Indigenous populations in the Asia-Pacific region have extremely high prevalence of diabetes mellitus and associated complications. This pattern has been attributed to genetic susceptibility combined with rapid changes in lifestyle and nutrition over the past 60 years. Studies in populations of European descent showed body mass index (BMI) > 25 kg/m² strongly predicts incidence of diabetes. These studies formed the basis of clinical guidelines attributing to genetic susceptibility components. However, work suggested a much lower “healthful” BMI range among other populations and Australian Aboriginals.

More recently, waist circumference (WC) has been used to predict diabetes risk in populations. Other studies found waist-to-hip ratio (WHR) a more useful measure of obesity to identify individuals with cardiovascular disease risk factors. Incidence studies in different populations provide some insight into these trajectories, and help clarify potential causal pathways, some of them modifiable. These studies also give an indication of the future burden of diabetes to the health care system and community, and assist in planning for preventive interventions, as well as secondary and tertiary level services.

Except for a follow-up study of central Australian Aborigins reported a decade ago and a more recent report from the Torres Strait, there are few diabetes incidence data for Indigenous Australians, which impedes the understanding of the causes of diabetes in this population. The aims of this study were to estimate the incidence of type 2 diabetes in two ethnically distinct groups of Australian Indigenous adults from rural communities in north Queensland, and to identify anthropometric and metabolic measures that predict the development of diabetes in these groups.

RESEARCH DESIGN AND METHODS

Study population
Baseline data were collected from 1814 Indigenous people in 19 rural Indigenous communities in Far North Queensland, who participated in the “Well Person’s Health Check” between 1999 and 2000 (methods for this cross-sectional study have been reported in detail elsewhere). The follow-up data were collected between 2004 and 2007. The study was approved by the Cairns and Hinterland Health Service District Ethics Committee, with support from the relevant peak Indigenous health councils.

Measurements
Fasting blood levels of total cholesterol, high-density lipoprotein (HDL) cholesterol, triglycerides and glucose were directly measured, as were height, weight and WC. Physical activity, smoking and alcohol intake measurement tools were similar to those used in other studies. Incident diabetes was defined as clinical diagnosis verified by the participant’s medical records, or a 2-hour glucose tolerance test result (blood glucose level >11.1 mmol/L, 2-hour post-glucose load), or fasting blood glucose level (> 7 mmol/L). WHR cutpoints were defined according to World Health Organization (WHO) criteria (>1.0 for men and >0.85 for women). Metabolic syndrome (MetS) was defined using the International Diabetes Federation criteria. Definition of physical activity levels followed the American Heart Association criteria. High-risk alcohol consumption using 7-day recall was defined according to Australia’s National Health and Medical Research Council criteria.

Analysis
Data analysis excluded participants who identified as non-Indigenous, were aged under 15 years, or had a diagnosis of diabetes at baseline.

The follow-up period for participants with incident diabetes was the time from entering the baseline study to the diagnosis date. For those who did not develop diabetes, the follow-up period was the interval between the day of the baseline survey and the follow-up. Each participant was followed up once. The age- and sex-specific cumulative incidence rate stratified by ethnicity was calculated by dividing the...
number of new diagnoses by the total person follow-up years of the corresponding subgroups. Direct standardisation was conducted using the 2007 Australian Bureau of Statistics national data as the reference population.

Cox proportional hazard models were used to explore the association of baseline BMI and MetS with diabetes incidence in each population. Baseline BMI was categorised using WHO criteria. WC measures were categorised using WHO gender-specific criteria: obese \( \geq 88 \) cm in women and \( \geq 102 \) cm in men. Univariate Cox models were used to estimate crude and adjusted hazard ratios. The analysis was carried out using Stata, version 10 (StatCorp, College Station, Tex, USA).

**RESULTS**

Among the 1814 eligible participants at baseline, 554 completed the follow-up health check; 137 (7.6%) died before follow-up; 319 (17.6%) changed place of residence before follow-up, and 28 (1.5%) were in prison at the time of follow-up. The other 776 (42.8%) were invited to the follow-up but did not attend. Participants who were followed-up were older (mean age ± standard error of the mean: 37 ± 0.6 vs 34.4 ± 0.4) and more likely to identify as Torres Strait Islanders (47% vs 33%) than those not followed-up; however, there was no sex difference.

Among the 554 participants who completed follow-up, 225 self-identified as Aboriginal, 262 as Torres Strait Islanders and 67 claimed both Aboriginal and Torres Strait Islander descent. Aboriginal participants had higher blood levels of triglycerides but lower WC and BMI than Torres Strait Islanders. Levels of self-reported physical activity were also lower among Aboriginal participants. Aboriginal women were older than Torres Strait Islander women, and Aboriginal men had the highest diastolic blood pressure (BP) and the highest proportion of “high-risk” drinking. There were no significant differences between the groups in smoking, systolic BP or blood levels of glucose and HDL cholesterol. Despite large differences in baseline BMI and WC, WHR was similar for both groups (Box 1).

The age- and sex-specific incidence of diabetes stratified by ethnicity is shown in Box 2. The mean follow-up interval was 6.3 years and the total follow-up was 3412 person-years (py). One hundred participants were newly diagnosed with diabetes after an average of 4.2 years of follow-up. Most participants who developed diabetes were aged 25–54 years, and 22 were over 55 years at baseline. The overall cumulative diabetes incidence rate was 29.3 per 1000 py (95% CI, 24.1–35.7) — 31.3 per 1000 py (95% CI, 24.0–40.9) in women and 27.2 per 1000 py (95% CI, 20.4–36.4) in men. Aboriginal participants had an incidence rate of 29.7 per 1000 py (95% CI, 20.4–38.4) compared with 29.0 per 1000 py (95% CI, 21.8–38.6) among Torres Strait Islanders. Participants aged 15–24 years showed an incidence rate of 9.8 per 1000 py; those aged 25–34 years, 21.8; those aged 35–54, 37.4; and those 55 years and older, 47.6 (trend test \( P < 0.001 \)). The direct age-standardised incidence rate was 30.5 per 1000 py in the study population (95% CI, 16.2–44.8), nearly four times that found in the AusDiab 2005 study.10

Among Aboriginals who were not obese (by WC) at baseline, incidence was 22.3 per 1000 py, more than three times the rate in Torres Strait Islanders, although the rates were not different in the obese category between the two populations. For those with MetS at baseline, incidence rates were 51.3 per 1000 py for Aboriginals and 48.0 per 1000 py for Torres Strait Islanders (Box 3).

Compared to normal body size defined by WC, obesity defined by WC doubled the risk of developing diabetes in Aborigi-

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**Table 1: Baseline characteristics of 554 Indigenous Australians completing follow-up survey in rural communities in north Queensland**

<table>
<thead>
<tr>
<th></th>
<th>Aboriginals</th>
<th>Torres Strait Islanders</th>
<th>Joint descendents</th>
</tr>
</thead>
<tbody>
<tr>
<td>Women</td>
<td>n = 113</td>
<td>n = 129</td>
<td>n = 38</td>
</tr>
<tr>
<td>Age (years)*</td>
<td>38.4 (35.5–41.3)</td>
<td>34.4 (32.2–36.6)</td>
<td>31.0 (27.3–34.7)</td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>124.0 (120.2–127.9)</td>
<td>125.5 (122.2–128.7)</td>
<td>119.1 (114.7–123.4)</td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td>69.2 (66.8–71.7)</td>
<td>67.3 (64.9–69.7)</td>
<td>65.2 (61.8–68.5)</td>
</tr>
<tr>
<td>Fasting glucose (mmol/L)</td>
<td>4.8 (4.6–4.9)</td>
<td>4.9 (4.8–5.0)</td>
<td>4.8 (4.5–5.0)</td>
</tr>
<tr>
<td>Total cholesterol (mmol/L)</td>
<td>4.7 (4.5–4.9)</td>
<td>4.9 (4.7–5.0)</td>
<td>4.4 (4.1–4.6)</td>
</tr>
<tr>
<td>HDL cholesterol (mmol/L)</td>
<td>1.1 (1.0–1.2)</td>
<td>1.1 (1.0–1.4)</td>
<td>1.1 (1.0–1.2)</td>
</tr>
<tr>
<td>Triglycerides (mmol/L)*</td>
<td>1.7 (1.5–2.0)</td>
<td>1.4 (1.2–1.6)</td>
<td>1.1 (1.0–1.3)</td>
</tr>
<tr>
<td>WC (cm)*</td>
<td>93.6 (90.3–96.9)</td>
<td>102.3 (99.5–105.1)</td>
<td>99.9 (93.6–106.3)</td>
</tr>
<tr>
<td>WHR</td>
<td>0.91 (0.89–0.93)</td>
<td>0.89 (0.88–0.90)</td>
<td>0.89 (0.86–0.91)</td>
</tr>
<tr>
<td>BMI (kg/m²)*</td>
<td>26.0 (24.7–27.3)</td>
<td>31.7 (30.5–33.0)</td>
<td>31.1 (28.2–34.1)</td>
</tr>
<tr>
<td>Smokers</td>
<td>48.7% (39.4–57.9)</td>
<td>51.2% (42.5–59.8)</td>
<td>57.9% (42.0–73.8)</td>
</tr>
<tr>
<td>Drinkers</td>
<td>54.5% (45.2–63.9)</td>
<td>58.4% (49.7–67.1)</td>
<td>66.7% (51.0–82.3)</td>
</tr>
<tr>
<td>Risky drinkers</td>
<td>33.6% (13.0–50.1)</td>
<td>28.7% (20.8–45.6)</td>
<td>36.9% (21.3–52.4)</td>
</tr>
<tr>
<td>PA sufficient</td>
<td>34.2% (31.6–29.7)</td>
<td>27.9% (20.1–23.7)</td>
<td>15.8% (4.0–27.6)</td>
</tr>
</tbody>
</table>

Data are means or percentages (95% CI). BMI = body mass index. DBP = diastolic blood pressure. HDL = high-density lipoprotein. PA = physical activity. SBP = systolic blood pressure. WC = waist circumference. WHR = waist-to-hip ratio. * Significant at \( P < 0.05 \) using analyses of variance or \( \chi^2 \) tests.
DEFINING THE GAP — RESEARCH

nals (rate ratio [RR], 2; 95% CI, 1.1–3.6) and increased the risk more than six times in Torres Strait Islanders (RR, 6.3; 95% CI, 2.5–16.1). The association between obesity defined by BMI and the risk of diabetes was the same as that defined by WC in Aboriginals but less marked in Torres Strait Islanders (Box 3). After adjusting for age, sex, smoking, drinking and physical activity, obesity did not significantly predict diabetes incidence in Aboriginals but remained a significant predictor in Torres Strait Islanders. The presence of the MetS increased the risk of developing diabetes in both Aboriginals (adjusted hazard ratio [HR] 2.7; 95% CI, 1.8–4.2) and Torres Strait Islanders (adjusted HR 2.9; 95% CI, 1.6–5.2) compared with absence of the MetS at baseline after adjustment in both populations.

DISCUSSION

This study found an incidence of diabetes in two Indigenous groups that was nearly four times higher than that reported in the general Australian population and appears to be about 50% higher than that in the first published incidence study over a decade ago (which found an incidence rate of 20.3 per 1000 py in an Aboriginal population in central Australia).
These findings reflect those reported from studies of North American Native populations, with higher incidence at each BMI, age and sex stratum than among resident non-Indigenous populations. The finding of a similar incidence of diabetes despite very large differences in baseline BMI and WC between the two Australian Indigenous groups is striking. However, WHR and MetS appeared to predict incidence more uniformly across the ethnic groups than BMI or WC. Notably, in this study, Aboriginal men had significantly higher fasting triglyceride levels and lower BMI and WC than Torres Strait Islanders, and Aboriginal men reported much higher rates of “risky” alcohol consumption and possibly smoking at baseline. The combination of high triglyceride levels and WC in men (the “hypertriglyceridemic waist”) has been found to predict high risk of coronary artery disease in populations of European ancestry. In Indigenous Australians, central obesity measured by WC is highly associated with non-alcoholic fatty liver, independently of lifestyle behaviours (odds ratio, 2.7, 95% CI, 1.2–6.0). Thus, it would seem that several pathways lead from central obesity and disordered lipid metabolism to insulin resistance and clinical diabetes, and that the relative importance of each might differ between individuals and populations, depending on the interplay of epigenetic, socioeconomic, environmental and individual exposures over the life course. Low birthweight, combined with rapid weight gain in adulthood and an energy-dense but nutrient-deficient diet, is a common experience in Indigenous communities in recent decades.

This study has several limitations, including relatively small numbers of participants and low follow-up rates, which limit comparison with the earlier Australian study and its antecedents deserves further exploration in the pathogenesis of diabetes in these populations. Achieving “metabolic fitness”, including waist reduction and lowering the excess of free fatty acids, should be a target for the treatment of insulin resistance. Improving nutrition, including availability and affordability of healthy food, is a key part of such a strategy.

ACKNOWLEDGEMENTS

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COMPETING INTERESTS

None identified.

AUTHOR DETAILS

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