tend to confirm that this is the general rule since, except for the 2 instances mentioned above, there was no inhibition of glycogen breakdown in our animals.

The excessive gluconeogenesis in both phlorhizin and pancreatic diabetes, although caused by different mechanisms, nevertheless, leads to the same consequences, namely, continued glycosuria, low liver glycogen values, the accumulation of fat in the liver, ketosis and lowered R.Q. To this extent the two conditions are similar. The presence of insulin in the phlorhizinized animal, however, enables it to maintain the normal relationship between the height of the blood sugar level and the rate of entry of blood sugar into the tissues.[9, 18] This differs from the depancreatized animal, which requires a higher blood sugar level to allow a normal rate of entry.

Summary. At similar blood sugar levels, the phlorhizinized dog utilizes dextrose at the same rates as does the normal animal. The similarities and differences between phlorhizin diabetes and pancreatic diabetes are briefly discussed.

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Effect of Vitamin A Deficiency on the Rate of Apposition of Dentin.*

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Sixty albino rats placed on complete Vitamin-A-deficiency diet were given 2 intraperitoneal injections of 0.5 cc of a 2% solution of alizarine Red S (Table I). Alizarine is deposited as a red line in the dentin growing and calcifying at the time of each injection.[1] By measuring, in ground sections of incisors, the distance between the 2 injection effects and dividing this by the time interval daily rates of apposition were obtained.

The daily rate of normal dentin apposition averages 16 μ in both the labial and lingual portions of the tooth. In the experimental animals the rate of dentin apposition increased on the labial and

[18] Soskin, S., and Levine, R., *Am. J. Physiol.*, 1938, **123**, 192.

* Aided by a grant from Mead Johnson and Co.

[1] Schour, I., and Hoffman, M. M., PROC. SOC. EXP. BIOL. AND MED., 1938, **37**, 710.

TABLE I.
History of 60 Albino Rats on a Vitamin-A-Deficient Diet and the Daily Rate of Dentin Apposition in Their Incisor Teeth.

Group No.	No. of animals in group*	Age (days) when Alizarine Red S was injected	Replacement therapy and ages (days) at which started	Age in days at death	Avg rate of apposition per 24 hr in microns			
					Labial	Disto-lingual	Mesio-lingual	Mid-lingual
1	45	30, 40	None	45	16.14	14.38	13.76	13.42
		43, 54		73	16.79	13.11	12.48	10.86
		47, 57		77	17.91	12.85	12.13	9.57
		50, 60		65	18.68	11.14	10.75	7.84
		64, 72		77	19.63	11.29	9.01	6.43
2	8	47, 51	Suboptimum vitamin A replacement as blue gramma grass at 47 days	52	18.32	12.47	11.83	8.91
3	7	50, 60	Full replacement as cod-liver-oil at 50 days	75	16.12	15.78	15.76	15.81
		60, 70		75	15.97	15.83	15.80	15.91

*All rats were placed on vitamin-A-deficient diet at 21 days of age (weaning).

decelerated on the lingual portions in direct proportion to the duration of Vitamin-A deficiency (Fig. 1, Table I).

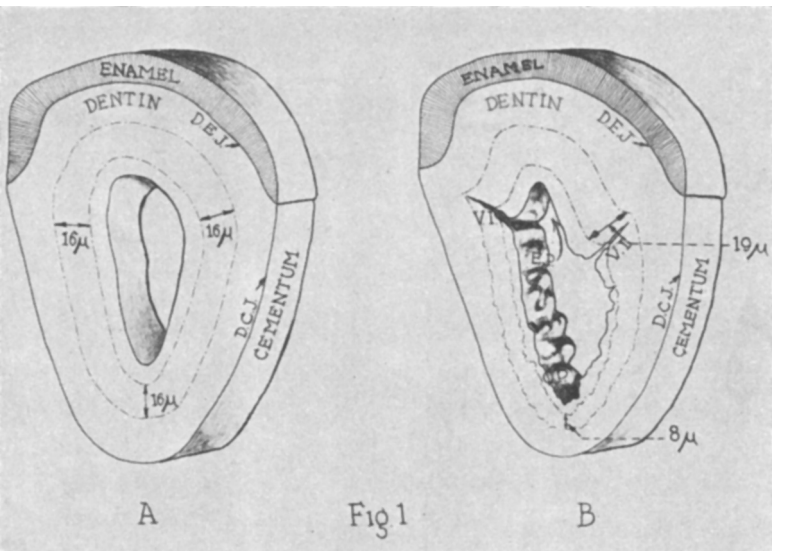

FIG. 1.

A. Tracing and reconstruction of a transverse ground section of the lower incisor taken at the level of the mental foramen of a normal control rat. This animal was injected with alizarine Red S on the 50th and 60th days of life and sacrificed on the 65th day. The alizarine effects (dotted lines) are parallel to the pulpal wall and dentino-enamel, D.E.J., and dentino-cemental, D.C.J. junctions. × 31.

B. Similar field of a rat with same history as in A but placed on vitamin-A deficiency when 21 days old. The enamel-covered dentin is increased and the cementum-covered dentin is decreased in width. The pulpal outline is distorted and narrow at E.P. and wide at C.P. V.I., vascular inclusions. Note alterations in daily appositional rate. × 37.

The disturbances in the rate of appositional growth are manifest between 9 and 19 days following institution of Vitamin-A deficiency (Table I), long before the cytologic and histologic changes become apparent.[2] The final width of the dentin is due primarily to the differences in the rate of growth.

Suboptimal replacement therapy of 4 days' duration did not produce a measurable change in appositional rate. However, full replacement therapy had an immediate effect, causing the normal rate of apposition to be resumed within a period of one to 5 days (Table I).

Summary. The rate of apposition of dentin is selectively altered in Vitamin-A deficiency, while the life span of the formative cells is

[2] Wolbach, S. B., and Howe, P. R., *Am. J. Path.*, 1933, **9**, 275.

not affected. The rate of apposition is accelerated in the enamel-covered portion and decelerated in the cementum-covered portion. The findings indicate the delicate response of the rate of dentin apposition to Vitamin-A deficiency and suggest the possible use of this reaction as a biologic method of measuring the Vitamin-A content of foods.

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A Pneumonia-Producing Filtrable Agent from Stock Mice.*

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A virus causing respiratory disease in stock mice has been reported by Dochez, Mills and Mulliken.[1] We have encountered a similar infectious agent, have made a preliminary pathological study of it, and have attempted to compare it with the Dochez virus and with other agents of disease found in stock mice. The chief purpose of this report, however, is to call attention to the ease with which the disease caused by this agent may be confused in mice with infection due to influenza virus.

Pharyngeal washings from patients with a variety of illnesses (common cold, atypical pneumonia, influenza-like illness) were inoculated intranasally into mice which were sacrificed on the sixth day. The lungs were removed aseptically, emulsified, and passed intranasally to normal mice which were in turn sacrificed, the series being continued indefinitely. As controls, serial passages were made from mice inoculated intranasally with pharyngeal washings from normal persons, with broth, and with normal mouse lungs. In every instance, after a varying number of transfers, lung lesions resembling those produced by influenza virus began to appear in the mice. Their initial appearance varied from the first to the ninth passage and they became maximum in 2 or 3 further passages, killing the mice usually on the third or fourth day. White mice purchased in the open market and weighing 5 to 10 g have been used exclusively, and lung

* This work was supported in part by a grant from the Fenger Memorial Fund.

1 Dochez, A. R., Mills, K. C., and Mulliken, B., PROC. SOC. EXP. BIOL. AND MED., 1937, **36**, 683.