might have accumulated during their preparation. Chemical estimations were made of the lactic acid content of the tissues and medium at the beginning and end of the experiments to determine whether this substance could have served as a substrate. The respiratory measurements were made in Ringer-phosphate solution of pH 7.4, to which glucose and IAA were added as desired. Glycolysis was measured manometrically and chemically in Ringer-bicarbonate solutions exposed to an atmosphere of 95 per cent. nitrogen-5 per cent. carbon dioxide. Concentrations of neutralized IAA were chosen which abolished all but minimal amounts of glycolysis, yet allowed a satisfactory maintenance of respiration. The results of typical experiments have been assembled in Table 1.

To judge from the R.Q.'s obtained with brain and all three types of mammalian muscle, oxidation of carbohydrate appears to take place as readily in the presence of sufficient IAA to inhibit glycolysis as in its absence. Furthermore, not only is preformed carbohydrate oxidized under these conditions, but added glucose is as effective in elevating the R.Q. in the presence of IAA as in the controls. Chemical analyses showed that preformed lactate was not responsible for these elevated quotients.

These results are in accord with those of Stannard and of Saslow on frog muscle. The disagreement between our results and those of Meyerhof and Boyland, of Krebs and of Quastel and Wheatley may be due to the greater concentrations which these other investigators employed. It may well be that toxic effects were produced by the higher concentrations quite apart from the specific influence of the poison on cell metabolism. Furthermore, Krebs and Quastel and Wheatley depended entirely on measurements of oxygen consumption to judge of inhibition of carbohydrate oxidation. The effect of lactate on respiration and the R.Q. of IAA poisoned tissue, pointed out by the early workers, can readily be confirmed, but much of the significance of this observation would be lost in view of the persistence of the capacity to burn glucose under the same conditions.

Our experiments lend no particular support either to the theory that carbohydrate must go through a lactic stage before it can be oxidized, or to the unitary theory of oxidation and fermentation. Carbohydrate oxidation, at least under these conditions, appears to be independent of the anaerobic mechanism. Nor does IAA specifically affect the phosphorylating process, if esterification is a necessary preliminary to glucose oxidation. The mechanism of the specific action of IAA on intermediary carbohydrate metabolism warrants further investigation in the light of these findings.

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TABLE 1

Tissue	Respiratory Quotient				Oxygen Consumption				Inhibition of Glycolysis	Concentration IAA
	Non-nutrient	N.N. IAA	Glucose 0.2 per cent.	Glucose IAA	Non-nutrient	N.N. IAA	Glucose 0.2 per cent.	Glucose IAA		
					cu mm/moist mg/hr.				per cent.	
Skeletal muscle (dog)	0.86	0.89			0.31	0.21			94*	1/10,000
	0.94		1.03	0.97	0.26		0.26	0.17	98*	1/10,000
Smooth muscle (cat)	0.85		0.95	0.95	0.30		0.34	0.26	90†	1/10,000
	0.95		1.05	1.01	0.25		0.24	0.23	94†	1/10,000
Heart (cat)	0.87		0.96	0.99	0.33		0.37	0.37	90*	1/100,000
Heart (dog)	0.83	0.81	0.91	0.90	0.60	0.58	0.61	0.58	99*	1/75,000
Brain gray matter (cat)	0.97	0.98	0.97	1.12	0.42	0.35	0.66	0.44	97*	1/100,000
	1.06	1.09	1.02	0.96	0.34	0.30	0.61	0.47	94*	1/100,000

* Lactic acid determined chemically. † Lactic acid estimated manometrically.

CRYSTALLINE FACTOR I

OF the five known components of the vitamin B complex, three of them—thiamine, riboflavin and nicotinic acid—have been available in crystalline form. The other two,[1] factor 1 and factor 2, have so far been known as extracts. Factor 1 has now been crystallized by a procedure to be described elsewhere. The crystals are colorless rods which aggregate mostly as rosettes and sometimes in fan shapes. The dry crystalline material has a very slight yellowish tinge which is probably due to a slight amount of impurity. Dermatitis of the peripheral parts of the body of rats on factor 1-deficiency diets[1] involving the feet, paws, ears and areas around the mouth[2] is promptly cured with a daily dose of 10 micrograms of crystalline factor 1; 5 micrograms daily will clear up the dermatitis somewhat more slowly. Rats on factor 1-free diets, in which growth had ceased, responded immediately with gains in weight on administration of crystalline fac-

[1] S. Lepkovsky, T. H. Jukes and M. E. Krause, *Jour. Biol. Chem.*, 115, 557, 1936.

[2] T. W. Birch, P. György and L. J. Harris, *Biochem. Jour.*, 19, 2830, 1935.

tor 1. The potency of this preparation of factor 1 as measured by such gains in weight is given in Table I.

TABLE I
WEIGHT INCREASE OF RATS FED CRYSTALLINE FACTOR 1 FOR 14 DAYS

Daily intake of factor 1 Micrograms	Average daily gain in weight Grams
25.0	3.4
20.0	3.4
10.0	3.4
5.0	2.5
2.5	2.4

An independent check of the potency of crystalline factor 1 was made by Mrs. M. K. Dimick at the Biological Laboratory of the Vitab Products, Inc., with similar results. In this experiment, rats which had ceased to grow on the factor 1-deficient diet,[1] gained an average of 3 grams daily when fed 10 micrograms of crystalline factor 1 daily; and when 5 micrograms were fed daily, the rats made an average daily gain of 2 grams. The factor 1-deficient diets were similar in composition,[1] the only difference being in the factor 2 concentrate, a liver filtrate being used by Mrs. Dimick, and a rice bran filtrate, in the laboratory of the Poultry Division.

This work was greatly facilitated by a financial grant from Eli Lilly and Co.; by crude factor 1 concentrates made for us by Eli Lilly and Co., and the Vitab Products, Inc.; and by materials and personnel from the WPA (project No. 8261).

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ANEURIN AND THE ROOTING OF CUTTINGS[1]

WITH the increase of our knowledge concerning the principles of plant growth and development, it has been possible to solve some interesting practical problems by a purely scientific approach. Thus the discoveries, in rapid succession, of the growth hormone (auxin), of its chemical nature, of the identity of the growth hormone with one of the hormones of root formation and of the growth hormone activity of indole derivatives have led to a number of practical applications, particularly in the rooting of cuttings.[2] It soon became clear, however, that root initiation and subsequent root growth are conditioned by a complex set of factors of which auxin, although of great importance, is but one. Sugar and biotin were soon recognized as additional factors in root formation (Went and Thimann[3]). An independent line of research has led to the recognition of aneurin (vitamin B_1) as a hormone of root growth.[4] Under normal conditions the extremely small amounts of aneurin which are required for root growth (as judged by the amounts required by roots *in vitro*) are supplied by other parts of the plant. Without aneurin, or derivatives of it, no root growth is possible. This consideration has led us to expect that under certain conditions aneurin should be the limiting factor for root development on cuttings. That this is actually the case is shown by the following experiments: etiolated pea stems were treated basally for ca 20 hours with indole acetic acid (20–200 mgs per liter), and were then transferred to bottles containing 5 cc of 2 per cent. sucrose solution. At different times after the auxin treatment, aneurin, over a wide range of concentrations, was added to the sugar solutions for different periods of time. With the exception of the highest concentrations (100–20 mgs per liter) applied soon after the auxin treatment, all the aneurin treatments caused a marked increase in the number and size of the visible roots. Concentrations of 1 mg per liter or lower, applied 5 to 9 days after the auxin treatment, gave the most vigorous response, which amounted to several hundred per cent. more visible roots than in the non-aneurin treated controls. Histological investigations show that 5 days after the above auxin treatment large numbers of root primordia have been formed. The number of these primordia which grow out is, as shown by the above experiments, limited by the available aneurin. That it is not root initiation which is primarily affected by aneurin is shown by the fact that aneurin without previous auxin treatment is without influence upon the root formation of these cuttings.

A number of other experiments were carried out under practical nursery conditions. Leafy lemon cuttings were treated overnight with indole acetic acid (200 mgs per liter) and were then allowed to stand for one week in sand in a propagating frame. The bases of these cuttings were then placed for 24 hours either in water or in an aneurin solution (1 mg per liter). Thirteen days later the control cuttings, after-treated with water, had 8.1 roots apiece, while the aneurin-treated plants had 16.3 roots each. These roots were in addition much longer than those of the water after-treated cuttings. The controls which were not treated with indole acetic acid had only 0.3 roots per cutting. Still more striking was an experiment with leafy Camellia cuttings, which are notably slow in rooting. After repeated indole acetic acid treatment (200 mgs per liter for 20 hours each time), 30 out of 200 cuttings

[1] Report of work carried out under the auspices of the Works Progress Administration, Official Project Number 165-03-6999, Work Project Number 6330-6989.

[2] These discoveries are sometimes erroneously attributed to the investigators of the Boyce Thompson Institute.

[3] F. W. Went and K. V. Thimann, "Phytohormones," New York, 1937.

[4] J. Bonner, SCIENCE, 85: 183, 1937; W. Robbins and M. Bartley, SCIENCE, 85: 246, 1937.