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Review Article

Childhood infections in the tropical north of Australia

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Abstract: In the tropical north of Australia there are high rates of infections in Aboriginal children living in remote communities. In addition to the burden of respiratory infections, diarrhoeal disease and skin sepsis, there are high rates of acute rheumatic fever, outbreaks of poststreptococcal glomerulonephritis and gonococcal conjunctivitis, endemic trachoma and various intestinal parasites. A number of infections generally restricted to the tropics are also present and can cause disease in both indigenous and non-indigenous children. These include melioidosis, Murray Valley encephalitis and dengue on the east coast. With global warming, these infections may become more common and more widespread within Australia and the potential for establishment of introduced infections such as Japanese encephalitis and malaria may increase.

Key words: Aborigines; global warming; melioidosis; Murray Valley encephalitis virus; rheumatic fever; scabies.

Tropical Australia lies north of the Tropic of Capricorn (latitude 23.5°S). It covers 40% of the continent, with a population well below one million. There is a great diversity of climates and terrains, including coastal monsoonal tropical rainforests, mangrove and sand dune coastal habitats, eucalypt woodlands and open savanna and arid desert in the interior. For those working in the region there are two important health issues in addition to those encountered elsewhere in Australia – Aboriginal health and specific diseases of the tropics.

INFECTIOUS DISEASES IN ABORIGINAL CHILDREN

There is continuing disparity in morbidity and mortality between Aboriginal people and other Australians. While differential mortality rates between Aboriginal and non-Aboriginal people are still highest for various infectious diseases, non-communicable diseases such as circulatory and chronic respiratory illnesses now cause more deaths in Aboriginal adults.[1] The high levels of chronic diseases in Aboriginal adults are being increasingly attributed in part to antecedents *in utero* and in early childhood, including recurrent skin and respiratory infections.[2,3] Although the current burden of infectious diseases in Aboriginal communities can be ascribed primarily to socioeconomic disadvantage, addressing this situation raises complex issues including those of self-determination and community control.[4] Living conditions in many remote Aboriginal communities remain harsh, with continuing overcrowding and difficulties with water supply and sanitation.[5,6] The strong links between poor health and low educational attainment have increased the focus on initiatives to address the barriers to formal education for Aboriginal children, especially in remote communities.[7]

Table 1 shows the range of infectious diseases seen in children in Aboriginal communities in central and northern Australia,[2,8] including some currently or ever rarely seen in non-indigenous Australians such as rheumatic fever, trachoma,[9] various intestinal parasites, poststreptococcal glomerulonephritis,[2,10] and gonococcal conjunctivitis.[11]

Table 2 shows the range of public health programs and interventions addressing specific infectious diseases in Aboriginal communities.

Respiratory infections

In many of the remote Aboriginal communities in the Northern Territory, most children are colonized in the nasopharynx soon after birth by multiple strains of *Streptococcus pneumoniae* and non-typeable *Haemophilus influenzae* (*H. influenzae*).[12] Chronic middle ear effusions are common and up to half of all children in some communities have had at least one perforated eardrum by their first birthday. The rates of invasive pneumococcal disease reported in Aboriginal children from central Australia are amongst the highest documented in the world, with an attack rate in those aged under 2 years of 2053 per 100 000 persons per year.[13] Rates of invasive disease from *H. influenzae* serotype b (Hib) were also very high in the Northern Territory until its virtual elimination by vaccination.[14] The introduction of the first licensed conjugate pneumococcal vaccine for infants occurred in June 2001. It is anticipated to have a major impact on invasive disease, with 58% of invasive isolates in indigenous children under 2 years of age in the

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Table 1 Infections in children in Aboriginal communities in central and northern Australia

Respiratory infections:
Otitis media – especially chronic suppurative otitis media
Bacterial pneumonia
Tuberculosis – especially nodal disease*
Influenza – may be epidemic and 'unseasonal'
Infectious diarrhoea:
Escherichia coli
Salmonella and shigella
Rotavirus
Campylobacter
Cryptosporidium
Giardia
Intestinal nematodes:
Hookworm* – *Ancylostoma duodenale,* causes iron deficiency anaemia, retards growth
Whipworm* – *Trichuris trichiura,* diarrhoea and can cause rectal prolapse
Strongyloides* – *Strongyloides stercoralis,* can cause swollen belly syndrome in young children and occasionally disseminated infection
Skin infections:
Impetigo (skin sores) – usually *Streptococcus pyogenes*
Boils – usually *Staphylococcus aureus*
Scabies*
Tinea corporis – usually *Trichophyton rubrum,* often with nail involvement
Bone, joint and muscle infections:
Septic arthritis, often *Staphylococcus aureus*
Osteomyelitis, usually *Staphylococcus aureus*
Pyomyositis*, usually *Staphylococcus aureus*
Others:
Bacterial meningitis
Acute rheumatic fever*, including rheumatic chorea
Poststreptococcal glomerulonephritis – almost always from impetigo, can be epidemic
Bacterial conjunctivitis – occasionally epidemic gonococcal conjunctivitis*
Trachoma* – *Chlamydia trachomatis*
Hepatitis B – only older children – universal neonatal vaccination from 1988
Hepatitis A – almost universal in childhood and usually asymptomatic – vaccination program commenced in north Queensland, not in NT as yet

*Absent or extremely uncommon in non-Aboriginal children.

Table 2 Public Health Programs for coordinating control of specific infections in children in Aboriginal communities

Ongoing programs:
Immunization – including pneumococcal vaccination and bacille Calmette-Guerin vaccine (BCG)
Routine deworming – often linked to school screening
Rheumatic fever – secondary prophylaxis program
Tuberculosis – curative treatment, contact tracing, community screening
Protocols for responses to specific outbreaks or cases:
Community-based scabies program
Community-based trachoma program
Meningococcal meningitis response protocol
Gonococcal conjunctivitis response protocol
Poststreptococcal glomerulonephritis response protocol

Northern Territory covered by the seven-valent vaccine.[15] However, the magnitude of any impact of pneumococcal vaccination on ear disease remains to be determined and concerns about serotype replacement following vaccine selection pressure will require careful longitudinal evaluation.[16] Recently, the recommendations for the 23-valent polysaccharide pneumococcal vaccine have been expanded in the Northern Territory to include all Aboriginals aged 15 years and over plus those aged 2 to 5 years in central Australia.[15]

Rheumatic fever, streptococcal infections and scabies

In the Aboriginal communities of northern and central Australia, the incidence of acute rheumatic fever and prevalence of established rheumatic heart disease are also amongst the highest documented in the world. In the Top End of the Northern Territory, the annual incidence of confirmed acute rheumatic fever between 1987 and 1996 was 224 per 100 000 Aboriginal people aged 5–14 years, with a more accurate estimate of 508 per 100 000 for 12 selected communities with good ascertainment.[17] The point prevalence of established rheumatic heart disease amongst all ages in Aboriginal people was 11.8 per 1000, with a rate of 22.4 per 1000 in the 12 selected communities. Rheumatic fever and rheumatic heart disease are also common in Aboriginal communities in the Kimberley and north Queensland.[18–20]

It has been considered that rheumatic fever only follows *Streptococcus pyogenes* (*S. pyogenes*) infection of the throat.

The paradox in Aboriginal communities is that throat carriage rates of *S. pyogenes* are usually very low (< 5%) and symptomatic pharyngitis is uncommon.[21] However, streptococcal pyoderma is endemic, with up to 70% of children in some communities being affected at any time.[22] Much of the streptococcal pyoderma is secondary to the current overlapping epidemic cycles of scabies, with the prevalence of scabies in children in some regions up to 50%.[22]

Intestinal parasites

Intestinal parasites remain common in many Aboriginal communities in the region, especially in the Kimberley and the Top End of the Northern Territory.[23–29] Routine community treatment of children with pyrantel, and more recently albendazole, has led to dramatic reductions in hookworm. *Strongyloides stercoralis* and *Trichuris trichiura* are now the most common stool parasites isolated from children at Royal Darwin Hospital, each found in about 3.5% of stool examinations. Despite being common in neighbouring Papua New Guinea and Timor, *Ascaris lumbricoides* is not found in Aboriginal communities in the Kimberley and the Northern Territory.

Diarrhoeal disease

Diarrhoeal disease is common in Aboriginal communities and reflects overcrowding and difficulties with sanitation and hygiene. Aboriginal children in the Top End of Australia have higher hospitalization rates and more severe manifestations of diarrhoeal disease (e.g. hypokalaemia, acidosis and lactose intolerance) than non-Aboriginal children.[30,31] For example, acidosis (bicarbonate < 18 mmol/L) was documented in 58% and hypokalaemia (< 3.5 mmol/L) in 69% of recent paediatric diarrhoeal admissions to Royal Darwin Hospital. These complications have been shown to be related to the severity of underlying small bowel damage as measured by the lactulose/rhamnose (L/R) test of intestinal permeability.[31] In Aboriginal children with diarrhoeal disease, the degree of small bowel mucosal damage correlated highly with the presence of acidosis, hypokalaemia and severe dehydration assessed both clinically and by weight change after rapid intravenous rehydration.[31,32] Aboriginal control children without diarrhoea or malnutrition, had over twice the mean L/R ratio of non-Aboriginal control children, indicating the presence of an asymptomatic environmental/tropical enteropathy syndrome which studies from overseas have attributed to contamination of food and water, and to unhygienic living conditions.[33] Small intestinal malabsorption in asymptomatic residents of the developing world is often referred to as the tropical enteropathy syndrome. It has been found to affect 30–50% of the population, including children, in almost all poor tropical areas studied. It is now clear that this syndrome is due to small intestinal mucosal damage from exposure to multiple enteric pathogens in a contaminated environment. This is highly relevant to Aboriginal children in northern Australia since the poor environmental health conditions in remote communities have already been well documented.[6] Although there was controversy initially about the nutritional significance of subclinical malabsorption in children, more recent studies using intestinal permeability ratios have documented that this underlying small bowel damage contributes to over 40% of growth failure in weanlings.[34] This raises the question as to the contribution of abnormal intestinal permeability or subclinical malabsorption in asymptomatic Aboriginal children to the high prevalence of growth retardation and frank malnutrition, which remain major problems for Aboriginal children in remote communities in central and northern Australia.[30,35]

Assessment of the microbiology of diarrhoeal disease in Aboriginal children has been limited by the unavailability of *Escherichia coli* (*E. coli*) probes for testing stool cultures in regional laboratories. Studies from the Kimberley have identified various *E. coli,* including enterotoxigenic, verotoxigenic, enteroaggregative and cell-detaching *E. coli.*[36–39] However, while sometimes associated with diarrhoea, many of these *E. coli* were also found in stools from asymptomatic Aboriginal children.[36,39] Other stool pathogens commonly found have been a wide range of Salmonella serovars, Shigella, Campylobacter, Cryptosporidium and rotavirus, again associated with both symptomatic and asymptomatic infection.[36] The very high rates of salmonellosis and shigellosis in remote Aboriginal communities are directly attributable to the difficulties with hygiene and water supply, which in turn reflect socioeconomic disadvantage. In Darwin, the most frequently isolated stool pathogens in hospitalized Aboriginal children are: enteroaggregative *E. coli*, rotavirus and enteropathogenic *E. coli,* followed by *Salmonella* spp., enterotoxigenic *E. coli, Cryptosporidium parvum* and *Strongyloides stercoralis* (R Kukuruzovic, D Brewster, 2000, unpub. data.). However multiple pathogens were frequently isolated in these children and even controls without diarrhoea had organisms isolated (particularly *Salmonella* spp., *Giardia duodenalis* and enteroaggregative *E. coli*). The greatest improvements in mean L/R permeability ratios from days 1–5 in hospital occurred for enteroaggregative *E. coli*, rotavirus, enteropathogenic *E. coli* and *Strongyloides stercoralis*, whereas *Cryptosporidium parvum* was the organism consistently associated with the most severe and prolonged mucosal damage, a finding also in other studies.[34]

SPECIFIC DISEASES OF THE TROPICS

Malaria and dengue

The last *indigenous* case (unrelated to an *imported* case) of malaria infection in Australia was in 1962 in the Northern Territory.[40] Occasional local cycles of transmission (involving local mosquitoes infected from an imported case), called *introduced* malaria, have occurred in north Queensland, especially in the Torres Strait Islands where there are close geographical and cultural links to Papua New Guinea.

Dengue and its main vector mosquito, *Aedes aegypti* (*A. aegypti*), have been introduced to north Australia periodically since the 1880s but public health measures resulted in the eradication of dengue in 1955. However a resurgence of *A. aegypti* and re-introduction of dengue occurred in north Queensland in the 1980s[41] and there have subsequently been a series of outbreaks in the region.[42] *A. aegypti* and hence dengue remain eradicated from the Northern Territory and Western Australia, although surveillance is essential because occasional imported mosquitoes or larvae are detected and increasing numbers of travellers are entering Australia with dengue acquired from endemic countries to the north of the country. Dengue haemorrhagic fever can be a fulminant illness, especially in children.

Arboviruses

The Murray Valley encephalitis virus (MVE) is endemic in the Kimberley region of northern Western Australia and the adjacent Northern Territory. Since the 1974 epidemic, which spread to southern states and especially the Murray and Darling watersheds, all confirmed cases were in tropical northern Australia until a cluster in central Australia following exceptionally heavy rains in early 2000.[43] Serological studies show considerable rates of exposure in the endemic region,[44] consistent with a low clinical attack rate (estimated to be 1 in 1000 of those infected), despite the often devastating nature of the encephalitis in those few who develop clinical illness. Cases in children since 1974 have been mostly in Aboriginal children from remote communities in the Kimberley and the Top End of the Northern Territory,[44] presumably reflecting increased exposure and possible higher viral infecting dose. In clinical cases, fever and seizures are common, together with tremor, cerebellar, spinal cord and brainstem signs (pseudopoliomyelitis).[44] Mortality in cases is 20% and residual neurological deficit occurs in up to a further 40%.

Occasional cases of 'Australian encephalitis' are caused by Kunjin virus rather than MVE. Another closely related flavivirus with clinical features in children similar to MVE is Japanese encephalitis virus. This virus is not endemic to Australia, but was introduced to the Torres Strait in 1995,[45] and a case occurred in 1998 on the Australian mainland.[46] To date there has been no indication of spread on the mainland, although the large number of feral pigs across northern Australia are potential amplifying hosts, making this virus a major threat to the region.

While more common in the tropical north of Australia, Ross River and Barmah Forest viruses cause outbreaks throughout Australia. Infection in children is usually asymptomatic, and it is likely that infection in childhood accounts for the very low incidence of clinical disease in adults in Aboriginal communities in northern Australia despite high rates of seropositivity.[47,48]

Melioidosis

Melioidosis, infection with the soil and water bacterium *Burkholderia pseudomallei*, is an important cause of sepsis across tropical Australia. However, disease in children is uncommon, with only 12 paediatric cases and one death out of 289 culture-confirmed cases in the 11-year prospective study in the Top End of the Northern Territory.[49,50] While pneumonia is the commonest presentation of melioidosis, spread to any organ can occur, with a spectrum from mild cutaneous lesions such as ulcers or boils to fulminant septic shock with multiple abscesses in internal organs. An uncommon but important presentation to recognize is neurological melioidosis, with various combinations of brainstem encephalitis (usually with facial palsy), peripheral motor weakness and acute flaccid paraparesis.[51] The differential diagnosis includes MVE, Japanese encephalitis and tuberculous meningitis.

A number of other infections endemic in northern Australia have not been clinical problems for children to date in the Top End. These include scrub typhus, now recognized to be endemic in Litchfield Park,[52] and leptospirosis.[53]

Global warming

Global warming may well impact on infectious diseases in children in the region. It is predicted that northern Australia will become warmer and wetter. This would extend the receptive area for malaria further south, mainly by expanding the range of the major mosquito vector *Anopheles farauti.* Transmission of dengue, MVE and Japanese encephalitis viruses would be facilitated and the endemic region for melioidosis would expand southward.[48]

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