Diabetes Incidence in an Australian Aboriginal Population

An 8-year follow-up study

MARK DANIEL, PHD
KEVIN G. ROWLEY, PHD
ROBYN MCDERMOTT, MBBS
ARUL MYLVAGANAM, PHD
KERIN O'DEA, PHD

OBJECTIVE — To examine prospectively the association between age, BMI, and subsequent incidence of type 2 diabetes in Australian aboriginal people.

RESEARCH DESIGN AND METHODS — We performed a stratified analysis of incidence data from a community-based longitudinal study. Measures included fasting and 2-h postload glucose concentrations, and BMI, stratified into four categories. Subjects were 882 male and female participants in diabetes screening initiatives in two remote Australian aboriginal communities, free from diabetes at baseline, ages 15–77 years.

RESULTS — There were 46 incident cases of diabetes over 2,808 person-years of follow-up. BMI modified strongly the sex- and community-adjusted association between age and diabetes incidence ($P < 0.001$). Adjusted for age, sex, and community, the population diabetes incidence rate was 20.3 cases/1,000 person-years, with BMI-specific rates of 10.7–47.2 cases/1,000 person-years, and relative risks (95% CI) for BMI strata beyond the reference category (<25 kg/m^2) of 3.3 (1.5–7.0), 2.7 (1.1–6.8), and 4.4 (1.7–11.6), respectively. The population's attributable risk (95% CI) associated with BMI beyond the reference category was 70.1% (58.1–82.4).

CONCLUSIONS — BMI-specific diabetes incidence rates in Australian aboriginal people are among the highest in the world. Diabetes incidence in the lowest BMI category (10.7 cases/1,000 person-years) is two to five times greater than corresponding rates for non-aboriginal populations. An urgent need exists to prevent weight gain associated with diabetes. Further study is required to determine for aboriginal people an optimal range of BMI, likely lower than that suggested for non-aboriginal populations.

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Type 2 diabetes and its sequelae, such as cardiovascular and renal diseases, are major causes of premature mortality in Australian aboriginal populations (1). While ~2–5% of the non-aboriginal population in Australia has type 2 diabetes (2), the prevalence of diabetes among many Australian aboriginal populations is much higher. Rates of 15–25% are common (2).

Obesity is considered the strongest nonmetabolic risk factor for type 2 diabetes (3,4). We have reported previously for aboriginal populations a strong linear age-adjusted relationship between BMI and diabetes risk (5) and high insulin and postload glucose concentrations (6). The prevalence of overweight and obesity among Australian aboriginal populations is related to degree of Westernization (5,7) together with inappropriate diet and low physical activity (8). Obesity is increasing in indigenous populations in Australia and elsewhere (9,10), especially among women (11,12).

In populations of European ancestry, a monotonic relationship holds for BMI and the incidence of type 2 diabetes. The Nurses' Health Study found a strong linear association between diabetes risk and BMI >25 kg/m^2 (13), in alignment with earlier work indicating that BMI of 20–25 kg/m^2 is associated with low mortality in both men and women (14). Such studies are the basis of new clinical guidelines defining overweight as BMI 25–29.9 kg/m^2 and obesity as BMI ≥30 kg/m^2 (15). Whether these guidelines have utility for indigenous populations is unclear. BMI is a proxy for body fat, and the relation between adiposity and BMI is population-specific (16,17). Preliminary data suggest that a more healthful range of BMI is lower for aboriginal than for non-aboriginal people (18). For indigenous Australians, as for Native Americans (19), it is important to know how obesity modifies the risk of developing diabetes.

Data indicating time trends for diabetes prevalence in Australian aboriginal people have not been published, and the incidence of diabetes is unknown. Further, the relation of BMI to the risk of developing diabetes, and the potentially modifying effects of age and sex, have not been examined for Australian aboriginal populations. Such information is important for identification of optimal healthful BMI ranges for Aborigines and for public health interventions to stem the epidemic of noncommunicable diseases among indigenous populations. We report here the results of an 8-year follow-up of a cohort of aboriginal people in central Australia. The focus of our analysis is diabetes incidence and variation in risk by BMI and age.

From the Department of Health Behavior and Health Education (M.D.), School of Public Health, University of North Carolina, Chapel Hill, North Carolina; the Centre for Population Health and Nutrition (K.G.R., K.O.), Institute of Public Health and Health Services Research, Monash University, Melbourne, Victoria; the Tropical Public Health Unit (R.M.), Queensland Health, Cairns, Queensland; and the Department of Epidemiology and Biostatistics (A.M.), School of Public Health, Curtin University of Technology, Perth, Western Australia, Australia.

Address correspondence and reprint requests to Dr. Mark Daniel, Department of Health Behavior and Health Education, School of Public Health, University of North Carolina, CB #7400, Rosenau Hall, Room 302, Chapel Hill, NC 27599. E-mail: mark.daniel@sph.unc.edu.

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A table elsewhere in this issue shows conventional and Système International (SI) units and conversion factors for many substances.

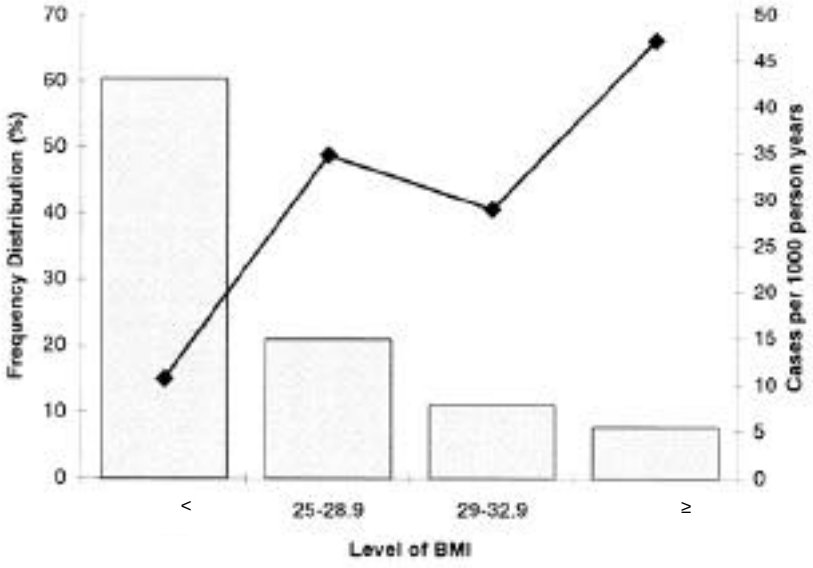

Figure 1—*Frequency distribution of baseline BMI (kg/m²) (bars) and age, sex, and community-adjusted incidence of diabetes (—◆—) for Australian aboriginal people followed for up to 8 years (1987–1995)* (n = *464*).

RESEARCH DESIGN AND METHODS

Subjects

Two remote central Australian aboriginal communities were surveyed. Subjects were volunteers for diabetes and cardiovascular disease diagnostic and risk factor screening initiatives. Under agreement with the two communities involved, we refer to them as "A" and "B," respectively. Baseline measures were taken in 1987 for community A and in 1988 for community B; both communities were followed up in 1995. Details of the population and recruitment methods have been published previously (20,21). Briefly, the population we classified as adult men and women (aged ≥15 years) was identified by household census. Community meetings to explain the survey facilitated recruitment into the study. The proportion of adults participating in baseline screening was 83.1% (*n* = 530) for community A and 80.3% (*n* = 437) in community B. Response rates did not differ between communities, nor did response rates vary by sex between communities. Across communities, however, response rates were greater for women (86.1%) than for men (79.7%).

Classification of diabetes at baseline and follow-up was based on 2-h oral glucose tolerance tests with 75-g load (2-h glucose ≥11.1 mmol/l) (22). Of 967 people screened at baseline, 85 were classified as having diabetes. The prevalence of diabetes at baseline was 12% for community A; 9% for community B. We report on people free from diabetes at baseline, a cohort of 882.

The Alice Springs Institutional Ethics Committee and Deakin University, Melbourne, Australia (the institution responsible for the study throughout the data collection phases), granted ethical approval. All participants provided their informed written consent.

Blood samples and analytic methodology

For screening purposes, participants provided a fasting (8–12 h) venous blood sample collected in fluoride heparin anticoagulant. Plasma was obtained on-site by centrifugation at 2,500 rpm. Samples were frozen for transport to Melbourne for analysis. Glucose concentrations were determined from fluoride heparin plasma samples. Two-hour glucose concentrations were determined from blood samples drawn 2 h after consumption of a 75-g carbohydrate load, taken after fasting samples were drawn. Glucose concentrations were assessed using standard enzymatic methods (Boehringer-Mannheim, Sydney), with coefficients of variation <2.2% for all assays.

Anthropometric measurements

Participants wore light clothing, with footwear removed. Weight was measured to the nearest 0.1 kg using a digital electronic scale. Height was measured to the nearest 0.1 cm using an anthropometer. BMI was calculated as weight divided by height squared (kg/m²).

Statistical analysis

In risk estimation, we used Poisson regression and linear additive excess relative risk models based on person-years at risk. This method yielded results similar to cumulative incidence in relating diabetes risk to BMI. Stratum-specific diabetes incident cases were numerators and person-years denominators, for morbidity rates. EPICURE software (23) was used for regression analyses. Person-years at risk were allocated in a time-dependent manner to cross-classified strata using an SAS algorithm developed by one of the authors (24). Individual length of follow-up was defined as the interval between oral glucose tolerance tests at baseline and follow-up. This method will have underestimated the true incidence rate, as an assumption is that all events occurred at follow-up when in fact the true time of onset was at some point during the interim period.

Potential confounders—age, sex, and community—were stratified into three age-groups (15–24, 25–34, and ≥35 years) and the two communities and sexes. Incidence and risk estimates were adjusted for age-group, sex, and community. Waist-to-hip ratio was not included in the regression model because waist and hip girths were not assessed at baseline. Exact *P* values and 95% CIs are reported. Hypothesis tests were two-tailed. Statistical significance was set at 0.05.

We used BMI categories consistent with those used by Colditz et al. (13) to evaluate the relation of BMI (<25, 25–28.9, 29–32.9, and ≥33 kg/m²) to the risk of developing diabetes. These categories were appropriate given the distribution of BMI at baseline for people followed over time (Fig. 1). The potentially modifying effects of age at follow up were assessed by examining risk estimates across age and BMI strata, controlling for sex and community.

Given small sample sizes and sparse data for cross-classifications of age and BMI, we have not age-standardized rates to a reference population. Instead, results are discussed in relation to other populations in terms of ranges of age and BMI.

RESULTS — Of 882 people free from diabetes at baseline, 52.6% (*n* = 464) were followed up in 1995. Reasons for nonparticipation in 1995 were: *1*) moved from community, 44.0% (*n* = 184); *2*) absent from community at time of screening, 2.6%

(*n* = 11); *3*) died, 19.1% (*n* = 80); and *4*) nonspecified, 34.2% (*n* = 143). In a comparison (mean ± SEM) at baseline of those successfully followed with those lost to follow-up, there were no differences in age (31.7 ± 0.7 vs. 31.8 ± 0.8 years), BMI (24.3 ± 0.3 vs. 23.9 ± 0.3 kg/m^2), fasting glucose (4.73 ± 0.03 vs. 4.74 ± 0.03 mmol/l), or 2-h glucose (5.86 ± 0.08 vs. 5.67 ± 0.09 mmol/l). Only the ratio of men to women differed (P = 0.008): 41:59 for people followed up; 50:50 for those lost to follow-up.

For people followed up the distribution of sex did not differ between the two communities (Table 1) and was consistent with that of aboriginal communities in central Australia (57% women, 43% men) (25). Mean age (range: 15–77 years) was lower by five years for community A than for community B, reflecting relatively fewer individuals <25 years in community B (Table 1). For the two communities together, the distribution of age among people followed up was similar to that for aboriginal communities in central Australia, ~37% aged <25 years, 25% aged 25–34 years, and 38% aged ≥35 years (25).

There were 46 incident cases of diabetes over 2,808 person-years (8 calendar years) of follow-up among 464 people. The population-weighted diabetes incidence rate, adjusted for age, sex, and community, was 20.3 cases/1,000 person-years. Likelihood ratio tests indicated that the risk of developing diabetes was related to community (P = 0.003) as well as age-group after accounting for community (P < 0.000). Sex was not related to risk of developing diabetes, after accounting for community and age-group (P = 0.339).

The interaction of age and BMI on the sex and community–adjusted risk of developing diabetes (P < 0.001), relative to the reference category of age 15–24 years and BMI <25 kg/m^2, is shown in Table 2. BMI >25 kg/m^2 modified substantially the risk of diabetes associated with each age-group. Relative to the reference category, risks were statistically significant for cross-classifications including and beyond age 25–34 years and BMI 25–28.9 kg/m^2.

The range of the incidence of diabetes per 1,000 person-years observation, adjusted for age, sex, and community, was 10.7–47.2 across BMI categories (Fig. 1). A strong linear trend was apparent, with greater incidence associated with greater BMI (P < 0.001). Relative risks associated with BMI strata beyond the reference category (<25 kg/m^2) were, respectively, 3.3 (P < 0.001), 2.7 (P = 0.023), and 4.4 (P = 0.001); however, the test for trend in relative risk was not statistically significant (P = 0.615). Age-adjusted rates of change in fasting glucose were statistically significant for BMI strata ≥25 kg/m^2, ranging from 0.78 to 2.25 mmol/l per 10 years; for 2-h glucose, rates of change were statistically significant for all BMI strata, ranging from 1.10 to 3.31 mmol/l per 10 years.

As a proportion of the population incidence rate, the population's attributable risk percentage for the age, sex, and community—adjusted incidence rate associated with BMI beyond the reference category (BMI <25 kg/m^2), for the corresponding population structure, ranged from 63.1 to 73.6% between the 2nd and 4th BMI strata (Table 2). The overall population attributable risk (95% CI), adjusted for age, sex, and community, was 70.1% (58.1–82.4).

Table 1—*Baseline characteristics of study participants initially free of diabetes in two central Australian aboriginal communities followed up to 8 years (1987–1995)*

	Community A	Community B
n	224	240
Age (15–77 years)	29.6 (26.9–32.3)	34.8 (32.0–37.6)
Age distribution (%)		
<25 years	54.4 (47.7–61.1)	34.9 (29.0–41.4)
25–34.9 years	17.8 (13.1–23.5)	24.1 (18.9–30.1)
≥35 years	27.8 (21.9–34.0)	41.0 (34.9–47.8)
Sex (%)		
Male	43.3 (36.9–49.9)	39.2 (32.9–45.6)
Female	56.7 (49.9–63.3)	60.8 (54.8–67.5)
Weight (kg)	73.7 (70.3–77.2)	62.8 (60.2–65.4)
Height (cm)	167.4 (165.9–168.9)	166.5 (165.1–167.9)
BMI (kg/m^2)	26.3 (25.1–27.5)	22.6 (21.8–23.4)

Data are *n* or means (95% CI).

CONCLUSIONS — This is the first report ever on diabetes incidence in Australian Aborigines. Adjusted for age, sex, and community, the BMI-specific diabetes incidence rates (11–47 cases/1,000 person-years) in the central Australian population assessed are among the highest in the world, corresponding closely with age- and sex-adjusted BMI-specific rates for Pima Indians (26). There are major cultural and genetic differences between Pima Indians and Australian Aborigines. Both populations, however, have only relatively recently undergone "Westernization"—the rapid process by which indigenous societies in industrialized countries have been diluted by and made dependent on modern or "Western" ways of living and external resources inconsistent with traditional patterns (27). We have addressed elsewhere the impact of social disadvantage, dependence living, and low autonomy on diabetes and its risk factors, including obesity (28).

The relationship between diabetes incidence and BMI in our study population differs from that of the mostly European-derived (female) population of the U.S. Nurses' Health Study, where diabetes incidence increased with BMI up to 35 kg/m^2. In contrast, for the aboriginal cohort, diabetes incidence jumped from 10.7 cases/1,000 person-years for BMI <25 kg/m^2 to an average (person-years–weighted) of 35.6 cases/1,000 person-years for BMI strata ≥25 kg/m^2 (Fig. 1). Thus, while the test for trend for increasing incidence across BMI strata was significant (P < 0.001), the test for trend in risk relative to the reference category of BMI <25 kg/m^2 was not. A major difference between our study and the Nurses' Health Study is that we screened for diabetes and did not define status from self-reports. The relevant issue, however, is the dissimilar relationship between BMI and diabetes incidence, not differentials in incidence that, to a degree, can be explained by different assessment strategies. We observed no effect of sex on the incidence of diabetes, consistent with a recent analysis of six prospective studies of predictors of diabetes (4).

In an analysis of 1,972 men screened for diabetes over 25 years in the Normative Aging Study cohort, Cassano et al. (29) reported a nearly twofold greater age-adjusted risk of developing diabetes for men in the upper tertile for BMI (≥26.9

Table 2—*Adjusted relative risk of developing diabetes by baseline BMI and age at follow-up, and BMI-specific adjusted incidence, relative risk, and attributable risk percentage of developing diabetes, for Australian aboriginal people followed up to 8 years (1987–1995)* (n = 464)

BMI (kg/m²)	Relative risk (95% CI)			Adjusted incidence/ 1,000 person-years	Adjusted relative risk (95% CI)	Attributable risk % (95% CI)
	Aged 15–24 years	Aged 25–34 years	Aged ≥35 years			
<25	1.0 (referent)	7.0 (0.8–60.3)	6.3 (0.7–54.8)	10.7	1.0 (referent)	—
25–28.9	3.5 (0.2–55.8)	22.6 (2.7–182.1)	25.4 (3.2–199.4)	34.9	3.3 (1.5–7.0)	63.1 (43.4–83.6)
29–32.9	—	15.9 (1.4–175.3)	22.9 (2.8–185.9)	29.0	2.7 (1.1–6.8)	79.9 (57.8–96.7)
≥33	—	27.6 (2.8–271.7)	37.9 (4.5–321.1)	47.2	4.4 (1.7–11.6)	73.6 (52.9–90.0)

Relative risk for age-groups is adjusted for sex and community; adjusted relative risk is adjusted for age, sex, and community; attributable risk is adjusted for age, sex, and community and represents population attributable risk percentage.

kg/m²) over that for men in the lowest tertile for BMI (<24.6 kg/m²). Njølstad et al. (30) reported that for 11,654 men and women followed up over 12 years in the Finnmark Study, the age-adjusted risk of diabetes was ~1.1 for people in the 2nd and 3rd quartiles for BMI (23.2–25.0 and 25.1–27.0 kg/m², respectively), and ~10 for those in the highest quartile for BMI (≥27.1 kg/m²), relative to people in the lowest quartile for BMI (<23.2 kg/m²). For the aboriginal cohort in this study, the risk of developing diabetes was greater by 3.3 for people with BMIs of 25–28.9 kg/m², and greater by 4.4 for people in the highest BMI category (≥33 kg/m²), than for people in the lowest BMI category (<25 kg/m²) (Table 2). This contrast highlights the greater risk of diabetes for Aborigines with small elevations above the recommended healthful limit of BMI <25 kg/m². Relative to non-aboriginal populations, a lesser relative risk of diabetes for obese (not overweight) aboriginal people reflects the greater incidence of diabetes in aboriginal populations at low levels of BMI.

In the Normative Aging Study, diabetes incidence was <4 cases/1,000 person-years for men in the lowest tertile for BMI and ~10 cases/1,000 person-years for men in the upper tertile for BMI. BMI-specific incidence rates were not reported for men and women in the Finnmark study. Participants in these studies were substantially older at baseline (mean age: mid-40s) than the aboriginal cohort in this study (mean age: early 30s), and age clearly modifies the risk associated with BMI (Table 2). The greatest incidence of 10 cases/1,000 person-years for men in the highest tertile for BMI in the Normative Aging Study is similar to the lowest incidence seen for aboriginal people in this study in the lowest BMI category (<25 kg/m²): 10.7 cases/1,000 person-years. Knowler et al. (26) reported a similar incidence of ~10 cases/1,000 person-years for Pima Indians (men and women) with BMIs in the same range. It is of considerable importance that the age-adjusted incidence of diabetes is ~10 cases/1,000 person-years in Australian Aborigines and Pima Indians at what is considered (for U.S. and European populations) to be a "healthful" BMI (<25 kg/m²).

The relationship between body composition and BMI is different for aboriginal people than for Europeans. For any given BMI, aboriginal people have more body fat than Europeans (31). The impact of greater adiposity is amplified by body fat distribution. Body fat is deposited preferentially around the abdomen in aboriginal men and women, yielding a greater risk of diabetes for any level of adiposity (6,32). Risk of diabetes is further compounded by poor dietary quality (e.g., high total and saturated fat intake, low intake of complex carbohydrate) (8,33), and low levels of physical activity (34). Thus, BMI within and slightly above the "healthful" range for American and European populations is associated with greater risk of diabetes among aboriginal people. For African-Americans relative to Caucasian Americans, the risk of diabetes is also greater at any level of BMI (35), particularly low BMI (36).

We were unable to evaluate the risk of diabetes relative to a reference category with an upper cutpoint <24.9 kg/m², because of sparseness of data. Nevertheless, we did determine diabetes incidence for a level of BMI lower than that reported in Table 2. At BMI <22 kg/m² there was just one incident case, corresponding to an incidence of 0.9 cases/1,000 person-years. For BMI in the range of 22–24.9 kg/m², we observed 10 incident cases, for an incidence of 15.8 cases/1,000 person-years. The age-adjusted risk (95% CI) of diabetes for people with BMI of 22–24.9 kg/m², relative to people with BMI <22 kg/m², was 18.4 (3.0–409.1). This estimate suffers an unstable reference denominator; however, it supports the notion that, for Aborigines, BMI <22 kg/m² may be more appropriately considered an optimal healthful range than BMI <25 kg/m².

Rapid early weight gain leading to overweight and obesity is likely a strong determinant of diabetes incidence in the aboriginal population in this study. For aboriginal children and adolescents followed up from 1989 to 1994 in the remote Kimberley region of Western Australia, the proportion of youth who were overweight grew by 15% (10). A review of nine studies of adults in aboriginal communities throughout Australia cited prevalence of obesity as high as 53% (5). Rapid weight gain and duration of obesity are associated with a high incidence of diabetes among Pima Indians (37,38).

As given by the 95% confidence bounds for the population attributable risk percentage for the aboriginal population surveyed here, 58.1–82.4% of cases of diabetes might be prevented by preventing gains in BMI beyond 25 kg/m². This estimate is more than that determined by Manson and Spelsberg, 50–75%, based on a review of the epidemiological and biological evidence for prevention of diabetes by control of obesity (39). The prevalence of obesity and its contribution to diabetes is high for aboriginal peoples, however (5). Both prevention and reduction of obesity can prevent development or ameliorate the impact of metabolic risk factors as well as the actual risk of developing diabetes (37,40). Therefore, we see weight reduction as a priority public health issue for Australian aboriginal populations. Weight reduction strategies must reach beyond purely individual responsibility for health to include enabling environmental supports (41). Especially for disadvantaged aboriginal populations, an individualistic emphasis on diabetes prevention is coun-

terproductive in diverting attention from systemic determinants of lifestyles, thus perpetuating social risk conditions supporting ill health (28).

Relative to other high-risk populations also screened using World Health Organization criteria, the overall incidence of diabetes for Australian Aborigines ≥15 years of age in this study (20.3 cases/1,000 person-years) is lower than rates of >30 cases/1,000 person-years for Pima Indians ≥15 years of age (26) but aligned with rates of 20–24.9 cases/1,000 person-years for Nauruans ≥20 years of age (42), Wanigelas 15–69 years of age in Papua New Guinea (43), and Indians 35–69 years of age in Trinidad (44). Diabetes incidence in this study is greater than rates of 15–19.9 cases/1,000 person-years for urban Samoans ≥20 years of age (43), Mauritian Indians 25–74 years of age (43), and Japanese Americans ≥40 years of age (45). Intermediate rates from 5–14.9 cases/1,000 person-years define Creole and Chinese Mauritians aged 25–74 years (43), Africans aged 35–69 years in Trinidad (44), Maltese aged ≥29 years (46), Mexican Americans aged 25–64 years (47), and rural Samoans aged ≥20 years (43). By comparison, diabetes incidence is <2.5 cases/1,000 person-years for European (30,48) and U.S. (13,49,50) populations.

Limitations of this study do not include possible bias in blood test results if participants failed to fast before blood samples were drawn. Diagnosis of diabetes in all cases was made on the basis of plasma glucose concentrations 2 h after ingestion of a 75-g glucose load. Measurement error in blood and anthropometric measures is possible, and would bias relative risk estimates toward the null. That more women than men participated in follow-up examinations is unlikely to have biased results, given the lack of association of sex with the risk of developing diabetes.

The results are probably generalizable, to some degree, to aboriginal populations beyond central Australia. Though not probability-based, the sample was representative of indigenous people of central Australia in terms of the distribution of age and sex and the prevalence of diabetes. Australian aboriginal populations are heterogeneous and it is likely that longitudinal studies in other parts of the continent would generate similar findings. Cross-sectional data from aboriginal populations from different parts of northern and central Australia indicate a reasonably consistent age-adjusted relationship between BMI and the prevalence of diabetes (21,51–53). Replication of our analyses in other indigenous Australian populations may further clarify the influence of BMI on the risk of developing diabetes. It is particularly important to establish an optimal healthful range of BMI for Australian aboriginal people.

In conclusion, we examined prospectively the relations between age, BMI, and the incidence of diabetes in Australian aboriginal people. BMI strongly modified the association between age and diabetes incidence. As for other high-risk populations, we observed a high incidence of diabetes among people aged <35 years. Sex was not associated with risk of developing diabetes. Adjusted for age, sex, and community, the overall population-weighted diabetes incidence rate was 20.3 cases/1,000 person-years, with BMI-specific rates of 10.7–47.2 cases/1,000 person-years. Of particular importance, BMI-specific incidence rates corresponded closely with those for Pima Indians, the highest in the world. The high incidence of diabetes (10.7 cases/1,000 person-years) in the lowest BMI category (<25 kg/m^2) is two to five times greater than that for non-aboriginal populations. Preventing gains in BMI beyond 25 kg/m^2 could prevent an average of 70.1% of cases of diabetes. An urgent need exists for community-based initiatives to prevent and reduce weight gain that may become associated with diabetes. Further study is required to define for aboriginal people an optimal range of BMI, inasmuch as the upper recommended "healthful" limit of 24.9 kg/m^2 is too high for this population.

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