especially those which have a relatively low virulence for experimental animals under normal conditions. Studies of this type of deficiency in experimental animals of other species are in progress.

SUMMARY

Riboflavin deficiency, even in a relatively early stage, greatly lowers the resistance of the rat to endemic typhus, thereby resulting in a fatal disease. Not only the serosal cells, but also the endothelial cells in several organs, notably the Kupfer cells of the liver, become greatly distended with rickettsiae under these conditions. Preliminary experiments strongly suggest that advanced vitamin A deficiency, even when making animals markedly cachetic, does not have a comparable effect. Riboflavin deficient animals remain alive for several weeks with an abnormal intracellular metabolism. This type of deficiency is worthy of further study as a possible method of approach in the investigation of other intracellular parasites and filtrable viruses. Speculation regarding the mechanism concerned also suggests the desirability of a study of the effects of this deficiency upon the production of immune bodies in general.

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PRELIMINARY NOTE ON THE MODE OF UNION OF THE GALACTURONIC RESIDUES IN PECTIC ACID

CONSIDERABLE progress has been made in recent years in the field of the chemistry of pectic acid, particularly through the investigations of Link and his co-workers. However, the essential points in its structure are still missing, namely, the exact position of the linkage of the individual residues of the galacturonic acid, their ring structure and the length of the chain (if pectic acid has a straight chain structure). Levene and Kreider recently limited the place of union of the individual residues to positions (4) or (5). The choice between the two has not yet been made.

For the purpose of obtaining the missing information, pectic acid has been exhaustively methylated. The product thus obtained had a composition corresponding most satisfactorily to a structure composed of approximately six units.

$C_{56}H_{90}O_{37}$.	Calculated.	OCH_3, 45.80
	Found.	OCH_3, 45.40

On saponification the substance had the following methoxyl value:

$C_{50}H_{78}O_{37}$.	Calculated.	OCH_3, 34.18
	Found.	OCH_3, 34.30

Whether the material is only a methylated fragment of pectic acid can as yet not be stated. The deduction as to the length of the chain of this material is tentative, inasmuch as it is based only on the methoxyl value in the fully methylated and in the saponified material. More precise information on this point is expected from the analysis of the methylated polygalactoside, obtained on reduction of the above material. This substance has now been prepared by heating the exhaustively methylated material with copper chromite catalyst in an atmosphere of hydrogen at a temperature of 175° and a pressure of 3,500 pounds per square inch during 6 hours. The product has been obtained in only a fair degree of purity, having the following composition:

$C_{56}H_{102}O_{31}$.	Calculated.	C 52.9, H 8.1, OCH_3, 48.81
	Found.	C 53.8, H 8.0, OCH_3, 47.33

As far as can be judged from the rate of hydrolysis of the fully methylated pectic acid, the galacturonic residues seem to have a furanose structure, for after 2½ hours heating of the product with 0.01 *N* hydrochloric acid, at 100° C., about 1½ equivalents of reducing groups are developed and after 15 hours, about 3 equivalents. The methyl ester of 2,3,4-trimethyl α-methyl-d-galacturonide under identical conditions remained unchanged. (R. S. Tipson.)

Thus, it seems suggestive that the galacturonic acid residues of pectic acid have a furanose structure and hence the union of the individual residues is through carbon atom (5).

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VITAMIN E AND NUTRITIONAL MUSCULAR DYSTROPHY

IN 1931 Goettsch and Pappenheimer[1] described a dietary deficiency disease in rabbits and guinea pigs characterized by dystrophy of the voluntary muscles. Morgulis and Spencer,[2] Morgulis, Wilder and Eppstein,[3] found that at least two factors, both contained in whole wheat germ, were required for the prevention or cure of the disease. One factor was removed from wheat germ, previously extracted with petroleum ether, by 70 per cent. ethanol. The other factor was found in the unsaponifiable fraction of wheat germ oil. This suggested that the fat-soluble factor might be vitamin E. However, the dystrophy producing diet apparently contains a significant amount of vitamin E, since both Goettsch and Pappenheimer[1] and Morgulis[4] found

[1] M. Goettsch and A. M. Pappenheimer, *Jour. Exp. Med.*, 54: 145, 1931.
[2] S. Morgulis and H. C. Spencer, *Jour. Nutrition*, 11: 573, 1936.
[3] S. Morgulis, V. M. Wilder and S. H. Eppstein, *Jour. Nutrition*, 16: 219, 1938.
[4] S. Morgulis, "Nutritional Muscular Dystrophy," Hermann and Cie., Paris, 1938.