

Zinc deficiency and Aboriginal health

You can see the kids running round tyepe-tyepe (lively or happy) and healthy. They eat bush tucker: goannas, turkey, perentie. See, they're healthy.

People didn't get much sickness in the olden days. They been living on good food. We got white man's disease today. We got white man's tucker.

These comments made by Northern Territory Aborigines* show the effects of the past 200 years on their health and traditional life style. In this last quarter of the twentieth century, Australia's Aborigines remain the sickest group of people living on the continent — a Third World nation within a developed country.

In their 1979 report on Aboriginal health, the House of Representatives Standing Committee on Aboriginal Affairs documented the poor health facilities of the communities. The findings of the National Trachoma and Eye Health Program's Report (1980) provided evidence of the high incidence of communicable and degenerative diseases among Aborigines and of its clear association with unsanitary and poorly serviced living environments.

Patterns of morbidity and mortality among these people are alarming. According to calculations in the 1978/79 Northern Territory Department of Health Annual Report, Aborigines living in the Territory can expect an infant mortality rate of 48 deaths per 1000 births, compared with 9 per 1000 for the non-Aboriginal population. They have an average life expectancy of 50.

Indeed, the whole Australian Aboriginal population has a 'top-heavy' age structure. It contains almost twice the proportion of children and adolescents found in non-Aboriginal communities, and about half the proportion of aged. A typical Aboriginal family has twice as many dependants as a typical non-Aboriginal family.

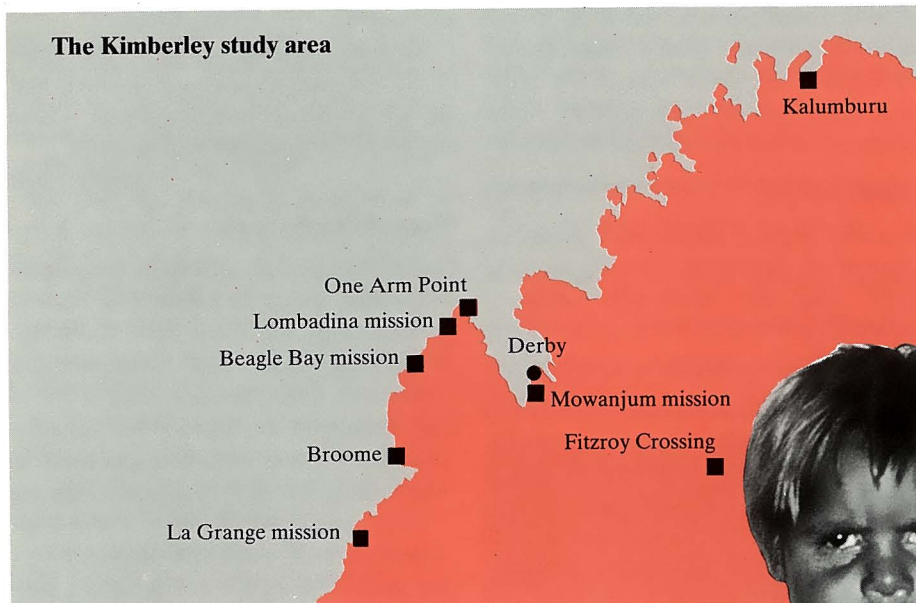
Most Aborigines in northern Australia will experience during their lifetime some



These women are preparing bush turkey in a camp oven.

or all of the chronic ailments that afflict their communities: underdevelopment during childhood and adolescence, reduced immunity to disease, failure of wounds to heal, inability to mobilize vitamin A, eye problems (particularly trachoma), and skin and renal problems.

Inadequate diet is often identified as one of the immediate causes of the Aboriginal health problem, with its effects most obvious in the young. In 1974, the Princess Margaret Hospital in Perth, W.A., recorded 932 admissions of 624 Aboriginal infants and children, of whom more than 50% were malnourished. In 1976, although



*quoted in 'Health Business'. P. Nathan and D.L. Japanangka. (Heinemann Educational: Melbourne 1983.)



Aborigines under 15 years made up only 4% of Western Australia's population, they accounted for 39% of State hospital admissions for gastroenteritis in the State.

In his study of the medical aspects of Aboriginal health, Dr Peter Moodie from Sydney gave a summary of the disease burden of a 'not unusual' Aboriginal child admitted to hospital with 'a small wardful of illness in the same body': the presenting illness (say bronchopneumonia) + iron-deficiency anaemia + roundworm and whipworm infestation + giardiasis + ringworm + pediculosis + diarrhoea + lactose intolerance + chronic suppressive otitis media + severe dental caries + a behavioural disturbance'.

Zinc is essential for the activity of a least 100 enzymes.



Witchetty grubs — a well-known bush food.

No fully documented studies of the nutrition of Australian Aboriginal children have been made. However, results of a joint investigation by the CSIRO Division of Human Nutrition, the Western Australian Department of Public Health, and the University of Adelaide have identified a possible source of many of the above illnesses — a deficiency of the trace element, zinc.

Brisk growth response

In 1977, a team headed by Professor Donald Cheek of the University of Adelaide and Dr Randolph Spargo of the Western Australian Department of Public Health carried out a study of the protein reserves of Aboriginal children in the Alice Springs and Derby areas. The findings indicated that the children had very low levels of zinc in their blood (a condition known as hypozincaemia). The researchers made a further study of zinc, copper, and iron con-

centrations in the blood serum and plasma of 363 Aborigines aged 5–70 years living in two inland settlements in the Kimberley region of Western Australia.

At these two settlements, Mowanjum and Fitzroy Crossing, researchers found not only hypozincaemia but also a generally low blood level of iron (hypoferraemia) and a high level of copper (hypercupraemia), especially in the younger age groups.

Why study zinc? The metal is one of the human body's trace elements; that is, it occurs in the body at concentrations of less than 0.01%. Unlike the macro-elements (carbon, hydrogen, nitrogen, calcium, and phosphorus), which are more abundant in the body and often serve in a structural capacity, trace elements generally perform a regulatory or catalytic role.

Zinc is essential for the activity of at least 100 enzymes, including those involved in the metabolism of nucleic acid (DNA and RNA) and in protein synthesis. This means that it is important to the growth of the cell itself and to cell multiplication. In the immune system, lack of it leads to a lack of cell-mediated immunity. Zinc cannot be stored by the human body in reserves, but must be continually absorbed from food items such as meat, fish, and nuts.

Affected children showed a brisk growth response when given zinc.

Human zinc deficiency was first reported in 1961 by Dr Ananda Prasad of Wayne State University, Michigan, U.S.A. He and his co-workers found low plasma zinc concentrations in severely growth-retarded Iranian children. A study of the children's diet revealed that they ate very little animal protein. Instead, they consumed large amounts of unleavened whole-grain bread. The phytate (inositol hexaphosphate, a substance associated with plant products such as those of soybean and cereals) present in this bread has been shown to bind with zinc, preventing the small amount of zinc in an already deficient diet from being completely absorbed in the gut.

Since this study, nutritionists in other parts of the world have observed that growth retardation and lack of immunity to disease accompany zinc deficiency. In some cases this deficiency, indicated by low levels of the metal in blood plasma, was associated with low iron and high copper levels.

Diet is not the only cause of the deficiency. Research on Egyptian adolescents

— by Dr Harold Sandstead of the Human Nutrition Laboratories, North Dakota, U.S.A. — revealed a different mode of depletion — zinc loss through bleeding into the bowel as a result of hookworm infestation.

Another contributing factor in both studies in the Middle East was the practice of geophagia (the eating of soil) among children. Minerals in clay readily bind with zinc and some of the available zinc in the digestive tract is adsorbed onto the clay particle surface.

Whatever the cause of the deficiency, the outcome of trials in which children were given zinc supplements left little doubt as to its nature. In Egypt, and more recently in Jamaica, affected children showed a brisk growth response when given zinc.

After they became aware of the existence of hypozincaemia in the inland missions of Western Australia, Dr Spargo, Professor Cheek, and their colleagues directed their attention to coastal areas, where meat, fish, and fresh fruit are more likely to appear in the diet. Throughout the region, flour and sugar products contribute heavily to the total calorie intake. Additionally, the rate of hookworm infestation is lower in these coastal communities than inland. They wanted to see whether the differences in climate, environmental factors, and parasite infestation led to a different health picture in the Aboriginal communities.

Instead, the studies at the coastal settlements of One Arm Point, Lombadina, and Beagle Bay produced the same results — hypozincaemia, hypercupraemia, and hypoferraemia were prevalent and the children were significantly underweight.

Hair zinc

During the coastal mission survey, Dr Richard Smith of the Division of Human Nutrition in Adelaide began an analysis of the zinc levels in the hair of 208 children, aged 5–15 years, in coastal missions. Hair analysis provides evidence of zinc status over the period of hair shaft growth, complementing plasma and serum zinc studies, which are an index of immediate zinc concentrations.

Dr Smith's initial results showed extremely high copper levels in the hair of children at One Arm Point. Later, an explanation for this surfaced — local copper contamination through erosion of copper pipes by bore water. Nevertheless, in non-contaminated environments, in which hair copper levels were not elevated, zinc levels were still abnormally low in both the hair and plasma, and plasma copper levels were consistently high.

In their most significant finding, Dr Smith and his colleagues established that, weight for height, Aboriginal children from the Kimberley region of Western Australia fell well below the 'normal' growth patterns derived from United States National Centre for Health statistics and from a control group of Caucasian schoolchildren in Adelaide.

Examination of similar growth data from central, northern, and western Australian settlements and communities showed that a uniform pattern existed for children aged 1–18 years, regardless of location. Aboriginal children in settlements across the whole of northern Australia slowed down in growth after 6 months of age and continued to develop slowly into maturity, with individual weight being more affected than height.

In 1982, Dr Smith and Dr Spargo again visited the missions at One Arm Point, Lombadina, and Beagle Bay. They examined 166 children, aged 3–16 years, for height, weight, arm circumference, blood plasma, and scalp hair. Additionally, they asked 120 children to recall the food they had eaten in the previous 24 hours. The researchers then compared this information with foodstore records for the previous 12 months from two of the missions. They converted the dietary and foodstore data into quantities of nutrients, using a computer program based on food composition tables.

Again they used a group of Adelaide schoolchildren as a reference population — and again found significantly lower zinc and iron levels in the plasma and hair in the Aboriginal children than in the Caucasian reference group.

They saw serious sickness as being caused by bad spirits.

Plasma copper levels were higher than those found in non-Aboriginal children in the region. About 85% of this copper could be accounted for in caeruloplasmin, the usual protein carrier of copper in the blood, but the identity of the remaining 15% was uncertain.

Further investigation indicated that the 'left-over' copper represented a genetically determined condition, and that Aboriginal ancestry was the dominant factor. While non-Aboriginal children in the region had lower copper levels, any Melanesian or Caucasian contributions to the Aboriginal ancestry conferred no decrease in plasma copper among children of mixed racial origin. Dr Smith suggests that some uniden-



Oysters such as these at One Arm Point showed below-average zinc levels.

The fruit and vegetable garden at La Grange.

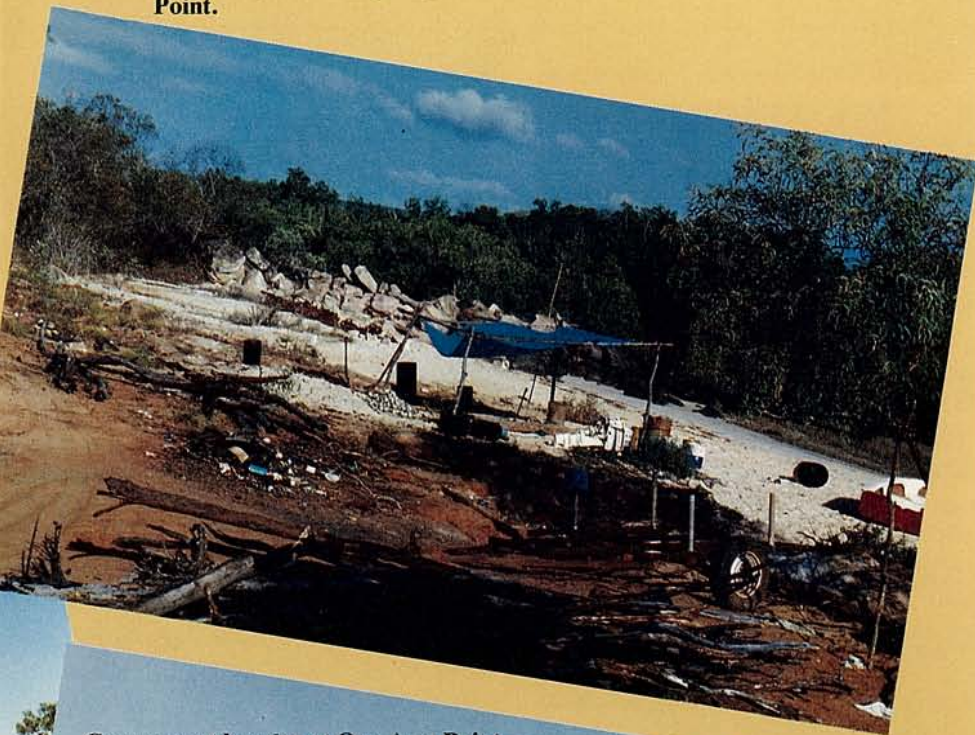
Their diet probably used to be high in protein, roughage, and vitamins.



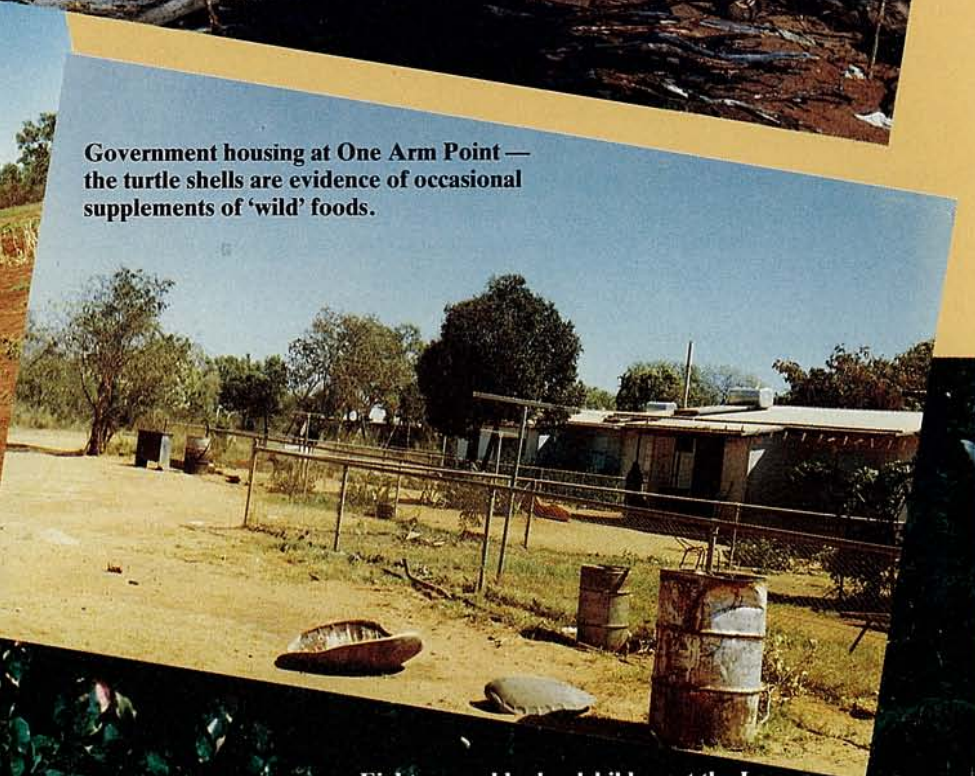
Children fishing — who needs bait?



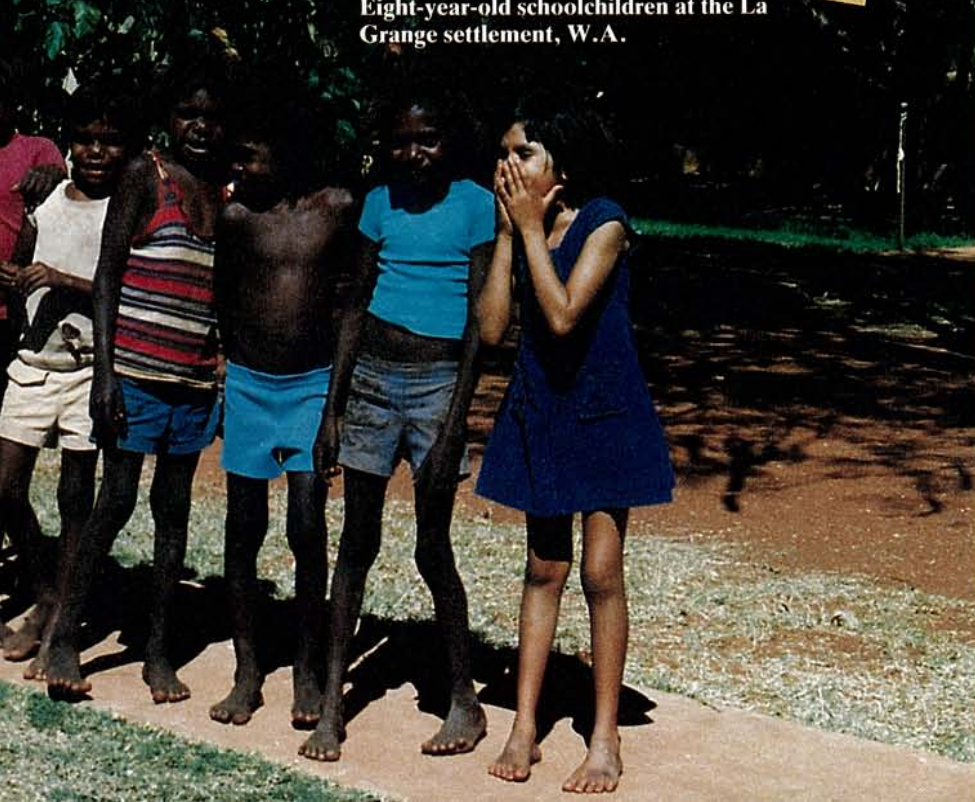
An Aboriginal fishing camp at One Arm Point.



Government housing at One Arm Point — the turtle shells are evidence of occasional supplements of 'wild' foods.



Eight-year-old schoolchildren at the La Grange settlement, W.A.



tified blood component that binds copper may be responsible for the excess, since copper ions at such high concentrations are toxic or lethal.

Oysters and mangoes

For all of the communities — whether inland or coastal — bread, damper, sugar, and tea were staple foods. Most of the coastal missions had fruit trees, and occasionally meat and fish from hunting and fishing excursions appeared in the diet there, in addition to meat purchased from the stores.

Two lines of evidence suggested that the area around the coastal settlements is poor in zinc. First, fruit trees, especially mangoes, showed a marked growth improvement when fertilized with zinc. Second, analysis of some local oysters revealed zinc concentrations of 20 p.p.m, a value well below the 400 p.p.m. usually found in these shellfish elsewhere.

Fresh vegetables and dairy products were rare items in the daily food intake. Dairy products are rich in calcium, yet another important element that occurred at relatively low levels in the mission children's diet. Dr Smith thinks that the estimated low intake of calcium and folic acid is further cause for concern about the nutritional status of Aborigines in the region.

He believes that the evidence to date is 'consistent with a marginally zinc-deficient diet, leading to depressed growth'.

But causes and symptoms are often indistinguishable from each other. Developmental impairment and gastrointestinal disease associated with poor nutrition cannot be considered in isolation: they partially constitute the complex of disease, mostly infectious, affecting many Aboriginal children. Chronic gastrointestinal disease leads directly to loss of zinc via the bowels. This in turn impairs the immune system and renders the body even more susceptible to disease, including further diseases of the gut. Low levels of vitamin A and plasma albumin may, respectively, contribute to and result from a high incidence of infectious disease, and both conditions are exacerbated by the systemic zinc deficiency, and so on.

Sweat loss

Are factors other than diet in the community environment contributing to the condition of hypozincaemia? Professor Cheek's research led him to believe that, unlike the situation in Egypt, hypozincaemia among Aborigines in north-western Australia was not caused primarily by hookworm or other parasites. For example, at one inland mission, Fitzroy Crossing, the incidence of

Zinc — a recommended daily requirement?

The human body contains between 1 and 2 g of zinc, much of it found in the prostate gland and eye membranes. Although the metal is distributed throughout the body's tissues, it is not stored in a reserve that the body can draw on as other trace elements are. Because zinc is non-cumulative, toxicity through overdose of the metal rarely occurs.

Meat, fish, nuts, and some legumes are good sources of readily available zinc, while vegetables such as spinach are, surprisingly, poor sources. Some foods contain substances — notably fibre and phytate, which is found in soybeans and cereals — that reduce zinc absorption by the body. And, as an added complication, calcium is known to enhance the phytate binding of zinc.

Normally, the body only takes up about one-third of the total zinc ingested, absorbing this through the small intestine. From his investigation of zinc-deficient Iranian dwarfs, Dr Prasad found evidence that the proportion absorbed can increase in cases of serious deficiency. Urinary excretion of zinc is small, about 0.5 mg per day, although up to 1 mg per day can be lost in sweat, which has roughly the same concentration of zinc as plasma.

Zinc is found at the active, catalytic site of many of the body's 'metalloenzymes' and it also appears to regulate the activity of other enzymes. It is involved in the synthesis of proteins and DNA and in the stabilization of cell membranes.

Since Dr Prasad made the first study of human zinc deficiency in the Middle East, studies in the United States, Canada, Scotland, and Sweden have revealed a number of susceptible groups within the population. Often the deficiency can be traced to poor nutrition, as happens for example in economically underprivileged communities and among the elderly.

hookworm among 5- to 20-year-olds was higher and that of hypozincaemia lower than at coastal settlements. Professor Cheek suggested that geophagia may also contribute to zinc deficiency among Aboriginal communities in the region.

Another likely source of severe zinc loss leading to an increased zinc requirement in Aborigines is sweat loss. In 1976, the late Dr Victor MacFarlane of Adelaide University demonstrated that, in summer, desert Aborigines produce up to twice as much sweat as non-Aboriginal Australians, losing up to 6 litres per day. During walks across the desert at 35–38°C, desert Aborigines

Tracking different zinc needs		
group	requirement (mg zinc per day)	recommended dietary intake (mg zinc per day)
infants		
0–6 months	3	3–6
7–12 months	3–4	4.5–6
children		
1–3 years	3–4	4.5–6
4–7 years	4–6	6–9
8–11 years	6–9	9–14
12–15 years	8–12	12–18
16–18 years	8–12	12–18
adults	8–11	12–16
pregnancy	extra 3–4	extra 4–5
lactation	extra 4	extra 6

Dr Dreosti's proposal for zinc requirements and recommended dietary intakes for Australians. The figures are expressed as mean daily intake.

In some groups, the cause is an increased requirement for the trace element, which occurs during adolescent and childhood growth, pregnancy, lactation, and convalescence. Damage to the gut associated with hookworm or inflammatory bowel disease may set up an excessive zinc demand. The heritable diseases acrodermatitis, enteropathica, and possibly sickle-cell anaemia cause their victims to suffer impaired zinc absorption. Increased excretion of zinc can result from kidney disease, liver cirrhosis, hepatitis, diabetes, drugs, trauma, or excessive sweating. Chronic alcoholics often have a poor zinc status, due partly to inadequate nutrition and possibly to increased urinary excretion.

Symptoms of zinc deficiency are similar in man and animals. They include anorexia, growth retardation, delayed wound healing, skin lesions, impaired sexual development, increased susceptibility to infection, behavioural anomalies, and mental

lethargy. In many cases, these defects can be corrected by zinc supplementation.

Nutritionists use intake–excretion balance studies and radioisotope investigations to estimate the dietary requirement for zinc. From the available data, the United States Food and Nutrition Board puts the daily requirement for adults at 15 mg of zinc per day, allowing for the one-third absorption factor.

The Australian National Health and Medical Research Council's 1976 Market Basket Survey listed the mean zinc levels in 44 individual foods sampled around the country. Dr Ivor Dreosti of the CSIRO Division of Human Nutrition used these results to estimate daily zinc intakes of different age groups within Australia. He concluded that most Australians receive an adequate supply of zinc, but stressed the need to pay particular attention to zinc intake when the requirement may be high (as, for example, among pregnant women, nursing mothers, and convalescents), or when intake is below average (such as among vegetarians, the poor, and alcoholics).

So, in drawing up an Australian recommended dietary intake for zinc, Dr Dreosti assigned specific values to a range of age groups, recognizing the changing requirements of the various growth phases of the human body. In the 1983 edition of its publication, 'Recommended Dietary Intakes for Use in Australia', the National Health and Medical Research Council adopted Dr Dreosti's recommendations, including his proposal that zinc be regarded as an essential nutrient.

Zinc. I.E. Dreosti. *Journal of Food and Nutrition*, 1982, **39**, 167–73.

Trace elements in nutrition. I.E. Dreosti. *Medical Journal of Australia*, 1980, **2**, 117–23.

lost 13.6 mL of sweat per kg per hour. Under similar circumstances, Europeans lost 6.5 mL per kg per hour. (The sweat of Aborigines contained twice as much urea and half as much sodium as that of Europeans.)

While Dr MacFarlane made no observations on zinc losses, studies on people of European ancestry have shown that substantial body zinc losses may occur via sweat, which has the same zinc concentration as plasma, and dangerous losses of the metal can occur in hot, humid conditions. In north-western Australia, not only does year-round atmospheric heat persist during

the day, but night temperatures of 30°C are common.

Dr Prasad noted recently that most measurements do not adequately identify a zinc deficiency. Researchers emphasize that, although hair and plasma zinc levels provide statistically acceptable indices for populations, they cannot be used as evidence of a zinc deficiency in an individual.

The researchers also admit that one of the problems they face is an inability to identify a suitable control group of adequately nourished Australian Aboriginal children for comparison of hair and plasma zinc levels.

Zinc, pregnancy, and alcoholism

Trace elements, including zinc, play a significant part in foetal development, and human studies in places as far apart as the United Kingdom, the Middle East, the United States, Turkey, West Germany, and Sweden have indicated that a link exists between sub-optimal zinc status (often as a result of inadequate dietary intake) and congenital defects in the offspring.

During pregnancy, plasma copper levels rise and zinc levels fall. The rise in copper appears to be due to oestrogen-induced synthesis of the blood's copper-carrying protein, caeruloplasmin, and the fall in zinc due to the altered binding activity of zinc-binding proteins and to zinc uptake by the foetus and foetal-maternal membranes.

Dr Dreosti, in collaboration with a group led by Dr Anthony McMichael of the Division of Human Nutrition and researchers from the Queen Victoria Hospital, Adelaide, measured copper and zinc levels in 1000 pregnant women at two stages during pregnancy. In addition, they took umbilical cord blood samples at delivery.

They found that copper levels rose in the maternal serum during gestation, while zinc fell below non-gestational levels. The cord serum copper concentration in the umbilical cord was lower than the mother's level, but cord zinc was higher.

Neither the weight and age of the mother nor, in most cases, the stage of pregnancy appeared to exert a significant effect on zinc and copper levels, but obesity was associated with abnormally high serum copper levels.

In three Aboriginal women included in the study, serum zinc levels rose during pregnancy, which may reflect a genetic difference in zinc metabolism — the researchers are giving this possibility further attention.

Within the general population, pregnant women with low plasma zinc levels tended to produce high-birthweight babies. This challenges the conventional wisdom about low zinc levels in pregnant women. Past animal studies have indicated that low plasma levels associated with inadequate dietary zinc in the mother cause the neonate to be underweight. But among well-nourished pregnant women in the Adelaide study, reduced levels of zinc in the blood seem to result from the foetus channelling a lot of zinc from the mother, resulting in a robust new-born baby.

During the past 10 years, Dr Dreosti has investigated the role of zinc in brain development in animals. His results from

experiments in rats have shown that a maternal zinc deficiency causes physical deformity in the embryo even when the restriction is imposed only briefly during pregnancy. During the early stages of brain development in particular, zinc restriction affects the primitive neural tube, which is the embryonic forerunner of the central nervous system.

Although defects occur in all organ systems as a result of zinc deficiency, Dr Dreosti has observed that the highest incidence of abnormalities — including hydrocephalus ('doming' of the head), anophthalmia (failure of eye development), and exencephalus (congenital fissure of the skull and massive brain deformity, often with development of the brain outside the skull) — occurs in the foetal central nervous system.

Many researchers believe that the deformities may arise from impaired DNA synthesis during embryonic development, which in turn affects cell division in the growing foetal tissues. Dr Dreosti has demonstrated that zinc-deficient rat embryos exhibit reduced cell activity. After injecting a radioactively labelled component of DNA into a number of rat embryos, he found that the incorporation of the labelled substance into DNA inside the foetal brain was significantly reduced in zinc-deficient foetuses relative to controls.

The activity of brain enzymes may also be affected by lack of zinc. Dr Dreosti, together with Mr Ian Record and Ms Susan Manual, also of the Division, showed that higher levels of the zinc-containing enzyme brain alkaline phosphatase occurred in the foetal rat brain than in the adult. Maternal zinc deprivation in 17- to 20-day-old foetuses considerably reduced enzyme levels, highlighting the possibility of further biochemical disturbances that could cause neural tube abnormalities.

In rats, late prenatal and early postnatal zinc deficiency causes behavioural abnormalities as the young rat matures. No apparent deformities of the central nervous system have been noted. Dr Sandstead's group in Grand Forks, North Dakota, U.S.A. suggested that zinc deficiency during the critical period for brain growth not only decreased the total cell count in the brain, but may also interfere with the myelin casing of nerve cell and the formation of interconnections between them.

Apart from an involvement of zinc in the cell and tissue development of the rats' central nervous system, depletion of the

metal may also modify brain function at a neurophysiological level. Two regions of the brain, the cerebellum and the hippocampus, undergo marked development in the first 3 or 4 weeks after birth; and researchers have established that zinc accumulates in the hippocampus during this time.

When Dr Dreosti and his co-workers at the Division carried out studies on young zinc-deprived rats, they found that late prenatal or early postnatal zinc depletion does not seriously affect cerebellar and hippocampal DNA synthesis. The activities of two enzymes associated with neural function, however, are affected. These enzymes may be involved in the transmission of nerve impulses and the function of the hippocampus, an area that is intrinsic to the expression of emotion and the integration of memory. Changes in their activity may contribute to the observed behavioural changes.

Few studies or observations have been made on the effect of zinc status on human brain development and function. However, some evidence of the effects of severe zinc deficiency in humans has come from studies of pregnant women with the disease acrodermatitis enteropathica. This rare disease causes defective absorption of zinc from the intestine. Acrodermatitis babies exhibit the disease on withdrawal of human milk, which appears to contain a zinc-binding compound similar to that responsible for zinc absorption in the intestine.

Very few females with acrodermatitis have produced offspring, but among the pregnancies reported, one terminated in spontaneous abortion and a further two suffered from major birth defects, including anencephaly.

Trace element interactions and alcohol in adult and fetal rats. I.E. Dreosti. *Proceedings of CIBA Symposium on In Utero Alcohol Damage, London, September 1983* (in press).

Fetal and maternal serum copper and zinc levels in human pregnancy. I.E. Dreosti, A.J. McMichael, G.T. Gibson, R.A. Buckley, J.M. Hartshorne, and D.P. Colley. *Nutrition Research*, 1982, **2**, 591-602.

Zinc and brain development. I.E. Dreosti. In 'Fetal Brain Disorders — Recent Approaches to the Problem of Mental Deficiency', ed. B.S. Hetzel and R.M. Smith. (Elsevier-North Holland Biochemical Press: Amsterdam 1981.)

Most available evidence suggests that genetic differences are probably less important than environmental factors in determining rates of growth of children. A group led by Dr Jean-Pierre Habicht of the Institute of Nutrition in Guatemala City, South America, compared weights and heights of individuals from birth to 7 years of age for a range of racial and ethnic groups living under very different conditions. They found that racial differences caused only minor variations in growth rates, whereas environmental effects such as nutrition and disease were far more significant.

The likelihood that a zinc-deficient diet may be the largest contributing cause of retarded growth of Aboriginal children in the north-west is reinforced by comparable data from other countries. Similar hair zinc levels were found in malnourished Brazilian children, and nutritionists in Denver, U.S.A., noticed a marked growth response to supplementary zinc in severely malnourished children with low plasma zinc levels.

In 1983, Dr Smith and Dr Spargo began intervention trials using zinc supplements. The trials are double-blind; that is, both zinc supplements and placebos are administered, with neither the recipient nor the

Growth and zinc status		
	Aboriginal	non-Aboriginal
age (years)	9.2	9.6
body weight (kg)	24.6	32.1
height (cm)	128.1	134.4
arm circumference (cm)	17.5	20.9
plasma zinc (µg/mL)	0.7	0.8
hair zinc (µg/mL)	105.6	119.7

Dr Smith found that the growth of Aboriginal children in the Kimberleys fell below normal patterns shown by a control group of non-Aboriginal schoolchildren in Adelaide. The figures are averages for the two groups.

communities ill-health appears to be the norm and is an inevitable product of the way in which Aborigines now live.

Some 200 years ago, Aboriginal men hunted large game such as kangaroo, bush turkey, and emu, while women and children gathered grubs, fruit, seeds, various roots, and green leaves, and caught goanna and

beliefs are still strong in remote places like the Kimberleys and often the people consult the *ngangkere* before considering a visit to a doctor or clinic. In many cases, they remain mystified by, and often frightened of, Western health institutions.

In many Aboriginal families, the major source of income is not wages or salaries but pensions and child endowment, and sometimes unemployment benefits. The ensuing feelings of dependency and powerlessness often lead to stress and its associated ill-health problems. Further, the pension cheque schedule dictates the pattern of life. Food must be cheap and able to be stored without refrigeration or else must be rapidly consumed. Unfortunately, tinned goods, processed cereals, and soft drinks readily fit this bill.

For Aborigines in northern Australia, settlement living with its sedentary life style and poor diet produces a wide range of life-style diseases and conditions. As well as zinc-deficiency symptoms, these people suffer widespread chronic diabetes, hypertension, and obesity, and secondary associated conditions include alcohol and drug abuse.

Results of studies on nutritional deficiencies can spell out the immediate resources necessary to treat the diseases that characterize too many Aboriginal communities. But, as Dr Smith and Professor Cheek emphasize, the present life style is what causes and perpetuates disease there. Clearly, medical technology can only be a small part of the answer.

Mary Lou Considine

More about the topic

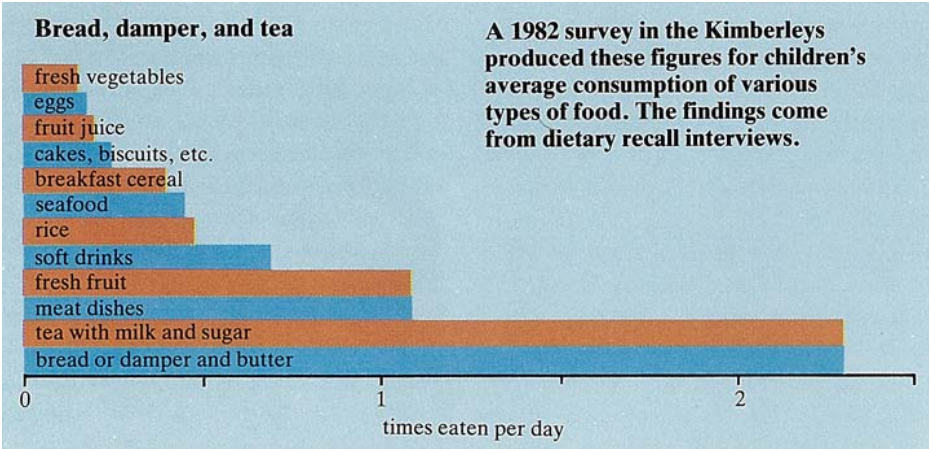
Zinc status and growth of Aboriginal children in the north-west of Australia. R.M. Smith, R.M. Spargo, and D.B. Cheek. *Proceedings of the Nutrition Society of Australia*, 1982, 7, 37-44.

Hair and plasma zinc levels in Aboriginal Australians. D.B. Cheek, R.M. Smith, and R.M. Spargo. In 'Clinical Applications of Recent Advances in Zinc Metabolism', ed. A. Prasad, B.S. Hetzel, and I.E. Dreosti. (Alan R. Liss Inc: New York 1982.)

Evidence for zinc deficiency in Aboriginal settlements in north-western Australia. D.B. Cheek, R.M. Spargo, and A.B. Holt. *Medical Journal of Australia (special supplement)*, 1981, 1, 4-5.

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donor knowing which is which. This strategy helps eliminate experimental bias and will resolve the question of whether zinc deficiency is or is not responsible for the depressed growth rate.

A problem within a problem

Health experts are becoming more aware that short-term programs, dealing with temporary solutions to an immediate problem such as zinc deficiency, do not provide durable answers to the fundamental life-style inadequacies of which the deficiency itself is often only a symptom.

The World Health Organization counsels that health-care planning should revolve around good health as a norm rather than focus on ill-health. Yet in many Aboriginal

smaller game. Their diet was probably high in protein, roughage, and vitamins, and low in fats and carbohydrates.

In this century, health authorities attempted to implement among Aborigines a medical system developed in cities of the Western industrialized world. But the people did not share the culture, the disease profile, or the aspirations of white Australians. For Aborigines the 'old ways' were pre-eminent, and so they saw serious sickness as being caused by bad spirits, to punish those who infringed dietary or social taboos.

Only the tribal healer or *ngangkere* knew the sacred law and could see the cause of an illness. Bad spirits were coaxed out of a body or treated with herbal cures. These