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 occurs 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; Guiggiani, 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 parenchymatous organs 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. Toksikol.* 1968, 31(3), 364–5 (Russ). Polyethylene polyamine (750 mg./kg.) administered s.c. to rats and mice increased the hepatic monoamine oxidase and histaminase activities, decreasing the tyramine and histamine levels in the blood and organs of the poisoned animals. The pathol. disturbances observed in mice and rats with acute polyethylene polyamine poisoning may be due to the increased oxidative deamination of endogenous amines with the consequent formation of aldehydes, NH_3, and H_2O_2. BJJR

50598w Effect of niacin on phagocytosis in chronic trinitrotoluene poisoning. Pidemskii, E. L.; Chukichev, E. M.; Mul'menko, A. M. (Perm. Univ., Perm, USSR). *Farmakol. Toksikol.* 1968, 31(3), 365–6 (Russ). Trinitrotoluene (TNT) administered orally at 30 mg./kg. daily for 6 days to rats progressively decreased phagocytosis. Niacin administered simultaneously at 1 mg./kg. s.c. prevented this decrease and even increased the phagocytic activity of the leukocytes to 1.5-times the level in control rats. BJJR

50599x Pathogenic effect of chronic exposure to vinyl chloride on rabbits. Vazin, A. N.; Plokhova, E. I. (Inst. Gig. Tr. Profzabol., Gorki, USSR). *Farmakol. Toksikol.* 1968, 31(3), 369–72 (Russ). In rabbits exposed to vinyl chloride (9–10 mg./l. air) for 4 hrs. daily for 5.5 months altered β-waves (frequency >80 Hz.) appeared on the electroencephalogram from the anterior hypothalamic nuclei, and the potentials of the anterior and posterior nuclei increased by 18–30 and 70–85%, resp., over control values. Concomitant changes in the cardiovascular system (bradycardia, arrhythmia, decreased voltage of the individual electrocardiogram peaks or whole complexes, decreased duration of systole, increased arterial pressure, and retarded blood flow) also occurred. The functional changes in the anterior and posterior hypothalamic nuelci may have a role in the pathogenesis of toxic angioneurosis due to vinyl chloride. BJJR

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

50601s Brown FK. IV. Cytopathic effects of Brown FK 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), 13–24 (Eng). Administration of 2 or 3 massive (1 g./kg.) oral doses of Brown FK to rats induced a myopathy in cardiac and skeletal muscles characterized by multiple vacuoles ~1–2 μ in diam. Ultrastructurally, these consisted of areas of fibrillolysis, affecting initially the A-band. Histochem., the myopathy was accompanied by a moderate increase in acid phosphatase activity and by a loss of phosphorylase activity. Subsequently, complete lysis of the affected fibers ensued. In the heart, lysis was followed by macrophage invasion and fibroblastic proliferation, and in skeletal muscle, by regeneration. The occurrence of lipofuscin in muscle fibers and in macrophages was scanty and 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 lysosomes was observed ultrastructurally during week 2–3 of the test. These changes were accompanied by a marked elevation of histochem. demonstrable acid phosphatase. Progressive deposition of lipofuscin was the principal pathological feature during week 3–12. Initial damage to the A- and I-bands in both cardiac and skeletal muscle distinguishes the Brown FK myopathy from that known to be produced by high doses of corticosteroids, thyroxine, chloroquine, or plasmocid, from ischemic damage and from muscle damage resulting from deprivation of K, Ca, or Mg. The lysosomal changes and accumulation of lipofuscin are suggestive of primary lysosomal damage which

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