rather significant rôle. The observation has been made that certain toxic substances such as cobra venom when mixed with lecithin cause it to lose its unsaturated fatty acid radicle.[12] The resulting lecithin compound contains a relatively high proportion of saturated fatty acids. The question thus arises as to whether the bacterial toxins of an acute infection could have the same influence on the plasma phospholipids. Most logical at the present time is the assumption that the intensity of the fat metabolism during an acute infection and the presence of the bacterial toxins in the blood may both be responsible for the ultimate fall in the plasma phospholipids to abnormal levels with an associated extensive loss of their unsaturated fatty acids. Should this actually be found to occur, it would give the phospholipids an important part in the phenomena of immune reactions.

9374 P

Photoelectric Spectrophotometry Applied to Studies in Fat Metabolism.*

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Recently new methods have been applied for following the rate of movement and metabolism of fats. Schoenheimer and Rittenberg[1] have published a series of papers on the use of deuterium as an indicator in fatty acid metabolism. Sinclair and coworkers[2] have made use of elaidin as an indicator of rate of movement and metabolism of fats in various rat tissues.

The common fats and fatty acids show very low absorption of light from 2000 to 8000 A.U., except for a weak band at 2300 A.U.[4] However, there are 2 exceptional cases. α-Eleostearic acid of tung oil has a very strong maximum at 2700 A.U.[3] Moore[4] has shown that linseed oil fatty acids become strongly "absorptive" on

12 Peters, John P., and Van Slyke, Donald, *Quantitative Clinical Chemistry*, Williams and Wilkins Company, Baltimore, 1931, Vol. 1.

* Aided by grants from the Rockefeller Foundation, the Medical Research Fund of the Graduate School of the University, and the National Live Stock and Meat Board.

1 Rittenberg, D., and Schoenheimer, R., *J. Biol. Chem.*, 1937, **117**, 485.

2 McConnell, K. P., and Sinclair, R. G., *J. Biol. Chem.*, 1937, **118**, 123.

3 Dingwall, A., and Thomson, J. C., *J. Am. Chem. Soc.*, 1934, **56**, 899.

4 Moore, T., *Biochem. J.*, 1937, **31**, 138.

prolonged saponification. These acids are digestible and cure fatty acid deficiency symptoms. It has also been shown that highly unsaturated fatty acids are changed to an absorptive form by the cow and secreted in milk.[5]

Using apparatus similar to that described by Hogness, Zscheile and Sidwell,[6] small amounts of a strongly absorbing lipid can be determined in the presence of the usual body lipids. This method offers the advantage of speed and accuracy of determination on very small samples. Less than 10 mg. of a lipid are required.

Tung oil was chosen for the first work because of its ready digestibility and very high absorption. At 2700 A.U. $E^{1\%}_{1\,cm.} = 1400$ in the specimen of oil used. At the same wave length adipose tissue of a rat has an $E^{1\%}_{1\,cm.}$ of less than one. Therefore, a very small amount of the tung oil acids deposited in a rat tissue is readily detected.

Three mature rats on stock diet were starved over night (18 hours) and fed at 9 a. m. 10 gm. of the same stock diet mixed with 1 gm. of tung oil. Uneaten food was removed from the cage in the afternoon and total oil consumption calculated. Regular stock diet was put in the cage so that there was no starvation following oil intake. One rat was killed at 9 a. m. each succeeding morning, that is, at 24, 48 and 72 hours after the first intake of tung oil. Table I summarizes the data.

TABLE I.

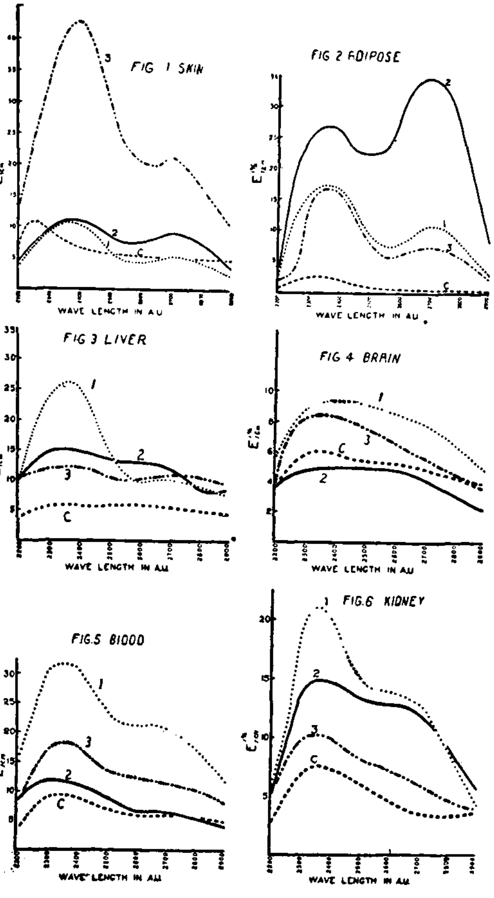
Case	Rat No.	Body wt. g.	Tung oil consumed g.	Time killed after first oil intake hr.
1	36194	180	0.9	24
2	36210	222	0.9	48
3	36212	189	0.64	70

At autopsy the blood, liver, kidneys, brain, skin and adipose tissue were collected. Solid tissues were ground with glass and the fat removed by prolonged alcohol-ether extractions. Solvents were removed *in vacuo,* true lipids taken up in petroleum ether, which was then removed *in vacuo* in a tared flask. The samples were made to volume in purified ether and aliquots used for the spectroscopic studies. Some results are summarized in Figs. 1 to 6.

[5] Dann, W. J., Moore, T., Booth, R. L., Golding, J., and Kon, S. K., *Biochem. J.*, 1935, **29**, 138.

[6] Hogness, T. R., Zscheile, F. P., Jr., and Sidwell, A. E., Jr., *J. Phys. Chem.*, 1937, **41**, 379.

Full interpretation of the curves is not possible at this time. First, it should be pointed out that the animal very quickly changes eleostearic acid, causing a loss of the band at 2700 A.U. and a rise in absorption at 2350 A.U. This fact must be considered in evaluating the data.



FIGS. 1-6.
Absorption curves of lipids extracted from six tissues of three experimental rats and a control. Curves numbered 1, 2 and 3 represent rats killed 24, 48, and 70 hours respectively after consumption of the first tung oil. C is the control on stock diet, but with no tung oil.

However, certain very interesting differences in tissues are at once apparent. According to the rapidity with which the 2700 A.U. band disappears during the first day, the tissues fall in the following order: liver, kidney, blood, adipose and skin. Adipose tissue received a large amount of unchanged acid (Fig. 2, curve 2) during the second day, which was largely gone by the third day. The new acid with high absorption at 2350 A.U. persisted through the third day. The skin received almost none of this material for 2 days, but on the third day large amounts of the changed acid were deposited (Fig. 1, curve 3). The brain showed a small but appreciable response to tung oil feeding. The order of the curves is the same as for blood, which suggest that the immediate blood supply may control the results; but the amount of blood present can not account for all of the absorptive acid, as the brain lipids were several times the total blood lipids.

Conclusions. Eleostearic acid is quickly changed to a new acid *in vivo.* Distribution and metabolism of this acid has been followed by spectroscopic analysis and large differences found among several tissues.

9375 P

Reaction of the Supraoptic Nucleus to Hypophysectomy.*

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The origin and significance of the hypothalamico-hypophyseal neural system are still controversial. Keller, Noble and Hamilton[1] and Mahoney and Sheehan,[2] who interrupted these fibers in the infundibulum, rather minimize their rôle. On the other hand, Ranson and co-workers seem to have established a very close connection between lesions in this system and at least diabetes insipidus.

The origin is generally regarded as being the paired supraoptic nuclei, 2 more dorsally situated paraventricular (filiform) nuclei and some less definitely localized cells in the tuber cinereum. In the dog retrograde degeneration of the supraoptic nuclei 10-16 days after destructive lesions in the processus infundibuli and infundibulum was noted by Kary,[3] Lewy,[3] Maiman[3] and Broers.[4] Hare[5] re-

* Aided by a grant from the Research Fund, Graduate School, University of Minnesota.

1 Keller, A. D., Noble, W., and Hamilton, J. W., Jr., *Am. J. Physiol.*, 1936, **117**, 467; PROC. SOC. EXP. BIOL. AND MED., 1936, **34**, 794; Keller and Noble, *Proc. Am. Physiol. Soc.*, 1936, **48**, 90.

2 Mahoney, W., and Sheehan, D., *Brain*, 1936, **59**, 61.

3 Kary, K., *Virchows Arch. f. path. Anat. u. Physiol.*, 1924, **252**, 734; Lewy, F. H., *Zentralb. f. d. ges. Neurol. u. Psych.*, 1934, **37**, 398; Maiman, R. M., *Z. f. d. ges. Neur. u. Psych.*, 1930, **129**, 666.

4 Broers, H., *Diss. Inaug. Kemink en Zoon*, Utrecht, 1932.

5 Hare, H., *Proc. Am. Physiol. Soc.*, 1937, **49**, 70.