

THE experiments of Heyroth and Loofbourow have established a close correlation between the physiological activity of concentrates of vitamin B₁ (after eliminating inactive purines and pyrimidines, which absorb in the same spectral region), and the intensity of the absorption band at 2600 Å. They have thus provided a welcome confirmation of the proof which we gave in our letter to NATURE of May 14, that vitamin B₁ is characterised by an absorption covering the mercury line at 2537 Å. The detection in their concentrates of inactive substances which absorb light of similar wave-length is of great value as a guide to the conditions under which the intensity of the band may be used as a measure of the concentration of the vitamin; but the identification of the characteristic band by the method of monochromatic irradiation was independent of the presence or absence of these impurities in the apparently homogeneous crystals which were used in our experiments.

In general, a molecule cannot be destroyed by light which it does not absorb. The only exception to this rule is provided by the phenomenon of photosensitisation. Thus, if light is absorbed by alien molecules of type A, giving rise to chemically active products, such as atoms of chlorine, it may happen that molecules of type B can be destroyed indirectly by the secondary chemical changes which these products are able to effect. This phenomenon, however, depends on a series of contingencies which we regard as unlikely, especially with so stable a vitamin. Moreover, an additional coincidence would be required to account for the parallel effects produced by irradiation and by the action of alkali, as described below.

If this indirect mechanism is excluded, the destruction of physiological activity which resulted from irradiation with the mercury line 2537 Å. proves that light of this wave-length is absorbed selectively by the vitamin and that the absorption band at this wave-length is photochemically active.

The correlation of this band with the biological activity of the vitamin was confirmed by experiments in which both were destroyed by the action of alkali; but this action is less specific than that of monochromatic light, and was therefore cited only as collateral evidence in support of the more rigid proof which was made possible by the method of monochromatic irradiation.

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Potency of Vitamin B₁ Preparations

RECENTLY (1932),¹ we have advanced indirect evidence for the belief that crystalline specimens of vitamin B₁ from baker's yeast, prepared by our methods, were more potent than those of Windaus, Tschesche *et al.* (1932).² Owing to their courtesy, we have been able to confirm this by direct test. Comparative tests upon pigeons (by curative method) have been made. As birds developed characteristic symptoms, they have been given alternately by mouth, approximately 14 γ (0.014 mgm.) of each preparation. (Results by mouth are usually 30 per cent. lower than by injection.) The results were as follows:

Prep.	No. of birds	Dose given.	Average day dose	Standard error of mean	Vitamin B ₁ units/mgm. ²
G. (Windaus, Tschesche <i>et al.</i>)	10	14.2 γ	4.31 γ	0.63	279
	8	23-28 γ	4.58 γ	0.41	262
E. (our own)	10	13.8 γ	2.56 γ	0.35	469

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The mean difference in potency $G : E$ is 1 : 1.75; that is, ours proved to be one and three quarter times as potent. Tested by the usual statistical formula, this difference would occur by chance less than once in fifty trials. It is supported by the tests upon larger doses, and also by reckoning the percentage cures with 14 γ dose. For G we have 10/13 cured, and for E 10/10. As we have found it possible to fractionate our crystals still further, we have no hesitation in concluding that more potent vitamin B₁ can be prepared than preparation G , and that this cannot be therefore pure vitamin B₁.

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* 12 mgm. Janson acid clay = 1 pigeon dose.

¹ *J. Physiol.*, **76**, 1, 1932.

² Windaus, Tschesche, Ruhkopf, Laquer and Schultz, *Z. physiol. Chem.*, **204**, 123, 1932.

A Growth-Stimulating Substance in Fatigued Muscle

MUSCULAR exercise results in development of the muscles concerned, and it influences other parts of the body. A metabolic product of muscular activity is probably responsible for the muscle hypertrophy, and it is possible that such a hormone may circulate in the blood and stimulate other organs. To our knowledge the only experimental study of this question is that of Bělehrádek¹, who fed tadpoles with artificially fatigued frog muscle. The weight of these tadpoles was increased by 28 per cent compared with controls fed on resting frog muscle, and they metamorphosed earlier than the controls. This has been confirmed by Siebert and Petow.²

We have extended this work by feeding blow-fly larvæ with frog muscle fatigued by electrical stimulation through the nerve. In 16 out of 18 experiments the larvæ fed on fatigued muscle grew larger than those fed on resting muscle, the average excess weight being 9 per cent. The larvæ fed on fatigued muscle did not metamorphose earlier than the controls, and their oxygen consumption was unchanged; but the rate of heart beat of the former exceeded that of the latter by 14 per cent.

Work is now being continued on the substances responsible for this growth stimulation and on their mode of action.

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¹ *Arch. Int. Physiol.*, **22**, 376, 1924.
² *Z. klin. Med.*, **102**, 434, 1925.

Limiting Mobilities of Some Monovalent Ions and the Dissociation Constant of Acetic Acid at 25°

VOGEL and Jeffery in a recent letter,¹ with the same heading as the above, have directed attention to the fact that we omitted, in our recent paper,² to mention that they have published³ different figures from ours for the limiting mobilities of ions. Though we should, possibly, have referred to their work in that particular connexion, it was not ignored. The conductance measurements of these authors and the