East Kimberley
Impact Assessment Project

THE HEALTH OF KIMBERLEY ABORIGINES:
A PERSONAL 15-YEAR PERSPECTIVE

by

Michael Gracey[1] MD, PhD, FRACP

and

Randolph M. Spargo[2] MB, BS, MPH

A Joint Project Of The:

Centre for Resource and Environmental Studies
Australian National University

Australian Institute of Aboriginal Studies

Anthropology Department
University of Western Australia

Academy of the Social Sciences in Australia

The aims of the project are as follows:

1. To compile a comprehensive profile of the contemporary social environment of the East Kimberley region utilising both existing information sources and limited fieldwork.

2. Develop and utilise appropriate methodological approaches to social impact assessment within a multi-disciplinary framework.

3. Assess the social impact of major public and private developments of the East Kimberley region's resources (physical, mineral and environmental) on resident Aboriginal communities. Attempt to identify problems/issues which, while possibly dormant at present, are likely to have implications that will affect communities at some stage in the future.

4. Establish a framework to allow the dissemination of research results to Aboriginal communities so as to enable them to develop their own strategies for dealing with social impact issues.

5. To identify in consultation with Governments and regional interests issues and problems which may be susceptible to further research.

Views expressed in the Project's publications are the views of the authors, and are not necessarily shared by the sponsoring organisations.

Address correspondence to:

The Executive Officer
East Kimberley Project
CRES, ANU
GPO Box 4
Canberra City, ACT 2601

THE HEALTH OF KIMBERLEY ABORIGINES:
A PERSONAL 15-YEAR PERSPECTIVE

by

Michael Gracey[1] MD, PhD, FRACP

and

Randolph M. Spargo[2] MB, BS, MPH

[1]   Princess Margaret Children's Medical Research Foundat on,
       Department of Child Health, University of Western Australia,   Perth.

[2]   Western Australian Department of Heal h, Derby, Western Aust tralia

EXPLANATORY NOTE

This paper is an attempt to bring data together from publicly available reports which have appeared in the past 15 years or so about the health of Aboriginal people in the Kimberley region of Western Australia. Inevitably, the content is subjective by virtue of focusing on subjects of particular interest to the authors. We have tried to overcome this bias by inviting colleagues who have worked in the Kimberley to provide data for inclusion and comment. They did so very generously but our coverage of their work and that of others may still be incomplete. This working document is presented to stimulate interest in the likely future trends of health for Aboriginal people in the Kimberley. These will need to be assessed in relation to the rapid physical and social changes which will occur as a result of increased mining, tourism and development in the region over the next 20 years.

SUMMARY

Up till about 20 years ago Aborigines in the remote Kimberley region of Western Australia experienced a pattern of health/illness which was dominated by physical isolation, nutritional deprivation and infectious diseases, particularly in infants and young children. Rapid sociological and political changes in Aboriginal society in general and in the far North-West of Australia in particular have brought in a new set of circumstances in which the so-called 'lifestyle' diseases now dominate. These include a very high incidence of diet-related obesity, hypertension, type-2 diabetes and coronary heart disease. Alcoholism and its related disorders, sudden and violent deaths - particularly in young men - and a very high rate of sexually transmitted diseases, are now major causes for disturbingly high levels of morbidity and mortality in Aborigines in the Kimberley region, particularly in young Aboriginal men.

TABLE OF CONTENTS

Introduction

Awareness of widespread and serious health problems in Aborigines began to emerge in Australia in the late 1960s. Malnutrition and highly prevalent infectious diseases in Aboriginal children became the initial focus for public and political attention. As a paediatrician with a special interest in gastrointestinal and nutritional disorders, one of us (M.G.) was first asked to assess these problems in the Kimberley in 1971 because of severe overcrowding in the children's ward of the Derby District Hospital, where newspapers had reported patients being nursed in cardboard beer cartons on the hospital verandah. At that time the other author (R.M.S.) had already spent some years as a Government Medical Officer in the region.

The Kimberley was much more remote then from the rest of Australia, in a communications sense, than it is today. Scheduled air services were far less frequent than they are today, there were no direct flights from Perth to Derby, the West Kimberley regional administrative centre, and booked seats were often cancelled at short notice because of emergency medical evacuations to Perth. Many of these patients were undernourished infants and young children with acute diarrhoea and dehydration. Their mortality rate was high. Telephone connections were often difficult and sometimes 'out' for hours or days because 'the lines are down'. There was no telex and no television.

Similarly, the medical infrastructure of the Kimberley was sparse and almost primitive by comparison with today. Government Medical Officers were few and scattered over vast distances, community and public health nurses had just begun to be appointed in the main towns and in the bigger remote communities and Aboriginal Health Workers had not yet been brought into service in their own communities.

The non-Aboriginal population of the far North-West of the nation was then much smaller than today. Tourism was restricted to the most adventurous, tourist facilities and accommodation were almost non-existent, and major mineral and oil discoveries were still to be made. Most of the roads, even between the six main Kimberley towns were unsurfaced and subject to intermittent interruption by floods which could isolate communities for many days or weeks. All these factors had a significant bearing on the health problems of the Kimberley at that time, and the subsequent physical and communications changes have helped alter the health patterns.

Gastroenteritis

Gastroenteritis was a major problem in Aboriginal infants and young children in the 1960s and 1970s (Jose and Welch, 1970; Walker and Harry, 1972); in the Kimberley, outbreaks of 'gastro' were common especially in 'the wet' and many severely dehydrated children were admitted to the main hospitals in Derby, Broome, Halls Creek and Wyndham. Knowledge about treatment of dehydration was very limited then, even in major centres, and the accepted method was intravenous therapy. This technique can be very difficult for personnel unfamiliar with the treatment of sick children and very sick patients were often evacuated urgently by air to the Princess Margaret Hospital in Perth, the only children's hospital in Western Australia. By the time patients were admitted to hospital in Perth, or

closer to home, their clinical condition was often complicated and their medical management was difficult; their outlook was correspondingly poor.

From mid-1970 to mid-1972 two hundred and fifty-one (251) Aboriginal infants and children with diarrhoea were admitted to the Princess Margaret Hospital from all parts of Western Australia, many from the Kimberley (Gracey, 1973). Almost two-thirds (65%) of these patients were under 12 months of age and more than one-quarter (27%) were between one and two years old. Their mortality rate was 5% which was much higher than experienced at that time by non-Aboriginal children with gastroenteritis.

Many patients were admitted to hospital on multiple occasions and their mean stay in hospital was 19 days. Forty percent of the patients were malnourished and more than one-third were anaemic. Nineteen patients had marasmus or marasmic kwashiorkor, severe forms of malnutrition. There was a remarkably high rate of isolation of intestinal pathogens in these patients (50%) including such organisms as enteropathogenic *E. coli*, *Salmonella*, *Shigella* and *Giardia lamblia*. Twenty-five percent of patients had temporary sugar intolerance, particularly to lactose in their feeds. Other complications, particularly infections such as bronchitis, pneumonia, otitis media, osteomyelitis and septicaemia, were prominent problems which made medical management difficult; multiple infections were common. The clinical features experienced by this group of poorly nourished Aborigines were characteristic of the pattern of gastrointestinal and other infections which occurs in undernourished children in developing countries.

Childhood deaths and malnutrition

Necropsy evidence from 13 Aboriginal children who died in Princess Margaret Hospital emphasized the importance of malnutrition in these patients (Gracey and Bower, 1973). Six patients had clinical evidence of malnutrition; diarrhoea was present in most patients and most had intestinal pathogens isolated; six patients had severe pneumonia; eight patients were anaemic. A remarkable finding was fatty infiltration of the liver which, in many children, was grossly enlarged, very pale and showed visible fat macroscopically when cut. This pathological change in the liver had been recognized previously as a significant feature of childhood malnutrition (Halliday, 1967) but had not been reported to occur in Aboriginal children. It was suggested that fatty change in the liver might indicate a pre-kwashiorkor condition in young Aborigines (Gracey and Bower, 1973).

Personal experience of Government Medical Officers in the Kimberley indicated that the livers of Aboriginal children, when examined at post-mortem, were often large and pale and their cut surface often oozed fatty droplets. Further evidence for malnutrition in Aboriginal children came from a group of patients investigated in Princess Margaret Hospital in whom the plasma levels of *b*- lipoprotein were low and who had thorny red blood cells (acanthocytes) in their blood (Gracey, Wilson and Patersen, 1972); in contrast to previous reported studies (Bassen and Kornzweig, 1950), these features were found to be reversible with nutritional rehabilitation.

Other workers found evidence of widespread malnutrition in Aboriginal children. As early as 1958 Crotty had commented on the prevalence of

anaemia and malnutrition in Aboriginal children in the Northern Territory, then Kettle (1966) showed that the growth rates of young Aborigines in Arnhem Land were substantially behind that of the accepted normal patterns of growth in non-Aboriginal Australian children. Reports from other parts of Australia which appeared about the same time made it clear that this was then a widespread problem (Kirke 1969; Maxwell and Elliott, 1969; Jose and Welch, 1970; Walker and Harry, 1972). There was indirect evidence, too, for undernutrition, especially in young Aborigines and in lactating and pregnant Aboriginal women; this included biochemical evidence of deficiencies of various vitamins (Kamien *et al*, 1974; 1975). Regrettably, there is very little reliable, quantitative information about dietary practices and nutrient intakes in Kimberley Aborigines which might help explain the prevalence of nutritional disorders. Another difficulty in assessing the nutritional state of Aboriginal people is the absence of 'normal' growth charts for Aborigines; the growth standards which are used are based on measurements of non-Aboriginal children (usually American, British or Australian standards) which may not be appropriate for Aboriginal children, particularly in view of the differences in size and shape of the Aboriginal skeleton compared to that of Europeans (Abbie, 1974).

Overall, the published information available in the early 1970's left the impression that Aborigines in the Kimberley region of Western Australia shared a standard of health more in keeping with developing or Third World countries than what was expected by the average Australian at that time. Many government-sponsored and other strategies were implemented in the 1970's to help improve this inequitable situation. Is there reliable information available to show whether these efforts have been successful?

Changing patterns

Western Australia was the first State in the nation to code hospital admissions according to race. This computerised Hospital Morbidity Data Collection applies to every hospital admission, separation (discharge) and transfer and includes clinical information, the major diagnosis, age, sex, race and postcode. This now allows a retrospective analysis so that some idea of trends over time can be assessed (Berry and Gracey, 1981).

A recently published report (McNeilly *et al*, 1983) has analysed data on admissions of all Aborigines and non-Aborigines aged 0-15 years in all parts of the State for gastroenteritis, lower respiratory tract infections and other infections for the decade 1971 to 1980. Overall, there was a decline in the number of Aboriginal infants and children admitted to hospital with infections. The most marked decline occurred in admissions for gastroenteritis and other infections in the Kimberley region.

The rates of hospitalization of Aboriginal infants for gastroenteritis in the Kimberley fell sharply from 393% in 1971 to 24% in 1980; this means that on average, each Aboriginal infant was hospitalized four times in 1971 before the age of 12 months, while in 1980 only one in four infants was hospitalized on one occasion before their first birthday. The rates for 'other infections' fell from 260% p.a. in 1971 to 47% p.a. in 1980. For children under 5 years of age (i.e. 1-4.9 years) rates of admission to hospital for gastroenteritis fell very sharply in the first half of the decade in Aboriginal children (78% per year to 24%

per year) and continued to fall to 9% in 1980. The rates of admission for 'other infections' dropped dramatically from 132% in 1971 to 20.5% in 1980. Rates for lower respiratory tract infections were 8% in 1971, peaked at 18% in 1972, and had fallen to 2% by 1980.

These are very encouraging improvements which show how serious public health problems can be made to respond to appropriate preventive health programmes; they have been accompanied by a similar drop in deaths of Aboriginal infants and young children from gastroenteritis (Berry and Gracey, 1981). However, there is no room for complacency or, perhaps, even for satisfaction with these results. There remains a very wide gap between hospitalization rates for infectious diseases of Aboriginal and non-Aboriginal infants and children in Western Australia. For example, in 1980, the rate of hospital admissions for gastroenteritis of Aboriginal infants was still *10 times* that of non-Aboriginal infants and children up to five years of age (McNeilly *et al*, 1983). A similar gap exists for other infectious diseases. The improvements which have been documented (see above) are considered due to 'several interrelated factors, including improvement in the general standard of living, housing and hygiene, and the provision of comprehensive, community-based health programmes'. There is an urgent need for maintenance and reinforcement of projects aimed at improving these factors. Very significantly, 'environmental contamination' has been singled out as a major factor causing continuing ill health in Aboriginal communities (McNeilly *et al*, 1983). This boils down to better standards of living, hygiene and nutrition, improved water supplies and sewage disposal, and more appropriate individual and community hygiene practices. These must all play major roles in improving the health of Kimberley Aborigines.

Nutrition of older children

Most studies of the nutritional state of Aboriginal children are confined to the first few years of life (Coles-Rutishauser, 1979; Gracey *et al*, 1983; Gracey, 1986); not much is known about their subsequent growth patterns in later childhood and adolescence - whether, for example, they have a period of 'catch-up' growth or whether some children remain permanently stunted and wasted. We have analysed anthropometric data from almost all Aboriginal schoolchildren in the Kimberley (1887 subjects in all) in relation to age, sex and whether the children lived in the main Kimberley towns or in remote communities (Hitchcock *et al* unpublished data). We found that Aboriginal children from remote communities were lighter and shorter than those living in the towns; these differences were present at birth and throughout childhood. Overall, the median weights and heights of Aboriginal children were similar to the 10th centile for weight and the 25th centile for height of non-Aboriginal children; in other words, there was a significant growth deficit in Aboriginal children which was more severe in remote communities and more marked for weight (wasting) than for height (stunting).

Maternal health and nutrition

Ill-health in Aboriginal infants and children is still a major concern in the Kimberley. When considering factors responsible for this, one must consider how the health and well-being of Aboriginal mothers might influence that of their infants. Low-birth-weight babies (LBW) are

prevalent in Aborigines (Seward and Stanley, 1981) and a recent cross-sectional anthropometric survey of almost 400 Aboriginal girls and women of child-bearing age in the Kimberley has shown that nutritional disturbances are widespread in that nutritionally vulnerable group (Gracey *et al*, 1984). Less than 60% of these females were satisfactorily nourished; one-sixth were undernourished and one-quarter, mostly older women, were overweight. We have recently shown that undernourished Aboriginal mothers have depressed levels of the anti-infective breast milk protein, lactoferrin, in their breast milk which might help explain why their infants are particularly susceptible to gastrointestinal and respiratory infections (Houghton *et al*, 1985). We are presently undertaking a study of the inter-relating factors which influence growth, nutrition and health patterns in Aboriginal infants born into several remote communities in the Fitzroy Valley area. This is a prospective, longitudinal study in which the babies will be studied until they are 2 years of age. Results from the study should start emerging in 1987.

Trace element deficiency?

Workers from the CSIRO Division of Human Nutrition in Adelaide have concentrated on the possibility that impaired growth and undernutrition in Aboriginal children may be due, at least in part, to trace element deficiency. Smith *et al (1982) showed widespread growth deficits, more marked for weight than for height, in older Aboriginal children (5-15 years) in the Kimberley and the same children had depressed hair zinc and plasma zinc levels when compared with values found in non-Aboriginal control subjects. Earlier work by Cheek and his colleagues had shown a deficit of protein reserves in Aboriginal children in relation to body length (Cheek et al.* 1978) suggesting that inadequate protein intakes might be contributing to the poor nutritional state of Aboriginal children. Other studies by the same group have detected low levels of zinc and high levels of copper in the blood in Aborigines in coastal communities in Dampier Land (Beagle Bay, Lombadina and One Arm Point) which they speculated might be due to poor zinc absorption because of phytates in damper, inadequate intakes in the diet or excessive losses in the sweat; pica was thought also to be a possible explanation (Cheek *et al*, 1981). Aborigines at other locations including La Grange, Mowanjum, Fitzroy Crossing and Kalumburu were also shown to be affected (Cheek *et al*, 1982). A later study (Cheek *et al*, 1984) showed that the zinc content of white blood cells (lymphocytes and granulocytes) and red blood cells (erythrocytes) were significantly reduced in Aboriginal boys aged 6 to 13 years; because the turnover of white blood cells is relatively fast, they suggested that white cell zinc content might be a better indicator of the individual's zinc status. Later work confirmed that levels of zinc in blood cells, urine and hair were depressed; furthermore, there is chronic growth retardation, sexual maturation is delayed as measured by plasma levels of dehydroisoepiandrosterone sulphate (DHEAS), cell mass is reduced, and nitrogen turnover is probably increased: all these observations tending to support the case for zinc deficiency in these Aboriginal children (Cheek *et al*, 1985).

These studies led to a double blind intervention trial of supplementary zinc involving 200 Aboriginal children aged 5-15 years in the Kimberley; however, despite evidence of subnormal zinc status there was no response to supplemental zinc (Smith *et al*, 1985). It is not really surprising that these children did not respond to supplementation

of their diet with a single substance (in this case, zinc) when they have other, multiple nutrient deficiencies and when repeated and chronic infections contribute so substantially to their unsatisfactory growth patterns. Recent work by the Adelaide group has, in fact, shown that dietary patterns of Aboriginal children in the Kimberley are suspect with potential deficiencies of Vitamin A, folic acid, calcium, and vitamins B, and B6 being quite likely due to their habitual dietary patterns as assessed by questionnaire/recall methods and checks on foodstore purchasing patterns (King *et al*, 1985). The main nutrient deficiency to emerge from these studies, and very importantly, is inadequate dietary energy. Much more work is needed to be done on this very important and practical aspect of the nutrition of Aboriginal people in the Kimberley. Before we have reliable information about what current dietary patterns exist, it will be very difficult to set about improving them and impossible to assess what changes are very likely to be introduced after the introduction of mining, tourism and the spread of urban patterns of food distribution and consumption in the region. This is already happening with 'take-aways' and other convenience foods in the larger towns, particularly with the increasing mobility of Aboriginal people from even the most remote settlements.

Urbanization and life-style disease

The rapid loss of traditional lifestyle and hunter-gatherer activities has already had a profound effect on the health and morbidity levels of Aborigines in Western Australia. In a 'Report on the Health of Kimberley Natives' (Elphinstone, 1960) the following comments are very pertinent:

> Obesity is now the commonest ... disorder of nutrition found in the Kimberley natives. The cause is the obvious one of overeating and under-exertion ... The habit of overeating is presumably a relic of the days when the nomadic native lived mainly by hunting and was obliged to gorge after a successful hunt to tide him over to the next kill. Although he can now be sure of his next meal, the instinct to gorge is still there. .... 'the carbohydrates are proportionately excessive' .... 'the native is very fond of very sweet tea, sweet biscuits, soft drinks and canned fruits.

It should be borne in mind, of course, that Dr Elphinstone's comments were made before drinking rights were obtained by Aborigines and before alcohol abuse had become widespread and entrenched in the lifestyle of many remote Aboriginal people. Elphinstone goes on to remark that:

> One encounters now, much more often than in the past, those diseases commonly associated with obesity - (including) hypertension (and) diabetes (among others).'

Also that:

> The easy, almost casual, childbirth traditionally associated with native women, is no longer the rule.

Dr L.J. Holman of Derby believes that the native female's bony pelvis is adequate for the small foetus of a mother living on natural bush food, but

too small for the larger foetus of a mother living on white man's food.'
Subsequent studies have clearly documented some of those risk factors to
which Elphinstone alludes.

Bastian (1979) surveyed 248 full-blood Aborigines in the West
Kimberley area (mainly from Derby, Mowanjum, Looma and Fitzroy Crossing)
for the prevalence of coronary heart disease. He found that 7% of
Aboriginal men and 12.6% of Aboriginal women had evidence of 'probable'
coronary heart disease (c.f. 6% in Busselton); 7% of the Aborigines
studied had electrocardiographic evidence of cardiac ischaemia (c.f. 4% in
Busselton). Bastian found that the major risk factors contributing to the
high prevalence of coronary heart disease in these Kimberley Aborigines
were hypertension (40% prevalence in males, 34% in females), diabetes
mellitus (14.5% in males and 20.7% in females; c.f. <2% in non-
Aborigines) and obesity.

Studies by O'Dea and her colleagues have thrown considerable light on
the problem of diabetes mellitus in Aborigines over the past several
years; much of this has been community-based field work in remote
Aboriginal groups in the Kimberley region. Surveys from several parts of
Australia (Wise et al, 1970; Kamien, 1976; Wise et al, 1976; Duffy et
al, 1981) including the Kimberley (Finlay-Jones and McCormish, 1972) have
suggested an overall prevalence of diabetes from 8% to 19% in Aborigines
compared to the rate of 2.3% quoted for non-Aboriginal people as judged
from work done in the Busselton community in Western Australia (Welborn et
al, 1968). O'Dea et al (1982) found an overall incidence of diabetes of
4.5% in Aborigines over 25 years of age in the remote, coastal community
of Kalumburu and an incidence of 9.4% for those over 35 years of age in
the same community. An 'urbanized' community of Aborigines living at
Mowanjum and studied at the same time showed similar patterns of glucose
tolerance and insulin response to oral glucose, although the men at
Kalumburu had significantly lower fasting glucose concentrations.
Responses to insulin in the two Aboriginal groups were very similar and
50% higher than in non-Aboriginal subjects. The authors considered that
elevations in fasting cholesterol and glucose concentrations were related
to lifestyle but that mild impairment of glucose tolerance and high
insulin responses in Aborigines might be inherited characteristics (O'Dea
et al, 1982).

O'Dea and Spargo (1982) found that reversion of Kimberley Aborigines
to a traditional diet for 2 weeks had important effects on metabolic
responses in these people. The traditional diet was derived almost
exclusively from estuarine seafood (fish, shellfish), snails and berries
and was low in fat, extremely low in carbohydrate and high in protein.
This 'experimental' diet was accompanied by initial weight loss, a fall in
plasma triglyceride concentrations and a small but significant improvement
in glucose tolerance and a small reduction in insulin response - all
indicating an effective adaptation to the diet. More detailed analysis of
the diet showed the tropical seafood to be a natural source of
polyunsaturated fatty acids (O'Dea and Sinclair, 1982), particularly
arachidonic acid (Sinclair et al, 1983). A later, more prolonged dietary
study of these Mowanjum people (O'Dea and Sinclair, 1985) involved a 2-
week diet of tropical seafood, followed by a 3-week diet in which kangaroo
and freshwater fish (rich in linoleic acid and arachidonic acid) were the
main sources of fat; both diets were very low in fat. This dietary
manipulation was accompanied by increased incorporation of arachidonic
acid into plasma lipids and an increased bleeding time.

Healthy, lean, young Aborigines at Kalumburu, where diabetes is common in older adults, had mild-impairment of glucose tolerance, hyperinsulinaemia, and elevated levels of very-low-density lipoprotein (VLDL) lipids (O'Dea et. al, 1982; O'Dea, 1983). It has been suggested that these characteristics somehow enhanced survival in the past among hunter-gatherer groups, the so-called 'thrifty gene' (Neel, 1963), but made these people very prone to type 2 (non-insulin-dependent) diabetes mellitus when they adopted an urbanized style of life (Reaven et al, 1976) when life was no longer a series of 'feasts and famines' and food became plentiful at all times. The main metabolic abnormalities of type 2 diabetes can be either greatly improved or completely corrected by reverting to their traditional hunter-gatherer lifestyle for several weeks (O'Dea, 1984). Alterations included a fall in fasting glucose, improved post-prandial glucose clearance, halving of plasma insulin concentrations, improved insulin responsiveness and a marked fall in fasting plasma triglycerides, largely because of the drop in VLDL concentration. There are at least three factors which were operating in these middle aged diabetics which could contribute to these improvements: (i) weight loss, (ii) the fact that the traditional diet was low-fat in content, and (iii) increased physical activity which their resumed hunter-gatherer activities entailed. O'Dea (1983, 1984) has emphasized the public health importance of these observations and the importance of regular physical activity, low-fat diets and control of body weight in the prevention and management of type-2 diabetes in Aboriginal people. These observations and comments are very relevant to the long-term effects of diet and lifestyle in childhood and early adult life on the development of cardiovascular and other degenerative diseases in Aboriginal people in later life.

Enteric disease surveillance

The Enteric Diseases Section of the State Health Laboratories in Perth has done extensive studies over many years on intestinal pathogens throughout Western Australia. Among other findings, they have shown the rich diversity of types of *Salmonellae* to be found in the Kimberley region, including several varieties of *Salmonella* which were discovered there and entered into the international register of *Salmonella* species; these include *S. fitzroy*, *S. jubilee*, *S. kalumburu*, *S. kimberley* and *S. mowanjum*. Their regular reports show a consistently high rate of isolation of *Salmonellae* and other bacterial and parasitic intestinal pathogens from the Kimberley region, particularly from Aborigines. Their work has also included studies of isolation of enteric pathogens in environmental sources and a wide variety of animals including livestock, feral animals, and wild indigenous animals such as mammals, marsupials, birds and reptiles. The report by Jones (1980) has re-emphasized the importance of intestinal parasite infections in Aborigines from the Kimberley, particularly infections with hookworm, *Strongyloides stercoralis*, *Giardia* and *Hymenolepis*.

The work from the State Health Laboratories (e.g. Iveson, 1983; Iveson, 1978; Iveson and Bamford, *submitted*; Iveson et al. 1969; Iveson, 1973; How et al, 1983; State Health Laboratory Services, 1985) is very valuable since it highlights the risks to local inhabitants of environmentally acquired gastrointestinal and other infections. The Health Department and its State Health Laboratory Services would be a very important resource which might be able to be used to monitor changes in these environmentally acquired infections in future, consequent on changes

in standards of hygiene which might result from pressures from tourism, mining, other industries and increased population in remote parts of the Kimberley.

Chronic ear infections

Sunderman and his colleagues have clearly documented the serious nature of chronic ear disease in Aboriginal children in the Kimberley region (Sunderman and Dyer, 1984). This is a major cause of hearing loss in young Aborigines and is a widespread and pressing problem which causes a great deal of personal distress and discomfort, frequent and prolonged medical and nursing care, and very serious educational disadvantages in Aboriginal children who already have many other social and political reasons for being educationally disadvantaged in comparison with their non-Aboriginal peers. These workers believe that chronic infection with the organism, *Chlamydia trachomatis*, is a very important cause of chronic otitis media in Aborigines; further work is needed on this point and about what are the most appropriate preventive measures which could be undertaken to control this serious problem. Dr. Sunderman has a very extensive bibliography about ear, nose and throat diseases in Aborigines which can be made available to interested persons, on request from the E.N.T. Clinic, Sir Charles Gairdner Hospital, Perth.

Eye disease

The National Trachoma and Eye Health Program (Royal Australian College of Ophthalmologists, 1980) included the Kimberley in their studies of trachoma and other eye disease in remote Australia. In the area they called 'Cattle Country', which takes in most of the inland Kimberley and crosses over into the northern part of the Northern Territory, 72% of Aborigines had evidence of trachoma (66.7% of those under 20 years and 91.7% of those aged 60 and over); only 2.3% of non-Aboriginal people in the Cattle Country were found to have trachoma.

There has been significant improvement in the prevalence of trachoma in the Kimberley over recent years. The overall prevalence of follicular trachoma fell from 22% in 1977, to 12% in 1985, while the rates for children up to 9 years of age have fallen from 52% to 15% over the same period (Department of Health, Western Australia, *Departmental Report*, 1986.

Thyroid function

Dick and Watson (1980) found that among Aborigines surveyed in the Halls Creek area, 40% had thyroid-binding globulin (TBG) levels below the lower reference limit for Europeans and 18% of Aboriginal subjects had levels between half and one-third of the lower limit. Such subjects have low levels of circulating thyroxine (by European standards) and their tri-iodothyronine uptake (T_3U) tests – a marker of normal thyroid function – would give the false impression of impaired thyroid activity. This apparent variation in thyroxine-binding globulin (TBG) is not confined to Kimberley Aborigines but occurs throughout Aboriginal people in Western Australia.

Kirk and his colleagues at the Australian National University (Canberra) have studied genetic characteristics of Aboriginal people in various parts of Australia, including the Kimberley. A recent study (Blake and Spargo, in press) has examined red cell enzyme systems in Kimberley Aborigines from 13 tribal groups which has emphasized the uniqueness of these people (Blake, 1979).

Leprosy

Leprosy is a unique scourge which has affected susceptible individuals in the Aboriginal population of the Kimberley this century and has caused considerable public interest and concern because of the usual perceptions of this infectious disease. The numbers of inpatients with this disease in the Derby leprosarium peaked in the 1940s (Table 1).

Table 1:   Leprosarium inpatients as at 31st of December

YEAR	PATIENTS
1936	86
1946	231
1956	198
1966	178
1976	38
1986	7

Effective treatment of clinical leprosy with drugs only became possible with the finding of the bacteriostatic effect of the Sulphones in the 1940s. After prolonged periods of treatment with this drug, patients could become negative on skin testing. The official policy adopted was to continue treatment until the patient had been negative for two years then to discharge the patients back to their communities. In those days there was no way to supervise outpatient drug treatment so therapy was ceased on discharge. There was subsequently a high rate of relapse among patients discharged.

During 1952 and 1953, there were 52 patients discharged after prolonged treatment for lepromatous leprosy. Five years after discharge, 25% had relapsed; by 10 years after discharge, 42% had relapsed.

Various methods of follow-up treatment were tried but supervision was inadequate and the relapse rate remained high. Of 26 patients discharged in 1962 and 1963, 38 percent had relapsed after 10 years. Two developments subsequently had considerable impact upon the relapse rate; in the early 1970s a network of community based nurses was established in the Kimberley. Later, a depot preparation of Dapsone (Acedapsone) was introduced. Out-patient drug management was then removed from the patient and given to the health team   - overcoming the difficult problem of compliance. During 1972 and 1973, 36 patients were discharged from the

leprosarium to supervised out-patient maintenance drug regimes. After 10 years only 5.5 percent had relapsed.

As there is no method of primary prevention (with the possible exception of neonatal BCG vaccination) leprosy control programmes are very dependent on secondary prevention. This means reducing the number of infective people occurring in the community. Overcoming the compliance problem helps reduce the frequency of relapse, so reducing the number of infective foci present in the community. From Table 2 the impact of the reduction in relapsed lepromatous leprosy is evident.

Table 2: Admissions to Derby Leprosarium, 1964-1983

Categories of admission		The decade 1964-73	The decade 1974-83
LEPROMATOUS	NEW	60	48
	RELAPSED	94	22
	TOTAL	156	70
TUBERCULOID		105	36

A neonatal BCG immunization programme was introduced in the late 1960s. Since then, every infant of Aboriginal descent born in the Kimberley has had a BCG vaccination. Table 2 shows the number of cases of tuberculoid leprosy admitted has decreased significantly from 105 in the decade 1964-73 to 36 in the next decade. With the recognition that the leprosy bacillus (*Mycobacterium leprae*) was becoming resistant to Dapsone and with episodes of Dapsone insensitivity being reported from many countries, it became important to introduce multiple drug regimes to manage lepromatous leprosy. Two important therapeutic advances have been the introduction of B663 (Clofazamine) and Rifampicin.

The use of these drugs combined with Dapsone has had significant impact upon the management of lepromatous leprosy:

(i) Severe reactions in lepromatous leprosy are now not occurring,

(ii) The time to obtain negative split skin smears is markedly reduced.

Important recent knowledge about leprosy includes the following:

(i) It is more infectious than previously thought,

(ii)   Only a small percentage of an infected population will develop clinical disease,

(iii)   Prolonged contact is not necessary to become infected,

(iv)   Nose-to-nose-spread-by droplets is highly-likely;

(v)   *M. leprae* can remain viable outside the human host for at least several days,

(vi)   There is a long incubation period which complicates its control. [The average incubation period for multibacillary leprosy admitted to Derby Leprosarium between 1979 and 1983 was more than 12 years],

(vii) Each individual incubating *M leprae* helps encourage widespread transmission, long before the development of clinical disease, long before the case can be detected and treatment implemented,

(viii) There is strong presumptive evidence that the incidence and Prevalence of *M. leprae* infection far exceeds clinical leprosy in endemic areas,

(ix)   A major impediment to the control of leprosy is the non-availability of an effective and practical method of specific protection of those exposed to *M leprae* (primary prevention).

    The number of patients admitted to Leprosarium who have a Kimberley origin are shown in Table 3.

    At the present time (mid-1986), there are approximately 400 patients with leprosy on the Kimberley Register; 217 are on treatment and 179 are on surveillance only. The effectiveness of control measures over leprosy in the Kimberley region has permitted the Derby Leprosarium to be closed (September 1986).

Table 3:   Admissions of patients with leprosy to Derby Leprosarium

Year	New Paucibacillary	Multibacillary New	Multibacillary Relapse
1960	7	12	22
1965	12	7	19
1970	16	5	11
1975	-	5	6
1980	2	1	2
1985	-	1	0

It is now possible, given the opportunity to closely supervise multi-drug schedules, for prolonged periods to prevent mutilation and deformity in leprosy. This is the real triumph in any leprosy programme. It is expected that the endemic will continue in northern Australia, so long as there are individuals in the endemic area who have not yet been exposed to *Mycobacterium leprae* and as communities are reached by the slow spread southward of the endemic area.

When considering possible future trends in leprosy in Kimberley Aborigines, the following factors should be considered. The genetically most susceptible pool of persons in northern Australia has now been reduced, at least in those areas where transmission has occurred. Subclinical infection on a wider scale has resulted in significant group immunity. The neonatal BCG immunization programme has probably enhanced resistance to *M. leprae* in Aborigines in the Kimberley and, lastly, chemotherapy has been moderately successful in controlling relapses.

It is expected that there still are individuals in the endemic area who are susceptible to intense transmission. Occasionally subclinical infections will escape detection in the elderly and there will be communities on the edges of the endemic area who have not been exposed to the leprosy bacillus. The occurrence of poorly understood factors, e.g. virulence factors in different species of background environmental mycobacteria, will help determine whether transmission leading to clinical infection will occur in future.

Sexually transmitted diseases

Sexually transmitted diseases are widespread throughout the Aboriginal population of the Kimberley. Syphilis became notified much more frequently in Western Australia from the late 1960s and spread rapidly through the Aboriginal population, establishing itself in the hinterland of the Kimberley region. From time to time Aborigines living an integrated lifestyle in Kimberley towns are involved in the transmission of the disease following intermittent contact with the isolated hinterland groups.

The increasing prevalence of treponemal infections is not confined to the Kimberley - other regions in Western Australia, the Northern Territory and other States have reported an increasing frequency of syphilis in Aboriginal populations. The numbers of notifications of gonorrhoea and syphilis in Western Australia from 1953 to 1985 are shown in Figure 1; this shows quite clearly the sharp upswing in notifications, initially for gonorrhoea and then a couple of years later for syphilis, commencing in the late 1960s. Subsequently, a disproportionate number of notifications for sexually transmitted diseases has been from Aborigines, particularly in the Kimberley and Pilbara regions. In 1977, for example, more than 50% of the State's notifications for gonorrhoea, syphilis and granuloma inguinale were from these two relatively sparsely populated regions alone. This may be partly explained by the higher level of screening, case finding and contact tracing, and certainly a higher compliance with notification of STD in those remote regions in comparison with the rest of Western Australia. For the State as a whole, compliance with notification for STD is estimated to be only 25-40% (CDJ Holman - personal communication).

Syphilis in the remote Kimberley region behaves unlike the disease in urban Australia where it is mostly confined to a relatively small, particular behavioural group. In remote, northern Australia this disease is widely endemic in the Aboriginal population and the very few Europeans who are infected in the region are, without exception, 'frontier type' males who have a lifestyle closely associated with the Aboriginal population.

The transmission of syphilis in Kimberley Aborigines is not always venereal; this is because of ecological and behavioural characteristics of the Aboriginal groups in hinterland communities which facilitate the transmission of the disease non-venereally. This explains the occasional presentation of syphilis in sexually inactive children and the elderly. Clinical presentation in such individuals is usually at the secondary stage of the disease and methods of case detection and treatment are available to treat these patients.

The first cases of granuloma inguinale (G.I.) were detected about 1980 in the Aboriginal population of the south-east Kimberley who have regular contact with remote Aboriginal groups in the Northern Territory. Subsequently, there has been a slow spread of the disease to involve individuals in communities in the more arid parts of the region. The precise mode of spread of this disease is uncertain; however formal investigation of contacts has confirmed the presence of G.I. in many of the sexual partners of index cases. There were 18 cases diagnosed in the Kimberley in 1985.

The occurrence of gonorrhoea in Kimberley Aborigines should be seen as a consequence of rapidly changing social factors in today's Kimberley community. With Aborigines in a period of hasty transition, many young Aboriginal adults now seek freedom from traditional cultural constraints and they are now being increasingly influenced by wider social attitudes of the whole population in the region. These younger people are much more 'independent' and have increased mobility and tend now to live in a type of forced integration with larger population groups than was the case even a few years ago. Social security payments have fostered the financial independence and physical mobility of these people. Increased use of alcohol as a lubricant or catalyst for their individual and group interaction has facilitated the altered behaviour patterns which are occurring. As a result, Kimberley Aborigines have entered their first era of sexual freedom which is causally related to the high frequency of gonorrhoea which they are experiencing.

Deaths due to 'external causes' in young adults

Social changes in the Kimberley in the past decade have encouraged a new and modern lifestyle which has its own attendant risks. An example of this is the rapidly increasing ownership of motor vehicles by groups and individuals in country towns and remote communities. These vehicles are often driven over large distances and on sub-standard roads and are sometimes heavily over-loaded with passengers in their cabins and unprotected external carrying compartments. Accidents involving such vehicles have caused single and multiple deaths in the region over recent years. As in other parts of Australia, alcohol usage is involved in a significant proportion of these incidents. Some of these accidents are responsible for the unexpectedly high numbers of deaths of young adult

males. Unfortunately, these deaths are recorded only as being due to 'injuries and poisoning' in officially available reports (Holman and Quadros, 1986). In 1983-84, ten out of 14 deaths of Aboriginal males in the age group 20-35 years and 3 out of 5 deaths of Aboriginal females in the 15-30 years age group were in this category. The risk to young adult males who are not Aboriginal seems to be just as high : in 1983-84 all but 2 of the 13 deaths in the 20-35 years age group were due to 'injuries and poisonings' (1 out of 2 deaths of non-Aboriginal females was in the same category). Homicide and suicide are responsible for some of the sudden and violent deaths in young Aboriginal men over recent years.

Conclusion

In conclusion, the following emerge as important trends in the changes affecting health of Aboriginal people in the Kimberley region from the early 1970's to the present (in no particular order):

- greatly improved infant mortality;

- reduction of preventible infectious diseases including gastroenteritis, respiratory infections and 'immunizable' diseases such as tuberculosis, polio myelitis, whooping cough and measles;

- decreased deaths and hospitalization from those childhood diseases;

- improved medical, nursing and hospital staffing, facilities and services. Preventive services in obstetrics and child health have been specially important;

- improved communications and transport;

- improved living conditions and standards of hygiene;

- rapid changes in socio-political circumstances and 'lifestyles' which are being accompanied; by

- very high rates of preventible 'lifestyle' diseases such as obesity, diabetes, coronary heart disease and hypertension

- alarmingly high rates of morbidity and mortality association with alcohol abuse, personal and communal violence, and sexually transmitted diseases.

These dramatic changes in health and disease pose many challenges for the Aboriginal Medical Services which are now assuming responsibility for Aboriginal health in the region.

GAINS AND LOSSES IN THE HEALTH OF ABORIGINES IN THE KIMBERLEY OVER THE PAST 20 YEARS

Improvements	Deterioration
Improved infant mortality	Increased prevalence of obesity, hypertension, diabetes and coronary heart disease
Suppression of infectious diseases through immunization (e.g. tuberculosis)	Alcoholism and related disorders
Control and suppression of leprosy	Sudden, violent deaths including motor vehicle accidents, homicide and suicide
Decreased hospitalization of children for infections such as gastroenteritis and respiratory tract infections	Increased incidence of sexually transmitted diseases including syphilis
Improved obstetric outcomes	

References

Abbie, A.A. 1974.
Physical standards of nomadic Aboriginal children.
Med. J. Aust. 1: 470.

Bassen F.A., Kornzweig, A.L. 1950.
Malformation of the erythrocyte in a case of atypical retinitis pigmentosa.
Blood 5: 381.

Bastian P. 1979.
Coronary heart disease in tribal Aborigines - The West Kimberley Survey.
Aust. NZ J. Med. 9: 284.

Berry, R.J., Gracey, M. 1981.
Diarrhoeal disease in Aboriginal and non-Aboriginal infants and young children in Western Australia.
Med. J. Aust. 1: 479.

Blake, N.M. 1979.
Genetic variation of red cell enzyme systems in Australian Aboriginal populations.
Occasional papers in Human Biology, 2. Canberra : Australian Institute of Aboriginal Studies.

Blake, N.M., Spargo, R.M. 1986.
Population genetic studies in the Kimberley of Western Australia.
Human Heredity 36: 286.

Cheek, D.B., Graystone, J.E., Holt, A.B., Sutherland, G.C., Chopra, S.A., Spargo, R.M. 1978.
Assessment of protein reserves (cellular mass) in Aboriginal children.
Am. J. Clin. Nutr. 31: 1328.

Cheek, D.B., Ness, D., Hay, H.J., Ludgwigsen, N., Lattanzio. L., Spargo, R. 1984.
Zinc content of red and white blood cells in Aboriginal children.
Aust. NZ J. Med. 14: 638.

Cheek, D.B., Smith, R.M., Spargo, R.M. 1982.
Hair and plasma zinc levels in Aboriginal Australians.
In *Clinical Applications of Recent Advances in Zinc Metabolism*: 151-167. New York : Alan R. Liss Inc.

Cheek, D.B., Smith, R.M., Spargo, R.M., Francis N. 1981.
Zinc, copper and environmental factors in the Aboriginal peoples of the North-West.
Aust. NZ J. Med. 11: 508.

Cheek, D.B., Wishart, J, Phillipou, G., Field, G., Spargo, R. 1985.
Zinc status, body composition, and maturation in Aboriginal boys.
In R.K. Chandra (ed) *Trace Elements in Nutrition of Children*: 209-227. New York : Nestle Nutrition, Vevey/Raven Press.

Coles-Rutishauser, I.H.E. 1979.
Growing up in Western Australia : if you are Aboriginal.
Proceedings of the Nutrition Society of Australia 4: 27.

Crotty, J.M. 1958.
Anaemia and nutritional disease in Northern Territory native children.
Med. J. Aust. 2: 322.

Dick, M., Watson, F. 1980.
Prevalent low serum thyroxine-binding globulin level in Western Australian Aborigines. Its effect on thyroid function tests.
Med. J. Aust. 1: 115.

Duffy. P., Morris, H., Neilson, G. 1981.
Diabetes mellitus in the Torres Strait region.
Med. J. Aust., Special Supplement 1: 8.

Elphinstone, J.T. 1960.
In *Report of the Commissioner of Public Health, Western Australia*: 59-62. Perth.

Finlay-Jones, R.A., McCormish, M.J. 1972.
Prevalence of diabetes in Aboriginal lepers : the Derby survey.
Med. J. Aust. 2: 135.

Gracey, M. 1973.
Enteric disease in young Australian Aborigines.
Aust. NZ J. Med. 3: 576.

Gracey, M., Bower, G. 1973.
Fatal diarrhoeal disease in Australian Aboriginal children.
Aust. NZ J. Med. 3: 255.

Gracey, M. 1985.
Aborigines and health (Leading Article).
Med. J. Aust. 143: S43-S44.

Gracey, M., Murray, H., Hitchcock, N.E., Owles, E.N., Murphy, B.P. 1983.
The nutrition of Australian Aboriginal infants and young children.
Nutr Res. 3: 133.

Gracey, M., Spargo, R.M., Bottrell, C., Hammond, K., Mulholland. K.,
Valentine, J. 1984.
Maternal and childhood nutrition among Aborigines of the Kimberley region.
Med. J. Aust. 141: 506.

Gracey, M, Wilson, R.G, Petersen, M. 1972.
Transient Acanthocytosis and Hypobetalipoproteinaemia.
Aust. NZ J. Med. 4: 397.

Hitchcock, N.E., Gracey, M., Maller, R.A., Spargo, R.M. (submitted)
Physical size of Aboriginal schoolchildren in the Kimberley region.

Holman, C.D.J., Quadros, C.F. 1986.
Health and disease in the Aboriginal population of the Kimberley
region of Western Australia 1980-1985.
Perth : Health Department of Western Australia.

Houghton M.R., Gracey. M., Burke, V., Bottrell, C., Spargo, R.M. 1985.
Breast milk lactoferrin levels in relation to maternal nutritional
status.
J. Pediatr. Gastroenterol. Nutr. 4: 230.

How, R.A., Bradley, A.J., Iveson, J.B., Kemper, C.M., Kitchener, D.J.,
Humphreys, W.F. 1983.
The natural history of *Salmonellae* in mammals of the tropical
Kimberley region, Western Australia.
Ecology of Disease 2: 9.

Iveson, J.B. 1973.
Enrichment procedures for the isolation of *Salmonella, Arizona,
Edwardsiella* and *Shigella* from faeces.
J. Hyg. (Cambridge) 71: 349.

Iveson, J.B. 1978.
Enteric parasites in Western Australia 1960-1977. A summary of
protozoa and helminths identified in humans.
West Australian Health Surveyor: 19-26, September.

Iveson, J.B. 1979.
Ecological aspects of Salmonella infections in Western Australia with
particular reference to the Kimberley region.
West Australian Health Surveyor: 37-41, June.

Iveson, J.B. 1983.
Regional aspects of *Salmonella* in humans, livestock, feral animals
and wildlife in Western Australia.
West Australian Health Surveyor, Autumn.

Iveson, J.B., Bamford, V.W. (submitted)
Arizona and *Edwardsiella* in developing tropical regions and urban
areas of Western Australia.

Iveson, J.B., Mackay-Scollay. E.M., Bamford. V. 1969.
Salmonella and *Arizona* in reptiles and man in Western Australia.
J. Hyg. (Cambridge) 67: 135.

Jones, H.I. 1980.
Intestinal parasite infections in Western Australian Aborigines.
Med. J. Aust. 2: 375.

Jose, D.G., Welch, J.S. 1970.
Growth retardation, anaemia and infection, with malabsorption and
infestation of the bowel. The syndrome of protein-calorie
malnutrition in Australian Aboriginal children.
Med. J. Aust. 1: 349.

Kamien, M. 1976.
 The physical health of Aboriginal adults in Bourke, New South Wales.
 Med. J. Aust. 1: 38, Special Supplement.

Kamien, M., Nobile, S., Cameron, P., Rosevear, P. 1974.
 Vitamin and nutritional status of a part-Aboriginal community.
 Aust. NZ J. Med. 4: 126.

Kamien, M., Woodhill, J,M., Nobile, S., Rosevear, P., Cameron, P.,
 Winston, J.M. 1975.
 Nutrition in the Australian Aborigine : A dietary survey of two
 Aboriginal families.
 Food Tech Aust. 27: 93.

Kettle, E.S. 1966.
 Weight and height curves for Australian Aboriginal infants and
 children.
 Med. J. Aust. 1: 972.

King, R.A., Smith, R.M., Spargo, R.M. 1985.
 Dietary patterns of Aboriginal children in the Kimberley.
 Proceedings of the Nutrition Society of Australia 10: 173.

Kirke, D.K. 1969.
 Growth rates of Aboriginal children in Central Australia.
 Med. J. Aust. 2: 1005.

Maxwell, G.M., Elliott, R.B. 1969.
 Nutritional state of Australian Aboriginal children.
 Am. J. Clin. Nutr. 22: 716.

McNeilly, J., Cicchini, C., Oliver, D., Gracey, M. 1983.
 Infectious disease in Aboriginal infants and children in Western
 Australia.
 Med. J. Aust. 2: 547.

Neel, J.V. 1963.
 Diabetes mellitus : a 'thrifty' genotype rendered detrimental by
 progress?
 Am. J. Hum. Genet. 14: 353.

O'Dea, K. 1982.
 The relationship between urbanisation and diabetes in Australian
 Aborigines.
 Proceedings of the Nutrition Society of Australia 7: 30.

O'Dea, K. 1983.
 Lifestyle change and diabetes in Australian Aborigines.
 Aboriginal Health Project Information Bulletin 4: 17, Canberra.

O'Dea, K. 1984.
 Marked improvement in carbohydrate and lipid metabolism in diabetic
 Australian Aborigines after temporary reversion to traditional
 lifestyle.
 Diabetes 33: 596.

O'Dea, K., Sinclair, A.J. 1982.
Increased proportion of arachidonic acid in plasma lipids after 2 weeks on a diet of tropical seafood.
Am. J. Clin. Nutr. 36: 868.

O'Dea, K., Sinclair, A.J. 1985.
The effects of low-fat diets rich in arachidonic acid on the composition of plasma fatty acids and bleeding time in Australian Aborigines.
J. Nutr. Sci. Vitaminol. 31: 441.

O'Dea, K., Spargo, R.M. 1982.
Metabolic adaptation to a low carbohydrate - high protein ('traditional') diet in Australian Aborigines.
Diabetologia 23: 494.

O'Dea, K., Spargo, R.M., Nestel, P.J. 1982.
Impact of Westernization on carbohydrate and lipid metabolism in Australian Aborigines.
Diabetologia 22: 148.

Reaven, G.M., Bernstein. R., Davis, B., Olefsky, J.M. 1976.
Non-ketotic diabetes mellitus : Insulin deficiency or insulin resistance?
Am. J. Med. 60: 80.

Royal Australian College of Opthalmologists. 1980.
National Trachoma and Eye Health Program.
Sydney.

Seward, J.F., Stanley, F.J. 1981.
Comparison of Births to Aboriginal and Caucasian mothers in Western Australia.
Med. J. Aust. 2: 80.

Sinclair, A.J., O'Dea, K., Naughton, J.M. 1983.
Elevated levels of arachidonic acid in fish from Northern Australian coastal waters.
Lipids 18: 877.

Smith, R.M., King, R.A., Spargo, R.M., Cheek, D.B., Field, J.B., Veitch, L.G. 1985.
Growth-retarded Aboriginal children with low plasma zinc levels do not show a growth response to supplemental zinc.
Lancet i: 923 (letter).

Smith, R.M., Spargo, R.M., Cheek, D.B. 1982.
Zinc status and growth of Aboriginal children in the North-West of Australia.
Proceedings of the Nutrition Society of Australia 7: 37.

State Health Laboratory Services. 1985.
Quarterly Report, April-June 1985, Enteric Pathogens isolated in Western Australia.
Perth : Health Department, Western Australia.

Sunderman, J., Dyer, H. 1984.
  Chronic ear disease in Australian Aborigines.
  Med. J. Aust. 140: 708.

Walker, A.C., Harry, J.G. 1972.
  A survey of diarrhoeal disease in malnourished Aboriginal children.
  Med. J. Aust. 1: 904.

Welborn, T.A., Curnow, D.H., Wearne, J.T., Cullen, K.J., McCall, M.G.,
    Stenhouse, N.S. 1968.
  Diabetes detected by blood sugar measurement after a glucose load :
  report from the Busselton Survey, 1966.
  Med. J. Aust. 2: 778.

Wise, P.H., Edwards, F.M., Craig, R.J., Evans, B., Murchland, J.B.,
    Sutherland, B., Thomas, D.W. 1976.
  Diabetes and associated variables in the South Australian Aboriginal.
  Aust. NZ J. Med. 6: 191.

Wise, P.H., Edwards, F.M., Thomas, D.W., Elliott, R.B., Hatcher, L.,
    Craig, R. 1970.
  Hyperglycaemia in the urbanised Aboriginal - the Davenport Survey.
  Med. J. Aust. 2: 1001.

Legend to figure

Notifications of Syphilis (•) and Gonorrhoea (o) in Western Australia, 1953-1985.