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Inst. Hyg. and Occupational Diseases, Gorki, U.S.S.R. *Toksikol. Novykh Pestitsidov i Klinika Otravlenii, Dokl. 2-oi [Vtoroi] Vses. Konf. 1962, 350-3.* Single peroral doses of 0.5-7.0 g. phenyldimethylurea (I)/kg. did not cause death of mice and rats. Repeated inhalation of I dust (1.0-1.4 mg. I/l. air) was followed by hypertrophy of adrenal glands up to double their normal wt. Repeated spreading of I on skin resulted in depilation lasting 1-3 months; thus, I is classified as a low-toxicity herbicide. Miloslav Kalab

The mechanism of action of carbon tetrachloride in inducing variations of some serum enzymes of probable hepatic origin. A. Sabatini Pellegrini (Univ. Milan). *Boll. Soc. Ital. Biol. Sper.* 39(23), 1435-9(1963). The effect of CCl_4 in olive oil soln., given by gastric probe to male rats of 300 g. av. wt., on lactic dehydrogenase (LDH), glutamic-pyruvic transaminase (GPT) and glutamic-oxalacetic transaminase (GOT) was studied. Expts. were made with normal rats, adrenalectomized rats, and adrenalectomized rats treated with 10 mg./kg. corticosterone (I) injection. The enzymes were analyzed spectrophotometrically. In normal rats there was a sharp increase of GOT and GPT 2 hrs. after the CCl_4 administration, and the level reached more than twice the normal value after 4 hrs. The increase of LDH was similar, but less pronounced. In animals which had been adrenalectomized 72 hrs. before the expt. there was no increase in enzyme levels; at the same time, the body temp. decreased by about 5.5° . Adrenalectomized rats to which I was given showed the same high value of LDH as normal rats after CCl_4 treatment; GOT and GPT were sharply increased, but not quite as in normal rats after CCl_4 administration. If the drop of body temp. was prevented in the adrenalectomized animals by keeping them in a thermostatically controlled room, their enzyme levels were as high as in the adrenalectomized rats that received I. It is surmised that hypothermia is a self-defense of the liver against CCl_4 damage. H. G. Figgdor

Metabolism of ^{32}P -labeled Shell SD-4294 in a lactating ewe. W. F. Chamberlain (U.S. Dept. of Agr., Kerrville, Texas). *J. Econ. Entomol.* 57(1), 119-21(1964). A study was made of the absorption, elimination, and metabolism of orally administered ^{32}P -labeled Shell SD-4294 (Ciodrin, α -methylbenzyl 3-hydroxycrotonate dimethyl phosphate) in a lactating ewe. The peak radioactivity in both the blood and urine occurred at 6 hrs., which indicated a moderate rate of absorption but rather rapid elimination. Some 61-90% of the radioactivity in the urine was dimethyl phosphoric acid; the other metabolites occurred only in small quantities. The ratio of α to β isomer in the blood was similar to that in the original sample of SD-4294, whereas the very small residues in the milk consisted of β isomer only. RCJS

Biochemical studies on minamata disease. Makio Uchida (Univ. Kumamoto, Japan). *Seikagaku* 35, 430-9(1963). A causative agent of the disease was isolated from poisonous mussel, *Hormomys mutabilis*, and was found to be $(\text{CH}_3\text{Hg})_2\text{S}$ (I). Animal test with synthesized I caused a typical minamata disease. 54 references. T. Akazawa

Hazardous substances; benzene, petroleum distillates, toluene, xylene; labeling requirements. Anon. *Federal Register* 29, 1802-3(Feb. 6, 1963); cf. *CA* 55, 25163g. Human experience establishes that products contg. more than 5% of the title materials are hazardous and require special labeling under the Federal Hazardous Substances Labeling Act. William Horwitz

Toxicology of mercaptophos. S. I. Loktionov. *Gigiena i Toksikol. Novykh Pestitsidov i Klinika Otravlenii, Dokl. 2-oi [Vtoroi] Vses. Konf. 1962, 220-2.* In rats, cats, and dogs to which mercaptophos was administered the av. cholinesterase activity of the brains was 75% lower than in control animals. Heavy vessel destruction and dystrophic changes were observed in the central nervous system of dead exptl. animals. Cats and dogs had approx. equal sensitivity to this poison: L.D.₅₀ was 3.9 mg./kg. for cats and 3.65 for dogs. St. Janderka

Cigaret smoking and blood lipids. Alfred Kershbaum and Samuel Bellet (Philadelphia Gen. Hosp., Philadelphia, Pa.). *J. Am. Med. Assoc.* 187(1), 32-6(1964). Cigaret smoking and nicotine have a biol. significant effect on lipid metabolism. Smoking in humans and nicotine in dogs caused a rapid and consistent rise in serum free fatty acid (FFA) levels and patients with myocardial infarction developed an elevation more than twice that of normal subject or noncoronary patients. An increase in urinary catechol amine excretion accompanied the FFA response to smoking. Sympathetic ganglionic blockade prevented or inhibited the FFA rise. Adrenalectomized patients showed a negligible rise or none. Smoking had a variable effect on fat tolerance except in subjects with fasting hypertriglyceridemia in whom smoking caused an impairment of fat tolerance. In dogs given nicotine daily for 6 weeks there was a 50% rise in the serum cholesterol level. Rachel Brown

Histopathology of chloroquine retinal toxicity. Dennis H. Wetterholm and Frank C. Winter (Stanford Univ., Palo Alto, Calif.). *Arch. Ophthalmol.* (Chicago) 71(1), 82-7(1964). In a patient on prolonged chloroquine (I) therapy an irreversible retinal pathology developed which included destructive lesions of

in the outer nuclear and outer plexiform layers. John F. R. Kuck, Jr. 40:9816e
The specificity of the cytotoxic action of silica. A study in vitro. R. W. I. Kessel, L. Monaco, and M. A. Marchisio (Univ. Milan). *Brit. J. Exptl. Pathol.* 44, 351-64(1963). In monolayer cultures of macrophages from guinea pig, rabbit, mouse, rat, and man, the production of lactic acid and the O uptake are rapidly decreased by tridymite, but not by hematite, C, or vitreous Si. Tridymite when coated with amorphous Si or Al_2O_3 was rendered nontoxic. A special surface is therefore necessary for the cytotoxic effect of Si. Polymorphonuclear leukocytes were not injured by tridymite, nor were several other types of cell cultures. M. L. C. Bernheim

Changes in the protein composition of the blood serum in experimental lead poisoning. A. T. Aldanazarov, T. Kh. Aitbaev, and N. A. Kutyrykina. *Izv. Akad. Nauk Kaz. SSR, Ser. Med. Nauk* 1963(2), 48-53. In Pb poisoning there is no great change in the total protein of dogs as detd. by refractometry, but paper electrophoresis shows that the albumin and γ -globulin concns. are reduced and those of the α - and β -globulins are increased. John Howe Scott

Effects of chlorine gas on the lungs of rats with spontaneous pulmonary disease. P. C. Elmes and Deirdre Bell (Queen's Univ., Belfast, Ire.). *J. Pathol. Bacteriol.* 86, 317-26(1963). Rats with spontaneous lung disease exposed to Cl develop acute necrosis of the bronchial mucosa, and acute inflammation with a cellular exudate and blockage of the peripheral bronchi. Cl produces excessive mucus secretion and focal damage to the lung periphery. John T. Myers

Effect of guanidine and histamine in CCl_4 toxicity in animals. T. P. Veselova, Yu. A. Velikovskaya, and L. M. Gordeeva. *Tr. Vses. Inst. Gel'mintol.* 10, 178-84(1963). As the clin. results of histamine, guanidine, and CCl_4 intoxication is very similar, the effects of guanidine and histamine in the toxic process were investigated. The L.D.₅₀ for rabbits was 2 mg./kg. of histamine-2HCl and 335 mg./kg. of guanidine. Guanidine increased the toxicity of histamine. Preliminary administration of CaCl_2 , glucose, Na lactate, and dimedrol prevented histamine shock and guanidine intoxication. In expts. with rabbits histamine was 183 times as toxic as guanidine. Introduction of CCl_4 caused an increase of the amt. of guanidine in the blood of animals, while guanidine initiated the liberation of surplus labile histamine. St. Janderka

The effect of histamine on the mechanism of CCl_4 toxicity in cattle. T. P. Veselova, Yu. A. Velikovskaya, and L. M. Gordeeva. *Tr. Vses. Inst. Gel'mintol.* 10, 169-78(1963). It was observed in expts. with 16 cows (11 of them fluke infected) that the histamine level in blood plasma rose from 3-8 γ % to 13-25.6 γ % in 24 hrs. after intramuscular administration of 4-5 ml./100 kg. wt. of CCl_4 dild. in an equal amt. of oil and to 33-40 γ % after enteral administration of this drug. Antagonistic effect of substances of antihistamines was only of short duration (3 hrs. with dimedrol, *p*-aminobenzoic acid, adenosine triphosphate, and citral. St. Janderka

An electron microscopy study of the effect of asbestos dust on the lung. J. M. G. Davis (Inst. Pathol., Cambridge, Engl.). *Brit. J. Exptl. Pathol.* 44, 454-64(1963). Rats and guinea pigs were subjected to the inhalation of chrysotile asbestos dust. Small nodular giant cell lesions of dust-carrying macrophages are formed, followed by fibrosing interstitial pneumonia, and finally consolidation of the lung. Alveolar macrophages are the only cells which can phagocytose the dust. Aggregates of ferritin occur around the chrysotile particles. The capillary walls are sometimes thickened. M. L. C. Bernheim

Clinical and laboratory data in chronic poisoning due to trichloropropene. F. Linari, F. Capellaro, and P. C. Gaido (Univ. Turin, Italy). *Rass. Med. Ind.* 31(3), 227-38(1962). Four cases of exposure to 1,3,3-trichloropropene vapor were described. The effects to a large extent were similar to those of trichloroethylene. It is an irritant to the mucous membranes. Its immediate effect was a disturbance of the central nervous system producing headaches, dizziness, asthenia, and restlessness. After exposure the dominant symptoms centered on the gastrointestinal system. Exposures reported here were brief and moderate except in one case where a worker was splashed with the liquid. In this worker electrocardiographic alterations were noted that persisted for some time, while in the others the findings were neg. Routine blood and urine analysis results were in all cases within normal limits, as were liver function tests. The Fujiwara reaction was pos. in all cases, but the Nash test which is specific for chloro derivs. of EtOH was consistently neg. Leonard D. Pagnotto

Sanitary-hygienic working conditions and pneumoconiosis morbidity rate among electric welders in the Ural automobile factory. K. E. Bakaleinik-Kazachkova. *Gigiena Truda i Tekhn. Bezopasnosti pri Elektrosvarochn. Rabotakh, Moscow* 1962, 54-9. The working conditions and death rate were studied in the welding and assembly-welding shops of a car factory. The compn. and amt. of dust generated during welding with the electrodes