TABLE III.—*Distribution of Sera Giving Different*Ratios: $\frac{\text{PR8 Titre}}{\text{BAR Titre}}$

Ratio: $\frac{\text{PR8 Titre}}{\text{BAR Titre}}$	1 or less	1	2	4	8	16	32	Over	Total No. Sera
Percentage of sera (using haemagglutination-inhibition tests) ..	2	2	11	19	23	23	8	1	47
Percentage of sera (using complement-fixation tests) ..	6	23	34	29	4	4	0	0	46

Note.—Titres are expressed as the reciprocal of the final dilution at the end-point. These figures are obtained using the assumption of a titre of 1 for any titre less than 2.

The average PR8 titre is 215 (H.I.T.) and 4.3 (C.F.T.), and the average BAR titre 20.0 (H.I.T.) and 1.4 (C.F.T.).

The ratio $\frac{\text{average PR8 titre}}{\text{average BAR titre}} = 10$, for haemagglutination-inhibition tests, and 4, for complement-fixation tests.

It is unlikely that these high PR8 antibody levels represent the result of contact with this virus at any period of more than a year or two ago, since it is known that, following the initial response to infection, specific antibody levels fall steadily over a period of one to two years by from 5 to 100 times in the absence of subsequent infection (cf. Stuart Harris *et al.*, 1938). It is possible that high PR8 titres might be maintained by the persistence of that strain in a form in which it cannot be isolated by present methods. Current strains of virus such as BAR may then represent variants of this latent PR8 form, which are capable of causing epidemics, capable of growing in eggs or infecting ferrets or mice, and therefore capable of being isolated.

Finally, and most probably, the present findings may be taken as falling into line with the work quoted earlier, especially that of Stuart-Harris and Miller (1947) and Morgan *et al.* (1948), leading to the conclusion that the PR8 antibody levels represent a non-specific response to infection or subinfection by a current virus strain, of which BAR is an example.

Summary

Haemagglutination-inhibition and complement-fixation tests were carried out on 50 normal serum samples, using influenza A strain PR8 (classical) and BAR (recently isolated), and the titres against these strains compared.

It was found that PR8 titres were on the average 10 times higher than titres against BAR virus by the haemagglutination-inhibition method, and four times higher by complement-fixation.

Acknowledgments are due to Professor T. J. Mackie and Dr. R. H. A. Swain for their advice and criticism; to Dr. Cummings, of the Blood Transfusion Service; and to blood donors for their co-operation in the supply of sera.

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PRESENCE OF CERTAIN SEROLOGICAL TYPES OF BACT. COLI IN THE HUMAN INTESTINE

BY

W. W. WALTHER, M.B., B.S.

With the Technical Assistance of

E. G. MILLWOOD, F.I.M.L.T.

(From the Area Pathological Laboratory, Whipps Cross Hospital, London, E.11)

Stevenson (1950) demonstrated the presence of *Bact. coli* D433 in the faeces of adults suffering from diarrhoea, but suggested that these organisms might be the normal inhabitants of the duodenum or jejunum and be carried down by increased intestinal activity due to other causes. This suggestion clearly required further study. Accordingly, during the period July 21 to October 30, 1950, swabs were taken from various parts of the alimentary tract of 51 cases (50 adults and a child of 7 months) examined *post mortem*, none of which had recently had diarrhoea or any treatment likely to interfere with the natural intestinal flora. These swabs were examined for the presence of *Bact. coli* of serological types D433, Aberdeen β , E893, E990, Vincent, and E611. Each swab was plated on half a Petri dish containing McConkey's agar medium and incubated aerobically at 37° C. for 18 to 24 hours. A comprehensive sample of the resulting growth was taken by sweeping a platinum loop across the surface of the medium, and this was first tested with a 1 in 500 solution of acriflavine in physiological saline.

Joan Taylor (personal communication) has found that some strains of *Bact. coli* other than D433 may be agglutinated on a slide by the specific sera prepared against *Bact. coli* D433, but most of these are also agglutinated by the acriflavine solution. On the other hand, none of the many strains so far examined that belong to the serological types which were the subject of this inquiry have ever been found to agglutinate in acriflavine. The acriflavine test is therefore very useful for preliminary "screening."

If no agglutination resulted in the acriflavine solution 12 individual colonies were tested with D433 K, Aberdeen β K, and a composite serum containing agglutinins to strains E893, E990, Vincent, and E611. If agglutination occurred in the acriflavine solution some four to six individual colonies were tested with the specific sera and a further sweep was subcultured on to plain nutrient agar. Those colonies which agglutinated in any of the specific sera were also subcultured to find if they should become acriflavine-negative. This routine was repeated on the following day, when only "acriflavine-negative" colonies were tested with the specific sera, at least 12 such colonies from each agar plate being tested before the swab was pronounced "negative." The slide-agglutination technique was used throughout.

The results are summarized in the Table.

The following is a list of the conditions from which the patients died: coronary thrombosis (6), carcinoma of the lung (5), congestive cardiac failure following hypertension (5), bronchopneumonia (5), haematemesis (3), carcinoma of stomach (3), senile myocardial degeneration (2), chronic nephritis (2), carcinoma of oesophagus (2); and one from each of the following: pyonephrosis, renal failure, carcinoma of breast, carcinoma

Agglutinin	Duodeno-jejunal Junction		Ileum		Transverse Colon	
	+	-	+	-	+	-
Acridiflavine . . .	21	30	22	29	24	27
Acridiflavine negative after subculture . .	16		15		15	
D433 K . . .	0	16	0	15	0	15
Aberdeen β K . . .	0	16	0	15	0	15
Composite E893, E990, Vincent, E611 K . .	0	16	0	15	0	15

+ Indicates agglutination. - Indicates no agglutination.

of rectum, carcinoma of liver, lymphosarcoma, chromophobe adenoma of pituitary, senile pulmonary tuberculosis, mitral stenosis, cor pulmonale, pulmonary abscess, cerebral abscess, multiple pulmonary infarction, pyelonephritis, peritonitis, aortic aneurysm, cerebral thrombosis, and osteogenesis imperfecta.

Summary

Swabs taken from the duodeno-jejunal junction, the ileum, and the transverse colon at post-mortem examination of 50 adults and one infant failed to provide any evidence of the presence in them of the serological types of *Bact. coli* D433, Aberdeen β , E893, E990, Vincent, and E611.

We are grateful to Dr. Joan Taylor for much helpful advice and criticism and for supplying the specific sera and the acridiflavine solution.

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Medical Memoranda

Pyrexia of Unusual Origin

One of the principles of medicine is to avoid multiple diagnoses whenever possible. Thus it is particularly satisfying to be able to supply one diagnosis which satisfactorily explains a series of illnesses previously considered to be quite unrelated to one another. The case described below illustrates this point. The history was obtained partly from the patient and partly from her medical records; consequently some of the evidence is hearsay and not completely conclusive. However, I think it is convincing enough to be of interest.

Case Report

The patient was the only child of divorced parents, both of whom had remarried and subsequently had further children. She was brought up in a convent and was studious and retiring. She felt unwanted and unloved. After failing a university scholarship examination she had a nervous breakdown and was treated with electric convulsion therapy and possibly with some form of insulin therapy. She joined the A.T.S. in August, 1947.

In December, 1947, while in a military hospital for a septic finger, she developed abdominal pain, for which her appendix was removed. The pathology was found to be inconsistent with the symptomatology. She was later suspected of tampering with her wound and creating a temperature by illicit means. She was interviewed by a psychiatrist, who considered discharging her from the Army but recommended her retention because of her unsatisfactory family background. "Psychopathic personality with emotional abnormality" was diagnosed, and she returned to duty as a medical orderly in an M.I. room.

In April, 1948, she entered a second hospital with erythema nodosum and another septic finger. She developed jaundice, diagnosed as infective hepatitis, a few days later. One month later, two days after a tooth extraction, she developed enlarged lymph nodes, "a blood picture suggestive of glandular fever and a strongly positive Paul-Bunnell." She next had whooping-cough, followed on June 26 by maxillary sinusitis. Some three weeks later she had a swelling over the right parotid gland and an abscess in the right thigh from which *Bact. coli* was cultured. A hard nodule from the region of the right knee was excised, and sections showed "fibrosis with many fat phagocytes, suggesting that trauma had played a part in its origin." She was discharged on sick leave in September, 1948.

On October 17 the patient was admitted to the Royal Victoria Hospital, Netley, with a cellulitis of her right thigh. This was treated with penicillin and sulphathiazole, and it subsided without incision. An evening pyrexia of 101-102° F. (38.4-38.9° C.) with severe night sweats persisted, and she complained of pain in her knees and ankles. By November 6 examination revealed no abnormality whatsoever except for some small scars on her fingers, thighs, and lower legs—residua from old abscesses. However, she remained febrile, and, though she was watched closely, there was no evidence that she was falsifying her temperature. X-ray examination of the chest, blood cultures, Wassermann and Kahn tests, catheter specimen of urine, and examination of the stools for pathogens were negative. The E.S.R. was 6 mm. in 1 hour; haemoglobin, 13.7 g. per 100 ml.; leucocytes 5,700 per c.mm. (neutrophil polymorphs 39%, lymphocytes 55%). The Widal agglutinations were:

Typhoid . . .		"O" 1:320; "H" 1:1,280
Paratyphoid A . . .		"O" 1:80; "H" 1:1,280
Paratyphoid B . . .		"O" 1:320; "H" 1:1,280
Typhoid Vi antigen . . .		1:12
<i>Br. abortus</i> and <i>melitensis</i>		negative
Paul-Bunnell . . .		1:128

Although there was no superficial lymphadenopathy and the spleen was not palpable, it was considered that her glandular fever was running an unusually prolonged course. We were unable to explain the Widal reaction, as she stated, and this was confirmed by her paybook, that she had never been inoculated.

On November 22 a parcel arrived at the unit for redirection to the patient. The circumstances were peculiar and suspicious. The parcel was opened and contained a syringe, needles, and numerous empty tetanus toxoid bottles. When taxed with this she admitted that over a period of some three years she had been taking various unspecified tablets and inoculating herself with tetanus toxoid and T.A.B., partly as an innocent experiment and partly in a desire for self-mortification. She admitted producing many, but not all, of the curious bumps found at the second hospital and diagnosed as erythema nodosum. She denied any malicious intentions, but felt that there must be something wrong with her head to make her behave like this, and, although depressed, she welcomed treatment.

In May, 1949, following treatment, she was well, was working, and had made a good social recovery.

Comment

The chief interest in this case lies in attempting to assess to what extent this patient's many symptoms can be attributed to her repeated self-inoculation.

Glandular fever was diagnosed at another hospital on the strength of enlarged lymph nodes, "a blood picture suggestive of glandular fever and a strongly positive Paul-Bunnell." Glandular fever cells were not specifically mentioned, and it is not known what technique was used for the Paul-Bunnell test. It has been shown that the agglutinin in glandular fever is absorbed by ox cells but not by guinea-pig's kidney, while the agglutinin of serum sickness is absorbed by both (Stuart, Welch,