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Brief Communication

MASSIVE LIPID ACCUMULATION IN MINK LIVER STELLATE CELLS MAY BE CAUSED BY FUSARIUM MYCOTOXINS IN THE FEED

During the last 3 years there has been an apparent increase in the number of animals submitted to the National Veterinary Institute for necropsy, having an anamnesis of stillbirths, disturbances of the central nervous system and infertility. The animals mostly affected have been mink, calves and chicken (*Karppanen & Rizzo* 1983). Mycotoxins have long been recognized as major causes of feed toxicoses in animals, and they are suspected to be the major cause also in these cases. The problems began shortly after a large quantity of raw material for the production of animal feed was imported to Finland. The following Fusarium-toxins have been isolated from industrially produced animal feed in Finland: deoxynivalenol or vomitoxin, nivalenol, fusarenone-X, diacetoxyscirpenol, T-2 toxin and HT-2 toxin (*Karppanen et al.* in press).

At histological examination of the livers of affected mink, an accumulation of lipid droplets in perisinusoidal cells was frequently seen. Perisinusoidal or stellate cells have been demonstrated in several mammalian species, including man (*Wake* 1980). They are reported to occupy less than 2 % of the normal liver parenchymal volume. The cells are able to store large doses of retinyl esters in intracytoplasmatic lipid droplets. Empty cells have a high structural similarity with fibroblasts. The cells are probably able to synthesize collagen, and they may be involved in the development of liver fibrosis (*De Leeuw et al.* 1984).

In order to study the ultrastructure of the livers of affected animals, some female mink, with an anamnesis of stillbirths, were delivered alive to the institute. After killing the animals, blood and liver samples were immediately obtained and processed. Small pieces of liver tissue were fixed in 3 % glutaraldehyde buffered with 0.1 mol/l phosphate buffer, pH 7.2 (osmolality approximately 555 mosmol/kg), for 1 h at 20°C. After post-fixation in 1 % osmium tetroxide, the samples were dehydrated and embedded in Epon. For light microscopy semithin sections were stained with 1 % toluidine blue in saturated sodium bicarbonate. For electron microscopy thin sections were contrasted

with lead citrate and uranyl acetate. Vitamin A (retinol) was determined chemically from serum and liver homogenates.

Stellate cells filled with lipid droplets of varying size occupied up to 3 % of liver parenchymal volume (Figs. 1 and 2). The cytoplasm was very scanty, containing mainly rough endoplasmic reticulum and, in some cells, a Golgi complex and microtubules. Some fat infiltration of hepatocytes was seen. More evident was a scanty smooth endoplasmic reticulum and glycogen content in these cells. Compared to vitamin A values in clinically normal mink taken as controls (∞ 20 USP/ml in serum and ∞ 1000 USP/g in liver homogenate), the values in mink with lipid accumulation in stellate cells were elevated up to almost 7 and 20 times, respectively.

Based on these preliminary results it seems possible that mycotoxins disturb vitamin A metabolism in mink liver.

L.-A. Lindberg, Y. Gröhn and *E. Karppanen*
The Departments of Anatomy and Animal Hygiene, College of Veterinary Medicine, and the National Veterinary Institute, Helsinki, Finland.

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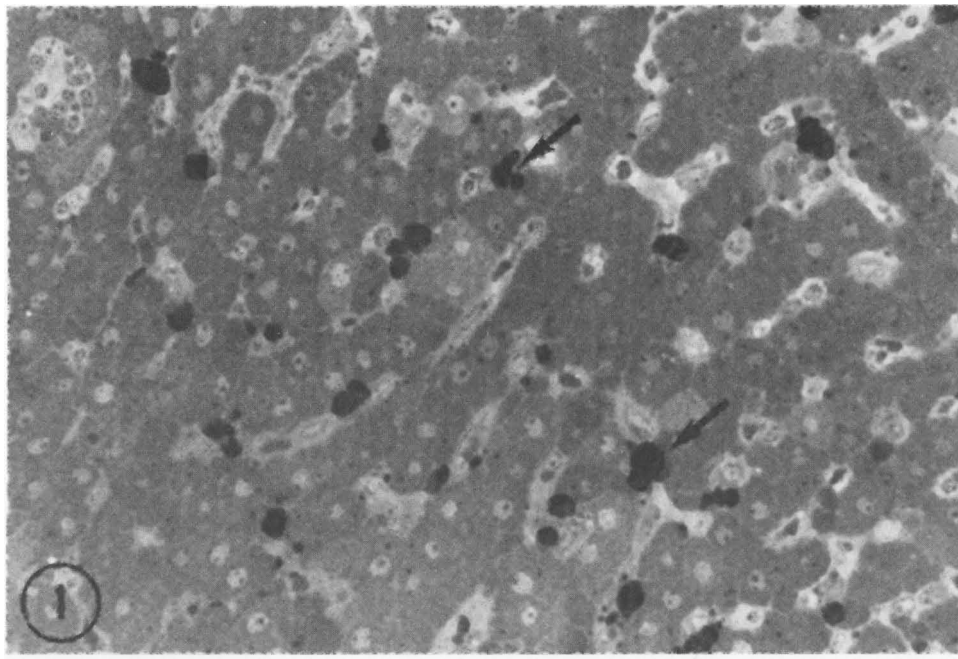
Reprints may be requested from: L.-A. Lindberg, the Department of Anatomy, College of Veterinary Medicine, P. O. Box 6, SF-00551 Helsinki, Finland.



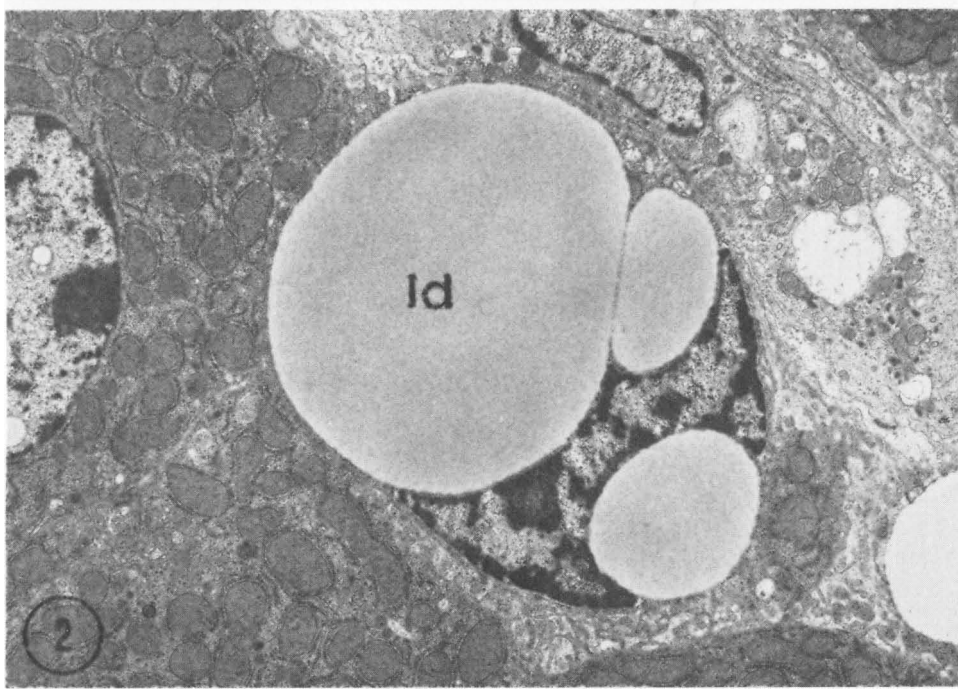


Figure 1. 1 μm Epon-section from the liver of an affected mink. Heavily contrasted lipid droplets in stellate cells are seen (arrows). x 450.

Figure 2. Electron micrograph of a stellate cell (ld), showing three lipid droplets and a scanty cytoplasm. The cell is surrounded by some hepatocytes and sinusoidal cells. x 6000.