PNG Med J 2001 Sep-Dec;44(3-4):164-170

Diabetes in Australian Aboriginal and Torres Strait Islander peoples

KEVIN G. ROWLEY[1] AND KERIN O'DEA[2,3]

University of Melbourne, Victoria, Australia and Menzies School of Health Research, Tiwi, Australia

SUMMARY

Type 2 diabetes arises from a complex and multifactorial set of factors, including genetic susceptibility, behaviour (including diet and exercise), early nutrition, obesity and psychosocial stress, leading to insulin resistance and pancreatic failure. These factors in turn are influenced by social and physical environmental factors. Each of these may be important determinants of the high prevalence and incidence of type 2 diabetes in Australian Aboriginal and Torres Strait Islander people. Public health interventions for primary and secondary prevention need to recognize this complexity. Although a reduction in the prevalence of obesity or diabetes in the short-medium term is rarely if ever achieved, there are documented examples of community-based programs which have been effective in reducing the risk of developing type 2 diabetes and its cardiovascular complications. Such interventions need to be community-directed and appropriate to local circumstances in order to be effective.

Prevalence, incidence and relationship to adiposity

Available evidence indicates that before the European colonization of Australia, obesity, diabetes and cardiovascular disease (CVD) were rare among Aboriginal and Torres Strait Islander people (1-3). Today, cardiovascular diseases are the leading cause of death among Indigenous Australians, and in the 25-44 year age group cardiovascular mortality is 10 times greater than for the wider Australian population (4). Diabetes, a major risk factor for CVD, is also occurring at epidemic rates in many communities. Reported prevalences of diabetes range up to 21% (5) of adults in Aboriginal and Torres Strait Islander communities. In an analysis of aggregated data collected in the course of community-based risk factor surveys in 15 remote communities in central and north-western Australia and Cape York (including over 2000 participants), we estimated an overall prevalence of diabetes of 15% (M. Daniel, K. G. Rowley, R. McDermott and K. O'Dea, Diabetes and impaired glucose tolerance in Aboriginal Australians: prevalence and risk, submitted manuscript). These surveys were conducted during the period 1983-1997 and the prevalence of impaired glucose tolerance (a mildly hyperglycaemic state associated with a high rate of progression to overt diabetes) was high (15%), so the figure of 15% for diabetes probably represents an underestimate of the current true prevalence. Furthermore, comparison of total population prevalences between Indigenous and non-Indigenous Australians can be quite misleading as it does not account for the very different population age structures and the early (and decreasing) age of onset of type 2 diabetes in Aboriginal people. For example, the prevalence of diabetes in the 25-34 year age group in one central Australian community was 13% compared to 0.3% in the Australian population (6), which represents an almost 40-fold higher prevalence. In the same community diabetes in the 15-24 year age group, almost absent in 1987, was becoming apparent by 1995 in line with a sharp increase in the prevalence of obesity (5). A rapidly increasing prevalence of diabetes has been observed in some (5), but not

1 University of Melbourne, Department of Medicine, St Vincent's Hospital, Fitzroy, Victoria 3065, Australia

2 Menzies School of Health Research, PO Box 41096, Casuarina, Northern Territory 0811, Australia

3 Corresponding author

all (7), communities. The age-specific prevalence of diabetes in the aggregated dataset cited above is shown in Figure 1. All cases identified in these surveys were of type 2 (adult-onset) diabetes. Extremely high prevalences occur in the age range 35-74 years. The apparent decrease in prevalence after age 64 years is at least partly due to survival bias: there is an extremely high mortality from cardiovascular and other chronic diseases in the preceding decades of life. A young age of onset for type 2 diabetes is of major concern because of the greatly increased potential for the development of complications such as nephropathy, retinopathy, peripheral vascular disease and heart disease at a relatively young age. There is also a greatly increased risk of diabetes complicating pregnancies. While these data are not necessarily representative of Aboriginal people in other parts of Australia, similarly high prevalences have been reported for communities in south-eastern Australia (8). The prevalence of diabetes among Torres Strait Islander people was already high in 1960 (9), and remains so (10).

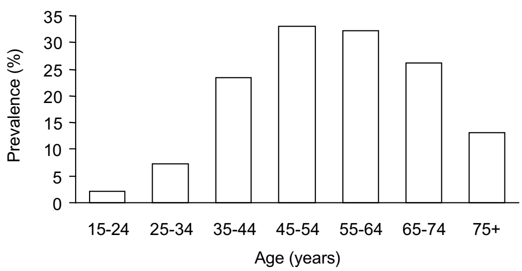

Figure 1. Age-specific prevalence of diabetes in Australian Aboriginal people, screened between 1983 and 1997 as part of community-based risk factor prevalence surveys (N = 818, 703, 426, 296, 227, 126 and 54 in order of increasing age categories). Derived from M. Daniel et al., submitted manuscript.

In all populations, a major risk factor for diabetes is excess body fat. The standard measure for defining individuals or groups as ‘overweight’ or ‘obese’ is the body mass index (BMI; body weight/height^2). BMI ranges defined as ‘healthy’ or otherwise have been derived from examination of health outcomes in populations of European descent. However, ethnic groups differ widely with respect to the amount of body fat associated with a particular BMI range. For example, for any given BMI, Aboriginal women from north-western Australia have a significantly greater amount of subcutaneous fat (11). We have observed similar results in other groups of Australian Aboriginal people (12) (L.S. Piers et al., submitted manuscript). The anatomical location of excess body fat is also important in conferring increased risk of diabetes: abdominal obesity, and in particular intra-abdominal as opposed to subcutaneous fat, is a stronger risk factor than is peripheral (hips and limbs) fat deposition. The studies cited above made use of skinfold measurements to examine body fat and its distribution, which does not account for intra-abdominal fat. Among those groups of Aboriginal people screened to date, abdominal fat deposition (indicated by waist-to-hip circumference ratio) is proportionately greater than in comparable Europeans. Torres Strait Islanders exhibit greater prevalence of obesity and greater mean BMI than Aboriginal people (13), but this difference only partly reflects a greater degree of adiposity. In common with Pacific peoples, Torres Strait Islanders have a greater degree of lean tissue mass (muscle) for any given BMI compared to Europeans. Hence the ‘healthy’ BMI range is likely to be different again for this ethnic group.

Consistent with these different relationships of BMI to body fat, the incidence of diabetes in a cohort of central Australian Aboriginal people (15 years and older) was high in the so-called ‘healthy’ BMI range of 20-25 kg/m^2: 11 cases per 1000 person-years, compared to less than 3 cases per 1000 person-years in (older, and including the full range of BMI) populations of European descent (14). Higher BMI categories were associated with greater risk of developing diabetes (Figure 2): the relative risk (95% confidence interval) for the BMI categories 25.0-28.9, 29.0-32.9 and 33.0+ kg/m^2 compared to BMI <25.0 kg/m^2 were 3.3 (1.5-7.0), 2.7 (1.1-6.8) and 4.4 (1.7-11.6) respectively (14). No cases of incident diabetes were observed among Aboriginal persons with a BMI less than 22 kg/m^2 at baseline. In cross-sectional analyses, BMI was also strongly related to diabetes and other CVD risk factors commonly recognized as components of the ‘metabolic syndrome’: dyslipidaemia, hypertension and albuminuria (12). A healthy BMI range for Torres Strait Islanders with respect to diabetes risk remains to be definitively determined.

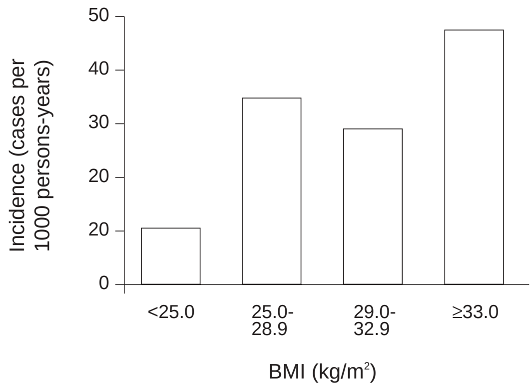

Figure 2. Incidence of diabetes according to BMI in a cohort of Aboriginal people followed for up to 8 years (n = 464, aged 15 years and older at baseline). Data are adjusted for age, gender and community of residence (14).

On the aetiology of obesity and diabetes

The aetiology of type 2 diabetes is complex and multifactorial (Figure 3). As physical activity decreases and body fat increases, the sensitivity to the glucoregulatory actions of insulin is decreased, leading to lower rates of uptake of glucose by skeletal muscle and a lack of inhibition of hepatic gluconeogenesis. As a result, insulin secretion from the pancreas must increase to achieve the same circulating concentrations of glucose, that is, the individual becomes more insulin resistant. Insulin resistance precedes glucose intolerance, and compensatory increases in pancreatic insulin release are sufficient to prevent diabetes in many individuals. Hyperglycaemia occurs when insulin release cannot be increased sufficiently to overcome insulin resistance. Overt diabetes is manifested when pancreatic β-cell failure becomes evident as insulin secretion falls. The precise cause of this pancreatic β-cell failure remains unclear, but oxidative damage may contribute. Important pro-oxidant factors common in many high-risk populations are smoking and hyperglycaemia and a low intake of dietary antioxidants.

Insulin resistance and high incidence of type 2 diabetes are common to many populations undergoing a transition from traditional lifestyles to more sedentary, urbanized (‘westernized’) modes of living. Given the diversity of populations exhibiting high rates of diabetes upon westernization, one could propose that populations of European origin are unusual in being particularly resistant to type 2 diabetes (although diabetes prevalence is also rising in many populations of European descent as they become more obese). The nature of historical and nutritional factors which may have promoted selection for genes

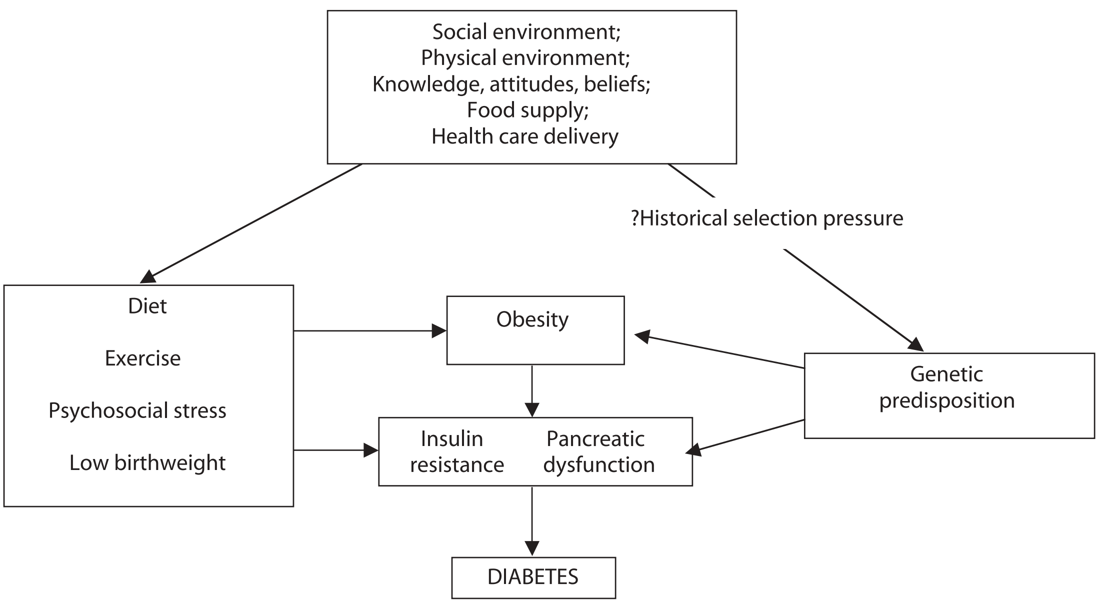

Figure 3. Some known and potential contributors to diabetes risk. The list is not exhaustive and does not identify the many complex interactions which may occur between these factors.

conferring variable insulin sensitivity between populations is not clear. The characteristics of populations with a high prevalence of diabetes and other metabolic syndrome components has often been proposed to arise from a 'feast and famine' dietary regime. In this model, a genotype which allowed efficient deposition of adipose tissue during times of food abundance would confer a survival advantage during times of scarcity. In hunter-gatherer societies, a 'feast' would typically consist of a large, non-domesticated animal (ie, high protein, low fat, low carbohydrate). Under such circumstances, insulin resistance could promote the efficient conversion of amino acids to a) glucose (hepatic gluconeogenesis) as a readily available source of energy, and b) fat for storage in adipose tissue, available for use during times of scarcity (3). Exposure of individuals exhibiting this metabolic efficiency to excessive caloric intake and low physical activity then unmasks a predisposition to obesity and diabetes.

It is apparent that, whatever the cause, there is variation within and between populations in the risk of developing diabetes. It is becoming clear that no single gene polymorphism can account for variation in diabetes risk. A polygenic origin is more plausible but is yet to be identified definitively, despite great interest in identifying candidate genes. The potential for complex gene-gene and gene-environment interactions also needs consideration. Hence the identification of a genotype which can predict diabetes with sufficient sensitivity and specificity is somewhat problematic. In terms of public health and the design of community-based interventions to reduce diabetes risk, genetic information is of limited utility at the present time.

Regardless of the historical factors determining insulin sensitivity in modern populations, insulin resistance is clearly a determinant of diabetes risk. Two major correlates of insulin resistance are body fat and physical activity. The two are not independent, habitual physical activity (energy expenditure) being one of the factors directly determining body fat deposition. Habitual diet (both quality and quantity) also determines body weight, and diet in turn is determined by nutrition knowledge, behaviour and the available food supply. Greater consumption of vegetables and fruit is associated with lower mean BMI and lower risk of diabetes at the population level. Saturated fat intake may also adversely affect insulin sensitivity. Weight loss and increased exercise, separately or in combination, unequivocally improve insulin sensitivity and reduce the risk of diabetes (15-17).

The degree to which early life (intrauterine and early childhood) environmental influences modulate the expression of insulin resistance and diabetes in adulthood is also a matter of some interest (18). Low birthweight has been associated with greater risk of diabetes in adulthood in several cohorts. Furthermore, offspring of diabetic mothers, who may exhibit high rather than low birthweight, are also at increased risk of insulin resistance, obesity and diabetes as adults. This may arise as a consequence of the maternal diabetic milieu stimulating fetal hyperinsulinaemia. Experimental evidence suggests that inappropriate fetal nutrition can compromise pancreatic function, further increasing risk of subsequent diabetes. The potential for permanent alteration of gene expression in response to early life environment is likely to become an important area of research in the aetiology of diabetes. The implications for populations which experience simultaneously high prevalences of both low birthweight and adult obesity are serious. High prevalences and early age of onset of obesity and diabetes among women occurs in Australian Aboriginal populations and other high-risk groups (19).

In western industrialized countries, chronic disease risk is inversely related to socioeconomic indicators. This is not only due to availability of material resources and differences in health behaviours such as smoking, but is believed to also arise from psychosocial factors related to degree of control ('mastery') over life circumstances. The social gradient identified in studies such as the Whitehall Study is thought to be mediated partly through the hypothalamic-pituitary axis (the 'stress' pathway), leading to excessive cortisol release. It is manifested as increasing abdominal fat deposition, insulin resistance, circulating fibrinogen and glycaemia and consequently increased cardiovascular risk (20-22).

Public health interventions to address diabetes and its complications

Hence a number of factors directly linked to the social and physical environment in which individuals live can contribute to diabetes risk: psychosocial stress, opportunities for physical activity and food supply (especially availability of fresh vegetables and fruit). Health promotion initiatives to raise awareness of diet, exercise and smoking need to be designed and delivered in conjunction with local workers in order for messages to be accessible and realistic in the context of broader social circumstances. For example, in Torres Strait supplies of fresh vegetables and fruit were reported to be of extremely limited quality and quantity despite a tradition of extensive local production. This was partly due to long shipping times from southern Australian sources and quarantine regulations (23). This limited availability is common to many remote areas of Australia. Thus initiatives to improve dietary quality in remote communities require modification of food supply in addition to health promotion and education. In Torres Strait, reference to traditional gardening practices has led to the implementation of programs such as 'Gadin KaiKai' which promotes production of local food varieties (10).

We have evaluated several community-directed intervention programs which were associated with significant improvements in population risk of diabetes. In Western Australia, in the Looma Healthy Lifestyle program, a community-directed project to reduce the risk of chronic disease, substantial improvements in risk factors related to diet and exercise have been documented. These included fasting insulin concentration (a marker of insulin sensitivity, Figure 4), cholesterol (which is related to saturated fat intake) and diet-derived antioxidants (24,25). This was achieved through a combination of locally designed and implemented health promotion initiatives including regular sporting competitions, appointment of a community member as manager of the single community store with a mandate to improve the quality of food supplied and policy changes at the level of the Community Council. In contrast to these improvements, there was no significant change in the prevalence of obesity or diabetes over a 4-year period (Figure 4). This is not surprising, however, as well-established diabetes is generally considered an irreversible condition. For this reason a focus on secondary prevention through improvements in risk factors for the major complications of diabetes remains an important successful outcome in the short to medium term. There are few if any examples of falls in the prevalence of obesity in populations, outside situations of war or famine. Thus, as noted below, programs targeted at the primary prevention of diabetes must focus on the prevention of overweight and obesity in children and adolescents and throughout adult life.

A similar lack of change in diabetes prevalence was observed in a central Australian population (7). In this community, consisting of numerous outstations located largely on traditional lands – most of which were remote from services such as a store – there was a relatively low baseline prevalence of diabetes (9%, still well above the national Australian figure in age-adjusted terms) which remained static over a 7-year period. During this time, the community initiated a primary prevention program, which was associated with a major decrease in the prevalence of impaired glucose tolerance from 23% to 10%. This suggested that the population was at substantially lower risk of incident diabetes. Regarding trends in BMI, there was a significant increase in average BMI only among those community members who were resident adjacent to a store: those people living on more remote homelands did not show an increase in average BMI. This observation speaks to the importance of the quality of food supply and the role of habitual physical activity patterns incorporated into daily routines.

These and other successful programs (26) are characterized by high levels of community control over intervention processes, as opposed to the imposition of programs designed and run by external organizations. They also highlight the importance of setting realistic goals: a reduction in the prevalence of obesity and diabetes is not feasible in the short to medium term, whereas significant improvements in cardiovascular disease risk factors (smoking,

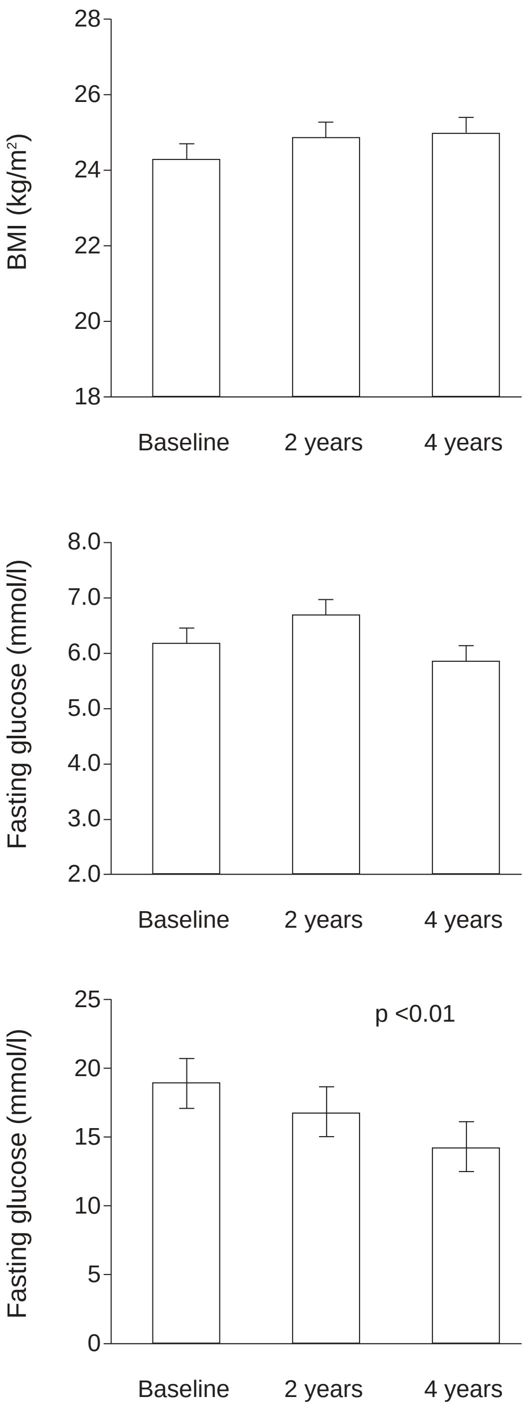

Figure 4. Trends in BMI and fasting plasma glucose and insulin over the course of a healthy lifestyle program in an Aboriginal community. Data are means (s.e.m.), except for insulin, which is geometric mean (95% confidence interval), adjusted for age and gender. N = 199, 181 and 125 at baseline, 2 years and 4 years respectively (24).

insulin resistance and diet-related risk factors such as plasma cholesterol, homocysteine and antioxidants) are achievable through modest improvements in diet, exercise and other health behaviours. Primary prevention of obesity and diabetes is required to ultimately reduce the prevalence of these conditions and their associated mortality from complications. Thus the aims of the Looma Healthy Lifestyle program, for example, eventually broadened to include a focus on improving nutrition education and practices among school-age children in order to prevent the development of obesity in adolescence and early adulthood. Major primary prevention programs are currently under way in indigenous communities in North America (27,28). Given the potential role of maternal nutrition and health in determining risk of diabetes in subsequent generations, interventions must also consider ways of preventing diabetes in young women and optimizing glycaemic control in diabetic pregnancies.

Conclusions

Diabetes and its complications arise from a multifactorial and complex set of risk factors related to genetics, behaviour, the social and physical environment, and influences of early life. Interventions at the population and community level should recognize this complexity. Interventions appropriate to local communities, designed and implemented in collaboration with community members, can achieve substantial improvements in modifiable risk factors for diabetes and other chronic diseases. However, if health outcomes are to be substantially improved at the population level, such interventions will need substantial long-term investment.

REFERENCES

1 **Abbie AA.** Studies in Physical Anthropology, Volumes I and II. Canberra: Australian Institute of Aboriginal Studies, 1975.

2 **Haddon AC.** Anthropological Expedition to the Torres Straits. Cambridge: Cambridge University Press, 1912.

3 **O'Dea K.** Diabetes in Australian Aborigines: impact of the western diet and lifestyle. *J Intern Med* 1992;232:103-117.

4 **Field P, Wakerman J, Nash K.** Report of the workshop to review current research and determine future research priorities for cardiovascular disease in rural and remote settings, and in Aboriginal and Torres Strait Islander people. National Heart

Foundation and Centre for Remote Health, Alice Springs, 2000.

5 **McDermott R, Rowley KG, Lee AJ, Knight S, O'Dea K.** Increase in prevalence of obesity and diabetes and decrease in plasma cholesterol in a central Australian Aboriginal community. *Med J Aust* 2000;172:480-484.

6 **Dunstan D, Zimmet P, Welborn T, Sicree R, Armstrong T, Atkins R, Cameron A, Shaw J, Chadban S.** Diabesity and associated disorders in Australia 2000. Melbourne: International Diabetes Institute, 2001.

7 **Rowley KG, Gault A, McDermott RG, Knight S, McLeay T, O'Dea K.** Reduced prevalence of impaired glucose tolerance and no change in prevalence of diabetes despite increasing BMI among Aboriginal people from a group of remote homeland communities. *Diabetes Care* 2000;23:898-904.

8 **Cameron WI, Moffitt PS, Williams DRR.** Diabetes mellitus in the Australian Aborigines of Bourke, New South Wales. *Diabetes Res Clin Pract* 1986;2:307-314.

9 **Winterbotham HJ.** Diabetes mellitus at Mabuiag Island, Torres Straits, 1960. *Med J Aust* 1961;1:780-781.

10 **Torres Strait and Northern Peninsula Area Health Council Inc.** Meriba Zageth for diabetes. Report of the Torres Strait and Northern Peninsula Area Health Council Diabetes Summit, Thursday Island, 1996.

11 **Rutishauser IHE, McKay H.** Anthropometric status and body composition in Aboriginal women of the Kimberley region. *Med J Aust* 1986;144:S8-S10.

12 **Rowley KG, Best JD, McDermott R, Green EA, Piers LS, O'Dea K.** Insulin resistance syndrome in Australian Aboriginal people. *Clin Exp Pharmacol Physiol* 1997;24:776-781.

13 **O'Dea K, Rowley KG.** Macrovascular disease risk factors and insulin resistance in Aboriginal and Torres Strait Islander people. *J Diabetes Complications,* in press.

14 **Daniel M, Rowley KG, McDermott R, Mylvaganam A, O'Dea K.** Diabetes incidence in an Australian Aboriginal population. An 8-year follow-up study. *Diabetes Care* 1999;22:1993-1998.

15 **Pan XR, Li GW, 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, Bennet 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* 1997;20:537-544.

16 **Eriksson KF, Lindgärde F.** Prevention of type 2 (non-insulin-dependent) diabetes mellitus by diet and physical exercise. The 6-year Malmö feasibility study. *Diabetologia* 1991;34:891-898.

17 **Tuomilehto J, Lindström J, Eriksson JG, Valle TT, Hämäläinen H, Ilanne-Parika P, Keinänen-Kiukaanniemi S, Laakso M, Louheranta A, Rastas M, Salminen V, Uusitupa M.** Prevention of type 2 diabetes mellitus by changes in lifestyle among subjects with impaired glucose tolerance. *N Engl J Med* 2001;344:1343-1350.

18 **Barker DJP.** The Wellcome Foundation Lecture, 1994. The fetal origins of adult disease. *Proc R Soc London B Biol Sci* 1995;262:37-43.

19 **O'Dea K.** Clinical implications of the 'thrifty genotype' hypothesis: where do we stand now? *Nutr Metab Cardiovasc Dis* 1997;7:281-284.

20 **Brunner EJ, Marmot MG, Nanchahal K, Shipley MJ, Stansfeld SA, Juneja M, Alberti KGMM.** Social inequality in coronary risk: central obesity and the metabolic syndrome. Evidence from the Whitehall II study. *Diabetologia* 1997;40:1341-1349.

21 **Räikkönen K, Keltikangas-Järvinen L, Aldercreutz H, Hautanen A.** Psychosocial stress and the insulin resistance syndrome. *Metabolism* 1996;45:1533-1538.

22 **Björntorp P.** Neuroendocrine perturbations as a cause of insulin resistance. *Diabetes Metab Res Rev* 1999;15:427-441.

23 **Leonard D, Beilin R, Moran M.** Which way kaikai blo umi? Food and nutrition in the Torres Strait. *Aust J Public Health* 1995;19:589-595.

24 **Rowley KG, Daniel M, Skinner K, Skinner M, White GA, O'Dea K.** Effectiveness of a community-directed 'healthy lifestyle' program in a remote Australian Aboriginal community. *Aust NZ J Public Health* 2000;24:136-144.

25 **Rowley KG, Su Q, Cincotta M, Skinner M, Skinner K, Pindan B, White GA, O'Dea K.** Improvements in circulating cholesterol, antioxidants, and homocysteine after dietary intervention in an Australian Aboriginal community. *Am J Clin Nutr* 2001;74:442-448.

26 **Lee AJ, Bonson APV, Yarmirr D, O'Dea K, Mathews JD.** Sustainability of a successful health and nutrition program in a remote Aboriginal community. *Med J Aust* 1995;162:632-635.

27 **Macaulay AC, Paradis G, Potvin L, Cross EJ, Saad-Haddad C, McComber A, Desrosiers S, Kirby R, Montour LT, Lamping DL, Leduc N, Rivard M.** The Kahnawake Schools Diabetes Prevention Project: intervention, evaluation, and baseline results of a diabetes primary prevention program with a native community in Canada. *Prev Med* 1997;26:779-790.

28 **American Journal of Clinical Nutrition.** Prevention of obesity in American Indian children: the Pathways study. *Am J Clin Nutr* 1999;69:745S-824S.