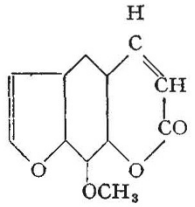

This constitution is identical with that of xanthotoxin, which had been isolated by Thoms from the fruits of *Fagara xanthoxyloides*[3] and from the berries of *Luvanga scandens* by Bose and Mookerjee[4], and which had been synthesized by Späth and Pailer[5]. We have found that ammoidin possesses the same properties as those described for xanthotoxin; for example, the product, m.p. 233°, was obtained from ammoidin under the same conditions which were followed for the preparation of nitroxanthotoxin[3]. The identity of ammoidin and xanthotoxin was finally proved by the mixed melting point (no depression) with an authentic sample of xanthotoxin; this was kindly done for us by Prof. Ebert of the University of Vienna.

Pharmacological aspect. The powdered fruits of *Ammi majus* are taken by mouth by the public in Egypt to cure leukoderma; the patient exposes the leukodermic patches repeatedly to the sun for half an hour after administration of the crude drug.

Xanthotoxin (ammoidin) has been given internally to leukodermic patients or applied as a paint over the leukodermic patches, resulting in the same curative effect as shown by the crude drug but without the toxic effects of the latter[6].

Note added in proof. Fahmy and Abushady have isolated a second crystalline substance which we have shown to be imperatorin[7].

ALEXANDER SCHÖNBERG
ALY SINA

Chemistry Department,
Faculty of Science,
Fouad I University,
Cairo. Jan. 12.

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Active Penetration of Fat into Adipose Tissue

WHEN adipose tissue of the rat, depleted of part of its fat by prolonged hunger, is incubated with blood or serum of the same rat, a decrease in the fat content of the medium can be demonstrated. Killing the tissue by heating to 80° C. abolishes this effect. The addition to the medium of sodium fluoride ($M/20$ or $M/40$), or of sodium cyanide ($M/500$) also completely inhibits the disappearance of fatty acids. Sodium azide ($M/1{,}000$) had no inhibiting effect. The most marked decrease in the fat content of the medium was obtained with adipose tissue and blood of rats starved to 25 per cent loss of weight. With fat-laden adipose tissue of normal rats the effect was negligible or absent. No decrease in the fat content of the blood was found when the incubation was carried out at room temperature. The accompanying table summarizes these results.

No. of expts.	Tissue	Medium	Average decrease in fat content of medium
17	Depleted groin fat	Blood of same rat	40±2·6 per cent
5	,, heated to 80° C.	,, ,, ,,	0
6	Fat-laden groin fat	,, ,, ,,	10±8 ,, ,,
4	Depleted groin fat	,, with $M/20$ NaF	0
4	,, ,, ,,	,, with $M/40$ NaF	5 ,, ,,
5	,, ,, ,,	Serum, with $M/500$ NaCN	0
3	,, ,, ,,	Blood, with $M/1{,}000$ NaN_3	34 ,, ,,
3	,, ,, ,,	Blood, at room temperature	0

100 mgm. of groin fat are cut with scissors to a fine brei and incubated with 1 ml. of blood or serum, in air, at 38° C. The fatty acid content is estimated in 0·1 ml. of the medium after half an hour (initial value) and after 3½ hours of incubation, according to the method of Marenzi and Cardini[1].

The quantity of fatty acids disappearing from the blood, by incubation with depleted adipose tissue for 3 hours, is between 1 and 2 mgm. The oxidation of fat by the tissue accounts for only 0·025 mgm. (calculated from the value of 0·18 mm.³ oxygen consumed per mgm. fat-depleted adipose tissue per hour). The main quantity of the disappearing fat must thus have been taken up by the tissue without any profound change.

The results of these experiments show that the penetration of fat into adipose tissue is an active process, depending on the physiological activity of the tissue.

The physiological and pathological conditions influencing this penetration are under investigation.

B. SHAPIRO V. BENTOR
D. WEISSMANN E. WERTHEIMER
Laboratory for Pathological Physiology,
Hebrew University, Jerusalem.
Dec. 10.

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Peripheral Action of Botulinum Toxin

AN investigation has been made of the pharmacological properties of *Cl. botulinum* (type *A*) toxin. Rabbits injected intravenously with the toxin developed severe œdema with focal hæmorrhages in the lungs. These lung lesions were also found when 'local' botulism was produced in a number of tissues, but with larger doses of toxin. It was therefore possible to achieve a local concentration of toxin sufficiently high to paralyse certain nerve-endings without killing the animals prematurely by the injury to the lungs.

Intra-ocular injections of the toxin produced a total paralysis of the nerves to the sphincter pupillæ, which failed to react to light or to stimulation of the oculomotor nerve. This action was analysed and was found to be due neither to any injury of the smooth muscle itself nor to a neuro-muscular block of the type produced by atropine (as suggested by Edmunds and Long[1]), since the sphincter reacted normally to acetylcholine.

The injurious effect of the toxin appears, therefore, to be on the cholinergic nerve fibres proper and is restricted to them, since stimulation of the cervical sympathetic nerve still dilates the pupil, and reflexes