• *New Members*

Active

George M. Adams, Biophysicist, U.S. Navy, Naval Medical Research Institute, Bethesda, Md.

Walter D. Alvin, Chief Cosmetic Chemist, Les Parfumes de Dana (Dana Perfumes Corp.), Mountaintop, Pa.

Joseph P. Bain, Manager, Development and Technical Services, Union Camp Corp., Savannah, Ga.

Richard J. Bloomfield, Manager, Quality Control, Emery Industries, Santa Fe Springs, Calif.

Clarence J. Broussard, Chemist, Supervisor in Analytical Services, Carnation Research Laboratories, Van Nuys, Calif.

William E. Cornatzer, Professor and Head of Department Biochemistry, Director, Ireland Research Laboratory, University of North Dakota, Medical School, Grand Forks, N.D.

Hector J. De Muelenaere, Controller, Research Laboratory, Hind Bros. & Co. Ltd., Durban, South Africa.

Robert C. Edmondson, Assistant to Manager of Manufacturing, Swift & Co., Refinery and Margerine Department, Chicago, Ill.

Tom F. Fite, Foreman, Anderson, Clayton & Co., Jacksonville, Ill.

Leonard J. Gigilo, Director of Research, George W. Luft (House of Tangee) Long Island City, N.Y.

Arley L. Goodenkauf, Chemist, Metropolitan Utilities District, Omaha, Neb.

James B. Himes, Administrative Assistant to the Director of Research and Development, The Richardson Company, Melrose Park, Ill.

Hildegard I. Hof, Post-doctoral fellow, University of Illinois, Department of Neurology, Presbyterian-St. Lukes Hospital, Chicago, Ill.

Ching-Tseng Keng, Mill Manager, Malayan Flour Mills Ltd., Lumut, Perak, Malaysia.

Maxwell R. Kleeman, Sales Manager, Union Carbide Corp., New York, N.Y.

Josef-Peter Leiher, Research Chemist, Emery Industries, Inc., Cincinnati, Ohio.

Thomas W. McGinnis, Chemical Engineer, Lever Bros., Research Division, Edgewater, N.J.

Roy E. Martin, Manager, Vegetable Protein Products, Swift & Co., Chicago, Ill.

Harry J. Mersmann, Research Associate, State University at Buffalo New York, Buffalo, N.Y.

Darle L. Nieneker, Technical Service Representative, Jefferson Chemical Co. Inc., Austin, Texas.

Leonard H. Ponder, Research Chemist, American Enka Corporation, Enka, N.C.

Bengt I. Samuelsson, Professor, Department Medical Chemistry, Royal Veterinary College, Stockholm 50, Sweden.

Thomas L. Scott, Chemical Engineer, A. E. Staley Manufacturing Co., Decatur, Ill.

Lauren C. Stenroos, Biochemist, Minneapolis V. A. Hospital, Minneapolis, Minn.

David M. Takahashi, Chemist, Food & Drug Administration, Department of Health, Education and Welfare, San Francisco, Calif.

Frank M. Yatsu, Assistant Professor of Neurology, University of California School of Medicine, San Francisco, Calif.

Individual Associate

Richard Flower, 590 Shenandoah Drive, Port Credit, Ontario, Canada.

Karl W. Klein, Sales Engineer, DeLaval Separator Co., Poughkeepsie, N.Y.

John F. Kraker, Vice President, Polyester Corporation, New York, N.Y.

Active Junior

Ching-kuang Chow, Graduate Student, University of Illinois, Urbana, Ill.

William R. Karski, Graduate Student, Research Assistant, Rutgers University, New Brunswick, N.J.

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HEPATIC TRIGLYCERIDE SECRETION IN RELATION TO LIPOGENESIS AND FREE FATTY ACID MOBILIZATION IN FASTED AND GLUCOSE-REFED RATS. N. Baker, A. S. Garfinkel and M. C. Schotz (Radioisotope Res., Veterans Adm. Center, Los Angeles, Calif. 90073). *J. Lipid Res.* **9**, 1–7 (1968). Plasma triglyceride concentrations were significantly lowered by a single feeding of glucose to rats that had been fasted for 22 hr. Three feedings of glucose produced a similar effect. In the glucose-refed animals mobilization of free fatty acids from adipose tissue was impaired more rapidly than hepatic lipogenesis was restored from its low fasting level. These effects of glucose were shown by both a 50% fall in plasma free fatty acid concentration and an 84% decrease in free fatty acid release by isolated epididymal fat pads within 30 min after a single refeeding of glucose. Hepatic lipogenesis from either acetate-1-^{14}C or glucose-U-^{14}C was not restored even after glucose had been fed three times at hourly intervals. Triton-induced hypertriglyceridemia was used to measure the hepatic triglyceride secretary rate; it was found that glucose refeeding decreased this rate in all but one of several experiments. This decreased secretion rate was sufficient to account for the nearly complete disappearance of triglyceride in very low density lipoproteins ($d < 1.019$) that occurred within 1 hr after a single glucose intubation.

BILE SALT EVOLUTION. G. A. D. Haslewood (Guy's Hospital Med. School, London S.E. 1, England). *J. Lipid Res.* **8**, 535–50 (1967). Views against the background of known or supposed biosynthetic pathways for cholic and chenodeoxycholic acids in man and laboratory animals, the chemical nature of bile salts in more primitive animals clearly indicates that evolution from C_{27},5α-alcohol sulfates to C_{24},5β-acids has taken place. Stages in this evolution, some of which are intermediates in the biosynthesis of C_{24} bile acids, are described for representatives of all the chief vertebrate groups. "Unique" primary C_{24} bile acids may be considered as hydroxylated chenodeoxycholic acids; the possible taxonomic significance of these is discussed. A closer study of the biochemical mechanisms underlying bile salt differences may be expected to throw new light on the nature of the evolutionary process itself.

PHOSPHATIDYL GLYCEROPHOSPHATE PHOSPHATASE. Y. Chang and E. P. Kennedy (Dept. Biol. Chem., Harvard Med. School, Boston, Mass.). *J. Lipid Res.* **8**, 456–62 (1967). An enzyme (phosphatidyl glycerophosphate phosphatase) that catalyzes the formation of phosphatidyl glycerol from phosphatidyl glycerophosphate has been rendered soluble by treatment of the particulate fraction of *E. coli* with Triton-X-100 in the presence of EDTA, and has been partially purified. The enzyme is specific for phosphatidyl glycerophosphate and does not catalyze the hydrolysis of other simple phosphomonoesters. It required Mg^{++} for activity and is inhibited by sulfhydryl agents. Some other properties of the enzyme are also described.

BIOSYNTHESIS OF PHOSPHATIDYL GLYCEROPHOSPHATE IN ESCHERICHIA COLI. *Ibid.*, 447–55. The enzyme, devoid of phosphatidyl glycerophosphatase activity is specific for L-glycerol 3-phosphate and is completely dependent upon added Mg^{++} or Mn^{++} for activity. It has high affinity for CDP-diglyceride and can be used for the assay of this nucleotide. Other properties of the enzyme are also described.

SEPARATION AND SIZE DETERMINATION OF HUMAN SERUM LIPOPROTEINS BY AGAROSE GEL FILTRATION. S. Margolis (Depts. of Med. and Physiol. Chem., The Johns Hopkins Univ. School of Med., Baltimore, Md.). *J. Lipid Res.* **8**, 501–07 (1967). A method is described for the separation of the three major classes of human serum lipoproteins by gel filtration on columns of 4 and 6% agarose gel. After calibration of the columns, the elution volumes of the lipoproteins were used to calculate the molecular sizes and molecular weights of these macromolecules. The technique was employed to demonstrate aggregation of low density lipoprotein following partial delipidation, partial proteolysis, or mild heat denaturation. Agarose gel filtration shows promise as a useful method for the isolation, purification, and characterization of liproproteins.

FATTY LIVER INDUCED BY INJECTION OF L-TRYPTOPHAN. Yukiko Hirata, T. Kawachi and T. Sugimura (Biochem. Div., National Cancer Center Res. Inst., Tsukiji, Chuo-ku, Tokyo). *Biochim. Biophys. Acta* **144**, 233–41 (1967). L-Tryptophan caused the accumulation of neutral lipids in liver within 2.5 hr. after its intraperitoneal injection into rats. This accumulation of neutral lipids continued for about 24 hr. Peripheral fatty liver was diagnosed histologically by Sudan III staining. The minimal effective dose was 0.5 mg/g of body weight. The level of