

proteins of the intestinal wall. Or (2) the amino acids are utilized while they are being absorbed, and thus the arginine would be incorporated into the protein of the intestinal wall without having been exposed to dilution.

Bloch's first explanation cannot account for the observation that orally administered methionine (labeled with S^{35}) shows the highest incorporation in the proteins of the intestinal mucosa, as reported by Tarver and Schmidt (2). Bloch's second explanation was tested in our laboratory by injecting labeled methionine *by way of the jugular vein*. (Bloch administered the amino acid orally.) Again the protein of the intestinal mucosa showed the highest isotope concentration. The same result is obtained when tyrosine (labeled with C^{14}) is injected intravenously.

The interesting finding that the intestinal mucosa has the highest specific activity is explained by us as being due to actual increased protein synthesis rather than to the conditions of the experiment. The intestinal wall secretes enzymes and mucous-proteins which are lost in enormous quantities (unlike other enzymes and proteins within the body). To compensate for this loss the intestinal wall may be more active in protein synthesis than other organs.

If this explanation is correct, then the pancreas should also show a high turnover rate; pancreatic juice contributes many enzymes needed for digestion. Actually Tarver and Schmidt (2) found that the pancreas has the second highest specific activity among the organs of animals treated with isotopic methionine.

References

1. BLOCH, K. *J. biol. Chem.*, 1946, 165, 469.
2. TARVER, H., and SCHMIDT, C. L. A. *J. biol. Chem.*, 1942, 146, 69.

Lipoid Substance in the Cells of Proximal Convoluted Tubules of the Kidneys of Young Rats on a Choline-deficient Diet

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Descriptions of the histological changes in the acute "hemorrhagic" kidney of young choline-deficient rats make little, if any, mention of the appearance in frozen sections of any demonstrable fat (1). In a series of such sections stained with Sudan IV, from kidneys of rats three to four weeks of age and weighing 35-45 grams, fatty droplets have been consistently observed in the cells of the proximal convoluted tubules. The animals were killed at daily intervals after being placed on a choline-deficient diet. These fatty changes were usually first seen on the third day, but in one instance were observed as early as the second day. The droplets of fat increased in size and number, reaching a maximum on the sixth or seventh day, when congestion, hemorrhage, and cortical necrosis were readily demonstrable.

The appearance of this fat may be secondary to degenerative changes in the tubular epithelium resulting from dietary lack of the lipotropic factor, choline; but the possibility that it

may be important in the production of the other lesions of the "hemorrhagic" kidney is being investigated. The fatty droplets are observed before the onset of congestion, necrosis, hemorrhage, or cast formation. It has already been demonstrated (3) that the lipase content of the proximal convoluted tubule is diminished or absent in such kidneys. Reduction of the ratio of the phospholipid fraction to the total lipid content has also been established (2). Further biochemical and histochemical investigations of the nature of the histologically demonstrable fat are being undertaken to determine its relationship, if any, to the kidney lesions which result from choline deficiency.

References

1. CHRISTENSEN, K. *Arch. Path.*, 1942 34, 633; COPELAND, D. H., and SALMON, W. D. *Amer. J. Path.*, 1946, 22, 1059; GYÖRGY, P., and GOLDBLATT, H. *J. exp. Med.*, 1940, 72, 1.
2. PATTERSON, JEAN M., and MCHENRY, E. W. *J. biol. Chem.*, 1944, 156, 265.
3. WACHSTEIN, M. *J. exp. Med.*, 1946, 84, 25.

Quick Decline of Orange Trees—A Virus Disease

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Recent results of experimental tests set up in southern California in June 1945 have shown that the highly destructive quick-decline disease of oranges is infectious, and these new results, now viewed in connection with other experiments, indicate that it is a virus disease. Quick decline, known only since 1939, is similar in many respects to the disease known in Brazil as *tristeza*, which, within the last 8 or 9 years, is reported to have almost completely destroyed all orange trees on sour orange root stocks in Argentina and Brazil. The quick-decline disease has been known to occur only on sweet orange varieties on sour orange root stock. Consequently, a test was made with 200 one-year-old healthy Valencia orange trees grown on sour orange stock. Live buds from quick-decline trees were placed in 100 of these healthy trees, and 100 trees were used as checks. Healthy buds were placed in 50 of the latter group, and 50 remained unbudded.

In September 1946 a few trees into which diseased buds had been inserted began to show symptoms. On November 11, 1946, 36 per cent of the trees in which diseased buds had been inserted showed symptoms; only 2 per cent showed disease in each of the two groups of the check trees. Since all these test trees were healthy trees that had originated in a non-diseased area and were planted in a quick-decline area in the open, the 2 per cent was presumably caused by natural infection.

A description of the transmission tests and of many other experiments planned for investigation of other phases of the quick-decline problem will be found in the April, October, and December 1946 numbers of *California Citrograph*. The more detailed evidence on which this preliminary statement is made will appear later.

The great preponderance of diseased trees in the inoculated