Rising incidence of invasive meningococcal disease caused by *Neisseria meningitidis* serogroup W in Victoria

Invasive meningococcal disease (IMD) caused by *Neisseria meningitidis* is one of the most rapidly progressive sepsis syndromes, often resulting in significant morbidity and mortality. Since the introduction of meningococcal C conjugate vaccine in 2003, IMD in Victoria has decreased from 2.5/100,000 to 0.6/100,000 population.\(^1\)

Epidemiological typing of *N. meningitidis* isolates is by serogroup, multilocus sequence typing and phylotyping. In Victoria, from January 2014 to September 2015, the number of cases of IMD caused by *N. meningitidis* serogroup W (MenW) increased. Previously uncommon (<5% of IMD overall in the period from 2008 to 2013 \(n = 260\)), MenW as a proportion of IMD has increased: four of 33 cases in 2014 and 12 of 41 cases in 2015 (Microbiological Diagnostic Unit, University of Melbourne, unpublished data). Over this period, the median age of cases was 55 years, compared with 19 years for serogroup B, with many non-classical presentations including pneumonia, epiglottitis, septic arthritis and pericarditis. There has been one death in a healthy young adult. No epidemiological links between cases have been observed (Victorian Government Department of Health and Human Services [DHHS], unpublished data).

Globally, MenW has been responsible for an increasing proportion of IMD since outbreaks associated with the Hajj pilgrimage in 2000.\(^2\) Large outbreaks predominately due to MenW strain type P1.5-2: F1-1: ST11 have been reported in South America and the United Kingdom.\(^2,3\) In the UK, MenW cases doubled year on year from <2% of IMD prior to 2009–10 to 25% in 2014–15, prompting a change in vaccination guidelines.\(^4\) Initially, almost 25% of these IMD cases were older adults with non-classical presentations.\(^3,4\)

From 1 January 2014 to 30 September 2015, molecular characterisation, including whole-genome sequencing (WGS), of Victorian MenW strains was undertaken at the Microbiological Diagnostic Unit. Of the 16 MenW isolates, 11 were strain type P1.5-2: F1-1: ST11; two were ST184; one was ST22; one was a new type; and one was polymerase chain reaction-positive only and thus unable to undergo WGS. Comparison of these 11 isolates with international strains using the PubMLST *Neisseria* database (http://pubmlst.org/neisseria) revealed that the Victorian isolates fall within a cluster formed by

Phylogenetic tree for Victorian and international meningococcal isolates


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UK–South American outbreak strains and are distinct from Hajj outbreak strains (Box). Within the UK–South American cluster, nine Victorian isolates appear as an exclusive group of taxa. The close genetic relationship between the nine isolates, long branch length compared with other UK–South American cluster isolates, and lack of identified epidemiological links between cases suggest that these isolates may be representative of a *N. meningitidis* clone arising from a single introduction event that is undergoing widespread endemic transmission in Victoria. The location of the remaining two UK–South American cluster isolates in the tree indicates independent introduction events into Victoria.

While IMD due to MenW in Victoria remains low in absolute case numbers, the rise in incidence is concerning. The Victorian DHHS has instigated enhanced surveillance measures with full molecular characterisation of future isolates to inform ongoing public health responses. National surveillance with enhanced molecular characterisation will improve understanding of the current epidemiology of meningococcus in Australia.

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