



Insulin resistance in a rural Maori community

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Abstract

Aim To determine the prevalence of insulin resistance, impaired fasting glycaemia, impaired glucose tolerance, and diabetes mellitus in a rural Maori community, and to compare different methods for identifying individuals with insulin resistance.

Methods 589 randomly selected individuals from the Ngati Porou Hauora Register aged 25 years and over and resident on New Zealand's East Coast north of Gisborne were invited to participate in the study. A questionnaire was administered, anthropometric measures made, and blood samples taken for an oral glucose tolerance test and biochemical analysis. Impaired fasting glycaemia, impaired glucose tolerance, and diabetes mellitus were defined according to World Health Organization (WHO) diagnostic criteria, and among those persons with normal glucose tolerance, insulin resistance was calculated according to the McAuley formula and three other recognised methods for calculating insulin sensitivity.

Results The overall age-standardised prevalence of diabetes (both known and newly diagnosed) was 10.6% and the age-standardised prevalence of insulin resistance was 37.0%. Age-specific diabetes rates were high among the older age groups, peaking at 34.1% for 60–69 year olds, whereas age-specific insulin resistance rates were high among the young age groups with the highest rate (44.3%) occurring among 30–39 year olds. Persons identifying as insulin-resistant reported higher rates of gout and family history of diabetes—and were found to have a higher waist circumference, blood pressure, and lower high-density lipoprotein (HDL) cholesterol than those without a glucose metabolism disorder.

Conclusion Diabetes is a common disorder among this population, but insulin resistance is even more prevalent, especially among young age groups. This is considerable cause for concern given that insulin resistance is believed to be the underlying cause of most cases of type 2 diabetes mellitus, and is confirmed by these data to be associated with a high degree of cardiovascular risk.

The prevalence of diabetes is increasing worldwide.¹ In New Zealand, only limited prevalence data are available, but evidence suggests that this increase is also occurring.^{2–9} The most recent New Zealand Health Survey (NZHS) found the prevalence of self-reported diabetes for people aged over 45 years to be 8.1% for females and 10.0% for males.⁶ Prevalence surveys have consistently shown diabetes (both known and newly diagnosed) to be more common among Maori compared with New Zealanders of European descent. Most recently, in the NZHS self-reported diabetes prevalence among Maori aged over 45 years was 21.4% and 13.0% for males and females, respectively—compared with 8.6% and 7.5% for non-Maori males and females, respectively.⁶

Previously, Simmons et al⁸ found the prevalence of known diabetes mellitus in South Auckland was 6.9% among Maori compared with 2.8% among Europeans—and in the New Zealand Multiracial Workforce Survey, the prevalence of known diabetes mellitus was 5.3% among Maori compared with 1.1% among Europeans.⁷

In New Zealand, information about the prevalence of impaired glucose tolerance (IGT) and impaired fasting glycaemia (IFG) is limited^{2,3,7,9}. Even less is known for any population regarding the prevalence of insulin resistance, a condition generally present prior to the development of IGT and IFG, and a major risk factor for cardiovascular disease, but it has been estimated that approximately 25% of people of European descent have insulin resistance.¹⁰

While there is no information on prevalence of insulin resistance in New Zealand, Simmons et al⁹ found that, compared with Europeans, Maori and Pacific people have poorer insulin sensitivity—when applying the Homeostasis Model Assessment (HOMA) of fasting glucose and insulin as a proxy measure of insulin resistance. This finding was attributed to the high rates of obesity among Maori and Pacific people, rather than of inherent insulin resistance among people of Polynesian descent.

Trials have demonstrated that progression from IGT to type 2 diabetes can be halted through lifestyle changes,^{11–13} but approximately 40% of those people still develop type 2 diabetes despite lifestyle intervention. This may be due to lack of compliance, but also likely to be due to the considerable beta-cell dysfunction already present in those with IGT. Thus, lifestyle intervention among those with insulin resistance, but normal glucose tolerance, may be a more effective approach to preventing or delaying the onset of type 2 diabetes, as well as reducing cardiovascular risk.

However, people with poor insulin sensitivity need to be easily identified, and this study examines different methods to achieve this goal in the clinical setting. A Maori Primary Health Organisation (PHO), Ngati Porou Hauora, has initiated a 2-year community lifestyle intervention programme (the Ngati and Healthy Programme) aimed at reducing the prevalence of insulin resistance among a predominantly Maori community living along the rural East Coast area, north of Gisborne, in the North Island of New Zealand. The outcome of the intervention will be assessed by pre- and post-intervention prevalence surveys of insulin resistance, as well as IFG, IGT, and diabetes. This paper presents results of the pre-intervention prevalence survey.

Methods

This study was based on the sparsely populated East Coast of New Zealand, among communities from Tolaga Bay (50 km north of Gisborne) to Potaka (near Te Araroa). (See the map of the East Coast region; Figure 1.)

The East Coast region has a population of approximately 6000 people. The main types of employment are forestry and farming. Ngati Porou Hauora has over 13,500 enrolled patients in Gisborne and the East Coast, and provides comprehensive services to the East Coast communities through six community clinics and is the only primary care provider in this rural region.

The Ngati Porou Hauora East Coast Enrolled Patient Register was used to obtain a random sample stratified by sex, age group, and ethnicity. Ethical approval was obtained from the Tairāwhiti Ethics Committee in March 2003 and the study took place throughout May to December 2003.

The Project Co-ordinator, and Ngati Porou Hauora rural health nurses and kaiāwhina, (community health workers) invited selected individuals to participate in the survey by letter, and if necessary by phone and home visit (up to three visits in some cases).

Figure 1: The survey was based on the East Coast region of New Zealand's North Island—from Tolaga Bay (50 km north of Gisborne) to Potaka (near Te Araroa)



Of the 741 individuals selected to participate in the study, two individuals were excluded because they had a terminal illness or died, and 150 were unable to be contacted because they had moved away from the East Coast study area. Thus, 589 individuals received an invitation to participate in the study.

At clinics undertaken at four different sites in the East Coast region, a questionnaire including demographic information, relevant medical history, and exercise and dietary history was administered. Height, weight, and waist circumference (midpoint between the anterior superior iliac crest and the lowest rib) were measured, body mass index (BMI) calculated, and blood pressure recorded (after 10 minutes of rest using random zero sphygmomanometers).

Duplicate measures were taken for each of the anthropometric measures, and the average of the two measures used in the analysis. A 75 g oral glucose tolerance test (OGTT) was performed with glucose and insulin measured at 0 and 120 minutes post-glucose load. Participants with documented diabetes did not have an OGTT. Blood was also taken for fasting lipids. All samples were spun and separated after collection. The plasma insulin samples were frozen and transported to Gisborne Laboratory in a mobile freezer (approximately -15°C) either the same day of collection or the following day.

Blood samples were packaged with Bio-freeze Blue Ice bottles to keep the samples at approximately -15°C , and sent immediately to Canterbury Health Laboratory (Christchurch, New Zealand), where they were processed. All other samples were stored in polystyrene boxes for transportation the same or the following day, and were processed on arrival at Gisborne Hospital's Laboratory (New Zealand).

Plasma glucose, total cholesterol, and triglycerides were measured using an enzymatic colorimetric method (Ortho-Clinical Diagnostic reagents). HDL cholesterol was measured using the direct magnetic

method. In accordance with the Royal College of Pathologists of Australasia Quality Assurance Programme, coefficients of variation were 2.2% for glucose, 3.7% for total cholesterol, 6% for HDL, and 3.7% for triglycerides.

Canterbury Health Laboratory, using a Roche Elecsys 2010 automated analyzer with polyethylene glycol to remove antibodies, measured plasma insulin after extraction. The assay detection limit is 0.4 mIU/L. The intra-assay coefficient of variation was 6%.

IFG, IGT, and diabetes were defined according to WHO diagnostic criteria.¹⁴ Insulin resistance was predicted using the McAuley formula based on fasting insulin, triglycerides and BMI,¹⁵—where predicted insulin sensitivity was expressed as exponent ($3.29 - 0.25 \ln[\text{fasting insulin}] - 0.22 \ln[\text{body mass index}] - 0.28 \ln[\text{fasting triglycerides}]$). Normoglycaemic individuals with calculated values = $6.3 \text{ M} \cdot \text{mU}^{-1} \cdot \text{l}^{-1}$ were defined as insulin resistant. As there is no internationally agreed simple method for predicting insulin sensitivity, insulin resistance was also estimated using three other methods: the Homeostasis Model Assessment, HOMA Calculator computer model (version 2.1, 2004),¹⁶ based on fasting insulin and fasting glucose, the National Education Program (NCEP) Adult Treatment Panel (ATP III) definition,¹⁷ which uses a set of clinical criteria (based on blood pressure, waist circumference, triglycerides, HDL, and fasting glucose) and an insulin sensitivity index (based on the average of a fasting and 2-hour glucose, and the average of fasting and 2-hour insulin).¹⁸

The ATP III criteria were applied to all study participants, while HOMA 2.1 and ISI₁₂₀ calculations excluded known diabetics taking oral hypoglycaemic medications or insulin.

Data were entered into a Microsoft Access-based software program. Regression analysis was used to estimate differences between groups after adjustment for sex.

Results

289 people agreed to participate in the study, giving an overall response rate of 48.7%. Males aged 25–29 years had the lowest response rate (24.0%)—whereas males aged 60 years and over, and females aged 30 years and over, had response rates higher than 50%, the highest being 76% for females aged 50–54 years. The female:male ratio was 1.5, and 249 (86%) respondents self-identified as Maori. The following results are for Maori participants only.

Table 1 shows the demographic and clinical characteristics of Maori respondents. The mean BMI was 33.4 kg/m^2 , and more than 90% of both females and males were either overweight or obese, defined as a BMI of 25 kg/m^2 or more.

Table 2 shows the age-standardised prevalence of insulin resistance, IFG or IGT, and diabetes (estimated using our equation based on fasting insulin, triglycerides, and BMI). Overall, the age-standardised prevalence of diabetes, both known and newly diagnosed, was 10.6%. The age-standardised prevalence of known diabetes was about twice that for newly diagnosed diabetes. IGT or IFG was relatively uncommon, whereas the age-standardised prevalence of insulin resistance was 40.3% for females and 36.0% for males.

Figure 2 shows the overall age-specific prevalence rates for diabetes (both known and newly diagnosed) and insulin resistance with normal glucose tolerance. Insulin resistance was more common among the young age groups—with the 30–39 year age group having the highest age-specific rate (44.3%), whereas the prevalence of diabetes increased with age, peaking in the 60–69 year age group at 34.1%.

The characteristics of the group identified as being insulin resistant were compared with the group that did not have any disorders of glucose metabolism (Table 3). The mean age of these groups was similar, as was the proportion who smoked. A history of gout and a family history of diabetes were more common among the insulin-resistant group. Also, individuals in this insulin-resistant group were more likely to be

overweight or obese and have an elevated blood pressure, and an elevated triglyceride level. Total cholesterol and LDL levels were similar.

Table 1. The demographic and clinical characteristics of study participants by sex

Variable	Female (n=153)	Male (n=94)	Total (n=247)
Age (years)	47.8 (±14.1)	51.8 (±14.2)	49.3 (±14.2)
Current smoker (%)	44.4	31.9	39.7
Family history of diabetes (%)	44.4	40.4	42.9
Weight (kg)	85.9 (± 22.4)	92.4 (± 16.1)	88.4 (± 20.4)
BMI (kg/m ²)	33.9 (± 8.2)	32.7 (± 5.2)	33.4 (± 7.2)
Waist (cm)	98.9 (± 17.1)	101.8 (± 11.7)	100.0 (± 15.3)
Systolic BP (mmHg)	124.4 (± 15.1)	125.0 (± 11.8)	124.7 (± 13.9)
Diastolic BP (mmHg)	82.5 (± 13.3)	81.9 (± 10.9)	82.2 (± 12.4)
Total cholesterol (mmol/L)	5.34 (± 0.98)	5.53 (± 1.10)	5.41 (± 1.03)
Triglycerides (mmol/L)	1.70 (± 1.08)	2.01 (± 1.73)	1.82 (± 1.37)
HDL (mmol/L)	1.31 (± 0.35)	1.23 (± 0.31)	1.28 (± 0.34)
Fasting insulin (mIU/L)	15.2 (± 11.5)	13.9 (± 15.9)	14.7 (± 13.3)
Overweight (25≤BMI<30) (%)	26.1	21.3	24.3
Obese (BMI≥30) (%)	64.7	71.3	67.2

Mean values and standard deviations are presented unless otherwise stated; BP=blood pressure; HDL=high-density lipoprotein; BMI=body mass index.

Table 2. Age-standardised prevalence of insulin resistance, IFG or IGT, and diabetes in adults aged 25 years and over

Variable	Female (n=153)		Male (n=94)		Total (n=247)	
	%	(95% CI)	%	(95% CI)	%	(95% CI)
Known diabetes	8.2	(3.5–12.9)	6.2	(1.2–11.2)	7.1	(4.0–10.2)
New diabetes	3.4	(0.6–6.3)	3.5	(0.0–7.6)	3.6	(1.4–5.3)
Total diabetes	11.6	(6.1–17.1)	9.7	(3.5–15.8)	10.6	(6.8–14.4)
IFG or IGT	2.5	(0.2–4.7)	5.9	(0.9–10.9)	4.1	(1.6–6.6)
Insulin resistance*	40.3	(29.6–50.9)	36.0	(19.1–52.9)	37.0	(28.6–45.5)

Age-standardised to the WHO world population; IFG=impaired fasting glycaemia; IGT= impaired glucose tolerance; *Insulin resistance calculated using the McAuley formula among those with normal glucose tolerance.

Table 4A shows different estimates of age-specific prevalence of glucose metabolism disorders in three age categories using our prediction equation and the ATP III criteria. Comparable age trends are evident with the two approaches. As no cut-offs to define insulin resistance have been applied to the HOMA2.1 and the ISI_{0,120} method, means and standard deviations are presented in Table 4B rather than prevalence rates. Those persons taking diabetes medications were excluded for the calculation of the HOMA2.1 as fasting insulin levels are less meaningful in this setting, and as this group did not have an OGTT, they could not be included in the ISI_{0,120} calculation.

Figure 2. Age-specific prevalence of diabetes (known and newly diagnosed) and insulin resistance with normal glucose tolerance

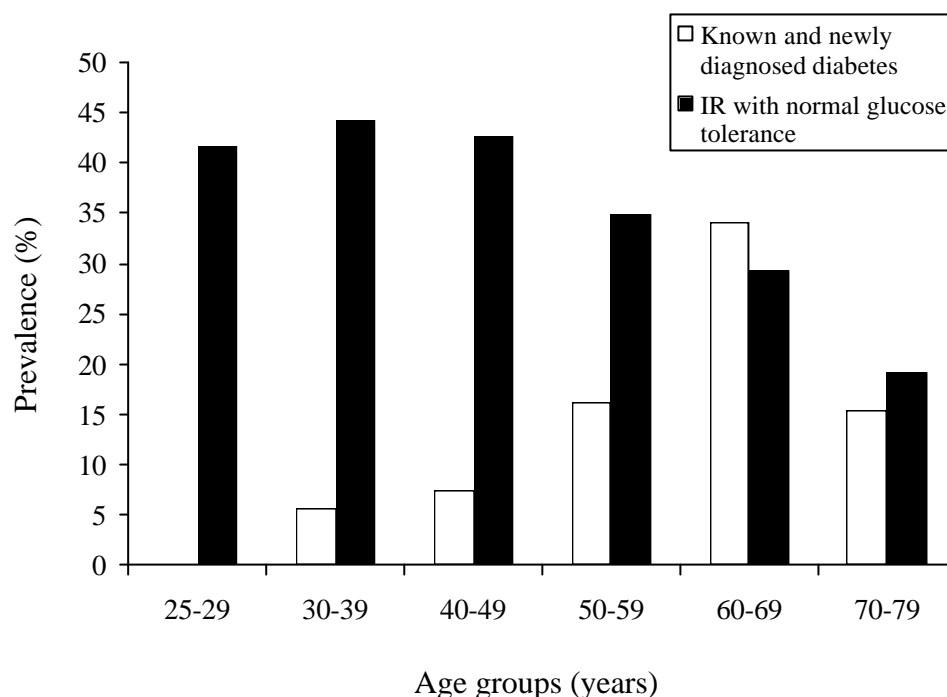


Table 3. Characteristics of the insulin resistance but normal glucose tolerance group and the 'healthy' group

Variable	Insulin resistance (n=91)		'Healthy' (n=112)		Difference (95%CI)
Sex (male)	31.9 %		40.2 %		
Age (yrs)	46.2	(12.8)	48.7	(14.8)	-2.5 (-6.4, 1.3)
Current smoker	42.9 %		44.6 %		0.9 (0.5, 1.6)*
History of gout	20.9 %		7.1 %		4.2 (1.8, 10.6)*
History of hypertension	12.1 %		16.1 %		0.7 (-0.3, 1.6)*
History of IHD	8.8 %		5.4 %		1.9 (0.6, 5.7)*
Family history of diabetes	51.6 %		31.3 %		2.3 (1.3, 4.2)*
Family history of IHD	33.0 %		27.7 %		1.2 (0.7, 1.2)*
Weight (kg)	98.9	(19.5)	77.5	(15.3)	22.1 (17.4, 26.8)
BMI (kg/m ²)	37.0	(6.9)	29.5	(5.3)	7.4 (5.7, 9.1)
Waist (cm)	107.5	(13.0)	90.4	(11.4)	17.5 (14.1, 20.8)
Systolic BP (mmHg)	125.8	(15.7)	120.9	(10.3)	4.9 (1.3, 8.6)
Diastolic BP (mmHg)	84.0	(12.5)	79.0	(11.7)	5.0 (1.6, 8.3)
Total cholesterol (mmol/L)	5.54	(1.00)	5.19	(1.02)	0.37 (0.09, 0.65)
HDL cholesterol (mmol/L)	1.16	(0.28)	1.44	(0.35)	-0.29 (-0.38, -0.20)
Triglycerides (mmol/L)	2.35	(1.24)	1.04	(0.38)	1.3 (1.1, 1.6)
Fasting insulin (mU/L) [†]	17.81	(15.91, 19.95)	6.49	(5.84, 7.21)	2.72 (2.33, 3.18)

Data presented are mean values and standard deviations unless otherwise stated; Differences or odds ratios are adjusted for sex; IHD=ischaemic heart disease; BP=blood pressure; HDL=high-density lipoprotein; BMI=body mass index; *Odds ratio (95% CI); †Geometric means (95% CI) and their ratio (95% CI) based on a log transformation.

Table 4A. Comparison of age-specific prevalence rates of insulin resistance using our formula and the ATP III criteria

Variable	Age groups (years)					
	25–39 (n=82)		40–59 (n=97)		60–79 (n=67)	
	Number	(%)	Number	(%)	Number	(%)
McAuley formula*						
Diabetes	4		11		18	
IFG or IGT	2		5		4	
Insulin resistance	36		38		17	
Total insulin resistance	42	(51.2)	54	(55.7)	39	(58.2)
ATP III criteria						
Total insulin resistance	28	(34.1)	35	(36.1)	28	(41.8)

*McAuley formula = $\exp[3.29 - 0.25\ln(\text{insulin}) - 0.22\ln(\text{BMI}) - 0.28\ln(\text{TAG})]$. (The formula was developed to predict insulin sensitivity, values = $6.3 \text{ M} \cdot \text{mU} \cdot \text{l}^{-1}$ define those who are insulin resistant.); IFG=impaired fasting glycaemia; IGT=impaired glucose tolerance.

Table 4B Comparison of means and standard deviations by age groups for HOMA 2.1 and ISI_{0,120}

Calculation	Age groups (years)					
	25–39 (n=79)		40–59 (n=90)		60–79 (n=52)	
	Mean	(SD)	Mean	(SD)	Mean	(SD)
HOMA 2.1 - %S* [†]	87.4	(119.0)	101.7	(84.7)	104.8	(83.4)
ISI _{0,120} * [†]	145.1	(76.5)	149.4	(103.5)	110.7	(51.4)

* %S is derived from the HOMA 2.1 computer model, and is a measure of insulin sensitivity with 100% defined as normal, and higher numbers signifying greater sensitivity; ISI_{0,120} is a calculated measure of sensitivity with higher numbers signifying greater sensitivity; [†] HOMA 2.1 and ISI_{0,120} calculations exclude those on diabetes medications.

Discussion

Slightly less than half the eligible participants (49%) completed all components of the survey. While a higher response rate would have been desirable, this rate was comparable with that of similar surveys such as the recent AUSDIAB Study.¹⁹ The low overall response rate in our study can to a considerable extent be explained by the poor response among the younger age group, especially males. The forestry industry employs a high proportion of the young men, and because their working days begin early, many young men were unable to obtain time away from work.

The population of the East Coast is spread over a large geographic area, and the distances required to travel to the survey centres was a major disincentive to participation in all age groups. However, the coordinated efforts of the study coordinator, kaiawhina, and rural health nurses to provide frequent reminders and to arrange transport to the survey centres resulted in a much higher response rate among middle aged and older individuals. Among women aged 40 years and over, the response rate was 62%, which compares favourably with the 68% response rate among 40–79 year old Maori women who were invited to complete a questionnaire

and have anthropometric measures and a random blood glucose in a 1995/96 South Auckland survey.⁹ Comparison of the response rate for males is less favourable (43% vs 63%). The high response rate (93% of households) in an earlier South Auckland survey involved only the completion of a questionnaire and suggests that the oral glucose tolerance test (OGTT) and physical examination may be disincentives.⁸ However, an OGTT is an essential component of any study which aims to assess the prevalence of disturbances of carbohydrate metabolism.

The limited number of prevalence studies among Maori populations in New Zealand are not directly comparable, and it was not possible to assess changes in diabetes prevalence over time. However, the comparable prevalence of known diabetes among women aged over 45 years in the East Coast population studied in the present survey (14%) and of self-reported diabetes in the 2002/03 NZHS (13%)⁶ provides strong confirmation of the overall high prevalence. Of interest, are the appreciably higher rates of self reported diabetes among males aged over 45 years in the NZHS (21%) than in the present study (10%), where the diagnosis was confirmed. The difference may be partly explained by the low response rate among East Coast men, but we cannot explain this difference with certainty.

Interestingly, in our data, newly diagnosed diabetes rates were only half that of known diabetes whereas most other surveys have reported comparable rates of known and newly diagnosed cases.^{3,7,9} This may well be due to the high level of awareness of diabetes in the study area. This in turn has resulted in more frequent screening of high-risk individuals.

A key purpose of the present study was to assess the prevalence of insulin resistance. Individuals with insulin resistance in the general population urgently need to be targeted for diabetes prevention and cardiovascular risk reduction strategies, but no universally accepted method for predicting insulin sensitivity exists. Euglycaemic clamps and intravenous glucose tolerance tests (IVGTTs) are limited to research settings. Various surrogate methods for predicting insulin sensitivity have been used in studies and a number of newer approaches have been suggested.²⁰

The most widely published method for predicting insulin sensitivity is the Homeostasis Model Assessment based on fasting glucose and insulin,²¹ which should ideally be based on three separate blood measurements taken 5 minutes apart and calculated using the model programme, but in most cases is based on a single measure and is estimated using a simplified formula.¹⁶ Only the HOMA-model has been shown to correlate well with the euglycaemic clamp.^{16,18}

Our study and others have found the HOMA formula to be no better than a fasting insulin in this regard.^{15,18} The HOMA formula is generally applied to those with normal glucose tolerance (NGT), IFG, IGT, and diabetes with several caveats for use in those on sulphonylureas and exogenous insulin.¹⁶

No cut-off has been proposed to identify a group with poor insulin action. Furthermore, there has been criticism of choosing surrogates to predict insulin sensitivity that correlate well with a euglycaemic clamp, a dynamic test under non physiological conditions. Thus, we have selected a further method for predicting insulin resistance, developed by Gutt et al, based on the average of the fasting and 2-hour glucose and insulin levels. This has been shown in prospective studies to be the best method for predicting the development of type 2 diabetes.²⁰

The failure to show a deterioration in insulin sensitivity with age (Table 4B) with both these measures reflects the fact that those with diabetes on medication have been excluded because of the difficulty in interpreting their insulin sensitivity data using this approach. It is clear that HOMA 2.1 and $ISI_{0,120}$ formulae are currently inappropriate to determine prevalence of insulin resistance. However they are likely to be of value in assessing response to intervention programmes aimed at improving insulin sensitivity, and will be used for this purpose in the Ngati and Healthy programme.

To date, the most frequently used approach for determining frequency of insulin resistance or identifying insulin resistant individuals has been to use a set of surrogate clinical and laboratory criteria. We have compared our equation¹⁵ which has been independently validated²² with the ATP III criteria¹⁷ for the definition of those with the metabolic syndrome. Our equation combines fasting insulin, triglycerides and BMI as continuous variables, so that those who would have fallen just outside a particular cut off can still be included depending on the other variables.

An arbitrary cut off (of less than or equal to $6.3 \text{ M}\cdot\text{mU}\cdot\text{l}^{-1}$) is applied to select those with poor insulin sensitivity, based on the lowest quartile for a lean population. Inevitably, the cut-off point is somewhat arbitrary but a similar difficulty applies to the ATP III criteria which might be expected to miss an even greater number of insulin resistant individuals since arbitrary cut offs are applied to several clinical and metabolic variables.

Table 4A shows that using our approach, more than half the population have insulin resistance, and this increases with increasing age, when those with IFG, IGT, and type 2 diabetes are included. Not surprisingly, the ATP III criteria gives rates substantially lower than this, but the same pattern of increasing rates across age groups is observed.

It has been estimated that as many as 25% of adults of European descent may be insulin resistant.¹⁰ The appreciably higher rates observed here (40% among women, 36% among men) represent considerable cause for concern given that this condition is believed to be the underlying cause of most cases of type 2 diabetes mellitus as well as being an important contributor to cardiovascular risk. Of especially great concern are the high rates among young individuals (Figure 2). This suggests that the future burden of diabetes and other diseases associated with insulin resistance and the metabolic syndrome is likely to escalate in the near future unless effective intervention programmes are in place.

The Ngati Porou Hauora Ngati & Healthy Programme is one such pioneering programme, which will be formally evaluated using well established methods.²³ A national diabetes prevalence survey, which would include estimates of IFG, IGT, and insulin resistance as well as associated clinical, anthropometric, and metabolic variables and assessment of nutritional status, is imperative since no such national data exist. Such information is essential for health care planning for what is arguably the most important epidemic disease in New Zealand and for assessing the effects of national strategies aimed at reducing obesity and diabetes rates.

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References:

1. King H, Aubert RE, Herman WH. Global burden of diabetes, 1995-2025: prevalence, numerical estimates, and projections. *Diabetes Care*. 1998;21:1414–31.
2. Bourn D, Mann J. Screening for noninsulin dependent diabetes mellitus and impaired glucose tolerance. *N Z Med J*. 1992;105:413.
3. Brown CR, Hider PN, Scott RS, et al. Diabetes mellitus in a Christchurch working population. *N Z Med J*. 1984;97:487–9.
4. Lintott CJ, Hanger HC, Scott RS, et al. Prevalence of diabetes mellitus in an ambulant elderly New Zealand population. *Diabetes Res Clin Pract*. 1992;16:131–6.
5. Ministry of Health. Taking the pulse. The 1996/97 New Zealand Health Survey. Wellington: Ministry of Health; 1999. Available online. URL: <http://www.moh.govt.nz/moh.nsf/0/d7b3cf1eee94fefb4c25677c007ddf96?OpenDocument> Accessed December 2004.
6. Ministry of Health. A snapshot of health. Provisional results of the 2002/03 New Zealand Health Survey. Wellington: Ministry of Health; 2003. Available online. URL: <http://www.moh.govt.nz/moh.nsf/0/9ad4668e36fecf37cc256deb007c38b8?OpenDocument> Accessed December 2004.
7. Scragg R, Baker J, Metcalf P, Dryson E. Prevalence of diabetes mellitus and impaired glucose tolerance in a New Zealand multiracial workforce. *N Z Med J*. 1991;104:395–7.
8. Simmons D, Gatland B, Fleming C, et al. Prevalence of known diabetes in a multiethnic community. *N Z Med J*. 1994;107:219–22.
9. Simmons D, Thompson CF, Volklander D. Polynesians: prone to obesity and Type 2 diabetes mellitus but not hyperinsulinaemia. *Diabet Med*. 2001;18:193–8.
10. Ford ES, Giles WH, Dietz WH. Prevalence of the metabolic syndrome among US adults: findings from the third National Health and Nutrition Examination Survey. *JAMA*. 2002;287:356–9.
11. 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.
12. Pan XR, Li GW, Hu YH, et al. Effects of diet and exercise in preventing NIDDM in people with impaired glucose tolerance. The Da Qing IGT and Diabetes Study. *Diabetes Care*. 1997;20:537–44.
13. 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.

14. World Health Organisation Department of Noncommunicable Disease Surveillance. Definition, Diagnosis and Classification of Diabetes Mellitus and its complications. Part 1: Diagnosis and Classification of diabetes mellitus. Geneva: World Health Organisation; 1999.
15. McAuley KA, Williams SM, Mann JI, et al. Diagnosing insulin resistance in the general population. *Diabetes Care*. 2001;24:460–4.
16. Wallace TM, Levy JC, Matthews DR. Use and Abuse of HOMA Modeling. *Diabetes Care*. 2004;27:1487–95.
17. National Cholesterol Education Program. Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) final report. *Circulation*. 2002;106:3143–421.
18. Gutt M, Davis CL, Spitzer SB, et al. Validation of the insulin sensitivity index (ISI(0,120)): comparison with other measures. *Diabetes Res Clin Pract*. 2000;47:177–84.
19. Dunstan D, Zimmet P, Welborn T, on behalf of the AusDiab Steering Committee. *Diabetes & Associated Disorders in Australia - 2000. The Accelerating Epidemic. The Australian Diabetes, Obesity and Lifestyle Study (AusDiab)*. Melbourne: International Diabetes Institute; 2001.
20. Hanley AJ, Williams K, Gonzalez C, et al. Prediction of type 2 diabetes using simple measures of insulin resistance: combined results from the San Antonio Heart Study, the Mexico City Diabetes Study, and the Insulin Resistance Atherosclerosis Study. *Diabetes*. 2003;52:463–9.
21. Matthews DR, Hosker JP, Rudenski AS, et al. Homeostasis model assessment: insulin resistance and beta-cell function from fasting plasma glucose and insulin concentrations in man. *Diabetologia*. 1985;28:412–9.
22. Ascaso JF, Pardo S, Real JT, et al. Diagnosing insulin resistance by simple quantitative methods in subjects with normal glucose metabolism. *Diabetes Care*. 2003;26:3320–5.
23. Kirkwood BR, Cousens SN, Victora CG, de Zoysa I. Issues in the design and interpretation of studies to evaluate the impact of community-based interventions. *Trop Med Int Health*. 1997;2:1022–9.