FILE NAME: Chemical Abstracts (CHAB)

DATE: 1966

DOC#: CHAB055

OCUMENT DESCRIPTION: Abstract Originally Published in 1964 by Medek

1966

10–36 months, and wt. 6–15 kg.) given a single $^{238}PuO_2$ treatment. Translocation and sepn. through urine and feces were greatest within 1 month when aerosols with very small particle diams. were used. With particle sizes of 4.3 μ, the half-time for lung retention was ~300 days, with simultaneous accumulation in the lymph nodes, which gave a total body retention of Pu of ~1500–1800 days. The distribution was completely different after Pu inhalation than after the intravenous injections; for example, the urine sepn. rate with inhalations was ~5 times as high as with the injections. From *CZ* 1964(21), Abstr. No. 1427. a

MRCR

Presence of mediators in the blood in lead intoxication. II. Acetylcholine in experimental lead poisoning. N. N. Tikhonov. *Izv. Akad. Nauk Kaz.SSR, Ser. Med. Nauk* 1964(3), 65–9 (Russ). The concn. of acetylcholine in the blood of dogs given 1 ml. 2.5% $Pb(OAc)_2$ daily rose only slightly and remained at that level until about the 170th day when it rose sharply to about 150 γ/ml. as the condition of the animals became much worse. b

John Howe Scott

Effect of edetic acid, bis[2-[bis(carboxymethyl)amino]ethyl] ether, and diethylenetriaminepentaacetic acid on lead-210 retention in mice. Dariusz Brykalski and Danuta Depczyk (Inst. Med. Pracy, Lodz, Poland). *Med. Pracy* 14(6), 439–47(1963). ^{210}Pb (1–4 μc.) was given intravenously to mice in the form of $Pb(NO_3)_2$ in 0.2*N* acetate buffer and immediately followed by edetic acid (I), bis-[2-(dicarboxymethylamino)ethyl] ether (II), or diethylenetriaminepentaacetic acid (III), each given intraperitoneally in the form of the Ca complex dissolved in 0.9% NaCl (0.2–0.4 ml.). The animals were sacrificed 48 hrs. later and the radioactivity detd. in sep. organs. Accumulation of ^{210}Pb in liver, kidneys, femoral bones, other parts of the body, and in the 48-hr. urine and feces (percent of intake) were: with I, 6.3, 2.3, 0.7, 17.4, 66.4, resp.; with II, 6.8, 2.9, 1.6, 26.9, 52.9, resp.; with III, 5.7, 1.9, 0.5, 9.4, 68.2, resp.; in controls, 6.8, 6.4, 2.2, 32.5, 28.4, resp. When the chelating agents were administered in 5 × 40-millimole portions given every other day, the total retention of ^{210}Pb, as found in the 11th day of the expt., was 32.08%, with I 35.98%, with II 28.38%, with III, and 46.77% in controls. When administration of the chelating agents was started on the 5th day of the expt., the final retention values were 72.9, 85.5, and 70.8%, resp. c

Jerzy Lange

Serum proteins in asbestosis. Vladimir Medek and Jiri Radl (Oddeleni Nemoci Povolani Koniggratz, Czech.). *Pracovni Lekar.* 16(8), 349–52(1964)(Czech). There was a mild decrease in serum proteins and an increase in the γ-globulins, both in relative and abs. values and more markedly in women, in 30 patients whose serum was investigated by paper electrophoresis, flocculation reactions, and immunoelectrophoresis. Paper electrophoresis of serum proteins is thought of sufficient value as a diagnostic means in asbestosis. d

L. J. Urbanek

Elimination of solid particles [silicon dioxide] from the lungs and formation of antibodies. Juraj Ferin, Eva Reichrtova, Gertruda Urbankova, and Angelika Vlckova (Ceskoslov. Akad. Ved, Bratislava). *Pracovni Lekar.* 16(9), 410–12(1964)(Slo). There was a pos. correlation between the elimination of SiO_2 dust in rats exposed to forced inhalation and the spontaneous production of antibodies (hemolysin). Possible utilization of the results in occupational hygiene is discussed. L. J. Urbanek e

The influence of methemoglobinemia on the lethality of some toxic anions. I. Azide. Roy A. Abbanat and Roger P. Smith (Dartmouth Med. School, Hanover, N.H.). *Toxicol. Appl. Pharmacol.* 6(5), 576–83(1964). The antidotal capacity of methemoglobinemia against a variety of toxic anions was tested in mice, using $NaNO_2$ and *p*-aminopropiophenone (I) to induce the methemoglobinemia. $NaNO_2$ at a dose of 75 mg./kg. produced a max. methemoglobin level of 34% in about 40 min.; the max. concn. of 36% for I was achieved in about 10 min. The methemoglobinemia induced by either agent was effective in protecting against NaN_3 given 20 min. after the reagent. $NaNO_2$ increased the $L.D._{50}$ approx. 20% and I was at least as effective. No protection was afforded against F^-, CNO^-, CNS^-, SeO_4^{2-}, or BO_2^-, although survival was prolonged after F^-. The presence of an azide-methemoglobin complex was demonstrated in mouse red cells *in vitro*, and small amts. of the complex were identified *in vivo*. Presumably the mechanism of this protection involves formation of an intermediary complex between the toxic anion and methemoglobin, as is the case for CN^-. f

II. Sulfide. Roger P. Smith and R. E. Gosselin. *Ibid.* 584–92; cf. preceding abstract. Pretreatment with $NaNO_2$ or I protected armadillos, rabbits, and mice from parenterally administered Na_2S. The $L.D._{50}$ of Na_2S in female mice was increased more than 2-fold by 75 mg. of $NaNO_2$/kg. given intraperitoneally 20 min. before intraperitoneal injection of Na_2S. Pretreatment with either agent increased the survival time of mice exposed to 722, 985, or 1872 ppm. of H_2S; the protection was greatest in those mice exposed to the intermediate concn. of H_2S. Propylene glycol, used as a solvent for I also had some protective action against H_2S, probably due to its action in depressing respiration. The g

is discussed with reference to the prodn. of sulfmethemoglobin and its possible fate *in vivo*. Preliminary expts. with armadillo blood showed its oxyhemoglobin and ferricyanide-produced methemoglobin to have absorption spectra qual. like those of human blood pigments.

Frank A. Smith

Changes in thyroid ^{131}I activity in ozone-tolerant and ozone-susceptible rats. Edward J. Fairchild, II, Stuart L. Graham, Mark Hite, Richard Killens, and Lester D. Scheel (U.S. Dept. of Health, Educ., & Welfare, Cincinnati, Ohio). *Toxicol. Appl. Pharmacol.* 6(5), 607–13(1964). Single 5-hr. exposures of male rats to 1, 2, and 4 ppm. of O_3 inhibited the thyroid release of ^{131}I. Under exposure conditions known to produce tolerance to O_3, viz repeated 5-hr. exposures to 0.5 or 1 ppm., O_3 did not alter the ^{131}I content of the thyroid. Two 5-hr. exposures to 2 ppm. O_3 followed later by a challenge exposure to 4 ppm. resulted in an increased rate of release of ^{131}I. This tri-phasic response of the thyroid to O_3 indicates that the tolerance phenomenon induced by certain irritant gases is assocd. with, but not necessarily detd. by a resistant phase of thyroid activity, and suggests activation of humorally mediated mechanisms by O_3 at concns. as low as 0.5 ppm.

Frank A. Smith

Amanita phalloides poisoning. Guenter Obauer and Harald Schoen (Univ. Klin., Erlangen, Ger.). *Arzneimittel-Forsch.* 14 (11), 1257–9(1964)(Ger). Course and treatment of hepatic lesions induced by the administration of phalloidin were investigated. While comparable doses led to an increase in transaminase in rabbits, reaching a max. 40 hrs. after administration of the poison, mice succumbed within a few hrs. Thioctic acid proved to be an effective antidote.

Rudolph Seiden

Biochemical findings in mushroom-poisoned patients. H. E. Bock, F. W. Aly, M. Eggstein, W. Gerok, W. Kauffmann, G. Scheurlen, and H. D. Waller (Med. Univ.-Klin., Tuebingen, Ger.). *Klin. Wochschr.* 42(21), 1039–52(1964)(Ger); cf. *CA* [H. E. Bock, *et al.*, *Deut. med. Wochschr.* 89, 1617–22(1964)]. Observations on the poisoning of 10 patients by *Amanita phalloides* are reported and compared extensively with published work. The metabolic effects of the toxin are discussed. Protein concn. in the serum increased by hemoconcn. in the initial stages of intoxication. Electrophoresis indicated an initial decrease of albumin and γ-globulins, followed by a slower increase during the second week. The γ-globulin concn. eventually reached a high value indicative of liver damage. There was no evidence for a general severe hemolysis during the poisoning. There was no increase in bilirubin concn. in the serum, but the activities of serum enzymes increased; both results being characteristic of liver damage. Cholesterol (and sometimes phospholipids) decreased to <120 mg./100 ml. serum in 5 patients during the first days. A decrease in serum arginine was noted. At the start of intoxication, most patients showed a metabolic acidosis in the blood. Within 3 to 4 days the correct balance was restored by infusion of lactate-glucose and corticosteroids. The serum electrolytes were usually normal. The concns. of ATP and methemoglobin in the red blood cells were normal. The activities of glucose-6-phosphate dehydrogenase and of glutathione reductase were normal in 8 of 10 cases. In all cases the glutathione value of the red cells and the glutathione stability decreased and the Heinz-bodies increased in the early stages of intoxication. 74 references.

D. H. Hutson

Importance of atmospheric lead in public health. D. H. Hofreuter, E. J. Catcott, R. G. Keenan, and C. Xintaras (Robert A. Taft Sanitary Eng. Center, Cincinnati, Ohio). *Arch. Environ. Health* 3, 568–74(1961)(Eng). Approx. 1000 blood and 250 urine samples from humans were tested for Pb last year in 6 large cities and in a rural area. Although there were considerable differences in Pb content between the city and rural population and between the smokers and nonsmokers, the av. values were always within normal limits. Not a single value exceeded the normal amt. of 0.07 mg. Pb/100 g. of blood. From *CZ* 1964(24), Abstr. No. 1532.

MWCR

A liquid pump of precisely constant flow rate for the continuous measurement of air pollution. Saburo Yanagisawa, Yoshikazu Hashimoto, Shumpo Mitsuzawa, and Akira Hirose (Keio Gijuku Univ., Tokyo). *Bunseki Kagaku* 12(11), 1069–70(1963)(Japan). A simple pump using 2 counter-acting syringes operated by a synchronous motor was constructed to give a const. flow rate of Saltzmann reagent in which air was absorbed continuously to det. the NO_2 concn. in the air as a function of time.

Hideaki Chihara

The deposition of aerosols in the respiratory tract. I. Mathematical analysis and comparison with experimental data. J. M. Beeckmans (Univ. Toronto). *Can. J. Physiol. Pharmacol.* 43(1), 157–72(1965). Deposition of inhaled aerosol particles in the human respiratory tract was calculated, by electronic computer, as a function of the particle size and particle density of the aerosol, the manner of breathing, and various parameters which control the degree of mixing of inspired air with the dead space air and with the lung air. Agreement between the computed total deposition curves, and published exptl. data was very satisfactory. h

A. E. Teeri