Typical micrographs of replicas made in the way just described are shown in Fig. 1-3. They are from the periphery of plaques developed on Petri dishes inoculated with a mixture of *E. coli* and a T2 strain of its bacteriophage. A number of isolated bacteriophage particles adhering to the film are evident in the clear areas of Fig. 1; the film also shows numerous pits that are undoubtedly replicas of particles that remained on the agar. The mass at the left of this micrograph, which approximately outlines the remains of a single bacterial cell, is a typical example of how completely bacterial protoplasm becomes converted into bacteriophage particles after invasion by one of them. Similar masses within the confines of old cells are reproduced at higher magnifications in Fig. 2 and 3. The separate particles can readily be recognized even when, as is commonly the case, they have partially collapsed presumably as a consequence of drying. These pictures do not reveal in detail how the bacteriophage particles are formed but they do suggest that a careful study of films taken from cultures containing bacteria in various stages of infection may be expected to yield such information.

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Sugar Alcohols. XXV. Sorbitol and Sorbitan as Precursors of Liver-Glycogen in the Rat.*

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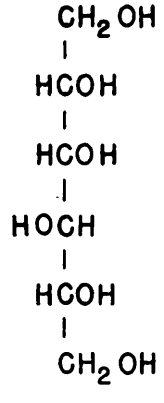
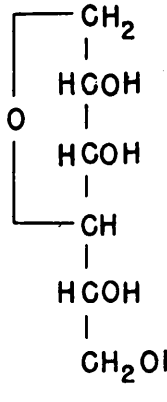
For many years we have been concerned with the fate of the sugar alcohols in the animal body.[1] Previously it was shown that mannitol served as a precursor of glycogen in the liver of the rat, whereas its anhydrides mannitan and isomannide were not available in this capacity. Sorbitol, an isomer of mannitol, surpasses the latter as a glycogen former. Having available crystalline sorbitan, anhydrosorbitol, it occurred to us to study its availability as a source of liver glycogen. Previously Carr and Krantz showed that polygalitol-1,5-anhydro-D-sorbitol was not available as a precursor of glycogen in the rat.[2] The relation of sorbitol to sorbitan is shown by the following formulas.

Experimental. Young white male rats from a uniform source, weighing from 150 to 200 g were fed a balanced ration (Purina Chow) for 3 days. They were then fasted for 48 hours and divided into the following experimental groups according to diet.

1. Cacao butter controls.
2. Cacao butter plus 30% crystalline sorbitol.



D-SORBITOL
M.p.97° $[\alpha]_D$ -1.97°



D-SORBITAN
Probable structure
M.p.113°

* The sorbitol and sorbitan were generously supplied by the Atlas Powder Company of Wilmington, Del.

1 Carr, C. J., and Krantz, J. C., Jr., *Advances in Carbohydrate Chemistry*, Vol. I, p. 175-192.

2 Carr, C. J., and Krantz, J. C., Jr., *J. Biol. Chem.*, 1934, **107**, 371.

TABLE I.
Glycogen Storage in Liver of White Rat After Feeding Sorbitol and Sorbitan in a Cacao Butter Diet.

Cacao butter		Sorbitol		Sorbitan	
No. of rats	Liver glycogen, %	No. of rats	Liver glycogen, %	No. of rats	Liver glycogen, %
3		3	0.85	3	0.05
3		8	1.16	3	0.02
3	Less than 0.1	5	1.37	3	0.04
3				3	0.04
3				3	0.05
				3	<0.01
				3	<0.01

3. Cacao butter plus 30% crystalline sorbitan.

Several animals in each group were divided into separate cages containing 3 to 8 rats each. The animals were fed their respective diets for 2 days. At the end of the feeding period the animals were anesthetized with intraperitoneal injections of amytal sodium and their livers extirpated. The livers in each group were pooled. The glycogen was determined by the method of Good *et al.*,[3] and the dextrose obtained by its hydrolysis was determined by the Munson-Walker method. The results are shown in Table I.

Conclusion. The data in Table I show that sorbitol and sorbitan behave like their isomers mannitol and mannitan[4] with respect to glycogen storage in the liver of the white rat. Mannitol and sorbitol are precursors of glycogen whereas their anhydrides are not.

[3] Good, C. A., *et al., J. Biol. Chem.*, 1933, **100**, 485.

[4] Carr, C. J., *et al., J. Biol. Chem.*, 1933, **102**, 721.

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Alteration of the Distribution and Excretion of Streptomycin.*

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Phagocytosis of colloidal particles by the reticulo-endothelium and the localization of such particles in inflamed areas under certain conditions are well known phenomena. These processes apparently depend upon several factors, among which are (a) charge of the particle, (b) particle size, and (c) capillary permeability.

It has been observed that penicillin[1] and streptomycin,[2] while effective in eliminating the septicemic phase of certain diseases, fail to provide a complete cure. In the light of previously reported evidence,[3] indicating that liver and spleen levels of streptomycin remain negative while blood levels are within a therapeutic range, it might reasonably be supposed that therapeutic failure[4] is due to

* Streptomycin used in these experiments was streptomycin hydrochloride.

[1] Robinson, H. J., Thesis, Rutgers University, New Brunswick, N.J., 1943, 43.

[2] Keefer, C. S., Blake, F. G., Lockwood, J. S., Long, P. H., Marshall, E. K., and Wood, B., Jr., *J. A. M. A.*, 1946, **132**, 9.

[3] Kornegay, G. B., Forgacs, J., and Henley, T. F., *J. Lab. and Clin. Med.*, 1946, **31**, 523.

[4] Kelly, E. G., and Henley, T. F., personal communication.