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IMPAIRMENT OF DRUG METABOLIZING SYSTEM IN EXPERIMENTAL DICROCOELIOSIS

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Dicrocoeliosis is a zoonotic disease caused by the liver fluke *Dicrocoelium dendriticum,* for which humans act as an accidental host. Cases of dicrocoeliosis both from Europe and the United States have been reported. Adequate pharmacological treatment of the disease is made difficult by the lack of information about its effect on hepatic function. We developed a hamster model for dicrocoeliosis that allows to investigate effects on liver drug biotransformation systems.

Methods. Dicrocoeliosis was produced by an oral administration of 50 metacercariae of *Dicrocoelium dendriticum*. Metacercariae were given suspended in 0.154M NaCl.

Results. By 12 weeks postinfection serum AST and ALT were significantly increased (+185% and +141% respectively). Hepatic microsomal cytochrome P-450 concentration was significantly reduced compared to healthy animals (0.196±0.034 nmol/mg prot vs 0.293±0.021 nmol/mg prot; $p<0.001$). Decreases in NADPH-cytochrome c reductase (-23%) and ethoxycoumarin O-deethylase (-31%) were observed. No change in microsomal membrane fluidity was detected.

Conclusion. Our data demonstrate that experimentally induced subclinical dicrocoeliosis causes an impairment in hepatic drug metabolizing capacity that is not due to alterations in membrane physical state. A need for the adjustment of drug dosage is suggested.