Parasitic Animals

Marketta Vanhorn

First Edition, 2012

ISBN 978-81-323-4203-8

Published by:
White Word Publications
4735/22 Prakashdeep Bldg,
Ansari Road, Darya Ganj,
Delhi - 110002
Email: info@wtbooks.com

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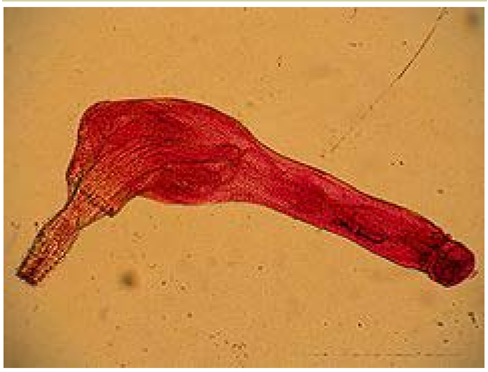
Acanthocephala

Acanthocephala	
	
Corynosoma wegeneri	
Scientific classification [e]	
Kingdom:	Animalia
(unranked):	Protostomia
(unranked):	Spiralia
(unranked):	Platyzoa
Phylum:	**Acanthocephala** Koelreuther, 1771

Acanthocephala is a phylum of parasitic worms known as **acanthocephales**, **thorny-headed worms**, or **spiny-headed worms**, characterized by the presence of an evertable proboscis, armed with spines, which it uses to pierce and hold the gut wall of its host. Acanthocephalans typically have complex life cycles, involving a number of hosts, including invertebrates, fishes, amphibians, birds, and mammals. About 1150 species have been described.

The Acanthocephala were thought to be a discrete phylum. Recent genome analysis has shown that they are descended from, and should be considered as, highly modified rotifers. This is an example of molecular phylogenetics.

Morphological characteristics

There are several morphological characteristics that distinguish acanthocephalans from other phyla of parasitic worms.

Digestion

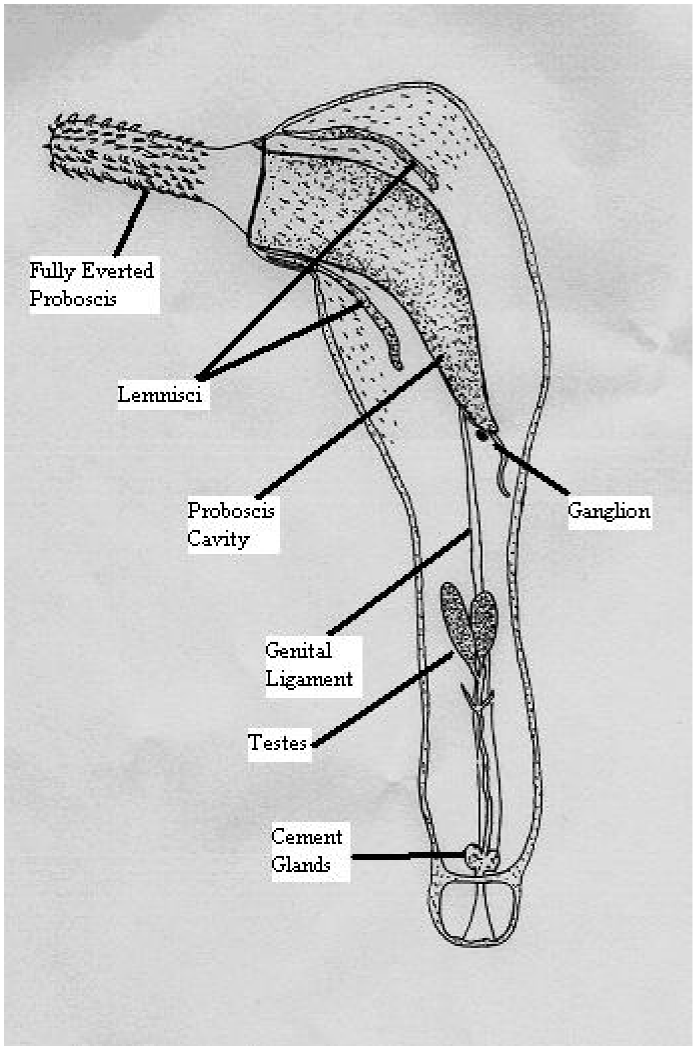
Acanthocephalans lack a mouth or alimentary canal. This is a feature they share with the cestoda (tapeworms), although the two groups are not closely related. Adult stages live in the intestines of their host and uptake nutrients which have been digested by the host, directly, through their body surface.

Proboscis

The most notable feature of the acanthocephala is the presence of an anterior, protrudible proboscis that is usually covered with spiny hooks (hence the common name: thorny headed worm). The proboscis bears rings of recurved hooks arranged in horizontal rows, and it is by means of these hooks that the animal attaches itself to the tissues of its host. The hooks may be of two or three shapes, usually, longer, more slender hooks are arranged along the length of the proboscis, with several rows of more sturdy, shorter nasal hooks around the base of the proboscis. The proboscis is used to pierce the gut wall of the final host, and hold the parasite fast while it completes its life cycle.

Like the body, the proboscis is hollow, and its cavity is separated from the body cavity by a *septum* or *proboscis sheath*. Traversing the cavity of the proboscis are muscle-strands inserted into the tip of the proboscis at one end and into the septum at the other. Their contraction causes the proboscis to be invaginated into its cavity. The whole proboscis apparatus can also be, at least partially, withdrawn into the body cavity, and this is effected by two retractor muscles which run from the posterior aspect of the septum to the body wall.

Some of the acanthocephalans (perforating acanthocephalans) can insert their proboscis in the intestine of the host and open the way to the abdominal cavity.



Some key features of acanthocephalan morphology

Phylogenetic relationships

Acanthocephalans are highly adapted to a parasitic mode of life, and have lost many organs and structures through evolutionary processes. This makes determining relationships with other higher taxa through morphological comparison problematic. Phylogenetic analysis of the 18S ribosomal gene has revealed that the Acanthocephala are most closely related to the rotifers, or may even belong in that phylum. The two are included among the Platyzoa.

Size

The size of the animals varies greatly, from forms a few millimetres in length to *Gigantorhynchus gigas*, which measures from 10 to 65 centimetres (3.9 to 26 in).

Skin

The body surface of the acanthocephala is peculiar. Externally, the skin has a thin cuticle covering the epidermis, which consists of a syncytium with no cell walls. The syncytium is traversed by a series of branching tubules containing fluid and is controlled by a few wandering, amoeboid nuclei. Inside the syncytium is an irregular layer of circular muscle fibres, and within this again some rather scattered longitudinal fibres; there is no endothelium. In their micro-structure the muscular fibres resemble those of nematodes.

Except for the absence of the longitudinal fibres the skin of the proboscis resembles that of the body, but the fluid-containing tubules of the proboscis are shut off from those of the body. The canals of the proboscis open into a circular vessel which runs round its base. From the circular canal two sac-like projections called the *lemnisci* run into the cavity of the body, alongside the proboscis cavity. Each consists of a prolongation of the syncytial material of the proboscis skin, penetrated by canals and sheathed with a muscular coat. They seem to act as reservoirs into which the fluid which is used to keep the proboscis "erect" can withdraw when it is retracted, and from which the fluid can be driven out when it is wished to expand the proboscis.

Nervous system

The central ganglion of the nervous system lies behind the proboscis sheath or septum. It innervates the proboscis and projects two stout trunks posteriorly which supply the body. Each of these trunks is surrounded by muscles, and this nerve-muscle complex is called a *retinaculum*. In the male at least there is also a genital ganglion. Some scattered papillae may possibly be sense-organs.

"Brain-jacking"

Thorny-headed worms begin their life cycle inside invertebrates that reside in marine or freshwater systems. *Gammarus lacustris*, a small crustacean that feeds near ponds and rivers, is one invertebrate that the thorny-headed worm may occupy. This crustacean is preyed on by ducks and hides by avoiding light and staying away from the surface. However, when infected by a thorny-headed worm it becomes attracted toward light and swims to the surface. *Gammarus lacustris* will even go so far as to find a rock or a plant on the surface, clamp its mouth down, and latch on, making it easy prey for the duck. The duck is the definitive host for the acanthocephalan parasite. In order to be transmitted to the duck, the parasite's intermediate host (the gammarid) must be eaten by the duck. This modification of gammarid behavior by the acanthocephalan is thought to increase the rate of transmission of the parasite to its next host by increasing the susceptibility of the gammarid to predation.

It is thought that when *Gammarus lacustris* is infected with a thorny-headed worm, the parasite causes serotonin to be massively expressed. Serotonin is a neurotransmitter involved in emotions and mood. Researchers have found that during mating *Gammarus lacustris* expresses high levels of serotonin. Also during mating, the male *Gammarus* *lacustris* clamps down on the female and holds on for days. Researchers have additionally found that blocking serotonin releases clamping. Another experiment found that serotonin also reduces the photophobic behavior in *Gammarus lacustris*. Thus, it is thought that the thorny-headed worm physiologically changes the behavior of the *Gammarus lacustris* in order to enter its final host, the bird.

Sex

The Acanthocephala are dioecious. There is a structure called the *genital ligament* which runs from the posterior end of the proboscis sheath to the posterior end of the body. In the male, two testes lie on either side of this. Each opens in a vas deferens which bears three diverticula or *vesiculae seminales*. The male also possesses three pairs of cement glands, found behind the testes, which pour their secretions through a duct into the vasa deferentia. These unite and end in a penis which opens posteriorly.

In the female, the ovaries are found, like the testes, as rounded bodies along the ligament. From these masses of ova dehisce into the body cavity and float in its fluid. Here the eggs are fertilized and segment so that the young embryos are formed within their mother's body. The embryos escape into the uterus through the *uterine bell*, a funnel like opening continuous with the uterus. At the junction of the bell and the uterus there is a second small opening situated dorsally. The bell "swallows" the matured embryos and passes them on into the uterus, and from there, out of the body via the oviduct. Should the bell swallow any of the ova, or even one of the younger embryos, these are passed back into the body cavity through the second, dorsal, opening.

The embryo passes from the body of the female into the alimentary canal of the host and leaves this with the feces.

Other features

A curious feature shared by both larva and adult is the large size of many of the cells, e.g. the nerve cells and cells forming the uterine bell. Polyploidy is common, with up to 343n having been recorded in some species. The acanthocephalans lack an excretory system, although some species have been shown to possess flame cells (protonephridia).

History

The earliest recognisable description of Acanthocephala - a worm with a proboscis armed with hooks - was made by Italian author Francesco Redi (1684). In 1771, I. T. Koelreuther proposed the name Acanthocephala. Müller independently called them *Echinorhynchus* in 1776. Karl Rudolphi in 1809 formally named them Acanthocephala.

Currently the phylum is divided into four classes – Palaeacanthocephala, Archiacanthocephala, Polyacanthocephala and Eoacanthocephala.

Life cycles

Adult *Pomphorhynchus* in a bluefish

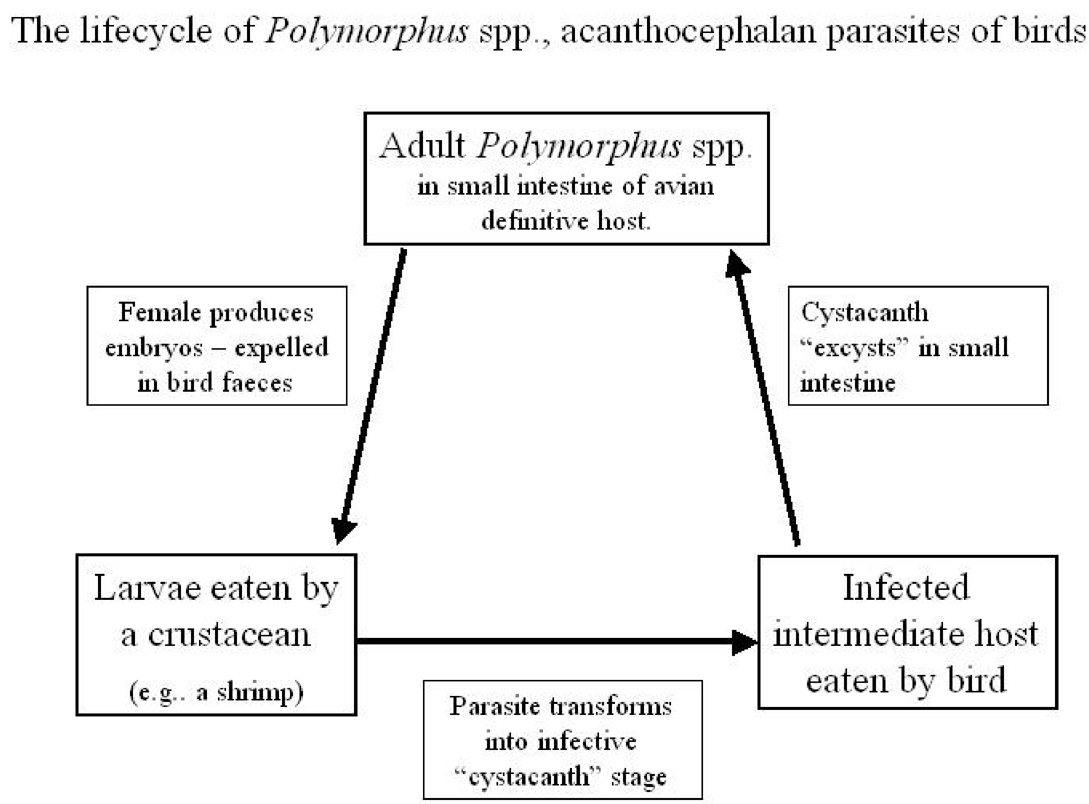
General patterns

Acanthocephalans have complex life cycles, involving a number of hosts, for both developmental and resting stages. Complete life cycles have been worked out for only 25 species.

Having been expelled by the female, the acanthocephalan embryo is released along with the feces of the host. For development to occur, the embryo needs to be ingested by an invertebrate, almost always a crustacean (there is one known life cycle which uses a mollusc as a first intermediate host). Inside the intermediate host, the acanthocephalan penetrates the gut wall, moves into the body cavity, encysts, and begins transformation into the infective *cystacanth* stage. This form has all the organs of the adult save the reproductive ones.

The parasite is released when the first intermediate host is ingested. This can be by a suitable final host, in which case the cystacanth develops into a mature adult, or by a paratenic host, in which the parasite again forms a cyst. When consumed by a suitable final host, the cycstacant *excysts*, everts its proboscis and pierces the gut wall. It then feeds, grows and develops its sexual organs. Adult worms then mate. The male uses the excretions of its *cement glands* to plug the vagina of the female, preventing subsequent matings from occurring. Embryos develop inside the female, and the life cycle repeats.

An example - *Polymorphus* spp.



A diagram of the life cycle of *Polymorphus* spp.

Polymorphus spp. are parasites of seabirds, particularly the Eider Duck (*Somateria mollissima*). Heavy infections of up to 750 parasites per bird are common, causing ulceration to the gut, disease and seasonal mortality. Recent research has suggested that there is no evidence of pathogenicity of *Polymorphus* spp. to intermediate crab hosts. The cystacanth stage is long lived and probably remains infective throughout the life of the crab.

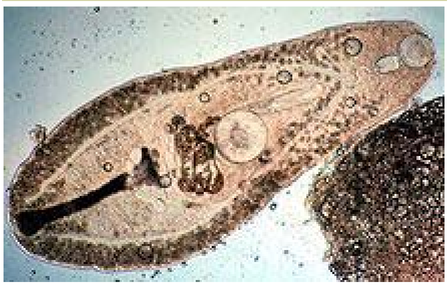
The life cycle of *Polymorphus* spp. normally occurs between sea ducks (e.g. eiders and scoters) and small crabs. Infections found in commercial-sized lobsters in Canada were probably acquired from crabs that form an important dietary item of lobsters. Cystacanths occurring in lobsters can cause economic loss to fishermen. There are no known methods of prevention or control.

Use in medicine

Acanthocephala have been used as a medical treatment for various diseases, particularly those involving an over active immune response. As humans have evolved with parasitic worms, proponents argue that they are needed for a healthy immune system. Scientists are looking to see if there is a connection between the prevention and control of parasitic worms and the increase in allergies such as hay-fever in developed countries. Acanthocephala and other parasitic worms may be able to damp down the immune system of their host, making it easier for them to live in the intestine without coming under attack. This may be one mechanism for their proposed medicinal effect.

Chapter 2

Digenea

Digenea	
	
Helicometra sp. (Plagiorchiida: Opecoelidae) from the intestine of a Flame Cardinal fish	
Scientific classification [e]	
Kingdom:	Animalia
Phylum:	Platyhelminthes
Class:	Trematoda
Subclass:	**Digenea** Carus, 1863
Orders (Families)	
Azygiida Echinostomida Opisthorchiida Plagiorchiida Strigeidida	

Digenea (Gr. *Dis* - double, *Genos* - race) is a subclass within the Platyhelminthes consisting of parasitic flatworms with a syncytial tegument and, usually, two suckers, one ventral and one oral. Adults are particularly common in the digestive tract, but occur throughout the organ systems of all classes of vertebrates. Once thought to be related to the Monogenea, it is now recognised that they are closest to the Aspidogastrea and that the Monogenea are more closely allied with the Cestoda. Around 6,000 species have been described to date.

Morphology

Key features

Characteristic features of the digenea include a syncitial tegument; that is, a tegument where the junctions between cells are broken down and a single continuous cytoplasm surrounds the entire animal. A similar tegument is found in other members of the Neodermata; a group of platyhelminths comprising the Digenea, Aspidogastrea, Monogenea and Cestoda. Digeneans possess a vermiform, unsegmented body-plan and have a solid parenchyma with no body cavity, as in all platyhelminths.

There are typically two suckers, an anterior oral sucker surrounding the mouth, and a ventral sucker sometimes termed the *acetabulum*, on the ventral surface. The oral sucker surrounds the mouth, while the ventral sucker is a blind muscular organ with no connection to any internal structure.

Monostome is a term used to describe worms with one sucker (oral). Flukes with an oral sucker and an acetabulum at the posterior end of the body are called *Amphistomes*. *Distomes* are flukes with an oral sucker and a ventral sucker, but the ventral sucker if somewhere other than posterior. These terms are common in older literature, when they were thought to reflect systematic relationships within the groups. They have fallen out of use in modern digenean taxonomy.

Reproductive system

The vast majority of digeneans is hermaphroditic. This is likely to be an adaptation to low abundance within hosts, allowing the life cycle to continue when only one individual successfully infects the final host. Fertilisation is internal, with sperm being transferred via the cirrus to the Laurer's Canal or genital aperture. A key group of digeneans which are dioecious are the schistosomes. Asexual reproduction in the first larval stage is ubiquitous.

While the sexual formation of the digenean eggs and asexual reproduction in the first larval stage (miracidium) is widely reported, the developmental biology of the asexual stages remains a problem. Electron microscopic studies have shown that the light microscopically visible germ balls consist of mitotically dividing cells which give rise to embryos and to a line of new germ cells that become included in these embryonic stages. Since the absence of meiotic processes is not proven, the exact definition remains doubtful.

Male organs

Protandry is the general rule among the Digenea. Usually two testes are present, but some flukes can have more than 100. Also present are vasa efferentia, a vas deferens, seminal vesicle, ejaculatory duct and a cirrus (analogous to a penis) usually (but not always) enclosed in a cirrus sac. The cirrus may or may not be covered in proteinaceous spines. The exact conformation of these organs within the male terminal genitalia is taxonomically important at the familial and generic levels.

Female organs

Usually there is a single ovary with an oviduct, a seminal receptacle, a pair of vitelline glands (involved in yolk and egg-shell production) with ducts, the ootype (a chamber where eggs are formed), a complex collection of glands cells called *Mehlis' gland*, which is believed to lubricate the uterus for egg passage.

In addition, some digeneans possess a canal called Laurer's Canal, which leads from the oviduct to the dorsal surface of the body. The function of this canal is debated, but it may be used for insemination in some species or for disposal of waste products from reproduction in other species. Most trematodes possess an ovicapt, an enlarged portion of the oviduct where it joins the ovary. It probably controls the release of ova and spaces out their descent down the uterus.

The uterus typically opens into a common genital atrium that also received the distal male copulatory organ (cirrus) before immediately opening onto the outer surface of the worm. The distal part of the uterus may be expanded into a metraterm, set off from the proximal uterus by a muscular sphincter, or it may be lined with spines, as in the Monorchiidae and some other families.

Digestive system

As adults, most digeneans possess a terminal or subterminal mouth, a muscular pharynx that provides the force for ingesting food, and a forked, blind digestive system consisting of two tubular sacs called caeca (sing. caecum). In some species the two gut caeca join posteriorly to make a ring-shaped gut or cyclocoel. In others the caeca may fuse with the body wall posteriorly to make one or more anuses, or with the excretory vesicle to form a uroproct. Digeneans are also capable of direct nutrient uptake through the tegument by pinocytosis and phagocytosis by the syncitium. Most adult digeneans occur in the vertebrate alimentary canal or its associated organs, where they most often graze on contents of the lumen (e.g., food ingested by the host, bile, mucus), but they may also feed across the mucosal wall (e.g., submucosa, host blood). The blood flukes, such as schistosomes, spirorchiids and sanguinicolids, feed exclusively on blood. Asexual stages in mollusc intermediate hosts feed mostly by direct absorption, although the redia stage found in some groups does have a mouth, pharynx and simple gut and may actively consume host tissue or even other parasites. Encysted metacercarial stages and free-living cercarial stages do not feed.

Nervous system

Paired ganglia at the anterior end of the body serve as the brain. From this nerves extend anteriorly and posteriorly. Sensory receptors are, for the most part, lacking among the adults, although they do have tangoreceptor cells. Larval stages have many kinds of sensory receptors, including light receptors and chemoreceptors. Chemoreception plays an important role in the free-living miracidial larva recognising and locating its host.

Life cycles

There is a bewildering array of variation on the complex digenean life cycle, and plasticity in this trait is probably a key to the group's success. In general, the life cycles may have two, three, or four obligate (necessary) hosts, sometimes with transport or paratenic hosts in between. The three-host life cycle is probably the most common. In almost all species, the first host in the life cycle is a mollusc. This has led to the inference that the ancestral digenean was a mollusc parasite and that vertebrate hosts were added subsequently.

The alternation of sexual and asexual generations is an important feature of digeneans. This phenomenon involves the presence of several discrete generations in one life-cycle.

A typical digenean trematode life cycle is as follows. Eggs leave the vertebrate host in faeces and use various strategies to infect the first intermediate host, in which sexual reproduction does not occur. Digeneans may infect the first intermediate host (usually a snail) by either passive or active means. The eggs of some digeneans, for example, are (passively) eaten by snails (or, rarely, by an annelid worm), in which they proceed to hatch. Alternatively, eggs may hatch in water to release an actively swimming, ciliated larva, the miracidium, which must locate and penetrate the body wall of the snail host.

After post-ingestion hatching or penetration of the snail, the miracidium metamorphoses into a simple, sac-like *mother sporocyst*. The mother sporocyst undergoes a round of internal asexual reproduction, giving rise to either *rediae* (sing. redia) or *daughter sporocysts*. The second generation is thus the daughter parthenita sequence. These in turn undergo further asexual reproduction, ultimately yielding large numbers of the second free-living stage, the *cercaria* (pl. cercariae).

Free-swimming cercariae leave the snail host and move through the aquatic or marine environment, often using a whip-like tail, though a tremendous diversity of tail morphology is seen. Cercariae are infective to the second host in the life cycle, and infection may occur passively (e.g., a fish consumes a cercaria) or actively (the cercaria penetrates the fish).

The life cycles of some digeneans include only two hosts, the second being a vertebrate. In these groups, sexual maturity occurs after the cercaria penetrates the second host, which is in this case also the definitive host. Two host life-cycles can be primary (there never was a third host) as in the Bivesiculidae, or secondary (there was at one time in evolutionary history a third host but it has been lost).

In three-host life cycles, cercariae develop in the second intermediate host into a resting stage, the *metacercaria*, which is usually encysted in a cyst of host and parasite origin, or encapsulated in a layer of tissue derived from the host only. This stage is infective to the definitive host. Transmission occurs when the definitive host preys upon an infected second intermediate host. Metacercariae excyst in the definitive host's gut in response to a variety of physical and chemical signals, such as gut pH levels, digestive enzymes, temperature, etc. Once excysted, adult digeneans migrate to more or less specific sites in the definitive host and the life cycle repeats.

Evolution

The evolutionary origins of the Digenea have been debated for some time, but there appears general agreement that the proto-digenean was a parasite of a mollusc, possibly of the mantle cavity. Evidence for this comes from the ubiquity of molluscs as first intermediate hosts for digeneans, and the fact that most aspidogastreans (the sister group to the Digenea) also have mollusc associations. It is thought that the early trematodes (the collective name for digeneans and aspidogastreans) likely evolved from rhabdocoel turbellarians that colonised the open mantle cavity of early molluscs.

It is likely that more complex life cycles evolved through a process of terminal addition, whereby digeneans survived predation of their mollusc host, probably by a fish. Other hosts were added by until the modern bewildering diversity of life cycle patterns developed.

Important families

The Digenea includes at least 50 families. Below is a list of the more commonly encountered ones.

Acanthocolpidae	Derogenidae	Haplosplanchnidae	Paramphistomidae
Accacoeliidae	Dicrocoeliidae	Hemiuridae	Paragonimidae
Allocreadiidae	Didymozoidae	Heronomidae	Philophthalmidae
Angiodictyidae	Diplostomidae	Heterophyidae	Plagiorchiidae
Apocreadiidae	Echinostomatidae	Lecithasteridae	Sanguinicolidae
Atractotrematidae	Enenteridae	Lepocreadiidae	Schistosomatidae
Azygiidae	Fasciolidae	Microphallidae	Sclerodistomidae
Bivesiculidae	Faustulidae	Monorchiidae	Strigeidae

Bucephalidae	Fellodstomidae	Nasitrematidae	Syncoeliidae
Campulidae	Gorgoderidae	Notocotylidae	Tandanicolidae
Cryptogonimidae	Gyliauchenidae	Opecoelidae	Transversotrematidae
Cyclocoelidae	Haploporidae	Opisthorchiidae	Zoogonidae

There are some digenean families that are either basal to or *incertae sedis* among the orders listed above:

- Acanthocollaritrematidae
- Echinoporidae
- Gekkonotrematidae
- Gyliauchenidae
- Jubilariidae
- Meristocotylidae
- Mesotretidae

Human digenean infections

Only about 12 of the 6,000 known species are infectious to humans, but some of these species are important diseases afflicting over 200 million people. The species that infect humans can be divided into groups, the schistosomes and the non-schistosomes.

Schistosomes

The Schistosomes occur in the circulatory system of the definitive host. Humans become infected after free-swimming cercaria liberated from infected snails penetrate the skin. These dioecious worms are long and thin, ranging in size from 10 to 30 mm in length to 0.2 to 1.0 mm in diameter. Adult males are shorter and thicker than females, and have a long groove along one side of the body in which the female is clasped. Females reach sexual maturity after they have been united with a male. After mating the two remain locked together for the rest of their lives. They can live for several years and produce many thousands of eggs.

The four species of schistosomes that infect humans are members of the genus *Schistosoma*.

Human Schistosomes		
Scientific Name	**First Intermediate Host**	**Endemic Area**
Schistosoma mansoni	*Biomphalaria* spp.	Africa, South America, Caribbean, Middle East

Schistosoma haematobium	*Bulinus* spp.	Africa, Middle East
Schistosoma japonicum	*Oncomelania* spp.	China, East Asia, Philippines
Schistosoma intercalatum	*Bulinus* spp.	Africa

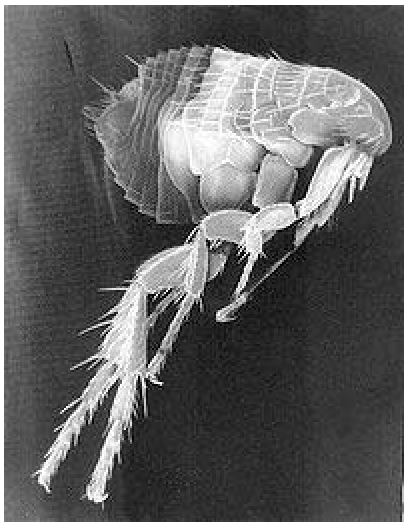
Non-schistosomes

There seven major species of non-schistosomes that infect humans are listed below. People become infected after ingesting metacercarial cysts on plants or in undercooked animal flesh. Most species inhabit the human gastrontestinal tract, where they shed eggs along with host feces. *Paragonimus westermani*, which colonizes the lungs, can also pass its eggs in saliva. These flukes generally cause mild pathology in humans, but more serious effects may also occur.

Human non-Schistosomes			
Scientific Name	**First Intermediate Host**	**Mode of Human Infection**	**Endemic Area**
Fasciolopsis buski	*Segmentina* sp.	Plants	Asia, India
Heterophyes heterophyes	*Pirinella*	Mullet, Tilapia	Asia, Eastern Europe, Egypt, Middle East
Metagonimus yokogawaii	*Semisulcospira* sp.	Carp, Trout	Siberia
Gastrodiscoides hominis	*Helicorbis* sp.	Plants	India, Vietnam, Philippines
Clonorchis sinensis	*Bulinus* sp.	Fish	East Asia, North America
Fasciola hepatica	*Galba truncatula*	Plants	Central America, North America, South America
Paragonimus westermani	*Oncomelania* sp.	Crabs, crayfish	Asia

Chapter 3

Flea

Flea Temporal range: Cretaceous–Recent	
	
Scanning electron microscope (SEM) depiction of a flea	
Scientific classification	
Kingdom:	Animalia
Phylum:	Arthropoda
Class:	Insecta
Subclass:	Pterygota
Infraclass:	Neoptera
Superorder:	Endopterygota
Order:	**Siphonaptera** Latreille, 1825
Infraorders	
Ceratophyllomorpha Hystrichopsyllomorpha Pulicomorpha Pygiopsyllomorpha	

Synonyms

Aphaniptera

Flea is the common name for insects of the order **Siphonaptera** which are wingless insects with mouthparts adapted for piercing skin and sucking blood. Fleas are external parasites, living by hematophagy off the blood of mammals (including bats and humans) and birds.

Some flea species include:

- Cat flea (*Ctenocephalides felis*)
- Dog flea (*Ctenocephalides canis*)
- Human flea (*Pulex irritans*)
- Moorhen flea (*Dasypsyllus gallinulae*)
- Northern rat flea (*Nosopsyllus fasciatus*)
- Oriental rat flea (*Xenopsylla cheopis*)

Over 2,000 species have been described worldwide.

Morphology and behavior

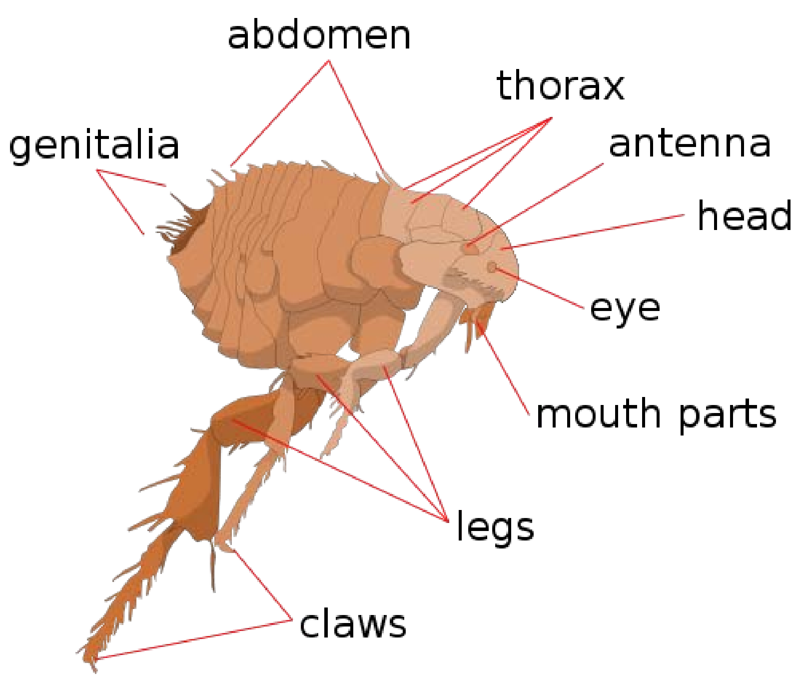
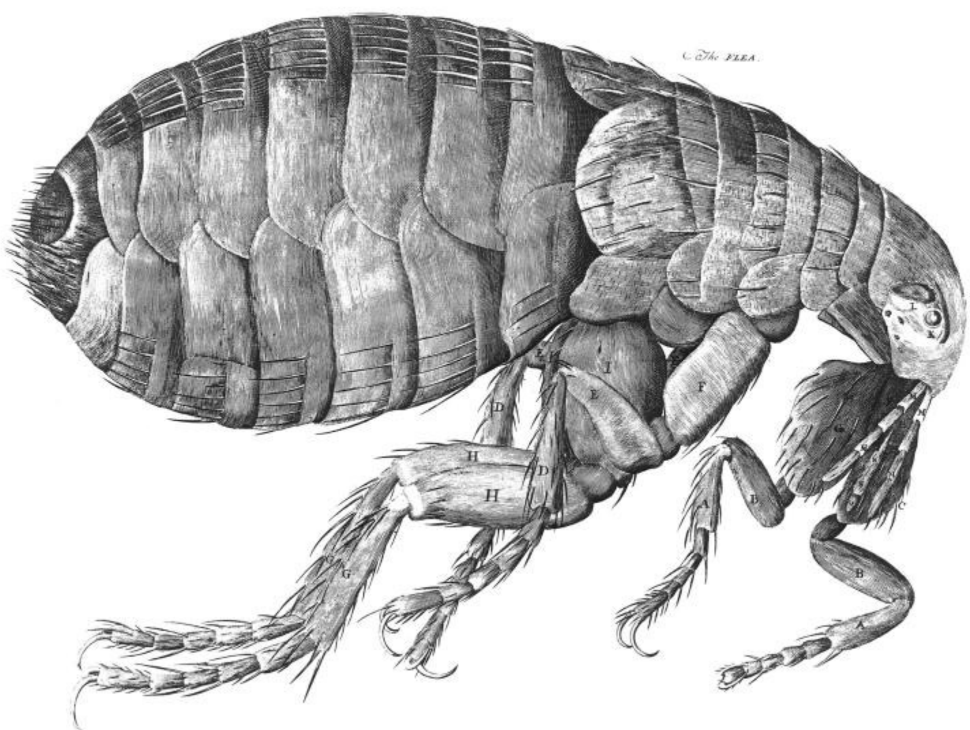


Diagram of a Flea

Fleas are small (1/16 to 1/8-inch (1.5 to 3.3 mm) long), agile, usually dark colored (for example, the reddish-brown of the cat flea), wingless insects with tube-like mouth-parts adapted to feeding on the blood of their hosts. Their legs are long, the hind pair well adapted for jumping: a flea can jump vertically up to 7 inches (18 cm) and horizontally up to 13 inches (33 cm). This is around 200 times their own body length, making the flea one of the best jumpers of all known animals (relative to body size), second only to the froghopper. According to an article in Science News , "researchers with the University of Cambridge in England have shown that fleas take off from their tibiae and tarsi — the insect equivalent of feet — and not their trochantera, or knees. The researchers report their conclusion in the March 1 Journal of Experimental Biology." It has been known that fleas do not use muscle power but energy stored in a protein named resilin but the researchers used high-speed video technology and mathematical models to discover where the spring action actually happens. Their bodies are laterally compressed (human anatomical terms), permitting easy movement through the hairs or feathers on the host's body (or in the case of humans, under clothing). The flea body is hard, polished, and covered with many hairs and short spines directed backward, which also assist its movements on the host. The tough body is able to withstand great pressure, likely an adaptation to survive attempts to eliminate them by mashing or scratching. Even hard squeezing between the fingers is normally insufficient to kill a flea. It is possible to eliminate them by pressing individual fleas with adhesive tape or softened beeswax (or "cheese" wax) or by rolling a flea briskly between the fingers to disable it then crushing it between the fingernails. Fleas also can be drowned in water and may not survive direct contact with anti-flea pesticides.



Hooke's drawing of a flea in *Micrographia*

Fleas lay tiny white oval-shaped eggs better viewed through a loupe. The larva is small, pale, has bristles covering its worm-like body, lacks eyes, and has mouthparts adapted to chewing. The larvae feed on various organic matter, especially the feces of mature fleas. The adult flea's diet consists solely of fresh blood. In the pupal phase, the larva is enclosed in a silken, debris-covered cocoon.

Life cycle and habitat

Fleas are holometabolous insects, going through the four life cycle stages of egg, larva, pupa, and imago (adult). The flea life cycle begins when the female lays after feeding. Adult fleas must feed on blood before they can become capable of reproduction. Eggs are laid in batches of up to 20 or so, usually on the host itself, which means that the eggs can easily roll onto the ground. Because of this, areas where the host rests and sleeps become one of the primary habitats of eggs and developing fleas. The eggs take around two days to two weeks to hatch.

Micrograph of a flea larva.

Fleas pass through a complete life cycle consisting of egg, larva, pupa and adult. A typical flea population consists of 50% eggs, 35% larvae, 10% pupae and 5% adults. Completion of the life cycle from egg to adult varies from two weeks to eight months depending on the temperature, humidity, food, and species. Normally after a blood meal, the female flea lays about 45 to 50 eggs per day up to 600 in a lifetime usually on the host (dogs, cats, rats, rabbits, mice, squirrels, chipmunks, raccoons, opossums, foxes, chickens, humans, etc.). Eggs loosely laid in the hair coat, drop out most anywhere especially where the host rests, sleeps or nests (rugs, carpets, upholstered furniture, cat or dog boxes, kennels, sand boxes, etc.). Eggs hatch in two days to two weeks into larvae found indoors in floor cracks & crevices, along baseboards, under rug edges and in furniture or beds. Outdoor development occurs in sandy gravel soils (moist sand boxes, dirt crawlspace under the house, under shrubs, etc.) where the pet may rest or sleep. Sand and gravel are very suitable for larval development which is the reason fleas are erroneously called "sand fleas."

Larvae are blind, avoid light, pass through three larval instars and take a week to several months to develop. Their food consists of digested blood from adult flea feces, dead skin, hair, feathers, and other organic debris. (Larvae do not suck blood.) Pupa mature to adulthood within a silken cocoon woven by the larva to which pet hair, carpet fiber, dust, grass cuttings, and other debris adheres. In about five to fourteen days, adult fleas emerge or may remain resting in the cocoon until the detection of vibration (pet and people movement), pressure (host animal lying down on them), heat, noise, or carbon dioxide (meaning a potential blood source is near). Most fleas overwinter in the larval or pupal stage with survival and growth best during warm, moist winters and spring.

Adult fleas cannot survive or lay eggs without a blood meal, but may live for one year without feeding. There is often a desperate need for flea control after a family has returned from a long vacation. The house has been empty with no cat or dog around for fleas to feed on. When the family and pets are gone, flea eggs hatch and larvae pupate. The adult fleas fully developed inside the pupal cocoon remains in a kind of "limbo" for a long time until a blood source is near. The family returning from vacation is immediately attacked by waiting hungry hordes of fleas. (In just 30 days, 10 female fleas under ideal conditions can multiply to over a quarter million different life stages.)

Newly emerged adult fleas live only about one week if a blood meal is not obtained. However, completely developed adult fleas can live for several months without eating, so long as they do not emerge from their puparia. Optimum temperatures for the flea's life cycle are 70°F to 85°F and optimum humidity is 70%.

Classification

Cat flea

Fleas are related to mecoptera, winged insects with good eyesight. The flightless boreid (snow scorpionfly) with its rudimentary wings seems to be close to the common ancestor of the 2000 or so known varieties of flea, which split off in many directions around 160 million years ago. Their evolution continued to produce adaptations for their specialized parasitic niche, such that they now have no wings and their eyes are covered over. The large number of flea species may be attributed to the wide variety of host species they feed on, which provides so many specific ecological niches to adapt to.

In the past, it was most commonly supposed that fleas had evolved from the flies (Diptera), based on similarities of the larvae. (Some authorities use the name **Aphaniptera** because it is older, but names above family rank need not follow the ICZN rules of priority, so most taxonomists use the more familiar name). Genetic and morphological evidence indicates that they are descendants of the Scorpionfly family Boreidae, which are also flightless; accordingly it is possible that they will eventually be reclassified as a suborder within the Mecoptera. In any case, all these groups seem to represent a clade of closely related insect lineages, for which the names Mecopteroidea and Antliophora have been proposed.

Flea systematics are not entirely fixed. While, compared to many other insect groups, fleas have been studied and classified fairly thoroughly, details still remain to be learned about the evolutionary relationships among the different flea lineages.

- Infraorder Pulicomorpha
  - Superfamily Pulicoidea
    - Family Hectopsyllidae – sticktight and chigoe fleas ("chiggers" of Latin America)
    - Family Pulicidae – common fleas
  - Superfamily Malacopsylloidea
    - Family Malacopsyllidae
    - Family Rhopalopsyllidae – hosts
    - Family Vermipsyllidae – hosts: carnivores
  - Superfamily Coptopsylloidea
    - Family Coptopsyllidae
  - Superfamily Ancistropsylloidea
    - Family Ancistropsyllidae
- Infraorder Pygiopsyllomorpha
  - Superfamily Pygiopsylloidea
    - Family Lycopsyllidae
    - Family Pygiopsyllidae
    - Family Stivaliidae
- Infraorder Hystrichopsyllomorpha
  - Superfamily Hystrichopsylloidea
    - Family Hystrichopsyllidae – hosts: rats and mice. Includes Ctenopsyllidae, Amphipsyllidae
    - Family Chimaeropsyllidae
  - Superfamily Macropsylloidea
    - Family Macropsyllidae
  - Superfamily Stephanocircidoidea
    - Family Stephanocircidae

Relationship with host

Flea bites on the back of a human

Flea bite on the waist of a human with no reaction

Fleas attack a wide variety of warm-blooded vertebrates including dogs, cats, humans, chickens, rabbits, squirrels, rats, ferrets, and mice.

Direct effects of bites

Fleas (Parasite) are not only a nuisance to humans and their pets, but can cause medical problems including flea allergy dermatitis (FAD), secondary skin irritations and, in extreme cases, anemia, tapeworms, stomach flu. Fleas can transmit murine typhus (endemic typhus) fever among animals and from animal to humans. Also, fleas can transmit bubonic plague and any other disease from human to rodent and from rodent to humans. Tapeworms normally infest in humans in severe cases. Although bites are rarely felt, it is the resulting irritation caused by the flea salivary secretions that varies among individuals. Some may witness a severe reaction (general rash or inflammation) resulting in secondary infections caused by scratching the irritated skin area. Others may show no reaction or irritation acquired after repeated bites over several weeks or months. Most bites usually found on the ankles and legs may cause irritation or pain lasting a few minutes, hours or days depending on one's sensitivity. The typical reaction to the bite is the formation of a small, hard, red, slightly raised (swollen) itching spot. There is a single puncture point in the center of each spot. (Ants and spiders leave two marks when they bite. Mosquitoes, bees, wasps and bedbugs cause a large swelling or welt.)

As a vector

Besides the problems posed by the creature itself, fleas can also act as a vector for disease. Fleas transmit not only a variety of viral, bacterial and rickettsial diseases to humans and other animals, but also protozoans and helminths.

- bacteria: Murine or endemic typhus. Fleas have helped cause epidemics by transmitting diseases such as the bubonic plague between rodents and humans by carrying *Yersinia pestis* bacteria. Fleas can transmit *Yersinia pestis*, *Rickettsia typhi*, *Rickettsia felis*, and *Bartonella henselae*.
- virus: myxomatosis.
- helminth: infestation of *Hymenolepiasis* tapeworm.
- protozoa: Trypanosome protozoans such as those of the subgenus *Herpetosoma*, use a variety of flea species opportunistically as vectors.

Fleas that specialize as parasites on specific mammals may use other mammals as hosts; therefore humans are susceptible to the predation of more than one species of flea.

Flea treatments

For humans

Fleas can become settled in a person's hair in less than ten minutes and cause soreness and itching. The itching associated with flea bites can be treated with anti-itch creams, usually antihistamines or hydrocortisone. Calamine lotion has been shown to be effective for itching.

For pets

Flea and tick repellant powder being applied to a dog

Modern flea control is approached using Integrated Pest Management (IPM) protocols at the host (pet) level. IPM is achieved by targeting fleas during at least two separate life stages, with at least two separate molecules. This is typically achieved using an adulticide to kill adult fleas and a insect development inhibitor (IDI), like lufenuron, or insect growth regulator (IGR), like methoprene, to prevent development of immature stages. Flea adults, larvae, or eggs can be controlled with insecticides. Lufenuron is a veterinary preparation (Program) that attacks the larval flea's ability to produce chitin but does not kill fleas. Flea medicines need to be used with care because many of them, especially the, also affect mammals.

Cedar oil, a non-toxic natural substance, has been proven effective in the eradication of infestations in pets. Cedar oil is being used to treat sand-flea infestation of US Military forces in the Gulf.

Since more than three quarters of a flea's life is spent somewhere other than on the host animal, it is not adequate to treat only the host; it is important also to treat the host's environment. Thorough vacuuming, washing linens in hot water, and treating all hosts in the immediate environment (the entire household, for example) are essential and if possible on a regular basis.

Contemporary commercial products for the topical treatment of flea infestations on pets contain pesticides such as imidacloprid, permethrin, and (S)-methoprene. All flea control products are recommended to be used at least half a year because the lifecycle of flea and tick can last to up to 6 months, and by using one of the flea and tick control products for so long, the infestation is highly prevented and, in the end, stopped. Although all these products are effective in fighting against flea and tick infestations, they have different active ingredients and, because cats cannot metabolize some of the compounds of the product, care must be taken in their use.

For the home

Combating a flea infestation in the home takes patience because for every flea found on an animal, there could be many more developing in the home. A spot-on insecticide will kill the fleas on the pet and in turn the pet itself will be a roving flea trap and mop up newly hatched fleas. The environment should be treated with a fogger or spray insecticide containing an insect growth regulator, such as pyriproxyfen or methoprene to kill eggs and pupae, which are quite resistant against insecticides. Frequent vacuuming is also helpful, but the vacuum bag must be disposed of immediately afterwards.

Flea "dirt" in the fur of a cat is actually excess blood from the host consumed by the adult flea and passed as feces

Diatomaceous earth can also be used as a home flea treatment in lieu of acetylcholinesterase inhibitory treatments or insecticides which carry with them a risk of poisoning for both humans and animals. However, diatomaceous earth is at least potentially dangerous to pets and people when inhaled, so care in use is recommended.

Dried pennyroyal has been suggested as a natural flea control, but is not recommended in homes with pets due to its high toxicity to mammals.

Borax is sold as a "Natural Laundry Booster" and can also be used as another home treatment for flea infestations. Borax contains boric acid which kills fleas by dehydrating them, but its safety for pets is untested.

Using dehumidifiers with air conditioning and vacuuming all may interrupt the flea life cycle. Humidity is critical to flea survival. Eggs need relative humidity of at least 70–75% to hatch, and larvae need at least 50% humidity to survive. In humid areas, about 20% of the eggs survive to adulthood; in arid areas, less than 5% complete the cycle. Fleas thrive at higher temperatures, but need 70° to 90°F (21° to 32°C) to survive. Lower temperatures slow down or completely interrupt the flea life-cycle. A laboratory study done at the University of California showed that vacuuming catches about 96% of adult fleas. A combination of controlled humidity, temperature, and vacuuming should eliminate fleas from an environment, and altering even one of these environmental factors may be enough to drastically lower and eliminate an infestation.

Chapter 4

Louse

Phthiraptera	

Light micrograph of *Fahrenholzia pinnata*

Scientific classification	
Kingdom:	Animalia
Phylum:	Arthropoda
Class:	Insecta
Subclass:	Pterygota
Infraclass:	Neoptera
Order:	**Phthiraptera** Haeckel, 1896
Suborders	

Anoplura
Rhyncophthirina
Ischnocera
Amblycera

Lice (singular: **louse**) is the common name for over 3000 species of wingless insects of the order Phthiraptera; three of which are classified as human disease agents. They are obligate ectoparasites of every avian and mammalian order except for Monotremes (the platypus and echidnas), bats, whales, dolphins, porpoises and pangolins.

Biology

Most lice are scavengers, feeding on skin and other debris found on the host's body, but some species feed on sebaceous secretions and blood. Most are found only on specific types of animal, and, in some cases, only to a particular part of the body; some animals are known to host up to fifteen different species, although one to three is typical for mammals, and two to six for birds. For example, in humans, different species of louse inhabit the scalp and pubic hair. Lice generally cannot survive for long if removed from their host.

A louse's color varies from pale beige to dark gray; however, if feeding on blood, it may become considerably darker. Female lice are usually more common than the males, and some species are even known to be parthenogenetic. A louse's egg is commonly called a nit. Many lice attach their eggs to their host's hair with specialized saliva; the saliva/hair bond is very difficult to sever without specialized products. Lice inhabiting birds, however, may simply leave their eggs in parts of the body inaccessible to preening, such as the interior of feather shafts. Living lice eggs tend to be pale white. Dead lice eggs are more yellow.

Lice are exopterygotes, being born as miniature versions of the adult, known as nymphs. The young moult three times before reaching the final adult form, which they usually reach within a month of hatching.

Ecology

Lice are optimal model organisms to study the ecology of contagious pathogens since their quantities, sex-ratios etc. are easier to quantify than those of other pathogens. The ecology of avian lice have been studied more intensively than that of mammal lice.

A few major trends

1. The average number of lice per host tends to be higher in large-bodied bird species than in small ones.
2. Louse individuals exhibit an aggregated distribution across bird individuals, i.e. most lice live on a few bird, while most birds are relatively free of lice. This

pattern is more pronounced in territorial than in colonial – more social – bird species.
3. Host taxa that dive under the water surface to feed on aquatic prey harbour fewer taxa of lice.
4. Bird taxa that are capable to exert stronger antiparasitic defense – such as stronger T cell immune response or larger uropygial glands – harbour more taxa of Amblyceran lice than others.
5. Temporal bottlenecks in host population size may couse a long-lasting reduction of louse taxonomic richness. E.g., birds introduced into New Zealand host fewer species of lice there than in Europe.
6. Louse sex ratios are more balanced in more social hosts and more female-biased in less social hosts, presumably due to the stronger isolation among louse subpopulations (living on separate birds) in the latter case.

A few effects of lousiness upon the host

1. Lice may reduce host life expectancy.
2. Lice may transmit microbial diseases or helminth parasites.
3. Ischnoceran lice may reduce the thermoregulation effect of the plumage, thus heavily infected birds loss more heat than other ones.
4. Lousiness is a disadvantage in the context of sexual rivalry.

Classification

The order has traditionally been divided into two suborders, the sucking lice (Anoplura) and the chewing lice (Mallophaga); however, recent classifications suggest that the Mallophaga are paraphyletic and four suborders are now recognised:

- Anoplura: sucking lice, occurring on mammals exclusively
- Rhyncophthirina: parasites of elephants and warthogs
- Ischnocera: mostly avian chewing lice, however, one family parasitize mammals
- Amblycera: a primitive suborder of chewing lice, widspread on birds, however, also live on South-American And Australian mammals

It has been suggested that the order is contained by the Troctomorpha suborder of Psocoptera.

Lice in humans

Humans host three different kinds of lice: head lice, body lice , and pubic lice. Lice infestations can be controlled with lice combs, and medicated shampoos or washes. Adult and nymphal lice can survive on sheep-shearers' moccasins for up to 10 days, but microwaving the footwear for five minutes in a plastic bag will kill the lice.

Human lice and DNA discoveries

Lice have been the subject of significant DNA research that has led to discoveries on human evolution. For example, recent DNA evidence suggests that pubic lice spread to humans approximately 2,000,000 years ago from gorillas. Additionally, the DNA differences between head lice and body lice provide corroborating evidence that humans started losing body hair, also about 2,000,000 years ago.

Ricinus bombycillae, an Amblyceran louse from the bohemian waxwing

Trinoton anserinum, an Amblyceran louse from a mute swan.

Damalinia limbata is an Ischnoceran louse from goats. The male is smaller than the female.

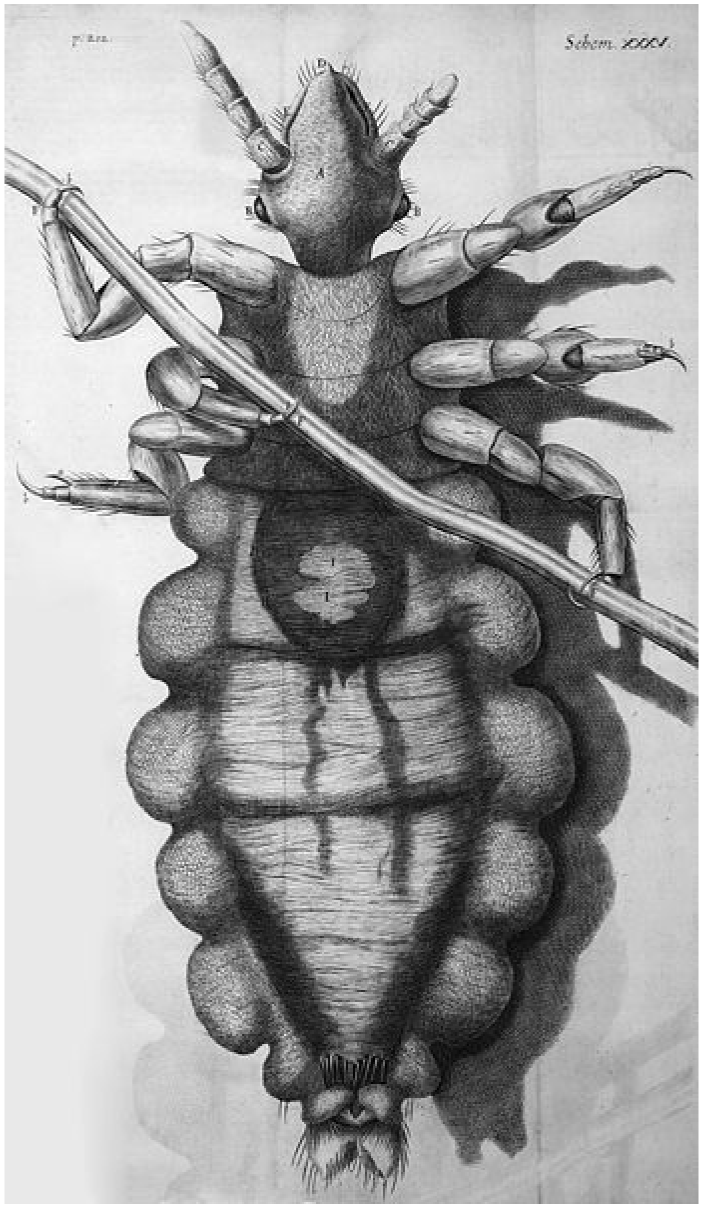


Diagram of a louse, by Robert Hooke, 1667.

Chapter 5

Tick

Ticks	
	
Adult deer tick, *Ixodes scapularis*	
Scientific classification	
Kingdom:	Animalia
Phylum:	Arthropoda
Class:	Arachnida
Subclass:	Acari
Superorder:	Parasitiformes
Order:	**Ixodida** Leach, 1815
Superfamily:	**Ixodoidea** Leach, 1815
Families	

- Ixodidae – hard ticks
- Argasidae – soft ticks
- Nuttalliellidae

Diversity

18 genera, c. 900 species

Tick is the common name for the small arachnids in superfamily **Ixodoidea** that, along with other mites, constitute the Acarina. Ticks are ectoparasites (external parasites), living by hematophagy on the blood of mammals, birds, and occasionally reptiles and amphibians. Ticks are vectors of a number of diseases, including Lyme disease, Q fever (rare; more commonly transmitted by infected excreta), Colorado tick fever, tularemia, tick-borne relapsing fever, babesiosis, ehrlichiosis and tick-borne meningoencephalitis, as well as bovine anaplasmosis.

Habitats and behaviors

Ticks are blood-feeding parasites that are often found in tall grass where they will wait to attach to a passing host. A tick will attach itself to its host by inserting its chelicerae (cutting mandibles) and hypostome (feeding tube) into the skin. The hypostome is covered with recurved teeth and serves as an anchor.

Seed ticks (tick larvae) also attack horses, cattle, moose, lions and other mammals, causing anemia, various diseases, paralysis and even death. Such infestations can be difficult to detect until thousands have attached themselves to an animal and eradication can be difficult.

Frequent grooming and chemical applications may control the spread of ticks.

Changes in temperature and day length are some of the factors signalling a tick to seek a host. Ticks can detect heat emitted or carbon dioxide respired from a nearby host. They will generally drop off the animal when full, but this may take several days. In some cases ticks will live for some time on the blood of an animal. Ticks are more active outdoors in warm weather, but can attack a host at any time.

Ticks can be found in most wooded or forested regions throughout the world. They are especially common in areas where there are deer trails or human tracks. Ticks are especially abundant near water, where warm-blooded animals come to drink, and in meadows wherever shrubs and brush provide woody surfaces and cover.

Population control

Ticks are a vector for a number of diseases including Lyme disease, Rocky Mountain spotted fever and other tick-borne disease.

Case study of the American deer tick

The black legged or deer tick (*Ixodes scapularis*) is dependent on the white-tailed deer for reproduction. Larval and nymph stages (immature ticks that cannot reproduce) of the deer tick feed on birds and small mammals. The adult female tick needs a large 3 day blood meal from the deer before she can reproduce and lay her 2000 or more eggs. Deer are the primary host for the adult deer tick and are key to the reproductive success of the tick.

Numerous studies have shown that abundance and distribution of deer ticks are correlated with deer densities.

When the deer population was reduced by 74% at a 248-acre (100 ha) study site in Bridgeport, Connecticut, for example, the number of nymphal ticks collected at the site decreased by 92%. The relationship between deer abundance, tick abundance, and human cases of Lyme disease was well documented in the Mumford Cove Community in Groton, Connecticut, from 1996 to 2004. The deer population in Mumford Cove was reduced from about 77 deer per square mile to about 10 deer per square mile (4 deer per square kilometer) after 2 years of controlled hunting. After the initial reduction the deer population was maintained at low levels. Reducing deer densities to 10 deer per square mile (4 deer per square kilometer) was adequate to reduce by more than 90% the risk of humans contracting Lyme disease in Mumford Cove.

A 2006 study by Penn State's Center for Infectious Disease Dynamics indicated that reducing the deer population in small areas may lead to higher tick densities, resulting in more tick-borne infections in rodents leading to a high prevalence of tick-borne encephalitis and creating a tick hot-spot.

Other control measures

Male tick size comparison to a match

The parasitic Ichneumon wasp *Ixodiphagus hookeri* has long been investigated for its potential to control tick populations. It lays its eggs into ticks; the hatching wasps kill their host.

Another natural form of control for ticks is the guineafowl, a bird species which consumes mass quantities of ticks. Just 2 birds can clear 2 acres (8,100 m^2) in a single year.

Topical (drops/dust) flea/tick medicines may be toxic to animals and humans. Phenothrin (85.7%) in combination with Methoprene was a popular topical flea/tick therapy for felines. Phenothrin kills adult fleas and ticks. Methoprene is an insect growth regulator that interrupts the insect's life cycle by killing the eggs. However, the U.S. Environmental Protection Agency required at least one manufacturer of these products to withdraw some products and include strong cautionary statements on others, warning of adverse reactions.

Classification

Engorged tick attached to back of toddler's head. Adult thumb shown for scale.

Head of *Ixodes ricinus* (sheep tick)

Ixodes hexagonus

There are three families of ticks, one of which - Nuttalliellidae comprises a single species, *Nuttalliella namaqua*. The remaining two families contain the hard ticks (Ixodidae) and the soft ticks (Argasidae).

Ixodidae

Dermacentor

- *Dermacentor variabilis*, the American dog tick, is perhaps the most well-known of the North American hard ticks. This tick does not carry Lyme disease but can carry Rocky Mountain spotted fever.

Ixodes

- *Ixodes scapularis* (formerly *Ixodes dammini*), known as the black-legged tick or deer tick, is common to the eastern part of North America and is known for spreading Lyme disease.
- *Ixodes pacificus*, the Western black-legged tick, lives in the western part of North America and is responsible for spreading Lyme disease and Rocky Mountain spotted fever. It tends to prefer livestock such as cows as its adult host.
- The most medically important species of tick in Australia is the paralysis tick, *Ixodes holocyclus*. It is found in a 20-kilometre band that follows the eastern coastline of Australia. Encounters with these parasites are relatively common as this is where much of the human population resides in New South Wales and South-East Queensland. Although most cases of tick bite are uneventful, some can result in life threatening illnesses including paralysis, tick typhus, and severe allergic reactions both in humans and pets.

Rhipicephalus

- The southern cattle tick, *Rhipicephalus microplus*, causes annual economic losses in the hundreds of millions of dollars to cattle producers throughout the world, and ranks as the most economically important tick globally. This tick also attacks sheep, horses, goats, and a few related species, but cattle are the most important hosts.

Amblyomma

- The Lone Star tick, *Amblyomma americanum*, is part of the Ixodidae family. The adult females are distinguished by a white dot or "lone star" on its back. The adult males can also be seen with dots and white streaks on the edge of their bodies. This tick has been associated with transmission of Southern Tick Associated Rash Illness (STARI) in humans. The etiologic agent causing STARI is unknown, but a spirochete, *Borrelia lonestari*, was detected in the skin of one patient and the lone star tick that bit him. However, subsequent study of over two dozen STARI patients has found no evidence of *B. lonestari* infection.

Argasidae

Members of this family include *Argas* and *Ornithodoros*.

Fossil record

Fossil ticks are common. Recent hypotheses based on total-evidence approach analysis place the origin of ticks in the Cretaceous (65 to 146 million years ago) with most of the evolution and dispersal occurring during the Tertiary (5 to 65 million years ago). The oldest example is an argasid (bird) tick from Cretaceous New Jersey amber. The younger

Baltic and Dominican ambers have also yielded examples, all of which can be placed in living genera.

Chapter 6

Bothriocephalus Acheilognathi and Bucephalus Polymorphus

Bothriocephalus acheilognathi

Bothriocephalus acheilognathi	
Scientific classification	
Kingdom:	Animalia
Phylum:	Cestoda
Class:	Cestoidea
Order:	Pseudophyllidea
Family:	Bothriocephalidae
Genus:	*Bothriocephalus*
Species:	***B. acheilognathi***
Binomial name	
Bothriocephalus acheilognathi	

Bothriocephalus acheilognathi, also known as the **Asian Tape Worm**, is a freshwater fish parasite that originated from China and Eastern Russia. *B. acheilognathi* has a fleshy, arrow-head scolex (head region) with an undeveloped terminal disc, a ribbon-like segmented body called proglottids, and two long attachment grooves called "bothria." The tapeworm is a generalized parasite that affects a wide variety of fish hosts, particularly cyprinids, contributing to its overall success. *B. acheilognathi's* favored host is the carp.

Pathology

The parasite attaches near the anterior portion of the intestine, just posterior to the bile duct. An accumulation of tapeworms in this area leads to digestive tract blockage that distends the intestinal wall leading to perforation. When attached, *B. acheilognathi* envelopes parts of the intestines and induces an inflammatory response. The inflammation can lead to hemorrhage and necrosis. Symptoms can also include, weight loss, anemia, and mortality (especially in young fishes). Infections can be detected by the presence of eggs or body parts in feces, and by the presence of the tapeworm in the gut of the fish.

Life cycle

The life cycle of *B. acheilognathi* involves a definitive host, a fish, and an intermediate host, a copepod. The adult tapeworm is hermaphroditic; each proglottid has a complete set of both male and female reproductive organs and produces eggs via self-fertilization. The tapeworm is sensitive to temperature, in addition the species is thermophilic; lower temperatures interfere and delay development and completion of the life cycle. The eggs are released into the water through the fish fecal material, where they hatch into free-swimming hexacanth (six-hooked) larvae. Between 1–28 days, the eggs will hatch according to the water temperature range it is in. Eggs that hatch within 1–5 days occur at temperatures between 28-30°C and eggs that hatch within 10–28 days occur at temperatures between 14- 15°C.

When the free-swimming larvae, called coracidia, are eaten by copepods (intermediate host), it penetrates into the gut wall, travels to the coelom, and develops into a second larval stage called a procercoid (infective form) all within 6–10 days. Once the infected copepods are eaten by the fish hosts, the procercoid rapidly transform into the plerocercoid stage and attaches to the intestinal gut wall, where it develops into the adult parasite over the course of 21–23 days.

Ecological Impact

Studies have shown that *B. acheilognathi* decreases the size of fish worldwide causing great economic loss in hatcheries and fish farms. The Asian Tape Worm was introduced globally via grass carp. Infestation is intermittent and follows a clear seasonal pattern with peak incidence in the summer. There are multiple chemotherapeutic solutions to fight infection. Tinostat, Yomesan, Droncit are examples of drugs (when mixed in fish food with oil) that are effective in relieving infection. Eliminating an infestation can be amplified by control of copepods in water.

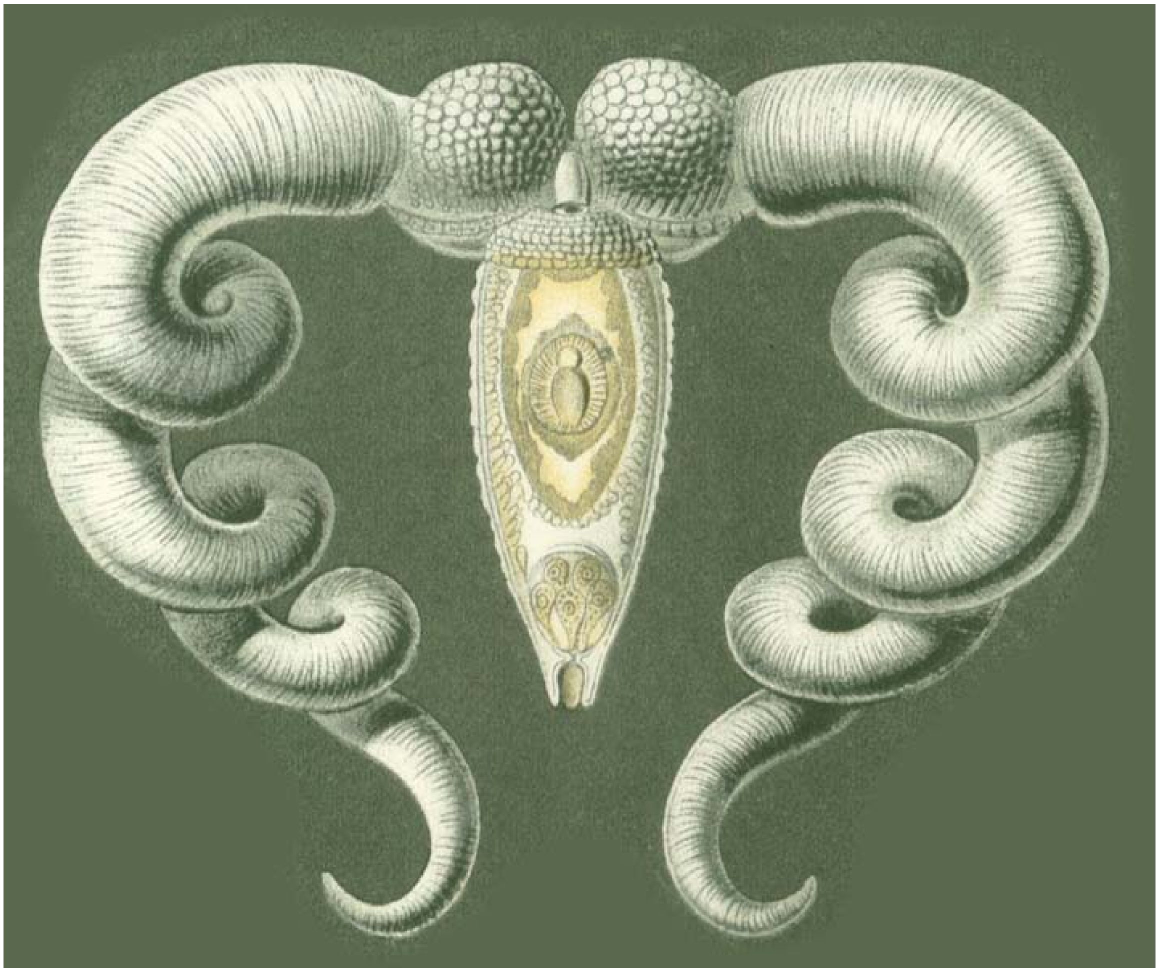
The discovery of the tapeworm’s substantial infections within the crucian carp (Carassius carassius) population in the UK is of particular concern because there have not been any known natural tapeworm parasites of these crucian carp. A plausible cause may be that the crucian carp have limited immunological defenses against this parasite.

Bucephalus polymorphus

Bucephalus polymorphus	
	
Cercaria larva of *B. polymorphus* from Encyclopaedia Britannica, Eleventh Edition	
Scientific classification	
Kingdom:	Animalia
Phylum:	Platyhelminthes
Class:	Trematoda
Subclass:	Digenea
Order:	Strigeidida
Superfamily:	Bucephaloidea
Family:	Bucephalidae
Genus:	*Bucephalus*
Species:	***B. polymorphus***
Binomial name	
Bucephalus polymorphus	

Bucephalus polymorphus is a species of the Bucephalidae family of Digenea, a subclass of Trematodes within the phylum Platyhelminthes. It is characterized by having a mouth near middle of body along with a sac-like gut. The adults occur in the centre of the ventral surface. The adults occur in the gut of marine and fresh-water fish. The metacercariae encyst in smaller fish, sometimes in the nervous system. These parasitic flatworms are dorso-ventrally flattened animals characterized by a bilaterally symmetrical body enclosed within a syncytial tegument. They have a distal, anucleate later (distal cytoplasm). The distal cytoplasm contains vesicular inclusions that are Golgi derived. The adults of these acoelomate worms are common in the digestive tract, but are also found in other organs of vertebrates. The adult parasite attaches via a characteristic anterior adhesive organ with tentacles. Bucephalus are native to North American fresh waters that parasitize freshwater bivalves.

Taxonomy

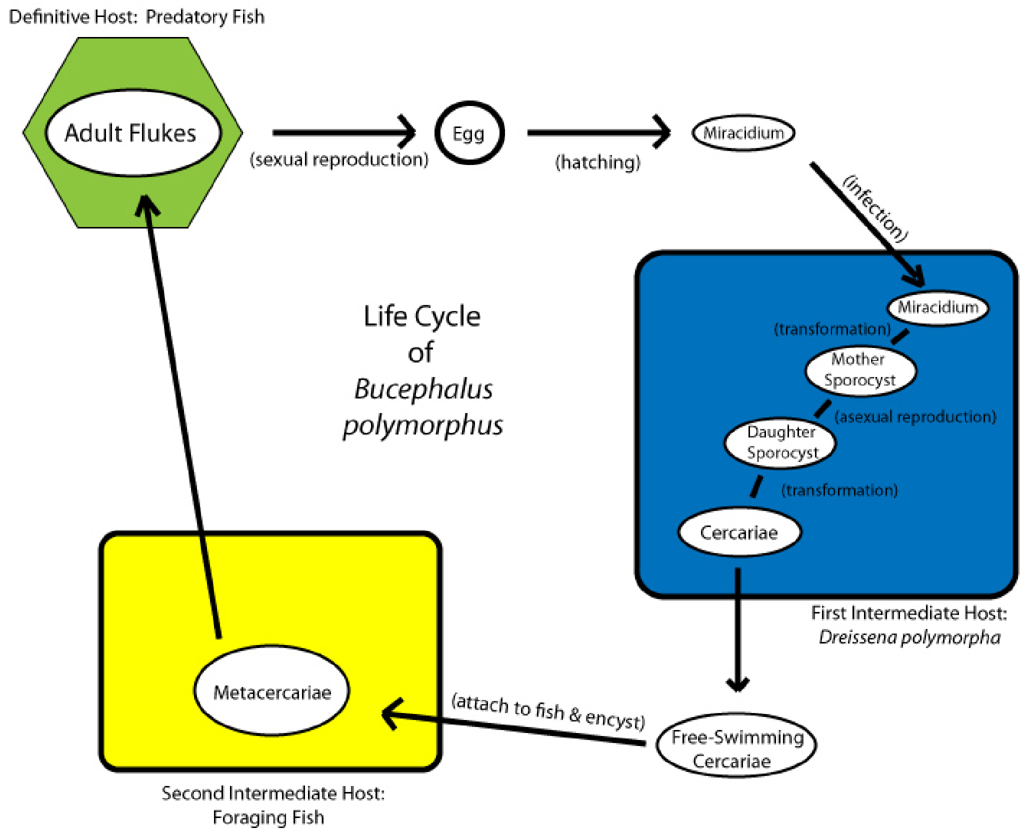


Bucephalid cercaria larva from Ernst Haeckel's Kunstformen der Natur (1904).

The genus *Bucephalus* was based on this species, which was the earliest known, initially described by Baer (1827) from its cercaria. Von Siebold (1848) believed that the adult bucephalid he named *Gasterostomum fimbriatum* represented an adult form of the same bucephalid, but this identity has never been proven.

Life cycle

Dignetic flatworm species require more than one host to complete a full life cycle. *Bucephalus polymorphus* requires three hosts. Dreissena polymorpha, a small freshwater mussel, is the first intermediate host parasitized by the hatching miracidium. Within the visceral mass of Dreissena, the miracidium transforms into a mother (primary) sporocyst. Asexual reproduction produces many daughter (secondary) sporocysts, which eventually become cercariae. Rapid proliferation of sporocysts results in a knotted white mass of tubules, which is found primarily in the gonads of the mussel. Released from the infected mussels, cercariae attach to fish (second intermediate host), encyst, and transforms into metacercariae. The third (definitive) hosts are predatory fish that consume the infected foraging fish. It has been shown experimentally that cercarial emergence exhibits a circadian rhythm of shedding with a peak in the dark period of a light:dark 12:12 h photoperiod .



Influence on Host

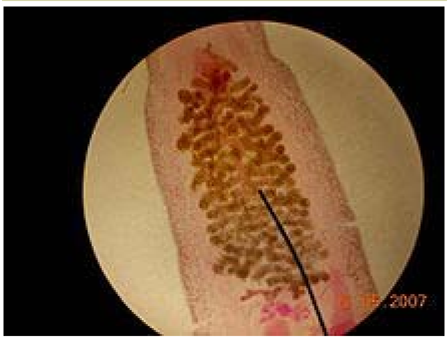
B. polymorphus is a parasite residing exclusively in host connective tissues. The gonads of its first intermediate host, *Dreissena*, is the primary target of infection and sporocyst proliferation. As infection intensifies, the sporocyst develops branches through connective tissue passages, emerges from the gonads, and can spread into other body regions. Such secondary sites of infection have been previously reported to occur in the digestive glands, the gills, the bundles of adductor muscle, and the mantle epithelium lining the interior of the shells. The study found that the digestive glands of infected bivalves appeared to be relatively normal when compared to full bodied, uninfected specimens. Within the sporocyst, the cercariae existed within a wide range of developmental stages, indicating that its development is asynchronous. It was also observed that heavy infection of the parasite led to host castration, which left the entire gonadal space often occupied by the sporocysts. By limiting the infection almost exclusively to the gonads, the parasites have developed an interesting strategy to only use the reproductive energy of their hosts, thereby minimizing the risk of host mortality. Extending the life of a bucephalus infected host is important because this allows the parasite to proliferate continuously from year to year, since infected gonads produce cercariae instead of gametes. The location of the sporocyst (primarily in gonads), its overall shape, irregular branches, and the morphology of its cercariae with a bifurcated tail, distinguish *B. polymorphus* from other trematode parasites of zebra mussels in histological sections.

Prevalence

Infection of *B. polymorphus* is geographically widely distributed. However, prevalence of infection in zebra mussel population is not common. A study by Lajtner *et al.*, which surveyed the zebra mussel population in the Drava River in Croatia, found a prevalence of 21.3%. In the most extensive study conducted to date, a prevalence of 73% was recorded in zebra mussel in South-Eastern France. Low rates of prevalence were also recorded: 1% (Kuperman *et al.* 1994), 1-4% (Baturo 1977), 2-5% (Smirnova and Ibrasheva 1967), 9% (Malloy *et al.* 1996), and 13-28% (de Kinkelin *et al.* 1968). Therefore, the prevalence of *Bucephalus polymorphus* can vary widely depending on the ecosystem.

Chapter 7

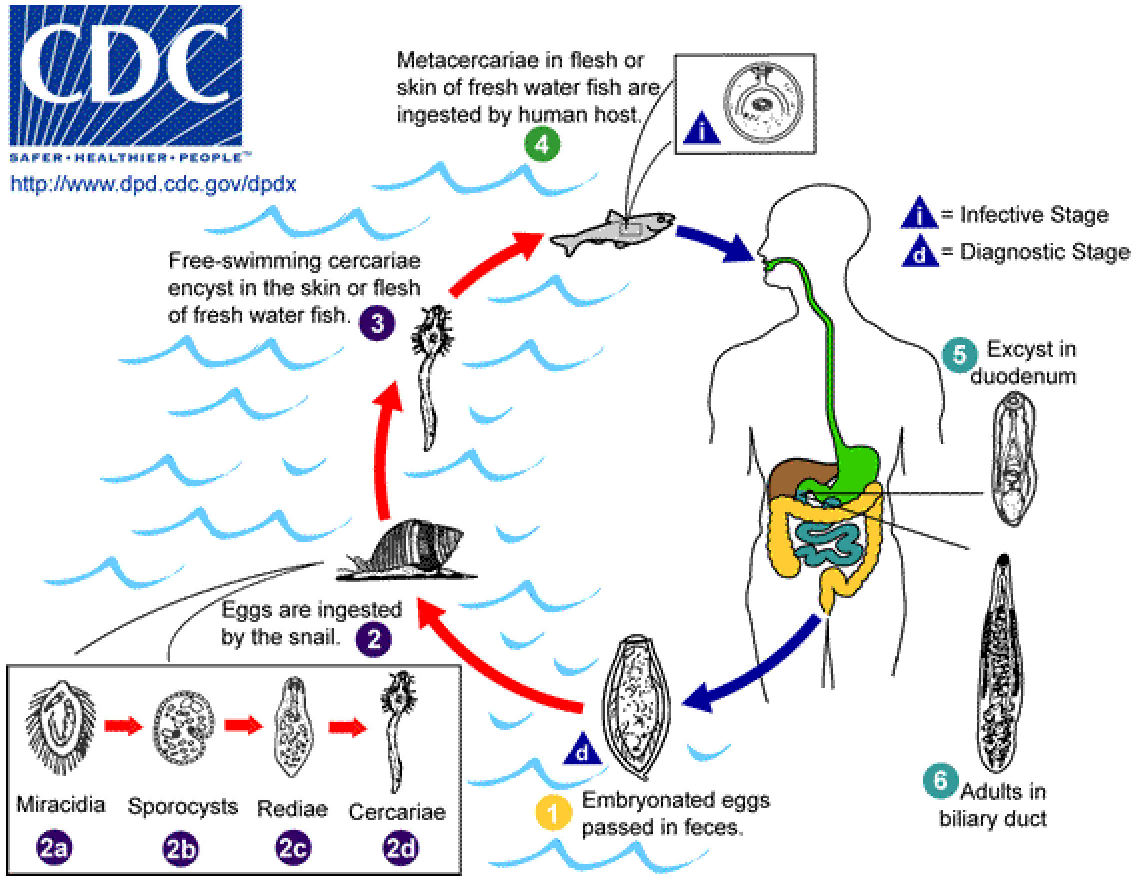
Clonorchis Sinensis

Clonorchis sinensis	
	
Clonorchis sinensis under a light microscope. Notice the ovaries. This species is monoecious,	
Scientific classification [e]	
Kingdom:	Animalia
Phylum:	Platyhelminthes
Class:	Trematoda
Order:	Opisthorchiida
Family:	Opisthorchiidae
Genus:	*Clonorchis*
Species:	***C. sinensis***
Binomial name	
Clonorchis sinensis Looss, 1907	

Clonorchis sinensis, the Chinese liver fluke, is a human liver fluke in the class Trematoda, Phylum Platyhelminthes. This parasite lives in the liver of humans, and is found mainly in the common bile duct and gall bladder, feeding on bile. These animals, which are believed to be the third most prevalent worm parasite in the world, are endemic to Japan, China, Taiwan, and Southeast Asia, currently infecting an estimated 30,000,000 humans.

Life cycle

An adult *Clonorchis sinensis* has these main body parts: oral sucker, pharynx, caecum, ventral sucker, vitellaria, uterus, ovary, Mehlis' gland, testes, exretory bladder.



Life cycle of *Clonorchis sinensis*

The egg of a *Clonorchis sinensis* (commonly: human liver fluke), which contains the miracidium that develops into the adult form, floats in freshwater until it is eaten by a snail.

First intermediate host

Freshwater snail *Parafossarulus manchouricus* - synonym: *Parafossarulus striatulus*, often serves as a first intermediate host for *Clonorchis sinensis* in China, Japan, Korea and Russia.

Other snail hosts include:

- *Bithynia longicornis* - synonym: *Alocinma longicornis* - in China
- *Bithynia fuchsiana* - in China
- *Bithynia misella* - in China
- *Parafossarulus anomalosiralis* - in China
- *Melanoides tuberculata* - in China
- *Semisulcospira libertina* - in China
- *Assiminea lutea* - in China
- *Tarebia granifera* - in Taiwan, China

Once inside of the snail body, the miracidium hatches from the egg, and parasitically grows inside of the snail. The miracidium develops into a sporocyst, which in turn house the asexual reproduction of redia, the next stage. The redia themselves house the asexual reproduction of free-swimming cercaria. This system of asexual reproduction allows for an exponential multiplication of cercaria individuals from one miracidium. This aids the *Clonorchis* in reproduction, because it enables the miracidium to capitalize on one chance occasion of passively being eaten by a snail before the egg dies.

Once the redia mature, having grown inside the snail body until this point, they actively bore out of the snail body into the freshwater environment.

Second intermediate host

There, instead of waiting to be consumed by a host (as is the case in their egg stage), they seek out a fish. Boring their way into the fish's body, they again become parasites of their new hosts.

Once inside of the fish muscle, the cercaria create a protective metacercarial cyst with which to encapsulate their bodies. This protective cyst proves useful when the fish muscle is consumed by a human.

Definitive host

The acid-resistant cyst enables the metacercaria to avoid being digested by the human gastric acids, and allows the metacercaria to reach the small intestine unharmed. Reaching the small intestines, the metacercaria navigate toward the human liver, which becomes its final habitat. *Clonorchis* feed on human bile created by the liver. In the human liver, the mature *Clonorchis* reaches its stage of sexual reproduction. The hermaphroditic adults produce eggs every 1–30 seconds, resulting in the rapid multiplication of inhabitants in the liver.

Effects on human health

Dwelling in the bile ducts, *Clonorchis* induces an inflammatory reaction, epithelial hyperplasia and sometimes even cholangiocarcinoma, the incidence of which is raised in fluke-infested areas.

One adverse effect of *Clonorchis* is the possibility for the adult metacercaria to consume all bile created in the liver, which would inhibit the host human from digesting, especially fats. Another possibility is obstruction of the bile duct by the parasite or its eggs, leading to biliary obstruction and cholangitis (specifically oriental cholangitis).

Central Serous Retinopathy (CSR) a report of 80 cases by Dr. John Chiao-nan Chang, M.D. and Dr. Yin-Ping Wang, M.D. Hong Kong on page 125 of their report observed that 19% of the cases of CSR in their sample tested positive for *Clonorchis sinensis*.

Treatment

Drugs used to treat infestation include triclabendazole, praziquantel, bithionol, albendazole and mebendazole.

Chapter 8

Cordylobia Anthropophaga

Cordylobia anthropophaga	
Scientific classification	
Kingdom:	Animalia
Phylum:	Arthropoda
Class:	Insecta
Order:	Diptera
Family:	Calliphoridae
Tribe:	Calliphorini
Genus:	*Cordylobia*
Species:	***C. anthropophaga***
Binomial name	
Cordylobia anthropophaga (Blanchard, 1872)	
Synonyms	
Ochromyia anthropophaga Blanchard, 1872	

Cordylobia anthropophaga, the *mango fly*, *tumbu fly*, *tumba fly*, *putzi fly* or *skin maggot fly* is a species of blow-fly common in East and Central Africa. It is a parasite of large mammals (including humans) during its larval stage .*C. anthropophaga* has been endemic in the subtropics of Africa for more than 135 years and is a common cause of myiasis in humans in the region.

Its specific epithet *anthropophaga* derives from the Greek word *anthropophagus*, "eater of men".

"The Mode of infection by the Cayor Worm. Doctors Rodhain and Bequaert conclude, from their observations in the Congo Free State, that *Cordylobia anthropophaga* (Grunberg) lays its eggs on the ground. The larvae, known generally as Cayor worms, crawl over the soil until they come in contact with man or a mammal, penetrate the skin and lie in the subcutaneous tissue, causing the formation of tumors. On reaching full growth, the larvae leave the host, fall to the ground, bury themselves and there pupate. This fly is said to be the most common cause of human or animal myiasis in tropical Africa, from Senegal to Natal. In the region of Bas-Katanga where these investigations were made, dogs appeared to be the principal hosts, although *Cordylobia* larvae were found also in guinea-pigs, a monkey and two white men. The larvae are always localized on those parts of the hosts which come in immediate contact with the soil." (*Ann. Soc. Entom. de Belgique*, Iv, pp. 192–197, 1911) summary translation in *Entomological News.* 1911 Vol. xxii:467.

History of Discovery

The larvae of the tumbu fly, *Cordylobia anthropophaga*, were first described in Senegal in 1862, and Blanchard first described the adult and gave it its name in 1893. In 1903, Grunbert placed the tumbu fly in a new genus, *Cordylobia*.

Life cycle

Female tumbu flies deposit 100-300 eggs in sandy soil often contaminated with animal feces. The hatched larvae can remain viable in the soil for 9–15 days until they need to find a host for development. If a larva finds a host, it will penetrate the skin and take 8–12 days developing through three larval stages before it reaches the prepupal stage. It will then leave the host, drop to the ground, bury itself, and pupate. It then becomes an adult fly able to reproduce and begin the cycle all over again.

Clinical Presentation in Humans

Successful penetrations in humans will result in furuncular (boil-like) myiasis, typically on the backs of arms or about the waist, lower back, or buttocks.

C. anthropophaga rarely causes severe problems, and mainly causes cutaneous myiasis. Geary et al. describe the presentation of cutaneous myiasis caused by the tumbu fly: "At the site of penetration, a red papule forms and gradually enlarges. At first the host may experience only intermittent, slight itching, but pain develops and increases in frequency and intensity as the lesions develop into a furuncle. The furuncle's aperture opens, permitting fluids containing blood and waste products of the maggot to drain."

Transmission

Female tumbu flies lay their eggs in soil contaminated with feces or urine or on damp clothing or bed linens. Damp clothing hanging to dry makes for a perfect spot. The larvae hatch in 2–3 days and attach to unbroken skin and penetrate the skin, producing swelling.

If the larvae hatch in soil, any disturbance of the soil causes them to wiggle to the surface to penetrate the skin of the host.

Reservoir and Vector

A reservoir is defined as an organism that can harbor a pathogen indefinitely with no ill effects. Although *C. anthropophaga* larvae can cause ill effects for animal hosts, because we are talking about myiasis in humans, we will consider any animal hosts as reservoirs.

Many animals are hosts of *C. anthropophaga*. The dog is the most common domestic host and several species of wild rats are the preferred field hosts. Domestic fowl are dead-end hosts, meaning that the larvae cannot develop when they enter the tissue of fowl.

Humans are in fact accidental hosts, which means that tumbu fly larvae do not usually infect humans and we are not necessary for the transmission cycle of the fly.

A vector is an organism that carries the parasites (the larvae) from one host to another. The tumbu fly itself is the vector in a loose sense, because the female deposits the eggs in soil or on damp cloth, where the larvae can hatch and attach to human or animal skin.

Diagnostics

Cutaneous myiasis caused by the tumbu fly should be suspected when a patient who has just spent time in Africa presents with ulcers or boil-like sores. A definitive diagnose can only be made when the larvae are found. They should be removed and allowed to develop into adult flies for identification purposes.

Treatment

In cases of cutaneous myiasis, the larvae are most often removed without an incision. Applying Vaseline to the skin blocks the breathing hole and cuts off the larva's air supply. This will force the maggot to the surface of the skin as it searches for air. The larvae can then easily be extracted from the skin. If this does not work, local anesthetic can be administered and an incision is made to extract the maggot.

Patients should be monitored for additional and subsequent lesions as development does not occur all at once and some larvae may take longer to reach the prepupal stage. To prevent bacterial infection after removal of the larvae, antibiotics can be administered.

Epidemiology

C. anthropophaga is the most common causes of myiasis in Africa (WHO).

The tumbu fly is endemic to the tropical regions of Africa, south of the Sahara desert. Myiasis caused by *C. anthropophaga* is the most common cause of myiasis in Africa but can be seen worldwide because of air travel, as human movements carry infestation outside endemic areas.

Public Health and Prevention Strategies

The fly commonly infects humans by laying its eggs on wet clothes, left out to dry. The eggs hatches in one to three days and the larvae (who can survive without a host for up to 15 days) then burrow into the skin when the clothes are worn. A prevention method is to iron all clothes (including underwear) which will kill the eggs/larvae.

Chapter 9

Emerald Cockroach Wasp and Fasciola Hepatica

Emerald cockroach wasp

Emerald cockroach wasp	

Ampulex compressa

Scientific classification	
Kingdom:	Animalia
Phylum:	Arthropoda
Class:	Insecta
Order:	Hymenoptera

Suborder:	Apocrita
Superfamily:	Apoidea
Family:	Ampulicidae
Genus:	*Ampulex*
Species:	***A. compressa***
Binomial name	
Ampulex compressa (Fabricius, 1781)	

The **emerald cockroach wasp** or **jewel wasp** (*Ampulex compressa*) is a solitary wasp of the family Ampulicidae. It is known for its unusual reproductive behavior, which involves stinging a cockroach and using it as a host for its larvae. It thus belongs to the entomophagous parasites.

Distribution

The wasp is mostly found in the tropical regions of South Asia, Africa and the Pacific islands. The flying wasps are more abundant in the warm seasons of the year.

A. compressa was introduced to Hawaii by F.X. Williams in 1941 as a method of biocontrol. This has been unsuccessful because of the territorial tendencies of the wasp, and the small scale on which they hunt.

Appearance

The wasp has a metallic blue-green body, with the thighs of the second and third pair of legs red. The female is about 22 mm long; the male is smaller and lacks a stinger.

Reproductive behavior and life cycle

As early as the 1940s it was reported that female wasps of this species sting a roach (specifically a *Periplaneta americana*, *Periplaneta australasiae* or *Nauphoeta rhombifolia*) twice, delivering venom. A 2003 study using radioactive labeling demonstrated that the wasp stings precisely into specific ganglia of the roach. It delivers an initial sting to a thoracic ganglion and injects venom to mildly and reversibly paralyze the front legs of its victim. This facilitates the second venomous sting at a carefully chosen spot in the roach's head ganglia (brain), in the section that controls the escape reflex. As a result of this sting, the roach will first groom extensively, and then become sluggish and fail to show normal escape responses. In 2007 it was reported that the venom of the wasp blocks receptors for the neurotransmitter octopamine.

The wasp proceeds to chew off half of each of the roach's antennae. Researchers believe that the wasp chews off the antenna to replenish fluids or possibly to regulate the amount of venom because too much could kill and too little would let the victim recover before the larva has grown. The wasp, which is too small to carry the roach, then leads the victim to the wasp's burrow, by pulling one of the roach's antennae in a manner similar to a leash. Once they reach the burrow, the wasp lays a white egg, about 2 mm long, on the roach's abdomen. It then exits and proceeds to fill in the burrow entrance with pebbles, more to keep other predators out than to keep the roach in.

With its escape reflex disabled, the stung roach will simply rest in the burrow as the wasp's egg hatches after about three days. The hatched larva lives and feeds for 4–5 days on the roach, then chews its way into its abdomen and proceeds to live as an endoparasitoid. Over a period of eight days, the wasp larva consumes the roach's internal organs in an order which guarantees that the roach will stay alive, at least until the larva enters the pupal stage and forms a cocoon inside the roach's body. Eventually the fully grown wasp emerges from the roach's body to begin its adult life. Development is faster in the warm season.

Adults live for several months. Mating takes about one minute, and only one mating is necessary for a female wasp to successfully parasitize several dozen roaches.

While a number of venomous animals paralyze prey as live food for their young, *Ampulex compressa* is different in that it initially leaves the roach mobile and modifies its behavior in a unique way. Several other species of the genus *Ampulex* show a similar behavior of preying on cockroaches. The wasp's predation appears only to affect the cockroach's escape responses. Research has shown that while a stung roach exhibits drastically reduced survival instincts (such as swimming, or avoiding pain) for approximately 72 hours, motor abilities like flight or flipping over are unimpaired.

Fasciola hepatica

Fasciola hepatica	
	
Fasciola hepatica - adult worm	
Scientific classification [e]	
Kingdom:	Animalia
Phylum:	Platyhelminthes
Class:	Trematoda
Order:	Echinostomida
Family:	Fasciolidae
Genus:	*Fasciola*
Species:	***F. hepatica***
Binomial name	
Fasciola hepatica Linnaeus, 1758	

Fasciola hepatica, also known as the **common liver fluke** or **sheep liver fluke**, is a parasitic flatworm of the class Trematoda, phylum Platyhelminthes that infects liver of various mammals, including humans. The disease caused by the fluke is called fascioliasis (also known as fasciolosis). *F. hepatica* is distributed worldwide and causes great economic losses in sheep and cattle.

Life cycle

In order to complete its life cycle, *Fasciola hepatica* requires a freshwater snail as an intermediate host such as *Galba truncatula*, in which the parasite can reproduce asexually.

Species in the family Lymnaeidae are serving as naturally or experimentally intermediate hosts of *Fasciola hepatica* and they include: *Austropeplea tomentosa*, *Austropeplea ollula*, *Austropeplea viridis*, *Radix peregra*, *Radix lagotis*, *Radix auricularia*, *Radix* *natalensis*, *Radix rubiginosa*, *Omphiscola glabra*, *Lymnaea stagnalis*, *Stagnicola fuscus*, *Stagnicola palustris*, *Stagnicola turricula*, *Pseudosuccinea columella*, *Lymnaea viatrix*, *Lymnaea neotropica*, *Fossaria bulimoides*, *Lymnaea cubensis*, *Lymnaea* sp. from Colombia, *Galba truncatula*, *Lymnaea cousini*, *Lymnaea humilis*, *Lymnaea diaphana*, *Stagnicola caperata* and *Lymnaea occulta*.

From the snail, minute cercariae emerge and swim through pools of water in pasture, and encyst as metacercariae on near-by vegetation. From here, the metacercariae are ingested by the ruminant, or in some cases, by humans eating un-cooked foods such as water-cress. Contact with low pH in the stomach causes the early immature juvenile to begin the process of excystment. In the duodenum, the parasite breaks free of the metacercariae and burrows through the intestinal lining into the peritoneal cavity. The newly excysted juvenile does not feed at this stage, but once it finds the liver parenchyma after a period of days, feeding will start. This immature stage in the liver tissue is the pathogenic stage, causing anaemia and clinical signs sometimes observed in infected animals. The parasite browses on liver tissue for a period of up to 5–6 weeks and eventually finds its way to the bile duct where it matures into an adult and begins to produce eggs. Up to 25,000 eggs per day per fluke can be produced, and in a light infection, up to 500,000 eggs per day can be deposited onto pasture by a single sheep.

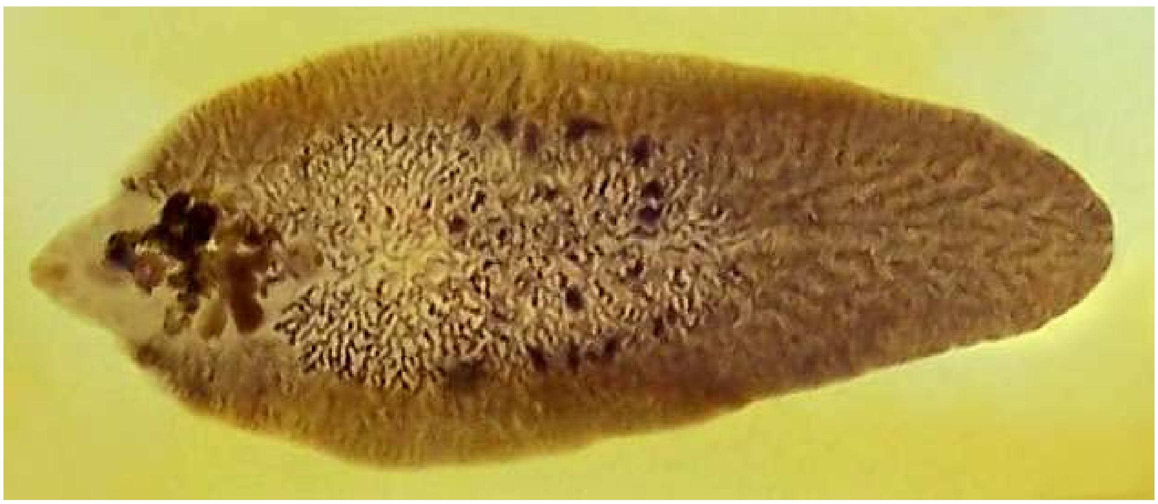
Disease biology

Egg of *F. hepatica*

In the United Kingdom, *Fasciola hepatica* is a frequent cause of disease in ruminants - this is most common between March and December. Cattle and sheep are infected when they consume the infectious stage of the parasite from low-lying, marshy pasture. The effects of liver fluke are referred to as fascioliasis, and include anaemia, weight loss and sub-mandibular oedema. Diarrhea is only an occasional consequence of liver fluke. Liver fluke is diagnosed by yellow-brown eggs in the faeces. They are not distinguishable from the eggs of *Fascioloides magna*, although the eggs of *F. magna* are very rarely passed in sheep, goats or cattle.

A serious consequence of the liver damage caused by fascioliasis is that latent *Clostridium novyi* spores can be activated by the low oxygen conditions in the damaged tracts the parasite forms in the liver - this can lead to "black disease", caused by *Clostridium novyi* type B or immune-mediated haemolytic anaemia (IMHA) leading to haemoglobinuria caused by *Clostridium novyi* type D.

Treatment



Slide showing its internal organs

The drug of choice in the treatment of fasciolosis is triclabendazole, a member of the benzimidazole family of anthelmintics. The drug works by preventing the polymerization of the molecule tubulin into the cytoskeletal structures, microtubules. However, resistance of *F. hepatica* to triclabendazole has already been recorded in Australia and Ireland. Artemether has been shown to be effective in a rat model of fascioliasis.

Chapter 10

Gongylonema Pulchrum

Gongylonema pulchrum	
Scientific classification	
Kingdom:	Animalia
Phylum:	Nematoda
Class:	Secernentea
Order:	Spirurida
Family:	Gongylonematidae
Genus:	Gongylonema
Species:	***Gonglylonema pulchrum***

Gonglylonema pulchrum is the only parasite of the genus Gongylonema capable of infecting humans. Gongylonema pulchrum infections are due to humans acting as accidental hosts for the parasite. There are seven genera of spirudia nematodes that infect human hosts accidentally: gnoathostoma, thelazia, gonyglonema, physaloptera, spirocerca, rictularia. The g. pulchrum parasite is a nematode worm of the order Spirudia. It is a relatively thin nematode, and like other worms within its class, it has no circulatory or respiratory system. In the Gongylonema family, it is the only species that can infect humans. Most other gongylonema species infect birds and mammals: there are 25 species found in mammals and 10 species found in birds.

This parasite is multi-cellular, and capable of movement, and it's phylum makes up a majority of the animalia kingdom, and 18% of it is parasitic. They have numerous rear mucosal projections, which assumedly assist propulsion through the thin layer of skin on the inside of the human host's mouth. They also have an excretory system possessing lateral canals. this parasite eats epithelial cells. Also, very often the canals are a place of inflammation, with accumulation of exudates in them. Gongylonema also swallows these exudates. They have no circulatory or respiratory system.

History of Discovery

Gongylonema pulchrum was first named and presented with its own species by Molin in 1857. The first reported case was in 1850 by Dr. Joseph Leidy, when he identified a worm "obtained from the mouth of a child" from the Philadelphia Academy. He originally described it as Filariae hominis oris, and initially considered the worm was a guinea worm (Dracunculiasis medeninsis), but because of the unique location of the worm (buccal cavity), and the relatively short size compared to the guinea worm, the hypothesis was disregarded. There have only been around 50 reported human cases of g. pulchrum world wide since 1864, and these infections have been widespread and globally ubiquitous. G. pulchrum infections have been notoriously and historically hard to diagnose due to symptom complaints by patients- this will be touched on later. Also, morphological diagnosis of the parasite is also somewhat complicated because of the variable size of adult worms, and the tendency of the worm to be different lengths depending on what host the worm is recovered from. This will also be discussed in the morphology section.

Transmission

Transmission to humans is due mostly to unsanitary conditions and the ingestion of infected coprophagous insects, mostly dung beetles and cockroaches. Beyond direct ingestion of infected intermediate hosts (insects), foods can become contaminated if unsanitary conditions pervade in the production of the food- coprophagous insects are found in the food, or in the production chain. Also, contaminated water sources, again with the intermediate hosts or the infective third stage larva, can lead to transmission to humans. The infection usually occurs when someone drinks contaminated water, or consumes an infected beetle. The buccal mucosa, which is the ideal environment for the parasite is the mucous membrane of the inside of the cheek. It is non-keratinized stratified squamous epithelium, and is continuous with the mucosae of the soft palate, under surface of tongue and the floor of the mouth.

Reservoir and Vectors

Gongylonema pulchrum, along with most other gongylonema nematodes, has a broad natural host range. This includes hedgehogs, cattle, dogs, cats, ruminants, rabbits, and skunks. The vector and intermediate host for gongylonema pulchrum infections are coprophagous insects (dung beetles and cockroaches).

Incubation

In humans, there can be an up to six week incubation period for worm development and symptoms may not appear until the second molting of the worm, in which the young adult worms begin migration to from the esophagus to the buccal and oral palate tissue. It is this movement through the mucosa of the mouth and lips that causes patients to complain of symptoms. Gongylonema pulchrum burrows in the mucosal lining of the esophagus and other parts of the buccal cavity. There the 14 cm (5.5 in) females lay their thick shelled eggs containing first stage larvae. The larvae all possess a cephalic hook and rows of tiny spines around a blunt anterior end, so when they hatch they may further infest their hosts.

Morphology

The morphology of the worm is as follows, from a 2000 Veterinary Medicine study: "The anterior end in both sexes was covered by numerous cuticular platelets. There was a pair of lateral cervical papillae. The buccal opening was small and extended in the dorsoventral direction. Around the mouth a cuticular elevation enclosed the labia, and eight papillae were located laterodorsally and lateroventrally. Two large lateral amphids were seen. On the lateral sides of the female's tail, phasmidal apertures were observed. The caudal end of the male was asymmetrically alate and bore 10 pairs of papillae and two phasmidal apertures." The average length for male worms is 29.1 mm (1.15 in), while the average length for adult females is 58.7 mm (2.31 in). The worm is highly mobile, as observed in patients' mouths and as evidenced by the morphological design of the worm.

Life cycle

In humans, the hypothesized lifecycle is as follows: Ingestion of contaminated food, water, or infected dung beetle. Infects upper esophagus, moves around and lays eggs in buccal cavity of human host, ingested eggs locate near esophagus, develop and mature into adult worms after two subsequent molting stages, migrate into buccal cavity, no eggs are ever found in human feces, which strengthens the assumption that humans are solely incidental, accidental, and dead end hosts for the gongylonema pulchrum parasite life cycle.

The pulchrum parasite has also been studied in vivo in rabbits. The lifecycle is as follows:

Infective third stage larva from naturally infected dung beetles (intermediate hosts and vectors), were orally given to rabbits. The larvae entered the upper gastrointestinal tract of the rabbits (esophagus and upper stomach), and then migrated upward into the buccal cavity- pharyngeal mucosa and tongue. A third molt took place 11 days after primary infection, and the final molt took place at 36 days after primary infection. Worms reached sexual maturity at about 8 weeks, and were found mostly in the esophagus of the rabbit. 72–81 days post primary infection, embryonated eggs appeared in the feces of the rabbits.

Symptoms

With initial infection, some patients have reported remembering a mild fever and flu-like symptoms about a month previous to extraction or identification of worm. The most common symptom is the complaint of sensation of a worm moving around the mouth, near the lips, and in the soft palate area. This movement is normally engendered by immature adult female worms. Symptoms, once noted, may continue from a month to a year if the worm is not surgically extracted. Eosinophilia is noted in some patients. Gongylonemiasis is the affliction caused by this parasite, which is simply protracted discomfort or sensation of movement in the buccal, oral or gingival areas associated with a sensation of foreign body. Subjects commonly pull worms from their gums, tongue, lips, and inner cheeks after days and even weeks of reported discomfort. In animals, this parasite quickly spreads down the esophagus, and into the upper digestive and respiratory tracts, making it more often than not, fatal. For humans, this parasite never makes it further than the oral cavity, and is often surgically or manually extracted.

Diagnosis

There is a danger of misdiagnosing infections of G. Pulchrum as delusional parasitosis. Diagnosis is often made by visible recognition of the worm moving through the tissue of the buccal cavity by either patient or doctor. Also, recovery of worm from patient is also a diagnostic technique. Microscopic identification of worm removed from patient's mouth or tissue is another diagnostic technique for determining the parasite infection type.

Treatment

Treatment for infections with gongylonema pulchrum include surgical/manual extraction and dosage of albendazole (400 mg BID (twice daily) X 21 days). Follow up measures include periodic of checks of buccal cavity and esophagus to ensure parasite infection has cleared.

Epidemiology

Infections of gongylonema pulchrum are not a huge public health concern. There have only been 50 recorded infections world wide since the first reported case in 1850. The infections of g. pulchrum have been widespread, and countries reporting human infections include the United States, Germany, Iran, Japan, Laos, Morocco, China, Italy, New Zealand, and Egypt, among others. Control measures for reducing infections include making sure vector and larval contamination of food and water sources does not occur-this could be included in basic sanitary practices. Another control measure is ensuring children and adults do not accidentally or purposefully ingest infected dung beetles and other coprophagous insects.

Individual Case Studies

In 1996, the first reported case of gongylonema pulchrum infection was reported in Japan. A 34-year old male complaining of irritable stomatitis on his lower lip went in to see his doctor, but the pain subsided spontaneously. However, it reoccurred several times in the next few months. When he went in to his doctor after one of these episodes, a thread like organism was seen protruding from his ulcer. The patient also had eosinophilia, but the ulcer healed with no scar once the organism was removed. The organism was identified as a female gongylonema pulchrum worm, and the patient needed no further treatment.

How the patient contracted the worm is still unknown. He didn't report eating any abnormal foods, nor had he traveled outside of Japan in the past few years. He also did not report drinking any water from possibly infected wells. It is possible that he ate food that had been contaminated in an endemic country and shipped to Japan. With the globalized food market now present, this is not out of the realm of possibility, and should be considered as a possible means of transmission into countries that have no previous history of g. pulchrum infection.

In 1999, a 41 year old female resident of New York City went in to her doctor complaining of the sensation of something moving in her mouth. She said she had had the feeling for the duration of one year. Supposedly, she had removed worms from her mouth on two separate occasions- one from her lip, and one from her gums. She submitted one of the specimens for microscopic identification, and it was found to be an adult female gongylonema pulchrum worm. She traveled frequently to visit relatives in Mississippi, so it is unknown whether she contracted the worm in New York or in the south. This was the first reported case of gongylonema in the United States since 1963.

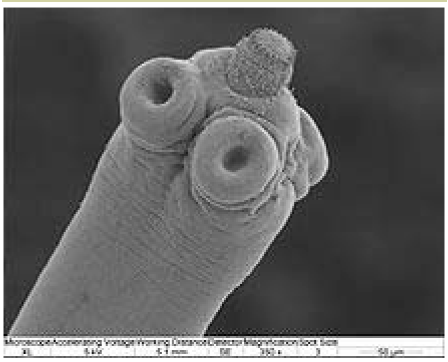
Also in 1999, a 38-year old woman of Cambridge, Massachusetts sought medical attention for the visible identification of a "migrating mass" in her cheek mucosa. Six months earlier, she had noted an irregular patch of mucosa on her cheek, but thought nothing of it. Previously in the year, she'd traveled to Mexico, Guatemala, and France. She didn't report ingesting any beetles, but she did eat raw foods when vacationing in Mexico. She described the foods as "raw, crunchy, and saladlike". Approximately 12 hours after eating the food, she and five other individuals she was traveling with had an acute attack of nausea, vomiting, and dizziness. The symptoms seemed to resolve themselves with no need of further treatment. A small female gongylonema worm was surgically removed from her cheek mucosa under local anesthesia, and follow up treatment included albendazole two times daily for three days. This was the eleventh reported case of g. pulchrum infection in the United States. Most cases reported in the US are reported from the southeastern part of the country.

There was a 1916 infection reported in a 16 year old girl from Mississippi. She presented with gastrointestinal pain, vomiting and a low fever (101.5 °F (38.6 °C)). She complained of a sensation of a worm moving around her lower lip, but was disregarded by her physician. As she continued to complain, the physician examined her mouth, and discovered the outline of a worm. He extracted the worm with a sewing needle, and the child's complaints stopped and she appeared to have no further symptoms of parasite infection.

Chapter 11

Hymenolepis Microstoma

Hymenolepis microstoma



Scientific classification

Kingdom:	Animalia
Phylum:	Platyhelminthes
Class:	Cestoda
Order:	Cyclophyllidea
Family:	Hymenolepididae
Genus:	*Hymenolepis*
Species:	***H. microstoma***

Binomial name

Hymenolepis microstoma
Dujardin, 1845

Synonyms

- *Vampirolepis microstoma* (Dujardin, 1854) Spasskii, 1954
- *Rodentolepis microstoma* (Dujardin, 1854) Spasskii, 1954
- *Taenia microstoma* Dujardin, 1845
- *Taenia brachydera*

Diesing, 1854

- *Taenia murisdecumani*
Diesing, 1863

Hymenolepis microstoma, also known as the **rodent tapeworm**, is an intestinal dwelling parasite. Adult worms live in the bile duct and small intestines of mice and rats, and larvae metamorphose in the haemocoel of beetles. It belongs to the genus *Hymenolepis*; tapeworms that cause hymenolepiasis. *H. microstoma* is prevalent in rodents worldwide, but rarely infects humans.

Ecology

Hymenolepis microstoma is an obligate parasite. Adults live in the bile duct and small intestine of rodents such as mice (*Mus musculus*), and larvae infect grain beetles such as *Tribolium* spp., in which they metamorphose from larvae into juvenile worms. Worms vary from 4 to 30cm in length, depending on the age and number of worms within the host. Adults have completely lost their mouth and intestine. Instead they use their skin (tegument) to absorb nutrients directly from the host gut.

Hymenolepis species and other tapeworms often exhibit a 'crowding effect' in which the total biomass of the worms stays more or less constant, regardless of the intensity of infection. Thus low intensity infections result in larger worms and high intensity infections produce smaller worms. Under laboratory conditions, *H. microstoma* adult infections in mice are typically limited to approximately 12 worms.

Life cycle

The cycle begins as arthropods become intermediate hosts by ingesting the parasite eggs. Oncospheral larvae are released from the eggs and use hooks and secreted enzymes to penetrate the gut of the beetles and enter the haemocoel. In the haemocoel the larvae undergo complete cellular reorganization (i.e. metamorphosis), transforming into cysticercoid larvae in approximately 7-10 days. The larvae can remain in the cysticercoid stage in the beetle as long as the lifespan of the adult beetle (up to 3 years), although any age-related decrease in viability has not been studied. When ingested, the eggs develop into cysticercoids. Rodents can become infected when they eat arthropods, such as flour beetles (*Tribolium* ssp). Humans, especially children, can ingest the arthropods as well and therefore become infected via the same mechanism. Rodents, especially rats, are definitive hosts and natural reservoirs of *H. microstoma*. As the definitive host (rats) eats an infected arthropod, cysticercoids present in the body cavity transform into the adult worm. Juvenile worms establish in the bile duct of mice after approximately 3 days movement within the upper gastrointestinal tract. Once established in the bile duct, the worms then mature sexually and begin producing eggs within approximately 1 week. Eggs are released with mouse faeces and thus dispersal is passive - through the movement and defecation of mice. Adult worm infections in mice held under laboratory conditions persist for 6-12 months.

Worms reproduce sexually via the cross fertilization of segments, each of which contains a complete complement of male and female reproductive organs (hermaphroditic). Shelled embryos develop in the ovaries through spiral cleavage, to become infective larvae with 3 pairs of hooks. When ingested by beetles, these larvae use their hooks and secretory glands to penetrate the gut of the beetle and enter the haemocoel where they undergo complete metamorphosis into cysticercoid larvae, replete with an adult scolex, ready for establishment in the final host.

Cytology

Like all flatworms (phylum Platyhelminthes), *H. microstoma* maintains totipotent stem cells (called *neoblasts* in flatworms) throughout its life cycle. These are located in the neck region of the adult worms and are responsible for the continual production of new organs during the process of strobilation (segment formation). Neoblasts divide in the neck region and become incorporated into new segments where they eventually differentiate into the reproductive organs and other elements of the body. The diploid chromosome number of *H. microstoma* is 12 and the total genome size has been estimated by the Sanger Institute to be 1.4 megabases (with GC-content of ~35%). These values are similar to the genomes of the fox tapeworm *Echinococcus multilocularis* and the pig tapeworm *Taenia solium*. All three species belonging to the tapeworm order Cyclophyllidea. Genome sizes outside of this order are presently unknown.

Evolution

The parasitic flatworms, which includes tapeworms, flukes and monogeneans, evolved from a single major lineage of free-living flatworm ancestors. The switch from a free-living to a parasitic lifestyle in the common ancestor of the parasitic flatworms involved a fundamental change in their tegument, which is found in all contemporary groups. Early-branching tapeworm groups are found in bony (e.g. teleost) and cartilaginous fishes (e.g. sharks and rays) and have entirely aquatic life cycles involving arthropod (e.g. copepods) first intermediate hosts and vertebrate (fish) final hosts. Tetrapod hosts (including mice and humans) were acquired later in tapeworm evolution and eventually part-aquatic life cycles led to the evolution of fully terrestrial life cycles, albeit still involving an arthropod intermediate host and a vertebrate definitive host. *Hymenolepis microstoma* is a member of the Cyclophyllidea, one of the youngest and most species-rich group of tapeworms.

Research impact

Most of our understanding of the basic biology of tapeworms, such as their anatomy, physiology and ultrastructure, stems from work on this genus. Species in the genus *Hymenolepis* (e.g. *H. diminuta*, *H. microstoma*, *H. nana*) have been maintained as laboratory models for studying tapeworm biology since the 1950s. They can be readily maintained *in vivo* in rodent and beetle hosts, which makes them useful for teaching and research purposes. They can also be grown in culture (*in vitro*), giving easy manipulation of the life cycle.

Infection and treatment

H. microstoma primarily infects rodents, and is only very rarely found in humans. Human *H. microstoma* infection is often asymptomatic, but abdominal pain, irritability, itching, and eosinophilia are among the existing symptoms in a few of the reported cases. Since data regarding praziquantel treatment of *H. microstoma* is sparse, scientists have recommended that every case and treatment of *H. microstoma* be reported for development of protocols and parasitological purposes.

Chapter 12

Moniezia Expansa and Nanophyetus Salmincola

Moniezia expansa

Moniezia expansa	
Scientific classification	
Kingdom:	Animalia
Phylum:	Platyhelminthes
Class:	Cestoda
Order:	Cyclophyllidea
Family:	Anoplocephalidae
Genus:	*Moniezia*
Species:	***M. expansa***
Binomial name	
Moniezia expansa Rudolphi, 1810	

Moniezia expansa is commonly known as **sheep tapeworm** or **double-pored ruminant tapeworm**. It is a large tapeworm inhabiting the small intestines of ruminants such as sheep, goats and cattle. It has been reported from Peru that pigs are also infected. There is an unusual report of human infection in an Egyptian. It is characterized by unarmed scolex (i.e., hooks and rostellum are absent), presence of two sets of reproductive systems in each proglottid, and each proglottid being very short but very broad.

Structure

M. expansa has a typical cestode body, consisting of the anterior scolex, followed by the neck and a highly extended body proper, the strobilus. It is an extremely long tapeworm, and can reach an enormous length up to 6–10 m. The scolex bears four large suckers, which are the holdfast organs to the host. There are no rostellum and rostellar hooks, and the suckers are devoid of spines. The boundary between the proglottids are studded with a row of interproglottid glands, which are yet undefined in terms of function. The tapeworm, being monecious, contains both male and female reproductive organs in an individual. Thus each proglottid is a complete reproductive unit. Moreover, one defining feature of the genus is that there are two sets of reproductive organs situated at lateral sides with the associated cirrus pouches and genital pores in each proglottid. The testes are numerous.

Life cycle

The complete life cycle requires two hosts, ruminants as definitive hosts, and oribatid mites as intermediate hosts. Eggs are passed out from the intestine of the ruminant host along the gravid proglottids in the feces into the soil. The eggs are eaten by soil mites. Eggs must reach the gut of mite hosts within 1 day of release otherwise they are desiccated. However, chances of development is very good as soil mites can be so numerous on a pasture that even if only 3% are infected (with 4-13 cysticercoids each), a grazing ruminant may ingest over 2,000 cysticercoids per kilogram of grass. Once inside the intestine of mites, the eggs hatch and the oncospheres penetrate into the haemocoel and develops to the cysticercoid stage. This stage may take up to 4 months. When the infected mite is eaten by the grazing ruminants, mature cysticercoids are digested out of the mite, and develop into mature tapeworms in the small intestine within 5–6 weeks.

Pathogenicity

M. expansa infections are generally harmless and asymptomatic, even when the tapeworms are present in large numbers in young lambs. However heavy infection may cause intestinal obstruction, diarrhea and weight loss.

Diagnosis and treatment

Diagnosis is done by analysis stool sample in which eggs can be detected, or often observation of the gravid proglottids in feces and anus. Niclosamide is most often used. Praziquantel is also 99–100% effective while albendazole is 19-75% effective; and praziquantel + levamisole combination is very effective in reducing worm burden and improvement of weight.

Nanophyetus salmincola

Nanophyetus salmincola	
Scientific classification	
Kingdom:	Animalia
Subkingdom:	Eumetazoa
(unranked):	Bilateria
Superphylum:	Platyzoa
Phylum:	Platyhelminthes
Class:	Trematoda
Genus:	*Nanophyetus*
Species:	***N. salmincola***
Binomial name	
Nanophyetus salmincola	

An egg of *Nanophyetus salmincola*

Nanophyetus salmincola may be the most common trematode endemic to the United States. In particular, the parasite is a food-borne intestinal trematode prevalent in the coast of the Pacific Northwest.

The life cycle of the *N. salmincola* requires three hosts. The first intermediate host is an *Oxytrema silicula* stream snail. The second intermediate host is a salmonid fish, though some non-salmonid fishes also play a role. Lastly, the definitive host is most commonly a canid, though many other mammals are also definitive hosts, including humans. Transmission of *N. salmincola* to the definitive host occurs upon ingestion of parasite-infected fish.

The parasite is most known for its association with “salmon poisoning disease", which, left untreated, proves to be fatal to dogs and other canids. However, canids are affected by the *Neorickettsia helminthoeca* bacteria, for which *N. salmincola* acts as a vector, and not by the parasite itself.

Very few known cases of naturally acquired human infection with *N. salmincola* are found in the literature, though it is likely that many cases are unreported, since most people are asymptomatic, or symptomatic with non-specific symptoms like gastrointestinal discomfort. Disease caused by *N. salmincola*, or nanophyetiasis, is easily preventable by thoroughly cooking fish before consumption. There are no known cases of human infection by the *Rickettsia* bacteria carried by *N. salmincola*.

A subspecific parasite, *Nanophyetus schikhobalowi*, is endemic to Siberia, where human cases of nanophyetiasis have been reported in scientific literature since 1931.

Agent (classification and taxonomy)

Kingdom: Animalia Phylum: Platyhelminthes Class: Trematoda Order: Digenea Family: Troglotrematidae Genus: Nanophyetus Species: Salmincola

Synonyms

Nanophyetus salmincola (Chapin), *Troglotrema salmincola*, and *Nanophyetus schikhobalowi* (Russian form)

History of Discovery

The first record of salmon poisoning disease (SPD) was reported in northwestern Oregon in 1814 when a writer for Henry's Astoria Journal noted the death of dogs after consumption of raw salmon., At first, investigators believed that SPD was caused by poisonous blood in the ingested fish. In 1911, small white cysts were observed in the kidneys of disease-causing salmon and trout, but the cysts were mistakenly identified as amebae. Small trematodes in the intestines of dogs that died after eating infected salmon were finally found in 1925 and the cysts present in the salmon were correctly identified as intermediate stages of the trematode. In an experimental follow-up study, researchers showed that the small intestinal parasite did in fact cause SPD in dogs, and that the cysts did develop into the adult worm found in the intestine.

The trematode was first named by Chapin as *Nanophyes salmincola* in 1926, as a member of the family Heterophyidae. Upon further examination of the morphology, Chapin reassigned the trematode to the family Troglotrematidae and renamed the parasite *Nanophyetus salmincola*, since *Nanophyes* was already taken. Discussions regarding the correctness of classification of the parasite continued as the trematode received further scientific attention and its morphology and behavior was further scrutinized. Ultimately, *Nanophyetus salmincola* was agreed upon, though *Troglotrema salmincola* remains a synonym.

In 1931, Skrjabin and Podjapolskaja describe a similar parasite, *Nanophyetus schikhobalowi*, which was endemic to East Siberia. Argument regarding whether or not *N. schikhobalowi* and *N. salmincola* were the same or different species recurred until 1966 when the two were granted subspecific status in order to reflect their biological and geographic differences, but little significant morphological differences.,, Since its discovery, *N. schikhobalowi* has been known to naturally infect humans and research reveals surveys indicating rates of infection in endemic Siberian villages of up to 98%.

In contrast, *N. salmincola* was not recognized to be a source of an infection until a researcher purposefully infected himself in a scientific experiment in 1958. Besides Philip experimentally infecting himself with the North American *N. salmincola*, the first naturally acquired human intestinal infection cases were observed between September 1974 and October 1985. The study revealed 10 patients who presented with positive *N. salmincola* stool samples and either gastrointestinal complaints or otherwise unexplainable peripheral blood eosinophilia. 7 patients recalled ingestion of undercooked or raw fish. Of those who were not given effective treatment, symptoms and/or eggs in stools persisted for 2 or more months before spontaneously resolving. It was hypothesized that the movement, attachment, and irritation of the adult worms in the small intestine mucosa was the likely cause of gastrointestinal symptoms and peripheral eosinophilia.

Two years after the first 10 cases of human infection with *N. salmincola* were reported in 1987, Fritsche et al. reported ten additional cases of human nanophyetiasis. Five presented with gastrointestinal complaints and the other five had unexplained peripheral eosinophilia. Nine out of ten recalled eating inadequately cooked fish. This time, praziquantel was the effective treatment of choice.

In 1990, the first case of human infection with *N. salmincola* without ingestion of raw or undercooked contaminated fish was reported. A man was infected through hand contamination while handling highly infected, fresh-killed, coho salmon. A diagnosis of nanophyetiasis was made based on gastrointestinal discomfort, peripheral blood esoinophilia and a positive stool sample. Treatment with praziquantel proved to be effective again.

None of the human cases of infection with either the North American or Siberian subspecies reveal infection by the *Neorickettsia helminthoeca* carried within the trematode, which was discovered in 1950. Infection by rickettsia helps to explain the more fatal outcome afflicting canids.

Clinical Presentation in Humans

Upon infection with *N. salmincola*, humans are normally asymptomatic. If symptoms are present, they are usually non-specific and mistaken for indication of other gastrointestinal problems. Symptoms include “diarrhea, unexplained peripheral blood eosinophilia, abdominal discomfort, nausea and vomiting, weight loss, and fatigue.” Eggs of *N. salmincola* appear in stools approximately one week after ingestion of infected fish.

Pathology in Dogs

Nanophysiasis in dogs is much more serious than in humans. Scientists noticed almost 200 years ago that dogs that consumed raw fish sometimes died rather quickly. This "salmon poisoning", while associated with the trematode *Nanophyetus salmincola* is not caused by the worm. The sickness is caused by *Neorickettsia helminthoeca*, a rickettsial bacteria that uses the *N. salmincola* as a host. Although only canines are susceptible to the disease raccoons show a raised temperature and lymphatic infection after being infected by the rickettsia, but both soon subside. The incubation period in dogs is 5–7 days, although it may take as long as 33 days. After onset, there is a sharp fever coupled with anorexia, vomiting and dysentery. The rickettsia attacks the canine's lymph system causing enlarging and eventually hemorrhaging many of the lymphnodes. The disease can spread to other tissues such as leucocytes. Death occurs 10–14 days after signs first appear.

Transmission

Nanophyetus salmincola is transmitted most commonly by the ingestion of raw, undercooked, or smoked salmon or steelhead trout. Usually this is meant to be ingestion of the muscle of the fish but there have been cases reported in which the suspected agent of transmission was Steelhead roe. Researchers hypothesize, in fish with especially high worm burdens, that the N. salmincola may migrate to many of the fishes tissues, not just the muscle tissue. In a case in 1990 Nanophyetiasis was diagnosed in an individual that is thought to have acquired the disease by simple handling of fresh-killed salmon. The infected individual, ironically, was a researcher studying N.salmincola in juvenile Coho salmon, had inadvertently initiated the infection by hand-to-mouth contact during the 3 month long study.

Reservoir

The reservoirs for *N. salmincola* are raccoons, mink, and skunks. Reservoirs are organisms that harbor parasites within themselves without suffering any signs of pathology, and spreading the parasites through their natural behavior. For example, raccoons naturally spread *N. salmincola* because they frequently eat fish and defecate parasitic eggs in or near the water, where subsequent larval stages can continue their life cycle.

Vector

Vectors are organisms that transmit parasites from one host to another. *Oxytrema silicula* stream snails are biological vectors for a larval stage of *N. salmincola*. Salmonid and some non-salmonid fish are vectors of the metacercariae of *N. salmincola.* Both fresh and ocean water fish can be parasitic vectors. Fish that act as second intermediate hosts are different species of the families Salmonidae, Cottidae, and Cyprinidae. Among the thirty-four natural and experimental secondary hosts found in scientific literature are the coastal cutthroat trout, rainbow trout, coho salmon, chum salmon, and kokanee salmon. More infection occurs in salmonid fish, rather than non-salmonid fish. In particular, salmonid fish of the genera Salmo, Oncorhynchus, and Salvelinus play a significant role in the *N. salmincola life cycle*. The parasite itself is a vector for *Neorickettsia helminthoeca*.

Definitive Hosts

Definitive hosts include fish-eating birds and mammals. The most common definitive hosts are the domestic dog, cat, and red fox. Humans are also definitive hosts for *N. salmincola*. A long list of experimental definitive hosts include the hamster and wood rat. Interestingly, an experimental study failed to infect two white rats and two white mice. Trematodes exist along the whole length of the small intestine in smaller animals like hamsters, while they exist only in the upper end of the small intestine in larger animals like dogs.

Incubation Period

After ingestion of fish infected with *N. salmincola*, it takes about 1 week for symptoms to occur, namely for eggs to be detected in the stool.

Morphology

Eggs of *N. salmincola* are light brown, ovoid, and operculate at one end, with a small blunt projection at the other end. They measure 0.087 mm to 0.097 mm by 0.038 mm to 0.055 mm. There are normally 5 to 16 eggs in the uterus, and their heaviness allows them to sink rapidly in water.

N. salmincola is a digenic trematode, which means that it is an unsegmented worm that is flattened dorsoventrally. Adult worms alternate shape from “a sphere to a long blunt rod.” The worms are 0.8 to 1.1mm long and 0.3 to 0.5 mm wide and are hermaphroditic, having both male and female reproductive organs in the same organism. The two large oval testes are 0.2 to 0.3 mm long and the round ovary is 0.07 to 0.11 mm in diameter. *N. salmincola* has a prominent cirrus pouch, or hollow organ surrounding the male copulatory organ, but no seminal vesicle. True to its character as a trematode, it has an oral sucker 0.15 to 0.18 mm in diameter, and a ventral sucker 0.12 to 0.13 mm in diameter. The oral and ventral suckers are used to grasp and crawl actively about the intestinal tissue of its host, though the worm leaves no extensive mechanical damage.

Life cycle

The adult lays eggs within the vertebrate host. The vertebrate passes out the eggs in its feces. The first larval stage, the miracidia, develop within the eggs, hatch, and swim away. The miracidia then penetrate the first intermediate host, the *Oxytrema silicula* stream snail. After further development in the stream snail, *N. salmincola* larva develop into rediae, which give rise to cercariae. The cercariae emerge from the snail and penetrate the second intermediate host, the salmonid (some non-salmonid) fish. The parasites develop into metacercaria and encyst within the kidneys, muscles, and fins of the salmonid fish. The parasites enter its final host, including canids and humans, upon ingestion of the infected fish, and develop into adult worms that produce eggs to be passed in the host's feces.

Detailed information regarding the life cycle stages

Eggs and miracidia: The eggs passed in the feces are unembryonated. Experimental studies demonstrate that eggs collected in room water temperature require 75 days to 200 days to hatch., The hatching rate of miracidia from eggs increases with decreasing temperatures, and egg mortality increases with increasing temperatures. Fully developed miracidia within the eggs contract and elongate repeatedly, and newly emerged miracidia swim in "characteristic, long graceful curves." Interestingly, the miracidia seem to have no attraction to host snails, bumping into the snails without attempting to penetrate and infect them.

Rediae: The rediae are the second larval stage of the trematode life cycle, that develops from the miracidum and contains germ cells that develop into cercariae. The rediae are found in the second intermediate host, the snail. Rediae can range from 0.45 mm to 3 mm, and the larger rediae can contain up to 76 cercariae. Rediae and cercariae are found in all tissues of the host snail, but primarily in the gonads and the digestive gland., Rediae destroy the gonads, invade the hepatopancreas, damage it by 1) increased pressure from rapid growth, 2) active ingestion by the parasites, and 3) the disposition of parasitic wastes. Furthermore, parasites take up glycogen and lipids from the hepatopancreas.

Cercariae: The cercariae measure 0.31 mm to 0.47 mm by 0.03 mm to 0.15 mm and live up to 48 hours in water. They have a tendency to infect snails that are at least 2.5 cm in length, though smaller snails have also been observed to shed cercariae. Cercariae shed intermittently by the thousands, entering the mantle cavity of the snail, and drifting out with the "exhalent water current on the right side of the snail's head.", Cercariae from snails in brackish water of a low salinity were found to survive longer than snails in freshwater.20

Once cercariae are shed from the snail, it contracts repeatedly until it contacts a fish and penetrates under its skin within 30 seconds to 2 minutes. The cercariae penetrate further into the renal portal blood system, into the kidney and deeper tissues into the base of the tail. Penetrations sights are easily visible, as the skin, fins, and tails of the fish appear to be heavily eroded and damaged. Cercariae can also indirectly infect the fish, if the fish eat the cercariae orally.

Metacercariae: The cercariae lose their tails in the act of penetration and encyst as metacercariae in almost any tissue of the salmonid fish. The new cyst wall is thin, transparent, and easily ruptured. If the cyst wall breaks, the metacercariae crawl out and re-encyst a few hours later in a tougher, larger cyst wall. While cysts can be found in all tissues of the fish, most encystment occurs in the kidneys and body muscles of the salmonid fish, and in the gills and fins of the non-salmonid fish. Cercariae penetrate less deeply in non-salmonid fish than in salmonid fish. Infected fish experience a decrease in their swimming activity and loss of equilibrium, and it is not uncommon for fish to have as many as 1000 to 2000 metacercariae in its tissues.,

Importantly, metacercariae can be destroyed either by cooking or freezing infected fish.

Snail: The *Oxytrema silicula* host snail is prevalent in coast streams and prefers large rocks, bridges, old planks, and debris on stream bed bottoms. It rarely migrates into shallow water. The infection of snails is high in comparison to the number of cercariae it sheds, since larval development continues slowly over a long period of time. Evidence of mixed infection varied between studies, but snails with large numbers of N. salmincola were not parasitized by other trematodes. It was also found that monthly incidences of infection in snails ranged from 9-52% after examining over 3000 snails every month for 10 months, and that mature cercariae infected snails in a seasonal manner. Mature cercariae were more likely to infect snails in late April to November.

Neorickettsia helmintheoca *Neorickettsia helmintheoca* is the etiological agent for salmon poisoning disease, found to be present in all stages of the trematode. It is 0.3 micrometers in size and a purple Giemsa stain indicates that it is Gram negative. Thus far, only canids are susceptible to disease by rickettsia and it is still uncertain how the rickettsia leave the trematode vector and reaches the host tissues. Experiments do show that the bacteria lead to necrosis of lymph follicles, ulceration, and severe hemorrhage in its host.

Diagnostic Tests

1. History of eating raw fish
2. Examination of feces for eggs of *N. salmincola*

   Because only a few eggs are contained within each adult worm, patients with light infections are likely to have negative stool tests. Using trichrome stained preparations rather than formalin-ethyl acetate concentrates was more sensitive to identify cases.

Management and Therapy

Praziquantel, 20 mg/kg body weight, three times a day. Praziquantel causes immobilized contraction of the worm, such that it can no longer grasp the intestinal walls, and can be eliminated from the body Three 2-g doses of niclosamide or two 50 mg/kg doses of bithionol have also proven to be effective when Praziquantel was either not available or treatment was refused. However, a single 2-g dose of niclosamine proved to be ineffective treatment therapy, as did 100 mg orally of mebendazole twice a day for three days. If diarrhea recurs, general supplements must also be provided in order to maintain electrolyte balance and meet nutritional requirements.

Epidemiology

Nanophyetus salmincola is limited to the geographic range of its intermediate hosts, primarily the US Pacific Northwest. Stream snails are found west of the Cascade Mountains in Oregon, north to the Olympic Peninsula in Washington, and in part of northern California. It is "the most common systemic trematode in the United States."

Public Health and Prevention Strategies

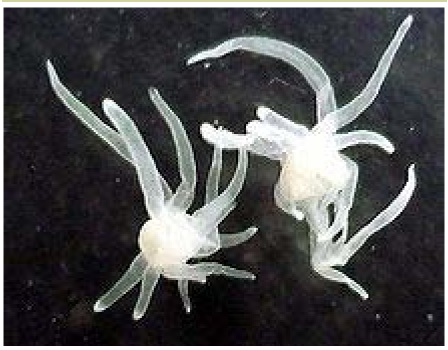
- Cook fish thoroughly
- Freeze fish for at least 24 hours
- Fish away from known snail endemic places (otherwise make especially sure to cook fish thoroughly)
- Take precautions when handling fish (prevent hand-to-mouth transmission of the metacercariae and be wary of hand contamination from heavily infected fish)
- Check fish periodically for signs of infection, such as cysts or sites of irritation from penetration of the cercarariae (pertinent to fishing companies, fish markets, and restaurants)
- Report sources of infected fish (bodies of water, fish companies, restaurants)
- Eat raw fish only from trusted sources, such as reputable restaurants
- Use molluscicides if can be practically used in smaller bodies of water
- Keep dogs away from streams, making sure they defecate away from snail habitats

Chapter 13

Polypodium Hydriforme and Ribeiroia

Polypodium hydriforme

Polypodium hydriforme	



Two specimens of free-living *Polypodium*

Scientific classification [e]	
Kingdom:	Animalia
Phylum:	Cnidaria
Subphylum:	Medusozoa
Class:	**Polypodiozoa** Raikova, 1994
Family:	**Polypodiidae**
Genus:	***Polypodium*** Ussov, 1885
Species:	***P. hydriforme***

Binomial name

Polypodium hydriforme
(Ussov, 1885)

Polypodium hydriforme is a species of a parasite attacking the eggs of sturgeon and similar fishes (Acipenseridae and Polyodontidae). It is one of few metazoans living inside the cells of other animals. It is also the only known intracellular cnidarian parasite.

Polypodium hydriforme is the only species in the genus ***Polypodium*** (monotypic genus). It is also the only species and genus within the whole family **Polypodiidae**.

Taxonomy

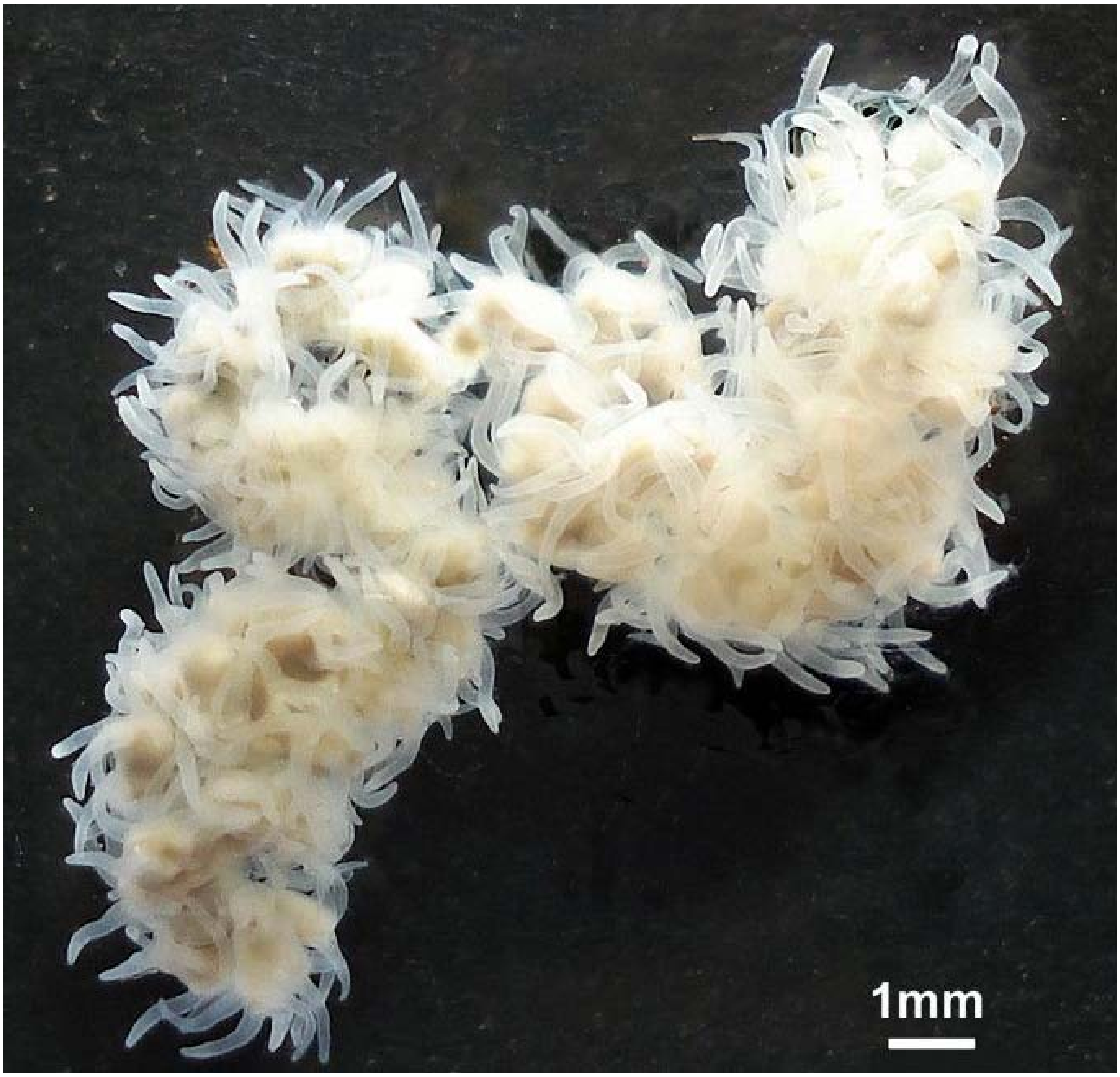
Unusual characteristics have led to much controversy regarding its phylogenetic position within metazoans.

Polypodium has traditionally been considered a cnidarian because it possesses nematocysts, the stinging structures characteristic of this phylum.

However, molecular phylogenetic studies using 18S rDNA sequence data have challenged this interpretation, and have shown that *Polypodium* is a close relative to myxozoans and together they share a closer affinity to bilaterians than cnidarians (Zrzavý & Hypša 2003). Due to the variable rates of 18S rDNA sequences, these results have been suggested to be an artifact of long branch attraction.

Evans et al. (2008) have performed phylogenetic analyses of metazoans with 18S and partial 28S rDNA sequences in a large dataset that includes *Polypodium* and a comprehensive sampling of cnidarian taxa. This support the placement of *Polypodium* within Cnidaria. This accords with the fact that *Polypodium* possesses nematocysts and a cnidarian-like body plan. The ML combined analysis places *Polypodium* as its own class **Polypodiozoa**. By contrast, the combined parsimony analysis and the ML analyses of 28S alone place *Polypodium* within the hydrozoan clade Leptothecata.

Description and life cycle



Free living stolon.

Polypodium hydriforme is an endocellular parasite with unusual life cycle, peculiar morphology, and high rates of DNA evolution. *Polypodium* spends most of its life inside the oocytes of acipenseriform fishes (sturgeons and paddlefish). Among its hosts include for example *Acipenser ruthenus*, *Polyodon spathula* and *Scaphirhynchus platorynchus*. During this time, *Polypodium* develops from a binucleate cell into an inside-out planuliform larva and then into an elongate inside-out stolon; the epidermal cell layer is located internal to the body and the gastrodermis is located externally. The embryo, larva and stolon are surrounded by a protective polyploid cell, which also functions in digestion. Just prior to host spawning, *Polypodium* everts to the normal position of cell layers, revealing tentacles scattered along the stolon. During eversion, the yolk of the host oocyte fills the gastral cavities of the parasite, supplying the future free-living stage with nutrients. Finally, upon emerging from the host egg in fresh water, the free-living stolon fragments into individual medusoid-like forms that go on to multiply by means of longitudinal fission, form sexual organs, and ultimately infect host fish with their gametophores.

Habitat

Habitat of *Polypodium hydriforme* is freshwater.

Although the fresh water habitat of *Polypodium* is unusual for cnidarians, it is not unheard of, especially within hydrozoans. For instance, the model organism *Hydra* and the jellyfish *Craspedacusta* are both exclusively fresh-water hydrozoans. *Hydra* and *Craspedacusta* are distantly related and they are not closely related to *Polypodium*. Thus, it appears that in the evolution of cnidarians, invasion to fresh-water habitats has happened at least three separate times.

Ribeiroia

Ribeiroia	
Scientific classification	
Kingdom:	Animalia
Subkingdom:	Eumetazoa
(unranked):	Bilateria
Superphylum:	Platyzoa
Phylum:	Platyhelminthes
Class:	Trematoda
Subclass:	Digenea
Order:	Echinostomida
Suborder:	Echinostomata
Family:	Psilostomatidae
Genus:	*Ribeiroia* Travassos, 1939
Species	
Ribeiroia congolensis	
Ribeiroia marini	
Ribeiroia ondatrae	

Ribeiroia is a group of trematode parasites (or flatworms) that sequentially infect freshwater snails in the family Planorbidae (ram's horn snails) as first intermediate hosts, fish and larval amphibians as second intermediate hosts, and birds and mammals as definitive hosts. In North America, infection by *Ribeiroia* has been linked to amphibians with limb malformations, such as extra limbs and digits, missing limbs and limb elements, and improperly formed limbs. The connection between parasitic infection and limb malformations has generated questions about (a) whether parasite-induced malformations in amphibians are increasing in frequency, severity, or geographic range, and (b) the consequences of such abnormalities for amphibian population conservation.

Ribeiroia *Taxonomy*

Ribeiroia is a genus of parasites in the class Trematoda, phylum Platyhelminthes. There are currently three recognized species of *Ribeiroia* and one subspecies: *R. ondatrae* in North America, *R. marini* in the Caribbean, *R. m. guadeloupensis* on the Caribbean island of Guadeloupe, and *R. congolensis* in Africa (Johnson et al. 2004). The trematode *Cercaria lileta* is also closely related to *Ribeiroia*, and molecular sequence data indicates that it may actually be a species of *Ribeiroia* (Johnson et al. 2004). All species of *Ribeiroia* share the distinctive morphological characteristic of esophageal diverticula (i.e., two short, dead-end branches that extend laterally from the esophagus). Other genera closely related to *Ribeiroia* include *Trifolium, Cathemasia*, and *Echinostoma* (Johnson et al. 2004).

Life cycle

Ribeiroia ondatrae has an indirect, complex life cycle (Beaver 1939; Basch and Sturrock 1969; Johnson et al. 2004). The adult worms live inside predatory birds or mammals (the definitive hosts), wherein they reproduce sexually if other worms are present. Mature adults release eggs into the host's intestinal tract, which are passed with the feces of the host into the aquatic environment. The eggs typically develop in 2-3 weeks (Beaver 1939; Basch and Sturrock 1969; Johnson et al. 2004), but the time varies depending on water temperature. Eggs hatch into miracidia, a ciliated free-living parasite stage. Miracidia infect the first intermediate host, ram's horn snails in the family Planorbidae, colonizing the snail's reproductive tissue and eventually forming rediae, a slow moving worm-like parasite stage. The rediae reproduce asexually, causing castration of the snail as they feed on host reproductive tissue. The infection becomes mature in about six weeks, when the rediae within the snail begin to release a second free-swimming stage called cercariae. The key identifying characteristic of *R. ondatrae* cercariae is the bifurcated esophagus (although note that this trait can occur in some closely related genera such as Trifolium). Cercariae infect amphibians or fish (the second intermediate hosts) wherein they encyst in either the limb buds or along the lateral line and scales of the head, body and gills, respectively. Encysted cercariae become metacercariae, a dormant parasite stage with a thin outer membrane. Metacercariae resemble cercariae without their tails and do not reproduce. The definitive hosts (birds and mammals) become infected when they consume a second intermediate host with encysted metacercariae. The life cycle is completed when the metacercariae emerge from their cyst and attach to the definitive host's intestinal tract, typically in the proventriculus of birds and the stomach of mammals (Johnson et al. 2004).

Ribeiroia Infection and Amphibian Malformations

Laboratory Studies

Experimental exposure to *Ribeiroia ondatrae* cercariae has been shown to cause limb malformations in various frog, toad and salamander species, including *Pseudacris regilla, Anaxyrus boreas, Lithobates pipiens, A. americanus, Ambystoma macrodactylum, L. clamitans* and *L. sylvatica* (Johnson et al. 1999; 2001; 2006; Stopper et al. 2002; Schotthoefer et al. 2003; Johnson and Hartson 2009). Cercariae appear to preferentially infect in and around the developing limb buds of larval amphibians, which can alter or inhibit limb development. The risk of malformation and mortality varies as a function of parasite exposure level, host developmental stage, and the amphibian species involved. As expected for macroparasitic infections, a dose-dependent relationship exists between cercariae exposure and pathology, particularly among larval amphibians at pre- or early-limb developmental stage (Schotthoefer et al. 2003). Cercarial penetration of host tissue involves proteolytic enzymes that may be one mechanism through which pathology occurs. While the exact mechanism through which limb development is altered is unknown, potential pathways include mechanical disturbance by invading parasites, release of a teratogenic chemical by parasites, or a combination of the two (Johnson et al. 1999; Stopper et al. 2002).

Interestingly, susceptibility to infection and the subsequent pathology differs among amphibian species. For example, gray treefrogs (*Hyla versicolor*) are largely resistant to infection, whereas toads (e.g. *A. americanus*) exhibit high frequencies of mortality and malformations following parasite exposure (Johnson and Hartson 2009). The types of limb malformations also vary among species and developmental stage of exposure. The most common errors in development associated with *Ribeiroia* exposure are skin webbings (cutaneous fusion), missing limbs and limb elements (ectro- and hemimelia), supernumerary limbs and digits (polymelia and polydactyly), and bony triangles. The factors determining variation in susceptibility among species remain poorly understood, but these observations suggest that the type of malformation alone is unlikely to be diagnostic of *Ribeiroia* exposure.

Field Studies

Ribeiroia infection has been linked to malformations in naturally occurring amphibian populations, especially in the western and midwestern USA (reviewed in Johnson et al. 2010). In a large-scale study in the western USA, both the presence and abundance of *Ribeiroia* infection predicted higher-than-baseline (e.g., >5%) frequencies of abnormalities in one or more amphibian species (Johnson et al. 2002). Limb malformations were observed in 9 species and ranged in frequency from <5% to nearly 90%. The role of *Ribeiroia* in explaining accounts of amphibian malformations in other regions is varied; *Ribeiroia* has been associated with malformation 'hotspots' in the midwestern and northeastern USA (Kiesecker 2002; Lannoo et al. 2003; Sutherland 2005; Johnson and Hartson 2009), but it was not detected in malformation surveys from

Vermont, Alaska, Bermuda, and Michigan (Gilliland and Muzzall 2002; Bacon et al. 2006; Skelly et al. 2007; Reeves et al. 2008).

Geographic Distribution

Ribeiroia ondatrae is widely distributed across the USA, including records from 22 species in 37 states (Johnson and McKenzie 2008). Most records of *Ribeiroia*-associated malformations have been reported from California, Oregon, Washington, Montana, Minnesota, Wisconsin, New Jersey and Pennsylvania. Less is known about the distribution of *Ribeiroia* in Canada and Central America. In amphibians, both the abundance of the parasite and the number of observations appear to correlate positively with major bird migratory flyways, with concentrations along the Pacific, Mississippi, and Atlantic flyways (Johnson et al. 2010). This observation suggests definitive (bird) host activity is an important determinant of parasite distribution at the continental scale, but this hypothesis has yet to be rigorously tested. *Ribeiroia ondatrae* is most often recovered from amphibians in lentic habitats, such as ponds, lakes, and wetlands, which support planorbid snails. Other species of Ribeiroia occur in the Caribbean and South America (*R. marini* and *R. marini guadeloupensis*) and in Africa (*R. congolensis*). However, whether these species infect amphibians or cause limb malformations has not been explored.

Evidence for Emergence

Dating back over 200 years, there have been reports of amphibian malformations suggesting that the general phenomenon is not a new occurrence in amphibian populations (Ouellet 2000). Despite the historical occurrence of amphibian malformations, the real question is whether such malformations are increasing in prevalence and severity in wild amphibian populations. Limited historical data on malformations coupled with the absence of baseline malformation data has complicated attempts to assess whether malformations are emerging. However, research over the past decade has demonstrated that malformations are emerging in some amphibian populations. For example, the background rate of malformations of northern leopard frogs in Minnesota increased from 0.4% in 1958-1963 to 2.5% in 1996-1997 (Hoppe 2000), with recently observed malformations also exhibiting a greater severity and diversity. Similarly, Johnson and Lunde (2005), in a comparison of recent (1990–2000) and historical (1899–1989) publications, found that recent reports document: 1) a wider range of more severe malformations, 2) a greater number of affected amphibian species, 3) a larger number of affected sites, and 4) a higher frequency range of affected individuals at reported sites. For both of these studies, however, the mechanisms driving the apparent increase in malformations were not identified. In a more rigorous study, Johnson et al. (2003) confirmed that historical malformations at six of eight sites (1947-1990) were associated with *Ribeiroia* infection. Moreover, three of these sites continued to support limb malformations at frequencies of 7-50% in one or more species. One pond in particular (Jette Pond in western Montana) showed increased prevalence of malformations in *Pseudacris regila* from 20% in 1960 to 46% in 2000, which was correlated with an increased intensity of *Ribeiroia* infection. Although more research is necessary, these studies provide evidence that malformations, especially those linked to *Ribeiroia* infection, are emerging in some amphibian populations.

Factors Influencing Infection

Nutrients - Nitrogen and phosphorus enrichment are widespread forms of anthropogenic environmental change that may influence parasite infection (Johnson and Carpenter 2008). Nutrient enrichment leads to eutrophication, which has been linked with emergence of *Ribeiroia ondatrae* through direct and indirect effects on aquatic food webs (Johnson and Chase 2004). The underlying mechanism through which eutrophication can increase infection involves increasing the density of infected first-intermediate snail hosts and by increasing the production of parasites by infected snails (Johnson et al. 2007).

Pesticides - Pesticides are still considered a potential factor influencing amphibian malformations (Johnson et al. 2010). However, recent investigations have focused on the interaction of pesticides with parasite infection (Kiesecker 2002; Rohr et al. 2008). Field experiments indicated a link between agricultural run-off and increased infection (Kiesecker 2002). Laboratory studies further demonstrated that pesticide exposure reduced host immunocompetence against parasites as the biological mechanism (Kiesecker 2002). Furthermore, there may be a link between nutrient addition and pesticide contamination jointly leading to increased infections by simultaneously increasing exposure and suppressing host immune systems (Rohr et al. 2008a, b).

Biodiversity - Host species diversity and community structure have been suggested to influence disease dynamics through a hypothesis termed the ‘dilution effect’ (Ostfeld and Keesing 2000). For *Ribeiroia ondatrae*, host species differences in susceptibility can influence infection patterns in multi-species communities (Johnson et al. 2008, Johnson and Hartson 2009). As a result, more diverse larval amphibian communities that include species with differing susceptibilities can alter parasite infection success and the resulting pathology in sensitive host species. For instance, larval toads (a sensitive species) raised experimentally alongside larval gray treefrogs (a resistant species) exhibited 37% less infection and fewer malformations (Johnson et al. 2008).

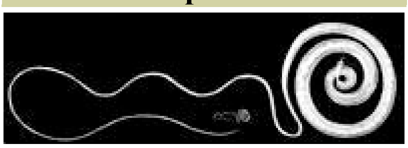
Parasite-induced Malformations and Amphibian Conservation

Perhaps the most unexplored, complex and vital aspects of trematode-caused amphibian malformations are the population-level consequences. Do these parasites and the malformations they induce pose a conservation risk? While no direct evidence linking trematode deformities and amphibian population declines exists, there are legitimate reasons for concern (Johnson and McKenzie 2008). For instance, in multiple species of frogs and toads, laboratory studies show that even low levels of *Ribeiroia* infection can induce 30-95% mortality. Correspondingly, multi-year field studies show that in localities of high infection and malformations in metamorphosing frogs, <2% of amphibians returning to breed exhibit malformations, suggesting *Ribeiroia* infection and malformations have deleterious consequences for individual survival and fecundity (Johnson et al. 2001). In specific wetlands that have historically exhibited a high prevalence of infection and malformations, several amphibian species have notably declined or disappeared. In light of these data, and the increasing evidence that *Ribeiroia* infections are on the rise (Johnson and McKenzie 2008), it is prudent to treat *Ribeiroia ondatrae* as a threat to amphibian populations and diversity, particularly in combination with other stressors.

Chapter 14

Trichuris Trichiura and Taenia asiatica

Trichuris trichiura

Whipworm	
	
Scientific classification	
Kingdom:	Animalia
Phylum:	Nematoda
Class:	Adenophorea
Order:	Trichurida
Family:	Trichuridae
Genus:	*Trichuris*
Species:	***T. trichiura***
Binomial name	
Trichuris trichiura (Linnaeus, 1771)	

The **human whipworm** (***Trichuris trichiura*** *or* ***Trichocephalus trichiuris***) is a roundworm, which causes trichuriasis when it infects a human large intestine. The name *whipworm* refers to the shape of the worm; they look like whips with wider "handles" at the posterior end.

Life cycle

The female *T. trichiura* produces 2,000–10,000 single celled eggs per day. Eggs are deposited from human feces to soil where, after two to three weeks, they become embryonated and enter the “infective” stage. These embryonated infective eggs are ingested and hatch in the human small intestine. This is the location of growth and molting. The infective larvae penetrate the villi and continue to develop in the small intestine. The young worms move to the cecum and penetrate the mucosa and there they complete development to adult worms in the large intestine. The life cycle from time of ingestion of eggs to development of mature worms takes approximately three months. During this time, there may be limited signs of infection in stool samples due to lack of egg production and shedding. The female *T. trichiura* begin to lay eggs after three months of maturity. Worms can live up to five years, during which time females can lay up to 20,000 eggs per day.

Recent studies using genome-wide scan revealed two quantitative trait loci on chromosome 9 and chromosome 18 may be responsible for genetic predisposition or susceptibility to infection of *T. trichiura* by some individuals.

Morphology

Trichuris trichiura has a narrow anterior esophageal end and shorter and thicker posterior anus. These pinkish-white worms are threaded through the mucosa. They attach to the host through their slender anterior end and feed on tissue secretions instead of blood. Females are larger than males; approximately 35–50 mm long compared to 30–45 mm. The females have a bluntly round posterior end compared to their male counterparts with a coiled posterior end. Their characteristic eggs are barrel-shaped, brown, and have bipolar protuberances.

Epidemiology

There is a worldwide distribution of *Trichuris trichiura*, with about 500 million human infections. However, it is chiefly tropical, especially in Asia and to a lesser degree, in Africa and South America. Within the United States, infection is rare overall but may be common in the rural Southeast where 2.2 million people are thought to be infected. Poor hygiene is associated with trichuriasis as well as the consumption of shaded moist soil, or food that may have been fecally contaminated. Children are especially vulnerable to infection due to their high exposure risk. Eggs are infective about 2–3 weeks after they are deposited in the soil under proper conditions of warmth and moisture, hence its tropical distribution.

Infection

Infection occurs through ingestion of eggs (which are usually found in dry goods such as beans, rice, and various grains) and is more common in warmer areas. The eggs hatch in the small intestine, and then move into the wall of the small intestine and develop. On reaching adulthood, the thinner end (the front of the worm) burrows into the large intestine and the thicker end hangs into the lumen and mates with nearby worms. The females can grow to 50 mm (2 inches) long. Neither the male nor the female has much of a visible tail past the anus.

Whipworm commonly infects patients also infected with *Giardia*, *Entamoeba histolytica*, *Ascaris lumbricoides*, and hookworms.

Symptoms and pathology

- Light infestations (<100 worms) are frequently asymptomatic.
- Heavy infestations may have bloody diarrhea.
- Long-standing blood loss may lead to iron-deficiency anemia.
- Rectal prolapse is possible in severe cases.
- Vitamin A deficiency may also result due to infection.

Mechanical damage to the mucosa may occur as well as toxic or inflammatory damage to the intestines of the host.

Diagnosis

Trichuriasis can be diagnosed when *T. trichiura* eggs are detected in stool examination. Eggs will appear barrel-shaped, unembryonated, having bipolar plugs and a smooth shell. Rectal prolapse can be diagnosed easily using defecating proctogram and is one of many methods for imaging the parasitic infection. Sigmoidoscopy show characteristic white bodies of adult hanging from inflamed mucosa (coconut cake rectum).

Treatment and control

Mebendazole is 90% effective in the first dose, and albendazole may also be offered as an anti-parasitic agent. Adding iron to the bloodstream helps solve the iron deficiency and rectal prolapse.

Infection can be avoided by proper disposal of human feces, avoiding fecal contamination of food, not eating dirt, and avoiding crops fertilized with night soil. Simple and effective proper hygiene such as washing hands and food is recommended for control.

Dog and cat whipworms

Egg of *Trichuris vulpis*

Egg of *Trichuris vulpis*

Whipworms develop when a dog swallows whipworm eggs, passed from an infected dog. Symptoms may include diarrhea, anemia, and dehydration. The **dog whipworm** (***Trichuris vulpis***) is commonly found in the U.S. It is hard to detect at times, because the numbers of eggs shed are low, and they are shed in waves. Centrifugation is the preferred method. There are several preventives available by prescription from a veterinarian to prevent dogs from getting whipworm.

The **cat whipworm** is a rare parasite. In Europe it is mostly represented by ***Trichuris campanula***, and in North America it is ***Trichuris serrata*** more often. Whipworm eggs found in cats in North America must be differentiated from lungworms, and from mouse whipworm eggs just passing through.

Whipworm as a therapeutic agent for IBD and other inflammatory disorders

The hygiene hypothesis suggests that various immunological disorders that have been observed in humans only within the last 100 years, such as Crohn's disease, or that have become more common during that period as hygienic practices have become more widespread, may result from a lack of exposure to parasitic worms (also called helminths) during childhood. The use of *Trichuris suis* ova (TSO, or pig whipworm eggs) by Weinstock, et al., as a therapy for treating Crohn's disease and to a lesser extent ulcerative colitis are two examples that support this hypothesis. There is also anecdotal evidence that treatment of inflammatory bowel disease (IBD) with TSO decreases the incidence of asthma, allergy, and other inflammatory disorders. Some scientific evidence suggests that the course of multiple sclerosis may be very favorably altered by helminth infection; TSO is being studied as a treatment for this disease.

Taenia asiatica

Taenia asiatica	
Scientific classification	
Kingdom:	Animalia
Phylum:	Platyhelminthes
Class:	Cestoda
Order:	Cyclophyllidea
Family:	Taeniidae
Genus:	*Taenia*
Species:	***T. asiatica***
Binomial name	
Taenia asiatica Eom and Rim, 1993	

Taenia asiatica is commonly known as **Asian taenia** or **Asian tapeworm** and is a parasitic tapeworm of humans and pigs. It is one of the three species of *Taenia* that infect humans and causes taeniasis. Discovered only in 1980s from Taiwan and other East Asian countries, it is notoriously similar to *Taenia saginata* that it was believed to be a slightly different strain. But the taxonomic consensus turns out to be that it is a unique species. Like for other taenids, humans are the definitive hosts, but in contrast both cattle and pigs can serve as intermediate hosts.

Discovery

T. asiatica was first recognized in Taiwan, and subsequently in Korea and other Asian countries; therefore it was originally known as Asian *Taenia saginata*, as it appeared to be exclusive to Asia. But studies on the biology began to throw light to its difference from the classical *T. saginata*. Firstly the tapeworm infects visceral organs such as liver, serosa and lungs of pigs, and liver of cattle; while *T. saginata* is known to infect only the muscle of cattle. Secondly there are significant morphological variations though their resemblance is overwhelming. For a time it was resolved to be a sub-species, and was named *T. saginata asiatica*. But further analyses imposed its taxonomic revision into a valid species *T. asiatica*. Comparison of the mitochondrial genome also provide further support to its taxonomic status.

Description

The body is yellowish white in colour, about 350 cm long and 1 cm broad, divided into the anterior scolex, followed by a short neck and a highly extended body proper called strobila. The strobila is composed a series of ribbon-like segments called proglottids. There are more than 700 proglottid]s in the strobila. The scolex bears of 4 simple suckers. The segments are made up of mature and gravid proglottids. It is an acoelomate animal with no digestive system. It is unique in having posterior protuberances in the gravid proglottid, which are absent in other taenids including *T. saginata*. The total number of proglottids is less than 1000 (~900), while *T. saginata* have more than 1000 proglottids. The protoscolex of cysticercus of the Taiwan *Taenia* has a sunken rostellum while that of *T. saginata* has only an apical pit. In addition, the rostellum is usually surrounded by two rows of rudimentary hooklets. The distinct rostellum on the scolex, the large number of uterine twigs and the existence of posterior protuberance in adult were the defining characters. Moreover, the metacestode was different morphologically from that of *T. saginata* in having wart-like formations on the external surface of the bladder wall.

Life cycle

The life cycle is indirect and complicated, and is completed in humans as the definitive host, and the intermediate host is mostly pigs, and cattle on rare occasion. The adult worm inhabits the small intestine of humans. Fertilized eggs are released through the faecesalong with the gravid proglottid which gets detached from the strobila. Pigs and cattle ingest the infective embryo while grazing. The digestive enzymes will break the thick shell of the egg and allow formation of the zygotes called "oncospheres". These zygotes then penetrate the mucous layer of the digestive tract and enter the circulation of the host. This is where the young larval stages form a pea-sized, fluid filled cyst, also known as “cysticercus”, which migrate to visceral organs like liver, serosa and lungs in pigs, and liver in cattle.

Epidemiology

The parasite is known in Asian countries including Taiwan, Korea, Indonesia, Thailand and China. In addition, molecular genotyping techniques have revealed that the disease also occurs in Japan, the Philippines, and Vietnam.

Diagnosis

The basic diagnosis is examination of a stool sample to find the parasite eggs. However there is a serious limitation as to the identification of the species because the eggs of all human taenids look the same. It is extremely difficult to identify *T. asiatica* from other taenids because of their striking resemblances. The species and *T. saginata* are frequently confused due to their morphological similarities and sympatric distribution. Identification often requires histological observation of the uterine branches and PCR detection of ribosomal 5.8S gene. The presence of rostellum on the scolex, a large number of uretine branches (more than 57) and prominent posterior protuberances in gravid proglottids, and wart-like formation on the surface of the larvae are the distinguishing structures.

To date the most relevant diagnosis of taeniasis due to *T. asiatica* is by enzyme-linked immunoelectrotransfer blot (EITB). EITB can effectively identify it from other taenid infections since serological test indicates that immunoblot band of 21.5 kDa exhibited specificity only to *T. asiatica*.

Treatment

Niclosamide (2 mg) was very effective against experimental infection in human. In general cestode infections are treated with praziquantel and albendazole.