

Changes to the management of meningococcal disease in Australia

The Communicable Diseases Network Australia (CDNA) has agreed to change its *Guidelines for the Early Clinical and Public Health Management of Meningococcal Disease in Australia*, June 2001, with regard to saliva.

Although salivary contact has in the past been regarded as a means of transmission of meningococci, there is little evidence to support this view. Indeed, the available evidence indicates that neither saliva nor salivary contact is important in the transmission of meningococci.

Saliva has been shown to inhibit the growth of meningococci. The inhibitory property is due to the presence of other bacteria in saliva, streptococci in particular.¹ As a result, meningococci can only rarely be isolated from saliva. In a study in the United Kingdom three swabs, one from the posterior nasopharyngeal wall, another from the tonsillar area, and the third from the front of the mouth, were taken from 258 college students and cultured for meningococci. In total, 32 per cent of the nasopharyngeal swabs and 19 per cent of the tonsillar swabs cultured *Neisseria meningitidis*. However, only one swab (0.4% of those collected from the front of the mouth) cultured the organism.²

Carriage of meningococci has not been convincingly shown to be associated with saliva contact. A case-control study of United Kingdom university students found no association between carriage of meningococci and sharing of drinks or cigarettes and a weak association with 'intimate kissing' (OR=1.4, CI from 1.0 to 1.8).³ It is unclear whether carriage in these circumstances is due to saliva contact rather than to droplets shed during household-like (i.e. close and prolonged) contact.

Invasive meningococcal disease has not been shown to be associated with salivary contact. A case-control study from Auckland, New Zealand found no increased risk ($p=0.07$) of invasive meningococcal disease in children <8 years of age who had shared an item of food, drink or pacifier in the two weeks prior to hospitalisation.⁴ A case-control study of college students in the United States of America found no association (in multi-variate analysis) between invasive meningococcal disease and kissing two or more contacts on the mouth in the month prior to the onset of the illness.⁵ Clusters of invasive meningococcal disease in people who have had a low level of salivary contact (e.g. footballers who have shared drink bottles, churchgoers who have shared a communion cup) appear to be very rare. Although clusters have

been described, for example, in association with sporting events⁶ and sports clubs,⁷ the reported details indicate that point-source salivary transmission was not involved. Secondary cases in situations where dribbling of saliva is common (e.g. child-care centres) are also rare.

As the available evidence does not support saliva or salivary contact as being important in the transmission of meningococci, chemoprophylaxis is not indicated for the following contacts of a case of invasive meningococcal disease unless they are either household-like, child-care or other very close contacts (e.g. sexual contacts):

- kissing contacts, even if mouth kissing was involved;
- food, drink (including drink bottle) sharing contacts;
- cigarette sharing, bong sharing contacts;
- communion cup, lip balm, wind instrument, referee's whistle sharing contacts; or
- any other similar low-level salivary contacts.

These revised recommendations are compatible with recently revised guidelines for the public health management of invasive meningococcal disease from the United Kingdom.⁸

The revised *Guidelines for the Early Clinical and Public Health Management of Meningococcal Disease in Australia* will be available from <http://www.cdna.gov.au/pubs/other/mening.htm>. The CDNA and will continue to review these guidelines and, when necessary, post further amendments on the website.

References

1. Gordon MH. The inhibitory action of saliva on growth of the meningococcus. Great Britain Medical Research Committee, Special Report Series 3, 1917: 106-111. (Original reference not seen; summarised in Cartwright K. Meningococcal carriage and disease. In: Cartwright K, ed. *Meningococcal disease*. Chichester: John Wiley & Sons, 1995: 115-146.)
2. Orr HJ, Gray SJ, Macdonald M, Stuart JM. Saliva and meningococcal transmission. *Emerg Infect Dis* 2003;9:1314-1315.

3. Neal KR, Nguyen-Van-Tam JS, Jeffrey N, Slack RCB, Madely RJ, At-Tahar K, *et al.* Changing carriage rate of *Neisseria meningitidis* among university students during the first week of term: a cross sectional study. *BJM* 2000; 320: 846-849.
4. Baker M, McNicholas A, Garrett N, *et al.* Household crowding a major risk factor for epidemic meningococcal disease in Auckland children. *Pediatr Infect Dis J* 2000;19:983-990.
5. Bruce MG, Rosenstein NE, Capparella JM, *et al.* Risk factors for meningococcal disease in college students. *JAMA* 2001;286:688-693.
6. Orr H, Kaczmarek E, Sarangi J, *et al.* Cluster of meningococcal disease in rugby match spectators. *Commun Dis Public Health* 2001;4:316-318.
7. Koh YM, Barnes GH, Kaczmarek E, *et al.* Outbreak of meningococcal disease linked to a sports club. *Lancet* 1998;352:706-707.
8. Public Health Laboratory Service Meningococcus Forum. Guidelines for public health management of meningococcal disease in the UK. *Commun Dis Public Health* 2002;5:187-204.