PORTAL ABSORPTION OF FATTY ACIDS IN LYMPH- AND PORTAL VEIN-CANNULATED RATS. S. A. Hyun, G. V. Vahouny and C. R. Treadwell (Dept. of Biochem. School of Med. The George Washington Univ., Washington, D.C.). *Biochim. Biophys. Acta* **137**, 296–305 (1967). The route and the rate of the intestinal absorption of oleic-1-^{14}C, caprylic-1-^{14}C and 2-ethyl-^{14}C-n-caproic acids have been studied using lymph and portal vein-cannulated rats. It was found that 85% of absorbed oleic acid was transported directly *via* the portal system. With short-chain fatty acids, between 94–98% of the absorbed acids were transported *via* the portal system. Studies on the rate of the intestinal absorption of these acids indicated that 2-ethyl-n-caproic acid was absorbed less completely and subsequently metabolized less effectively than the corresponding straight-chain fatty acid, caprylic acid. Studies on the distribution of radioactivity in lymph lipids showed that most of the radioactivity (85%) was present as triglycerides when oleic acid-^{14}C was administered to lymph and portal vein fistula rats. However, when the ^{14}C-labeled short-chain fatty acids were given, 96–102% of the radioactivity was present as free fatty acids. Studies on the distribution of radioactivity in lipid fractions of portal vein blood showed that 50 and 98–100% of the radioactivity present were in the form of free fatty acids when oleic acid-^{14}C and ^{14}C-labeled short-chain fatty acids, respectively, were administered to lymph and portal vein fistula rats.

MECHANISM OF STIMULATION OF CHOLESTEROL ABSORPTION BY 2-ETHYL-N-CAPROIC ACID IN VIVO. *Ibid.*, 306–14. Studies on the mechanism of stimulation of (7 α-^{3}H) cholesterol absorption of 2-ethyl-n-caproic acid have been carried out in lymph and portal-vein cannulated rats. About 15% of administered cholesterol was absorbed in 8 h when the sterol was given with oleic acid alone. The administration of 2-ethyl-n-caproic acid together with oleic acid and cholesterol significantly increased the absorption of cholesterol to 23%. Simultaneous determination of fatty acid absorption and esterification showed that 44% of the fed oleic acid-1-^{14}C was recovered in thoracic duct lymph in 8 h, and about 15% of the long-chain acid was transported directly via portal blood. However, in the presence of 2-ethyl-n-caproic acid, only 22% of the administered oleic acid-1-^{14}C was recovered in lymph, while 40% was transported directly into portal system. This effect of the branched-chain fatty acid was most pronounced 4–8 h after feeding. In this group there was a reduction in total lymph triglycerides and a slight increase in lymph free fatty acids. These data support the earlier suggestion that the primary effect of 2-ethyl-n-caproic acid is inhibition of triglyceride synthesis in intestinal mucosa, resulting in increased transport of free fatty acids via portal blood and increased availability of fatty acids for cholesterol esterification and absorption.

THE ROLE OF PLACENTA IN LYSOLECITHIN METABOLISM IN RATS AND MICE. S. Eisenberg, Y. Stein and O. Stein (Dept. of Medicine B and Lipid Research Laboratory, Hadassah Univ. Hospital, Jerusalem (Israel)). *Biochim. Biophys. Acta* **137**, 115–20 (1967). In 16–20-days pregnant rats a 50% fall in serum lysolecithin level was found. Following intravenous injection into pregnant rats and mice there was an extensive uptake of palmitoyl-1-^{14}C-lysolecithin by the placenta followed by a rapid conversion of the labeled lysolecithin to lecithin. During the first 10 min after injection of palmitoyl-1-^{14}C-lysolecithin-^{32}P, the ^{14}C/^{32}P ratio in the newly formed lecithin was the same as the injected lysolecithin, indicating that the conversion of lysolecithin to lecithin in the placenta was accomplished through the acylation pathway. It is concluded that the fall in serum lysolecithin is due to a selective uptake of lysolecithin by the placenta and this additional source of placental lecithin could be of importance in an organ with a pronounced tronsport function.

THE RESOLUTION OF (±)-CARNITINE AND THE SYNTHESIS OF ACYLCARNITINES. K. Brendel and R. Bressler (Dept. of Biochem. and Med., Duke Univ. Medical Center, Durham, N.C.). *Biochim. Biophys. Acta* **137**, 98–106 (1967). Procedures are described for the resolution of (±)-carnitine nitrile chloride into its optical isomers by salt formation with (+)-10-camphorsulfonic acid and (+)-dibenzoyltartaric acid. Procedures for the acylation of carnitine are described employing large excesses of the acylating agent in a homogenous reaction. The acylation of the carnitine benzyl ester can be carried out using lower ratios of acylating agent to carnitine.

METABOLISM OF 1-PALMITOYL DIOLEIN AND 3-PALMITOYL DIOLEIN BY ADIPOSE TISSUE. W. R. Wright and S. B. Tove (Depts. of Biochem. and Animal Sci., North Carolina State Univ.,

(Continued on page 522A)

Swedish Symposium on Metal Catalyzed Lipid Oxidation

The Swedish Institute of Food Preservation Research (SIK) in Göteborg is organizing a symposium on Metal Catalyzed Lipid Oxidation on Oct. 9 and 10, 1967.

Metal catalyzed lipid oxidation—as metal catalysis in general—plays an important role in terms of quality and stability in various branches of food technology. It therefore appeared desirable to gather experts with different lines of interest within this sphere at a symposium in order to promote scientific research and technical development.

Invitations were mainly sent to scientists engaged in research within this complex of problems and prepared to take an active part in the symposium.

The idea of this symposium has been accepted with great interest and scientists representing 15 countries were represented at SIK.

The symposium will be introduced by Dr. Ingold from the National Research Council, Ottawa, Canada, who will deliver a review on the subject in question. There was a section on analytical techniques, a section on fundamental research, and a section on the problems in various sectors of food technology and industry. Finally packaging problems were discussed.

Organizing secretary of the symposium was Dr. Reinhard Marcuse, SIK, Göteborg.

Basic Statistics and Evolutionary Operation Courses

Basic Statistical Methods for the Chemical and Process Industries

A two-day short course will be co-sponsored by the Chemical Division of ASQC and the Section on Physical and Engineering Sciences of ASA. This course will cover two days of practical, easy-to-learn statistical methods for the engineer and the applied scientist in industry—in the plant or in the laboratory. Principal topics to be covered are concepts, frequency distributions, control charts, comparison of means, comparison of variances, test precision and analysis of variance, regression, experimental design and evolutionary operation.

The instructors are D. S. Chambers, Professor of Statistics at the University of Tennessee, and Mr. H. O. Hehner, Manager of Quality Control of Monsanto's Organic Chemicals Division.

The registration fee is $100, dates are November 3 and 4, and place is the Midland Hotel in Chicago. For further information and application forms, contact Mr. Charles Ferczok, Operations Research Department, Swift & Company, 115 W. Jackson Blvd., Chicago, Illinois 60604. Telephone number is 312–431–2777.

Evolutionary Operation

A three-day short course on Evolutionary Operation will be co-sponsored by the Chemical Division of ASQC and the Section on Physical & Engineering Sciences of ASA.

Cost reduction, quality improvement and increased capacity are demonstrated results of EVOP. This method of process improvement has been so successful because it is applied directly to operating processes and designed to be used by regular plant personnel. This method of process improvement does not interfere with normal production.

The registration fee is $140, which includes all course materials and lunches. Dates are November 16, 17 and 18, in the Midland Hotel, Chicago, Ill. For further information and application forms, contact G. R. Wagner, Operations Research Dept., Swift & Company, 115 W. Jackson Blvd., Chicago, Illinois 60604, Telephone 312–431–2777.