Despite a suggestion that *H influenzae* infection in the United Kingdom may behave differently,[4] half the paediatricians questioned in our study were already following the American example in offering prophylaxis to siblings of contacts; we also now intend to follow this practice.

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Commentary

This case report describes the unusual, but well documented, occurrence of a case of systemic *Haemophilus influenzae* type b infection secondary to *H influenzae* type b meningitis in an older sibling. It stimulates discussion of several points. Glode *et al* were among the first to document the contagiousness of these infections.[1] Their data and that of others in North America have indicated a substantially increased likelihood that household contacts of a child with *H influenzae* type b meningitis will also contract invasive infection with the same organism.[2] A crucial point is that the risk of secondary disease is strongly correlated with the age of the household contact, being greatest in those aged ≤ 2 years.[3] Subsequent clinical studies showed that rifampicin could eradicate nasopharyngeal colonisation with *H influenzae* type b and also, but less convincingly, the strategy could decrease the risk of secondary disease. As a consequence, the advice of the American Academy of Pediatrics is that '. . . prophylaxis is recommended for all household contacts, including adults, in those households with at least one contact younger than 49 months old. . .'.

Two additional points should be emphasised. Firstly, the data resulting in this recommendation were obtained in North America. It should not be assumed that the epidemiology of *H influenzae* type b disease in North America is necessarily an appropriate basis for recommendations for the United Kingdom. *H influenzae* type b strains (carrier or disease isolates) show genotypic and phenotypic differences depending on their geographical origin. Therefore the epidemiological behaviour of different bacterial clones may be dissimilar owing to variation in virulence factors that are important to transmission and invasive potential. Secondly, the issue of what constitutes a secondary case is not straight-forward. A reasonable, but arbitrary, definition has included contacts contracting invasive disease within 30 days of exposure to the index case. Furthermore, this definition does not allow a distinction between coprimary and secondary infection.

Commendably, the authors draw attention to the disparate advice tended by paediatricians. The questionnaire, however, did not secure the answer to the following pertinent question: in a household with at least one child aged <4 years (other than the index case), how many paediatricians would recommend rifampicin for all household contacts and the index case? Those responding 'yes' would be following the advice of the American Academy of Pediatrics. The advice is sensible and the recommendation cited in the BPA *Manual on Infections and Immunisations in Children* says that: 'When there is another pre-school child in the house of a case of invasive type b infection, rifampicin prophylaxis is recommended for all household contacts. The index case should also receive rifampicin since standard treatment does not eradicate nasopharyngeal carriage'.

Finally, the 'bottom line' is that even if this policy were implemented, it has the potential to prevent only a minority of invasive *H influenzae* type b infections. Furthermore, there are documented instances in which rifampicin has apparently failed to prevent secondary cases. The controversy is likely to become academic as active immunisation should soon consign invasive *H influenzae* type b infections to the list of preventable communicable diseases and render rifampicin prophylaxis redundant.

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