U was supplied as 0.7 g of a fullers' earth eluate of the wheat bran extract. Pantothenic acid was supplied as 0.1 g of a charcoal eluate of the milk sugar concentrate. The results are shown in Table II. Rubidium exerted a slight and only temporary alleviating effect on potassium deficiency.

Discussion. A comparison of the potassium requirement of the rat, as reported in the literature, and that of the chick, as reported in this paper, indicates a greater sensitivity on the part of the latter to potassium deficiency. When rats were fed 0.037% potassium[2] and 0.033% potassium[4] they survived at least 112 days and 220 days respectively. The chicks, on the other hand, were all dead within 40 days when fed 0.076% potassium. At the same time chicks receiving adequate levels of potassium grew fairly well and were in good condition. Growth, however, was below that obtainable on our stock diet.

Summary. 1. At least 0.17 g of potassium per 100 g of diet was found necessary to secure approximately maximal growth of chicks under the conditions of these experiments. 2. More than 0.13 g of potassium per 100 g of diet was necessary to prevent heavy mortality. 3. Rubidium had a slight and only temporary alleviating effect on potassium deficiency.

11988

Formation of Pressor Amines in the Kidney.

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Holtz[1, 2] found that renal cortical extracts of different species were able to transform *l*-histidine, *l*-tyrosine and *l*-dopa (*l*-dihydroxyphenylalanine) into their corresponding amines. The presence of the amines was demonstrated by their effect on the blood pressure of a test cat. The action of the enzymes responsible for this decarboxylation could be demonstrated only under anaerobic conditions. Bing[3] showed that this process could also occur in the isolated kidney

[4] Osborne, T. B., and Mendel, L. B., *J. B. C.*, 1918, **34**, 131.

[1] Holtz, P., *Klin. Woch.*, 1937, **16**, 1561.

[2] Holtz, P., and Heise, R., *Arch. Exp. Path. und Pharm.*, 1939, **191**, 87.

[3] Bing, R. J., *Am. J. Physiol.*, 1941, March.

of the cat perfused *in vitro* with blood containing dopa. The injection of 5-6 cc of the perfusate into a test cat caused a rise in blood pressure of 120 mm Hg. The amount of the pressor substance produced, presumably hydroxytyramine, was found to vary inversely with the rate of blood flow through the kidney. Since these experiments suggested that renal hypertension is due to a similar transformation of amino acids by the ischemic kidney, experiments were performed to investigate (a) whether amino acids other than those examined by Holtz could be transformed into pressor substances by renal tissue extracts and by perfused kidneys, (b) whether the enzymes responsible for this process varied with different species, and (c) whether renin, a protein found in renal extracts[4] could transform dopa into hydroxytyramine.

For the perfusion and incubation experiments the technic described by one of us (R.J.B.) was used.[5] The perfusion fluid for the cat's kidney consisted of 120 cc of heparinized cat's blood; for the human kidney 120 cc of human blood containing 5% citrate were used. The lung was included in the perfusion circuit except in experiments on the human kidney. Cats anesthetized with nembutal (39 mg per kg body weight intraperitoneally) were used to ascertain the effect of the protein-free extracts and the kidney perfusates on the blood pressure. All samples were injected intravenously.

In 21 incubation experiments *l*-dopa,* thyroxine,† *d-l*-phenylalanine and *l*-tryptophane were incubated under nitrogen for 12-16 hours at 38° C with extracts from the renal cortex of the guinea pig, cat and man. An equal amount of the amino acids was incubated with the kidney extracts under oxygen. As seen in Table I, the anaerobic incubation of dopa with renal cortical extracts of these 3 species gave rise to a pressor substance. Both the height and duration of the response were enhanced by the previous injection of cocaine hydrochloride (6 mg per kg body weight intravenously) in the experiments on guinea pig's renal extracts. In experiments on extracts of the kidney of the cat, cocaine caused a slight decrease in the height of the blood pressure response, but usually increased its duration. The work of Holtz makes it probable that this pressor substance is identical with hydroxytyramine. The injection of 6 mg of dopa dissolved in Ringer's solution had no

[4] Helmer, O. M., and Page, I. H., *J. Biol. Chem.*, 1939, **127**, 757.

[5] Bing, R. J., *Am. J. Physiol.*, 1941, in press.

* Hoffman-LaRoche.

† Squibb, Thyroxin Crystals. Natural form possibly converted to racemic mixture by crystallization.

TABLE I.
Incubation of Renal Extracts under Nitrogen.

Species	Amino-acid	Mg amino-acid used	No. exp.	Cc concentrate inj.*	Rise in blood pressure* Before cocaine hydrochloride	Rise in blood pressure* After cocaine hydrochloride
Guinea pig	*l*-dopa	125	6	1	90	145
" "	thyroxine	10	1	2.5	0	
" "	*d-l*-phenylalanine	35	3	2.3	100	35
Cat	*l*-dopa	5	4	1	70	65
"	*d-l*-phenylalanine	35	1	2	0	
"	*l*-tryptophane	20	2	2	0	0
Human	*l*-dopa	35	1	2	110	
"	"	5	1	2	80	

*Typical experiment.

effect on the blood pressure. A pressor substance also originated during the anaerobic incubation of phenylalanine with renal cortical extracts of the guinea pig. Its pressor effect was decreased after the previous injection of cocaine. During the anaerobic incubation of extracts of phenylalanine with cat's kidneys, however, no pressor substance was formed. Protein-free extracts obtained from guinea pig's kidneys incubated with thyroxine and of cat's kidneys with tryptophane had no effect on the blood pressure of the test cat.

Holtz showed that an enzymatic de-amination of hydroxytyramine occurred when the incubation of dopa and renal extracts was carried out under oxygen.[2] Extracts of the cat's kidney, however, incubated under oxygen with 5 mg of dopa usually gave a pressor response, but the rise in blood pressure was always smaller than that caused by the anaerobically incubated extract.

In the perfusion experiments, cat and human kidneys were perfused with reduced blood flow. After removing a sample of blood to serve as a control, 240 mg of dopa were added to the perfusate and perfusion was continued for 2 hours. The effect of the injection of 3.5-5 cc of the perfused plasma on the blood pressure of the test cat was compared with that produced by an equal amount of control plasma. The control samples had no effect. As shown in Table II, dopa was converted into a pressor substance, presumably hydroxytyramine, by both the perfused human and cat's kidneys. No pressor substance was detected in plasma perfused through the cat's kidney with phenylalanine.

Since there was a possibility that the enzyme responsible for the decarboxylation was renin, 1 cc of renin solution‡ was incubated in

‡ Kindly supplied by Dr. I. H. Page of the Lilly Laboratory, Indianapolis.

TABLE II.
Perfusion Experiments.

Species	Amino-acid	Reduction in blood flow (cc/min)*	Cc plasma inj.†	Rise in blood pressure† (mm Hg) Before cocaine hydrochloride	Rise in blood pressure† (mm Hg) After cocaine hydrochloride
Cat	*l*-dopa	12	5	120	140
"	"	5	5	30	40
"	*d-l*-phenylalanine	5	4	0	
Human	*l*-dopa	20	3.5	157	
" ‡	"	20	4	30	35

*Average value before reduction of flow in the cat was 17.5 cc per min and in the human 40 cc per min.

†Typical experiment.

‡Kidney obtained 12 hours postmortem. Destruction of the decarboxylating enzyme by autolysis probably explains the small yield of pressor substance.

one experiment for 10 minutes, in another for 2 hours with 35 mg of dopa in 50 cc of phosphate buffer under nitrogen. After incubation, the renin was destroyed by heating. Neither preparation gave a pressor response when injected into a test cat. It can be concluded, therefore, that renin was unable to convert dopa into hydroxytyramine. Incubation of 0.5 cc of renin and 25 mg of dopa in 5 cc of Ringer's solution with 11 cc of serum, which contains renin activator,[6] also failed to produce hydroxytyramine.

Discussion. It is shown that during anaerobic incubation of the extracts of guinea pig kidneys with either dopa or phenylalanine, substances originate which raise the blood pressure of the anesthetized cat. It can be assumed on the basis of the work by Holtz, that these substances are amines produced by the decarboxylation of dopa or phenylalanine. Extracts prepared after the incubation of renal cortical extracts of the cat with tryptophane and of the guinea pig with thyroxine have no effect on the blood pressure of the cat. This observation confirms the conclusion of Holtz that the decarboxylases are specific for certain amino acids.[7] Both anaerobic incubation and perfusion of human kidneys with dopa give rise to a pressor substance, demonstrating that the human kidney contains a dopa decarboxylase.

In experiments in which extracts of cat's kidneys are anaerobically incubated with phenylalanine no pressor substance is formed, indicating that, in contrast to the guinea pig's kidney, that of the cat

[6] Kohlstaedt, K. G., Helmer, O. M., and Page, I. H., PROC. SOC. EXP. BIOL. AND MED., 1938, **39**, 214.

[7] Holtz, P., Credner, K., and Walter, H., *Z. f. physiol. Chem.*, 1939, **262**, 111.

probably does not contain a phenylalanine decarboxylase. Experiments in which the cat's kidney is perfused with blood containing phenylalanine confirm these results. These experiments demonstrate that the decarboxylating enzyme responsible for the transformation of phenylalanine into its corresponding amine is species specific.

Summary. The decarboxylating enzymes contained in the kidney are specific for certain amino acids and vary with the species.

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11989

Quantitative Spectroscopic Analysis of Stem "Tracheal" Fluids for Inorganic Constituents.*

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Many workers have reported quantitative spectrographic methods for the determination of major and trace elements necessary for plant growth. The major plant nutrition studies by spectrographic analysis have concerned either leaf tissue analysis or plant ash analysis Heretofore, the general practice consisted of volatilizing a homogenized ash in a 220 volt d-c arc with a current of 9-12 amperes. Estimations of the amounts of the constituents present have usually been carried out either by employing standard powders according to Nitchie[1] for comparison purposes, or by using suitable internal standards and determining the ratios of certain emission lines of the unknown to the suitable emission spectra of an internal standard as outlined by Gerlach.[2] Studies of ashed leaf tissue have furnished data concerning the number and respective amounts of the soil

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1 Nitchie, C. C., *Ind. and Eng. Chem.* Analyt. Ed., 1929, **1**, 1.

2 Gerlach and Schweitzer, *Foundations and Methods of Chemical Analysis by the Emission Spectrum.*