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**Fitness, Fatness and Fibre Type:
Predicting Insulin Resistance in Māori**

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‘Tama tu tama ora, tama noho tama mate’

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List of Abbreviations

ADA – The American Diabetes Association

BMI – Body Mass Index

CO₂ – Carbon dioxide

CRP – C - reactive protein

CVD – Cardiovascular disease

FFA – Free fatty acid

Gluc2hr – Glucose measurement 2 hours post-glucose load

GlucFast – Fasting glucose

HAD – 3-hydroxyacyl-CoA-dehydrogenase

HDL – High-density lipoprotein

HOMA-IR – Homeostasis model assessment – insulin resistance

IDDM – Insulin dependant diabetes mellitus

IMTG – Intra myocellular triacylglycerol

Ins2hr – Insulin measurement 2 hours post-glucose load

InsFast – Fasting Insulin

IR – Insulin resistance

LBM – Lean body mass

LDL – Low-density lipoprotein

MoH – Ministry of Health

NIDDM – Non-insulin dependant diabetes

NZDep – New Zealand Index of Deprivation

NZEO – New Zealanders of European origin

NZHS – New Zealand Health Survey

O₂ – Oxygen

OGTT – Oral glucose tolerance test

PFK – Phosphofructokinase

PHO – Primary health organisation

SDH – Succinate dehydrogenase

SPARC – Sport and Recreation New Zealand

T50 – Time to 50% of peak force (See Study 4 and 5)

TPF – Time to peak force

VO₂max – Maximal aerobic capacity

WHO – World Health Organization

Explanation of Māori terms

Hapu – clan, tribe, sub-tribe

Hauora – wellness, health

He Korowai Oranga – Māori health strategy developed to set the direction for Māori health development in New Zealand

Hinengaro – mind, thoughts: In the context of Māori health, hinengaro represents mental health and wellbeing

Iwi – tribe, nation, people; Often refers to a large group of people descended from a common ancestor

Kapahaka – Māori cultural performance group, performing Māori dances, haka, waiata (songs) etc. in a group

Karakia – chant, prayer

Kaupapa Māori research – research methodology based on Māori ideology, values

Rangatahi – younger generation, youth

Tamariki – children

Tapu – sacred, set apart, forbidden

Te Whare Tapa Whā – Māori health model with the four aspects of health represented as the four walls of a house; developed by Mason Durie

Tikanga – correct procedure, customs, habits, method

Tinana – physical body: In context of Māori health, tinana represents physical wellbeing

Tino rangatiratanga – The idea of self-determination, self-governance, Māori sovereignty.

Wairua – spirit, soul: represents spiritual wellbeing in the context of Māori health

Whakatātaka – The Māori health action plan

Whānau – extended family, family group

Whanaungatanga – relationship, kinship, sense of family connection

1 Introduction

The prevalence of type-2 diabetes has increased dramatically in recent years, in New Zealand and around the world. As the global burden of diabetes increases, certain ethnic groups seem to be hit hardest when compared with those of European descent. This is true in New Zealand where incidence, prevalence, death rates and complications associated with diabetes are all greater in Māori when compared to New Zealanders of European origin (NZEO). (Joshy & Simmons, 2006; Ministry of Health, 2008).

Insulin resistance, a metabolic disorder which reduces disposal of circulating glucose, has been identified as the underlying cause for type-2 diabetes. Increased adiposity (obesity) and an inability to oxidize lipid appears to disrupt the cellular response to insulin, leading to chronic hyperglycaemia, β -cell destruction, and ultimately diabetes.

While ethnicity is related to many socioeconomic factors that may contribute to increased prevalence of obesity and therefore diabetes, many ethnic groups appear to be more 'insulin resistant' even when socioeconomic factors are taken into account. This leads to the question of whether Māori and other high-risk ethnic groups have a genetic predisposition to diabetes.

Along with increased adiposity, a decreased oxidative capacity (aerobic fitness of skeletal muscle) (C. R. Bruce et al., 2003), as well as a higher percentage of type II (fast twitch) muscle fibre, are associated with insulin resistance and the obesity that leads to it. Nearly all of the studies observing these relationships have been done in Europeans however.

If identified early, insulin resistance can be treated before the disorder progresses to β -cell dysfunction and diabetes. Because use of health services is also low in Māori, it can be

difficult to identify those most at risk of insulin resistance. Thus, development of non-invasive methods to predict risk of metabolic disorder could prove valuable in the identification and prevention of type-2 diabetes in Māori.

Because of cultural barriers, participation of Māori in physiological research has been low, making it difficult to understand the physiological reasons, if any, behind ethnic health disparities in New Zealand. In order to overcome these barriers, the approach to research and intervention must also be led by Māori, and employ culturally appropriate methods.

2 Literature Review

2.1 Overview of Literature review

The aim of the first section of this review is to describe the incidence and prevalence of type-2 diabetes, specifically in New Zealand and within Māori. This background will include a brief definition and the diagnostic criteria for diabetes, discuss the inequalities regarding diabetes that are seen within the population, and present what is being done to lessen the burden of diabetes within New Zealand. It will also briefly address the issue of insulin resistance, although it will be discussed in more detail later.

The remainder of the literature review discusses the physiological mechanisms that lead to insulin resistance focusing on disruptions in lipid and glucose metabolism in skeletal muscle, and the effect of exercise on these metabolic processes. The review will also discuss possible physiological explanations for the disparities seen between different ethnicities in obesity and insulin resistance, and methods used to predict the disorder.

2.2 Diabetes and Insulin resistance in New Zealand and Globally

2.2.1 Definitions of diabetes & diagnostic criteria

Diabetes mellitus is considered a metabolic disorder, characterised by chronic hyperglycaemia and metabolic disturbances as a result of disrupted insulin secretion and/or insulin action (Alberti, Zimmet, & World Health Organisation, 1998). The word diabetes is derived from a Greek word meaning 'to pass' or 'urine', while mellitus is a Latin derivative meaning 'honey' or 'sweet'; The combination of these words was given due to the glucose-rich urine passed by those with the disorder ("Stedman's medical dictionary", 2000). Diabetes is classified into a number of categories, including type-1, type-2 and gestational diabetes; this review will focus on the most common of the classifications; Type-2 diabetes, previously known as 'non-insulin dependent diabetes mellitus' (NIDDM) and 'adult onset diabetes'.

Type-1 diabetes, previously identified as 'insulin dependant diabetes mellitus' and 'juvenile onset diabetes', is a result of disrupted insulin secretion due to an initial disorder within pancreatic β -cells. This disorder can occur in non-obese individuals and most often presents in children. Although the cause of the initial β -cell dysfunction in type-1 diabetes is unknown, it is thought to be genetically linked (Atkinson & Maclaren, 1994). Because dysfunction of β -cells disrupts insulin secretion, blood glucose levels remain chronically high signalling the diagnosis of diabetes. The anti-lipolytic role of insulin is also absent, so ketosis, and loss of body weight, are symptoms upon presentation (Alberti, Zimmet, & World Health Organisation, 1998).

Type-2 diabetes is also characterised by decreased insulin secretion and hyperglycaemia, but the β -cell disorder that leads to the diabetic state stems initially from a condition known as

insulin resistance. In this case, blood glucose levels become chronically elevated similar to type-1 diabetes, but initially the glucose level remains high regardless of normally sufficient insulin secretion. In response to this glucose intolerance, β -cells compensate by secreting more insulin, and the hyperglycaemia is controlled depending on the β -cells ability to maintain the elevated levels of insulin needed (Reaven, 1988). Thus, in type-2 diabetes, rather than the disorder initiating from disrupted insulin secretion, elevated blood glucose levels remain high due to myocytes and adipocytes 'resisting' the insulin signal; hence the term 'insulin resistance'. Although the precise mechanisms responsible for insulin resistance are still not fully known, the disorder is broadly a result of obesity (B. B. Kahn & Flier, 2000; Reaven, 1988), and can be avoided with lifestyle change (Kelly, 2000).

Since French physician Lancereaux first made the distinction between 'fat' and 'thin' diabetes in the late 1800s (cited in Gale, 2001), the definition of diabetes has been altered many times. During a meeting between The World Health Organisation (WHO) and The American Diabetes Association (ADA), it was recommended that the terms 'insulin dependant diabetes mellitus' and 'non insulin dependant diabetes mellitus' and their acronyms 'IDDM' and 'NIDDM' be no longer used. This recommendation was made because the previously described terms caused confusion and often led to patients being classified according to their treatment rather than pathogenesis (Alberti, Zimmet, & World Health Organisation, 1998). Similarly, the terms 'juvenile' and 'adult' onset diabetes are no longer used because children are presenting with type-2 diabetes in increasing numbers and type-1 can be first diagnosed in adulthood.

Along with its definition, the diagnostic criteria for diabetes have also changed many times, and variation in criteria between different health organisations has led to confusion regarding

its diagnosis. While symptoms that prompt diagnosis include an increased thirst and urine volume, recurrent infections and unexplained weight loss, clinical diagnosis of diabetes is often made by measuring fasting glucose concentration and performing an oral glucose tolerance test (OGTT). With an OGTT, blood glucose levels are measured while fasted and then at incremental periods for 2 hours after an oral glucose load of 75g (Matsuda & DeFronzo, 1997). The rationale behind the OGTT is that those who are insulin sensitive will return to fasting (or near fasting) glucose levels within 2 hours of the glucose load, while a relatively high blood glucose level, 2 hours after the glucose load, is indicative of glucose intolerance and diabetes. In 1998 the WHO lowered their diagnostic criteria for diabetes from a fasting plasma glucose concentration of 7.8 mmol L⁻¹ and above, to the currently accepted value of 7.0 mmol L⁻¹ and above, and/or a plasma concentration of 11.1 mmol L⁻¹ and above for a 2-hour post glucose load (Alberti, Zimmet, & World Health Organisation, 1998).

2.2.2 Prevalence, incidence and mortality

In 1998, WHO predicted that non-communicable diseases, such as type-2 diabetes, would become more prevalent in developing countries due to adopted Western lifestyles and the risk factors, such as smoking, high energy diets and lack of exercise, that accompany such a lifestyle (WHO, 1998). Since then diabetes has become a global epidemic, increasing in prevalence and incidence in most of the developed world. One study estimated the global prevalence of diabetes at 2.8% in 2000, and predicted that this would increase to 4.4% in 2030 (Wild, Roglic, Green, Sicree, & King, 2004). In New Zealand the burden of diabetes has become an area of concern as well, with national diabetes prevalence exceeding global estimates. The 1996/97 New Zealand Health Survey (NZHS) showed that 1 in 27 (3.7%) adults in New Zealand, were diagnosed with diabetes (Ministry of Health, 1999b). Ten years later, the 2006/07 survey showed that prevalence of diagnosed diabetes had increased to

approximately 5.0% (1 in 20 adults) (Ministry of Health, 2008). What's more, the burden of this disorder will likely be compounded in years to come, as the population of the groups that seem to be most at risk of diabetes (Māori, Pacific Islanders, Indian and Asian) are increasing in numbers dramatically (Moore & Lunt, 2000). As is the case in most countries, type-2 diabetes accounts for the majority of diabetes cases in New Zealand (Ministry of Health, 2004b, , 2008).

Table 2-1 – Age adjusted prevalence of diabetes within New Zealand by year

Year	Prevalence
NZHS 1996/97	3.7%
NZHS 2002/03	4.3%
NZHS 2006/07	5.0%

The prevalence of diabetes differs between ethnicities in New Zealand similar to other countries. The 1996/97 New Zealand Health Survey (NZHS) showed the prevalence of diabetes at 3.1% in Europeans compared with 8.3% of the Māori population (Ministry of Health, 1999b). The age standardized rate ratios in the 2006/07 NZHS (where 1.00 equals the national average) were 1.74 for Māori men and 1.61 in Māori women, compared to just 0.76 in European men and 0.82 in European women (Ministry of Health, 2008). Another study done in South Auckland found that prevalence of the metabolic syndrome and known diabetes was greater than 50% in the Māori and Pacific people aged 40 years and older that were tested, much higher than observed in European groups (David Simmons & Thompson, 2004).

2.2.3 Prevalence of obesity

As mentioned earlier, obesity, as a result of chronic caloric excess i.e. relatively low energy expenditure for energy intake, has been identified as the key factor leading to insulin resistance, and is therefore associated with type-2 diabetes (Hohepa, Schofield, & Kolt, 2004). A population-based study in the US showed that obesity was the most important risk factor for insulin resistance in adolescents, independent of sex, age or ethnicity (J. M. Lee, Okumura, Davis, Herman, & Gurney, 2006). Others have shown that 'Western' dietary patterns are associated with obesity and a higher risk of insulin resistance, while 'healthy' dietary patterns on the other hand, are associated with lower risk of insulin resistance (Esmailzadeh et al., 2007). Accordingly, studies in Europe have revealed that reduction of weight within a population is associated with a similar reduction in diabetes incidence (Astrup, 2001). Intervention programs in the United States, which aimed at reducing weight with physical exercise and healthy diet, also reduced the risk of diabetes in those who participated (Hamman et al., 2006).

These studies portray the close association between obesity and diabetes prevalence. It is therefore no surprise that the prevalence of obesity in New Zealand is increasing at a rate similar to the increased prevalence of insulin resistance and diabetes. Results of the 2002/2003 New Zealand Health Survey (NZHS) showed that 35.2% of adult New Zealanders (15 years plus) were considered overweight, and 20.9% were obese (Parnell, Scragg, Wilson, Schaaf, & Fitzgerald, 2003). Four years later, the 2006/2007 NZHS showed that the prevalence of overweight adults in New Zealand had increased to 36.2%, and obesity had increased to 26.5% (Ministry of Health, 2008).

Notably, the rates of participation in sporting clubs and organised teams has fallen considerably (Bauman et al., 2003). Not only is participation in sport and leisure decreasing but advances in technology promote a sedentary lifestyle and decrease the need for physical labour. This decrease in daily physical activity coupled with less costly, readily available high-energy foods, are the primary cause for the growing prevalence of obesity and the accompanying co-morbidity of type-2 diabetes.

2.2.4 Mortality

Since it is the complications of diabetes that cause death and not diabetes *per se*, coding of diabetes-related mortality statistics can be flawed (Joshy & Simmons, 2006). In 2000, 3% of all deaths in New Zealand were attributed to diabetes (Ministry of Health, 2004a). Accordingly, a report prepared by the New Zealand Ministry of Health showed that 8% of all New Zealand deaths in 1996/97, were associated with physical inactivity (Ministry of Health, 1999b) making it the second highest preventable risk factor among New Zealanders (behind tobacco smoking).

Just as many illnesses are disproportionately higher in Māori than NZEO, so too is the general mortality rate compared to that in non-Māori (N. Pearce, Pomare, Marshall, & Borman, 1993). In 2004, the overall death rate was 7.0 (deaths per 1000) for all New Zealanders and 4.3 for Māori. When adjusted for age however, age standardized death rates for Māori, who are younger than the general NZ population, were 7.6 per 1000 compared with 4.5 per 1000 in the general New Zealand population respectively (Statistics New Zealand, 2006a, , 2006c). In 2005/07, life expectancy for Māori was 70 and 75 years for males and females respectively, compared with 79 years in non-Māori males and 83 in non-Māori females (Statistics New Zealand, 2008).

Similarly, ethnic disparities exist in diabetes-related mortality. In the United States, the risk of diabetes-related death in Indigenous Americans was 2.37 times higher than Caucasian Americans. However, even higher still was diabetes-related mortality in Māori which was 5.81 times higher than European New Zealanders (Bramley, Hebert, Tuzzio, & Chassin, 2005). In fact, this same study showed that, in regard to many health indicators, the disparity between indigenous and non-indigenous groups in New Zealand i.e. Māori and non-Māori, was greater than the disparity between indigenous and non-indigenous groups in the US.

The reason for the higher mortality rate in Māori, even compared to other indigenous groups, is not clear, though Moore and Lunt speculate that delivery of medical services, and the absence of a Māori focus, may contribute (Moore & Lunt, 2000). Interestingly, results of the 2006/07 NZ Health Survey showed that Māori were significantly less likely to report that a) their health provider had treated them with respect and dignity ‘all of the time’, b) their health care provider listened carefully to what they had to say ‘all the time’, and c) that their health care provider had discussed their health care as much as they wanted ‘all of the time’, when compared to the total population (Ministry of Health, 2008). In context with ethnic health disparities, use of medical services is partly reflected in the high prevalence of undiagnosed diabetes in Māori. One study showed that 7% of Māori respondents were undiagnosed diabetics, while 15.5% had impaired glucose tolerance (Joshy & Simmons, 2006). Another study found that in a cohort of Māori where 7.1% (n=247) were known diabetics, an additional 3.6% were undiagnosed diabetics (Tipene-Leach et al., 2004). This result is probably modest considering the response rate in this study (49%). Furthermore, 37% of those that did respond were insulin resistant.

2.2.5 Inequalities in diabetes prevalence

2.2.5.1 Age

Epidemiological studies show that diabetes prevalence increases with age (Ministry of Health, 2008). Nevertheless, where type-2 diabetes was once a disease common only among older age groups, it is now becoming a serious problem among adolescents. This increase in the incidence of type-2 diabetes in adolescents and children, is directly tied to the increasing prevalence of childhood obesity (Rocchini, 2002). Hotu et al. observed a 6-fold increase in adolescents with type-2 diabetes attending the Adolescent Diabetes Service in New Zealand between 1996 and 2002 (Hotu, Carter, Watson, Cutfield, & Cundy, 2004). Studies also indicate that the onset age of diabetes in Māori is almost 10 years earlier than Europeans (Joshy & Simmons, 2006). Accordingly, the median age for Māori is considerably lower than the median for New Zealand (22.7 years compared with 35.9 years) (Statistics New Zealand, 2006c), which may partly explain the earlier onset age of diabetes in Māori. Alternatively, Moore and Lunt suggest that the earlier onset age of diabetes in Māori, could be due to a longer lifetime exposure to conditions causing hyperglycaemia; namely caloric imbalance (Moore & Lunt, 2000) (See section 2.2.5.2).

2.2.5.2 Socio-economic status,

The prevalence of diabetes in those that live in the most deprived quintile of the NZDep2006 (quintile 5), is more than twice as high as those in the least deprived (quintile 1) (Ministry of Health, 2008). Accordingly, obesity is also highest in the most deprived quintile (Ministry of Health, 2008). A number of epidemiological findings may help to explain this association. Firstly, the 2006/07 NZHS showed that the time children spent watching television, as well as their 'fizzy drink' and 'fast food' consumption, were higher in areas of high neighbourhood deprivation (quintile 5) than in areas of low deprivation (Ministry of Health, 2008). Fittingly,

insulin resistance and diabetes have been shown to be associated with time spent watching television (Hu et al., 2001; Hu, Li, Colditz, Willett, & Manson, 2003), high consumption of 'soft drink' (Schulze et al., 2004) and 'fast food' (Pereira et al., 2005), and a high fat/high sugar (high energy) diet (Gittelsohn et al., 1998; Ministry of Health, 2008).

Another possible explanation is that physical activity and participation in sports and leisure may be perceived by the socioeconomically deprived as costly, discouraging those with low incomes from participating (Estabrooks, Lee, & Gyurcsik, 2003). A report by the Ministry of Health also suggests that those who are least deprived are better able to respond to social pressures associated with weight gain (Tobias, Paul, & Yea, 2006).

These trends have major implications for Māori who are proportionately over represented in the most deprived quintile, having an annual income approximately 20% lower than NZEO (Statistics New Zealand, 2006b). Accordingly, Māori children spend more time watching TV, and consume more 'fizzy drink' and 'fast food' than the general NZ population (Ministry of Health, 2008). What's more, hazardous drinking patterns are significantly higher in Māori adults which compounds the 'high-risk' health behaviours of Māori considering the relatively high calorie density of alcohol and its association with obesity (P. M. Suter, Hasler, & Vetter, 1997). Interestingly however, Māori are reportedly as active as, or more active than NZEO (Ministry of Health, 2008; SPARC., 2004). Another interesting finding is that the relationship between obesity and socioeconomic status in Māori men is counterintuitive; that is, elevated markers of overweight and obesity are associated with higher income and education (Tobias, Paul, & Yea, 2006). This suggests that the effect of socioeconomic status on obesity is modified by ethnicity and culture. One possible explanation for this may be the difference in cultural perceptions of body image and preferred body shape (Tobias, Paul, & Yea, 2006).

2.2.5.3 Ethnicity

Diabetes research comparing ethnic groups in New Zealand paints a grave portrait for the future of Māori health if this epidemic is allowed to continue on its current path. Among Māori, prevalence of known diabetes, as well as diabetes risk factors such as obesity and low HDL are higher than the general New Zealand population (Ministry of Health, 2008; David Simmons & Thompson, 2004). The prevalence of insulin resistance is also much higher in Māori even when BMI and body fat percentage is similar to European New Zealanders (K. McAuley, Williams, Mann, Goulding, & Murphy, 2002). Though studies show Māori to have higher levels of fat-free mass and lower body fat percentage than Europeans of the same BMI (Swinburn, Ley, Carmichael, & Palnk, 1999), Māori appear to be at greater risk of obesity related diseases at a lower BMI. This indicates that obesity related health risk differs between ethnic groups, with Māori being much more sensitive to extra body fatness (adiposity). One study by Simmons et al. challenged these findings and showed that when adjusted for obesity, Māori and other Polynesian groups seem to have relatively normal insulin sensitivity and do not have the usual marker of hyperinsulinaemia found in other high-risk ethnic groups (D. Simmons, Thompson, & Volklander, 2001). This finding would suggest that the high prevalence of diabetes is due to increased obesity in Māori and not necessarily an ethnic susceptibility to risk factors such as insulin resistance.

Although much debate surrounds the use of current BMI standards in Māori and other ethnic groups (see section 2.4.2), Māori appear much more obese than Europeans, when defined by current BMI standards (Moore & Lunt, 2000). The 2006/07 NZ Health Survey showed that prevalence of obesity within Māori was 41.7% compared with just 24.3% in Europeans (Ministry of Health, 2008). Pacific Islanders were even higher at 63.7%. In accordance with a

higher prevalence of obesity and diabetes, Māori diets are higher in fat than the general population (Ministry of Health, 1999a; Parnell, Scragg, Wilson, Schaaf, & Fitzgerald, 2003).

Not only is diabetes more prevalent among Māori, but Māori have more than double the risk of the disease progressing into associated complications such as blindness and renal failure (Joshy & Simmons, 2006). The fact that smoking has been associated with the progression of diabetes complications as well, makes the current situation even worse, as the MoH have shown that almost half of Māori adults are smokers, compared to 23% of European adults (Ministry of Health, 1999b). Correspondingly, Māori and Pacific people have poorer medium term metabolic control (HbA1c > 8%) than Europeans (40%, 51% and 23% respectively) (Joshy & Simmons, 2006).

Similar to New Zealand, ethnic disparities in diabetes prevalence can be seen around the world. For example, one study from a multicultural community in Hawaii showed that even when energy intake, physical activity, BMI and waist-hip ratio were accounted for, prevalence of type 2 diabetes and glucose intolerance was significantly higher among the Hawaiians, Phillipinos and Japanese when compared with those who identified as Caucasian (Grandinetti et al., 2007). Likewise, the prevalence of diabetes among some indigenous groups in Canada is more than 3 times that of the general population (Dannenbaum, Kuzmina, Lejeune, Torrie, & Gangbe, 2008), while the Pima Indians of Arizona are believed to have the highest prevalence of diabetes of any group in the world (Pratley, 2007).

A number of theories which have been postulated to explain these ethnic disparities in relation to genetic makeup, will be discussed later in this review (See section 2.7). On the other hand, environmental factors such as lifestyle play a large part in ethnic health disparities.

Accordingly, Zimmet (Zimmet, 1979) observed that prevalence of diabetes was low in aboriginal people of Australia and the Pacific that continued to live a traditional, physically active lifestyle, but high in those who adopted a sedentary, westernized way of living. Similarly, O'Dea (O'Dea, 1984) showed that the metabolic abnormalities associated with type-2 diabetes in a group of diabetic Australian Aborigines were greatly improved, and in some cases normalized, with just 7 weeks of living a traditional lifestyle. One implication of these findings is that intense exercise is not necessary for the maintenance of health in Māori, but that the everyday activities of a traditional lifestyle, such as farming, cultivation of land, fishing and physical labour, in conjunction with a healthy diet, may suffice.

Lastly, studies show that Māori are less likely to make use of government-based health services (Moore & Lunt, 2000). For example, the results of the Get Checked programme in 2004 showed that 63% of European diabetics and 92% of Pacific diabetics had a free annual check while only 27% of Māori with diabetes took advantage of this free check (Joshy & Simmons, 2006). Whether this is due to expense of health care, accessibility of the service and/or cultural perception is not entirely clear, however, this has been discussed in more detail in section 2.2.4.

2.2.6 The cost of diabetes

The estimated cost of obesity related health care alone in New Zealand in 2002 was NZ\$303 million per year (Ministry of Health, 2003), however, more recent costs would no doubt be greater. SPARC estimated the direct cost of physical inactivity in New Zealand to be NZ \$180 million per year, compared to Australia, with an estimated cost of AUS \$400 million per year (SPARC., 2004), proportionally lower considering the relatively larger population. Other figures show that the economic burden of Type-2 diabetes in New Zealand during 2001 was

close to NZ\$400 million which is predicted to rise to NZ\$1 billion by 2021 (PricewaterhouseCoopers, 2001). Much of these costs are due to lifestyle choices and could be greatly reduced with relatively small lifestyle changes. In fact, one study estimated that an increase in physical activity as small as 5% across the population would result in savings of NZ\$25 million per year (Bauman, 1997), though this figure may be much greater as it does not take into account the direct effect of physical activity upon diabetes cost. What's more, lifestyle change for those most at risk of insulin resistance and diabetes, is more cost effective at a personal level when compared with pharmaceutical interventions (Herman et al., 2005).

2.2.7 Summary

Population data paints a grave portrait for Māori who, when compared to NZEO, are worse effected by diabetes at all stages. Type-2 diabetes prevention begins with the promotion of healthy behaviours; a balanced diet along with adequate and consistent physical activity to ensure maintenance of a healthy body composition (Young, Reading, Elias, & O'Neil, 2000). Thorough research and appropriate interventions will come at a price, but considering diabetes is a preventable disease, and the potentially vast economic burden stemming from diabetes in coming years, it would be a wise public health investment.

2.3 Diabetes and Insulin Resistance: A metabolic disorder

2.3.1 Glucose Transport and Insulin signalling

After a carbohydrate-containing meal, complex sugars (polysaccharides) are broken down in the gut into simple sugars (monosaccharide) such as glucose (Crabtree & Garlick, 1993). Following absorption into the bloodstream, blood glucose concentration becomes elevated and in response β -cells in the pancreas secrete insulin, the hormone responsible for signalling the uptake of glucose into adipocytes (fat cells) and myocytes (muscle cells) (Mathers & Wolever, 2002; Wilcox, 2005). Insulin is then detected by receptor proteins on the cell membrane of these tissues, setting off a signalling cascade which facilitates the translocation of GLUT4, a transport protein which allows the transport of glucose across the cell membrane and into the cell (Bryant, Govers, & James, 2002). After glucose enters muscle cells it is quickly phosphorylated, then either stored as glycogen or used as a substrate for glycolysis. Adipose tissue also has a role in glucose disposal and responds to insulin by storing excess glucose as fat (Flatt, 1995; Winegrad & Renold, 1958). Although other glucose transporters exist, such as GLUT1 and GLUT5, GLUT4 is the most important glucose transporter in skeletal muscle and adipose tissue.

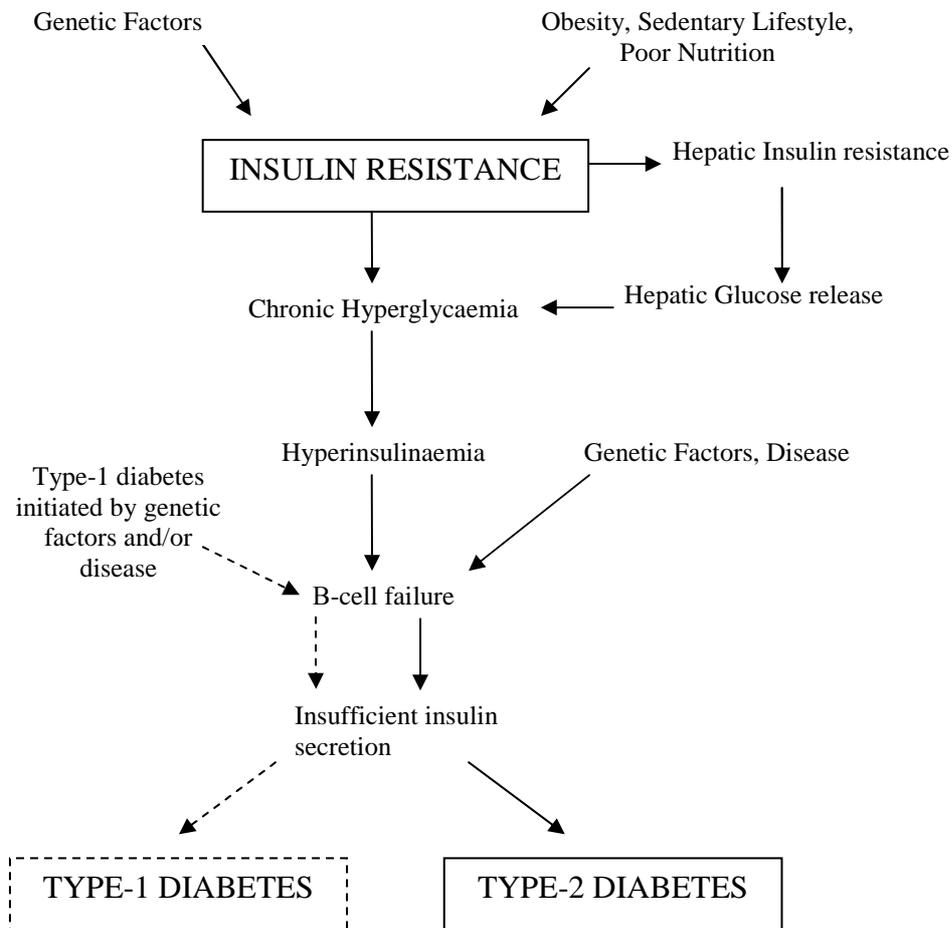
Along with signalling the uptake of glucose into myocytes, insulin also signals a reduction in the rate of lipolysis and increases the rate of esterification of free fatty acids (FFA) for storage as triglycerides in adipocytes (Campbell, Carlson, Hill, & Nurjhan, 2006). Similarly the liver responds to insulin by taking up glucose to be stored as glycogen, at the same time inhibiting gluconeogenesis and glucose output (Michael et al., 2000; Saltiel & Kahn, 2001). Thus, skeletal muscle, adipose tissue and the liver all contribute and play a major role in the regulation of lipid and glucose metabolism. Nevertheless, skeletal muscle is responsible for

up to 75% of glucose disposal following a meal (Baron, Brechtel, Wallace, & Edelman, 1988).

2.3.2 Insulin resistance and its role in type-2 diabetes

In insulin resistant individuals the insulin-signalling pathway in the insulin-sensitive tissues are disrupted and insulin-mediated uptake of glucose by myocytes and adipocytes is impaired. Initially, insulin is secreted in response to increasing blood glucose as expected, but the signalling cascade within the cell, which normally results in GLUT-4 translocation to the cell surface, is blunted (Garvey et al., 1998; Roden et al., 1996), reducing sensitivity to the insulin signal, 'insulin sensitivity', within these myocytes and adipocytes.

In response to the resultant hyperglycaemia, β -cells in the pancreas compensate by secreting more insulin to maintain control of blood glucose levels (Warram, Martin, Krolewski, Soeldner, & Kahn, 1990). This hyperinsulinaemia, if left untreated, worsens to the point where the pancreas cannot secrete enough insulin to maintain normal blood glucose levels, and blood glucose levels remain chronically elevated (Weir & Bonner-Weir, 2004). If β -cells cannot cope with the chronically elevated blood glucose levels and the continual resistance to secreted insulin, β -cell dysfunction can occur and blood glucose levels eventually reach the point at which diabetes is diagnosed i.e. a fasting blood glucose concentration of ≥ 7.0 mmol/l (Alberti, Zimmet, & World Health Organisation, 1998; Reaven, 1988). On the other hand, all persons with insulin resistance do not necessarily go on to develop hyperglycaemia. Large ranges exist in fasting insulin concentrations before hyperglycaemia occurs (Hollenbeck & Reaven, 1987), suggesting that some individuals possess a greater 'compensatory' ability to secrete insulin in response to hyperglycaemia.

Figure 1 - Development of type-1 and type-2 diabetes mellitus

It is believed that dysregulation of lipid metabolism, as a consequence of excess fatty acid (FA) availability and intracellular lipid accumulation, somehow interferes with the signalling cascade and translocation of GLUT4 in skeletal muscle, causing insulin resistance in myocytes (Boden & Shulman, 2002; Lewis, Carpentier, Adeli, & Giacca, 2002). What's more, when the liver becomes resistant to insulin, hepatic glucose release is no longer inhibited, increasing blood glucose further and compounding the hyperglycaemic state (Boden, 1997) (See **Figure 1**). These points highlight the importance of all three tissues (adipose, liver and muscle) in the development of insulin resistance and type-2 diabetes; as well as highlighting the complexity of the disorder which involves many different tissues and

physiological processes. The pathophysiology of insulin resistance will be discussed further in section 2.4.1.

2.3.3 Techniques to measure whole-body insulin sensitivity

The ‘gold standard’ method for measurement of whole-body insulin sensitivity is the glucose clamp technique (Ferrannini & Mari, 1998). The glucose clamp, or hyperinsulinaemic-euglycemic clamp, measures the amount of glucose that is needed to balance an artificial increase in insulin, without causing hypoglycaemia. Insulin is first infused into a peripheral vein followed by infusions of glucose at increasing rates, to compensate for the increased insulin and maintain blood sugar levels at 5 – 5.5 mmol/l. Glucose levels in the blood are monitored every 5 to 10 minutes for around 2 hours until a steady state of infusion is reached (DeFronzo, 1979). This usually occurs during the last 30 minutes of the test, where the ratio of glucose infusion to insulin concentration during this time is then calculated to determine insulin sensitivity (Wallace & Matthews, 2002). However, because this technique involves intra-venous (IV) infusion of insulin, frequent blood samples over a 2-3 hour period, and continuous infusions of glucose, the glucose clamp technique is not practical for large scale population studies.

A less costly and less arduous means of estimating insulin sensitivity is by using the glucose tolerance test (GTT). This is performed either by ingesting (oral glucose tolerance test, OGTT) or infusing (intravenous glucose tolerance test, IGTT) glucose, and then measuring the blood glucose and insulin response during the next two to three hours, while there is restoration of normal glycaemia. Mathematical models can then be applied to this data to infer insulin action. Perhaps the most popular model to date is the Homeostasis Assessment Model (HOMA-IR) which was first described in 1985 by Mathews et al. (Matthews et al., 1985).

HOMA-IR can be used to infer insulin sensitivity and β -cell function in the basal state, requiring only fasted insulin and glucose measurements. Thus, it is a simple and practical method of estimating insulin sensitivity and β -cell function in population studies and clinical practice. Nevertheless, some have shown that measurement of fasting insulin alone is just as effective at predicting insulin sensitivity as an OGTT (Stumvoll et al., 2000). In fact, studies in Māori have shown that HOMA-IR is no better than measuring fasting insulin alone, at predicting insulin sensitivity in this ethnic group (Bell, McAuley, Mann, Murphy, & Williams, 2004). Therefore, a fasting blood sample, and analysis of insulin, may be all that is required to screen for insulin resistance in Māori and others.

2.3.4 Summary

Type II diabetes is a disorder initially resulting from resistance to insulin, within insulin sensitive tissue. While the exact mechanisms which blunt the insulin response are not entirely understood, it appears that high concentrations of circulating and intracellular lipid, associated with obesity, interfere with the insulin signalling cascade, preventing GLUT-4 translocation and thus uptake of glucose from the blood.

2.4 Body Composition and Diabetes

2.4.1 Obesity and its role in insulin resistance

Mechanisms behind the relationship between body fatness and insulin sensitivity are still not fully understood. Nevertheless, obesity is associated with increased circulating free fatty acids (FFA) and intramyocellular triacylglycerols (IMTG or IMCL) concentrations (Goodpaster, Theriault, Watkins, & Kelley, 2000). This increase in FFA and IMTG is also associated with insulin resistance in skeletal muscle (Jacob et al., 1999; D. A. Pan et al., 1997). Because muscle is the most important insulin sensitive tissue (to glucose disposal), changes in muscle insulin sensitivity affect whole body insulin sensitivity.

Still, resistance to insulin in the liver also plays a role in whole body insulin sensitivity. As discussed earlier, under normal circumstances, insulin inhibits gluconeogenesis within the liver (Saltiel & Kahn, 2001). On the other hand, increased FFA has been shown to reduce the inhibiting effect of insulin on hepatic glucose release, adding to the hyperglycaemic condition in insulin resistant individuals (Boden, 1997). Interestingly, elevated lipid availability increases triglyceride accumulation in the liver (Oakes et al., 1994), similar to how IMTG accumulates in skeletal muscle, and this accumulation appears to be associated with hepatic insulin resistance (Seppälä-Lindroos et al., 2002). What's more, extraction of insulin, which is largely controlled by the liver, is reduced during hepatic insulin resistance, exacerbating the compensatory hyperinsulinemia in response to whole body insulin resistance (Polonsky et al., 1988).

Because insulin resistance can occur within liver, muscle and adipose tissue, there is debate regarding the primary site of insulin resistance (Caldwell, Ikura, Iezzoni, & Liu, 2007). Some

propose that excess adiposity causes insulin resistance within adipocytes primarily, which then initiates whole-body insulin resistance (Bergman & Mittelman, 1998). In a normal state, insulin secretion suppresses lipolysis (break down of fat) within adipocytes, facilitating storage of triglycerides into adipose tissue. When this tissue becomes insulin resistant however, lipolysis continues which increases circulating FFA (Boden & Shulman, 2002). As discussed previously, increased concentration of circulating FFA is associated with insulin resistance. However, it appears that IMTG accumulation has an even stronger relationship with insulin sensitivity, suggesting that increased FFA drives accumulation of IMTG, rather than effecting glucose uptake in skeletal muscle directly (Shulman, 2000). Certainly, situations which encourage increased FFA, also increase concentrations of IMTG in healthy persons, though with a delay (Stannard et al., 2002). On the other hand, general weight loss is associated with a decrease in IMTG (Goodpaster, Theriault, Watkins, & Kelley, 2000) and improvements in insulin sensitivity. However, Klein et al. found that liposuction decreased weight and body fat percentage but did not improve insulin sensitivity (Klein et al., 2004), suggesting that a decrease in adipose tissue alone is not enough to improve insulin sensitivity, but must also be accompanied by a negative energy balance. Although IMTG appears to be a marker of insulin resistance in healthy people, this was not observed in Māori (Stannard, Holdaway, Sachinwalla, & Cunningham, 2007), suggesting that the relationship between IMTG content and insulin sensitivity may be effected by ethnicity. It is unlikely however, that IMTG accumulation directly impairs insulin action. Rather, other related lipid compounds that build up within muscle cells, as IMTG accumulates, alter the insulin signalling pathway (Ellis et al., 2000).

One theory suggests that increased fat oxidation, from an oversupply of fat, competes with glucose as a substrate (Randle, Garland, Hales, & Newsholme, 1963; Shulman, 2000).

Randle et al. hypothesized that FFA induces insulin resistance by increasing the oxidation of fat relative to carbohydrate (Boden & Shulman, 2002; Randle, Garland, Hales, & Newsholme, 1963). They showed that an increase in fatty acids increased intramitochondrial acetyl CoA/CoA and NADH/NAD⁺ ratio causing inactivation of pyruvate dehydrogenase. This leads to an increase of intracellular citrate, inhibiting phosphofructokinase (PFK), an important enzyme which controls the rate of glycolysis. This would then lead to accumulation of glucose-6-phosphate (G-6-P) and inhibition of hexokinase II activity, therefore increasing intracellular glucose and reducing glucose uptake.

Others challenge this theory, suggesting that glucose transport is the primary factor effecting insulin sensitivity while enzyme activities, such as hexokinase II in glycogen synthesis, are secondary (Boden, Chen, Ruiz, White, & Rossetti, 1994; Dresner et al., 1999; Roden et al., 1996). These studies found that in healthy persons, the reduction in muscle glycogen synthesis associated with elevated FFA, was preceded by a fall in intramuscular G-6-P, rather than an increase as would be expected with Randle's theory, decreasing intramuscular glucose. These observations indicate that an increase in plasma fatty acid concentrations, inhibit glucose transport across the cell membrane, which in turn reduces the rate of phosphorylation (rather than mitochondrial ratios). This then results in reduced glycogen synthesis and glucose oxidation. Shulman suggests that fatty acid accumulation interferes with an earlier step in insulin stimulation of GLUT4 directly or the signalling pathways that signal GLUT4 translocation (Shulman, 2000). A series of later studies by this group confirm these findings (Boden & Shulman, 2002). As discussed previously, rather than FFA directly inhibiting glucose uptake, it is thought that metabolites of IMTG directly interfere with one or more stages of GLUT4 translocation. For example, long-chain fatty acyl CoAs, the activated form of intracellular FFA, and 1,2-Diacylglycerol (DAG), another intermediate of lipid

metabolism, have been shown to disrupt insulin signalling through activation of protein kinase C (PKC) which is associated with glucose disruption (Hegarty, Furler, Ye, Cooney, & Kraegen, 2003). Another lipid intermediate, ceramide, has been shown to directly inhibit the phosphorylation of insulin signalling molecules in muscle and adipose cells (Hajdich et al., 2001; Summers, Garza, Zhou, & Birnbaum, 1998). Nevertheless, the precise mechanism/s responsible for disturbed glucose transport is not entirely clear.

Interestingly, fasting studies have shown that extended periods of fasting result in elevated FFA concentrations, IMTG accumulation and impaired glucose uptake in healthy persons, similar to the insulin resistance seen in obese subjects (N A Johnson et al., 2006; Stannard et al., 2002). These findings suggest that, in an evolutionary context, IMTG accumulation and insulin resistance may have ensured an intra muscular fuel supply when food was scarce, whilst at the same time preventing muscle from taking up glucose to preserve it for the brain (Neel, 1962; Stannard et al., 2002).

To further compound the pathological effects of obesity, it appears that FFAs may have a direct effect upon secretion of insulin and β -cell dysfunction as well, independent of insulin resistance and compensatory hyperinsulinemia (Boden & Shulman, 2002; Unger, 1995; Unger & Zhou, 2001). Crespín et al. showed that FFA infusion stimulated secretion of insulin in dogs (Crespín, Greenough Iii, & Steinberg, 1969). Furthermore, larger increases in FFA infusion resulted in correspondingly higher insulin levels. It also appears that elevated FFA is associated with increased insulin secretion in humans (Boden, Chen, Rosner, & Barton, 1995). These observations suggest that in obese, insulin resistant individuals, elevated FFA may stimulate secretion of insulin directly, placing further strain on already overcompensating

β -cells. If this is the case, obesity has a multifaceted effect upon type-2 diabetes; through disruption of glucose disposal as well as dysfunction of insulin secretion.

While there is a considerable amount of research regarding insulin resistance and its relation to body composition and/or aerobic fitness, these studies have been done almost entirely with European/Caucasian people (Cauza et al., 2005; Goodpaster, He, Watkins, & Kelley, 2001; Jacob et al., 1999; Kasa-Vubu, Lee, Rosenthal, Singer, & Halter, 2005; Thamer et al., 2003; Uusitupa et al., 2003). To date it appears that only one study exists on the relationship between IMTG and insulin resistance using a Māori cohort (Stannard, Holdaway, Sachinwalla, & Cunningham, 2007). Unfortunately this cross-sectional study only involved a relatively small cohort of convenience, and thus cannot be confidently generalised to the wider Māori population. Nevertheless, this study showed that body fatness and maximal aerobic capacity (VO_{2max}) expressed per kg lean body mass, were both strong predictors of fasting insulin concentration in apparently healthy, young Māori men.

In addition, most studies which have identified the relationship between adiposity and insulin sensitivity, have used less-direct methods to assess adiposity, such as BMI or skin-fold thickness (Bogardus, Lillioja, Mott, Hollenbeck, & Reaven, 1985). Surprisingly few have utilized hydrodensitometry; the 'gold standard' method of body composition assessment. On the other hand, body fat distribution, namely central adiposity, appears to be more closely associated to insulin resistance and other obesity related illnesses than total fat *per se* (Blair, Habicht, Sims, Sylwester, & Abraham, 1984). Following on from this, visceral fat appears to be more closely associated with insulin resistance than subcutaneous fat, fat in the extremities and lower body fat (Abate, Garg, Peshock, Stray-Gundersen, & Grundy, 1995; Björntorp, 1991; Ross, Aru, Freeman, Hudson, & Janssen, 2002). Others suggest that subcutaneous

abdominal adiposity alone is more closely associated with insulin sensitivity (Abate, Garg, Peshock, Stray-Gundersen, & Grundy, 1995; Goodpaster, Thaete, Simoneau, & Kelley, 1997). On the other hand, Fabbrini et al. (Fabbrini et al., 2009) reveal that intrahepatic fat is an independent indicator of ‘multi-organ’ insulin resistance and that visceral fat is only associated with insulin sensitivity because of the association between visceral fat and intrahepatic fat. Indeed, it is suggested that the anatomical location of visceral fat in the abdomen allows easy transport of FFA to the liver (Bergman et al., 2006). Regardless of whether visceral/abdominal fat effects insulin sensitivity directly or indirectly, measuring total body fat may actually be less effective than a simple measure of waist circumference. In light of this, one might suppose that the higher risk of obesity-related illness seen in Māori and Pacific Islanders, at a relatively low BMI, may be due to a genetic predisposition to harmful fat storage. However, the literature suggests that this is not the case (E. Rush et al., 2004; E. C. Rush, Freitas, & Plank, 2009). Nevertheless, waist circumference will be discussed in more detail in section 2.4.2.4.

Given the importance of body composition in the prediction of insulin action, techniques used to assess body composition must be accurate. What’s more, body composition (P. Deurenberg, Yap, & van Staveren, 1998; Luke et al., 1997; Swinburn, Ley, Carmichael, & Palnk, 1999) as well as body fat distribution (Raji, Seely, Arky, & Simonson, 2001) differ between ethnic groups, so it is important that research in this area be ethnicity specific to better understand diabetes risk in those groups at greatest risk of metabolic disorders.

2.4.2 Techniques used to measure body composition

During the last 2 decades, literature regarding anthropometric techniques has become abundant and modern, more complex techniques have been compared to less intricate

techniques of the past. Body composition techniques rely on reference data regarding the composition of the human body. This reference data is based primarily on the chemical analysis of just a few human cadavers. Because only a limited amount of cadavers have actually been analyzed in the past century (Clarys, Martin, & Drinkwater, 1984), assumptions that are used in body composition models are based on reference data that is not necessarily indicative of the population being measured (Heyward & Wagner, 2004).

2.4.2.1 BMI

The body mass index (BMI) was first developed to meet the needs for screening those at high risk of obesity related illnesses such as hypertension, heart disease and diabetes, and is based on a simple measurement of an individual's weight in relation to their height (BMI = weight/height²).

In the early 90's 'The Expert Committee on Clinical Guidelines for Overweight in Adolescent Preventive Services' was established in the United States to develop specific criteria to identify adolescents most at risk of obesity and its co-morbidities (Himes & Dietz, 1994). This committee proposed that a BMI above the 85th percentile, or 25 kg/m², was a representation of overweight, while those above the 95th percentile, or 30kg/m², were considered obese. Three years later the International Obesity Task Force (IOTF) held a workshop in Dublin to explore the strengths and limitations of existing approaches to measure childhood obesity, including the previously mentioned BMI cut-off points (Dietz & Bellizzi, 1999). Although a number of limitations were identified, it was concluded from this workshop that BMI was a reasonable way to assess obesity and overweight in adolescents. Similar BMI standards of 25 kg/m² and 30kg/m², representing overweight and obesity respectively, have been accepted as appropriate cut-off points for health screening in adults (D. Gallagher et al.,

2000). The reference data for these cut-off points is derived from studies showing that BMI is associated with risk of diabetes, cardiovascular disease (CVD) and mortality (Calle, Thun, Petrelli, Rodriguez, & Heath, 1999; I. M. Lee, Manson, Hennekens, & Paffenbarger Jr, 1993; Lohman, 1981b; WHO, 1995). BMI has also been shown to correlate well with body fatness (Paul Deurenberg, Weststrate, A., & Seidell, 1991). There are obvious problems that arise from adopting a single set of rules for BMI cut-off points however, as reference data is derived from populations that are not representative of all groups. Therefore, what may be applicable in a predominantly Caucasian group may not be an appropriate measure of health risk in populations without Caucasian heritage.

Because BMI is inexpensive, easy to calculate [$\text{weight (kg)/height}^2 \text{ (m)}$] and non-invasive, it has become a practical and popular tool in the assessment of obesity-related disease risk in populations. However, because it is excess adiposity and not body weight per se that causes obesity related illness (D. Gallagher et al., 2000; Janssen, Katzmarzyk, & Ross, 2004), relying solely on BMI as a measurement of obesity, has its problems (Prentice & Jebb, 2001). Research shows that BMI and its relation to adiposity varies among ethnic groups (P. Deurenberg, Yap, & van Staveren, 1998; Prentice & Jebb, 2001; Seidell, 2000; Swinburn, Ley, Carmichael, & Palnk, 1999), between age groups (D. Gallagher et al., 2000; Mei et al., 2002) and differs with gender (D. Gallagher et al., 1996). These variations lead to the issue of appropriate BMI values that can accurately express overweight and obesity, most commonly known in the literature as BMI cut-off points. For example, when using the currently accepted BMI cut-off points, 7 time Mr. Olympia Arnold Schwarzenegger, who at the apex of his bodybuilding career was branded by the Guinness book of records as “the most perfectly developed man in the history of the world”, would at the same time be considered well in the obese range with a BMI of 33.4. Similarly, world renowned rugby winger Jonah Lomu, who

could run 100 metres in 11 seconds in his prime, would have carried the label of obese, with a BMI of 31. On the other hand, a study by O'Dea et al. (O'Dea, White, & Sinclair, 1988) revealed that Australian Aborigine adults living a traditionally oriented lifestyle all displayed a BMI categorised as 'underweight' by current BMI standards (13.4-19.3 kg/m²), but showed no biochemical evidence of malnutrition or poor health. Although these examples do not represent the body composition of an average NZ man or women, their examples outline a need for specificity in BMI standards.

Much of the recent literature regarding the body mass index deals with the association of BMI to adiposity and how it differs between populations. Because public health strategies rely on accurate representation of health trends within populations, and considering the extensive research that relies on BMI use, it is important that the effectiveness of BMI as a tool in predicting obesity related illness and appropriate cut-off points is re-evaluated. This is especially important for Māori, who are at greater than average risk from obesity-related morbidity and mortality.

BMI & Age

In his studies of ageing and its effect on body composition, S. H. Cohn showed that a typical healthy male will have an increase of body fat from 15% to 29% from the age of 25 to 70, indicating an increase in the ratio of fat to lean body mass as ageing occurs (Cohn, 1987(citation)). Accordingly, a review by Serdula et al. showed that obese children were at least twice as likely to become obese adults when compared with non-obese children, and that the degree of obesity in childhood increased the risk of adult obesity (Serdula et al., 1993). BMI has also been shown to increase with age, but BMI as a representation of body fat, underestimates the magnitude of the increase of adipose tissue associated with ageing, and

therefore fails to detect the age-associated decrease of lean mass (Prentice & Jebb, 2001). What's more, the validity of employing BMI to determine obesity in children has been questioned (Paul Deurenberg, Weststrate, A., & Seidell, 1991). These findings highlight the need for age-specific BMI cut-off points. In fact, a publication by Deurenberg et al. outlines the effectiveness of age-specific formulas, showing that with such formulas BMI becomes an accurate predictor of body fat stores with a prediction error similar to skin-fold thickness and bioelectrical impedance measurements (Paul Deurenberg, Weststrate, A., & Seidell, 1991).

BMI & Ethnicity

Most ethnic comparative studies of body composition show differences in the relationships between BMI, percentage body fat, and health risks between ethnic groups (P. Deurenberg, Yap, & van Staveren, 1998; Luke et al., 1997; Prentice & Jebb, 2001), while some have shown no difference (D. Gallagher et al., 1996). The international cut-off points set by the WHO in 1993 (WHO, 1995) come from definitions of overweight and obesity derived mainly from data collected by Lohman et al. (Lohman, 1981a). More recently researchers have found that some ethnic groups such as Asians and Indians have a higher percentage of body fat than Caucasians when controlled for BMI, age and sex (WHO, 2004). Polynesians and African Americans on the other hand, are characterised by a lower body fat percentage than Caucasians (P. Deurenberg, Yap, & van Staveren, 1998; Swinburn, Ley, Carmichael, & Palnk, 1999) because of higher proportions of fat-free mass (Swinburn, Ley, Carmichael, & Palnk, 1999). Prentice and Jebb calculate that in order to have the same body fat proportion as a Caucasian, a Polynesian would need to have a BMI 5kgm^{-2} higher than a Caucasian of the same age and gender (Prentice & Jebb, 2001). In Asians and Indians however, the risk of obesity related illnesses such as diabetes seem to increase rapidly at a BMI that falls in the acceptable range for Europeans (Kosaka, Kuzuya, Yoshinaga, & Hagura, 1996; Prentice &

Jebb, 2001). Because it is excess adipose tissue and not excess weight that causes obesity related health risk (P. Deurenberg, Yap, & van Staveren, 1998), BMI scores that are unadjusted for ethnic differences may lead to an inaccurate diagnosis of risk. The implication is that those with relatively lower BMI may be at risk of obesity related illness at a BMI lower than the cut-off, and therefore are ignored in the screening process as they fall within a healthy range. Those with a high BMI due to greater fat-free mass, such as Polynesians and African Americans, would be wrongly categorized as obese. BMI cut-off points representing overweight and obesity should therefore be population specific (P. Deurenberg, Yap, & van Staveren, 1998).

To complicate this issue, differences in the BMI/body fat relationship have been observed between populations with the same or similar ethnic background. Luke et al. (Luke et al., 1997) studied the relationship between BMI and body fat in three different black populations from Nigeria, Jamaica and the US, who all shared a common West African ancestry. They found that at any given BMI, the percentage of body fat differed significantly between the three populations. For example, males at a BMI of 25 had a body fat percentage of 25.8% for the US sample, while Jamaicans had 22.2% and Nigerian men just 16.4%. Deurenberg et al. also observed differences between the Americans and Europeans within a 'white' cohort of their study (P. Deurenberg, Yap, & van Staveren, 1998). These findings indicate that environmental factors along with ethnic make-up may influence the relationship between BMI and body fat percentage. More importantly, the difference in obesity-related disease risk between these genetically close groups, that are geographically disparate, requires investigation to identify important environmental factors. Luke et al. suggests that differences in energy intake and expenditure (the availability of food and the amount of physical activity)

between these populations are the main environmental factors influencing these differences (Luke et al., 1997).

BMI & body composition

BMI is one of a number of ways to assess obesity, and studies of its effectiveness compared to other methods of measurement are well reported in current literature. BMI alone does not measure body composition or the amount of adipose tissue (Prentice & Jebb, 2001). Body composition is also the reason for the obvious differences in the BMI/body fat relationship between populations. In those that are highly muscular, whether from resistance training or a natural disposition, BMI gives an overestimation of body fat (Prentice & Jebb, 2001). Some populations such as Polynesians also appear to have a higher bone mineral density (Swinburn, Ley, Carmichael, & Palnk, 1999), while the body fat of populations with naturally small frames is underestimated by BMI because of a lower level of muscle mass (P. Deurenberg, Yap, & van Staveren, 1998; Swinburn, Ley, Carmichael, & Palnk, 1999).

2.4.2.2 Underwater weighing (Hydrodensitometry)

Underwater weighing, also known as hydrostatic weighing or hydrodensitometry, has been accepted as an effective method of measuring body composition (Heyward & Wagner, 2004; Nord & Payne, 1995; van der Ploeg, Gunn, Withers, Modra, & Crockett, 2000). This method uses Archimedes principle to calculate body density from values of an individual's mass in air and their mass underwater, taking into account the water temperature (van der Ploeg, Gunn, Withers, Modra, & Crockett, 2000). Good practice also measures the volume of gas in the respiratory system while an individual is fully submerged in water (known as residual lung volume), because this alters buoyancy. Values from assumed density of fat and fat-free mass are then used to estimate body fat percentage according to algorithms based on cadaver

studies of body density (Brozek, Grande, Anderson, & Keys, 1963; Norton, 1996; Siri, 1961). Because underwater weighing is the benchmark that other methods of body composition assessment have been compared to, it is difficult to verify the validity of underwater weighing. What's more, more modern techniques of body composition appear to exceed hydrodensitometry in its precision of body composition assessment (Heymsfield, Wang, Heshka, Kehayias, & Pierson, 1989) (See section 2.4.2.6).

The main problem with this method is the need for accurate measures of residual lung volume which can be difficult to obtain and uncomfortable for participants (Wilmore, 1969; Wilmore, Vodak, Parr, Girandola, & Billing, 1980). Calculation of body fat percentage from individuals, whose bone density or total body hydration deviates from the standard by assumption of the four compartment model, will also result in errors (Nord & Payne, 1995) because of the relatively small cadaver-based data sets from which the density equations are drawn.

Underwater weighing can be very difficult to perform, for both the participant being measured and for those administering the test. It requires a pool or tub big enough to fully submerge a person, comfortably warm water, and easy access in and out of the pool. This is difficult for old, large and/or physically incapable persons. Furthermore, it can be quite time consuming, because a number of measures are needed, as well as calibration, so it is not practical for use in population studies or in clinical assessment.

2.4.2.3 Bioelectrical Impedance

Bioelectrical impedance (BIA) machines have become a popular tool for measuring body composition. BIA is quick, non-invasive and recent models are simple to use. BIA does not

measure body fat directly, but measures total body water and segmental water content through the impedance of tissues when a small electrical current is passed through the body. Based on the impedance of this current and certain assumptions regarding water content in FFM (~73% water), body fat and fat-free mass can then be estimated using knowledge from prior experiments associating impedance with body composition (Lukaski, 1987; National Institutes of Health Technology, 1996). Fat-free mass contains a greater proportion of electrolytes and therefore conducts electricity better than fat which has a relatively low water content (Khaled et al., 1988). This implies that the impedance of the electrical current as it passes through the body is related to the amount of fat it has to pass through. This method relies on calculations involving the length and cross sectional area of the body measured (Ursula G. Kyle et al., 2004). There are obvious problems with this however as the length and cross sectional area of the human body is much more complex than a basic cylinder.

Since BIA produces data quickly and is relatively inexpensive, it is a practical tool for obtaining body composition data in clinical screening and population research. The reliability of BIA is a major concern however. Individuals measured with BIA have recorded variations in body fat percentage of up to 10% because of differences in machines and the methodologies used by manufacturers (National Institutes of Health Technology, 1996). It also seems that like BMI, BIA is limited by its need for age, gender and ethnicity-specific equations due to the differences between populations (National Institutes of Health Technology, 1996; Swinburn, Ley, Carmichael, & Palnk, 1999). It is important that the most appropriate prediction equation is used for each individual.

The body of literature regarding bioelectrical impedance is massive, and most of the research is focused on developing the BIA methods and equations in order to produce more valid

measurements of body composition, when compared to more direct measurements such as hydrodensitometry and DEXA. While the skill of the technician taking the BIA measurement is not a major source of error, there are many other possible sources of error when using the BIA method. In the late 80's, Caton et al. found that body composition measurements obtained from BIA were significantly affected by changes in skin temperature when ambient air temperature was changed (Caton, MolÉ, Adams, & Heustis, 1988). In warmer conditions, fat mass was significantly lower than in the cooler conditions. Another study 10 years later also repeated this observation and found that skin hydration (similar to sweating) also affected impedance (Cornish, Thomas, & Ward, 1998). These findings highlight the importance of standardized environmental conditions when performing BIA measures.

Because BIA is a measure of electrical impedance of body tissues, variation in bone and muscular densities between individuals can affect the validity of BIA. In fact, BIA was found to overestimate fatness in lean males and underestimate it in overweight subjects, when compared with hydrostatic weighing (Heath, Adams, Daines, & Hunt, 1998; Segal, Gutin, Presta, Wang, & Van Itallie, 1985). What's more, not only do Māori and Polynesians have a greater muscle mass when compared with Europeans of the same BMI, but their bone density is also greater (Reid, Mackie, & Ibbertson, 1986; Swinburn, Ley, Carmichael, & Palnk, 1999). Durenberg et al. also showed that differences in body build, including arm and leg length in proportion to the trunk, was a major cause of the errors in BIA results between ethnic groups (P. Deurenberg, Deurenberg-Yap, & Schouten, 2002). They also showed however, that when the differences in body build were accounted for in the BIA equations relating impedance to body fatness, the error disappeared. Though there are many studies on the different confounders that effect BIA measurements, only a few studies exist regarding the affect of

ethnicity on the validity of BIA methods. Therefore, appropriate equations are needed for Māori, as well as other ethnic groups.

Theoretically, over hydration should give relatively higher electrical resistance measurements, while dehydration will give relatively lower resistance levels (Khaled et al., 1988). A review by Kyle et al. (U. G. Kyle et al., 2004) revealed that BIA results are affected most by whether subjects were in the fasted or fed state. For example Gallagher et al. (M. R. Gallagher, Walker, & O'Dea, 1998) showed that measuring BIA 3-5hrs post-consumption of food significantly underestimated body fat when compared to fasting measurements. Kushner et al. (Kushner, Gudivaka, & Schoeller, 1996) showed similar effects of eating, but also reported that the effect of eating was not apparent until one hour after food consumption.

Thus, for the most accurate and reliable results, development of universally standardized protocols are needed for BIA (U. G. Kyle et al., 2004). This means that BIA measurements should be taken at the same time of the day, in a fasted state, with controls to reduce the effects of other confounders such as hydration status and exercise.

2.4.2.4 Waist circumference

Measurement of abdominal adiposity is a good, independent predictor of chronic diseases such as CVD and diabetes (Bell, McAuley, Mann, Murphy, & Williams, 2004; Lean, Han, & Seidell, 1998; Zhu et al., 2002). In fact, measurement of waist circumference (WC), is a better predictor of obesity-related illness than BMI alone (Janssen, Katzmarzyk, & Ross, 2004; Zhu et al., 2002). In a study of over 182,000 people from 63 different countries, Balkau et al. showed that WC was a stronger predictor of CVD and diabetes than BMI (Balkau et al., 2007). What's more, body fat distribution i.e. a greater proportion of fat in the abdominal

area, is a more important indicator of cardio-vascular (Blair, Habicht, Sims, Sylwester, & Abraham, 1984) and metabolic health (Ross, Aru, Freeman, Hudson, & Janssen, 2002), than total body fat per se. Because of the close association between central adiposity and insulin resistance, WC could become a practical method of assessing risk of insulin resistance and diabetes. .

Body fat distribution and the relationship between abdominal adiposity and obesity-related co-morbidities differ with ethnicity. One study by Raji et al. showed that Asian Indians had greater abdominal fat when compared to Caucasians of a similar BMI (Raji, Seely, Arky, & Simonson, 2001). Accordingly, at a comparable BMI and age, the Asian Indian group also demonstrated an increase of markers associated with insulin resistance and CVD. Similarly, Okosun et al. reported that the value of WC as a predictor of diabetes and CVD, differed between ethnicities in a cohort of American Caucasian, African American and Mexican American people (Okosun, Liao, Rotimi, Choi, & Cooper, 2000). Because of these differences, ethnic specific criteria is required in the use of WC as a measure of adiposity (Misra, Wasir, & Vikram, 2005; Okosun, Tedders, Choi, & Dever, 2000). Nevertheless, regardless of the differences in the relationship between WC and health risk, WC has still proven to be a reliable predictor of obesity-related co-morbidities in many high risk ethnic groups, including Mexican Americans (M. Wei, Gaskill, Haffner, & Stern, 1997), African Americans (Okosun, Cooper, Rotimi, Osotimehin, & Forrester, 1998) and Māori (Bell, McAuley, Mann, Murphy, & Williams, 2004).

2.4.2.5 Skinfold thickness

Another way of measuring excess adiposity is through skin-fold measurements which measure thickness of subcutaneous adipose tissue from various sites on the body. The sum of

the skin-folds taken from the sites mentioned above may then be used on their own or put into an equation to estimate fat stores, based on reference data (usually obtained from hydrodensitometry). There are over a hundred such population-specific equations in use, but generalized equations have been developed also (Heyward & Wagner, 2004). This type of measurement is inexpensive and easily measured, though skill is required for reliability (Heyward & Wagner, 2004). Measuring skin-fold thickness is invasive however, and concerns about the reliability of this method arise because skin-fold thickness measurements are often only taken from a few body sites (Lohman, 1981b(Citation); Mei et al., 2002). Intertechnician reliability is also a cause for error as improper location and measurement of skin-fold sites between technicians, as well as choice of an appropriate equation, can cause significant differences in %BF interpretation (Lohman, Pollock, Slaughter, Brandon, & Boileau, 1984) The use of reporting body fatness from skin-fold measurements also has questionable validity since it completely ignores variations in the subcutaneous/visceral fat relationships which exist (Norton, 1996).

2.4.2.6 Dual energy x-ray absorptiometry

A more sophisticated way of measuring body composition is through dual energy x-ray absorptiometry (DEXA or DXA). This method distinguishes, then quantifies fat mass, lean mass and total-body bone mineral, through the use of two x-rays of differing energy levels (Mazess, Barden, Bisek, & Hanson, 1990; Svendsen, Haarbo, Hassager, & Christiansen, 1993). It does this by measuring the different attenuation by tissues (muscle, fat, bone etc.) of emitted photons at two energy levels; these measurements are then used to calculate proportions of different body tissues (Heymsfield, Wang, Heshka, Kehayias, & Pierson, 1989). DXA has proven to be a very valuable method of measuring body composition as it is able to differentiate between fat and fat-free mass, in regions as well as total body (Mei et al.,

2002; Svendsen, Haarbo, Hassager, & Christiansen, 1993). In fact, some results would suggest that DXA is more precise than hydrodensitometry (Heymsfield, Wang, Heshka, Kehayias, & Pierson, 1989), highlighting its use as a method of measuring small changes in body composition. However, the use of DXA as a measure of body composition in clinical screening at a population level is not practical, as it is time consuming and expensive, limiting its use primarily to research settings (Prentice & Jebb, 2001).

2.4.2.7 Māori and body composition

BMI has been adopted by clinicians as a practical way of determining body composition in New Zealand. As previously mentioned however, the problem with relying solely on BMI as a measure of obesity in this priority group is that Māori and Pacific people have a different BMI/body fat ratio to NZEO. With this in mind, Swinburn et al. has recommended a BMI of 26-32 as overweight and 32 as obese in Māori and Polynesians, for clinical and epidemiological purposes (Swinburn, Ley, Carmichael, & Plank, 1999). At the standard BMI cut-off for overweight (25kg/m^2) Polynesians have more fat-free mass and less fat than Caucasians (Prentice & Jebb, 2001; Swinburn, Ley, Carmichael, & Plank, 1999), suggesting the need for Māori/Polynesian-specific equations. Other studies suggest that BMI is not an adequate measure of fatness in a mixed European, Māori and Pacific population and other methods of assessment such as BIA (E. C. Rush, Puniani, Valencia, Davies, & Plank, 2003) or waist circumference (Bell, McAuley, Mann, Murphy, & Williams, 2004) would be more appropriate. On the other hand, it has been argued that cut-off points should reflect the risk of co-morbidities associated with a given BMI, rather than an individual's lean body mass (K. McAuley, Williams, Mann, Goulding, & Murphy, 2002). In their study on a group of Māori and non-Māori women, McAuley et al. showed that Māori women were more insulin resistant than Europeans even when BMI levels were similar.

2.4.3 Summary

Increased body fat is identified as a major cause of insulin resistance. Fat is an important substrate during exercise, although in sedentary individuals excess fat from energy rich meals is not used and fat is stored in adipose tissue and muscle cells. A high concentration of IMTG is associated with diabetes, but can also be seen in healthy endurance trained individuals. This suggests that the turnover of intramuscular lipids, rather than the concentration per se, affects the insulin signalling pathway. Because of body fat's relationship to insulin resistance, obtaining accurate measures of body composition is important in predicting many health risks. Of the methods of measuring or estimating body fatness discussed above, only DEXA and underwater weighing have been shown to be reliable and accurate measures. However, these are techniques which require expensive equipment and trained technicians to obtain the measurements. Simple and quick means of accurately measuring body composition must be sought if large scale studies are to become more valid.

2.5 Exercise and Diabetes

2.5.1 Fuel selection during endurance and high intensity exercise

The increasing energy requirement during exercise, due to increased skeletal muscle metabolism, is predominantly fuelled by oxidation of lipids and carbohydrate (CHO). The total fuel requirement is determined by the absolute work rate and duration of exercise, while the proportion of fuel used, i.e. the ratio of fat to CHO, is determined by the relative intensity of the exercise for a particular duration (Brooks & Mercier, 1994). Generally speaking, during low intensity exercise, particularly as duration increases, oxidation of fat provides most of the energy for contraction, although CHO still contributes in part. At the onset of exercise, and particularly at higher intensities, glycolytic flux predominates and fatty acid oxidation plays a more minor role (Hawley, 2001).

Circulating glucose is an important substrate for contracting muscle. It has been repeatedly shown that if circulating glucose concentrations can be supported or even increased in the latter stages of prolonged exercise, then muscle performance similarly is maintained (Coggan & Coyle, 1987; Coyle et al., 1983). The rate of blood glucose utilization by contracting muscle, is controlled by various factors including glycogen status (Fell, Terblanche, Ivy, Young, & Holloszy, 1982), lipid availability (Ferrannini, Barrett, Bevilacqua, & DeFronzo, 1983), muscle temperature (Febbraio, Snow, Stathis, Hargreaves, & Carey, 1994), exercise duration (Ahlborg, Felig, Hagenfeldt, Hendler, & Wahren, 1974), and exercise intensity (Hawley, 2001). Glucose uptake into muscle fibre increases as a function of contraction (Ploug, Galbo, & Richter, 1984), whilst an exercise-induced increase in blood flow increases substrate availability (Goodyear & Kahn, 1998). In recovery (at rest), glucose uptake increases to replenish muscle glycogen stores, but some continuing oxidation takes place. At

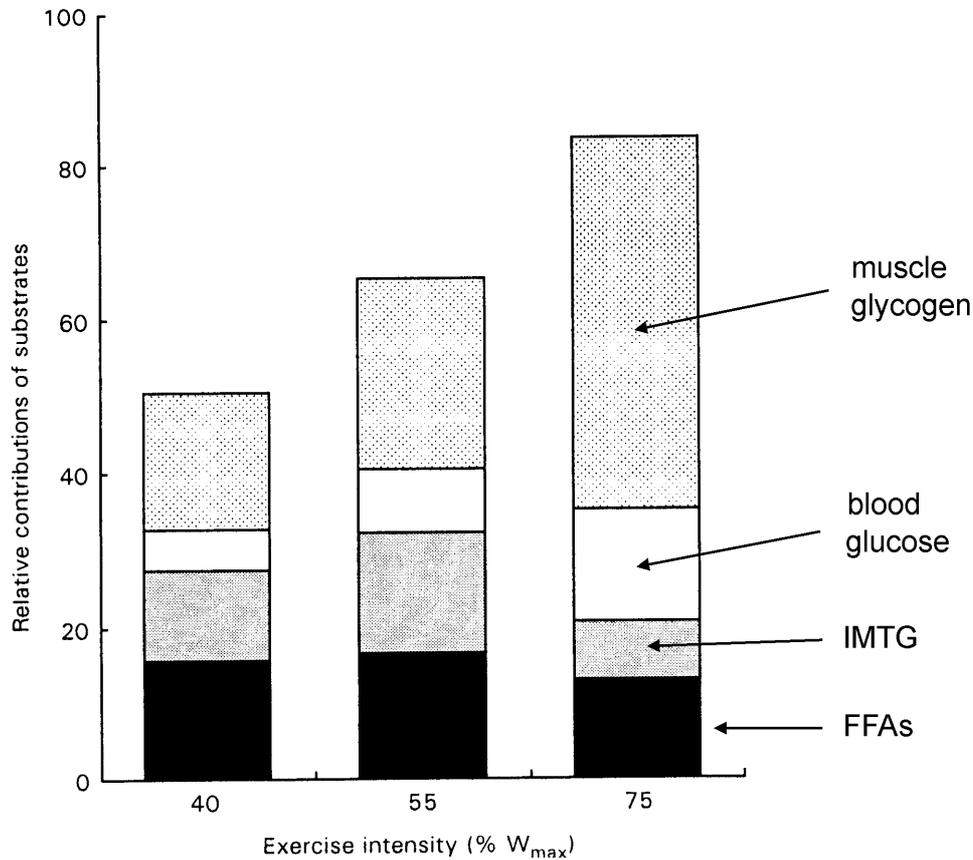
rest, following recovery and in the fed or postprandial state, it is not usually availability of glucose during exercise that limits oxidation of glucose, but the process of glucose transport into the cell. As touched upon earlier, GLUT4, the main transport protein responsible for glucose transport, is not stimulated to the cell surface (to enable glucose uptake) by insulin secretion alone but GLUT4 translocation is also independently stimulated by contraction (Ploug, Galbo, & Richter, 1984). For these reasons, exercise and adequate physical activity are important in maintaining glucose transport and preventing type-2 diabetes (J. Eriksson, Taimela, & Koivisto, 1997; Ivy, 1997).

2.5.2 Effects of an acute exercise bout on glucose transport

A single bout of exercise improves insulin-mediated glucose disposal in insulin resistant, diabetic and even insulin sensitive subjects (J. Eriksson, Taimela, & Koivisto, 1997; E. Henriksen, 2002). The insulin sensitive response to acute exercise vanishes within a few days however, emphasizing the need for chronic exercise (J. Eriksson, Taimela, & Koivisto, 1997; Richter, Garetto, Goodman, & Ruderman, 1982).

A single bout of exercise also reduces body fat, albeit in small amounts, through increased oxidation of intramuscular lipid, and mobilization of fatty acids from adipose tissue. At exercise intensities below 75% of maximal workload, utilization of fat as a substrate is at its highest (See **Figure 2**). Past this point, the relative contribution of glycogen and glucose increases, while fat oxidation decreases (van Loon, Greenhaff, Constantin-Teodosiu, Saris, & Wagenmakers, 2001). This is likely the reason why sub-maximal exercise has become the recommended form of exercise for those with insulin resistance or at risk of the disorder.

Figure 2 – Relative contribution of substrate utilized at different exercise intensities (L.J.C van Loon, 2004)



However, because GLUT4 translocation responds to muscle contraction independent of insulin sensitivity, other types of exercise, such as resistance training, have proven effective in improving glucose tolerance through greater expression and increased translocation of GLUT4 transport proteins (Holten et al., 2004). Host et al. (Host, Hansen, Nolte, Chen, & Holloszy, 1998) found that the increase of GLUT4 seen after training in animals, reversed after just 2 days of detraining. Thus, in order for exercise to be effective in maintaining glucose tolerance, exercise must be performed regularly. Interestingly, the period just after exercise is also associated with increased insulin sensitivity within muscle cells (Richter,

Mikines, Galbo, & Kiens, 1989), which re-enforces the potential benefits of exercise in improving and maintaining insulin sensitivity.

2.5.3 Effects of Chronic exercise on glucose transport

While the relationship between physical activity and insulin resistance is not entirely understood, many studies have showed that progression from a glucose intolerant state to diabetes, is prevented by physical exercise (K. F. Eriksson & Lindgärde, 1991; Lindstrom et al., 2003; X. R. Pan et al., 1997; Tuomilehto et al., 2001). In some cases, exercise and a healthy diet have even led to remission of diabetes (K. F. Eriksson & Lindgärde, 1991). Furthermore, type-2 diabetics who are physically fit live longer than diabetics who are unfit (Ming Wei, Gibbons, Kampert, Nichaman, & Blair, 2000). Physical activity not only reduces adiposity, but improves insulin sensitivity independent of weight loss (Duncan et al., 2003; Katzmarzyk, Church, Janssen, Ross, & Blair, 2005; S. Lee et al., 2005; S. J. Lee et al., 2005), suggesting that diabetes develops not just because of obesity, but from physical inactivity. This leads to the idea that an overweight person who is physically fit is protected from insulin resistance, while an unfit lean person may be at higher risk of insulin resistance and diabetes.

Aerobic fitness is largely a function of the oxidative capacity of myocytes; the ability of muscle to oxidize substrate. Although there are many mechanisms underlying the increase in physical fitness that results from training, classic studies by Holloszy (J. O. Holloszy, 1967), as well as Gollnick and King (Cited in J O Holloszy & Coyle, 1984), revealed that exercise training increased mitochondrial content, density and function within muscle fibres of rats. Later, studies by Hoppeler et al. and Morgan et al. (Cited in J O Holloszy & Coyle, 1984) confirmed these findings in humans. Conversely, studies have shown that mitochondrial density and content are reduced in obese (Toledo, Watkins, & Kelley, 2006) and type-2 diabetic patients (D. E. Kelley, He, Menshikova, & Ritov, 2002). However, exercise increases

mitochondrial function and content in these groups as well (Toledo et al., 2007; Toledo, Watkins, & Kelley, 2006). These findings highlight the role of mitochondrial content and density as determinants of aerobic fitness, and their importance in regulating metabolic health. What's more, oxidative capacity also mediates the rate of lipid oxidation which is associated with an individual's ability to maintain or lose weight (David E. Kelley, Goodpaster, Wing, & Simoneau, 1999).

Interestingly, ageing is also associated with a diminished oxidative capacity (Conley, Jubrias, & Esselman, 2000; Papa, 1996) and insulin sensitivity (Fink, Kolterman, Griffin, & Olefsky, 1983). On the other hand, Rimbart et al. showed that aerobic capacity is not diminished with age, but is associated with the physical inactivity that accompanies aging (Rimbart et al., 2004). This observation implies that a reduction of physical activity in later years, rather than age itself, as the name 'adult-onset diabetes' suggests, explains in part, the association between age and insulin resistance.

2.5.4 Aerobic Capacity, Oxidative Capacity and Insulin Resistance

Chronic endurance exercise also increases the oxidative capacity of skeletal muscle and therefore its ability to use fat as a substrate. Aerobic fitness is also closely associated with insulin sensitivity. For example, oxidative enzyme activity is closely related to insulin sensitivity (Rimbart et al., 2004; J.-A. Simoneau & Kelley, 1997). Some even suggest that oxidative capacity is a better predictor of insulin resistance than adiposity (C. R. Bruce et al., 2003). Although measuring oxidative enzyme activity is a relatively accurate representation of a muscle's aerobic fitness, the problem with this method is that invasive muscle biopsies are required. A test of maximal oxygen consumption (VO_{2max}) on the other hand, is less invasive and the only discomfort felt is the discomfort associated with intense exercise.

However, maximal aerobic capacity is probably limited by O₂ delivery to the working muscle, rather than O₂ utilization (González-Alonso & Calbet, 2003). Nevertheless, a high VO₂max requires a good oxidative capacity (Bassett Jr & Howley, 2000).

Literature observing the relationship between insulin sensitivity and VO₂max is limited, though one study found that VO₂max was a better predictor of insulin resistance than BMI or even adiposity in a group of post-pubertal adolescent females (Kasa-Vubu, Lee, Rosenthal, Singer, & Halter, 2005). Likewise, Stannard et al. revealed a similar relationship between VO₂max and insulin sensitivity in a cohort of healthy Māori men (Stannard, Holdaway, Sachinwalla, & Cunningham, 2007).

There are problems using maximal oxygen consumption as a measure of aerobic fitness however. When used in research, VO₂max is most often expressed relative to total body weight (ml.kg *total body mass*⁻¹.min⁻¹), which includes fat mass. Because physical fitness is a risk factor independent of fatness, and because skeletal muscle (and not adipose tissue) is responsible for the increase in oxygen consumption during exercise, it seems more sensible to express fitness in terms of lean body mass (VO₂maxLBM) according to the methodology of two previous studies i.e. (ml.kg *lean body mass*⁻¹.min⁻¹) (Goodpaster, Wolfe, & Kelley, 2002; Stannard, Holdaway, Sachinwalla, & Cunningham, 2007).

2.5.5 Endurance training & IMTG accumulation: A paradox

As discussed earlier, resting IMTG levels are elevated in sedentary obese individuals and those with insulin resistance. A paradox exists regarding this association however as endurance trained athletes also have elevated levels of IMTG even though their insulin sensitivity is high (Hoppeler et al., 1985; Morgan, Short, & Cobb, 1969). Schrauwen-

Hinderling et al. (Schrauwen-Hinderling et al., 2003) observed that IMTG content increased in untrained healthy males after just 2 weeks of endurance training, but that this increase was not associated with a reduction of insulin sensitivity. The authors suggest that 1) increased IMTG accumulation is amongst the earliest responses to training, 2) The presence of IMTG alone does not have detrimental effects on insulin sensitivity, and 3) that IMTG contributes to the fuel used during prolonged exercise. This is further supported by Goodpaster et al. (Goodpaster, He, Watkins, & Kelley, 2001) who showed that in sedentary individuals, IMTG content was inversely associated with insulin sensitivity, while endurance-trained subjects were insulin sensitive despite having higher IMTG content than the sedentary group. Thamer et al. (Thamer et al., 2003) further suggest that the correlation between IMTG and insulin resistance is modified by the extent of aerobic fitness, and that interpretation of IMTG measurements and how they relate to insulin resistance must include measurements of aerobic fitness.

The difference between the obese and endurance trained individual is that the endurance trained person has muscle with a higher fat oxidation capacity and is frequently involved in exercise, both of which are conditions enabling turnover of IMTG. Stannard and Johnson suggest that in a context of exercise, the ability to store large amounts of IMTG aids in increasing the availability of lipid as a substrate during prolonged bouts of exercise, or at the onset of exercise when alternative intramuscular substrate, glycogen, has not been replenished (Stannard & Johnson, 2004).

In contrast, the skeletal muscle of sedentary individuals does not have a well developed capacity for fat oxidation, and is usually not involved in regular acute exercise bouts. Furthermore, even when they do exercise, fat oxidation is lower than in non-obese individuals

for the same relative intensities (Blaak, 2007). Blaak et al. (Blaak et al., 2000) showed that 50% of fatty acids taken up by non-diabetic muscle were directly oxidised during β -adrenergic stimulation, but in diabetic muscle, which had impaired FFA uptake, no oxidation was detected. In addition, Goodpaster et al. (Goodpaster, Katsiaras, & Kelley, 2003) observed that in obese individuals, improvement in insulin sensitivity resulting from a combined diet and exercise intervention, was associated with an increased reliance on fat oxidation during fasted conditions. Thus, it is believed that an imbalance between uptake and oxidation may lead to accumulation of lipid intermediates in skeletal muscle, thereby affecting the insulin signalling pathway and leading to insulin resistance (Blaak, 2007; Moro, Bajpeyi, & Smith, 2008). In summary, the 'paradox' surrounding IMTG and how increased storage of IMTG leads to insulin resistance in obese individuals but not the endurance trained, suggests it is the oxidation of lipid, and therefore aerobic fitness and participation in regular exercise bouts, that dictate insulin sensitivity (Goodpaster, He, Watkins, & Kelley, 2001).

2.5.6 Exercise physiology research with Māori

To the authors knowledge, only one study exists which investigates the relationship between aerobic capacity and insulin sensitivity in Māori (Stannard, Holdaway, Sachinwalla, & Cunningham, 2007). This study showed that the aerobic capacity, or $VO_{2maxLBM}$ ($mL.kg^{-1} LBM.min^{-1}$) in Māori men, was able to help predict insulin sensitivity as measured by HOMA-IR. Only one other published study has made the comparison between insulin sensitivity and aerobic fitness, using $VO_{2maxLBM}$. This study was done in a cohort of Caucasian men, and no observed relationship between $VO_{2maxLBM}$ and insulin sensitivity in this cohort was apparent (Coon, Bleecker, Drinkwater, Meyers, & Goldberg, 1989).

2.5.7 Summary

A single exercise bout stimulates uptake of glucose and FFA, and if the exercise is of a sufficient duration, the turnover of IMTG. This reduction in intramuscular lipid lowers the risk of insulin resistance and increases insulin sensitivity. Muscle contraction also increases expression of glucose transporters and their translocation, improving glucose tolerance, independent of insulin action. Because of this, the demand on the pancreas and β -cells to secrete insulin is reduced with exercise, further reducing the risk of diabetes and β -cell failure. Chronic exercise improves the muscle cells ability to maintain healthy metabolic function (flexibility), by increasing mitochondria content, which in turn improves oxidative capacity and enhances skeletal muscle's ability to utilize lipid as a substrate (J. O. Holloszy & Booth, 1976). A higher aerobic capacity has also been shown to independently predict insulin sensitivity in Māori (Stannard, Holdaway, Sachinwalla, & Cunningham, 2007). Thus, regular and consistent exercise is likely an important means of maintaining glucose disposal in a healthy state, and reducing hyperglycaemia in the glucose intolerant.

2.6 Muscle Fibre type and Diabetes

2.6.1 A history of fibre type and how it's measured

In the late 19th century, French anatomist Louis Antoine Ranvier observed that some muscle in rabbits differed in colour and that the redder muscle contracted at a slower rate than the muscle with a more pale coloration (Needham, 1926). From this simple observation, the basis of skeletal muscle fibre type study was formed and the understanding that muscle was a heterogeneous tissue was established. Just shortly after Ranvier's experiments, Grutzner (cited in Needham, 1926) proposed that all muscle contained a mixture of two different fibres (red and white) and that the response of a muscle to stimulation (whether fast or slow) depended on the proportion of these two fibres within the muscle. These early in-vivo studies were done with animals, until the advancement of surgical techniques made it possible to take samples of human muscle by biopsy (Zierath & Hawley, 2004). In the 1960's, the needle biopsy technique was re-introduced to the field of physiology (Bergström & Hultman, 1966), enabling physiologists to obtain human muscle tissue while subjects were awake. Using this procedure, samples of human muscle became more readily available, allowing further development in the study of fibre-type.

In the late 1960's and early 1970's, studies by Edstrom and Kugelberg (Edström & Kugelberg, 1968), as well as Burke et al. (Burke, Levine, Zajac Iii, Tsairis, & Engel, 1971) showed a relationship between physiological parameters and histochemistry within motor units. These studies paved the way for histological and biochemical analysis of muscle samples, leading to current classifications based on histochemical, biochemical, morphological or physiological characteristics (Scott, Stevens, & Binder-Macleod, 2001).

Since these early studies, many techniques have been developed to classify fibre type, making the interpretation of fibre type data more difficult, as classifications from different techniques do not always agree (Staron, 1997). At present, the most common way of identifying muscle fibres in humans is by dividing them into different categories according to the myosin heavy chain (MHC) isoform found within the fibre. Because myosin isoform content in a given muscle is quite stable and relatively unaffected by contraction, it has been identified as a suitable basis for fibre-type classification (Astrand, Rodahl, Dahl, & Stromme, 2003). Furthermore, measurement of MHC isoforms is probably the best method of fibre typing currently available, because MHC isoforms are identified within single muscle fibres by electrophoresis, meaning the analysis is quantitative.

Another way muscle fibres are often identified is through histochemical staining to measure ATPase activity. The activity of ATPase, the enzyme responsible for breaking down ATP, is known to be associated with myosin isoform content (Billeter, Heizmann, Howald, & Jenny, 1981) and the speed of muscle shortening (Barany, 1967). When histochemically analyzed, muscle fibres with high ATPase activity are identified as type II muscle fibres, while those fibres with low ATPase activity are identified as type I fibres. Differing pH sensitivities within type II fibres can then be used to separate type II fibres into two subgroups; type IIA and type IIX (also commonly known as IIB) (Brooke & Kaiser, 1970 - citation only; Rosen, 1969). The problem with measuring ATPase activity with histochemical staining is that it is based on qualitative analysis of the different staining intensities, so that any given fibre could be classified differently by different researchers (Scott, Stevens, & Binder-Macleod, 2001).

Presently there are three common fibre type divisions in humans based on MHC content; type I – slow oxidative, type IIA – fast oxidative and type IIX– fast glycolytic. There are

limitations in trying to define muscle fibre types however, as functional properties vary within groups of muscle fibres that have the same fibre type designation (Astrand, Rodahl, Dahl, & Stromme, 2003). This is largely due to the ‘plasticity’ of muscle fibres; their ability to transition from fast to slower fibre types and vice versa, in response to certain stimulus which will be discussed later. Because of this ‘plasticity’, many sub-categories of fibre type, those that are transitioning between ‘pure’ fibre types, and therefore express two MHC isoforms, have been identified within human muscle (Pette, 2001). However, this review will only deal with the 3 most commonly used classifications of fibre type, identified above.

The needle biopsy technique also has many limitations. Within humans, fibre type varies between different muscles as well as within a given muscle (Blomstrand & Ekblom, 1982; J. A. Simoneau, Lortie, Boulay, Thibault, & Bouchard, 2008). Because biopsies are usually taken from only a few sites, this inter and intra-muscular variation is often not accounted for, and muscle samples are falsely considered as representative of the fibre type proportion of the whole muscle (Lexell, Taylor, & Sjostrom, 1985). Blomstrand and Ekblom reported that fibre type percentage varied between two biopsies taken from the same leg by 6% for type I fibres, 4% for type IIA and 5% for type IIX (Blomstrand & Ekblom, 1982). When the samples were taken from both legs this variation increased to 12%, 7% and 7% respectively. Furthermore, it appears that fibre type distribution varies depending on the depth of the muscle sample, with a greater proportion of type II fibres close to the surface and type I fibres in deeper regions of the muscle (Lexell, Henriksson-Larsen, & Sjostrom, 1983). The varying distribution of fibre type within whole muscle raises concern regarding the repeatability of obtaining muscle samples by biopsy, and the biopsy technique’s reliability in obtaining muscle samples to identify those with a certain ‘fibre type proportion’. What’s more, the muscle biopsy

technique is invasive and requires the skill of a trained physician with specialized equipment (Bergström, 1975), making it impractical for many research and clinical settings.

In accordance with early observations of different fibre types having different functional properties (Needham, 1926), more recent studies have shown, using non-invasive techniques, that contractile function of muscle, such as time to peak torque and fatigability measured externally, is related to fibre type proportion in-vivo (Ivy, Withers, Brose, Maxwell, & Costill, 1981; MacIntosh, Herzog, Suter, Wiley, & Sokolosky, 1993; Sadoyama, Masuda, Miyata, & Katsuta, 1988; E. Suter, Herzog, Sokolosky, Wiley, & Macintosh, 1993; Thorstensson & Karlsson, 1976). To the author's knowledge, these techniques have not yet been used as a surrogate measure of fibre type proportion in larger studies. Nevertheless, the relationships observed between histo/biochemically determined fibre type and functional characteristics, indicate that methods which measure muscle contractile properties externally, may provide a non-invasive way to estimate fibre type proportion in future studies. Therefore, further development of these techniques is required.

One method of modelling muscle contraction which has been used a great deal in the clinical setting is electrical stimulation (ES). ES enables muscle to contract independent of the central nervous system and is therefore useful for maintenance of strength, muscle mass and physical activity levels during muscle immobilization/paralysis (Delitto & Snyder-Mackler, 1990). This technique is useful in a research setting as the magnitude of muscle contraction can be controlled with ES, and psychological factors such as motivation are bypassed. For example, electrical stimulation has been used to study the role of the nervous system in force production. By administering an electrical impulse to a maximal voluntary contraction, and observing that greater force was produced with the addition of electrical stimulation, early

researchers showed that neural factors limited maximal force production rather than the muscle structure (Shield & Zhou, 2004). The most important factor to consider when administering percutaneous ES is the location of electrode on the skin. Placement of electrode pads should ensure that antagonist muscles are not being stimulated at the same time as agonists as incorrect placement of electrodes can alter force measurements (Shield & Zhou, 2004). Nevertheless, apart from error associated with electrode placement, technical error is small, as the administration, the timing and magnitude of the twitch, is controlled electronically.

Although the ES technique has been used extensively in a research setting as a substitute for voluntary exercise, its use in stimulating muscle to measure contractile properties has hardly been employed. The only published study I know of which has employed ES to measure muscle contractile properties is one by Blimkie et al. (Blimkie, Sale, & Bar-Or, 1990). Thus, although validity and limitations have been discussed in regard to the use of electrical stimulation, these discussions have focused on the validity of ES as a tool for simulating exercise rather than creating twitches for measurement of contractile properties. Therefore, the validity of ES for use in this way, and its limitations, has not been addressed.

2.6.2 Metabolic and functional characteristics of fibre type

A muscle's contractile properties are determined largely by the myosin heavy chain (MHC) protein that is expressed in the muscle fibres within (Bottinelli, Betto, Schiaffino, & Reggiani, 1994). However, it seems that the innervation of muscle fibres may be the primary determinant of MHC, and thus fibre type. A classic study by Buller et al. (Buller, Eccles, & Eccles, 1960) showed that slow-twitch muscles become faster when reinnervated by the motor neuron that originally innervated a fast muscle, and vice versa. This suggests that the

contractile characteristics of a muscle are ultimately the result of the activity of the motor neuron which innervates it and that any changes in structure, phenotype or contractile protein content, are secondary functions in response to the innervation of the fibre.

Enzyme activities also differ between fibre types. Essen et al. found that activity of SDH (succinate dehydrogenase) and HAD (3-hydroxyacyl-CoA-dehydrogenase), important oxidative enzymes, are higher in slow-twitch fibres, while activity of PFK (phosphofructokinase), an important glycolytic enzyme, is higher in fast-twitch fibre (Essén, Jansson, Henriksson, Taylor, & Saltin, 1975). Others have reported a similar relationship between fibre type and glycolytic/oxidative enzyme activity (He, Watkins, & Kelley, 2001). These relationships are not surprising, considering the recruitment of these different fibres during exercise. In accordance with Henneman's 'size principle' (cited in Enoka & Stuart, 1984), slow-twitch fibres, which are part of a relatively small motor unit, are recruited during low-intensity activity and can maintain contraction (albeit at low-intensities) for prolonged periods. Accordingly, slow-twitch fibres require a greater oxidative capacity to provide fuel during prolonged exercise. Fast-twitch fibres on the other hand, are part of a larger motor unit and are therefore recruited during more intense bouts of exercise that require maximal contractions fuelled mainly by the glycolytic system.

It appears that glucose transport is fibre type specific as well. Henriksen et al. showed that expression of GLUT4 transport protein was greatest in the slow-twitch fibres of rats (E. J. Henriksen et al., 1990). Similarly, Dagaard et al. (Dagaard et al., 2000) demonstrated that in humans GLUT4 expression was slightly higher ($P < 0.05$) in slow-twitch than in fast-twitch fibres, both before and after a 2 week training protocol.

Table 2-2 – Metabolic, structural & functional characteristics of different muscle fibre types

(Adapted from Wilmore & Costill, 2005 & Zierath & Hawley, 2004)

Characteristic	Slow-twitch	Fast-twitch (oxidative)	Fast-twitch (glycolytic)
MHC	MHC I	MHC IIA	MHC IIX
Type of myosin ATPase	Slow	Fast	Fast
Time to peak tension	High	Low	Low
Fibres per motor neuron	Few	Many	Many
Motor unit force	Low	High	High
Aerobic capacity (oxidative)	High	Moderate	Low
Anaerobic capacity (glycolytic)	Low	High	High
Mitochondrial density	High	Moderate	Low
GLUT4 Content	High	Medium	Low
Cross sectional area	Small	Medium	Large
Motor neuron size	Small	Large	Large
Nerve conduction velocity	Slow	Fast	Fast
Fatigue resistance	High	Moderate	Low
Insulin sensitivity	High	Medium	Low
Capillary density	High	Moderate	Low

2.6.3 Fibre type and exercise

As can be seen in the table above, type II fibres have a lower oxidative capacity than type I fibres. This is important to note as it is thought that the oxidative capacity of skeletal muscle is related directly to its ability oxidize fat (David E. Kelley, Goodpaster, Wing, & Simoneau, 1999), and therefore take up glucose in response to insulin. If this is the case, an individual with a greater proportion of type II fibres in their muscles would be expected to be at greater

risk of lipid accumulation (obesity) and whole body insulin resistance, than one with a high proportion of type I fibres.

Although exercise is well known to increase insulin sensitivity, Cortez et al. (Cortez, Torgan, Brozinick, & Ivy, 1991) showed that the effect of exercise on insulin sensitivity is only significant within the fibres recruited during the exercise. Their studies on obese Zucker rats revealed that with low-intensity training, glucose transport only improved in fast-twitch red fibres (type IIA). On the other hand, glucose transport in fast-twitch white fibres (type IIX) only improved after high-intensity training. Accordingly, Dugaard et al. (Dugaard et al., 2000) showed that after endurance-type training in humans, GLUT4 expression only increased in slow-twitch fibres. What's more, Koopman et al. (Koopman et al., 2006) found that IMTG only decreased in type-1 muscle fibres after resistance training in untrained healthy males, but that the decrease was closely related to fibre-type specific oxidative capacity. On the other hand, this same study showed that the greatest decrease in glycogen content was in type IIX fibres. These findings all suggest that the increased GLUT4 content and increased glycogen and IMTG turnover associated with exercise training, is more a function of the oxidative capacity and activity level of the muscle fibre, rather than the fibre type per se (Dugaard & Richter, 2001).

As discussed earlier, the recruitment of type II fibres increase as increased spatial recruitment is required, such as occurs during high intensity exercise. Type I fibres are recruited first to provide the majority of tension during endurance type exercise. Hence, the intensity of exercise prescribed to the glucose intolerant individual, should be carefully considered. At present, biopsies are required to obtain a direct measure of fibre type proportion, but this procedure is very invasive and not practical for large studies. Thus, developing a less invasive

way of estimating muscle composition could provide a means of identifying those who are most at risk of obesity and metabolic disorders, and allow prescription of fibre-type specific exercise.

2.6.4 Fibre type and insulin resistance

As previously discussed, insulin-stimulated glucose uptake and metabolism within skeletal muscle is influenced by fibre type (Hickey, Weidner et al., 1995). Accordingly, fibre type proportion has been related to whole body insulin sensitivity and obesity. Lillioja et al. (Lillioja et al., 1987a) found that a higher proportion of type IIX (fast-twitch B) fibres is associated with many risk factors of type-2 diabetes, while insulin sensitivity correlates positively with the percentage of type-1 fibres. Likewise, Wade et al. (Wade, Marbut, & Round, 1990) found an inverse relationship between fatness and proportion of slow-twitch fibres in a group of untrained males. Other studies have also shown increased proportions of type IIX fibres in type-2 diabetics (Hickey, Carey et al., 1995; Marin, Andersson, Krotkiewski, & Bjorntorp, 1994). Tanner et al. (Tanner et al., 2002) showed that morbidly obese women with a higher proportion of type-1 fibre, had a greater capacity for weight loss 12 months after a surgical weight loss intervention, when compared with those with more type-2 fibres. This finding is further supported by a study by Wade et al. (Wade, Marbut, & Round, 1990) who found that untrained men with a greater proportion of slow-twitch fibre, had a greater ability to oxidize fat during exercise, when compared to those with more fast-twitch fibres. Along with the fact that slow-twitch fibres have a greater capillarization and mitochondrial content than fast-twitch fibres (Ingjer, 1979), these findings are consistent with the idea that impaired insulin action in skeletal muscle is a function of an inability to properly oxidize lipids (Goodpaster, Katsiaras, & Kelley, 2003).

It has been reported that the balance between oxidative and glycolytic enzymes in the muscle of obese individuals is related to insulin resistance (J.-A. Simoneau & Kelley, 1997). IMTG content is also strongly related to insulin resistance (Shulman, 2000). Fittingly, He et al. showed that oxidative enzyme activity was reduced in all fibre types of obese and diabetic individuals when compared with lean subjects (He, Watkins, & Kelley, 2001). They also demonstrated that the ratio of oxidative enzyme activity to lipid content within all fibre types was significantly lower in obese and type-2 diabetic individuals. Though type I fibres have a higher concentration of IMTG, this fibre type's greater oxidative capacity and ability to mobilize fatty acids allows for a greater turnover of the accumulated lipid (Blaak, 2007; Moro, Bajpeyi, & Smith, 2008). This suggests furthermore, that the rate of turnover, rather than the concentration of IMTG, determines insulin sensitivity. Considering that fibre-type is related to the ability to utilize fat during exercise, an individual's muscle composition may play a role in determining the effectiveness of exercise interventions in reducing adiposity, and improving insulin sensitivity.

2.6.5 Fibre type proportion: Genetically or environmentally determined?

When observing a variety of sporting and athletic events at the elite level, it is difficult not to notice an ethnic bias towards success in particular events. It is common to see an African-American running the 100m sprint at the Olympics, while Caucasians usually find success in long-distance events (Cited in Suminski, Mattern, & Devor, 2002). On the other hand it is not uncommon to see an African Marathon runner take a gold medal, or a European dominate the power lifting. It is often remarked that some ethnicities are genetically predisposed to excel in different athletic events (Hunter, 1998). Alternatively factors like socio-economic status or exposure to certain sports may also explain these ethnic biases.

From their classic studies which investigated fibre type proportion in monozygous and dizygous twins, Komi et al. (Komi et al., 1977) were the first to provide evidence that fibre type proportion is mostly determined by genetic influences. In accordance with these findings, Ama et al. showed that a cohort of Black men had a lower percentage of type-1 fibres when compared to Caucasian men (Ama et al., 1986). However, the differences in fibre type proportion between the two ethnic groups in this study, was only slightly larger than the sampling error which occurs when using the needle biopsy to collect muscle tissue. However, a study in the US found that obese African-American woman had a lower percentage of type-1 fibres than obese Caucasian women (Tanner et al., 2002), while others have showed in a cohort of sub-elite endurance runners, that African men had lower proportions of type I muscle fibre than European men (Kohn, Essen-Gustavsson, & Myburgh, 2007). Others challenge this idea however, suggesting that environmental factors explain a large part of the variation (J. A. Simoneau & Bouchard, 1995).

Simoneau and Bouchard (J. A. Simoneau & Bouchard, 1995) suggest that fibre type proportion can be partly explained by inherited factors (45%) and partly by environmental factors (40%). Elite athletes involved in endurance exercise appear to have a high proportion of slow-twitch fibres, while sprinters have more fast-twitch fibres (Costill et al., 1976). While this seems to imply a fibre-type change with training, selection into events by individuals possessing a natural endowment can occur (P. D. Gollnick, Armstrong, Saubert, Piehl, & Saltin, 1972), which may in part explain the ethnic grouping observed in certain athletic events.

Interestingly, studies have shown that muscle fibre proportions can change with training. Adams et al. (Adams, Hather, Baldwin, & Dudley, 1993) found that resistance training led to

a decrease in type IIB fibres and an increase in type IIA fibres in a cohort of healthy men. Staron et al. (Staron et al., 1991) also saw an increase of type IIA fibres and a decrease in type IIB fibres after resistance training in a cohort of women that had detrained previous to the study. Both of these studies also showed that detraining causes the conversion in reverse (from IIA to IIB). Similar to resistance training, it appears that the shift from IIB to IIA occurs with endurance training also (Schantz, Billeter, Henriksson, & Jansson, 1982). A reduction in the proportion of type I fibres is seen in the denervated muscle of paraplegics, but can be reversed with muscle contraction by way of electrical stimulation (Martin, Stein, Hoepfner, & Reid, 1992; Rochester et al., 1995). All of these findings suggest that chronic contractile activity induces a conversion from faster, fatigable muscle fibres, to slower, fatigue resistant fibres. Detraining or denervation on the other hand, induces the conversion in the opposite direction, from slow to fast fibres. Nevertheless, it appears that in normal individuals fibre type conversion in response to training or detraining, is limited to the conversion from IIA to IIX and vice versa (P. Andersen & Henriksson, 1977; Houmard et al., 1993). While, studies in rats have revealed that electrical stimulation within denervated fast-twitch muscle fibres, can 'transform' these fibres into slow-twitch muscle (Windisch, Gundersen, Szabolcs, Gruber, & Lømo, 1998), no conclusive research exists to show conversion from type IIA to type I fibres in humans (Daugaard & Richter, 2001). Some even propose that exercise increases the oxidative capacity of muscle fibres rather than actually changing fibre type composition (P. D. Gollnick, Armstrong, Saubert, Piehl, & Saltin, 1972).

2.6.6 Summary

In summary, though it was previously thought that fibre type was solely genetically determined, it has more recently been shown that conversion between fibre types does exist, although to a small degree, with environmental stimulus. Ethnic differences in fibre type

proportion also exist, but caution must be used in interpreting the results of these studies as confounding factors that also relate to ethnicity, such as socioeconomic status, may explain part of the variation. It appears that chronic exercise induces a switch from faster, less insulin sensitive muscle to slower, more insulin sensitive fibres. Inactivity on the other hand, causes a shift from slower to faster muscle fibres. If this is so, the physiological benefits of exercise would be two-fold for those at risk of insulin resistance; first, from reductions in lipid content and improved oxidative capacity of individual muscle fibres, and second from an increased proportion of the slower, more insulin sensitive fibre types. Genetics, rather than determining a set fibre type proportion, may instead provide the limits of fibre type conversion in response to stimulus. Because of the invasive nature of the muscle biopsy, research on fibre type proportion has been limited in humans. Less invasive procedures, which measure muscle contractile properties externally, have produced results that are closely related to the fibre type proportions obtained through biopsy (E. Suter, Herzog, Sokolosky, Wiley, & Macintosh, 1993). However, it appears that these methods have not been utilized as a substitute to the biopsy technique in research, or to identify those at risk of insulin sensitivity. Exercise induced improvements in glucose tolerance are specific to the fibre types recruited, thus developing a non-invasive means to measure fibre type proportion could be valuable in prescription of fibre-type specific exercise.

2.7 The influence of environment and genes on diabetes prevalence

2.7.1 Ethnicity and health research

While obesity, by way of caloric imbalance, has been identified as the major factor associated with insulin resistance, it appears that some populations are still predisposed to this condition when body fatness is taken into account (Scragg, Baker, Metcalf, & Dryson, 1991; David Simmons & Thompson, 2004). Because most data revealing an ethnic disparity in health is based on national surveys or community questionnaires where ethnicity is self-identified, there are problems when interpreting this data in a genetic or physiological context. Accordingly, there is much debate surrounding the collection and analysis of ethnicity data in New Zealand and around the world.

First of all, because the meaning of words such as ‘race’, ‘ethnicity’ and ‘culture’ are often confused in the literature (D. R. Thomas, 2001), interpretation and application of such data is difficult. For example, of the 80 participants that were categorised as Māori in the *Auckland Region Coronary or Stroke Study* (ARCOS) only 50% (40) were classified as Māori in the death registration data (Graham, Jackson, Beaglehole, & De Boer, 1989). The reason for this discrepancy was that participants in the ARCOS study were classified by “cultural affiliation”, while ethnicity in the death registration was classified according to “biological origin – half or more Māori blood” (D. R. Thomas, 2001). Additionally, Thomas points out that changes in the census question regarding an individual’s ethnicity, which happened from 1981 to 1996, may not have been taken into account when interpreting health data over this time period (D. R. Thomas, 2001). Secondly, because the concepts of race and ethnicity are more representative of socio-cultural identification, rather than a common genetic ancestry

(Rebbeck & Sankar, 2005), the application of ethnicity data, in identifying physiological bases for health disparities, has been deemed invalid by some (Goodman, 2000).

In spite of this, there is clear evidence that self-identified ethnicity is closely associated with genetic variability (Tang et al., 2005), suggesting that while ethnicity may be more closely linked to socio-cultural factors, there is still a genetic basis to ethnicity. Nevertheless, methods of differentiating groups according to genetic ancestry are limited. In fact, only one study to my knowledge, has observed disparities in type-2 diabetes prevalence according to genetic ancestry (Gardner et al., 1984), and this study did show that type-2 diabetes did, in fact, correlate with genetic ancestry. However, the method used in this study to quantify ancestry was skin colouration, which is not necessarily an accurate representation of ancestry (Parra, Kittles, & Shriver, 2004).

Self-identified ethnicity is a complex concept which is difficult to interpret in the context of physiological research. Therefore, further research and discussion is required to ensure that ethnicity data is collected, analysed and interpreted appropriately when applied to health, medicine and scientific research.

2.7.2 Environment vs. genetics

Because problems exist in the interpretation of ethnicity data, it is difficult to understand the relative contribution of environmental and genetic influences in ethnic health disparities. For example, Diamond (Diamond, 2003) described how the lowest prevalence of diabetes is in rural third world areas where food is scarce and people are forced to expend energy to obtain food. Whereas, prevalence is highest in rural areas where food is easily obtained and a

sedentary lifestyle has become the norm, emphasizing the role of environmental factors on ethnic disparities in obesity and diabetes.

On the other hand, Diamond points out that prevalence of diabetes is relatively low among European groups living urban lifestyles and highest among Nauru islanders, Pima Indians of Arizona and Wanigela people in Papua New Guinea, all of which have only (relatively) recently adopted a 'Western' way of life. Thus, although Māori and other 'high-risk' ethnic groups appear to have an abnormal predisposition toward obesity and its co-morbidities, it is actually European groups who are unique, in that the prevalence of obesity and diabetes is relatively low even in a similar environment.

From the literature, a number of physiological characteristics can be identified which may be responsible, in part, for the disparity in metabolic control that can be seen between Europeans and other ethnic groups, such as NZEO and Māori in New Zealand. Physiological factors associated with insulin resistance include unfavourable body fat distribution (S. E. Kahn et al., 2001), a decreased fat-storage capacity in adipose tissue (Lewis, Carpentier, Adeli, & Giacca, 2002), a greater tendency for fat accumulation in muscle (D. A. Pan et al., 1997) and liver tissue (Seppälä-Lindroos et al., 2002), a greater proportion of fast-twitch muscle fibre (Lillioja et al., 1987a), reduced mitochondrial content/function (D. E. Kelley, He, Menshikova, & Ritov, 2002), and diminished insulin secretion (β -cell function) (Hollenbeck & Reaven, 1987).

Although literature is abundant which supports the stance that environmental factors determine ethnic health disparities, the purpose of this part of the review is to focus on physiological explanations for ethnic health disparities.

2.7.3 Genetic adaptation and ethnic health disparities

In view of the physiological characteristics associated with lipid accumulation and insulin resistance, many authors have sought to explain predispositions to obesity and poor metabolic health, in the context of genetic adaptation to environmental stresses. For example, Diamond reasons that, in response to gradually increasing food supply in Europe between the fifteenth and eighteenth century, Europeans may have developed a protective adaptation to the effects of caloric imbalance (Diamond, 2003). Alternatively, Fridlyand and Philipson (Fridlyand & Philipson, 2006) propose that the physiological characteristics which appear to protect Europeans from unfavourable lipid accumulation and insulin resistance, can be explained by, what they term, 'cold climate genes'. Their theory is based on the thought that as humans migrated to colder areas in the North, the metabolic functions associated with thermogenesis, adapted to aid survival in cold climates. Thermogenesis is largely associated with the basal metabolic rate (BMR), which is determined by metabolic processes in the mitochondria of skeletal muscle. In context of obesity and insulin resistance, basal metabolic processes are responsible for almost all expenditure of energy for an individual living a sedentary lifestyle. The implication is that those with an increased metabolic rate, by way of a genetic adaptation to cold climate, would have an advantage in reducing adiposity over others living a sedentary lifestyle, through an increased ability to oxidize excess substrate. Fridlyand and Philipson point out that while the ancestors of Europeans, who lived in many far North areas, would have benefited from such an adaptation, the adaptation would not have occurred in the ancestors of Africans, South East Asians and Pacific Islanders, who originate from equatorial areas. In connection with this, African American women were found to have a lower RMR than Caucasian women, even when lean body mass was accounted for (Forman, Miller, Szymanski, & Fernhall, 1998). Likewise, a study by Rush et al. (E. C. Rush, Plank, & Robinson, 1997) revealed that the resting metabolic rate of young Māori and Polynesian

woman was lower than in young Caucasian women. These authors also propose that this decreased resting metabolic rate may predispose Māori/Polynesians to obesity and the onset of diabetes. Fridlyand and Philipson observe that an increase in the number of mitochondria, or increased mitochondrial activity, could be the mechanism that adapts in a cold climate, increasing basal metabolic rate and heat production (Fridlyand & Philipson, 2006). Others have also suggested that physiological adaptation to cold climate is associated with increased mitochondrial content or activity (Lowell & Spiegelman, 2000). Interestingly, mitochondrial content is also associated with aerobic capacity and fibre type, both of which are associated with insulin sensitivity.

As discussed previously, fibre type proportion differs between ethnicities and a greater proportion of fast-twitch muscle fibre is associated with increased risk of insulin sensitivity and diabetes. It is interesting to note that in many instances the same ethnic groups which have been identified as predominantly 'fast twitch' in physiological studies, are also those who are identified as metabolically disadvantaged in epidemiological studies. For instance Tanner et al. found that obese African American women possessed a greater proportion of fast-twitch fibre when compared to obese Caucasian men (Tanner et al., 2002). What's more, Ama et al. found that sedentary Black men had a greater proportion of fast twitch fibre compared with Caucasian men (Ama et al., 1986). In accordance with these findings, the 1999/2002 National Health and Nutrition Examination Survey (NHANES) showed that 11% of African-Americans were diabetic compared with just 5.2% of Caucasian Americans (Cowie et al., 2006). Likewise, diabetes is more than twice as prevalent in Māori than in NZEO (when adjusted for age) (Ministry of Health, 2004b, , 2008). Thus, it is possible that a genetically determined fibre type proportion may be an underlying factor in predisposing, or protecting, certain ethnic groups from the detrimental effects of a sedentary lifestyle.

Nevertheless, no studies to date have investigated fibre type proportion in Māori, so the application of this theory to Māori is limited to assumptions gained from fibre-type studies done in other ethnic groups who share a disproportionate prevalence of obesity and diabetes.

Similar to the theories previously mentioned, Neel's 'thrifty gene' theory also seeks to explain ethnic disparities in insulin resistance as a result of survival adaptations. Neel's theory however, is based on the metabolic adaptations geared toward survival from starvation, rather than cold climate. This theory suggests that insulin resistance is a natural response to food scarcity, which preserves blood glucose for brain function (Neel, 1962; Stannard & Johnson, 2004). When combined with excessive feeding and a sedentary lifestyle however, over supply of substrate and energy imbalance makes this mechanism for survival a destructive condition.

In recent years, many more theories have been postulated such as the 'adipose tissue overflow hypothesis' (Sniderman, Bhopal, Prabhakaran, Sarrafzadegan, & Tchernof, 2007) which proposes that some ethnic groups, in this case South-East Asians, may have a reduced capacity to store fat in subcutaneous adipose tissue, leading to a greater accumulation of abdominal fat. Thus, because a greater distribution of fat in the abdominal area is associated with a higher-risk of metabolic disorder (S. E. Kahn et al., 2001; Ross, Aru, Freeman, Hudson, & Janssen, 2002), and because Asian populations seem to have greater abdominal adiposity compared with Caucasians of the same BMI (Wang et al., 1994), the effect of caloric imbalance on insulin sensitivity is greater in this ethnic group than those who are less prone to central adipose storage. Similar to the 'cold climate' theory discussed previously, Sniderman et al. suggest that the reduced capacity to store fat in subcutaneous adipose tissue could be an adaptive response to climatic influences. However, 'adipose tissue overflow' may not be a valid explanation in Māori and Polynesians, who are more muscular than Asians and

Caucasians of the same BMI (P. Deurenberg, Yap, & van Staveren, 1998; Swinburn, Ley, Carmichael, & Palnk, 1999), and have a relatively lower abdominal distribution of fat (E. Rush et al., 2004).

Although none of the theories discussed have been investigated in Māori, these theories warrant further investigation regarding the contribution of environmental and genetic factors to the development of obesity and its associated metabolic disorders. When taking into account the theories presented, it is possible that Māori, who likely descend from ancestors that lived close to the equator, and also faced periods of starvation, have a double genetic disadvantage when faced with a lifestyle of caloric imbalance by having ‘thrifty genes’, without the advantage of ‘cold climate genes’.

2.7.4 Summary

A number of theories have been developed to explain the genetic adaptations responsible for ethnic disparities in metabolic control seen in developed countries. When taking these theories into account, Māori are genetically disadvantaged in a modern, sedentary lifestyle. It appears that a genetically determined fibre type proportion may also increase the risk of obesity and therefore insulin resistance and diabetes. While some studies have shown fibre type to be genetically determined, it appears that environmental factors, such as training, may be just as important in determining fibre type proportion. A greater understanding of fibre type, and whether metabolic processes differ between ethnic groups, may shed some light on why the disparities exist in obesity, insulin resistance and diabetes. Because ethnicity is a social construct and not necessarily biological, developing methods to measure someone’s ancestry could also prove valuable.

3 The Interface between Exercise Science and Māori Health

3.1 Introduction

When the World Health Organisation (WHO) was first established, it defined health as a state of complete physical, mental and social wellbeing and not merely the absence of disease or infirmity (WHO, 1946). Similarly, a Māori view of health is holistic and focuses on wellbeing rather than just absence of illness (M. H. Durie, 1985). Past experience however, suggests that the focus of mainstream health systems and health research seems to be on the treatment of illness (Booth, Gordon, Carlson, & Hamilton, 2000). Furthermore, indigenous views are often overlooked as health is approached in a Western manner (Sachdev, 1998). Because we view the world differently, a Māori approach to health and research (along with all other aspects of life) differs from that of the 'norm' taken by non-Māori. Cultural sensitivity is important and can become an ethical issue when assumptions are made that certain requirements for research and interventions which are appropriate for non-Māori must also be appropriate for Māori. Because the Māori view of hauora (often incorrectly used synonymously as a translation of the English word for 'health') differs from a non-Māori view of health, the measures of success for hauora also differ from those expressed for the idea of non-Māori health. Hauora includes a spiritual and holistic view and is interdependent, involving friends and whānau, compared with the naturalistic, individualistic view of mainstream health models (M. Durie, 1994).

As lifestyles in developed countries, such as New Zealand, Australia and the US become more sedentary, and food supply more abundant, prevalence of obesity related illnesses such as diabetes have increased dramatically (Diamond, 2003). The different ethnicities living

within these developed countries seem to be disproportionately affected by this growing epidemic however, with indigenous groups suffering most (Gohdes; Moore & Lunt, 2000; Young, Reading, Elias, & O'Neil, 2000; Zimmet, 1979). Similar to indigenous people of other westernized nations, Māori in New Zealand are effected more by diabetes than New Zealand Europeans (K. McAuley et al., 2001; David Simmons & Thompson, 2004).

Although intervention programs have been developed to slow the increasing prevalence of diabetes, recruitment of Māori into many research and health intervention programs has not been successful (Murphy et al., 2003; Ni Mhurchua et al., 2007; Tuttle, 2002). While programs aimed at improving Māori well-being have good intentions, success of these programs is not dependant on the quality of the information presented but whether its approach is culturally appropriate. Programs that are aimed at Māori are no doubt beneficial to Māori health but cannot be expected to succeed if recruitment and delivery of services continues to be approached in a Western manner. Māori will continue to respond poorly if researchers and those who develop interventions continue to overlook Māori views; and according to current trends, if Māori continue to respond poorly and today's interventions are unsuccessful, an unsustainable burden on the health system (PricewaterhouseCoopers, 2001) and the endangerment of the Māori people is inevitable.

3.2 The Scientific Approach

Western science tends to be reductionist, and therefore seeks to understand behaviour and actions by breaking specific scientific units or concepts into smaller parts. According to a reductionist view, diseases and health conditions can be explained by understanding the cellular pathogenesis associated with the condition (H. Andersen, 2001). When related to diabetes for instance a biomedical view would seek to understand the condition's relationship

to cellular mechanisms and observe molecular imbalances within the cell. Though the physiological mechanisms of the condition are better understood due to this reductionist approach, the pharmaceutical interventions developed from this understanding does not seem to prove more effective than the prescription of whole body exercise (Stannard & Johnson, 2006). It has also been argued that the reduction of many diseases to cellular and molecular mechanisms is so complex that no conclusive explanation of disease pathology can be obtained through reduction anyway (H. Andersen, 2001). This complexity could explain why the mechanism/s that leads to insulin resistance still elude researchers as research suggests that insulin resistance is a result of a disorder in more than one part of cellular metabolism or physiology (B. B. Kahn & Flier, 2000; Stannard & Johnson, 2004).

A scientific understanding of physiological mechanisms is important however as it allows us to better understand the reason behind trends observed by epidemiological studies. Physiological findings have also been a driving force in motivating legislative change related to health problems (Booth, Gordon, Carlson, & Hamilton, 2000).

Another characteristic of Western science is that it is naturalistic in that it disregards any supernatural phenomenon as an explanation (Harris & Mercier, 2006). A disregard for that which many indigenous people may consider as spiritual knowledge, whether intended or not, can be a barrier to Māori acceptance of scientific research as a valuable tool in their development.

Although science exists as a vehicle to understand the unexplained, it has drawn a clear distinction between itself and indigenous understanding. Because indigenous knowledge includes a spiritual aspect, approaches research differently and is based on a different set of

values, it is pushed aside and branded irrelevant to scientific knowledge. Science in its purest and most basic sense however is not just the standard chemistry, physics and biology associated with modern sciences, but is defined as knowledge, whether obtained through the usual scientific methodology of observation, experimentation and theory or not (Harris & Mercier, 2006).

3.3 The Holistic Approach – The Māori Approach

The WHO and other health organisations are taking a more holistic view of health than typical medical sciences have taken in the past. A holistic approach maintains that all levels of social, psychological and biological health are linked to each other so that if one changes, the others are affected. According to this view, the characteristics of wellbeing, a collective outcome, cannot be understood by focusing on the characteristics of each component separately (H. Andersen, 2001).

Where Māori views differ from that of the holistic view taken by the WHO however is that the Māori holistic view of health includes a spiritual aspect. This spiritual aspect does not only encompass the Māori link to God and the divine, but includes a strong sense of unity with their environment, which link is thought to be the most defining element of indigeneity (Mason Durie, 2004; Kame'eleihiwa, 1992). Indigenous people globally have holistic views similar to the Māori concept of health and wellbeing, known to Māori as hauora (Smylie, Anderson, Ratima, Crengle, & Anderson, 2006). This concept includes a balance between the mental (hinengaro), physical (tinana), social/extended family (whānau) and spiritual (wairua) dimensions of health (M. H. Durie, 1985; Smylie, Anderson, Ratima, Crengle, & Anderson, 2006). Māori Health models such as Whare Tapa Wha, developed by Mason Durie, represent these four aspects of health and have been adopted by many as a contemporary Māori view of

health. Māori are also more concerned about the health and wellbeing of whānau collectively rather than that of the individual (M. Durie, 1994).

Though it may be seen that science has disregarded the legitimacy of indigenous knowledge, indigenous people often dismiss the scientific method due to its failure to recognise spiritual aspects of learning (Mason Durie, 2004). In order for Māori to develop in a colonized world, Māori health researchers must ask the question “How can Māori health issues be better understood through Western-science, while still maintaining cultural integrity?”

3.4 Exercise Science – The Bridge

Though research would suggest many indigenous groups to be physiologically susceptible to diabetes and its complications, a few studies show that prevalence of diabetes is low in indigenous people that continue to live a traditional lifestyle, but high in those who adopt a westernized way of living (Eaton & Konner, 1985; Zimmet, 1979). Though it is unlikely and impractical to think that Māori could (or would) return to a completely traditional way of living, the implication is that intense exercise (as prescribed in a gym culture) may not be necessary for the maintenance of health in Māori, but that the everyday activities similar to a traditional lifestyle, such as cultivation of land, hunting, physical labour or even kapahaka, would suffice. It is important then that the benefits of exercise (or increased physical activity) are understood by Māori.

Exercise physiology is the study of how our bodies’ structures and functions are altered when we are exposed to acute and chronic bouts of exercise (Wilmore, Costill, & Kenney, 2005). Literature regarding the research approach of exercise physiology is scarce however, though one editorial in the *Journal of Physiology* concluded ‘that the main regulatory and adaptive

responses to acute and chronic exercise defy simple reductionist explanations' (Joyner & Saltin, 2008). This implies that the study of exercise physiology is best approached at a holistic level.

As type-2 diabetes is a disorder within skeletal muscle, it would be ideal to study muscle in a contracting state in order to better understand the effects of exercise on the physiological mechanisms associated with diabetes. Nevertheless, because it is not (yet) possible to study muscle cells directly in vivo during exercise, the responses to exercise of other systems (e.g. respiratory, cardiovascular systems) are observed instead. These systems, which also adjust to the energy requirements of contracting muscle at the onset of exercise, are related to skeletal muscle metabolism and are therefore an indirect way of observing the metabolic changes that occur in vivo. Therefore in order to understand diabetes and its relationship to muscular metabolism, the body must ultimately be studied as a system.

Exercise physiology does follow the scientific methodology of observation, experiment and theory, but because it tends to be more holistic in its approach to health issues than other physiological sciences, it could prove a valuable tool in bridging a gap between western science and indigenous knowledge. Durie expresses that research and methods need to make sense to Māori (M. Durie, 1994), and because exercise science retains this systems approach and requires an understanding of the whole body response to exercise, its application is easy and often appeals more to those that are inexperienced in scientific research.

Understanding the physiology of exercise in Māori also allows us to better understand the quantity and the type of exercise that could best benefit Māori (Booth, Gordon, Carlson, & Hamilton, 2000). Regardless of molecular or cellular explanation, the simple truth is that

physical activity and exercise is likely to reduce the risk of diabetes in Māori similarly to non-Māori (Burstein, Epstein, Shapiro, Charuzi, & Karnieli, 1990; Cauza et al., 2005), along with many other chronic diseases. While the study of exercise physiology may appear to focus on just one aspect of the four part health model, it is interesting that literature suggests that the effects of exercise are not only limited to tinana but cross to other aspects of the hauora model by improving mental health, whānau health and overall well being (Fox, 2007; Penedo & Dahn, 2005).

It is my concern as a Māori exercise physiologist however, that the emphasis on the holistic view of health among Māori and their movement toward such multi-dimensional health models as Whare Tapa Wha, may lead some Māori to under-emphasize the importance of physical health (tinana) in Māori development. Though tinana is only one part of a multi-dimensional view of health, it must not be neglected as less important than the other parts. If traditional views incorporated into everyday life are the desire for Māori, then physical activity and healthy living, normal parts of the ‘traditional Māori lifestyle’, must be placed near the top of priorities. Whether consciously for the benefit of health or not, pre-European Māori were physically active. In saying this it is not intended to take away from the other very important parts of hauora represented in such successful models as Te Whare Tapa Wha, but to emphasize the importance of re-introducing, rather than ‘educating’, Māori to the physically active lifestyles their bodies are genetically designed for (Booth, Gordon, Carlson, & Hamilton, 2000; Stannard & Johnson, 2006). Exercise physiology will allow Māori to better understand exercise and how it relates to their wellbeing, and could therefore prove a valuable tool toward achieving hauora. The idea that exercise and eating healthy is a Western view, introduced by Pakeha, is false. However the problem may be in the way the ‘physically

active lifestyle' is presented in New Zealand, which may not appeal to Māori and cause the message to appear as 'Western medicine'.

3.5 What is happening in New Zealand?

In their 2000 world health report, the WHO made the statement that one of three principal goals for health-care systems should be 'responsiveness to the expectations of the population' (Smylie, Anderson, Ratima, Crengle, & Anderson, 2006). In the past the New Zealand health system, dominated by European influence (HWAC, 2006), has not been 'responsive to the expectations' of Māori, as their views and cultural concepts toward health have not been included. Recently however, the New Zealand government has attempted to better meet expectations of its Māori population with strategies such as He Korowai Oranga and Whakatātaka which were developed by the Ministry of Health as part of a framework for the public health sector to follow in its support of Māori communities and whānau. The main goal of He Korowai Oranga is achieving 'whānau ora'; the realisation of maximum health and well-being among Māori families and communities. These strategies acknowledge the importance of a close relationship between the government and Māori at all levels i.e. Iwi (tribe), hapu (sub-tribes), community and whānau (family) levels etc. An underlying theme that runs through all of these government strategies is that Māori need to be involved in all aspects of Māori health and that the incorporation of a Māori approach is necessary for successful implementation.

3.6 Implications – The Interface

Throughout the world much has been said regarding the incorporation of indigenous values into national health systems. During a national Māori health conference held in 1984, Hui

Whakaoranga, Māori recommended that 'health and educational institutions recognise culture as a positive resource' (*Hui Whakaoranga*, 1984). Harris & Mercier also comment that learning from more than one knowledge system can only increase knowledge and help people make better-informed decisions in a multi-cultural world (Harris & Mercier, 2006). Just as the scientific world can benefit from the addition of Māori knowledge and views, Māori can benefit from scientific knowledge and methods of research. Māori researchers agree that kaupapa Māori research is not against the use of scientific methods (Cram, Smith, & Johnstone, 2003; Mason Durie, 2004), but are concerned about cultural sensitivity and whether Māori benefit directly from the research they're involved in (Bishop, 1998). In order for Māori to benefit from science however, Māori must first be able to trust in researchers and scientific research systems. This will be possible with more Western-trained Māori scientists and an understanding and acceptance of Māori values among researchers and research institutions (Harris & Mercier, 2006).

Focusing research on the prevention of diabetes, rather than treatment, is the key to ridding Māori of the burden of diabetes and other obesity related chronic illnesses. When related to health, many physiologists take a reductionist approach, racing to understand the molecular mechanisms of diabetes in search of the 'diabetes pill', and therefore aiming at the secondary/tertiary treatment of the disorder. Exercise physiology on the other hand, is focused on primary prevention (preventing obesity, insulin resistance, chronic illness etc.) and the response of the whole body system to exercise. While pharmaceutical interventions have proven effective in treating insulin resistance, it is often at the detriment of other bodily systems (Doggrell, 2006; Stannard & Johnson, 2006). In contrast, exercise improves insulin sensitivity better than pharmaceutical intervention and affects other body systems (cardiovascular, respiratory, neural systems) in a positive way (Stannard & Johnson, 2006).

Similarly, traditional Māori health practices focused on primary prevention. Although procedures existed for the treatment of disease, the concept of ‘tapu’ which applied to all aspects of Māori life, was preventative by nature, acting as a type of illness and accident prevention (M. Durie, 1994). Not only is it more humane to avoid the suffering of diabetes but it is also much more cost effective in terms of the economic burden on health care (Booth, Gordon, Carlson, & Hamilton, 2000).

3.7 My Research

The aim of my research is not only to better understand how insulin resistance leads to diabetes in Māori, but to develop a suitable methodology for research at the interface of science and Māori health and to help Māori develop trust in scientific research techniques. As discussed earlier, exercise physiology and Māori health are focused on primary prevention, which is the main purpose for studying a cohort of non-diabetic Māori men that are clinically and self-identified as healthy.

Māori are under-represented as scientific researchers (Harris & Mercier, 2006) as well as participants in scientific research, but one who observes my insulin resistance study is able to witness a rather rare scene of Māori participants participating as part of a whānau-like group in exercise physiology testing procedures, conducted by, and under, the direction of a team of Māori researchers. Much like the approach taken in public health studies, this research involves larger cohorts than most exercise physiology studies with between 24 and 50 subjects in each study.

Because the achievement of hauora is the ultimate goal of Māori research, there also needs to be changes when measuring the outcome of interventions and research to observe overall

wellbeing rather than just the absence of illness (Mason Durie, 2004). I hope to apply this to my research by including questionnaires and group-interviews based on Māori perspectives of health, as part of the research process. By doing this, I will be able to gain an understanding of how participants feel about exercise and the methods of research that were used in my research, and the effect of these methods on wairua, hinengaro, tinana and whānau; similar to principles of a framework developed by Māori health researchers to measure mental health outcomes (M. H. Durie & Kingi, 1998).

Research also suggests that data collected on Māori and other indigenous groups is taken but often not compiled and fed back to the groups or communities from which they came (Smylie, Anderson, Ratima, Crengle, & Anderson, 2006). In my research however, I have compiled results of findings and reported back to the individuals and the group as a priority activity, explaining the implications of my research findings and how it may affect the individual participant as well as Māori collectively.

3.8 Limitations of the methodology

As this approach is an innovation it suffers the same limitations as all innovations:

a) There are no examples to follow, b) there may be incompatible principles/methods which need to be reconciled, and c) It may or may not be relevant to participants of other ethnic groups/cultures

Also, as Māori are usually represented at a lower socioeconomic status and health status, it can often be difficult to recruit Māori participants that show a true representation of the NZ Māori community. Many of the Māori who participated in this research came from friend,

church, employment or university networks so are likely more healthy, better educated and better employed than average NZ Māori.

What's more, ethnicity is self-identified in the cohorts I studied. As discussed previously (see Section 2.7.1), ethnicity is more closely associated with socio-cultural identity than genetic make-up, making the interpretation of ethnicity data difficult when observing physiological characteristics. Thus, those who identified as Māori in my studies did not necessarily share genetic traits. Nevertheless, this problem is not unique to my studies, and discussion regarding the issue of ethnicity data in scientific research, is abundant in current literature.

3.9 Discussion

The paradox with Western science is that while it discovers treatment and technologies to better understand and conquer diabetes, the same advances in technology have led to the sedentary lifestyle that supports the condition. The purpose of this paper is not to dispute the value of scientific research which obviously speaks for itself, nor argue that a Māori approach to research is more effective, but to present the possibility of using both methods of research to benefit Māori and non-Māori alike. As indigenous people, it is our right to have Māori views used in the research process and in the application of findings into intervention, especially when we are hardest hit by the research topic. Although Māori are becoming more involved in Māori-focused research, the ultimate goal in this sense should be Māori-driven scientific research that is developed by Māori for Māori, using a combination of scientific and Māori methods while maintaining cultural integrity. The scientific idea that indigenous knowledge is only applicable to pre-colonization times is a barrier that will only hinder health research. The New Zealand health system, which is burdened by 'unhealthy Māori', would also benefit greatly by investing more into improved Māori wellbeing.

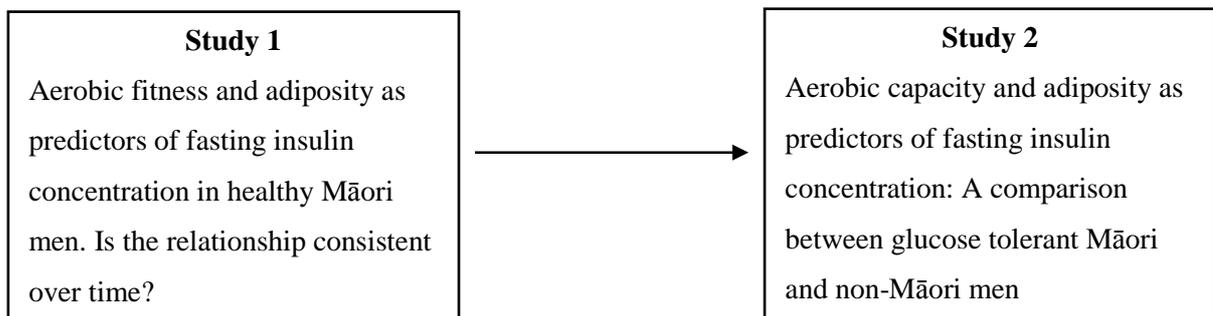
Research in the past has been focused on the secondary and tertiary treatment of diabetes, whereas a shift in focus is needed toward primary prevention. As habits are formed early in life, these interventions should appeal to rangatahi (young people) and tamariki (children). The editors of *The New England Journal of Medicine* wrote “We need to do a better job of educating people about healthful diets...Encouraging lifelong, regular exercise in children may well have the greatest effect in terms of preventing obesity”(Kassirer & Angell, 1998). On the other hand, while interventions such as “healthy eating – healthy action” (also known as Oranga kai- Oranga Pumau or HEHA)(Ministry of Health, 2007) have focused on ‘educating’ Māori about key health issues such as the need for exercise and a balanced diet, it is ignorant to believe that Māori are not aware of the dangers of a sedentary lifestyle and bad diet already. These well planned strategies with their high funded research and interventions may yield the greatest success among Māori when the approach is made culturally appropriate for Māori.

The notion of applying Māori knowledge and views to current health research, alongside a physiological approach, will only benefit Māori and scientific knowledge equally. Durie puts it simply that exploring the interface of Māori views and science allows us to shift the focus from “proving the superiority of one system over another to identifying opportunities for combining both”(Mason Durie, 2004). While we are at the exploratory stages of the interface between exercise physiology and Māori health research, the combination of these two methods could become a powerful tool in the future, to better understand the problems facing Māori and allow the development of more effective interventions.

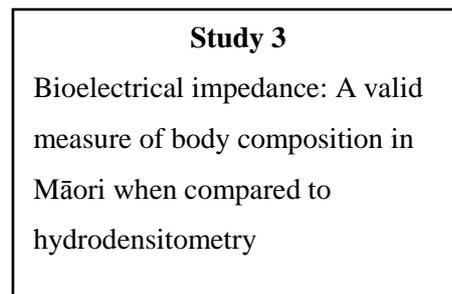
4 Hypotheses and Research Design

4.1 Research Design

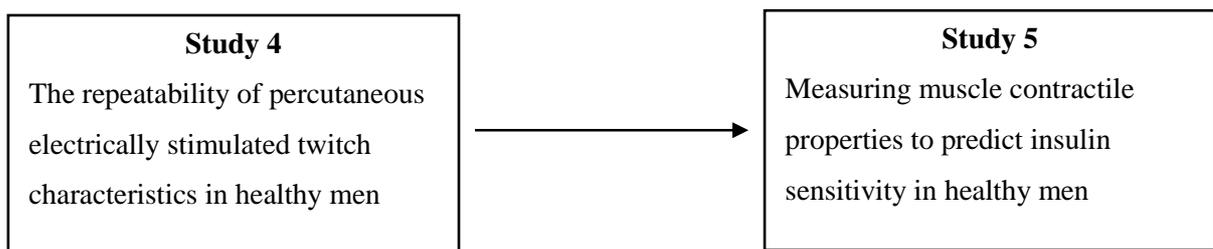
The following five studies were chosen to investigate three separate physiological factors, each of which have been shown to be associated with obesity and insulin sensitivity: Aerobic capacity (*fitness*), body composition (*fatness*), and skeletal muscle fibre-type proportion (*fibre-type*). With regard to Māori, no published studies have investigated fibre type proportion and only one study has investigated aerobic capacity. As Māori are worse affected by obesity and its co-morbidities, these studies were chosen to better understand how the aforementioned physiological factors might contribute to the development of insulin resistance and diabetes in Māori.



Study 1 and *Study 2* specifically investigate the relationship between aerobic capacity, body composition and markers of insulin sensitivity, so as to better understand the interaction between fitness, adiposity and insulin sensitivity. *In Study 1*, aerobic capacity, adiposity and markers of insulin sensitivity, were re-tested in a Māori cohort from whom data was collected in a previous study. This was done to confirm the findings of that previous work by observing whether those relationships remained constant over time despite small changes in the independent variables. *Study 2* included a cohort of non-Māori men, the purpose of which was to observe whether the aforementioned relationships differed between Māori and non-Māori.



Following on from the investigation of body composition and insulin sensitivity, *Study 3* sought to validate the use of BIA as a suitable method for body composition assessment in Māori. BMI is the most frequently used method of assessment in population studies and clinical assessment, but ‘cut-off points’ do not take into account ethnicity-related differences in body composition. On the other hand, the most accurate means of measuring body fatness, such as hydrodensitometry, requires expensive equipment, trained technicians and is often impractical for those being measured. Thus, if BIA, which is now relatively cheap and easy to administer, proves valid in Māori it could provide a simple means of measuring body fatness in the laboratory and field.



In addition to the focus of fitness and fatness as physiological determinants of insulin sensitivity, *Study 4* and *Study 5* were aimed at understanding the relationship between insulin sensitivity and another physiological determinant: muscle fibre type. Because measurement of fibre type proportion requires muscle biopsies, *Study 4* was chosen to investigate the

feasibility of employing an innovative, non-invasive technique, developed to measuring muscle contractile properties (and therefore muscle composition indirectly). *Study 5* then focused on whether the contractile properties measured with this technique, could predict insulin sensitivity.

4.2 Hypotheses

Study 1: The relationship between aerobic capacity, body fatness and blood markers of insulin sensitivity in a group of healthy Māori men will remain, even if variables such as body fatness and aerobic capacity change over time.

Study 2: The relationship between aerobic capacity, body fatness and blood markers of insulin sensitivity are similar in a group of healthy Māori and non-Māori men.

Study 3: Measures of body composition obtained with a bioelectrical impedance apparatus correlate well with those obtained by hydrodensitometry, in healthy Māori and non-Māori men.

Study 4: Measuring time to peak force (and half peak force) of an electrically evoked twitch in the vastus lateralis, will produce repeatable results as well as variance between subjects.

Study 5: Twitch characteristics obtained with the method previously described, will accurately predict insulin sensitivity in a cohort of healthy men.

5 Study 1: Aerobic fitness and adiposity as predictors of fasting insulin concentration in healthy Māori men. Is the relationship consistent over time?

5.1 Introduction

Obesity, as a result of excessive energy intake and a sedentary lifestyle, is primarily responsible for accumulation of fatty acids and triglycerides within myocytes (muscle cells). This accumulation is associated with impaired insulin signalling within these cells; a disorder known as insulin resistance. Insulin-mediated glucose uptake from the blood is reduced due to this insulin resistance, and eventually contributes to the hyperglycaemia which defines diabetes (Goodpaster, Thaete, Simoneau, & Kelley, 1997; Phillips et al., 1996).

A single bout of exercise on the other hand, improves insulin action for at least 48 hours post-exercise (Mikines, Sonne, Farrell, Tronier, & Galbo, 1988). Although the mechanisms as to why this occurs are not completely understood, it is widely known that the muscle's fuel requirement increases during contractile activity (exercise), increasing the rate of fat oxidation and thereby reducing intramyocellular triglycerides (IMTG) (N. A. Johnson et al., 2003), which are closely associated with the insulin signalling disorder (Goodpaster, Theriault, Watkins, & Kelley, 2000; Greco et al., 2002; Koopman et al., 2006; Luc J. C. van Loon, 2004). The maximal rate of fat oxidation within muscle cells is limited by the mitochondrial content of the cell (J. O. Holloszy, 1967; J O Holloszy & Coyle, 1984).

Chronic contractile activity (exercise training) not only decreases body fat, which independently improves insulin sensitivity (Golay, Felber, Dusmet, Gomez, & Curchod, 1985), but increases the body's ability to oxidise lipid such as IMTG, improving insulin

signalling independently of weight loss (DeFronzo, Sherwin, & Kraemer, 1987; Dengel, Pratley, Hagberg, Rogus, & Goldberg, 1996; Duncan et al., 2003). This improvement in muscle oxidative capacity, is largely due to an increase in mitochondrial content in response to chronic muscular contraction (J O Holloszy & Coyle, 1984).

Surprisingly, endurance trained athletes also have an elevated concentration of IMTG, yet are very insulin sensitive (Goodpaster, He, Watkins, & Kelley, 2001; Morgan, Short, & Cobb, 1969; Schrauwen-Hinderling et al., 2003). However, acute exercise bouts along with increased muscle oxidative capacity within endurance trained individuals, results in a rapid turnover of these fats and probably ensures insulin sensitivity (Goodpaster, He, Watkins, & Kelley, 2001). Thus, it appears that insulin sensitivity is more a function of the rate of fat oxidation within myocytes, rather than the concentration of fat per se.

It has been previously shown that in a group of young, apparently healthy Māori men, variations in fasting insulin concentrations and insulin sensitivity (HOMA-IR) can be partially accounted for by variations in both whole body adiposity and aerobic fitness (Stannard, Holdaway, Sachinwalla, & Cunningham, 2007). Whilst maximal aerobic fitness ($VO_2\text{max}$) may be limited by oxygen delivery to the working muscles (cardiac output) (González-Alonso & Calbet, 2003), a high $VO_2\text{max}$ also requires high oxidative capacity of the working muscle to utilize that oxygen (Bassett Jr & Howley, 2000).

Although a number of studies have investigated aerobic fitness and its relationship to insulin sensitivity, almost all of these have been done with Caucasian subjects, and have measured oxidative capacity using invasive biopsy procedures (C. R. Bruce et al., 2003; David E. Kelley, Goodpaster, Wing, & Simoneau, 1999). Only the previously mentioned study by

Stannard et al. has investigated this relationship in Māori, and they used a non-invasive method to measure the aerobic fitness of the working muscle ($VO_2\text{maxLBM}$). Because this study was the first of its kind within Māori, and its results differed somewhat from the results of others using similar testing methods in Caucasian men (Coon, Bleecker, Drinkwater, Meyers, & Goldberg, 1989), it is important that further studies be conducted to confirm these findings. One method of confirming these relationships is to retest the same study participants after a period of time to see if the same associations exist. This proves particularly powerful if there are changes in one related variable such as fitness or adiposity and the other related variable also changes accordingly.

Thus, the purposes of this study was to investigate whether the relationships observed between %BF, aerobic capacity and blood markers of insulin sensitivity in a cohort of Māori men, would exist over time after changes in fitness and body composition had occurred.

5.2 Methods

5.2.1 Study Design

Three years prior to this study, 24 ‘healthy’ Māori men participated in a study to investigate the relationship between aerobic capacity (as measured by $VO_2\text{maxLBM}$), body composition and markers of insulin sensitivity (Stannard, Holdaway, Sachinwalla, & Cunningham, 2007). The original study was the first of its kind using a cohort of young Māori men and produced results that differed to previous studies using only persons of Caucasian heritage. It was the intention of this study to follow-up with these same 24 Māori men and to observe whether the relationships established in the first study were consistent over time when variables such as body weight, body fat %, and aerobic capacity ($VO_2\text{maxLBM}$) had changed.

5.2.2 Participants

24 Māori men, aged 31 ± 6 (mean \pm SD), who were part of a study investigating these relationships 2-3 years earlier were contacted to be re-tested. Only 12 of the 24 participants from the first study responded and agreed to be tested in the repeated study. All participants were originally recruited from Māori service providers and a sports group from Whanganui, Manawatu and Wellington. All were of varying physical activities though none were ‘well-trained’.

All participants completed a health screening questionnaire¹ and none reported diagnosed diabetes or cardiovascular disease, though the results of one participant suggested insulin resistance in the previous (Sydney) study.

Table 5-1 – Participant physical characteristics

Name	Age (yrs)	Height (m)	Weight (kg)	BMI (kg/m²)
LB	22	1.74	70.50	23.4
RW	23	1.82	88.20	26.6
TD	22	1.89	99.50	27.9
SW	25	1.81	94.50	28.8
NB	32	1.78	75.00	23.7
RC	32	1.82	90.00	27.2
CH	37	1.80	102.70	31.7
BC	36	1.83	91.40	27.3
JH	35	1.77	79.10	25.2
TK	38	1.74	94.70	31.3
JG	32	1.79	85.50	26.7
JR	27	1.82	152.70	46.1
Mean	31	1.80	93.02	28.6
<i>Standard Deviation</i>	<i>6.0</i>	<i>0.04</i>	<i>20.9</i>	<i>6.0</i>

¹ The questionnaire used was based upon the Physical Activity Readiness Questionnaire (PAR-Q) which originates from the British Columbia Dept of Health (Canada), as revised by Thomas et al. (S. Thomas, Reading, & Shephard, 1992) and Cardinal et al. (Cardinal, Esters, & Cardinal, 1996) and with additional requirements of the Massey University Human Ethics Committee.

5.2.3 Protocol

Each participant began a water-only fast in the evening after eating dinner. In the morning (after at least 10 hours fasted) participants underwent an oral glucose tolerance test (OGTT) in order to establish fasting insulin/glucose levels and insulin/glucose levels 2 hours after a glucose load. After a light breakfast following the OGTT, body composition was measured by hydrodensitometry. Participants then performed a treadmill test to establish their VO_{2max} .

In following with a kaupapa Māori approach, I aimed to promote whanaungatanga among the research participants, a concept associated with building relationships and finding common ground, so testing sessions were done in groups of 3-4 men, rather than individually. Participants also shared meals, before and after testing, in order to build relationships between researchers and participants. In most instances, karakia (prayers) were also offered before testing sessions. At the conclusion of the study, all participants received a compiled report of their results as well as the mean results of the group and population norms.

5.2.3.1 Blood biochemistry

After an overnight fast (10-12 hours post-prandial), participants underwent a standard oral glucose tolerance test (OGTT) at an accredited blood pathology lab (Aotea Pathology Ltd, Wellington). Fasting blood samples were collected for analysis of fasting glucose (FastGluc) and fasting insulin (FastIns) before a 75ml glucose beverage was given. Two hours after the glucose load was taken, blood samples were again collected for analysis of glucose and insulin, 2 hours post-glucose load (2hrGluc & 2hrIns).

Analysis of blood was also performed at these accredited laboratories where serum was extracted and frozen for analysis. Serum Glucose concentrations were measured using a

Roche 917 autoanalyser with Gluco-quant® kits (Roche Diagnostics, Mannheim, Germany). Serum insulin was measured with a Roche Elecsys® 2010 via an immunometrical method using electrochemical luminescence. Blood biochemistry followed the same methods used by Stannard et al. (Stannard, Holdaway, Sachinwalla, & Cunningham, 2007). Extraction and analysis of blood was performed at the same lab in Wellington for both arms of the study.

Insulin sensitivity was estimated using the homeostasis model assessment-insulin resistance (HOMA-IR) model (Matthews et al., 1985).

5.2.3.2 Maximal oxygen uptake

Respiratory exchange of participants was measured during an incremental exercise protocol on a WOODWAY® treadmill (the *Desmo '05', Wisconsin, USA*). The Bruce protocol (R. A. Bruce, 1971) was chosen as it was used in the initial study and was practical for those that were unfamiliar with treadmill running or were relatively unfit. Participants were encouraged to continue exercising for as long as possible, as the speed and gradient of the treadmill increased every 3 minutes, in order to obtain intensities capable of eliciting maximal VO_2 . Volitional fatigue was defined as when the subject grasped the safety railing of the treadmill to finish the treadmill test.

Expired respiratory gases were sampled throughout the entire exercise test by an online gas analysis system using Moxus (version 2.1.05) software (*AEI technologies Ltd, Illinois, USA*). Expired air was collected via a Hans Rudolf mouthpiece with a turbine attached to measure expired volume. Expired air entered a mixing chamber and was analyzed for expired fraction of CO_2 using an infrared carbon dioxide sensor and expired oxygen using a zirconium cell-based oxygen sensor (AEI, Illinois, USA).

Both the analysers and software were calibrated each morning before testing was performed using a predetermined mixture of O₂ and CO₂ (4.9% CO₂, 15.6% O₂ in Nitrogen) while the turbine was calibrated with a 3000ml Hans Rudolf calibration syringe. Data were averaged by the software and displayed in 10 second intervals. VO₂max was defined as the highest rolling 1 minute average VO₂ (six 10 second recordings) obtained during the test. VO₂max was expressed relative to lean body mass (VO₂maxLBM) rather than relative to whole-body mass as it better represents the aerobic capacity of working muscle, independent of fatness (Coon, Bleecker, Drinkwater, Meyers, & Goldberg, 1989; Stannard, Holdaway, Sachinwalla, & Cunningham, 2007).

5.2.3.3 *Body composition*

Hydrodensitometry was performed in the morning between 1-2 hours after a small meal (the first meal post-fast) for determination of body volume. Participants wore tight fitting shorts or a swimming costume while in the water tank. Body weight on land was measured using a Sartorius electric scale (EA Model, Goettingen, Germany) and height was measured using a wall mounted 'Harpenden' stadiometer (*Holtain Ltd, Crosswell, UK*).

Participants were given up to 10 minutes to practice the submersion technique and familiarize themselves with the procedure. They were suspended in the water by a harness-like seat attached by ropes to a force transducer sited above the tank. The transducer output, once calibrated would be amplified and converted to a digital signal and used to indicate the underwater weight of the subjects. Once familiar with what was required, subjects were asked to exhale as much air from their lungs as was comfortable while submerging completely underwater by bowing the head forward as far as was comfortable. Once the subject was fully submerged and relatively stable, the measurement of underwater weight began using Labview

software (Eagle Scientific, USA) programmed by our group. Participants were required to stay submerged for 5 seconds as underwater weight was measured. The software provided a graph of underwater weight, during the 5 second period. An average of the 5 second reading was taken, in order to account for the sinusoidal fluctuations in transducer output that occurred from the natural motion of the water.

After the 5 second underwater period, participants were given a signal from the operator to raise their head out of the water. After participants had raised their head out of the water, residual lung volume was measured via oxygen dilution using an oxygen re-breathing technique described by van der Ploeg et al. (van der Ploeg, Gunn, Withers, Modra, & Crockett, 2000). The re-breathing apparatus consisted of a 5L (rubber) anaesthetic bag (Vacumed, California, USA) containing 4.25L of pure oxygen attached to Hans Rudolf 3 way 'sliding typeTM' Manual directional control valve (2870 series, Missouri, USA). A disposable 'SureGard' respiratory filter was used as a mouthpiece for participants, and the control valve allowed the operator to open or close the flow of oxygen from the bag to the mouthpiece.

After raising their head out of the water after the weighing, the mouthpiece of the apparatus was guided into their mouth and participants were asked not to take their first breath until the mouthpiece was securely in the mouth, the lips sealed around it, and the valve opened that allowed the participant to breathe the closed circuit oxygen. After 5-6 breaths in the apparatus, the valve was closed and the gas analysed for O₂ and CO₂ concentration using the same gas analysers used in the exercise test above. Body fat percentage was then calculated using previously established methods (Brozek, Grande, Anderson, & Keys, 1963).

Each participant performed the measurement protocol at least 3 times. Percentage body fat was taken as the average of the 3 measurements. If deviation outside 2% body fat occurred, a fourth measurement was also taken, and measurements that deviated outside 2% body fat were disregarded.

Two point calibration of the load cell was performed (once with no weight and again with a 5kg weight) each morning before testing took place.

5.2.4 Statistical Analyses

Paired t-tests were used to identify any significant changes in the variables measured, between the first test (Sydney) and the second test (Wellington). Simultaneous multiple regression was performed to ascertain whether the dependant variables relating to insulin sensitivity (HOMA-IR, Fast-Ins, 2hr-Ins, Fast-Gluc, 2hr-Gluc), could be significantly predicted by the independent variables (%BF, $VO_2\max$ LBM). Testing date was also included as an independent variable, to observe the effect of time on these relationships. SPSS version 10 was used for all statistical analyses, while graphs were made using Microsoft excel 2003.

5.3 Results

It was my initial intention to re-test all of the 24 Māori men that participated in the initial study (Sydney study). However, only 12 of the participants from the Sydney study were available to take part in the repeated test (Wellington study). Of the original 24 participants, six had moved out of the country, five were unable to be contacted, one was not interested in participating and one could not participate due to injury.

The results of the 12 participants in this study will only be compared to the results of these same participants in the Sydney study. Because of this, the results from the Sydney study of the 12 participants referred to in this text, may vary from those described in the original study (Stannard, Holdaway, Sachinwalla, & Cunningham, 2007), which included all 24 participants.

Tables of individual results for this study are found in the appendix (Appendix A – Study 1).

5.3.1 Body composition and aerobic capacity

Mean body weight increased from $92.3\text{kg} \pm 20.7$ (mean \pm SD) in the initial study to $93.7\text{kg} \pm 20.9$ in the repeated study, and %BF decreased from $22.4\% \pm 9.2$ to $20.9\% \pm 8.5$, although neither of these changes were statistically significant ($p > 0.05$) (Table 1.2). Of all the variables measured, only LBM, which increased from an average of $70.3\text{kg} \pm 8.7$ to $71.9\text{kg} \pm 10.0$, and VO_2maxLBM , which decreased from 59.4 ± 6.4 ml/kg l.b.m/min to 50.3 ± 2.1 ml/kg l.b.m/min, changed significantly ($p < 0.05$) between the two testing periods. Only one participant had an increase in VO_2maxLBM between testing sessions (from 50.08 to 60.84).

Table 5-2 – Body composition and aerobic capacity

	Sydney		Wellington		Paired
	Study		Study		t-Test
	Mean	SD	Mean	SD	p
Weight (kg)	92.3	20.7	93.7	20.9	0.535
BMI (kg/m²)	28.5	6.1	28.8	6.0	0.595
%BF	22.4	9.2	20.9	8.5	0.223
LBM (kg)	70.3	8.7	71.9	10.0	0.019
VO₂max LBM (ml.kgLBM⁻¹.min⁻¹)	59.4	6.4	50.3	7.4	0.002

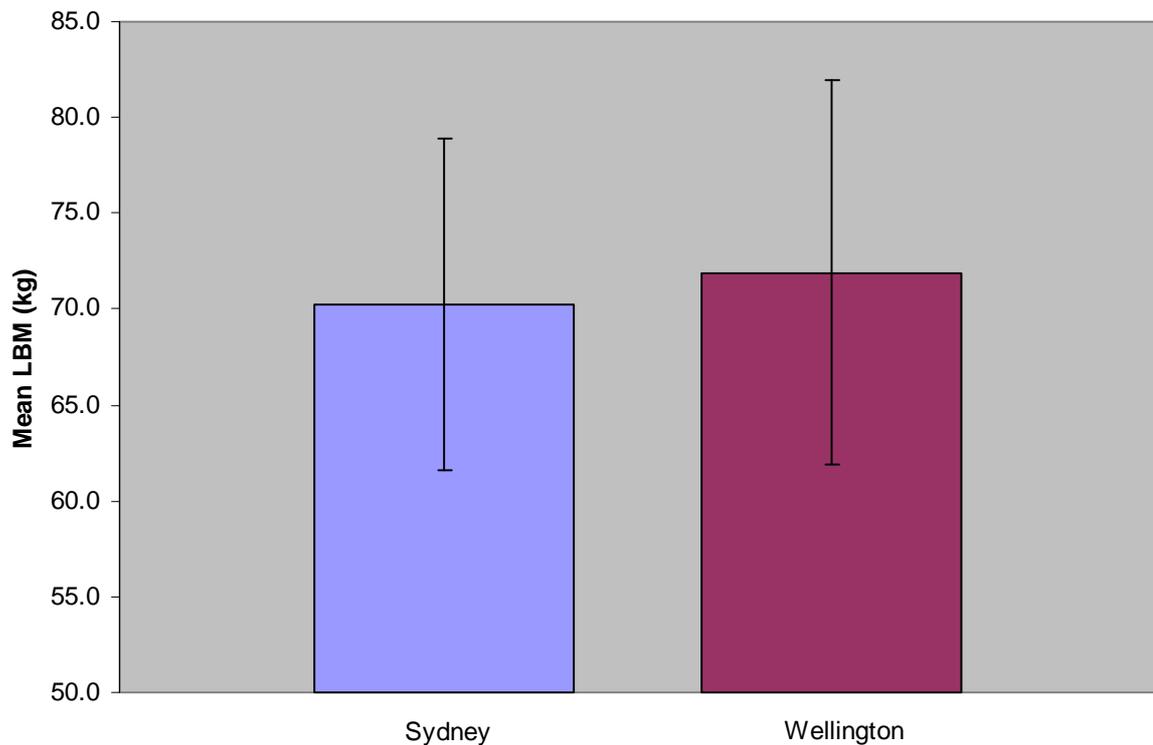
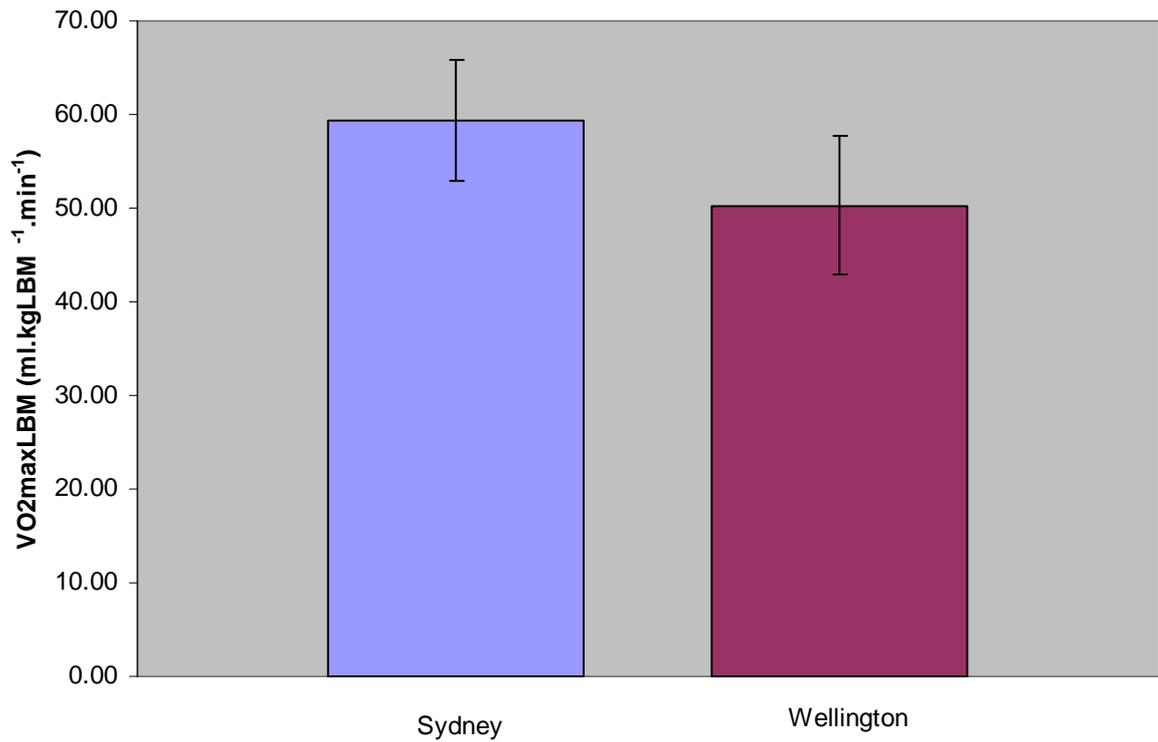
Figure 3 – Group mean of lean body mass (LBM) in Sydney and Wellington study

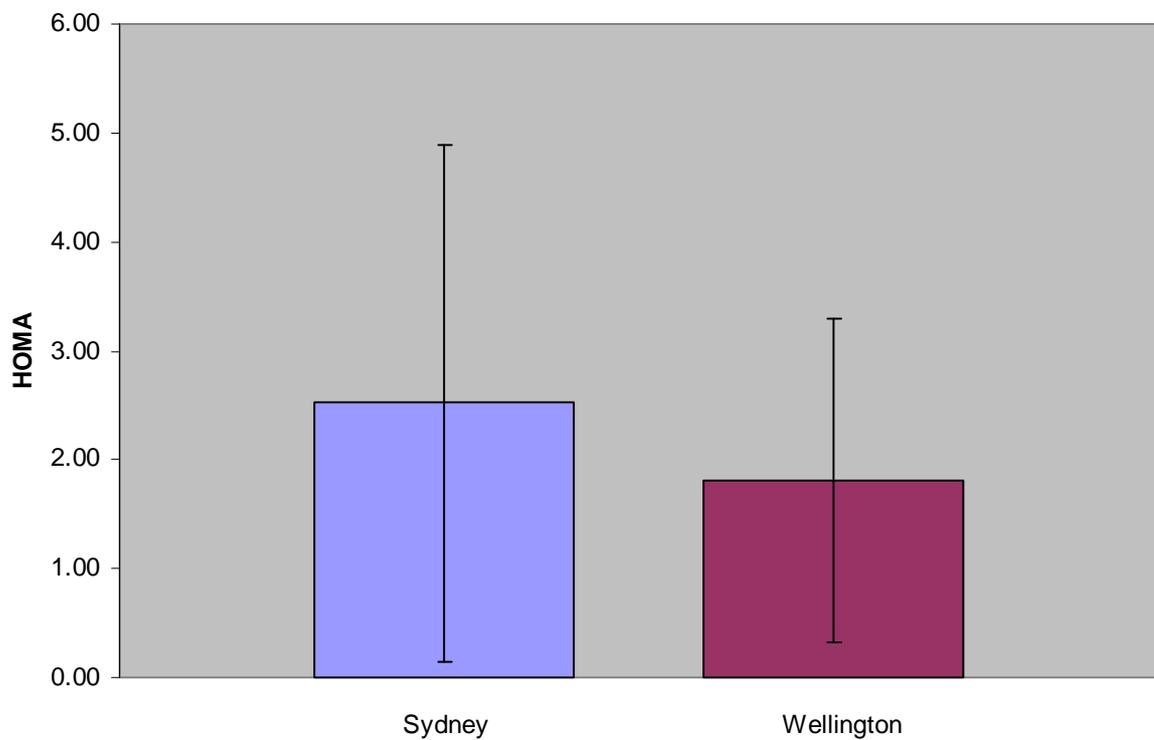
Figure 4 – Group mean for $VO_2\text{maxLBM}$ in Sydney and Wellington study

5.3.2 Blood biochemistry

HOMA-IR decreased along with fasting insulin from 75.2 ± 64.9 to 58.3 ± 46.6 and fasting glucose from 5.1 ± 0.5 to 4.9 ± 0.3 . While 2hrIns increased from 117.3 ± 76.8 to 178.1 ± 226.7 along with 2hrGluc from 4.1 ± 0.9 to 4.3 ± 1.1 . None of the changes in blood biochemistry were statistically significant ($p > 0.05$).

Table 5-3 – Blood Biochemistry within group

	Sydney Study		Wellington Study		Paired t-Test
	Mean	SD	Mean	SD	<i>p</i>
HOMA-IR	2.5	2.4	1.8	1.5	0.055
FastIns (pmol.L⁻¹)	75.2	18.7	58.3	46.6	0.068
FastGluc (mmol.L⁻¹)	5.1	0.5	4.9	0.3	0.111
2hrIns (pmol.L⁻¹)	117.3	76.8	178.1	226.7	0.395
2hrGluc (mmol.L⁻¹)	4.1	0.9	4.3	1.1	0.593

Figure 5 – Group mean for HOMA-IR in Sydney and Wellington study

5.3.3 Multiple regression analysis

Simultaneous multiple regression analysis was performed with the results of the Wellington and Sydney study combined using the 'testing date', i.e. Wellington or Sydney, as an independent variable.

The simultaneous multiple regression analysis showed that 84% of the variance in HOMA-IR could be explained by %BF ($p < 0.001$), $VO_2\text{maxLBM}$ ($p = 0.013$) and testing date ($p = 0.027$) combined. Similarly, 86% of the variation in FastIns could be explained by %BF, $VO_2\text{maxLBM}$ ($p < 0.05$) and testing date which was marginally significant ($p = 0.051$). Sixty percent of FastGluc variance could be attributed to $VO_2\text{maxLBM}$ and testing date ($p < 0.05$), but %BF was not significant ($p > 0.05$). $VO_2\text{maxLBM}$ was the only significant predictor of 2hrIns.

When multiple regression analysis was performed on the two studies separately, %BF ($p = 0.004$) and $VO_2\text{maxLBM}$ ($p = 0.041$) were significant contributors to the variation in HOMA-IR and FastIns in the Wellington study, although $VO_2\text{maxLBM}$ was marginally significant ($p = 0.054$). In the earlier Sydney study however (including only the 12 participants re-tested in Wellington), only %BF was a significant contributor to the variation in HOMA-IR and FastIns. No other significant relationship existed between the independent variables of %BF and $VO_2\text{maxLBM}$, and the other dependent variables analyzed (FastGluc, 2hrIns, 2hrGluc) (See Appendix **Table 0-5**)

Table 5-4 – Multiple regression analysis for combined results

Dependant variable	Independent variable	<i>r</i>	<i>r</i>²	<i>t</i>	<i>P</i>
HOMA-IR	-	0.844	0.712	-	<0.001
	<i>% body fat</i>	-	-	5.001	<0.001
	<i>VO₂maxLBM</i>	-	-	-2.712	0.013
	<i>Testing date</i>	-	-	-2.392	0.027
Fasting serum insulin	-	0.863	0.745	-	<0.001
	<i>% body fat</i>	-	-	5.804	<0.001
	<i>VO₂maxLBM</i>	-	-	-2.496	0.021
	<i>Testing date</i>	-	-	-2.077	<u>0.051</u>
Fasting serum glucose	-	0.602	0.362	-	0.027
	<i>% body fat</i>	-	-	-1.732	0.099
	<i>VO₂maxLBM</i>	-	-	-2.787	0.011
	<i>Testing date</i>	-	-	-3.095	0.006
2hr serum insulin	-	0.690	0.477	-	0.008
	<i>% body fat</i>	-	-	1.500	0.151
	<i>VO₂maxLBM</i>	-	-	-2.784	0.012
	<i>Testing date</i>	-	-	-0.442	0.664
2hr serum glucose	-	0.527	0.277	-	0.970
	<i>% body fat</i>	-	-	-1.159	0.261
	<i>VO₂maxLBM</i>	-	-	-2.601	0.018
	<i>Testing date</i>	-	-	-0.951	0.354

5.4 Discussion

The purpose of this study was to observe the relationship between adiposity, aerobic fitness and markers of insulin sensitivity in a group of healthy Māori men, and investigate whether those relationships changed over time.

Both the separated and combined results from Sydney and Wellington showed that %BF predicts fasting insulin levels and HOMA-IR values in young, healthy Māori men. This finding is consistent with studies in non-Māori, which have shown that body composition is associated with insulin sensitivity (Bogardus, Lillioja, Mott, Hollenbeck, & Reaven, 1985; Yki-Jarvinen & Koivisto, 1983). Bogardus et al. (Bogardus, Lillioja, Mott, Hollenbeck, & Reaven, 1985) point out that early reports of this relationship may have been misleading due to the indirect methods which essentially infer body composition, such as BMI and skin-fold measurement, employed to assess body composition. In fact, although the association between body fat and insulin sensitivity is commonly assumed, the use of valid methods to measure body composition, when observing its relationship to insulin sensitivity, has rarely been used. Consequently, body composition in this study was measured using hydrodensitometry, which is termed the 'gold standard' method to measure body composition. Then again, this method is not practical for large scale studies due to the time and skill required to perform the test, nor is it appropriate for certain populations, such as the obese or elderly. For this reason, development of appropriate techniques to measure, rather than infer body composition, could prove valuable in early identification of Māori that are at risk of type-2 diabetes; before glucose intolerance occurs. On the other hand, because body fat distribution is more closely related to insulin sensitivity than body fat percentage, methods of body composition measurement should actually focus on quantifying abdominal/visceral fat independent of subcutaneous fat. In this regard, hydrodensitometry does not take into account body fat

distribution and therefore %BF derived from this method is made up in large part by subcutaneous fat stores. Accordingly, a simple measure of abdominal adiposity, such as waist circumference, may provide a simple and more effective way of predicting diabetes risk. Accordingly, Bell et al. found that the combination of measuring waist circumference and blood triglycerides was a simple and accurate method of predicting insulin sensitivity in Māori (Bell, McAuley, Mann, Murphy, & Williams, 2004). For that reason, this study would have benefited from the inclusion of waist circumference measures.

A second major finding of the present study is that the time between the first and second tests (a representation of ageing, albeit small) had a significant effect upon HOMA-IR, FastIns and FastGluc ($p < 0.05$), revealing a relationship between ageing and insulin sensitivity in this cohort. Surprisingly however, the effect of age on insulin sensitivity was not a detrimental effect i.e. increased HOMA-IR, fasting insulin etc. In fact FastIns, FastGluc and HOMA-IR actually decreased, although the decrease was not significant for FastGluc ($p = 0.111$), approached significance for FastIns (0.068) and was only marginally significant for HOMA-IR ($p = 0.055$). This is in contrast to most epidemiological and physiological studies, which indicate that aging is associated with insulin resistance and diabetes (Fink, Kolterman, Griffin, & Olefsky, 1983; Ministry of Health, 2008; Rowe, Minaker, Pallotta, & Flier, 1983). On the other hand, although type-2 diabetes was originally thought to be a disorder associated with ageing, hence the name 'mature/adult onset diabetes', research now shows that there is no association between age and insulin sensitivity after accounting for differences in %BF and $VO_2\text{max}$ (Bogardus, Lillioja, Mott, Hollenbeck, & Reaven, 1985). Although my results could be confounded by technical error associated with collection and analysis of repeated blood samples, this is not likely a factor in this study as blood was collected and analyzed in the same location (Aotea Pathology, Wellington) for both arms of the study (See section 5.2.3.1).

Therefore, in these participants, the improvement in insulin sensitivity with age is likely a result of the significant increase in lean body mass within this cohort. Because muscle is a major site of insulin-mediated glucose disposal in the body, an increase in muscle mass would improve whole-body insulin sensitivity, and thus the glycaemic response to a glucose load. The reduction in relative fat mass in this cohort, albeit small, might also improve insulin sensitivity.

In these studies (study 1 and 2 of the thesis), fasting insulin/glucose, 2 hour insulin/glucose and HOMA-IR, have been used to represent 'insulin sensitivity'. Because these measures do not give a clear understanding of the dynamic response of glucose to insulin secretion over time, more precise methods and models, such as the glucose/insulin clamp and frequently sampled glucose tolerance test, have been advocated in lab-based studies (Breda, Cavaghan, Toffolo, Polonsky, & Cobelli, 2001; Caumo, Bergman, & Cobelli, 2000; DeFronzo, 1979). While these methods are more precise measures of 'insulin sensitivity', the simple measures (fasting insulin, HOMA-IR) used in this study are currently the standard diagnostic tool for insulin resistance, glucose intolerance and diabetes in the clinical setting and are likely more acceptable for participants than methods involving regular measurement (See section 2.3.3).

On another note, $VO_2\text{maxLBM}$ was also a significant predictor of HOMA-IR in the Wellington study but not in the Sydney study. However, when the results of all 24 participants from the original Sydney study are referred to (Stannard, Holdaway, Sachinwalla, & Cunningham, 2007), $VO_2\text{maxLBM}$ was a significant predictor of HOMA-IR in this group as well, signalling a lack of statistical power in the individual 'arms' of this repeated study. However, when the results of both 'arms' (Sydney and Wellington) were combined, and the year tested was analysed as an independent variable, $VO_2\text{maxLBM}$, was a significant

predictor of HOMA-IR, FastIns, FastGluc. This supports the findings of Bruce et al., although unlike my study where %BF ($p < 0.001$) was a more significant contributor than $VO_{2maxLBM}$ ($p = 0.013$), they showed that oxidative capacity and VO_{2max} were even better predictors of insulin sensitivity than adiposity (C. R. Bruce et al., 2003). This could be due to the difference in methods used. Bruce et al. expressed VO_{2max} relative to total body weight; whereas VO_{2max} in my study was expressed relative to lean body mass only ($VO_{2maxLBM}$).

During exercise, muscle is the main tissue for energy utilization and oxygen consumption within the body, whereas adipose tissue does not contract and consumes relatively little oxygen. Therefore, expressing VO_{2max} relative to total body weight, as is common place in exercise physiology, underestimates the relative 'fitness' of the muscle. What's more, considering the role of body fatness upon insulin sensitivity, including fat in the measurement of relative aerobic capacity could confound results. Alternatively, expressing VO_{2max} relative to lean body mass alone represents, in part, the maximal oxidative capacity of the muscle. Thus, in this study, measurement of relative aerobic capacity was made independent of body fatness. The only other study that I know of which investigates these relationships, using VO_{2max} relative to lean body mass ($VO_{2maxLBM}$) as a measure of aerobic fitness, found that $VO_{2maxLBM}$ was not a significant predictor of insulin sensitivity at all (Coon, Bleecker, Drinkwater, Meyers, & Goldberg, 1989). However, Coon et al. used a much older, cohort (46-73 years old), which likely had a very different ethnic background and much larger range of ages than my Māori participants. In contrast, on two separate occasions, $VO_{2maxLBM}$ has been shown to be a significant predictor of insulin sensitivity in a cohort of Māori men. These findings further support the possibility of using VO_{2max} , relative to lean body mass, as a non-invasive way of identifying Māori at risk of insulin resistance, who do not yet show impaired glycaemia.

Another interesting finding from this study was the degree to which VO_2max decreased between tests. Maximal oxygen consumption (VO_2max) reportedly decreases with age, and the significant point of decline within sedentary individuals occurs when people reach their 20s and 30s (Hawkins & Wiswell, 2003). Accordingly, in the present study aerobic capacity relative to lean body mass, decreased significantly between testing sessions (over 2-3 years) within this cohort of Māori men, all of which were in their 20s or 30s. Although one participant had a 21% increase in VO_2maxLBM , the rest of the cohort (minus the one participant with the increase) had an average decline in VO_2maxLBM of 18%. This result is quite dramatic considering that researchers propose the age-related decline in VO_2max to be just 10% per decade (Buskirk & Hodgson, 1987; Hawkins & Wiswell, 2003). LBM within the group, which actually increased from the first study, was the only other variable which had a significant change between tests ($p < 0.05$). Although this increase in LBM would lower VO_2maxLBM calculation and partially explain the observed decrease in VO_2maxLBM , even absolute VO_2max decreased by 16% on average in this group (not including the participant who showed an increase). Taken at face value, these findings suggest that young Māori men may have a greater rate of decline in oxidative capacity with age when compared with other populations. Nevertheless, this cohort's level of physical activity in the years between testing sessions was not recorded, so it is difficult to explain this marked decrease in aerobic capacity. Furthermore, whilst the method of gas composition analysis was the same between both the Sydney and Wellington arms of the study, the method of assessing gas volume was different. Douglas bags were used in the Sydney study and a turbine was used in the Wellington study. Both methods were calibrated, but it is possible that the observed reduction in VO_2max could be explained by this technical difference.

Since insulin sensitivity is positively correlated to aerobic fitness, it is interesting that HOMA-IR did not significantly increase as $VO_2\text{maxLBM}$ declined. However, as discussed previously, the increase in lean body mass and small decrease in relative fat mass likely counteracted any possible decrease in aerobic capacity, and ageing. There are a number of applications from these findings. Firstly, in contrast to common belief that cardiovascular exercise is the most appropriate mode of training for ‘metabolic health’, exercise such as resistance training which is aimed at increasing muscle mass (hypertrophy) without necessarily improving aerobic fitness, may be just as effective in improving and maintaining metabolic health in Māori. This is important considering that some may find it difficult or unacceptable to perform particular types of cardiovascular exercise. Secondly, in the short-term at least, the supposedly adverse effects of ageing on insulin sensitivity can be prevented or even reversed with the maintenance or improvement of lean body mass.

5.4.1 Chapter Summary

This study strengthens the findings of the original study by Stannard et al. (Stannard, Holdaway, Sachinwalla, & Cunningham, 2007), and shows that even over time, a relationship exists between adiposity, aerobic fitness and insulin sensitivity in Māori men. My results have also shown that the detrimental effects of age and reduced aerobic fitness upon ‘metabolic health’ are reduced with increased muscle mass and reduced body fat. Clearly however, further studies of this relationship in both Māori and non-Māori would make the observations of this study stronger if a larger sample size can be employed.

6 Study 2: Aerobic capacity and adiposity as predictors of fasting insulin concentration: A comparison between glucose tolerant Māori and non-Māori men.

6.1 Introduction

When compared with New Zealanders of European ancestry, Māori in New Zealand are disproportionately afflicted by diabetes and its precursor condition insulin resistance (David Simmons & Thompson, 2004). Physiological characteristics of an individual, such as skeletal muscle fibre type proportion (Lillioja et al., 1987a), aerobic capacity (C. R. Bruce et al., 2003), and body composition (Bogardus, Lillioja, Mott, Hollenbeck, & Reaven, 1985), are all associated with risk of insulin resistance. It is thought that these physiological variables all impact on whether there is accumulation of lipid, and subsequent impairment of insulin-mediated glucose disposal, within liver, and particularly, skeletal muscle (Seppälä-Lindroos et al., 2002; Stannard & Johnson, 2004).

On the other hand, population-based studies have identified lower standards of employment, education, and living standards as contributors to an increase in diabetes prevalence (Ministry of Health, 2004b). When taken in isolation, the latter approach suggests that Māori, who are overrepresented in the lower deciles of socioeconomic status, have a lower degree of 'metabolic' health because they are exposed to a lifestyle that is conducive to obesity and insulin resistance. Alternatively, it is possible that a physiological disposition for lipid

accumulation and/or reduced lipid turnover within Māori predisposes this population to greater risk of obesity and metabolic disorder.

Separating socioeconomic from physiological factors is difficult however, because any phenotype, including that which results in diabetes, develops from the interaction between inherent traits and environmental factors (Neil Pearce, Foliaki, Sporle, & Cunningham, 2004). Furthermore, comparing Māori and European participants in a physiological study can be difficult as ethnicity is a self identified trait and not necessarily a genetic or biological characteristic. On the other hand, because epidemiological data is also based on self-identification of ethnic identity, it would be expected that a comparative study of physiological factors between two different self-identified ethnic groups would produce similar trends to epidemiological data. Nevertheless, an understanding of which physiological characteristics may be associated with increased risk of developing insulin resistance and diabetes may prove useful in preventing insulin resistance and prescribing treatment.

Previous research has identified both aerobic capacity ($VO_2\text{max}$) of the lean body mass, and body adiposity independently, as predictors of insulin resistance in young Māori men (Stannard, Holdaway, Sachinwalla, & Cunningham, 2007). Others have also shown the significance of adiposity and associated increases in blood lipids in predicting insulin sensitivity in Māori (K. McAuley et al., 2001). Aerobic capacity is, in part, a function of the oxidative capacity of the muscle and thus also its ability to utilize (oxidise) lipids. Accordingly, oxidative capacity of skeletal muscle and resting whole body lipid oxidation rates have been shown to be significant predictors of whole body insulin sensitivity (Goodpaster, He, Watkins, & Kelley, 2001).

Regardless of the epidemiological trends previously discussed, Māori actually appear to be more physically active than New Zealanders of European Origin (NZEO) (Utter, Scragg, Schaaf, & Fitzgerald, 2006), and sporting participation rates in Māori are high (Chadwick & Palmer, 2006). Furthermore, Māori have less adiposity when compared to NZEO of a similar BMI (Swinburn, Ley, Carmichael, & Palnk, 1999), and even at a similar %BF, Māori are still more insulin resistant than NZEO (K. McAuley, Williams, Mann, Goulding, & Murphy, 2002). These data beg the question as to whether the observed relationship between physical fitness, adiposity and insulin sensitivity exist in Māori and non-Māori. Understanding these relationships better, may assist in shedding light upon the reasons why Māori are more predisposed to developing type-2 diabetes in New Zealand. To date, only one published study has shown that the relationship between aerobic capacity, body composition and insulin sensitivity exists in Māori (Stannard, Holdaway, Sachinwalla, & Cunningham, 2007). Additionally, I have shown that this relationship is consistent over time (see Chapter 5). Nevertheless, neither of these studies tried to compare these relationships between two ethnic groups.

Thus, the purpose of this study was to investigate whether the relationships observed between %BF, aerobic capacity and blood markers of insulin sensitivity, differed between Māori and non-Māori men.

6.2 Methods

6.2.1 Study Design

The methods used in this study, including the measurement of blood biochemistry, aerobic capacity and body composition, were identical to those in Study 1. However, this was a cross-sectional study rather than a repeated measures study, including a non-Māori cohort for comparative purposes.

6.2.2 Participants

Forty eight healthy men (31 Māori, 17 non-Māori) aged 28 ± 5 (mean \pm SD) years old. All participants answered a health questionnaire (see chapter 5) and had no cardiovascular, respiratory or metabolic disorders. All participants were randomly selected from employment, university or church networks.

Participants were asked to self-identify their ethnicity. All those who were included as Māori identified as such, while in the non-Māori group all but one participant (Asian) identified as European. Pacific Islanders were excluded from all studies in this body of research.

Table 6-1 – Participant physical characteristics

	Entire Group		Māori		Non-Māori	
	n = 48		n = 31		n = 17	
	<i>Mean</i>	<i>SD</i>	<i>Mean</i>	<i>SD</i>	<i>Mean</i>	<i>SD</i>
Age (yrs)	27.9	5.40	28.5	5.5	26.7	5.1
Height (m)	1.80	0.07	1.80	0.07	1.79	0.07
Weight (kg)	87.8	17.2	93.7	17.4	77.0	10.4
BMI (kg/m²)	27.1	4.7	28.8	4.8	24.1	2.7

6.2.3 Statistical Analyses

Independent samples t-tests were used to identify any significant differences in the variables measured, between the Māori and non-Māori cohort. Simultaneous multiple regression was performed to ascertain whether the dependant variables relating to insulin sensitivity (HOMA-IR, Fast-Ins, 2hr-Ins, Fast-Gluc, 2hr-Gluc), could be significantly predicted by the independent variables (%BF, VO₂maxLBM). Ethnicity was also included as an independent variable, to determine the effect of ethnicity on these relationships. SPSS version 10 was used for all statistical analyses.

6.3 Results

Although 61 participants took part in this study (39 Māori, 22 non-Māori), I was unable to collect accurate VO₂max measures for 13 of these subjects (8 Māori and 5 non-Māori) so data are reported only for 48 participants.

6.3.1 Body Composition

The Māori group had a significantly greater mean weight (93.7kg Māori, 77.0kg non-Māori), BMI, %BF and LBM than the non-Māori group ($p < 0.01$). Neither age nor height were significantly different between groups ($p > 0.05$).

Table 6-2 – Body composition (percent body fat and total lean body mass)

	Entire Group		Māori		Non-Māori	
	Mean	SD	Mean	SD	Mean	SD
%BF	19.7	7.0	21.5	7.4	16.5	5.0
LBM (kg)	69.3	9.3	72.4	9.2	63.8	6.5

6.3.2 Aerobic capacity

The non-Māori group had a significantly greater $VO_2\max$ LBM than the Māori group (60.8 L/min/kg l.b.m and 53.0 L/min/kg l.b.m: $p < 0.05$). The non-Māori group also had a higher absolute $VO_2\max$ than the Māori (3853 ml/min and 3805 ml/min), though this was not significant ($p > 0.05$).

Table 6-3 – Aerobic capacity (absolute and relative to lean body mass)

	Entire Group		Māori		Non-Māori	
	Mean	SD	Mean	SD	Mean	SD
$VO_2\max$ (ml)	3822	507	3805	505	3853	525
$VO_2\max$LBM (ml.kgLBM⁻¹.min⁻¹)	55.8	8.6	53.0	6.9	60.8	9.3

6.3.3 Blood Biochemistry

Eight of the 48 participants did not receive 2hr insulin blood samples, and 5 did not receive 2hr glucose samples. None of the blood markers measured were significantly different between the Māori and non-Māori group ($p>0.05$).

Table 6-4 – Blood Biochemistry

	Entire Group			Māori			Non-Māori		
	Mean	SD	n	Mean	SD	n	Mean	SD	n
HOMA-IR	1.67	1.28	48	1.88	1.32	31	1.46	1.23	17
Fast Ins (pmol.L⁻¹)	55	38.7	48	60	39.3	31	47	37.2	17
Fast Gluc (mmol.L⁻¹)	4.8	0.4	48	4.8	0.5	31	4.8	0.2	17
2hr Ins (pmol.L⁻¹)	143	141.3	40	161	165.0	26*	110	75.5	14*
2hr Gluc (mmol.L⁻¹)	4.3	1.1	43	4.4	1.1	27*	4.0	1.0	16*

* 8 participants did not have 2hr insulin samples, 5 did not have 2hr glucose

6.3.4 Statistical Analysis

The results of the simultaneous multiple regression analyses are found in **Table 6-5**. While ethnicity was used as an independent variable (along with %BF and VO₂maxLBM) in the initial multiple regression analysis of the entire group, it was not a significant predictor of any of the dependant variables ($p>0.05$). The results of the entire group seen in **Table 6-5** are from a simultaneous multiple regression analysis which used only %BF and VO₂maxLBM as independent variables, ethnicity was excluded.

Table 6-5 – Simultaneous multiple linear regression analysis

		Entire Group					Māori					non-Māori				
Dependant variable	Independent variable	<i>r</i>	<i>r</i> ²	<i>t</i> -value	<i>P</i> -value	<i>n</i>	<i>r</i>	<i>r</i> ²	<i>t</i> -value	<i>P</i> -value	<i>n</i>	<i>r</i>	<i>r</i> ²	<i>t</i> -value	<i>P</i> -value	<i>n</i>
<i>HOMA-IR</i>	-	0.55	0.30	-	<0.001	<i>n</i> =4	0.66	0.44	-	<0.001	<i>n</i> =3	0.34	0.11	-	0.423	<i>n</i> =1
	% body fat	3	6	2.902	0.006	8	6	3	3.465	0.002	1	0	6	0.396	0.698	7
	VO ₂ maxLBM	-	-	-3.172	0.003	-	-	-	-3.222	0.003	-	-	-	-1.354	0.197	-
<i>Fasting insulin serum</i>	-	0.57	0.33	-	<0.001	<i>n</i> =4	0.69	0.48	-	<0.001	<i>n</i> =3	0.35	0.12	-	0.386	<i>n</i> =1
	% body fat	9	6	3.317	0.002	8	4	1	4.018	<0.001	1	7	7	0.435	0.670	7
	VO ₂ maxLBM	-	-	-3.194	0.003	-	-	-	-3.151	0.004	-	-	-	-1.428	0.175	-
<i>Fasting glucose serum</i>	-	0.27	0.07	-	0.179	<i>n</i> =4	0.37	0.13	-	0.123	<i>n</i> =3	0.20	0.04	-	0.732	<i>n</i> =1
	% body fat	1	4	-1.189	0.241	8	3	9	-1.174	0.250	1	9	4	0.548	0.592	7
	VO ₂ maxLBM	-	-	-1.547	0.129	-	-	-	-1.771	0.087	-	-	-	-0.721	0.483	-
<i>2hr serum insulin</i>	-	0.56	0.31	-	0.001	<i>n</i> =4	0.67	0.45	-	0.001	<i>n</i> =2	0.42	0.17	-	0.340	<i>n</i> =1
	% body fat	0	4	2.436	0.020	0	3	2	2.827	0.010	6	2	8	-0.814	0.433	4
	VO ₂ maxLBM	-	-	-3.141	0.003	-	-	-	-3.285	0.003	-	-	-	-1.049	0.317	-
<i>2hr serum glucose</i>	-	0.26	0.07	-	0.225	<i>n</i> =4	0.34	0.11	-	0.219	<i>n</i> =2	0.27	0.07	-	0.597	<i>n</i> =1
	% body fat	8	2	0.946	0.350	3	5	9	1.177	0.251	7	6	6	-1.023	0.325	6
	VO ₂ maxLBM	-	-	-1.398	0.170	-	-	-	-1.293	0.208	-	-	-	0.166	0.871	-

Multiple regression analysis showed that both %BF and $VO_2\text{maxLBM}$ were significant predictors of HOMA-IR, Fast-Ins and 2hr-Ins in the entire group as well as the Māori cohort. %BF and $VO_2\text{maxLBM}$ were not significant predictors of any of the dependant variables (HOMA-IR, Fast-Ins, Fast-Gluc, 2hr-Ins, 2hr-Gluc) in the non-Māori cohort however.

6.4 Discussion

Population studies have shown that Māori are more obese and insulin resistant than NZEO (K. McAuley et al., 2002; Ministry of Health, 2008). Since Māori are also overrepresented in lower socioeconomic levels, it is believed that the health disparity between Māori and NZEO is due to environmental factors rather than a physiological disposition. Accordingly, Pearce et al. suggest that historical, cultural and socioeconomic factors, which influence lifestyle and access to health care, are responsible for ethnic differences in health (Neil Pearce, Foliaki, Sporle, & Cunningham, 2004). On the other hand, it appears that Māori are more insulin resistant than NZEO even after adiposity is adjusted for (K. McAuley, Williams, Mann, Goulding, & Murphy, 2002), suggesting an inherent disposition toward insulin resistance. While differences in insulin sensitivity and obesity between Māori and NZEO are clear from population studies, almost no studies have investigated how ethnicity may alter the relationship between the physiological factors related to such metabolic disorders. Thus, the purpose of this study was to investigate whether the relationship between aerobic capacity, body composition and markers of insulin sensitivity differed between Māori and non-Māori.

We were able to show that $VO_2\text{maxLBM}$ and %BF independent of each other are both significant predictors of blood markers associated with insulin sensitivity in a healthy group of young, adult New Zealand males. When this group was split into two self-identified ethnic groups, $VO_2\text{maxLBM}$ and %BF were only predictors of these blood markers in the Māori

group, with no significant interactions seen in the non-Māori group. While the relationship observed in the Māori cohort is in accordance with the only published study of these relationships done in Māori (Stannard, Holdaway, Sachinwalla, & Cunningham, 2007), the absence of a relationship between insulin sensitivity and $VO_2\text{maxLBM}/\%BF$ in the non-Māori group is a surprise. Interestingly, Bogardus et al. (Bogardus, Lillioja, Mott, Hollenbeck, & Reaven, 1985) showed that aerobic capacity and %BF were significantly correlated with insulin sensitivity in Caucasian and Native American men, but that these associations were only marginally statistically significant. What's more, Bruce et al. (C. R. Bruce et al., 2003) reported that $VO_2\text{max}$ and oxidative capacity were even better predictors of insulin sensitivity than percentage body fat. However, Coon et al. note that most studies using aerobic capacity ($VO_2\text{max}$) to predict insulin sensitivity have reported $VO_2\text{max}$ relative to total body weight rather than lean mass, so in these studies the influence of $VO_2\text{max}$ is not entirely independent of body fatness (See Section 5.4). In contrast, Coon et al. showed that when $VO_2\text{max}$ was expressed relative to lean body mass, it was not significantly correlated with markers of insulin sensitivity in a cohort of American men (Coon, Bleecker, Drinkwater, Meyers, & Goldberg, 1989).

Although part of the result may be due to a lack of statistical power in the non-Māori cohort, Coon et al. found no significant interaction between $VO_2\text{maxLBM}$ and insulin sensitivity in a cohort over four times the size of the non-Māori group I tested (Coon, Bleecker, Drinkwater, Meyers, & Goldberg, 1989). However, this does not explain why no significant relationship was observed between %BF and insulin sensitivity in my non-Māori group, as has been shown in these, and other studies (Boden, Chen, DeSantis, & Kendrick, 1993). On the other hand, our non-Māori cohort were fitter (had a higher $VO_2\text{maxLBM}$) and leaner (lower %BF) than the Māori group and the lack of data spread in the non-Māori cohort was a weakness in

this study. Thus, environmental factors such as physical activity levels or nutritional status, rather than ethnicity, may explain part of the result of this study. With this in mind, recording physical activity levels of participants prior to testing would have been ideal. Another possible confounder in regard to measurement of insulin sensitivity in this study was that the amount of glucose administered during the OGTT was the same (75g) for all participants regardless of body weight and LBM. Because of the role of skeletal muscle in the uptake of glucose from the blood, a greater total LBM should increase glucose disposal and therefore confound postprandial indices of insulin sensitivity (2hr insulin, 2hr glucose). On the other hand, an OGTT is the standard diagnostic test for insulin sensitivity in New Zealand and is used to identify insulin resistance/glucose tolerance in Māori and non-Māori. Furthermore, if the fasting insulin values, a measure unaffected by the glucose load, are analyzed against the independent variables of this study alone, the differences between the Māori and non-Māori cohort still remain. Indeed, fasting insulin has been shown to be as effective as HOMA-IR in predicting insulin sensitivity in Māori (Bell, McAuley, Mann, Murphy, & Williams, 2004) and is identified as an adequate means of measuring insulin sensitivity in glucose tolerant individuals such as the participants of this study (K. McAuley et al., 2001).

Regardless of confounding factors, the results of the present study viewed in conjunction with the findings of the previously referred to studies by Coon et al. and Bogardus et al., suggest that the relationship between aerobic capacity and insulin sensitivity may be ethnically determined. If, as the results of this study suggest, the association between aerobic capacity and insulin sensitivity is strong in Māori but not NZEO, the focus for Māori-targeted intervention for reducing diabetes risk, would obviously benefit from inclusion of physical fitness rather than simply weight loss alone. Perhaps an emphasis on physical *inactivity* as a more important risk factor should be considered in Māori.

Currently, the government vehicle of choice for increasing public physical activity levels is the ‘*Push Play*’ campaign delivered by SPARC (*Sport and Recreation New Zealand*) (SPARC., 2005). Through media and other types of community based promotion, ‘Push Play’ encourages people to engage in 30 minutes of physical activity per day (60 minutes for children), by incorporating physical activity in to everyday activities such as playing with the kids or walking to work. Similarly, the 10,000 Steps Program (and similar pedometer-based walking initiatives) promote increased physical activity by encouraging people to take at least 10,000 steps per day, often incorporated into their occupational time. Although well intentioned, it is difficult to assess the success of such campaigns among Māori. As discussed previously (see section 3.5), in order for interventions to be effective for Māori, they must be culturally appropriate for Māori, and therefore involve Māori at all levels; from planning to implementation (Ministry of Health, 2002).

Furthermore, such initiatives may motivate sedentary individuals to become more active, but the recommendations of these programs may not be sufficient to reduce adiposity or the effects of obesity and insulin resistance in Māori and others. On the other hand, the mode of exercises recommended to improve metabolic control (usually cardiovascular exercise such as walking, running, cycling, swimming etc) are difficult or unacceptable for some, particularly the already obese, to perform in public. Alternatively, resistance training has been shown to be an effective training method to improve insulin sensitivity in non-Māori (Evas & Plotnikoff, 2006; Ibanez et al., 2005; Poehlman, Dvorak, DeNino, Brochu, & Ades, 2000), and may be a more appropriate form of exercise for the obese and elderly. This is in accordance with the results of my previous study (see chapter 5), which suggests that increasing muscle mass (through resistance training), may be just as effective in improving

insulin sensitivity in Māori, as cardiovascular exercise. Resistance training can be done indoors and perhaps could form the basis of a marae-based exercise initiative.

There are limitations when comparing measures of health within separate ethnicities, as ethnicity is often more closely associated to cultural and social identity than it is to biological or genetic factors (Neil Pearce, Foliaki, Sporle, & Cunningham, 2004). Because of this, identifying what appears to be a physiological disposition to a disorder such as insulin resistance in Māori may actually be a result of a certain lifestyle adopted by those who identify as Māori. Identifying participants according to 'heritage' rather than self-identified 'ethnicity' may be more appropriate to detect genetic dispositions in health.

6.4.1 Chapter Summary

We have shown that VO₂maxLBM and %BF are predictors of insulin sensitivity, in glucose tolerant New Zealanders. When ethnicity was accounted for however, VO₂maxLBM and %BF were only predictors of insulin sensitivity in the Māori group, but not the non-Māori group. Although literature is limited in Māori, the findings of this study are in accordance with other studies involving Māori. While reduced statistical power within the non-Māori cohort may have affected the result in that group, other studies in non-Māori, have found similar results.

7 Study 3: Bioelectrical impedance: A valid method of measuring body composition in Māori, when compared to hydrodensitometry.

7.1 Introduction

Adiposity is inversely related to insulin sensitivity, and it is believed that excess lipid accumulation, or stagnancy, is responsible for disruption of the insulin signalling cascade in insulin sensitive tissues (Boden, 1997; Boden, Chen, Ruiz, White, & Rossetti, 1994; Roden et al., 1996). Likewise, population studies have identified obesity as a strong predictor of diabetes, other non-communicable diseases such as stroke and breast cancer, and all-cause mortality (Solomon & Manson, 1997). Armed with this knowledge, researchers, clinicians, and other health practitioners have sought precise and reliable methods to assess body fat percentage (%BF) in individuals, so as to predict the development of insulin resistance, diabetes and other obesity related illness.

The body mass index (BMI) was originally developed as a simple means of identifying those at risk of obesity related illnesses, with distinct “cut off” points developed to categorize the level of risk. These cut off points have come under scrutiny in both lay and scientific media, as healthy people are often mistakenly categorized as overweight or obese, while other groups are at greater risk of obesity, and its co-morbidities, at a relatively low BMI (Kosaka, Kuzuya, Yoshinaga, & Hagura, 1996; K. McAuley, Williams, Mann, Goulding, & Murphy, 2002).

Body mass index is derived from a person’s weight in relation to their height (BMI = weight (kg)/height (m²)), measures that are easily and quickly obtained in the clinical healthcare

setting. Although a direct measure of body fat percentage is a more valid predictor of health status, obtaining an accurate measure of an individual's body fat percentage can involve difficult and costly procedures.

Epidemiological evidence indicates that particular BMI ranges, defined by these cut-offs, are associated with differing levels of mortality and risk of other 'life-style' diseases (Solomon & Manson, 1997). Whether an individual's measured BMI lies within a particular range is then often extrapolated to indicate the presence of obesity-related disease or mortality risk. However, the use of BMI for such purposes makes the broad assumption that increasing BMI is a function of increasing adiposity. Whilst at a population level this is likely true, for an individual this assumption may not hold. Thus, for diagnosis at an individual level, a more direct method for estimating body fatness must be found.

A second, but important issue is that the sample from which population-based BMI risk assessment is based may not necessarily represent other populations (WHO, 1995). A case in point is the use of BMI cut offs derived from populations of European decent, to predict health status in Māori. What is considered a "healthy" BMI range for non-Māori may be different from Māori as physical characteristics, and the association with disease risk factors, probably differ significantly between these ethnic groups. For example, Asians and Indians have been shown to have more body fat than Europeans at a given BMI, while Polynesians and Māori appear to have less adiposity than Caucasians at the same BMI (Swinburn, Ley, Carmichael, & Palnk, 1999; WHO, 2004).

Hydrodensitometry, also known as hydrostatic weighing or underwater weighing (UWW), has become the 'gold standard' procedure for obtaining an accurate measure of percent body fat

(%BF). This procedure is based on the Archimedes principle of water displacement, and relies on assumptions regarding density of body tissues in order to calculate %BF (Heyward & Wagner, 2004). Accurate measurement also requires calculation of residual lung volume whilst submerged; this is then accounted for when calculating true underwater weight. Thus, hydrodensitometry is a difficult procedure to perform, especially for those who are lacking in mobility, such as the clinically obese. It is therefore impractical for use in population studies or clinical assessment in heterogeneous groups.

Bioelectrical impedance machines (BIA) on the other hand, have become more popular in recent years because they are now relatively cheap and easy to use. These machines relay a small electrical current through the body, and based on the current's impedance through tissues, an estimation of total body water (TBW) can be obtained. Water is a good conductor of electricity, so the current flows more easily through fat-free mass (FFM) which has a high water content (~73% water), while a body with more fat 'impedes' the electrical current because of fat's relatively small water content (Heyward & Wagner, 2004). From estimates of TBW, FFM (and therefore %BF) can then be predicted (See section 2.4.2.3). The whole procedure takes less than a minute and requires little expertise from the operator or demand of the individual being tested. If proven reliable, BIA could provide a practical solution for obtaining measures of body composition in population studies and clinical assessment.

Thus the purpose of this study was to investigate the reliability and applicability of BIA for measuring body composition in a Māori and non-Māori cohort, when compared with hydrodensitometry.

7.2 Methods

7.2.1 Participants

Forty eight men (28 Māori & 20 non-Māori) aged 27 ± 5 (mean \pm SD) years old participated in this study. All participants completed a written health questionnaire and were considered healthy with no cardiovascular, respiratory or metabolic disorders.

Table 7-1 – Participant physical characteristics

	Māori (n=28)		Non-Māori (n=20)		Entire Group (n=48)	
	Mean	SD	Mean	SD	Mean	SD
Age (years)	26.6	5.1	27.0	4.4	26.7	4.8
Height (M)	1.80	0.07	1.79	0.06	1.80	0.06
Weight (kg)	95.7	18.2	76.8	9.0	87.8	17.6
BMI (kg/m²)	29.4	5.1	23.9	2.4	27.1	5.0

7.2.2 Protocol

7.2.2.1 Hydrodensitometry

Methods have been described in a previous chapter (Chapter 1).

7.2.2.2 Bioelectrical impedance

BIA measures were taken 1-2 hours after a small (standard) meal (the first meal after a 10 hour fast). As BIA measurements were taken just before underwater weighing, subjects wore only the same swimsuits that were worn in the underwater weighing tank during the BIA procedure. The BIA apparatus used was the Biospace 230 INBODY by GBC Biomed NZ, Technipro PTY. This model used ‘direct segmental multifrequency’ impedance measures, and

had an '8 point tactile electrode system' to make impedance measurements i.e. two points of electrode contact per hand and two points of contact per foot. Electrode contact was made with the palms of the hands and at each thumb, as the participant grasped the electrode handles, as well as with the soles and balls of the feet, as the participant stood barefoot upon the electrode plate. The operator would then enter the participant's name, age, gender, height and weight, and the analyser would take approximately 30 seconds to make the impedance measurement and produce a printout of %BF, total body water (TBW) and segmental impedance measures from the right leg, left leg, right arm, left arm and trunk.

Participants avoided diuretics, exercise, and alcohol prior to testing; all factors which may reduce the reliability of the measurement (Heyward & Wagner, 2004).

7.2.3 Statistical Analyses

Independent samples t-tests were used to identify any significant differences in the variables measured, between the Māori and non-Māori cohort. Bivariate correlation analysis was performed, to determine the relationship between measures of percentage body fat from underwater weighing and BIA. Simultaneous multiple regression analysis was also employed to determine the effect of ethnicity on this relationship. All statistical analyses were performed using SPSS version 10, while scatter plots were made using Microsoft excel 2003.

7.3 Results

Forty eight people participated in this study (28 Māori and 20 non-Māori).

7.3.1 Body composition

The average %BF from BIA and underwater weighing, as well as the BMI of the entire group, Māori only and non-Māori only, are shown in the table below. Māori had significantly higher %BF, LBM and BMI values, compared with the non-Māori group ($p < 0.001$).

Table 7-2 – BMI and percentage body fat from underwater weighing and BIA

	Māori		Non-Māori		Whole Group	
	Mean	SD	Mean	SD	Mean	SD
BMI (kg/m^2)	29.4	5.1	23.9	2.4	27.1	5.0
%BF (UWW)	22.9	7.0	15.9	5.0	20.0	7.1
%BF (BIA)	20.6	7.9	13.6	3.7	17.7	7.3

7.3.2 Statistical Analyses

Bivariate correlation revealed that %BF obtained from the BIA apparatus was a significant predictor of %BF obtained using hydrodensitometry in the entire cohort ($P < 0.001$), the Māori cohort ($P < 0.001$) and the non-Māori cohort ($P = 0.017$). BIA values explained 77% of variation in the entire group, 76% in the Māori group and 53% in the non-Māori group.

Multiple regression analysis was also performed using ethnicity and %BF_{BIA} as independent variables to ascertain the effect of ethnicity on the relationship between %BF_{BIA} and %BF_{UWW}. Ethnicity however, had no significant effect upon this relationship ($P > 0.05$).

Figure 6 – Correlation between underwater weighing and BIA (entire cohort)

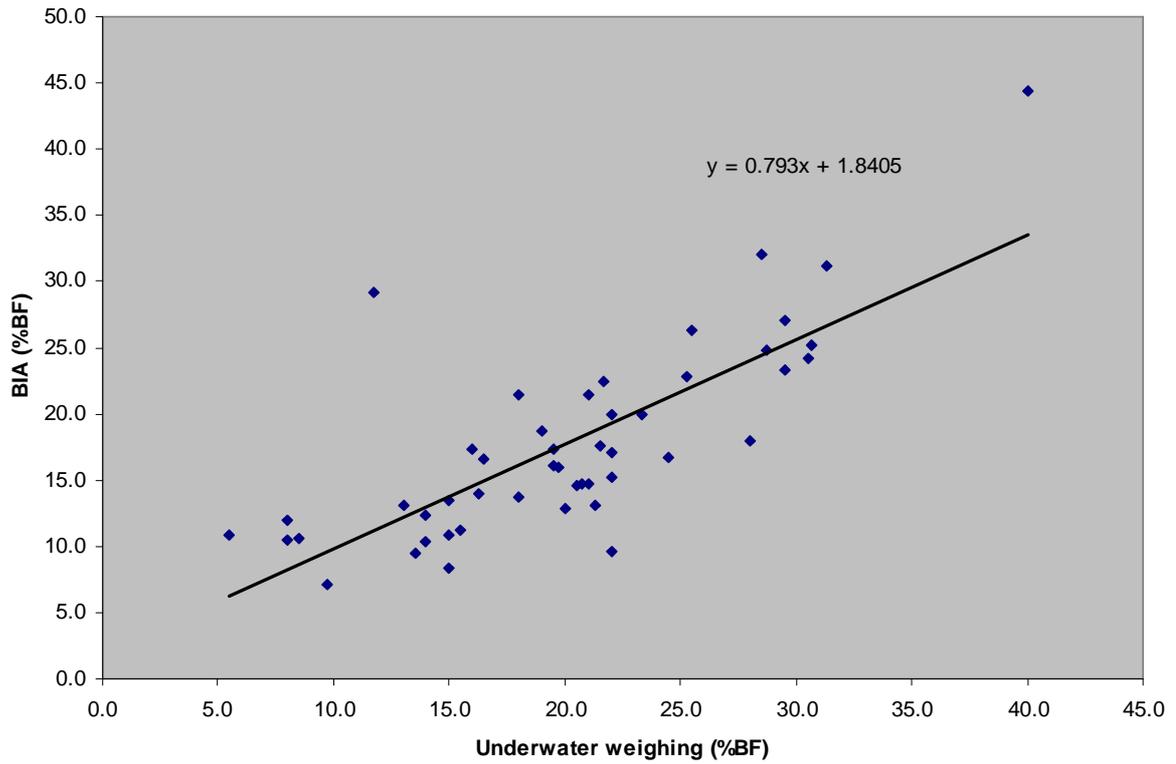


Figure 7 – Correlation between underwater weighing and BIA (Māori cohort only)

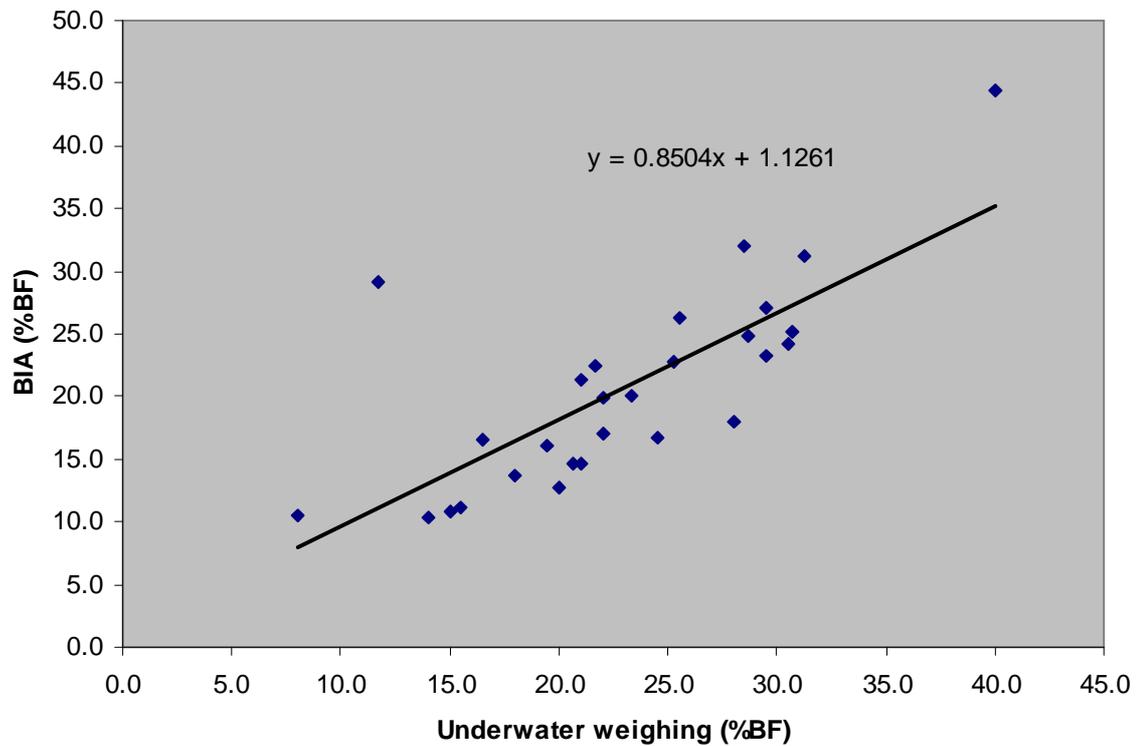
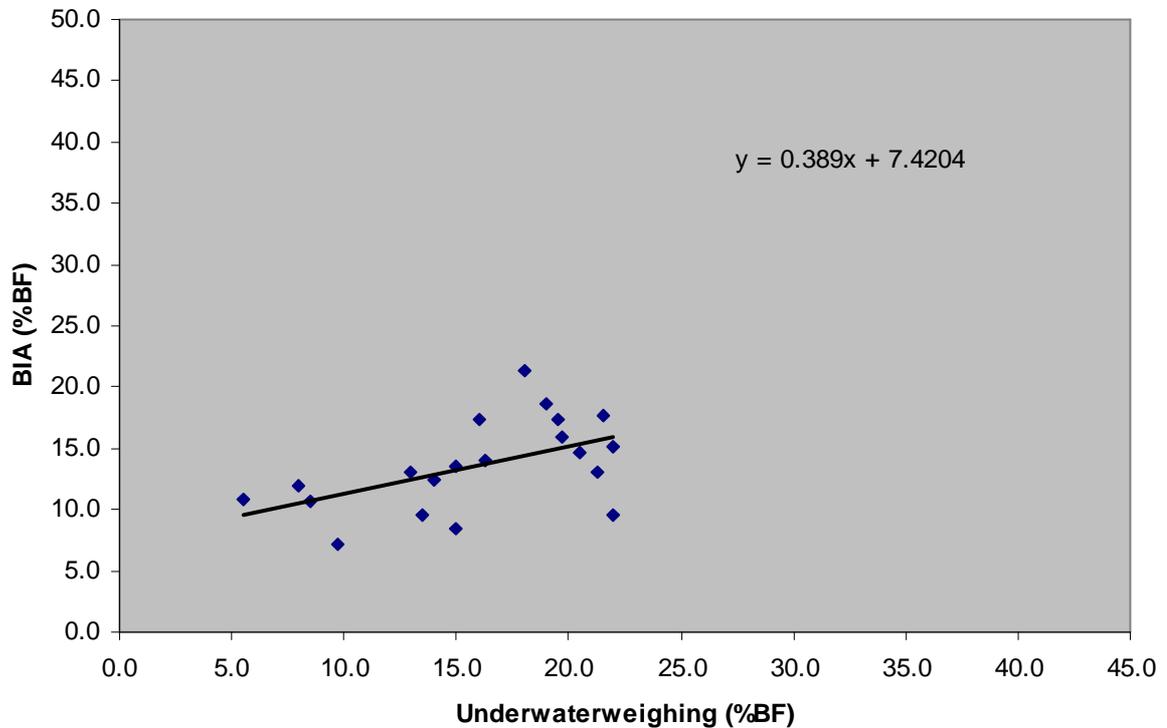


Figure 8 – Correlation between underwater weighing and BIA (non-Māori cohort only)

7.4 Discussion

Because of the association between adiposity and ‘lifestyle illnesses’ such as diabetes and cardiovascular disease, accurate measurement of body composition is important in identifying those most at risk of these disorders. Although useful to identify obesity trends in population studies, the body mass index (BMI) is not a direct measure of adiposity and can therefore under/overestimate obesity and associated health risks in certain populations. However, proven methods of body composition assessment, such as hydrodensitometry and DEXA, are not practical for population studies. On the other hand, BIA provides an easy and direct way to measure fatness, and therefore risk of insulin resistance, but its accuracy in certain populations has been questioned. Thus the purpose of this study was to investigate the

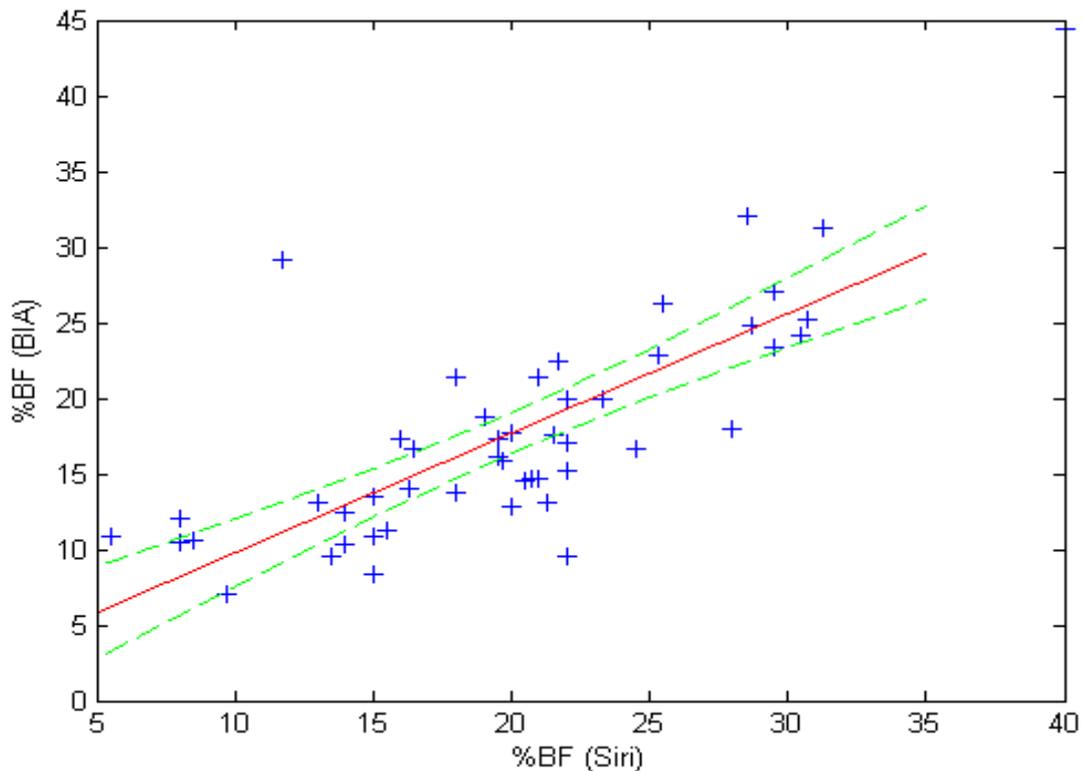
accuracy of employing BIA to measure %BF in a group of Māori and non-Māori males, when compared to hydrodensitometry.

We have shown that %BF values obtained with the BIA apparatus correlated well with %BF measures from underwater weighing. Using an earlier version of the apparatus I've used, Malavolti et al. showed that whole-body fat-free mass and appendicular lean body mass obtained from BIA, correlated well with measurements from DEXA (Malavolti et al., 2003). In conjunction with these findings, my results suggest that BIA could provide a valid and easy way to measure %BF in both Māori and non-Māori. Although the relationship between BIA and UWW seemed weaker in the non-Māori group ($r=0.76$ Māori vs. $r=0.53$ non-Māori), this is most likely due to an insufficient spread of data in this group.

Although my results indicate that it may be appropriate for obtaining population data, the use of BIA for individual assessment must be approached with some caution. While it correlated well with UWW, BIA did underestimate %BF by an average of 2.3% in all three groups tested, when compared with UWW. This inaccuracy is relatively modest compared with the findings of other studies comparing BIA to hydrodensitometry. Heath et al. revealed that BIA under predicted %BF by 5.7% in men and 9.1% in women (Heath, Adams, Daines, & Hunt, 1998). Studies have also shown that, when compared with hydrostatic weighing, BIA overestimates fatness in lean individuals and underestimates it in overweight individuals (Heath, Adams, Daines, & Hunt, 1998; Segal, Gutin, Presta, Wang, & Van Itallie, 1985). Accordingly, my study showed that BIA underestimated %BF for 21 of the 25 participants that were over 20% BF, and over estimated %BF in 4 out of the 5 'lean' participants (%BF<10%), supporting the idea that BIA underestimates %BF in overweight individuals and

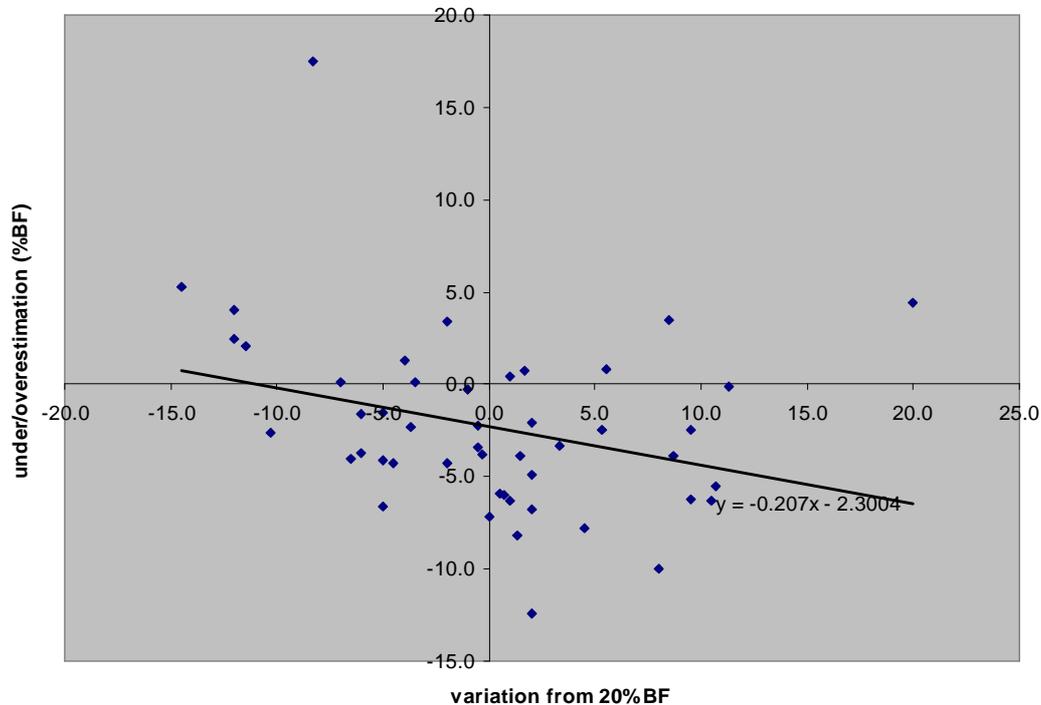
overestimated it in lean individuals (Heath, Adams, Daines, & Hunt, 1998; Segal, Gutin, Presta, Wang, & Van Itallie, 1985).

Figure 9 - Correlation between underwater weighing and BIA (entire cohort) 95% confidence intervals added



What's more, the degree to which BIA under/overestimates %BF, can be relatively large. In this study BIA under/overestimated %BF by at least 5% for 14 of the 48 participants, 3 of which were more than 10% under/overestimated. Other studies have recorded similarly large under/overestimations of %BF from BIA when compared with hydrostatic weighing (Heath, Adams, Daines, & Hunt, 1998). If technical error is responsible for these deviations, the re-breathing protocol during underwater weighing is likely responsible, rather than the BIA apparatus.

Figure 10 – The relationship between body fatness and the degree to which BIA under/overestimates percent body fat



In order to obtain an accurate measurement of %BF from underwater weighing, residual lung volume, the volume of air remaining in the lungs after maximal expiration, should be accounted for (Buskirk, 1959). Buoyancy of an individual is increased as RV increases, so that a greater RV can make a person appear lighter under water than they actually are and therefore overestimate body fatness. This suggests that if RV is measured and accounted for, the amount of air in the lungs during the measurement of underwater weight should make no difference. On the other hand, Welch and Crisp (Welch & Crisp, 1958) revealed that body density was significantly different between lung volume measured at half expiration and full expiration. If so, measuring RV at full expiration is important, but because maximal expiration can be uncomfortable underwater and is measured subjectively, measuring RV is not without its limitations. After having been underwater for up to 10 seconds with air fully

expired, many of the participants of this study found it difficult to stop themselves from inhaling, as was required of them before connection to the re-breathing apparatus. Although measures were taken to ensure that participants did not inhale before the re-breathing apparatus was in their mouths and the circuit open, it is possible that slight inhalation may have occurred which could alter the dilution and RV measurement.

Another limitation of BIA is its need for specific equations to accurately calculate %BF. In order to make BIA valid for certain ethnic groups, ethnic specific equations are required. Rising et al. showed that BIA %BF measures calculated by the software provided with the analyser they used, underestimated lean body mass in Pima Indians when compared to underwater weighing, by 5.3 ± 8.6 kg (Rising, Swinburn, Larson, & Ravussin, 1991). Similarly, Jakicic et al. found that BIA underestimated LBM in Caucasians, while overestimating LBM in African-Americans (Jakicic, Wing, & Lang, 1998). In my study however, ethnicity did not have a significant effect upon this relationship ($p > 0.05$), even though the same equation was used for both Māori and non-Māori. Furthermore, although BIA underestimated %BF when compared to UWW, the underestimation was the same in both ethnic groups (2.3%).

This study also revealed that BMI correlated well with the %BF measures obtained in the group tested. Indeed the correlation between BMI and hydrodensitometry was slightly stronger than that for BIA and hydrodensitometry in the entire group ($r = 0.791$ vs. $r = 0.773$) and the Māori group ($r = 0.775$ vs. $r = 0.756$), but very similar in the non-Māori group ($r = 0.516$ vs. $r = 0.526$). This suggests that BMI in this group of healthy Māori men is an acceptable way to identify the degree of insulin sensitivity. Although this implies that BMI may be a valid index of risk in the group which I tested, caution must still be used in applying BMI to

individual assessment as it is not a measure of adiposity, which is the actual risk factor associated with metabolic disorder.

In this study, just one type of BIA analyser was used. Studies have shown that impedance measures obtained from different BIA analysers can produce %BF measurements which differ significantly (Heyward & Wagner, 2004). While this does not affect the results of my study, it is important to understand this if different analysers are used interchangeably during population research and clinical practice. For example, if a BIA analyser is used to obtain repeated measures of body composition during a longitudinal study, the same analyser should be used. I was unable to obtain information regarding the equations this apparatus used to determine %BF, so it is questionable whether the results obtained with the Biospace 230 INBODY BIA are comparable with other machines.

The source of error, as a result of technician skill, is quite minor due to the nature of the apparatus. However, the position of electrode placement has been shown to be a major source of error in other tetra polar BIA analysers. While placement of electrodes in other methods requires technicians to place electrodes at certain points on the wrist and ankle while the participant lies supine, the specific apparatus used in this study is less intricate. Impedance with this apparatus is measured while the subject stands on the lower body electrode plates, meaning that contact with electrodes are with the soles of the feet, while upper body electrode contact comes from the palms of the hands while grasping the electrode handles. Because of this, electrode contact with the feet and hands is not completely standardized, creating another possible source of error.

Hydration and food consumption is also a source for error when using BIA. Participants in this study were not in the fasted state, as is suggested by Heyward and Wagner (Heyward & Wagner, 2004), but food consumption was within 1-2hr postprandial. This consumption time should have little effect on the BIA measurements in this study however, considering most studies have shown food consumption and hydration to have little effect on impedance in the first 2hrs post-consumption (P. Deurenberg, Weststrate, Paymans, & Van Der Kooy, 1988; M. R. Gallagher, Walker, & O'Dea, 1998). Kushner et al. conclude that eating and drinking have little effect on the impedance measures until 2-4hr post consumption (Kushner, Gudivaka, & Schoeller, 1996), although others have found consumption to effect measures at just 1hr post meal (Rising, Swinburn, Larson, & Ravussin, 1991).

Percentage body fat (%BF) obtained from BIA is based on assumptions of the impedance of certain tissues (See section 2.4.2.3). Whenever assumptions are made from one population to another, there is a possibility of error when applying the assumption to a different population. Because underwater weighing is the standard to which BIA is usually compared, and this method is based on assumptions regarding the density of certain tissues obtained from reference data in just a few cadavers (Heyward & Wagner, 2004), it is important that the principles and assumptions behind underwater weighing are understood when applied to the context of different ethnic groups. Bone density has been shown to differ between ethnicities, with Māori and Polynesian having denser bone than Caucasians (Reid, Mackie, & Ibbertson, 1986; Swinburn, Ley, Carmichael, & Palnk, 1999). Because bone makes up a significant part of LBM in the 2 component model, applying assumptions of LBM density to ethnic groups with greater bone density must be done with caution.

Interestingly, considering the body of literature dedicated to developing more direct measures of body composition to predict health status, no accepted guidelines for a healthy percentage body fat range exist. In 2000, Gallagher et al. published a paper which provided the ground work for establishing international healthy body fat ranges (D. Gallagher et al., 2000), but almost a decade later and, to my knowledge, standard body fat ranges have still not been established. Until such ranges are established, development of easier to use methods of body composition measurement, are of little value in public health assessment. On the other hand, the realization that body fat distribution is a more significant predictor of insulin sensitivity and health risk than total body fatness, may have halted the development of appropriate body fat ranges as the focus shifts toward body fat distribution and the measurement of ectopic fat stores.

7.4.1 Chapter Summary

We have shown that the Biospace 230 INBODY BIA analyser, provided %BF measures that correlated well with the ‘gold standard’ method of underwater weighing in a group of healthy males but that with the manufacturers equation, BF% was underestimated by an average of 2.3% in the entire group (see **Table 7-2**) and this was similar in Māori and non-Māori. Also, BIA may not be appropriate for individual assessment, as it caused significant under/over estimation of %BF. While the results of this study do not demonstrate a significant contribution from ethnicity like other studies, this could be due to the small sample size employed.

8 Study 4: The repeatability of percutaneous electrically stimulated twitch characteristics in healthy men

8.1 Introduction

Within the body, skeletal muscle is the main site of insulin-mediated glucose disposal (Baron, Brechtel, Wallace, & Edelman, 1988) and lipid oxidation (cited in Kim, Hickner, Cortright, Dohm, & Houmard, 2000). Thus, skeletal muscle insulin sensitivity largely determines whole body insulin sensitivity.

A whole skeletal muscle is innervated by many motor neurons; each motor neuron innervates a set of muscle fibres (multi-nucleate cells called myocytes). The motor neuron and the muscle fibres it innervates are called the motor unit. The fibres in each motor unit area are all innervated together and possess similar functional characteristics and physiological properties. However, between motor units these fibre characteristics and properties are different and can be compared and classified by either histochemical, biochemical, morphological or physiological characteristics (Scott, Stevens, & Binder-Macleod, 2001). Although current methods of this “fibre typing” rely mostly on biochemical and histochemical analysis of muscle samples obtained through biopsy, muscle fibres are also characterized by certain contractile characteristics. In fact, the earliest fibre-typing research was based on observations of different contractile behaviour between different muscle groups in animals and humans (Needham, 1926). Fast twitch fibres were shown to have a shorter twitch time, a higher twitch tension and a greater fatigability than slow twitch fibres (Sadoyama, Masuda, Miyata, & Katsuta, 1988; Thorstensson & Karlsson, 1976). Twitch characteristics and

muscular fatigability measured with methods other than biopsy, have also proven to correlate well with fibre type composition (E. Suter, Herzog, Sokolosky, Wiley, & Macintosh, 1993).

The proportion of these varying fibre types, within a whole muscle, has also been shown to relate closely with body composition and insulin sensitivity. In this context, if a person possesses a greater proportion of fast twitch (type IIX glycolytic) muscle fibre, they are more likely to have increased adiposity and reduced insulin sensitivity (Hickey, Carey et al., 1995; Tanner et al., 2002; Wade, Marbut, & Round, 1990). Conversely, those characterised by more slow twitch (type I oxidative) fibres, are less likely to be obese or insulin resistant (Lillioja et al., 1987a). This association between muscle fibre-type and insulin sensitivity is likely due to the variation in oxidative capacity between muscle fibres, which alter the rate of lipid metabolism and therefore glucose transport (Daugaard et al., 2000) (See section 2.6.3 and 2.6.4).

It is thought that fibre type is genetically determined (Komi et al., 1977) and a number of studies have shown an ethnic difference in fibre type proportion in well-trained and obese individuals (Ama et al., 1986; Kohn, Essen-Gustavsson, & Myburgh, 2007; Tanner et al., 2002). However, more recent studies have revealed that fibre type proportion can change in response to training or inactivity (Adams, Hather, Baldwin, & Dudley, 1993; Schantz, Billeter, Henriksson, & Jansson, 1982; Staron et al., 1991). Denervation, as occurs in the leg muscles of paraplegics, reduces the relative number of type 1 fibres (Burnham et al., 1997; Martin, Stein, Hoeppe, & Reid, 1992) and fatigue resistance (Rochester et al., 1995). These adaptations can be reversed by repeated electrical stimulation (Martin, Stein, Hoeppe, & Reid, 1992; Rochester et al., 1995), demonstrating that contractile activity alone, can induce a shift in the muscle fibre from fast, fatiguing fibres (IIX), to slower, less-fatiguing fibres (IIA

and I). However, in able-bodied people, changes in fibre type with training are generally small, restricted to conversion of type IIB to type IIA (P. Andersen & Henriksson, 1977; Houmard et al., 1993), or non-existent (P D Gollnick et al., 1973). Simoneau and Bouchard (J. A. Simoneau & Bouchard, 1995) suggest that fibre type proportion is partly a function of both genetics and environment.

Given the inherent nature of an individual's fibre type and the apparently protective effects of slow fibre types against the development of insulin resistance, understanding an individual's muscle composition could potentially be a means to identify early, those most at risk of metabolic disorders such as diabetes. Also, because the effects of different training intensities are fibre type specific (Cortez, Torgan, Brozinick, & Ivy, 1991), identifying fibre-type proportion could provide more effective exercise prescription.

In order to obtain an estimate of muscle fibre proportion, muscle biopsies are usually required. Once a sufficient sample of muscle is obtained through biopsy, the individual fibres are identified by biochemical or histochemical analysis, or myosin heavy chain identification. The biopsy procedure is very invasive, contains some risk, and because of inter-muscular, as well as intra-muscular variation of fibre type, the reliability of obtaining a true representation of muscle composition has been challenged (Lexell, Henriksson-Larsen, & Sjostrom, 1983; Lexell, Taylor, & Sjostrom, 1985). Furthermore, even if biopsy samples are reliable, the classifications obtained through the different analysis techniques mentioned above do not always agree (Staron, 1997).

Percutaneous (via the skin) electrical stimulation has been utilized for decades in many clinical settings to maintain muscle mass and strength during times of immobilization (Delitto

& Snyder-Mackler, 1990; Lake, 1992). Electrical stimulation allows the muscle to produce force while bypassing the nervous processes involved with a voluntary contraction. In a research context, electrical stimulation has provided a way to simulate voluntary muscle contraction, and has been used in many physiological studies as a substitute for voluntary exercise. Only one study that I know of has applied it to the measurement of contractile properties however (Blimkie, Sale, & Bar-Or, 1990).

Therefore, it was the intention of this study to a) observe whether twitch characteristics obtained by measuring the time to peak tension of an electrically stimulated twitch were repeatable, and b) whether variation could be observed between subjects, in order to ascertain the feasibility of investigating the electrically evoked twitch method and its relation to muscle fibre type.

8.2 Methods

8.2.1 Participants

Nine men aged 27 ± 4.3 years (mean \pm SD) volunteered to participate in this study. All participants completed a health questionnaire; none reported any cardiovascular problems, diabetes, used a pacemaker, or chronic injuries that would prevent them from performing the movements required. All of the participants participated in social sport but none were well-trained. Their physical characteristics are described in the table below.

Table 8-1 – Participant physical characteristics

Participant	Age (yrs)	Height (m)	Weight (kg)	BMI (kg/m²)	%BF
IW	25	1.95	110.2	29.0	28.0
NP	32	1.59	61.0	24.1	14.0
HM	18	1.74	69.4	22.9	8.0
WR	25	1.94	75.8	20.1	13.5
GE	32	1.67	62.4	22.4	8.0
CM	29	1.81	84.5	25.8	14.0
TM	28	1.67	62.8	22.5	15.0
DN	26	1.82	71.5	21.6	21.3
JM	28	1.78	68.6	21.7	16.0
Mean	27	1.77	74.0	23.3	15.3
<i>Standard Deviation</i>	4.3	0.12	15.4	2.7	6.2

8.2.2 Protocol

Participants attended the Massey University Human Performance Laboratory at the Turitea campus, in the morning after breakfast, but before any form of exercise for that day. All were asked not to perform any unnecessary leg exercise in the 3 days leading up to the test.

Usually participants came in groups of 2-4, though this was not always the case. Each participant was asked to sit in a seated position on a specially designed rig and was secured to the seat by tightening a safety belt around the waste. A load cell (Sensatronics – Class; C3 DIV 3000) was then attached to the participant's dominant leg (identified as the leg they would naturally kick a ball with) just above the ankle (maleolus) with a Velcro[®] strap which could be adjusted to limit the slack between relaxation and contraction. This was connected to a custom made D.C amplifier and then to the 'Power Lab 4/25' (ADInstruments, Australia). This circuit of apparatus measured the amount of force that the lower leg exerted during an electrically stimulated leg extension. Percutaneous electrode pads were attached to the lateral part of the thigh on the *vastus lateralis*. One electrode was placed $2/5^{\text{th}}$ of the distance from hip to knee and the second at $4/5^{\text{th}}$ the distance. *Chart v5.4.2* software by *ADInstruments, Australia* was used to collect data as well as control the stimulation, by way of a Digitimer Stimulator (model DS7A, *Digitimer Ltd, England*). This software provided numerical and graphical data of absolute force produced per unit time. Two point calibration of the load cell was performed each morning before testing.

After being attached to the measuring apparatus each participant went through a familiarisation routine involving a series of evoked twitches controlled by the Digitimer stimulator. These were all at 10V and increased in amperage from 100-900 mA. Appropriate testing amperage was obtained when the maximum force produced by the electrical

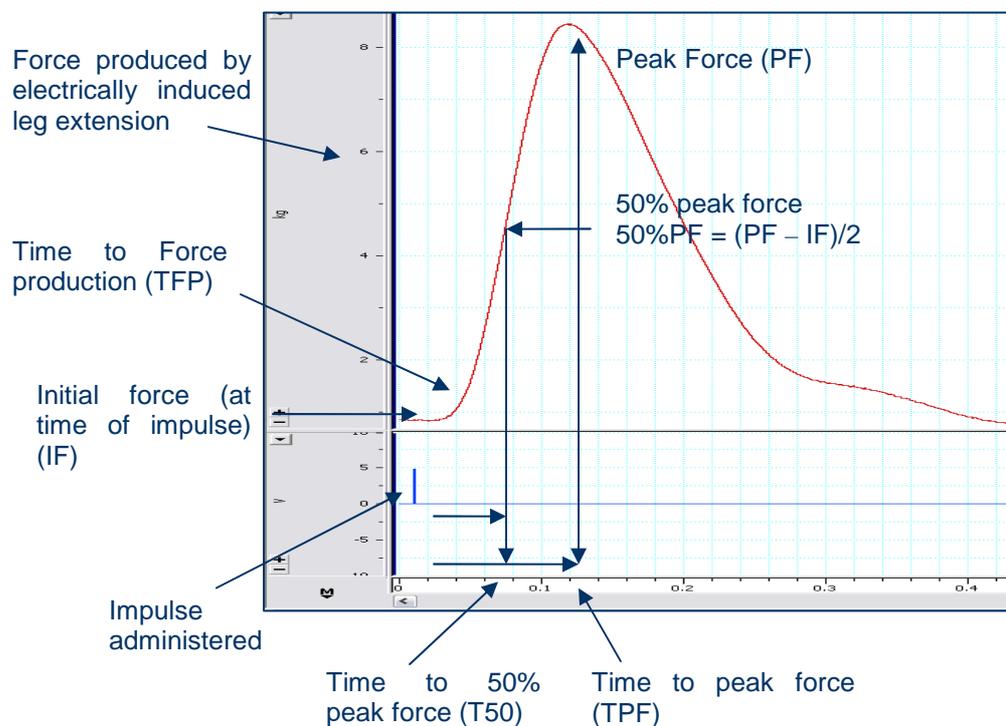
stimulation reached a plateau and no longer increased with an increase in amperage, or when the participant no longer felt comfortable with an increase in amperage.

Five twitches were then measured at the 'plateau amperage' with a 30 second break between each administration. Each twitch was then analysed for time to peak-force (TPF), and time to 50% peak-force (T50).

Each participant returned to the laboratory two more times to conduct this repeat this testing protocol. The three testing sessions were 3-5 days apart for each participant.

8.2.3 Analysis of twitch

Figure 11 – Computer analysis of electrically evoked twitch



Each of the 5 twitches obtained at each testing session was analysed for time to peak force (TPF) and time to 50% peak force (T50) using the Chart v5.4.2 software (ADInstruments, Australia) (table 4.2).

8.2.4 Statistical Analyses

Repeat-measures analysis of variance was performed to investigate differences in the dependent variables (TPF, T50): a) within a testing session (individual twitch number), b) between testing days (session), c) between participants, and d) interactions between a), b), and c). Significance required a 95% level of confidence to detect a real effect ($P \leq 0.05$).

8.3 Results

All participants completed all three trials with minimal discomfort and no reported side effects.

8.3.1 Reliability of twitch characteristics

Analysis of variance revealed that there were no significant interactions between individual twitches and subject, nor was there an interaction between individual twitch and session ($p>0.05$). However, significant interaction was revealed between subject and day, indicating that some subjects did differ from one day to another in TPF and T50 ($P<0.001$ for both), even though overall the difference between trial days was not significant within the group ($P>0.05$).

There was no significant difference between individual twitches taken within each trial day either ($P>0.05$), indicating the repeatability of the test within sessions and between sessions. The analysis of variance also showed that there was significant variation between subjects in both TPF ($P<0.001$) and T50 ($P<0.01$). Table **Figure 12** shows the repeatability of the T50 and **Figure 13**, the repeatability of TPF ($n=9$)

Figure 12 – Repeatability of the T50 measure for each participant, and the variation between participants (each colour represents a single participant)

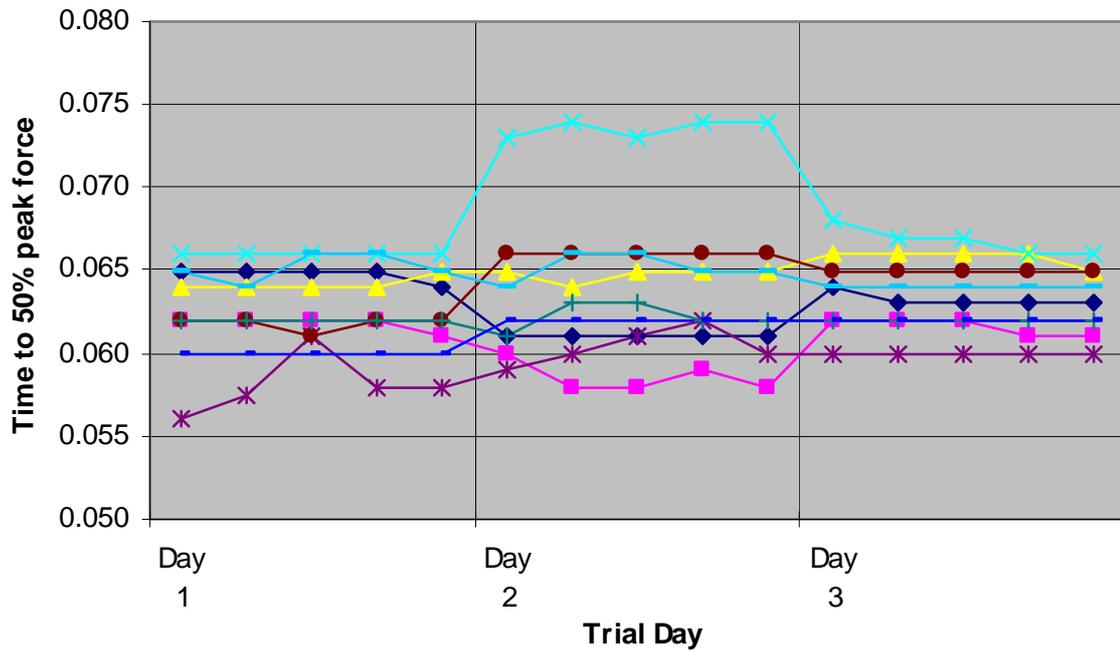
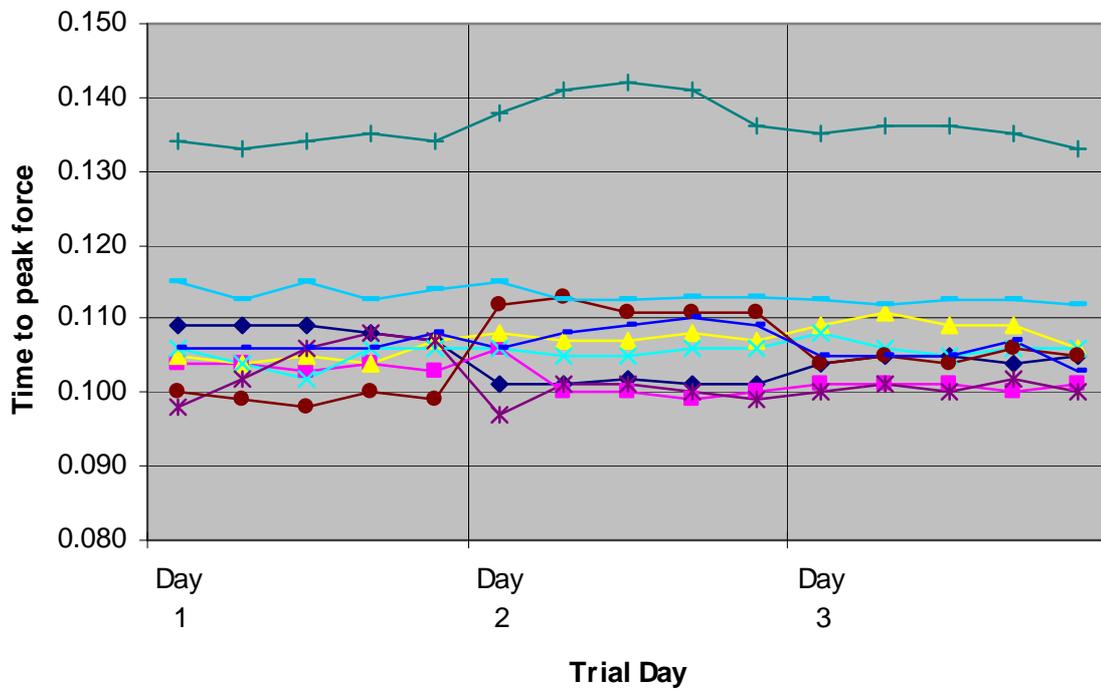


Figure 13 – Repeatability of the TPF measure for each participant, and the variation between participants (each colour represents a single participant)



8.4 Discussion

Because a higher proportion of fast-twitch muscle fibre is associated with a greater risk of obesity, insulin resistance and type-2 diabetes, measuring an individual's fibre-type proportion may, in theory, aid in identifying those with a greater risk of developing type-2 diabetes. The metabolic effects of different forms of exercise also appear to be fibre-type specific (Cortez, Torgan, Brozinick, & Ivy, 1991), so that identifying an individual's fibre type may provide more specific exercise recommendations to maintain and improve insulin sensitivity. Due to the invasive nature of the biopsy procedure required for histochemical and biochemical identification of muscle fibre type, recruitment into fibre type-based studies and the scope of research in this area has been limited. A few studies suggests that contractile properties measured externally, are related to fibre-type proportion obtained through biopsy (Ivy, Withers, Brose, Maxwell, & Costill, 1981; Sadoyama, Masuda, Miyata, & Katsuta, 1988). Thus, it was the purpose of this study to investigate the utility and reliability of twitch characteristics (measured as time to peak tension), during a percutaneous electrically stimulated twitch. It was not the intention of this study to investigate whether the electrically evoked twitch characteristics reported correlated well with fibre type proportions obtained through biopsy and histochemical analysis, but to investigate whether the measures were a) individual, and b) repeatable, and therefore warranted further investigation.

A number of studies have made the comparison between contractile characteristics obtained with external stimuli and histo/bio-chemically analysed fibre type proportion from muscle samples, but using different methods to characterise contractile property (Ivy, Withers, Brose, Maxwell, & Costill, 1981; Sadoyama, Masuda, Miyata, & Katsuta, 1988; E. Suter, Herzog, Sokolosky, Wiley, & Macintosh, 1993). These studies have shown that conduction velocity (Sadoyama, Masuda, Miyata, & Katsuta, 1988), peak torque, peak power and the rate of

power production (Ivy, Withers, Brose, Maxwell, & Costill, 1981) are related to histochemically determined fibre type composition. Others have also shown a relationship between muscle fatigue and histochemically determined fibre type (E. Suter, Herzog, Sokolosky, Wiley, & Macintosh, 1993; Thorstensson & Karlsson, 1976).

In the present study I have been able to show that evoked twitch characteristics in the quadriceps muscles of an individual, in terms of time to peak tension at least, are highly repeatable within a testing session. While some individuals produced significantly different results on different days, overall there was no significant difference in both TPF and T50 between testing sessions, suggesting that this measure is largely repeatable. It is important to note that there was a significant variation between the twitch characteristics of subjects tested, suggesting that a person's muscle twitch characteristics are inherent and not subject to temporal or environmental influences. To my knowledge, only one previous study has employed percutaneous electrical-stimulation to determine twitch characteristics, although this study measured peak dynamic torque with a dynamometer (Blimkie, Sale, & Bar-Or, 1990). One other study also used a force transducer, along with EMG, to measure fibre conduction velocity, but the measurement was taken during a voluntary contraction, rather than an electrically evoked contraction (Sadoyama, Masuda, Miyata, & Katsuta, 1988). Because a maximal voluntary contraction can be influenced by psychological factors such as motivation, an electrically stimulated twitch may provide a way to overcome the error associated with such psychological factors. To date there are no published studies which report measuring the time to peak force of an electrically stimulated twitch.

Because I was unable to ascertain whether the measures I took (TPF, T50) were associated with other indicators of fibre type characteristics, or whether these measures truly represented

the twitch characteristics of the muscle, the next obvious step in this line of research would be to compare these twitch characteristics to biopsy samples, in order to determine whether these twitch measures are correlated with fibre type proportion. As fibre type is also associated with insulin sensitivity and %BF, it would also be of worth to investigate the correlation of these twitch characteristics with measures of body composition and insulin sensitivity, and whether measurement of contractile properties can be employed to predict insulin sensitivity.

Because of the innovative nature of the methods used in this study, presently, no reviews exist which discuss the percutaneous electrical stimulation method and its limitations in determining twitch characteristics as such. Nevertheless, care was taken in placing the electrode pads in the same spot on the thigh for each session. Besides placement of electrode pads, possibility for technical error from impulse administration was small, because the timing and magnitude of the impulse was controlled electronically. The variation that some participants showed in TPF and T50, between testing sessions, could be explained by either muscular fatigue during sessions. Also, attachment of the lower leg to the force transducer has been identified as a possible source of operational error.

For field studies, or in laboratories where a dynamometer is not available, measurement of peak tension with a simple force transducer may be more appropriate. On the other hand, because T50 and TPF have not been compared with fibre type determined from muscle samples, as fatigue, peak torque, peak power and rate of power production have been (Ivy, Withers, Brose, Maxwell, & Costill, 1981), it is possible that these two measurements may not relate to fibre type, or even muscle contractile properties. Nevertheless, if further investigation demonstrates that this techniques correlates well with fibre type proportion and measures of insulin sensitivity, it could provide a non-invasive way to predict insulin

sensitivity in population studies, and aid in the development of fibre-type specific exercise recommendations.

8.4.1 Chapter Summary

The time to peak force (TPF) and 50% peak force (T50) of an electrically evoked twitch in the vastus-lateralis is repeatable within a session of 5 twitches and between 3 separate testing sessions. Variability of twitch characteristics between subjects is significant. Further study is recommended, to assess the validity of this method, as a true representation of muscle contractile properties, and whether the contractile properties measured can predict insulin sensitivity.

9 Study 5: Measuring muscle contractile properties to predict insulin sensitivity in healthy men

9.1 Introduction

Skeletal muscle fibres vary in functional and metabolic properties which meet the varying requirements placed upon them. Slow twitch fibres contain a slower myosin ATPase, a greater mitochondrial volume/density, oxidative enzyme activity, and greater capillarization than fast twitch fibres, making them more efficient and less fatigable during prolonged exercise. Fast twitch fibres on the other hand, are better suited to high-power, short bouts of muscular activity, and evidently contain a greater proportion (volume/volume) of glycolytic enzymes than slow twitch fibres (Wilmore, Costill, & Kenney, 2005; Zierath & Hawley, 2004). Whole-body aerobic fitness, measured as maximal oxygen uptake, is largely determined by blood supply to the working muscle (González-Alonso & Calbet, 2003), but also relies upon sufficient mitochondrial content and oxidative enzyme activity to utilize this delivered oxygen; as indicated above, all of the latter are associated with slow twitch muscle fibre (Essén, Jansson, Henriksson, Taylor, & Saltin, 1975; He, Watkins, & Kelley, 2001; Zierath & Hawley, 2004).

These noted variations between muscle fibres have implications for health status in humans. Chronic endurance exercise (endurance training) increases an individual's oxidative capacity, mainly due to an increase of mitochondrial volume density and capillarization of the trained muscle (J. O. Holloszy & Booth, 1976).

Table 9-1 – Twitch speed, aerobic capacity and fatigability of varying muscle fibre types

Fibre Type	Twitch speed	Aerobic capacity	Fatigability
Type 1 (slow twitch/oxidative)	Slow	High	Low
Type 2A (fast twitch/oxidative)	Fast	Medium	Medium
Type 2B (fast twitch/glycolytic)	Fast	Low	High

This increase in aerobic/oxidative capacity is associated with improved whole body insulin sensitivity (C. R. Bruce et al., 2003) and an improved ability to oxidize lipid (Goodpaster, Katsiaras, & Kelley, 2003). Chronic muscle contraction, which occurs with endurance training, also appears to induce a conversion, albeit slow, from fast twitch to slower twitching fibres (Schantz, Billeter, Henriksson, & Jansson, 1982). Interestingly, possession of a greater proportion of slow twitch muscle fibres is associated with greater whole body insulin sensitivity (Lillioja et al., 1987a).

One functional characteristic of fibre type is its time to reach peak force, or ‘twitch speed’. As the name suggests, fast twitch (type II) muscle fibres contract, but also relax at a greater velocity than slow twitch fibres (Harridge et al., 1996; Reiser, Moss, Giulian, & Greaser, 1985). Consequently, the fast twitch fibres are unable to maintain this force production, and fatigue more rapidly than slow twitch fibres when forced to contract repeatedly (Thorstensson & Karlsson, 1976). Work by Suter et al. (E. Suter, Herzog, Sokolosky, Wiley, & Macintosh, 1993) showed that the rate at which relative torque decreased after 60 maximal leg extensions, was related to the proportion of type II muscle fibres in the *vastus lateralis* identified by histochemical analysis.

The literature suggests that improved oxidative capacity, as a result of a high proportion of well-trained, slow twitch (oxidative) muscle, is a major factor in determining insulin sensitivity. Consequently, identifying those with a higher proportion of fast twitch muscle fibre may provide a glimpse at those most at risk of insulin resistance and diabetes. Although research is still inconclusive as to the roles of genetics and exercise in determining muscle composition, some studies suggest that ethnicity influences fibre-type proportion (Ama et al., 1986; Kohn, Essen-Gustavsson, & Myburgh, 2007; Tanner et al., 2002). Thus, it is possible that high-risk populations, such as Māori, may be more prone to insulin resistance due to a genetically determined greater proportion of fast twitch muscle fibres.

Although it is well known that fibre type is related to obesity and insulin resistance, no studies to my knowledge have attempted to observe a relationship between muscle contractile properties, measured externally using methods other than biopsy, and markers of insulin sensitivity. Certainly, no study has investigated these relationships in Māori. While I have shown in Study 4 that time to peak and 50% peak tension (TPF and T50) are repeatable and that there is variation between participants, these two measurements are unique to this study and have not been compared with fibre type in the way that fatigue, peak torque, peak power and rate of power production have been (Ivy, Withers, Brose, Maxwell, & Costill, 1981). Thus, T50 and TPF may not be a representation of fibre type proportion, or even contractile properties of muscle, at all. Nevertheless, it was the aim of this study to observe whether T50 and TPF induced by an electrically stimulated isometric twitch, as well as the fatigability of muscle, were related to markers of insulin sensitivity.

9.2 Methods

9.2.1 Participants

Twenty one men (12 Māori and 9 non-Māori) aged 27 ± 5 (mean \pm SD) years old participated in this study. All participants completed a health questionnaire; none reported any cardiovascular problems, diabetes, used a pacemaker or had chronic injuries that would prevent them from performing the movements required. All of the participants participated in social sport but none were well-trained. Their physical characteristics are described in the table below.

Table 9-2 – Participant Physical characteristics

	Mean	SD
Age (yrs)	27	5.0
Height (m)	1.79	0.09
Weight (kg)	83.6	17.2
BMI (kg/m²)	26	4.6

9.2.2 Protocol

The methods used to obtain measures of blood biochemistry, body composition and aerobic capacity in this study, were identical to those previously described (see section 5.2).

9.2.2.1 Electrically evoked twitch test

Participants followed a protocol previously described in the methods section of Study 4, except they only participated in one session of 5 electrically stimulated twitches instead of the three separate sessions required of the participants in the feasibility study.

9.2.2.2 Fatigue Test

This test was performed on the Biodex isokinetic dynamometer (Biodex Medical systems, NY, USA), and was designed similar to a protocol used by Suter et al. (E. Suter, Herzog, Sokolosky, Wiley, & Macintosh, 1993).

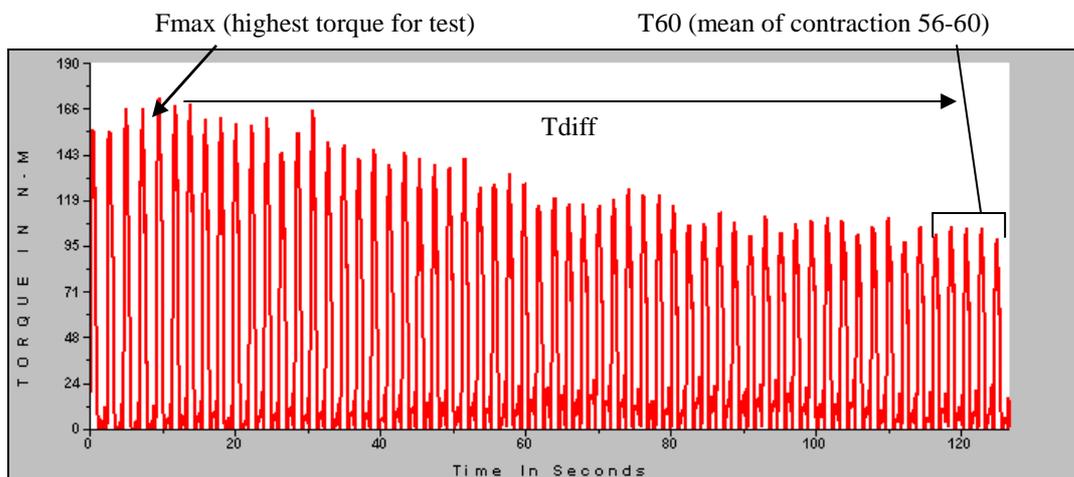
Participants were asked to sit in the seat of the Biodex apparatus and their dominant leg (see section 8.2) was attached to the dynamometer just above the ankle, with a specially designed padded strap. The arm of the dynamometer ran parallel to the lower leg and its pivot point was adjusted to be in line with, and 10-15mm from, the lateral condyle of the femur.

After a warm up including stretching and 3 sets of 4 leg extensions at various velocities on the apparatus, participants became familiar with the force they would be exerting during the test. For the testing protocol, participants were asked to perform 60 knee extensions at an angular velocity of 90 degrees per second. Participants were encouraged throughout the test to put forth a maximal effort for each contraction. Peak torque was recorded for each contraction throughout the test. For a detailed description of the dynamometer apparatus and its use, see Thorstensen et al. (Thorstensson, Grimby, & Karlsson, 1976).

The Biodex software (Biodex System 3) provided graphs (see **Error! Reference source not found.**) and tables of the peak torque of all 60 contractions throughout the test. These tables were used to calculate the fatigability of an individual. To calculate fatigability i.e. the drop off of torque after 60 contractions, the mean of the peak torque for the last five contractions (mean of 56-60), was subtracted from the peak torque recorded in the test (T_{max}), to obtain

the absolute difference (Tdiff). The relative fatigue, the drop off as a percentage of Tmax (%diff), was also recorded.

Figure 14 – Computer Analysis of Tdiff by subtracting T60 from Fmax



9.2.3 Statistical analyses

VO₂max was expressed relative to lean body mass (l_{bm} – derived from two compartment hydrodensitometry) rather than relative to whole body mass because it: a) better represents the aerobic performance of the working muscle (Coon, Bleecker, Drinkwater, Meyers, & Goldberg, 1989); and b) provides for a truly independent variable in the multiple regression analysis.

Simultaneous multiple regression was performed on the dependent variables which were markers of insulin sensitivity or glucose tolerance (HOMA-IR, fasting glucose, fasting insulin, two-hr glucose two-hr insulin). The independent variables included in the analysis were percentage body fat, relative VO₂max/l_{bm} (ml.kg⁻¹ l_{bm}.min⁻¹), and T50 or TPF. Bivariate correlation was used to describe the relationship between T50 and the dependent variables. Bivariate correlation was also used to describe the relationship between measures

from the fatigue study (PerDecl, Absdiff and Fmax), and the previously noted dependent variables. Significance was ascribed at a confidence level of 95% (two tailed). All statistical analyses were performed on specialist software (SPSS Version 10).

9.3 Results

We were unable to obtain a *2hr glucose sample for one participant and *2 hr insulin samples for four participants.

9.3.1 Blood Biochemistry

Table 9-3 – Blood biochemistry

	Mean	SD	<i>n</i>
HOMA-IR	1.76	1.5	21
Fast Ins (pmol.L⁻¹)	56	42	21
Fast Gluc (mmol. L⁻¹)	4.7	0.5	21
*2hr Ins (pmol.L⁻¹)	145	111	17
*2hr Gluc (mmol. L⁻¹)	4.3	0.9	20

9.3.2 Body Composition and Aerobic Capacity

Table 9-4 – Percent body fat, lean body mass and VO₂maxLBM

	Mean	SD
%BF	18.0	7.8
LBM (kg)	68.0	9.4
VO₂max LBM (ml.kgLBM⁻¹.min⁻¹)	55.43	7.8

9.3.3 Twitch Test

9.3.3.1 Multiple regression analysis

Simultaneous multiple regression analysis revealed that 46% of the variance in HOMA-IR, 45 % of the variance in fasting insulin, and 56% of the variance in 2hr insulin, could be predicted by the T50 value ($p < 0.05$). TPF was not a significant predictor of any of the independent variables analyzed ($p > 0.05$).

9.3.3.2 Bivariate Correlation

Bivariate correlation showed that T50 and TPF were not significantly correlated with $\text{VO}_2\text{max/lbm}$ ($r < 0.2$, $p > 0.05$). My results did show however that T50 was directly correlated with HOMA, fasting insulin, 2hr insulin and 2hr glucose ($p < 0.05$). T50 was also significantly related to %BF as well as absolute body weight ($p < 0.01$). TPF however, was not significantly related to any of the variables tested. There was no significant relationship between T50 and ethnicity or T50 and age ($p > 0.05$).

9.3.4 Fatigue Test

9.3.4.1 Multiple regression analysis

Simultaneous multiple regression revealed no significant contribution to markers of insulin sensitivity or glucose tolerance from Fmax, PerDecl or AbsDiff, when included with the other independent variables used ($\text{VO}_2\text{max/lbm}$ and %BF) ($p > 0.05$)

9.3.4.2 Bivariate Correlation

Bivariate correlations showed that Fmax (maximal force produced during the fatigue test) and Absdiff (absolute difference between Fmax and the mean of the last 5 twitches) were significantly correlated with T50 and %BF ($p < 0.01$). Fmax and Absdiff were also related to

HOMA-IR measurements ($p < 0.05$). PercDecl (The mean of the last 5 twitches as a percentage of Fmax), which represented the relative decline of force, was not related to T50, TPF, $VO_2\text{max/lbm}$, %BF or any of the blood markers ($p > 0.05$).

Table 9-5 – Simultaneous multiple regression analysis

Dependant variable	Independent variable	R	r ²	t-value	P-value	n
HOMA-IR	T50	0.675	0.455	2.963	0.009	21
	$VO_2\text{max/lbm}$			-1.563	0.137	
	%BF			-1.484	0.156	
Fasting serum insulin	T50	0.670	0.448	2.803	0.012	21
	$VO_2\text{max/lbm}$			-1.650	0.117	
	%BF			-1.241	0.232	
Fasting serum glucose	T50	0.524	0.275	2.318	0.033	21
	$VO_2\text{max/lbm}$			-0.109	0.915	
	%BF			2.318	0.033	
2hr serum insulin	T50	0.746	0.557	3.353	0.005	17
	$VO_2\text{max/lbm}$			-1.282	0.222	
	%BF			-1.702	0.113	
2hr serum glucose	T50	0.619	0.383	1.650	0.119	20
	$VO_2\text{max/lbm}$			-1.243	0.232	
	%BF			0.350	0.731	

9.4 Discussion

Muscle fibre type proportion is related to athletic performance as well as metabolic health. Measuring fibre type proportion in an individual can be difficult and invasive however, as it requires muscle samples obtained through biopsy. Fibre type proportion is also associated with a person's muscle contractile characteristics and fatigability measured externally. Thus, the purpose of this study was to investigate the relationship between electrically evoked twitch characteristics, muscular fatigue and markers of insulin sensitivity.

Although studies have shown that insulin sensitivity is associated with an individual's histochemically determined fibre type proportion, to the author's knowledge, none have investigated whether contractile characteristics measured externally could predict insulin sensitivity. In this study, I was able to show that electrically evoked twitch characteristics were significantly related to estimates of insulin sensitivity (HOMA-IR). There was no significant interaction between twitch characteristics and age, or twitch characteristics and ethnicity in this cohort. While the TPF measure was not a significant predictor of any of the variables studied, T50 was a significant predictor of fasting insulin, 2hr insulin (post glucose load) and 2 hr glucose (post glucose load). T50 was also significantly related to BF% in this cohort, but not $VO_2\text{max/lbm LBM}$.

According to findings of twitch characteristics and its relationship to fibre type, if a correlation was observed between T50 and insulin sensitivity, it was expected to be an inverse relationship, as fast twitch fibres are associated with obesity and insulin resistance i.e. the shorter the T50, the higher the %BF and HOMA values. Surprisingly however, these were not inverse relationships as expected. That is, those with the highest HOMA-IR score (more

insulin resistant) were characterised by slower twitch speeds. This observation can probably be explained according to the methodology I've employed.

Electrical conductivity in the thigh may explain the relationship between slower twitch time and higher HOMA-IR, because a higher HOMA-IR is strongly associated with increased %BF. While muscle is a relatively good electrical conductor, adipose tissue is not. Indeed it is possible that conduction of the electrical impulse may be different, but repeatable between individuals because of some other inherent or physical factor such as subcutaneous fatness (of the thigh). Therefore, the electrical impulse administered percutaneously would be impeded in those with greater adiposity surrounding the vastus lateralis, increasing T50. In order to account for the impedance, I also observed whether there was a relationship between adiposity and 'time to force initiation' (identified as the time from impulse administration to the moment contractile force went beyond 3 standard deviations from initial force). No significant relationship existed between 'time to force initiation' and obesity however ($P > 0.05$), neither was there a significant change in the results when T50 and TPF were adjusted for 'time to force initiation'. These findings suggest that factors associated with adiposity, other than impulse impedance, are responsible for the 'adiposity/slow-twitch' relationship.

Furthermore, I expected to see a relationship between a subject's ability to resist fatigue in the fatigability test, and their $VO_2\text{max/lbm}$ Suter et al. (E. Suter, Herzog, Sokolosky, Wiley, & Macintosh, 1993) showed that the decline in force during a dynamometer test similar to mine, was associated with fibre type. They found that those with a greater proportion of fast twitch fibres had a greater relative decline than those with a high proportion of slow twitch fibre. This study revealed that the relative decline was not significantly related to markers of insulin

sensitivity or aerobic fitness (expressed as $\text{VO}_2\text{max/lbm}$). Relative decline was not significantly related to T50 or TPF either.

It appears that the methods I used to obtain twitch characteristics, namely TPF and T50 of an evoked twitch, are probably not valid as measures of fibre type and thus require further development. Indeed, T50 and TPF are innovative measures which have not been compared with muscle samples, so these two measures may not actually be associated with twitch characteristics in the muscle at all. However, the fatigue protocol, which has been shown to correlate with fibre type, did not produce any significant relationships between fatigability and markers of insulin sensitivity either. This could be due to the use of healthy subjects in my studies, as variations in oxidative capacity and insulin sensitivity do not only exist between different fibre types, but between the same fibre types of different subjects i.e. It is possible to have insulin sensitive type 2 fibres. Thus, observations of the protective qualities of type 1 fibre would only be distinguished in unhealthy subjects. Nonetheless, there are a number of useful applications for measuring fibre type proportion, warranting further development of non-invasive methods of fibre-typing.

Because fibre type is associated with obesity and the ability to oxidize lipids, measuring an individual's fibre type proportion, may allow early identification of those who are most susceptible to obesity and metabolic disorder, long before the individual has reached an unhealthy state.

The type of exercise i.e. endurance-type or high intensity, and its effectiveness in improving metabolic profile, is also influenced by fibre-type, so that measurement of a person's fibre type proportion may allow prescription of exercise that is more specific to an individual's

metabolic needs. 'Fibre-type dependant' exercise prescription would apply to primary prevention of obesity and insulin resistance, as well as the treatment of those who are obese and/or glucose intolerant. In regards to reducing body fat within the obese, Tanner et al. (Tanner et al., 2002) found that obese individuals with a higher proportion of slow-twitch fibres were better able to reduce body mass with intervention, when compared with those who had a higher proportion of fast-twitch fibre.

Studies have shown that muscle fibre type proportion differs between ethnic groups. Interestingly, the same ethnic groups that are identified in the literature as having a greater proportion of fast-twitch muscle fibre (Ama et al., 1986; Tanner et al., 2002), are also identified in epidemiological studies as those with greater risk of obesity and diabetes (Cowie et al., 2006). The data highlighting this relationship is limited to only a few ethnic groups however, and certainly, there is no muscle fibre-type data from Māori. Because of the invasive methods required to accurately measure the proportion of skeletal muscle fibre types, it has not been possible to obtain population data comparing the fibre type proportions of different ethnic groups. Nevertheless, based on studies in other ethnic groups, it remains possible that Māori have a greater predisposition for obesity, insulin resistance and diabetes than NZEO, because of a genetically determined, higher proportion of fast-twitch muscle fibre.

9.4.1 Chapter Summary

The results of this study did reveal a relationship between electrically-evoked twitch characteristics and blood markers for insulin resistance. There was also a relationship between adiposity and twitch characteristics, but neither of these relationships were inverse as expected i.e. twitch time in this study increased as adiposity increased, suggesting flaws in the

methods used. Fatigability, which has been shown to correlate with fibre type proportion measured in-vivo, was not a significant predictor of insulin sensitivity, adiposity or aerobic fitness either. The association between fibre type and metabolic health warrants further development of non-invasive techniques to enable further investigation in this area, and to explore whether fibre type can partly explain ethnic health disparities.

10 Conclusions

The line of research presented in this thesis was undertaken in an effort to help explain why Māori appear more predisposed to insulin resistance and type-2 diabetes than New Zealanders of European Origin. I acknowledge the importance of socio-cultural and socio-economic factors, and the role they play in the prevalence of this condition, but this thesis has focussed on the physiological factors which may contribute. I also recognise the limitations of including ethnicity data in this physiological research. What's more, I acknowledge that the study participants were by and large homogenous, particularly the Māori cohort; thus the results may only be applicable to young Māori men. Nevertheless, it is the age represented by these participants which is likely critical in the development of diabetes and cardiovascular disease; Māori men have arguably the lowest life expectancy of any group in NZ (Statistics New Zealand, 2008), as well as a lower onset age for diabetes (Joshy & Simmons, 2006).

10.1 Summary of findings

The results of the studies here-in have confirmed that the aerobic capacity of lean body mass ($VO_2\text{max LBM}$) and percentage body fat (%BF) both help to predict insulin sensitivity (HOMA-IR) in a healthy group of young, Māori males. Furthermore, as body composition and fitness change with time, $VO_2\text{max LBM}$ and %BF still predict insulin sensitivity in this self-identified ethnic group. This relationship may vary between ethnicities however, though the reason for this remains unclear.

As a consequence of the relationship between %BF and insulin sensitivity, and thus risk of diabetes, it is important that appropriate methods become available to measure body fatness in

Māori as, to date, there are no scientifically determined Māori-specific BMI cut-off values. Accordingly, I have shown that BIA is a valid and acceptable means of assessing body fatness in young Māori men, when compared to a two compartment, established method of measuring body composition (hydrodensitometry).

The scientific literature indicates that muscle fibre type is also independently related to obesity and insulin resistance. As part of my studies, I have attempted to develop a non-invasive method of apportioning fibre type distribution by measuring the functional characteristics of muscle. Whilst this method proved reliable and acceptable to the participants, its validity in representing twitch characteristics representative of fibre type proportion, is questionable. Furthermore, and likely as a consequence of its invalidity, twitch characteristics obtained with this method were not able to predict markers of insulin sensitivity.

10.2 Applicability

Measuring aerobic fitness, in combination with body composition, is a valid way of identifying risk of insulin resistance, as represented by elevated fasting insulin, in Māori men who maintain normoglycaemia. It is important to note the importance of measuring both, as both body fatness and aerobic fitness of the lean body mass are independent predictors, but the latter requires knowledge of lean body mass. In a clinical and public health setting, however, both may be difficult to obtain because of the facilities and equipment required to accurately make these measures. However, there are valid and reliable means of predicting both of these. Indeed, in this thesis, I have described the validity of BIA to predict body fatness in young Māori men. Maximal aerobic fitness, on the other hand, is more difficult to measure. There are some tests available, such as a ‘beep’ test which can infer maximal

aerobic capacity from performance, though skill may play some role (Léger & Lambert, 1982). Sub maximal testing may be more useful in the clinical, or public health setting, as such tests have shown to be valid in large population studies (Noonan & Dean, 2000).

In connection with the findings of others, aerobic capacity (of lean body mass) may not be associated with insulin sensitivity in non-Māori however. Since aerobic capacity contributed significantly to the variance in insulin sensitivity in the Māori but not the non-Māori cohort, ethnicity may determine the extent to which aerobic fitness effects insulin sensitivity. This knowledge may help explain why Māori have reduced insulin sensitivity when compared to Europeans of the same BMI. It also highlights the possibility of a genetic influence on the development of insulin resistance. In context of the theories of genetic adaptation discussed earlier (on page 64), Māori and other ethnic groups which had to expend relatively more energy to obtain food, would have benefited from a physiological mechanism which controlled glucose uptake relative to energy requirements. Europeans on the other hand, may have adapted mechanisms which control glucose uptake irrespective of aerobic fitness, in response to increased food availability that did not require great energy expenditure.

Fibre-type proportion has also been identified as a possible factor in ethnic health disparities, probably because of the varying oxidative properties between different muscle fibres, and the necessity of adequate oxidative capacity of muscle in maintaining sensitivity to insulin-mediated glucose uptake. Thus, although I can only speculate, it is possible that a larger proportion of fast or slow-twitch muscle may influence the relationship between aerobic fitness and insulin sensitivity. Findings from my study of twitch characteristics, and their relationship with obesity and insulin sensitivity are unable to resolve this issue because of methodological issues. Specifically, the electrical stimulation method (which measured the

time to peak force) showed that the speed of the electrically evoked twitch (measured by a force transducer) is probably not a function of fibre type, but of the absolute size of the participant, and thus probably the cross-sectional area of the thigh muscle which was stimulated. Nevertheless, these results do not rule out the future possibility of employing contractile characteristics to predict health risk, or in obtaining a representation of fibre type proportion if an appropriate tool can be developed and validated.

Another aim of the research described within the thesis was to observe any changes, over a time period of two years, in health-related markers in a small group of young Maori men. Interestingly, the reduction in aerobic capacity seen within this group was not accompanied by a reduction in insulin sensitivity. However, the relationship between aerobic fitness per unit lean body mass and insulin sensitivity remained. These results are likely due to the increased lean mass seen within this group after two years. It may be that since the initial tests, participants grew a little or undertook some form of resistance training and hypertrophy resulted. However, it is also likely that the reduction in aerobic capacity observed in Study 1 may reflect a small but consistent difference in maximal ventilation measurement, because two slightly different methods were used at the different time points (Macfarlane, 2001). Douglas bags were used during the initial (Sydney) study, but in the second (Wellington) study, a turbine was used to measure ventilation; the use of which, has been shown by some to result in error when measuring ventilation (Yeh, Adams, Gardner, & Yanowitz, 1987).

10.3 Implications

The implication of my findings can be applied to public health studies, health interventions, and future physiological studies involving Māori. Firstly, public health studies often rely upon BMI, an indirect measure of fatness, to indicate risk of obesity-related illnesses such as

diabetes and CVD. The results of my study imply that BIA, which actually measures body fatness, though indirectly by way of 'total body water', can be utilized within a Māori population. On the other hand, BIA can significantly under/overestimate %BF in some people, so employing BIA for individual clinical assessment should be approached with some caution.

Perhaps the most important contribution from this body of research is that improved physical fitness and a healthy body composition are associated with better metabolic health and a reduced risk of diabetes among young Māori men. The now proven association between aerobic fitness, in relation to lean body mass, and fasting insulin concentration in Māori, advocates the development of simple sub-maximal exercise tests to provide health promoters and physicians with an additional way to predict insulin sensitivity within Māori. However a VO₂max test, as performed in these studies, may not be appropriate for the obese or elderly, so development of an appropriate alternative exercise test, which correlates well with aerobic capacity, may be more suitable. The association between aerobic capacity and insulin sensitivity also highlights the importance of increasing physical fitness among Māori who are most at risk of metabolic disorders. Thus, for Māori at least, monitoring ones fitness may be more important than just monitoring their weight.

On the other hand, increasing lean body mass, through resistance training, allows maintenance of insulin sensitivity independent of aerobic fitness. Thus, resistance training may offer an alternative means of exercise for Māori, to maintain and improve glucose tolerance. Yet, this means of physical activity has not been researched nor promoted in Māori to date, even though, anecdotally, it is well accepted in young Māori men.

The implications of fibre-type proportion upon health, also warrants further research in this area which has been limited to relatively small studies because of the invasive muscle biopsies required. Although the method I used to measure contractile properties was not a valid way to predict insulin sensitivity, further development of techniques may provide a method that does. The effectiveness of exercise in improving metabolic control is dependent upon the recruitment of fibres, so being able to identify fibre-type proportion within individuals, will allow exercise prescription which maximizes the effects of exercise specific to each individual.

Importantly, the participation of Māori within this research was an innovation in itself. Māori health researchers have discussed the interface between ‘Western’ and ‘kaupapa Māori’ research methods, and have described ways to achieve this. Yet, physiological research involving Māori participants remains limited. The fact alone, that this research was conducted by a Māori exercise physiologist, is also a positive step toward desires of Māori to have leadership in issues relating to Māori; tying in with Māori health strategies and the principle of rangatiratanga. Additionally, I have sought to conduct my research at this interface according to principles discussed earlier (see chapter 3). As with any innovation however, there is no literature to compare this approach to, or measure the effectiveness of the methodology against. Nevertheless, the approach employed in this body of research may provide a methodological basis to be used and/or built upon for further scientific studies involving Māori. Thus, follow up with participants would be beneficial in assessing this approach and understanding the participants experience and view of this research.

10.4 Future Directions

Physical activity, obesity and diabetes have a cyclic relationship (Hohepa, Schofield, & Kolt, 2004). In other words, obesity is partly caused by physical inactivity but can also lead to further inactivity, as obese individuals may find it difficult to perform the physical tasks required to improve their body composition. Because of this, policy and interventions should focus on increasing awareness of physical activity levels among those who are still physically capable of making lifestyle changes. This falls inside the realm of prevention rather than treatment. Accordingly, SPARC have stated that efforts to increase physical activity should be focused on young people as well as two other groups; Māori and Pacific people (SPARC., 2004).

If Māori are the focus of policy efforts however, it is important that Māori are included in the development and implementation of policy. According to He Korowai Oranga (The Māori Health Strategy) this should be done at whānau (family), community and Iwi (tribal) levels for maximum Māori participation (Ministry of Health, 2002). District Health Boards (DHBs) and Public Health Organisations (PHOs) are also involved in the promotion of health care among Māori and the general population and should therefore play a part in policy making. In a study of diabetes among Canada's Aboriginal people, Young et al. make the observation that non-Aboriginal professionals, in this case non-Māori, involved in health care and policy making, need to understand how Māori interpret the experience of illness and respond to treatments prescribed, and to respect the logic of what may be to some a foreign system of thought (Young, Reading, Elias, & O'Neil, 2000). Training in cultural sensitivity and Māori systems of health is essential to develop policies that will achieve the outcomes of reducing the obvious health inequalities among Māori. Strategies such as He Korowai Oranga and Whakatātaka were developed as part of a framework for the public health sector to follow in

its support of Māori communities and whānau, the main goal of these strategies being the achievement of ‘whānau ora’; the realisation of maximum health and well-being among Māori families and communities.

Future policy should also involve the reduction of environments that promote sedentary, energy saving lifestyles and the excessive consumption of high energy food. Obesity during childhood also adds to the risk of obesity continuing or worsening in adulthood (Guo, Roche, Chumlea, Gardner, & Siervogel, 1994), suggesting that policy should be targeted toward the prevention of obesity in children and adolescents. Indeed, healthy eating plans are currently being piloted in some NZ schools, as the MoH implement the Healthy Eating – Healthy Action plan (HEHA) (Ministry of Health, 2003). However, because children with obese parents are more likely to become obese adults themselves (Whitaker, Wright, Pepe, Seidel, & Dietz, 1997), interventions may be more effective when parents and family are involved. Because of the part whānau (family) plays in the achievement of hauora, this approach may also be more culturally appropriate for Māori.

Along with policy changes, there are areas of research that need to be addressed. Firstly, because screening and assessment of health is often dependent upon measures such as BMI and waist circumference, Māori specific norms for these measurements should be established. Secondly, due to the importance of exercise in reducing obesity and risk of diabetes, identifying the most effective forms of exercise for Māori should be a priority. Although this should include investigation of the most effective forms of exercise in improving physiological markers associated with diabetes, it may be even more important to understand cultural, social and individual preferences to exercise. Without identifying preferred modes of

exercise, participation of 'high-risk' groups in exercise and physical activity will not likely improve.

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Appendices

10.5 Appendix A – Study 1

Table 0-1 – Participant physical characteristics

Name	Sydney Study						Wellington Study					
	Age	Height	Weight	BMI	% BF (Siri)	LBM	Age	Height	Weight	BMI	% BF (Siri)	LBM
LB	20	1.74	75.80	25.2	21.9	59.20	22	1.74	70.50	23.4	16.5	58.40
RW	21	1.82	73.60	22.2	16.2	61.68	23	1.82	88.20	26.6	19.5	63.40
TD	20	1.89	93.50	26.2	11.7	82.56	22	1.89	99.50	27.9	13.0	84.75
SW	23	1.81	103.10	31.5	36.7	65.26	25	1.81	94.50	28.8	29.5	66.25
NB	30	1.78	80.85	25.5	18.7	65.73	32	1.78	75.00	23.7	13.0	65.00
RC	30	1.82	82.95	25.2	17.7	68.27	32	1.82	90.00	27.2	23.5	68.80
CH	33	1.77	79.00	25.2	20.0	63.20	37	1.80	102.70	31.7	20.3	81.40
BC	34	1.80	91.00	28.1	17.7	74.89	36	1.83	91.40	27.3	22.0	70.65
JH	34	1.83	90.00	27.0	24.6	67.86	35	1.77	79.10	25.2	19.5	63.48
TK	37	1.74	95.40	31.5	27.4	69.26	38	1.74	94.70	31.3	26.0	69.95
JG	31	1.79	90.50	28.2	14.0	77.83	32	1.79	85.50	26.7	7.5	79.35
JR	25	1.82	152.00	45.9	42.5	87.40	27	1.82	152.70	46.1	40.0	91.20
Mean	28	2	92	28	22	70	30	1.80	93.65	28.8	20.9	71.89

Table 0-2 – Blood Pathology (Sydney)

Name	Ins pM	Ins 2 hr	Glucose	2 hr Glucose	Total Chol	HDL	LDL	Chol/LDL	Ins iU	HOMA
LB	53	114	4.5	4.4	5.1	1.30	2.6	3.9	7.63	1.53
RW	21	108	4.5	4.7	4.7	1.25	3.8	2.4	3.02	0.60
TD	39	109	6.1	3.2	-	-	-	-	5.62	1.52
SW	128	34	4.6	2.8	5.5	1.42	3.6	3.9	18.43	3.77
NB	67	136	5.5	5.4	6.1	1.27	3.7	4.8	9.65	2.36
RC	53	14	4.8	3.8	8.6	1.54	6.3	5.6	7.63	1.63
CH	71	191	5.5	5.2	4.0	1.02	2.7	3.9	10.22	2.50
BC	52	29	5.4	2.7	3.5	1.22	2.0	2.9	7.49	1.80
JH	46	114	5.0	4.3	4.7	1.13	3.2	4.2	6.62	1.47
TK	43	249	4.9	4.1	5.6	1.01	4.1	5.5	6.19	1.35
JG	65	-	5.0	3.3	5.0	1.00	2.8	4.9	9.36	2.08
JR	264	184	5.7	4.3	5.8	1.37	3.8	4.2	38.01	9.63
Mean	51	117	5	4	5	1	3	4	11	3

Table 0-3 – Blood Pathology (Wellington)

Name	Ins pM	Ins 2 hr	Glucose	2 hr Glucose	Total Chol	HDL	LDL	Chol/LDL	Ins iU	HOMA
LB	36	49	5.1	3.9	4.5	1.50	2.30	3.0	5.2	1.17
RW	54	65	4.2	2.5	5.0	1.13	3.30	4.4	7.8	1.45
TD	32	-	4.9	-	3.9	2.59	1.20	1.5	4.6	1.00
SW	85	165	4.5	3.4	4.6	1.72	2.40	2.7	12.2	2.45
NB	32	213	4.9	6.3	6.3	1.40	4.00	4.5	4.6	1.00
RC	28	78	4.7	4.2	7.9	1.42	5.40	5.6	4.0	0.84
CH	54	114	5.0	4.3	4.6	0.97	2.70	4.7	7.8	1.73
BC	31	42	5.1	3.1	4.9	1.19	3.40	4.1	4.5	1.01
JH	34	155	4.7	5.2	3.8	1.03	2.60	3.7	4.9	1.02
TK	72	96	4.9	4.1	4.6	0.92	3.10	5.0	10.4	2.26
JG	46	152	5.4	4.6	5.2	0.95	3.40	5.5	6.6	1.59
JR	195	804	5.0	5.5	5.6	1.20	3.60	4.7	28.1	6.24
Mean	58.3	176	4.9	4.3	5.1	1.34	3.12	4.1	8.4	1.81

Table 0-4 – Aerobic capacity

	Sydney			Wellington		
Name	VO2max	VO2max/kg	VO2max/LBM	VO2max	Vo2max/kg	VO2max/LBM
LB	3550	46.83	59.96	3269	46.37	55.97
RW	3833	52.09	62.15	3571	40.49	56.32
TD	4567	48.85	55.32	3748	37.67	44.22
SW	4202	40.75	64.38	3641	38.53	54.96
NB	3796	46.95	57.75	3161	42.15	48.63
RC	4409	53.15	64.58	3363	37.37	48.88
CH	3165	40.06	50.08	4151	40.42	51.00
BC	4979	54.71	66.48	4104	44.90	58.09
JH	4375	48.61	64.47	3862	48.82	60.84
TK	3843	40.28	55.49	2802	29.59	40.06
JG	4212	46.54	65.09	3762	44.00	47.41
JR	4075	26.81	46.62	3340	21.87	36.62
Mean	4084	45.47	59.36	3564	39.35	50.25

Table 0-5 – Multiple regression analysis (Sydney)

Dependant variable	Independent variable	r	r²	t	P
<i>HOMA-IR</i>	% body fat	0.847	0.717	3.417	0.008
	VO ₂ maxLBM			-1.797	0.106
<i>Fasting serum insulin</i>	% body fat	0.861	0.741	3.952	0.003
	VO ₂ maxLBM			-1.452	0.181
<i>Fasting serum glucose</i>	% body fat	0.65	0.423	-1.287	0.230
	VO ₂ maxLBM			-2.544	0.032
<i>2hr serum insulin</i>	% body fat	0.781	0.610	-0.060	0.954
	VO ₂ maxLBM			-3.366	0.010
<i>2hr serum glucose</i>	% body fat	0.543	0.295	-0.774	0.459
	VO ₂ maxLBM			-1.942	0.084

Table 0-6 – Multiple regression analysis (Wellington only)

Dependant variable	Independent variable	<i>r</i>	<i>r</i>²	<i>t</i>	<i>P</i>
<i>HOMA-IR</i>	% body fat	0.873	0.763	3.829	0.004
	VO ₂ maxLBM			-2.388	0.041
<i>Fasting serum insulin</i>	% body fat	0.879	0.772	4.116	0.003
	VO ₂ maxLBM			-2.214	<u>0.054</u>
<i>Fasting serum glucose</i>	% body fat	0.508	0.258	-1.391	0.198
	VO ₂ maxLBM			-1.473	0.175
<i>2hr serum insulin</i>	% body fat	0.764	0.584	1.824	0.106
	VO ₂ maxLBM			-1.794	0.111
<i>2hr serum glucose</i>	% body fat	0.496	0.246	-0.801	0.446
	VO ₂ maxLBM			-1.608	0.146

10.6 Appendix B – Study 2

Table 0-7 – Participant physical characteristics (non-Māori)

Name	Age	Height	Weight	BMI	% BF (Siri)	LBM
W	32	1.81	69.3	21.2	16.3	57.8
WR	25	1.94	75.8	20.1	13.5	65.5
NC	23	1.74	86.7	28.6	21.5	68.1
SW	24	1.83	90.9	27.1	20.5	72.3
SC	25	1.82	87.6	26.4	19.0	70.9
GE	32	1.67	62.4	22.4	8.0	57.2
DM	35	1.84	83.2	24.6	22.0	64.4
CM	29	1.81	84.5	25.8	14.0	72.4
MV	24	1.79	81.9	25.6	13.0	71.0
AR	35	1.75	76.0	24.8	22.0	59.5
KM	18	1.78	66.2	20.9	5.5	62.4
JG	28	1.81	70.7	21.6	15.0	53.5
TM	28	1.67	62.8	22.5	15.0	59.8
BG	28	1.75	72.0	23.5	18.0	59.0
CB	20	1.85	98.2	28.7	25.0	73.2
KM	19	1.73	72.5	24.2	17.0	60.1
JM	28	1.78	68.6	21.7	16.0	56.7
Mean	28	1.81	80.1	24.5	16.9	66.1

Table 0-8 – Participant physical characteristics (Māori)

Name	Age	Height	Weight	BMI	% BF (Siri)	LBM
RP	26	1.78	80.0	25.2	24.5	60.6
ES	25	1.81	95.2	29.1	21.7	74.3
AW	32	1.84	115.4	34.1	28.7	82.3
KT	36	1.81	97.8	29.9	30.7	68.2
WM	28	1.79	94.7	29.6	25.3	71.0
KT	37	1.81	101.2	30.9	11.7	89.1
PT	26	1.84	83.4	24.6	15.0	71.0
CT	23	1.88	94.6	26.8	21.0	74.7
BE	34	1.82	85.4	25.8	20.0	68.3
IW	25	1.95	110.2	29.0	28.0	79.7
LR	25	1.70	111.3	38.5	33.0	74.4
ND	30	1.70	84.8	29.3	23.3	65.0
LT	28	1.87	118.6	33.9	30.5	82.2
ST	23	1.81	82.2	25.1	15.5	71.8
CW	22	1.83	88.6	26.5	22.0	68.8
CW	23	1.79	100.0	31.2	21.0	79.7
NP	32	1.59	61.0	24.1	14.0	52.6
JC	30	1.87	107.3	30.7	22.0	83.9
HM	18	1.74	69.4	22.9	8.0	63.7
Mean	28	1.80	94.4	29.0	22.7	72.6

Table 0-9 – Blood Pathology (Māori)

Name	Ins pM	Ins 2 hr	Glucose	2 hr Glucose	Total Chol	HDL	LDL	Chol/LDL	Ins iU	HOMA
RP	75	118	4.0	5.7	-	-	-	-	10.8	1.92
ES	32	32	4.7	5.0	-	-	-	-	4.6	0.96
AW	111	308	5.1	5.3	5.8	0.9	3.8	6.2	16.0	3.62
KT	46	99	4.1	5.1	3.9	1.4	2.2	2.8	6.6	1.21
WM	53	68	4.9	2.6	-	-	-	-	7.6	1.66
KT	138	329	5.6	4.6	-	-	-	-	19.9	4.95
PT	40	41	4.5	3.5	-	-	-	-	5.8	1.15
CT	35	127	4.4	-	-	-	-	-	5.0	0.99
BE	35	61	4.2	5.6	-	-	-	-	5.0	0.94
IW	33	39	3.6	5.3	6.6	1.1	4.6	5.8	4.8	0.76
LR	103	453	5.1	6.5	4.0	0.8	2.6	4.9	14.8	3.36
ND	52	80	5.1	3.3	-	-	-	-	7.5	1.70
LT	105	-	5.4	4.5	-	-	-	-	15.1	3.63
ST	41	-	4.6	2.7	-	-	-	-	5.9	1.21
CW	106	-	4.8	4.2	-	-	-	-	15.3	3.26
CW	76	-	4.7	-	-	-	-	-	10.9	2.29
NP	12	136	4.6	4.2	5.8	1.1	4.4	5.3	1.7	0.35
JC	40	202	5.3	-	5.5	0.8	3.9	6.8	5.8	1.36
HM	33	156	5.2	4.1	3.4	0.4	2.7	8.0	4.8	1.10
Mean	63.6	147.8	4.7	4.5	5.2	1.1	3.5	5.0	9.2	2.00

Table 0-10 – Blood Pathology (non-Māori)

Name	Ins pM	Ins 2 hr	Glucose	2 hr Glucose	Total Chol	HDL	LDL	Chol/LDL	Ins iU	HOMA
W	33	-	5.0	-	-	-	-	-	4.8	1.07
WR	81	90	4.7	4.5	3.9	1.0	2.5	4.1	11.7	2.44
NC	81	264	4.6	5.0	4.8	1.1	3.2	4.4	11.7	2.38
SW	41	17	4.9	6.1	5.5	1.2	3.7	4.6	5.9	1.29
SC	47	158	4.8	4.0	4.8	0.7	3.5	7.2	6.8	1.44
GE	41	138	5.0	4.2	4.3	1.0	3.0	4.5	5.9	1.31
DM	50	42	4.8	2.7	4.2	1.6	2.3	2.6	7.2	1.54
CM	165	-	5.2	3.8	3.3	0.8	2.1	4.3	23.8	5.49
MV	42	-	4.8	4.3	5.2	0.9	4.1	6.2	6.0	1.29
AR	8	32	4.5	1.7	4.4	1.0	3.1	4.4	1.2	0.23
KM	29	199	4.5	4.5	4.5	1.0	3.1	4.4	4.2	0.84
JG	16	57	4.5	3.8	4.4	1.1	3.0	4.0	2.3	0.46
TM	13	54	4.7	3.5	4.3	1.3	2.6	3.4	1.9	0.39
BG	49	209	5.2	4.1	4.1	0.7	3.0	5.5	7.1	1.63
CB	56	49	5.0	3.2	4.8	1.0	3.3	4.9	8.1	1.79
KM	22	113	4.9	4.7	4.5	1.0	2.9	4.7	3.2	0.69
JM	17	122	4.5	4.1	4.6	1.1	3.2	4.4	2.4	0.49
Mean	47	110	4.8	4.0	4.5	1.0	3.0	4.6	6.7	1.52

Table 0-11 – Aerobic capacity (Māori)

Name	VO2max	Vo2max/kg	VO2max/LBM
RP	3520	44.00	58.09
ES	3852	40.46	51.84
AW	4674	40.50	56.79
KT	4108	42.00	60.23
WM	4728	49.93	66.59
KT	4007	39.59	44.97
PT	3460	41.49	48.73
CT	3649	38.57	48.85
BE	3868	45.29	56.63
IW	4506	40.89	56.54
LR	3667	32.95	49.29
ND	3940	46.46	60.62
LT	3808	32.11	46.33
ST	3792	46.13	52.81
CW	4000	45.15	58.14
CW	4139	41.39	51.93
NP	3267	53.59	62.11
JC	5136	47.87	61.22
HM	3075	44.30	48.27
Mean	3982	42.70	54.55

Table 0-12 – Aerobic capacity (non-Māori)

Name	VO2max	Vo2max/kg	VO2max/LBM
W	3846	55.50	66.54
WR	3408	44.96	52.03
NC	4213	48.59	61.86
SW	4672	51.40	64.62
SC	4001	45.67	56.43
GE	3221	51.62	56.31
DM	3481	41.84	54.05
CM	4059	48.04	56.06
MV	3704	45.23	52.17
AR	3572	47.00	60.03
KM	2723	41.13	43.64
JG	4591	64.92	85.81
TM	4244	67.58	70.97
BG	3521	48.90	59.68
CB	4647	47.32	63.48
KM	3788	52.25	63.03
JM	3802	55.42	67.05
Mean	3853	50.43	60.81

10.7 Appendix C – Study 3

Table 0-13 – non-Māori participants

Name	Age	Height	Weight	BMI	% BF UWW	LBM	% BF BIA	BIA Lean Mass
W	32	1.81	69.3	21.2	16.3	57.8	14.0	59.6
WR	25	1.94	75.8	20.1	13.5	65.5	9.5	68.6
NC	23	1.74	86.7	28.6	21.5	68.1	17.6	71.2
SW	24	1.83	90.9	27.1	20.5	72.3	14.6	77.4
SC	25	1.82	87.6	26.4	19.0	70.9	18.7	71.2
GE	32	1.67	62.4	22.4	8.0	57.2	12.0	54.6
DM	35	1.84	83.2	24.6	22.0	64.4	15.2	70.7
CM	29	1.81	84.5	25.8	14.0	72.4	12.4	72.2
MV	24	1.79	81.9	25.6	13.0	71.0	13.1	72.8
AR	35	1.75	76.0	24.8	22.0	59.5	9.6	65.2
KM	18	1.78	66.2	20.9	5.5	62.4	10.8	58.3
JG	28	1.81	70.7	21.6	15.0	53.5	8.4	64.2
TM	28	1.67	62.8	22.5	15.0	59.8	13.5	54.1
BG	28	1.75	72.0	23.5	18.0	59.0	21.4	57.0
DN	26	1.82	71.5	21.6	21.3	56.3	13.1	62.1
JM	28	1.78	68.6	21.7	16.0	56.7	17.3	56.8
AW	26	1.82	78.1	23.6	8.5	71.3	10.6	69.8
KE	23	1.85	88.8	25.9	19.5	71.5	17.3	73.4
KC	21	1.78	71.6	22.6	9.7	68.4	7.1	66.5
JH	28	1.81	86.8	26.5	19.7	70.0	15.9	73.0
Mean	27	1.79	76.8	23.9	15.9	64.4	13.6	65.9

Table 0-14 – Māori participants

Name	Age	Height	Weight	BMI	% BF UWW	LBM	% BF BIA	BIA Lean Mass
RP	26	1.78	80.0	25.2	24.5	60.6	16.7	66.6
ES	25	1.81	95.2	29.1	21.7	74.3	22.4	73.9
AW	32	1.84	115.4	34.1	28.7	82.3	24.8	86.8
KT	36	1.81	97.8	29.9	30.7	68.2	25.2	73.1
WM	28	1.79	94.7	29.6	25.3	71.0	22.8	73.2
KT	37	1.81	101.2	30.9	11.7	89.1	29.2	71.7
PT	26	1.84	83.4	24.6	15.0	71.0	10.9	74.3
CT	23	1.88	94.6	26.8	21.0	74.7	14.7	80.7
BE	34	1.82	85.4	25.8	20.0	68.3	12.8	74.4
PH	28	1.84	97.1	28.7	20.7	76.9	14.7	82.8
AR	20	1.76	112.0	36.2	25.5	83.8	26.3	82.5
IW	25	1.95	110.2	29.0	28.0	79.7	18.0	90.1
ND	30	1.70	84.8	29.3	23.3	65.0	20.0	68.0
LT	28	1.87	118.6	33.9	30.5	82.2	24.2	89.6
ST	23	1.81	82.2	25.1	15.5	71.8	11.2	72.7
CW	22	1.83	88.6	26.5	22.0	68.8	17.1	72.8
CW	23	1.79	100.0	31.2	21.0	79.7	21.4	78.0
NP	32	1.59	61.0	24.1	14.0	52.6	10.3	54.3
HM	18	1.74	69.4	22.9	8.0	63.7	10.5	61.4
SB	22	1.77	99.1	31.6	31.3	67.9	31.2	68.2
DA	26	1.79	118.0	36.8	29.5	83.6	27.0	86.7
JE	20	1.76	107.5	34.7	28.5	77.1	32.0	73.0
DP	27	1.88	84.8	24.0	18.0	69.6	13.7	73.2
LB	22	1.74	70.50	23.4	16.5	58.40	16.6	58.9
RW	23	1.82	88.20	26.6	19.5	63.40	16.1	72.0
SW	25	1.81	94.50	28.8	29.5	66.25	23.3	72.0
BC	36	1.83	91.40	27.3	22.0	70.65	19.9	72.9
JR	27	1.82	152.70	46.1	40.0	91.20	44.4	84.6
Mean	27	1.80	95.7	29.4	22.9	72.6	20.6	74.6

10.8 Appendix D – Study 4

Table 0-15 – Participant physical characteristics

Name	Age	Height	Weight	BMI	% BF
NP	32	1.59	61.0	24.1	14.0
HM	18	1.74	69.4	22.9	8.0
WR	25	1.94	75.8	20.1	13.5
JM	28	1.78	68.6	21.7	16.0
DN	26	1.82	71.5	21.6	21.3
IW	25	1.95	110.2	29.0	28.0
GE	32	1.67	62.4	22.4	8.0
CM	29	1.81	84.5	25.8	14.0
TM	28	1.67	62.8	22.5	15.0
Mean	27	1.77	74.0	23.3	15.3

Table 0-16 – T50 & TPF

Name	Twitch#	Day 1		Day 2		Day 3	
		T50 (Lillioja et al.)	TPF (Lillioja et al.)	T50	TPF	T50	TPF
CM	1	0.065	0.109	0.061	0.101	0.064	0.104
	2	0.065	0.109	0.061	0.101	0.063	0.105
	3	0.065	0.109	0.061	0.102	0.063	0.105
	4	0.065	0.108	0.061	0.101	0.063	0.104
	5	0.064	0.107	0.061	0.101	0.063	0.105
GE	1	0.062	0.104	0.060	0.106	0.062	0.101
	2	0.062	0.104	0.058	0.100	0.062	0.101
	3	0.062	0.103	0.058	0.100	0.062	0.101
	4	0.062	0.104	0.059	0.099	0.061	0.100
	5	0.061	0.103	0.058	0.100	0.061	0.101
TM	1	0.064	0.105	0.065	0.108	0.066	0.109
	2	0.064	0.104	0.064	0.107	0.066	0.111
	3	0.064	0.105	0.065	0.107	0.066	0.109
	4	0.064	0.104	0.065	0.108	0.066	0.109
	5	0.065	0.107	0.065	0.107	0.065	0.106
DN	1	0.066	0.106	0.073	0.106	0.068	0.108
	2	0.066	0.104	0.074	0.105	0.067	0.106
	3	0.066	0.102	0.073	0.105	0.067	0.105
	4	0.066	0.106	0.074	0.106	0.066	0.106
	5	0.066	0.106	0.074	0.106	0.066	0.106
HM	1	0.056	0.098	0.059	0.097	0.060	0.100
	2	0.058	0.102	0.060	0.101	0.060	0.101
	3	0.061	0.106	0.061	0.101	0.060	0.100
	4	0.058	0.108	0.062	0.100	0.060	0.102
	5	0.058	0.107	0.060	0.099	0.060	0.100

Table 0-17 – T50 & TPF (continued)

Name	Twitch#	Day 1		Day 2		Day 3	
		T50 (Lillioja et al.)	TPF (Lillioja et al.)	T50	TPF	T50	TPF
WR	1	0.062	0.100	0.059	0.099	0.065	0.104
	2	0.062	0.099	0.058	0.097	0.065	0.105
	3	0.061	0.098	0.059	0.098	0.065	0.104
	4	0.062	0.100	0.058	0.099	0.065	0.106
	5	0.062	0.099	0.058	0.098	0.065	0.105
NP	1	0.062	0.134	0.061	0.138	0.062	0.135
	2	0.062	0.133	0.063	0.141	0.062	0.136
	3	0.062	0.134	0.063	0.142	0.062	0.136
	4	0.062	0.135	0.062	0.141	0.062	0.135
	5	0.062	0.134	0.062	0.136	0.062	0.133
JM	1	0.060	0.106	0.062	0.106	0.062	0.105
	2	0.060	0.106	0.062	0.108	0.062	0.105
	3	0.060	0.106	0.062	0.109	0.062	0.105
	4	0.060	0.106	0.062	0.110	0.062	0.107
	5	0.060	0.108	0.062	0.109	0.062	0.103
IW	1	0.065	0.115	0.064	0.115	0.064	0.113
	2	0.064	0.113	0.066	0.113	0.064	0.112
	3	0.066	0.115	0.066	0.113	0.064	0.113
	4	0.066	0.113	0.065	0.113	0.064	0.113
	5	0.065	0.114	0.065	0.113	0.064	0.112

Table 0-18 – ANOVA table for TPF & T50

ANOVA for TTP					
Source of variation	Degrees of freedom	SSQ	MSQ	F	Significance (p)
Between subjects	8	0.0145758	0.0018220	61.1409	<0.001
Between days	2	0.0000279	0.0000140	0.4698	NS
Subject/Day Error (1)	16	0.0004772	0.0000298	17.5294	<0.001
Between trials	26	0.0150809	0.0005800		
Test	4	0.0000061	0.0000015	0.8824	NS
Subject/Test	32	0.0001018	0.0000032	1.8824	NS
Day/Test	8	0.0000164	0.0000020	1.1765	NS
Error (2)	64	0.0001098	0.0000017		
Total	134	0.0153151			
ANOVA for T50					
Between subjects	8	0.0009534	0.0001192	5.2743	<0.01
Between days	2	0.0000297	0.0000148	0.6549	NS
Subject/Day Error (1)	16	0.0003621	0.0000226	75.3333	<0.001
Between trials	26	0.0013452	0.0000517		
Test	4	0.0000021	0.0000005	1.6667	NS
Subject/Test	32	0.0000142	0.0000004	1.3333	NS
Day/Test	8	0.0000040	0.0000005	1.6667	NS
Error (2)	64	0.0000217	0.0000003		
Total	134				

10.9 Appendix E – Study 5

Table 0-19 – Participant physical characteristics

Name	Age	Height	Weight	BMI	% BF
WM	28	1.79	94.7	29.6	25.3
PT	26	1.84	83.4	24.6	15.0
CT	23	1.88	94.6	26.8	21.0
LT	28	1.87	118.6	33.9	30.5
ST	23	1.81	82.2	25.1	15.5
NP	32	1.59	61.0	24.1	14.0
HM	18	1.74	69.4	22.9	8.0
WR	25	1.94	75.8	20.1	13.5
JM	28	1.78	68.6	21.7	16.0
DN	26	1.82	71.5	21.6	21.3
KT	36	1.81	97.8	29.9	30.7
KT	37	1.81	101.2	30.9	11.7
BE	34	1.82	85.4	25.8	20.0
IW	25	1.95	110.2	29.0	28.0
LR	25	1.70	111.3	38.5	33.0
GE	32	1.67	62.4	22.4	8.0
CM	29	1.81	84.5	25.8	14.0
MV	24	1.79	81.9	25.6	13.0
KM	18	1.78	66.2	20.9	5.5
TM	28	1.67	62.8	22.5	15.0
BG	29	1.75	72.0	23.5	18.0
Mean	27	1.79	83.6	26.0	18.0

Table 0-20 – Blood pathology

Name	Ins pM	Ins 2 hr	Glucose	2 hr Gluc	HOMA
WM	53	68	4.9	2.6	1.66
PT	40	41	4.5	3.5	1.15
CT	35	127	4.4	-	0.99
LT	105	-	5.4	4.5	3.63
ST	41	-	4.6	2.7	1.21
NP	12	136	4.6	4.2	0.35
HM	33	156	5.2	4.1	1.10
WR	81	90	4.7	4.5	2.44
JM	17	122	4.5	4.1	0.49
DN	56	271	5.5	5.0	1.97
KT	46	99	4.1	5.1	1.21
KT	138	329	5.6	4.6	4.95
BE	35	61	4.2	5.6	0.94
IW	33	39	3.6	5.3	0.76
LR	103	453	5.1	6.5	3.36
GE	41	138	5.0	4.2	1.31
CM	165	-	5.2	3.8	5.49
MV	42	-	4.8	4.3	1.29
KM	29	199	4.5	4.5	0.84
TM	13	54	4.7	3.5	0.39
BG	49	209	5.2	4.1	1.63
Mean	56	152	4.8	4.3	1.77

Table 0-21 – Aerobic capacity, fatigue test and twitch data

Name	VO2lbn	TTP	TT50%	Fmax	Fmean of 56-60 (T60)	T60 as percentage of max (%)	Diff (Fmax-T60)	Percentage decline
WM	66.59	0.115	0.065	215.5	92.9	43.1	122.6	56.9
PT	48.73	0.101	0.060	231.7	62.0	26.7	169.7	73.3
CT	48.85	0.106	0.064	233.4	87.8	37.6	145.6	62.4
LT	46.33	0.127	0.068	288.2	129.6	45.0	158.6	55.0
ST	52.81	0.106	0.060	211.9	97.6	46.0	114.3	54.0
NP	62.11	0.134	0.062	144.4	56.5	39.2	87.9	60.8
HM	48.27	0.104	0.058	178.3	83.7	46.9	94.6	53.1
WR	52.03	0.099	0.062	161.0	77.9	48.4	83.1	51.6
JM	67.05	0.106	0.060	187.9	81.8	43.6	106.1	56.4
DN	64.69	0.106	0.066	167.5	38.0	22.7	129.5	77.3
KT	60.23	0.116	0.067	266.6	105.9	39.7	160.7	60.3
KT	44.97	0.110	0.066	280.3	91.3	32.6	189.0	67.4
BE	56.63	0.111	0.064	201.8	75.1	37.2	126.7	62.8
IW	56.54	0.114	0.065	233.9	82.8	35.4	151.1	64.6
LR	49.29	0.113	0.071	294.0	77.5	26.3	216.5	73.7
GE	56.31	0.104	0.062	182.4	80.6	44.2	101.8	55.8
CM	56.06	0.108	0.065	218.7	82.0	37.5	136.7	62.5
MV	52.17	0.101	0.063	201.0	75.6	37.6	125.4	62.4
KM	43.64	0.104	0.063	156.3	44.7	28.6	111.6	71.4
TM	70.97	0.105	0.064	171.7	104.9	61.1	66.8	38.9
BG	59.68	0.107	0.065	153.5	69.5	45.3	84.0	54.7
Mean	55.43	0.109	0.064	208.6	80.8	39.3	127.7	60.7

10.10 Appendix F – Participant Information Forms & Health

Questionnaires



10.11 Pre-Exercise Health Screening Questionnaire

Name: _____

Address: _____

Phone: _____

v

Age: _____

Please read the following questions carefully. If you have any difficulty, please advise the medical practitioner, nurse or exercise specialist who is conducting the exercise test.

Please answer all of the following questions by ticking only one box for each question:

This questionnaire has been designed to identify the small number of persons (15-69 years of age) for whom physical activity might be inappropriate. The questions are based upon the Physical Activity Readiness Questionnaire (PAR-Q), originally devised by the British Columbia Dept of Health (Canada), as revised by ¹Thomas *et al.* (1992) and ²Cardinal *et al.* (1996), and with added requirements of the Massey University Human Ethics Committee. The information provided by you on this form will be treated with the strictest confidentiality.

Qu 1. Has your doctor ever said that you have a heart condition and that you should only do physical activity recommended by a doctor?

Yes No

Qu 2. Do you feel a pain in your chest when you do physical activity?

Yes No

Qu 3. In the past month have you had chest pain when you were not doing physical activity?

Yes No

Qu 4. Do you lose your balance because of dizziness or do you ever lose consciousness?

Yes No

Qu 5. Is your doctor currently prescribing drugs (for example, water pills) for your blood pressure or heart condition?

Yes No

Qu 6. Do you have a bone or joint problem that could be made worse by vigorous exercise?

Yes No

Qu 7. Do you know of any other reason why you should not do physical activity?

Yes No

Qu 8. Have any immediate family had heart problems prior to the age of 60?

Yes No

Qu 9. Have you been hospitalised recently?

Yes No

Qu 11. Do you have any infectious disease that may be transmitted in blood?

Yes No

You should be aware that even amongst healthy persons who undertake regular physical activity there is a risk of sudden death during exercise. Though extremely rare, such cases can occur in people with an undiagnosed heart condition. If you have any reason to suspect that you may have a heart condition that will put you at risk during exercise, you should seek advice from a medical practitioner before undertaking an exercise test.

I have read, understood and completed this questionnaire.

Signature: _____ Date: _____

References

1. Thomas S, Reading J and Shephard RJ. Revision of the Physical Activity Readiness Questionnaire (PAR-Q). *Can J Sport Sci* 17(4): 338-345.
2. Cardinal BJ, Esters J and Cardinal MK. Evaluation of the revised physical activity readiness questionnaire in older adults. *Med Sci Sports Exerc* 28(4): 468-472



Participant Information Summary

Insulin resistance in Māori males.

Who are the researchers and where can we be contacted?

Dr Stephen Stannard	Prof. Chris Cunningham	Isaac Warbrick
Institute of Food Nutrition & Human Health Massey University Palmerston North	Research Centre for Māori Health & Development, Massey University, Wellington	Research Centre for Māori Health & Development, Massey University, Wellington

What is this study about? This study is about type-2 diabetes which affects Māori at a greater rate than non-Māori. Specifically we will investigate whether the increased incidence of type-2 diabetes seen in the Maori population may involve elevated muscle lipid concentrations. Muscle lipid is an important ‘fuel’ for exercising muscle and a better understanding of its role in producing insulin resistance may enable the development of more effective strategies for preventing type-2 diabetes.

If you choose to participate in this study, you will:

- Complete a health questionnaire
 - Complete an exercise test of your oxygen consumption on a treadmill.
 - Perform an oral glucose tolerance test.
 - Provide two 10 ml venous blood samples.
 - Have your body composition assessed via underwater weighing and Bioelectrical Impedance®
- (Full details of these procedures are given in the attached pages).

What are the benefits of the research?

This research will give you the opportunity to have an input into a study which is designed to understand the risks Māori have in relation to type-2 diabetes.

Your Rights as Participants

You are under no obligation to accept this invitation. If you decide to participate, you have the right to:

- decline to answer any particular question;
- withdraw from the study;
- ask any questions about the study at any time during participation;
- provide information on the understanding that your name will not be used unless you give permission to the researcher;
- be given access to a summary of the project findings when it is concluded.

Project Contacts

Please contact any of the researchers if you have any concerns or require further information.



Participant Full Information Sheet

Insulin resistance in Māori males.

Aims of this study

This research will use techniques to measure the oxidative capacity and insulin resistance for young Māori men. Blood samples will be taken to estimate insulin sensitivity, blood lipid profile, glucose tolerance, C-reactive protein and other markers of insulin resistance.

Participation

Participants in this study must be male Māori who are physically active, aged between 18 and 45. You will be fully informed of your involvement in this study and will complete a Medical History questionnaire and a Physical Activity Readiness questionnaire (PAR Q) to assess your readiness to participate in the study. Following these, you will be required to sign a consent form after reading this “Participant Information Statement” and clarifying any matters that remain unclear.

You will be asked to undergo one maximal exercise bout, two measures of body composition, an oral glucose tolerance test, and to give two venous blood samples. It should be emphasised that you may withdraw from this study any time you wish. If you choose to do so, there will be no compromise in the relationship between you and the investigators. If you are a Massey University student, participation in, or withdrawal from the study will not influence your academic progress in any manner. In deciding to partake in this study, you will be subjecting yourself to the following requirements:

1. Screening Procedures

You will answer a demographic, Medical History and Physical Activity Readiness questionnaire.

2. Testing Procedures

Body Composition: You will attend the RCMHD Research Laboratory at Massey University, Wellington Campus and have your body composition assessed in an underwater weighing tank. This will require you to dress down to a pair of swimming shorts only and sit immerse yourself in a warm water tank. If you feel uncomfortable, you can exit the tank anytime during the procedure at your own request.

VO_{2max} test: After the underwater weighing measurement, you will undergo an exercise test on a treadmill to measure your maximal oxygen uptake (VO_{2max}). You will begin the test by walking on the treadmill at a slow speed. Each two minutes the treadmill speed and gradient will be increased until you are unable to continue.

During exercise, your oxygen consumption will be measured by collected expired gases from your mouth using the Moxus gas analysis system. This equipment measures volumes of air and oxygen passing into and out of your lungs and from this we can determine the rate at which you use oxygen. Heart rate is measured electronically using the commonly available Polar chest band and wristwatch combination. All exercise testing will be performed on the research quality Woodway treadmill located in the RCMHD Research Laboratory at Massey University, Wellington Campus.

Oral glucose tolerance test and venous blood sample: Prior to this test, a resting 10 ml blood sample will be taken from a vein in your arm by an experienced staff member. The sample will be frozen and later analysed for blood lipids, glucose, C-reactive protein and insulin. The glucose tolerance test involves drinking a sugary (glucose) solution before breakfast. One further blood sample will be taken exactly two hours after drinking the solution to test your blood glucose and insulin levels again. Following analysis, any remaining blood will be returned to the earth by our kaumätua following due tikanga.

Potential Risks and Discomforts

As with any research of this nature, there are some potential risks and discomforts, which you should be aware of. The researchers will attempt to minimise these through careful, consistent monitoring of your response to the testing procedures. Every effort will be made by the researchers to ensure your safety, comfort and familiarity with the testing procedures.

a. Blood test and oral glucose tolerance test: Sampling of venous blood may cause a degree of transient discomfort, and some bruising may occur at the point of insertion up to 48 hours afterwards. This discomfort is of no lasting consequence. There is also a slight risk of fainting during blood sampling. The risks and discomforts will be minimised, as the procedure will be performed under sterile conditions by highly experienced staff.

b. Exercise procedure: The exercise procedure during the VO_{2max} test will not be easy, but the time of exercise at high intensity is short (less than 2 minutes) and you are able to terminate the test at any time at which you feel fatigued. This test is often performed on both athletes and sedentary

individuals to measure aerobic fitness. As in any physical activity, there is a very small possibility of injuries that include, but are not restricted to, muscle, ligament or tendon damage, breathing irregularities and dizziness. However, the exercise protocols are commonly performed in exercise physiology laboratories and potential risks to participants have been minimised.

c. Body composition. There are no risks associated with these techniques. During the underwater weighing you will be fully submerged in a warm water bath, and must hold your breath for about 20 second whilst the measurements are taken. This is a little disconcerting and if you feel uncomfortable you are free to exit the water at any time. During this procedure you will need to wear a swimming costume.

Right of withdrawal

Your participation in this study is completely voluntary. You may choose to withdraw from the study at any time for any reason. By signing the consent form you are indicating that the tests and procedures of this study have been explained to you and understood by you.

Inquiries

All inquiries regarding requirements and procedures used in this study are encouraged. Please contact either of the investigators below if you have any questions.

Mr Isaac Warbrick	(06) 350 5799 ext 5264	I.Warbrick@massey.ac.nz
Dr Stephen Stannard:	(06) 350 5799 ext 7465	S.Stannard@massey.ac.nz
Prof. Chris Cunningham:	(04) 801 5799 ext 6027	C.W.Cunningham@massey.ac.nz

This project has been reviewed and approved by the Massey University Human Ethics Committee, PN Application 04/110.

If you have any concerns about the conduct of this research, please contact Professor John O'Neill, Chair, Massey University

Campus Human Ethics Committee: Palmerston North, telephone 06 350 5249, email humanethicspn@massey.ac.nz.



Research Centre for Māori Health & Development
College of Humanities & Social Sciences

Participant Information Summary

Insulin resistance in Maori & non-Maori men

Who are the researchers and where can we be contacted?

Mr Isaac Warbrick	Prof. Chris Cunningham	Dr Stephen Stannard
Research Centre for Māori Health & Development, Massey University, Wellington	Research Centre for Māori Health & Development, Massey University, Wellington	Institute of Food Nutrition & Human Health Massey University Palmerston North

What is this study about? This study is about type-2 diabetes which affects Māori at a greater rate than non-Māori. Specifically we will investigate whether protection from type-2 diabetes seen in the Maori population may involve good aerobic fitness of your lean body mass which is mostly muscle, compared to non-Maori. Muscle has an important role in the development of insulin resistance and ensuring aerobic fitness of muscle may be an important strategy to prevent type-2 diabetes.

If you choose to participate in this study, you will:

- Complete a health questionnaire
 - Complete an exercise test of your oxygen consumption on a treadmill.
 - Perform an oral glucose tolerance test.
 - Provide two 10 ml venous blood samples.
 - Have your body composition assessed via underwater weighing and Bioelectrical Impedance®
- (Full details of these procedures are given in the attached pages).

What are the benefits of the research?

This research will give you the opportunity to have an input into a study which is designed to understand the risks Māori have in relation to type-2 diabetes and why they are more susceptible to the disease than non-Maori.

Your Rights as Participants

You are under no obligation to accept this invitation. If you decide to participate, you have the right to:

- decline to answer any particular question;
- withdraw from the study;
- ask any questions about the study at any time during participation;
- provide information on the understanding that your name will not be used unless you give permission to the researcher;
- be given access to a summary of the project findings when it is concluded.

Project Contacts

Please contact any of the researchers if you have any concerns or require further information.

This project has been reviewed and approved by the Massey University Human Ethics Committee, PN Application 07/60. If you have any concerns about the conduct of this research, please contact Professor John O'Neill, Chair, Massey University Campus Human Ethics Committee: Palmerston North, telephone 06 350 5249, email humanethicspn@massey.ac.nz.



Research Centre for Māori Health & Development
College of Humanities & Social Sciences

Participant Full Information Sheet

Insulin resistance in Māori & non-Māori men

Aims of this study

This research will use techniques to measure aerobic capacity and insulin sensitivity in a cohort of young Māori and non-Maori men. Blood samples will be taken to estimate insulin sensitivity, blood lipid profile, glucose tolerance, C-reactive protein and other markers of insulin resistance. A treadmill exercise test will be used to assess aerobic fitness.

Participation

Participants in this study will be part of two groups i) Maori and ii) non-Maori. Participants will be male who are physically active, aged between 18 and 45. You will be fully informed of your involvement in this study and will complete a Medical History questionnaire and a Physical Activity Readiness questionnaire (PAR Q) to assess your readiness to participate in the study. Following these, you will be required to sign a consent form after reading this “Participant Information Statement” and clarifying any matters that remain unclear.

You will be asked to undergo one maximal exercise test on a treadmill, two measures of body composition, an oral glucose tolerance test, and to give two venous blood samples. It should be emphasised that you may withdraw from this study any time you wish. If you choose to do so, there will be no compromise in the relationship between you and the investigators. If you are a Massey University student, participation in, or withdrawal from the study will not influence your academic progress in any manner. In deciding to partake in this study, you will be subjecting yourself to the following requirements:

1. Screening Procedures

You will answer a demographic, Medical History and Physical Activity Readiness questionnaire.

2. Testing Procedures

Venous blood sample and oral glucose tolerance test: Prior to eating breakfast in the morning you will attend the Medlab Clinic to provide a 10 ml blood sample. This will be taken from a vein in your arm by an experienced venepuncturist. The sample will be frozen and later analysed for blood lipids, glucose, C-reactive protein and insulin. The glucose tolerance test will then be performed with involves drinking a sugary (glucose) solution. One further blood sample will be taken exactly two hours after drinking the solution to test your blood glucose and insulin levels again. We will then provide you with breakfast. Following analysis, any remaining blood will be returned to the earth by our kaumätua following due tikanga.

Body Composition: You will attend the RCMHD Research Laboratory at Massey University, Wellington Campus and have your body composition assessed in an underwater weighing tank. This will require you to dress down to a pair of swimming shorts only and sit immerse yourself in a warm water tank. You will be asked to breathe out then bend forward to fully submerge your body for approximately twenty seconds whilst we measure your body weight underwater. If you feel uncomfortable, you can exit the tank anytime during the procedure at your own request.

VO_{2max} test: After the underwater weighing measurement, you will undergo an exercise test on a treadmill to measure your maximal oxygen uptake (VO_{2max}).

You will begin the test by walking on the treadmill at a slow speed. Each two minutes the treadmill speed and gradient will be increased until you are unable to continue.

During exercise, your oxygen consumption will be measured by collecting expired gases from your mouth using the Moxus gas analysis system. This equipment measures volumes of air and oxygen passing into and out of your lungs and from this we can determine the rate at which you use oxygen. It will require you to breathe through a mouthpiece during the test and have a clip on your nose. During the test we will measure your heart rate using the commonly available Polar chest band and wristwatch combination. All exercise testing will be performed on the research quality Woodway treadmill located in the RCMHD Research Laboratory at Massey University, Wellington Campus.

Potential Risks and Discomforts

As with any research of this nature, there are some potential risks and discomforts, which you should be aware of. The researchers will attempt to minimise these through careful, consistent monitoring of

your response to the testing procedures. Every effort will be made by the researchers to ensure your safety, comfort and familiarity with the testing procedures.

a. Blood test and oral glucose tolerance test: Sampling of venous blood may cause a degree of transient discomfort, and some bruising may occur at the point of insertion up to 48 hours afterwards. This discomfort is of no lasting consequence. There is also a slight risk of fainting during blood sampling. The risks and discomforts will be minimised, as the procedure will be performed under sterile conditions by people highly experienced in this procedure.

b. Exercise procedure: The exercise procedure during the $\text{VO}_{2\text{max}}$ test will not be easy, but the time of exercise at high intensity is short (less than 3 minutes) and you are able to terminate the test at any time at which you feel fatigued. This test is often performed on both athletes and sedentary individuals to measure aerobic fitness. As in any physical activity, there is a very small possibility of injuries that include, but are not restricted to, muscle, ligament or tendon damage, breathing irregularities and dizziness. However, the exercise protocols are commonly performed in exercise physiology laboratories and potential risks to participants have been minimised.

c. Body composition. There are no risks associated with these techniques. During the underwater weighing you will be fully submerged in a warm water bath, and must hold your breath for about 20 second whilst the measurements are taken. This is a little disconcerting and if you feel uncomfortable you are free to exit the water at any time. During this procedure you will need to wear a swimming costume.

Right of withdrawal

Your participation in this study is completely voluntary. You may choose to withdraw from the study at any time for any reason. By signing the consent form you are indicating that the tests and procedures of this study have been explained to you and understood by you.

Inquiries

All inquiries regarding requirements and procedures used in this study are encouraged. Please contact either of the investigators below if you have any questions.

Mr Isaac Warbrick	(06) 350 5799 ext 5264	I.Warbrick@massey.ac.nz
Dr Stephen Stannard:	(06) 350 5799 ext 7465	S.Stannard@massey.ac.nz
Prof. Chris Cunningham:	(04) 801 5799 ext 6027	C.W.Cunningham@massey.ac.nz



10.12 Muscle contraction properties as predictors of insulin sensitivity in Māori and non-Māori men

10.13 Pre-Exercise Health Screening Questionnaire

Name: _____

Address: _____

Phone: _____

Age: _____

Please read the following questions carefully. If you have any difficulty, please advise the medical practitioner, nurse or exercise specialist who is conducting the exercise test.

Please answer all of the following questions by ticking only one box for each question:

This questionnaire has been designed to identify the small number of persons (15-69 years of age) for whom physical activity might be inappropriate. The questions are based upon the Physical Activity Readiness Questionnaire (PAR-Q), originally devised by the British Columbia Dept of Health (Canada), as revised by ¹Thomas *et al.* (1992) and ²Cardinal *et al.* (1996), and with added requirements of the Massey University Human Ethics Committee. The information provided by you on this form will be treated with the strictest confidentiality.

Qu 1. Has your doctor ever said that you have a heart condition and that you should only do physical activity recommended by a doctor?

Yes No

Qu 2. Has a member of your family died below the age of fifty (50) as a result of a heart condition?

Yes No

Qu 3. In the past month have you had chest pain when you were not doing physical activity?

Yes No

Qu 4. Do you have any current injury, bone or joint problem in your legs, or previous injury that could be affected by resistance exercise?

Yes No

Qu 5. Are you taking any prescribed medication?

Yes No

Qu 6. Do you know of any other reason why you should not do physical activity and/or resistance exercise?

Yes No

Qu7. Have you ever had persistent or regular back pain?

Yes No

Qu 8. Do you have a bleeding disorder?

Yes No

Qu9. Do you currently have, or have you ever had renal and/or hepatic disease?

Yes No

Qu 10. Are you diabetic?

Yes No

Qu 11. Have you had/do you have a medical condition that may effect your ability to sense pain or discomfort?

Yes No

Qu 12. Do you use a pacemaker?

Yes No

Qu 12. Do you have a skin allergy or condition that may be put yourself and others at risk during exercise testing?

Yes No

You should be aware that even amongst healthy persons who undertake regular physical activity there is a risk of sudden death during exercise. Though extremely rare, such cases can occur in people with an undiagnosed heart condition. If you have any reason to suspect that you may have a heart condition that will put you at risk during exercise, you should seek advice from a medical practitioner before undertaking an exercise test.

I have read, understood and completed this questionnaire.

Signature: _____ Date: _____

References

3. Thomas S, Reading J and Shephard RJ. Revision of the Physical Activity Readiness Questionnaire (PAR-Q). *Can J Sport Sci* 17(4): 338-345.
4. Cardinal BJ, Esters J and Cardinal MK. Evaluation of the revised physical activity readiness questionnaire in older adults. *Med Sci Sports Exerc* 28(4): 468-472



Participant Information Sheet

Muscle contraction properties as predictors of insulin sensitivity in Māori and non-Māori men

Who are the researchers and where can we be contacted?

Dr Stephen Stannard	Prof. Chris Cunningham	Isaac Warbrick
Institute of Food Nutrition & Human Health Massey University Palmerston North	Research Centre for Māori Health & Development, Massey University, Wellington	Research Centre for Māori Health & Development, Massey University, Wellington

What is this study about? This study is part of a doctoral thesis for the primary researcher, Isaac Warbrick. His doctoral research focuses on the development of insulin resistance and type-2 diabetes; a condition which affects Māori at a greater rate than non-Māori. Because insulin effectiveness in muscle is linked to skeletal muscle function we will investigate the relationship between blood markers of insulin resistance (a condition which leads to diabetes), and the peak power and contraction characteristics which are related to a persons muscle fibre type. Measures of peak muscle power will be obtained through a number of leg extensions performed on the Biodex, while twitch characteristics will be obtained through electrically stimulated contractions on specially designed apparatus. We also hope to compare our findings between Maori and non-Maori. Little is known regarding these relationships in Maori or whether they differ between ethnicities, but better understanding them may enable the development of more effective strategies for preventing type-2 diabetes in both groups.

Ethnicity of participants in this study will be self-identified by each individual participant.

If you choose to participate in this study, you will be invited to:

- Complete a health questionnaire

- Complete 3-5 leg extensions against resistance on the Biodex.
 - Participate in an oral glucose tolerance test.
 - Provide two 10 ml venous blood samples.
 - Complete 3-5 electrically stimulated leg extensions.
- (Full details of these procedures are given in the attached pages).

If any of the following apply:

- *You have a known heart or cardiovascular condition or if a member of your family died below the age of fifty (50) as a result of a heart condition.*
 - *You have any current or previous injury to your legs*
 - *You have had an injury or medical condition that you think may affect your ability to sense pain or discomfort.*
 - *You have ever had persistent or regular lower back pain.*
 - *You are taking prescribed medication.*
 - *You have cultural or religious sensitivities about human body measurements.*
 - *You have any other reason to consider that you are not in good health and of average, or better than average, fitness.*
 - *You have a bone or joint problem in your legs that could be made worse by resistance exercise*
 - *You are diabetic.*
 - *You have a bleeding disorder.*
 - *If you currently have or have had renal and/or hepatic disease*
 - *You use a pacemaker*
 - *You have a skin allergy or condition that may put yourself or others at risk*
- ...you should **NOT** participate in this project.

What are the benefits of the research?

This research will give you the opportunity to have an input into a study which is designed to understand the risks Māori and non-Maori have in relation to type-2 diabetes. In completing the study we will also be able to give you an indication of your (quadriceps) muscle fibre type.

Your Rights as a Participant

You are under no obligation to accept this invitation. If you decide to participate, you have the right to:

- decline to answer any particular question;
- withdraw from the study;
- ask any questions about the study at any time during participation;
- provide information on the understanding that your name will not be used unless you give permission to the researcher;
- be given access to a summary of the project findings when it is concluded.

Project Contacts

Please contact any of the researchers if you have any concerns or require further information

STUDY DETAILS

Aims of this study

This research will use laboratory techniques to measure the peak muscular force production and twitch characteristics during a leg extension, and how these measures relate to insulin resistance in both young Māori men and New Zealand non-Māori men. Fasting blood samples will be taken to estimate insulin sensitivity.

Participation

Participants in this study will be male Māori and non-Maori who are physically active, aged between 18 and 45. You will be fully informed of your involvement in this study and will be invited to complete a Medical History questionnaire and a Physical Activity Readiness questionnaire (PAR Q) to assess your readiness to participate in the study. Following these, you will be invited to sign a consent form after reading this “Participant Information Statement” and clarifying any matters that remain unclear.

You will be asked to undergo one bout of leg extensions on the Biodex, one bout of electrically stimulated contractions on the electrical stimulation rig, an oral glucose tolerance test, and to give two venous blood samples. It should be emphasised that you may withdraw from this study any time you wish. If you choose to do so, there will be no compromise in the relationship between you and the investigators. If you are a Massey University student, participation in, or withdrawal from the study will not influence your academic progress in any manner. In deciding to partake in this study, you will be subjecting yourself to the following requirements:

1. Screening Procedures

You will be invited to answer each of a Demographic, Medical History, and Physical Activity Readiness questionnaire.

2. Testing Procedures

Muscular Fatigue: Muscular Fatigue will be measured on the Biodex isometric dynamometer. You will be invited to sit in a chair while your lower leg is strapped to a mechanical arm that measures the velocity and force that your thigh muscle (quadriceps) is able to produce when you straighten your leg at maximal force against a known resistance for 60 repetitions. This is very similar to using a leg extension machine found in most gyms.

Muscle twitch characteristics: The twitch characteristics of the knee extensors (quadriceps) will be measured while you are seated in an adjustable straight-backed chair. During the test your ankle is strapped and the straps are connected to a load cell which records the forces produced by the quadriceps muscle during each electrical impulse. Electrical stimulation will be achieved by placing two large pads covered with electrode gel onto the skin on the belly of the Quadriceps muscles (front of thigh). A commercially available electrical stimulator will then deliver the impulses to the electrodes on your leg causing the quadriceps to contract. A suitable current will be obtained by increasing the current slowly until you find the sensation to be just tolerable. If at anytime, you feel uncomfortable you may stop the testing.

Oral glucose tolerance test and venous blood sample: One morning, before breakfast, you will be asked to attend an accredited blood testing laboratory where a resting 10 ml blood sample will be taken from a vein in your arm by an experienced phlebotomist. You will then be asked to undertake a glucose tolerance test which involves drinking a sugary (glucose) solution. One further blood sample will be taken exactly two hours after drinking the solution to test your blood glucose and insulin levels again. The blood samples taken will be analysed for glucose and insulin concentrations to estimate your insulin sensitivity.

Note: *The researchers are not medically trained to offer advice on treatment of illnesses that may be identified with the blood analysis. However, if abnormalities are observed in the blood results, you will be notified by phone by Mr. Warbrick and advised to see a physician. The results of the tests will be made available to you.*

Potential Risks and Discomforts

As with any research of this nature, there are some potential risks and discomforts, which you should be aware of. The researchers will attempt to minimise these through careful, consistent monitoring of your response to the testing procedures. Every effort will be made by the researchers to ensure your safety, comfort and familiarity with the testing procedures.

a. Blood test and oral glucose tolerance test: Sampling of venous blood may cause a degree of transient discomfort, and some bruising may occur at the point of insertion up to 48 hours afterwards. This discomfort is of no lasting consequence. There is also a slight risk of fainting during blood sampling. The risks and discomforts will be minimised, as the procedure will be performed under sterile conditions by highly experienced staff.

b. Muscular Contractions on the Biodex: You are likely to experience the fatigue of the thigh muscles associated with strenuous exercise during the contractions on the Biodex. Nevertheless, as in any physical activity, there is a very small possibility of injuries that include, but are not restricted to, muscle, ligament or tendon damage and dizziness. However, all protocols are commonly performed in exercise physiology laboratories and potential risks to participants have been minimized.

c. Electrically stimulated muscle contractions: Electrical Stimulation (ES) provides a small electrical stimulus to the belly of the muscle. At low frequencies (20Hz) participants may feel a ‘tingling’ in the muscle at higher frequencies (80Hz) participants may feel a ‘tugging’ in the muscle.

Right of withdrawal

Your participation in this study is completely voluntary. You may choose to withdraw from the study at any time for any reason. By signing the consent form you are indicating that the tests and procedures of this study have been explained to you and understood by you.

Inquiries

All inquiries regarding requirements and procedures used in this study are encouraged. Please contact either of the investigators below if you have any questions.

Mr Isaac Warbrick	(06) 350 5799 ext 5264	I.Warbrick@massey.ac.nz
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This project has been reviewed and approved by the Massey University Human Ethics Committee: Southern A, Application 08/56. If you have any concerns about the conduct of this research, please contact Professor John O’Neill, Chair, Massey University Human Ethics Committee: Southern A, telephone 06 350 5799 x 8771, email humanethicsoutha@massey.ac.nz