series is not the method of choice: to show an association between use of penicillin and a decreased frequency of meningococcal disease in close contacts a controlled study is required. It should also be noted that a recent monograph on meningococcal disease states that the use of penicillin in Norway to prevent secondary infection "is no longer recommended."

In conclusion, there is no evidence to support the use of penicillin, with or without chemoprophylaxis, to reduce the risk of close contacts developing meningococcal disease. We fear that the only effect of this editorial will be to confuse general practitioners and others who may not be well acquainted with the subject. In our opinion the *BMJ* should instead have given prominence to the recent guidance on meningococcal disease issued by the Public Health Laboratory Service's Meningococcal Infections Working Group.⁴

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Cases are "unavoidably unpredictable"

EDITOR.—Bjørn-Erik Kristiansen and Arne-Birger Knapskog point out that, typically, 3-4% of contacts of patients with meningococcal disease carry the pathogenic strain of meningococci but fail to draw out the implications of this.¹ One of the hardest messages to get across to the public is that it is irrational to focus all attention on an isolated case and close contacts of the patient. At the time that a sporadic case presents there will be many other people in the community who are carrying the same pathogenic strain. Contacts of these community carriers (who are not readily identifiable) are also at slightly increased risk. The reason why particular people develop invasive disease is not known, although age, time of the year, genetic susceptibility, and overcrowding are all associated factors.² More recently it has been suggested that viral infection of the respiratory tract may predispose to meningococcal disease.³

The situation is thus one of shifting clusters of risk within the community, with the next case being "unavoidably unpredictable."⁴

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British guidelines should have been considered

EDITOR.—Guidelines on the management and control of meningococcal disease in England and Wales were published last December.¹ They were developed after extensive consultation between the Public Health Laboratory Service's Meningococcal Infections Working Group and consultants in communicable disease control and represent a consensus based on best available evidence as it pertains to Britain. It would have been helpful if Bjørn-Erik Kristiansen and Arne-Birger Knapskog had considered these guidelines before making policy suggestions for Britain on preventing secondary cases of meningococcal disease.²

People living in the same household as a patient with meningococcal disease are at increased risk of the disease. It is unclear whether giving antibiotics to these household contacts reduces the risk, although there is evidence of temporary protection.^{3,4} In a retrospective study of household clusters in England and Wales between 1993 and 1995 no household contacts developed disease within one to seven days of taking rifampicin, although cases occurred subsequently (L Hastings, unpublished data). Rifampicin will not always clear carriage, but Kristiansen and Knapskog do not provide references to support a policy of a combined antibiotic approach, and we understand that this is not the national policy in Norway. The suggestion that preventive treatment should be confined to those aged under 15 would have no logical basis in Britain as age specific attack rates are higher in 15-19 year olds than 6-14 year olds.⁵

Evidence of protection from randomised controlled trials does not exist and would be difficult to obtain, given the few secondary cases and the likely small differences in efficacy between policies. We believe that current recommendations in England and Wales to give rifampicin to household contacts are reasonable in the light of available evidence. The use of sulphonamides as second line drugs is not advocated because one third of strains of meningococci in England and Wales are resistant to sulphonamides and antibiotic sensitivities are usually not known when prophylaxis is given.

In outbreaks the urgency of the situation means that it is inappropriate for consultants in communicable disease control to wait for the results of surveys of swabs before taking action. We agree that increasing use of polymerase chain reaction testing could make this approach more feasible in future.

New conjugated vaccines against disease due to serogroups A and C meningococci hold great promise; vaccines against serogroup C are currently undergoing phase II trials in Gloucester and Oxfordshire. Whatever antibiotic policies are followed for the prevention of secondary cases, most (over 95%) cases of meningococcal disease are single, sporadic cases. The greater challenge of primary prevention can be met only through the development of effective vaccines.

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Authors' reply

EDITOR.—In considering the prevention of secondary cases of meningococcal disease we can divide infected people into two groups: those with incubating infections and carriers. As Andrew J Pollard and colleagues state, the most widely used chemoprophylactic agent, rifampicin,^{1,2} is satisfactory for eradicating carriage. Penicillin is unsatisfactory. Incubating infections require both eradication and treatment. They usually occur in the patient's close family.³ As M J Martin and colleagues and Pollard and colleagues agree, rifampicin is not reliable for treating incubating infections. Begg states that penicillin is more reliable than rifampicin for treating incubating infections,¹ and Wall *et al* advocate the use of parenteral penicillin to prevent some of the early infections.⁴ We therefore advocate the Norwegian practice of giving penicillin to susceptible household contacts. Treatment with penicillin provides protection from disease as long as it is maintained, but it fails to tackle the problems of (re)infection from adult contacts or the persistence of meningococci in the patient's throat flora. This is perhaps why the practice has been criticised and why we insist that it must be combined with prophylaxis with rifampicin for all close contacts.

Does oral penicillin cure incubating systemic meningococcal disease or does it, as Tim Stokes and colleagues suggest, merely hold the infection in check? We know that oral penicillin protects while it is maintained; more than 4000 households in Norway have been treated in this way without any breakdown of treatment being reported. We also know that parenteral penicillin has been successfully used to treat systemic infections for decades. The fact that oral penicillin cures incubating infection remains unproved, but do we have a better alternative for dealing with the threat of incubating disease in close contacts?

In our opinion the most important way of preventing secondary meningococcal disease, apart from vaccination, is to eradicate carriage by chemoprophylaxis. We believe that giving supplementary oral penicillin to susceptible household members provides added protection. If it could be shown that single dose ceftriaxone or ciprofloxacin cures incubating meningococcal disease as well as eradicating carriage then we would agree with Martin and colleagues that these drugs would be excellent alternatives for preventing meningococcal disease.

It was not the purpose of the editorial to recapitulate the British guidelines; had it been, the editor would surely not have invited Norwegians to write it. Unfortunately, the British guidelines were not available to us when we wrote it last December.

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