EDITORIAL

Dengue in Australia

The history of dengue in Australia extends over more than 120 years. The earliest reference described the importation of eight cases by ship from Mauritius in 1873; the first indigenous outbreaks probably occurred in Queensland at Townsville in 1879 and Rockhampton in 1885 [1]. Several epidemics were described in the 1890s and the early part of the 20th century. The first cases in northern New South Wales were reported in 1898, but epidemic activity did not extend southwards until 1925–26 when cases were described as far south as Newcastle. Dengue was reported from Western Australia in 1909–10 and was declared notifiable there in 1912. In 1914, dengue was reported from Darwin in the Northern Territory.

The early epidemic activity in Queensland and northern New South Wales led to a number of seminal observations on the epidemiology and pathogenesis of dengue, and on the role of *Aedes aegypti* mosquitoes in the transmission of the disease. Thus Hare [2] described the first cases of dengue haemorrhagic fever; Bancroft [3], building on earlier findings of the Yellow Fever Commission of the United States Army, described the first experimental transmission of dengue by *A. aegypti*; Cleland *et al.* [4–6] confirmed and extended the findings of Bancroft and demonstrated that dengue only occurred in those areas in which the mosquito was prevalent. Bancroft not only carried out transmission studies to implicate *A. aegypti* (unlike earlier observations in Syria which appeared to show that *Culex fatigans* was the vector of dengue [7]), but provided epidemiological support for this contention by his observations on the diurnal habit of *A. aegypti* compared to the nocturnal habit of *C. fatigans*. He also believed that the causative agent of dengue was probably ultra-microscopic.

The only vector of dengue in Australia is *A. aegypti*, which may have been introduced with the first fleet or shortly after [8], or have arrived in the mid-19th century with settlement of the tropics and subtropics [9]. By the end of the 19th century it was quite widely distributed in the north-east coastal areas. Spread then followed the movement of people, through coastal steamers, and along road and rail links. Major breeding sites were provided by rainwater tanks and water-holding domestic containers [10, 11]. The dis-

tribution of *A. aegypti* in Australia has been described by Taylor [12] and O’Gower [13].

Dengue has not occurred in New South Wales since 1925–26, apart from some isolated cases on the far north coast in 1942; no cases have been reported in Western Australia since the 1940s; and the last epidemic activity in the Northern Territory occurred in Darwin in 1955 [11, 14, 15]. The decrease in dengue activity has been due to a decline in the distribution of *A. aegypti*. This species disappeared from New South Wales [11] and Western Australia in the 1950s, and from the Northern Territory in the 1960s [14]. There was also a reduction in its distribution in Queensland in the 1960s and 1970s, although further spread has occurred over the past 15 years [10, 16, 17]. Several factors have influenced the decline of *A. aegypti* throughout Australia. The conversion of urban water supplies from household rainwater tanks to a reticulated supply has certainly been the most important single factor, but the change from steam to diesel locomotives, the use of refrigerators instead of water-cooled ‘Coolgardie’ safes, the use of domestic insecticides, the advent of the motor mower, and greater awareness by local health officers together with public education have all contributed.

Dengue re-appeared in North Queensland in 1981–82 after an absence of >25 years. Whereas the previous epidemic in North Queensland and the Northern Territory in 1955 had been caused by dengue virus type 3 [18, 19], the 1981–82 epidemic was due to dengue virus type 1 [20, 21]. Several hundred serologically confirmed cases were reported from Cairns, Townsville and Thursday Island. Further cases due to dengue type 1 were reported in 1990–91. This was followed in 1992–93 by a large outbreak of dengue virus type 2, centred in Townsville (population 120 000) and in Charters Towers (population 10 000) with 900 serologically confirmed cases and an additional 950 cases inferred on clinical grounds [22, 23] (J. Sheridan and M. Pearce, personal communication). A serosurvey of 1000 randomly recruited residents in Charters Towers suggests that 20% of the population of this small town were infected with Dengue 2 during this outbreak (MacBride *et al.*, personal communication), emphasising that estimates based on serology and clinical grounds underestimated the prevalence of infection. This could be because of asymptomatic infections and of failure to report to

Correspondence should be sent to: Professor A. L. Cunningham, Virology Department, Level 3, ICPMR, Westmead Hospital, NSW 2145, Australia.

doctors for other reasons. One case of dengue haemorrhagic fever (DHF) was reported from Charters Towers [23] during this outbreak.

The available evidence strongly suggests that dengue is not endemic in Australia, and that epidemics arise from virus introduced by viraemic travellers [24]. Imported cases of dengue in travellers are regularly diagnosed throughout Australia and dengue is the most commonly diagnosed flavivirus infection in the country. Over the past 5 years *c.* 30–50 cases/year have been diagnosed serologically and reported through the major state reference virology laboratories. However, the real figure is probably at least twice that number. Dengue is endemic in many of Australia's south-east Asian and Pacific neighbours and has been identified in travellers returning from Thailand and Indonesia, Melanesia including Papua New Guinea, Vanuatu, Noumea, Fiji and India.

Dengue usually presents as a febrile illness with headache, myalgia, arthralgia and a metallic taste in the mouth. A maculo-papular rash which may become petechial occurs in *c.* 50% of patients. The leucocyte count is normal or marginally low and thrombocytopenia is almost invariable. Marginally abnormal liver biochemistry is common. Uncommon presentations of dengue include frank hepatitis or encephalopathy. Its most important life-threatening complications are DHF and dengue shock syndrome, which are heralded by a rising haematocrit and hypotension.

Diagnosis is confirmed serologically, but the interpretation of flavivirus serology is complicated by the extensive cross-reactivity exhibited by this group of viruses. There are *c.* 70 flaviviruses including the four serotypes of dengue (at least 10 are found in Australia). Although these viruses cross-react in serological tests the antibodies produced do not cross-protect. Frequently a patient presents with flavivirus IgG antibody (from a previous infection or a vaccination) but no detectable IgM. In these cases it is essential to obtain second samples and to look for changes in the IgG level or the appearance of IgM, or both, before recent infection can be excluded.

In the southern temperate zones of Australia where the dominant mosquito species are incapable of transmitting dengue, cases mostly provide a test of diagnostic acumen to infectious diseases specialists. However, in northern Queensland where *A. aegypti* is a common peri-domestic mosquito and where there is much travel between Australia and countries in Asia and the Pacific where dengue is endemic, there are major risks of re-establishment of the disease through local transmission of imported virus as occurred in 1992–93. The greatest fear is the possibility of epidemic DHF and shock occurring in Australian children if a strain of dengue other than type 2 becomes prevalent with sequential infection of children who have

previously had type 2. This concern has been aggravated by recent reports of different serotypes of dengue being imported into North Queensland. In addition there was a limited outbreak of dengue – but fortunately again of type 2 [25]. Following the 1992–93 outbreak, public health authorities have intensified surveillance for cases of dengue in Queensland and the Northern Territory and have a rapid response plan for low-level spraying to control mosquitoes in the event of an outbreak being identified or if high numbers of *A. aegypti* are identified near where a patient with dengue has been residing. There are regular community programmes to educate householders in the elimination of casual water as a breeding site for *A. aegypti*.

Although *A. aegypti* is presently confined to areas of Queensland, its wide distribution in Australia in the past necessitates continued vigilance in case it may spread to other states, particularly the Northern Territory. Of equal concern is the possible importation of other exotic dengue vectors such as *A. albopictus*. The rapid spread of this species in the United States following its importation in used car tyres provides a salutary lesson on the need for active surveillance programmes. Indeed, over the past 16 years 11 instances of vector importations, mainly larvae, have been detected in the Northern Territory in vessels arriving at Darwin; of these, six were *A. aegypti*, four were *A. albopictus* and one vessel contained larvae of both species [26].

J. S. MACKENZIE, J. T. LA BROOY, LINDA HUESTON* and
A. L. CUNNINGHAM*

University of Queensland,
North Queensland Clinical School and
*Virology Department, ICPMR,
Westmead Hospital NSW 2145,
Australia.

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