Does meeting physical activity guidelines in normal weight females influence body fatness?

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Abstract

Purpose: To investigate the associations among objectively measured physical activity and markers of body composition in normal weight, New Zealand European women.

Methods: Anthropometric measures were performed in 107 women aged 16-45 years with a BMI between 18.5 to 25 kg/m^2. Accelerometers were worn over 7 days to assess sedentary time (<100 counts per minute), light (100 - 2019), moderate (2200 - 5998) and vigorous (>5999) physical activity. Independent *t*-tests were used to compare associations between participants with normal (<30%) and high (≥30%) body fat. Partial correlations examined the independent associations of physical activity behaviours on body fat.

Results: Participants with normal body fat completed significantly more moderate to vigorous physical activity (MVPA) minutes per week (P = .002) and MVPA% (P =.021). Achieving current physical activity recommendations of ≥ 150 mins/week of MVPA, resulted in lower body fat (P =.038). Achieving ≥300 mins/week of moderate physical activity showed a trend towards significance for lower body fat (P = .076), while achieving ≥150 mins/week of vigorous activity showed significantly lower body fat% (P = .022). Partial correlations determined the significance of MVPA on body fat% independent of sedentary (r [104] = -.258 P = 0.008) and light activity (r [104] = -.273 P = 0.005).

Conclusion: Achieving current exercise recommendations was associated with lower body fat % in normal weight women. Our data suggest this association is stronger for vigorous activity, and is independent of the amount of sedentary activity achieved. Increasing vigorous physical activity may be important for improving body composition in this group.

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Contributions

Richard Swift (Masters Student). Performed the phlebotomy and processing of the blood samples* for the EXPLORE study, was responsible for the analysis of the accelerometer data and performed all the statistical analysis.

Dr Pamela von Hurst and Dr Sarah Shultz (Supervisors). Both Dr von Hurst and Dr Shultz were investigators on the EXPLORE study and helped with supervision of the writing of the thesis. Dr von Hurst also performed DEXA* assessments for the EXPLORE study.

Dr Philip Fink: Dr Fink developed the code for the MATLAB software that allowed the accelerometer data to be analysed

Dr Rozanne Kruger: Dr Kruger was the principal investigator on the EXPLORE study and also performed the BodPod and DEXA* assessments.

Dr Kathryn Beck, Dr Cathryn Conlon: Both were co – investigators on the EXPLORE project and also helped with data collection required for this project.

PhD Candidate: Wendy O'Brien: Wendy co-managed the EXPLORE study and also performed the BodPod assessment and the accelerometer station.

PhD Candidate: Shakeela Jayasinghe: Shakeela co-managed the EXPLORE study, managed the taste testing station* and also helped with data collection required for this project.

Masters Students: Zara Houston and AJ Hepburn: Both Zara and AJ helped with the initial screening for inclusion into the EXPLORE study, facilitated the completion of questionnaires* and helped with data collection required for this project.

*data from these assessments were not utilised in this study

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1.0 Introduction

1.1 Background

New Zealand is currently experiencing an obesity epidemic, it has one of the highest rates of obesity globally and obesity rates are continuing to rise, placing pressure on our health resources and infrastructure (Ng et al., 2014). Obesity is a well-known risk factor for Cardiovascular Disease (CVD) and other chronic illness, and being overweight and having a high body mass index is second only to tobacco use, as a risk factor contributing to ill health and disability and shortening life expectancy in New Zealand (Ministry of Health, 2014).

1.1.1 Body mass index

Body Mass Index (BMI, kg/m^2) is the most commonly used method to identify obesity, however, underlying the rise in obesity, is the increase in metabolic dysregulation that is being seen without obesity, and it has been suggested that BMI in its simplistic form cannot accurately characterise obesity (Oliveros et al., 2014). Fat distribution and percentage of body fat may be different in individuals with the same BMI, with characteristics of metabolic dysregulation exhibited that cannot be explained by BMI alone (Romero-Corral et al., 2008). This is a concern as many individuals, because of their weight and age may go undiagnosed until they have developed a chronic condition that may have been prevented with appropriate intervention. Further, specific areas of fat disposition are already recognised as increasing risk of disease, especially in the waist area (central obesity). This is seen independent of BMI, suggesting the obesity definition should be developed further to target these populations (Cleeman et al., 2001) .

1.2 Normal weight obesity

Recently, efforts have been made to classify obesity based on more specific anthropometric and metabolic profiles (Oliveros et al., 2014). A phenotype of metabolically obese but normal weight individuals has been proposed, but with differing measures of what defines metabolically obese. This has included hyperinsulinemia, presence of disease associated with obesity, impaired insulin sensitivity, meeting the criteria for metabolic syndrome and increased visceral adiposity among others (Oliveros et al., 2014).

It has been shown that adult women, who were classified as metabolically obese normal weight (MONW) due to decreased insulin sensitivity, determined by Homeostatic Model Assessment (HOMA), have a lower energy expenditure, are less aerobically fit and have increased total cholesterol levels when compared to metabolically healthy controls (Conus et al., 2004).

The concept of the Normal Weight Obese (NWO) has also been suggested, with the classification based on the amount of body fat rather than BMI or metabolic measures as described above. There is however, little consensus about a normal or ideal level of body fat. Neither is there agreement at to what percentage of body fat causes an increase in health risk. Underlying this, and in the context of this study, the association between physical activity and anthropometric markers has not been well documented in individuals classified with a normal weight.

The influence of physical activity or sedentary behaviour on the health risks of those with a normal BMI is not well understood. Increased physical activity is associated with lower body fat levels in a wide range of populations and weight categories (den Hoed & Westerterp, 2008; Sternfeld et al., 2005; Tucker & Peterson, 2003). Those classified as NWO showed a lower resting metabolic rate (RMR) (De Lorenzo et al., 2005) and in those classified as MONW physical activity energy expenditure was shown to be lower compared to a normal weight metabolically healthy control group (Dvorak et al., 1999). But no measure of physical activity behaviours have been utilised in a group of normal weight women.

1.3 Physical activity

The physical activity guidelines have a comprehensive evidence base for promoting health and reducing the risk of chronic disease. Until recently most national physical activity guidelines have focused on establishing behaviours based on the association of improved markers of health with moderate and vigorous physical activity (MVPA) (Katzmarzyk, 2010). This has been promoted rather than reducing the amount of sedentary behaviour achieved by an individual, although the evidence for increased adverse health risk caused by a lack of physical activity has been evident since the 1950's. Early research by Morris et al. (1953) showed that

London bus drivers, with a greater time spent sitting, had a higher relative incidence of myocardial infarction compared to conductors who spent less time sitting. Further evidence of the protective nature of physical activity came with the Harvard Alumni study (Paffenbarger et al., 1993). Using questionnaires to determine physical activity levels and two follow up periods over a 23-year period, results showed that small increases in energy expenditure, approximately 30 minutes walking per day, was associated with a 30% reduced risk of premature death.

The benefits of exercise have been shown for the prevention of chronic disease including cardiovascular disease and associated risk factors, specific cancers and diabetes (Warburton et al., 2006). But while there has been a focus on this public health message, there has also been a rapid rise in overweight and obesity, even with increases in leisure time physical activity witnessed in some countries (Bauman et al., 2008). The focus on MVPA, an important, but limited element in the context of the overall physical activity spectrum has been suggested as a plausible explanation for this paradox, as most waking hours are spent in sedentary behaviours, not at this activity level (Dunstan et al., 2010b).

Physical activity has shown benefits in preventing weight gain, with those performing more activity after a five year follow up compared to baseline showing weight loss suggesting a dose response for physical activity (Di Pietro et al., 2004). The effect of exercise intensity appears important, with vigorous activity resulting in lower body fat, as measured by skinfolds, and waist to hip ratio independent of the amount of total energy expenditure achieved (Tremblay et al., 1990). Further to this, middle aged and older women achieving recommended exercise guidelines showed lower visceral fat (Pelclova et al., 2012).

1.4 Sedentary behaviour

The impact of sedentary behaviour on disease risk is less understood when compared to physical activity. Sedentary behaviour has been defined by three basic behaviours, where the individual expends very little energy, $\leq$ 1.5 metabolic equivalents, they are sitting or lying down and they are awake (Sedentary Behaviour Research Network, 2012), with common sedentary behaviours including

TV viewing, video game playing, computer use, driving automobiles and reading (Pate et al., 2008). Overseas evidence suggests that the majority of adults non-sleeping time is spent being sedentary (57%) with light intensity (39%) and MVPA (4%) making up the remainder (Healy et al., 2008c). Because of the amount of time spent in sedentary behaviour, assessing the impact of this behaviour has become a focus for many health researchers, as the majority of an individual's waking day is spent in sedentary activity. Therefore, strategies focussing on reducing this time have been theorised to have a major effect on the risk profile of an individual, independent of the amount of physical activity they perform (Owen et al., 2010).

Studies evaluating the impact of sedentary behaviour on health have highlighted the importance of this association, with research indicating that sedentary behaviour is a risk factor for cardiovascular disease independent of time spent in moderate to vigorous physical activity (greater than three metabolic equivalents) (Healy et al., 2008b; Healy et al., 2008c). However recent evidence has emerged suggesting this relationship is not so clear, with physical activity attenuating the association between sedentary behaviour and metabolic syndrome (Scheers et al., 2013).

1.5 Summary

The mechanisms by which sedentary behaviours increase disease risk and mortality are unclear. Correlation evidence suggests that high levels of sedentary behaviour can impact negatively on health, and are associated with being overweight and obese (Cameron et al., 2003; Hu et al., 2003; Tudor-Locke et al., 2010), an increased risk of various chronic diseases such as the metabolic syndrome (Healy et al., 2008c), diabetes (Healy et al., 2008b; Hu et al., 2003), cardiovascular disease (Ford & Caspersen, 2012; Katzmarzyk et al., 2009; Warren et al., 2010) and premature mortality (Dunstan et al., 2010a; Katzmarzyk et al., 2009). Furthermore, evidence examining the association between sitting and mortality, after controlling for bodyweight, showed that sitting remained a significant predictor of mortality (Katzmarzyk et al., 2012).

While there is an established link between sedentary behaviour and the development of obesity, there is little known about sedentary behaviour, total body

fat and regional adiposity. A recent study identified no relationship between regional body composition and sedentary behaviour, although this was performed in a sample of inactive, overweight to obese individuals (McGuire & Ross, 2012).

Further to this, there is currently little known about the epidemiology of sedentary behaviour of New Zealand women of European ancestry, and no specific guidance in our national activity guidelines, on how to reduce sedentary behaviour, and to what level.

2.0 Aims and objectives

2.1 Aim

To objectively assess sedentary behaviour and physical activity levels in New Zealand European females aged 16 – 45 with a normal BMI, and compare this to body fatness, central adiposity and blood pressure as surrogate measures of health risk .

To assess the comparable effects of sedentary behaviour and MVPA on these health markers to help inform public health policy on promoting reductions in sedentary behaviour, increasing physical activity or both.

2.2 Objectives

1. Assess accelerometer data for New Zealand European women with a normal BMI and stratify participants into groups based on body fat percentage levels and achievement of 150 minutes of MVPA activity per week.
2. Examine relationships between anthropometric measures and differing levels of physical activity.

2.3 Hypotheses

Null	That the anthropometric measures will not differ between the group that achieves 150 minutes of MVPA activity and the group that does not achieve this level of activity.
Experimental 1.	That the measured group will exhibit similar profiles to those witnessed in overseas research of approximately 60% of waking time spent in sedentary behaviour.
Experimental 2.	That participants who achieve higher levels of sedentary behaviour will exhibit poorer anthropometric measures than those with less time spent in sedentary behaviour.

3.0 Review of the literature

3.1 Introduction

Considering that human movement is a complex behaviour that is influenced by personal motivation, health and mobility, genetics and the social and physical environments in which people live, aiming to limit sedentary behaviour, rather than focusing on Moderate to Vigorous Physical Activity (MVPA) has become a potential public health paradigm shift (Katzmarzyk, 2010). This study aims to assess the links between different body fat profiles in normal weight individuals, and therefore the risk for chronic disease, as suggested through anthropometric measures of height, weight, BMI, body fat percentage and blood pressure. It also aims to assess how physical activity patterns may impact on this risk. Evidence has shown that individuals with normal weight and normal BMI values can have excessive body fat and this 'hidden' body fat or normal weight obesity is a key factor in the emerging obesity epidemic and related disease risk (Oliveros et al., 2014). How physical activity (PA) or sedentary behaviour affects this body fat profile in normal weight females is not well understood.

3.2 Epidemiology of health in New Zealand

In order to understand the significance of excess body fat on the health risk of the New Zealand population, a brief review of the epidemiology of chronic disease follows.

3.2.1 Obesity

The World Health Organization (WHO) released an international classification of obesity in 2000 defining the classification of weight and obesity based on BMI values (Table 1). New evidence shows that the levels of overweight and obesity in New Zealand are among the highest in the developed world with 71.4% (95% Uncertainty Index: 69·6–73·3%) of Men $\geq$ 20 years and 60.0 % (57.8 – 62.2%) of women $\geq$ 20 years obese or overweight (Ng et al., 2014).

Table 1: The international classification of adult underweight, overweight and obesity according to BMI

Classification	BMI(kg/m^2) cut-off points
Underweight	<18.50
Severe thinness	<16.00
Moderate thinness	16.00 - 16.99
Mild thinness	17.00 - 18.49
Normal range	18.50 - 24.99
Overweight	≥25.00
Pre-obese	25.00 - 29.99
Obese	≥30.00
Obese class I	30.00 - 34.99
Obese class II	35.00 - 39.99
Obese class III	≥40.00

WHO 2000 (World Health Organization, 2000).

This is significant, especially as the American Heart Association and the American College of Cardiology guidelines present obesity as a major modifiable, CVD risk factor (Eckel et al., 1998). Obesity is associated with higher rates of CVD, diabetes and hypertension as well as forming part of the metabolic syndrome. Obesity has also been linked with obstructive sleep apnoea, non-alcoholic fatty liver disease, breast and colon cancer.(Eckel et al., 1998) Further, obesity is associated with an increase in mortality in a normally healthy population (Calle et al., 2005).

However BMI, although relatively easy to measure, fails to account for behaviours, such as physical activity, and the associated improvement to cardiorespiratory fitness which is associated with reductions in CVD and mortality (Janiszewski & Ross, 2007). Further, waist circumference and visceral fat may be reduced in response to physical activity, with little or no change to body weight (Ross & Janiszewski, 2008). Ethnic differences in disease risk for any given BMI are also evident, with research from the Nurses' Health study showing after a 20 year follow

up, women of Asian descent had double the risk of developing type 2 diabetes compared to Caucasians of the same BMI (Shai et al., 2006).

3.2.2 Cardiovascular disease

CVD is the leading cause of death and hospitalisation in New Zealand, with 2005 measures suggesting that in those aged 35 or older, approximately 151,000 have experienced a heart attack, stroke or have angina (Wells et al., 2006). When considering risk of experiencing a new CVD event in five years, men are three times as likely as pre-menopausal women to be considered at high or very high risk. In developed countries, after menopause, CVD becomes the leading cause of mortality and morbidity in women aged over 50 (Rosano et al., 2007).

Hypertension, cigarette smoking, obesity (BMI $\geq$ 30), physical inactivity, dyslipidaemia, diabetes mellitus, microalbuminuria or an estimated Glomerular Filtration Rate <60 mL/min, age (>55 years for men, >65 years for women), or a family history of premature cardiovascular disease (men <55 years or women <65 years) are all established risk factors for CVD (Chobanian et al., 2003).

3.2.3 Hypertension

Elevated blood pressure is caused by increases in cardiac output, total peripheral resistance or both (American College of Sports Medicine, 2010). Cardiac output is increased by any condition that increases heart rate or stroke volume, whereas peripheral resistance is increased by any factor that increases blood viscosity or reduces vessel diameter, particularly arteriolar diameter. These increases lead hypertension to be a major risk factor worldwide for heart attacks, strokes, kidney disease and premature death (American College of Sports Medicine, 2010).

The Seventh Report of the Joint National Committee on Prevention, Detection Evaluation and Treatment of High Blood Pressure (JNC7) Classifies blood pressure into four stages (Table 2).

Table 2: Classification of blood pressure

BP classification	Systolic BP, mm Hg		Diastolic BP, mm Hg
Normal	<120	And	<80
Prehypertension	120-139	Or	80-89
Stage 1 hypertension	140-159	Or	90-99
Stage 2 hypertension	≥160	Or	≥100

Adapted from (Chobanian et al., 2003)

Data from the NZ health survey shows a mean (±SD) systolic BP of 126 ± 18 mm Hg for adults aged 15 and over with men 130 ± 16 mm Hg and women 122 ± 19 mm Hg. Mean diastolic BP was 75 ± 12 mm Hg, with men 75 ± 12 mm Hg and women 73 ± 12 mm Hg (McLean et al., 2013). The prevalence of hypertension as defined by systolic BP ≥ 140mm Hg and diastolic ≥ 90 mm Hg or self-reported use of anti-hypertensive medication was 30.8% (95% CI 28.7, 32.9) with aged standardised data showing a prevalence of 29.3% (95% CI 26.7, 31.9) in men and 22.7 (95% CI 20.7, 24.6) in women.

3.2.4 Diabetes

Using a glycated haemoglobin (HbA1c) level of ≥ 6.5% as a measure of undiagnosed diabetes and an HbA1c result between 5.7% (39mmol/mol) and 6.4% (46mmol/mol) as pre diabetes, a sample of 4,721 New Zealanders, as part of the 2008/09 New Zealand Adult Nutrition survey, showed a prevalence of diabetes of 7.0% and prevalence of pre diabetes of 25.5% (Coppell et al., 2013). The prevalence of diabetes was higher in men (8.3%; 95% CI: 6.4, 10.1) than in women (5.8%; 95% CI: 4.7, 7.0) and showed a higher prevalence in those measured as obese (14.2%; 95% CI: 11.6, 16.9) compared with the normal weight group (2.5%; 95% CI: 1.4, 3.6).

3.2.5 Metabolic syndrome

Using the 2001 Third Report of the Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (ATP III) definition (Table 3), the prevalence of metabolic syndrome in 4022 participants in the Auckland region showed that 32% of Maori and 41% of Pacific People had significantly higher prevalence than other ethnicities 16% (mostly European) (Gentles et al., 2007).

Table 3: Clinical identification of the metabolic syndrome

NCEP ATP III Diagnosis is based on any three of the following:

Risk Factor	Defining Level
Abdominal obesity	Waist circumference
Men	>102 cm
Women	>88 cm
Triglycerides	≥1.69mmol/L
HDL cholesterol	
Men	<1.03mmol/L
Women	<1.29mmol/L
Blood pressure	≥130 / ≥85mmHg
Fasting glucose	≥6.1mmol/L

Adapted from (*Third report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (adult treatment panel III)* 2001)

3.3 Normal weight obesity

As discussed, the global prevalence of obesity has doubled in the past 30 years and is now considered an epidemic, with New Zealand exhibiting overweight and obesity levels that are some of the highest in the developed world (Ng et al., 2014). Much research has focused on this problem including best practice in exercise volume, intensity and diet therapy, with a review of the subject showing a lack of recent evidence and methodological concerns such as confounding issues, reverse causality and measurement errors influencing results and therefore recommendations (Wareham et al., 2005). As yet there is still conflicting evidence over the best way to avoid weight gain, Hu (2008) proposes that most cross sectional studies show a strong inverse association between physical activity and weight gain, but the direction of causality is unclear. It is further suggested that prospective studies show a general, but inconsistent trend, that increasing physical activity attenuates weight gain or waist circumference during midlife (Hu, 2008).

Individuals with a normal BMI and high body fat percentage have been shown to have a high degree of metabolic dysregulation, with an increased risk for metabolic syndrome, cardio-metabolic dysfunction and higher mortality (Oliveros et al.,

2014). With this increase in knowledge there have been calls for an updated definition of obesity based on adiposity, not on body weight.

The American Association of Clinical Endocrinologists define obesity as a BMI greater than 30 kg/m^2 as a proxy for excessive adiposity (Mechanick et al., 2012), however there is no consensus on how to define an excessive fat mass range, either by absolute measures or relative body fat percentage. Sex and age differences have also been proposed but there is no consensus about the best cut-off to define excessive body fat, with levels from 20 – 25% for men and 30 – 37% for women being proposed (Oliveros et al., 2014).

Although BMI is a popular epidemiological measure due to its ease of use, its main limitation is that it cannot differentiate body fat from lean mass and central from peripheral body fat. The classic subgroup used to explain this limitation is athletes, who through enhanced lean muscle tissue may be classified as obese when using only BMI to define obesity and conversely, individuals with low lean muscle tissue but high body fat levels may present with a normal BMI.

As yet it is unclear as to what extent metabolically dysregulated individuals, in the sub group of individuals with a normal BMI, could have this dysfunction explained by increases in total body fat, high body fat percentage or increased visceral fat.

3.3.1 Metabolic measures of obesity

Assessing participants with normal weight and BMI, Karelis et al. (2004) described subgroups of individuals who display multiple metabolic abnormalities including insulin resistance, hyper-insulinaemia, and dyslipidaemia. They suggest that the presence of metabolic and cardiovascular disease may go undetected for years because of the general characteristics of young age and normal body weight in these individuals.

Dvorak et al. (1999) assessed young women with normal BMI (Defined as <26.3 kg/m^2) and either with or without metabolic dysregulation measured by insulin sensitivity using the hyperinsulinaemic/-euglycemic clamp. Plasma insulin concentration was raised by insulin infusion into a peripheral vein, and kept at a

constant rate of 240 pmol/m $^{-2}$. A variable infusion of a 20% dextrose solution was then used to keep plasma glucose concentration constant. This steady-state condition of euglycemia, was used to assess insulin sensitivity via blood glucose measures as the dextrose infusion rate equals the glucose uptake by all the tissues in the body , with the average rate of glucose infusion during the last thirty minutes of the test determined the insulin sensitivity. Low results (<8.0 mg/min/kg lean body mass) indicating low insulin sensitivity and high results (>8.0 mg/min/kg lean body mass) suggesting high insulin sensitivity. Whilst there was no difference in age, body mass, BMI, and fat free mass between the groups, total fat mass, body fat percentage truncal fat and subcutaneous and visceral adiposity was significantly higher in the group with lower insulin sensitivity (31.8 ± 5.9%) compared to the normal group (27.4 ± 5.5%). The author's referred to this group as metabolically obese normal weight (MONW). Subcutaneous fat and visceral fat as measured by DXA was also significantly higher in the MONW.

Dvorak et al. (1999) also measured physical activity energy expenditure by doubly labelled water and found that this was significantly lower (- 412kcal) in the MONW group. This led the investigators to suggest that physical activity may affect insulin sensitivity and other CVD risk factors through its effects on body composition, and the lower levels of physical activity seen in the normal weight metabolically obese group may favour positive energy balance therefore influencing an increase, in part, of total fat mass.

3.3.2 Body fat measures of obesity

De Lorenzo et al. (2005) used the term normal weight obesity (NWO) to describe those individuals with a normal body weight based on BMI (<25 kg/m^2) with increased body fat percentage (>30%). An evaluation of 74 females with normal BMI showed that those classified as NWO (N = 28) and pre obese (BMI >25 kg/m^2, body fat percentage >30%, N = 26) had significant differences from the control group (BMI <25 kg/m^2, body fat percentage <30%, n = 20) in HDL cholesterol and the LDL/HDL cholesterol ratio. Those in the NWO group had a significantly lower resting metabolic rate (RMR) compared to the pre obese group of approximately 200 kcal/day, as well as significantly lower oxygen consumption.

The authors suggested that this was due to reduction in metabolically active fat free mass.

Using the same criteria as De Lorenzo et al. (2005) above, Di Renzo et al. (2010) showed that the NWO and pre-obese-obese women (defined as BMI > 25 kg/m^2 and clinically healthy), had significantly greater oxidative stress, with reduced glutathione and nitric oxide metabolites, when compared to healthy controls. The authors highlight this specifically, as oxidative stress is a central pathogenic mechanism in obesity related metabolic syndrome. It also plays an important role in the development of atherosclerosis, cancer, CVD and diabetes mellitus. Using BMI as a measure to identify at-risk individuals will miss a significant proportion of the population that may be at risk of developing these conditions.

Further Romero-Corral et al. (2010) has identified that NWO is associated with increased CVD mortality. Using the highest tertile of body fat percentage (> 33.3% in women and >23.1% in men) to identify NWO, 6171 participants from the National Health and Nutrition Examination Survey III (NHANES), were followed for a median of 8.8 years. Women in the NWO group were 2.2 times more likely to die from CVD, compared to the lowest body fat percentage group. The NWO group also displayed a higher prevalence of dyslipidaemia and CVD, with men only showing higher incidence for hypertension.

The understanding of how physical activity or sedentary behaviours affects the NWO group has not been well researched. To our knowledge there have been no studies examining the relationship between body composition and physical activity behaviours in this group.

Evidence from Scheers et al. (2013) showed that physical activity had a significant effect in both men and women and an effect for sedentary behaviour in women on body fat percentage. However no interaction effects were observed, so those who spent >60 mins/day in MVPA had significantly lower body fat percentage than those who did not achieve this target, irrespective of the amount of time spent in

sedentary behaviour. This research was conducted in men and women within a normal to overweight BMI range (26.0 ± 3.8 and 24.3 ± 3.9 kg/m^2 respectively).

A recent study looking at athletes used self-reported sedentary time using the International Physical Activity Questionnaire (IPAQ) and found that sedentary behaviour predicated total fat mass and trunk fat mass but not abdominal fat and that these results were independent of age, weekly training time and residual mass (Judice et al., 2014). This indicates, conversely to Scheers et al. (2013), that high MVPA levels did not influence the association of sedentary behaviour and body fatness in a highly trained population.

3.3.3 Summary of section

Although there is evidence that increasing sedentary behaviour increases body composition measures unfavourably, this has not been researched in normal weight individuals with differing body fat profiles. This is important as people with normal BMI and high BF are at an increased risk for metabolic dysregulation, inflammation and mortality, and that this increased risk may be related to increased body adiposity that is not detected by BMI (Oliveros et al., 2014).

3.4 Physical activity

3.4.1 Physical activity versus physical fitness

Physical activity has been defined as any bodily movement produced by skeletal muscles, resulting in energy expenditure. (Caspersen et al., 1985) This energy expenditure can be measured (In kilojoules and/or kilocalories) and may be categorised into occupational, sports, conditioning, household or other activities.

Exercise is a subset of physical activity, which constitutes similar characteristics with physical activity, but is not synonymous. Exercise is planned, structured and repetitive movement, performed to improve or maintain one or more components of physical fitness NIH (National Institutes of Health) (1996).

Physical fitness contrasts with physical activity, in that it is considered a set of attributes that people may have or may achieve, rather than movements that they perform. This may include concepts of vigour, alertness, fatigue and enjoyment

which are not easily measured. Other health concepts of physical fitness are measureable such as cardiorespiratory endurance, muscular endurance and strength, body composition and flexibility (Caspersen et al., 1985).

3.4.2 MET and MVPA definition

Measures of activity and fitness have been designed to allow researchers to quantify activity and/or exercise habits. Commonly used is the concept of metabolic equivalents (MET). A MET can be defined as the resting metabolic rate, or conventionally the amount of oxygen consumed at rest, sitting quietly in a chair, which is approximated to 3.5ml O_2/kg/min (Jetté et al., 1990). The MET of a task is used to indicate the amount of oxygen the body uses during physical activity, and it express the ratio of that task compared to the individual's metabolic rate at rest. Practically it allows comparison of the level of exertion and energy cost of the same physical activity task performed by people of different weight. It also can allow comparison of the aerobic intensity and energy expenditure of various physical activities.

Physical activity performed at an intensity of 3-6 METS is considered moderate, while activities with an intensity level greater than 6 METS are considered vigorous (Pate et al., 2008). It should be noted that these values are arbitrary, and the associated percent of maximal capacity may vary with age and physical fitness (Hu, 2008). Further MET values derived from normal weight subjects may be less accurate when compared to obese subjects (Saris et al., 2003). The metabolic cost of activity has generally been derived from younger subjects and tend to overestimate energy expenditure in older adults (Rikli, 2000) .

3.4.3 Benefits of physical activity

Physical activity has been widely postulated as a method of staving off chronic disease for over 50 years (Haskell et al., 2007). Recently a paradigm shift has occurred which suggests that sedentariness will also have an impact on chronic disease and health risk. The early evidence by Morris et al. showed that more sedentary bus drivers and mail sorters had a higher incidence of heart attacks, than more physically active bus conductors and postmen (Morris et al., 1953). Other

occupational based studies confirmed these findings and were used to quantify the level of physical activity by occupation (Paffenbarger & Hale, 1975; Paffenbarger et al., 1970). Although more recent evidence has suggested the benefits of occupational activity are not as clear as once believed, with long term sickness absence (LTSA) increased with moderate and high occupational physical activity (Holtermann et al., 2011). The authors noted a reduction in LTSA with increased leisure time activity, highlighting the differences in mechanical, repetitive type activity (occupational) versus more cardiovascular, dynamic styled exercise with the ability to rest once fatigue sets in with leisure time activity.

These paradoxes aside, it is generally accepted based on existing evidence that physical activity is associated with health benefits, particularly cardiovascular disease, which is one of the leading causes of death. Also, most research is based on leisure time activity, and due to the decrease in physical activity of most modern day jobs, this is perhaps more relevant to the current population than the earlier studies cited above. Generally, the health benefits of physical activity have been well researched, with the suggestion that there is a linear relationship between physical activity and health status (Warburton et al., 2006). A brief review of these benefits is discussed and specific mechanisms for physical activity improving health are examined, with a specific focus on women, where available.

3.4.4 Mortality and physical activity

The updated Surgeon General 2008 Guidelines for Physical Activity, involved a thorough review of all studies relating to physical activity and mortality (Physical Activity Guidelines Advisory Committee, 2008). This included 73 studies, mainly prospective cohort studies, published up to 2008. Of these studies, 67 reported a significant reduction of risk in the physically active groups or those with the highest physical activity compared with no physical activity or the lowest physically active groups. The results suggested a 30% reduction in the risk of death.

Similarly, a recent meta-analysis of 21 prospective studies on physical activity and cardiovascular disease from 1980 to 2010 showed a relative risk (RR) of 0.76 (95% CI 0.70 – 0.82, P <0.001) for men and 0.73 (95% CI 0.68 – 0.78, P <0.001) for women for developing CVD when high leisure time physical activity was compared

with low leisure time physical activity (Li & Siegrist, 2012). Other recent meta analyses have suggested women show a greater reduction in risk attributable to physical activity, when compared to men (Brown et al., 2012; Löllgen et al., 2009; Woodcock et al., 2011).

This gender effect is may be explained by various observations. Women are protected against CVD up until the menopausal period, whereby the risk of CVD events increases (Löllgen et al., 2009). Further as women age, the least active group in women is generally less active than the least active group in men, maybe explaining the apparently stronger association in older men compared to older women (Löllgen et al., 2009). Also as there are fewer cohort studies with women tan with men, there is chance for a larger standard error in combining the effect of these studies, which may result in selection bias of the various meta-analysis performed so far (Löllgen et al., 2009).

3.4.5 Cardiovascular disease, coronary heart disease and physical activity

The evidence from observational epidemiological studies to support an inverse relationship between physical activity and CVD and Coronary Heart Disease (CHD) risk has been described as substantial (Shiroma & Lee, 2010).

Studies on the Framingham cohort have shown there to be an inverse association with physical activity levels and the development of CHD and overall CVD mortality (Reiner et al., 2013). Data from the Harvard Alumni health study looked at stroke (Lee & Paffenbarger, 1998) and CHD (Sesso et al., 2000) with results showing there was a U-shaped pattern for energy expenditure and RR, with 2000 – 2999 kcal of additional energy required to maximally reduce stroke. CHD showed the same U-shaped curve, but the energy expenditure required for risk reduction was lower at 1000 kcal. The National Health Nutrition Examination Study 1 (NHANES 1) follow up study, of 5852 men and women aged 24 – 74 at baseline, showed the same U-shaped pattern for men, but greater physical activity was negatively associated with the incidence of stroke in women (Gillum et al., 1996). Nocon et al. (2008) in a meta-analysis, showed a pooled relative risk ratio (RRR) of 35% for CVD in both men and women with an all-cause mortality RRR of 33%.

A seminal review on physical activity and CHD showed an inverse association between physical activity and CHD incidence and that the RR of inactivity was similar in magnitude to hypertension, hypercholesterolemia and smoking (Powell et al., 1987). Macera et al. (2003) highlights this as an important concept, as being physically inactive or sedentary is more prevalent in the general population than smoking or hypertension, thus making this an important public health risk measure. An early, comprehensive, meta-analysis on physical activity and CHD followed this review, and showed an increased relative risk (RR) of death from CHD for sedentary individuals, that was almost double of that for active individuals (Berlin & Colditz, 1990).

Although early research did not generally include women in the analysis, evidence would suggest that women respond well to lower intensity activities. Females in the Nurses' Health Study showed an inverse relationship for total mortality and total physical activity (Rockhill et al., 2001). The greatest decrease in risk occurred with an increase in activity from less than one hour per week to 1.9 hours per week, with the remaining decreases in risk with increasing duration suggested to be relatively minor. A meta-analysis of women only, showed that there was a dose response relationship between physical activity and reduced risk of CVD in women, with similar results found for CHD and stroke, which is consistent with reviews of male data on CVD and CHD (Oguma & Shinoda-Tagawa, 2004).

3.4.6 Mechanisms effecting cardiovascular health

3.4.6.1 Improved body composition

The Aerobics Centre Longitudinal Study (ACLS) showed that daily physical activity was negatively associated with weight gain in 2501 healthy men aged 22 – 55, when baseline weight measures where compared to results from a five year follow up (Di Pietro et al., 2004). Those whose physical activity levels increased over the follow up period showed weight loss whereas those whose physical activity levels declined showed weight gain

The CARDIA study included 4995 men and women between 18-30 years at baseline, who were re-examined after 2, 5, 7, 10 and 15 years (Gordon-Larsen et

al., 2009). After 15 years a negative association was shown between 30 minutes of walking per day and weight gain and an increased likelihood of weight loss. This association was dependent on baseline weight with those in the lowest 25th percentile of baseline weight showing no association with weight gain whereas those in the 50th and 75th percentiles showed delayed gain with 30 minutes walking.

Not only does regular physical activity appear to have a positive effect on preventing weight gain and promoting weight maintenance, but also in the redistribution of this weight. Middle aged and older women who achieved the recommended physical activity guidelines showed lower visceral fat, with no difference noted in the Fat Free Mass Index (FFMI) (Pelclova et al., 2012). Of note in this study was that 76.6% of participants achieved the physical activity Guidelines for America (accumulation of 150 minutes per week), with only 41.9% achieving the ACSM/AHA recommendations of moderate physical activity for 30 minutes, five days per week, with those achieving 150-300 minutes of accumulated activity per week having significantly worse body composition values than those achieving greater than 300 minutes per week. The researchers also showed that achieving 10,000 steps per day exhibited slightly better body composition values than the previously mentioned groups. This highlights the challenge in providing effective public health messages, with different recommendations achieving different results.

Finally, a recent study by Larsen et al. (2014) also showed that greater physical activity was associated with less visceral fat, as well as lower intra-thoracic subcutaneous and intramuscular fat in older adults (mean age 65), with stronger associations seen for men than women.

3.4.6.2 Effects of physical activity on blood pressure

A recent review showed that both aerobic exercise (30 to 40 minutes of training at 60 – 85% of predicted maximum heart rate, most days of the week) and resistance training (three to four sets of eight to 12 repetitions three days per week) significantly improves blood pressure (Pal et al., 2013). Aerobic exercise also exhibited improvements in the augmentation index (an assessment of arterial stiffness), carotid artery compliance and restoring vascular endothelial function.

3.4.7 Diabetes and physical activity

The development of type 2 diabetes has a known behavioural component as well as genetic factors which include being overweight or obese, improper nutrition and physical inactivity (Williamson et al., 2004). Regular physical activity has been shown to not only lower the risk of developing diabetes but can prevent and/or reduce the complications of the disease. In the primary prevention of type 2 diabetes, weight loss achieved through diet and exercise, both aerobic and resistance training, reduced incidence among high risk individuals by 40 – 60% over four years (Williamson et al., 2004).

A recent meta-analysis showed physical activity alone to be more effective at preventing type 2 diabetes, than most medications (excluding glitazones) but not as effective as physical activity and diet interventions and bariatric surgery (Merlotti et al., 2014) (It should be noted that the bariatric group had a significantly larger BMI to the other participants included in the analysis).

3.4.8 Mechanisms effecting diabetes risk

3.4.8.1 Increased insulin sensitivity

Regular physical activity has been shown to be a major determining factor for insulin sensitivity, independent of the intensity of exercise or the time spent in sedentary behaviours (Balkau et al., 2008). Physically trained subjects show greater insulin sensitivity when compared to untrained subjects, demonstrating increased up regulation of muscle GLUT 4 protein, increased muscle enzyme capacities and muscle capillarisation (Borghouts & Keizer, 2000). Further, in people with obesity, physical training induces beneficial changes in blood lipid profiles, lipid metabolism and body composition values that are known to positively influence insulin sensitivity.

3.4.8.2 Improved lipid profile

The association with serum cholesterol levels and CVD outcomes has been well established, with LDL- C and apolipoprotein B shown to have the highest correlation (Di Angelantonio et al., 2009). Physical activity has shown a correlation with reducing triglycerides, apolipoprotein B, increasing HDL – C and changing LDL particle size, independent of weight change, all of which have been shown to

be significant risk factors for CVD. The change in LDL particle size, rather than reducing LDL -C concentration is believed to be a major factor in how exercise reduces CVD risk (Strasser, 2013).

3.4.9 Osteoporosis and physical activity

Routine physical activity may have a direct impact in primary prevention of osteoporosis by preventing loss of bone mineral density especially in post-menopausal women and the elderly (Warburton et al., 2006). Musculoskeletal fitness is associated with improved glucose control, functional independence, mobility, psychological wellbeing and quality of life, and negatively associated with fall risk and premature mortality (Warburton et al., 2001).

3.4.9.1 Physical activity associations with musculoskeletal health

Evidence suggests that enhanced musculoskeletal fitness benefits overall health and is associated with a reduction in the risk of disease and disability, and routine physical activity has been shown to improve musculoskeletal fitness (Warburton et al., 2006). Mason et al. (2007) showed that musculoskeletal fitness was a significant predictor of weight gain during a 20 year follow up with low musculoskeletal fitness associated with significantly higher odds of gaining 10 kg in this time (OR: 1.78, 95% CI: 1.14–2.79).

3.4.10 What are the guidelines for physical activity?

Early exercise recommendations were developed to combat the rising cost of cardiovascular disease, and specifically guide clinicians working in cardiac rehabilitation (Blair et al., 2004). Because of the increase in the evidence linking physical fitness to a wide array of physical and mental health benefits the American College of Sports Medicine (ACSM) released recommendations for the dose of physical activity required to improve physical fitness in 1978 which included 15-60 minutes per day of moderate intensity physical activity, to be performed 3- 5 days a week (Blair et al., 2004). These were subsequently updated in 1990 and 1998, when there started to be a shift away from performance related fitness to including recommendations for both performance and health related outcomes. A focus on lower-intensity exercise, and cumulative totals (10 min minimum) rather than continuous exercise were the general consensus for the later position stands.

This focus on lower intensity was to aim to improve the activity levels of the estimated 40 – 50 million US adults who were considered sedentary at the time (Blair et al., 2004). Evidence had emerged that showed the health benefits could be accrued with moderate amounts and intensities of regular exercise, and this sedentary population was unlikely to have a physical capacity required to engage in greater quantities of higher intensity exercise. The 1998 position stand also included a recommendation for resistance training (1 set of 8-12 reps for 8 – 10 exercises), highlighting the growing importance of this mode of exercise. Recommendations from the Surgeon General's Office (1996) and the American Heart Association (AHA, 2000) suggested similar durations and intensities.

In contrast, the Institute of Medicine (IOM) released guidelines in 2002, called for 60 minutes of activity per day, mainly aimed at countering the rising obesity epidemic, suggesting that 30 minutes of activity was insufficient to affect weight control (Blair et al., 2004). The most recent recommendations for adults issued by the ACSM and AHA suggest that adults aged 18 – 65 complete moderate intensity aerobic (endurance) exercise for a minimum of 30 minutes five days per week or vigorous intensity exercise for 20 minutes three days per week.

However, as the ideal combination of frequency, intensity, time and type of exercise remains unclear, the 2008 Surgeon General's Physical Activity Guidelines for Americans are simpler, in the suggestion that an individual accumulate 150 minutes per week of various moderate intensity activities or 75 minutes per week of vigorous intensity activity. This was also adopted by the WHO in their global physical activity guidelines released in 2010.

In New Zealand, the Guidelines for Physical Activity, released in 2005 were developed by Sport and Recreation New Zealand, and adapted from the US Surgeon General recommendations (1996). They promote four aspects of physical activity:

1. View movement as an opportunity, not an inconvenience.
2. Be active every day in as many ways as possible.

3. Put together at least 30 minutes of moderate-intensity physical activity on most if not all days of the week.

4. If possible, add some vigorous exercise for extra health and fitness (SPARC: Sport and Recreation in New Zealand, 2005).

3.4.11 How many people achieve the physical activity targets?

The WHO reports that physical inactivity is the fourth leading risk factor for global mortality. This inactivity accounts for 6% of the global death burden (WHO 2010) Analysis on the global burden of disease estimates that physical inactivity is responsible for 6% of the incidence of CHD, 7% of the incidence of type 2 diabetes, 10% of the incidence of breast cancer and 10% of the incidence of colon cancer. Furthermore the authors suggest that if physical inactivity was reduced by 10 to 25% between 533,000 and 1.3 million deaths may be averted each year (Lee et al., 2012).

Data from the Health of New Zealand Adults 2011/12 survey shows that over half of all adults (54%) are physically active for at least 30 minutes on five or more days of the week, with men (57%) more likely to meet the physical activity recommendations than women (51%) (Mason et al., 2012). This is similar to the levels found in the 2002/03 survey. Only 12% of adults reported that they had performed little or no exercise in the past week (less than 30 minutes of exercise in total over the course of the week), with women more likely than men to have performed little or no exercise in the past seven days.

3.4.12 Summary

In summary physical activity has been clearly defined, with clear and measurable health benefits. The mechanisms behind these benefits are also becoming better understood. These benefits have allowed the development of physical activity targets, set globally as well as country specific targets, with NZ data showing that approximately half the population is achieving this requirement.

3.5 Sedentary behaviour

There is growing evidence that sedentary behaviours present a distinct health risk that is independent of, and not mediated by, time spent in physical activity including MVPA (Thorp et al., 2011). A focus on increasing structured and leisure

time activity has dominated public health policy through the evolution of the physical activity guidelines. But alongside this development there has been a decline in the background level of physical activity performed by individuals, with changes in personal transportation, communication, workplace, and domestic entertainment technologies significantly reducing the demands to be active (Dunstan et al., 2012).

As opposed to the large body of work in physical activity, the measurement and understanding of sedentary behaviour is a more recent discipline. Sedentary behaviour has been defined, but not specifically measured in large amounts of research; instead physical activity behaviours that were not classified as light, moderate or vigorous were classified as sedentary. This has led to misrepresentation of the concept and how it relates to health. This is because activity levels performed at the lower end of the physical activity spectrum have been defined as sedentary, rather than a direct measurement of sedentary behaviour (Pate et al., 2008). Also no studies have withdrawn MVPA from lifestyle and studied the physiologic impact on health, besides studies of bed rest. Operationally, sedentary behaviour can be defined as activities that involve energy expenditure at the level of 1.0 to 1.5 METS. Light physical activity is often grouped with sedentary but is in essence different.

A more recent development is the use of accelerometer technology in the measurement of physical activity. Accelerometer data allows classification of cumulative time spent in sedentary behaviours. As such, this can identify individuals who meet activity guidelines but who still engage in excessive sedentary behaviour (Pate et al., 2008).

This chapter describes the measures used to estimate sedentary behaviour, and discusses the evolution of sedentary behaviour measurement from studies of bed rest to prospective cohort studies, focusing on subjective and objective measurements.

3.5.1 Definition of sedentary behaviour

Sedentary behaviours are multi-faceted, including behaviours at work, home, school, during transport and in leisure time, although typically screen time (TV viewing and computer use) have been measured, to categorise time spent in sedentary activities (Shields & Tremblay, 2008). Reading, meditating, relaxing, thinking, talking on the telephone, listening to music, writing letters playing cards and riding in a car are other examples of sedentary behaviour suggesting that most sedentary activities involve sitting, but, the tabled MET values for different types of sitting range from 1.0 to >2.0 METS (Ford & Caspersen, 2012). This suggests that although not equivalent, sitting and sedentary behaviour do overlap. To help further quantify this, sedentary behaviour has recently been defined into three basic behaviours, and in order to qualify as sedentary or exhibit a sedentary behaviour the individual must (1) be expending very little energy ($\leq$ 1.5 METS) (2) they must be sitting or lying down (3) and they must be awake (Sedentary Behaviour Research Network, 2012).

It has been proposed that it is the act of sedentary behaviour itself that causes a change in health risk, rather than the displacement of MVPA activity, and that sedentary behaviour is a distinct concept from physical activity with high levels of sedentary behaviour being able to co-exist with high levels of physical activity (Owen et al., 2010) .

3.5.2 Measures of physical activity and sedentary behaviour

3.5.2.1 Bed rest

Bed rest studies have shown that periods of inactivity are associated with metabolic changes in a short period of time, as little as three days (Katzmarzyk, 2010). Bed rest has been shown to impair insulin sensitivity, decrease fat oxidation, increase fat storage (Bergouignan et al., 2011).

An interesting follow up study (n=5) compared the changes in physical fitness from baseline following three weeks of bed rest in 1966 and changes from baseline after 30 years of aging when baseline results were measured again in 1996 (McGuire et al., 2001). There was a significant increase in body weight and fat

percentage over the 30 year period, but although aerobic fitness declined, this decline was less than what was witnessed after the three weeks of bed rest.

However, as the authors of a review of studies of bed rest noted, although bed rest can give an insight into the physiological mechanisms of sedentary behaviour, these studies are not ideal as they induced a level of physical inactivity that is different from the general population and that do not mimic all aspects of sedentary behaviour, such as sitting (Bergouignan et al., 2011)

3.5.3 Observational measures of sedentary behaviour

Sedentary behaviour measurements have evolved over the years. Initially this information was collected as interviews, activity logs or questionnaires.

A face to face or phone interview generally will involve participants recalling past behaviour, ranging from 24 hours to one week, with surrogate measures such as TV viewing, time spent sitting or transportation also collected. Recall bias and social desirability can affect the responses of the individual (Ainsworth, 2009).

A logbook may be used to record various physical activity factors such as the type, purpose, duration, intensity, body position, for activities completed within a defined period. Generally recorded every 15 minutes, METs can be determined using the compendium of physical activity with:

Kcal = MET x hours of activity x body weight (kg) (Ainsworth, 2009)

Logbooks have a very high administration burden on the participant, poor compliance and thus are subject to recall bias (Ainsworth, 2009).

Questionnaires have commonly been employed in epidemiological research, due to their relatively low cost and ease of use. They can be classified into three types;

(1) Global questionnaires are brief surveys that ask about general physical activity levels.
(2) Recall questionnaires involve 7 – 20 questions. Ordinal scales identify frequency duration and type over the past day, week or month. The

questionnaire reflects domains of activity, exercise or leisure as well as occupation, transportation and family care.

(3) Quantitative questionnaires have approximately 50 items and recall the past year or lifetime (Ainsworth, 2009).

Although most subjective measures of physical activity have shown good test retest reliability for physical activity, most PAQs do not have enough face validity, when compared with doubly labelled water (DLW) to estimate energy expenditure (Ainsworth, 2009). A review of activity energy expenditure (AEE) estimated through questionnaires, showed generally poor correlations compared to DLW. This highlighted methodology concerns with existing research with no study incorporating over 80 participants, and generally, DLW was not assessed at the same time as the PAQ (Neilson et al., 2008). When compared to objective measures, measuring sedentary behaviour by self-recall (sitting estimates) has been shown to underestimate sedentary behaviour by two to four hours (Johnson-Kozlow et al., 2006)

3.5.4 Observationally assessed mortality and adverse health outcomes

3.5.4.1 Television and screen viewing

Early research into sedentary behaviour was focused on using sedentary activities to define behaviours that did not meet the energy requirements of activity classed as MVPA (Pate et al., 2008). Due to a lack of technology available to objectively measure physical activity, efforts were focussed on assessing leisure time activities. Television viewing and recreational computer use, commonly referred to as screen time, were the sedentary behaviours that were mostly classified and investigated.

A landmark study by Dietz and Gortmaker (1985) used data from the NHANES survey (cycles II and III) to measure the association of television viewing and obesity in children using both a cross sectional and longitudinal design. They found significant association with the time spent watching television and the prevalence of obesity. Also for 12 – 17 year olds the prevalence of obesity increased by 2% for each additional hour of television watched. This association still existed when prior

obesity, region, season, population density, race socioeconomic class and other variables were controlled for.

A 21 year follow up of 7744 men in the Aerobics Centre Longitudinal Study (ACLS) study included 377 CVD deaths, and showed that self-reported TV viewing time was significantly higher in the CVD death group 10.0 ± 7.4 hours /week compared to the non CVD death group 9.1 ± 7.6 hours/week (Warren et al., 2010)

Dunstan et al. (2010a) looked at TV viewing time as a risk for all cause, CVD and cancer mortality in 8800 men and women ≥ 25 years old as part of the Australian Diabetes, Obesity and Lifestyle Study (AusDiab) When comparing less than two hours of television viewing time to greater than four hours, all-cause mortality adjusted hazard ratio (aHR) =1.46 (95% CI: 1.04 - 2.05) and CVD mortality aHR=1.80 (95% CI: 1.00-3.25) were significantly correlated.

The European Prospective Investigation into Cancer and Nutrition (EPIC) – Norfolk study included 13197 participants with a median follow up time of 9.5 years and recorded 1270 deaths (373 CVD, 570 cancer) (Wijndaele et al., 2010). TV viewing time was assessed by summing responses to four questions regarding viewing before and after six pm on week and weekend days. Those who died from any cause watched 0.4 hours/day of TV more than survivors, with CVD greater at 0.6 hours/day and cancer smaller at 0.3 hours/day. A one hour increase in TV viewing time was associated with an increased all cause aHR 1.04 (95% CI : 1.01–1.09) and CVD mortality aHR 1.07 (95% CI: 1.01–1.15)

Data from the National Health and Nutrition Examination Survey (NHANES), involving 7,350 with a median follow up of 5.8 years, was assessed for sedentary behaviours using questionnaires to assess screen time usage (Ford, 2012). There were 542 deaths recorded, which included 190 deaths from diseases of the circulatory system. Screen time was measured in hourly increments from less than one hour to greater than five hours and included watching TV, videos or using a computer outside of work. After adjusting for potential confounders, the aHR for

all-cause mortality was 1.33 (95% CI: 0.85 -2.09) and CVD 1.13 (95% CI: 0.57-2.24).

A further prospective study using data from the Scottish Health Survey involved 4512 adults aged 35 and older who were followed from 2003 to 2007, during which time 325 deaths (215 CVD deaths) occurred (Stamatakis et al., 2011). When comparing those who watched television, used a computer or played video games for < two hours per day, those who participated in ≥ four hours of these activities had an all cause aHR of 1.48 (95% CI: 1.04-2.13) and CVD aHR of 2.25 (95% CI: 1.04 – 2.13)

A recent Meta-analysis, considered two hours of TV viewing time per day and reported a pooled relative risk for all-cause mortality of 1.13 (95% CI: 1.07-1.18), fatal and non-fatal CVD of 1.15 (95% CI, 1.06-1.23) and 1.20 (95% CI: 1.14 – 1.27) for type 2 diabetes (Grøntved & Hu, 2011).

In summary, most observational assessment of screen time, involving larger cohorts have shown a consistent benefit to mortality and CVD by reducing screen time hours. The level of risk reduction is modest but significant in most studies. Ecological validity concerns over the use of questionnaires to assess screen time may influence the interpretation of these results.

3.5.4.2 Sitting time

Other studies have estimated sedentary behaviour and health risk by quantifying the amount of sitting an individual has done (Manson et al., 2002) . The Women's Health Initiative Study involved 73,743 women, aged 50 – 79 and free of CVD and cancer at baseline that were followed for an average of 5.9 years. Compared to women who reported sitting for less than four hours per day, those who spent greater than 16 hours per day sitting had a RR of developing CVD of 1.68 (95% CI: 1.07-2.64.)

Using Data from the 1981 Canada Fitness Survey, (Katzmarzyk et al., 2009) compared sitting time and all-cause mortality in 17,013 Canadians with a mean age of 42 years. Sitting time was assessed by an interviewer led questionnaire as being

either 1) almost none of the time, 2) approximately one quarter of the time, 3) approximately half of the time, 4) approximately three quarters of the time, or 5) almost all of the time, over the course of a week. Age adjusted hazard ratios increased across successive groups for all-cause mortality, (P <0.0001 for trend), and CVD (P <0.0001).

Participants in the CPS-II Nutrition Cohort categorised based on leisure time sitting, into less than three, three to five or greater than six hours per day (Patel et al., 2010). Leisure time sitting of greater than six hours was associated with an increased all-cause mortality when compared to less than three hour in men RR = 1.18 (95% CI: 1.12 – 1.25) and women RR = 1.37 (95% CI = 1.12 – 1.25). Similar associations were noted for CVD, with the relationship again appearing stronger in women than men.

Again there is a trend for greater risk of mortality and disease with greater sitting time. Research investigations have assessed both total and leisure time sitting and still came to similar conclusions suggesting that the time spent sitting, may not need to be of great duration to have an impact on the RR of an individual. However whether the increased risk was due to sitting time directly or sitting time displacing physical activity was not assessed in the above studies.

In summary, prospective studies have given a consistent indication that time spent in sedentary behaviour is associated with an increase in all-cause mortality in both men and women that is independent of BMI (Thorp et al., 2011). It should however be noted that the relationship is not always consistently significant, and there is more evidence for certain diseases (CVD, CVD) than others (eg cancer, not discussed at length in this review).

3.5.5 Evidence for adverse effects on obesity and body composition

Early research using results from the Nurses' Health Study, involving 68,497 women aged 30 - 55 showed positive relationships with TV viewing time, sitting at work, sitting away from home and driving time with an increased risk of obesity. Time spent watching TV was categorised into five quintiles (0-1, 2-5, 6-20, 21-40 and >40 hours per week). The risk for each time period (RR = 1.0, 1.23, 1.42, 1.68

and 2.0 respectively) showed a significant trend ($p < 0.001$) for developing obesity (BMI ≥30) (Hu et al., 2003).

The Australian Diabetes, Obesity and Lifestyle study (AusDiab) also highlighted a relationship between sedentary behaviours and obesity (Cameron et al., 2003). When time spent watching TV (obtained via questionnaire) was categorised into quintiles, measures of BMI in males, odds ratio (OR): 1.86 (95% CI: 1.30 – 2.67) and females OR: 1.82 (95% CI: 1.19 – 2.76) were significantly greater in the highest versus the lowest quintile. Waist circumference was also significantly higher in both sexes in the highest versus the lowest quintile (Males: OR: 1.97 (95% CI: 1.48 – 2.63), Females: OR: 2.27 (95% CI: 1.55 – 3.32)).

A recent review on the health outcomes of sedentary behaviour from longitudinal studies has shown a consistent relationship between greater sedentary time and increased risk for obesity (Thorp et al., 2011). This is especially true for sedentary time in childhood and developing adulthood obesity. There have been mixed results when specific measures of obesity have been utilised, such as waist circumference. This led the authors to conclude that there was limited evidence to suggest a longitudinal relationship exists between sedentary behaviour and weight gain or risk of obesity in adults, mainly due to limitations in the measurement of sedentary behaviour, with only three studies reviewed utilising device measured sedentary behaviour (Thorp et al., 2011).

3.5.6 Objective measures of sedentary behaviour

The use of objective measures of physical activity has addressed some of the concerns with self-reported measures. Accelerometer technology has been the main measuring tool that has been used to assess sedentary behaviour, although inclinometers, pedometers and heart rate monitors have also been used, and are discussed in specific studies briefly below.

Accelerometers allow for an objective measure of physical activity. Body movement is categorised into activity counts (counts per minute or cpm) with high counts being associated with physical activity and low counts associated with physical inactivity and sedentary behaviours. Calibration studies have allowed the

determination of cut-points for the activity counts (generally expressed as sedentary, light moderate and vigorous) which allows the data to be defined as metabolic equivalents (METS). This in turn allows for estimations of energy expenditure.

3.5.6.1 Objective measures of physical inactivity

The relationship between physical activity and body composition was assessed in 134 participants (80 women and 54 men), with an average age of 21 years. Physical activity was measured via accelerometer over two weeks, with body fat percentage determined via underwater weighing. Body fat percentage was associated with all levels of activity above "low". In women, body fat percentage and physical activity were significantly associated, but in men this was only the case when seasonality was accounted for (den Hoed & Westerterp, 2008).

Sternfeld et al. (2005) looked at body fat distribution in 248 white and Chinese, midlife women and the association with physical activity and menopause. An accelerometer worn on the waist over seven days and a log book were used to assess physical activity. A 60 second epoch was used with total activity and mean MVPA recorded (moderate activity 1000-5000 cpm and vigorous greater than 5000 cpm) Sedentary behaviour was not directly identified. Higher levels of physical activity were associated with a decreased body fat percentage and a smaller waist circumference, although not statistically significant, in Chinese women. Waist circumference decreased from 96.2cm in those doing no vigorous activity to 81.4cm in those performing 10 minutes or more per day.

Park et al. (2011) studied the relationship between body composition and physical activity using DLW and accelerometer, with 100 females aged 31 to 69 involved in the study. Movement data was categorised into eleven activity levels (0, 0.5 and 1 to 9). METS were applied to each activity level and the intensity of activity was defined as; light - >3 METS, moderate - >3, 6<METS Vigorous -6 <METS. Those women with high body fat had a lower level of physical activity as assessed by both DLW and accelerometer suggesting that there is clear relationship between physical activity and body composition. This study also highlighted the ability of accelerometer technology to identify this relationship.

A study from the Czech Republic looked at the association between volume and frequency of moderate intensity physical activity as determined via accelerometer derived steps and body composition (Pelclova et al., 2012). Accelerometer measures over seven days were recorded in 167 females, with a mean age of 62.8 years (±4.8) who had measures of BMI, body fat mass index (BFMI = body fat mass (kg) divided by the height squared (m^2)), fat free mass index (FFMI = fat free mass (kg) divided by the height squared (m^2)), WHR and visceral fat mass (VFM). Moderate physical activity and steps per day were significantly associated with observed body composition parameters. Women spending greater than 300 min/week in moderate physical activity showed significantly lower values of Body Fat Mass Index than those who spent 150-300 min/week. Carrying out moderate physical activity for 30 min five days a week was also significantly associated with lower BMI, BFMI and VFM. Higher amounts of daily steps were significantly associated with lower BMI, BFMI, VFM and WHR. An association was found between physical activity guidelines and body composition variables for the women examined in this study, however it was noted that step counts were better associated with body composition, than physical activity.

These studies highlight a concern in accelerometer and activity based research with no consistent approach used to measure activity. Both hip and waist wearing devices have been used, and different cut points assessed, and steps rather than activity counts used to quantify the exercise volume and intensity. However the above studies indicate a relationship between unfavourable changes in body composition and an absence of physical activity. The independent influence of sedentary behaviour cannot be identified, as sedentary behaviour was not directly measured. Further studies have measured sedentary behaviour and are discussed below.

3.5.6.2 Accelerometer derived measures of sedentary behaviour

A study looking at the accelerometer profiles of 3,522 US men and women as part of the NHANES survey, used epochs of 60 seconds, with greater than 10 hours of wear time used to define a valid day (Tudor-Locke et al., 2010). Counts per minute (cpm) were used to define physical activity levels with sedentary behaviours

measured at <100 cpm, Moderate at 2020 cpm (100 –2019) and Vigorous at >5999 cpm. Only 3.2% of individuals achieved physical activity guidelines of greater than 30 minutes of MVPA accumulated in 10 min bouts on at least five days per week. Gradients were used to assess trends across BMI categories (normal, overweight and obese), and showed a significant increase in sedentary time with increasing BMI in males, and significantly decreased BMI with increased moderate and vigorous intensity activity. Sedentary behaviour showed no significant differences in BMI groups in females but did so for MVPA.

Research from the AusDiab study looked at sedentary behaviours and showed that this was positively associated with waist circumference and triglycerides (Healy et al., 2008c). Each 105 minute increase in sedentary time was associated with a 3.1 cm larger waist circumference. It should be pointed out that males and females waist measures were assessed together, confounding these results. Independent of time spent in MVPA there were still significant associations with sedentary behaviour and waist circumference suggesting that regardless of the amount of MVPA performed, sedentary behaviour still negatively influences waist circumference, but the researchers did not note any sex differences within their data. Sedentary time and time in light activities were correlated but not sedentary time and time spent in MVPA.

The same researchers also assessed physical activity and breaks in sedentary time with a uniaxial accelerometer over seven days, with 168 males and females aged 30-87 involved in the study (Healy et al., 2008a). Sedentary behaviour was defined using a cut point of 100 cpm, with a break in sedentary time defined when the accelerometer count rose above 100 counts for longer than 10 consecutive minutes. There was a significant correlation with fewer breaks in sedentary time and significantly increased waist circumference, BMI, triglycerides and two hour fasting glucose levels. No significant associations were seen with HDL, BP or fasting glucose. Those in the highest quartile of breaks in sedentary behaviour had a lower waist circumference, and lower two-hour fasting glucose levels. Males and females were assessed together, which may have been a significant confounder, especially with waist circumference measures.

Contrasting this result, a study with 442 Flemish adults, using objectively measured sedentary behaviour and physical activity, showed that adults who spent ≥ 60 mins/ day in MVPA had significantly lower body fat percentages (as measured via BIA) than those with less than 60 minutes per day in MVPA, regardless of the time spent in sedentary behaviours (Scheers et al., 2013) . The authors note that this suggests that engaging in high levels of sedentary behaviour can be compensated for by engaging in sufficient MVPA.

A similar result was seen by Ekelund et al. (2009) in 192 participants with a family history of type 2 diabetes, with time spent in MVPA a significant predictor of fasting insulin at a one year follow up (β = -0.004 [95% CI: -0.007, -0.0001], P = 0.022), with insulin sensitivity, measured by HOMA-IR, approaching significance (β = -0.003 [95% CI: -0.007, -0.0002], P = 0.052). This was independent of time spent sedentary, light intensity exercise and other lifestyle behaviours such as smoking. No association was seen for sedentary behaviour or light intensity exercise and meeting the physical activity guidelines (> 30 minutes of MVPA per day), and being in a lower risk BMI group (normal, overweight or obese) predicted a lower fasting insulin status on follow up (P_{trend} = 0.050 and 0.004 respectively).

In a similar population with known risk factors for type 2 diabetes, Henson et al. (2013) found contrasting results, with sedentary behaviour predicting 2 hour fasting glucose (β=0.220±0.060, P <0.001), triacylglycerol (β=−0.123±0.056, P =0.029) and HDL- Cholesterol (β = 0.206±0.061, P =0.001) after adjusting for BMI and MVPA. MVPA was associated with lower BMI (β=−0.215±0.041, p <0.001) and waist circumference (β =−0.228 ± 0.043, P <0.001) after adjusting for sedentary time.

Recent evidence from Green et al. (2014) has shown that sedentary time was significantly associated with triglycerides and lipid accumulation product (an independent risk factor for CVD and diabetes calculated from waist circumference and triglyceride levels) independent of MVPA, body mass and $\dot{V}O_2$ peak. They assessed 50 young adult women, and also found that $\dot{V}O_2$ peak was an important

confounding variable when assessing the association with sedentary behaviour, physical activity and cardio metabolic health.

A prospective trial, using heart rate monitoring as an objective measure of sedentary behaviour, showed that independent of sex, age, baseline physical activity energy expenditure, baseline sedentary behaviour smoking and social economic status (SES), baseline measures of body weight, BMI, fat mass and waist circumference all predicted an increase in the amount of sedentary behaviour over a 5.6 year follow up period (Ekelund et al., 2008). However sedentary behaviour itself did not predict gains in body weight.

These data show conflicting evidence from a wide range of studies, with sedentary behaviour generally predicting cardio metabolic health but with less evidence for anthropometric measures such as BMI and waist circumference, where MVPA appears to be a better indicator. This may be due to the maintenance of lean tissue through high intensity exercise, helping to prevent the accumulation of adipose tissue (Boutcher, 2011). Also in the studies mentioned above, there has been a wide range of anthropometric profiles used, rather than a consistently homogeneous range, potentially confounding results, as they relate to body weight and fat gain or maintenance.

3.5.7 Mechanisms of increased health risk

3.5.7.1 Physiological

Whilst the mechanisms of how sedentary behaviours increase health risk are not well understood at present, current evidence suggests that these behaviours may have a direct influence on metabolism, bone mineral content and vascular health (Tremblay et al., 2010).

3.5.7.2 Metabolic health

Lipoprotein lipase (LPL) is an enzyme that facilitates the uptake of free fatty acids into skeletal muscle and adipose tissue. Lower levels are associated with an increase in circulating triglyceride levels, decreased HDL cholesterol and an increased risk of CVD (Hamilton et al., 2007).

Using rat models, Bey and Hamilton (2003) have shown that physical inactivity significantly reduces lipoprotein lipase activity, with no significant difference in the drop in LPL activity between a single day of inactivity and 11 days inactivity. This has important implications, as it implicates an acute, rather than an accumulative effect, similar to a day spent at work, having an effect on an individual's metabolic risk. Also of note is that LPL activity returned to normal levels after four hours of light intensity walking, suggesting a protective and restorative factor for exercise, although it is unclear at present what intensity and duration are required to provide this exercise benefit. The authors suggested the changes in LPL activity in sedentary behaviour appear to be due to transcriptional changes, rather than to changes in LPL mRNA levels, which are seen with exercise (Bey & Hamilton, 2003). This suggests that there are different mechanisms acting upon the LPL pathway in sedentary behaviour compared to physical activity.

Studies of human bed rest have shown similar metabolic changes, with 20 days of bed rest significantly decreasing LPL activity, alongside fasting HDL while VLDL and triglycerides increased (Yanagibori et al., 1998). In another study, five days of bed rest significantly increased total cholesterol, triglycerides, glucose, insulin and insulin resistance, as measured by Homeostasis Model Assessment-insulin Resistance (HOMA-IR) (Hamburg et al., 2007).

Sedentary behaviour is also reported to have effects on carbohydrate metabolism through changes in muscle glucose transporter (GLUT). Evidence shows decreases in muscle GLUT – 4 content and insulin stimulated glucose uptake in response to muscle denervation. Patients with spinal cord injuries have also shown dramatic increase in muscle GLUT content (both GLUT 1 and GLUT 4 transporters) with very low intensity exercise, as well as improved oral glucose tolerance. This has been witnessed at intensities far lower than what would be considered moderate physical activity (Tremblay et al., 2010).

A prospective study, involving 376 middle aged adults followed over 5.6 years and using heart rate monitoring as an objective measure of physical activity and sedentary behaviour, showed that time spent in sedentary behaviour predicted

higher levels of fasting insulin, independent of the time spent in MVPA (Helmerhorst et al., 2009). This result was independent of time spent in MVPA, further indicating benefits to metabolic health in limiting sedentary behaviour time.

Conversely, recent research has looked at the relationship sedentary behaviour and metabolic syndrome (MetS) as defined by National Cholesterol Education Program Adult Treatment Panel III guidelines. Three hundred and seventy Flemish adults wore a SenseWear armband for seven days allowing for estimation of METs, and sedentary behaviour was defined as activities with a MET value ≤ 1.5 during waking hours. MVPA was also recorded as activities >3 METs. Total sedentary time and the average duration of sedentary bouts were positively associated with MetS (OR = 1.07–1.47) and some of the individual components of this (Waist circumference, Triglycerides, HDL cholesterol, Diastolic Blood Pressure). However when MVPA was accounted for this association was no longer significant, indicating that engaging in enough MVPA may counteract the effects of sedentary behaviours in the development of MetS and its components (Scheers et al., 2013)

3.5.7.3 Bone density

A loss of bone mineral density is another documented detrimental effect of sedentary behaviour, with both animal and human studies showing reductions in bone mass following long periods spent in zero gravity, with bed rest and in cases with spinal cord injuries (Tremblay et al., 2010).

A 14 day bed rest study, designed to simulate a microgravity environment, measured bone formation and resorption markers in 11 healthy young male participants (age 22 ± 1 year) (Kim et al., 2003). Urinary deoxypyridinoline concentration and urinary type I collagen cross-linked N-telopeptides both increased significantly from baseline by the end of the study. Both these measures are highly specific markers of bone resorption indicating a risk of decreasing bone mineral density. Also both serum and urinary calcium increased significantly from baseline, whereas markers of bone formation were less affected, with early marker bone alkaline phosphatase increased, and late marker osteocalcin decreased.

3.5.7.4 Vascular health

Vascular health may also be affected by sedentary behaviours. Hamburg et al. (2007) measured significant increases in systolic blood pressure, and decreases in brachial artery diameter after five days of bed rest. Reactive hyperaemia, an important measure of microvascular function was also decreased after five days in both the forearm and calf.

Other studies simulating a microgravity environment have found similar results including decreased endothelium dependant vasodilation and increased endothelial cell damage (Tremblay et al., 2010).

Prospective studies have shown sedentary behaviour to be a risk factor for hypertension. The SUN cohort used 11,837 Spanish university graduates with a mean age of 36 years and followed for an average of 40 months (Beunza et al., 2007). Using questionnaires to assess sedentary behaviour and incident hypertension the authors reported a higher risk of hypertension when comparing upper and lower quartiles for sedentary time (Hazard ratio 1.48 95% CI 1.01 – 2.18).

3.5.8 What are the guidelines for sedentary behaviour?

The current WHO Global recommendations on Physical Activity for Health (2010) do not have any specific recommendations regarding sedentary behaviours. There is reference to reducing physical inactivity, identifying this as the fourth leading cause of global mortality. Sedentary behaviour is identified as an area of new research and is proposed as a topic for review in 2015.

3.5.8.1 Worldwide

Only a limited number of countries have released specific guidelines for sedentary behaviour. The Australian Physical Activity & Sedentary Behaviour Guidelines for Adults (18-64 years) have general guidelines regarding sedentary behaviour, with advice to minimise the amount of time spent in prolonged sitting and to break up periods of sitting as often as possible. In addition, there are specific recommendations on how to be more active to reduce sedentariness, with tips on building more activity into the day, how to be active at work and active indoors

with specific recommendations on how to reduce sedentary behaviour also included (Australian Government: Department of Health, 2014).

Canada have released sedentary behaviour guidelines for children aged 0-4 years and 5 to 11 years, and for youth aged 12 – 17 but there are no specific guidelines for adults or older adults other than increasing physical activity (Canadian Society for Exercise Physiology, 2012).

3.5.8.2 New Zealand

There are no specific recommendations for sedentary behaviour in New Zealand. The physical activity guidelines make no mention of this, although the Ministry of Health website advises to increase the time you spend being physically active and to reduce the time you spend being sedentary, but with no suggestions on how to achieve the latter.

3.5.9 What is the prevalence of sedentary behaviour?

3.5.9.1 Worldwide

Evidence of the prevalence of sedentary behaviours comes from various self-reported, interviewer assessed and objectively measured studies. This review focuses on only those objectively measured studies.

Ekelund et al. (2006) used accelerometer data as a tool to validate a self-report tool (International Physical Activity Questionnaire – I-PAQ) for both exercise and sedentary time. Seven days of accelerometer wear time was evaluated in 185 Swedish workers, with sedentary behaviour defined as a cut point of >100 cpm. Average time in sedentary activity was 7 hours and 7 minutes for males (± 74 minutes) and 6 hours 54 minutes for females (± 69 minutes). This was estimated to be approximately 54% of waking time for both sexes.

Using results from the NHANES 2003/2004 cohort, Matthews et al. assessed accelerometer data for 6329 participants of both sexes aged 6-85 (Matthews et al., 2008). Using at least one day of valid wear time, and estimating sedentary behaviour with a cut point of >100 cpm, males spent 7.73 hours/day and females 7.74 hours per day in sedentary activities. Adult sedentary behaviour increased by

age bracket from 7.48 hours /day in the 20 – 29 age group to 9.28 hours/ day in the 70 – 85 age group with. Younger and adult females were more sedentary than younger and adult males, but this trend was reversed in those aged over 60 years.

The AusDiab study used seven days of accelerometer data from 169 adults (67 men and 102 women), with sedentary time defined as <100 cpm. Results showed that 57% of wear time was spent in sedentary behaviour, with 39% in light intensity activities and 4% in MVPA (Healy et al., 2008c).

3.5.9.2 New Zealand

There is a lack of specific evidence on sedentary behaviours in New Zealand. A birth cohort study from Dunedin measured self-reported TV viewing time in 992 participants aged 21 (Hancox et al., 2004). Mean TV viewing time was 3.07 hours, with greater television viewing hours at an earlier age associated with being overweight at 21. Further evidence is provided in Statistics NZ 2009/10 time use survey, which shows 2 hours and 8 minutes spent was spent watching television which is an increase of six minutes since 1998/99 (Bascand, 2011).

3.5.10 Summary of section

The evidence shows a clear relationship between increasing levels of sedentariness and mortality and CVD. The relationship between sedentary behaviours and obesity and specific body composition changes is less clear with some studies showing a relationship, and others showing no relationship. Furthermore this is confused further by some studies indicating the relationship between sedentary behaviour and increasing weight is independent of the amount of MVPA performed, whilst others show the opposite, that MVPA reduces weight independent of sedentary behaviours.

Sex differences exist in regards to the risk of sedentary behaviour, but little is understood about the mechanisms of this risk. The effects of lipoprotein lipase have been assessed, mainly in animal models, with bed rest and anti-gravity studies showing further correlates with poor health outcomes, especially in regards to the metabolic syndrome.

More countries are developing sedentary behaviour guidelines, although there is no global strategy for this at present, even though sedentary levels have been noted to make up the majority of waking time in most epidemiological studies. There is currently a lack of evidence on the prevalence of sedentary behaviour in New Zealand.

3.6 Summary

There has been a recent shift in physical activity research from focusing on differing identifying optimal volume and intensities of exercise and activity to focuses on reducing sedentary behaviour. The evidence regarding the prevalence of sedentary behaviour in New Zealand is very limited, making public health recommendations about reducing sedentary behaviour challenging. Although there is a clear link between sedentary behaviour mortality and increased chronic disease risk, there is little objective evidence about the prevalence of this behaviour in New Zealand adults.

The link between physical activity and decreasing CVD and Diabetes risk is well recognised, and further specific outcomes such as improved BMI, blood pressure and decreased MetS risk classification have been established. The link between sedentary behaviour and these outcomes is less clear and has not been presented in a New Zealand population, although there is a clear benefit to reducing sedentary behaviour by decreasing all cause and CVD mortality.

Further how sedentary behaviours and physical activity correlate with body composition and health in those without obesity but with higher levels of body fat has not been assessed. This study aims to objectively assess sedentary behaviour and physical activity levels in New Zealand European females aged 16 – 45 with a normal BMI, and compare this to body fatness, central adiposity and blood pressure as surrogate measures of health risk. Further it aims to assess the comparable effects of sedentary behaviour and MVPA on these health markers to help inform public health policy on promoting reductions in sedentary behaviour, increasing physical activity or both.

4.0 Methods

4.1 Research design

The study, Examining the Predictors Linking Obesity-Related Elements (EXPLORE) is a comparative cross sectional analysis of New Zealand European, Maori and Pacifica women, that aims to investigate whether body fat profiles (hidden or apparent) are associated with an increased metabolic disease risk. It also aims to investigate dietary and physical activity patterns as predictors of body fat profiles.

The current study was a cross sectional investigation into the clinical benefits of MVPA in otherwise sedentary women. It included accelerometer-derived physical activity data from the New Zealand European women sub group of the EXPLORE participants. The physical activity data, classified into sedentary, light, moderate and vigorous activity using standard cut-off points (Troiano et al., 2008), will define the independent variable in this research. Height, weight, Body Mass Index (BMI), waist and hip circumferences, waist to hip ratio, body fat percentage and blood pressure were considered dependent variables.

4.2 Participants

Women, aged 16 – 45 years were recruited from the wider Auckland area for the EXPLORE study. Participants were recruited from local members of the North Shore (Auckland) community, universities, local businesses, newspaper advertisements, magazine articles, radio and word of mouth. Of the 775 women recruited for the EXPLORE study, 190 were included in this cross-sectional analysis. Participants were included in the study at the first stage if they were aged between 16- 45, were post-menarche (defined by at least one complete year of regular menstrual cycle), and were pre-menopausal (defined by a continuous regular menstrual cycle for at least the past one year). Participants were excluded if they were pregnant, currently breastfeeding, or showed the presence of any illness (Coronary Heart disease, diabetes, cancer, gut disorders interfering with the digestion and absorption of food, Endocrine disease, thyroid disease, kidney disease, liver disorders and blood borne diseases such as Hepatitis B).

Participants were of New Zealand European descent. This was established as those that identified with NZ European ethnicity on the initial health questionnaire and were born in New Zealand or had been living in New Zealand for five or more years. If participants identified with another ethnicity, as well as New Zealand European, then participants where included if one parent identified their ethnicity as New Zealand European.

4.3 Sequence of tests

The EXPLORE study was conducted in three stages. Stage one involved the screening of participants for inclusion in the analysis, with a health questionnaire, height and weight measurements to calculate Body Mass Index (BMI) and bioelectrical impedance testing for estimating body fat percentage to categorise participants into one of three body composition profiles:

- Group one; normal BMI and normal body fat percentage.
- Group two; normal BMI and high body fat percentage.
- Group three; high BMI and high body fat percentage.

Those meeting the criteria for acceptance into the study were invited for the testing of health markers (total and regional body composition and blood pressure). For the purpose of this cross-sectional study, only women classified in groups one and two were included.

The final process involved the participants wearing preprogramed accelerometer for seven days (Figure 1).

4.4 Anthropometric measurements

Height was initially measured at screening, to allow the calculation of body fat percentage and BMI, via a calibrated stadiometer. The procedure for measuring height involved checking hair was not interfering with measurement, feet were positioned together and looking straight ahead, the jaw was lifted with both hands so the participant's eye socket (orbitale) was horizontal with their ear canal (tragion).

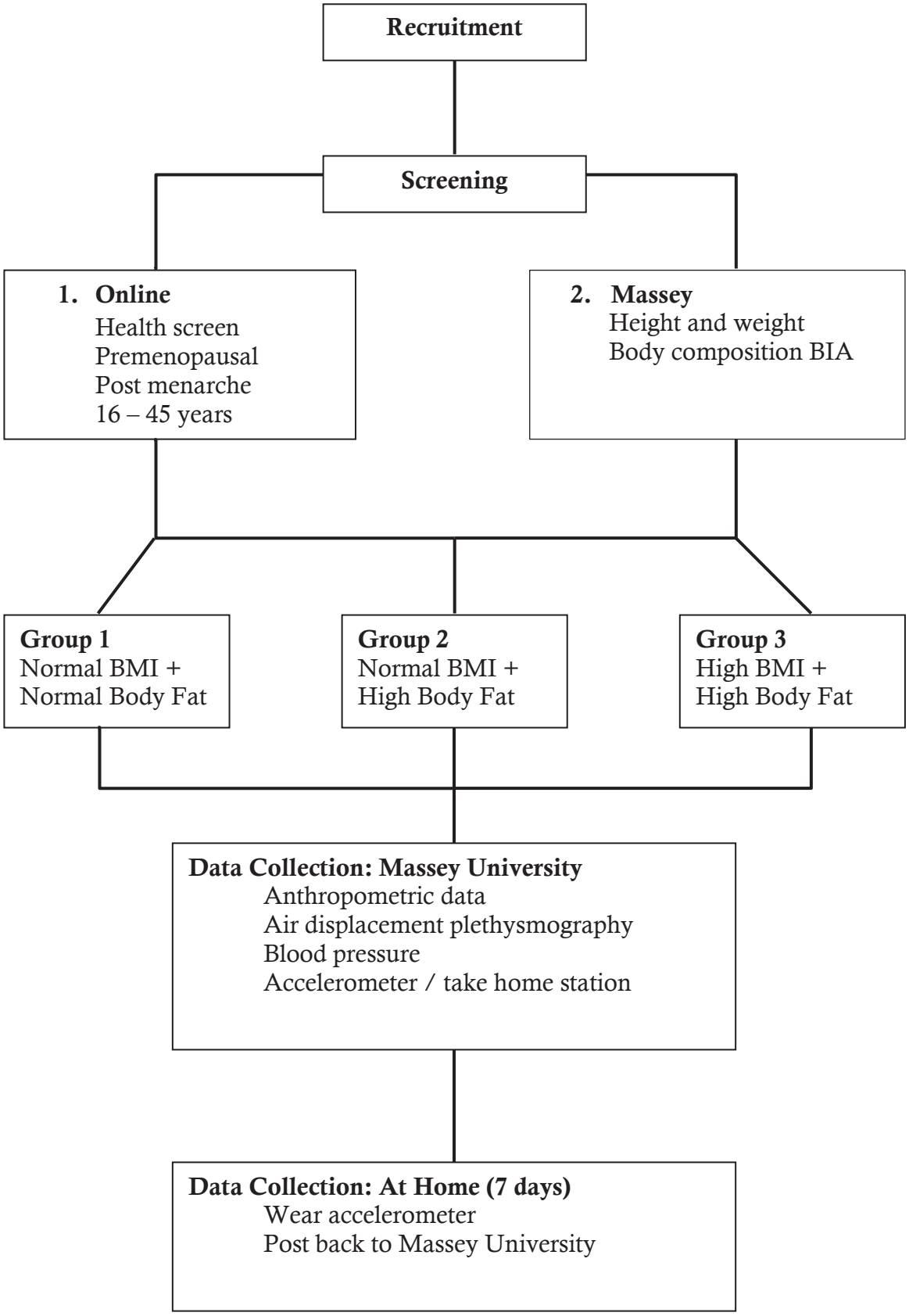

Figure 1: Flow chart of testing process

The participant was then asked to inhale and measurement was taken at full height with the headboard touching the vertex of the skull. Height was recorded to the nearest 0.1 cm as the average of two measurements. A third measure was taken if the 2nd measure was not within 1% of the first measure.

A bioelectrical impedance analyser (Inbody 230, Biospace, Cerritos, CA) was used to estimate body fat percentage. Participants were instructed to remove light clothing and shoes so that they could be measured in bare feet and a weight measurement was taken whilst standing stationary on the device. After self-calibration of the BIA analyser, participant's identification number, age, height measures and sex (all female participants) were entered into the device, and participants were instructed to remain still whilst holding the handgrips, slightly abducted from the body, for 30 seconds while the measurement was completed. The InBody 230 BIA has shown to have a strong, significant correlation for percentage body fat when compared with Dual energy X-ray absorptiometry (DXA) (r=0.97 p=0.01) (Karelis et al., 2013)

The measurement of weight from the device allowed the calculation of BMI (kg/m^2). Normal BMI was defined as a body mass index of 18.5 - 24.9 with high BMI defined as above 25.0.

On acceptance into the study, participants attended testing at the Nutrition Research Facility at Massey University's Albany campus, which was timed to coincide with the first two weeks of the individual's menstrual cycle. All measurements were taken in the morning before 10am and participants were instructed to not exercise or eat or drink anything in the two hours prior to the measurement.

A pre testing questionnaire was completed that identified menstrual status, employment history, smoking status and the amount of alcohol consumed weekly. Participants were asked if they had partaken in any smoking, caffeine, vigorous physical activity or recreational drugs in the last hour. If the participant answered

yes to any of these questions the testing procedure was delayed and the participant rebooked for analysis on a later day

The Bod Pod system has been shown to have high intra class correlation (ICC), in a heterogeneous population, when used to measure body density (ICC = 0.996, P = 0.001) (Noreen & Lemon, 2006). Height was re-measured as per the above protocol, and entered into the Bod Pod system to the nearest 0.1cm.

Participants were instructed to wear a tight-fitting swimsuit and cap and empty their bladder prior to testing. All jewellery was asked to be removed. Waist and hip circumference was measured at this time. A flexible steel tape measure (Lufkin W600PM, Apex Tool Group, NC, USA) was used with the cross hand technique. Waist measures were determined at the narrowest point between the lower costal border and the iliac crest, with the measure taken at the end of a normal expiration and measured twice, or three times if more than 1cm difference was noted. Hip measures were taken at the widest part of the buttocks and measured twice or three times if there was more than a 1cm difference. This data was entered into the Bod Pod device to the nearest cm.

Volume calibration was performed with a 50-L cylinder. A weight calibration was also performed with two 10kg weights place on the Bod Pod scale. Participants were weighed in the Bod Pod and had this weight recorded into the Bod Pod device. Participant's lung volume was measured by the evacuation of thoracic lung volume. The body volume procedure required the participant to sit quietly for 50s, and two measures were taken.

Body density was measured by the BodBod computer and body fat percentage was estimated using the Siri equation:

Body Fat = (495 / Body Density) – 450 (Siri, 1961).

4.5 Blood pressure

An automated blood pressure monitor (Riester Ri Champion N digital monitor; Rudolf Riester GmbH, Jungingen, Germany) was used for determining blood pressure. The participant was seated in a chair and any loose clothing was

removed. The cuff was appropriately sized by measuring arm circumference, and fastened to the participant. The participant was left to rest quietly for five minutes in this position with feet uncrossed. Three readings, with one minute intervals between each reading were performed, and the average was recorded to the nearest 1 mmHg (O'Brien et al., 2003).

4.6 Physical activity measures

The accelerometer (WGT3X, Actigraph, Pensacola, FL, USA) and appropriate software (Actilife 6, Actigraph, Pensacola, FL, USA) were used to assess physical activity over a period of seven days during the study. Participants were shown how to wear the device by trained IFNHH staff. Participants were instructed to wear the accelerometer at all times (including to bed, but to be removed for showering, swimming or other water based activities) during a typical week. A typical week was defined as one during which they attended work/school/university for five days and participated in usual after work and weekend activities. Weeks during which participants were taking holidays or other days off (public holidays, school holidays, or special events) occur were to be avoided.

The device was fitted on the participant on the right hip. An activity diary was also given to participants to record any activities performed with the accelerometer removed and to record any structured exercise that was completed. The accelerometer was programmed to start recording at midday on the day of testing and to stop at midday on the eighth day following testing. After this the device was to be returned to the IFNHH staff in a pre-paid envelope. Once received, data stored in the accelerometer was checked for completeness and downloaded to a computer.

4.7 Data handling

After removing participants with a BMI $\geq$ 25 kg/m^2 (63 participants) a total of 127 participants were included in the analysis (see figure 2). Of this group nine had no accelerometer data, either due to accelerometer malfunction, lost accelerometers or unworn accelerometer. A further seven participants had incomplete accelerometer

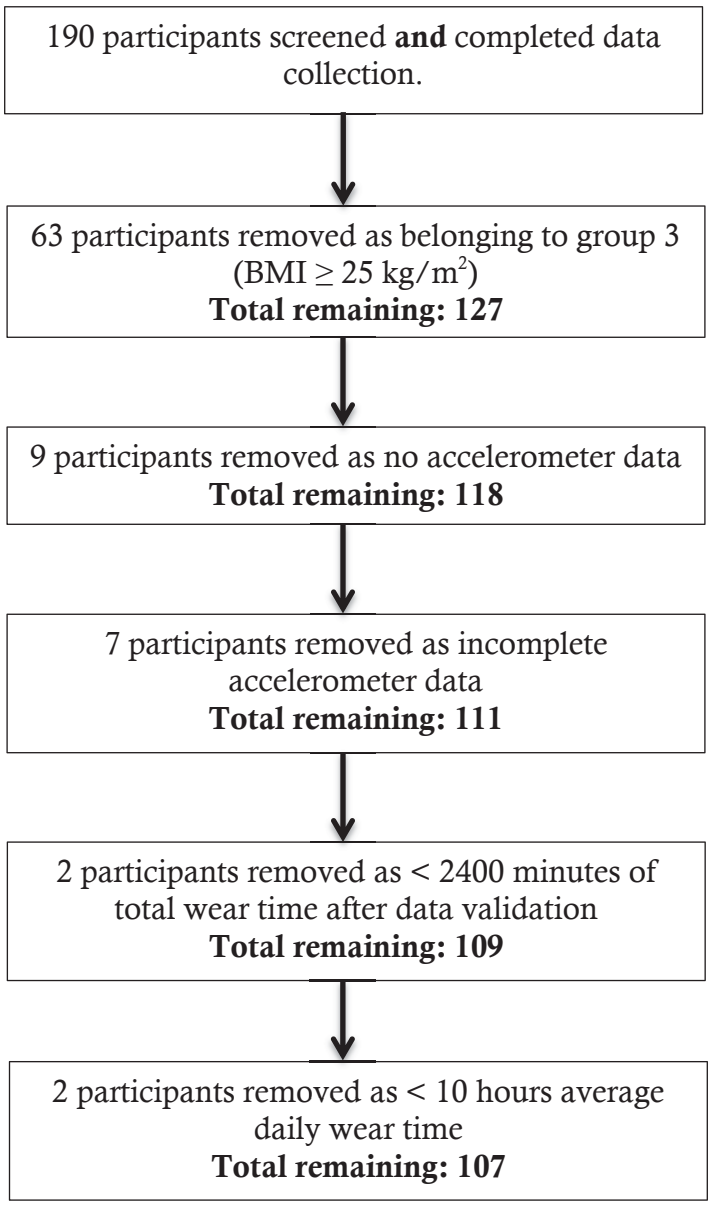

Figure 2: Flow chart of data handling procedure.

data, defined as less than seven complete days of accelerometer recording. This was due to accelerometer malfunction.

As participants were instructed to wear their accelerometer at all times, including to bed, sleep time was assessed and removed from the analysis using the algorithm designed by Tudor-Locke et al. (2014). This analysis was defined to differentiate zero counts from sleeping and zero counts from being sedentary. The remaining 111 accelerometer profiles were processed using this algorithm via computer software (MATLAB and Statistics Toolbox Release 2012b, The MathWorks, Inc., Natick, Massachusetts, United States).

Four weekdays, including one weekend day has been accepted as a valid week, (Healy et al., 2011) and participants with less than 2,400 minutes of total wear time were excluded. It was not able to be determined via this method, what days of the week were included in the each participant's analysis (total of two participants).
A valid day was defined as at greater than 10 hours of wear time, which has been commonly cited as acceptance of a valid day (Atkin et al., 2012). Participants who achieved less than 10 hours average wear time over the seven days were excluded (total of two participants). This left a sample size of 107 participants.

4.8 Accelerometer cut point selection

Assessing the Crouter 2006 and 2010 regression models, NHANES cut points and Matthews cut-points, Crouter et al. showed none were better for estimating free living physical activity, when compared against indirect calorimetry (Crouter et al., 2013). As one of the aims of this study was sedentary behaviour analysis, it was noted that a cut-point of 100 Counts Per Minute (CPM) is commonly used to define sedentary behaviour, although there are limited validation studies of this cut point (Atkin et al., 2012). As mentioned previously, sedentary behaviour has been defined as an energy expenditure at the level of $\leq$1.5 METS with MVPA greater than three METS (Pate et al., 2008). A cut point of 150 CPM has recently been suggested for sedentary behaviour, as this was shown to have the lowest bias in a validation study of the GT3X, over estimating sedentary time by 1.8%, whereas a cut point of 100 CPM underestimated sedentary time by 4.9% (Kozey-Keadle et al., 2011). The limitation in this validation is that the assessment was over two, six hour periods, rather than the 10 hours, over seven days of wear that was used in this study for acceptance of a valid day. Also, as this cut point has been seldom used in the literature, a comparison of results against other studies would be more difficult. Given the lack of agreement, the Troiano et al. (2008) NHANES cut points were utilized, due to the recent validation study by Crouter above and the wider use of the cut point in current research (Troiano et al., 2008). These are defined as:

- Sedentary: 0 - 99 CPM
- Light: 100 - 2019 CPM
- Moderate: 2020 - 5998 CPM

- Vigorous: 5999 - ∞ CPM

4.9 Body fat cut point selection

We used a cut point of <30% to indicate a healthy body fat level. This is a somewhat subjective measure of health, as there is no consensus as to what defines obesity via fat mass calculations or fat percentage (Oliveros et al., 2014). This is mainly due to direct measures such as water-displacement plethysmography or magnetic resonance considered too cumbersome and expensive to be utilised in large populations. However Dvorak et al. (1999) used the same value to define NWO and for ease of use this value was chosen.

4.10 Statistical analysis

Statistical analysis was completed using IBM SPSS statistics package version 20 (IBM corporation, New York, USA). Variables were tested for normality using the Kolmogorov –Smirnov and Sharipo Wilk tests and for homogeneity using the Levene's test. Normally distributed data was reported as mean ± SD and geometric mean [95% CI], and not normally distributed data expressed as median [25^{th}, 75^{th} percentiles]. A *P* value of less than 0.05 was considered to be significant. For variables that showed statistically significant differences between groups, the effect size was calculated, with 0.2 considered a 'small' effect size, 0.5 represented a 'medium' effect size and 0.8 a 'large' effect size (Cohen, 1992).

Analysis of differences in dependent variables between two groups, were examined using the independent *t*-test for parametric data and the Mann-Whitney test for non-parametric data.

Quartiles were created by ranking the amount of MVPA, with any ties separated by the mean method with the same value assigned the average rank. For comparisons of more than two groups, one way ANOVA was performed, with post hoc analysis by Tukey method used on significant results, to determine where the difference lay and the level of significance.

Pearson's correlations were performed using physical activity measures and health measures. Both bivariate and partial correlations were performed.

Participants who were excluded from the study were compared with participants who completed the study using these tests. Two side tests were used for all analyses.

4.11 Funding

The study was funded by a Massey University Institute of Food, Nutrition and Human Health Research Grant, Nutricia Research Foundation, Netherlands and New Zealand Lottery Health.

5.0 Results

5.1 Participant characteristics

The characteristics of those included (N=107) and excluded from the study (N=53) were assessed for differences. Reasons for exclusion have previously been discussed. No significant differences were noted between the two groups in age or accelerometer measures.

Participant characteristics are present in Table 4. Included in the sample were 67 (62.3%) participants who were classified with high body fat (≥30%), with 40 (37.4%) participants classified as with normal body fat (<30%). As previously discussed all included participants had a normal BMI.

Table 4: Participant Characteristics (n= 107).

Variable	Mean ± SD	Range
Demographics		
Age (yrs)*	30.6 (25.6 – 39.2)	16.6 – 44.9
Weight (kg)	61.8 ± 6.7	43.7 – 77.0
Height (cm)	167.7 ± 6.5	149.4 – 182.5
BMI (kg/m^2)	22.0 ± 1.6	18.7 – 24.9
Body Fat (%)	27.9 ± 5.6	17.3 – 43.9
Waist (cm)	71.4 ± 4.8	59.0 – 85.3
Hip (cm)	98.4 ± 5.0	86.0 – 108.3
Waist/Hip ratio	0.73 ± 0.04	0.63 – 0.84
Systolic BP (mmHg)	114.0 ± 9.0	89.0 – 149.0
Diastolic BP (mmHg)	71.0 ± 7.0	52.0 – 97.0
Accelerometer measures		
Sedentary (%)	61.33 ± 8.04	40.39 – 79.52
Light (%)	34.42 ± 7.47	18.62 – 51.51
MVPA (%)	4.25 ± 2.14	0.57 – 10.99

* Median (25th – 75th Percentile)

(BP: Blood Pressure; MVPA: Moderate to Vigorous Physical Activity)

No participants had a high risk waist circumference, (≥ 88 cm), 4.7% presented with an elevated systolic blood pressure (≥ 130 mmHg) and 1.9% with an elevated diastolic blood pressure (≥ 85 mmHg).

Participants spent on average 9.96 (61%) hours of their waking day exhibiting sedentary behaviours, with 5.56 (34%) hours in light activities and 41 (4%) minutes per day in MVPA. This is similar to previous research from Australia of a comparable sample size (Healy et al., 2008c), and recent evidence from the USA (Green et al., 2014).

5.2 Body composition

Participants were then stratified by body fat percentage, with a value of <30% considered normal and ≥ 30% considered high (Table 5). A significant difference between groups was noted in the amount of MVPA completed, with those having a normal body fat percentage (318.9 ± 158.0 minutes/week) completing more MVPA than those with high body fat percentage (236.3 ± 114.6). (t=2.881, 105 df, P =.002, r = 0.27)

Table 5: Independent samples T - Test for differences in blood pressure and activity levels between normal and high body fat groups.

	Body Composition Group		*P*
	BF <30% (n = 67)	BF ≥30% (n = 40)	
Systolic BP (mmHg)	113.0 (106.0 – 118.0)	114.5 (109.3 – 120.5)	.379
Diastolic BP (mmHg)	70.0 (65.0 – 75.0)	70.0 (67.0 – 77.0)	.171
Sedentary (mins/week)	4232 ± 762.4	4105 ± 686.0	.390
Sedentary (%)	60.78 ± 8.45	62.26 ± 7.32	.358
Light (mins/week)	2394 ± 536.4	2247 ± 555.5	.179
Light (%)	34.61 ± 7.74	34.11 ± 7.07	.739
MVPA (mins/week)	**318.9 ± 158.0**	**236.3 ± 114.6**	**.002**
MVPA (%)	**4.61 ± 2.28**	**3.63 ± 1.75**	**.021**

(BF: Body fat; BP: Blood Pressure, MVPA: Moderate to Vigorous Physical Activity).

5.3 Activity guidelines

Participants were separated into groups based on achievement of physical activity guidelines and all health measures and activity levels were assessed between the two groups (Table 6). Participants displaying a level of ≥ 150 minutes of MVPA per

week were considered to have achieved the recommendations and < 150 minutes of MVPA was considered to have not achieved the target. Percentage body fat was different between the two groups, with those not achieving the physical activity guidelines showing significantly higher body fat percentage (30.3% ± 4.08%) than those achieving the guidelines 27.4 ± 5.7 (t = -2.107, 105 df, *P* =.038 (two tailed), small effect *r* = 0.20). No other significant differences were noted between the groups.

Table 6: Independent samples T - Test for differences in health variables with those meeting physical activity recommendations (≥150 mins MVPA/week) and those not meeting the recommendations (<150 mins MVPA/week).

	Do not achieve MVPA	Achieve MVPA	*P*
Number of participants (%)	19 (17.7)	88 (82.3)	
Body Fat%	**30.3 ± 4.08**	**27.4 ± 5.7**	**.038**
Systolic BP (mmHg)	113.9 ± 8.3	113.9 ± 9.0	.991
Diastolic BP (mmHg)	70.8 ± 7.3	70.7 ± 7.0	.747
Waist (cm)	71.3 ± 4.3	71.5 ± 5.0	.894
WHR	.72 ± .04	.73 ± .04	.602

(MVPA: Moderate to Vigorous Physical Activity; BP: Blood Pressure; WHR: Waist to Hip ratio).

To assess the importance of moderate and vigorous components of activity on health risk, the cohort was separated firstly into two groups based on the achievement of 300 minutes of moderate physical activity per week (Table 7). This showed no significant difference in the waist (*P*=0.894) or blood pressure measures between the two groups (SBP *P*= 0.991, DBP *P*= 0.747). Although body fat was not significantly different between the groups, it was trending towards significance (t = 1.793, 105 df, *P* = .076).

Table 7: Independent samples T - Test for differences in health risk between those achieving 300 minutes of moderate level physical activity (3-6 Mets) and those not achieving 300 minutes.

	Do not achieve 300 mins MPA	Achieve 300 mins MPA	*P*
Number of participants (%)	83 (77.6)	24 (22.4)	
Body Fat (%)	28.4 ± 5.6	26.1 ± 4.9	.076
Systolic BP (mmHg)	114.3 ± 9.1	112.5 ± 8.2	.377
Diastolic BP (mmHg)	70.9 ± 6.9	70.3 ± 7.5	.731
Waist (cm)	71.6 ± 4.6	71.0 ± 5.7	.631
WHR	.73 ± .04	.72 ± .04	.601

(MPA = moderate physical activity, WHR = waist to hip ratio, BP = blood pressure).

When the cohort was separated into groups according to the achievement of 150 minutes per week of vigorous exercise, a significant difference in body fat between the two groups was observed (Table 8). Those who achieved 150 minutes had significantly lower body fat (24.5 ± 4.3%) than those who did not achieve 150 minutes (28.3 ± 5.5%) (t = 2.317 105 df, *P* = .022 r = 0.22). No other health measures showed differences between the two groups.

Table 8: Differences in health risk between those achieving 150 minutes of Vigorous Level Physical Activity (> 6 Mets) and those not.

	Do not achieve 150 mins VPA.	Achieve 150 mins VPA.	*P*
Number of participants (%)	95 (88.8)	12 (11.2)	
Body Fat%	**28.3 ± 5.5**	**24.5 ± 4.3**	**.022**
Systolic BP (mmHg)	113.6 ± 8.8	116.3 ± 9.7	.319
Diastolic BP (mmHg)	70.8 ± 7.1	70.5 ± 6.5	.905
Waist (cm)	71.4 ± 5.0	71.7 ± 3.9	.859
WHR	.73 ± .04	.72 ± .03	.692

(VPA = vigorous physical activity, BMI = Body mass index WHR = waist to hip ratio, BP = blood pressure).

5.4 Activity quartiles

When the percentage of MVPA recorded was split into quartiles, interquartile differences were seen with body fat (F = 4.896, (3,103) df, *P* =0.003, r = 0.35). Post

hoc analysis by the Tukey method showed a significant difference between quartile one (30.2 ± 4.7%) and quartile four (25.2 ± 5.2%). Other quartile differences were not significant, after the post hoc analysis (Table 9). Diastolic blood pressure showed significant differences between groups (F = 2.803, (3,103) df, *P* =0.044, r = 0.27). However post hoc analysis showed no significant differences in any of the quartiles.

Table 9: Differences in health risk between MVPA quartiles.

	MVPA quartiles				*P*
	1 (n =26)	2 (n =27)	3 (n = 27)	4 (n = 27)	
Range (mins/week)	(39 -172)	(174 – 254)	(255 – 364)	(365-718)	
Age (years)	32.8 ± 8.6	32.9 ± 8.8	31.7 ± 7.3	31.2 ± 6.9	.826
Body Fat%	**30.2 ± 4.7**	**29.3 ± 6.5**	**27.0 ± 4.3**	**25.2 ± 5.2#**	**.003**
Weight (kg)	61.1 ± 6.3	61.4 ± 5.1	63.0 ± 7.6	61.8 ± 7.8	.736
Waist (cm)	72.0 ± 5.2	71.6 ± 4.1	71.3 ± 5.2	70.9 ± 5.0	.444
WHR*	0.72 (0.70 - 0.76)	0.72 (0.70 -0.76)	0.73 (0.70 - 0.76)	0.71 (0.70 - 0.73)	.906
Systolic BP (mmHg)	115.4 ± 8.0	115.1 ± 10.4	112.0 ± 7.6	113.3 ± 9.2	.462
Diastolic BP (mmHg)	**72.1 ± 7.1**	**72.2 ± 7.3**	**67.5 ± 4.7**	**71.2 ± 7.8**	**.044**

All tests are ANOVA (Mean ± SD) unless stated.

*Kruskal-Wallis ANOVA: Median (25^{th} – 75^{th} percentile).

#significant difference from quartile 1

(MVPA: Moderate to Vigorous Physical Activity; BP: Blood Pressure; WHR: Waist to Hip ratio).

5.5 Correlations

Pearson's correlations were performed to assess relationships between body fat and physical activity. MVPA was the only physical activity variable to have a significant correlation with a health measure, body fat percentage, explaining 7.4% of the variance. Partial correlations were then performed to determine the significance of MVPA independent of sedentary and light activity on body fat. This was found to be statistically significant for both sedentary (r [104] = -.258 *P* = 0.008) and light activity (r [104] = -.273 *P* = 0.005) but not both (r [103] = -.001 *P* = .994).

6.0 Discussion

6.1 Findings

The central aim of this study was to investigate the associations of objectively measured physical activity behaviours on health risk in women with a normal BMI, but differing body fat levels. Using this approach, it was evident that those who achieved the recommended level of physical activity (defined as achieving 150 minutes of MVPA/week) had a significantly lower body fat compared to those not achieving the recommended levels.

Furthermore, when the intensity of the MVPA was separated into moderate and vigorous activity components, vigorous activity (achieving 150 minutes of activity greater than six METS per week) showed a significantly lower body fat percentage profile, whilst achieving 300 minutes of moderate (3-6 MET) activity per week demonstrated no differences in body fat. This suggests that it is the vigorous component of exercise (> 6 METS) that is associated with a reduced body fat profile, although achieving the recommended activity levels of 150 minutes per week still showed benefits on controlling this health risk. Simple correlations showed that the association of body fat with MVPA was still significant, even after adjusting for the amount of sedentary and light behaviour but not both.

This study helps to extend the current evidence by objectively measuring sedentary behaviour levels and physical activity levels and simple health risk measures, and assessing the associations in a homogenous group of participants with a normal BMI, and shows a benefit for MVPA and especially vigorous activity, with no clear benefits for reduced sedentary behaviours with respect to body fat.

The evidence for the association with body fat percentage and physical activity has been acknowledged previously. den Hoed and Westerterp (2008) assessed physical activity via accelerometers over a two week period, and determined body fat percentage via underwater weighing in 134 participants (80 women and 54 men). Body fat percentage was associated with higher activity levels, and in women body fat percentage and physical activity were significantly associated, independent of

height, body mass and seasonality. Sternfeld et al. (2005) looked at body fat distribution in midlife women and the association with physical activity. Only vigorous physical activity was associated with a decreased body fat percentage with waist circumference decreasing from 96.2 cm in those doing no vigorous activity to 81.4 cm in those performing 10 minutes or more per day. Other evidence shows that higher intensity and/or longer duration physical activity was associated with lower body fat percentages when compared with shorter duration, lower intensity physical activity (Tucker & Peterson, 2003).

Recent evidence of the relationship between sedentary behaviour and health show that high levels of sedentary behaviour are associated with increased mortality and cardio-metabolic risk, though most studies have focussed on using TV viewing time as a surrogate to measuring total sedentary behaviour (Dunstan et al., 2010a; Hu et al., 2003; Katzmarzyk et al., 2009). However, TV viewing time is only one behavioural aspect of sedentariness, and it is important to include more objective measures as this may not be reflective of the overall pattern of an individual's sedentary behaviour. Although linked to increasing BMI, there is little objective data linking sedentary behaviours with increased adiposity, with most cross sectional and prospective studies using subjective assessments and BMI as an indicator of adiposity (Cameron et al., 2003; Hu et al., 2003; Vandelanotte et al., 2009).

Our results suggest women who spend a greater period of time engaging in MVPA, and specifically vigorous physical activity, have a significantly lower body fat percentage than those who do not exercise at the same intensity and duration. This was found irrespective of the time spent in sedentary behaviour or light activity. This finding supports previous research by Scheers et al. (2013) who found similar results, suggesting that risks associated with high amounts of sedentary behaviour can be compensated for by engaging in sufficient MVPA.

Our data, suggesting that MVPA has a greater influence on health risk, in this case adiposity, when compared to sedentary behaviour, is similar to other research that has focussed specifically on sedentary behaviour and cardio metabolic health

(Ekelund et al., 2009; Scheers et al., 2013) but contradicts evidence from others (Green et al., 2014; Healy et al., 2008c; Henson et al., 2013). These studies however used participants from a wider range of BMI levels, age, sex and had various degrees of metabolic impairments included in the study cohort. As our population was more homogenous, with normal BMI, it may be that to prevent increases in body fat, MVPA is the important behaviour to emphasise, whilst to decrease body fat in those already classified as overweight or obese a focus on MVPA as well as aiming to decrease sedentary behaviours is important. This concept may be seen in the data from Henson et al. (2013), who assessed a cohort of already overweight and obese participants with known risk factors for type II diabetes. They found a stronger association for sedentary behaviour than MVPA on cardio metabolic health (2 hour glucose, triacylglycerol, HDL – cholesterol), with associations for BMI and waist circumference associated with MVPA, which contrasts our findings for sedentary behaviour in normal weight individuals.

Current evidence suggests that to prevent weight gain (defined as <3% change in body weight) a target of 150-250 minutes per week of moderately vigorous exercise is recommended (Donnelly et al., 2009). Our data would add weight to this recommendation, and suggest that aiming for 150 minutes per week of vigorous exercise correlates with significantly lower adiposity. This suggestion may have significant implications on exercise recommendations, as a major focus of activity research has been placed on identifying the dose response required to reduce the risk of mortality and chronic disease, and 150 minutes per week has been well evidenced to achieve this (Hu et al., 2005) (Leitzmann et al., 2007). However, this may not be enough exercise to prevent weight gain, with Lee et al. suggesting that 420 minutes per week of moderate intensity activity is required to reduce risk of significant weight gain in women over a 13 year period (Lee et al., 2010). Recent evidence from Norway shows that reduced weight gain over a 22 year period was only associated with activity levels greater than the current recommendations (≥ 150 minutes per week of moderate intensity, or ≥ 60 minutes per week of vigorous intensity activity per week) (Moholdt et al., 2014) .

This contrast in exercise outcomes therefore invites the question: if current recommendations are suitable for reducing chronic disease and mortality risk but not enough to prevent weight gain, is it feasible to be overweight or obese but still active and healthy with a reduced mortality risk?

This question becomes more relevant when evidence shows that the levels of overweight and obesity in New Zealand are among the highest in the developed world (Ng et al., 2014), and when it is considered that the majority of people who lose weight do not maintain this weight loss over time (Katan, 2009). Data from the ACLS studies show that cardio respiratory fitness reduces the risk of all cause CVD and cancer mortality associated with obesity (LaMonte & Blair, 2006). Blair (2009) suggests that data from the ACLS studies show that obesity is more strongly associated with the risk of type 2 diabetes than is physical activity, but physical activity is as important as obesity in the development of type 2 diabetes. This suggests that physical activity is important, but especially in the prevention of weight gain to limit obesity development and the associated health risks that come with this. Our data again emphasises the importance of vigorous intensity exercise in maintaining "healthy" body composition levels which may prevent the development of obesity.

There are limited studies that have prospectively followed changes in body composition, especially FFM or body fat, related to exercise habits. A prospective study in women over 20 months, using accelerometer derived physical activity measures and body fat percentages, showed that those who increased their physical activity tended to show a decrease in body fat whilst those who decreased their physical activity tended to show increases in body fat (Bailey et al., 2007). As a whole the cohort increased their body fat percentage and body weight over the study period. Using the EPIC study data, Ekelund et al. (2011) found that, although physical activity was not significantly associated with weight gain, it predicted waist circumference.

We found no effects on any other health measure other than body fatness, but this is only one component of metabolic dysfunction that can be assessed. The National

Cholesterol Education Program (NECP) defined the criteria for metabolic syndrome, with three or more risk factors required for a diagnosis (Cleeman et al., 2001). Along with elevated waist circumference and blood pressure, elevated triglyceride levels, low high density lipoprotein (HDL) levels and fasting glucose levels are used. Evidence suggests that even with a normal BMI, elevated body fat is associated with metabolic dysfunction (De Lorenzo et al., 2005; Di Renzo et al., 2010; Dvorak et al., 1999; Karelis et al., 2004).

Dvorak et al. (1999) showed significantly higher body fat levels in those defined as metabolically obese normal weight and Karelis et al. (2004) described subgroups of individuals with normal weight and BMI, but differing body fat profiles, who display multiple metabolic abnormalities including insulin resistance, hyperinsulinaemia, and dyslipidaemia. Others have assessed markers of the MetS in normal weight individuals (<25 kg/m^2) with increased body fat percentage (> 30%) compared to a normal body weight and body fat control group and those with elevated BMI (≥ 25 kg/m^2) (De Lorenzo et al., 2005).Significant differences from the control group in HDL cholesterol and the LDL/HDL cholesterol ratio were noted, indicating some degree of metabolic dysfunction.

Interestingly, the research by Green et al. (2014), although contradictory in its conclusion to ours, albeit with a differing BMI range, used a similarly aged population and has shown that $\dot{V}O_{2peak}$ may be an important mediator of cardio metabolic health independent of physical activity behaviours. $\dot{V}O_{2peak}$ has been shown to be an independent risk factor for cardio metabolic disease (Abdulnour et al., 2010). The authors suggested that this may be an important factor to control for in further research, which we have not done. They also suggest that in addition to developing interventions to reduce sedentary behaviour, a focus on increasing $\dot{V}O_{2peak}$ rather than MVPA may be important for reducing CVD risk. Our results may suggest similar, with better body fat profiles seen in those individuals achieving higher MET activity, which is a key method of increasing $\dot{V}O_{2peak}$ (Gormley et al., 2008; Helgerud et al., 2007).

Further, it has previously been shown that those classified as NWO have a significantly lower resting metabolic rate (RMR) compared to the pre-obese-obese (defined as BMI > 25 kg/m^2 and clinically healthy) of approximately 200 kcal/day (De Lorenzo et al., 2005). It has also been seen that physical activity expenditure measured by doubly labelled water in individuals defined as metabolically obese normal weight was significantly lower when compared to healthy controls (Dvorak et al., 1999). Higher intensity activity and resistance training may help promote an increase in lean tissue, mediating changes in metabolic rate and promoting better metabolic control (Strasser, 2013).

The lack of association with sedentary behaviours and adiposity may be considered surprising given that there is a demonstrated relationship between sedentary behaviour and increased risk of becoming overweight (Hamilton et al., 2007), and increased waist circumference (Healy et al., 2008c). Recent evidence has also shown that sedentary behaviour increase cardio metabolic risk, regardless of the amount of daily accumulated MVPA (Green et al., 2014).

As mentioned, underlying characteristics of metabolic dysfunction were not assessed, but previous evidence has demonstrated a relationship between sedentary behaviour and markers of insulin resistance (Dunstan et al., 2007; Hu et al., 2003). However this was seen in populations where body weight was not controlled for, as in this study. Also we did not measure bouts of sedentary behaviour, which has been shown to correlate with metabolic risk (Healy et al., 2008a), so this adjunctive measure, and measures of metabolic health, might show some associations that we have not assessed.

6.2 Recommendations

Making public health recommendations for physical activity is difficult, as many factors need to be considered, not least of which is how realistic the targets are. When comparing overweight and obese participants to normal weight participants matched for age gender and height, only 13% of overweight and obese and 26% of normal weight participants met the Institute of Medicines (2002) recommendations of 60 minutes of moderate intensity exercise daily (Davis et al., 2006). Our data is similar, with 22% meeting 300 minutes of moderate intensity exercise per week and

11% meeting 150 minutes of vigorous intensity exercise per week suggesting that although the benefits in favourable body composition are present as a public health message this may be unobtainable for a large percentage of the population. Future research should identify if further metabolic benefits can be achieved with these activity levels. Conversely, 82% of individuals in this study met the current recommendations, which still showed significant benefits to body fat percentage when they were achieved.

This study extends the current literature on the understanding of risk factors in those with NWO and correlations with physical activity in this group, which have not been well researched. Strengths of the study include the use of an objective measure to assess sedentary behaviour and physical activity, and the use of accurate body composition assessment measures.

6.3 Limitations

It is important to note however that as a cross sectional study this result cannot identify cause and effect. Thus it is not known if those exercising at higher volumes and intensities have lower body fat because of the exercise they perform or if their body fat levels encourage them to participate in more intensive exercise. A lack of generalisability to the main population may also be considered a limitation of the study due to the specific cohort of women used and further research should assess if ethnic differences exist. We have used an arbitrary value to determine healthy body composition at <30% body fat. This has previously been utilised by Dvorak et al. (1999), and other research has indicated a similar value (30.7%) when using the highest quintile of body fat % to classify risk of CVD (Tanaka et al., 2002). Further research should aim to clarify healthy body fat levels.

6.4 Future research

As discussed, more investigations into metabolic differences between normal and excessive body fat, normal weight individuals, and comparisons between exercise habits should also be investigated. Behavioural aspects of sedentary behaviour and physical activity, such as the assessment of bouts of activity or sedentary behaviour, could further clarify the effects of physical activity in this cohort.

Further research should assess if there are differences in the metabolic make-up of the NWO group that can be explained by physical activity behaviours. As well, there needs to be a greater emphasis on evidence from prospective studies, with more specific body composition measures, in order to link changes in mortality or disease risk with body composition, adiposity and exercise habits.

6.5 Conclusions

In conclusion, achieving 150 minutes per week of MVPA was associated with a lower body fat percentage in a population of women with a normal BMI by differing body fat profiles. Achieving 300 minutes of moderate activity per week showed no significant differences but achieving 150 minutes per week of vigorous activity showed further improvements in body fat. Further, percentage of time spent in MVPA was inversely associated with body fat percentage, even when controlling for the amount of time spent sedentary. The results of this study suggest that aiming for more vigorous activity may be important for maintaining a reduced body fat profile in those with normal bodyweight.

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8.0 Appendices

Appendix A: Consent form

Appendix B: Anthropometry and blood pressure data collection form

Appendix C: Accelerometer information sheet

Appendix D: Personal information, health and demographics questionnaire

Appendix A

MASSEY UNIVERSITY
COLLEGE OF HEALTH
TE KURA HAUORA TANGATA

Institute of Food, Nutrition and Human Health
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T 09 414 0800
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EXPLORE Women's study

PARTICIPANT CONSENT FORM

This consent form will be held for a period of five (5) years

I have read the Information Sheet and have had the details of the study explained to me.

My questions have been answered to my satisfaction, and I understand that I may ask further questions at any time.

I agree to participate in this study under the conditions set out in the Information Sheet.

Signature: ..... **Date:** .....

Full Name - printed

Appendix B

EXPLORE Women's Study (2014)

Anthropometry & blood pressure
Data sheet - phase 2

Body Composition INDICATOR	MEASUREMENT	FINAL VALUE
Height	1.	
	2.	
	3.	
RECORDER:		
Weight		
Waist circumference	1.	
	2.	
	3.	
Hip circumference	1.	
	2.	
	3.	
RECORDER:		
Blood Pressure	1.	
	2.	
	3.	
RECORDER:		

Appendix C

MASSEY UNIVERSITY
COLLEGE OF HEALTH
TE KURA HAUORA TANGATA

Women's EXPLORE Study

What is an ActiGraph device?

- An ActiGraph device is a motion sensor which measures your movement. It is an expensive piece of equipment so it is important you take good care of it and return it as soon as you have finished wearing it!
- Your ActiGraph device is threaded onto an elastic belt on which it must remain.

How do I wear the ActiGraph device?

- The elastic belt with the ActiGraph device on it should fit firmly around your hips (under your clothes).
- The ActiGraph device can be taken on and off using the clip fastener on the elastic belt.
- The ActiGraph device must be positioned on the **right hand side of your waist/hip**, just above your hip bone and in line with your armpit as shown in Fig 1 and 2.

Correct

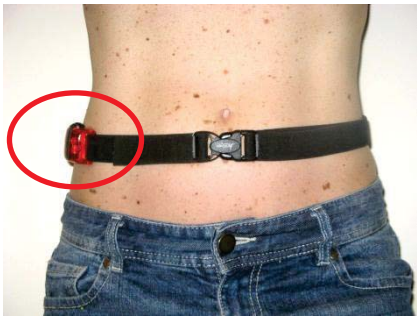

Fig 1 Right hip/waist

Correct

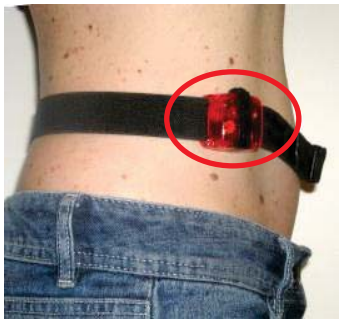

Fig 2 Right side

- The ActiGraph device must be worn on the **right waist/hip**, not in the centre of your body as in Fig 3.
- The elastic belt must **not sag or be twisted** (Fig 2) and must be **FIRM** around your hips (Fig 1).
- Make sure the ActiGraph device is in an upright position and is not tilted as in Fig 4.

Incorrect

Fig 3 Not in centre of body

Incorrect

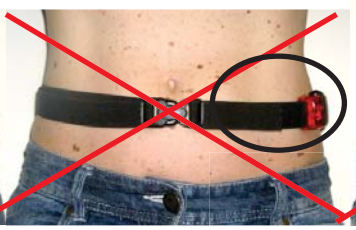

Fig 4 Not on left side

Incorrect

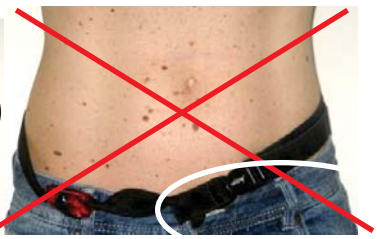

Fig 5 Not loose, must be firm around waist/hip

Please DO NOT wear the ActiGraph device in an incorrect place or position. ActiGraph devices worn incorrectly (as in Fig 3, 4 or 5) will NOT work properly.

When do I wear the ActiGraph device?

You need to wear your ActiGraph device for **8 days** ……..

Starting today when you receive the device

Finishing any time after midday on

_____

- Please wear the ActiGraph device **ALL THE TIME (**except for showering or water based activities like swimming), including to bed and during sport or exercise.
- Reposition the ActiGraph device above your right hip first thing in the morning as soon as you wake up and any time during the day when it slips or moves.
- The ActiGraph device may be taken off **ONLY** when doing **water based activities** where it would get completely wet or submerged (e.g., swimming or showering). The device should be worn for other water activities such as kayaking where it won't get completely wet.
- **Remember,** if the ActiGraph device is removed at any time (e.g. swimming, showering):
    - Put it back on as soon as you have finished.
    - Be sure to record in your activity diary when the ActiGraph device was removed and put back on.
- If possible, wear the ActiGraph device under clothing at all times.
- The ActiGraph device must remain on the elastic belt at all times and should be put on and taken off using the clip fastener.

What information do I need to record in the Physical Activity Diary?

- We need to know when the ActiGraph was worn and not worn during each day (e.g., taken off for a shower).
- **If the ActiGraph device is taken off,** please note the time when it was removed and replaced and what activity you were doing during that time.
- We'd also like to know about any activities you do with the aim of improving your health or fitness, or for sport, e.g. if you go for a walk, or to the gym or bike riding. For these activities, record for each day, the start time, length of time you did the activity, and the intensity (light, moderate or vigorous as explained on your diary).

If you have any queries, questions or concerns whilst wearing the ActiGraph, please don't hesitate to contact: Wendy O'Brien on 027 276 7796 or w.j.obrien@massey.ac.nz

Appendix D

Subject Number:

MASSEY UNIVERSITY
COLLEGE OF HEALTH
TE KURA HAUORA TANGATA

Women's EXPLORE study

Personal Information, Health and Demographics Questionnaire

First name: _____

Family name: _____

Name you would like to be called by: _____

Medical Practitioner: _____

Address: _____

Phone: _____

What is your first language?

English ☐

Other ☐

If other, please state:_____

I would like to receive a brief report summarizing the main findings of the project:

Yes □ No □

I am willing to be contacted in future research projects within the Institute of Food, Nutrition and Human Health:

Yes □ No □

Health and Demographic information

Do you have children? Yes □ No □

- How many children do you have? _____

- When was your youngest child born? __ / __ / ____ (DD/MM/YYYY)

When did your last period start? (Day / month / year) _____

Are you pregnant? Yes □ No □

Do you have any surgical or cosmetic implants? Yes □ No □

Are you currently in paid employment? Yes □ No □

If yes,

Full time Yes □ No □

Part time Yes □ No □

If yes, specify hours per week:_____

Describe your job or paid employment or work:

TITLE / DESCRIBE HOURS PER WEEK

_____

_____

Do you follow a specific diet for health reasons? Yes □ No □

Please explain

_____

_____

Do you follow any diet for cultural or religious reasons? Yes □ No □

If yes, what type of diet do you follow?

_____

_____

_____

Are you taking any form of medication, including traditional or homeopathic medicine and contraception?

Yes □ No □

Please specify the condition, the medication and the dosage in the table provided.

Condition	Medication	Dosage	Frequency

Are you taking any form of supplements, including tablets or drinks? Yes □ _____ No □

If yes, what are the name, brand and dosage of the supplements you are taking? _____

(Will send details by email Yes □ No □)

Supplement	Brand	Dosage	Frequency

Do you smoke cigarettes? Yes □ No □

If yes, approximately how many cigarettes per

day:_____

Do you drink alcohol? Yes □ No □

If yes, approximately how many standard drinks per week:_____

_____ _____

[1 standard drink = a glass of wine (120ml), 1 bottle/can of beer, I tot of spirits (45mL)]

Do you have any allergies? Yes □ No □

Please specify _____

_____

Please tell us how you found out about the Women's EXPLORE study. Did you found out from:

- A friend?
- If yes, what is him/her Name?.....
- An email list?
    - If yes, what is the name of the email list?.....
- At an event?
    - If yes, which event?.....
- Flyer on noticeboard?
    - If yes, where was the noticeboard?

      .....
- Other……

  ……..……

  ……