



ferrous hydroxide reduction of *d*-urobilin are suitable for the racemization of the pigment, and this would explain the formation of *i*-urobilin in Watson's experiments.

d-Urobilin IX α also undergoes racemization in alkali. It is possible that *i*-urobilin, prepared from mesobilirubinogen formed by amalgam reduction of bilirubin, is a true meso compound, but the racemized pigment must contain equal quantities of enantiomorphs. It should therefore be possible to obtain, in addition to the above pigments, a levorotatory urobilin IX α which is not identical with stercobilin as well as an *l*-dehydro-urobilin IX α which is enantiomorphous with *d*-urobilin.

The quantitative hydrogenations were kindly carried out by Miss J. Cuckney, of the Micro-analytical Department of the Imperial College of Science and Technology.

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¹ Gray, C. H., and Nicholson, D. C., *Nature*, **180**, 336 (1957).

² Lowry, P. T., Ziegler, N. B., and Watson, C. J., *Bull. Univ. Minn. Hosp. and Med. Found.*, **24**, 166 (1952).

³ Gray, C. H., Nicholson, D. C., and Nicolaus, R. A., *Nature*, **181**, 183 (1958).

Excretion of 17-Ketogenic Steroids in Subjects with Complete Transverse Lesions of the Spinal Cord

ELEVATION of the blood adrenaline-level, whether by intravenous infusion or in response to stress stimuli, leads to an increase in the concentration of adrenocortical hormones in blood¹⁻⁴. It is believed that this increase is mediated by the anterior hypophysis, which responds to the increased blood adrenaline levels by releasing adrenocorticotrophic hormone.

The peripheral blood contains adrenaline, though in very low concentrations, even in the resting state. It has been shown that subjects with complete transverse lesions of the cervical cord have blood adrenaline-levels which are significantly lower than those of normal subjects⁵. More recent work in this Centre has shown that subjects with complete transverse lesions above fifth thoracic vertebra (*T*.5), that is, subjects whose adrenal medullae are isolated from the control of the higher nervous centres, also show low blood adrenaline-levels.

It was of some interest, therefore, to determine the rate of excretion of adrenocortical hormones in the urine of subjects with complete transverse lesions of the spinal cord above *T*.5. The adrenocortical hormones determined were the 17-ketogenic steroids.

The 24-hr. excretion of 17-ketogenic steroids was determined on 16 male subjects with complete transverse lesions of the spinal cord above *T*.5, and on a comparable group of 17 male subjects with transverse spinal cord lesions complete below *T*.5.

The results are summarized in Table 1.

Table 1. 24-HR. 17-KETOGENIC STEROID EXCRETIONS

Subjects	No. of determinations	Mgm. 17-ketogenic steroids excreted/day (mean \pm S.D.)
Normals (from Norymberski <i>et al.</i> (ref. 6))	8	13.15 \pm 3.48
Subjects with lesions below <i>T</i> .5	17	14.0 \pm 3.19
Subjects with lesions above <i>T</i> .5	16	5.8 \pm 2.26

For the purpose of statistical analysis, the results of excretion of 17-ketogenic steroids by normal men obtained by Norymberski *et al.*⁶ have been used. The results obtained from subjects with lesions complete above *T*.5 are significantly lower ($P < 0.01$) than results from subjects with lesions complete below *T*.5 and than from results from normal subjects; there is no significant difference between the results from normal subjects and those from subjects with lesions below *T*.5.

The results are in harmony with the view that adrenaline is concerned in the release of adrenocortical steroids not only in stress conditions but also under resting conditions.

Studies are at present in progress on the plasma 17 α -hydroxycorticosterone-levels in subjects with spinal cord lesions. The results of these studies, together with those reported above, will be published *in extenso* at a later date.

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¹ Vogt, M., *J. Physiol.*, **103**, 317 (1944).

² Long, C. N. H., and Fry, E. G., *Proc. Soc. Exp. Biol.*, N.Y., **59**, 67 (1948).

³ Pickford, M., and Vogt, M., *J. Physiol.*, **112**, 133 (1951).

⁴ Long, C. N. H., in "Symposium on Shock" (U.S. Army Medical Service Graduate School, Washington, 1951).

⁵ Robinson, R., and Munro, A. F., *Acta Physiol. Scand.* (in the press).

⁶ Norymberski, J. K., Stubbs, R. D., and West, H. F., *Lancet*, **i**, 1276 (1953).

Isolation of a New Flavin produced by an Actinomycete

SINCE the discovery that riboflavin is produced by *Eremothecium ashbyii* and *Ashbya gossypii*¹, a great number of bacteria and fungi have been found capable of producing this and other closely related substances². Among the *Actinomycetales* only a few species of *Mycobacterium*³ have been reported capable of producing riboflavin, while no true Actinomycete has ever been found to possess such a capacity.

During the isolation of an antibiotic produced by *Streptomyces* 476, a still unidentified species probably belonging to the *griseus* group, a considerable yellow-green fluorescence of the metabolic liquids was noted. The riboflavin potency of such liquids was assayed with mutant strains of *Lactobacillus casei* and *Leuconostoc mesenteroids*, giving an activity corresponding to a riboflavin content of 0.9–1.2 μ gm./ml.

The fluorescent pigments could be easily extracted with *n*-butanol and purified by absorption on charcoal and elution with aqueous pyridine. Paper chromatography of such preparations and of the original brews were then run in a *n*-butanol/acetic acid/water system according to Crammer's technique⁴. The