Fascioliasis and Fasciolopsiasis: Similar Names, Similar Diseases?

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ABSTRACT

This article reviews plant-borne helminth infections caused by *Fasciola hepatica/gigantica* and *Fasciolopsis buski*. Besides having similar names, both infections are caused by trematodes (flatworms or flukes). As with nearly all helminth infections, eosinophilia may be present, there is usually a delayed clinical presentation, and diagnosis is made with the proper identification of parasite eggs in the stool or serological testing. However, fascioliasis and fasciolopsiasis have more similarities including: egg morphology, parasite development, the involvement of aquatic plants and snails in the lifecycle, and preventive measures. Despite these similarities there are some important differences including: geographical distribution, definitive hosts, clinical presentation, and treatment. The SOF medical professional will have a greater understanding and be able to more easily identify both of these infections by being able to compare and contrast the two. Though these are not the most common helminth infections, these diseases are prevalent and may be of particular importance to providers working in Southeast Asia or South America.

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OBJECTIVES

1. Understand the epidemiology and geographic distribution of fascioliasis and fasciolopsiasis.
2. Be able to describe the lifecycle of fascioliasis and fasciolopsiasis.
3. Know the key similarities and differences in the clinical presentation, diagnosis, and treatment of fasci oliasis and fasciolopsiasis.
4. Describe prevention strategies for fascioliasis and fasciolopsiasis.

Introduction

Parasitic worms (helminths) are a great success in an evolutionary sense, but are equally successful at causing a massive burden of human and animal disease. It has been said by one renowned parasitologist, "There are more different *species* of parasitic worms in the world than there are people in the world."[1,2] Thus, many people suffer from infections of multiple worm species. Indeed, common helminth infections such as schistosomiasis, onchocerciasis, and soil-transmitted helminths (*Ascaris* species, hookworms, pinworms) affect hundreds of millions of people worldwide. These diseases receive much attention from the World Health Organization (WHO) through well-established public health programs such as the Partners for Parasite Control and Onchocerciasis Control Program.[3,4] However, other helminth infections that do not receive as much attention also contribute to the overall burden of helminth-associated disease. These helminths include "food-borne" trematode infections.[5] Two of these infections are fascioliasis (caused by *Fasciola hepatica* or *Fasciola gigantica*) and fasciolopsiasis (caused by *Fasciolopsis buski*).

Fascioliasis and fasciolopsiasis are infections caused by "aquatic plant-borne" trematodes.[6,7] In order to better understand this classification a quick review of helminth taxonomy is warranted. Helminths are classified several ways. Parasitologists classify helminths morphologically into annelids (segmented worms like leeches), nematodes (roundworms like *Ascaris lumbricoides*), and platyhelminthes (flatworms).[1] Platyhelminthes are commonly classified as cestodes (tapeworms like *Taenia solium*, pork tapeworm) and trematodes (flukes).[1] Healthcare professionals typically further classify helminths by the tissue or organ system in

Table 1. Key comparisons between fasciolopsiasis and fascioliasis[8]

Disease	Fasciolopsiasis	Fascioliasis
Organism	*Fasciolopsis buski*	*Fasciola hepatica, Fasciola gigantica*
Type of parasite	Trematode (Fluke, flatworm)	Trematode (Fluke, flatworm)
Adult	Large, up to 7cm in length	3cm in length
Eggs	130-159 x 78-98 µm	120-150 x 63-90 µm
	Oval, thin shelled, yellow, operculum not noticable	Oval, thin shelled, yellow, operculated
Human Reservoir	Intentional	Unintentional
Animal Reservoir	Pigs	Sheep
	Dogs	Cattle
		Water buffalo
Intermediate host	Snails (planorbid)	Snails (lymnaeid)
Aquatic plant	Water chesnuts, water bamboo, water caltrop	Watercress
Site of infection	Small intestine	Liver, bile ducts, skin
Clinical presentation	Diarrhea	RUQ pain
	Vomiting	Elevated LFTs
	Anorexia	Biliary colic
	Abdominal pain, intestinal obstruction	Obstructive jaundice
	Edema of face, abdominal wall, and legs	Migratory inflamation of skin (ectopic infection)
	Acities	
Diagnosis	Eggs in feces apprear in 3 months	Eggs in feces apprear in 3-4months
Geographic distribution	Rural SE Asia	Worldwide, more common in:
	Central and South China	South America (Bolivia, Ecuador, Peru)
	India	Egypt
	Thailand	Georgia
		Russia
		Vietnam
Treatment	Praziquantel	Triclabendazole - NOT FDA approved, bithionol

which they reside or cause infection (for example, schistosomes are classified as "blood flukes"). Still another classification scheme is by the parasite's intermediate host (for example, dracunculiasis is copepod-mediated, while schistosomiasis is snail-mediated). In the case of *F. hepatica/gigantica* and *F. buski*, classification is by second-intermediate host – an aquatic plant. Infections result from eating parasite-encysted plants, thus they are also food-borne infections. In addition to *Fasciola hepatica*, *Fasciola gigantica*, and *Fasciolopsis buski,* three other plant-borne trematodes cause disease. These include *Gastrodiscoides hominis*, *Watsonius watsoni*, and *Fischoederius elongates*.[6]

Fascioliasis and fasciolopsiasis have important similarities that go beyond names, taxonomy, and clinical features common to all helminthic infections. Indeed, important differences exist as well. The remainder of this article details these key similarities and differences that are summarized in Table 1.

Epidemiology

It is estimated that worldwide 40 million people have food-borne trematode infections out of the 700 million people at risk. Accordingly, the reported prevalence of fascioliasis is 2.39 million people infected out of the more than 180 million at risk.[5,9] Others place the estimated prevalence much higher: Up to 17 million people may be infected worldwide (the discrepancy in the reported and estimated prevalence is due to underreporting of the disease in many countries).[5] Over the past several decades, fascioliasis has been reported in 61 countries and has a geographic distribution throughout Asia, Africa, South America, Europe, and Oceania.[5-8] In 51 countries, the incidence of fascioliasis is increasing, making it an emerging disease throughout the world.[5,10] The following countries have the highest reported prevalence: Bolivia, Ecuador, Egypt, France, Iran, Peru, and Portugal. The most reliable data on prevalence was obtained from eight countries used in a WHO study group report (the above mentioned countries excluding France, but including China and Spain).[5] Parasitologists have studied some hyperendemic areas in depth and there is good epidemiological data from published reports. For example, the Bolivian altiplano (high plain) is a hyperendemic area in South America that has been well studied; in some communities the prevalence of people shedding eggs was 65 to 92 percent.[5,11] An estimated 360,000 people in Bolivia are infected.[5] In the nearby countries of Ecuador and Peru, as many as 20,000 and 792,000 are infected, respectively.[5] Infections occur mainly in rural areas where the consumption of aquatic plants such as watercress is high. A recent case-control study conducted in Peru, found fascioliasis to be positively associated with familiarity with aquatic plants, drinking alfalfa juice, and dog ownership.[12] Other studies revealed that females shed more eggs and have a higher rate of infection than males.[5,6] It is thought that this gender difference exists due to cultural or behavioral factors such as females being the members of the family who wash household items in water, and prepare food including aquatic plants.

Fasciolopsiasis is present mainly in Southeast Asian countries with formal epidemiological surveys taking place in the following countries: China, Taiwan, Bangladesh, India, Vietnam, and Thailand.[6,8] Fasciolopsiasis has also been detected in Laos, Cambodia, Indonesia, Singapore, Myanmar (formerly Burma), Malaysia, and the Philippines.[6,8] There are no reported cases of fasciolopsiasis in Japan or Korea.[6] Most of these studies are prevalence surveys of villages, local regions, or specific age groups making it difficult to make a general statement about the prevalence of fasciolopsiasis. In one large study in central Thailand there was an estimated 100,000 people infected in a population of 500,000.[6] Another study conducted in northern Thailand reported a prevalence of 7 percent.[6] In China, a survey that included 10 provinces found a very wide range of prevalence from 1 to 85 percent.[6] Other epidemiological relationships include females being more infected than males, and children under 15 years of age being more infected than adults. The 10 to 14-year-old age group was the most heavily infected.[6] These relationships were found in other countries too, including Thailand, Bangladesh, and Taiwan.[6] Higher rates of infection were also found to be associated with: high infection rate in pigs, one's proximity to plantations that cultivate aquatic plants, socioeconomic status, and villages that lack food inspection and sanitation programs.[6] It is hypothesized that the globalization of aquaculture is a major factor in the worldwide emergence of food-borne trematode infections since this industry has grown exponentially in recent decades.[10] Several studies have shown that the closer one lives to freshwater, the greater one's risk of having a foodborne trematode infection.[10]

In the United States, fascioliasis is rare and occurring cases are usually imported.[13] In one case series article, it was reported that 7 of 58 specimens submitted to a major U.S. reference laboratory tested positive for *F. hepatica*.[13] Likewise, there have been only few cases of imported fasciolopsiasis in the USA.[5,8]

Identification of adult flukes and eggs

Adult *Fasciola hepatica* flukes are 20 – 30mm long and 8 – 12mm wide.[14] *Fasciola gigantica* is reported to be slightly larger; however, the sizes of *F. he-*

patica and *F. gigantica* overlap. It has been reported that the life span of *F. hepatica* in humans is 9 to 13.5 years.[6] Eggs are elliptical in appearance with dimensions of 120 – 150µm by 63 – 90µm.[15] They are thin shelled and have a "cap" at top called an operculum.

F. buski is one of the largest flukes that infect humans and with a commonly reported length of 7.5cm (up to 10cm according to some parasitologists[6]) and width of 2.0cm (range 2.0 – 7.5cm by 0.8 – 2.0cm).[16] The eggs range in size from 130 to 159µm by 78 to 98µm and are operculated.[16] The operculum, however, is less noticeable in *F. buski* eggs than in *F. hepatica* eggs.

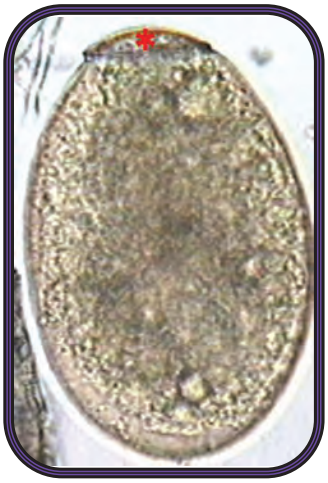

Figure 1. *Fasciola hepatica* egg. Note the crescent shaped operculum that "caps" the top of the egg (denoted in the figure by the *****). Eggs of *Fasciolopsis buski* are similar in size, shape, and color, but the operculum is usually less noticeable. Image available for public use through the Carlo Denegri Foundation Atlas of Human Parasitiology available at: http://www.cdfound.to.it/_atlas.htm.

Lifecycle, plant and animal hosts

The lifecycles of these two trematodes are nearly identical as depicted in Figure 2. Differences in the lifecycle are noted in **bold type** below.

Fasciola hepatica/gigantica: Unembryonated eggs are discharged from adult trematodes into the **biliary ducts** and thus the stool of humans or other mammals. As stool is excreted into freshwater, eggs mature and release miracidia, which invade a suitable snail intermediate host of the family ***Lymnae***. In the snail, the trematodes develop into cercariae. The snail releases cercariae and they attach to aquatic plants where they encyst as metacercariae. Humans and other mammals become infected by ingesting metacercariae attached to aquatic plants. Metacercariae exist in the duodenum and migrate through the intestinal wall, the peritoneal cavity, and the liver parenchyma into the biliary ducts, where they develop into adults. They develop into adult flukes in approximately three to four months and occupy the biliary ducts.[15]

Fasciolopsis buski: Unembryonated eggs are discharged from adult trematodes into the **intestine** and stool of humans or other mammals. As stool is excreted into freshwater, eggs mature and release miracidia, which invade a suitable snail intermediate host of the family ***Planorba***. In the snail, the trematodes develop into cercariae. The snail releases cercariae and they attach to aquatic plants where they encyst as metacercariae. Humans and other mammals become infected by ingesting metacercariae attached to aquatic plants. Metacercariae excyst in the duodenum and **attach to the intestinal wall**. There they develop into adult flukes in approximately **three months**, attached to the intestinal wall where they will live for about one year.[16]

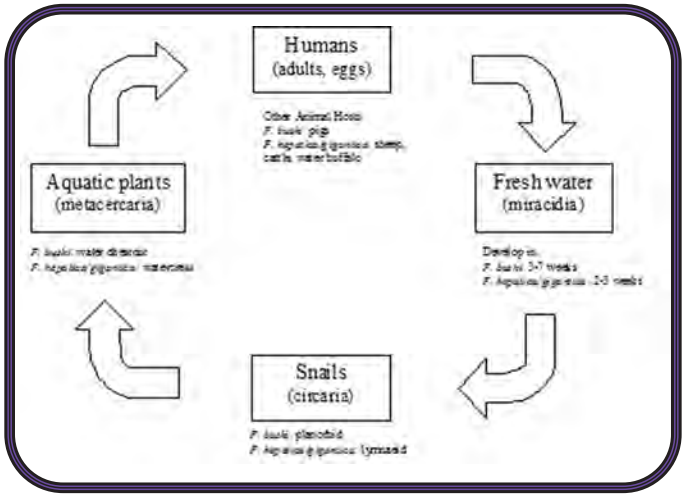

Figure 2 Lifecycle of *Fasciola hepatica/gigantica* and *Fasciolopsis buski*.

First intermediate hosts: snails

As mentioned above, Lymneid snails serve as first intermediate hosts of *F. hepatica/gigantica*. This family of snails has a worldwide distribution. *Galba truncatula* is the Lymneid mainly found in Europe, while *Pseudosuccinea columella* is mainly found in the Americas.[5,6] The ability of these Lymneid species to adapt to new environments (including some very high altitude regions) has made fascioliasis an emerging disease throughout the world.[5,6] The common Lymneids that serve as hosts for *F. gigantica* are from the genus *Radix*, which are less adaptable to new environments than the Lymneids that serve as hosts for *F. hepatica*. Thus, there is a more limited geographic distribution of *F. gigantica*.[6]

Planorbid snails of genera *Segmentia*, *Hippeutis*, and *Gyraulus* serve as first intermediate host of *F. buski*.

Depending on temperature and specific snail species, it can take 46 to 59 days for the parasite to undergo development in the snail tissues.[6] In some infected planorbid snails the mortality rate is 100 percent because developing *F. buski* causes permanent damage to snail tissues.[6] The effect on the parasite lifecycle is that there is a short time period allowed for development in the snail.[6] Curiously, there are unconfirmed instances where Planorbid snails serve as intermediate hosts of *F. hepatica*.[6]

Second intermediate hosts: aquatic plants

Watercress (*Nasturtium officinale*) is classically reported as the second intermediate plant host of *F. hepatica/gigantica*.[14,17,18] Watercress is an aquatic plant that grows in freshwater. It is a green leafy vegetable that grows in most temperate and tropical areas of the world. Wild watercress is collected and eaten, but it is also cultivated in small family gardens and farms.[18] The plant is also produced commercially on large farms and sold in supermarkets. A fascioliasis outbreak of 18 cases in France in 2002 was associated with consuming cultivated watercress produced at a commercial farm.[18] The investigation revealed that Lymneid snails were present in watercress beds as well as runoff water from a neighboring cattle farm. Other vegetables upon which metacercaraie are known to encyst includes varieties of wild, aquatic mint.[5,9] Experimental studies revealed that metacercariae preferentially encyst on plants that have a rough as opposed to a smooth epidermis.[5] These studies also found that lettuce, parsley, and clover were shown to support *Fasciola* metacercariae.[5]

Figure 3 Watercress (*Nasturtium officinale*) pictured here was obtained from a commercial farm in the United States. Watercress is the aquatic plant classically associated with fascioliasis. Cultivated watercress played a role in a fascioliasis outbreak in France in 2002. *Photographed by author, 2007.*

Water chestnut (*Eliocharis tuberosa*) is classically reported as the second intermediate aquatic plant host of *F. buski*.[5,14] It mainly grows in Southeast Asia. Other aquatic plants that serve as hosts for *F. buski* include: water caltrop (*Trapa natans* or *Trapa bicornis*), water lotus, water lily, water bamboo, water morning glory, water hyacinth, and duckweed.[5,6] Not surprisingly, *F. buski* metacercariae may also encyst upon watercress.

It is also important to know that metacercariae encyst on the water surface and there is sufficient evidence to show that human and animal infections are due to ingesting infected drinking water.[6]

Figure 4 Aquatic plant environments like this one in the Southern Philippines can harbor infective metacercariae of aquatic plant-borne trematodes. *Photographed by author, 2006.*

Definitive hosts: humans and other mammals

Fascioliasis is a disease of large grazing herbivores such as sheep, goats, cattle, buffalo, and horses. It is probably the most common helminth infection in cattle.[9] One alternative name given to the disease is "Sheep Liver Rot." Pigs, deer, elk, donkeys, camels, llamas, and rabbits are also reported as less common hosts.[6-8] Research has shown that *F. hepatica* is highly adaptable to new definitive hosts and experts regard humans as unintentional definitive hosts. Research also shows that *F. hepatica* seems to more readily infect humans than *F. gigantica*.[6]

Figure 5 Fascioliasis is probably the most common helminth infection in cattle. Infected cattle grazing near a river (in this case the Niger River in Mali) could introduce the parasites into freshwater sources. This, in turn, could lead to infection in humans when contaminated water (or plants growing in the water) is consumed.
Photographed by author, 2007.

Fasciolopsisasis is a disease in which humans are intentional definitive hosts.[6,8] Pigs are the most common non-human definitive host, but usually harbor fewer parasites than humans.[6] Rabbits, monkeys, and guinea pigs were also found to be susceptible to infection in laboratory tests. Cattle, buffalo, and horses do not serve as hosts for *F. buski*.[6] There is some debate among experts whether dogs are important definitive hosts for *F. buski*.[1,6]

Clinical presentation

In fascioliasis, immature flukes migrate from the gastrointestinal tract to the liver and biliary tract. In this acute phase of infection, symptoms may include abdominal pain, hepatomegaly, fever, diarrhea, vomiting, and eosinophilia.[14,19] Occasionally, urticaria, dypnea, cough, and dyspepsia are also present.[5] These may be the only symptoms for several months. In the chronic phase, adult flukes reside in the hepatic and common bile ducts and patients may experience biliary colic, obstructive jaundice, ascending cholangitis, and pancreatitis.[14,19,20]

Ectopic infections occur in which flukes migrate to the skin or the eye. There was a reported case in which an 11 year-old girl from Vietnam was found to have a fluke inside a puncture wound over her knee.[21] In another case from Vietnam, a small, hard, migrating puritic mass was noticed on a 48 year-old woman's chest.[21] When incised, an immature fluke appeared. In these cases, the flukes were identified using DNA analysis and found to be *F. gigantica.* There is at least one reported occurrence of *F. hepatica* infecting the anterior chamber of a woman's left eye in a highly endemic area of Iran.[22]

In fasciolopsiasis, adult flukes live in the host's duodenum and jejunum exclusively, unless there is a very heavy parasite burden. Clinical manifestations stem from the fluke's direct effects on these organs. Tissue changes include ulceration, hemorrhage, and inflammation.[6,14] The pathological changes of the bowel are manifested as clinical signs related to physical trauma, obstruction, or toxicity of the small bowel.[6,14] Light infections are usually asymptomatic, but may cause diarrhea. The diarrhea is mainly watery; however, undigested food and mucus may be present due to malabsorption. Accompanying signs and symptoms may include periods of constipation, abdominal pain, eosinophilia, headaches, dizziness, and abdominal distention. Moderate infections may cause nausea, vomiting, fever, and bowel obstruction. In heavy infections, poor appetite, malnutrition including vitamin B12 deficiency, ascities, and facial edema progressing to anasarca may be part of the presentation.[6,14] Ascites, edema, and anasarca are attributed to the body's absorption of toxic and allergic metabolites produced by the parasite.[6]

Diagnosis and treatment

In fascioliasis, the cornerstone of diagnosis is the identification of eggs in the feces. However, stool examinations may be negative in newly acquired infections (since flukes do not mature and produce eggs until about three to four months after initial infection), or in even older infections (due to the intermittent nature of fluke egg production).[8,14] Another pitfall is the possibility of "false" fascioliasis — that is, eggs are identified in the feces but are due to the patient's ingestion of infected raw liver or another food that contains eggs that are simply passing through the digestive tract in the process of digestion.[6] It is also possible to identify eggs from bilary or bile aspirate. Since egg identification can be difficult, the use of serological tests such as complement fixation and electrophoresis are available. These tests have the advantage of being positive soon after infection and much earlier than a stool exam.[6,14,23] Enzyme-linked immunoabsorbent assay (ELISA) tests are commonly used and are highly sensitive and specific. Radiographic imaging studies prove very useful in the diagnosis of fascioliasis. Ultrasound and computed tomography are the most commonly used modalities. Findings include: common bile duct and intrahepatic bile duct dilatation, bile duct wall thickening, nodular lesions, and flukes in the gallbladder.[20,24] Saba et. al., classify fascioliasis based on motile echogenic images and symptom duration.[25]

Acute fascioliasis is defined as symptoms of infection for a duration of less than four months and no motile echogenic images in the gallbladder. Chronic fascioliasis is defined as symptoms of infection for a duration of longer than four months or motile echogenic images in the gallbladder. Endoscopic retrograde cholangiopancreatography (ERCP) is both diagnostic and therapeutic. Findings in this study include: motile curved filling defects and irregularities of the bile duct wall. Once identified, one can extract flukes using a balloon or basket. In one case, 20 flukes were removed from a single patient.[20] Magnetic resonance cholanigography proved useful in at lease one case to monitor a patient's response to drug therapy.[26] Percutaneous liver biopsy is not useful since it rarely reveals eggs or parasites.[20]

In fasciolopsiasis, diagnosis cannot be made based on clinical signs and symptoms alone. Diagnosis is made by the identification of eggs in the feces that resemble those of *F. hepatica*. It is rare to recover actual flukes from a patient; however, there was one reported case in which a Vietnamese boy vomited several flukes.[27] Sequences of DNA extracted from these flukes were compared to sequences of known *F. buski* DNA in the GenBank database. The sequences were nearly identical, thus confirming the diagnosis of fasciolopsiasis.

Drug therapy for fascioliasis includes the use of triclabendazole, bithionol, and praziquantel. Among these triclabendazole has proven especially efficacious in the treatment of fascioliasis.[28] Studies showed that the cure rate was 79 percent after one dose and 93-100 percent after two doses.[6] Unfortunately, triclabendazole for human use (Egaten®, Novartis, Basel, Switzerland) is not commercially available in the United States. However, triclabendazole for veterinary use (Fasinex®, Novartis, Basel, Switzerland) is available and has been used effectively to treat livestock since 1983.[13] The mechanism of action of triclabendazole is not completely understood, but the drug probably acts to disrupt the fluke's microtubules.[29,30] In addition, triclabendazole seems to inhibit the release of proteolytic enzymes. These effects cause immobility and death of the fluke. Once absorbed (a fatty meal will increase absorption) it reaches peak serum concentrations in eight hours.[29] Most of the drug (and its metabolites) is eliminated in the feces. In animal studies, there have been no adverse fetal effects. Triclabendazole is excreted in breast milk. Side effects may include transient diarrhea, and abdominal cramps. Treatment with triclabendazole may induce biliary obstruction, which may occur due to dying flukes. Though dosage has not been established, a single dose of 10mg/kg body weight is the usual pediatric and adult dose.[29,31,32] In more severe cases, two doses at 20mg/kg body weight 12 hours apart is recommended.[29] Triclabendazole, manufactured for humans, comes in 250mg tablets. Tablets should be stored at less than 104 degrees Fahrenheit (40 degrees Celsius).[29] SOF healthcare providers should know that triclabendazole in not an FDA approved medication. However, it is important to be familiar with the drug because foreign healthcare providers may use it.

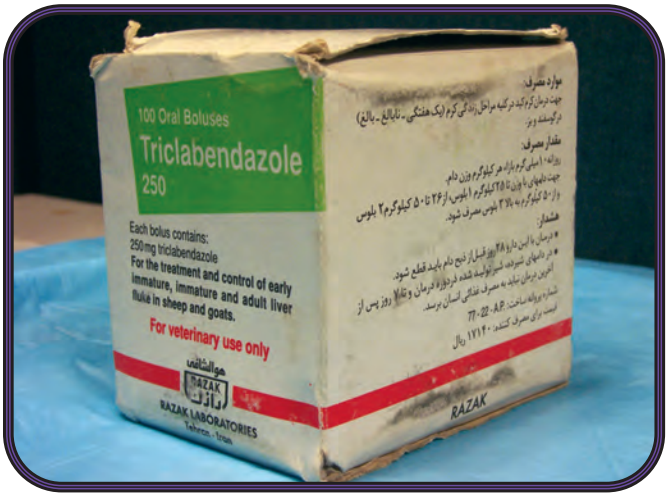

Figure 6 Triclabendazole pictured here is the drug of choice for the treatment of fascioliasis. However, it is not FDA-approved for human use. This veterinary formulation was manufactured in Iran where fascioliasis is common (in animals and humans) in areas near the Caspian Sea.
Photographed by author, 2007.

Bithionol is an investigational drug and is available through the CDC Drug Service.[33] Its mechanism of action involves blocking oxidative phosphorylation of the parasite.[31] The dosing regimen for bithionol is 30 to 50mg/kg/day divided into three times per day on alternating days for 5 to 15 days.[31,32] Side effects of bithionol include nausea, vomiting, loss of appetite, abdominal pain, dizziness, photosensitivity, and purtitis.[31] One or more of these side effects may be present in 50% of patients taking bithionol.[13]

Praziquantel is the drug of choice for treatment of fasciolopsiasis. Praziquantel (Biltricide®, Bayer, West Haven, CT) acts to increase the fluke's cell membrane permeability.[31,34] Increased permeability leads to the loss of intracellular calcium. This causes rapid muscle contraction and paralysis. In addition, bleb formation and vacuolization occurs, leading to disintegration of the tegument. Praziquantel is rapidly absorbed, undergoes extensive first-pass metabolism, is highly protein binding, reaches peak serum concentrations in less than 1.5 hours, has a half-life of one to three hours, and is excreted in the urine.[34] It is a FDA Pregnancy Category B drug.

It is excreted in breast milk and nursing mothers are advised to stop breast feeding for 72 hours once the drug is started. Potential side effects include: dizziness, drowsiness, fever, abdominal cramps, nausea, vomiting, (bloody) diarrhea, increased sweating, and skin rash.[31,34] Contraindications include known praziquantel hypersensitivity and ocular cysticercosis (permanent ocular lesions result from death of parasites in the eye).[34] Key drug interactions (through the induction of cytochrome p450 enzymes) that act to decrease serum levels of praziquantel include: rifampin, phenytoin, and dexamethasone.[34] Drug interactions that increase praziquantel levels (through the decrease of cytochrome p450 enzyme activity) include: ketoconazole, cimetidine, and erythromycin.[34] Caution should be exercised when using praziquantel in patients with liver disease, as they will have increased serum drug concentrations. For fasciolopsiasis, the dose is 25mg/kg three times a day (dose spaced 4 to 6 hours apart) for one day.[31,32,34] This is the accepted dose for adults and children over four years of age. Dosing has not been established in children younger than four years of age. Praziquantel comes in 600mg tablets scored to allow use of ¼ of a tablet. Tablets should be stored at less than 86 degrees Fahrenheit (30 degrees Celsius).

Prevention strategies

Knowledge of specific foods that may contain infectious metacercariae is important for individual avoidance and protection. Khat (*Catha edulis*) is a plant that is chewed for its psychotropic effects. It is popular in Yemen and Horn of Africa nations. It requires moisture to thrive and serves as a host plant for *F. hepatica*. The practice of storing freshly picked plants in wet banana leaves probably contributes to the persistence of metacercariae.[14,35] Zeitoon-parvardeh is an appetizer served in Iran. It is prepared by mixing a wild aquatic plant called choochagh (*Eryngium coucasicum*), walnuts, olives, garlic, and spices.[17] It is usually eaten immediately or stored for up to two weeks.[17] Delar is another food served in Iran.[17] It is a paste prepared by mixing an aquatic plant called khlivash (*Mentha pulegium*) with salt. This preparation can be stored and eaten over several months. Experimental studies have shown that foods prepared with raw liver such as liver-sushi in Japan, or a dish called "tab-wan" (raw liver, lemon and chili) from Thailand may cause infection.[36]

There are many traditional food preparation and storage practices that contribute to the presence of plant-borne trematode infections. Targeting changes in these practices may help prevent disease. The most common practice is simply eating raw plants that are infected. Another practice is the tradition of peeling water caltrop with one's teeth prior to consumption.[1,14] The storage of aquatic plants in basins of water or damp covers for an extended period of time is yet another practice that may propagate infection. Several studies have examined methods of food treatment to include: chemical treatments, drying, heating, freezing, and irradiation.[17] To different extents these treatments will detach, inactivate, or kill metacercariae (for a comprehensive review see the article by Ashrafi et. al.).[17] The quickest and most effective ways to kill or inactivate *F. buski* metacercariae are boiling; soaking plants in vinegar, salt solution, or soy sauce; and exposure to direct sunlight.[17] Barriers to using effective treatments include extended preparation time and changes in food characteristics such as taste, temperature, and texture.[17]

There are many community level prevention strategies available to halt plant-borne trematode infections. At the heart of prevention are the use of clean water and the proper treatment of human and animal excreta. Programs to increase disease awareness among consumers, producers of raw aquatic plants, and livestock farmers also play a role.[6] Controlling agriculture practices such as curbing the runoff from animal farms into watercress beds, and stopping the practice of using night soil or other untreated manure is also necessary. The use of molluscicides to kill snail hosts may also be an effective strategy.[8] In some endemic areas, mass chemotherapy of livestock, humans, or both have been advocated. As the aquaculture industry grows, proper regulations need to be put in place to halt the continued emergence of plant-borne trematode infections. Currently, there are no vaccines available to protect against plant-borne trematode infections, but Polish researchers have developed a recombinant enteral vaccination that was up to 80 percent effective in preventing *F. hepatica* infection in rats.[37]

Conclusion

The SOF healthcare professional should be familiar with fascioliasis and fasciolopsiasis because deployed Soldiers living in austere conditions in endemic areas are at risk of acquiring these infections. Fascioliasis and fasciolopsiasis are two infections caused by trematodes that have a similar lifecycle involving snails, water, and aquatic plants. Egg morphology and symptoms of early infection are also similar. Key differences include adult fluke size, geographic distribution, late clinical presentation, and treatment drug of choice. Disease transmission occurs when parasites, snails, water, aquatic plants, and humans (or other mammalian definitive hosts) meet to complete the lifecycle. Prevention and control of

these diseases depend on breaking the parasite lifecycle. Knowledge of local foods, consumption of clean water, and avoidance of raw or undercooked aquatic plants in endemic areas are the best strategies for prevention in individuals. Sanitary agriculture practices, molluscicides, and mass chemotherapy are large scale, community-based, programs that may stop disease transmission. SOF healthcare professionals should suspect disease transmission in endemic areas and look for opportunities to implement these community prevention programs whenever possible.

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