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.

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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.

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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

group as compared with the checks, together with much additional data which indicate that the disease is not due primarily to organisms, nutritional unbalance, or other factors, lead to the definite conclusion that it is of the nature of a virus disease.

A Quantitative Study of the Complement-Hemolysin Relation

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A precise spectrophotometric method employing the end-point of 50 per cent hemolysis (*2*) has been used to measure the changes in complement activity that occur when a standard suspension of sheep's erythrocytes is sensitized with graded amounts of a hemolytic antiserum. Complement was titrated without preliminary incubation, and the 50 per cent units were determined by the method of least squares. The latter were then plotted against the corresponding amounts of antiserum to give the curve of the complement-hemolysin relation (*5*). Analysis of the experimental findings indicated that this curve may be rectified by plotting the ratio x/y, as ordinate, against x, as abscissa, where x represents hemolytic antiserum, and y, the unit of complement. A graph of the results obtained in a

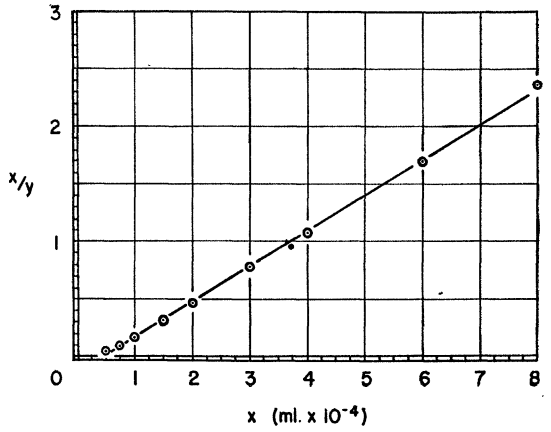

FIG. 1. Graph of x/y vs. x, where x represents hemolytic antiserum; y, the 50 per cent unit of complement.

representative experience is shown in Fig. 1. In fitting the line

$$(x/y) = bx + a \qquad (1)$$

to such data, the method of least squares was used to find values of b, the slope, and a, the intercept on the ordinate axis. For the example given, it was found that b = 0.308 and a = −0.137. To test the applicability of Equation 1 to the experimental points, the ratios x/y were calculated by substituting the predetermined values of a and b in the equation and allowing x to take on those used experimentally. The standard deviation of the observed from the calculated ratios was estimated (*3*) from

$$s'^2 = \Sigma\,[(x/y)_{observed} - (x/y)_{calculated}]^2/_{N-2}. \qquad (2)$$

The estimate, $s' \leqq 0.022$, was obtained for the results plotted in Fig. 1.

Solving Equation 1 for y gives

$$y = \frac{x}{bx + a}, \qquad (3)$$

which would express the curve of the complement-hemolysin relation. Vertical and horizontal asymptotes of this curve are demonstrable (*1a*), respectively, at

$$x = \frac{-a}{b} \text{ and } y = \frac{1}{b}.$$

If the axes are translated to a new origin at the point $(\frac{-a}{b}, \frac{1}{b})$, the equation assumes the standard form for a rectangular hyperbola referred to its asymptotes as axes (*1b*).

Curves based upon Equation 3 have fitted the experimental points over a wide range of hemolysin quantities but deviate somewhat when low concentrations of the reagent are used.[2]

TABLE 1

EFFECT OF HEMOLYSIN CONCENTRATION UPON THE CONSTANT, 1/n, IN VON KROGH'S ALTERNATION FORMULA

x Hemolytic antiserum (ml. × 10^{-4}).....	0.50	0.75	1.0	1.5	2.0	3.0	4.0	6.0	8.0
y 50% unit of complement (ml. × 10^{-3}) (calculated*)	29.41	7.98	5.85	4.62	4.18	3.81	3.65	3.51	3.44
1/n.....	0.366	0.354	0.254	0.221	0.199	0.175	0.166	0.180	0.189
Complement required for 99% hemolysis (ml. × 10^{-3}) (calculated†).....			18.79	12.76	10.43	8.51	7.83	8.03	8.20

* Values of y from Equation 3, where b = 0.308 and a = −0.137.
† From the Von Krogh formula (*4*).

It is of interest that the highest concentrations employed failed to produce an inhibition of complement activity such as has been ascribed to agglutination of erythrocytes by concentrated antiserum. Such inhibitions have, nevertheless, been observed occasionally in previous experiments, and Equation 3 would not be expected to express the complement-hemolysin relation in the affected range.

The data were also examined with regard to the effect of

[1] With the technical assistance of Helen M. Conway and Willie Mae Bodie.

[2] As a result of consultations with W. R. Thompson, of the Division of Laboratories and Research, New York State Department of Health, it appears that a better fit may be obtained with the more general form of rectangular hyperbola, $(x - x_0)(y - y_0) = k$, where x_0, y_0, and k are parametric constants. The application of this formula will be discussed in a more comprehensive report.