Trends in Parasitology | Vector of the Month

Aedes albopictus (Asian Tiger Mosquito)

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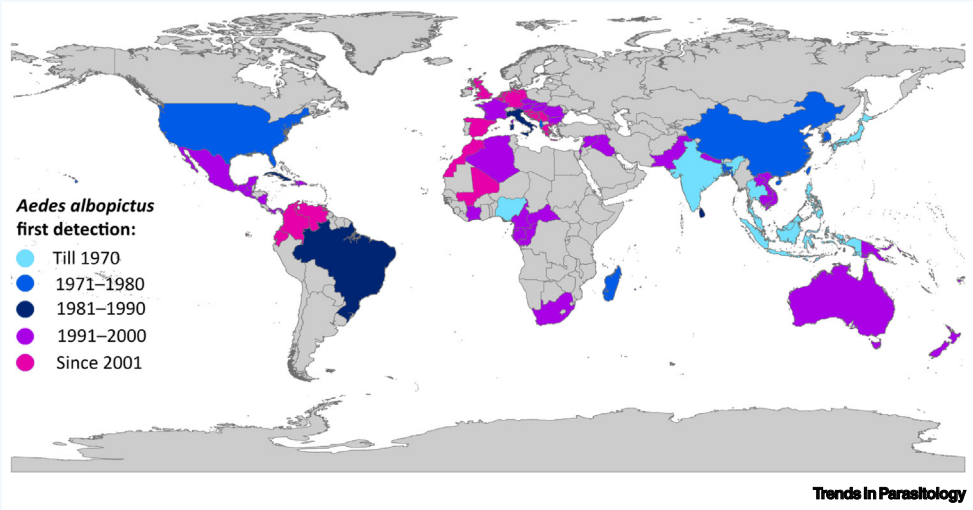
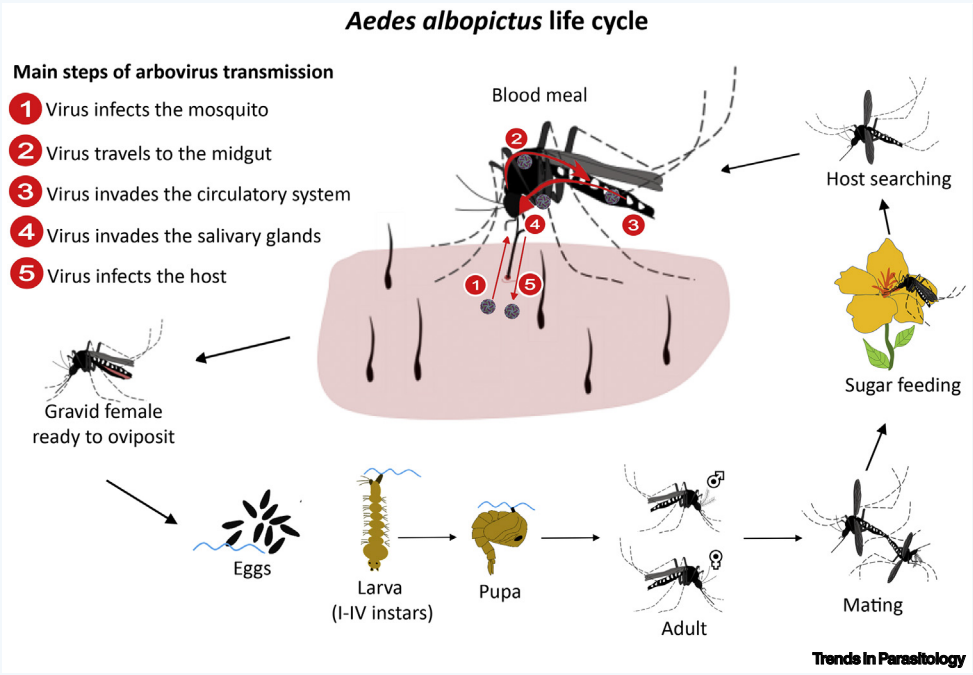


Aedes albopictus originated in the tropical forests of Southeast Asia. It is currently ranked among the top 100 invasive species worldwide and can be found on all continents. It is a vector of chikungunya, dengue, and Zika viruses, and the filarial worms that cause dirofilariasis, among other agents. *Ae. albopictus* is a zoophilic species, but a preference for human blood meals is known. It has high levels of ecological and physiological plasticity (e.g., drought-resistant eggs, cold-acclimated adults exploiting various breeding sites, and 5–17 generations per year), allowing its fast adaptation to urban/suburban environments and colder regions. Notably, trade and travel globalization, climate change, superior competition for food over other *Aedes* species, as well as the lack of reliable surveillance and effective control tools boost its worldwide-scale invasion. Its resistance to commonly used larvicides and adulticides is well recognized, and the development of novel control tools with proven epidemiological impact is challenging.



TRANSMISSION FACTS:
Ae. albopictus transmits >25 arboviruses, including chikungunya, dengue, and Zika viruses, and filarial worms such as *Dirofilaria* spp. It is susceptible to infection by a few *Plasmodium* species (*Plasmodium gallinaceum* and to a lesser extent *Plasmodium relictum*).

Intense day-biting activity, mainly outdoors.

A wide host range, from main host mammals to birds, reptiles, and amphibians, representing a bridge vector of zoonotic pathogens to humans.

Some *Wolbachia* endosymbionts may induce cytoplasmic incompatibility and reduce arbovirus transmission.

CONTROL FACTS:
Conventional control is mainly based on temephos and *Bacillus thuringiensis* subsp. *israelensis* (*Bti*) larvicides. Other biocontrol tools include entomopathogenic fungi and larvivorous natural enemies (e.g., copepods).

If larvicidal control fails, or in emergency situations, space spraying with pyrethroids or organophosphates can be used against adults.

The use of synthetic insecticides is hampered by the quick resistance development in exposed populations.

Removing urban breeding sites is crucial; promising results were obtained with toxic sugar baits.

Insect repellents and insecticide-treated materials help to reduce vector-biting activity on humans and pets.

Control approaches based on the sterile insect technique (SIT) and the incompatible insect technique (IIT)-SIT achieved positive results.

TAXONOMY AND CLASSIFICATION:
PHYLUM: Arthropoda
CLASS: Insecta
ORDER: Diptera
FAMILY: Culicidae
GENUS: *Aedes*
SPECIES: *Ae.* (*Stegomyia*) *albopictus* (Skuse 1894)

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Resources

www.ecdc.europa.eu/en/disease-vectors/facts/mosquito-factsheets/aedes-albopictus
www.cabi.org/isc/datasheet/94897
www.invasivespeciesinfo.gov/profile/asian-tiger-mosquito
www.who.int/news-room/detail/14-11-2019-mosquito-sterilization-offers-new-opportunity-to-control-chikungunya-dengue-and-zika

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Trends in Parasitology | Parasite of the Month

Ancylostoma ceylanicum

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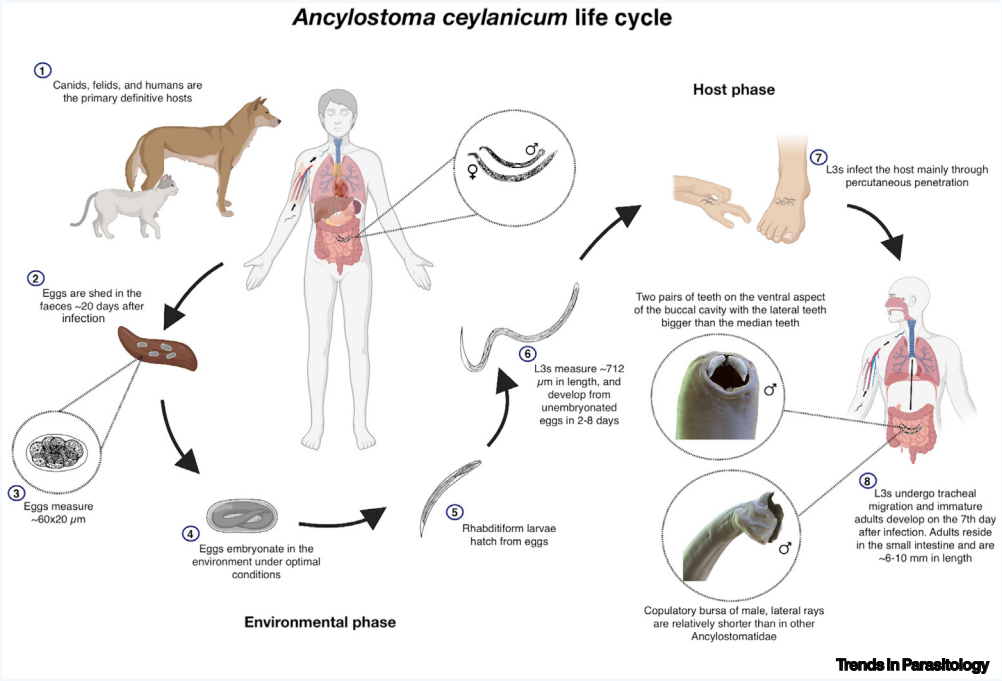
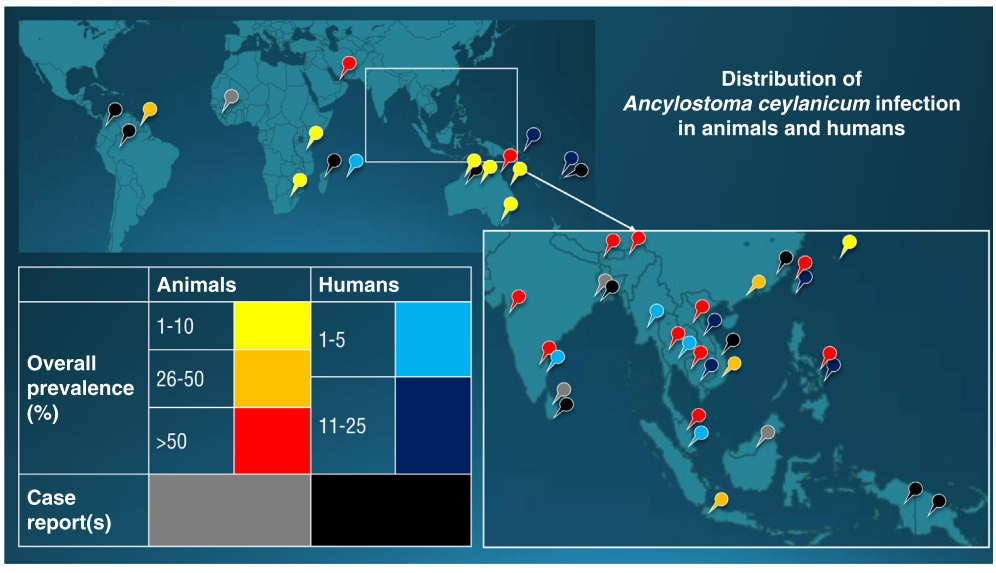


Ancylostoma ceylanicum is a soil-transmitted helminth recognised as the second most common hookworm species (after *Necator americanus*) infecting humans in the Asia Pacific region. In contrast to the human-specific hookworms *N. americanus* and *Ancylostoma duodenale*, *A. ceylanicum* is zoonotic, with canids as the primary reservoir for human infection. Thus, the distribution of human infections largely mirrors that of dogs. *A. ceylanicum* displays a direct life cycle with adult parasites residing in the small intestine of the definitive hosts. Humans acquire infection via oral ingestion or percutaneous penetration of infective third-stage larvae (L3s) from the environment. L3s make their way into the blood circulation and undergo tracheal migration before moulting to fifth-stage larva 7 days postinfection. Juveniles develop to adult worms, mate, and produce eggs 14 days after infection in animals and 18–35 days in humans.



KEY FACTS:
Microscopy-based diagnostic techniques are unable to differentiate hookworm eggs at species level. Molecular tools can identify hookworm species directly from eggs in faeces and have led to the discovery of *A. ceylanicum* as a hookworm infecting humans with an estimated 100 million cases worldwide.

A. ceylanicum/hamster is used as a laboratory model for studying human hookworm infection.

Control strategies for hookworms aim to reduce morbidity through periodic treatment of at-risk human populations with albendazole or mebendazole.

A One Health approach will be fundamental to achieve sustained long-term control of this zoonotic hookworm.

DISEASE FACTS:
Clinical outcomes of *A. ceylanicum* infections in humans include increase in eosinophil count, temporary ground itch, abdominal pain, fatigue, weight loss, fever, diarrhoea, melena, vomiting, and dyspnoea.

Hookworm infection causes blood loss and iron-deficiency anaemia, which may have serious implications for women of child-bearing age, pregnant women, and children living in hookworm-endemic countries.

In dogs, heavy infections result in large quantities of blood loss in the stool followed by microcytic hypochromic iron-deficiency anaemia.

Further studies are urgently needed to characterise *A. ceylanicum*-associated morbidity at a population scale.

TAXONOMY AND CLASSIFICATION:
PHYLUM: Nematoda
CLASS: Chromadorea
ORDER: Strongylida
FAMILY: Ancylostomatidae
GENUS: *Ancylostoma*
SPECIES: *A. ceylanicum*

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Declaration of interests

The authors declare no competing interests.

Resources

https://parasite.wormbase.org/Ancylostoma_ceylanicum_prjna72583/Info/SpeciesLanding/
www.cdc.gov/parasites/hookworm/index.html
www.troccap.com/canine-guidelines/gastrointestinal-parasites/hookworms/

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Anisakis pegreffii

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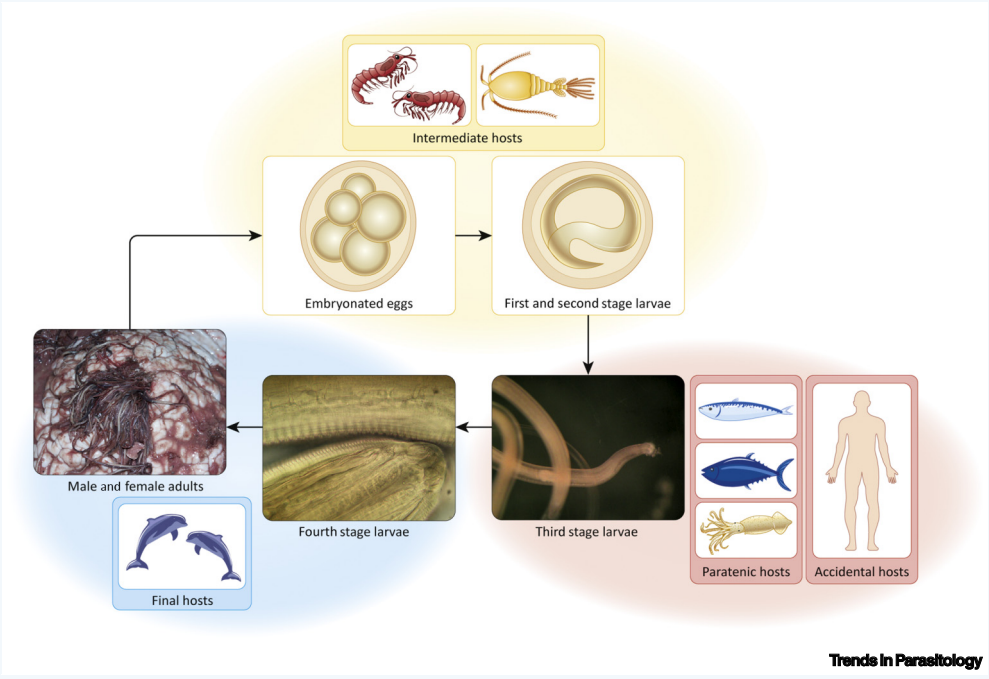
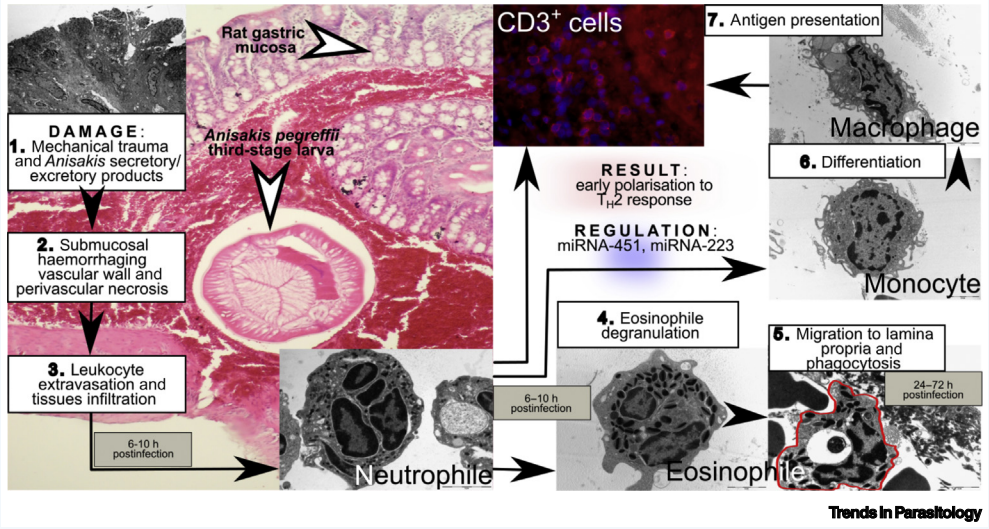


Anisakis pegreffii belongs to the *Anisakis simplex* species complex that causes anisakiasis, an important fish-borne zoonosis worldwide. The parasite typically reproduces in the gastric chambers of toothed whales (final hosts). Larvae hatch from the eggs that are expelled in seawater; the larvae are ingested by crustaceans and small fish (intermediate hosts). Many fish and cephalopod species (paratenic hosts) that prey upon them transport infective third-stage larvae (L3) to the final host. Humans are accidentally infected by L3 through consumption of uncooked seafood. Anisakiasis is characterised by four main types: gastric, intestinal, ectopic, and gastroallergic that elicit mostly mild, only occasionally severe symptoms. Recently, a fifth type has been proposed based on many asymptomatic *Anisakis*-IgE seropositive subjects, predominately in countries with high per capita fish consumption. In humans, L3 fails to develop into the adult stage, dies, and decomposes in infected tissues, evoking a local proinflammatory response characterized by eosinophilic granulomatosis.



KEY FACTS:
The L3 excretory gland cell generates excretory and secretory products engaged in host tissue degradation, larval penetration and migration, feeding and ecdysis, antigenic interaction with host immunity, and antimicrobial activity.

Lesions in dolphins (final host) range from ulcerative gastritis, chronic granulomatous gastritis, focal oedema, and fibrosis to multifocal granulomatous jejunitis, and can have a secondary role in mammalian stranding.

Early (32 h) lesions in the rat (model accidental host) include severe inflammatory and haemorrhagic changes in stomach and muscle tissues with neutrophil, eosinophil, and macrophage infiltration.

DISEASE FACTS:
Anisakiasis is considered to be the fifth most important food-borne parasitosis in Southwest Europe.

The estimated frequency is 2000 cases/year in Japan, 200 in South Korea, and 20–500 in some European countries, the latter being most likely underestimated.

Symptoms in the gastric form are epigastric pain, nausea, and vomiting 1–12 h postingestion; in the intestinal form they are abdominal pain 48–72 h postingestion; in the ectopic form they are damage associated with larval migration through body cavities; in the gastroallergic form they range from urticaria to anaphylactic shock. Live L3 is generally considered necessary for the first sensitisation.

TAXONOMY AND CLASSIFICATION:
PHYLUM: Nematoda
CLASS: Chromadorea
ORDER: Rhabditida
FAMILY: Anisakidae
GENUS: *Anisakis*
SPECIES: *A. pegreffii* (in the *A. simplex* species complex)

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Resources

http://parasite-project.eu/outcomes
www.cdc.gov/parasites/anisakiasis/index.html
www.fao.org/3/y4743e/y4743e0c.htm

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Trends in Parasitology | Parasite of the Month

Anncaliia algerae

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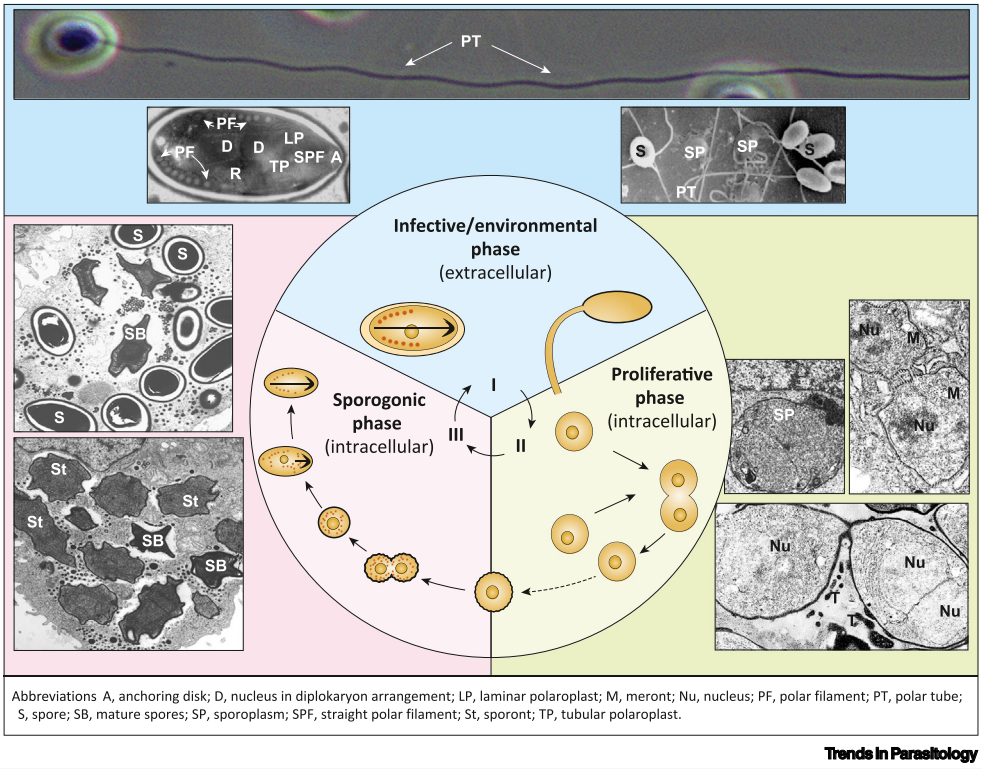
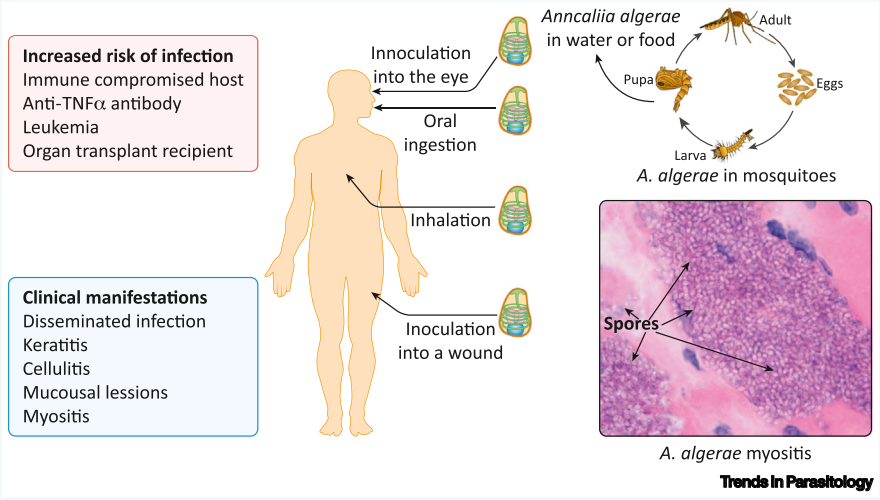


Anncaliia algerae belongs to the microsporidia, a group of obligate intracellular pathogens originally classified as early branching 'primitive' protozoa but now understood to be related to the Cryptomycota as a basal branch in the fungal kingdom. *A. algerae* has emerged as a rare opportunistic human pathogen in immune compromised patients such as those taking immune suppressive medications for arthritis, hematologic malignancy, or organ transplantation. It was originally identified as a pathogen of mosquitoes and is probably transmitted to humans by food or water through ingestion, inhalation, or contamination of ocular tissue or wounds with environmental spores. *A. algerae* infection primarily causes myositis; however, vocal cord, skin, corneal (in immune competent hosts), and disseminated infections have been reported. Human infection has also been reported with other members of the *Anncaliia* genus: *Anncaliia vesicularum* in a HIV patient, and *Anncaliia connori* in an infant with thymic dysplasia.



KEY FACTS:

A. algerae was previously called *Nosema algerae* and then *Brachiola algerae* before being reclassified to *Anncaliia.*

A. algerae can be grown *in vitro* in either mammalian or insect cells.

The genome sequences for human (ATCC PRA 109) and insect (ATCC PRA339, also called Undeen) isolates are available. The genome contains a large number of transposable elements and long terminal repeat (LTR) retrotransposons.

It has a worldwide distribution and is a pathogen of mosquitoes and other insects.

DISEASE FACTS:

Most cases have occurred in immune suppressed patients who either received immune modulating antibodies for arthritis or immune suppressive drugs for organ transplantation.

Myositis may be associated with central nervous system or cardiac involvement.

Infection is diagnosed by finding spores and other developmental forms in tissue. PCR based on the SSU-rRNA gene can also be used for diagnosis.

Management of *A. algerae* infection usually requires minimizing immunosuppression.

Albendazole has *in vitro* efficacy and has resulted in clinical improvement in several cases. The *A. algerae* tubulin sequence has amino acids associated with sensitivity to albendazole. The addition of fumagillin to albendazole has been needed for successful therapy in some cases.

TAXONOMY AND CLASSIFICATION:

PHYLUM: Microsporidia
CLASS: Microsporea
ORDER: Microsporidia
SUPERFAMILY: Tubulinosematoidea
FAMILY: Tubulinosematidae
GENUS: *Anncaliia*
SPECIES: A. *algerae*

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Declaration of interests

The authors declare no competing interests.

Resources

https://microsporidiadb.org/micro/app
www.ATCC.org
https://onlinelibrary.wiley.com/doi/book/10.1002/9781118395264

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Anopheles stephensi (Asian Malaria Mosquito)

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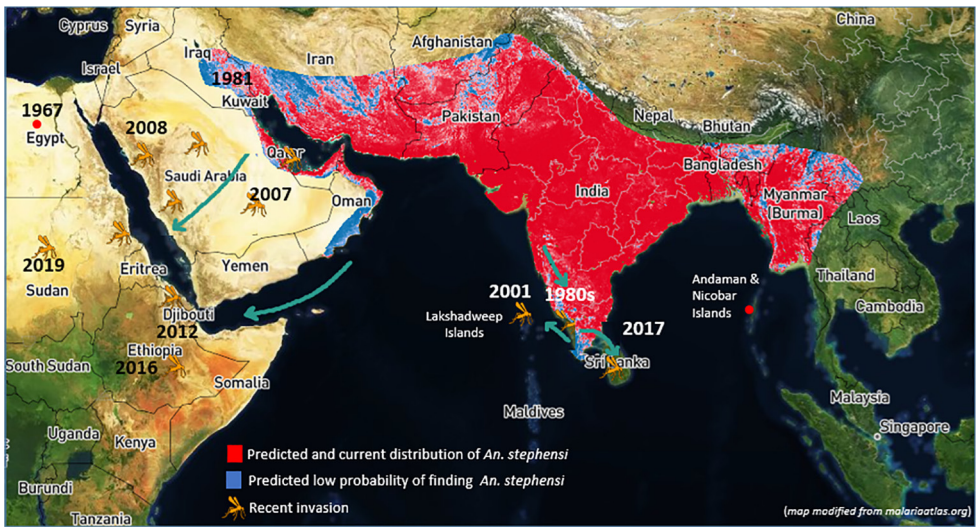
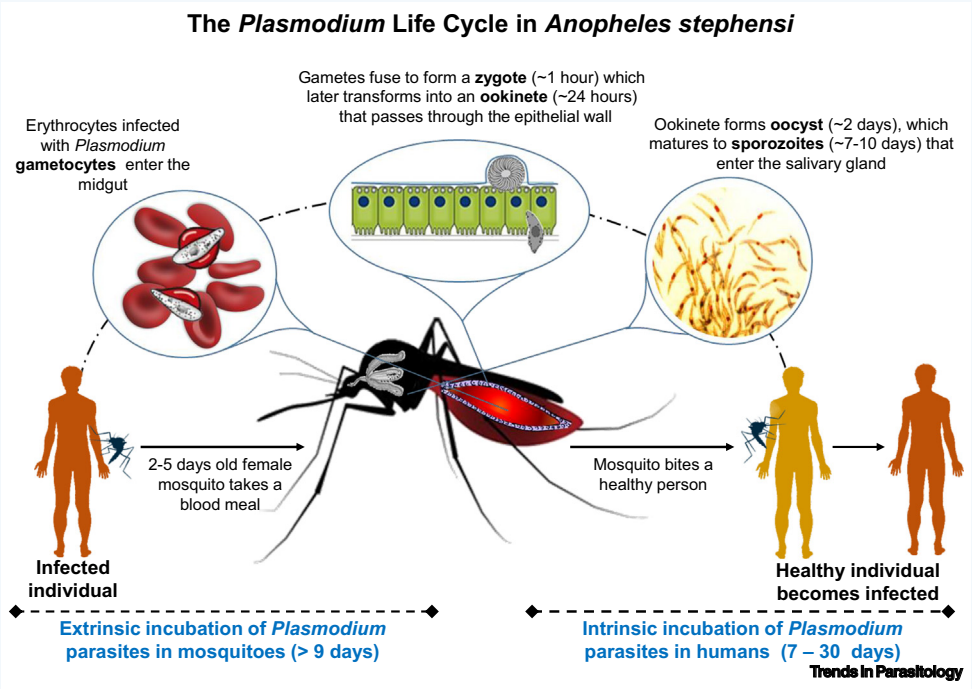


Anopheles stephensi originated in Southeast Asia and the Arabian Peninsula. It has recently emerged as an efficient and invasive urban malaria vector. There are three known forms, 'type', 'intermediate', and 'mysorensis', of which the type and intermediate forms are efficient vectors in both rural and urban environments. Typical breeding sites are artificial containers, such as domestic wells, cisterns, overhead water tanks, and roof gutters, with clean and unpolluted water allowing rapid expansion to urban environments. *An. stephensi* has rapidly extended its geographical range, with the type form being reported in the Lakshadweep Islands (2001), in countries in the Horn of Africa (2012), Sri Lanka (2017), and most recently in the Republic of Sudan (2019). The introduced *An. stephensi* exhibits resistance to several classes of insecticides, posing challenges in controlling the spread to new areas.



TRANSMISSION FACTS:
An. stephensi transmits *Plasmodium vivax* and *Plasmodium falciparum* parasites. It is a day-biting, anthropophilic mosquito, found mainly in cattle sheds near human dwellings (endophilic) and feeds indoors (endophagic).

Perennial, with seasonal peaks mainly in July–August and October–December. It can survive extremely high temperatures during the dry season when malaria transmission usually reaches a seasonal low.

Some *Wolbachia* endosymbionts may induce cytoplasmic incompatibility and reduce *Plasmodium* transmission.

CONTROL FACTS:
Conventional methods include removal of larval habitats and installation of hermetically sealed lids to water storage containers to prevent vector breeding.

Larval source management using biocontrol agents (copepods) and botanical larvicides following World Health Organization (WHO) guidelines.

International health regulations should be enforced to ensure that airports and other points of exit are free of mosquitoes.

Adult mosquitoes can be controlled with long-lasting insecticidal nets, indoor residual spraying, and installation of mechanical barriers to reduce vector-biting activity on humans.

Control approaches based on the CRISPR-Cas9 system have achieved positive results in the laboratory but are yet to be field-tested.

TAXONOMY AND CLASSIFICATION:
PHYLUM: Arthropoda
CLASS: Insecta
ORDER: Diptera
FAMILY: Culicidae
GENUS: *Anopheles*
SPECIES: *An. stephensi* (Liston, 1901)

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Declaration of Interests

There are no interests to declare.

Resources

www.who.int/publications/i/item/vector-alert-anopheles-stephensi-invasion-and-spread
https://malariaatlas.org/
www.ncbi.nlm.nih.gov/assembly/GCF_013141755.1

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Trends in Parasitology | Parasite of the Month

Batrachochytrium dendrobatidis

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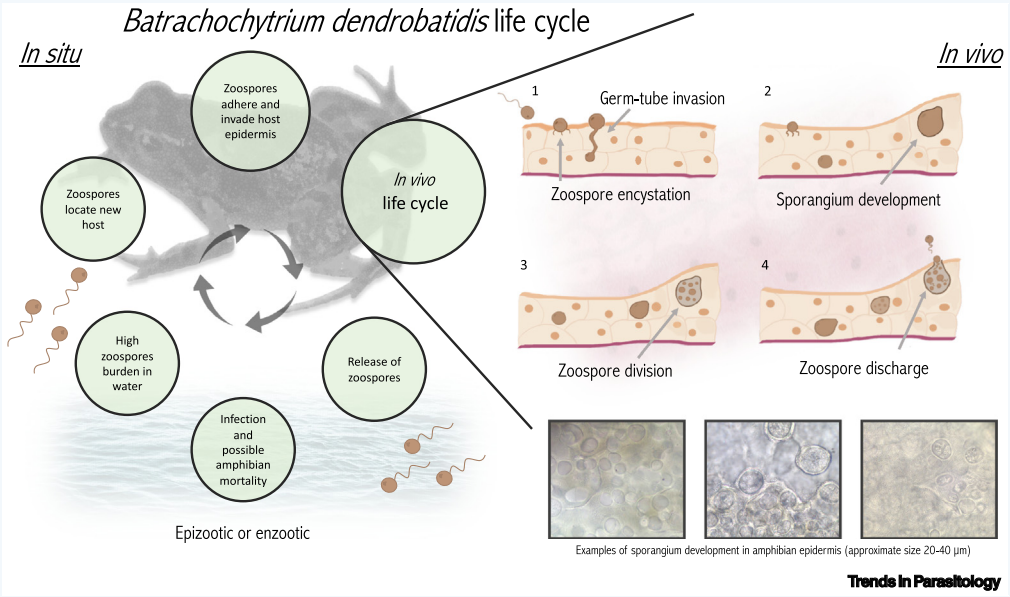
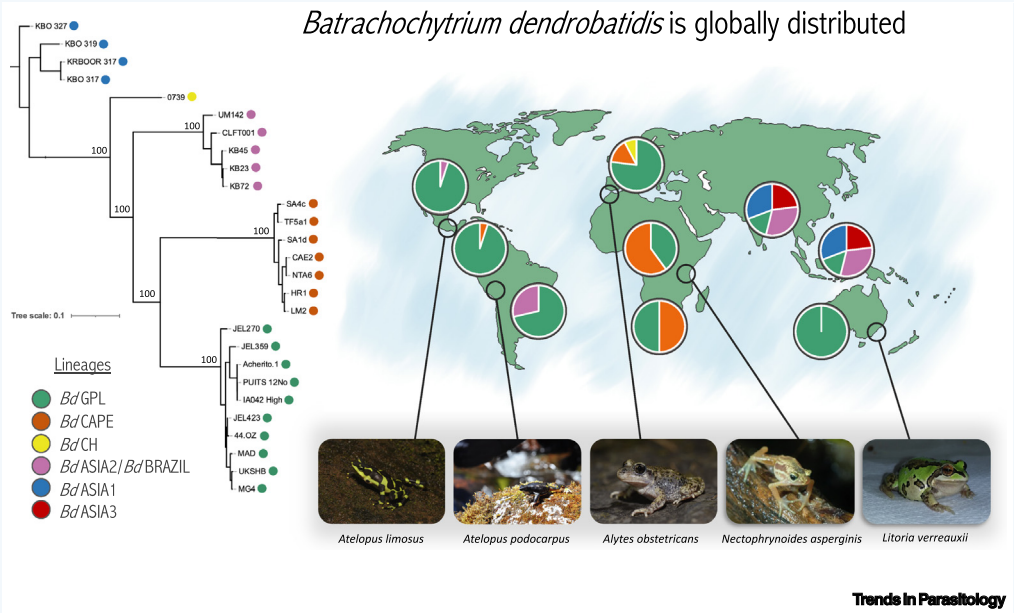


Batrachochytrium dendrobatidis is a zoospore-forming aquatic fungus and the etiological agent of chytridiomycosis, a skin infection affecting all orders of amphibians. *B. dendrobatidis* emerged worldwide during the 20th century, causing a panzootic that has contributed to widespread declines in the abundance of at least 501 amphibian species and 90 presumed extinctions. The pathogen has a two-stage life cycle, whereby flagellate-motile zoospores seek out amphibians by means of chemotaxis before attaching, encysting, and developing intracellularly into zoosporangia, which gestate and discharge new zoospores. Factors leading to the onset of chytridiomycosis following exposure to zoospores are complex, consisting of biotic and abiotic components that influence host–pathogen interactions. This results in differing clinical outcomes within and amongst species that range from no or mild symptoms to death and declines.



Key facts:
Chytridiomycosis was first observed in Australia and the Neotropics in 1998. *B. dendrobatidis* was later discovered as the cause of the devastating declines in amphibian abundance across the Neotropics, formally named in 1999 and listed as a notifiable species by the World Organization for Animal Heath (OIE) in 2008.

Panzootic chytridiomycosis is thought to have emerged from an endemic range in East Asia (Korea). Five genetic lineages of *B. dendrobatidis* are believed to have emerged out of Asia within the past century, resulting in a complex phylogeographically correlated population genetic structure.

Disease facts:
Panzootic chytridiomycosis is driven primarily by the emergence and expansion of a highly virulent *B. dendrobatidis* genotype named *Bd*GPL (Global Panzootic Lineage). International spread occurs in traded amphibians.

B. dendrobatidis infects the epidermis of postmetamorphic anurans, where it disrupts cutaneous osmoregulatory function, leading to electrolyte imbalance and, in some cases, death by asystolic cardiac arrest.

B. dendrobatidis produces a germ tube that physically invades epithelial cells, allowing the pathogen to become intracellular. Zoosporangium then form intracellularly before migrating towards the epidermal surface to release infectious zoospores.

Taxonomy and classification:
Phylum: Chytridiomycota
Class: Chytridiomycetes
Order: Rhizophydiales
Genus: *Batrachochytrium*
Species: *B. dendrobatidis*

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Declaration of interests

The authors declare no competing interests.

Resources

www.broadinstitute.org/fungal-genome-initiative/batrachochytrium-genome-project
www.cabi.org/isc/datasheet/109730
https://twitter.com/ChytridCrisis
https://amphibiandisease.org

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Trends in Parasitology | Parasite of the Month

Blastocystis

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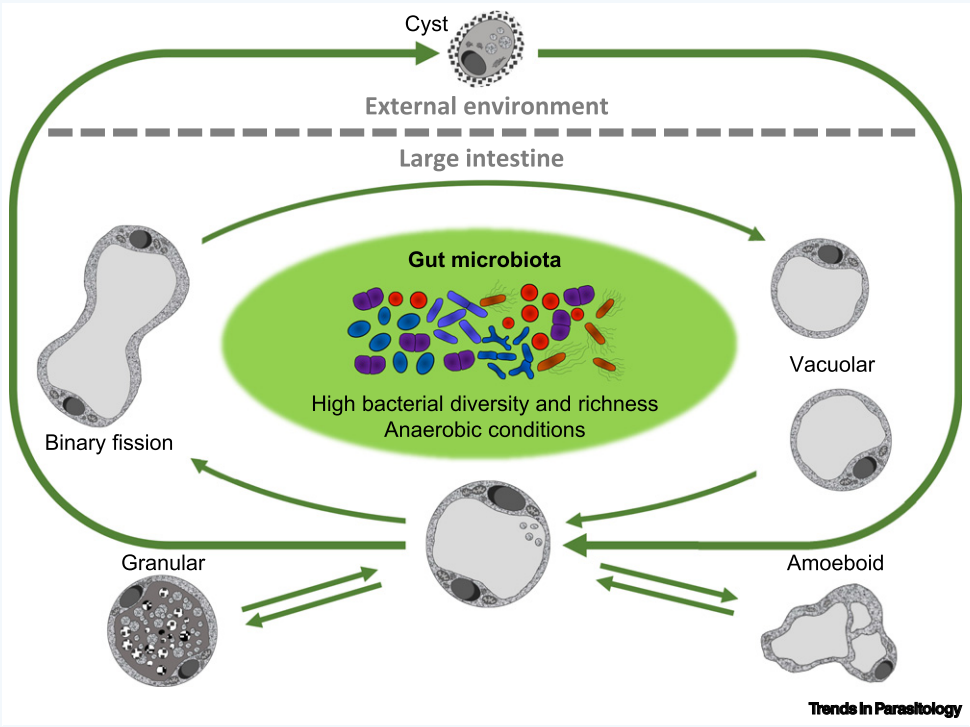
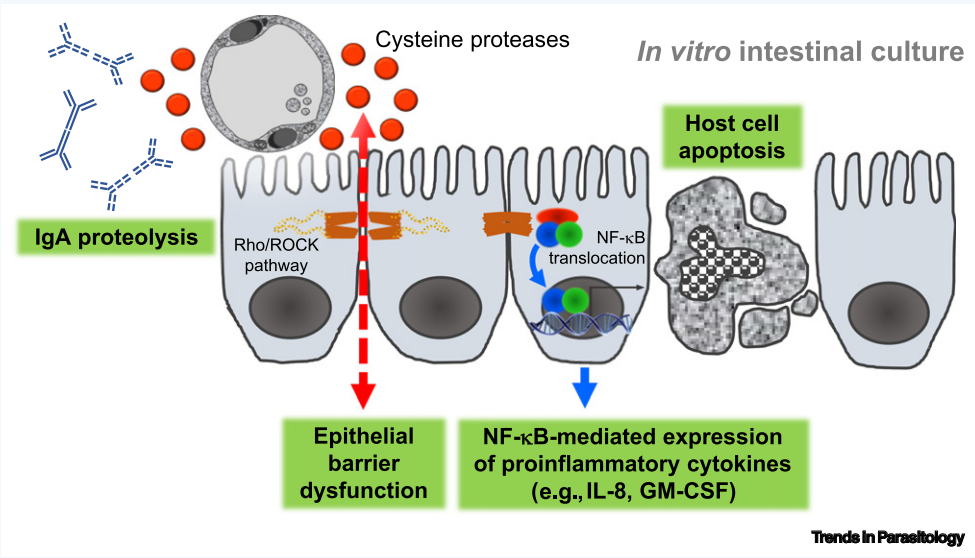


Blastocystis colonizes the large intestine and divides by binary fission. *In vitro*, *Blastocystis* can adhere to intestinal mucin and secrete cysteine proteases that contribute to pathogenesis through degradation of secretory IgA, Rho/ROCK-mediated tight-junction compromise, NF-κB-mediated secretion of inflammatory cytokines, and host cell apoptosis. It is currently unknown whether this occurs *in vivo*. Most gut microbiota studies that include *Blastocystis* report that *Blastocystis* is a common constituent of the healthy gut microbiota and is associated with higher bacterial richness, and that long-term asymptomatic carriage is common. In contrast, a couple of recent studies have suggested that *Blastocystis* decreases beneficial gut bacteria, leading to a dysbiotic state. Such discrepant observations have led to confusion on the clinical relevance of the parasite. *Blastocystis* is relatively rare in patients with inflammatory bowel disease, and its role in irritable bowel syndrome is still controversial.



KEY FACTS:

Blastocystis from mammals and birds can be classified into at least 17 subtypes (STs) currently, based on small subunit (SSU) rRNA genes. STs are as divergent as species or even genera.

Humans can host ST1–9 and 12; more than 90% of human *Blastocystis* strains belong to ST1–4.

Reservoir hosts have been identified for all subtypes except ST9; cryptic host specificity exists for at least some of them.

Two genomes: a nuclear genome of 12.9–18.8 Mb (depending on ST) encoding 5713–6544 proteins, and a mitochondrial genome of 27.7–29.3 kb.

Blastocystis can be cultured easily in Jones' and other media with fecal bacteria. A genetic manipulation method for ST7 has been described recently.

Subtype nomenclature was introduced when it became clear that the names of previous species were invalid or represented multiple very distinct entities.

DISEASE FACTS:

Despite more than 1 billion carriers worldwide, the public health significance remains unknown.

Blastocystis has been found more commonly in the gastrointestinal tract of healthy individuals.

Gut bacterial diversity and richness are mostly higher in *Blastocystis*-positive individuals. ST7 has been shown to decrease the levels of beneficial gut bacteria such as *Bifidobacterium* and *Lactobacillus*.

The zoonotic contribution to human *Blastocystis* colonization is probably low.

TAXONOMY AND CLASSIFICATION:

KINGDOM: Sar
PHYLUM: Stramenopiles
CLASS: Bigyra
ORDER: Opalinata
FAMILY: Blastocystidae
GENUS: *Blastocystis*
SPECIES: Currently not applicable

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  https://doi.org/10.1016/j.pt.2019.12.008

Resources

www.pubmlst.org/blastocystis

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  https://doi.org/10.1016/j.pt.2019.12.008

Trends in Parasitology | Parasite of the Month

Clonorchis sinensis

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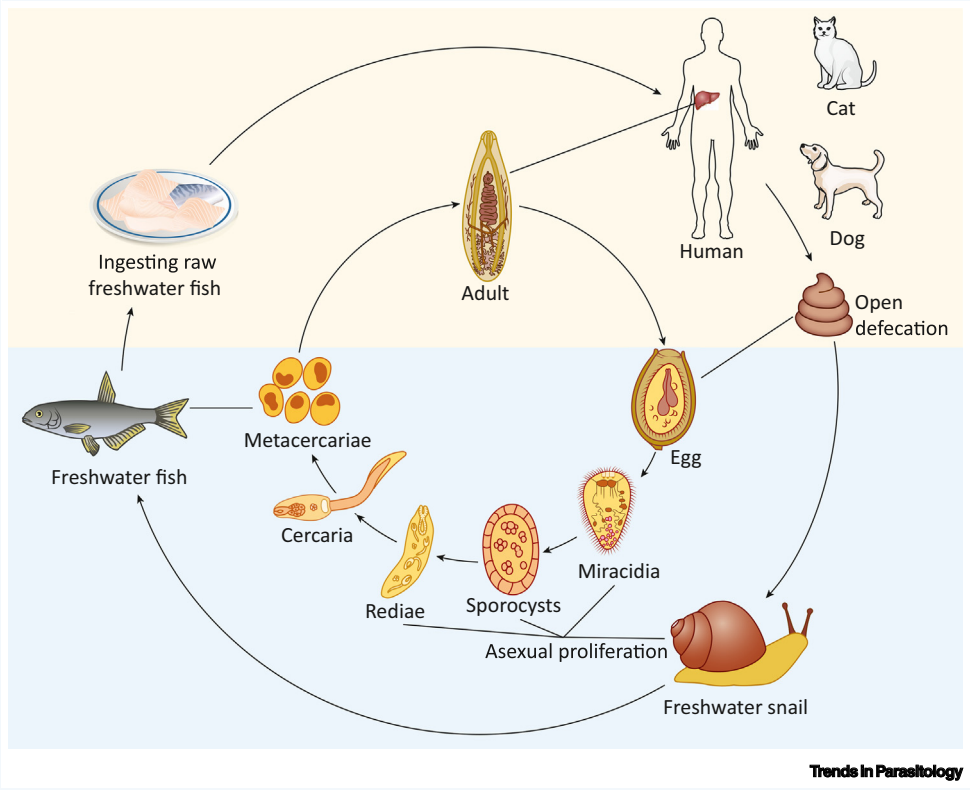
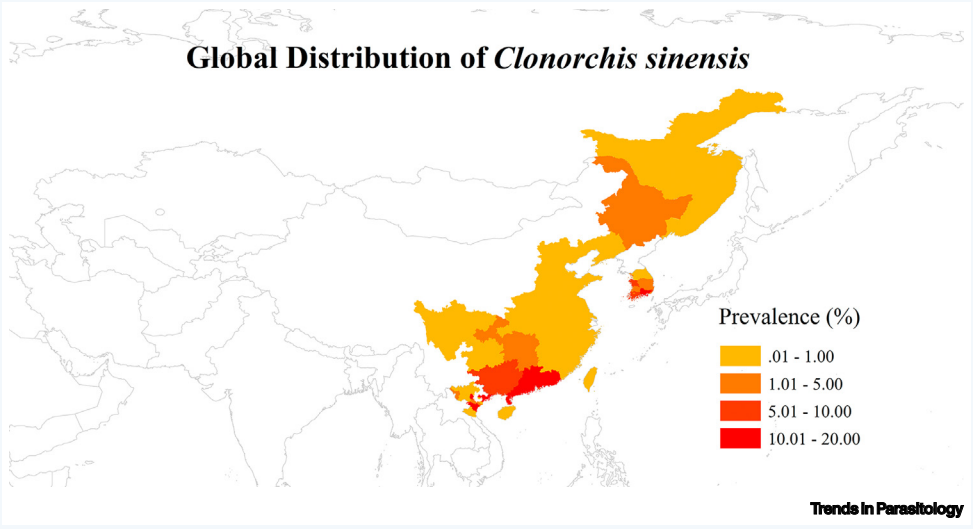


The life cycle of *Clonorchis sinensis* involves freshwater snails and fishes as intermediate hosts and humans and piscivorous animals as definite hosts. Humans are infected through ingestion of raw or undercooked freshwater fish containing *C. sinensis* metacercariae. Around 15 million people are infected in China, South Korea, northern Vietnam, and far east of Russia. A high burden is exerted due to diverse hepatobiliary morbidity (e.g., cholangitis, cholecystitis, cholelithiasis, and cholangiocarcinoma). A vaccine is not currently available. Chemotherapy for morbidity control is the mainstream intervention against clonorchiasis. Mass drug administration is adopted in high endemic areas, while selective chemotherapy is used in moderately endemic areas, targeting those ingesting raw freshwater fish. Environmental modification to block feces contamination and education to promote behavioral change on raw-eating habits are needed to increase the effectiveness and sustainability of control.



KEY FACTS:

The *C. sinensis* genome has a size of 550–560 Mb, containing 13 000–15 000 protein-coding genes.

Geographical distribution varies highly due to the different distribution of first intermediate hosts and habits in ingesting raw freshwater fish.

Piscivorous animals, especially dogs and cats, participate in the circulation, increasing the challenge to block transmission.

The practice of ingesting raw freshwater fish in children is highly influenced by their parents' practice.

Excretory–secretory products play important roles in pathogenesis and carcinogenesis and they could be used as markers in serological diagnosis.

DISEASE FACTS:

Adult worms can live in human bodies for up to 20 years and thus cause persistent damage. Morbidity is related to worm burden, indicated by eggs in feces.

High infection and intensity as well as subsequent morbidity are presented in adults, especially men, due to frequent ingestion of raw freshwater fish.

Infection is carcinogenic, causing ~5000 human cholangiocarcinoma cases annually.

Detection of eggs in feces is the gold standard for diagnosis. Immunological tests, imaging techniques, and molecular methods are also useful.

Praziquantel is recommended for both individual treatment and population chemotherapy, while albendazole is also efficacious as an alternative.

TAXONOMY AND CLASSIFICATION:

PHYLUM: Platyhelminthes
CLASS: Trematoda
ORDER: Opisthorchiida
FAMILY: Opisthorchiidae
GENUS: *Clonorchis*
SPECIES: *C. sinensis*

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We modified published figures from Qian *et al.* [5].

Declaration of interests

The authors declare no competing interests.

Resources

www.cdc.gov/dpdx/clonorchiasis/index.html
www.who.int/health-topics/foodborne-trematode-infections#tab=tab_1
https://parasite.wormbase.org/species.html

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  https://doi.org/10.1016/j.pt.2021.05.011

Trends in Parasitology | Parasite of the Month

Cryptosporidium parvum

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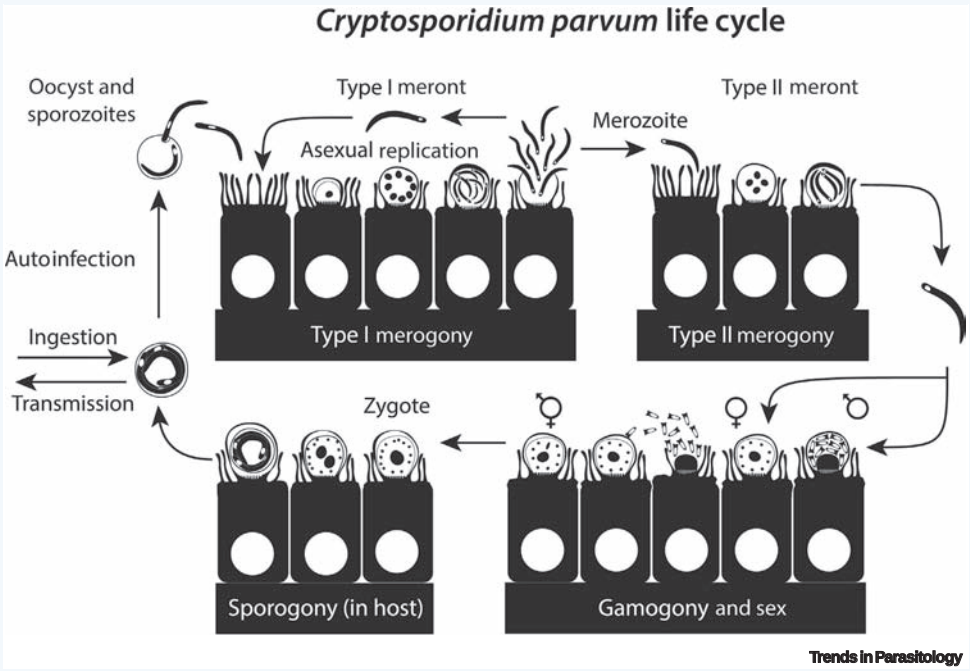
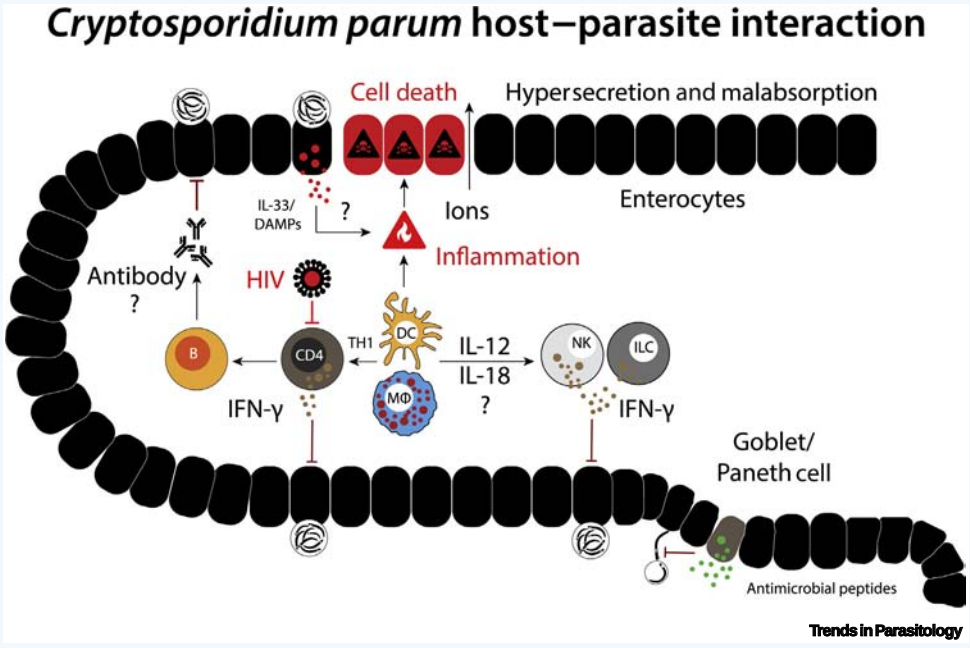


Cryptosporidium is a leading cause of diarrheal disease in young children and untreated AIDS patients in resource-limited countries worldwide. Transmission occurs via the fecal–oral route, and sources of *Cryptosporidium* infection include contaminated water or food, or contact with infected people or animals. Upon ingestion of the infective parasite oocysts, motile sporozoites emerge and invade epithelial cells of the small intestine where they develop in an intracellular but extracytoplasmic niche. *Cryptosporidium* completes its complex life cycle in a single host, with both asexual and sexual stages present in the intestine. Replication of the parasite, and the resulting immune response contribute to the development of severe, watery diarrhea in infected individuals. Currently, there is no vaccine, and only one drug (nitazoxanide), which has limited efficacy in those most susceptible.



KEY FACTS:

Human infections are caused by *C. parvum* and *Cryptosporidium hominis* but transmission of multiple additional species occurs locally.

Zoonotic *C. parvum* strains appear genetically distinct from anthroponotic strains.

C. parvum invasive stages resemble those of other apicomplexans, but invasion and intracellular development show important differences.

C. parvum has a minute genome (9.1 Mbp encoding 4020 genes), lacks an apicoplast and mitochondrial DNA, has greatly reduced metabolic capabilities, and relies on host metabolism.

Recent advances: genetic engineering, cryopreservation, culture in organoids, tractable life cycle, phenotypic screens delivered potent drug leads, a natural mouse model to study protective immunity.

DISEASE FACTS:

Cryptosporidiosis is a major cause of global child mortality, particularly under the age of two.

With advanced water treatment, outbreaks are still frequent due to oocyst resistance to water chlorination.

The main disease symptoms are severe watery diarrhea, nausea, vomiting, and wasting.

Chronic infection causes villus blunting, nutrient malabsorption, and stunted growth.

Infection results in protective immunity, albeit not sterile and not in a single infection. T cells are required to clear the infection, and interferon-γ is a key mediator of parasite restriction.

TAXONOMY AND CLASSIFICATION:

SUPERPHYLUM: Alveolata
PHYLUM: Apicomplexa
CLASS: Conoidasida
ORDER: Cryptogregarinorida
FAMILY: Cryptosporidiidae
GENUS: *Cryptosporidium*
SPECIES: *C. parvum*

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Resources

www.cdc.gov/parasites/crypto/index.html
https://cryptodb.org/cryptodb/

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Trends in Parasitology | Vector of the Month

Culicoides imicola (Biting Midge)

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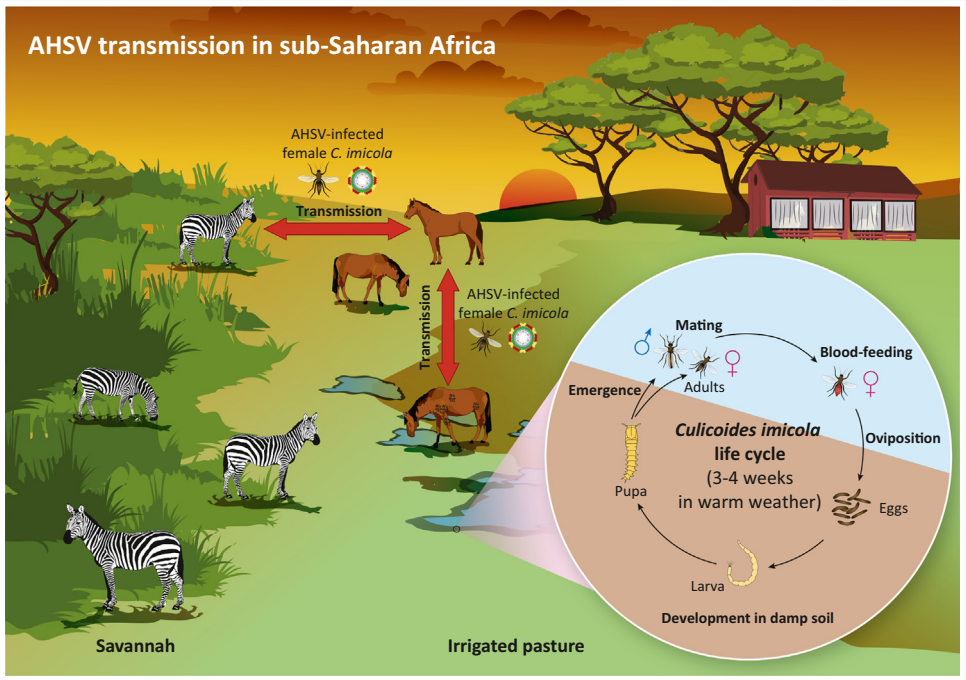
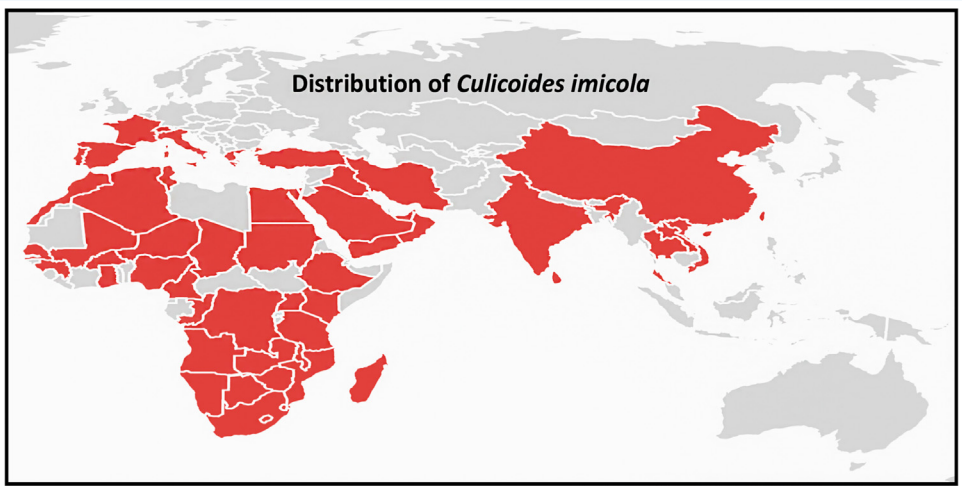


The biting midge *Culicoides imicola* is a small, haematophagous fly that plays a key role in the biological transmission of viral diseases including bluetongue and African horse sickness (AHS) that inflict damage on both subsistence and intensive livestock as well as companion animals and wildlife. Within the subgenus *Avaritia*, the monophyletic Imicola group includes ten species, although cryptic diversity may hide the existence of additional species closely related to *C. imicola*. *C. imicola* has the broadest geographic range in the genus *Culicoides*, including most of the African continent, the Mediterranean basin and parts of southern Europe, the Middle East, India, and Southeast Asia to Taiwan. The broad distribution of *C. imicola* illustrates exploitation of anthropogenic habitats and may allow future expansion into new areas. Long-distance movement of adults, especially over the sea, has been proposed and may facilitate this geographic expansion.



TRANSMISSION FACTS:
C. imicola is the primary vector of arboviruses within the families Reoviridae and Peribunyaviridae, including bluetongue virus (BTV) and AHS virus (AHSV).

Only females are haematophagous, ingesting viruses from viraemic hosts and transmitting them to uninfected hosts following a temperature-dependent extrinsic incubation period.

C. imicola has a broad mammophilic host range, including wildlife species, with the greatest abundance in proximity to domesticated large mammals, particularly species in the Bovidae and Equidae families.

CONTROL FACTS:
A range of control measures for adult and immature life stages have been defined based on the impact on *C. imicola* populations or host–vector contact, but no quantitative evidence has linked these techniques directly to a reduction in arbovirus transmission.

Mitigation techniques can significantly reduce host–vector contact, such as stabling of susceptible animals at night in completely enclosed buildings with insecticide-treated mesh, or host treatment with insecticides and/or repellents.

Control of *C. imicola*-borne arboviruses remains highly dependent on vaccines that are often too expensive to develop, unaffordable in a subsistence context, or rely on technologies with known limitations.

TAXONOMY AND CLASSIFICATION:
PHYLUM: Arthropoda
CLASS: Insecta
ORDER: Diptera
FAMILY: Ceratopogonidae
GENUS: *Culicoides*
SUBGENUS: *Avaritia*
SPECIES: *C. imicola* (Kieffer, 1913)

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Declaration of Interests

There are no interests to declare.

Resources

www.iikculicoides.net/taxa/imicola.html
http://avabase.cirad.fr/taxon.php?id=17&name=C.%20iraquis
www.gnatwork.ac.uk
https://figshare.com/articles/journal_contribution/VectorNet_Data_Series_3_Culicoides_Abundance_Distribution_Models_for_Europe_and_Surrounding_Regions/12932844?file=24621365
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Echinococcus granulosus sensu lato

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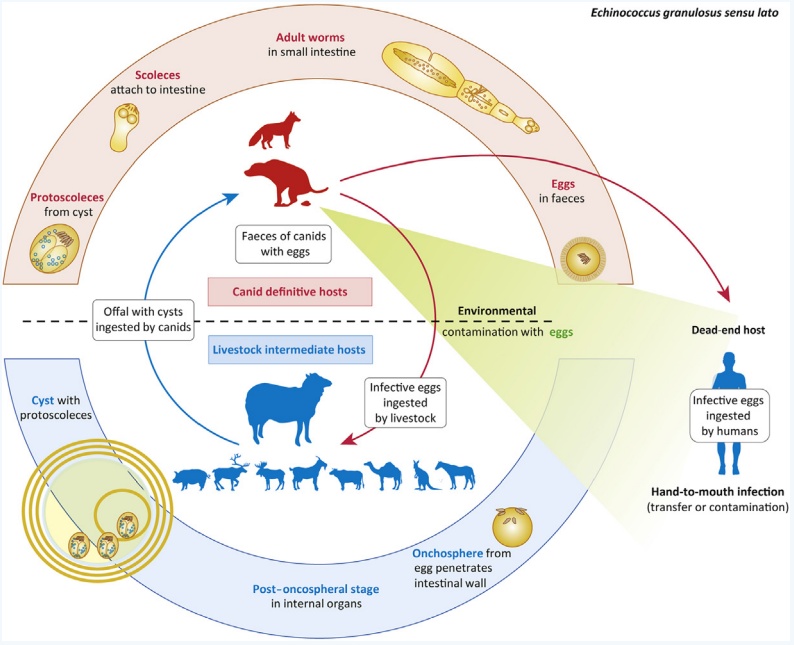
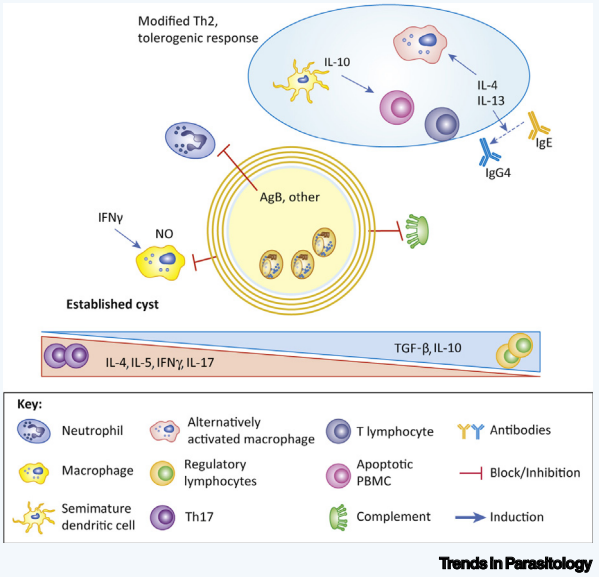
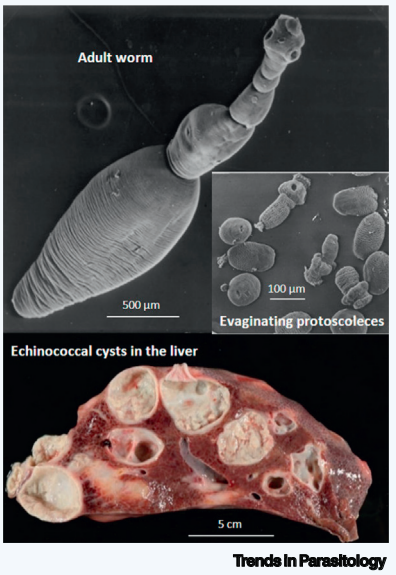


Trends in Parasitology

The larval stage of *Echinococcus granulosus sensu lato* (*sl*) causes cystic echinococcosis (CE), a neglected and chronic parasitic zoonotic disease infecting over an estimated one million people worldwide. *E. granulosus sl* is a species complex with uncertain taxonomic status, including several cryptic species (genotypes): *E. granulosus sensu stricto* (*ss*) (G1/3); *E. canadensis* cluster [G6/7, G8, G10, proposed to be split into *E. intermedius* (G6/7), *E. borealis* (G8), and *E. canadensis* (G10)]; *E. ortleppi* (G4); *E. equinus* (G5); and *E. felidis*. Canids, in particular dogs, are definitive hosts for the sexual stage adult worms, while livestock, predominantly sheep, are intermediate hosts for the asexual larval stage metacestodes. Adult worms induce no evident pathology in canids, but metacestodes moderately reduce livestock productivity. Humans are accidental dead-end intermediate hosts who are infected by the ingestion of eggs shed in the feces of infected canids.



Trends in Parasitology



Trends in Parasitology

KEY FACTS:
CE is most prevalent in poor pastoral communities in close contact with competent hosts.

E. granulosus ss (G1/3) causes the majority of human infections, followed by *E. canadensis* (G6/7).

CE is 100% preventable, and the WHO advocates concerted efforts for its control.

Control measures aim to interrupt the transmission cycle by dog deworming with praziquantel, abattoir control, culling of aged sheep, and lamb vaccination with the EG95 vaccine.

Most frequent parasite transmission pathways to humans, and risk factors for infection, are difficult to define, due to its complex life cycle and the temporal lag between infection and diagnosis.

DISEASE FACTS:
Human CE is a chronic, disabling disease mainly affecting the liver and lungs. Fluid-filled echinococcal cysts develop through different stages and grow concentrically, mainly causing compression on neighboring structures.

Imaging, in particular with ultrasound, is the reference technique for human CE diagnosis based on the visualization of pathognomonic signs, while serology is only supportive.

The management of CE envisages four clinical options, guided by cyst-stage classification as seen in imaging: surgery, percutaneous treatments, antiparasitic treatment with albendazole, and watch and wait (active surveillance by imaging).

TAXONOMY AND CLASSIFICATION:
PHYLUM: Platyhelminthes
CLASS: Cestoda
ORDER: Cyclophyllidea
FAMILY: Taeniidae
GENUS: *Echinococcus*
SPECIES: *E. granulosus sensu lato*

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Resources

www.who.int/echinococcosis/en/
www.cdc.gov/parasites/echinococcosis/index.html
www.heracles-fp7.eu/index.html
www.who.int/echinococcosis/resources/WHO_HTM_NTD_NZD_2017.01/en/

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  https://doi.org/10.1016/j.pt.2019.05.006

Echinococcus multilocularis

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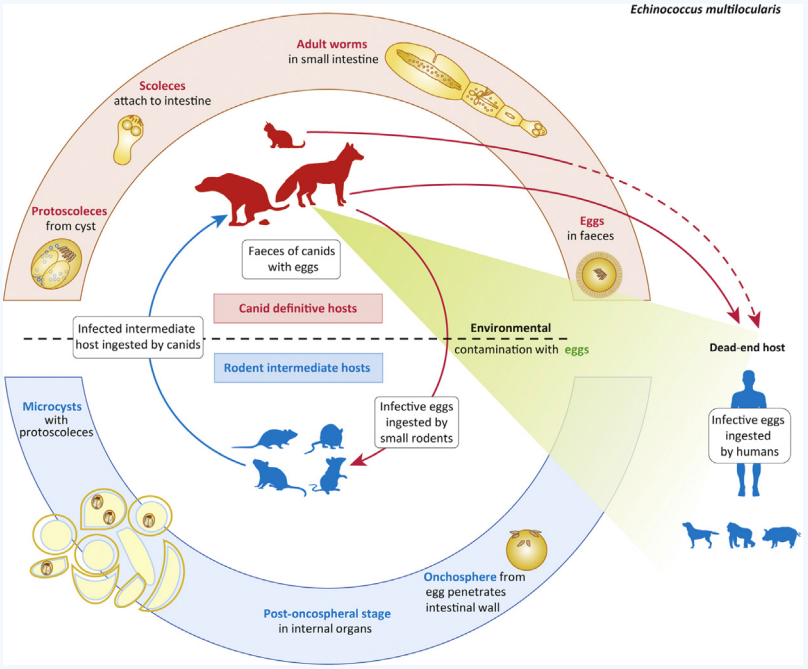
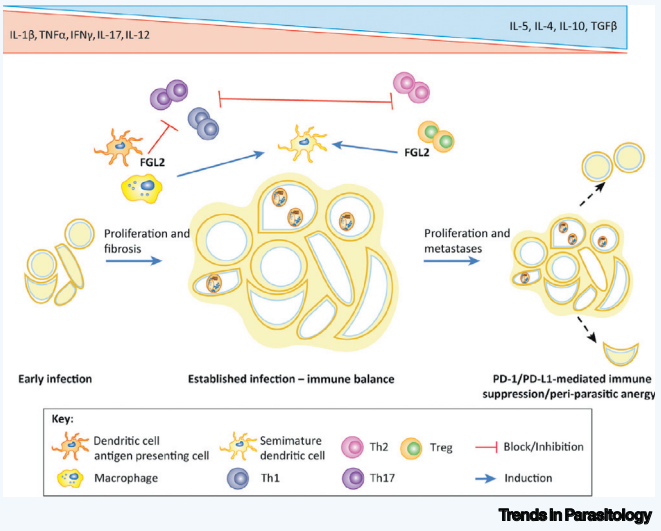


The larval stage of *Echinococcus multilocularis* is the etiological agent of alveolar echinococcosis (AE), a parasitic zoonotic disease distributed in the Northern hemisphere, with an estimated 17 400 new infections/year, most of which occur in China. The life cycle of *E. multilocularis* involves small rodent intermediate hosts, such as arvicolids and, depending on the epidemiological settings, wild or domestic canid definitive hosts, such as red or arctic foxes, jackals, wolves, or dogs. Humans are aberrant intermediate hosts acquiring the infection through ingestion of eggs shed in the feces of definitive hosts. AE is a devastating clinical condition characterized by the silently progressing infiltrative proliferation of the parasite, mimicking a malignancy. AE is of increasing concern globally due to the geographical spread of the parasite, its increasing prevalence in animals from endemic areas, the absence of a vaccine, and the lack of active control measures to prevent the infection.



KEY FACTS:
Human AE is a rare, neglected zoonotic disease, prioritized by the WHO for control.

Epidemiological research suggests *E. multilocularis* expansion in some endemic areas, although greater awareness and improved diagnostic tools may have contributed to increased detection of animal and human infections.

Risk factors for infection and parasite transmission pathways to humans are difficult to define due to the complex life cycle of the pathogen and the temporal lag between infection and onset of symptoms.

Control measures are logistically challenging due to the presence of wildlife hosts, and they focus mainly on baiting definitive hosts with anthelmintic praziquantel.

DISEASE FACTS:
AE primarily affects the liver and behaves like an infiltrative and eventually metastasizing tumor, with a high fatality rate if untreated. Immune suppression dramatically exacerbates disease progression.

The gold standard for AE diagnosis is the identification of parasite structures/genome in samples obtained invasively; imaging techniques demonstrate characteristic features of the lesions, while serology is complementary.

Human AE can be cured by radical surgery. However, many patients are no longer candidates for radical surgery due to late diagnosis. Infection in these cases is managed by life-long albendazole therapy.

TAXONOMY AND CLASSIFICATION:
PHYLUM: Platyhelminthes
CLASS: Cestoda
ORDER: Cyclophyllidea
FAMILY: Taeniidae
GENUS: *Echinococcus*
SPECIES: *E. multilocularis*

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Resources

www.who.int/echinococcosis/en/
www.cdc.gov/parasites/echinococcosis/index.html
www.who.int/echinococcosis/resources/WHO_HTM_NTD_NZD_2017.01/en/

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  https://doi.org/10.1016/j.pt.2019.05.005

Entamoeba histolytica

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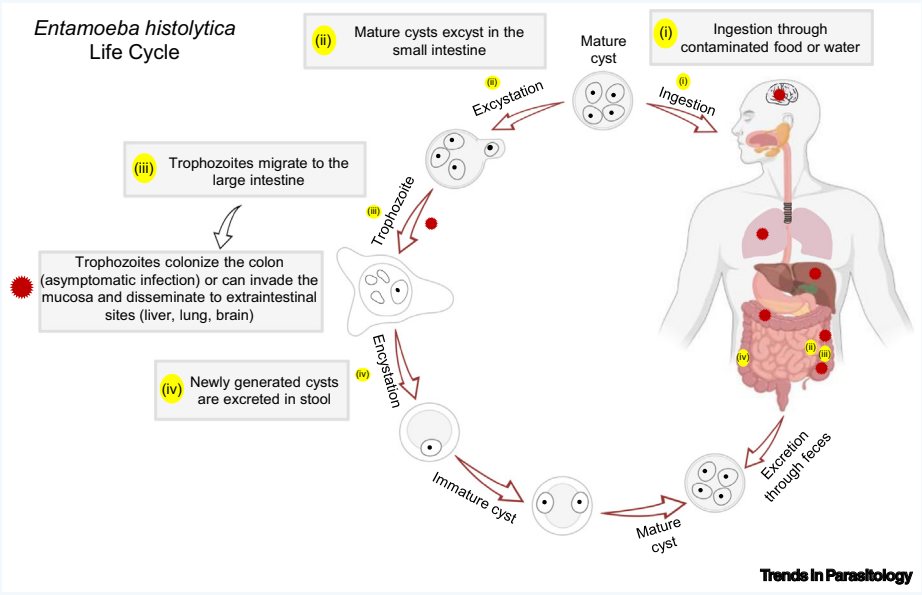
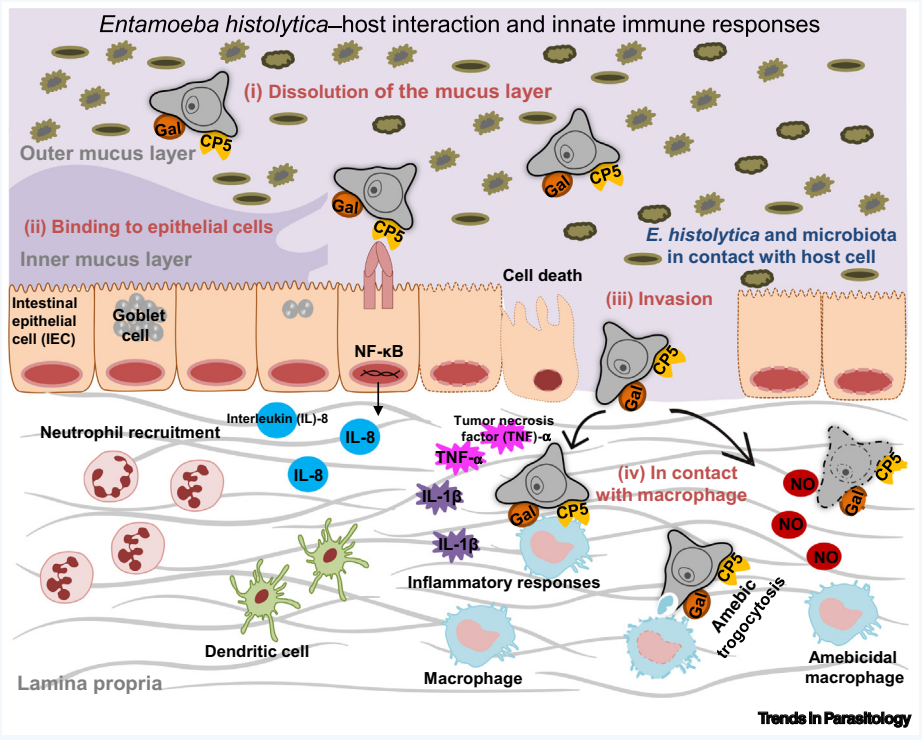


Entamoeba histolytica is a human enteric protozoan parasite responsible for the disease amebiasis. Infection starts through the ingestion of *E. histolytica* cysts contaminating food or water. The vast majority (90%) of infected individuals are asymptomatic carriers in which the parasite resides firmly within the lumen of the colon and excrete cysts in stool to complete the direct life cycle. In 10% of infected cases (symptomatic), *E. histolytica* breaches the intestinal mucosa and invades the underlying lamina propria where it interacts with host immune cells, triggering a raging proinflammatory cytokine response resulting in tissue damage and the hallmark amebic 'flask-shaped ulcer' formation. It is not known why a large proportion of *E. histolytica*-colonized individuals do not progress to invasive disease. Several parasite virulence factors along with host genetics, microbiota, and immune responses, are likely to drive the complex pathogenesis.



KEY FACTS:

Friedrick Lösch discovered *E. histolytica* in 1873 and was the first to describe the relationship between the parasite and disease, although reports of bloody diarrhea, consistent with amebiasis, date back to 1000 BC.

E. histolytica is morphologically identical to the noninfectious *Entamoeba dispar* which lacks key cysteine proteinases that are important for pathogenesis.

E. histolytica exists as an infectious cyst with four nuclei. Upon ingestion, it excysts in the terminal ileum to give rise to disease-causing motile trophozoites through its pseudopodium.

Virulence factors allow the parasite to colonize colonic mucus and to overcome innate epithelial barrier function.

DISEASE FACTS:

E. histolytica is prevalent in developing countries due to poor sanitation and hygiene but it has also emerged globally from returning travelers and is spread by sexual transmission through oral–anal contact.

~100 million cases occur annually. In 2013, 11 300 deaths from amebiasis ranked it the fourth leading cause of parasitic death.

Infection can lead to mild to bloody diarrhea, cramping and fever, abdominal pain, and weight loss. If left untreated, it may contribute to extraintestinal amebiasis, mostly liver abscess.

Stool sample analysis for cysts or trophozoites, PCR, ELISA, and serological evaluation for diagnosis.

Nitroimidazoles: metronidazole, are the mainstay drug therapy for invasive amebiasis.

TAXONOMY AND CLASSIFICATION:

PHYLUM: Amoebozoa
CLASS: Lobosea
ORDER: Amoebida
FAMILY: Entamoebidae
GENUS: *Entamoeba*
SPECIES: *E. histolytica*

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Declaration of Interests

The authors declare no competing interests.

Resources

https://amoebadb.org/
www.who.int/ith/diseases/amoebiasis/en/
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Glossina palpalis gambiensis (Tsetse Fly)

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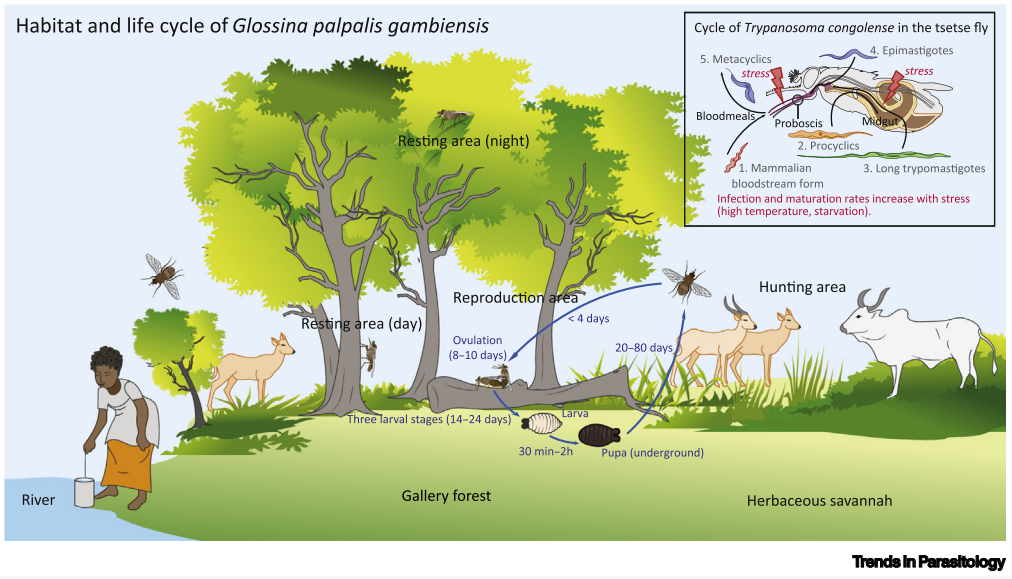
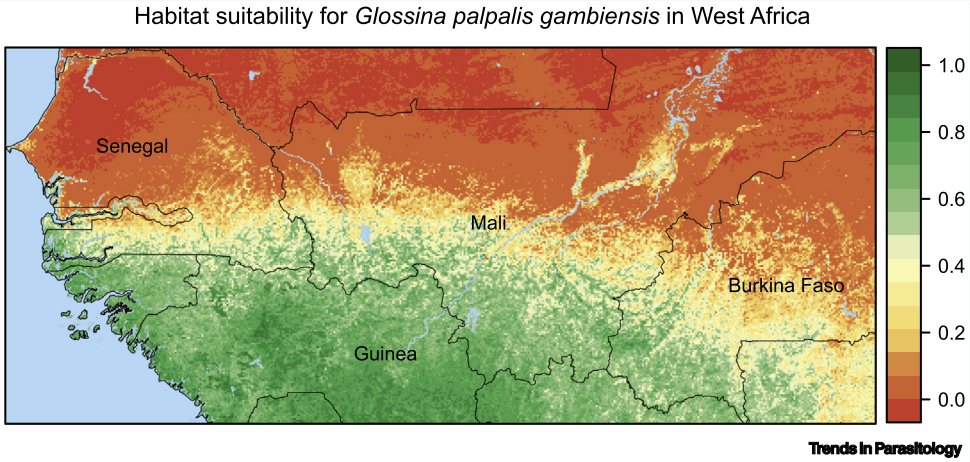


Glossina palpalis gambiensis is a riverine tsetse species endemic in West Africa and thriving in riparian vegetation of the savannah areas from Burkina Faso and Mali to Guinea and Senegal. It is a major vector of human and animal trypanosomosis (sleeping sickness and nagana, respectively) in that region. *G. p. gambiensis* is an opportunistic species, feeding on a wide range of hosts from reptiles to pigs and cattle, with humans as one of its preferred hosts. Like most tsetse species, it has a narrow range of acceptable temperature and humidity, a low reproduction rate, and is thus very sensitive to climate change but can adapt to human modification of its environment and survive in polluted and densely populated areas. Its presence in the Niayes area of Senegal, where rainfall is below 500 mm a year, and in the Parc de Hahn of Dakar reveals an extraordinary plasticity. In the Niayes area it is presently targeted by an eradication program, including a sterile insect technique component.



TRANSMISSION FACTS:
G. p. gambiensis can transmit all *Trypanosoma* spp., particularly *Trypanosoma brucei gambiense* in humans and *Trypanosoma vivax* in cattle.

It picks up the bloodstream form of trypanosomes from a host and injects the metacyclic form into the skin of another host after an extrinsic cycle of 10–30 days depending on the parasite species.

It is a day-biter, with a peak of activity conditioned by temperature. Its distribution, density, lifespan and infection rate are also temperature-dependent.

Its learning capability increases the hunting efficiency of older flies, that is, the host selected for the first bloodmeal can influence host selection for the second meal.

CONTROL FACTS:
Conventional control relies on insecticide-baited traps. Cattle are generally treated with pyrethroid pour-ons.

In Guinea, its control was instrumental in reducing the incidence of sleeping sickness.

Its ecology and preferred habitats (riverine and dense forest vegetation) partially protect it against sequential aerosol spraying of insecticides that is more efficient against savannah tsetse species.

It was eradicated from a 3000 km^2 area in Burkina Faso (Sidéradougou) in the 1980s using an integrated strategy including the sterile insect technique, but the cleared area was then reinvaded because the target area was not isolated.

TAXONOMY AND CLASSIFICATION
PHYLUM: Arthropoda
CLASS: Insecta
ORDER: Diptera
FAMILY: Glossinidae
GENUS: *Glossina*
SPECIES: *G. palpalis gambiensis* (Vanderplank 1949)

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Resources

www.fao.org/paat/resources/atlases/tsetse-and-aat/en/
https://books.openedition.org/irdeditions/10532?lang=fr
www.anipedia.org/resources/vectors-tsetse-flies/1109

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Trends in Parasitology | Parasite of the Month

Hepatocystis

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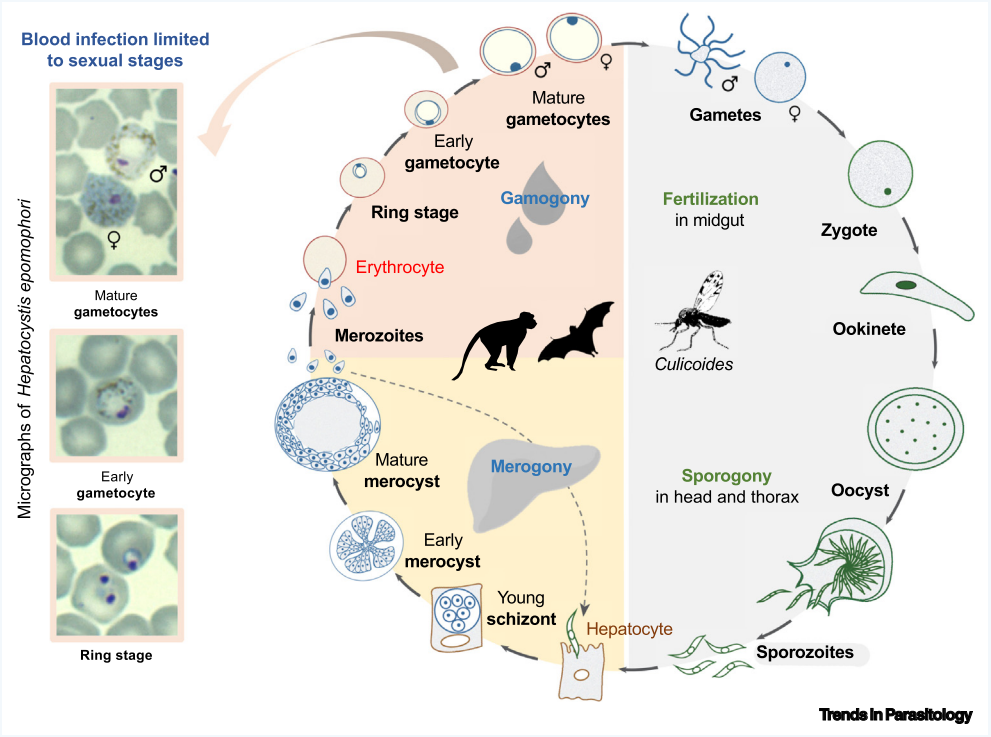
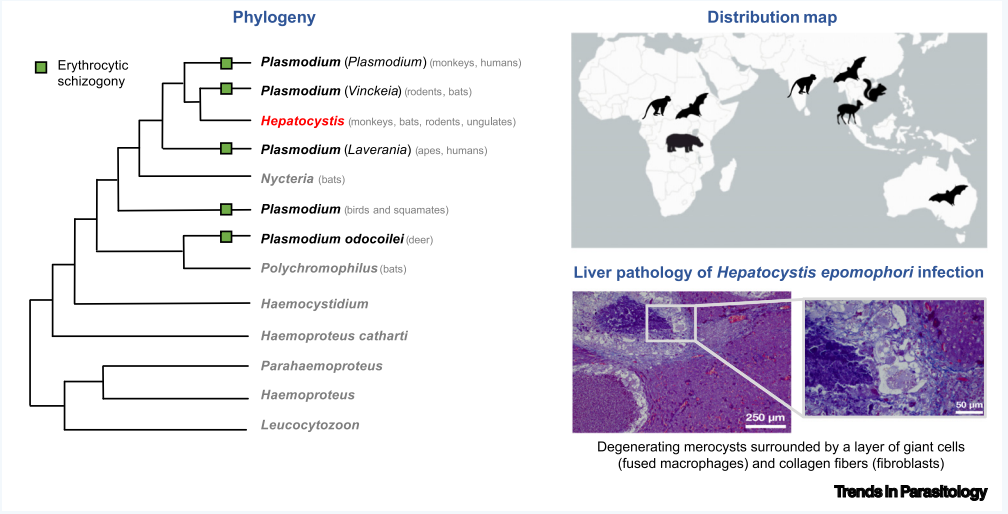


Hepatocystis parasites are the closest relatives of *Plasmodium* species of mammals. They infect monkeys, bats, squirrels, and ungulates in Africa, Asia, and Australia. A prevalence of up to 100% has been documented in fruit bats and monkeys. Twenty-five morphospecies have been described, and cross-species transmission, divergent *Hepatocystis* lineages, and species complexes are reported in primate and bat hosts. Biting midges (*Culicoides*) are the only known vectors. In the vertebrate, merogony occurs exclusively in the liver, resulting in formation of macroscopic merocysts. Merozoites invade erythrocytes and transform directly into sexual gametocytes, thereby omitting asexual replication and associated health conditions. Gametocytes can persist for several weeks and fertilize after a bloodmeal in the *Culicoides* midgut. The *Hepatocystis* genome features unique gene families, a low number of *Plasmodium* interspersed repeat (*pir*) genes, and an absence of the reticulocyte-binding protein family.



KEY FACTS::
Hepatocystis is phylogenetically nested within (paraphyletic) mammalian *Plasmodium* clades.

Vector *Culicoides adersi* has been confirmed for primate-infecting species *Hepatocystis kochi*; experimental infection of *Culicoides nubeculosus* was reported with *Hepatocystis* of Australian bats.

Liver merocysts differ in size and structure among *Hepatocystis* species and mature within 2 months.

Merozoites are discharged into sinusoids, while a small proportion likely reinvades hepatocytes for a secondary merogony.

Ookinetes penetrate the midgut and transform to oocysts in the thorax and head of *Culicoides*. Sporozoites accumulate in the hemocoel.

Three genomes in the nucleus (19.95 Mb encoding 5341 genes), mitochondrion (6.6 kb), and apicoplast (27 kb).

Adaptations to the *Culicoides* vector are recognizable in the genome/transcriptome.

DISEASE FACTS::
Infections rarely cause disease, but anemia has been documented.

Cyclical fevers do not occur as blood stages are limited to gametocytes that do not provoke cytokine responses.

Liver merocyst maturation leads to cellular infiltration and inflammation, followed by granuloma formation.

Hemozoin pigment is deposited in liver and spleen.

Identification of apparent selection of resistance alleles in the promoter region of the Duffy blood group antigen/chemokine receptor DARC in *H. kochi*-infected monkeys.

TAXONOMY AND CLASSIFICATION:
PHYLUM: Apicomplexa
CLASS: Aconoidasida
ORDER: Haemosporida
FAMILY: Plasmodiidae/ Haemoproteidae
GENUS: *Hepatocystis*

  https://doi.org/10.1016/j.pt.2020.07.015

Acknowledgments

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www.ebi.ac.uk/ena
https://github.com/adamjamesreid/hepatocystis-genome

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Leishmania infantum

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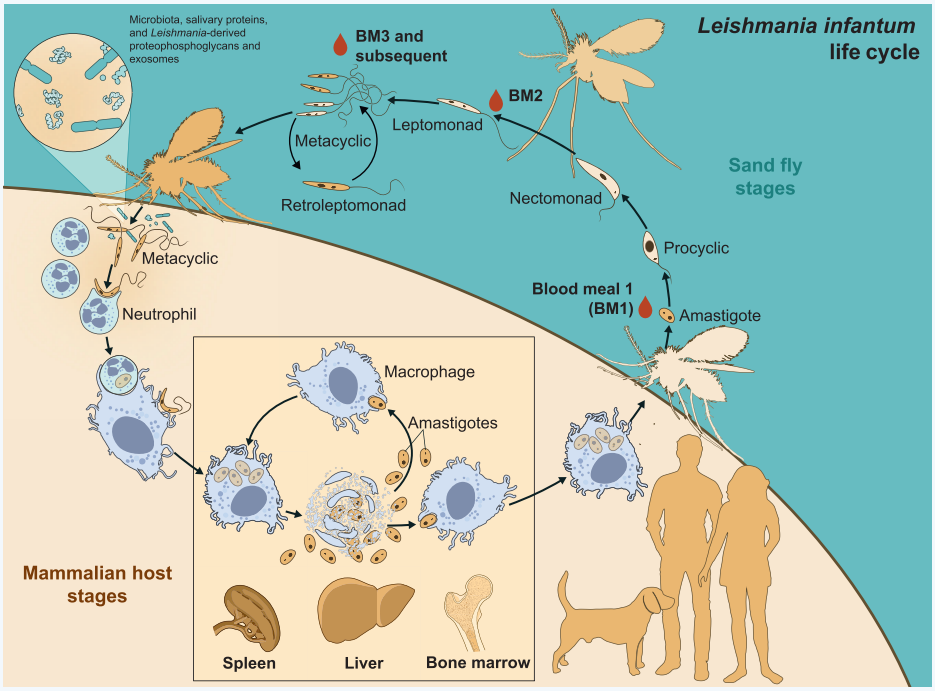


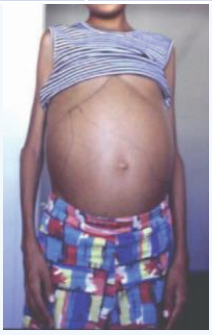
Trends in Parasitology

Leishmania infantum is the etiological agent of visceral leishmaniasis (VL) in South America, the Mediterranean basin, and West and Central Asia. It can also cause cutaneous lesions, particularly in the Mediterranean. The most affected country, Brazil, reported 4297 VL cases in 2017. *L. infantum* is transmitted by female phlebotomine sand flies during successive blood meals. After being picked up by the insect vector during a bite on a reservoir host, the parasites become extracellular and undergo a series of morphological changes culminating in the rise of the metacyclic infective stage, which is then inoculated into the host skin during a blood meal. Metacyclic parasites will infect multi- and mononuclear cells in the host and become intracellular amastigotes. Parasite visceralization results in impaired function of the liver, spleen, and bone marrow with fatal consequences in many cases. To date, no human vaccine is available, and therapeutic drugs, some with severe side effects, are used to achieve a clinical curative response.

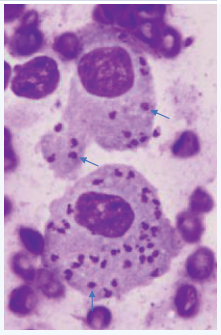
Human immune responses to *Leishmania infantum* infection			
Asymptomatic	Presence of specific anti-*Leishmania* antibodies	Positive in skin test	Respond to *Leishmania* antigen *in vitro* (Il-2, IFNγ, and Il-12)
Diseased	Hypergammaglobulinemia	Negative in skin test	Lack of response to *Leishmania* antigens *in vitro*
Cured	Long-term specific anti-*Leishmania* antibodies	Positive in skin test	Recovery of cellular immunity to *Leishmania* antigens *in vitro*

Blood fed sand fly vector *Lutzomyia longipalpis*

Scanning electron micrograph of *L. infantum* metacyclic promastigote



Chronic case of visceral leishmaniasis in infant in Northeast Brazil - rare occurrence nowadays



Giemsa staining of macrophages harboring several *L. infantum* amastigotes (arrows)

Trends in Parasitology

KEY FACTS:

L. infantum was introduced into the New World during Portuguese and Spanish colonization; it was formerly named *Leishmania chagasi*.

The primary hosts are humans and canids. Domestic dogs are the main parasite reservoir. Wild hares serve as sylvatic reservoirs in southern Europe. It is controversial whether humans act as reservoirs.

The main vectors are: *Lutzomyia longipalpis* in Brazil; *Phlebotomus perniciosus* in Spain, Portugal, and Italy; and *Phlebotomus ariasi* in France and Portugal.

During a blood meal, parasites are inoculated into the host skin together with sand fly gut microbiota and salivary proteins, as well as *Leishmania*-derived proteophosphoglycans and exosomes.

Multiple blood meals (also uninfected) are critical for successful sand fly infection and parasite development to the infective stage.

Genetic markers are associated with drug resistance.

The genome consists of 36 chromosomes, ~32 Mb.

DISEASE FACTS:

Disease is caused by amastigote forms of the parasite living in macrophages.

It mainly affects children under 10 years of age and immunocompromised adults.

The most severe cases occur in South America.

Symptoms include persistent fever and hepatosplenomegaly. Cutaneous lesions are an uncommon manifestation of infection.

Poor nutritional status is associated with disease progression.

TAXONOMY AND CLASSIFICATION:

KINGDOM: Protozoa
PHYLUM: Euglenozoa
CLASS: Kinetoplastea
ORDER: Kinetoplastida
FAMILY: Trypanosomatidae
GENUS: *Leishmania*
SUBGENUS: *Leishmania*
SPECIES: *L. infantum*

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Resources

www.who.int/leishmaniasis/en/
www.cdc.gov/parasites/leishmaniasis/
www.paho.org/hq/index.php?option=com_topics&view=article&id=29&Itemid=40754&lang=en
www.dndi.org/diseases-projects/leishmaniasis/
https://tritrypdb.org/tritrypdb/

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Leishmania Viannia guyanensis

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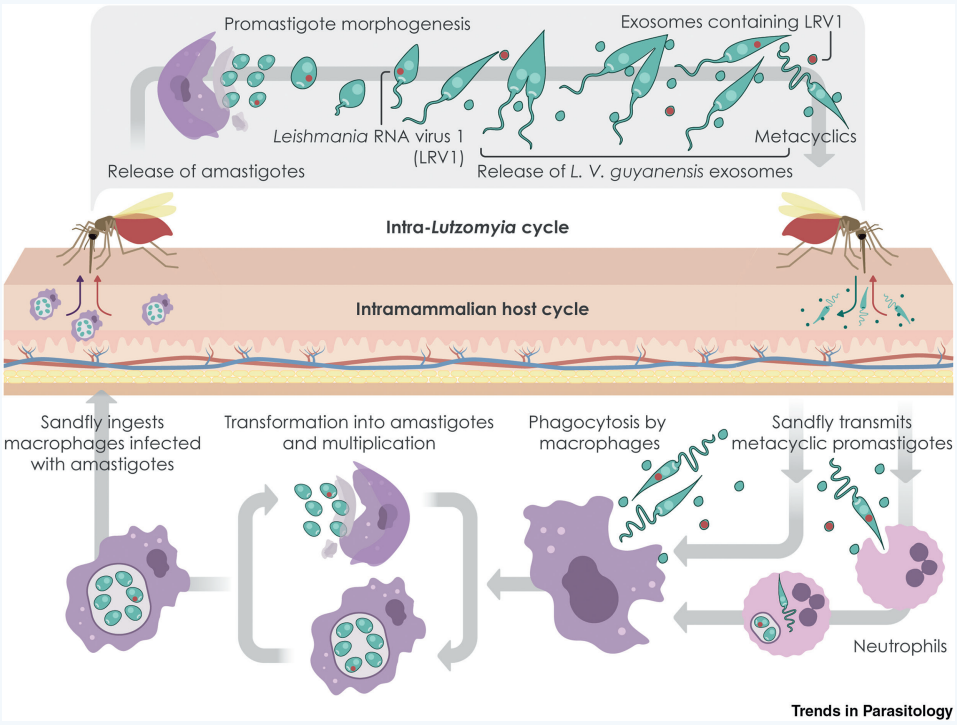
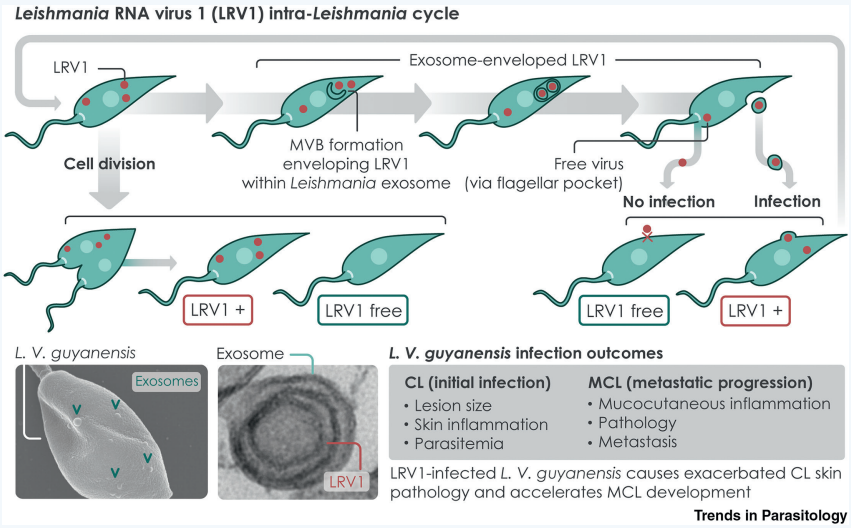


Leishmania of the Viannia subgenus, including *Leishmania Viannia guyanensis*, is the agent responsible for cutaneous and mucocutaneous leishmaniasis (CL and MCL) in the Americas from the USA to Argentina. 48 000 new cases of Cl and MCL are reported yearly, among which 1/10 are associated to *L. V. guyanensis* infection transmitted by female *Lutzomyia* sandflies during the blood meal. Inoculated metacyclic promastigotes, coupled with *Leishmania* exosomes, will infect various inflammatory cells at inoculation sites, where they rapidly transform into amastigotes. Parasites divide and progress in the intramacrophage form, leading to an initial CL skin ulceration. Depending on the inoculation site and host health condition, parasites may metastasize to the nasopharyngeal tissues within a few months. *L. V. guyanensis* is occasionally infected with *Leishmania* RNA virus 1 (LRV1) that can be enveloped by exosomes and is believed to accelerate MCL development.



KEY FACTS:

L. V. guyanensis infection is mainly found in the amazon basin of South America, including Bolivia, Brazil, and Peru.

The primary hosts are the two-toed sloth, the lesser anteater, and the opossum.

After being inoculated into the skin by female *Lutzomyia* sandflies, themetacyclic promastigotes infect phagocytes at the injection site, leading to localized skin lesions that can evolve to MCL.

Some *guyanensis* strains are infected by LRV1 endovirus, causing a potentially more aggressive form of MCL. LRV1 is not necessary for the development of MCL skin pathology.

LRV1 is maintained in *L. V. guyanensis* by cell division but has been shown to exploit *Leishmania* exosomal pathways to exit promastigotes and infect naïve *Leishmania* from the Viannia subgenus.

DISEASE FACTS:

MCL outcomes are influenced by initial CL lesion site, size, and delayed healing.

Disfiguring MCL skin pathology, which affects the mouth and nasopharyngeal tissue, may develop anywhere from several months to 10–20 years after the CL episode.

The nose is mainly affected; however, 1/3 of infected individualsmay develop invasive lesions of the pharynx/larynx and upper lip.

Final stages of MCL can lead to major disfiguration, tissue destruction, and nasal obstruction.

While CL can heal spontaneously, MCL never heals by itself.

TAXONOMY AND CLASSIFICATION:

PHYLUM: Euglenozoa
CLASS: Kinetoplastea
ORDER: Kinetoplastida
FAMILY: Trypanosomatidae
GENUS: *Leishmania*
SUBGENUS: *Viannia*
SPECIES: *L. V. guyanensis*

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Resources

www.who.int/news-room/fact-sheets/detail/leishmaniasis
www.cdc.gov/parasites/leishmaniasis/health_professionals/index.html
www.paho.org/leishmaniasis
www.paho.org/hq/dmdocuments/2016/2016-cha-leish-epi-report-americas.pdf

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  https://doi.org/10.1016/j.pt.2019.06.008

Trends in Parasitology | Vector of the Month

Lutzomyia longipalpis (Sand Fly)

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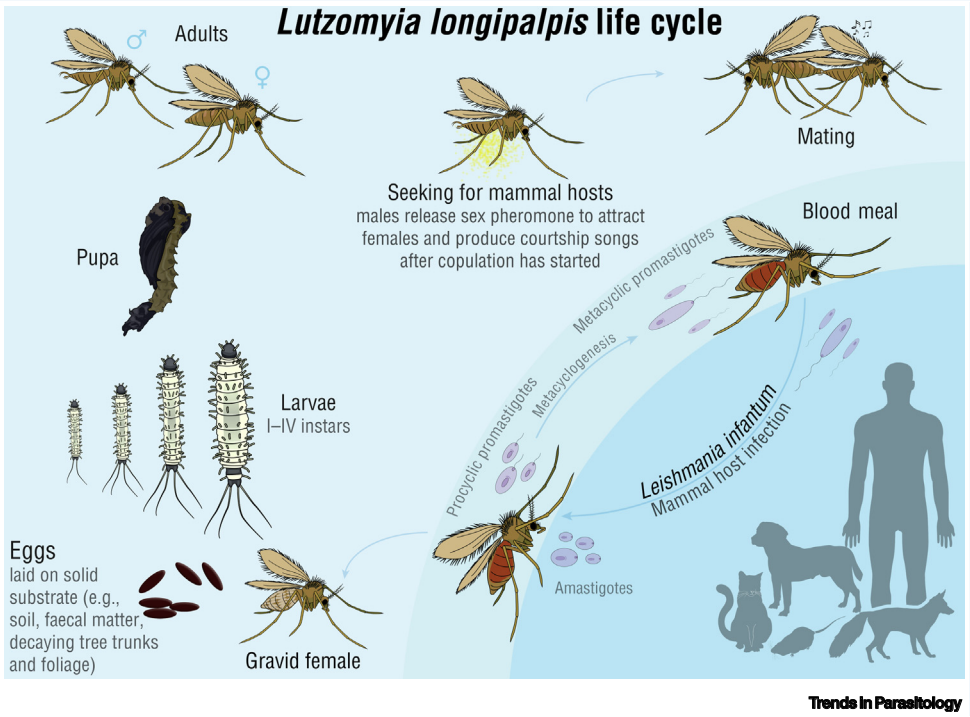
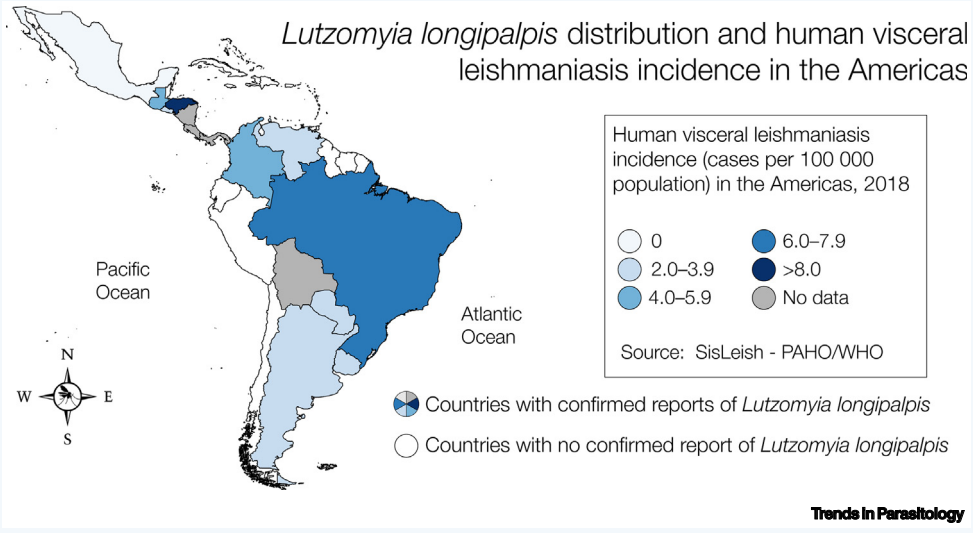


Lutzomyia longipalpis appears primarily in Central and South America and is the main vector of visceral leishmaniasis (VL) caused by *Leishmania infantum*. In Brazil, the country reporting the highest number of human VL cases in the region, this sand fly is reported in 24 of 27 states. *L. longipalpis* is adapted to human dwellings, which contributes to its spreading in rural and urban areas. Female sand flies are catholic blood feeders with remarkable anthropophilic and endophilic behaviour. The presence of dogs at home and higher dog seropositivity in nearby areas are risk factors for VL. Current control strategies target adult stages. The limited knowledge of *L. longipalpis* breeding sites, which are strictly terrestrial, is a hurdle for controlling the preimaginal stages. In addition, *L. longipalpis* composes a species complex, harbouring an uncertain number of cryptic species. Further research may reveal that some of these cryptic species are more efficient vectors of *L. infantum* than others.



TRANSMISSION FACTS:
L. longipalpis adults may rest in human houses and animal shelters during the day. The biting activity of females is crepuscular and nocturnal.

Sequential blood meals by *L. infantum*-infected *L. longipalpis* females increase infective forms in their gut, potentially augmenting their infectiousness.

L. infantum transmitted by some *L. longipalpis* populations with low amounts of maxadilan (a salivary peptide) may cause cutaneous lesions in Central America.

The sand fly promastigote secretory gel and gut microbiota are egested into host skin during the bite, playing a role in the establishment and visceralization of *Leishmania* infections.

L. longipalpis is widely used as model for experimental transmission, with high biting rate on chicken skin membranes. It is also permissive to several *Leishmania* spp. under laboratory conditions.

CONTROL FACTS:
Insecticide-treated nets and indoor residual spraying can reduce indoor transmission. Both strategies can be boosted when combined with synthetic sex-aggregation pheromones, which attracts both males and females.

Applying topical insecticides (e.g., pyrethroid-based products) on dogs can reduce their exposure to the vectors. The extended use of this strategy in Brazil has not increased *L. longipalpis* insecticide resistance.

TAXONOMY AND CLASSIFICATION:
PHYLUM: Arthropoda
CLASS: Insecta
ORDER: Diptera
FAMILY: Psychodidae
GENUS: *Lutzomyia*
SPECIES: *L. longipalpis* (Lutz and Neiva 1912)

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Resources

http://www.cvbd.org/en/sand-fly-borne-diseases/about-sand-flies/sand-fly-feeding/host-seeking-behaviour/
https://www.who.int/leishmaniasis/disease/vector/en/
https://www.troccap.com/
http://www.leishvet.org/

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Mansonella ozzardi

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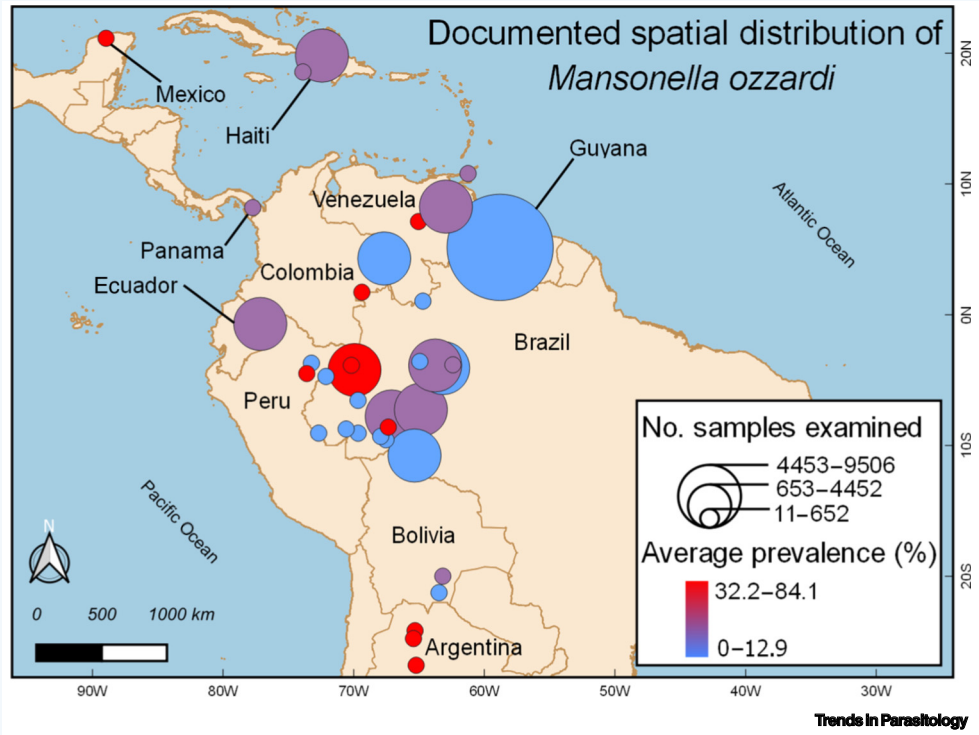
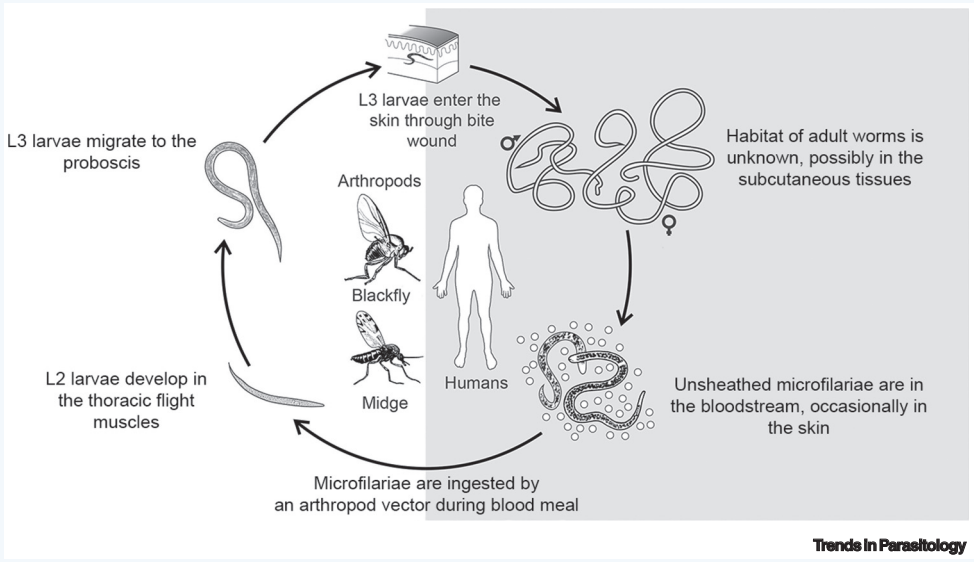


The human filarial nematode *Mansonella ozzardi* occurs widely in the Neotropical region from southern Mexico to northwestern Argentina. It causes mansonellosis and is transmitted by blackflies of the genus *Simulium* and biting midges of the family Ceratopogonidae. The embryonic unsheathed microfilariae with sharp, unnucleated tails are detectable in the blood (and occasionally in the skin) 1 day after being released by the fertilized adult female worms. After being ingested by a vector, the microfilariae reach the insect's thoracic musculature through the hemocoel and develop, after two moults, into infective L3 larvae that migrate to the head and mouth parts of the vector. Cross-sectional surveys in endemic areas show an increase in both *M. ozzardi* prevalence and microfilarial load with patients' age until they reach their 60s. Most cases of mansonellosis appear to be asymptomatic, but mild symptoms and a recently recognized ocular pathology have been associated with this infection.



KEY FACTS:

The synonymous terms mansonellosis and mansonelliasis were exclusively used for *M. ozzardi* infections until the taxonomic revisions in the 1980s included other human filarial parasites in the genus *Mansonella*.

Nuclear genome 80 Mb, with 9000 predicted genes; circular mitogenome 13.6 kb.

M. ozzardi harbors superclade F *Wolbachia* endosymbionts, with potential therapeutic implications.

Although filarial nematodes typically downregulate host immunity to limit inflammation-mediated tissue damage, such effect remains to be demonstrated for mansonellosis.

In endemic areas, infection rates are the lowest in urban areas, among women and the young, being linked to increased exposure to infectious vector bites.

DISEASE FACTS:

Most infections are asymptomatic, but fever, headache, joint pain, lower-limb chills, and ocular lesions may occur.

The overall disease burden and the visual impairment caused by ocular lesions have not yet been quantified.

Diagnosis is usually done with light microscopy, sometimes with concentration methods. PCR and loop mediated isothermal amplification (LAMP) can improve diagnostic sensitivity and specificity.

An ivermectin dose of 0.15 mg/kg clears microfilariae but not adult worms.

Future curative treatments may come from *Wolbachia*-targeting drugs currently being developed for onchocerciasis and lymphatic filariasis.

TAXONOMY AND CLASSIFICATION:

PHYLUM: Nematoda
CLASS: Chromadorea
ORDER: Rhabditida
FAMILY: Onchocercidae
GENUS: *Mansonella*
SPECIES: *M. ozzardi*

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Resources

www.ncbi.nlm.nih.gov/Taxonomy/Browser/wwwtax.cgi?mode=Info&id=122354&lvl=3&lin=f&keep=1&srchmode=1&unlock
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  https://doi.org/10.1016/j.pt.2020.03.005

Naegleria fowleri

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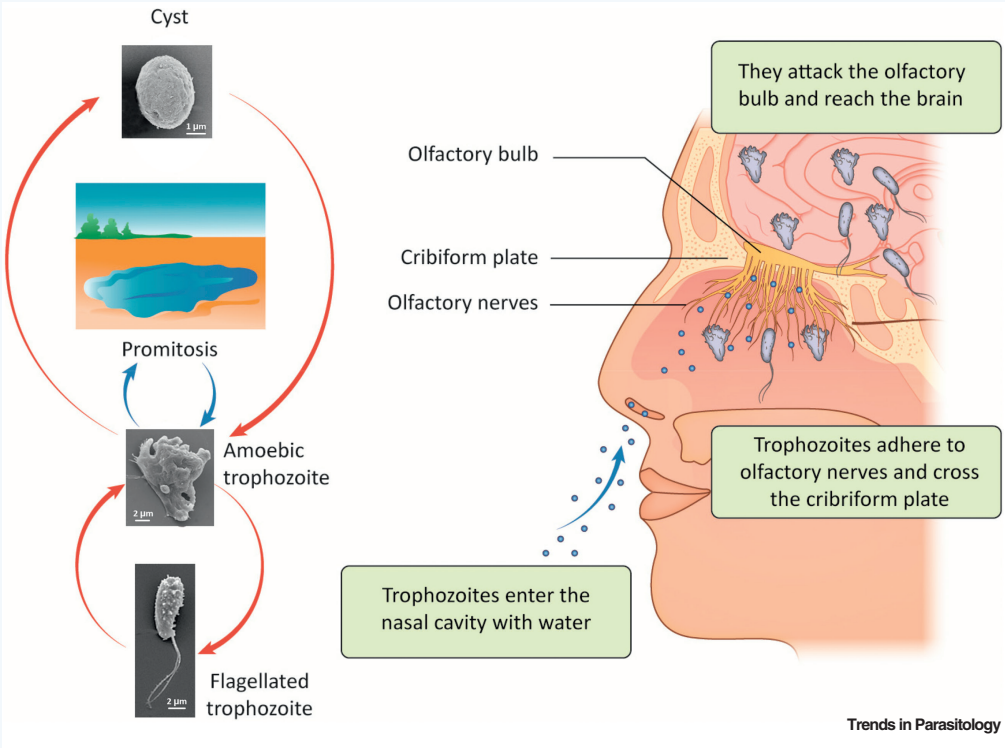
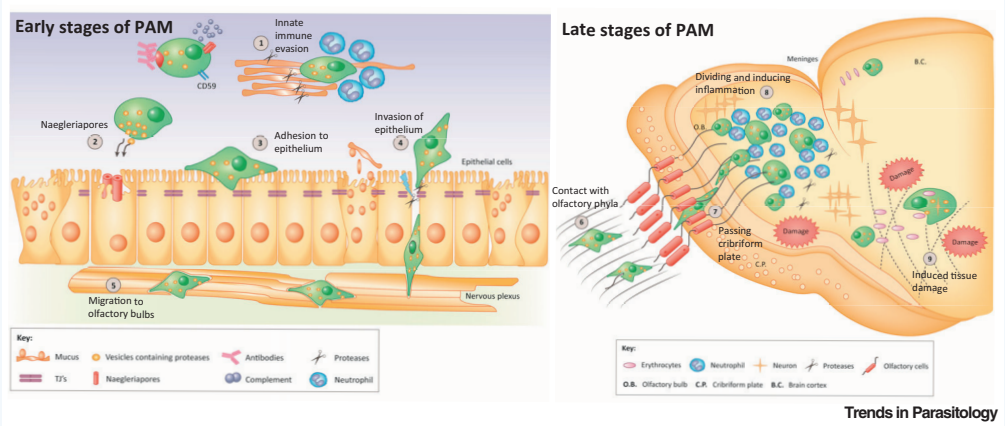


Naegleria fowleri, also known as the 'brain-eating amoeba', is a free-living amoeba capable of living in the environment, especially in bodies of warm water. Three distinct life stages exist: amoeboid trophozoite, flagellated trophozoite, and cyst. In the amoeboid trophozoite phase cell division occurs by mitosis. The sizes of the trophozoite forms range from 15 to 25 µm. Like most protists, the trophozoites form cysts under unfavorable conditions. The cysts measure between 7 and 10 µm and are covered by a thin double wall with one or two pores. It is possible that all three phases of *N. fowleri* reach or invade the human nasal mucosa, but only the trophozoite amoeboid form has been isolated in cerebrospinal fluid or tissue. It is feasible that, upon reaching the nasal area, the cyst quickly becomes a trophozoite and forms flagella before it invades the nasal and olfactory nerve tissue. Infection occurs when individuals swim or dive in *Naegleria*-contaminated warm freshwater bodies such as lakes or rivers.



KEY FACTS:

N. fowleri has been isolated from both soils and different types of water bodies, such as swimming pools, thermal waters, tap water, and lakes.

N. fowleri can cause an aggressive infection of the human central nervous system (CNS), which is called primary amoebic meningoencephalitis (PAM).

Most reported cases of PAM patients present a history of contact with N. fowleri-contaminated water bodies in the week prior to the appearance of the first symptoms.

DISEASE FACTS:

N. fowleri penetrates through the nasal passage, where it adheres to the olfactory nerves and makes its way to the brain through the cribriform plate.

The amoeba attacks the olfactory bulb, producing an inflammatory process and leading to brain infection, which is fatal in more than 97% of cases.

Clinical symptoms are characterized by the sudden onset of bifrontal or bitemporal headache, high fever, stiff neck, followed by nausea, vomiting, and irritability. In advanced stages of the infection, photophobia and neurological alterations are observed, such as lethargy, seizures, confusion, coma, diplopia, or strange behavior, which leads to death within a week.

The treatment is based on the use of amphotericin B in combination with other drugs such as rifampin, fluconazole, azithromycin, miltefosine, and dexamethasone.

TAXONOMY AND CLASSIFICATION:

PHYLUM: Percolozoa
CLASS: Heterolobosea
ORDER: Schizopyrenida
FAMILY: Vahlkampfiidae
GENUS: *Naegleria*
SPECIES: *N. fowleri*

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Resources

www.cdc.gov/dpdx/freelivingamebic/
www.cdc.gov/parasites/naegleria/general.html
www.jordansmelskifoundation.org/

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Trends in Parasitology | Parasite of the Month

Perkinsus marinus

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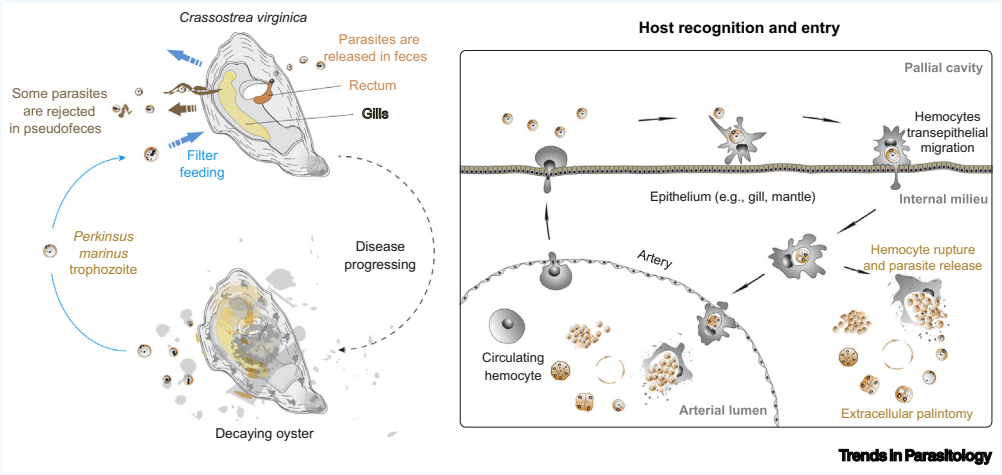
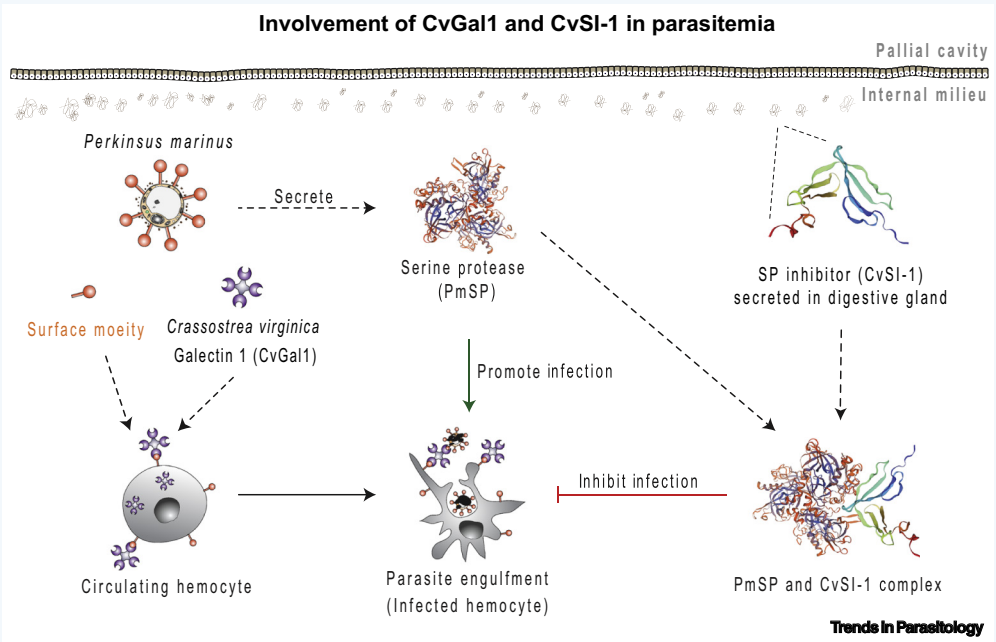


Perkinsus marinus is a facultative intracellular marine protozoan parasite responsible for the Dermo disease in *Crassostrea virginica* oysters. Associated with mass mortalities in the Gulf Coast and Chesapeake Bay (USA), it remains one of the main hurdles for oyster reef restoration and aquaculture. Oysters take up the parasite by filter-feeding; in the pallial cavity it can be phagocytosed by the hemocytes via CvGal1, gaining access to the internal milieu. Inside the parasitophorous vacuole, the parasite resists oxidative stress and acquires nutrients. Propagation strategies include binary fission, budding, palintomy, and schizogony. Although the effect on humans upon consumption of raw infected oysters has not been studied, humanized HLA-DR4 mice fed with *P. marinus* do not develop noticeable pathology but elicit systemic immunity. Parasite culture in host-free media, and the use of genetic tools, make it a tractable genetic model and a heterologous expression and vaccine-delivery system.



KEY FACTS:

P. marinus is phylogenetically close to dinoflagellates and apicomplexans.

Two genomes in the nucleus (86 Mb encoding >23 600 proteins) and mitochondrion, using mRNA *trans*-splicing with a conserved 21–22 nt spliced leader.

With a direct life cycle, trophozoites are released into the water with pseudofeces and feces, or from decaying oysters.

It remains controversial whether *P. marinus* produces zoospores as do other *Perkinsus* spp.

Continuous culture in the absence of host cells and transfection methodology enable the study of physiology, cell biology, and host–parasite interactions.

It offers a heterologous expression system for human pathogen genes (e.g., *Plasmodium falciparum*, *Toxoplasma gondii*, *Cryptosporidium parvum,* and Ebola virus).

DISEASE FACTS:

P. marinus infection is one of the World Organization of Animal Health (OIE)-listed diseases.

Once described in the Gulf of México, it is now found in both North and South America.

High water temperature is the main environmental clue associated with oyster mass mortalities.

A protease inhibitor (CvSI-1) isolated from the plasma of eastern oysters inhibits the parasite's proliferation *in vitro* and appears to be involved in the resistance to Dermo disease.

With numerous chemical inhibitors of *in vitro* propagation, treatment of diseased oysters in the natural environment remains unrealistic.

TAXONOMY AND CLASSIFICATION:
PHYLUM: Perkinsozoa
CLASS: Perkinsea
ORDER: Perkinsida
FAMILY: Perkinsidae
GENUS: *Perkinsus*
SPECIES: *P. marinus*

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National Science Foundation (NSF) 1701480 (J.A.F.R.), National Oceanic and Atmospheric Administration (NOAA) NA15NMF4270303 (J.A.F.R.), and JSPS KAKENHI – Grant-in-Aid for JSPS Fellows 19J00148 (K.U.).

Resources

www.dfo-mpo.gc.ca/science/aah-saa/diseases-maladies/pmdoy-eng.html
www.ncbi.nlm.nih.gov/genome/280
www.atcc.org
www.protocols.io

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Trends in Parasitology | Parasite of the Month

Plasmodium falciparum

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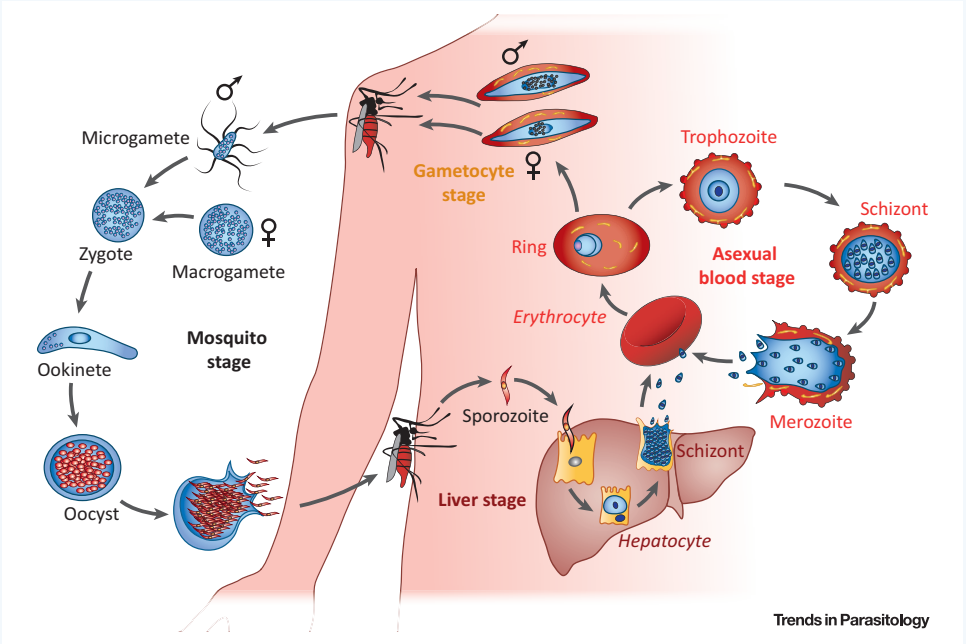
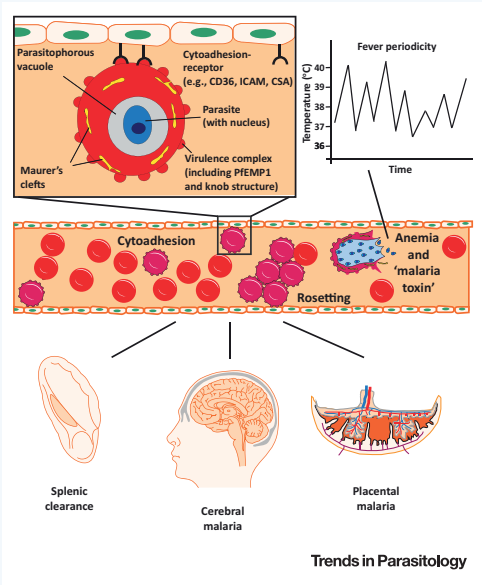
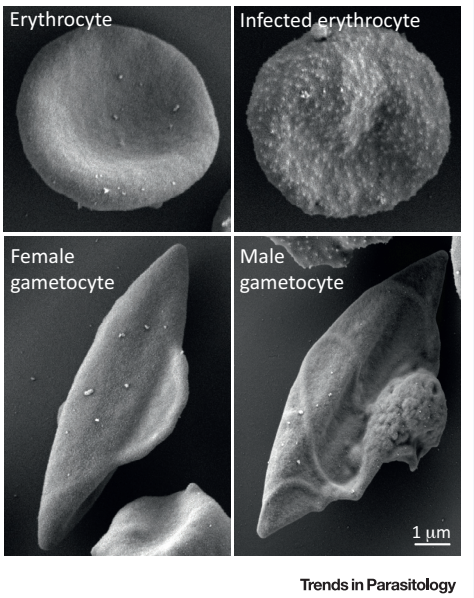


Plasmodium falciparum is the etiological agent of malaria *tropica*, the leading cause of death due to a vector-borne infectious disease, claiming 0.5 million lives every year. The single-cell eukaryote undergoes a complex life cycle and is an obligate intracellular parasite of hepatocytes (clinically silent) and erythrocytes (disease causing). An infection can progress to a wide range of pathologies, including severe anemia and cerebral malaria, which can lead to death. *P. falciparum* repeatedly replicates over the course of 48 h inside erythrocytes, resulting in exponential growth and rapid disease progression. As the single most important infectious disease afflicting children, no other pathogen has exerted a higher selection pressure on the human genome. Over 20 polymorphisms, including the sickle-cell trait, have been selected in human populations, despite severe fitness costs, since they offer protection against fatal *P. falciparum* infections. No effective vaccine exists, but several curative treatments are available.





KEY FACTS:

No known reservoir; *P. falciparum* has a tight species barrier.

Sporozoites are injected into the skin by female Anopheles mosquitoes, travel to the liver and initiate silent expansion in hepatocytes.

Tissue sequestration due to the expression of parasite-encoded proteins leads to knob formation on the infected erythrocyte membrane.

Three genomes: a nuclear genome (23.2 Mb encoding 5,370 genes); a mitochondrial genome (6 kb) and an apicoplast genome (35 kb).

Continuous red blood cell infections and complete life cycle can be maintained in lab setting alongside both forward and reverse genetics strategies.

DISEASE FACTS:

Major cause of infant mortality, mostly in sub-Saharan Africa. Major factor for poverty in resource-poor settings.

Disease is caused by the asexual blood stages. Symptoms include signature fever-chill periodicity, fatigue and headache. Morbidity and mortality are due to a broad spectrum of pathologies, including metabolic acidosis, organ failure, anemia and coma.

Slow acquisition of partial, short-lived immunity even after years of continuous re-infections.

Treatment with artemisinin-based combination therapy is lifesaving, but no causal prophylactic drug in use. Resistance in the field has developed to all available antimalarial drugs.

Insecticide-treated bed nets have major impact on reducing transmission.

TAXONOMY AND CLASSIFICATION:

PHYLUM: Apicomplexa
CLASS: Aconoidasida
ORDER: Haemosporida
FAMILY: Plasmodiidae
GENUS: *Plasmodium*
SPECIES: *P. falciparum*

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Resources
www.who.int/malaria/en/
www.cdc.gov/malaria/
www.plasmodb.org

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  https://doi.org/10.1016/j.pt.2018.11.010

Trends in Parasitology | Parasite of the Month

Plasmodium relictum

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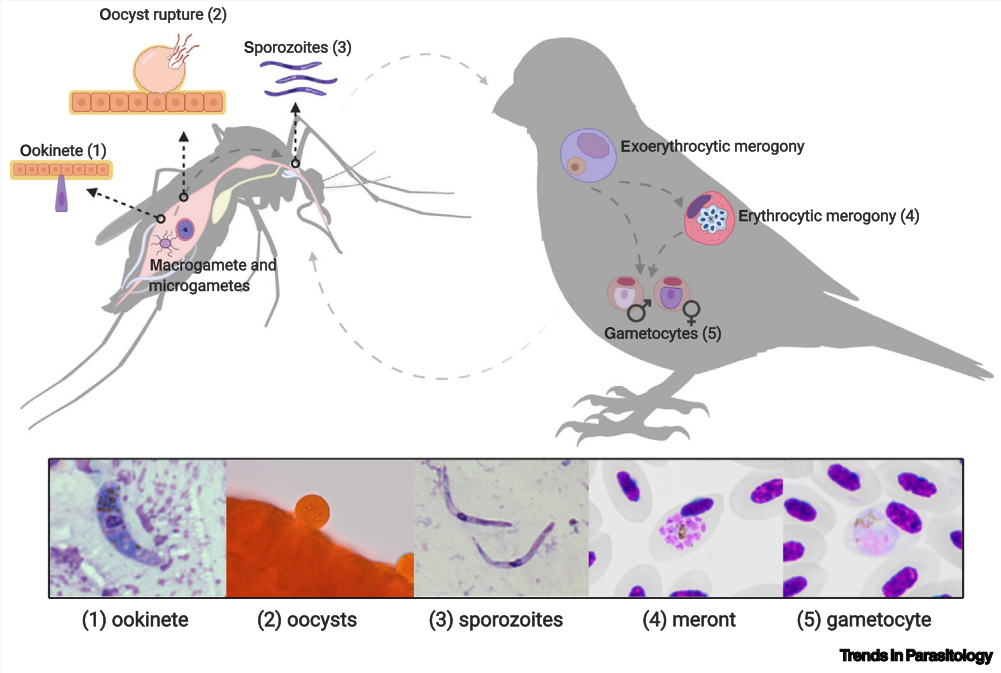
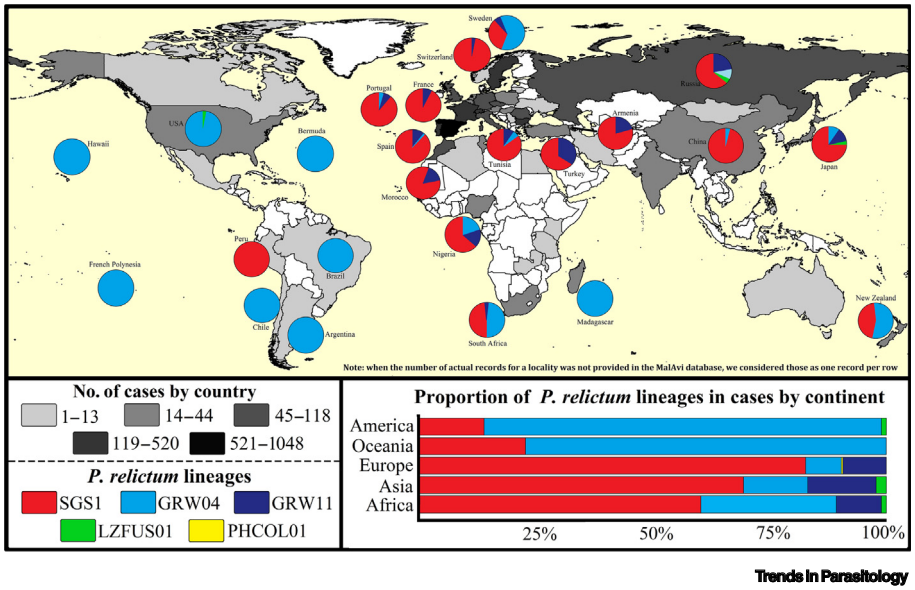


Plasmodium relictum is a widespread haemosporidian parasite infecting over 300 bird species from all continents except Antarctica. Based on sequences of the cytochrome *b* gene, that has become the barcoding region for avian haemosporidians, five different mitochondrial haplotypes (lineages) have been linked to *P. relictum* (SGS1, GRW04, GRW11, LZFUS01, and PHCOL01). *Culex* mosquitoes are the main vectors in *P. relictum* transmission, while other potential vectors include *Aedes, Lutzia, Culiseta*, and *Anopheles* species. Introduction of the lineage GRW04 to Hawaii in the first half of the 20th century, where *Culex* vectors were previously introduced – in synergy with anthropogenic impacts and infections of other pathogens (i.e., avian pox virus) – resulted in dramatic population declines of native bird species. As a result of this and other invasion events, *P. relictum* is nowadays catalogued as one of the 100 world's worst invasive species.



KEY FACTS:
Human and avian *Plasmodium* spp. are transmitted by different Culicidae species but have overall similar life cycles between mosquito vector and vertebrate host.

The role of mosquitoes in the transmission of malarial parasites was first demonstrated, using experimental infections of *P. relictum*, by Ronald Ross, who received the Nobel Prize in 1902 for this discovery.

P. relictum is one of only two avian *Plasmodium* spp. with sequenced genomes.

P. relictum is commonly used as a model organism in ecological and evolutionary experimental studies of malarial parasites.

DISEASE FACTS:
Molecular tools have identified the lineage SGS1 infecting more species of birds than any other *Plasmodium* lineage.

Unlike most other *Plasmodium* parasites, transmission of *P. relictum* takes place as far north as northern Norway.

In Europe, the findings of the lineage GRW04 are restricted to tropical migratory birds after they return from winter quarters, suggesting the absence of active transmission on breeding grounds.

Infection virulence varies among bird species and transmission areas. Upon infection with *P. relictum*, some bird species (especially in endemic regions) develop light transient parasitemias, while in other species it may lead to acute anemia and organ pathology.

TAXONOMY AND CLASSIFICATION:
PHYLUM: Apicomplexa
CLASS: Aconoidasida
ORDER: Haemosporida
FAMILY: Plasmodiidae
GENUS: *Plasmodium*
SPECIES: *P. relictum*

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Resources

www.ncbi.nlm.nih.gov/Taxonomy/Browser/wwwtax.cgi?lvl=0&id=85471
http://130.235.244.92/Malavi/

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Trends in Parasitology | Parasite of the Month

Plasmodium vivax

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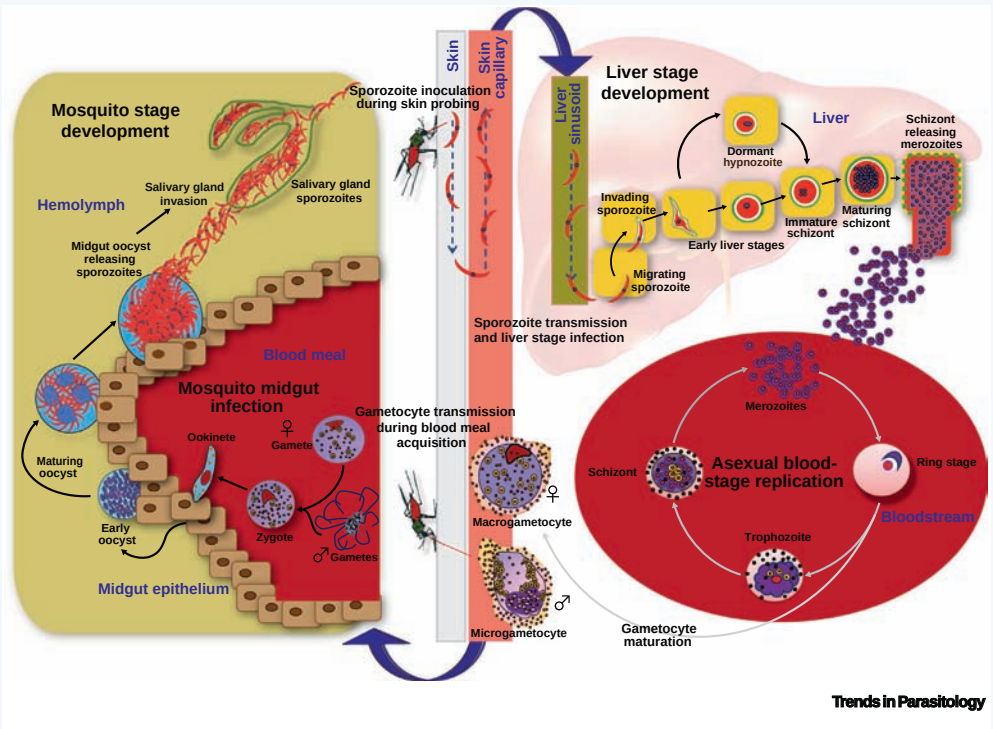
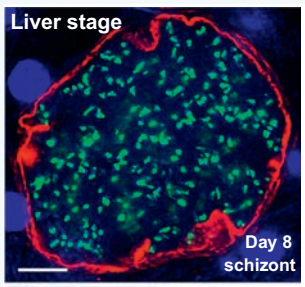


Plasmodium vivax is the most widely distributed of several plasmodial species that cause human malaria, a disease associated with blood-stage parasite replication. About 2.5 billion people are at risk of *P. vivax* infection; they live mainly in Southeast Asia and the Americas, where *P. vivax* accounts for approximately 72% of malaria cases. In Africa, widespread lack of the Duffy antigen constrains transmission. The dormant liver form of the parasite, the hypnozoite, which can reactivate long after the primary infection and give rise to a relapsing blood-stage infection, complicates eradication. In fact, hypnozoites are the origin of most blood-stage infections. Primaquine and tafenoquine are the only drugs that prevent relapse. However, neither is used during pregnancy or by people with glucose-6-phosphate dehydrogenase deficiency; and tafenoquine is not yet approved for treating children. Thus, this species of malaria-causing parasite is a unique challenge in eradication campaigns.



Key biological and epidemiological differences

Species	*Plasmodium vivax*	*Plasmodium falciparum*
Infective gametocytes in bloodstream	Present earlier (leads to earlier transmission)	Transmission occurs later
Sporogony in mosquito	Duration shorter (facilitates transmission)	Longer duration
Development in mosquito	Can occur in temperate regions, thus widespread geographically	Higher temperatures required and therefore less widespread
Parasite density in peripheral blood	Low (infection very easily overlooked)	Can be very high
Parasite population genetic diversity	High global diversity	Less diverse
Hypnozoite stage in liver	Yes	Not known to occur
Mortality	Infrequent	Frequent
Immunity	Acquired quickly	Acquired slowly

Trends in Parasitology

KEY FACTS:
Sporozoites of *P. vivax* are injected into the human skin by a mosquito and migrate to the liver, where a clinically silent phase of either parasite multiplication (schizogony) or dormancy occurs per sporozoite. Both forms of the parasite (schizont and hypnozoite) can exist simultaneously in the liver.

The transition from liver stage to blood stage results in asexual parasite expansion in erythrocytes. Sexual-stage gametocytes also develop in erythrocytes and are taken up during a mosquito blood meal, after which mature gametes fuse. Ultimately, midgut oocysts mature, releasing sporozoites that travel to the mosquito's salivary glands.

The nuclear genome is 29 Mb, encoding 6642 genes; the mitochondrial genome is 6 kb; and the apicoplast genome is 29.6 kb.

DISEASE FACTS:
Asexual blood-stage reproduction leads to illness involving febrile episodes, anemia, diarrhea, abdominal pain, nausea and vomiting, headache, and muscular pains. Disease consequences include organ failure, respiratory problems, splenomegaly, splenic rupture and occasionally death.

P. vivax is a major cause of suffering in resource-poor settings.

Continuous reinfection or superinfection is frequent, but is usually subclinical.

Patients may experience relapses after weeks to years, because of (presumed) hypnozoite activation.

TAXONOMY AND CLASSIFICATION:
PHYLUM: Apicomplexa
CLASS: Aconoidasida
ORDER: Haemosporida
FAMILY: Plasmodiidae
GENUS: *Plasmodium*
SPECIES: *P. vivax*

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  https://doi.org/10.1016/j.pt.2019.04.005

Resources

www.malariaeradication.org/
www.who.int/malaria/en/
www.cdc.gov/malaria/
www.plasmodb.org
www.mmv.org

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  https://doi.org/10.1016/j.pt.2019.04.005

Trends in Parasitology | Parasite of the Month

Schistosoma mansoni

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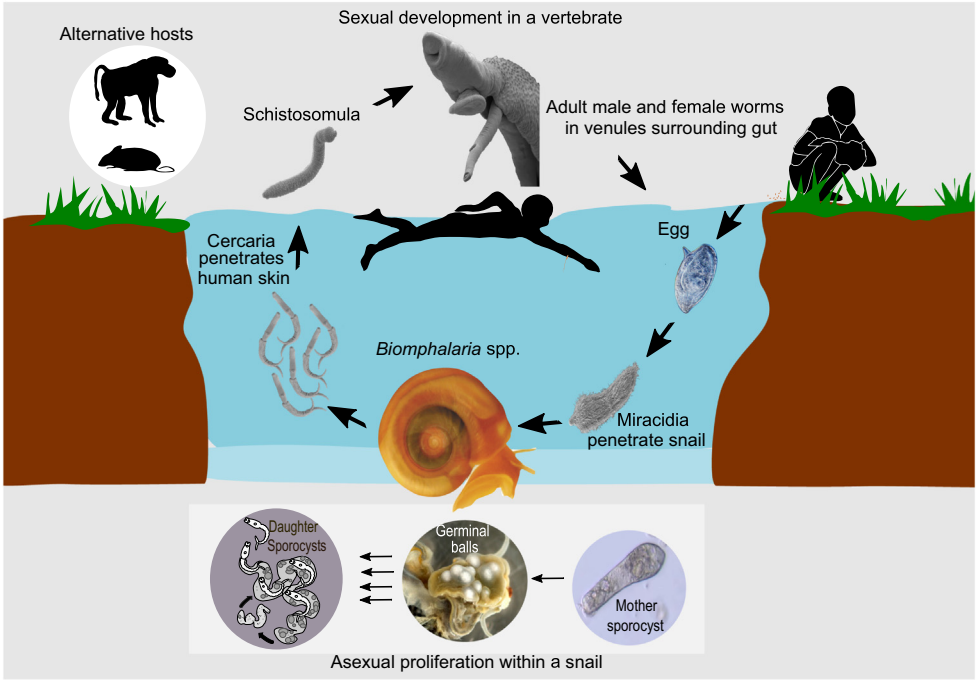
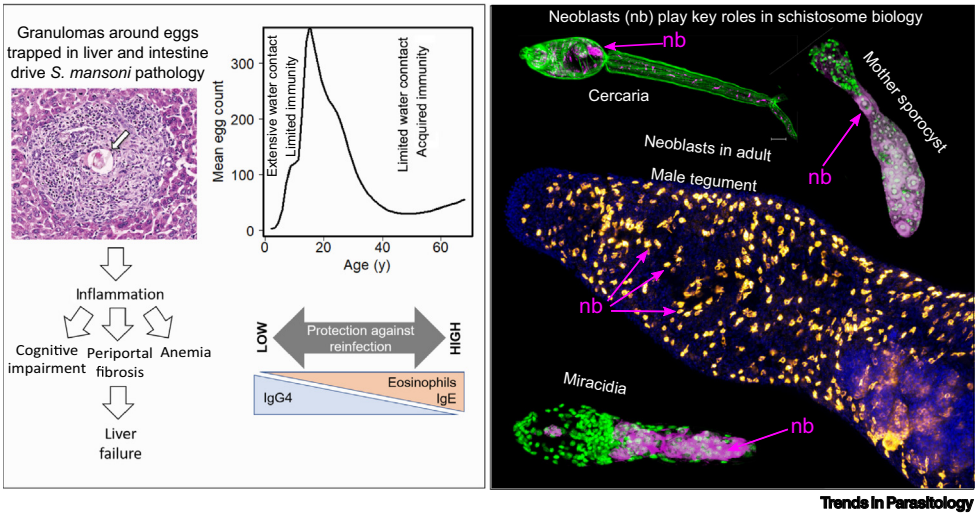


Schistosoma mansoni is the causative agent of intestinal schistosomiasis and infects ~54 million people annually, causing significant mortality and morbidity. This parasitic trematode is endemic in sub-Saharan Africa and the Middle East, and colonized South America during the transatlantic slave trade. Parasites transition between five distinctive body plans, with asexual proliferation in the snail host and sexual proliferation in the vertebrate host, and motile free-living stages**.** Transmission results from contact with water containing infected *Biomphalaria* spp. snails. Infection prevalence and intensity peaks in school-age children; both reduced water contact and acquired immunity reduces infection in adults. Pathology in the human host results from granulomas that form around eggs trapped in the liver and gut. There is no effective vaccine available: treatment of infected patients with praziquantel is the mainstay of control efforts.



KEY FACTS:

The *S. mansoni* life cycle is easily maintained in the laboratory using *Biomphalaria* spp. snails and hamster or mouse vertebrate hosts.

The parasite's genome (363 Mb, 10 144 protein genes, 7 autosomes, ZW sex determination) has been fully sequenced and assembled.

Developing a functional tool kit for this organism includes methodology for cell and stem cell biology, and functional genetic analysis (RNAi, transfection, and CRISPR).

The experimental tractability, biomedical importance, and developed genomic and cell biology resources make *S. mansoni* ideal for investigating both fundamental and applied aspects of helminth biology.

DISEASE FACTS:

Pathology results from granulomas around eggs trapped in the liver, leading to portal hypertension and liver failure. Heavy infections are associated with elevated pathology.

Diagnosis by fecal egg counts or circulating cathodic antigen test.

Adult worms remain in the bloodstream for many years and avoid immune destruction by continuous renewal of the tegument, but they do not cause pathology.

The human immune response to invading schistosomulae is predominantly T helper (Th)1, while the egg antigens stimulate a Th2 response.

S. mansoni infection castrates and reduces survival of the snail host, leading to strong coevolutionary interactions between snails and parasites.

TAXONOMY AND CLASSIFICATION:

PHYLUM: Platyhelminthes
CLASS: Trematoda
ORDER: Diplostomida
FAMILY: Schistosomatidae
GENUS: *Schistosoma*
SPECIES: *S. mansoni*

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Resources

https://parasite.wormbase.org/Schistosoma_mansoni_prjea36577/Info/Index/ (Genomic)
www.afbr-bri.org/schistosomiasis/ (Reagents: Schistosomiasis Resource Centre)
http://hydra.bio.ed.ac.uk/ (Conferences: Parasitic Helminths: New Perspectives in Biology and Infection)

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  https://doi.org/10.1016/j.pt.2020.06.003

Trends in Parasitology | Parasite of the Month

Thelazia callipaeda

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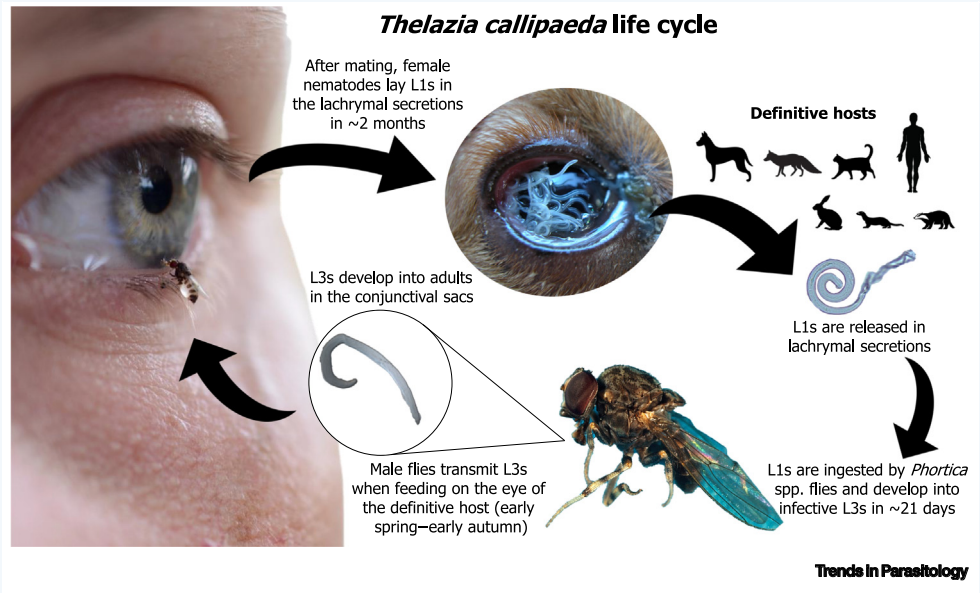
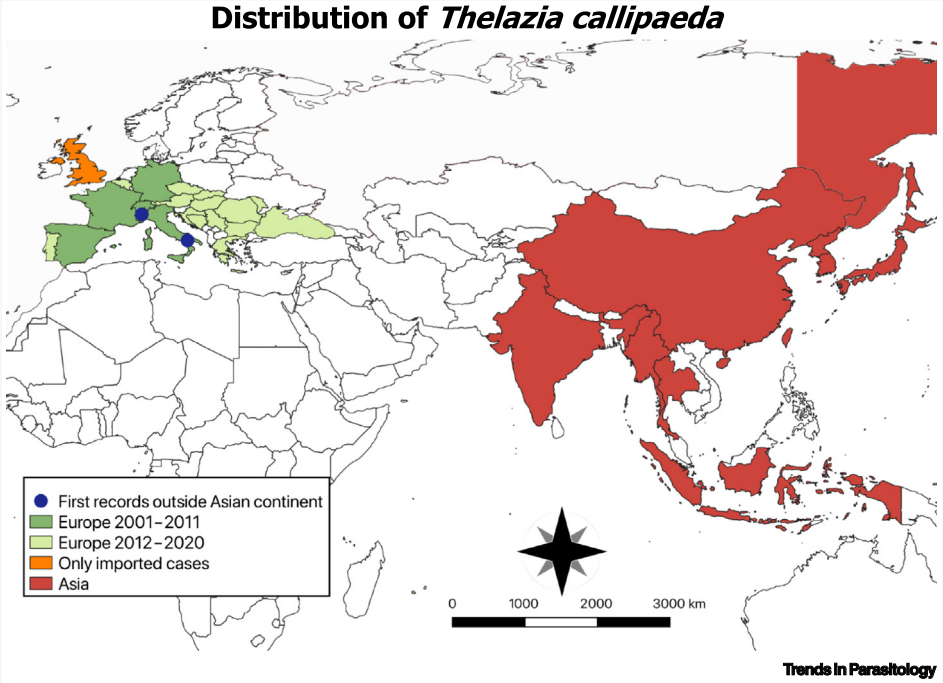
[2]Faculty of Veterinary Sciences, Bu-Ali Sina University, Hamedan, Iran
[3]Laboratory of Immunoparasitology, Department of Immunology, Aggeu Magalhães Institute, Oswaldo Cruz Foundation (Fiocruz), 50740-465, Recife, Pernambuco, Brazil



Thelazia callipaeda is a nematode living on the surface of the eyes of domestic and wild carnivores and lagomorphs, being transmitted by zoophilic drosophilids belonging to the genus *Phortica*. It also infects humans, mainly children and the elderly in poor economic settings. For a long time it has been referred to as the oriental eyeworm for its distribution in many areas of southeast Asia (i.e., from China to Indonesia) and India. Since the early 1990s it has also been reported in Europe, arising in some spots in Italy. In the last 30 years this parasite has been detected throughout Europe in almost all countries as well as in the Balkans. *T. callipaeda* may cause from mild clinical signs (e.g., lachrymation, conjunctivitis, and keratitis) to corneal ulcers and even blindness, depending on the parasite burden and individual susceptibility. Control strategies are focussed on topical or systemic anthelmintic treatments, whereas the use of repellents seems to be ineffective against the vectors.



KEY FACTS::
The *Phortica variegata* male fly is the vector of *T. callipaeda* in Europe, whereas *Phortica okadai* is the vector in China.

T. callipaeda first-stage larvae (L1s) are ingested while the flies feed on the lachrymal secretions of infected animals.

Inside the fly, L1s develop within ~21 days to L2s and L3s, the infective stage.

The parasites are transmitted when male flies harbouring L3s feed on the eye of a suitable host.

P. variegata may overwinter and transmit *T. callipaeda* in early spring until summer, and adult nematodes can survive in the definitive host during winter.

Experimental infections have demonstrated that *T. callipaeda* may develop in *P. variegata* flies from the USA, suggesting the potential occurrence of its infection in that country.

DISEASE FACTS::
T. callipaeda adults may cause variable clinical signs, including inflammation, lachrymation, and a foreign-body sensation.

In heavy infections, other clinical signs may occur, such as photophobia, oedema, corneal ulceration, conjunctivitis, and even blindness.

The use of anthelmintic drugs is useful for curing a pre-existing infection and even for preventing new infections.

TAXONOMY AND CLASSIFICATION:
PHYLUM: Nematoda
CLASS: Secernentea
ORDER: Spirurida
FAMILY: Thelazidae
GENUS: *Thelazia*
SPECIES: *T. callipaeda* (Railliet and Henry 1910)

https://doi.org/10.1016/j.pt.2020.04.013

Acknowledgments

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Resources

www.cvbd.org/en/triatomine-and-fly-borne-diseases/thelaziosis/
www.cdc.gov/dpdx/thelaziasis/index.html
www.troccap.com/

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  https://doi.org/10.1016/j.pt.2020.04.013

Toxoplasma gondii

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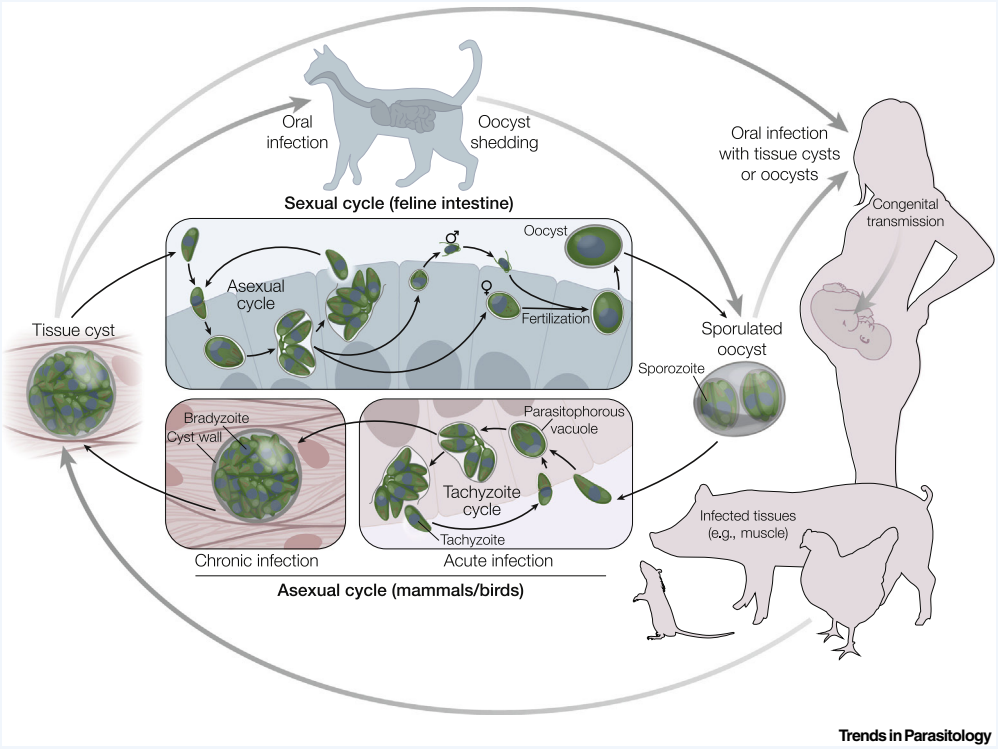
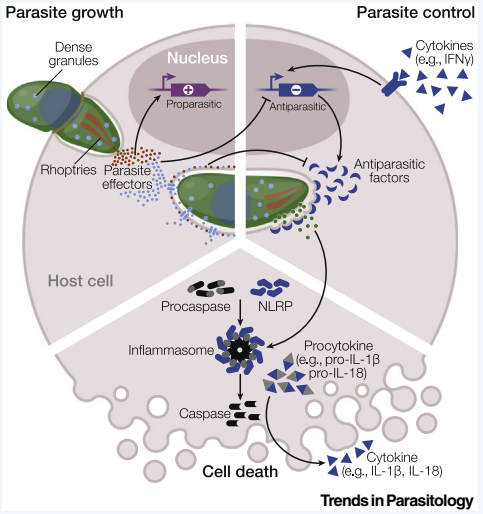
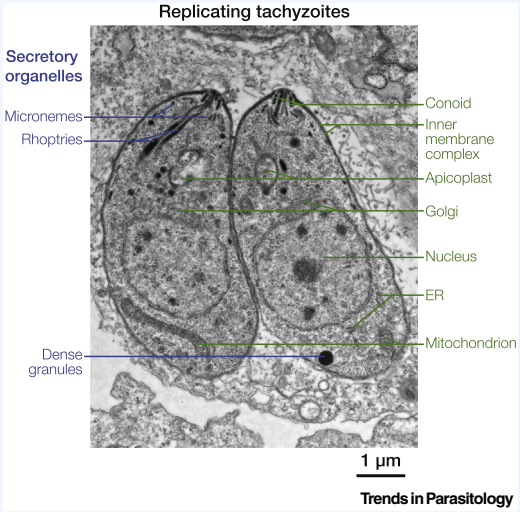


Toxoplasma gondii is an obligate intracellular eukaryotic parasite from the phylum Apicomplexa that infects up to one-third of the global population. Although most infections are asymptomatic, some cause retinal lesions and, in immunocompromised individuals or when contracted congenitally, can lead to life-threatening disseminated infections involving the central nervous system. Parasites can enter a chronic state, resistant to current therapies, which can be a reservoir for recrudescence. Felines are the strict definitive hosts for *T. gondii*, in which the parasite can sexually develop, forming highly infectious, environmentally resistant oocysts. Virtually all warm-blooded animals can act as intermediate hosts, acquiring the infection from consuming oocysts or tissues of chronically infected animals. A variety of specialized organelles release proteins that mediate motility and invasion and modulate host pathways. Extensive genetic tools make *T. gondii* a tractable model to dissect the biology of the phylum.





KEY FACTS:

Broad host range, infecting warm-blooded animals from birds to humans, with sexual recombination restricted to felines.

Following infection of the intestine from consumption of oocysts or tissue cysts, parasites disseminate systemically to replicate in a variety of nucleated cell types.

Slow-replicating chronic stages may persist life-long as tissue cysts within neurons and muscle cells.

Three genomes in the nucleus (66 Mb encoding ~8320 genes), mitochondrion, and apicoplast.

Continuous asexual culture and extensive genetic approaches enable the study of physiology, cell biology, and host–parasite interactions.

DISEASE FACTS:

Most infections are asymptomatic, although ocular lesions can occur in immunocompetent individuals and are frequently associated with recrudescence.

Parasites can enter immunologically privileged sites by crossing biologically restrictive barriers like the blood–brain and placental barriers.

Reactivation of chronic infection in immunocompromised individuals can cause life-threatening infections of the central nervous system.

Congenital transmission can occur during an untreated primary infection of the mother, commonly resulting in chorioretinitis and hydrocephalus in the newborn.

Chemotherapy is effective against the acute disease but fails to clear chronic stages.

TAXONOMY AND CLASSIFICATION:

PHYLUM: Apicomplexa
CLASS: Conoidasida
ORDER: Eucoccidiorida
FAMILY: Sarcocystidae
GENUS: *Toxoplasma*
SPECIES: *T. gondii*

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Resources

www.toxodb.org
www.cdc.gov/parasites/toxoplasmosis/
www.llamp.net

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Trichinella britovi

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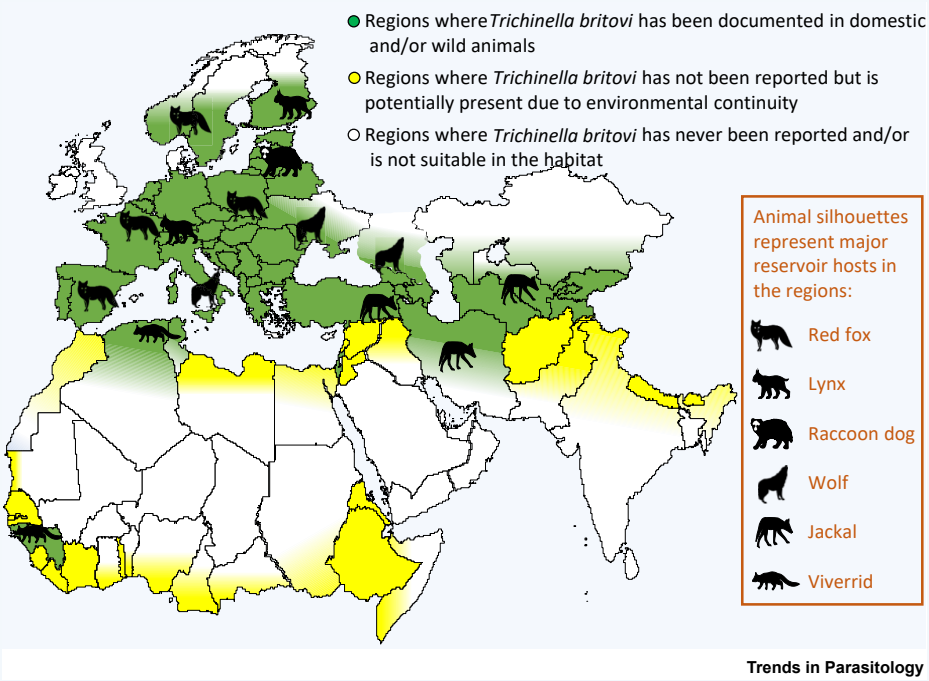
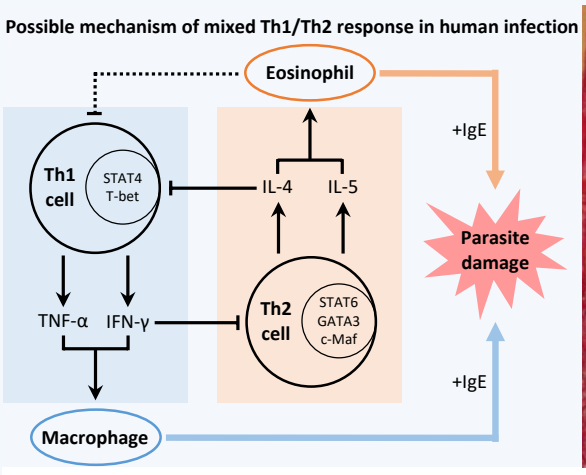
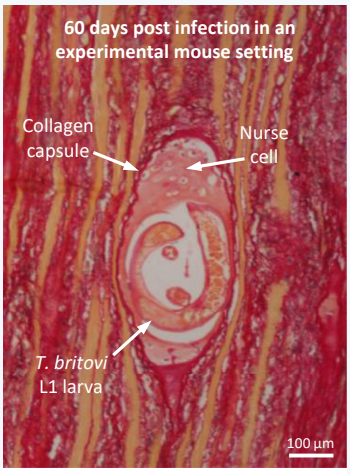


Trichinella britovi belongs to the encapsulated clade of the genus *Trichinella* and shows a wide distribution covering most of Europe (excluding some islands and the very north), north and west Africa, and Southwest Asia. After a host, including humans, ingests infected muscles, the larvae are released in the stomach before they reach the small intestine, penetrate the villi, and undergo four molts within 2 days postinfection (dpi), developing to the adult stage. Male and female worms mate, and the females begin to produce newborn larvae 6 dpi for 2–3 weeks in a mouse model. Once released, the larvae migrate into the lymphatic and blood vessels, then enter the blood circulation and travel to skeletal muscle cells, where they develop to the L1 infective stage in about 15 dpi. A completely developed collagen capsule around L1 worms can be detected from 24–42 dpi. In the modified muscle cells (nurse cells), larvae can survive for years waiting to be ingested by a new host. In humans, *T. britovi* infection generally shows a low severity.





Trends in Parasitology

KEY FACTS:

The natural infection cycle occurs in carnivore and omnivore mammalians, including humans. Carnivores act as reservoirs, whereas domestic and wild swine play a minor role.

In Europe, infection prevalence in wild carnivores and boars is much higher in the north than in the south due to environmental conditions favoring larva survival in decaying muscles of animal carcasses.

Larvae can survive in frozen muscles of swine and carnivores for up to 3 weeks and 1 year, respectively.

The main infection sources for humans are meat from wild boars, pigs, horses, and sometimes carnivores.

DISEASE FACTS:

The clinical picture is usually mild due to low fertility of female worms in humans. However, electromyography changes may be present for years.

Facial/periorbital edema, the typical sign of trichinellosis, occurs in 47–85% of cases.

Seroconversion has been documented up to 2 months postinfection. Circulating antibodies disappear within 6 months in half of the patients, while most patients become seronegative within 3 years. Specific antibodies have been detected 15 years postinfection.

Patient T cell clones reveal a mixed T helper (Th)1/Th2 response 3 months after infection.

No deaths were reported in *T. britovi* outbreaks.

Treatment: albendazole (800 mg/day for 1 week) + prednisone (25 mg *per os* with de-escalating doses for 10 days).

TAXONOMY AND CLASSIFICATION:

KINGDOM: Animalia
PHYLUM: Nematoda
CLASS: Enoplea
ORDER: Trichocephalida
FAMILY: Trichinellidae
GENUS: *Trichinella*
SPECIES: *T. britovi*

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Acknowledgments

The authors are indebted to Dr Francesca Panvini and Stefano Mazzoni for preparing the histology image.

Resources

www.trichinellosis.org
www.trichinella.iss.it

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  https://doi.org/10.1016/j.pt.2019.10.003

Trends in Parasitology | Parasite of the Month

Trichomonas vaginalis

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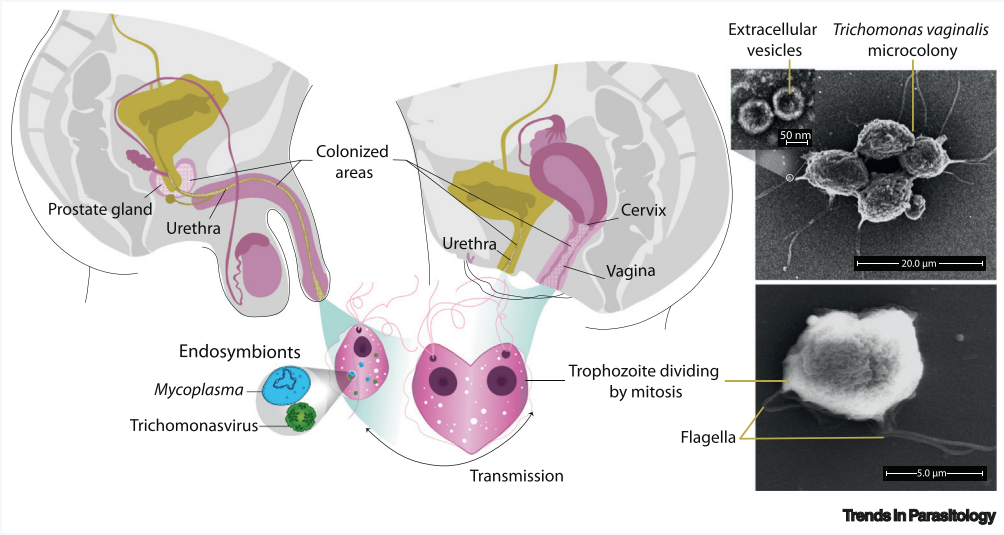
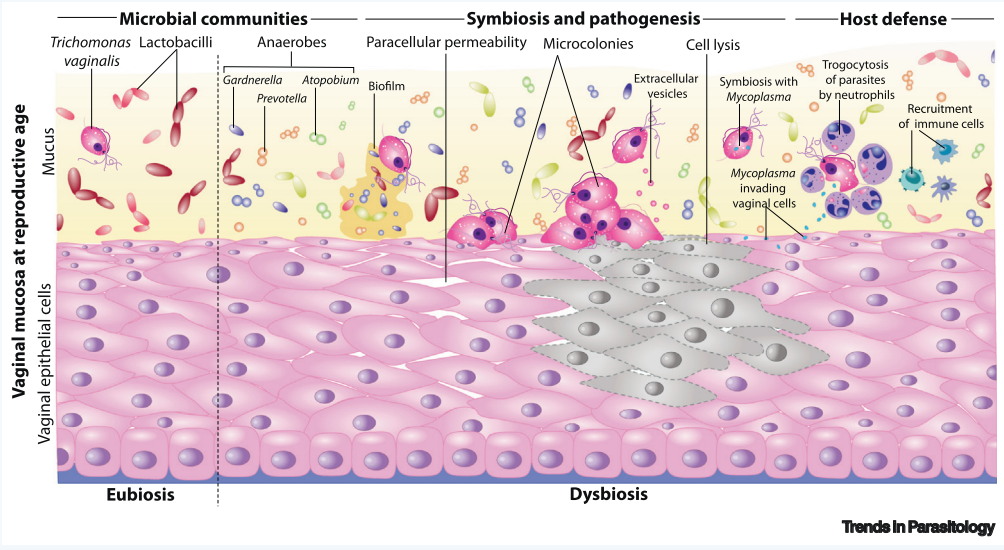
[2]All authors made equal contributions



The extracellular protozoan parasite *Trichomonas vaginalis* colonizes the lower urogenital tract of humans: the vagina, ectocervix, urethra, and prostate, where trophozoites divide asexually and the transmission depends on sexual contact. Trichomoniasis is the most common, nonviral sexually transmitted infection worldwide, accounting for ~270 million cases each year. Pathogenesis has been well characterized in the female genital tract, where reproductive outcomes are clinically relevant, resulting in vaginitis with discharge. *T. vaginalis* often carries endosymbionts (*Mycoplasma* and Trichomonasvirus spp.) and is accompanied with vaginal dysbiotic microbiota containing mostly anaerobic bacteria. Host cell adhesion, phagocytosis, and lysis are the major virulence traits of *T. vaginalis*, with levels varying among strains. Immunopathogenesis is modulated by endosymbionts and the associated microbiota. Despite drug resistance being documented for decades, 5-nitroimidazoles remain the only treatment option.



KEY FACTS:

While parasite colonization is inhibited by protective lactobacilli in the vaginal microbiota, *T. vaginalis* and the bacteria causing bacterial vaginosis amplify disease synergistically.

Metabolic interactions with *Mycoplasma* enhance the growth and weaken the macrophage-mediated killing of *T. vaginalis*.

5-Nitromidazole treatment of trichomoniasis neither eliminates *Mycoplasma* nor counteracts the vaginal microbiome disturbances; hence novel therapies are necessary.

Despite hurdles of genome size (~160 Mbp and 60 000 protein-coding genes) and repetitiveness, CRISPR/Cas9 editing should advance genetic studies.

DISEASE FACTS:

Trichomoniasis is associated with poor birth outcomes and increased risks of HIV transmission and cervical cancer.

Parasite clumping is strain dependent. Size-variable microcolonies dysregulate epithelium permeability or promote its destruction.

Parasite extracellular vesicles are immunomodulatory, 'priming' host cells and parasites for adherence.

T. vaginalis-induced immunomodulation contributes to pathology, HIV spread, and immune evasion.

Neutrophils kill the parasites by trogocytosis, but reinfections are common due to insufficient immunity.

TAXONOMY AND CLASSIFICATION:

KINGDOM: Protozoa
PHYLUM: Parabasalia
CLASS: Trichomonadea
ORDER: Trichomonadida
FAMILY: Trichomonadidae
GENUS: *Trichomonas*
SPECIES: *T. vaginalis*

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A.S.-B. thanks the Health Research Council of New Zealand for funding of the original research proposal on *T. vaginalis* and the vaginal microbiota (HRC 11/314); this led to many of the findings summarized here. The authors are grateful for the technical support on microscopy from Dr Adrian Turner and Ms Catherine Hobbis, University of Auckland.

Resources

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  https://doi.org/10.1016/j.pt.2020.01.010

Troglostrongylus brevior

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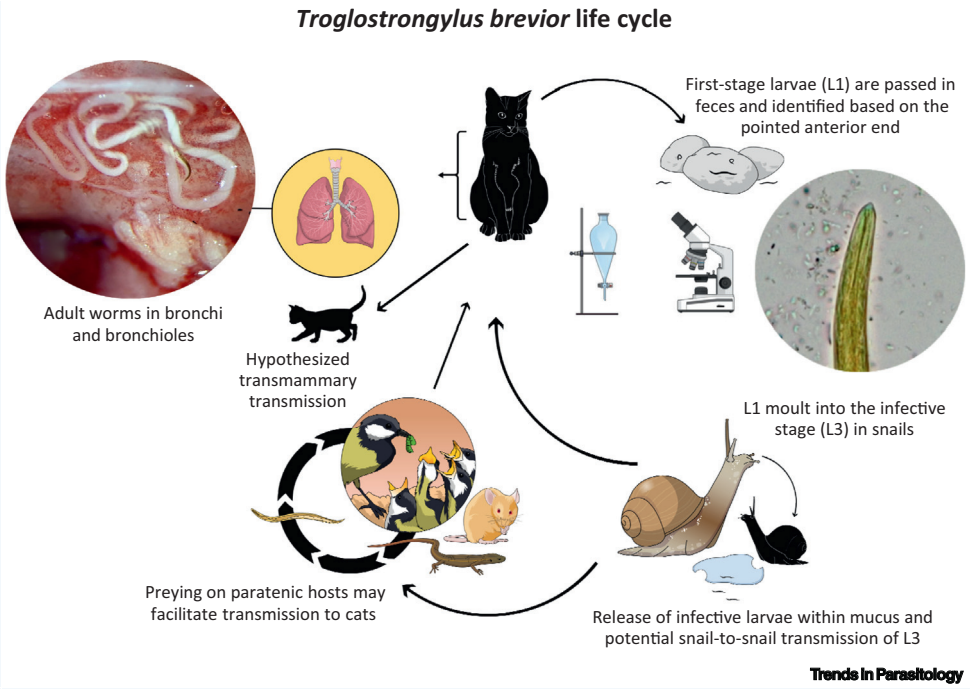
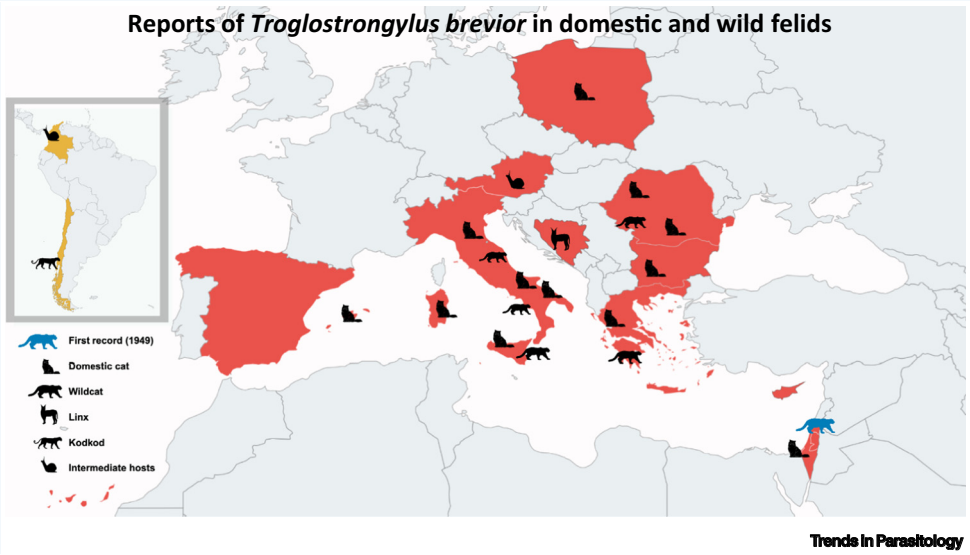
[5]Faculty of Veterinary Sciences, Bu-Ali Sina University, Hamedan, Iran



Troglostrongylus brevior is a metastrongyloid lungworm of felids. Similar to the better-known lungworm species *Aelurostrongylus abstrusus*, *T. brevior* completes its life cycle in snails and potentially in many species of paratenic hosts. This lungworm was first described by Gerichter in 1949 and has been reported increasingly in cats from many countries in recent years. Epidemiological data indicate *T. brevior* as the second most diagnosed lungworm in domestic cats in Europe. Wildcats have been suggested as natural reservoir hosts, given the high proportion of these animals found with *T. brevior* infection. *T. brevior* infection may induce respiratory conditions characterized by severe clinical signs (often fatal) in kittens. Macrocyclic lactones and emodepside have been shown to have efficacy in the treatment of the infection.



KEY FACTS:

First-stage larvae (L1) are passed with feces and could be morphologically differentiated from those of *A. abstrusus* on the basis of their pointed anterior end.

In the environment, L1 use snails as intermediate hosts in which they develop to infective third-stage larvae (L3). L3 may be shed within the mucus of snails and/or infect other snails through intermediesis transmission.

A large number of animals (e.g., birds, rodents, and reptiles) may potentially serve as paratenic hosts, increasing the likelihood of infecting cats.

Direct transmission from infected mother cat to kittens via the mammary route has also been hypothesized.

DISEASE FACTS:

Because of the larger size of adult worms and their localization in the lungs (i.e., bronchi and bronchioles), *T. brevior* has been thought to be more pathogenic than *A. abstrusus*.

Minimal to severe respiratory signs have been reported in naturally infected cats, including cough, dyspnea, and sneezing.

Coinfections with other lungworms or concurrent diseases may exacerbate the severity of signs.

Fatal outcomes have been documented mainly in kittens. This finding, along with the higher frequency of infection reported in young cats, suggests that troglostrongylosis is primarily a pediatric disease.

TAXONOMY AND CLASSIFICATION

PHYLUM: Nematoda
CLASS: Chromadorea
ORDER: Strongylida
FAMILY: Crenosomatidae
GENUS: *Troglostrongylus*
SPECIES: *T. brevior* Gerichter 1949

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Acknowledgments

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Resources

https://youtu.be/4Vl90gAbP-E

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  https://doi.org/10.1016/j.pt.2020.11.006

Trypanosoma brucei

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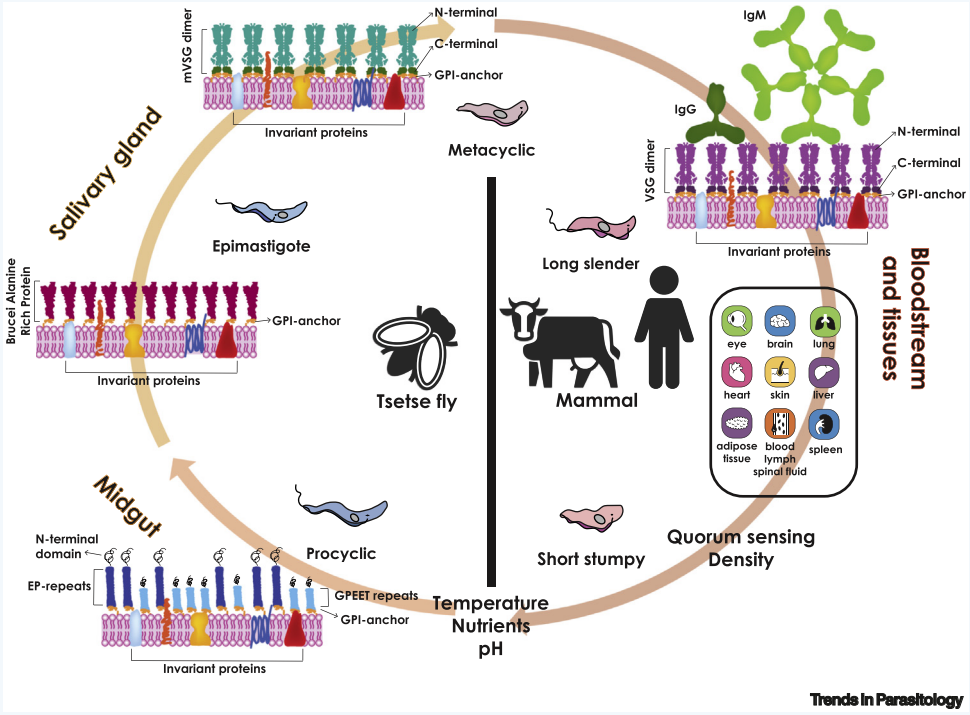
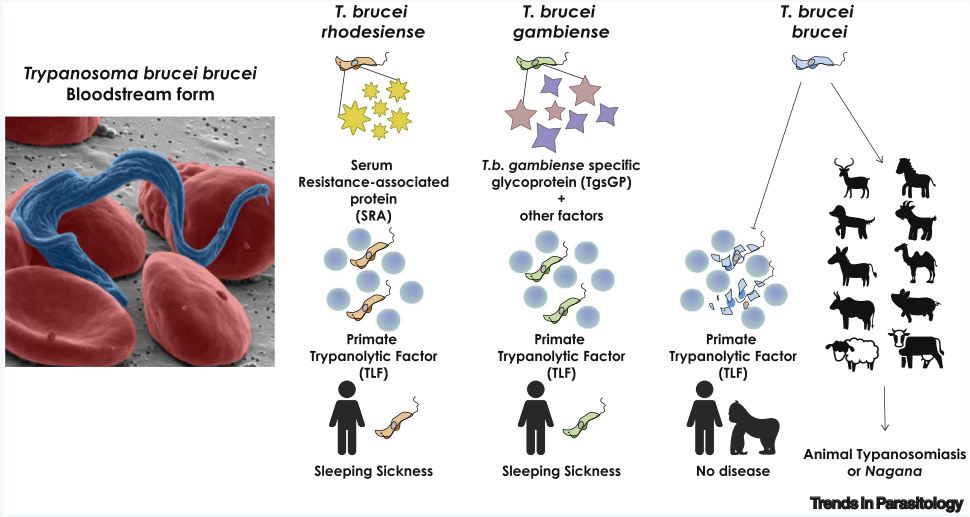


Trypanosoma brucei causes African trypanosomiasis in humans and nagana in domestic animals. This vector-borne parasite, transmitted by the tsetse fly, affects rural areas in sub-Saharan Africa. When injected by the fly, metacyclic-form parasites are introduced into the host dermis and then disseminate into the bloodstream as replicative long slender forms. Throughout its life cycle, *T. brucei* is entirely extracellular. To evade host antibody recognition, the parasite uses antigenic variation: it periodically changes a dense coat of only one kind of variant surface glycoprotein (VSG), drawing from a genomic repertoire of about 2000 VSG-encoding genes. Using quorum sensing mechanisms, slender forms develop into stumpy forms that are preadapted to the insect environment. Once taken up by the fly, the parasite replaces its VSG coat with procyclins and progresses through procyclic and epimastigote stages. Finally, the parasites become VSG-expressing metacyclic trypomastigotes.



KEY FACTS:

T. brucei possesses a unique organelle, the kinetoplast: a network of circular DNA inside a single mitochondrion.

The 35 Mb diploid genome contains three types of chromosome: megabase, intermediate, and minichromosomes.

The 11 megabase chromosomes contain the majority of transcriptionally active genes arranged in polycistronic units, as well as subtelomeric arrays of silent VSG genes.

Minichromosomes and intermediate chromosomes contain VSG genes and DNA repeats.

T. brucei typically invades tissues, including blood, lymph, bone marrow, skin, brain, eye, and heart.

DISEASE FACTS:

T. brucei is lysed by a primate serum component called trypanosome lytic factor (TLF), rendering it noninfectious to humans. *Trypanosoma brucei gambiense* and *Trypanosoma brucei rhodesiense* are resistant to TLF and can infect humans.

Symptoms in the early stage of infection: fever, joint pain, and swollen lymph nodes. In the late stage the central nervous system is affected.

Detection of parasites in body fluids by microscopy is required for diagnosis.

After treatment, patients are monitored for relapse by a periodic check for parasites and leukocyte counts in the cerebrospinal fluid.

Affected populations live in areas with limited access to diagnosis and treatment, but human African trypanosomiasis is on its way to elimination in most endemic countries.

TAXONOMY AND CLASSIFICATION:

PHYLUM: Euglenozoa
CLASS: Kinetoplastida
ORDER: Kinetoplastida
FAMILY: Trypanosomatidae
GENUS: *Trypanosoma*
SPECIES: *T. brucei*

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Resources

www.cdc.gov/parasites/sleepingsickness/
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Trypanosoma cruzi

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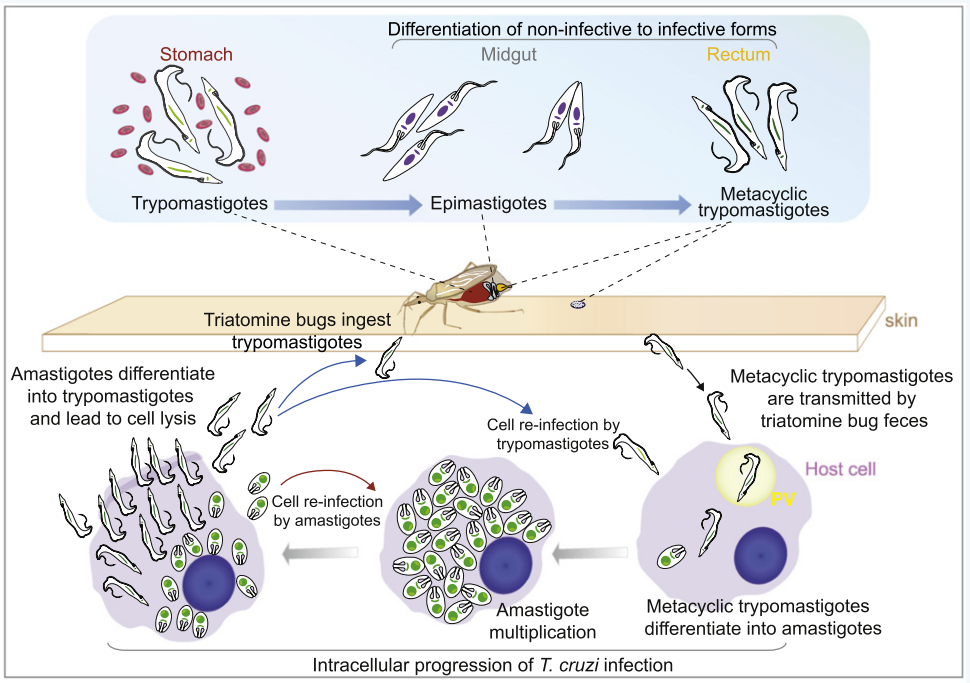
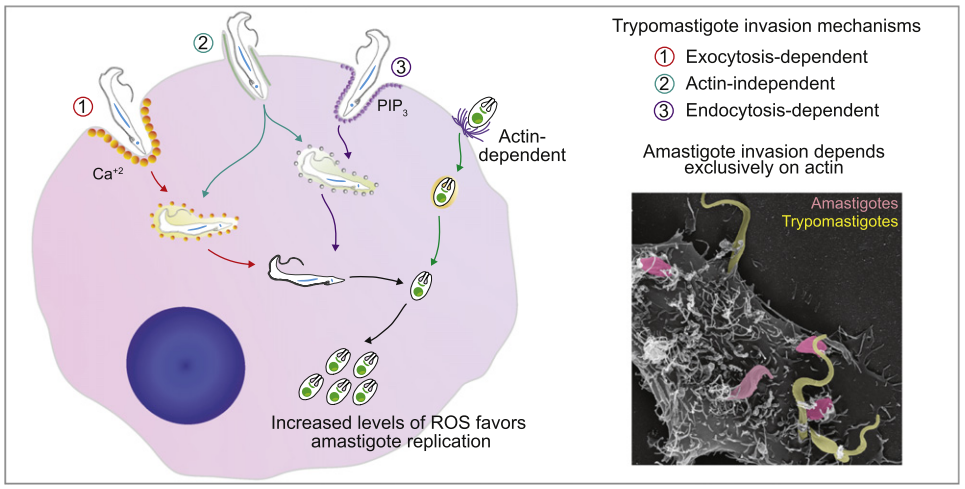


Trypanosoma cruzi is the etiological agent of American trypanosomiasis, or Chagas disease, and is transmitted mainly by blood-sucking reduviid insects in endemic countries. Metacyclic trypomastigotes released in the feces during the insect blood meal enter a mammalian host through skin wounds or mucosal membranes and invade surrounding cells. After cell invasion, metacyclic trypomastigotes are restrained within a parasitophorous vacuole (PV), from where they escape, transform into amastigotes, and multiply in the cytosol. Later, following binary division, amastigotes differentiate back into highly motile trypomastigotes that are released upon cell lysis. They can infect neighboring cells, migrate to different tissues, or be ingested by an insect vector. The parasites in the tissues, associated with an immune response, contribute to the chronic symptoms of the disease. Reactive oxygen species (ROS), among other factors, play an important role during parasite multiplication and interstage transformation.



KEY FACTS:

The Brazilian physician Carlos Chagas first discovered the parasite and defined its life cycle, vector, and disease symptoms.

T. cruzi presents replicating and nonreplicating forms. Transition between these forms relies on environmental signals, including ROS.

T. cruzi is classified into seven discrete typing units (DTUs), TcI–TcVI and Tcbat, based on genetic and biological diversity, which show different clinical manifestations and drug sensitivity.

The parasite can invade and replicate within many cell types, including macrophages and smooth and striated muscle cells.

DISEASE FACTS:

The disease is endemic in southern USA and 21 countries across Latin America, with ~7 million people infected and 70 million at risk.

Migratory movements are increasing the disease risk in the USA, Europe, and Asia.

T. cruzi is also transmitted congenitally, by blood transfusion, by organ transplantation, and by ingestion of contaminated food and drink.

The acute phase happens at any age and is usually asymptomatic. Parasites are found in the peripheral circulation.

The chronic phase manifests ~20 years from the first infection, with low parasite burden, causing cardiomyopathy, megaesophagus, and megacolon.

Benznidazole and nifurtimox are available drugs and are effective in the acute phase but less dependable for chronic-phase cure.

TAXONOMY AND CLASSIFICATION:

KINGDOM: Protozoa
PHYLUM: Euglenozoa
CLASS: Kinetoplastea
ORDER: Kinetoplastida
FAMILY: Trypanosomatidae
GENUS: *Trypanosoma*
SPECIES: *T. cruzi*

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Resources

www.dndi.org/diseases-projects/chagas/
www.cdc.gov/parasites/chagas/
www.who.int/chagas/en/

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