

been suggested⁹⁻¹¹ that the sign of the specific rotation of the aporphine alkaloids at 589 m μ is a true criterion of their absolute configuration. On the basis of these correlations one would expect that phanostenine (If)¹² laurelliptine, and N-methyl-laurelliptine with the same 9-methoxy-10-hydroxy substitution pattern in ring D would have the same sign of rotation. This is not so. Laurelliptine and N-methyl-laurelliptine are dextrorotatory while phanostenine is levorotatory. Hence we must conclude that we have an exception to one of these correlations.

On the basis of this proposed relationship between substituents and absolute configuration SHAMMA⁸ has suggested that thalicmidine¹³, a levorotatory, monophenolic alkaloid of the glaucine series has structure (Ig). Although the validity of this relationship is perhaps questionable the results published in this communication prove that the above structure for thalicmidine is correct. Of the four possible structures for a mono-O-des-methyl-glaucine three have now been reported. (Ih) is N-methyl-laurotetanine, (Ii) is glaucetrine and (Ik) is N,O-dimethyl-laurelliptine. As the properties of these bases do not correspond with those of thalicmidine, the structure of this latter base, by exclusion must be (Ig).

Experiments are in hand to synthesise laurelliptine and thalicmidine¹⁴.

Zusammenfassung. Dem neuen aus *Beilschmiedia elliptica* isolierten Alkaloid Laurelliptin wurde die Struktur 1,9-Dimethoxy-2,10-dihydroxy-noraporphin (III) zugeschrieben. Die früher (von SHAMMA) vorgeschlagene Struktur für Thalicmidin (Ig) wurde bestätigt.

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⁹ K. W. BENTLEY and H. M. E. CARDWELL, *J. chem. Soc.* 1955, 3252.

¹⁰ M. SHAMMA, *Exper.* 16, 484 (1960).

¹¹ C. DJERASSI, K. MISLOW, and M. SHAMMA, *Exper.* 18, 53 (1961).

¹² M. TOMITA and I. KIKAWA, *Yakugaku Zasshi* 77, 1011 (1957); *Chem. Abstr.* 52, 3833c (1958).

¹³ S. YUNUSOV and N. N. PROGRESSOV, *Zhur. Obshchei Khim.* 20, 1151 (1950); *Chem. Abstr.* 45, 1608c (1951); *Zhur. Obshchei Khim.* 22, 1047 (1952); *Chem. Abstr.* 48, 2731c (1954).

¹⁴ We are grateful to the Phytochemical Survey Unit of the Division of Organic Chemistry, C.S.I.R.O. for the collection of material, to Dr. MANSKE for samples of glaucine and its methiodide and to Dr. MARION for the picate of compound (II).

Steroid Hormones and Monolayers

In a recent review¹, WILLMER proposed a model to explain the manifold physiological effects of steroid hormones. One of its salient features involves the interaction between these hormones and lipids of the cell membrane; only those steroids which have the 'flat' shape similar to cholesterol are said to penetrate the lipids of the membrane, and consequently are active as hormones. The attractiveness of this idea prompted us to determine whether steroid hormones indeed penetrate into monomolecular lipid films and remain there. Clearly, demonstration of such interactions with simple models, would lend considerable support to WILLMER's theory.

A Langmuir-type film balance was used to obtain surface pressure (± 0.05 dynes/cm)-area data; simultaneously, surface potentials (± 10 mV) were measured using a Ra²²⁶ electrode and an electrometer. In all cases, benzene-alcohol (20:1) was used as the spreading solvent for the monolayers. Three types of films were prepared: (a) hormonally active and inactive steroids were spread as monolayers on water (pH6 and pH2) to determine their orientation at the air/water interface; (b) equimolar mixtures of representative steroids with cholesterol or cholesterol-stearic acid (1:3) were spread on water; and (c) monolayers of stearic acid, cholesterol or cholesterol-stearic acid (1:3) were spread on the surface of a saturated aqueous solution of cortisol.

In the first series (a), results obtained with estrone, androsterone, and etiocholanolone indicated that the steroids form gaseous monolayers without reorienting to a vertical position. Collapse pressures were less than 0.5 dynes/cm for each compound; surface potentials were +10, +40, and +35 mV, respectively; surface pressures and potentials were stable.

In the second series (b), the addition of androsterone, etiocholanolone, and progesterone had no effect on either the surface pressure or the potential of the monolayers of cholesterol, or cholesterol-stearic acid.

In the last series (c), cortisol had very little effect on the surface pressure-area relations for the three monolayers (stearic acid, cholesterol, or stearic acid-cholesterol (3:1)); above 3 dynes/cm all three surface pressure-area curves coincided with those of the hormone-free systems. The surface potential in each case was reduced by ~ 150 mV from the values observed for the hormone-free system.

If film penetration by the steroids had occurred, one would expect to find more pronounced effects than those observed. Even carcinogens which do not form films show fairly strong interactions with stearic acid and cholesterol monolayers². Our results do not support the mechanism proposed by WILLMER, but indicate non-specific adsorption of steroid hormones at the monolayer/water interface, rather than film penetration. MUNCK³ also found steroids to orient horizontally at the heptane/water interface, in agreement with our findings.

Zusammenfassung. Der Einfluss verschiedener Vertreter der Steroidhormone auf die Eigenschaften monomolekularer Filme von Cholesterin und Stearinsäure wurde mittels der Langmuir-Waage geprüft. Es konnte kein Eindringen der Steroide in die Filme festgestellt werden, sodass die Vorstellungen von Willmer über die Wirkungsweise der Steroidhormone experimentell nicht zu stützen waren.

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National Institutes of Health, National Institute of Arthritis and Metabolic Diseases, Bethesda (Maryland, U.S.A.), September 24, 1962.

¹ E. N. WILLMER, *Biol. Rev.* 36, 368 (1961).

² W. W. DAVIS, M. E. KRAHL, and G. H. A. CLOWES, *J. Amer. chem. Soc.* 62, 3080 (1940).

³ A. MUNCK, *Biochim. biophys. Acta* 24, 507 (1957).