17B (r = 0.33); estrone and calcium (r = 0.40); estrone and plasma free fatty ocid (r = 0.40) and calcium and plasma free fatty acids (r = 0.45). Correlation between corticosterone and free fatty acids (r = 0.20) was also significant ($P < 0.05$).

THE ROLE OF CYTOCHROME P-450 IN CHOLESTEROL BIOGENESIS AND CATABOLISM. Sandra D. Atkin, Eileen D. Palmer, P.D. English, B. Morgan, M.A. Cawthorne and J. Green (Beecham Res. Labs., Nutr. Res. Centre, Walton Oaks, Tadworth, Surrey, U.K.). *Biochem. J.* 128, 237–42 (1972). Adjuvant-induced arthritis in rats is accompanied by a loss of activity of the drug-metabolizing enzyme system and a decrease in hepatic cytochrome P-450. Arthritic rats have normal serum and liver cholesterol concentrations. The rate of biogenesis of cholesterol in vivo and in vitro from either [^{14}C]acetate or [^{14}C]mevalonate in arthritic rats was the same as or greater than that found in control rats. Treatment of rats with carbon disulphide (1 ml/kg) resulted in a loss of drug-metabolizing-enzyme activity and increased cholesterol biogenesis. The activity of cholesterol 7α-hydroxylase in adjuvant-induced arthritic rats did not differ significantly from that in control rats. Rats fed with cholestyramine had an elevated hepatic cholesterol 7α-hydroxylase activity, but neither the concentration of cytochrome P-450 nor the activity of the drug-hydroxylating enzyme, aminopyrine demethylase, was affected. The relationships between drug hydroxylation and cholesterol metabolism are discussed.

BINDING OF IRON AND COPPER TO BOVINE HEART MITOCHONDRIA. III. ROLE OF MITOCHONDRIAL PHOSPHOLIPIDS AND THIOLS. A.I. Cederbaum and W.W. Wainio (Dept. of Biochem., Rutgers College, Rutgers—State Univ. of N.J., New Brunswick, N.J. 08903). *J. Biol. Chem.* 247, 4615–20 (1972). Extraction of phospholipids with 10% H_2O-90% acetone did not affect Cu^{2+} binding by bovine heart mitochondria when the external concentration of added Cu^{2+} was 0.5 to 1 mM. At 2.5 mM Cu^{2+}, there was a decrease in Cu^{2+} binding upon removal of the phospholipids. Readdition of phospholipids restored Cu^{2+} binding. Local anesthetics which interact with phospholipids had an inhibitory affect on Cu^{2+} binding only at high Cu^{2+} concentrations. Therefore, Cu^{2+} binding does not apparently involve phospholipids except at high Cu^{2+} concentrations. Disulfide-generating agents, external thiols and *p*-chloromercuribenzoate inhibited Cu^{2+} binding. A $Cu^{2+} + e^- \leftrightarrows Cu^+$ transformation probably occurs during Cu^{2+} binding. It is concluded that copper may be bound to the same proteins that bind Zn^{2+}. Fe^{3+} binding was not affected by the removal of the phospholipids nor by the presence of the local anesthetic procaine-HCl. Mitochondrial thiols did not seem to be involved in the binding to intact mitochondria. Fe^{3+} and copper appear to bind to different loci on mitochondrial proteins. Maximal binding of Fe^{2+} required the presence of cardiolipin in the mitochondria. Removal of phosphatidylcholine and phosphatidylethanolamine with 10% H_2O-90% acetone, or interaction of phospholipids with local anesthetics caused an increase in Fe^{2+} binding. Thiols were shown to play a role in Fe^{2+} binding.

FETAL RAT LUNG DEVELOPMENT: LIPIDS AND SURFACE TENSION PROPERTIES AFTER DECAPITATION IN UTERO. W.R. Blackburn, D.M. Potter, H. Travers, L.L. Gassenheimer and R.A. Rhoades (Dept. of Pathol. and Lab. for Human Performance Res. and Center for Air Environment Studies, Penn. State Univ., Hershey, Pa. 17033). *Proc. Soc. Exp. Biol. Med.* 140, 885–9 (1972). Decapitation in utero produces hormonal deficiencies which do not influence lung organogenesis but retards pneumocyte differentiation at the organelle and molecular levels. Such lungs contain decreased quantities of phospholipid and functionally are impaired in their ability to reduce surface tension to normal levels. The mechanisms by which these alterations are produced are most likely related to hormonal deficiencies during fetal development. These deficiencies may specifically alter lung phospholipid metabolism. Retardation of phospholipid metabolism may, however, be secondary to a more generalized retardation of pneumocyte differentiation. We favor the latter hypothesis in so far as pituitary failure occurring in man and animals with fully mature lungs does not lead to clinical respiratory distress. Further inquiry into the manner in which hormones influence lung metabolism will require evaluation of the effect of specific hormonal replacements on in utero decapitated or hypophysectomized fetuses and on lung explants differentiating in vitro.

A COMPARISON OF MAGNESIUM DEFICIENCY, COLD ACCLIMATION AND THYROXINE ADMINISTRATION ON MITOCHONDRIAL FATTY ACID COMPOSITION. W.S. Platner, B.C. Patnayak and R.R.J. Chaffee (Dept. of Physiol., Univ. of Mo., Schl. of Med., Columbia, Mo. 65201). *Proc. Soc. Exp. Biol. Med.* 140, 857–61 (1972). A study was made to compare the effects of dietary magnesium deficiency, cold acclimation and thyroxine administration on liver mitochondrial fatty acid composition. Groups of singly caged rats were fed synthetic diets containing either a control level (61 mg%) or a low level (4 mg%) of magnesium, and of these some were kept in the cold (5 ± 2C) and others at normal colony room temperature (23 ± 2C). A selected number of these rats were made hyperthyroid by ip injection of 2 mg/100 g body weight of L-thyroxine sodium daily for 8 days before sacrifice. The fatty acid composition of the mitochondrial lipids was determined by gas chromatography. Cold acclimation alone significantly decreased the percentages of palmitic and arachidonic acids and increased the percentages of myristoleic, stearic, and linoleic acids. Thyroxine treatment increased the percentage of stearic acid and decreased linoleic and arachidonic acids. Magnesium deficiency, on the other hand, had little effect on the major components of the mitochondrial fatty acids and no effect on the total unsaturated fatty acids. It is concluded that mitochondrial swelling and uncoupling produced by magnesium deficiency does not seem to be related to changes in the total unsaturation of mitochondrial fatty acids.

EFFECTS OF THIAMINE DEFICIENCY AND OCTANOATE ADMINISTRATION, IN VIVO, ON GLUCONEOGENESIS IN RAT KIDNEY SLICES AND ON AMINO ACID PROFILE IN RAT LIVER. R.J. Paquet, C.R. Mackerer and M.A. Mehlman (Dept. of Biochem., Univ. of Nebraska, College of Med., Omaha, Neb. 68105). *Proc. Soc. Exp. Biol. Med.* 140, 934–6 (1972). The effects of a single dose of octanoate (3.7 mmoles/kg, ip) on gluconeogenesis in kidney slices and on amino acid profile in liver were determined in thiamine-deficient and pair-fed control rats. Kidney slices from the deficient rats produced glucose, from lactate or pyruvate, at greatly reduced rates. Octanoate, in vivo, increased the rates of glucose synthesis in slices from both control and deficient rats. Thiamine deficiency caused increased levels of threonine and decreased levels of aspartate but glutamate, glutamine, serine, glycine and alanine were unchanged. Octanoate administration, in vivo, to both control and diabetic rats, reduced the concentrations of all liver amino acids, except glycine. It was concluded that thiamine-deficiency inhibited gluconeogenesis indirectly, possibly via a primary lesion at pyruvate dehydrogenase, but that the inhibition could be circumvented by providing a fatty acid (e.g. octanoate).

OBSERVATIONS ON HYPERVITAMINOSIS *A* AND HYDROPERICARDIUM IN CHICKS. L.W. McCuaig, H.C. Carlson and I. Motzok (Univ. of Guelph, Guelph, Ontario, Canada). *Poultry Sci.* 51, 1206–10 (1972). Male chicks developed severe hydropericardium when fed a 20% tallow diet for the first four weeks of life followed by a tallow-free ration containing 3,250,000