- Sun, G. Y. & Horrocks, L. A. (1971) *J. Neurochem.* **18**, 1963
 Webster, G. R. & Cooper, M. (1968) *J. Neurochem.* **15**, 795
 Wells, M. A. & Dittmer, J. C. (1967) *Biochemistry* **6**, 3169

Cerebroside Sulphate Biosynthesis in Brain

By B. K. BACHHAWAT, SHALINI KOKRADY and S. SAXENA (*Neurochemistry Laboratory, Department of Neurological Sciences, Christian Medical College Hospital, Vellore, S. India*)

Earlier reports from our laboratory have shown the presence in brain of a sulphotransferase that catalyses the transfer of sulphate from adenosine 3'-phosphate 5-sulphatophosphate to an endogenous protein-bound galactose cerebroside. This enzyme has been partially purified from sheep brain, and was found to be activated by EDTA and thiols such as GSH and cysteine. Attempts to use exogenous cerebroside as a substrate for this enzyme were unsuccessful, and the use of detergents such as sodium deoxycholate, Triton X-100 and Brij-96 resulted in a marked decrease in its activity (Bhandrai & Bachhawat, 1967). Recently it has been possible to purify the endogenous acceptor partially by DEAE-Sephadex chromatography, and indirect evidence has been obtained that indicates that it is a protein-bound cerebroside. This sulphotransferase system seems to be distinct from the one described by Cumar *et al.* (1968) and McKhann & Ho (1967). Recently, by using purified microsomal and synaptosomal fractions, we have been able to show that these preparations are very active in our system and that detergents markedly inhibit the activity. Solubilization of the microsomal fraction by deoxycholate led to a marked decrease in the activity, even though detectable amounts of cerebroside were present in the solubilized preparation, which further supports our previous observation that this cerebroside sulphotransferase is distinct. It also explains the failure of Kopaczky & Radin (1965) to detect the formation of any sulphatide after injections into rats of cerebroside containing radioactive non-hydroxy fatty acid. This can be explained from our observation, because exogenous cerebroside is not an appropriate substrate for the synthesis of the product in our system. T.l.c. patterns of the isolated cerebroside suggest that both hydroxy and non-hydroxy fatty acids containing sulphatides are synthesized in our system.

- Bhandari, V. R. & Bachhawat, B. K. (1967) *Proc. 1st. Int. Meet. International Society for Neurochemistry, Strasburg*, p. 24
 Cumar, F. A., Barra, H. S., Maccioni, H. J. & Caputto, R. (1968) *J. Biol. Chem.* **243**, 3807
 Kopaczky, K. C. & Radin, N. S. (1965) *J. Lipid Res.* **6**, 140
 McKhann, G. M. & Ho, W. (1967) *J. Neurochem.* **14**, 717

Absorption of Lipids

By J. GANGULY, P. V. SUBBAIAH and S. PARTHASARATHY (*Department of Biochemistry, Indian Institute of Science, Bangalore-12, India*)

Recent work on the absorption of triglycerides has shown that during the digestive phase the dietary triglycerides are hydrolysed by the pancreatic lipase mainly to the 2-monoglyceride stage, after which the products of lipolysis appear in a micellar solution along with bile salts. From such a micellar solution the 2-monoglycerides and free fatty acids diffuse into the mucosal cell to be reassembled into triglycerides. By using triglycerides containing ^{14}C -labelled fatty acids at different positions Raghavan & Ganguly (1967) have produced convincing evidence that during the process of digestion and resynthesis about 80% of the fatty acid at the β -position of the triglycerides is retained at the same place.

Our sustained work on the mechanism of absorption of retinyl and cholesteryl esters has similarly shown that the retinyl esters are partially hydrolysed while in the fat phase, after which the retinol and its esters appear in a micellar solution (David & Ganguly, 1967). Both retinol and its esters then diffuse into the cell, where the hydrolysis of the ester is completed and retinol is re-esterified preferentially with palmitic acid. The cholesteryl esters, on the other hand, are totally hydrolysed while in the fat phase, so that only the de-esterified sterol appears in the micelle; the micellar sterol is then admitted into the cell, where it is re-esterified.

We have also demonstrated that the rat mucosal cell is rich in phospholipase A activity and that considerable proportions of this activity are localized in the brush border (Subbaiah & Ganguly, 1970). Our work further demonstrated the presence of a very active acyl-CoA-lysophosphatidylcholine acyltransferase in the microsomal fraction of rat mucosa (Subbaiah *et al.*, 1970). Sufficient evidence has since been gathered to demonstrate that during absorption the fatty acid at the β -position of phosphatidylcholine (lecithin) undergoes rapid turnover. Here also it appears that the phosphatidylcholine is hydrolysed by the pancreatic phospholipase A to lysophosphatidylcholine, which being readily soluble in water can diffuse into the cell to be reacylated by the mucosal acyltransferase.

Thus it appears that there are certain similarities in the absorption of different lipids, in that they are hydrolysed by the lipolytic enzymes in the intestinal lumen to such a state that they can be made water-soluble and thereby can readily diffuse into the mucosal cell, to be re-esterified intracellularly to the parent compounds and transported through the lymph. The re-esterification of cholesterol and