



## Prevalence of undiagnosed diabetes, impaired glucose tolerance, and impaired fasting glucose among Māori in Te Wai o Rona: Diabetes Prevention Strategy

David Simmons, Elaine Rush, Nic Crook

### Abstract

**Aims** To describe the prevalence of undiagnosed diabetes, impaired glucose tolerance (IGT) and impaired fasting glucose (IFG) (“dysglycaemia”) among Māori.

**Methods** Te Wai o Rona: Diabetes Prevention Strategy was a trial of lifestyle change among Māori families in the Waikato/Lakes areas of New Zealand. All Māori family household members aged  $\geq 28$  years, without known diabetes, were invited to participate through primary care, community, and media approaches. Participants were invited to have an oral glucose tolerance test (OGTT).

**Results** Of the 3817 eligible Māori, mean BMI was  $32.9 \pm 7.8$  kg/m<sup>2</sup> (women) and  $33.1 \pm 6.7$  kg/m<sup>2</sup> (men). The age standardised prevalence of undiagnosed diabetes was higher among men than women (6.5[5.8–7.4]% vs 4.2[3.6–4.8]%), as was that for IFG (5.4[4.7–6.1]% vs 3.0[2.3–3.5]%), but not IGT (8.5[7.6–9.4]% vs 9.7[8.7–10.6]%) with no rural-urban differences. The prevalence of dysglycaemia increased with increasing BMI with no clear inflection point and was 1.33(1.11–1.60) greater among those with a community services card after adjusting for age, sex and BMI.

**Conclusions** Undiagnosed diabetes, IGT, and IFG remain common among Māori, particularly men, the very obese, and those with greater socioeconomic disadvantage. There remains significant opportunity to reduce Māori morbidity and premature mortality through diabetes case-finding and intervention.

The management of known Type 2 diabetes and its complications accounts for a significant proportion of health expenditure in New Zealand.<sup>1</sup> However, much of diabetes remains undiagnosed<sup>2</sup> and levels of hyperglycaemia below the diagnostic threshold for diabetes (impaired glucose tolerance [IGT] and impaired fasting glucose [IFG]) are also associated with an excess risk of cardiovascular disease.<sup>3</sup> Such levels of hyperglycaemia are also at high risk of progressing to Type 2 diabetes, progression now known to be preventable by intensive lifestyle change and some medications.<sup>4–6</sup>

The potential benefits of detecting any abnormal glucose tolerance vary between populations depending on risk of developing diabetes and risk of complications once diabetes has been diagnosed. Calculating the costs and benefits of intervening at an early stage require detailed information including the proportion with undiagnosed diabetes, IGT and IFG.

Māori have higher rates of diabetes than European New Zealanders and disproportionately higher rates of many of the complications caused by diabetes.<sup>2</sup> As such, a successful diabetes prevention strategy among Māori is crucial for New Zealand and Māori alike.

Te Wai o Rona: Diabetes Prevention Strategy was a 4-year randomised controlled trial among Māori communities in the Waikato and Lakes District Health Board areas,<sup>7</sup> registered with the Australasian Controlled Trials Registry (ACTRN012605000622606). The trial did not proceed after the first 3 years for funding reasons. However, the baseline data, being population based may be able to be used to provide the needed detailed information regarding the proportion with undiagnosed diabetes, IGT, and IFG and we now present these data and discuss the caveats behind the data.

## Methods

**Participants**—All Māori resident within the boundaries of the Waikato DHB, and the tribal area of Ngati TuWharetoa in the neighbouring Lakes DHB were invited to participate with their families. The age cutoff for entry was taken as  $\geq 28$  years on 30 September 2005. Recruitment was by personal invitation from local general practitioners in association with media releases (television, radio, posters, newspapers) announcing times/venues of screening, workplace screening, and personal contact.

Personal contact through different health organisations and their staff, and announcements at a number of Māori community activities became increasingly important through the recruitment phase. Those who attended were asked to inform other family members and friends.

Participants were asked to attend after a 10-hour overnight fast and were advised that breakfast would be provided. Advice was also given that attending non-fasting was also acceptable although full testing (including OGTT) would not then be performed. Those unfit to sign a consent form, with terminal disease, or not permanently residing in the study area at the time of the baseline data collection were excluded.

Ethical approval was provided by both the Waikato and Bay of Plenty Ethics Committees. All participants gave signed informed consent.

Screening sessions were held 0700–1400 hours in a variety of community venues across the study area. Where possible, transport was provided for participants. After registration/consent and ascertainment of fasting status, fasting participants had a finger-prick glucose (venous plasma equivalent) using a glucose meter (Advantage, Roche, Switzerland) and venesection. Those who were non-fasting had a single venesection for glucose and HbA1c (Bio-rad Diamat Variant, [upper limit of reference range 6.4%], Bio-Rad Laboratories, USA).

Samples for HbA1c were sent to the same laboratory for analysis. Glucose samples were centrifuged, separated, and refrigerated within 30 minutes on site in a mobile laboratory and subsequently measured using the Hitachi 911 (Hitachi Limited, Tokyo, Japan). All assays were within target limits specified by the RCPA Quality Assurance Program. These assays were carried out by the Waikato District Health Board Laboratory which has IANZ ISO9002 Accreditation.

Fasting participants with a fingerprick glucose  $\geq 4.4$  mmol/L were advised to undertake a 75g 2 hour oral glucose tolerance test (OGTT), although those with values below this were also invited to have an OGTT.

Trained staff facilitated standard questionnaire and measurement completion. Questionnaires including demographic data were completed. Ethnicity was determined by self identity. During the OGTT, other measurements included height $\pm 0.5$  cm (portable height scale PE087; Mentone Education Centre, Victoria, Australia) and weight $\pm 0.1$  kg (Wedderburn TI-TH316 Personal scales or Wedderburn TI-BWB800 Personal scales [up to 200kg] for oversize participants).

Based upon previous research including Māori,<sup>8</sup> if no OGTT had been completed and the fasting glucose was  $\geq 5.3$  mmol/L, or a random glucose  $\geq 5.3$  mmol/L or the HbA1c  $\geq 5.3\%$ , participants were asked to subsequently attend the local community laboratory for an OGTT (screen positive subjects).

Screen-negative subjects were defined as those with HbA1c, fasting, and random glucose results below these criteria. Diabetes, IFG, and IGT were diagnosed using 1998 World Health Organization criteria.<sup>9</sup> If no OGTT was undertaken and the fasting glucose was  $\geq 7.0$  mmol/L and/or the random glucose was  $\geq 11.1$  mmol/L, diabetes was considered to be present.

**Statistics**—The overall prevalence of diabetes (among those aged  $\geq 30$  years) was calculated by direct age standardization to the 2006 Census.<sup>10</sup> Comparisons are made by either Chi-squared test or by

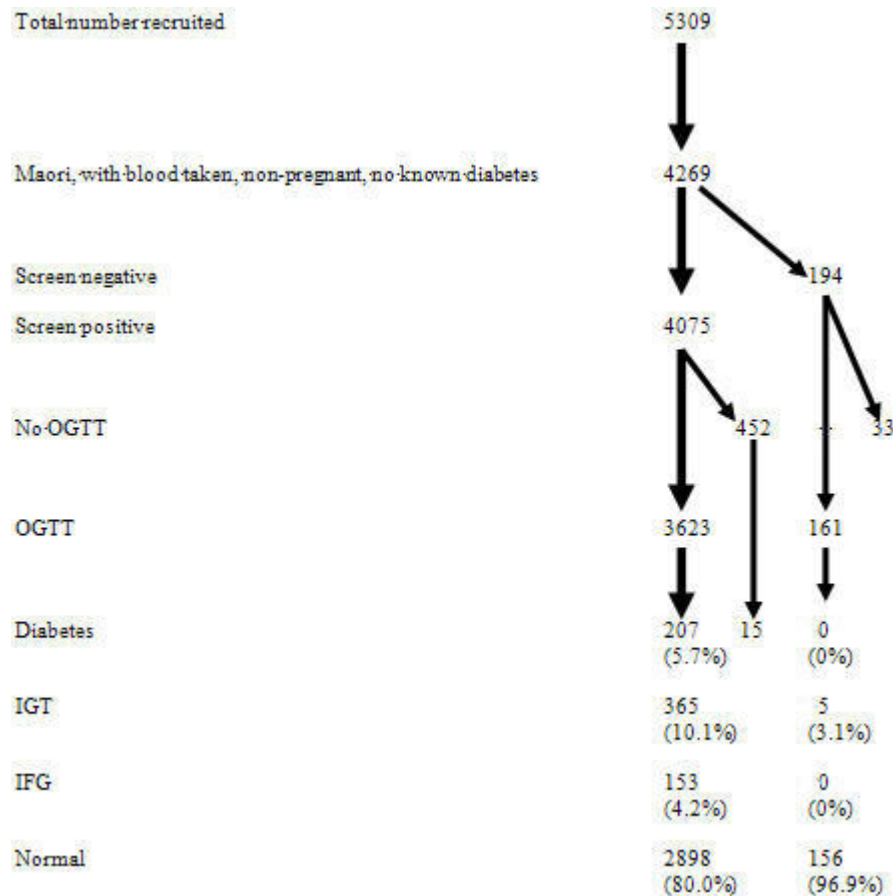
comparing 95% confidence intervals. Tests are 2-tailed with  $p < 0.05$  taken as significant. Mantell Haenszel test was used to compare the prevalence of dysglycaemia by community services card use, after adjustment for age and gender.

## Results

Of the 5059 non-pregnant adults screened for diabetes, 4269 were Māori aged  $\geq 28$  years (approximately 13% of the comparably aged Māori population in the recruitment area).<sup>10</sup>

Figure 1 shows the attendance at OGTT ( $n=3784$ ) including among those with a negative screen. Among screened Māori, 2726 (63.9%) were women, the mean age was  $48 \pm 12$  years, 67.4% were rural residents, and 65.9% were known to have a family member with diabetes. Very few (3.7%) of those with a negative screen had IGT and none had diabetes or IFG.

**Figure 1. Response to screening and crude proportions with diabetes, impaired glucose tolerance (IGT), and impaired fasting glucose (IFG)**



Among men, 86.7% and 79.2% of those above and below the criteria for OGTT respectively had an OGTT, the proportions being 85.2% and 89.9% respectively among women. Among those not attending OGTT and screen-positive (above the

threshold for OGTT), 15 were considered to have diabetes on the basis of their screening test.

Among the screen-positive participants, the fasting blood glucose (n=109), random blood glucose (n=338), and HbA1c were similar between those attending and not attending OGTT (5.3±0.9 vs 5.5±2.1 mmol/L; 5.5±1.0 vs 5.6±1.8 mmol/L; 6.0±0.6 vs 6.0±1.0% respectively). As the proportion with a “negative screen” was small (4.5%), and most of these had an OGTT (among whom few had IGT and none with diabetes or IFG), analyses assume that the negative screen group were all normal and that those not having an OGTT were comparable to the overall cohort. Age-specific prevalence is therefore calculated from those who had an OGTT or who were screen negative without an OGTT (n=33).

Table 1 shows that the prevalence of diabetes increased with age, although no Māori aged over 80 years was found to have undiagnosed diabetes. The age standardised prevalence of undiagnosed diabetes was higher among men than women (6.5[5.8–7.4]% vs 4.2[3.6–4.8]%), as was that for IFG (5.4[4.7–6.1]% vs 3.0[2.3–3.5]%), but not IGT (8.5[7.6–9.4]% vs 9.7[8.7–10.6]%).

**Table 1. Response to screening and estimated prevalence of undiagnosed diabetes, IGT, and IFG among Māori**

<b>Females (age groups)</b>	<b>28–29</b>	<b>30–39</b>	<b>40–49</b>	<b>50–59</b>	<b>60–69</b>	<b>70–79</b>	<b>80+</b>
N	93	630	737	537	328	128	15
Diabetes	2.2%	2.4%	3.8%	5.0%	8.5%	7.0%	0%
IGT	5.4%	6.5%	7.9%	10.6%	15.9%	14.8%	53.3%
IFG	1.1%	1.3%	2.4%	4.8%	6.7%	3.1%	0%
<b>Males (age groups)</b>	<b>28–29</b>	<b>30–39</b>	<b>40–49</b>	<b>50–59</b>	<b>60–69</b>	<b>70–79</b>	<b>80+</b>
N	44	301	413	286	206	89	10
Diabetes	2.3%	2.3%	7.7%	8.4%	11.2%	12.4%	0%
IGT	2.3%	4.3%	5.8%	12.6%	17.5%	14.6%	20.0%
IFG	0%	3.3%	7.7%	5.6%	4.9%	6.7%	0%
Female vs male	ns	ns	<0.001	ns	ns	ns	ns

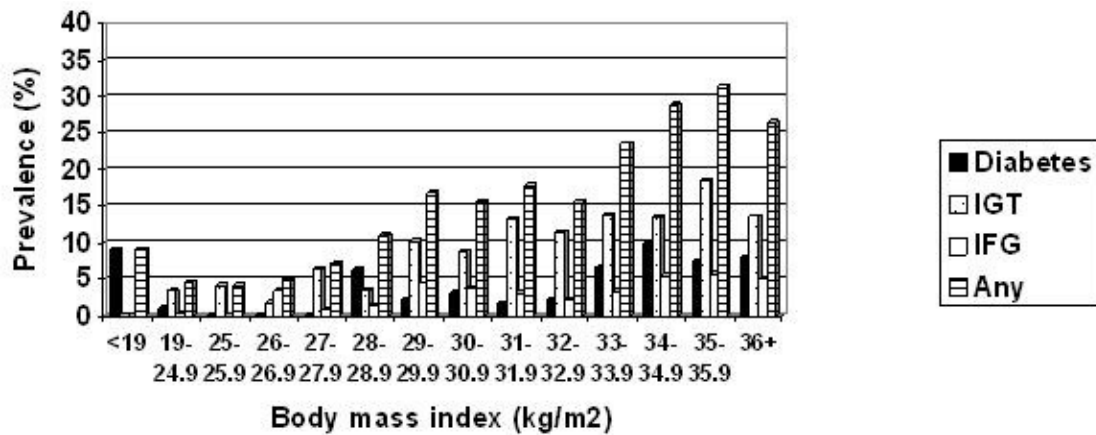
Those who were screen negative were <15.0% in all age-sex groups.

There was no significant difference in prevalence of diabetes, IGT, or IFG between those living in rural and urban areas, nor between those living in different rural tribal areas. Among those classified as having diabetes, 12.8% had a fasting glucose <6.1 mmol/L and 8.2% had a fasting glucose <5.6 mmol/L. Among those with IGT, 76.2% had a fasting glucose <6.1 mmol/L and 46.7% had a fasting glucose <5.6 mmol/L.

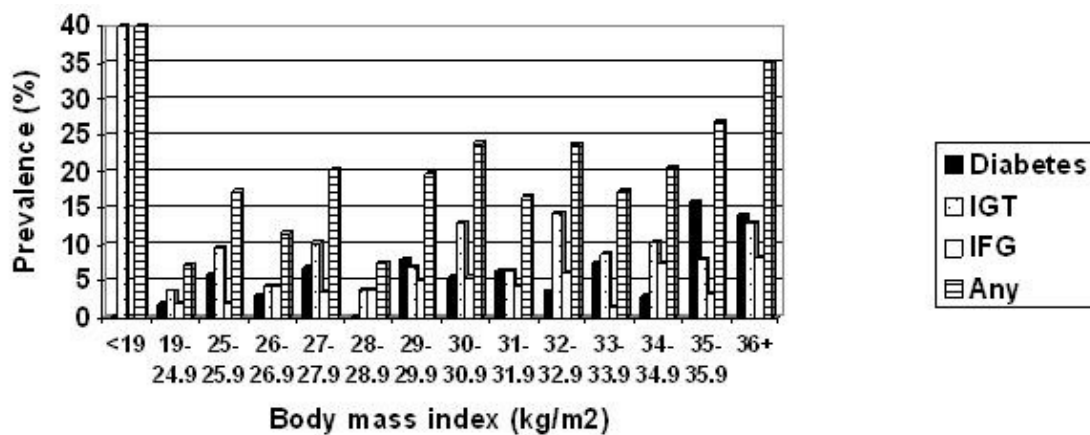
Mean BMI among women was 32.9±7.8 kg/m<sup>2</sup> and among men was 33.1±6.7 kg/m<sup>2</sup>. The prevalence of diabetes, IGT, and IFG are shown by body mass index (BMI) group in Figure 2 (women) and Figure 3 (men). The BMI groups were defined by 1 kg/m<sup>2</sup> across the overweight and obese ranges (25–36 kg/m<sup>2</sup>).

There was no significant age difference between the BMI groups. Prevalence of dysglycaemia increased with increasing BMI with no clear inflection point. Among the 29.4% of women and 28.4% of men with a BMI ≥36 kg/m<sup>2</sup>, over one-third had some degree of dysglycaemia, particularly IGT.

**Figure 2. Prevalence of diabetes, IGT, and IFG by body mass index (women)**



**Figure 3. Prevalence of diabetes, IGT, and IFG by body mass index (men)**



**Note:** The 1 subject with a BMI <19 kg/m<sup>2</sup> had IGT.

The prevalence of dysglycaemia was 1.38(1.16–1.65) fold greater among those with a community services card after age and gender and 1.33(1.11–1.60) fold greater after additionally adjusting for BMI. Undiagnosed diabetes alone was 1.24(0.91–1.69) fold higher among those with a community services card (i.e. non significant).

## Discussion

This is one of several studies reporting the prevalence of undiagnosed diabetes, IGT, and IFG among Māori in different parts of New Zealand.<sup>2</sup> The prevalence of undiagnosed diabetes was higher than in the Diabetes Heart and Health Study<sup>11</sup> and comparable to that in the Ngati and Healthy Project.<sup>12</sup>

Abnormal glucose tolerance, particularly undiagnosed diabetes, was particularly high among Māori men and the very obese. As diabetes, IGT, and IFG remain harbingers

of significant cardiovascular disease,<sup>3</sup> and lifestyle and pharmacological intervention can significantly reduce the risk of future morbidity and mortality, we need to find ways to identify such dysglycaemia as early as possible.

Those identified as having diabetes in this study were followed up for microvascular disease (retinopathy and nephropathy) and while retinopathy was prevalent in very few, microalbuminuria and albuminuria were present in 29.6% and 7.7% respectively.<sup>7</sup>

We have previously shown that microalbuminuria is often present before diagnosis of diabetes in Māori and that it is more related to the familial risk of renal disease than diabetes.<sup>13</sup> The low prevalence of retinopathy was heartening and is a measure of the duration that individuals remained undiagnosed.<sup>14</sup>

The prevalence of known diabetes in the area is unknown, and the low rate of retinopathy at diagnosis might suggest a greater uptake of screening in this district. A combined diabetes specialist clinic and retinal screening register has estimated the prevalence of known diabetes among Waikato Māori to be 1.4% (30–39 years), 4.0% (40–49 years), 10.9% (50–59 years), and 17–20% (60+ years).<sup>15</sup>

These data exclude some important patients (e.g. those attending eye clinic but not the diabetes specialist clinic) and are not gender-specific, but suggest that (perhaps) over 50% of diabetes is undiagnosed in those 30–49 years, but that 25–40% are undiagnosed over this age.

In view of the risk from undiagnosed Type 2 diabetes in pregnancy among Māori women,<sup>16</sup> and the risk of renal disease and other long-term complications from uncontrolled diabetes among Māori,<sup>2</sup> diabetes screening and diagnostic strategies among Māori under 50 years warrants greater attention.

With such a high prevalence of undiagnosed diabetes shown in these analyses, and the excess risk of cardiovascular disease associated with dysglycaemia, there is clearly scope to increase identification of Māori with any form of dysglycaemia, who would benefit from intervention.

Whether this would help address the higher mortality rates among Māori, particularly Māori men<sup>17</sup>, is yet to be seen. This difference is known to be associated with greater deprivation, and in our study, use of a community services card, accessible only to those on reduced incomes, was associated with a greater prevalence of dysglycaemia. The lack of a significant association with new diabetes was possibly due to insufficient statistical power.

Of interest was the lack of difference in prevalence between rural and urban Māori (as well as between tribal areas), suggesting a balance between access to diabetes screening between rural and urban Māori and actual diabetes/dysglycaemia risk.

We have carefully looked at the prevalence of IGT, IFG, and undiagnosed diabetes in relation to obesity as measured by BMI. Naturally, prevalence increased with increasing BMI, but there was no natural inflection point to assist with defining obesity within Māori.

A range of studies have recommended that Māori have different criteria for obesity using BMI in view of their lower fat content at a given BMI.<sup>18</sup> Clearly what is of



importance in defining a cut off for risk is relating the cutoff to a hard end point such as diabetes or CVD. While such analyses should preferably be prospective using a baseline BMI, in reality, BMI changes over time and a greater weight gain is associated with greater diabetes risk.<sup>19</sup>

In our study, using cross-sectional data, there is no BMI between 25 and 36 which would help define overweight or obesity. These data do need to consider penetration of screening, which may have been greater among more obese Māori.

There are a range of caveats to interpreting these data. These are recruits into a trial of lifestyle change to prevent diabetes. We are aware that some participants attended to be screened for diabetes (i.e. the trial may have attracted those who were symptomatic). Conversely, those attending for a lifestyle trial are possibly more likely to lead a healthier life and to have started making healthier food and physical activity choices, which would impact on risk of dysglycaemia.

Notwithstanding this self selection, the vast majority were obese and hence this is unlikely to have had a major impact on the nature of the cohort. The cohort represents approximately 25% of Māori women and <15% of Māori men in the area, again suggesting that caution should be used in extrapolating these findings to the wider local or national Māori population.

One of the major strengths of the cohort is the high proportion who underwent OGTT without reliance on a fasting test alone. The criteria used to avoid OGTT were largely ignored by participants, resulting in a high proportion of screen negative individuals having an OGTT. This was important, with the high proportion of Māori with diabetes and IGT with a lower fasting glucose. These data are important to inform future screening campaigns and emphasising the importance of the OGTT in identifying those with undiagnosed dysglycaemia who could benefit from intervention.

In conclusion, we have shown that undiagnosed diabetes, IGT, and IFG remain common in this Māori cohort (particularly in men and the very obese) and that there remains significant opportunity to reduce Māori morbidity and premature mortality through case-finding and intervention.

**Competing interests:** None known.

**Author information:** David Simmons, Lead Community Diabetologist, Institute of Metabolic Science, Cambridge University Hospitals NHS Foundation Trust, Cambridge, England; Elaine Rush, Professor of Nutrition, Centre for Physical Activity and Nutrition Research, AUT University, Auckland, New Zealand; Nic Crook, Diabetes Consultant, Lakes District Health Board, Rotorua, New Zealand

**Funding and acknowledgements:** Funding was provided by Health Research Council, Waikato District Health Board, Lakes District Health Board, Ministry of Health, Sport and Recreation New Zealand, Southern Trust, Waikato Local Diabetes Team, and Merck Sharp and Dohme. Support in kind was provided by Roche Diagnostics, Pathlab, Medlab, University of Auckland, Auckland University of Technology, Wintec, Te Hotu Manawa Māori, Eggs Inc, Vodafone, Rivermill Bakers, and Sun Fruit. We thank the investigator group, Kaitiaki, Māori Community Health Workers, Te Wai o Rona: Diabetes Prevention Strategy Project team, and local health

service staff for their varied contributions to the study. DS also thanks NIHR Cambridge Biomedical Research Centre for its support.

**Correspondence:** Professor David Simmons, Institute of Metabolic Science, Cambridge University Hospitals NHS Foundation Trust, Cambridge, UK CB2 2QQ. Fax: +44 (0)1223 217080; Email: [dsworkster@gmail.com](mailto:dsworkster@gmail.com)

## References:

1. Diabetes New Zealand. Type 2 diabetes: Managing for better health outcomes. Price Waterhouse Cooper Economic Report for Diabetes New Zealand Inc; 2001. <http://www.diabetes.org.nz/resources/pwcreport.html>
2. Joshy G, Simmons D. The epidemiology of diabetes in New Zealand: Revisit to a changing landscape. *N Z Med J.* 2006;119(1235). <http://www.nzma.org.nz/journal/119-1235/1999>
3. Unwin N, Shaw J, Zimmet P, Alberti KG. Impaired glucose tolerance and impaired fasting glycaemia: the current status on definition and intervention. *Diabetic Med.* 2002;19:708–23.
4. Tuomilehto J, Lindstrom J, Eriksson JG, et al. Prevention of type 2 diabetes mellitus by changes in lifestyle among subjects with impaired glucose tolerance. *N Engl J Med.* 2001;344:1343–50.
5. Ramachandran A, Snehalatha C, Mary S, et al. The Indian Diabetes Prevention Programme shows that lifestyle modification and metformin prevent type 2 diabetes in Asian Indian subjects with impaired glucose tolerance (IDPP-1). *Diabetologia.* 2006;49:289–97.
6. Knowler WC, Barrett-Connor E, Fowler SE, et al. Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. *N Engl J Med.* 2002;346:393–403.
7. Lim S, Chellumuthi C, Crook N, Rush E, Simmons D. Low prevalence of retinopathy, but high prevalence of nephropathy among Maori with newly diagnosed diabetes: Te Wai o Rona: Diabetes Prevention Strategy. *Diab Res Clin Prac.* 2008;80:271–4.
8. Simmons D, Thompson CF, Engelgau MM. Controlling the diabetes epidemic: how should we screen for undiagnosed diabetes and dysglycaemia? *Diabet Med.* 2005;22:207–12.
9. World Health Organization, International Diabetes Federation. Definition and diagnosis of diabetes mellitus and intermediate hyperglycemia – report of a WHO/IDF consultation. Geneva, Switzerland: World Health Organization; 2006
10. Statistics New Zealand. New Zealand Census. Wellington: Statistics New Zealand; 2006.
11. Sundborn G, Metcalf P, Scragg R, et al. Ethnic differences in the prevalence of new and known diabetes mellitus, impaired glucose tolerance and impaired fasting glucose. Diabetes Heart and Health Survey (DHAH) s002-2003, Auckland, New Zealand. *N Z Med J.* 2007;120(1257). <http://www.nzma.org.nz/journal/120-1257/2607/>
12. Tipene-Leach D, Pahau H, Joseph N, et al. Insulin resistance in a rural Maori community. *N Z Med J.* 2004;117(1207). <http://www.nzma.org.nz/journal/117-1207/1208>
13. Thompson C, Simmons D, Collins JF, Cecil A. Predisposition to nephropathy in Polynesians is associated with family history of renal disease, not diabetes mellitus. *Diabet Med.* 2001;18:40–6.
14. Harris MI, Klein R, Welbourn TA, Knudman MW. Onset of NIDDM occurs at least 4-7 years before clinical diagnosis. *Diab Care.* 1992;15:815–9.
15. Joshy G, Lawrenson R, Dunn P. Diabetes patients in Waikato & their hospital admissions (Proceedings of the Waikato Clinical School Research Seminar on 15th March 2007). *N Z Med J.* 2007;120(1257). <http://www.nzma.org.nz/journal/120-1257/2625/>
16. Simmons D, Thompson CF, Conroy C. Incidence and risk factors for neonatal hypoglycaemia among women with gestational diabetes mellitus in South Auckland. *Diabet Med.* 2000;17:830–4.
17. Bramley D, Hebert P, Tuzzio L, Chassin M. Disparities in Indigenous Health: A cross country comparison between New Zealand and the United States. *Am J Public Health.* 2005;95:844–50.



18. Rush EC, Goedecke JH, Jennings C, et al. BMI, fat and muscle differences in urban women of five ethnicities from two countries. *Int J Obes Relat Metab Disord.* 2007;31:1232–9.
19. Will JC, Williamson DF, Ford ES, et al. Intentional weight loss and 13 year diabetes incidence in overweight adults. *Am J Pub Health.* 2002;92:1245-8.  
<http://www.ajph.org/cgi/content/full/92/8/1245>