Ten-year incidence of diabetes in older Australians: the Blue Mountains Eye Study

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ABSTRACT

Objective: To estimate the incidence of diabetes and impaired fasting glucose (IFG), and increased risk associated with the metabolic syndrome, in a representative population-based sample of older Australians.

Design, setting and participants: The Blue Mountains Eye Study examined 3654 residents aged 49 + years (82.4% response rate) during 1992–1994, and re-examined 2335 (75.1% of survivors) during 1997–1999 and 1952 (75.6% of survivors) during 2002–2004; 2123 participants with normal blood glucose levels at baseline were considered at risk of developing incident diabetes.

Main outcome measures: Incident diabetes (or IFG) was defined in participants at risk who were newly diagnosed by a physician during the follow-up or found to have a fasting blood glucose level ⩾ 7.0 mmol/L (or 5.6–6.9 mmol/L). Kaplan–Meier cumulative 10-year incidence was calculated.

Results: The overall 10-year incidence of diabetes and IFG was 9.3% and 15.8%, respectively. Participants with metabolic syndrome at baseline had a higher risk of incident diabetes than those without metabolic syndrome (29.2% v 8.6%). Baseline factors associated with incident diabetes were elevated fasting glucose level (adjusted odds ratio [OR], 4.5; 95% CI, 3.4–6.1 per mmol/L), obesity (OR, 2.0; 95% CI, 1.3–2.8), diabetes family history (OR, 1.7; 95% CI, 1.2–2.5), current smoking (OR, 1.6; 95% CI, 1.0–2.7) and high density lipoprotein cholesterol level < 1.0 mmol/L (OR, 2.4; 95% CI, 1.5–3.8). Similar baseline factors were associated with incident IFG.

Conclusion: This population-based study provides data on the incidence of diabetes and IFG in an older, predominantly white population, and confirms that metabolic and lifestyle factors are major risk factors for diabetes.

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Diabetes is a major threat to public health.[1-4] Many studies have estimated or assessed the incidence of diabetes in adult populations during the past 10 years.[5-11] The reported incidence varied from 2–8 per 1000 person-years[5,7,8,10,11] to 10–15 per 1000 person-years.[6,12] A recent secular increase in diabetes incidence has also been reported; for example, in the San Antonio Heart Study, diabetes incidence tripled from 5.7% to 15.7% in Mexican Americans and from 2.6% to 9.4% in non-Hispanic whites over 9 years.[6] Importantly, diabetes incidence is increasing in both developed and developing countries.

In an Australian Aboriginal population, diabetes incidence was previously reported as 9.9% over 8 years.[8] To our knowledge, there are no current reports on the incidence of diabetes and impaired fasting glucose (IFG) from non-Aboriginal population-based cohorts in Australia, although the AusDiab Study will provide such data.[13]

The Blue Mountains Eye Study (BMES) is a population-based cohort study of predominantly white, older Australians. We previously reported a diabetes prevalence of 8.8% at baseline in this study.[14] With completion of BMES 5- and 10-year examinations (BMES II and III), we now report the incidence of both diabetes and IFG, plus associated risk factors, including components of the metabolic syndrome, in this study population.

METHODS

Population sample

The BMES was conducted in an older community within a geographically defined area, west of Sydney. The study was approved by the Western Sydney Area Human Research Ethics Committee and was conducted in adherence to the tenets of the Declaration of Helsinki.

Details of the BMES methods were previously reported.[14,15] The study population was representative of the Australian population for demographic and socioeconomic status, although slightly older on average. After a private door-to-door census of 38 Australian Bureau of Statistics (ABS) Census Collection Districts of two postcodes in the Blue Mountains region of New South Wales (2780 and 2782), all permanent, non-institutionalised residents with birthdates before 1 January 1943 were invited to participate. Of the 4433 eligible residents, 3654 (82.4%) participated in baseline examinations (BMES I) during 1992–1994. A comparison of characteristics of participants and non-participants was previously reported.[15] Surviving baseline participants were invited to attend follow-up examinations after 5 years (1997–1999) and 10 years (2002–2004).

Data collection

All three examinations used similar survey methods. A detailed questionnaire was administered by trained interviewers and included demographic information, eye and general medical history, and medications used. All participants were asked to undertake a fasting blood test within a month of the interview to assess fasting blood glucose, lipids, creatinine and fibrinogen concentrations. Blood was collected in fluoride/oxalate tubes and centrifuged on site within 1 hour to separate plasma. Samples were then refrigerated before transportation on ice by courier within 4 hours of collection to the Institute of Clinical Pathology and Medical Research, Westmead Hospital, Sydney, for blood glucose analysis by the hexokinase method.

Diabetes definitions

Criteria for a diagnosis of diabetes were:

- self-reported diabetes history and current use of diabetic medications, or
- a fasting plasma glucose concentration ⩾ 7.0 mmol/L, according to the World Health Organization diabetes classification.[3]

IFG was defined as:

- fasting plasma glucose concentration 5.6–6.9 mmol/L among participants not using diabetes medications, as recommended by the American Diabetes Association (ADA).[16]

1 Baseline factors associated with the incidence of diabetes

Baseline characteristic	No. with incident diabetes (%*)	Odds ratio (95% CI)	
		Age- and sex-adjusted	Multivariate-adjusted†
Age (per decade)		1.04 (0.85–1.25)	1.01 (0.98–1.24)
Female sex	105 (8.5%)	1.24 (0.90–1.71)	1.73 (1.19–2.52)
Family history of diabetes	58 (13.0%)	2.04 (1.46–2.85)	1.72 (1.19–2.47)
Obesity	55 (16.4%)	2.89 (2.05–4.06)	1.95 (1.33–2.84)
Current smoker	27 (10.5%)	1.54 (0.99–2.38)	1.64 (1.01–2.65)
Hypertension			
Stage I	42 (7.1%)	1.27 (0.81–1.99)	1.05 (0.65–1.71)
Stage II	85 (9.7%)	1.90 (1.27–2.85)	1.26 (0.81–1.95)
Gout	21 (11.2%)	1.80 (1.10–2.95)	
Fasting plasma glucose (continuous) (mmol/L)		5.14 (3.93–6.71)	4.54 (3.41–6.06)
Fasting plasma glucose			
< 5 mmol/L	45 (3.9%)	1.0	
5 to < 6 (5.96) mmol/L	85 (9.7%)	2.84 (1.96–4.11)	
6 to < 7 (6.97) mmol/L	35 (39.8%)	19.13 (11.59–31.66)	
Hypercholesterolaemia (≥ 5.2 mmol/L)	129 (8.2%)	0.95 (0.64–1.40)	
Hypertriglyceridaemia (≥ 2 mmol/L)	62 (12.5%)	1.98 (1.43–2.75)	1.07 (0.73–1.56)
Low serum HDL level (≤ 1 mmol/L)	33 (18.2%)	3.28 (2.15–5.00)	2.37 (1.47–3.82)
Metabolic syndrome	47 (23.2%)	4.35 (3.03–6.26)	

* Percentage of number at risk. † After adjusting for the factors listed in the column using the multiple discrete logistic regression models. HDL = high density lipoprotein cholesterol. ◆

Diabetes incidence was defined in participants who were free of diabetes at baseline but who developed diabetes, diagnosed before or at the 5- or 10-year follow-up examination. Similarly, IFG incidence was defined in participants who were free of diabetes or IFG at baseline but who developed IFG, diagnosed before or at the 5- or 10-year follow-up examination.

Risk factor assessment

The 2003 WHO/International Society of Hypertension guidelines[17] were used to classify participants as:

- pre-hypertensive if systolic blood pressure (BP) was 120–139 mmHg or diastolic BP was 80–89 mmHg;
- hypertensive stage I if systolic BP was 140–159 mmHg or diastolic BP was 90–99 mmHg; and
- hypertensive stage II or higher if the individual was previously diagnosed as hypertensive and was using antihypertensive medications or had a systolic BP ≥ 160 mmHg or diastolic BP ≥ 100 mmHg at examination.

Obesity was defined as body mass index (BMI) ≥ 30 kg/m^2. Participants were classified as smokers if they currently smoked or had stopped smoking less than 12 months before examination.

Total cholesterol, triglycerides and high density lipoprotein (HDL) were measured from fasting blood samples using a 747 Biochemistry Analyzer (Hitachi, Tokyo, Japan). Hypercholesterolaemia was defined as a serum cholesterol level ≥ 5.2 mmol/L, hypertriglyceridaemia as serum triglyceride ≥ 2.0 mmol/L, and abnormal HDL cholesterol as ≤ 1.0 mmol/L.

Renal function was estimated based on serum creatinine level and creatinine clearance (using the Cockcroft–Gault formula). Adjusted creatinine clearance was then calculated as creatinine clearance × 1.73/body surface area.[18] Renal impairment was considered present if the adjusted creatinine clearance was < 60 mL/min/1.73 m^2. All participants were asked about a history of angina, acute myocardial infarction, stroke or gout.

The metabolic syndrome was defined according to the new International Diabetes Federation definition[19] as central obesity (defined as BMI > 30 kg/m^2) plus any two of the following four factors:

- serum triglyceride level ≥ 1.7 mmol/L, or specific treatment for this lipid abnormality;
- serum HDL cholesterol < 1.03 mmol/L in men and < 1.29 mmol/L in women, or specific treatment for this lipid abnormality;
- systolic BP ≥ 130 mmHg or diastolic BP ≥ 85 mmHg, or treatment of previously diagnosed hypertension; or
- fasting plasma glucose ≥ 5.6 mmol/L.

A history of diabetes in the participant's family was sought during the study interview. First-degree relatives included parents and siblings, while second-degree relatives included both maternal and paternal relatives. Participants with unknown family history of diabetes in both parents were considered to have no family history of diabetes.

Data analysis

Data were analysed using SAS version 9.1 (SAS Institute Inc, Cary, NC, USA). Ten-year cumulative incidence rates were calculated using the Kaplan–Meier (product-limit) method, which allowed participants who were censored at the 5-year follow-up examination to contribute to the estimates. Multi-

2 Risk of developing diabetes by the presence of obesity* and number of additional metabolic syndrome (MS) traits at baseline

Baseline	No. at risk	No. with incident diabetes (%†)	Odds ratio (95% CI)	
			Age- and sex-adjusted	Multivariate-adjusted‡
No risk factors for MS	1762	108 (6.1%)	1	1
Obesity + 0–1 MS trait	132	8 (6.1%)	0.93 (0.45–1.94)	0.97 (0.46–2.02)
Obesity + 2 MS traits	104	19 (18.3%)	3.27 (1.94–5.48)	2.99 (1.73–5.17)
Obesity + 3 MS traits	79	20 (25%)	5.43 (3.23–9.14)	4.98 (2.94–8.43)
Obesity + 4 MS traits	20	8 (40%)	9.34 (4.06–21.47)	8.07 (3.45–18.89)
P for trend			< 0.0001	< 0.0001

* Obesity defined as body mass index > 30 kg/m^2. † Percentage of number at risk. ‡ After adjusting for age, sex, current smoking, and positive family history of diabetes in multiple discrete logistic regression models. ◆

3 Risk of developing diabetes by family history of diabetes

Diabetes history	No. with incident diabetes (%*)	Multivariate-adjusted odds ratio (95% CI)†		
		Both sexes	Women	Men
Paternal history	22 (15.7%)	2.23 (1.36–3.65)	2.55 (1.42–4.59)	1.32 (0.50–3.50)
Maternal history	24 (10.8%)	1.33 (0.83–2.13)	1.31 (0.75–2.29)	1.24 (0.49–3.15)
Sibling history	14 (15.1%)	1.86 (1.02–3.38)	1.94 (0.95–3.96)	2.08 (0.68–6.32
First-degree relative	40 (13.9%)	2.05 (1.40–3.01)	2.15 (1.35–3.42)	1.78 (0.89–3.57)
Second-degree relative	16 (9.6%)	1.04 (0.59–1.83)	1.11 (0.59–2.10)	0.70 (0.19–2.57)

* Percentage of number at risk. † After adjusting for age, sex, obesity, current smoking, low level of serum high density lipoprotein (< 1.0 mmol/L), and family history of diabetes in the multiple logistic regression models. ◆

variate-adjusted discrete logistic models (by means of the PHREG SAS procedure) were used to assess factors associated with incident diabetes, using three time points when presence or absence of the outcome event was recorded.[20] Participants with incomplete data were excluded from analyses. Variables tested for association with diabetes incidence included fasting plasma glucose level, obesity, serum cholesterol level, serum HDL cholesterol level, serum triglycerides, current smoking, hypertension and family history of diabetes. Trends for increasing incidence with increasing age were tested for significance using the Mantel–Haenszel χ^2 test. Odds ratios and 95% CIs are presented. Tests for interaction between sex and selected risk factors (family history of diabetes, HDL and obesity) showed they were not significant.

RESULTS

Incidence of diabetes and associations

Of the 3654 baseline participants, 2335 (75.1% of survivors) returned to the 5-year follow-up examination, and 1952 (75.6% of survivors) to the 10-year follow-up examination. Including participants seen at either or both of these examinations, 2564 participants were followed up after baseline. Surviving participants who did not return at either the 5- or 10-year follow-up examinations were more likely to have been younger than 60 years of age and current smokers at baseline.

Exclusions were 163 participants with diabetes at baseline, 52 with missing diabetes data or fasting blood samples at baseline, and 226 with no fasting blood tests at either the 5- or 10-year examinations. Thus, 2123 participants (1242 women and 881 men) had complete data available and were included in the assessment of 10-year incident diabetes.

The 10-year incidence of diabetes in the study population estimated by the Kaplan–Meier method was 9.3% ($n = 165$). Of these 165 participants, 52 (31.5%) were diagnosed solely on the basis of an elevated fasting plasma glucose level at examination. The remaining 113 (68.5%) gave a history of physician-diagnosed diabetes and were receiving treatment (insulin, oral therapy or diet).

More women (incidence, 10.0%, $n = 105$) than men (8.2%, $n = 60$) developed diabetes, although this difference was not statistically significant ($P = 0.20$). The age-specific incidence of diabetes was 9.1%, 8.6%, and 11.2%, in the age groups < 60, 60–69 and 70 + years, respectively (P for age-related trend = 0.73). After multivariate adjustment, baseline plasma glucose level, low HDL cholesterol, presence of obesity, current smoking and family history of diabetes were all significantly associated with an increased risk of diabetes, but presence of hypertension was not (Box 1). Past history of angina, acute myocardial infarction, stroke or gout were also not found to be associated with diabetes incidence, nor was body surface area-adjusted creatinine clearance (data not shown).

The 10-year cumulative diabetes incidence was substantially higher among the 203 participants who had metabolic syndrome at baseline (28.1%) than among those without metabolic syndrome (7.6%). Box 2 shows the effect of metabolic syndrome traits on the incidence of diabetes. The risk of diabetes increased with a greater number of metabolic syndrome traits, in addition to a BMI > 30 kg/m^2. A two-, four- and eightfold increased risk of diabetes was associated with the presence of two, three and four metabolic syndrome traits, respectively, in addition to a BMI > 30 kg/m^2.

The 10-year incidence of diabetes was higher in participants with IFG at baseline (30.0%) than in those with normoglycaemia (6.5%). Of the 194 participants with IFG at baseline who did not develop diabetes, 94 (48.5%) were normoglycaemic at the 5-year examination, and 15 (7.7%) at the 10-year examination, using the new ADA definition. Of the 94 who were normoglycaemic at the 5-year examination, nine (9.6%) reverted to IFG at the 10-year examination. The corresponding return rates from IFG at baseline to normoglycaemia at follow-up examinations using the previous ADA definition (6.1–6.9 mmol/L) were also assessed: 34/50 (68%) became normoglycaemic at the 5-

4 Baseline factors associated with the incidence of impaired fasting glucose (IFG)

Baseline characteristic	No. with incident IFG (%*)	Odds ratio (95% CI)	
		Age- and sex-adjusted	Multivariate-adjusted†
Age (per decade)		1.24 (1.06–1.45)	1.20 (1.01–1.36)
Female sex	140 (13.5%)	0.80 (0.61–1.04)	0.87 (0.65–1.16)
Family history of diabetes	48 (13.9%)	0.99 (0.71–1.38)	
Obesity	56 (23.6%)	2.14 (1.55–2.97)	1.83 (1.28–2.61)
Current smoker	46 (22.0%)	1.99 (1.39–2.84)	1.96 (1.33–2.89)
Hypertension			
Stage I	80 (15.9%)	1.57 (1.10–2.24)	1.43 (0.98–2.11)
Stage II	115 (16.9%)	1.69 (1.21–2.38)	1.52 (1.06–2.19)
Fasting plasma glucose (continuous) (mmol/L)		6.84 (4.61–10.16)	6.92 (4.57–10.47)
Hypercholesterolaemia (≥ 5.2 mmol/L)	201 (15.5%)	1.16 (0.83–1.62)	
Hypertriglyceridaemia (≥ 2 mmol/L)	69 (18.7%)	1.36 (1.01–1.83)	1.15 (0.84–1.58)
Low serum HDL level (≤ 1 mmol/L)	23 (18.4%)	1.17 (0.73–1.87)	

* Percentage of number at risk. † After adjusting for the factors listed in the column in multiple discrete logistic regression models. HDL = high density lipoprotein. ◆

year examination, and two (4%) at the 10-year examination. Of the 34 who returned to normoglycaemia after 5 years, three (9%) developed IFG at the 10-year examination.

A history of diabetes in first-degree relatives, or a paternal history of diabetes, was also a significant predictor of incident diabetes (Box 3).

Incidence of IFG and associations

After excluding participants with diabetes or IFG at baseline and those with missing fasting blood samples, 1756 participants were considered at risk of incident IFG. The overall 10-year incidence of IFG was estimated as 15.8% (17.2% in men versus 14.7% in women; $P = 0.11$). The age-specific IFG incidence was 11.0%, 16.0%, and 18.6% in participants aged < 60, 60–69, and 70 + years, respectively (P for age-related trend = 0.03). Higher baseline plasma glucose level, current smoking, presence of obesity and stage II hypertension were all associated with increased IFG incidence (Box 4). However, family history of diabetes, low HDL cholesterol level, and high total cholesterol or triglyceride levels were not significantly associated with incident IFG, nor was history of gout, angina, stroke, or acute myocardial infarction, or the presence of renal impairment (data not shown).

Using the previous ADA definition (fasting plasma glucose, 6.1–6.9 mmol/L),[21] the 10-year IFG incidence was 5.7%. Risk factors significantly associated with incident IFG of this definition included higher plasma glucose levels, stage II hypertension and hypertriglyceridaemia.

DISCUSSION

In this white Australian population aged 49 + years at baseline, we found that the 10-year cumulative incidence of type 2 diabetes was 9.3%, with only a weak age-related trend. Elevated fasting plasma glucose level, traits of the metabolic syndrome, current smoking and family history of diabetes were all independent predictors of type 2 diabetes. The 10-year incidence of IFG was 15.8%. We are unaware of other current large prospective population-based studies in Australia that have reported the long-term incidence of type 2 diabetes among non-Aboriginal Australians. Increasing age, current smoking and obesity were significant factors predicting higher risk of incident IFG. We found no apparent association between hypertension and diabetes incidence. An explanation for our finding that the age-related trend for incident diabetes, reported from many other studies, was only weak is not clear, but could reflect selective mortality in this older population sample. A clear age-related trend was found for incident IFG.

Notably, the 9.3% diabetes incidence over 10 years in this older, predominantly white Australian population is only slightly lower than the incidence of 9.9% over 8 years reported for an Australian Aboriginal population,[8] a group well known to have higher diabetes prevalence, suggesting that the incidence of diabetes is rising. However, it is important to consider the differences between the two studies. The BMES was undertaken 5 years after the study in Australian Aboriginals, and the diagnostic criteria for diabetes differed: BMES used fasting plasma glucose level, while the study in Australian Aboriginals used 2-hour glucose tolerance. This is a major limitation of our study. The 2-hour oral glucose tolerance test and fasting plasma glucose level measure somewhat different metabolic abnormalities, and both have a certain degree of misclassification.[22] Hence, we may have underestimated the incidence of diabetes.

A 60% higher risk of diabetes was found among current smokers compared with non-smokers or ex-smokers at baseline, independent of other risk factors. Our findings that smoking predicts both incident diabetes and IFG are comparable to those from other recent large population-based studies.[23,24] This link could be the result of increased insulin resistance in smokers, either directly due to pancreatic damage, or indirectly due to increased hepatic lipase activity or impaired insulin-stimulated glucose transport in skeletal muscles, which has been observed in smokers.[23,24] Efforts to encourage smoking cessation could thus be cost effective in reducing the burden of diabetes.

We observed that the presence of two or more metabolic syndrome traits in addition to a BMI > 30 kg/m^2 significantly increased the risk of diabetes, in keeping with findings from other studies.[2,25] Given that the metabolic syndrome now affects nearly a quarter of the adult population in developed countries,[25] continued increases in both the prevalence and incidence of diabetes are likely.

The strengths of this study include its population-based sample and reasonable follow-up at both 5 and 10 years. Fasting plasma glucose level was measured in most participants, and incident diabetes was defined either by a physician diagnosis of diabetes or elevated fasting plasma glucose level. A major drawback of our study is the "surrogate" definition of central obesity. As other anthropometric measures of abdominal fatness were not taken at baseline examinations of our study cohort, we were unable to study the association between waist circumference and diabetes incidence. Although central obesity can be presumed in a middle-aged person with a BMI > 30 kg/m^2,[19] participants with increased waist circumference but normal BMI would have been misclassified in our analysis. Cross-sectional analytical data from the AusDiab Study indicated that, although waist circumference and waist/hip ratio were superior to BMI in terms of the diabetes association in men, no differences were found in these three measurements in women.[26]

In summary, in this population-based study of older Australians, incidence rates for diabetes and IFG over a 10-year period were 9.3% and 15.8%, respectively. Baseline cardiovascular risk factors, particularly traits of the metabolic syndrome, and family history of diabetes, were shown to predict incident diabetes. Addressing factors that predict the metabolic syndrome would help decrease the future incidence of diabetes. Preventive strategies should also focus on people with a first-degree family history of diabetes and those who smoke.

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COMPETING INTERESTS

None identified.

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REFERENCES

1 Jonsson B. The economic impact of diabetes. *Diabetes Care* 1998; 21 Suppl 3: C7-C10.
2 Wilson PW, D'Agostino RB, Parise H, et al. Metabolic syndrome as a precursor of cardiovascular disease and type 2 diabetes mellitus. *Circulation* 2005; 112: 3066-3072.

3 World Health Organization. Definition, diagnosis and classification of diabetes mellitus and its complications. Part 1: diagnosis and classification of diabetes mellitus. Geneva: WHO, 1999.
4 King H, Aubert RE, Herman WH. Global burden of diabetes, 1995-2025: prevalence, numerical estimates, and projections. *Diabetes Care* 1998; 21: 1414-1431.
5 de Vegt F, Dekker JM, Jager A, et al. Relation of impaired fasting and postload glucose with incident type 2 diabetes in a Dutch population: the Hoorn Study. *JAMA* 2001; 285: 2109-2113.
6 Burke JP, Williams K, Gaskill SP, et al. Rapid rise in the incidence of type 2 diabetes from 1987 to 1996: results from the San Antonio Heart Study. *Arch Intern Med* 1999; 159: 1450-1456.
7 Rockwood K, Awalt E, MacKnight C, et al. Incidence and outcomes of diabetes mellitus in elderly people: report from the Canadian Study of Health and Aging. *CMAJ* 2000; 162: 769-772.
8 Daniel M, Rowley KG, McDermott R, et al. Diabetes incidence in an Australian Aboriginal population. An 8-year follow-up study. *Diabetes Care* 1999; 22: 1993-1998.
9 Garancini MP, Gobbi C, Errera A, et al. Age-specific incidence and duration of known diabetes. The Cremona Study. *Diabetes Care* 1996; 19: 1279-1282.
10 Meisinger C, Thorand B, Schneider A, et al. Sex differences in risk factors for incident type 2 diabetes mellitus: the MONICA Augsburg cohort study. *Arch Intern Med* 2002; 162: 82-89.
11 Bonora E, Kiechl S, Willeit J, et al. Population-based incidence rates and risk factors for type 2 diabetes in white individuals: the Bruneck study. *Diabetes* 2004; 53: 1782-1789.
12 Brancati FL, Kao WH, Folsom AR, et al. Incident type 2 diabetes mellitus in African American and white adults: the Atherosclerosis Risk in Communities Study. *JAMA* 2000; 283: 2253-2259.
13 Cameron AJ, Welborn TA, Zimmet PZ, et al. Overweight and obesity in Australia: the 1999–2000 Australian Diabetes, Obesity and Lifestyle Study (AusDiab). *Med J Aust* 2003; 178: 427-432.
14 Mitchell P, Smith W, Wang JJ, et al. Diabetes in an older Australian population. *Diabetes Res Clin Pract* 1998; 41: 177-184.
15 Attebo K, Mitchell P, Smith W. Visual acuity and the causes of visual loss in Australia. The Blue Mountains Eye Study. *Ophthalmology* 1996; 103: 357-364.
16 Genuth S, Alberti KG, Bennett P, et al. Follow-up report on the diagnosis of diabetes mellitus. *Diabetes Care* 2003; 26: 3160-3167.
17 2003 World Health Organization (WHO)/International Society of Hypertension (ISH) statement on management of hypertension. *J Hypertens* 2003; 21: 1983-1992.
18 Huynh SC, Kifley A, Strippoli GF, et al. Is renal impairment a predictor of the incidence of cataract or cataract surgery? Findings from a population-based study. *Ophthalmology* 2005; 112: 293-300.
19 Zimmet P, Magliano D, Matsuzawa Y, et al. The metabolic syndrome: a global public health problem and a new definition. *J Atheroscler Thromb* 2005; 12: 295-300.
20 Hosmer DW, Lemeshow S. Applied logistic regression. New York: John Wiley & Sons Inc, 1989.
21 The Expert Committee on the Diagnosis and Classification of Diabetes Mellitus. Report of the expert committee on the diagnosis and classification of diabetes mellitus. *Diabetes Care* 1998; 21 Suppl 1: S5-S19.
22 Unwin N, Shaw J, Zimmet P, et al. Impaired glucose tolerance and impaired fasting glycaemia: the current status on definition and intervention. *Diabet Med* 2002; 19: 708-723.
23 Patja K, Jousilahti P, Hu G, et al. Effects of smoking, obesity and physical activity on the risk of type 2 diabetes in middle-aged Finnish men and women. *J Intern Med* 2005; 258: 356-362.
24 Will JC, Galuska DA, Ford ES, et al. Cigarette smoking and diabetes mellitus: evidence of a positive association from a large prospective cohort study. *Int J Epidemiol* 2001; 30: 540-546.
25 Tkac I. Metabolic syndrome in relationship to type 2 diabetes and atherosclerosis. *Diabetes Res Clin Pract* 2005; 68 Suppl 1: S2-S9.
26 Dalton M, Cameron AJ, Zimmet PZ, et al. Waist circumference, waist-hip ratio and body mass index and their correlation with cardiovascular disease risk factors in Australian adults. *J Intern Med* 2003; 254: 555-563.

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