

Comparative ultrastructural studies of the alterations to mouse hepatic parenchyma during *Trichinella spiralis* or *Toxocara canis* infection

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Both *Trichinella spiralis* and *Toxocara canis* have tissue dwelling larvae stage, which migrate through the soft tissues such as hepatic parenchyma of many hosts and produce many pathological changes in liver. On contrary to *T. spiralis* larvae, which only pass through the liver on their way from intestine to skeletal muscles, *T. canis* larvae become widely disseminated throughout the body. Most *T. canis* larvae remain in a developmentally arrested state and they induce many alterations in the hepatic parenchyma. The aim of our comparison is to show the pattern of changes in the hepatic parenchyma at the electron microscopic level of mice experimentally infected with *T. spiralis* or *T. canis*. Each mouse was orally infected with 800 *T. spiralis* larvae or 1000 eggs of *T. canis*. At 12 or 21 days post infection, mice were sacrificed and the fragments of liver, were obtained and standard processed for electron microscopy. The tissue samples were fixed with a mixture of 2% paraformaldehyde and 2.5% glutaraldehyde and postfixed in 1% OsO₄. Ultrathin sections were examined using a JEM 1200 EX transmission electron microscope.

The predominant cell in the liver was the hepatocyte, which numerically comprised 60 % of the total cell population and accounted for 80% of the liver's volume. We observed the destruction of these cells in mice group infected with both species of nematodes. The lipid drops and glycogen accumulation were present inside hepatocytes, mainly in group of mice infected with *T. canis*. We investigated also that mitochondria of hepatocytes were often swollen and they had paracrystalloid inclusions inside in mice infected with *T. spiralis*. The perisinusoidal space (space of Disse), which was located between a hepatocyte and a sinusoid, was extended and we observed many hepatic stellate cells (Ito cells) associated with collagen fibers in this space. The sinusoids were also often dilated and filled with inflammatory cells such as eosinophils or lymphocytes, mainly in mice infected with *T. canis*. The ultrastructural studies demonstrated that infection with *T. canis* larvae initiated mainly hepatic steatosis, which was a result of the response to liver damage. In contrast *T. spiralis* larvae migrating through the hepatic parenchyma evoked mainly fibrous degeneration of hepatocytes and the development of intramitochondrial paracrystalloid inclusions, which were described in patients with cirrhosis.