Efficacy of whole-body vibration on exercise tolerance and functional performance on the lower limbs of people with chronic obstructive pulmonary disease

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EFFICACY OF WHOLE-BODY VIBRATION ON EXERCISE TOLERANCE AND FUNCTIONAL PERFORMANCE OF THE LOWER LIMBS OF PEOPLE WITH CHRONIC OBSTRUCTIVE PULMONARY DISEASE

Submitted by

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A thesis submitted in total fulfilment of the requirement of the degree of Doctor of Philosophy

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01/08/2012
STATEMENT OF AUTHORSHIP AND SOURCES

This thesis contains no material published or extracted in whole or part from a thesis by which I have qualified for or been awarded another degree or diploma. No parts of this thesis have been submitted towards the award of any other degree or diploma in any other tertiary institution. No other person’s work has been used without date acknowledgement in the main text of the thesis. All research procedures reported in the thesis received the approval of the relevant Ethics/Safety Committees (where required).

Signed: _______________________________ Date: ____________________

Name: Trentham Phillip Furness
ABSTRACT

**Aims:** The general aim of this research is to advance knowledge of effects of whole-body vibration on exercise tolerance and functional performance of the lower limbs of people with chronic obstructive pulmonary disease in a community setting. Achieving the general aim of this research would determine efficacy of a whole-body vibration intervention to: (1) avoid exacerbations of chronic obstructive pulmonary disease that add to physical inactivity and, (2) improve performance of activities of daily living of people with chronic obstructive pulmonary disease. To meet the general aim of this research, specific aims were to: (1) establish validity of a WBV platform, (2) determine safety of a single session of whole-body vibration for people with chronic obstructive pulmonary disease by quantifying rating of perceived dyspnoea and selected physiological responses to physical activity, (3) describe transmission of whole-body vibration about the knee of people with chronic obstructive pulmonary disease, (4) establish reliability of the test procedure for the major intervention study, and (5) determine efficacy of a six week whole-body vibration intervention on rating of perceived dyspnoea, selected physiological responses to physical activity, and functional performance of the lower limbs of people with chronic obstructive pulmonary disease compared with a six week placebo intervention.

**Scope:** Chronic obstructive pulmonary disease can be treated with an ongoing pulmonary rehabilitation program. The beneficial effects of components of pulmonary rehabilitation programs incorporating aerobic conditioning and resistance training have been confirmed for improving exercise tolerance and functional performance of the lower limbs. However, effects of whole-body vibration as a mode of physical activity are yet to be explored in a community setting for people with chronic obstructive pulmonary disease even though whole-body vibration has been beneficial as a mode of physical activity for other sub-optimal health population sub-groups. This research therefore, is designed as a non-randomised placebo cross-over efficacy trial conducted in the home of participants affected with chronic obstructive pulmonary disease. Dependent variables of exercise tolerance and functional performance were tested across a 14 week intervention period. Participants first completed a six week whole-body vibration intervention, then after a 2 week washout period, completed a six week placebo intervention. Sixteen community-dwelling adults with stable chronic obstructive pulmonary disease provided voluntary informed consent to participate in this 14 week research (mean ± SD age = 72 ± 7 years, stature = 1.7 ± 0.1 metres, body mass = 85.7 ± 20.4 kilograms).

**Conclusions:** The general aim of this research was met, thus supporting the efficacy of whole body vibration as a safe mode of physical activity for people with chronic obstructive pulmonary disease. Results showed that whole-body vibration did not exacerbate symptoms of the disease associated with physical inactivity. Furthermore, the whole-body vibration intervention improved tests to mimic activities of daily living such as rising from a chair, turning, and walking gait with greater effect than a placebo intervention. As such, if a placebo effect was systemic to a whole-body vibration intervention, the effect was negligible. In conclusion, whole-body vibration was a safe mode of physical activity for people with stable chronic obstructive pulmonary disease that did not negatively effect exercise tolerance or exacerbate the disease, while concurrently improving functional performance of the lower limbs.
STATEMENT OF APPRECIATION

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# TABLE OF CONTENTS

<table>
<thead>
<tr>
<th>Section</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Statement of Authorship and Sources</td>
<td>ii</td>
</tr>
<tr>
<td>Abstract</td>
<td>iii</td>
</tr>
<tr>
<td>Statement of Appreciation</td>
<td>iv</td>
</tr>
<tr>
<td>Table of Contents</td>
<td>v</td>
</tr>
<tr>
<td>List of Figures</td>
<td>ix</td>
</tr>
<tr>
<td>List of Tables</td>
<td>xi</td>
</tr>
<tr>
<td>Glossary of Abbreviations &amp; Definition of Terms</td>
<td>xiii</td>
</tr>
<tr>
<td>List of Publications of this Research</td>
<td>xvi</td>
</tr>
<tr>
<td><strong>Chapter 1. General Introduction</strong></td>
<td>1</td>
</tr>
<tr>
<td>1.1 Statement of the Problem</td>
<td>1</td>
</tr>
<tr>
<td>1.2 General Aim of this Research</td>
<td>6</td>
</tr>
<tr>
<td>Specific Aims of this Research</td>
<td>7</td>
</tr>
<tr>
<td>Limitations</td>
<td>7</td>
</tr>
<tr>
<td>Delimiters</td>
<td>8</td>
</tr>
<tr>
<td><strong>Chapter 2. Literature Review</strong></td>
<td>9</td>
</tr>
<tr>
<td>2.1 Chronic Obstructive Pulmonary Disease</td>
<td></td>
</tr>
<tr>
<td>2.1.1 Assess and Monitor the Disease</td>
<td>10</td>
</tr>
<tr>
<td>2.1.2 Reduce Risk Factors</td>
<td>11</td>
</tr>
<tr>
<td>2.1.3 Managing Stable COPD</td>
<td>14</td>
</tr>
<tr>
<td>Aetiology of Poor Exercise Tolerance and Functional Performance of the Lower Limbs</td>
<td>16</td>
</tr>
<tr>
<td>Non-Pharmacologic Treatment</td>
<td>18</td>
</tr>
<tr>
<td>Pulmonary Rehabilitation</td>
<td>20</td>
</tr>
<tr>
<td>Peripheral Muscle Training</td>
<td>21</td>
</tr>
<tr>
<td>Contraindications to Exercise Participation and Recruitment for Research</td>
<td>23</td>
</tr>
<tr>
<td>Pharmacologic Treatment</td>
<td>24</td>
</tr>
<tr>
<td>2.1.4 Manage Exacerbations</td>
<td>26</td>
</tr>
<tr>
<td>2.1.5 Systematic Review of Peripheral Muscle Training – Resistance</td>
<td>26</td>
</tr>
<tr>
<td>Training</td>
<td>28</td>
</tr>
<tr>
<td>Evidence Level I</td>
<td></td>
</tr>
<tr>
<td>Evidence Level II</td>
<td>29</td>
</tr>
<tr>
<td>Systematic Review of Resistance Training and Functional Performance of People with COPD</td>
<td>33</td>
</tr>
<tr>
<td>Evidence Level V</td>
<td>35</td>
</tr>
<tr>
<td>Summary of Systematic Review</td>
<td>35</td>
</tr>
<tr>
<td>2.1.6 General Summary</td>
<td>36</td>
</tr>
<tr>
<td><strong>2.2 Whole-Body Vibration</strong></td>
<td>37</td>
</tr>
<tr>
<td>2.2.1 Whole-Body Vibration as a Mode of Physical Activity</td>
<td>40</td>
</tr>
<tr>
<td>Quantifying WBV / Vibration Platform Descriptors</td>
<td>41</td>
</tr>
<tr>
<td>Practical Application of WBV</td>
<td>46</td>
</tr>
<tr>
<td>2.2.2 Aetiology of Performance Improvement after WBV</td>
<td>48</td>
</tr>
<tr>
<td>The Motor Cortex</td>
<td>51</td>
</tr>
<tr>
<td>The Stretch Reflex</td>
<td>50</td>
</tr>
<tr>
<td>The Tonic Vibration Reflex</td>
<td>54</td>
</tr>
<tr>
<td>The Hoffmann Reflex</td>
<td>55</td>
</tr>
<tr>
<td>Stochastic Resonance</td>
<td>56</td>
</tr>
<tr>
<td>2.2.3 Whole-Body Vibration Guidelines</td>
<td>57</td>
</tr>
<tr>
<td>Individual</td>
<td>59</td>
</tr>
</tbody>
</table>
Chapter 3. Methods

3.1 Overview

3.2 Pilot Test 1: Validity of a Vibration Platform
   Overview
   Introduction
   Methods
   Instruments and Test Procedure
   Statistical Procedures
   Results
   Discussion and Summary

3.3 Pilot Test 2: Transmission of WBV about the Knee of Healthy Young Adults
   Overview
   Introduction
   Methods
   Participants
   Test Procedure
   Instruments
   Statistical Procedures
   Results
   Discussion
   Summary
   Limitations to the Planned Research

3.4 Pilot Test 3: Reliability of Selected Dependent Variables of Major Study 1 and Major Study 2
   Overview
   Methods
   Participants
   Selection Criteria, Selection Protocol and Instruments
   Tests of Cognition, Motor Function and Visual Acuity
     The Mini-Mental State Examination
     The Romberg Test
     The Snellen Eye Chart
     The Melbourne Edge Test
   Data Collection
   Exerciser Tolerance: The Borg CR-10 Visual Analogue Scale
   Exercise Tolerance: Heart Rate and Saturation of Haemoglobin
Descriptive Statistics of Participants 160
Effects of WBV and PLACEBO Interventions on Exercise Tolerance of People with COPD 162
Effects of WBV and PLACEBO Interventions on Functional Performance of the Lower Limbs of People with COPD
   Long-Term Effects on the TUG Test and 5-Chair Test 167
   Long-Term Effects on Kinematics of Gait 170
   Acute Effects on Kinematics of Gait 171
5.4 Discussion 175
   Major Findings 175
      Exercise Tolerance 176
         Fitness – Long-Term Compared with Instantaneous Results 177
         Exacerbations – Long-Term Compared with Instantaneous Results 179
   Functional Performance of the Lower Limbs 183
      Simulated ADLs 183
      Gait 184
      Possible Mechanisms of Improvement of Functional Performance 185
   Maintenance of Functional Performance of the Lower Limbs and a Placebo Effect 188
   Compliance and Drop-Out 189
   Anecdotal Responses and Future Directions 190
5.5 Summary 192
Chapter 6. Conclusion 194
References 195
List of Appendices 239
   Appendix A Concurrent validity of an accelerometer 240
   Appendix B Validation of vibration platform frequency 243
   Appendix C Transmission of WBV of healthy young adults 245
   Appendix D ACU HREC approval of transmission of young adults 250
   Appendix E ACU HREC approval of transmission of COPD 251
   Appendix F Southern Health information letter for WBV and COPD 252
   Appendix G ACU HREC approval of major study and reliability 253
   Appendix H Southern Health HREC approval of WBV and COPD 254
   Appendix I Tests of cognition and visual acuity 255
   Appendix J The Borg CR-10 VAS 258
   Appendix K ACU information letter/ advertising for safety of WBV 259
   Appendix L ACU HREC approval of WBV and COPD safety 260
   Appendix M The challenge of participant recruitment 261
   Appendix N The placebo vibration platform 262
   Appendix O Raw data of major study 2 264
LIST OF FIGURES

Figure 1.1. A person with COPD stands on a side alternating vibration platform. Assistance/support to an individual may be supplied. 5
Figure 2.1. Global tobacco rates among the top 10 most prolific nations (WHO, 2008b). 15
Figure 2.2. The dyspnoea spiral showing how reduced lung function leads to reduced physical activity, and deconditioning (Préfaut, Varray & Vallet, 1995). 17
Figure 2.3. Whole-body vibration can be used as a mode of physical activity in the commercial health and fitness industries. 39
Figure 2.4. Side alternating WBV (left) and synchronous WBV (right). Adapted from Cardinale & Wakeling (2005). 40
Figure 2.5. Pure sinusoidal WBV. 42
Figure 2.6. Multiple vibration frequency types. Whole-body vibration as a mode of physical activity is described as ‘sinusoidal’. Taken from Griffin (1994). 43
Figure 2.7. Peak-to-peak displacement and amplitude of pure sinusoidal vibration. The peak-to-peak displacement was 2.0 mm. The amplitude was 1.0 mm. 44
Figure 2.8. The motor cortex showing how γ-motoneurons regulate sensitivity of a muscle spindle (modified from Latash, 1998, p. 84). 50
Figure 2.9. The stretch reflex (Latash, 1998, p. 65). 51
Figure 2.10. The motor cortex and spinal region for γ-motoneuron and α-motoneurons activity (modified from Latash, 1998, p. 84). The γ-motoneurons regulate sensitivity of the muscle spindles. The α-motoneurons regulate contraction of skeletal muscle. 53
Figure 3.1. Overview of the design of this research to describe efficacy of WBV on exercise tolerance and functional performance of the lower limbs of people with COPD. 84
Figure 3.2. A tri-axial accelerometer (CXL25GP3, Crossbow Technology, San Jose, USA) and a 3D motion analysis camera (VICON Oxford Metrics, Oxford, UK). The images are not to scale. 86
Figure 3.3. Superior view of a vibration platform showing locations of reflective markers and the accelerometer. The reflective markers are identified by the 3D motion analysis system. 87
Figure 3.4. Bland-Altman plots shown the 95% limit of agreement among VICON 3D motion analysis system (VICON Oxford Metrics, Oxford, UK) and a tri-axial accelerometer (CXL25GP3, Crossbow Technology, San Jose, USA). 89
Figure 3.5. Demonstration of 40° knee flexion angle upon the vibration platform. The accelerometer can be seen at the knee and on the vibration platform. 94
Figure 3.6. An example of transmission, where the maximum acceleration about the knee is less than the maximum acceleration of the platform. For this example, the transmission would be < 1.00. Note, data are m.sec$^{-2}$ rather than RMS because negative values are squared when calculating RMS values. 95
Figure 3.7. Walkway length showing data sensitive area and body length protocol. 114
Figure 3.8. Spatial kinematic parameters of gait from the GAITRite® Electronic Walkway. Source: CIR Systems Inc., GAITRite® Electronic Walkway Technical Reference (WI-02-15). Stride length was the length in metres of the line of progression from points A and G.

Figure 5.1. Flow of participation in the study based on the CONSORT flow chart for reporting randomised controlled trials.

Figure 5.2. Instantaneous effects of WBV (blue) and PLACEBO (red) interventions on rating of perceived dyspnoea of people with COPD. Columns are mean data. Error bars are one standard deviation shown in the ‘positive’ direction.

Figure 5.3. Instantaneous effects of WBV (blue) and PLACEBO (red) interventions on heart rate of people with COPD. Columns are mean data. Error bars are one standard deviation shown in the ‘positive’ direction.

Figure 5.4. Instantaneous effects of WBV (blue) and PLACEBO (red) interventions on saturation of haemoglobin of people with COPD. Columns are mean data. Error bars are one standard deviation shown in the ‘positive’ direction. * indicates \( p \leq 0.05 \).

Figure 5.5. Effects of the washout period on functional performance of the TUG test. Week 6 results were long-term, Week 9 results were prior to the first PLACEBO session. Error bars are one standard deviation. * indicates \( p \leq 0.05 \) among Week 6 and Week 9 results.

Figure 5.6. Effects of the washout period on functional performance of the 5-chair test. Week 6 results were long-term, Week 9 results were prior to the first PLACEBO session. Error bars are one standard deviation.

Figure 5.7. Effects of WBV and PLACEBO interventions on stride length of people with COPD. Results were both long-term (blue) and acute (red). Results are mean stride length. Error bars are one standard deviation.

Figure 5.8. Effects of WBV and PLACEBO interventions on stride time of people with COPD. Results were both long-term (blue) and acute (red). Results are mean stride time. Error bars are one standard deviation. * indicates \( p \leq 0.05 \) within Week 6 long-term and acute stride times.

Figure 5.9. Effects of WBV and PLACEBO interventions on stride velocity of people with COPD. Results were both long-term (blue) and acute (red). Results are mean stride velocity. Error bars are one standard deviation.
LIST OF TABLES

Table 2.1  Spirometry classification of COPD severity according to GOLD  12
Table 2.2  Independent variables of resistance training for a skeletal muscle or skeletal muscle groups  22
Table 2.3  NHMRC Australia and GOLD levels of evidence  27
Table 2.4  Search strategy of Medline and Cochrane for evidence level I (to March 2012)  28
Table 2.5  Reviews of COPD and peripheral muscle training  29
Table 2.6  Criteria for a randomised controlled trial acceptable for this review  30
Table 2.7  Acceptable randomised controlled trials for various COPD samples  32
Table 2.8  Functional performance of the lower limbs after resistance training of people with COPD  34
Table 2.9  Statements of effectiveness of resistance training and associated evidence level after a systematic review of resistance training and functional performance  36
Table 2.10  Recommendations and practical applications of WBV as a mode of physical activity for various population sub-groups  47
Table 2.11  Components of WBV that can effect dependent variables regardless of population sub-group  58
Table 2.12  Commonly referenced published studies of WBV  73
Table 2.13  Examples of published studies of WBV  74
Table 2.14  Range of various variables for WBV interventions (tables 2.12 and 2.13)  75
Table 2.15  All published research of WBV and sub-optimal health populations  77
Table 3.1  Descriptive statistics and 95% limit of agreement among VICON and an accelerometer for various manufacturer defined vibration platform frequencies  88
Table 3.2  Anthropometric and descriptive statistics of the healthy young adults  98
Table 3.3  Transmission of WBV about the knee for various vibration platform frequencies, stance widths, and stance postures.  98
Table 3.4  Pre-determined stance postures and knee flexion angles by ‘Siliconcoach’  99
Table 3.5  Spirometry classification of COPD severity (GOLD, 2007)  105
Table 3.6  Procedure for recording forced vital capacity  107
Table 3.7  Sample descriptors of participants with COPD  117
Table 3.8  Spirometry descriptors of the participants with COPD prior to and after bronchodilator medication  118
Table 3.9  Inter-class correlation reliability results of all dependent variables across three test occasions for people with COPD  119
Table 3.10  Raw data of Furness & Maschette (2009) for healthy community dwelling older adults at baseline and after six weeks of WBV  124
Table 3.11  Descriptive statistics of raw data of Furness & Maschette (2009) for healthy community dwelling older adults at baseline and after six weeks of WBV  124
Table 3.12  Descriptive statistics of raw data after reciprocal transformation  125
Table 4.1  Contraindications for WBV and people of sub-optimal health (Cardinale & Rittweger, 2006)  132
Table 4.2  Anthropometric and descriptive statistics of the participants with COPD  137
Table 4.3  Spirometry descriptors of the participants with COPD prior to and after bronchodilator medication  138
Table 4.4  Effects of WBV on rating of perceived breathlessness, heart rate, and saturation of haemoglobin  139
GLOSSARY OF ABBRVIATIONS & DEFINITION OF TERMS

**Acute data:** Data collected within two minutes of the final bout of a WBV or PLACEBO session.

**ADLs:** Activities of daily living such as non assisted walking and independent living.

**Aerobic conditioning:** A purposeful component of pulmonary rehabilitation with the aim of improving cardiovascular fitness.

**Amplitude:** The maximum distance from the equilibrium position of pure sinusoidal vibration.

**ANZCTR:** Australian New Zealand Clinical Trials Registry.

**Borg CR-10:** A visual analogue scale to quantify prating of perceived breathlessness (i.e., dyspnoea).

**Community dwelling:** A potential participant had to be living in a fully-independent residence, and needed to have the capacity to complete ADLs (i.e., functional independence).

**COPD:** Chronic Obstructive Pulmonary Disease.

**Dyspnoea:** A clinical term of breathlessness

**Exercise:** Structured and purposeful physical activity.

**Exercise tolerance:** The ability to participant in exercise (i.e., exercise intolerance).

**FEV₁:** The forced expiratory volume after one second during a spirometry test.

**FVC:** The forced vital capacity of a spirometry test.

**Frequency:** The quantification of motion of pure sinusoidal vibration measured by the number of cycles of motion per second (Hertz; Hz).

**Functional performance:** The ability of the lower limbs to assist with performance of ADLs and walking gait.

**Gait:** The manner in which participants with COPD walked over a straight, flat and even surface as a self-selected comfortable speed.

**GOLD:** Global Initiative for Chronic Obstructive Lung Disease.

**Gravitational force:** A quantification of WBV intensity (i.e., g force).

**HR:** Heart rate. Quantified also with the heart rate reserve (HRR) and the percentage of heart rate reserve (%HRR).

**Hypoxemia:** A > 4% drop in saturation of haemoglobin to resting levels.
**Instantaneous data:** Data collected during the final bout (after 30 sec) of a WBV or PLACEBO session.

**ISO:** International Organisation for Standardisation.

**Isometric stance posture:** Maintaining a static knee flexion angle during a WBV or PLACEBO bout (i.e., isotonic skeletal muscle contractions).

**Isotonic stance posture:** Performing both eccentric and concentric skeletal muscle contractions during a WBV bout.

**Kinematics:** Spatiotemporal variables of gait.

**Long-term data:** Data collected at least 48 hours after a WBV or PLACEBO bout.

**Mid-test:** A level of the ‘test occasion’ independent variable. Data collected at week 3 and week 11.

**Muscular deconditioning:** A loss of skeletal muscle strength and power.

**NHMCR Australia:** National Health and Medical Research Council Australia.

**Peak-to-peak displacement:** The peak movement in one direction to the peak movement in the opposite direction of pure sinusoidal vibration.

**Peripheral muscle training:** Strengthening the upper and lower limbs to improve exercise tolerance and functional performance.

**Physical activity:** Any mode of activity that can increase the resting metabolic rate.

**Post-test:** A level of the ‘test occasion’ independent variable. Data collected at week 6 and week 14.

**Pre-test:** A level of the ‘test occasion’ independent variable. Data collected at week 1 (i.e., baseline) and week 9.

**PLACEBO:** A six week intervention consisting of 12 bouts of a PLACEBO vibration intervention where the amplitude of the vibration platform was ~0.0 mm.

**Pulmonary rehabilitation:** The comprehensive management of COPD.

**Reliability:** The test-retest-retest test procedure used to calculate the intra-class correlation coefficient (ICC), standard error of measurement (SEM) and coefficient of variation (CV) of the dependent variables of this research.

**Resistance training:** A component of pulmonary rehabilitation with the aim of improving skeletal muscle strength and power.

**SpO2:** Saturation of haemoglobin.

**Side alternating:** A vibration platform that creates pure sinusoidal vibration. The amplitude is changed by changing stance width about the axis of rotation.
Skidding: A method to validate the intensity of WBV (i.e., Hz and peak-to-peak displacement).

Stride length: The line of progression between two consecutive footprints of the same foot.

Stride time: Stride time was the time taken in seconds between the first contacts of two consecutive footballs of the same foot.

Stride velocity: The ratio of stride length to stride time and quantified as metres per second (m.sec⁻¹).

Spirometry: A mode of clinical diagnosis of COPD to quantify airflow limitation.

Sub-optimal health population: Any sample population with pathology.

The 5-Chair Stands Test: A dependent variable of functional performance. A valid measure of performance ADLs.


Transmission of WBV: The measurement of attenuation of WBV about various bony landmarks of the body.

Vibration: Shock that is ISO regulated for industrial exposure to vibration that can be detrimental to health with both acute and chronic exposure.

Washout: A duration of two weeks within the WBV and PLACEBO interventions where the participants were instructed not to commence any form of physical activity.

WBV: Whole-body vibration. A six week intervention consisting of 12 bouts of WBV intervention where the amplitude of the vibration platform was ~1.0 mm.

WHO: World Health Organisation.
LIST OF PUBLICATIONS OF THIS RESEARCH

Peer Review Journal


Peer Review Conference Proceeding


CHAPTER 1. GENERAL INTRODUCTION

1.1 Statement of the Problem

The World Health Organisation’s (WHO) current definition of chronic obstructive pulmonary disease (COPD) is a lung ailment fundamentally characterised by “persistent blockage of airflow from the lungs” (WHO, 2008a, p. 1). In 2004 the WHO estimated 64 million global cases of COPD and later predicted COPD would become the third leading cause of death by 2030 (WHO, 2008b). In Australia, COPD was the third leading cause of burden of disease and injury behind ischaemic heart disease and stroke (ABS 1301.0, 2001). Although not always the case, tobacco smoke is the leading risk factor for COPD (WHO, 2011). Somewhat understandably, tobacco usage was listed with physical inactivity as the two leading risk factors eliciting burden of disease in Australia (ABS 1301.0, 2001). While the cost of COPD in Australia has not been directly estimated, respiratory disease alone accounted for AU$3.31 billion (6.3%) of the total allocated health expenditure in Australia (ABS 1301.0, 2010).

Given that COPD diminishes the ability of the lungs to supply the body with oxygen, most activities of functional independence lead to breathlessness if the disease is progressed. Perceived breathlessness, or dyspnoea, leads to physical inactivity and consequently compounds the risk of burden of disease to those individuals. The combined effect of tobacco smoke and physical inactivity was responsible for 17% of the total burden of disease in Australia (ABS 1301.0, 2001). As a treatment strategy, physical activity is routinely incorporated into the management of stable COPD (GOLD, 2007; Cooper 2009).
Habitual physical activity practices among people with COPD have not been reported by leading health authorities and as such, the effect of COPD on physical activity in adults was limited (Vorrink, Kort, Troosters & Lammers, 2011). However, physical inactivity among older adults, regardless of health status, was a concern for health professionals (e.g., the ‘Swap It’ campaign recently introduced by the Federal Australian Government, www.swapit.gov.au). In Australia, no more than 65% of people over the age of 40 years engaged in physical activity (ABS 4156.0.55.001, 2011). Among adults aged 65 years or older, fewer than one in two adults were participating in physical activity (ABS 4156.0.55.001, 2011).

Based on recommendations from the WHO, the American Thoracic Society, the European Respiratory Society and the Thoracic Society of Australia and New Zealand, and other professional bodies, physical activity is now integral to pulmonary rehabilitation programs in public health settings to assist individuals affected by COPD (GOLD 2007). A goal of physical activity within pulmonary rehabilitation is to improve or maintain exercise tolerance and functional independence of people with COPD. Improvement of exercise tolerance and functional independence can increase the ability to complete activities of daily living (ADLs).

Maintenance and improvement of ADLs was central for functional independence (GOLD, 2007; O’Shea, Taylor & Paratz, 2009). Muscular strength, muscular power and gait ability are integral to the functional independence needed to complete ADLs. Specifically, activities requiring strong and powerful muscular contraction are fundamental and include rising from a chair, climbing stairs, turning and adjusting posture, and stumbling to avoid a fall (i.e., functional performance). People with COPD have less strength and power than healthy age matched controls (Hamilton, Killian, Summers & Jones, 1995). Subsequently, people with
COPD may have poorer exercise tolerance due to muscular weakness (Gosselink, Troosters & Decramer, 1996; Cooper 2009), a reduction of saturation of haemoglobin, and dyspnoea (GOLD, 2011). A diagnosis of COPD therefore, is synonymous with muscular deconditioning/dysfunction leading to poor performance of ADLs and poor exercise tolerance that is commonly addressed with pulmonary rehabilitation (GOLD, 2007).

Primary objectives of pulmonary rehabilitation are to combat poor exercise tolerance and poor functional performance. The components of pulmonary rehabilitation to beneficially effect exercise tolerance and functional performance can vary, but include exercise training (both aerobic conditioning and resistance training), healthy lifestyle education, and nutritional counselling (GOLD, 2011). Initially, pulmonary rehabilitation can be implemented at an outpatient setting, usually followed by a home based intervention that relies mostly on the compliance of the patient. The efficacy of both outpatient pulmonary rehabilitation (Donner & Muir, 1997; Dhein, Münks-Lederer & Worth, 2003; Fromer, Barnes, Garvey, Ortiz, Saver & Yawn, 2010) and home based pulmonary rehabilitation (Cigna & Turner-Cinga, 2005; Bartoli, Zanaboni, Maselle & Ursini, 2009; Vieira, Maltais & Bourbeau, 2010) is supported by systematic reviews.

During pulmonary rehabilitation, aerobic conditioning improved exercise tolerance at intensities over 50% of peak oxygen consumption ($VO_{2peak}$) (Mahler, 1998; GOLD, 2011). Although resistance training improved muscular strength (Shaw, Shaw & Brown, 2009), muscular power (Sayers & Gibson, 2010), and variables of gait (Fahlman, McNevin, Boardley, Morgan & Topp, 2011), dedicated strategies for resistance training of the lower limbs to improve functional performance lack thorough recommendations for people with COPD. A recent literature review reported efficacy of resistance training of the lower limbs
for people with COPD to elicit appreciable gains in muscular strength that may carry over to performance of some ADLs (O’Shea, Taylor & Paratz, 2009). However, resistance training was conducted in conjunction with other modes of training such as variants of aerobic conditioning. The independent effect of resistance training therefore, could not be established.

The need for safe and valid exercise interventions specifically for people with COPD is salient, and would add to the scientific evidence essential for public health initiatives to promote physical activity and reduce burden of the disease. Two common modes of physical activity; aerobic conditioning and resistance training, exacerbate dyspnoea for people with COPD and may lead to reduced physical activity because of fear of breathlessness (e.g., Normandin, McCusker, Connors, Vale, Gerardi & ZuWallack, 2002). The clinical and social merit of modes of physical activity that can minimise dyspnoea may add to the benefit of pulmonary rehabilitation interventions for people with COPD. Whole-body vibration (WBV) may be such a mode of physical activity.

Whole-body vibration is a mode of physical activity during which an individual stands on a vibration platform. The device can create acceleration predominantly in the vertical (Fz) direction (Figure 1.1). The acceleration forces are transmitted to the body and postulated to elicit physiological responses comparable to other modes of physical activity such as aerobic conditioning and resistance training (Pollock, Martin & Newham, 2012; von Stengel, Kemmler, Engelke & Kalender, 2012). Recent recommendations of nomenclature by the International Society of Musculoskeletal and Neuronal Interactions stipulate that vibration platforms are either; synchronous or side alternating (Rauch, Sievanen, Boonen, Cardinale, Degens, Felsenberg, et al., 2010).
Independent of type of vibration platform, WBV increased leg muscular strength and muscular power (Bosco, Iacovelli, Tsarpela, Cardianle, Bonifazi, Tihanyi et al., 2000; Furness & Maschette, 2009), oxygen consumption (Rittweger, Schiessl & Felsenberg, 2001; Rittweger, Ehring, Just, Mutschelknauss, Kirsch & Felsenberg, 2002), growth hormone, and testosterone levels (Bosco et al., 2000; Roelants, Delecluse, Goris & Verschueren, 2004) among a variety of healthy young and older adult sample groups. Furthermore, WBV may be a safe and effective mode to improve muscular strength, body balance and mechanical competence of bone for older adults with low bone mineral density (Torvinen, Kannus,
Sievänen, Järvinen, Pasanen, Kontulainen et al., 2002; Yue & Mester, 2002). A growing body of literature has reported benefit of WBV for sub-optimal health populations such as people suffering with cystic fibrosis (Rietschel, van Koningsbruggen, Fricke, Semler & Schoenau, 2008), multiple sclerosis (Jackson, Merriman, Vanderburgh & Braher, 2008) and stroke (van Nes, Latour, Schils, Meijer, Kuijik & Geurts, 2006). To date, trials into efficacy of WBV in patients with COPD are scarce in the literature.

Since the completion of the data collection period of this research, one paper has been published investigating efficacy of WBV to improve muscular strength and muscular power of people with COPD. In conjunction with a three week pulmonary rehabilitation intervention, WBV “may” have enhanced exercise tolerance of a multidisciplinary rehabilitation program (Gloeckl, Heinzelmann, Baeuerle, Damm, Schwedhelm, Diril et al., 2012, p. 79). These authors suggested that long-term studies are needed to determine optimal intensity and duration of WBV for people with COPD, though WBV seemed to be a “promising new exercise mode” (Gloeckl et al., 2012, p. 76).

1.2 General Aim of this Research

The general aim of this research is to advance knowledge of effects of WBV on exercise tolerance and functional performance of the lower limbs of people with COPD in a community setting. Achieving the general aim of this research would determine efficacy of a WBV intervention to: (1) effect exercise tolerance while avoiding exacerbations of COPD that add to physical inactivity, and (2) improve performance of ADLs of people with COPD.
Specific Aims of this Research

The specific aims of this research were to:

- Establish validity of a WBV platform,
- Determine safety of a single session of WBV for people with COPD by quantifying rating of perceived dyspnoea and selected physiological responses to exercise,
- Describe transmission of WBV about the knee of people with COPD,
- Establish reliability of the test procedure for the major intervention study, and,
- Determine efficacy of a six week WBV intervention on rating of perceived dyspnoea, selected physiological responses to exercise, and functional performance of the lower limbs of people with COPD compared with a six week PLACEBO intervention.

Limitations

Given the nature of COPD and potential exacerbations expected of people affected with it, this research is an efficacy trial and as such is not conducted as a randomised controlled trial. Rather than randomise the order of treatment intervention (i.e., WBV and PLACEBO interventions), it was thought necessary to first conduct the WBV intervention, and then the PLACEBO intervention. The main reason for that decision was to maximise participant numbers in the WBV group, with anticipated drop-out over the 14 weeks of this research. Other factors, considered beyond the scope of this research may have limited the results of this research. As such, this research design did not examine:

- The nutrition, exercise history, motivation and other environmental support mechanisms of community dwelling older adults,
- Current levels of physical activity,
• Current or past pharmacological treatment for COPD other than bronchodilator or corticosteroid treatment,
• Assessment and monitoring of the disease,
• Risk factors associated with the disease, and,
• Exacerbations of the disease earlier than six months prior to recruitment to the study.

**Delimiters**

Considering limitations, this research was delimited to a study of:

• People with well managed and stable COPD whom were independent living in metropolitan Melbourne and the Mornington Peninsula.
• Exercise associated dyspnoea and functional performance of the lower limbs for people with stable COPD,
• People with stable COPD whom were not engaged in pulmonary rehabilitation associated with usual outpatient hospital care, but were community dwelling and physically active in their independent living,
• Community based (field) tests of functional performance of the lower limbs designed to show improvement of muscular strength, muscular power, and ADLs,
• Selected linear kinematic variables of gait quantifiable by a portable electronic walkway,
• A six week WBV intervention and a six week PLACEBO intervention (interspersed with a two week washout period) with a session frequency of two sessions per week, and,
• Portable vibration platforms, able to be transported among the homes of the community dwelling participants.
CHAPTER 2. LITERATURE REVIEW

In the first section of this literature review COPD is defined and the mandate developed by the Global Initiative for Chronic Obstructive Lung Disease (GOLD) is selected to describe COPD and the methods of COPD assessment and management. Then a narrative review of literature describing pulmonary rehabilitation of people with COPD is presented. The more general narrative review is followed by a systematic review of literature specifically relating to peripheral muscle training (i.e., the legs) of people with COPD. It was conducted in an effort to more objectively understand contemporary knowledge and recommendations for improving exercise tolerance and functional performance of the lower limbs of people with COPD.

In the second section of this literature review, WBV is defined, and then literature describing WBV as a mode of exercise is reviewed. Also, theoretical perspectives of effects of WBV are reviewed in order to describe the physiological responses elicited by WBV. Further, published research recruiting sub-optimal health sample groups is reviewed. Concurrently, research involving healthy sample groups is included due to limited publications of sub-optimal health sample groups. Intervention methods of WBV are appraised in order to clarify the current understanding of WBV recommendations. Published literature targeting children, adolescents, and WBV during sitting, prone, and supine body positions are excluded because they are beyond the scope of this research research.
2.1 Chronic Obstructive Pulmonary Disease

A disease of the lungs, chronic obstructive pulmonary disease (COPD) is a leading cause of mortality worldwide (Casanova, Cote, Marin, Pinto-Plata, de Torres, Aguirre-Jaíme et al., 2008). The disease diminishes functional capacity of the pulmonary system, the goal of which is gaseous exchange (Celli, MacNee, Agusti, Anzueto, Berg, Bulst et al., 2004). Data of the Australian Bureau of Statistics report COPD to be more common than most cancers, road traffic accidents, coronary artery disease and diabetes (ABS 3303.0, 2007). An estimated 2.1 million Australians are affected with COPD, with predictions escalating that total to 4.5 million by 2050 (ABS 3303.0, 2007; Access Economics, 2008). Considering the known effect of COPD on mortality and a projected increase of incidence of COPD, research about assessment, prevention, management, and treatment of the disease is salient.

The disease is characterised by breathlessness (dyspnoea), excessive sputum production, chronic cough, bronchitis, and emphysema (Foy, Wickley, Adair, Lang, Miller, Rejeski et al., 2006). Chronic lung disease is typically not amenable to cure (Bemt, Schermer, Smeele, Bischoff, Jacobs, Grol et al., 2008). Functionally, exercise tolerance is negatively affected by COPD, resulting in for example, difficulty in performing ADLs (O’Shea, Taylor & Paratz, 2004), due partly to dyspnoea (Foy et al., 2006), and skeletal muscle dysfunction (Debigaré, Côté & Maltais, 2001). Major risk factors include tobacco smoke, occupational dust and chemicals as well as indoor and outdoor pollution (GOLD, 2007). For those reasons, COPD is considered as a preventable disease.
A unified approach was taken by the World Health Organisation and the United States National Heart, Lung, and Blood Institute to combat COPD resulting in the creation of the Global Initiative for Chronic Obstructive Lung Disease (GOLD) in 1998. The GOLD mandate has four specific considerations for COPD: (1) assess and monitor the disease, (2) reduce risk factors, (3) manage stable COPD, and (4) manage exacerbations.

2.1.1 Assess and Monitor the Disease

A clinical diagnosis of COPD must include measurement of airflow limitation with spirometry the recommended method (GOLD, 2007). A spirometer is a device for measuring flow and volume of inspired and expired air (Miller, Hankinson, Brusasco, Burgos, Casaburi, Coats et al., 2005). Given that COPD is a progressive deterioration of lung function, the GOLD committee proposed a classification scale of COPD severity to assist assessment (table 2.1).
Table 2.1

Spirometry classification of COPD severity according to GOLD

<table>
<thead>
<tr>
<th>Stage</th>
<th>FEV$_1$.FVC$^{-1}$ Spirometry classification</th>
<th>Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage I: Mild</td>
<td>FEV$_1$.FVC$^{-1} &lt; 0.70$</td>
<td>Possible chronic cough and sputum production</td>
</tr>
<tr>
<td></td>
<td>FEV$_1$ ≥ 80% predicted</td>
<td></td>
</tr>
<tr>
<td>Stage II: Moderate</td>
<td>FEV$_1$.FVC$^{-1} &lt; 0.70$</td>
<td>Shortness of breath, likely chronic cough and sputum</td>
</tr>
<tr>
<td></td>
<td>FEV$_1$ &lt; 80% predicted</td>
<td></td>
</tr>
<tr>
<td>Stage III: Severe</td>
<td>FEV$_1$.FVC$^{-1} &lt; 0.70$</td>
<td>Reduced exercise capacity and fatigue</td>
</tr>
<tr>
<td></td>
<td>FEV$_1$ &lt; 50% predicted</td>
<td></td>
</tr>
<tr>
<td>Stage IV: Very severe</td>
<td>FEV$_1$.FVC$^{-1} &lt; 0.70$</td>
<td>Respiratory failure, low quality of life</td>
</tr>
<tr>
<td></td>
<td>FEV$_1$ &lt; 30% predicted</td>
<td></td>
</tr>
</tbody>
</table>

Note: FEV$_1$: forced expired volume of oxygen in the first second. FVC: forced vital capacity of the lungs. Predicted FEV$_1$ can be calculated based on age, sex, height, and race (GOLD, 2007).

The key indices of spirometry for diagnosing COPD include forced vital capacity (FVC) and forced expiratory volume after one second (FEV$_1$) (Miller et al., 2005). The severity of COPD can be determined by comparing spirometry indices to predicted normal values because people with COPD present both a reduced FVC and FEV$_1$. However, universally applicable reference values are not available due to alterations to predicted estimates by age, sex, height, and race (GOLD, 2007). Predicted FEV$_1$ after one second of a 65 year old, 165 cm Caucasian female can vary between 2.41 L and 2.83 L (e.g., Cherniack & Raber, 1972; Crapo, Morris & Gardner, 1981; Roberts, MacRae, Winning, Adams & Seed, 1991; Enright, Kronmal, Higgins, Schenker & Haponik, 1993). Greater disparity exists among ethnic groups. For example, a 65 year old African American female with a stature of 165 cm would have a predicted FEV$_1$ of 1.97 L (Stinson, McPherson, Hicks, Scott, Sykes, Cobbs et al., 1981). Multiple prediction equations can lead to difficulty when comparing among COPD sample groups.
A more precise definition of the lower limits of normal for spirometry values were recently established for Caucasians (Stanojevic, Wade, Stocks, Hankinson, Coats, Pan et al., 2008). Stanojevic et al. (2008) drew data from over 3500 Caucasian participants by modelling from previous research including the third National Health and Nutrition Examination Survey (NHANES III) and results from Hankinson, Odencrantz and Fedan (1999).

Accurate assessment and ongoing monitoring of COPD enables appropriate modification of therapy and identification of exacerbations (van den Bemt, Schermer, Smeele, Bischoff, Jacobs, Groll et al., 2008). From a research perspective, accurate assessment of COPD is necessary to add validity and reliability of treatment interventions. The trend of published literature however, was not to rigidly abide the GOLD spirometry classification of COPD severity. Some published works clustered participants with large (30%) variance in predicted FEV<sub>1</sub> (Clark, Cochrane, Mackay & Paton, 2000; Bjørnshave & Korsgard, 2005; Vagra, Porszasz, Boda, Casaburi & Somfay, 2007; Casanova et al., 2008). By using the GOLD classification method, such variance covered Stage II, Stage III and Stage IV COPD. The severity of COPD affects symptoms (table 2.1) and should therefore be considered when planning research interventions.

Other studies have disregarded the GOLD method. One study reported FEV<sub>1</sub> values ranging between 23-68% of predicted, yet assigned those participants, with a large range in airway function, to the same cycle ergometer protocol (van Helvoort, van de Pol, Heijdra & Dekhuijzen, 2005). Another study recruited Stage III and Stage IV COPD participants to the same aerobic conditioning intervention group (Dreher, Walterspacher, Sonntag, Prettin, Kabitz & Windisch, 2008). Therefore, internal and external validity issues prevail in studies that recruited people with COPD for exercise interventions.
Accurate reporting of severity of COPD is required for exercise interventions because severity can determine intensity and duration of physical activity. Significant differences in ADLs as well as recreational physical activity were found among groups of COPD severity within ~2400 participants classified according to the GOLD method (Garcia-Aymerich, Lange, Benet, Schnohr & Antó, 2006). Subsequently, problems arise when interpreting results from populations with significant differences in their functional performance due to COPD severity.

In contrast, low variances in FEV$_1$ prediction values have also been reported. One study recruited 24 Stage III COPD participants with a 4% variance in the predicted FEV$_1$ (Mador, Bozkanat, Aggarwal, Shaffer & Kufel, 2004). The variance was 3% for another study of Stage III COPD participants although the number of participants with COPD was six (Radom-Aizik, Kaminski, Hayek, Halkin, Cooper & Ben-Dov, 2007). The FEV$_1$ values reported in those two studies are externally valid because a clear, specific population was identified, rather than a broad sample of people with COPD. For studies to be reliable and externally valid, recommendations, such as that of the GOLD committee should be followed.

### 2.1.2 Reduce Risk Factors

Smoking cessation is the most effective method to reduce COPD risk (GOLD, 2007). World Health Organisation data show over one billion tobacco smokers globally, with 10 nations accounting for almost two thirds of global use (WHO, 2008b) (figure 2.1). Over 80% of smokers live in third world and developing nations, while tobacco use is declining in developed nations (WHO, 2008b). The daily smoking rate in Australia, for example, has reduced from ~50% of the population in the 1940s (Scollo & Winstanley, 2008) to ~20% of
the population, with a further 4% of the population occasionally smoking (Australian Institute of Health and Welfare, 2009).

![Figure 2.1. Global tobacco rates among the top 10 most prolific nations (WHO, 2008b).](image)

Passive smoking and occupational exposure to indoor and outdoor air pollution are other risk factors for COPD. The WHO reported that over 50% of the world’s children are affected by poor air quality (WHO, 2008a). Given that COPD is a progressive disease, predominantly affecting those over 40 years of age, a case for early intervention strategies to reduce risk factors is strong. Reducing risk factors of COPD however, can be compounded by other factors related to aging such as sarcopenia, arthritis and accessibility to health related education (Chodzko-Zajko, Proctor, Fiatarone Singh, Minson, Nigg, Salem et al., 2009).
### 2.1.3 Manage Stable COPD

Although COPD is an irreversible disease, stable COPD can prevail but it is dependent upon the severity of the disease and clinical status of the patient (GOLD, 2007). Clinical and perceived measures of stability such as saturation of haemoglobin and dyspnoea can facilitate a diagnosis of stability. Alternatively, people with COPD were only included to an aerobic conditioning and resistance training intervention when free from respiratory infections and exacerbations two months prior (Marrara, Marino, de Held, de Oliveria Jn, Jamami & Di Lorenzo, 2008). Management strategies of stable COPD have focussed on both non-pharmacological and pharmacological treatments with the goal to prevent disease progression, relieve symptoms and improve quality of life (GOLD, 2007).

### Aetiology of Poor Exercise Tolerance and Functional Performance of the Lower Limbs

A recently published review provided evidence that airflow obstruction did not impair peripheral muscle (i.e, arms and legs) structure and function for people with COPD (Wüst & Degens, 2007). That evidence may be attributed to the net effect of gaseous exchange meeting demand because exercise capacity improved after various cardiovascular training interventions among people with COPD (Bernard, Whittom, LeBlanc, Jobin, Belleau, Bérubé et al., 1999; Berry Rejeski, Adair & Zaccaro, 1999; Arnardóttir, Sörensen, Ringqvist & Larsson, 2006; Marrara et al., 2008; Skumlien, Skogedal, Ryg & Bjørtuft, 2008).
Although airflow limitation may not directly affect peripheral muscle structure and function, perceived breathlessness (i.e., dyspnoea) negatively impacts physical activity (Préfaut, Varray & Vallet, 1995; figure 2.2). The so called ‘dyspnoea spiral’ is a useful model to visualise effects of lung function impairment (i.e., airflow limitation).

Figure 2.2. The dyspnoea spiral showing how reduced lung function leads to reduced physical activity, and deconditioning (Préfaut, Varray & Vallet, 1995).

Shown in figure 2.2, a sedentary lifestyle leads to deconditioning. For people with COPD, deconditioning of peripheral muscle may result in a loss of muscular strength, power and endurance, muscle cross sectional area, and muscle fibre Type distribution (Gosker, Wouters, van der Vusse & Schols, 2000; Storer, 2001; Degens & Always, 2006). Specifically, the age-related atrophy of Type II muscle fibres of the quadriceps femoris was exacerbated in the presence of COPD (Hughes, Katz, Sahgal, Campbell, Hartz & Shields, 1983).
The underlying physiological mechanism for augmented atrophy may include systemic inflammation, nutritional depletion, reduced saturation of haemoglobin, hypoxemia, and hypercapnia (Serres, Hayot, Préfaut & Mercier, 1998; Debigaré, Côté & Maltais, 2001; Augusti, Norguera, Sauleda, Sala, Pons & Busquets, 2003; GOLD, 2007; Wüst & Degens, 2007). Given that the major roles of Type II skeletal muscle fibres is to contract with great speed and power (Saltin, Henriksson, Nygaard & Anderson, 1977; ACSM, 1998a; Mahoney, & Tarnopolsky, 2005), modes of training to modify effects of underlying physiological mechanisms, or at least reduce the rate of atrophy through peripheral muscle training are justified (Chodzko-Zajko et al., 2009).

Fast and powerful muscular contractions are used for ADLs such as standing from a chair and climbing stairs. Understandably therefore, the deconditioning of Type II skeletal muscle fibres may lead to sedentary behaviours. However, functional measures of performance specific to speed and power have rarely been reported in the COPD literature, and therefore forms part of the rationale of this research.

Non-Pharmacologic Treatment

The third component of the GOLD mandate, to manage stable COPD, can be partly addressed by improving or maintaining exercise tolerance with non-pharmacologic methods. Following a review of literature, GOLD reported that all people with COPD benefit from structured physical activity programs in relation to exercise tolerance (GOLD, 2007). The dependent variables of exercise tolerance were; maximal exercise capacity during walking, cycling (O’Shea, Taylor & Paratz, 2004) and stair climbing (Foy et al., 2006; Dreher et al., 2008), lower limb cross sectional area, muscular strength and power (Bernard, LeBlanc, Whittom,
Jobin, Belleau & Maltais et al., 1998; Storer, 2001), and six-minute-walk-test distance (Dreher et al., 2008). Of the variables frequently listed to test exercise tolerance, stair climbing and the six-minute-walk-test distance should be considered to mimic ADLs. Furthermore measurement equipment and skill requirement for stair climbing and walking are low compared with for example, maximal capacity testing using a cycle ergometer.

To date, research into improvement in exercise tolerance and functional performance has been mainly directed at individuals with Stage III COPD. People with severe COPD almost always have a reduced quality of life and subsequently have a high need for medical attention (GOLD, 2007). It is therefore understandable that people with no greater than a Stage III COPD diagnosis are the most accessible for research. Generally, COPD is diagnosed at Stage II. At Stage II, not Stage I, individuals begin to seek advice for management of their condition (GOLD, 2007). Despite the GOLD spirometry classification system of COPD severity, limited literature is available on exercise interventions among people with Stage I and Stage II COPD. The relative lack of management advice in the early onset of the disease may be due to the classification description of COPD because individuals in the less severe Stages are generally not aware of abnormal lung function (GOLD, 2007) (i.e., table 2.1).

Non-pharmacologic treatments for all people with stable COPD were shown to reduce dyspnoea, and improve exercise tolerance (Nici, Donner, Wouters, Zuwallack, Ambrosino, Bourbeau et al., 2006). The treatment is evidence-based, multidisciplinary, and comprehensive. As such, that method is more commonly described as ‘pulmonary rehabilitation’.
Pulmonary Rehabilitation

Pulmonary rehabilitation methods for treatment of stable COPD involve patient assessment, exercise training, education, nutritional intervention and psychological support (Nici et al., 2006). Case specific, the rehabilitation method may also incorporate supplemental oxygen therapy or surgical intervention (e.g., lung transplant) (GOLD, 2007).

The content of a multidisciplinary and comprehensive pulmonary rehabilitation program should consist of more than breathing and relaxation exercises (e.g., Strijbos, Postma, van Altena, Gimeno & Koëter, 1996), or respiratory machines and upper arm strengthening exercises (e.g., Martinez, Vogel, Dupont, Stanopoulos, Gray & Beamis, 1993). Recently, a collective effort of the American Thoracic Society, the European Respiratory Society and GOLD, agreed on a more thorough understanding of appropriate and effective pulmonary rehabilitation. Pulmonary rehabilitation is now viewed as the cornerstone of comprehensive management of patients with COPD (Nici et al., 2006).

Although clinical evidence of the benefits of pulmonary rehabilitation have wide acceptance, replication based on the methods of published literature can be difficult. One study for example, beneficially affected people with COPD after a comprehensive multidisciplinary pulmonary rehabilitation intervention including formal education sessions (Normandin et al., 2002). However, nondisclosure of the precise components of the intervention makes replication impossible.
A comprehensive pulmonary rehabilitation program must incorporate strength training of skeletal muscle. Strengthening the upper and lower limbs is known as peripheral muscle training (O’Shea, Taylor & Paratz, 2004). The training methods to improve exercise tolerance and functional performance of the lower limbs of people with COPD are systematically reviewed later in this chapter (2.1.5 Systematic Review of Peripheral Muscle Training – Resistance Training). The following section, however, describes some peripheral muscle training modes.

**Peripheral Muscle Training**

People with COPD have lower muscle mass than healthy counterparts (Hamilton et al., 1995; Gosselink, Troosters & Decramer, 1996). As a consequence, skeletal muscular strength of the leg has been described as less than age and sex matched controls (Hamilton et al., 1995), although the exact cause (i.e., atrophy) remains speculative (Decramer, Lacquet, Fagard & Rogiers, 1994). Subsequent purpose designed peripheral muscle training interventions to improve muscular strength of the legs among people with COPD have concurrently improved various kinematic variables of gait (Panton, Golden, Broeder, Browder, Cestaro-Seifer & Seifer, 2004; Alexander, Phillips & Wagner, 2008). Strength of skeletal muscle is a required fitness component for maintenance of ADLs among people with COPD (GOLD, 2011) and can be correlated with improved gait performance of people with COPD (Alexander, Phillips & Wagner, 2008). Maintenance or improvement of gait can describe functional independence because non assisted gait can be quantified to describe performance of ADLs.
Resistance training is a primary peripheral muscel training mode to beneficially alter skeletal muscle (GOLD, 2007). The independent variables germane to resistance training and the American College of Sports Medicine (ACSM) recommendations are listed in table 2.2. Articulate and detailed disclosure of the training method needs to be reported in literature to enable replication. Recently, resistance training with an emphasis on strength surrounding the knee was incorporated into a training regime in an attempt to improve maximal leg strength of a sample with COPD (Skumlien et al., 2008). The authors did not however, report rest intervals. Lack of disclosure, or justification of resistance training protocols is not uncommon among COPD studies (e.g., Clark et al., 2000; Ortega, Toral, Cegudo, Villagomez, Sánchez, Castillo et al., 2002; Rooyackers, Berkeljon & Folgering, 2003; Mador et al., 2004). Again, such non-disclosure makes precise replication, comparisons and meta-analyses challenging.

Table 2.2

<table>
<thead>
<tr>
<th>Variable</th>
<th>Definition</th>
<th>Position stand (minimum recommendation for healthy adults)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Repetition</td>
<td>A single lift</td>
<td>8-12</td>
</tr>
<tr>
<td>Set</td>
<td>Number of repetitions completed without rest</td>
<td>1</td>
</tr>
<tr>
<td>Sessions</td>
<td>Number of sets per workout</td>
<td>8-10</td>
</tr>
<tr>
<td>Frequency</td>
<td>Sessions per week</td>
<td>2-3</td>
</tr>
<tr>
<td>Load</td>
<td>Resistance</td>
<td>8-12 Repetition Maximum</td>
</tr>
<tr>
<td>Rest Interval</td>
<td>Time in seconds between sets</td>
<td>60-180 seconds</td>
</tr>
<tr>
<td>Length of Intervention</td>
<td>Weeks of intervention</td>
<td>Ongoing</td>
</tr>
<tr>
<td>Overload</td>
<td>Increment in load over time</td>
<td>2-10%</td>
</tr>
</tbody>
</table>

Peripheral muscle training interventions have also incorporated aerobic conditioning for people with COPD. Various methods were used at different intensities. Intensity on cycle ergometers ranged from at least 50% of predicted peak work (Ortega et al., 2002; Mador et al., 2004; van Helvoort et al., 2005) to 80% of peak work (Radom-Aizik et al., 2007). Treadmill walking at 60% of the average speed of the six-minute-walk-test (Spruit, Gosselink, Troosters, De Paepe & Decramer, 2002), and 70% VO$_2$max were also used (Puente-Maestu, Sánz, Sánz, Cubillo, Mayo & Casaburi, 2000). Only one study, (Ortega et al., 2002) reported participant drop-out due to COPD exacerbations ($n = 4$) and lack of motivation ($n = 3$). Nevertheless, the six-minute-walk-test was probably the most representative of ADLs.

The limited available evidence about peripheral muscle training independent of other modes of pulmonary rehabilitation allowed three subjective conclusions: (1) moderate intensity peripheral muscle training exercises appear tolerable to people with COPD, (2) moderate intensity peripheral muscle training exercises were beneficial to people with COPD, and (3) functional measures of performance were not commonly assessed after moderate intensity peripheral muscle training exercises in people with COPD.

**Contraindications to Exercise Participation and Recruitment for Research**

Chronic obstructive pulmonary disease is not a contraindication to exercise. Some medications however, are contraindicated for exercising in people with COPD. Generally, medications that suppress respiratory drive or alleviate mucus are contraindicated for exercise among people with COPD (American Medical Directors Association, 2003).
By definition, the effect of COPD on airway obstruction is irreversible (GOLD, 2007). Subsequently, people with COPD were excluded from exercise interventions if their expiratory airflow limitation was reversible after bronchodilator inhalation and/or they had a long-term history of oral steroid therapy (Berry, Rejeski, Adair & Zaccaro, 1999; Clark et al., 2000). Prospective participants have also been excluded if self-reported breathing difficulty (i.e., dyspnoea) was present after performing ADLs such as: walking a city block, grocery shopping, completing household chores, lifting objects, walking up stairs, and rising from a chair (Berry et al., 1999).

Oversights of the GOLD statements (2007 & 2011) include the absence of detailed discussion around exercise training heart rate, subjective measures of perceived exertion to determine maximum intensity exercise, and subsequent suitability to certain exercise protocols. Although to establish exercise guidelines are beyond the scope of this research, the oversight obscures the management of stable COPD using resistance training and aerobic conditioning because the importance of peripheral muscle training had already been established.

**Pharmacologic Treatment**

A full review of pharmacological treatment is beyond the scope of this research. In this section, some common pharmacologic treatments are described due to their possible impact on the variables to be measured in this research. Participant screening protocols should include current pharmacologic treatment because the effect of some medications is to improve exercise capacity. Thorough screening allows replication for future research and removes the confounding potential of medication as an extraneous variable.
Usual pharmacologic treatment of COPD focuses on pulmonary function. The goal of pharmacologic treatment is to treat or prevent exacerbations (GOLD, 2007). Typical exacerbations are characterised by changes in a patient’s baseline dyspnoea, cough or sputum beyond normal daily variation (GOLD, 2007).

In the past, nitric oxide was prescribed for disease management because it assisted pulmonary vasodilatation. More recently, nitric oxide was shown to worsen gaseous exchange (Barberá, Roger, Roca, Rovira, Higenbottom & Rodriguez-Roisin, 1996; Jones & Evans, 1997). Corticosteroids are frequently prescribed to reduce exacerbations and increase exercise capacity for individuals with COPD (Gartlehner, Hansen, Carson & Lohr, 2006). However, the long-term use of corticosteroids is not recommended (GOLD, 2007) because habitual and substantial dosage may alter bone metabolism and bone health (Richy, Bousquet, Ehrlich, Meunier, Israel, Morii et al., 2003).

Contemporary pharmacologic treatments use bronchodilators to prevent and control symptoms, reduce exacerbations, and improve exercise tolerance for individuals with COPD (GOLD, 2007). Bronchodilators also increase exercise capacity for people with COPD (Man, Mustfa, Nikoletou, Kaul, Hart, Rafferty et al., 2004) and are subsequently viewed as performance enhancing. Salbutamol, a short-acting β₂-adrenergic receptor agonist is commonly used to assess airflow limitation reversibility (Johannessen, Lehmann, Omenaas, Eide, Bakkle & Gulsvik, 2006). Salbutamol can be used in the recruitment of participants with COPD for research to assess airflow limitation (Berry et al., 1999; Clark et al., 2000; GOLD 2007).
2.1.4 Manage Exacerbations

As mentioned, exacerbations of COPD change ‘baseline’ dyspnoea, cough or sputum beyond normal day to day variation (Rodrigues-Roisin, 2000; Burge & Wedzicha, 2003). Generally, respiratory infection and poor air quality cause exacerbations (GOLD, 2007). Exacerbations can be successfully treated with pharmacologic treatment and pulmonary rehabilitation (Puhan, Scharplatz, Troosters, Walters & Steurer, 2009).

Exercise can also change baseline dyspnoea for people with COPD. Dyspnoea was perceived as ‘somewhat severe’ to ‘severe’ for some patients with COPD during supervised cycling (Oliveira, Carrascosa, Borghi-Silva, Berton, Queiroga Jn, Ferreira et al., 2010) and walking (Breyer, Breyer-Kohansal, Funk, Dornhofer, Spruit, Wouters et al., 2010). It was recommended that exercise should be terminated if saturation of haemoglobin reduced to ≤ 85% (Poulain, Durand, Palomba, Ceugni, Desplan, Varray et al., 2003; Jenkins, Hill & Cecins, 2010). However, despite perceived breathing discomfort and a risk of reduced saturation of haemoglobin for people with COPD during exercise, peripheral muscle training remains integral to pulmonary rehabilitation.

2.1.5 Systematic Review of Peripheral Muscle Training – Resistance Training

The objective of this section is to identify and review published literature of peripheral muscle training (i.e., resistance training) of people with COPD. The aim is to systematically quantify evidence levels for effective peripheral muscle training interventions using National Health and Medical Research Council (NHMRC Australia), GOLD evidence level, and CONSORT guidelines (table 2.3). A level of evidence V category is also added to incorporate position statements of professional bodies, associations and societies. Interventions of combined
aerobic and resistance training are excluded unless the research design allowed the independent effect of resistance training to be known. Literature of aerobic conditioning is excluded due to the ‘power continuum’. The continuum describes the divide among metabolic power and mechanical power for anaerobic (e.g., resistance training) and aerobic energy pathways (Knuttgen, 2007). Interventions of less than six week duration are excluded because long-term structural change is of interest rather than acute motoneuron changes (Sale, 1988).

Table 2.3

**NHMRC Australia and GOLD levels of evidence**

<table>
<thead>
<tr>
<th>Level</th>
<th>Intervention</th>
<th>Diagnostic accuracy</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>A systematic review / meta-analysis of level II studies</td>
<td>A systematic review of level II studies</td>
</tr>
<tr>
<td>II</td>
<td>A randomised controlled trial</td>
<td>A study of test accuracy with: an independent, blinded comparison with a valid reference standard, among consecutive persons with a defined clinical presentation</td>
</tr>
<tr>
<td>III-1</td>
<td>A pseudo-randomised controlled trial</td>
<td>A study of test accuracy with: an independent, blinded comparison with a valid reference standard, among non-consecutive persons with a defined clinical presentation</td>
</tr>
<tr>
<td>III-2</td>
<td>A comparative study with concurrent controls</td>
<td>A comparison with reference standard that does not meet the criteria required for: Level II and III-1 evidence</td>
</tr>
<tr>
<td>III-3</td>
<td>A comparative study without concurrent controls</td>
<td>Diagnostic case-controlled study</td>
</tr>
<tr>
<td>IV</td>
<td>Case series with either post-test or pre-test/post-test outcomes</td>
<td>Study of diagnostic yield</td>
</tr>
<tr>
<td>V*</td>
<td>Panel Consensus Judgement</td>
<td>Based on clinical experience or knowledge</td>
</tr>
</tbody>
</table>

Note: * Not part of NHMRC or GOLD evidence level recommendations.
Evidence Level I

To meet the aim and specially answer the question “what resistance training protocols are beneficial for people with COPD?”, the search terms and MeSH headings for COPD were established a priori, and are listed in table 2.4. The abstract for each paper was screened for content and accepted/rejected for review (table 2.5). An accepted paper had to show clear method of search strategy and primary outcome measures central to the aim. One paper was highly acceptable.

Table 2.4

Search strategy of Medline and Cochrane for evidence level I (to March 2012)

<table>
<thead>
<tr>
<th>Search</th>
<th>Terms</th>
<th>Results Medline</th>
<th>Cochrane Library</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>&quot;COPD&quot; or &quot;chronic obstructive pulmonary disease&quot; or &quot;obstructive lung disease&quot; or &quot;chronic obstructive lung disease&quot; or &quot;chronic obstructive airway disease&quot; or &quot;chronic airways limitation&quot; or &quot;chronic airways obstruction&quot; or &quot;chronic bronchitis&quot; or &quot;pulmonary emphysema&quot; or &quot;chronic airflow obstruction&quot; or &quot;obstructive lung disease&quot;. mp. [mp=title, original title, abstract, name of substance word, subject heading word] AND</td>
<td>70759</td>
<td>7163</td>
</tr>
<tr>
<td>2</td>
<td>&quot;strength training&quot; or &quot;strength exercise&quot; or &quot;weight training&quot; or &quot;weight lifting&quot; or &quot;resistance exercise&quot; or &quot;resistance training&quot; or &quot;progressive resistance exercise&quot; or &quot;progressive resistance training&quot;. mp. [mp=title, original title, abstract, name of substance word, subject heading word]</td>
<td>127</td>
<td>39</td>
</tr>
<tr>
<td>3</td>
<td>1 and 2 to “review articles” “English language” “humans”</td>
<td>34</td>
<td>4</td>
</tr>
</tbody>
</table>
Table 2.5

Reviews of COPD and peripheral muscle training

<table>
<thead>
<tr>
<th>Authors</th>
<th>Review</th>
<th>Inclusion criteria*</th>
<th>Primary Outcome</th>
<th>Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>O’Shea et al. (2004)*</td>
<td>Systematic, Meta-Analysis</td>
<td>Evidence level: II, III-1, III-2 &lt; 6 week intervention</td>
<td>Resistance training</td>
<td>Overall N = 202, δ = 0.90 favours treatment to ↑ knee extensor strength</td>
</tr>
</tbody>
</table>

Note: * The authors did not use NHMRC evidence level guidelines nor CONSORT guidelines. # Inclusion criteria based on NHRMC evidence level guidelines used in this research.

O’Shea et al. (2004) conducted meta-analysis based on findings of five papers of varying NHMRC evidence. Review of the methods of those studies revealed that effective resistance training occurred in outpatient clinics when the participants used machine weights. However, because of inconsistent study descriptions and an oversight of CONSORT guidelines, a knowledge gap remained. Furthermore, the authors did not nominate an effective resistance training protocol for people with COPD. Given that the most recent review was published in 2004, it was considered necessary to revisit the literature to seek any additional knowledge in resistance training interventions. The search strategy of table 2.4 was used. The CONSORT checklist was used as a method of quality control for NHMRC Level II evidence.

**Evidence Level II**

At this level of evidence, only randomised controlled trials (NHMRC evidence level II) were considered for review since they can establish superior efficacy of a health care intervention and minimise potential bias, confounders, and group contaminant (Schulz, Altman, Moher, & the CONSORT Group, 2010). The literature search was limited to publications in the English language. For the purpose of this review, a randomised controlled trial is determined as a
study “in which the subjects were randomly allocated to a new treatment, to a control group or to an existing treatment group” (Peat, Mellis, Williams & Xuan, 2001, p. 22). The inclusion criteria for randomised controlled trials are shown in table 2.6.

Table 2.6

<table>
<thead>
<tr>
<th>Type</th>
<th>Cohort</th>
<th>Cohort with condition (i.e., COPD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>RCT 1</td>
<td>Control</td>
<td>Experimental (control)</td>
</tr>
<tr>
<td>RCT 2</td>
<td>Experimental</td>
<td>Experimental</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Experimental + Existing treatment</td>
</tr>
</tbody>
</table>

Note: Cohort: a group randomly gathered not currently receiving a condition such as resistance training. Cohort with condition: the condition may be an existing treatment, e.g., resistance training or pharmacologic treatment.

The exclusion criteria stated that the following methods and designs were unacceptable: (1) parallel design, (2) control or intervention group drawn from a different cohort, and, (3) participants were not randomly recruited (Peat et al., 2001; Schulz et al., 2010). Specific to this research, the exclusion criteria extended to: combined aerobic and resistance training groups and/or resistance training of the upper limbs only.

Applying the exclusion criteria resulted in a total of four acceptable papers (table 2.7). Of those papers however, three were susceptible to the Hawthorne Effect (changed behaviour via the process of being recruited to a study, independent of true experimental effect) and the remaining paper (i.e., Kongsgaard, Backer, Jørgensen, Kjaer, & Beyer et al., 2004) did not report the administered control procedure (e.g., non-activity, pamphlet or information session). One paper did not report control data (i.e., Ortega et al., 2002). Criteria for meta-
analysis were not met because dependent variables were not common among studies. Statement of allocation concealment was absent among all studies. Of the four papers in table 2.7, the method of one study was to assess FEV$_1$ after bronchodilator (i.e., Ortega et al., 2002). Therefore, of the other three studies the participant’s “COPD” may be reversible (i.e., airflow limitation), and thus, is not considered COPD to the GOLD classification method.

Unjustified sample sizes and resistance training protocols (i.e., reliability and validity), were evident in existing randomised controlled trials using resistance training in populations with COPD. Specifically, the number and intensity of repetitions was not justified or consistent, and the number of sets and rest intervals lacked clarity. The resistance training methods appeared perfunctory rather than systematic. Despite this, two strong trends emerged: (1) muscular strength improved for people with COPD after at least eight weeks of resistance training, and (2) resistance training interventions were well tolerated by people with COPD. While measures of muscular strength and power such as one repetition maximum (1RM) and work rate (J) are commonly reported, they do not necessarily transfer to functional performance of ADLs. Given the employed search strategy was unable to reveal any NHMRC evidence level II studies for effects of resistance training on functional performance of people with COPD, the systematic search strategy is expanded to lower evidence levels.
<table>
<thead>
<tr>
<th>Authors</th>
<th>Sample (included in analysis)</th>
<th>Groups</th>
<th>Intervention</th>
<th>Findings</th>
<th>Adverse effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Simpson et al. (1992)</td>
<td>3 sessions/week 8 weeks</td>
<td>G1: Resistance training</td>
<td>3 sets, 10 reps, 50-85% 1RM (overload)</td>
<td>G1: 1 RM ↑ for all muscle groups G2: no change</td>
<td>n = 0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>G2: No-activity control §</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>28 Stage III (FEV₁ SD = 20%)</td>
<td></td>
<td>Hip flexors &amp; leg extensors</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Clark et al. (2000)</td>
<td>2 sessions/week 12 weeks</td>
<td>G1: Resistance training</td>
<td>3 sets, 10 reps, 70% 1RM</td>
<td>G1: Work (J) ↑ for all muscle groups G2: no change</td>
<td>Not reported</td>
</tr>
<tr>
<td></td>
<td></td>
<td>G2: No-activity control §</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>43 Stage II (FEV₁ SD = 23%)*</td>
<td></td>
<td>Hip flexors &amp; extensors, leg extensors</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ortega et al. (2002)</td>
<td>3 sessions/week 12 weeks</td>
<td>G1: Resistance training</td>
<td>4 sets, 6-8 reps, 70-85% 1RM</td>
<td>G1: 1 RM ↑ for all muscle groups G2: data not reported</td>
<td>n = 1 due to exacerbations</td>
</tr>
<tr>
<td></td>
<td></td>
<td>G2: Control §</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>35 Stage III (FEV₁ SD = 14%)*†</td>
<td></td>
<td>Leg flexors &amp; extensors</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kongsgaard et al. (2004)</td>
<td>2 sessions/week 12 weeks</td>
<td>G1: Resistance training</td>
<td>4 sets, 8 reps, 80% 1RM</td>
<td>G1: 5 RM ↑ for all muscle groups G2: no change</td>
<td>n = 5 unrelated to the study</td>
</tr>
<tr>
<td></td>
<td></td>
<td>G2: Control §</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>13 Stage III (FEV₁ SD = 4%)*</td>
<td></td>
<td>Hip flexors, leg flexors &amp; extensors</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note: *: Did not report prediction equation used. †: post-bronchodilator. §: Susceptible to Hawthorne Effect. ‡: Protocol was not justified with known evidence of efficacy. SD: standard deviation.
Systematic Review of Resistance Training and Functional Performance of People with COPD

Functional independence is of considerable importance to people with COPD and of equal concern is the maintenance of ability to complete ADLs (GOLD, 2007). Measurement tools to quantify such have been used in a variety of settings through published research. This section therefore, focuses on published research in which the primary outcomes were measures of functional performance specific to ADLs. The included studies were listed in table 2.8. The search terms; “functional performance" or "health status" or "mobility" or "function" or "functional fitness” or “functional independence”, were combined with search terms from table 2.4.

The additional search produced 38 papers. Two papers, shown in table 2.8, were acceptable according to the criteria that the following methods and designs were excluded: (1) Evidence Level V, (2) combined aerobic and resistance training groups and resistance training of the upper limbs only, and (3) dependent variables of a primarily aerobic nature.

Of the functional performance tests identified, validity and reliability had been previously established for the sit-to-stand repetitions in 30 seconds test (Rikli & Jones, 2001), but not for the sit-to-stand repetitions in 60 seconds test. Both studies of table 2.8 did not establish reliability or validity of the dependent variables. Furthermore, one study combined data of people with Stage II and Stage III COPD severity (Normandin et al., 2002). Although later, COPD severity was shown to affect functional performance (Garcia-Aymerich et al., 2006). Despite this, resistance training was shown to improve functional performance of people with COPD.
Table 2.8

Functional performance of the lower limbs after resistance training of people with COPD

<table>
<thead>
<tr>
<th>Authors</th>
<th>Sample (included in analysis)</th>
<th>Groups</th>
<th>Intervention</th>
<th>Findings</th>
<th>Adverse effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normandin et al. (2002)</td>
<td>40 Stage II and Stage III (FEV₁ s.d = 18%)*+</td>
<td>G1: Resistance training</td>
<td>Sets unknown, 8-10 reps</td>
<td>Chair stand repetition ↑ from 16 to 18.8 per minute</td>
<td>n = 14 due to exacerbations</td>
</tr>
<tr>
<td></td>
<td></td>
<td>G2: Aerobic training</td>
<td>Resistance unknown</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>G3: Pulmonary rehabilitation</td>
<td>Hip &amp; leg flexors &amp; extensors</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kongsgaard et al. (2004)</td>
<td>13 Stage III (FEV₁ s.d = 4%)*</td>
<td>G1: Resistance training</td>
<td>4 sets, 8 reps 80% 1RM</td>
<td>Stair climbing ↓ from 4.7 sec to 3.9 sec</td>
<td>n = 5 unrelated to the study</td>
</tr>
<tr>
<td></td>
<td></td>
<td>G2: Control</td>
<td>Hip flexors, leg flexors &amp; extensors ‡</td>
<td>Chair stand repetition ↑ from 17.0 to 18.3 per minute</td>
<td></td>
</tr>
</tbody>
</table>

Note: *: Did not report prediction equation used. ‡: Protocol was not justified with known evidence of efficacy. +: Did not separate groups according to COPD severity
Evidence Level V

A rationale for the use of peripheral muscle training to improve skeletal muscle function in people with COPD has been addressed by GOLD, the American Thoracic Society, the European Respiratory Society, the Thoracic Society of Australia and New Zealand, the Australian Lung Foundation, the International Federation of Sports Medicine, and the American Department of Veterans Affairs. Authoritative health bodies supported the importance of aerobic conditioning and resistance training, yet specific practical guidelines for the implementation of resistance training were not described. Specifically, the joint American Thoracic Society/European Respiratory Society statement (Nici et al., 2006) recommended more research to identify appropriate resistance training intensities for people with COPD. Such research could aid the creation of universal guidelines that may be followed by health professional in an effort to improve the third consideration of the GOLD mandate (i.e., to manage stable COPD), with an overarching goal to improve or maintain exercise tolerance and functional performance.

Summary of Systematic Review

A component of pulmonary rehabilitation, peripheral muscle training of the lower limbs improved exercise tolerance of people with COPD (tables 2.7 and 2.8). In particular, resistance training improved muscular strength and power, and performance of ADLs with, at most, evidence level II (table 2.9). Despite evidence, variation exists among resistance training interventions and exercise guidelines have not been established. Calls for guidelines from professional bodies at the forefront of clinical practice remain without a united response. Specifically, after a systematic review of literature, an optimal resistance training method has not been established (table 2.9).
Table 2.9

Statements of effectiveness of resistance training and associated evidence level after a systematic review of resistance training and functional performance

<table>
<thead>
<tr>
<th>Statement</th>
<th>Level of Evidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Resistance combined aerobic training beneficially effect exercise tolerance and health status for people with COPD</td>
<td>Level I</td>
</tr>
<tr>
<td>Resistance training beneficially effects exercise tolerance and health status for people with COPD</td>
<td>Level II</td>
</tr>
<tr>
<td>Resistance training is well tolerated for people with COPD</td>
<td>Level II</td>
</tr>
<tr>
<td>Resistance training was most effective when set at between 50-85% 1RM</td>
<td>Level II</td>
</tr>
<tr>
<td>Resistance training was most beneficial when conducted at least two sessions per week</td>
<td>Level II</td>
</tr>
<tr>
<td>Resistance training beneficially effects measures of functional performance of activities of daily living</td>
<td>Level II &amp; Level III-I</td>
</tr>
</tbody>
</table>

*Guidelines of resistance training for people with COPD*  
None

2.1.6 General Summary

Creation of the Global Initiative for Chronic Obstructive Lung Disease, backed by the most internationally respected health organisation (i.e., the WHO) has allowed narrative and systematic analyses of the COPD literature. These analyses established four mandates for the consideration of COPD: (1) assess and monitor the disease, (2) reduce risk factors, (3) manage stable COPD, and (4) manage exacerbations. The third mandate; to manage stable COPD, can be addressed partly by improving or maintaining exercise tolerance and functional performance.
Resistance training has been routinely incorporated to pulmonary rehabilitation interventions (table 2.4). However, study protocols could not allow the independent benefit of resistance training to be identified. As such, the independent benefit of resistance training has been limited to very few studies (table 2.7). Furthermore, effects of resistance training on functional performance of ADLs appear almost to have been ignored by researchers working with people affected with COPD (table 2.8). The independent effect of resistance training is only known for stair climbing and chair standing repetitions (i.e., Normandin et al., 2002; Kongsgaard et al., 2004). Other field tests, such as the timed-up-and-go test (TUG test) and the 5-chair stands test (5-chair test), and/or kinematic variables of gait with known reliability and validity could be used to describe effects of resistance training on exercise tolerance and functional performance of the lower limbs of people with COPD.

Further research is required to thoroughly establish effective interventions to beneficially improve or maintain exercise tolerance and functional performance of the lower limbs of people with COPD. Ideally, interventions should be randomised controlled trials and may focus on functional effects of resistance training related to ADLs.

**2.2 Whole-Body Vibration**

Vibration may occur in most environments. Workers are exposed to vibration when they use jack hammers and drive trucks. The vibrations are generated by the machinery. Athletes are exposed to vibration when they run or ride a horse. Vibrations in this case are generated by the contact of the foot upon the running surface and the movements of the horse. In everyday activities, people are exposed to vibration when riding a train or driving a car.
Typically, vibration affects the whole-body and henceforth is referred to as whole-body vibration (WBV). Further, WBV may be moving in alternate directions simultaneously (Griffin, 1994). More specifically, vibration may be evident in the frontal, sagittal and transverse planes (Hamill & Knutzen, 2003; Marieb, 2004). The International Organisation for Standardisation (ISO) devised guidelines within which WBV magnitude must adhere. These limits are to ensure that deleterious conditions such as gastrointestinal upsets (Ishitake, Ando, Miyazaki & Matoba, 1998; Ishitake, Mitazaki, Noguchi, Ando & Matoba, 2002) and biological structural damage are avoided (Necking, Dahlin, Fridén, Lundborg, Lundström & Thornell, 1992).

While ISO guidelines are established for occupational environments, such guidelines do not exist for athletic, social exercise, exercise training, and exercise rehabilitation environments, despite a theory that WBV may cause structural damage to bones (Kiiski, Heinonen, Järvinen, Kannus & Sievänen, 2008). Furthermore, because WBV research in exercise training environments is relatively recent, scientific knowledge of effects of WBV on all body systems are not thoroughly understood. Unsurprisingly, and possibly due to the variability of physiological demands across sport and exercise environments, ISO standards and scientific guidelines for safe and effective WBV have not been established.

Despite the known deleterious effects of occupational WBV (ISO 2631-1, 1997), as an exercise training mode, WBV has been created by vibration platforms and used for both healthy young adult and older adult populations. Moreover, within the commercial health and fitness industries, WBV represents an exercise mode in which an individual stands on a functioning vibration platform and may improve a variety of physiological components such as body balance, muscular strength and power, and bone metabolism (figure 2.3).
Furthermore, WBV has been advertised and marketed as an essential tool with multiple capabilities such as eliminating cellulite and fat and improving the users’ golf swing. However, claims from the commercial health and fitness industry are based almost exclusively upon anecdotal opinion and lack scientific evidence.

![Whole-body vibration can be used as a mode of physical activity in the commercial health and fitness industries.](image)

**Figure 2.3.** Whole-body vibration can be used as a mode of physical activity in the commercial health and fitness industries.

Vibration platforms may deliver side alternating vibration or synchronous vibration to an individual (figure 2.4). Primarily, side alternating WBV occurs when one places the feet equidistant about the axis of rotation. Synchronous WBV occurs when one places the feet at any point upon the vibrating platform.
2.2.1 Whole-Body Vibration as a Mode of Physical Activity

During interventions, WBV is used when a participant stands upon a vibrating platform. Exercise protocols have varied among several sets of 30 second bouts, to bouts as long as four minutes (Torvinen, Kannus, Sievänen, Järvinen, Pasanen, Kontulainen et al., 2003; Bogaerts, Verschueren, Delecluse, Claessens & Boonen, 2007; Rees, Murphy & Watsford, 2008).

Whole body vibration has been reported to increase/improve; jump height (Bosco, Cardinale, Tsarpela, Colli, Tihanyi, von Duvillard et al., 1998; Roelants, Delecluse, Goris & Verschueren, 2004; Rønnestad, 2004), knee extensor strength (Delecluse, Roelants & Verschueren, 2003; Roelants, Delecluse & Verschueren, 2004), explosive lower limb strength (Russo, Lauretani, Bandinelli, Bartali, Cavazzini, Guralnik et al., 2003), neuromuscular activation as measured by surface electromyography (Abercromby, Amonette, Layne, McFarlin, Hinman & Paloski, 2007a), oxygen uptake (Rittweger et al., 2002), postural control (Cheung, Mok, Qin, Sze, Lee & Leung, 2007; Rees, Murphy & Watsford, 2008), and hormonal responses such as testosterone and growth hormone (Bosco et al., 2000) among various healthy young and old adult populations. Increases and improvements within the
human body however, have been contradicted by other research. Whole body vibration failed to improve; jump height (Cochrane, Legg & Hooker, 2004; Delecluse, Roelants, Diels, Koninckx & Verschueren, 2005), lower limb muscular strength and power (de Ruiter, van Raak, Schilperoort, Hollander & de Haan 2003; Bautmans, Van Hees, Lemper & Mets, 2005) and hormonal responses such as testosterone and growth hormone (Cardianle, Leiper, Erskine, Milroy & Bell, 2006) among various young and old adult populations.

Different findings among studies may be due to variation of study methods. Participant number, gender, age and health status, WBV intervention type (i.e., frequency, intensity, type and time; FITT), dependent variables, measurement instruments, statistical analyses, and study location (i.e., laboratory or community based) varied among published literature. The quantification of WBV as a mode of exercise is in need of further understanding and standardisation. Accurate reporting of vibration platform descriptors and disclosure of WBV interventions methods will allow replication among future studies.

**Quantifying WBV / Vibration Platform Descriptors**

Theoretically, vibration platforms generate pure sinusoidal WBV (figure 2.5). Such WBV allows investigation and application of a single frequency of motion rather several alternate and simultaneous frequencies (Griffin, 1994; Cardinale & Bosco, 2003). The frequency of motion is measured by the number of cycles of motion per second (Hertz) (Griffin, 1994). The system international unit for frequency is Hertz (Hz).
Alternate and simultaneous frequency can occur in any environment; occupational or recreational. Alternate WBV frequency refers to shock WBV; whilst simultaneous refers to numerous alternate vibrations acting in conjunction (Griffin, 1994). Figure 2.6 shows other forms of vibration that occur in all environments. Most notably, the frequency of the motion is inconsistent. Further, not shown in figure 2.6, the motion frequencies may be occurring simultaneously and in different planes.

Figure 2.5. Pure sinusoidal WBV.
Figure 2.6. Multiple vibration frequency types. Whole-body vibration as a mode of physical activity is described as ‘sinusoidal’. Taken from Griffin (1994).

As mentioned, vibration may be evident in the frontal, sagittal and transverse planes in conjunction. With reference to vibration platforms and WBV however, the vibration moves through the transverse plane, in a vertical direction or about a sagittal axis.
The magnitude of pure sinusoidal motion is measured by the displacement of the vibration (Griffin, 1994). Within literature, however, inconsistency exists because several nouns are used (Lorenzen, Maschette, Koh & Wilson, 2009). Amplitude, displacement, peak-to-peak amplitude and peak-to-peak displacement have been used, and at times, used synonymously (e.g., Delecluse, Roelants & Verschueren, 2003). Those authors initially referred to amplitude, only later to refer to peak-to-peak displacement of the same magnitude.

Griffin (1994) suggested displacement is defined by the peak movement in one direction to the peak movement in the opposite direction (i.e., peak-to-peak displacement) (figure 2.7). The definition would find support from Knight (2000) who suggested amplitude was the maximum distance from the equilibrium position, whilst Mester, Spitzempfeil and Yue (2002) defined magnitude of the motion as amplitude (mm) of a half wave form. Figure 2.8 shows peak-to-peak displacement of 2.0 mm and amplitude of 1.0 mm.

![Diagram of sinusoidal vibration with annotations for peak-to-peak displacement and amplitude]

Figure 2.7. Peak-to-peak displacement and amplitude of pure sinusoidal vibration. The peak-to-peak displacement was 2.0 mm. The amplitude was 1.0 mm.
Using the frequency (Hz) and amplitude (mm) of a vibration platform, it is possible to quantify WBV at the surface of the platform. Equation 1 shows the calculation of gravity \( g \), which can be reported within WBV literature along with frequency and amplitude (Rittweger, Schiessl & Felsenberg, 2001).

\[
g = \frac{A(2\pi f)^2}{9.81} \tag{equation 1}
\]

Where ‘\( A \)’ is the amplitude (m), ‘\( f \)’ is the frequency (Hz), ‘\((2\pi f)^2\)’ is the maximum acceleration of the platform, and 9.81 is the acceleration of gravity. If for example, peak-to-peak displacement was 2.0 mm and frequency was 25 Hz, gravity \( g \) was 2.52 g, because \( A \) was 0.001 m.

It seems that WBV has been quantified and, at times, reported in such a way to allow comparison among study methods. Comparison however, is limited because only frequency is the most commonly reported variable in the frequency range of 26 to 50 Hz (Bosco, Colli, Introini, Cardinale, Tsarpela, Madella et al., 1999; Cardinale & Lim, 2003; Delecluse, Roelants & Verschueren, 2003; Erskine, Smillie, Leiper, Ball & Cardinale, 2007).

Given that \( g \) is affected by \( A \), the inconsistent definition of \( A \) limits \( g \) calculation. Of the recent literature, Erskine et al. (2007) reported frequency 30 Hz, peak-to-peak displacement 4.0 mm and gravity 3.5 g. Using equation 1, gravity is 7.24 g. The authors did not disclose the gravity calculation. Other authors have neglected to report gravity, yet referred to peak-
to-peak amplitude (Rees, Murphy & Watsford, 2008). As a consequence, incomplete and incorrectly reported information limits comparison and accurate replication.

**Practical Application of WBV**

Independent of the quantification method of WBV and the subsequent effects found within the literature, WBV has been sparsely recommended for most population sub groups (table 2.10). That is, no strong evidence based statement exist to advocate the use of WBV as an exercise mode. Whole-body vibration studies incorporating healthy children, healthy young adults, healthy young athletes, community dwelling older adults, institutionalised older adults and samples of adults recovering from illness, or injury and coping with disease have been published mostly without useful and specific practical application of WBV. Table 2.10 list the only published recommendations of WBV as a mode of exercise.

The studies listed in table 2.10 were selected to represent some detailed information as to the practical application of WBV. Such information may be used by the clinician, coach, personal trainer, and individual. Those studies, however, comprised a small proportion of the total WBV literature base. More research is required with the aim to: (1) identify and describe physiological mechanisms that elicit performance improvement after WBV, (2) identify appropriate vibration platform descriptors and WBV protocols to elicit performance improvement, and (3) increase participant numbers and extend WBV interventions to examine long-term effects. Despite the recognised directions for future research, the capacity to address each of the three needs was beyond the scope of this research.
Table 2.10

Recommendations and practical applications of WBV as a mode of physical activity for various population sub-groups

<table>
<thead>
<tr>
<th>Cohort Condition</th>
<th>Authors</th>
<th>Year</th>
<th>Recommendations of WBV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Child athletes</td>
<td>Mahieu et al.</td>
<td>2006</td>
<td>strength training tool</td>
</tr>
<tr>
<td>Young athletes</td>
<td>Issurin &amp; Tenenbaum</td>
<td>1999</td>
<td>identify “hidden reserves” (p. 181)</td>
</tr>
<tr>
<td></td>
<td>Annino et al.</td>
<td>2007</td>
<td>improve lower limb muscular power</td>
</tr>
<tr>
<td>Young adults</td>
<td>Cormie et al.</td>
<td>2006</td>
<td>warm up tool</td>
</tr>
<tr>
<td></td>
<td>van den Tillaar</td>
<td>2006</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Rønnestad</td>
<td>2004</td>
<td>incorporate to resistance training interventions</td>
</tr>
<tr>
<td></td>
<td>Da Silva et al.</td>
<td>2007</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Da Silva et al.</td>
<td>2007</td>
<td>to stimulate weight loss</td>
</tr>
<tr>
<td></td>
<td>Garatachea et al.</td>
<td>2007</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Bakhtiary et al.</td>
<td>2007</td>
<td>prevent delayed onset of muscle soreness before eccentric exercise</td>
</tr>
<tr>
<td>Community dwelling older adults</td>
<td>Russo et al.</td>
<td>2003</td>
<td>prevention of bone and muscle strength loss</td>
</tr>
<tr>
<td></td>
<td>Roelants et al.</td>
<td>2004</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Verschueren et al.</td>
<td>2004</td>
<td>enhance balance ability</td>
</tr>
<tr>
<td></td>
<td>Cheung et al.</td>
<td>2007</td>
<td></td>
</tr>
<tr>
<td>Institutionalised older adults</td>
<td>Bautmans et al.</td>
<td>2005</td>
<td>improve balance and mobility</td>
</tr>
<tr>
<td>Those recovering from injury or illness or living with chronic disease</td>
<td>van Nes et al.</td>
<td>2004</td>
<td>proprioceptive control in stroke patients</td>
</tr>
<tr>
<td></td>
<td>Turbanski et al.</td>
<td>2005</td>
<td>part of an intervention for Parkinson’s disease</td>
</tr>
<tr>
<td></td>
<td>Ahlborg et al.</td>
<td>2006</td>
<td>increase gross motor performance in adults with cerebral palsy</td>
</tr>
<tr>
<td></td>
<td>Tihanyi et al.</td>
<td>2007</td>
<td>increase neuromuscular performance of the lower limbs</td>
</tr>
</tbody>
</table>
2.2.2 Aetiology of Performance Improvement after WBV

Recent systematic reviews concluded that WBV activated the stretch reflex due to muscle spindle excitation (Norlund & Thorstensson, 2007) and may trigger hormone responses such as testosterone and growth hormone (Rehn, Lidström, Skoglund & Lindström, 2007). Whole-body vibration has also been shown to effect other mechanisms of the autonomic nervous system in single intervention studies. Various intensities of WBV increased peripheral circulation of the lower limbs when measured with Doppler ultrasound among healthy young adults (Kerschan-Schindl, Grampp, Henk, Resch, Preisinger, Fialka-Moser et al., 2001; Lythgo, Eser, de Groot & Galea, 2009).

Mechanistic explanations are hypothesised to lie in theoretical perspectives such as the Hoffmann-reflex (H-reflex) and stochastic resonance (SR). Interestingly, because many WBV interventions in the sport science field were aimed at affecting skeletal muscle, a neuromuscular explanation was sought in an attempt to identify the physiological mechanisms underpinning WBV. Neuromuscular activity, for example, has been determined indirectly by surface electromyography (EMG). Specifically, EMG results indicated enhanced motor unit recruitment associated with improvement in neuromuscular performance (Rittweger, Mutschelknauss & Felsenberg, 2003). As such, it was postulated that the stretch reflex was activated to elicit improvement during WBV (Rittweger, Mutschelknauss & Felsenberg, 2003).

Motoneuron excitability may also be quantified in an effort to describe WBV effects, yet investigations of a neurophysiologic nature remain incomplete. In attempts to quantify effects of WBV, researchers have elicited muscular activity by transcranial magnetic stimulation.
(TMS) (Mileva, Botwell & Kossev, 2009), resistance training, and direct measurement of neuromuscular activity with intramuscular electrodes (Kidgell, Sale & Semmler, 2006) and stimulation of the H-reflex (Hamada, Sale, MacDougall & Tarnopolsky, 2000; Palmieri, Hoffman & Ingersoll, 2002; Hoffman, Palmieri & Ingersoll, 2003).

Transcranial magnetic stimulation of the motor cortex activated spinal motoneurons of the lower limbs (Brouwer & Ashby, 1992; Chen, Tam, Bütefisch, Corwell, Ziemann, Rothwell et al., 1998). Such a method provided access to descriptions of motor-evoked potentials in healthy men after WBV (Mileva, Botwell & Kossev, 2009). During a bout of WBV, a static squat posture increased tibialis anterior muscle activity above static squatting alone, suggesting that WBV increased motoneuron excitability for that sample of healthy men. Whole-body vibration however, did not potentiate the stretch reflex of healthy young adults (Hopkins, Fredericks, Guyon, Parker, Gage, Feland et al., 2009). The stretch reflex is a somatic spinal reflex and is an involuntary action because the higher centre is located in the grey matter of the spinal cord (Jenkins, Kemnitz & Tortora, 2007; Tortoda & Derrickson, 2009). As such, for healthy young adults, WBV may have activated the stretch reflex during WBV (due to increased motoneuron excitability observed with TMS), but it did not potentiate the reflex once a WBV bout was completed (Hopkins et al., 2009).

Current knowledge of the physiological mechanisms affected by WBV remains imperfect. Therefore, it was necessary to further explore the major theories of WBV mechanistic responses from a broader area. The following sections described the: (1) motor cortex, (2) stretch reflex, (3) tonic vibration reflex, (4) H-reflex, and (5) stochastic resonance.
The Motor Cortex

The motor cortex, a component of the cerebral cortex of the brain, is responsible for perceiving and interpreting sensory information, making conscious decisions and controlling voluntary movements (Latash, 1998). Standing in a squat position, for example, requires voluntary movement, affected by gamma (γ) -motoneurons (figure 2.8). The γ-motoneurons regulate sensitivity of the muscle spindles for afferent alpha (α) -motoneurons (Felton & Józefowicz, 2004).

![Diagram of motor cortex](image)

**Figure 2.8.** The motor cortex showing how γ-motoneurons regulate sensitivity of a muscle spindle (modified from Latash, 1998, p. 84).

The process of such voluntary activity depends on γ activity (Latash, 1998). As such, an increase in γ activity represents a lengthening of muscle. Such activity may then lead to α-motoneuron activity, more commonly associated with the stretch reflex.
The Stretch Reflex

Maintenance of body posture is dependent on the ability of skeletal muscle to maintain muscle tone. Skeletal muscles maintain a slightly toned state in preparation for quick contraction required to attenuate changes in body position (Luttgens & Hamilton, 1997). When skeletal muscles experience a brief, unexpected increase in length, the reflex response is to shorten the muscle to avoid adverse events including injury or unstable body posture. For example, when the patella tendon is tapped with a patella reflex hammer, a seemingly instant and involuntary contraction of the quadriceps muscle group occurs resulting in knee extension.

The process of initial stimulation to reaction is theorised to occur in less than 40 milliseconds (Abernethy, Kippers, Mackinnon, Neal & Hanrahan, 1996; Martini, 1998). As such, the role of the stretch reflex is to detect and respond to changes in skeletal muscle length (Luttgens & Hamilton, 1997). The usual response is a concentric contraction of the stretched muscle. As such, both sensory and motor mechanisms must react within the stretch reflex (figure 2.9).

Figure 2.9. The stretch reflex (Latash, 1998, p. 65).
Skeletal muscle comprises both intrafusal and extrafusal fibres. Intrafusal fibres of the stretch reflex have a sensory function, whilst extrafusal fibres are responsible for muscular contraction (Punkt, 2002; Martini, 2006). Of intrafusal fibres, muscle spindles convert changes in muscle length and tension to nerve impulses that enter the spinal cord (Hamill & Knutzen, 1995).

Muscle spindle afferent neurons, Type Ia or Type II, synapse in the dorsal root of the spinal cord with an interneuron. At the spinal cord, the afferent α-motoneuron synapses with the interneuron resulting in stimulation of a motor unit (Shumway-Cook & Woollacott, 2001; Enoka, 2002; McBride, Porcari & Scheunke, 2004; Jordan, Norris, Smith & Herzog, 2005). The fibres also play an inhibitory role because they stimulate the antagonist muscle to relax, thus allowing the agonist to be the prime mover (Shumway-Cook & Woollacott, 2001); a function known as reciprocal inhibition (Marieb, 1995).

Thus, γ-motoneurons regulate sensitivity of the muscle spindles for afferent α-motoneurons during voluntary movement (Felton & Józefowicz, 2004). Subsequently, α-motoneurons regulate contraction of skeletal muscle to produce movement if the muscle spindle is stimulated due to a lengthening of skeletal muscle (figure 2.10).
Figure 2.10. The motor cortex and spinal region for $\gamma$-motoneuron and $\alpha$-motoneurons activity (modified from Latash, 1998, p. 84). The $\gamma$-motoneurons regulate sensitivity of the muscle spindles. The $\alpha$-motoneurons regulate contraction of skeletal muscle.

As shown in figure 2.10, both $\gamma$- and $\alpha$-motoneurons may be activated during a voluntary isometric contraction if some stimulus lengthens the muscle. Researchers concluded that WBV was such a stimulus (Norlund & Thorstensson, 2007). Direct measurement of motor unit activity however, has only recently been established with WBV using TMS (Mileva, Botwell & Kossev, 2009). That result is in contrast to previous research reporting that the TMS did not stimulate muscle spindle activity (Armstrong, Nestle, Grinnell, Cole, Van Gilder, Warren et al., 2008).

The effect of WBV on physiological mechanisms of the stretch reflex requires further research to advance the understanding of the effect of WBV. If adding WBV during an isometric squat activates muscle spindles and, for example, Type Ia afferent neurons, then
what is the role of the antagonist muscles, the hamstrings? Within the context of reciprocal inhibition, should EMG activity of the antagonist be affected by WBV? More research is required. Are both the agonist and antagonist simultaneously activated to the same extent during WBV? If so, then is reciprocal inhibition constrained? Speculation about physiological mechanisms of WBV may have led to statements such as the following: WBV can be used to identify the “hidden reserves” of an athlete to allow performance improvement (Issurin & Tenenbaum, 1999, p. 181).

**The Tonic Vibration Reflex**

The neural response to vibration of high frequency and low amplitude applied directly to muscle is the activation of the tonic vibration reflex (TVR) (Griffin, 1994; Latash, 1998). The TVR indicates increased muscular activity through a large number of motor units, compared with voluntary contraction (Latash, 1998). The TVR was activated with 30 Hz vibration frequency (Seidel, 1988), though it was suggested about 100 Hz was required (Latash, 1998). Later EMG activation was significantly enhanced after 30 Hz vibration of the biceps brachii, yet the authors did not confirm that the TVR was the attributing factor (Bosco, Cardinale & Tsarpela, 1999).

The neural pathway of the TVR resembles that of the stretch reflex. Since the TVR refers only to vibration applied to the muscle belly or tendon, by definition, it cannot be a contributing factor of WBV effects. Possibly for that reason, the stretch reflex was used to describe WBV effects.
The Hoffmann Reflex

The Hoffmann reflex (H-reflex) can be quantified in EMG waves, usually of the triceps surae (Griffin, 1994; Latash, 1998). The H-reflex is analogous with the stretch reflex, yet is activated by electrical stimulation of, for example, the posterior tibial nerve (Latash, 1998, Scaglioni, Narici, Maffiuletti, Pensini & Martin, 2003; Voerman, Gregorič & Hermens, 2005). Therefore, activation of the H-reflex bypasses the muscle spindle (Armstrong et al., 2008).

The H-reflex may be a contributor to WBV effects since it represents motoneuron excitability (Hamada et al., 2000; Palmieri, Hoffman & Ingersoll, 2002; Hoffman, Palmieri & Ingersoll, 2003). Recently WBV was shown to affected the H-reflex (Armstrong et al., 2008). Links of WBV to the H-reflex should be expected because, as previously mentioned, TMS elicited greater activity of tibialis anterior during WBV (Mileva, Botwell & Kossev, 2009). However, because the H-reflex is electrically stimulated rather that mechanically stimulated (i.e., WBV), the role of the H-reflex and WBV remains unclear. Furthermore, the general mechanistic effect of WBV on the human body remains speculative although exposure to WBV is linked to greater muscular activity (Cardinale & Lim, 2003; Abercromby et al., 2007a). Intuitively, three reflexes, that share a similar neural pathway, may be activated during WBV. However, only the H-reflex has been directly quantified, despite not activating muscle spindles.
Stochastic Resonance

Another theoretical perspective of vibration effects is stochastic resonance (SR). While barely discussed in WBV literature, SR may be causal to WBV effects. Theoretically, it is a “phenomenon whereby a nonlinear system can detect an otherwise undetectable stimulus with the addition of a random stimulus to the input” (Fallon & Morgan, 2005, p. 928). The ‘nonlinear system’ in this case is the human body and the ‘random stimulus’ is the WBV.

It was thought that ‘noise’ could interfere with the ability to process relevant information about a signal (Ward, Doesburg, Kitajo, MacLean & Roggeveen, 2006). However, if the noise generated a resonance in tune with the nonlinear system, then SR would elicit for example, an osteogenic response to low-amplitude broad frequency (0 to 50 Hz) vibration (Tanaka, Alam & Turner, 2003).

Stochastic resonance (SR) therefore, may enhance the human body response to WBV. Previous studies had established SR in the sensory system of humans, and only recently SR was identified in the motor system as well (Martinez, Pérez, Mirasso & Manjarrez, 2007). Interestingly, vibration platform frequency for maximal EMG activity of the vastus lateralis during WBV was different for individual young adults (Di Giminiani, Tihanyi, Safar & Scrimaglia, 2009). The frequency range was 20 to 55 Hz. Despite the absence of mechanistic discussion, it is possible that WBV elicited a SR for the vastus lateralis muscle that was unique to each individual.
Musculoskeletal pain after resistance training of healthy young adults reduced after ‘stochastic resonance whole-body vibration’ (Elfering, Thomann, Schade & Radlinger, 2011). A trend towards improved functional performance of the lower limbs among healthy older adults was also reported (Rogan, Radlinger, Schmid, Herren, Hilfiker & de Bruin, 2012; Roga, Hilfiker, Schmid & Radlinger, 2012) when WBV frequency was set at 5 Hz. No explanations were presented for possible mechanisms of reduced pain or the trend towards improved functional performance, nor to the potential that the ‘random stimulus’ that may have been too low.

### 2.2.3 Whole-Body Vibration Guidelines

Whole-body vibration interventions vary within existing literature. However, no published guidelines for safe and effective WBV exist. Nevertheless, the absence of guidelines has not diminished proliferation of development and use of vibration platforms in community settings such as health and fitness clubs.

The benefit of safe and effective WBV guidelines are salient, yet such knowledge remains poorly understood from a scientific perspective. Establishing guidelines may be difficult because of a multitude of variables than can be manipulated to affect the intensity/effect of WBV upon the body (table 2.11). Whole-body vibration interventions within the existing literature have used multiple manipulations of variables that may be broadly categorised as: (1) individual, (2) vibration platform, and (3) intervention.
Table 2.11

Components of WBV that can effect dependent variables regardless of population sub-group

<table>
<thead>
<tr>
<th>Category</th>
<th>Variable</th>
<th>Intensity / Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Individual</td>
<td>Transmission</td>
<td>WBV as a mode of exercise</td>
</tr>
<tr>
<td></td>
<td>Stance posture: Static and Dynamic</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Additional load</td>
<td></td>
</tr>
<tr>
<td>Platform</td>
<td>Frequency (Hz)</td>
<td>Gravitational force (g)</td>
</tr>
<tr>
<td></td>
<td>Peak-to-peak displacement (mm)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Stance width</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Vibration direction</td>
<td></td>
</tr>
<tr>
<td>Intervention</td>
<td>Time per bout (sec)</td>
<td>WBV exposure</td>
</tr>
<tr>
<td></td>
<td>Bouts per session</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Session per week</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Weeks of intervention</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Rest interval (sec)</td>
<td></td>
</tr>
</tbody>
</table>

Generally, access to WBV is only possible in health and fitness clubs. This may be due to several reasons, but most probably, that vibration platforms are beyond the financial capacity of individuals for home based use. It may also be that WBV is seen as a gimmick, as a review of advertising strategies might suggest. Regardless, it seems logical that before such time that vibration platforms become more readily available to the general community, guidelines for safety and effective methods should be established.

The creation of guidelines is a large task, and is envisaged to comprise knowledge of vast numbers of variables across many population sub-groups (i.e., table 2.11). However, the scope of this research was to identify a safe and effective method of WBV for people with COPD by establishing a safe, valid and reliable WBV intervention.
**Individual**

The stance posture adopted during WBV can alter the transmission and location of WBV about the body. Changing body posture (particularly the angle of knee flexion) can alter the transmission of WBV through various bony landmarks (Harazin & Grzesik, 1998). Typically, transmission is the ratio of vertical acceleration at the surface of the vibration platform to vertical acceleration at a specific landmark of the body. Wobbling mass refers to all non-rigid parts of the body, namely; muscles, soft tissues, fluids, and organs (Yue & Mester, 2002). Generally, a transmission value does not discriminate among wobbling mass and rigid bodies (Yue & Mester, 2002). However, the value can usefully quantify the rate of vibration attenuation among various landmarks and across populations.

Transmission of WBV in the sitting posture has been thoroughly researched, yet literature describing transmission in the standing posture is lacking. Perhaps another explanation for the imperfect knowledge of physiological mechanisms activated by stance alterations during WBV may be in part due to lack of knowledge of transmission of WBV through the standing body. The exact degree of transmission remained uncertain following one WBV intervention, despite claims the intervention caused high-frequency loading of the skeleton (Verschueren, Roelants, Delecluse, Swinnen, Vanderschueren & Boonen, 2004).

**Transmission**

The International Organisation for Standardisation devised a vibration exposure threshold based upon occupational environments (ISO-2631-1, 1997). When WBV was in the sitting position and about the spine and head, the threshold considered frequency, magnitude, direction, and daily duration of vibration, but does not specify a SI unit (ISO-2631-1, 1997).
Vibration exposure below a value of 17.0 was considered safe. The ISO recommendation has subsequently been used for a WBV intervention and the estimated vibration dose was above 17.0 (Abercromby, Amonette, Layne, McFalin, Hinman, & Paloski, 2007b). The healthy young adults stood on a vibration platform and were exposed to a gravitational force of 7.24 g.

The intervention of Abercromby et al. (2007b) violated ISO defined safe daily exposure to WBV. Given that interventions of many studies have used similar and larger gravitational forces and durations, it is probable that the ISO recommendation has been ignored or unknown. Conversely, the safety of long-term WBV interventions above an estimated vibration dose value of 17.0 can also be questioned because occupational vibration was shown to negatively affect normal bodily functions in healthy adults (Ishitake et al., 1998; Ishitake, et al., 2002). Abercromby et al. (2007b) suggested that since participants in their study stood with knee flexion between 10 and 35º, transmission attenuation occurred and thus, the posture may have reduced deleterious effects of WBV about the spine and head.

Other computation methods have been used to determine a transmission value. The ‘cross-spectral density function method’ (Paddan & Griffin, 1993), the ‘transmission factor’ (Mester, Spitzenfell, Schwarzer & Seifriz, 1999), and the ‘transmissibility transfer function’ (Rubin, Pope, Fritton, Magnusson, Hansson & McLeod, 2003). Independent of the computation method used, the collective data demonstrate differences among the use of vibration platform and body landmarks.
Few studies have described transmission of WBV when the participant stood on a vibration platform. Transmission of synchronous WBV was described after participants stood with 45 and 70° knee flexion (Harazin & Grzesik, 1998). Another design incorporated participants standing with the knees bent so that they were vertically aligned to the toes (Matsumoto & Griffin, 1998). It should be noted that Matsumoto and Griffin (1998) did not measure proximal nor distal limb lengths to screen for difference among participants. At approximately 20 Hz, both studies reported vertical transmissibility (VT) about the knee of ~1.0 for each stance postures. Essentially, the vertical acceleration of the vibration platform was the same as the vertical acceleration about the knee.

The results of those two studies can lead to some practical considerations. First, WBV with an intensity of approximately 20 Hz, independent of stance posture, was not attenuated before the knee. The magnitude of the vibration therefore, must be attenuated at some other locations above the knee. Second, because peak-to-peak displacement was different among the two studies (Harazin & Grzesik = 1.40 mm; Matsumoto & Griffin = 0.45 mm), the resultant acceleration of the vibration platform would have been 11.05 m.s\(^{-2}\) (1.13 g) compared with 3.55 m.s\(^{-2}\) (0.36 g). Thus, despite the large differences in magnitude, transmission of WBV was not affected.

At 31.5 Hz however, VT was larger when participants increased knee flexion to 45° (Harazin & Grzesik, 1998). The VT was < 1.0 at that frequency. Subsequently, vertical acceleration was attenuated about the knee. Furthermore, VT about the knee was greater as flexion increased. At that frequency however, the peak-to-peak displacement of the vibration platform was 0.40 mm. Thus, vertical acceleration was estimated to be 7.83 m.s\(^{-2}\) (0.80 g).
It is difficult to draw conclusions from existing literature due to the inconsistent methods within the Harazin and Grzesik (1998) study and among studies. Furthermore, the validity of the vibration platforms was not reported. It seems that vibration platform frequency above 20 Hz attenuates VT about the knee. It is possible that a subsequent increase in vertical acceleration would explain such an occurrence, yet at 31.5 Hz, vertical acceleration was less than at 20 Hz due to the different peak-to-peak displacements among the studies.

A negative VT value represents a higher vertical transmission about a body location than at the surface of the vibration platform. Such occurrences have not been reported suggesting that attenuation of WBV occurred throughout the body. It was reported that the attenuation of WBV can partially be understood by observations of wobbling mass and rigid bodies (Yue & Mester, 2002). Researchers have used varying mathematical models to describe impact forces on wobbling mass (Gruber, Ruder, Denoth & Schneider, 1998; Yue, Kleinöder & Mester, 2001; Gittoes, Brewin & Kerwin, 2006). However, to date, mathematical modelling has not been applied to WBV.

Transmission of WBV in the standing posture has been measured directly (Rubin, et al., 2003). Pins with accelerometers were surgically inserted in the spinous process of L4 and the greater trochanter of the left femur of six participants. The method was time consuming and invasive and limited by a possibly understandable low number of participants. Nevertheless, transmission of WBV to the spine and hip was greatly attenuated below the hip when the knees were flexed at 20°.
Less invasive estimates of transmission research also exist in the WBV literature. Three studies indirectly identified transmission of WBV with accelerometers at the knee in the frequency range of 20 to 30 Hz. Two studies incorporated synchronous WBV (Harazin & Grzesnik, 1998; Kiiski et al., 2008) while another incorporated side alternating WBV (Crewther, Cronin & Keogh, 2004). Comparison of attenuation with the Rubin et al. (2003) study was lacking, though the bone insertion method was thought to be the most accurate method of transmission quantification (Kiiski et al., 2008).

Of published work, one study examined gravitational forces of synchronous WBV when participants stood in 60º knee flexion and 3-5º knee flexion at 20 and 30 Hz (Crewther, Cronin & Keogh, 2004). Given that a transmission value was not computed, interpretation of the results was limited. While gravitational force about the knees was 3.91 g, it was not possible to determine the vibration platform frequency, peak-to-peak displacement and subsequent gravitational force. Furthermore, the stance posture adopted by the healthy young adults was not reported. Therefore, it is difficult to comment further on the findings of that paper, or compare side alternating WBV with synchronous WBV, suffice to suggest that further research is required to gain knowledge about transmission with side alternating WBV.

**Stance Posture**

Stance posture can be sub-classed as static and dynamic. For example, a static stance posture occurs when an individual stands with the knees fixed at 70º knee flexion during WBV. An example of dynamic posture occurs when an individual bends the knees through a specified range of motion during WBV resulting in the knee both flexing and extending. The major difference among the two stance postures is the muscular contraction type about a joint. A
static stance posture demands an isometric contraction of skeletal muscle, while the dynamic stance posture demands isotonic skeletal muscle contractions; both eccentric and concentric contraction of the flexors and extensors.

Without WBV, the EMG activity of the vastus lateralis during unloaded squatting was observed to increase between 21 and 63% of maximal voluntary contraction (MVC) (Isear, Erickson & Worrell, 1997). Both isometric and isokinetic (concentric/eccentric) resistance training affected performance of a self-paced 40-step stair climbing speed to the same degree in 30 older adults (mean age = 73 years) (Symons, Vandervoort, Rice, Overend & Marsh, 2005). Regarding repetition maximum (RM), 10 RM training on a dynamometer was effective independent of skeletal muscle contraction type (Symons et al., 2005) even though dynamic stance demanded more oxygen than static stance (Rittweger, Schiessl & Felsenberg, 2001). Whole-body vibration should not be compared with RM training due to the fundamental differences of the two exercise modes, yet interestingly, an isometric squat (55° knee flexion) during WBV increased EMG activity of vastus lateralis from between 53.0 and 58.8% of MVC in healthy young adults (Roelants, Verschueren, Delecluse, Levin & Stijnen, 2006). Higher EMG activity of the vastus lateralis was also observed during static stance with WBV compared with non-WBV (Cardinale & Lim, 2003) although MVC data were not recorded. Though EMG activity of the lower limbs increases with WBV, a paucity of publications limits conclusions, practical applications, and external validity.

Accordingly, eight weeks of static and dynamic squats during WBV (maximal gravitational force = 10.88 g) did not improve the knee flexor and extensor strength of older adults (Rees, Murphy & Watsford, 2008). No significant improvement was found in ankle dorsiflexor strength and power, despite earlier findings that WBV acutely increased tibialis anterior EMG
activity during static and dynamic squatting (Abercromby et al., 2007a). Increased EMG activity of the lower limbs had been attributed to muscle length and/or vibration platform frequency in earlier research (Cardinale & Lim, 2003). Later though, muscle length (i.e., stance posture) and vibration platform frequency failed to alter maximal knee joint extension moments (Savelberg, Keizer, & Meijer, 2007).

Additional loads have been used to theoretically increase the intensity of WBV via dynamic stance posture although results varied among studies. Additional load of between 35 and 40% of body weight placed about the hips of healthy young adults elicited greater oxygen uptake during WBV (Rittweger, Schiessl & Felsenberg, 2001). Yet functionally, additional load of 6 to 10 RM during WBV did not improve the counter movement jump among a similar sample population (Rønnestad, 2004).

Essentially, dependent variables of WBV can be affected by the individual via manipulating the stance posture and the load placed on the individual. Such manipulation will change the transmission of WBV through the body, with the theory that attenuation of WBV occurs with the knees flexed to 20°. Such attenuation was thought to involve muscular activity measured by EMG, yet in both WBV and non-WBV conditions, muscle contraction type did not affect strength, even though EMG activity and oxygen uptake increased. Despite a recent increase in the volume of WBV literature, the effects of WBV on functional performance remains limited to counter movement jump performance and maximal strength after WBV. As such, the effects of WBV with static and dynamic stance postures on functional performance of varying sample groups remain inconclusive.
Platform

**Vibration Platform Frequency (Hertz)**

Vibration platform frequency can be manipulated in a WBV intervention with many methods: (1) the frequency (Hz) is increased as the intervention progresses, (2) the frequency (Hz) is maintained at the same frequency for the duration of the intervention, (3) the frequency (Hz) is reduced as the intervention progresses, (4) the frequency (Hz) is both increased and decreased across the duration of the intervention, and (5) the frequency (Hz) is randomly assigned for each WBV session/bout across the duration of the intervention.

Methods one and two were most common in the literature. Typically, vibration platform frequency increased from 15 Hz to 40 Hz across the duration of interventions for healthy young and old adults (Torvinen et al., 2002; Roelants et al., 2004; Rietschel et al., 2008; Roth, Wust, Rawer, Schnabel, Armbrecht, Beller et al., 2008; Furness & Maschette 2009). Vibration platform frequency range was also similar, but inconsistent across studies that maintained frequency for healthy sample groups (Lamont, Cramer, Bemben, Shehab, Anderson & Bemben, 2008; Edge, Mündel, Weir & Cochrane, 2009; Jacobs & Burns, 2009; Stewart, Cochrane & Morton, 2009).

Evidence was equivocal for methods one and two, and may explain why WBV guidelines remain unknown. Reporting a justification of each method used would assist future research. It seems the ‘overload principle’ was used in WBV interventions. Such theory however, has never been justified with previous WBV literature. Progressive overload theory seems logical since the perceived general consensuses is that lower increasing to higher frequency will allow adaptation and familiarity, and reduce injury risk (Crewther, Cronin & Keogh, 2004).
Precision identifying ‘lower’ and ‘higher’ vibration platform frequency however, remains equivocal.

The potential effects of methods three (the frequency is reduced as the intervention progresses), four (the frequency is both increased and decreased across the duration of the intervention), and five (the frequency is randomly assigned for each WBV session/bout across the duration of the intervention) have not been investigated. It seems an oversight within the literature that all methods to manipulate vibration platform frequency have not been investigated. While such an investigation is necessary and should be conducted in the future, it was beyond the scope of this research for several reasons: (1) limited availability of resources, (2) availability of participants, and their health status, and (3) it is difficult to justify such research because limited knowledge of people with COPD and WBV persists.

Recently, optimal vibration platform frequency was determined in a healthy sample group for peak counter movement jump height. Estimated peak power was higher when the vibration platform was operating at 50 Hz in conjunction with 6.0 mm peak-to-peak displacement (Adams, Edwards, Serviette, Bedient, Huntsman, Jacobs et al., 2009). Gravitation force at those platform dynamics would have been 30.18 g. However, vibration platform frequency of 30 Hz in conjunction with 4.0 mm peak-to-peak displacement was as effective for estimated peak power (Adams et al., 2009). Gravitation force at those platform dynamics would have been 7.24 g. The authors did not offer an explanation.
It may be that vibration platform frequency of 30 Hz was large enough to elicit physiological mechanisms, independent of peak-to-peak displacement. It should be noted that the methods of the study (Adams et al., 2009) were not adequately reported to allow reproduction. For example, the rest interval among each WBV bout was not disclosed. Also, a residual WBV effect cannot be discounted. In previous work, at least 24 hours was used to ensure adequate washout (Cochrane, Legg & Hooker, 2004; Furness & Maschette, 2009). Also, for the 30.18 g protocol, estimated peak power increased from 3225.6 W to 3245.6 W, an increase of 20 W (0.62%). The practical benefits of such improvement may have been more valid if, for example, jump height was reported. Therefore, the clinical significance and subsequent external validity of the study could be questioned.

**Peak-to-Peak Displacement (mm)**

Accurate reporting of peak-to-peak displacement is difficult because it has not always precisely described. Part of the confusion is due to inconsistent nomenclature (Lorenzen, Maschette, Koh & Wilson, 2009). Nouns such as amplitude and peak-to-peak displacement have been used interchangeably, yet true definition differs. Given that the vibration platform amplitude is used to calculate gravitational force, inaccurate reporting of amplitude affects interpretation.

Both peak-to-peak displacement and amplitude were used to describe the same value (5.0 mm) (Delecluse, Roelants & Verschueren, 2003). It is impossible to determine what the 5.0 mm value represented. However, the corresponding gravitational force was either 16.10 g or 32.19 g when vibration platform frequency was 40 Hz. In contrast, the measured motion of the vibration platform with an accelerometer was reported to have gravitational forces of 2.28
to 5.09 g. It should be noted that the concurrent validity of the accelerometer was not reported, nor the method used to incorporate the accelerometer and the vibration platform. Hence, precision in reporting forces can be confusing.

It seems that study designs to specifically determine the effect of vibration platform peak-to-peak displacement are lacking. As such, researchers have been designing studies omitting justification of the peak-to-peak displacement (Lorenzen, Maschette, Koh & Wilson, 2009). In context with vibration platform frequency, more research is required to further understand WBV effects.

Research designs may be limited by the specifications of the type of vibration platform used. Typically, peak-to-peak displacement can be changed by two means. A button can be pressed for synchronous vibration platforms that changes the mechanisms within the vibration platform. Of side alternating vibration platforms, changing the stance width will change the peak-to-peak displacement. A prototype side alternating vibration platform has been designed so that peak-to-peak displacement could be manipulated without changing stance width (Furness & Maschette, 2009; Maschette, Lorenzen & Furness, 2009) (Appendix N).

**Stance Width**

Few studies have examined EMG activity of the lower limbs during different stance widths in healthy adults. Without WBV, electrical activity of the lower limbs was between 12 and 64% larger with narrow (10 cm) than wide (32 cm) heel stance (Henry, Fung & Horak, 2000). As such, greater leg muscle activity occurred during the narrow heel stance and may be partly attributed to a narrower base of support (Henry, Fung & Horak, 2000).
Where EMG activity increased during WBV compared with non-WBV conditions (Abercromby et al., 2007a; Hazell, Jakobi & Kenno, 2007; Roelants et al., 2006) stance width was manipulated with the aim to describe effects of peak-to-peak displacement, not EMG. As such, the effect of stance width and WBV on EMG of the lower limbs remains unknown. Knowledge of stance is important because WBV may potentially be more effective at eliciting electrical activity of the lower limbs when the heels are 10 cm apart rather than for example, 21 cm (Abercromby et al., 2007a). Furthermore, manipulating stance width may introduce extraneous variables such as muscular co-activation or different motor unit recruitment patterns therefore, potentially changing a training effect of WBV.

Vibration Direction

Direction of WBV can effect practical considerations. The only group of researchers to compare WBV direction, reported that side alternating and synchronous WBV affect EMG activity of the lower limbs differently (Abercromby et al., 2007a). Furthermore, synchronous WBV was potentially more harmful because acceleration about the head was significantly greater, mindful of ISO guidelines (Abercromby et al., 2007b). That is, the transmission of WBV to the head was greater with synchronous WBV. It should be noted that the participants of those investigations were very fit young adults (n = 16 astronauts). More research is needed with synchronous and side alternating vibration platforms, with particular attention to transmission of WBV through the body.
**Intervention**

The variation of past WBV intervention protocols persists in relatively recently published studies. Single bouts of WBV ranged from 30 seconds (Rhea & Kenn, 2009), to six minutes (Jacobs & Burns, 2009). Those investigations were designed to view acute effects of WBV. Typically, data collected immediately after, or within 30 minutes of a WBV bout/session were considered acute (Cardianle & Lim, 2003; Humphries, Warman, Purton, Doyle & Dugan, 2004).

Interventions designed to collect long-term WBV data have also varied. Long-term data are recorded after a series of WBV sessions over an extended period (Luo, McNamara & Moran, 2005). As such, interventions have varied in length from five weeks to one year (Rønnestad, 2004; Bogaerts et al., 2007; Lamont et al., 2008; Di Giminiani et al., 2009). Such variance in intervention design does not allow a dose response explanation since outcome variables and study designs differ too greatly to draw meaningful conclusions.

For studies examining the long-term effects of WBV, another consideration for data are the acute/residual effects of WBV. A minimum 24 hour rest period was thought prudent to overcome residual effects of WBV (Roelants, Delecluse & Verschueren, 2004). Data collected before that time are considered acute. Recently, data were collected at least 48 hours after WBV to ensure long-term data responses were profiled (Cochrane, Legg & Hooker, 2004; Furness & Maschette, 2009).
Table 2.12 shows some of the most cited papers of WBV and the methods of each (cited 144 to 300 times). Table 2.13 shows subsequent publications. It seems that WBV interventions rarely coincide on time per bout, bouts per session, sessions per week, weeks of intervention, and rest intervals.

The range of participant number, age, maximal gravitational force, and maximum exposure are shown in table 2.14. Selected data were of commonly referenced and subsequent published WBV interventions (i.e., table 2.12 and table 2.13). Age range falls into the ‘young adulthood’ category (Gallahue & Ozmun, 2006). There is a large range of both maximum gravitational force and maximum WBV exposure time. Such range may highlight the lack of effective WBV guidelines.
Table 2.12
Commonly referenced published studies of WBV

<table>
<thead>
<tr>
<th>Authors</th>
<th>RCT</th>
<th>Participants</th>
<th>WBV Platform</th>
<th>Maximal gravitational force</th>
<th>WBV Duration</th>
<th>Protocol</th>
<th>Dependent variables</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bosco et al., (2000)</td>
<td>no</td>
<td>25.1 years</td>
<td>14</td>
<td>0% synchronous</td>
<td>17 g</td>
<td>60 sec, 60 sec rest</td>
<td>Isometric squat 100° knee flexion EMG of lower limbs and CMJ</td>
</tr>
<tr>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td>between</td>
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<tr>
<td>Cardinale &amp; Lim (2003)</td>
<td>no</td>
<td>23.5 years</td>
<td>16</td>
<td>100% unknown</td>
<td>50.33 g</td>
<td>60 sec, 60 sec rest</td>
<td>Isometric squat 100° knee flexion EMG of lower limbs</td>
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<tr>
<td></td>
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<td>between</td>
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<tr>
<td>Rittweger et al., (2003)</td>
<td>no</td>
<td>21.8 – 24.4 years</td>
<td>19</td>
<td>52.6% side alternating</td>
<td>16.32 g</td>
<td>unknown</td>
<td>Dynamic squats plus 40% body weight Blood lactate Jump height RPE</td>
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<tr>
<td>Torvinen et al., (2003)</td>
<td>yes</td>
<td>19 – 38 years</td>
<td>56</td>
<td>63% unknown</td>
<td>8.12 g</td>
<td>4 minutes</td>
<td>Dynamic squats Bone via DEXA, and pQCT CMJ Shuttle run</td>
</tr>
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</tbody>
</table>

Note: unknown: information regarding was not disclosed. RCT: randomised controlled trial. EMG: electromyography. CMJ: counter movement jump. RPE: Borg’s rating of perceived exertion. DEXA: dual-emission X-ray absorptiometry. pQCT: peripheral quantitative computed tomography.
Table 2.13

Examples of published studies of WBV

<table>
<thead>
<tr>
<th>Authors</th>
<th>RCT</th>
<th>Participants</th>
<th>WBV</th>
<th>Maximal gravitational force</th>
<th>WBV Duration</th>
<th>Protocol</th>
<th>Dependent variables</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adams et al., (2009)</td>
<td>no</td>
<td>23 – 39 years</td>
<td>n 20</td>
<td>Female 45%</td>
<td>synchronous</td>
<td>30.18 g</td>
<td>60 sec</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>2.27 rads</td>
<td></td>
</tr>
<tr>
<td>Di Giminiani et al., (2009)</td>
<td>yes</td>
<td>22.0 years</td>
<td>n 33</td>
<td>Female 55%</td>
<td>synchronous</td>
<td>12.17 g</td>
<td>60 sec, 60 sec rest between</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>90° knee flexion</td>
<td></td>
</tr>
<tr>
<td>Jacobs &amp; Burns (2009)</td>
<td>no</td>
<td>28.6 years</td>
<td>n 20</td>
<td>Female 50%</td>
<td>side alternating</td>
<td>unknown</td>
<td>6 minutes</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0 – 5° knee flexion</td>
<td></td>
</tr>
<tr>
<td>Lamont et al., (2009)</td>
<td>yes</td>
<td>20 – 30 years</td>
<td>n 36</td>
<td>Female 0%</td>
<td>synchronous</td>
<td>30.18 g</td>
<td>30 sec, 60 sec rest</td>
</tr>
<tr>
<td></td>
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</tr>
<tr>
<td>Rhea et al., (2009)</td>
<td>no</td>
<td>22.6 years</td>
<td>n 16</td>
<td>Female 0%</td>
<td>unknown</td>
<td>9.86 g</td>
<td>30 sec</td>
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</tbody>
</table>

Note: unknown: information regarding was not disclosed. RCT: randomised controlled trial. EMG: electromyography. HR: heart rate.
Table 2.14

Range of various variables for WBV interventions (tables 2.12 and 2.13)

<table>
<thead>
<tr>
<th></th>
<th>Age</th>
<th>Gravitational force</th>
<th>WBV Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>14-56</td>
<td>19-39</td>
<td>8.12 – 50.33 g</td>
<td>30 sec – 6 min</td>
</tr>
</tbody>
</table>

2.2.4 Whole-Body Vibration for Sub-Optimal Health Populations and Functional Performance

A sub-optimal health population, for the purpose of this research, is defined as any sample population with pathology. Older age is not considered sub-optimal, but for example, women with low bone mineral density or individuals of residential aged care (i.e., non-community dwelling) are considered as sub-optimal health sample groups. The volume of published literature of WBV as an exercise/rehabilitation intervention for sub-optimal health populations is small. The dearth is obvious when searching for papers of COPD and WBV, since only one published paper could be found. Possible explanations for the limited research include availability of equipment and knowledgeable/skilled staff, inconsistent finding in the body of WBV literature for perceived safe and effective protocols, and the absence of WBV guidelines for both acute and long-term effects.

The earliest intervention of WBV and a sub-optimal health sample was of chronic lower back pain (Rittweger, Just, Kautzsch, Reeg & Felsenberg, 2002). A more recent intervention used WBV as rehabilitation for Parkinson’s disease (Ebersbach, Edler, Kaufhold & Wissel, 2008). All other published interventions of WBV and sub-optimal health samples are listed in table 2.15 (inclusive of publication year 2008). A study of children and adolescents with
osteogenesis imperfecta in which WBV was delivered in an inclined position (i.e., the body was not perpendicular to the vibration platform) (Semler, Fricke, Vezyroglou, Stark, Stabrey & Schoenau, 2008), and a study of stroke patients who received WBV while balancing their body weight over one limb (Tihanyi, Horváth, Fazekas, Hortobágyi & Tihanyi, 2007) were excluded because the research directions were beyond the scope of this research.

Since 2009 and inclusive of January 2012, an additional 25 papers were published on WBV and sub-optimal health sample populations, highlighting the increased trend of WBV research of clinical populations. The majority of papers were of people with spinal cord injury (Ness & Field-Fote, 2009; Davis, Sanborn, Nichols, Bazett-Jones & Dugan, 2010; Sayenko, Masani, Alizadeh-Meghrazi, Popovic & Craven, 2010; Herrero, Menéndez, Gil, Martín, Martín García-López et al., 2011), multiple sclerosis (Schyns, Paul, Finlay, Ferguson & Noble, 2009; Broekmans, Roelants, Alders, Feys, Herbert & Eijnde, 2010; Wunderer, Schabrun & Chipchase, 2010) and fibromyalgia (Alentorn-Geli, Moras, Padilla, Fernández-Solà, Bennett, Lázaro-Haro et al., 2008; Sañudo, de Hoyo, Carrasco, McVeigh, Corral, Cabeza et al., 2010; Olivares, Gusi, Parraca, Adsuar & Del Pozo-Cruz, 2011).
Table 2.15

All published research of WBV and sub-optimal health populations

<table>
<thead>
<tr>
<th>Cohort Condition</th>
<th>Authors</th>
<th>Year</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cerebral palsy</td>
<td>Ahlborg et al.</td>
<td>2006</td>
</tr>
<tr>
<td>Cystic fibrosis</td>
<td>Rietschel et al.</td>
<td>2008</td>
</tr>
<tr>
<td></td>
<td>Roth et al.</td>
<td>2008</td>
</tr>
<tr>
<td>Heart transplant recipients</td>
<td>Crevenna et al.</td>
<td>2003</td>
</tr>
<tr>
<td>Multiple sclerosis patients</td>
<td>Schuhfried et al.</td>
<td>2005</td>
</tr>
<tr>
<td></td>
<td>Jackson et al.</td>
<td>2008</td>
</tr>
<tr>
<td>Nursing home residents</td>
<td>Bruyere et al.</td>
<td>2005</td>
</tr>
<tr>
<td>Parkinson’s disease</td>
<td>Turbanski et al.</td>
<td>2005</td>
</tr>
<tr>
<td>Stroke patients</td>
<td>van Nes et al.</td>
<td>2004</td>
</tr>
<tr>
<td></td>
<td>van Nes et al.</td>
<td>2006</td>
</tr>
<tr>
<td>Women with low bone mineral density</td>
<td>Gilsanz et al.</td>
<td>2006</td>
</tr>
<tr>
<td>Woman with fibromyalgia</td>
<td>Alentorn-Geli et al.</td>
<td>2008</td>
</tr>
</tbody>
</table>

Of all literature, adverse or negative effects of WBV are rarely reported. Crevenna, Fialka-Moser, Rödler, Keilani, Zöch, Nuhr et al. (2003) considered WBV performed to ‘subjective exhaustion’ safe for heart transplant recipients as soon as six months post surgery. The average WBV exposure time was 248 consecutive seconds (4.08 g), while heart rates rose from 98 beats.min\(^{-1}\) at baseline, to 121 beats.min\(^{-1}\) at subjective exhaustion.
One paper, published in 2012, was designed to investigate WBV for people with COPD as a mode of exercise to improve muscular strength, muscular power and functional performance of the lower limbs. In conjunction with traditional pulmonary rehabilitation, dynamic squats during WBV elicited a “striking” improvement in functional performance quantified with the six-minute walk test compared with traditional rehabilitation and dynamic squats without WBV (Gloeckl et al., 2012, p 78). The three week intervention caused six of 42 participants (14%) to drop out of the WBV intervention due to acute exacerbations of COPD (n = 5) and acute gonarthrosis (n = 1). Interestingly, WBV was proposed to beneficially affect gonarthrosis (Stein, Knoell, Faymonville, Kaulhausen, Siewe, Otto et al., 2010), though equivocal evidence of WBV for bone health persisted (Wysocki, Butler, Shamliyan & Kane, 2011).

Maintenance of ADLs was essential for people with COPD (GOLD, 2011). Effects of WBV on ADLs and functional performance however, remained unknown since the intervention was not completed independent to other modes of pulmonary rehabilitation (Gloeckl et al., 2012). Furthermore, the effects of a community based WBV intervention on valid and reliable field tests of ADLs such as the TUG test and the 5-chair test, or kinematic variables of gait remain unknown.

Although the effects of WBV on people with COPD have rarely been investigated, it was important to draw reference of previous published work of different sub-optimal health populations. For example, a WBV intervention was well tolerated by participants with cystic fibrosis (Roth et al., 2008). That is, six months of WBV did not cause exacerbations of cystic fibrosis, nor as expected, did it alter lung function. Subsequently, those findings were confirmed by results of another cystic fibrosis group (Rietschel et al., 2008).
Considering the limited body of literature of WBV and sub-optimal health populations, only speculative conclusions could be drawn. However, given that WBV had not negatively altered lung function, it should not be considered a component of pulmonary rehabilitation for lung function rehabilitation. It also seems that WBV is safe because it did not elicit exacerbations of pathology, even when exercise is performed to subjective exhaustion for heart transplant patients. It remains unknown however, if a long-term community based WBV intervention will cause acute exacerbations in people with COPD or improve/maintain exercise tolerance and functional performance of the lower limbs.

2.3 Summary and Significance of this Research Question

Whole-body vibration is used as a mode of exercise to impact the musculoskeletal, endocrine and cardiovascular systems of the human body. Researchers can choose to use several different vibration platform designs to expose participants to WBV. As a consequence, both effective and ineffective results after WBV have been reported. However, incidences of negative effects of WBV have been rarely reported.

Considering the growing WBV literature base, it is an oversight that guidelines for safe and effective WBV have not been established, despite increased popularity of vibration platforms in the health and fitness industry. However, it seems that many variables of a WBV intervention can be manipulated. Therefore, comparison among studies and subsequent guideline recommendations remains challenging. Further research is required to thoroughly understand WBV effects.
Transmission of WBV through the body for example, needs to be established for different vibration platforms, frequencies, peak-to-peak displacements, stance postures and widths, and sample groups. Electrical activity of skeletal muscle of the lower limbs should also be examined. Such information may assist in establishing both acute and long-term WBV protocols that primarily affects the wobbling mass of lower limbs rather than, for example, the rigid bodies of the spine and head.

Whole-body vibration is becoming increasingly used as an exercise intervention for people with sub-optimal health. Whole-body vibration has to date, limited application as an exercise intervention for people with COPD. As mentioned, further research is required in all sample populations. However, from the existing literature base of sub-optimal health populations, WBV rarely caused exacerbations.

A WBV intervention should be associated with safe exercise practices before it can be more widely incorporated into the lifestyle regimes of population sub-groups, especially those of sub-optimal health. Encouragingly, WBV did not negatively affect heart transplant recipients or people with cystic fibrosis. Whole-body vibration increased heart rate and oxygen consumption but did not affect FEV₁ or increase exacerbations. Current findings can only partially justify the use of WBV to improve or maintain exercise tolerance and functional performance of the lower limbs of people with COPD.

Despite existing knowledge of WBV, as a standalone mode of exercise, efficacy has not been established for improvement or maintenance of exercise tolerance and functional performance of the lower limbs of people with COPD. Furthermore, it remains unknown if a long-term
community based WBV intervention would elicit exacerbations of COPD, such as increased dyspnoea or reduced saturation of haemoglobin, or if the WBV intervention will be well tolerated and adhered to by community dwelling adults with COPD.

Future research of effects of WBV on exercise tolerance and functional performance of the lower limbs should be confirmed with robust research designs such as randomised controlled trials of people with COPD. However, before long-term interventions of effect of WBV on exercise tolerance and functional performance of the lower limbs can be conducted, safety and efficacy of WBV as a standalone intervention should first be established. As such, the research design of this project was established to first describe safety, and then efficacy of WBV for people with COPD. Since WBV had never been used as a standalone intervention in a community setting for people with COPD, this research design incorporated a non-randomised crossover placebo design to answer the question “Will WBV improve or maintain exercise tolerance and functional performance of the lower limbs of people with COPD?”

2.4 Research Hypotheses

The following research hypotheses were tested to determine and efficacy of WBV on exercise tolerance and functional performance of the lower limbs of people with COPD:

**Exercise Tolerance**

H$_i$: For people with COPD, a six week WBV intervention; two sessions per week of five 60 second bouts interspersed with 60 seconds passive rest will: (1) acutely increase dyspnoea during a WBV bout, (2) not elicit a long-term change of dyspnoea quantified with the Borg CR-10 visual analogue scale, (3) acutely increase heart rate
and reduce saturation of haemoglobin during a WBV bout, and (4) not elicit a long-term change of heart rate and saturation of haemoglobin.

H₁: For people with COPD, there will be a difference among a six week WBV intervention and a six week PLACEBO intervention (two sessions per week of five 60 second bouts interspersed with 60 seconds passive rest) for dyspnoea, heart rate and saturation of haemoglobin.

**Functional Performance**

H₁: For people with COPD, a six week WBV intervention; two sessions per week of five 60 second bouts interspersed with 60 seconds passive rest will improve: (1) performance of the timed-up-and-go test, (2) performance of the 5-chair stands test, and (3) stride time, stride length and stride velocity quantified with a portable electronic walkway.

H₁: For people with COPD, a six week WBV intervention will improve functional performance compared with a six week PLACEBO intervention (each intervention; two sessions per week of five 60 second bouts interspersed with 60 seconds passive rest).
CHAPTER 3. METHODS

3.1 Overview

This chapter comprises the selected procedures and protocols relevant to the pilot tests targeted for this research. Following the review of literature, three essential pilot studies were conducted to: (1) validate a vibration platform, (2) describe transmission of WBV about the knee of healthy young adults, and (3) describe reliability of selected psychological and physiological dependent variables of exercise tolerance and functional performance of the lower limbs of people with COPD. The third pilot study (section 3.4) described the shared methods of participant selection criteria, selection protocol, instruments and data collection of people with COPD used for Chapter 4 and Chapter 5.

Given that effects of WBV on people in a community setting with COPD had never been investigated, and in keeping with the ‘first do no harm’ philosophy of research, the first major study: safety of WBV for people with COPD was completed and described in Chapter 4. The second major study: to describe efficacy of WBV to improve or maintain exercise tolerance and functional performance of the lower limbs of people with COPD was completed and described in Chapter 5. Sample size calculations of major study 2 are described in section 3.5 of this chapter. Common statistical procedures of Chapter 4 and Chapter 5 were described in the final section of this chapter (section 3.6). The design of this research is shown in figure 3.1.
Efficacy of Whole-Body Vibration on Exercise Tolerance and Functional Performance of the Lower Limbs of People with Chronic Obstructive Pulmonary Disease

Pilot Testing (Chapter 3)
- Validity of a vibration platform
- Transmission of WBV
- Reliability of dependent variables
- Statistical methods

MAJOR STUDY 1 (Chapter 4)
Safety of WBV for people with COPD

MAJOR STUDY 2 (Chapter 5)
Efficacy of WBV for people with COPD

Figure 3.1. Overview of the design of this research to describe efficacy of WBV on exercise tolerance and functional performance of the lower limbs of people with COPD.
3.2 Pilot Test 1: Validity of a Vibration Platform

Overview

This pilot test was completed to validate the frequency (Hz) of the vibration platform to be used in the major studies of this research. This pilot test was presented in 2009 at the 7th Australasian Biomechanics Conference (Appendix A), and was based upon a previous project conducted by the student researcher to quantify vibration platform frequency (presented at the Australian Conference of Science and Medicine in Sport 2009, Appendix B). The major finding of the previous project (Appendix B) was that manufacturer specifications of vibration platform frequency could be validated with advanced and precise laboratory based biomechanical equipment. For research therefore, manufacturer specifications of the vibration platform should be confirmed in the laboratory to allow accurate description of frequency.

Introduction

Depending on specifications of a vibration platform, frequency (Hz) may be manipulated to alter intensity (g force) of WBV. Frequency needs to be accurately reported in literature to strengthen the rigour required to allow replication in laboratory and community settings. Measurement of frequency can occur in the laboratory with three-dimensional (3D) motion analysis systems, but portable, light weight and inexpensive measurement instruments are desired in community settings. The purpose of this pilot test therefore, is to concurrently validate a portable accelerometer with a 3D motion analysis system to: (1) quantify the range of frequency of the vibration platform at pre-determined speeds, and (2) determine if an accelerometer can be used to establish concurrent validity and potentially allow its use in community settings to quantify vibration platform frequency.
Methods

Instruments and Test Procedure

A side alternating vibration platform was used (Vibro-Trainer, Amazing Super Health, AUS). Vertical ($F_z$) data were concurrently recorded at 500 Hz with: (1) a 3D motion analysis system (VICON Oxford Metrics, Oxford UK, six cameras), and (2) a tri-axial accelerometer (CXL25GP3, Crossbow Technology, San Jose, USA) (figure 3.2). The accelerometer was placed at the lateral end of the vibration platform surface, ~ 26 cm from the axis of rotation. Small (1 cm diameter) reflective ball markers were placed at both lateral ends of the vibration platform (figure 3.3). Data were analysed for five seconds during steady-state operation of the vibration platform for three common vibration platform frequencies: 20, 25 and 30 Hz.

Figure 3.2. A tri-axial accelerometer (CXL25GP3, Crossbow Technology, San Jose, USA) and a 3D motion analysis camera (VICON Oxford Metrics, Oxford UK). The images are not to scale.
Figure 3.3. Superior view of a vibration platform showing locations of reflective markers and the accelerometer. The reflective markers are identified by the 3D motion analysis system.

Statistical Procedures

To quantify vibration platform frequency, descriptive statistics of mean and standard deviation were computed for 20, 25 and 30 Hz. Concurrent validity was described with 95% limits of agreement and shown with Bland-Altman plots. Descriptive statistics were computed with the Statistical Package for Social Scientists (SPSS) 17.0 for Windows (SPSS Inc., Chicago, USA).
Results

Descriptive statistics are shown in table 3.1. The 95% limits of agreement are shown in figure 3.4. The difference in vibration platform frequency was less than 0.50 Hz among a 3D motion analysis system and a tri-axial accelerometer. To the nearest whole number, manufacturer determined vibration platform frequency was the same as those quantified with a 3D motion analysis system and a tri-axial accelerometer. The 95% limit of agreement was within ± 2.1 Hz for vibration platform frequency. As such, if the vibration platform was operating at 25 Hz, and the peak-to-peak displacement was 2.0 mm, then the gravitational force would be between 2.1 g and 3.0 g.

Table 3.1

Descriptive statistics and 95% limit of agreement among VICON and an accelerometer for various manufacturer defined vibration platform frequencies

<table>
<thead>
<tr>
<th>Manufacturer determined Hz</th>
<th>VICON Mean</th>
<th>VICON SD</th>
<th>Accelerometer Mean</th>
<th>Accelerometer SD</th>
<th>Mean difference (VICON-Accelerometer)</th>
<th>95% Limit of Agreement</th>
</tr>
</thead>
<tbody>
<tr>
<td>20</td>
<td>19.9</td>
<td>0.2</td>
<td>19.7</td>
<td>0.5</td>
<td>0.2 Hz</td>
<td>-2.1 Hz and 2.1 Hz</td>
</tr>
<tr>
<td>25</td>
<td>24.9</td>
<td>0.4</td>
<td>24.5</td>
<td>1.9</td>
<td>0.4 Hz</td>
<td></td>
</tr>
<tr>
<td>30</td>
<td>29.8</td>
<td>0.8</td>
<td>30.2</td>
<td>0.9</td>
<td>-0.4 Hz</td>
<td></td>
</tr>
</tbody>
</table>
Figure 3.4. Bland-Altman plot showing the 95% limit of agreement among VICON 3D motional analysis system (VICON Oxford Metrics, Oxford UK) and a tri-axial accelerometer (CXL25GP3, Crossbow Technology, San Jose, USA).

**Discussion and Summary**

Results of this pilot test show that manufacturer defined vibration platform specifications and frequency quantified with laboratory and field based instruments are similar. Furthermore, concurrent validity of a tri-axial accelerometer and a 3D motion analysis system was acceptable. If laboratory conditions can be replicated in a community setting, such concurrent validity may allow data collection with a tri-axial accelerometer to quantify vibration platform frequency or other components of vibration description such as transmission and acceleration of WBV about the vibration platform and various locations about the human body.
3.3 Pilot Test 2: Transmission of WBV about the Knee of Healthy Young Adults

Overview

This phase of pilot testing was a preliminary investigation into transmission of side alternating WBV. Given that the major aim of this research is to describe efficacy of WBV for people with COPD (i.e., major study 2, Chapter 5), it was considered crucial to enhance the level of understanding of transmission and side alternating WBV. Results of this phase of the pilot testing were presented in 2010 at the 28th International Conference on Biomechanics in Sport (Appendix C).

The data reported in this section describe transmission of WBV when the vibration platform frequency was 25 Hz and the peak-to-peak displacement of the vibration platform was 2.0 mm even though the test procedure describes other, more detailed data collection. The vibration platform descriptors (25 Hz and 2.0 mm) were selected to be inclusive of anticipated descriptors of major studies 1 and 2 (Chapter 4 and Chapter 5). The entire data collection protocol of the pilot test however, is reported to show the effort afforded to control for intervention bias and a potential learning effect. Specifically, there were three independent variables: (1) vibration platform frequency (20, 25 and 30 Hz), (2) stance width (10 and 20 cm), and (3) knee flexion angle (20, 40 and 60º).

Introduction

The mechanistic understanding of improved performance following WBV is uncertain. For example, transmission of WBV throughout the body is not thoroughly understood. Although increasingly available in gymnasiums and rehabilitation centres, the evidence base of WBV for transmission lacks empirical data. A search of existing literature showed transmission had
only been investigated for side alternating WBV among healthy young adults (Abercromby et al., 2007b; Crewther, Cronin & Keogh, 2004) (Chapter 2, section 2.2.3). As such, a greater understanding of transmission and attenuation of WBV with a side alternating vibration platform is required to assist with WBV intervention planning for people with COPD.

Previously, transmission was calculated by placing accelerometers on a vibration platform and the knee (Harazin & Grzeski, 1998), head (Abercromby et al., 2007a), tibial shaft and thigh (Cook, Mileva, James, Zaidell, Goss, & Botwell, 2011), pelvis and spine (Mansfield & Griffin, 2002) or directly into bone (Rubin et al., 2003; Nsiah Edwards, Meardon, & Ward, 2006). Some methods were more invasive than others, yet an acknowledged error was created when accelerometers were attached to skin. A data correction method to eliminate effects of local tissue-accelerometer resonance from surface measurements of vibration about the spine was proposed (Kitazaki & Griffin, 1995), yet since then, the method has not been routinely used during research.

Generally, peak-to-peak displacement and vibration platform frequency were manipulated in WBV interventions to alter physiological responses, though a consensus of specifications has not been attained. Nevertheless, for side alternating vibration platforms, changing stance width about the sagittal axis of rotation can change peak-to-peak displacement. Currently, no published evidence exists of transmission of WBV for different stance width when participants are using a side alternating vibration platform, even though stance width altered EMG activity of the lower limbs among healthy young adults (Henry, Fung & Horak, 2000).
Typically, vibration platform frequency is manipulated in an intervention by either increasing the frequency (Hertz) as the intervention progressed (Torvinen et al., 2002; Roth et al., 2008), or by maintaining the frequency for the duration of the intervention (Jacobs & Burns, 2009; Stewart et al., 2009). Regardless of method, the vibration platform frequency range to date has been 15 to 50 Hz. Investigations of transmission of WBV therefore, should quantify transmission in a similar range.

Two studies have identified transmission of WBV with accelerometers at the knee in the frequency range of 20 to 30 Hz with synchronous vibration platforms (Harazin & Grzesik, 1998; Kiiski et al., 2008). Accelerometer results showed WBV was attenuated to some extent about the knee, though dependent on vibration platform frequency and knee flexion angle. Accelerometer assessments using a side alternating vibration platform are yet to be published for transmission about the knee.

Knee flexion angle during WBV alters transmission of vibration when standing upon a synchronous platform (Harazin & Grzesik, 1998). Previously, knee flexion angle was investigated at for example, 20 (Rubin et al., 2003), 31-35 (Abercromby et al., 2007b) and 70\(^\circ\) (Harazin & Grzesik, 1998). Effects of knee flexion angle upon attenuation about the knee with a side alternating vibration platform however, are unknown.

Recently, noise of skin-mounted accelerometers was shown to minimally impact acceleration quantified during gait compared with bone-mounted accelerometers (Nsiah et al., 2006). As such, skin-mounted accelerometers were considered a good predictor of skeletal impact accelerations. Alternatively, 3D motion analysis systems were used to quantify transmission
of WBV in order to eliminate error associated with skin-mounted accelerometers (Smith Bressel, & Snyder, 2009). For interventions in a community setting, laboratory procedures and access to complex equipment to identify precision are often unsuitable. Therefore, research based in a community setting generally acknowledges the limitation (i.e., noise) of skin-mounted accelerometers but persist with their use because of ease of operation, cost, and transportability.

The purpose of this pilot test therefore, was to quantify transmission of WBV about the knee of healthy young adults to determine the most effective knee flexion angle for vibration attenuation. If the methods used in the pilot test allow quantification of transmission, then the methods could be adopted and replicated in a community setting to subsequently quantify transmission about the knee of people living independently with COPD.

Methods

Participants

Fifteen healthy, recreationally active males and females (mean ± SD age = 20 ± 1 years, stature = 1.8 ± 0.1 metres, mass = 70.5 ± 10.6 kilograms) (table 3.2) provided informed voluntary consent to participate in the pilot test. Participants were free from lower limb injury in the month prior to testing and had no known articular joint injuries. A convenient sample was recruited from the undergraduate degree within the School of Exercise Science, Melbourne. Data were collected in the postgraduate laboratories within the School of Exercise Science in 2009. The project was approved by the Australian Catholic University Human Research Ethics Committee (Appendix D).
Test Procedure

Each participant received six bouts of side alternating WBV delivered by a sinusoidal oscillating vibration platform (Vibro-Trainer, Amazing Super Health, AUS). Each bout lasted 60 seconds and consisted of a predetermined stance posture (i.e., knee flexion angle) and stance width (i.e., peak-to-peak displacement) while vibration platform frequency was randomly assigned. A minimum of 60 seconds of passive rest occurred between bouts. Stance posture consisted of 20, 40 and 60º knee flexion, for which 0º knee flexion corresponded with full knee extension (figure 3.5). Stance width was 10 and 20 cm from the axis of rotation for each leg as measured to the second toe. As such, the peak-to-peak displacement was 1.0 and 2.0 mm respectively. Vibration platform frequencies were 20, 25 and 30 Hz (cognisant of the findings of pilot study 1). Data were recorded for five seconds at each vibration platform frequency after platform steady-state was achieved.

Figure 3.5. Demonstration of 40º knee flexion angle upon the vibration platform. The accelerometer can be seen at the knee and on the vibration platform.
The independent variables were: (1) vibration platform frequency (20, 25, and 30 Hz), (2) stance width (10, and 20 cm), and knee flexion angle (20, 40, and 60º). The dependent variable, transmission, was calculated from the ratio of root mean square (RMS) knee acceleration (K_{RMS}) to RMS platform (P_{RMS}). A transmission value of 1.00 represented parity among the platform and knee. A transmission value less than 1.00 represented a larger P_{RMS} than K_{RMS} (figure 3.6).

Figure 3.6. An example of transmission, where the maximum acceleration about the knee is less than the maximum acceleration of the platform. For this example, the transmission value would be < 1.00. Note, data are m.sec^{-2} rather than RMS because negative values are squared when calculating RMS values.
Instruments

Two 25 g tri-axial accelerometers (CXL25GP3, Crossbow Technology, San Jose, USA) sampling at 250 Hz were used to quantify: (1) vibration platform vertical acceleration, and (2) knee vertical acceleration. The mass of each accelerometer was 46 gm. One accelerometer was mounted to the vibration platform. Another accelerometer was firmly taped to the centre of the left patella of a participant to reduce skin movement. That location was determined at the intersection of midpoints of the transverse and sagittal planes. The left leg was used due to laboratory room width. Knee flexion angle was constant for each WBV bout and checked manually with a goniometer. The accelerometer about the knee was checked for vertical alignment before and during each WBV bout.

Left leg length was measured with the ‘total true shortening’ method (McRae, 1999). A tape was placed from the left anterior superior iliac spine to the left medial malleolus. The participant lay in the supine position. The data were recorded because it was thought leg length may impact on transmission of WBV given that wobbling mass and rigid bodies affect transmission (Yue & Mester, 2002).

Knee flexion angle was filmed with a digital camcorder (NV-GS11, Matsushita Electric Industrial Co., Osaka JPN) and digitised with Siliconcoach Pro 7 (Siliconcoach, San Francisco, USA). Small reflective markers (1 cm diameter) were adhered on the skin to the right; greater trochanter, lateral condyle, and lateral malleolus. Data were filmed for each knee flexion angle independent of stance width and vibration platform frequency.
**Statistical Procedures**

Although this pilot test included several independent variables, only knee angle was used for inferential analysis. Descriptive statistics were calculated to quantify sample statistics, root mean square acceleration, knee angle, and WBV transmission. Normality was tested by: (1) homogeneity of variance should not be violated, and (2) the data should not exceed ± 2 for skewness and kurtosis (Bluman, 1997; Macellari, Giacomozzi & Saggini, 1999; Vincent, 1999; Portney & Watkins, 2000; Coakes, Steed & Dzidic, 2006). Independent sample *t*-tests were computed for gender effects. A one-way ANOVA was computed to describe effects of knee angle on transmission at 25Hz with 2.0 mm peak-to-peak displacement. Data were imported to SPSS 17.0 for Windows (SPSS Inc., Chicago, USA). Significance was accepted at *p* ≤ 0.05. Some root mean square acceleration data were lost due to technical difficulties. For vibration platform frequency 25 Hz and peak-to-peak displacement 2.0 mm, data were analysed for no less than 9 participants and no more than 11 participants respectively.

**Results**

Descriptive and inferential statistics are shown to describe transmission of WBV about the knee of healthy young adults. Participant anthropometric and selected descriptive statistics are shown in table 3.2. Descriptive and inferential statistics of transmission are shown in table 3.3. Descriptive statistics of knee angle are shown in table 3.4.

The males and females in this pilot test were different for stature and mass, but not leg length. Males were 8.0 cm taller and 12.6 kg heavier than females (*p* < 0.05). Left leg length (where the accelerometer was placed) was not significantly different (mean difference = 4.5 cm), so data were pooled for transmission inferential analysis.
Table 3.2

Anthropometric and descriptive statistics of the healthy young adults

<table>
<thead>
<tr>
<th>Sex</th>
<th>n</th>
<th>Stature (m)</th>
<th>Δ mean</th>
<th>Mass (kg)</th>
<th>Δ mean</th>
<th>Left leg length (cm)</th>
<th>Δ mean</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>mean</td>
<td>SD</td>
<td></td>
<td>mean</td>
<td>SD</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>7</td>
<td>1.7</td>
<td>0.1</td>
<td>0.1*</td>
<td>63.8</td>
<td>7.2</td>
<td>12.6*</td>
</tr>
<tr>
<td></td>
<td>8</td>
<td>1.8</td>
<td>0.1</td>
<td>76.4</td>
<td>9.7</td>
<td>93.2</td>
<td>3.9</td>
</tr>
</tbody>
</table>

Note: * significant difference $p \leq 0.05$

Root mean square acceleration was reported as transmission of WBV about the knee to best support the description of the effect of knee flexion angle (table 3.3). There was no effect of the three knee flexion angles on root mean square acceleration of WBV about the knee ($F(2,28) = 1.45, p = 0.25$). Transmission, the ratio of root mean square acceleration about the knee to root mean square acceleration at the surface of the vibration platform, was greater when the knee was flexed to 60°. Attenuation of WBV about the knee was least when the knee was flexed to 40°.

Table 3.3

Transmission of WBV about the knee for various vibration platform frequencies, stance widths, and stance postures

<table>
<thead>
<tr>
<th>Knee angle</th>
<th>RMS Acceleration Knee (m/sec(^2))</th>
<th>95% Confidence Interval for Mean</th>
<th>RMS Acceleration Vibratation Platform (m/sec(^2))</th>
<th>Transmission RMS(_K)-RMS(_P) (^{\text{SD}})</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
<td>Lower Bound</td>
<td>Upper Bound</td>
</tr>
<tr>
<td>20°</td>
<td>20.0</td>
<td>2.2</td>
<td>12.6</td>
<td>27.4</td>
</tr>
<tr>
<td>40°</td>
<td>23.0</td>
<td>6.4</td>
<td>16.9</td>
<td>28.9</td>
</tr>
<tr>
<td>60°</td>
<td>16.1</td>
<td>5.4</td>
<td>11.9</td>
<td>20.2</td>
</tr>
</tbody>
</table>

Note: RMS: root mean square. RMS\(_K\): root mean square knee. RMS\(_P\): root means square vibration platform.
Table 3.4 shows variability of pre-determined knee flexion angle during each WBV bout. Although the 20º knee flexion stance posture was most accurately maintained, variability across all stance postures was almost identical.

Table 3.4

Pre-determined stance postures and knee flexion angles by ‘Siliconcoach’

<table>
<thead>
<tr>
<th>Pre-determined knee flexion angle</th>
<th>Mean</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>20º</td>
<td>22.1º</td>
<td>4.2º</td>
</tr>
<tr>
<td>40º</td>
<td>36.6º</td>
<td>4.1º</td>
</tr>
<tr>
<td>60º</td>
<td>54.7º</td>
<td>4.2º</td>
</tr>
</tbody>
</table>

Discussion

In this pilot test, transmission of WBV was quantified by the ratio of root mean square acceleration of the vibration platform and root mean square acceleration about the knee. Three major findings emerged: (1) knee flexion angle influences transmission, though the differences among each of the knee flexion angles were not significant (2) transmission can be quantified with a tri-axial accelerometer, and (3) speculatively, the methods of accelerometer based transmission assessment may be used in a community setting to quantify transmission of WBV about the knee in other populations such as people living with COPD if the laboratory conditions can be adopted and replicated.
A shortage of knowledge exists about WBV transmission with side alternating vibration platforms. Although not specific to the vibration platform descriptors of this pilot test, for synchronous WBV at 20 Hz, independent of stance posture, WBV was not attenuated about the knee (Harazin & Grzesik, 1998; Matsumoto & Griffin, 1998). The results of this pilot test showed attenuation of WBV about the knee when transmission was quantified at 25 Hz. As such, attenuation of WBV, due to wobbling mass and rigid bodies were confirmed by the results of this pilot test.

Although males were taller and heavier than females, limb length was not significantly different. As such, wobbling mass of recreationally active, healthy adults may affect transmission of WBV. The same case may be made of rigid bodies (i.e., femur and tibia length) although further investigation is required. Furthermore, the effect of wobbling mass and rigid bodies on transmission force attenuation about the hip, spine, and head is relatively unknown with side alternating vibration platforms and should be further investigated.

About the head, synchronous WBV created more acceleration than side alternating WBV for healthy adults (Abercromby et al., 2007b). The remainder of the WBV was transmitted about other locations of the body (i.e., hip, spine and head). Given the overall aim of this current research was to improve or maintain exercise tolerance and functional performance by increasing muscular strength and muscular power of the lower limbs of people with COPD, transmission of WBV about the head was not considered. The quantification of transmission of WBV at other locations superior to the knee was beyond the scope of this research, although such information could enhance understanding of WBV and transmission.
Quantification of transmission was assessed in this pilot test, despite the known limitation of skin-mounted accelerometers. The accelerometers as such, were sufficiently sensitive to detect attenuation of WBV. Although in the current research noise of skin mounted accelerometer data was not accounted for, the error appeared systematic rather than random (i.e., figure 3.6).

Variability of knee angle, vertical acceleration of the vibration platform, and individual difference in the ratio of wobbling mass and rigid bodies may have affected statistical results. Such variability may imply that stance width and knee flexion angle needs to be considered for each individual when quantifying efficacy of WBV training interventions. The results of this pilot test show greater transmission at 60º knee flexion angle. The sustainability of that posture, however, for people with muscular deconditioning (i.e., people with COPD) could be questioned even though several authors have reported increased EMG activity of vastus lateralis during WBV with an isometric squat of 55º knee flexion compared with more erect stance postures (Cardinale & Lim, 2003; Roelants et al., 2006). Given there was no difference for knee flexion angle and transmission in the current pilot test ($p = 0.25$), people with muscular deconditioning could stand with a knee flexion angle between 20 and 60º, thus, at a preferred stance posture to aid compliance to a long-term WBV intervention.
Summary

Transmission of side alternating WBV was quantified in this pilot test. The results showed for healthy young adults, knee flexion angle did not alter transmission of WBV about the knee ($p = 0.25$). Greater knee flexion angles are likely to be less sustainable for people with poor muscular conditioning and should be carefully considered when undertaking a WBV intervention. The accelerometer used in this study allowed quantification of WBV transmission and could be used in a community setting to allow similar quantification if the laboratory conditions could be reproduced.

Limitations to the Planned Research

A pilot test to quantify transmission of WBV with skin-mounted accelerometers about the knee of people with COPD in a community setting was undertaken with approval from the Australian Catholic University Human Research Ethics Committee (Appendix E). Participants were recruited through community based COPD support groups. The design of the current pilot test was to be replicated, in which the independent variables were: (1) vibration platform frequency (20, 25 and 30 Hz), (2) stance width (10 and 20 cm), and (3) knee flexion angles (20, 40 and 60º). The aim was to identify the combination of variables that created the largest attenuation of vibration about the knee.

The pilot test was abandoned because the specific laboratory procedures could not be replicated in the 11 homes of the participants. Specifically, several unexpected and uncontrollable extraneous variables led to the abandonment: (1) mounting the accelerometer to the skin of the participants to reduce noise caused subcutaneous bleeding at the knee when the adhering tape was removed and added to the relative inconvenience to the participants of
disruption of the normal arrangements of the home (i.e., movement of furniture), (2) limited space in the home of each participant to establish an adequate field of view to capture knee angle with the camcorder, (3) a lack of funding to transport participants to the laboratory at Australian Catholic University and general inconvenience of such travel to the participant, and (4) only the student researcher was given ethical permission to enter the house of each participant to conduct the pilot test in the community setting (i.e., knee angle could not be manually checked during a WBV bout) resulting in a largely expanded data collection time period and subsequent burden to the participant.
3.4 Pilot Test 3: Reliability of Selected Dependent Variables of Major Study 1 and Major Study 2

Overview

This pilot test was completed to establish inter-day reliability of dependent variables of exercise tolerance and functional performance in a community setting. The dependent variables of exercise tolerance collected at rest were: (1) rating of perceived dyspnoea (Borg CR-10 visual analogue scale), (2) heart rate, and (3) saturation of haemoglobin. The dependent variables of functional performance were: (1) the 5-chair test, (2) the TUG test, (3) stride length, (4) stride time, and (5) stride velocity. Data were collected across a test-retest-retest design to allow a comprehensive assessment of reliability (Weir, 2005) interspersed with at least 48 hours.

Methods

Participants

Participants were recruited from the Department of Respiratory and Sleep Medicine, Monash Medical Centre. The Department patient database was accessed by the Clinical Trials Manager. The Clinical Trials Coordinator sent an information letter to potential participants whom were then invited to record their interest in the pilot test by contacting the student researcher (Appendix F). Medical consent was complicit within the approval of this research by the Department Head (Professor Philip Bardin). The Australian Catholic University Human Research Ethics Committee (Appendix G) and the Southern Health Human Research Ethics Committee A (Monash Medical Centre) (Appendix H) approved the procedures. A convenient sample of 16 community dwelling adult males and females with COPD provided
voluntary informed consent to participate (mean ± SD age = 72 ± 7 years, stature = 1.7 ± 0.1 metres, mass = 85.7 ± 20.4 kilograms). Data were collected in the home of each participant.

Selection Criteria, Selection Protocol and Instruments

Suitability for inclusion to the project was twofold: (1) the potential participant was affected with COPD, and, (2) the potential participant was community dwelling. To satisfy the first criterion spirometry testing was conducted to confirm COPD and determine disease severity (table 3.5).

Table 3.5

Spirometry classification of COPD severity (GOLD, 2007)

<table>
<thead>
<tr>
<th>Stage</th>
<th>FEV₁/FVC Spirometry classification</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage I: Mild</td>
<td>FEV₁/FVC &lt; 0.70</td>
</tr>
<tr>
<td></td>
<td>FEV₁ ≥ 80% predicted</td>
</tr>
<tr>
<td>Stage II: Moderate</td>
<td>FEV₁/FVC &lt; 0.70</td>
</tr>
<tr>
<td></td>
<td>FEV₁ &lt; 80% predicted</td>
</tr>
<tr>
<td>Stage III: Severe</td>
<td>FEV₁/FVC &lt; 0.70</td>
</tr>
<tr>
<td></td>
<td>FEV₁ &lt; 50% predicted</td>
</tr>
<tr>
<td>Stage IV: Very severe</td>
<td>FEV₁/FVC &lt; 0.70</td>
</tr>
<tr>
<td></td>
<td>FEV₁ &lt; 30% predicted</td>
</tr>
</tbody>
</table>

Note: Predicted FEV₁ is calculated based on height, age, gender, and ethnicity.

A SpiroPro PC connected spirometer (MicroMedical Ltd., Kent, UK) was used to conduct at least three FVC (FEV₁) lung function tests. The SpiroPro is concurrently validated with other laboratory and office based spirometers to detect COPD (Liistro, Vanwelde, Vincken, Vandevoorde, Verleden & Buffels, 2006). The protocol for an acceptable spirometry test is well described by Miller et al. (2005). A summation of the acceptable protocol is shown in
table 3.6. Spida 5 PC Software (Version 2.7; MicroMedical Ltd., Kent, UK) was used to automatically determine predicted FEV$_1$ using the third National Health and Nutrition Examination Survey (NHANES III) (Hankinson, Odencrantz & Fedan, 1999).

To be considered community dwelling, a potential participant had to be living in a fully-independent residence, and needed to have the capacity to complete ADLs such as non-assisted walking (Furness & Maschette, 2009). Selected tests of cognition and visual acuity were then used assist with determination of the status of ‘community dwelling’ (Appendix I). The tests were: (1) the Mini-Mental State Examination (MMSE), (2) the Romberg test, (3) the Snellen Eye Chart (six metres), and (4) the Melbourne Edge test. The tests were always conducted in the same order for every participant and data recorded to a results sheet. Participants unable to ‘pass’ all tests were not invited to participate further in the project. The selection criteria had been previously used to screen for healthy community dwelling older adults prior to inclusion to a WBV intervention (Furness & Maschette, 2009).
Table 3.6

Procedure for recording forced vital capacity

<table>
<thead>
<tr>
<th>Description</th>
<th>Descriptions/Instructions</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Check the spirometer calibration</td>
<td>3L syringe</td>
</tr>
<tr>
<td>2. Explain the test</td>
<td></td>
</tr>
<tr>
<td>3. Prepare the participant</td>
<td>Measure mass and stature</td>
</tr>
<tr>
<td>4. Wash hands</td>
<td></td>
</tr>
<tr>
<td>5. Instruct and demonstrate the test to the participant</td>
<td>Correct posture with head slightly elevated. Inhale rapidly and completely. Exhale with maximal force.</td>
</tr>
<tr>
<td>6. Perform manoeuvre</td>
<td>Have participant perform the correct posture. Attach nose clip, place mouthpiece in mouth and close lips around mouthpiece. Inhale completely and rapidly with a pause of &lt;1 s at total lung capacity. Exhale maximally until no more air can be expired while maintaining an upright posture. Repeat instructions as necessary, coaching vigorously. Repeat for a minimum of three manoeuvres.</td>
</tr>
</tbody>
</table>

Note: Adopted from Miller et al. (2005). The correct posture for a participant was to ensure each was person sitting erect in an armless chair. Following the joint guidelines of the American Thoracic Society and the European Respiratory Society, FEV1/FEV data were recorded prior to, and 15 minutes after Salbutamol (0.3 mg in spray form) inhalation from a spacer (Celli et al., 2004). The prediction equations of the third National Health and Nutrition Examination Survey (NHANES III) were used to classify COPD severity.

Tests of Cognition, Motor Function and Visual Acuity

The Mini-Mental State Examination

The MMSE was used to assess mental status. The reliability of the test was previously reported as ‘good’ to detect dementia (ICC ≥ 0.80) (Magni, Binetti, Bianchetti, Rozzini & Trabucchi, 1996). The test covers five areas of cognitive function: orientation, registration,
attention and calculation, recall and language, and is commonly used as a screening tool among community dwelling older adult groups (Kurlowicz & Wallace, 1999). A score of 23 out of 30 is considered a pass (Dite, & Temple, 2002; Hill & Schwarz, 2004; Melzer, Benjuya & Kaplanski, 2004). The MMSE was conducted with the ‘paper and pen’ method described in previous investigations (Kurlowicz & Wallace, 1999; Dite, & Temple, 2002; Hill & Schwarz, 2004; Melzer, Benjuya & Kaplanski, 2004).

The Romberg Test

The Romberg test was used to identify distorted proprioception (Khasnis & Gokula, 2003). The test is a good predictor of normal postural sway (Black, Wall, Rockette & Kitch, 1982). Each participant stood with their feet together and eyes closed for 30 seconds. The student researcher was close at hand in case the participant began to sway or fall, at which time the test was ended. A successful test was at least 30 seconds in duration, as recorded by a stopwatch.

The Snellen Eye Chart

The Snellen Eye Chart was used to establish how well each eye saw printed letters (Gerdhem, Ringsberg, Åkesson & Obrant, 2005). The chart provides a useful test for visual acuity in a community setting to assist with participant screening (Schwiegerling, 2004), although for ophthalmic diagnosis the test is limited (McGraw, Winn & Whitaker, 1995). Each participant was asked to read letters of the chart at six metres distance. The participant sat in a chair and read, one eye at a time, the chart as accurately as possible (prescription glasses optional). To pass the test the participant had to score at least 0.2 on the chart for the set distance. The chart was placed on a chair (46 cm high) six metres from the participant. The participant read the
chart from an identical chair. A tape measure was fixed to a point on the floor (directly below the chart) and drawn six metres from the chart. The centre of the participant’s seat was placed at that point.

The Melbourne Edge Test

The Melbourne Edge test was used to examine the contrast vision sensitivity of each participant (Hill & Schwarz, 2004) because poor performance of the test is linked to increased risk of a fall (Lord & Dayhew, 2001). The reliability of the test was previously reported as ‘good’ to detect contrast vision sensitivity (ICC = 0.880) (Haymers, & Chen, 2004). The participant was asked to use both eyes (prescription glasses optional) to detect the varying contrast among two colours. A score of at least 11 on the test represents a pass. The Melbourne Edge test was placed on a table directly in front of the participant. The participant sat on the same chair used for the Snellen Eye Chart assessment.

Data Collection

Data of the dependent variables of major study 1 and major study 2: rating of perceived dyspnoea, heart rate, saturation of haemoglobin, 5-chair test, TUG test, and kinematic variables of gait; stride length, stride time, and stride velocity were collected over a test-retest-retest procedure. Data were collected interspersed with at least 48 hours to allow inter-day reliability analyses.
Exercise Tolerance: The Borg CR-10 Visual Analogue Scale

The Borg CR-10 visual analogue scale (Appendix J) was used to quantify the rating of perceived level of breathing limitation so that dyspnoea could be anchored. Reliability of the visual analogue scale (VAS) for dyspnoea was previously established (Mahler, 1992; Mahler, Mejia-Alfaro, Ward & Baird, 2001; Borg, Borg, Larsson, Letzter & Sundblad, 2010). Population specific reliability of the VAS was considered ‘acceptable’ if the ICC was ≥ 0.700 (George & Mallery, 2003). Resting rating of perceived dyspnoea was collected after the participant had been sitting quietly in a chair for five minutes.

Exercise Tolerance: Heart Rate and Saturation of Haemoglobin

Heart rate (HR) and saturation of haemoglobin (SpO₂) were both quantified with the CARESCOPER™ V100 Vital Sign Monitor (GE Health Care, Milwaukee, USA). At rest, data were collected after the participant had been sitting quietly in a chair for five minutes. Population specific reliability of the device was ‘acceptable’ if the ICC was ≥ 0.700 (George & Mallery, 2003). An increase of resting heart rate is synonymous with physical activity (ACSM, 1998b), while a reduction of SpO₂ has been linked to poor diffusing capacity partly due to emphysematous change consistent with a diagnosis of COPD (West, 2008).

Functional Performance: The 5-Chair Stands Test

The 5-chair stands test (5-chair test) was most commonly used in community settings to quantify muscular power (Moreland, Richardson, Goldsmith & Clase, 2004) because chair rising power was fundamental to functional performance of older adults (Runge, Rittweger, Russo, Schiessl & Felsenberg, 2004). The reliability of the tests was ‘excellent’ for functional performance of community dwelling older adults (ICC > 0.900) (Furness &
Maschette, 2009). Each participant was asked to sit in a standard height (46 cm, including back rest) chair with their arms folded across their chest. The participant was then asked to stand and sit five times (Brill, Cornman, Davis, Lane, Mustafa, Sanderson & Macera, 1999; Runge, Rehfeld & Resnicek, 2000; Visser, Pluijm, Stel, Bosscher & Deeg, 2002; Runge et al., 2004).

The test began on the verbal instruction to “go” and ended when the participant sat with their back against the back rest of the chair. Participants were told their back must make contact with the back rest for all five chair stands. Time taken was recorded with a stopwatch. Participants were told the procedure to complete the test and were given one familiarisation test at the first of three test occasions. The timed test occurred 5 minutes after the familiarisation test to ensure recovery. Participants were asked to complete the test as they felt comfortable. One test was completed and used for reliability analyses at each test occasion separated by at least 48 hours.

**Functional Performance: The Timed-Up-and-Go Test**

The timed-up-and-go test (TUG test) was a modified version of the get-up-and-go test (GUG). The TUG test was reliable (inter-day ICC ≥ 0.970) and a valid quantifier of gait speed ($r^2 = 0.74$), and functional motor performance ability of older adults (Podsiadlo & Richardson, 1991). The TUG test involves a well recognised series of events used in active daily living (Isles, Low Choy, Steer & Nitz, 2004). Presented by Podsiadlo and Richardson (1991) the TUG test eliminated observer variance systemic to the GUG test by examining the time taken to complete the test rather than a performance rating scale (Podsiadlo & Richardson, 1991).
For the TUG test, the participant was asked to sit in a standard height (46 cm) arm (63 cm) chair. The participant was then asked to stand, walk three metres to a marker on the floor, turn, return, and sit on the chair (Brill et al., 1999; Dite & Temple, 2002; Cho, Scarpace & Alexander, 2004). The test began on the verbal instruction to “go” and ended when the participant sat with their back against the back rest of the chair. Participants were told their back must make contact with the back rest in order to complete the test.

Time taken was recorded with a stopwatch. Participants were told the procedure to complete the test and were given one familiarisation test at the first of three test occasions. The timed test occurred 5 minutes after the familiarisation test to ensure recovery. Participants were asked to complete the test as they felt comfortable. Three tests were completed on each of the three test occasions with 60 seconds passive rest (sitting in the chair) interspersed among tests. All three tests were used in the data analyses.

**Kinematic Variables of Gait**

**The GAITRite® Electronic Walkway**

The GAITRite mat was used for the capacity to collect a large number of kinematic datum per walking trial compared with the 5-chair test and the TUG test. The GAITRite mat comprised of seven sensor pads embedded in a 14 foot portable roll up mat. The data sensitive area of the mat (61 cm wide, by 360 cm long, by 0.6 cm high) allowed quantification of spatial and temporal kinematic variables of gait. Each centimetre of walkway was embedded with 38 sensors sampling at 80 Hz. Data were transferred to a notebook computer with GAITRite software (Version 3.8). The concurrent validity and reliability of the walkway had been previously established in the range of ‘good’ to ‘excellent’ (ICC > 0.800) (Selby-Silverstein...
& Besser, 1999; Cutlip, Mancinelli, Huber & DiPasquale, 2000; McDonough, Batavia, Chen, Kwon & Ziai, 2001; Nelson Zwick, Brody, Doran, Pulver, Rooz et al., 2002). For the participants of this pilot test, data were collected for stride length, stride time, and stride velocity.

Participants were instructed to walk at a self-selected comfortable speed along a straight, flat, and even walkway area containing the mat. Body length was measured from the base of the foot (unshod) to the crown of the head while standing in the anatomical position. During data collection, participants walked (shod) over the mat five times in a discontinuous process with an approach and departure distance equal to two body lengths (figure 3.7). The procedure was reliable for step length, step time, and velocity among older women (ICC range 0.860 to 0.940; mean age = 68 years) (Paterson, Hill, Lythgo & Maschette, 2008).

For reliability and subsequent use of this pilot test, stride length is quantified as the line of progression between two consecutive footprints of the same foot. In figure 3.8, stride length was represented in metres by the length of the line of progression between points A and G. Stride time was the time taken in seconds between the first contacts of two consecutive footballs of the same foot (i.e., the time among points A and G). Stride velocity was the ratio of stride length to stride time and quantified as metres per second (m.sec⁻¹).
Stride length was the length in metres of the line of progression from points A and G.

Figure 3.7. Walkway length showing data sensitive area and body length protocol.

Figure 3.8. Spatial kinematic parameters of gait from the GAITRite® Electronic Walkway. Source: CIR Systems Inc., GAITRite® Electronic Walkway Technical Reference (WI-02-15). Stride length was the length in metres of the line of progression from points A and G.
Data Analyses

Descriptive statistics were computed to describe the participants and the severity of their COPD. Reliability intra-class correlation coefficients; ICC (3, k) values were calculated to describe inter-day reliability of the dependent variables of exercise tolerance and functional performance. The model suited a repeated measures ANOVA, where k denoted mean ratings, and ‘ratings’ were the test occasions (Portney & Watkins, 2000). The repeated measures ANOVA was computed with the SPSS 19.0 for Windows spread sheet (SPSS Inc., Chicago, USA). Normality was tested by: (1) Mauchly’s sphericity test; and (2) the data should not exceed ± 2 for skewness and kurtosis (Bluman, 1997; Macellari, Giacomozzi & Saggini, 1999; Vincent, 1999; Portney & Watkins, 2000; Coakes, Steed & Dzidic, 2006).

Each ICC (3, k) was calculated with equation 1 (Vincent, 1999; Portney & Watkins, 2000). For equation 1, ‘BMS’ was the between-subjects mean square from the repeated measures ANOVA and ‘EMS’ was the error mean square from the repeated measures ANOVA. Intraclass correlation coefficients were computed between values of 0.000 and 1.000. An ICC value of 0.700 was ‘acceptable’, 0.800 was ‘good’, and, ≥ 0.900 was ‘excellent’ (George & Mallery, 2003).

\[
\text{ICC } (3, k) = \frac{\text{BMS} - \text{EMS}}{\text{BMS}}
\]

equation 1

High ICC values can overestimate reliability because the results do not show within subject differences across test occasion (Menz, Latt, Tidenamm, Kwon & Lord, 2004). Therefore, as absolute measures of reliability, standard error of measurement (SEM) and coefficient of
variation (CV) were calculated to show variability within test occasions (Portney & Watkins, 2000). The SEM was calculated with equation 2 (Portney & Watkins, 2000) where ‘SD’ was the standard deviation of all data and calculated with equation 3 (Weir, 2005), and ‘SS_Total’ was the sum of squares total computed by the repeated measures ANOVA and ‘n’ was the total number of datum.

\[
SEM = SD \times \frac{1 - ICC}{\sqrt{n}}
\]  
\[equation 2\]

\[
SD = \frac{SS_{Total}}{n-1}
\]  
\[equation 3\]

A 95% confidence interval (CI) of the SEM was calculated to describe the range of error for each dependent variable (Portney & Watkins, 2000) (equation 4). The 95% CI was calculated for the grand mean (x) of the three test occasions.

\[
95\% \ CI = x \pm 1.96 \times \left( \frac{SEM}{\sqrt{n}} \right)
\]  
\[equation 4\]

The CV was calculated with equation 5 (Portney & Watkins, 2000). For equation 5, ‘x’ was the grand mean and SEM was calculated with equation 2.

\[
CV = \frac{SEM}{x} \times 100\%
\]  
\[equation 5\]
Results

Table 3.7 shows the descriptive statistics of the participants in this reliability pilot. Mean age of the four females and 12 males was 72 years. Participant age ranged between 56 and 84 years. Mean body mass index (BMI) was 29.3 kg.m\(^2\), which was considered ‘normal’ (WHO, 2012).

Table 3.7

Sample descriptors of participants with COPD

<table>
<thead>
<tr>
<th>Descriptor</th>
<th>Mean</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>72</td>
<td>7</td>
</tr>
<tr>
<td>Stature (m)</td>
<td>1.7</td>
<td>0.1</td>
</tr>
<tr>
<td>Mass (kg)</td>
<td>85.7</td>
<td>20.4</td>
</tr>
<tr>
<td>BMI (kg.m(^{-2}))</td>
<td>29.3</td>
<td>6.1</td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>139</td>
<td>13</td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td>75</td>
<td>10</td>
</tr>
<tr>
<td>Resting HR (beats.min(^{-1}))</td>
<td>82</td>
<td>10</td>
</tr>
<tr>
<td>Predicted maximum HR (beats.min(^{-1}))</td>
<td>148</td>
<td>7</td>
</tr>
</tbody>
</table>

Note: N = 16 (4 females, 12 males). SBP: systolic blood pressure. DBP: diastolic blood pressure. HR: heart rate. Resting HR: After sitting quietly in a chair for five minutes. Predicted maximum HR: 220 beats.min\(^{-1}\) – age.

Spirometry testing results are shown in table 3.8. According to GOLD (2007), 15 participants were affected by ‘moderate’ Stage II COPD. Mean FEV\(_1\) was less than 80% predicted. Mean FER was less than 70%. One participant was affected with ‘severe’ Stage III COPD (FEV\(_1\) % predicted = 30%).
Table 3.8

Spirometry descriptors of the participants with COPD

<table>
<thead>
<tr>
<th>Descriptor</th>
<th>Mean</th>
<th>Pre SD</th>
<th>95% CI</th>
<th>Mean</th>
<th>Post SD</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>FEV₁ (L BTPS)</td>
<td>1.5</td>
<td>0.5</td>
<td>± 1.0</td>
<td>1.7</td>
<td>0.7</td>
<td>± 1.4</td>
</tr>
<tr>
<td>FEV₁ % predicted</td>
<td>53.0</td>
<td>16.7</td>
<td>± 32.7</td>
<td>58.5</td>
<td>19.0</td>
<td>± 37.2</td>
</tr>
<tr>
<td>FVC (L BTPS)</td>
<td>2.8</td>
<td>0.7</td>
<td>± 1.3</td>
<td>3.0</td>
<td>0.8</td>
<td>± 1.6</td>
</tr>
<tr>
<td>FVC % predicted</td>
<td>78.5</td>
<td>13.3</td>
<td>± 26.1</td>
<td>83.2</td>
<td>15.0</td>
<td>± 29.4</td>
</tr>
<tr>
<td>FER %</td>
<td>52.0</td>
<td>13.8</td>
<td>± 27.0</td>
<td>52.3</td>
<td>10.8</td>
<td>± 21.2</td>
</tr>
<tr>
<td>PEF (L sec⁻¹)</td>
<td>3.8</td>
<td>1.4</td>
<td>± 2.7</td>
<td>4.2</td>
<td>0.8</td>
<td>± 1.6</td>
</tr>
</tbody>
</table>

Note: Pre: before 0.3 mg Salbutamol inhalation. Post: 15 minutes after 0.3 mg Salbutamol inhalation. One participant was classified as severe Stage III COPD. FEV₁: forced expired volume of oxygen in the first second. FVC: forced vital capacity of the lungs. FER: forced expiratory ratio (FEV₁/FVC⁻¹). PEF: peak expiratory flow. L BTPS: Litres Body Temperature Pressure Saturated. 95% CI: confidence interval [x ± 1.96(SD)]

Table 3.9 shows the descriptive statistics and the result of the ICC and CV calculations for the dependent variables of exercise tolerance and functional performance. Data were collected across a test-retest-retest design interspersed with at least 48 hours. Reliability of the dependent variables was at least ‘acceptable’ (ICC ≥ 0.700). The maximum CV for exercise tolerance variables was 28.5% (i.e., Borg CR-10). The maximum CV for functional performance dependent variables was 4.3% (i.e., stride velocity).
### Table 3.9

Inter-class correlation reliability results of all dependent variables across three test occasions for people with COPD

<table>
<thead>
<tr>
<th>Dependent Variable</th>
<th>Test Occasion</th>
<th>n</th>
<th>Mean</th>
<th>SD</th>
<th>Skewness</th>
<th>Kurtosis</th>
<th>CV (%)</th>
<th>SEM (± 95% CI)</th>
<th>ICC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Borg CR-10 (VAS)</td>
<td>1</td>
<td>16</td>
<td>2</td>
<td>0</td>
<td>1.69</td>
<td>1.48</td>
<td>28.5</td>
<td>2 (± 1.12)</td>
<td>0.706</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>16</td>
<td>2</td>
<td>0</td>
<td>0.97</td>
<td>0.89</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>16</td>
<td>2</td>
<td>0</td>
<td>0.67</td>
<td>1.18</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heart Rate (beats.min⁻¹)</td>
<td>1</td>
<td>16</td>
<td>88</td>
<td>4</td>
<td>-0.12</td>
<td>-0.12</td>
<td>5.39</td>
<td>89 (± 9.40)</td>
<td>0.789</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>16</td>
<td>88</td>
<td>4</td>
<td>-0.27</td>
<td>0.25</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>16</td>
<td>90</td>
<td>2</td>
<td>0.24</td>
<td>1.43</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Saturation of haemoglobin (%)</td>
<td>1</td>
<td>16</td>
<td>96</td>
<td>1</td>
<td>-0.66</td>
<td>-0.98</td>
<td>0.70</td>
<td>96 (± 1.31)</td>
<td>0.700</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>16</td>
<td>96</td>
<td>0</td>
<td>-0.13</td>
<td>-0.88</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>16</td>
<td>95</td>
<td>1</td>
<td>-0.02</td>
<td>-0.07</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note: Continued on next page.
Table 3.9 (continued)

<table>
<thead>
<tr>
<th>Dependent Variable</th>
<th>Test Occasion</th>
<th>n</th>
<th>Mean</th>
<th>SD</th>
<th>Skewness</th>
<th>Kurtosis</th>
<th>CV (%)</th>
<th>SEM (± 95% CI)</th>
<th>ICC</th>
</tr>
</thead>
<tbody>
<tr>
<td>5-chair stands test (sec)</td>
<td>1</td>
<td>16</td>
<td>19.3</td>
<td>3.7</td>
<td>0.01</td>
<td>-1.56</td>
<td>1.27</td>
<td>3.67 (± 7.19)</td>
<td>0.881</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>16</td>
<td>19.3</td>
<td>4.1</td>
<td>0.09</td>
<td>-1.14</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>16</td>
<td>18.8</td>
<td>3.2</td>
<td>0.75</td>
<td>-0.34</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TUG test (sec)</td>
<td>1</td>
<td>46</td>
<td>11.8</td>
<td>2.1</td>
<td>0.18</td>
<td>-0.24</td>
<td>0.37</td>
<td>1.97 (± 3.86)</td>
<td>0.964</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>46</td>
<td>11.5</td>
<td>1.9</td>
<td>-0.13</td>
<td>-0.52</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>46</td>
<td>11.3</td>
<td>1.8</td>
<td>-0.05</td>
<td>-0.20</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stride time (sec)</td>
<td>1</td>
<td>182</td>
<td>1.14</td>
<td>0.09</td>
<td>0.61</td>
<td>-0.39</td>
<td>0.03</td>
<td>0.10 (± 0.20)</td>
<td>0.928</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>182</td>
<td>1.13</td>
<td>0.10</td>
<td>0.86</td>
<td>0.18</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>182</td>
<td>1.13</td>
<td>0.11</td>
<td>0.92</td>
<td>0.30</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stride length (m)</td>
<td>1</td>
<td>182</td>
<td>1.13</td>
<td>0.16</td>
<td>0.54</td>
<td>-0.10</td>
<td>3.28</td>
<td>0.15 (± 0.29)</td>
<td>0.952</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>182</td>
<td>1.14</td>
<td>0.14</td>
<td>0.25</td>
<td>-1.10</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>182</td>
<td>1.14</td>
<td>0.15</td>
<td>0.23</td>
<td>-0.93</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stride velocity (m.sec(^{-1}))</td>
<td>1</td>
<td>182</td>
<td>0.99</td>
<td>0.15</td>
<td>0.35</td>
<td>-0.66</td>
<td>4.30</td>
<td>0.14 (± 0.27)</td>
<td>0.900</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>182</td>
<td>1.01</td>
<td>0.12</td>
<td>0.84</td>
<td>0.18</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>182</td>
<td>1.02</td>
<td>0.14</td>
<td>0.29</td>
<td>-0.42</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note: The 5-chair test: 16 participants completed one trial on each test occasion, thus N = 48. The TUG test: 15 participants completed three trials on each of three test occasions and 1 participant completed one trial on each test occasion, thus N = 138. Stride time: at least 10 datum were recorded for each individual among the five trials. Stride length: at least 10 datum were recorded for each individual among the five trials. Stride velocity: At least 10 datum were recorded for each individual among the five trials.
Discussion and Summary

This pilot test established the inter-day reliability of dependent variables fundamental to major study 1 and major study 2. For the dependent variables of exercise tolerance (Borg CR-10, heart rate, and saturation of haemoglobin), reliability was at least ‘acceptable’ (ICC ≥ 0.700). The range of ICC’s was good (0.881) to excellent (0.964) for functional performance dependent variables (5-char test, TUG test, stride time, stride length, and stride velocity).

The Borg CR-10 VAS has been limited due to intra-individual variability across test occasions (Shen & Parsons, 1997). The CV results of this pilot test (CV = 28.5%) similarly show a large intra-individual variability of the Borg CR-10 VAS. Bearing in mind however, the acceptable reliability of the VAS (ICC = 0.706), the CV was considered to be satisfactory.

For healthy young adults CV of resting HR was between 4 and 17% (Haddad, Laursen, Chollet, Ahmaidi & Buchheit, 2011). In this pilot test CV of resting HR was similarly low at 5.39% although quantified with a different device. The similarity of HR and the acceptable reliability (ICC = 0.700) and CV (0.70%) of SpO2 in the current pilot test justifies the use of the CARESCOPE™ V100 Vital Sign Monitor to quantify the selected dependent variables of exercise tolerance of this research.

The reliability data for the functional performance dependent variables of this research are supported by other studies of reliability of the 5-chair test (Guralnik, Ferrucci, Simonsick, Salive, & Wallace, 1995; Moreland et al., 2004) and the TUG test (Isles et al., 2004). Kinematic gait data are also supported by the ‘good’ to ‘excellent’ ICC’s reported by Paterson et al. (2008) for community dwelling older adult females. Specifically, the two body length
approach and departure distance was reliable for people with COPD for stride time, stride length, and stride velocity.

For older adults, a comfortable self-selected walking pace with a 2 metre approach and departure distance was reliable for velocity and step length quantified with the GAITRite mat (ICC range 0.880 to 0.910) (Menz et al., 2004). In this pilot test, a two body length approach and departure distance elicited similar ICC reliability.

The CV values calculated in this pilot test were similar to Menz et al. (2004). In that study, CV for velocity was 3.5% and 3.3% for step length of healthy older adults (mean age = 81 years). In this pilot test, CV of stride velocity was 4.30%, and 3.28% for stride length of people with COPD. Although no limits of acceptability for CV were apparent in the literature, the CV for dependent variables of functional performance of people with COPD in this pilot test were low and similar to the CV results of other studies considered acceptable. The reliability data of this pilot test support the previous reports that the GAITRite® Electronic Walkway is reliable for quantifying kinematic variables of gait with older adults (Menz et al., 2004; Paterson et al., 2008).
3.5 Sample Size Calculations

Overview

Whole-body vibration has never been used as an intervention in a community setting to improve or maintain exercise tolerance and functional performance of the lower limbs of people with COPD. Therefore, sample size was estimated using data from a study of healthy community dwelling older adults (mean age = 72 years) who completed two sessions of WBV over a six week intervention (Furness & Maschette, 2009). The dependent variables were the 5-chair test and the TUG test. The intensity of WBV was 0.6 g (weeks 1 and 2), 0.8 g (weeks 3 and 4), and 1.3 g (weeks 5 and 6). The WBV sessions consisted of five, one minute WBV bouts interspersed with one minute passive rest. The data collection methods of this research for the 5-chair test and the TUG test were identical. To allow sample size calculation, the primary dependent variables focused on tests of functional performance: the 5-chair test and the TUG test.

Methods

Original Data

The raw data of Furness and Maschette (2009) are shown in table 3.10. Descriptive statistics of that study are shown in table 3.11. The value of kurtosis exceeded the assumption of normality (within ± 2) (Vincent, 1999).
Table 3.10.

Raw data of Furness & Maschette (2009) for healthy community dwelling older adults at baseline and after six weeks of WBV

<table>
<thead>
<tr>
<th>TUG Pre</th>
<th>TUG Post</th>
<th>CHAIR Pre</th>
<th>CHAIR Post</th>
</tr>
</thead>
<tbody>
<tr>
<td>8.39</td>
<td>7.16</td>
<td>10.22</td>
<td>9.97</td>
</tr>
<tr>
<td>6.58</td>
<td>6.25</td>
<td>18.61</td>
<td>11.55</td>
</tr>
<tr>
<td>9.05</td>
<td>7.52</td>
<td>14.64</td>
<td>11.79</td>
</tr>
<tr>
<td>9.28</td>
<td>6.95</td>
<td>20.40</td>
<td>16.48</td>
</tr>
<tr>
<td>14.77</td>
<td>14.50</td>
<td>12.26</td>
<td>11.99</td>
</tr>
<tr>
<td>10.19</td>
<td>9.41</td>
<td>24.61</td>
<td>18.73</td>
</tr>
<tr>
<td>18.60</td>
<td>16.00</td>
<td>21.56</td>
<td>12.93</td>
</tr>
<tr>
<td>13.55</td>
<td>9.80</td>
<td>15.79</td>
<td>12.93</td>
</tr>
<tr>
<td>10.19</td>
<td>8.44</td>
<td>15.10</td>
<td>12.38</td>
</tr>
<tr>
<td>9.61</td>
<td>8.44</td>
<td>18.44</td>
<td>16.40</td>
</tr>
<tr>
<td>10.43</td>
<td>9.28</td>
<td>11.42</td>
<td>10.98</td>
</tr>
<tr>
<td>11.44</td>
<td>9.16</td>
<td>15.12</td>
<td>12.93</td>
</tr>
<tr>
<td>7.22</td>
<td>6.86</td>
<td>13.11</td>
<td>12.93</td>
</tr>
<tr>
<td>8.84</td>
<td>8.44</td>
<td>13.48</td>
<td>11.32</td>
</tr>
</tbody>
</table>

Table 3.11.

Descriptive statistics of raw data of Furness & Maschette (2009) for healthy community dwelling older adults at baseline and after six weeks of WBV

<table>
<thead>
<tr>
<th>Variable</th>
<th>TUG Pre</th>
<th>TUG Post</th>
<th>CHAIR Pre</th>
<th>CHAIR Post</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>10.44</td>
<td>9.11</td>
<td>16.05</td>
<td>13.11</td>
</tr>
<tr>
<td>Standard Deviation</td>
<td>3.12</td>
<td>2.71</td>
<td>4.00</td>
<td>2.35</td>
</tr>
<tr>
<td>Variance</td>
<td>9.72</td>
<td>7.37</td>
<td>15.98</td>
<td>5.54</td>
</tr>
<tr>
<td><strong>Kurtosis</strong></td>
<td><strong>2.34</strong></td>
<td><strong>2.78</strong></td>
<td>-0.04</td>
<td>1.18</td>
</tr>
<tr>
<td>Skewness</td>
<td>1.47</td>
<td>1.76</td>
<td>0.66</td>
<td>1.24</td>
</tr>
</tbody>
</table>

Bold font indicates violation of normality.
Given the values of kurtosis violated the assumption of normality, reciprocal transformation (RT) was computed of the data (equation 1) (Portney & Watkins, 2000). With equation 1, ‘RT’ was the datum value of the reciprocal transformation computation and ‘x’ was the raw datum value. Descriptive statistics of the transformed data are shown in table 3.12.

\[ RT = \frac{1}{x} \]  
\[ \text{equation 1} \]

**Table 3.12.**

**Descriptive statistics of raw data after reciprocal transformation**

<table>
<thead>
<tr>
<th>Variable</th>
<th>TUG Pre</th>
<th>TUG Post</th>
<th>CHAIR Pre</th>
<th>CHAIR Post</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>0.10</td>
<td>0.12</td>
<td>0.07</td>
<td>0.08</td>
</tr>
<tr>
<td>Standard Deviation</td>
<td>0.03</td>
<td>0.03</td>
<td>0.02</td>
<td>0.01</td>
</tr>
<tr>
<td>Variance</td>
<td>0.01</td>
<td>0.01</td>
<td>0.01</td>
<td>0.01</td>
</tr>
<tr>
<td>Kurtosis</td>
<td>0.22</td>
<td>-0.39</td>
<td>-0.27</td>
<td>0.23</td>
</tr>
<tr>
<td>Skewness</td>
<td>-0.04</td>
<td>-0.59</td>
<td>0.35</td>
<td>-0.50</td>
</tr>
<tr>
<td>Correlation coefficient</td>
<td>0.920</td>
<td>0.810</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

The method of paired t-test (equation 2 and 3; Portney & Watkins, 2000, Table C.2 p, 720) was used to estimate sample size from the data of Furness and Maschette (2009). With equation 2, d’ was based on paired score and ‘s’ was the common standard deviation. After calculation of d’, the effect size index; d was calculated with equation 3, where ‘r’ was the correlation coefficient for the paired data.

\[ d' = \frac{x_1 - x_2}{s} \]  
\[ \text{equation 2} \]

\[ d = \frac{d}{1 - r} \]  
\[ \text{equation 3} \]
Calculation of the means of the pairs by equation 2 and the effect size index by equation 3 for the 5-chair test:

\[ d' = \frac{0.08 - 0.07}{0.02} \]

\[ \therefore d' = 0.50 \]

\[ d = \frac{0.50}{1 - 0.81} \]

\[ \therefore d = 1.15 \]

Calculation of the means of the pairs by equation 2 and the effect size index by equation 3 for the TUG test:

\[ d' = \frac{0.12 - 0.10}{0.03} \]

\[ \therefore d' = 0.67 \]

\[ d = \frac{0.67}{1 - 0.92} \]

\[ \therefore d = 2.37 \]

Sample size needed for a one tailed paired \( t \)-test \( \alpha \) 0.05 and \( n \) = 14 with power 0.90:

The 5-chair test estimated \( n = 16 \)

The TUG test estimated \( n = < 10 \)
3.6 Statistical Processes: Normality, Effect Size and Partial Eta Squared

Normality

All data were checked for normality according to an established standard (Bluman, 1997; Macellari, Giacomozzi & Saggini, 1999; Vincent, 1999; Portney & Watkins, 2000; Coakes, Steed & Dzidic, 2006). The assumptions of normality comprised: (1) the distribution of the population data scores were normal, (2) the data should not exceed ± 2 for skewness and kurtosis, (3) the assumption of sphericity was met, and (4) Levene’s statistics for homogeneity of variance was not violated (Macellari, Giacomozzi & Saggini, 1999; Vincent, 1999; Portney & Watkins, 2000; Coakes, Steed & Dzidic, 2006). When the assumption of sphericity was not met, the Greenhouse-Geisser correction factor was used (Portney & Watkins, 2000).

Effect Size

Effect size was calculated with equation 4, in which ‘SD_{pooled}’ was the pooled standard deviation of the baseline and instantaneous WBV data (Vincent, 1999; Thalheimer & Cook; 2001). Specifically, effect size was calculated for paired t-tests of Study 1 (Chapter 4). The effect size was expressed as Cohen’s $d$. Effect size 0.2 was considered small, 0.5 moderate and $> 0.8$ large effect (Winer, Brown & Michels, 1991).

\[
d = \frac{x_1 - x_2}{SD_{pooled}} \tag{equation 4}
\]

\[
SD_{pooled} = \frac{n_1 - 1 \cdot SD_1^2 + n_2 - 1 \cdot SD_2^2}{n_1 + n_2} \tag{equation 5}
\]
Partial Eta Squared

Partial eta-squared (Partial $\eta^2$) was computed to describe strength of association for repeated measures analysis of variance (RM-ANOVA) (Pallant, 2005). Specifically, Partial $\eta^2$ was calculated for dependent variables of Study 2 (Chapter 5). Partial eta-squared of 0.01 was small effect, 0.06 moderate effect and 0.14 large effect (Cohen, 1988).

$$\text{Partial } \eta^2 = \frac{SS_{between}}{SS_{total} + SS_{error}}$$ equation 6
CHAPTER 4. MAJOR STUDY 1: SAFETY OF WBV FOR PEOPLE WITH COPD

4.1 Introduction

A justification for the use of WBV for the management of stable COPD, specifically relating to exercise tolerance and functional performance was presented in Chapters 1 and 2. The rationale was speculative, because the efficacy of WBV has not been established as a standalone mode of exercise. Furthermore, safety of WBV has never been established for people with COPD. Three studies however, two of which involve WBV and lung function of cystic fibrosis sufferers (Roth et al., 2008; Rietschel et al., 2008) and another on WBV and heart transplant recipients (Crevenna et al., 2003) supported the safe use of WBV. Specifically, experiences with WBV did not exacerbate each pathology.

Position statements from respiratory bodies including the American Thoracic Society and the European Respiratory Society support the use of resistance training and aerobic conditioning. Generally, those modes of physical activity are regarded as safe and effective modes to improve exercise tolerance of people with COPD (Nici et al., 2006; GOLD, 2007). As such, people with COPD are expected to respond safely to varied modes of peripheral muscle training, yet it remains unknown if people with COPD would respond safely to WBV. Furthermore, WBV may be an easier mode of exercise to perform for people with COPD than resistance training and aerobic conditioning because of low skill demand. Whole-body vibration in a community setting is yet to be determined as an exercise mode well tolerated and capable of improving functional performance of the lower limbs of people with COPD.
However, the safety of a single session of WBV in a community setting should first be established before long-term interventions can be introduced.

4.2 Aim and Objectives

The aim of this research is to describe effects of a single exercise session of WBV on selected psychological and physiological responses in people with COPD. Specifically, the aim is to determine the safety of WBV by quantifying rating of perceived dyspnoea, heart rate, and saturation of haemoglobin for people with COPD during a WBV bout. Physical activity, such as treadmill walking (Clark et al., 2000) and resistance training (Panton et al., 2004) routinely increase dyspnoea, even for healthy participants, yet those modes were considered necessary of pulmonary rehabilitation (GOLD, 2007). Potentially less strenuous and less known, effects of WBV on COPD should be cautiously introduced. Therefore, the objectives of major study 1 were to:

- Quantify and describe effects of a single session of WBV on rating of perceived dyspnoea, heart rate, and saturation of haemoglobin prior to and during a bout of WBV for people with COPD.

Research Hypothesis

The following research hypothesis was tested to describe effects a single session of WBV on selected psychological and physiological responses of exercise of people with COPD:

\[ H_1: \text{For people with COPD, a single session of WBV: (1) will increase rating of perceive dyspnoea, (2) will increase heart rate, and (3) will reduce saturation of haemoglobin.} \]
4.3 Methods

Participants

Participants were recruited via two independent methods: (1) advertising brochure and information sessions at COPD support group meetings (Appendix K), and (2) mail out to patients of the Monash Medical Centre, Department of Respiratory and Sleep Medicine, Monash Medical Centre, Southern Health (Appendix F). After interest was attracted, each participant was provided with an information letter and consent form. The Australian Catholic University Human Research Ethics Committee (Appendix L) and the Southern Health Human Research Ethics Committee A (Monash Medical Centre) (Appendix H) approved the procedures. A convenience sample of 17 adults with COPD (mean age = 69 years ± 8, mean stature = 1.7 metres ± 0.9, mean mass = 83.9 kilograms ± 19.2) provided informed voluntary consent and were included in the study. The cohort was drawn from metropolitan Melbourne and the Mornington Peninsula, Victoria, Australia. Data were collected in the home of each participant, April through May, 2011.

Selection Criteria

The selection criteria and selection protocol used in this research are more comprehensively described in Chapter 3 (pages 105 to 109). Essentially participants were: (1) affected by COPD, and (2) community dwelling. A third criterion specific for WBV was added to this research: (3) the potential participant satisfied WBV intervention inclusion guidelines and is free of WBV contraindications.
To satisfy the third criterion, all participants had to report being free of self reported; vascular disease, reactive arthritis, vertigo, and risk of thromboembolism (Runge, Rehfeld & Resnicek, 2000; Roelants, Delecluse & Verschueren, 2004; Bruyere, Wuidart, Di Palma, Gourlay, Ethgen, Richy et al., 2005). A list of contraindications for WBV was tentatively suggested by Cardinale and Rittweger (2006). Table 4.1 shows the potential problems associated with varying sub-optimal health conditions and WBV. Notably, COPD is not a listed condition. Participants with any condition described in table 4.1 were not included in this research.

Table 4.1

Contraindications for WBV and people of sub-optimal health (Cardinale & Rittweger, 2006)

<table>
<thead>
<tr>
<th>Condition</th>
<th>Potential Problem</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diabetes with neuropathy and ulceration</td>
<td>Lack of sensation or worsening ulcers</td>
</tr>
<tr>
<td>Recent venous thrombosis</td>
<td>Pulmonary embolism</td>
</tr>
<tr>
<td>Recent fracture</td>
<td>Instability/re-fracture</td>
</tr>
<tr>
<td>Osteosynthesis in lower limbs containing metal implants</td>
<td>Discomfort</td>
</tr>
<tr>
<td>Spinal tumours or metastases</td>
<td>Instability</td>
</tr>
<tr>
<td>Acute vertebral disc herniation</td>
<td>Instability</td>
</tr>
<tr>
<td>Recent abdominal surgery</td>
<td>Operative result may be compromised</td>
</tr>
<tr>
<td>Pregnancy</td>
<td>Unknown risk</td>
</tr>
<tr>
<td>Hip or knee endoprosthesis or other metal implants</td>
<td>Unknown risk</td>
</tr>
<tr>
<td>Aortic aneurism</td>
<td>Risk of rupture</td>
</tr>
</tbody>
</table>
Test Procedure

Data were collected in the homes of the 17 participants. The community setting was selected ahead of the University laboratory and the Monash Medical Centre because: (1) the home was more suited to the convenience of the participant, (2) space within the Monash Medical Centre was unavailable, (3) the instruments were transportable, and (4) the safety of WBV that could be potentially used in a community setting needed to be established.

The independent variable was WBV. A side alternating vibration platform (Amazing Super Health, Melbourne, AUS) was used in the homes of the participants. For each vibration bout, the vibration platform frequency was set at 25 Hz with a peak-to-peak displacement at 2.0 mm, peak acceleration was ~24.67 m.s\(^{-2}\), and the resulting gravitational force was ~2.52 g (cognisant of the findings of pilot test 1). Validity of the vibration platform peak-to-peak displacement and frequency were established prior to commencement of the test procedure (pilot test 1, Appendix A). Foot placement (second toe) was equidistant, 20 cm from axis of rotation. The participant wore flat soled shoes. Skidding was checked according to the recommended method of the International Society of Musculoskeletal and Neuronal Interactions (Rauch et al., 2010). A piece of paper was placed under the foot during a vibration bout to ensure constant contact with the vibration platform (Rauch et al., 2010). The method allows determination of the WBV parameters (i.e., vibration platform frequency, peak-to-peak displacement, and resultant gravitational force) (Rauch et al., 2010). The participant stood with ~40° knee flexion. Despite the position being checked with a goniometer, previous work by the student researcher showed knee flexion angle to vary (37° ± 4°) independent of a request to maintain 40° knee flexion among healthy young adults (table 3.4 and Appendix C).
The single session of WBV consisted of five, 60 second vibration bouts interspersed with 60 seconds of passive rest. Although the most effective protocol is yet to be established, the selected protocol had been used previously to establish efficacy of WBV interventions in healthy young adults (Cardinale & Lim, 2003; Cronin, Oliver & McNair, 2004) and non-community dwelling old adults (i.e., nursing home residents, Bruyere, et al., 2005). During rest, the participant remained on the vibration platform, but was encouraged to stand with a posture that mimicked the ‘anatomical position’ in accordance with the guidelines of the International Society for the Advancement of Kinanthropometry. More specifically, the participant assumed a standing position with the feet close together, the arms to the side, and the head, eyes, and palms facing forwards.

Data Collection and Instruments

Anthropometric data (stature and mass) were collected at baseline. Data of selected physiological responses to exercise were also collected at baseline (before the first WBV bout) and during the fifth and final WBV bout (after 30 seconds of the 60 second bout). Clinical spirometry tests were conducted no more than 12 months prior to inclusion in the study as part of usual care for the participant at the Monash Medical Centre.

The Borg CR-10 VAS (Appendix J) was used to quantify the rating of perceived level of breathing limitation so that dyspnoea during WBV could be quantified. Reliability of the VAS for dyspnoea was previously established (Mahler, 1992; Mahler, Mejia-Alfar, Ward & Baird, 2001; Borg et al., 2010). Population specific reliability of the VAS was acceptable (ICC ≥ 0.700, George & Mallery, 2003) mindful that the CV was almost one third of the mean (Chapter 3, pilot test 3). To anchor rating of perceived breathlessness data during the
single session of WBV, resting rating of perceived dyspnoea was collected after each participant had been sitting quietly in a chair for five minutes.

Dependent variables were heart rate and saturation of haemoglobin (SpO₂), both quantified with the CARESCOPE™ V100 Vital Sign Monitor (GE Health Care, Milwaukee, USA). At baseline, those data were collected after each participant had been sitting quietly in a chair for five minutes. Population specific reliability of the device is acceptable (ICC ≥ 0.700, George & Mallery, 2003) for heart rate and saturation of haemoglobin (Chapter 3, pilot test 3).

Heart rate reserve (HRR) was calculated to allow comparison with other studies of physical activity. Heart rate reserve was calculated with the Karvonen method (Karvonen, Kentala & Mustala, 1957) (equation 2). Where ‘MRH_pred’ was the predicted maximum heart rate calculated by 220 beats.min⁻¹ – age in years, and ‘RHR’ was the resting heart rate quantified after each participant had been sitting quietly in a chair for five minutes.

\[
HRR = MRH_{pred} - RHR \quad \text{equation 2}
\]

The percentage of heart rate reserve (HRR%) was calculated with the method of Colberg, Swain & Vinik (2003) (equation 3). Where ‘Δ HR’ was the increment in heart rate from RHR during WBV.

\[
HRR\% = \frac{\Delta HR}{HRR} \times 100\% \quad \text{equation 3}
\]
The HRR can describe the relative acute effects of physical activity (Cornelissen, Verheyden, Aubert & Fagard, 2010). However, HRR% is calculated considering the HRR and is therefore relative to each individual even if the age in years is the same. As such, a HRR value of 0% is the equivalent of rest and can allow comparison among individuals even if their resting heart rates differ.

**Statistical Procedures**

The Statistical Package for Social Scientists (SPSS Version 19.0, Chicago IL™) was used to compute descriptive statistics of participant anthropometric, spirometry and dependent variables. Inferential statistics were computed to test the research hypothesis. Parametric statistical methods were used to calculate inferential statistics. Paired-sample \( t \)-tests were computed for Borg CR-10, heart rate and, saturation of haemoglobin. The paired analyses were within baseline and during WBV. Significance was accepted at \( p \leq 0.05 \). Effect size was calculated and expressed as Cohen’s \( d \) (Chapter 3, section 3.6).

**4.4 Results**

Participant anthropometric and selected descriptors are shown in table 4.2. Descriptive and inferential statistics are shown to describe psychological and physiological effects of a single session of WBV on people with COPD. Participant spirometry data are shown in table 4.3. Descriptive and inferential statistics of the research hypothesis are shown in table 4.4.
Mean age of the four females and 13 males was 69 years, which is ‘older adulthood’ (Gallahue & Ozmun, 2006). Mean body mass index (BMI) was 24.7, which is considered ‘normal’ (WHO, 2012). The oldest participant was 84 years. The youngest participant was 56 years (table 4.2).

Table 4.2

Anthropometric and descriptive statistics of the participants with COPD

<table>
<thead>
<tr>
<th>Descriptor</th>
<th>Mean</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>69</td>
<td>8</td>
</tr>
<tr>
<td>Stature (m)</td>
<td>1.7</td>
<td>0.9</td>
</tr>
<tr>
<td>Mass (kg)</td>
<td>83.9</td>
<td>19.2</td>
</tr>
<tr>
<td>BMI (kg.m⁻²)</td>
<td>24.7</td>
<td>5.1</td>
</tr>
<tr>
<td>Pack Years</td>
<td>53</td>
<td>48</td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>136</td>
<td>15</td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td>73</td>
<td>11</td>
</tr>
<tr>
<td>Resting HR (beats.min⁻¹)</td>
<td>81</td>
<td>12</td>
</tr>
<tr>
<td>Predicted maximum HR (beats.min⁻¹)</td>
<td>148</td>
<td>7</td>
</tr>
<tr>
<td>HRR (Karvonen)</td>
<td>67</td>
<td>14</td>
</tr>
</tbody>
</table>

Note: N = 17 (four females, 13 males). BMI: body mass index. Pack years: product of packets of cigarettes per day across years (i.e., one pack year = 365 packets of cigarettes). Pack years range was 10.5 to 140 pack years. SBP: systolic blood pressure. DBP: diastolic blood pressure. HR: heart rate. HRR: heart rate reserve with the Karvonen method.

According to GOLD (2007), 14 participants were affected by moderate Stage II COPD (table 4.3). Mean FEV₁ was less than 80% predicted. Mean FER was less than 70%. Three participants were affected by severe Stage III COPD (i.e., FEV₁ < 50 % predicted).
Table 4.3

Spirometry descriptors of the participants with COPD prior to and after bronchodilator medication

<table>
<thead>
<tr>
<th>Descriptor</th>
<th>Pre Mean</th>
<th>SD</th>
<th>95% CI</th>
<th>Post Mean</th>
<th>SD</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>FEV₁ (L BTPS)</td>
<td>1.4</td>
<td>0.5</td>
<td>± 1.0</td>
<td>1.6</td>
<td>0.6</td>
<td>± 1.2</td>
</tr>
<tr>
<td>FEV₁ % predicted</td>
<td>52.1</td>
<td>17.5</td>
<td>± 34.3</td>
<td>58.2</td>
<td>19.4</td>
<td>± 38.0</td>
</tr>
<tr>
<td>FVC (L BTPS)</td>
<td>2.8</td>
<td>0.8</td>
<td>± 1.6</td>
<td>3.1</td>
<td>0.8</td>
<td>± 1.6</td>
</tr>
<tr>
<td>FVC % predicted</td>
<td>80.3</td>
<td>16.1</td>
<td>± 31.6</td>
<td>84.5</td>
<td>17.3</td>
<td>± 33.9</td>
</tr>
<tr>
<td>FER %</td>
<td>52.0</td>
<td>13.7</td>
<td>± 26.9</td>
<td>52.7</td>
<td>11.8</td>
<td>± 23.1</td>
</tr>
<tr>
<td>PEF (L sec⁻¹)</td>
<td>3.8</td>
<td>1.4</td>
<td>± 2.7</td>
<td>4.2</td>
<td>0.9</td>
<td>± 1.8</td>
</tr>
</tbody>
</table>

Note: Three participants were classified as severe Stage III COPD. FEV₁: forced expired volume of oxygen in the first second. FVC: forced vital capacity of the lungs. FER: forced expiratory ratio (FEV₁/FVC⁻¹). PEF: peak expiratory flow. L BTPS: Litres Body Temperature Pressure Saturated. Pre: before 0.3 mg Salbutamol inhalation. Post: 15 minutes after 0.3 mg Salbutamol inhalation. 95% CI: confidence interval [x ± 1.96(SD)]

Selected psychological and physiological responses to a single session of WBV are shown in table 4.4. There was a moderate effect of WBV on rating of perceived dyspnoea (t = 4.01, df = 16, p = 0.01). With the Borg CR-10 VAS, rating of perceived dyspnoea increased from ‘very slight’ to ‘slight’. There was a large effect of WBV on heart rate (t = 10.8, df = 16, p = 0.01, Cohen’s d = 0.91). Resting HR was 55% of predicted maximum and 62% during WBV. Heart rate reserve was 16% (± 29%) during WBV; the range was 6 to 38%. Saturation of haemoglobin changed during WBV by 1% (t = 1.89, df = 16, p = 0.08).
Table 4.4

Effects of WBV on rating of perceived dyspnoea, heart rate, and saturation of haemoglobin

<table>
<thead>
<tr>
<th>Dependent Variable</th>
<th>Mean</th>
<th>SD</th>
<th>95% Confidence Lower</th>
<th>95% Confidence Upper</th>
<th>Δ</th>
<th>Sig.</th>
<th>Effect size</th>
</tr>
</thead>
<tbody>
<tr>
<td>Borg CR-10 baseline</td>
<td>1</td>
<td>2</td>
<td>0</td>
<td>2</td>
<td>1</td>
<td>0.01</td>
<td>0.55</td>
</tr>
<tr>
<td>Borg CR-10 during WBV</td>
<td>2</td>
<td>2</td>
<td>1</td>
<td>3</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HR baseline (beats.min⁻¹)</td>
<td>81</td>
<td>12</td>
<td>75</td>
<td>87</td>
<td>11</td>
<td>0.01</td>
<td>0.91</td>
</tr>
<tr>
<td>HR during WBV (beats.min⁻¹)</td>
<td>92</td>
<td>13</td>
<td>85</td>
<td>99</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SpO₂ baseline (%)</td>
<td>97</td>
<td>2</td>
<td>96</td>
<td>97</td>
<td>-1</td>
<td>0.08</td>
<td>0.55</td>
</tr>
<tr>
<td>SpO₂ during WBV (%)</td>
<td>96</td>
<td>2</td>
<td>95</td>
<td>97</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HRR %</td>
<td>16</td>
<td>29</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note: Effect size is Cohen’s $d$ where 0.2 was considered small, 0.5 moderate and > 0.8 large effect (Winer, Brown & Michels, 1991). SpO₂: saturation of haemoglobin. HR: heart rate. HRR%: percentage of heart rate reserve. 95% Confidence with standard error (SE).

4.5 Discussion

Major Findings

In this first major study, the safety of WBV was explored via selected psychological and physiological responses to exercise. Three major findings emerged to support the safeness of WBV for people with COPD: (1) WBV was associated with a minimal increase in rating of perceived dyspnoea from ‘very slight’ to ‘slight’ with moderate effect, (2) WBV was associated with an increased heart rate 7% higher than resting with large effect, and (3) WBV was associated with a minimal reduction of 1% in saturation of haemoglobin.

Dyspnoea

In this research, a single session of WBV was associated with an increase in dyspnoea from 1 ‘very slight’ at baseline to 2 ‘slight’ during the final WBV bout for people with COPD. The results are supported by other research using exercise modes of various intensities.
Resistance training increased Borg CR-10 to a value of 3 ‘moderate’ after three sets of 10 to 12 repetitions for people with moderate Stage II COPD (Panton et al., 2004). Treadmill walking to subjective exhaustion caused dyspnoea to increases to 4 ‘somewhat severe’ on the Borg CR-10 scale for moderate Stage II COPD participants (Clark et al., 2000). The six-minute walk test also raised dyspnoea to the same value of ‘4’ (Breyer et al., 2010). Dyspnoea was perceived to be as high as a value of 7 ‘very severe’ during 70 to 80% peak limit of tolerance cycling among people with Stage III COPD (Oliveira et al., 2010). The habitual sustainability of exercise that can cause dyspnoea to rise to levels perceived as ‘severe’ and above could however, be questioned.

At baseline, the participants of this research perceived ‘very slight’ dyspnoea. In another study, people with moderate Stage II COPD perceived dyspnoea as ‘slight’ during a similar period of rest (Pickard, Yang & Lee, 2011). Within the patients with moderate Stage II COPD, FEV₁ was 65.5% predicted (± 9.1%), while in the current study FEV₁ was 52.1% predicted (± 17.5%). The broader distribution of the spirometry data about the mean in the current research vary because Stage II and Stage III participants were analysed collectively. Although COPD disease severity affects physical activity participation (Garcia-Aymerich et al., 2006), the clinical merit of ‘slight’ and ‘very slight’ dyspnoea could be negligible.

In a recent study, people with severe Stage III COPD perceived resting dyspnoea as ‘slight’, increasing to ‘severe’ after three minutes of self determined ‘fast paced exercise’ (O’Driscoll, Neill, Pulakal & Turkington, 2011). Despite a definition of ‘fast paced’, the exact protocol of exercise was lacking. It seems that an increased level of perceived breathlessness among people with moderate and severe COPD is complicit with resistance training and aerobic
conditioning. Furthermore dyspnoea classified as ‘severe’ can be expected during physical activity.

Possible Mechanisms of Increased Dyspnoea with WBV

The mechanism for increased dyspnoea can partly be attributed to oxygen demand of the task and supply of oxygen through the respiratory system (i.e., emphysematous change). Given that many factors affect dyspnoea such as systemic inflammation, diet, oxygen supply at the cellular level, and diffusing capacity (Serres et al., 1998; Debigaré, Côté & Maltais, 2001; Augusti, et al., 2003; GOLD, 2007; Wüst & Degens, 2007), essentially, limitation of “exertional dyspnoea” during physical activity is primary for both pharmacological and non-pharmacological interventions among people with COPD (Nishiyama, Taniguchi, Kondoh, Kimura, Kato, Ogawa, et al., 2007, p 834). However, this research could only allow speculative comment about mechanisms of dyspnoea because none of the aforementioned variables were quantified.

Heart Rate and Saturation of Haemoglobin

COPD

In this research, heart rate increased from 81 beats.min⁻¹ at baseline to 92 beats.min⁻¹ during a single session of WBV. Observation of increased heart rate is supported by other research using exercise modes of various intensities in people with COPD. For people with moderate Stage II COPD, combined resistance training and aerobic conditioning increased heart rate to 125 beats.min⁻¹ (Panton et al., 2004). For people with severe Stage III COPD, heart rate increased from 86 to 99 beats.min⁻¹ after self determined ‘fast paced exercise’ (O’Driscoll et al., 2011) and to 115 beats.min⁻¹ at a treadmill walking speed determined to increase dyspnoea.
on the Borg CR-10 scale to ‘severe’ (Martin & Davenport, 2011). It may be that the increase in heart rates observed in this research can be attributed to an ‘exercise’ effect of WBV that is commonly associated with resistance training and aerobic conditioning.

Concurrently, saturation of haemoglobin significantly reduced to 90% during perceived ‘severe’ aerobic conditioning, yet those data were considered safe and standard for exercise training for people with COPD (Martin & Davenport, 2011). Other physical activity modes have reduced saturation of haemoglobin to 89% for people with COPD (Oliveira et al., 2010; O’Driscoll et al., 2011). Results from this research show saturation of haemoglobin was not significantly reduced (Δ mean 1%, p = 0.08). Saturation of haemoglobin was reduced from 97 to 96% during WBV however, the clinical significance may be negligible despite the moderate effect size. Given the negative correlation of diffusing capacity and saturation of haemoglobin (West, 2008), the slight reduction of SpO₂ was expected. Contemporary safety recommendations are that exercise should be terminated if saturation of haemoglobin reduces to ≤ 85% (Poulain et al., 2003; Jenkins, Hill & Cecins, 2010). The effect of WBV on saturation on haemoglobin in this study remained below the threshold of safe exercise.

**WBV**

For participants in this research %HRR was 16% during WBV. The result was similar to a study of heart transplant recipients, in whom %HRR during WBV was 14% (Crevenna et al., 2003). Interestingly, for community dwelling older adults, HRR during synchronous WBV increased to as much as 62% (Bogaerts, Delecluse, Claessens, Troosters, Boonen & Verschueren, 2009). Participants in that study however, performed dynamic movement on the
vibration platform that may have concurrently affected the results. The participants with COPD in this research maintained a static isometric squat.

The gravitational force of the vibration platform in this research was ~2.52 g. For non-community dwelling older adults, heart rate increased to 135 beats.min\(^{-1}\) when the gravitational force was 9.5 g (Bruyere et al., 2005). Exposure to WBV at 8 g in healthy adults elicited a %HRR to 8% (Jacobs & Burns, 2009). It may be that irrespective of gravitational force and health status, WBV elicited physical stress on the body that stimulated responses synonymous with exercise. Muscular contraction about the lower limbs quantified by EMG, for example, significantly increased during WBV (Bosco, Cardinale & Tsarpela, 1999; Rittweger, Mutschelknauss & Felsenberg, 2003; Di Giminiani et al., 2009). As such, increased muscular activity during WBV increased oxygen uptake and heart rate (Rittweger, Beller & Felsenberg, 2000).

Nevertheless, it is suspected that high gravitational forces caused by WBV could damage fragile bones (Kiiski et al., 2008). Subsequently, the appropriateness of prescribed doses of WBV should be at the forefront of exercise intervention planning for people with COPD. Perhaps due to long term medication (e.g., corticosteroids), limited mobility, or other physiological or lifestyle related factors, people with moderate Stage II COPD are known to have lower bone mineral density than healthy matched controls (Dam, Harrison, Fink, Ramsdell & Barrett-Connor, 2011; Duckers, Evans, Fraser, Stone, Bolton & Shale, 2011). Bone fragility therefore becomes an important consideration with WBV for people with COPD.
Rating of perceived dyspnoea may also increase during exercise due to uncontrolled psychological factors. As such, the Borg CR-10 VAS has been limited when participants have used the scale with an inconsistent procedure (Shen & Parsons, 1997). That is, mean ratings were confounded with dyspnoea and intra-individual variability. Recently though, the Borg CR-10 VAS was strongly correlated to leg fatigue during exercise for healthy young adults (Borg et al., 2010). It was noted that during moderate to strong intensity exercise, for people with COPD, symptom limited dyspnoea may be the major cause of discomfort rather than leg fatigue (Borg et al., 2010). For participants in this research however, the intensity of exercise was low, which was associated with a minimal increase in rating of perceived dyspnoea during WBV.

This research associated WBV to increase dyspnoea. Although to a lesser extent than other modes of exercise (Breyer et al., 2010; Oliveira et al., 2010), a single session of WBV in this research may have increased oxygen uptake for people with COPD. This may be because an increase in oxygen demand is expected during WBV with a static stance posture (Rittweger, Schiessl & Felsenberg, 2001). Specifically, static stance WBV increased oxygen uptake ($\text{VO}_2$) from 4.8 ml.kg$^{-1}$.min$^{-1}$ to 10.2 ml.kg$^{-1}$.min$^{-1}$ in healthy adults (Rittweger, Schiessl & Felsenberg, 2001). That was equivalent to a rise in energy expenditure of 1.5 MET (metabolic equivalents) to 2.9 MET. Recently 2.9 MET is the upper threshold of low intensity physical activity for older adults (Borjesson, Urhausen, Kouidi, Dugmore, Sharma, Halle et al., 2011). The long-term effect of WBV on oxygen uptake for healthy older adults was a 21% increase of $\text{VO}_2\text{peak}$ over 12 months (Bogaerts et al., 2009).
The aforementioned studies (Rittweger, Schiessl & Felsenberg, 2001; Bogaerts et al., 2009) show an immediate effect of WBV on selected physiological responses to exercise, and a long-term effect of WBV on the cardiovascular system. The gravitational force of those two studies was not less than 2.9 g. The gravitational force of WBV in this research was ~2.5 g. Quantification of oxygen uptake was not possible because data were collected in a community setting in this research. However, the rise in dyspnoea may be attributed to greater oxygen demand associated with WBV even though the gravitational force was lower than had been used for other interventions. The notion of increased energy demands with low intensity WBV (i.e., ~2.5 g) is supported by the observed increase of heart rate of people with COPD. Given the results of this research, it is possible that a cardiovascular stimulus may be elicited by exposure to low intensity WBV.

**Limitations and Considerations**

This research was limited because current medication (other than bronchodilators and corticosteroids) and history of medication were neither recorded nor controlled. Rather, the priority was given to recruitment of participants whose COPD is stable. Specifically, participants were ‘well’ medicated in order to avoid potential exacerbations, and may have influenced the effects of WBV. However, being ‘well’ medicated during WBV has been observed in a population living in sub-optimal health (Crevenna et al., 2003). Similarly, it is likely that people managing their COPD in the community would medicate responsibly to maintain functional independence.
External validity was considered carefully before the conduct of this research. Given this research targeted a population living in sub-optimal health, the validity of everyday pharmacologic management would be sacrificed if participants were to be deprived normal pharmacologic care. Normal daily practices in COPD management had to be well represented, as well as the strong ethical decision of not interfering with usual care. Furthermore, considering the mantra ‘first do no harm’, WBV for people with COPD needed to be cautiously introduced.

The effect of sitting and standing postures on heart rate should be considered. Heart rate is shown to increase as much as 22 beats.min\(^{-1}\) once an individual stood (MacWilliams, 1933; Rittweger, Beller & Felsenberg, 2000; Martinmäki, Häkkinen, Mikkola & Rusko, 2008) and can be broadly explained by changes in blood pressure and other autonomic nervous system mechanisms. A similar change in heart rate was quantified in this research. It may be that the task of standing on the vibration platform increased heart rate rather than WBV. Perhaps there was a concurrent effect of standing and WBV on heart rate. This research was limited because only resting sitting heart rate data were collected and then compared with standing heart rate data during WBV. Further study is needed to describe the independent effects of standing and WBV on heart rate to understand effects of WBV for people with COPD. However, a placebo design could also be used to describe the effect of WBV on standing heart rate to address the limitation.

Body mass index of participants in this research was ‘normal’. Considering the global trend of obesity as estimated by BMI, subsequent studies of WBV and people with COPD may draw participants classified as ‘overweight’ and ‘obese’. Recently, people with and without a classification of ‘obesity’ in addition to COPD responded differently to six-minute walk test
distance (Bautista, Ehsan, Normandin, Zuwallack & Lahiri, 2011), although relative to body mass, measures of respiration (respiratory rate, $V\text{EO}_2$, RER) were the same. Similarly aerobic conditioning (80% limit of tolerance) of ‘normal’, ‘overweight’ and ‘obese’ people with COPD elicited different effects on performance, though relative to body mass, magnitude of performance improvement was the same (Sava, Laviolette, Bernard, Breton, Bourbeau & Maltais, 2010). Regardless of BMI, people with COPD respond safely to exercise interventions, with exacerbations of the disease rarely elicited (GOLD, 2007; McKenzie, Abramson, Crockett, Glasgow, Jenkins, McDonald et al., 2007; Grove, 2010). While cognisant of co-morbidities and contraindicators of COPD, future studies involving WBV should be considered to beneficially affect exercise tolerance, avoid exacerbations of the disease, and possibly, improve functional performance of the lower limbs.

4.6 Summary

This was the first study to quantify and describe effects of a single exercise session of WBV on selected psychological and physiological responses to exercise for people with COPD with the aim to quantify safety of WBV. The specific aim was to quantify several markers of safety surrounding the use of WBV. For the participants of this research, considering the nature of COPD and the known contraindications of the disease, a single session of WBV was associated with markers of psychological and physiological safety. Negligible changes in dyspnoea, saturation of haemoglobin, and heart rate during the single session of WBV were expected and support the use of WBV as a safe mode of exercise for people with COPD. Furthermore, changes of the markers of safety compared with resting results did not exceed safe thresholds reported with other safe modes of exercise.
Given the data were collected in the home of each participant with COPD, this research is unique. Whole-body vibration can be safely completed in the home of the individual with moderate, but likely negligible effect on dyspnoea (table 4.5). Modes of exercise that can beneficially affect, or at least maintain exercise tolerance, avoid exacerbations, and improve functional performance of the lower limbs of people with COPD is salient. Further study of community based WBV and effects on exercise tolerance and functional performance of the lower limbs of people with COPD should be conducted to establish efficacy, because results of this research support the use of WBV as a safe mode of exercise for people with COPD.

Table 4.5

Summary of research hypothesis and decision after inferential statistical analyses

<table>
<thead>
<tr>
<th>Research Hypothesis</th>
<th>Accept</th>
<th>Reject</th>
<th>Data</th>
<th>Interpretation</th>
</tr>
</thead>
<tbody>
<tr>
<td>WBV will increase Borg CR-10</td>
<td>✔</td>
<td></td>
<td>Increased from ‘very slight’ to ‘slight’</td>
<td>Negligible clinical significance for people with COPD</td>
</tr>
<tr>
<td>WBV will increase HR</td>
<td>✔</td>
<td></td>
<td>Increased 7%</td>
<td>Within safe responses reported of other exercise modes for people with COPD</td>
</tr>
<tr>
<td>WBV will reduce SpO₂</td>
<td>✔</td>
<td></td>
<td>Reduced 1%</td>
<td>Negligible clinical significance for people with COPD</td>
</tr>
</tbody>
</table>
CHAPTER 5. MAJOR STUDY 2: EFFICACY OF WBV ON EXERCISE TOLERANCE AND FUNCTIONAL PERFORMANCE OF THE LOWER LIMBS OF PEOPLE WITH COPD

5.1 Introduction

Following on from the safe WBV protocol for people with COPD identified with major study 1 (Chapter 4), a second study (major study 2) was conducted. The general aim of this research was to advance knowledge of effects of WBV on exercise tolerance and functional performance of the lower limbs of people with COPD in a community setting. Achieving the general aim of this research would determine efficacy of a WBV intervention to: (1) effect exercise tolerance while avoiding exacerbations of COPD that add to physical inactivity, and, (2) improve performance of ADLs of people with COPD. This research is registered with the Australian New Zealand Clinical Trials Registry, ANZCTR12612000508875:


To meet the general aim, participants were allocated to: (1) a six week WBV intervention, and then (2) a six week PLACEBO intervention. A two-week wash out interval interspersed the WBV and PLACEBO interventions. Given the nature of COPD, and exacerbations expected of it, this research was designed as a community based efficacy trial and was not conducted as a randomised controlled trial. For each intervention, data were collected at baseline and subsequent fortnights over the 14 week intervention period (i.e., pre-test, mid-test, and post-test). The methods described in Chapter 3 were used for major study 2 (i.e., selection criteria, selection criteria protocol, instruments, and data collection). Although this research was not completed as a randomised controlled trail, the CONSORT flow diagram (used to enhance
reporting of randomised controlled trails) is generated to describe enrolment, intervention allocation, and analysis (Moher, Schultz, Altman & the CONSORT Group, 2001) (figure 5.1).

The multiple challenges of participant recruitment are presented in Appendix M. Specifically, substantial effort was taken over a 24 month period to recruit participants for the 14 week community based efficacy trial through modes including: (1) hospital outpatient clinics, (2) private practice respiratory specialists, (3) community support networks, and (4) public information sessions. As such, being enrolled at a university without direct clinical links was problematic and time inefficient, but not insurmountable once a link was established with the Monash Medical Centre. Despite the multiple challenges presented in Appendix M, results of this research were robust because: (1) the sample sizes of the primary dependent variables were met with calculations based about an almost identical WBV intervention (i.e., Furness & Maschette, 2009), (2) the participants acted as their own control group because of the cross-over design of this research, and (3) the number of datum to dependent variable for each individual was as large as 173:1.
Figure 5.1. Flow of participation in the study based on the CONSORT flow chart for reporting randomised controlled trials.
Research Hypotheses (from Chapter 2)

The following research hypotheses were tested to determine efficacy of WBV on exercise tolerance and functional performance of the lower limbs of people with COPD:

Exercise Tolerance

H\textsubscript{1}: For people with COPD, a six week WBV intervention; two sessions per week of five 60 second bouts interspersed with 60 seconds passive rest will: (1) acutely increase dyspnoea during a WBV bout, (2) not elicit a long-term change of dyspnoea quantified with the Borg CR-10 visual analogue scale, (3) acutely increase heart rate and reduce saturation of haemoglobin during a WBV bout, and, (4) not elicit a long-term change of heart rate and saturation of haemoglobin.

H\textsubscript{2}: For people with COPD, there will be a difference among a six week WBV intervention and a six week PLACEBO intervention (two sessions per week of five 60 second bouts interspersed with 60 seconds passive rest) for dyspnoea, heart rate and saturation of haemoglobin.

Functional Performance

H\textsubscript{1}: For people with COPD, a six week WBV intervention; two sessions per week of five 60 second bouts interspersed with 60 seconds passive rest will improve: (1) performance of the timed-up-and-go test, (2) performance of the 5-chair stands test, and, (3) stride time, stride length and stride velocity quantified with a portable electronic walkway.

H\textsubscript{2}: For people with COPD, a six week WBV intervention will improve functional performance compared with a six week PLACEBO intervention (each intervention;
two sessions per week of five 60 second bouts interspersed with 60 seconds passive rest).

5.2 Methods

Participants

Participants were recruited from the Department of Respiratory and Sleep Medicine, Monash Medical Centre. The Department patient database was accessed by the Clinical Trials Manager. The Clinical Trials Coordinator sent an information letter to potential participants whom were then invited to record their interest in the study by contacting the student researcher (Appendix F). Medical consent was complicit within the approval of this research by the Department Head (Professor Philip Bardin). The Australian Catholic University Human Research Ethics Committee (Appendix G) and the Southern Health Human Research Ethics Committee A (Monash Medical Centre) (Appendix H) approved the procedures. A convenient sample of 16 community dwelling adults with COPD provided voluntary informed consent to participate (mean ± SD age = 72 ± 7 years, stature = 1.7 ± 0.1 metres, body mass = 85.7 ± 20.4 kilograms). Data were collected in the home of each participant, June through December, 2011.

Test Procedure

Data were collected in the 16 participants’ homes. The community setting was selected ahead of the University laboratory and the Monash Medical Centre because: (1) the home was more suited to the convenience of the participant, (2) space within the Monash Medical Centre was unavailable, (3) the instruments were transportable, (4) the safety of WBV for people with
COPD had been established after major study 1, and (5) a home-based protocol would maximise compliance.

To describe effects of the test procedure on day to day variation of dyspnoea a one-way ANOVA was computed for PLACEBO intervention data. It was important to know that changes in dyspnoea may be associated with WBV rather than the demand of completing the test procedure. Data were collected with the Borg CR-10 VAS in Week 9 immediately after: (1) the 5-chair test but prior to the first PLACEBO bout, (2) the gait trials but prior to the first PLACEBO bout, and, (3) during a PLACEBO bout. There was no effect of the test procedure on dyspnoea (F(2,30) = 0.62, p = 0.55).

**Independent and Dependent Variables**

The independent variables were ‘intervention’ with two levels: (1) WBV and, (2) PLACEBO and ‘test occasion’ with three levels: (1) pre-test, (2) mid-test, and (3) post-test. The dependent variables were selected variables of: (1) exercise tolerance (rating of perceived dyspnoea, heart rate, and saturation of haemoglobin), and (2) functional performance (TUG test, 5-chair test, stride length, stride time, and stride velocity).

The Borg CR-10 VAS (Appendix J) was used to quantify the perceived level of breathing limitation. Heart rate, saturation of haemoglobin and blood pressure were quantified with the CARESCOPETM V100 Vital Sign Monitor (GE Health Care, Milwaukee, USA). Reliability of the test procedures was no less than ‘acceptable’ (ICC ≥ 0.700, Chapter 3) for the three dependent variables.
Selected dependent variables of functional performance (i.e., TUG test, and 5-chair test) were quantified with a stop-watch. Kinematic variables of gait: stride length, stride time, and stride velocity were quantified with the GAITRite® Electronic Walkway (CIR Systems Inc, Peekskill, USA). Reliability of the test procedures was no less than ‘good’ (ICC ≥ 0.881, Chapter 3) for the five dependent variables.

Data of all dependent variables were collected to quantify at least one of three different data classifications: (1) long-term, (2) instantaneous, and (3) acute (table 5.2). Long-term data were collected at least 48 hours after a WBV bout (Cochrane, Legg & Hooker, 2004; Furness & Maschette, 2009). Instantaneous data were collected during the final WBV bout (after 30 seconds of the 60 second bout). Acute data were collected within two minutes of a completed WBV bout (Cardinale & Lim, 2003; Humphries et al., 2004).

Data were collected with the same procedure for each occasion (table 5.1). For long-term data, exercise tolerance data were collected first, followed by functional performance data. The procedures were designed to ensure that the most physically exertive test was completed last (i.e., the 5-chair test). Data collection of the PLACEBO intervention was identical to the procedure shown in table 5.2.
Table 5.1

Data collection procedure showing long-term, instantaneous, and acute variables for the WBV intervention and washout period

<table>
<thead>
<tr>
<th>Occasion</th>
<th>Session</th>
<th>Variable</th>
<th>Data</th>
</tr>
</thead>
<tbody>
<tr>
<td>Week 1 (pre)</td>
<td>Pre WBV</td>
<td>Borg CR-10</td>
<td>Baseline</td>
</tr>
<tr>
<td></td>
<td></td>
<td>heart rate</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>blood pressure</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>SpO₂</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>TUG</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>5-chair</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Gait</td>
<td></td>
</tr>
<tr>
<td>During WBV bout 5</td>
<td>Borg CR-10</td>
<td>Instantaneous</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>heart rate</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>SpO₂</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Gait</td>
<td></td>
</tr>
<tr>
<td>Post WBV</td>
<td></td>
<td></td>
<td>Acute</td>
</tr>
<tr>
<td>Week 3 (mid)</td>
<td>Pre WBV</td>
<td>Borg CR-10</td>
<td>Long-term</td>
</tr>
<tr>
<td></td>
<td></td>
<td>heart rate</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>blood pressure</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>SpO₂</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>TUG</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>5-chair</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Gait</td>
<td></td>
</tr>
<tr>
<td>During WBV bout 5</td>
<td>Borg CR-10</td>
<td>Instantaneous</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>heart rate</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>SpO₂</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Gait</td>
<td></td>
</tr>
<tr>
<td>Post WBV</td>
<td></td>
<td></td>
<td>Acute</td>
</tr>
<tr>
<td>Week 6 (post)</td>
<td>Pre WBV</td>
<td>Borg CR-10</td>
<td>Long-term</td>
</tr>
<tr>
<td></td>
<td></td>
<td>heart rate</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>blood pressure</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>SpO₂</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>TUG</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>5-chair</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Gait</td>
<td></td>
</tr>
<tr>
<td>During WBV bout 5</td>
<td>Borg CR-10</td>
<td>Instantaneous</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>heart rate</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>SpO₂</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Gait</td>
<td></td>
</tr>
<tr>
<td>Post WBV</td>
<td></td>
<td></td>
<td>Acute</td>
</tr>
<tr>
<td>Washout</td>
<td>At least 48 hours post WBV</td>
<td>TUG</td>
<td>Long-term</td>
</tr>
<tr>
<td></td>
<td></td>
<td>5-chair</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Gait</td>
<td></td>
</tr>
</tbody>
</table>

Note: Gait: stride length, stride time, and stride velocity. SpO₂: saturation of haemoglobin. The same data collection procedure was used for both WBV intervention and PLACEBO intervention.
**Intervention Design**

The WBV intervention consisted of 12 WBV sessions over a consecutive six week period. The participants completed two vibration sessions per week interspersed with at least 48 hours. The session frequency was determined using past work by the student researcher of healthy community dwelling older adults as a guide (Furness & Maschette, 2009; Furness, Maschette, Lorenzen, Naughton & Williams, 2010). The results show no main effect among two and three vibration sessions per week, though both session frequencies were significantly more effective than one session per week to improve functional performance of the lower limbs over a six week WBV intervention (Furness & Maschette, 2009).

In this research, a WBV session consisted of five, 60 second vibration bouts interspersed with 60 seconds of passive rest. Evidence suggests the test procedure was safe for people with COPD (Chapter 4). The design of the intervention is shown in table 5.2.

**Table 5.2**

<table>
<thead>
<tr>
<th>Intervention</th>
<th>Week 1</th>
<th>Week 3</th>
<th>Week 6</th>
<th>Washout</th>
<th>Cross-over</th>
<th>Week 9</th>
<th>Week 11</th>
<th>Week 14</th>
</tr>
</thead>
<tbody>
<tr>
<td>WBV</td>
<td>pre-test</td>
<td>mid-test</td>
<td>post-test</td>
<td>2 weeks</td>
<td>PLACEBO</td>
<td>pre-test</td>
<td>mid-test</td>
<td>post-test</td>
</tr>
</tbody>
</table>

Note: pre-test: data collected before the first WBV or PLACEBO bout and immediately after the final bout. mid-test: data collected before the seventh session of the WBV or PLACEBO intervention and immediately after the final bout of the sixth session. post-test: data collected immediately after the final bout of the twelfth session of the WBV or PLACEBO intervention and at least 48 hours after the final bout.
Instruments

Vibration Platforms

The vibration platform and skidding method used in major study 1 was also used for the WBV intervention of major study 2 (Chapter 4, pages 133 to 134). A prototype vibration platform was developed to deliver the PLACEBO intervention (Appendix N). For each PLACEBO vibration bout, the selected vibration platform frequency was 25 Hz and peak-to-peak displacement was ~0.0 mm, peak acceleration was ~0.00 m.s\(^{-2}\), gravitational force was ~0.0 g (cognisant that the Earth’s gravitational force is constant 1.0 g). Foot placement (second toe) was equidistant, 20 cm from the axis of rotation. The participants wore flat soled shoes. When the vibration platform was operational, the participants could hear the motor running and may have felt vibration. Participants were told the PLACEBO intervention was an “ultra-low frequency” vibration intervention that is “very different” to the WBV intervention.

Participants were told to stand with a bent knee posture that could be maintained for the duration of each WBV and PLACEBO bout. The procedure was based on evidence that transmission of WBV about the knee of healthy young adults was not different for 20, 40 and 60° knee flexion (Chapter 3, section 3.3). Knee flexion was checked manually with a goniometer after 30 seconds of every bout with the procedure described in Chapter 3 (section 3.3). During WBV, the participants stood with the knees flexed to 53° (SD ± 10°). During PLACEBO, participants stood with the knees flexed to 44° (SD ± 14°).
Data Analysis

Data of dependent variables of exercise tolerance and functional performance: rating of perceived dyspnoea, heart rate, saturation of haemoglobin, TUG test, and 5-chair test were recorded with pen and paper, then transposed to a SPSS for Windows spread sheet (SPSS Version 19.0, Chicago IL™). Functional performance gait data were imported to SPSS from the GAITRite software package. After calculation, with 0.90 power, sample size was estimated to be $n < 10$ for the TUG test and $n = 16$ for the 5-chair test (Chapter 3, section 3.5).

Descriptive Statistics

Descriptive statistics were calculated for all participants. All data were checked for normal distribution with methods previously described (Bluman, 1997; Macellari, Giacomozzi & Saggini, 1999; Vincent, 1999; Portney & Watkins, 2000; Coakes, Steed & Dzidic, 2006). The assumptions of normality are described in Chapter 3 (section 3.6).

Inferential Statistics

To test the research hypotheses of major study 2, parametric statistical methods were used to calculate inferential statistics. Repeated measures analysis of variance (RM-ANOVA) with repeated contrasts (WBV - Week 1 to Week 3; Week 3 to Week 6; PLACEBO - Week 9 to Week 11, Week 11 to Week 14) were computed for the dependent variables (rating of perceived dyspnoea, heart rate, saturation of haemoglobin, TUG test, 5-chair test, and gait variables). Despite a washout period, the RM-ANOVA was more appropriate than several one-way ANOVAs or a multivariate ANOVA because data were collected of the same participants across the WBV and PLACEBO interventions (personal correspondence with Professor Jennifer Peat, Medical Biostatistician). One-way ANOVA with least significant
difference post-hoc comparisons were computed for WBV intervention and PLACEBO intervention to compare long-term with acute data.

5.3 Results

Participants’ anthropometric and selected descriptors are shown in tables 5.4 and 5.5. Descriptive and inferential statistics to test the research hypotheses are shown to describe efficacy of WBV on exercise tolerance and functional performance of the lower limbs of people with COPD in tables 5.6 through 5.9. Raw data are shown in Appendix O.

Descriptive Statistics of Participants

Of the four females and 12 males recruited to this research, the youngest person was 56 years and the oldest 83 years. Mean BMI was 29.3 kg.m$^{-2}$, which is within the 'normal' range (WHO, 2012). The range of age in years was 56 to 84 years (table 5.3).

The method of Miller et al. (2005), described in Chapter 3 (section 3.4) was used for spirometry testing. According to GOLD (2007), the participants were affected by ‘moderate’ Stage II COPD. Mean FEV$_1$ was less than 80% predicted. Mean FER was less than 70%. No participants were using supplemented oxygen therapy as part of usual care. One participant however, was affected by ‘severe’ Stage III COPD (FEV$_1$ = 30% predicted) (table 5.4).
### Table 5.3

Anthropometric and descriptive statistics of the participants with COPD at baseline

<table>
<thead>
<tr>
<th>Descriptor</th>
<th>Mean</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>72</td>
<td>7</td>
</tr>
<tr>
<td>Stature (m)</td>
<td>1.71</td>
<td>0.1</td>
</tr>
<tr>
<td>Mass (kg)</td>
<td>85.7</td>
<td>20.4</td>
</tr>
<tr>
<td>BMI (kg.m(^{-2}))</td>
<td>29.3</td>
<td>6.1</td>
</tr>
<tr>
<td>Pack Years</td>
<td>52</td>
<td>45</td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>139</td>
<td>13</td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td>75</td>
<td>10</td>
</tr>
<tr>
<td>Resting HR (beats.min(^{-1}))</td>
<td>82</td>
<td>10</td>
</tr>
<tr>
<td>Predicted maximum HR (beats.min(^{-1}))</td>
<td>148</td>
<td>7</td>
</tr>
<tr>
<td>HRR (Karvonen)</td>
<td>67</td>
<td>11</td>
</tr>
</tbody>
</table>

Note: N = 16 (4 females, 12 males). BMI: body mass index. Pack Years: product of packets of cigarettes per day across years (i.e., one pack year = 365 packets of cigarettes). Pack years range was 10.5 to 140 pack years. SBP: systolic blood pressure. DBP: diastolic blood pressure. HR: heart rate. HRR: heart rate reserve with the Karvonen method.

### Table 5.4

Spirometry descriptors of the participants with COPD (one participant was classified as severe Stage III COPD)

<table>
<thead>
<tr>
<th>Descriptor</th>
<th>Pre</th>
<th>Post</th>
</tr>
</thead>
<tbody>
<tr>
<td>FEV(_1) (L BTPS)</td>
<td>1.5</td>
<td>1.7</td>
</tr>
<tr>
<td>FEV(_1) % predicted</td>
<td>53.0</td>
<td>58.5</td>
</tr>
<tr>
<td>FVC (L BTPS)</td>
<td>2.8</td>
<td>3.0</td>
</tr>
<tr>
<td>FVC % predicted</td>
<td>78.5</td>
<td>83.2</td>
</tr>
<tr>
<td>FER %</td>
<td>52.0</td>
<td>52.3</td>
</tr>
<tr>
<td>PEF (L.sec(^{-1}))</td>
<td>3.8</td>
<td>4.2</td>
</tr>
</tbody>
</table>

FEV\(_1\): forced expired volume of oxygen in the first second. FVC: forced vital capacity of the lungs. FER: forced expiratory ratio (FEV\(_1\).FVC\(^{-1}\)). PEF: peak expiratory flow. L BTPS: Litres Body Temperature Pressure Saturated. Pre: before 0.3 mg Salbutamol inhalation. Post: 15 minutes after 0.3 mg Salbutamol inhalation. 95% CI: confidence interval [x ± 1.96(SD)].
Effects of WBV and PLACEBO Interventions on Exercise Tolerance of People with COPD

Effects of the independent variable ‘test occasion’ were computed with repeated measures ANOVA with repeated contrasts of long-term and instantaneous results of each intervention on exercise tolerance. Test occasion and intervention results are shown in table 5.5. Data were collected to show a potential ‘training’ and/or ‘placebo’ effect of WBV. The long-term data were collected at rest across each intervention. The instantaneous data were collected during a WBV or PLACEBO bout. Effects of the independent variable ‘test occasion’ on exercise tolerance (long-term data compared with instantaneous data) are shown in figures 5.2, 5.3 and 5.4.

There were no long-term, or instantaneous effects of WBV on rating of perceived dyspnoea and heart rate ($p > 0.05$) (table 5.5). Dyspnoea was as high as ‘severe’, while the maximum heart rate was 115 beats.min$^{-1}$. However, repeated contrasts showed an interaction within Week 3 and Week 6 for instantaneous dyspnoea ($F(1,15) = 4.63, p = 0.05$, partial eta square = 0.24, power 0.52). A significant long-term effect of WBV was found on saturation of haemoglobin ($F(2,30) = 8.33, p = 0.01$). The effect occurred within Week 3 and Week 6 in which saturation of haemoglobin reduced from 96 to 95% ($p = 0.02$, partial eta square = 0.31, power 0.69). There was no instantaneous effect of WBV on saturation of haemoglobin ($F(2,30) = .91, p = 0.41$).
Table 5.5

Long-term and instantaneous effects of WBV and PLACEBO interventions on rating of perceived dyspnoea, heart rate, and saturation of haemoglobin of people with COPD

<table>
<thead>
<tr>
<th>Long-Term</th>
<th>Intervention</th>
<th>Dependent Variable</th>
<th>Week 1</th>
<th>Week 3</th>
<th>Week 6</th>
</tr>
</thead>
<tbody>
<tr>
<td>WBV</td>
<td>Borg CR-10</td>
<td></td>
<td>1 ± 1</td>
<td>2 ± 1</td>
<td>1 ± 1</td>
</tr>
<tr>
<td></td>
<td>HR (beats.min(^{-1}))</td>
<td></td>
<td>82 ± 10</td>
<td>86 ± 11</td>
<td>83 ± 11</td>
</tr>
<tr>
<td></td>
<td>SpO(_2) (%)</td>
<td></td>
<td>97 ± 2</td>
<td>96 ± 2</td>
<td>95 ± 2*</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Week 9</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Week 11</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Week 14</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PLACEBO</td>
<td>Borg CR-10</td>
<td></td>
<td>2 ± 1</td>
<td>2 ± 1</td>
<td>2 ± 1</td>
</tr>
<tr>
<td></td>
<td>HR (beats.min(^{-1}))</td>
<td></td>
<td>81 ± 13</td>
<td>83 ± 10</td>
<td>83 ± 11</td>
</tr>
<tr>
<td></td>
<td>SpO(_2) (%)</td>
<td></td>
<td>95 ± 2</td>
<td>96 ± 2</td>
<td>94 ± 2‡</td>
</tr>
<tr>
<td>Instantaneous</td>
<td></td>
<td>Week 1</td>
<td>2 ± 2</td>
<td>1 ± 1</td>
<td>2 ± 1*</td>
</tr>
<tr>
<td>WBV</td>
<td>Borg CR-10</td>
<td></td>
<td>2 ± 2</td>
<td>1 ± 1</td>
<td>2 ± 1*</td>
</tr>
<tr>
<td></td>
<td>HR (beats.min(^{-1}))</td>
<td></td>
<td>92 ± 10</td>
<td>94 ± 12</td>
<td>95 ± 9</td>
</tr>
<tr>
<td></td>
<td>HRR</td>
<td></td>
<td>67 ± 11</td>
<td>63 ± 13</td>
<td>66 ± 13</td>
</tr>
<tr>
<td></td>
<td>HRR%</td>
<td></td>
<td>16 ± 6</td>
<td>14 ± 8</td>
<td>18 ± 7</td>
</tr>
<tr>
<td></td>
<td>SpO(_2) (%)</td>
<td></td>
<td>96 ± 2</td>
<td>96 ± 2</td>
<td>96 ± 1</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Week 9</td>
<td></td>
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<td>Week 11</td>
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<td></td>
<td></td>
<td>Week 14</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>PLACEBO</td>
<td>Borg CR-10</td>
<td></td>
<td>2 ± 2</td>
<td>2 ± 1</td>
<td>2 ± 1</td>
</tr>
<tr>
<td></td>
<td>HR (beats.min(^{-1}))</td>
<td></td>
<td>88 ± 13</td>
<td>88 ± 13</td>
<td>90 ± 8</td>
</tr>
<tr>
<td></td>
<td>HRR</td>
<td></td>
<td>64 ± 16</td>
<td>64 ± 15</td>
<td>66 ± 16</td>
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<tr>
<td></td>
<td>HRR%</td>
<td></td>
<td>7 ± 9</td>
<td>7 ± 10</td>
<td>11 ± 7</td>
</tr>
<tr>
<td></td>
<td>SpO(_2) (%)</td>
<td></td>
<td>96 ± 2</td>
<td>96 ± 1</td>
<td>95 ± 2</td>
</tr>
</tbody>
</table>

Note: Data are mean ± SD. * significant difference within Week 3 to Week 6 (p ≤ 0.05). ‡ significant difference Week 11 to Week 14 (p ≤ 0.05). SpO\(_2\): saturation of haemoglobin. HR: heart rate. HRR: heart rate reserve with Karvonen method. HRR%: percentage of heart rate reserve.

There were no long-term, or instantaneous effects of PLACEBO on dyspnoea and heart rate (p > 0.05) (table 5.6). There was a significant long-term effect of PLACEBO on saturation of haemoglobin (F(2,20) = 6.02, p = 0.01). The effect occurred within Week 11 and Week 14 in which saturation of haemoglobin reduced from 96 to 94% (p = 0.01, partial eta square = 0.52, power 0.84). There was no instantaneous effect of PLACEBO on saturation of haemoglobin (F(2,20) = .79, p = 0.47).
There was no mean difference of long-term compared with instantaneous dyspnoea ($F(7,100) = 1.59, p = 0.15$) and saturation of haemoglobin ($F(7,100) = 1.20, p = 0.31$) across the 14 week efficacy trial. Neither the WBV intervention nor the PLACEBO intervention caused an immediate rise in dyspnoea (figure 5.2) or reduction in saturation of haemoglobin (figure 5.3).

Figure 5.2. Instantaneous effects WBV (blue) and PLACEBO (red) interventions on rating of perceived dyspnoea of people with COPD. Columns are mean data. Error bars are one standard deviation shown in the ‘positive’ direction.
Figure 5.3. Instantaneous effects WBV (blue) and PLACEBO (red) interventions on saturation of haemoglobin of people with COPD. Columns are mean data. Error bars are one standard deviation shown in the ‘positive’ direction.

A main effect for heart rate occurred among the long-term and instantaneous data (F(7,100) = 3.67, p = 0.01) (figure 5.4). The WBV intervention immediately increased heart rate at Week 1 (p = 0.01) and Week 6 (p = 0.01) compared with instantaneous data. The PLACEBO intervention did not increase heart rate. During the WBV intervention, heart rate mean difference was 10 beats.min\(^{-1}\) at Week 1, and 12 beats.min\(^{-1}\) at Week 6. Mean difference was 7 beats.min\(^{-1}\) across the PLACEBO intervention.
Figure 5.4. Instantaneous effects WBV (blue) and PLACEBO (red) interventions on heart rate of people with COPD. Columns are mean data. Error bars are one standard deviation shown in the ‘positive’ direction. * indicates $p \leq 0.05$. 

![Bar chart showing heart rate (beats.min$^{-1}$) over weeks 1 to 14 for WBV and PLACEBO interventions, with error bars indicating standard deviation.](chart.png)
Effects of WBV and PLACEBO Interventions on Functional Performance of the Lower Limbs of People with COPD

Effects of the independent variable ‘test occasion’ were computed with repeated measures ANOVA with repeated contrasts of long-term and acute results of each intervention on functional performance. Test occasion and intervention results are shown in tables 5.6 through 5.8. Data were collected to show a potential ‘training’ and/or ‘placebo’ effect of WBV. The long-term data were collected at rest across the two interventions. The acute data were collected within two minutes of a completed WBV or PLACEBO session. Effects of the independent variable ‘intervention’ on functional performance variables were analysed with a one-way ANOVA and least significant difference post-hoc comparisons were used to describe differences. Intervention results are shown in figures 5.5 though 5.9.

Long-term Effects on the TUG Test and 5-Chair Test

There was a main effect of WBV on the TUG test ($F(2,94) = 47.52, p = 0.01$) and the 5-chair test ($F(1.41,21.11) = 20.75, p = 0.01$) (table 5.6). Performance of the TUG test improved after the first six WBV sessions (Week 1 and Week 3 repeated contrast; $p = 0.01$, partial eta square $= 0.28$, power $= 0.99$), and second six WBV sessions (Week 3 and Week 6 repeated contrast; $p = 0.01$, partial eta square $= 0.41$, power $= 1.00$). Performance of the 5-chair test also improved after the first six WBV sessions (Week 1 and Week 3 repeated contrast; $p = 0.01$, partial eta square $= 0.50$, power $= 0.95$), and second six WBV sessions (Week 3 and Week 6 repeated contrast; $p = 0.01$ (partial eta square $= 0.49$, power $= 0.94$).
There was no effect of PLACEBO on performance of the TUG test ($F(2,64) = 1.11, p = 0.34$) and the 5-chair test ($F(1.29,12.88) = 0.15, p = 0.76$) (table 5.6). The mean difference within the intervention was not more than $± 0.2$ sec for both tests. Overall, TUG test time reduced by 1.5 seconds and 5-chair stands test time reduced by 3.4 seconds after the six week WBV intervention. Improvement of functional performance measured with simulated ADLs after WBV was maintained over the washout period and across the PLACEBO intervention.

Table 5.6

<table>
<thead>
<tr>
<th>Occasion</th>
<th>TUG (sec)</th>
<th>CHAIR (sec)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
</tr>
<tr>
<td>* WBV Week 1</td>
<td>11.3</td>
<td>1.9</td>
</tr>
<tr>
<td>Week 3+</td>
<td>10.7</td>
<td>1.7</td>
</tr>
<tr>
<td>Week 6+</td>
<td>9.8</td>
<td>1.9</td>
</tr>
<tr>
<td>PLACEBO Week 9</td>
<td>10.8</td>
<td>2.2</td>
</tr>
<tr>
<td>Week 11</td>
<td>10.7</td>
<td>2.0</td>
</tr>
<tr>
<td>Week 14</td>
<td>10.6</td>
<td>1.7</td>
</tr>
</tbody>
</table>

Note: n WBV = 16, n PLACEBO = 11. * significant main effect after repeated measures ANOVA computation ($p ≤ 0.05$). + significant difference after repeated contrast. 95% CI: confidence interval [$x ± 1.96(SD)$].

Figure 5.5 shows effects of both the WBV and PLACEBO interventions on TUG test time. Post-hoc comparisons revealed a reduction in performance after the six week WBV intervention (Week 6 and Week 9; mean increase in time = 1.0 seconds, $p = 0.02$). Performance of the 5-chair test was not different across the same time interval (Week 6 and Week 9; mean increase in time = 1.0 seconds, $p = 0.38$) (figure 5.6).
Figure 5.5. Effects of the washout period on functional performance of the TUG test. Week 6 results were long-term, Week 9 results were prior to the first PLACEBO session. Error bars are one standard deviation. * indicates $p \leq 0.05$ among Week 6 and Week 9 results.

Figure 5.6. Effects of the washout period on functional performance of the 5-chair test. Week 6 results were long-term, Week 9 results were prior to the first PLACEBO session. Error bars are one standard deviation.
Long-term Effects on Kinematics of Gait

There was a main effect of WBV on stride length ($F(1.48,244.07) = 50.21$, $p = 0.01$) (table 5.7). Stride length was longer within Week 1 and Week 3 results ($p = 0.01$, partial eta square $= 0.24$, power $= 1.00$), and Week 3 and Week 6 results ($p = 0.01$, partial eta square $= 0.36$, power $= 1.00$). There was no effect of PLACEBO on stride length ($F(1.79,141.76) = 1.46$, $p = 0.24$).

There was a main effect of WBV on stride time ($F(1.63,269.58) = 8.20$, $p = 0.01$) (table 5.7). Stride time was faster within Week 1 and Week 3 results ($p = 0.01$, partial eta square $= 0.14$, power $= 1.00$), and Week 3 and Week 6 results ($p = 0.01$, partial eta square $= 0.03$, power $= 0.70$). There was no main effect of PLACEBO on stride time ($F(1.78,140.41) = 2.32$, $p = 0.11$) although stride time was 0.1 sec faster within Week 9 and Week 11 results ($p = 0.02$, partial eta square $= 0.07$, power $= 0.65$).

There was a main effect of WBV on stride velocity ($F(1.35,223.37) = 323.17$, $p = 0.01$) (table 5.7). Stride velocity increased within Week 1 and Week 3 results ($p = 0.01$, partial eta square $= 0.72$, power $= 1.00$), and Week 3 and Week 6 results ($p = 0.01$, partial eta square $= 0.72$, power $= 1.00$). There was no main effect of PLACEBO on stride velocity ($F(2,158) = 1.20$, $p = 0.30$).
Table 5.7

Long-term effects of WBV and PLACEBO interventions on kinematic variables of gait of people with COPD

<table>
<thead>
<tr>
<th>Intervention</th>
<th>Occasion</th>
<th>Stride Length (m)</th>
<th>Stride Time (sec)</th>
<th>Stride Velocity (m.sec$^{-1}$)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
<td>95% CI</td>
<td>Mean</td>
</tr>
<tr>
<td>WBV *</td>
<td>1.14</td>
<td>0.16</td>
<td>±0.31</td>
<td>1.11</td>
</tr>
<tr>
<td></td>
<td>1.19</td>
<td>0.17</td>
<td>±0.33</td>
<td>1.09‡</td>
</tr>
<tr>
<td></td>
<td>1.27</td>
<td>0.11</td>
<td>±0.22</td>
<td>1.10*</td>
</tr>
<tr>
<td>PLACEBO</td>
<td>1.22</td>
<td>0.11</td>
<td>±0.22</td>
<td>1.03</td>
</tr>
<tr>
<td></td>
<td>1.20</td>
<td>0.13</td>
<td>±0.25</td>
<td>1.02*</td>
</tr>
<tr>
<td></td>
<td>1.22</td>
<td>0.10</td>
<td>±0.20</td>
<td>1.02</td>
</tr>
</tbody>
</table>

Note: WBV n = 166 datum per test occasion, per variable. PLACEBO n = 80 datum per test occasion, per variable. * significant main effect after repeated measures ANOVA ($p \leq 0.05$). ‡ significant interaction Week 1 to Week 3 ($p \leq 0.05$). * significant interaction Week 3 to Week 6 ($p \leq 0.05$). # significant interaction Week 9 to Week 11 ($p \leq 0.05$). 95% CI: confidence interval [x ± 1.96(SD)].

Acute Effects on Kinematics of Gait

There was a main effect of WBV on stride length ($F(1.45,248.97) = 143.34, p = 0.01$) (table 5.8). Stride length was longer within Week 1 and Week 3 results ($p = 0.01$, partial eta square = 0.25, power = 1.00), and Week 3 and Week 6 results ($p = 0.01$, partial eta square = 0.34, power = 1.00). There was a main effect of PLACEBO on stride length ($F(2,150) = 4.48, p = 0.01$). Stride length was shorter within Week 9 and Week 11 results ($p = 0.01$, partial eta square = 0.10, power = 0.83), and longer within Week 11 and Week 14 results ($p = 0.03$, partial eta square = 0.34, power = 0.60).
There was a main effect of WBV on stride time (F(1.60,275.30) = 7.72, p = 0.01) (table 5.8). Stride time was faster within Week 1 and Week 3 results (p = 0.01, partial eta square = 0.14, power = 1.00). There was a main effect of PLACEBO on stride time (F(1.75,148.11) = 4.66, p = 0.02). Mean difference was a 0.20 sec reduction in time within the test occasions even though there were no differences in the contrast analyses (i.e., p > 0.05).

There was a main effect of WBV on stride velocity (F(1.86,319.23) = 159.73, p = 0.01) (table 5.8). Stride velocity increased within Week 1 and Week 3 results (p = 0.01, partial eta square = 0.29, power = 1.00), and Week 3 and Week 6 results (p = 0.01, partial eta square = 0.36, power = 1.00). There was no effect of PLACEBO and test occasions for stride velocity (F(1.78,133.67) = 1.84, p = 0.17).

Table 5.8

Acute effects of WBV and PLACEBO interventions on kinematic variables of gait of people with COPD

<table>
<thead>
<tr>
<th>Intervention</th>
<th>Occasion</th>
<th>Stride Length (m)</th>
<th>Stride Time (sec)</th>
<th>Stride Velocity (m.sec(^{-1}))</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Mean</td>
<td>SD</td>
<td>95% CI</td>
</tr>
<tr>
<td>WBV *</td>
<td>Week 1</td>
<td>1.12</td>
<td>0.17</td>
<td>±0.33</td>
</tr>
<tr>
<td></td>
<td>Week 3 ‡</td>
<td>1.17‡</td>
<td>0.17</td>
<td>±0.33</td>
</tr>
<tr>
<td></td>
<td>Week 6 +</td>
<td>1.27‡</td>
<td>0.12</td>
<td>±0.24</td>
</tr>
<tr>
<td>PLACEBO *</td>
<td>Week 9</td>
<td>1.24</td>
<td>0.13</td>
<td>±0.25</td>
</tr>
<tr>
<td></td>
<td>Week 11 #</td>
<td>1.20#</td>
<td>0.15</td>
<td>±0.29</td>
</tr>
<tr>
<td></td>
<td>Week 14 $</td>
<td>1.23$</td>
<td>0.13</td>
<td>±0.25</td>
</tr>
</tbody>
</table>

Note: WBV n = 173 datum per test occasion, per variable. PLACEBO n = 80 datum per test occasion, per variable. * significant main effect after repeated measures ANOVA (p ≤ 0.05). ‡ significant interaction Week 1 to Week 3 (p ≤ 0.05). † significant interaction Week 3 to Week 6 (p ≤ 0.05). # significant interaction Week 9 to Week 11 (p ≤ 0.05). $ significant interaction Week 11 to Week 14 (p ≤ 0.05). 95% CI: confidence interval [x ± 1.96(SD)].
There were main effects for long-term compared with acute stride length ($F(11,1808) = 15.40, p = 0.01$), stride time ($F(11,1808) = 9.13, p = 0.01$), and stride velocity ($F(11,1808) = 30.92, p = 0.01$) across all test occasions of the 14 week efficacy trial. However, interaction among each test occasion of the 14 week efficacy trial was of specific interest rather than interaction across all weeks. As such, there were no interactions among all test occasions (i.e., long-term compared with acute results for: Weeks 1, 3, 6, 9, 12, and 14 $p > 0.05$) for stride length (figure 5.7) and stride velocity (figure 5.9). An interaction occurred among Week 6 stride times ($p = 0.04$) (figure 5.8). As such, stride time was faster after the WBV session in Week 6. After the WASHOUT period, long-term and acute gait performance was unaffected ($p > 0.05$).

![Figure 5.7](image.png)

**Figure 5.7.** Effects WBV and PLACEBO interventions on stride length of people with COPD. Results were both long-term (blue) and acute (red). Results are mean stride length. Error bars are one standard deviation.
Figure 5.8. Effects WBV and PLACEBO interventions on stride time of people with COPD. Results were both long-term (blue) and acute (red). Results are mean stride time. Error bars are one standard deviation. * indicates $p \leq 0.05$ among Week 6 long-term and acute stride times.
Figure 5.9. Effects WBV and PLACEBO interventions on stride velocity of people with COPD. Results were both long-term (blue) and acute (red). Results are mean stride velocity. Error bars are one standard deviation.

5.4 Discussion

Major Findings

In this second major study, efficacy of a WBV intervention on exercise tolerance and functional performance of the lower limbs was explored by conducting a non-randomised placebo crossover trial of people with COPD. Selected dependent variables specific to exercise tolerance (rating of perceived breathlessness, heart rate and, saturation of haemoglobin) and functional performance (simulated ADLs and walking gait) were quantified over a 14 week research period. Prior to this 14 week research, pilot studies for validity and reliability of instruments and test procedures were established before the first major study of
safety of WBV for people with COPD was conducted. After the acute safety of WBV as a mode of exercise for people with COPD was confirmed, four major findings of this research emerged to support efficacy of WBV: (1) the WBV intervention failed to negatively affect exercise tolerance, (2) the WBV intervention did not cause exacerbations of COPD that can limit participation in physical activity modes, (3) the WBV intervention improved functional performance of the lower limbs, (4) the effects of WBV on functional performance of the lower limbs were at least maintained eight weeks after the WBV intervention, and (5) the effects of a WBV intervention on exercise tolerance and functional performance of the lower limbs were not susceptible to a placebo effect.

**Exercise Tolerance**

In this research there were no long-term or instantaneous effects of WBV on exercise tolerance. Exercise tolerance was maintained over the duration of this research. The dependent variables of exercise tolerance quantified in the current research can be viewed in two perspectives: (1) exercise tolerance can describe the potential ‘fitness training’ effect of WBV to improve aerobic performance commonly associated with ‘exercise’, and (2) exercise tolerance can describe exacerbations of COPD over the duration of a WBV intervention. In the first perspective, there was no ‘fitness training’ effect of a six week WBV vibration intervention on: (1) rating of perceived dyspnoea, (2) heart rate, and (3) saturation of haemoglobin. Although significant poorer results of saturation of haemoglobin (table 5.6: Week 3 to 6 WBV: 1% reduction, Week 11 to 14 PLACEBO: 2% reduction) and rating of perceived dyspnoea were found (table 5.6: Week 3 to 6 WBV: ‘very slight’ to ‘slight’), a lack of clinical importance persists because the results of exercise tolerance were: (1) expected after major study 1, and (2) considered safe after comparison with another sample of people with Stage II COPD during exercise (i.e., Pickard, Yang & Lee, 2011). In the second
perspective, the results of this research are similar to other beneficial peripheral muscle training interventions among people with COPD.

Fitness – Long-Term Compared with Instantaneous Results

The negligible changes in exercise tolerance in this research do not follow the trend of published literature for improvement in exercise tolerance (i.e., fitness training) among people with COPD after various methods of aerobic conditioning, and for healthy older adults after WBV. The mechanisms of improvement in exercise tolerance for humans (after WBV or indeed any mode of exercise training of COPD patients) can be demonstrated by increased oxygen uptake and VO$_{2\text{peak}}$ (Rittweger, Schiessl & Felsenberg, 2001), increased peripheral blood flow (Kerschan-Schindl, et al., 2001), hypertrophy of skeletal muscle of the lower limbs, and improved oxidative capacity within skeletal muscle fibres (Maltais, LeBlanc, Whitton, Simard, Marquis, Belanger et al., 2000; Haykowski, McGavock, Vonder Muhll, Koller, Mandic, Welsh et al., 2005).

The aforementioned variables of exercise tolerance were not quantified due to field-based testing. Such quantification is suited to a controlled laboratory setting rather than a community setting. Although WBV increased blood flow about the lower limbs (Kerschan-Schindl et al., 2001; Yamada, Kusaka, Miyamoto, Tanaka, Morita, Tanaka, et al., 2005; Lohman III, Petrofsky, Maloney-Hinds, Betts-Schwab & Thorpe, 2007; Lythgo et al., 2009), it is plausible that enhanced oxygen transport and uptake at the skeletal muscle could occur during WBV. Although for participants of this research, the WBV sessions increased heart rate, there was no fitness training effect because resting heart rate did not change. Given the known positive correlation of heart rate and oxygen uptake, and improvement of cardiac
output after fitness training (the product of stroke volume and heart rate), WBV may have increased oxygen uptake for people with COPD. However, the effect was not shown with instantaneous rating of perceived dyspnoea and saturation of haemoglobin results or resting heart rate results.

Efficacy of WBV to improve fitness of healthy older adults was recently established over a 12 month period (Bogaerts et al., 2009). During WBV, participants performed dynamic movement that allowed an increase of HRR to as much as 85%, which was within the aerobic fitness training zone (ACSM, 2005). The HRR of participants in the current research was 18% (± 7%), where 40% was the minimum threshold for aerobic conditioning improvement (ACSM, 2005). The negligible changes in exercise tolerance shown in the results in this research therefore, may be explained by an absence of a suitable ‘stimulus’ to enhance fitness.

Eliciting a suitable stimulus during exercise interventions for people with COPD can be compromised due to a reduced ability to deliver oxygen to peripheral skeletal muscle because of gas exchange inefficiency, flow limitation, and concurrent activation of large muscle groups (Troosters, Casaburi, Gosselink, & Decramer, 2005). Given that people with COPD present a reduced anaerobic threshold (Maltais, LeBlanc, Simard, Jobin, Bérubé, Bruneau et al, 1996) and an increased muscular fatigability represented by a trend towards Type II muscle fibre distribution (Gosker et al., 2000; Gosker, van Mameren, van Dijk, Engelen, van der Vusse, Wouters et al., 2002; Allaire, Maltais, Doyon, Noel, LeBlanc, Carrier et al., 2004), whole body exercises common with aerobic conditioning and resistance training increase ventilatory demand (Troosters et al., 2005). The net benefit of increased ventilatory stress can be modified reparatory techniques (Gigliotti, Coli, Bianchi, Romagnoli, Lanini, Binazzi et al., 2003) and reduced dynamic hyperinflation (Porszasz, Emtner, Whipp, Goto, Sonfay &
Casaburi, 2003), however compliance to intervention protocols remains problematic (Troosters et al., 2005). The results of this research show that ventilatory demand was not beyond normal daily variation for people with COPD (i.e., perceived breathlessness) and despite the absence of improvement in ‘fitness’ of the participants in this research, WBV did not reduce exercise tolerance of people with COPD.

The ongoing challenge of peripheral muscle training therefore, remains to design exercise interventions that stimulate the cardiovascular and musculoskeletal systems to allow physiological adaptations in skeletal muscle to improve exercise tolerance by avoiding exacerbations and reducing skeletal muscle dysfunction (Troosters et al., 2005). Skeletal muscle dysfunction is synonymous with the dyspnoea spiral, and can also be quantified by poor aerobic performance due to reduced muscular strength, endurance, and oxidative capacity (Maltais et al., 2000). However, the exact mechanisms of skeletal muscle dysfunction among people with COPD remain individualised and largely unknown (Troosters et al., 2005). Results of this research do not support efficacy of WBV to improve the generic notion of exercise tolerance (i.e., fitness training) among people with COPD. The possibility of simultaneously addressing fitness and musculoskeletal dysfunction in patients with COPD (as evidenced by improvements when healthy older adults added resistance training exercises to WBV) remains a challenge for the future.

**Exacerbations – Long-Term Compared with Instantaneous Results**

Results of this research suggest that WBV can be a mode of exercise for people with COPD that will not exacerbate the core limitations of the disease because a meaningful change in dyspnoea and saturation of haemoglobin did not occur across the WBV intervention. Results
of this research compare favourably to other investigations of resistance training and aerobic conditioning of people with COPD. After 12 weeks of both training modes, Borg CR-10 values were not different within and among resistance training and aerobic conditioning for people with COPD (Skumlien et al., 2008). However, during performance of the six-minute walk test, perceived dyspnoea increased to a value of ‘6’ (Skumlien et al., 2008). Concurrently, saturation of haemoglobin reduced from 95 to 87% (Skumlien et al., 2008) and has reduced to similar values across other exercise investigations of people with COPD (Poulain et al., 2003; O’Driscoll et al., 2011).

The use of WBV for people with COPD can be supported with quantification of saturation of haemoglobin to allow speculative description of hypoxemia. A method to define hypoxemia was to observe a > 4% drop in saturation of haemoglobin to resting levels (Poulain et al., 2003). Others have defined ‘severe’ hypoxemia at 90% saturation of haemoglobin (Stoller, Panos, Krachman, Doherty, Make & the Long-term Oxygen Treatment Trial Research Group, 2010; Liu, Chisholm, Ngeow, John, Shaw, Ma et al., 2011). Although not to the same values, the results of this research further support use of WBV as a mode of physical activity for people with COPD. Results of major study 1 supported the safety of a single session of WBV. Results of this research support the efficacy of a long-term WBV intervention to avoid exacerbations of COPD.

As a mode of exercise, WBV reduced muscle oxygenation (Yamada et al., 2005) and increased cortisol and circulating levels of insulin-like growth factor I (Cardinale, Soiza, Leiper, Gibson & Primrose, 2007) of healthy participants, while for people with mild to moderate COPD, long-term physical activity was shown to improve oxygen saturation (Barbera, Roca, Ramirez, Wagner, Ussetti & Rodriguez-Roisin, 1991). The markers of stress,
or physical exertion associated with WBV may be transferred to people with COPD so that if the lower limbs are shown to increase metabolic activity, physiological responses, such as increased breathlessness or reduced saturation of haemoglobin may be expected during WBV. For participants of this research however, there was an absence meaningful change of dyspnoea and saturation of haemoglobin across the WBV intervention. As such, if metabolic activity of people with COPD increased due to WBV, the change was not represented with the Borg CR-10 VAS and SpO2. Metabolic activity however, was shown to increase due to results of heart rate in the current research. During the 14 week research, heart rate increased more during WBV ($p \leq 0.05$, mean heart rate increase $\geq 10$ beats.min$^{-1}$) than during PLACBO ($p > 0.05$, mean heart rate increase = 7 beats.min$^{-1}$). Others have reported similar increases in heart rate during WBV for sub-optimal health (Crevenna et al., 2003) and healthy older adults (Bogaerts et al., 2009).

A consideration from major study 1 was further study to describe the independent effects of standing and WBV on heart rate. The current results indicated WBV increased metabolic activity, as demonstrated by heart rate, of people with COPD whereas PLACEBO did not. As such, the limitation of Chapter 4, that heart rate was quantified in a sitting position and compared to an anatomical position, can also be discounted. For humans, regardless of health status, exercise increases heart rate. This research has shown that a WBV bout increases heart rate for people with COPD. Those results were previously unknown for people with COPD. As such, and given the known and broad physiological responses of the human body to exercise, WBV may be viewed as an effective mode of exercise to increase metabolic activity of people with COPD.
Conversely, the WBV intervention of this research did not increase HRR as had been shown in a previous WBV intervention of healthy older adults (Bogaerts et al., 2009). However, because COPD is a progressive disease, with the progression to poor exercise tolerance, improvements in HRR or other physiological responses to exercise should be secondary to maintenance of current levels. Whole-body vibration was a well tolerated exercise intervention for people with COPD highlighted by the absence of exacerbations of COPD. Fluctuations (i.e., standard deviation) of results of this research can be partly explained by normal day-to-day variation and should be expected concurrently with WBV effects and people with COPD rather than caused by WBV.

The exercise tolerance results of this research show that participants had more trouble completing the 5-chair test than the WBV and PLACEBO interventions. There was no main effect of the test protocol on Borg CR-10 VAS results (page 163), yet, rating of perceived breathlessness was as high 6 (between ‘severe’ and ‘very severe’) immediately after the 5-chair test. Given the test mimics ADLs, as a mode of exercise, WBV could be added to the ‘normal’ daily routine of people with moderate Stage II COPD without causing dyspnoea commonly associated with other modes of physical activity common place among pulmonary rehabilitation interventions.
Functional Performance of the Lower Limbs

Simulated ADLs

The results of this research show the beneficial effect of WBV on functional performance of people with COPD. The results are supported by other WBV interventions of older adults and aerobic and resistance training interventions of people with COPD. The improvements in performance of the TUG test (13%) and 5-chair test (18%) after WBV for people with COPD in this research were greater than the respective 6% and 12% improvements reported after a two month WBV intervention of healthy older adults (Rees, Murphy & Watsford, 2007). Given the community dwelling sub-optimal health status of participants in this research, it is logical that the potential for improvement in performance of ADLs in people with COPD after WBV would be larger than healthy older adults when considering data of non-community dwelling older adults who’s TUG test time improved 48% after six weeks of WBV (i.e., Bruyere et al., 2005).

A three week resistance training combined with WBV intervention reduced time taken to complete the 5-chair test by 4.0 sec (± 4.8 sec) (Gloeckl et al., 2012). Results of this research show a 3.4 sec (± 2.8 sec) reduction of performance of the 5-chair test after a six week standalone WBV intervention (table 5.6). The results of combined resistance training and WBV however, should be cautiously appraised because the standard deviation is larger than the mean and reliability of the test procedure is not reported. Reliability of the 5-chair test procedure in this research was categorised as ‘good’ (ICC = 0.881, table 3.9, page 120).
For people with COPD, chair stands in 60 seconds and stair climbing have improved after resistance training and aerobic conditioning (Kongsgaard et al., 2004; Alexander, Phillips & Wagner, 2008; Arnardóttir et al., 2011). Compared with resistance training, WBV is not as effective for people with COPD because improvement of TUG test performance was 67% after 12 weeks of resistance training (Panton et al., 2004) compared with 13% in this research. A resistance training intervention in this population however, would require facilities not usually available in the home, and specialised supervision. As such the efficacy of sustainable and accessible exercise prescription in populations with COPD requires careful consideration.

Gait

Participants in this research walked with longer strides, in a faster time after the WBV intervention. Similarly, eight weeks of combined resistance training and WBV in healthy older adults lengthened step length from 61 cm to 65 cm (7%) (Kawanabe, Kawashima, Sashimoto, Takedu, Sato & Iwamoto, 2007). Stride length of participants in this research was 13 cm longer (11%), which is similar to the ≥ 14 cm improvement of healthy older adults after resistance training interventions (Persch, Uginowitsch, Pereira & Rodacki, 2009; Fahlman et al., 2011). Similar effects were reported for gait velocity after resistance training and aerobic conditioning for people with COPD. Specifically, velocity increased 13%, from 0.89 m.sec\(^{-1}\) to 1.01 m.sec\(^{-1}\) (Alexander, Phillips & Wagner, 2008). The 12% increase of stride velocity for people with COPD in this research is similar to previous results in the range of 14 to 30% (Berry et al., 1999; Kongsgaard et al., 2004; Panton et al., 2004) for people with COPD. For healthy older adults, a 12% improvement in gait velocity after resistance training was reported as an “attractive strategy to improve gait” (Persch et al., 2009, p 824). When
compared with this research, WBV may also be an attractive strategy to improve gait for people with COPD.

**Possible Mechanisms of Improvement of Functional Performance**

The possible mechanisms for improvement in functional performance during and after WBV were reviewed in Chapter 2. Specifically, improvement has been postulated to be linked to stimulation of various mechanisms including the stretch reflex (Nørlund & Thorstensson, 2007), the endocrine system (Rehn et al., 2007), and other components of the autonomic nervous system (Kerschan-Schindl et al., 2001; Torvinen et al., 2002; Lythgo et al., 2009; Mileva, Botwell & Kossev, 2009). Such mechanisms could be attributed to increased perceived exertion and blood lactate during WBV (Rittweger, Mutschelknauss & Felsenberg, 2003), and a similar explanation may be made for the increase in heart rate of participants with COPD in this research during WBV.

Given the known physiological benefits of aerobic conditioning and resistance training on people with COPD (e.g., skeletal muscle hypertrophy and increased oxidative capacity), it may be appropriate to view possible mechanisms of improvement in functional performance of people with COPD after this research with a similar perspective. However, given the predominance of Type II muscle fibre activity during exercise for people with COPD leading to increase anaerobic fatigue (Allaire et al., 2004), it may be possible that improvement of functional performance of people with COPD after WBV could be partly attributed to training of Type II skeletal muscle fibres rather than for example, an increase of oxidative capacity at the muscle.
Given that Type II muscle fibres are predominantly anaerobic and the simulated ADLs of this research were largely based about anaerobic energy supply (i.e., the phosphogen system and anaerobic glycolysis), improvement in performance may be due to an increase in anaerobic power, or an anaerobic training effect of WBV. This point may be supported by linking the exercise tolerance results of this research showing that the WBV ‘stimulus’ was too low to allow improvement in aerobic fitness, and the improvements in performance of ADLs after WBV reported by others (e.g., Bruyere et al., 2005; Rees, Murphy & Watsford, 2007). Given the known leg fatigae exhibited by people with COPD due to high-intensity exercise designed to improve anaerobic performance, and persistence of poor exercise compliance associated with symptom limited participation (Troosters et al., 2005; Kortianou, Nasis, Spetsioti, Daskalakis & Vogiatzis, 2010), WBV may be a more attractive mode of anaerobic exercise for people with COPD when comparing results across interventions.

If then, WBV has the capacity to improve anaerobic power of the skeletal muscles of the lower limbs of people with COPD, a purely physiological perspective may only partly explain the possible mechanisms leading to improvement in functional performance of the lower limbs. The gait of participants in this research for example, may have been enhanced due to increased motoneuron excitability observable with the H-reflex at the triceps surae. Given that WBV effected the H-reflex (Armstrong et al., 2008), the known role of the H-reflex in motoneuron excitability (Hamada et al., 2000; Palmieri, Hoffman & Ingersoll, 2002; Hoffman, Palmieri & Ingersoll, 2003), and the role of the triceps surae during gait, motoneuron enhancement may have occurred during WBV for people with COPD. It may be that WBV elicited either strength and power improvement, or improvement in motor unit recruitment as had been reported for healthy older adults (Mileva, Botwell & Kossev, 2009).
Improvement in functional performance of the lower limbs may also be partly attributed to the stretch reflex. After WBV to exhaustion of healthy adults, patella tendon reflex amplitudes were similar to, and sometimes exceeded baseline compared with a reduction in amplitudes among a control group (Rittweger, Mutschelknauss & Felsenberg, 2003). The results occurred independent of similar declines in motor performance in both groups (Rittweger, Mutschelknauss & Felsenberg, 2003). The authors attributed the results most likely to an enhanced central motor excitability resulting in patella tendon reflex activation (i.e., the stretch reflex). Given that resistance training interventions initially improve motoneuron performance before structural morphology occurs (Sale, 1988), it may be that the current WBV intervention improved neural activity to allow strength and power improvement of the lower limbs of people with COPD. Regardless of the neural mechanisms, functional performance of simulated ADLs of people with COPD was positively affected by the current WBV intervention. Specifically, functional measures of muscular strength and muscular power were improved after a six week WBV intervention. Essentially, participants were able to rise from a chair and perform other simulated daily activities with assumed synergies of greater muscular strength and muscular power.

It was beyond the scope of this research to quantify cellular components of muscle, fibre Type distribution, or more direct metabolic responses, yet long-term improvement of field tests designed to indirectly quantify muscular strength and muscular power were comparable to other modes of training such as resistance training and aerobic conditioning among people with COPD and healthy older adults. However, invasive laboratory procedures required for more mechanistic explanations are unlikely to be obtained from populations in sub-optimal health such as people with COPD.
Maintenance of Functional Performance of the Lower Limbs and a Placebo Effect

Results of this research show the maintenance of functional performance following a WBV intervention for people with COPD (i.e., figures 5.5 to 5.9). The trend of simulated ADLs results was an improvement in performance after WBV and then maintenance of the improved performance during the PLACEBO intervention. It may be that the participants reached a ‘ceiling’ that limited further improvement during the PLACEBO intervention and therefore, a placebo effect may have been systemic to the WBV intervention. However, performance of the TUG test was worse \( (p \leq 0.05) \) after the two week washout period, and did not improve over the duration of the PLACEBO intervention. It may also be that: (1) simply standing on the placebo vibration platform with the knees flexed was enough stimulus to maintain the improvement, or (2) participants were more physically active after the WBV intervention despite the requirement not to begin any new mode of physical activity during the 14 week research period.

The kinematic gait results of this research show that compared with a PLACEBO intervention, WBV improved functional performance. Although there were differences among PLACEBO and WBV results, the differences were not meaningful (i.e., \( p > 0.05 \)). After WBV for example, stride length was 1.27 m compared with 1.22 m after PLACEBO, while stride velocity was 1.16 m.sec\(^{-1}\) compared with 1.20 m.sec\(^{-1}\) (table 5.9). The observed improvement in stride velocity of the PLACEBO results may be explained by the difference in stride time among the interventions, because stride velocity is the product of stride time and stride length. The differences may also be due to a placebo effect.
Although to test for a placebo effect during WBV is not common among the WBV literature base, the evidence of a placebo effect is equivocal. The effect has been discounted when, for example, measuring TUG test performance of people with multiple sclerosis (Schuhfried Mittermaier, Jovanovic, Pieber, & Paternostro-Sluga, 2005). Equivocation arises because WBV was often added to resistance training or some other mode of training and compared with a placebo condition with the same mode of training (e.g., Artero, Espada-Fuentes, Argüelles-Cienfuegos, Román, Gómez-López, & Gutiérrez, 2012). Furthermore, highlighted by inconsistent WBV protocols (e.g., frequency and peak-to-peak displacement) a clear consensus of WBV and a potential placebo effect has not been confirmed. Results of this research show that at Week 14, eight weeks after the WBV intervention was completed, functional performance was better compared with baseline data, though the meaningful improvement occurred across the WBV intervention and not across the PLACEBO intervention.

**Compliance and Drop-Out**

All participants successfully completed all WBV and PLACEBO sessions however, the number of participants reduced over the 14 week duration of this research (figure 5.1). The drop-out of participants was due to circumstances beyond the demands of the research during the washout period. As the importance of compliance to pulmonary rehabilitation had been documented and problematic (e.g., Troosters et al., 2005) it was meaningful that participants complied 100% of the WBV intervention. As a mode of physical activity to maintain exercise tolerance, avoid exacerbations of COPD, and improve functional performance of the lower limbs, efficacy of WBV can be confirmed.
Compared with other outpatient based interventions, the compliance and drop-out rate in this research was unique. The drop-out rate of three month resistance training research projects ranged from 20% to 38% (Suzuki, Kim, Yoshida & Ishizaki, 2004; Means, Rodell & O’Sullivan, 2005). Compliance, in some instances among healthy adults was at best 79% (Brill et al., 1999) and as low as 75% (Suzuki et al., 2004) for resistance training interventions. Symptom limitation was a major reason for poor compliance among people with COPD during pulmonary rehabilitation (Troosters et al., 2005). Given that only 10% of older American adults regularly engage in resistance training (Seguin & Nelson, 2003), and only 48% of older Australians are physically active (ABS 4156.0.55.001, 2011), the requirement of exercise that does not exacerbate people living with sub-optimal health is salient. The results of the home-based WBV intervention used in this research of people with COPD confirm the ease and convenience of WBV highlighted by high compliance and an absence of drop-out during the WBV intervention.

**Anecdotal Responses and Future Directions**

Anecdotal responses, though biased and poorly controlled, can lead to additional research questions and outline the need for a placebo condition in WBV research. The following occurred during the WBV and PLACEBO interventions:

- Two participants lost body mass. One participant lost 10 kg over six weeks (~8% body mass) and was sure that was due to WBV because it was the only difference in his life. His weight did not change over the PLACEBO intervention. One female lost 2 kg (~2% body mass) after six weeks of WBV. She was adamant that she had tried “everything” over time and that WBV was the cause.

- One participant was sure the PLACEBO intervention was better than the WBV intervention, and he felt much stronger and “better” after PLACEBO.
Most participants, at the beginning of the study, felt leg fatigue due to the demand of standing on the vibration platform with the isometric squat. That fatigue was negligible after 14 weeks. Several participants also felt the need to rest for up to 60 minutes after a session, regardless of intervention.

Transmission of WBV about the knee and other landmarks of the body for people with COPD remains unknown. Metabolic activity such as oxygen uptake and responses of the musculoskeletal system to WBV such as EMG activity, and circulating blood lactate, cortisol and creatine kinase also remains unknown for people with COPD. More invasive experimental protocols however, are more suited to a controlled laboratory environment rather than a community setting used in this research. The combined effect of a pulmonary rehabilitation program and a WBV intervention requires further investigation because the results of this research support the contention that WBV may be a “promising new exercise mode” for people with COPD (Gloeckl et al., 2012, p 76).

Whole-body vibration intensity (i.e., g force) to elicit improvement of a component of exercise tolerance such as aerobic fitness should be investigated for people with COPD. The gravitational force of the WBV during this research (~2.52 g) did not provide a suitable stimulus to enhance aerobic fitness. Despite the need for aerobic fitness and the safe use of low (i.e., Furness & Maschette, 2009: 1.26 g) and high (i.e., Cardinale & Lim, 2003: 50.33 g) WBV intensity, as a mode of exercise, WBV should be prescribed mindful that older adults are susceptible to vertebral fracture with high intensity WBV (Kiiski et al., 2008).
5.5 Summary

This was the first study to quantify and describe effects of a WBV intervention on exercise tolerance and functional performance of the lower limbs of people with COPD in a community setting. Furthermore, a potential placebo effect of WBV was quantified with a PLACEBO intervention. Both the WBV and PLACEBO interventions occurred in the home of each participant, which maximised participant compliance to each intervention. Results of this research confirmed efficacy of WBV to improve functional performance of the lower limbs of people with COPD. Exercise tolerance was not negatively effected by WBV, nor did the intervention exacerbate the stable COPD of the participants.

Changes in the dependent variables of exercise tolerance were negligible or clinically meaningless across the duration of the WBV intervention (table 5.9). Improvement in performance of simulated ADLs and walking gait occurred associated with large effect sizes across the duration of the WBV intervention. The improvement in functional performance was at least maintained over the PLACEBO intervention. Interpretation of the results may suggest that if a WBV intervention is susceptible to a placebo effect, it was not evident after the non-randomised cross-over design of this research.
Table 5.9

Summary of research hypotheses and decision after inferential statistical analyses

<table>
<thead>
<tr>
<th>Research Hypothesis</th>
<th>Accept</th>
<th>Reject</th>
<th>Data</th>
<th>Interpretation</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>EXERCISE TOLERANCE</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>WBV will instantaneously increase dyspnoea</td>
<td>✓</td>
<td></td>
<td>Increased from ‘very slight’ to ‘slight’</td>
<td>Negligible clinical impact for people with COPD</td>
</tr>
<tr>
<td>WBV will not change long-term dyspnoea, heart rate or SpO₂</td>
<td>✓</td>
<td></td>
<td>Reduction of SpO₂ from 96 to 95%</td>
<td>Negligible clinical impact for people with COPD</td>
</tr>
<tr>
<td>WBV will instantaneously increase heart rate</td>
<td>✓</td>
<td></td>
<td>Significant increase</td>
<td>WBV is a mode of exercise to increase metabolic activity</td>
</tr>
<tr>
<td>WBV will instantaneously reduce SpO₂</td>
<td>✓</td>
<td></td>
<td>No difference</td>
<td>WBV did not elicit hypoxemia</td>
</tr>
<tr>
<td>WBV will be different to PLACEBO</td>
<td>✓</td>
<td></td>
<td>No difference</td>
<td>WBV has no effect on exercise tolerance, but does not exacerbate stable COPD</td>
</tr>
<tr>
<td><strong>FUNCTIONAL PERFORMANCE</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>WBV will improve performance of the TUG test</td>
<td>✓</td>
<td></td>
<td>13% improvement</td>
<td>WBV improved strength and power</td>
</tr>
<tr>
<td>WBV will improve performance of the 5-chair test</td>
<td>✓</td>
<td></td>
<td>18% improvement</td>
<td>WBV improved strength and power</td>
</tr>
<tr>
<td>WBV will improve selected kinematic variables of gait</td>
<td>✓</td>
<td></td>
<td>Significant improvement</td>
<td>WBV improved strength and power</td>
</tr>
<tr>
<td>WBV will be different to PLACEBO</td>
<td>✓</td>
<td></td>
<td>Functional performance improvement after WBV</td>
<td>PLACEBO did not improve nor negatively effect functional performance</td>
</tr>
</tbody>
</table>

As components of pulmonary rehabilitation, efficacy of resistance training and aerobic conditioning has been confirmed (GOLD, 2011). Results of this research support efficacy of WBV to improve functional performance of the lower limbs of people with stable COPD in a community setting, while avoiding exacerbations of the disease that can limit compliance to pulmonary rehabilitation interventions.
CHAPTER 6. CONCLUSION

This was the first research to advance knowledge of effects of WBV on exercise tolerance and functional performance of the lower limbs of people with COPD in a community setting. Safety and efficacy of a WBV intervention to maintain exercise tolerance and avoid exacerbations of COPD that add to physical inactivity, and improve performance of ADLs of people with COPD was determined.

The specific aims of this research were met by: (1) establishing validity of a WBV platform, (2) determining safety of a single session of WBV for people with COPD by quantifying rating of perceived dyspnoea and selected acute physiological responses to exercise, (3) establishing reliability of the test procedure of major study 2, and (4) determining efficacy of a six week WBV intervention on rating of perceived dyspnoea, selected physiological responses to exercise, and functional performance of the lower limbs of people with COPD compared with a six week PLACEBO intervention. All of the aims were met with exception of the aim to describe transmission of WBV about the knee of people with COPD in a community setting. This aim was not met mainly because the robustness of the laboratory could not be replicated with the limited resources of the community setting available at the time of data collection and the overall arduousness of the test protocol for people with COPD. Nevertheless, the results from this research determine sufficient evidence of efficacy to proceed to higher levels of evidence in both rehabilitative and disease management programs for community dwelling adults with COPD.
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## LIST OF APPENDICES

| Appendix A | Concurrent validity of an accelerometer |
| Appendix B | Validation of vibration platform frequency |
| Appendix C | Transmission of WBV of healthy young adults |
| Appendix D | ACU HREC approval of transmission of young adults |
| Appendix E | ACU HREC approval of transmission of COPD |
| Appendix F | Southern Health information letter for WBV and COPD |
| Appendix G | ACU HREC approval of major study and reliability |
| Appendix H | Southern Health HREC approval of WBV and COPD |
| Appendix I | Tests of cognition and visual acuity |
| Appendix J | The Borg CR-10 VAS |
| Appendix K | ACU information letter/ advertising for safety of WBV |
| Appendix L | ACU HREC approval of WBV and COPD safety |
| Appendix M | The challenge of participant recruitment |
| Appendix N | The placebo vibration platform |
| Appendix O | Raw data of major study 2 |
CONCURRENT VALIDITY OF AN ACCELEROMETER TO QUANTIFY VIBRATION PLATFORM FREQUENCY

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INTRODUCTION

Vibrational platforms are used to deliver whole-body vibration to an individual. Depending on the specifications of the platform, frequency (Hz) may be manipulated to affect intensity of whole-body vibration.

Frequency should be accurately reported in literature to enable replication in the laboratory and field. Such information is under researched. Measurement of frequency can occur in the laboratory with VICON, but portable, light weight and inexpensive measurement instruments are desired in the field. The purpose of this study, therefore, was to cross-examine validity of a portable accelerometer with the VICON motion analysis system.

METHODS

Two commercial vibration platforms were used, VibeTrain® (Australia) and VibeTrain® Advanced (Armstrong Sports Health, AUS). The platforms were 2009 models. Vertical data were concurrently recorded with a three-dimensional motion analysis system (VICON Oxford Metrics, Oxford UK; six cameras, 500 Hz) and a tri-axial accelerometer (CortexBone Technology, San Jose, USA; 500 Hz). Data were recorded for five seconds during steady-state for three vibration platform speeds. After normality testing, 93% limits of agreement were computed to describe concurrent validity between the accelerometers and VICON.

RESULTS & DISCUSSION

Descriptive statistics are shown in Table 1: The difference in vibration platform frequency was less than ±0.56 Hz between VICON and a tri-axial accelerometer. The results of limits of agreement computed are shown in Figure 1. The 93% limit of agreement of the difference between the two measurement instruments was between 2.07 Hz or -2.13 Hz for each platform speed.

Table 1: Descriptive Statistics of a Tri-axial Accelerometer to VICON for various vibration platform speeds.

<table>
<thead>
<tr>
<th>Platform speed</th>
<th>Hz (± SE)</th>
<th>Mean difference (VICON-Accelerometer)</th>
<th>93% Limit of Agreement</th>
</tr>
</thead>
<tbody>
<tr>
<td>20</td>
<td>VICON 19.92 (0.24)</td>
<td>0.19 Hz</td>
<td>-2.13 Hz and 2.07 Hz</td>
</tr>
<tr>
<td></td>
<td>Accel 19.73 (0.50)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>25</td>
<td>VICON 24.88 (0.30)</td>
<td>0.62 Hz</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Accel 24.46 (1.85)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>30</td>
<td>VICON 20.78 (0.38)</td>
<td>-0.38 Hz</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Accel 30.16 (0.90)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

CONCLUSIONS

In order to prescribe vibration resistant platform frequency must be known. The preliminary study establishes the concurrent validity of a tri-axial accelerometer. When used in the field, researchers should consider vibration platforms frequency measured with the tri-axial accelerometer to be within ±2.5 Hz of actual frequency established by a gold standard measurement device.
NOTE: peak-to-peak displacement can be calculated from the equations of gravity quantification (Chapter 2, page 45).

Using the frequency (Hz) and amplitude (mm) of a vibration platform, it is possible to quantify WBV at the surface of the platform. Equation 1 shows the calculation of gravity ($g$) which is was reported within WBV literature along with frequency and amplitude (Rittweger, Schiessl & Felsenberg, 2001).

\[
g = \frac{A(2\pi f)^2}{9.81} \quad \text{equation 1}
\]

Where $A$ is the amplitude (m), $f$ is the frequency (Hz), $2\pi f^2$ is the maximum acceleration of the platform and 9.81 is the acceleration of gravity. If peak-to-peak displacement was 1.0 mm and frequency was 25 Hz, for example, then gravity ($g$) was 1.26 $g$, since $A$ was 0.0005 m.

Not shown in the conference abstract, reflective ball markers were placed on the end of the vibration platform to determine peak-to-peak displacement with VICON. As such peak-to-peak displacement at the end on the vibration platform was $\sim$ 3.00 mm.
APPENDIX C

Proceedings of the 28th Conference of the International Society of Biomechanics in Sports

July 19-23, 2010
Northern Michigan University
Marquette, Michigan USA

Edited by
Randall Jensen, William Ebben, Erich Petushek, Chris Richter, & Karen Roemer

Department of Health Physical Education and Recreation
College of Professional Studies
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245
TRANSMISSION OF VIBRATION ABOUT THE KNEE

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The purpose of this study was to examine and describe effects of knee flexion angle, stance width and vibration platform frequency on the transmission of vertical acceleration about the knee. Fifteen adults were exposed to various vibration conditions while standing on a side-to-side vibration platform. Vertical acceleration data, expressed as transmission, were shown to be attenuated for all vibration conditions. A larger degree of knee flexion however, was conducive to greater attenuation about the knee. Such information may be used to develop vibration training programs with a more thorough understanding of effects of vibration.

KEYWORDS: transmission, vibration, root mean square acceleration

INTRODUCTION: The mechanistic understanding of improved performance following vibration is uncertain. Specifically, the transmission of vibration throughout the body is not thoroughly understood. When someone stands on a vibration platform, a transmission value can be calculated to assist with the description of the magnitude of the vibration imposed on body landmarks. Generally, a transmission value does not discriminate between wobbling masses and ridged bodies, yet the value may be useful in identifying the rate of vibration absorption inter- and intra-individually.

Previously, transmission has been calculated by placing accelerometers on a vibration platform and the knee (Harazin & Grzesik, 1998), head (Abercromby et al., 2007), shank and thigh (Cook et al., 2010), pelvis and spine (Mansfield & Griffin, 2002) or directly into bone (Nsiah et al., 2006). Some methods are more invasive than others, yet an acknowledged error is created when accelerometers are mounted to skin. A data correction method to eliminate effects of local tissue-accelerometer resonance from surface measurements of vibration about the spine was proposed (Kitazaki & Griffin, 1995), yet since then, correction methods have not always been used.

Recently, skin-mounted accelerometers were shown to minimally affect impact accelerations during gait compared with bone-mounted accelerometers (Nsiah et al., 2006). As such, skin-mounted accelerometers were thought of as a good predictor of skeletal impact accelerations. Another study used a three-dimensional motion analysis system to measure transmission of vibration in order to eliminate error associated with skin-mounted accelerometers (Smith, Bressel & Snyder, 2009). For field work however, laboratory procedures are often unsuitable. Therefore, field studies generally acknowledge the limitation of skin-mounted accelerometers but implement their use because of ease of operation and transportability.

Our previous pilot study had validated accelerometers for measuring vibration platform frequency (Joseph & Furness, 2009). To further that work, the purpose of this study was to explore and describe effects of knee angle, stance width and platform frequency on transmission of vibration measured by skin mounted accelerometers.

METHODS: Fifteen healthy females and males (mean age = 19.6 years ± 1.4, stature = 1.76 m ± 0.08, mass = 70.5 kg ± 10.6) freely consented to participate in the study. Participants were free from muscular injury in the previous month and had no known joint injuries. Each participant randomly received 6 bouts of side-to-side vibration delivered by a sinusoidal oscillating vibration platform (Vibro-Trainer Semi-Commercial, Amazing Super Health, AUS). Each bout lasted 60 seconds and consisted of a predetermined stance posture and stance width while vibration platform frequency was randomly assigned. Stance posture consisted of
20°, 40° and 60° knee flexion, where 0° knee flexion corresponded with full knee extension. Stance width was 10 cm and 20 cm from the axis of rotation for each leg. Vibration platform frequency was 20 Hz, 25 Hz and 30 Hz. Data were collected for five seconds at each frequency after platform steady state had been achieved. The project had University Human Research Ethics Committee approval.

The independent variables were: (1) vibration platform frequency (20 Hz, 25 Hz, 30 Hz), (2) stance width (10 cm, 20 cm) and knee flexion angle (20°, 40°, 60°). The dependent variable, transmission, was calculated from the ratio of root mean square (RMS) knee acceleration (KRMS) to RMS platform (PRMS). A transmission value of 1.00 represented parity between the platform and knee. A transmission value less than 1.00 represented a larger PRMS than KRMS (figure 1).

Figure 1. An example of transmission, where the maximum acceleration about the knee is less than the maximum acceleration of the platform. For this example, the transmission value would be < 1.00. Note, data are m.s⁻² rather than RMS since negative values are squared when calculating RMS values.

Two 25 g tri-axial accelerometers (CXL25GP3, Crossbow Technology, San Jose, USA) sampling at 250 Hz were used to quantify vibration platform vertical acceleration and knee vertical acceleration. The mass of each accelerometer was 46 gm. One accelerometer was attached to the vibration platform with double sided adhesive tape. Another accelerometer was firmly taped to the left patella of a participant to reduce skin movement. Knee flexion angle was constant for each vibration bout and checked with a manual goniometer. The accelerometer was checked for vertical alignment during each vibration bout.

Knee flexion angle was filmed with a digital camcorder (NV-GS11, Matsushita Electric Industrial Co., Osaka JPN) and digitised with Silicoconcoach Pro 7 (Silicoconcoach, San Francisco, USA). Small reflective markers (1 mm diameter) were adhered on the skin to the right; greater trochanter, lateral condyle and lateral malleolus. Data were filmed for each knee flexion angle independent of stance width and vibration platform frequency.

Left leg length was measured with the "total true shortening" method (McRae, 1999). A tape was placed from the left anterior superior iliac spine to the left medial malleolus. The participant lay in the supine position. The average of two readings was recorded. These data were recorded since it was thought leg length may affect vibration transmission.

Data were imported to SPSS 17.0 for Windows (SPSS Inc., Chicago, USA). Descriptive statistics were calculated to quantify sample statistics, root mean square, knee angle and vibration transmission.

RESULTS: Sample descriptive statistics are shown in table 1. Acceleration about the knee and platform are shown in table 2. Acceleration was 28.55 m.s⁻² for 40° knee flexion, 20 cm stance width and 30 Hz platform frequency.
Table 1. Sample Descriptors

<table>
<thead>
<tr>
<th>Sex</th>
<th>n</th>
<th>Stature (m)</th>
<th>Mass (kg)</th>
<th>Left leg length (cm)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>mean</td>
<td>SD</td>
<td>mean</td>
</tr>
<tr>
<td>Female</td>
<td>7</td>
<td>1.72</td>
<td>0.08</td>
<td>63.76</td>
</tr>
<tr>
<td>Male</td>
<td>8</td>
<td>1.80</td>
<td>0.07</td>
<td>76.35</td>
</tr>
</tbody>
</table>

Table 2. Knee and Platform Acceleration for Vibration Platform Frequencies, Stance Widths and Stance Postures

<table>
<thead>
<tr>
<th></th>
<th>10 cm</th>
<th>20 cm</th>
<th>10 cm</th>
<th>20 cm</th>
<th>10 cm</th>
<th>20 cm</th>
<th>10 cm</th>
<th>20 cm</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>mean (SD)</td>
<td>mean (SD)</td>
<td>mean (SD)</td>
<td>mean (SD)</td>
<td>mean (SD)</td>
<td>mean (SD)</td>
<td>mean (SD)</td>
<td>mean (SD)</td>
</tr>
<tr>
<td>20°</td>
<td>7.85 (3.63)</td>
<td>10.30 (2.06)</td>
<td>14.42 (5.10)</td>
<td>20.01 (2.16)</td>
<td>17.27 (4.91)</td>
<td>28.35 (7.26)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>40°</td>
<td>8.53 (2.35)</td>
<td>12.07 (2.84)</td>
<td>10.10 (2.16)</td>
<td>22.96 (6.38)</td>
<td>24.62 (6.08)</td>
<td>28.55 (7.85)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>60°</td>
<td>5.89 (1.96)</td>
<td>9.52 (3.14)</td>
<td>13.34 (5.59)</td>
<td>16.09 (5.40)</td>
<td>17.36 (3.43)</td>
<td>24.03 (5.49)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Platform</td>
<td>19.13 (1.57)</td>
<td>30.02 (3.92)</td>
<td>47.19 (4.02)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Transmission of vibration about the knee is shown in table 3. At 30 Hz, for example, a 20 cm stance width and 60° knee flexion angle caused a transmission value of 0.55. Generally, the 60° knee flexion stance posture elicited greater transmission about the knee.

Table 4 shows variability of pre-determined stance postures during each vibration bout. Although the 20° knee flexion stance posture was most accurately maintained, variability across all stance postures was almost identical.

Table 3. Transmission of Vibration About the Knee for Various Vibration Platform Frequencies, Stance Widths and Stance Postures

<table>
<thead>
<tr>
<th></th>
<th>10 cm</th>
<th>20 cm</th>
<th>10 cm</th>
<th>20 cm</th>
<th>10 cm</th>
<th>20 cm</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>mean</td>
<td>SD</td>
<td>mean</td>
<td>SD</td>
<td>mean</td>
<td>SD</td>
</tr>
<tr>
<td>20°</td>
<td>0.73</td>
<td>0.67</td>
<td>0.87</td>
<td>0.74</td>
<td>0.57</td>
<td>0.68</td>
</tr>
<tr>
<td>40°</td>
<td>0.80</td>
<td>0.69</td>
<td>0.54</td>
<td>0.80</td>
<td>0.83</td>
<td>0.64</td>
</tr>
<tr>
<td>60°</td>
<td>0.57</td>
<td>0.57</td>
<td>0.77</td>
<td>0.55</td>
<td>0.57</td>
<td>0.53</td>
</tr>
</tbody>
</table>

Table 4. Pre-determined Stance Postures and Actual Knee Flexion Angles by ‘Siliconcoach’

<table>
<thead>
<tr>
<th>Pre-determined knee flexion angle</th>
<th>Exact</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
</tr>
<tr>
<td>20°</td>
<td>22.1°</td>
</tr>
<tr>
<td>40°</td>
<td>36.6°</td>
</tr>
<tr>
<td>60°</td>
<td>54.7°</td>
</tr>
</tbody>
</table>

DISCUSSION: The major finding of this study was that knee angle, stance width and vibration platform frequencies concurrently affected transmission when standing upon a vibration platform (table 3). The 60° knee flexion stance posture generally caused the lowest transmission values suggesting that vibration was attenuated about the lower limbs to a greater extent than other stance postures. Knee angle however, was varied (table 4). The finding may be supported by previous work of electromyogram (EMG) data. Several authors have reported increased EMG activity of vastus lateralis during vibration with an isometric squat of 65° knee flexion (Roelants et al., 2008; Cardinale & Lim, 2003). Future research should concurrently investigate transmission and EMG activity to further understand biological responses to vibration. Methods of this study allowed quantification of transmission despite the known limitation of skin-mounted accelerometers. The accelerometers as such, were sufficiently sensitive enough to detect such transmission attenuation. Though we did not quantify the error of
potential skin-accelerometer resonance, the error appeared consistent rather that sporadic and may explain the variance of acceleration (table 2).

Since a common goal of vibration exercise was to improve muscular strength and power in various target populations, future research should first justify a training protocol and second monitor the effectiveness of the protocol throughout the intervention. To date, reports of vibration transmission pre-, during- and post-intervention are not reported in the literature. Such knowledge may enhance understanding of biological mechanisms manipulated by vibration training.

Since we have shown that various independent variables concurrently affect vibration transmission, we propose it should be reported and monitored in all vibration protocols. Not surprisingly, effects of vibration training on transmission are unknown about the knee and other body landmarks.

CONCLUSION: Collectively knee flexion angle, stance width and vibration platform frequency affect transmission of vibration. Future research should measure vibration transmission about various body landmarks and in both healthy and sub-optimal health populations in an effort to determine the most appropriate vibration training protocol.

REFERENCES:

Acknowledgement
The authors would like to thank Sofie Synahriska of Amazing Super Health for the use of the vibration platform.
Dear

In conjunction with the Australian Catholic University (ACU) we are conducting a free to participate research project into the benefits of whole body vibration as a very gentle exercise program for you.

This would mean a researcher from ACU coming to your home twice a week with a vibration platform, at times and days convenient to you. Each home visit will occupy about 15 minutes of your day. You will be asked to stand on that platform for five lots of one minute exercise with one minute rest in between (a total of about 9 minutes). There are two different types of platforms. You will stand on one of these over the first six weeks, have a two week break, and then stand on the second platform for another 6 weeks.

The idea of the project is to increase your exercise capacity without making you breathless, therefore improving your ability to complete typical everyday activities like walking about and getting out of a chair. Your total involvement is 14 weeks.

For more information please contact:

Name: Trent Furness  Name: Nicole Bate
School of Exercise Science  Dept Respiratory Medicine
Australian Catholic University  Monash Medical Centre
Ph: 9953-3041  OR  0432-299-956  Ph: 9594-2279
email: trentham.furness@acu.edu.au  email: nicole.bate@southernhealth.org.au
APPENDIX G

Human Research Ethics Committee
Committee Approval Form

Principal Investigator/Supervisor: Christian Lorenzen  Melbourne Campus
Co-Investigators: Melbourne Campus
Student Researcher: Trehani Furness Melbourne Campus

Ethics approval has been granted for the following project:
Whole Body Vibration and Chronic Obstructive Pulmonary Disease
for the period: 07/04/2011-07/05/2012
Human Research Ethics Committee (HREC) Register Number: V2011 01

Special Conditions of Approval

Prior to commencement of your research, the following permissions are required to be submitted to the
ACU HREC:
N/A

The following standard conditions as stipulated in the National Statement on Ethical Conduct in
Research Involving Humans (2007) apply:

(i) that Principal Investigators / Supervisors provide, on the form supplied by the Human
Research Ethics Committee, annual reports on matters such as:
• security of records
• compliance with approved consent procedures and documentation
• compliance with special conditions, and

(ii) that researchers report to the HREC immediately any matter that might affect the ethical
acceptability of the protocol, such as:
• proposed changes to the protocol
• unforeseen circumstances or events
• adverse effects on participants

The HREC will conduct an audit each year of all projects deemed to be of more than low risk. There will
also be random audits of a sample of projects considered to be of negligible risk and low risk on all
 Campuses each year.

Within one month of the conclusion of the project, researchers are required to complete a Final Report
Form and submit it to the local Research Services Officer.

If the project continues for more than one year, researchers are required to complete an Annual Progress
Report Form and submit it to the local Research Services Officer within one month of the anniversary date
of the ethics approval.

Signed: ___________________________ Date: 07/04/2011
(Research Services Officer, Melbourne Campus)

(Research Services Officer, Melbourne Campus)
24 August 2011

Prof Geraldine Naughton
School of Exercise Science
Australan Catholic University
Locked Bag 4115
Fitzroy MDC 3065

Dear Prof Naughton

Study title: Whole body vibration and chronic obstructive pulmonary disease
Southern Health HREC Ref: 11222A

The Southern Health HREC A reviewed the above application at the meeting held on 04 August 2011 In addition, the HREC is satisfied that the responses to our correspondence of 9 August 2011 have been sufficiently addressed.

The HREC approved the above application on the basis of the information provided in the application form, protocol and supporting documentation.

Approval

The HREC approval is from the date of this letter.

Approval is given in accordance with the research conforming to the National Health and Medical Research Council Act 1992 and the National Statement on Ethical Conduct in Human Research (2007). The HREC has ethically approved this research according to the Memorandum of Understanding between the Consultative Council and the participating organisations conducting the research.

Approval is given for this research project to be conducted at the following sites and campuses:

- Southern Health

You must comply with the following conditions:

The Chief Principal Investigator is required to notify the Administrative Officer, Research Directorate, Southern Health of:

1. Any change in protocol and the reason for that change together with an indication of ethical implications (if any)
2. Serious or unexpected adverse effects of project on subjects and steps taken to deal with them
APPENDIX I

The Mini-Mental State Exam (MMSE)

Participant_________________________ Examiner_________________________ Date__________

Maximum Score

Orientation

5 ( ) What is the (year) (season) (date) (day) (month)?
5 ( ) Where are we (state) (country) (town) (hospital) (floor)?

Registration

3 ( ) Name 3 objects: 1 second to say each. Then ask the patient all 3 after you have said them. Give 1 point for each correct answer. Then repeat them until he/she learns all 3. Count trials and record.

Trials ________

Attention and Calculation

5 ( ) Serial 7’s. 1 point for each correct answer. Stop after 5 answers. Alternatively spell “world” backward.

Recall

3 ( ) Ask for the 3 objects repeated above. Give 1 point for each correct answer.

Language

2 ( ) Name a pencil and watch.
1 ( ) Repeat the following "No ifs, ands, or buts"
3 ( ) Follow a 3-stage command:
"Take a paper in your hand, fold it in half, and put it on the floor."
1 ( ) Read and obey the following: CLOSE YOUR EYES
1 ( ) Write a sentence.
1 ( ) Copy the design shown.

_____ Total Score
Figure 1 The Snellen Eye Chart.
Figure 2 The Melbourne Edge Test.
## Shortness of Breath

**Modified Borg Dyspnea Scale**

<table>
<thead>
<tr>
<th>Scale</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Nothing at all</td>
</tr>
<tr>
<td>0.5</td>
<td>Very, very slight (just noticeable)</td>
</tr>
<tr>
<td>1</td>
<td>Very slight</td>
</tr>
<tr>
<td>2</td>
<td>Slight</td>
</tr>
<tr>
<td>3</td>
<td>Moderate</td>
</tr>
<tr>
<td>4</td>
<td>Somewhat Severe</td>
</tr>
<tr>
<td>5</td>
<td>Severe</td>
</tr>
<tr>
<td>6</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>Very Severe</td>
</tr>
<tr>
<td>8</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>Very, very severe (almost maximal)</td>
</tr>
<tr>
<td>10</td>
<td>Maximal</td>
</tr>
</tbody>
</table>

- TeleRehab™ Advantage Cardiopulmonary Monitoring System
- NICORE™ External Counterpulsation (ECP) Therapy System
- ROZINN™ Diagnostic Cardiology Suite (Holter, Event, ABP)

ScottCare Corporation  
4791 West 150th Street  
Cleveland, Ohio 44135  
A Scott Fetzer, Berkshire Hathaway Company

Phone: 800-243-9412, ext. 116  
Fax: 216.267.6129  
www.scottcare.com
APPENDIX L
# APPENDIX M

Table 1

Successful and Unsuccessful Modes of Participant Recruitment for the 14 Week Intervention

<table>
<thead>
<tr>
<th>Unsuccessful participant recruitment options</th>
<th>Successful participant recruitment options</th>
</tr>
</thead>
<tbody>
<tr>
<td>General Practitioners at four community medical centres based about the Mornington Peninsula.</td>
<td>COPD suppose group; Rosebud Better Breathers</td>
</tr>
<tr>
<td>St Vincent’s Hospital, Melbourne. Department of (Restoring Health Program)</td>
<td>Monash Medical Centre, Department of Sleep and Respiratory Medicine, Southern Health</td>
</tr>
<tr>
<td>Frankston Hospital. Department of Respiratory Specialists at 2 private practices in Frankston</td>
<td>McKinnon Sports Medicine Clinic</td>
</tr>
<tr>
<td>Catholic housing</td>
<td></td>
</tr>
<tr>
<td>COPD suppose group; Hawthorn Huffers. And a few more</td>
<td></td>
</tr>
</tbody>
</table>

n = 0  
n = 16
APPENDIX N

Picture 1. View of PLACEBO vibration platform.

Specifications of the prototype vibrating platform:

- Baldor Electrical Co USA
  - Industrial Motor Direct Current
  - 1750 RPM
  - 29.16 Hz
  - 180 V
  - 2.5 Amps
  - Cam wheel/lever arm set for 0.0 mm perk-to-peak displacement
  - Weight ~ 25 kg
Dimensions of the prototype vibrating platform:

- **Lateral View**
  - 55 cm long by 26 cm high
  - Total height (including handle) 130 cm
- **From Above**
  - 55 cm by 55 cm (78 cm diagonal)

Calibration of frequency for the prototype vibrating platform was performed with the same tri-axial accelerometers used in Appendix H.

**Picture 2.** Lateral view of platform.  **Picture 3.** Superior view of platform.
APPENDIX O

WBV INTERVENTION

**Figure 1.** Raw data of the TUG test.

**Figure 2.** Raw data of the 5-chair test.
Figure 3. Raw data of long-term stride length.

Figure 4. Raw data of long-term stride time.
**Figure 5.** Raw data of long-term stride velocity.

**Figure 6.** Raw data of acute stride length.
**Figure 7.** Raw data of acute stride time.

**Figure 8.** Raw data of acute stride velocity.
PLACEBO INTERVENTION

Figure 9. Raw data of the TUG test.

Figure 10. Raw data of the 5-chair test.
Figure 11. Raw data of long-term stride length.

Figure 12. Raw data of long-term stride time.
Figure 13. Raw data of long-term stride velocity.

Figure 14. Raw data of acute stride length.
Figure 15. Raw data of acute stride time.

Figure 16. Raw data of acute stride velocity.