

Schistosoma haematobium

Schistosoma haematobium is an important digenetic trematode, and is found in Africa and a locus of infection has been reported in India. It is a major agent of schistosomiasis; more specifically, it is associated with urinary schistosomiasis.

Adults are found in the venous plexuses around the urinary bladder and the released eggs travel to the wall of the urinary bladder causing haematuria and fibrosis of the bladder. Adults reside in the posterior mesenteric arteries and the eggs are laid in the walls of the bladder, ureters and urethra. Man is the only significant maintenance host of this species although the infection has been found in animals e.g baboons and monkeys in East Africa, rodents in Kenya and Southern Africa, Pigs in Nigeria and Chimpanzes in West Africa. The bladder becomes calcified, and there is increased pressure on ureters and kidneys otherwise known as hydronephrosis. Inflammation of the genitals due to *S. haematobium* may contribute to the propagation of HIV. Studies have shown the relationship between *S. haematobium* infection and the development of squamous cell carcinoma of the bladder.

Morphology

Adult schistosomes share all the fundamental features of the digenea. Adult males are 10 to 15 mm long. They have a basic bilateral symmetry, oral and ventral suckers, a body covering of a syncytial tegument, a blind-ending digestive system consisting of mouth, esophagus and bifurcated caeca; the area between the tegument and alimentary canal filled with a loose network of mesoderm cells, and an excretory or osmoregulatory system based on flame cells. They have deep grooves called gynecophoral canals in which adult females typically lie. Males have many small nodules (tubercles) on their dorsal surfaces and many tiny spines on their suckers and inside their gynecophoral canals. Females are longer (16-22 mm), smoother, and more slender. Both sexes have two suckers, one anterior and one ventral, which are used to grip venule walls. Eggs, which can be found in the urine of infected hosts, are 110-170 µm long by 40 to 70 µm wide. They are elongated with a distinctive terminal spine and look like microscopic American footballs with a spike on one end. The shells of the eggs are clear and contain

miracidia. Adult worms tend to be 10–20 mm (0.4–0.8 in) long and use globins from their hosts' hemoglobin for their own circulatory system.

Life cycle

The free swimming infective larval cercariae burrow into human skin when it comes into contact with contaminated water. The cercariae enter the blood stream of the host where they travel to the liver to mature into adult flukes. In order to avoid detection by the immune system inside the host, the adults have the ability to coat themselves with host antigen. After a period of about three weeks the young flukes migrate to the urinary bladder veins to copulate. The female fluke lays as many as 30 eggs per day which migrate to the lumen of the urinary bladder and ureters. These eggs measure 112-170µm by 40-70µm and are readily identified by a distinct terminal spine. The eggs are eliminated from the host into the water supply with micturition. The miracidia escapes from the egg capsules and swim in the water and actively seek snails to penetrate. Upon finding a suitable snail, (*Bulinus* spp., e.g. *B. globosus*, *B. gumerei*, *B. senegalensis*, *B. forskalii*, *B. nyassanus* and *B. truncatus*). The miracidia penetrate the soft tissues of the foot. After penetration the ciliated cells disappear and transformation into the first generation or mother sporocyst takes place.

The first generation sporocysts generally remain in the foot of snail near the point of penetration: they are non motile, convoluted sacs less than 1mm long.

With 2 - 6 weeks daughter or second generation sporocysts form in the central cavity of the mother sporocyst. The daughter sporocyst are somewhat larger than the mother sporocyst and are motile. Cercariae form in the central cavity of the daughter sporocyst and leave through a pore at the posterior end. Cercariae may be produced as early as 20 days following infection.

The Cercariae tend to accumulate in the area of the mantle collar of the snail in the region where escape to the outside is simple. The Cercariae break out of the epithelium and reach the water. Unicellular **escape glands** located in the anterior portion of the body are seen only in the unemerged Cercariae and presumably are instrumental in the organisms breaking out of the snail tissue. Cercariae of the blood flukes tend to leave the snail at certain times of the day e.g. *S. haematobium* between 9.30 am and 2.00pm.

The function of the Cercariae is to move from the intermediate host through a hazardous environment to the definitive host. The Cercariae swim in the water by means of their tails and actively seek host to penetrate. They can survive about a day, but infectivity declines in a few hours if they fail to find host. When they contact human skin, Cercariae are stimulated to penetrate by warmth, skin surface lipids and light. They shed their tails and begin the process of Penetration by using the secretions of the pre and post acetabular glands. The post acetabular glands secrete a substance that is probably a mucopolysaccharide. This material spreads out from the oral sucker and allows the attachment of the organism to the skin. The Cercariae often enter the unbroken skin through hair follicles or sebaceous glands. The Cercariae penetrate the skin through the muscular action of the oral sucker. Once the organism is in the skin the pre acetabular glands secrete an alkaline secretion that causes softening of the skin's keratin layer. Successful penetration occurs in several minutes. Once the organism penetrates the skin it loses its tails and secretes the substances from the various glands and are considered to be **schistosomules**. The schistosomules are clearly different from Cercariae in their ability to tolerate high osmotic pressure, their ability to survive in serum and their appearance which is worm like. The schistosomules are carried by the circulatory system to the lungs about a day after penetration. They then migrate to the liver. Development takes place in the liver and as the worms approach sexual maturity, they begin to migrate down the hepatic portal into the smaller vessels, draining the intestine or bladder. Sexual maturity reaches as early as 7 weeks following infection depending on the species.

Egg production per female worm per day ranges from 22 – 3500. The highest figure was found in *S. japonicum*. *S. mansoni* female produces perhaps 500 eggs per day whereas *S. haematobium* probably produces fewer than 100.

Transmission Transmission occurs in stagnant or slow-moving fresh water where infected *Bulinus* snails live. Transmission rates to populations that have frequent exposure to water (e.g. fishermen, farmers working in irrigation canals, women fetching water for home use, children who swim regularly) are especially high. Anthropogenic creation of new snail habitat through building dams or irrigation canals may increase rates of transmission to nearby human populations.

Diagnosis

The majority of diagnoses are made by examination of the urine for eggs. In chronic infections, or if eggs are difficult to find, an intradermal injection of schistosome antigen to form a wheal is effective in determining infection. Alternate diagnosis can be made by complement fixation tests. The most common way to diagnose *S. haematobium* infection is by identification of ova in urine or in biopsies of the bladder, rectum, or vaginal wall. Urine analysis may also reveal blood in the urine. Infected people often have anemia, high eosinophil levels, and/or low platelets in their blood. Antibody tests are also diagnostic, although they are rarely done.

Pathogenicity: Host parasite relationship

Schistosomiasis (Bilharziasis) is unusual amongst helminth disease for two reasons: much of the pathogenesis is due to the eggs (rather than larvae or adults) : and most of the pathology is caused by host immune responses (delayed type hypersensitivity and granulomatous reactions). The course of infection is often divided into 3 phase: **migratory, acute and chronic**. The migratory phase occurs when cercariae penetrates and migrate through the skin. This is often asymptomatic, but in sensitised patients, it may cause transient dermatitis (swimmers itch) , and occasionally pulmonary lesions and pneumonitis. The acute phase (sometimes called Katayama fever) is coincide with first egg release and is characterized by allergic responses. Serum sickness due to over whelming immune complex formation, resulting in pyrexia (fever), fatigue, aches, lymphadenopathy, gastrointestinal discomfort and eosinophilia. The chronic phase occurs in response to the cumulative deposition of fluke eggs in tissues and the host reactions that develops against them. Not all the eggs laid by female worms successfully penetrate the gut or the bladder walls, many are swept away in the circulation and become trapped in the organs where they elicit strong granulomatous responses. Eggs become surrounded by inflammatory cells forming granuloma which may coalesce to form larger granulomatous reactions (polyps). The encapsulated eggs die and eventually calcify. The resultant effects on host organs and tissues are manifold, and include intestinal polyposis, abdominal pain, diarrhea, glomerulonephritis, pulmonary arteritis, cardiovascular problems including heart failure and periportal (symmers's pipe- stem) fibrosis. Portal hypertension often leads to

hepatomegally, splenomegaly, ascites and sometimes gross enlargement of oesophagus and gastric veins which may burst. Cerebral granulomas have been associated with focal epileptic convulsions, while spinal cord granulomas may cause transverse myelitis. Infection by *S. haematobium* often causes haematuria and progressive disruption of bladder wall may lead to carcinoma.

Public Health and Prevention Strategies

Education: Teaching people who live in endemic areas how to avoid contact with fresh water containing snails allows them to make choices that will help prevent infections. This is particularly important for those who frequently come into contact with water, such as women fetching water for household use, fishermen, or children who play in water. It is also important that there be a safe source of water available; education about transmission can't do much good unless people have the means to put it into practice.

Molluscides: These chemicals are used to kill snails. The human health and ecosystem consequences of applying these poisons need to be taken into consideration before they are used.

Biologic control of snails: Snail pathogens and snail predators may be introduced to reduce snail populations. It is important to consider the consequences to the ecosystem of the introduction of a new species.

Environmental modification to reduce snail habitat: Water projects can be designed to minimize or eliminate snail habitat.

Sanitation practices: Providing clean water for cooking, drinking, and bathing gives people an alternative to making contact with water that harbors cercariae. Providing modern sewage systems gives an alternative to urinating in places where eggs can be released to fresh water containing snails.

A topical drug: Niclosamide 1% lotion on skin before going in water reduces the chance of infection.

Vaccination: There is currently no vaccine available to prevent *S. haematobium* infection. Animal trials have been carried out with mixed results.

Prevention

The main cause of schistosomiasis is the dumping of human waste into water supplies. Hygienic disposal of waste would be sufficient to eliminate the disease

Treatment

The drug of choice is praziquantel, a quinolone derivative.

