Glucose Indices, Health Behaviors, and Incidence of Diabetes in Australia

The Australian Diabetes, Obesity and Lifestyle Study

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OBJECTIVE — This national, population-based study reports diabetes incidence based on oral glucose tolerance tests (OGTTs) and identifies risk factors for diabetes in Australians.

RESEARCH DESIGN AND METHODS — The Australian Diabetes, Obesity and Lifestyle Study followed-up 5,842 participants over 5 years. Normal glycemia, impaired fasting glucose (IFG), impaired glucose tolerance (IGT), and diabetes were defined using World Health Organization criteria.

RESULTS — Age-standardized annual incidence of diabetes for men and women was 0.8% (95% CI 0.6–0.9) and 0.7% (0.5–0.8), respectively. The annual incidence was 0.2% (0.2–0.3), 2.6% (1.8–3.4), and 3.5% (2.9–4.2) among those with normal glycemia, IFG, and IGT, respectively, at baseline. Among those with IFG, the incidence was significantly higher in women (4.0 vs. 2.0%), while among those with IGT, it was significantly higher in men (4.4 vs. 2.9%). Using multivariate logistic regression, hypertension (odds ratio 1.64 [95% CI 1.17–2.28]), hypertriglyceridemia (1.46 [1.05–2.02]), log fasting plasma glucose (odds ratio per 1 SD 5.25 [95% CI 3.98–6.92]), waist circumference (1.26 [1.08–1.48]), smoking (1.70 [96% CI 1.11–2.63]), physical inactivity (1.56 [1.12–2.16]), family history of diabetes (1.82 [1.30–2.52]), and low education level (1.85 [1.04–3.31]) were associated with incident diabetes. In age- and sex-adjusted models, A1C was a predictor of diabetes in the whole population, in those with normal glycemia, and in those with IGT or IFG.

CONCLUSIONS — Diabetes incidence is 10–20 times greater in those with IGT or IFG than those with normal glycemia. Measures of glycemia, A1C, metabolic syndrome components, education level, smoking, and physical inactivity are risk factors for diabetes.

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The rapidly increasing prevalence of type 2 diabetes (1) and the potential for prevention through lifestyle change and behavioral measures such as weight reduction and exercise (2–4) mandate a more complete understanding of the epidemiology and natural history of diabetes, impaired fasting glycemia (IFG), and impaired glucose tolerance (IGT). Several longitudinal observational studies (5–9) and clinical trials (2,3) have provided inferences about incidence and etiology, with obesity, physical inactivity, and metabolic syndrome components emerging as the major risk factors (5,10–14). In the Brisighella Heart Study (10), a population-based study of 2,939 individuals followed for 8 years, BMI and hyperglycemia were independent risk factors for diabetes, while in the Bruneck Study (5), insulin resistance also significantly predicted diabetes. In Pima Indians, body size, lipids, insulinemia (13), and physical activity (15) were risk factors for diabetes. In studies reporting incidence, rates ranged from 7.6 per 1,000 person-years in low- to moderate-risk populations from Italy (5) to 21 per 1,000 person-years in a high-risk population such as Mauritians (16). However, the generalizability of these findings is limited, particularly in relation to the absolute incidence rates, since they have been based on populations that are restricted by age, ethnicity, occupation, or geography (7,12). Such incidence data can only be derived from following large, population-based cohorts in which diabetes is defined by the oral glucose tolerance test (OGTT). Indeed, there are no data on diabetes incidence from longitudinal, national, population-based studies conducted in a developed nation and using the OGTT.

The Australian Diabetes, Obesity and Lifestyle Study (AusDiab) recruited a national, population-based sample of Australian adults and included an OGTT at baseline and at the 5-year follow-up. It provides a unique opportunity to describe the incidence and risk factors for diabetes for Australian adults.

RESEARCH DESIGN AND METHODS

— The AusDiab baseline study methods and response rates are described in detail elsewhere (17,18). In brief, the baseline study was a cross-sectional, national, population-based survey of 11,247 adults aged ≥25 years in 1999–2000. Over 85% of the sample was from an Australian, New Zealand, or British background. A stratified cluster sample was drawn from 42 randomly selected census collector districts across Australia. Information was collected using a brief household interview, followed by a bio-

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Abbreviations: 2hPG, 2-h plasma glucose; AusDiab, Australian Diabetes, Obesity, and Lifestyle Study; FPG, fasting plasma glucose; IFG, impaired fasting glucose; IGT, impaired glucose tolerance; OGTT, oral glucose tolerance test.

medical examination. The response rate to the baseline biomedical examination was 55%. In 2004–2005, all living eligible participants were invited to attend follow-up. Those who were considered ineligible included those who refused further contact ($n = 128$), were deceased ($n = 310$), had moved overseas or into a nursing facility classified for high care, or had a terminal illness ($n = 21$).

At baseline and follow-up, the physical examination included blood samples, anthropometric measurements, and questionnaires. All participants except for those currently receiving treatment for diabetes or who were pregnant underwent a standard 75-g OGTT (19). In 1999–2000, fasting plasma glucose (FPG) and 2-h plasma glucose (2hPG) levels were determined by a glucose oxidase method using an Olympus AU600 automated analyser (Olympus Optical, Tokyo, Japan), and in 2004–2005 a spectrophotometric-hexokinase method utilizing a Roche Modular (Roche Diagnostics, Indianapolis, IN) was used. To compare results from the two assays, 195 stored baseline FPG samples and 171 2hPG samples were analyzed on the Roche assay used at follow-up. The median (10th–90th percentile) difference between the pairs of FPG values was 0.2 mmol/l (0.1–0.4), and the median (10th–90th percentile) difference between the pairs of 2hPG values was 0.2 mmol/l (0.0–0.4), with higher values from the baseline assay. Total glycated hemoglobin analysis used high-performance liquid chromatography (Bio-Rad Variant Hemoglobin Testing System; Bio-Rad, Hercules, CA) with standardized conversion to A1C values. Blood pressure was measured using Dinamap or a standard mercury sphygmomanometer with appropriate adjustments made as previously described (20). The study was approved by the International Diabetes Institute Ethics Committee.

Definition of diabetes, IFG, and IGT

Glucose tolerance status was classified according to the 1999 World Health Organization criteria (19). Diabetes was classified on the basis of FPG ≥7.0 mmol/l or 2hPG ≥11.1 mmol/l or current treatment with insulin or oral hypoglycemic agents. For those not reporting treatment for diabetes, FPG <7.0 mmol/l and 2hPG ≥7.8 mmol/l but <11.1 mmol/l indicated IGT, FPG 6.1–6.9 mmol/l and 2hPG <7.8 mmol/l indicated IFG, and both FPG <6.1 mmol/l and 2hPG <7.8 mmol/l indicated normal glycemia. An incident case of diabetes was defined as an individual who had normal glycemia, IFG, or IGT at baseline but had developed diabetes at follow-up. Incident cases of IFG were defined as people who had normal glycemia at baseline but had developed IFG at follow-up. Incident cases of IGT were defined as people who had normal glycemia or IFG at baseline but had developed IGT at follow-up.

Risk factors

Hypertriglyceridemia was defined as serum triglycerides ≥2.0 mmol/l, and abnormal HDL cholesterol was defined as HDL cholesterol <1.03 and <1.29 mmol/l for men and women, respectively. Hypertension was defined as having systolic blood pressure ≥140 mmHg or diastolic blood pressure ≥90 mmHg or reporting antihypertensive medication use.

Data on education, smoking, leisure-time physical activity, and family history of diabetes were collected by an interviewer-administered questionnaire. Education was classified into four categories: *1*) university/further education, *2*) completed secondary school, *3*) some secondary school, or *4*) primary school/never attended school. Smoking history (current smoker, past smoker, or never smoked) was collected using a questionnaire on tobacco use, which has been validated in Australian adults (21). Total leisure-time physical activity reported for the previous week (none, insufficient: 1–149 min; sufficient: ≥150 min) was measured using the Active Australia questionnaire, which is the standard instrument for population surveillance and has acceptable reliability (22,23). Family history of diabetes was defined as a mother or father being diagnosed as having diabetes.

Statistical analysis

Five-year cumulative incidence was calculated and standardized to the 1998 Australian population using the direct method (7). In brief, age- and sex-specific 5-year cumulative incidences of diabetes were applied to the equivalent age and sex strata from the Australian population of 1998, who were free of diabetes. The 1998 Australian diabetes-free population was calculated by applying age- and sex-specific diabetes prevalence estimates from the baseline AusDiab data to the 1998 Australian population and subtracting the diabetic population from the total population to give a nondiabetic population. The incidence of IFG and IGT were age and sex standardized using the same method but applying the relevant IFG- and IGT-free populations rather than the diabetes-free population. Annual incidence (% per year) was calculated from the 5-year cumulative incidence by applying the following formula: $[-\ln(1 - S)]/t$; where S is the proportion of new cases over t years and t equals the time of follow-up. Baseline characteristics of the attendees ($n = 6,537$) were compared with those of the nonattendees ($n = 4,710$) with an independent t test, Mann-Whitney U test, or Pearson's χ^2 test, where appropriate. The incidence of diabetes is restricted to those who did not have diabetes at baseline ($n = 5,842$) but developed diabetes at follow-up.

Risk factors for diabetes were identified by multivariate logistic regression. The covariates included in the model were age, sex, waist circumference, smoking status, FPG, 2hPG, A1C, hypertension, physical activity, education level, family history of diabetes, total cholesterol, HDL cholesterol, and triglycerides. Triglycerides, 2hPG, FPG, and A1C were log transformed. Since waist circumference was a better predictor of diabetes than BMI, it was used as the measure of adiposity in the model. The risk of diabetes associated with age was calculated per 10 years, and for waist circumference, FPG, 2hPG, and A1C, the risk of diabetes was estimated per 1 SD. In models comparing the incremental value of 2hPG and A1C to FPG, the test procedure was based on maximum likelihood estimators, Akaike's information criterion (AIC statistic), and the area under the receiver-operating characteristic curve. Analyses were conducted using Stata statistical software version 9.2 (StataCorp, College Station, TX).

RESULTS

Response rates

Of 10,788 participants eligible for testing in 2004–2005, 6,400 (59.3%) attended the full physical examination. A further 137 (1.3%) participants had blood and urine tests only, and another 2,261 (21.0%) completed a telephone questionnaire only. Thus, the response rate for the follow-up was 60.6% (6,537 of 10,788).

Compared with those who did not attend ($n = 4,710$), attendees ($n = 6,537$) were significantly less likely to be hypertensive, to have a lower level of education attainment, or to be smokers and had lower 2hPG, A1C, and smaller waist circumferences at baseline. Furthermore, a significantly larger proportion of nonattendees had abnormal triglyceride or

HDL cholesterol levels at baseline compared with attendees (data not shown).

Incidence of diabetes

Of the population at risk of diabetes (n = 5,842), 54.3% were female and 88.5% were of Australian, New Zealand, or British background. The mean (range) for age and BMI was 50.9 years (25–88) and 26.7 kg/m^2 (14.5–59.4), respectively. The baseline characteristics of those who developed diabetes and those who did not are described in Table 1. Table 2 describes the incidence of diabetes, IFG, and IGT. At follow-up, there were 224 new cases of diabetes. More men (4.4%) than women (3.4%) developed diabetes (P = 0.09). The incidence of diabetes among those with IFG was significantly higher in women than in men, while the opposite applied to those with IGT at baseline. Among those with IGT, the excess risk of diabetes among men (adjusted for age and waist circumference) was attenuated and became nonsignificant with adjustment for baseline FPG. Among those with IFG, the excess risk of diabetes among women (adjusted for age) was reduced and was no longer significant after adjustment for baseline waist circumference and was further attenuated, following adjustment for baseline 2hPG (data not shown).

When the fasting glucose values from follow-up were adjusted via a regression equation (derived from comparing the 195 samples from baseline that were retested at follow-up), 244 new cases of diabetes (compared with 224 new cases of diabetes observed in the main analysis) occurred over follow-up. The annual incidence of self-reported diabetes was 0.5% (95% CI 0.4–0.6) in the 6,537 (5,842 free of diabetes) attendees in the main analysis and 0.5% (0.4–0.7) in the 2,261 (1,990 free of diabetes) nonattendees who only completed health questionnaires. After adjusting for age and sex, the odds of self-reporting incident diabetes remained similar in these two groups (odds ratio 1.0 [95% CI 0.7–1.4]).

Table 3 shows that waist circumference, current smoking, primary school level of education only/never attended school, hypertension, family history of diabetes, FPG, and high triglycerides were positively associated with incident diabetes. An increased risk of diabetes was observed in those who did not achieve the public health recommendations for physical activity (the inactive and the insufficiently active), compared with those who achieved the recommended levels (sufficiently active: ≥150 min/week). Low HDL cholesterol was only associated with incident diabetes in adjusted models, which did not include triglycerides. Each of the three measures of glycemia had significant relationships with incident diabetes that were similar to each other in magnitude. These relationships remained significant among those with normal glycemia and among those with IFG or IGT (data not shown), with similar and significant results in men and women.

To determine the additional value of 2hPG and A1C to other risk factors, these parameters were incorporated into to a model comprising FPG, waist circumference, age, sex, triglycerides, hypertension, education level, family history of diabetes, physical activity category, and smoking status. The sequential addition of 2hPG and A1C significantly improved the maximum likelihood ratio (2hPG: χ^2 = 131.8, P < 0.001) (A1C: χ^2 = 48.1, P < 0.001) and reduced the AIC statistic of the model (data

Table 1—*Baseline characteristics according to diabetes status at follow-up: the AusDiab study*

	Diabetes status at follow-up		
	With diabetes	Without diabetes	*P* value
n	224	5618	
Male subjects (%)	51.3	45.0	<0.001
Age (years)	55.8 ± 12.0	50.7 ± 12.6	0.06
Waist circumference (cm)			
Men	104.1 ± 11.6	96.4 ± 10.6	<0.001
Women	92.7 ± 14.7	83.8 ± 12.4	<0.001
BMI (kg/m^2)			
Men	29.3 ± 0.4	26.9 ± 0.1	<0.001
Women	29.7 ± 0.6	26.3 ± 0.1	<0.001
Smoking status (%)			0.003
Nonsmoker	51.8	59.8	
Ex-smoker	32.6	29.1	
Current smoker	15.6	11.1	
Education (%)			<0.001
Never attended school or attended primary school only	10.7	4.2	
Some high school	38.4	35.1	
Completed high school	17.9	18.9	
University/technical and further education	33.0	42.0	
Physical activity (min/week) (%)			<0.001
Sufficient (≥150 min/week leisure-time physical activity)	40.7	54.4	
Insufficient (1–149 min/week leisure-time physical activity)	37.6	30.7	
Inactive (0 min/week leisure-time physical activity)	21.7	15.0	
Hypertension (%)*	54.6	27.6	<0.001
Family history of diabetes (%)	30.5	17.8	<0.001
FPG (mmol/l)	6.0 (5.5–6.4)	5.3 (5.0–5.6)	<0.001
2hPG (mmol/l)	8.0 (6.7–9.4)	5.7 (4.8–6.7)	<0.001
A1C (percent)	5.5 (5.2–5.7)	5.1 (4.9–5.3)	<0.001
Total cholesterol (mmol/l)	5.9 ± 1.0	5.6 ± 1.1	<0.001
Low HDL cholesterol (%)†	37.1	21.4	<0.001
Hypertriglyceridemia (%)‡	41.5	19.2	<0.001
Glucose tolerance status (%)			<0.001
Normal glycemia	25.9	82.9	
IFG	19.6	5.8	
IGT	54.5	11.3	

Data are means ± SD, median (25th–75th percentile), or percent. *Hypertension was defined as systolic blood pressure ≥140 mmHg or diastolic blood pressure ≥90 mmHg or reporting antihypertensive medication use. †Low HDL cholesterol was defined as HDL cholesterol <1.03 mmol/l for men and <1.29 mmol/l for women. ‡Hypertriglyceridemia was defined as triglycerides ≥2.0 mmol/l.

Table 2—*Incidence of diabetes, impaired fasting glucose and impaired glucose tolerance: the AusDiab study*

	Age- and sex-standardized incidence			
	Male subjects	Female subjects	*P* value*	Total
Incidence of diabetes according to baseline glucose tolerance status				
Whole population	0.8 (0.6–0.9)	0.7 (0.5–0.8)	0.09	0.7 (0.6–0.8)
Among normal glycemic subjects	0.2 (0.1–0.3)	0.2 (0.2–0.3)	0.91	0.2 (0.2–0.3)
Among IGT subjects	4.4 (3.3–5.6)	2.9 (2.1–3.7)	0.02	3.5 (2.9–4.2)
Among IFG subjects	2.0 (1.3–2.8)	4.0 (2.2–5.9)	0.03	2.6 (1.8–3.4)
Incidence of IFG†	0.7 (0.6–0.9)	0.4 (0.3–0.5)	<0.001	0.5 (0.4–0.6)
Incidence of IGT‡	1.1 (0.9–1.4)	1.3 (1.1–1.5)	0.10	1.2 (1.1–1.4)

Data are incidence percent per year (95% CI), age and sex standardized to the 1998 Australian population. *Men versus women. *P* value derived in age-adjusted models using logistic regression. †The population at risk of developing IFG were those with normal glycemia at baseline. ‡The population at risk of developing IGT were those with IFG or normal glycemia at baseline.

not shown). The basic model with FPG yielded an area under the curve of 0.82 (95% CI 0.79–0.85), which increased by 0.05 and 0.01 by the addition of 2hPG and A1C, respectively. The differences in area under the curve upon the addition of 2hPG ($P < 0.001$) and A1C ($P = 0.003$) were statistically significant at each point.

In a model including all three measures of glycemia (per 1 SD), as well as other independent risk factors, each measure was a significant, independent predictor of diabetes (A1C: odds ratio 2.9 [95% CI 2.1–3.9], FPG: 2.3 [1.7–3.2], and 2hPG: 3.0 [2.4–3.8]).

Table 3—*Risk factors associated with incident diabetes: the AusDiab study*

	Odds ratio (95% CI)	
Characteristics	Age and sex adjusted	Multivariate adjusted*
Age (per 10 years)	1.37 (1.23–1.52)	1.12 (0.97–1.29)
Female sex	0.79 (0.61–1.04)	1.22 (0.89–1.68)
Waist circumference (per SD)	1.84 (1.62–2.09)	1.26 (1.08–1.48)
Smoking status		
Nonsmoker	1.00	1.00
Former smoker	1.15 (0.84–1.57)	1.03 (0.74–1.44)
Current smoker	1.83 (1.23–2.74)	1.70 (1.11–2.63)
Education		
University/further education	1.00	1.00
Completed secondary school	1.20 (0.81–1.78)	1.23 (0.81–1.87)
Some secondary school	1.24 (0.90–1.72)	0.95 (0.68–1.37)
Primary school/never attended school	2.16 (1.30–3.61)†	1.85 (1.04–3.31)†
Physical inactivity (min/week)‡		
Sufficient (≥150 min/week leisure-time physical activity)	1.00	1.00
Insufficient (1–49 min/week leisure-time physical activity)	1.71 (1.26–2.33)	1.51 (1.01–2.25)
Inactive (0 min/week leisure-time physical activity)	1.99 (1.39–2.86)†	1.56 (1.12–2.18)†
Hypertension§	2.64 (1.94–3.60)	1.64 (1.17–2.28)
Family history of diabetes	2.19 (1.60–2.13)	1.82 (1.30–2.52)
Log FPG (per SD)	7.11 (5.51–9.16)	5.25 (3.98–6.92)
Log A1C (per SD)	8.39 (6.50–10.84)	3.94 (2.92–5.32)
Log 2hPG (per SD)	4.83 (3.94–5.92)	3.49 (2.77–4.40)
Hypertriglyceridemia‖	2.80 (2.12–3.69)	1.46 (1.04–2.02)
Low HDL cholesterol¶	2.32 (1.75–3.07)	1.09 (0.78–1.53)
Total cholesterol (per mmol/l)	1.21 (0.91–1.60)	Not included in model

Data are odds ratio (95% CI) from univariate and multivariate logistic regression of incident diabetes and risk factors. *Analyses adjusted for all factors listed in Table 3 except for A1C and 2hPG. †*P* for trend <0.05. ‡Physical activity time for the previous week was calculated as the sum of the time spent performing moderate activity (e.g. walking) plus double the time spent in vigorous activity. §Hypertension was defined as systolic blood pressure ≥140 mmHg or diastolic blood pressure ≥90 mmHg or reporting antihypertensive medication use. ‖Hypertriglyceridemia was defined as triglycerides ≥2.0 mmol/l. ¶Low HDL cholesterol was defined as HDL cholesterol <1.03 mmol/l for men and <1.29 mmol/l for women.

CONCLUSIONS — Despite the obvious importance of accurate diabetes data, there are no national, population-based studies reporting the incidence of diabetes in a developed country. There are, however, two previous studies reporting the incidence of diabetes in high-risk Australians. The incidence of diabetes in Australian Aborigines (24) and in older (aged ≥49 years) Australians from the Blue Mountains Eye Study (25) was 9.9% over 8 years and 9.3% over 10 years, respectively. The data from both of these studies equate to slightly higher incidences than we report here for AusDiab, in keeping with the higher risks of these populations. Compared with other recent studies reporting incidence of diabetes using an OGTT, our study has a slightly lower incidence than has been reported for South African Indians (26), a very similar incidence than has been reported in Italians (5), and a much lower incidence than that reported in high-risk populations such as Mauritians (16) and Pima Indians (7).

The incidence of diabetes was higher for those with IGT than for those with IFG. However, as shown in Table 3, the relationship between baseline FPG and incident diabetes was slightly stronger than was the relationship between baseline 2hPG and incident diabetes. This apparent inconsistency is due to the fact that the World Health Organization classifications include all those who have combined IFG and IGT within the IGT group, while IFG includes only those with isolated IFG and a completely normal 2hPG.

The incidence of diabetes in those with IFG was higher in women than in men, while at baseline, the prevalence of IFG was higher in men (18). Conversely, the incidence of diabetes in those with IGT was higher in men than in women, but the prevalence of IGT was slightly higher in women. It appears that although more women had IGT, men with IGT were more likely to convert to diabetes. In contrast, while more men had IFG, women with IFG were more likely to convert to diabetes. While this may appear counterintuitive, it is explained by sex differences in glucose levels. Among those with IGT, men have a higher FPG, leading to a higher risk of progressing to diabetes. Among those with IFG, women have a higher 2hPG, leading to a higher risk of progression to diabetes. We have previously reported these glucose differences (27) and observed that women have a larger difference between FPG and 2hPG than do men. This in itself may be due to the use of a fixed glucose load (75 g) in men and women, despite women being shorter than men.

Traits of the metabolic syndrome were associated with an increased risk of diabetes, as has been shown previously (5,13,14,28). Relationships with HDL cholesterol were less important in our study than in other studies (25).

The effect of age in the model of incident diabetes was attenuated after adjustment for other age-related covariates. This finding is consistent with other reports. Cugati et al. (25) showed that age was only weakly associated with incident diabetes in a cohort of older Australians. Similar to the findings in this study, family history of diabetes was also associated with incident diabetes (7,25). Consistent with data from other large recent prospective studies, smokers were found to be at increased risk of diabetes compared with nonsmokers (29,30).

In terms of other lifestyle factors, education level, a likely surrogate for socioeconomic status, was associated with increased risk of diabetes. Those having only a primary school level of education or who reported having no education were almost two times more likely to develop diabetes, and there was a significant trend across categories of education attainment, suggesting that risk of diabetes increased with lower levels of education. This finding confirms early research showing that a low level of education predicted diabetes in Mexicans Americans (31).

Our findings of an increased diabetes risk among those who are physically inactive confirm our earlier cross-sectional results (32–34), as well as longitudinal and interventional data from other studies (2,4,15). These findings further reinforce the message that the adoption and maintenance of a physically active lifestyle must be a fundamental element of any approach to prevent diabetes.

The strength of the association between A1C and incident diabetes was noteworthy. Despite the fact that diabetes is defined by blood glucose, A1C was as good a predictor of diabetes as was 2hPG, suggesting a possible clinical role for the measurement of A1C (when the methods are better standardized) in the prediction of diabetes risk and not just in the management of those with established diabetes.

The strengths of the current study are *1*) inclusion of men and women of a wide age range; *2*) a large national, population-based sample of Australian adults; *3*) diagnosis of diabetes using an objective measure, the OGTT; and *4*) evaluation of a large array of candidate risk factors of incident diabetes. This study is also not without limitations. The response rate at baseline was 55% (17), and only 60% of all participants were followed-up. Therefore, it could be said that the results may not be reflective of the whole Australian population. This, however, is offset by the observation that the incidence of self-report diabetes among 1,990 participants who only completed questionnaires (and were not included in the main analysis) was very similar to the incidence of diabetes in 5,842 participants in the main analysis. It is unlikely that the remaining ~20% of participants for whom we do not have any follow-up information would have had an appreciable impact on the overall incidence of diabetes. If anything, the differences between attendees and nonattendees suggests that a healthy cohort bias may have occurred in this study and that these findings could be a slight underestimate of the true incidence of diabetes in Australia. Another limitation relates to the number of cases of diabetes ($n = 224$); it is possible that some effects of covariates may have been missed due to insufficient power.

This study provides the first national, population-based data on the incidence of diabetes in a developed country and, as such, includes information of significant utility in both clinical and health-planning settings. The findings confirm the high risk of those with IFG and IGT, as well as the importance of physical activity, adiposity, and cigarette smoking as well as other previously identified risk factors, while also suggesting the potential value of A1C in the prediction of diabetes.

In Australia, the prevalence of diabetes doubled from 1981 to 2000 (18), and it is projected to increase further by the year 2025 (35). Obesity has been identified as a key driver of diabetes in this study, and the expected increases in the prevalence of obesity (34) are likely to further increase the incidence and prevalence of diabetes, contributing significantly to the total burden of ill health in Australia.

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