viewpoints

PSEUDOMEMBRANOUS COLITIS — More Evidence Implicating Clostridium difficile

☐ Previous studies of the aetiology of pseudomembranous colitis (PMC) has implicated *Clostridium difficile* and its production of a toxin neutralised by antitoxin to *C. sordellii [see Inpharma No. 130: p11 (1 Apr 1978)].* Although the exact identity of the causal organism may seem clinically unimportant, it is essential for the making of prophylactic and therapeutic preparations. This week another study reports results confirming the role of *C. difficile* in PMC. It is possible that the sporadic nature of the condition reflects the rarity of human colonisation by *C. difficile*, as only a small number of organisms have been isolated from normal individuals. Dr Larson and his colleagues suspect that *C. difficile* is not a normal intestinal inhabitant.

> **'The next question is: are the toxins produced by *C. difficile* directly responsible for the symptoms, or do they have a more indirect effect — operating, for example, via production from trypsin of the powerful inflammatory agent *p*-creosol?'**

Editorial: Lancet 1: 1080 (20 May 1978)

☐ Screening of bacterial isolates from 5 patients with PMC found *Clostridium* strains from 4 patients that produced *in vitro* a toxin similar to that found in PMC faecal suspension. Oral inoculation of the strains from 2 patients into hamsters caused fatal enterocolitis in those pretreated with vancomycin. The *C. difficile* that produced the toxin *in vitro* was re-isolated from hamster canal contents. This suggests that *C. difficile* causes PMC in individuals made susceptible by previous antibiotic therapy. *C. difficile* was not isolated from any of 11 healthy adults or 20 patients with other bowel disorders, but it was isolated from 5 of 8 healthy neonates and toxin was found in the faeces of 2. **'The weight of our evidence is against *C. difficile* being a permanent resident in the gastrointestinal tract.'**

Larson, H.E. et al.: Lancet 1: 1063 (20 May 1978)