sponding spots, eluting the II with EtOH and detg. colorimetrically. The extn. yield was ~80%. Urinary tests showed no II in clin. healthy personnel, while among the staff of I-processing plants, 80% of the sampled persons gave a pos. reaction to the method. Extensive tests showed that most of the pos. reactions were obtained with staff employed 2–5 years in the plants. In these cases the proteinograms were modified, the α-globulin fraction being higher and the γ-globulin fraction being lower than in the persons with neg. reactions to the presence of II in the urine. It was concluded that the capacity of the organism to metabolize II decreased after 2 years.

M. Ben Eliezer

1240g Effect of hexachloro-p-xylene ingestion on the oxidative phosphorylation of rat liver mitochondria. Hsia-Ying Chang and Chen-To Yuan (Sino-Soviet Friendship Hosp., Peking, China). *Sheng Wu Hua Hsueh Yu Sheng Wu Li Hsueh Pao* 6(3), 273–5(1966)(Ch). Oral administration of hexachloro-*p*-xylene (I, 0.5 g./kg./day) to rats uncoupled the oxidative phosphorylation of the liver mitochondria, in extent proportional to the duration of I treatment. The decrease in the production of ATP with insufficient energy supply of tissue may be the basis of the toxic reactions of I such as generalized weakness, fatigue, dizziness, headache. BHJJ

1241q Adrenaline-inactivation capacity of the normal and of the CCl_4-poisoned liver perfused in vitro. Emma Costiner, L. Vaisler, and Viorica Chivu (Acad. Rep. Soc., Romania, Bucharest). *Studii Cercetari Endocrinol.* 17(4), 355–7(1966)(Rom). Normal rat livers, and livers originating from CCl_4-treated rats were perfused with adrenaline in vitro. The latter showed a lower rate of adrenaline degradation as compared with the former. BTJQ

1242x Pyruvic acid content in acute barbiturate poisoning. B. F. Murashov (S. M. Kirov Mil. Med. Acad., Leningrad). *Ter. Arkh.* 38(10), 103–4(1966)(Russ). In deep coma due to barbiturate intoxication, 1.2 mg. % pyruvic acid (I) was found in the blood. Its concn. decreased with recovery to 0.77 mg. %. When the I concn. reached 1.8–2 mg. %, complications occurred and the respiration was disturbed. The I concn. was directly proportional to the severity of intoxication, as observed in a total of 32 poisoned humans. Miloslav Kalab

1243e Inhibition of biosynthesis of protein and nucleic acids by phenolic compounds in vivo. G. V. Kukushkina, L. B. Gorbacheva, and N. M. Emanuel (Inst. Chem. Phys., Moscow). *Vopr. Med. Khim.* 12(5), 452–5(1966)(Russ). Mice with the Ehrlich ascites carcinoma or solid hepatoma XXII were injected with a mixt. of uniformly ^{14}C-labeled L-amino acids (I) (10 μc./mouse), Na formate ^{14}C (II) (5 μc.) or adenine-8-^{14}C (III) (17 μc.). After 30 min., one group was injected intraperitoneally with Pr gallate (IV) in 0.9% NaCl and another, with 4-methyl-2,6-di-*tert*-butylphenol (V) in 3% aq. Tween-80. Controls were injected with the resp. solvents. Ascitic fluid samples were taken from mice with Ehrlich tumors at time intervals from 0 to 180 min. and the cells washed with a 5:4:1 mixt. of 0.9% NaCl, 0.3*M* Na_2HPO_4, and 0.3*M* KH_2PO_4. Mice with solid tumors were sacrificed after 120 min. and the tumors, liver, kidneys, and spleen were removed and homogenized. When I was used, the cells were treated with cold 5% Cl_3CCOOH and the pptd. proteins isolated and examd. When II and III were used, 5% Cl_3CCOOH washings were extd. with Et_2O, the aq. layer evapd., and the residue (acid-sol. fraction) examd. for radioactivity. The acid-insol. fraction was washed with cold 96% EtOH and heated for 10 min. at 50° with EtOH–Et_2O (2:1) to remove lipids. A sample was examd. for radioactivity and the rest extd. with 10% NaCl (pH 7.4–7.6) for 30 min. at 95°. Nucleic acids were pptd. by adding 3 vols. of cold 96% EtOH and washed with 70% EtOH. In mice with ascites tumors, injection of 75 mg./kg. of V decreased I incorporation into protein by 30% as compared with the controls; 200 mg./kg. of V inhibited I incorporation almost completely. Injection of 100 mg. of V/kg. decreased III incorporation into nucleic acids by 20%; 200 mg. of V/kg. inhibited III incorporation almost completely. Injection of 80 mg. of IV/kg. or of 120 mg. of V/kg. after 150 min. decreased II incorporation into acid-insol. fraction by 43–55%, into nucleic acid by 54–61%, into acid-sol. fraction by 50–77%, and into proteins, by 39–44%. In mice with solid hepatoma, injection of 150 mg. of IV/kg. after 150 min. decreased I incorporation into tumor proteins by 26%; 200 mg. of IV/kg. decreased I incorporation by 63%. Injection of 150–200 mg./kg. of IV had no effect on protein biosynthesis in kidneys and the liver of normal mice and in kidneys of tumor-bearing mice which showed some activation of I incorporation in the liver. Injection of 200 mg. of IV/kg. decreased I incorporation into spleen proteins of normal and tumor-bearing mice by 30%; 150 mg./kg. had no effect on protein biosynthesis in the spleen. Felix Borek

1244n Experimental intoxication by ammonium salts. Protection by some amino acids and their mechanism of action. F. Cimino and F. Salvatore (Univ. Naples). *Biochim. Appl.* 13 (2), 57–85(1966)(Ital). The biochem. conversion of NH_3 into urea is discussed relative to the protection afforded in acute and chronic NH_3 intoxication by some compds. assocd. with the conversion process. Exptl acute NH_3 poisoning was produced in rats by administering LD_{50} NH_4OAc, preceded by varying doses of protective amino acids and their mixts. Based on mortality and convulsions, arginine (I) provides the most effective protection at as low a dose as 0.5 millimole/kg. A mixt. (*A*) of L-ornithine and L-aspartic acid at a dose of 1.0 millimole/kg. protects as effectively as I. A comparison of blood NH_3 and urea levels using I and *A* shows I to be more effective a protector than *A*. In rats intoxicated with a LD_{50} of NH_4OAc and protected by *A*, α-methylaspartic acid injected intraperitoneally in a dose of 3.4 millimoles/kg. produces approx. 54% mortality and 90% convulsions. Utilizing this same procedure but substituting I for *A*, the mortality and convulsions percentages decrease. Further, a dose of 0.5 millimole I/kg. produces 8.8% mortality and 20% convulsions, while a dose of 1.0 millimole I/kg. produces 21% mortality and 38% convulsions. 67 references. Domenic A. Vavala

1245v Erythrocytic ALA dehydratase and lead. D. Bonsignore, P. Calissano, and C. Cartasegna (Univ. Genoa). *Panminerva Med.* (Engl. Ed.) 8(7–8), 282–4(1966)(Eng). Persons having Pb poisoning showed an increased urinary excretion of aminolevulinic acid (ALA) (I), which was assocd. with a significantly decreased activity of I dehydratase in the erythrocytes. The assay of I dehydratase may be of significance in assessing the gravity of Pb poisoning, since in exptl. animals the level of I dehydratase was related to the extent of the poisoning.

B. S. S. R. Rao

1246c Relations between the porphyrin metabolism and the nicotinic acid metabolism in saturnine poisoning. L. Pecora, A. Silvestroni, and A. Brancaccio (Univ. Naples). *Panminerva Med.* (Engl. Ed.) 8(7–8), 284–8(1966)(Eng). Poisoning of rabbits with $Pb(OAc)_2$ decreased nicotinic acid (I) by 75% in the blood and urine and markedly increased erythrocyte-free protoporphyrin (II) (13–247 γ %) and urinary coproporphyrin (III) (14–341 γ/24 hrs.), aminolevulinic acid (IV) (0.2–21 mg./l.), porphobilinogen (V) (trace–6.6 mg./l.), and xanthurenic acid (VI) (1.0–18.3 mg./24 hrs.). I administration either concurrently with or 15 days after Pb poisoning increased nicotinemia and nicotinuria and markedly inhibited (by 50–80%) the effects of Pb poisoning on II, III, IV, V, and VI. The effects of Pb poisoning are probably mediated through inhibition of pyridine coenzyme synthesis directly or indirectly through vitamin B_6 inhibition. HFJN

1247k Porphyrins in tetraethyl lead poisoning. M. Crepet and P. Chiesura (Univ. Padua, Italy). *Panminerva Med.* (Engl. Ed.) 8(7–8), 295–301(1966)(Eng). The levels of aminolevulinic acid, Pb, and porphyrins were detd. in blood and urine samples of persons having Pb poisoning due to inorg. Pb salts or Pb tetraethyl and also in exptl. animals with exptl. Pb poisoning. Pb poisoning due to Pb tetraethyl caused a significant increase in the levels of free protoporphyrin in erythrocytes, while Pb poisoning due to inorg. Pb salts caused an increased urinary excretion of aminolevulinic acid and coproporphyrin.

B. S. S. R. Rao

1248t Aflatoxin B_1 injury in rat and monkey liver. Donald J. Svoboda, Harold Grady, and John Higginson (Univ. of Kansas, Med. Center, Kansas City). *Amer. J. Pathol.* 49(6), 1023–51(1966)(Eng). Oral administration of pure aflatoxin B_1 (0.45–3.78 mg./kg.) or intraperitoneal injection of a 0.167-mg./kg. dose into rats produced periportal parenchymal cell necrosis and slight bile duct proliferation, together with sepn. of the granular and fibrillar components of the nucleolus with the formation of nucleolar caps. The alterations in fine structure were also found in monkeys after oral administration of 0.45 mg. purified aflatoxin B_1/kg. or intraperitoneal injection of a 2.5-mg./kg. dose. The livers of rats receiving an oral 0.45-mg./kg. dose 24–72 hrs. before sacrifice showed decreased levels of RNA and protein in both the nucleus and cytoplasm. The RNA-to-DNA ratio in a total liver homogenate decreased by 40%, and that in a nuclear fraction by 8%; the protein-to-DNA ratio in the total homogenate decreased by 24%. P:O ratios showed decreasing values with increasing functional damage to the hepatic mitochondria after administration of aflatoxin B_1. Both O consumption and phosphorylation were inhibited, although the essentially normal O consumption 72 hrs. after administration of the toxin may have indicated recovery of respiration without the concomitant return to normal phosphorylation levels. The acute hepatic lesions shown by light microscopy of liver from treated monkeys resembled the changes in human liver during acute viral hepatitis. Administration of pure aflatoxin B_1 (1 ppm. of a synthetic diet fed ad libitum to rats for 33 weeks) did not produce the nuclear, nucleolar, and cytoplasmic alterations found after the acute administration, although well-differentiated hepatocellular carcinomas appeared in rats whose livers otherwise showed little abnormality. 55 references. BPJN

1249a Distribution and excretion of cyclamate-^{14}C sodium in animals. Jonathan P. Miller, L. E. Michael Crawford, Robert C. Souders, and Earl V. Cardinal (Dept. of Pharmacol., Abbott Labs., North Chicago). *Biochem. Biophys. Res. Comm.* 25(2), 153–7(1966)(Eng). Na cyclamate-^{14}C distribution, excretion,