating, and is thus a surrogate of force (Brown *et al.*, 1967:778). It inhibits motor output via polysynaptic spinal pathways and its information is converged with that from cutaneous receptors, spindle afferents, joint receptors and others (Lundberg *et al.*, 1975:83).

Passive muscle vibration causes a reflex contraction, also known as the tonic vibration reflex (Matthews, 1966:204). The reflex is characterized by its gradual onset and can be voluntary suppressed, suggesting supra-spinal control (Burke *et al*., 1976:695). During active knee extension, vibration applied to the patella tendon lead to enhanced co-contraction of the hamstring musculature (Rothmuller & Cafarelli, 1995:857), suggesting that the centrally mediated co-contraction command supersedes spinal reciprocal inhibition of the antagonist. Inconsistent results have been reported for the stretch reflex, with some authors reporting enhancement (Shinohara *et al.*, 2005:1835) and others depression (Roll *et al.*, 1980:1227) of the Hoffmann reflex after termination of single-muscle vibration.

Enhancement of stretch reflexes (Jendrassik maneuver) (Melnyk *et al.,* 2008:842) and increased Hoffmann reflex (H-reflex) responses (Nishihira et al., 2002:877) during WBV have been reported. Unfortunately, only a small number of studies are available on this

topic and the exercise parameters (such as exposure time and loads) are quite heterogeneous.

2.8 Parameters of vibration exercise

2.8.1 Frequency

There is little scientific documentation suggesting appropriate vibration frequency (*f*) for exercise prescription. The majority of researchers investigated vibration frequency using the effectiveness of muscle activation and power and not cardiovascular efficiency as such (Mester *et al.,* 2006:1056). Keeping in mind that cardiovascular efficiency depends greatly upon the effectiveness of muscle activation, discussion thereof seems appropriate.

Upon the inception of commercialized vibration platforms, Nazarov and Spivak (1985:447) studied frequency of 23 Hz WBV on athletes. The reason for the specific frequency was that they presumed higher frequencies would disappear in the tissue during transmission from the vibration device to the musculature. Later, Bosco *et al.* (1989:157) exposed handball and water polo athletes to intermittent WBV of 26 Hz and found increased vertical jump height of 12%. No rationale was given as to why 26 Hz was selected.

In 2003, Cardinale and Lim (2003:621) introduced surface electromyography (EMG) to validate vibration frequency. They found that when standing in a half-squat position (knee angle 100˚) EMG response of the vastus lateralis muscle, was significantly higher in acute 60 s vibration sessions of 30 Hz, 10 mm peak-to-peak (p-p) amplitude, compared to 40 and 50 Hz. Likewise, Delecluse *et al.* (2003:1033) found increased EMG-activity in the rectus femoris and medial gastrocnemius muscles in the same half-squat position (*f*=35 Hz, *A*=5 mm, *g*=3.9) compared to the placebo condition (*a*=3.9 *g*).

Recently, dose-response relationships between different vibration frequencies and muscle performance (vertical jump height) were investigated. Da Silva *et al.* (2006:267) found 30 Hz to be more effective in generating muscle power in a male population than

20 Hz, whereas 40 Hz decreased both muscle power and strength. Bazett-Jones *et al.* (2008:144) found 30 Hz and 50 Hz to improve muscle power in women ($p<0.01$), but not in men, suggesting gender should be considered during training. In addition, Di Giminiani *et al.* (2009:169) found surprisingly beneficial results when vibration frequencies were individualized according to surface electromyographic (EMG) properties of the participants. Increases of 11% and 18% were found in the squat jump and jump height activities respectively after eight weeks of individualized WBVT three times per week.

From the results it seems best to individualize WBVT-prescriptions according to each person's ability. Whether surface elctromyography is the most appropriate measure of WBV ability, is yet to be established. Furthermore, the equipment is expensive and may not be available to all practitioners in exercise settings.

2.8.2 Amplitude

In platforms that produce side-alternating vertical sinusoidal vibration, the body experiences rotation around an antero-posterior horizontal axis. So when feet are further from the axis, this results in larger vibration amplitude. Other platforms producing synchronous vibration, such as the Power-Plate®, vibration is distributed simultaneously and symmetrically to both sides of the body, regardless of foot-placement (Cochrane, 2011:76).

At a fixed vibration frequency, Cardinale *et al.* (2006:380) found no difference in testosterone or insulin growth factor 1 (IGF-1) when participants were exposed to amplitudes of 3 mm, 1.5 mm and 0 mm. The type of vibration platform was, however not specified. In a study using a sinusoidal vibration platform, fixed frequency of 26 Hz and three different levels of amplitude (2.5 mm, 5 mm, and 7.5 mm) were induced on participants standing at a 10˚ knee-flexion angle. Oxygen cost increased proportionately at all three levels where A=7.5 mm induced the largest significant difference (Rittweger *et al.*, 2002a:428).

On a synchronous vibration platform (Power-Plate®) Lythgo *et al.* (2009:53) found a 27% increase in mean blood-cell velocity when frequency was progressively increased from 5 to 30 Hz and amplitude ranging from 2.5 mm to 4.5 mm. On the same platform, Adams *et al.* (2009:237) found a significant increase in vertical jump power after acute vibration at *f*=50 Hz and *A*=4-6 mm, compared to *f*=30 Hz and *A*=2-4 mm.

Direct comparisons between sinusoidal and synchronous vibration platforms have been vaguely researched. Pel *et al.* (2009:937) reported no change in amplitude when sinusoidal and synchronous vibration platforms were loaded with two different body masses. It was, however, noted that acceleration (*g*) was reduced when frequency (*f*) was increased from 30 to 40 Hz on the sinusoidal vibration platform. This evidence might suggest caution when WBVT programs are prescribed for heavier individuals. A single study, however, is insufficient to critically assess the effect of body mass on frequency.

Specific details such as how amplitude was calculated – whether by mathematical equation, accelerometer or other method – is lacking in most studies. Moreover, terminology is inconsistent. The terms amplitude, peak-to-peak amplitude, and displacement are not distinguished in any detail.

2.8.3 Duration

Duration refers to the exposure time to vibration. Currently, little scientific evidence is available on the optimal duration for intermittent and/or continuous sessions. Even so, Adams *et al*. (2009:237) found no significant difference in vertical jump power after acute vibrations of 30 s, 45 s, or 60 s (*f*=30-50 Hz, *A*=2-4, and 4-6 mm) synchronous vibration platform in an untrained population. In comparison to decrements in peak torque after 4-6 min of continuous vibration, Stewart *et al.* (2009:50) found increased peak torque of 3.8% after 2 min continuous vibration (*f*=26 Hz *A*=4 mm) in participants standing on a synchronous vibration platform (5˚ knee-flexion angle).

In long-term studies covering 6 weeks to 8 months, various vibration exposure times have been reported (Abercromby *et al.,* 2007:1794; Cardinale *et al*., 2006:380). It seems that intermittent protocols may be preferred over continuous ones as vibration exposure of more than 1 minute at a time are likely either to involve lower levels of acceleration (with positive strength and power adaptations), or greater injury risk (if high duration and accelerations are combined). In intermittent protocols, muscle may be stimulated and fatigue limited. More research is needed to clarify optimal duration for performance enhancement and health benefits.

2.9 Safety considerations

The International Organization for Standardization (ISO) 2631-5 (2004) has defined limits of vibration tolerance in industrial exposure for three categories, namely: comfort, performance proficiency, and safety. Each category's safety is determined by a combination of frequency, direction of vibration and time of exposure.

International Organization for Standardization 2631-5 standards have been based on data obtained from aircraft pilots and drivers (ISO, 2004) with contact areas being their gluteal area. In this situation the detrimental effect concerns are focused on the vertebral column and internal organ tolerance to vibration. As previously discussed, legs help to reduce vibration transmission to the trunk and are much more tolerant of vibration than the trunk and head. This may largely be due to the spine, which consists of bony parts transmitting vibration easier and absorbing vibration to a lesser extent than muscle and other soft tissues. Safety may be underestimated for certain groups of patients, such as those suffering from acute trauma; whereas it may be overestimated for patients suffering from chronic mechanical lower-back pain as evidence suggests that WBV relieves this condition (Rittweger *et al.*, 2002b:1829).

Detrimental incidents during WBVT are uncommon. However, side-effects such as hot feet, lower-leg erythema, vertigo and hip discomfort have been reported in untrained participants when exposed to acute vibration frequencies (10, 20, 30 Hz) with amplitudes 1.25, 3, and 5.25 mm (Crewther *et al.,* 2004:41). Likewise, Cronin *et al.*

(2004:74) found participants complaining of jaw, neck and lower-limb pain (f=26 Hz; A=6 mm) which subsided after 7-10 days of physiotherapy treatment.

In addition, certain sports such as skiing commonly exposes participants to vibrations well above the ISO2631-1 (Spitzenpfeil & Mester, 1997:209). It seems therefore that safety standards should be modified for sports and exercise rehabilitation environments.

2.10 General exercise-mediated effects on the human body

In Western societies the statistical average of physical activity is believed to be far below the genetic background. The tendency of sedentary lifestyle in combination with excess food intake has surpassed smoking as the number one preventable cause of death in the United States (Blair, 2009:1; Mokdad *et al*., 2004:356). Gielen *et al.* (2010:1221) speculate that, in comparison to ancestral survival activity level, most of the molecular changes during exercise are probably only a return to normal values and not necessarily improvement.

They have summarized the exercise-mediated effects on the human body (Figure 2.2) (Gielen *et al*., 2010:1222). Types of training and frequencies, intensities and time of sessions are not specified in the summary, yet the table gives a valuable nutshell overview of exercise-mediated effects on the human body. Compared to aerobic and resistance training, whole-body vibration is a vaguely explored area in the milieu of exercise training. The effect of WBVT on the human body, whether it may be beneficial or detrimental, is inconclusive for many bodily systems including the cardiovascular, neuro-hormonal, and autonomic nervous systems. Research in this area is especially lacking at molecular level, making it difficult to explain possible mechanisms of findings. The summary by Gilien *et al.* (2010:1222) could therefore be useful as a means of establishing the place of WBVT in the milieu of exercise and exercise training.

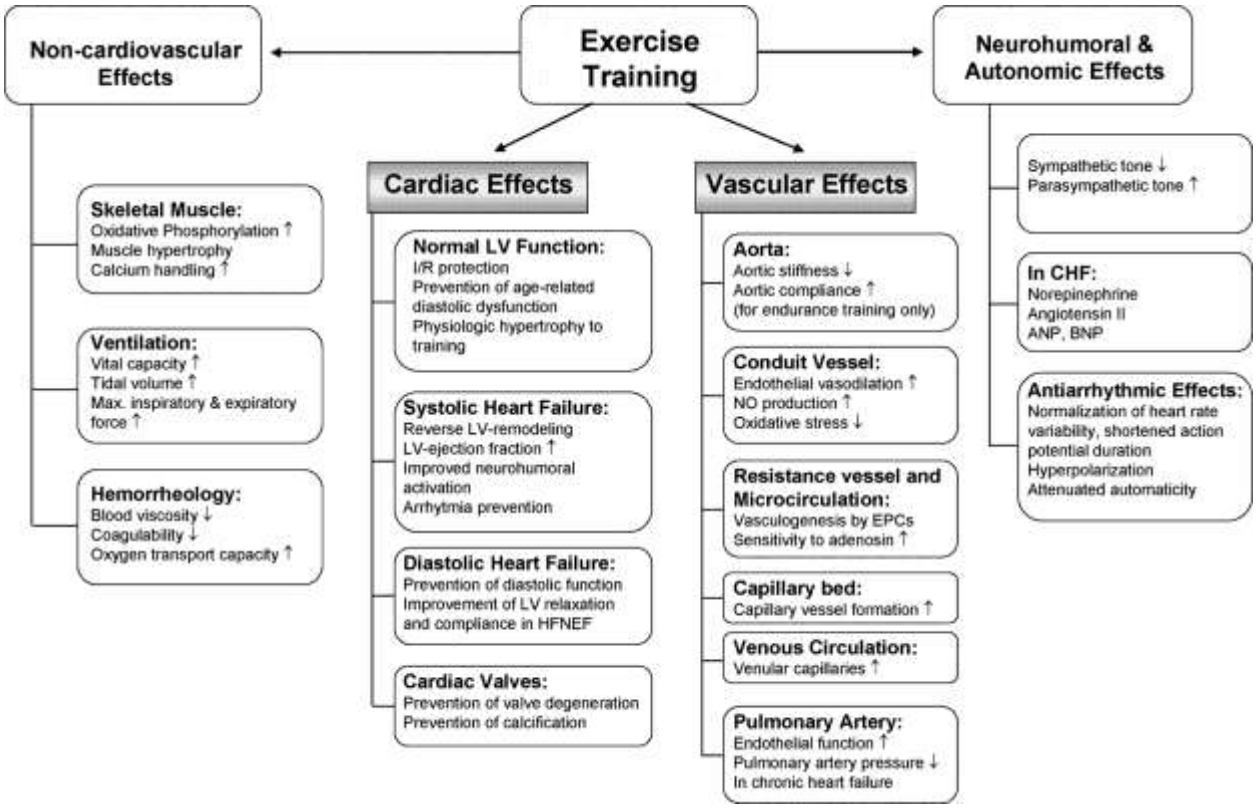


Figure 2.2: Summary of exercise-mediated effects on the human body (Adapted from Gielen *et al.*, 2010:1222)

LV = Left ventricle; NO = nitro-oxide; EPC = endothelial progenitor cells; CHF = congestive heart failure; Max. = maximum; HFNEF = heart failure with normal ejection fraction; ANP = arterial natriuretic peptide; BNP = brain natriuretic peptide; I/R = Ischemia/Reperfusion

2.11 Effects of WBVT on the human body

2.11.1 Body Composition

Body composition implies different components in the body that make up a person's body weight. The human body is composed of different tissue types, including lean tissues (muscle, bone, and organs) that are metabolically active and fat (adipose) tissue that is metabolically inactive (Quinn, 2009).

The term obesity, on the other hand, describes individuals that are excessively overweight and as a result thereof are at higher risk for premature death and the development of chronic diseases such as heart disease (Shaper *et al.*, 1997:1311), diabetes (Colditz *et al.*, 1990:501), cancer (Giovannucci *et al.*, 1995:332), hyperlipidaemia (Hershcopf *et al.*, 1982:112), and hypertension (Flegal *et al.*, 1998:44).

To estimate disease risk, obesity can be determined by calculating one or more risk indicators including body mass index (BMI), percentage body fat (%BF), and waist circumference or waist-to-hip ratio (WHR). WHR is different to %BF and BMI in that it is rather a descriptor of fat distribution than a value of body mass or total body fatness. Men aged 18-40 years are considered overweight or at increased disease risk when meeting the criteria set out in Table 2.4. Fat free mass (FFM) is an additional value indicating the metabolically active tissue of a person. FFM is not used to determine cardiovascular risk, but assists in evaluating the effectiveness of certain lifestyle intervention programs such as weight training (Hunter *et al.,* 2008:1045).

Energy balance in the body is reached when energy input and output are equal. In order to gain or lose body weight, each side of the equation needs to be adjusted. Exercise helps to increase energy output and consequently plays a critical role in weight control (Marra *et al*., 2005:39).

Table 2.4: Disease risk based on the classification of anthropometric measurements for obesity in 18-40 year old males (ACSM, 2010:63-71)

Method	Value for onset of disease risk
Body Mass Index (BMI)	≥ 25.0 $kg.m^{-2}$
Percentage body fat (%BF)	≥ 20%
Waist circumference (WC)	≥ 102 cm
Waist-to-hip ratio (WHR)	≥ 0.95

Evidence suggests that aerobic exercise training lowers %BF (Jakčić *et al.*, 2003:1323; Van Aggle-Leijssen *et al.*, 2002:1307) as well as body weight, body-mass index, and waist circumference (Slentz *et al.*, 2004:31). There is, however, controversy over the ideal intensity of exercise as both low intensity as well as high intensity aerobic conditioning has proved to be successful (Jakčić *et al.,* 2003:1323). Aerobic training does, however, not seem to influence fat-free mass (Hunter *et al.*, 2008:1045). Resistance training, on the other hand, conserves (Hunter *et al.*, 2008:1045) or increases (Adams *et al.,* 2003:1613; Campbell *et al.*, 1994:167) fat-free mass, whether performed in isometric or dynamic fashion. It also decreases waist circumference (Tsuzuku *et al.*, 2007:549), and fat mass (Campbell *et al.,* 1994:167).

The ACSM (2010:254) suggests a combined aerobic and resistance training regime for overweight and obese individuals to improve their body composition and reduce cardiovascular risk. Intensity for aerobic training should start low (40-60% HRR) and increase over time to higher intensity activities (50-75% HRR). Exercise duration should be 30-60 min. per day to a total of 150 min. per week, progressing to 300 min. and exercise frequency 5-7 days per week. Resistance training should involve large muscle groups and be executed 2-3 times per week with a volume of 8-10 exercises, 2-3 sets per exercise and 15-25 repetitions per set. Loads should be low (50% 1RM) and at least one day of rest between resistance training sessions should be incorporated (ACSM, 2010:170).

In studies investigating the effect of WBVT on body composition, certain essential variables such as frequency, intensity, time, and population type have not been reported sufficiently. It is therefore difficult to generalize findings and predict results in different populations.

Bonner Physical Therapy (2003) investigated the BMI of six subjects. BMI improved by 10.6% after six weeks of WBVT intervention. Roelants *et al*. (2004:1) found no significant changes in weight, %BF or skinfold thickness in untrained females after 24-weeks of WBVT. However, a significant increase in FFM (2.2%; $p \leq 0.05$) was reported, which could be due to the strength-training properties of WBVT. Higher quantities of FFM consequently increase the metabolically active tissue, which increases metabolic rate and consequently energy expenditure. It seems, however, as though the increase in FFM was not sufficient enough to beneficially change body weight, skinfold thickness, and %BF. Fjeldstad *et al.* (2009:79) reported a significant decrease ($p \leq 0.05$) in %BF and significant increase in bone-free FFM in postmenopausal women after 8 months of combined resistance and vibration training (f=30-50 Hz, A=3 mm, time=15-60 s).

Bogaerts *et al.* (2007:630) investigated older men (≥60 years) on a 12-month WBVT program. Muscle mass increased significantly ($p \leq 0.01$) with 3.4%. No other body composition variables were investigated. By contrast, Verscheuren *et al.* (2004:352) found no change in lean body mass subsequent to vibration training, but reported a 2% decrease in %BF in older adults. Again, the possible mechanism responsible for the findings might be the increase in metabolically active tissue. Muscle tissue is metabolically more active than adipose tissue and is affected by exercise training, especially resistance training. Unlike adipose cells, muscle cells have the capacity to produce ATP that provides energy during activity (Seeley *et al.,* 2011:949). Consequently, individuals with well-developed muscles have higher basal metabolic rates than unconditioned individuals. Given that basal metabolic rate accounts for about 60% of daily energy expenditure, individuals with higher metabolic rates will use more energy during rest in comparison to unconditioned individuals. In this way the energy balance is negatively affected and body weight is reduced. During rest, fat is

utilized as primary energy source, explaining the decrease in %BF as a result of possibly higher basal metabolic rates. The increase in muscle mass and the reduction in %BF that was recorded by Bogaerts *et al.* (2007:630) and Verscheuren *et al.* (2004:352) are comparable to the effects of resistance training. WBVT as a resistance training modality is thus stressed.

The above findings suggest that WBVT could be an alternative treatment modality for those suffering from obesity as FFM may increase and %BF may decrease over a period of six or more weeks.

Additionally, Power-plate International (2008) advises against the use of vibration platforms for patients who are more than 10 kg over-weight (method for determining excess weight has not been given). The advice to abstain from WBVT is given most likely due to the increase in injury risk, but also due to the harm that might be caused in overweight individuals that consequently possess higher cardiovascular disease risk. However, Mester *et al.* (2006:1063) suggest that transmissibility of wobbly mass is abolished at frequencies higher than 15 Hz and consider training at frequencies higher than 20 Hz to be safe. No studies could be found reporting injury of overweight subjects due to WBVT.

2.11.2 Cardiovascular function

The cardiovascular system is a closed unit comprising the cardiac and circulatory systems. The cardiac system refers to the structure of the heart (endocardium, myocardium, and pericardium) and its function during systole and diastole, where the circulatory system refers to blood vessels (arteries, arterioles, capillaries, venules, and veins) and their function as a transport aid for oxygen, nutrients and waste products (Brooks *et al*., 2004:313). Control of the system is mainly directed toward maintaining blood pressure.

2.11.2.1 Blood pressure

Blood pressure as measured in the brachial artery is considered optimal when systolic pressure (SBP) is lower than 120 mmHg and diastolic blood pressure (DBP) is lower than 80 mmHg (ACSM, 2010:49). Blood pressure can be elevated by numerous factors such as excessive sodium and alcohol intake, and lack of physical activity, but is mostly idiopathic. Values of ≥140/≥90 mmHg at rest are considered hypertensive and increase an individual's risk for developing cardiovascular disease (ACSM, 2010:47). Lifestyle modification such as diet adjustments and increased physical activity is considered as cornerstone treatment for hypertension (ACSM, 2010:48). Evidence supporting the positive effect of regular exercise on blood pressure reduction is well documented and proves its valuable contribution as part of lifestyle modification (Hagberg *et al.*, 2000:193; Paffenbarger *et al.*, 1991:319).

Dynamic aerobic training reduces resting blood pressure in individuals with normal blood pressure and in those with hypertension (Whelton *et al.*, 2002:493; Fagard, 2001:491). The decrease in blood pressure appears to be more pronounced in hypertensive than in normotensive subjects (Kelley *et al.*, 2001:73). Aerobic training also reduces ambulatory blood pressure and blood pressure measured at a fixed sub-maximal work load (Tsai *et al.*, 2002:571). Characteristics of the aerobic exercise training programs, that is, frequency, intensity, time, and type have not been sufficiently explained. The mechanism underlying the decreases in blood pressure in individuals participating in endurance training is most likely through a reduction in systemic vascular resistance, in which the sympathetic nervous system and the renin-angiotensin system appear to be involved (Fagard & Cornelissen, 2007:12). Weight loss might partially explain the findings.

According to Bacon *et al.* (2004:307) a drop in SBP of 1 mmHg and DBP of 0.8 mmHg is expected for every kilogram of body weight reduction. This is because weight loss due to exercise training significantly increases muscle fiber capillarization irrespective of the fiber composition of the muscle, whereas weight loss due to calorie restriction

increases the efficiency of the capillary bed in relation to body weight (Kern *et al.*, 1999:4185). A larger blood-vessel network accompanied by smaller body area can therefore decrease blood pressure. Blood pressure reduction has, however, not been expressed in terms of fat mass, but only in terms of body weight. It would be more accurate to express the decrease in blood pressure in relation to the decrease in fat mass seeing that factors other than fat, such as carbohydrate depletion and subsequent body-water loss, can contribute to weight loss.

Resistance training has also proven to have a beneficial effect on resting blood pressure in normotensive, hypertensive and borderline-hypertensive individuals (Fagard & Cornelissen, 2007:12; LaFontaine, 1997:7; Harris & Holly, 1987:250). The reduction is accounted to increased muscle-fibre capillarization (Bell *et al*., 2000:418) by means of training itself, but also by weight reduction due to calorie restriction, which increases the efficiency of the capillary bed (Neter *et al.*, 2003:878; Kern *et al.*, 1999:4185). A larger blood-vessel network for the same, or smaller, body area decreases total peripheral resistance and consequently blood pressure.

There is evidence that, during an acute exercise bout, resistance exercise increases systolic blood pressure considerably more in hypertensive individuals than in normotensives. The increase seems greater during routines where lower loads are used, but performed to the point of exhaustion (De Souza Nery *et al.*, 2010:271). Monitoring exertion levels during exercise is therefore a priority in hypertensive individuals. Even though blood pressure response is elevated during acute bouts of resistance exercise, repetitive exposure to these regimes causes favourable adaptation of the cardiovascular system and consequently lowers blood pressure.

For hypertensive individuals, a combination approach of endurance and resistance training is advised. The ACSM (2004:533) recommends moderate intensity endurance training (40-60% of $\dot{V}O_{2\text{-max}}$) for 30 minutes per day on most, but preferably all days of the week. This low-to-moderate intensity exercise targets fat oxidation that lowers fat mass and body weight. Fat- and body mass decreases contribute significantly to the

lowering of blood pressure both at rest and during exercise (Bacon *et al.,* 2004:307). In addition, endurance training should be supplemented by resistance training.

Resistance training improves blood pressure in two possible ways. Firstly, it increases FFM that enhances metabolic rate and energy expenditure (Adams *et al.,* 2003:1613). Subsequently body composition is affected beneficially and contributes to lowering blood pressure (Bacon *et al.,* 2004:307). Secondly, resistance training increases skeletal muscle vascularization (Bell *et al.*, 2000:418) causing a larger area for circulation and consequently reduces total peripheral resistance (Brooks *et al.,* 2004:358). According to Poiseuille's law, flow through a tube is directly proportional to the differences in pressure (Parker *et al*., 2009:1357). It varies directly to the fourth power of the radius of the tube and is inversely proportional to the length of the tube and the viscosity of the fluid. If the length of the tube is increased – as in the case of additional vascularization in skeletal muscles – blood pressure is consequently reduced.

Whole-body vibration seems to have little effect on heart rate and blood pressure (Hazell *et al.*, 2008:903; Otsuki *et al*., 2008:189). Acute responses of blood pressure during vibration training (*f*=26 Hz, synchronous vibration [Power-Plate®]) have been investigated in a heterogeneous group of young adults (Rittweger *et al.,* 1999:134). Exercise was carried out until exhaustion (Rate of Perceived Exertion [RPE] of 18) where blood pressure and heart rate reached 126/58 mmHg and 128 beats.min^{-1} respectively. With near-exhaustion exertion levels, heart rate and blood pressure were expected to increase significantly as they increase according to perceived exertion levels in healthy subjects (Lambrick *et al.,* 2011:1). Interestingly, heart rate and blood pressure reached only the intensity of a light cardiovascular workout. The type of muscle contractions (isometric or dynamic) and the main source of energy were not reported and could help explain these findings. Isometric muscle contraction limits oxygen delivery to the involved muscles causing phosphocreatine (PCr) and glucose to become the primary energy sources. Due to the rapid depletion of energy, early fatigue is induced in the musculature while at the same time the cardiovascular system is only minimally stressed.

Furthermore, Kerschan-Schindl *et al.* (2001:337) observed no change in HR, SBP and DBP after acute WBV (*f*=26 Hz, *A*=3 mm, sinusoidal vibration [Galileo]). Otsuki *et al.* (2008:189) found no increase in blood pressure or heart rate in ten healthy young men after 10 sets of 60-second repetitions. A rest period of 60 seconds between repetitions was, however, imposed and might have been sufficient time for heart rate and blood pressure to return to baseline levels.

Mester *et al.* (2006:1061) raised a point of caution for patients with high blood pressure engaging in vibration training. The researchers reason that vibration (especially lateral vibration) might have a high deformation effect on smaller arteries, increasing the total peripheral resistance and thereby increasing blood pressure. It could also affect damaged arteries significantly. Arteries that are damaged by atherosclerotic plaque are more rigid with unstable intimae than undamaged arteries. When external vibration is applied to the damaged arteries, plaques may detach and be transported to smaller arteries where they may cause occlusion and consequently infarction. This may also apply to individuals with widely-spread atherosclerosis and dislipidaemia. These statements have, however, not yet been proven true in practice and remain a matter of theory as no literature could be found regarding how hypertensive patients react to WBV.

Blood pressure and heart-rate responses to WBV – in all categories of risk – remain inconclusive as literature is lacking and contradictory. The necessity of research in these areas is therefore essential, and is addressed in this study.

2.11.2.2 Cardiac performance

The effect of WBVT on cardiac performance has not been investigated yet. Therefore, this section will give an overview of conservative training in the milieu of cardiac performance.

The cardiac cycle consists of an active phase (cardiac contraction) called systole and a relaxation phase called diastole (Mauritz *et al.*, 2011:473). During diastole, the ventricles relax and the chambers fill with blood in preparation for the next systole

(George *et al.*, 2010:1). During exercise, the cardiac cycle accelerates. In abnormal heart rhythms, such as ventricular tachycardia, heart rate can increase to the point where filling and stroke volume are compromised.

Cardiac performance is determined by preload, afterload, contractility, and heart rate (Figure 2.3). The relationship between myocardial stretch and stroke volume is known as the Frank-Starling mechanism (Zimmer, 2002:181). Increased myocardial stretch induced by preload, causes increased pressure in the ventricles, which leads to an increase in stroke volume and stroke work. The mechanism is most important in increasing end-diastolic volume during low intensity exercise of up to 60% of $\dot{V}O_{2\text{-max}}$. During maximal exercise, the Frank-Starling mechanism can enhance cardiac output if heart rate is reduced (Brooks *et al.,* 2004:303). For example, administering β-blocking drugs, which decrease heart rate and cardiac contractility to healthy subjects running on a treadmill, decreases maximal exercise by 40% (Bevilacqua *et al.*, 1989:853). However, stroke volume increases according to the Frank-Starling mechanism, so cardiac output remains the same. The Frank-Starling mechanism is limited in its ability to increase cardiac output by the structure of cardiac muscle (Bielen *et al.*, 1990:7).

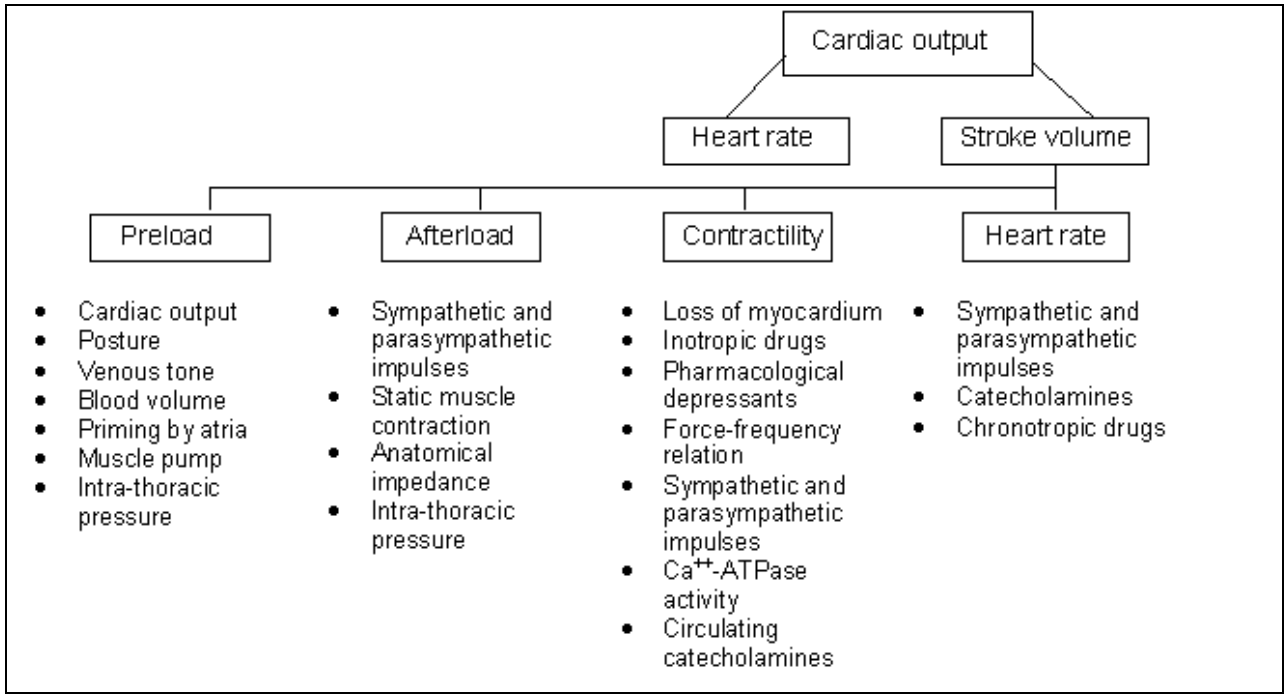


Figure 2.3: Factors affecting cardiac output during exercise (Brooks *et al.*, 2004:300)

a) Ventricular systolic function

Systolic function in terms of stroke volume and ejection fraction has not yet been investigated in the area of whole-body vibration training, but has been described in acute aerobic-type exercise studies. Research shows that the response of stroke volume to exercise may depend on many factors, including age (McCole *et al.*, 1999:2334; Wiebe *et al.*, 1999:684), fitness level (Zhou *et al.*, 2001:1849), and gender (Proctor *et al.*, 1998:599). Genetics may also play a role, as higher stroke volumes have been detected in some individuals with genetically higher blood volumes (Martino *et al*., 2002:966).

Incremental exercise protocols that investigate stroke-volume changes with increasing exercise intensities are most prevalent in the literature. Gledhill *et al.* (1994:1116) investigated young males and females and found that both systolic and diastolic time intervals decreased as acute exercise intensity increased. This leads to a plateau in stroke volume. On the contrary, diastolic filling as well as ventricular emptying was

enhanced in young endurance-trained individuals, causing progressive increase in stroke volume during exercise to maximal oxygen uptake (Ferguson *et al.*, 2001:1114; Krip *et al.*, 1997:1469; Gledhill *et al.*, 1994:1116). When endurance-trained individuals were compared to untrained individuals, the trained individuals presented significantly longer ventricular ejection times (Gledhill *et al.,* 1994:1114), greater myocardial contractility (Sullivan *et al*., 1991:1405), greater left ventricular diameter and mass (Hoogsteen *et al.*, 2003:220), and shorter diastolic filling times (Krip *et al*., 1997:1469) than untrained ones, both during rest and activity.

Spina *et al.* (1992:2458) executed a combined cycling and treadmill-running intervention study on twelve young men and women to assess the effect of endurance training on maximum oxygen uptake and stroke volume. Initially a progressive decline in stroke volume was detected when exercise was carried out to maximum oxygen uptake ($\dot{V}O_{2\text{-max}}$). This may be due to the rise in heart rate above sub-maximal levels of exertion that causes proportionately decreased stroke volume. After the training period of 12 weeks a plateau rather than a progressive decline in stroke volume was evident. The stretch-shortening capacity of the heart increased as a result of the endurance training enabling stroke volume to remain constant, while $\dot{V}O_{2\text{-max}}$ increased from 42 $ml.kg^{-1}.min^{-1}$ to 50 $ml.kg^{-1}.min^{-1}$. Later, Spina *et al.* (1993:849) found the same tendency in an older population of men (63 years) and women (64 years) who participated in a treadmill-only intervention for 9-12 months. Other researchers also found improvements in cardiac function with 12 weeks of endurance training (Sullivan *et al*., 1991:1405) and combined-type training of the same duration (Schrauwen-Hinderling, 2010:1932).

It seems that endurance-trained individuals depend more on the Frank-Starling mechanism (preload) as exercise intensity increases, compared with untrained subjects. These findings emphasize the positive effects that endurance training may have on cardiac health. The lack of longitudinal studies leave room for research which could be valuable in understanding the training effect of different kinds of exercise regimes, such

as aerobic, weight training, combined-type training and whole-body vibration training. Cardiac function is currently a barren area in the milieu of WBVT – in cross-sectional as well as longitudinal research designs. This study provides new evidence of the training effect of WBVT on cardiac function, which may provide a basis for future research.

b) Ventricular diastolic function

Cardiac function, with specific reference to left ventricular diastolic function, has not yet been explored in the area of acute WBV exercise or WBVT. The effect of conventional exercise training (resistance and endurance training) on diastolic function is also limited, but acute exercise effects are better represented.

There is no single measure of overall diastolic function. The most clinically relevant parameters of diastolic function are ventricular relaxation, myocardial or chamber compliance, and filling pressures (Table 2.5). Table 2.5 outlines the measurements of left ventricular diastolic filling and indicate which of the measurements were included in the current study.

Table 2.5: Doppler measures of left ventricular diastolic filling (Otto, 2004:171)

Variable	Measured in this research study
Left ventricular inflow velocities	
Early diastolic filling velocity *(E)*	Yes
Filling velocity after atrial contraction *(A)*	Yes
Ratio of *E/A*	Yes
Intervals	
Isovolumic relaxation time (IVRT)	No
Deceleration time (DT)	Yes
Atrial filling period	No
Acceleration/Deceleration	
Time from mitral valve opening to *E* velocity	No
Maximal acceleration	No
Early diastolic deceleration slope	No
Filling rates/volumes	
Peak rapid filling rate	Yes
Peak atrial filling rate	No
Stroke volume	Yes
Fractional filling rates	No
Doppler myocardial tissue velocities	
Early diastolic myocardial tissue velocity *(E_m)*	No
Diastolic myocardial tissue velocity after atrial contraction *(A_m)*	No
Ratio of E_m/A_m	No
Color M=Mode Doppler	
Propagation velocity	No
Mitral regurgitant jet signal	
Rate of decline in LV pressure in early diastole	No

The gold standard measure of LV diastolic function (tau) represents the pressure decline in the LV (Frais *et al.*, 1990:1071). Measurement technique used to derive tau is, however, invasive (Weiss *et al.*, 1976:494). Non-invasive imaging techniques of echocardiography (tissue Doppler, color Doppler and M-mode and pulsed-wave Doppler, and 2D speckle tracking) have emerged in the last three decades and are used to accurately assess LV-diastolic function. Echocardiography provides significant

prognostic information for predicting mortality and major cardiac events in patients with normal electrocardiographic testing (Bouzas-Mosquera *et al.*, 2010:1129). Trans-mitral flow velocities (peak early [*E*] and peak late [*A*]), *E*/*A* ratio, time velocity interval (TVI), deceleration time (DT), and longitudinal myocardial velocities (*E', A'*) are among the measurements for diastolic function.

Cardio-respiratory fitness enhances systolic function that has to be matched by LV-diastolic function (Warburton *et al.*, 1999:800). When diastolic function is impaired, systole can consequently not be supported sufficiently and may result in cardiac failure over time. The importance of impaired LV-diastolic function is thus underlined by its key role in the development and progression of a multitude of cardiovascular diseases (George *et al*., 2010:1).

Acute exercise places significant demand on the cardiovascular system for oxygen and nutrient delivery. These are transported via the arteries to metabolically active tissues. Libonati (1999:1741) states that in a closed circulation LV diastolic filling matches the increase in cardiac output in response to exercise. Given a rise in cardiac output of up to eightfold (Ekblom & Hermansen, 1968:619) during acute exercise, diastolic filling time decreases from ~0.55 s at 70 beats.min^{-1} to ~0.12 s at 195 beats.min^{-1}.

Diastolic function is augmented during acute exercise. Specifically, peak *E, E', A, A'* are reported to be HR-dependent (Peverill *et al.*, 2004:1146; Ha *et al.*, 2003:114). The combination of *E* and *E'* (*E/E'*) assess both rest and exercise LV diastolic pressures (Burgess *et al.*, 2006:1891), and increases in direct proportion to exercise intensity during incremental exercise (Quintana *et al.*, 2004:451).

After prolonged acute exercise of different modes, duration and distances, a drop in *E* and *E/A* is noted (Middleton *et al.,* 2006:681; Neilan *et al.,* 2006:1079; George *et al.,* 2004:1709). Interestingly, Neilan *et al*. (2006:1079) reported that the acute depression in both the septum and lateral wall velocities persisted for 3-4 weeks post-marathon. Similarly, *E'* had not recovered after 48 hours of rest in cyclists completing 20 prolonged exercise stages over 22 days (Williams *et al.,* 2009:97).

The effect of exercise training on diastolic function has also not been explored in the area of WBVT. Exercise training plays a major role in improving the health of diseased populations, reducing the risk for cardiovascular incidents and optimizing quality of life in all age groups and genders (ACSM, 2010:7). WBVT is a possible modality of training for the purpose of optimizing health and is increasingly popular in exercise settings. The current lack of research that answers to important health-related questions such as the effect of WBVT on cardiac function, stresses once again the importance of this research study. Current evidence for diastolic cardiac function entails the use of conventional exercise modalities and has been derived from a blend of athlete-control cross-sectional studies, with fewer longitudinal intervention studies performed in sedentary adult controls and children.

Cross-sectional studies reporting peak *E* filling velocities yielded mixed results. Karjalainen *et al.* (1997:531) reported increased *E* wave velocity in athletes, whereas other researchers reported no difference between athletes and control groups (Baldi *et al.,* 2003:2570; Sharma *et al.,* 2002:1431; Triposkiadis *et al.,* 2002:16). Conversely, peak *A* filling velocity is decreased in athletes compared to matched controls (Sharma *et al*., 2002:1431; Triposkiadis *et al.,* 2002:16; Guazzi *et al.,* 2001:139) with Baldi *et al.,* (2003:2570) suggesting no difference.

E/A ratio is more commonly reported as an index of diastolic function. Numerous studies report the resting *E/A* to be unchanged (Gates *et al.,* 2004:859; Pelá *et al*., 2004:205; Fagard, 2003:1455; Baldi *et al*., *2003*:2570; Sharma *et al.,* 2002:1431) or slightly increased in athletes (Triposkiadis *et al*., 2002:16; Schmidt-Trucksäss *et al.,* 2001:189). Studies of Olympic speed skaters (Poh *et al.,* 2008:346), professional soccer players (Tümüklü *et al.,* 2008:25), and endurance trained individuals (Nottin *et al.,* 2008:4721) which reported elevated tissue Doppler measures of *E'* and *E'/A'*. *E'*, reflecting myocardial relaxation after aortic valve closure and during early mitral inflow, was found to be higher in trained older individuals compared to untrained ones (Arab-Zadeh *et al.,* 2004:1799).

Longitudinal comparisons also yield mixed results. Healthy sedentary men who underwent six months of moderate intensity aerobic training, yielded increased septal and lateral *E'* myocardial tissue velocities (Rodrigues *et al.,* 2006:1089). Similary, three months of endurance training in previously sedentary individuals enhanced global and regional diastolic relaxation (Kivistö *et al.,* 2006:321). Conversely, in a one-year study of endurance training in 16 sedentary men, training was not associated with changes in resting *E/A* ratio, even though resting heart rate decreased (Sadaniantz *et al.,* 1996:1345). This contrasting finding is strange as population type, frequency and intensity of training, and session time corresponded in all three studies. It is expected that the longer intervention time (1 year) will cause results greater than or at least equal to the shorter studies (3-6 months).

In the shortest available intervention study on sedentary individuals (obese children), 8 weeks of resistance training improved *E'* and decreased *E/E'* (a marker of left atrial pressure). This suggests that resistance training may advance early diastolic tissue velocity in obese children, independent of changes in LV morphology or loading conditions (Naylor *et al.,* 2008:2027). If this beneficial change was independent of cardiac morphological changes and loading conditions, some unknown factor worth exploring may have caused the result.

Baggish *et al.* (2008:1121) explored possible differences in ventricular diastolic function between endurance and strength-training athletes. Endurance athletes who trained for three months demonstrated improvements in *E'* in both ventricles. Conversely, strength-trained athletes showed reduction in diastolic tissue velocities. Neglecting the complexity of the haemodynamic response to rowing, it seems that endurance training induces biventricular dilation with improved diastolic relaxation, while resistance training induces concentric hypertrophy and reduces diastolic function.

Measurements such as time velocity integral (TVI) and shortening fraction are lacking in the available research. Also, due to the many cross-sectional and fewer longitudinal

studies, it is difficult to clearly determine the impact of exercise training on diastolic function in the long run.

Moreover, this unexplored area in WBVT leaves much room for research as WBV becomes increasingly popular in performance and health settings.

2.11.3 Blood glucose

Inability of the body to transport glucose from the circulating blood to muscle tissue, results in impaired glucose tolerance (IGT) which is a state of dysglycemia that is associated with insulin resistance and increased risk of cardiovascular pathology (American Heart Association, 2007:151). Lifestyle adjustments such as regular exercise and diet modifications are considered first-line treatments for these conditions and have been widely researched (Zanuso *et al.*, 2010:15). Conventional aerobic and resistance training, as well as a combination thereof, has been investigated in the literature. Whole-body vibration as exercise modality has, however, been neglected.

Aerobic exercise has a beneficial, statistically and clinically significant effect on glycemic control as reflected by glycosylated hemoglobin (HbA_{1c}) (Boulé *et al.*, 2001:1071). Boulé *et al.* (2001:1071) found that aerobic exercise intensity had a greater effect on HbA_{1c} in adults with type 2 diabetes than exercise volume. Higher intensity programs have shown to improve insulin sensitivity more so than lower intensities in two ways. Firstly, higher intensity exercise targets carbohydrate oxidation for the supply of energy and improves insulin sensitivity. Secondly, it increases EPOC (post-exercise oxygen consumption) that results in higher quantities of energy expenditure after exercise has seized and subsequently reduces visceral and subcutaneous fat in the long run (Gill, 2007:47; Coker *et al.*, 2006:443; Dipietro *et al.*, 2006:142; O'Donovan *et al.*, 2005:522).

Resistance exercise has also been recognized to improve insulin action on tissue, leading favorably to prevention and treatment of type 2 diabetes (Holten *et al.*, 2004:294; Baldi & Snowling, 2003:419; Dunstan *et al.*, 1998:53). Holten *et al.* (2004:294), Baldi & Snowling (2003:419), and Dunstan *et al.* (1998:53) agree that

resistance training improves glycemic control by increasing the skeletal muscle storage of glucose. However, there are opposing opinions as to whether this effect is due to increase in muscle size or qualitative changes of certain muscular functions. In the case of increased muscle size (hypertrophy or hyperplasia) the total catalytic activity of the muscle improves, whereas the change in muscle function mainly results in higher amounts of glycolytic enzymes. Decreased abdominal fat has also been suggested as a contributing factor (Ibanez *et al*., 2005:662). Despite the controversy, resistance training seems to match the effectiveness of aerobic training in the treatment of type 2 diabetes and impaired glucose tolerance.

Given the effectiveness of aerobic and resistance training as separate entities, a combination of the two seem a promising treatment method. This is indeed the case. Research has shown an additional statistically significant improvement in HbA_{1c} with combined training in comparison to aerobic training alone and resistance training alone (Sigal *et al.*, 2007:357; Balducci *et al.*, 2004:841; Cuff *et al.*, 2003:2977). Light-to-moderate intensity aerobic and resistance training is therefore recommended for patients with impaired glucose tolerance and type 2 diabetes, with the main aim being abdominal and subcutaneous fat loss and increase in muscle mass.

No studies could be found investigating the effect of WBVT on blood glucose levels. However, a single study considered the effect of WBV on the blood glucose regulating hormones in the body. Di Loreto *et al.* (2004:323) found that WBV exercise did not affect insulin and glucagon levels, most likely due to enhanced glucose uptake from the blood into the musculature. They suggest that WBV exercise may lead to reduction of blood glucose levels.

This single study is insufficient to prove either potentially positive or negative effects of WBVT on blood glucose levels. However, taking into account the possible improvements in conditions secondary to impaired glucose tolerance, vibration training may be a viable option for these subjects. Secondary conditions that are improved by

WBVT include obesity (Bogaerts e*t al.,* 2007:630; Bonner Physical Therapy, 2003), and impaired circulation to the periphery (Mark *et al.*, 2008).

2.11.4 Blood lipids

Another vaguely explored subject in the area of WBVT is the lipid profile reaction of subjects training on vibration platforms. Disruption in the lipid profile is termed dislipidaemia and is considered an independent risk factor for the development of atherosclerotic diseases that include cardiac, peripheral, and cerebro-vascular disease (ACSM, 2010:48). It is therefore of vital importance to control the lipid profile.

Because lipids are not water soluble, they are transported as components of lipoproteins. Lipoproteins are composed of cholesterol, protein, triglycerides, and phospholipids. There are five types of lipoproteins classified by density, composition, and size. In order of increasing density, the five types are very low-density lipoproteins (VLDL), low-density lipoproteins (LDL), intermediate density lipoproteins (IDL), high density lipoproteins (HDL), and chylomicrons. Total cholesterol is the sum total of cholesterol carried by VLDL, LDL, HDL, and chylomicrons (Brooks *et al*., 2004:583). The regulation of cholesterol and lipoproteins is shown in Figure 2.4.

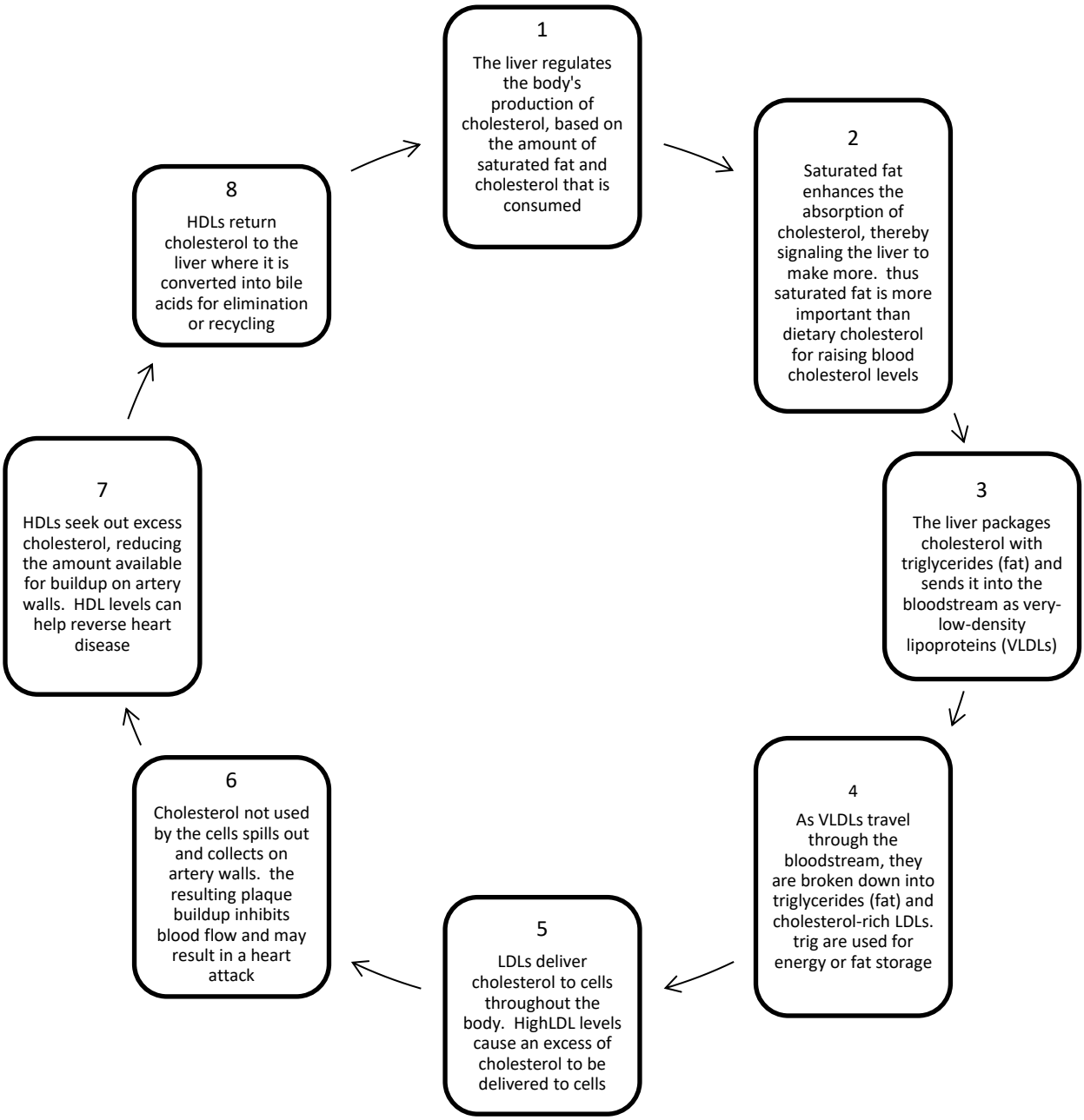


Figure 2.4: Regulation of cholesterol (Brooks *et al.*, 2004:584)

VLDL = very low-density lipoproteins; LDL = low-density lipoprotein; HDL = high density lipoprotein

Moderate-to-high intensity aerobic training (60-85% of $\dot{V}O_{2-max}$), have shown to improve the lipid profile in populations with elevated (Stein *et al.*, 1990:278) and normal (King *et al.*, 1995:2600) total cholesterol (TC) levels. Modalities most commonly reported in lipid profile trials are walking (Motoyama *et al.*, 1995:128; Ready *et al.*, 1995:909), jogging (Stefanick *et al.*, 1998:16), and cycling (Sunami *et al*., 1999:986). Benefits of aerobic training mainly include increased high density lipoprotein cholesterol (Barr *et al.*, 1991:797; Despres *et al.*, 1991:164; Stein *et al*., 1990:278). TC, low-density lipoprotein cholesterol (LDL), and triglycerides (TG) are influenced to a lesser extent (Whitehurst & Mendes, 1991:100).

Resistance training prescriptions of trials yielding beneficial lipid profile results include frequencies of 3 times per week, resistance intensities of 12-15 repetition maximum, volumes of 8-12 exercises, 1-3 sets, and 8-20 repetitions. The positive effect of resistance exercise marks out a trend for both LDL-cholesterol (Prahbakaran *et al.*, 1999:193; Boyden *et al.*, 1993:98) and HDL-cholesterol (Fahlman *et al.*, 2002:57; Joseph *et al.*, 1999:1478). TC and TG levels seem to be affected by resistance training to a lesser extent.

Combined aerobic and resistance exercise show improvements in all lipid profile variables in obese women (Park *et al.*, 2003:133). Males and females older than 40 years also show improvements in TC, HDL-cholesterol, and LDL-cholesterol levels when performing combined aerobic and resistance exercise at a frequency of 3 times per week for a period of 12 weeks or more (Kodama *et al.*, 2007:1075; Verney *et al.*, 2006:294; Tokudome *et al.*, 2004:159).

Endurance training increases LDL receptor sensitivity, HDL and lipoprotein lipase that enhances the elimination of cholesterol in the bile and may be the principal mechanism through which exercise reduces the risk of cardiovascular disease (Brooks *et al*., 2004:585). Resistance training also improves the lipid profile, but not to the same extent as endurance training. The contribution of resistance training lies primarily in its ability to influence body composition, blood pressure and glucose regulation

beneficially. Consequently the risk for the development of CVD is lowered. It seems that the main goal in conventional exercise prescription for dislipidaemia should therefore be one that includes a combination of endurance and resistance training with the emphasis on endurance training. Ehrman *et al.* (2003:180) suggest endurance training at intensities of 60-80% of $\dot{V}O_{2\text{-max}}$ that should be supplemented by moderate intensity resistance training.

Considering WBVT, a single study by Goto and Takamatsu (2005:279) reported an increase in serum-free fatty acids, but not in glycerol, 150 min after WBV exercise. The effect may have been related to the non-significant increase in growth hormone rather than catecholamine action due to the significant time lag. No other studies were found determining the effect of WBVT on the lipid profile.

Even though WBVT can be manipulated to address combined-type exercise training, it is not yet known whether the mechanism of vibration exercise is beneficial for individuals with dislipidaemia. In persons with developed atherosclerotic disease the macrophage effect enhances the development of thrombi in the areas of the blood vessels where atherosclerotic plaques have accumulated. When the already unstable intimae of the affected blood vessels are exposed to external tri-directional vibration, further disruption may be caused. Plaque fragments might come loose as a result of turbulent flow and mechanical deformation, and increase the risk of acute myocardial or cerebrovascular infarction. It is important to note that dislipidaemia often occurs in combination with other CVD risk factors – mainly obesity (Powers & Howley, 2009:478) which makes it difficult to determine the effect of exercise on dislipidaemia only.

2.11.5 Metabolic rate and energy expenditure

Metabolism implies the sum total of processes occurring in a living organism, indicated by the rate of heat production that ultimately depends on biological oxidation (Brooks *et al.*, 2004:43). During exercise, the body has the means to derive energy from the degradation of substances such as carbohydrates, fat or protein with or without the immediate use of oxygen (Brooks *et al.,* 2004:52). Common measures of metabolism include oxygen uptake ($\dot{V}O_2$), heart rate, substrate oxidation (carbohydrates, fat, and protein), and the respiratory exchange ratio (RER). The RER is determined by the ratio between oxygen uptake and carbon dioxide secretion, and is also referred to as the respiratory quotient (RQ) by some sources (Nilsson *et al.*, 2010; Peronnet & Massicott, 1991:23).

The RER assists in the calculation of the energy equivalent of the specific non-protein substrate that has been oxidized. A table designed by Lusk (Peronett & Massicott, 1991:23; Lusk, 1924:41) proposes percentages of energy derived from non-protein sources i.e. glucose (a carbohydrate) and fatty acids within extreme ranges of the RER (0.7036 – 0.9960). The lowest RER range entails fat oxidation only, and the highest range, glucose (carbohydrate) oxidation only (Peronett & Massicott, 1991:24).

The regulation of fuel selection during exercise is under complex control and is dependent upon several factors, including circulating hormones (Tataranni *et al.*, 1996:317), diet (Westerterp-Plantenga *et al.*, 2009:414) and the intensity and duration of exercise (Brooks & Mercier, 1994:2253). Other factors that may influence metabolic rate, is age, body temperature and stress (Johnstone *et al.*, 2005:941).

In acute exercise circumstances, the ATP-PC system is the first bioenergetic pathway to provide energy to the muscles, followed by glycolysis and, finally, aerobic energy production (Powers & Howley, 2009:51). Energy to perform high intensity exercise lasting up to 45 sec is generated primarily from anaerobic metabolic pathways i.e. the ATP-PC or glycolysis system (Hultman, 1973:56; Knuttgen & Saltin, 1972:690). High intensity events lasting longer than 45 seconds use a combination of the ATP-PC

system, glycolysis, and the aerobic system to produce the needed ATP for muscular contraction (Powers & Howley, 2009:56). The energy to perform low intensity activities lasting longer than 10 min originates primarily from aerobic metabolism (Hagberg *et al*., 1978:381).

During rest, $\dot{V}O_2$ amounts to around 3.5 $ml.kg^{-1}.min^{-1}$ in an average 70 kg male, which is equal to one metabolic unit (1 MET) (ACSM, 2010:3). During maximal exercise, however, oxygen uptake can reach levels up to 70 $ml.kg^{-1}.min^{-1}$ or 20 METs in endurance-trained individuals such as elite cyclers (About.com, 2011). Heart rate and oxygen uptake are directly related. Therefore heart rate will increase proportionately to exercise intensity, as is the case in oxygen uptake. For example, during normal resistance-type exercise, heart rate increases in proportion to the muscle mass used and according to the percentage of maximum voluntary contraction (Pollock *et al.*, 2000:828).

Indirect calorimetry has become a famous, non-invasive, and accurate way of determining the substance from which energy is derived. After a period of fasting, fat is the primary energy source (Soeters *et al.,* 2009:589), and is usually lower in obese, compared to normal-weight individuals (Pérez-Martin *et al.,* 2001:466). During high intensity exercise, carbohydrates are generally the major fuel source (Brooks & Trimmer, 1995:1205). During prolonged exercise, however, there is a gradual shift from carbohydrate metabolism toward fat metabolism (Ladu *et al.*, 1991:404). Proteins contribute less than 2% of the fuel used during exercise of less than one hour's duration (Van Hall *et al.*, 1995:251). High intensity exercise can cause release of hormones such as glucagon, growth hormone, and epinephrine that stimulate lipolysis and increase the amount of circulating triglycerides (Phillips *et al.,* 1996:2182; Felsing *et al.,* 1992:157).

Basal metabolic rate can be increased by resistance training but is hardly, if at all, influenced by aerobic training (Hunter *et al.*, 2008:1045; Tsuzuku *et al.,* 2007:549). WBV is considered a special method of strength training and is therefore expected to

yield similar results to that of resistance training. There is, however, a scarcity of research regarding the effect of WBV-exercise and/or WBV-training, on metabolism. The available studies also lack description of specific fuel sources that provide energy during exercise.

Wang and Kerrick (2002:2409) found increases in ATP turnover when vibration is applied to intact or skinned single-fiber preparations. Accordingly, a certain amount of energy demand is expected arising from the exposure to vibration. This is indeed the case and was proven by Rittweger *et al.* (2001:169; 2002a:428).

Energy turnover appears to vary according to the exercise position of the person, the frequency and amplitude of the vibration plate, external load added during exercise (Rittweger *et al.,* 2002a:428), and pre-exercise food consumption (Da Silva *et al.*, 2007:470). Under various conditions such as standing and squatting (f=26 Hz, A=3 mm), Rittweger *et al.* (2001:169) found that oxygen consumption amounts to 14.0±2.7 $ml.kg^{-1}.min^{-1}$. In addition, Da Silva *et al*. (2007:470) found energy expenditure reaching a level of 4.5 $kcal.min^{-1}$ during WBV (f=30 Hz, A=4 mm) in active men.

Zange *et al.* (2008:265) studied muscular ATP turnover during vibration (f=20 Hz, A=2 mm) by means of magnetic resonance spectroscopy. Vibration-related ATP consumption decreased intracellular Phospho-creatine (PCr) levels when arteries were suppressed due to isometric muscle contraction. A possible explanation for the use of PCr may be that when the arteries are suppressed, less blood flow is allowed to the musculature and consequently limited oxygen and nutrients are delivered to the muscle cells. In this way the muscle cells are forced to utilize energy anaerobically from CHO stores. Consequently, oxidative phosphorylation and ATP regeneration was hampered (Zange *et al.,* 2008:265).

Cook *et al.* (2000:7) stated that WBV may not be a viable option for directly losing body fat. Assuming an energy equivalent of 39 kJ for each gram of body fat, a typical 70 kg person would consume ≈20 liters of oxygen and use approximately 10 g of fat for each hour while performing a standard WBV-exercise regimen. Similarly WBVT may not

increase aerobic fitness significantly as energy turnover and cardiorespiratory responses are only moderately affected (Rittweger *et al*., 2001:169; Rittweger *et al.* 2002a:428).

2.12 Summary

The health-promoting effects of endurance and resistance training on the human body has been well documented, whereas whole-body vibration training has not been investigated extensively. The literature is inconclusive as to whether this unique type of training is beneficial or detrimental to the body. This seems to be the result of an insufficient number of studies and inadequate description of testing and intervention details.

During acute whole-body vibration, the cardiovascular system seems to be minimally stressed whilst muscle power output and flexibility are enhanced. Additional symptoms reported during acute WBV-exercise are lower-leg erythema, headache, vertigo, and nausea. Training on vibration platforms for 6 weeks or longer seems to improve body composition and resting blood pressure, and holds promising possibilities for glucose control. Mechanisms responsible for findings at this stage are merely speculative as there is a lack of research on molecular level. Furthermore, blood lipids, metabolism and cardiac performance are neglected areas in WBVT, most likely due to four reasons, namely (i) WBVT is a newer method of training and currently provides a smaller scope of research topics; (ii) exercise intervention studies are labour and time intensive ; (iii) equipment used for testing metabolism, cardiac function and cardiovascular risk-related matters are costly; (iv) individuals with established blood lipid, metabolic and cardiac abnormalities have greater risk for cardiovascular incidents during exercise than those individuals without these conditions. Consequently, these areas leave much room for future research.

Whole-body vibration training appears to be a promising exercise modality for the purpose of health promotion. However, care should be taken when dealing with

diseased populations as the lack of research supply little support for safe training on vibration platforms.

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3

Methods and procedures

3.1 Introduction

This chapter describes the protocol that was designed to investigate the objectives stated in chapter one. A description of the instruments that were used as well as the technique for every measurement will be discussed. In preparation for this thesis, literature was collected from electronic databases such as Kovsiekat, Pubmed, EbscoHost (Academic Search Elite and Medline), academic journals and textbooks. This study was approved by the Ethics Committee of the Faculty of Health Sciences, University of the Free State (ethics no. ETOVS 113/09)

3.2 Research design

A quasi-experimental design comprising a before-after study on an availability population was used. The group was subjected to a pre-evaluation followed by a 12-weeks intervention regimen performed on the Power-plate tri-directional vibration platform. After the intervention period, a post-evaluation was performed. In this way the effect of whole-body vibration training (WBVT) on the cardiovascular and other systems could be determined.

3.3 Selection of participants

A convenient sample of fifty-seven male subjects (age 31±7.5 years) had initially been recruited. Thirty participants served as the experimental group and underwent WBVT for a period of 12 weeks while the other 27 stayed sedentary for the same time period and served as the control group. A larger experimental group was selected to compensate for anticipated drop-outs in the intervention group.

3.3.1 Recruitment

Recruitment involved advertising on the main campus of the University of the Free State, at First National Bank branches in the city of Bloemfontein (which was the only bank that gave permission to recruit their employees) and informal distribution of two thousand flyers at several locations in the city. All advertising was conducted with permission of the relevant authorities.

3.3.2 Selection criteria

Participants were approved or disapproved for selection if they adhered to the inclusion and exclusion criteria as stated below.

3.3.2.1 Inclusion criteria

a) Gender: Male
b) Age: 18-40 years
c) CVD-risk: Asymptomatic – with or without CVD risk factors (i.e. low risk)
d) Physical activity Status: Sedentary (Physical Activity Index of 16 or less according to the index of Sharkey & Gaskill (2007:8))

3.3.2.2 Exclusion criteria

a) Known cardiovascular, metabolic or pulmonary disease.
b) Blood pressure: Resting value of ≥140/≥90.
c) Total cholesterol (TC): ≥6.2 mmol.L^{-1}
d) Fasting glucose: ≥7 mmol.L^{-1}
e) Physically or mentally disabled
f) Bone or joint problems such as joint replacements and recently fitted coils, metal pins, bolts or plates
g) Acute hernia, discopathy, spondylolysis
h) Open wounds
i) Epilepsy
j) Cancer (any type)
k) A tendency to regular heavy migraine at rest or during exercise
l) Medicine intake for any of the above conditions, including anabolic steroids, and/or sports enhancing agents.

3.4 Selection instrument

Upon application, the prospective subject completed a selection questionnaire containing the following five parts (Appendix A).

3.4.1 Demographic information

Demographic information was gathered for the purpose of correspondence and identification. A project number was assigned to each participant.

3.4.2 Physical Activity Index (PAI) questionnaire (Sharkey & Gaskill, 2007:8)

This self-report questionnaire by Sharkey & Gaskill (2007:8) was used to quantify the physical activity status of participants. Numerical values were awarded to each level of intensity, duration and frequency in the form of a Likert scale.

Table 3.1: Physical Activity Index (Sharkey & Gaskill, 2007:8)

Intensity	Not tired	1
	A little tired	2
	Tired	3
	Very tired	4
	Exhausted	5
Duration	<10 min	1
	10-19 min	2
	20-30 min	3
	>30 min	4
Frequency	1 x per month	1
	Few times per month	2
	1-2 times per week	3
	3-5 times per week	4
	Every day	5

Values were then multiplied and presented as an index of the physical activity status of the participant. E.g. if a participant exercised at exhaustive intensities (INTENSITY = 5) for duration of 20-30 minutes (DURATION = 3) at a frequency of 3-5 times per week (FREQUENCY = 4), then the participant's PAI would be calculated as:

PAI	=	Intensity x Duration x Frequency
	=	5 x 3 x 4
	=	60

A value of ≤16 represented inactivity; values from 17-44 indicated moderate activity levels, and highly active participants scored values equal to or greater than 45 (Kriel, 2002:64). In this study, sedentary individuals were selected; therefore, participants with physical activity indexes of higher than 16 were excluded.

3.4.3 Physical Activity Readiness questionnaire (PAR-Q), (Canadian Society for Exercise Physiology, 2002).

The PAR-Q is a tool for self-guided screening for joining a physical activity program. It contained questions on health risk and physical activity and was used in this study to partially report on the inclusion and exclusion criteria for the subjects.

3.4.4 Power-Plate screening form for contra-indications to whole body vibration exercise (Power-Plate International, 2008).

This form included the contra-indications for participation in WBVT. If a person met any of the conditions that contra-indicated WBV exercise, they were excluded from the study.

3.4.5 Informed consent and declaration of accurate information

The testing procedures, risks, benefits, and confidentiality of information were explained to each participant in a language in their command. Participants then signed an informed consent form (Appendix J) declaring the giving of accurate information, voluntary participation, and the use of information for scientific purposes.

3.5 Pre-test and post-test procedures, measurements and equipment

Pre-test and post-test procedures refer to testing before and after the 12-week intervention period respectively. Pre- and post-test procedures were divided into two sessions, each scheduled on a separate day. Both sessions required subjects to be in a fasting state (12 and 4 hours respectively). Subjects were tested individually wearing comfortable clothing fit for exercise. Subjects were referred to a medical practitioner of their choice when an abnormality that could endanger their health was detected. However, no such incident occurred.

Both sessions are described as the order of testing may influence results.

3.5.1 Session 1

Subjects fasted for at least twelve hours prior to testing (Burtis *et al.*, 2006:956-957). After test procedures were explained to the participant, testing commenced. At station 1 informed consent was completed and subjects verified the selection questionnaire handed in during the selection period (Appendix A). At station 2 body mass, stature, resting blood pressure, resting heart rate, and blood lipids and glucose were tested, and at station 3, ultrasound imaging of the heart was conducted. All data was captured on a data sheet (Appendix B). Testing time for session one was approximately 45 minutes per subject.

3.5.1.1 Blood pressure and heart rate

Resting blood pressure (BP) was measured with a standard sphygmomanometer after the subject rested for five minutes in a seated position. Measurement technique described by Bloomfield *et al.* (2006:609) was followed. Heart rate was measured, also in seated position, according to the technique described by Talley & O'Connor (2010:24).

3.5.1.2 Blood lipid profile and glucose

Each subject's lipid profile (HDL-cholesterol, LDL-cholesterol, total serum cholesterol and triglycerides) as well as fasting glucose were determined with a portable Cardio

Chek hand-held analyzer (Cardio Chek P.A). This analyzer uses capillary whole-blood for measurement obtained from a finger prick. Gentle pressure to the finger was applied to enlarge the drop of blood as the capillary tube must be filled to ensure sufficient specimen size (Cardio Chek, 2011:23).

The Cardio Chek P.A. does however not measure LDL-cholesterol directly; therefore the Friedewald equation was used to calculate LDL-cholesterol (Friedewald *et al.,* 1972:499-502). For accurate results when using the equation, participants were to precede testing with twelve hours of fasting (Burtis *et al.,* 2006:956-957). Certain assumptions and limitations apply. The Friedewald equation is explained in Table 3.2.

Table 3.2: Friedewald equation (Friedewald *et al.*, 1972:499-502)

Background	The ultracentrifugal measurement of LDL is time consuming, expensive and requires specialist equipment. For this reason, LDL-cholesterol is most commonly estimated from quantitative measurements of total and HDL-cholesterol and plasma triglycerides (TG) using the empirical relationship of Friedewald et. al. (1972).
Equation	LDL = TC – HDL – [TG/2.2] Where all concentrations are given in $mmol.L^{-1}$. If calculated using all concentrations in $mg.dL^{-1}$ then the equation is: LDL = TC – HDL – [TG/5].
Assumptions	The quotient [TG/5] is used as an estimate of Very-low density lipoprotein (VLDL) cholesterol concentration. It assumes that: 1. Virtually all of the plasma TG is carried on VLDL 2. The TG:cholesterol ratio of VLDL is constant at about 5:1 Neither assumption is strictly true.
Limitations	The Friedewald equation should not be used under the following circumstances: • When chylomicrons are present • When plasma triglyceride concentration exceeds 4.52 $mmol.L^{-1}$ • In patients with dysbetalipoproteinaemia (type III hyperlipopreteinemia)

TC = Total Cholesterol; HDL = High density Lipoprotein; LDL = Low density Lipoprotein; VLDL = Very-low Density Lipoprotein; TG = Triglycerides

Single testing strips as well as panel testing strips are available for analysis in the Cardio Chek P.A range. In this study the panel testing strips were used. A unique memory chip identified each lot of testing strips to match with the analyzer (Cardio Chek P.A., 2011:22). When strips have expired, the memory chip prevented testing.

Internal validation for lipid lots, manufactured under controlled PTS processes, was tested using standard quality-control procedures. Lot-specific correlations to the reference laboratory were established, and lot-specific memory chips were programmed as per standard process. These lipid lots were then reevaluated in an internal "clinical applications" study where blood was analyzed on the internal reference method which has been calibrated against the CRMLN laboratory. Correlation values for the lipid profile were as follows: Total Cholesterol, r≥0.9; HDL-cholesterol, r≥0.9; and Triglyccerides, r≥0.98 (Springer *et al.* 2010:2).

3.5.1.3 Cardiac ultrasound

Ultrasound recordings were performed by a certified cardiac sonographer. Recordings were taken with patients in the left lateral decubitus position. M-mode traces were recorded at a speed of 25 $mm.s^{-1}$ and the Doppler signals at 50 $mm.s^{-1}$. Three consecutive cycles were averaged for every parameter. Left ventricle diameter, fractional shortening, and ejection fraction were determined from M-mode traces which were recorded from the parasternal long-axis view. Measurement technique complied with established standards as suggested by Nagueh *et al.* (2009:165).

Standardized trans-thoracic echocardiography, Doppler, and pulsed-wave tissue Doppler examinations were performed using commercially available equipment in all subjects (Eseote Mylab 50 cv Gold). Specific views included the parasternal long-axis view and the apical four chamber view.

Tissue Doppler interrogation was performed on the septal and lateral anular segments of the left ventricle. In the apical four-chamber view a Doppler sample volume was placed in correspondence of both the left ventricle basal septum and the basal lateral wall.

3.5.2 Session 2

Session 2 comprised two stations, namely, anthropometry and WBV-exercise testing. For this session a fasting period of four hours was required during which time participants may not have consumed any food or drinks other than pure water. No

training, sauna, swimming or showering was allowed during this time as exercise and heat produce increased blood flow in the skin with a concomitant increase in skinfold thickness (Marfell-Jones *et al*., 2006:56). Also, no smoking was allowed for two hours prior to testing. Fasting (4 hours) ensured accurate measurement of metabolic rate during exercise testing. Anthropometrical data was captured on a data sheet (Appendix C) as was whole-body vibration exercise test (Appendix D). Total testing time for station 2 was approximately 90 minutes.

3.5.2.1 Anthropometry

Anthropometric measurements (Appendix C) enabling the calculation of percentage body fat (%BF), fat-free mass (FFM), Body mass index (BMI), and waist-to-hip ratio (WHR) was performed on each subject. Measurements were carried out by a qualified anthropometrist and measurement technique followed the guidelines of the International Standards for Anthropometric Assessment (Marfell-Jones *et al.,* 2006:21-117) unless otherwise stated.

Three skinfolds (chest, abdominal, and mid-thigh) were measured in duplicate to the nearest 0.2 mm with a Harpenden skinfold caliper. This caliper had constant pressure of 10 $g.mm^{-2}$ (Cambridge Scientific Instruments, Cambridge, MA). The two measured values were averaged. In cases where the measurement error was greater than 2 mm, a third measurement was taken. The median of the 3 measurements was taken as the final value (Marfell-Jones *et al.,* 2006:8). For standardization and re-test validity skinfolds were measured on the right side of the body unless otherwise stated.

Girths were measured to the nearest 0.1 cm and included the chest, biceps (tensed), waist, umbilicus, gluteal, mid-thigh, knee and calf. Knee girth was measured according to the prescriptions of Lohman *et al.* (1988:41). The tool that was used for girth measurement was a Lufkin (Cooper Tools, Apex, NC) unstretchable metal measuring tape.

The Bi-iliocristal bone breadth was measured to the nearest 0.1 cm with a large sliding caliper (Campbell 54cm: Rosscraft). In cases where the measurement error of the first two readings was greater than 2 mm, a third measurement was taken. The median of the three measurements were taken as the final value (Marfell-Jones *et al.,* 2006:8).

Equations that were used to calculate body-composition values are outlined in Table 3.3.

Table 3.3: Equations for calculation of body-composition components

Component	***Equation***	***Reference***
Body mass index (BMI)	BMI ($kg.m^{-2}$) = weight(kg)/height$(m)^2$	ACSM, 2010:63
Waist-to-hip ratio (WHR)	WHR = waist girth/gluteal girth	ACSM, 2010:64
Body density (Db)	Db (g/cc) = 1.109380 – 0.0008267 (∑3SKF) + 0.0000016 $(∑3SKF)^2$ – 0.0002574 (age)	Jackson & Pollock, 1985:76
Percentage body fat (%BF) *Males 18-59 years*	%BF = 495/Db – 450	Heyward & Wagner, 2004:9
Fat free mass (FFM) *Males 18-40 years*	39.625 + 1.0932(BW) +0.8370 (bi-iliac crest D) + 0.3297 (AB_1 C) - 1.0008 (AB_2 C) – 0.6478 (knee C)	Wilmore & Behnke, 1969:25

SKF = skinfold; BW = body weight; D = diameter; AB_1 = smallest circumference of the abdominal area; AB_2 = umbilical circumference; C = circumference

3.5.2.2 Whole-body vibration exercise test

The Power-Plate® pro5 AIRdaptive™ model was used for the testing as well as the intervention. This tri-directional vibration platform provides a large range of settings for frequency (*f*=30-50 Hz), amplitude (*A*=2-6 mm), and time (*t*=30-60 s) with 45 kg of extra load-bearing capacity and three air settings (Power-Plate International Ltd., 2010).

Participants were then connected to:

- A 12-lead electrocardiogram (ECG) for accurate HR-measurement, and to detect cardiac abnormalities.

- A spiro-ergometer (ZAN600 CPET) via a breathing mask and ergo flow-sensor. Using indirect calorimetry, peak oxygen uptake ($\dot{V}O_2$) and substrate use during rest and exercise was measured (see Figure 3.1) (nSpire, 2008:132).

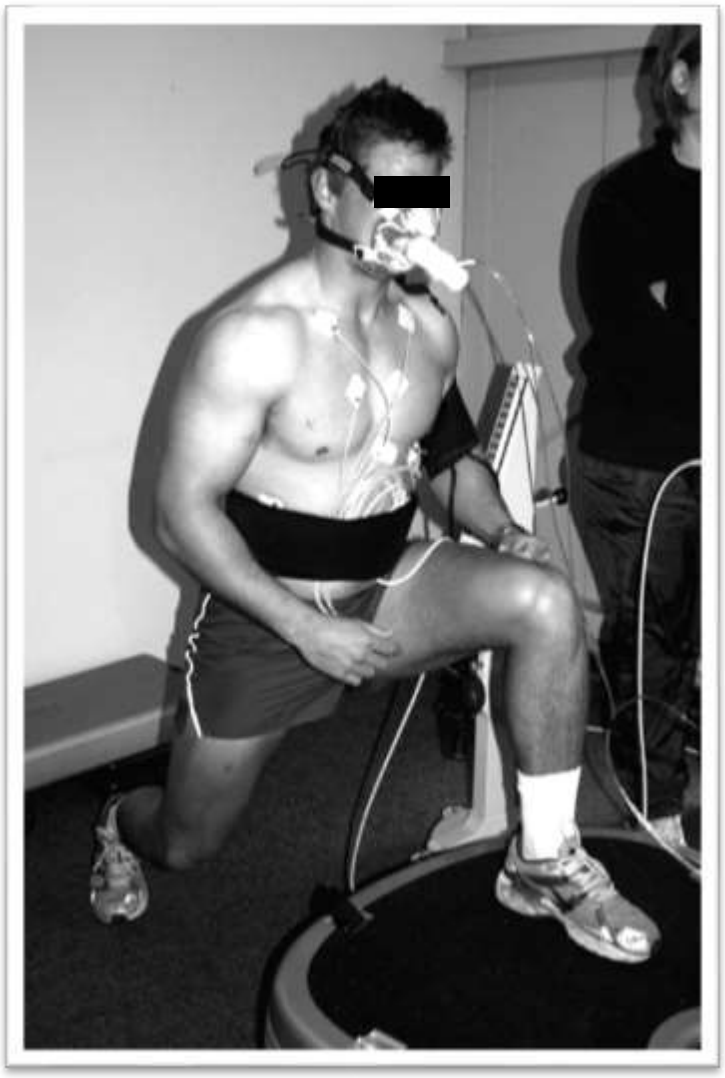

Figure 3.1: Exercise-test setup with participant performing an isometric lunge

Participants then executed twenty exercises, ten lower body and ten upper body (Table 3.4). Each isometric exercise was kept in correct position for 60 s. Tri-directional vibration at a frequency (*f*) of 40 Hz and amplitude (*A*) of 4-6 mm was induced. No resting time between exercises was allowed. Blood pressure was measured in a standing position at the end of the third, sixth, and 10th exercise of each session. A recovery period of three minutes followed exercise where blood pressure was measured

in seated position directly after the first and third minute of recovery. The rationale of recovery in seated position, rather than in standing position, was to allow maximal rest of musculature. Whole-body vibration exercise test data was captured on a data sheet (Appendix D). An illustrated version of the exercise test protocol can be viewed in Appendix E.

Table 3.4: Exercises of the test protocol

No	Lower body	Upper body
1	45º squat	Push-up on the knees
2	Deep squat	Triceps dip
3	Wide squat	Bicep curl
4	Pelvic lift (bridging)	Lateral deltoid raise
5	Double-leg heel raise	Row
6	Lunge (right leg)	Push-up on the knees
7	Lunge (left leg)	Triceps dip
8	Pelvic lift (bridging)	Bicep curl
9	Double-leg heel raise	Lateral deltoid raise
10	Wide squat	Row

3.6 Intervention

The intervention program took the form of a 12-week WBVT-program designed by Power-plate International (2008). The Power-plate International (2008) exercise program has a basic structure of strength training (both upper and lower body), stretching, massaging and relaxation. Duration per session ranges between 20 and 30 minutes, depending on the repetition time and resting periods. Intervention frequency was 3 times per week. Weekly progressions of the program followed the guidelines of Power-plate® International (2008) for an introductory whole-body vibration training program (Table 3.5).

For an illustrated version of the exercise program (first session), see Appendix F.

Table 3.5: Weekly progressions of the exercise program

		Specifiatons of exercise sessions											
		Phase 1			Phase 2			Phase 3			Phase 4		
		week 1	*week 2*	*week 3*	*week 4*	*week 5*	*week 6*	*week 7*	*week 8*	*week 9*	*week 10*	*week 11*	*week 12*
Strength	*sets*	1	1	1	1	1	1	1	1	1	1	1	1
	duration	30s	45s	60s	30s	45s	60s	30s	45s	60s	30s	45s	60s
	frequency	30	30	30	30	30	30	40	40	40	40	40	40
	amplitude	low	low	low	high	high	high	low	low	low	high	high	high
	rest	30s	15s	0s	30s	15s	0	30s	15s	0s	30s	15s	0s
Stretching	*sets*	1	1	1	1	1	1	1	1	1	1	1	1
	duration	30s	30s	30s	30s	30s	30s	30s	30s	30s	30s	30s	30s
	frequency	30	30	30s	30	30	30	30	30	30	30	30	30
	amplitude	low	low	low	low	low	low	low	low	low	low	low	low
	rest	0s	0s	0s	0s	0s	0s	0s	0s	0s	0s	0s	0s
Massage	*sets*	1	1	1	1	1	1	1	1	1	1	1	1
	duration	60s	60s	60s	30s	30s	30s	30s	60s	60s	30s	30s	30s
	frequency	40	40	40	40	40	40	40s	40	40	40	40	40
	amplitude	high	high	high	high	high	high	high	high	high	high	high	high
	rest	0	0	0s	0s	0s	0s	0s	0s	0s	0s	0s	0s
Relaxation	*sets*	1	1	1	1	1	1	1	1	1			
	duration	60s	60s	60s	60s	60s	60	30s	60s	60s			
	frequency	40	40	40	30	30	30	40	40	40			
	amplitude	low	low	low	low	low	low	low	low	low			
	rest	30s	30s	30s	0s	0s	0s	30s	30s	30s			

	Amount of exercises per category per session											
	Phase 1			Phase 2			Phase 3			Phase 4		
	week 1	*week 2*	*week 3*	*week 4*	*week 5*	*week 6*	*week 7*	*week 8*	*week 9*	*week 10*	*week 11*	*week 12*
Strength	9	9	9	11	11	11	13	13	13	15	15	15
Stretching	4	4	4	4	4	3	3	1	1	1	1	1
Massage	5	5	5	3	3	4	4	4	4	3	4	4
Relaxation	3	3	3	2	2	2	1	1	1	0	0	0

Low = 2-4 mm; high = 4-6 mm

3.7 Pilot study

A pilot study with five subjects was conducted ten months prior to the intervention regimen. It consisted of testing only without intervention with the primary aim to determine the effectiveness of the proposed questionnaires, data sheets, equipment, and protocols. Additionally, duration of testing per subject as well as the continuity of testing was evaluated in order to properly plan testing schedules for the study.

3.7.1 Outcomes

Questionnaires, data sheets, equipment, and protocols were found effective in testing the proposed objectives. A few concerns did, however, arise. During exercise, three of the five participants presented with signs of cardiac ischemia i.e. ST-segment depression of up to 2 mm as well as multi-focal premature ventricular complexes

(PVC's). One subject presented with twenty six multi-focal PVC's in the fifth minute of exercise. The particular subject was referred to a cardiologist.

Another concern was the rise in diastolic blood pressure within the first five minutes of exercise. A rise in DBP of more than 10 mmHg and even up to 20 mmHg was prevalent in three of the five subjects.

Possible reasons for the above findings:

- Age. Subjects' ages ranged from 52-60 years which predisposed them to arteriosclerosis and consequently DBP increases during exercise.
- Dislipidaemia. Two subjects used chronic medication for dislipidaemia, even though their total cholesterol reading on the day of testing was 5.01 and 6.0 $mmol.L^{-1}$ respectively.
- Limited physical activity. Subjects had a Physical activity Index of twenty four or less, suggesting moderate or low levels of activity (Sharkey & Gaskill, 2007:5).

3.7.2 Adjustments to the inclusion and exclusion criteria of subjects

Experimental group age was adjusted to 18-40 years as this category proposes least risk for CVD in adults. Subjects were also not allowed to take chronic medication for any condition. A sedentary lifestyle was compulsory so as to maximize training effect of WBV.

3.8 Statistical analysis

Data was captured from the data sheets to Microsoft Excel by the researcher. Further analysis was done by a statistician using SAS Version 9.1.3. Frequencies and percentages were calculated for categorical data. For numerical data, means and standard deviations were calculated where data was evenly distributed, and medians and percentiles were calculated where data were skew. The Student's t-test was used to compare mean values between the two groups (Research methods knowledge base, 2006). The appropriate p-values and/or confidence intervals were reported. The mean differences were calculated within the groups for the dependent data. The Student's t-

test was used to determine the significance of differences between pre-test and post-test values in the same group, as well as differences between the groups. Statistical significance was set at a level of $p \leq 0.05$ for this study and marked with a single asterisk sign (*) where applicable. Additionally, levels of significance equal to or smaller than 0.001 ($p \leq 0.001$) were marked with a double asterisk sign (**).

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4

Results and discussion

4.1 Introduction

In a complex biological system such as the exercising human body, many physiological processes occur simultaneously to establish ongoing homeostasis. Numerous bodily systems such as the cardiovascular, musculo-skeletal, respiratory, endocrine, and nervous systems integrate constantly to support the body for optimal function. In this study the reaction of sedentary young males to a less researched type of training, namely whole-body vibration, was investigated. Discussion of results will address health-related effects and will not include sports performance whatsoever.

Results and discussion of results are combined in this chapter. Results of each category of variables will be displayed in tables and followed by a discussion of the findings. The group of men (age 31±7.5 years) that underwent 12 weeks of whole-body vibration training (WBVT) will henceforth be called the exercise group (Ex group), where the group of men (age 30±6.3 years) that did not take part in any intervention will be called the control group (Con group).

For many of the variables, data distribution was skew, therefore tables not only display mean values with accompanying standard deviations (Mean±SD), minimum, and maximum values, but also median values with accompanied lower- and upper-quartile values. Applicable values of each variable are printed in bold to highlight which distribution type is relevant. For the sake of consistency in this discussion, only mean values and standard deviations will be reported in the text.

Differences within (intra-group) and between (inter-group) groups were calculated by student's t-tests. These values are displayed in separate tables and marked with one asterisk (*) where the significance level was $p \leq 0.05$, and two asterisks (**) where the significance level was $p \leq 0.001$. Inter-group differences were statistically corrected for (ANCOVA) so as to consider initial differences between the groups.

Demographic information of both groups will be addressed first, followed by the variables that were investigated, and the whole compass of information will be concluded by a summary of the main findings. For the sake of consistency inter- and

intra-group differences will be discussed in the same order and manner under each category of variables.

Both groups (Ex and Con) were tested alike before and after the intervention period of 12 weeks. The Ex group was subjected to WBVT intervention of progressive nature during that time and the Con group did not undergo any form of exercise intervention. Results displayed were limited to those who complied with the pre- and post-testing regulations. Additionally, only participants in the Ex group who completed a minimum of 80% of the prescribed exercise sessions were included.

4.2 Demographic information

Demographically the two groups of sedentary men are described by age, stature and body weight (BW). Results are displayed in Tables 4.1 and 4.2.

Table 4.1: Demographic information

Variable	N	Lower Quartile	Median	Upper Quartile	Mean	SD	Minimum	Maximum
Exercise group								
Age (yrs.)	23	**27.0**	**31.0**	**36.0**	31	7.5	20.0	39.0
Stature (m)	23	**1.73**	**1.79**	**1.81**	1.79	0.07	1.69	2.02
BW (kg)	23	77.5	84.7	102.0	**88.4**	**14.8**	**64.1**	**112.7**
Control group								
Age (yrs.)	17	**25.0**	**26.0**	**37.0**	30	6.3	22.0	39.0
Stature (m)	17	**1.71**	**1.72**	**1.74**	1.71	0.05	1.61	1.79
BW (kg)	17	64.4	68.4	77.8	**71.3**	**12.3**	**51.4**	**101.8**

BW = body weight

Bold print indicates distribution type.

Table 4.2: Inter-group differences of demographic information

Variable	Pre-test	Post-test
Age (yrs.)	0.76	0.76
Stature (m)	0.0008**	0.54
BW (kg)	0.0004**	0.08

BW = body weight

* p≤0.05; ** p≤0.001

Post-test inter-group differences are corrected for initial differences.

4.2.1 Inter-group differences

Apart from age, the exercise (Ex) and control (Con) groups initially differed significantly from each other in terms of body weight and stature (p≤0.001). The Ex group was taller than the Con group with stature of 1.79±0.07 m compared to 1.71±0.05 m, and heavier than the Con group with body weight 88.4±14.8 kg compared to 71.3±12.3 kg. With

these dissimilarities corrected for, no significant difference in any demographic variable was revealed in the post-test.

4.3 Body composition

The body composition of the two groups is described by selected measures of body dimensions. Variables in this category which influence an individual's risk for developing cardiovascular and metabolic diseases are body mass index (BMI), waist circumference (WC), waist-to-hip ratio (WHR), and percentage body fat (%BF). Onset of disease risk is recognized by a BMI of higher than 24.9 $kg.m^{-2}$, WC larger than 102 cm, WHR higher than 0.86, and %BF higher than 20% (ACSM, 2010:66).

Two measurements, namely fat free mass (FFM) and excess fat (ExF) were calculated additionally. FFM represents the difference in body weight and fat weight whereas excess fat represents the absolute value of fat, expressed in kilograms, exceeding the recommended upper range of 20% for young men.

Body composition variables for the Ex and Con groups are presented in Tables 4.3-4.5.

Table 4.3: Body composition of the exercise group

Variable	N	Lower Quartile	Median	Upper Quartile	Mean	SD	Minimum	Maximum
Pre-test								
BW (kg)	23	77.5	84.7	102.0	**88.4**	**14.8**	**64.1**	**112.7**
BMI ($kg.m^{-2}$)	23	24.3	28.3	30.4	**27.5**	**3.9**	**20.3**	**34.1**
% BF	23	18.3	22.4	26.3	**22.9**	**6.3**	**8.4**	**35.4**
FFM (kg)	23	63.9	67.2	71.1	**68.4**	**9.3**	**51.7**	**94.0**
ExF (kg)	20	3.7	6.9	11.7	**8.9**	**6.9**	**1.4**	**24.5**
WC (cm)	23	89.5	96.6	101.8	**96.3**	**10.7**	**76.2**	**121.6**
WHR	23	**0.84**	**0.88**	**0.91**	0.89	0.095	0.81	1.27
Post-test								
BW (kg)	23	77.2	81.1	104.7	**87.7**	**14.9**	**63.9**	**112.3**
BMI ($kg.m^{-2}$)	23	23.6	27.2	30.6	**27.2**	**4.0**	**21.0**	**34.9**
% BF	23	16.5	20.7	27.7	**22.3**	**6.6**	**7.9**	**34.0**
FFM (kg)	23	64.1	66.8	70.7	**67.4**	**7.7**	**53.6**	**83.4**
ExF (kg)	20	1.9	6.9	13.8	**8.2**	**7.5**	**-2.4**	**22.7**
WC (cm)	23	85.9	93.9	100.2	**94.3**	**11.1**	**76.5**	**120.0**
WHR	**23**	**0.83**	**0.87**	**0.91**	0.87	0.06	0.75	1.00

BW = body weight; BMI = body mass index; %BF = percentage body fat; FFM = fat free mass; ExF = excess fat; WC = waist circumference; WHR = waist-to-hip ratio

Bold print indicates data distribution type.

Table 4.4: Body composition of the control group

Variable	N	Lower Quartile	Median	Upper Quartile	Mean	SD	Minimum	Maximum
Pre-test								
BW (kg)	17	64.4	68.4	77.8	**71.3**	**12.3**	**51.4**	**101.8**
BMI ($kg.m^{-2}$)	17	21.8	23.1	26.4	**24.2**	**3.3**	**19.7**	**31.5**
% BF	17	9.1	16.6	20.2	**14.8**	**5.9**	**5.1**	**22.2**
FFM (kg)	17	56.2	59.3	64.4	**60.7**	**7.5**	**48.8**	**80.9**
ExF (kg)	8	1.5	3.4	4.0	**3.2**	**1.6**	**0.1**	**5.0**
WC (cm)	17	73.3	82.1	89.5	**81.3**	**8.6**	**66.9**	**93.9**
WHR	17	**0.83**	**0.86**	**0.9**	0.93	0.28	0.76	1.99
Post-test								
BW (kg)	17	64.4	70.0	80.3	**72.0**	**13.1**	**49.9**	**106.9**
BMI ($kg.m^{-2}$)	17	22.1	23.9	26.7	**24.5**	**3.4**	**19.4**	**33.6**
% BF	17	10.1	15.6	22.1	**15.7**	**6.6**	**4.1**	**25.1**
FFM (kg)	17	55.9	59.5	64.3	**60.5**	**8.0**	**47.9**	**80.5**
ExF (kg)	8	1.5	5.3	5.7	**4.7**	**3.7**	**-0.9**	**10.3**
WC (cm)	17	75.8	80.3	88.0	**80.6**	**9.0**	**62.7**	**94.6**
WHR	17	**0.81**	**0.83**	**0.89**	0.82	0.13	0.76	0.97

BW = body weight; BMI = body mass index; %BF = percentage body fat; FFM = fat free mass; ExF = excess fat; WC = waist circumference; WHR = waist-to-hip ratio

Bold print indicates data distribution type.

Table 4.5: Inter- and intra-group differences for body composition

Variable	Inter-group differences (Pre-test)	Inter-group differences (Post-test)	Intra-group differences (pre-test vs. post-test)	
			Exercise group	Control group
BW (kg)	0.0004**	0.08	0.14	0.31
BMI ($kg.m^{-2}$)	0.008*	<0.0001**	0.12	0.24
% BF	0.0002**	0.14	0.49	0.07
FFM (kg)	0.39	0.009*	0.17	0.74
ExF (kg)	0.013*	0.11	0.38	0.1
WC (cm)	<0.0001**	0.12	0.0003**	0.22
WHR	0.49	0.13	0.41	0.07

BW = body weight; BMI = body mass index; %BF = percentage body fat; FFM = fat free mass; ExF = excess fat; WC = waist circumference; WHR = waist-to-hip ratio
*p≤0.05; **p≤0.001
Post-test inter-group differences are corrected for initial differences.

4.3.1 Inter-group differences

The two groups were significantly (p≤0.05) different from one another in terms of body weight and stature. It was therefore expected that body mass index would be significantly different between the groups as BMI expresses body weight in terms of body surface. This was indeed the case. When initial differences were corrected for, body weight and stature were not significantly different after the intervention period. The significant difference in BMI (p≤0.05) after the intervention might be explained by the decrease in the Ex group (27.5±3.9 vs. 27.2±4.0 $kg.m^2$) with an increase in the Con group (24.2±3.3 vs. 24.5±3.4 $kg.m^2$).

With regard to body fat estimates, the Con group was initially leaner (14.8±15.9 %) than the Ex group (22.9±6.3 %), with less excess fat (3.2±1.6 kg) than the Ex group (8.9±6.9 kg). When these differences were corrected for, body fat measurements revealed no significant difference in the post-test, proposing that the WBVT-intervention was not effective for reducing excess body fat. Waist circumference followed the same trend

providing a significant difference (p≤0.001) between the groups in the pre-test, but not when corrected for in the post-test. When waist circumference was expressed in relation to hip circumference (waist-to-hip ratio), the Con group presented with slightly higher central fat distribution (0.93±0.28) than the Ex group (0.89±0.095). No significant differences were noted in either group, both in the pre-test or post-test.

The quantity of FFM in the two groups did not differ, suggesting that their metabolically active tissue might have been proportionately equal enough not to cause differences in performance. When the initial difference was corrected for, a significant decrease in FFM was established in the Ex group after the intervention. Surprisingly FFM decreased more so in the Ex group (68.4±9.3 vs. 67.4±7.7 kg) that trained on the vibration platform than in the Con group (60.7±7.5 vs. 60.5±8.0 kg) who did not engage in any structured exercise training during the intervention time. This result indicates that WBVT negatively affected the quantity of FFM in the Ex group and is in contrast to the increases in FFM found by Osawa *et al.* (2011:102), Fjeldstad *et al.* (2009:79), Bogaerts *et al.* (2007:630), and Roelants *et al.* (2004:3) as a result of WBVT. Consequently, the decrease in FFM in the group that participated in WBVT is strange and might be the result of the small sample size.

4.3.2 Intra-group differences

a) Control group

After 12 weeks of ongoing sedentary behaviour, no significant change in any of the body composition variables was found in the Con group. Minor increases were noted in body weight, from 71.3±12.3 to 72.0±13.1 kg, BMI, from 24.2±3.3 to 24.5±3.4 $kg.m^{-2}$, excess fat, from 3.2±1.6 to 4.7±3.7 kg, and %BF, from 14.8±5.9 to 15.7±6.6 %. Conversely, a decrease in fat free mass from 60.7±7.5 to 60.5±8.0 kg was noted. The investigation period was scheduled during the winter months. The negative changes in body composition might be the result of an even stronger trend toward sedentary behaviour and additional food consumption in winter compared to summer time. One contradictory finding was that both WC (81.3±8.6 to 80.6±9.0 cm) and WHR (0.93±0.28 to 0.82±0.13) decreased in this group. Taking into account the unfavourable changes in

the other body composition variables, the positive decreases in these measurements seem strange.

b) Exercise group

After 12 weeks of WBVT, the only significant change in body composition was found in waist circumference (WC). A decrease of 2 cm around the waist from 96.3±10.7 to 94.3±11.1 cm ($p \leq 0.001$) was a positive change as waist circumference is considered to be an independent risk factor for CVD (ACSM, 2010:66). The decrease is further emphasized by taking into account that the group had initially not been in a category of risk for this specific variable. WC and WHR are directly related in that the WHR is calculated by dividing WC by the circumference of the hip. Both measures give an indication of intra-abdominal fat that increases an individual's risk for developing cardiovascular disease. Ratios for WHR higher than 0.95 indicate risk in male populations. Given the significant decrease in WC, the decrease in WHR from 0.89±0.09 to 0.87±0.06 consequently comes as no surprise.

Body weight (BW) as well as excess fat (ExF) both decreased by 0.7 kg during the intervention period, which was statistically insignificant ($p > 0.05$). Older men seem to react the same as the currently investigated group, as Bogaerts *et al.* (2007a:630) and Verschueren *et al.* (2004:352) found that BW did not change significantly in older men. Fjeldstad *et al*. (2009:79), Roelants *et al.* (2004:1), and Bonner physical therapy (2003) found similar results in younger and post-menopausal women, indicating that BW is not significantly affected by WBVT of durations up to 12 months in either gender, irrespective of age. These researchers did not investigate excess fat (Fjeldstad *et al*., 2009:79; Bogaerts *et al.*, 2007a:630; Roelants *et al.,* 2004:1; Verschueren *et al.,* 2004:352; Bonner physical therapy, 2003).

The change in body weight was most likely due to fat loss as the measurements were more or less the same. The difference in pre-test and post-test values for BW and ExF were not found to be statistically significant. However, when compared to a single procedure of liposuction, where about half a kilogram of fat is removed at a high cost, the 0.7 kg reduction in fat is both practically of value as well as economic.

Even though the decrease in percentage body fat (%BF) was not found to be statistically significant (p≤0.05), the change of 0.6% (from 22.9±6.3 to 22.3±6.6 %) was consistent with the findings of Roelants *et al.* (2004:1), and Bonner physical therapy (2003) who induced WBVT of 12 weeks and shorter in female populations, but in contrast with the significant decrease (p≤0.05) that Fjeldstad *et al.* (2009:79) reported in a female population that adhered to WBVT of 8 months (*f*=30 Hz, *A*=3 mm, time=15-60 s). Whether the change was a function of intensity or intervention time cannot be argued as training intensity has not been reported adequately in the other studies.

Reduction in fat free mass (FFM) of 1.5% from 68.4±9.3 to 67.4±7.7 kg opposes the increases found in previous WBVT-studies. In previous studies WBVT proved to be as effective as weight training for improving FFM with increases of up to 3.5% (Osawa *et al.*, 2011:102; Bogaerts *et al.,* 2007:630; Roelants *et al.,* 2004:3). Exercise time, frequency, and amplitude of the studies were similar to that of the present investigation (*t*=30-60 s; *f*=30-40 Hz; *A*=3-6 mm). Two differences were, however, noted; (i) intervention time of the current study was 12 weeks whereas intervention time in the majority of previous studies ranged from 6-12 months (Fjeldstad *et al.,* 2009:79; Bogaerts *et al.,* 2007:630; Roelants *et al.,* 2004:3), (ii) there was a difference in the way in which the exercises were performed (isometric in the current study vs. dynamic in previous studies). The first difference (intervention time) may have a significant effect on the development of FFM as longer intervention studies reported an increase. However, Osawa *et al.*, (2011:102) also conducted a 12-week intervention study and accomplished a 3.5% increase in FFM. With regards to the second difference (the way in which exercises are preformed), resistance training in both isometric and dynamic mode produces skeletal muscle hypertrophy (Adams *et al.,* 2004:1613). It was therefore expected that WBVT in either fashion would produce similar results and is for this reason not valued as an influential factor.

Taking into account the slight decreases in BW, from 88.4±14.8 to 87.7±14.9 kg; BMI, from 27.5±3.9 to 27.2±4.0 $kg.m^{-2}$; and %BF, from 22.9±6.3 to 22.3±6.6 %; WBVT seems to have the ability to alter fat distribution without significantly changing the other body composition variables. This finding was consistent with a single 12-week WBVT-

intervention study (Osawa *et al.,* 2011:101) and a conventional resistance training study on the elderly (Tsuzuku *et al.,* 2007:549). Slight changes in the other variables (BW, BMI, FFM, %BF, and WHR) are consistent with WBVT intervention studies of shorter duration i.e. 12 weeks or less (Osawa *et al.,* 2011:101; Verschueren *et al*., 2004:630), but contrast with those of longer intervention periods (8-12 months) (Fjeldstad *et al.*, 2009:79; Bogaerts *et al.*, 2007:630). Beneficial changes in body composition seem therefore to be a function of intervention time.

The contribution that exercise intensity might make to alterations in body composition should not be excluded. The literature lacks description of amplitude height, frequency of vibration, type of exercise (dynamic or static), and time of the repetition(s) or set(s), making it difficult to interpret the effect of WBVT intensity on body composition. Also, waist circumference (WC), waist-to-hip ratio (WHR) and excess fat (ExF) have not been investigated in previous WBVT studies. This study consequently offers a unique contribution to the current literature base.

It should be noted that the majority of participants in this study were overweight with BMI-values between 25.0 $kg.m^{-2}$ and 30.0 $kg.m^{-2}$. Individuals in other body composition categories of risk, such as the underweight and obesity class I-III categories, might respond differently to the same intervention protocol.

4.4 Cardiovascular function

The cardiovascular system is one of several systems in the body that help maintain a constant internal environment by delivering necessary substances to tissues and eliminating metabolic end products. The system consists of the heart and blood vessels that work as a unit. Control of the system is directed toward maintaining blood pressure, which is balanced by the need for more blood to active tissues and organs and the need to maintain the pressure in the system. In resting state, the cardiovascular system has little difficulty supplying oxygen and fuels to the tissues, and easily removes waste products to help maintain homeostasis (Brooks *et al.,* 2004:343). However, during exercise the cardiovascular system is challenged, as active tissues require more oxygen and nutrients and deliver higher quantities of waste products that

need to be removed. Regular exposure to physical activity and exercise brings about favourable adaptations that promote health, and reduce the risk for developing chronic diseases.

Cardiovascular-specific variables were recorded at rest after an overnight fasting period of 12 hours. Measurements such as blood pressure and heart rate were manually determined, whereas echocardiographic measurements such as systolic and diastolic volumes, flow velocities, and shortening fraction of cardiac muscle were measured by means of ultra sound, Doppler- and pulsed-wave-tissue Doppler techniques. Metabolic demand on the heart was calculated in terms of the double product and is reported additionally. The results of both groups are presented in Tables 4.6-4.8.

Table 4.6: Cardiovascular measurements of the exercise group

Variable	N	Lower Quartile	Median	Upper Quartile	Mean	SD	Minimum	Maximum
Pre-test								
SBP (mmHg)	23	120	132	142	**132**	**12**	**110**	**158**
DBP (mmHg)	23	78	86	94	**85**	**10**	**60**	**100**
HR (beats.min^{-1})	23	**64**	**68**	**76**	70	8	60	92
RPP	23	**8060**	**9372**	**9768**	9176	1275	7040	12880
End-Diast.Vol. (ml)	23	249	294	363	**312**	**84**	**174**	**540**
End-Syst.Vol. (ml)	23	**101**	**119**	**166**	141	73	53	414
SV (ml)	23	141	174	202	**171**	**71**	**48**	**319**
EF (%)	23	57	62	68	**63**	**8**	**51**	**81**
SF (%)	23	31	33	38	**35**	**6**	**26**	**49**
TVI (cm.s^{-1})	20	18	21	25	**21**	**4**	**13**	**28**
DT (ms)	22	100	120	157	**129**	**38**	**84**	**233**
E (cm.s^{-1})	22	67	71	77	**74**	**9**	**56**	**94**
A (cm.s^{-1})	22	39	52	56	**49**	**14**	**29**	**76**
E'SEP (cm.s^{-1})	22	10	11	12	**11**	**2**	**9**	**15**
A'SEP (cm.s^{-1})	22	9	11	11	**10**	**2**	**6**	**14**
S'SEP (cm.s^{-1})	22	**8**	**9**	**9**	9	1	7	11
E'LAT (cm.s^{-1})	22	14	16	17	**16**	**2**	**11**	**20**
A'LAT (cm.s^{-1})	22	8	10	13	**10**	**3**	**5**	**15**
S'LAT (cm.s^{-1})	22	**10**	**11**	**13**	12	2	8	17

SBP = systolic blood pressure; DBP = diastolic blood pressure; HR = heart rate; EF = rjection fraction; RPP = rate pressure product (double product); SV = stroke volume; SF = shortening fraction; End-Diast.Vol = end diastolic volume; End-Syst.Vol = end systolic volume ; TVI – time velocity integral; DT = deceleration time; E = trans mitral velocity during passive relaxation; A = trans mitral velocity during active relaxation; E'SEP = peak cell velocity during passive relaxation at the septum; A'SEP = peak cell velocity during active relaxation at the septum; S'SEP = peak cell velocity during systole at the septum; E'LAT = peak cell velocity during passive relaxation at the lateral wall of the left ventricle; A'LAT = peak cell velocity during active relaxation at the lateral wall of the left ventricle; S'LAT = peak cell velocity during systole at the lateral wall of the ventricle

Bold print indicates data distribution type.

Table 4.6 continued (Cardiovascular measurements of the exercise group)

Post-test								
	N	**Lower Quartile**	**Median**	**Upper Quartile**	**Mean**	**SD**	**Minimum**	**Maximum**
SBP (mmHg)	23	112	118	124	**116**	**11**	**90**	**130**
DBP (mmHg)	23	78	82	84	**78**	**9**	**60**	**90**
HR (beats.min^{-1})	23	64	74	78	**72**	**8**	**55**	**88**
RPP	23	**7050**	**8512**	**9600**	8446	1464	5580	11264
End-Diast.Vol (ml)	23	371	424	487	**437**	**134**	**198**	**724**
End-Syst.Vol (ml)	23	**100**	**136**	**202**	153	63	77	302
SV (ml)	23	232	270	337	**285**	**121**	**74**	**578**
EF (%)	23	60	66	70	**66**	**8**	**49**	**81**
SF (%)	23	**32**	**37**	**39**	36	6	25	50
TVI (cm.s^{-1})	23	19	22	24	**21**	**3**	**14**	**27**
DT (ms)	23	104	127	159	**126**	**32**	**73**	**178**
E (cm.s^{-1})	23	67	72	78	**73**	**14**	**49**	**102**
A (cm.s^{-1})	23	40	46	59	**49**	**12**	**33**	**77**
E'SEP (cm.s^{-1})	23	10	11	12	**11**	**2**	**7**	**16**
A'SEP (cm.s^{-1})	23	8	9	11	**10**	**2**	**6**	**13**
S'SEP (cm.s^{-1})	23	**8**	**9**	**10**	9	1	7	11
E'LAT (cm.s^{-1})	23	12	15	18	**15**	**5**	**8**	**25**
A'LAT (cm.s^{-1})	23	8	11	13	**11**	**4**	**7**	**21**
S'LAT (cm.s^{-1})	23	10	11	15	**12**	**3**	**8**	**18**

SBP = systolic blood pressure; DBP = diastolic blood pressure; HR = heart rate; EF = rjection fraction; RPP = rate pressure product (double product); SV = stroke volume; SF = shortening fraction; End-Diast.Vol = end diastolic volume; End-Syst.Vol = end systolic volume ; TVI – time velocity integral; DT = deceleration time; E = trans mitral velocity during passive relaxation; A = trans mitral velocity during active relaxation; E'SEP = peak cell velocity during passive relaxation at the septum; A'SEP = peak cell velocity during active relaxation at the septum; S'SEP = peak cell velocity during systole at the septum; E'LAT = peak cell velocity during passive relaxation at the lateral wall of the left ventricle; A'LAT = peak cell velocity during active relaxation at the lateral wall of the left ventricle; S'LAT = peak cell velocity during systole at the lateral wall of the ventricle

Bold print indicates data distribution type.

Table 4.7: Cardiovascular measurements of the control group

Variable	N	Lower Quartile	Median	Upper Quartile	Mean	SD	Minimum	Maximum
Pre-test								
SBP (mmHg)	17	118	126	130	**124**	**9**	**110**	**142**
DBP (mmHg)	17	74	80	88	**81**	**10**	**60**	**100**
HR (beats.min^{-1})	17	64	69	78	**71**	**11**	**56**	**98**
RPP	17	**7680**	**8662**	**10368**	8902	1671	6384	11564
End-Diast.Vol (ml)	17	328	365	400	**363**	**61**	**206**	**458**
End-Syst.Vol (ml)	17	81	97	114	**101**	**27**	**60**	**158**
SV (ml)	17	224	270	298	**261**	**72**	**48**	**374**
EF (%)	17	60	68	73	**67**	**7**	**56**	**77**
SF (%)	17	32	38	42	**38**	**5**	**29**	**46**
TVI (cm.s^{-1})	17	18	21	22	**21**	**3**	**14**	**26**
DT (ms)	17	94	110	131	**118**	**30**	**81**	**175**
E (cm.s^{-1})	17	73	79	89	**81**	**12**	**60**	**102**
A (cm.s^{-1})	17	43	49	54	**49**	**10**	**31**	**67**
E'SEP (cm.s^{-1})	17	10	12	13	**12**	**2**	**8**	**18**
A'SEP (cm.s^{-1})	17	8	9	10	**9**	**2**	**6**	**12**
S'SEP (cm.s^{-1})	17	**7**	**8**	**8**	8	1	6	12
E'LAT (cm.s^{-1})	17	15	18	21	**18**	**4**	**13**	**27**
A'LAT (cm.s^{-1})	17	8	9	10	**9**	**2**	**6**	**14**
S'LAT (cm.s^{-1})	17	**10**	**11**	**12**	11	2	8	16

SBP = systolic blood pressure; DBP = diastolic blood pressure; HR = heart rate; EF = rjection fraction; RPP = rate pressure product (double product); SV = stroke volume; SF = shortening fraction; End-Diast.Vol = end diastolic volume; End-Syst.Vol = end systolic volume ; TVI – time velocity integral; DT = deceleration time; E = trans mitral velocity during passive relaxation; A = trans mitral velocity during active relaxation; E'SEP = peak cell velocity during passive relaxation at the septum; A'SEP = peak cell velocity during active relaxation at the septum; S'SEP = peak cell velocity during systole at the septum; E'LAT = peak cell velocity during passive relaxation at the lateral wall of the left ventricle; A'LAT = peak cell velocity during active relaxation at the lateral wall of the left ventricle; S'LAT = peak cell velocity during systole at the lateral wall of the ventricle

Bold print indicates data distribution type.

Table 4.7 continued (Cardiovascular measurements of the control group)

Post-test								
	N	**Lower Quartile**	**Median**	**Upper Quartile**	**Mean**	**SD**	**Minimum**	**Maximum**
SBP (mmHg)	17	112	120	130	**122**	**12**	**104**	**148**
DBP (mmHg)	17	78	88	88	**84**	**9**	**60**	**96**
HR (beats.min^{-1})	17	60	65	75	**67**	**8**	**58**	**84**
RPP	17	**7040**	**7540**	**9408**	8261	1430	6696	11040
End-Diast.Vol (ml)	17	312	356	377	**350**	**66**	**242**	**476**
End-Syst.Vol (ml)	17	**72**	**89**	**116**	101	35	61	194
SV (ml)	17	215	253	283	**249**	**66**	**154**	**387**
EF (%)	17	59	63	68	**64**	**6**	**57**	**74**
SF (%)	17	**31**	**34**	**38**	35	4	30	42
TVI (cm.s^{-1})	17	18	22	23	**21**	**3**	**14**	**26**
DT (ms)	17	108	133	147	**130**	**33**	**72**	**203**
E (cm.s^{-1})	17	75	84	89	**81**	**11**	**59**	**94**
A (cm.s^{-1})	17	38	47	53	**47**	**11**	**29**	**70**
E'SEP (cm.s^{-1})	17	11	12	12	**12**	**1**	**9**	**15**
A'SEP (cm.s^{-1})	17	8	9	10	**9**	**2**	**5**	**13**
S'SEP (cm.s^{-1})	17	**8**	**8**	**9**	9	1	7	11
E'LAT (cm.s^{-1})	17	15	18	19	**18**	**4**	**11**	**27**
A'LAT (cm.s^{-1})	17	8	9	12	**10**	**3**	**5**	**15**
S'LAT (cm.s^{-1})	17	10	11	13	**11**	**2**	**7**	**15**

SBP = systolic blood pressure; DBP = diastolic blood pressure; HR = heart rate; EF = rjection fraction; RPP = rate pressure product (double product); SV = stroke volume; SF = shortening fraction; End-Diast.Vol = end diastolic volume; End-Syst.Vol = end systolic volume ; TVI – time velocity integral; DT = deceleration time; E = trans mitral velocity during passive relaxation; A = trans mitral velocity during active relaxation; E'SEP = peak cell velocity during passive relaxation at the septum; A'SEP = peak cell velocity during active relaxation at the septum; S'SEP = peak cell velocity during systole at the septum; E'LAT = peak cell velocity during passive relaxation at the lateral wall of the left ventricle; A'LAT = peak cell velocity during active relaxation at the lateral wall of the left ventricle; S'LAT = peak cell velocity during systole at the lateral wall of the ventricle

Bold print indicates data distribution type.

Table 4.8: Inter- and intra-group differences for cardiovascular measurements

Variable	Inter-group differences (pre-test)	Inter-group differences (post-test)	Intra-group differences (pre-test vs. post-test)	
			Exercise group	Control group
SBP (mmHg)	0.05*	0.005**	<0.0001**	0.38
DBP (mmHg)	0.21	0.06	0.02*	0.18
HR (beats.min^{-1})	0.74	0.07	0.17	0.09
RPP	0.56	0.04*	0.01*	0.11
End-Diast.Vol (ml)	0.04*	0.003**	<0.0001**	0.35
End-Syst.Vol (ml)	0.02*	0.032*	0.25	1
SV (ml)	0.0001**	0.077	0.001**	0.23
EF (%)	0.098	0.45	0.09	0.05*
SF (%)	0.12	0.35	0.08	0.04*
TVI (cm.s^{-1})	0.76	0.75	0.57	0.84
DT (ms)	0.04*	0.6	0.96	0.29
E (cm.s^{-1})	0.97	0.06	0.58	0.9
A (cm.s^{-1})	0.32	0.64	0.79	0.09
E'SEP (cm.s^{-1})	0.54	0.37	1	0.48
A'SEP (cm.s^{-1})	0.14	0.32	0.26	0.87
S'SEP (cm.s^{-1})	0.04*	0.2	0.66	0.08
E'LAT (cm.s^{-1})	0.02*	0.8	0.96	0.54
A'LAT (cm.s^{-1})	0.19	0.12	0.33	0.31
S'LAT (cm.s^{-1})	0.15	0.34	0.51	0.33

SBP = systolic blood pressure; DBP = diastolic blood pressure; HR = heart rate; EF = rjection fraction; RPP = rate pressure product (double product); SV = stroke volume; SF = shortening fraction; End-Diast.Vol = end diastolic volume; End-Syst.Vol = end systolic volume ; TVI – time velocity integral; DT = deceleration time; E = trans mitral velocity during passive relaxation; A = trans mitral velocity during active relaxation; E'SEP = peak cell velocity during passive relaxation at the septum; A'SEP = peak cell velocity during active relaxation at the septum; S'SEP = peak cell velocity during systole at the septum; E'LAT = peak cell velocity during passive relaxation at the lateral wall of the left ventricle; A'LAT = peak cell velocity during active relaxation at the lateral wall of the left ventricle; S'LAT = peak cell velocity during systole at the lateral wall of the ventricle

* $p \leq 0.05$; ** $p \leq 0.001$.

Post-test inter-group differences are corrected for initial differences.

4.4.1 Inter-group differences

Resting blood pressure of both the Ex (132/85 mmHg) and Con (124/81 mmHg) groups was categorized as pre-hypertensive, which ranges between normal (<120/<80 mmHg) and hypertensive (≥140/≥90 mmHg) values for adults (ACSM, 2010:47). The underlying cause of hypertension is not known in 90% of cases, and is termed essential hypertension (ACSM, 2010:248). In the other 10% of cases, hypertension is secondary to numerous factors among which excessive sodium and alcohol intake, lack of physical activity, obesity and fear of the health-care provider (white-coat hypertension), serve (Ehrman *et al.*, 2003:283). Risk for cardiovascular disease has been associated with resting blood pressure readings as low as 115/75 mmHg. According to the National Heart, Lung, and Blood Institute (2004), the risk for cardiovascular disease doubles with every increment of rise in SBP by 20 mmHg and/or 10 mmHg in DBP.

A significant difference ($p \leq 0.05$) in systolic blood pressure (SBP) was found between the Ex (132±12 mmHg) and Con (124±9 mmHg) groups (Table 4.6 and 4.7). As mentioned earlier, the difference could be explained by the higher BMI and WC values of the Ex group as these constituted distinctive differences between the groups that relate to elevated blood pressure. Diastolic blood pressure (DBP), heart rate (HR), and double product (RPP) did not vary significantly between the two groups (Table 4.6 and 4.7). Given the significantly higher SBP value of the Ex group, it was expected that RPP will also be significantly higher in the Ex group compared to the Con group, which was not the case.

In the post-test (Table 4.6 and 4.7), the initial differences between the groups were corrected for statistically. As a result of the intervention, SBP significantly decreased in the Ex group (12%) compared to the Con group (1.5%), indicating that WBVT (t=30-60 s; f=30-40 Hz; A=3-6 mm) for a period of 12 weeks might be an effective modality for decreasing SBP. This finding is consistent with aerobic (Whelton *et al.*, 2002:493; Kelley *et al.*, 2001:73), as well as resistance training regimes (Fagard & Cornelissen, 2007:12; LaFontaine, 1997:7; Harris & Holly, 1987:250) in normotensive, hypertensive, and borderline-hypertensive populations. A similar result was, however, not achieved

for DBP. No significant difference between the groups was established for this variable as a result of the intervention.

Other cardiac variables that were measured by means of echocardiography also indicated significant difference between the two groups in the pre-test (Table 4.6 and 4.7). End-systolic volume was significantly higher in the Ex group (141±73 ml) compared to the Con group (101±27 ml) ($p \leq 0.05$), whereas end-diastolic volume was significantly lower in the Ex group (312±84 ml) compared to the Con group (363±61 ml) ($p \leq 0.05$). Consequently stroke volume was significantly lower in the Ex group (171±71 ml) in comparison to the Con group (261±72 ml). After correction for these differences, the post-test revealed significant differences ($p \leq 0.05$) in end-systolic and end-diastolic volumes, but not in stroke volume. WBVT (*t*=30-60 s; *f*=30-40 Hz; *A*=3-6 mm) seems to beneficially affect left ventricular volumes over a period of 12 weeks. These differences between the trained (Ex) and untrained (Con) groups are similar to the tendency that is found between endurance trained, and sedentary individuals (Krip *et al.*, 1997:1469; Sullivan *et al.*, 1991:1405).

Deceleration time reveals the capacity of the ventricles to actively decelerate during diastole. A shorter deceleration time indicates greater activation of the Frank-Starling mechanism resulting in greater ventricular contraction during systole (Glower *et al.*, 1985:994). Before intervention, deceleration time of the Con group was shorter (118±30 $m.s^{-1}$) than the Ex group (129±38 $m.s^{-1}$) indicating more efficient contribution of the Frank-Starling mechanism in the Con group compared to the Ex group. In view of the systolic and diastolic volumes, stroke volume, ejection fraction, blood pressure, and heart rate, the cardiovascular system of the Con group seemed to be in a more optimal state than that of the Ex group. In the Con group larger blood volumes were accommodated by and ejected from the ventricles. A compliant circulatory system was able to accommodate the ejected volumes of blood and maintain blood pressure at healthier levels than the Ex group. In the post-test, however, no significant difference in DT was established between the two groups. WBVT (*t*=30-60 s; *f*=30-40 Hz; *A*=3-6 mm) appears not to affect DT significantly over a period of 12 weeks.

Myocardial cell velocities during systole (*S'*) as well as during active (*A'*) and passive (*E'*) relaxation (diastole) on lateral and septal sides of the left ventricle are indicators of myocardial efficiency. Deficiencies in myocardial cell velocity during diastole negatively influence the Frank-Starling mechanism, resulting in impaired systolic function. In turn, deficiencies in myocardial cell velocity during systole results in poor ejection capacity. These values were quite similar between the groups. However, significant differences ($p \leq 0.05$) in favour of the Con group were measured for systolic velocity of the septal left ventricular region (S'sep), and for passive relaxation of the lateral region of the left ventricle (E'lat). In the post-test, the initial differences were corrected for and revealed that 12 weeks of progressive WBVT (*t*=30-60 s; *f*=30-40 Hz; *A*=3-6 mm) did not significantly improve myocardial cell velocities in the left ventricle.

These results are in contrast with a cross-sectional study conducted by Karjalainen *et al.* (1997:531) who reported increased myocardial cell velocities during passive ventricular filling, and Sharma *et al.* (2002:1431) who reported decreased myocardial cell velocities in athletes compared to sedentary controls. However, the 12-week WBVT intervention yielded similar results to other researchers who reported no difference between athletes and control groups (Baldi *et al.* 2003:2570; Sharma *et al.* 2002:1431; Triposkiadis *et al.* 2002:16). In longitudinal studies, however, literature supports myocardial cell velocity improvement after moderate intensity aerobic training of 3 months or longer (Kivistö *et al.* 2006:321; Rodrigues *et al.* 2006:1089), and reports reduction in a resistance-trained population (Baggish *et al.*, 2008:1121). In this study, WBVT of similar duration did not yield comparable results in sedentary men to either aerobic training or resistance training. WBVT might therefore neither be as effective as aerobic training to improve, nor as detrimental as resistance training to decrease myocardial cell velocities in the left ventricle.

Flow velocities were not significantly different in the groups, both during the pre-test and post-test. Previous literature does not address the effect of training on flow velocities during active (A) and passive (E) ventricular filling. Consequently, even though these results indicate insignificant changes, the study provides groundwork in the area of flow velocities for future research in exercise science.

The distance that blood traveled during one cardiac cycle, termed the time velocity integral (TVI), followed the same trend. With the significant differences in end-diastolic volume and stroke volume, significant differences in flow velocities were expected, but not proven. This might not necessarily reflect the physiologic adaptation of the Ex group to WBV, as the values for velocity was small in relation to the cardiac volumes, with accompanying small standard deviations.

4.4.2 Intra-group differences

a) *Control group*

After the 12-week period of sustained sedentary behaviour, the Con group demonstrated unfavorable changes ($p \leq 0.05$) in two variables, namely ejection fraction (EF) and shortening fraction (SF). These findings are consistent with ongoing sedentary behaviour. The body adapts in relation to the demands that are imposed upon it (Silva, 1990:5). When the body remains sedentary, its systems will not adapt favorably over time. Inactivity predisposes the body to chronic diseases as bodily systems deteriorate (Tremblay *et al.,* 2010:729). The other cardiovascular variables only presented with minor fluctuations.

Ejection fraction (EF) is a percentage expression of stroke volume in relation to end-diastolic volume (Brooks *et al.,* 2004:297). EF is therefore dependent upon the change in stroke volume and end-diastolic volume. Stroke volume decreased from the pre-test (261±72 ml) to post-test (249±66 ml), as did end-diastolic volume from 363±61 ml in the pre-test to 350±66 ml in the post-test. The expected decrease in EF from 67±7% to 64±6% was further underlined by the reduction in shortening fraction. With a lower end-diastolic volume, less cardiac-muscles stretch is induced, resulting in weaker contraction. Consequently the reduced shortening fraction of the cardiac cells was expected. SF decreased from 38±5% in the pre-test to 35±4% in the post-test. These unfavourable changes may be the result of even less activity during the winter intervention time as the heart will decondition according to the level of sedentary behavior (Tremblay *et al.,* 2010:732).

SBP decreased by 2% from 124±9 mmHg to 122±12 mmHg where DBP increased by 4% from (81±10) to (84±9) mmHg ($p>0.05$). HR decreased by 6% from (71±11) to (67±8) beats.min^{-1} as did double product by nearly 7% ($p\leq0.05$). It seems that the decrease in both SBP and heart rate was not sufficient to decrease the metabolic demand on the heart significantly. The findings are consistent with the persistent sedentary behavior of the group. The group gained an overall 0.5 kg in body weight and 0.7 kg in fat mass (Table 4.4). According to Bacon *et al.* (2004:307), a drop in SBP of 1 mmHg and DBP of 0.8 mmHg is expected for every kilogram of body weight that is reduced. Conversely, weight gain will increase blood pressure. Due to weight gain in this group, a rise in blood pressure was expected, but was only evident in DBP. The minor drop in average SBP was negligible.

Changes in cardiovascular measurements were consistent with sedentary behavior.

b) Exercise group

In Tables 4.6 and 4.8, it is evident that SBP of the Ex group decreased favorably from a pre-hypertensive value of 132±12 mmHg to an optimal value of 116±11mmHg ($p\leq0.001$) after 12 weeks of WBVT (t=30-60 s; f=30-40 Hz; A=3-6 mm). Similarly, DBP decreased significantly ($p\leq0.05$) from a pre-hypertensive value of 85±10 mmHg to an optimal value of 78±9 mmHg. The reduction in blood pressure may be directly related to weight loss (Bacon *et al.,* 2004:307).

According to Bacon *et al.* (2004:307), a drop in SBP of 1 mmHg and DBP of 0.8 mmHg is expected for every kilogram of body weight lost. This is because weight loss significantly increases muscle fiber capillarization irrespective of the fiber composition of the muscle (Kern *et al.*, 1999:4185). The underlying mechanism for this phenomenon is attributed to an increase in the oxidative capacity of the muscles. In overweight individuals who lost body weight over a period of time, higher quantities of the enzyme succinate dehydrogenase, a key mitochondrial enzyme in the Krebs cycle, is found. This is a sign of higher mitochondrial activity (Kern *et al*., 1999:4189). Even though this effect can also be established by exercise training (Bell *et al*., 2000:418), weight-loss stimulated mitochondrial activity was not related to habitual exercise changes. As the

mitochondrial activity of the muscle fibers increase, the muscle is compelled to form new capillary vessels to supply the muscle with larger quantities of oxygen, and this consequently increases the oxidative capacity of the muscles.

A larger blood vessel network accompanied by smaller body area decreases total peripheral resistance and consequently blood pressure. Blood pressure decrease has, however, not been expressed in terms of fat mass. It would be more accurate to express the decrease in blood pressure in relation to the decrease in fat mass, given that factors other than fat, such as carbohydrate depletion and subsequent body water loss, can contribute to weight loss.

According to the weight loss of 0.7 kg in the Ex group, which was also the amount of fat mass lost, a drop in SBP of around 0.7 mmHg and in DBP of 0.5 mmHg was expected (Neter *et al.*, 2003:878). A much larger drop than expected was evident in this group and may be explained by factors other than weight loss. Mester *et al.* (2006:1061) mentioned the possibility that vibration (especially lateral vibration) could cause deformation in smaller arteries, which in turn could cause turbulent blood flow. The turbulence would increase total peripheral resistance and consequently elevate blood pressure. Individuals with developed atherosclerotic plaque, arteriosclerosis, diabetes or peripheral vascular disease may therefore court disaster when exposed to WBV. It may damage the already diseased blood vessels even further, causing discomfort, pain, elevated blood pressure, and even stroke, lung embolism or myocardial infarction, if plaque is shaken loose.

However, exposure to WBV may have a beneficial side with regard to blood pressure. Regular exposure to vibration may improve the elastic properties of blood vessels as they adapt to endure strain (Van der Meer *et al.*, 2007:111). If blood pressure is then acutely elevated by the turbulent flow and subsequent higher peripheral resistance, the vascular system will need to adapt in order to maintain homeostasis. This might be the reason for the decrease in blood pressure over and above what was anticipated as a result of weight loss. Furthermore, Rittweger *et al.* (1999:134) found that whole-body vibration improves circulation to the periphery on the basis of muscle contraction called

a secondary pump mechanism. In this way, larger quantities of oxygen and nutrients are delivered, and healing is accelerated. This unique quality of WBV could be used for the benefit of individuals with restricted circulation to the periphery.

The favourable reduction in systolic blood pressure that resulted from 12 weeks of WBVT was consistent with the effect gained by conventional modalities of aerobic training (Whelton *et al.*, 2002:493; Kelley *et al*., 2001:73) as well as resistance training (Fagard & Cornelissen, 2007:12; LaFontaine, 1997:7; Harris & Holley, 1987:250) interventions of the same or longer training periods.

In contrast to the reduction in blood pressure, heart rate increased at rest by an average of 3%, from 70±8 to 72±8 beats.min^{-1} (Tables 4.6-4.7). This contrasts with the effect achieved by endurance training. Endurance training increases parasympathetic tone to the SA-node from the vagus nerve and decreases sympathetic influence on the heart. Consequently intrinsic rhythmicity of the heart will be lowered (Carter *et al.*, 2003:33). Resting heart rate did not decrease, most likely because whole-body vibration is a form of strength training rather endurance training.

Even with the increase in heart rate, double product – as calculated by the product of SBP and HR – decreased significantly by about 10% ($p \leq 0.05$). The reduction indicates that the metabolic demand on the heart lessened due to the intervention and that the reduction in SBP was sufficient to accomplish this positive outcome, despite the increase in heart rate. In retrospect the main possibility for the initial elevated blood pressure might have been the absence of regular physical activity.

Cardiac volumes changed significantly (Tables 4.6 and 4.8). End-diastolic volume at rest increased by 40% from 312±84 ml in the pre-test to 437±134 ml in the post-test $p \leq 0.001$. Larger end-diastolic volumes cause greater stretching of the myocardium and greater activation of the Frank-Starling mechanism. Increased stroke volume and ejection fraction consequently follows. Significant increase in stroke volume by 67% from the pre-test (171±71 ml) to the post-test (285±121 ml) was evident ($p \leq 0.001$), and was accompanied by higher ejection fraction from 63±8% to 66±8%. These findings

were consistent with endurance-trained subjects (Spina *et al.,* 1992:2458; Sullivan *et al*., 1991:1405) as well as subjects who adhered to combined-type training (Schrauwen-Hinderling *et al.*, 2010:1932). As a result of the higher ejection volume, lower end-systolic volume was expected. Conversely end-systolic volume increased by 9% from 141±73 ml to 153±63 ml. The insignificant decrease in end-systolic volume of 9% might be explained in relation to the 3% increase in ejection fraction.

Increase in end-diastolic volume is usually a function of the increase in plasma volume that results after a few days of training (Hopper *et al.*, 1988:404). The reason for the increased volume is twofold. Firstly, rennin and vasopressin levels increase greatly, which result in the increased retention of sodium and water by the kidneys. Secondly, the plasma protein albumin increases, causing increased osmolality of the blood, allowing it to hold more fluid (Brooks *et al.*, 2004:332). Increased plasma volume in itself holds little advantage for an individual, apart from thermoregulation, which helps prevent fatigue. However, it increases stroke volume and consequently cardiac output which improves the capacity of the cardiovascular system. Higher stroke volume also supplies the myocardium with larger quantities of oxygen via the coronary arteries enhancing performance of the heart (Brooks *et al.,* 2004:358).

Training also leads to increased numbers of blood vessels in skeletal muscle (Bell *et al.*, 2000:418). This adaptation occurs most readily in type I (slow-twitch) muscle fibers, and reflects the degree of chronic metabolic demand placed on the tissues. Increased vascularity of skeletal muscles facilitates diffusion of oxygen, substrates, and metabolites in the muscles, and also decreases peripheral resistance (Bell *et al.*, 2000:418). The decrease in peripheral resistance may explain the significant decrease in blood pressure that was found in the Ex group.

With the increase in stroke volume and the decrease in blood pressure, a decrease in the distance blood travels in one cardiac cycle (TVI) is expected. This may be explained by Poiseuille's law (Parker *et al*., 2009:1357):

$$F = \frac{(P_1 - P_2)\pi R^4}{8LN}$$

where *F* is flow rate, $(P_1 – P_2)$ is the drop in pressure, π is a constant value of 3.1416, *R* is the radius of the tube, *L* is the length of the tube and *N* is the viscosity of the fluid (Parker *et al.,* 2009:1357).

This law states that flow through a tube varies directly with the differences in pressure. It varies to the fourth power of the radius of the tube and is inversely proportional to the length of the tube and the viscosity of the fluid. Of the four factors in this equation, length is the only one that does not vary in the physiological system. Assuming that the viscosity of blood and the radius of the blood vessels remains constant, the significant decrease in blood pressure predicts a decrease in flow. For this reason a decrease in the distance that blood would flow during one cardiac cycle was expected.

The Time Velocity Integral (TVI) actually remained constant during rest at an average value of 21 $cm.s^{-2}$ (Table 4.6). For this value to remain constant, either the radius of the blood vessels increased, or the viscosity of the blood decreased. Most likely the viscosity of the blood decreased as plasma volume increases as a result of training and consequently lowers the shear forces of the blood cells against the blood vessel walls (Hopper *et al.*, 1988:404). TVI had only been investigated during acute aerobic exercise and not in resting state as a function of training. It is therefore difficult to compare the current training results to any previous studies that investigated the TVI.

Measures of diastolic function reflect the capacity of the heart to match systolic function. Impaired diastolic function can predict cardiac disease in advance and be treated to prevent manifestation. Previously in the text, results revealed larger stroke volumes during systole as a result of larger filling volumes during diastole. In order for the heart to match systolic function, it has to adapt its diastolic function. Stroke volume can only be increased if the compliance of the left ventricle has the capacity to contract forcefully after stretching (the Frank-Starling mechanism). If diastolic filling increases and stroke volume does not, the systolic function of the myocardium is impaired. Myocardial velocities are helpful measurements to evaluate diastolic function.

Deceleration time (DT) of the cardiac muscles measures the capacity of the myocardium to decelerate during active relaxation. A shorter deceleration time affects the Frank-Starling mechanism as it influences contractility that reacts to stretching of the myocardium. DT shortened from pre-test (129±38 $m.s^{-1}$) to post-test (126±32 $m.s^{-1}$) in contrast to the lengthened period of the Con group. Other myocardial cell velocities at septal and lateral regions of the left ventricle remained constant during passive relaxation (*E'*), active relaxation (*A'*) and systole (*S'*). Peak mitral flow during early (E) and late (A) diastole also did not change during the 12 weeks of WBVT.

Diastolic function (Table 4.6 and 4.8) has improved slightly as a result of WBVT, but only in the component of deceleration time. Diastolic cell velocities i.e. (*E'*), and (*A'*) did not show improvement, which may be due to the increase in heart rate as these velocities are heart rate dependent (Peverill *et al.*, 2004:1146). In sedentary individuals, passive relaxation of the cardiac fibers (*E')* in both lateral and septal regions of the left ventricle is higher as a result of moderate endurance training of 12 weeks or longer (Rodrigues *et al.*, 2006:1089; Kivistö *et al.*, 2006:321). By contrast, strength-trained athletes (American football players) show reduction in diastolic tissue velocities (George *et al*., 2010:7). It seems that progressive WBVT over a period of 12 weeks may be neither as beneficial as endurance training, nor as detrimental as strength training on myocardial tissue velocities.

4.5 Blood lipid profile and glucose

Blood lipids and glucose were determined from capillary whole blood after a 12-hour overnight fasting period. Results of the Ex and Con groups are presented in Tables 4.9-4.11.

Table 4.9: Blood lipid profile and glucose of the exercise group

Variable mmol.L^{-1}	N	Lower Quartile	Median	Upper Quartile	Mean	SD	Minimum	Maximum
Pre-test								
TC	23	**3.17**	**3.89**	**4.72**	3.99	0.99	2.59	5.79
HDL	23	**0.54**	**0.67**	**0.89**	0.73	0.24	0.41	1.23
LDL	18	**2.63**	**2.99**	**3.23**	2.91	0.65	1.82	4.1
Trig.	23	**0.68**	**1**	**2**	1.41	0.98	0.57	3.89
Glc.	23	**3.51**	**3.9**	**4.2**	3.9	0.42	3.2	4.7
Post-test								
TC	23	**2.76**	**3.7**	**4.19**	3.7	0.9	2.59	5.74
HDL	23	**0.51**	**0.67**	**0.85**	0.71	0.33	0.39	1.93
LDL	16	**1.5**	**2.29**	**3.32**	2.32	1.11	0.43	4.52
Trig.	23	**0.76**	**1.37**	**2.4**	1.92	0.93	0.57	2.38
Glc.	23	3.7	4	4.3	**3.93**	**0.52**	**2.67**	**4.8**

TC = total cholesterol; HDL = high density lipoprotein; LDL = low density lipoprotein;
Trig = triglycerides; Glc. = glucose

Bold print indicates data distribution type.

Table 4.10: Blood lipid profile and glucose of the control group

Variable mmol.L^{-1}	N	Lower Quartile	Median	Upper Quartile	Mean	SD	Minimum	Maximum
Pre-test								
TC	17	**2.66**	**3.17**	**4.13**	3.45	0.94	2.59	5.49
HDL	17	**0.77**	**0.98**	**1.25**	1.03	0.42	0.39	2.09
LDL	9	**1.28**	**2.52**	**2.64**	2.03	0.74	1.15	2.72
Trig.	17	**0.61**	**1.06**	**1.39**	1.32	1.21	0.57	5.65
Glc.	17	**3.7**	**4**	**4.3**	4.15	0.6	3.5	5.3
Post-test								
TC	17	**2.99**	**3.33**	**4.09**	3.59	0.91	2.59	5.89
HDL	17	**0.72**	**0.89**	**1.29**	1	0.38	0.55	1.91
LDL	12	**1.05**	**1.96**	**2.36**	1.92	0.99	0.57	3.68
Trig.	17	**0.98**	**1.3**	**1.82**	1.49	0.86	0.57	3.76
Glc.	17	3.8	3.9	4.3	**4.01**	**0.59**	**2.73**	**5.2**

TC = total cholesterol; HDL = high density lipoprotein; LDL = low density lipoprotein; Trig = triglycerides; Glc. = glucose

Bold print indicates data distribution type.

Table 4.11: Inter- and intra-group differences for blood lipids and glucose

Variable mmol.L^{-1}	Inter-group differences (pre-test)	Inter-group differences (post-test)	Intra-group differences (pre-test vs. post-test)	
			Exercise group	Control group
TC	0.066	0.71	0.03*	0.33
HDL	0.012*	0.62	0.39	0.56
LDL	0.006*	0.28	0.05*	0.64
Trig.	0.72	0.32	0.06	0.13
Glc.	0.34	0.69	0.57	0.86

TC = total cholesterol; HDL = high density lipoprotein; LDL = low density lipoprotein; Trig = triglycerides; Glc. = glucose

* $p \leq 0.05$; ** $p \leq 0.001$

Post-test inter-group differences are corrected for initial differences.

4.5.1 Inter-group differences

In Table 4.10 it is evident that significant differences ($p \leq 0.05$) existed between the groups for two blood lipid variables, namely high density lipoprotein cholesterol (HDL) and low density lipoprotein cholesterol (LDL). The Con group had an HDL-value of 1.03±0.42 mmol.L^{-1} compared to 0.73±0.24 mmol.L^{-1} of the Ex group. HDL-values of the Ex group was lower than the optimal range of 1.0-1.55 mmol.L^{-1} suggested by the ACSM (2010:48), whereas the Con group's HDL-value was just within the norm. For LDL, the Con group presented a value of 2.03±0.74 mmol.L^{-1}, which was lower, compared to the 2.91±0.65 mmol.L^{-1} of the Ex group, but optimal compared to the norm of <2.59 mmol.L^{-1} (ACSM, 2010:48). The LDL-value of the Ex group was above the optimal value. Total cholesterol (TC), and triglycerides (Trig.) did not show significant differences between the two groups, and were considered desirable compared to the norms suggested by the ACSM (2010:48,50).

With the initial differences between the two groups taken into account, none of the blood lipid values indicated statistical significance in the post-test. This is in contrast to the beneficial effects of aerobic training that mainly marks out a trend to increasing HDL (Barr *et al.*, 1991:797) as well as resistance training that increases HDL (Fahlman *et al.*, 2002:57), and decreases LDL (Prahbakaran *et al.,* 1999:193)

Similarly, blood glucose did not indicate significant difference after the 12-week intervention period. This is in contrast with conventional training regimes such as aerobic (Boulé et al., 2001:1071; Gill, 2007:47), resistance (Holten *et al.*, 2004:294; Baldi & Snowling, 2003:419), and combined-type training (Sigal *et al.,* 2007:357; Balducci *et al.,* 2004:841) which all improves blood glucose control.

It seems therefore that WBVT (t=30-60 s; f=30-40 Hz; A=3-6 mm) over a period of 12 weeks may not be as effective as conventional training in improving blood lipids and glucose levels in sedentary males.

4.5.2 Intra-group differences

a) Control group

No significant change in any of the lipid profile variables or blood glucose level was detected in the Con group after 12 weeks (Table 4.10). Slight fluctuations in the lipid profile were visible, but blood glucose remained almost constant with a value around (4.0±0.59) mmol.L^{-1}. Minor increases were observed in TC, from 3.45±0.94 to 3.59±0.91 mmol.L^{-1} (4% change), and triglycerides, from 1.32±1.21 to 1.49±0.86 mmol.L^{-1} (13% change). This was in contrast to the decreases noted in LDL, from 2.03±0.74 to 1.92±0.99 mmol.L^{-1} (5% change), and HDL, from 1.03±0.42 to 1.0±0.38 mmol.L^{-1} (3% change).

b) Exercise group

Significant improvements in TC and LDL were prevalent in the Ex group after 12 weeks of WBVT (Table 4.9 and 4.11). TC decreased by 7% from 3.99±0.99 to 3.7±0.9 mmol.L^{-1} ($p \leq 0.05$) and LDL by 20%, from 2.91± 0.65 to 2.32±1.11 mmol.L^{-1} ($p \leq 0.05$). Although not significant, an increase of 36% in triglycerides from 1.41±0.98 to 1.92±0.93 mmol.L^{-1} was prevalent. HDL and glucose remained constant at 0.73±0.33 mmol.L^{-1} and 3.9±0.52 mmol.L^{-1} respectively.

Results were similar to what is found in resistance training interventions, as resistance training mainly decreases LDL-cholesterol (Prahbakaran *et al.*, 1999:193; Boyden *et al*., 1993:98). However, resistance training also increases HDL-cholesterol (Fahlman *et al.*, 2002:57; Joseph *et al.*, 1999:1478) which was in contrast to what was found in this study. TC and Trig levels are only slightly affected by conventional aerobic and resistance training interventions (Whitehurst & Mendes, 1991:100). Similar results were found in this WBVT-study.

For beneficial change in the lipid profile, literature favors combination-type training (aerobic and resistance training) (Park *et al*., 2003:133). Prescription entails a frequency of at least 3 times per week for a period of 12 weeks or longer (Kodama *et al.*, 2007:1075; Verney *et al.*, 2006:294; Tokudome *et al.*, 2004:159). The current

WBVT-study corresponded with the frequency and time of the suggested conventional exercise prescription, but did not yield the same magnitude of benefits.

Consequently, on a first-impression basis, WBVT seems the less effective exercise option to improve the lipid profile in comparison to combined-type conventional training. It might, however, be underestimated. Taking into account the normal initial values of TC and Trig, and the near-normal LDL and HDL-values of the Ex group, improvements reflect the noteworthy ability of WBVT to improve the lipid profile. These effects are doubly acclaimed when taking into account that conventional-training studies mainly investigated populations that were dislipidaemic in composition.

Fasting glucose remained unchanged (Table 4.9) and seemed not to be affected by WBVT which contrasted with the effect of aerobic training (Gill, 2007:47; Boulé *et al.*, 2001:1071), resistance training (Holten *et al.*, 2004:294; Baldi & Snowling, 2003:419), and combined aerobic and resistance training (Sigal *et al.*, 2007:357). These regimes all improve insulin action and glucose control, and consequently lower blood glucose levels.

WBVT has been found inadequate regarding the improvement of blood glucose. However, it should be noted that glucose values in the Ex group were in normal range to start with, making further improvement unlikely.

4.6 Metabolism

Preceded by a fasting period of 4 hours, peak oxygen uptake ($\dot{V}O_2$), heart rate, the respiratory exchange ratio, fat (FAT) oxidation, and carbohydrate (CHO) oxidation were measured. Measurements in this category were recorded at rest as well as during activity (Chapter 3, section 4.2.2), which was an exception to the other categories where measurements were reported only at rest. Measurements are therefore reported as (i) metabolism at rest (pre-test vs. post-test), (ii) difference in metabolism between rest and activity (pre-test vs. post-test), and (iii) metabolism during activity (pre-test vs. post-test). In addition to the significance levels (p-values) reported for rest and activity (Table 4.14), activity-induced differences in metabolism are included. These p-values reveal the level of significance for metabolic changes from resting state to activity (pre-test vs. post-test).

Oxygen uptake ($\dot{V}O_2$) is expressed in milliliters of oxygen consumed per kilogram of body mass per minute, where fat and carbohydrate usage is expressed in grams per 24 hours. Both units have limitations. The unit for oxygen consumption ($ml.kg^{-1}.min^{-1}$) assumes that every kilogram of bodily tissue consumes the same amount of oxygen per minute, regardless of its composition (fat mass, fat free mass, or body water). This is not the case as muscle tissue is metabolically more active than fat tissue (Johnstone *et al.*, 2005:941), and water has no metabolic properties. On the other hand, the unit for substrate oxidation ($g.24h^{-1}$) assumes that the subject will oxidize a certain amount of the substrate if he/she continues in the same fashion and intensity of exercise for 24 hours. Firstly, it is highly unlikely for a subject to continue with such set activity for 24 hours. Secondly, even if so, the measurement does not take into account the metabolic and hormonal changes that may occur during such an extended period of activity. Nevertheless, these units will remain as is throughout the text and will be interpreted accordingly.

Only 2 studies regarding the effect of WBV on metabolism could be found. One study addressed energy expenditure (Da Silva *et al.,* 2006:267) and the other, oxygen uptake (Ritweger *et al.*, 2001:169) during acute WBV. Due to the lack of applicable research,

relevant literature from conventional aerobic and resistance training studies will serve as points of reference.

Metabolic variables measured during rest and activity are presented in Table 4.12-4.13. Significance levels are reported in Table 4.14.

Table 4.12: Metabolic rate and energy expenditure of the exercise group

Variable	N	Lower Quartile	Median	Upper Quartile	Mean	SD	Minimum	Maximum
Pre-test								
$\dot{V}O_2$ Rest ($ml.kg^{-1}.min^{-1}$)	23	3.6	4.5	5.2	**4.5**	**1.8**	**1.3**	**7.5**
FAT Rest ($g.24h^{-1}$)	23	**68.5**	**105**	**186**	195	345	12	1740
CHO Rest ($g.24h^{-1}$)	23	**155**	**289**	**625**	402	326	1	1087.5
RER Rest	23	**0.77**	**0.83**	**0.87**	0.82	0.11	0.56	0.99
HR Rest ($beats.min^{-1}$)	23	**64**	**68**	**76**	70	8	60	92
$\dot{V}O_2$ Activity ($ml.kg^{-1}.min^{-1}$)	23	12.8	15.1	16.8	**15.1**	**4.6**	**6.7**	**25.7**
FAT Activity ($g.24h^{-1}$)	23	**82**	**110**	**196**	145	94	33	361
CHO Activity ($g.24h^{-1}$)	23	**717**	**1147**	**1347**	991	451	28	1675
RER Activity	23	**0.89**	**0.97**	**1.0**	0.95	0.11	0.65	1.15
HR Activity ($beats.min^{-1}$)	23	**100**	**114**	**126**	114	20	77	155
Post-test								
$\dot{V}O_2$ Rest ($ml.kg^{-1}.min^{-1}$)	23	3.5	3.8	5.0	**4.4**	**1.7**	**1.0**	**8.1**
FAT Rest ($g.24h^{-1}$)	23	**126**	**193**	**241**	217	131	37	476
CHO Rest ($g.24h^{-1}$)	23	**43.3**	**96.8**	**308.2**	186.7	197.7	0	704.1
RER Rest	23	**0.75**	**0.75**	**0.85**	0.8	0.12	0.65	1.15
HR Rest ($beats.min^{-1}$)	23	64	74	78	**72**	**8**	**55**	**88**
$\dot{V}O_2$ Activity ($ml.kg^{-1}.min^{-1}$)	23	10.7	11.9	14.1	**12.4**	**4.0**	**4.2**	**23.2**
FAT Activity ($g.24h^{-1}$)	23	**152**	**208**	**283**	221	93	63	464
CHO Activity ($g.24h^{-1}$)	23	**318**	**525**	**730**	557	254	246	1067
RER Activity	23	**0.83**	**0.85**	**0.88**	0.86	0.06	0.77	1.09
HR Activity ($beats.min^{-1}$)	23	**95**	**116**	**124**	112	18	82	144

$\dot{V}O_2$ = peak oxygen uptake; FAT = fat; CHO = carbohydrates; RER = respiratory exchange ratio; HR = Heart rate
Bold print indicates data distribution type.

Table 4.13: Metabolic rate and energy expenditure of the control group

Variable	N	Lower Quartile	Median	Upper Quartile	Mean	SD	Minimum	Maximum
Pre-test								
$\dot{V}O_2$ Rest ($ml.kg^{-1}.min^{-1}$)	17	2.9	4.6	5.1	**3.9**	**2.1**	**1.0**	**7.4**
FAT Rest ($g.24h^{-1}$)	17	**106**	**170**	**235**	269	319	43	1337
CHO Rest ($g.24h^{-1}$)	17	**102**	**227**	**293**	221	154	0	592
RER Rest	17	**0.73**	**0.77**	**0.85**	0.79	0.14	0.45	0.93
HR Rest ($beats.min^{-1}$)	17	64	69	78	**71**	**11**	**56**	**98**
$\dot{V}O_2$ Activity ($ml.kg^{-1}.min^{-1}$)	17	11.3	14.1	15.7	**13.3**	**4.3**	**6.0**	**21.5**
FAT Activity ($g.24h^{-1}$)	17	**95**	**122**	**151**	124	58	17	253
CHO Activity ($g.24h^{-1}$)	17	**408**	**760**	**975**	730	430	41	1425
RER Activity	17	**0.88**	**0.92**	**0.98**	0.91	0.12	0.58	1.11
HR Activity ($beats.min^{-1}$)	17	**96**	**102**	**111**	106	15	88	146
Post-test								
$\dot{V}O_2$ Rest ($ml.kg^{-1}.min^{-1}$)	17	3.0	3.8	4.5	**3.8**	**1.3**	**1.7**	**6.7**
FAT Rest ($g.24h^{-1}$)	17	**119**	**130**	**187**	225	308	87	1404
CHO Rest ($g.24h^{-1}$)	17	**41**	**105**	**222**	198	234	28	846
RER Rest	17	**0.73**	**0.77**	**0.84**	0.8	0.1	0.7	1.09
HR Rest ($beats.min^{-1}$)	17	60	65	75	**67**	**8**	**58**	**84**
$\dot{V}O_2$ Activity ($ml.kg^{-1}.min^{-1}$)	17	10.8	11.9	13.5	**12.4**	**2.3**	**7.5**	**15.3**
FAT Activity ($g.24h^{-1}$)	17	**127**	**182**	**213**	168	60	60	252
CHO Activity ($g.24h^{-1}$)	17	**360**	**561**	**589**	484	220	41	895
RER Activity	17	**0.83**	**0.87**	**0.9**	0.87	0.06	0.76	0.97
HR Activity ($beats.min^{-1}$)	17	**94**	**106**	**113**	109	20	87	158

$\dot{V}O_2$ = peak oxygen uptake; FAT = fat; CHO = carbohydrates; RER = respiratory exchange ratio; HR = Heart rate
Bold print indicates data distribution type.

Table 4.14: Intra- and inter-group differences for metabolic variables

Variable	Rest				Activity			
	Inter-group difference (pre-test)	Inter-group difference (post-test)	Intra-group difference (pre-test vs. post-test)		Inter-group difference (pre-test)	Inter-group difference (post-test)	Intra-group difference (pre-test vs. post-test)	
			Ex	Con			Ex	Con
$\dot{V}O_2$ ($ml.kg^{-1}.min^{-1}$)	0.56	0.17	0.72	0.62	0.44	0.79	<0.0001**	0.19
FAT ($g.24h^{-1}$)	0.129	0.92	0.028*	0.617	0.924	0.047*	0.04*	0.56
CHO ($g.24h^{-1}$)	0.095	0.12	0.02*	0.57	0.08	0.345	<0.0001**	0.01*
RER	0.547	0.98	0.21	0.255	0.279	0.49	<0.0001**	0.028*
HR ($beats.min^{-1}$)	0.742	0.067	0.235	0.270	0.080	0.69	0.775	0.809

$\dot{V}O_2$ = peak oxygen uptake; FAT = fat; CHO = carbohydrates; RER = respiratory exchange ratio; HR = Heart rate

*p≤0.05; **p≤0.001

Post-test inter-group differences are corrected for initial differences.

4.6.1 Inter-group differences

No significant difference was revealed between the two groups in $\dot{V}O_2$, CHO, RER, and HR, both before and after intervention. This was the case during rest as well as during activity. However, when the initial differences were corrected for, FAT revealed to have increased significantly during activity ($p \leq 0.05$) in the Ex group compared to the Con group. This indicated that WBVT (*t*=30-60 s; *f*=30-40 Hz; *A*=3-6 mm) for a period of 12 weeks, effectively improved fat oxidation in comparison to sedentary controls. The single significant difference was unusual when the significant inter-group differences in body composition (body mass index, and fat free mass) are taken into account. As mentioned earlier, metabolic activity is different in various bodily tissues (Johnstone *et al.*, 2005:941). Significant variance between the two groups was therefore expected in terms of metabolic measures, as their fat mass and fat free mass differed significantly.

Even though the differences were not statistically significant, the Ex group initially oxidized less FAT (15%) during activity compared to the Con group (28%). The discrepancy can most likely be explained by the higher amount of body fat in the Ex group compared to the Con group. This difference is consistent with the findings of Pérez-Martin *et al.* (2001:466) who found significantly lower fat-oxidation levels in obese individuals compared to lean ones. After correcting for the pre-test differences between the groups, the Ex group oxidized a significantly ($p \leq 0.05$) larger percentage FAT (45%) than the Con group (42%) which may be primarily attributed to the loss in body fat in the Ex group, but also to the adaptation of the body to utilize intra-muscular triglycerides (Phillips *et al.,* 1996:2182). Fat oxidation during rest was more or less equal in both instances (pre-test and post-test), and did not indicate significant differences between the groups for either instance.

Other factors that may influence metabolic rate are age, body temperature, diet, stress, and circulating thyroxin (Johnstone *et al.*, 2005:941). Age is constant for the two groups, but body temperature, diet, stress and thyroxin have not been measured and can therefore not be excluded as contributing factors for the small metabolic variation between the groups.

The large standard deviations that sometimes exceeded the average values of fat and carbohydrate metabolism indicate considerable variability between individuals. This may explain the insignificant inter-group differences for fat and carbohydrate oxidation when significant differences were expected.

4.6.2 Intra-group differences

The body has the means to derive energy from the degradation of substances such as carbohydrates, fat or protein with or without the immediate use of oxygen (Brooks *et al.*, 2004:43). Oxygen uptake and carbon dioxide secretion were measured in order to calculate the respiratory exchange ratio (RER). RER will henceforth be reported with the percentage ratio of CHO and FAT oxidation in brackets, e.g. 0.79(39:69).

It should be noted that the origin from where the substrate (CHO or FAT) is utilized cannot be determined by means of indirect calorimetry. In other words whether CHO was utilized from the liver or from muscle tissue, cannot be depicted. Thus, a cumulative value for CHO and FAT is presented. Furthermore, values in the Lusk table (Lusk, 1924:41) cannot easily be interpolated. The reason for this is that the relationships between the RER and the percentage energy provided from glucose and fat oxidation, and the energy equivalent of oxygen, is not linear (Peronnet & Massicott, 1991:23). Lusk's table can be viewed in Appendix H.

a) Control group

(i) Metabolism at rest

At rest, no metabolic measure indicated any noteworthy change in the Con group after sustained sedentary behavior over a period of 12 weeks. Along with the 6% decrease in heart rate from 71±11 to 67±8 beats.min^{-1} (which was most likely due to the higher environmental temperature), the total metabolic activity at rest lessened. Oxygen uptake ($\dot{V}O_2$) decreased 3% from 3.9±2.1 to 3.8±1.3 ml.kg^{-1}.min^{-1}, FAT oxidation with 16% from 269±319 to 225±308 g.24h^{-1}, and CHO oxidation with 10% from 221±154 to 198±234 g.24h^{-1}. Reduction in these metabolic measures indicates less energy demand during rest than was measured initially. Even though these changes are small,

the involvement of some factor may have induced positive change. FAT yielded the highest value for substrate oxidation both before and after the period of 12 weeks. This indicates FAT to be the primary energy source during rest, as is to be expected in fasting state (Soeters *et al.,* 2009:589). Statistical significance ($p \leq 0.05$) was not evident in any of the changes, as was expected in a group that did not undergo any form of exercise intervention.

It is important to note the large standard deviations for substrate oxidation. The considerable variability between individuals in the group for substrate oxidation resulted in relatively large pre-test and post-test differences that did not indicate statistical significance. The large variability between subjects in the same group also stresses the individuality of energy metabolism.

(ii) Difference in metabolism between rest and activity

Exercise intensity can be expressed by various physiological and subjective measures. Oxygen uptake ($\dot{V}O_2$), and metabolic equivalents (METs) are among the physiological measured whereas the "rate of perceived exertion" scale by Borg (1974:131) is among the subjective measures. To clarify, $\dot{V}O_2$ is the peak amount of oxygen ($ml.kg^{-1}.min^{-1}$) the body requires to sustain energy demands at a certain time and is proportional to the intensity of exercise (ACSM, 2010:72). A metabolic equivalent (MET) is a unit that expresses oxygen cost at rest and is usually equal to 3.5 $ml.kg^{-1}.min^{-1}$ (ACSM, 2010:161). On the other hand, the "rate of perceived exertion" scale is a tool that numerically expresses the subjective intensity rating of a person. The tool is likert-wise scaled from 6 to 20 with 7 representing extremely light effort, and 19 representing extremely hard effort (Borg, 1974:131). Borg's scale can be viewed in Appendix I.

Whole-body vibration training has been identified as a special method of strength training and functions according to the second law of Newton (Van der Meer *et al.,* 2007:24). In order to increase the force exerted by the body, WBV manipulates acceleration, rather than mass, as is the case with conservative strength training. In conventional resistance training, muscle contractions are voluntary, whereas in WBV,

muscle contractions are involuntary and stretch-reflex induced. Currently literature favours energy expenditure in WBV when compared to conventional resistance training in terms of energy expenditure, the respiratory exchange ratio and perceived exertion (Da Silva *et al.*, 2007:473; Rittweger *et al.*, 2001:171).

On the contrary, heart rate is not significantly altered by WBV (Da Silva *et al.*, 2007:173; Rittweger *et al.*, 2001:171). During normal resistance-type exercise, heart rate increases in proportion to the muscle mass used and according to the percentage of maximum voluntary contraction (Pollock *et al.*, 2000:828). In the specific whole-body vibration test protocol of the current study, large muscle groups were targeted and exercise intensity relied to a large extent on the voluntary contraction of the participant's muscles. The more forcefully the participants contracted their muscles, the greater the vibration transmission to the muscles, and the more difficult the activity.

In the pre-test, the mean heart rate increased by 49% from rest (71±11) to activity (106±15 beats.min^{-1}) and reached 55% of age-predicted maximum heart rate. The mean heart rate values represent the average heart rate of the 20-min. protocol with combined upper- and lower-body exercises. In comparison to younger men (18.3±0.24 yrs.), WBV also caused an increase of 49% in heart rate and reached 50% of age-predicted maximal heart rate (Da Silva *et al.*, 2007:473). In the post-test, heart rate increased 63% from rest (67±8) beats.min^{-1} to activity (109±20 beats.min^{-1}) which is a larger difference than during the pre-test, however, not significant ($p<0.05$). This time the group reached 56% of their age-predicted maximum heart rate which was slightly higher than during the pre-test. The heart rate responses correlated well to what is found in isometric-type of resistance exercise (Pollock *et al.,* 2000:829).

Subjective rating of perceived exertion (RPE) in the group was 11 (light) in both instances (pre-test as well as post-test). When this sedentary group of men was compared to younger, physically active men participating in lower-body WBV, RPE was higher by 2 levels. Younger men rated their perceived exertion as 9 on Borg's rate of perceived exertion scale. It seems therefore that sedentary individuals experience WBV

harder than physically active, younger men. Also, the low subjective level of exertion may indicate that the group did not participate maximally in the voluntary contraction.

Metabolic demand only increased by 3.8 METs as derived from the change in $\dot{V}O_2$ from basal value (3.9±2.1 ml.kg^{-1}.min^{-1}) to activity (13.3±4.3 ml.kg^{-1}.min^{-1}). This increment is slightly lower than the 14.0±2.7 ml.kg^{-1}.min^{-1} that was reached by a heterogenous group of individuals (25.2 yrs.) during squatting (Rittweger *et al.*, 2001:170) and the 16.4±2.6 ml.kg^{-1}.min^{-1} that is reached by sedentary men participating in cycle ergometry up to the same level of exertion, namely RPE=11 (Faulkner *et al.,* 2007:401). This finding indicates that oxygen demand is almost similar during WBV of higher intensity (*f*=40 Hz and *A*=4 mm) compared to lower intensities (*f*=26 Hz, *A*=3 mm), but lower than during cycling for the same subjective intensity rating.

About the same increments of change were noted for these variables during the post-test. Oxygen uptake reached approximately the same levels as during the pre-test with a value of 3.8±1.3 ml.kg^{-1}.min^{-1} during rest and 12.4±2.3 ml.kg^{-1}.min^{-1} during activity. Energy demand of 3.5 METs was reached. These pre-test vs. post-test values are consistent with ongoing sedentary behavior.

Exercise intensity for the purpose of health promotion in sedentary individuals is recommended to equal sub-maximal levels of exertion. It entails 57-67% of age-predicted maximal heart rate (ACSM, 2010:166), 5-7 MET's (ACSM, 2010:175), and rate of perceived exertion of 11-13 (ACSM, 2010:166) for a period of 20-30 minutes on most days of the week. Whole-body vibration exercise of the required duration only reached 55% of age-predicted maximum heart rate, and half the metabolic equivalents. Perceived exertion reached 11 which is equal to a light intensity exercise, and only the lower end of the required intensity rating. These values indicate that the cardiovascular system was only minimally stressed.

In this whole-body vibration study, however, an interesting observation was made. Higher RER-values accompanied lower levels of oxygen uptake. This means that, even though the cardiovascular stress during exercise was low, the anaerobic capacity of the

muscle cells was challenged. The RER represents the ratio of oxygen (O_2) consumption and carbon dioxide (CO_2) secretion. When this ratio equals 1, carbohydrates are primarily utilized for energy supply, indicating that the exercise intensity is high. This was evident in the results for substrate oxidation.

The pre-test quantity of FAT halved from rest (269±319 $g.24h^{-1}$) to activity (124±58 $g.24h^{-1}$) where the average quantity of CHO increased 3.3 times from rest (221±154 $g.24h^{-1}$) to activity (730±430 $g.24h^{-1}$). The fat-to-carbohydrates-ratio, as derived from the RER, revealed the same tendency. RER increased from 0.79(39:69) at rest to 0.91(72:28) during activity. The post-test results were very similar to those of the pre-test. Once again, the main energy source changed from FAT at rest to CHO during activity. FAT decreased insignificantly by a quarter from 225±308 to 168±60 $g.24h^{-1}$, where CHO increased significantly ($p \leq 0.001$) from 189±324 to 484±220 $g.24h^{-1}$. Inter-group variability remained high as is evidenced by the large standard deviations. Da Silva *et al.* (2007:473) recorded RER-levels exceeding 1.2 during WBV; however, participants consumed a pre-exercise snack an hour before testing. Comparison is therefore biased.

The fat-to-carbohydrates-ratio, as derived from the RER, increased significantly, but this time revealed less CHO oxidation and more FAT oxidation. RER increased from 0.8(34.5:65.5) to 0.87(58.5:41.5). The quantity of oxidized FAT in activity was approximately similar in the pre- and post-tests, but CHO oxidation was much less. So, even though the tendency was the same for both instances, it seems that adaptation in CHO-oxidation was established during the 12 weeks. The reason for this is, however, unclear.

The results might be clarified by the type of muscle contraction that was induced during the exercise protocol. Absorption of oxygen into the muscles depends mainly upon two factors, namely the rate of diffusion of oxygen from the blood into the cells, and the capacity of the mitochondria to utilize oxygen (Brooks *et al.,* 2004:344). Mitochondrial integrity could not be measured, but the rate of diffusion of oxygen was manipulated. During isometric contractions, arteries are suppressed, allowing less blood flow to the

musculature and consequently, a limited delivery of oxygen and nutrients to the muscle cells. In this way the muscle cells are forced to utilize energy anaerobically from CHO or FAT stores, causing accumulation of metabolites and inducing a quicker fatigue rate (Katayama *et al.,* 2010:1269). In such cases, regular training on vibration platforms that equal the intensity of testing predict adaptation in favor of CHO utilization.

Note that the CHO value during activity was a cumulative average of the 20 min. activity period. It is therefore assumed that CHO oxidation remained higher than FAT oxidation throughout the 20 min. session. The ability of the sedentary participants to keep up the high intensity for longer than 10 min. is ascribed to the position change every 60 s. Exercises in the testing protocol alternated in such a way that different muscles were targeted at different times. Some exercises were repeated, but not consecutively, allowing phospho-creatine (PC) to regenerate in the muscle cells.

The minor variations between pre- and post-test values indicated no significant adaptation after 12 weeks without exercise intervention.

(iii) Metabolism during activity

When pre-test and post-test values during activity were compared, large differences were noted. Average $\dot{V}O_2$ decreased by 7% from 13.3±4.3 to 12.4±2.3 $ml.kg^{-1}.min^{-1}$. Average fat oxidation increased by 35% from 124±58 to 168±60 $g.24h^{-1}$, and average carbohydrate oxidation decreased by 34% from 730±430 to 484±220 $g.24h^{-1}$. A shift in the RER from 0.91(71.9:28.1) to 0.87(58.5:41.5) was noted, with carbohydrates remaining the primary energy source during activity. The decrease in carbohydrate oxidation during activity indicated statistical significance ($p \leq 0.01$), even though the percentage difference of average fat oxidation was higher.

Decrease in the RER during activity, accompanied by a large increase in fat oxidation and a large decrease in carbohydrate oxidation, point to noteworthy metabolic adaptation in favour of fat oxidation. This is in contrast to what was expected in the Con group as they neither participated in regular physical activity nor were they exposed to whole-body vibration training in particular during the 12-week intervention period. Food

intake before exercise is one of the factors that influence substrate oxidation. However, the participants adhered to a fasting period of 4 hours prior to testing. The possible influence of pre-testing food consumption is consequently eliminated. Some unrecorded stimulus is thought to be responsible for the findings.

b) Exercise group

(i) Metabolism at rest

Significant change ($p \leq 0.05$) in FAT and CHO oxidation was recorded in the Ex group during rest. FAT oxidation increased by 11% from 195±345 to 217±131 $g.24h^{-1}$ with an accompanying decrease in CHO oxidation of 54% from 402±326 to 187±198 $g.24h^{-1}$. The primary energy source changed from CHO in the pre-test to FAT in the post-test. These measures were in contrast to the RER-value of 0.82(41.4:58.6) that suggest FAT to be the main energy source at rest. This contrast may arise because the relationships between the RER and the percentage energy provided from glucose and fat oxidation, and the energy equivalent of oxygen are not linear (Peronnet & Massicott, 1991:23).

The differences in standard deviation of FAT and CHO between the pre-test and post-test are noteworthy. It seems that intra-group variability was larger for both variables in the pre-test compared to the post-test. This is in contrast to what was found in the Con group, as intra-group variability remained high in both testing sessions. Adaptation in favor of FAT oxidation was thus evident through these changes and was supported by the RER-value that decreased from 0.82(41:59) to 0.80(34.5:65.5).

The change in substrate oxidation holds positive prospects for overweight and obese individuals. Energy expenditure during rest constitutes about 75% of total energy expenditure in sedentary men (Segal *et al.*, 1984:999). If fat oxidation is elevated in this period, improvement in excess fat is predicted. WBVT has indeed improved excess fat of the Ex group, with a decrease of 0.7 kg, which is an 8% improvement from the initial value.

The other metabolic variables, namely oxygen uptake and heart rate were less affected by the training regime. Oxygen uptake decreased slightly from (4.5±1.8 $ml.kg^{-1}.min^{-1}$) to

(4.4±1.7 ml.kg^{-1}.min^{-1}) where heart rate increased by 1 beat.min^{-1}. These findings are comparable to what was found in the Con group.

(ii) Difference in metabolism between rest and activity

During the transition from rest to activity, the Ex group responded much the same as the Con group for both the pre- and post-tests. Significant change ($p \leq 0.05$) for $\dot{V}O_2$, CHO oxidation, RER and HR was induced by the activity, but no significant features were indicated regarding the change in FAT oxidation.

In the pre-test, heart rate increased by 63% from rest (70±8 beats.min^{-1}) to activity (114±20 beats.min^{-1}) and reached 60% of their age-predicted maximum heart rate. This increment of increase is higher than what was found in the Con group (49% increase) that reached 55% of age-predicted maximum heart rate, and in physically active younger individuals (18.3±0.24 yrs.) that achieved a 49% increase during lower-body WBV and reached 50% of age-predicted maximum heart rate (Da Silva *et al.,* 2007:473). In the pre-test the Ex group seems less fit than the Con group.

Metabolic demand reached 4.3 METs as oxygen uptake increased from rest (4.5±1.8 ml.kg^{-1}.min^{-1}) to activity (15.1±4.6 ml.kg^{-1}.min^{-1}) at a subjective intensity rating of 13 on Borg's RPE-scale. This value is higher than the 14.0±2.7 ml.kg^{-1}.min^{-1} that is reached by younger individuals during lower-body WBV (Rittweger *et al.,* 2001:171), but lower than the 21.7±3.7 ml.kg^{-1}.min^{-1} that is reached by sedentary men participating in cycle ergometry up to the same level of exertion (Faulkner *et al.,* 2007:401). Physically active individuals reach an RPE of 9 during WBV (Da Silva *et al.*, 2007:473) indicating that this sedentary group experienced WBV harder than physically active individuals. The intensity of WBV in the study of Da Silva *et al.* (2007:473) was, however, not stipulated.

Similar responses were noted in the post-test, but with indications of adaptation. Heart rate increased only by 55% from rest (72±8) to activity (112±18 beats.min^{-1}) and reached 59% of age-predicted maximal heart rate which was less than during the pre-test. Metabolic demand was also less in the post-test with reduced increase in $\dot{V}O_2$ from rest (4.4±1.7) to activity (12.4±4 ml.kg^{-1}.min^{-1}) and accompanying measure of 3.5

METs. The subjective rating decreased to 11 on Borg's perceived exertion scale, indicating "moderate".

Even though the induced stress on the group's metabolism was equally significant in both testing sessions, the Ex group proved that they had adapted to the stimulus. With the same exercise stimulus, physiological and subjective intensity ratings were lower. For the purpose of health promotion, intensity measures were still too low, rendering WBVT an unviable option for improving cardiovascular fitness.

Whilst the intensity of the activity seems too low to gain health benefits, other variables indicated favorable change. In the pre-test, the Ex group generated energy mainly from CHO, both at rest and during activity, where FAT decreased slightly from rest to activity. CHO increased significantly ($p \leq 0.05$) by 1.5 times from 402±326 to 991±451 $g.24h^{-1}$. This is a smaller increment of increase in comparison to the 3.3 times of the Con group. FAT decreased insignificantly by a quarter from 195±345 to 145±94 $g.24h^{-1}$ which was less than the decrease by a half that was prevalent in the Con group. RER-value increased significantly ($p \leq 0.001$) from 0.82(41.4:58.6) to 0.95(85.1:14.9) underlining the high intensity of the activity. The large standard deviations yet again stress the individuality of metabolism. In younger individuals (18.3±0.24 yrs.) participating in lower body WBV, an RER-value exceeding 1.2 was reached (Da Silva *et al.*, 2007:471). However, these individuals consumed a pre-exercise snack one hour before testing, making comparison to the Ex group of the current study inaccurate.

In the post-test, the main energy supply changed from FAT during rest to CHO during activity. As was expected, significant increases in RER and CHO oxidation were seen ($p \leq 0.05$) in the transition from rest to activity. However, an insignificant increase in FAT oxidation was also prevalent. The increase in both FAT and CHO was contradictory because when oxidation of either energy source increases, a decrease is expected in the other. These energy sources do not compete for energy supply to the body, but rather function in synchronization. It was thus expected that during high intensity exercise, CHO oxidation will gradually increase and FAT oxidation will gradually decrease. In the post-test, CHO increased 3 times from 187±198 to 557±254 $g.24h^{-1}$

accompanied by a 2% increase in the average FAT oxidation from 217±131 to 221±93 $g.24h^{-1}$.

On the other hand, RER proved what was expected during activity, namely an increase in CHO oxidation and a decrease in fat oxidation. RER increased from 0.80(34.5:65.5) to 0.86(55.1:44.9) in the transition from rest to exercise which revealed a 7.5% increment of change. In the pre-test, the increment of increase was 16%, indicating adaptation in favor of FAT oxidation.

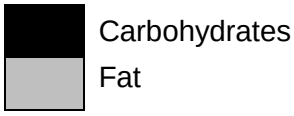
Response of the Ex group to this type of activity (whole-body vibration at *f*=40 Hz; *A*=4-6 mm, *t*=20 min) revealed the same trend as that found in the Con group. Therefore the possible explanatory causes that were applicable in the Con group, also apply to the Ex group. Firstly, the exercises were executed in 60 s bouts of active isometric contractions, thus restricting blood flow to the active muscles and forcing the cells to generate energy anaerobically from mainly CHO stores. When the exercise position changed to target a new set of muscles, blood flow and oxygen delivery is allowed back into the musculature for energy stores to be refueled. The cycle then repeats itself for the next 60 s bout of activity. Secondly, the reported values for substrate oxidation during activity are average values for the 20 min. period, suggesting that the intensity of the activity remained high throughout the testing period. Possibly the sedentary participants could keep up with the high intensity activity for longer than 10 minutes due to position changes every 60 s.

Repetitive exposure to this type of training predicts adaptation in favor of CHO utilization during exercise. But the contrary was evident. The ratio of CHO to FAT oxidation changed in favor of FAT oxidation both during rest and activity. A possible explanation for the adaptation might be that hormones such as glucagon, growth hormone, and epinephrine are secreted in response to high intensity exercise (Felsing *et al.,* 1992:157). In turn, these hormones stimulate lipolysis that may result in increased circulating triglycerides, which was evident in the Ex group, as circulating triglycerides increased by 36%. Higher quantities of circulating triglycerides incline the body to use fat over carbohydrate oxidation.

(iii) Metabolism during activity

When pre-test and post-test values during activity were compared, significant improvements ($p \leq 0.05$) were noted in $\dot{V}O_2$ and CHO oxidation, FAT oxidation and RER. Oxygen uptake decreased by 18% from 15.1±4.6 to 12.4±4.0 $ml.kg^{-1}.min^{-1}$. Average fat oxidation increased by 34% from 145±94 to 221±93 $g.24h^{-1}$, and average carbohydrate oxidation decreased by 44% from 991±451 to 557±254 $g.24h^{-1}$. A shift in the RER from 0.95(85.1:14.9) to 0.86(55.1:44.9) was noted, with carbohydrates remaining the primary energy source during activity. Heart rate did not adapt significantly from pre-test (114±20) to post-test (112±18 $beats.min^{-1}$).

One incident of exhaustion was documented during activity. In the pre-test, a participant collapsed from muscle fatigue after the third lower-body exercise. At the time his heart rate was 143 $beats.min^{-1}$, blood pressure 118/80 mmHg, RER-value 1.6, oxygen uptake 10.7 $ml.kg^{-1}.min^{-1}$, FAT oxidation zero, and CHO oxidation 2227 $g.24h^{-1}$. He reported the intensity of the exercise as 18 on the “rate-of-perceived-exertion” scale. The participant was able to resume activity after 30 s of rest in a seated position, and completed the test. After the intervention of 12 weeks, the same participant was able to complete the test uninterrupted. At the same time during the test in which the incident had previously occurred, his heart rate was 101 $beats.min^{-1}$, blood pressure146/86 mmHg, RER-value 0.72, oxygen uptake 6.6 $ml.kg^{-1}.min^{-1}$, FAT oxidation 473 $g.24h^{-1}$, and CHO oxidation 94 $g.24h^{-1}$. This time he reported the difficulty of the exercise as 13 on the “rate-of-perceived-exertion” scale. This participant also shed 3.5 kg of his excess fat in the 12-week intervention period.



Exercise group

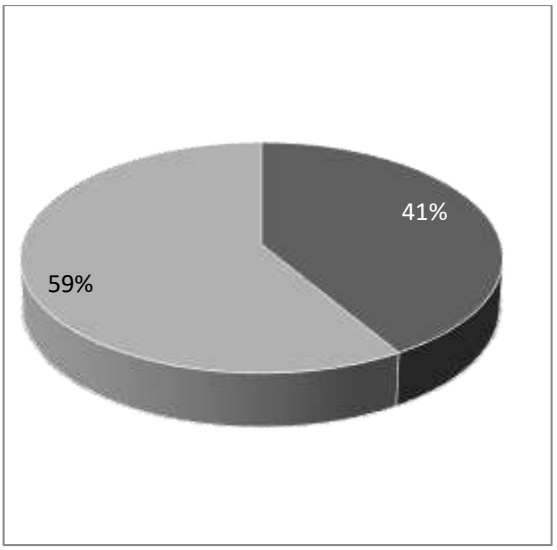


Figure 4.1: Mean substrate oxidation at rest before intervention

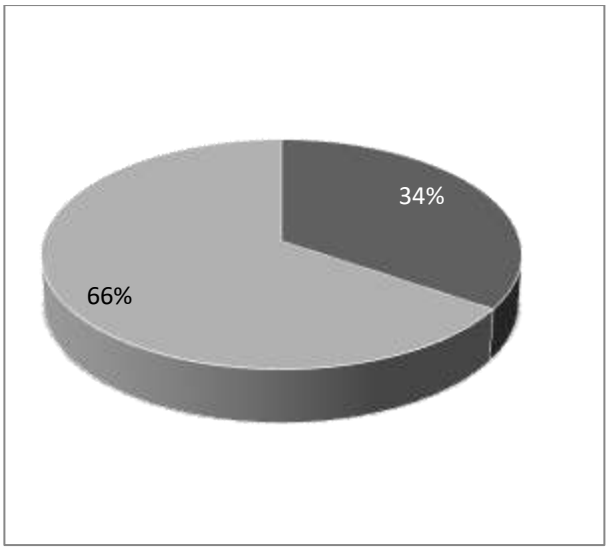


Figure 4.2: Mean substrate oxidation at rest after intervention

Control group

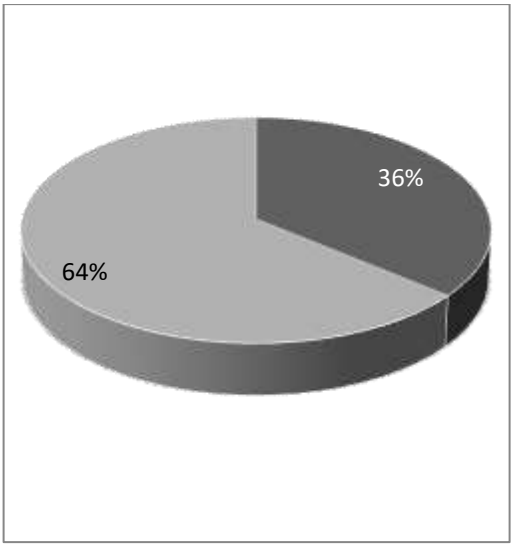


Figure 4.3: Mean substrate oxidation at rest before intervention

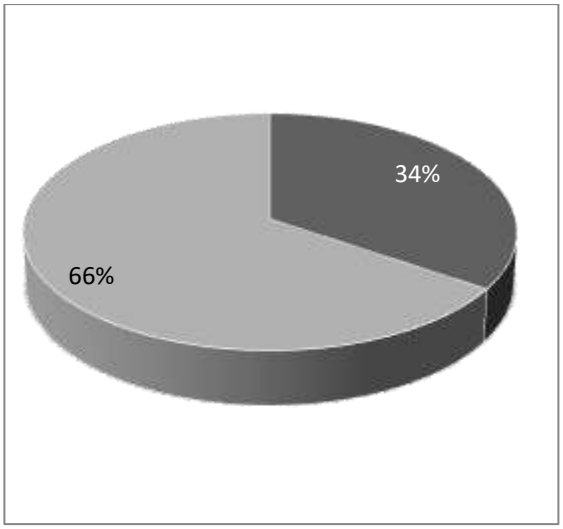
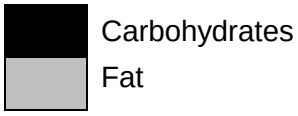


Figure 4.4: Mean substrate oxidation at rest after intervention



Exercise group

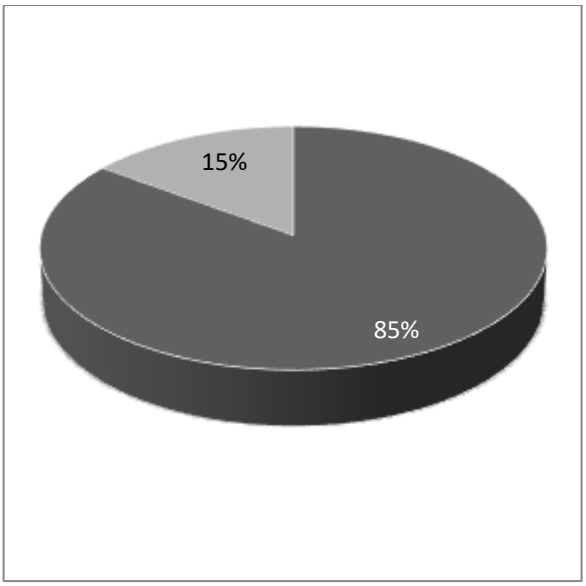


Figure 4.5: Mean substrate oxidation during activity, before intervention

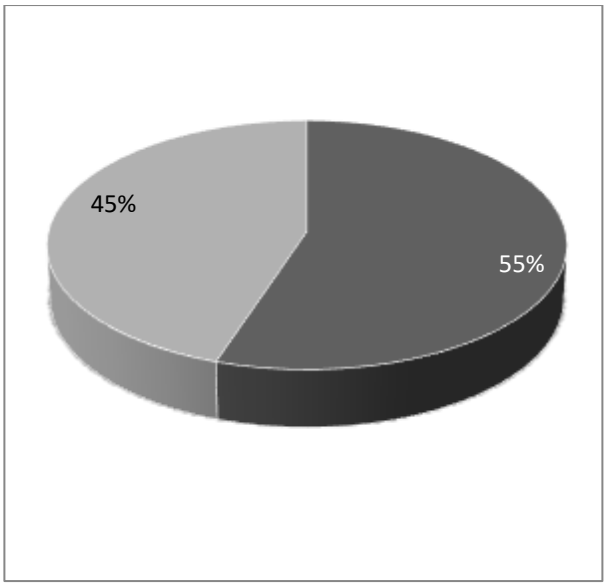


Figure 4.6: Mean substrate oxidation during activity, after intervention

Control group

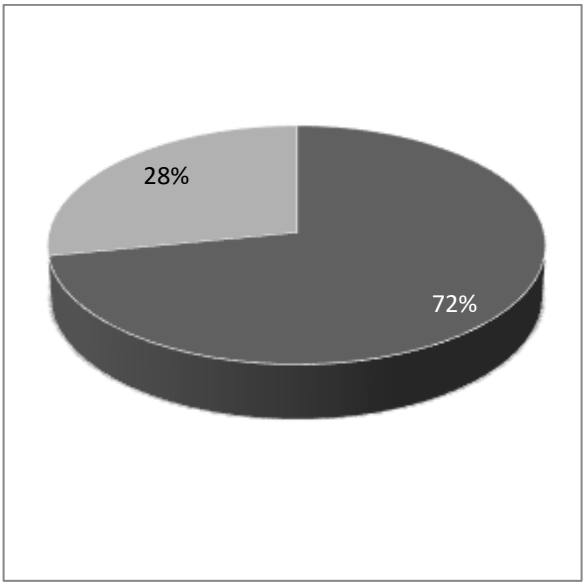


Figure 4.7: Mean substrate oxidation during activity, before intervention

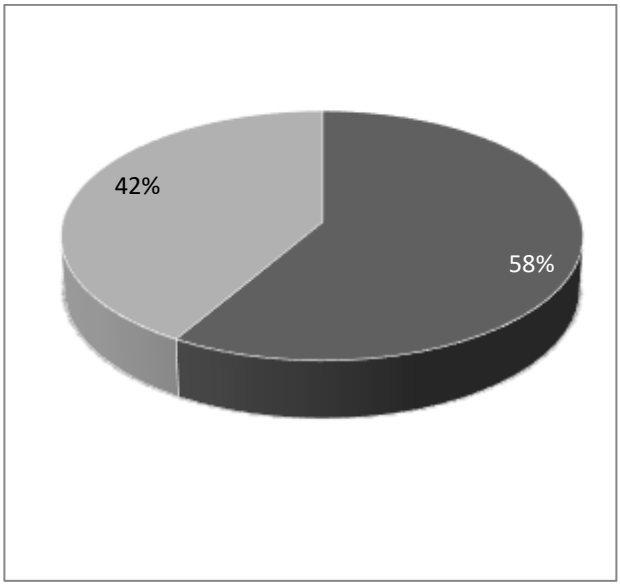


Figure 4.8: Mean substrate oxidation during activity, after intervention

4.7 Summary of findings

The Ex and Con groups were significantly different in a number of variables initially. In the body composition category the Ex group was taller and heavier than the Con group, with more body fat, a larger waist circumference, and more fat free mass. Lipid profile measurements revealed higher low density lipoprotein cholesterol, and, on the positive side, higher high density lipoprotein cholesterol. Furthermore, systolic blood pressure was higher and the left ventricle chamber less compliant than that of the Con group. Conversely, the Con group presented a significantly higher ($p \leq 0.05$) stroke volume than the Ex group, which was positive. Total risk for the development of cardiovascular disease was consequently higher in the Ex group than the Con group. When initial inter-group differences were corrected for statistically, WBVT (t=30-60 s; f=30-40 Hz; A=3-6 mm) proved to be beneficial in improving systolic blood pressure, metabolic demand of the heart, end-diastolic volume, stroke volume, and fat oxidation.

The Con group persisted in sedentary behavior for the 12-week intervention period and showed significant changes in haemodynamics and metabolism. Unfavorable decreases in ejection fraction and shortening fraction of the myocardial tissue were found, which are consistent with sedentary behavior. Beneficial change in favor of fat oxidation was also noted during the whole-body vibration exercise test. This finding contrasts with those relating to sedentary behavior.

After 12 weeks of progressive whole-body vibration training, the Ex group gained overall benefit in the 4 tested categories (body composition, cardiovascular function, blood lipids and glucose, and metabolism). The total of 44 variables yielded different levels of significance, therefore the overall benefit could not be expressed in a single p-value. Reduction in the group's risk for developing cardiovascular disease was accomplished. The group improved in body weight, fat mass, and waist circumference, but showed a decrease in fat free mass. The decrease in fat free mass was in contrast to the current available literature and attributed to the small sample size. Blood pressure and double product dropped significantly, and chamber compliance, end-diastolic volume and stroke volume increased. In addition to the cardiovascular benefits, total cholesterol

and low density lipoprotein cholesterol were significantly reduced. In the metabolism category, oxygen uptake improved, and substrate oxidation changed in favor of fat-oxidation.

A summary of the findings is presented in Table 4.15.

Table 4.15: Summary of the study's findings for both groups (Ex and Con) in all investigated categories

Variable	Inter-group difference		Intra-group differences					
			Exercise group			Control group		
	Pre-test	Post-test	Change	Beneficial (+) Detrimental (-) Constant (c)	Level of significance	Change	Beneficial (+) Detrimental (-) Constant (c)	Level of significance
DEMOGRAPHICS								
Age	0.76	-	0	C	n.s	0	C	n.s
Stature	0.0008**	0.54	0	C	n.s	0	C	n.s
BODY COMPOSITION								
Body weight	0.0004**	0.08	↓	+	n.s	↑	-	n.s
BMI	0.008*	<0.0001**	↓	+	n.s	↑	-	n.s
% BF	0.0002**	0.14	↓	+	n.s	↑	-	n.s
FFM	0.39	0.009*	↓	-	n.s	↓	-	n.s
Excess fat	0.013*	0.11	↓	+	n.s	↑	-	n.s
Cir. Waist	<0.0001**	0.12	↓	+	*	↓	+	n.s
WHR	0.49	0.13	↓	+	n.s	↓	+	n.s
CARDIOVASCULAR								
SBP	0.05*	0.005*	↓	+	*	↓	+	n.s
DBP	0.21	0.06	↓	+	*	↑	-	n.s
HR	0.74	0.07	↑	-	n.s	↓	+	n.s
Double Product	0.56	0.04*	↓	+	*	↓	-	n.s
End-diast.vol	0.04*	0.003*	↑	+	*	↓	-	n.s
End-syst.vol	0.02*	0.032*	↑	-	n.s	↓	+	n.s
Stroke volume	0.0001**	0.077	↑	+	*	↑	+	n.s
Ejection Fraction	0.098	0.45	↑	+	n.s	↓	-	*
Shortening Fraction	0.12	0.35	↑	+	n.s	↓	-	*
Time Velocity Integral	0.76	0.75	0	C	n.s	0	C	n.s
Deceleration time	0.04*	0.6	↓	+	n.s	↑	-	n.s
E	0.97	0.06	↓	+	n.s	0	C	n.s
A	0.32	0.64	0	C	n.s	↓	+	n.s
E'SEP	0.54	0.37	0	C	n.s	0	C	n.s
A'SEP	0.14	0.33	0	C	n.s	0	C	n.s

Level of significance: * p≤0.05; **p≤0.001; n.s. = not significant

Change: 0 = no change; ↑ = increase; ↓ = decrease

Abbreviations: BMI = body mass index; %BF = percentage body fat; FFM = fat free mass; Cir.Waist = waist circumference; WHR = waist-to-hip ratio; SBP = systolic blood pressure; DBP = diastolic blood pressure; HR = heart rate; End-diast.vol = end-diastolic volume; End-syst.vol = end-systolic volume; E = Trans-mitral velocity during passive relaxation; A = Trans-mitral velocity during active relaxation; E'SEP = Peak cell velocity during passive relaxation at the septum

Table 4.15 Continued…

Variable	Inter-group differences		Intra-group differences					
			Exercise group			Control group		
	Pre-test	Post-test	Change	Beneficial (+) Detrimental (-) Constant (c)	Level of significance	Change	Beneficial (+) Detrimental (-) Constant (c)	Level of significance
S'SEP	0.04*	0.2	0	C	n.s	0	C	n.s
E'LAT	0.02*	0.8	↓	+	n.s	0	C	n.s
A'SEP	0.14	0.33	0	C	n.s	0	C	n.s
S'SEP	0.04*	0.2	0	C	n.s	0	C	n.s
E'LAT	0.02*	0.8	↓	+	n.s	0	C	n.s
A'LAT	0.19	0.12	↑	-	n.s	↑	-	n.s
S'LAT	0.15	0.34	↑	-	n.s	0	-	n.s
LIPIDS AND GLUCOSE								
TC	0.066	0.71	↓	+	*	↑	-	n.s
LDL	0.012*	0.62	↓	+	*	↓	+	n.s
HDL	0.006*	0.28	0	C	n.s	↓	-	n.s
Triglycerides	0.72	0.32	↑	-	n.s	↑	-	n.s
Glucose	0.34	0.69	0	C	n.s	0	C	n.s
METABOLISM								
$\dot{V}O_2$ rest	0.56	0.17	↓	+	n.s	↓	+	n.s
$\dot{V}O_2$ activity	0.44	0.79	↓	+	**	↓	+	n.s
Fat rest	0.129	0.92	↑	+	*	↓	-	n.s
Fat activity	0.924	0.047	↑	+	*	↓	-	n.s
CHO rest	0.095	0.12	↓	+	*	↓	+	n.s
CHO activity	0.08	0.34	↓	+	**	↓	+	*
RER rest	0.547	0.98	↓	+	n.s	↑	-	n.s
RER activity	0.279	0.49	↓	+	**	↓	+	*
HR rest	0.742	0.067	↑	-	n.s	↓	+	n.s
HR activity	0.080	0.69	↓	+	n.s	↑	-	n.s

Level of significance: * p≤0.05; **p≤0.001; n.s. = not significant

Change: 0 = no change; ↑ = increase; ↓ = decrease

Abbreviations:
S'SEP = Peak cell velocity during systole at the septum; E'LAT = Peak cell velocity during passive relaxation at the lateral wall of the left ventricle; A'LAT = Peak cell velocity during active relaxation at the lateral wall of the left ventricle; S'LAT = peak cell velocity during systole at the lateral wall of the ventricle TC = total cholesterol; LDL = low density lipoprotein cholesterol; HDL = high density lipoprotein cholesterol; $\dot{V}O_2$ = peak oxygen uptake; CHO = carbohydrates, RER = respiratory exchange ratio; HR = heart rate

4.8 References

ACSM see AMERICAN COLLEGE OF SPORTS MEDICINE

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5

Summary, conclusions and recommendations for future research

5.1 Summary

Biokineticists frequently treat patients at risk for developing cardiovascular disease (CVD) and those with established cardiovascular disease. Research indicates that aerobic and resistance training aid in both primary (initial event) and secondary (another event) prevention of cardiovascular events (Blair, 2009:1; Fagard & Cornelissen, 2007:12; Tsuzuku *et al.*, 2007:549; Jakčić *et al.*, 2003:1323; Whelton *et al.*, 2002:493). Exercise is consequently an integral part of the treatment of CVD-risk patients.

With the escalating popularity of whole-body vibration as a modality of training (Signorile, 2008:20), increasing numbers of health-related questions arise, several of which research has not found answers to yet. Some of the areas include variables associated with cardiovascular disease risk such as blood pressure, blood lipids, blood glucose, body composition, and cardiac function. Since biokineticists deal with these variables in patients on a regular basis, clarity is essential. The purpose of the study was to investigate the effect of progressive whole-body vibration training on variables associated with CVD risk (Chapter 1).

Chapter 2 dealt with whole-body vibration as training modality and was divided into two main parts. The first part addressed the principles, mechanism, terminology and safety considerations of whole-body vibration, whereas the second part discussed the effect of

whole-body vibration training on selected physical (body composition), hemodynamic (blood pressure and cardiac function), physiological and biological (blood lipids, blood glucose, and metabolism) parameters that are associated with cardiovascular disease risk. The majority of WBVT-studies only investigated acute responses, most likely due to the time-consuming demand of intervention-based research designs. Only a few intervention-based studies reported improvements in body composition and resting blood pressure, and hold promising possibilities for glucose control for those who participate in WBVT for 6 weeks or longer. Mechanisms that may explain the findings are, at this stage, speculative. Due to the scarcity of literature in these areas, evidence from conventional training modalities such as aerobic and resistance exercise was added. Blood lipids, metabolism and cardiac performance have been recognized as neglected areas in the WBVT continuum, which stresses the importance of the research study.

In Chapter 3 the equipment used during testing as well as the procedures of testing and intervention were explained. The method of data collection and statistical analysis was also addressed.

The results of the investigation and discussion thereof were included in Chapter 4. Results of each category of variables, which were obtained over a period of 12 weeks, were presented in tables and discussed accordingly. For the sake of consistency, each section included a discussion of the relevant data under two headings, namely, inter-group differences (differences between the two groups), followed by the intra-group differences (differences within the same group) at the pre- and post-testing occasions. A summary of the main findings concluded the chapter.

5.2 Conclusions

From the results of the investigation, the following conclusions can be drawn:

Hypothesis 1: Body composition will improve significantly.

Whole-body vibration training of a progressive nature improved the following body-composition variables:

- Body weight
- Body-mass index
- Percentage body fat
- Excess fat
- Waist circumference, and
- Waist-to-hip-ratio

Fat-free mass, however, did not improve.

Twelve weeks of progressive WBVT seems to be an effective intervention strategy for improving the body composition of sedentary young men. However, it is not sufficient to improve fat-free mass.

Hypothesis 1 is partially accepted.

Hypothesis 2: Systolic and diastolic blood pressure will improve significantly.

Systolic and diastolic blood pressure improved after 12 weeks of progressive whole-body vibration training.

As a result of the intervention, blood pressure was effectively lowered by progressive WBVT over 12 weeks. Furthermore, systolic blood pressure seems to be more sensitive to WBVT than diastolic blood pressure as the decrease in systolic blood pressure was statistically significant, and larger than what was found in diastolic pressure. Diastolic blood pressure did not improve significantly.

Hypothesis 2 is partially accepted.

Hypothesis 3: Blood lipids will improve significantly.

Blood lipids improved significantly in terms of the following variables:

- Total cholesterol, and
- Low-density lipoprotein cholesterol

The findings are positive seeing that initial total cholesterol levels were desirable and low-density lipoprotein cholesterol was near optimal. Other blood lipid variables,

namely high-density lipoprotein cholesterol and triglycerides, did not improve. The initial HDL-level was lower than normal and was expected to increase as a result of training, whereas the triglyceride level was normal and likely to decrease. This was not, in fact, the case, as high-density lipoprotein cholesterol remained constant, indicating neither improvement nor detriment, whereas triglycerides increased, indicating a possible negative trend.

Progressive whole-body vibration training seems to significantly improve the two major blood lipid components that increase an individual's risk for developing cardiovascular disease. This result is of particular value seeing that initial total cholesterol and low-density lipoprotein cholesterol values in this population of sedentary men were normal.

Hypothesis 3 is partially accepted.

Hypothesis 4: Blood glucose will improve significantly.

Blood glucose remained constant.

Whole-body vibration training of a progressive nature seems not to affect the fasting blood glucose of sedentary men. However, the optimal initial blood glucose level of the group might be the reason for the finding.

Hypothesis 4 is rejected.

Hypothesis 5: Cardiac function will improve significantly.

Cardiac function improved significantly in terms of the following variables:

- Double product
- End-diastolic volume, and
- Stroke volume

Other cardiac function variables that improved, but did not indicate significance, were:

- Ejection fraction
- Shortening fraction
- Deceleration time

- Myocardial cell velocities at lateral regions of the left ventricle during active filling and systole

Variables that remained more or less constant after the 12-week intervention period were the following:

- Time velocity integral
- Myocardial cell velocities at septal regions of the left ventricle during passive and active filling, as well as during systole (E'sep, A'sep, S'sep)
- Active filling velocity

Variables that were negatively affected by progressive WBVT were as follows:

- Passive filling velocity
- Myocardial cell velocity at lateral regions of the left ventricle during passive filling

Reduced filling velocities (velocity of blood flow through the mitral valve) and slower cell velocities (velocity of cell lengthening during passive ventricular filling) indicate less effective ventricular contractility according to the Frank-Starling mechanism. These findings are negative.

It appears that 12 weeks of progressive whole-body vibration training improves overall cardiac function in sedentary men. The two variables that were negatively affected did not carry any statistical significance and consequently did not counteract the improvements gained.

Hypothesis 5 is partially accepted.

Hypothesis 6.1: Oxygen uptake ($\dot{V}O_2$) will remain unchanged during rest (pre-test vs. post-test) and decrease during activity (pre-test vs. post-test).

Oxygen uptake ($\dot{V}O_2$) at rest decreased insignificantly (pre-test vs. post-test), whereas oxygen uptake during activity decreased significantly (pre-test vs. post-test)

Hypothesis 6.1 is partially accepted.

Hypothesis 6.2: Fat oxidation will decrease during rest (pre-test vs. post-test) and activity (pre-test vs. post-test).

Fat oxidation increased significantly both at rest (pre-test vs. post-test), and during activity (pre-test vs. post-test).

Hypothesis 6.2 is rejected.

Hypothesis 6.3: Carbohydrate oxidation will increase during rest (pre-test vs. post-test) and activity (pre-test vs. post-test).

Carbohydrate oxidation decreased significantly at rest (pre-test vs. post-test) as well as during activity (pre-test vs. post-test).

Hypothesis 6.3 is rejected.

Hypothesis 6.4: Respiratory exchange ratio will remain constant during rest (pre-test vs. post-test) and decrease during activity (pre-test vs. post-test).

The respiratory exchange ratio (RER) decreased insignificantly at rest (pre-test vs. post-test), and significantly during activity (pre-test vs. post-test).

Hypothesis 6.4 is partially accepted.

Hypothesis 6.5: Heart rate will decrease during rest (pre-test vs. post-test) as well as during activity (pre-test vs. post-test).

Heart rate increased insignificantly at rest (pre-test vs. post-test) and decreased insignificantly during activity (pre-test vs. post-test).

Hypothesis 6.5 is partially accepted.

5.3 Recommendations for future research

During the course of the study, certain problems, questions, and uncertainties became apparent. These have been identified as possible areas for investigation in future.

5.3.1 A health-related WBVT-testing protocol in risk patients is lacking. Such a protocol, which will effectively test an individual's tolerance to WBV and consequently his/her safety during exercise, is especially necessary for health professionals and exercise specialists who recommend and prescribe this unique type of training. Research regarding health-related testing-protocol design is recommended.

5.3.2 In this study, a group of men with low risk for developing cardiovascular disease was investigated. Individuals with higher risk for CVD might respond differently to WBVT than the current group. More research is recommended in higher-risk populations. These may include older populations, obese, hypertensive, and/or smoking patients with established dislipidaemia, a positive family history of coronary artery disease and those suffering from stress.

5.3.3 The current intervention program consisted of strengthening exercises for the upper- and lower body, flexibility, massaging and relaxation techniques that were manipulated in progressive fashion in terms of frequency, time, rest periods and number of exercises. In order to optimize health benefits, research is needed as to which exercises contribute best to health promotion, with the accompanying frequency, amplitude, type of muscle contraction and time of exposure.

5.3.4 Whole-body vibration is a popular training modality in female circles, most likely due to its cosmetic properties. Equivalent research regarding health-promotion is recommended in female populations.

5.3.5 The mechanical effect of tri-directional vibration on smaller arteries is a matter of concern, but still only a theoretic proposition. Evidence is required to clarify the matter.

5.3.6 WBV is considered to be a special method of strength training. In many health-related studies combined-type training (aerobic training in conjunction with resistance training) is recommended. Whether WBVT can replace, or even surpass, the role of resistance training is yet to be investigated.

5.4 References

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Appendix A: Selection questionnaire

Please answer ***all*** questions below in order to be considered as a participant in this research study. Fax through to: 051 401 9243 and mark for attention Berna de Kock or email to: dekockb@ufs.ac.za.

Name & Surname:_____ **Date**:_____

Tel: _____ **Email:** _____

Age:____**years** **Weight:**_____**kg** **Height:**_____**(m)**

I would like to be in the (underline): *Experimental group exercising for 12 weeks* **OR** *control group undergoing only the before-and-after testing.*

1. ***Mark/circle*** the number for each category (intensity, duration and frequency) that best describes your level of activity.
   To better understand the table, ask yourself: When/if I exercise, I feel (intensity) for how long (duration) how many times per week/month (frequency).

Physical-activity Index

Intensity	Not tired	1
	A little tired	2
	Tired	3
	Very tired	4
	Exhausted	5
Duration	<10 min	1
	10-19 min	2
	20-30 min	3
	>30 min	4
Frequency	1 x per month	1
	Few times per month	2
	1-2 times per week	3
	3-5 times per week	4
	Every day	5

2. The following questions consider your general health and your readiness for physical activity. Please complete the questionnaire.

Physical Activity Readiness Questionnaire (PAR-Q)

	Question	Yes	No
1	Has your doctor ever said you have heart trouble? If yes, specify:		
2	Do you frequently have pains in your heart and chest?		
3	Do you often feel faint or have spells of severe dizziness?		
4	Has a doctor ever told you that your blood pressure was too high?		
5	Do you smoke? If yes, specify:		
6	Does your mother or father suffer from any of the following conditions: Heart disease, stroke, obesity, diabetes mellitus, high blood pressure, high cholesterol levels? If so, specify:		
7	Are you suffering from insulin resistance or diabetes mellitus?		
8	Do you have a high blood cholesterol level?		
9	Are you currently taking any medication? If yes, specify:		
10	Do you currently have a disability or a communicable disease? If yes, specify:		
11	Do you suffer from any problems of the lower back, i.e., chronic pain or numbness?		
12	Has your doctor ever told you that you have a bone or joint problem that can be aggravated by exercise? Specify:		
13	Do you see yourself as a person who stresses easily and often? Explain:		
14	Is there any reason, not mentioned here, why you should not follow an activity program even if you wanted to?		

3. Please tick the condition(s) that are applicable to you

Wearing a pacemaker		Hip- and knee replacements	
Acute thrombosis conditions		Acute hernia, dicopathy, spondylolysis	
Fresh wounds resulting from an operation or surgical intervention		Wearing recently fitted coils, metal pins, bolts or plates	
Cancer		Taking anabolic steroids	
Heavy migraine		Taking performance enhancing agents	
Osteoporosis in an advanced stage (<-3.5)		Epilepsy	

I, _____ declare that the above information (p.1-3) is accurate and correct, and that I have not omitted any relevant information. I understand that the questionnaire is aimed at protecting my health and that the information will be treated confidentially.

_____ _____

Signature Date

Contact the researcher at
Fax: 051 401 9243
Tel: 051 401 3395
E-mail: dekockb@ufs.ac.za

Appendix B: Data sheet session 1

Participant number:_____ Date:_____

GENERAL

Stretch Stature (cm)		Body weight (kg)	
Heart rate (beats/min)		Blood pressure (mmHg)	

LIPOGRAM AND GLUCOSE

Total Cholesterol (mmol/L)	
HDL (mmol/L)	
LDL (mmol/L)	
Triglyccerides (mmol/L)	
Glucose (mmol/L)	

Station Manager General

Station Manager Lipogram & Glucose

CARDIAC ULTRA SOUND

Systolic volume (ml)		A	
Diastolic volume (ml)		E'sep	
Ejection fraction (%)		A'sep	
Shortening fraction (%)		S'sep	
Time-velocity integral (TVI) ($cm.s^{-1}$)		E'lat	
Deceleration time ($cm.s^{-1}$)		A'lat	
E		S'lat	

Station Manager Cardiac Morphology

Appendix C: Data sheet for body composition

Subject number:		Date:	
Measuring side:	Right / Left	Time:	
Stretch Stature (cm)		Weight (kg)	

Skinfolds (mm)				**Circumferences (cm)**			
	Meas1	Meas2	Meas3		Meas1	Meas2	Meas3
Triceps				Chest			
Subscapular				Waist			
Biceps				Umbilicus			
Chest				Hip			
Iliac crest				Thigh			
Supra iliac				Knee			
Umbilicus				Calf			
Mid Thigh				Arm flex			
Calf							

Bone breadths

	Meas1	Meas2	Meas3
Humerus			
Femur			
Bi-iliac crest			

Station Manager
Body Composition

Appendix D: Data sheet for whole-body vibration exercise test

Participant number:_____ Date:_____

Lower-body exercise						
	Resting	After 3rd exercise	After 6th exercise	After 10th exercise	1 min rest	3 min rest
BP (mmHg)						
HR (beats.min^{-1})						
RER						
VO_2 (ml.kg^{-1}.min^{-1})						
Fat (g.24h^{-1})						
Prot (g.24h^{-1})						
CHO (g.24h^{-1})						

Upper-body exercise						
	Resting	After 3rd exercise	After 6th exercise	After 10th exercise	1 min rest	3 min rest
BP (mmHg)						
HR (beats.min^{-1})						
RER						
VO_2 (ml.kg^{-1}.min^{-1})						
Fat (g.24h^{-1})						
Prot (g.24h^{-1})						
CHO (g.24h^{-1})						

Appendix E: Illustration of exercise test protocol

LOWER BODY		
Nu	**Exercise**	**Execution technique**
A01		**Squat** - Start by standing with correct posture - Bend legs only slightly - Bum slightly backwards and upper body straight up - Raise heels slightly for comfort if necessary - Touch handlebars lightly for balance only if necessary **Pre-tension** - Calves, thighs, abdominals, back
A02		**Deep Squat** - Start by standing with correct posture - Bend legs naturally to halfway position - Push buttocks slightly backwards - Keep knees above toes - Look up to keep upper body as straight as possible - Raise heels slightly for comfort if necessary - Touch handlebars lightly for balance only if necessary **Pre-tension** - Thigh (quadriceps, hamstrings); buttocks (gluteal complex)
A03		**Wide Stance Squat** - Start by standing with correct posture - Feet wide apart, pointing out at 45º - Bend legs naturally to halfway position – point knees in direction of feet - Push buttocks slightly backwards - Keep knees above toes - Look up to keep upper body as straight as possible - Raise heels slightly for comfort if necessary - Touch handlebars lightly for balance only if necessary **Pre-tension** - Inner thigh (adductors) – squeeze knees and feet together without actually moving them - Thigh (quadriceps and hamstrings) - Gluteal complex
A05		**Calve raise** - Legs straight, but soft knees – perfect posture - Lift heels until calf feels good contraction - Keep weight evenly distributed over all toes **Pre-tension** - Calves (gastrocnemius/soleus complex)

A06		**Pelvic bridge** - Lie on back on bench with hands on side of bench - Feet in centre of plate and hip width apart - Knees bent at 90º - Raise hips halfway up, keeping back in neutral - Toes lifted up, dig heels into plate and pull feet towards you. **Pre-tension** - Hamstrings, buttocks, and lower back
A04		**Lunge** - Start on floor standing with perfect posture - Take a big step forward onto the middle of the platform - Bend legs until front leg reaches a 90º angle. Point knees in direction of feet. Lift the heel of your back foot. - Keep front knee at a line behind the toes and distribute weight evenly over both feet - Push front foot into the platform - Look up to keep upper body as straight as possible - Raise heels slightly for comfort if necessary - Touch handlebars lightly for balance only if necessary **Pre-tension** - Gluteal complex - Thigh (quadriceps and hamstrings)

	UPPER BODY	
A08		**Push-Up** - In front of plate, knees on floor, hands at shoulder width on platform (on rubber mat) while maintaining perfect posture (shoulders, hips and knees in a straight line) - Point fingers inwards, bend arms slightly, keep shoulders above hands **Pre-tension** - Chest (pectoral muscles), and triceps
A09		**Triceps Dip** - Sit on platform facing away from column, knees bent 90º. - Hands at shoulder width on platform, fingers over the edge of the plate (on rubber mat) - Shoulders straight above hands - Shoulders back, elbows back, look up - Move hips forward off platform and bend elbows slightly to take weight of body. - Keep hips close to the platform **Pre-tension** - Triceps

A10		**Biceps Curl** - Stand in front of platform, perfect posture, knees slightly bent - Straps positioned in front slots - Straps at correct length to allow elbows bent at 90º - Elbows tight at sides, keep hands at shoulder width apart - Pull up as hard as possible – try to lift machine up - Keep wrists firm, not bent - Do not lean backward **Pre-tension** - Biceps
A14		**Lateral Raise** - Stand in front of platform, perfect posture, knees slightly bent - Straps positioned in side slots - Straps at correct length to allow arms to be just below shoulder height sideways - Arms straight but 'soft' - Pull up sideways as hard as possible – try to lift machine up - Keep wrists firm, not bent **Pre-tension** - Deltoid muscles
A11		**Bent-over pull** - Stand in front of platform, perfect posture, knees slightly bent, bend over 45º - Straps positioned in front slots - Straps at correct length to allow elbows bent at 90º while elbows at sides - Pull up as hard as possible – try to lift machine up - Concentrate on pulling elbows towards ceiling, and squeezing shoulder blades together - Keep wrists firm, not bent - Turn hands outward **Pre-tension** - Rhomboids and trapezius

Appendix F: Illustration of exercise session 1

Strength (lower body)									
		**1**		**2**		**3**		**4**	
Sets		1		1		1		1	
Duration		30 s		30 s		30 s		30 s	
Frequency		30 Hz		30 Hz		30 Hz		30 Hz	
Amplitude		Low		Low		Low		Low	
Rest		30 s		30 s		30 s		30 s	
Strength (upper body)									
		**5**		**6**		**7**		**8**		**9**
Sets		1		1		1		1		1
Duration		30 s		30 s		30 s		30 s		30 s
Frequency		30 Hz		30 Hz		30 Hz		30 Hz		30 Hz
Amplitude		Low		Low		Low		Low		Low
Rest		30 s		30 s		30 s		30 s		30 s
Stretching									
		**10**		**11**		**12**		**13**	
Sets		1		1		1		1	
Duration		30 s		30 s		30 s		30 s	
Frequency		30 Hz		30 Hz		30 Hz		30 Hz	
Amplitude		Low		Low		Low		Low	
Rest		0 s		0 s		0 s		0 s	
Massage									
		**14**		**15**		**16**		**17**		**18**
Sets		1		1		1		1		1
Duration		60 s		60 s		60 s		60 s		60 s
Frequency		40 Hz		40 Hz		40 Hz		40 Hz		40 Hz
Amplitude		High		High		High		High		High
Rest		0 s		0 s		0 s		0 s		0 s
Relaxation										
		**19**		**20**		**21**				
Sets		1		1		1				
Duration		60 s		60 s		60 s				
Frequency		40 Hz		40 Hz		40 Hz				
Amplitude		High		High		High				
Rest		0 s		0 s		0 s				

Appendix G: Advertisement

Appendix H: Table of Lusk

RER	Glucose	Fatty Acids	RER	Glucose	Fatty Acids
0.7036	0.00	100.0	**0.8750**	60.20	39.8
0.7050	0.50	99.5	**0.8800**	61.90	38.1
0.7100	2.30	97.7	**0.8850**	63.60	36.4
0.7150	4.20	95.8	**0.8900**	65.30	34.7
0.7200	6.00	91.0	**0.8950**	66.90	33.1
0.7250	7.80	92.2	**0.9000**	68.60	31.4
0.7300	9.60	90.4	**0.9050**	70.30	29.7
0.7350	11.40	88.6	**0.9100**	71.90	28.1
0.7400	13.20	86.8	**0.9150**	73.60	26.4
0.7450	15.00	85.0	**0.9200**	75.30	24.7
0.7500	16.80	83.2	**0.9250**	7.69	23.1
0.7550	18.60	81.4	**0.9300**	78.60	21.4
0.7600	20.40	79.6	**0.9350**	80.20	19.8
0.7650	22.10	77.9	**0.9400**	81.80	18.2
0.7700	23.96	76.1	**0.9450**	83.50	16.5
0.7750	25.70	74.3	**0.9500**	85.10	14.9
0.7800	27.40	72.6	**0.9550**	86.70	13.3
0.7850	29.20	70.8	**0.9600**	88.40	11.6
0.7900	31.00	69.0	**0.9650**	90.00	10.0
0.7950	32.70	67.3	**0.9700**	91.60	8.4
0.8000	34.50	65.5	**0.9750**	93.20	6.8
0.8050	36.20	63.8	**0.9800**	94.80	5.2
0.8100	38.00	62.0	**0.9850**	96.40	3.6
0.8150	39.70	60.3	**0.9900**	98.00	2.0
0.8200	41.40	58.6	**0.9950**	99.60	0.4
0.8250	43.20	56.8	**0.9600**	88.40	11.6
0.8300	44.90	55.1	**0.9650**	90.00	10.0
0.8350	46.60	53.4	**0.9700**	91.60	8.4
0.8400	48.30	51.7	**0.9750**	93.20	6.8
0.8450	50.00	50.0	**0.9800**	94.80	5.2
0.8500	51.70	48.3	**0.9850**	96.40	3.6
0.8550	53.40	46.6	**0.9900**	98.00	2.0
0.8600	55.10	44.9	**0.9950**	99.60	0.4
0.8650	56.80	43.2	0.9960	**100.00**	0.0
0.8700	58.50	41.5			

Appendix I: Borg's scale of perceived exertion

Nu	Description	% effort
6		20
7	Very, very light	30
8		40
9	Very light	50
10		55
11	Fairly light	60
12		65
13	Somewhat hard	70
14		75
15	Hard	80
16		85
17	Very hard	90
18		95
19	Very, very hard	100
20		