

The Rising Prevalence of Diabetes and Impaired Glucose Tolerance

The Australian Diabetes, Obesity and Lifestyle Study

DAVID W. DUNSTAN, PHD¹
PAUL Z. ZIMMET, MD¹
TIMOTHY A. WELBORN, PHD²
MAXIMILIAN P. DE COURTEN, MD¹
ADRIAN J. CAMERON, MPH¹
RICHARD A. SICREE, MPH¹
TERRY DWYER, PHD³

STEPHEN COLAGIURI, MD⁴
DAMIEN JOLLEY, MSC⁵
MATTHEW KNUIMAN, PHD⁶
ROBERT ATKINS, MD⁷
JONATHAN E. SHAW, MD¹
ON BEHALF OF THE AUSDIAB STEERING
COMMITTEE

OBJECTIVE — To determine the population-based prevalence of diabetes and other categories of glucose intolerance (impaired glucose tolerance [IGT] and impaired fasting glucose [IFG]) in Australia and to compare the prevalence with previous Australian data.

RESEARCH DESIGN AND METHODS — A national sample involving 11,247 participants aged ≥ 25 years living in 42 randomly selected areas from the six states and the Northern Territory were examined in a cross-sectional survey using the 75-g oral glucose tolerance test to assess fasting and 2-h plasma glucose concentrations. The World Health Organization diagnostic criteria were used to determine the prevalence of abnormal glucose tolerance.

RESULTS — The prevalence of diabetes in Australia was 8.0% in men and 6.8% in women, and an additional 17.4% of men and 15.4% of women had IGT or IFG. Even in the youngest age group (25–34 years), 5.7% of subjects had abnormal glucose tolerance. The overall diabetes prevalence in Australia was 7.4%, and an additional 16.4% had IGT or IFG. Diabetes prevalence has more than doubled since 1981, and this is only partially explained by changes in age profile and obesity.

CONCLUSIONS — Australia has a rapidly rising prevalence of diabetes and other categories of abnormal glucose tolerance. The prevalence of abnormal glucose tolerance in Australia is one of the highest yet reported from a developed nation with a predominantly European background.

Diabetes Care 25:829–834, 2002

The prevalence of type 2 diabetes varies widely between populations, reflecting differences in both environmental influences and genetic susceptibility (1). The aging of popula-

tions and the effects of modernization of lifestyle have led to a dramatic increase in the prevalence of diabetes globally with very high rates in developing nations, particularly in Asia and the Pacific (1). Cur-

From the ¹International Diabetes Institute, Melbourne, Australia; the ²Department of Medicine, University of Western Australia, Perth, Australia; the ³Menzies Centre for Population Health Research, University of Tasmania, Hobart, Australia; the ⁴Department of Endocrinology, Diabetes and Metabolism, the Prince of Wales Hospital, New South Wales, Sydney, Australia; the ⁵School of Health Sciences, Deakin University, Melbourne, Australia; the ⁶Department of Public Health, University of Western Australia, Perth, Australia; and the ⁷Department of Nephrology, Monash University, Melbourne, Australia.

Address correspondence and reprint requests to Dr. David Dunstan, International Diabetes Institute, 250 Kooyong Road, Caulfield, Victoria, Australia 3162. E-mail: ddunstan@idi.org.au.

Received for publication 21 January 2002 and accepted in revised form 21 January 2002.

Abbreviations: 2hPG, 2-h plasma glucose; AusDiab, Australian Diabetes, Obesity and Lifestyle Study; CD, Census Collector District; FPG, fasting plasma glucose; IFG, impaired fasting glucose; IGT, impaired glucose tolerance; KDM, known diabetes mellitus; NDM, newly diagnosed diabetes mellitus; OGTT, oral glucose tolerance test; WHO, World Health Organization.

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

rently, it is estimated that 150 million people in the world have diabetes (2). This number is expected to increase to 300 million by the year 2025; most of these cases will be type 2 diabetes (2).

The heightened susceptibility and high prevalence of type 2 diabetes of Micronesia and Polynesian Pacific Islanders (3), Native Americans (4), Indigenous Australians and Torres Strait Islanders (5), and Asian Indians (6) has been well documented. Despite the large body of epidemiological data now available on the high prevalence of diabetes in developing countries, a paucity of data exists on the prevalence of diabetes in developed nations. In fact, despite increasing awareness of the growing problem of diabetes and the recent publication of a number of predictions of current and future prevalences of diabetes worldwide, the U.S. is the only country in the developed world with reliable data on national prevalence (7).

In Australia, there are only two previous population-based studies of diabetes prevalence based on the oral glucose tolerance test (OGTT). In 1981, a study from the rural Western Australia town of Busselton (8) showed a prevalence of 3.4% (2.5% known cases and 0.9% newly diagnosed) in subjects aged ≥ 25 years. The prevalence of impaired glucose tolerance (IGT) was 2.9%. In 1992, a study from rural Victoria reported a diabetes prevalence of 3.4% (1.6% known and 1.8% newly diagnosed) among European adults ≥ 15 years (9). The IGT prevalence was 6.0%. Based on national data on self-reported diabetes only in 1989 and 1990, the prevalence of previously diagnosed diabetes was 1.9% in men and 2.0% in women (10).

The Australian Diabetes, Obesity and Lifestyle Study (AusDiab) (11) is the first national study of the prevalence and impact of diabetes in Australia. The results of this study are compared with those of the 1981 study from Busselton (8) to determine to what extent changes in preva-

Table 1—Prevalence of KDM and NDM, according to age and sex for the Australian population

	Age (years)							
	25–34	35–44	45–54	55–64	65–74	75+	≥25	40–74
Men								
KDM	0.0	1.2	2.7	8.9	12.8	13.9	4.3	5.8
NDM	0.1	1.5	3.9	7.8	7.9	9.6	3.7	5.1
Women								
KDM	0.3	0.9	3.8	4.0	6.6	8.8	3.1	3.8
NDM	0.1	1.3	2.0	5.5	9.0	13.9	3.7	4.0
All subjects								
KDM	0.2	1.0	3.3	6.5	9.4	10.9	3.7	4.8
NDM	0.1	1.4	2.9	6.6	8.5	12.1	3.7	4.6
All diabetes	0.3	2.4	6.2	13.1	17.9	23.0	7.4	9.4

Data are %.

lence of diabetes can be explained by changes in obesity.

RESEARCH DESIGN AND METHODS

Survey design

A detailed description of the methodology has been published elsewhere (11). A representative sample of the national population was drawn from 42 randomly selected urban and nonurban areas (Census Collector Districts [CDs]) across Australia (six CDs in each of the six states and the Northern Territory). CDs containing <100 individuals aged ≥25 years, CDs classified as 100% rural, or CDs with a population comprising >10% Aboriginal or Torres Strait Islanders were excluded. Within each CD, all homes were approached, and adults aged ≥25 years who were usual residents were invited to attend the survey. The survey work took place from May 1999 to December 2000 and consisted of a short household interview followed by a biomedical examination (including blood sampling) at a study examination site within or close to the selected residential areas.

Households not responding to the initial doorstep approach were recontacted up to four more times. Of those homes in which a response was obtained and at least one eligible adult was residing, 70% took part in the household questionnaire. The final survey sample (those attending the biomedical examination) included 11,247 adults, representing 55.3% of those completing the household interview.

Survey procedures

Between 7:00 and 10:00 A.M. each day, ~40 individuals were invited to attend the survey site. Participants were asked to fast overnight and not to take hypoglycemic medication on the day of the test. If their reported fasting time was less than 8 h, they were asked to return on another day. After registration, an OGTT was performed on all participants, except those on insulin or oral hypoglycemic drugs or those who were pregnant. The OGTT was performed according to World Health Organization (WHO) specifications (12). All subjects were given a 300-ml beverage containing 75 g glucose and were asked to consume it in <5 min. Blood specimens were collected into fluoride/oxalate tubes immediately before and 2 h after the glucose load. The specimens were centrifuged, plasma was separated immediately, and plasma glucose levels were determined using an Olympus AU600 automated analyzer, which uses a glucose oxidase method. Height and weight were measured with the subjects wearing light clothing and no shoes, and BMI was calculated as weight (kg)/height (m²).

Glucose tolerance was classified according to the WHO criteria (12). Participants who reported a history of physician-diagnosed diabetes and who were 1) taking oral hypoglycemic tablets or insulin injections or 2) had a fasting plasma glucose (FPG) level ≥7.0 mmol/l or 2-h plasma glucose (2hPG) level ≥11.1 mmol/l were classified as having known diabetes mellitus (KDM). Subjects not reporting diabetes and who had FPG ≥7.0 mmol/l or 2hPG ≥11.1 mmol/l were classified as having newly diagnosed

diabetes mellitus (NDM). For those without KDM, FPG <7.0 mmol/l and 2hPG ≥7.8 mmol/l but <11.1 mmol/l indicated IGT; impaired fasting glucose (IFG) was defined as FPG ≥6.1 mmol/l and <7.0 mmol/l, with 2hPG <7.8 mmol/l; and normal glucose tolerance was defined as FPG <6.1 mmol/l and 2hPG <7.8 mmol/l.

The details of the 1981 Busselton survey have been published previously (8). The Busselton study included 2hPG collected with no FPG. For all comparisons with the Busselton data, only the 2hPG was used for each data set, and subjects on any dietary treatment for diabetes were also classified as having KDM, irrespective of their 2hPG. BMI was calculated as for AusDiab, and such data were available on 2,900 of the 3,196 (90.7%) Busselton survey participants and 11,067 of the 11,247 (98.4%) AusDiab participants.

Statistical analysis

To adjust for nonresponse, the data have been weighted to match the age and gender distribution of the 1998 estimated residential population of Australia aged ≥25 years (13). The weighting factor was based on the probability of selection in each cluster. Therefore, all prevalences provided relate to the total 1998 Australian population aged ≥25 years (13). To account for the clustering and stratification of the survey design, prevalences and 95% CIs were calculated using Stata Statistical Software Release 6.0 (StataCorp, College Station, TX), which adjusts the 95% CIs to account for these aspects of survey design. Unweighted data were calculated using SPSS Version 10.0.5 for Windows (SPSS, Chicago, IL).

The weighting was not used for comparisons with the Busselton data in the section on secular changes, but both populations were adjusted (age and sex) to the 1998 Australian population by the direct method.

Logistic regression was used to determine to what extent differences in age, sex, and BMI could account for differences in diabetes prevalence between Busselton and AusDiab.

RESULTS— There were 11,247 respondents to the biomedical examination. The following significant differences were found between responders and nonresponders. Compared with nonresponders, responders were less likely to

Table 2—Prevalence of IGT and IFG according to age and sex for the Australian population

	Age (years)							40–74
	25–34	35–44	45–54	55–64	65–74	75+	≥25	
Men								
IGT	2.1	4.7	9.0	14.8	20.4	24.8	9.2	12.0
IFG	3.4	8.4	9.3	12.8	11.5	4.6	8.1	10.7
Women								
IGT	4.9	8.9	11.0	15.7	21.9	22.1	11.9	14.0
IFG	0.5	2.1	5.1	4.5	4.3	8.4	3.4	4.2
All subjects								
IGT	3.4	6.5	10.0	15.2	21.2	23.2	10.6	13.0
IFG	2.0	5.2	7.2	8.7	7.6	6.8	5.8	7.4

Data are %.

be men (44.9% [95% CI 44.0–45.8] vs. 51.3% [50.2–52.1]) and were slightly older (mean age 51.5 years [50.7–52.3] vs. 47.7 years [46.6–48.8]). Responders were more likely to be married (71.5% [68.8–74.2] vs. 67.0% [64.4–69.5]), English-speaking (96.0% [94.6–97.4] vs. 93.6% [91.2–96.3]), and born in the U.K. (11.3% [9.7–12.8] vs. 8.2% [7.2–10.2]) (11). Also, the percentage of those who suspected they had diabetes was higher in responders than nonresponders to the biomedical examination (1.5% [1.3–1.7] vs. 0.5% [0.4–0.7]). However, given the low prevalence of those who suspected they had diabetes, this observed difference would be expected to have only a negligible effect on total diabetes prevalence estimates (11).

Diabetes

The total diabetes prevalence (known and newly diagnosed cases) was 7.4% (5.9–8.8) (Table 1). The prevalence was 0.3% in the youngest age group (25–34 years), increasing to 23.0% in those aged ≥75 years. The prevalence of KDM was 3.7%, and that of NDM was 3.7%. Therefore, half of all those identified as having diabetes were undiagnosed, and this varied little across the age groups.

Impaired glucose metabolism (IFG and IGT)

Until 75 years of age, the prevalence of IFG was found to be considerably higher in men than women. Overall, the prevalence of IFG was 8.1% in men and 3.4% in women (5.8% in total; Table 2). For IGT, there was less of a difference between men and women, but it was more common in women (11.9 vs. 9.2%) and 10.6% in total.

Total glucose intolerance

The prevalence of abnormal glucose tolerance (individuals with IGT, IFG, or diabetes) in men was 25.3%, whereas in women, the frequency was 22.2%. The total prevalence of abnormal glucose tolerance was 23.7%. The prevalence of abnormal glucose tolerance was 5.7% in the youngest age group (25–34 years), increasing to 53.1% in those aged ≥75 years.

Table 3 shows prevalence data comparing the American Diabetes Association (ADA) criteria for epidemiological studies (14), based on FPG ≥7.0 mmol/l, with the WHO criteria, based on the OGTT (12). With the fasting criterion alone, the prevalence of newly diagnosed diabetes was 1.8%—substantially lower than the 2.9% rate using 2hPG alone or the 3.7% using the FPG and 2hPG criteria based on WHO recommendations. Furthermore, there were differences in the individuals who were identified by the two tests. Of all those with NDM, only 28.3% were diabetic on both FPG and 2hPG criteria, and only 15.6% of all individuals with IGT or IFG had both conditions.

Table 3—Prevalence of categories of glucose tolerance according to FPG and 2hPG diagnostic criteria for the Australian population

FPG (mmol/l)	2hPG (mmol/l)			Total
	<7.8	7.8–11.1	≥11.1	
<6.1	76.3	8.0	1.0	85.4
6.1–6.9	5.7	2.6	0.8	9.1
≥7.0	0.3	0.5	1.0	1.9
Total	82.3	11.1	2.9	96.3*

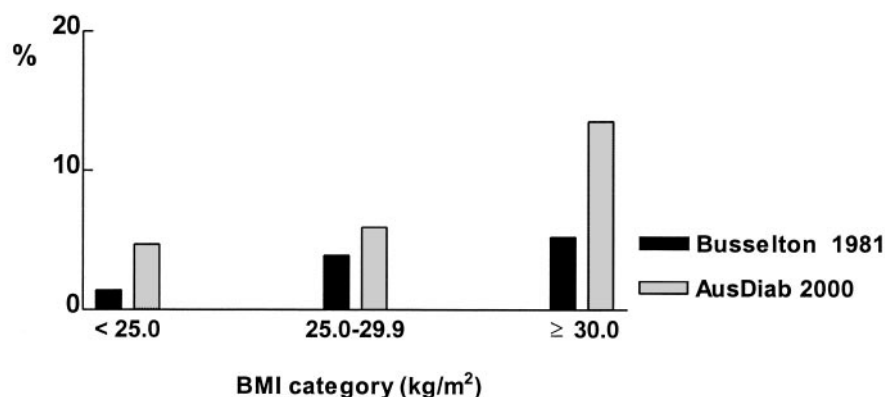
Data are %. *Total value is 96.3%, because 3.7% of the population had previously diagnosed diabetes.

Secular trends: 1981 to 1999–2000

Using the diagnostic criteria of the Busselton survey (8) and after age/sex adjustment of both populations to the 1998 Australian population, the prevalences of known diabetes among men was 4.8% for the AusDiab and 2.9% for the Busselton survey populations. For women, the comparative prevalences were 4.0% and 2.8%. For NDM, the prevalences were 2.7% for the AusDiab and 0.7% for the Busselton men and 2.9% for the AusDiab and 0.7% for the Busselton women. Overall, the total prevalence of diabetes had increased from 3.4 to 7.2% since 1981. Using the same diagnostic criteria, the prevalence of diabetes was 7.3% in the seven rural towns ($n = 2,186$) that were included in AusDiab and 6.1% in the state of Western Australia ($n = 1,542$).

Figure 1 shows the prevalence of diabetes by BMI category for both the Busselton and AusDiab surveys. It shows that for each category of BMI, there has been an increase in diabetes prevalence between the two surveys. Logistic regression (Table 4) shows that when the two data sets were combined, BMI was a significant predictor of diabetes status, but survey (AusDiab versus Busselton) was also an independent predictor. Participants in AusDiab were more than twice as likely as Busselton participants to have diabetes, even after accounting for the effects of BMI, age, and sex. This held true when men and women were analyzed separately and when individuals with previously diagnosed diabetes were excluded from the analysis.

CONCLUSIONS— AusDiab is the largest national diabetes prevalence study in the developed world to have used the OGTT. This study has revealed that Australia has one of the highest recorded prevalences of diabetes for a developed



Mean BMI

Busselton	22.5	27.1	33.0
AusDiab	22.4	27.3	34.1

Figure 1—Diabetes prevalence (KDM and NDM) according to survey and BMI. Mean values for each BMI category are provided for both surveys. Prevalence data on BMI were adjusted for each population to the 1998 Australian resident population.

nation. When IFG and IGT are also considered, almost one in four adult Australians has abnormal glucose tolerance. The prevalence of diabetes is similar to that reported from the U.S. (7) for non-Hispanic whites (7.3% for those aged ≥20 years) but not as high as rates reported for Hispanics (7), Asian Indians (6,15), American Pima Indians (16), or Micronesian Nauruans (17). The prevalence in Australia seems to be higher than in northern Europe. In the Hoorn study of Dutch people aged 50–74 years (18), the prevalence was 8.3%, and among 60-year-old Danes, the prevalence was 12.3% in men and 6.8% in women (19). These figures are both lower than the relevant age-specific data for AusDiab.

The study has also demonstrated a significant secular increase (more than a doubling over 20 years) in the prevalence of diabetes in Australia, which is con-

sistent with increasing trends in self-reported diabetes prevalence reported previously (10). Given the high prevalence of IGT, it can reasonably be expected that the prevalence of diabetes will continue to increase in Australia in the near future. Some caution, however, is required in comparing the current AusDiab data with the earlier Busselton results. The primary limitation is the survey sample. Busselton is a rural town in Western Australia, which even after adjustment for age, sex, and BMI, may not be entirely representative of the national picture in 1981. However, a major increase in prevalence is apparent (3.4 vs. 7.2%), and this approximate doubling of the prevalence since 1981 was the same whether Busselton was compared with the whole AusDiab sample, with other rural towns, or with the state of Western Australia. Marked increases in diabetes prevalence

over the last three decades have also been reported in a variety of developed and developing populations (6,7,20,21). Despite the relatively modest response rate achieved, the current AusDiab results are in keeping with a recent (1992–1996) large population-based study from the Australian state of Victoria (22). In that study, 5.1% of the population aged ≥40 years reported previously diagnosed diabetes. This compares to 5.4% for the same age group in AusDiab.

Some of the causes for the increasing diabetes prevalence are obvious. Aging populations will be expected to have higher prevalences of age-related diseases, such as type 2 diabetes. Obesity is strongly linked to diabetes, and has been observed to have increased in many countries over recent decades, including Australia (23). Indeed, the intimate relationship between diabetes and obesity has given rise to the term “diabesity” to characterize the close association of these two disorders (24).

Interestingly, this study, by comparing the current data with the earlier Australian data (Busselton 1981) (22), while confirming the importance of obesity in the etiology of type 2 diabetes, showed that neither this nor increasing age fully explained the difference in diabetes prevalence between the two surveys. One reason for this may have been the obesity measure used. Only BMI was measured in 1981, whereas it is recognized now that measures of central obesity, such as waist circumference or waist-to-hip ratio, show better correlations with components of the Metabolic Syndrome (25) and with mortality than does BMI (26). Another possible explanation is duration of obesity. Obesity is now being seen at younger ages than in the past (27,28), and it is therefore possible that those who were obese in the AusDiab population had

Table 4—Logistic regression identifying independent predictors of diabetes (KDM and NDM)* in a combined data set of AusDiab (1999–2000) and Busselton (1981)

Variable as diabetes predictor	β	95% CI for β	Odds ratio (95% CI)	P value
Sex (male versus female)	0.273	0.14–0.41	1.31 (1.15–1.50)	<0.001
Age (per year)	0.063	0.058–0.069	1.07 (1.06–1.07)	<0.001
Survey (AusDiab versus Busselton)	0.851	0.64–1.06	2.34 (1.90–2.90)	<0.001
BMI (per unit increase)	0.117	0.10–0.13	1.12 (1.10–1.14)	<0.001
Constant	–8.90	–9.45 to –8.35		

β is the regression coefficient for the exponential. Sex, age, survey site, and BMI were the only variables entered into the regression. NDM was identified by 2hPG only in both surveys.

been obese for longer than those in the Busselton survey. Duration of obesity is known to be important in the risk of diabetes (29).

Although obesity is recognized as an obvious factor associated with the increasing rates of diabetes (20,21,30), the role of physical inactivity is likely to be important and closely inter-related (31). Although there are no long-term trend data for physical activity patterns in Australia, there is evidence elsewhere that the frequency of physical inactivity has steadily increased in recent decades and shows very close parallels with the rising prevalence of obesity (31). In observational epidemiological studies, leisure time physical activity seems to provide strong protection against the development of type 2 diabetes. This is clearly established in both cross-sectional and prospective studies in women and in men, where its effect is independent of obesity (32,33). The link between physical activity and diabetes is further strengthened by intervention studies. Three trials have shown that lifestyle intervention among obese subjects with IGT reduces the rate of progression to diabetes by 40–60% over a 3- to 6-year period (34–36). The impact of increasing physical activity was clearly additive to that of dietary changes (34,35) and was apparent even in those who were unable to lose weight (35). Notably, in the AusDiab participants, 50% of adults undertook little or no physical activity on any regular basis (37).

Type 2 diabetes has traditionally been considered a disease of the middle-aged and elderly. The current data continue to show a clear relationship between age and diabetes prevalence, but whereas earlier Australian studies such as Busselton showed that the increase in diabetes prevalence only begins in those older than 50 years (8), the current data show that this increase begins in the 35- to 44-year age group.

This study continues to show that the number of people with undiagnosed diabetes remains high. Despite increasing awareness of the problems of type 2 diabetes, our findings showed that 50% of all those with diabetes were undiagnosed, although like many other epidemiological studies, it is possible that this could be an overestimate, because for clinical purposes, a second measurement within the diabetic range is required to confirm the diagnosis of diabetes. Part of the reason for this finding may lie in

the high proportion of people whose diabetes was only diagnosed based on the 2hPG. Of all those with undiagnosed diabetes, 50.2% had a nondiabetic FPG. This distribution is similar to that seen in a range of other populations (38–40) and emphasizes the need to maintain the OGTT as part of routine clinical practice as well as epidemiological studies. This is especially important, because it now seems that 2hPG is a stronger predictor of mortality and cardiovascular disease than FPG (41,42).

AusDiab was designed to obtain national prevalence estimates and did not attempt to provide a representative picture of diabetes in the Indigenous population. This minority group constitutes ~2% of the total Australian population but only 0.8% of the AusDiab sample (13). Diabetes is an enormous health problem in Australian Aborigines and Torres Strait Islanders; the overall prevalence is estimated to be 10–30% (43). Furthermore, the associated macrovascular and microvascular complications result in significant premature mortality and ill health among the Australian Indigenous population (44). Further research, including sample surveys using the AusDiab methodology, is planned to address the significant gap in our knowledge of the complex mechanisms that underlie the high prevalence of diabetes and its complications in Aboriginal and Torres Strait Islander communities.

In summary, this study shows a high prevalence of diabetes, IGT, and IFG and a doubling of the prevalence of diabetes within two decades. It is one of the first studies to demonstrate such a dramatic secular increase in a developed nation. Because there are few national studies from developed countries, AusDiab should ring alarm bells for governments and public health planners. Diabetes, together with its associated complications and diseases, is set to be one of the major contributors to health costs in all nations during the 21st century.

Acknowledgments—J.E.S. received a grant from the Institute for Diabetes Discovery, Branford, CT.

We thank the following for their support of the study: The Commonwealth Department of Health and Aged Care, Eli Lilly (Australia), Janssen-Cilag (Australia), Knoll Australia, Merck Lipla, Alphapharm, Merck Sharp & Dohme (Australia), Roche Diagnostics, Servier Laboratories (Australia), SmithKline Beecham Interna-

tional, Pharmacia and Upjohn, BioRad Laboratories, HITECH Pathology, the Australian Kidney Foundation, Diabetes Australia (Northern Territory), Queensland Health, South Australian Department of Human Services, Tasmanian Department of Health and Human Services, Territory Health Services, Victorian Department of Human Services and the Health Department of Western Australia. Also, for their invaluable contribution to the field activities of AusDiab, we thank Annie Allman, Marita Dalton, Adam Meehan, Claire Reid, Alison Stewart, Robyn Tapp, and Fay Wilson.

APPENDIX— The AusDiab Steering Committee consists of Dr. R. Atkins, Dr. S. Bennett, Dr. S. Chadban, Prof. S. Colagiuri, Dr. M. de Courten, Dr. M. D'Embden, Dr. D. Dunstan, Prof. T. Dwyer, Dr. D. Jolley, Dr. P. Magnus, Prof. J. Mathews, Dr. D. McCarty, Prof. K. O'Dea, Dr. P. Phillips, Dr. P. Popplewell, Mr. I. Kemp, Prof. H. Taylor, Prof. T. Welborn, and Prof. P. Zimmet.

References

1. Amos A, McCarty D, Zimmet P: The rising global burden of diabetes and its complications: estimates and projections to the year 2010. *Diabet Med* 14:S1–S85, 1997
2. King H, Aubert R, Herman W: Global burden of diabetes, 1995–2025: prevalence, numerical estimates and projections. *Diabetes Care* 21:1414–1431, 1998
3. Zimmet P, King H, Taylor R, Raper LR, Balkau B, Borger J, Heriot W, Thoma K: The high prevalence of diabetes mellitus, impaired glucose tolerance and diabetic retinopathy in Nauru—the 1982 survey. *Diabetes Res* 1:13–18, 1984
4. Knowler W, Bennett P, Hamman R, Miller M: Diabetes incidence and prevalence in Pima Indians: a 19-fold greater incidence than in Rochester, Minnesota. *Am J Epidemiol* 108:497–504, 1978
5. O'Dea K: Westernisation, insulin resistance and diabetes in Australian Aborigines. *Med J Aust* 155:258–264, 1991
6. Ramachandran A, Snehalatha C, Latha E, Vijay V, Viswanathan M: Rising prevalence of NIDDM in an urban population in India. *Diabetologia* 40:232–237, 1997
7. Harris MI, Flegal KM, Cowie CC, Eberhardt MS, Goldstein DE, Little RR, Wiedmeyer HM, Byrd-Holt DD: Prevalence of impaired fasting glucose, and impaired glucose tolerance in U.S. adults: the Third National Health and Nutrition Examination 1988–1994. *Diabetes Care* 21:518–524, 1998
8. Glatthaar C, Welborn TA, Stenhouse NS, Garcia-Webb P: Diabetes and impaired glucose tolerance: a prevalence estimate based on the Busselton 1981 survey. *Med*

- J Aust* 143:436–440, 1985
9. Guest C, O'Dea K, Hopper J, Nankervis A, Larkins R: The prevalence of glucose intolerance in Aborigines and Europids of south-eastern Australia. *Diabetes Res Clin Pract* 15:227–235, 1992
 10. Welborn T, Knuiiman M, Bartholomew H, Whittall D: 1989–90 National Health Survey: prevalence of self-reported diabetes in Australia. *Med J Aust* 163:129–132, 1995
 11. Dunstan D, Zimmet P, Welborn T, Cameron A, Shaw J, de Courten M, Jolley D, McCarty D, on behalf of the AusDiab Steering Committee: The Australian Diabetes, Obesity and Lifestyle Study (AusDiab): methods and response rates. *Diabetes Res Clin Pract* 2002 (In press)
 12. World Health Organization: *Definition, Diagnosis and Classification of Diabetes: Mellitus and its Complications. Part 1: Diagnosis and Classification of Diabetes Mellitus*. Geneva, Department of Noncommunicable Disease Surveillance; 1999
 13. Australian Bureau of Statistics: *Population by Age and Sex, Australian States and Territories*. Canberra, Australian Bureau of Statistics, 1999
 14. 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* 20:1183–1197, 1997
 15. Dowse GK, Gareeboo H, Zimmet PZ, Alberti KG, Tuomilehto J, Fareed D, Brissonnette LG, Finch CF: High prevalence of NIDDM and impaired glucose tolerance in Indian, Creole and Chinese Mauritians. *Diabetes* 39:390–396, 1990
 16. Bennett P, Burch T, Miller M: Diabetes mellitus in American (Pima) Indians. *Lancet* 2:125–128, 1971
 17. Zimmet P, Taft P, Guinea A, Guthrie W, Thoma K: The high prevalence of diabetes mellitus on a Central Pacific island. *Diabetologia* 13:111–115, 1977
 18. Mooy J, Grootenhuys P, de Vries H, Valkenburg HA, Bouter LM, Kostense PJ, Heine RJ: Prevalence and determinants of glucose intolerance in a Dutch Caucasian population: the Hoorn study. *Diabetes Care* 18:1270–1273, 1995
 19. Drivsholm T, Ibsen H, Schroll M, Davidsson M, Borch-Johnsen K: Increasing prevalence of diabetes mellitus and impaired glucose tolerance among 60-year-old Danes. *Diabet Med* 18:126–132, 2001
 20. Midthjell K, Krüger Ø, Holman J, Tverdal A, Claudi T, Bjorndal A, Magnus P: Rapid changes in the prevalence of obesity and known diabetes in an adult Norwegian population. *Diabetes Care* 22:1813–1820, 1999
 21. Collins V, Dowse G, Toelue P, Imo TT, Aloaina FL, Spark RA, Zimmet PZ: Increasing prevalence of NIDDM in the Pacific island population of Western Samoa over a 13-year period. *Diabetes Care* 17: 288–296, 1994
 22. McKay R, McCarty C, Taylor H: Diabetes in Victoria, Australia: the visual impairment project. *Aust N Z J Public Health* 24: 565–569, 2000
 23. World Health Organization: *Obesity: Preventing and Managing the Global Epidemic. Report of a WHO Expert Committee*. Geneva, World Health Org., 1997
 24. Ziv E: Psammomys obesus: nutritionally induced NIDDM-like syndrome on a “thrifty gene” background. In *Lessons From Animal Diabetes*. London, Smith-Gordon; 1995, p. 285–300
 25. Haffner S, Stern M, Hazuda H, Pugh J, Paterson J: Do upper-body and centralized adiposity measure different aspects of regional body-fat distribution? Relationship to non-insulin dependent diabetes mellitus, lipids, and lipoproteins. *Diabetes* 36:43–51, 1987
 26. Larsson B, Svardsudd K, Welin L, Welhelmsen L, Bjorntorp P, Tibblin G: Abdominal adipose tissue distribution, obesity, and risk of cardiovascular disease and death: 13 year follow up of participants in the study of men born in 1913. *BMJ* 288:1401–1404, 1984
 27. Troiano R, Flegal K, Kuczumski R, Campbell S, Johnson C: Overweight prevalence and trends for children and adolescents: the National Health and Nutrition Examination Survey 1963 to 1991. *Arch Pediatr Adolesc Med* 149:1085–1091, 1995
 28. Magarey A, Daniels L, Boulton T: Prevalence of overweight and obesity in Australian children and adolescents: reassessment of 1985 and 1995 data against new standard international definitions. *Med J Aust* 174:561–564, 2001
 29. Modan M, Karasik A, Halkin H, Fuchs Z, Lusky A, Shitrit A, Modan B: Effect of past and concurrent body mass index on prevalence of glucose intolerance and type 2 (non-insulin dependent) diabetes and on insulin response: the Israeli Study of Glucose Intolerance, Obesity and Hypertension. *Diabetologia* 29:82–89, 1986
 30. Mokdad A, Bowman B, Ford E, Vinicor F, Marks J, Kaplan J: The continuing epidemic of obesity and diabetes in the United States. *JAMA* 286:1195–2000, 2001
 31. Prentice A, Jebb S: Obesity in Britain: glutony or sloth? *BMJ* 311:437–439, 1995
 32. Manson J, Rimm E, Stampfer M, Colditz GA, Willett WC, Krolewski AS, Rosner B, Hennekens CH, Speizer FE: Physical activity and incidence of non-insulin-dependent diabetes mellitus in women. *Lancet* 338:774–778, 1991
 33. Helmrich SP, Ragland DR, Leung RW, Paffenbarger RS: Physical activity and reduced occurrence of non-insulin-dependent diabetes mellitus. *N Engl J Med* 325: 147–152, 1991
 34. Pan XR, Li GW, Hu YH, Wang JX, Yang WY, An ZX, Hu ZX, Lin J, Xiao JZ, Cao HB, Liu PA, Jiang XG, Jiang YY, Wang JP, Zheng H, Zhang H, Bennett PH, Howard BV: Effects of diet and exercise in preventing NIDDM in people with impaired glucose tolerance: the Da Qing IGT and Diabetes Study. *Diabetes Care* 20:537–544, 1997
 35. Tuomilehto J, Lindstrom J, Eriksson J, Valle TT, Hamalainen H, Ilanne-Parikka P, Keinanen-Kiukaanniemi S, Laakso M, Louheranta A, Rastas M, Salminen V, Uusitupa M, Finnish Diabetes Prevention Study Group: Prevention of type 2 diabetes mellitus by changes in lifestyle among subjects with impaired glucose tolerance. *N Engl J Med* 344:1343–1350, 2001
 36. Diabetes Prevention Program Research Group: Reduction in the incidence of type 2 diabetes with lifestyle intervention on metformin. *N Engl J Med* 346:393–403, 2002
 37. Dunstan D, Zimmet P, Welborn T, Sicree R, Armstrong T, Atkins R, Cameron A, Shaw J, Chadban S, on behalf of the AusDiab Steering Committee: *Diabetes and Associated Disorders in Australia 2000: The Accelerating Epidemic*. Melbourne, International Diabetes Institute, 2001
 38. DECODE Study Group on behalf of the European Diabetes Epidemiology Group: Is fasting glucose sufficient to define diabetes? Epidemiological data from 20 European studies. *Diabetologia* 42:647–654, 1999
 39. Shaw J, de Courten M, Boyko E, Zimmet P: Impact of new diagnostic criteria for diabetes in different populations. *Diabetes Care* 22:762–766, 1999
 40. Harris M, Eastman R, Cowie C, Flegal K, Eberhardt M: Comparison of diabetes diagnostic categories in the US population according to 1997 American Diabetes Association and 1980–1985 World Health Organization diagnostic criteria. *Diabetes Care* 20:1859–1862, 1997
 41. DECODE Study Group: Glucose tolerance and mortality: comparison of WHO and American Diabetes Association diagnostic criteria. *Lancet* 354:617–621, 1999
 42. Shaw J, Hodge A, de Courten M, Chitson P, Zimmet P: Isolated post-challenge hyperglycaemia confirmed as a risk factor for mortality. *Diabetologia* 42:1050–1054, 1999
 43. de Courten M, Hodge A, Dowsett GHK, Vickery J, Zimmet P: *Review of the Epidemiology, Aetiology, Pathogenesis and Preventability of Diabetes in Aboriginal and Torres Strait Island Populations*. Canberra, OATSIH & DHFS, 1998
 44. Hoy W, Kelly A, Jacups S, McKendry K, Baker P, MacDonald S, Wang Z, Punguatji N, Kerinaua J, Tipiloura E, Tipiloura E, Harrison C: Stemming the tide: reducing cardiovascular disease and renal failure in Australian Aborigines. *Aust N Z J Med* 29: 480–483, 1999