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 (*ceylon.*)	Anti-205 (*dispar*)	Anti-171 (*dispar*)
Shigella ceylonensis	167	11	+	+	+	+	±	1:12,800	1:12,800	1:1,600
Shigella dispar	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 organism, 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.

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Daft, Ashburn and Sebrell[1] 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 Sebrell[2] 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.

that the diets into which these sulfonamide drugs were incorporated were marginal or possibly deficient in vitamin E but that hyalinized or necrotic muscle fibers were seen but rarely in control rats. We wish to report at this time the occurrence of these muscle lesions (hyalinization, necrosis and calcification) in a group of rats given a somewhat similar purified diet containing sulfasuxidine and their prevention by α-tocopherol.*

Twelve litters of 3 rats each were placed on experiment at weaning at 21 to 25 days of age. Each group of litter-mates consisted of rats of the same sex. One rat in each litter was given experimental diet No. 735, which contains sulfasuxidine, and a weekly oral supplement of 3 mg of α-tocopherol (Merck) in ethyl laurate; another was given the same diet (No. 735) but no α-tocopherol supplement; and the third was given the control diet No. 736, which does not contain sulfasuxidine, and no α-tocopherol supplement. The control diet No. 736 consists of leached and alcohol extracted casein 25 g, sucrose 63 g, salt mixture 550[3] 4 g and lard 8 g, into which is incorporated 1 mg of thiamine hydrochloride, 2 mg of riboflavin, 1 mg of pyridoxin hydrochloride, 4 mg of calcium pantothenate, 2 mg of niacin, 100 mg of choline chloride and 400 μg of 2-methyl-1,4-naphthohydroquinone diacetate (vitamin K). The experimental diet No. 735 is identical except that 1% of sulfasuxidine replaces an equivalent amount of sucrose. Twice weekly each rat was given an oral supplement of 0.25 cc of corn oil containing 2000 units of vitamin A and 200 units of vitamin D (Natola).

Twenty-two of the 24 rats receiving sulfasuxidine died after 36 to 84 experimental days. Eleven of these had been given the α-tocopherol supplement and 11 had not; their average survival times were 48 and 58 days, respectively. The remaining 2 animals receiving sulfasuxidine and 9 control animals were sacrificed at the end of 3 months, the other 3 controls at the end of 4 months on the experiment.

The incidence of muscle lesions is shown in Table I. These lesions have been described in detail previously.[2]

TABLE I.
Incidence of Muscle Lesions.

No. rats in group	Sulfasuxidine in diet	Individual weekly supplement of α-tocopherol	No. rats with muscle lesions
12	0	0	0
12	1%	0	9
12	1%	3 mg	0

From the negative findings with the control rats, it does not appear that the basal diet which we have employed will produce muscle damage in these young rats during the experimental period used. The high incidence of muscle lesions in the rats which were given sulfasuxidine in an otherwise identical diet appears to implicate this drug in their production while the preventive action of α-tocopherol indicates that these changes are due to a deficiency of vitamin E. It is not clear at the present time whether this deficiency arises through an increased demand for the vitamin, through interference with utilization of an otherwise adequate supply, or through a decrease in the amount available to the tissues.

While α-tocopherol is effective in preventing muscle lesions it evidently does not completely neutralize the toxic effects of sulfasuxidine as is evidenced by its failure to prolong the life of the experimental animal.

Conclusions. (1) Hyalinization, necrosis and calcification of voluntary muscle occur in rats given succinyl sulfathiazole in certain purified diets. (2) These lesions may be prevented by the oral administration of α-tocopherol.

* A purified diet, containing sulfaguanidine instead of sulfasuxidine but otherwise identical to one (No. 735) used in the present study has been fed for 3 months and has failed almost completely to bring about the occurrence of muscle lesions. The differences between our earlier[1] and present experimental conditions are under investigation.

3 Spicer, S. S., Daft, Floyd S., Sebrell, W. H., and Ashburn, L. L., *Pub. Health Rep.*, 1942, **57**, 1559.