Escherichia coli O148 and Diarrhoea in Adults

Sir,—Your leading article "Dropouts' Diarrhoea" (10 August, p. 373) referred to the discovery of the O148 serogroup of Escherichia coli during an investigation of travels to Aden.1 Subsequent work in the U.S.A. demonstrated its pathogenicity in laboratory systems as well as in human volunteers2 and showed that this enteropathogen was due to an enterotoxin that was not pathogenic in experimental animals. This hypothesis has been confirmed by further field studies, though the results have not been published. Because of the withdrawal of British troops from Aden these confirmatory studies were carried out in the Gulf area and in the Arabian Gulf. As in the Aden study, the work involved a newly arrived unit of about 620 men, and the bacteriological and epidemiological observations followed the scheme used in Aden.1

The incidence of diarrhoea in the newly arrived troops was almost three times that seen in the seasoned garrison troops. In the newly arrived troops shigellosa infection was found in 18% of the cases of diarrhoea and E. coli O148 was isolated from 55% (25%) of those cases in which shigellae were not found. The cases with E. coli O148 showed a peak incidence in the first three weeks after arrival, whereas the shigella infections occurred in later weeks. In the seasoned troops shigellosa infection was found in 29% of cases whereas E. coli O148 occurred in only 4%. Thus the incidence of infection with E. coli O148 was much lower in seasoned troops, but seasoned and newly arrived troops were affected equally by the shigella infections. In both groups the peak incidence of the bacillary diarrhoea occurred between the second and third week and was mainly caused by two outbreaks due to Shigella dysenteriae 3 and Sh. dysenteriae 6, though there were some cases due to other shigella serotypes.

Samples of all meals served by the camp kitchen were examined bacteriologically and E. coli O148 was found in 12%. It was found in samples of all types of meal over the whole period of the study and therefore no single food could be exclusively incriminated as the source of infection. Faeces from 140 food handlers were examined and three were excreting E. coli O148, one being a local civilian and two British soldiers. Flies occurred in the kitchen and area and 8% were infected with E. coli O148.

As in the Aden study, E. coli O148 was clearly related to the diarrhoea and 83% of all the isolates were from specimens taken during the acute phase of the disease. Serial faecal specimens were taken from as many subjects as possible and these showed that E. coli O148 was acquired 3-7 days before the onset of diarrhoea, appeared in pure culture in the acute phase, and remained for a variable period in the convalescent stages. This is a similar pattern to that seen in infantile enteritis due to enteropathogenic E. coli.

E. coli O148 was the commonest single serogroup found in faecal specimens, accounting for 8% of all isolates; similarly in the meals it was the commonest serogroup, accounting for 23% of all E. coli isolates. During the 1968 study it was not possible to investigate in detail the origin of E. coli O148 in the meals, but in a later study it was shown that the food supplied to the kitchen was free from E. coli O148 but became infected during preparation and serving, either from infected food handlers or from flies.

In Aden the isolates of E. coli O148 were all flagellar type H28, whereas in Sharjah the epidemic strain was isolated from 50% of the men and 15% of the women of a new arrival unit with a few isolates of flagellar type H30. In the studies of infantile enteropathogenic E. coli it has been observed that within an O group more than one H type may cause disease.3

It appears that E. coli O148 is prevalent in Arabia but uncommon in the British Isles; since 1968 we have identified approximately 20,000 strains of E. coli isolated in the British Isles, and of these strains only 20 were E. coli O148. In the present studies it seemed that persons newly arrived in Arabia became infected with this enteropathogenic serogroup and developed diarrhoea. It must be stressed that E. coli O148 is not the only cause of travellers' diarrhoea but seems responsible for a significant proportion in Arabia.—I am, etc.,

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Tetracycline-resistant Group A Streptococci

Sir,—Dr Emslie's interesting letter (12 August, p. 467) has stimulated me to see if similar variations in the prevalence of tetracycline-resistant streptococci were recorded from cultures received from north Glasgow. Over the whole period of the study the rate of isolation of these organisms was 3% from 1,960 women and 0.7% from 200 men, with a ratio of 2-7. Similar results have been reported from European countries.3 4

The S. pyogenes strains isolated in Scotland were classified into 20 serotypes, and it was observed that strains of serotype 1A were more resistant to tetracycline. Both pathogenic and non-pathogenic strains were isolated, and a total of 40% of pathogenic S. pyogenes strains isolated in Scotland were resistant to tetracycline. This is similar to findings in other parts of the world.4

The tetracycline-resistant strains isolated from Scottish patients were resistant to tetracycline and chloramphenicol, but sensitive to penicillin and erythromycin. The resistance to tetracycline was plasmid-mediated.5

The presence of these resistant strains in Scotland may have implications for future antibiotic therapy. These results highlight the need for continuing monitoring of the antibiotic sensitivity of S. pyogenes to ensure that appropriate antibiotic therapy is available for patients with infections due to these organisms.