Thiamin has a more marked accelerating effect in similar combinations, but the effect is just as evident in the cultures containing 1% oleic acid as in those containing .01%.

With .01% oleic acid and 0.5% asparagin and thiamin, good growth is obtained in 5-10 days. At this time flasks without thiamin show only a trace, but after 4 weeks both show heavy and apparently equal growth. These effects were observed when as little as 1 gamma of the vitamin was added to 50 cc of medium. On the other hand, amounts of pyridoxin and thiamin up to 2.5 mg per 50 cc were without effect in the absence of oleic acid.

Conclusions. The *Pityrosporum ovale* grows in the presence of inorganic salts, glucose and oleic acid. The addition of asparagin accelerates growth and increases the final yield.

Thiamin and pyridoxin accelerate development but are not essential to the growth of this fungus under the conditions studied.

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Non-Availability of Gum Acacia as a Glycogenic Foodstuff in the Rat.

J. VICTOR MONKE. (Introduced by John C. Krantz, Jr.)

From the Departments of Physiology and Pharmacology, School of Medicine, University of Maryland, Baltimore.

No biochemical analyses have been made to test whether the gum acacia molecule passes entirely undigested through the gastrointestinal tract. Observations on poorly controlled feeding experiments, cited in the older literature,[1] are reported to have shown that gum acacia, without other dietary supplement, is a deficient foodstuff. Since gum acacia is a rather important physiological tool,[1] any information concerning its chemistry or physiological properties is of some interest.

The question arose whether all of the sugar components of the molecule[2] are tightly bound into a "main" chain molecule, or whether some of them are possibly attached as "side" chains to the main molecule. In the latter case, they might be vulnerable to the digestive enzymes, and thus become materials available for glycogenesis.

1 Amberson, W. R., *Biol. Rev.*, 1937, **12**, 48.

2 Butler, C. L., and Cretcher, L. H., *J. Am. Chem. Soc.*, 1929, **51**, 1519.

Two groups of 15 young male rats (avg. wt. 140 g) were removed from a normal diet of dog mash, placed in individual cages, and fasted for 48 hours. Five individuals in each group were given 10 g of cacao butter, the remaining ten, 10 g of a mixture containing 34% of white powdered gum acacia (Arthur H. Thomas) and 66% of cacao butter. Tests for free reducing substances in the gum acacia were negative. At the end of 72 hours each rat was anesthetized with 0.5 cc of 10% sodium amytal administered intraperitoneally. The liver was immediately extirpated and placed in a tared 50 cc centrifuge tube containing 15 cc of 30% potassium hydroxide. Glycogen was obtained by means of Good's modification[3] of Pfluger's method. Glucose was determined upon an aliquot of the hydrolyzed glycogen according to the method of Shaffer and Hartman.[4]

The livers of the 2 control groups contained an average of 0.15% and 0.13% glycogen by weight, respectively, while those of the acacia-fed groups contained an average of 0.24% and 0.17% glycogen by weight, respectively. The variation in the percent of glycogen by weight between the livers of the individual rats in the control groups ranged from 0.03% to 0.29%, with a mean in each group of 0.12% and 0.17%, respectively. Variations in percent glycogen by weight in the livers of the acacia-fed groups ranged from 0.04% to 0.31% in the various individuals. These had a mean value for each group of 0.27% and 0.18%, respectively.

In previous papers reported by Krantz, *et al.*,[5, 6] it has been established that the assay of glycogenic activity on the part of a compound administered as it was in these experiments does not become significant unless the percentage increase has an average value of the order of 300% or more.

It is concluded that the difference in liver glycogen between the control and the acacia-fed rats is insignificant. No part of the gum acacia molecule is subject to disintegration by the enzymes of the digestive tract of the rat. Orally administered gum acacia is excreted unchanged in the feces.

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3 Good, C. A., Kraemer, H., and Somogyi, M., *J. Biol. Chem.*, 1933, **100**, 485.

4 Shaffer, P. A., and Hartmann, A. F., *J. Biol. Chem.*, 1920, **45**, 349.

5 Carr, C. J., Musser, R., Schmidt, J., and Krantz, J. C., Jr., *J. Biol. Chem.*, 1933, **102**, 721.

6 Krantz, J. C., Jr., Evans, W. C., and Carr, C. J., *Quart. J. Pharm. and Pharmacol.*, 1935, **8**, 213.