

Letter to the Editor

Understanding the role of toxigenic *Corynebacterium diphtheriae* in skin infections in northern Australia

Hannah Gooding and Allison Hempenstall

The current outbreak of toxigenic *Corynebacterium diphtheriae* across northern Australia is the most significant within Australia in the last 100 years. As of 24 August 2026, there have been 538 cases nationally in 2026, with 289 (54%) from the Northern Territory and 226 (42%) from Western Australia.¹ Most confirmed cases have had cutaneous or mild respiratory clinical manifestations, many of which are clinically indistinguishable from other common infections in the region such as *Staphylococcus aureus* or group A streptococcus (GAS). During the current outbreak, in the Northern Territory, *C. diphtheriae* has most frequently been isolated alongside *S. aureus*, beta-haemolytic streptococci, or *Arcanobacterium haemolyticum*.² This pattern is consistent with reports from France,³ Canada,⁴ and New Caledonia.⁵ Three-quarters of toxigenic cutaneous *C. diphtheriae* infections in New Caledonia were co-infected with *S. aureus* or GAS.⁵ Similarly, of cutaneous non-toxigenic *C. diphtheriae* isolates from cases in Vancouver,⁴ 97% (32/33) were cultured with other pathogens.

At present, it is unclear which skin sores and sore throats should raise clinicians' index of

suspicion for a toxigenic diphtheria diagnosis. Many confirmed cases have not presented with a classic, non-healing cutaneous ulcer or toxin-mediated respiratory disease, as would be seen in an unvaccinated population. As the outbreak evolves, several important questions remain unanswered. Could *C. diphtheriae* be a secondary pathogen to the more common skin and soft tissue pathogens, *S. aureus* or GAS? Or could these organisms interact in ways that enhance *C. diphtheriae* tox gene expression or pathogenicity? In the absence of baseline carriage data, the prevalence of asymptomatic colonisation with toxigenic and non-toxigenic *C. diphtheriae* remains unknown. Likewise, the contribution of toxigenic *C. diphtheriae* to wound pathogenesis, when co-isolated with established skin pathogens, has yet to be defined. Improved understanding of the clinical manifestations and pathogenic mechanisms of toxigenic cutaneous *C. diphtheriae* infection would clarify its clinical significance and would help clinicians to identify patients who are most likely to benefit from targeted microbiological testing, treatment and public health management.

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