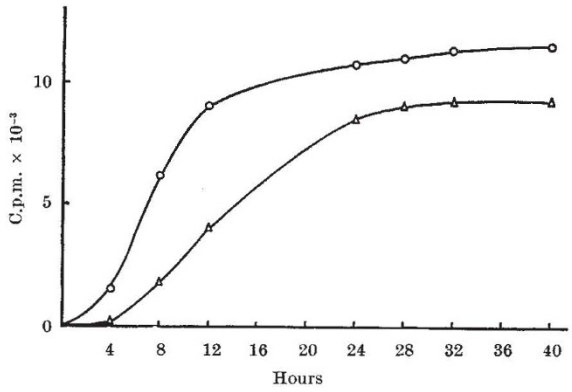

Fig. 1. Absorption of 1-^{14}C lignoceric acid by rat intestine: A neutralized, aqueous suspension of 1-^{14}C lignoceric acid (4 μmoles, 20,000 c.p.m./μmole) was given by stomach tube (△) or intraduodenal injection (○). The thoracic duct was cannulated, the lymph collected and the radioactivity of the lipids determined

acids) thus released is absorbed and afterwards transported as neutral glycerides in the lymph. It has previously been shown[1] that lignoceric acid, in the form of neutral lipids, is metabolized by the liver. In addition, 1-^{14}C lignoceric acid, when injected into rats, is oxidized to respiratory $^{14}CO_2$ (ref. 1). Therefore, it is reasonable to assume that the dietary lignoceric acid is transported to the liver, released from its linkage to glycerol and then either oxidized or again incorporated into the sphingolipids or glycerides.

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M. Fields (Gellei)*
S. Gatt

Department of Biochemistry,
The Hebrew University-Hadassah Medical School,
Jerusalem, Israel.

* Present address: University Department of Surgery, The General Infirmary, Leeds 1.

[1] Gatt, S., and Shapiro, B., *Nature*, **185**, 461 (1960).
[2] Bollman, J. L., Cain, J. C., and Grindley, J. M., *J. Lab. Clin. Med.*, **33**, 1349 (1948).
[3] Borgstroem, B., *Acta Physiol. Scand.*, **25**, 101 (1952).
[4] Bergstroem, S., Borgstroem, B., Carlsten, A., and Rottenberg, M., *Acta Chem. Scand.*, **4**, 1142 (1950).
[5] Borgstroem, B., *Acta Chem. Scand.*, **5**, 643 (1951).
[6] Bergstroem, S., Borgstroem, B., and Rottenberg, M., *Acta Physiol. Scand.*, **25**, 120 (1952).
[7] Borgstroem, B., *Acta Physiol. Scand.*, **25**, 140, 291, 315 (1952).
[8] Bloom, B., Chaikoff, I. L., Rheinhardt, W. O., Entenman, C., and Dauben, W. G., *J. Biol. Chem.*, **184**, 1 (1950).
[9] Bloom, B., Chaikoff, I. L., Rheinhardt, W. O., and Dauben, W. G., *J. Biol. Chem.*, **189**, 261 (1951).

Occurrence of *trans*-Octadec-16-enoic Acid in Sheep and Ox Perinephric Fats

In their investigations on the fatty acid composition of butter-fat, Backderf and Brown[1] deduced the presence of *trans*-octadec-16-enoic acid, an acid hitherto unknown in Nature. Later this acid was isolated from butter-fat by Hansen and Cooke[2] and its physical and chemical characteristics were established. The present communication reports the detection by gas-liquid chromatography of this acid in sheep and ox fats using as a basis for identification the gas-liquid chromatographic data previously obtained for the methyl esters of pure *trans*-octadec-16-enoic acid isolated from butter-fat. The methyl esters of both sheep and ox fats were analysed with a chromatograph fitted with a strontium-90 ionization detector and an 8 ft. × ¼ in. column. With a 'Celite' column impregnated with 20 per cent (w/w) polydiethylene glycol adipate at 207° a peak appeared in both fats with R_F value 1·26 corresponding with that recorded for the methyl ester of *trans*-octadec-16-enoic acid[2], but with a 'Celite' column containing 5 per cent (w/w) 'Apiezon *L*' at 207° this component was not apparent. As was earlier found with the polydiethylene glycol adipate stationary phase[2], resolution from other *cis* and *trans* C_{18} isomers and from methyl stearate was complete whereas with a 'Celite' column impregnated with 5 per cent (w/w) 'Apiezon *L*' the methyl ester of *trans*-octadec-16-enoic acid was not separated from methyl stearate. To confirm the presence of *trans*-octadec-16-enoic acid and other *trans* acids in sheep fat and ox fat, work is in progress which is designed to isolate and identify these fatty acid constituents.

R. P. Hansen

Fats Research Laboratory,
Department of Scientific and Industrial Research,
Wellington, New Zealand.

[1] Backderf, R. H., and Brown, J. B., *Arch. Biochem. Biophys.*, **76**, 15 (1958).
[2] Hansen, R. P., and Cooke, N. J., *Biochem. J.*, **81**, 237 (1961).

Content of Extractable Gangliosides in Rat Brain during Ethanol Intoxication

McIlwain *et al.* have established that the content of extractable gangliosides (measured by their *N*-acetylneuraminic acid moiety) is directly related to the excitability of the nervous tissue[1]. The *N*-acetylneuraminic acid is suggested to form a hydrophilic path for the passage of cations through the lipid membrane[2]. We wished to check whether the decreased metabolic response of rat brain cortex slices to electrical stimulation during ethanol intoxication[3] could be accounted for by a decrease in the *N*-acetylneuraminic acid groups available.

Rats of Wistar strain (weight 350–430 g) received 550 mg of ethanol per 100 g of body weight as a 33 per cent (v/v) aqueous solution by stomach tube. The control animals received an equal volume of water. The rats were killed after 1 h by decapitation and their brains dissected immediately at room temperature. The brains were severed at the caudal level of the cerebral hemispheres and the cranial parts were analysed. The gangliosides were determined according to Long and Staples[4] with the modifications that the first supernatant in chloroform–methanol mixture was centrifuged instead of filtered, and that, after addition of a solution of potassium chloride, the layers were allowed to separate overnight instead of being centrifuged. The standard curve was based on two samples of *N*-acetylneuraminic acid (a gift from Prof. L. Svennerholm, of Gothenburg, Sweden, and a commercial preparation from L. Light and Co., Ltd., Colnbrook, England), which agreed within the experimental error.

The content of extractable *N*-acetylneuraminic acid, in μmoles per 100 mg of fresh weight, was in the brains of: (*a*) ethanol-intoxicated rats $0{\cdot}211 \pm 0{\cdot}011$ ($n = 10$); (*b*) control rats $0{\cdot}188 \pm 0{\cdot}011$ ($n = 9$).

It is evident that no decrease of *N*-acetylneuraminic acid occurs during intoxication but rather an increase ($t = 4{\cdot}6$, $P < 0{\cdot}001$), for which we cannot offer any explanation at present. It may be relevant, though, that the metabolism of certain amino-acids, including glutamic acid and glutamine, is disturbed during ethanol intoxication[5].

Hertta-Maija Häkkinen
E. Kulonen

Research Laboratories of the
State Alcohol Monopoly (Alko),
Helsinki,
Finland.

[1] McIlwain, H., *Biochem. J.*, **76**, 16 P (1960); **78**, 24 (1961). Balakrishnan, S., and McIlwain, H., *ibid.*, **81**, 72 (1961).
[2] McIlwain, H., Woodman, R. J., and Cummins, J. T., *Biochem. J.*, **81**, 79 (1961).
[3] Wallgren, H., and Kulonen, E., *Biochem. J.*, **75**, 150 (1960).
[4] Long, C., and Staples, D. A., *Biochem. J.*, **73**, 385 (1959).
[5] Häkkinen, H.-M., and Kulonen, E., *Biochem. J.*, **78**, 588 (1961).