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التوثيق الالكتروني والميكروفيلم

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بالرسالة صفحات

لم ترد بالأصل

HOSSAM MAGHRABY

Histochemical studies *in vitro* and *in vivo* on *Fasciola* worms under the effect of bithionol and praziquantel

Thesis

Submitted to the Medical Research Institute

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OF HISTOCHEMISTRY AND CELL BIOLOGY

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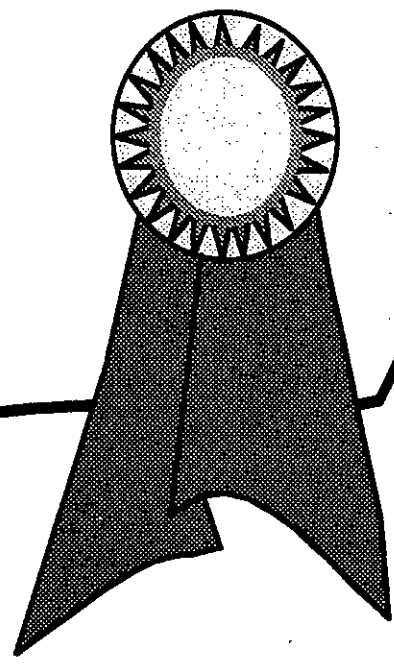
ABBREVIATIONS

F	:	*Fasciola.*
G	:	Gut.
E	:	Egg.
IS	:	Interstitial Strands.
M	:	Muscle.
P	:	Parenchymal cell
S	:	Spine.
SuL	:	Subtegumental Layer
T	:	Tegument.
V	:	Vitellaria cells.
↑	:	Necrosis.
+	:	Vacuolization.
I	:	Invagination
↑↑	:	Celluar infiltrations.
^	:	Disruption
AMPS	:	Acid mucopolysaccharides.
ACPase	:	Acid phosphatase.
ATPase	:	Adenosine triphosphatase.
G6PDase	:	Glucose-6- phosphate dehydrogenase.
B	:	Bithionol
PZQ	:	Praziquantel.

CONTENT

INTRODUCTION



INTRODUCTION

Fasciolosis is essentially a disease of herbivorous animals, while human infections occur accidentally. The economic importance of the common liver fluke lies in the damage it causes to cattle and sheep, but its presence in other mammals is also of interest.

In the last few years, human fasciolosis was reported more frequently from different parts of the world and it is becoming a world-wide problem caused mainly by the presence of *Fasciola gigantica and/*or *Fasciola hepatica.*

In Egypt, until 1978, only sporadic cases were diagnosed; these were usually considered spurious. Since then, an increasing number of human infections with both species have been diagnosed by clinical services and laboratories in certain provinces of the Nile Delta, in one province in Upper Egypt, and in the city of Alexandria. In some rural areas prevalence rates varied between 2% and 17%. The population at risk is 27.7 million individuals and the number infected is at least 830000, based on the estimated overall prevalence of 3% in the population at risk[1].

Human infection with *Fasciola* spp. was reported to result from ingestion of the encysted metacercariae attached to vegetables and plants eaten raw as salads[2].

The major pathological changes are seen during the migration of juvenile worms through the liver parenchyma before they enter the biliary tree. Juvenile worms digest hepatic tissue and cause extensive traumatic and necrotic lesions in the liver parenchyma. They are sometimes trapped in the organ and die leaving cavities filled with necrotic debris. After arrival to the bile ducts, they develop into adults, provoking inflammatory and adenomatous changes of the biliary epithelium, with enveloping fibrosis of the ducts. Pressure atrophy of the liver parenchyma and extensive periductal fibrosis may ensue if the pathological process in the biliary tract continues and the number of worms are considerable. Moreover, the gall-bladder frequently undergoes the same pathological changes as the bile ducts and may also be invaded. If the juvenile worms deviate during their normal migration to the liver, they may cause ectopic fasciolosis in the intestine, pancreas, lungs, heart, brain, skin, and other organs[1].

The currently recommended treatment for human fasciolosis is bithionol and triclabendazole[1]. A wide range of treatment schedules were monitored as albendazole, levamisole, mebendazole, praziquantel, and dehydroemetine[1].