

# Epidemiology of group A streptococcus in Australia



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**The Australian population experiences the extremes of group A streptococcal (GAS) disease. The majority of our population lives an affluent, middle class existence and experiences the same types and rates of GAS disease as are found in most industrialised countries. By contrast, the Aboriginal and Torres Strait Islander populations, particularly those living in remote and rural settings, experience a developing-country profile of GAS disease, which is almost entirely attributable to poverty,**

**particularly as it is manifest by overcrowded housing, poor environmental health and reduced access to health care.**

The group A streptococcus causes the broadest range of diseases of any bacterium (Figure 1). Infection with this organism can lead to asymptomatic carriage, superficial infection of the upper respiratory tract (pharyngitis) and skin (impetigo or pyoderma), or invasive disease (bacteraemia or focal infection such as osteomyelitis, pneumonia, meningitis and so on). But GAS also has the potential to release exotoxins, resulting in scarlet fever or streptococcal toxic shock syndrome. And to top it all off, this is one of the few organisms that unequivocally causes autoimmune disease (acute rheumatic fever, ARF and acute post-streptococcal glomerulonephritis, APSGN). These last two manifestations can in turn lead to chronic sequelae; rheumatic heart disease (RHD), may follow ARF<sup>1</sup> and chronic renal failure is linked to APSGN<sup>2</sup>.

Globally, GAS is responsible for at least 517,000 deaths per year (Table 1)<sup>3</sup>. There are more than 18 million people living with a severe sequel of GAS infection, with at least 1.8 million new

Table 1. Minimum summary estimates of the global burden of group A streptococcal diseases as of 2002.

Disease	Number of existing cases	Number of new cases each year	Number of deaths each year
Rheumatic heart disease	15.6 million	282,000*	233,000†
History of acute rheumatic fever without carditis, requiring secondary prophylaxis	1.88 million	188,000*	
RHD-related infective endocarditis		34,000	8,000
RHD-related stroke	642,000	144,000	108,000
Acute post-streptococcal glomerulonephritis	§	472,000	5,000
Invasive group A streptococcal diseases		663,000	163,000
<b>Total severe cases</b>	<b>18.1 million</b>	<b>1.78 million</b>	<b>517,000</b>
Pyoderma	111 million		
Pharyngitis		616 million	

All estimates rounded off. Note that these estimates assume constancy of incidence and prevalence over time.

\* New RHD cases were calculated based on the proportion of incident ARF cases expected to develop RHD. The remainder of incident ARF cases are included in the 'History of ARF without carditis' row. Therefore, the total number of new ARF cases each year is 282,000+188,000=470,000.

† Includes ARF deaths. RHD deaths are based on the proportion of existing RHD cases expected to die each year.

§ No attempt has been made to quantify the prevalence of APSGN-induced chronic renal impairment or end-stage renal failure

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cases each year. Almost all of these cases and deaths occur in settings of poverty, mainly in developing countries. As more and better quality data are collected from developing countries, these estimates will almost certainly rise<sup>4</sup>. Based on estimates from the World Health Organization, GAS causes a similar number of deaths each year as rotavirus, measles, *Haemophilus influenzae* type B and hepatitis B and is only exceeded as a single-organism cause of death by HIV, *Plasmodium falciparum* and *Streptococcus pneumoniae*<sup>3</sup>. Given that GAS causes more long-term morbidity than most of these pathogens, it is clear that its importance to global health has been underestimated.

### Streptococcal epidemiology in the non-Indigenous population

The few population-based studies that have been conducted suggest that GAS infections affect the majority of the Australian population much as they affect populations in industrialised countries around the world. Statewide surveillance of invasive

GAS infections in Victoria over 2.5 years from 2002 to 2004 confirmed an annual incidence of 2.7 cases per 100,000 people, peak incidences in the elderly and young children, case fatality of 7.8%, streptococcal toxic shock in 14% (with a case fatality rate of 23%) and that five *emm* types of GAS accounted for 54% of all cases, with *emm* type 1 the most common *emm* type<sup>5</sup>. These data support an earlier hospital-based study from Melbourne, suggesting that the incidence and virulence of invasive GAS infections increased in Victoria during the 1980s and 1990s, as had been seen in North America and Europe<sup>6</sup>. The Victorian surveillance produced an estimate of all-age incidence remarkably similar to the pooled estimate (2.45 cases per 100,000) from 16 industrialised countries in a systematic review of the global burden of disease, although the case fatality rate was somewhat lower than the global pooled estimate (15%)<sup>3</sup>.

A 16-month prospective, population-based cohort study in Melbourne during 2001-2 found an incidence of culture-positive GAS pharyngitis in 5-12 year old children of 13% per year, while

Figure 1. The broad spectrum of GAS diseases

- **Superficial infection**
  - Pharyngitis
  - Impetigo
- **Invasive diseases**
  - Septicaemia
  - Pneumonia, osteomyelitis...
  - Necrotising fasciitis
- **Toxin mediated diseases**
  - Scarlet fever
  - Streptococcal toxic shock syndrome
- **Post-streptococcal autoimmune sequelae**
  - Acute rheumatic fever/rheumatic heart disease
  - Post-streptococcal glomerulonephritis



A. Typical GAS pharyngitis. B. Aboriginal child with multiple impetigo lesions. C. Necrotising fasciitis (photo courtesy Alfred Hospital). D. Chest X-ray of a 15-year-old Aboriginal boy with severe mitral and aortic regurgitation due to RHD. The child died the next day. E. Aboriginal man with traditional scarification and an iatrogenic scar from a mitral valve replacement for RHD (Photo courtesy B Currie).

serologically-confirmed cases occurred at an incidence of 8% per year<sup>7</sup>. This study also found a high burden on families due to GAS pharyngitis: 20% of families experienced at least one GAS culture positive sore throat case per year and of families with a case of GAS pharyngitis, at least one secondary case occurred in 43% (in more than half, at least two secondary cases occurred).

Until recently, little was known of the epidemiology of rheumatic fever outside the Northern Territory (NT), north Queensland or the Kimberley. Since October 2007, the Australian Paediatric Surveillance Unit (APSU) has been conducting voluntary surveillance among Australian paediatricians for cases of ARF. In the first 20 months of surveillance, 69 confirmed cases were notified, of which 84% were Indigenous<sup>8</sup>. Of the eight Caucasian children notified, all presented with their first episode of ARF and there was a tendency to quite severe cardiac lesions (four had mild-moderate valve lesions and one had severe valvular disease). A separate audit of admissions with ARF to Westmead Children's Hospital in Sydney from 2000-2008 suggests that the APSU surveillance may be underascertaining cases in urban centres<sup>9</sup>. That audit found 29 cases (an average of 3.5 per year) with confirmed ARF presenting to just one hospital in Sydney. Eighteen per cent were Aboriginal or Torres Strait Islander, 36% were Pacific Islanders and 64% came from regions having the two lowest quintiles in the Index of Relative Socioeconomic Disadvantage and Advantage.

### Group A streptococcal diseases in Aboriginal and Torres Strait Islander people

If non-Indigenous Australians demonstrate a typical industrialised-country pattern and burden of GAS diseases, Aboriginal and Torres Strait Islander Australians experience the other extreme. With the exception of pharyngitis, every GAS disease described to date in Aboriginal and Torres Strait Islander people living in rural or remote Australia has occurred at among the highest rates reported anywhere in the world. Unfortunately, there are no reliable population-based data relating to GAS disease epidemiology in Aboriginal and Torres Strait Islander people living in urban settings.

### Rheumatic fever and RHD

For two decades, reports have continued to emerge from northern and central Australia that confirm that Aboriginal people have the highest reported incidences in the world of ARF and among the highest reported prevalences in the world of RHD. Two small studies during the 1980s found extremely high incidence rates of ARF in school-aged children: 815 per 100,000 in a single central Australian community<sup>10</sup> and 375 per 100,000 in a single hospital study in Western Australia<sup>11</sup>. The central Australian study also found a prevalence of RHD of 12.3 per 1,000<sup>10</sup>. A report in 1978 stated that the prevalence of rheumatic

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valvular disease in Torres Strait Islanders was no different to that found in non-Indigenous people; this interpretation is likely to have been incorrect, as the data showed that the prevalence was at least 4 per 1,000 and may have been as high as 11 per 1,000<sup>12</sup>.

More recent surveillance in the Northern Territory has provided more accurate estimates of ARF and RHD rates. The incidence in Top End Aboriginal children aged 5-14 years over 10 years to 1996 was 224 per 100,000 per year, but in 12 Aboriginal communities with more complete ascertainment, the incidence was 508 per 100,000<sup>13</sup>. The same study reported a point prevalence of RHD among all-age Aboriginal people in the Top End during 1997 of 11.8 per 1,000 but in the 12 communities it was 22.4 per 1,000. Data from the NT RHD registers, first established in the Top End in 1997 and in Central Australia in 2001, confirm that ARF incidence has remained relatively constant (ranging between 186 and 375 per 100,000 per year in Top End Aboriginal school-aged children between 2002 and 2007) and that the true all-age prevalence of RHD is more than 2% (increasing from 12.5 per 1,000 in 1998 to 23.4 per 1,000 in 2006, due to improved ascertainment of cases as a result of the control program)<sup>14, 15</sup> [Unpublished data, NT RHD Control Program].

Until recently, similar quality data were not available from other states in Australia. In Queensland, reviews of cardiac surgery lists and paediatric outreach clinics found that RHD was the overwhelming factor leading to valvular surgery between 2001 and 2006 and that RHD prevalence was among the highest in Australia based solely on children seen by paediatricians<sup>16, 17</sup>. A study of ARF incidence in North Queensland between 1999 and 2004 found incidences in 5-14 year old Indigenous children comparable to those in the NT: overall the incidence was 162 per 100,000 with peak rates in the Torres Strait (349 per 100,000) and Cape York (230 per 100,000)<sup>18</sup>. There are no recent data from Western Australia, but reports from clinicians in the Kimberley and Pilbara support the contention that RHD is a major issue for all Aboriginal people living in rural and remote northern and central Australia.

## APSGN

This acute illness may follow a GAS upper respiratory or skin infection, although in rural and remote Aboriginal people APSGN almost always follows an episode of impetigo. Unlike ARF, which has never been described in epidemics in developing countries or other populations with very high rates of GAS disease (such as Australian Aboriginal people) APSGN occurs both sporadically and in intermittent epidemics in northern Australian Aboriginal communities<sup>19-21</sup>. These epidemics occur in association with the epidemic occurrence of particular *emm* types of GAS (particularly *emm* 55) in impetigo lesions. Traditionally, epidemic APSGN has been considered relatively benign, but in the Aboriginal

population of the NT, childhood APSGN is associated with a five- to sixfold increased risk of later chronic renal disease<sup>2</sup>.

Until studies were done in the Australian Aboriginal population, the highest incidences in the world of APSGN were found in NZ Maori (50.5 per 100,000) and Pacific Islander (46.5 per 100,000) children during the 1980s<sup>22</sup>, with rates between 18 and 24 per 100,000 in Nigeria, Kuwait, Chile and the French Caribbean, also in the 1980s<sup>23-27</sup>.

In the Top End of the NT between 1993 and 1995, based on hospitalisation data only, the incidence of APSGN in Aboriginal children aged <15 years was 239 per 100,000 whereas it was only 6 per 100,000 in non-Aboriginal children in the same region<sup>28</sup>. A recent review of the NT Notifiable Diseases Database between 1991 and 2008 found 415 notified cases of APSGN in Aboriginal people, with an incidence in Aboriginal children <15 years of 94.3 per 100,000 (95% CI 84.2-105.3) and an Aboriginal: non-Aboriginal rate ratio of 53.6 (95% CI 32.6-94.8)<sup>29</sup>. Interestingly, during this period, large epidemics occurred every 5 years in the Top End, perhaps suggesting that population immunity to virulent serotypes may not be long-lasting.

## Invasive GAS diseases

Australian Indigenous people living in remote and rural settings experience dramatically higher incidence rates and mortality due to invasive GAS infections than other Australians. Retrospective studies in the Top End (1991-6) and north Queensland (1996-2001) found all-age incidences of 32.2 (Top End) and 82.5 (Qld) per 100,000 in Indigenous people compared to 6.4 and 10.2, respectively, in non-Indigenous people living in the same regions<sup>30, 31</sup>. Mortality was 13% in the Top End study and 7% in the Queensland study. These rates in Indigenous people are the highest described in any population. The Top End study found a clear association with skin infection. It also found a broad diversity of *emm* types causing invasive disease, which was recently also found in Fiji<sup>32</sup>, whereas in affluent settings most invasive infections are due to a relatively limited number of *emm* types. Globally, there are estimated to be more than 660,000 cases of invasive GAS disease each year, with more than 160,000 resultant deaths<sup>3</sup>: it is increasingly clear that in developing countries and in Aboriginal Australians, invasive GAS diseases have been underestimated previously as important causes of morbidity and mortality.

## Impetigo

There have been no population-based studies of the incidence or prevalence of impetigo in non-Indigenous children in Australia, but we know that it continues to occur from time to time, including as occasional outbreaks of 'school-sores' and that the aetiology is increasingly due to *Staphylococcus aureus*



rather than GAS<sup>33</sup>. However, in Indigenous communities in rural and remote regions, impetigo remains the most common manifestation of GAS disease and affects more than half of the children at any one time in many communities<sup>34, 35</sup>. Children are often affected by multiple impetigo lesions. As with middle-class Australia, *S. aureus* is increasingly isolated from impetigo lesions, often together with GAS. The relative contribution of each organism to the pathogenesis of many sores is unknown, although community-associated MRSA is also a major pathogen associated with impetigo in northern Australian communities<sup>36</sup>. Impetigo is the major factor underlying post-streptococcal glomerulonephritis and invasive GAS disease in Aboriginal communities and is also postulated to have a role in pathogenesis of rheumatic fever in this population<sup>37</sup>. The treatment of impetigo traditionally has been with penicillin, which targets the GAS but has no activity against most *S. aureus*. It is increasingly clear that the effectiveness of penicillin in impetigo in Aboriginal communities needs to be evaluated; our group at the Menzies School of Health Research is currently doing this, as part of a randomised trial comparing intramuscular benzathine penicillin G with various regimens of oral co-trimoxazole.

### Pharyngitis

During the 1990s, our group in the Northern Territory noted that Aboriginal children in the Top End had a low throat carriage rate of GAS and, anecdotally, infrequently presented with symptomatic GAS pharyngitis<sup>34</sup>. Yet the same population experienced the highest documented incidence in the world of ARF<sup>13</sup>. These data were difficult to reconcile with the traditional dogma that ARF could only follow an episode of GAS pharyngitis<sup>38</sup>. Our further work has confirmed that the incidence of sore throat is low in Aboriginal children in the Top End (incidence density 8 per 100 person-years, 95% CI 4–15) and that symptomatic GAS pharyngitis is rarely seen (incidence density zero in our study of 4,842 person-visits)<sup>39</sup>. Interestingly, repeated surveillance in this same region confirms that group C and G streptococci are isolated from throats of Aboriginal children much more commonly than group A<sup>34, 39</sup>. The putative roles of these other beta-haemolytic streptococci in causing more severe disease, such as ARF, has yet to be clarified.

### Conclusions

The 'gap' between the health of Indigenous and non-Indigenous Australians is perfectly mirrored in the epidemiology of GAS diseases, which are in many ways a barometer of socio-economic conditions. On the one hand, the majority of the Australian population lives an affluent, comfortable lifestyle and experiences frequent but usually mild episodes of GAS pharyngitis in childhood and rare but often severe episodes of invasive GAS diseases throughout life, but particularly at the

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extremes of age. On the other hand, Aboriginal and Torres Strait Islander people living in rural and remote settings, characterised by poverty, overcrowded housing and poor access to medical care, experience extraordinarily high rates of impetigo and its severe sequelae, particularly invasive GAS disease, APSGN and ARF/RHD. These latter diseases cause substantial morbidity and mortality and add further to the arguments that immediate action is needed to provide high quality medical and preventive care, but also to address the underlying determinants of these diseases of poverty.

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