[illegible] IN RATS AND [illegible] . Harris, R. C. Hodge, [illegible] A. [illegible] & H. J. [illegible] Jr. ARCH. IND. HEALTH 1[illegible], p. [illegible] ([illegible])

BEHAVIOR OF THE ORGANISMS INFLUENCED BY VARIOUS PLASTICIZERS. (toxicity studies) G. Dormann et al. Z. LEBENSMITT-UNTERSUCH 103, p. 413 (1956)

PROPOSALS FOR EVALUATION OF SYNTHETIC MATERIALS USED IN THE FOOD INDUSTRY. (identification of plasticizers and stabilizers for packaging films, and evaluations of their toxicity) H. Staub. MITT. LEBENSM., HYG. 49, p. 1 (1958)

THE TOXICITY OF PLASTICS MATERIALS. PACKAGING 41, No. 342, p. 72 (December 1958)

TOXICITY EXAMINATION OF PVC FOILS. O. Berndt. PLASTE u. KAUT. 6, No. 7, p. 347 (July 1959)

INVESTIGATIONS OF THE TOXICITY OF POLYVINYL CHLORIDE STABILIZERS. I. V. Nebel & O.R. Klimmer. ARZNEIMITTEL-FORSCHUNG 10, No. 1, p. 44 (January 1960)

THE TOXICITY OF PLASTICS. (especially lead stabilizers) S. F. Chancellor. NATURE 185, No. 4716, p. 841 (March 19, 1960)

PLASTICS. (Review of toxicity references) R. H. Wilson & W. E. McCormick. ARCH. IND. HEALTH 21, No. 6, p. 536 (1960)

THE TOXICOLOGICAL PROBLEMS OF PLASTICS USED IN FOOD PACKAGING. IND. PLASTIQUES MOD. 12, p. 59 (June 1960)

CHRONIC TOXICITY STUDIES ON TWO EPOXIDIZED SOYBEAN OILS IN THE RAT AND DOG. P. S. Larson et al. TOXICOLOGY & APPL. PHARMACOLOGY 2, No. 6, p. 649 (November, 1960)

THE ACUTE TOXICITY OF THE TRIARYL PHOSPHATE USED AS PLASTICIZERS. H. F. Bondy et al. BRIT. J. IND. MED. 17, p. 190 (1960)

PHYSIOLOGICAL AND TOXICOLOGICAL PROPERTIES. (Toxicity of vinyl resin stabilizers) R. Lefaux. PLASTIQUES INFORMATIONS 12, No. 257, p. 4 (1961)

SANITARY-CHEMICAL STUDY OF PVC-COATED FABR[illegible] N. I. Slepak. UCH. ZAP. MOSK. NAUCH. ISSLED. INST. GIGIENY, No. 9, p. 121 (1961)

THE TOXICITY OF RIGID PVC DRINKING WATER CONDUITS. A. Rodeyns. REV. BELGE MAT. PLASTIQUES 3, No. 1, p. 17 (January 1962)

TOXIC AND HEALTH EFFECTS OF PLASTICS AND RESINS. J. A. Zapp. ARCHIVES ENVIRONMENT. HEALTH 4, p. 335 (March, 1962)

A MINIMUM STANDARD TO MAKE PLASTICS SAFE IN MEDICAL APPLICATIONS. Dr. John Autian.

PLASTICS WORLD 20, No. 3, p. 12 (March, 1962)

HEALTH AND SAFETY IN THE PLASTICS INDUSTRY. D. Kenwin Harris. PLASTICS INST. TRANS. 30, No. 86 (April 1962)

THE PROBLEM OF NON-TOXICITY OF ADDITIVES IN THE DOMAIN OF PLASTICS. E. LeClerc. IND. PLASTIQUES MOD. 14, No. 10, p. 37 (Dec. 1962)

48. VISCOSITY & FLOW PROPERTIES

MEASUREMENT OF THE FLOW TEMPERATURE OF THERMOPLASTIC MOLDING MATERIALS. L. W. A. Meyer. A.S.T.M. BULL. No. 105, p. 23 (August, 1940)

FLOW TEMPERATURE OF THERMOPLASTICS. L. W. A. Meyer. MOD. PLASTICS 18, No. 4, p. 59 (December 1940)

FLOW PROPERTIES OF THERMOPLASTICS. I. W. G. Wearmouth & J. S. Small. BRITISH PLASTICS 12, No. 144, p. 377 (May 1941)

WORKING-RANGE FLOW PROPERTIES OF THERMOPLASTICS. F. E. Wiley. IND. ENG. CHEM. 33, No. 11, p. 1377 (November, 1941)

SOME RHEOLOGICAL PROPERTIES OF POLYVINYL CHLORIDE. L. Bilmes. J. SOC. CHEM. IND. 63, No. 6, p. 182 (June 1944)

HIGH TEMPERATURE, HIGH PRESSURE RHEOMETER FOR PLASTICS. H. K. Nason. J. APPLIED PHYS. 16, No. 6, p. 338 (June 1945)

RHEOLOGICAL PROBLEMS IN THE PROCESSING OF PLASTICS. R. Buchdahl & H. K. Nason. IND. ENG. CHEM. 40, No. 4, p. 642 (April 1948)

VISCOELASTIC PROPERTIES OF [illegible] HIGH POLYMERS. M. Horio et al. J. APPLIED PHYS. 22, No. 7, p. 966 (July 1951)

RHEOLOGY AND THERMOPLASTICS. E. E. Atkinson & H. A. Nancarrow. PLASTICS INST. TRANS. 19, No. 38, p. 23 (October, 1951)

APPLICATION OF NUTTING'S EQUATION TO VISCOELASTIC BEHAVIOR OF CERTAIN POLYMERIC SYSTE[illegible] R. Buchdahl & L. E. Nielsen. J. APPLIED PHYSICS 22, No. 11, p. 1344 (November, 195[illegible])

THE ELASTIC-VISCOUS BEHAVIOR OF PLASTICIZED POLYVINYL CHLORIDE AT ELEVATED TEMPERATURE. W. Tsuji & K. Shiozawa. BULL. INST. CHEM. RESEARCH, KYOTO UNIV. 27, p. 67 (1951)

TWO EXTRUSION PLASTOMETERS FOR USE WITH POLYVINYL CHLORIDE. R. Hayes. PLASTICS 17, No. 177, p. 104 (April 1952); and CHEM. & IND. (1952) p. 421 and p. 1069

VISCO-ELASTICITY OF HIGH POLYMERS. II. KIN[illegible] BEHAVIOR OF PLASTICIZED POLYVINYL CHLORIDE NEAR THE TRANSITION POINT. S. Nohara & S. Ouchi. CHEM. HIGH POLYMERS (Japan) 10, p. 286 (1953)

POLYVINYL CHLORIDE RESIN. IV. VISCOMETRIC STUDIES OF MASTICATED PVC. K. Goto et al. CHEM. HIGH POLYMERS (Japan) 11, p. 437 (19[illegible]

A KINETIC INTERPRETATION OF THE RHEOLOGICAL BEHAVIOR OF HIGH POLYMERS. Junji Furukawa. J. POLYMER SCI. 15, No. 79, p. 193 (Januar[illegible] 1955)

INTERPRETATION OF BROOKFIELD VISCOSITIES. R. L. Bowles, R. P. Davie & W. D. Todd. MOD. PLASTICS 33, No. 3, p. 140 (November, 1955)

[illegible]

[illegible]
[illegible]

CHEM. AGE
19, 1957)

VINYL CHLO-
C. A. Kumins
. 10, p. 1290

ILMS. W. E.
N PLASTICS 36,

RS TO 90%
ietrick and
ER SCI. 2, No.
1959)

SUPPORTED
, C. N.
. APPLIED
(September/

IN CONTACT
. Woelk.
5 (December

TO GASES. B.
O KAGAKU

POLYMERS TO
rlin and K. P.
2252

GH POLYMERIC
tt and R. M.
CI. 3, No. 7,

LMS FOR GASES
THE DEGREE OF
TION ON THE
SHI KAGAKU

YMERS. H. H.
POLYMER SCI.
)

D PVC. H.
. 12, No. 7,

D PVC. H.
5, p. 359

TIC FILMS.
uary 1961)

EABILITY C
. VI. THE
. Myers et
nuary, 1961)

ERS ON THE
OUGH HIGH
KAGAKU
ry, 1961)

S OF
nd W. A.
26, No.

[illegible], p. 117 (February, [illegible])

ON THE WATER VAPOR [illegible] POWDERED FOODSTUFFS [illegible] PROOFING BY SOME PACKAGING FILMS. T. Makino et al. KOGYO KAGAKU ZASSHI 64, No. 7, p. 1255 (July 1961)

THE PERMEABILITY OF PVC-CELLULOSE LAMINATES TOWARDS HYDROGEN SULFIDE AND AMMONIA. H. Braunish and H. Lenhart. KOLLOID-ZEITS 177, No. 1, p. 24 (July 1961)

PERMEABILITY OF PLASTICS. I. Phillips and D. V. Bartlett. BRITISH PLASTICS 34, No. 10, p. 533 (October, 1961)

DIFFUSION OF GASES AND VAPORS THROUGH POLYVINYL CHLORIDE- POLYVINYL ACETATE COPOLYMER FILMS. I. GLASS TRANSITION EFFECT. II. THE EFFECT OF POLYMER SEGMENT MOBILITY. C. A. Kumins and J. Roteman. J. POLYMER SCI. 55, No. 162, p. 683 (December 1961)

WVT THROUGH MULTIPLE BARRIERS. K. W. Ninnemann and L. E. Simerl. MODERN PACKAGING 35, No. 4, p. 137 (December 1961)

PERMEABILITY OF PLASTIC MATERIALS TO WATER VAPOR. Marie Korte-Palinski. J. CHIM. PHYS. 59, No. 1, p. 27 (January 1962)

DIFFUSION OF GASES AND VAPORS THROUGH UNPIGMENTED VINYL FILMS -- GLASS TRANSITION EFFECT. C. A. Kumins & D. J. Waythomas. OFF. DIG. J. PAINT TECH. & ENG. 34, No. 445, p. 117 (February 1962)

[illegible] PERMEABILITY OF HIGH POLYMERS. J. [illegible] u. KAUT. 7, No. 3, p. 117 (March 1962)

ACTION OF LOW-MOLECULAR LIQUIDS ON CERTAIN POLYMERIC MATERIALS. (effects of immersion in water and gasoline) V. A. Voskresenskii. ZHUR. PRIKLAD KHIM. 35, No. 3, p. 696 (1962); and J. APPL. CHEM. U.S.S.R. 35, No. 3, p. 676

DETERMINATION OF THE PERMEABILITY OF PLASTIC CONTAINERS TO OXYGEN. A. Dousa and V. Simak. OBALY 8, No. 4, p. 104 (1962)

GAS PERMEABILITY OF PLASTICS. Coleman J. Major and Karl Kammermeyer. MODERN PLASTICS 39, No. 11, p. 135 (July 1962)

HYGROPHOTOGRAPHIC STUDIES ON THE PERMEABILITY TO WATER AND MOISTURE OF PACKAGING MATERIALS. J. Sivadjian and F. Corral. J. APPL. POLYMER SCI. 6, No. 23, p. 561 (September 1962)

ISOSTATIC-CHEMICAL ANALYSIS METHOD TESTS FILM'S GAS PERMEABILITY. R. G. Marlett. PACKAGE ENG. 7, No. 10, p. 60 (October 1962)

WATER VAPOR TRANSMISSION THROUGH POROUS FILMS. D. Satas and D. G. Carr. J. APPLIED POLYMER SCI. 6, No. 24, p. S-64 (November/December 1962)

GAS CHROMATOGRAPH MEASURES FILM PERMEABILITY. H. L. Fricke. PACKAGE ENG. 7, No. 12, p. 51 (December, 1962)

47. TOXICITY

See topic 38, Packaging, for general references dealing with toxicity problems.

SKIN TROUBLES FROM TEXTILES. W. Schweisheimer. TEXTILE COLORIST 66, p. 69 (Feb. 1944)

ACUTE AND SUBACUTE TOXICITY OF DI (2-ETHYLHEXYL) PHTHALATE -- ITS METABOLISM. C. B. Shaffer et al. J. INDUS. HYG. & TOX. 27, No. 5, (May 1945)

HEALTH HAZARDS FOR PVC AND ACRYLICS? PLASTICS 12, No. 129, p. 85 (February 1948)

TOXICITY OF O-TRICRESYL PHOSPHATE. O. Blomqvist. SVENSKA PLAST. FOR TEK. MED. 5, No. 1 (1950)

THE TOXICITY OF TRICRESYL PHOSPHATE. N. Nelson. MED. BULL. NEW JERSEY 10, p. 114 (April 1950)

STUDIES ON THE TOXICITY AND SKIN EFFECTS OF COMPOUNDS USED IN THE RUBBER AND PLASTICS INDUSTRIES. II. PLASTICIZERS. F. S. Mallette & E. Von Haam. ARCH. INDUS. HYG. & OCCUP. MED. 6, No. 3, p. 231 (September, 1952)

TOXICITIES OF BUTYL STEARATE, DIBUTYL SEBACATE, DIBUTYL PHTHALATE, AND [illegible] OLEATE. C. C. Smith. ARCH. INDUST. HYG. & OCCUP. MED. 7, p. 310 (April, 1953)

TOXICITY OF 2-ETHYLHEXYL DIPHENYL PHOSPHATE. I. IMMEDIATE TOXICITY AND EFFECTS OF LONG-TERM FEEDING EXPERIMENTS. J. F. Treon, F. R. Deutra & P. F. Cleveland. ARCH. IND. HYG. OCCUPATIONAL MED. 8, p. 170 (August, 1953)

TOXICITY OF 2-ETHYLHEXYL DIPHENYL PHOSPHATE. III. STUDIES OF THE EXTRACTION OF SANTICIZER 141 FROM POLYVINYL CHLORIDE FILMS BY FOODSTUFFS UNDER VARIOUS CONDITIONS OF STORAGE. J. Clark Kaskie. ARCH. IND. HYG. OCCUPATIONAL MED. 8, p. 281 (1953)

OCCUPATIONAL DISEASE IN THE MANUFACTURE OF POLYVINYL CHLORIDE PRODUCTS. (when containing chlorinated naphthalene) J. Roubal & F. Pokorny. PRACOVNI LEKARSTVI 5, p. 144 (1953)

TOXICOLOGY OF PLASTICS AND RUBBER -- PLASTOMERS AND MONOMERS. Rex H. Wilson & Wm. E. McCormick. IND. MED. & SURGERY 23, p. 479 (November, 1954)

FURTHER STUDIES OF POLYMERS AS CARCINOGENIC AGENTS IN ANIMALS. B. S. Oppenheimer et al. CANCER RESEARCH 15, p. 333 (1955)

SOME ASPECTS OF THE TOXICOLOGY OF PLASTICS. R. Forquet. MATERIE PLASTICHE 22, No. 2, p. 100 (February 1956)

CHRONIC ORAL TOXICITY OF 2-ETHYL HEXYL

OLI 5792

was mixed with diphtheria toxin (0.[illegible] ml.) and incubated with antitoxin at 37° for 5-6 days in medium 199. The min. toxin concn. detd. by the metabolic pH-indicator test using phenol red was 10^{-4}-10^{-5} Lf/ml. The test was applied using a range of inoculum sizes, vols., HCO_3^- concn. and antitoxin concn. Variance was 20% compared with 7% in the cytopathogenicity test, but the method was easier and was suitable for serial diln. application. Peter M. Molton

59105g Toxicological investigation of cyanides. Ortega, Manuel; Vallejo, E. (Inst. Nac. Toxicol., Madrid, Spain). *An. Real Acad. Farm.* 1966, 32(4-5), 479-87 (Span). Blood (1-2 ml.) or minced viscera (2-3 g.) was placed in a Conway cell for 4 hrs. with 4-5 drops of 10% H_2SO_4 and HCN evolved was absorbed in 0.1*N* NaOH. To the latter were added 2 ml. *M* NaH_2PO_4 and 1 ml. 0.25% chloramine-T, then after 2-3 min., 3 ml. reagent (3 g. barbituric acid, 15 ml. pyridine, 3 ml. HCl, and water to 50 ml.). The red to reddish purple color was read at 580 mμ after 10 min. Beer's law was obeyed for 0.5-2 γ KCN/ml. With time, cadaver CN^- is converted to CNS^-. To det. this, blood or viscera was deproteinized by adding an equal amt. of H_3PO_4, heating to boiling for 10 min., and adding 50 mg. of powd. picric acid/g. of tissue. After 12-24 hrs. at room temp., it was filtered and 1/3 vol. 5% $K_2Cr_2O_7$ and 2/3 vol. 50% H_2SO_4 were added to the filtrate in a Conway cell. Detn. of CN^- was as above. Recovery of added CNS^- was 54% (7% if not deproteinized). Disappearance of CN postmortem is approx. exponential and the amt. present at death cannot be accurately calcd. from subsequent detns. J. R. Gwilt

59106h Identification of hashish (Cannabis sativa). Chambon, Paul (Fac. Med. Pharm. Lyon, Lyons, Fr.). *Bull. Trav. Soc. Pharm. Lyon* 1968, 12(1), 43-6 (Fr). A sample of hashish was identified using color reactions and thin-layer chromatog. on silica gel G. The principal constituents were cannabinol, cannabidiol, and tetrahydrocannabinol. Dorothy J. Buchanan-Davidson

59107j Identification and determination of nonvolatile organic poisons by infrared spectroscopy in forensic toxicology. Corneteau, H.; Monnet, R.; Boiteau, H. L. (Fac. Mixte Med. Pharm. Nantes, Nantes, Fr.). *Med. Leg. Domm. Corpor.* 1968, 1(4), 352-9 (Fr). Eleven alkaloids and 9 neuroleptic agents were identified and estd. by ir spectrometry after extn. and sepn. by thin-layer chromatog. on silica gel. The chromatogram eluates were concd. on KBr micropellets. Spots contg. 10 γ of compd. could be estd. with a precision of 8-15%. L. A. Dehennin

59108k Utilization of an anti-γ-globulin immune serum for determining the human origin of blood stains. Tran Van Ky, Philippe; Lenoir, L.; Muller, P. (Inst. Med. Legale Lille, Lille, Fr.). *Med. Leg. Domm. Corpor.* 1968, 1(4), 392-5 (Fr). Extn. of a blood spot with isotonic phosphate buffer of pH 7.4 exts. the antigen. Monospecific antisera are obtained from rabbits immunized with human γG-globulins. The immunologic tests are performed by double diffusion on gelose. L. A. Dehennin

59109m Evaluation of postmortem blood sugar levels and their correlation with the glycogen content of the liver. Ramu, M.; Robinson, Ann E.; Camps, F. E. *Med. Sci. Law* 1969, 9, 23-6 (Eng). Satisfactory blood glucose estns. may be made by using blood samples collected into tubes contg. F^- up to 48 hrs. after death, provided that a specific assay procedure is used. The blood glucose concn. for samples from the left side of the heart and the upper and lower extremities statistically belong to 1 group whereas the right heart blood and the liver blood belong, statistically, to 2 different groups. There were no significant variations in the blood glucose concn. with the time interval 12-48 hrs. after death. There is a significant inverse relation between liver blood glucose concns. and the glycogen content of the liver as assayed chem. Irving Sunshine

59110e Relations between the levels of alcohol in the blood and in the breath. Paolucci, G. *Riv. Med. Aeronaut. Spaz.* 1968, 31(3-4), 401-9 (Ital). Tests were made with 20 subjects (2 female) of av. age 35 who consumed, in a single dose, 0.50 g. EtOH/kg. in the form of cognac. Exhaled air was collected every 15 min. up to 2 hrs. after ingestion and blood was sampled at 45 and 120 min. in 15 of the subjects and every 15 min. in the remaining 5. To det. whether differences existed between the air exhaled in 1 profound expiration and alveolar air, the latter was collected in equal vol. immediately after the former. The blood EtOH increased regularly up to 60 min., then decreased irregularly up to 2 hrs. The ratio of EtOH in [illegible] ml. exhaled air to that in 1 ml. blood was about 1:2 for the first hr. after ingestion, varying with the individual. Blood EtOH is detectable up to 2 hrs. in subjects with considerable adipose tissue but has practically disappeared after 1 hr. in lean subjects. J. I. M. Jones

59111f Fractionation analysis of metallic poisons. Krylova, A. N. *Vopr. Sudebnoi Med., Min. Zdravookhr. SSSR* 1968, 314-21 (Russ). From *Ref. Zh., Khim.* 1969, Abstr. No. 2G175. A comparison of the accepted H_2S method and the new fractiona[illegible] is made, with relation to a group of 12 poisons (Pb, Ba, As, Sb, Bi, Hg, Cu, Cd, Ag, Zn, Cr, and Mn). Qual. and quant. anal. methods take 1-2 working days instead of the 8 required for the H_2S method, with greater sensitivity and accuracy. MQRK

59112g Separation of copper and cadmium by extraction in forensic chemical investigations. Krylova, A. N. *Vopr. Sudebnoi Med., Min. Zdravookhr. SSSR* 1968, 322-7 (Russ). From *Ref. Zh., Khim.* 1969, Abstr. No. 2G176. A method of extn. and complexometric detn. of Cu and Cd in human organs is described. Mineralize the sample with HNO_3-H_2SO_4, add 20 ml. 20% K Na tartrate, 10 ml. glycerol (1:10), and 30% KOH until neutral and 5 ml. in excess. Add 10 ml. 1% Na diethyldithiocarbamate (I) and 10-15 ml. $CHCl_3$, shake, and repeat the extn. until a colorless $CHCl_3$ layer is obtained. Reext. Cd from the combined ext. by shaking with 10 ml. *N* HCl (3 times). Dil. the Cd ext. to 100 ml. with H_2O and bring to pH 8. Titrate Cd with 0.01*N* EDTA until the red-violet color changes to dark blue. Wash the $CHCl_3$ ext. 3 times with 10 ml. 5*N* HCl (to remove Bi and Tl) and then with H_2O until neutral reaction. Ext. Cu by shaking with 1% $HgCl_2$ until colorless, and det. in the ext. by titrn. with EDTA in the presence of murexide and 1-2 g. KI at pH 8 (color change from yellow to violet). MQRK

59113h Biochemical modifications induced by acute and subacute intoxication with white phosphorus in the rat. I. Action of repeated parenteral doses. Truhaut, Rene; Claude, Jean R.; Warnet, Jean M. (Fac. Pharm., Paris, Fr.). *Ann. Pharm. Fr.* 1969, 27(1), 17-23 (Fr). Repeated administration of white P to rats, parenterally, decreased body wt. A dose of 2.5 mg./kg. decreased liver wt. without visible steatosis; liver triglycerides were increased and phospholipids decreased, leaving the total lipid content nearly const. In the blood both total lipids and triglycerides were lowered. With 5 mg./kg. several rats died after 7-8 days following the 3rd injection, and all showed varying degrees of liver steatosis with increase in triglycerides and in total lipids. Since the activity of glucose-6-phosphate dehydrogenase remained normal it is unlikely that the biosynthesis of fatty acids and triglycerides was increased. Ruth M. Chester

59114j Toxicity of pyrolysis products of vinyl plastics. Cornish, Herbert H.; Abar, Ellen L. (Sch. of Public Health, Ann Arbor, Mich.). *Arch. Environ. Health* 1969, 19(1), 15-21 (Eng). Poly(vinyl chloride) (I) and vinyl chloride-vinyl acetate polymers (II) were pyrolyzed in air by gradually raising the air temp. from ambient to 600°. Rats were exposed to the pyrolysis products air stream dild. with room air to twice its initial vol. Exposure to air contg. the pyrolyzed products from 1-2 g. I resulted in death to 50% of the animals. Most deaths were due to CO, and carboxyhemoglobin levels correlated well with the amt. of pyrolyzed I. Little histol. evidence of lung damage was evident. When the air stream was dild. with O, pulmonary edema and interstitial hemorrhage developed. The lungs of animals exposed to high levels of II pyrolysis products showed focal edema and intraalveolar hemorrhage. I formulations contg. additives and inert materials were generally less toxic per g. of sample pyrolyzed. BZJN

59115k Validity of a critical blood level for prevention of dieldrin intoxication. Keane, William T., Jr.; Zavon, Mitchell R. (Agr. Chem. Div., Shell Chem. Co., New York, N.Y.). *Arch. Environ. Health* 1969, 19(1), 36-44 (Eng). In dogs given a single dose of 5 mg./kg. of dieldrin, dieldrin concn. in the blood reached a max. in 2-4 hrs. and declined after 10 hrs. to a relatively const. level which persisted for the following 22 hrs. All intoxications should therefore occur within 2-4 hrs. after feeding dieldrin. In dogs given 1.0 mg./kg. of dieldrin for 5 days, 0.2 mg./kg. for the next 57 days, and then 2 mg./kg. daily until intoxication occurred, the 1st muscular spasm occurred at a baseline concn. of 55 μg./100 ml. of whole blood. In both acute and subacute intoxication, the baseline concn. prior to intoxication was 62 μg./100 ml. After administration of the daily dose and subsequent appearance of intoxication, the blood concns. of dieldrin were, resp., 74 and 83 μg./100 ml. blood in the acute and subacute groups. The difference was not significant. Correlation between dieldrin concns. in blood and body fat was 0.81. The results support the hypothesis of a threshold level for dieldrin in blood which, when exceeded, results in intoxication independently of the type or duration of exposure. Raymond Zehnpfennig

59116m Alteration of the locomotor activity of mice poisoned with sublethal doses of lead acetate. Terzin, A. L.; Vujkov, V. V. (Med. Fac., Novi Sad, Yugoslavia). *Arh. Hig. Rada Toksikol.* 1968, 19(4), 4[illegible]-511 (Eng). Injection of $Pb(OAc)_2$ (0.4-20 mg. i.p.) into female mice (21-33 g.) produced dose-dependent changes in locomotor activity. Single doses produced depression of locomotor activity while chronic poisoning increased locomotor activity. Single or multiple i.p. injections of saline did not alter locomotor activity. Animals poisoned with lethal doses of $Pb(OAc)_2$ developed stippled cells during the 2nd or 3rd week while significant changes in locomotor activity were evident 1 day after the last injection. Mice poisoned with sublethal doses (0.4-1.0 mg.) showed no stippled cells but displayed changes in locomotor activity. P. H. Redfern

OLI 5793

... compn. of the ... between HCl ...
... N. Constantsas.

... ization of liver damage caused by ... silymarin as a model of antihepatotoxin therapy. ... Ined. Inst. Madaus, Cologne, ... Forsch. 1969, 19(1), 61-... (Ger). Silymarin, ... present in the seeds of *Silybum marianum*, the toxic effects of phalloidin in mice. Silymarin was ... in reversing the toxic effects of phalloidin in mice ... first with this compd. and then treated with silymarin ... antiphalloidin effect of silymarin depended on the ... between intoxication and treatment, and on the ... liver damage. BQJG

... Evidence for the bioactivation of slaframine. Aust, ... (Michigan State Univ., East Lansing, Mich.). *Biochem. Pharmacol.* 1969, 18(4), 929-32 (Eng). Max. salivary ... in pentobarbital-anesthetized cats occurred ~1 hr. ... administration of slaframine (0.3 mg./kg.); the duration ... was extremely long, usually lasting for 6 hrs. ... dose. In addn. to salivation, most of the exocrine ... specifically stimulated by slaframine. Upon administration of slaframine to cows, sheep, and goats with ... there was also a substantial delay before an increase in pancreatic flow was observed. In mice, a longer delay after i.v. injection of 2 mg./kg. than with i.p. administration of the same dose; this delay was decreased by injection into the portal vein. Delay of the effect of the drug at ... was significantly extended by briefly isolating (with ...) the liver of rats; the effect was completely eliminated ... permanent isolation of the liver. Common inducers of ... metabolizing enzymes consistently reduced the delay ... action of salivation in mice, while inhibitors of the enzymes consistently lengthened the delay. Thus, slaframine ... require activation by the liver before stimulating exocrine ... The enzymes involved in this activation are ... those responsible for the metabolism of most xeno... BYJN

... Metabolism of phospholipids in the brain and liver ... during intoxication with organophosphorus compounds. ... V. Ya.; Tofilo, A. P. (Pavlov Inst. Physiol., Leningrad, USSR). *Byull. Eksp. Biol. Med.* 1969, 67(5), 50-2 (Russ). ...phosphorus compds. LG-63 (5 mg./kg.) and GA-81 (0.4 ...) administered i.m. to rats did not affect the level of ...lipids in the brain and liver or the rate of restoration of ...lipids in the brain. The metabolism of phospholipids ... was increased 24.8% by organophosphorus compd. ... 32.4% by GA-81. The increased intensity of phospholipid metabolism may be connected in some way to increased ... of the hepatic cells during detoxication of the cholinesterase inhibitors. BJJR

... Toxicity studies in mice treated with 1-β-D-arabino...cytosine (ara-C). Leach, William B.; Laster, W. ... Jr.; Mayo, Joseph G.; Griswold, Daniel P., Jr.; ... Frank M., Jr. (Med. Center, Univ. of Alabama, ...am, Ala.). *Cancer Res.* 1969, 29(3), 529-35 (Eng). ... therapeutic dosage schedules of 1-β-D-arabinofuranosylcytosine (ara-C) administered i.p. in mice (15 mg./kg., every ... 24 hrs.) result in karyorrhectic damage to the crypt cells of the intestinal mucosa. This damage is most ... hrs. (earliest observations made) after the end of the ..., after which there is a rapid recovery to completion ... The damage is thus transitory and resembles that of ... This cycle of transitory intestinal damage followed by recovery occurs after each therapeutic dosage schedule as long as the schedule is not repeated more often than ... day. Progressive damage to the intestinal mucosa ... by changes in the hematopoietic tissues occurs ... optimum therapeutic dose (15 mg./kg., every 3 hrs.) ... uninterrupted to the LD_{100} dose (360 mg./kg. in ...) and beyond. Irreversible changes are seen in the intestinal mucosa, and a progressive depopulation of the bone ... with ultimate aplasia ensues as the LD_{100} dose is reached ... RCTT

... Effect of butylated hydroxytoluene (BHT) on rat ...chrome oxidase activity. Pascal, Gerard; Terroine, ... (Centre Rech. Nutr., C.N.R.S., Bellevue, Fr.). *C. R. ... Paris, Ser. D* 1969, 268(11), 1529-31 (Fr). The oral ... of BHT (0.05% in the food, a concn. 50-fold ... that authorized for human food) for 8 weeks caused ... decrease in hepatic cytochrome oxidase activity ... sexes. The addn. of BHT to rat liver homogenate ... of 10 mg. ... liver caused a 12% decrease in ... oxidase activity. Thus, ... appears ... effect on in vivo metabolism. DHJF

... Activity of guanine deaminase in rat kidney, liver, ... during experimental uranyl nitrate intoxication. ... Janine; Bastide, Pierre (Fac. Mixte Med. Pharm., ...ferrand, Fr.). *C. R. Soc. Biol.* 1968, 162(5-6), ... (Fr). Guanine deaminase activity (I) was studied in ... kidney, and blood of rats during exptl. intoxication with uranyl nitrate ... body wt.). ... I was noted in all organs, but there was a definite lowering of I in the kidney for the first 12 hrs. after the start of intoxication.
Dorothy J. Buchanan-Davidson

113458u Excretion of tetraethylgermanium in the rat. ... J.; Clavel, M. J.; Petet, ... (Centre Rech. Toxicol., C.N.R.S., Toulouse, Fr.). *C. R. Soc. Biol.* 1968, 162(5-6), 1226-9 (Fr); cf. G. Petet (1967). Et_4Ge (I), a very resistant compd. in vitro, is degraded very easily in the rat. Although I has a very high b.p., it is eliminated in large amts. during respiration. *The metabolites of I found in rat urine are GeO_2 or the germanates.* The metabolism of I is different from that of tetraethyllead (II) or tetraethyltin (III). No evidence was found for the formation of triethylgermanium, which explains the weak toxicity of I compared to II and III.
Dorothy J. Buchanan-Davidson

113459v α-Hydroxybutyrate dehydrogenase activity of rat kidney homogenate fractions after experimental uranyl nitrate intoxication. Bastide, Janine; Bastide, Pierre (Fac. Mixte Med. Pharm., Clermont-Ferrand, Fr.). *C. R. Soc. Biol.* **1968**, 162(8-9), 1492-3 (Fr). Most of the α-hydroxybutyrate dehydrogenase activity of the rat kidney is found in the supernatant after centrifugation at 24,000 g for 2 hrs. This activity decreased with time during exptl. uranyl nitrate poisoning, whereas the contents of protein, DNA, and RNA were relatively low and underwent but little variation. C. W. Ackerson

113460p Effect of ethyl bromide on the liver. Karimullina, N. K.; Gizatullina, A. A. (Ufim. Nauch.-Issled. Inst. Gig. Profzabol., Ufa, USSR). *Farmakol. Toksikol. (Moscow)* 1969, 32(2), 165-7 (Russ). EtBr fumes (2.4 mg./l.) inhaled by rats and rabbits for 4 hrs. daily for 6 months disrupted the functional ability of the liver, decreased the hepatic glycogen and fat levels, and prolonged hexenal sleep. Granular dystrophy in the hepatic cells and a decreased RNA level in the cytoplasm were detected. BJJR

113461q Hemopoiesis in chronic heliotrine poisoning of Wistar rats. Levin, G. S.; Novachenko, Z. I. (Uzb. Nauch.-Issled. Inst. Gematol. Pereliv. Krovi, USSR). *Farmakol. Toksikol. (Moscow)* 1969, 32(2), 170-1 (Russ). Heliotrine administered s.c. at 3-10 mg./100 g. once a week for 1-8 months to Wistar rats caused hepatic lesions, hypochromic anemia, severe reticulocytosis, normoblastosis, and leukopenia. Erythroblastic sprout hyperplasia, an increased no. of mitoses, and an increased no. of reticular and esp. plasmatic cells were observed. The observed changes in the blood and bone marrow suggest development of an autoimmune form of hemolytic anemia in response to chronic heliotrine poisoning. BJJR

113462r Benzene metabolism in the liver of experimental animals and man. Tiunov, L. A.; Sokolova, T. I.; Bandman, A. L. (USSR). *Farmakol. Toksikol. (Moscow)* 1969, 32(2), 185-8 (Russ). The rate of C_6H_6 metabolism during 24 hrs. of incubation was similar in liver homogenates from humans and rats, more rapid in homogenates from guinea pigs, and slower in rabbit liver homogenates. Species-specific differences seemed to be involved in C_6H_6 conversion, since the activities of aryl-4-hydroxylase and glucuronyltransferase increased while the sulfonating system activity did not change in C_6H_6-treated rats. In C_6H_6-treated rabbits the sulfate adenyltransferase and aryl sulfotransferase activities decreased during disruption of the sulfonating system. The high rate of C_6H_6 conversion in guinea pigs may be connected with the relatively high liver catalase activity. BJJR

113463s Effect of the sodium salt of adenosine triphosphate on experimental cyanide poisoning of rats (mechanism of the antidotal action). Mosketi, K. V.; Ganzhara, P. S. (Odess. Med. Inst., Odessa, USSR). *Farmakol. Toksikol. (Moscow)* 1969, 32(2), 214-15 (Russ). Na ATP (0.5 ml. of a 1% soln.) given s.c. or i.m. to rats 1 hr. prior to administration of the min. lethal dose of NaCN prolonged survival. When given s.c. immediately after lethal cyanide poisoning it substantially extended the life span of 50% and prevented death in the other 50% of the rats. The antidotic effect of ATP may result from its ability to restore respiration by bypassing cyanide-sensitive enzymes. BKJR

113464t Changes in the cardiac activity of rats chronically exposed to vinyl chloride vapors. Vazin, A. N.; Plokhova, E. I. (Gor'k. Nauch.-Issled. Inst. Gig. Tr. Prof. Zabol., Gorki, USSR). *Farmakol. Toksikol. (Moscow)* 1969, 32(2), 220-2 (Russ). Chronic (5 months) exposure of rats to vinyl chloride vapors at 0.03-0.04 mg./l. disrupted cardiac work rhythm, induced bradycardia and arrhythmia, and reduced the relative duration of I-II and T-II sound intervals. The relative duration of the QRS complex did not change significantly. Within 15 days after termination of poisoning, the rat cardiac activity rhythm returned to normal, but the duration of the I-II and T-II sound intervals remained below the initial level for another 15 days. The maximal permissible concn. of vinyl chloride apparently is significantly less than the 0.03 mg./l. level previously estd. BJJR

20 113464t

OLI 5794

daily for 60 days increased both β and γ globulin levels and decreased the albumin to globulin ratio, α-globulin levels were not affected. BEJR

85811h Effect of selenophene on liver and kidneys during carbon tetrachloride intoxication. Zolotarevskii, V. B.; Tugarinova, V. N.; Miklashevskii, V. E. (Mosk. Med. Inst. im. Sechenova, Moscow, USSR). *Farmakol. Toksikol. Prep. Selena, Mater. Simp.* 1967, 87-9 (Russ). Two groups each comprising 10 male rabbits (2-3 kg.) were used. All animals received twice a week s.c. doses of 0.3 ml./kg. of a 40% oil soln. of CCl_4. To one of these groups 0.5 ml./kg. of 1% selenophene soln. was s.c. injected 2 hrs. later. During the 1st 10-12 days of CCl_4 intoxication approx. 60% of the rabbits died in both groups. Selenophene (5000 μg./kg. = approx. 100 μg. % or 1/32 of an LD_{50}) protected kidney tissue against CCl_4 intoxication, but dystrophic and necrotic changes of liver were more extensive and more important in animals to whom a combination of CCl_4 with selenophene was given. Thus, the exptl. dose of selenophene was too great and showed no protective but a destructive effect on the liver, although smaller doses of 2-3 μg. % of Se are necessary to maintain normal liver function. Addn. of 20 μg. % of Se to the normal diet of rabbits prevented dietary liver necrosis in cases of hyperthyroidism. R. Pavlak

85812j Toxicology of some polymers (Povinols). Dvoskin, Ya. G.; Rakhmanina, N. A.; Dem'yanko, F. V.; Men'shikova, T. A.; Erofeeva, L. F.; Lashkina, A. V. (Nauch.-Issled. Inst. Gig. Vod. Transp., Moscow, USSR). *Gig. Sanit.* 1969, 34(1), 7-11 (Russ). Three samples of poly(vinyl chloride) polymers (povinols) contg. di-Bu phthalate (I), tricresyl phosphate, and chlorinated hydrocarbons as plasticizers, released I into the surrounding air in amts. of 1.6-3.73 mg./m.³ in a sealed container and 0.53 during a single air exchange. All 3 samples had unfavorable physiol. effects on exptl. animals and are not recommended for use as finishing materials in shipbuilding. Exposure of rats to I (0.2-8 mg./m.³) for 1-5 days disrupted conditioned reflex activity and nonspecific immunity, and, to a lesser extent, the blood protein compn. and phagocytic index, and the liver wt. I-induced changes were dose-dependent. BJJR

85813k Mechanism of boron effect on warm-blooded animals. Popov, T.; Angelieva, R. (Nauch.-Issled. Inst. Gig., Sofia, Bulg.). *Gig. Sanit.* 1969, 34(1), 78-81 (Russ). Of 60 mg. B given to rats, the distribution was approx. as follows: 50 mg. in the bones, 0.62 mg. in the liver, 0.06 mg. in the kidneys, and 0.1 mg. in the brain. The max. was reached in 12 hrs. in the liver and kidneys, after 24 hrs. in the bones and brain. B was principally excreted in the urine. Complete excretion of the accumulated B required a long time, but the majority was gone in 2 days after reaching the max. in each tissue. John Howe Scott

85814m Effect of ascorbic acid on various signs of vanadium toxicity. Matantseva, E. I. (Inst. Gig. Tr. Profzabol., Sverdlovsk, USSR). *Gig. Tr. Prof. Zabol.* 1968, 12(12), 22-5 (Russ). I.v. administration to dogs (8-16 kg.) of 3, 4, or 5 mg./kg. of ammonium vanadate, or 3-11 mg./kg. of Na metavanadate increased blood inorg. P, glucose, and lactic acid, decreased blood glutathione, and caused salivation, dyspnea, increased respiratory and heart rates, urination, and defecation. Na ascorbate, 10-50 mg./kg. given i.v. simultaneously with, or 10 min. after the administration of V compds., alleviated or prevented the toxic and biochem. effects. One dog given 5 mg./kg. of both ammonium vanadate and Na ascorbate died of lung edema. The severe toxicity of V compds. was attributed to increased vascular permeability and depressed glycolysis and oxidn. S. K. Vanov

85815n Comparative toxicity of some aralkylphenols and their quaternary ammonium salts. Gadzhibalaev, A. A.; Goryaev, M. I.; Dakhno, O. V.; Kanaulova, L. P.; Potapov, S. V.; Slepushev, V. S.; Churakov, V. I.; Dozorova, A. D.; Zharkova, I. I.; Fedrushkova, I. N. (Khim.-Tekhnol. Inst., Chimkent, USSR). *Gig. Tr. Prof. Zabol.* 1968, 12(12), 44-7 (Russ). The phenols *p*- and *o*-hydroxydiphenyldimethylmethane (I and II), *p*- and *o*-hydroxydiphenylmethylmethane (III and IV), the quaternary *N*-[β-[β-(*p*-methylbenzylphenoxy)ethoxy]ethyl]pyridinium chloride (V), and a mixt. (VI) of V and its ortho isomer had in frogs the following LD_{50} values: 334.9, 584.7, 320.0, 126.4, 42.5, and 42.5 mg./kg. V and VI were more toxic, presumably because of better water soly. which permitted better absorption. In mice and frogs II or IV first caused increased motility and convulsions, then marked central nervous system depression. All other compds. caused depression. All compds. applied topically to the skin or in the eyes of rabbits caused local irritation, pain, and necrosis. In rabbits skin absorption was evidenced by the occurrence of toxic signs and death (in 2-3 months) after topical application of 20, 12, and 5% solns. S. K. Vanov

85816p Tolerance to lethal doses of metals in mice pretreated with low doses. Yoshikawa, Hiroshi (Nat. Inst. Ind. Health, Kawasaki, Japan). *Ind. Health (Kawasaki, Jap.)* 1968, 6(1-2), 88-9 (Eng). Rats were pretreated with metal ions at 10% of the challenge dose. Pretreatment decreased the mortality after challenge by Cd, Hg, In, and Pb, had no effect with Mn and Cu and increased mortality in Fe and Zn. When the challenge metal was different from the pretreatment metal, Cd, Hg, In, and Pb developed cross-tolerance. D. Barbara Sankar

85817q Reversible necrosis in striated muscle fibers of the rat after severe intoxication with various cholinesterase inhibitors. Ariens, A. Th.; Cohen, E. M.; Meeter, E.; Wolthuis, O. L. (Med. Biol. Lab., Nat. Def. Res. Organ. T.N.O., Rijswijk, Neth.). *Ind. Med. Surg.* 1968, 37(11), 845-7 (Eng). Albino rats (200 g.) were injected i.v. with just sublethal doses of paraoxon, diisopropyl phosphorofluoridate, or tabun, and sacrificed at various intervals after injection. The diaphragm, intercostal muscles, gastrocnemius-soleus muscle group, and the psoas muscle were dissected, fixed in 10% formalin, and examd. histol. Local necrosis of muscle fibers is caused by the abnormally long-lasting presence of acetylcholine around the end plates. Muscle fiber necrosis may also occur in man after anticholinesterase poisoning. R. R. Rowlands

85818r Chemical Mace: ocular effects in rabbits and monkeys. MacLeod, Ian F. (Med. School, Univ. of Michigan, Ann Arbor, Mich.). *J. Forensic Sci.* 1969, 14(1), 34-47 (Eng). Direct corneal contact with liquid Mace produces lasting opacities and melanosis. Spray exposure resembling that anticipated for actual use of a Mace weapon causes only transitory lesions of the face and eyes with possible sunburnlike sequellae. A Mace weapon should not be aimed directly at the face if it is discharged at less than 6 ft. The propellant does not seem to be responsible for the injuries; they seem to be due to the lacrimator, chloroacetophenone. Irving Sunshine

85819s Cerebral amines and acute hyperbaric oxygen toxicity. Blenkarn, G. Douglas; Schanberg, Saul M.; Saltzman, Herbert A. (Med. Center, Duke Univ., Durham, N.C.). *J. Pharmacol. Exp. Ther.* 1969, 166(2), 346-53 (Eng). Acute central nervous system (CNS) toxicity impedes treatment with hyperbaric O (OHP) for ischemic illness which led to a study of relations between cerebral amines and OHP-induced CNS toxicity in rats after injection of selected drugs. Brain levels of norepinephrine and serotonin were unchanged after 1 hr. of OHP at 4.95 atm. abs. Injection of pargyline (40-60 mg./kg.), a monoamine oxidase (MAO) inhibitor, 30 min. prior to OHP (4.95 atm. abs.) exposure for 2.5 hrs. increased (54%) the latent period before the onset of convulsions, decreased the frequency of convulsions from 92 to 33%, and increased 72-hr. survival from 20 to 59% (n = 66). Iproniazid (25-75 mg./kg.) proved equally effective; isoniazid (50-100 mg./kg.) was ineffective. Although inhibitors of norepinephrine synthesis (α-methyl-*p*-tyrosine, 100 mg./kg.) and of 5-hydroxytryptamine synthesis (DL-*p*-chlorophenylalanine, 300 mg./kg.) lowered resp. amine levels without changing OHP tolerance, the protective action of pargyline remained unaltered. Amine precursors 5-hydroxytryptophan (10-25 mg./kg.) and 3,4-dihydroxyphenylalanine (25-50 mg./kg.) also failed to alter O tolerance. The data indicate that premedication with certain MAO inhibitors can protect rats from OHP-induced CNS toxicity. These studies suggest that both the CNS toxicity induced by hyperbaric O and the protection from toxicity afforded by certain MAO inhibitors are not mediated through alterations in the brain metabolism of norepinephrine and 5-hydroxytryptamine. RCHP

85820k Effect of Coprinus atramentarius on the metabolism of ethanol in mice. Coldwell, Blake B.; Genest, Klaus; Hughes, David William (Res. Lab., Food Drug Dir., Ottawa, Can.). *J. Pharm. Pharmacol.* 1969, 21(3), 176-9 (Eng). The EtOH-sol. material extd. from *C. atramentarius* was more toxic to mice, as measured by the LD_{50} and potentiation of the EtOH-induced sleeping time, than the whole mushroom or the residue remaining after EtOH extn. Also, the EtOH-sol. fraction when fed to mice 4 hrs. before administration of EtOH markedly increased the blood HAc and EtOH levels. RCHQ

85821m Salivary gland regeneration after DL-ethionine poisoning. Ulmansky, Mario; Rubinow, A.; Ungar, Henry (Hadassah Med. Sch., Hebrew Univ., Jerusalem, Israel). *Lab. Invest.* 1969, 20(3), 230-3 (Eng). After ethionine feeding, there is increased structural damage during the first 24 hrs. to the salivary glands of male white rats. Recovery begins in 48 hrs. After 72 hrs., mitotic figures are apparent, and after cessation of ethionine feeding for 2 weeks, morphological and histochem. indicators of the salivary glands are normal. Anna Lee Waldo

85822n Changes induced by cadmium, zinc, and lead in renal and peripheral vascular resistances in the anesthetized rat. Ciampolini, Girolamo; Costantini, S.; Cirla, A. M. (Univ. Milano, Milan, Italy). *Med. Lav.* 1968, 59(8-9), 522-33 (It). Cd at 100-500 μg. into the jugular vein of anesthetized rats produced a drop in the vascular resistance of the renal artery which decreased approx. with dose in magnitude and duration. At the highest dose it was almost absent. The effect on the arterial system below the aortic bifurcation (measured at the carotid) varied. There was an increase in vascular resistance at low dosages and a slight redn. at higher dosages. Zn and Pb ...

OLI 5795

components used in manufacture of the polyvinyls. W. L. Guess, Sol Haberman, D. F. Rowan, R. L. Bower, and John Autian (Univ. of Texas, Austin) *Amer. J. Hosp. Pharm.* 24(9), 494-501(1967)(Eng). The techniques useful in detecting the subtle toxicity of some ingredients used in the manuf. of poly(vinyl chloride) plastics, used in the production of containers and closures for certain biologicals, are discussed. Methods of detection include tissue culture tests, animal expts., and effects on antibody reactions and on washed blood cells. Certain heat stabilizers showed a marked incompatability with the biol. systems used. Reactions with 5 plastisizers were more subtle. The results suggest the need for further research into the relations between various levels of toxicity and potential harm in humans.
H. M. Burlage

20561r Some chemical aspects concerning the occurrence of aflatoxin in foods and feeds. W. D. Raymond (Trop. Prod. Inst., London). *Ann. Ist. Super. Sanita* 3(Pt. 3-4), 289-306 (1967)(Ital). This paper reviews various aspects of the problem aflatoxin in foods and feeds. The agricultural, mycological chem., anal., biochem., toxic, com., and public health aspects of the problem are discussed. The presence of mycotoxins in foods and foods also is summarized. 47 references.
Katherine H. Fisher

20562s The effect of diet on carbon tetrachloride metabolism. A. A. Seawright and A. E. M. McLean (Med. Res. Council Labs., Carshalton, Engl.). *Biochem. J.* 105(3), 1055-60 (1967)(Eng). Blood and liver concns. of CCl_4 were measured at intervals after an oral dose, in rats given stock and protein-free diets. The values did not correlate with the resistance to poisoning found in the rats given protein-free diets. Metabolism of CCl_4 to CO_2 in vivo and in liver microsomal prepns. was depressed in animals given protein-free diets. Rats given a single dose of DDT are highly sensitive to CCl_4 poisoning. The livers of such animals had an increased microsomal protein content and greatly increased microsomal activity in the demethylation of Pyramidon and in the conversion of $^{14}CCl_4$ into $^{14}CO_2$. The incorporation of leucine-^{14}C into protein by liver slices is depressed by CCl_4. This effect is decreased by the addn. of SKF 525A and in slices from rats given protein-free diets. It is suggested that the toxicity of CCl_4 is closely linked to its metabolism.
RCFG

20563t An attempt to influence the collagen defect in experimental lathyrism. K. Trnavsky, Z. Trnavska, B. Skrovina, and L. Cebecauer (Res. Inst. Rheumatic Diseases, Piestany, Czech.). *Biochim. Physiol. Tissu Conjonctif, Conf. Commun. Symp. Int., Lyon* 1965, 663-70(Pub. 1966)(Eng). To study the influence of hydrocortisone (I) and Na salicylate (II) on the soly. of collagen proteins and on changes in the epiphyseal growth plates in lathyric rats, exptl. lathyrism was induced in 36 male rats by feeding a 60% concn. of sweetpea seeds (*Lathyrus odoratus*) for 30 days. Twelve of these rats were simultaneously treated with I (10 mg./kg. body wt. daily), i.m.; another group of 12 were treated with II (250 mg./kg. daily), i.p. The 3 exptl. groups were compared with 3 nonlathyric control groups treated similarly. After 30 days, total hydroxyproline (III), dialyzable III, and acid-sol. III were detd. in dorsal skin exts. For histol. evaluation, the tibial growth plates from decalcified hind limbs were microscopically examd. Total III was not significantly changed in any of the groups studied. I in intact (control) animals resulted in decreased levels of the neutral salt-sol. III and dialyzable III. II had no effect on the sol. fractions. Feeding with sweetpea increased the neutral and acid sol. III; the greatest part of the lathyric neutral sol. fraction being dialyzable. In lathyric rats, I treatment depressed the elevated amts. of sol. III as well as dialyzable III. Even more effective in restoring normal conditions in lathyric rats was II treatment. Histol., the cells of the epiphyseal plate in lathyric animals showed elevated mitotic activity but delayed maturation. The synthesis of ground substance in the intercellular spaces was also disturbed with demasked collagen fibers, granular decompn., and some cystic areas. I depressed lathyric toxicity, while II restored the tibial growth plate to nearly normal. 19 references.
L. K. Allen

20564u Toxicity of 1,2-dichloroethane-extracted fish protein concentrate. I. C. Munro and A. B. Morrison (Food Drug Res. Labs., Dept. Natl. Health Welfare, Ottawa). *Can. J. Biochem.* 45(11), 1779-81(1967)(Eng); cf. *CA* 67: 52812d. Wistar rats were fed a protein-free diet with 50% protein from lyophilized or extd. cod fish (lyophilized cod extd. for 24 hrs. with iso-PrOH or 1,2-dichloroethane (I), then heated for 2 hrs. at 60° in vacuo) which contained 40 mg. % chlorocholine chloride II. Cod fillets extd. with I were toxic to the growing rat. The toxicity was not due to II but was concd. in a MeOH ext. of the I-treated fish. However the MeOH-extd. fish did not support growth even when supplemented with cystine and histidine.
Dorothy J. Buchanan-Davidson

20565v Significance of carbon monoxide from cigaret smoking inside a car. Milan Srch (Karlova Univ., Hradec-Kralove, Czech.). *Deut. Z. Gesamte Gerichtl. Med.* 60(3), 89-9(1967) (Ger). A forensic opinion from the author was needed in the case of a 30 year old woman who had died in a closed car in a garage. In a test on 4 persons, 2 of them smokers and 2 nonsmokers, who sat in a closed car with a cubature of 2 m and smoked 5 cigarets with inhalation during 60 min., the hemoglobin concn. in the blood of the smokers rose from ... 10%, that in the blood of the non-smokers from 2 to 5%. ... such circumstances a fatal intoxication was deemed not po... Consequences for traffic safety are discussed. D. H. Ste...

20566w Histoautoradiographic studies on mesenchymal ... duct cells of thioacetamide-induced cirrhosis of the liver in ... Balthasar Wohlgemuth (Karl-Marx-Univ., Leipzig, ... *Exp. Pathol.* 1(1), 64-71(1967)(Ger). White mice (19-2... were fed 0.25-0.45 mg. thioacetamide (I) in the drinking w... Controls without I were treated further by the same proced... After 70 days the animals were injected i.p. with 3 μci. of th...dine (II)-^3H/g. body wt. After this the animals were k... and the livers were fixed in HCHO; sections (5 μ) were wa... with inactive II to remove unincorporated II-^3H, and autor... grams were exposed for 4, 6, 8, and 19 days at 15°. Af... days the development of cirrhosis had proceeded widely ... treated animals. The parenchyma was penetrated by ret... and collagenous fibrillar structures as well as with mesench... and duct cells, which increased in relative no. The incor... tion rate of II-^3H into hepatocytes increased from 0.03 (con... to 0.24% in exptl. animals; in Kupffer's cells the resp. lab... indexes were 1.67 and 2.41%. Labeling of hepatic capsul... endothelial cells or of the bile duct epithelium showed no ... ference between controls and I-treated animals. The duct ... exhibited a labeling rate similar to that of Kupffer's cells ... treated animals. As early as 1 hr. after II application, black... mitotic figures could be seen in Kupffer's cells, both in con... and in I-treated animals. A possible shortening of th... phase due to unexplained reasons was considered. 34 refere...
E. Talafa...

20567x Histamine level and histamine fixation in the ... tissues and skin in rats under the effect of industrial pois... R. S. Eizengart (Sanepidst., Moscow). *Farmakol. Tok...* 30(5), 608-9(1967)(Russ). Homogenates from rat brain ... from rat skin were tested chromatographically for hista... concn. and histamine fixation after rats had been exposed ... to threshold concns. or to toxic doses of methylcyclohe... ethylene sulfide, diethylaminoethyl mercaptan, or 2-meth... vinylpyridine. At threshold levels there was less hista... fixation in skin and more in brain tissue. After toxic d... the changes were irregular both in concn. and in hista... fixation. Julian F. Sm...

20568y The toxic peptides of Amanita phalloides. Th... Wieland (Goethe-Univ., Frankfurt/M., Ger.). *Fortschr. C... Org. Naturst.* 25, 214-50(1967)(Eng). A review is given; ... mushroom poisoning is almost exclusively attributable to ... bers of the genus *Amanita*. Their toxins are completely ... sorbed from the gastrointestinal tract, while their acti... primarily on the liver; no specific therapeutic agent is know... present. Thus, the understanding of the chem. structure... the mechanism of the action of the toxins is of great impor... from the therapeutic standpoint. Isolation of the toxi... gredients, the chem. structure of the toxic peptides, the ... loidin and the amanitin group, and synthetic expts. with a... acids and cyclopeptides are covered. 86 references. CS...

20569z Comparative toxicity of mercaptobenzimidazole ... its zinc salt. N. V. Mezentseva. *Gig. Otsenka Khim. Fa... Vnesh. Sredy* 1966, 119-20(Russ). Mercaptobenzimidazo... and its Zn salt (II) were studied in acute (mice), subacute (... and chronic (rabbits) expts. LD_{50} internally for mice was ... for I and 737 mg./kg. for II. The animals were exposed ... and II dust for 2 hrs. daily for 15 days in concns. of 400 ... m.^{3} A lag in wt., increase in the content of amino N in ... urine, and disturbances in the nervous system were obser... Morphological alterations were found only in the lung tissue ... bronchitis with slight atrophy of the bronchial mucosa) ... chronic expts. on rabbits I caused disorder in the protein-f... ing function of the liver. II increased the lipid content ... blood. I increased the aldolase content, and II decrease... cholinesterase activity of the blood. I decreased the erythr... count in the blood to 20,000. A proliferative reaction was ... in the lungs; edema and fatty dystrophy were observed i... liver, and punctate hemorrhages were observed in the ... cardium. II caused slight edema in parenchymatous or... I had no irritating effect on the skin or mucosa of the ... whereas II caused moderate irritation. From *Ref. Zh., Far... Khimoterap. Sredstva, Toksikol.* 1967, Abstr. No. 7.54.861.
MV...

20570t Methods for the qualitative and quantitative ... termination of aflatoxins. E. H. Marth (Univ. of Wis... Madison). *J. Milk Food Technol.* 30(10), 317-20(1967)... Qual. and quant. measurements of aflatoxins by means of ... layer chromatog. can be improved by substituting a de... metric "reading" of the thin-layer plates for visual ... Examn. of small samples (down to 0.3 mg.) can be accomp... by a thin-layer chromatog. procedure which employs a ...

OLI 5796

wick, N.J.). *Can. J. Physiol. Pharmacol.* 1968, 46(4), 609-16 (Eng). A series of expts. has shown that the mechanisms by which the metabolism of EtOH occur are strongly influenced by the partial pressures of the O present in the aerating medium. For the most frequently used gas mixt., 95% O_2 + 5% CO_2, the EtOH consumption was abnormally large, and proportional to the concn. of EtOH in the perfusate. However, when the system was aerated with 18% O_2 + 5% CO_2 + 77% N_2, the consumption of EtOH was similar to that found in the intact rat. The perfusate concn. of Me_2CO, HOAc, pyruvic acid, and lactic acid was measured in all expts. When EtOH was consumed at these two different rates, increases in the lactic:pyruvic ratio and acetate levels of the perfusate were noted in both types of expts. However, the utilization of glucose by isolated perfused liver was inhibited, and acetone levels increased markedly, when EtOH was consumed at abnormally rapid rates, although no effect was noted in the perfusate levels of these metabolites when EtOH was consumed at normal rates. 27 references. RCYD

50591p Action of beryllium ions on primary cultures of swine cells. Vegni Talluri, Maria; Guiggani, Viviana (Univ. Siena, Siena, Italy). *Caryologia* 1967, 20(4), 355-67 (Eng). The effect of Be^{++} poisoning on mitosis and morphology of swine kidney cell and lymphocyte cultures was studied. Treatment with Be^{++} had a harmful effect on all the mitotic phases and abnormality of chromosomes was frequent. At high concns. the action of Be^{++} was less pronounced on kidney cells than on lymphocytes. Be^{++} exerts its toxic action by competition with Mg^{++} either in the activation of DNA polymerase or in the binding of DNA to histones. L. Mirone

50592q Protective effect of ethyl alcohol towards allyl alcohol-induced hepatic lesions. Schwarzmann, V.; Infante, R.; Raisonnier, A.; Caroli, J. (CHU Saint-Antoine, Paris, Fr.). *C. R. Soc. Biol.* 1967, 161(12), 2425-9 (Fr). A 90-min. perfusion of isolated rat liver with blood contg. allyl alc. (0.018 ml./180 ml.) caused a release of lactic dehydrogenase, malic dehydrogenase, and glutamic-pyruvic transaminase into the perfusate and a simultaneous depletion of these enzymes from the liver; alc. dehydrogenase and glutamic dehydrogenase activities were not detected in the perfusate and were not decreased in the liver. Biliary secretion was irreversibly arrested after 18-20 min. of perfusion, and hepatic lesions occurred. A 20-min. perfusion with blood contg. EtOH prior to the treatment with allyl alc. prevented the enzyme release, the hepatic lesions and the effect on biliary secretion, and did not cause the release of alc. dehydrogenase or glutamic dehydrogenase activities. BPJF

50593r Effect of ethanol on hepatic metabolism and enzyme activities. Nelson, Patricia Ann (Indiana Univ., Bloomington, Indiana). 1967, 104 pp. (Eng). Avail. Univ. Microfilms, Ann Arbor, Mich., Order No. 68-7228. From *Diss. Abstr. B* 1968, 28(11), 4688. SNDC

50594s Energy-linked calcium transport in liver mitochondria during carbon tetrachloride intoxication. Carafoli, Ernesto; Tiozzo, Roberta (Univ. Modena, Modena, Italy). *Exp. Mol. Pathol.* 1968, 9(1), 131-40 (Eng). In rats intoxicated by the gastric intubation of 0.5 ml. CCl_4/200 g., Ca^{2+} concn. in liver mitochondria increased ~10-fold within 18-20 hrs. However, i.p. injected ^{45}Ca was not taken up by hepatic mitochondria in intoxicated rats as rapidly as in normal animals. In vitro, hepatic mitochondria from CCl_4-intoxicated rats did not show increased Ca^{2+} uptake, but Ca^{2+} release induced by oxidative phosphorylation uncouplers was greatly decreased. This suggested that in CCl_4 intoxication, the balance between ADP phosphorylation and Ca^{2+} uptake is shifted in favor of the latter, thus contributing to the accumulation of Ca^{2+} in mitochondria. DDJN

50595t Protective effects of alpha-tocopherol on the hepatotoxicity of carbon tetrachloride: an electron microscope study. Meldolesi, Jacopo (Inst. Pharmacol., Univ. Milan, Milan, Italy). *Exp. Mol. Pathol.* 1968, 9(1), 141-7 (Eng). The oral administration of 2.5 ml./kg. CCl_4 dild. 1:2 with liq. paraffin to rats produced severe hepatic lesions within 24 hrs. However, liver damage was markedly reduced in rats i.p. injected with the lipid antioxidant, α-tocopherol succinate (125 mg./kg.), before CCl_4 administration. This suggested that lipid peroxidn. is the primary mechanism of CCl_4 hepatotoxicity. 27 references. DDJN

50596u Distribution and toxicity of aliphatic hydrocarbons in body tissues. Shugaev, B. B. (Yaroslav. Med. Inst., Yaroslavl, USSR). *Farmakol. Toksikol.* 1968, 31(3), 360-3 (Russ). Gas chromatog. was used to study the distribution of 6 volatile hydrocarbons in the mouse and rat body tissues. The volatile hydrocarbon content of the brain and ... was similar, but the level in the fatty tissue was significantly higher than in the brain, liver, kidneys, or spleen. There was a parallel between the toxicity and effective cerebral concn. of isoprene, divinyl, and isobutylene, but not of butane, 2-methylpent-1-ene, or 2-methylpent-2-ene. The divinyl concn. was higher in the medulla oblongata than in the cerebellum or cerebral cortex. BJJR

50597v Some biochemical changes in animals following acute polyethylene polyamine poisoning. Solomiskaya, E. A. (Nauch.-Issled. Inst. Onkol., Leningrad, USSR). *Farmakol. Tok* 1968, 31(3), 361-5 (Russ). Polyethylene polyamine (750 ... kg.) administered s.c. to rats and mice increased the ... monoamine oxidase and histaminase activities, decreasing tyramine and histamine levels in the blood and organs of poisoned animals. The pathol. disturbances observed in ... and rats with acute polyethylene polyamine poisoning may ... due to the increased oxidative deamination of endogenous am... with the consequent formation of aldehydes, NH_3, and H_2O ... BJJ...

50598w Effect of niacin on phagocytosis in chronic trini... toluene poisoning. Pidemskii, E. L.; Chukichev, E. ...; Mul'menko, A. M. (Perm. Univ., Perm, USSR). *Farma... Toksikol.* 1968, 31(3), 365-6 (Russ). Trinitrotoluene (T... administered orally at 30 mg./kg. daily for 6 days to rats p... gressively decreased phagocytosis. Niacin administered sim... taneously at 1 mg./kg. s.c. prevented this decrease and even ... creased the phagocytic activity of the leukocytes to 1.5-times ... level in control rats. BJJ...

50599x Pathogenic effect of chronic exposure to vinyl chlor... on rabbits. Vazin, A. N.; Plokhova, E. I. (Inst. Gig. Tr. Pr... zabol., Gorki, USSR). *Farmakol. Toksikol.* 1968, 31(3), 3... 72 (Russ). In rabbits exposed to vinyl chloride (9-10 mg ... air) for 4 hrs. daily for 5.5 months altered β-waves (frequen... >80 Hz.) appeared on the electroencephalogram from the ... terior hypothalamic nuclei, and the potentials of the ante... and posterior nuclei increased by 18-30 and 70-85%, resp., ov... control values. Concomitant changes in the cardiovascular sy... tem (bradycardia, arrhythmia, decreased voltage of the ... dividual electrocardiogram peaks or whole complexes, decrea... duration of systole, increased arterial pressure, and retard... blood flow) also occurred. The functional changes in the a... terior and posterior hypothalamic nuclei may have a role in t... pathogenesis of toxic angioneurosis due to vinyl chloride. BJJR

50600r Brown FK. III. Administration of high doses ... rats and mice. Grasso, P.; Gaunt, I. F.; Hall, D. E.; Golber... L.; Batstone, Elizabeth (Brit. Ind. Biol. Res. Assoc., Carsha... ton, Engl.). *Food Cosmet. Toxicol.* 1968, 6(1), 1-11 (En... Acute oral toxicity studies with Brown FK gave LD_{50} values > ... g./kg. and 8 g./kg. in mice and rats, resp. The values for i... injection were 1.5-2 g./kg. in mice and 0.75-1.15 g./kg. in rat... When repeated daily oral doses ranging from 0.1 to 2 g./kg. we... given to rats, a precipitous wt. loss and a vacuolar myopath... of the heart and skeletal muscles accompanied by lipofusc... formation occurred with doses of 0.5 g./kg. and over. At t... highest dose employed (2 g./kg.), there was also hepatic centr... lobular necrosis and renal tubular degeneration. Pretreatmen... with antibiotics reduced the incidence of muscle lesions. Re... peated i.p. injection did not have any effect on the heart or skele... muscle. Only 2 of the 6 components of Brown FK, 2,4-diamin... 5-(p-sulfophenylazo)toluene (I) and 1,3-diamino-4-(p-sulf... phenylazo)benzene (II), produced muscle damage after repeate... oral doses of 0.5 g./kg. Comparable doses of mixts. contg. t... other components as well as I or II were considerably less toxic. After daily oral doses of 1 g./kg., muscle lesions were found ... rats, rabbits, and guinea-pigs, but none were seen in mice o... hamsters. The results suggest that the intestinal micoflora ... responsible for the breakdown of the components of Brown FK to toxic products. Variations between species and between ani... mals of the same species may be due to qual. and quant. di... ferences in the intestinal microbial population. RCNP

50601s Brown FK. IV. Cytopathic effects of Brown F... on cardiac and skeletal muscle in the rat. Grasso, P.; Muir, A.; Golberg, L.; Batstone, Elizabeth (Brit. Ind. Biol. Res. Assoc., Carshalton, Engl.). *Food Cosmet. Toxicol.* 1968, 6(1), 13-2... (Eng). Administration of 2 or 3 massive (1 g./kg.) oral dos... of Brown FK to rats induced a myopathy in cardiac and skelet... muscles characterized by multiple vacuoles ~1-2 μ in diam... Ultrastructurally, these consisted of areas of fibrillolysis, a... fecting initially the A-band. Histochem., the myopathy wa... accompanied by a moderate increase in acid phosphatase activity and by a loss of phosphorylase activity. Subsequently, complet... lysis of the affected fibers ensued. In the heart, lysis was fol... lowed by macrophage invasion and fibroblastic proliferation and in skeletal muscle, by regeneration. The occurrence o... lipofuscin in muscle fibers and in macrophages was scanty an... erratic. When Brown FK was given in the diet at a level of 2%, fibrillolysis and an increase in the no. and electron d. of lyso... somes was observed ultrastructurally during week 2-3 of t... test. These changes were accompanied by a marked elevat... of histochem. demonstrable acid phosphatase. Progressiv... deposition of lipofuscin was the principal pathological featur... during week 3-12. Initial damage to the A- and I-bands in bot... cardiac and skeletal muscle distinguishes the Brown FK myop... athy from that known to be produced by high doses of corti... costeroids, thyroxine, chloroquine, or plasmocid, from ischemi... damage and from muscle damage resulting from deprivation o... K, Ca, or Mg. The lysosomal changes and accumulation of lipofuscin are suggestive of primary lysosomal damage whic...

... which are analogous to those observed in individual ... ephthera. Vladimir Kostka

See also: Mammalian Pathological Biochemistry, Section 12. ... synthesis of rat immunoglobulin: cell-free system for ... synthesis prepared from lymph node microsomal vesicles ... 2) 18658j. Immunochem. studies on Salmonella: detn. of ... of specific polysaccharide of Salmonella strasbourg, ... Salmonella (Sect. 2) 18790f. Topochem. arrangement ... glycosides in erythrocyte membrane (Sect. 2) 18808h. ... rative study of reaction of heparin with hexammine cobalt ... and lysozyme (Sect. 2) 18724c. Ascorbic acid oxidase ... from different plant materials (Sect. 3) 19064z. Effect ... mone prepn. on protein fractions of immunized animal ... (Sect. 4) 19263p. Changes in body compn. during long-... treatment with cortisone and anabolic steroids in asthmatic ... (Sect. 4) 19188t. Simplified batch method for isolation ... glycoproteins from normal and pathological serums and ... (Sect. 6) 19409r. Biol. activities of polyelectrolytically ... macromols. (Sect. 8) 19711h. Immunoelectrophoretic ... ysis of antigenic components of Bacillus megaterium (Sect. 8) 19757h. Sepn. and characterization of cell adhesive type ... components (Sect. 8) 19716p. Structure and kinetics of formation of antibody in turtle (Sect. 9) 19946p. Difference in proteolytic activity of spleen cells of young and immunologically mature rabbits (Sect. 11) 20213d. Corticosteroid-binding globulin: biochemistry, physiology, and phylogeny (Sect. 11) 20107z. Detection of embryo-specific globulins in serum of embryos and fullterm human infants (Sect. 11) 20150f. Comparative immunoglobulin detns. in human serum (Sect. 11) 20178w. Sensitivity to some fungal allergens during penicillin prepn. (Sect. 14) 20587d. Role of histamine in production of immediate reactions of wheal and angioneurotic type (Sect. 15) 20554p. Effect of some ganglion-blocking prepns. on development and course of anaphylactic shock (Sect. 15) 20830c. Influence of chloramphenicol in immune reactions in rats (Sect. 15) 20800z. Methodology and trends in study and prevention of air pollution by chem. allergens (Sect. 59) 15779p. Antigen with nucleoside determinant (Sect. 63) 16099d. Fractionation of significant quantities of biol. active prepns. by prolonged preparative paper electrophoresis (Sect. 2) 18711w. Response of lymphocytes to penicillin; comparison with skin tests and circulating antibodies in man (Sect. 15) 20706s.

14—TOXICOLOGY

T. R. TORKELSON

...546q **Chemical analysis of aflatoxin.** R. Monacelli and M. Manzone (Inst. Super. Sanita, Rome). *Ann. Ist. Super. ...* 3(Pt. 3–4), 316–26(1967)(Ital). A review with 23 references. Antonio A. Rizzoli

...547r **Toxicity of mercury vapor.** G. A. Neville. *Can. ... Educ.* 3(1), 4–7(1967)(Eng). A review is given of Hg ...city, covering its history, threshold limits, mode of absorption and distribution, and treatments for Hg poisoning. 33 references. CRJN

...548s **Fluorocarbon toxicity and biological action.** J. Wesley Clayton, Jr. (E. I. du Pont de Nemours and Co., Wilmington, Del.). *Fluorine Chem. Rev.* 1(2), 197–252(1967)(Eng). This extensive review summarizes the known toxicology of the fluoroalkanes, the fluoroalkenes, and the fluoropolymers, and discusses various aspects of their biol. action. Much more information is needed on the mechanism of action of these substances. Understanding their mode of action in relation to their chem. constitution should be a governing factor in further investigations of fluorocarbon toxicity. 57 references. M. M. Judge

...549t **The effect of fluorides on livestock, with particular reference to cattle.** James L. Shupe and Ernest W. Alther (... Agr. Res. Serv., Logan, Utah). *Handbuch Exp. Pharmakol.* 20(1), 307–54(1966)(Eng). A review of the effects of fluorides on sheep, swine, horses, turkeys, and, esp. cattle. The effects of fluorosis in cattle on teeth, bones, hair coat, skin, ..., soft tissue, kidney, liver, reproduction, blood, placental transfer, milk production, digestion, metabolism, and enzymes were discussed. A comprehensive guide for diagnosing and evaluating fluorosis was given. 89 references. Patricia W. Heenan

...550m **The mammalian toxicology of organic compounds containing fluorine.** J. Wesley Clayton, Jr. *Handbuch Exp. Pharmakol.* 20(1), 459–500(1966)(Eng). A review of mammalian toxicology, history, and uses of fluorocarbons, including discussion of the toxicology of fluoroalkanes, fluorocycloalkanes, fluoroalkenes, fluoroalcohols, and fluoroaromatic ..., their acute and chronic inhalation toxicities, acute oral, ... or eye toxicities, and their biol. activities. 72 references. Patricia W. Heenan

...551n **Botulism.** Anton Skulberg (Forskningsutvalget ... ..., Oslo, Norway). *Norsk Pelsdyrblad* 41(8), 259–61 ...(Norweg). A review of the subject in connection with ... cases of poisoning of mink in Telemark, Norway, due to [Clostridium] *botulinum* toxin in the mink feed. Vallborg Aschehoug

...552p Botulism in mink. Steinar Hauge, Anton Skulberg, ... Grini (Norges Veterinaerhoegskole, Oslo, Norway). ... *Pelsdyrblad* 41(18), 510–11(1967)(Norweg). A review ... subject in connection with the recent cases of poisoning in ... farms with *Cl[ostridium] botulinum* in Telemark and Aust-..., Norway. As mink is an animal very sensitive to *C. ...num* toxin, vaccination of the puppies should be recommended as a prophylactic means. Vallborg Aschehoug

... Short term chemical danger. Georges Le Moan (..., Paris). *Pharm. Fr.* 1967(15), 199–50(Fr). ... is given of the hazards of explosion, poisoning, etc., ... of the precautions that should be taken against them. ... references. George M. Hocking

...554t **Neurotoxicity of anticholinesterase agents. Antagonistic action of various centrally acting drugs.** G. Bignami ... L. Gatti (Ist. Super. Sanita, Rome). *Proc. Eur. Soc. Study Drug Toxicity* 8, 93–106(1967)(Eng). The report summarizes the behavioral effects of anticholinesterases, and evaluates the usefulness of behavioral methods for toxicological investigations with Para-oxon, parathion, Chlorthion, Systox phosphamide, physostigmine, and galanthamine. 45 references. P. S. Sarin

20555s **Neurotoxic side effects of certain organophosphorus compounds.** W. N. Aldridge and J. M. Barnes (Med. Council Labs., Carshalton, Engl.). *Proc. Eur. Soc. Study Drug Toxicity* 8, 102–8(1967)(Eng). A review of delayed neurotoxic effects of some organophosphorous anticholinesterases. 20 references. Jenifer A. Edwards

20556t **Biochemical and physiological causes of the toxicity of alcohol.** Ryszard Wiktor Schramm and Halina Schrammowa (Univ. A. Mickiewicza, Poznan, Poland). *Psychiat. Pol.* 1(3), 343–7(1967)(Pol). A review with 47 references. Y. Pomeranz

20557u **In vivo deiodination of Rose Bengal-^{131}I in rats after administration of CCl_4.** H. Bergner (Humboldt-Univ., Berlin). *Acta Biol. Med. Ger.* 16, 294–9(1966)(Ger). About double the amt. of ^{131}I-labeled Rose Bengal was deiodinated in vivo in rats intoxicated by 0.1 ml. CCl_4/100 g. as compared with controls. With a protein-free diet, the effect was smaller and could not be detected after 6 days. From *CZ* 1967(30), Abstr. No. 1564. MZCR

20558v **Effect of acute and chronic hepatitis and of fatty liver degeneration on erythropoiesis.** B. Sari, S. V. Kirilina, Sh. Dan, and L. Oros. *Acta Conv. Med. Internae Hung., 3rd., Budapest* 1965, 923–8(Russ). Rabbits were orally given 3 doses of 0.25 mg. CCl_4/kg. and then i.m. 3 doses of 15 mg. phenylhydrazine (I)/kg. Consequently, plasma enzymes (alk. phosphatase, aldolase, glutamic-oxalacetic transaminase) increased, but erythrocyte enzymes did not change or slightly decreased. After a few days, serum enzymes normalized and administration of I did not further alter their level. At the same time, erythrocyte enzymes increased in activity due to I and the increase was correlated with the no. of reticulocytes. Later, these enzymes normalized as well, with the exception of glucose-6-phosphate dehydrogenase and aldolase, whose increase outlasted normalization of the reticulocytes, indicating the presence of a large no. of young erythrocytes in the peripheral blood. These changes were similar to those in erythrocyte enzymes in animals given only I without CCl_4; it is concluded, therefore, that a slight acute injury to the liver does not affect erythropoiesis. Chronic injury decreases erythrocyte survival time and accelerates erythropoiesis. M. Kalab

20559w Poisoning with organic mercury compounds in the Niigata prefecture along the Agano river. I. Detection of methylmercury compounds in hair of patients by gas chromatography. Yukio Takizawa and Takao Kosaka Fak., (Med. Univ. Niigata, Japan). *Acta Med. Biol.* (Niigata) 14(3), 153–61 (1966)(Ger). Eight patients exhibited poisoning attributable to organomercury compds. Hair from the patients was treated with detergent, water, HCl, $CHCl_3$, and dithizone before anal. by gas chromatog. was carried out with a Shimazu GC-1C app. with electron capture detector (ECD-IA) with the conditions: column 1.5 m., inner diam. approx. 3 mm.; column temp. 190–5°, 15 or 25°; diethylene glycol succinate on 60–80 mesh Simalite; N carrier gas at 35 ml./min.; detector temp. 200°; sample size 0.5 μl. Sepn. of methyl-, ethyl-, and butylmercury chlorides was achieved with the ECD-IA detector, but not by flame ionization. D. S. Plaut

~~20560x Characterization of subtle toxicity of certain plastic~~ X

68 20560x

OLI 5798

oxidn. of DPNH which results from alc. metabolism inhibits oxidn. of fatty acids, acetyl-CoA generation, and subsequently gluconeogenesis. 24 references. FHJN

1699a Determination of the toxicity of quercimeritrin, its aglucone, and the amount of flavonoids obtained from cotton flowers. D. E. Tuniyants and L. A. El'kind (Tashkentsk. Gos. Medinst., Tashkent). *Dokl. Akad. Nauk Uzb. SSR* 24 (8), 50-1(1967)(Russ). The toxicities of quercetin 7-glucoside (I), its aglucone quercetin (II), and flavonoids (III) extd. from cotton flower (108-F) were studied in 5 groups of 6 mice. Preps. were given perorally in 1 ml. of distd. water daily for 12 days. II was administered in a dose of 1 g./kg. body wt., doses of I and III were not given. The av. increase in wt. was detd. Then the animals were decapitated and internal organs were studied histol. No morphological changes were noted in the brain, lungs, heart, stomach, pancreas, liver, kidneys, adrenal glands, and gonads of animals administered I, II, or III, except protein dystrophy observed in the liver and adrenal gland cortex zone of some exptl. as well as control animals. F. Vitek

1700u Effect of small concentrations of thiophene and chloroisocyanates on the ascorbic acid content in rat organs. N. N. Pushkina, I. A. Zibireva, and Sh. S. Khikmatullaeva (Inst. Obshch. Kommun. Gig. im. Sysina, Moscow). *Gig. Sanit.* 32(10), 103-5(1967)(Russ). Inhalation of thiophene (3 or 20 mg./m.^{3}), *p*-chlorophenylisocyanate (0.03 mg./m.^{3}), or *m*-chlorophenylisocyanate (0.1 mg./m.^{3}) by rats over an 80-day period decreased the adrenal, kidney, liver, and brain ascorbic acid (vitamin C) levels. This decrease was accompanied by dehydroascorbic acid and diketogulonic acid accumulation in the liver and increased urinary excretion of 17-keto steroids. BJJR

1701v An experimental study in dogs on the usefulness of mannitol, fursemide, and methyl phenidate in phenobarbital intoxication. S. V. Gokhale, K. K. Gudibanda, D. G. Patel, and U. K. Sheth (Seth G. S. Med. Coll., Bombay). *Indian J. Med. Res.* 55(10), 1107-20(1967)(Eng). Acute intoxication by phenobarbital (I) (100 mg./kg., i.p.) was studied in dogs. One hr. after I, i.v. infusion of 20% mannitol (II) (375 ml. in 110-30 min.) or i.v. injection of fursemide (III) (20 mg.) increased the I excretion to 13.58% and 5.8%, resp., as compared to 1.78% of the injected dose in controls. A combination of II and III did not act synergistically. II (20%) in 3 infusions of 375 ml. interspersed with isotonic saline (500 ml.) caused an excretion of I of 47.7%. Saline alone was less effective than II. Methyl phenidate, 10 mg./kg. i.v. 1, 3, and 5 hrs. after I increased the urinary excretion to 5.2%. This was thought to be due to high blood pressure and hyperventilation produced by methyl phenidate. 27 references. FHJN

1702w Toxicity of dimethyl sulfoxide (DMSO) to fish. Wayne A. Willford (Bur. of Sport Fisheries & Wildlife, La Crosse, Wis.). *Invest. Fish. Contr.* No. 20, 8 pp.(1967)(Eng). Toxicities of DMSO to rainbow trout, brook trout, lake trout, carp, black bullhead, channel catfish, green sunfish, bluegill and yellow perch were detd. in 24-, 48-, and 96-hr. static bioassays at 12°. Toxicity was observed around 30 parts/thousand and was not seriously affected by water quality, although increased temp. increased the toxicity to rainbow trout. A preliminary test indicated that DMSO has little effect on the toxicity of antimycin to bluegill. S. Allen

1703x Accidental organic phosphorus poisoning: the use of propranolol to counteract vagolytic cardiac effects of atropine. A. Valero and D. Golan (Med. Dept. B, Rambam Govt. Hosp., Haifa). *Israel J. Med. Sci.* 3(4), 582-4(1967)(Eng). The shortening of the time for coronary filling caused by the excessive tachycardia resulting from atropine administration after org. P poisoning has serious deleterious effects on the heart in the presence of hypoxia. The patient, a young healthy woman, demonstrated electrocardiogram changes after one day of sinus tachycardia at 150-180 beats/min. Shortly after injection of 5 mg. of propranolol, the electrocardiogram reverted to normal. This procedure was extremely safe, even in the presence of heart disease. Independent of the slowing of the heart rate it is thought that propranolol also has specific antiarrhythmic action, making this drug dually beneficial in counteracting the vagolytic effects of atropine and in guarding against ventricular fibrillation. Mahmoud A. Fayez

1704y Organic phosphate intoxication. Wendell L. Fairbanks and Kenneth C. Hoffman. *Neb. State Med. J.* 52(8), 376-9(1967)(Eng). A discussion of 2 cases of org. phosphate poisoning, emphasizing the role of cholinesterases in resistance and of atropine and the oxime drugs in treatment. The mechanism of toxicity and the rationale of treatment are detailed. William F. Bruce

1705z Histological and histochemical aspects of the morphologic alterations in the heart muscle caused by alcoholic intoxication. V. I. Yakovleva (Nauch.-Issled. Inst. Sudebnoi Med., Moscow). *Sudebno-Med. Ekspertiza, Mat. Zdravookhr. SSSR* 10(3), 25-30(1967)(Russ). Morphological alterations in the heart muscle of 8 persons who had died from alc. intoxication and 16 persons who had died from atherosclerosis and alc. intoxication (2.3‰ alc. in the blood) were studied histol. and histochem. An increased size and wt. of the heart caused by a complex of pathol. processes was noted in persons with alc. intoxication. A damaged permeability of blood vessels and stromal edema connected with the accumulation of acid mucopolysaccharides, protein, and fatty myocardium dystrophy were observed. N. Mastalir

1706a Toxicity profiles of vinyl and polyolefinic plastics and their additives. Wallace L. Guess and Sol Haberman (Coll. of Pharm., Univ. of Texas, Austin). *Tech. Pap. Reg. Tech. Conf., Soc. Plast. Eng., N.Y. Sect.* 1967(Sept.), 15-23(Eng). Plastics were implanted into rabbit muscle tissue by cutting slivers of plastics into sections 1 mm. × 1 cm. These slivers were placed in a hypodermic needle and inserted into the paravertebral muscle of anesthetized rabbits. One week later the rabbits were sacrificed and the implantation sites examd. for evidence of a histopathol. tissue reaction around the plastics. A cell culture technique was also used (*CA* 62:9672e; 63:16545c). Exts. of plastics were prepd. by extg. for 24 hrs. in a Soxhlet app. using 95% EtOH; the alc. was removed by gentle heat in vacuo and the residue was reconstituted with normal saline. These exts. were then evaluated by mouse i.p. injection, rabbit intradermal injection, cell culture techniques, and hemolysis liability to a 2% suspension of washed red blood cells. Alc. extns. of one of the polyvinyl chloride (PVC) resins revealed that the supposedly pure PVC resin contained an av. of 3.5% extractable material, which appeared to be a waxy-like semisolid material. Some of this material was pushed through a needle into the muscle of a rabbit and gave visible necrosis and microscopic eosinophilia after 1 week. Two different lots of PVC resin contained toxic material which was leachable into a biol. system and an extreme eosinophilic response was noted. A large group of chem. agents (17 phthalates, 5 sebacates, 6 adipates, 7 citrates, and 9 miscellaneous compds.) currently being used or having potential use in various plastics formulations were investigated for toxic potential. As a group, the stabilizers showed the greatest degree of toxicity and if these were used in conjunction with a plasticizer in a plastics formulation, the toxic potential was greatly increased due to an increased opportunity to migrate from the plastics. M. M. Fetkovich

1707b Changes in resistance to acids of erythrocytes during poisoning by some industrial poisons. M. G. Polyak, P. A. Balander, V. N. Fedyanina, and T. I. Nevolina (Nauch.-Issled. Sanit. Inst., Novosibirsk). *Vop. Biofiz., Biokhim. Patol. Eritrotsitov, Akad. Nauk SSSR, Sib. Otd., Inst. Fiz.* 1967, 173-9 (Russ). The effect of mesidine (I), dichloroisobutylene (II), and dioxydiphenylpropane (III) on erythrocytes was tested in 50 rats and 28 rabbits. After I inhalation of concns. of 1.8-4.7 mg./l., close to the LD, the no. of erythrocytes was not affected, but methemoglobin increased by 3-5%; this increase, nevertheless, was not correlated with the severity of poisoning. In rabbits chronically poisoned with 0.01 mg. I/l., reduced glutathione in the blood increased after 4 months, whereas lower doses of I were without effect on its level. Acid resistance of the erythrocytes showed a left shift after poisoning with 0.001 mg. I/l.; after 0.01 mg./l. shifts both to the left and to the right occurred. II caused injury to the liver and was more toxic than CCl_4: 3 mg./l. caused hemolysis in rats, their erythrocyte no. decreased from 8.3 to 5.8 × 10^6/mm.^{3} and Hb decreased from 14.8 to 9.0 g. %. At the same time, reticulocytes increased from 460 to 708 × 10^3/mm.^{3} Changes on erythrograms of rats chronically intoxicated with 0.6 mg. II/l. for 3-5 weeks were similar to the changes after acute poisoning, but the no. of erythrocytes, reticulocytes, and the concn. of Hb did not differ from intact animals. III had effects similar to phenol; erythrograms normalized within 25 days. Crystn. of native IIb occurred in the erythrocytes of rats poisoned with II or III. M. Kalab

1708c Glucuronide formation in cases of toxic liver damage. K. Beck, H. A. Kuehn, and B. Nouraie (Med. Univ., Freiburg/Br., Ger.). *Acta Hepato-Splenol.* 14(5), 280-91(1967)(Ger). Rabbits were poisoned either with a single s.c. injection of 1.5 ml. CCl_4/kg. of body wt., 0.3 ml. CCl_4/kg. of body wt. each 3rd day 6 times, a total of 40 ml. CCl_4 over a long period of time, or with daily s.c. injections of 0.4 ml. 5% aq. allyl alc. (I) soln./kg. body wt. for 23 days to test the liver capacity of glucuronide (II) synthesis. The rise of plasma II-glucuronic acid (III) as well as the urinary excretion of III after loading with 1 g. *N*-acetyl-*p*-aminophenol showed that the max. blood levels were reached only after 3 hrs. (in normal animals after 1 hr.), while the total increase was also considerably lower than in normal animals with a concomitant delayed urinary excretion of III. The disturbances of the II synthesis are not as marked in cases of subacute poisoning, while in chronic poisoning (CCl_4 cirrhosis) only a shift of the max. to the 2nd hr. was observed. Poisoning with I produced no significant influence on II synthesis. These results confirm findings in humans in which a disturbance of II formation (after loading with menthol) only occurs in cases with

hemagglutination reaction—extn. and purification of antigen—selection of inactivation method (Rezepova) 66:73e. Some factors that influence the hemolysin of ox serum (Glover) 72443g. Immunochem. studies of insol. wheat pro... bains) 72465r. Fluorescence depolarization ... pyrene butyric acid bovine serum conjugates (L...

14—TOXICOLOGY

T. R. TORKELSON

74452h A simple apparatus for the estimation of some gaseous or volatile toxins. H. L. Boiteau and Cl. Mousson (Fac. Med. Pharm., Nantes, France). *Ann. Biol. Clin.* (Paris) **25**(1-2), 215-27(1967)(Fr). The app. is made up of 2 compartments connected through a glass stopper. The first one, of known vol. (approx. 100 ml.) contains the sample to be analyzed. The second one (approx. 10 ml.) contains the reagents used in the detn. By connecting the 2 containers, the reaction can proceed and the detn. carried out colorimetrically or volumetrically. With this app., CO, CO_2, N_2O_4, HCN, and trichloroethylene were detd. within the limits of the sensitivity of the specific detn. Rene Evard

74453j Poison chamber and a setup for creating constant vapor concentrations under dynamic conditions. N. Sh. Vol'berg and A. A. Golubev (Inst. Ind. Hyg. Prof. Diseases, Leningrad). *Gig. Tr. Prof. Zabol.* 10(12), 49-50(1966)(Russ). A chamber is described which contains small flasks for individual mice. The flasks are supplied with glass capillaries to ensure const. flow of poison-contg. gas. This app. was suggested for poisoning mice with halosilanes. To avoid halosilanes decompn. with moisture, air was dried in a column of silica gel and then on zeolite NaA (mol. sieve). Inlet of toxic fluid terminates on an elec. heated coil, where the fluid evaps. and enters the air stream. Miloslav Kalab

74454k Toxicity studies of latex rubber and soft poly(vinyl chloride) as tubing materials. Comparison between in vitro tests and results of prolonged administration to rats. L. H. Eklund, C. A. Grant, and V. Willners (Kgl. Farm. Inst., Stockholm). *Acta Pharm. Suecica* 2(5), 349-56(1965)(Eng). A comparison has been made between in vitro and in vivo properties of water exts. of latex and poly(vinyl chloride) products. Sixty rats were injected 1 ml./100 g. of body wt. i.p. for 12 weeks. Hematological, liver and kidney tests, and pathol. examns. were carried out. At the end of the study, no appreciable differences from the control animals were observed. The value of in vitro tests as a relevant parameter of toxicity of polymer products is discussed. RCTT

74455m Alterations in microsomal electron transport, oxidative N-demethylation, and azo-dye cleavage in carbon tetrachloride and dimethylnitrosamine-induced liver injury. E. A. Smuckler, E. Arrhenius, and T. Hultin (Wenner-Gren Inst. Exptl. Biol., Stockholm). *Biochem. J.* 103(1), 55-64(1967)(Eng). The effect of administration of CCl_4 and dimethylnitrosamine in vivo on hepatic microsomal function related to drug metabolism was measured. The capacity of isolated microsomes to demethylate dimethylaniline was diminished during the 1st hr. after CCl_4 poisoning and during the 2nd hr. after dimethylnitrosamine poisoning. Thereafter the microsomes from CCl_4-poisoned livers showed a continuous decline in activity so that at 24 hrs. there was little residual capacity to undertake demethylation. Microsomes from dimethylnitrosamine-poisoned animals were not different from controls at 24 hrs. During the first 3 hrs. there was a transient rise in the accumulation of the *N*-oxide intermediate in CCl_4-poisoned livers, with a subsequent fall to below control values. In dimethylnitrosamine poisoning there was a parallel decrease in *N*-oxide accumulation with decreased demethylation. In the latter part of the first 24 hrs., the ratio of *N*-oxide accumulation to demethylation was increased in both instances. At 2 hrs. after poisoning with either compd., there was no evidence of altered NADPH-dependent neotetrazolium redn. or lipid peroxidn. NADPH-dependent azo-dye cleavage was decreased. There was no difference in microsomal cytochrome b_5 content, but there was a decrease in the amt. of cytochrome P-450. This latter change was correlated with the decreased capacity for NADPH-dependent oxidative demethylation. It is suggested that dimethylnitrosamine is assocd. with a defect in microsomal NADPH-dependent electron transport at the level of cytochrome P-450. In addn. to affecting cytochrome P-450, CCl_4 is assocd. with a 2nd severe block involving the release of formaldehyde from the *N*-oxide intermediate. RCTG

74456n The effect of the aflatoxins B_1, G_1, and G_2 on protein and nucleic acid synthesis in rat liver. Janet I. Clifford, K. R. Rees, and M. Elizabeth M. Stevens (Univ. Coll. Hosp. Med. School, London). *Biochem. J.* 103, 258-64(1967)(Eng). A comparison has been made of the difference spectra obtained by causing various aflatoxins (B_1, G_1, and G_2) to interact with calf thymus DNA. The effect of these toxins on RNA and protein synthesis by rat liver slices has been measured. The extent of their inhibitory action on the synthetic reactions was proportional [illegible] with DNA. It is proposed that their toxicity depends ... action. It was demonstrated that the RNA ... nucleoli isolated from the livers of aflatoxin B_1-poison... inhibited. This finding is in agreement with th... mechanism for the hepatotoxic action of aflatoxin.

74457p Effect of pretreatment with polycyclic h... on the metabolism of dimethylbenzanthracene-12-¹⁴C ... and other tissues. P. H. Jellinck and Barbara G... British Columbia, Vancouver, Can.). *Biochem.* ... 16(1), 131-41(1967)(Eng). Pretreatment of rats w... polycylic hydrocarbons (3-methylcholanthrene, ... anthracene, benzo(*a*)pyrene, anthracene, phenan... naphthalene) alters the metabolism of 7,12-dim... anthracene (DMBA) by hepatic tissue from side-ch... hydroxylation. No change in the metabolism of DM... adrenal glands was observed under these condition..., hydroxylated metabolite of the carcinogen was form... tissue. It is suggested that protection by aromatic hy... against DMBA-induced adrenal necrosis is achieved by ... the yield of the 7-hydroxymethyl deriv. of DMBA ... This product, rather than the unchanged carcinogen, ... the proximal necrotic agent by virtue of its structural ... to the adrenocortical steroids. The cytotoxic eff... adrenals may be due to damage of cellular mem... release of lysosomal enzymes. 24 references.

74458q Metabolism of chloroethanol in the rat. ... Johnson (Med. Res. Council Labs., Carshalton, En... *chem. Pharmacol.* 16(1), 185-99(1967)(Eng). Wh... ethanol is given orally to rats, liver GSH (reduced gl... is rapidly depleted and *S*-(carboxymethyl)glutathion... Release of $^{36}Cl^-$ from chloroethanol-^{36}Cl has been studi... and shown to require stoichiometric amts. of GSH (1 ... NAD (2 moles). *S*-(Carboxymethyl)glutathione ... identified as the product in vitro. Ethanol inhibits th... of GSH in vivo and release of $^{36}Cl^-$ in vitro. Chloro... readily dehydrogenated in vitro by purified yeast and ... alc. dehydrogenases. The route from chloroethanol ... boxymethyl)glutathione has been studied in vitro. *S*... methyl)glutathione is degraded by kidney homogenate ... glutamic acid, and S-(carboxymethyl)cysteine. ... metabolism of the latter compd. is discussed. It is ... that the toxicity of chloroethanol is due to its con... chloroacetaldehyde in vivo.

74459r The effect of carbon tetrachloride on rat ... somes. David H. Alpers and Kurt J. Isselbacher ... Med. School, Boston, Mass.). *Biochim. Biophys.* ... 33-42(1967)(Eng). The effects of CCl_4 on isolated ... and rat liver slices were examd. The addn. of CCl_4 ... lysosomes caused the release of β-glucuronidase, ace... tase, and acid RNase activity. The release occurre... lag period and was linear. In addn., the rate of ... dependent on the CCl_4 concn. CCl_4 added to liver sl... a redistribution of the lysosomal enzyme activity su... greater percent was found in the supernatant vs. the ... fraction. Under these conditions leucine-^{14}C incorp... protein was inhibited. The effects on lysosomal enzy... leucine incorporation were roughly comparable for a ... of CCl_4. Studies of the kinetics of these changes ... to be linear and to occur without any detectable ... Lysosomes were isolated by 2 different methods, su... peroxidn. occurred to a much greater extent in one ... pared with the other. Despite this difference, CCl_4 ... comparable release of lysosomal enzyme activity in b... Furthermore, in neither lysosomal fraction did CCl_4 ... lipid peroxidn. at a time when its effect on enzyme ... max. 32 references.

74460j Veterinary toxicology, to make a diagnosi... Buck and Virginia Marshall (Iowa State Univ., ... *State Univ. Vet.* 28(1), 7-9(1966)(Eng). ... chem. toxicants and their diagnosis in veterinary ... discussed. Mar...

74461k Histochemical enzyme levels in experi... cirrhosis. T. Tashev, L. Krustev, and L. Bala... *Inst. Khranene, Bulgar. Akad. Nauk* 5, 29-38 ... Exptl. liver cirrhosis was induced in rats by daily s... injection of CCl_4 (0.05 ml.) during 3 months. Alk. ... was neg. in normal liver parenchyma and bile du... strongly pos. in hepatic cells of cirrhotic areas. ... changes were observed with acid phosphatase; its ... decreased in half of the exptl. animals with liver cir...

i.6 74454 k

OLI 5800

Immunofluorescent localization of calcitonin in C cells of pig and dog thyroid (Bussolati) 63179y. Differentiation and functional expression of potential antibody-producing cells in presence of chloramphenicol (Schoenberg) 64010y. Action of heparin and aminoguanidine on anaphylactic, anaphylatoxin- and histamine-induced shock in guinea pigs (Giertz) 64178j. Effect of anticoagulants on delayed hypersensitivity reactions (Cohen) 6414?x. Side effects of drugs on granulopoiesis and thrombopoiesis (Hoppe) 63912g. Inhibition of chlorophyllin derivs. against complement activities and anaphylactic reaction (Sindo) 64140r. Effect of Na Cu chlorophyllin as complement inhibitor on allograft reaction (Fujii) 64141s. Effect of Imuran (azathioprine) on susceptibility of guinea pigs to allergic encephalomyelitis (Field) 64064u. Elucidation of structure of Vi-antigen isolated from (Escherichia coli) 5396/38 (Heyns) 65785s. Effect of chloroprene on immunologic reactivity of organism in persons vaccinated against typhoid fever (Mikaelyan) 58658z. Analysis of the crystal antigens of Bacillus thuringiensis by gel diffusion (Pendleton) 62794h. Interferon and interferon-like substances—present state of research (Lackovic) 62728q.
Patent: Allergen prepns. (Aktiebolag Cernelle) 58845h.

—TOXICOLOGY

T. R. TORKELSON

crease in that of neutral lipids were observed in mitochondria from alc.-fed rats, compared with controls. The compn. of the phospholipid fraction was nearly identical in the 2 groups of animals. 22 references. BXJN

63866v Diuretic therapy of acute intoxication with hypnotics. G. A. Krueger (Evangelischen Krankenhauses, Muelheim, Ger.). *Anaesthetist* 16(2), 40–4(1967)(Ger). Furosemide (20 mg.), injected i.v. into patients with barbiturate intoxication and added to running infusions dependent on the progress of the case (an av. of 20 mg./1000 ml.), was an effective therapeutic measure against the intoxication. The drug was easily administered and could be safely used in case of diabetes or complications caused by depression of cardiac, circulatory, or liver functions. It considerably reduced the time of unconsciousness and hospital treatment. Circulatory analeptics, particularly ethyldiphenylpropenamine, were effectively used in cases of hypotension in order to reestablish normal blood pressure values. 46 references. BKJG

63867w Electroencephalographic changes in chronic poisoning with trichloroethylene. K. Schwartzova (Karlova Univ., Plzen, Czech.). *Cesk. Neurol.* 29(6), 369–77(1966)(Czech). Examns. of 78 persons, exposed for 7.5 years on the av., revealed insufficient mental relaxation, lowered vigility, and disturbance of the α rhythm (on the border of abnormality) in 46.1%, small focal lesions and few synchronous θ waves (slight abnormality) in 5.1%, and frequent disperse or diffuse θ waves with synchronous exacerbations or focally accentuated (moderate abnormality) in 34.5%, of the cases. L. J. Urbanek

63868x Depression of potential myocardial contractility accompanying chronic ethyl alcohol administration. John Elwood Maines, III (Tulane Univ., New Orleans, La.). *Univ. Microfilms* (Ann Arbor, Mich.), *Order No.* 66-10,767, 88 pp.; *Diss. Abstr. B* 27(5), 1561–2(1966)(Eng). SNDC

63869y Subacute toxicity of ether extracts of PVC. S. Homrowski, H. Piekacz, and H. Mazur (Staatliches Inst. Hyg., Warsaw). *Ernaehrungsforschung* 11(3), 398–402(1966)(Ger). Toxicity assessments of H_2O_2-free Et_2O-extd. plates of a poly(vinyl chloride) (PVC) plastic (consisting of PVC 89.5, Palatinol AH (dioctyl phthalate) as plasticizer 7, Advastab 17M (S-contg. org. dibutyl Sn compd.) as stabilizer 1.0, Advawax 360 as lubricant 0.5, and TiO_2 as pigment 2.0%) were carried out by thin-layer chromatography of blood specimens obtained periodically from Wistar rats of both sexes with starting wts. of 100–125 g. In addn. to a standard diet, by means of a stomach tube, 60 animals received 1 or 100 mg. PVC ext./kg. body wt./day as a 0.3 or 30.0% suspension, resp., in olive oil, for 3 months, and 10 of these animals received 1 g. undild. PVC ext./kg. body wt./day throughout the 4th month. All exptl. animals exhibited normal behavior, appearance, and eating habits; body wt. gains and blood-constituent values were similar to those of olive oil-treated control animals. No significant macro- or microscopic lesions of liver, spleen, kidneys, or stomach were revealed following removal after sacrifice of animals by Et_2O narcosis, with the exception of a statistically significant liver enlargement (about 28% heavier than for controls) among animals receiving large doses of ext. Only about 25% of the total content of non-PVC-contg. plastic constituents was extd. by the Et_2O, of which 90% was plasticizer and 3% stabilizer; it was assumed that the other 7% consisted of the lubricant, since the pigment is practically insol. in Et_2O. On the basis of reports, the plasticizer and lubricant, both with an LD_{50} range of 33–35 g./kg. body wt., were practically nonpoisonous, whereas the LD_{50} value for the stabilizer was 500–600 mg./kg. body wt., indicating that any toxicity evident in plastics exts. was due to the stabilizer. Doses of 100 and 1000 mg. PVC ext./kg. body wt., which corresponded to about 3 and 30 mg. stabilizer/kg. body wt., resp., did not elicit any adverse reactions; the heavier wt. of the liver, as a result of hemorrhage of its blood vessels, was considered to be evidence of an enhanced activity of this organ in handling the increased amt. of ext. content. Roy Solkot

63870s Hepatotropic action of N-methylformamide. V. N. Vasil'eva and T. M. Sukharevskaya (Inst. Ind. Hyg. Prof. Diseases, Moscow). *Gig. Tr. Prof. Zabol.* 10(12), 53–4(1966)(Russ). A case history is presented of a person who drank 50 ml. of *N*-methylformamide; even after washing the stomach with water, acute injury to the liver developed, accompanied by proconvertin (I) decrease (by 24%) in the blood and increase of plasma fibrinogen to 910 mg. %. I normalized more slowly than any other biochem. index. Miloslav Kalab

63871t Toxicity of phthalodinitrile and tetrachlorophthalodinitrile. I. Acute toxicity in mice. Hiroshi Yoshikawa and Kiyoyuki Kawai (Nat. Inst. Ind. Health, Kawasaki). *Ind. Health* (Kawasaki, Japan) 4(1–2), 11–15(1966)(Eng). Five to 30 min. after intraperitoneal, subcutaneous, or oral administration of *o*-phthalodinitrile (I), mice had generalized convulsions lasting 10–15 sec. The convulsive attacks were repeated several times with 30–60 sec. intervals and death occurred within 5 hrs. Upon administration of the meta isomer of I, convulsions were less marked and animals receiving the para isomer showed only weakness. The resp. LD_{50} values of *o*-, *m*-, and *p*-I were 34.5, 481.3 and 698.6 mg./kg. LD_{50} values for *o*-, *m*-, and *p*-tetrachlorophthalodinitrile administered intraperitoneally were 66.4, 2.5, and 1581 mg./kg. Convulsions were absent with the chlorinated derivs. Emanuel Kaplan

63872u Hepatotoxicity of a series of organotin esters. David J. Galley, W. L. Guess, and J. Autian (Univ. of Texas, Austin). *J. Pharm. Sci.* 56(2), 240–3(1967)(Eng). Four organotin esters having use or potential use in the formulation of medically used plastics were evaluated for their toxic effects on the liver following oral administration. The liver damage is apparently the cumulative type, requiring several days to manifest damage in the form of elevated serum glutamic-pyruvic transaminase values and prolonged hexobarbital narcosis. The toxic potential was, in general, soly. related. RCYG

63873v Effect of Vandid on cardiac functions in experimental trichloroethylene poisoning. A. Kubacki, A. Mrozikiewicz, A. Skubiszynska, and A. Wachowiak (Akad. Med., Poznan, Poland). *Med. Pracy* 17(3), 196–200(1966)(Pol). Electrocardiograph curves were surveyed in rabbits exptl. poisoned with trichloroethylene. Administration of Vandid (*N,N*-diethylvanillamide) (5 mg./kg.) produced no arrhythmia symptoms, which occurred, however, when adrenaline (0.1 mg./kg.) was used as the cardiac stimulant. Helena Lange

63874w Histochemical evaluation of liver extracts in prophylaxis of experimental carbon tetrachloride poisoning. Kazimierz Zajusz and Marek Zacharewicz (Inst. Med. Pracy, Zabrze, Poland). *Med. Pracy* 17(4), 317–28(1966)(Pol). CCl_4 administered by stomach tube to rats (0.6 ml./animal) caused an initial increase of the liver acid and alk. phosphatases and ATPase activities, but this trend soon discontinued and the enzymes became inhibited almost completely. Liver succinic dehydrogenase, phosphorylase, and the branching enzyme were also totally inhibited. Enhanced reactions for alk. and acid phosphatases were observed in the kidneys of the poisoned animals. The effects of CCl_4 were markedly moderated by prophylactic administration of hepatic exts. Helena Lange

63875x Pathomorphologic changes in white rat organs during chronic combined inhalation of methanol, aqueous ethanol, and furfural. R. U. Ubaidullaev and P. V. Panov. *Med. Zh. Uzb.* 1966(9), 70–3(Russ). Three different concns. of a mixt. of vapors of MeOH, aq. EtOH, and furfural were used in an inhalation expt. of 3-months duration. Severe histol. changes were found at the highest concn. only (5.79 mg. MeOH/m.³–28.48 mg. EtOH/m.³–0.3 mg. furfural/m.³). RNA of brain and glycogen of liver decreased, accompanied by fatty liver dystrophy. J. Prokes

63876y Morphological changes in the organs of animals in acute and chronic intoxications with α-methylstyrene and styrene. V. V. Veselova and G. A. Ogleznev. *Nauch. Tr. Omsk. Med. Inst.* No. 61, 89–95(1965)(Russ). Expts. were conducted on mice and rats administered intragastrically an aq. soln. of α-methylstyrene (I) or styrene (II). Internal administration of max. tolerance doses of I (3 for mice and 4 g./kg. for rats) and II (5 and 2 g./kg., resp.) caused no marked morphological changes. In acute I intoxications, grave hemodynamic and dystrophic changes in the parenchymatous organs, most pronounced in the lungs, were recorded. The group of morphol. changes found in the central nervous system of animals with I intoxication was a syndrome of toxic encephalopathy. Morphol. changes caused by I intoxication did not essentially differ from II-caused changes. Chronic administration of II (0.014 mg./kg.) to rats caused development of dystrophic changes in the central nervous system more often than did 0.5 mg. I/kg. under the same conditions.

Í1
that
th chloro-
otein con-
by an un-
fluence of
were incon-
ector in this
, were con-
with chloro-
activities of
uric trans-
nd Frankel
levski units.
blood, liver,
nimals. In
rnal tissues
e spleen the
enzymic ac-
roprene re-
higher level
of pyridoxal
that the in-
its reaction
Redlighian
holesterol in
and hepato-
erke-Krank-
(9), 748-50
of choles-
istration of
ion of ethio-
dized follow-
/kg.) given
CMJG
rat) toxicity
). D. E.
Ind. Biol.
oxicol. 4(4),
studies have
thylene gly-
in both
given
ect. The
DDGB was
-day study
, and 2.5%
both sexes at
was attribut-
h this effect
ematological
d 2.5% dict-
nd the rela-
reased. No
to DDGB.
tary level of
ys could not
ted.
RCTT
s. Norman
erck Chem.
Chem. Soc.
reacts with
n relatively
reported in-
explained in
plexing and
RCJC
ing agents.
m. Pharma-
of effective-
mustard, L-
produced a
d intracere-
v included a
ns of central
e behavior,
tonic exten-
amide, and
in doses of
nytoin (100
effects in-
barbitone
a (1 g./
ting the
CMJN
ent on the
B. Meyers,
Greenfield,
(Eng). A

method using rats that can be of value in predicting the effects of chronic treatment with psychotherapeutic on the acute toxicity of other medicinal agents ... Results obtained by this method showed that nortriptyline, like amitriptyline, enhanced the toxicity of most ... 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 alc. 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 2.0 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 1101 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(8), Pt. II, Abstr. No. 81414. 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. BPJN

1238f 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 Toba. *Rev. Chim.* (Bucharest) 17(8), 499-501(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. alc. 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 elutant 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. ... concd. NH_3 ... heated for 5 min. at ... ninhydrin in MeOH, and dried. The II is indicated by ... violet spots. For distinct development, the strips are sprayed finally with 10% $Cu(NO_3)_2$ (carmine red spots). The R_f at 24° 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 $Cu(NO_3)_2$. The extn. of II from urine was demonstrated by treating a soln. of 25 μg. II/cc. in urine as above, cutting the corresponding 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 ... 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 Elieser

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 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, I. 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

1243c 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 30-44%. 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 ... of IV/kg. decreased I incorporation into spleen protein ... and tumor-bearing mice by 30%; 150 mg./kg. had ... on protein biosynthesis in the spleen. Felix Borsk

1244n Experimental intoxication by ammonium salts. Protection by some amino acids and their mechanism of action. I. 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 con

OLI 5802