

the ingestion of these hydrocarbons by rats is followed by the excretion of acid-decomposable hydrocarbon precursors either such compounds are excreted in very small amounts or their

decomposition occurs under conditions which differ from those which bring about the breakdown of the naphthalene precursor excreted after the administration of naphthalene.

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Antigenic Relationships of the Species *Shigella dispar*.

PHILIP L. CARPENTER. (Introduced by C. A. Stuart.)

From the Department of Bacteriology, Rhode Island State College, Kingston, R.I.

Castellani¹ isolated from the feces of patients with clinical dysentery a bacterium which he named "*Bacillus ceylonensis* B." Five years later² he reported the isolation from normal human stools of *Bacillus madampensis*. Both organisms were gram-negative, nonmotile rods, fermenting many carbohydrates, including lactose, with production of acid only, and producing indol. *Bacillus ceylonensis* B fermented dulcitol, whereas *Bacillus madampensis* did not. Both fermented sucrose. Andrewes³ included these 2 species, together with some other lactose-fermenting dysentery-like varieties, in his "*Bacillus dispar*." Current terminology places these microorganisms in the genus *Shigella*.

Castellani stated that *Bacillus ceylonensis* B was a serologically homogeneous species. Glynn and Starkey,⁴ working with 15 strains, reported *Shigella dispar* antigenically heterogeneous. The present investigation was undertaken to study further the antigenic structure of strains designated *Shigella dispar* or *Shigella ceylonensis*.

Thirty-seven strains of bacteria are included in this preliminary report. Five were received from the American Type Culture Collection; 10 were received from Dr. K. M. Wheeler, and were isolated in Connecticut within the past 4 years; 3 were isolated in Texas in 1942, and were received from Dr. MacDonald

Fulton; two were isolated recently in Rhode Island by Dr. C. A. Stuart; the remaining 17 were isolated in a number of different laboratories along the Atlantic seaboard. Eleven strains of *Sh. ceylonensis* were received, and 26 strains of *Sh. dispar*.

All 37 strains produced acid from dextrose, lactose, maltose, and mannitol; failed to ferment salicin, and were indol positive. Further fermentation reactions are noted in Table I.

Antiserums were prepared for 2 strains of *Sh. dispar* and one of *Sh. ceylonensis*. Table I presents a summary of the results obtained when the 37 strains were tested for agglutination in the various antiserums. The titers stated are those for the type strains listed, but all organisms grouped together behaved similarly.

It is apparent that the 11 strains of *Sh. ceylonensis* are serologically identical or closely related to *Sh. dispar* type 205. Most of the type 205 strains were pigmented, the color varying from a faint yellowish-orange to a deep orange. None of the *Sh. ceylonensis* strains were pigmented, but there appears to be no correlation between pigment production and antigenic structure.

Two other antigenic types, 171 and 221, are evident. Thus, there are 3 well defined serological groups, the major group including 21 of the 26 strains of *Sh. dispar*, together with all 11 strains which were designated *Sh. ceylonensis* on the basis of their fermentation reactions.

This grouping of the strains is confirmed by adsorption experiments. Antiserums in 1:100 dilution were adsorbed twice for one hour at

¹ Castellani, A., *J. Hyg.*, 1907, **7**, 1.

² Castellani, A., *C. f. Bakt.*, 1912, I, Orig., **65**, 262.

³ Andrewes, F. W., *Lancet*, 1918, **194**, 560.

⁴ Glynn, J. H., and Starkey, D. H., *J. Bact.*, 1939, **37**, 315.

TABLE I.
Fermentation and Agglutination Reactions of *Shigella ceylonensis* and *Shigella dispar*.

Species	Type strain	Total strains	Fermentation of*					Serum agglutination†		
			Sucrose	Dulcitol	Xylose	Sorbitol	Rhamnose	Anti-167 (<i>ceylon.</i>)	Anti-205 (<i>dispar</i>)	Anti-171 (<i>dispar</i>)
<i>Shigella ceylonensis</i>	167	11	+	+	+	+	±	1:12,800	1:12,800	1:1,600
<i>Shigella dispar</i>	205	21	±	—	±	±	+	"	"	"
	171	2	+	—	+	+	+	1:100	1:100	1:25,600
	221	3	—	±	±	+	+	<1:50	<1:50	<1:50

* + indicates acid production,

± indicates acid production by some strains.

† Tests were read after 12-18 hours at 55°C.

37°C with heavy antigenic suspensions. Some of the results are shown in Table II.

TABLE II.
Reciprocal Adsorption of Anti-*ceylonensis* and Anti-*dispar* Serums.

Antiserum	Adsorbing strain	Test strain	Agglutination titer × 100
167	Unadsorbed	167	1:128
	205	"	<1:1
	171	"	1:64
205	Unadsorbed	205	1:128
	167	"	<1:1
	171	"	1:128
171	Unadsorbed	171	1:256
	167	"	1:256
	205	"	1:256

It is evident that strains 167 and 205 are serologically identical. Since these cultures represent *Sh. ceylonensis* and *Sh. dispar* respectively, the conclusion seems warranted that these 2 names designate the same organ-

ism, differing only in pigmentation and in one or two biochemical characteristics. Type 171 (Tables I and II), while antigenically related to type 205 (*Sh. dispar*), possesses a major antigenic fraction not shared by type 205. On the other hand, type 221, biochemically related to *Sh. dispar*, appears to have no antigens in common with type 205. The present work confirms the antigenic heterogeneity of *Sh. dispar*, as shown by Glynn and Starkey; nevertheless, 86% of the 37 strains used in this investigation were antigenically closely related or identical.

Leaving in abeyance for the present the question of priority, it is tentatively suggested that bacterial groups designated *Shigella ceylonensis* and *Shigella dispar* be combined and defined as gram-negative, non-sporeforming rods which produce indol, ferment dextrose and lactose with production of acid only, and may or may not ferment sucrose or dulcitol.

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Vitamin E Deficiency in Rats Given Succinyl Sulfathiazole in Purified Diets.

FLOYD S. DAFT, K. M. ENDICOTT, L. L. ASHBURN, AND W. H. SEBRELL.

From the National Institute of Health, U. S. Public Health Service, Bethesda, Md.

Daft, Ashburn and Sebrell¹ reported the occurrence of various lesions in rats given sulfaguanidine (sulfanilylguanidine) or sulfasuxidine (succinyl sulfathiazole) in purified diets. These lesions included hyalinization,

necrosis and calcification of voluntary muscle. Ashburn, Daft, Endicott and Sebrell² remarked on the similarity of these lesions to those ascribed to vitamin E deficiency. They noted

¹ Daft, Floyd S., Ashburn, L. L., and Sebrell, W. H., *Science*, 1942, **96**, 321.

² Ashburn, L. L., Daft, Floyd S., Endicott, K. M., and Sebrell, W. H., *Pub. Health Rep.*, 1943, **57**, 1883.