Letters to the Editor

SHIGA BACILLUS: WEST BENGAL TO BRISTOL

SIR,—Dr Pal (June 30, p 1462) describes the outbreak of *Shigella dysenteriae* type 1 in West Bengal. We wish to report the rapid intercontinental spread of this outbreak.

The 7-year-old son of a healthy Bangladeshi restaurateur was admitted to hospital on June 20, 1984, with a 2-day history of colicky abdominal pain, fever, and profuse diarrhoea with blood and mucus in the stool. He had never been abroad, but the family were being visited by two cousins from Sylhet in northern Bangladesh, adjacent to west Bengal. They were both well, with no gastrointestinal symptoms. The patient's stool was cultured and grew *S dysenteriae* type 1 sensitive to nalidixic acid, gentamicin, and ampicillin but resistant to tetracycline, co-trimoxazole, and chloramphenicol. This appears to be the organism reported by Pal. For several days the patient had severe diarrhoea, which gradually settled on treatment with nalidixic acid.

The immediate family, including the two relatives from Bangladesh, were screened for shigella infection. No positive stool culture was obtained. On questioning it was discovered that the cousins had imported cheese, mangoes, and fresh vegetables for consumption by the family. None of these items were available for examination but we believe contaminated food to be the probable source of infection. Routine public health measures to avoid spread via the local Tandoori restaurant were instituted and no further cases have appeared.

This case yet again illustrates the speed with which rare infections, even from remote areas, may spread, assisted by modern air travel. This is especially likely to occur in those with close family ties in epidemic areas.

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PYLORIC CAMPYLOBACTER SEROLOGY

SIR,—Like your correspondents[1-3] we have been intrigued by the possibility of replacing endoscopic biopsy of the gastric antrum with a blood test for gastritis. We have been using a passive haemagglutination test based on sonicated pyloric campylobacter (PC) organisms. The test detects antibody to pyloric campylobacter (PC) in most patients with gastritis. In 5 consecutive patients with undiagnosed dyspepsia and positive serology subsequent antral biopsy has proved PC to be present. In 5 patients cleared of PC and gastritis with tripotassium dicitrato bismuthate and antibiotic therapy, serial testing has shown a fourfold fall in titre in the subsequent twelve weeks. The antibody does not withstand 6-mercaptoethanol incubation, indicating that it is probably an IgM.

Table I shows that a titre of 320 or more is always associated with PC colonisation of the gastric antrum. Conversely, duodenal ulcer is rarely present in patients with completely negative serology (titres below 20).

Assessment of young male blood donors indicates an incidence of PC gastritis similar to that found in medical students by Langenberg et al[4] (table II). The use of this test in further epidemiological studies of PC gastritis and comparison with the known epidemiology of peptic ulceration will indicate whether the two diseases are one.

TABLE I—PYLORIC CAMPYLOBACTER SEROLOGY IN PATIENTS UNDERGOING GASTROSCOPY IN WHOM SERUM WAS TAKEN

Group	Reciprocal of titre				
	⩽20	80	320	1280	⩾5120
PC absent, gastritis absent*	25	5	0	0	0
PC gastritis, without ulcer	1	4	7	8	16
PC gastritis, with ulcer	2	4	2	12	8

*Includes 1 patient with duodenal ulcer and 5 patients with gastric ulcer.

TABLE II—PYLORIC CAMPYLOBACTER SEROLOGY IN 104 CONSECUTIVE CROSSMATCHED CHILDREN AND 216 BLOOD DONORS

Group	Reciprocal of titre	
	<320	⩾320
Males		
Children	57	7 *(11%)*
Adults (⩽30yr)	24	7 *(23%)*
Adults (>30yr)	51	21 *(29%)*
Females		
Children	38	2 *(5%)*
Adults (⩽30yr)	38	2 *(5%)*
Adults (>30yr)	55	18 *(25%)*

TABLE III—PYLORIC CAMPYLOBACTER SEROLOGY IN 61 FEMALES ATTENDING VENEREAL DISEASES CLINIC

TPHA*	Reciprocal of titre	
	<320	⩾320
Negative	29	1 *(3%)*
Positive	19	12 *(38%)*

*Treponemal passive haemagglutination test. The TPHA negatives were of average age 26 (5 over 30), the TPHA positives were of average age 24 (6 over 30).

Serological testing may also help answer the question—"How do you acquire pyloric campylobacter and from whom"? Without further comment we present the data in table III.

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CLEARANCE OF LUNG METASTASES OF PROSTATE CARCINOMA AFTER TREATMENT WITH LH-RH AGONIST

SIR,—Agonistic analogues of luteinising hormone releasing hormone (LH-RH) have proved effective in patients with prostate carcinoma. We describe here the radiological disappearance of lung metastases and bone lesions in a patient with prostate carcinoma after treatment with D-Trp-6-LH-RH.

This 71-year-old man was admitted to hospital in Brazil in April, 1983, with urinary obstruction, bone pain, and debilitation and a history of gross haematuria and, in 1981, prostatectomy to relieve urinary retention. A chest X-ray revealed bilateral hilar adenopathy and numerous nodular densities (fig 1). These findings, together with the results of a needle biopsy of the prostate and a skeletal survey, led to a diagnosis of bone and pulmonary metastases from an adenocarcinoma of the prostate.

D-Trp-6-LH-RH (kindly provided by Debiopharm SA, Lausanne) was administered subcutaneously every 24 h at 500 μg daily for the first 7 days and then 100 μg daily. After 4 weeks of treatment with D-Trp-6-LH-RH plasma testosterone levels had fallen from 400 to 35 ng/dl, with a striking decrease in the bone pain and improvement in the urinary symptoms. After 14 weeks the chest X-ray was clear (fig 2), and chest X-rays in November, 1983, and in March, 1984, after he had been on D-Trp-6-LH-RH for 11 months, continued to show a complete resolution of lung lesions. A bone survey in March, 1984, was negative. Before treatment, this patient had been bedridden. After 14 weeks of therapy with the LH-RH analogue he was able to resume all his activities. Since