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# Cestode Zoonoses of Aquatic Animals

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## Abstract

The cestode zoonoses of *Diphyllobothrium*, and *Spirometra* and Sparganosis are discussed from the point of view of their transmission and epidemiology.

Zoonoses, for the purpose of this discussion, are diseases and infections naturally transmitted between animals and man. In conformity with other American workers (Faust et al.;<sup>7</sup> Noble and Noble,<sup>20</sup>), I recognize both vertebrate and invertebrate animals as the source of human infection, although I acknowledge that some authorities employ a more limited definition of zoonosis. The Joint WHO/FAO expert committee<sup>1</sup> limit the definition to vertebrates and man, whereas the Soviets would restrict zoonoses to diseases transmissible among animals alone. There is a long list of epidemiological terminology and neologisms among which one must cautiously thread his way, even if he is a parasitologist, and I would refer the general worker to the summary article by Nelson<sup>18</sup> and a more recent discussion by Sprent.<sup>23</sup>

Within this broad concept of zoonoses, which includes the invertebrates, the list includes a few species of pseudophyllidean Diphylobothriidae that, except for a few incidental host records (*Diplogonoporus balaenopterae* Lönnberg, 1892 [= *D. grandis* Blanchard (1894)], *Ligula intestinalis* [Lin., 1758], and *Schistocephalus solidus* [Mueller, 1776]), are limited to the genera *Diphyllobothrium* and *Spirometra*.

Since both the source of zoonoses and the biological role played by man in the epidemiology are different, it is necessary that the species of each genus involved be considered separately. No attempt will be made to consider all the named species and reported cases since some are of doubtful validity and there is insufficient documentation of some records (e.g., *Diphyllobothrium cordatum* [Leuckart, 1863]).

Instead, I shall concentrate upon the well established cestode zoonoses of aquatic animals, and lay stress on the aspects of the problem which have relevance to us in the United States.

## Diphyllobothrium

Several species of *Diphyllobothrium* are involved in cestode zoonoses of aquatic animals. Table 1 lists three infrequently reported species. The commonest tapeworm of the north-temperate region is *Diphyllobothrium latum*, the broad fish tapeworm, which reaches sexual maturity in such fish-eating mammals as man, dogs, and bears. It is endemic to central and northern Europe, especially in the Baltic countries and the U.S.S.R., and in Siberia, Manchuria, and Japan. It has also spread to North America, where it occurs throughout much of the western Great Lakes region. Since the first case of human diphylobothriasis indigenous to the United States was reported in 1906, the infections have become so common that they are not report-worthy unless they occur far removed from the endemic area.

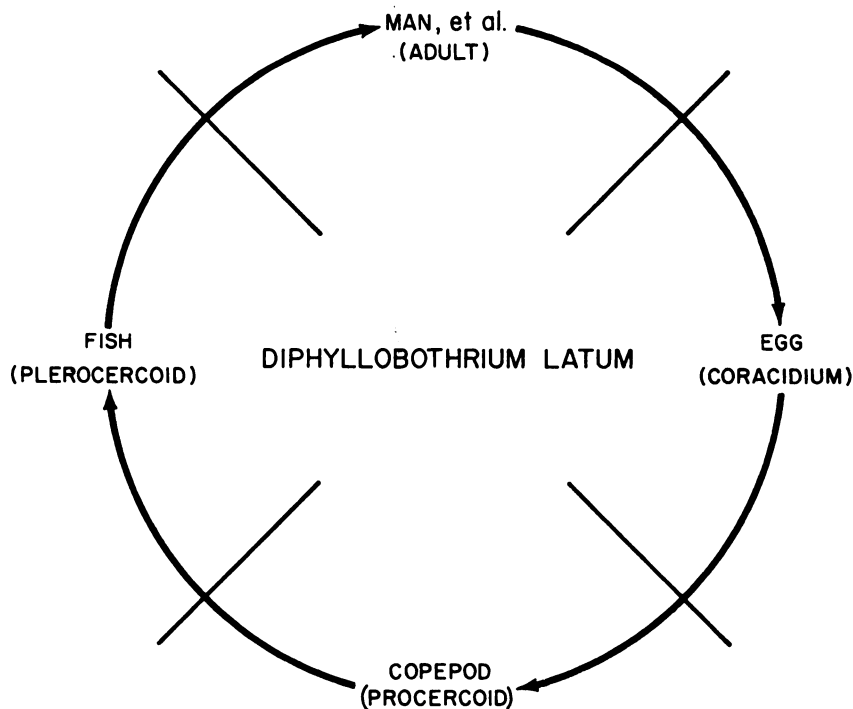
This cestode has a remarkable life cycle. When the eggs, undeveloped when passed with the host feces, reach water, the oncosphere or coracidium hatches. When the errant coracidium is eaten by a suitable copepod [species of *Diaptomus* serve best (Humes<sup>10</sup>)], it develops in the body cavity of the crustacean into a

TABLE 1. Zoonotic *diphyllbothrium*, other than *D. latum*

Species	Locality	Reference
<i>Diphyllbothrium dalliae</i> Rausch, 1956	Alaska	Hilliard <sup>6</sup> Rausch et al. <sup>21</sup>
<i>D. dendriticum</i> * (Nitsch, 1824)	Norway U.S.S.R. (Lake Baikal)	Vik <sup>28</sup> Chizhova et al. <sup>4</sup>
<i>D. pacificum</i> (Nybelin, 1931)	Peru	Baer et al. <sup>2</sup> Miranda et al. <sup>14</sup>

\* This apparently is a widely distributed species. *Diphyllbothrium minus* Kholodkovskii, 1916 and *D. strictum* (Talyzin, 1932) have recently been concluded by Chizhova (Med. Parazit. Parazit. Bolez. 26: 710-714, 1957) and Chizhova, Gofman-Kadoshnikov and Kravtsov (Ibid. 31: 213-223, 1962) as conspecific with *D. dendriticum*. Also *D. norvegicum* Vik, 1957 has been synonymized with *D. dendriticum* by Wikgren (Societas Scientiarum Fennica Commentationes Biologicae, 27: 1-26, 1964 and Bylund (Tiedoksianto-Information, 10: 3-17, 1969).

procercoid. The procercoid is liberated when the copepod is eaten by certain freshwater fish and it, in turn, bores through the intestine to reach the body wall or viscera of the piscine host, where it develops into a plerocercoid. In North America, pike (*Esox*) and walleyes (*Stizostedion*) are the most important hosts, but elsewhere other fish may be involved. The plerocercoids are liberated when the host fish is eaten, and proceed to develop into adults in the intestine of the final host (Fig. 1).

Figure 1. Life cycle of *Diphyllbothrium latum*.

Plerocercoids recovered from fish and implanted into frogs may remain viable for a month or longer, but no growth occurs.<sup>11,17</sup> Only fish are involved in this zoonosis. When plerocercoids enter the intestine of an unsuitable host, except in fish, where they may migrate through the intestinal wall and reestablish themselves, they are lost from the alimentary tract.

There is good reason to believe that man is the most important host in the cycle of transmission. Opinion is divided as to the importance of animal hosts in the epidemiology of *D. latum*. Essex and Magath<sup>6</sup> and Kuhlow<sup>13</sup> maintain that the dog plays no substantial role, while Vergeer<sup>24,26,28</sup> believes that dogs and bears are important in its dissemination. Experimental feeding infections have shown that the cat is a poor host.<sup>12,29</sup>

In addition to the general susceptibility of *Diaptomus* to the coracidia, two features of *D. latum* are especially noteworthy. First, the source of the zoonosis is limited to fish and second, man serves as the final host in the transmission cycle.

Personal preventive measures against diphyllobothriasis consist simply in abstinence from fish that are not thoroughly cooked. Salminen et al.<sup>25</sup> have shown that plerocercoids are quickly destroyed by a temperature of 56° C, but fish must be cooked for a length of time proportionate to its weight in order to insure penetration of heat to the center.

#### Spirometra and Sparganosis

While there is some confusion about the species of *Spirometra*, in the Orient it is usually regarded as *S. mansoni*, and in the United States, where it is distributed over a broad area of the Atlantic and Gulf States, it is normally recognized as *S. mansonioides*.

When the proceroid-infected copepods [species of *Cyclops* serve best (Mueller<sup>20</sup>)], are eaten by certain amphibians, reptiles, and mammals the plerocercoid stage develops in their tissues (Mueller,<sup>15</sup> Corkum.<sup>8</sup> Infection of the final host, cats and related mammals, comes from eating one of the second intermediate hosts containing the viable plerocercoids (Fig. 2). Experimental studies by Mueller<sup>17</sup> have shown that fish, as a result of their resistance to both proceroids and plerocercoids, are unsuitable intermediate hosts.

When the plerocercoids of *Spirometra*, for which the name *Sparganum* was used before their adult stage was known, gets into an unsuitable final host, (frogs, reptiles and some mammals) the larvae migrate through the intestinal wall and reestablish themselves in the subcutaneous tissues and muscles where they grow in size, until a host is reached in which maturity can be attained in the intestine. This is of much more than academic interest, because man is included among the unsuitable final hosts.

Thus, in addition to the general susceptibility of *Cyclops* to the coracidia, *Spirometra* differs from *Diphyllobothrium* in two important respects: first, the fish, which are obligatory hosts for the plerocercoids of *Diphyllobothrium*, are replaced by amphibians, reptiles, and some mammals (including man) in *Spirometra*; and second, man is the preferred final host of *D. latum*, while that of *Spirometra* is the cat.

Sparganosis as a zoonosis results from man: i) drinking water containing proceroid-infected *Cyclops* ii) eating amphibians, reptiles, and mammals containing the viable plerocercoids; iii) applying plerocercoid-infected flesh of frogs, snakes and possibly mammals to wounds or inflamed eye as a poultice.

While the use of meat-poultices is a common practice in the Orient, where the spargana of *S. mansoni* is involved, they can be ruled out for America. Until recently

it was presumed that zoonotic sparganosis in the United States came from drinking water containing the proceroid-infected copepods. But the finding of spargana in a hog in Florida (Becklund<sup>4</sup>) and the discovery of natural infections of *S. mansonioides* in two amphibian, eight reptilian, and three mammalian species in Louisiana by Corkum,<sup>5</sup> who showed experimentally that hogs are susceptible to spargana, suggest that human infection may occasionally result from consuming wild animals or pork containing the viable larvae.

It therefore behooves those living in an area exposed to *Spirometra* to give close attention to the epidemiology of the zoonosis and to practice effective control measures. Drinking water must be boiled or properly filtered; before being eaten, animal flesh should be cooked sufficiently to destroy the spargana, and the application of an animal poultice to an inflamed part of the body should be avoided.

Nelson et al.,<sup>19</sup> reporting on an area in equatorial Africa where man is the only known host of spargana, suggest that man may serve an essential role in the epidemiology of the zoonosis through his custom of leaving his dead and dying relations for hyenas to eat. In this area, hyenas are heavily infected with what is presumably the corresponding adult cestode. Elsewhere, the spargana are withdrawn from the transmission cycle when they get into man.

The first case of human sparganosis indigenous to the United States was reported in 1908. To date there are 44 cases; most of them have been observed in southern states, and the discovery of cases has taken an upward swing over the past decade. Recent reviews of the literature on the occurrence and distribution of human sparganosis can be found in Huang and Kirk<sup>9</sup> for the Orient, and McQuay et al.<sup>13</sup> for this country.

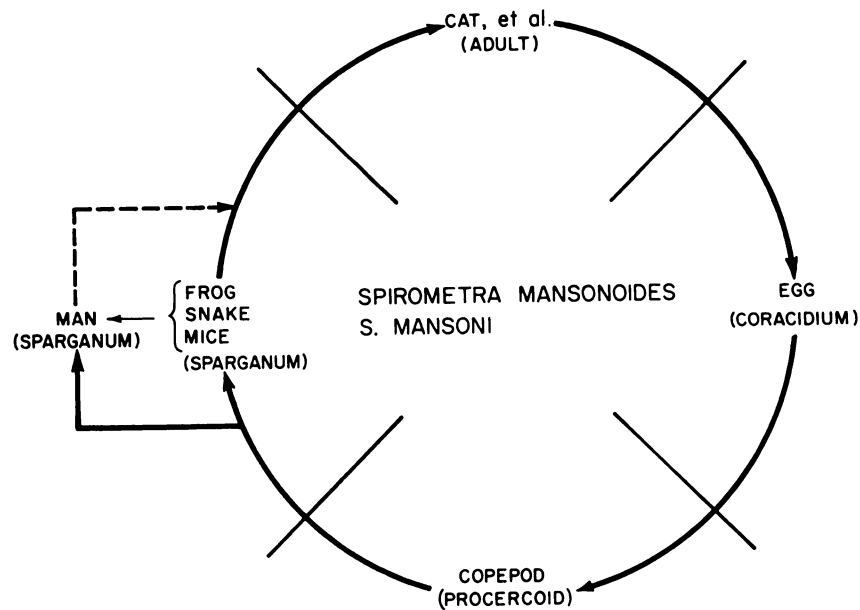


Figure 2. Life cycle of *Spirometra mansonioides* and *S. mansoni*.

It is hardly necessary to say that the number of known cases should not be taken as reflecting the true incidence of the zoonosis in the United States, because the number of cases is always greater than the actual records indicate. One should also bear in mind that the geographical distribution of a species of *Spirometra* and the pattern of a corresponding zoonosis may bear no relationship to one another. In order for a spargana zoonosis to occur, man's habits and behavior must be of a favorable nature, in the presence of the etiologic agent, for him to become involved in the zoonosis.

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#### Literature Cited

1. ANON. 1959. Joint WHO/FAO Expert Committee on Zoonoses. 2nd rept., Tech. Rep. Series Wld. Hlth. Org., No. 169, 83 pp.
2. BAER, J. G., H. MIRANDA, W. FERNANDEZ, and J. MEDINA T. 1967. Human diphyllbothriasis in Peru. *Zeit. Parasitenk.* 28: 277-289.
3. BECKLUND, W. W. 1962. Occurrence of a larval trematode (Diplostomidae) in a larval cestode (Diphyllbothriidae) from *Sus scrofa* in Florida. *J. Parasitol.* 48: 286.
4. CHIZHOVA, T. P., P. B. GOFMAN-KADOSHNIKOV, and E. G. KRAVTSOV. 1962. Plerocercoids in fish in Karelia and their epidemiological importance. *Med. Parazitol. Parazit. Bolez.* 31: 213-223.
5. CORKUM, K. C. 1966. Sparganosis in some vertebrates of Louisiana and observations on a human infection. *J. Parasit.* 52: 444-448.
6. ESSEX, H. E., and T. B. MAGATH. 1931. Comparison of the viability of ova of the broad fish tapeworm, *Diphyllbothrium latum*, from man and dogs: its bearing on the spread of infestation with this parasite. *Amer. J. Hyg.* 14: 698-704.
7. FAUST, E. C., P. C. BEAVER, and R. C. JUNG. 1968. *Animal Agents and Vectors of Human Disease*, 3rd ed., Philadelphia, Lea & Febiger, 461 pp.
8. HILLIARD, D. K. 1960. Studies on the helminth fauna of Alaska. XXXVIII. The taxonomic significance of the eggs and coracidia of some diphyllbothriid cestodes. *J. Parasitol.* 46: 703-716.
9. HUANG, C. T., and R. KIRK. 1962. Human sparganosis in Hong Kong. *J. Trop. Med.* 65: 133-138.
10. HUMES, A. G. 1950. Experimental copepod hosts of the broad tapeworm of man, *Dibothriocephalus latus* (L.). *J. Parasit.* 36: 541-547.
11. KUHLOW, F. 1953. Beiträge zur Entwicklung und Systematik heimischer *Diphyllbothrium*-Arten. *Zeit. Tropenmed. Parasit.* 4: 203-234.
12. ———. 1955. Untersuchungen über die Entwicklung des Breiten Bandwurmes (*Diphyllbothrium latum*). *Zeit. Tropenmed. Parasit.* 6: 213-225.
13. McQUAY, R. M., S. VEIGA, and W. A. FRUMOVITZ. 1966. Sparganosis in a Chicago resident originally from Arkansas. *Amer. J. Clin. Path.* 46: 645-648.
14. MIRANDA, H., W. FERNANDEZ, and R. BOCANEGRA. 1967. Diphyllbothriasis. Estado actual en el Peru. Descripción de nuevos casos. *Arch. Peru. Patol. Clin. (Lima)*, 21: 53-69.
15. MUELLER, J. F. 1937. The hosts of *Diphyllbothrium mansonoides* (Cestoda: Diphyllbothriidae). *Proc. Helm. Soc. Washington* 4: 68-69.

16. ————. 1938. The life history of *Diphyllobothrium mansonoides* Mueller, 1935, and some considerations with regard to sparganosis in the United States. *Amer. J. Trop. Med.* 18: 41-66.
  17. ————. 1960. The immunologic basis of host specificity in the sparganum larva of *Spirometra mansonoides*. In *Libro Homenaje al Doctor Eduardo Caballero y Caballero, Mexico, D. F.*, pp. 435-442.
  18. NELSON, G. S. 1960. Schistosome infections as zoonoses in Africa. *Trans. Royal Soc. Trop. Med. Hyg.* 54: 301-324.
  19. ————, F. R. N. PESTER, and R. RICKMAN. 1965. The significance of wild animals in the transmission of cestodes of medical importance in Kenya. *Trans. Royal Soc. Trop. Med. Hyg.* 59: 507-524.
  20. NOBLE, R. R., and G. A. NOBLE. 1964. *Parasitology. The Biology of Animal Parasites*, 2nd ed., Philadelphia, Lea & Febiger, 724 pp.
  21. RAUSCH, R. L., E. M. SCOTT, and V. R. RAUSCH. 1967. Helminths in Eskimos in western Alaska, with particular reference to *Diphyllobothrium* infection and anaemia. *Trans. Royal Soc. Trop. Med. Hyg.* 61: 351-357.
  22. SALMINEN, K., H.-L. KUOSMA, and L. REINIUS. 1966. The effect of customary Finnish heat preparation methods on the infestiveness of *Diphyllobothrium latum* from fish to man. *Act Vet. Scand.* 7: 101-124.
  23. SPRENT, J. F. A. 1969. Helminth "Zoonoses": An analysis. *Helm. Abs.* 38: 333-351.
  24. VERGEER, T. 1928. Dissemination of the broad tapeworm by wild Carnivora. *Canad. Med. Assn. J.* 19: 692-694.
  25. ————. 1929. The dog a reservoir of the broad tapeworm. *J. Amer. Med. Assn.* 92: 607-608.
  26. ————. 1930. Causes underlying increased incidence of broad tapeworm in man in North America. *J. Amer. Med. Assn.* 95: 1579-1581.
  27. ————. 1937. No asexual reproduction in *Diphyllobothrium*. In *Papers in Helminthology, Moscow*, pp. 755-757.
  28. VIK, R. 1957. Studies on the helminth fauna of Norway. I. Taxonomy and ecology of *Diphyllobothrium norvegicum* n. sp. and the plerocercoid of *Diphyllobothrium latum* (L.). *Nytt Mag. Zool.* 5: 26-93.
  29. WARD, H. B. 1929. Studies on the broad fish tapeworm in Minnesota. *J. Amer. Med. Assn.* 29: 389-390.
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