

# **A Comparison of the Anatomical and Biomechanical Parameters of the Foot in Māori and non-Māori**

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This thesis is submitted to AUT University in partial fulfilment of the requirements for the degree of the Masters of Health Science (MHSc)

**2014**

**School of Rehabilitation & Occupation Studies**

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## **Attestation of Authorship**

“I hereby declare that this submission is my own work and that, to the best of my knowledge and belief, it contains no material previously published or written by another person (except where explicitly defined in the acknowledgements), nor material which to a substantial extent has been submitted for the award of any other degree or diploma of a university or other institution of higher learning”.

Date: 22<sup>nd</sup> May, 2014

Signed

A handwritten signature in black ink, consisting of a series of loops and a horizontal line extending to the right.

## Acknowledgements

First and foremost, I would like to thank the participants for their time. My journey would not be possible without your support. Renee Muru and Dr Nathan Joseph, Kia Ora for allowing me to use the clinical rooms at Turuki Health Centre. Your passion and commitment to improve health outcomes, most notably for Māori is both motivational and inspirational. I am grateful to Morehu Kara for his wise words and invaluable guidance. Thank you for sharing your knowledge of Tikanga Māori with me. Many thanks also to Vicky Moeava for your support with recruitment and keeping it real.

To my supervisory team, Associate Professor Wayne Hing – thanks for showing me "the big picture". You sowed the seeds and set me on this pathway. Professor Keith Rome, you have shown phenomenal patience and much kindness. The little deep and meaningful and encouraging pats on the back have been amazing. Without your belief in me, this journey may not have been possible. Thank you for helping me reach this point. I read once, you may know when a storm is coming, but you have to have the skills to be able to dance through the rain - you must be a fantastic dancer.

Cheers to my whanau, thanks for having me and listening to my highs and lows. Just to let you know, it's only the start of more to come so I thank you in advance. My sister Rowena, thanks for reviewing and editing this section to a PGR rating.

Thank you Podiatry New Zealand, your sponsorship has helped in many ways, including the provision of gifts for my participants. Dr Nick Garrett I have appreciated your clever biostatistical knowledge and can-do attitude. Finally, thank you Bruce from Horizon Scanning for your assistance and expertise. Finally, Matthew Carroll, thanks for the hugs and final formatting, you're an awesome friend.

Arohanui

Bee



## **Ethical Approval**

This research was approved by the Northern X Regional Ethics Committee (Appendix A) on the 19<sup>th</sup> September, 2011 (NTX/11/09/082). Ethical approval was also approved by the AUT University Ethics Committee (AUTEC) on the 7<sup>th</sup> October, 2011 (Appendix B).

## Abstract

**Background:** Māori have poorer health outcomes compared to non-Māori and are over-represented in amputation and age-standardised mortality rates in Aotearoa. There is limited knowledge on the biomechanical parameters of Māori feet with diabetes and peripheral neuropathy.

**Aims:** The primary aim was to evaluate differences in plantar fascia thickness, peak forefoot plantar pressures and pressure time integrals under the forefoot region between Māori and non-Māori with and without diabetes and peripheral neuropathy. A secondary aim was to determine the relationship between plantar fascia thickness and plantar forefoot pressures under the forefoot in Māori with diabetes.

**Method:** A cross-sectional observational study was conducted on 36 participants recruited from two clinical sites (South Auckland and North Shore, Auckland). Participants who met the inclusion criteria were divided into four groups: Māori with diabetes, Māori with no diabetes, non-Māori with diabetes and non-Māori with no diabetes. Plantar fascia thickness was measured by ultrasound. Forefoot peak plantar pressure and pressure time integrals were evaluated. Data of the four groups were analysed using a Kruskal Wallis test and a Pearson's *r*-correlation to analyse the relationship between plantar fascia thickness and plantar pressure.

**Results:** No significant differences were found between age, gender, body mass index (BMI) or diabetes duration (for people with diabetes) across the four groups ( $p > 0.05$ ). Plantar fascia thickness showed significant differences between the groups ( $p = 0.02$ ). Post-hoc analysis demonstrated significant increases between Māori with diabetes and non-Māori with no diabetes ( $p = 0.04$ ); and non-Māori with diabetes and non-Māori with no diabetes ( $p = 0.01$ ). Peak plantar pressure demonstrated significant differences across the groups for the 2<sup>nd</sup>/3<sup>rd</sup> MPJ ( $p = 0.01$ ) and 4<sup>th</sup>/5<sup>th</sup> MPJ ( $p = 0.02$ ), but no difference for the 1<sup>st</sup> MPJ ( $p = 0.10$ ). No significant differences in pressure time integrals were found in the four groups across the forefoot region ( $p > 0.05$ ). In Māori with diabetes, a significant relationship was found between plantar fascia thickness and peak plantar pressure at the 4<sup>th</sup>/5<sup>th</sup> MPJ ( $r = 0.77$ ;  $p = 0.01$ ).

**Conclusion:** The preliminary results found biomechanical changes to the plantar fascia and increased plantar pressures in the lateral forefoot of Māori with diabetes and peripheral neuropathy. Evaluating other biomechanical risk factors may be used in future studies to evaluate predictors that may contribute to diabetic foot complications in Māori with diabetes.

## **Chapter 1: Introduction**

### **1.1 Statement of the problem**

Diabetes Mellitus (DM) is a long term chronic condition associated with considerable morbidity and mortality (Skriver, Støvring, Kristensen, Charles & Sandbæk, 2012; Wild, Roglic, Green, Sicree & King, 2004). The economic burden associated with diabetes related morbidity and mortality is underestimated due to the association of this condition with other long term conditions such as ischaemic heart disease, cerebrovascular diseases and a variety of life-threatening cancers (Coppell et al., 2013). Coppell et al. (2013) report that the prevalence of diagnosed DM in Māori is higher (7%) as well as pre-diabetes in Māori (2.2%); compared to non-Māori for diagnosed DM (4.5%) and pre-diabetes (1.5%). Globally, the prevalence of DM is estimated to rise from 2.8% from the year 2000, to 4.4% in the year 2030; which is the same prevalence predictions for Aotearoa (Wild et al, 2004; Ministry of Health, 2007).

The Ministry of Health (2012), state that the age-standardised mortality rates of DM in Aotearoa for Māori were five times higher than for non-Māori (age-standardised rate of 49.0 for Māori versus 9.8 for non-Māori). Māori are also less likely to continue with on-going DM management and follow up care (Garrett & Ihaka, 2013; Simmons & Fleming, 2000), for which the reasons may be multifactorial in nature and contribute to the higher associated morbidity and mortality rates in Māori (Browne & Garrett, 2010). Although these issues have been highlighted in terms of management and resources for this population (Browne & Garrett, 2010; Mauri Ora Associates, 2008; Scott, Marwick & Crampton, 2003), there is still a burgeoning gap between health outcomes of Māori compared to non-Māori.

DM related foot complications are a threat to all people with diabetes, although predisposing lesions (hyperkeratosis) and major lesions such as plantar foot ulcerations are more common in Māori (Ihaka, Bayley, Rome, 2012). Hyperkeratosis acts as a foreign body forming as a result of repetitive stress increasing localised pressure to the underlying tissues (Veves, Murray, Young & Boulton, 1992). Any breach to skin integrity can lead to devastating consequences such as ulceration, and it is for this reason that diabetes related foot complications is an important health issue in Aotearoa. This is of particular importance to Māori, as it has been identified that much of the associated

morbidity and mortality could be prevented if Māori received appropriate and effective care (MOH, 2013). A recent audit of a district health board in Aotearoa found that Māori were twice as likely to undergo a lower limb amputation ( $RR = 2.1$ ), compared to non-Māori ( $RR = 0.92$ ) (Garrett & Ihaka, 2013). These statistics highlight an urgent need to re-evaluate the underlying mechanisms contributing to adverse outcomes for this population. It also demonstrates that effective care does not only consider the role of the health care system to provide the service; but the skill of the practitioner to deliver effective care, as well as identify and have access to appropriate referral channels (Browne & Garrett, 2010; Garrett & Ihaka, 2013; Ihaka et al., 2012).

Up to 15% to 25% of all people with DM will experience a diabetic foot ulceration in their lifetime (Boulton, 2010; Singh, Armstrong, Lipsky, 2005; Ulbrecht, Cavanagh, Caputo, 2004); with early studies indicating that 28%-100% of this group will continue to suffer with recurrent ulcerations five years post treatment (Apelqvist, Larsson, Agardh, 1993; Chantelau & Haage, 1994; Dargis, Panteljeva, Jonushaite, Vileikyte & Boulton, 1999; Uccioli, et al., 1995). Distal symmetrical sensorimotor neuropathy (DSSN) is a consequence of either a long history of diabetes or hyperglycaemia (Boulton, 2005), and is the underlying feature of the majority of diabetic foot complications. Damage to the sensorimotor and autonomic nerves elicits structural and functional changes to the foot. Sensory dysfunction causes loss of protective sensation and can inevitably predispose the foot to repeated and undetected trauma (Mueller, Zou, Bohnert, Tuttle & Sinacore 2008). Lack of motor nerve conduction can lead to intrinsic muscle weakness altering plantarflexion of the metatarsophalangeal joints hence predisposing these areas to abnormally high pressures intensifying functional and anatomical alterations (van Schie, Carrington, Vermigli & Boulton, 2004). Autonomic dysfunction has a sudomotor effect on the skin allowing callous formation, increasing the plantar pressure under localised areas (Brem, Sheehan, Boulton, 2004). However, the coexistence of both peripheral neuropathy (sensorimotor neuropathy) and autonomic neuropathy is not necessarily habitual (Tentolouris, Pagonai, Tzonou, Katsilambros, 2001).

DSSN has been implicated as the primary cause of diabetic foot ulcerations (Edmonds, Foster, 2006; Ulbrecht et al., 2004; Watkins, 2003). Gerstein & Hayness (2001) report that DSSN may develop in 6.1% of people with DM per year, and up to 50% of older

people with DM will have evidence of DSSN (Boulton, 2010). However, foot ulcerations do not occur in isolation and are linked to other risk factors such as limited joint mobility, increased plantar pressures, plantar fascia thickening, hyperglycaemia, body mass, age, footwear and ethnicity (Boulton, 2010; Chuter & Payne, 2001; Owings et al., 2009; Solano, Prieto, Varon, Moreno & Boulton, 2008; van Schie et al., 2004; Zimny, Schatz, Pfohl, 2004); potentially leading to significant disability and a reduction in quality of life (Nabuurs-Franssen, Huijberts, Kruseman, Willems, & Schaper, 2005; Vileikyte, 2001).

The plantar fascia or plantar aponeurosis, plays an integral part in maintaining the arch during heel strike to allow for shock absorption by acting as a truss, before stabilising the arch and locking the midtarsal joints during propulsion where it acts as a beam known as the 'Windlass' mechanism (Hicks, 1954, as cited in Giacomozzi, D'Ambrogi, Uccioli & Macellari, 2005). However, tendons and ligaments are particularly susceptible to increased thickness and loss of elasticity as a consequence of metabolic changes resulting from hyperglycaemia enhancing the development of glycosylated proteins and the accumulation of advanced glycosylation end-products (Cameron, Eaton, Cotter, Tesfaye, 2001; Giacomozzi et al., 2005). Such changes within this soft tissue may lead to structural changes of the foot-ankle complex, as well as altering the overall function of the foot as a result of an altered Windlass mechanism (D'Ambrogi et al., 2005). This may invariably predispose the foot to areas of high pressure as a result of altered function.

Plantar pressure assessment has been widely used to measure the foot-to-floor interface of vertical ground reaction force in people with diabetes and DSSN to determine the level of risk of ulceration (Bennetts, Owings, Erdemir, Botek & Cavanagh, 2013; Giacomozzi & Martelli, 2006). Peak plantar pressure is the measure of choice because it is thought to be the primary indicator of skin stress leading to skin breakdown. Cut off values to determine the level of ulcer risk have been reported to be between 200 KPa to >700 KPa (Owings et al., 2009). The range in values is a result of different measurement systems in which sensor placement varies, as well as comparing participants with a history of prior ulceration, no ulceration and peripheral neuropathy. Therefore, comparisons of ranges need to be interpreted with caution. Whilst barefoot measurement of the foot-floor interface may demonstrate details of the effects of any foot deformity; in-shoe measurements are particularly useful to determine therapeutic cut off values for insole

and shoe modifications to high plantar pressure areas (Bus, Ulbrecht & Cavanagh, 2004; Bus, Maas, de Lange, Michels & Levi, 2005; Bus, Haspels & Busch-Westbroek, 2011). Although high plantar pressures will contribute to a shorter time-course for ulcerations, low-pressures over a period of time can also lead to ulceration (Wu, Driver, Wrobel, Armstrong, 2007).

Few studies have compared plantar pressure assessment to determine diabetic foot ulcer risk to different ethnic groups with diabetes and DSSN. Solano et al. (2008) found that Hispanics with diabetes and moderate DSSN showed lower peak plantar pressures as compared to their Caucasian counterparts. Although not measured, it was suggested by the authors that a difference in skin thickness and an increased range of motion at the first metatarsophalangeal joint may contribute to these findings. Whilst Gurney, Kuch, Rosenbaum, and Kersting (2012) found that Māori with diabetes and without DSSN displayed high plantar peak pressures at the central forefoot, as well as Māori with no diabetes. The authors postulated this finding may be attributable to tissue stiffness changes rather than foot function; and, it was also reported that Māori with no diabetes displayed a flatter foot. A recent study compared skin thickness changes to the epidermis in people with no diabetes, with diabetes, with diabetes and peripheral neuropathy; and, with diabetes, peripheral neuropathy and prior ulceration (Chao, Zheng, Cheing, 2011). The authors found a disproportionate increase in skin thickness in people with diabetes (6%) as compared to a reduction in the prior ulceration group (15%) and 9% reduction in epidermal thickness in people with neuropathy compared to the control. This may indicate that changes to soft tissue occur prior to the development of DSSN.

Diabetic foot ulceration often precedes lower limb amputations (Giacomozzi & Martelli, 2006). A number of risk factors have been identified in Māori which can lead to diabetic foot ulcerations such as high plantar pressures, presence of hyperkeratosis and structural deformity (Gurney et al., 2012; Ihaka et al., 2012). However, no studies have compared plantar fascia thickness and plantar pressure measurements in this population. Therefore, little is known about the specific soft tissue and function changes of Māori to determine why this population is significantly more susceptible to adverse outcomes compared to non-Māori.

## **1.2 Aims of the study**

The primary aim was to evaluate significant differences in plantar fascia thickness, pressure time integrals and peak forefoot plantar pressures between Māori and non-Māori with and with no diabetes. The secondary aim was to determine the relationship between plantar fascia thickness and plantar forefoot pressures in Māori with diabetes.

## **1.3 Hypothesis**

Null Hypothesis 1: There will be no significant difference in plantar fascia thickness between Māori and non-Māori with and with no diabetes.

Null Hypothesis 2: There will be no significant difference in forefoot peak plantar pressures between Māori and non-Māori with and with no diabetes.

Null Hypothesis 3: There will be no significant difference in forefoot pressure time integrals between Māori and non-Māori with and with no diabetes.

## **1.4 Significance of the study**

The findings from this study will inform future investigations that will explore predictors of foot ulcerations in Māori. It is intended that further exploration of these predictors will lead to an intervention study that will be aimed at reducing the impact of foot ulcerations in Māori and increase the body of evidence for ethnic groups globally.

## **Chapter 2: Review of Literature**

### **2.1 Distal symmetrical sensorimotor neuropathy**

Diabetic foot ulcerations will occur in up to 25% of people with DM (Boulton, 2010; Singh et al., 2005; Ulbrecht et al., 2004), with a reported cost of diabetic foot ulcer and infection admission in excess of NZ\$1,000 per day excluding outpatient care (Ellis, Ballance, Lunt & Lewis, 2010). Although many extrinsic and intrinsic risk factors have been reported (Boulton, 2010; Chuter & Payne, 2001; Owings et al., 2009; Solano et al., 2008; van Schie et al., 2004; Zimny et al., 2004), the common denominator is neuropathy (Edmonds & Foster, 2006; Ulbrecht et al., 2004; Watkins, 2003).

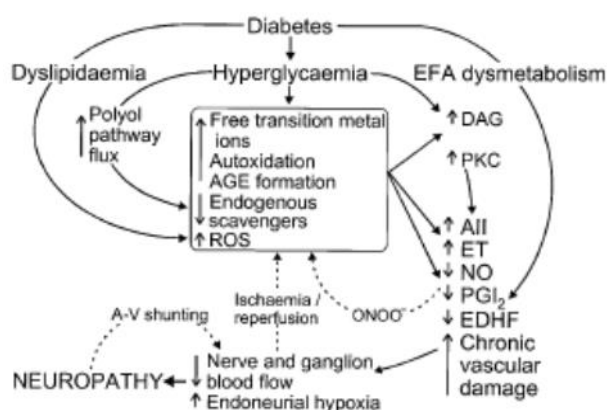
Peripheral neuropathy (PN) is comprised of many syndromes that affect the sensory, motor and autonomic nerves, occurring at various anatomical locations, with differing clinical time/course and fluctuating symptoms (Jack & Wright, 2012). However, not all people with diabetes will experience symptoms, and there is a lack of evidence to determine if symptoms will be painful or painless (Jack & Wright, 2012). The most common form of PN in diabetes is distal symmetrical sensorimotor neuropathy (DSSN) which occurs distal and symmetrical in distribution, and evidence of axonal degeneration and demyelination of the myelin sheath or epineural tissue is observed (Jack & Wright, 2012; Vinik, Strotmeier, Nakave & Patel, 2008). However, PN is commonly used to describe DSSN, and will be used throughout the current study to describe DSSN.

Although the underlying cause of PN has yet to be established, metabolic and vascular factors have been implicated in the pathogenesis, as well as other aetiologies that includes neuro-hormonal growth factor deficiencies, autoimmune, oxidative and nitrosative stress (Cameron et al., 2001; Vinik, Park, Stansberry & Pittenger, 2000; Vinik et al., 2008). Regardless of the aetiology, hyperglycaemia and lipid dysmetabolism ultimately causes a derangement in nerve function due to endoneural vasculopathy and reduced nerve perfusion (Cameron et al., 2001; Vinik et al., 2008). (Figure 2.1).

Hyperglycaemia stimulates a chain of events including activation of the polyol pathway, non-enzymatic glycation of structural proteins, oxidative stress reactions and activation of protein kinase C; rendering hyperglycaemia as an independent risk factor for PN (Cuaderes, Lamb, Khan & Lawrence, 2009; Huijberts, Schaper & Schalkwijk, 2008). Although the duration of DM has also been implicated as an independent risk factor for PN, it is important to



acknowledge that microvascular complications will still develop in people regardless of diabetes duration or adequate blood glucose levels (Boulton, 2005; Tesfaye et al., 1996).



**Figure 2.1: Metabolic and vascular factors that contribute to neuropathy.** AII, angiotensin; AGE, advanced glycation end product; A-V, arterio-venous; DAG, diacylglycerol; EDHF, endothelium-derived hyperpolarising factor; EFA, essential fatty acid; ET, endothelin-1; NO, nitric oxide; ONOO<sup>•</sup>, peroxynitrite; PGI<sub>2</sub>, prostacyclin; PKC, protein kinase C; ROS, reactive oxygen species. Reprinted with permission from Cameron et al., (2001).

The majority of people with DM will have a mixture of small and large fibre neuropathy. Large nerve fibres are usually affected first due to their length, and then sensory or motor nerves or both are affected (Vinik et al., 2000). Sensory dysfunction causes loss of protective sensation and denotes any chance of detection of changes in joint position which can inevitably predispose the foot to repeated and undetected trauma (Mueller et al., 2008). Lack of motor input can lead to intrinsic muscle weakness, reduced tendon reflexes, limited joint mobility and distal migration of the fat pad underneath the forefoot (Abouaisha, van Schie, Griffiths, Young, & Boulton 2001; Vinik et al., 2000). Small-fibre neuropathy manifests as burning pain, allodynia, and inefficient thermal sensation (Vinik et al., 2000). Autonomic neuropathy will involve any system of the body (e.g. cardiovascular, endocrine, sudomotor) and may occur sub-clinically (Vinik, Maser, Mitchell, Freeman, 2003). However specific to the foot is the loss of peripheral sympathetic tone leading increased blood flow and decreased sweating leading to fissures and callous on the feet. Therefore, damage to the sensory, motor and autonomic nerves can elicit functional changes to the foot.

Previous studies have reported that PN is an underlying feature in 90% of people with diabetic foot complications such as ulcerations (Abouaisha et al., 2001; Huijberts et al., 2008; Tesfaye, 2011). Extrinsic and intrinsic characteristics such as limited joint mobility, increased plantar

pressures, plantar fascia thickening, increased blood glucose levels, BMI, age, footwear and ethnicity have been postulated as associated risk factors for ulcerations in the diabetic population (Chuter & Payne, 2001; Zimny et al., 2004; Solano et al., 2008; Owings et al., 2009). These in combination will contribute to increased areas of plantar pressure distribution. However, in the absence of neuropathy, areas exposed to high plantar pressures will not necessarily lead to ulceration as the person is able to avoid repetitive pressure due to effective feedback mechanisms (Mueller et al., 2008).

It is the formation of advanced glycation end products (AGE's) as a result of hyperglycaemia that is thought to be the most important feature involved in diabetic foot complications. AGE's are attached to tissue proteins and three mechanisms have been identified as the drivers of these changes (Craig et al., 2008; Figueroa-Romero, Sadidi & Feldman, 2008; Huijberts et al., 2008). The first mechanism is AGE accumulation in the extracellular matrix leading to collagen cross-linking which leads to a reduction of elasticity in blood vessels (Craig et al., 2008; Figueroa-Romero et al., 2008; Huijberts et al., 2008). The second mechanism includes AGE binding to AGE-receptors on different cell types. This process leads to modulation of gene expression in vascular cells (Figueroa-Romero et al., 2008; Miyazaki, Nakayama & Horiuchi, 2002; Stern, Yan, Yan & Schmidt, 2002). The final mechanism includes non-enzymatic glycosylation of the basic fibroblast growth factor (bFGF) alongside intracellular sugars impeding endothelial function. The final result is unavoidable microangiopathy and hypoxia (Tesfaye, 2011). However, regardless of the mechanism, the outcome of reduced nerve perfusion and irreversible neuropathy remains the same (Tesfaye, 2011; Vinik et al., 2000).

AGE production will accelerate age-related changes to soft tissue including the skin, cartilage, tendons, muscles and joints (Craig et al., 2008); and earlier studies in people with type 1 diabetes (T1DM) reported cartilage related changes leading to limited joint mobility strongly associated to retinopathy and nephropathy (Rosenbloom, Malone, Yucha, & Van Cader, 1984; Silverstein et al., 1985; as cited in Craig et al., 2008). These structural changes may be eminent before the onset of peripheral neuropathy due to collagen cross-linking and abnormal storage of collagen (Giacomozzi et al., 2005). Collagen-rich tendons such as the Achilles and plantar fascia are particularly vulnerable to AGE production and impairment of these tendons will cause marked changes in gait performance (D'Ambrogi et al., 2003; Abouaisha et al., 2001).

In terms of limited joint mobility, the periarticular cartilage becomes stiffer, reducing adequate range of motion of foot joints (Duffin et al., 1999; Smith, Burnet & McNeil, 2003). The

epidermal skin has been shown to become thinner whilst the underlying soft tissues thicken in people with PN and prior ulceration compromising cushioning properties (Chao et al., 2011; Sun et al., 2011). These changes alter plantar loading patterns increasing plantar pressures in localised areas and predisposing these areas to hyperkeratosis (Formosa, Gatt & Chockalingam, 2013). Various studies have found that plantar soft tissue thickness is the strongest predictor of plantar peak pressure in the diabetic population (Sun et al., 2011; Abouaisha et al., 2001; Abouaisha, van Schie, Armstrong & Boulton, 2004).

In summary, hyperglycaemia may inevitably cause soft tissue changes in the foot due to the glycosylation of structural proteins. These changes may be subclinical, and appear before the onset of PN. Soft tissue characteristics will alter due to stiffness and thickness changes occurring in the tendons, joints muscles and skin; and when PN becomes eminent, the inability to detect trauma and altered joint sense position may alter the structure of the foot.

### **2.1.2 Anatomical and biomechanical function of the plantar fascia**

Soft tissue changes as a consequence of PN, can lead to high plantar pressures due to altered biomechanics (Formosa et al., 2013; van Schie et al., 2004). The key principles of the foot are to provide a rigid lever during propulsion to accelerate the body forward, and to become a mobile adaptor to uneven surfaces and terrain (van Schie, 2005). This is carried out by the interplay of adequate range of motion, muscle and tendon tractability as well as appropriate feedback mechanisms. Figure 2.2 illustrates the plantar fascia, which is a tendinous aponeurosis originating in the calcaneal tuberosity and extending to the proximal phalanges of the metatarsals (Duffin, Lam, Kidd, Chan & Donaghue, 2002). The plantar fascia appears as a homogeneous echogenic band on ultrasound (US), comprising of internal linear interfaces in longitudinal sections (Rathleff, Moelgaard & Olesen, 2011; Sabir, Demirlenk, Yagci, Karabulut, & Cubukcu, 2005).

The role of the plantar fascia during gait was first described by Hicks (1954) as the Windlass mechanism. The plantar fascia acts like a beam to secure the midtarsal joints to stabilise the arch during propulsion occurring at the beginning of heel lift when the person is loading on the forefoot. This action allows for greater shock absorption and inhibits the arch from collapsing when landing, by converting in action to work as a truss (Bojsen-Møller, 1978). The effects of increased thickness of the plantar fascia in response to PN have been linked to the development of a cavoid foot ultimately affecting the windlass mechanism (Giacomozzi et al., 2005).



**Figure 2.2: View of plantar fascia from a cadaveric study. Reprinted with permission from Pavan, Stecco, Darwish, Natali, De Caro (2001).**

During gait, ground reaction forces increase to more than 120-150% of a person's body weight, proportional to the area that is in contact with the ground (van Schie, 2005). These are mainly vertically directed forces, which cause tissue deformation. With changes in foot structure and loss of cushioning properties in the setting of PN, higher plantar pressures can occur due to increased duration of pressures, increased magnitude of pressure and / or increased number of pressures (van Schie, 2005). However, it has been suggested that the force applied to a localised area is more harmful in terms of tissue breakdown than force distributed over a larger area (Formosa et al., 2013). Therefore, repetitive plantar pressures can increase the risk of diabetic foot ulcerations.

## **2.2 Plantar fascia thickness and plantar pressures in the diabetic foot**

The majority of studies are concerned with either plantar pressure measurements or plantar fascia thickness measurements as risk factors for vasculopathy or for tissue breakdown leading to diabetic foot ulcerations (Duffin et al., 2002; Mueller et al., 2008; Rich & Veves, 2000; Rosenbloom et al., 1984; Silverstein et al., 1985; as cited in Craig et al., 2008; Zou, Mueller & Lott, 2007).

Plantar fascia thickness measurements have long been associated with T1DM and used as indicators for microvascular complications (Benitez-Aguirre et al., 2012; Duffin et al., 2002). However, more interest in plantar fascia thickness and its impact on plantar pressure in people with type 2 diabetes (T2DM) is slowly emerging as approaches to plantar ulceration have relied solely on PN as the root cause hindering the discovery of other causes (Giacomozzi et al., 2005). With AGE stimulating changes in collagen structures before the clinical onset of PN,

subclinical structural changes may already be inhibiting foot function, thus leading to increased areas of pressure.

In terms of imaging location, measurement of the plantar fascia using ultrasound (US) varies (Hashefi, 2011; Wearing et al., 2007). Previous studies have suggested measurements near the insertion of the plantar fascia, and other studies 1/3-5 from the insertion (Genc, Saracoglu, Nacir, Erdem, & Kacar, 2005; Ozdemir et al., 2005). There is no general consensus regarding patient positioning in terms of knee flexion, and the foot in a neutral position; however, most authors agree that the patient should be prone (Duffin et al., 2002; Ozdemir et al., 2005; Wearing et al., 2004; Wearing et al., 2007). These features complicate protocol standardization which makes comparisons between studies difficult.

Normal plantar fascia thickness with US also varies which could be a reflection on the location of the region of interest. However, previous studies suggest that in a healthy population, the width varies between 2-4mm (Hashefi, 2011; Mahowald, Legge & Grady, 2011; Wearing et al., 2007), and that any measurement above this reference or with a mean of 2mm (0.5) above mean reference values is considered pathological (Fabrikant & Park, 2011; Genc et al., 2005; Ozdemir et al., 2005; Wearing et al., 2004). An advantage of US is the relative low risk involved to the patient and the practitioners' ability to analyse in real time (Blankenbaker & De Smet, 2006). The plantar fascia can also be visualized using magnetic resonance (MRI) and computed tomography (CT) imaging, where the reference value for a non-pathological tendon is also between the 2-4mm range (Bolton, Smith, Pilgram, Mueller & Bae, 2005; Chimutengwende-Gordon, O'Donnell & Singh, 2010; Narv ez et al., 2000). However, these techniques are less popular due to cost and time required from participants.

Plantar pressure measures are dynamic indicators of vertical and horizontal stress that are associated in the development of diabetic foot ulcerations (Bus & Waaijman, 2013). Peak plantar pressures represent the peak pressure measured over an area during gait (Bus & Waaijman, 2013). Pressure time integrals signify the amount of time spent over an area which is multiplied by the amount of time required to complete the propulsive phase of gait (Bus & Waaijman, 2013; Solano et al., 2008). The pressure time integrals add value to plantar pressure measures as they signify time as a factor in the development of ulceration (Hsi, Chai & Lai, 2002).

Plantar pressures can be measured with the participant either shod or barefoot. In-shoe measurement systems are particularly useful for determining management options for healed ulcers (Bus et al., 2011). These are primarily used as outcome measures for insole and footwear prescription due to inadequate patient feedback because of sensory denervation (Bus et al., 2011). Barefoot dynamic measurements provide information regarding dynamic loading of the foot during gait. These are particularly useful measures to indicate the level of risk the foot is at prior to skin breakdown leading to ulceration. However, these measurements can only be used as indicators and cut-off values differ in the literature based on subject selection, pressure systems, and regions selected for analysis, with early studies indicating <700kPa as a cut-off point in people who have had a previous ulceration (Armstrong, Peters, Athanasiou & Lavery, 1998). Normative values cannot be detected with certainty and the degree of prediction of an ulcer, suggesting a combination of other factors contributing to skin breakdown. A limitation of barefoot measurements is the rate and loading of pressure going beyond the saturation level of the sensors (Owings et al., 2009; Armstrong et al., 1998).

Previous studies have reported the relationship between plantar fascia thickness and increased plantar pressure in the diabetic foot (D'Ambrogi et al., 2003; D'Ambrogi et al., 2005; Giacomozzi et al., 2005). The studies compared sixty-one diabetic patients without peripheral neuropathy (D), with peripheral neuropathy (DN), with prior ulceration and peripheral neuropathy (DPNU) to twenty-one controls, utilising the neuropathy disability score and bioesthesiometer to determine presence of peripheral neuropathy (Young, Boulton, MacLeod, Williams & Sonksen, 1993 as cited in D'Ambrogi et al., 2005). A 8-10 MHZ linear array transducer was used to measure the length of the plantar fascia with the participant prone, knee flexed at 90°, and ankle held in neutral position. The thickness of the plantar fascia was taken at the insertion into the calcaneus and expressed in millimetres (mm). Pathological thickness of the plantar fascia was defined as mean (SD) 2 (0.5)mm above mean reference values (2-4mm) (Fabrikant & Park, 2011; Genc et al., 2005; Ozdemir et al., 2005; Wearing et al., 2004). There was a significant difference between groups, with the most significant difference occurring in the diabetic group with neuropathy and with prior ulceration.

Plantar pressure elements obtained included centre of pressure coordinates, pressure distribution, contact area, and pressure / time and force / time integrals (D'Ambrogi et al., 2003; D'Ambrogi et al., 2005; Giacomozzi et al., 2005). The foot-floor interface was measured utilizing a piezo-dynamometric platform taking the mean of six footsteps with the participants

unshod. The areas of interest included the hallux, metatarsals and heel. The location of these sites was determined geometrically by the participants' footprint and analysed by four different operators. The intra-operator differences were not statistically significant. The authors concluded that plantar fascia thickness was thicker in DM participants compared to controls leading to increased loading times and force integrals under the metatarsals in the same groups. The overall findings suggested an altered Windlass mechanism due to changes affecting soft tissues altered function during gait, particularly during propulsion (D'Ambrogi et al., 2003; D'Ambrogi et al., 2005; Giacomozzi et al., 2005).

In summary, the imaging findings were not compared to a control population with neuropathy and with no diabetes. A systematic review suggested that US has low sensitivity and low accuracy of plantar fascia thickness measurements and that magnetic resonance imaging (MRI) was reproducible (de Oliveira, Lemos, de Castro Silveira, da Silva & de Moraes, 2011). However, the practicality, cost and invasive nature of this type of imaging needs to be considered.

## **2.3 Methodological issues in measuring plantar fascia thickness and plantar pressures**

Given the limited resources available in the literature, a review of plantar fascia thickness and plantar pressure methodology was undertaken.

### **2.3.1 Search Strategy**

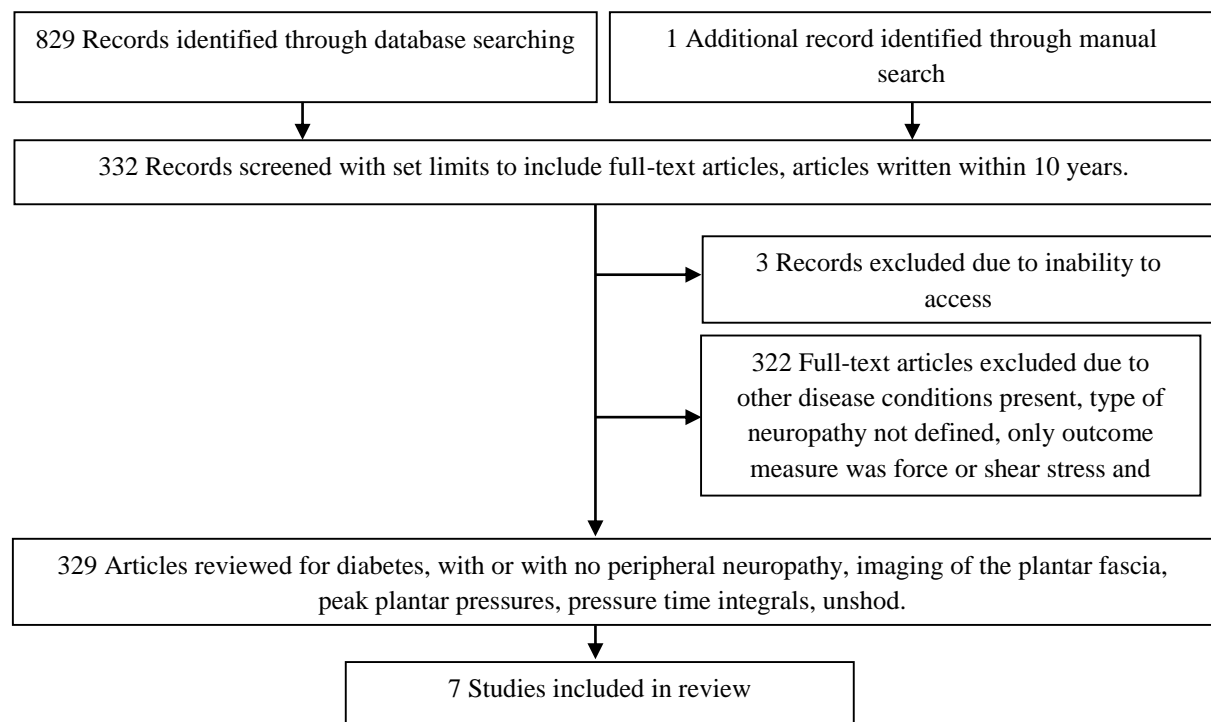
A search strategy to identify appropriate studies was performed electronically using the following databases: Cochrane Database of Systematic Reviews (2000-2013); Cumulative Index to Nursing and Allied Health Literature (CINAHL, 1976-2013); EBSCO Health Databases (1979-2013); Ovid Medline (1946); SCOPUS (from 1946-2013); which AUT University Library maintains subscription. The search was conducted between November 2012 and July 2013. The reference lists of the studies were then reviewed manually to detect further studies that met the inclusion criteria.

*Inclusion Criteria:* The criteria for selecting studies relating to the structure and function of the foot include those studies including people with diabetes with or with no peripheral neuropathy and those investigating imaging of the plantar fascia as well as plantar pressures using unshod

pressure measurement systems. Studies that reported patients with type 1 diabetes were included if they also reported patients with type 2 diabetes.

*Exclusion Criteria:* Studies were excluded if the participants suffered from any other form of chronic disease (inflammatory arthritis or neuromuscular conditions) or the type of neuropathy was not defined. Studies were also excluded if the outcome measure of plantar pressure included only force or shear stress. Studies with interventions were also excluded.

Limitations were set to include only full-text articles, articles written in English, articles published in peer-reviewed journals, and articles written within the last 10 years (Figure 2.3). A total of 7 articles were included in this review after meeting the inclusion criteria.



**Figure 2.3:** Flow chart of literature search for plantar pressure and imaging modalities.



**Table 2.1: Summary of papers reviewed.**

Author	Subjects	Age (SD) (years)	Methodology	Findings	Conclusion
Bolton et al. (2005).	DM: 16 with PN (4F, 12M); ND: 10 (2F, 10 8M)	DM: 55.2 (10.5) ND: 53.8 (9.1)	3D CAT: plantar fascia thickness.	Mean (SD) plantar fascia thickness: DM: 4.2 (0.9)mm; ND: 3.6 (0.8)mm.	Plantar fascia thickness significant between groups ( $P = 0.04$ ).
Udoh et al. (2010).	DM: 200 (89F, 111M) ND: 200 (95F, 105M)	DM: 60.7 (6.6) ND: 39.5 (3.5)	MUS: plantar fascia thickness.	Mean (SD) overall plantar fascia thickness: DM: 4.4 (0.94) mm; compared to ND: 3.0 (0.26) mm in females, ND: 3.3 (0.11) mm in males.	Plantar fascia thickness significant ( $P < 0.05$ ) in DM group.
Bus & de Lange (2005).	DM: 14 with PN (8 F, 6 M)	58.6 (6.1)	Plantar Pressure: Peak plantar pressures and pressure time integrals, EMED-NT pressure platform.	No difference between peak plantar pressures and pressure time integrals using different step protocol (1-step, 2-step, 3-step).	All barefoot pressures can be reproduced using any of the step protocols, although the 2-step protocol requires the least amount of repeated trials.
Mueller et al. (2008).	DM: 12 with PN and ulceration (6 F, 6 M); ND: 12 (7F, 5M)	54 (8)	Plantar Pressure: Peak plantar pressures, one foot analysed using EMED ST P-2 platform. Three trials using 2-step protocol.	Peak plantar pressure in the forefoot showed a 34% difference between groups. Mean (SD) total forefoot: DM: 998 (215) KPa ND: 739 (268) KPa	PN prevents detection of high pressure and pain to avoid undetected trauma.
Owings et al. (2009).	DM: 49 with prior history of ulceration (11F, 38M)	62.9 (10.3)	Plantar Pressure: Peak plantar pressures unshod, both feet analysed using EMED pressure platform. 1-step method employed.	Mean peak plantar pressure in the forefoot 566 (316) KPa with a range 107 – 1192 KPa. Some peak plantar pressures exceeded the saturation level of the pressure system.	Mean peak plantar pressures at prior ulceration sites lower than previous studies reported.
Solano et al. (2008).	DM: 35 with PN / Caucasian (22F, 13M); DM: 44 with PN / Hispanic (23F, 21M); ND: 33 Caucasian (11F, 22M); ND: 41 Hispanic (20F, 21M)	DM: Caucasian 60.8 (11.7) DM: Hispanic 64.2 (11.3) ND: Caucasian 56.8 (11.7) ND: Hispanic 59.5 (9.3)	Plantar Pressure: Peak plantar pressures and pressure time integrals, unshod, both feet analysed using EMED-SF-4 pressure platform. 2-step method employed.	Peak plantar pressures were lower between DM groups: DM: Hispanic KPa 464.1 (222.6) DM: Caucasian KPa 699.6 (323.1). This was the same for pressure time integrals: DM: Hispanic KPa/s 463.2 (39.3) DM: Caucasian KPa/s. No significant differences between ND groups.	Dynamic plantar pressures may be different when comparing ethnic groups with DM and PN.
Yavuz et al. (2008).	DM: 15 with PN (3F, 12M); ND: 20 (8F, 12M).	DM: 60.5 (10.1) ND: 45.8 (19.8)	Plantar Pressure: Peak plantar pressures and pressure time integrals using a custom built platform. 2-step method employed.	No significant difference found between groups for mean peak pressure although DM group had a higher mean value (23%). Mean pressure time integral: DM: 54% higher with $P = 0.013$ .	Higher pressure time integrals may be associated with longer contact time in DM with PN.

DM = diabetes mellitus; ND = no diabetes; PN = peripheral neuropathy.

### **2.3.2 Imaging measurements in diabetes**

Only two studies met the inclusion criteria for analysis (Table 2.1). Bolton et al. (2005) utilized computed tomography to quantify the thickness of the plantar fascia in controls (n=10), people with DM and PN with a past history of ulceration (n=16). Participants were then matched for age and weight. Measurements were taken 1/5 of the distance from the plantar fascia insertion to the first metatarsophalangeal joint. The foot was placed in a neutral position, and one foot was analysed. A Siemens Plus4 CT scanner (Siemens Medical Systems Inc., PA, USA) was utilized to capture CT images, set at 120 kVp, 220 mAs, 3 mm collimation, and 3 mm table increment per rotation on a 512 x 512 matrix. A soft tissue algorithm was used to analyse the data set at 1mm increments taken from the sagittal slice with the thickest aponeurosis at the location of interest. Measurement reliability of these measures were good with bias in the linear measurements (-0.31 to 0.53 mm) and variability (0.53 to 2.62 mm). Mean plantar fascia thickness measurements were found to be thicker in people with DM (4.2 mm), compared to controls (3.6 mm). The results for the DM group with prior ulceration indicate variance between groups. Although prior studies comparing CT to US have demonstrated lower mean plantar fascia thickness measures of cases and controls; this may be due to the preciseness of the CT processing which involves the generation of a three-dimensional image from a series of two-dimensional X-ray images.

Udoh, Ezeokpo, Agwu and Okpala (2010), investigated the comparison of plantar fascia thickness using US before and after administering hypoglycaemic medication in a Nigerian population with DM with no prior ulceration (n=200), and comparing the findings to a healthy population (n=200). People with prior ulcerations were excluded from the study, and there was no match between age and weight. A digital grey scale SonoAce 5500 (Medicol, Korea) and 10MHz transducer were used to obtain thickness measurements which were taken 3 cm from the insertion of the plantar fascia with the patient prone and knee flexed at baseline; and then after 3 weeks of prescription of anti-glycaemic agents. The mean (SD) plantar fascia thickness in the control group was 3.13 (0.15) mm compared to 4.20 (1.31) mm in the DM population. The results indicated higher than normal reference values of the plantar fascia width for the DM group, with the plantar fascia thickness in the DM group ranging between 3.13mm to 7.6mm. The authors concluded that other factors such as hormonal constitution between male and female subjects may have contributed to the

findings. However, there are several limitations which may account for these findings. Firstly, the control group had a mean HbA1c of 6.5%. This is a feature of pre-diabetes, a state of impaired glucose tolerance where underlying vascular changes could be taking place prior to the diagnosis of DM. Secondly, there was a significant difference in age and BMI, that is, the DM population were older (DM; 60.7 years versus control 39.5 years), and heavier (DM 26.7 Kg/m<sup>2</sup> versus control 22.6 Kg/m<sup>2</sup>). Furthermore, subjects were not screened for PN to show differences between early onset plantar fascia thickening; and the method for patient position did not suggest if the plantar fascia was put under load for adequate investigation. Also, it is not reported how many measurements were obtained to calculate the means or if the findings reported in Table 1 and 2 are the baseline measurement or post-medication (also not reported) measurements. Therefore, this study is not robust enough to draw definitive conclusions.

In summary, the two studies demonstrate a lack of comparison for the standardized assessment protocols and imaging technique employed. However, Bolton et al. (2005) demonstrated reliability and statistical differences between control groups and those with DM. A limitation of some of the studies is the combination of T1DM and T2DM patients. Changes in plantar fascia thickness can occur at a younger age in people with T1DM which may have some bearing on these results. However, the overall findings supports suggestions that plantar fascia thickening occurs in people with DM and is vital to the understanding of factors leading to the development of skin breakdown and ulceration.

### **2.3.3 Plantar pressure measurements in diabetes**

Five studies were included in this review (Table 2.1). Mueller et al. (2008) reported on the differences in stress variables between the forefoot and rearfoot in participants with DM and PN; and current plantar ulceration. Twelve people with DM, PN and a plantar ulcer were employed in one group, with evidence of PN indicated by absence of sensation to 5.07 monofilament. The control group consisted of twelve people with no history of DM or PN. People were excluded from the study if they did not have an active ulceration; had severe midfoot deformity and / or a partial amputation. One foot was utilised in this study, with the control group using the same foot used by the DM group as the comparison. A two-step method of three walking trials was used for data collection and analysis. The results

demonstrated mean peak plantar pressures expressed as a total in the forefoot of 988 KPa in the DM group compared to 739 KPa in the control group.

Owings et al. (2009) recruited 49 subjects with T2DM and prior ulceration (38 men and 11 women, matched for age, height and weight). Patients were included in the study if the ulcers were of neuropathic origin and the ulcerative site had remained healed for 90-days. Exclusion criteria included major vascular disease, reduced toe pressure ( $<35\text{mmHg}$ ), trauma as cause of ulceration, Charcot fracture, prior foot surgery involving amputation and inability to walk. Five first-step trials were collected and expressed as a mean at three sites (hallux; first metatarsal head; second-fifth metatarsal and total mean of the forefoot). The main aim of this study was to measure in-shoe plantar pressures and other characteristics in a population with ulcerations which had remained healed. The authors found barefoot mean peak plantar pressures of 566 KPa in the forefoot, with a range between 107-1192 KPa reaching above the saturation level.

These mean peak plantar pressure values for the forefoot where healed ulcerations have occurred are lower than those reported for current ulcerations by Mueller et al. (2008). Owings et al. (2009) suggested that  $>700\text{ KPa}$  be used as a reference to indicate an at-risk of ulceration cut-off point. However a limitation of this study is the inability to compare findings to a control group.

Yavuz, Tajaddini, Botek and Davis (2008) aimed to determine the differences in pressure time integrals and peak plantar pressure of the forefoot in DM versus controls using a custom built plantar pressure system. The authors recruited fifteen people without DM and twenty people with DM and PN as determined by 5.07monofilament and a bioesthesiometer. A two-step method was employed and only one foot was used for analysis (not matched). A significant difference for mean (SD) pressure time integrals between the control 167 (54) KPa/s and DM group 258 (142) KPa/s. The study showed no significant difference in peak plantar pressures between the two groups (control 498 versus DM 614 KPa). To note from this study is the recruitment of a DM population without a current or previous ulcer; however, the mean peak plantar pressures reported are higher than those that were reported by Owings et al. (2009) in a population who had suffered a prior ulceration. This indicates that plantar pressure platforms vary in terms of their sensor placement indicating that results between plantar pressure systems should be analysed thoughtfully.

Bus and de Lange (2005) compared the difference between one, two and three-step methods in fourteen DM participants with PN, which was determined by a bioesthesiometer and a 5.07gm monofilament. Ten repeated trials per step-method were employed. Each step protocol was randomised. The foot was divided into six anatomical regions for statistical analysis of pressure time integrals and peak plantar pressure. The results demonstrated no significant difference between the three different step protocols for pressure time integrals and peak plantar pressure. Intraclass correlation coefficients (ICC) were calculated to demonstrate reliability in each protocol and showed acceptable reliability ( $ICC > 0.85$ ) in 1-step protocol if four collected trials for peak plantar pressure and seven trials for pressure time integrals were undertaken; and 3 to 4 trials respectively of the 2-step method, and 4 to 5 trials if utilising the 3-step method.

In terms of step method, Owings et al. (2009) showed good reliability by utilising five trials using the 1-step method, whereas Meuller et al. (2008) and Yavuz et al. (2008) only employed three trials using the 2-step method. This method was also employed by Solano et al. (2008). The aim of the study was to detect significant differences in ethnic populations with and with no DM and PN (Hispanic and Caucasian with DM & PN; versus Hispanic and Caucasian with no DM). Participants were included based on the neuropathy disability score. The results demonstrated higher peak plantar pressures in the entire foot and all regions in the Caucasian group (810 KPa) compared to the Hispanic group with DM (228 KPa). Similar findings were found for pressure time integrals. In the forefoot, the Hispanic group with DM showed significant reduced peak plantar pressure after adjusting for age, weight, gender and DM duration compared to the Caucasian group (464 KPa versus 700 KPa), suggesting that moderate PN may not necessarily elevate plantar pressures in this ethnic group.

The majority of studies included in the review utilised the EMED plantar pressure system, with the same methods for diagnosis of PN and similar selection for step protocol. However, the subject selection differed (current ulceration, prior ulceration, no ulceration), as well as the use of a customised plantar pressure platform. These features invariably impede comparisons between studies. A limitation of these studies was that none utilised people with DM in the absence of PN, which may have had some impact on gait style. This may have also given different pressure time integrals comparisons as well, with the expectation

that pressure time integrals would be higher in the group with neuropathy due to loss of tactile sensitivity increasing contact time and delaying propulsion as demonstrated in the study by Yavuz et al (2008). These results could be used as a cut off value in the prediction of skin breakdown leading to ulcerations.

## **2.4 Summary**

Changes in thickness of the plantar fascia have been reported in the literature. Although imaging choice and standardization of assessment varies, there is a consensus that plantar fascia thickness occurs in people with T2DM, which is observed before the clinical onset of PN. There is also limited evidence that plantar fascia thickness may be different across ethnic groups with only two studies reporting differences. One study lacked vital information regarding the characteristics of the study participants. The other study comparing peak plantar pressures and pressure time integrals reported ethnic differences in plantar pressure distribution, and suggested that PN may not necessarily lead to increased plantar pressures. The two studies suggested a structural and functional difference across ethnic groups, however, the methodological quality of these studies were not robust to come to a definite conclusion.

The ultrasound plantar fascia thickness studies identified that the changes to the soft tissues occur at the insertion of the plantar fascia. This feature may have functional consequences in terms of altering the Windlass mechanism during gait. In the presence of PN, the thickness increases, as does dynamic plantar pressures in people with DM compared to control groups. High dynamic plantar pressures have been demonstrated in the literature as pre-cursors for skin breakdown and ulceration. Higher pressure time integrals and peak plantar pressures are observed for those who have suffered prior diabetic foot ulceration, but there are no absolute cut off values to determine when skin breakdown will occur. Therefore, comparing plantar fascia thickness, pressure time integrals and peak plantar pressures in controls with and with no diabetes and peripheral neuropathy, to a different ethnic population may help identify the missing gaps in the literature.

## **Chapter 3: Methodology**

### **3.1 Study Design**

Cross-sectional observational study design to examine plantar fascia thickening and plantar pressure of the forefoot of Māori and non-Māori with and with no diabetes.

### **3.2 Participants**

Participants were recruited from two different locations, Turuki Health Centre and the AUT University Podiatry Clinic, Auckland. Māori self-determined their ethnicity status when consenting to the study. Protection of Māori tikanga and culture was preserved through participant anonymity. The project was specifically designed for Māori participation. Consultation with a Māori representative from Turuki Health Centre occurred at the inception of the idea of the study and prior to the recruitment process to allow for the provision of acknowledgement of the key principles of the Treaty of Waitangi as including principles of partnership, participation and protection, and the related ethical responsibilities of the researchers. Participants were recruited directly at both sites and through an advertising poster placed in the waiting areas of both locations. Participants were provided with information forms with the researchers contact details (Appendix C). This allowed the participants to ask the researcher questions directly so to make an informed choice, in line with the right of Māori for self-determination (Appendix D). Ethical approval was granted by the Northern X Regional Ethics Committee on the 19<sup>th</sup> September, 2011 (Appendix A).

*Inclusion Criteria:* Māori and non-Māori aged  $\geq 18 - 65$  years with and with no diabetes. Furthermore, only people with peripheral neuropathy as determined by monofilament and bioesthesiometry were included.

*Exclusion Criteria:* Participants were excluded from the study if they were aged  $\geq 65$  years; current history of neurological conditions (other than diabetes) affecting balance and coordination; current diabetic ulcer or a lower limb amputation.

#### **3.2.1 Clinical characteristics**

Information regarding participant clinical demographics was recorded such as age (years), height (m), weight (Kg) and BMI ( $\text{Kg/m}^2$ ). Other clinical information such as self-reported

blood glucose level averages (mmol/L), current diabetes medication, and diabetes duration (years) were also recorded.

### **3.2.2 Foot specific characteristics**

The participant's foot type using the Foot Posture Index (FPI) was recorded using a validated index which is an observational measure of the participant during relaxed standing position where a deviation from a score of zero indicates either supinated characteristics (high arch) or pronated characteristics (flat foot) (Redmond, Crosbie & Ouvrier, 2006; Keenan, Redmond, Horton, Conaghan & Tennant, 2007). Foot width, length and length of arch measurements were also recorded using a specialised shoe fitting device (The Brannock Device Co, NY, USA).

Type of footwear information was recorded based on a validated footwear assessment form which has been used to determine footwear styles in long term conditions (Barton, Bonanno, Menz, 2009; Silvester, Williams, Dalbeth & Rome, 2010; Rome, Frecklington, McNair, Gow & Dalbeth, 2011). A foot examination was conducted by the researcher (BI) to determine areas of callous that required debridement. The callous was removed prior to pressure measurements as these areas have been reported to cause altered walking patterns (Abouaesha et al., 2001).

### **3.2.3 Screening for peripheral neuropathy**

Participants with and with no diabetes underwent screening tests to determine eligibility for the study. Loss of sensation was determined by using a 5.07 Semmes-Weinstein monofilament (Bailey Instruments Ltd, UK) and a Bioesthesiometer (Bio-medical Instrument Company, Newbury, Ohio) to measure vibrational pain threshold (VPT). Participants were included in the study if they were unable to detect the monofilament at one or more sites of the plantar surface of either foot (hallux, first metatarsal, third metatarsal, fifth metatarsal); or demonstrated a loss of vibration perception threshold (VPT  $\geq 25V$ ) using the Bioesthesiometer at the apex of the hallux (Young, Breddy, Veves & Boulton, 1994; Smieja et al., 1999).

## **3.3 Instrumentation**

### **3.3.1 Plantar fascia thickening**



A portable ultrasound machine model 8300 (Chison, Wuxi, China) with a 256 gray scale imager and a 7.5 MHz linear array transducer was used to measure plantar fascia thickening. Real-time plantar fascia thickness was captured in B-mode (Figure 3.1). The system monitor comprised of 10-inch super video graphics array (SVGA) of high resolution.

#### **3.3.1.1 Intra-tester reliability of measuring plantar fascia thickening**

Prior to data collection, an intra-tester reliability (BI) test was conducted of five healthy subjects. The right foot was measured on two separate occasions on the same day, one hour apart. The results demonstrated an intra-class correlation coefficient ( $\alpha = 0.84$ ) with 95% CIs [-0.67, 0.86] indicating very good intra-tester reliability (ICC >0.75 indicates good-to-excellent intra-tester reliability) similar to a previous study evaluating intra-tester reliability of the plantar fascia (Rathleff et al., 2011).

#### **3.3.2 Plantar pressure measurements**

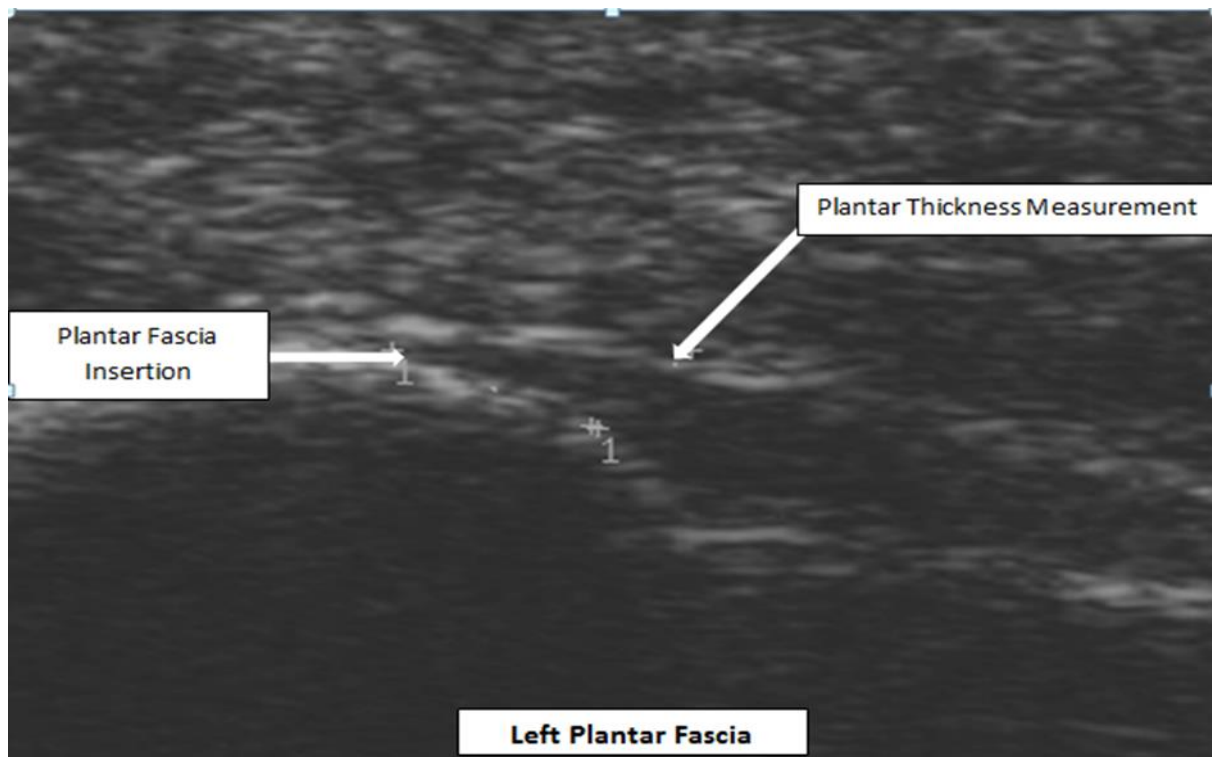
Plantar pressure measurements were captured by recording unshod plantar pressure measurements of both feet using the MatScan® version 6.61 (Tekscan, Boston, USA). The MatScan® is a portable low-profile floor mat of 5mm thickness (sensing area 435.9 x 368.8 mm) comprising of 2,288 sensors (1.4 sensors/cm<sup>2</sup>) capturing dynamic events at scan rate of 100 Hertz. The TekScan software maps the plantar pressures detected by the hardware into the pressure data displayed in a real-time window. The intra- and inter- session reliability of the MatScan® system has been shown to be reliable using this instrumentation in healthy subjects (Zammit, Menz, Munteanu, 2010). Prior to the commencement of the study, the researcher was trained to use MatScan® instrumentation and data analysis software.

### **3.4 Procedure**

#### **3.4.1 Plantar fascia thickening**

Plantar fascia thickness was measured non-weight-bearing with the participant lying prone on a plinth. Dorsiflexory force was applied to allow for maximal tension to the plantar fascia as well as definition of the tendon (Cardinal, Chhe, Beauregard, Aubin & Petlleteir, 1996). Transmission gel was then applied over the medial tubercle of the calcaneus (Fabrikant & Park, 2011). A longitudinal measurement was taken from the proximal end of the plantar fascia where it crosses the anterior-inferior border of the calcaneus to 5mm from its insertion

(Cardinal et al, 1996; Gibbon & Long, 1999). The width (mm) of the plantar fascia was measured three times and an average was accurately measured, using the built in callipers of the US machine. The plantar fascia was measured in the sagittal plane (Figure 3.1). The normal appearance of the plantar fascia is homogenous and echogenic with an average thickness of 2-4mm (Wearing et al, 2004; Genc et al., 2005; Ozdemir et al, 2005; Fabrikant & Park, 2011; Hashefi, 2011); as opposed to hypoechoic and diffuse in the presence of pathology (Rathleff et al, 2011). Mean plantar fascia width thickness for participants with diabetes has been reported to be 3.0 mm (Craig et al, 2008; Giacomozzi et al., 2005; D'Ambrogi et al., 2005). Three repetitions of each foot were captured by the researcher who is experienced with musculoskeletal ultrasound. The US images were converted to digital frames 800 x 600 pixels (Bitmaps).



*Figure 3.1: Longitudinal view of plantar fascia measurement from the anterior-inferior insertion.*

### **3.4.2 Plantar pressure measurements**

Three walking trials were performed in order for the participants to familiarise walking on the mat. A marker set at each end as a starting reference point, determined by each individuals stride length. A walk-calibration test was performed according to the

manufacturer's instructions. A 3-step protocol was used due its type-reliability in testing barefoot pressure measurements in people with peripheral neuropathy (Bus & de Lange, 2005; Caselli, Pham, Giurini, Armstrong & Veves, 2002). The first and last strides were excluded; therefore, a total of seven strides were averaged from each walking trial (Rome et al, 2011).

The TekScan Research Pressure Measurement System version 6.61 was used to analyse the data. The regions of interest (ROI) include the 1<sup>st</sup> metatarsophalangeal joint (1<sup>st</sup> MPJ), 2<sup>nd</sup> metatarsophalangeal joint and 3<sup>rd</sup> metatarsophalangeal joint (2/3<sup>rd</sup> MPJ), 4<sup>th</sup> and 5<sup>th</sup> metatarsophalangeal joints (4/5<sup>th</sup> MPJ). The outcome measures included peak plantar pressure (KPa) and pressure time integrals (KPa/s). A walk calibration was performed for both feet of each participant according to the participant's weight (kg). The mat was set to trigger a recording each frame of the stance phase of gait as soon as the participant walked on the pressure system.

### **3.5 Data Analysis**

Statistical Package for the Social Sciences (SPSS) Version 20.0 was used for all data analysis. Descriptive statistics (mean, SD) were used to report age, BMI, diabetes duration, blood glucose level and foot posture index. Plantar pressure measurements and plantar fascia thickness measurements were also recorded as mean (SD). Measurements for the right and left foot were averaged to represent an overall finding. Footwear information was reported as a percentage (%). Sample size calculations were not performed but determined by considering a minimum between-group difference and specific comparisons between groups. The Kruskal Wallis test was applied due to small sample sizes in each group; uneven sample sizes of each group and because this test does not assume the normal distribution of data (Guo, Zhong, Zhang, 2013). This is a non-parametric test, and its parametric equivalence is the Analysis of Variance (ANOVA) which would assume the normal distribution of data.

Least significant difference was used as a post hoc test to determine the mean scores between groups. To analyse the relationship between plantar pressure and plantar fascia thickness a Pearson's *r*-correlation was undertaken. All data was tested at  $P = 0.05$  level.

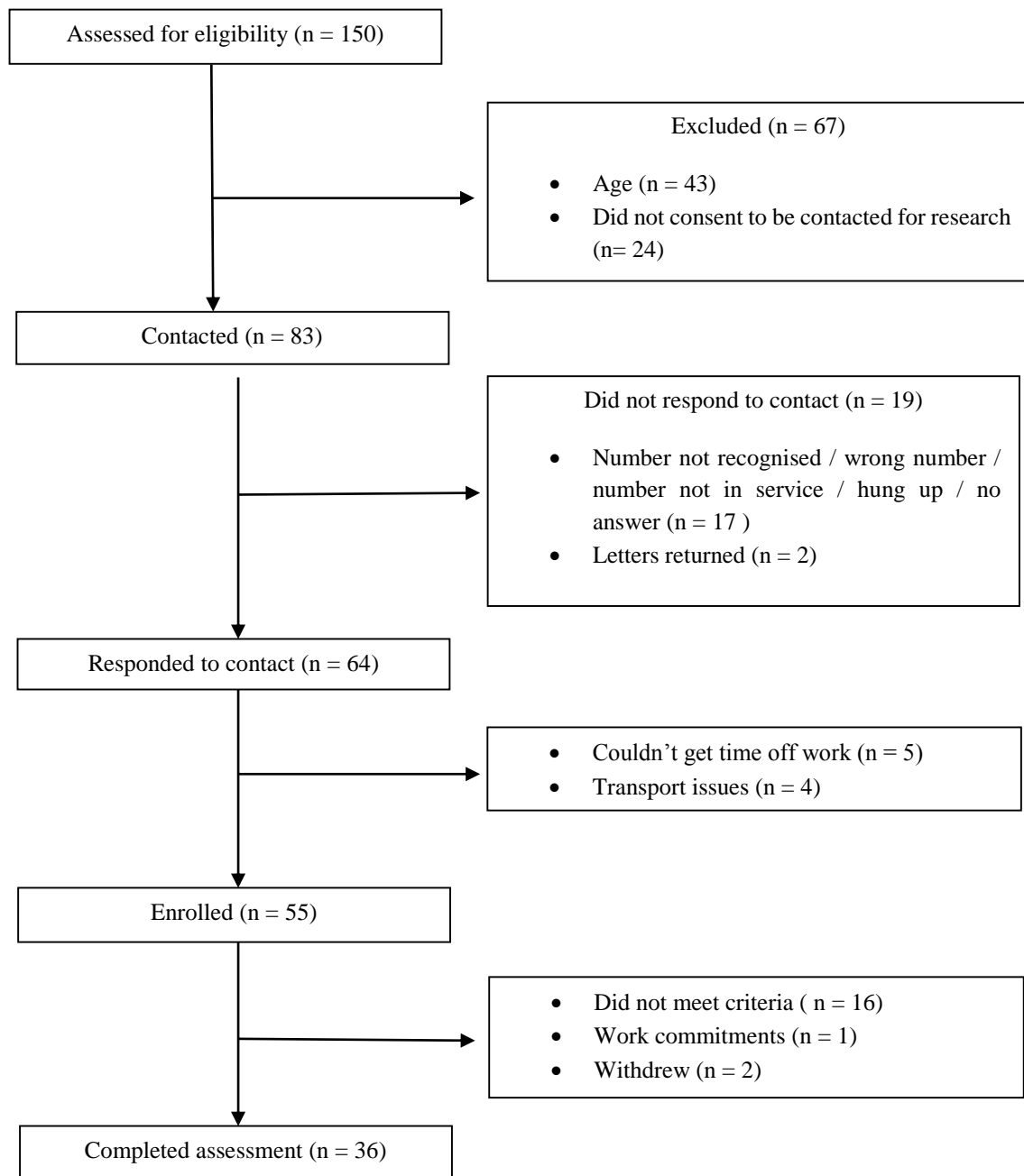
## Chapter 4: Results

### 4.1 Demographic Characteristics

Thirty six (n=36) participants were recruited. Figure 4.1 illustrates a flowchart of participant's recruitment into the study. Twenty one (n = 21, 58%) were male and fifteen (n = 15, 42%) were female. Ten (10) participants who identified as being Māori with DM; seven (n = 7) participants who identified as being non-Māori with DM; ten (n = 10) participants who identified as being Māori without DM and nine (n = 9) participants who identified as being non-Māori without DM. A total of 56% of the cohort identified as being Māori as compared to 44% who identified as being non-Māori. The mean (SD) age was 58 (7.9) years. The mean (SD) for BMI of the participants was 34.2 (10.9) Kg/m<sup>2</sup>, with the diabetes participants having a higher BMI overall (Table 4.1). A total of seventeen participants (47%) had a history of diabetes. Diabetes duration for the non-Māori with DM was longer compared to Māori with DM, but not significant ( $P = 0.17$ ). Both DM groups demonstrated mean blood glucose levels above acceptable range ( $>7\text{mmol/L}$ ). The Foot Posture Index scores demonstrated no significant differences in foot type between the four groups ( $P = 0.12$ ). Foot width and length characteristics are demonstrated in Appendix E.

The mean (SD) duration of DM for the cohort was 13 (10.8) years, ranging between 2-37 years. The majority of participants who identified as having DM were taking hypoglycaemic agents (oral hypoglycaemic agents (n = 8, 47%); insulin (n = 3; 18%); oral hypoglycaemic agents and insulin (n = 3; 18%); diet alone (n = 3; 18%).

In this cohort, we found 33% (n = 12) attended with walking shoes; 17% (n = 6) attended with sandals; 11% (n = 4) attended with athletic shoes; 8% (n = 3) attended with boots; 6% (n = 2) attended barefoot or with flip flops or moccasins; and 14% (n = 5) attended with oxford, ugg boot, slipper, backless slipper or court shoe styled footwear.



*Figure 4.1: Recruitment flow chart.*

**Table 4.1: Demographic Characteristics.**

Group					
Variable	Non-Māori no DM Mean (SD)	Māori no DM Mean (SD)	Non-Māori with DM Mean (SD)	Māori with DM Mean (SD)	<i>P</i> -value
Age (years)	62.3 (2.5)	57 (7.4)	58.9 (5.8)	54.4 (11.4)	0.17
BMI (kg/cm <sup>2</sup> )	29.4 (7.6)	32.9 (12.3)	37.5 (8.9)	37.6 (12.6)	0.32
Diabetes Duration (years)	NA	NA	17 (12.9)	10.4 (8.8)	0.22
Blood Glucose Level (mmol/L)	NA	NA	9 (.0)	7.5 (2.9)	0.52
Foot Posture Index	4 (4.2)	7 (2.4)	8 (2.3)	7 (3.6)	0.12

## 4.2 Plantar fascia thickness

Table 4.2 demonstrates the mean (SD) thickness differences between each group. The results demonstrated a significant difference between the four groups ( $P = 0.02$ ).

**Table 4.2: Plantar fascia thickness measurements (mm).**

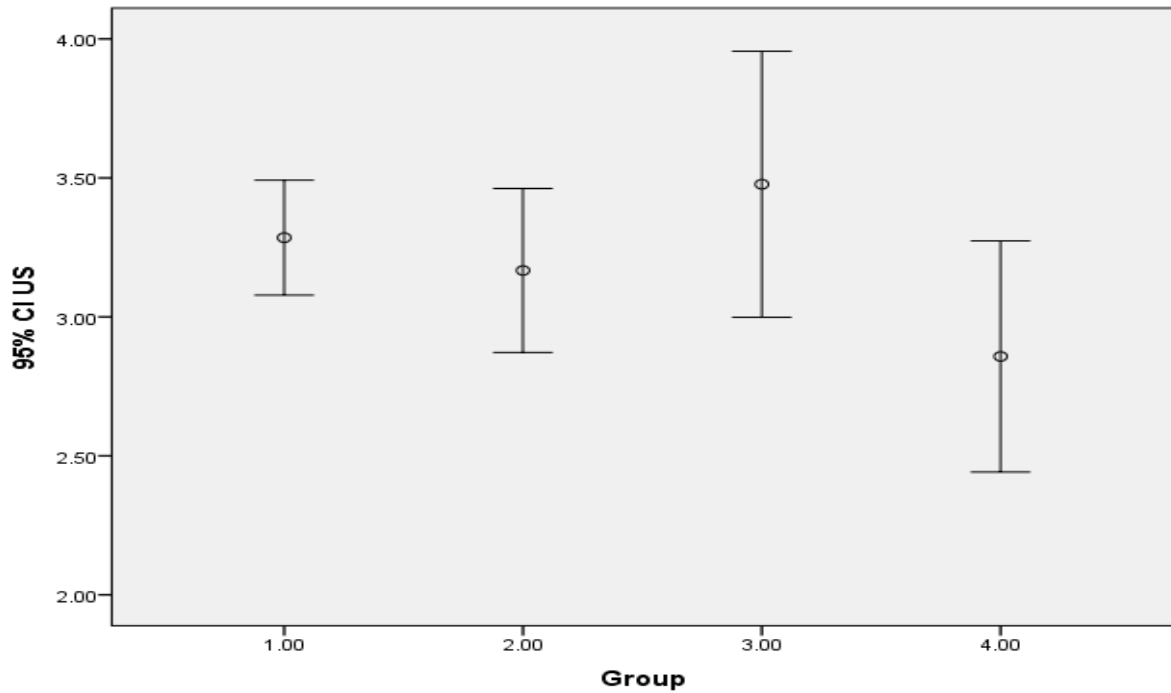
Group					
Variable	Non-Māori no DM Mean (SD)	Māori no DM Mean (SD)	Non-Māori with DM Mean (SD)	Māori with DM Mean (SD)	<i>P</i> -value
Plantar fascia thickness (mm)	2.85 (0.5)	3.16 (0.4)	3.47 (0.5)	3.28 (0.2)	0.02

A post-hoc test was performed to determine the differences across the four groups (Table 4.3). Significant differences were found between Māori with DM and non-Māori with no DM ( $P = 0.04$ ); and non-Māori with DM and non-Māori with no DM ( $P = 0.01$ ). Figure 4.2 illustrates the 95% confidence intervals of ultrasound results. We therefore reject null hypothesis 1 and state there is a significant difference in plantar fascia thickness between Māori with diabetes and non-Māori with no diabetes.

**Table 4.3: Post Hoc results of plantar fascia thickness between groups.**

Variable	Group	Mean Difference (mm)	Overall $P$ -value
Plantar fascia thickness (mm)	Māori with DM vs. Non-Māori with no DM	0.42	0.04*
	Non-Māori with DM vs. Non-Māori with no DM	0.61	0.01*
	Māori with no DM vs. Non-Māori with DM	0.31	0.20
	Non-Māori with DM vs. Māori with DM	0.19	0.40
	Māori with DM vs. Māori with no DM	0.11	0.60

\*mean difference is significant at the 0.05 level



**Figure 4.2: 95% Confidence Intervals for Plantar Fascia thickness (mm). 1 = Māori with DM, 2 = Māori without DM, 3 = Non-Māori with DM, 4 = Non-Māori without DM.**

## 4.3 Plantar pressure

### 4.3.1 Peak plantar pressure

Table 4.4 demonstrates the mean (SD) findings of the metatarsophalangeal joints (MPJ) for peak plantar pressure measurements. There were no significant differences found between the four groups for the 1<sup>st</sup> MPJ ( $P = 0.10$ ). Significant differences between the four groups for the 2<sup>nd</sup> / 3<sup>rd</sup> MPJ ( $P = 0.01$ ) and 4<sup>th</sup> / 5<sup>th</sup> MPJ measurements ( $P = 0.02$ ) were found.



**Table 4.4: Peak plantar pressure measurements (KPa).**

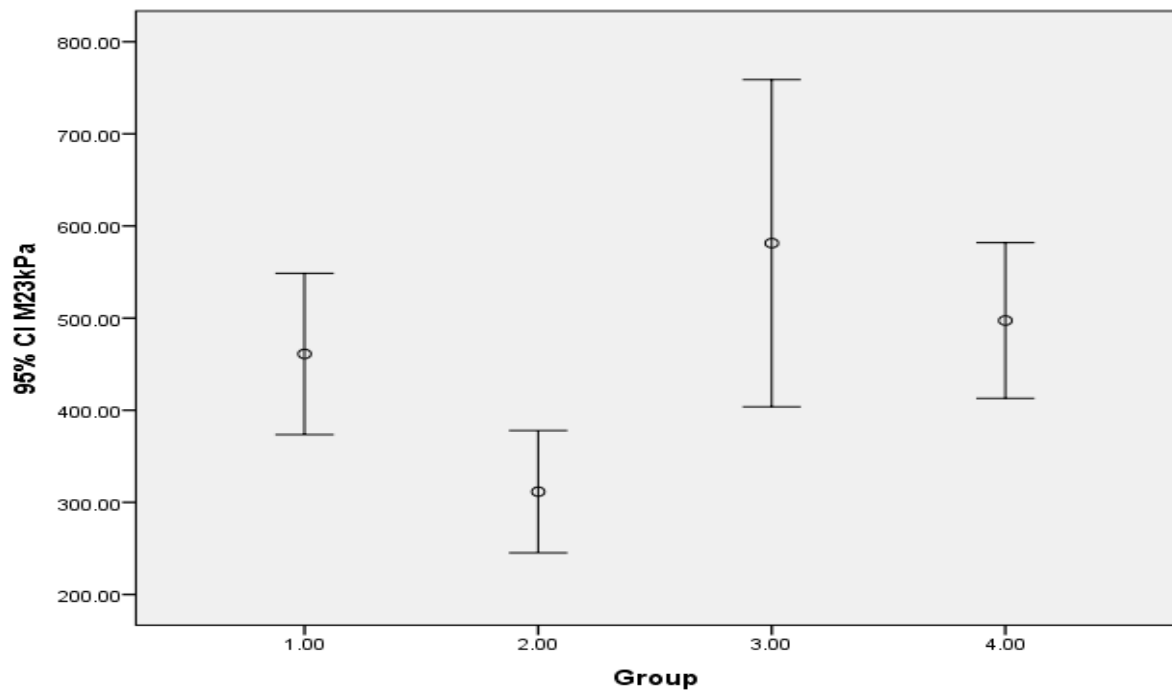
Group					
Peak pressure (KPa)	Non-Māori no DM Mean (SD)	Māori no DM Mean (SD)	Non-Māori with DM Mean (SD)	Māori with DM Mean (SD)	<i>P</i> -value
1 <sup>st</sup> MPJ	173.3 (69.9)	199.8 (64.1)	256.3 (94.0)	216.2 (73.3)	0.10
2 <sup>nd</sup> /3 <sup>rd</sup> MPJ	497.3 (109.9)	311.5 (92.7)	581.3 (191.9)	461.1 (122.2)	0.01
4 <sup>th</sup> /5 <sup>th</sup> MPJ	352.5 (128.5)	329.1 (90.9)	464.0 (126.6)	293.6 (84.5)	0.02

Table 4.5 demonstrates post-hoc testing between the four groups for peak plantar pressure. There was a significant difference between Māori with DM and Māori with no DM for the 2<sup>nd</sup> / 3<sup>rd</sup> MPJ ( $P = 0.01$ ); Māori with no DM and non-Māori with DM ( $P = 0.00$ ); and non-Māori with no DM and Māori with no DM ( $P = 0.04$ ). There were also significant differences between Māori with DM and non-Māori with DM for the 4<sup>th</sup> / 5<sup>th</sup> MPJ ( $P = 0.03$ ); Māori with no DM and non-Māori with DM ( $P = 0.02$ ) for the 4<sup>th</sup> / 5<sup>th</sup> MPJ. Figures 4.3 and 4.4 illustrate the 95% confidence intervals for plantar pressure readings of the 2<sup>nd</sup> / 3<sup>rd</sup> and 4<sup>th</sup> / 5<sup>th</sup> MPJ (KPa). We can therefore reject the null hypothesis 2: and state there are significant differences in forefoot peak plantar pressures between Māori and non-Māori with and without diabetes.

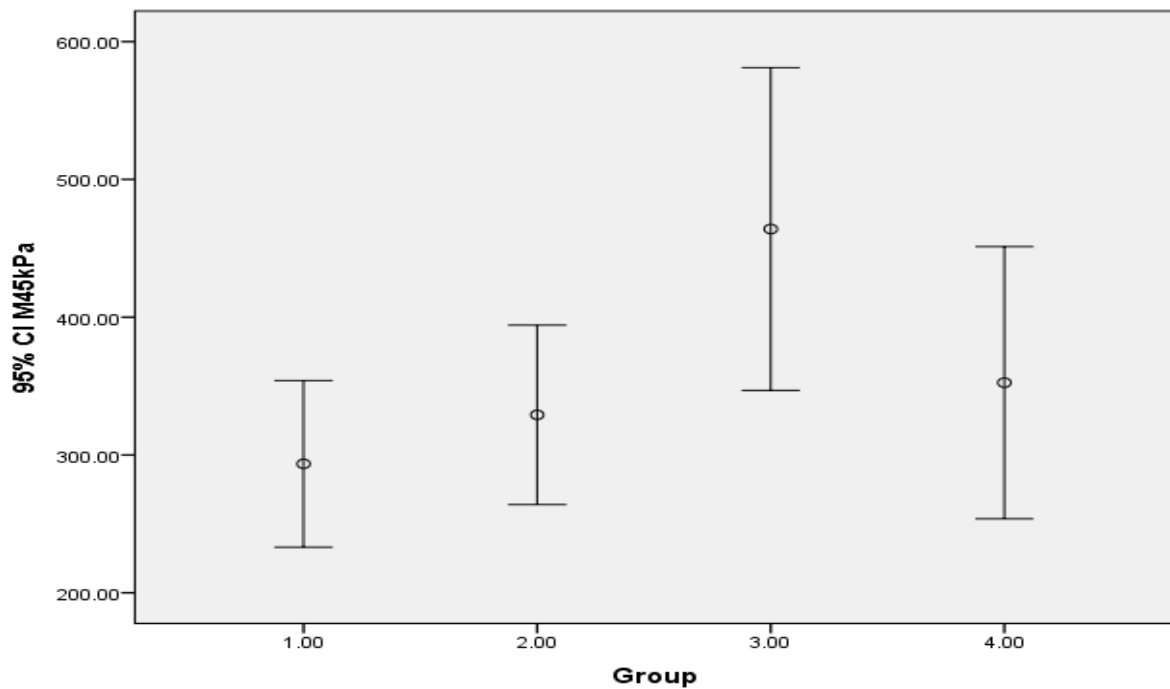
**Table 4.5: Post Hoc results for peak plantar pressure of the forefoot between groups.**

Variable	Group	Mean Difference (KPa)	P-value
1 <sup>st</sup> MPJ	Māori with DM vs. Māori with no DM	16.4	0.60
	Māori with DM vs. Non-Māori with DM	49.2	0.20
	Māori with DM vs. Non-Māori with no DM	42.9	0.20
	Māori no DM vs. Non-Māori with DM	65.6	0.80
	Māori no DM vs. Non- Māori with no DM	26.5	0.40
	Non- Māori with DM vs. Non- Māori with no DM	92.0	0.02*
2 <sup>nd</sup> /3 <sup>rd</sup> MPJ	Māori with DM vs. Māori with no DM	149.5	0.01*
	Māori with DM vs. Non-Māori with DM	120.3	0.70
	Māori with DM vs. Non-Māori with no DM	36.2	0.50
	Māori no DM vs. Non-Māori with DM	269.8	0.00*
	Māori no DM vs. Non- Māori with no DM	185.8	0.04*
	Non- Māori with DM vs. Non- Māori with no DM	84.0	0.20
4 <sup>th</sup> /5 <sup>th</sup> MPJ	Māori with DM vs. Māori with no DM	35.6	0.50
	Māori with DM vs. Non-Māori with DM	170.4	0.03*
	Māori with DM vs. Non-Māori with no DM	58.9	0.20
	Māori no DM vs. Non-Māori with DM	134.6	0.02*
	Māori no DM vs. Non- Māori with no DM	23.4	0.60
	Non- Māori with DM vs. Non- Māori with no DM	111.5	0.05

\*mean difference is significant at the 0.05 level



**Figure 4.3: 95% Confidence Intervals for 2<sup>nd</sup>/3<sup>rd</sup> MPJ (KPa). 1 = Māori with DM, 2 = Māori without DM, 3 = Non-Māori with DM, 4 = Non-Māori without DM.**



**Figure 4.4: 95% Confidence Intervals for 4<sup>th</sup>/5<sup>th</sup> MPJ (KPa). 1 = Māori with DM, 2 = Māori without DM, 3 = Non-Māori with DM, 4 = Non-Māori without DM.**

#### 4.3.2 Pressure time integrals

There were no significance differences across the forefoot regions. Table 4.6 demonstrates the mean (SD) findings of the forefoot MPJ regions for pressure time integral measurements (KPa/s). We can therefore accept the null hypothesis 3 and state there are no significant differences in forefoot pressure time integrals between Māori and non-Māori with and with no diabetes.

**Table 4.6: Pressure Time Integral measurements (KPa/s).**

Group					
Pressure Time Integral (KPa/s)	Non-Māori no DM Mean (SD)	Māori no DM Mean (SD)	Non-Māori with DM Mean (SD)	Māori with DM Mean (SD)	<i>P</i> -value
1 <sup>st</sup> MPJ	65.9 (32.2)	62.5 (27.9)	94.9 (44.0)	83.4 (34.5)	0.20
2 <sup>nd</sup> /3 <sup>rd</sup> MPJ	186.4 (88.7)	152.5 (54.8)	226.7 (87.5)	188.47 (53.3)	0.20
4 <sup>th</sup> /5 <sup>th</sup> MPJ	155.8 (108.1)	104.6 (25.7)	195.5 (100.4)	126.3 57.0)	0.10

#### 4.4 Correlation between plantar fascia thickness and forefoot plantar pressure in Māori

Table 4.7 demonstrates a correlation between plantar fascia thickness and plantar pressure in Māori with diabetes. This data reflects our secondary aim which was to determine the relationship between plantar fascia thickness and plantar pressures in Māori with diabetes.

**Table 4.7: Relationship between US and plantar pressure in Māori with DM**

Plantar fascia thickness (mm)	1 <sup>st</sup> MPJ (KPa)	2 <sup>nd</sup> /3 <sup>rd</sup> MPJ (KPa)	4 <sup>th</sup> /5 <sup>th</sup> MPJ (KPa)	1 <sup>st</sup> MPJ (KPa/s)	2 <sup>nd</sup> /3 <sup>rd</sup> MPJ (KPa/s)	4 <sup>th</sup> /5 <sup>th</sup> MPJ (KPa/s)
Pearson R-correlation	0.62	0.62	0.77	0.52	0.40	0.45
Sig. (2-tailed)	0.06	0.06	0.01	0.12	0.25	0.19

## **Chapter 5: Discussion**

The aim of this study was to evaluate significant differences in plantar fascia thickness, peak forefoot plantar pressures and pressure time integrals between Māori and non-Māori with and without diabetes. Peripheral neuropathy (sensory and motor) is responsible for changes in both structure and function of the foot in people with diabetes. Atrophy and reduced volume of intrinsic muscles, deformities of the foot structure such as claw toes and prominent metatarsal heads, reduced strength of the lower limb muscles, alterations of plantar soft tissue and denervation of tendinous and ligamentous structures have been reported in clinical observations and experimental studies (Andersen, Gadeberg, Brock, & Jakobsen A, 1997, Morag & Cavanagh, 1999; Robertson et al., 2002).

### **5.1 Plantar fascia thickness**

In the current study, the ultrasound examination demonstrated an overall increased thickening in the plantar fascia between Māori with diabetes and non-Māori with no diabetes; and non-Māori with and with no diabetes. Our findings are similar to previous studies of increased plantar fascia thickness in people with diabetes (Bolton et al., 2005; D'Ambrogi et al., 2003; D'Ambrogi et al., 2005; Duffin et al., 2002). Histomorphological and mechanical differences in soft tissue properties in people with diabetes compared to controls may reflect the thickness changes (Chao et al., 2011; de Oliveira et al., 2013; Klaesner, Hastings, Zou, Lewis & Mueller, 2002; Pai & Ledoux, 2010; Wang, Lee, Ledoux, 2011).

Previous studies have suggested that tendon thickness may be increased in physiological conditions because of the deposition of newly formed collagen fibres as by effect of exercise in subjects performing sport activities (Heinemeier & Kjaer, 2011; Parkinson, Samiric, Ilic, Cook, Handley, 2011). However, in diabetes, tendon thickness is associated to an increased packing density and abnormal morphology of collagen fibrils, which appear, at electron microscopy, twisted, curved, overlapping, and otherwise highly disorganized (Grant et al., 1997); water retention, due to the deposition of hydrophilic proteoglycans and/or inflammation, may contribute.

According to previous studies, these alterations result from the formation of advanced glycation end products (AGEs) in chronic hyperglycaemia, and from the subsequent cross-

linking within collagen fibres, which can deteriorate the biological and mechanical function of tendons and ligaments (Abate, Schiavone, Pelotti, & Salini, 2010; Abate, Schiavone, Pelotti, & Salini, 2011). Besides that, AGEs react with cell surface receptors, which, in turn, trigger cell-specific signalling, resulting in enhanced generation of reactive oxygen species and in a sustained upregulation of pro-inflammatory mediators (Shoji et al., 2006). Furthermore, AGEs may enhance cellular apoptosis in tendons via the expression of pro-apoptotic cytokines (Alikhani et al., 2005). Current evidence suggests that soft tissue changes can occur as early as twenty four days *in vivo* (de Oliveira et al., 2013). This may suggest that changes to the structure of soft tissue occurs before the clinical diagnosis of peripheral neuropathy.

Plantar fascia thickness has been reported to be thicker in Nigerians with diabetes compared to a control group (Udoh et al., 2010). The average plantar fascia thickness measurements in the Udoh et al (2010) study were similar to our findings for the control groups (Nigerian Control, 3.10mm *versus* non-Māori / Māori no diabetes control, 3.0mm). However, plantar thickness measurements were greater in the Nigerian group with diabetes compared to current findings (Nigerian with diabetes, 4.2mm *versus* non-Māori / Māori with diabetes, 3.4mm). The differences between our work and Udoh et al (2010) was the larger sample size, people without peripheral neuropathy and an older age group.

There are a number of risk factors that may have contributed to the increase in plantar fascia thickness. A previous study reported a correlation between increasing body mass index (BMI) and plantar fascia thickness in people with diabetes but with no peripheral neuropathy (Abate, Schiavone, Di Carlo & Salini, 2012). We found Māori with diabetes to be obese with BMI over 35. A recent systematic review reported that obesity is strongly associated with planus (low-arched) foot posture, pronated dynamic foot function and increased plantar pressures when walking (Butterworth, Landorf, Gilleard, Urquhart & Menz, 2014). Previous studies have also reported on differences in foot morphology in different ethnic groups (Dunn et al., 2006 as cited in Golightly, Hannan, Dufour & Jordan, 2012; Gurney et al., 2012). Evidence specific to the population in Aotearoa suggests that foot morphology differs between ethnic groups (Gurney, Kersting, & Rosenbaum, 2009). We found the foot type to be pronated in Māori with and with no diabetes, and in non-Māori with diabetes. This concept differs from previous studies where pes cavus had an incidence of over 50% in the diabetic population (Hastings et al., 2000), especially in patients with long-standing diabetes (Ledoux

et al., 2003). In studies carried out in people with diabetes, an increased prevalence of calcaneal pronation as a consequence of increased eversion and support of the inner foot arch was observed, resulting in stiffness of the plantar fascia, a more inefficient lower extremity, and a more unstable gait (Kirby, 2000; D'Ambrogi et al., 2005). In non-diabetic populations, a greater predisposition to developing pronated feet has been observed when BMI values are high (Messier et al., 1994).

## **5.2 Plantar forefoot pressures**

A flattening of the plantar arch and a tendency toward pronation with increased plantar pressure in the midfoot are increasingly identified in diabetic neuropathic patients (Armstrong, Todd, Lavery, Harkless & Bushman, 2003). High plantar pressures have been associated with foot ulceration in people with diabetes, who can experience loss of protective sensation due to peripheral neuropathy (Bennetts et al., 2012). We found lower peak plantar pressures for Māori compared to non-Māori with and with no diabetes. Solano et al. (2008) reported similar lower dynamic plantar peak pressures in a Hispanic population with diabetes and peripheral neuropathy compared to a Caucasian matched population. The findings were independent of age, weight and duration of diabetes and both the peak plantar pressures and pressure time integrals were significantly higher in the Caucasian population for the entire foot, forefoot and specifically in the 5<sup>th</sup> MPJ. The 5<sup>th</sup> MPJ has been reported to a predominant site for foot ulceration (Solano et al., 2008). Elevated pressures are believed to increase the risk of ulceration in the diabetic foot, particularly when combined with deformity and peripheral neuropathy (Lavery et al., 2003). Periyasamy, Anand and Ammini (2012) found in a North Asian Indian population that the plantar pressure distribution across the foot increased specifically in those with peripheral neuropathy, suggesting that peripheral neuropathy is the underlying feature in the alteration of foot pressure distribution. In terms of specific regions of interest, our findings showed the peak plantar pressure was increased in the 4<sup>th</sup> / 5<sup>th</sup> MPJ in both diabetes groups.

Previous studies have reported differences in peak plantar pressures across different ethnic groups in people with diabetes compared to controls (Gurney et al., 2012; Periyasamy et al., 2012; Solano et al., 2008; McPoil, Yamada, Smith & Cornwall, 2001). Gurney et al. (2012) reported significant peak plantar pressure differences between Māori and non-Māori with diabetes at the central forefoot. McPoil et al. (2001) found peak plantar pressure was higher



in the central forefoot in American Indians with peripheral neuropathy. Our findings showed non-Māori with diabetes demonstrated the highest peak plantar pressure at the central forefoot. van Schie et al. (2011) reported that peak plantar pressures were reduced in an Asian population with diabetes. Our findings are similar to Solano et al. (2008) and van Schie et al (2011).

A study of healthy Māori and non-Māori reported differences in pressure time-integrals particularly between the 2/3<sup>rd</sup> MPJ, where Māori had higher pressure time integrals compared to Caucasians (Gurney et al., 2009). These findings are similar to current findings. However, we found no significant differences between the four groups across the forefoot for pressure time integrals. Bus and Waaijman (2013) questioned the relevance of reporting both peak plantar pressures and pressure-time integrals and state that the outcome of each parameter is interchangeable. Bus & Waaijman (2013) undertook a systematic review on studies reporting both peak plantar pressure and pressure time integral in the diabetic foot. The aim of the review was to determine the value of reporting pressure time integral in addition to peak plantar pressure. This is due to a majority of studies collecting pressure time integral values, but not showing differences between pressure time integrals or reporting the meaning of these differences. Of thirty five eligible articles, fifteen discussed pressure time integral but only five gave meaningful explanations in comparison to peak plantar pressure measurements. The authors concluded that the limitations noted in the studies provided sufficient evidence for researchers to report both or use one as the main indicator of plantar pressures. For individual regions, peak plantar pressure is more discriminative. Pressure time integral may be useful in studies using different walking speeds and sensor size.

### **5.3 Plantar fascia thickness and increased plantar pressures in Māori**

The secondary aim of this study was to determine the relationship between plantar fascia thickness and plantar forefoot pressures in Māori with diabetes. A significant correlation between plantar fascia thickness and peak plantar pressure at the 4 / 5<sup>th</sup> MPJ was observed in Māori with diabetes. These results support earlier findings by Solano et al. (2008) and represent a region that maybe a potential area for high risk of ulceration for Māori. This is the first study to support that these characteristics are different between two populations in Aotearoa. The plantar fascia plays an important role in sustaining the longitudinal arch of

the foot during propulsion, when it acts as a beam and the metatarsals undergo a bending stress to absorb impact forces at the midtarsal joint during landing and stance, where it acts as a truss and the metatarsals undergo a compressive stress (Hicks, 1954; Salathé, Arangio & Salathé, 1986). In order to correctly act under these two conditions, the plantar fascia needs adequate elasticity to manage the tensile force due to the synergic action of Achilles tendons and metatarsophalangeal joints. Although joint range of motion data was not obtained in the current study, the findings suggest that in Māori, the Windlass mechanism may have been affected by plantar fascia thickening and altering pressure distribution prior to propulsion.

## **5.4 Limitations**

The current work was a pilot study to determine plantar fascia thickness and plantar pressure measurements in Māori with diabetes with a view for further investigation. As the sample size was low, we cannot generalise the current findings to all Māori. The small sample size also enhances the possibility of a type 1 statistical error. The reasons for the small sample size were limited access to resources and flexibility in appointment times. We did not measure 1<sup>st</sup> MPJ joint range of motion and dynamic function of the plantar fascia. The Windlass mechanism describes how the plantar fascia performs during weight-bearing and is concerned with the amount of 1<sup>st</sup> MPJ range of motion. Therefore, 1<sup>st</sup> MPJ dorsiflexion stiffness cannot be ruled out as a contributing factor to the correlation between plantar fascia thickness and plantar pressures in Māori and is a recommendation for further research (Chuter & Payne, 2001). Another limitation of the study was determining if footwear choice impacted on the main outcomes of the study. This could be a recommendation of future studies.

## **5.5 Future recommendations**

The preliminary findings in the current study require further exploration to determine predictors of ulcer formation particularly for Māori with diabetes and peripheral neuropathy. However, a larger sample population would allow data to be generalizable and applicable within the context of Aotearoa. Although Māori displayed lower peak plantar pressures overall, our findings demonstrated peak plantar pressures that were above the barefoot threshold of 700 KPa for the entire forefoot (mean Māori with diabetes: 971 KPa) that has been suggested as the cut-off point prior to skin breakdown (Armstrong, Peters, Athanasiou,

& Lavery, 1998; Maluf & Mueller, 2003). However, other variables, for example BMI, foot morphology, soft tissue thickness, dynamic gait characteristics and previous history of foot ulceration, should be considered as risk factors leading to high plantar pressures which complicates defining specific barefoot thresholds (Patry, Belley, Côté, Chateau-Degat, 2013). Exploring these predictors utilising the current study population may help explain why Māori demonstrated lower peak plantar pressures across groups.

Future recommendations may include the use ultrasound measurements of the plantar fascia (central, lateral and medial bands) and Achilles tendon (calcaneal insertion), with both tendons placed under slight tension to represent of the tendon during dynamic motion (Giacomozzi et al., 2005; Vohra, Kincaid, Japour & Sobel, 2002). The Achilles tendon and plantar fascia work synergistically to maintain the Windlass mechanism, therefore, a prospective observational study to determine this mechanism would be useful. An adjunct to this observation would be to consider the major muscles that contribute to the balance between pronation and supination affecting the integrity of the Windlass mechanism to include the posterior tibialis, flexor digitorum longus, flexor hallucis longus and peroneus longus muscles (Bolgla & Malone, 2004).

Early studies reported that shifting from an ankle to hip strategy is required in people with diabetes (Andersen et al., 1997; Hunt, Smith & Torode, 2001 as reported in Giacomozzi et al. (2002). This was demonstrated by decreases in medio-lateral and longitudinal centre of pressure excursions contributing to a functional flatfoot and a hip-based walking strategy in people with diabetes and neuropathy (Giacomozzi et al., 2002). These functional changes require further exploration, particularly in terms of soft tissue changes occurring prior to the clinical onset of peripheral neuropathy and the consequences to joint structure and gait parameters. To our knowledge, there is no information regarding walking strategy in Māori with diabetes and peripheral neuropathy, or comparatively to people without neuropathy. However, a study involving a similar population (American Indians) who are at the highest risk of amputation in America (Kernozek, Greany & Heizler, 2013) have found that gait patterns, specifically double support time and unsteadiness during barefoot walking were significant in this population with peripheral neuropathy (Najafi, Khan, Fleischer & Wrobel, 2013). These characteristics are linked to an increased risk and fear of falling particularly in the aging diabetes population (Kelly et al., 2013). Therefore future directions necessitates the use of 3D gait analysis to determine spatiotemporal parameters that would provide

information concerning the parameters above including walking speeds and a wider base of gait in people with diabetes and neuropathy (Wrobel, Crews, & Connolly, 2009; Wrobel & Najafi, 2010).

Management of long term conditions in Aotearoa is patient-centred and requires an integrated approach. Future studies require patient outcome measures such as activities of daily living and quality of life measures. These recommendations form the basis of a larger study that necessitates collaboration and integration of community and district wide services to improve the understanding of the structural and biomechanical predictors of diabetic foot ulcerations in Māori.

## **Chapter 6: Conclusion**

Diabetic foot ulcerations are not a spontaneous event, and Māori are more vulnerable to foot related diabetes complications than non-Māori. We are not aware of any studies identifying soft tissue and plantar pressure differences between Māori and non-Māori with peripheral neuropathy which have been discussed in relation to the impact on biomechanical function in diabetes.

The results from this study have provided preliminary data on plantar fascia thickness and plantar pressures in the foot of Māori with diabetes. Our findings reject two of three null hypotheses, stating that there are significant differences in plantar fascia thickness and peak plantar pressures between Māori and non-Māori with and with no diabetes. It accepts the third hypothesis that there were no significant differences in forefoot pressure time integrals between Māori and non-Māori with and with no diabetes. However, a relationship between plantar fascia thickness and plantar forefoot pressures was demonstrated at the 4<sup>th</sup> and 5<sup>th</sup> metatarsophalangeal joint in Māori with diabetes. Limitations of this study have been discussed in terms of small sample sizes and have been taken into consideration for future research. Future recommendations include exploring other variables that have contributed to these findings in an effort to identify predictors that lead to diabetic foot ulcerations in Māori with diabetes.

## Chapter 7: References

- Abate, M., Schiavone, C., Pelotti, P., & Salini, V. (2010). Limited joint mobility in diabetes and ageing: recent advances in pathogenesis and therapy. *International Journal of Immunopathology & Pharmacology*, 23(4), 997-1003.
- Abate, M., Schiavone, C., Pelotti, P., & Salini, V. (2011). Limited joint mobility (LJM) in elderly subjects with type II diabetes mellitus. *Archives of gerontology and geriatrics*, 53(2), 135-140.
- Abate, M., Schiavone, C., Carlo, L.D., & Salini, V. (2012). Achilles tendon and plantar fascia in recently diagnosed type II diabetes: role of body mass index. *Clinical Rheumatology*, 31, 1109-1113. doi: 10.1007/s10067-012-1955-y.
- Abouaesha, F., van Schie, C.H., Griffiths, G.D., Young, R.J., & Boulton, A.J.M. (2001). Plantar tissue thickness is related to peak plantar pressure in the high-risk diabetic foot. *Diabetes Care*, 24(7), 127-1274. doi:10.2333/diacare.24.7.1270.
- Abouaesha, F., van Schie, C.H.M., Armstrong, D.G., & Boulton, A.J.M. (2004). Plantar soft-tissue thickness predicts high peak plantar pressure in the diabetic foot. *Journal of the American Podiatric Association*, 94(1), 39-42.
- Alikhani, Z., Alikhani, M., Boyd, C.M., Nagao, K., Trackman, P.C., Graves, D.T. (2005). Advanced glycation end products enhance expression of pro-apoptotic genes and stimulate fibroblast apoptosis through cytoplasmic and mitochondrial pathways. *The Journal of Biological Chemistry*, 280(13), 12087-95. doi:10.1074/jbc.M406313200.
- Andersen, H., Gadeberg, P.C., Brock, B., & Jakobsen, J. (1997). Muscular atrophy in diabetic neuropathy: a stereological magnetic resonance imaging study. *Diabetologia*, 40, 1062-1069.
- Andersen, H., Gjerstad, M.D., & Jakobsen, J. (2004). Atrophy of intrinsic foot muscles - a measure of diabetic neuropathy? *Diabetes Care*, 27, 2382-2385.
- Apelqvist J., Larsson, J., & Agardh, C.D. (1993). Long-term prognosis for diabetic patients with foot ulcers. *Journal of Internal Medicine*, 233(6), 485-491. doi:10.1111/j.1365-2796.1993.tb01003.x.
- Armstrong, D.G., Peters, E.J., Athanasiou, K.A., & Lavery, L.A. (1998). Is there a critical level of plantar foot pressure to identify patients at risk for neuropathic foot ulceration? *Journal of Foot and Ankle Surgery*, 37(4), 303-307.
- Armstrong, D.G., Todd, W.F., Lavery, L.A., Harkless, L.B., Bushman, T.R. (2003). The natural history of acute Charcot's arthropathy in a diabetic foot specialty clinic. *Journal of the American Podiatric Medical Association*, 87(6):272-8. doi: 10.1002/(SICI)1096-9136.
- Barton, C. J., Bonanno, D., & Menz, H.B. (2009). Development and evaluation of a tool for the assessment of footwear characteristics. *Journal of Foot and Ankle Research*, 2, 1-10. doi:10.1186/1757-1146-2-10.

- Benitez-Aguirre, P.Z., Craig, M.E., Jenkins, A.J., Gallego, P.H., Cusumano, J., Duffin, A.C....Donaghue, K.C. (2012). Plantar fascia thickness is longitudinally associated with retinopathy and renal dysfunction: A prospective study from adolescence to adulthood. *Journal of Diabetes Science and Technology*, 6(2), 348-355.
- Bennetts, C.J., Owings, T.M., Erdemir, A., Botek, G. & Cavanagh, P.R. (2013). Clustering and classification of regional peak plantar pressures of diabetic feet. *Journal of Biomechanics*, 4(46), 19-25. doi:10.1016/j.jbiomech.2012.09.007.
- Blankenbaker, D., & De Smet, A. (2006.). The role of ultrasound in the evaluation of sports injuries of the lower extremities. *Clinical Sports Medicine*, 897. doi:10.1016/J.CSM.2006.06.004.
- Bojsen-Møller, F. (1979). Calcaneocuboid joint stability of the longitudinal arch of the foot at high and low gear push off. *Journal of Anatomy*, 129, 167-176.
- Bolgla, L.A., Malone, T.R. (2004). Plantar fasciitis and the Windlass mechanism: A biomechanical link to clinical practice. *Journal of Athletic Training*, 39(1), 7782.
- Bolton, N.R., Smith, K.E., Pilgram, T.K., Mueller, M.J., & Bae, K.T. (2005). Computed tomography to visualize and quantify the plantar aponeurosis and flexor hallucis longus tendon in the diabetic foot. *Clinical Biomechanics*, 20, 540-546. doi:10.1016/j.clinbiomech.2004.12.007.
- Boulton, A.J.M. (2005). Management of diabetic peripheral neuropathy. *Clinical Diabetes*, 23(1), 9-15.
- Boulton, A.J.M. (2010). What you can't feel can't hurt you. *Journal of Vascular Surgery*, 52(3 Supplement), 28S-30S. doi:10.1016/j.jvs.2010.06.005.
- Brem, H., Sheehan, P., & Boulton, A.J.M. (2004). Protocol for treatment of diabetic foot ulcers. *The American Journal of Surgery*, 187(5), S1-S10. doi:10.1016/S0002-9610(03)00299-X.
- Browne, J., Garrett, N. (2010). *An intervention trial for Māori at risk of diabetes-related lower limb pathology*. Retrieved from AUT University, Taupua Waiora Centre for Māori Health Research website:  
[http://www.aut.ac.nz/\\_data/assets/pdf\\_file/0005/389075/FEET-FOR-LIFE-Final.pdf](http://www.aut.ac.nz/_data/assets/pdf_file/0005/389075/FEET-FOR-LIFE-Final.pdf)
- Bus, S.A., Yang, Q.X., Wang, J.H., Smith, M.B., Wunderlich, R., & Cavanagh, P.R. (2002). Intrinsic muscle atrophy and toe deformity in the diabetic neuropathic foot: a magnetic resonance imaging study. *Diabetes Care*, 25, 1444-1450.
- Bus, S.A., Ulbrecht, J.S., & Cavanagh, P.R. (2004). Pressure relief and load redistribution by custom-made insoles in diabetic patients with neuropathy and foot deformity. *Clinical Biomechanics*, 19, 629-638. doi:10.1016/j.clinbiomech.2004.02.010.
- Bus, S.A., & de Lange, A. (2005). A comparison of the 1-step, 2-step, and 3-step protocols for obtaining barefoot plantar pressure data in the diabetic neuropathic foot. *Clinical Biomechanics*, 20, 892-899. doi:10.1016/j.clinbiomech.2005.05.004.

- Bus, S.A., Mass, M., de Lange, A., Michels, R.P.J., & Levi, M. (2005). Elevated plantar pressure in neuropathic diabetic patients with claw/hammer toe deformity. *Journal of Biomechanics*, 38(9), 1918-1925. doi:10.1016/j.jbiomech.2004.07.034.
- Bus, S.A., Haspels, R., & Busch-Westbroek, T.E. (2011). Evaluation and optimization of therapeutic footwear for neuropathic diabetic foot patients using in-shoe plantar pressure analysis. *Diabetes Care*, 34, 1595-1600. doi:10.2337/dc10-2206.
- Bus, S. A., & Waaijman, R. (2013). The value of reporting pressure-time integral data in addition to peak pressure data in studies on the diabetic foot: A systematic review. *Clinical Biomechanics*, 28, 117-121. doi:10.1016/j.biomech.2012.12.002.
- Butterworth, P.A., Landorf, K.B., Gilleard, W., Urquhart, D.M., Menz, H.B. (2014). The association between body composition and foot structure and function: a systematic review. *Obesity Reviews*, 15(4), 348–357. doi:1111/obr.12130.
- Cameron, N. E., Eaton, S.E.M., Cotter, M.A., & Tesfaye, S. (2001). Vascular factors and metabolic interactions in the pathogenesis of diabetic neuropathy. *Diabetologia*, 44, 1973-1988.
- Cardinal, E., Chhem, R.K., Beauregard, C.G., Aubin, B., & Pelletier, M. (1996). Plantar fasciitis: sonographic evaluation. *Radiology*, 201(1), 257-259.
- Caselli, A., Pham, H., Giurini, J.M, Armstrong, D.G., & Veves, A. (2002). The forefoot-to-rearfoot plantar pressure ratio is increased in severe diabetic neuropathy and can predict foot ulceration. *Diabetes Care*, 25(6), 1066-1071. doi:10.2337/diacare.25.6.1066.
- Chantelau, E., & Haage, P. (1994). An audit of cushioned diabetic footwear: relation to patient compliance. *Diabetes Medicine*, 11(1), 114-116. doi:10.1111/j.1464-5491.1994.
- Chao, C.Y.L., Zheng, Y.P., & Cheing, G.L.Y. (2011). Epidermal thickness and biomechanical properties of plantar tissues in diabetic foot. *Ultrasound in Medicine & Biology*, 37(7), 1029-1038. doi:10.1016/j.ultramedbio.2011.04.004.
- Chimutengwende-Gordon, M., O'Donnell, P., & Singh, D. (2010). Magnetic resonance imaging in plantar heel pain. *Foot & Ankle International*, 31(10), 865-870. doi:10.3113/FAI.2001.0865.
- Chuter, V., & Payne, C. (2001). Limited joint mobility and plantar fascia function in Charcot's neuroarthropathy. *Diabetes Medicine*, 18(7), 558-561. doi:10.1046/j.1464-5491.2001.00827.
- Coppell, K.J., Mann, J.I., Williams, S.M., Jo, E., Drury, P.L., Miller, J., & Parnell, W.R. (2013). Prevalence of diagnosed and undiagnosed diabetes and prediabetes in New Zealand: findings from the 2008/09 Adult Nutrition Survey *New Zealand Medical Journal*, 126(1370), 23-42.
- Craig, M.E., Duffin, A.C, Gallego, P.H., Lam, A., Cusumano, J., Hing, S., et al. (2008). Plantar fascia thickness, a measure of tissue glycation, predicts the development of



- complications in adolescents with type 1 diabetes. *Diabetes Care*, 31(6), 1201-1207. doi:10.2337/dc07-2168.
- Cuaderes, E., Lamb, W.L., Khan, M., & Lawrence, G. (2009). Weight-bearing activity and foot parameters in native Americans with diabetes with and without foot sensation. *Journal of Cultural Diversity*, 16(1), 10-20.
- D'Ambrogi, E., Giurato, L., D'Agostino, M.A., Giacomozzi, C., Macellari, V., Caselli, A., et al. (2003). Contribution of plantar fascia to the increased forefoot pressures in diabetic patients. *Diabetes Care*, 26(5), 1525-1529.
- D'Ambrogi, E., Giacomozzi, C., Macellari, V., & Uccioli, L. (2005). Abnormal foot function in diabetic patients: The altered onset of Windlass mechanism. *Diabetic Medicine*, 22, 1713-1719. doi:10.1111/j.1464-5491.2005.01699.x.
- Dargis, V., Pantelejeva, O., Jonushaite, A., Vileikyte, L., & Boulton, A.J.M. (1999). Benefits of a Multidisciplinary Approach in the Management of Recurrent Diabetic Foot Ulceration in Lithuania. A prospective study. *Diabetes Care*, 22(9), 1428-1431. doi:10.2337/diacare.22.9.1428.
- de Oliveira, R.R., Lemos, A., de Castro Silveira, P.V., da Silva, R.J., & de Moraes, S.R.A. (2011). Alterations of tendons in patients with diabetes mellitus: a systematic review. *Diabetic Medicine*, 28, 886-895. doi:10.1111/j.1464-5491.2010.03197.x.
- Duffin, A.C., Donaghue, K.C., Potter, M., McInnes, A., Chan, A.K., King, J., Silink, M. (1999). Limited joint mobility in the hands and feet of adolescents with Type 1 diabetes mellitus. *Diabetes Medicine*, 16(2), 125-130. doi:10.1046/j.1464-5491.1999.00030.x.
- Duffin, A.C., Lam, A., Kidd, R., Chan, A.K.F., & Donaghue, K.C. (2002). Ultrasonography of plantar soft tissue thickness in young people with diabetes. *Diabetic Medicine*, 19, 1009-1013.
- Dunn, J.E., Link, C.L., Felson, D.T., Crincoli, M.G., Keysor, J.J., & McKinaly, J.B. (2004). Prevalence of foot and ankle conditions in a multi-ethnic community sample of older adults. *American Journal of Epidemiology*, 159(5), 491-498. doi:10.1093/aje/kwh071.
- Edmonds, M.E., & Foster, A.V. (2006). Diabetic foot ulcers. *British Medical Journal*, 18(332), 407-410. doi:10.1136/bmj.332.7538.407.
- Ellis, E., Ballance, K., Lunt, H., & Lewis, D. (2010). Diabetes outpatient care before and after admission for diabetic foot complications. *Journal of Wound Care*, 19(4), 150-152.
- Fabrikant, J., & Park, T. (2011). Plantar fasciitis (fasciosis) treatment outcome study: Plantar fascia thickness measured by ultrasound and correlated with patient self-reported improvement. *The Foot*, 21(2), 79-83. doi:10.1016/j.foot.2011.01.015.
- Figuerola-Romero, C., Sadidi, M., & Feldman, E.L. (2008). Mechanisms of disease: the oxidative stress theory of diabetic neuropathy. *Reviews in Endocrine and Metabolic Disorders*, 9, 301-314. doi:10.1007/s11154-0087-9104-2.

- Formosa, C., Gatt, A., & Chockalingam, N. (2013). The importance of clinical biomechanical assessment of foot deformity and joint mobility in people living with type-2 diabetes within a primary care setting. *Primary Care Diabetes*, 7, 45-50. doi:10.1016/j.pcd.2012.12.003.
- Frykberg, R.G., Lavery, L.A., Pham, H., Harvey, C., Harkless, L., & Veves, A. (1998). Role of neuropathy and high foot pressures in diabetic foot ulceration. *Diabetes Care*, 21(10), 1714-1719.
- Garrett, M., & Ihaka, B. (2013, May). *Save our Soles: An audit of a District Health Board incentive to improve amputation rates*. Paper presented at the meeting of the Sydney Diabetic Foot Conference, Sydney, Australia.
- Genc, H., Saracoglu, M., Nacir, B., Erdem, H., & Kacar, M. (2005). Long-term ultrasonographic follow-up of plantar fasciitis patients treated with steroid injection. *Joint, Bone & Spine*. 72(1), 61-65. doi:10.1016/j.jbspin.2004.03.006.
- Gerstein, H. & Hayness, R. (Eds.). (2001). Peripheral Nerve Disease. In *Evidence-Based Diabetes Care* (pp. 466-479). BC Decker: London.
- Giacomozzi, C., Caselli, A., Macellari, V., Giurato, L., Lardieri, L., & Uccioli, L. (2002). Walking strategy in diabetic patients with peripheral neuropathy. *Diabetes Care*, 25(8), 1451-1453.
- Giacomozzi, C., D'Ambrogi, E., Uccioli, L., & Macellari, V. (2005). Does the thickening of the Achilles tendon and plantar fascia contribute to the alteration of diabetic foot loading? *Clinical Biomechanics*, 20, 532-539. doi:10.1016/j.clinbiomech.2005.01.011.
- Giacomozzi, C., & Martelli, F. (2006). Peak pressure curve: an effective parameter for early detection of foot functional impairments in diabetic patients. *Gait & Posture*, 23(4), 464-470. doi:10.1016/j.gaitpost.2005.06.006.
- Gibbon, W., & Long, G. (1999). Ultrasound of the plantar aponeurosis (fascia). *Skeletal Radiology*, 28, 21-26.
- Golightly, Y.M., Hannan, M.T., Dufour, A.B., & Jordan, J.M. (2012). Racial differences in foot disorders and foot type. *Arthritis Care & Research*, 64(11), 1756-1759. doi:10.1002/acr.21752.
- Grant, W.P., Sullivan, R., Sonenshine, D.E., Adam, M., Slusser, J.H., Carson, K.A., Vinik, A.L. (1997). Electron microscopic investigation of the effects of diabetes mellitus on the Achilles tendon. *Journal of Foot & Ankle Surgery*, 36(4), 272-8.
- Guo, S., Zhong, S., Zhang, A. (2013). Privacy-preserving Kruskal-Wallis test. *Computer Methods and Programs in Biomedicine*, 112, 135-145. doi:10.1016/j.cmpb.2013.05.023.
- Gurney, J.K., Kersting, U.G., & Rosenbaum, D. (2009). Dynamic foot function and morphology in elite rugby league athletes of different ethnicity. *Applied Ergonomics*, 40, 554-559. doi:10.1016/j.apergo.2008.11.001.

- Gurney, J.K., Kuch, C., Rosenbaum, D., & Kersting, U.G. (2012). The Māori foot exhibits differences in plantar loading and midfoot morphology to the Caucasian foot. *Gait & Posture*, 36(1), 157-159. doi:10.1016/j.gaitpost.2012.01.013.
- Hashefi, M. (2011). Ultrasound in the diagnosis of noninflammatory musculoskeletal conditions. *Seminars in Ultrasound, CT and MRI*, 32, 74-90. doi:10.1053/j.sult.2010.12.005.
- Hastings, M.K., Mueller, M.J., Sinacore, D.R., Salsich, G.B., Engsberg, J.R., Johnson, J.E. (2000). Effects of a tendo-Achilles lengthening procedure on muscle function and gait characteristics in a patient with diabetes mellitus. *Journal of Orthopaedic & Sports Physical Therapy*, 30(2), 85–90. doi: 10.2519/jospt.2000.30.2.85.
- Heinemeier, K.M., Kjaer, M. (2011). In vivo investigation of tendon responses to mechanical loading. *Journal of Musculoskeletal & Neuronal Interactions*, 11(2), 115–123.
- Hicks, J.H. (1954). The mechanics of the foot. II. The plantar aponeurosis and the arch. *Journal of Anatomy*, 88, 25-30.
- Hsi, W.L., Chai, H.M., & Lai, J.S. (2002). Comparison of pressure and time parameters in evaluating diabetic footwear. *American Journal of Physical Medicine in Rehabilitation*, 81(11), 822-899.
- Huerta, J.P., & Garcia, J.M.A. (2007). Effect of gender, age and anthropometric variables on plantar fascia thickness at different locations in asymptomatic subjects. *European Journal of Radiology*, 62, 449-453. doi:10.1016/j.ejrad.2007.01.002.
- Huijberts, M.S.P., Schaper, N. C., & Schalkwijk, C.G. (2008). Advanced glycation end products and diabetic foot disease. *Diabetes /Metabolism Research and Reviews*, 24 (Suppl 1), S19-S24.
- Hunt, A.E., Smith, R.M., & Torode, M. (2001). Extrinsic muscle activity, foot motion and ankle joint moments during the stance phase of walking. *Foot & Ankle International*, 22, 31-41.
- Ihaka, B., Bayley, A., & Rome, K. (2012). Foot problems in Māori with diabetes. *New Zealand Medical Journal*, 125(1360), 48-56.
- Jack, M., & Wright, D. (2012). Role of advanced glycation endproducts and glyoxalase I in diabetic peripheral sensory neuropathy. *Translational Research*, 159(355-365).
- Keenan, A., Redmond, A., Horton, M., Conaghan, P., & Tennant, A. (2007). The foot posture index: Rasch analysis of a novel, foot-specific outcome measure. *Archives of Physical Medicine and Rehabilitation*, 88(1), 88-93. doi:10.1016/j.apmr.2006.10.005.
- Kelly, C., Fleischer, A., Yalla, S., Grewal, G.S., Albright, R., Berns, D...Najafi, B. (2013). Fear of falling is prevalent in older adults with diabetes mellitus but is unrelated to level of neuropathy. *Journal of the American Podiatric Medical Association*, 103(6), 480-488. doi:10.7547/1030480.
- Kernozek, T.W., Greany, J.F., Heizler, C. (2013). Plantar loading asymmetry in American Indians with diabetes and peripheral neuropathy, with diabetes only, and without

- diabetes. *Journal of the American Podiatric Medical Association* 103(2), 106-112. doi: 10.7547/1030106.
- Kirby, K.A. (2000). Biomechanics of the normal and abnormal foot. *Journal of the American Podiatric Medical Association*, 90(1), 1–5. doi: 10.7547/87507315-90-1-30.
- Klaesner, J.W., Hastings, M.K., Zou, D.Q., Lewis, C., & Mueller, M.J. (2002). Plantar tissue stiffness in patients with diabetes mellitus and peripheral neuropathy. *Archive of Physical Medicine and Rehabilitation*, 83, 1796-1801. doi:10.1053/apmr.2002.356661.
- Ledoux, W.R., Shofer, J.B., Ahroni, J.H., Smith, D.G., Sangeorzan, B.J., Boyko, E.J. (2003). Biomechanical differences among pes cavus, neutrally aligned, and pes planus feet in subjects with diabetes. *Foot & Ankle International*, 24(11):845–9. doi:10.1177/107110070302401107.
- Mahowald, S., Legge, B.S., & Grady, J.F. (2011). The correlation between plantar fascia thickness and symptoms of plantar fasciitis. *Journal of the American Podiatric Association*, 101(5), 385-389.
- Maluf, K.S., Mueller, M.J. (2003). Comparison of physical activity and cumulative plantar tissue stress among subjects with and without diabetes mellitus and a history of recurrent plantar ulcers. *Clinical Biomechanics*, 18(7), 567-575. doi:10.1016/S0268-0033(03)00118-9.
- Mauri Ora Associates. (2008). *Best health outcomes for Māori: Practice implications*. Medical Council of New Zealand. Retrieved from <http://www.mcnz.org.nz/assets/News-and-Publications/Statements/best-health-Māoricomplete.pdf>
- McPoil, T.G., Yamada, W., Smith, W., & Cornwall, M. (2001). The distribution of plantar pressures in American Indians with diabetes mellitus. *Journal of the American Podiatric Association*, 91(6), 280-287.
- Messier, S.P., Davies, A.B., Moore, D.T., Davis, S.E., Pack, R.J., Kazmar, S.C. (1994). Severe obesity: effects on foot mechanics during walking. *Foot and Ankle International*, 15, 29–34. doi:10.1177/107110079401500106.
- Melai, T., Ijzerman, T.H., Schaper, N.C., de Lange, T.L.H., Willems, P.J.B., Meijer, K., et al. (2011). Calculation of plantar pressure time integral, an alternative approach. *Gait & Posture*, 34, 379-383. doi: 10.1016/j.gaitpost.2011.06.005.
- Ministry of Health. (2007). *Diabetes Surveillance Population-based estimates and projections for New Zealand, 2001–2011* (Public Health Intelligence Occasional Bulletin No. 46). Retrieved from [http://www.moh.govt.nz/moh.nsf/pagesmh/6796/\\$File/diabetes-suveillance-population-estimates-projections-2001-2011.pdf](http://www.moh.govt.nz/moh.nsf/pagesmh/6796/$File/diabetes-suveillance-population-estimates-projections-2001-2011.pdf)
- Ministry of Health. (2012). *Mortality and Demographic Data 2009*. Retrieved from 3, <http://www.health.govt.nz/publication/mortality-and-demographic-data-2009>

- Ministry of Health. (2013). *Māori Health*. Retrieved from <http://www.health.govt.nz/our-work/populations/Māori-health>
- Miyazaki, S., Nakayama, H., & Horiuchi, S. (2002). Scavenger Receptors that Recognize Advanced Glycation End Products. *Trends in Cardiovascular Medicine*, 12(6), 258-262. doi:10.1016/S1050-1738(02)00171-8.
- Morag, E., Cavanagh, P.R. (1999). Structural and functional predictors of regional peak pressures under the foot during walking. *Journal of Biomechanics*, 32(4), 359-70. doi:10.1016/S0021-9290(98)00188-2.
- Mueller, M.J., Hastings, M., Commean, P.K., Smith, K.E., Pilgram, T.K., Robertson, D., et al. (2003). Forefoot structural predictors of plantar pressures during walking in people with diabetes and peripheral neuropathy. *Journal of Biomechanics*, 36, 1009-1017. doi:10.1016/S0021-9290(03)00078-2.
- Mueller, M.J., Zou, D., Bohnert, K.L., Tuttle, L.J., & Sinacore, D.R. (2008). Plantar stresses on the neuropathic foot during barefoot walking. *Physical Therapy*, 88(11), 1375-1384.
- Nabuurs-Franssen, M.H., Huijberts, M.S.P., Kruseman, A.C.N, Willems, J., & Schaper, S.C. (2005). Health-related quality of life of diabetic foot ulcer patients and their care-givers. *Diabetologia*, 48, 1906-1910. doi:10.1007/s00125-005-1856-6.
- Narváez, J.A., Narváez, J., Ortega, R., Aguilera, C., Sánchez, A., & Andía, E. (2000). Painful Heel: MR imaging findings. *Radiographics*, 20, 333-352.
- Najafi, B., Khan, T., Fleischer, A., Wrobel, J. (2013). The impact of footwear and walking distance on gait stability in diabetic patients with peripheral neuropathy. *Journal of the American Podiatric Medical Association*, 103(3), 165-173. doi:10.7547/1030165.
- Owings, T.M., Apelqvist, J., Stenström, A., Becker, M., Bus, S.A., Kalpen, A., et al. (2009). Plantar pressures in diabetic patients with foot ulcers which have remained healed. *Diabetic Medicine*, 26, 1141-1146. doi:10.1111/j.1464-5491.2009.02835.x.
- Ozdemir, H., Yimaz, E., Murat, A., Karakurt, L., Poyraz, A., & Ogur, E. (2005). Sonographic evaluation of plantar fasciitis and relation to body mass index. *European Journal of Radiology*, 54, 443-447. doi:10.1016/j.erad.2004.09.004.
- Pai, S., & Ledoux, W.R. (2010). The compressive mechanical properties of diabetic and non-diabetic plantar soft tissue. *Journal of Biomechanics*, 43, 1754-1760. doi:10.1016/j.jbiomech.2010.02.021.
- Parkinson, J., Samiric, T., Ilic, M.Z., Cook, J., Handley, C.J. (2011). Involvement of proteoglycans in tendinopathy. *Journal of Musculoskeletal & Neuronal Interactions*, 11(2), 86-93.
- Patry, J., Belley, R., Côté, M., Chateau-Degat, ML. (2013). Plantar pressures, plantar forces, and their influence on the pathogenesis of diabetic foot ulcers. A Review. *Journal of the American Podiatric Medical Association*, 103(4), 322-332. doi:10.7547/1030322.

- Pavan, P.G., Stecco, C., Darwish, S., Natali, A.N., & De Caro, R. (2011). Investigation of the mechanical properties of the plantar aponeurosis. *Surgical and Radiologic Anatomy*, 33, 905-911. doi:10.1007/s00276-011-0873-z.
- Periyasamy, R., Anand, S., & Ammini, A.C. (2012). Prevalence of standing plantar pressure distribution variation in north Asian Indian patients with diabetes mellitus: A study to understand ulcer formation. *Journal of Engineering in Medicine*, 227(2), 181-189. doi:10.1177/095441.
- Rathleff, M., Moelgaard, C., & Olesen, J. (2011). Intra- and interobserver reliability of quantitative ultrasound measurement of the plantar fascia. *Journal of Clinical Ultrasound*, 39(3), 128-134.
- Redmond, A., Crosbie, J., & Ouvrier, R. (2006). Development and validation of a novel rating system for scoring standing foot posture: The foot posture index. *Clinical Biomechanics*, 21, 89-98. doi:10.1016/j.clinbiomech.2005.08.002.
- Rich, J., & Veves, A. (2000). Forefoot and rearfoot plantar pressures in diabetic patients: Correlation to foot ulceration. *Wounds*, 12(4), 82-87.
- Robertson, D.D., Mueller, M.J., Smith, K.E., Commean, P.K., Pilgram, T., Johnson, J.E. (2002). Structural changes in the forefoot of individuals with diabetes and a prior plantar ulcer. *Journal of Bone & Joint Surgery*, 84(8), 1395-1404.
- Rosenbloom, A.L., Malone, J.I., Yucha, J., Van Cader, T.C. (1984). Limited joint mobility and diabetic retinopathy demonstrated by fluorescein angiography. *European Journal of Pediatrics*, 141, 163-164.
- Sabir, N., Demirlenk, S., Yagci, B., Karabulut, N., & Cubukcu, S. (2005). Clinical utility of sonography in diagnosing plantar fasciitis. *Journal of Ultrasound Medicine*, 24, 1041-1048.
- Salathé, E.P. Jnr., Arangio, G.A., Salathé, E.P. (1986). A biomechanical model of the foot. *Journal of Biomechanics*, 19(12), 989-1001. doi: 10.1016/0021-9290(86)90116-8
- Scott, K.M., Marwick, J.C., & Crampton, P.R. (2003). Utilization of general practitioner services in New Zealand and its relationship with income, ethnicity and government subsidy. *Health Services Management Research*, 16, 45-55.
- Silverstein, J.H., Fennell, R., Donnelly, W., Banks, R., Stratton, R., Spillar, R...Rosenbloom, A.L. (1985). Correlates of biopsy-studied nephropathy in young patients with insulin-dependent diabetes mellitus. *Journal of Pediatrics*, 106, 196-201.
- Silvester, R., Williams, A., Dalbeth, N., & Rome, K. (2010). 'Choosing shoes': a preliminary study into the challenges facing clinicians in assessing footwear for rheumatoid patients. *Journal of Foot and Ankle Research*, 3(24), 1-8. doi:10.1186/1757-1146-3-24.
- Simmons, D., & Fleming, C. (2000). Prevalence and characteristics of diabetic participants with no ongoing care in South Auckland. *Diabetes Care*, 23, 1791-1793. doi:10.2337/diacare.23.12.1791.

- Singh, N., Armstrong, D.G., & Lipsky, B.A. (2005). Preventing foot ulcers in people with diabetes. *The Journal of the American Medical Association*, 293(2), 217-228. doi:10.1001/jama.293.2.217.
- Skriver, M.V., Støvring, H., Kristensen, J.K., Charles, M., & Sandbæk, A. (2012). Short-term impact of HbA1c on morbidity and all-cause mortality in people with type 2 diabetes: a Danish population-based observational study. *Diabetologia*, 55, 2361-2370. doi:10.1007/s00125-012-2614-1.
- Smieja, M., Hunt, D., Edelman, D., Etchells, E., Cornuz, J., & Simel, D. (1999). Clinical examination for the detection of protective sensation in the feet of diabetic patients. *Journal of Internal Medicine*, 14(7), 418-424. doi:10.1046/j.1525-1497.1999.05208.x.
- Smith, L.L., Burnet, S.P., & McNeil, J.D. (2003). Musculoskeletal manifestations of diabetes mellitus. *British Journal of Sports Medicine*, 37(1), 30-35. doi:10.1136/bjism.37.1.30.
- Shoji, T., Koyama, H., Morioka, T., Tanaka, S., Kizu, A., Motoyama, K., ... & Nishizawa, Y. (2006). Receptor for advanced glycation end products is involved in impaired angiogenic response in diabetes. *Diabetes*, 55(8), 2245-2255. doi: 10.2337/db05-1375.
- Solano, M.P., Prieto, L.M., Varon, J.C., Moreno, M., & Boulton, A. (2008). Ethnic differences in plantar pressures in diabetic patients with peripheral neuropathy. *Diabetic Medicine*, 25, 505-507. doi:10.1111/j.1464-5491.2008.02381.x.
- Stern, D., Yan, S.D., Yan, S.F., & Schmidt, A.M. (2002). Receptor for advanced glycation endproducts: a multiligand receptor magnifying cell stress in diverse pathologic settings. *Advanced Drug Delivery Reviews*, 54(12), 1615-1625. doi:10.1016/S0169-409X(02)00160-6.
- Sun, J., Cheng, B.K., Zheng, Y., Huang, Y., Leung, J.Y., & Cheing, G.L. (2011). Changes in the thickness and stiffness of plantar soft tissues in people with diabetic peripheral neuropathy. *Archives of Physical Medicine and Rehabilitation*, 92, 1484-1489. doi:10.1016/j.apmr.2011.03.015.
- Tentolouris, N., Pagoni, S., Tzonou, A., & Katsilambros, N. (2001). Peripheral neuropathy does not invariably coexist with autonomic neuropathy in diabetes mellitus. *European Journal of Internal Medicine*, 12(1), 20-27. doi:10.1016/S0953-6205(00)00128-X.
- Tesfaye, S., Stevens, J.K., Stephenson, J.M., Fuller, J.H., Plater, M., Ionescu-Tirgoviste, C., et al. (1996). Prevalence of diabetic peripheral neuropathy and its relation to glycaemic control and potential risk factors: The EURODIAB IDDM complications study. *Diabetologia*, 39(11), 1377-1384.
- Tesfaye, S. (2011). Recent advances in the management of diabetic distal symmetrical polyneuropathy. *Journal of Diabetes Investigation*, 2(2), 33-42. doi:10.1111/j.2040-1124.2010.00083.x.

- Uccioli, L., Faglia, E., Monticone, G., Favales, F., Durola, L., Aldeghi, A., et al. (1995). Manufactured shoes in the prevention of diabetic foot ulcers. *Diabetes Care*, 18(10), 1376-1378. doi:10.2337/diacare.18.10.1376.
- Udoh, B.E., Ezeokpo, B.C., Agwu, K.K., & Okpala, O.C. (2010). Effects of type two diabetes mellitus on the plantar aponeurosis of Nigerians. *World Journal of Medical Sciences*, 5(4), 81-84.
- Ulbrecht, J.S., Cavanagh, P.R., & Caputo, G.M. (2004). Foot problems in diabetes: An overview. *Clinical Infectious Diseases*, 39(Supplement 2), S73-S82. doi:10.1086/383266.
- van Schie, C., Carrington, A., Vermigli, C., & Boulton, A. (2004). Muscle weakness and foot deformities in diabetes. Relationship to neuropathy and foot ulceration in Caucasian diabetic men. *Diabetes Care*, 27(7), 1668-1673.
- van Schie, C.H. (2005). A review of the biomechanics of the diabetic foot. *The International Journal of Lower Extremity Wounds*, 4(3), 160-170. doi:10.1177/15347346605280587.
- van Schie, C.H.M., van der Linden, M.L., & Boulton, A.J.M. (2011). Foot pressures, peripheral neuropathy, and joint mobility in Asian and European patients with diabetes. *Wounds*, 23(7), 216-227.
- Veves, A., Murray, H.J., Young, M.J., & Boulton, A.J.M. (1992). The risk of foot ulceration in diabetic patients with high foot pressure: a prospective study. *Diabetologia*, 35(7), 660-663. doi:10.1007/BF00400259.
- Vileikyte, L. (2001). Diabetic foot ulcers: a quality of life issue. *Diabetes /Metabolism Research and Reviews*, 17(4), 246-249. doi:10.1002/dmrr.216.
- Vinik, A., Strotmeyer, E., Nakave, A., & Patel, C. (2008). Diabetic neuropathy in older adults. *Geriatric Medicine*, 24(3), 407-435. doi:10.1016/j.cger.2008.03.011.
- Vinik, A.I., Park, T.S., Stansberry, K.B., & Pittenger, G.L. (2000). Diabetic neuropathies. *Diabetologia*, 43, 957-973.
- Vinik, A.I., Maser, R.E., Mitchell, B.D., & Freeman, R. (2003). Diabetic autonomic neuropathy. *Diabetes Care*, 26, 1553-1579.
- Vohra, P.K., Kincaid, B.R., Japour, C.J., Sobel, E. (2002). Ultrasonographic Evaluation of Plantar Fascia Bands. *Journal of the American Podiatric Medical Association*, 92(8), 444-449. doi:10.7547/87507315-92-8-444.
- Waaijman, R., Bus, S.A. (2012). The interdependency of peak pressure and pressure-time integral in pressure studies on diabetic footwear: No need to report both parameters. *Gait & Posture*, 35, 1-5. doi: 10.1016/j.gaitpost.2011.07.006.
- Wang, Y., Lee, K., & Ledoux, W.R. (2011). Histomorphological evaluation of diabetic and non-diabetic plantar soft tissue. *Foot & Ankle International*, 32, 802-810. doi:10.3113/FAI.2011.0802.



- Watkins, P.J. (2003). The diabetic foot. *British Medical Journal*, 326(3), 977-979. doi:10.1136/bmj.326.7396.977.
- Wearing, S., Smeathers, J., Yates, B., Sullivan, P., Urry, S., & Dubois, P. (2004). Sagittal movement of the medial longitudinal arch is unchanged in plantar fasciitis. *Medicine & Science in Sports & Exercise*, 1761-1767. doi:10.1249/01MMSS.0000142297.10881.11.
- Wearing, S.C., Smeathers, J.E., Sullivan, P.M., Yates, B., Urry, S.R., & Dubois, P. (2007). Plantar fasciitis: are pain and fascial thickness associated with arch shape and loading? *Physical Therapy*, 87(8), 1002-1008.
- Wild, S., Roglic, G., Green, A., Sicree, R., & King, H. (2004). Global prevalence of diabetes: estimates for the year 2000 and projections for 2030. *Diabetes Care*, 27, 1047-1053. doi:10.2337/diacare.27.5.1047.
- Wrobel, J.S., Crews, R.T., Connolly, J.E. (2009). Clinical factors associated with a conservative gait pattern in older male veterans with diabetes. *Journal of Foot and Ankle Research*, 2, 11. doi:10.1186/1757-1146-2-11.
- Wrobel, J.S., Najafi, B. (2010). Diabetic foot biomechanics and gait dysfunction. *Journal of Diabetes Science Technology*, 4(4), 833-845. doi: 10.1177/193229681000400411.
- Wu, S.C., Driver, V.R., Wrobel, J.S., & Armstrong, D.G. (2007). Foot ulcers in the diabetic patient, prevention and treatment. *Vascular Health Risk Management*, 3(1), 65-76. doi:10.2147/VHRM.S.
- Yavuz, M., Tajaddini, A., Botek, G., & Davis, B.L. (2008). Temporal characteristics of plantar shear distribution: Relevance to diabetic patients. *Journal of Biomechanics*, 41, 556-559. doi:10.1016/j.biomech.2007.10.008.
- Young, M., Breddy, J., Veves, A., & Boulton, A. (1994). The prediction of diabetic neuropathic foot ulceration using vibration perception thresholds. A prospective study. *Diabetes Care*, 17(June), 557-560. doi:10.2337/diacare.17.6.557.
- Young, M.J., Boulton, A.J.M., Macleod, A.F., Williams, D.R.R., & Sonksen, P.H. (1993). A multicentre study of the prevalence of diabetic peripheral neuropathy in the United Kingdom hospital clinic population. *Diabetologia*, 36, 150-154.
- Zammit, G.V., Menz, H.B., & Munteanu, S.E. (2010). Reliability of the TekScan MatScan® system for the measurement of plantar forces and pressures during barefoot level walking in healthy adults. *Journal of Foot and Ankle Research*, 3, 11. doi:10.1186/1757-1146-3-11.
- Zimny, S., Schatz, H., & Pfohl, M. (2004). The role of limited joint mobility in diabetic patients with an at-risk foot. *Diabetes Care*, 27(4), 942-946. doi:10.233/diacare.27.4.942.
- Zou, D., Mueller, M.J., & Lott, D.J. (2007). Effect of peak pressure and pressure gradient on subsurface shear stresses in the neuropathic foot. *Journal of Biomechanics*, 40, 883-890. doi:10.1016/j.biomech.2006.03.005.

## **Glossary of Abbreviations**

AGE	Advanced glycation end products
BGL	Blood glucose level
BMI	Body mass index
DM	Diabetes mellitus
DSSN	Distal symmetrical sensorimotor neuropathy
FPI	Foot posture index
HbA1c	Glycated Hemoglobin
KPa	Mean peak plantar pressure
KPa/s	Pressure time integral
LJM	Limited joint mobility
MPJ	Metatarsophalangeal joint
mm	Millimetres
mmol/L	Millimoles per litre
PN	Peripheral neuropathy
T1DM	Type 1 Diabetes Mellitus
T2DM	Type 2 Diabetes Mellitus
US	Ultrasound Imaging
VPT	Vibration Perception Threshold

## Appendices

### Appendix A: Northern X Regional Ethics Committee



**Northern X Regional Ethics Committee**  
Private Bag 92522  
Wellesley Street  
Auckland 1141  
Phone: (09) 580 9105  
Fax: (09) 580 9001  
Email: northernx\_ethicscommittee@moh.govt.nz

19 September 2011

Professor Keith Rome c/- Ms Belinda Ihaka  
School of Podiatry, Dept of Rehabilitation and Occupation Studies,  
AUT University, Northshore Campus  
Akoranga Drive, Northcote  
Auckland 0627

Dear Keith

Re: Ethics ref: **NTX/11/09/082** (please quote in all correspondence)  
Study title: The evaluation of anatomical and biomechanical parameters of the feet in Maori with diabetes: a feasibility study  
Investigators: Professor Keith Rome (Principal), Ms Belinda Anne Ihaka, Associate Professor Wayne Hing  
Localities: Auckland University of Technology, Turuki Medical Centre

We thank Belinda Ihaka and yourself for attending the meeting when the Northern X Regional Ethics Committee considered your study on 13 September 2011.

#### Discussion

The Committee supports this worthwhile project; the changes required are minor. The Committee **approved** the above study **subject to the following conditions**

#### Application

- Please remove the word, “feasibility” but can say it is a small, underpowered study.
- Please be specific that it is a “comparative” study between Maori and non-Maori; put “comparison” into the title.
- Please ensure the protocol accurately describes the study.

#### Participant Information Sheet

- Please change wording on *koha* to “reimbursement of expense”.
- Please add “will consult/inform GP” in the Information Sheet.
- Please insert Deaf Interpreter box in Consent Form.
- Please insert the same version number and date for the Information Sheet and Consent Form, eg. version 1 and the current date.
- Please amend last paragraph in the Information Sheet to “This study has received ethical approval from the Northern X...”

You may **not** proceed with your study until ethical approval has been given. In order to obtain ethical approval from the Committee, please forward evidence that the above conditions have

been met, with the required documents and one copy of amended documentation (they can be sent via e-mail), including:

- amended pages only of the National Application Form (please highlight the changes).
- a full copy of the amended Information Sheet/Consent Form with **updated version number and date** (please highlight changes).

Provided the conditions above have been met, final approval for your study will be given by the Chairperson of the Committee. You will receive a letter advising you that final approval has been given, and may then proceed with your study.

Please don't hesitate to contact me for further information.

Yours sincerely



Cheh Chua  
Administrator  
Northern X Regional Ethics Committee

## Appendix B: AUTECH Ethics Approval



# MEMORANDUM

## Auckland University of Technology Ethics Committee (AUTECH)

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To: Keith Rome  
From: **Dr Rosemary Godbold** Executive Secretary, AUTECH  
Date: 7 October 2011  
Subject: Ethics Application Number 11/281 **A comparison between Māori and non-Māori with diabetes of the anatomical and biomechanical parameters of the foot.**

---

Dear Keith

Thank you for providing written evidence as requested. I am pleased to advise that it satisfies the point raised by a subcommittee of the Auckland University of Technology Ethics Committee (AUTECH) and I have approved your ethics application. This delegated approval is made in accordance with section 5.3.2.3 of AUTECH's *Applying for Ethics Approval: Guidelines and Procedures* and is subject to endorsement at AUTECH's meeting on 31 October 2011.

Your ethics application is approved for a period of three years until 7 October 2014.

I advise that as part of the ethics approval process, you are required to submit the following to AUTECH:

- A brief annual progress report using form EA2, which is available online through <http://www.aut.ac.nz/research/research-ethics/ethics>. When necessary this form may also be used to request an extension of the approval at least one month prior to its expiry on 7 October 2014;
- A brief report on the status of the project using form EA3, which is available online through <http://www.aut.ac.nz/research/research-ethics/ethics>. This report is to be submitted either when the approval expires on 7 October 2014 or on completion of the project, whichever comes sooner;

It is a condition of approval that AUTECH is notified of any adverse events or if the research does not commence. AUTECH approval needs to be sought for any alteration to the research, including any alteration of or addition to any documents that are provided to participants. You are reminded that, as applicant, you are responsible for ensuring that research undertaken under this approval occurs within the parameters outlined in the approved application.

Please note that AUTECH grants ethical approval only. If you require management approval from an institution or organisation for your research, then you will need to make the arrangements necessary to obtain this.

When communicating with us about this application, we ask that you use the application number and study title to enable us to provide you with prompt service. Should you have any further enquiries regarding this matter, you are welcome to contact me by email at [ethics@aut.ac.nz](mailto:ethics@aut.ac.nz) or by telephone on 921 9999 at extension 6902.

On behalf of AUTECH and myself, I wish you success with your research and look forward to reading about it in your reports.

Yours sincerely

Dr Rosemary Godbold  
**Executive Secretary**  
**Auckland University of Technology Ethics Committee**

Cc: Belinda Ihaka belinda.ihaka@aut.ac.nz, Wayne Hing

## Appendix C: Participant Information Form

Professor Keith Rome  
Department of Podiatry  
Faculty of Rehabilitation & Occupation Studies  
Telephone: 64 9 921 9999 extn 7688  
Facsimile: 64 9 921 9839 Email: krome@aut.ac.nz

September, 2011

### Participant Information Sheet

## **A comparison between Māori and non-Māori with diabetes of the anatomical and biomechanical parameters of the foot**

### **An Invitation**

You are invited to take part in a research study. Before you decide, it is important for you to understand why the research is being done and what it will involve. This is part of a Master's study being undertaken at AUT University by Belinda Ihaka (researcher).

### **What is the purpose of this research?**

We are interested in finding out why foot ulcerations occur in New Zealand Māori people with diabetes than in other populations. There has been a lot of work to suggest that loss of sensation is a major contributing factor to foot ulcerations, however, there are very few research studies examining why Māori are disproportionately affected by foot ulcerations. Because of this problem, I would like to look at your feet.

### **What will happen in this research?**

I would like to ask you questions about your blood glucose levels, how many years you have had diabetes (if present); height, weight and what shoes you currently wear. I will also take measurements of your feet and then check your feet to make sure you have no areas of hard skin. If you do, you will be asked if these areas could be removed by me, so that I can check your foot's sensation. I will then ask you to lie on the clinic bed, and I will take 3 readings of the sole of each foot using a portable ultrasound machine. There are no risks associated with ultrasound measurements. Then you will be asked to walk along a portable walkway to determine how much force is being applied under your feet. Three walking trials will occur in order for you to familiarise yourself with the equipment.

Your participation is entirely voluntary (your choice). If you change your mind about participating after your study visit, you can withdraw your data up to one month after the study visit without giving a reason. If you wish to withdraw your data, please contact Professor Rome (contact details above). You do not have to take part in this research study, and if you decide not to take part or withdraw from the study, this will not affect your normal medical care in any way. The assessment will take about 45 minutes to complete. We will consult/inform your GP about the study

**What are the benefits?**

The research team will be happy to give you information about the progress of the project and will be happy to send you a final report. I will keep you informed of the results of the study. Please note that there may be a delay between your study visit and when the results are made public.

**What are the costs of participating in this research?**

No payments are being made to any doctors or researchers for including patients in this study.

**Will I receive feedback on the results of this research?**

The research team plan to publish results from this study in scientific journals so that the information is freely available to other doctors, scientists and the public. Patients will not be identified in any report or publication and indeed all information about your identity will be kept strictly confidential. Although no names will be used in the publication, age, sex, ethnicity, foot type and diagnosis will be reported.

**What compensation is available for injury or negligence?**

In the unlikely event of a physical injury as a result of your participation in this study, you may be covered by ACC under the Injury Prevention, Rehabilitation and Compensation Act. ACC cover is not automatic and your case will need to be assessed by ACC according to the provisions of the 2002 Injury Prevention Rehabilitation and Compensation Act. If your claim is accepted by ACC, you still might not get any compensation. This depends on a number of factors such as whether you are an earner or non-earner. ACC usually provides only partial reimbursement of costs and expenses and there may be no lump sum compensation payable. There is no cover for mental injury unless it is a result of physical injury. If you have ACC cover, generally this will affect your right to sue the investigators.

**What do I do if I have concerns about this research?**

If you have any questions or medical problems during this study, you should call the study doctor Professor Keith Rome who is in charge of this research or one of the study staff. The study doctor or study staff will also answer any questions you have about this research study or your participation in the study. You have the right to ask questions about this study at any time.

Study Doctor: Professor Keith Rome  
Telephone Number: 921 9999 extension 7688

Researcher: Belinda Ihaka  
Telephone Number: 921 9015

If you have any queries or concerns regarding your rights as a participant in this study you may wish to contact an independent Health and Disability Advocate

Telephone: 0800 555 050

Free Fax: 0800 2787 7678 (0800 2 SUPPORT)



Email:      advocacy@hdc.org.nz

**How do I agree to participate in this research?**

If you choose to help us with our research we ask you to sign a consent form to show that you agree to the above. Thank you for reading this.

This study has received ethical approval from the Northern X Regional Ethics Committee.

## Appendix D: Consent Form

Professor Keith Rome  
Department of Podiatry  
School of Rehabilitation & Occupation Studies  
Telephone: 64 9 921 9999 extn 7688  
Facsimile: 64 9 921 9839  
Email: krome@aut.ac.nz

September, 2011

### Participant Consent Form

#### A comparison between Māori and non-Māori with diabetes of the anatomical and biomechanical parameters of the foot

English	I wish to have an interpreter.	Yes	No
Deaf	I wish to have a NZ sign language interpreter	Yes	No
Māori	E hiahia ana ahau ki tetahi kaiwhakaMāori/kaiwhaka pakeha korero.	Ae	Kao

I have read the study information sheet (Version 2 September, 2011) for people taking part in the study to understand foot health problems in diabetes

The study has been explained to me by: Prof/Miss\_\_\_\_\_

I have been given the opportunity to ask questions and discuss this study with the investigator and my whanau/family, and I have received satisfactory answers to all my questions. **YES/NO**

I have received enough information about the study and have had enough time to think about it.

**YES/NO**

I understand that taking part in this study is voluntary (my choice). If I change my mind after I have attended a study visit, I understand that I can withdraw my data up to one month after this visit, without having to give a reason for withdrawing and without affecting my future medical care.

**YES/NO**

I know who to contact if I have any questions about the study. **YES/NO**

I understand that my participation in this study is confidential and that no material which could identify me will be used in any reports on this study. **YES/NO**

I agree to take part in this study. **YES/NO**

I wish to receive a copy of the results. **YES/NO**

Signed.....Date.....

(NAME IN BLOCK CAPITALS).....

Investigator's signature.....Date: .....  
(NAME AND ROLE IN BLOCK CAPITALS).....

## Appendix E: Post Hoc Test of Foot Characteristics

Non-Māori with and with no diabetes demonstrated larger foot size and arch length compared to the other groups overall as demonstrated below. When the data is combined (i.e. left and right foot) all groups demonstrated a narrow foot width.

Group				
Variable	Non-Māori no DM Mean (SD)	Māori no DM Mean (SD)	Non-Māori with DM Mean (SD)	Māori with DM Mean (SD)
Left foot width	1.1 (0.3)	1.3 (0.4)	2.0 (0.8)	1.3 (0.4)
Right foot width	1.2 (0.4)	1.2 (0.4)	1.7 (0.7)	1.9 (2.0)
Left foot length	8.7 (2.6)	9.8 (1.3)	10.7 (2.5)	9.3 (1.5)
Right foot length	8.7 (2.7)	9.7 (1.4)	11.35 (2.4)	8.7 (8.7)
Left foot arch length	9.4 (2.8)	10.0 (2.4)	10.8 (3.0)	9.9 (1.8)
Right foot arch length	9.1 (2.7)	9.8 (2.0)	11.3 (3.7)	9.5 (2.3)

**DM = Diabetes.**