activity. G. V. Martinyan. *Biol. Zh. Armenii* 19(8), 36–41 (1966)(Armenian). In previous studies it was established that the decompn. of proteins is accelerated by poisoning with chloroprene. This process can be interrupted and the protein contained in cells rapidly restored with hyposulfite soln. by an unknown mechanism. Studies made to elucidate the influence of chloroprene and hyposulfite on cathepsin enzymes were inconclusive, and it is suggested that the most important factor in this modification is related to the transaminases. Expts. were conducted using normal rats (control) and rats poisoned with chloroprene (8 mg./l., 2 hrs. daily, 2–3 months). The activities of glutamic-oxalacetic transaminase and glutamic-pyruvic transaminase were measured by using the Reitman and Frankel method (*CA* 51, 13[illegible]) and are expressed in Vroblevski units. The activities of the enzymes decreased 10–19% in blood, liver, and kidneys and 41–51% in the spleen of poisoned animals. In in vitro expts. the decrease in enzyme activity in normal tissues was 4.7–8.4% and in poisoned animals 2.3–7.1%; in the spleen the decrease was 0.4–0.5%. Hyposulfite also lowers the enzymic activity; however, the combination, hyposulfite–chloroprene restores it to the initial level and in some cases even a higher level is reached. Chloroprene does not affect the activity of pyridoxal phosphate. These results lead to the assumption that the inhibitory action of chloroprene can be explained by its reaction with the protein part of the enzyme. L. B. Bedighian

1231c Changes in the esterified fatty acids and cholesterol in serum and liver of rats after application of orotic acid and hepatotoxic substances. F. Musil and J. Sueva (Skoda-Werke-Krankenhauses, Plzen, Czech). *Fette Seifen Anstrichm.* 68(9), 748–50 (1966)(Ger.). Changes in the serum and hepatic levels of cholesterol and esterified fatty acids following oral administration of CCl_4 (0.5 ml./kg.) for the intraperitoneal administration of ethionine (250 mg./kg.) to rats were in some cases normalized following oral administration of orotic acid (100–250 mg./kg.) given as either a preventive or as a therapeutic agent. CMJG

1232k Acute (mouse and rat) and short-term (rat) toxicity studies on dibutyl(diethylene glycol bisphthalate). D. E. Hall, Patricia Austin, and F. A. Fairweather (British Ind. Biol. Res. Assoc., Carshalton, Engl.). *Food Cosmet. Toxicol.* 4(4), 383–8(1966)(Eng). Acute and short-term feeding studies have been carried out on a sample contg. 80% dibutyl(diethylene glycol bisphthalate) (DDGB). The intraperitoneal LD_{50} in both mice and rats was approx. 11.2 g./kg. This dose when given orally was tolerated by both species without ill effect. The toxicity by either route in mice was enhanced when DDGB was administered in arachis oil (50% vol./vol.). In a 90-day study in rats given dietary levels of 0.0 (control), 0.25, 1.0, and 2.5% DDGB, significant growth retardation occurred in both sexes at all levels apart from males on 0.25%. This effect was attributable, at least in part, to reduced food intake, although this effect was less marked in females. There were no hematological changes or effects on kidney function. At the 1.0 and 2.5% dietary levels, the relative liver and heart wts. in males and the relative brain wts. in both sexes were significantly increased. No pathol. changes were found that could be attributed to DDGB. In view of the effect on growth at the lowest dietary level of 0.25%, a no-effect level in the diet of rats for 90 days could not be established for the particular batch of DDGB tested. RCTT

1233t Reaction of hydroxocobalamin with thiols. Norman Adler, Thomas Medwick, and T. J. Poznanski (Merck Chem. Div., Merck & Co., Inc., Rahway, N.J.). *J. Am. Chem. Soc.* 88(21), 5018–20(1966)(Eng). Hydroxocobalamin reacts with thiol compds., as exemplified by glutathione, to form relatively weak 1:1 inner coordination complexes. Previously reported inconsistencies in the generality of this reaction are explained in terms of the simultaneous role of thiol compds. as complexing and reducing agents. KCJC

1234a Neurotoxic effects induced by alkylating agents. M. G. Donelli, R. Rosso, and S. Garattini. *J. Pharm. Pharmacol.* 18(11), 768–2(1966)(Eng). In decreasing order of effectiveness, DL-tryptophan mustard, D-sarcolysine, glycine mustard, L-sarcolysine, DL-sarcolysine, and alanine mustard produced a typical pattern of neurotoxicity when administered intracerebrally to rats. The typical pattern of neurotoxicity included a latency time of about 30 to 60 min., followed by signs of central stimulation, incoordinate movements, and stereotypic behavior, and ending with clonic convulsions and occasional tonic extensions. Mannitol mustard, tetramine, cyclophosphamide, and chlorambucil were not neurotoxic when administered in doses of 100 γ/rat. Intraperitoneal administration of Na phenytoin (100 mg./kg.) was ineffective in preventing the neurotoxic effects induced by the alkylating agents, whereas Na phenobarbitone showed a clear protective effect. Cysteine and thiourea (1 g./kg., intraperitoneally) were also ineffective in preventing the neurotoxic effects. CMJN

1235h Effect of chronic nortriptyline pretreatment on the acute toxicity of various medicinal agents in rats. D. H. Meyers, D. O. Kamyuck, and R. C. Anderson (Eli Lilly & Co., Greenfield, Indiana). *J. Pharm. Sci.* 55(11), 1317–18(1966)(Eng). A method using rats that can be of value in predicting the effects of chronic treatment with psychotherapeutics on the acute toxicity of other medicinal agents is described. Results obtained by this method showed that nortriptyline, like amitriptyline, enhanced the toxicity of most cerebral depressants in an additive manner. Both of the antidepressive compds. markedly potentiated the toxicity of physostigmine and to a lesser degree pilocarpine. Ephedrine toxicity was significantly antagonized by thymoleptic pretreatment. The test failed to verify reports that the tricyclic antidepressives dangerously potentiate the toxicity of ale. or monoamine oxidase inhibitors. Results indicated that a variety of medicinal agents can be used in the nortriptyline treated patient without any problem of significant drug interaction. RCYG

1236r Chlorinated anilines and their sanitary-toxicological characteristics. G. D. Khamuev. *Khim. Faktory Vneshn. Sredy i Ikh Gigien. Znachenie, Sb., Moscow* 1965, 108–10(Russ). Monochloroaniline is used in the production of insecticides and dyes. *m*-Monochloroaniline (I) and *p*-monochloroaniline (II) dissolve in H_2O (5 g./l.), in which they cause an unpleasant taste and odor. The odor and taste perception thresholds were 2.6 and 7.2 for I and 1.5 and 3–6 mg./l. for II, resp. In a concn. of 150 mg./l., I and II lowered the B.O.D. of water. Threshold concns. are 1.5 mg./l. for I and 0.75 mg./l. for II. The $L.D_{50}$ values (stomach administration) for I and II, resp., were 1104 and 401 for white mice, 1104 and 368 for white rats, and 750 and 350 mg./kg. for guinea pigs. A 3-month sub-acute expt., conducted on white rats with stomach administration of 0.1 $L.D_{50}$ levels, caused the formation of methemoglobin and reduced the hemoglobin level and the erythrocyte count. From *Ref. Zh., Khim.* 1966(18), Pt. II, Abstr. No. 18414. MVRK

1237y Toxicity of thioacetamide. Z. Hruban, W. Gradman, A. Slesers, and M. Lubran (Univ. of Chicago). *Lab. Invest.* 15 (11), 1748–60(1966)(Eng). Thioacetamide (I), fed to rats in a diet contg. 50 mg./kg. body wt./day for 5 days, produced high serum lactic dehydrogenase levels and centrolobular necrosis of the livers within 24 hrs.; the enzyme levels decreased and the necrotic areas were resorbed within 3 days of feeding the diet. Severe degenerative changes in the kidneys, hematuria, and increased serum urea N and serum creatinine levels occurred within 48 hrs. of treatment. The lethal effects of the above level of I were correlated with the renal damage. Excess dietary casein hydrolyzate, methionine, or guanosine reduced the mortality and renal toxicity of I, although these compds. did not prevent the nuclear and nucleolar enlargement of hepatic cells and of the cells of the proximal convoluted tubules of the kidney occurring in treated rats. 67 references. EPJN

1235f Poisoning by grass. C. H. Gallagher, J. H. Koch, and H. Hoffmann (Univ. Sydney). *New Scientist* 31(510), 412–14(1966)(Eng). Acute and chronic neuromuscular disorders occur in sheep feeding on the pasture grass *Phalaris tuberosa*. This grass contained 0.3% (dry wt.) tryptamine alkaloids. Tissues from sheep undergoing acute poisoning showed no histol. alterations, whereas the brain and kidneys of chronically poisoned animals showed deposits of green pigment. The pigment was noted in the mitochondria cells of the mid-brain, brain stem, and spinal cord. It is suggested that these changes are related to the oxidative deamination of *N,N*-dimethyltryptamine and 5-methoxy-*N,N*-dimethyltryptamine. The protective action of Co against the chronic form of the disease, reported by others, may be related to the prevention of degenerative changes in nerve tissue or to the acceleration of alkaloid metabolism. W. L. Downs

1239p Vinyl chloride; industrial toxicologic aspects. I. Grigorescu and Gh. Tăbăa. *Rev. Chim.* (Bucharest) 17(8), 498–501(1966)(Rom). A hypothesis was put forward, claiming that $CH_2{:}CHCl$ (I) could react with H_2O at the level of the hepatic cell, yielding ethylene monochlorohydrin, which could be oxidized to chloral and chloroacetic acid (II). II could be eliminated as such in the urine or be metabolized further through amination to glycocol. An anal. method was developed for detection of II in urine, assuming that the only other source of II could be massive ingestion of wine yeast or inhalation of trichloroethylene. An aliquot (200 cc.) of urine, acidified with 50% H_2SO_4 (2 cc.) is extd. with Et_2O (200 cc.), shaking vigorously, and settling for 24 hrs. The ext. is dried (anhyd. Na_2SO_4), evapd. to dryness under reduced pressure, and the residue is dissolved in 50% aq. ale. soln. (0.5 cc.). One drop of the soln. (25–30 µl.) is placed on Whatman No. 1 paper strips (25 × 3 cm.), dried, and chromatographed by ascending method, using as eluant the mixt. NH_4OH–EtOH–H_2O (1:20:4) for a period of 6 hrs. The strips, dried in air, are kept 24 hrs. in a closed NH_3 atm., heated for 5 min. at 80°, sprayed with 0.1% ninhydrin in MeOH, and dried. The II is indicated by reddish-violet spots. For distinct development, the strips are sprayed finally with 10% $Cu(NO_3)_2$ (carmine red spots). The R_f of II was established with a soln. of 20–30 µg. II/cc., as 0.80. The limit of sensitivity is 0.1 µg. II, and is improved by the use of $Co(NO_3)_2$. The extn. of II from urine was demonstrated by treating a soln. of 25 µg. II/cc. in urine as above, cutting the corre-

sponding spots, eluting the II with EtOH, and detg. colorimetrically. The extn. yield was ~80%. Preliminary 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 2–3 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-Te Yuan (Sino-Soviet Friendship Hosp., Peking, China). *Sheng Li Hua Hsueh Yu Sheng Li 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

1241g **Adrenaline-inactivation capacity of the normal and of the CCl_4-poisoned liver perfused in vitro.** Emma Costner, L. Vaisler, and Viorica Chivu (Acad. Rep. Soc., Romania, Bucharest). *Studii Cercetari Endocrinol.* 17(4), 335–7(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), 459–70(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-^{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; 250 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 45–55%, into nucleic acid by 54–61%, into acid-sol. fraction by 50–77% and into proteins, by 30–44%. In mice with solid hepatoma, injection of 150 mg. of IV/kg. after 150 min. decreased I incorporation into tumor proteins by 25%; 200 mg. of IV/kg. decreased I incorporation by 62%. 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 Ilowek

1244a **Experimental intoxication by ammonium salts. Protection by some amino acids and their mechanism of action.** F. Cuomo and F. Salvatore (Univ. Naples). *Boll. Soc. Ital. Biol. Sper.* 42(2), 87–8(1966)(Ital). The biological conversion of NH_3 into urea is discussed relative to the protection afforded in acute and chronic NH_3 intoxications by some compds. assayed, with the conversion process. Exptl., acute NH_3 poisoning was produced in rats by administering L.D.$_{50}$ 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 L.D.$_{50}$ 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. Cartaserena (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 porphyria 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), 234–8(1966)(Eng). Poisoning of rabbits with PbOAc$_2$ decreased nicotinic acid (I) by 75% in the blood and urine and markedly increased erythrocyte-free protoporphyrin (II) (12–247 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 **Porphyrias in tetraethyl lead poisoning.** M. Crenet and P. Chicsura (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 34 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. Sanders, and Earl V. Cardinal (Dept. of Pharmacol., Abbott Labs., North Chicago). *Biochem. Biophys. Res. Commun.* 25(2), 156–7(1966)(Eng). Na cyclamate-^{14}C distribution, excretion,

streptomycin ascorbinate, dihydrostreptomycin pantothenate, paromycin, florimycin and kanamycin are presented. Complex compounds dihydrostreptomycin with ascorbic, pantothenic or p-aminosalicylic acid are less toxic than streptomycin sulfate or dihydrostreptomycin. All above dihydrostreptomycin derivatives may be used in cases with side effects of streptomycin sulfate. Paromycin is preferable since it combines 2 main antituberculous preparations (dihydrostreptomycin and PAS). Florimycin and kanamycin are more toxic than streptomycin sulfate or dihydrostreptomycin and their derivatives. These antibiotics should be limited by patients with progressive processes and streptomycin resistance. Florimycin and kanamycin are ototoxic and should be used only in stationary conditions with constant examination of hearing.--Authors.

† 30951. TAYLOR, T. G., K. M. L. MORRIS, and JEAN KIRKLEY. (Agr. Res. Counc. Poultry Res. Cent., Edinburgh, Scot., UK.) Effect of dietary excesses of vitamins A and D on some constituents of the blood of chicks. BRIT J NUTR 22(4): 713-721. 1968.--Groups of chicks were given diets containing 4 levels of vitamins A and D, 1, 10, 100 and 1000 times the basal level, in all 16 combinations, with the object of investigating a possible antagonism between the 2 vitamins. Only diets containing 1000 times the basal level (approx. 1700 times the dietary requirements) of 1 or both vitamins depressed growth and induced changes in the blood. The packed cell volume was substantially reduced from 4 wk of age in the chicks given the highest level of vitamin A. This was probably due to an effect of the vitamin on the fragility of the red cells and thus on their life span. Chicks given the toxic level of vitamin D showed an increase in plasma Ca and a decrease in plasma inorganic P. The highest level of vitamin A depressed plasma Ca without influencing plasma inorganic P. Increasing amounts of vitamin A given in combination with the highest level of vitamin D caused a progressive increase in the plasma inorganic P. The highest level of dietary vitamin A significantly increased the activity in the plasma of 3 lysosomal enzymes: acid phosphatase, β-glucuronidase and arylsulphatase. Excess vitamin D given in conjunction with the basal level of vitamin A significantly depressed the plasma acid phosphatase and the activity of this enzyme increased with increasing amounts of vitamin A. *Excess* vitamin D had no influence on the other hydrolases studied. A marked antagonism between the effects of excessive amounts of the 2 vitamins occurred only in respect of their actions on the plasma levels of Ca, inorganic P and acid phosphatase, all of which are involved in bone metabolism. --Authors.

30952. TUMARKIN, R. I. (N. F. Gamaleya Inst. Epidemiol. Microbiol., Acad. Med. Sci. USSR, Moscow, USSR.) K voprosu o mekhanizme toksicheskogo deistviya antibiotikov tetratsiklinovoi gruppy na pechen'. [Mechanism of the toxic effect of the tetracycline group of antibiotics on the liver.] ANTIBIOTIKI 13(6): 547-552. 1968. [Engl. sum.] --Chlortetracycline in high doses in rats increased histidine deaminase and urokinase activity. Oxytetracycline had a slight effect on histidinedeaminase activity and suppressed urokaninase activity. Tetracycline effect was similar to that of oxytetracycline. Chlortetracycline has a higher toxicity.--Author.

30953. TYUL'MANKOV, V. N., and B. K. LUGOVSKII. Sosudistye oslozhneniya pri lechenii psikhicheski bol'nykh aminazinom. [Vascular complications during aminazine [chlorpromazine] treatment of mental patients.] ZH NEUROPATOL PSIKHIAT IM S S KORSAKOVA 68(1): 111-115. 1968. [Engl. sum.]--The main syndromes were cerebral hemorrhages and thrombotic vascular affections of vitally important organs. Aminazine profusely increases coagulability. A thorough control (including laboratorial and tests) of old patients and patients with hypertensive disease must be made in the process of aminazine treatment.--From auth. sum.

† 30954. WEISS, HARVEY J. (Mt. Sinai Hosp., New York, N. Y., USA.), LOUIS M. ALEDORT, and SHAUL KOCIWA. The effect of salicylates on the hemostatic properties of platelets in man. J CLIN INVEST 47(9): 2109-2116. Illus. 1968.--Ingestion of 1.5 g of aspirin, but not of sodium salicylate, produced a significant prolongation of the bleeding time in 6 normal male subjects when compared with the effects of a placebo. Similar differences in the effect of the 2 drugs on platelets was also observed. Aspirin ingestion resulted in impaired platelet aggregation by connective tissue and was associated with a decreased release of platelet ADP; sodium salicylate had no effect on these values. *In vitro*, incubation of platelet-rich plasma with an optimum aspirin concentration of 0.045 mg/ml inhibited both the adhesion of platelets to connective tissue and the release of ADP as well as the 2ndary wave of platelet aggregation produced with ADP or epinephrine. Sodium salicylate had no effect on these reactions, which were also normal in patients with von Willebrand's disease. Inhibitory effect produced by ingesting a single 1.8 g dose of aspirin was detectable for 4-7 days at which time salicylate was no longer detectable in the blood, which suggested an irreversible effect on the platelet. Aspirin also inhibited the release of platelet ATP, but had no effect on the platelet surface charge, available platelet ATP or ADP, or the destruction of ADP by plasma ADPase. These studies support the hypothesis that ingestion of aspirin, in contrast to sodium salicylate, prolongs the bleeding time by inhibiting the release of platelet ADP, perhaps reflecting the findings in other cell systems that aspirin alters membrane permeability.--Authors.

30955. ANONYMOUS. The kidney and oral calcium therapy. ANN INTERN MED 68(5): 1021-1022. 1968.--Though $CaCO_3$ is valuable as an antacid, severe and possibly irreversible injury to the kidney may occur from its prolonged use in conjunction with a high milk intake. Evidence of renal function disturbance may be shown by a rise in serum creatinine and renal N. During treatment with $CaCO_3$ a patient's serum Ca level should be watched for early signs of Ca nephropathy within the 1st 7 days of treatment.

INDUSTRIAL TOXICOLOGY

(See also "Pathology" under Respiratory System; "Environmental Health - Occupational Health," and "Radiation Health" under Public Health)

30956. CANDEVIA, BRYAN. (Div. Thorac. Med., Univ. N. S. W., Sydney, N. S. W., Aust.) Pulmonary function in asbestos workers: A three-year follow-up study. AMER REV RESP DIS 96(3): 425-427. Illus. 1967. [Span. and Fr. sum.]--Ventilatory capacity and ventilatory response to a standard exercise test were measured in a series of 41 male workers in a factory making asbestos products, and was repeated about 3 1/3 yr later. Twelve men left the industry during the intermin. They had a significantly lower vital capacity after adjustment for age, and a significantly higher ventilatory requirement for exercise than those who remained in the industry. This pattern of functional abnormality contrasts with that found in subjects admitting to persistent cough and sputum, in whom a significant reduction was demonstrated in age-adjusted forced expiratory volume at 1 sec. In a control series, observed and predicted changes in pulmonary function over the observation period showed reasonable agreement. The asbestos workers showed a greater decrease in forced expiratory volume at 1 sec. than predicted, and possibly greater changes in vital capacity and ventilatory requirement for exercise. The trend toward functional abnormality is consistent with, although not indicative of, the development of asbestosis. Serial pulmonary function tests are likely to be more informative than serial roentgenograms.--From auth. sum.

30957. HARRIS, D. KENWIN, and W. G. F. ADAMS. Acro-osteolysis occurring in men engaged in the polymerization of vinyl chloride. BRIT MED J 5587: 713-714. Illus. 1967.--Two cases of acro-osteolysis occurring in men engaged in the polymerization of vinyl chloride are described. The bones affected were the terminal phalanges of the fingers and the sacroiliac joints, but in 1 case the patella and in the other the phalanges of the feet were involved. The condition was accompanied by Raynaud's phenomenon and skin lesions. This condition is probably a self-limiting disease.--Authors.

30958. SAMEDOV, I. G. *O nespetsificheskikh proyavleniyakh deistviya uglevodorodov v malykh kontsentratsiyakh.* [Nonspecific manifestations of low concentrations of hydrocarbons.] AZERB MED ZH 6: 56-61. 1967. From: REF ZH OTD VYP FARMAKOL KHIMIOTER SREDSTVA TOKSIKOL, 1968, No. 4.54.915. (Translation) --Workers were tested at a petroleum-processing plant where the concentration of hydrocarbon vapors did not exceed the maximum allowable (0.07-0.48 mg/l. Rabbits and guinea pigs were treated under various static regimes with the vapors of gasoline B-70 at 0.95-1.1 ml/l, 4 hr/day, 6 times/week for 4 mo. The workers and the animals showed a rise in the concentration of blood acetylcholine and epinephrine-like substances. Animals treated constantly and evenly with increasing hydrocarbon concentrations showed these tendencies, but they were less marked. Cholinesterase activity in the workers did not differ from that of controls, but it was reduced in treated animals, especially when the treatment was with fluctuating concentrations of hydrocarbons. Pseudocholinesterase activity did not change in either persons or animals. Daily urinary excretion of 17-ketosteroids by treated rabbits was of a phasic character: the elevated activity of the adrenocortical function was soon replaced by reduced activity. Only under the action of sharply fluctuating hydrocarbon concentrations was there a prolonged rise in 17-ketosteroid excretion. Maximum depression in the concentration of vitamins C and B_1 in the blood, urine, and organs was noted in animals treated with steadily and evenly increasing and also sharply fluctuating hydrocarbon concentrations. Changes in the electrolyte metabolism with low concentrations was insignificant. The principal changes with low hydrocarbon concentrations were in the functional condition of the autonomic and endocrine systems (changes in the metabolism of mediating substances and vitamins and in adrenocortical function). These changes are probably nonspecific and of a compensatory-adaptational nature. Over a certain period they may lead to persistent and even organic changes.

30959. SPAK, IVAR. (Dep. Surg., Lanslasarettet, Hassleholm, Swed.) Finger injury caused by paint spray gun. ACTA CHIR