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Ultrastructural changes in the hepatocytes under the influence of alcohol were studied. The greatest changes were found in the mitochondria. Physical exertion and a low protein diet have a marked effect on the degree of alcohol poisoning. The first factor reduces whereas the second aggravates the harmful action of alcohol.

KEY WORDS: mitochondria; liver; alcohol

The mitochondria of hepatocytes are very sensitive to the action of alcohol [2, 10, 11, 14, 17]. This can be attributed to its toxicity, its high calorific value, and other properties of alcohol [5, 8, 13, 20].

The object of this investigation was to compare changes in the mitochondria of hepatocytes in alcoholics and in experimental animals receiving alcohol.

EXPERIMENTAL METHOD

Experiments were carried out on male albino rats weighing 150-200 g and divided into five groups: 1) control (37 animals); 2) rats receiving 50% alcohol by gastric tube in a dose of 0.5-0.8 ml/100 g body weight daily (50 animals); 3) rats also exposed to physical exertion (running on a treadmill) for 10-30 min daily (30 animals); 4) rats on a low protein diet (17 animals); 5) rats on a low protein diet and receiving 50% alcohol by gastric tube in a dose of 0.5-0.8 ml/100 g body weight (20 animals). The animals were killed on the 60th and 150th days after the beginning of the experiments. The liver was examined histologically and electron-microscopically.

Pieces of the right lobe of the liver were taken by punch biopsy from 15 chronic alcoholics and examined histologically and electron-microscopically.

EXPERIMENTAL RESULTS

Histological examination of the liver of the 15 chronic alcoholics showed marked or moderate fatty degeneration (3), fatty degeneration and persistent chronic hepatitis (5), persistent chronic hepatitis without fatty degeneration (1), and postnecrotic cirrhosis (1). No significant changes were found in the liver of five subjects.

Electron-microscopic examination revealed moderate swelling, lengthening, and deformation of the mitochondria, increased translucency of their matrix, some disorganization of the cristae, and the appearance of transversely arranged paracrystalline structures in the mitochondria. In the presence of marked fatty degeneration and postnecrotic cirrhosis the number of mitochondria in the hepatocytes was reduced. Material of fibrous appearance was found in the cytoplasm of the hepatocytes in one case of alcoholic hepatitis.

In the animals receiving alcohol fatty degeneration of the hepatocytes and lengthening and polymorphism of the mitochondria were observed [1, 3, 21*]. Physical exertion reduced the harmful effect of alcohol on the liver cells: the fatty degeneration and ultrastructural changes under these circumstances were less marked than in rats exposed to the action of alcohol only.

In animals kept on a low protein diet, on the other hand, the effects of alcohol on the hepatocytes were intensified. These animals developed considerable fatty degeneration and marked changes in the mitochondria (lipid and myelin degeneration; see Fig. 1).

*References 21 and 22 omitted from Russian original - Consultants Bureau.

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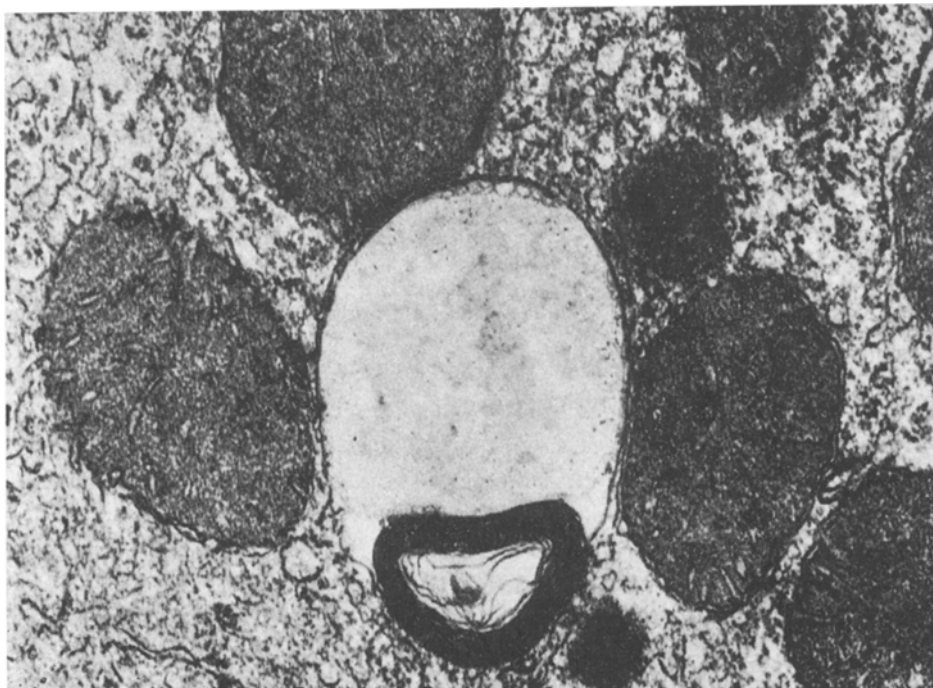


Fig. 1. Changes in mitochondria of hepatocytes under the influence of alcohol and low protein diet; 23,000 \times .

Some degree of parallel was thus established between the changes in the liver under the influence of alcohol in man and in the experimental animals. Direct correlation was found between the dose of alcohol and the ultrastructural changes in the hepatocytes, and certain additional factors also were shown to affect the course of alcohol damage to the liver.

Unlike in investigations by other workers, in the present case there was virtually no sign of the so-called alcoholic hyalin or Mallory's bodies in the liver cells [4, 6, 7, 9, 12, 18, 22]. This may perhaps be due to the fact that in the present observations alcoholic damage to the liver was moderate in degree. The results of these experiments add to the information in the literature on the important role of the mitochondria in intracellular metabolic processes [15, 16, 19].

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