pended in aerated water in the dark. When the radicles were ten centimetres in length, one-millimetre portions were cut off and assayed for vitamin C using 2,6-dichlorophenolindophenol. The points shown in the graph represent means of forty-nine one-millimetre portions.

The graph shows a high concentration of vitamin C in the first portion. A fall in the vitamin concentration occurs in the second to the fourth portions, and this is followed by a rise in the seventh portion. Thus the region of maximum growth, as shown by Sachs's classical experiment, does contain a reasonably high quantity of the vitamin, as might be expected if vitamin C is of importance in growth.

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Role of Glutaminase in the Production of Urinary Ammonia

IF, as Van Slyke, Philips, Hamilton, Archibald, Futcher and Hiller[1] suggest, urinary ammonia is mainly produced in the kidney from glutamine, it is possible that changes which increase or decrease the amount of ammonia produced might increase or decrease the amount of renal glutaminase. Such adaptation, if it occurred, would support the view that glutaminase is in fact involved in the production of urinary ammonia.

This possibility was investigated by a quantitative study of L-glutaminase in the kidneys of rats rendered chronically acidotic or alkalotic. The animals were litter-mate female albino rats of the Wistar strain and weighed about 170 gm. at the beginning of the experiment. They were fed on a mixture of bread, casein, cane sugar, arachis oil, cod liver oil, dried yeast and salt mixture. They were divided into three groups, each of six animals, and were given to drink, ad libitum, either water or 0·05 N hydrochloric acid or 0·1 N sodium bicarbonate. At the end of three months, the average daily excretion per rat of ammonia (as ammonia–nitrogen) was 7·5, 13·8 and 2·9 mgm. respectively, while the average weight of the animals was still similar in the three groups.

The activity of glutaminase in each rat was then determined in the Warburg apparatus by the method of Krebs[2]. Slices of kidney (cortex and medulla) were incubated in the presence of 4 mgm. of L-glutamine for 30 min. and the ammonia produced estimated by a modification of the method of Van Slyke and Cullen[3]. The tissue slices used were dried and weighed at the end of the experiment. The ammonia produced in the control flasks (by kidney tissue in the absence of glutamine) was deducted from that produced by the same amount of tissue in the presence of glutamine.

Preliminary experiments had shown that there was no linear relationship between ammonia production and the amount of tissue. It was therefore decided to use four different weights of tissue from each animal. A curve was then constructed from the results with each animal, and from it was calculated the amounts of ammonia produced by four arbitrary

ACTIVITY OF L-GLUTAMINASE IN RAT KIDNEY SLICES

Mean intrapolated values ± S.E., from six rats in each group, expressed in μgm. ammonia-nitrogen produced in 30 min.

Dry wt. of tissue (mgm.)	Normal	Acidotic	Alkalotic
6	67 ± 2·8	125 ± 15·3	43 ± 4·1
8	87 ± 2·4	157 ± 17·5	66 ± 3·9
10	97 ± 3·0	181 ± 16·6	77 ± 4·2
12	101 ± 3·8	195 ± 15·9	81 ± 4·6

The differences between the means of any two groups are significant ($P < 0.01$).

dry weights of tissue : 6 mgm., 8 mgm., 10 mgm. and 12 mgm. The mean values for each group of animals, calculated in this way, are given in the accompanying table.

The results show that acidosis produced an increase in glutaminase, whereas alkalosis produced a decrease in the enzyme. These results support the hypothesis that L-glutaminase is concerned in the production of urinary ammonia.

The findings reported here contradict those of Handler, Bernheim and Bernheim[4], who found no difference in ammonia produced from glutamine by kidney slices of normal, acidotic or alkalotic rats. Inspection of their results, however, reveals that not only did they use only one rat of each sort, but also that the ammonia produced in the Warburg apparatus represents about 90 per cent hydrolysis of the glutamine. It is clear that differences in enzyme activity might easily not be revealed under these conditions.

Further work is proceeding on the effect of acidosis and alkalosis on the metabolism by the kidney of other amino-acids. So far, it has been shown that renal deamination of L-aspartic acid is unaffected by changes in the excretion of urinary ammonia. It will be of particular interest to study the effect on amino-acids which are oxidized by L-amino-acid oxidase, since Lotspeich and Pitts[5] have shown that intravenous infusion of these increased the excretion of ammonia in acidotic dogs more than the infusion of amino-acids which are not oxidized by this enzyme.

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Utilization of Vitamin A₁ by Freshwater Fish

WALD[1] has pointed out that freshwater fish contain vitamin A_2 whereas marine fish contain vitamin A_1. This was confirmed by Gillam[2] and Lederer et al.[3]. It is only migratory fish which contain a mixture of both vitamins A_1 and A_2.

Karrer et al.[4] suggested that vitamin A_2 is not utilized by mammals and may even be toxic to them.