METABOLISM OF UNSATURATED FATTY ACIDS IN PROTOZOA. Ann M. Lees and E. D. Korn (Lab. of Biochem., Sec. on Cellular Physiology, NHI, Nat. Inst. H. E. W., Bethesda, Maryland 20014). *Biochemistry* 5, 1475–81 (1966). *Tetrahymena pyriformis*, when grown on a fatty acid-free medium, contains only two polyunsaturated fatty acids, 9,12-octadecadienoate and 6,9,12-octadecatrienoate. When grown in the presence of 11,14-eicosadienoate, 8,11,14-eicosatrienoate, 11-eicosenoate, or 11-octadecenoate, the protozoa incorporated the fatty acids into their neutral lipids and phospholipids. Despite profound changes in fatty acid composition, the protozoa were normal in growth rate, appearance, and cell motility. Some of these fatty acids were desaturated or elongated. *T. pyriformis* incorporated 5,8,11,14-eicosatetraenoate into its lipids with nonreproducible effects on growth, rate, appearance and motility. 9,12,15-Octadecatrienoate, 6-octadecenoate, and 6,9,12-octadecatrienoate, but not 9,12-octadecadienoate, were very toxic to *T. pyriformis*. *Acanthamoeba* sp., which normally contains only ω-6-polyunsaturated fatty acids converted 9,12,15-octadetrienoate into several ω-3-polyunsaturated fatty acids which were incorporated into the lipids of the amoebae with no apparent toxicity.

THE METABOLISM OF SPHINGOMYELIN. I. J. N. Kanfer, O. M. Young, D. Shapiro and R. O. Brady (Lab. of Neurochem. Natl. Inst. of Neurological Diseases and Blindness, N.I.H., Bethesda, Md.). *J. Biol Chem.* 241, 1081–4 (1966). A sphingomyelin-cleaving enzyme has been found in rat liver tissue. The enzyme, originally present in subcellular particulate fractions, could be released in a soluble form by treatment with appropriate degents and was partially purified by conventional procedures. The most highly purifed enzyme preparations catalyzed the hydrolysis of sphingomyelin, whereas lecithin and phosphatidylethanolamine were unaffected. The products of the reaction were identified as phosphorylcholine and ceramide. Lecithin was a competitive inhibitor of the reaction.

CHANGES IN SERUM LIPID LEVELS OF HYPERLIPEMIC PATIENTS FOLLOWING THE FEEDING OF STARCH, SUCROSE AND GLUCOSE. N. A. Kaufmann, Rachel Poznanski, S. H. Blondheim and Yechezkiel Stein (Hadassah-Univ. Hosp. and Hebrew Univ.-Hadassah Med. School, Jerusalem, Israel). *Am. J. Clin Nutr.* 18, 261–9 (1966). Serum triglyceride and serum cholesterol responses to the interchange of starch with sucrose or glucose in four patients with carbohydrate-induced hypertriglyceridemia, one with the mixed type of hypertriglyceridemia and one with essential hypercholesterolemia are reported. In all cases feeding of sucrose or glucose caused a marked increase in serum triglycerides whereas feeding of starch reduced serum triglyceride levels. In general, serum cholesterol followed the same pattern as serum triglyceride. Metabolic differences between starch and di- or monosaccharides, which might explain their different effect on the blood lipids, are discussed.

INFLUENCES OF DIETARY FAT ON ALCOHOLIC FATTY LIVER. D. P. Jones and Elizabeth A. Greene (Thorndike Memorial Lab., Boston, Mass.). *Am. J. Clin Nutr.* 18, 350–7 (1966). Groups of rats were fed nutritionally adequate diets differing in fat content for three week periods. When alcohol was included in the diets as 36% of the total calories, hepatic lipids increased only when the dietary fat exceeded 20% of the total calories. When a high fat diet (43%) including alcohol was fed, liver fat increased three-to fourfold whether the dietary fat was highly saturated (coconut oil) or unsaturated (safflower oil). With the high fat diets (43%), the fatty acid composition of liver triglycerides resembled that of the diet whether alcohol was fed or not. In one volunteer subject, twice as much hepatic fat accumulated when he was fed alcohol and a high fat diet than when he was fed a low fat diet and alcohol. In both rats and man, the excess hepatic fat resulting from high fat diets and alcohol is probably derived from the diet to a significant extent.

FAILURE OF D- AND L-THYROXINE TO PROTECT CHOLESTEROL AND OIL FED COCKERELS AGAINST CORONARY ATHEROGENESIS. S. Jain, Ruth Pick, P. J. Johnson and L. N. Katz (Cardiovascular Inst. and Dept. of Cardiovascular Disease, Div. of Med., Michael Reese Hosp. and Med Center, Chicago, Ill.). *Circulation Res.* 18, 519–24 (1966). In cockerels, L-thyroxine was significantly more active biologically than an equivalent dose of D-thyroxine, even four times the dose, as demonstrated by the effects on body weights, organ weights and comb index. In cockerels fed an atherogenic diet, the plasma cholesterol concentrations in all the thyroxine-treated groups were significantly lower than in the control groups. No consistent effect of D-thyroxine on thoracic aorta atherosclerosis was observed. However, L-thyroxine had some protective action in the one experiment done.

(Continued on page 326A)

• *New Members*

Active

Dexter H. C. Beach, Plant Superintendent, Agra Vegetable Oil Products, Ltd., Nipawin, Saskatchewan, Canada.

James William Blankenship, Biochemist, International Nutrition Research Foundation, Riverside, Calif.

Paul A. Cardenas, Production Manager, Lance Hnos, S. A., Mexico, Mexico.

Raymond Caren, Research Associate, Cedars Sinai Med. Research Institute, Los Angeles, California.

Hans Ulrich Daeniker, Director of Research, Givaudan Corp., Clifton, N. J.

Robert L. Dryer, Associate Professor in Biochemistry, University of Iowa, Iowa City, Iowa.

Hardy Malcom Edwards, Jr., Associate Professor, University of Georgia, Athens, Ga.

James Byrd Edwards, Chemical Engineer, Procter and Gamble Co., Cincinnati, Ohio.

George Frankl, Chemist, Hunt Foods & Industries, Fullerton, Calif.

John Philip Friedrich, Principal Chemist, Northern Regional Research Laboratories, Peoria, Ill.

Charles Ted Gammon, Chemist, Drew Chemical Corp. Boonton, N.J.

Shimon Gatt, Senior Lecturer, Hebrew University, Hadassan Medical School, Jerusalem, Israel.

Peter William Gilderson, Technologist, Shell Chemical Co., New York, N.Y.

Thomas Grier, Chemist, U.S. Public Health Service, Heart Disease Control, San Francisco, Calif.

Gerald W. Hart, Plant Chem. Engineer, Procter and Gamble Mfg. Co., Chicago, Ill.

Gerald R. Hegarty, Head, Meat Research Section, Armour and Co., Oak Brook, Ill.

Arthur Mitchell Hochhauser, Chemists, DCA Food Industries, New York, N.Y.

William H. Jennings, Self Employed, Consultant, Norfolk, Va.

Dale W. Johnson, Vice President, Soypro International Inc., Chicago, Ill.

Reino Emil Kallio, Director of Life Sciences, School of Life Science, University of Illinois, Urbana, Ill.

William L. Kohlhase, Principal Chemist, USDA Northern Regional Research Laboratory, Peoria, Ill.

Sherman G. Lee, Chemist, Durkee Famous Foods, Berkeley, Calif.

Gummuluri V. A. Pantulu, Reserach Fellow, University of Minnesota, Minneapolis, Minn.

James Bristow McLeod Rattray, Associate Professor of Chemistry, University of Guelph, Guelph, Canada.

John Gray Robertson, Scientific Officer, Plant Chemistry Division, D.S.I.R., Palmerston, New Zealand.

James W. Ryder, Technical Advisor Marketing, Humble Oil Refining Co., Los Angeles, Calif.

Charles Waldo Smith, Chief Chemist, Consumer Products, Corn Products Co., Argo, Ill.

Vincent Anthony Stallone, Production Superintendent, Glyco Chemicals, Inc., Painesville, Ohio.

Haruo Uzawa, Assistant Professor, Kyushu University, Fukuoka, Japan.

Individual Associate

Salih J. Wakil, Professor of Biochemistry, Duke University, Durham, N.C.

George J. Wetterholt, Chief Chemist, D. A. Stuart Oil Co., Ltd., Somerville, N. J.

Dao Lip Lee, Technical Director, Industria e Comercio Lotus S. A., Sao Paulo, Brasil.

Active Junior (first year free)

Shukri M. El-Khatib, Graduate Student, Texas A & M University, College Station, Texas.

David Graham Guy, Graduate Student, Ohio State University, Columbus, Ohio.

Barbara Jeanne Zook, Michigan State University, East Lansing, Mich.