ascorbic acid and decreased by vitamin E. Irradiation causes formation of lipid peroxides in lysosomes, and enzyme release increases with lipid peroxide formation. The authors suggest that lipid peroxide formation leads to rupture of the lysosome membrane and allows release of the contained hydrolytic enzymes.

MECHANISMS OF LIPID PEROXIDE FORMATION IN ANIMAL TISSUES. E. D. Wills. *Ibid.*, 667–76. Homogenates of rat liver, spleen, heart and kidney form lipid peroxides when incubated *in vitro* and actively catalyze peroxide formation in emulsions of linoleic or linolenic acids. In liver, catalytic activity is distributed throughout the nuclear, mitochondrial and microsomal fractions and is present in 100,000g supernatant. Activity is weak in the nuclear fraction. Ascorbic acid increases the rate of peroxidation of unsaturated fatty acids catalyzed by whole homogenates of liver, heart, kidney and spleen at pH 6.0 but not at pH 7.4. Catalysis of peroxidation of unsaturated fatty acids by the mitochondrial and microsomal fractions of liver is inhibited by ascorbic acid at pH 7.4 but the activity of the supernatant fraction is enhanced. Inorganic iron or ferritin are active catalysts in the presence of ascorbic acid. Lipid peroxide formation in linoleic or linolenic acid emulsions catalyzed by tissue homogenates is partially inhibited by EDTA but stimulated by *o*-phenanthroline. Cystein or glutathion inhibits peroxide formation catalyzed by whole homogenates, mitochondria or hemo-protein. Inhibition increases with increase of pH.

CHROMATOGRAPHIC EVIDENCE FOR THE OCCURRENCE OF OLEIC ACID METABOLITES IN ERYTHROCYTES FROM ESSENTIAL FATTY ACID-DEFICIENT RATS. B. L. Walker (Univ. of Guelph). *Arch. Biochem. Biophys.* 114, 465–71 (1966). Rats were made EFA-deficient by feeding a synthetic diet containing 10% hydrogenated coconut oil as the fat. Methyl esters, prepared from the erythrocyte lipids of these rats, were fractionated by thin-layer chromatography on silver nitrate-impregnated silica gel. The fractions, which differed in degree of unsaturation were analyzed by gas liquid chromatography. Three peaks, not previously reported in chromatograms of erythrocyte fatty acids, were detected and tentatively identified on the basis of the chromatographic data and of the metabolic interrelationships existing between the various acids of EFA-deficient animals. These acids are believed to be 18:2ω9, 22:3ω9, and 22:4ω9 (X:YωZ, where X is the number of carbon atoms in the acid, Y is the number of double bonds, and X is the number of carbon atoms after the methyl-terminal double bond). Inability to detect these compounds on chromatograms of total methyl ester mixtures is due to the similarity of their retention times with those of more commonly occurring esters.

STUDIES ON LIPOGENESIS IN VIVO. EFFECT OF DIETARY FAT OR STARVATION ON CONVERSION OF C^{14}-GLUCOSE INTO FAT AND TURNOVER OF NEWLY SYNTHESIZED FAT. G. R. Jansen, C. F. Hutchison and M. E. Zanetti (Merck Institute for Therapeutic Research). *Biochem. J.* 99, 323–32 (1966). Lipogenesis was studied *in vivo* by giving mice 250 mg meals of U-C^{14}-glucose and measuring the disposition and incorporation of label. About 48% of the label was eliminated as CO_2 in the first two hours. At 60 minutes after administration, 1.0, 1.9 and 11.9% of the administered dose was recovered as liver glycogen, liver fatty acid and carcass fatty acid, respectively. Of the labeled glucose converted into fat in the epididymal pads about 90% was present as glyceride fatty acid and 10% as glyceride glycerol. Hepatic synthesis of fatty acid was depressed by dietary fat to a much greater extent than was synthesis outside the liver. Both feeding with fat and starvation decreased the proportion of the label taken up by adipose tissue present as fat (triglyceride) and increased the proportion of triglyceride label present as glyceride glycerol. These results are consistent with the hypothesis that the primary action of both these conditions in decreasing fat synthesis is to inhibit synthesis of fatty acids. Turnover of body fat labeled *in vivo* from U-C^{14}-glucose was estimated from the decline in radioactivity measured over the first 24 hours of the experiment. The half-life of liver and extrahepatic fatty acids (excluding epididymal fat) was 16 hours and 3 days, respectively. In contrast, no measurable decrease in radioactivity of the fatty acids of epididymal fat was observed for 7 days after administration of the radioactive glucose.

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• *New Products*

LABCONO, Kansas City, Mo., has a new line of fire retardant and chemical resistant fiberglass hoods. The new hoods feature larger motors, bigger all-fiberglass blowers, automatic air by-pass and many other engineering and design improvements.

INTERNATIONAL CRYSTAL LABORATORIES, Irvington, N.J., has announced a new line of flow cells for use in UV, visible and near IR spectrophotometers. Cell path length extends from less than 1 mm to 100 mm. Cell material is available in quartz, IR transmitting quartz, and glass.

BIO-RAD LABORATORIES, Richmond, Calif., has added polyacrylamide gels with fast flow rates and improved resolution to the Bio-Gel P series of porous polyacrylamide beads. The spherical beads separate materials by differences in molecular weights when used in chromatographic columns.

KOMLINE-SANDERSON ENGINEERING CORPORATION, Peapack, N.J., has introduced a continuous screw conveyer centrifuge for continuous process application. Especially suited to crystalline, powdery and short-fibrous materials, it provides efficient liquid-solid separation with minimum retention time.

MALLINCKRODT CHEMICAL WORKS, St. Louis, Mo., has a new line of solvents for use in GLC work, column, thin-layer, and paper chromatography, with total residue-after-evaporation at about 0.5 ppm. Mallinckrodt also has new precoated TLC plates with a separation time of between 15 and 30 minutes.