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# Fascioliasis Due to *Fasciola hepatica* and *Fasciola gigantica* Infection: An Update on This 'Neglected' Neglected Tropical Disease

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## Abstract

Fascioliasis, an infection due to the food- and water-borne trematodes *Fasciola hepatica* and *Fasciola gigantica*, is among the most neglected of the neglected tropical diseases. Among the estimated 91.1 million humans at risk for infection worldwide, as many as 17 million may be infected. Certain areas of the world bear the burden of the highest prevalence of infection. There, school-age children are the most likely to be infected. In the United States, rare cases have been reported among immigrants from endemic areas, returned travelers to endemic regions, and individuals residing in Hawaii, California, and Florida. Indigenous cases have almost always been associated with consumption of watercress. Diagnosis is made serologically most often, although stool examination for the eggs is fruitful if obtained when the adult worm is laying eggs. With an appropriate index of suspicion, laboratory and imaging studies often confirm the suspected diagnosis. Triclabendazole is the treatment of choice.

**Keywords:** Fascioliasis, *Fasciola hepatica*, *Fasciola gigantica*, trematodiasis, neglected tropical diseases, liver fluke

After reading this article, readers should be able to recognize the different morphologies of eggs from a variety of human parasites, understand the role of laboratory testing in the diagnosis of human fascioliasis, and appreciate the clinical and epidemiologic circumstances in which the diagnosis of infection with *Fasciola* is appropriate.

**Microbiology exam 71101** questions and corresponding answer form are located after this CE Update on page ##.

The neglected tropical diseases (NTD) comprise a group of (mostly infectious) conditions uncommonly recognized or diagnosed in the United States and the developed world; are less well understood than more common infections due to a of lack of research interest and/or insufficient funding; and, lastly, remain mysterious or unknown to health care providers because of minimal or no instruction regarding the diseases during our training.[1] However, the outlook for the NTD is improving. For example, a new journal, *PLoS Neglected Tropical Diseases*, recently appeared with support of the Bill and Melinda Gates Foundation, which is also a pioneer in supporting the study of these diseases.[2] However, certain food-borne trematode infections in particular remain "neglected" NTD, according to the World Health Organization.[1] These include clonorchiasis (Chinese liver fluke disease), fascioliasis (sheep liver fluke disease), opisthorchiasis (fish liver fluke disease), and paragonimiasis (lung fluke disease). These diseases most often significantly affect large numbers of poverty-stricken individuals, generally in resource-limited regions, and receive very little interest from funding or government agencies.[1] As these diseases have complex life cycles and are rarely encountered in the United States, they receive little attention in the education of physicians, which furthers their enigmatic status.[3] This continuing education update will address the most commonly encountered (in the United States) of these "neglected" NTD, fascioliasis. The interested reader is referred to recent reviews of this topic for further information.[3-11]

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## Abbreviations

NTD, neglected tropical diseases; Fh, *Fasciola hepatica*; Fg, *Fasciola gigantica*; FBT, food-borne trematodiases; FAST-ELISA, Falcon assay screening test - enzyme-linked immunosorbent assay; EITB, enzyme-linked immunoelectrotransfer blot; ERCP, endoscopic retrograde cholangiopancreatography; MRCP, magnetic resonance cholangiopancreatography

## *Fasciola* Epidemiology

Fascioliasis, a food- or water-borne trematodiasis due to infection by *Fasciola hepatica* (*Fh*) or *F. gigantica* (*Fg*), is currently

**Glossary**

| | |
|---|---|
| **Autochthonous** | Originating where found; indigenous |
| **Coproantigens** | Antigens found in stool specimens |
| **Coprological** | Having to do with stool (for example, stool studies) |

believed to affect as many as 17 million people worldwide,[12] with 91.1 million individuals at risk for infection.[4] *Fasciola hepatica* infects humans on all continents (except Antarctica), having the widest latitudinal, longitudinal, and altitudinal distribution of the food-borne trematodiases (FBT) and other parasitic and vector-borne diseases.[13] In contrast, *Fg* infection is more geographically constricted, occurring in the tropical regions of Africa, the Middle East, and Asia, where infection due to either species may occur.[5] Contrary to early thinking, prevalence of veterinary disease is not predictive of prevalence of human disease in endemic regions.[13] Prior to 1980, fascioliasis was thought to be a zoonosis of only mild, sporadic, or local importance. In the past 20 years however, a more complete understanding of disease epidemiology has emerged.[14] Mas-Coma and colleagues have proposed an epidemiologically useful classification, based on more recent studies, to better understand fascioliasis.[14]

Imported cases are those acquired in an endemic area but diagnosed in a region lacking indigenous fascioliasis, even in animals. Imported fascioliasis in the returned traveler exemplifies this situation.

Autochthonous, isolated, non-constant cases occur in areas where animal and human infections are recognized but are sporadic and not endemic. Indigenous cases resulting from ingestion of contaminated watercress in Hawaii are examples.

Endemic regions can be described as hypoendemic, mesoendemic, or hyperendemic, depending on whether coprological diagnosis on inhabitants reveals a prevalence of less than 1%, 1%-10%, or greater than 10%, respectively. Typically, in hypoendemic regions sanitation usually involves latrines or sewage disposal systems and outdoor defecation is unusual. In contrast, in hyperendemic areas, indiscriminate defecation is common, as latrines and waste management systems are absent. Human egg shedding contributes significantly to disease transmission in this circumstance.

Epidemic outbreaks can be characterized as occurring in areas where fascioliasis is endemic in animals but not humans and in areas where the disease is endemic in both animals and humans.

Areas of the world with the highest prevalence of fascioliasis include the highlands of Bolivia (the Bolivian Altiplano has the highest prevalence in the world[14]), Ecuador, and Peru; Cuba; Portugal and Spain; Turkey; the Nile Delta of Egypt and elsewhere in Africa; the northern regions of the Islamic Republic of Iran and elsewhere in Asia; and central Vietnam.[7]

Imported fascioliasis in the United States (and other developed countries) has been extensively reviewed.[8,15-19] Twenty-three cases of imported fascioliasis diagnosed in the United States have been summarized.[19] A handful of indigenous cases, primarily associated with consumption of contaminated watercress, have been identified in Hawaii, California, and Florida, as well.[19] Most cases seen in the United States will involve immigrants from endemic areas and returned travelers infected while visiting such regions (**Figure 1**).

The life cycle of *Fasciola* is shown in **Figure 2**.[20] Understanding the interaction between the parasite and its accidental human host at a molecular pathophysiological level is resulting from recent investigations.[21] Unlike the other FBT, in which consumption of raw or undercooked seafood is necessary for infection, humans most often become accidental hosts of *Fasciola* when they ingest aquatic vegetation on which the metacercariae have encysted. Implicated plants include (most commonly) watercress,[6] water morning glory (pak boong),[8] other aquatic plants, salads in endemic areas such as the highlands of South America,[22] and alfalfa juice (which is used as a medicinal tonic in Peru).[23] Another source of infection is drinking water contaminated with free-living (non-encysted) metacercariae. The least common mode of transmission is the consumption of raw or undercooked liver infected with immature or adult forms of the worm.

**Figure 1_Key Points**

- Most cases of fascioliasis seen in the United States will occur in immigrants and travelers returning from endemic regions
- Most cases worldwide occur in school-age children living in endemic areas (but those cases do not come to medical attention in the United States)
- Unlike other FBT, fascioliasis most often follows ingestion of contaminated aquatic vegetation or water—so vegetarians are at risk
- A high index of suspicion is necessary
- Acute disease is typified by fever, abdominal pain, and eosinophilia but no eggs in the stool
- Chronic disease is most often asymptomatic, but eggs may be shed and serology is positive
- Human *Fasciola* infection cannot be speciated on the basis of egg size
- Imaging studies often support the laboratory diagnosis

## Clinical Manifestations

Given that more than half of cases are subclinical (asymptomatic),[6] human infection can be classified as acute or chronic based upon clinical manifestations and laboratory findings. These 2 classifications have been further subdivided into 6 phases (**Table 1**).[7] In a review of 173 cases,[15] clinical manifestations included abdominal pain in 125 (72%), fever in 99 (57%), constitutional symptoms in 76 (44%), urticaria in 22 (13%), itching in 17 (10%), respiratory symptoms in 14 (8%), headache in 8 (5%), and cardiac symptoms in 2 (1%). Laboratory and other evaluations revealed eosinophilia (>500/mm$^3$) in 96%, leukocytosis (>10,000/mm$^3$) in 64%, eggs in feces in 40%, and positive computed tomography in 70%. In chronic infection beyond the latent phase, clinical manifestations are those of the complications of fascioliasis, namely ascending cholangitis, cholelithiasis, cholecystitis, pancreatitis, biliary cirrhosis, and hepatic fibrosis. Unlike other FBT, fascioliasis has not yet been associated with the development of cholangiocarcinoma.[24] Typical fascioliasis results from the migration of immature worms through Glisson's

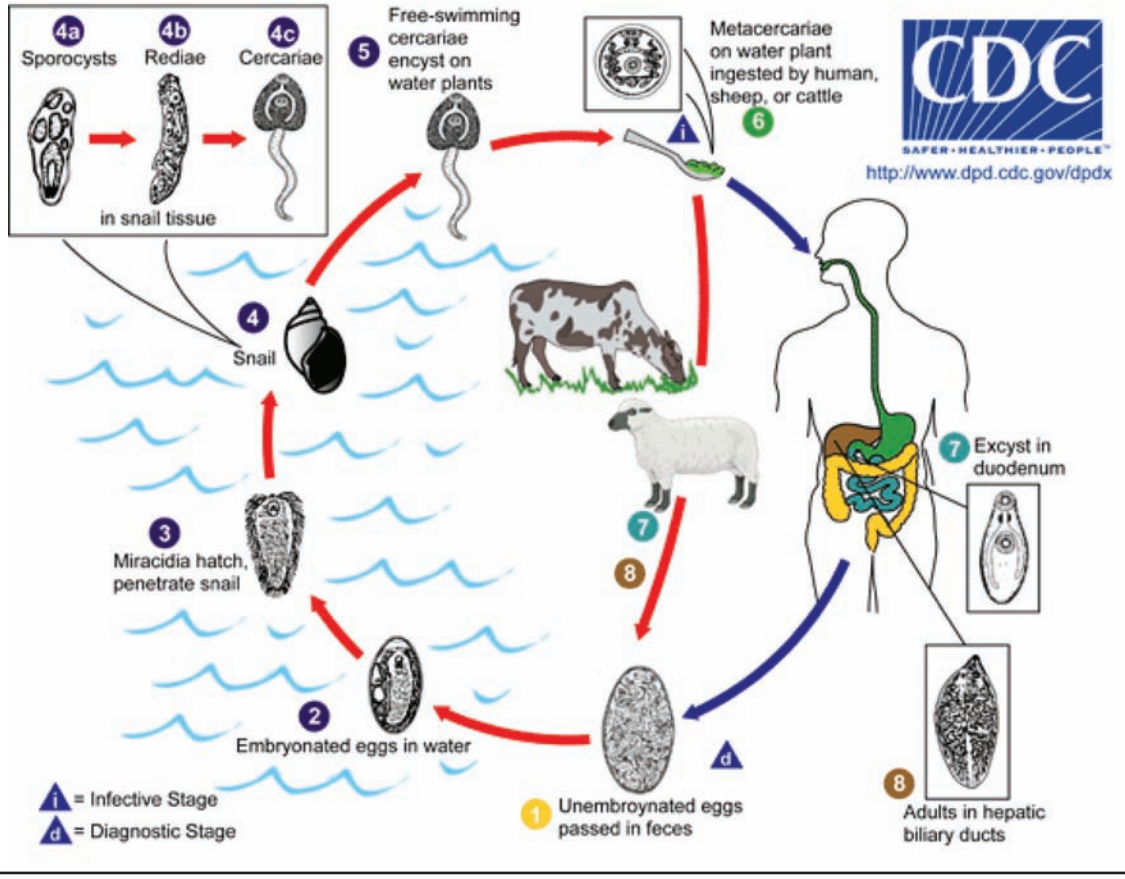


**Figure 2**_Life cycle of *Fasciola.* Immature eggs are discharged in the biliary ducts and in the stool ❶. Eggs become embryonated in water ❷, eggs release miracidia ❸, which invade a suitable snail intermediate host ❹, including the genera *Galba*, *Fossaria*, and *Pseudosuccinea*. In the snail the parasites undergo several developmental stages (sporocysts ❹a, rediae ❹b, and cercariae ❹c). The cercariae are released from the snail ❺ and encyst as metacercariae on aquatic vegetation or other surfaces. Mammals acquire the infection by eating vegetation containing metacercariae. Humans can become infected by ingesting metacercariae-containing freshwater plants, especially watercress ❻. After ingestion, the metacercariae excyst in the duodenum ❼ and migrate through the intestinal wall, the peritoneal cavity, and the liver parenchyma into the biliary ducts, where they develop into adults ❽. In humans, maturation from metacercariae into adult flukes takes approximately 3 to 4 months. The adult flukes (*F. hepatica*: up to 30 mm by 13 mm; *F. gigantica*: up to 75 mm) reside in the large biliary ducts of the mammalian host. *F. hepatica* infect various animal species, mostly herbivores. From the Centers for Disease Control and Prevention Laboratory Identification of Parasites of Public Health Concern website.[20]

**Table 1_Classification of Fascioliasis by Clinical Manifestations and Laboratory Findings**

| | Phase | Time Frame | Pathophysiology | Clinical Manifestations | Laboratory Findings |
|---|---|---|---|---|---|
| Acute | Incubation | Few days to few months | From ingestion of metacercariae to appearance of symptoms when they reach the liver parenchyma or other ectopic site | None | None |
| | Invasive | 2-4 months | Worms migrate through liver tissue, causing necrosis, hemorrhage, and inflammation | Fever, abdominal pain, gastrointestinal disturbances, rashes, cough, hepatosplenomegaly, ascites, jaundice, anemia, right upper quadrant tenderness | High levels of eosinophils and immunoglobulin E often noted. Imaging and/or histopathologic studies often abnormal |
| | Latency | Months or years | Parasites mature and start to lay eggs when they reach the bile ducts | None or mild gastrointestinal disturbances with vague abdominal pain or tenderness | Intermittent eosinophilia may occur; eggs are shed in the stool intermittently |
| | Obstructive | Months or years | Parasites, fragments, and/or debris obstruct the biliary tree | Biliary colic, nausea, epigastric pain, jaundice, fever, right upper quadrant tenderness | Leukocytosis with cholangitis; anemia may ensue; imaging may be consistent with cholecystitis or reveal worms in the bile ducts |
| Chronic | Advanced chronic | Years | Consequences of long-term obstruction include calculi, bacterobilia, cholangitis, and cholecystitis | Chronic cholecystitis and cholangitis | Findings consistent with cholecystitis or cholangitis; egg laying ceases |
| | Post-infectious | Years | Most flukes have died, but calculi may persist with biliary cirrhosis and gallbladder atony | Chronicity of cholecystitis and/or cholangitis | Findings consistent with chronic cholecystitis or cholangitis may be seen |

**Figure 3_Reference Laboratory for *Fasciola* Serology**

Falcon assay screening test—enzyme-linked immunosorbent assay (FAST-ELISA) and enzyme-linked immunoelectrotransfer blot (EITB)[28,29]

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Reference Laboratory for *Fasciola* Immunofluorescence on Worm Sections

For clinical consultation and immunofluorescence on worm sections:

Professor Jean Dupouty-Camet
Parasitologie-Mycologie
UFR Cochin
27 Fauberg St. Jacques, 75014
Paris, France

Telephone: 33-1-4234-1497
Fax: 33-1-4234-1496
Email: dupouyca@imaginet.fr and
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capsule and the liver parenchyma en route to the bile ducts, where they mature and take up permanent residence. Occasionally the metacercariae reach ectopic destinations, resulting in a cutaneous or visceral larva migrans picture reminiscent of strongyloidiasis, taeniasis, or gnathostomiasis.[25-27] The clinical findings with ectopic migration will depend upon the organs or tissues invaded.

## Diagnostic Approach to Fascioliasis

Clearly, the appropriate diagnostic approach to the patient with possible fascioliasis depends first and foremost on a high index of suspicion, as well as the stage of the infection and the resources and expertise available.

## Laboratory Evaluation

Eosinophilia, leukocytosis, and elevated inflammatory markers are common in acute infections (**Table 1**); anemia and/or elevated serum hepatic transaminases, bilirubin, or alkaline phosphatase are only occasionally transiently present during chronic infection. Within 2 to 4 weeks of becoming infected, a *Fasciola*-specific serologic response ensues, allowing confirmation of infection 5 to 7 weeks before eggs appear in



**Table 2_Differential Morphology of the Diagnostic Stages of Helminths Found in Humans: Eggs (Trematodes)**

| Species | Size | Shape | Color | Stage of Development When Passed |
|---|---|---|---|---|
| *Schistosoma mansoni* | 140 µm × 66 µm Range, 114-180 µm × 45-73 µm | Elongated with prominent lateral spine near posterior end. Anterior end tapered and slightly curved. | Yellow or yellow brown | Embryonated. Contains mature miracidium. |
| *Schistosoma japonicum* | 90 µm × 70 µm Range, 68-100 µm × 45-80 µm | Oval. Small lateral spine is often seen or may appear as a small hook or "knob" located in a depression in the shell. | Yellow or yellow brown | Embryonated. Contains mature miracidium. |
| *Schistosoma haematobium* | 143 µm × 60 µm Range, 112-170 µm × 40-70 µm | Elongated with rounded anterior end and terminal spine at posterior end. | Yellow or yellow brown | Embryonated. Contains mature miracidium. |
| *Schistosoma intercalatum* | 175 µm × 60 µm Range, 140-240 µm × 50-85 µm | Elongated with tapered anterior end and terminal spine. Sometimes spindle-shaped. | Yellow or yellow brown | Embryonated. Contains mature miracidium. |
| *Schistosoma mekongi* | 69 µm × 56 µm* Range, 51-73 µm × 39-66 µm | Spherical. Small lateral spine, not always visible or may appear as a small "knob" in a depression in the shell. | Yellow or yellow brown | Embryonated. Contains mature miracidium. |
| *Clonorchis sinensis* | 30 µm × l6 µm Range, 27-35 µm × 11-20 µm | Small, ovoidal, or elongated with broad rounded posterior end and a convex operculum resting on "shoulders." A small "knob" may be seen on the posterior end. | Yellow brown | Embryonated. Contains mature miracidium. |
| *Opisthorchis felineus* | 30 µm × 12 µm Range, 26-30 µm × 11-15 µm | Elongated with operculum on anterior end and pointed terminal "knob" on posterior end. | Yellow brown | Embryonated. Contains mature miracidium. |
| *Heterophyes heterophyes* | 28 µm × 15 µm Range, 28-30 µm × 15-17 µm | Small, elongated, or slightly ovoidal. Operculum. Slight "knob" at posterior end. | Yellow brown | Embryonated. Contains mature miracidium. |
| *Metagonimus yokogawai* | 28 µm × 17 µm Range, 26-30 µm × 15-20 µm | Small, elongated, or ovoidal. Operculum. No "shoulders" at anterior end. Small "knob" often seen on posterior end. | Yellow or yellow brown | Embryonated. Contains mature miracidium. |
| *Paragonimus westermani* | 85 µm × 53 µm Range, 68-118 µm × 39-67 µm | Ovoidal or elongated with thick shell. Operculum is slightly flattened and fits into shoulder area of shell. Posterior end is thickened. Egg often asymmetrical with 1 side slightly flattened. | Yellow brown to dark brown | Unembryonated. Filled with yolk material in which a germinal cell is imbedded. Cells are irregular in size. |
| *Fasciola hepatica* | 145 µm × 80 µm Range, 120-150 µm × 63-90 µm | Ellipsoidal, thin shell. Small, indistinct operculum. | Yellow to light brown | Unembryonated. Filled with yolk cells in which an indistinct germinal cell is imbedded. |
| *Fasciolopsis buski* | 140 µm × 80 µm Range, 130-159 µm × 78-98 µm | Ellipsoidal, thin shell. Small, indistinct operculum. | Yellow brown. | Unembryonated. Filled with yolk cells in which an indistinct germinal cell is imbedded. |

*From the Centers for Disease Control and Prevention Parasites and Health Web site,[41] based on sizes of eggs in human fecal specimens.[42,4]*

the feces. In the United States, serology is not commercially available and the Centers for Disease Control and Prevention refers *Fasciola* serology requests to a reference laboratory (**Figure 3**).[28,29] Despite some cross-reactivity of the Falcon assay screening test-enzyme-linked immunosorbent assay (FAST-ELISA) with serum from those with schistosomiasis (which generally can be clarified by the clinical scenario), the sensitivity of the test is reported to be 95%.[30] The sensitivity of the confirmatory enzyme-linked immunoelectrotransfer blot (EITB), using 12-, 17-, and 63-kiloDalton antigens, is reported to be 100%.[30] Positive serology can also be used to document infection in the chronic phase, when egg release may be intermittent or absent. Serology should revert to negative within a year of successful treatment. A variety of serodiagnostic methods have been described,[31-39] including 2 commercially available testing kits available in Europe and elsewhere.[38,39]

An ELISA assay to detect *Fh* coproantigens in preserved human stool samples has been described,[40] but the standard fecal study for the diagnosis of fascioliasis is the identification of eggs in the stool. In hyperendemic settings, children may shed thousands of eggs per gram of feces.[3] A tabular comparison (**Table 2**)[41-43] and **Figure 4**[41] illustrate the similarities and differences among the eggs of most human trematodes. **Figure 5** shows unstained wet mounts of *Fh* eggs.[20] Infection due to *Fh* and *Fg* classically has been differentiated by the geographic distribution of the organisms, when the 2 do not overlap. In regions endemic for both, however, it was thought that the size of the eggs shed in the feces could differentiate the 2 kinds. In livestock, the eggs of *Fg* are significantly larger than those shed by *Fh*, and this was thought to be true for human shedding as well. Recent work corrects this teaching, demonstrating that *Fh* eggs shed by humans are larger than those shed by livestock, while *Fg* eggs shed by humans are smaller than those shed by livestock, with considerable overlap. Thus, one cannot speciate human fascioliasis on the basis of the size of the eggs shed in the stool.[44]

Adult worms are found in the biliary tract, while migrating larvae can be found anywhere in the human host (most often in the skin or other sites of ectopic migration). Rarely, a worm will be retrieved from the patient (for example, by endoscopic retrograde cholangiopancreatography [ERCP]).[45] A comparison among the adult visceral trematodes of humans is demonstrated in **Figure 6**.[41] Unstained, formalin-fixed and carmine-stained adult *Fh* worms are shown in **Figure 7**.[20] An immunofluorescent antibody test with 90% sensitivity and specificity can be performed on worm sections, if the diagnosis is in doubt (**Figure 3**).

| Specific Features and Variations |
|---|
| Lateral spine. Found in feces; in rare cases, in urine also. Eggs are discharged at irregular intervals and may not be found in every stool specimen. They are rare in chronic stages of infection. |
| Found in feces. Often coated with debris and may be overlooked. |
| Terminal spine. Found in urine, occasionally in feces. Egg often covered with debris. |
| Terminal spine long, slender with bent tip. Resembles *S. haematobium* egg except it is longer, is thinner, and has a longer spine. Found in feces. May have debris adhering to shell. |
| Found in feces. Closely resembles *S. japonicum* egg except it is smaller. May be coated with debris. |
| Small size, operculum and "knob" on posterior end. Shell often is covered by adhering debris. |
| Lacks prominent shoulders characteristic of *Clonorchis* and has more tapered end. |
| Resembles *Clonorchis* egg but with less distinct "shoulders." Operculum is broader than in *Clonorchis*. |
| Resembles *Clonorchis* and *Heterophyes* eggs. Shell is slightly thinner than *Heterophyes*. Operculum is broader than *Clonorchis*. |
| Found in sputum, occasionally in feces. Resembles egg of *D. latum* but is larger, slightly asymmetrical, and the operculum is smaller and flatter. The widest part of the *Paragonimus* egg is usually anterior to the center; in a *D. latum*, the widest area is around the center. |
| Large size. Broadly oval eggs. |
| Large size. Resembles *F. hepatica* egg and cannot be easily distinguished from *Fasciola*. |

## Diagnostic Imaging

In the appropriate clinical context, imaging studies can be very useful to confirm the diagnosis of fascioliasis.[46-48] Typical findings include subcapsular hemorrhage; parenchymal nodular, ill-defined lesions, which may coalesce into tortuous or tubular tracks; filling defects in the biliary tract; and subcapsular, peribiliary, or periportal indistinct nodules and abscesses. Ultrasound, computed tomography, magnetic resonance imaging, and, to a lesser extent, nuclear medicine scanning all have a role. Endoscopic retrograde cholangiopancreatography and magnetic resonance cholangiopancreatography (MRCP) have been found to be useful in chronic disease when the adult worms occupy the biliary tree.

## Histopathology

Rarely, a biopsy is performed, and a tissue diagnosis is made, most often when the diagnosis has not been considered. With ERCP removal of a worm, diagnosis can be confirmed as above. In ectopic fascioliasis, removal of the migrating metacercariae is diagnostic, as well. A liver biopsy typically reveals necrosis, acute and chronic inflammatory changes, debris, and occasionally fragments of migrating larvae.

## Treatment of Fascioliasis

Unlike other helminth infections, fascioliasis responds very poorly to treatment with albendazole or praziquantel. The reason(s) for this lack of treatment effectiveness are not understood. Currently, the drug of choice is triclabendazole,[49-52] which is not commercially available in the United States. It may be ordered from Victoria Pharmacy, Zurich, Switzerland.[53] Outside of the United States, it is available from the manufacturer, Novartis (Basel, Switzerland), for both treatment of individual cases and for mass administration as part of

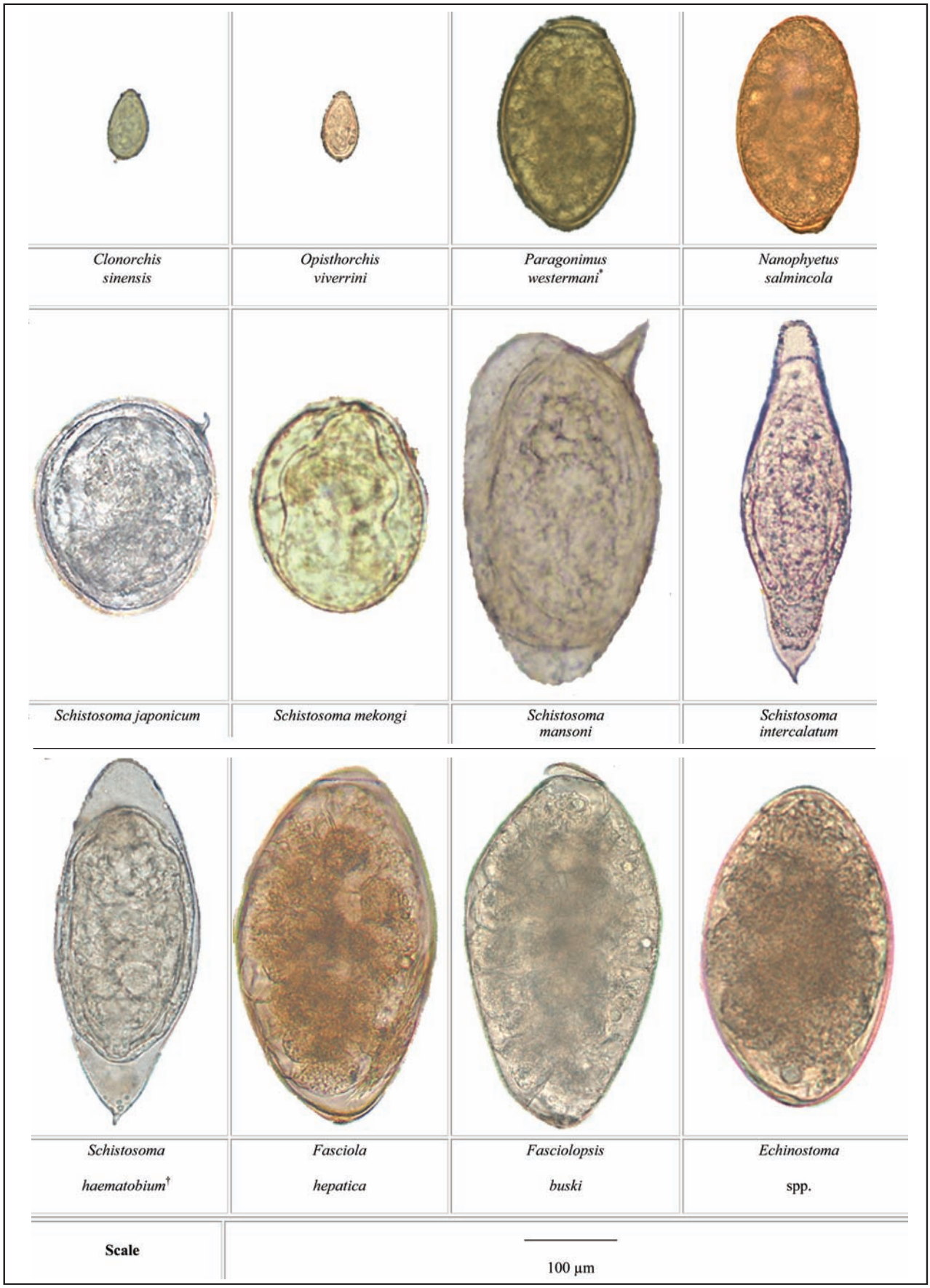


**Figure 4**_Trematode eggs found in stool specimens of humans. From the Centers for Disease Control and Prevention Parasites and Health Web site.[41]
*Usually found in respiratory specimens.
†Usually passed in urine.

**Figure 5**_Eggs of *F. hepatica*.
Eggs of *F. hepatica* are broadly ellipsoidal, operculated, and measure 130-150 µm long by 60-90 µm wide. The eggs are unembryonated when passed in feces. The eggs of *F. hepatica* can be difficult to distinguish from *Fasciolopsis* spp., although the abopercular end of the former often has a roughened or irregular area. Images from unstained wet mounts, taken at 400x magnification. From the Centers for Disease Control and Prevention Laboratory Identification of Parasites of Public Health Concern Web site.[20]

national control strategies.[7] The dose is 10 mg/kg by mouth once or twice, given with food. Alternatives include bithionol, available from the Centers for Disease Control and Prevention Drug Service, Atlanta, GA. The dose is 30-50 mg/kg by mouth on alternate days for 10-15 doses.[53] More recently, studies have suggested that nitazoxanide may be an effective alternative as well.[53,54] The dose is 500 mg by mouth twice daily for 7 days for adults. The dose is reduced to 100 mg twice daily for children aged 1-3 years and 200 mg twice daily for children aged 4-11 years. Those 12 years and above receive the adult dose.[53] There may be a role for treatment with artesunate in certain settings, based upon a pilot study.[55] As with all such diseases, prevention of infection by provision of clean water and safe food, control of sewage and waste, and healthy travel hygiene is preferable to treatment of established infection.

## Conclusion

Fascioliasis is a disease rarely encountered in the United States. As a result, correct diagnosis is often delayed because the infection is not considered. As the laboratory plays an important role in confirming the diagnosis, it is important to consider the disease in a patient with risk factors and proceed with appropriate testing and treatment.

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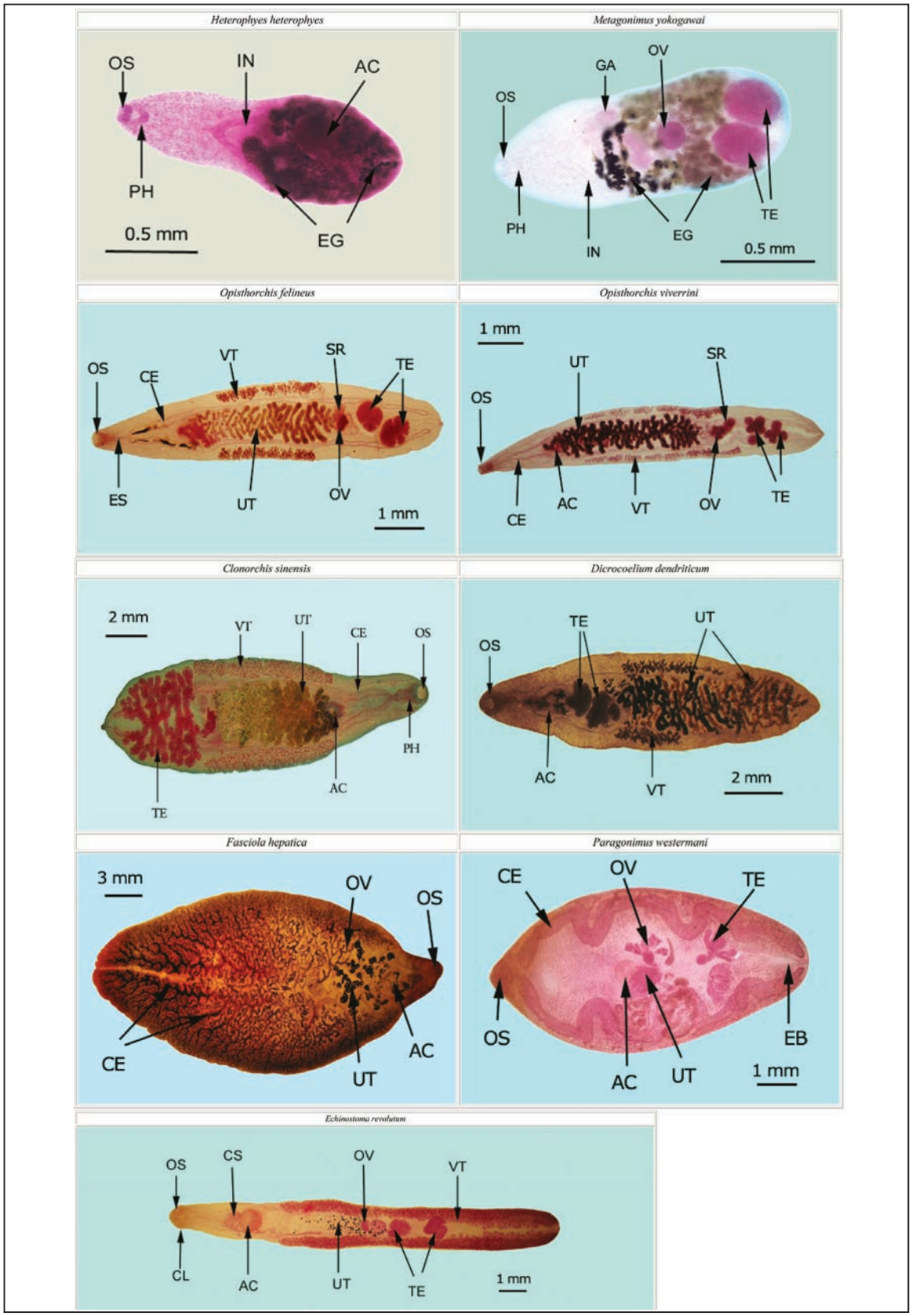


**Figure 6**

- AC = acetabulum (ventral sucker)
- CE = cecum
- CL = collar
- CS = cirrus sac
- IN = intestine
- OS = oral sucker
- OV = ovary
- PH = pharynx
- EB = excretory bladder
- EG = eggs (within uterus)
- ES = esophagus
- GA = genitoacetabulum
- SR = seminal receptacle
- TE = testes
- UT = uterus
- VT = vitellaria

From the Centers for Disease Control and Prevention Parasites and Health Web site.[41]


**Figure 7**_Adult *Fasciola hepatica.* Adults of *F. hepatica* are large and broadly flattened, measuring up to 30 mm long and 15 mm wide. The anterior end is cone shaped, unlike the rounded anterior end of *Fasciolopsis buski.* Adults reside in the bile ducts of the liver in the definitive host. Formalin-fixed, unstained (left) and carmine-stained adult *F. hepatica* (right). From the Centers for Disease Control and Prevention Laboratory Identification of Parasites of Public Health Concern Web site.[20]


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