Diabetes, Hyperinsulinemia, and Hyperlipidemia in Small Aboriginal Community in Northern Australia

Kerin O'Dea, PhD
Robin J. Lion, DipHSc
Amanda Lee, BSc, GradDipDiet
Kathy Traianedes, BAppSc
John L. Hopper, PhD
Cheryl Rae, DipNFS, CertDiet

A small rural Aboriginal community in northern Australia was surveyed for diabetes, impaired glucose tolerance (IGT), hyperinsulinemia, and lipid levels. Of the 122 adults >17 yr of age who participated (95% response rate), 11.5% had diabetes, 7.4% had IGT, and the remaining 81.1% had normal glucose tolerance. Both diabetes and IGT were strongly age related. This high frequency of diabetes occurred, despite the population being relatively lean. Although the body mass index (BMI) increased with age in both men and women, only 25% of the population overall had BMI >25 kg/m^2. There were wide ranges of insulin responses to glucose, with the upper tertile of 2-h insulin levels being more than seven times higher than the lower tertile (144 ± 13 vs. 19 ± 1 mU/L). Hyperinsulinemia was associated with IGT, elevated triglycerides, and lower high-density lipoprotein cholesterol levels. Lipid abnormalities were much more frequent among men than women. Cholesterol levels were an average of 0.55 mM higher and triglycerides an average of 1.05 mM higher in men than in women, and both increased with age. In conclusion, this small isolated Aboriginal population from northern Australia had an unexpectedly high frequency of diabetes (in view of their relative leanness) in association with a high frequency of metabolic abnormalities indicative of insulin resistance (hyperinsulinemia, IGT, hypertriglyceridemia). *Diabetes Care* 13:830–35, 1990

The diabetes prevalence among Australians of European descent has been estimated to be 3.4% from the results of the most recent Busselton survey in western Australia (1). Prevalence rates two to six times higher have been reported for Aboriginal communities around the country (2–9), being highest in the most westernized communities (2,3,6) and lower in the least westernized groups (5,9) or in those with significant non-Aboriginal genetic admixture (7). No data are available on the prevalence of diabetes and related abnormalities of carbohydrate and lipid metabolism among Aborigines in the Northern Territory. Herein, we report the findings of a survey of diabetes and hyperlipidemia in a small isolated Aboriginal community in the northwest of the Northern Territory.

The community is situated on a Catholic mission that was established in 1956 and encompasses parts of their traditional country, which has helped the Aboriginal people who live there to retain strong links with the land and continue to hunt and gather traditional foods (mainly on weekends and the month-long bush holiday in the dry season). However, their dietary staples (refined sugar, flour, beef) are purchased at the local community store. Although this community is nominally "dry" (i.e., alcohol cannot be brought into the mission premises), alcohol is available regularly at the mission club, which is open 2 h/day. In addition, a hotel is located 4 km outside the mission, thus alcohol is freely available to those who want it. Alcohol consumption is much more common among men than women.

RESEARCH DESIGN AND METHODS

The subjects in this study came from an Aboriginal community situated on a river in the northwest region of the

From the Department of Human Nutrition, Deakin University, Geelong, Victoria; the Department of Medicine, Royal Melbourne Hospital, Victoria; the Northern Territory Department of Health and Community Services, Casuarina; and the Menzies School of Health Research, Casuarina, Northern Territory, Australia.

Address correspondence to Kerin O'Dea, PhD, Department of Human Nutrition, Deakin University, Victoria 3217, Australia.

Received for publication 5 October 1989 and accepted in revised form 28 March 1990.

Northern Territory (latitude 13° south, longitude 130° east) 150 km southwest of Darwin. The community is accessible by road, air, and from the sea. Food supplies are brought in by road, and air service is available 2 times/wk. The community has a health clinic staffed by community health-care nursing sisters and Aboriginal health-care workers. Other medical and allied health-care workers visit on a regular basis.

One hundred twenty-two adults >17 yr of age participated in the survey. This represented >95% of all adult residents in the community at the time.

Apparent food and nutrient intake in the community over the 3 mo immediately preceding the survey was estimated from invoices of all food supplied to the community store by use of the Microdiet software package, which is based on the British Food Tables (10), and incorporating Australian data where possible (11). Apparent per capita daily intake of food and nutrients was calculated by denomination of the mean population relying on the store for their food purchases over 3 mo. The total population of 262 comprised 134 adults and 128 children <18 yr of age. Results pertaining to food and nutrient profiles (the relative contributions of macronutrients to total energy intake) are independent of the population estimation and may be used with more confidence to describe the style of the diet.

Because this method does not include alcohol or bush foods as sources of energy and nutrients, it is an underestimate. However, both these items were impossible to quantify with any accuracy over the extended 3 mo of the store turnover analysis. The contribution of bush foods to total energy intake was considered to be small (averaging <5%) because half of the population seldom consumed any, and of the remainder, bush foods accounted for <50% of 1 day's food intake/wk. The women were more active in hunting and gathering of bush foods and as a result more physically active than the men. However, it was not possible to quantify physical activity levels in this population.

Alcohol was more of a problem. Wide variations between individuals and within individuals over time make an average daily consumption figure almost meaningless, even if it could be obtained with accuracy.

After an overnight fast of 10–12 h, subjects were tested either at the clinic or in their homes. A fasting blood sample was taken (12 ml for measurement of glucose, insulin, cholesterol, high-density lipoprotein cholesterol [HDL-chol], and triglycerides), after which the subjects drank 75 g glucose monohydrate dissolved in water. A second blood sample was taken 2 h later (2 ml for glucose and insulin measurements). Blood samples were centrifuged, and the plasma was stored frozen until it could be analyzed. The following anthropometric measurements were made: height, weight, and circumferences of the waist and hips (12).

Glucose concentrations were measured in fluoride heparin plasma by the glucose oxidase method. Immunoreactive insulin concentrations in fluoride heparin plasma were measured with kits purchased from Pharmacia (Uppsala, Sweden) with human insulin standard. The range of assay is 5–240 mU/L, with an interassay coefficient of variation of 5%. Fasting cholesterol and triglyceride concentrations were measured after enzymatic hydrolysis with a commercially available kit (Boehringer Mannheim, Mannheim, FRG) via a Cobas Bio centrifugal analyzer. HDL-chol levels were measured by the same method after precipitation of low-density lipoprotein and very-low-density lipoprotein with polyethylene glycol (6000, 15% wt/vol). The normal range of cholesterol concentrations in fasting plasma from Whites was 3.5–5.5 mM, 0.5–2.0 mM for triglycerides, and 0.8–2.0 mM for HDL-chol.

Diagnostic criteria for diabetes. World Health Organization criteria based on venous plasma glucose concentrations 2 h after 75 g oral glucose were used (13): diabetes, 2-h oral glucose tolerance 11.1 mM; impaired glucose tolerance (IGT), 2-h oral glucose between 7.8 and 11.1 mM; normal glucose tolerance, 2-h oral glucose tolerance <7.8 mM.

Statistical analysis. Statistical analyses (unpaired *t* tests and multiple regression) were performed by SPSS. Linear regression was used to determine the direct relationship between variables of interest. Stepwise forward selection and backward elimination were performed to determine a parsimonious model that incorporates only those predictor variables that are independently significant at the 0.05 level (8).

RESULTS

Per capita daily energy intake averaged 3064 cal/day, with 9.5% energy from protein, 59.3% from carbohydrate, and 31.2% from fat. Sugars, accounted for primarily by sucrose (196 g · person^{-1} · day^{-1}), contributed 37.1% of total energy. Fruit and vegetable intake was low (115 g wet wt · person^{-1} · day^{-1}), as was the intake of dietary fiber (<13 g · person^{-1} · day^{-1}). Fat (106 g · person^{-1} · day^{-1}) was derived primarily from meats and snack foods.

Anthropometric data for the population as a function of age are shown in Table 1. Although body mass index (BMI) increased with age in both men and women, the population was not obese by White standards. More than half of those <35 yr of age (59% of men, 58% of women) had BMI <20 kg/m^2. BMI, however, varied greatly, ranging from 14 to 34 kg/m^2 in the population. The waist-hip ratio rose with age ($P < 0.01$). Both men and women had a central or android pattern of fat distribution.

The frequency of diabetes and IGT is shown in Table 2. Fourteen (11.5%) had diabetes, and 9 (7.4%) had IGT. Diabetes and IGT were both more common in those aged ≥35 yr ($P < 0.001$).

Plasma glucose and insulin concentrations before and 2 h after 75 g oral glucose are reported in Table 3. Mean fasting glucose levels increased with age in men ($P < 0.02$) but not women. Mean 2-h glucose levels increased with age in both men and women, with the effect being more pronounced in the men. There were

TABLE 1
Age and anthropometric data of population

	Men		Women	
	15–34 yr	>35 yr	15–34 yr	>35 yr
n	33	17	37	35
Age (yr)	24.0 ± 0.9	47.1 ± 2.7	24.8 ± 0.7	46.0 ± 1.7
Height (m)	1.74 ± 0.01	1.77 ± 0.01	1.64 ± 0.01	1.65 ± 0.01
Weight (kg)	61.9 ± 2.0	73.6 ± 4.0	54.9 ± 2.2	61.5 ± 2.5
Body mass index (kg/m^2)	20.4 ± 0.6	23.5 ± 1.2	20.5 ± 0.9	22.7 ± 0.9
Waist-hip ratio	0.895 ± 0.011	0.951 ± 0.015	0.857 ± 0.010	0.915 ± 0.017

Values are means ± SE.

no significant differences in fasting insulin levels between men and women and no increase with age. However, 2-h insulin levels were higher in the men >35 yr of age than in those <35 yr of age, but once adjustment was made for BMI, these differences disappeared. Although there was a similar trend for 2-h insulin levels in women to increase with age, it was not significant and also disappeared completely once adjustment was made for BMI.

The interrelationships between age and BMI and the major parameters of carbohydrate and lipid metabolism measured in this study (fasting glucose, insulin, triglyceride, cholesterol and HDL-chol concentrations, 2-h glucose, and 2-h insulin) were examined in men and women with multiple regression to determine which parameters were related to each other independently when the influence of the other parameters was taken into account (Table 4). Diabetic subjects were excluded from analysis. The 2-h glucose and insulin values were not modeled as predictors of the fasting parameters.

The 2-h glucose was determined primarily by 2-h insulin and fasting glucose levels (Table 4). Similarly, 2-h insulin was determined by 2-h glucose and fasting insulin levels. In turn, fasting glucose was determined by the fasting insulin with additional determinants (excluding 2-h glucose), i.e., age, sex (higher in men), and triglycerides (inversely related). Fasting insulin levels were determined by fasting glucose, BMI, and triglycerides and inversely related to cholesterol.

TABLE 2
Frequency of diabetes and impaired glucose tolerance

		Normal glucose tolerance		Impaired glucose tolerance		Diabetes	
Age (yr)	*n*	*n*	%	*n*	%	*n*	%
Men							
<35	33	30	90.9	1	3.0	2	6.1
>35	17	9	53.0	4	23.5	4	23.5
Women							
<35	37	35	94.6	2	5.4	0	
>35	35	25	71.4	2	5.7	8	22.9
Total							
<35	70	65	92.9	3	4.3	2	2.8
>35	52	34	65.4	6	11.5	12	23.1
All ages	122	99	81.1	9	7.4	14	11.5

Fasting plasma lipid levels are shown in Table 5. Cholesterol levels were an average of 0.55 mM higher in men than women and increased with age ($P < 0.001$). HDL-chol levels were similar in men and women and did not change significantly with age. Triglyceride levels were an average of 1.05 mM higher in men than women ($P < 0.001$) and increased with age in women but not men. Lipid abnormalities were much more frequent among men than women: the frequency of hypercholesterolemia (>5.5 mM) was 24 vs. 3% in men and women <35 yr of age and 47 vs. 23% in men and women >35 yr of age. Similarly, hypertriglyceridemia (>2.0 mM) occurred much less frequently among women: 6 vs. 36% in women and men <35 yr of age and 43 vs. 71% in women and men >35 yr of age.

One of the most striking characteristics of this population was the wide range of 2-h insulin levels and the high frequency of hyperinsulinemia. In Table 6, the major anthropometric and metabolic characteristics of the population are presented as a function of 2-h insulin levels. The mean 2-h insulin concentration of the upper tertile of the population was more than seven times higher than that of the lower tertile. Differences in fasting insulin levels between the two groups were much less pronounced. Although those in the upper tertile had a higher mean BMI, the range of BMIs in both tertiles was almost identical (16–30 kg/m^2 in lower tertile and 15–31 kg/m^2 in upper tertile). The tertiles were similar in age range, and there were no differences in waist-hip ratios. There were, however, some important differences between the groups: impairment of glucose tolerance was much more common in the upper tertile, and triglyceride levels were higher and HDL-chol levels lower in the upper tertile.

DISCUSSION

The most striking result of this study was the high prevalence of diabetes in this population, despite their relative leanness. Diabetes prevalence increased sharply with age, being almost eight times more common in those >35 yr of age than in those

TABLE 3
Glucose and insulin concentrations before (fasting) and 2 h after 75 g oral glucose

	Men		Women	
	15–34 yr	>35 yr	15–34 yr	>35 yr
n	31	13	37	27
Glucose (mM)				
Fasting	4.5 ± 0.1	4.9 ± 0.2	4.5 ± 0.1	4.5 ± 0.1
Two hour	4.9 ± 0.2	6.5 ± 0.6	5.1 ± 0.3	5.5 ± 0.2
Insulin (mU/L)				
Fasting	15 ± 0.2	19 ± 0.3	15 ± 0.2	15 ± 0.2
Two hour	50 ± 0.7	101 ± 34	60 ± 0.7	71 ± 10

Values are means ± SE, excluding diabetic subjects.

<35 yr of age. IGT was also age related, but the differential was much less pronounced. Comparisons with other Aboriginal communities in northern Australia are interesting. The population in this study had more than twice as much diabetes as a population of coastal Aborigines who were similarly lean (5) and significantly more diabetes than even desert Aborigines who were not as lean (8). Thus, although both fasting and 2-h glucose levels were correlated with BMI in a univariate analysis in this study, other factors must be examined in an attempt to explain the high diabetes prevalence rates.

Aborigines have been shown to have a linear body build (i.e., with shorter trunks and longer limbs than almost every other ethnic group; 14). Body composition studies have shown that, for a given BMI, Aboriginal women have more body fat than White women and that the fat is centrally distributed (15,16). These data imply that, not only are BMI standards derived from one population not necessarily appropriate for another, but also that distribution of body fat may differ greatly between populations. Both men and women in the population studied here had a central distribution of body fat, and the waist-hip ratio increased with increasing BMI. There was little difference in body fat distribution between men and women, in contrast to the usual pattern in Europeans (12). This central distribution of fat has been shown in Europeans to be associated with increased risk of non-insulin-dependent diabetes mellitus, hypertriglyceridemia, and hyperinsulinemia, all of which were highly prevalent in this Aboriginal population. Thus, although obesity as such was rare, the centrally located fat deposits observed in this population could be interpreted as indicating chronic disease risk. However, this is unlikely to be a sufficient explanation for the findings itself, because a high waist-hip ratio was much more common than hyperinsulinemia, hyperlipidemia, or abnormal glucose tolerance and was not a predictor of chronic disease risk factors in the multivariate analysis.

One of the most striking characteristics of the population was the wide range of 2-h insulin levels. The highest 2-h insulin levels were associated with IGT and

TABLE 4
Interrelationships between age, body mass index (BMI), and major parameters of carbohydrate and lipid metabolism

Dependent variable	Predictor variable	B ± SE*	*P*
Fasting glucose (mM)	Fasting insulin	0.024 ± 0.005	0.0001
	Age	0.009 ± 0.004	0.02
	Triglycerides	−0.093 ± 0.039	0.02
	Sex†	−0.251 ± 0.101	0.02
Two-hour glucose (mM)	Two-hour insulin	0.010 ± 0.002	0.0001
	Fasting glucose	0.604 ± 0.228	0.01
Fasting insulin (mU/L)	BMI	0.686 ± 0.172	0.0001
	Triglycerides	2.601 ± 0.419	0.0001
	Fasting glucose	4.612 ± 1.532	0.003
	Cholesterol	−1.850 ± 0.667	0.007
Two-hour insulin (mU/L)	Two-hour glucose	22.862 ± 4.316	0.0001
	Fasting insulin	2.221 ± 0.568	0.0002
Cholesterol	Age	0.027 ± 0.007	0.0004
	High-density lipoprotein cholesterol	1.205 ± 0.284	0.0001
	Triglycerides	0.163 ± 0.060	0.008
	Sex†	−0.551 ± 0.212	0.01
Triglycerides (mM)	Fasting insulin	0.086 ± 0.017	0.0001
	Cholesterol	0.428 ± 0.137	0.0007
	Fasting glucose	−0.894 ± 0.295	0.02
	High-density lipoprotein cholesterol	−1.295 ± 0.458	0.006
	Sex†	−1.048 ± 0.301	0.0008
High-density lipoprotein cholesterol (mM)	Cholesterol	0.128 ± 0.027	0.0001
	BMI	−0.025 ± 0.0007	0.004
	Triglycerides	−0.070 ± 0.017	0.0001

*B ± SE is estimated regression coefficient and its standard error.
†Negative value for sex indicates that parameter was higher in men than in women.

TABLE 5
Cholesterol, triglyceride, and high-density lipoprotein cholesterol concentrations in fasting plasma

	Men		Women	
	15–34 yr	>35 yr	15–34 yr	>35 yr
n	31	13	37	27
Cholesterol (mM)	4.73 ± 0.21	5.38 ± 0.27	4.05 ± 0.14	4.41 ± 0.21
Triglycerides (mM)	2.56 ± 0.50	2.67 ± 0.48	1.25 ± 0.08	1.62 ± 0.13
High-density lipoprotein cholesterol (mM)	1.38 ± 0.08	1.15 ± 0.08	1.24 ± 0.05	1.23 ± 0.07

Values are means ± SE, excluding diabetic subjects.

hypertriglyceridemia. Hyperinsulinemia was much more pronounced 2 h after 75 g oral glucose than in the fasting state. Nevertheless, fasting and 2-h insulin levels were strongly correlated. The association of fasting glucose and insulin and 2-h glucose and insulin values are both consistent with underlying insulin resistance. It was particularly interesting that neither excess body fat (BMI) nor its location (waist-hip ratio) was associated with insulin resistance. In terms of their 2-h insulin response, the lower tertile of the population had the same range of BMI and waist-hip ratio values as the upper tertile. There were obese individuals with a central location of body fat who had excellent glucose tolerance and insulin sensitivity (as assessed by a low insulin response to oral glucose), and there were lean individuals who had glucose intolerance and pronounced hyperinsulinemia (interpreted as indicating insulin resistance).

What is the source of this insulin resistance? Is it primarily hepatic or peripheral? Is it a cause of or a response to hyperinsulinemia?

Hyperinsulinemia is not in itself proof of increased insulin secretion. Without concurrent measurements of C-peptide concentrations we are unable to differentiate unequivocally between increased insulin secretion or reduced hepatic extraction of insulin as the source of the elevated insulin concentrations observed. Obesity, hyperinsulinemia, and IGT have all been shown to be associated with reduced hepatic extraction of insulin (17–19). However, what is unequivocal is that the hyperinsulinemia 2 h after oral glucose, whatever its cause, was ineffective in normalizing plasma glucose levels. Hyperinsulinemia and resistance to the glucose-lowering effects of insulin have been described previously for Aborigines (5,8,20) and other populations exhibiting high rates of diabetes when subjected to rapid life-style change, e.g., Pima Indians (21), Nauruans (22), other Pacific islanders (23), and Mexican Americans (24).

Lipid levels had a pattern similar to that observed in other Aboriginal populations (5,8), the major feature of which was high triglyceride levels consistent with insulin resistance. Both triglyceride and cholesterol concentrations were higher in men than women. Dietary and life-style practices may have contributed to the large sex difference in triglyceride levels. Excessive alcohol consumption was much more frequent in men than women, and the women were generally more physically active than the men; the relatively high HDL-chol levels in the younger men in particular are consistent with high alcohol intake (25). The higher cholesterol levels in men may have been related to their high consumption of fatty cuts of meat (mostly beef).

In conclusion, this population had an unexpectedly high prevalence of diabetes and IGT in view of their relative leanness. Markedly elevated insulin levels 2 h after oral glucose appeared to be the best indicator of abnormal carbohydrate metabolism and may be used as an independent predictor of diabetes risk within the population. Consistent with data from other Australian

TABLE 6
Anthropometric and metabolic characteristics of subjects as function of their 2-h insulin levels

	Lower tertile	Upper tertile	*P*
n	36	35	
Two-hour insulin (mU/L)	19 ± 0.1	144 ± 13	0.0001
Fasting insulin (mU/L)	13 ± 0.1	20 ± 0.2	0.002
Age (yr)	32.9 ± 2.3	34.5 ± 2.2	NS
Body mass index (kg/m^2)	20.5 ± 0.6	22.1 ± 0.8	NS
Waist-hip ratio	0.894 ± 0.013	0.899 ± 0.015	NS
Fasting glucose (mM)	4.6 ± 0.2	5.0 ± 0.2	NS
Two-hour glucose (mM)	4.7 ± 0.3	7.0 ± 0.5	0.001
Cholesterol (mM)	4.44 ± 0.20	4.76 ± 0.21	NS
Triglycerides (mM)	1.62 ± 0.19	2.03 ± 0.24	0.001
High-density lipoprotein cholesterol (mM)	1.47 ± 0.07	1.19 ± 0.08	0.0001

Values are means ± SE, excluding diabetic subjects.

Aboriginal groups (2–8), the usual sex difference in diabetes prevalence was not evident in this population; e.g., women were at a similar risk to men. However, women had much healthier lipid profiles than men. The results of this study provide a strong rationale for the development of intervention strategies aimed at minimization of insulin resistance through life-style modification. There is no doubt that, when diet and life-style are modified, as in temporary reversion to traditional hunter-gatherer life-style, the abnormalities of carbohydrate and lipid metabolism associated with diabetes and IGT in Aborigines are greatly ameliorated (20, 26,27). However, for intervention strategies to be successful over the long term, it is essential that they be developed by the Aboriginal communities themselves to ensure that they are culturally appropriate and acceptable and therefore able to be "owned" by the community rather than imposed in a well-meaning fashion from the outside.

ACKNOWLEDGMENTS

This work was supported by a grant from the National Health and Medical Research Council of Australia to K.O'D.

We are grateful to the people of Nauiyu Nanambiyu, without whose help and cooperation this work could not have been conducted.

REFERENCES

1. 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* 143:436–40, 1985
2. Wise PH, Edwards FM, Thomas DW, Elliott RB, Hatcher L, Craig R: Diabetes and associated variable in the South Australian Aboriginal. *Aust NZ J Med* 6:191–96, 1976
3. Bastian P: Coronary heart disease in tribal Aborigines: the West Kimberley survey. *Aust NZ J Med* 9:284–92, 1979
4. Duffy P, Morris H, Neilsen G: Diabetes mellitus in the Torres Strait region. *Med J Aust* 1 (Suppl. 2):S8–11, 1981
5. O'Dea K, Spargo RM, Nestel PJ: Impact of westernisation on carbohydrate and lipid metabolism of Australian Aborigines. *Diabetologia* 22:148–53, 1982
6. Cameron WI, Moffitt PS, Williams DDR: Diabetes mellitus in the Australian Aborigines of Bourke, New South Wales. *Diet Res Clin Pract* 2:307–14, 1986
7. Williams DDR, Moffitt PS, Fisher JS, Bashir HV: Diabetes and glucose tolerance in New South Wales coastal Aborigines: possible effects of non-Aboriginal genetic admixture. *Diabetologia* 30:72–77, 1987
8. O'Dea K, Traianedes K, Hopper JL, Larkins RG: Impaired glucose tolerance, hyperinsulinemia, and hypertriglyceridemia in Australian Aborigines from the desert. *Diabetes Care* 11:23–29, 1988
9. O'Dea K, White NG, Sinclair AJ: An investigation of nutrition-related risk factors in an isolated Aboriginal community in northern Australia: advantages of a traditionally-orientated lifestyle. *Med J Aust* 148:177–80, 1988
10. Paul AA, Southgate DAT: *McCance and Widdowson's The Composition of Foods*. 4th ed. London, Her Majesty's Stationery Office, 1978 (Med. Res. Council Rep. Ser. 293)
11. Cashel K, English R, Lewis J: *Composition of Foods Australia*. Canberra, Australia, Aust. Govt. Printing Office, 1989
12. Krotkiewski M, Bjorntorp P, Sjostrom L, Smith U: Impact of obesity in metabolism in men and women: importance of regional adipose tissue distribution. *J Clin Invest* 72:1150–62, 1983
13. World Health Organization: *WHO Expert Committee on Diabetes Mellitus: Second Report*. Geneva, World Health Org., 1980 (Tech. Rep. Ser., no. 646)
14. Abbie AA: Physical characteristics. In *Aboriginal Man in South and Central Australia*. Adelaide, Australia, Aust. Govt. Printing Office, 1966, p. 9–45
15. Rutishauser IHE, McKay H: Anthropometric status and body composition in Aboriginal women of the Kimberley region. *Med J Aust* 144 (Suppl.):S8–10, 1986
16. O'Dea K: Body fat distribution and health outcome in Australian Aborigines. *Proc Nutr Soc Aust* 12:56–65, 1987
17. Meistas MT, Margolis S, Kowarski AA: Hyperinsulinemia of obesity is due to decreased clearance of insulin. *Am J Physiol* 245:E155–59, 1983
18. Bonara E, Zavaroni I, Coscelli C, Butturini U: Decreased hepatic insulin extraction in subjects with mild glucose intolerance. *Metabolism* 32:438–46, 1983
19. Polansky KS, Rubenstein AH: C-peptide as a measure of the secretion and hepatic extraction of insulin. *Diabetes* 33:486–94, 1984
20. O'Dea K, Spargo RM, Akerman K: The effect of transition from traditional to urban life-style on the insulin secretory response in Australian Aborigines. *Diabetes Care* 3:31–37, 1980
21. Aronoff SL, Bennett PH, Gorden P, Rushforth N, Miller M: Unexplained hyperinsulinemia in normal and prediabetic Pima Indians compared with normal Caucasians: an example of racial differences in insulin secretion. *Diabetes* 26:827–40, 1977
22. Balkau B, King H, Zimmet P, Raper LR: Factors associated with the development of diabetes in the Micronesian population of Nauru. *Am J Epidemiol* 122:594–605, 1985
23. Zimmet P, Whitehouse S, Kiss J: Ethnic variability in the plasma insulin response to oral glucose in Polynesian and Micronesian subjects. *Diabetes* 28:624–28, 1979
24. Haffner SM, Stern MP, Hayuda HP, Pugh JA, Patterson JK: Hyperinsulinemia in a population at high risk for non-insulin dependent diabetes mellitus. *N Engl J Med* 315: 220–24, 1986
25. Taskinen M-R, Välimäki M, Nikkilä EA, Kuusi T, Ehnholm C, Ylikahri R: High density lipoprotein subfractions in alcoholic men before and after alcohol withdrawal. *Metabolism* 31:1168–74, 1982
26. O'Dea K: Marked improvement in carbohydrate and lipid metabolism in diabetic Australian Aborigines after temporary reversion to traditional lifestyle. *Diabetes* 33:596–603, 1984
27. O'Dea K: The hunter-gatherer lifestyle of Australian Aborigines: implications for health. In *Current Problems in Nutrition, Pharmacology and Toxicology*. McLean AJ, Wahlqvist ML, Eds. London, Libbey, 1988, p. 26–36