THE RELATIONSHIP BETWEEN URBANISATION AND DIABETES IN AUSTRALIAN ABORIGINES

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Summary

In common with certain other populations, Australian Aborigines are particularly susceptible to Type 2 diabetes when they make a rapid transition from traditional to urban lifestyle. In an attempt to understand the relative contributions of genetic and lifestyle influences, we have conducted a series of metabolic studies on Aborigines from the Kimberley region of Western Australia with particular reference to degree of urbanisation and the effects of temporary reversions to traditional lifestyle.

The results of these studies suggest that certain metabolic characteristics are strongly influenced by lifestyle (fasting plasma glucose, and plasma total, LDL and HDL cholesterol concentrations), while others appear to have a strong genetic component (impairment of glucose tolerance, insulin resistance and elevations in VLDL lipids). These latter metabolic characteristics may favour efficient fat deposition through enhanced hepatic lipogenesis and, although they could have been considered a survival advantage under conditions of traditional lifestyle (the 'thrifty gene'), they would promote obesity in the urban environment with its altered diet and level of physical activity. The increased insulin resistance of obesity may be a critical factor in precipitating diabetes in these people when they urbanise. Indeed, diabetes was uncommon in a small, rural community, from the same geographical area, in which obesity was not widespread.

The impairment of glucose tolerance and insulin resistance were both ameliorated in urban Aborigines by temporary reversion to traditional lifestyle, raising the possibility that such a procedure may have both therapeutic and preventive potential as a new approach to the increasing problem of diabetes in Aboriginal communities.

1. INTRODUCTION

Particularly high prevalence rates for Type 2 (non-insulin-dependent, or maturity-onset) diabetes have been observed in some populations which have undergone rapid transitions from traditional to urban lifestyles (e.g. Pima Indians (Bennett et al. 1971), Nauruans (Zimmet et al. 1977), Australian Aborigines (Wise et al. 1976; Bastian 1979)). The high prevalence rates have been preceded by widespread and often severe obesity in the communities concerned. Thus, although such people appear to be highly susceptible to diabetes, they do not develop the disease without the presence of certain lifestyle-associated trigger factors related to diet and level of physical activity. Although the precise nature of the genetic susceptibility to Type 2 diabetes is yet to be defined, it has been suggested that the 'initial lesion' is related to insulin excess (insulin resistance?) rather than insulin deficiency (Jackson et al. 1972; Reaven et al. 1976). A high insulin response may have conferred survival advantage to people in a hunter-gatherer environment with its feast-and-famine pattern of food intake by facilitating

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efficient fat deposition in times of food abundance - the 'thrifty gene' (Neel 1963).

A recent survey in the West Kimberleys in Western Australia revealed that 17% of Aborigines had diabetes (Bastian 1979). This compares with 2-3% for Australians of European descent (Welborn et al. 1968). The study of such high-risk communities can provide insight into both the metabolic characteristics of susceptible people and the environmental trigger factors operating to precipitate Type 2 diabetes in these people.

Over the past 5 years we have conducted a series of metabolic studies on Aborigines from the West Kimberleys with particular reference to the degree of urbanisation and the effects of temporary reversions to traditional lifestyle. The area in which these studies were carried out is shown in Figure 1.

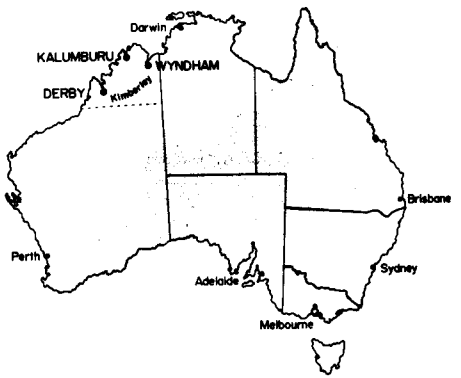

Fig. 1. Map of Australia showing the location of Kalumburu and Derby in the Kimberley region of Western Australia.

II. EXPERIMENTAL AND RESULTS

(a) Effect of temporary reversion to tradutional lifestyle

There are many groups of Aborigines in outback regions of Australia who, despite living in an urban setting for most of the time, retain the knowledge and ability to survive in their traditional environment as hunter-gatherers. This has provided us with what appears to be a unique opportunity to compare urban and traditional metabolic responses in a population that responds to urbanisation by developing high prevalence of obesity and Type 2 diabetes.

'Urban' refers to the environment of the majority of Aborigines in this region who live near towns: high unemployment, physically inactive, high alcohol consumption and a diet rich in refined carbohydrate and fat.

'Traditional' is used in the sense that the only food eaten was that hunted and gathered by the Aborigines themselves in an isolated location at least 100 km from the nearest settlement. Under these conditions the Aborigines were physically active and had no access to alcohol or refined food. This diet was high in protein and low to moderate in carbohydrate and fat.

In the first of these studies (O'Dea at al. 1980), 13 full-blood Aborigines from the Mowanjum Community spent 3 months living as nomadic hunter-gatherers. The major sources of food were kangaroo, birds, reptiles and fish, supplemented with vegetable foods such as yams, palm hearts, wild fruits and honey. At the conclusion of this 3-month 'traditional' period, and again 3 months after they had returned to their urban environment, the Aborigines were given a carbohydrate-loading test after a 12-h overnight fast.

Similar tests were conducted on a group of Caucasians of comparable age and weight.

The major findings, the details of which have been published elsewhere (O'Dea et al. 1980), were as follows.

(1) The insulin responses of the Aborigines in the urban environment were twice as high as in the Caucasians: AUC 289 ± 35 vs 153 ± 22 μU/mL h respectively (Figure 2).

(2) The insulin response was reduced in the traditional environment (203 ± 21 μU/mL h) but was still 50% higher than in Caucasians (Figure 2).

(3) In the traditional environment the Aborigines showed a small improvement in glucose tolerance, no change in fasting plasma cholesterol concentrations (4.6 mM traditional; 4.6 mM urban), a significant reduction in fasting plasma triglyceride concentration (1.13 mM traditional; 1.92 mM urban) and a reduction in body weight.

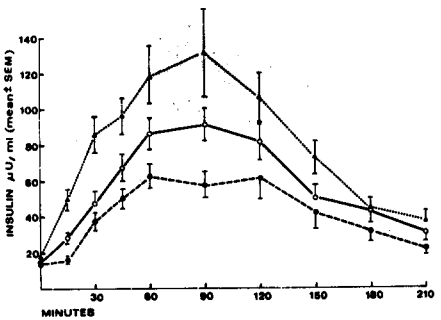

Fig. 2. Changes in plasma insulin concentrations, following oral ingestion of 70 g starch, in 13 Aborigines in the urban (.....) or traditional (————) environments and in 16 Caucasians (------). (O'Dea, K., Spargo, R.M. and Akerman, K. (1980). Diabetes Care 3: 31.)

The second study in this series (O'Dea and Spargo 1982) was an examination of short-term adaptation to the low-carbohydrate, high-protein traditional diet. The glucose and insulin responses to 75 g glucose were measured before and after 2 weeks on a diet derived almost exclusively from seafood in 12 young Aborigines. There was a small but significant improvement in glucose tolerance ($P < 0.05$) (Figure 3) which was accompanied by a similar small, significant reduction in the insulin response ($P < 0.05$) (Figure 4). Together these findings suggest an improvement in glucose utilisation and peripheral insulin sensitivity. There was no change in the fasting plasma cholesterol concentration (4.26 ± 0.57 mM before; 3.93 ± 0.31 mM after), while fasting triglyceride concentrations fell from 1.33 ± 0.23 mM before to 0.61 ± 0.08 mM after ($P < 0.001$).

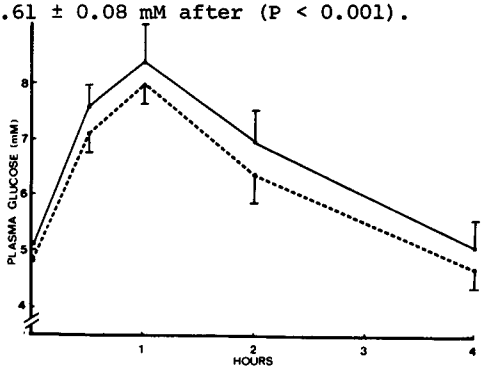

Fig. 3. Changes in plasma glucose concentration following 75 g oral glucose in 12 Aborigines before (————) and after (------) 2 weeks on a low-carbohydrate high-protein diet. (O'Dea, K. and Spargo, R.M. (1983). Diabetologia (in press).)

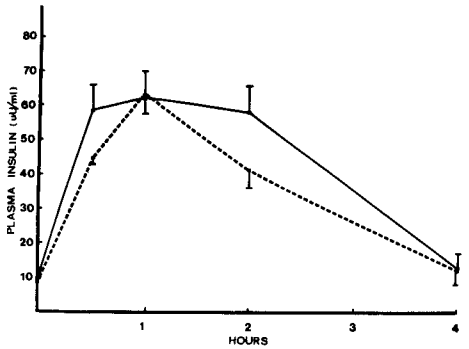

Fig. 4. Changes in plasma insulin concentration following 75 g oral glucose in 12 Aborigines before (———) and after (------) 2 weeks on a low-carbohydrate, high-protein diet. (O'Dea, K. and Spargo, R.M. (1983). Diabetologia (in press).)

(b) Influence of degree of urbanisation

Kalumburu Mission is situated on the northern coast of Western Australia (Figure 1), 750 km north-east of Derby, and is accessible by road for only 6-8 months of the year. The Aboriginal community there has had less exposure to many aspects of urban life than other Aboriginal groups in the Kimberleys. The people lead a rural existence growing their own fruit and vegetables and breeding cattle, pigs and poultry. Beef and vegetable stew and rice were available twice daily. Soft drinks, sugar and other processed foods could be purchased at a store, but alcoholic drinks were excluded from the Mission at the time of this study and during the years preceding it.

We conducted a survey in this small population (O'Dea et al. 1982) and found that the frequency of diabetes and impaired glucose tolerance (National Diabetes Data Group 1979) was 4.5% and 11.9% respectively. No one under 54 had diabetes. Proportionately more men than women were glucose intolerant despite having somewhat lower body-mass indices (BMI men 22.2 ± 0.6 kg/m^2, women 23.8 ± 0.8 kg/m^2).

We also conducted more detailed metabolic studies on lean, young Aboriginal men from this rural community and made comparisons with urban Aborigines from the same geographical region (Mowanjum, Derby) as well as with Caucasians (O'Dea et al. 1982). The three groups were closely matched for age and body-mass index. The main findings were as follows.

(1) Plasma total, HDL and LDL cholesterol concentrations were significantly lower in the rural Aboriginal men than in either the urban Aborigines or the Caucasians. However, both Aboriginal groups had significantly higher VLDL lipid concentrations than the Caucasians.

Plasma lipoprotein lipid concentrations in Aboriginal and Caucasian men < 35 years (mmol/L) (Mean ± SEM)

	Caucasians	Aborigines	
		Urban	Rural
Number of Subjects	17	19	15
Total			
Cholesterol	4.89±0.29	5.00±0.09	3.89±0.27
Triglyceride	1.20±0.19	1.86±0.29	1.73±0.38
HDL			
Cholesterol	1.14±0.12	1.17±0.09	0.85±0.04
Triglyceride	0.60±0.11	0.42±0.06	0.56±0.10
VLDL			
Cholesterol	0.19±0.05	0.49±0.10	0.57±0.17
Triglyceride	0.10±0.04	0.50±0.13	0.46±0.22
LDL			
Cholesterol	3.61±0.36	3.34±0.23	2.46±0.16
Triglyceride	0.45±0.09	0.95±0.12	0.70±0.08

(2) The fasting plasma glucose concentrations in the rural Aboriginal men (4.1 mM) were significantly lower than in either the urban Aborigines (5.1 mM) or the Caucasians (4.7 mM).
(3) Both Aboriginal groups had mild impairment of glucose tolerance as evidenced by elevated plasma glucose level 2 h after a 75 g glucose load (Figure 5).
(4) The insulin responses to 75 g oral glucose in both Aboriginal groups were 50% higher than in Caucasians, although fasting levels were similar in all groups (Figure 6).

These latter results suggest that elevations in fasting glucose, total cholesterol, HDL and LDL cholesterol concentrations are lifestyle-associated, but that mild impairment of glucose tolerance, high insulin response and elevated VLDL lipid levels in the Aborigines may be inherited metabolic characteristics.

III. DISCUSSION

In the studies briefly described above we have attempted to distinguish between environmental and genetic influences upon glucose tolerance and insulin response in a population which has been shown to be highly susceptible to Type 2 diabetes upon changing from a traditional to an urban way of life.

By measuring glucose tolerance and insulin response to glucose in lean young Aboriginal men from rural and urban lifestyles and comparing the results with those from closely-matched urban Caucasians, we have been able to speculate on the relative contributions of genes and environment. The urban Aborigines and Caucasians showed certain similarities in that they both had higher fasting plasma concentrations of glucose, and of HDL and LDL cholesterol, than the rural Aborigines, which suggested that these parameters were strongly influenced by lifestyle. On the other hand, the urban and rural Aboriginal groups were very similar in their mild impairment of glucose tolerance, elevated insulin response and increased VLDL lipid levels, suggesting that these parameters had a strong genetic component.

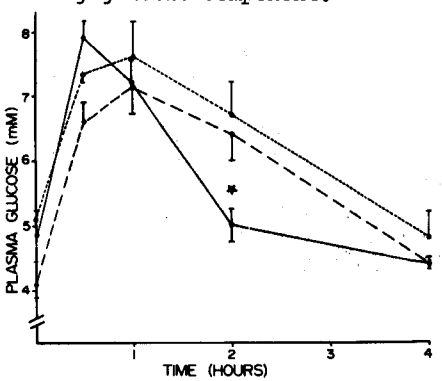

Fig. 5. Changes in plasma glocose concentrations, following 75 g oral glucose, in 15 rural Aborigines ( ), 19 urban Aborigines ( ) and 17 Caucasians (———). O'Dea, K., Spargo, R.M. and Nestel, P.J. (1982). Diabetologia 22 : 148.)

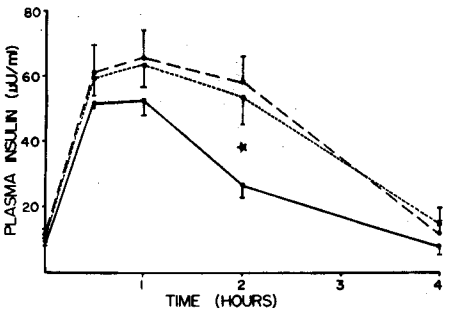

Fig. 6. Changes in plasma insulin concentrations, following 75 g oral glucose, in 15 rural Aborigines ( ), 19 urban Aborigines ( ), and 17 Caucasians ( ). O'Dea, K., Spargo, R.M. and Nestel, P.J. (1982). Diabetologia 22 : 148.)

Unlike results on glucose tolerance and insulin response from other populations at risk for diabetes, such as the Pima Indians (Aronoff et al. 1977), our own data were not complicated by the presence of obesity in the subjects under investigation. Thus, the insulin resistance, or impaired glucose utilisation, displayed by the lean young Aborigines in this study may be a critical factor in their eventual susceptibility to diabetes when older and obese. Reaven et al. (1976) have suggested that the primary defect in Type 2 diabetes is insensitivity of peripheral tissues to the action of insulin, which is responsible for the initial impairment of glucose tolerance. This is followed by a progressive deterioration in the β-cell's capacity to respond to hyperglycemia as a stimulus for insulin secretion. As a result of insulin resistance in peripheral tissues such as muscle and adipose tissue, a greater proportion of a glucose load would be taken up by the liver. In the presence of the elevated insulin concentrations hepatic lipogenesis would be stimulated. The hyperinsulinaemia would also stimulate adipose tissue lipoprotein lipase activity and thereby provide a means by which newly synthesised VLDL triglyceride could be efficiently transferred from liver to adipose tissue. Thus, peripheral insulin resistance may be a means by which excess energy could be directed, via hepatic lipogenesis, predominantly to adipose tissue lipid rather than muscle glycogen. The increased levels of VLDL triglyceride in both Aboriginal groups studied are consistent with such a scheme (O'Dea et al. 1982). It may also explain the susceptibility to obesity which, in turn, worsens the insulin resistance. In this way it may be possible to link efficient fat deposition (the 'thrifty gene') with susceptibility to Type 2 diabetes; i.e. insulin resistance may be a necessary precondition for efficient fat deposition as well as predisposing towards Type 2 diabetes. In all populations which are predisposed to Type 2 diabetes when they westernise (Pima Indians, Polynesians, Aborigines, Micronesians, Melanesians) the increase in diabetes prevalence is preceded by widespread and often extreme obesity.

If the primary defect in Type 2 diabetes is peripheral insulin resistance, then factors which act to precipitate the disease in susceptible people should also increase insulin resistance. The most general precipitating factor in all high-risk populations so far studied is a rapid change from traditional to urban lifestyle. In both our studies on the metabolic effects of temporary reversion to traditional lifestyle (3-month, 2-week) there was an associated reduction in peripheral insulin resistance. Several factors probably contributed to this improvement. Weight loss and increased physical activity have been shown to improve insulin sensitivity (Pfeiffer 1974); Björntorp et al. 1970) while the high consumptions of fat and sugar in the urban diet impair it (Himsworth 1935; Beck-Nielsen et al. 1978).

These data have potential therapeutic implications. By understanding why Aborigines are susceptible to diabetes and which aspects of modern western lifestyle act to trigger the disease, we may be able to suggest new approaches to what has become a serious health problem. Reverting to traditional lifestyle periodically may be an effective and acceptable means of treating/preventing diabetes in Aborigines in remote areas of Australia.

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REFERENCES

ARONOFF, S.L., BENNETT, P.H., GORDEN, P., RUSHFORTH, N. and MILLER, M. (1977). Diabetes 26: 827.

BASTIAN, P. (1979). Aust. N.Z. Jl Med. 9: 284.

BECK-NIELSEN, H., PEDERSEN, O. and SCHWARTZ-SORENSEN, N. (1978). Diabetologia 15: 289.

BENNETT, P.H., BURCH, T.A. and MILLER, M. 1971). Lancet ii: 125.

BJÖRNTORP, P., de JOUNGE, K., SJÖSTRÖM, L. and SULLIVAN, L. (1970). Metabolism 19: 631.

HIMSWORTH, H.P. (1935). Clin. Sci. 2: 67.

JACKSON, W.P.U., Van MIEGHAM, W. and KELLER, P. (1972). Lancet i: 1040.

NATIONAL DIABETES DATA GROUP (1979). Diabetes 28: 1039.

NEEL, J.V. (1963). Am. J. hum. Genet. 14: 352.

O'DEA, K. and SPARGO, R.M. (1983). Diabetologia (in press)

O'DEA, K., SPARGO, R.M. and AKERMAN, K. (1980). Diabetes Care 3: 31.

O'DEA, K., SPARGO, R.M. and NESTEL, P.J. (1982). Diabetologia 22: 148.

PFEIFFER, E.F. (1974). Horm. Metab. Res. Suppl. 4: 143.

REAVEN, G.M., BERNSTEIN, R., DAVIS, B. and OLEFSKY, J.M. (1976). Am. J. Med. 60: 80.

WELBORN, T.A., CURNOW, D.H., WEARNE, J.T., CULLEN, K.J., McCALL, M.G. and STENHOUSE, N.S. (1968). Med. J. Aust. 2: 778.

WISE, P.H., EDWARDS, F.M., CRAIG, R.F., EVANS, B., MARCHLAND, J.B., SUTHERLAND, B. and THOMAS, D.W. (1976). Aust. N.Z. Jl Med. 6: 191.

ZIMMET, P., TAFT, P., GUINEA, A., GUTHRIE, W. and THOMA, K. (1977). Diabetologia 13: 111.