Abstracts

EDITOR: F.A. Kummerow

ABSTRACTORS: J.C. Harris, M.A. Kokatnur, F.A. Kummerow, G. List, B. Matijasevic, R.A. Reiners, and P.Y. Vigneron

Biochemistry and nutrition

INTERACTION OF APOLIPOPROTEIN B FROM HUMAN SERUM LOW-DENSITY LIPOPROTEIN WITH EGG YOLK PHOSPHATIDYLCHOLINE. R.M. Watt and J.A. Reynolds (Dept. of Physiology, Duke University Medical Center, and Whitehead Medical Res. Institute, Durham, North Carolina 27710) *Biochemistry 20*(13): 3897-3901 (1981). A binary complex of apolipoprotein B and egg yolk lecithin has been formed which contains 250-350 mol of lipid/500,000 g [*sic*] of protein. This particle retains many of the structural properties of native human low-density serum lipoprotein (LDL) as evidenced by the state of association of the protein, the circular dichroic spectrum, and immunological characteristics. Apolipoprotein B does not interact with lipid vesicles but rather binds a small number of phospholipid molecules in water-soluble form. This study represents the first partial reconstitution of native LDL from the delipidated apoprotein and is the initial step in a systematic investigation of the lipid binding properties of apolipoprotein B.

ENZYMIC SYNTHESIS OF STEROID SULPHATES. XIV. PROPERTIES OF HUMAN ADRENAL STEROID ALCOHOL SULPHOTRANSFERASE. J.B. Adams and D. McDonald (Sch. of Biochem., Univ. of New South Wales, Kensington, N.S.W., Australia) *Biochim. Biophys. Acta 664*:460-468 (1981). Pure steroid alcohol sulphotransferase (EC 2.8.2-) has the property of sulphurylating hydroyxl groups on different positions of the steroid ring. It has now been established that although only monosulphates are formed from substrates such as 3,17-diols, the position of the sulphate group depends on the relative configuration of the hydroxyl groups. The enzyme exhibits non-Michaelis-Menten kinetics within physiological concentrations (0-1 μM) of the substrate dehydroepiandrosterone and evidence was obtained for the presence of multiple interacting steroid-binding sites. A regulatory role for the enzyme in the secretion of dehydroepiandrosterone from the human adrenal gland is proposed.

HEPATIC 3-HYDROXY-3-METHYLGLUTARYL COENZYME A REDUCTASE ACTIVITY AND BILIARY LIPID COMPOSITION IN MAN: RELATION TO CHOLESTEROL GALLSTONE DISEASE AND EFFECTS OF CHOLIC ACID AND CHENODEOXYCHOLIC ACID TREATMENT. J. Ahlberg, B. Angelin and K. Einarsson (Departments of Medicine and Surgery, Karolinska Institutet at Serafimerlasarettet and Huddinge University Hospital, Stockholm, Sweden) *J. Lipid Res. 22*(3):410-422 (1981). The present work was undertaken in order to study whether or not there is a relation between hepatic HMG CoA reductase, hepatic cholesterol concentration, and biliary lipid composition. In 55 patients a liver biopsy together with gallbladder and hepatic bile were obtained at laparotomy under standardized conditions. Of the gallstone patients, twelve have been treated with cholic acid and ten with chenodeoxycholic acid in a dose of 15 $mg \cdot kg^{-1} \cdot d^{-1}$ for 6-8 weeks prior to operation. Treatment with cholic acid reduced the cholesterol saturation of hepatic bile, although supersaturation persisted. During chenodeoxycholic acid treatment, hepatic bile became unsaturated in most of the patients. Hepatic cholesterol concentration was about 20% higher in patients with cholesterol gallstone disease than in gallstone-free controls. During treatment with cholic acid or chenodeoxycholic acid, hepatic cholesterol concentration was normalized. In gallstone-free controls and in bile acid-treated but not in untreated gallstone patients, saturation of hepatic bile correlated with HMG CoA reductase activity. It is concluded that treatment with chenodeoxycholic acid but not with cholic acid results in saturated hepatic bile. This unsaturation may in part be explained by a decreased hepatic HMG CoA reductase activity.

INFLUENCE OF DIETARY FAT ON GROWTH AND LIVER LIPID CONTENT, GLUCOSE 6-PHOSPHATE AND 6-PHOSPHOGLUCONATE DEHYDROGENASES, AND ALDOLASE ACTIVITIES IN THE CHICK. A.I. Akinwande (Dept. of Biochem., Col. of Med. of the Univ. of Lagos, Lagos, Nigeria) *Poultry Sci. 60*:1259-1263 (1981). The influence of fat in chick diets containing 18, 21 and 24% protein on growth and liver lipid content, glucose 6-phosphate, and 6-phosphogluconate dehydrogenases, and aldolase activities was evaluated. Twelve percent fat in diets containing 21 and 24% protein increased the rate of growth. The fat level also decreased liver lipid content at 24% dietary protein. Improved growth was attributed to higher feed intake. However, dietary fat did not affect the activities of the above enzymes indicating their inadaptive nature to dietary fat.

PHOSPHOLIPID SYNTHESIS IN FETAL LUNG ORGANOTYPIC CULTURES AND ISOLATED TYPE II PNEUMOCYTES. A.S. Anian, S.A. Kaplan and C.T. Barrett (Dept. of Ped., U.C.L.A., Sch. of Med., Los Angeles, CA) *Biochim. Biophys. Acta 664*:498-512 (1981). Type II pneumocytes from fetal rabbit lungs were grown in an organotypic system and used to study surfactant phospholipid synthesis. This organotypic system was further employed as a means for isolating purified type II cells which were grown in monolayer cultures. Phospholipid synthesis properties for these purified type II cells at different stages of culture were studied using the radioactively labeled substrates: palmitate, choline, and acetate. These observations indicate that the organotypic system is a useful model for examining fetal lung surfactant phospholipid synthesis and may also be employed as a simple means for isolating fetal type II pneumocytes.

ACTIVATION OF ADENYLATE CYCLASE OF RAT BRAIN BY LIPID PEROXIDATION. A. Baba, E. Lee, A. Ohta, T. Tatsuno, and H. Iwata (Dept. of Pharm., Faculty of Pharm. Sci., Osaka Univ., Osaka, Japan) *J. Bio. Chem. 256*(8):3679-3684 (1981). The relationship between adenylate cyclase activity in the synaptic membrane fraction (M_1) of rat brain and lipid peroxidation of these membranes was examined. In the presence of 5 mM dithiothreitol (DTT), 1 to 10 μM Fe^{2+} activated adenylate cyclase 2- to 4-fold. Of several metal ions, Fe^{2+} was the most effective. Other enzymes in M_1, such as Mg^{2+}-ATPase, $(Na^+\text{-}K^+)$-ATPase, 5'-nucleotidase, acetylacholinesterase, and phosphodiesterase, were not activated by Fe^{2+} plus DTT. Activation of adenylate cyclase by Fe^{2+} plus DTT was accompanied by production of malondialdehyde, a product of lipid peroxidation. Formation of malondialdehyde was completely parallel with enzyme activation. Ascorbic acid or a NADPH system also stimulated enzyme activity and caused lipid peroxidation. These results indicate that lipid peroxidation of synaptic membranes was accompanied by specific stimulation of adenylate cyclase activity.

EFFECT OF DIETARY ENERGY, ENVIRONMENTAL TEMPERATURE, AND SEX OF MARKET BROILERS ON LIPOPROTEIN COMPOSITION. W.L. Bacon, A.H. Cantor and M.A. Coleman (Dept. of Poultry Sci., Ohio Ag. Res. and Development Center, Wooster, OH) *Poultry Sci. 60*:1282-1286 (1981). Broiler chicks were fed starter diets (days 1 to 28) containing 3.09, 3.20, or 3.31 kcal ME/g, grower diets (days 29 to 43) containing 3.14, 3.25, or 3.36 kcal ME/g, and then finisher diets (days 44 to 49) containing 3.20, 3.31 or 3.42 kcal ME/g, respectively. For each dietary treatment there were two replicate floor pens containing 78 male and 78 female chicks in a cool environment and two replicate pens in a warmer environment. TLC and TG levels were significantly elevated in females fed the highest energy level. No other dietary effects were observed.

ANALYTICAL FRACTIONATION OF HUMAN LIVER MICROSOMAL FRACTIONS: LOCALIZATION OF CHOLESTEROL AND OF THE ENZYMES RELEVANT TO ITS METABOLISM. S. Balasubramaniam, K.A. Mitropoulos, S. Venkatesan, N.B. Myant, T.J. Peters, A. Postiglione and M. Mancini (Med. Res. Council Lipid Met. Unit, Hammersmith Hosp., London) *Clin. Sci. 60*:435-439 (1981). The submicrosomal distribution of three enzymes concerned in cholesterol metabolism, and of free and esterified cholesterol, was determined in human liver by analytical isopycnic centrifugation on sucrose gradients. Most of the free cholesterol in the microsomal preparations was present in smooth membranes from the Golgi apparatus and in vesicles from plasma-membrane fragments. The distribution of esterified cholesterol was multimodal and extended throughout the whole gradient.

CONCENTRATION OF CALCIUM, PHOSPHORUS, AND 1,25-DIHYDROXYVITAMIN D IN PLASMA OF DAIRY COWS DUR-

ING THE LACTATION CYCLE. B.A. Barton, R.L. Horst, N.A. Jorgensen and H.F. DeLuca (Dept. of Dairy Sci. and Biochem., Univ. of Wis., Madison, WI) *J. Dairy Sci. 64*:850-852 (1981). Concentration of calcium, phosphorus, and 1,25-dihydroxyvitamin D in plasma of four young, four nonparetic aged, and four paretic aged cows were measured during the lactation cycle. Concentration of 1,25-dihydroxyvitamin D in plasma was elevated significantly in paretic aged cows as compared to non-aretic aged and young cows from the day of calving to 3 days postcalving. In paretic aged cows, severe hypocalcemia and hypophosphatemia developed on the day of calving, whereas hypocalcemia and hypophosphatemia were only transient in nonparetic aged and young cows at this time. Changes were only minor in concentration of 1,25-dihydroxyvitamin D in plasma from 7 days postcalving to 7 days precalving in all cows.

A COMPARISON OF BILE SALT BINDING TO LYMPH AND PLASMA ALBUMIN IN THE RAT. G.J. Beckett, P. Armstrong and I.W. Percy-Robb (Dept. of Clin. Chem., Univ. of Edinburgh Med. Sch., Royal Infirmary, Edinburgh, U.K.) *Biochim. Biophys. Acta 664*:602-610 (1981). The binding of bile salts to proteins in rat plasma and rat lymph has been investigated. Under the non-equilibrium conditions of gel chromatography no binding of glycochenodeoxycholate or glycocholate to any of the lymph proteins was observed. In contrast, plasma bound a proportion of both bile salts. When lymph was treated with charcoal, binding of glycochenodeoxycholate to a protein with a molecular weight identical to albumin occurred. Equilibrium binding studies showed that the binding of glycocholate to partially purified plasma albumin exhibited saturation kinetics with a dissociation constant of $2 \cdot 10^{-4}$ M. In contrast, the binding of glycocholate to lymph albumin was nonsaturable. It is suggested that the high free fatty acid concentrations found in lymph inhibit the binding of bile salts to albumin.

PLASMA LIPOPROTEIN CHANGES RESULTING FROM IMMUNOLOGICALLY BLOCKED LIPOLYSIS. S.R. Behr, J.R. Patsch, T. Forte and A. Bensadoun (Dept. of Nutritional Biochemistry, Cornell University, Ithaca, NY 14853) *J. Lipid Res. 22*(3): 443-451 (1981). The role of lipoprotein lipase (LPL) in the generation of low density lipoprotein (LDL) and high density lipoprotein (HDL) was investigated. Intravenous injections of high titer goat antiserum against highly purified chicken LPL into fasted roosters quantitatively blocks the removal of plasma VLDL triglyceride. Analyses of the chemical components of lipoproteins after 8 hr of LPL inhibition showed that the very low density lipoprotein (VLDL) concentration increased over 10-fold, while LDL and HDL concentrations decreased by 5-fold and 48%, respectively. LDL and HDL cholesterol levels decreased logarithmically over the 8-hr period. The Stokes' radii of HDL were determined by gel filtration on Biogel A5M and Ultrogel AcA 22: the radius of experimental HDL (44.9 Å) was smaller than that of control HDL (55.4 Å). These measurements were confirmed by electron microscopy (43 and 54 Å, respectively). After rate zonal ultracentrifugations of plasma samples, control LDL was clearly resolved, while no LDL could be detected in the experimental samples. Rate zonal ultracentrifugations of plasma samples also indicated that control HDL had a higher flotation rate than experimental HDL. Equilibrium zonal ultracentrifugation showed experimental HDL to be more dense than control HDL with hydrated densities of 1.118 and 1.113 g/ml, respectively. These experiments provide in vivo evidence that LDL is a direct metabolic product of VLDL and that LPL plays a role in the transfer of surface constituents from VLDL to HDL.

MECHANISM OF ACTIVATION OF GLUCOCEREBROSIDASE BY CO-β-GLUCOSIDASE (GLUCOSIDASE ACTIVATOR PROTEIN). S.L. Berent and N.S. Rarin (Mental Health Res. Inst. and Dept. of Biol. Chem., Univ. of MI, Ann Arbor, MI) *Biochim. Biophys. Acta 664*:572-582 (1981). The nature of the stimulatory action of the protein "coglucosidase" on glucocerebrosidase was investigated with the use of highly purified cofactor from bovine spleen, radioactive glucosyl ceramide and methylumbelliferyl-β-glucoside. A complex between coglucosidase and either substrate could not be detected under equilibrium and non-equilibrium binding conditions. Complex formation between stimulating protein and the enzyme could be shown by the binding of the enzyme to an affinity column containing coglucosidase. This binding could be blocked by adding phosphatidylserine to the enzyme. The lipid also stimulated the enzyme. Additional evidence for binding of the enzyme to the two kinds of stimulators was the finding that they protected the enzyme against inactivation by *N*-ethylmaleimide and chloromercuriphenylsulfonate. It is proposed that lipids, particularly acidic ones, act on solubilized glucocerebrosidase to produce an enzyme conformation which allows binding and stimulation by coglucosidase. At higher lipid concentrations, the acidic lipids bind, in competition with glucosidase, to the latter's binding site on the enzyme.

ESSENTIAL FATTY ACID DEFICIENCY: EFFECTS OF CROSS-FOSTERING MICE AT BIRTH ON BRAIN GROWTH AND MATURATION. S.E. Berkow and A.T. Campagnoni (Univ. of Maryland, Nutr. Sci. Program, Dep. of Chem., College Park, MD) *J. Nutr. 111*(5):886-894 (1981). The effects of brain development of cross-fostering mice at birth from essential fatty acid (EFA)-deficient to EFA-sufficient diets and vice versa were examined. Four groups of animals were studied: (C), animals reared on an EFA-sufficient diet throughout pre- and postnatal life; (D), animals reared on an EFA-deficient diet throughout pre- and postnatal life; (C→D), animals receiving an EFA-sufficient diet prenatally and cross-fostered to an EFA-deficient diet at birth; and (D→C), animals receiving an EFA-deficient diet prenatally and cross-fostered to an EFA-sufficient diet at birth. Indices of brain growth (i.e. wet weight, DNA and protein content) and mylination (brain proteolipid and galactolipid content) were measured on animals ranging in age from 0 to 9 weeks in the four experimental groups. These results suggest that mice "rehabilitated" from EFA-deficient to EFA-sufficient diets at birth may possess abnormal myelin for at least 2 months during a period of rapid brain maturation.

CARBONYL COMPOUNDS IN COW AND BUFFALO MILK FAT. G.S. Bhat, M.K.R. Murthy and M.B. Rao (Southern Regional Station, Nat. Dairy Res. Inst., Bangalore, India) *J. Dairy Sci. 64*:588-593 (1981). Cow and buffalo milk fat were analyzed for carbonyls over 1 yr to study the interrelationship among classes of carbonyls. Correlation between total carbonyls and ketoglycerides was positive. Monocarbonyl content of buffalo milk fat was higher than that of cow milk fat. Cow milk fat contained higher quantities (nearly twice) of β-ketoglycerides than buffalo milk fat. The percentage proportion of methyl ketones was much higher in cow than in buffalo milk fat. Colostral fat contained about 65 to 70% of carbonyls in normal milk fat. Monocarbonyls increased rapidly during the early period of lactation and gradually during the remaining period. However, ketoglycerides remained steady after a rapid increase during the early part of lactation.

DETERMINATION OF CHOLESTEROL ASYMMETRY BY RAPID KINETICS OF FILIPIN-CHOLESTEROL ASSOCIATION: EFFECT OF MODIFICATION IN LIPIDS AND PROTEINS. R. Bittman, L. Blau, S. Clejan and S. Rottem (Dept. of Chemistry, Queens College of the City University of New York, Flushing, NY 11367) *Biochemistry 20*(9):2425:2432 (1981). The rapid kinetic behavior of filipin association with cholesterol was unaffected by binding of water-soluble proteins to vesicles and mycoplasma membranes and by proteolytic digestion of mycoplasma membrane proteins. The kinetic properties were, however, dependent on the membrane phospholipids, in that the initial rate of filipin association with cholesterol was enhanced by phospholipase A_2 treatment, by the incorporation of lysophosphatidylcholine, and by increasing the degree of unsaturation in phospholipid vesicles and mycoplasma membranes. The second-order rate constant was also dependent on the mol% of cholesterol in small unilamellar vesicles but not in large unilamellar vesicles. The ratio of rate constants in intact mycoplasma cells relative to isolated membranes provides an estimate of cholesterol distribution in membranes. This ratio was unaffected by proteolytic digestion of intact cells and by the incorporation of exogenous phospholipids into the *Mycoplasma capricolum* cell membrane. The results of these experiments are discussed in relation to the use of the rapid kinetics of filipin binding as a probe of cholesterol distribution.

EFFECTS OF STEROLS ON PERMEABILITY AND PHASE TRANSITIONS OF BILAYERS FROM PHOSPHATIDYLCHOLINES LACKING ACYL GROUPS. R. Bittman, S. Clejan, M.K. Jain, P.W. Deroo, and A.F. Rosenthal (Dept. of Chem., Queens Col. of the City Univ. of New York, Flushing, NY) *Biochemistry 20*: 2790-2795 (1981). Sonicated vesicles were prepared from synthetic phosphatidylcholines lacking acyl groups. The phospholipids used were diether phosphatidylcholines bearing 1,2-ditetradecyl, 1,2-dihexadecyl, and 1-*cis*-9'-octadecenyl and 2-hexadecyl chains and akkyl analogues such as (2-octadecyl-eicosyl)phosphorylcholine, which has no diacylglycerol or glycerol diether moiety. The effect of increasing cholesterol concentrations on the thermotropic behavior of (2-octadecyleicosyl)-phosphorylcholine was not altered in its analogues having increased steric bulk in the polar head group region, suggesting that no direct or specific PC polar head group-cholesterol interaction occurs.

SERUM TRIGLYCERIDES, TO BE OR NOT TO BE A RISK FACTOR FOR ISCHAEMIC HEART DISEASE? L.A. Carlson and L.E. Böttiger (King Gustaf V Res. Inst. and Dept. of Intern. Med., Karolinska Hosp., Stockholm, Sweden) *Atherosclerosis 39*:287-291 (1981). The Stockholm Prospective Study—in a 14.5 year follow-up of 3486 men—found plasma triglycerides but not plasma cholesterol to be an independent risk factor for ischaemic heart disease. This finding stands in sharp contrast to the oppposite results of the 8.5 year follow-up of the Western Collaborative Study.

Differences between the two studies are discussed as one way of explaining the varying results—the most important probably being the use of different end-points for the diagnosis of ischaemic heart disease, but geographical, environmental and ethnic differences may also be of importance.

THE UTILIZATION BY RABBIT AORTA OF CARBOHYDRATES, FATTY ACIDS, KETONE BODIES AND AMINO ACIDS AS SUBSTRATES FOR ENERGY PRODUCTION. K.V. Chace and R. Odessey (Dept. of Physio., Sch. of Med., Univ. of Vir., Charlottesville, VA) *Cir. Res. 48*(6):850-858 (1981). The ability of rabbit aorta to oxidize various substrates was studied to determine which of these compounds may be energy substrates for vascular smooth muscle (VSM). Glucose, ketone bodies, medium-chain length fatty acids, branched-chain amino acids, and glutamine all are oxidized at comparable rates on a molar basis. Some other amino acids, long chain fatty acids, pyruvate and glycerol also are oxidized, but at lower rates. The oxidation of 6 amino acids could not be detected. The oxidation rate of all the exogenous substrates together is calculated to account for less than half of the oxygen consumption; this finding indicates that an exogenous substrate must also be utilized.

EVIDENCE INDICATING THAT INACTIVATION OF 3-HYDROXY-3-METHYLGLUTARYL COENZYME A REDUCTASE BY LOW DENSITY LIPOPROTEIN OR BY 25-HYDROXYCHOLESTEROL REQUIRES MEDIATOR PROTEIN(S) WITH RAPID TURNOVER RATE. T.-Y. Chang, J.S. Limanek and C.C.Y. Chang (Dept. of Biochem., Dartmouth Med. Sch., Hanover, NH) *J. Biol. Chem.* The half-life ($t_{1/2}$) of 3-hydroxy-3-methylglutaryl coenzyme A (HMG-CoA) reductase of Chinese hamster ovary cells grown in fetal calf serum medium is approximately 2 h. When cells are switched to grow in delipidated serum medium (DeL-M) for more than 24 h, the $t_{1/2}$ of the enzyme is found to be drastically latered to approximately 13 h. Exposure of low density lipoprotein (LDL) (100 μg of protein/ml) or 25-hydroxycholesterol (1 μg/ml) to cells grown in DeL-M suppresses reductase activity more rapidly than would be expected solely if reductase synthesis were suppressed, showing that inactivation of reductase activity by sterols, previously demonstrated using only analogs of cholesterol, is a normal mechanism for regulation of HMG-CoA reductase activity by the physiologically important sterol source (LDL).

EFFECT OF DIETARY FAT ON PROTEIN INTAKE REGULATION IN YOUNG OBESE AND LEAN MICE. K.M. Chee, D.R. Romsos and W.G. Bergen (Depts. of Food Sci. and Human Nutr., and An. Sci., MI State Univ., East Lansing, MI) *J. Nutr. 111*(4):668-677 (1981). Young female obese (ob/ob) and lean mice were allowed to self-select from two diets varying in protein and carbohydrate, protein and fat or carbohydrate and fat for 36 days. Obese and lean mice offered a choice between two diets varying in protein and carbohydrate consumed 35 and 30%, respectively, or energy from protein. When two diets varying in protein and fat were fed, both obese and lean mice initially self-selected a higher percentage of energy from protein than when diets varying in protein and carbohydrate were fed. This pattern was rapidly reversed in lean mice and more gradually reversed in obese mice. In summary, replacement of dietary carbohydrate with fat lowered the percentage of energy self-selected as protein. Obese mice, however, continued to consume more energy and more protein than lean mice.

ZINC DEFICIENCY-INDUCED CHANGES IN THE COMPOSITION OF MICROSOMAL MEMBRANES AND IN THE ENZYMATIC REGULATION OF GLYCEROLIPID SYNTHESIS. S. Clejan, V.T. Maddaiah, M. Castro-Magna and P.J. Collipp (Nassau County Med. Center, Dept. of Ped., East Meadow, NY) *Lipids 61*(6):454-460 (1981). The effects of zinc deficiency and/or castration on the lipid composition of microsomal membranes of liver, small intestine and testes were studied in rats. The results showed that feeding a zinc-deficient diet to castrated rats decreased phospholipid content and consequently increased the cholesterol-to-phospholipid ratio in liver microsomes. An increase in cholesterol-to-phospholipid ratio occurred also in small intestine and testis microsomes from rats fed the zinc-deficient diet. It is postulated, therefore, that zinc deficiency alters the lipid composition and fluidity of microsomal membranes.

EFFECT OF INCUBATION OF GUINEA-PIG TAENIA COLI IN POTASSIUM-FREE MEDIA ON ARACHIDONATE RELEASE AND LIPID METABOLISM. R.F. Coburn, M. Cunningham and J.F. Strauss, III (Department of Physiology, University of Pennsylvania School of Medicine, Philadelphia, PA 19104) *Biochim. Biophys. Acta 664*(1):188-199 (1981). We studied effects of immersion of guinea-pig taenia coli strips in potassium-free media on arachidonate stores and other lipid fractions. Control studies obtained with the strips in Krebs solution showed that greater than 97% of arachidonate was found esterified in phospholipid with the following distribution: phosphatidylethanolamine > phosphatidylcholine > phosphatidylserine plus phosphatidylinositol. 30 min incubation of the strips with [^{3}H] arachidonate complexed to albumin resulted in incorporation of this isotope into phospholipid and neutral lipid fractions, phosphatidylcholine > phosphatidylethanolamine. 30 min incubations with $^{32}PO_4^{2-}$ resulted in isotope incorporation into phospholipids, phosphatidylcholine > phosphatidylserine plus phosphatidylinositol > phosphatidylethanolamine. Arachidonate specific activity fell and arachidonate content increased in the phosphatidylserine plus phosphatidylinositol fraction. We conclude that exposure of taenia coli to potassium-free media activates turnover of phosphatidylinositol, which results in release of arachidonate.

THE EFFECT OF FEEDING HIGH ENERGY DIETS CONTAINING SUPPLEMENTAL FAT OR BROILER WEIGHT GAIN, FEED EFFICIENCY, AND CARCASS COMPOSITION. C.N. Coon, W.A. Becker and J.V. Spencer (Dept. of Animal Sci. and Food Sci., Washington State Univ., Pullman, WA) *Poultry Sci. 60*:1264-1271 (1981). We studied the effect of feeding two levels of energy in starter diets (3135 kcal metabolizable energy (ME)/kg low energy and 3410 kcal ME/kg high energy) from 0 to 28 days and finishing diets (3190 kcal ME/kg low energy and 3465 kcal ME/kg high energy) from 28 to 56 days upon broiler weight gain, feed conversion, feed consumption, calorie consumption, and abdominal fat content. A sex-diet interaction was observed ($P<.05$) for regressed abdominal fat weight on live body weight.

BIOCHEMICAL EVIDENCE FOR A CYTOPLASMIC 1α,25-DIHYDROXYVITAMIN D_3 RECEPTOR-LIKE PROTEIN IN RAT YOLK SAC. J.L. Danan, A.C. Delorme and P. Cuisinier-Gleizes (Unité de Recherches sur le Métabolisme Hydro-Minéral, Institut National de la Santé et de la Rocherche Médicale U. 120, 44 Chemin de Ronde, 78110 Le Vésinet, France) *J. Biol. Chem. 256*(10):4847-4850 (1981). The yolk sac in rats is an organ of exchanges between the mother and the fetus. A vitamin D-dependent calcium-binding protein (CaBP) has been recently described in this organ. This led us to investigate the presence of 1,25-dihydroxyvitamin D_3 (1,25-$(OH)_2D_3$) receptor-like proteins in the yok sac cytosol. For this purpose we have utilized sucrose gradient centrifugation, Scatchard analysis, and DNA-cellulose chromatography. Our results show that cycosol prepared from rat yolk sacs contains a 3.3 S binding protein for 1,25-$(OH)_2D_3$. The binding is a highly specific, saturable process with high affinity (2×10^{-10} M at 25 C). The sterol-protein complex binds to DNA-cellulose. The 1,25-$(OH)_2D_3$ binding protein is present in the yolk sac from at least the 15th day until the 21st day of gestation. In contrast, such a binding protein is not found in the amnion, the other component of fetal membranes. The biochemical parameters of the 1,25-$(OH)_2D_3$ binding protein in the yolk sac are similar to those of 1,25-$(OH)_2D_3$ cytosolic receptors in vitamin D target organs. This strongly suggests that the 3.3 S protein in the yolk sac may function as a specific receptor, indicating that this organ may be a new target organ for vitamin D.

STUDIES OF THE RATE OF EFFLUX OF CHOLESTEROL FROM CULTURED HUMAN SKIN FIBROBLASTS. R.J. Daniels, L.S. Guertler, T.S. Parker and D. Steinberg (Div. of Metabolic Disease, Dept. of Medicine, Univ. of California, San Diego, School of Medicine, La Jolla, CA 92093) *J. Biol. Chem. 256*(10):4978-4983 (1981). The cholesterol content of normal human skin fibroblasts was increased (approximately doubled) by incubating cells in the presence of a high concentration of low density lipoprotein. Cholesterol efflux from these cells was then studied as a function of time and as a function of acceptor concentration. High density lipoprotein from which essentially all of the cholesterol had been removed by heptane extraction was used as a model receptor (chole - sterol-depleted high density lipoprotein). Using a sensitive enzymatic assay, it was possible to measure the increase in medium cholesterol and the decrease in cell cholesterol content simultaneously. Release was approximately a linear function of time for at least 6-12 h. A maximal rate of release was obtained at 20 μg of protein/ml (50% of excess stored sterol released in about 12 h); increasing the acceptor concentration 10-fold (to 200 μg/ml) failed to increase efflux rate. Comparison of the rates of fall of free and ester cholesterol levels suggested that hydrolysis of the ester may be rate-limiting when cholesterol-depleted high density lipoprotein is used as the acceptor. The results imply that above saturating concentrations of acceptor, acceptor-cell interaction is no longer limiting and that the rate of efflux of cholesterol under such conditions depends on intracellular processes necessary to make cholesterol available to the acceptor. Whether or not the concentrations of acceptor bathing cells *in vivo* is normally rate limiting remains to be determined.

ABDOMINAL FAT OF BROILERS AS INFLUENCED BY DIETARY LEVEL OF ANIMAL FAT. J.W. Deaton, J.L. McNaughton, F.N. Reece and B.D. Lott (U.S. Dept. of Ag., Sci., and Ed. Admin., Ag. Res., South Central Poultry Res. Lab., Mississippi

State, MS) *Poultry Sci. 60*:1250-1253 (1981). In an attempt to determine the effect of dietary energy source on the quantity of abdominal fat in broilers, three levels of dietary animal fat (4, 7 and 10%) in isocaloric and isonitrogenous diets were fed in place of a carbohydrate energy source. Three trials were conducted. As dietary animal fat increased, the amount of abdominal fat in broilers increased under both a moderate- and a high-temperature rearing regimen. However, as dietary animal fat increased, the body weight gain in broilers tended to increase. It is possible that the benefits of increased growth outweigh the disadvantages of increased abdominal fat when dietary fat is added. Data are presented to provide the industry with figures to help in this decision.

ACETOACETYL-CoA REDUCTASE ACTIVITY OF LACTATING BOVINE MAMMARY FATTY ACID SYNTHASE. P.F. Dodds, M.G.F. Guzman, S.C. Chalberg, G.J. Anderson and S. Kumar (Dept. of Chem., Georgetown Univ., Washington, DC) *J. Biol. Chem. 256*(12):6282-6290 (1981). Fatty acid synthase, purified from lactating bovine mammary gland, utilizes coenzyme A esters of acetoacetic, 3-hydroxybutyric, and crotonic acids as substrates for its partial reactions at micromolar concentrations. The NADPH: acetoacetyl-CoA reductase had a K_m of 5 μM acetoaceyl-CoA and a V_{max} of about 4 μmol of NADPH oxidized min^{-1} mg^{-1}. In contrast, the K_m for the model compound, acetoacetyl pantetheine was 820 μM and that of S-acetoacetyl-*N*-acetylcysteamine was over 40 mM. The reduction of acetoacetyl-CoA was observed with the enzyme from rat tissue also but not with those from avian tissues or yeast. With the bovine mammary enzyme, the reaction was found to oxidize 2 mol of NADPH for every mol of acetoacetyl-CoA consumed.

INCORPORATION OF (1-^{14}C) ACETATE INTO FATTY ACIDS OF THE CRUSTACEANS *Daphnia magna* AND *Cyclops strenus* IN RELATION TO TEMPERATURE. T. Farkas, K. Kariko and I. Csengeri (Inst. of Biochem., Bio. Res. Center, Hungarian Academy of Sci., Szeged, Hungary) *Lipids 16*(6):418-422 (1981). *Daphnia magna* and *Cyclops strenus* were maintained in aquaria containing sodium (1-^{14}C)acetate and the effect of temperature on labeling of their lipids was investigated. Incorporation of radioactivity in total lipids was slowed by a factor of 4 in cold-exposed (5 C) specimens compared to those incubated at 25 C. There was no significant difference in the distribution of label in the lipid classes of animals incubated at the two exteme temperatures. Decrease of the temperature from 25 to 5 C brought about a considerable reduction in the formation of palmitic and stearic acids and an increase in labeling of monounsaturated (18:1) fatty acids in *D. magna*. Docosapolyenoic acids were absent from lipids of this crustacean. *C. strenus* directed a higher proportion of radioactivity in both oleic and docosahexanoic acids upon cold exposure. In response to decrease of the temperature, *D. magna* formed a less unsaturated fatty acid population, as judged from dpm ratios of total saturated to total unsaturated fatty acids, than *C. strenus*. Inability to form and accumulate docosapolyenoic fatty acids by *D. magna* might be related to their poor survival at reduced temperatures.

RELATIONSHIP BETWEEN STRUCTURE AND FATTY ACID METABOLISM IN MITOCHONDRIA ISOLATED FROM ISCHEMIC RAT HEARTS. D. Feuvray and J. Plouët (Lab. de Physio. Comparee, Univ. Paris-Sud, Orsay, France) *Circ. Res. 48*:740-747 (1981). We studied mitochondrial structure and intermediates of fatty acid metabolism in mitochondria isolated from ischemic hearts. By electron microscopy, no structural difference was detected between mitochondria isolated from control hearts and from ischemic hearts receiving glucose as the only substrate. However, major differences were observed in mitochondria obtained from control and ischemic hearts receiving both glucose and palmitate. These hearts contained a higher portion of damaged mitochondria. Levels of long-chain acyl-CoA in mitochondria isolated from hearts receiving glucose alone were practically the same for control and ischemic hearts and were only slightly increased in mitochondria of ischemic hearts receiving both glucose and palmitate. On the other hand, levels of long-chain acyl carnitine in mitochondria of ischemic hearts were twice those found in control hearts. This rise in long-chain acyl carnitine levels in mitochondria isolated from ischemic hearts receiving palmitate may be related to modifications of the mitochondrial structure and to the appearance of amorphous densities.

RAT PLATELET PROSTAGLANDIN, CYCLIC AMP AND LIPID RESPONSE TO VARIATIONS IN DIETARY FAT. K.M. Fine, J. Dupont and M.M. Mathias (Dept. of Food Sci. and Nutr., Colorado State Univ., Fort Collins, CO) *J. Nutr. 111*(4):699-707 (1981). Diets supplying 20 or 40% of the calories as fat with linoleate to saturated fatty acid ratios of 0.4, 0.8 and 5.5 were fed to male weanling rats for 8 and 11 weeks. Recalcification clotting time was not affected by dietary treatment. Concentration of cAMP in the platelets was significantly elevated at the highest level of dietary linoleate. Cyclic AMP was negatively correlated with concentration of dihomo-γ-linolenic acid in platelet phospholipids and positively correlated with prostaglandin E_1, suggesting that dihomo-γ-linolenic acid is rapidly converted to prostaglandin E_1, causing a rise of cAMP due to stimulation of adenylate cyclase. Serum concentrations of cholesterol were significantly higher in rats fed the 20% fat diet than in rats fed the 40% fat diet. Platelet cholesterol content of platelets appears to reflect the lipid environment of the plasma.

PLASMA LIPID AND LIPOPROTEIN CHOLESTEROL CONCENTRATION IN ADULT MALES CONSUMING NORMAL AND HIGH CHOLESTEROL DIETS UNDER CONTROLLED CONDITIONS. E. Flaim, L.F. Ferreri, F.W. Thye, J.E. Hill and S.J. Ritchey (Dept. of Human Nutr. and Foods and Dairy Sci., VA Polytechnic Inst. and State Univ., Blackburg, VA) *Am. J. Clin. Nutr. 34*:1103-1108 (1981). Twenty-three young adult males were fed diets containing either 400 or 1400 mg of cholesterol per day under controlled conditions for 4 wk. There were minimal differences between the two diets in total protein, carbohydrate, fat and the P/S fatty acid ratio. In both diets 400 mg of cholesterol was aupplied from nonegg food sources; the additional 1000 mg of cholesterol was from four whole eggs. Blood samples were collected after a 12- to 14-h fast at the beginning of the study, weekly throughout the experimental period, and 1 wk after completion of the study. Plasma total cholesterol and triglycerides and high-density, low-density, and very low-density lipoprotein cholesterol levels were measured. The importance of changes in the properties and metabolic activity of individual lipoproteins induced by dietary cholesterol with or without gross changes in the cholesterol levels remains to be determined.

PURIFICATION AND CHARACTERIZATION OF A PHOSPHOLIPID-DEPENDENT α-MANNOSIDASE FROM RABBIT LIVER. W.T. Forsee and J.S. Schutzbach (Dept. of Micobiol., The Diabetes Res. and Training Center, and Dept. of Biochem., Univ. of Alabama in Birmingham, Birmingham, AL) *J. Biol. Chem. 256*(13):6577-6582 (1981). An α-mannosidase specific for the hydrolysis of α-1,2-mannosyl-mannose linkages has been solubilized and partially purified from rabbit liver microsomes. The enzyme is inhibited by EDTA and has optimal activity in the presence of calcium ions. The purified enzyme has a requirement for nonionic detergents or for specific phospholipids. At detergent concentrations appreciably below the critical micelle concentration, the enzyme is active in the presence of phosphatidylcholine or phosphatidylethanolamine but not with phosphatidylinositol, phosphatidylglycerol, or phosphatidic acid. At concentrations of phosphatidylcholine which provide optimal activity, the enzyme is strongly inhibited by phosphatidylinositol or phosphatidylglycerol. The substrate specificity of the α-mannosidase toward oligosaccharide substrates suggests that the enzyme may be involved in the processing of the oligosaccharide chains of mammalian glycoproteins.

CHARACTERIZATION OF 1,25-DIHYDROXYVITAMIN D_3-DEPENDENT CALCIUM UPTAKE IN CULTURED EMBRYONIC CHICK DUODENUM. R.T. Franceschi and H.F. DeLuca (Dept. of Biochem., College of Ag. and Life Sci., Univ. of Wisc.-Madison, Madison, WI) *J. Biol. Chem. 256*(8):3840-3847 (1981). To confirm that embryonic chick duodenum in organ culture is a suitable model for studying the mechanism by which 1α,25-dihydroxyvitamin D_3 stimulates intestinal calcium transport, we have further characterized this system using the following criteria: calcium uptake properties, responsiveness to vitamin D_3 analogs, and properties of a 1α,25-dihydroxyvitamin D_3 receptor-like binding protein. Our studies suggest that this *in vitro* system is quite similar to the rachitic chick system by all criteria examined. These studies provide further support for the use of embryonic chick duodenum as a model for studies on the mechanism of action of 1α,25-dihydroxyvitamin D_3 in intestine.

THE EFFECT OF INHIBITORS OF PROTEIN AND RNA SYNTHESIS ON A 1α,25-DIHYDROXYVITAMIN D_3-DEPENDENT CALCIUM UPTAKE IN CULTURED EMBRYONIC CHICK DUODENUM. R.T. Franceschi and H.F. DeLuca (Dept. of Biochem., Col. of Ag. and Life Sci., Univ. of Wisconsin-Madison, Madison, Wisconsin) *J. Bio. Chem. 256*(8):3848-3852 (1981). To determine whether 1α,25-dihydroxyvitamin D_3-dependent increases in intestinal calcium uptake require *de novo* protein and RNA synthesis, the effects of several inhibitors of these processes have been reexamined *in vitro* using cultured embryonic chick duodenum. To minimize the contributions of antibiotic toxicity to the interpretation of results, care was taken to examine inhibitor effects at early times after the onset of the 1α,25-dihydroxyvitamin D_3 response. These results further strengthen the hypothesis that 1α,25-dihydroxyvitamin D_3 stimulates intestinal calcium transport via a nuclear mechanism involving new gene expression.

TOTAL ENZYMIC SYNTHESIS OF CHOLESTEROL FROM

LANOSTEROL. CYTOCHROME b_5-DEPENDENCE OF 4-METHYL STEROL OXIDASE. H. Fukushima, G.F. Grinstead and J.L. Gaylor (Dept. of Biochemistry, University of Missouri, Columbia, MO 65212) *J. Biol. Chem. 256*(10):4822-4826 (1981). Methyl sterol oxidase of microsomal synthesis of cholesterol from lanosterol is a mixed-function oxidase that is dependent upon reduced pyridine nucleotide. The methyl sterol oxidase, as well as NADH-cytochrome c reductase, in intact rat liver microsomes are inhibited by anti-cytochrome b_5 immunoglobulin, but NADPH-cytochrome c reductase is not affected. There is a decreased time lag prior to onset of reoxidation of steady state levels of reduced cytochrome b_5 when 4-methyl sterol oxidase substrates are present. Trypsin treatment of microsomes destroys cytochrome b_5 with loss of methyl sterol oxidase activity. Activity is restored by addition of purified cyctochrome b_5 to trypsin-treated microsomes. Initial attempts to solubilize and purify 4-methyl sterol oxidase have been only partially successful due to the extreme lability of the oxidase. However, DEAE-cellulose column chromatography of a detergent extract of microsomes yields a fraction that contains the oxidase, lipids, and NADH-cytochrome b_5 reductase but is free of cytochrome b_5. Oxidation of 4α[30-^{3}H] methyl-5α-cholest-7-en-3β-ol by methyl sterol oxidase in this isolated fraction can be fully restored by the addition of purified liver microsomal cytochrome b_5. These results strongly support the suggestion that membrane-bound cytochrome b_5 of rat liver microsomes is an obligatory electron carrier from NADH to 4-methyl sterol oxidase.

EFFECT OF POLYESTRADIOL ON LECITHIN:CHOLESTEROL ACYLTRANSFERASE IN MALE AND FEMALE RATS. J.M. Gandarias, M. Lacort, B. Ochoa and M. Quiroga (Dept. of Physio. and Biochem., Faculty of Med., Univ. of Pais Vasco, Bilbao, Spain) *Lipids 16*(6):449-453 (1981). The effects of two doses of polyestradiol phosphate on lecithin:cholesterol acylttransferase activity and on liver and plasma cholesterol levels have been studied on female and male rats. Both treatments increased the hepatic content of esterified cholesterol, but the LCAT activity expressed as a percentage of cholesterol esterifications was unaltered. The progress of esterification was not affected by the administration of the hormone. The LCAT activity in terms of the initial rate of esterification was decreased by the high dose of estradiol. This decrease was associated with a reduction of free plasma cholesterol level, as there is a significant positive correlation between these two parameters. The findings suggest that the increased esterified cholesterol in liver of estradiol-treated rats is not mediated by an alteration in the LCAT activity.

THE EFFECTS OF VITAMIN A-DEFICIENT DIETS CONTAINING LACTOSE IN PRODUCING BLADDER CALCULI AND TUMORS IN RATS. S.N. Gershoff, R.B. McGandy (Tufts University Nutrition Institute, Medford, MA 02155) *Amer. J. Clin. Nutr. 34*(4):483-489 (1981). This report describes studies in which diets have been developed which when fed to rats commonly result in the formation of primary urinary bladder calculi. The bladder walls of most of these rats but not those with urinary stones were grossly hypertrophied. Microscopic examinations in one of the studies indicated that about a quarter of the bladders containing stones showed histological changes consistent with those characterized as grade I to II transitional cell carcinomas. In all cases animals showing these histological abnormalities consumed vitamin A-deficient diets in which the carbohydrate was supplied mostly by lactose.

EFFECTS OF OCTANOIC ACIDEMIA ON PALMITIC ACID METABOLISM IN RAT LIVER. J.L. Glenn and W.B. Hinshaw (Dept. of Biochem., Albany Med. Center, Albany, NY) *Pro. Soc. Exp. Bio. Med. 167*:36-39 (1981). This study examined the effects of octanoic acidemia on the metabolism of radioactive palmitic acid in rat liver. Octanoic acidemia, produced either by an intraperitoneal bolus injection or sustained intravenous infusion of sodium octanoate, markedly influenced biochemical pathways which are concerned by the hepatic disposition of palmitic acid into the different classes of rat liver lipids. During acute octanoic acidemia, the incorporation of (16-^{14}C)palmitic acid into hepatic triacylglycerols was greatly increased, while uptake of this fatty acid into phospholipids, especially phosphatidylcholine, was sharply reduced. When the octanoic acidemia resulted from intravenous infusion of sodium octanoate for 4 hr prior to the injection of (15-^{14}C)palmitic acid, the pattern of isotope incorporation was one which would support hepatic steatosis.

VITAMIN D METABOLITES IN PLASMA OF COWS FED A PREPARTUM LOW-CALCIUM DIET FOR PREVENTION OF PARTURIENT HYPOCALCEMIA. H.B. Green, R.L. Horst, D.C. Beitz, and E.T. Littledike (Dept. of Animal Science, Iowa State University and National Animal Disease Center, Ames, IA 50011) *J. Dairy Sci. 64*(2):217-226 (1981). Our objective was to characterize changes in vitamin D metabolites of plasma in Jersey cows fed a prepartum low-calcium diet. Eight cows were fed a high-calcium diet (80 g/day) and eight were fed a low-calcium diet (8 g/day) at least 14 days before parturition. Calcium concentrations in plasma decreased after initiation of feeding either diet, but cows fed low-calcium diet tended to have lower prepartum calcium and phosphorus and greater peripartal calcium in plasma. Hydroxyproline in plasma was greater during peripartal period in cows fed low-calcium diet. Prepartum 1,25-dihydroxyvitamin D in plasma tended to be greater in cows fed low calcium. Increases in 1,25-dihydroxyvitamin D were only 2 and 3 days after initiation of the low-calcium diet; during the first 2 days after parturition, however, 1,25-dihydroxyvitamin D tended to be lower in those cows fed low calcium. As parturition neared, 24,25-dihydroxyvitamin D tended to be lower in cows fed the low-calcium diet. Usual early postpartum changes in calcium, phosphorus, magnesium, 1,25-dihydroxyvitamin D, and hydroxyproline were seen during first few days after initiation of feeding low calcium. Thus, we propose that the preventative action of the low-calcium diet is associated with preparation of the calcium homeostatic mechanism several days before the calcium demand of initiation of lactation.

LONG CHAIN POLYENOIC ACID LEVELS IN VIABLY SORTED, HIGHLY ENRICHED MOUSE TESTIS CELLS. W.M. Grogan, W.F. Farnham and B.A. Szopiak (Dept. of Biochem., Med. Col. of Virginia Campus, Virginia Commonwealth Univ., Richmond, VA) *Lipids 16*(6):401-410 (1981). Twenty- and 22-carbon fatty acids of the linoleic (n-6) and linolenic (n-3) acid families were measured in murine spermatogonia and preleptotene spermatocytes (early), pachytene primary spermatocytes (1°), round spermatids (RS), condensing spermatids (CS) and Leydig cells enriched by staput velocity sedimentation at 1 G, followed by viable microflow sorting on the basis of ligth scatter and DNA content. 22:5(n-6) increased progressively from 2 to 20% of total fatty acid in the progression of germinal cell differentiation, early → 1° → RS → CS, but decreased in mature sperm. The precursor 20:4(n-6) showed a roughly reciprocal relationship. 22:6(n-3) showed no significant correlation with cell type. 22:5(n-6) was found highest in triglycerides of later differentiation stages whereas 20:4(n-6) and 22:6(n-3) were found primarily in phospholipid in all cell fractions.

FUNCTION OF HEPATIC TRIGLYCERIDE LIPASE IN LIPOPROTEIN METABOLISM. J. Grosser, O. Schrecker and H. Greten (Klinisches Institut für Herzinfarktforschung an der Medizinischen Universitätsclinik Heidelberg, Bergheimer Strasse 58, D-6900 Heidelberg, Federal Republic of Germany) *J. Lipid Res. 22*(3):437-442 (1981). Rat hepatic triglyceride lipase (H-TGL) was purified from liver tissue extracts by affinity chromatography on Sepharose 4B with covalently linked heparin. The purified rat H-TGL exhibited the properties previously described for this enzyme. Enzyme protein was injected into rabbits for anti-H-TGL antibody production. Antirat-H-TGL did not react against rat lipoprotein lipase (LPL) but inhibited H-TGL-activity both in vitro and in vivo >90%. These antibodies were injected into rats and lipoprotein analyses were performed over a 36-hr period. It could be shown that inactivation of H-TGL by anti-H-TGL γ globulins in vivo led to an increase in total triglyceride concentration from 70 mg/dl to 230 mg/dl due to an increase in very low density lipoprotein (VLDL) and low density lipoprotein (LDL) triglycerides 4 hr after antibody injection; a marked increase in high density lipoprotein (HDL) phospholipid concentration was observed with almost no change in HDL-cholesterol and HDL-triglycerides. This study documents the ability of antirat-H-TGL γ-globulins to inhibit H-TGL in vitro and in vivo. Furthermore, the inhibition of triglyceride removal in vivo demonstrated that this enzyme together with LPL is responsible for the catabolism of VLDL-triglyceride.

EFFECT OF ALLOXAN DIABETES ON PHOSPHATIDYLCHOLINE BIOSYNTHETIC ENZYMES. D.R. Hoffman, J.A. Haning and W.E. Cornatzer (Guy and Bertha Ireland Res. Lab., Dept. of Biochem., Univ. of North Dakota, Sch. of Med., Grand Forks, ND) *Pro. Soc. Exp. Biol. Med. 167*:143-146 (1981). Phosphatidylethanolamine methyltransferase, and choline phosphotransferase enzymatic activities (nmole PC formed min/mg protein) have been determined in liver microsomes of alloxan diabetic rats. There was a significant reduction in the methylation pathway in the conversion of phosphatidylethanolamine to phosphatidylcholine as demonstrated in the low volume of the phosphatidylethanolamine methyltransferase and phosphatidyldimethylethanolamine methyltransferase in those diabetic rats with blood glucose levels greater than 600 mg%. The reduction was 49 and 48% decrease over controls, respectively. There was a significant increase in the choline phosphotransferase in the diabetic rats. The increase was 92% over controls for the rats with blood glucose of 461 mg% and 55% over controls for the rats with blood glucose 946 mg%.

ALTERATION OF CHOLESTEROL METABOLISM BY 4-0-METHYLASCOCHLORIN IN RATS. T. Hosokawa, M. Sawada, K. Ando and G. Tamura (Dept. of Ag. Chem., Faculty of Ag., Univ.

of Tokyo, Tokyo, Japan) *Lipids 16*(6):433-438 (1981). The effect of 4-O-methylascochlorin (MAC), an experimental hypocholesterolemic agent, on cholesterol metabolism was investigated in rats in two separate experiments. The administration of MAC for 2 and 6 consecutive weeks at daily doses of 100-135 mg/kg resulted in reduction in serum cholesterol levels of 16% after 2 weeks of treatment in the first experiment, and 13% after 6 weeks in the second experiment in comparison to the corresponding controls. It appears that the mechanism of serum cholesterol lowering due to MAC is related to the enhancement of hepatic bile acid synthesis and the increase in biliary and fecal excretion of bile acids.

LIVER BLOOD FLOW AND OXYGEN CONSUMPTION DURING METABOLIC ACIDOSIS AND ALKALOSIS IN THE GREYHOUND. R.L. Hughes, R.T. Mathie, W. Fitch and D. Campbell (Univ. of Glasgow, Dept. of Anaesthesia at the Royal Infirmary, Glasgow, Scotland) *Clin. Sci. 60*:355-361 (1981). Hepatic arterial and portal venous blood flow and hepatic oxygen consumption were measured in two groups of greyhounds anaesthetized with pentobarbitone. Flows were measured with electromagnetic flowmeters. In the first group the effects of metabolic acidosis produced by the infusion of a molar solution of lactic acid were studied. In the second group the effects of metabolic alkalosis produced by the infusion of a molar solution of sodium bicarbonate were studied. It is concluded that metabolic acidosis reduces the supply of oxygen to the liver owing to the reduction in hepatic arterial blood flow and is therefore potentially harmful, whereas metabolic alkalosis probably has no biologically significant effect on liver blood flow.

BINDING OF PLASMA LOW DENSITY LIPOPROTEINS TO ERYTHROCYTES. D.Y. Hui, J.G. Noel and J.A.K. Harmony (Chem. Dept., Indiana Univ., Bloomington, IN) *Biochim. Biophys. Acta 664*:513-526 (1981). Low density lipoproteins (LDL) containing apolipoprotein B bind to intact, freshly isolated erythrocytes. The LDL-erythrocyte interaction is of low affinity, with a K_d of $1.1 \cdot 10^{-6}$ M. Binding is noncooperative. There are about 200 binding sites per cell and, within the limits of experimental uncertainty, these sites comprise a homogenous class. Binding of LDL is a temperature-independent process. The maximum amount of LDL bound increases following proteolytic digestion of the cells with trypsin or chymotrypsin. Chemically modified and native LDL exchange cholesterol with erythrocytes at equal rates and to nearly equal extents. Taken together, the data suggest that the binding sites for LDL on the erythrocyte membrane are distinct from the LDL receptors at the surface of other cells which do not bind HDL and which do not recognize LDL with derivatized arginine or lysine residues. It is proposed that the biological function of the erythrocyte binding sites is to mediate the exchange of cholesterol between the cell membrane and lipoproteins.

EFFECT OF OXIDIZED FISH OIL, DL-α-TOCOPHERYL ACETATE AND ETHOXYQUIN SUPPLEMENTATION ON THE VITAMIN E NUTRITION OF RAINBOW TROUT (*Salmo gairdneri*) FED PRACTICAL DIETS. S.S.O. Hung, C.Y. Cho and S.J. Slinger (Dept. of Nutr., College of Bio. Sci., Univ. of Guelph, Guelph, Ontario) *J. Nutr. 111*(4):648-657 (1981). A factorial experiment was conducted using two degrees of oxidation of the 7.5% supplemental fish oil (peroxide values of 5 and 120 meq/kg oil), two levels of supplemental DL-α-tocopheryl acetate (0 and 33 mg/kg diet) and two levels of ethoxyquin (0 and 125 mg/kg diet) supplementation. Dietary thiobarbituric acid number, weight percentage of polyunsaturated fatty acids and omega-three fatty acids in the total fatty acids were significantly ($P < 0.05$) different between diets with fresh and highly oxidized oil. Dietary RRR-α-tocopheryl was significantly ($P < 0.05$) reduced by the addition of highly oxidized oil after 24 weeks storage of the feed while supplemental DL-α-tocopheryl acetate level was not changed.

BOLL WEEVIL FEEDING DETERRENTS FROM TUNG OIL. M. Jacobson, M.M. Crystal and J.D. Warthen (Bio. Active Natural Products Lab. and Insect Reproduction Lab., Ag. Res., Sci. and Ed. Admin., U.S. Dept. of Ag., Beltsville, MD) *J. Agric. Food Chem. 29*: 591-593 (1981). α-Eleostearic acid and *erythro*-9,10-dihydroxy-1-octadecanol acetate have been identified as the components responsible for the feeding deterrency of tung oil to the adult boll weevil, and methods have been developed for isolating large quantities of the acid from the oil and for synthesizing *erythro*-9,10-dihydroxy-1-octadecanol acetate. Although α-eleostearic acid is too unstable for practical use as a feeding deterrent under field conditions, its methyl ester is much more stable and equally effective as a deterrent.

INTERACTIONS OF DIPALMITOYL- AND DIMYRISTOYLPHOSPHATIDYLCHOLINES AND THEIR MIXTURES WITH APOLIPOPROTEIN A-I. A. Jonas and W.R. Mason (Dept. of Biochemistry and the School of Basic Medical Sciences, Univ. of Illinois, Urbana, IL 61801) *Biochemistry 20*(13):3801-3805 (1981). Human and bovine A-I apolipoproteins were incubated with multibilayer liposomes of dimyristoylphosphatidylcholine (DMPC) and dipalmitoylphosphatidylcholine (DPPC) and several mixtures of these two lipids. The results indicate a decrease in reaction rates with increasing DPPC contents of the mixtures, consistent with the higher stability of DPPC bilayers. The lower lipid/protein ratio of DPPC complexes (100:1 mol/mol) is compensated by the longer acyl chains of this lipid, such that the acyl chain area of both complexes stabilized by apolipoprotein is essentially identical.

STRUCTURE OF TWO SUBFRACTIONS OF NORMAL PORCINE (*Sus domesticus*) SERUM LOW-DENSITY LIPOPROTEINS. X-RAY SMALL-ANGLE SCATTERING STUDIES. G. Jürgens, G.M.J. Knipping, P. Zipper, R. Kayushina, G. Degovics, and P. Laggner (Institut für Röntgenfeinstrukturforschung der Österreichischen Akademie der Wissenschaften und des Forschungszentrums Graz, Steyrergasse 17, A-8010 Graz, Austria) *Biochemistry 20*(11): 3231-3237 (1981). Two subfractions of low-density lipoproteins (LDL) were isolated from normal pig (*Sus domesticus*) serum by a combined method including precipitation, ultracentrifugation, and gel chromatography. The fractions recovered from the buoyant density ranges 1.020-1.050 and 1.050-1.090 g/mL, denoted as LDL_1 and LDL_2, respectively, were studied with regard to structure and thermotropic behavior by X-ray small-angle scattering and were compared to human serum low-density lipoprotein of density 1.020-1.063 g/mL. The average molecular weights determined from the scattering intensities on an absolute scale were 2.6×10^6 and 2.0×10^6 for LDL_1 and LDL_2, respectively. The maximum particle diameters were found to be 24 and 21 nm, respectively. Both species were found to have quasi-spherical symmetry and to display the thermotropic transition of the apolar lipids within the particle core similar to human LDL. The width of the transition was approximately 9 °C in both cases, but the midpoint transition temperature was higher by 8 C for LDL_1 (33 C) than for LDL_2 (25 C). Despite their different sizes and thermotropic behavior, the two porcine LDL subfractions appear to be built according to the same structural principle as human LDL in the molecular organization of the apolar lipids within the particle core.

DETERMINATION OF PHOSPHOLIPIDS BY A COMBINED LIQUID CHROMATOGRAPH-AUTOMATED PHOSPHORUS ANALYZER. J.K. Kaitaranta and S.P. Bessman (Tech. Res. Centre of Finland, Food Res. Lab., 02150 Espoo 15, Finland) *Anal. Chem. 53*:1232-1235 (1981). An automatic phosphorus analyzer was evaluated for phospholipid quantitations and as a detector in the HPCL analysis of phospholipids. The relative standard deviation of the analyses with 10 nmol loads of individual phospholipids averaged 3.2% (range 1.1-4.7%). Considering the reproducibility and the linearity of the method, the range from 1 to 100 nmol is suggested as a practical working range. A two-step elution system was developed to separate individual phospholipids from natural samples on a silica column. Neutral lipids present in the total lipid extracts do not interfere with the phospholipid determination if the phosphorus analyzer is used for detection.

SERUM LIPOPROTEINS IN PATIENTS WITH CLINICAL SIGNS OF ATHEROSCLEROSIS: PERIPHERAL VASCULAR AND CORONARY HEART DISEASE. J. Kaliman, K. Widhalm and W. Strobl (Dept. of Cardio. and Ped., Med. Sch. Univ. Vienna, Austria) *Artery 8*(6):547-552 (1980). Lipids and lipoproteins were examined in 7 males patients with coronary heart disease, 10 males with coronary heart and peripheral vascular disease, 25 males with peripheral vascular disease and 10 females with peripheral vascular disease aged between 50-60 years. In general our results demonstrate that in all patients with different locations of atherosclerosis elevated concentrations of total cholesterol, LDL-cholesterol and triglycerides can be found. However, low HDL-cholesterol levels measured in all groups should be pointed out. From our data it is concluded that there does not exist a typical lipid or lipoprotein pattern in patients with different locations of atherosclerosis. Only minor differences exist concerning the concentrations of the major lipids and lipoproteins.

ANTIBODY AGAINST LOW DENSITY LIPOPROTEIN RECEPTOR BLOCKS UPTAKE OF LOW DENSITY LIPOPROTEIN (BUT NOT HIGH DENSITY LIPOPROTEIN) BY THE ADRENAL GLAND OF THE MOUSE IN VIVO. T. Kita, U. Beisiegel, J.L. Goldstein, W.J. Schneider, and M.S. Brown (Depts. of Molecular Genetics and Internal Medicine, University of Texas Health Science Center at Dallas, Dallas, TX 75235) *J. Biol. Chem. 256*(10):4701-4703 (1981). The adrenal gland of the mouse takes up intravenously administered ^{125}I-labeled human low density lipoprotein (LDL) by a high affinity, receptor-mediated mechanism. Uptake is enhanced by treatment of mice with a combination of 4-aminopyrazolopyrimidine, which eliminates endogenous mouse lipoproteins from the plasma, and adrenocorticotropin, which increases the number of adrenal LDL receptors. In the current studies, we show

that adrenal uptake of ^{125}I-LDL is blocked when the mice have received a prior intravenous injection of a rabbit antibody directed against the LDL receptor purified from bovine adrenal cortex. The antibody-mediated inhibition of ^{125}I-LDL uptake persisted for 6 h and was reversed by 19 h. Adrenal uptake of ^{125}I-labeled high density lipoprotein uptake by the adrenal gland is mediated by a receptor that differs from the LDL receptor. The current studies illustrate the usefulness of antibodies in probing the process of receptor-mediated endocytosis in intact animals.

ADIPOCYTE CHOLESTEROL STORAGE: EFFECT OF STARVATION. B.R. Krause, M. Balzer, and A.D. Hartman (Dept. of Physio., Louisiana State Univ. Med. Center, New Orleans, LA) *Pro. Soc. Exp. Bio. Med. 167*:407-411 (1981). The content of free and esterified cholesterol in epididymal and subcutaneous adipocytes was measured during starvation in adult male rats in order to test the hypothesis that adipose tissue cholesterol esters are mobilized concomitantly with free cholesterol and triglyceride during starvation. Plasma and liver lipid concentrations were also determined as a function of time during food deprivation. Although neither cholesterol ester turnover nor hydrolase activity was directly measured, the results appear to be inconsistent with the hypothesis that an adipose tissue cholesterol ester hydrolase plays a role in the hydrolysis of stored esters during acute starvation, and further suggest that the turnover of cholesterol and cholesterol esters is probably different in adipose tissue.

CONTROL OF ARACHIDONIC ACID ACCUMULATION IN BONE MARROW-DERIVED MACROPHAGES BY ACYLTRANSFERASES. E.E. Kroner, B.A. Peskar, H. Fischer, and E. Ferber (Max-Planck-Institut fur Immunbiologie and Pharm. Inst. der Univ. Freiburg, Freiburg, West Germany) *J. Bio. Chem. 256*(8):3690-3697 (1981). The turnover of phospholipid fatty acid moieties of bone marrow-derived macrophates was analyzed by separate determination of degrading and acylating activities. Acylating activities were followed intact cells by incubation with excess arachidonic acid and degradation of phospholipids was followed in cells prelabeled with fatty acids. Significant phospholipase A_2 activity was detectable only if the reutilization of liberated fatty acid was inhibited, e.g. by *p*-chloromercuribenzoate. These results suggest that in the cells studied, the level of free arachidonic acid is mainly controlled by the activity of the lysophosphatide acyltransferase.

INACTIVATION OF CHICKEN LIVER FATTY ACID SYNTHETASE BY MALONYL COENZYME A. EFFECTS OF ACETYL COENZYME A AND NICOTINAMIDE ADENINE DINUCLEOTIDE PHOSPHATE. S. Kumar and K.R. Srinivasan (Dept. of Biochem., College of Med. andDentistry of NJ, NJ Med. Sch., Newark, NJ) *Biochem. 20*:3393-3400 (1981). Chicken liver fatty acid synthetase complex is irreversibly inactivated by one of the substrates, malonyl-CoA. Acetyl-CoA has a dual role. At concentrations less than or comparable to those of malonyl-CoA, the rate of inactivations is enhanced, whereas at acetyl-CoA/malonyl-CoA ratios greater than 2, the rate of inactivations is slowed down. $NADP^+$ at low concentrations (25 μM) affords considerable protection against malonyl-CoA mediated inactivation whereas NAD^+ even at 1.0 mM concentration has no effect. The process of inactivation is accompanied by enhanced covalent binding of malonyl groups such that approximately 6 mol of the acyl group is bound per mol of the enzyme at complete inactivation. The available evidence suggests that the inactivation of the enzyme results from the binding of malonyl group(s) at or near the condensing site of the enzyme.

SEX DIFFERENCES IN HEPATIC UPTAKE OF LONG CHAIN FATTY ACIDS IN SINGLE-PASS PERFUSED RAT LIVER. M.C. Kushlan, J.L. Gollan, W.L. Ma, and R.K. Ockner (Dept. of Medicine and Liver Center, University of California School of Medicine, San Francisco, CA 94143) *J. Lipid Res. 22*(3):431-436 (1981). Recent studies of liver cell suspensions have shown that in immature, adult, castrated, and hormone-treated rats, sex steroids exert striking effects on [^{14}C] oleate uptake and utilization (which were significantly increased by estradiol and diminished by testosterone). To determine whether these observed sex differences in fatty acid uptake also were present in the inact liver, single-pass [^{14}C] oleate uptake was measured in isolated perfused livers. Livers from sexually mature female and male rats were perfused single-pass with albumin-bound [^{14}C] oleate in Krebs-Ringer bicarbonate buffer. Under all conditions, oxidation of [^{14}C] oleate in female liver equaled or exceeded that in male liver, indicating that the increased incorporation into triglycerides and other glycolipids was not simply the result of differences in the distribution of [^{14}C] oleate among cellular metabolic pathways. These studies demonstrate that in the intact liver, as in isolated hepatocytes, there are profound sex differences in the uptake of long chain fatty acids. This difference may account in part for the observed sex steroid effects on hepatic triglyceride biosynthesis and VLDL production. The mechanism of these uptake differences remains to be determined.

NOVEL POLAR LIPIDS FROM METHANOGEN *METHANOSPIRILLUM HUMGATEI* GP1. S.C. Kushwaha, M. Kates, G.D. Sprott, and I.C.P. Smith (Department of Biochemistry, University of Ottawa, Ottawa, Ontario, K1N 9B4, Canada) *Biochim. Biophys. Acta 664*(1):156-173 (1981). The methanogenic bacterium *Methanospirillum hungatei* GP1 has been shown to contain two unusual phosphoglycolipids (phosphoglycolipid I and phosphoglycolipid II) that account for 64% (by wt.) of the total cellular lipids. These lipids are derivatives of the dibiphytanyldiglycerol tetraether. One of the free hydroxyls of this tetraether is esterified with glycerophosphoric acid and the other is linked glycosidically to a disaccharide with structure α-Glop-(1→2)-βGal*f* in phosphoglycolipid I and β-Gal*f*-(1→6)-βGal*f* in phosphoglycolipid II. Smaller amounts of the *sn*-2,3-diphytanylglycerol analog of phosphatidylglycerol and diglycosyldiphytanylglycerol ethers (DGD-I and DGD-II) containing the same disaccharide residues as in phosphoglycolipid I and phosphoglycolipid II, respectively, were identified, together with very small amounts of diglycosyldibiphytanyldiglycerol tetraethers (DGT-I and DGT-II) containing the same disaccharide residues as in phosphoglycolipid I and phosphoglycolipid II, respectively. A biosynthetic pathway involving head-to-head condensation of phosphatidylglycerol with DGD-I or DGD-II to form phosphoglycolipid I or phosphoglycolipid II, respectively, is proposed.

THE INITIAL ACTION OF THROMBIN ON PLATELETS. CONVERSION OF PHOSPHATIDYLINOSITOL TO PHOSPHATIDIC ACID PRECEDING THE PRODUCTION OF ARACHIDONIC ACID. E.G. Lapetina, M.M. Billah, and P. Cuatrecasas (Dept. of Mo. Bio., Wellcome Res. Lab., Res. Triangle Park, NC) *J. Biol. Chem. 256*(10):5037-5040 (1981). Measurements of phosphatidylinositol (PI) and phosphatidic acid (PA) by phosphorus assays and by radioactivity ([^{14}C] arachidonate) indicate that thrombin induces the degradation of a given fraction of the total PI to PA. The maximal conversion of PI to PA represents approximately one-third of the total PI which can be degraded by thrombin. This same amount of PI is converted to PA even in the presence of 1 mM quinacrine, which completely inhibits the release of arachidonic acid from phospholipids and which reduces by two-thirds the loss of labeled PI. In this case the fall in PI is equal to the amount of PA formed. If thrombin is added to platelets previously maximally stimulated by ionophore A23187, PA is produced from PI in amounts equal to those produced by thrombin in the absence of other stimuli. The simplest scheme is one in which thrombin specifically produces an active fraction of PA which in some way results in the subsequent production of arachidonic acid from various phospholipids (including PI), perhaps by activation of quinacrine-sensitive phospholipase A_2.

THE IDENTITY AND PROPERTIES OF TWO FORMS OF ACTIVATED COLIPASE FROM PORCINE PANCREAS. A. Larsson and C. Erlanson-Albertsson (Department of Physiological Chemistry, University of Lund, P.O. Box 750, S-220 07 Lund 7, Sweden) *Biochim. Biophys. Acta 664*(3):538-548 (1981). Colipase is excreted as a procolipase, colipase$_{101}$. It is activated by low concentrations of trypsin, hydrolyzing the N-terminal pentapeptide. With higher concentrations of trypsin or in the presence of Ca^{2+} a smaller form of colipase, containing 85 amino acids, appears. It has glycine as the N-terminal and arginine as the C-terminal amino acid residue and has lost 11 amino acids in the C-terminal chain. The ability of colipase$_{85}$ to activate lipase with tributyrin as substrate is about the same as for colipase$_{96}$ and colipase$_{85}$ are similar and about five times higher than that of colipase$_{101}$. With intralipid as substrate colipase$_{85}$ enables lipase to reach the triacylglycerol substrate more rapidly than colipase$_{96}$, having about six times shorter lag-time for a given concentration. Colipase$_{84}$, obtained by splitting off the C-terminal arginine from colipase$_{85}$, has a lagtime somewhere between colipase$_{85}$ and colipase$_{96}$, pointing to the importance of arginine$_{85}$ for Intralipid activity. The binding between lipase and colipase has about the same strength for procolipase, colipase$_{96}$, and for colipase$_{85}$, K_d being about 10^{-6} M either in buffer or in the presence of 2 mM taurodeoxycholate at pH 7. Addition of long chain fatty acids in the presence of bile salts increases the binding strength between colipase and lipase 100-fold, both for colipase$_{96}$ and colipase$_{85}$.

EFFECTS OF CARNITINE ISOMERS ON FATTY ACID METABOLISM IN ISCHEMIC SWINE HEARTS. A.J. Liedtke, S.H. Hellis, and L.F. Witesell (Cardio. Div., Penn. State Univ., Hershey Med. Center, Hershey, PA) *Cir. Res. 48*(6):859-866 (1981). We studied the hemodynamic and metabolic effects of treatments with the L- and DL-isomers of caritine in four groups (n=42) of intact working swine hearts rendered mildly ischemic (-46% reduction in global perfusion). In three groups (n=34), free fatty acids (FFA) in the coronary perfusate were augmented with labeled palmitate (0.72 μmol/ml). These data suggest that carnitine reduces availability or incorporation of FFA intracellularly, and this benefits the heart

mechanically during ischemic restrictions in coronary flow. The L-isomer appears to be the more biologically active.

FORMATION OF URSO- AND URSODEOXY-CHOLIC ACIDS FROM PRIMARY BILE ACIDS BY *CLOSTRIDIUM ABSONUM*. I.A. Macdonald, D.M. Hutchison and T.P. Forrest (Depts. of Med., Biochemistry, and Chem., Dalhousie Univ., Halifax, Nova Scotia, Canada, B3H 4H7) *J. Lipid Res. 22*(3):458-466 (1981). Eight strains of *Clostridium absonum* were shown to form ursocholic acid (UC) from cholic acid (C) and ursodeoxycholic acid (UDC) from chenodeoxycholic acid (CDC) but did not transform deoxycholic acid (DC) in whole cell cultures. The structures of UC and UDC were verified by mass spectroscopy, and by thin-layer chromatography using Komarowsky's spray reagent. The organism transformed C and CDC at concentrations below 1.5×10^{-3} M and 5.0×10^{-4} M, respectively. Optimal yields of the final products were realized at about 15-22 hr and 9-15 hr of incubation, respectively. With longer periods of incubation, increasing yields of 7K-DC and 7K-LC and decreasing yields of UC and UDC were observed. These time course studies suggest that 7K-DC and 7K-LC are intermediates in the formation of UC and UDC from the primary bile acids. We propose the occurrence of C$\rightarrow$7K-DC$\rightleftarrows$ UC and CDC$\rightleftarrows$7K-LC$\rightleftarrows$ UDC with increasing dominance of back reaction of the second step on aging of the culture. Formation of UC from C was much slower than that of UDC from CDC. In contrast, *C. paraperfringens* transformed none of the above bile acids. We propose the *C. absonum*, or a biochemically similar species, may be present in the human gut and give rise to UDC (and UC) in vivo.

DECREASE IN SERUM LEVELS OF 1,25-DIHYDROXYCHOLECALCIFEROL IN RATS AND CHICKENS FED A VITAMIN D-DEFICIENT DIET. J.P. Mallon, A. Boris and G.F. Bryce (Dept. of Cell Biol., Roche Res. Center, Hoffmann-La Roche Inc., Nutley, NJ) *J. Nutr. 111*(4):665-667 (1981). Twenty-one-day-old rats placed on a viatmin D-deficient diet showed no decrease in serum, 1,25-dihydroxy vitamin D levels after 13 days on this diet. Between 13 and 20 days on this D-deficient diet there was a 50% decrease in serum 1,25-dihydroxy vitamin D. After 34 days, the level of 1,25-dihydroxy vitamin D in serum had dropped to near zero. With a vitamin D-deficient diet lacking calcium, there was an apparent stimulation of 1-hydroxylase, resulting in higher 1,25-dihydroxy vitamin D serum levels after 6-20 days on the diet. Measurement of 1,25-dihydroxy vitamin D levels decreased to near biochemical indicator of vitamin D deficiency in chicks and rats which will complement other established biological criteria for vitamin D deficiency.

TRANSPORT OF LONG AND MEDIUM CHAIN FATTY ACIDS BY *ESCHERICHIA COLI* K12. S.R. Maloy, C.L. Ginsburgh, R.W. Simons and W.D. Nunn (Dept. of Mol. Bio. and Biochem., Univ. of Calif., Irvine, Calif.) *J. Bio. Chem. 256*(8):3735-3742 (1981). Kinetic, metabolic and physical parameters of long and medium chain fatty acid transport by *Escherichia coli* K12 were determined. Uptake of long chain fatty acids (C_{11}-$C_{18:1}$) mediated by the *fadL* gene involves concentrative transport. These results present evidence for separate mechanisms of long and medium chain fatty acid transport in *E. coli*.

PHOSPHOLIPID METABOLISM OF DOG LIVER UNDER HYPOXIC CONDITIONS INDUCED BY LIGATION OF THE HEPATIC ARTERY. J. Matsumoto, T. Tanaka, M. Gamo, K. Saito and I. Honjo (Third Dept. of Internal Med., Kansai Med. Sch., Moriguchi, Osaka, Japan) *Biochim. Biophys. Acta 664*:527-537 (1981). Ischemic hypoxic liver was induced in dogs by ligation of the hepatic artery. About 67% of the dogs died of liver necrosis within 1 or 2 days (severe cases), and the rest survived (mild cases). In the severe cases, the decreases in the contents of total lipids, phospholipids and proteins of the liver after 24 h were 24, 46 and 12%, respectively, of the original values. The marked decrease in phospholipids was due to decreases in the microsomal and mitochondrial fractions. In the mild cases, similar but smaller decreases occurred and decrease of phospholipids occurred only in the microsomal fraction. The main phospholipids were choline and ethanolamine glycerophospholipids, and their molecular species were analyzed. In the severe cases, ligation resulted in relative increases in mono- and diene species and a decrease in polyene species. No increase in phospholipase activity was found at various times after ligation of the hepatic artery. Penicillin-treated dogs all survived and showed little decrease in liver phospholipids.

THIN-LAYER AND GAS-LIQUID CHROMATOGRAPHIC IDENTIFICATION OF NEUTRAL STEROIDS IN HUMAN AND RAT FECES. D.J. McNamara, A. Proia, and T.A. Miettinen (The Rockefeller University, New York, NY 10021) *J. Lipid Res. 22*(3):474-484 (1981). Natural steroids from rat and human feces were fractionated by sequential thin-layer chromatography (TLC) on Florisil, silica gel, and silver nitrate-impregnated silica gel and analyzed by gas-liquid chromatography (GLC). Cholesterol, coprostanol, and coprostanone accounted for more than 95% of the endogenous neutral steroid in human feces, the remainder being predominantly cholestanol. In addition, evidence was obtained for the presence in human feces of trace amounts of epicoprostanol and cholestanone. In rat feces, several cholesterol precursors that probably originated in the skin (and were ingested during fur-licking) were detected in relatively large amounts, accounting for as much as 27% of the total fecal neutral steroids, whereas these steroids were quantitatively trivial in human feces. As with cholesterol, the major dietary plant sterols (sitosterol, campesterol, and stigmasterol) were converted by intestinal bacteria to the corresponding coprostane and ketonic derivatives during intestinal transit in both human beings and rats. This combined us of TLC and GLC provided for the separation of steroids of endogenous and dietary origin that could not be resolved by either system alone. A majority of the fecal steroids could be tentatively identified by their chromatographic behavior in different TCL systems and on GLC, even when reference standards were unavailable.

ORIGINS OF FECAL NEUTRAL STEROIDS IN RATS. T.A. Miettinen, A. Proia, and D.J. McNamara (The Rockefeller University, New York, NY 10021) *J. Lipid Res. 22*(3):485-495 (1981). The origins of rat fecal neutral steroids were studied in male and female animals fed a sterol-free diet and maintained in an isotopic steady state. The specific activity of fecal cholesterol was found to be consistently lower than that of plasma cholesterol and of the fecal bile acids, indicating that a considerable portion of the fecal neutral steroids was derived from cholesterol not in equilibrium with the rapidly exchangeable pool of body cholesterol. This non-exchanging fraction of neutral steroids was larger in male than in female rats; it appeared to have at least two origins: skin surface lipids licked off fur, and sterols newly synthesized by the intestinal mucosa and secreted into the gut lumen. When the ingestion of skin sterols rich in cholesterol precursors was minimized, the proportion of the non-exchanging fraction of fecal neutral sterols fell somewhat, but the output of cholesterol precrusors dropped markedly. This suggests that a significant portion of the non-exchanging fecal cholesterol fraction originated in the intestinal wall by secretion. It can be concluded that the fecal neutral steroids of rats originate primarily from three sources: 1) de novo cholesterol synthesis by the intestinal mucosa, 2) ingested dietary, skin, and fecal sterols, and 3) a rapidly exchangeable cholesterol pool excreted through bile, the intestinal wall, or both.

CHOLESTEROL TRANSFER FROM NORMAL AND ATHEROGENIC LOW DENSITY LIPOPROTEINS TO MYCOPLASMA MEMBRANES. J.J. Mitschelen, R.W. St. Clair, and S.H. Hester (Dept. of Pathology, Arteriosclerosis Res. Center, Bowman Gray School of Medicine of Wake Forest University, Winston-Salem, NC) *Arteriosclerosis 1*(2):134-143 (1981). The purpose of this study was to determine whether the free cholesterol of hypercholesterolemic low density lipoprotein (LDL) from cholesterol-fed nonhuman primates has a greater potential for surface transfer to cell membranes than does the free cholesterol of normal LDL. The LDLs were isolated from normal and hypercholesterolemic rhesus and cynomolgus monkeys, incubated with membranes from *Acholeplasma laidlawii*, a mycoplasma species devoid of cholesterol in its membranes, and the mass transfer of free choelsterol determined by measuring membrane cholesterol content. When added at an equivalent particle concentration, there was greater enrichment of membranes with free cholesterol from hypercholesterolemic LDL. Calculations on the basis of equivalent free cholesterol from hypercholesterolemic LDL. Calculations on the basis of equivalent free cholesterol content showed no difference in either the rate or extent of free cholesterol transfer from normal or hypercholesterolemic LDL. These studies indicate that marked differences in the cholesterol composition of normal and hypercholesterolemic LDLs do not result in a greater chemical potential for surface transfer of free cholesterol. Thus, if a difference in the surface transfer of free cholesterol is responsible for the enhanced ability of hypercholesterolemic LDL to promote cellular cholesterol accumulation, it must be the result of differences in the interaction of the hypercholesterolemic LDL with the complicated mammalian cell membranes.

ACCUMULATION OF INTERMEDIATE DENSITY LIPOPROTEIN IN PLASMA AFTER INTRAVENOUS ADMINISTRATION OF HEPATIC TRIGLYCERIDE LIPASE ANTIBODY IN RATS. T. Murase and H. Itakura (Third Dept. of Internal Med., Univ. of Tokyo, Hongo, Tokyo, Japan) *Atherosclerosis 39*:293-300 (1981). In an attempt to define the role of hepatic triglyceride lipase in plasma lipoprotein metabolism, in vivo experiments using an antibody specifically prepared against this enzyme were conducted in rats. The antibody gamma globulins were injected into rats three times during a 40 min period. Control rats received non-immune rabbit gamma globulins prepared in the same way as the immune gamma globulins. After treatment, blood was taken and the plasma

was separated. Plasma lipoproteins were fractionated by ultracentrifugation into VLDL, IDL, LDL, and HDL. The data of the present study indicate that hepatic triglyceride lipase mediates the catabolism of remnant lipoproteins by the liver.

INCREASED PROPORTION OF MEDIUM CHAIN FATTY ACIDS IN NYSTATIN-RESISTANT YEAST MUTANTS. J. Nagai, S. Yokoe, M. Tanaka, H. Hibasami and T. Ikeda (Dept. of Biochem., Mie Univ. Sch. of Med., Tsu, Japan) *Lipids 16*(6):411-417 (1981). Fatty acid composition of phospholipids and steryl esters from four nystatin-resistant mutants of *Saccharomyces cerevisiae* was compared to that from the wild strain. All the mutant strains which produce several ergosterol intermediates incorporated two- to three-fold as much medium chain fatty acids, especially 14:0 and 14:1 in phospholipids, and 12:0, 14:0 and 14:1 in steryl esters as the wild strain did. The increase in the relative amount of medium chain fatty acids in these mutants was found at all the growth temperatures and the growth phases examined, and in all the phospholipid species.

DETERMINATION OF VITAMIN A STABILITY IN MASH AND PELLETED FEEDS BY A BIOLOGICAL PROCEDURE. I. Nir, R. Cohen and I. Kafri (Hebrew Univ. of Jerusalem, Faculty of Ag., Rehovot, Israel) *Poultry Sci. 60*:1022-1025 (1981). A study was conducted with chicks to determine the effect of pelleting commercial feeds on vitamin A stability. Recovery of vitamin A by a chemical procedures was compared to a biological method in which hepatic stores of vitamin A were determined. Pelleting of the feeds, composed essentially of cereals, soybean meal, and fish meal did not cause an appreciable reduction in the stability and availability of gelatin-coated vitamin A acetate. Chemical determination of vitamin A in feeds was highly correlated to its determination by the biological procedure (r=.88).

PLATELET LIPID COMPOSITION AND PLATELET AGGREGATION IN HUMAN LIVER DISEASE. J.S. Owen, R.A. Hutton, R.C. Day, K.R. Bruckdorfer and N. McIntyre (Department of Biochemistry and Chemistry, Royal Free Hospital School of Medicine, University of London, London, WCIN 1BP) *J. Lipid Res. 22*(3): 423-430 (1981). We hypothesized that, in patients with liver disease, platelets would have an increased cholesterol/phospholipid ratio and that this might affect aggregation in vitro. Platelet aggregation by adrenaline and ADP was measured in 34 patients with a variety of liver diseases and in 20 normal subjects and the values were related to platelet lipid composition. The platelet cholesterol/phospholipid ratio was 13% higher in the patients. Platelet aggregation was reduced in most of the patients. Cross-incubation and hemostasis studies indicated that there were no inhibitory factors present in the plasma; the defect was in th platelets. The phospholipid and fatty acid compositions of our patient platelets were abnormal: the lecithin/sphingomyelin ratio was increased and was inversely correlated with aggregation; the proportion of arachidonic acid was decreased and positively correlated with the aggregation. In our patients with liver diseases the effects of the altered phospholipid and fatty acid composition presumably overrode those of the increased cholesterol content so that instead of enhanced aggregation, only reduced or normal aggregation was seen. We conclude that the reduced platelet aggreagation seen in liver disease may reflect a decrease in arachidonic acid availability for prostaglandin and/or thromboxane production.

LIPOPROTEINS IN FAMILIAL HYPERALPHALIPOPROTEINEMIA. W. Patsch, I. Kuisk, C. Glueck and G. Schonfeld (Depts. Prev. Med. and Med., Washington Univ. School of Med., St. Louis, MO) *Arteriosclerosis 1*(2):156-161 (1981). To describe the plasma lipoproteins in familial hyperalphalipoproteinemia more completely, a kindred consisting of the proband, her affected father, and her two affected brothers was studied. Fasting plasmas were analyzed for lipoprotein lipids by combined preparative ultracentrifugal and precipitation methods. Levels of apolipoprotein A-I and apolipoprotein A-II, the major apoproteins of high density lipoproteins, were assayed by radioimmunoassay. The flotation properties of very low desnity, low density and high density lipoprotein were determined by zonal ultracentrifugation, and the isolated high density lipoprotein subfractions were characterized according to their lipid and apoprotein compositions. Total cholesterol of all subjects was normal, but triglycerides were elevated (above the 90th percentile) in the two brothers. The high density lipoprotein cholesteorl and apolipoprotein A-I and A-II values for the relatives are above the 95th percentile for sex and age, while the levels of the proband are the highest recorded in our laboratories. On zonal ultracentrifugation HDL_2 was grossly elevated, accounting for most of the rise in the high density lipoprotein fraction. Thus, in this kindred, high density lipoprotein was quantitatively rather than qualitatively unusual. It is of interest that hypertriglyceridemia and hyperalphalipoproteinemia coexisted in the siblings. These concurrent elevations differ from the expected reciprocal relationship between high density lipoprotein and very low density lipoprotein levels in plasma, and suggests that the two abnormalities may be independently transmitted.

ODD-NUMBERED FATTY ACIDS IN PHOSPHATIDYLCHOLINE VERSUS PHOSPHATIDYLETHANOLAMINE OF VITAMIN B_{12}-DEPRIVED RATS. J.J. Peifer and R.D. Lewis (Dept. of Foods and Nutr., Univ. of GA, Athens, GA) *Pro. Soc. Exp. Bio. Med. 167*:212-217 (1981). In response to a 20-week dietary deficiency of vitamin B_{12} rats accumulated two to four times more heptadecanoic acid (17:0) and pentadecanoic acid (15:0) in phosphatidyl choline (PC) of their cerebrum. Considerably smaller amounts of these odd-numbered fatty acids (ONFA) were present in phosphatidylethanolamine (PE) of either the cerebrum or liver of rats deprived of, or supplemented with, vitamin B_{12}. Vitamin B_{12}-deficient rats had twice as much ONFA in PC of their cerebrum than in PC of their liver, and the vitamin deficiency had little or no effect on the amount of ONFA in PE of the cerebrum or liver. The greater incorporation of ONFA into cerebral PC appears to be correlated with a greater abundance of palmitic acid (16:0) and related chain length—even-numbered fatty acids (14:0 and 16:1) in the phospholipid of the rat. Similar relationships between the abundance of ONFA and 16:0 in neural PC were previously found in an infant with geneticall-ly defective B_{12}-coenzyme systems.

THE ROLE OF GONADAL STEROIDS IN ARACHIDONATE-INDUCED MORTALITY IN MICE. J.C. Penhos, F. Rabbani, A. Myers, E. Ramey and P. Ramwell (Dept. of Physiology, Georgetown Univ. Medical Center, Washington, DC 200007) *Proc. Soc. Exp. Biol. Med. 167*(1):98-100 (1981). Female mice are significantly more resistant than male mice to intravenous arachidonate. Estradiol treatment of castrated males, but not females or intact males, provides protection. Testosterone treatment, on the other hand, increased the mortality rate of the intact or castrated males but had no significnat effect on the responsiveness to arachidonate in female mice. Progesterone pretreatment did not modify the mortality rate in any of the groups studied. Another steroid, cortisone, is much more protective than the ovarian steroids. In both males and females, intact or castrated, exogenous cortisone greatly increased the ability to withstand an arachidonate injection. These data suggest that gonadal and cortical steroids act at a critical step in tissue response to arachidonate and alter its ultimate effect on mobidity and mortality.

EFFECTS OF LONG-TERM PROTEIN DEFICIENCY ON PLASMA LIPOPROTEIN CONCENTRATIONS AND METABOLISM IN RHESUS MONKEYS. O.W. Portman, M. Alexander, M. Neuringer, D.R. Illingworth and S.S. Alam (Oregon Regional Primate Res. Center, Beaverton, OR) *J. Nutr. 111*(4):733-745 (1981). Lipoprotein concentrations and metabolism were studied in 5- and 9-year-old rhesus monkeys (*Macaca mulatta*). Both age groups had been divided into control (13.8% of the calories as protein) and low-protein (3.7% protein) subgroups at birth. All were tested before and after their dietary lipid was changed from corn oil to butter plus cholesterol. The concentrations of very-low-density and high density$_2$ lipoproteins (VLDL and HDL_2) tended to be higher in monkeys of the low-protein group and butter plus cholesterol accentuated the difference. All monkeys of the low protein group had elevated levels of at least one of these two classes of lipoproteins.

INCORPORATION OF PHOTOSENSITIVE FATTY ACID INTO PHOSPHOLIPIDS OF *ESCHERICHIA COLI* AND IRRADIATION-DEPENDENT CROSS-LINKING OF PHOSPHOLIPIDS TO MEMBRANE PROTEINS. S.C. Quay, R. Radhakrishnan, and H.G. Khorana (Dept. of Bio. and Chem., Mass. Inst. of Tech., Cambridge, MA) *J. Biol. Chem. 256*(9):4444-4449 (1981). In an approach to the study of phospholipid-protein interactions in biological membranes, the photoactivable fatty acids, ω-(*m*-azidophenoxy)-undecanoic acid (I) and ω-(*m*-diazirinophenoxy)-hexadecanoic acid (II), were incorporated biosynthetically into the phospholipids of the *Escherichia coli* fatty acid auxotroph, strain K1060-B5. The extent of incorporation of the two fatty acids was 43% and 21%, respectively, of the total fatty acid content of the phospholipids. Membrane vesicles prepared from cells grown on the fatty acid supplements and $[^{32}P]H_3PO_4$ were irradiated at suitable wavelengths to generate the reactive nitrene or carbene intermediates. The present results, together with the previously observed nonreactivity of the nitrene generated from I to undergo C-H insertion, show that the use of carbene precursors such as II is promising for chemical analysis of specific phospholipid-protein interactions in bacterial membranes under biologicall meaningful conditions.

IDENTIFICATION OF THE MANNOSYL DONORS INVOLVED IN THE SYNTHESIS IF LIPID-LINKED OLIGOSACCHARIDES. J.I. Rearick, F. Fujimoto and S. Kornfeld (Wash. Univ. Sch. of Med., Depts. of Med. and Bio. Chem., St. Louis, Missouri) *J. Bio. Chem. 256*(8):3762-2769 (1981). Thy-1$^-$ mutant mouse lymphoma

cells of the class E complementation group lack GDP-mannose: dolichol-P mannosyltransferase and therefore are unable to interconvert GDP-mannose and dolichol-p-mannose. We have used this cell line to define the donor for each of the mannose residues of the major lipid-linked oligosaccharide which has the composition glucose-$_3$mannose$_9$*N*-acetylglucosamine$_2$. Membrane preparations from the mutant cells were incubated with GDP-(^{14}C)mannose and dolichol-P-(^{3}H)mannose and the labeled lipid-linked oligosaccharides wer isolated. This finding indicates that the manosyltransferase involved in the synthesis of the lipid-linked oligosaccharide are highly specific with respect to the mannosyl donor and somewhat less specific with respect to the oligosaccharide acceptor.

BLOOD LIPIDS AND THEIR DISTRIBUTION IN LIPOPROTEINS IN HYPERINSULINEMIC SUBJECTS FED THREE DIFFERENT LEVELS OF SUCROSE. S. Reiser, M.C. Bickard, J. Hallfrisch, O.E. Michaelis, and E.S. Prather (Beltsville Human Nutr. Res. Center, Human Nutr., Sci. and Ed. Admin., U.S. Dept. of Ag., Beltsville, MD) *J. Nutr. 111*:1045-1057 (1981). The effects of dietary sucrose on blood lipids and their distribution in lipoprotein fractions were determined in 12 males and 12 females diagnosed as carbohydrate-sensitive on the basis of an exaggerated insulin response to a sucrose load. The subjects were fed diets containing 5%, 18% or 33% of the total calories as sucrose for 6 weeks each in a crossover design. Initial body weights were essentially maintained. These results indicate that sucrose intake at levels now common in the American diet by carbohydrate-sensitive males could lead to a blood lipid profile associated with coronary risk.

COMPARATIVE EFFECTS OF CHOLIC, CHENODEOXYCHOLIC, AND URSODEOXYCHOLIC ACIDS ON MICELLAR SOLUBILIZATION AND INTESTINAL ABSORPTION OF CHOLESTEROL. M.O. Reynier, J.C. Montet, A. Gerolami, C. Marteau, C. Crotte, A.M. Montet, and S. Mathieu (Unité de Recherches de Pathologie Digestive, U 31 INSERUM, 46 Boulevard de la Gaye, 13009 Marseille, France) *J. Lipid Res. 22*(3):467-473 (1981). Cholesterol absorption was studied in mice receiving cholic, chenodeoxycholic, or ursodeoxycholic acids (0.2% of the diet) for 2 months. Cholesterol absorption was greater with cholic acid (79%) than with chenodeoxycholic acid feeding (60%) and the lowest levels were observed during ursodeoxycholic acid feeding (37%). Under the three diets, bile acid pool and bile acid secretion were not different. Biliary cholesterol secretion was increased by cholic acid. The bile acid fed represents at least 80% of total bile acids. Micellar solubilization of oleic acid and cholesterol in the presence of each tauro-conjugated bile salt (10 mM) was determined in vitro by the coprecipitation method. Whatever the pH conditions, taurochenodeoxycholate solubilized significantly more cholesterol and more oleic acid than taurocholate. Tauroursodeoxycholate had the poorest detergent properties for both lipids. The differences between the three bile salts for cholesterol solubilization were enlarged by lowering pH and by high oleic acid concentration. Therefore the decrease in cholesterol absorption observed during ursodeoxycholic acid feeding could be explained by the poor detergent properties of this bile salt species. On the other hand, there is no relationship between the detergent properties of taurochenodeoxycholate and taurocholate and their effects of cholesterol absorption in mice. These results suggest that, in this particular case, micellar solubilization is not the rate limiting step in cholesterol absorption.

INTESTINAL CHOLESTEROL UPTAKE FROM MIXED MICELLES. IN VITRO EFFECTS OF TAUROCHOLATE, TAUROCHENODEOXYCHOLATE AND TAUROURSODEOXYCHOLATE. M.O. Reynier, J.C. Montet, C. Crotte, C. Marteau and A. Gerolami (Unite de Recherches de Pathologie Digestive, U 31, INSERM, 46 Boulevard de la Gaye, 13009 Marseilles, France) *Biochim. Biophys. Acta 664*:616-619 (1981). Cholesterol uptake by everted rat jejunal sacs is lower from mixed micelles containing tauroursodeoxycholate than from those with taurocholate or taurochenodeoxycholate. This occurs in spite of a greater saturation with cholesterol of tauroursodeoxycholate micelles as measured by equilibrium solubility studies. The results suggest that cholesterol saturation of solutions containing tauroursedeoxycholate is overestimated when calculated with reference to solubility in micellar form.

EFFECT OF LEAD ON HEMOGLOBIN-CATALYZED LIPID PEROXIDATION. S.R. Ribarov, L.C. Benov and I.C. Benchev (Dept. of Biophys., Univ. Sch. of Med., Pleven, Bulgaria) *Biochim. Biophys. Acta 664*:453-459 (1981). Lead significantly increases the rate of hemoglobin-catalyzed lipid peroxidation. The inhibition of this effect by superoxide dismutase and catalase suggests that superoxide radicals and H_2O_2 are somehow involved. Furthermore, lead catalyzes methemoglobin formation both in pure hemoglobin solutions and in hemolysates in which all protecting systems are present. It is speculated that a superoxide radical released in lead-catalyzed hemoglobin autoxidation may initiate the peroxidation of unsaturated fatty acids in red cell membrane. This hypothesis is supported by the fact that the preliminary conversion of oxyhemoglobin to methemoglobin decreases the rate of peroxidation. A conclusion is drawn that in native erythrocyte Pb^{2+} may exert a pro-oxidant effect, possibly by interacting with hemoglobin.

CELLULARITY OF BOVINE ADIPOSE TISSUES: DEVELOPMENTAL CHANGES FROM 15 TO 65 PERCENT MATURE WEIGHT. J. Robeline (Laboratoire de la Production de Viande, Centre de Recherches de Clermont-Ferrand, I.N.R.A. Theix, 63110 Beaumont, France) *J. Lipid Res. 22*(3):452-457 (1981). Changes in the cellularity of various adipose tissues in growing cattle were analyzed. Fifty male cattle were slaughtered between 15 and 65% of mature weight. The whole adipose mass, separated by dissection, was divided into three parts: subcutaneous, intermuscular, and internal adipose tissues. Lipid content, cell size and distribution, as well as cell number of these three parts were determined. Adipose cells became 15 times greater from 15 to 65% mature weight, whereas total adipose cell number increased only 1.8-fold. However, a significant hyperplasia. Over the whole period studied (15-65% mature weight), hyperplasia was far higher in subcutaneous adipose tissue than in other tissues. This is discussed as related to the higher relative growth of this tissue. In each fatty tissue, two identical developmental periods were observed. Each of them began by an increase in small-sized cells (hyperplasia) followed by the filling of these cells (hypertrophy). These two periods were particularly clear in the case of subcutaneous tissue, in which the second hyperplasia occurred slightly later than in other fatty tissues. So, in all respects, subcutaneous fatty tissue appears to develop later than other tissues studied.

EFFECT OF SOY PROTEIN, CASEIN AND TRYPSIN INHIBITOR ON CHOLESTEROL, BILE ACIDS AND PANCREATIC ENZYMES IN MICE. D.M. Roy and B.O. Schneeman (Dept. of Nutr., Univ. of Calif., Davis, CA) *J. Nutr. 111*(5):878-885 (1981). Dietary vegetable proteins may lower plasma cholesterol compared to animal proteins. We considered the possibility that lower digestibility and the trypsin inhibitor (TI) content of plant proteins could lend to alteratiosn in bile acid metabolism and exocrine pancreatic function mediating some of this change. Mice were fed cholesterolemic diet of different protein source and TI content: casein, soy protein isolate, or casein plus TI for 4 weeks. Plasma and liver cholesterol were measured; pancreata, intestinal contents and mucosal scrapes were collected for bile acid, trypsin, chymotrypsin, amulase, and lipase assays. Therefore, soyben TI does not seem to affect cholesterol metabolism, though it greatly affects pancreatic secretion. On the other hand, soy protein has a marked effect on bile acid and cholesterol metabolism, which may be a function of protein quality.

DIETARY ETHANOL-INDUCED MODIFICATIONS IN HYPERLIPOPROTEINEMIA AND ATHEROSCLEROSIS IN NONHUMAN PRIMATES (*Macaca nemestrina*). L.L. Rudel, C.W. Leathers,M.G. Bond and B.C. Bullock (Arteriosclerosis Res. Center and Dept. of Comparative Med., Bowman Gray School of Medicine of Wake Forest Univ., Winston-Salem, NC) *Arteriosclerosis 1*(2):144-155 (1981). Male *Macaca nemestrina* were studied in an experiment with a 2 × 2 factorial design. Diets contained low vs high cholesterol levels and no ethanol or ethanol, as 36% of the calories substituted isocalorically for carbohydrate. After receiving their diets for 18 months, the monkeys had blood samples drawn for lipoprotein analyses, and then were killed for evaluation of the extent of atherosclerosis. Ethanol-fed groups had significantly increased concentrations of serum cholesterol, triglycerides, low density lipoprotein (LDL), and high density lipoprotein (HDL). Dietary cholesterol had the effect of increasing the concentration of LDL and decreasing the concentration of HDL. Significant interactions were found between the effects of ethanol and cholesterol on HDL and LDL. Ethanol significantly decreased the cholesterol-induced atherosclerosis found in the aorta and coronary arteries. Highly significant correlations between coronary artery atherosclerosis and LDL molecular weight, inverse HDL concentration, and LDL cholesterol ester pattern were found. Different relationships with aortic atherosclerosis were found; LDL molecular weight and cholesterol ester pattern were highly correlated, while HDL concentration was not. This suggests that the effect of ethanol in reducing the development of atherosclerosis may have been mediated through its effects on the plasma lipoproteins.

HIGH DENSITY LIPOPROTEIN UTILIZATION BY DISPERSED RAT LUTEAL CELLS. L.A. Schuler, K.L. Langenberg, J.T. Gwynne and J.F. Strauss (Dept. of Obstetrics and Gynecology and Physio., Univ. of Penn., Sch. of Med., Philadelphia, PA) *Biochim. Biophys. Acta 664*:583-601 (1981). Utilization of lipoproteins by cells prepared by collagenase dispersion of ovaries of immature gonadotropic-primed rats was studied. Human and rat HDL increased basal progestin secretion and incorporation of (^{14}C)oleate into cellular sterol esters 2-fold during a 2 h incubation, with

maximal stimulation occuring at a lipoprotein sterol concentration of 125 μg/ml. This concentration of HDL cholesterol also increased progestin production by cells stimulated with dibutyryl cyclic AMP. We conclude that lipoprotein-carried cholesterol is an important substrate for rat luteal cells and that these cells possess a specific mechanism for the uptake of HDL.

SELECTIVE INDUCTION OF DE NOVO PROSTAGLANDIN BIOSYNTHESIS IN RABBIT KIDNEY CORTEX. M. Schwartzman and A. Raz (Dept. of Biochem., The George S. Wise Center of Life Sci., Tel Aviv Univ., Ramat Aviv, Tel Aviv, Israel) *Biochim. Biophys. Acta 664*:469-474 (1981). Ureter-obstructed kidney develops during perfusion an enhanced responsiveness to brandy-kinin-stmulated prostaglandin release. This enhance prostaglandin generation results from de novo synthesis of prostaglandin synthetase and acylhydrolase enzymes during the perfusion and is therefore unaffected by acylhydrolase enzymes during the perfusion and is therefore unaffected by acetylsalicyclic acid (asprin) inhibition of prostaglandin synthesis prior to initiation of perfusion. Studies were carried out to identify the renal cellular site in which the newly synthesizing prostaglandin generating system is localized. It thus appears that the induced coritcal acylhydrolase and prostaglandin synthease activities are tightly coupled and that the true molecular form of precursor arachidonate for this prostaglandin generating system is esterified and not free arachidonate.

REGULATION OF BILE ACID SYNTHESIS. MEASUREMENT OF CHOLESTEROL 7α-HYDROXYLASE ACTIVITY IN RAT LIVER MICROSOMAL PREPARTIONS IN THE ABSENCE OF ENDOGENOUS CHOLESTEROL. S. Shefer, F.W. Cheng, S. Hauser, A.K. Batta and G. Salen (Dept. of Med., Col. of Med. and Dent. of NJ, NJ Med. Sch., Newark, NJ) *J. Lipid Res. 22*:532-536 (1981). A rapid procedure was developed to measure hepatic cholesterol 7α-hydroxylase activity in the absence of endogenous microsomal cholesterol. This method involves the preparation of an acetone powder from the microsomal fraction of rat liver that remains its cholesterol 7α-hydroxylase activity and contains virtually no endogenous cholesterol. The enzyme activity is measured in the presence of labeled exogenous cholesterol as the only substrate source, and can be expressed in terms of picomoles of produce formed when a simple isotope incorporation procedure is employed. Optimal assay conditions were determined and the reproducibility of the acetone powder cholesterol 7α-hydroxylase assay was established.

EFFECT OF CANDICIDIN ON CHOLESTEROL AND BILE ACID METABOLISM IN THE RAT. A.K. Singhal, E.H. Mosbach and C.P. Schaffner (Dept. of Surgery, Beth Israel Med. Center, New York, NY) *Lipids 16*(6):423-426 (1981). Sterol metabolism studies were carried out in rats maintained on a diet containing a polyene antibiotic, candicidin, (30 mg/kg/day) for 2-½ months. Compared to the controls, the candicidin-treated animals had a smaller food intake and weight gain during this period. There was no difference between the 2 groups in serum cholesterol levels, biliary cholesterol or bile acid concentrations. However, in the experimental group, liver cholesterol content decreased by 27% and hepatic HMG-CoA reductase increased by 36%. Candicidin administration produced an 84% increased in neutral sterol output without change in bile acid output. Cholesterol absorption was reduced 80% by candicidin feeding. The weight of ventral prostate was reduced 33% by candicidin administration. Prostatic HMG-CoA reductase levels were 3 times higher than those of the liver, but enzyme activity was unchanged by candicidin treatment.

PLASMA LIPIDS IN TROPICAL SPRUE. C. Tiruppathi, P.G. Hill and V.I. Mathan (Wellcome Res. Unit, Christian Med. College Hosp., Vellore, India) *Am. J. Clin. Nutr. 34*:1117-1120 (1981). Previous studies indicated that many patients with tropical sprue in southern India have triglyceride accumulation within the cells of the intestinal mucosa. This could be due to essential fatty acid deficiency as a result of steatorrhea in subjects on a diet normally low in linoleic acid. Plasma lipids have, therefore, been studied in patients with tropical sprue and the results compared to values observed in healthy controls. All of these observations indicate essential fatty

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acid depletion although unequivocal evidence of essential fatty acid deficiency was not present in any of the patients. The degree of essential fatty acid depletion observed is unlikely to be the cause of the mucosal accumulation of triglyceride in tropical sprue.

METABOLIC HETEROGENEITY OF APOLIPOPROTEIN B IN THE RAT. C.E. Sparks and J.B. Marsh (The Medical College of Pennsylvania, Dept. of Physiology and Biochemistry, Philadelphia, PA 19129) *J. Lipid Res. 22*(3):519-527 (1981). Triglyceride-rich lipoprotein apoprotein catabolism was studied in rats from 5 to 60 min after intravenous injection of ^{125}I-labeled lipoproteins. The plasma and liver labeled apoprotein content was analyzed by gel filtration column chromatography using an elution buffer containing 1% sodium dodecyl sulfate. The method resolved two B apoproteins of lower (apo B_l) and higher (apo B_h) molecular weight. The findings suggest that there is differential hepatic catabolism of a subpopulation of triglyceride-rich lipoproteins containing apo B_l 1 A population of triglyceride-rich lipoproteins containing apo B_h preferentially enters the low density lipoprotein pool with a slower catabolism. The results are consistent with an hypothesis that apo B_l mediates binding and rapid hepatic catabolism of its associated lipoproteins. Metabolic heterogeneity of the triglyceride-rich lipoproteins may be explained by the molecular heterogeneity of apoprotein B.

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