Effects of Commuter Cycling on Physical Activity and Cardiovascular Health

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A thesis submitted for the degree of Masters of Physical Education at the University of Otago, Dunedin, New Zealand

30/06/2015
STUDENT DECLARATION

DECLARATION CONCERNING THE THESIS PRESENTED FOR THE DEGREE OF

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☐ The material has not previously been accepted in whole, or in part, for any other degree or diploma

Signature: Brendon James Novis Date: 30/8/2015

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ABSTRACT

Few have utilised a randomised controlled study design to monitor cardiovascular health in adults who replace automotive forms of transport with cycling\(^1,2\). Previous commuter cycling (CC) interventions observed enhanced cardiorespiratory fitness, mixed effects on body composition, whereas markers of insulin resistance have not been investigated. Furthermore, studies to date have omitted measures of physical activity (PA) and energy expenditure (EE) prior to initiating a routine of CC and therefore the contribution of the novel commuter cycling on existing PA and EE is unknown. The purpose of this study was to examine effects of CC on existing PA and EE, body composition and parameters of cardiovascular and metabolic health. Methods: Males and females who commuted by motorised transport were recruited and completed a graded exercise test on a cycle ergometer to determine $\dot{V}O_2$max, completed weighed food records, PA log books, and a validated PA questionnaire. Group assignment was randomised and matched for sex and fitness. The cycling group (CYC) (n=14, age 39±7 yr, body mass 77±11 kg, $\dot{V}O_2$max 2.8±0.8 l•min\(^{-1}\)) were given bicycles and asked to cycle commute for 100+ min/wk for 10 weeks. The control group (CON) (n=14, age 34±8 yr, body mass 70±7 kg, $\dot{V}O_2$max 2.6±0.7 l•min\(^{-1}\)) continued using motorised transport. Baseline (wk0) testing included cholesterol fractions, triglycerides, C-reactive protein, fasting insulin and glucose (HOMA-IR) and body composition (mass, skin folds), blood pressure, resting heart rate and measures were repeated wk10. CYC also underwent pre and post-intervention dual-energy x-ray
absorptiometry (DXA) scans for assessment of body composition. Energy expenditure was estimated with energy intake data (wk0, wk4, wk8) with adjustment for energy balance from measures of fasted body mass at the same time points. Data were analysed using ANOVA for the group change from wk0 to wk10, and repeated measures ANOVA for data collected at 3 or more time points.

Results: Analyses included 13 participants per group. CYC cycled 152 ± 60 min/wk, but did not alter (P>0.05) total levels of PA, energy intake or estimated EE. Consequently, no changes in body fat (P=0.69) or mass (P=0.61) were observed. VO₂max (CYC: 10±17%, CON: -1.5±11%, P=0.03) and resting HR (CYC: -5±6%, CON: 3±9%, P=0.03) were improved, whereas diastolic BP increased in CON (CYC: -1±7%, CON 12±16%, P=0.02). There were no significant changes within or between groups for blood parameters. Conclusion: Commuter cycling for ~150 min/wk does not increase total PA and EE, which may be due to a reduction in non-cycling physical activities or the instruments used for PA and EE measures may have lacked resolution to capture change to short-lasting and spontaneous PA. Nevertheless, major markers for cardiovascular health were enhanced in just 10 weeks and the seasonal increase in diastolic BP observed in CON was prevented with CC in CYC. The lack of significant changes in blood parameters may be due to inadequate volume of exercise or duration of intervention. Research was supported by the New Zealand Heart Foundation and School of Physical Education, Sport and Exercise Sciences at University of Otago.
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**ABBREVIATIONS**

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
</tr>
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<tbody>
<tr>
<td>ANOVA</td>
<td>(analysis of variance)</td>
</tr>
<tr>
<td>BP</td>
<td>(blood pressure)</td>
</tr>
<tr>
<td>BMI</td>
<td>(body mass index)</td>
</tr>
<tr>
<td>BPM</td>
<td>(beats per minute)</td>
</tr>
<tr>
<td>CC</td>
<td>(commuter cycling)</td>
</tr>
<tr>
<td>CM</td>
<td>(centimetre)</td>
</tr>
<tr>
<td>CON</td>
<td>(control group)</td>
</tr>
<tr>
<td>CRP</td>
<td>(C-reactive protein)</td>
</tr>
<tr>
<td>CVD</td>
<td>(cardiovascular diseases)</td>
</tr>
<tr>
<td>CYC</td>
<td>(cycling group)</td>
</tr>
<tr>
<td>DLW</td>
<td>(doubly-labelled water)</td>
</tr>
<tr>
<td>DXA</td>
<td>(dual-energy x-ray absorptiometry)</td>
</tr>
<tr>
<td>EE</td>
<td>(energy expenditure)</td>
</tr>
<tr>
<td>g</td>
<td>(gram)</td>
</tr>
<tr>
<td>HDL</td>
<td>(high-density lipoprotein)</td>
</tr>
<tr>
<td>HOMA-IR</td>
<td>(homeostatic model of assessment)</td>
</tr>
<tr>
<td>HR</td>
<td>(heart rate)</td>
</tr>
<tr>
<td>HR_{max}</td>
<td>(maximal heart rate)</td>
</tr>
<tr>
<td>IL-6</td>
<td>(interlukin-6)</td>
</tr>
<tr>
<td>IPAQ</td>
<td>(International Physical Activity Questionnaire)</td>
</tr>
<tr>
<td>Kcal</td>
<td>(kilocalorie)</td>
</tr>
<tr>
<td>Kg</td>
<td>(kilogram)</td>
</tr>
<tr>
<td>kJ</td>
<td>(kilojoule)</td>
</tr>
<tr>
<td>LDL</td>
<td>(low-density lipoprotein)</td>
</tr>
<tr>
<td>Max</td>
<td>(maximum)</td>
</tr>
<tr>
<td>Min</td>
<td>(minutes)</td>
</tr>
<tr>
<td>min/wk</td>
<td>(minutes per week)</td>
</tr>
<tr>
<td>ml</td>
<td>(millilitre)</td>
</tr>
<tr>
<td>mmHg</td>
<td>(millimetre of mercury)</td>
</tr>
</tbody>
</table>
MVPA  (moderate and vigorous physical activity)
PA     (physical activity)
QOL    (quality of life)
RER    (respiratory exchange ratio)
RPM    (revolutions per minute)
SF-36  (short-form 36 questionnaire)
$\dot{V}O_2$max (maximal oxygen consumption)
wk     (week)
w      (watts)
WHR    (waist-to-hip ratio)
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Associate Professor Nancy Rehrer for endless support, time, effort and above all, incredible patience. As a friend, supervisor, colleague, and fellow cyclist, you have shaped the many facets of my life and I wouldn’t be the person I am today without your friendship.

The School of Physical Education, Sport and Exercise Sciences at the University of Otago for the generous resources that assisted me directly with this thesis research, as well as with my professional development.

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And lastly, Dr. Tina Manos for never being short of a great idea.
CHAPTER ONE: Introduction

Robust evidence supports the role of physical activity (PA) to reduce risk for developing various diseases, most notably those that affect metabolic and cardiovascular function (for review, see Haskell, et al., 2007). Participation in sport, recreation and active living are all practical means to accumulate public health recommendations for moderate intensity PA of 150+ min/wk (Haskell, et al., 2007). Active living may support integration of, and adherence to, PA into a daily routine better than group activities and facility-based programs (e.g. gyms) (Hillsdon, et al., 1995). Commuter cycling is an efficient and practical example of active living, as it assists with the incorporation of PA into the time of day that would otherwise be unproductive. Although walking is convenient for short distances, cycling can cover a greater distance per time and is performed at a physiological intensity that generally supports greater cardiovascular benefits than those from walking (Oja, et al., 1991). If performed by a large proportion of the population, commuter cycling may alleviate traffic congestion, air pollution, and some public health concerns such as those caused by physical inactivity (e.g. obesity).

New Zealand rates third highest in the world for vehicle ownership in proportion to its population, with 607 vehicles per 1000 people (The Economist, 2008; Tin Tin, et al., 2009). The high rate of vehicle ownership may increase the frequency of automotive trips, which may help to explain why two-thirds of all vehicle trips are six km or less, with half of those trips three km or less (Tin Tin, et al., 2009). To reduce the high rate of vehicle use, the Ministry of Transport aims to increase the total number of trips made
by active transport from less than 3% up to 30% by year 2040 (Ministry of Transport, 2008; Tin Tin, et al., 2009). The optimistic active-transportation goals set by the Ministry of Transport will necessitate promotional strategies to encourage individuals to replace short vehicle trips with physically active modes of transport. Health promotion is sometimes utilised as a marketing tool to promote societal change, and health promotion campaigns were observed to increase participation in active transportation in Northern Europe (Vuori, et al., 1998; Oja & Vuori, 2000). However, the health benefits achieved from initiating a routine of commuter cycling are not well understood, which limits the use of health promotional strategies by government organisations to achieve their active transportation goals.

Recreational cycling (i.e. sport or leisure-time cycling) is known to sufficiently strain metabolic and cardiovascular systems providing a protective effect against heart disease, stroke, high blood pressure, unfavorable blood lipid profiles, metabolic syndrome, some forms of cancers, improved cardiorespiratory and muscular fitness, reduced depression, and better cognitive function (for review, see Oja, et al., 2011). Commuter cycling differs considerable from recreational cycling and may not offer the same benefits to health. For example, the time of day that cyclists commute will generally coincide with peak traffic hours. Increased ventilation with exercise combined with increased car emissions at peak traffic times may have adverse health effects (Bigazzi & Figliozzi, 2014). Peak traffic may also impose frequent periods of inactivity at intersections and the physiological intensity of cycling may be lower than that of recreational cycling due to social norms in the workplace regarding body sweat, odor and attire, particularly
if showering facilities are not present. Finally, cycling on the road increases the risk of injury and mortality compared to motorised transport (including public transport) and walking (Tin Tin, et al., 2010). Acquiring an injury can decrease quality of life and health (e.g. Moreira, et al., 2014). Although evidence of improved health with recreational cycling is robust, it is unclear if commuter cycling offers the same benefits.

Few have examined the role of cycling in a transportation context for reduced risk of developing disease (Oja, et al., 1991; Hendriksen, et al., 2000; de Geus, et al., 2008; Møller, et al., 2011). Aforementioned authors all reported significant improvements to \( \dot{V}O_{\text{2max}} \) from commuter cycling. Improvements to other health parameters from commuter cycling are not well supported. For example, the findings of decreased body fatness (Møller, et al., 2011) and a lack of change in inflammation markers (de Geus, et al., 2008) come from one investigation each, with confirmation from other commuter cycling interventions lacking. Several studies have concluded that commuter cycling is ineffective in altering blood pressure (de Geus, et al., 2008; Møller, et al., 2011), blood cholesterol and triglycerides (Oja, et al., 1991; de Geus, et al., 2008), in 8, 10, and 52 week trials. Although the aforementioned findings are supported in multiple studies, the varying length of commuter cycling studies and load of cycling performed weakens the evidence from the repetitive findings. Whether or not commuter cycling can enhance insulin and glucose dynamics has yet to be investigated in a peer-controlled intervention. Furthermore, previous commuter cycling interventions excluded pre-intervention measures of physical activity and energy expenditure. Therefore, it is unknown if compensation in existing PA and EE occurred, which could reduce the
benefits from initiating a routine of cycle commuting. On the other hand, novel commuter cycling may have inspired PA beyond that accumulated from cycling. Thus, health gains could be attributed to the commuter cycling and other forms of PA combined.

In summary, health promotion initiatives will require evidence of health gains with commuter cycling to promote participation in commuter cycling to improve social problems such as traffic congestion, air pollution, as well as possibly improve personal health. Controlled interventions monitoring the health gains of commuter cycling are few, and vary in design and quality, thus preventing generalisations about the health benefits of this form of cycling. Furthermore, it is unknown how initiating a routine of commuter cycling will impact total PA and EE and whether health responses obtained, or lack thereof, are related to these parameters. Therefore it was the purpose of this thesis research to investigate the effects of bicycle commuting at a self-selected intensity for 10 weeks on existing PA and EE, and on parameters of health.

The objectives were to determine the extent to which incorporating bicycle commuting into the daily routine 1) increases overall physical activity and energy expenditure, 2) improves body composition, 3) enhances cardiovascular and metabolic health and 4) improves health related quality of life. The hypotheses were that commuter cycling will 1) increase total levels of physical activity, 2) increase cardiovascular fitness and 3) improve high-density lipoprotein, fasting insulin and glucose.
CHAPTER TWO: Literature Review

2.1 Introduction

Commuter cycling is a utilitarian activity that offers the convenience of accumulating physical activity into the time of day generally reserved for physically inactive transport to/from work, whilst potentially saving costs from fuel and parking, reducing carbon emissions compared to automotive transport and generally offers greater cardiovascular and metabolic strain than walking (Oja, et al., 2011; Oja, et al., 1991). However, when compared to recreational cycling, cycling for transport may be less intense, of shorter duration and have a higher potential for adverse health effects (e.g. air pollutants, traffic incidents) (Oja, et al., 2011). Although recreational cycling is well established to enhance cardiovascular health, commuter cycling has received considerably less attention in research literature and little is known about the health gains to be expected upon incorporating commuter cycling into a daily routine (Oja, et al., 2011). Large population studies utilising cross-sectional research methods report that commuter cyclists have favourable markers for cardiovascular health above those who use motorised transport (Hamer & Chida, 2008). However, randomised controlled trials on the protective effect from cardiovascular disease with commuter cycling are ambiguous. Current evidence on the role of commuter cycle on parameters of health (e.g. blood cholesterol and inflammation, blood pressure, glucose/insulin dynamics, abdominal body fat) are equivocal due to a lack of repetition in the research literature, or a complete absence of investigative evidence. Evidence is further hindered because the impact that novel commuter cycling has on participants’ physical activity (PA) and
energy expenditure (EE) is unknown. Without first understanding the impact of novel PA on existing activity and EE, the benefits of the PA cannot be ascertained. The forthcoming chapter will review known evidence on the health benefits gained from physically active transportation, with emphasis on commuter cycling where evidence is available. The mechanisms responsible for the reduced risk of cardiovascular disease through commuter cycling will be examined, as well as the effects of novel exercise on total physical activity and energy expenditure will be explored.

2.2 Physical Activity

Effects of commuter cycling on physical activity

The effect of commuter cycling on total PA has not been investigated with an experimental research design. In a systematic review on the associations between physically active transport and total PA, 14 of the 15 included studies reported positive associations between physically active transport and increased total PA (Wanner, et al., 2012). Five of the 16 studies reported associations between all PA variables; nine reported some association, one study observed no association between physically active transport and PA (Wanner, et al., 2012). However, most studies combined walking and cycling as modes of transport and are inclusive of all forms of transport, not just commuting to and from the workplace, which limits generalisations for the role of
commuter cycling. Four studies from the review also reported the relationship of cycling for transport on total PA (Table 2.1). Wanner et al. (2012) identified four studies that observed an association with cycling for transport and greater scores in some PA variables “(i.e. time engaged in PA and/or frequency of PA bouts per week) over non-active transportation users (Boone-Heinonen, et al., 2009; Titze, et al., 2008; Gómez, et al., 2005; Wanner, et al., 2012). Wanner et al. (2012) developed a rating scale to define the quality of included studies, which was based on the objectivity of methods for PA and transport measures, validity of measurement tools, sample size, research design and control for other variables (e.g. age, gender, education, income). The aforementioned studies are presented in table 2.1, which averaged a rating of four on a one (lowest) to ten (highest) scale. The relatively low-quality score for the studies on the role of cycling for transport on increased total PA, in conjunction with no known data from experimental study designs warrants further investigation into the potential for commuter cycling to be utilised to increase total PA.

*Time spent commuter cycling*

Novel commuter cycling in previous longitudinal studies was generally performed below government-issued recommendations on the amount of moderate-intensity PA to enhance or maintain health (e.g. 150 min/wk moderate PA) (Oja, et al., 1991; Hendriksen, et al., 2000; de Geus, et al., 2008; de Geus, et al., 2009), with one exception (Møller, et al., 2011). Time engaged in commuter cycling by participants from Møller et al. (2011), was not measured directly; rather it was estimated from the distance
participants cycled. An estimate of 200 min/wk of commuter cycling was performed by participants.

![Image](image_url)

Table 2.2 Summary of studies on the association between cycling for transport on increased total physical activity, adapted from Wanner et al., 2012.

<table>
<thead>
<tr>
<th>Author, location</th>
<th>Sample size</th>
<th>Method for CYC measures</th>
<th>Method for PA measures</th>
<th>Statistical analyses</th>
<th>Association of CYC on PA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Butler et al., 2007. Canada</td>
<td>77,953</td>
<td>Self-report Categories: cyclist or non-cyclist</td>
<td>Self-report Categories: inactive, moderate, active</td>
<td>Logistic regression: OR (95% CI)</td>
<td>OR for CYC according to PA category: Men: OR = 1.93 (1.63, 2.29); Women: OR = 1.80 (1.49, 2.20).</td>
</tr>
<tr>
<td>Gomez et al., 2005. Columbia</td>
<td>1,464</td>
<td>Self-report Categories: regular cycling activity, irregular cycling activity, inactive</td>
<td>Self-report Categories: regular (meeting PA guidelines), irregular (some PA), inactive (no PA)</td>
<td>Logistic regression: OR (95% CI)</td>
<td>OR for bicycling related to LTPA: OR irregPA = 1.84 (1.26, 2.69); OR regPA = 1.10 (0.60, 2.02).</td>
</tr>
<tr>
<td>Titze et al., 2008. Austria</td>
<td>998</td>
<td>Self-report Categories: cycling ≥1d/wk, non-cyclist &lt;1d/wk</td>
<td>Self-report Categories: inactive, moderately active, highly active</td>
<td>Factor analysis OR (95% CI)</td>
<td>OR for being a cyclist (≥1/week) according to PA category: OR mod = 0.82 (0.52, 1.30); OR high = 2.30 (1.59, 3.32).</td>
</tr>
<tr>
<td>Boone-Heinonen et al., 2009. U.S.A.</td>
<td>2,717</td>
<td>Self-report Categories: cyclist or non-cyclist</td>
<td>Self-report categories: high or low</td>
<td>Multinomial logistic regression: OR (95% CI)</td>
<td>OR for CYC compared to car according to PA category: to recreational facility: OR high = 1.52 (0.82, 2.82); to park: OR high = 1.75 (1.24, 2.47); to grocery store: OR high = 1.35 (0.87, 2.11); to public transit: OR high = 0.96 (0.39, 2.36).</td>
</tr>
</tbody>
</table>

Abbreviations: day (d), week (wk), cycling (CYC), physical activity (PA), leisure-time physical activity (LTPA), confidence intervals (CI), odds ratio (OR), moderately active (mod), regular physical activity (reg), irregular physical activity (irreg).
Whether or not this increased PA levels beyond pre-intervention levels was not reported, but authors reported significantly improved body composition (i.e. skin fold thickness) after 8 weeks of commuter cycling, which indicates the load of PA from cycling likely increased total PA levels (Møller, et al., 2011). Improved body composition wasn’t observed in any other commuter cycling study (detailed in subsequent section), but in those studies, participants cycled less than the public recommendations for PA. In another commuter cycling intervention, participants cycled less than 150 min/wk, but exceeded the amount of time recommended for weekly PA through combined commuter cycling and leisure-time PA (de Geus, et al., 2008, 2009). However, the impact that commuter cycling had to existing PA is unknown, as de Geus et al. (2008, 2009) didn’t report pre-intervention levels of PA. In summary, only one study contained participants that cycled for transport above public recommendations for PA and another study observed participants to meet PA guidelines through combined cycling and existing PA habits. It is unknown however, if commuter cycling enhances existing levels of PA.

*Time spent commuter cycling*

Novel commuter cycling in previous longitudinal studies was generally performed below public recommendations on the amount of moderate-intensity PA to enhance or maintain health (e.g. 150 min/wk moderate PA) (Oja, et al., 1991; Hendriksen, et al., 2000; de Geus, et al., 2008; de Geus, et al., 2009), with one exception (Møller, et al., 2011). Time engaged in commuter cycling by participants from Møller et al. (2011), was
not measured directly; rather it was estimated from the distance participants cycled. An estimate of 200 min/wk of commuter cycling was performed by participants. Whether or not this increased PA levels beyond pre-intervention levels was not reported, but authors reported significantly improved body composition (i.e. skin fold thickness) after 8 weeks of commuter cycling, which indicates the load of PA from cycling likely increased total PA levels (Møller, et al., 2011). Improved body composition wasn’t observed in any other commuter cycling study (detailed in subsequent section), but in those studies, participants cycled less than the public recommendations for PA. In another commuter cycling intervention, participants cycled less than 150 min/wk, but exceeded the amount of time recommended for weekly PA through combined commuter cycling and leisure-time PA (de Geus, et al., 2008, 2009). However, the impact that commuter cycling had to existing PA is unknown, as de Geus et al. (2008, 2009) didn’t report pre-intervention levels of PA. In summary, only one study contained participants that cycled for transport above public recommendations for PA and another study observed participants to meet PA guidelines through combined cycling and existing PA habits. It is unknown however, if commuter cycling enhances existing levels of PA.

**Physiological intensity of commuter cycling**

Although measures of the duration of cycling and existing PA in previous studies lacked objectivity, measurements of the physiological intensity of commuter cycling was performed with highly standardised measurement procedures in several studies (Oja,
et al., 1991; Hendriksen, et al., 2000; de Geus, et al., 2007, 2008, 2009). In general, commuter cycling is performed at 60-65% of participants' VO$_2$max (Oja, et al., 1991; Hendriksen, et al., 2000; de Geus, et al., 2008). However, one group of participants cycled at 75% VO$_2$max when performing a field test along their typical commuting route (de Geus, et al., 2007). Intensity of the field test was measured with a portable, indirect calorimetry system. Although the indirect calorimetry is a gold standard method for measuring respiratory function from a free-living activity, participants rode significantly faster during the field test than their normal commute and therefore the measure of intensity is an over estimate of those participants' normal commute (de Geus, et al., 2007). Data indicates commuter cycling is generally performed at moderate-intensity, which provides enough cardiovascular strain to promote adaptations that are known to reduce risk for developing disease (Haskell, et al., 2007; Lee, et al., 1995). This evidence hints at the prospect of commuter cycling being a viable means to increase health, however, the duration people are willing to engage in commuter cycling per week, as well as the effects it may or may not have on total level of PA is still unknown and requires investigation to determine if commuter cycling can be promoted as a health enhancing activity.

Compensation of Energy Expenditure and Physical Activity

Initiating novel PA into the daily routine may or may not result in increased levels of EE or PA. Individuals may respond to novel PA by reducing non-exercise activities (Goran & Poehlman, 1992). Non-exercise activities include spontaneous movements
associated with posture, daily living and fidgeting (Goran & Poehlman, 1992). Both biological and behavioural mechanisms may explain the compensation of activity and EE (Garland, et al., 2010). For example, biological sources for compensation may be attributed to sympathetic response to restore energy balance after PA (Meijer, et al., 1991). Alternatively, individuals may knowingly restore energy balance by physically resting or by increasing caloric intake (King, et al., 2006). However, any account on the mechanisms that drive energy compensation should be approached with caution as underlying mechanisms are not well understood and involve hormones, circulating and stored substrates, feedback from peripheral tissues and substrate utilisation to higher centres (for review, see Garland, et al., 2010).

Compensation from non-exercise activity

Studies that have monitored the effects of exercise on the energy expended from non-exercise activities are surprisingly sparse and even fewer studies exist on the detriment, or lack thereof, that EE compensation has on health-related outcome variables. For example, compensation of EE (Goran & Poehlman, 1992) and PA (Meijer, et al., 1999) has been observed with elderly participants who have partaken in an exercise training program. Goran and Poehlman (1992) observed a return to pre-intervention levels of EE during the last 10 days of the eight week exercise program, utilising the doubly labelled water (DLW) technique. Similarly, Meijer, et al., (1999) observed a significant decrease of non-exercise activities on training days during the 12th and final week of their exercise training intervention. Nevertheless, training programs from both studies
improved $\text{VO}_2\text{max}$ among the elderly participants, even though compensation of PA and EE was observed (Goran & Poehlman, 1992; Meijer, et al., 1999).

In support of the aforementioned studies, Colley et al. (2010) reported compensation of EE among obese women initiating a walking intervention. The walking intervention increased participant EE between ~4200-6300 kJ/wk. However, total EE remained unchanged from pre-intervention measures, assessed by DLW method (Colley, et al., 2010). Colley et al. (2010) suggested that the lack of change in total EE during the intervention was due to compensation from non-exercise activities, an increase in sleep time and reduction in light-intensity PA. Authors also commented that it is unknown if the reduction in light activities resulted from decreased frequency and/or duration of light PA, or if light activities were increased into moderate-intensity activities (Colley, et al., 2010).

Research on elderly participants (Goran & Poehlman, 1992; Meijer, et al., 1999) and obese women (Colley, et al., 2010) indicates that novel PA may decrease EE spent during non-exercise and light-intensity activities. In support of this, a meta-analytic review reported that within-individual variation of daily EE is 8% and increases to 10% after three months of training and variation may rise to 15% with long-term training (Black & Cole, 2010). Surprisingly, similar observations of EE compensation have been observed among young adults (Wickel & Eisenmann, 2006). Using the DLW technique, Wickel and Eisenmann (2006) observed EE from light-intensity activities to change inversely with the amount of moderate and vigorous PA performed by participants.
Few have reported evidence for compensation with novel PA, nevertheless it has been observed in elderly, young adults and obese women. Despite the presence of compensation, participants' VO$_{2\text{max}}$ was significantly improved. How other markers of health (plasma cholesterol, fasting glucose, blood pressure, adiposity, etc.) respond to exercise training when compensation occurs is not clear.

**Summary**

Commuter cycling is generally performed at moderate-intensity, which favours improved long-term health and fitness. On the other hand, the duration of commuter cycling is often performed below government-issued recommendations for weekly PA. Although participants may have meet PA recommendations with combined commuter cycling and leisure-time PA, the duration of leisure-time PA is only measured from one sample of participants (de Geus, et al., 2008, 2009). Furthermore, no existing research has monitored the effects of initiating a routine of commuter cycling on existing PA levels. Some cross-sectional evidence indicates that those who cycle for transport may have greater total PA compared to motorised transport users, however the quality of evidence is reportedly low (Wanner, et al., 2012). Previous authors utilising controlled trials did not report the effects of commuter cycling on existing PA, and in general, the effects of novel exercise on EE and PA in adults is seldom investigated. Research investigating the impact of novel PA on health outcomes need to monitor existing PA and EE to validate the load of PA has on total EE and the consequent changes to health (if any) from that load.
2.3 Body composition

Unhealthy waist to hip ratio (WHR) (> 0.95 for men, > 0.80 for women) is associated with increased risk for several ailments including cardiovascular disease, diabetes, and some forms of cancer (eg. Plodkowski & St. Jeor, 2003; Pitsavos, et al., 2007). A multitude of cross sectional and prospective cohort studies have resulted in mixed findings for associations between PA levels and body mass (eg. Williamson, et al., 1993; Schoeller, et al., 1997; Di Pietro, et al., 2004). Similar to the large population studies, intervention trials have also resulted in mixed results for the role of PA on body composition. (eg. Møller, et al., 2011; Lehmann, et al., 1997; Rice, et al., 1999; Yamamoto, et al., 2007). The discrepancy in results between studies may be due to compensation of reduced EE and/or increased energy intake with exercise training. For example, caloric intake has been observed to increase with physical activity (eg. Donnelly & Smith, 2005; King, et al., 2007). Thus, compensatory behaviour will diminish exercise-induced reductions in energy balance and consequent loss of body fatness and mass. Furthermore, without the use of advanced research instruments such as Dual-energy X-ray absorptiometry (DXA), gains in muscle mass may not be detected, adding further ambiguity to previous research.

**Body composition among active transport users**

It is unclear if physically-active transportation produces enough strain among users to reduce long-term energy balance or affect skeletal muscle mass. Interestingly, some evidence indicates that BMI is lower among those who use physically active transport
It is important to note however, that BMI does not directly measure body fatness or composition, but may allow for generalisations of body composition (Lee, et al., 1999). A prospective study by Gordon-Larsen et al. (2009) reported lower BMI in male participants that used active forms of transportation for an average of 20 mins/day, but not females, after a twenty-year follow-up. Authors commented that females performed significantly less (15%) active transport than males, which may explain the gender differences they observed (Gordon-Larsen, et al., 2009). Whereas, others also observed lower BMI among active transport users, in both genders (Hayashi, et al., 1999; Wagner, et al., 2001; Hu, et al., 2002). Although there isn’t complete consensus in the research literature on the role of habitual use of active transport and it’s effect on BMI, there is evidence to suggest it may be a useful mode of PA to enhance body composition, but the use of objective measures for body composition (i.e. WHR and DXA) are required.

In research specific to commuter cyclists, evidence for enhanced body composition is equivocal. Møller, et al., (2011) observed reduced skin fold thickness in cyclists after eight weeks of commuter cycling. Contextualising the results to other studies is hampered because the volume of CC performed was not measured. In a longer controlled trial, body mass and BMI were unchanged at the 26 week follow-up, and interestingly, de Geus et al. (2008) observed an increase over time in body mass among cyclists and a decrease in body mass in controls between weeks 26 and 52. Authors reported a decrease in the amount of time spent cycling between weeks 26 and 52,
which may explain the increase in body mass with cycling, but rationale for improvements among controls was not speculated on (de Geus, et al., 2008).

In summary, cross-sectional and longitudinal investigations have observed lower BMI among users of physically-active modes of transport when compared to those who use automotive transport; however, BMI is not a direct measure of body composition. Two controlled trials aimed to understand the role of CC on body composition and mass, where one reported an unexpected finding of increased body mass, and the other observed reduced body fatness, but the volume of cycling to achieve the result was unreported. Further confounding the evidence is the amount EE required from PA to improve body composition varies per individual due compensatory behaviours that result in increased caloric intake (e.g. Colley, et al., 2010) or decreased EE from other activities (Wickel & Eisenmann, 2006). Further research may enhance our understanding on the energy requirements of commuter cycling, the occurrence and degree of compensation in EE from other physical activities, and the effects commuter cycling has on muscle and fat mass by use of objective research tools (i.e. DXA).

2.4 Cardiovascular Diseases

Cardiovascular diseases (CVD) is a collection of diseases affecting cardiac muscle and blood vessels. The disease progresses with age, generally due to a habitually unhealthy lifestyle and the most serious acute events are myocardial infarction and stroke, caused
by atherosclerosis (Squires, 2005). Atherosclerosis is caused by chronic inflammation in the arteries because of dyslipidaemias, hypertension and other modifiable risk factors (e.g. Ross, 1986; Nabel & Braunwald, 2012). Prolonged presence of risk factors increases exposure of endothelial cells to oxidative, inflammatory, and biochemical stimuli (e.g. Furchgott & Zawadzki, 1980; Nabel & Braunwald, 2012). Through chronic exposure to CVD risk factors, endothelial cells change in permeability to allow entry of LDL cholesterol particles eventuating into lesions of plaque in the artery wall that obstructs blood flow in the artery (Furchgott & Zawadzki, 1980). When atherosclerosis results in occlusion of blood flow, nutrient and oxygen supply to the heart, brain or lower limbs can be compromised and result in a cardiovascular event (Squires, 2005).

For research groups, atherosclerosis is invasive and expensive to measure directly, therefore researchers often study the risks of developing atherosclerosis and CVD. Risk factors include unfavourable blood concentrations of low-density and high-density lipoprotein (LDL and HDL) cholesterol, high concentrations of triglycerides, C-reactive protein, fasting glucose and insulin, high blood pressure, smoking, significant distribution of adipose tissue around the abdomen, physical inactivity, among others (Kannel & Larson, 1993; Kannel, 1990; Gordon, et al., 2005). Of interest to this thesis research is how PA is either inversely correlated to the risk factors specified above, or has a direct protective effect (Shepard, et al., 1997; Pollock, et al., 1997; Verdaet, et al., 2004).
Epidemiological findings on the positive effects of exercise are also supported by data from robust experimental research trials (Braith & Stewart, 2006; Donnelly, et al., 2000; Jakicic, et al., 1999). However, exercise in previous research is generally supervised for intensity and adherence. Thus, the effects of exercise at a self-selected duration and intensity on CVD risk are less clear due to limited research that utilises free-living activities, opposed to laboratory-based exercise programs (Gray, et al., 2009). Exercise interventions in a free-living population have challenges with compliance and the volume of exercise performed is difficult to quantify. Commuter cycling is an example of a free-living PA where exercise duration is influenced by distance between participants' home and place of work, and intensity is self-selected and potentially influenced by motivation, traffic and weather conditions. Commuter cycling trials to date have seen improved fitness with cycling, but other, more robust predictors for CVD have not been observed. On the other hand, longitudinal research has reported less frequent incidences of CVD events and reduced risk for developing CVD among those people who habitually use physically-active modes of transportation (Hamer & Chida, 2008; Wang, et al., 2010). Although physically-active transportation appears to offer a protective effect against CVD, the research isn’t specific to commuter cycling, and longitudinal studies generally utilise research methods that are less adept than randomised controlled trials for establishing a cause and effect relationship due to unknown influences from covariates. To date, evidence for the role of commuter cycling on the reduction in CVD risk is sparse and necessitates further investigation.
2.5 Blood Pressure

High blood pressure (BP) is robustly correlated to risk of cardiovascular disease and every increase of 20/10mmHg of systolic/diastolic BP over 115/75mmHg doubles CVD risk (Chobanian, et al., 2003). Long-term high systolic blood pressure increases risk of CVD through damage to the inner arterial walls, which increases susceptibility to the entry of cholesterol and inflammation factors, resulting in the hardening of arteries (i.e. atherosclerosis) (Alexander, 1995; Chae, et al., 2001). In addition to the protective effects of exercise on atherosclerosis, as previously discussed, several have reported PA as an effective method to reduce blood pressure (BP) among normotensive and hypertensive individuals (e.g. Arroll & Beaglehole, 1992; Kelley & McClellan, 1994; Kelley & Tran, 1995; Halbert, et al., 1997). Favourable changes in BP from PA result from reduced systemic vascular resistance due to involvement of the sympathetic nervous system and renin-angiotensin system (Fagard, 2006; Guyenet, 2006). Reduced sympathetic tone lowers activity of arterial smooth muscle causing vasodilation of arterial endothelium and therefore reduced vascular resistance (Guyenet, 2006; Young, 2010). The reduction in vascular resistance is the primary mechanism for decreased arterial blood pressure at rest with regular exercise (DeSouza, et al., 2000; Tashiro, et al., 1993). Mechanisms for reduced BP appear responsive to both low and high-intensity PA (Roman, et al., 1981; Matsusaki, et al., 1992; Tashiro, et al., 1993; Rogers, et al., 1996). However, controlled commuter cycling studies have not observed lower BP among cyclists compared to control participants.
The effects of commuter cycling on BP have been investigated with an eight week controlled trial (Møller, et al., 2011) and a 52 week non-randomised trial (de Geus, et al., 2008). Diastolic BP was lower by ~4.6 mmHg after eight and 52 weeks of commuter cycling, but no changes were observed compared to controls (de Geus, et al., 2008; Møller, et al., 2011). In contrast to de Geus et al. (2008), Møller et al. (2011) also observed a decrease in systolic BP over time amongst cyclists, but again no group effect was present. Interestingly, Møller et al. (2011) reported lower BP among controls during the same intervention period and therefore it is unclear if changes over time with the cycling group were a result of the cycling or other factors (e.g. seasonal) that also affected the control group.

The lack of differences between cycling and control groups contradicts robust evidence for the role of PA on BP. Diastolic blood pressure appears most responsive to change with regular commuter cycling, but findings should be interpreted cautiously due to the lack of change between experimental groups. However, the reduction seen over time is an interesting finding that deserves further investigation.

2.6 Resting Heart Rate

Decreased resting heart rate (HR) is a reliable, non-invasive indicator for reduced risk for a cardiac event (i.e. sudden cardiac death, myocardial infarction), which has prognostic value of CVD risk independent of other risks factors (Smith, et al., 1989; Kannel, et al., 1987). Although the effects of PA on resting HR haven’t been
investigated in the context of physically active transport, robust evidence supports aerobic endurance training to reduce resting HR (Barnard, 1975; Scheuer & Tipton, 1977; Moore & Korzick, 1995). For example, interventions with aerobic exercise have resulted in significant reductions in HR of 3 BPM after 20 weeks (Wilmore, et al., 1996), 10 BPM (Byrd, et al., 1974) and 11 BPM (Edwards, 1974) after 12 weeks and 11 BPM in 10 weeks (Macial, et al., 1985). Endurance training in those studies was likely to expand plasma volume resulting in increased stroke volume of the heart, thus requiring less heart beats to maintain resting cardiac output (Convertino, et al., 1991). Furthermore, exercise training increases parasympathetic autonomic control and decreases sympathetic autonomic control (Smith, et al., 1989; Wilmore, et al., 1996).

Finer control of the autonomic nervous system decreases secretion of epinephrine and norepinephrine from the adrenal medulla, which in turn reduces heart rate (Young, et al., 2010). Resting HR is therefore an indicator of resting cardiac output as it relates to vascular resistance and stroke volume of the heart and a training effect is possible with regular aerobic exercise. There is however no known evidence that commuter cycling is generally performed at an intensity and frequency reduce resting HR.

More research should utilise resting HR as an outcome measure to enhanced knowledge of its effects with various types of exercise modalities and loads. With modest equipment and training, resting HR can be measured by the general public, empowering people to gain feedback on improvements to resting HR with exercise training, and awareness of cardiac function in comparison to the general population.
2.7 Aerobic capacity

Low cardiorespiratory fitness is defined by \( \dot{V}O_2 \text{max} \) of 25 ml/kg/min for people under 30 years of age and 16 ml/kg/min for people over 60 years of age (McArdle, et al., 2001). Low fitness is a well-established risk factor for all-cause mortality, particularly death from cardiovascular events (e.g. Paffenbarger, et al., 1986; Williams, 2001). Improved fitness can independently counteract the life-shortening effects of smoking, unhealthy body mass, genetic predisposition to early mortality and increase longevity in healthy adults (Paffenbarger, et al., 1986; Lee, et al., 1995). Further, fitness is a predictor of CVD risk of equal magnitude to other major risk factors such as smoking, unfavourable cholesterol and blood pressure (Powell, et al., 1987).

Cycling for recreation, sport and commuting consistently improves cardiovascular fitness (Oja, et al., 2011). Commuter cycling studies to date have resulted in improved \( \dot{V}O_2 \text{max} \) compared to control participants (Table 2.2). However, the role of commuter cycling to improve \( \dot{V}O_2 \text{max} \) within the cycling groups (i.e. within group change) is less clear (Table 2.2). The absence of within-group change for \( \dot{V}O_2 \text{max} \) with commuter cycling may be due to seasonal changes in fitness when testing was performed after winter (de Geus, et al., 2008), gender differences as a result one gender group had no change and may have precluded total group change (Hendriksen, et al., 2000), or no within-group statistics reported (Oja, et al., 1991).

Robust evidence supports commuter cycling as beneficial for improved cardiovascular fitness above controls. However, future controlled trials with commuter cycling should
include \( \dot{V}O_2\text{max} \) as an outcome measure to compare the effect (i.e. magnitude of change) in fitness as a basis to compare the effects for other outcome measures.

Table 2.7: Summary of studies on the improvements to \( \dot{V}O_2\text{max} \) with commuter cycling. Abbreviations: CYC (cycling), CON (control).

<table>
<thead>
<tr>
<th>Author / location</th>
<th>Study design</th>
<th>Sample size</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oja, et al., 1991. Tampere, Finland.</td>
<td>Randomised controlled trial; 10 wk follow-up.</td>
<td>ACTIVE group: n=26cyclists; n=39walkers. CON group: n=33</td>
<td>Cyclists higher ( (P = 0.01) ) than walkers. ACTIVE group higher ( (P = 0.02) ) than CON group. Change over time not reported.</td>
</tr>
<tr>
<td>Hendriksen, et al., 2000. Amsterdam, The Netherlands.</td>
<td>Randomised controlled trial; 26 wk follow-up.</td>
<td>CYC group: n=57 CON group: n=58</td>
<td>CYC group higher ( (P &lt; 0.01) ) than controls. Group change over time not reported, CYC group males increased ( (P &lt; 0.01) ) over time, but not females.</td>
</tr>
<tr>
<td>de Geus, et al., 2008. Brussels, Belgium.</td>
<td>Non-randomised intervention trial; 26 and 52 wk follow-up.</td>
<td>CYC group: n=72 CON group: n=15</td>
<td>CYC group higher ( (P &lt; 0.01) ) than controls at wk 52. N.C. over time.</td>
</tr>
<tr>
<td>Møller, et al., 2011. Funen, Denmark.</td>
<td>Randomised controlled trial; 8 wk follow-up.</td>
<td>CYC group: n=25 CON group: n=23</td>
<td>CYC group higher ( (P &lt; 0.01) ) than CON group and increased over time ( (P &lt; 0.01) ). CON group increased ( (P &lt; 0.01) ) over time.</td>
</tr>
</tbody>
</table>

2.8 Blood Cholesterol and Triglycerides

Unfavourable levels of plasma cholesterol and triglycerides are well established to increase risk of morbidity from heart disease (Rhoads, et al., 1976; Fuster, et al., 1996). Specifically, high concentrations of LDL cholesterol and triglycerides are correlated with increased risk of a coronary event, whereas high concentrations of HDL cholesterol are inversely correlated to risk of a coronary event (e.g. Rhoads, et al., 1976; Gordon, et al., 1977). Protective effects of exercise on cholesterol and triglyceride levels
are well established (e.g. Lloyd-Jones, et al., 2004; Mann, et al., 2014). The volume of PA to reverse unfavourable cholesterol and triglycerides depends on the duration and intensity of exercise, and existing levels of cholesterol and exercise habits (Mann, et al., 2014).

Short interventions (i.e. 12 +/- weeks) have produced mixed findings on changes to cholesterol and triglyceride levels (El-Sayed, 1996; Spate-Douglas & Keyser, 1999; Houmard, et al., 2004). Nevertheless, a review paper that included results from 13 meta-analyses reported that cholesterol and triglycerides can change favourably through habitual physical activity (Pedersen & Saltin, 2006). High-intensity PA is generally more effective to improve blood levels of cholesterol and triglycerides than lower intensity activities (Tambalis, et al., 2009). It could be that higher intensity aerobic PA increases energy expenditure and therefore skeletal muscles utilise a greater amount of lipids (Mann, et al., 2014). Compensation in EE with novel PA may not increase the amount of plasma lipids utilised by skeletal muscle and therefore cholesterol and triglycerides may remain unchanged. Change to LDL cholesterol with exercise may also improve through increased high-density lipoprotein (HDL) cholesterol (Shaw, et al., 2009; Mann, et al., 2014). The increase of HDL and associated enzymes (i.e. lecithin-cholesterol acyltrans and ester transfer protein) assists with removal of cholesterol from the blood and prevents HDL from being converted to other lipoproteins (Mann, et al., 2014). Data on the effects of PA on cholesterol in free-living individuals, whilst controlling for existing PA and EE habits is obscure and further research would
enhance knowledge on the practicality of using exercise to prevent and reduce high cholesterol in minimally supervised exercise programs.

**Effects of physically active transport on cholesterol and triglycerides**

Two cross-sectional studies measured the role of physically active transport on levels of blood cholesterol (Hu, et al., 2001; Gordon-Larsen, et al., 2009). Hu et al. (2001) observed favourable changes in triglycerides, HDL and LDL cholesterol among participants who actively commuted. Authors reported improvements to cholesterol were independent of leisure-time PA in their linear regression analysis, but the duration and intensity of leisure-time PA and active transportation were not reported (Hu, et al., 2001). Results published by Gordon-Larsen et al. (2009) were less supportive, and reported lower triglyceride levels among male participants, but not females (Gordon-Larsen, et al., 2009). Male participants were more physically active than females during the research period and were likely to have influenced gender differences in the results (Gordon-Larsen, et al., 2009). Furthermore, after adjustment for BMI, smoking and alcohol consumption, all cholesterol measures lacked statistical significance (Gordon-Larsen, et al., 2009).

Two intervention trials have measured blood cholesterol and triglyceride levels among commuter cyclists (Oja, et al., 1991; de Geus, et al., 2008). Oja et al. (1991) observed no change to triglycerides or LDL cholesterol, but a trend (i.e. $P = 0.06$) was observed for increased HDL cholesterol between physically active commuters and controls after 10 weeks. Specific results for the effects of blood parameters from commuter cyclists were
not reported. On the other hand, de Geus et al. (2008) reported improved blood levels of total cholesterol, LDL cholesterol, HDL cholesterol, but not triglycerides after 26 weeks of commuter cycling. However, improved blood parameters were only significant over time, not between the cyclists and the non-randomised control group. Repeat measures of cholesterol at week 52 did not indicate any further improvements from measures at week 26 (de Geus, et al., 2008). The lack of change between cycling and control groups may be due to unexpected and significant decrease in total and LDL cholesterol among controls during the intervention period (de Geus, et al., 2008). The study had methodological shortcomings including a non-randomised group selection, inclusion of office workers only, and post-intervention testing took place after a significant decline in PA and commuter cycling, potentially due to Northern European winter conditions.

**Summary**

Current evidence for improved blood cholesterol with physically active transport is limited by methodological inconsistencies in cross-sectional and controlled intervention trials. Cross-sectional research has resulted in mixed effects for improved blood parameters amongst physically active transport users. Discrepancies in the findings may have resulted from imprecise measures of PA, such as an unknown contribution of walking versus cycling for transport and unknown physiological intensity of participants’ PA. In the very few controlled commuter cycling studies to date, cholesterol has not been observed to change in cyclists compared to controls and
it is unclear if the physiological strain of commuter cycling is insufficient to cause change in cholesterol, or if the aforementioned short comings in previous research efforts has hampered the effects of the cycling intervention. Due to this scarcity of randomised controlled studies on the topic, further study is necessary for consensus on the role of commuter cycling for potential benefit in blood levels of cholesterol.

2.9 C-reactive protein

Although blood cholesterol levels are a well-established method for predicting CVD risk, some research indicates half the individuals who experience a cardiac event have normal lipid values (Ridker, et al., 2002). C-reactive protein (CRP) on the other hand, independently predicts risk of CVD and has demonstrated greater reliability than cholesterol alone for predicting CVD events (Ridker, et al., 1998; Ridker, et al., 2002). Combined cholesterol fractions with CRP in medical screening further enhances the predictive power for CVD (Ridker, et al., 2002).

Infection and tissue damage result in an acute-phase, innate immune response, which increase some analytes (i.e. C-reactive protein (CRP), interlukin-6 (IL-6), fibrinogen) whilst others decrease (i.e. transferrin and iron) (Young, et al., 1991; Thompson, et al., 1992; Gambino, 1997). Increases in analytes such as CRP are used as a clinical measure of an immune response to injury or infection. C-reactive protein is a reliable inflammation marker for clinical use due to the quick onset and high magnitude of its
release from the liver in response to tissue damage (Kushner, et al., 1978; Morley & Kushner, 1982). In addition to the liver, CRP is also produced in smooth muscle, such as the muscle in arterial walls in response to inflammation (for review, see Bassuk, et al., 2004; Plaisance & Grandjean, 2006). C-reactive protein is hypothesised to directly contribute to the formation of arterial lesions (Bassuk, et al., 2004). Specifically, chronic exposure to CRP may increase expression of endothelial surface adhesion molecules (Pasceri, et al., 2000), reduce nitric oxide bioavailability resulting in reduced vasodilation function (Venugopal, et al., 2002), increase LDL uptake by macrophages that accelerate formation of arterial lesions (Zwaka, et al., 2001), among other mechanisms (Bassuk, et al., 2004). Therefore, CRP is the leading biochemical marker for estimating CVD risk due to its direct and indirect role on arterial inflammation (Gordon, et al., 2005).

The role of physical activity and weight loss on CRP

Physical activity interventions that decrease body mass, waist circumference or percentage body fat often observe reduced CRP (Okita, et al., 2004; Martins, et al., 2010; Donges, et al., 2010). The mechanism for reduced CRP through improved body composition is supported by the hypothesis that IL-6 released from adipose tissue promotes the release of CRP (Bastard, et al., 1999). Therefore, reduced body fat will result in a lower circulating concentration of IL-6 and consequently less CRP.

Okita et al. (2004) observed significantly lower body mass and CRP in participants after eight weeks of PA between 60 to 80% of maximal heart rate (HR$_{max}$). Similarly, Donges
et al. (2010) implemented an aerobic exercise intervention at 70% HR_{max} that resulted in reduced waist circumference compared to controls, reduced body fat within the exercise group and a trend (i.e. $P = 0.06$) towards reduced CRP in 10 weeks. Another group of participants from the aforementioned study underwent the same aerobic program in addition to a resistance exercise program, which resulted in significantly lower CRP (Donges, et al., 2010). In contrast, others have not observed changes to CRP despite exercise induced changes to body composition. For example, a six month intervention of aerobic training at 50% of maximal heart rate reduced participants’ body mass while CRP remained unchanged (Arsenault, et al., 2009). Another study resulted in decreased body fat and body mass after 16 weeks of moderate-intensity or high-intensity training, with no significant effects to CRP (Marcell, et al., 2005). As previously discussed, inflammation induces a large and quick increase in CRP, and therefore the variability of the CRP levels may fluctuate from day to day; best practice for measuring CRP in prolonged exercise trials is not yet clear.

One study monitored participants’ CRP levels during a moderate-intensity, commuter cycling intervention (de Geus, et al., 2008). The intervention resulted in no change to body composition measures or CRP, compared to controls at 26 and 52 week follow-up (de Geus, et al., 2008). The results were consistent with a previous study from a 26 week intervention consisting of moderate aerobic PA (Hammett, et al., 2004).
Summary

Cross-sectional investigations provide evidence for an inverse relationship between cardiovascular fitness and total physical activity on CRP levels (Abramson & Vaccarino, 2002; Panagiotakos, et al., 2005). Despite this, current evidence from randomised controlled trials generally indicates that exercise training is more likely to enhance CRP with concurrent loss of body mass, particularly from adipose tissue. Interestingly, weight-loss without change to PA habits has been reported to decrease CRP (Plaisance & Grandjean, 2006). Nevertheless, reduced CRP without improved body composition is generally limited to untrained or diseased populations such as CVD patients (Milani, et al., 2004; Goldhammer, et al., 2005) post-menopausal, breast cancer survivors (Fairey, et al., 2005) and elderly participants (Kohut, et al., 2006). Evidence for PA induced reductions in CRP without changes to body composition is sparse among younger and disease-free populations. Given the strong prognostic value of CRP as a leading biochemical marker of cardiovascular health, further study is warranted to enhance knowledge on the role of exercise to preclude adverse levels of CRP in seemingly healthy adults.

2.10 Impaired glucose management

Impaired glucose management is a condition by which blood levels of glucose are abnormally high due to resistance of insulin in peripheral cells and/or a lack of insulin
production from beta cells in the pancreas (Muniyappa, et al., 2008; Antuna-Puente, et al., 2011). This condition is clinically defined as fasting glucose levels ≥7.0 mmol/L or ≥126mg/dL (Alberti & Zimmet, 1998; McAuley, et al., 2001). Robust evidence supports the role of impaired glucose management in the progression of insulin resistance and type-2 diabetes, which increases risk for CVD. For example, insulin resistance is associated with dyslipidaemia, myocardial infarction, and hyperuricemia (Pyörälä, et al., 1987; Lillioja, et al., 1988; Stamler, et al., 1993; Wingard & Barrett-Connor, 1995; Haffner, et al., 1998). A variety of risk factors contribute to the development insulin resistance in adults. Age and genetic predisposition are non-modifiable risk factors, whereas obesity, accumulation of adipose tissue around the abdomen (android distribution), poor diet and physical inactivity are risks that can be reduced through behaviour change (eg. Ferrannini, et al., 1990; Wang, et al., 1989; Wareham, et al., 2005). Physical activity is indisputably beneficial for people who have impaired blood glucose are insulin management (e.g. Sigul, et al., 2006). Exercise training enhances glucose management through several mechanisms including increased skeletal muscle dependence on lipids through enhanced mitochondrial density and greater capacity for beta-oxidisation, which spares glycogen (O’Gorman & Krook, 2008; Sigal, et al., 2006). Sensitivity of insulin is enhanced through increased signalling in skeletal muscle as well as improved glucose transport predominantly through increased concentrations of Glut-4 enzyme with frequent exercise (O’Gorman & Krook, 2008; Sigal, et al., 2006).

The volume of exercise required by apparently healthy and normal-weight adults to reduce risk of insulin resistance is not very clear due to surprisingly few studies that
have investigated exercise training on glucose or insulin management in this population. Aerobic exercise training improved glucose and insulin dynamics in obese men with 100-150 min/wk of PA in 16 weeks (Leon, et al., 1979; Rice, et al., 1999) and in middle aged women with 12 weeks (Yamamoto, et al., 2007), and older obese men with combined aerobic and resistance exercises (Davidson, et al., 2009). Only one aforementioned study utilised the gold-standard method for determining insulin resistance, a euglycemic insulin clamp (Davidson, et al., 2009). The variety of methodology used by others (e.g. OGTT, HOMA, HbA1c) challenges efforts to directly compare results between studies, nevertheless, aerobic exercise appears to be beneficial in obese and in middle to older aged adults.

In general, people who are insulin resistant can improve management of glucose and insulin through regular PA and the adaptations that improve insulin resistance have also been observed in healthy adults (O'Gorman & Krook, 2008). However, comparatively few have investigated insulin and glucose dynamics with exercise training in a healthy adult population. the intensity of the PA performed may be a determining factor in preventing insulin resistance in young adults. High-intensity PA has been demonstrated to have a greater effect on insulin and glucose dynamics (Seals, et al., 1984; Tjønna, et al., 2008), albeit others have reported that intensity was of less importance when controlling for total energy expenditure (Hansen, et al., 2009).
Diabetes risk among physically active transport users

Two studies were conducted to assess risk for developing type-2 diabetes among physically active transport users (Gordon-Larsen et al., 2009; Hu et al., 2003). In a cross-sectional investigation, Gordon-Larsen et al. (2009) reported lower fasting insulin measures among men, but not women who actively commuted. However, their analysis did not control body composition, which is correlated with glucose management (Meigs, et al., 1998). In their prospective cohort study, Hu et al. (2003) reported reduced type-2 diabetes prevalence among those who use physically active transport. When controlling for gender, the effect remained significant for women, but not men. But gender differences diminished when BMI was controlled for (Hu, et al., 2003). To date, no known study has investigated blood glucose and insulin dynamics exclusively amongst habitual commuter cyclists, or in an intervention that promotes the new use of cycling for transport.

Summary

A strong association between risk for developing diabetes and risk for CVD has been established (Stern, et al., 1985; Rodriguez, et al., 1996). Epidemiological research suggests active commuting may reduce risk for developing type-2 diabetes (Gordon-Larsen, et al., 2009; Hu, et al., 2003). However, direct risk factors for type-2 diabetes (i.e. insulin sensitivity / resistance) have not been investigated with commuter cycling. Interventions utilising other forms of PA have observed enhanced fasting insulin and glucose in adults. Thus, future research is warranted to clarify the relationship between
PA, insulin and glucose to establish whether commuter cycling is sufficient to reduce risk of developing insulin resistance and associated health impairments such as metabolic and cardiovascular diseases.

2.11 Quality of Life

Physical activity is correlated with improved mental health (Glenister, 1996). Specifically, PA increases emotional well-being and decreases anxiety and depression compared to sedentary people (Salmon, 2001). The commonly used Short Form 36 questionnaire uses eight components to define and measure QOL in a health-related context, which includes vitality, physical functioning, mental functioning, bodily pain, general health perceptions, mental health, emotional functioning and social functioning (Ware Jr. & Sherbourne, 1992).

Cross-sectional research has consistently demonstrated a correlation between time engaged in PA and health-related QOL. That relationship is described by a bell-shaped curve in which those who exercise the least and the most have inferior QOL to those in the two middle quartiles of time engaged in PA (Stewart, et al., 1993; Brown, et al., 2003; Vuillemin, et al., 2005). Longitudinal studies generally support data from cross sectional studies, albeit evidence is less consistent (e.g. Partonen, et al., 1998; Malmberg, et al., 2005; Brand, et al., 2006; Tessier, et al., 2007). The longitudinal trials
however, have been criticised for a lack of instrument validation, inappropriate analyses and inadequate randomisation procedures (for review, see Bize, et al., 2007).

**Quality of life and commuter cycling**

In a non-randomised controlled trial, de Geus et al. (2008) explored the effects of 12 months of moderate-intensity CC and leisure time PA over 150 min/day on health-related QOL as measured by the Short Form 36 (Ware Jr. & Sherbourne, 1992). Cycling participants reported increases in Vitality and role limitation due to physical health over time, but not between groups, whereas Physical Functioning was enhanced over time and above controls (de Geus, et al., 2008). Cycling and control groups differed in some QOL variables at baseline, which decreases the internal validity of their between-group QOL analyses (de Geus, et al., 2008). Nevertheless, within-subject improvements to QOL variables with commuter cycling are encouraging, but require confirmation.

**Summary**

People with habitual PA routines generally have lower occurrence of anxiety and depression. Some longitudinal studies support the role of PA on enhanced mood, but not all. Quality of life instruments also vary in quality between studies, further confounding generalisations on the role of PA and QOL. In the one study that measured QOL with commuter cycling, cycling enhanced some QOL measures, but cycling and control groups were different at baseline, which obscure the effects of commuter cycling on QOL parameters (de Geus, et al., 2008). Although acute increases in mood
with exercise are well established, whether a training effect can be observed in healthy people is less clear, and data amongst commuter cyclists are few.

2.12 Conclusion

The role of PA to reduce risk of CVD is indisputable. Even short-term PA interventions improved risk factors for CVD, however mixed findings exist for enhanced body composition, insulin resistance and C-reactive protein in healthy populations. Different initial values for parameters of health amongst participants in previous studies, as well as the different load and duration of PA, may have influenced the contrast in results between studies. This is particularly true for moderate-intensity PA trials, which are less likely to improve indices of health compared to high-intensity PA. Although both moderate and high-intensity PA is beneficial for health and longevity, trials with high-intensity PA enhances health parameters in a shorter duration.

The self-selected pace for commuter cycling is generally at a moderate-intensity (~60\% \text{\dot{V}O}_\text{max}) and performed for less than the public health recommendation of 150 min/wk of moderate PA. However, commuter cycling may assist with meeting public recommendations for PA when combined with existing leisurely PA and/or occupational PA. The role of commuter cycling on existing PA is unknown and largely under reported in research on healthy populations.
Epidemiological and cross-sectional research indicates a reduction of risk for CVD mortality and favourable changes to CVD risk factors amongst people who habitually use physically active modes of transportation. Short and long-term interventions of commuter cycling support epidemiological findings for cardio-respiratory fitness, with inconclusive findings for other parameters such as body composition, blood pressure, inflammation and cholesterol. Resting heart rate and fasting insulin have not been investigated in the context of commuter cycling.

Barriers that prevent generalisations of which health parameters are likely to change with commuter cycling are due to a lack of repeatability in the parameters of health monitored, as well as varying duration of commuter cycling trials (i.e. 8 wk vs 52 wk). Generalisations of the benefits from commuter cycling are further confounded by the lack of control for existing levels of PA and EE before and during research studies. The role of novel commuter cycling on total daily PA and EE has not been investigated. Health parameters may have only improved minimally in current trials, in contrast to interventions with different exercise modes, due to a relatively small contribution of commuter cycling on total levels of EE and PA. Or it may be that commuter cycling doesn’t offer enough cardiovascular strain to enhance major CVD risk factors; only further investigation will enhance knowledge on this form of PA.

Research on how commuter cycling may reduce risk for CVD, improve body composition and QOL is not yet complete. Further research is warranted to repeat and support existing findings and explore other health parameters that have not previously
been studied in the context of commuter cycling (i.e. insulin/glucose dynamics and resting heart rate). In doing so, a deeper understanding may develop for the contribution of commuter cycling towards improved health.
CHAPTER THREE: Research Methods

3.1 Overview

To enhance knowledge of the health gains that may be achieved by introducing commuter cycling to the daily routine, a quasi-randomised controlled study was undertaken to evaluate effects of a 10 week commuter cycling intervention. Twenty-eight men and women who commuted by motorised transport were allocated to cycling and control groups. Those in the cycling group were provided with bicycles and cycled to and from work for a minimum of 100 min/wk for 10 consecutive weeks, whilst the controls were asked to continue their existing transportation and lifestyle habits, and were provided with public transport or petrol vouchers. Existing PA and EE was monitored before and throughout the 10 weeks. Markers of cardiovascular health, body composition and quality of life were measured before and after the intervention in cyclists and controls. Furthermore, the intensity and caloric expenditure of commuter cycling were also measured. This study was approved by the University of Otago Human Ethics Committee, reference number 10/153.

3.2 Power Analysis

Two power analyses, to determine sample size, were conducted using published studies that implemented interventions similar in duration and physiological intensity as the one proposed. Maximal oxygen consumption ($\dot{VO}_2$ max) as a measure of aerobic fitness was chosen as a primary outcome variable as it is highly correlated with reduced
cardiovascular disease risk and reduced morbidity. Thus, the first power analysis was performed on this variable and expected changes over this time period. The paired-sampled analysis was conducted assuming an increase in $\dot{V}O_2$max of 14%, standard deviation of 15% and a power of 0.80 indicated a sample size of 13 in each group was necessary (Kukkonen-Harjula, et al., 1998).

A second power analysis was conducted on high-density lipoprotein (HDL cholesterol), which is also correlated with cardiovascular health and expected to change with increased PA. A paired-sample analysis with an increase of 33%, and standard deviation of 27% and a power of 0.80 indicated a sample size of 14 participants per group (Spate-Douglas & Keyser, 1999). Therefore a minimum of 28 volunteers were sought for participation in this study.

3.3 Participant selection

A questionnaire was distributed to prospective participants who responded to fliers and emails advertising the study. The questionnaire assessed physical activity readiness and lifestyle factors associated with their current work, travel and PA habits (Appendix I). Exclusion criteria included smoking, active commuting, unemployed or working from home, a BMI greater than 30, under the age of 20 or over the age of 55, or participating in a structured training program, training to compete in sport and contraindications to exercise as defined in the Physical Activity Readiness Questionnaire (Thomas, et al.,
ninety-six individuals applied for the study and 33 (18 men and 15 women) met the inclusion criteria and were invited to pre-intervention testing.

3.4 Testing overview

Eligible participants were invited to the School of Physical Education research laboratory for two testing sessions prior to the intervention and those allocated to the cycling group underwent a third session at the Dunedin Public Hospital (Figure 3.1). In response to the invitation, one male declined and one female discovered she was pregnant and withdrew from the study. Measures taken at the first testing session were quality of life (QOL) questionnaire, skin-fold thickness, incremental VO₂max with a cycle ergometer and power output at VO₂max. Thirty one people (17 male, 14 female) participated in the first testing session. One male did not complete the VO₂max test and another was excluded from participation upon disclosing plans for overseas travel during the intervention period. Twenty nine people (15 males, 14 females) completed the first testing session and were invited to the second testing session. Upon group allocation to cycling (CYC) and control (CON) groups, one male withdrew from the study. Twenty-eight participants (14 male, 14 female) underwent the second testing session, which involved a physical activity questionnaire, resting measures of heart rate and blood pressure, blood sample, and instructions for completion of diet records and PA log books. All 28 participants completed the second testing session. The 14
participants in CYC underwent a third testing session at the Dunedin Public Hospital for a dual-energy x-ray absorptiometry (DXA) scan.

<table>
<thead>
<tr>
<th>Time period</th>
<th>Measures</th>
</tr>
</thead>
<tbody>
<tr>
<td>14 – 8 days before intervention</td>
<td>QOL, Skinfolds, ( \dot{V}O_2_{\text{max}} )</td>
</tr>
<tr>
<td>7 – 1 days before intervention</td>
<td>wk 0 Resting HR &amp; BP, WHR, blood sample, IPAQ, fasted BM, diet record, log book, DXA (CYC only)</td>
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<tr>
<td></td>
<td>wk 1 Log book, fasted BM</td>
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<td>wk 2 Log book</td>
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<td></td>
<td>wk 3 Log book</td>
</tr>
<tr>
<td></td>
<td>wk 4 Log book, fasted BM, diet record, IPAQ, HR sampling (CYC only)</td>
</tr>
<tr>
<td></td>
<td>wk 5 Log book, fasted BM, sub-maximal ( \dot{V}O_2 ) cycle test (CYC only)</td>
</tr>
<tr>
<td></td>
<td>wk 6 Log book</td>
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<td></td>
<td>wk 7 Log book</td>
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<tr>
<td></td>
<td>wk 8 Log book, fasted BM, diet record, IPAQ, HR sampling (CYC only)</td>
</tr>
<tr>
<td></td>
<td>wk 9 Log book, fasted BM</td>
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<tr>
<td></td>
<td>wk 10 Log book</td>
</tr>
<tr>
<td>1 – 7 days after intervention</td>
<td>Resting HR &amp; BP, WHR, blood sample, IPAQ, fasted BM, DXA (CYC only)</td>
</tr>
<tr>
<td>8 – 14 days after intervention</td>
<td>QOL, Skinfolds, ( \dot{V}O_2_{\text{max}} )</td>
</tr>
</tbody>
</table>

*Figure 3.1: Timeline of data collection. Abbreviations: wk (week), QOL (quality of life), \( \dot{V}O_2_{\text{max}} \) (maximal oxygen consumption), HR (heart rate), BP (blood pressure), WHR (waist-to-hip ratio), IPAQ (international physical activity questionnaire), BM (body mass), DXA (Dual-energy X-ray absorptiometry), CYC (cycling group).*
3.5 Preliminary testing measures

The preliminary testing session took place between fourteen and eight days before the intervention (Figure 3.1). At least two days before the first testing session, participants were informed of the testing procedures and preparedness for the test (i.e. appropriate clothing for exercise, hydration, no meals within 2 hours of testing, abstain from vigorous or otherwise exhausting PA the day prior). Upon arrival to the lab, participants completed an informed consent to participate.

Health-related quality of life

Health-related QOL was assessed by the Short Form 36 questionnaire (SF-36, Ware & Sherbourne, 1992). The questionnaire measured eight multi-itemed variables for physical, social and mental functioning. The variables include: ten-item physical functioning, two-item social functioning, four-item role limitations due to physical problems, three-item role limitations due to emotional problems, five-item mental health, four-item mental energy and vitality, two-item pain, five-item perception of general health, and a single-item question on changes to health in the previous year.

Skin folds

Skinfold thickness was measured at four sites (triceps, biceps, sub-scapular and suprailiac) to the nearest 0.2mm with John Bull callipers (British Indicators Ltd., St Albans, England). Measures were performed in duplicate and measures with variability over one millimetre were repeated a third time and the closest two measures used. Body
density and percentage body fat were calculated with formulas from Durnin & Womersley (1974).

*Maximal oxygen consumption*

An incremental exercise test to exhaustion was performed on a Velotron Elite cycle ergometer (RacerMate Inc., Seattle, Washington, USA) to determine $\dot{V}O_2$ max and maximum power (watts). A silicon face mask (Hans Rudolph, Inc, Kansas City, USA) was used to capture respiratory gases, measured breath-by-breath with open circuit spirometry (Cosmed Cardio Pulmonary Exercise Testing, CosmedSrl, Rome, Italy) and averaged into 30 s blocks.

The protocol for the cycle test started with a five minute warm up at 75 w with a pedalling cadence between 80-95 revolutions per minute (rpm). Visual and verbal feedback was used to assist with maintaining pedalling cadence. The protocol started at 100 w and increased in 50 w increments every three minutes until a heart rate (HR) of 165 BPM was achieved. After 165 BPM, stages were increased in 25 w increments until exhaustion, defined by plateau in $\dot{V}O_2$ with increased workload. If a plateau was not observed, exhaustion was defined as inability to pedal greater than 60 rpm in addition to one of the following criteria: RER of $\geq$ 1.1 or maximal age-predicated HR ($\pm$ 10 BPM). A warm-down was then performed on the cycle ergometer for a minimum of five minutes. For safety purposes, participants stayed at the laboratory until HR was below 100 BPM.
If participants were unable to complete the VO2 max protocol, a second attempt was permitted before exclusion from the study, which took place prior to the start of the second testing session. Two females completed the test on second attempt in which the resistance on the cycle ergometer was reduced to a 50 w warm up, with the first stage at 75 w and 25 w increases every three minutes until exhaustion, as defined above.

3.6 Second testing session

Overview

The second testing session took place in the seven days prior to the intervention (wk 0, Figure 3.1). Participants were encouraged to select an appointment time between 6:30am and 10am to suit their normal morning routine and were asked to arrive at least 48 hours after their last bout of moderate or high intensity exercise, at least 24 hours from any exercise, at least a 24 hour fast from alcohol and 12 hour fast from food and caffeinated beverages. Upon arrival to the laboratory, body mass and waist and hip circumferences were measured, participants were then fitted with a recordable HR monitor and seated to complete a physical activity questionnaire, followed by rest in the supine position. Blood collection consumables were hidden from participants’ view until BP and HR measures were collected. While participants remained supine, blood collection was performed. Upon completion of testing, participants were provided with breakfast and were instructed on procedures for completing a four-day weighed food record and log book (if not already advised).
International Physical Activity Questionnaire

Physical activity was assessed with the short form version of the International Physical Activity Questionnaire (IPAQ). In a systematic review, total PA assessed by the short form IPAQ was correlated between 0.09 and 0.39 to objective measures of PA, whereas vigorous and moderate intensity PA had a stronger, albeit more variable correlation (-0.18 and 0.76) compared to objective PA measures (Lee, et al., 2011).

Body composition and mass measures

Fasted body mass, waist and hip circumferences were performed after a 12 hour fast from food. Participants were instructed to void and asked to remove excess clothing before body mass measure on electronic scales (Model 770, Seca alpha, Hamburg, Germany). Participants were clothed for waist and hip circumference measures, which were performed in duplicate by the same technician for pre and post-intervention testing. Pre-intervention results were blinded from the technician during post-intervention testing.

Resting blood pressure

Systolic and diastolic BP was manually recorded for the first and fifth Korotkoff sounds from the antecubital artery. Blood pressure measurements were performed in duplicate with a mercury sphygmomanometer (Nihon Seimitsu Sokki Co. Ltd., Gunma, Japan) and stethoscope by the same technician for all participants during pre and post-
intervention testing. Pre-intervention results were blinded from the technician during post-intervention testing.

*Resting heart rate*

Resting heart rate was recorded with a Polar RS400 (Polar Electro Inc., Kempele, Finland) throughout 15 min of supine rest. Data was exported to Microsoft Excel for Windows (2010) where HR values were averaged into 15 s blocks, and the lowest value was recorded.

*Blood collection*

Plasma samples for fasting insulin, glucose, triglycerides, cholesterol (HDL and LDL), and C-reactive protein (CRP) were collected after resting HR and BP measures. A technician drew blood from an antecubital vein and collected in plastic 6 ml BD vacutainers (BD New Zealand, Auckland, New Zealand) with 10.8mg K$_2$ EDTA coagulant. Vacutainers were gently inverted eight times, then immediately spun in a ~4°C centrifuge (Beckman GS-15R, Beckman Instruments, Solna, Sweden) at 3000 rpm for 10 min. Plasma was then extracted from the vacutainer into four micro centrifuge tubes in the following amounts: 200 $\mu$L for glucose, 400 $\mu$L for insulin, 350 $\mu$L for CRP, 500+ $\mu$L for cholesterol and triglycerides. Plasma samples were frozen at -80°C and analysed in one batch after post-intervention samples had been collected.
Food records

Estimated energy intake and macro nutrient intake was assessed by weighed food records at weeks zero, four and eight. Participants were provided with verbal and written instructions, food scales, and portion-size picture book to measure total food intake for three weekdays and one weekend day in the seven days prior to the intervention. Fasted body mass was measured before and after the four day food dairy. Although food records were kept by both participant groups, only CYC results were intended for analysis. CON performed the food records to ascertain any effects of keeping a food diary, which has previously been shown to affect eating behavior (Poslusna, et al., 2009). We acknowledge that the analyses of EE estimates would be more robust if performed on both groups. However, availability of human resources prevented data entry of food records from CON. Energy and macronutrient intakes of CYC were assessed with Diet Cruncher Software (Way Down South Software, Dunedin, New Zealand).

Physical activity log books

Time engaged in physical activity prior and throughout the intervention was recorded with a daily PA log book. Participants were provided with log books and access to a website specific to this study, depending on their preference, to record PA information. To establish baseline PA levels, a daily log was kept by all participants on the duration of light activities lasting 10 or more minutes, and moderate and vigorous-intensity PA lasting at least five minutes in duration, for one week prior to the intervention (wk 0).
and throughout the study. To improve reliability of perceived intensity recorded in activity diaries, log books including definitions and examples for activities of moderate and vigorous-intensity, which was adapted from the validated IPAQ.

3.7 Group allocation

Twenty-eight participants were randomised into CYC and CON with stratification for gender and $\dot{V}O_2\text{max}$. Each participant was paired with one other participant of the same gender with a similar $\dot{V}O_2\text{max}$ (i.e. $\pm 3 \text{ ml} \cdot \text{kg} \cdot \text{min}^{-1}$) and randomly split into CYC and CON.

3.8 Third testing session

Participants allocated to CYC underwent DXA scans at the Dunedin Public Hospital before and after the intervention. Lean and fat masses were determined for the whole body, as well as for legs, arms, trunk, waist and hips using methods from Taylor et al. (1998). Funding for DXA was adequate for 28 scans only and statistical power was insufficient to perform scans on a subset of participants from both groups at both time points. Therefore DXA scans were performed on CYC before and after the intervention because most change to body composition was expected in this group.
3.9 Bicycle safety, maintenance and route selection

Each participant in the intervention group was met with individually to discuss road rules regarding bicycle use and common hazards from various road surfaces and road users. During the session, participants were provided with an information pack which outlined the bicycle function and road rules. A discussion of potential route choices followed, including an action plan for the days they intend to cycle and a backup plan if a scheduled active commute was missed. Participants were instructed to report the occurrence of illness during weekly phone calls / emails with research assistant, if A) was a cause for not meeting the minimum 100 min/wk of cycling or B) if it was a week of data collection (e.g. body mass and food diaries wks 4 and 8, submaximal testing wk 5, or testing sessions before/after the intervention). Additionally, strategies for managing weather, clothing choices and transport of work materials (i.e. business suits, briefcases, text books etc.) were discussed and questions answered.

During the third week of the intervention period, two bicycle maintenance clinics were conducted to support participants in learning how to replace a bicycle tube, perform a safety check and answer any bicycle maintenance questions.

3.10 Intervention

Initiation of commuter cycling was staggered based on timing of recruitment, testing and availability of lab equipment, with six females and one male starting seven days after the other seven CYC participants. CYC were asked to cycle commute at least 20
min/day, 3-5 days/wk at a self-selected intensity for 10 consecutive weeks. Participants were encouraged to accumulate at least 150 min/wk of commuter cycling. However, a minimum ride time of 100 min/wk, accumulated on three or more days was necessary to remain in the project. Participants were allowed one week of less than 100 minutes of cycling (e.g. illness, travel) but were made up by adding an extra week to the intervention. CYC were provided with bicycles, high-visibility wet-weather clothing, front and rear bicycle lights, tube repair kits, and a portable tire pump, but supplied their own helmet. Correspondence with participants took place weekly to ensure adherence and completion of PA log books and offer any assistance required by the participants. Correspondence was performed in person when data was collected on weeks four, five, seven and eight. Remaining contact took place by email or phone, based on the participants’ preference. As reward for participation in the study, participants in the CYC were given the bicycles and accessories provided to them for the intervention.

CON followed the intervention period of the first seven CYC participants. CON were asked to maintain their present transport, physical activity and dietary habits but were encouraged to record any changes they made into their PA log book/website entries. Reward for participation was $150 voucher for their preference of petrol or public transportation.
3.11 Intermediate measures during the intervention period

Levels of physical activity, food intake, body mass and HR sampling were monitored at regular intervals during the intervention (*Figure 3.1*).

*Monitoring physical activity*

The IPAQ (short form) administered at week zero, was repeated weeks four and eight. The questionnaire was implemented in pencil and paper form at the research laboratory.

*Monitoring dietary intake*

The four-day weighed food record performed week zero, was repeated on weeks four and eight. Body mass measurements were performed before and after completion of each four-day food record, within a seven-day window.

*Heart rate sampling*

CYC were instructed to wear a downloadable HR monitor (Polar RS400, Polar Electro Inc., Kempele, Finland) during all waking hours on week four and for each one-way cycle commute on week eight. Heart rate data for their commuting activities was extracted from the data set to determine the relative work rate (percent of maximum HR and VO₂max) of commuter cycling.
Sub-maximal cycling test

At week five, CYC performed an incremental exercise test on a cycle ergometer to determine steady state oxygen consumption at various cycling intensities. These data were used to estimate energy expenditure of commuter cycling based from HR data collected at weeks four and eight. Equipment used for the test was the same as described for the VO₂max test in the first testing session.

Participants were fitted with HR monitors and warmed up for 5 minutes with a self-selected cadence between 50 to 100 W of resistance, depending on fitness level. For warm up, participants were instructed to use a pedalling cadence similar to their normal bicycling habits and were instructed to maintain the same cadence throughout the sub-maximal test. A visual representation of their real-time cadence was present on a computer screen in front of the cycle ergometer.

The exercise test started at 40% of VO₂max and increased in 10% increments until termination at 80% of VO₂max. Resistance on the cycle ergometer corresponding to targeted percentage of VO₂max was calculated based participants’ power output from their pre-intervention VO₂max test. Each workload was performed for five minutes or until a steady state had been achieved for 60 s. Respiratory gases were monitored continuously throughout the sub-maximal test for total oxygen consumption and for respiratory exchange ratio.
3.12 Post-intervention testing

The three testing sessions performed by participants prior to the intervention were repeated upon completion, with replication of the procedures and in the order that the earlier tests were implemented (Figure 3.1). Measures of PA and diet habits were excluded from post-intervention testing as they were monitored to ascertain changes between baseline and the intervention period.

3.13 Analyses

Blood measures

Plasma samples of C-reactive protein, total cholesterol, HDL cholesterol and triglycerides were analysed with COBAS Mira Plus analyser (Roche Diagnostic, Basel, Switzerland). Participants’ pre and post-intervention samples were analysed in the same batch. LDL cholesterol was determined by Friedewald equation, where cholesterol values are expressed as mmol/L (Friedewald, et al., 1972).

\[
LDL \text{ Chol.} = Total \text{ Chol.} - (Triglycerides \div 2.18) - HDL \text{ Chol.}
\]

Glucose was analysed in duplicate with a Cobas c 111 (Roche Instrument Centre, Rotkreuz, Switzerland) and insulin was analysed with electrochemiluminescence method using analyser Elecsys2010 (Roche/Hitachi, Tokyo, Japan). Homeostatic model
assessment for insulin resistance (HOMA-IR) was calculated using the method by Matthews et al. (1985).

\[
\text{HOMA(IR)} = \frac{(\text{Insulin (uU/ml)} \times \text{Glucose(mmol/L)})}{22.5}
\]

Defrost and preparation of plasma samples was performed in accordance to manufacturer’s instructions. Plasma was analysed with kits from Roche/Hitachi (Roche Diagnostics, Mannheim, Germany). The coefficient of variation (CV) for glucose was (1.6%), insulin (1.6%), cholesterol (1.7%), HDL cholesterol (4.7%), triglycerides (1.2%) and CRP (6.7%).

**Energy expenditure of commuter cycling**

The HR Flex method was utilised to differentiate active from non-active periods in HR dataset by defining PA as 10 BPM above the highest average sedentary HR (Spurr et al., 1988). To define physiological intensity (i.e. \(\dot{VO}_2\text{max} \%)\) of cycling, week four HR data was compared to the initial \(\dot{VO}_2\text{max} \) test, whereas HR data from week eight was compared to the post-intervention test. To determine energy expenditure of commuter cycling, HR from cycling was compared to HR from the week five submaximal test and corresponding steady-state \(\dot{VO}_2\) value. Calculation for energy expenditure was estimated from oxygen consumption and expiratory carbon dioxide using the Weir (1949) method.
Energy expenditure from weighed food records

Energy expenditure was estimated based on energy intake at weeks zero, four and eight with correction for change in fasted body mass. Change in mass was added/subtracted to energy intake with the assumption that one pound (0.45kg) of mass equals 14,644 kJ (Wishnofsky, 1952; McArdle, Katch and Katch, 2001).

\[ \text{kcal} \cdot \min^{-1} = [(1.1 \times \text{RQ}) + 3.9] \times \dot{V}O_2 \]

3.14 Statistical Analyses

Overview

Statistical Packages for the Social Sciences (SPSS, Version 20.0, SPSS INC, Chicago, IL) was used to carry out statistical analysis, with significance set at \( P \leq 0.05 \). Paired t-tests were performed for measures analysed from CYC only (i.e. DXA, food records), repeated measures ANOVA were used to detect any time-by-group interactions with measures collected in both groups at three or more time points (i.e. PA). The percentage change between pre and post-intervention measures was performed on data collected at two time points (e.g. body composition, cardiovascular parameters, quality of life) and analysed by one-way ANOVA with group (i.e. CYC, CON) as independent variable.

Preliminary analysis

The use of ANOVA assumes normality of the distribution of data (Tabachnick & Fidell, 2007). Each variable was tested for normality with Shapiro-Wilk statistic prior to use
in analysis. A significant ($P \leq 0.05$) Shapiro-Wilk result indicates a violation of the assumption of normality, in which case a transformation of the data was performed prior to further analysis (Tabachnick & Fidell, 2007). Outliers were included in analyses unless instrumentation error or lack of participant compliance to research procedures was the suspected cause for the inconsistent data value, in which case the data were removed from the analysis. Whether a transformation was performed and the type of transformation used is indicated in Chapter Four.

In addition to the assumption of normality, repeated measures ANOVA also has the underlying assumption of sphericity and homogeneity of variance (Tabachnick & Fidell, 2007). A significant ($P \leq 0.05$) result from Mauchly’s Test of Sphericity indicates the assumption of sphericity is violated. In this case, results with Greenhouse-Geisser correction are reported. Homogeneity of variance was examined with Levene’s test of Equality of Error Variances with alpha set at $P \leq 0.05$. All data are presented as mean ± standard deviation, unless indicated otherwise.

**Physical activity**

Total and MVPA was analysed with a two-way analysis (group by time) with repeated measures ANOVA to compare the change from baseline PA (min/wk) between groups each week of the intervention. Mean change in PA from baseline for weeks one through ten combined was analysed by one-way ANOVA for group difference for total PA and combined moderate and vigorous PA. Similarly, to determine if time engaged in non-cycling PA changed, total PA (minus cycling) and moderate and vigorous PA
(minus cycling) was analysed by one-way ANOVA for mean change from baseline to weeks one through ten combined.

A two-way analysis (group by time) with repeated measures ANOVA was used to compare group difference in the change of PA (min/wk) from baseline measured by IPAQ at weeks four and eight. The analysis included PA and EE at vigorous-intensity, moderate-intensity, combined moderate and vigorous activity, but not for total PA due to low compliance on the 7-day recall for the amount of time spent walking in IPAQ. Data are presented as median and inter-quartile range, as recommended by the IPAQ research group (Sjöström, et al., 2005).

**Food records**

Repeated measures ANOVA was used to assess change from baseline for total energy intake (kJ/day), total and percentage intake from carbohydrates, fat and protein, and estimated energy expenditure (kJ/day) at weeks four and eight for CYC.

**Body composition**

One-way ANOVA with group as a factor was used to determine the percentage change in BMI, mass, sum of skin folds, body fat, hip circumference, waist circumference, and waist-to-hip ratio from baseline. Paired t-tests were used to measure differences between pre and post-intervention DXA scans for CYC. The analyses on body composition measures were repeated using sex as a factor to detect any differences between males and females.
**Cardiovascular measures**

Percentage change in $\dot{V}O_2$max, resting HR, blood pressure and blood parameters were analysed with one-way ANOVA, with group as a factor. Triglycerides, LDL-cholesterol and C-reactive protein yielded significant ($P < 0.05$) results from the Shapiro-Wilk statistic and were log-transformed prior to analysis.

**Quality of life questionnaire**

Each of the eight multi-itemed variables of the SF36 survey were summated and transformed to a scale of 0 (worst state of health) to 100 (best state of health) (Ware, et al., 2000). The percentage change from baseline was analysed with one-way ANOVA, with group as a factor.

**Other Measures**

Percentage change for fasting insulin and glucose, HOMA and maximum power achieved in $V_0$max test were analysed by one-way ANOVA with group as a factor.
CHAPTER FOUR: Results

4.1 Compliance

Twenty-seven participants completed intervention testing. Three CON participants (2 female, 1 male) did not complete all intermediate measures performed during the intervention (i.e. log books, food records & IPAQ). Two of these aforementioned participants completed post-intervention testing and one female withdrew due to recurring illness. All 14 CYC participants completed the intervention. However, one female CYC participant was excluded from analysis upon her admission that she was commuter cycling prior to the intervention.

Intervention

Participants reported cycling for $148 \pm 38$ min/wk, with a range of $105 - 248$ min/wk and this remained consistent ($P = 0.82$) (Figure 4.1a). Females cycled a similar amount as males ($168 \pm 59$ min/wk, $131 \pm 49$ min/wk, resp.) and this did not change over time (Time $P = 0.86$, Group $P = 0.53$, Interaction $P = 0.13$) (Figure 4.1b).
Figure 4.1a Time spent (mean ± SD) commuter cycling (n = 13) each week of the intervention period (ANOVA: P = 0.82).

Figure 4.1b Time spent (mean ± SD) commuter cycling each week of the intervention period by males (n = 7) and females (n = 6) (ANOVA: Time P = 0.86, Group P = 0.53, Interaction P = 0.13).
**Illness**

Both groups had a high prevalence of self-reported illness throughout the intervention period *(Figure 4.1c)*. When post-intervention testing was delayed due to illness, testing was rescheduled three days upon relief of symptoms for CON. When post-intervention testing was delayed to illness for CYC, testing was rescheduled upon alleviation of symptoms and after resuming commuter cycling for the same number of days that cycling was not performed due to illness (e.g. seven days of illness resulted in seven day continuation of the intervention).

*Figure 4.1c* Incidence of self-reported illness where each dot represents either one cyclist (●) or one control (○) participant.

### 4.2 Participant Characteristics

Characteristics of participants who completed intervention testing are summarised in Table 4.2. Fasted body mass was greater *(P = 0.01)* in CYC than CON and a trend *(P = 0.06)* for BMI to be greater in CYC than in CON at baseline was observed (Table 4.2). Fitness and age were similar between groups.
Table 4.2 Baseline physical characteristics (mean ± SD) for cycling and control groups at baseline.

<table>
<thead>
<tr>
<th>Cycling group</th>
<th>Age (years)</th>
<th>Body mass (kg)</th>
<th>BMI</th>
<th>(\bar{\text{V}}\text{O}_{2}\text{max} ) (l/min)</th>
<th>(\bar{\text{V}}\text{O}_{2}\text{max} ) (ml/kg/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Female n = 6</td>
<td>39.2 ± 9.0</td>
<td>75.9 ± 8.5</td>
<td>25.8 ± 2.9</td>
<td>2.3 ± 0.4</td>
<td>30.5 ± 3.9</td>
</tr>
<tr>
<td>Male n = 7</td>
<td>38.0 ± 6.0</td>
<td>80.7 ± 9.5</td>
<td>26.3 ± 1.9</td>
<td>3.3 ± 0.9</td>
<td>40.7 ± 8.9</td>
</tr>
<tr>
<td>Total n = 13</td>
<td>39.0 ± 7.0</td>
<td>76.8 ± 10.7</td>
<td>25.7 ± 2.7</td>
<td>2.8 ± 0.8</td>
<td>35.9 ± 8.2</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Control group</th>
<th>Age (years)</th>
<th>Body mass (kg)</th>
<th>BMI</th>
<th>(\bar{\text{V}}\text{O}_{2}\text{max} ) (l/min)</th>
<th>(\bar{\text{V}}\text{O}_{2}\text{max} ) (ml/kg/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Female n = 6</td>
<td>32.5 ± 6.5</td>
<td>68.7 ± 8.5</td>
<td>24.8 ± 2.4</td>
<td>2.2 ± 0.4</td>
<td>31.8 ± 7.1</td>
</tr>
<tr>
<td>Male n = 7</td>
<td>36.0 ± 7.6</td>
<td>70.8 ± 5.2</td>
<td>23.7 ± 2.3</td>
<td>3.1 ± 0.6</td>
<td>44.4 ± 8.4</td>
</tr>
<tr>
<td>Total n = 13</td>
<td>34.5 ± 7.0</td>
<td>69.8 ± 6.7</td>
<td>24.3 ± 2.3</td>
<td>2.7 ± 0.7</td>
<td>38.6 ± 9.9</td>
</tr>
</tbody>
</table>

BMI, body mass index; \(\bar{\text{V}}\text{O}_{2}\text{max}\), maximal oxygen consumption.

\(a P = 0.01\) ANOVA difference between groups.

4.3 Commuter Cycling

Duration

Participants cycled 65 ± 38 min per round trip, which is the total time of daily cycling to and from work combined. This was determined by the monitoring of heart rate whilst commuting at wk 4 and wk 8. Mean round trip duration was similar (\(P = 0.51\)) between wk 4 and wk 8 (wk 4 66 ± 39, wk 8 64 ± 38 min). One-way commute to work was shorter in duration (\(P = 0.05\)) than the commute home at wk 4, but not at wk 8 (\(P = 0.13\)) (wk 4: to work 29 ± 23, to home 37 ± 17; wk 8: to work 31 ± 21, to home 37 ± 18).
Intensity

Based upon the \( \dot{V}O_2 \text{max} \) tests and heart rates during commuting, commuter cycling was performed at 65 ± 9% of HR_max, which correlates with 56 ± 11% \( \dot{V}O_2 \text{max} \) from pre-intervention \( \dot{V}O_2 \text{max} \) tests. Intensity of commuter cycling was similar at wk 4 and wk 8 (Table 4.3). Heart rate response to cycling was higher during travel home from work at wk 4 \( (P=0.02) \) and wk 8 \( (P=0.04) \) than travel to work (wk 4: to work 60 ± 12%, to home 69 ± 7%; wk 8: to work 65 ± 10%, to home 69 ± 10% HR_max) (absolute values in Table 4.3). Similarly, cycling intensity expressed as percentage \( \dot{V}O_2 \text{max} \) was higher during travel home from work at wk 4 \( (P=0.02) \) and wk 8 \( (P=0.02) \) than travel to work (wk 4: to work 52 ± 18%, to home 60 ± 12%; wk 8: to work 46 ± 17%, to home 61 ± 9% \( \dot{V}O_2 \text{max} \)) (absolute values in Table 4.3).

Energy expenditure

Mean energy expenditure of a round trip commute, as estimated by heart rate while commuting and \( \dot{V}O_2 \) data from a lab-based sub-maximal cycling test was 2435 ± 1774 (kJ). Roundtrip commute at wk 4 was similar \( (P=0.64) \) to wk 8 (wk 4 2397 ± 1820, wk 8 2460 ± 1820 kJ).

Energy expenditure was lower for travel to work than travel home from work at wk 4 \( (P=0.03) \) and a similar trend occurred at wk 8 \( (P=0.07) \) (wk 4: to work 987 ± 1104, to home 1405 ± 790; wk 8: to work 1096 ± 1071, to home 1514 ± 806 kJ).
Table 4.3 *Physiological measures of intensity (mean ± SD) while commuter cycling (n=13) at wk 4, wk 8 and average of wk 4 and 8.*

<table>
<thead>
<tr>
<th>Average</th>
<th>To work</th>
<th>To home</th>
<th>$P^a$</th>
<th>Round trip</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR (b·min$^{-1}$)</td>
<td>111 ± 24</td>
<td>129 ± 11</td>
<td>0.02</td>
<td>120 ± 20</td>
</tr>
<tr>
<td>$\dot{V}O_2$ (l·min$^{-1}$)</td>
<td>1.4 ± 0.5</td>
<td>1.8 ± 0.5</td>
<td>0.02</td>
<td>1.6 ± 0.6</td>
</tr>
<tr>
<td>EE (kJ·min$^{-1}$)</td>
<td>31 ± 11</td>
<td>39 ± 12</td>
<td>0.05</td>
<td>35 ± 12</td>
</tr>
</tbody>
</table>

Wk 4

<table>
<thead>
<tr>
<th>Average</th>
<th>To work</th>
<th>To home</th>
<th>$P^a$</th>
<th>Round trip</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR (b·min$^{-1}$)</td>
<td>111 ± 22</td>
<td>127 ± 10</td>
<td>0.03</td>
<td>119 ± 12</td>
</tr>
<tr>
<td>$\dot{V}O_2$ (l·min$^{-1}$)</td>
<td>1.4 ± 0.4</td>
<td>1.8 ± 0.6</td>
<td>0.03</td>
<td>1.6 ± 1.4</td>
</tr>
<tr>
<td>EE (kJ·min$^{-1}$)</td>
<td>29 ± 8</td>
<td>38 ± 8</td>
<td>0.01</td>
<td>33 ± 8</td>
</tr>
</tbody>
</table>

Wk 8

<table>
<thead>
<tr>
<th>Average</th>
<th>To work</th>
<th>To home</th>
<th>$P^a$</th>
<th>Round trip</th>
<th>$P^b$</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR (b·min$^{-1}$)</td>
<td>112 ± 29</td>
<td>130 ± 15</td>
<td>0.02</td>
<td>121 ± 19</td>
<td>0.39</td>
</tr>
<tr>
<td>$\dot{V}O_2$ (l·min$^{-1}$)</td>
<td>1.4 ± 0.6</td>
<td>1.9 ± 0.5</td>
<td>0.03</td>
<td>1.6 ± 1.4</td>
<td>0.54</td>
</tr>
<tr>
<td>EE (kJ·min$^{-1}$)</td>
<td>29 ± 12</td>
<td>38 ± 12</td>
<td>0.03</td>
<td>33 ± 8</td>
<td>0.18</td>
</tr>
</tbody>
</table>

HR, heart rate; $\dot{V}O_2$, oxygen consumption; EE, energy expenditure

$^a$Paired t-test between to work and to home

$^b$Paired t-test between wk 4 and wk 8

4.4 Levels of Physical Activity

*International Physical Activity Questionnaire*

At wk 0, the majority of participants had moderate levels of physical activity, defined by IPAQ as 30 min of moderate intensity PA on most days per week (CYC n = 10, CON n = 9). Several participants (CYC n = 2, CON n = 2) had high PA levels, defined as 60 min of daily moderate activity or 30 min of vigorous activity above basal PA level (basal defined as 5,000 steps/day or the PA equivalent). Some participants had low levels of PA, defined as not meeting the requirements for moderate or vigorous categories (CYC n = 1, CON n = 2).
Table 4.4 Median (upper and lower quartiles) weekly physical activity from International Physical Activity Questionnaire in control (CON n = 11) and commuter cycling (CYC, n= 12) groups before (wk 0) and 4 weeks (4 wk) and 8 weeks (8wk) into the intervention.

<table>
<thead>
<tr>
<th></th>
<th>wk 0</th>
<th>wk 4</th>
<th>wk 8</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>CYC</td>
<td>CON</td>
<td>CYC</td>
</tr>
<tr>
<td>MVPA</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(min)</td>
<td>150 (50, 307)</td>
<td>90 (0, 375)</td>
<td>200 (132, 330)</td>
</tr>
<tr>
<td>VPA</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(min)</td>
<td>55 (0, 131)</td>
<td>0 (0, 60)</td>
<td>115 (12, 230)</td>
</tr>
<tr>
<td>MPA</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(min)</td>
<td>40 (30, 157)</td>
<td>90 (0, 410)</td>
<td>110 (41, 147)</td>
</tr>
</tbody>
</table>

MVPA, moderate and vigorous intensity physical activity combined; VPA, vigorous intensity physical activity; MPA, moderate intensity physical activity.

\( a P = 0.005 \) ANOVA change from baseline \((P > 0.05\) for group effect and time-by-group interaction)  
\( b P = 0.05 \) ANOVA change from baseline \((P > 0.05\) for group effect and time-by-group interaction)  
\( c P = 0.03 \) ANOVA change from baseline \((P > 0.05\) for group effect and time-by-group interaction)

There were no group differences at wk 0 for combined moderate and vigorous PA according to reported minutes active in defined intensity categories (CYC 160 ± 119, CON 169 ± 262 min/wk, \( P = 0.91 \)), vigorous PA (CYC 85 ± 100, CON 24 ± 32 min/wk, \( P = 0.09 \)) nor moderate PA (CYC 75 ± 82, CON 141 ± 257 min/wk, \( P = 0.39 \)).

Moderate and vigorous PA combined changed \((P = 0.005)\) from baseline and a trend \((P = 0.06)\) for PA to be reported higher in CYC than CON was observed (wk 4: CYC Δ114 ± 131, CON Δ0 ± 119; wk 8: CYC Δ38 ± 117, CON Δ−67 ± 200 min/wk) (absolute values in Table 4.4). Vigorous PA also changed from baseline (wk 4: CYC Δ55 ± 85, CON Δ26 ± 70; wk 8: CYC Δ20 ± 78, CON Δ−5 ± 31 min/wk) (Table 4.4) but the large variation
between individuals precluded a significant difference between groups or interaction (Time $P = 0.05$, Group $P = 0.90$, Interaction $P = 0.38$) (Table 4.4). Moderate PA changed from baseline (wk 4: CYC $\Delta 45 \pm 75$, CON $\Delta -26 \pm 176$; wk 8: CYC $\Delta 5 \pm 79$, CON $\Delta - 70 \pm 193$ min/wk) and there was no difference between groups nor was there a significant interaction (Time $P = 0.03$, Group $P = 0.20$, Interaction $P = 0.93$) (Table 4.4).

**Physical Activity Log Books**

The contribution of cycling to total weekly PA, as recorded in log books, in CYC and CON is illustrated in Figure 4.4a. Groups were similar at wk 0 for mean total PA ($P = 0.68$) (Figure 4.4a) and for combined moderate and vigorous PA ($P = 0.42$) (Figure 4.4b). Difference from baseline in weekly total PA did not change over time and there was no difference between groups over the ten week period (Time $P = 0.19$, Group $P = 0.24$, Interaction $P = 0.46$) (absolute values in Figure 4.4a). Further, mean change in PA of weeks one through ten combined was similar ($P = 0.24$) to wk 0 (absolute values in Figure 4.4c, Appendix II). Difference from baseline in reported weekly moderate and vigorous PA was higher in CYC than CON, but no change was detected over time nor was there a significant interaction (Time $P = 0.26$, Group $P = 0.003$, Interaction $P = 0.31$) (absolute values in Figure 4.4b). However, when analysed as the mean of weeks one through ten of moderate and vigorous PA, CYC increased ($P = 0.003$) from baseline compared to CON (CYC $\Delta 112 \pm 101$, CON $\Delta -10 \pm 82$ min/wk) (absolute values in Figure 4.4d Appendix II).
Figure 4.4a Total physical activity per week (mean ± SD) in control (n = 12) and commuter cycling (n = 13) groups, the latter differentiating that from cycle commuting and other physical activities (ANOVA for difference from baseline: Time P = 0.19, Group P = 0.24, Interaction P = 0.46).

Figure 4.4b Vigorous and moderate intensity physical activities combined per week (mean ± SD) in cycling (n=13) and control (n=12) groups (ANOVA for difference from baseline: Time P = 0.26, Group P = 0.003, Interaction P = 0.31).
Non-cycling physical activity from log books

Weekly reported total PA unrelated to commuter cycling did not change over time and there was no difference between groups (Time $P = 0.22$, Group $P = 0.23$, Interaction $P = 0.37$) (Figure 4.4e). Further, the ten week average for time engaged in PA unrelated to commuter cycling was unchanged ($P = 0.23$) from wk 0 (absolute values in Figure 4.4f, Appendix II). Weekly moderate and vigorous PA did not change over the 10 week period and there was no difference between groups (Time $P = 0.36$, Group $P = 0.81$, Interaction $P = 0.13$) (Figure 4.4g). Likewise, moderate and vigorous PA for weeks one through ten combined also did not change ($P = 0.81$) (absolute values in Figure 4.4h, Appendix II). In conclusion, no change to PA outside of time spent commuter cycling was observed.
Figure 4.4e Total physical activity per week (mean ± SD) outside of commuter cycling in cycling (n=13) and control (n=12) groups (ANOVA for difference from baseline: Time $P = 0.22$, Group $P = 0.23$, Interaction $P = 0.37$).

Figure 4.4g Time spent in vigorous and moderate intensity physical activities combined (mean ± SD) outside of commuter cycling in cycling and control groups (ANOVA for difference from baseline: Time $P = 0.36$, Group $P = 0.81$, Interaction $P = 0.13$).
4.5 Energy Expenditure

*International Physical Activity Questionnaire*

Energy expenditure calculated as metabolic equivalent minutes per week (MET-min/wk) for combined MET-min from moderate and vigorous PA ($P = 0.16$), and moderate PA ($P = 0.39$) but a trend ($P = 0.07$) for vigorous PA to be greater in CYC than CON was observed (Table 4.5a).

Difference from baseline for MET-min/wk from moderate and vigorous PA changed over time ($P = 0.005$), but no main group effect ($P = 0.16$) or interaction ($P = 0.25$) were observed (wk 4: CYC $\Delta 666 \pm 823$, CON $\Delta 136 \pm 413$; wk 8: $\Delta$CYC $134 \pm 882$, CON $\Delta -103 \pm 567$ MET-min/wk). MET-min/wk from moderate PA transiently increased over time at wk 4 ($P = 0.05$), with no observed changes to the main group effect ($P = 0.66$) or interaction ($P = 0.77$) (wk 4: CYC $\Delta 93 \pm 429$, CON $\Delta 5 \pm 309$; wk 8: CYC $\Delta -56 \pm 407$, CON $\Delta -107 \pm 477$ MET-min/wk) (Table 4.5a). MET-min/wk from vigorous PA also increased over time ($P = 0.02$) and again no observed changes between groups ($P = 0.10$) or interaction ($P = 0.22$) (wk 4: CYC $\Delta 572 \pm 654$, CON $\Delta 130 \pm 328$; wk 8: CYC $\Delta 190 \pm 645$, CON $\Delta 3 \pm 223$ MET-min/wk)
Table 4.5a *Calculated energy expenditure (Median (upper and lower quartiles)) from IPAQ questionnaire with 10 wk commuter cycling intervention and in a control group.*

<table>
<thead>
<tr>
<th></th>
<th>wk 0</th>
<th>wk 4</th>
<th>wk 8</th>
<th>Time</th>
<th>Group</th>
<th>Interaction</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>MVPA MET[min/wk]</strong></td>
<td>CYC 942 (338, 1784)</td>
<td>CON 409 (0, 1272)</td>
<td>CYC 1730 (815, 2732)</td>
<td>CON 630 (0, 1139)</td>
<td>CYC 1312 (687, 2382)</td>
<td>CON 466 (129, 682)</td>
</tr>
<tr>
<td><strong>MPA MET[min/wk]</strong></td>
<td>CYC 224 (145, 821)</td>
<td>CON 157 (0, 763)</td>
<td>CYC 528 (153, 761)</td>
<td>CON 331 (0, 584)</td>
<td>CYC 477 (42, 650)</td>
<td>CON 212 (43, 681)</td>
</tr>
<tr>
<td><strong>VPA MET[min/wk]</strong></td>
<td>CYC 680 (0, 1206)</td>
<td>CON 0 (0, 508)</td>
<td>CYC 1361 (263, 2455)</td>
<td>CON 220 (0, 584)</td>
<td>CYC 631 (169, 1772)</td>
<td>CON 0 (0, 363)</td>
</tr>
</tbody>
</table>

MET, metabolic equivalent; MVPA, moderate and vigorous physical activity; MPA, moderate physical activity; VPA, vigorous physical activity.

*ANOVA for the difference from baseline*
Food Records

No change in total energy, carbohydrate, fat or protein intakes were observed over time in CYC (Table 4.5b). Similarly, estimated energy expenditure from food records with correction for body mass changes resulted in no change ($P = 0.26$) over time in CYC (Figure 4.5).

Table 4.5b Total energy intake, carbohydrate intake, fat intake and protein intake for 13 commuter cyclists at week 0 and weeks 4 and 8.

<table>
<thead>
<tr>
<th></th>
<th>Wk 0</th>
<th>Wk 4</th>
<th>Wk 8</th>
<th>$P^a$</th>
</tr>
</thead>
<tbody>
<tr>
<td>TEI (kJ/d)</td>
<td>10,206 ± 2,236</td>
<td>10,367 ± 1,623</td>
<td>9,949 ± 2,735</td>
<td>0.52</td>
</tr>
<tr>
<td>CHO (g/d)</td>
<td>316 ± 117</td>
<td>320 ± 68</td>
<td>303 ± 120</td>
<td>0.71</td>
</tr>
<tr>
<td>CHO (E%)</td>
<td>52 ± 9</td>
<td>52 ± 8</td>
<td>51 ± 7</td>
<td>0.34</td>
</tr>
<tr>
<td>Fat (g/d)</td>
<td>86 ± 22</td>
<td>91 ± 22</td>
<td>87 ± 18</td>
<td>0.54</td>
</tr>
<tr>
<td>Fat (E%)</td>
<td>32 ± 7</td>
<td>32 ± 6</td>
<td>33 ± 5</td>
<td>0.67</td>
</tr>
<tr>
<td>Protein (g/d)</td>
<td>91 ± 18</td>
<td>86 ± 18</td>
<td>85 ± 20</td>
<td>0.92</td>
</tr>
<tr>
<td>Protein (E%)</td>
<td>15 ± 3</td>
<td>14 ± 2</td>
<td>15 ± 2</td>
<td>0.16</td>
</tr>
</tbody>
</table>

TEI, Total energy intake; CHO, carbohydrate; kJ/d, kilojoules per day; g/d, grams per day; E%, percentage daily energy intake.

$^a P \leq 0.05$ paired t-tests on the difference from baseline.
Figure 4.5 Estimated daily energy expenditure (mean ± SD) for commuter cyclists (n=13) determined by the average of 4-day food records at week 0 and weeks 4 and 8 with correction for body mass change (ANOVA P = 0.26).

4.6 Body Composition

**Anthropometry**

Baseline data were similar between groups for percentage body fat ($P = 0.28$), sum of five skin folds ($P = 0.37$), waist circumference ($P = 0.15$), hip circumference ($P = 0.71$), WHR ($P = 0.19$) (Table 4.6a). Fasted body mass was greater ($P = 0.01$) in CYC than CON at baseline and a similar trend ($P = 0.06$) for BMI was observed (Table 4.2a). No effects of commuter cycling on body mass, sum of skin folds, percentage body fat, waist circumference or BMI were observed over time or between groups (Table 4.6a). Waist to hip ratio (WHR) decreased in CYC compared to CON (CYC -5.1 ± 5.8%, CON -0.2 ± 2.4%, $P = 0.01$) (Table 4.6a). Change in WHR resulted from an increase in hip
circumference with commuter cycling (CYC 5.3 ± 4.0\%, CON 1.4 ± 3.1\%, \(P = 0.01\)) (Table 4.6a). Waist circumference did not change (\(P = 0.36\)) (Table 4.6a).

\textit{Dual energy x-ray absorptiometry (DXA)}

Commuter cycling had no effect (\(P > 0.05\)) on lean and fat mass determined by DXA. (Table 4.6b). Circumferences and ratio of waist and hip determined from the DXA were not observed to be altered (\(P > 0.05\)).

\begin{table}[h]
\centering
\begin{tabular}{lcccc}
\hline
 & \textbf{CYC (n=13)} & & \textbf{CON (n=13)} & \\
 & \textbf{Baseline} & \textbf{Follow-up} & \textbf{Baseline} & \textbf{Follow-up} & \textbf{\(P^a\)} \\
\hline
Body mass (kg) & 78.5 ± 9.0 & 78.7 ± 9.0 & 69.8 ± 6.7 & 70.3 ± 6.7 & 0.61 \\
4-site skinfolds (mm) & 76 ± 36 & 76 ± 35 & 62 ± 26 & 63 ± 24 & 0.95 \\
Body fat (%) & 30.2 ± 8.6 & 30.3 ± 8.4 & 26.8 ± 7.7 & 26.9 ± 7.9 & 0.69 \\
BMI & 26.1 ± 2.4 & 26.2 ± 2.4 & 24.2 ± 2.3 & 24.4 ± 2.3 & 0.61 \\
WHR & 0.85 ± 0.08 & 0.81 ± 0.08 & 0.80 ± 0.06 & 0.81 ± 0.07 & 0.01 \\
Waist (cm) & 83.3 ± 7.2 & 83.2 ± 8.7 & 78.1 ± 6.5 & 79.0 ± 6.9 & 0.36 \\
Hip (cm) & 98.3 ± 9.1 & 103.4 ± 7.5 & 96.5 ± 3.5 & 97.8 ± 4.2 & 0.01 \\
\hline
\end{tabular}
\caption{Body composition measures (mean ± SD) at baseline and after 10 weeks (Follow-up) by those commuter cycling (CYC) and a control group (CON).}
\end{table}

BMI, body mass index; WHR, waist-to-hip ratio.

\(^a P \leq 0.05\) ANOVA for the group difference in change from baseline to follow-up.
Table 4.6b: Dual-energy X-ray absorptiometry measures of body composition (mean ± SD) at baseline and after 10 weeks of commuter cycling (n = 13; 7 males, 6 females).

<table>
<thead>
<tr>
<th>Measure</th>
<th>Baseline</th>
<th>Follow-up</th>
<th>(P^a)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total body fat (%)</td>
<td>29.42 ± 9.67</td>
<td>29.46 ± 9.82</td>
<td>0.93</td>
</tr>
<tr>
<td>Total body lean (%)</td>
<td>66.55 ± 9.54</td>
<td>66.50 ± 9.71</td>
<td>0.90</td>
</tr>
<tr>
<td>Waist fat (%)</td>
<td>37.06 ± 8.15</td>
<td>36.13 ± 8.51</td>
<td>0.92</td>
</tr>
<tr>
<td>Hip fat (%)</td>
<td>36.62 ± 10.44</td>
<td>36.65 ± 11.10</td>
<td>0.92</td>
</tr>
<tr>
<td>Hip lean (%)</td>
<td>63.36 ± 10.45</td>
<td>63.34 ± 11.08</td>
<td>0.95</td>
</tr>
<tr>
<td>Trunk fat (%)</td>
<td>32.93 ± 8.50</td>
<td>33.28 ± 8.99</td>
<td>0.57</td>
</tr>
<tr>
<td>Trunk lean (%)</td>
<td>67.07 ± 8.50</td>
<td>66.71 ± 8.99</td>
<td>0.57</td>
</tr>
<tr>
<td>Legs fat (%)</td>
<td>31.10 ± 13.17</td>
<td>30.71 ± 13.11</td>
<td>0.34</td>
</tr>
<tr>
<td>Legs lean (%)</td>
<td>68.89 ± 13.17</td>
<td>69.28 ± 13.11</td>
<td>0.34</td>
</tr>
<tr>
<td>Arms fat (%)</td>
<td>25.99 ± 11.41</td>
<td>25.91 ± 11.21</td>
<td>0.82</td>
</tr>
<tr>
<td>Arms lean (%)</td>
<td>74.01 ± 11.41</td>
<td>74.08 ± 11.21</td>
<td>0.82</td>
</tr>
<tr>
<td>Hips (cm)</td>
<td>104.6 ± 8.8</td>
<td>103.8 ± 7.3</td>
<td>0.42</td>
</tr>
<tr>
<td>Waist (cm)</td>
<td>88.1 ± 7.9</td>
<td>87.4 ± 8.5</td>
<td>0.31</td>
</tr>
<tr>
<td>WHR</td>
<td>0.85 ± 0.09</td>
<td>0.84 ± 0.09</td>
<td>0.90</td>
</tr>
</tbody>
</table>

WHR, waist-to-hip ratio.

\(^a P \leq 0.05\) determined by paired t-tests.

4.7 Cardiovascular Measures

Maximum oxygen consumption

Groups were similar (\(P = 0.71\)) in absolute \(\dot{\text{VO}_2}\)max (l · min⁻¹) and relative to body mass (ml · kg · min⁻¹) (\(P = 0.45\)) at baseline (Table 4.2a). Absolute \(\dot{\text{VO}_2}\)max increased after 10 weeks of commuting in CYC, however, in CON it decreased (CYC 10.4 ± 17.3 %, CON -1.5 ± 11.3 %, resp., \(P = 0.05\)) (Table 4.7a). A similar pattern was observed when calculated relative to body mass (CYC 10.5 ± 16.2 %, CON -2.8 ± 12.3 %, resp., \(P = 0.03\)) (Table 4.7a).
Table 4.7a *Maximum oxygen consumption (mean ± SD) at baseline and at 10 weeks (follow-up) in commuter cyclists (CYC) and controls (CON).*

<table>
<thead>
<tr>
<th></th>
<th>CYC (n = 13)</th>
<th>CON (n = 13)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Baseline</td>
<td>Follow-up</td>
</tr>
<tr>
<td>Absolute $\dot{V}O_2$max (1·min$^{-1}$) $^a$</td>
<td>2.76 ± 0.8</td>
<td>2.97 ± 0.8</td>
</tr>
<tr>
<td>Relative $\dot{V}O_2$max (ml·kg·min$^{-1}$) $^b$</td>
<td>35.9 ± 8.2</td>
<td>38.7 ± 6.5</td>
</tr>
</tbody>
</table>

$^a$ $P = 0.05$ ANOVA of the group difference in change from baseline to follow-up.
$^b$ $P = 0.03$ ANOVA of the group difference in change from baseline to follow-up.

*Resting heart rate*

Groups were similar ($P = 0.27$) in resting heart rate at baseline. Resting heart rate decreased in CYC (-4.8 ± 6.2 %, $P = 0.03$) and increased in CON (2.6 ± 9.1 %) (*Figure 4.7a*).

*Resting blood pressure*

Groups were similar at baseline for systolic ($P = 0.18$) and diastolic ($P = 0.08$) blood pressure. No effect ($P = 0.20$) of commuter cycling on systolic BP was detected (*Figure 4.7b*). Diastolic BP decreased ($P = 0.02$) in CYC (-1.2 ± 7.3 %) and increased in CON (11.8 ± 16.5%) (*Figure 4.7b*).
Figure 4.7a Resting heart rate (mean ± SD) at baseline and at 10 weeks (follow-up) in commuter cyclists and controls (ANOVA for group change from baseline to follow-up $P = 0.03$).

Figure 4.7b Systolic ($P = 0.20$)$^a$ and diastolic ($P = 0.02$)$^a$ blood pressure (mean ± SD) at wk. 0 and at 10 weeks in commuter cyclists and controls.

$^a$ ANOVA for the group difference in change from baseline to follow-up.
**Blood Parameters**

One CYC participant was excluded from C-reactive protein (CRP) analysis due to elevated CRP levels that indicated an underlying ailment (Haran, et al., 2013). No group differences were observed at baseline for total cholesterol ($P = 0.66$), HDL ($P = 0.66$), LDL ($P = 0.29$), triglycerides ($P = 0.96$), or CRP ($P = 0.12$). There were no significant effects of commuter cycling on any blood parameters (Table 4.7b).

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>Follow-up</th>
<th>$P^a$</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>CYC</td>
<td>CON</td>
<td>CYC</td>
</tr>
<tr>
<td>Total Cholesterol</td>
<td>4.98 ± 0.78</td>
<td>5.39 ± 1.05</td>
<td>4.79 ± 0.67</td>
</tr>
<tr>
<td>HDL Cholesterol</td>
<td>0.95 ± 0.32</td>
<td>1.15 ± 0.29</td>
<td>0.95 ± 0.32</td>
</tr>
<tr>
<td>LDL Cholesterol</td>
<td>3.24 ± 0.45</td>
<td>3.72 ± 1.03</td>
<td>3.20 ± 0.53</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>1.61 ± 1.69</td>
<td>1.13 ± 0.62</td>
<td>1.35 ± 0.91</td>
</tr>
<tr>
<td>C-Reactive Protein (b)</td>
<td>0.74 ± 0.72</td>
<td>0.85 ± 0.97</td>
<td>0.99 ± 0.68</td>
</tr>
</tbody>
</table>

HDL, high-density lipoprotein; LDL, low-density lipoprotein.

\(a\) $P \leq 0.05$ ANOVA for the group difference in change from baseline to follow-up.

\(b\) Analyses included 12 CYC participants
4.8 Quality of life

Groups were similar at baseline for all health-related quality of life variables. No significant effects ($P > 0.05$) were observed after 10 weeks commuter cycling (Table 4.8). However, a trend ($P = 0.06$) for social functioning to decrease was observed (CYC $-2.2 \pm 18.4$, CON $-5.2 \pm 12.6\%$) (Table 4.8).

Table 4.8 Health-related quality of life parameters (mean $\pm$ SD) at baseline and after 10 weeks (follow-up) commuter cycling (CYC) and a control group (CON).

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>Follow-up</th>
<th>$P^a$</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>CYC</td>
<td>CON</td>
<td>CYC</td>
</tr>
<tr>
<td>Physical functioning</td>
<td>96.2 $\pm$ 4.6</td>
<td>95.8 $\pm$ 5.7</td>
<td>96.9 $\pm$ 4.8</td>
</tr>
<tr>
<td>Role physical</td>
<td>94.2 $\pm$ 14.9</td>
<td>73.1 $\pm$ 42.7</td>
<td>90.4 $\pm$ 28.0</td>
</tr>
<tr>
<td>Bodily pain</td>
<td>84.6 $\pm$ 13.3</td>
<td>81.2 $\pm$ 13.9</td>
<td>80.3 $\pm$ 10.3</td>
</tr>
<tr>
<td>Mental health</td>
<td>80.3 $\pm$ 14.2</td>
<td>78.2 $\pm$ 10.4</td>
<td>78.8 $\pm$ 14.1</td>
</tr>
<tr>
<td>Role emotional</td>
<td>94.9 $\pm$ 18.5</td>
<td>79.5 $\pm$ 37.4</td>
<td>94.9 $\pm$ 8.5</td>
</tr>
<tr>
<td>Social functioning</td>
<td>92.3 $\pm$ 15.9</td>
<td>96.6 $\pm$ 5.3</td>
<td>88.0 $\pm$ 12.4</td>
</tr>
<tr>
<td>Vitality</td>
<td>61.9 $\pm$ 23.9</td>
<td>62.3 $\pm$ 13.9</td>
<td>65.0 $\pm$ 15.8</td>
</tr>
<tr>
<td>General health</td>
<td>73.3 $\pm$ 22.7</td>
<td>70.5 $\pm$ 12.1</td>
<td>76.8 $\pm$ 19.2</td>
</tr>
</tbody>
</table>

$^a$ ANOVA for the group difference in change from baseline to follow-up.
4.9 Other Measures

Glucose and insulin measures

One CYC participant was excluded from glucose, insulin and homeostatic model assessment – insulin resistance (HOMA-IR) analyses due to uncertainty for the cause in the 81% lower plasma insulin level at follow-up than at baseline. The change in plasma insulin was not typical based on other eight to twelve week PA interventions (Yamamoto, et al., 2007, Gray, et al., 2009). Therefore error may have derived from participant compliance to fasting in the baseline measure or error during plasma analysis. Groups were similar at baseline for fasting plasma glucose ($P = 0.85$), insulin ($P = 0.45$) and HOMA-IR ($P = 0.71$). There were no significant effects of commuter cycling on glucose ($P = 0.69$), insulin ($P = 0.61$) or HOMA-IR ($P = 0.61$) (Table 4.9a).

Table 4.9 Fasted plasma levels of glucose, insulin and HOMA-IR (mean ± SD) at baseline and after 10 weeks (follow-up) of commuter cycling (n=12) and control group (n=13).

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>Follow-up</th>
<th>$P^a$</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>CYC</td>
<td>CON</td>
<td>CYC</td>
</tr>
<tr>
<td>Glucose (mmol/L)</td>
<td>5.41 ± 0.77</td>
<td>5.46 ± 0.54</td>
<td>5.25 ± 0.36</td>
</tr>
<tr>
<td>Insulin (uU/ml)</td>
<td>6.79 ± 3.04</td>
<td>5.94 ± 2.43</td>
<td>7.00 ± 3.07</td>
</tr>
<tr>
<td>HOMA-IR</td>
<td>1.57 ± 0.71</td>
<td>1.47 ± 0.69</td>
<td>1.63 ± 0.76</td>
</tr>
</tbody>
</table>

HOMA-IR, homeostatic model assessment – insulin resistance.

$^a P \leq 0.05$ ANOVA for the group difference in change from baseline to follow-up.
Cycling power output

Groups were similar ($P = 0.65$) for peak power (w) output from the incremental $\dot{V}O_2$ max cycling at baseline. Peak power improved ($P < 0.001$) in CYC (7.9 ± 11.7 %) whereas it decreased in CON (-9.8 ± 7.7 %) (Figure 4.9).

![Bar graph showing peak power output during incremental $\dot{V}O_2$ max test on cycle ergometer at baseline and after 10 weeks (follow-up) by those commuter cycling and a control group (ANOVA for the group difference in change from baseline to follow-up $P < 0.001$).]
CHAPTER 5: Discussion

5.1 Overview

The present study is the first to investigate the effect of introducing commuter cycling to an individual’s daily routine on existing physical activity and cardiovascular health and to compare this with a control group over the same time period. Following 10 weeks of cycling totalling around 150 min/wk, cardiovascular fitness and resting heart rate were enhanced. Furthermore, an increase is diastolic blood pressure observed in the control group may have been thwarted amongst cyclists due the intervention. Unexpectedly, these health gains occurred despite the fact that there was no measurable change in the total amount of time engaged in physical activity. Perhaps even more surprising was the finding that parameters of cardiovascular health were improved without significant change in total body mass or proportion or distribution of fat mass and lean mass. Due to the relatively short time frame over which this trial was conducted, the possibility that if the commuter cycling was continued for a prolonged period, body composition changes may have occurred. Nevertheless, these data do support others demonstrating that physical fitness, health & longevity can be improved irrespective of body mass or composition (Lee, et al., 1999). The improvement in health without change in overall PA may have resulted from the increased energetic demands of cycling above that of most other activities.
5.2 Physical activity

Total physical activity

Total PA, including commuter cycling and daily activities, as measured by logbooks was not observed to be altered. In the one other study that also examined the effect of a CC intervention on total PA, a greater total PA (inclusive of cycling) in cyclists than controls was observed (de Geus, et al., 2008). Although de Geus et al. (2008) observed greater PA in cyclists at the 26 and 52 week follow-up assessments, pre-intervention PA habits were not reported and therefore it is not known whether cyclists were already more active prior to the non-randomised group selection. Furthermore, de Geus et al. (2008) defined PA as leisure-time PA and cycling, which excludes PA from daily living (e.g. gardening, playing with children, occupation, etc.). In contrast, participants from our CC trial were instructed to report all types of PA, not just those from exercise.

The lack of change in total PA with the addition of ~150 min/wk CC suggests that compensation in other physical activities occurred. Also in support of compensation in PA was the finding that energy intake (as determined by weighed food records) and body composition (DXA) were unaltered by cycling. The addition of CC without change to energy intake or change in body composition indicates energy expenditure (EE) was not significantly altered and was likely maintained at pre-intervention levels due to a decrease in PA outside of time spent cycling. However, despite the probable decrease in other activities, results showed no change in the recorded time engaged in PA outside of cycling. It may be that the method of capturing daily activities did not
have the resolution to identify changes in incidental, short lasting activities or that a shift of intensity of activities occurred. The increase in cardiorespiratory fitness in cyclists above that of controls favours the likelihood that short lasting, lower intensity activities were replaced with cycling at a greater intensity. Intensity of exercise generally has a greater effect than volume towards the adaptations that increase \( \dot{V}O_{2\text{max}} \) (Gutin, et al., 2002; Wenger & Bell, 1986; Thomas, et al., 1984).

Although DXA measures of body composition support our conclusions that EE was unaltered by CC, it is important to acknowledge that estimating EE from food diaries has an unknown level of validity due to potential errors from self-reporting, which generally results in underestimated EE values (Jackson & Wootton, 1990). Furthermore, it is unknown if participants’ body mass was stable in the months prior to the intervention, which is another limitation of estimating EE from diet records.

**Moderate and vigorous physical activity**

Although total PA was unaltered by CC in the present study, time engaged in moderate and vigorous-intensity PA combined (MVPA) as reported in the International Physical Activity Questionnaire was greater at weeks four and eight than at baseline. The validity of IPAQ has been observed to be variable, ranging from low to high compared to objective PA measures (Lee, et al., 2011). Since objective measures of PA were not used in this study, the validity of IPAQ results is unknown. Nevertheless, with the removal of light intensity PA from daily logbook analyses, cyclists engaged in more MVPA than controls during the intervention period, even though groups were similar
(p > 0.05) at baseline, which supports the IPAQ results. The increase in MVPA with CC is corroborated by heart rate data. In conjunction with laboratory-based \( \dot{V}O_2 \)max tests, heart rate data indicate that the intensity at which cycling was performed was \( 56 \pm 11\% \dot{V}O_2 \)max, which corresponds to moderate-intensity PA. Therefore, CC increased the amount of time cyclists performed MVPA, but was insufficient to affect total PA and energy balance. The self-report instruments used to collect PA data may have contributed to the lack of observable changes (Vanhees, et al., 2010). Furthermore, the power analysis performed to determine the sample size for this study was not conducted on PA variables, and it is possible that this study is lacking in statistical power to draw firm conclusions about the role of CC on existing levels of PA. Still, the addition of commuter cycling has likely increased time engaged in MVPA, which is supported by our results for enhanced cardio respiratory function among cyclists.

5.3 Cardiovascular health

Maximal oxygen consumption
The 10.5% increase in \( \dot{V}O_2 \)max that we observed among commuter cyclists corroborates the findings of previous short term (i.e. 8-10 weeks) CC studies and demonstrates that the present study produced similar cardiovascular strain as previous investigations, which found a 7.3% (Oja, et al., 1991) and 12.5% (Møller, et al., 2011) increase in \( \dot{V}O_2 \)max. Interestingly, our participants were, on average, younger (16%) and fitter (12%) than those from previous investigations, indicating that CC even has benefits for
young adults who lead active lives (Oja, et al., 1991; de Geus, et al., 2008; Møller, et al., 2011). Furthermore, $\dot{V}O_2$max is a robust predictor for cardiovascular health and morbidity (Haskell, et al., 2007). Therefore, findings from the present and previous studies indicate that cycling for transport, at a self-selected pace, can improve cardiovascular function in healthy adults in as little as 10 weeks.

In their meta-analytic review, Kodema et al., (2009) performed a dose-response analysis to determine the role of fitness on risk of CVD events and mortality. The authors reported a 13% decrease in risk with each 1 MET (3.5ml/kg/min) increase in fitness from their analysis of 33 studies (Kodama, et al., 2009). The robustness of the evidenced presented by Kodema et al., (2009) for the role of fitness on CVD outcomes is not clear, and may be confounded by covariates that may influence the results. Nevertheless, the results from Kodama, et al., (2009) indicate a goal for the minimum amount of increase in fitness that should occur to see meaningful, long term benefits. In CC studies to date, only one group observed increases in fitness above 1 MET, but the authors omitted results on the volume or intensity of exercise performed by cyclists (Møller, et al., 2011). We observed a 2.8ml/kg/min increase in $\dot{V}O_2$max with 150 mins/wk of moderate-intensity cycling and Oja, et al., (1991) observed a 3.1ml/kg/min increase with an estimated 210 min/wk of commuter cycling, also at moderate intensity. The increase in $\dot{V}O_2$max below 1 MET generally observed in CC trials may explain why prospective cohort studies have observed enhancements to CVD risk factors (i.e. fasting plasma glucose, triglycerides, systolic blood pressure) with habitual commuter cyclists,
but results haven’t been repeated in in short term CC trials (for reviews, see Hamer & Chida, 2008; and Oja, et al., 2011).

Numerous adaptations occur with aerobic training that will increase \( \dot{V}O_2 \text{max} \). An increase in left ventricular mass results in an increase in end-diastolic volume, thus leading to improved stroke volume and cardiac output per heartbeat (Hickson, et al., 1985). Although aerobic training, including cycling, is well established to increase ventricle mass, the exercise load from the present study was unlikely to have created enough strain to develop that adaptation in 10 weeks (Fagard, 1997; Hickson, et al., 1985; Sipola, et al., 2009). Although skeletal, cardiac and enzymatic adaptations were not directly measured in this study, adaptations that are likely to have occurred with 10 weeks of CC are increased mitochondrial density in skeletal muscle, quantity of oxidative enzymes and capillary density, which improves saturation and utilisation of oxygen at the muscle (Nadel, 1985; Andersen & Hendriksson, 1977; Klausen, et al., 1981). A more prominent adaptation responsible for improved \( \dot{V}O_2 \text{max} \) with CC is greater cardiac output through expanded blood volume, which increases the amount of circulating oxygen and thus allowing greater utilisation of oxygen at the muscle for aerobic metabolism (Gledhill, et al., 1999; Akgun, et al., 1974; Convertino, et al., 1991).

If CC was performed long-term, further adaptations to cardiac muscle and vasculature may occur, further enhancing \( \dot{V}O_2 \text{max} \) and may even reduce CVD risk factors that longitudinal studies have observed among commuter cyclists, but allusive in CC trials, such as the present study.
Blood pressure and resting heart rate

A group difference in diastolic blood pressure was observed due to an increase in DBP among controls. Two other research groups monitored blood pressure and observed no change compared to controls after eight weeks (Møller, et al., 2011) and one year (de Geus, et al., 2008) of commuter cycling. In the present study, systolic blood pressure was not altered, which supports previous findings that CC is generally performed at an intensity and/or duration that is insufficient to strain the cardiovascular system enough to enhance BP. However, aforementioned authors observed a decrease in DBP amongst their participants who were commuter cycling, but they did not observe a significant group effect (Møller, et al., 2011; de Geus, et al., 2008). The group effect for DBP observed in the present study should be approached with caution, as CC did not directly enhance DBP in cyclists. Nevertheless, this result is of importance because the time of year and location in which the study took place would likely have imposed seasonal fluctuations for time engaged in PA and consequent changes to health parameters. For example, we observed controls to have decreased cycling power output (10±7%), increased resting heart rate (3±9%), decreased \( \dot{V}O_2 \)max (3±12%), and increased DBP (12±16%) at the end of the 10 week intervention period. The intervention period started in autumn and finished in winter and the study location was at latitude 45.8 degrees south in which winter temperatures and daylight hours are less favourable for outdoor activities than regions further north. Seasonal influences on PA (Matthews, et al., 2001), risk of mortality from heart disease (Dunnigan, et al., 1970) and low levels of Vitamin D (Webb, et al., 1988) are well established. Low levels of Vitamin D contribute
to seasonal variation for blood pressure by working as a negative endocrine regulator on the renin-angiotensin system, which contributes to the regulation of blood pressure through reno-cardiovascular functions (Li, et al., 2002; Li, et al., 2004). In the present study, the seasonal increase in diastolic blood pressure observed in controls may have been prevented in cyclists and if this was the case, it was most likely through change of activity from the autonomic nervous system (Guyenet, 2006).

Decrease of sympathetic tone, and to a lesser extent, an increase in parasympathetic tone is the probable mechanism for the prevention of seasonal DBP change (Tashiro, et al., 1993; Young, 2010). The decrease in sympathetic tone reduces activity of arterial smooth muscle causing endothelium vasodilation and thus reduces systemic vascular resistance (Guyenet, 2006; Young, 2010). The reduction in vascular resistance is the primary mechanism for decreased arterial blood pressure at rest with regular exercise (DeSouza, et al., 2000; Tashiro, et al., 1993).

A change to the autonomic nervous system at rest is also most likely the primary mechanism for the reduced resting heart rate that we observed after 10 weeks of CC (Ekblem, et al., 1973). Reduced sympathetic activity alters the contractility of the cardiac muscle at rest through reduced tone of adrenergic cardiac nerves to the atria and ventricles to lower norepinephrine secretion, as well as on the adrenal medulla and consequent reduction of circulating epinephrine and norepinephrine (Young, 2010). The finer control of cardiac muscle contractions, combined with the increased stroke
volume that likely occurred from an expanded plasma volume with CC, would result in fewer heart beats per minute to maintain resting cardiac output.

Evidence of the relationship between morbidity and blood pressure is robust (He & Whelton, 1999; Cook, et al., 1995). Similarly, resting heart rate is an indirect measure of vascular resistance as it relates to stroke volume and resting cardiac output, all of which are indicators of cardiovascular function and independently linked to risk of mortality by cardiovascular disease (Fox, et al., 2007; Diaz, et al., 2005; Jouven, et al., 2005). Adaptations required to improve blood pressure and heart rate at rest are well established with exercise training (For review, see Arroll & Beaglehole, 1992; Chen & Dicarlo, 2009). The present study is the first to observe that the introduction of commuter cycling to an individual’s daily routine improves these variables above that of controls. It is important to note, however, that this is the only known controlled study to monitor resting heart rate with novel commuter cycling and, therefore, these results require confirmation.

5.4 Energy expenditure and body composition

Energy expenditure

Energy expenditure of CC, as estimated by heart rate while commuting and \( \dot{V}O_2 \) data from a lab-based sub-maximal cycling test, was approximately 5600 kJ/wk. The addition that cycling had on EE did not alter DXA measures of lean or fat mass in cyclists’ appendages, trunk or whole body. An absence of change to body composition and
energy intake despite an increase in EE further supports our suggestion that a decrease in non-cycling PA took place during the cycling intervention. How EE is affected long-term by introduction of a new PA habit (e.g. commuter cycling) has not been thoroughly investigated in healthy adults, and in general is under reported in physical activity interventions, particularly those using a free-living population (i.e. activity behaviour unmanipulated by participation in research) (Garland, et al., 2010). Wickel and Eisenmann (2006) are the authors of the only known study that has investigated compensation of EE in healthy adults. They reported that EE (assessed using the gold-standard, doubly-labelled water technique) from light-intensity activities to change inversely with the amount of moderate and vigorous PA performed by participants. In support of this, a meta-analytic review reported that within-individual variation of daily EE is relatively stable and low at 13% in a free-living population (Black & Cole, 2010). Therefore EE from MVPA may increase total EE, but only up to a point (i.e. 13%) before compensatory mechanisms limit further changes to total daily EE.

The role of exercise-induced EE on improved health parameters is widely accepted. However, the present study resulted in improvements to major cardiovascular markers of health without change to total EE through a compensation of light-intensity activities. The mechanisms involved in the regulation of EE are complex, involving hormones, circulating and stored substrates, feedback from peripheral tissues and substrate utilisation to higher centres (For review, see King et al, 2006; Garland, et al., 2010). The role of compensation of EE and the effects it has on cardiovascular gains from novel exercise warrants further investigation.
Body composition

Ten weeks of commuter cycling for 150 min/wk at moderate intensity (56 ± 11% \( \dot{V}O_{2\text{max}} \)) had no effect on adiposity in the present study. Møller et al. (2011) also investigated body composition change with novel commuter cycling. They reported that skin fold thickness decreased after eight weeks and more so in the cycling group than controls. Study characteristics were similar between the present study and that of Møller et al. (2011). Both utilised a randomised control group, included males and females who had similar body mass, BMI, and \( \dot{V}O_{2\text{max}} \). The only difference was that participants in Møller et al. (2011) were on average five years older. Møller et al. (2011) estimated 40 min of daily cycling (200 min/wk) was performed, which is 25% greater than cyclists from the present study. However, authors did not report the volume of cycling performed, but in the present study, participants averaged 5624 ± 1776 kJ of weekly cycling, based from sub-maximal \( \dot{V}O_2 \) testing and heart rate responses to cycling, which was insufficient to alter adiposity in normal-weight adults after ten weeks.

In contrast to DXA measures, manual measures of waist-to-hip ratio (WHR) decreased with CC at follow up, due to an increase in hip circumference in cyclists. WHR was similar (i.e. \( P=0.78 \)) between manual measures and DXA, indicating validity between the two measures at pre-intervention testing. However, circumference measures differed between instruments. The difference in waist measurements from manual measures and DXA remained consistent before (~4.8 cm) and after the intervention (~4.2 cm). However, the difference in hip circumference before (~6.3 cm) and after
~0.5 cm) the intervention was significant (p < 0.05). Unaltered DXA measures of lean and fat mass in the hip region combined with inconsistencies in manual hip circumference measures indicate that the measures contain errors, which produced a false finding in manual WHR results.

5.5 Blood parameters

Blood cholesterol

We observed no effect of 10 weeks of CC on triglycerides and cholesterol, which agrees with findings from Oja et al. (1991) who also observed no change in high-density lipoprotein (HDL) and low-density lipoprotein (LDL) cholesterol as well as triglycerides in their 10 week commuter cycling intervention. Commuter cycling for 26 and 52 weeks also appears to be insufficient to improve triglycerides and cholesterol fractions (de Geus, et al., 2008). A lower threshold exists for improvement to HDL with exercise training than LDL cholesterol (Mann, et al., 2014). Some evidence indicates a minimum amount of EE is required from PA to improve HDL and 4600 kJ/wk has been suggested as the lower threshold for improvement (Ferguson, et al., 1998; Mann, et al., 2014; Harrison, et al., 2012). Although we could not identify compensatory behaviours with CC, it is likely that some form of activity compensation reduced the impact of CC on total EE and thus HDL was unaltered from 10 weeks of CC. Furthermore, regular PA has been shown to increase lecithin cholesterol acyltrans enzyme, which promotes the transfer of cholesteryl ester to HDL and consequent removal of LDL through the liver (Riedl, et al., 2010; Calabresi & Franceschini, 2010). Without increased HDL from
CC, adaptations that reduce LDL were unlikely to have occurred. Even if the CC was continued over a longer period of time it is unclear if LDL would have improved, as changes to LDL with aerobic exercise with longer trials (e.g. 26+ weeks) is supported by some (Grandjean, et al., 1996; Tikkanen, et al., 1999; Dunn, et al., 1999), but not others (Wing, et al., 1998; de Geus, et al., 2008; Hersey, et al., 1994). Conflicting results are likely due to differences in health and fitness of participants at the onset of intervention between studies (Tambalis, et al., 2009). Furthermore, reduced LDL cholesterol is more responsive when accompanied with altered diet (Jenkins, et al., 2011; Kelley, et al., 2012) or with training at high-intensity (e.g. > 60% of VO$_{2}$max) (Ponjee, et al., 1995; Lindheim, et al., 1994; Tikkanen, et al., 1999). Neither scenario was observed in the present study, but LDL cholesterol was included as an outcome measure because de Geus et al (2007) observed commuters cycling at high-intensity (i.e. 75% VO$_{2}$max) in their study. But we observed commuter cyclists to generally cycle at moderate intensity, which was insufficient to alter cholesterol levels in 10 weeks.

Glucose and insulin

Commuter cycling did not alter blood levels of glucose and insulin after 10 weeks. To ensure our results were not affected by any acute effects, fasting blood samples were obtained between two and seven days after post-intervention VO$_{2}$max testing. Energy expenditure directly enhances insulin and glucose dynamics up to 24 hours from a single bout of exercise, whereas exercise training may offer sustained effects up to seven days of detraining (Devlin & Horton, 1985; Mann, et al., 2014). Others have used a
similar exercise load and duration to the present study and observed enhanced glucose regulation (Mourier, et al., 1997; Raz, et al., 1994). However, those studies included participant groups who had impaired glucose management. The present study used a younger group of adults (39 ± 7 years) without impaired blood glucose management. Data on changes to glucose and insulin dynamics with PA in non-elderly and non-obese adults with normal fasting blood glucose are surprisingly sparse (Sénéchal, et al., 2014). Participants from our study had low values of fasted blood glucose and therefore only small changes are likely to occur to glucose and insulin levels, which may be more difficult to detect.

Comparatively few data exist for contextualising our results; however the lack of observable change to EE has likely contributed to the absence of improvements to fasting glucose and insulin. Energy expenditure from moderate-intensity cycling among active and non-obese men has previously enhanced blood glucose management with 3760 kJ/wk, in which authors proposed as the minimum threshold to improve insulin sensitivity in that population (Magkos, et al., 2008). In support of findings from Magkos et al. (2008), insulin sensitivity was unaltered with exercise training that was below 3760 kJ/wk (Kang, et al., 1996; Cusi, et al., 2000). Evidence for the importance of intensity versus total EE on glucose management is unclear. Whether an increase in the intensity of activities without change to EE has the same or greater effect than total EE from PA of any intensity requires further investigation with normoglycaemic adult participants.
**C-reactive Protein**

C-reactive protein (CRP) was unaltered by 10 weeks CC. Others also observed commuter cycling to have no effect on CRP at weeks 26 and 52 (de Geus, et al., 2008). It is possible that the intensity of commuter cycling is not high enough to alter CRP, which has been suggested to respond best to high-intensity training, but more evidence on the role of intensity for this leading biochemical predictor for CVD is required (Plaisance & Grandjean, 2006; Chen, et al., 2014). Further obscuring change to CRP was the high prevalence of self-reported illness amongst cyclists towards the end of our CC intervention (*figure 4.1c*). Illnesses such as the common cold or any injury which results in an inflammatory response increases CRP, which may be responsible for the unexpected, but non-significant 34% mean increase we observed in CRP with CC (Haran, et al., 2013).

5.6 Health-related quality of life

Health-related quality of life (QOL) was not enhanced with CC; moreover, a trend for decreased social functioning was observed. Mean scores for social functioning were lower in both groups after the intervention and therefore were likely to have been caused by factors unrelated to the intervention per se, but rather to environmental factors (e.g. season, day length, weather). Utilising the same Short-Form 36 QOL questionnaire, de Geus et al. (2008) also observed changes in both participant groups, with enhanced role-physical and vitality reported in cyclists and controls after a 52
week CC intervention. The physical activity performed in our study, as well as that of de Geus et al., (2008), may have a lesser influence on QOL than factors outside of the intervention, evidenced by the changes in both participant groups in each study.

As previously mentioned the present study started in autumn and finished in winter, whereas participants from de Geus et al (2008) were tested in spring after CC in winter. Seasonal changes to QOL are well documented, with winter conditions worsening QOL measures (Lubetkin & Jia, 2009). Therefore a lack of improvements from CC in the present study, but enhancement is some QOL measures by de Geus et al. (2008) may be explained, in part, with timing of testing sessions in relation to the seasons. Differentiating the effect of QOL from seasonal changes and those from commuter cycling cannot be determined with current available evidence. Furthermore conclusions are further hindered by the scale of QOL instruments that are not purpose designed for young and healthy populations.

5.7 Conclusions

Commuter cycling (~150 min/wk) improves indices of cardiovascular health. These improvements in cardiovascular health occurred without measurable changes in overall physical activity, energy intake, estimated energy expenditure or body composition. It appears that more intensive commuter cycling replaced other lower intensity activities that were below the resolution of our instruments to quantify. Blood parameters were unaltered, but as a weekly minimum increase in EE of 4600 and 37600 kJ/wk are recommended to improve HDL cholesterol and blood glucose handling, respectively, it
may be that the estimated EE from cycling of 5600 kJ/wk was insufficient to alter total EE, or that the duration was too short to elicit these changes. It is also possible that compensation of EE may have reduced the metabolic strain that is necessary to substantially alter these parameters. Few data exist on the impact of adding a novel form of physical activity on other pre-existing physical activities (i.e. compensation) in healthy adults. Typically, research participants are asked to not alter anything else in their daily regimen. Although it appears that some compensation did occur in our intervention, this did not pre-empt the increase in cardiovascular fitness. Based on our findings it appears that self-paced, moderate-intensity commuter cycling can improve cardiovascular health without changes in energy balance or body mass or composition. Thus, one may conclude, that at an individual level, focussing on BMI or body mass may be counterproductive when attempting to encourage and assess the benefits of increased physical activity.
CHAPTER 6: Conclusions

Many people are not achieving public health recommendations for the amount of physical activity known to reduce risk for disease. A major barrier to increased participation of physical activity is the amount of free time perceived to be available (Sallisa, et al., 2015). Physically active modes of transport can support the accumulation of physical activity in a time efficient manner by incorporating physical activity in the time of day that would otherwise be used to travel. Additionally, government and health agencies are promoting use of physically active forms of transportation to improve health, reduce traffic congestion and pollution.

Our study adds clarity to existing knowledge of commuter cycling. We observed commuter cycling to enhance major markers of cardiovascular health in just 10 weeks, which offers health promoters evidence that cycling to work will have personal health benefits. Furthermore, an increase in energy expenditure is not necessary to reduce markers associated with risk of cardiovascular disease. However, our study has highlighted an under-reported issue in physical activity research, that adding novel daily PA may not increase total physical activity or energy expenditure in healthy adults. The extent that metabolic and cardiovascular strain was reduced due to the lack of increase in energy expenditure is unclear. Nevertheless, it is encouraging that other markers of cardiovascular health such as VO₂max and resting heart rate can improve, despite the small contribution cycle commuting made to participants' physical activity levels. In addition to these positive findings, the duration of time and physiological
intensity that people are willing to spend cycling to and from work has now been defined in a New Zealand context.

Results from the present study demonstrate that commuter cycling at moderate intensity for ~150 min/wk for ten weeks is not sufficient to improve body composition, health-related quality of life or select blood parameters (i.e. cholesterol, fasting insulin and glucose, C-reactive protein, or triglycerides) in healthy, normal weight adults. It is unknown if a greater amount of physical activity and energy expenditure, negative energy balance or duration of intervention is necessary to affect these parameters in this population.

Prospective research direction

Measuring the health gains acquired from novel commuter cycling is challenging for researchers. Short term trials lessen the opportunity for mechanisms to take place that enhance health, whereas longer trials report lower compliance, particularly through winter months. Further confounding our understanding of commuter cycling is that individuals who are likely to choose cycling as a means of transport over the conveniences of an automobile tend to be young or middle-aged adults who are already physically active (Damant-Sirois & El-Geneidy, 2015; Wanner, et al., 2012). Habitually active people generally have improved markers of cardiovascular health than their sedentary peers and have less to gain with novel PA. We therefore suggest that future research focus on defining the frequency, intensity and duration of cycling performed by habitual commuter cyclists. Furthermore, the load of commuter cycling should be
contextualised in rural verses urban environments and geographical location as it relates to weather and typography. If generalisations about the load of PA performed with commuter cycling can be defined, potential health benefits can then be extrapolated based on findings from numerous existing data on the effects of aerobic cycling on health.

Summary

Our results indicate that commuter cycling at a self-selected intensity is sufficient to enhance aerobic fitness, resting heart rate and prevent seasonal increases in diastolic blood pressure, which are all predictors of cardiovascular health. The addition of commuter cycling may result in a reduction in other forms of PA, although this doesn’t preclude all its health benefits. Noteworthy is that enhancement in cardiovascular health occur without significant alterations in energy expenditure, body weight or composition.
REFERENCES


Jenkins, D. J., Jones, P. J., Lamarche, B., Kendall, C. W., Faulkner, D., Cermakova, L., Gigleux, I., Ramprasath, V., de Souza, R., Ireland, C., Patel, D., Srichaikul, K.,


**APPENDIX I**

Participant ID ___________ (office use only)

Name: ___________________ Date: __________

The purpose of this questionnaire is to assess the types of physical activities you perform and the amount of time you spend performing each activity.

Please answer each question as completely as possible and feel free to use the bottom of the sheet if you need more space to write. If you have any questions, please ask James at novbr02p@otago.co.nz or 021-067-0519.

**Occupational activities:**

1. What is your occupation?

2. Which suburb do you work in?

3. How do you currently travel to work? (e.g. car, bus, walk, bike, etc)

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<th>Type of transport</th>
<th>Duration (each way)</th>
<th>Frequency (times per week)</th>
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<td><em>Example:</em> bus, then walk</td>
<td>12min (bus) &amp; 5min (walk)</td>
<td>10</td>
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4. How many minutes/hours a day do you spend standing while at work (approximately)?

5. How many days a week do you work?

6. Please list any physical activities you frequently perform while at work:
   *For example: walking, heavy lifting, talking the stairs, etc.*

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<th>Activity</th>
<th>Duration (minutes)</th>
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Recreational activities:

1. Have you competed in any sport competitions in the last 3 months? If yes, please provide details of the event(s).

2. have you trained or intend to train for a competition in the previous or next 3 months?

3. Please list any recreational activities you frequently perform:
   *for example: playing sport, gym workouts, hiking/walking, swimming, cycling, etc.*

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<tr>
<th>Activity</th>
<th>Duration (minutes)</th>
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4. Do you plan to start performing any new recreational activities in the next three months?

3. Do you plan to stop performing any of your current recreational activities in the next three months?
Activities of daily living:

1. Please list any recurring physical activities you perform around your home:
   *For example, mowing the lawns, gardening, walking the dog, playing with your children.*

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2. which suburb of Dunedin do you live in?
**APPENDIX II**

*Figure 4.4c* Time spent in total physical activity (mean ± SD) at week 0 and average of weeks 1-10 in cycling (n=13) and control (n=12) groups (one-way ANOVA for difference from baseline $P = 0.24$).

*Figure 4.4d* Time spent in vigorous and moderate intensity physical activities combined (mean ± SD) at week 0 and average of weeks 1 – 10 in cycling (n=13) and control (n=12) groups (one-way ANOVA for difference from baseline: $P = 0.003$).
Figure 4.4f Time spent in total physical activity (mean ± SD) outside of commuter cycling, at week 0 and average of weeks 1 through 10 in cycling and control groups (one-way ANOVA for difference from baseline: \( P = 0.23 \)).

Figure 4.4h Time spent in vigorous and moderate physical activity (mean ± SD) outside of commuter cycling, at week 0 and average of weeks 1 through 10 in cycling and control groups (one-way ANOVA for difference from baseline: \( P = 0.81 \)).