Incidence of type 2 diabetes in two Indigenous Australian populations: a 6-year follow-up study

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ABSTRACT

Objective: To estimate the incidence of type 2 diabetes in two ethnically distinct Indigenous populations in north Queensland, Australia.

Design, setting and participants: A community-based follow-up study of 1814 Australian Aboriginal and Torres Strait Islander adults from 1999 to 2007. Participants were initially free of diabetes and lived in 19 remote communities in Far North Queensland.

Main outcome measures: Fasting blood glucose level; diagnosis of diabetes; blood lipid levels; weight; waist circumference (WC); and blood pressure.

Results: Of the 554 adults who completed the study, 100 developed diabetes over 3412 person-years (py) of follow-up. The incidence of diabetes was similar for Aboriginals (29.7 [95% CI, 20.4–38.4] per 1000 py) and Torres Strait Islanders (29.0 [95% CI, 21.8–38.6] per 1000 py) despite large differences in baseline body mass index (BMI) and WC. The age-standardised incidence for both populations was 30.5 per 1000 py. Obesity defined by WC increased the risk of developing diabetes for Aboriginals (rate ratio [RR], 2.0 [95% CI, 1.1–3.6]) and for Torres Strait Islanders (RR, 6.3 [95% CI, 2.5–16.1]) compared with normal WC. Presence of the metabolic syndrome (MetS) was a strong predictor of incident diabetes (adjusted hazard ratio, 2.4 [95% CI, 1.6–3.7]). For both groups, waist-to-hip ratio and the presence of the MetS better predicted diabetes than WC or BMI.

Conclusions: The incidence of diabetes in these Indigenous Australians is nearly four times higher than for the non-Indigenous population and 50% higher than the incidence reported 10 years ago in Australian Aboriginals. Currently used BMI cut-off points are not appropriate for Indigenous Australians to predict diabetes.

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Indigenous populations in the Asia–Pacific region have extremely high prevalence of diabetes mellitus and associated complications.[1] 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.[2] These studies formed the basis of clinical guidelines defining a "healthful" BMI as < 25 kg/m^2 and "obese" as ⩾ 30 kg/m^2.[3] However, other work suggested a much lower "healthful" BMI range among other populations[4] and Australian Aboriginals.[5]

More recently, waist circumference (WC) has been used to predict diabetes risk in populations.[6] Other studies found waist-to-hip ratio (WHR) a more useful measure of obesity to identify individuals with cardiovascular disease risk factors.[7] 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 Aboriginals reported a decade ago[5] and a more recent report from the Torres Strait,[8] 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[9]). 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.[10] 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).[3] WHR cutpoints were defined according to World Health Organization (WHO) criteria (> 1.0 for men and > 0.85 for women).[4] Metabolic syndrome (MetS) was defined using the International Diabetes Federation criteria.[11] Definition of physical activity levels followed the American Heart Association criteria.[12] High-risk alcohol consumption using 7-day recall was defined according to Australia's National Health and Medical Research Council criteria.[13]

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

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

	Aboriginals	Torres Strait Islanders	Joint descendents
Women	*n* = 113	*n* = 129	*n* = 38
Age (years)*	38.4 (35.5–41.3)	34.4 (32.2–36.6)	31.0 (27.3–34.7)
SBP (mmHg)	124.0 (120.2–127.9)	125.5 (122.2–128.7)	119.1 (114.7–123.4)
DBP (mmHg)	69.2 (66.8–71.7)	67.3(64.9–69.7)	65.2 (61.8–68.5)
Fasting glucose (mmol/L)	4.8 (4.6–4.9)	4.9 (4.8–5.0)	4.8 (4.5–5.0)
Total cholesterol (mmol/L)	4.7 (4.5–4.9)	4.9 (4.7–5.0)	4.4 (4.1–4.6)
HDL cholesterol (mmol/L)	1.1 (1.07–1.2)	1.1 (1.06–1.14)	1.1 (1.0–1.2)
Triglycerides (mmol/L)*	1.7 (1.5–2.0)	1.4 (1.2–1.6)	1.1 (1.0–1.3)
WC (cm)*	93.6 (90.3–96.9)	102.3 (99.5–105.1)	99.9 (93.6–106.3)
WHR	0.91(0.89–0.93)	0.89 (0.88–0.90)	0.89 (0.86–0.91)
BMI (kg/m^2)*	26.0 (24.7–27.3)	31.7 (30.5–33.0)	31.1 (28.2–34.1)
Smokers	48.7% (39.4–57.9)	51.2% (42.5–59.8)	57.9% (42.0–73.8)
Drinkers	54.5% (45.2–63.9)	58.4% (49.7–67.1)	66.7% (51.0–82.3)
Risky drinkers	33.6% (13.0–28.1)	28.7% (20.8–36.5)	36.9% (21.3–52.4)
PA sufficient	20.4% (12.9–27.8)	27.9% (20.1–35.7)	15.8% (4.0–27.6)
Men	*n* = 112	*n* = 133	*n* = 29
Age (years)	41.1(38.7–43.49)	38.4 (35.9–40.9)	34.7 (29.2–40.1)
SBP (mmHg)	135.0 (131.8–138.2)	136.5 (134.0–139.5)	130.1 (124.8–135.4)
DBP (mmHg)*	78.4 (76.0–80.9)	74.2 (72.0–76.4)	75.2 (65.8–74.6)
Fasting glucose (mmol/L)	4.8 (4.7–5.0)	5.0 (4.9–5.1)	4.8 (4.5–5.0)
Total cholesterol (mmol/L)	5.2 (5.0–5.4)	5.3 (5.1–5.5)	4.6 (4.3–5.0)
HDL cholesterol (mmol/L)	1.2 (1.1–1.23)	1.1 (1.06–1.2)	1.1 (1.05–1.2)
Triglycerides (mmol/L)*	2.5 (2.1–2.9)	1.9 (1.7–2.1)	1.3 (1.1–1.5)
WC (cm)*	90.9 (88.4–93.5)	100.7 (97.9–103.5)	99.1 (94.3–103.9)
WHR	0.96 (0.94–0.97)	0.95 (0.94–0.96)	0.98 (0.92–1.03)
BMI (kg/m^2)*	24.3 (23.4–25.2)	29.6 (28.5–30.6)	29.1 (27.3–30.9)
Smokers	65.8% (56.9–74.7)	56.1% (47.5–64.6)	51.7% (33.2–70.2)
Drinkers	86.6% (79.9–92.8)	80.3% (73.5–87.1)	93.1% (83.7–102.5)
Risky drinkers*	68.8% (60.1–77.4)	49.6% (41.1–58.2)	51.7% (33.2–70.3)
PA sufficient*	20.5% (13.0–28.1)	35.3% (27.2–34.5)	31.0% (13.9–48.2)

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 χ^2 tests. ◆

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 obesity and MetS with diabetes incidence in each population. Baseline BMI was categorised using WHO criteria.[14] WC measures were categorised using WHO gender-specific criteria: obese ⩾ 88 cm in women and ⩾ 102 cm in men.[14] Univariate Cox models were used to estimate crude and adjusted hazard ratios. The analysis was carried out using Stata, version 10 (Statacorp, 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: 37.3 ±0.6 v 34.4 ±0.4) and more likely to identify as Torres Strait Islanders (47% v 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 aged 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-

2 Diabetes incidence by age, sex and ethnicity among 554 Indigenous Australians in north Queensland

Age group	Aboriginals (*n* = 225) Case/py (no.)	Aboriginals (*n* = 225) Incidence per 1000 py (95% CI)	Torres Strait Islanders (*n* = 262) Case/py (no.)	Torres Strait Islanders (*n* = 262) Incidence per 1000 py (95% CI)	Joint descendants (*n* = 67) Case/py (no.)	Joint descendants (*n* = 67) Incidence per 1000 py (95% CI)	Total Incidence
Women							
15–34 y	10/346.5 (53)	28.9 (15.5–53.6)	6/497.8 (78)	12.1 (5.4–26.8)	3/123.1 (24)	24.4 (7.9–75.5)	19.6 (12.5–30.8)
35–54 y	9/249.9 (38)	36.0 (18.7–69.2)	10/243.0 (41)	41.2 (22.1–76.5)	4/60.3 (13)	66.3 (24.9–176.7)	41.6 (27.6–62.6)
55 + y	6/137.7 (22)	43.6 (19.6–97.0)	6/58.5 (10)	102.0 (46.0–228.1)	0/6.8 (1)	0	59.1 (33.6–104.1)
Subtotal	25/734.1 (113)	34.1 (23.0–50.4)	22/799.4 (129)	27.5 (18.1–41.8)	7/190.2 (38)	36.8 (17.5–77.2)	31.3 (24.0–40.9)
Men							
15–34 y	2/229.3 (34)	8.7 (2.2–35.0)	7/407.7 (60)	17.2 (8.1–36.0)	2/93.4 (17)	21.4 (5.4–85.6)	15.1 (8.3–27.2)
35–54 y	13/365.8 (60)	35.5 (20.6–61.2)	12/316.3 (54)	37.9 (21.6–66.8)	0/41.1(8)	0	34.6 (23.4–51.2)
55 + y	3/120.8 (18)	24.8 (8.2–76.9)	6/97.0 (19)	61.9 (27.8–237.7)	1/17.5 (4)	57.1 (8.0–405.4)	42.5 (22.9–78.9)
Subtotal	18/715.9 (112)	25.1 (15.9–39.9)	25/821.0 (133)	30.5 (20.6–45.1)	3/152.0 (29)	19.7 (6.4–61.2)	27.2 (20.4–36.4)
Total	43/1450 (225)	29.7 (20.4–38.4)	47/1620.4 (262)	29.0 (21.8–38.6)	10/342.2 (67)	29.2 (15.7–54.3)	29.3 (24.1–35.7)

Case = number of participants who developed incident diabetes. no. = number of participants in subgroup from the designated ethnicity background. py = person-years. y = years.

3 Diabetes incidence per 1000 person-years (95% CI) by obesity and metabolic syndrome among 554 Indigenous Australians in north Queensland

Predictors	Aboriginals (*n* = 225)	Torres Strait Islanders (*n* = 262)	Joint ethnicity (*n* = 67)	Total (*n* = 554)	Crude HR	Adjusted HR*
Abdominal obesity by WC (> 88 cm in women and > 102 cm in men)†						
No	22.3 (14.4–34.3)	7.1 (2.9–16.9)	6.8 (1.0–48.6)	14.8 (10.1–21.7)	1.0	1.0
Yes	42.7 (28.4–61.3)	45.1 (33.2–61.3)	45.9 (23.9–88.1)	44.4 (35.3–55.9)	3.1 (2.0–4.8)	3.4 (2.0–5.8)
Abdominal obesity by WHR (> 1.0 in men and > 0.85 in women)†						
No	19.2 (11.6–31.9)	16.3 (10.0–26.6)	5.6 (0.8–39.5)	16.4 (11.7–23.3)	1.0	1.0
Yes	42.3 (29.2–61.2)	47.2 (33.0–67.5)	55.4 (28.8–106.5)	45.9 (36.1–58.3)	2.9 (1.9–4.4)	3.1 (1.9–5.2)
Obesity by body mass index						
No (< 30 kg/m^2)	25.6 (17.5–36.2)	13.1 (6.6–23.4)	10.6 (26.4–42.2)	19.7 (14.6–26.4)	1.0	1.0
Yes (≥ 30 kg/m^2)	50.1 (26.2–86.1)	43.9 (30.6–60.8)	52.3 (26.2–104.6)	46.2 (35.4–60.4)	2.4 (1.6–3.6)	2.7 (1.7–4.2)
Obesity by metabolic syndrome‡						
No	20.0 (12.9–31.0)	17.6 (11.1–27.9)	11.4 (3.7–35.4)	18.0 (13.2–24.4)	1.0	1.0
Yes	51.3 (34.1–77.2)	48.0 (33.4–69.1)	87.6 (41.8–183.8)	52.1 (40.4–67.3)	3.0 (2.0–4.4)	2.4 (1.6–3.7)

HR = hazard ratio. WC = waist circumference. WHR = waist-to-hip ratio.
* Adjusted for age, sex, ethnicity, smoking, alcohol-drinking and physical activity. † Abdominal obesity defined with World Health Organization WC and WHR criteria.[16] ‡ Metabolic syndrome defined by International Diabetes Federation criteria.[12]

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[10] 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).[5]

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.[15] 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 "hypertriglyeridaemic waist") has been found to predict high risk of coronary artery disease in populations of European ancestry.[16] 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).[17] 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.[18] 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.[19]

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 suggest potential selection bias and possible unmeasured confounding. Also, central obesity was estimated from WC, which is a crude measure and cannot distinguish between visceral, subcutaneous and other loci and types of fat accumulation, which may have different metabolic effects and different relationships to free fatty acid metabolism between individuals and groups. Despite this, it has been clear for some time that there are significant ethnic differences in "cut-offs" for obesity and overweight categories which predict metabolic and cardiovascular risk, and different cut-off points have been suggested for specific groups.[20] A recent study across 52 countries found WHR a better predictor of myocardial infarction attributable to obesity than BMI.[21]

Some advantages of this study include the use of measured, rather than self-reported, variables. The role of disordered triglyceride metabolism 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.

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COMPETING INTERESTS

None identified.

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