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Letters to the Editor

Effect of growth at 43°C on phage typing of staphylococci

During 1975 we noted that a number of initially untypable strains of *Staphylococcus aureus* had become susceptible to lysis by one or more of the basic set of typing phages after varying periods of storage at room temperature on slopes of nutrient agar. During an investigation of this phenomenon it was shown that loss of resistance to phage lysis could also be greatly accelerated by growing the cultures of staphylococci in broth at 43°C before they were typed. The results of testing 110 random isolates in parallel after growth in broth culture at 37°C and 43°C are shown in Tables 1 and 2. There was a 10% increase in the number of typable strains at routine test dilution (RTD), and a further 30% increase in typability in strains that required examination at 100 RTD, with no apparent loss in discrimination.

Although Ma and Mandle (1961) reported a similar observation 15 years ago the effect of growth at elevated temperature on typability does not appear to be widely known. I draw attention to it because it may be useful in routine typing.

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Reference

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Table 1 Results of typing *Staphylococcus aureus* strains at RTD and 100 RTD after growth in broth at 37°C and 43°C

	RTD		100 RTD	
	37°C	43°C	37°C	43°C
Number of strains typed	68	73	16	23
Number of strains NOT typed	42	37	16	9
Total	110	110	32	32

Table 2 Different typing patterns of *Staphylococcus aureus* obtained after growth at 37°C and 43°C

Strain No.	RTD	
	37°C	43°C
1	NT	80
2	NT	52/52A/80
3	NT	54
4	NT	94/96
5	NT	95
6	NT	52/52A/80
7	77	NT
8	83A/85	42E/54/83A/85
9	80/95	29/52/52A/80/95
10	80/95	52/52A/80/95
11	95	52A/80/95/53
100 RTD		
	37°C	43°C
12	NT	29/52/52A/79/53/54
13	NT	52/80/85
14	NT	47/53
15	NT	84/85
16	NT	52/80
17	NT	52A/79
18	NT	85
19	80	52/52A/80
20	47	6/47/54/75/84/85
21	52	29/52/52A
22	77	42E/47/53/54/75/77

Outbreaks of food poisoning attributed to lecithinase-negative *Clostridium welchii*

The ability of *Clostridium welchii* type A to produce a lecithinase which splits the insoluble fatty material in human serum was first described by Nagler (1939). Since then this reaction, and its specific inhibition, has been recommended and widely used as a rapid diagnostic and confirmatory test for *Cl. welchii* (Hayward, 1943; McClung and Toabe, 1947).

In the investigation of food poisoning outbreaks where *Cl. welchii* type A is suspected as the causative agent, typical colonies from the primary isolation medium, after anaerobic incubation, are streaked across a plate of egg yolk medium. Half of this has previously been spread with 2 or 3 drops of standard *Cl. welchii* type A antitoxin and allowed to dry. After anaerobic incubation growth of *Cl. welchii* is surrounded by a zone of opalescence (Nagler reaction). This is inhibited on the other half of the plate by the specific antitoxin. *Cl. bifermentans*, *Cl. sordellii*, and *Cl. centrosporogenes* also produce a lecithinase partially inhibited by *Cl. welchii* type A antitoxin. In the past two years we have encountered three outbreaks of food poisoning attributed to lecithinase-negative strains of *Cl. welchii*.

Outbreak 1

In October 1974, 105 of the patients and staff in a hospital suffered symptoms of food poisoning. *Cl. welchii* was isolated from 16 of 20 faecal specimens. Roast lamb and gravy were suspected as the cause of the outbreak but samples were not available for examination.

Outbreak 2

In August 1975, 18 of 36 people attending

an old people's luncheon club suffered from diarrhoea 12 hours after eating a meal consisting of steak and kidney, potatoes, cabbage, and gravy. *Cl. welchii* was isolated from the faeces of four affected persons and from steak and kidney in a specimen meal (1.1×10^7 /g).

Outbreak 3

During July 1976, 77 patients in a geriatric hospital experienced diarrhoea 12-15 hours after consuming reheated roast beef. *Cl. welchii* was isolated from 68 of the patients and from the roast beef (1.5×10^8 /g).

In all three outbreaks the strains isolated were lecithinase-negative on nutrient egg yolk medium. All strains had identical serological typing reactions (Hughes *et al.*, 1976) agglutinating with the unusual combination of four antisera, types 25, 27, 31, and 68. The three outbreaks occurred within 25 miles of each other and a common source of the organism is possible.

Representative strains from the three outbreaks were examined at the Wellcome Research Laboratories for the production of toxins. A very weak alpha toxin was produced which exhibited a very weak α -haemolytic activity but no demonstrable lecithinase activity. With these strains, therefore, the haemolytic test is more sensitive than the lecithinase test for detecting alpha toxin. The strains were considered to be atypical *Cl. welchii* type A. A strain from one of the outbreaks has been deposited with the National Collection of Type Cultures (reference number NCTC 11144).

The implication of these results is clear. If laboratories are relying on the production of lecithinase as the sole confirmatory test for *Cl. welchii* then it is possible that some strains will be missed. It is recommended, therefore, that organisms that are lecithinase-negative but which grow well and colonially resemble *Cl. welchii* on neomycin blood agar should be tested for motility, gelatin hydrolysis, nitrate reduction, action on litmus milk, and fermentation of glucose, lactose, sucrose, salicin, maltose, and starch.

We are unaware of any previous reports of outbreaks of *Cl. welchii* food poisoning where the cultures isolated were lecithinase negative and would be interested to hear whether other workers have encountered a similar problem.

We are grateful to Dr P. D. Walker and Mrs G. Roper of the Wellcome Research Laboratories for their help.

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Book reviews

Cerebrospinal Fluid Cytology. An Introduction and Atlas. By Manfred Oehmichen. Pp. vii + 208; illustrated; £27.75. Philadelphia, London, Toronto: W. B. Saunders. 1976.

Many of us can remember when the cerebrospinal fluid (CSF) contained only three kinds of cell, and even these could be difficult to identify in the counting chamber. Until the introduction of Sayk's sedimentation method about 15 years ago, only very dedicated clinical pathologists succeeded in making satisfactory smears from fluids with low cell counts. Anybody can do it now, either with a sedimentation chamber or with its successor, the cytocentrifuge. Most of the numerous publications on this subject are in German and have been largely ignored by workers in America and, I am afraid, in Britain.

Oehmichen's book shows many photographs in half-tone and in colour, mainly from sediment-chamber preparations; but it is not just a picture-book. The text discusses the pathophysiology of the cells and their origins and functions from studies performed in large part by the author. Far from being an 'introduction' as the subtitle states, it covers pretty well all that is known at present about the cells of CSF.

There are a few cell identifications about which I feel doubtful. The 'mast cells' in Plate 2 look very unlike the usual tissue mast cells and would pass more easily for promyelocytes. The statement that medulloblastoma cells are almost indistinguishable from lymphoma cells is wrong, because the former nearly always occur in coherent clusters, the latter never. One would also like some evidence that the cells represented as those of oligodendroglioma and pinealoma are really tumour cells and not reactive lymphoid cells.

In the next edition a revision of the text could remove a number of awkwardnesses resulting from the translator's difficulty in rendering medical German into medical English; but these do not interfere with comprehension.

Advances in knowledge of cell function (particularly of the lymphocytes and macrophages) will make future editions of this book progressively more important. With its exhaustive and up-to-date bibliography and thorough index it