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Schistosomiasis: The Scourge of the Third World. Part 1. Etiology

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Schistosomiasis is a waterborne, parasitic disease, found in much of the Third World. The World Health Organization (WHO) ranks schistosomiasis as one of the predominant tropical diseases in terms of socioeconomic and public health importance.¹ It is endemic in 74 countries and threatens as many as 600 million people as they perform daily activities related to water such as swimming, fishing, farming, washing, and bathing. In extreme cases, schistosomes, the parasites causing schistosomiasis, can survive for decades in their human host. While schistosomiasis is predominantly a disease of the rural poor, travelers to endemic areas are also subject to infection.

Schistosomes belong to the phylum Platyhelminthes, the flatworms, and are commonly known as blood flukes. Figure 1 shows the complex life cycle of the schistosome, which involves an aquatic snail intermediate host, the human or animal definitive host, and the mutual presence of both snail and definitive host in the environment in which transmission occurs.

Adult flukes live in human blood vessels, particularly those supplying the intestines, liver, and bladder. Kenneth S. Warren, director for health sciences, Rockefeller Foundation, New York, notes that while these flukes produce large numbers of eggs in the vessels of these organs, they do not multiply in the human body.² Some of the eggs pass out of the body via the urine or feces and hatch if they reach fresh water. Free-

swimming larvae, or miracidia, are then released. These miracidia need an intermediate host, a freshwater snail of a particular genus, for completion of their life cycle.

After 25 to 40 days in the snail, the miracidia develop into large numbers of infective larvae, called cercariae, which emerge from the snail. These cercariae, infective for as long as 24 hours after they are released into the water, burrow through the skin of humans they encounter in the water.

Three major species have been identified as the causative agents of schistosomiasis. There are striking differences among these three species, including geographic locations, snail hosts, patterns of egg laying, modes of transmission, disease patterns, and responses to treatment.

Schistosoma haematobium is found throughout Africa and Cyprus and is also common in the Middle East. *S. haematobium* requires snails of the genus *Bulinus* to complete its life cycle. Larry W. Laughlin, director, US Naval Medical Research Unit No. 2, Jakarta Detachment, Indonesia, notes that the origin of *S. haematobium* was probably the lake plateau of Africa.³ (p. 708)

Schistosoma mansoni, found in South America, the Caribbean, the eastern Mediterranean, and Africa, most likely originated in the Upper Nile River basin. It requires snails of the genus *Biomphalaria* as the intermediate host. *Schistosoma japonicum* probably originated in the Yangtze River Valley and requires

snails of the genus *Oncomelania*. It is endemic in Southeast Asia and in the Western Pacific.³

Although schistosomiasis is an ancient disease, Warren notes that schistosomes were identified as the causative agent relatively recently, in Egypt in 1851, by a young German pathologist, Theodor Bilharz. In fact, the older name for schistosomiasis is bilharziasis. According to Peter Jordan, director, Research and Control Department, Ministry of Health, St. Lucia, and medical parasitologist Gerald Webbe, School of Hygiene and Tropical Medicine, University of London, UK, schistosomiasis was described in the Kahun Papyrus, written about 1900 B.C.⁴ (p. 1) In 1910 M.A. Ruffer, then president of the Sanitary, Maritime, and Quarantine Council of Egypt, Alexandria, found large numbers of calcified schistosome eggs in the kidneys of Egyptian mummies from the twentieth dynasty (1250-1000 B.C.).⁵ Warren notes that the Chinese found schistosome eggs in bodies from Hunan and Hubei provinces dating back 2,000 years.⁶

Symptoms

Schistosomiasis proceeds in three stages in humans, and its severity is related to the intensity of the parasite burden. The first stage occurs soon after the cercariae penetrate the skin, producing a rash known as swimmer's itch. When the parasites migrate to the vascular system, mate, and produce eggs, an illness results known as Katayama fever, named for an endemic area of schistosomiasis in Hiroshima province, Japan. Occurring three to nine weeks after infection, this acute stage can cause complications including diarrhea, bloody stools, and enlarged liver and spleen.⁴ (p. 66)

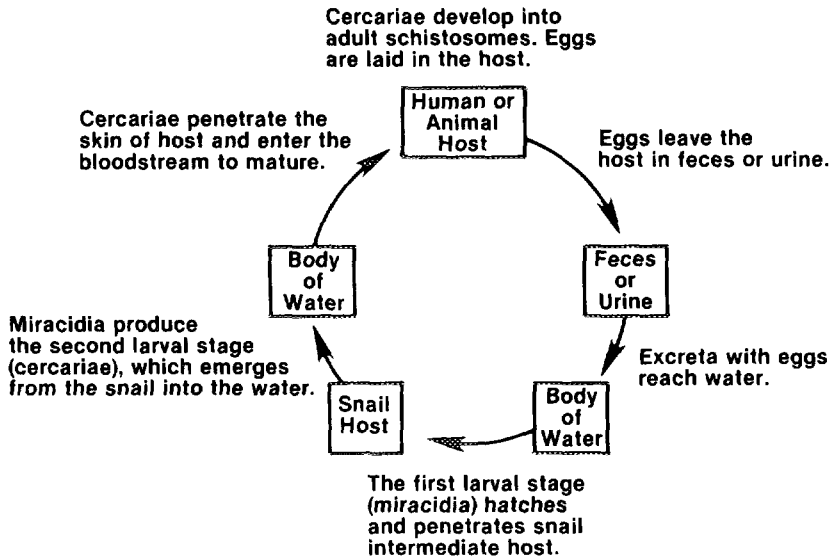
A chronic stage of infection affects those with high parasite burdens of long duration. The chronic disease results from the host's immune response to schistosome eggs deposited in tissues. Warren indicates that each pair of schistosomes can produce between 300 and

3,000 eggs each day.⁷ Some of these eggs are excreted, while others remain in the body. Eggs trapped in tissues release enzymes and other substances that spark an immune response by activating host lymphocytes and other immune cells. These cells make up compact cellular infiltrates called egg granulomas that in turn cause physical complications.³ Frequent signs of chronic schistosomiasis are the enlargement of the liver, spleen, and abdominal wall veins.

S. japonicum and *S. mansoni* are found in the mesenteric veins and the small and large intestines. Here the egg granulomas can be numerous and, according to Hari V. Iyer and colleagues, Departments of Surgery and Pathology, Methodist Hospital, Brooklyn, New York, can cause bowel obstruction. In addition, liver damage results from physical obstruction due to the granulomas or from tissue compression.⁸ In *S. haematobium* infection, egg granulomas are found in the urinary tract where they can cause obstruction. Kamal G. Ishak, Hepatic Branch, Armed Forces Institute of Pathology, Washington, DC, and colleagues note that these granulomas have been associated with the later development of bladder cancer.⁹

These distinctive symptoms have been used to speculate on the cause of the physical problems that constantly plagued Napoleon Bonaparte during the latter part of his life. In his book *Napoleon's Glands and Other Ventures in Biohistory*, Arno Karlen, former executive editor, *Physicians World*, noted that between 1798 and 1799, Napoleon spent some time in Egypt, where he was known to take long baths. After his return from Egypt, Napoleon apparently developed symptoms of urinary pain, an enlarged liver, pain in the right side, skin rashes, and swollen ankles. Karlen proposes that the Egyptian bathwaters were possibly infected with schistosomes. He notes that "it would take modern microscopic study to know if this was the case, but bilharzia does neatly fit Napoleon's great array of symptoms over two decades."¹⁰ (p. 20)

Figure 1: Schistosome life cycle.



Prevalence

Urinary schistosomiasis, caused by *S. haematobium*, has historically been the most prevalent form of the disease in Egypt. However, in a study comparing the pattern of schistosomiasis in the Nile Delta in Egypt in 1935 and in 1979, M.F. Abdel-Wahab, Department of Tropical Medicine, Cairo University School of Medicine, and colleagues report that, with the advent of artificial irrigation projects, the ecology of the snail has changed, and conditions now favor the *Biomphalaria* snail more than the *Bulinus* snail. Consequently, *S. mansoni* infection is now the most prevalent form of the disease in the Nile Delta.¹¹

While a large percentage of the population is infected in endemic areas, schistosomiasis is most prevalent in certain age groups. Fisseha Haile-Meskal, National Research Institute of Health, Addis Ababa, Ethiopia, and colleagues studied the prevalence of urinary schistosomiasis in the Middle Awash Valley of eastern Ethiopia.¹² They found that persons from 5 to 19 years of age had the

highest rate of infection associated with water-contact activities that increase from childhood through adolescence and decrease thereafter. Adel A.F. Mahmoud, Department of Medicine, Case Western Reserve University School of Medicine, Cleveland, Ohio, suggests that, aside from water contact, the development of immunity may partially explain the age-related infection pattern.¹³

The prevalence of schistosomiasis varies according to sex as well as age. These sex-related differences vary depending on the area studied and the cultural habits of the community. For example, Haile-Meskal and colleagues found that in the seminomadic Afar group, females regardless of age have more frequent water contact than males.¹² The resulting higher incidence of infection in females is attributable to occupational differences between the sexes. Females collect water plants for food and cut the tall aquatic grass for construction of houses. While the rural Afar females are more frequently infected than males, this is not the case in the city of Dar es Salaam, Tanzania. R.K. Sarda, Depart-

ment of Parasitology/Medical Entomology, Muhimbili Medical Centre, Dar es Salaam, and colleagues reported that more males than females are affected.¹⁴ The authors suggest that this sex difference may result from the boys in this community playing in streams and ponds more frequently than girls.

Environmental Factors

In emerging nations, enhancing agricultural productivity by developing and expanding water resources is an economic necessity. However, Laughlin notes that the incidence of schistosomiasis is rising due to increased use of irrigation for agricultural development, population increases and redistribution, and inadequate control measures.³ In addition, new hydroelectric and irrigation projects have created new habitats for the snail intermediate hosts in areas previously free of the disease.

H.M. Gilles, Liverpool School of Tropical Medicine, UK, and colleagues studied the effect of building a dam at Ruwan Sanyi in Nigeria and the resulting change from subsistence farming to mechanization in the Malumfashi district of Nigeria. They found a sharp increase in *S. haematobium* infection two years after the dam was built.¹⁵ In a study of *S. mansoni* and *S. haematobium* infection in Liberia six years after the establishment of swamp-rice farms, James W. Kazura, Department of Medicine, Case Western Reserve University, and colleagues reported that 87 percent of the population sampled was infected with *S. mansoni*, while 42 percent was infected with *S. haematobium*. Comparatively, in a nearby town where swamp-rice farms had not been established, the prevalence of schistosomiasis was much lower. Only 9 percent of the population was infected with *S. mansoni* and 11 percent with *S. haematobium*.¹⁶

Ecological factors play an important role in schistosome transmission. Two years after the 1973 drought in the Sahelian area of North Africa, the Gilles group found that the incidence of *S.*

haematobium infection decreased by over 20 percent.¹⁵ This period of drought decreased the amount of human contact with water and dried up the habitat of the snail host.

Generally regarded as a problem in developing countries, schistosomiasis has been brought to the attention of medical practitioners in nonendemic areas due to recent shifts in world populations. For example, the recent influx of Asian refugees from endemic areas into the US and other Western nations makes schistosomiasis a significant consideration for diagnosis. In addition, Warren warns that American tourists visiting endemic areas are not immune to the disease. In a five-year period, about 20 million Americans travel to areas where schistosomiasis is endemic.² Fortunately, Mahmoud makes it clear that the schistosome life cycle cannot become established in this country because of the absence of susceptible snails.¹³ But it is still important that physicians in the US be aware of this potential health problem.

A recent screening of the Yemeni population living in northern England yielded a 5 percent infection rate, according to P.L. Ganvir and colleagues, Eccles Health Centre, Manchester, UK.¹⁷ Similarly, 10 years earlier, Warren studied a Yemeni population in California and found that 56 percent of 218 agricultural workers examined were infected.¹⁸

Since schistosomiasis is found in much of the Third World, the question arises as to whether the economic development of those nations is hindered by the presence of this disease in the work force. The research findings are contradictory. For example, economists Robert E. Baldwin and Burton A. Weisbrod, University of Wisconsin, studied an agricultural community in the West Indian island of St. Lucia and found no significant effect on work capacity. However, this study did not measure the infection intensity.¹⁹ In a later study, M.A. Awad El Karim, London School of Hygiene and Tropical Medicine, MRC

Environmental Physiology Unit, and colleagues did measure the infection intensity and reported that infection with *S. mansoni* reduced the work capacity of canal cleaners in Managaza, Sudan, by 16 to 18 percent compared to schistosomiasis-free workers.²⁰

As we have seen, there are a variety of ecological and cultural factors involved in the transmission of schistosomiasis. Control of this disease requires both ecological and immunological measures. According to the WHO, there is evidence that humans have some degree of immunity to schistosomiasis; however, the exact nature of this immunity

and whether it plays a significant role in the control of the disease is still debated.²¹ The second part of this essay will examine the diagnosis and treatment of schistosomiasis, as well as ways of preventing the disease, including the possibility of a vaccine. We will review ISI's research fronts to learn the present status of schistosomiasis research.

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REFERENCES

1. **World Health Organization.** Schistosomiasis. *In Point of Fact* No. 24, 1984. (Newsletter.)
2. **Warren K S.** The relevance of schistosomiasis. *N. Engl. J. Med.* 303:203-6, 1980.
3. **Laughlin L W.** Schistosomiasis. (Strickland G T, ed.) *Hunter's tropical medicine.* Philadelphia: Saunders, 1984. p. 708-40.
4. **Jordan P & Webbe G.** *Human schistosomiasis.* London: William Heinemann Medical Books, 1969. 212 p.
5. **Ruffer M A.** Note on the presence of "Bilharzia haematobia" in Egyptian mummies of the twentieth dynasty [1250-1000 B.C.]. *Brit. Med. J.* 1:16, 1910.
6. **Warren K S.** Precarious odyssey of an unconquered parasite. *Natur. Hist.* 83(5):46-53, 1974.
7. -----, Water-poison disease. *World Health,* December 1984. p. 5-6.
8. **Iyer H V, Abaci I F, Rehnke E C & Enquist I F.** Intestinal obstruction due to schistosomiasis. *Amer. J. Surg.* 149:409-11, 1985.
9. **Ishak K G, Le Golvan P C & El-Sebai I.** Malignant bladder tumors associated with schistosomiasis. A gross and microscopic study. (Mostofi F K, ed.) *Bilharziasis.* New York: Springer-Verlag, 1967. p. 58-83.
10. **Karlen A.** *Napoleon's glands and other ventures in biohistory.* Boston: Little, Brown, 1984. 277 p.
11. **Abdel-Wahab M F, Strickland G T, El-Sahly A, El-Kady N, Zakaria S & Ahmed L.** Changing pattern of schistosomiasis in Egypt 1935-79. *Lancet* 2:242-4, 1979.
12. **Haile-Meskal F, Woldemichael T & Lakew M.** Endemicity of urinary schistosomiasis in Enta-doyta village, Gewane flood-plain, eastern Ethiopia. *Ethiopian Med. J.* 23:107-15, 1985.
13. **Mahmoud A A F.** Schistosomiasis. (Wynngaarden J B & Smith L H, eds.) *Cecil textbook of medicine.* Philadelphia: Saunders, 1985. p. 1809-15.
14. **Sarda R K, Simonsen P E & Mahlkwanu L F.** Urban transmission of urinary schistosomiasis in Dar es Salaam, Tanzania. *Acta Trop.* 42:71-8, 1985.
15. **Gilles H M, Greenwood B M, Greenwood A M, Bradley A K, Blakebrough I, Pugh R N H, Musa B, Shehu U, Tayo M & Jewsbury J.** The Malumfashi Project—an epidemiological, clinical and laboratory study. *Trans. Roy. Soc. Trop. Med. Hyg.* 77:24-31, 1983.
16. **Kazura J W, Neff M, Peters P A S & Dennis E.** Swamp rice farming: possible effects on endemicity of schistosomiasis mansoni and haematobia in a population in Liberia. *Amer. J. Trop. Med. Hyg.* 34:107-11, 1985.
17. **Ganvir P L, Packer J M V & Mallard R H.** Screening for schistosomiasis among the Yemeni population in Eccles. *Brit. Med. J.* 290:751-2, 1985.
18. **Warren K S, Mahmoud A A F, Cummings P, Murphy D J & Houser H B.** Schistosomiasis mansoni in Yemen in California: duration of infection, presence of disease, therapeutic management. *Amer. J. Trop. Med. Hyg.* 23:902-9, 1974.
19. **Baldwin R E & Weisbrod B A.** Disease and labor productivity. *Econ. Develop. Cult. Change* 22:414-35, 1974.
20. **El Karim M A A, Collins K J, Brotherhood J R, Dore C, Welner J S, Sukkar M Y, Omer A H S & Amla M A.** Quantitative egg excretion and work capacity in a Gezira population infected with *Schistosoma mansoni.* *Amer. J. Trop. Med. Hyg.* 29:54-61, 1980.
21. **World Health Organization, Expert Committee.** *Epidemiology and control of schistosomiasis.* Geneva: WHO, 1980. Technical Report Series, No. 643.