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or 15% fat, dietary choline decreased (P < 0.01) the concentration of hepatic lipid and moisture but did not significantly alter choline. Increasing dietary fat from 5 to 15% decreased (P < 0.01) food intake, weight gain and concentration of moisture but not of choline in the liver; and 15% fat caused a greater accumulation of lipid in the liver in the absence of choline than did 5 or 10%. There was significant interaction between dietary choline and methionine only in respect to weight gain and liver weight (P < 0.05), and between choline and fat in respect to lipid and moisture in fresh liver (P < 0.01).

THE BINDING OF ESTRADIOL-17 $\beta$  TO HUMAN BREAST CANCERS AND OTHER TISSUES IN VITRO. H. Johansson, L. Terenius and L. Thorén (Depts. of Pharmacol. and Surgery, Univ. of Uppsala, Uppsala, Sweden). Cancer Res. 30, 692–98 (1970). Material from malignant and benign mammary tumors in women was incubated with tritium-labeled 1,3,5(10)-estratriene-3,17 $\beta$ -diol (estradiol-17 $\beta$ ) under conditions which allowed an estimation of the "receptor"-bound estradiol-17 $\beta$ . Of the 31 cancers 14 showed significant binding of estradiol-17 $\beta$  while of the 26 benign tumors only 2 showed significant binding. There was no obvious correlation between the estradiol-17 $\beta$  binding capacity of a cancer and factors of the disease at the time of operation (clinical stage, histopathological classification, or differentiation of the tumor) or of the menopausal stage. There was never significant binding of estradiol-17 $\beta$  in the normal tissue surrounding the tumor. It was found possible to store tumor material (from experimental mammary tumors) at 0° for 24 hours without changes in estradiol-17 $\beta$  binding while lower storage temperature destroyed the binding capacity.

THE METABOLISM OF GLYCERIDE GLYCOLIPIDS. IV. ENZYMATIC HYDROLYSIS OF MONOGALACTOSYL AND DIGALACTOSYL DIGLYC-ERIDES IN RAT BRAIN. K. Subba Rao, D. Wenger and R. Pieringer (Dept. of Biochem., Temple U. School of Med., Philadelphia, Penn. 19140). J. Biol. Chem. 245, 2520-24 (1970). Particulate fractions of rat brain catalyze the hydrolysis of radiochemically pure monogalactosyl and digalactosyl diglycerides, which had been synthesized in vitro by enzymes derived from rat brain or spinach chloroplasts. Monogalactosyl-U-<sup>14</sup>C diglyc-

## • Local Section News . . .

The Program Planning Committee of the Northeast Section has outlined and approved the 1970–1971 schedule of meetings for the section. The dates and subjects were selected as follows:

On Sept. 15, 1970, a meeting will be held at Whyte's Restaurant in New York. The Northeast Section AOCS will present to S. S. Chang its Achievement Award. Dr. Chang will also speak on "The Volatile and Non-Volatile Decomposition Products Produced During Deep Fat Frying of Foods." Meeting Chairman is M. Eijadi.

On Oct. 21, 1970, a meeting will be held in Philadelphia at the Franklin Motor Inn. The evening's speaker will be Bob Casparian on the topic "Water Waste Pollution." George Raupp is Meeting Chairman.

On Dec. 8, 1970, a meeting will be held at the Military Park Hotel in Newark, N.J. The topic of the meeting will be "The Application of Nuclear Magnetic Resonance in Fats and Oils," and the speaker will be W. A. Bosin. Meeting Chairman is J. P. McNaught.

On Feb. 16, 1971 the Northeast Section AOCS will hold a joint meeting with the Filtration Engineers' Society in the midtown area of New York City. The topic for the evening will be "Bleaching and Refining," and the Meeting Chairman is August Rossetto.

April 13, 1971 is the date of the section's Symposium on "Fatty Acids: Tall Oils, Iso-Acids, Solvent Crystallization." The meeting will include a speaker on labor relations, among other subjects to be taken up. The Symposium Chairman is Frank Naughton.

The final meeting of the season will be the traditional Ladies' Night to be held in Greenwich Village, New York. The "School Feeding Program" will be the evening's topic with guest speaker P. A. Lachance. The Meeting Chairman is S. S. Chang. eride yields <sup>14</sup>C-galactose when incubated with the mitochondrial fraction of brain at the optimum pH of 4.4 (galactosidase conditions) and monogalactosyl-U-<sup>14</sup>C glycerol when incubated with the microsomal fraction of brain at the optimum pH of 7.2 (galactolipase conditions). Biosynthesized diagalactosyl-U-<sup>14</sup>C diglyceride is degraded in a similar way. There is no cross-contamination of the two degradative enzyme systems.

COINDUCTION OF BAT LIVER BRANCHED CHAIN a-KETO ACID DEHY-DROGENASE ACTIVITIES. R. M. Wohlhueter and A. E. Harper (Dept. of Biochem., Univ. of Wis., Madison, Wisc. 53706). J. Biol. Chem. 245, 2391-2401 (1970). By use of carboxyllabeled a-ketoisocaproic, a-ketoisovaleric, and a-keto- $\beta$ -methylvaleric acids, branched chain keto acid dehydrogenase activities were measured in rat tissues. a-Ketoisocaproic acid dehydrogenase was shown to be a mitochondrial enzyme and was found almost exclusively in liver, in contrast to leucine-a-keto-glutarate aminotransferase which was found predominantly in kidney. a-Ketoisocaproic, a-ketoisovaleric, and a-keto- $\beta$ -methylvaleric acid dehydrogenase specific activities in liver increased linearly and in constant proportion to each other as the protein content of the diets was increased from 0 to 30%. Modulation of dehydrogenase activity appears to be the resultant of at least two superimposable mechanisms. One, cycloheximide-sensitive, is apparently an adaptation to dietary protein intake. The other cycloheximide-insensitive, involves a daily activationdeactivation cycle.

BIOSYNTHESIS OF 3-HYDROXY-3-METHYLGLUTARATE AND MEVALO-NATE BY RAT LIVER HOMOGENATES IN VITRO. L. W. White and H. Rudney (Dept. of Biochem., Case Western Reserve Univ., Cleveland, Ohio 44106). Biochemistry 9, 2713-24 (1970). Methods have been developed for studying regulation of 3hydroxy-3-methylglutaryl-coenzyme A (HMG-CoA) and mevalonate biosynthesis in fractionated rat liver homogenates. Using methods described here, accurate delineation of early regulatory points in the cholesterol biosynthetic pathway may be performed. The validity of previous studies, the precautions required and the usefulness and importance of these methods are discussed.

REGULATION OF 3-HYDROXY-3-METHYLGLUTARATE AND MEVALO-NATE BIOSYNTHESIS BY RAT LIVER HOMOGENATES. EFFECTS OF FASTING, CHOLESTEROL FEEDING, AND TRITON ADMINISTRATION. *Biochemistry* 9, 2725-30 (1970). Regulation of biosynthesis of 3-hydroxy-3-methylglutarate (HMG) and mevalonate has been studied in fractionated rat liver homogenates using methods that permit evaluation of possible control points between acetate and mevalonate. Fasting results in depression of HMG-GoA reductase activity and decreased activation of acetate. Triton WR 1339 administration is associated with increased acetate activation and HMG-CoA reductase activity. By contrast cholesterol feeding is associated with inhibition of both HMG-CoA condensing enzyme and HMG-CoA reductase; however the major effect is on the reductase and the inhibition is generally greater than 90%, thereby accounting for the major part of the decrease in cholesterol synthesis.

STUDIES ON THE CONTROL OF FATTY ACID METABOLISM. G. Weeks and S. J. Wakil (Dept. of Biochem., Duke Univ. Med. Center, Durham, N. Carolina 27706). J. Biol. Chem. 245, 1913–21 (1970). When Lactobacillus plantarum is cultured in a medium containing certain fatty acids, the synthesis of fatty acid by the organism is reduced. Of the fatty acids tested, oleic and cis-vaccenic acids exert the most pronounced inhibition, but significant inhibition is also produced by palmitoleic, lactobacillic, elaidic, linoleic and eicosenoic acids. Linolenic acid, petroselenic acids, have little or no effect. The exogenous fatty acids are incorporated into the cellular lipids, thus altering the fatty acid composition.

REMOVAL OF THE 4,4-DIMETHYL CARBONS IN THE ENZYMIC CON-VERSION OF LANOSTEROL TO CHOLESTEROL; INITIAL LOSS OF THE 4a-METHYL GROUP. R. Rahman, K. Sharpless, T. Spencer and R. Clayton (Dept. of Psychiatry, Stanford Univ. School of Med., Stanford, Calif. 94305). J. Biol. Chem. 245, 2667-71 (1970). The sequence of removal of methyl substituents at C-4 of 4,4,14a-trimethyl-5a-cholesta-8,24-dien-3 $\beta$ -ol (lanosterol) in the course of its conversion to cholesterol has been reexamined. From a homogenate of rat liver incubated with DL-mevalonic acid-2-<sup>14</sup>C-5-<sup>3</sup>H, squalene, lanosterol, 4,4,14atrimethyl-5a-cholest-8-en-3 $\beta$ -ol, 4a-methyl-5a-cholest-7-en-3 $\beta$ -ol and cholesterol have been isolated. It is concluded from measurements of the <sup>3</sup>H:<sup>14</sup>C ratios found in these various compounds that the 4a-methyl group of lanosterol is removed before the 4 $\beta$ -methyl group and that the 4a-methyl group of 4a-methyl-5a-cholest-7-en-3 $\beta$ -ol originates as the 4 $\beta$ -methyl group of lanosterol.