

1 mg/kg of reserpine, respectively. All the doses proved to be lethal for the dogs, and the 0.5 and 1 mg/kg doses were lethal for the majority of the rats.

The daily urinary excretion of 5-HIAA in the experimental animals is shown in the Tables I and II. In parentheses is the 5-HIAA content per ml of urine (in μg).

SHORE *et al.*¹, ERSPAMER⁵, and FISCHER and LECOMTE⁶ observed that the rate of excretion of 5-HIAA markedly increased during the first hours following the administration of high doses of reserpine (2–5 mg/kg, intraperitoneally). This was now confirmed in dogs and in group B of rats.

It clearly appears, however, from the tabulated data that, after this initial increase in the 5-HIAA urinary output, the daily excretion of the metabolite of 5-HT returns to normal values, in spite of the continuous administration of reserpine and the persistent very low levels of 5-HT in serum and spleen tissue of reserpine treated rats. An apparent, unexplained exception is group B of rats in which the urinary excretion of 5-HIAA remained constantly above the normal levels.

Present results show that reserpine, even in lethal doses, does not appreciably interfere in the biosynthesis of 5-HT in the dog and rat organism. This is in accordance with the observations of HAVERBACK *et al.*⁷, who found that reserpine, in a dosage known to lower platelet 5-HT, did not change, in normal human subjects, the excretion of the 5-HT metabolite.

We can conclude that the only hitherto demonstrated action of reserpine on 5-HT is that of causing a more or less conspicuous liberation of the amine from some body depots.

5-HT creatinine sulphate and 5-HIAA were synthesized in the Farmitalia Research Laboratories, Milan.

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Zusammenfassung

Wiederholte intraperitoneale Reserpidosen (0.1–1 mg/kg), die bei vielen Tieren tödlich wirken, sind beim Hund und bei der Ratte kaum imstande, die Biosynthese des 5-Oxytryptamins (Enteramin) zu beeinflussen, obwohl sie eine erhebliche Ausschüttung der Substanz aus einigen Körperdepots verursachen können.

⁵ V. ERSPAMER, *Exper.* 12, 63 (1956); *Naturwissenschaften* 43, 61 (1956).

⁶ P. FISCHER and J. LECOMTE, *C. r. Soc. Biol. Paris* 150, 1026 (1956).

⁷ B. J. HAVERBACK, A. SJOERDSMA, and L. L. TERRY, *New England J. Med.* 255, 270 (1956).

Absence of Uricolytic Activity in Human Parotid Glands*

For the past fifty years investigators have unsuccessfully sought for an uricase in human tissues¹. The

* This work was performed during the tenure of Grants A 139 and A 139-C from the National Institute of Arthritis and Metabolic Diseases, Department of Health, Education and Welfare, Public Health Service.

¹ W. WIECHOWSKI, *Beiträge chem. Physiol.* 9, 295 (1907); 11, 109 (1908). – A. SCHITTENHELM, *Z. physiol. Chem.* 63, 248 (1909). – A. A. CHRISTMAN, *Physiol. Rev.* 32, Suppl. 1, 333 (1952).

search has been given new impetus by the observation that the amount of urate lost from the body pool is 100–250 mg greater than that excreted in the urine². This finding is suggestive evidence for the presence of uricolytic activity in the body. GEREN *et al.*³ have shown that N-labelled uric acid, administered by mouth to humans, was extensively degraded, a result quite different from that obtained when the same preparation was administered parenterally. Thus, the localization of a uricase in humans may reside in either (or both) the intestinal flora or in the host tissues.

Recently STERN and IGLESIAS claim to have demonstrated the presence of a uricolytic ferment in human saliva and particularly in human parotid gland⁴. Their method of uricase detection was not described, nor did the authors present their scheduled paper orally at the International Physiological Congress, Montreal, 1953.

Inasmuch as the occurrence of a uricase in human tissues would be of prime importance in considerations of purine metabolism and gout we endeavored to confirm the presence of this enzyme in human parotids. Two fresh human parotid glands were minced, passed through a tissue press, homogenized and diluted with phosphate buffer. An aliquot of each suspension (equivalent to 130 mg of parotid) was incubated with urate under conditions of pH, temperature and time which permit the direct relation of uricase concentration to disappearance of urate in mammalian (other than primate) liver and kidney homogenates⁵. Urate concentration was measured by standard photometric procedures.

There was absolutely no disappearance of urate from the parotid tissue, urate-containing medium. Thus uricase, as determined by conventional methods is absent in human parotid. This conclusion is in keeping with the very many past failures to demonstrate uricase in any or all human tissues.

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Zusammenfassung

In homogenisierter menschlicher Speicheldrüse liess sich entgegen den Angaben von STERN und IGLESIAS⁴ keine Urikase nachweisen.

² J. D. BENEDICT, P. H. FORSHAM, and D. STETTEN jr., *J. biol. Chem.* 181, 183 (1949). – J. BUZARD, C. BISHOP, and J. H. TALBOTT, *J. biol. Chem.* 196, 179 (1952).

³ W. GEREN, A. BENDICH, O. BODANSKY, and G. B. BROWN, *J. biol. Chem.* 183, 21 (1950).

⁴ W. STERN and O. IGLESIAS, *Abstr. XIX. Int. Physiol. Congress, Montreal, September 1953*, p. 803.

⁵ A. H. SCHEIN, E. PODBER, and A. B. NOVIKOFF, *J. biol. Chem.* 190, 331 (1951).

Electrophysiological Investigation on the Antennal Receptors of the Silk Moth During Chemical and Mechanical Stimulation

Progress in the biochemical isolation of the sexual attracting-substance of the Silkworm (*Bombyx mori*) was recently achieved by the use of new methods (BUTENANDT¹ and HECKER²). The extract of the attracting

¹ A. BUTENANDT, *Naturwiss. Rdsch. (Stuttgart)* 8, 457 (1955).

² E. HECKER, *Chem. Ber.* 88, 1666 (1955); *Verteilungsverfahren im Laboratorium* (Verlag Chemie, Weinheim 1955).