Malnutrition is prevalent amongst children in many parts of the world, particularly in the so-called developing countries. Scrimshaw et al. in their pioneering studies helped to delineate the contribution of infectious diseases to the aetiology of childhood malnutrition. This role was reinforced by longitudinal field-based studies in rural areas of Central America and Bangladesh which documented the contribution particularly of episodes of diarrhoea, to acute and chronic malnutrition, and high rates of death in infants and young children in such environments. We now understand that, because of the negative impact of malnutrition on humoral, mucosal and cellular immune defence mechanisms, children from vulnerable backgrounds become enmeshed in a vicious cycle of 'malnutrition — infection — malnutrition'. Infectious diarrhoeas are particularly important in this context because of anorexia, vomiting, gastrointestinal losses of fluids, nutrients and electrolytes, loss of body weight and the negative effect of infection on energy balance. Furthermore, this situation is often complicated by co-existing diseases such as respiratory tract infections, intestinal parasites and, in many parts of the world, malaria. Despite intense local and international efforts over recent years, malnutrition, vaccine-preventable diseases and other infections kill millions of infants and children annually around the world.

MALNUTRITION IN YOUNG AUSTRALIAN ABORIGINES

In the 1960s it was shown that malnutrition and infections were widespread in Aboriginal infants and young children in this country, an observation which shocked many Australians, caused a public outcry and led to the development and implementation of many government-sponsored and other initiatives to try to correct this inequity. In the Northern Territory, Ellen Kettle had attempted to develop normal growth curves for Aboriginal children by studying, prospectively and monthly, more than 200 Aboriginal infants from coastal mission settlements in Arnhem Land. However, many of her subjects had gastrointestinal and respiratory infections, parasitic infestations and severe anaemia which often cause unsatisfactory growth patterns in early childhood. Crotty had previously described anaemia and malnutrition in Aboriginal children in the Northern Territory and later work from Central Australia re-emphasised the widespread nature of growth faltering and failure to thrive in young Aborigines. Moodie highlighted the poor standards of health by collating information about morbidity and mortality in Aboriginal children from different parts of Australia in a book and an annotated bibliography on this subject. This has been brought up to date to the mid-1980s. Jose and Welch, in more than 2,000 children from six Aboriginal settlements in Queensland, found half of those aged six months to three years were growth retarded, one half of that group being anaemic and infected as well. They described this as 'growth retardation, anaemia and infection with malabsorption and infestation of the bowel' and, drawing the analogy with the situation in children in developing countries, referred to this as 'the syndrome of protein-calorie malnutrition'. Such reports and many subsequent ones established that undernutrition and infections are common and serious problems in Australian Aboriginal children.

*This paper is an abridged version based on the Annie B. Cunning Lecture delivered at the Annual Scientific Meeting of the Royal Australasian College of Physicians, held in Perth, WA, May, 1991.
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GASTROINTESTINAL INFECTIONS

Gastrointestinal and respiratory tract infections predominate as causes of morbidity and mortality in young Aborigines. Moodie46 showed that 'dysentery and gastroenteritis' and 'influenza and pneumonia' between them caused about half the deaths in Aboriginal children under five years of age in Western Australia, South Australia, New South Wales and the Northern Territory. In 1969, although only about 4% of the childhood population of Western Australia, of admissions to the State's only children's hospital, Aborigines accounted for 46% of 'deficiency anaemias', 27% of nutritional disorders, 19% of rheumatic fever, 13% of pneumonia and 9% of gastrointestinal infections.18 During the eight years 1971 to 1978, although only 3.7% of the under-five-year population of Western Australia in 1976 (a census year), Aborigines accounted for 42% of admissions to Princess Margaret Hospital for Children, Perth, for gastroenteritis and 58% of bed occupancy for that disease.16 The age-specific admission rates for Aboriginal infants (0-12 months) were more than 20 times those for non-Aboriginal infants and for Aboriginal children aged 12 months to five years, 16 times. However, over the period of that study (early to late 1970s) there was a substantial decline in hospital admissions of Aboriginal infants and young children in Western Australia for diarrhoeal diseases. This was confirmed in a later report yet the hospitalisation rate for gastroenteritis for Aboriginal infants by the mid-1980s was still 20 to 25 times the rate for other infants and their rate of hospital bed usage was 40 to 50 times that for other infants.16 Part of the reason for the even greater discrepancy in 'bed usage' is that Aborigines spend more than twice as long in hospital as other patients for gastroenteritis and 58% of bed occupancy for that disease.16 The age-specific admission rates for Aboriginal infants (0-12 months) were more than 20 times those for non-Aboriginal infants and for Aboriginal children aged 12 months to five years, 16 times. However, over the period of that study (early to late 1970s) there was a substantial decline in hospital admissions of Aboriginal infants and young children in Western Australia for diarrhoeal diseases. This was confirmed in a later report yet the hospitalisation rate for gastroenteritis for Aboriginal infants by the mid-1980s was still 20 to 25 times the rate for other infants and their rate of hospital bed usage was 40 to 50 times that for other infants.16 Part of the reason for the even greater discrepancy in 'bed usage' is that Aborigines spend more than twice as long in hospital as other patients for gastroenteritis13,16,19,20 because of more serious disease as well as distance, lack of transport, immobilisation by severe weather, difficulties with follow-up supervision and the reluctance of medical practitioners to discharge patients into environments or family circumstances where re-infection is likely.17

Aboriginal infants and children experience diarrhoea caused by viruses, bacteria and a wide variety of intestinal parasites. The parasites most commonly encountered include *Giardia lamblia* and *Hymenolepis nana*,21-23 *Trichiuris trichiura*, *Strongyloides stercoralis* and hookworm.24-25 Their distribution varies geographically; hookworm, for example, tends to occur in moister coastal communities in the tropics or temperate regions but can occur in arid areas where permanent puddles are allowed to develop (e.g. under leaking air conditioners or near leaking taps or drains) and where environmental contamination is not actively discouraged. Hookworm infestation is hyperendemic in many coastal communities in northern Australia, sometimes occurring in more than half the children under five years,26 and is a frequent cause of severe anaemia from gastrointestinal bleeding in young Aborigines.27 Infestation with *Strongyloides stercoralis* can be so heavy as to cause partial intestinal obstruction.28 In Aboriginal children with diarrhoea, intestinal bacterial infections are important, for example, enterotoxigenic *Escherichia coli* (ETEC), enterotoxigenic *Achromonas* *Salmonella*, *Shigella* and *Campylobacter*. Intestinal parasites such as *Giardia lamblia* are very common but are as frequent in Aboriginal children without diarrhoea as from those who have it.29 Viral infections have been associated with outbreaks of diarrhoea in Central Australia.30 Rotaviruses and coronavirus-like particles have both been detected in faecal samples from Aborigines.31 Gastrointestinal infection with *Candida* species also occurs in malnourished Aboriginal children.32

Undernourished Aboriginal children have, in addition, bacterial contamination of upper intestinal secretions33 as occurs in malnourished children in other parts of the world.34-36 They also have extensive histological damage in the upper intestinal mucosa,37,38 associated with secondary lactose intolerance.39 After weaning, Aboriginal children have a high prevalence of late-onset hypolactasia, with lactose malabsorption.40,41

A problem with most of the published reports is that they are from patients in hospitals, hospital admission statistics or *ad hoc* cross-sectional surveys and therefore provide little information about the extent or severity of diarrhoeal disease in children in Aboriginal communities. A study in remote communities in tropical north-western Australia showed that isolations of enterotoxigenic *E. coli* (ETEC) were much more frequent in the wet monsoonal summer than in the dry winter season even in children under five years who did not have diarrhoea and were believed not to have had diarrhoea recently.42 A recent, year-long, prospective, community-based study of more than 100 Aboriginal children in the Kimberley region showed that isolations of bacterial or viral pathogens were significantly associated with diarrhoea.43 A range of bacterial agents including ETEC, *Salmonella* spp., *Shigella* spp. and *Campylobacter* as well as the intestinal parasites *Giardia*, *Cryptosporidium* and *Strongyloides* were isolated frequently from Aboriginal children in their first two years of life in the same region.44

Respiratory tract infections cause three or four times the number of admissions of Aboriginal infants and young children that gastroenteritis does.18 Chronic respiratory disease, sometimes resulting in bronchiectasis, has been recognised as a common problem in Aborigines for many years.11,45-48 Rates of admission of young Aboriginal children (12 months to five years) and infants are five to ten times those of non-
Aborigines of the same ages. These illnesses may have serious long-term consequences since they are the single most important cause of admission to hospital of Aborigines of all ages.

Lobar pneumonia occurred 10 times more frequently in Aboriginal than in non-Aboriginal children in the district of Bourke (NSW) in the first three years of life and multiple recurrences occurred in more than one-third of Aboriginal children. Pneumonia tended to occur when siblings were affected by the disease or when families lived in sub-standard housing. A more recent study in Bourke showed that admission rates for pneumonia and diseases such as gastroenteritis and eye and ear infections declined, despite deterioration in employment opportunities and adult health, possibly because of better nutrition in early childhood, better housing conditions and better access to health care.

OTHER INFECTIONS
Other endemic infections of particular note in Aboriginal children are diseases of the skin and scalp, chronic otitis media, often associated with hearing loss, trachoma, meningitis, and post-streptococcal glomerulonephritis.

Trachoma, a troublesome eye infection caused by *Chlamydia trachomatis*, has left many Aborigines in remote communities permanently blinded and is very closely related to living conditions. Its prevalence increased dramatically in the 'Top End' of the Northern Territory between 1950 and 1955 then declined substantially by about 2% per annum, i.e. before mass treatment programmes were begun in the late 1970s; in Central Australia the prevalence seems constant or even increasing, highlighting the need for better living conditions for Aborigines. An outbreak of gonococcal conjunctivitis in more than 100 Aboriginal children in Central Australia reminds us of this possibility. Appropriate antibiotic treatment results in dramatic improvement.

Meningitis is a serious problem and in the Northern Territory *Haemophilus influenzae* is the most important cause of bacterial meningitis in Aboriginal children; vaccination may be required to reduce the risk, particularly below 18 months of age. The incidence of bacterial meningitis in West Australian Aboriginal children is seven times that of other children; 60% of episodes are due to *H. influenzae* and the case fatality rate is 10% compared with the non-Aboriginal rate of 4.1%.

Chronic ear disease and hearing loss have important educational and social dimensions as well as medical consequences. In a recent study from Western Australia, 3.2% of ears in Kwinana (near Perth), 10% in Wiluna and 13.8% in La Grange were perforated, which seems substantially fewer than in earlier studies.

Chronic renal disease is increasing in Aboriginal adults, considerable numbers requiring dialysis or renal transplantation. Much of this later morbidity is probably related to earlier post-streptococcal disease and urinary tract infections associated in turn with urinary tract calculi which are much more common in Aboriginal children.

Communicable diseases such as hepatitis B, dengue, hydatid disease, and melioidosis, are particularly important in Aborigines, no doubt because of geographical location and the unfavourable environments in which most of them live. Several recent deaths from measles in Central Australia emphasise the importance of protection of these vulnerable children against vaccine-preventable diseases. A survey of more than 200 children in that region showed only 73% were fully immunised; 23% were partly immunised and 4% were unimmunised or information was unavailable.

MULTIPLE INFECTIONS
Aboriginal children often have multiple infections and multiple causes for diarrhoea such as viruses, bacteria or intestinal parasites. This is reflected in hospital admission patterns in remote areas; in the regional hospital in Derby, WA, in 1984 90% of admissions involved Aboriginal children, 60% of admissions were for children under two years of age and multiple conditions such as respiratory and gastrointestinal infections, renal disease, failure to thrive and anaemia were common. This heavy burden of infections is well illustrated by a community-based, prospective study of 48 infants born into several Kimberley communities, examined monthly for 24 months; 60% of infants were hospitalised in their first six months, 65% from two to 12 months of age, 67% from 12 to 18 months and 56% between 18 months and two years, most frequently for respiratory and gastrointestinal infections, failure to thrive, conjunctivitis, other febrile illnesses, anaemia, social reasons or because of being abandoned.

GROWTH PATTERNS IN ABORIGINAL INFANTS AND CHILDREN
Very high infection rates in Aboriginal infants and children are not surprisingly associated with widespread growth faltering, failure to thrive and frank malnutrition which have been documented repeatedly since the 1960s (see earlier and other references, e.g. 76-79).

The 'average' Aboriginal infant is a little lighter than non-Aboriginal infants at birth but growth appears to be adequate in the first several months of life. However, attained weight-for-age often falls behind the median (or 50th centile) body weight at about six months (see Figure 1) although weight velocity (i.e. the rate of weight gain) begins to slow well before this
Faltering of growth between six and 12 months of age is characteristic in Aboriginal infants and this period is the time of main negative impact on growth. Weight gain rates in Aboriginal children do not recover until 18 months to two years of age, by which time the 'average' Aboriginal boy is 1.5 kg lighter and 4.5 cm shorter and the 'average' Aboriginal girl 1.1 kg lighter and 3.6 cm shorter than the expected median measurements. In Aboriginal children in north-west Australia, 'catch up' growth is usually lacking or ineffective. In 2,000 Aboriginal school children, substantial deficits persisted till at least 15 years of age (Table 1). This is in agreement with experience with Guatemalan children stunted in the first five years of life who were likely to be permanently stunted as adults. The study by Hitchcock et al. showed that Aboriginal children in more remote communities were smaller in weight and height than those in towns; retrospectively, these differences had been present at birth and throughout childhood.

Low birthweight (<2,500 g) is more than twice as common in Aboriginal babies than in the rest of the community and is the earliest detectable nutritional setback in young Aborigines. It is probably related to maternal factors including undernutrition, urinary tract infections, anaemia, hypertension, inadequate antenatal supervision, smoking and alcohol consumption. It has been linked to impaired growth in Aboriginal children for at least the first five years of life.

There is some evidence to suggest that standards of nutrition and growth of Aboriginal children have improved over recent years. Patterns of growth in Aboriginal children in Cherbourg, Queensland, in the early 1980s were very similar to international reference values. This is a very important point because it is sometimes argued that genetic differences between Aborigines and other people invalidate the use of international reference values based on heterogeneous samples of the United States population. There is general agreement among auxologists that these values, which have been adopted by the National Health and Medical Research Council, are valid for comparisons...

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Figure 1: Mean weights of Kimberley Aboriginal boys (*) monthly from birth to two years of age in comparison with international 50th centile reference values (x-x) (NCHS, 1977) at intervals over the same age period (from Gracey and Sullivan, 1989 — with permission from 'Annals of Human Biology').

Figure 2: Mean weight velocities of Kimberley Aboriginal boys (*) from birth to three months, three to six months, six to nine months, nine to 12 months, 12-18 months, and from 18-24 months compared to the mean weight velocities between international (NCHS, 1977) reference values (x-x) over the same age intervals (from Gracey and Sullivan, 1989 — with permission from 'Annals of Human Biology').
of growth and that children from almost all populations will grow very much like the NCHS values, given adequate nutrition and environmental circumstances. Kimberley Aboriginal children grow satisfactorily in comparison with the international references, depending on maternal and environmental factors.87,88 Favourable maternal factors include absence of anaemia and other illnesses during pregnancy, adequate pregnancy weight gain, regular antenatal supervision and lack of smoking or alcohol consumption; favourable environmental factors relate to personal and family hygiene, rubbish disposal and access to food supplies.

CONCLUSION

Growth responds to a combination of genetic and environmental factors. For Aboriginal infants and children to reach their potential for growth, living conditions, standards of hygiene and nutrition must be improved. In doing so, long-term consequences and health patterns must be anticipated. We should recognise, for example, the trend for increasing body mass index (BMI) in Aborigines, even during childhood41 and its association with obesity, cardiovascular disease and non-insulin dependent diabetes mellitus in Aboriginal adults.76,91 Simply ‘feeding up’ Aboriginal children will achieve little for them without cognisance of more comprehensive present and future health needs.

Acknowledgements

I wish to thank the many colleagues who have assisted with my studies of Aboriginal health over 20 years including my wife, Valerie Burke, and Helen Sullivan and Randolph Spargo. Particular thanks are given to Aboriginal Health Workers and other members of the Health Department of Western Australia for their co-operation and friendship.

Date of submission: 11 June 1991

CHILDHOOD MALNUTRITION IN ABORIGINES

| Age (years) | Boys | Girls |
|-------------|------|-------|
|             | Kimberley NCHS | Diff | Kimberley NCHS | Diff |
| 5           | 17.2 | 16.9  |   -1.5 | 17.7 | -0.8 |
| 10          | 27.4 | 30.2  |   -2.8 | 32.6 | -2.4 |
| 15          | 52.3 | 45.4  |   -6.9 | 53.7 | -7.7 |

*The observed 50th centile measurements are shown along with the 50th centile NCHS values (National Center for Health Statistics, 1977).

TABLE 1

Observed and Expected Sizes* of Kimberley Aboriginal Children (Hitchcock et al.)

| Age (years) | Weight (kg) | Height (cm) |
|-------------|-------------|-------------|
|             | Kimberley NCHS | Diff | Kimberley NCHS | Diff |
| 5           | 110.1 109.9 +0.2 | 109.6 108.4 +1.2 | 52.7 52.5 -0.2 |
| 10          | 135.8 137.5 -1.7 | 138.8 138.3 +0.5 | 58.5 58.3 -0.2 |
| 15          | 165.2 169.0 -3.8 | 155.6 161.8 +5.2 | 63.7 63.5 -0.2 |

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