Abstracts

Biochemistry and nutrition

EFFECT OF 1,3-BUTYLENE GLYCOL ON GROWTH AND *IN VIVO* AND *IN VITRO* LIPOGENESIS BY TURKEY POULTS. R.W. Rosebrough and N.C. Steele (Nonruminant Animal Nutr. Lab., U.S. Dept. of Agr., Beltsville, MD) *Poultry Sci. 60*:1448-1453 (1981). A series of feeding trials lasting 21 days was conducted with Large White turkey poults to determine the effects of 0, 12.5, and 25% energy as 1,3-butylene glycol (BG) on growth and on both *in vivo* and *in vitro* lipogenesis. The substitution of 12.5 and 25% of the energy as BG depressed growth and feed efficiency of 21-day-old poults ($P<.01$). The relative liver size was increased by BG ($P<.01$) by BG. *In vivo* lipogenesis, determined by the incorporation of tritiated water into liver fatty acids, was decreased ($P<.05$) by BG. The evolvement of CO_2 from both (1-^{14}C) acetate and from (U-^{14}C) glucose was decreased by BG. The results of this study indicate that while lipogenesis can be decreased by BG, growth is also decreased. Therefore, the regulation of growth parallels the regulation of lipid synthesis in the turkey poults.

STABILIZATION OF LIVER LIPASE IN VITRO BY HEPARIN OR BY BINDING TO NON-PARENCHYMAL LIVER CELLS. K. Schoonderwoerd, W.C. Hülsmann, and H. Jansen (Dept. of Biochemistry I, Medical Faculty, Erasmus University Rotterdam, P.O. Box 1738, 3000 DR Rotterdam, The Netherlands) *Biochim. Biophys. Acta 665*(2):317-321 (1981). The effect of heparin on the secretion of acylglycerol hydrolase activity by isolated parenchymal liver cells was studied. In the presence of heparin, the lipase activity, secreted in 3 h, was almost doubled. Heparin did not influence the activity of the enzyme, but affected the stability of the enzyme. In the absence of heparin, the triacylglycerol hydrolase activity declined to 50% of the initial value during 1 h incubation at 37 C. The addition of heparin prevented this loss of activity almost completely. The optimal stabilization of enzyme activity was reached at 15 U heparin/ml NaCl (1 M) and protamine sulphate (120 μg/ml) abolished this effect of heparin. Instead of heparin, liver lipase activity could also be stabilized by binding to non-parenchymal liver cells. The results are discussed in connection with binding of the enzyme in vivo.

RELEASE AND METABOLISM OF ARACHIDONIC ACID IN HUMAN NEUTROPHILS. C.E. Walsh, B.M. Waite, M.J. Thomas, and L.R. DeChatelet (Dept. of Biochem., Bowman Gray School of Medicine, Wake Forest Univ., Winston-Salem, North Carolina 27103) *J. Biol. Chem. 256*(14):7228-7234 (1981). Dual radiolabel incorporation of [^{3}H] arachidonic acid and [^{14}C] palmitate or [^{14}C] stearate by human neutrophils was employed to study both the release and metabolism of arachidonic acid. Results indicate the involvement of a phospholipase A_2 mechanism causing [^{3}H]-arachidonate release from membrane phospholipid. Phosphatidylinositol and phosphatidylcholine were the sources of [^{3}H]-arachidonate; about twice as much radiolabeled phosphatidylinositol was degraded as phosphatidylcholine. Challenge of neutrophils with opsonized zymosan and calcium ionophores caused the release of [^{3}H] arachidonate; however, ionophores but not opsonized zymosan led to the production of [^{3}H] hydroxyicosatetraenoic acid and [^{3}H] dihydroxyicosatetraenoic acid. These products were preferentially released by neutrophils into the extracellular milieu in contrast with free [^{3}H] arachidonate which remained cell associated. One-third of the [^{3}H] hydroxyicosatetraenoic acid but not [^{3}H] dihydroxyicosatetraenoic acid was reincorporated into cellular lipid, primarily phospholipid. No significant production of [^{3}H]-prostaglandin or [^{3}H] thromboxane was detected. In contrast to zymosan and ionophore, phorbol myristate acetate, another potent stimulant of neutrophil oxidative metabolism and degranulation, did not release [^{3}H] arachidonate.

DEGRADATION OF PHOSPHATIDYLINOSITOL BY SOLUBLE ENZYMES OF RAT GASTRIC MUCOSA. M.K. Wassef and M.I. Horowtiz (Department of Biochemistry, New York Medical College, Valhalla, NY 10595) *Biochim. Biophys. Acta 665*(2):234-243 (1981). Rat gastric mucosa homogenates contain two enzymatic systems for hydrolyzing phosphatidylinositol: a deacylation activity yielding lysophosphatidylinositol and free fatty acid, and a phospholipase C-like activity producing 1,2-diacylglycerol and inositol phosphates. These activities were found mainly in the 105000 × *g* supernatant and could be distinguished by differential stabilities, metal requirements and the action of deoxycholate and mepacrine. Each lipolytic reaction displayed a major pH optimum at 7.5 and a minor pH optimum at 5.5. The deacylation system was 8-10 times as active as the phospholipase C, with an apparent K_m of 0.63 mM towards 1-acyl-2-arachidonylphosphatidylinositol at pH 7.5. The phospholipase C activity, on the other hand, hydrolyzed 1-acyl-2-arachidonylphosphatidylinositol or 1-acyl-2-arachidonylphosphatidylethanolamine and yielded 1-acyl-2-arachidonyl-*sn*-glycerol. This 1,2-diacylglycerol could be phosphorylated to form 1-acyl-2-[^{14}C] arachidonyl-*sn*-phosphoglycerol (phosphatidic acid), but could not be hydrolyzed to produce free [^{14}C] arachidonic acid using stomach mucosal microsomes. Phospholipase A_2 and phospholipase C attack 1-acyl-2-arachidonylphosphatidylethanolamine and phosphatidylinositol equally well, but hydrolyze 1-acyl-2-arachidonylphosphatidylcholine poorly.

INHIBITION OF MEMBRANE-BOUND HEPATIC 3 HYDROXY-3 METHYL GLUTARYL CoA REDUCTASE AS THE CONSEQUENCE OF ALTERED MEMBRANE FLUIDITY. E. Wulfert, G. Boissard, C. Legendre and C. Baron (Centre de Recherches, Laboratoires FOURNIER-DIJON, 42-rue de Longvic, 21300 CHENOVE, FRANCE) *Artery 9*(2):120-131 (1981). The activity of hydroxy methyl glutaryl-CoA reductase in microsomes from rat and from human liver was inhibited in a non-competitive manner by fenofibric acid. High affinity of the microsomal preparation for the ligand allowed a one-step purification of the microsomal enzyme preparation, using a Sepharose gel coupled to the phenol analogue of fenofibric acid. The Arrhenius plots of partially purified hydroxy methyl glutaryl-CoA reductase in the microsomal fraction from rat liver showed that the break in the activation energy at 11 C was abolished by the ligand. The results in the present study may be consistent with a modulation of membrane-bound HMG-CoA reductase activity.

METABOLISM OF FATTY ACIDS IN RAT BRAIN MICROSOMAL MEMBRANES. E.E. Aeberhard, M. Gan-Elepano, and J.F. Mead (Laboratory of Nuclear Medicine and Radiation Biology, University of California, 900 Veteran Ave., Los Angeles, CA 90024) *Lipids 16*(10):705-713 (1981). Using a technique in which substrate fatty acids are incorporated into microsomal membranes followed by comparison of their rates of desaturation with those of exogenous added fatty acids, it has been found that the desaturation rate may be greater for the membrane-bound substrate than for the added fatty acid. Moreover, the product of the membrane-bound substrate is incorporated into membrane phospholipid whereas the product of the exogenous substrate is found in di- and triacyl glycerols and in free fatty acids, as well. These and other findings point to a normal sequence of reaction of membrane lipids with membrane-bound substrates involving transfer of fatty acid from phospholipid to the coupled enzyme systems without facile equilibration with the free fatty acid pool.

ON THE MECHANISM OF PLASMA CHOLESTEROL REDUCTION IN THE RAT GIVEN PROBUCOL. S. Balasubramaniam, D.M. Beins, and L.A. Simons (Lipid Research Div., St. Vincent's Hospital and School of Med., Univ. of New South Wales, Sydney, N.S.W., Australia) *Clin. Sci. 61*(5):615-619 (1981). 1. The effects of the cholesterol-lowering drug probucol on lipoprotein metabolism and on the key enzymes that regulate hepatic cholesterol metabolism in the rat were studied. 2. Probucol given for 2 weeks was accompanied by a significant reduction in plasma concentrations of low-density and high-density lipoproteins (LDL,HDL). The fractional catabolic rates of the apolipoproteins of HDL and LDL (apoHDL, apoLDL) were not affected by probucol, although the absolute rates of catabolism of both the apolipoproteins were significantly reduced. 3. The activities of 3-hydroxy-3-methylglutaryl-coenzyme A (CoA) reductase and cholesterol 7α-monooxygenase, as well as the rate of hepatic sterol synthesis, were unchanged during the first 2 weeks of probucol. More prolonged

probucol led to inhibition of the activity of these enzymes and reduction in sterol synthesis, although the liver cellular content of cholesterol significantly increased. 4. It is postulated that a principal mode of action of the drug is to reduce the rate of lipoprotein synthesis.

ASSESSMENT OF THE ESSENTIAL FATTY ACID REQUIREMENT IN GERBILS BY POLYUNSATURATED FATTY ACID RATIO. S-H.W. Chu and D.M. Hegsted (Dept. of Nutrition, Harvard School of Public Health, Boston, MA 02115) *J. Nutr. 111*(9): 1548-1555 (1981). Essential fatty acid status in the gerbil was assessed using the ratio of 20:3ω9 (5,8,11-eicosatrienoic acid) to 20:4ω6 (arachidonic acid) derived from liver phospholipid. Both a fat-free diet and a diet containing 20% hydrogenated coconut oil produced an essential fatty acid deficiency. The minimum requirement for 18:2ω6 (linoleic acid) in the gerbil, like that in the rat, was estimated at 1% kcal of the diet when graded levels of safflower oil were added to a purified diet containing hydrogenated coconut oil. Dietary cholesterol did not affect the minimum requirement, but accentuated the triene:tetraene ratio when the dietary linoleate level was below the requirement.

SIZE AND NUMBER OF ADIPOCYTES AND MEASURES OF BODY FAT IN BOYS AND GIRLS 10 TO 18 YEARS OF AGE. W.C. Chumlea, J.L. Knittle, A.F. Roche, R.M. Siervogel, and P. Webb (Fels Research Institute and Depart. of Pediatrics, Wright State Univ. School of Med., Yellow Springs, OH 45387) *Am. J. Clin. Nutr. 34*(9):1791-1797 (1981). In 111 boys and girls, 10 to 18 yr of age, body density was measured by underwater weighing, and the size of adipocytes in adipose tissue from the buttocks was measured by the osmium tetroxide method. From these two measures, estimates of percentage body fat, total body fat, and adipocyte number were computed for most of the children. Their skeletal age was also calculated by an acceptable method. Across chronological age, the girls have significantly larger mean values of total and percentage body fat and larger and more numerous adipocytes than the boys. The mean number of adipocytes of each sex is within adult levels, as is the mean size of the adipocytes in the girls. The boys' mean adipocyte size is below the adult level. There are negative, significant correlations between percentage body fat and chronological or skeletal age in the boys, and positive significant correlations between total body fat and chronological or skeletal age in the girls. Also, adipocyte size is positively correlated with percentage body fat but only in the boys. With the effects of chronological age removed, percentage body fat was significantly and negatively correlated with skeletal age in boys only. All other correlations among the variables were not statistically significant.

INFLUENCES OF DIETARY VITAMIN E AND SELENIUM ON THE OXIDANT DEFENSE SYSTEM OF THE CHICK. G.F. Combs, Jr. (Department of Poultry and Avian Science and Division of Nutritional Sciences, Cornell University, Ithaca, NY) *Poultry Sci. 60*(9):2098-2105 (1981). The effects of dietary vitamin E and selenium on the oxidant defense system (glutathione peroxidase, catalase, glutathione reductase, reduced glutathione, and superoxide dismutase) were investigated in the chick. Two-week-old chicks were reared using a vitamin E-free, low-selenium, semipurified basal diet alone or supplemented with vitamin E (100 IU/kg) and/or selenium (.10 ppm). Whereas vitamin E sustained chick growth, survival, and protection from exudative diathesis (ED), it did not significantly affect the enzymatic components of the oxidant defense system. Dietary selenium promoted chick growth and protection against ED in the absence of vitamin E and sustained glutathione peroxidase activity in several tissues. The latter effect was associated with decreases in reduced glutathione concentrations observed in liver and blood. Catalase and superoxide dismutase activities were increased in liver and brain in selenium deficiency. Glutathione reductase activities in liver, kidney, lung, and brain were not affected by diet.

TISSUE DISTRIBUTION, UPTAKE, AND REQUIREMENT FOR α-TOCOPHEROL OF RAINBOW TROUT (*Salmo gairdneri*) FED DIETS WITH A MINIMAL CONTENT OF UNSATURATED FATTY ACIDS. C.B. Cowey, J.W. Adron, M.J. Walton, J. Murray, A. Youngson, and D. Knox (N.E.R.C. Institute of Marine Biochemistry, St. Fittick's Road, Aberdeen, AB1 3RA, U.K.) *J. Nutr. 111*(9): 1556-1567 (1981). The metabolism of and requirement for α-tocopherol in rainbow trout fed diets containing 1% linolenic acid as sole source of unsaturated fat and graded levels of tocopherol (0.06-10 mg/100 g) were examined. Fish grew 5-fold over a 16-week period. In liver, tocopherol was concentrated in mitochondria with little in cytosol. Orally administered (^{3}H)-tocopherol was rapidly taken up by plasma and liver but uptake into erythrocytes and white muscle was much slower; in most tissues radioactivity reached a plateau after about 3 days but in red muscle radioactivity increased over a 10-day period. Activities of enzymes that prevent free radical initiated tissue damage did not change in tocopherol deficiency. Tocopherol-deficient trout had no gross or subcellular pathologies even though liver and muscle were severely depleted of the vitamin. Ascorbic acid-stimulated lipid peroxidation in liver organelles indicated a tocopherol requirement of 2-3 mg/100 g diet; the molar ratios of polyunsaturated fatty acids to tocopherol in livers of trout fed diets lacking or supplemented with tocopherol (10 mg/100 g) were 980 and 170, respectively.

EFFECTS OF CLOFIBRATE AND TIADENOL ON THE ELIMINATION OF LIPIDS AND BILE ACIDS IN RAT BILE. P. Cuchet, C. Morrier, F. Cand, and C. Keriel (Laboratoire de Physiologie animale, Universite Scientifique et Medicale de Grenoble, B.P. 53 X-38041, Grenoble, Cedex, France) *Lipids 16*(10): 732-738 (1981). The aim of the work presented here was to compare the biliary elimination of cholesterol and the different bile acids of rats that had been made hypolipidemic by short-term treatments with clofibrate or tiadenol. Both treatments induced a significant decrease in cholesterol output in the bile. The analysis of the different bile acids showed a decrease in dihydroxylated acids elmination (especially CDC acid) without any difference between the 2 sexes. This decrease was associated with an increase in cholic acid excretion. These results are directly correlated with the dose of the administered hypolipidemic drug. The drugs caused a significant increase in the ratio of trihydroxylated acids to dihydroxylated acids. The maximal effect on the concentration of the biliary acids of the bile and on the output was obtained, for both drugs, with a treatment of 200 mg/kg/day. Clofibrate had a greater effect than tiadenol at this dose. Both drugs show a greater effect on lowering serum lipid levels in female animals when compared to males, whereas elimination of bile cholesterol and modifications of bile acids were greater in male animals than female animals.

COMPARATIVE STUDY OF AN ADENOSINE TRIPHOSPHATASE TRIGGER-FUSED LIPID VESICLE AND OTHER VESICLE FORMS OF DIMYRISTOYLPHOSPHATIDYLCHOLINE. J-P. Dufour, R. Nunnally, L. Buhle, and T.Y. Tsong (Department of Radiology, University of Texas Health Science Center, Dallas, TX 75235) *Biochemistry 20*(19):5576-5586 (1981). Several known forms of bilayer vesicles of dimyristoylphosphatidylcholine exhibit the gel to liquid-crystalline phase transition in the temperature range convenient for membrane enzyme reconstitution studies. This warrants a systematic investigation of their physical characteristics and their phase transition behaviors. We have employed electron microscopy, gel chromatography, ^{31}P nuclear magnetic resonance, differential scanning micro-calorimetry, and fluorescence spectroscopy to determine several physical parameters of the limiting size microvesicle, the larger vesicle form of Enoch and Strittmatter, the multilamellar vesicle, and an ATPase-trigger-fused macrovesicle. This latter vesicle form was produced by a spontaneous fusion of the complex of the plasma membrane ATPase of *Schizosaccharomyces pombe* and the lipid microvesicles at a low ratio of enzyme to vesicle concentrations, and at a low temperature. The ATPase-trigger-fused vesicle are unilamellar and have an intact ionic permeation barrier at 30 C and a gel to liquid-crystalline transition temperature at 24.4 C with a transition heat of 5.64 kcal/mol. Thus, this vesicle form should be a valuable tool for studying possible proton-pumping activity of this ATPase. In contrast to data found in the literature, which show lack of the pretransition around 15 C for all the vesicle forms examined. Moreover, the transition widths of unilamellar vesicles are much broader than those of the multilamellar vesicles, suggesting that in the latter system interlayer interactions may contribute to the cooperativity of the transition.

EFFECTS OF DIETARY CHOLESTEROL ON ANTIBODY-DEPENDENT PHAGOCYTOSIS AND CELL-MEDIATED LYSIS IN GUINEA PIGS. A.K. Duwe, M. Fitch, and R. Ostwald (Dept. of Nutr. Sciences, Univ. of California, Berkeley, CA 94720) *J. Nutr. 111*(9):1672-1680 (1981). The effect of dietary cholesterol on antibody-dependent phagocytosis and cell-mediated cytotoxicity (ADCC) by peritoneal cells and on the susceptibility to lysis of erythrocytes was studied in the guinea pig. We found that peritoneal cells from cholesterol-fed animals (CHOL PEC) demonstrated a decreased ability to both phagocytose and lyse antibody-coated (Ab) guinea pig erythrocytes than did those from control guinea pigs (CONT PEC). This decrease was equal in groups fed cholesterol for 5½-13 weeks, preanemic or anemic, and with normal or enlarged spleens. Dose response curves varying Ab concentration showed that CHOL PEC required higher concentrations of Ab to effect phagocytosis and lysis than did CONT PEC. Dietary cholesterol, while rapidly inducing morphological changes such as spurring in guinea pig erythcytosis in this assay system. These findings suggest that the increased incidence of infection in cholesterol-fed guinea pigs may be due to impaired phagocytic function and that the anemia observed in guinea pigs after 8-10 weeks of feeding cholesterol is not due to increased antibody-dependent removal of spurred erythrocytes by the phagocytic system.

EFFECTS OF DIETARY NUTRIENTS ON INTESTINAL TAUROCHOLIC ACID ABSORPTION. J.D. Fondacaro and R.H. Wolcott (Dept. of Physiology, Univ. of Cincinnati College of Medicine, Cincinnati, Ohio 45627) Proc. *Soc. Exp. Biol. Med. 168*(2):276-281 (1981). In order to assess the intraluminal event that may be responsible for bile acid malabsorption in cystic fibrosis, taurocholic acid absorption was determined in the presence and absence of representative unhydrolyzed dietary nutrients in animal models. Triglyceride (corn oil) significantly reduced taurocholic acid uptake by villi isolated from hamster ileum. Likewise, when combinations of nutrients were were studied, only those combinations of nutrients containing triglyceride inhibited taurocholic acid absorption. Neither starch nor albumin or a combination of these two substrates altered this process. Triglyceride also produced significant reductions in taurocholic acid absorption in perfused segments of terminal ileum of rats as determined by reduced biliary recovery of absorbed bile acid. Again, starch and albumin had no effect *in vivo*. These findings support an "intraluminal theory" of bile acid malabsorption in cystic fibrosis limited to only the adverse influence of unhydrolyzed lipid on this normal physiological process.

EFFECT OF FEEDING A LOW PROTEIN DIET DURING NEONATAL LIFE ON SUBSEQUENT CHOLESTEROL AND BILE ACID METABOLISM IN ADULT GUINEA PIGS. A.S. Hassan, L.S. Gallon, R.L. Yunker, and M.T.R. Subbiah (Departments of Pathology and Medicine, University of Cincinnati Medical Center, Cincinnati, OH 45267) *J. Nutr. 111*(11):2024-2029 (1981). The effect of feeding a low-protein (LP) diet during neonatal life of guinea pigs on subsequent cholesterol and bile acid metabolism when the animals were being fed a) stock diet and b) 0.25% cholesterol-containing diet, was investigated. Feeding a 10% protein diet caused a significant ($P < 0.05$) increase in the fecal excretion of neutral sterols and bile acids without any change in plasma cholesterol. The LP-fed guinea pigs continued to excrete significantly ($P < 0.05$) greater amounts of neutral sterols and bile acids even after they had been switched to stock diet for several weeks. The pool sizes of lithocholic and chenodeoxycholic acids were lower in the LP group during the stock diet period. Upon challenge with 0.25% cholesterol diet, no significant differences between the two groups, regarding the above-mentioned parameters, were noted. The data suggest that neonatal exposure to a low-protein diet can affect sterol and bile acid metabolism such that the effect persists even when the animals have been switched to stock diet for several weeks.

VITAMIN D AND ITS METABOLITES IN HUMAN AND BOVINE MILK. B.W. Hollis, B.A. Roos, H.H. Draper, and P.W. Lambert (Endocrinology and Mineral Metabolism, VA Medical Center and School of Med., Case Western Reserve Univ., Cleveland, OH 44106) *N. Nutr. 111*(7):1240-1248 (1981). Human and bovine milk were analyzed for vitamin D, 25-hydroxyvitamin D, 24,25-dihydroxyvitamin D, 25,26-dihydroxyvitamin D and 1,25-dehydroxyvitamin D using exhaustive chromatographic purification procedures coupled with ligand binding assays. Human milk contained the following amounts of antirachitic sterols) pg/ml, mean ± SD, n = 5): 39 ± 9 vitamin D; 311 ± 31 25-hydroxyvitamin D; 52 ± 8 24,25-dihydroxyvitamin D; 32 ± 9 25,26-dihydroxyvitamin D; 5.1 ± .03 1,25-dihydroxy-vtamin D. Normal bovine milk contained levels of these sterols comparable to those found in human milk. Increasing the oral dose of vitamin D to the cows was reflected by an increase of the parent vitamin and 25-hydroxyvitamin D in the milk. Vitamin D-binding protein concentration in human milk whey, determined by Ouchterlony immunodiffusion and radioimmunoassay, was 1-2% of the levels observed in the plasma and was dependent on the stage of lactation. Vitamin D and its metabolite were shown initially to be present in the whey portion but with time migrated into the fat portion of milk. The antirachitic sterols detected account for approximately 25 IU/liter and 25 IU/liter of antirachitic activity in human and bovine milk, respectively. In both species 25-hydroxyvitamin D comprised the majority of the antirachitic sterols detected in normal milk.

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