a tube agglutination method, and those found to be *E coli* 0157 were grown in a modified brain heart infusion broth and tested for verotoxin production. Samples were also examined for free faecal verotoxin; if this was found in the absence of *E coli* 0157 then other toxigenic serogroups were sought by culture of the faecal sample on ordinary MacConkey agar (Oxoid CM7b) and by checking all *E coli* isolates for verotoxin production as above. All samples were also examined for the presence of other recognised enteric pathogens. All methods have been detailed in previous reports.[2 3]

We found VTEC in 31 (78%) of 40 patients with haemorrhagic colitis, but in only two (0·9%) of 229 control patients ($p < 0·001$). With the exception of one verotoxin producing *E coli* 0128, all VTEC isolated were sorbitol negative *E coli* 0157. Other recognised enteric pathogens were not isolated from any patients with haemorrhagic colitis; they were recovered from 52 (23%) of 229 control patients.

Thus our findings differ sharply from those of Larson and Welch.[1] Our findings are, however, similar to those of workers who studied sporadic cases of haemorrhagic colitis in England and Wales[4] and Canada.[5] We therefore disagree with the view expressed by Larson and Welch, and indeed regard VTEC, particularly serogroup 0157, as the maor aetiological agent in sporadic cases of haemorrhagic colitis.

PA CHAPMAN,
DJ WRIGHT,
P NORMAN,
Public Health Laboratory,
Northern General Hospital,
Herries Road,
Sheffield S5 7AU

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3 Chapman PA, Mitchelmore DL. A two-year survey of the incidence of heat-labile enterotoxin-producing Escherichia coli and other enteric pathogens in travellers returning to the Sheffield area. *Epidemiol Infect* 1988;**101**:239–47.
4 Smith HR, Rowe B, Gross RJ, Fry NK, Scotland SM. Haemorrhagic colitis and vero cytotoxin-producing Escherichia coli in England and Wales. *Lancet* 1987;**i**:1062–5.
5 Pai CH, Gordon R, Sims HV, *et al.* Sporadic cases of haemorrhagic colitis associated with Escherichia coli 0157:H7. *Ann Intern Med* 1984;**101**:738–42.

Dr Larson comments:

If acute diarrhoea in adult patients caused by *Salmonella, Shigella, Campylobacter* and *Clostridium* species are regarded as separate clinical entities, haemorrhagic colitis not due to these organisms is the single most common clinical presentation of acute diarrhoea to our infectious disease unit.[1] It was patients with this clinical presentation whom we subsequently studied for evidence of infection with verotoxin producing *Escherichia coli* and failed to find it. If these cases are, in fact, caused by infection with *E coli* 0157, this organism would be the single most prevalent gastrointestinal pathogen in the community. But I continue to be sceptical about this conclusion.

Drs Chapman, Wright, and Norman do not describe the patient population which was the source of the faecal samples they tested. Thus it is not possible to estimate a prevalence for *E coli* 0157 infection from their data (nor from data in their references 6 and 7) to compare it with our own. The percentage of cases of haemorrhagic colitis they found to be due to *E coli* 0157 is very high, however, significantly higher even than that found by Smith *et al*, or Pai *et al*. This suggests that they describe not sporadic but epidemic haemorrhagic colitis.

Reference

1 Jewkes J, Larson HE, Price AB, Sanderson PJ, Davies HA. Aetiology of acute diarrhoea in adults. *Gut* 1981;**22**:388–92.

Staphylococcus lugdunensis endocarditis

We were interested to read the description of a new type of staphylococcal endocarditis by Smyth *et al*.[1] No species identification was done for the staphylococcal strain which has most of the characteristics of *Staphylococcus lugdunensis*, a new coagulase negative staphylococcal species.[2] This species produces smooth, glossy colonies, initially cream-coloured, but becoming pale yellow to golden after five days. Using the API Staph gallery (AIP-System, Montalieu-Vercieu, France), *S lugdunensis* is incorrectly recognised as *S hominis* biotype 3. Such strains are identified as *S lugdunensis* if they have an ornithine decarboxylase and a fibrinogen affinity factor. They also have a thermostable DNAase activity, like *S aureus* but not like *S epidermidis*.

S lugdunensis, however, is probably more responsible for infections such as infective endocarditis. Three cases of infective endocarditis due to *S lugdunensis* occurred in France in 1977, 1982, and 1983.[3] The three strains, primarily recognised as coagulase negative staphylococci close to *S hominis*, but with atypical characters, were correctly identified in 1988.[2] Unlike the usual hospital *S epidermidis* isolates, the strains were susceptible to all the antibiotics tested (benzylpenicillin, meticillin, aminoglycosides, chloramphenicol, tetracycline, macrolides, fusidic acid, vancomycin . . .).

If *S lugdunensis* seems, like *S epidermidis* and *S saprophyticus*, to be a coagulase negative staphylococcus isolated from human infections, its correct identification is a necessity and can be done easily. Coagulase and fibrinogen affinity factor (clumping factor) must both be detected, and the positivity of the second test suggests only that the isolate is *S lugdunensis*; its definite identification is achieved by the other biochemical tests, including ornithine decarboxylase detection.[1 2]

J ETIENNE,
Y BRUN,
J FLEURETTE
Centre National de Référence
des Staphylocoques,
Laboratoire de Bactériologie,
Faculté de Médecine Alexis Carrel,
Rue Guillaume Paradin,
69372 Lyon Cedex 8. France

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1 Smyth EG, Wright ED, Marples RR. New type of staphylococcal endocarditis. *J Clin Pathol* 1988;**41**:809–14.
2 Freney J, Brun Y, Bes M, *et al.* Staphylococcus lugdunensis sp. nov. and Staphylococcus schleiferi sp. nov., two species from human clinical specimens. *Int J Syst Bacteriol* 1988;**38**:168–72.
3 Etienne J, Pangon B, Leport C, *et al.* Staphylococcus lugdunensis endocarditis. *Lancet* 1989;**i**:390.

Dr Smyth *et al* comment:

We agree that the organism we described as causing endocarditis can probably be identified as *S lugdunensis*, a new species first described by Etienne *et al* in 1988. The description was not available to us at the time we studied the strain from this patient, but we were cognisant that ornithine decarboxylase positive staphylococci resembling *S*