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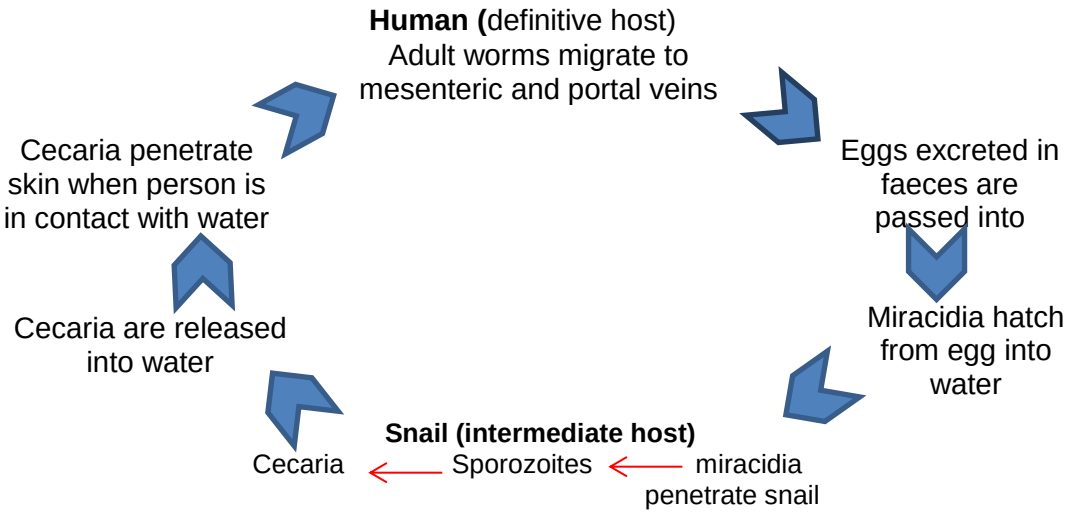
Schistosoma mansoni

Introduction

The Schistosomes belong to the Phylum *Platyhelmintha*. They are blood trematodes, have separate sexes, and require definitive and intermediate hosts to complete their life cycle. There are 3 species of Schistosomes responsible for human disease i.e.. *S. mansoni, S. haematobium* and *S. japonicum*. *S. mekongi* and *S. intercalatum* are less common.

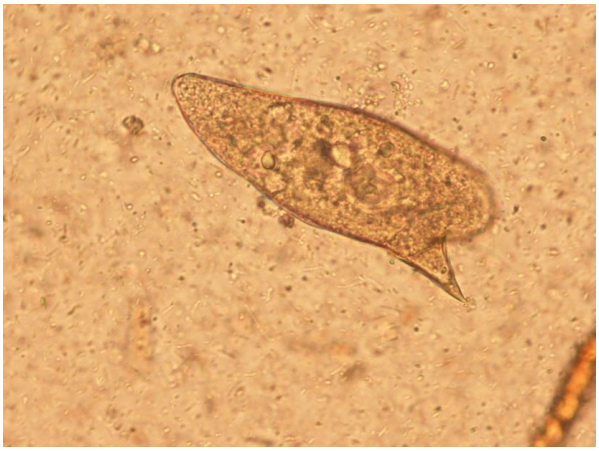
S. mansoni occurs in West and Central Africa, Egypt, Malagasy, the Arabian Peninsula, Brazil, Surinam, Venezuela and the West Indies. The intermediate host is Biomphalaria, an aquatic snail.

Life Cycle



Morphology

The ova of *S.mansoni* are 114-175um long by 45-68 um wide. They are light yellowish brown, elongate and possess a lateral spine. The shell is acid fast when stained with modified Ziehl-Neelsen. The eggs are often viable when passes in fresh unpreserved stool and the miracidia show flickering of an excretory flame cell. A non viable egg is dark coloured and shows no internal structural detail or flame cell movement. Eggs can become calcified after treatment and are usually smaller, appear black and often distorted with a less distinct spine.



An ovum of *Schistosoma mansoni*

Clinical Disease

The clinical disease is related to the stage of infection, previous host exposure, worm burden and host response. Cecarial dermatitis (swimmers itch) follows skin penetration and results in a maculopapular rash which may last 36 hours or more.

After mating, the mature flukes migrate to the venules draining the large intestine. There, eggs are laid and they penetrate the intestinal wall. They are excreted in the faeces often accompanied by blood and mucus.

It is the eggs and not the adult worms which are responsible for the pathology associated with *S. mansoni* infections. The adult flukes acquire host antigen which protects them from the host's immune response.

The host's reaction to the eggs which are lodged in the intestinal mucosa, leads to the formation of granulomata and ulceration of the intesinal wall. Some of the eggs reach the liver via the portal vein. The granulomatous response to these eggs can result in enlargement of the liver with fibrosis, ultimately leading to portal hypertension and ascites. The spleen may also become enlarged. Other complications may arise as a result of deposition of the eggs in other organs e.g. lungs.

Katayama fever is associated with heavy primary infection and egg production. Clinical features include high fever, hepatosplenomegaly, lymphadenopathy eosinophilia and dysentery. This syndrome occurs a few weeks after primary infection.

Laboratory Diagnosis

Microscopy

Laboratory confirmation of *S. mansoni* infection can be made by finding the eggs in the faeces. When eggs cannot be found in the faeces a rectal biopsy can be examined.

Serology

Serological tests are of value in the diagnosis of schistosomiasis when eggs cannot be found. An enzyme linked immunosorbent assay (ELISA) using soluble egg antigen, is employed at HTD.