On the basis of the assays, the purified diet contained traces of all amino acids studied except isoleucine, even though urea was the only nitrogen source added. The presence of these amino acids in the purified diet appears to be a slight protein contamination from the cornstarch, sugar, and lard. The rumen material contained 9 to 20 times more of the amino acids that the diet fed (Table 1).

From a calculation of the amounts of amino acids furnished by the daily ration it would appear that the losses in feces and urine considerably exceeded the dietary intake. Obviously, this could not be the case because the animals were storing nitrogen and gaining weight. If the amino acid content of the rumen material is used to estimate the amounts available to the animals, a retention of the amino acids is indicated, which would explain the ability of the animals to grow. The fact that the animals have continued to gain in weight on the urea diet, containing no protein, for over 3 months is further evidence of the formation of amino acids.

Similar studies were carried out using a purified diet containing glycine as the only source of nitrogen. Rumen samples from sheep fed the glycine diet again indicated synthesis of the amino acids but at a lower level.

Results of these experiments show that the ten essential amino acids are synthesized in large amounts in ruminants fed urea as the only dietary source of nitrogen.

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Experimental Amyloidosis in the Guinea Pig[1]

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In the course of a pathological study of chronic scurvy, we unexpectedly observed deposits of amyloid in various organs. So far as we know, this is the first report of experimental production of amyloidosis in guinea pigs.

Thirty young guinea pigs were fed scorbutogenic diets for varying lengths of time as shown in Table 1; 12 received the Sherman-La Mar diet as modified by Rinehart (*10*), and 18 the Crampton No. 5 diet (*3*). The control animals received 2.0 mg/day ascorbic acid, the acute scurvy group none, and the chronic scurvy group 0.2 mg/day. No amyloid was found either in the control or in the acute scurvy animals. Among the chronic scurvy guinea pigs, amyloid was demonstrated only in those animals which were sacrificed 8 weeks or longer after the beginning of the experiment. Six out of seven animals in this last group showed distinct amyloidosis. The lone exception was that of a guinea pig on the Crampton No. 5 diet, which was sacrificed at 19 weeks following a period of 4 weeks of unexplained clinical improvement.

TABLE 1

EFFECT OF DIET AND ASCORBIC ACID ON AMYLOID DEPOSITION IN GUINEA PIGS

Diet	Ascorbic acid supplement (mg/day)	Number of animals	Time sacrificed (weeks)	Pathological findings		
				Inanition	Scurvy	Amyloid
Rinehart	0	4	4–5	0	acute	0
	0.2	5	9–18	+	chronic	+ (in all 5)
	2.0	3	1–18	0	0	0
Crampton No. 5	0	7	4–5	0	acute	0
	0.2	2	8 and 14	+	chronic	+ (in both)
	0.2	5	1, 1, 2, 6, 19	+ (6–19 weeks)	+ (6–19 weeks)	0
	2.0	4	8–17	0	0	0

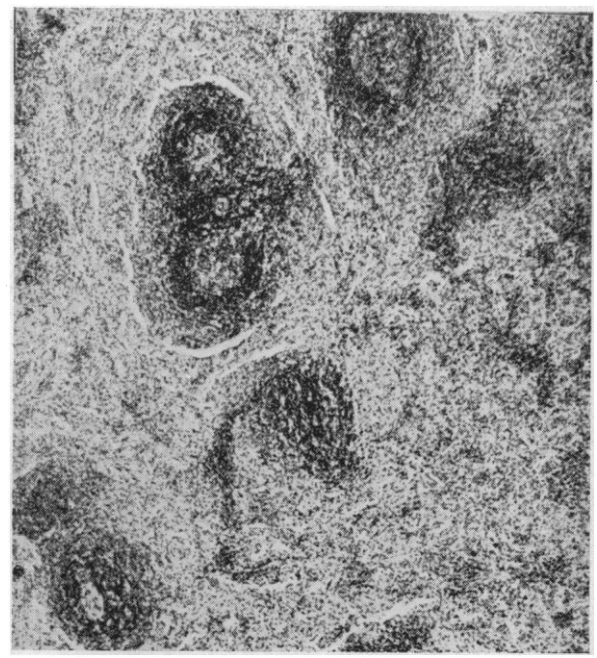

FIG. 1. Spleen from a control guinea pig (hematoxylin-eosin; × 65). Army Institute of Pathology.

[1] The opinions expressed in this paper are those of the authors and do not necessarily represent the official views of any governmental agency.

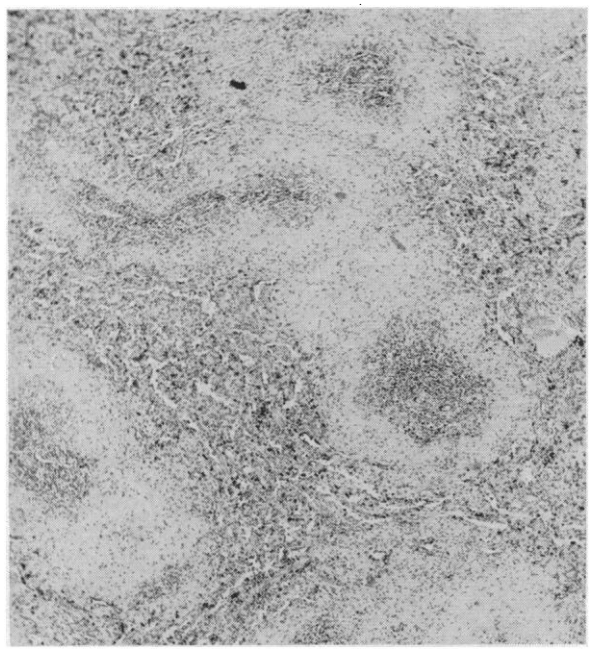

FIG. 2. Spleen from a guinea pig with chronic scurvy (hematoxylin-eosin ; × 48). Army Institute of Pathology.

In animals showing amyloid deposition, the spleen was severely involved, the liver moderately, and the adrenal cortex only minimally in a few cases. Amyloid was not found in organs other than those mentioned in the chronically scorbutic guinea pigs.

Microscopically, the amyloid appeared in the spleen in the form of a thick band in the peripheral portion of the Malpighian corpuscles, extending at times to a minor degree into the pulp. (Figs. 1 and 2.) The central portion of the corpuscles appeared normal. The pulp was relatively poor in cells and contained prominent sinusoids. In the liver, amyloid was noted in moderate amounts between the hepatic cords and the walls of the sinusoids. The peripheral two-thirds of the hepatic lobules were as a rule more severely affected, the hepatic cells in these areas appearing small, atrophic, and compressed by the amyloid material. No definite intracellular amyloid was noted in the spleen or in the liver. In the adrenal cortex amyloid was noted in small amounts, again in close apposition with, and at times completely surrounding, the walls of capillaries lying among the cortical cells.

Iodine and sulfuric acid tests on the gross specimens, and crystal violet and Congo red stains on the sections, were all positive. However, the tinctorial response of the amyloid, particularly with Congo red, was not as brilliant as is usually seen in pathological specimens from man. The results of the tests with special stains, together with the typical distribution of the amyloid in the immediate neighborhood of capillaries and of reticuloendothelial elements, is characteristic of this disease, in both man and experimental animal. The minor differences of the tinctorial response from that usually observed in man can probably be explained on the basis of age of deposition and molecular arrangement of the amyloid.

At the present time the exact chemical composition of amyloid is not known and the etiology and pathogenesis of amyloidosis are not clear. Experimentally, amyloidosis has been produced in mice, rats, hamsters, rabbits, dogs, and horses by various means (*1, 2, 4, 5, 7, 8, 9, 11, 13*). These include: supplementation of the diet with cheese; injection of bacteria, casein, sodium caseinate, pentose nucleotides, human serum, sulfur, selenium, or sodium silicate; inoculation with *Leishmania donovani;* and implantation of homologous tissues. Allegedly, amyloidosis has also been obtained in the experimental animal by injection of sodium bicarbonate, sodium hydroxide, hydrochloric acid, or animal tar (*12*). In addition, amyloid has been found in tumor-bearing mice (*6*).

Our observations do not warrant any positive conclusion, however attractive, as to the possible role of ascorbic acid or inanition, either alone or in combination in the pathogenesis of amyloid deposition. We feel, however, that these observations should be reported for two reasons: because to our knowledge, amyloidosis has never been detected in the guinea pig, and because amyloidosis has never been produced in animals by means of a deficient diet.

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Radiophosphorus and Radiostrontium in Mosquitoes. Preliminary Report

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The possibility of employing radioactive isotopes for marking mosquitoes for subsequent recognition has led to a study of behavior of P^{32} and Sr^{89} in colonized *Aëdes aegypti,* with a view to developing techniques by which such marking might be applied to large numbers of mosquitoes under field conditions. The usual methods of marking involve relatively drastic procedures, such as dusting and spraying the captive adult mosquitoes before

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