Abstracts

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Biochemistry and nutrition

EXPERIMENTAL NEPHROTIC SYNDROME INDUCED IN THE RAT BY PUROMYCIN AMINONUCLEOSIDE: HEPATIC SYNTHESIS OF NEUTRAL LIPIDS AND PHOSPHOLIPIDS FROM ^{3}H-WATER AND ^{3}-H-PALMITATE. E. Gherardi and S. Calandra (Istituto di Patologia Generale, Universita degli Studi di Modena Via Campi 287, 41100 Modena, Italy) *Lipids 15*(2), 108-12 (1980). Experimental nephrotic syndrome (ascites, proteinuria, hypoalbuminemia, and hyperlipidemia) was induced in male Wistar rats by seven daily subcutaneous injections of puromycin aminonucleoside (20 mg/kg). Hepatic lipogenesis from ^{3}H-water and ^{3}H-palmitate was investigated in nephrotic and pair fed control rats by using liver slices. The difference in hepatic lipogenesis between nephrotic and control rats was even more pronounced if the data were corrected for the total liver weight which was significantly increased in the nephrotic rats (11.3 ±.3 vs. 8.5 ±.1 g, $p < .001$). These findings indicate that the synthesis of neutral lipids from both ^{3}H-water and ^{3}H-palmitate is elevated in rat with aminonucleoside-induced nephrotic syndrome. The possible role of the increased hepatic lipogenesis in the pathogenesis of the nephrotic hyperlipidemia is discussed.

REGULATION OF CHOLESTEROL BIOSYNTHESIS IN CULTURED CELLS BY PROBABLE NATURAL PRECURSOR STEROLS. G.F. Gibbons, C.R. Pullinger, H.W. Chen, W.K. Cavenee and A.A. Kandutsch (Med. Res. Council, Lipid Metabolism Unit, Hammersmith Hosp., London, W12 OHS, United Kingdom) *J. Biol. Chem. 255*(2), 395-400 (1980). Lanosterol derivatives bearing an additional oxygen function at carbon 32 are generally considered to be natural cholesterol precursors. Two such oxysterols, 5α-lanost-8-ene-3β,32-diol and 3β-hydroxy-5α-lanost-8-en-32-al, strongly inhibited the incorporation of [1-^{14}C] acetate into sterols in cultures of Chinese hamster lung cells, mouse L-cells, and fetal mouse liver cells. In the latter two cell types, this inhibition could be accounted for predominantly by a decrease in the activity of the enzyme hydroxymethylglutaryl-CoA reductase (NADPH) (EC 1.1.1.34). In the lung cells, lanosterol 14α-demethylation was also suppressed. The oxygenated lanosterols had no effect on reductase activity in a mutant line of Chinese hamster lung cells selected for resistance to 25-hydroxycholesterol (cholest-5-ene-3β,25-diol). In these cases, only lanosterol 14α-demethylation was inhibited. All these effects were also observed with the Δ^7 bond isomers of the above compounds. Studies of the metabolism of the 32-oxygenated lanostenols in L-cells and mouse fetal liver cells revealed that, while all were converted into C_{27} sterols (including cholesterol), to a varying degree, each was more extensively metabolized to both polar and nonpolar products. Similar products also arose during the metabolism of each of the C-15 epimers of 5α-lanost-8-ene-3β, 15-diol and of their respective Δ^7 bond isomers.

INTERACTION OF HUMAN PLASMA HIGH DENSITY LIPOPROTEIN HDL_2 WITH SYNTHETIC SATURATED PHOSPHATIDYLCHOLINES. E.L. Gong and A.V. Nichols (Donner Lab., Lawrence Berkeley Lab., Univ. of Calif., Berkeley, CA 94720) *Lipids 15*(2), 86-90 (1980). The interaction of human plasma high density lipoprotein HDL_2 (d 1.063-1.125 g/ml) with sonicated dispersions of synthetic saturated phosphatidylcholines, dipalmitoyl- ($diC_{16}PC$), dimyristoyl- ($diC_{14}PC$), didodecanoyl- ($diC_{12}PC$), didecanoyl- ($diC_{10}PC$), and dioctanoyl- (diC_8PC) L-alpha phosphatidylcholine, was investigated. Incubation (4.5 hr, 37 C) of HDL_2 with $diC_{14}PC$, $diC_{12}PC$, $diC_{10}PC$ and diC_8PC followed by gradient gel electrophoresis or preparative ultracentrifugation resulted in a redistribution of apolipoprotein A-I (apoA-I). The extent of redistribution depended on the molar ratio of the phospholipid to HDL_2 in the incubation mixture. Redistributed apoA-I occurred as lipid-free apoA-I and/or as complexes of apoA-I with phosphatidylcholine. Increasing the length of time of ultracentrifugation of the interaction mixtures did not increase the extent of redistribution. No redistribution of apoA-I was detected following incubation and gradient gel electrophoresis or preparative ultracentrifugation of mixtures of HDL_2 with dispersions of $diC_{16}PC$.

BIOSYNTHESIS OF CHOLIC ACID IN RAT LIVER: FORMATION OF CHOLIC ACID FROM 3α, 7α, 12α-TRIHYDROXY- AND 3α, 7α, 12α, 24-TETRAHYDROXY-5β-CHOLESTANOIC ACIDS. J. Gustafsson (Dept. of Chem., Karolinska Institutet, Stockholm, Sweden) *Lipids 15*(2), 113-21 (1980). Conversion of 3α7α12α-trihydroxy-5β-cholestanoic acid into 3α,7α,12α24-tetrahydroxy5β-cholestanoic and cholic acids was catalyzed either by the mitochondrial fraction fortified with coenzyme A, ATP, $MgCl_2$ and NAD or by the combination of microsomal fraction and 100,000 x g supernatant fluid fortified with coenzyme A, ATP and NAD. 24-Hydroxylation and formation of cholic acid occurred at similar rates with the 25R- and the 25S-forms of 3α,7α,12α-trihydroxy-5β-cholestanoic acid. The 25R- and 25S-forms of 3α,7α,12α-trihydroxy- and 3α,7α,12α,24-tetrahydroxy-5β-cholestanoic acids were administered to bile fistula rats. Labeled cholic acid was isolated from the bile. The initial specific radioactivity of cholic acid was higher and the disappearance of radioactivity more rapid after administration of 3α,7α,12α-trihydroxy-5β-cholestanoic acid than of 3α,7α,12α,24-tetrahydroxy-5β-cholestanoic acid. The findings are discussed in relation to the assumed pathway for side chain cleavage in cholic acid biosynthesis.

THE EFFECTS OF DIETARY PHOSPHORUS, VITAMIN D_3, AND 25-HYDROXY VITAMIN D_3 LEVELS ON FEED INTAKE, PRODUCTIVE PERFORMANCE, AND EGG AND SHELL QUALITY IN TWO STRAINS OF FORCE-MOLTED WHITE LEGHORNS. R.M.G. Hamilton (Animal Res. Inst., Res. Branch, Agri. Canada, Central Experimental Farm, Ottawa, Canada) *Poult. Sci. 59*(3), 598-604 (1980). A total of 576 force-molted hens were used to investigate the effect of dietary phosphorus level, and the source and level of vitamin D_3 on egg production and shell quality. The experiment was a 2x2x2x3 factorial design with two strains of hens, two levels of available phosphorus (.34 and .60%), and two sources of D_3 (25-hydroxy vitamin D_3 and vitamin D_3) at three levels (8, 16, 24 μg/kg); the diets were provided *ad libitum* for 84 days (from 646 to 730 days of age). Egg and shell quality measurements were taken on eggs laid over a 4-day period when the hens were 727 days of age. Dietary phosphorus level, and D_3 source and level had no significant ($P > .05$) effect on 730-day body weight, feed intake and efficiency, egg production and yield, shell weight, percent shell, shell weight per unit surface area, Haugh units, blood spots, egg specific gravity, nondestructive deformation, compression fracture force, and shell thickness. There were significant differences among strains for the aforementioned variables except feed intake, egg weight, Haugh units, and blood spot incidence. Few interactions were found between the main effects. Results indicate that no improvement in shell quality of eggs from force-molted hens was obtained by decreasing the level of phosphorus or substituting 25-hydroxy vitamin D_3 for vitamin D_3 in the diet.

STEREOCHEMISTRY OF THE SIDE CHAIN OXIDATION OF 5β CHOLESTANE-3α,7α,12α-TRIOL IN MAN. R.F. Hanson, P. Szczepanik-VanLeeuwen, and G.C. Williams (Dept. of Internal Med., Univ. of Minn., Minneapolis, MN 55455) *J. Biol. Chem. 255*(4), 1483-5 (1980). Previous *in vitro* studies have shown that the oxidation of the side chain of bile acid precursors can start with either microsomal or mitochondrial enzyme systems. The microsomal system oxidizes the terminal methyl group (C-26) of the side chain that originates from C-2 of mevalonic acid, and the mitochondrial system oxidizes the terminal methyl group (C-27) derived from C'-3 of mevalonic acid. We administered [2-^{14}C] mevalonic acid to a patient with a complete bile fistula and isolated from the bile 3α,7α,12α-trihydroxy[1,7,15,22,26-^{14}C] 5β-cholestanoic acid, a precursor in the synthesis of cholic acid which has undergone partial oxidation of the side chain. This study suggests that the major pathway of side chain oxidation of 5β-cholestane-3α,7α,12α-triol starts with hydroxylation of the methyl group derived from C'-3 of mevalonate (C-27) by mitochondrial enzymes. It is also concluded that the 3α,7α,12α-trihydroxy-5β-cholestan-27-oic acid formed in man has the R configuration at carbon 25.

HYDROXYLATIONS IN BIOSYNTHESIS AND METABOLISM OF BILE ACIDS. CATALYTIC PROPERTIES OF DIFFERENT FORMS OF CYTOCHROME P-450. R. Hansson and D. Wikvall (Dept. of Pharmaceutical Biochem., Univ. of Uppsala, Biomed. Center, Box 578, S-751 23 Uppsala, Sweden) *J. Biol. Chem. 255*(4), 1643-9 (1980). Three fractions of cytochrome P-450 were prepared from liver microsomes of phenobarbital-treated rabbits. The fractions appeared homogeneous on sodium dodecyl sulfate-polyacrylamide gel electrophoresis and contained 11 to 19 nmol of cytochrome P-450/mg of protein. Two of the fractions were apparently identical with cytochromes P-450 LM_2 and LM_4. Two fractions of cytochrome P-450 were purified from liver microsomes of phenobarbital-treated rats. The fractions were apparently homogeneous on sodium dodecyl sulfate-polyacrylamide gel electrophoresis and contained 14 to 18 nmol of cytochrome P-450/mg of protein. Two fractions of cytochrome P-450 were purified from liver microsomes of cholestyramine-treated rats. The fractions showed one major band on sodium dodecyl sulfate-polyacrylamide gel electrophoresis and contained 14 to 15 nmol of cytochrome P-450/mg of protein.

FATTY ACID SYNTHETASES FROM *EUGLENA GRACILIS*. SEPARATION OF COMPONENT ACTIVITIES OF THE ACP-DEPENDENT FATTY ACID SYNTHETASE AND PARTIAL PURIFICATION OF THE β-KETOACYL-ACP SYNTHETASE. R.W. Hendren and K. Bloch (James Bryant Conant Laboratories, Harvard Univ., Cambridge, Mass. 02138) *J. Biol. Chem. 255*(4), 1504-8 (1980). The component enzymes of the chloroplast-associated, acyl carrier protein (ACP)-dependent, fatty acid synthetase (FAS-II) from *Euglena gracilis* have been independently examined by gel filtration chromatography of crude extracts from photoautotrophic cells. Under certain conditions, several of the *E. gracilis* FAS-II component activities may aggregate noncovalently to form a weak complex. A partial purification of the β-ketoacyl-ACP synthetase by ammonium sulfate fractionation, DEAE-cellulose chromatography, and hydroxylapatite chromatography resulted in its complete separation from the enoyl-ACP reductase activity. When the separated β-ketoacyl-ACP synthetase and enoyl-ACP reductase activities were recombined and subjected to gel filtration chromatography, the two activities migrated distinctly and with lower apparent molecular weights, 118,000 and 56,500, respectively, than when similarly measured in the crude extract.

LIVER AND SERUM LIPIDS AND LIPOPROTEINS OF RATS FED 5% L-LYSINE. P. Hevia and W.J. Visek (Schools of Basic Med. Sci. and Clin. Med., Univ. of Illinois, Urbana, IL 61801) *Lipids 15*(2), 95-9 (1980). Soybean protein and casein supplemented with 1% Arginine were compared for their ability to prevent fatty livers caused by excess dietary Lysine. The concentrations of serum lipids and lipoproteins of rats fed 5% Lysine and having fatty livers were also compared with those of rats fed the identical diet but lacking fatty livers when killed. Overall, excessive dietary Lysine caused fatty livers which were prevented by varying the diet or length of feeding. Excess Lysine feeding altered lipoprotein metabolism shown by decreased serum HDL and a substantial elevation in LDL. The latter was more apparent when the fat accumulation in liver was less severe or absent. The data suggest that the fatty liver from Lysine excess is probably unrelated to increased fat mobilization from storage, decreased fat oxidation or to a major block in the transport of triglycerides from the liver to the circulation.

DECREASED FORMATION OF PROSTAGLANDINS DERIVED FROM ARACHIDONIC ACID BY DIETARY LINOLENATE IN RATS. D.H. Hwang and A.E. Carroll (Human Nutr. and Food, School of Home Economics, Louisiana State Univ., Baton Rouge, LA 70803) *Am. J. Clin. Nutr. 33*(3), 590-7 (1980). Accumulated evidence now suggests that availability of precursor acid is an important factor controlling the biosynthesis of prostaglandins (PG's). Since linolenic acid inhibits the conversion of linoleic acid to arachidonic acid (PGE_2, $PGF_2\alpha$, and thromboxane A_2 precursor), rats receiving more linolenic acid are expected to have less arachidonic acid and thus less PG's synthesized from arachidonic acid than those receiving linoleic acid alone. Essential fatty acid-deficient rats, induced by feeding hydrogenated coconut oil diet for 15 weeks, were divided into six groups and fed graded amounts of purified methyl linolenate for 6 weeks. Each group of rats showed that the level of arachidonic acid in serum lipids and serum concentrations of PG's synthesized from arachidonic acid by platelets decreased as the amount of dietary linolenate increased. This indicated that biosynthesis of PG's in platelets can be influenced by the availability of precursors, and thus it can be modified by the manipulation of dietary fatty acids.

MECHANISM OF ACTION OF MILK LIPOPROTEIN LIPASE AT SUBSTRATE INTERFACES: EFFECTS OF APOLIPOPROTEINS. R.L. Jackson, F. Pattus and G. de Haas (Dept. of Pharmacology and Cell Biophysics, Biochem. and Med., Univ. of Cincinnati Coll. of Med., Cincinnati, OH 45267) *Biochemistry 19*(2), 373-8 (1980). The mechanism of action of bovine milk lipoprotein lipase was studied by using a monomolecular film of 1,2-didecanoylglycerol. The apparent rate of hydrolysis of diglyceride increased with increasing surface pressures above 12 mN/m; the enzyme was inactive at pressures less than 12 mN/m. We have measured the effects of four plasma apolipoporteins (apoC-II, apoC-III, apoA-I, and apoE), bovine serum albumin, porcine pancreatic colipase, heparin, and NaCl on the kinetics of lipid hydrolysis. At a surface pressure of 15 mN/m, all of the proteins, with the exception of colipase, gave increased enzyme activity compared to lipase alone; apoC-II gave maximal activation. At 25 mN/m, apoC-II at concentrations of less than 0.25 μ g/mL showed a specific activation, whereas the other proteins had no effect. Heparin activated at both high and low surface pressures; NaCl had little or no effect in this system. At a higher concentration of apoC-II (0.50 μ g/mL), the apoprotein inhibited the enzyme. The addition of apoC-III, apoA-I, or apoE (final concentration 0.25 μ g/mL), but not albumin or colipase, to apoC-II (0.25 μ g/mL) caused an increase in surface pressure of 5-6 mN/m and an apparent rate which was less than half that found for lipase alone, suggesting that all of the apoproteins inhibit the apoC-II specific activation.

EFFECT OF SUCROSE POLYESTER ON FECAL STEROID EXCRETION BY 24 NORMAL MEN. R.J. Jandacek, F.H. Mattson, S. McNeely, L. Gallon, R. Yunker and C.J. Glueck (Miami Valley Lab., Procter and Gamble Co., Cincinnati, OH 45247) *Am. J. Clin. Nutr. 33*(2), 251-9 (1980). The effect of the nonabsorbable fat-like substance, sucrose polyester (SPE), on neutral steroid excretion was determined in 24 healthy men. Initially the subjects received for 10 days a basal diet that was high (800 mg/day) or low (300 mg/day) in cholesterol or for 21 days a basal diet containing $<$ 50 mg cholesterol per day. These diets were isocaloric. Over three subsequent, consecutive periods of 10 days each, 8, 16, or 25 g/day of liquid SPE or 19, 38, or 62 g/day of an 80/20 mixture of SPE and completely hydrogenated palm oil was added to the diet. The amounts of C27 steroids, i.e., cholesterol and its conversion products formed by intestinal bacteria, were measured in the feces of each subject. In each individual, the intestinal bacteria converted β-sitosterol to its characteristic products in the same proportion as was cholesterol. The addition of SPE to the diet caused a lesser amount of the sterols to be modified by the bacteria. This could be measured more readily in the converter population. There, each gram of SPE that was ingested resulted in 1.1% decrease in conversion for those who received liquid SPE and a 0.6% decrease for those who received the SPE-hydrogenated palm oil mix. The decrease in conversion is a probable consequence of the sterols being dissolved in the oil phase of SPE in the lumen of the intestinal tract and hence unavailable to the bacteria.

PROTEIN-LIPID INTERACTIONS. STUDIES OF THE M13 COAT PROTEIN IN DIMYRISTOYLPHOSPHATIDYLCHOLINE VESICLES USING PARINARIC ACID. D. Kimelman, E.S. Tecoma, P.K. Wolber, B.S. Hudson, W.T. Wickner and R.D. Simoni (Dept. of Biological Sciences and the Dept. of Chem., Stanford Univ., Stanford, CA 94305) *Biochemistry 18*(26), 5874-80 (1979). Addition of the M13 virus coat protein to dimyristoylphosphatidylcholine vesicles decreases the amplitude of the lipid bilayer phase transition observed with parinaric acid fluorescence intensity. According to these measurements, the solid-phase bilayer is disordered by the protein, while the fluid phase is not appreciably affected. The results are consistent with a perturbation of the bilayer by the coat protein at low temperature. The structure of the bilayer near the protein results in a spatial distribution of *trans*-parinaric acid such that this quencher avoids the donor protein. The intensity and partitioning/quenching behavior used to characterize the effect of the protein on the solid bilayer do not reveal any bilayer structural changes for the fluid-phase bilayer. However, fluorescence polarization measurements with parinaric acid reveal a marked decrease in the rotational mobility of the probe relative to that of the fluid bilayer. The utility of parinaric acid fluorescence methods in the study of membrane-bound proteins is emphasized.

THE EFFECT OF ISOMERIC *TRANS*- 18:1 ACIDS ON THE DESATURATION OF PALMITIC, LINOLEIC AND EICOSA-8,11,14-TRIENOIC ACIDS BY RAT LIVER MICROSOMES. M.M. Mahfouz, S. Johnson and R.T. Holman (The Hormel Inst., Univ. of Minn., Austin, MN 55912) *Lipids 15*(2), 100-7 (1980). The inhibitory effects of the positional isomers of *trans*- 18:1 acids on the desaturation of palmitic acid to palmitoleic (Δ9-desaturase), linoleic to γ-linolenic(Δ6-desaturase) and eicosa-8,11,14-trienoic to arachidonic acid (Δ5-desaturase) were investigated. These *trans*-18:1 acids were found to be inhibitory for the microsomal Δ6-, Δ9- and Δ5-desaturases of rat liver. The position of the double bond in the *trans*-18:1 acids seems to be important in determining the degree of inhibition. At inhibitor/substrate ratio of 3:1, the Δ6-desaturase was most strongly inhibited by *trans*-Δ3, -Δ4, -Δ7 and -Δ15-18:1 isomers, whereas the Δ9-desaturase was most strongly inhibited by

trans-Δ3, -Δ5, -Δ7, -Δ10, -Δ12, -Δ13 and -Δ16 isomers. At inhibitor/substrate ratio of 6:1, the Δ5-desaturase was most strongly inhibited by Δ3-, Δ9-, Δ13- and Δ15-isomers. When 18:0 was added to the incubations of 16:0, 18:2 and 20:3 at the same I/S ratios used for the *trans*-18:1 acids, weak inhibition for Δ9-desaturase and no inhibition for Δ5- and Δ6-desaturases was observed.

IMMUNOCHEMISTRY OF HUMAN VERY LOW DENSITY LIPOPROTEINS: APOLIPOPROTEIN C-III. S.J.T. Mao, P.K. Bhatnagar, A.M. Gotto, Jr., and J.T. Sparrow (Dept. of Med., Baylor College of Med. and The Methodist Hosp., Houston, TX 77030) *Biochemistry 19*(2), 315-20 (1980). Apolipoprotein C-III (apoC-III) is a major protein constituent of human plasma very low density lipoproteins (VLDL) and a minor constituent of high density lipoproteins (HDL). The apoprotein is a singly polypeptide chain of 79 amino acids and occurs in several forms differing only in their content of sialic acid. In the present report a quantitative radioimmunoassay (RIA) has been developed in order to study the immunochemical properties of apoC-III. Two individual rabbit antisera were used. Since the conformation of the apoprotein has drastically changed upon the addition of dimyristoylphosphatidylcholine (DMPC), this finding indicates that the gross conformational change of apoC-III_1 does not affect the immunochemical properties and that the antigenic reactive sites are probably located at the surface in apoC-III_1-DMPC complexes. The immunoreactivity of apoC-III was also found to be approximately the same in HDL or VLDL as that of the delipidated apoHDL or apoVLDL. Thus, the antigenic sites of apoC-III must be fully exposed on the surface of the lipoproteins.

STUDIES ON THE POLYMORPHISM OF HUMAN APOLIPOPROTEIN A-I. A.C. Nestruck, G. Suzue and Y.L. Marcel (Institut de Recherches Cliniques de Montréal, 110, Avenue des Pins ouest, Montréal H2W 1R7, Québec, Canada) *Biochim. Biophys. Acta 617*(1), 110-21 (1980). Upon preparative isoelectric focussing of human apo-HDL, four major forms of apolipoprotein A-I have been isolated. These forms of apolipoprotein A-I were shown to have identical migration on polyacrylamide gel electrophoresis, molecular weights of 26 000 on sodium dodecyl sulfate gel electrophoresis and a common antigenicity with antisera against apolipoprotein A-I or $A\text{-}I_1$. Each form had very similar amino acid compositions with the exception of form apolipoprotein $A\text{-}I_4$. All forms but apolipoprotein $A\text{-}I_4$ were activators of lecithin:cholesterol acyltransferase, the latter was inhibitory to the reaction. From these results, it was concluded that apolipoprotein $A\text{-}I_1$, $A\text{-}I_2$ and $A\text{-}I_3$ are equivalent forms of apolipoprotein A-I whereas apolipoprotein $A\text{-}I_4$ is different or heterogeneous. Upon refocussing, the polymorphs were shown to be stable at their p*I* and not affected by changes in concentration and by the presence of urea or ampholytes. Exposure of a form of apolipoprotein A-I to alkaline pH partially regenerated the original heterogeneity; however, apolipoprotein $A\text{-}I_4$ regenerated from apolipoprotein $A\text{-}I_1$ did not contain isoleucine, which further demonstrates form apolipoprotein $A\text{-}I_4$ heterogeneity.

PUBLICATIONS ABSTRACTED

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