

- 735 Tolerance for Residues of Ethylene Oxide. Anon. Federal Register 22, 931 (Feb. 14, 1957).

A tolerance of 50 ppm is established for residues of ethylene oxide in or on whole spices, under the Federal Food, Drug, and Cosmetic Act. -- Chem. Absts.

- 736 Toxic Liver Injury. The Metabolism of Dimethylnitroso Amine. P. N. Magee. Biochem. J. 64, 677-682 (1956).

The metabolism, distribution in the body, and excretion of dimethylnitroso amine were studied after its administration to rats, mice, and rabbits. It has been shown previously that this compound is a selective liver poison in these species, and that it can produce malignant primary liver tumors in the rat. The evidence suggests that dimethylnitroso amine is rapidly metabolized, and that the liver is the main, probably the only organ concerned. The compound is distributed uniformly in most tissues of the body, and there is reason to believe that it penetrates into the intracellular space. There is no selective concentration in the liver. SKF 525A (2-diethylaminoethyl diphenylpropylacetate hydrochloride) inhibits the metabolism of dimethylnitroso amine in the mouse, but only when given in large doses. The findings are discussed in relation to the pathogenesis of the acute liver necrosis caused by dimethylnitroso amine. -- Chem. Absts.

- 737 Acute Changes in the Liver after Administration of Thioacetamide. D. N. Gupta. J. Pathol. Bacteriol. 72, 183-192 (1956).

Thioacetamide produces consistent liver necrosis after 24 hours in rats when a dose of 200 mg. per kg. or more is given by various routes. The first change apparent after six hours is the loss of glycogen and of basophilic bodies in the centrilobular liver cells. The central veins and the adjacent sinusoids dilate and edema channels appear which connect the central veins with each other. Necrosis develops in the centrilobular cells and spreads along the edema channels; by 24 hours it is well established. In 42-48 hours, the necrotic liver cells were cleared away by mononuclear cells which move into the necrotic zone at about 12 hours. Regeneration extends from the periportal zone and healing is complete after a week. The histological sequence of events to acute thioacetamide intoxication is non-specific, since it resembles that of many other liver injuries. The centrilobular necrosis is probably due to a number of factors. -- Author's summary (Biol. Absts.)

- 738 Biochemical Changes in Liver in Acute Thioacetamide Intoxication. C. H. Gallagher, D. N. Gupta, J. D. Judah, and K. R. Rees. J. Pathol. Bacteriol. 72, 193-201 (1956).

The observed inhibitions in enzyme systems in liver are due to the accumulation of calcium in the affected liver cell. Administration of antihistamines protects against the action of thioacetamide. Adrenalectomy did not protect against thioacetamide intoxication. The evidence so far obtained suggests that the primary lesion in the liver cell is a loss of the semi-permeable properties of the cell wall with the entry of calcium ions. It is likely that the loss of this semi-permeable property is due to a direct attack on the cell wall rather than to a secondary effect resulting from a failure of the metabolic activity of the cell. -- Authors' summary (Biol. Absts.)

- 739 Tolerance for Residues of Sodium 2, 2-Dichloropropionate. Anon. Federal Register 22, 637 (Jan. 31, 1957).

A tolerance is established under the Federal Food, Drug, and Cosmetic Act for residues of sodium 2, 2-dichloropropionate as 2, 2-dichloropropionic acid of 35 ppm in or on cottonseed oil and 5 ppm in or on sugar beets and sugar-beet tops. -- Chem. Absts.