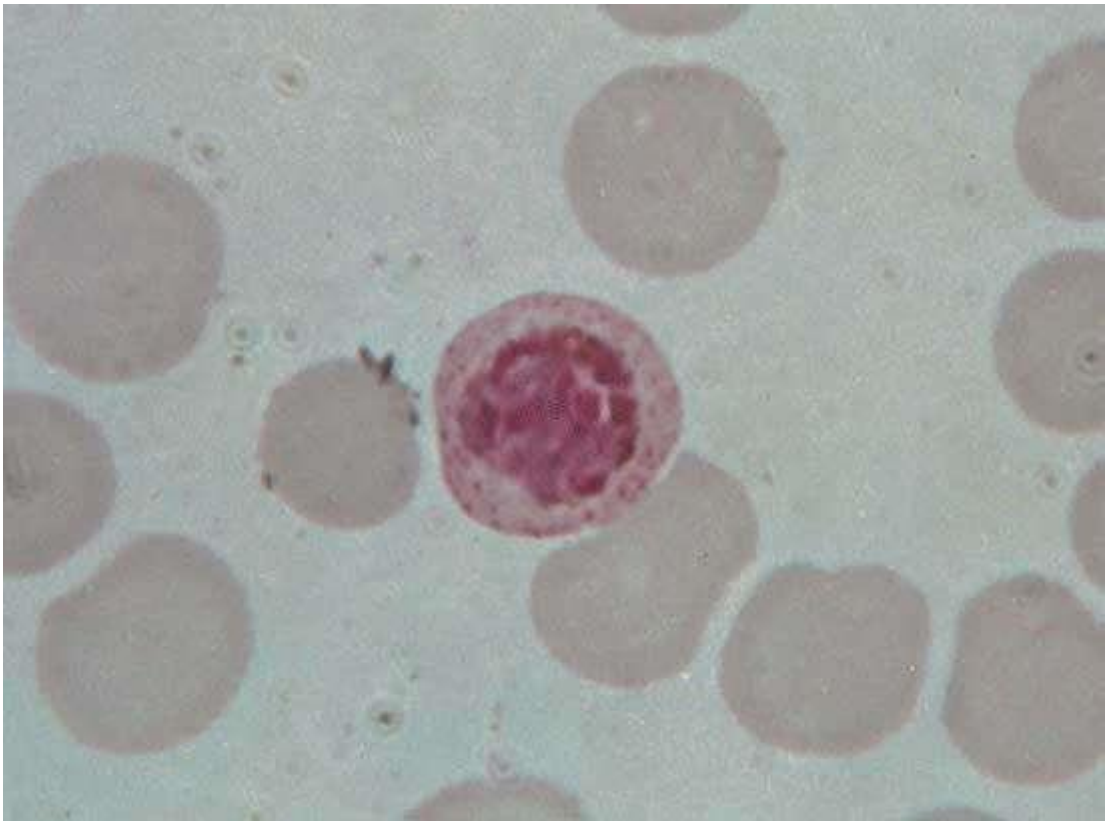
Malaria

Malaria, serious relapsing infection in humans, characterized by periodic attacks of chills and fever, anemia, splenomegaly (enlargement of the spleen), and often fatal complications. It is caused by one-celled parasites of the genus *Plasmodium* that are transmitted to humans by the bite of *Anopheles* mosquitoes. Malaria can occur in temperate regions, but it is most common in the tropics and subtropics. In many parts of sub-Saharan Africa, entire populations are infected more or less constantly. Malaria is also common in Central America, the northern half of South America, and in South and Southeast Asia. The disease also occurs in countries bordering on the Mediterranean, in the Middle East, and in East Asia. In Europe, North America, and the developed countries of East Asia, malaria is still encountered in travelers arriving or returning from affected tropical zones.

The Course Of The Disease

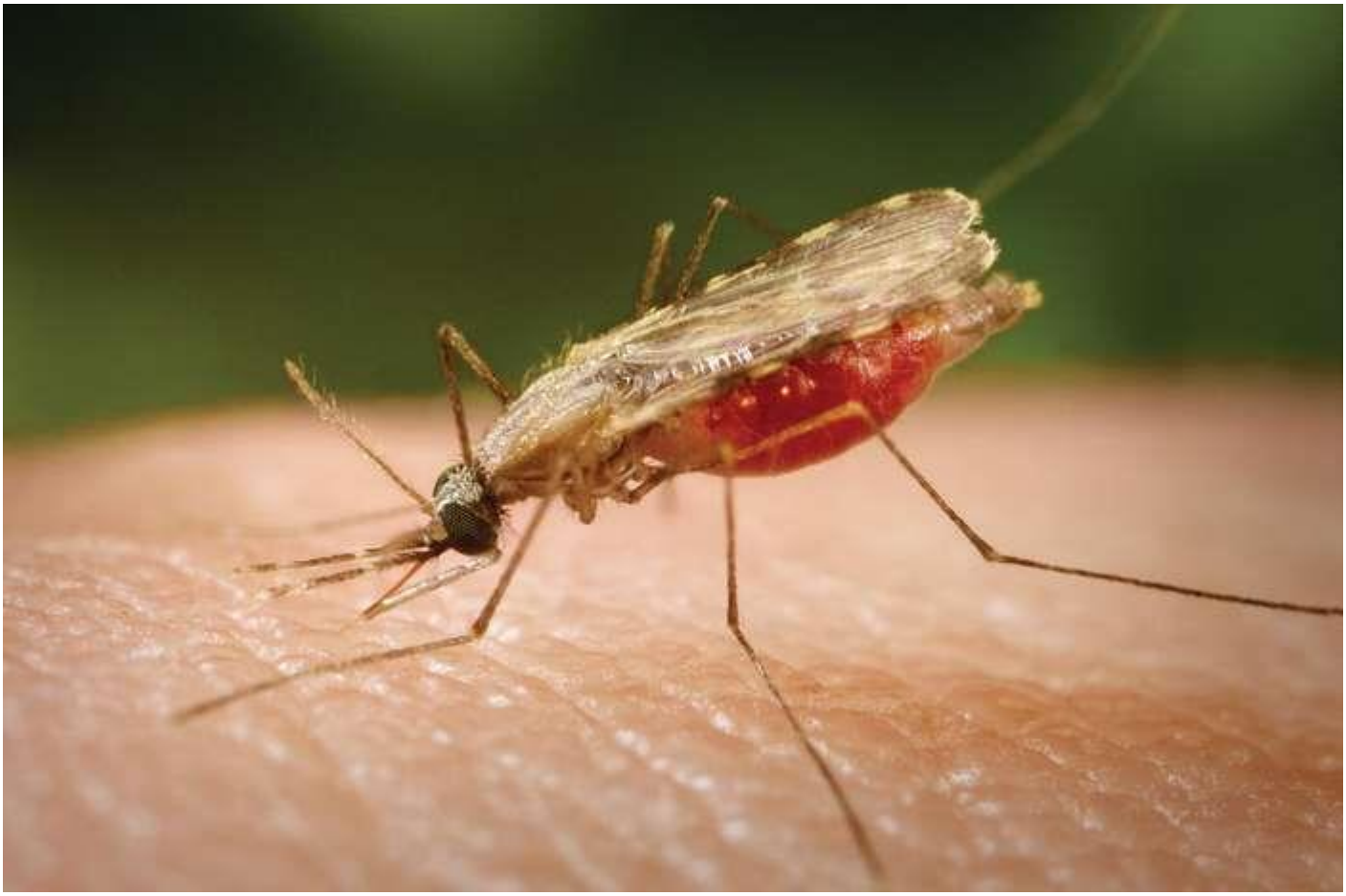
Malaria in humans is caused by five related protozoan (single-celled) parasites: *Plasmodium falciparum*, *P. vivax*, *P. ovale*, *P. malariae*, and *P. knowlesi*. The most common worldwide is *P. vivax*. The deadliest is *P. falciparum*. In 2008 *P. knowlesi*, which was thought to infect primarily Old World monkeys and to occur only rarely in humans, was identified as a major cause of malaria in humans in Southeast Asia, accounting for as many as 70 percent of cases in some areas. *P. knowlesi* was found to be easily confused with *P. malariae* during microscopic examination, resulting in many cases being attributed to *P. malariae* when in fact they may have been caused by *P. knowlesi*.



***Plasmodium vivax*, malaria parasite**The malaria parasite *Plasmodium vivax* inside a red blood cell.*A.L. Leu*

Plasmodium parasites are spread by the bite of infected female *Anopheles* mosquitoes, which feed on human blood in order to nourish their own eggs. While taking its meal (usually between dusk and dawn), an infected mosquito injects immature forms of the parasite, called sporozoites, into the person's bloodstream. The sporozoites are carried by the blood to the liver, where they mature into forms known as schizonts. Over the next one to two weeks each schizont multiplies into thousands of other forms known as merozoites. The merozoites break out of the liver and reenter the bloodstream, where they invade red blood cells, grow and divide further, and destroy the blood cells in the process. The interval between invasion of a blood cell and rupture of that cell by the next generation of merozoites is about 48 hours for *P. falciparum*, *P. vivax*,

and *P. ovale*. In *P. malariae* the cycle is 72 hours long. *P. knowlesi* has the shortest life cycle—24 hours—of the known human *Plasmodium* pathogens, and thus parasites rupture daily from infected blood cells.



mosquito: malaria vector Mosquito (*Anopheles minimus*) feeding on a human. *A. minimus* is a major malaria vector in Asia.*James Gathany/CDC*

Most merozoites reproduce asexually—that is, by making identical copies of themselves rather than by mixing the genetic material of their parents. A few, however, develop into a sexual stage known as a gametocyte. These will mate only when they enter the gut of another mosquito that bites the infected person. Mating between gametocytes

produces embryonic forms called ookinetes; these embed themselves in the mosquito's gut, where they mature after 9 to 14 days into oocysts, which in turn break open and release thousands of sporozoites that migrate to the insect's salivary glands, ready to infect the next person in the cycle.

Anopheles* mosquitoAnopheles* mosquito, carrier of the malarial parasite.© *Razvan Cornel Constantin/Dreamstime.com*

Typically, victims who are bitten by malaria-carrying mosquitoes experience no symptoms until 10 to 28 days after infection. The first clinical signs may be any combination of chills, fever, headache, muscle ache, nausea, vomiting, diarrhea, and abdominal cramps. Chills and fever occur in periodic attacks; these last 4 to 10 hours and consist first of a stage of shaking and chills, then a stage of fever and severe headache, and finally a stage of profuse sweating during which

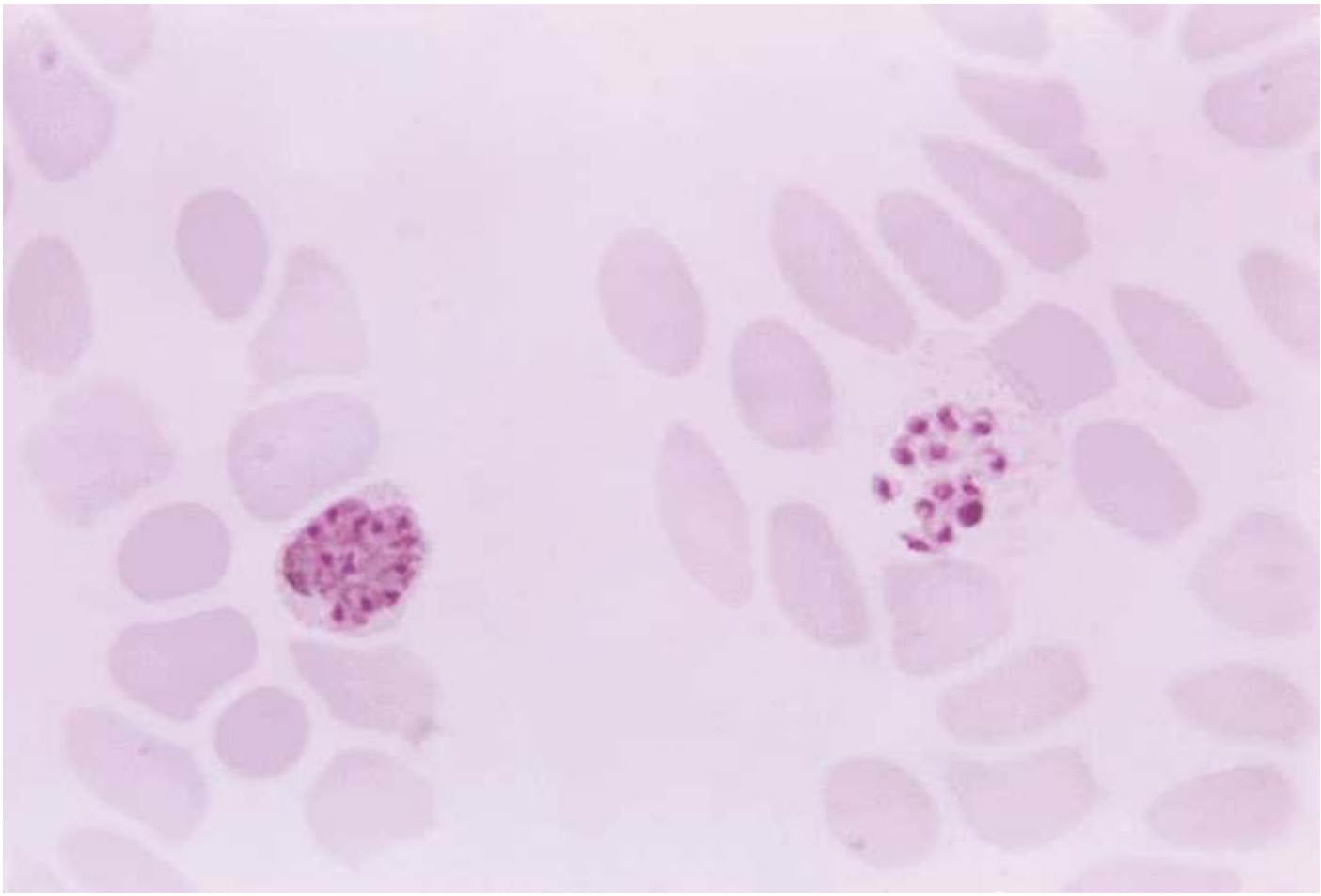
the temperature drops back to normal. Between attacks the temperature may be normal or below normal. The classic attack cycles, recurring at intervals of 48 hours (in so-called tertian malaria) or 72 hours (quartan malaria), coincide with the synchronized release of each new generation of merozoites into the bloodstream. Often, however, a victim may be infected with different species of parasites at the same time or may have different generations of the same species being released out of synchrony—in which case the classic two- or three-day pattern may be replaced by more frequent rigours of chills, fever, and sweating. The parasites continue to multiply—unless the victim is treated with appropriate drugs or dies in the interim.

Besides attacks, persons with malaria commonly have anemia (owing to the destruction of red blood cells by the parasites), enlargement of the spleen (the organ responsible for ridding the body of degenerate red blood cells), and general weakness and debility. Infections due to *P. falciparum* are by far the most dangerous. Victims of this "malignant tertian" form of the disease may deteriorate rapidly from mild symptoms to coma and death unless they are diagnosed and treated promptly and properly. The greater virulence of *P. falciparum* is associated with its tendency to infect a large proportion of the red blood cells; patients infected with that species will exhibit ten times the number of parasites per cubic millimetre of blood than patients infected with the other three malaria species. In addition, red blood cells infected with *P. falciparum* have a special tendency to adhere to the walls of the tiniest blood vessels, or capillaries. This results in obstruction of the blood flow in various organs, but the consequences are gravest when capillaries in the brain are affected, as they often are. It is this latter complication—known as cerebral malaria and manifested by confusion, convulsions, and coma—that frequently kills victims of *P. falciparum* malaria. Several strains of *P. falciparum* have developed that are resistant to some of the drugs used to treat or prevent malaria.

Infections of *P. vivax* and *P. ovale* differ from the other two types of malaria in that some of the sporozoites may remain dormant in the liver in a "hypnozoite" stage for months or even years before emerging to attack red blood cells and cause a relapse of the disease.

Diagnosis

If diagnosis is based on clinical symptoms alone, malaria may easily be confused with any of several other diseases. For example, an enlarged spleen can also sometimes be caused by other less-prevalent tropical infections such as schistosomiasis, kala-azar (a type of leishmaniasis), and typhoid fever. For this reason the most reliable method of diagnosis is a laboratory test in which a trained technician is able to distinguish between the four species of parasites when a smear of blood from the infected person is examined under a microscope. The method has drawbacks, however. For example, the test is time-consuming, may fail to detect cases where there are very few parasites, and relies on a laboratory and skilled staff. Therefore, symptoms will continue to be an important clue in detecting malaria, especially for people who live in rural areas that lack sophisticated laboratory facilities but also for international travelers. Most travelers will not develop symptoms until they return home to countries where malaria may not be endemic. This makes it vital that they recognize the possible early signs of infection themselves and tell their doctors where they have been. Otherwise, their illness may be dismissed as flu, with potentially fatal consequences. In some cases, malaria can kill within hours.



malariaTwo *Plasmodium vivax* schizonts. The mature form is on the right, and the immature form is on the left.*Dr. Mae Melvin/CDC*

Treatment

An effective treatment for malaria was known long before the cause of the disease was understood: the bark of the cinchona tree, whose most active principle, quinine, was used to alleviate malarial fevers as early as the 17th century. Quinine has been extracted from cultivated cinchona trees since the early 19th century. Despite a range of side effects such as tinnitus (ringing in the ears), blurred vision, and, less commonly, blood disorders and various allergic reactions, it is still used, especially for severe malaria and in cases in which the parasites are resistant to other, newer drugs. Chief among these newer drugs are chloroquine, a combination

of pyrimethamine and sulfadoxine, mefloquine, primaquine, and artemisinin—the latter a derivative of *Artemisia annua*, a type of wormwood whose dried leaves have been used against malarial fevers since ancient times in China. All of these drugs destroy the malarial parasites while they are living inside red blood cells. For the treatment of malignant or cerebral malaria, the antimalarial drug must be given intravenously without delay, and measures are taken to restore the red blood cell level, to correct the severe upset of the body's fluids and electrolytes, and to get rid of urea that accumulates in the blood when the kidneys fail.

cinchona bark Cinchona tree bark.*lognetic/Pond5.com*

In their initial decades of use, chloroquine and related drugs could relieve symptoms of an attack that had already started, prevent attacks altogether, and even wipe out the plasmodial infection entirely. By the

late 20th century, however, some strains of *P. vivax* as well as most strains of *P. falciparum* had become resistant to the drugs, which were thus rendered ineffective. As a result, the incidence of malaria began to increase after having steadily declined for decades. In the second decade of the 21st century, evidence of artemisinin-resistant *P. falciparum* emerged in Southeast Asia, a region of the world that previously had been the site of origin for the development of other antimalarial-resistant strains of the parasite. Artemisinin resistance was a source of major concern because of the significant role that artemisinin-based combination therapies had come to serve in the global fight against malaria.

In 2008 scientists reported the discovery of a group of proteins synthesized by *Plasmodium* that mediate the parasite's ability to make human red blood cells "sticky." Stickiness causes the infected human cells to adhere to the walls of blood vessels, allowing the parasite to evade transport to the spleen and hence destruction by the host's immune system. Scientists found that blocking the synthesis of one of the proteins involved in mediating this adherence process renders the parasite susceptible to elimination by the host's immune system. These adherence proteins represent possible targets for the development of novel antimalarial drugs.

Natural Immunity

Unlike some infectious diseases, infection with malaria induces the human body to develop immunity very slowly. Unprotected children in tropical countries acquire sufficient immunity to suppress clinical attacks only after many months or a few years of constant exposure to *Plasmodium* parasites by hungry mosquitoes. Even then, the immunity is effective only against the specific parasite to which the child has been exposed, and the immunity wanes after several months if the child is removed from constant exposure. One interesting group that shows unusual resistance to malaria are carriers of a gene for the sickle-cell trait (*see* sickle cell anemia). Infection of the red blood

cells induces the sickling effect, and the cells are destroyed along with the parasites.

Vaccines And Other Forms Of Prevention

The first malaria vaccine to be approved was RTS,S (brand name Mosquirix), which was developed by GlaxoSmithKline and which gained approval in 2015 in Europe, enabling WHO to formulate recommendations for its use in Africa. RTS,S was approved specifically for use in infants and young children aged 6 weeks to 17 months. In a study involving nearly 16,000 young children in Africa, the vaccine successfully prevented malarial infection in about 46 percent of young children aged 5 to 17 months and 27 percent of infants aged 6 to 12 weeks. RTS,S was a recombinant vaccine engineered to express *P. falciparum* proteins capable of stimulating antibody production against the parasite.

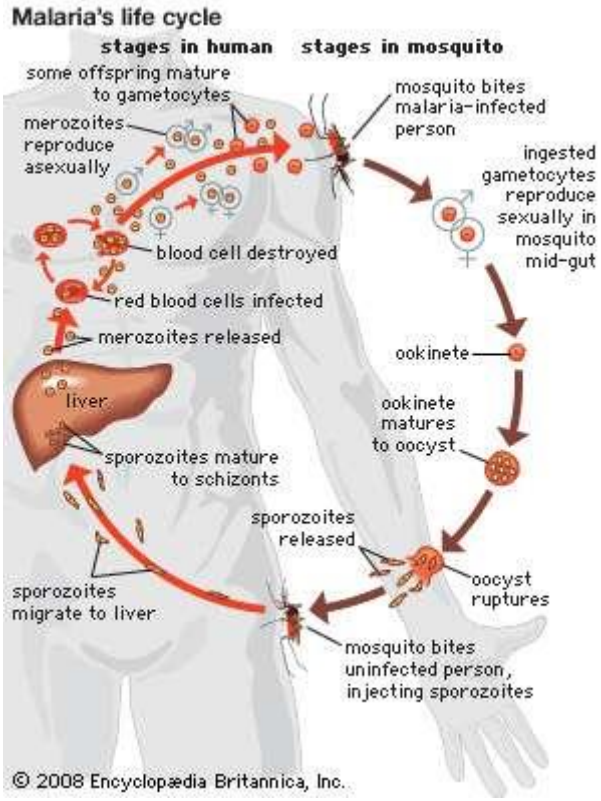
Other vaccines were also being studied. Of particular interest was a vaccine made of attenuated *P. falciparum* sporozoites (PfSPZ). In 2013 PfSPZ demonstrated early clinical success in protecting healthy volunteers against malaria. Individuals who received the highest doses of PfSPZ gained the highest levels of protection.

Another strategy was to develop an “antidisease” vaccine, which would block not the infection itself but rather the immune system’s responses to infection, which are responsible for many of the harmful symptoms. A third approach, known as the “altruistic” vaccine, would not stop either infection or symptoms but would prevent infection from spreading to others by blocking the ability of the parasites to reproduce in the gut of the mosquito.

Despite progress on malaria vaccines, the mainstay of prevention in much of Africa and Southeast Asia is the insecticide-treated bed net, which has reduced mortality significantly in some areas. For example,

in western Kenya the use of bed nets reduced mortality among children by 25 percent. Bed nets can be washed but must be re-treated with insecticide about every 6–12 months, depending on the frequency of washing. Long-lasting insecticide-treated nets (LLINs), in which insecticide forms a coating around the net's fibres or is incorporated into the fibres, can be used for at least three years before re-treatment is required. Frequent washing, however, may render LLINs less effective over time. In addition, a report published in 2011 concerning the use of deltamethrin-treated LLINs over a two-and-a-half-year period in Senegal revealed that some 37 percent of *Anopheles gambiae* mosquitoes were resistant to the insecticide. Prior to the study, only 8 percent of *A. gambiae* mosquitoes carried the genetic mutation responsible for resistance. Although longer-term investigations were needed to confirm the association between LLINs and insecticide resistance, the findings raised important questions for the future of malaria prevention and control. Furthermore, there were concerns that because bed nets reduced exposure to mosquito bites, the nets might also lead to reduced acquired immunity to malaria. This concern was highlighted by the marked increase in infection rates in the Senegal LLIN study.

For travelers to malarial regions, essential equipment in addition to a bed net would include a spray-on or roll-on insecticide such as diethyl toluamide. Travelers should also take antimalarial drugs prophylactically, though none is completely effective against the parasites. The most comprehensive method of prevention is to eliminate the breeding places of *Anopheles* mosquitoes by draining and filling marshes, swamps, stagnant pools, and other large or small bodies of standing freshwater. Insecticides have proved potent in controlling mosquito populations in affected areas.



Life cycle of a malaria parasite.

Chloroquine

Chloroquine, synthetic drug used in the treatment of malaria. Chloroquine, discovered in 1934 and introduced into medicine in the 1940s, is a member of an important series of chemically related antimalarial agents, the quinoline derivatives. Chloroquine is administered orally as chloroquine phosphate. It also can be given by intramuscular injection as chloroquine hydrochloride. Chloroquine is effective against susceptible strains of the malarial parasites *Plasmodium vivax*, *P. ovale*, and *P. falciparum* as well as certain parasitic worms and amoebas. It is also used in the treatment of inflammatory rheumatic diseases, such as lupus erythematosus and rheumatoid arthritis.

Side effects can occur with chloroquine use. Examples of mild side effects include headache and abdominal cramps, which are common to antimalarials. Persons taking chloroquine sometimes also experience

skin rash, muscle weakness, nausea, vomiting, tinnitus (ringing in the ears), and changes in behaviour. Visual impairment, in the form of retinal damage, may occur with long-term use of chloroquine; this condition is known as chloroquine retinopathy.

Chloroquine interacts with a number of other medications, including antacids, certain types of antibiotics—e.g., ampicillin and erythromycin—and antiarrhythmics (drugs used to treat defects in heart rhythms). Drug-drug interactions can alter chloroquine levels in the body, such as by blocking chloroquine metabolism, resulting in toxic chloroquine accumulation in the body. Alternatively, chloroquine can alter levels of other drugs, increasing the risk of side effects and toxicity caused by those agents.

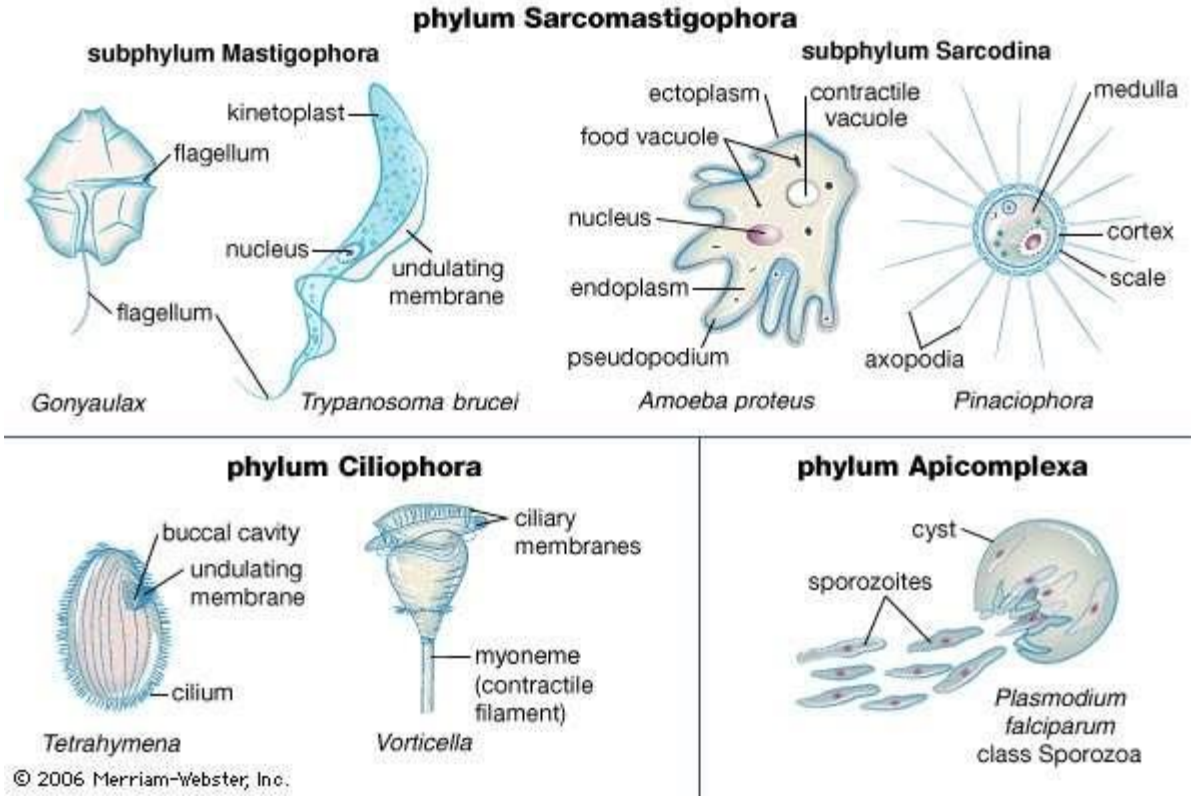
Chloroquine is closely related to hydroxychloroquine, another type of quinoline derivative. Hydroxychloroquine is also used in the treatment of malaria and inflammatory rheumatic diseases. Hydroxychloroquine has many of the same side effects as chloroquine, including an elevated risk of retinopathy, but generally is considered to be less toxic.

Chloroquine phosphate, given orally, is a drug used for the prevention and treatment of uncomplicated cases of malaria, which is caused by species of *Plasmodium*. In regions where chloroquine-resistant *P. falciparum* is encountered, mefloquine or doxycycline may be used for prevention of the disease. Infection with chloroquine-resistant *P. falciparum* may be treated with quinine sulfate, often in combination with pyrimethamine and sulfadoxine, or with artemisinin, in combination with agents such as mefloquine or amodiaquine. A high level of quinine in the plasma frequently is associated with cinchonism, a mild adverse reaction associated with such symptoms as a ringing noise in the ears (tinnitus), headache, nausea, abdominal pain, and visual disturbance. Primaquine phosphate is given orally to prevent malaria after a person has left an area where *P.*

vivax and *P. ovale* are endemic and to prevent relapses with the same organisms.

Plasmodium

Plasmodium, a genus of parasitic protozoans of the sporozoan subclass Coccidia that are the causative organisms of malaria. *Plasmodium*, which infects red blood cells in mammals (including humans), birds, and reptiles, occurs worldwide, especially in tropical and temperate zones. The organism is transmitted by the bite of the female *Anopheles* mosquito. Other insects and some mites may also transmit forms of malaria to animals.



representative protozoans Representative protozoans. The phytoflagellate *Gonyaulax* is one of the dinoflagellates responsible for the

occurrence of red tides. The zooflagellate *Trypanosoma brucei* is the causative agent of African sleeping sickness. The amoeba is one of the most common sarcodines. Other members of the subphylum Sarcodina, such as the radiolarians, heliozoans, and foraminiferans, usually possess protective coverings. The heliozoan *Pinaciophora* is shown covered with scales. The phylum Ciliophora, which includes the ciliated *Tetrahymena* and *Vorticella,* contains the greatest number of protozoan species but is the most homogeneous group. The malaria-causing *Plasmodium* is spread by the bite of a mosquito that injects infective spores (sporozoites) into the bloodstream.

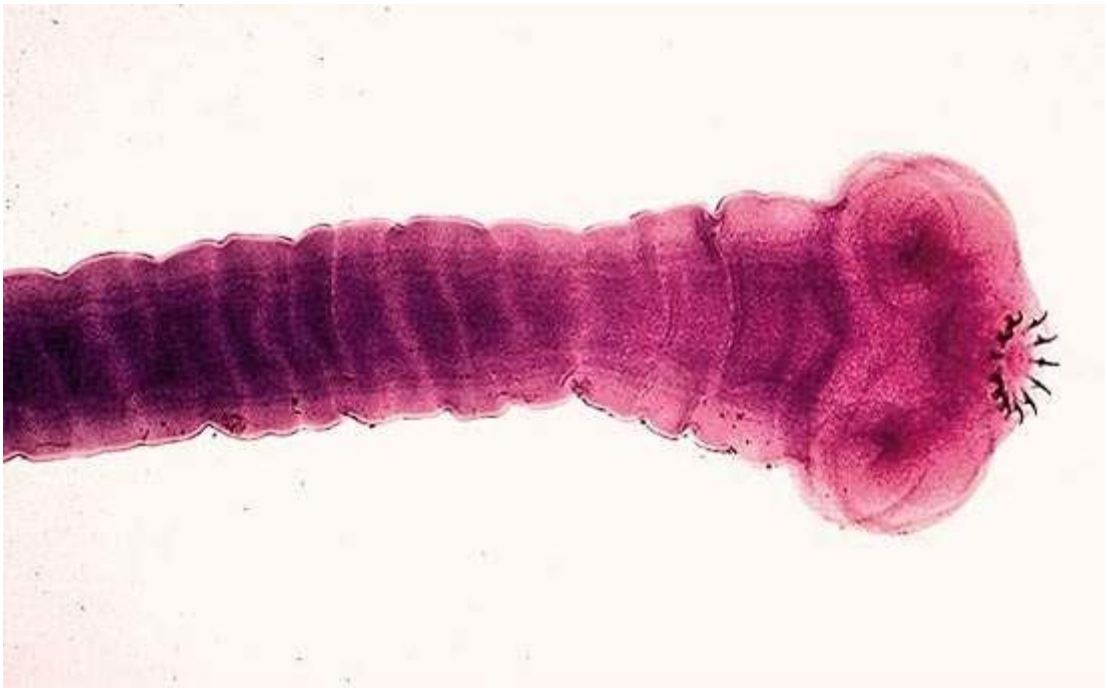
Five species cause human malaria: *P. vivax* (producing the most widespread form), *P. ovale* (relatively uncommon), *P. falciparum* (producing the most severe symptoms), *P. malariae*, and *P. knowlesi*. There are several species that have been isolated from chimpanzees, including *P. reichenowi* and *P. gaboni*. *P. falciparum*, *P. gaboni*, and other species have been isolated from gorillas. Examples of parasites found in reptiles include *P. mexicanum* and *P. floridense*, and those in birds include *P. relictum* and *P. juxtanucleare*.

Plasmodium species exhibit three life-cycle stages—gametocytes, sporozoites, and merozoites. Gametocytes within a mosquito develop into sporozoites. The sporozoites are transmitted via the saliva of a feeding mosquito to the human bloodstream. From there they enter liver parenchyma cells, where they divide and form merozoites. The merozoites are released into the bloodstream and infect red blood cells. Rapid division of the merozoites results in the destruction of the red blood cells, and the newly multiplied merozoites then infect new red blood cells. Some merozoites may develop into gametocytes, which can be ingested by a feeding mosquito, starting the life cycle over again. The red blood cells destroyed by the merozoites liberate toxins that cause the periodic chill-and-fever cycles that are the typical symptoms of malaria. *P. vivax*, *P. ovale*, and *P. falciparum* repeat this chill-fever cycle every 48 hours (tertian malaria), and *P. malariae* repeats it every 72 hours (quartan

malaria). *P. knowlesi* has a 24-hour life cycle and thus can cause daily spikes in fever.

HELMINTHS:-

Anthelmintic



cestodiasisScolex (head) of the tapeworm *Taenia solium*. The hooks of the scolex enable the tapeworm to attach to the intestinal wall.*Dr. Mae Melvin/Centers for Disease Control and Prevention (CDC)(Image Number: 1515)*

Helminths can be divided into three groups: cestodes, or tapeworms; nematodes, or roundworms; and trematodes, or flukes. The helminths differ from other infectious organisms in that they have a complex body structure. They are multicellular and have partial or complete organ systems (e.g., muscular, nervous, digestive, and reproductive). Several of the drugs used to treat worm infections affect the nervous system of the parasite and result in muscle paralysis. Other drugs affect the uptake of glucose and thus energy stores. All are chemical agents and are generally administered orally, and many are

used in both human and veterinary medicine. No anthelmintic, however, is completely effective, completely without toxic effect upon the host, or equally active against all worms.

Cestode Anthelmintics

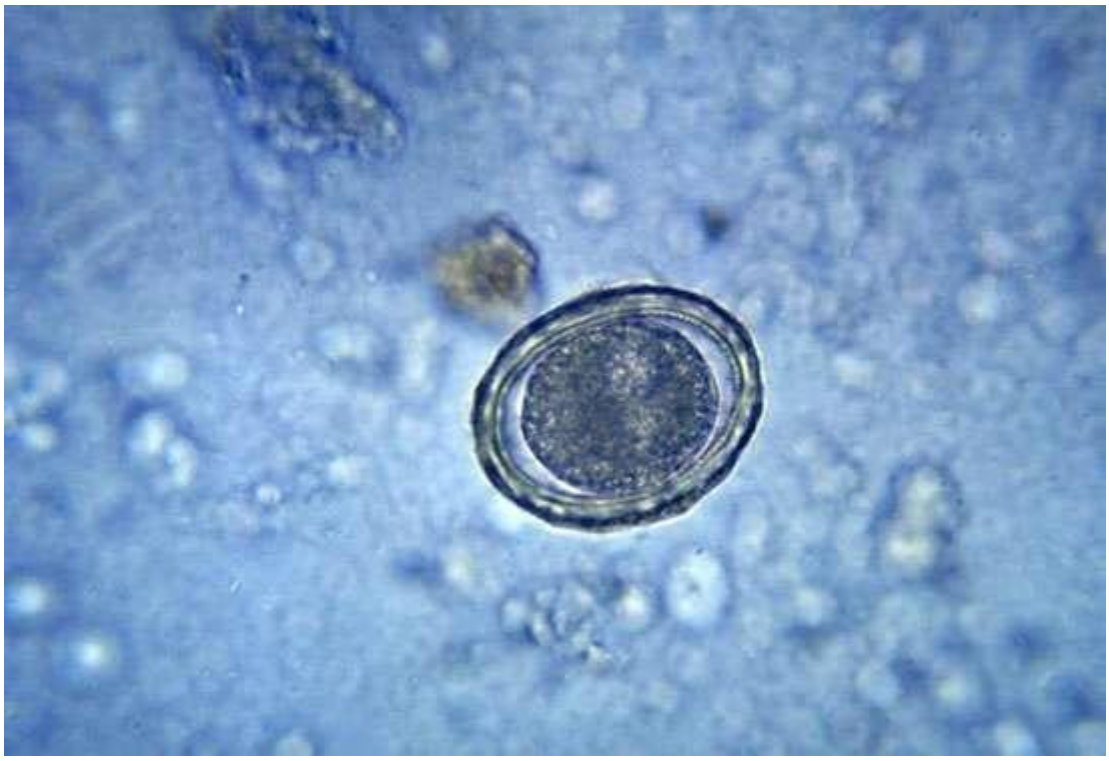
Tapeworms attach to the intestinal tract by a sucker or a sucking groove on the head (scolex). Unlike the nematodes and trematodes, tapeworms do not enter the host tissues. As a result, tapeworm infections in general are more easily treated than infections caused by worms that penetrate host tissues. In addition, because tapeworms are confined to the intestinal tract, they need not be killed by the drug, and the drug need not be absorbed when given by mouth. Thus, there usually is a wider margin of safety for cestode anthelmintics than for anthelmintics used to treat worm infections in sites other than the intestine. The term *vermifuge* is often applied to remedies that are used to remove intestinal worms.

The primary drugs used for cestode infections are albendazole and praziquantel. Albendazole inhibits the uptake of glucose by the helminth and therefore the production of energy. It has a spastic or paralytic effect on the worm. Praziquantel also produces tetanus-like contractions of the musculature of the worm. Unlike albendazole, praziquantel is readily absorbed from the intestinal tract. It is a broad-spectrum anthelmintic affecting both flukes and tapeworms.

Quinacrine, an early synthetic antimalarial later superseded, is often used as an anthelmintic for the treatment of tapeworm infection in dogs, cattle, and other animals.

Nematode Anthelmintics

Treatment of roundworms is complicated by the fact that some live in blood, lymphatics, and other tissues (filarial worms) and thus require use of drugs that are absorbed from the intestinal tract and penetrate into tissues. Others are found primarily or solely in the intestinal tract (intestinal nematodes). Diethylcarbamazine and ivermectin, used for treating filarial worm infections, are absorbed from the intestinal tract. Blood levels are reached quickly, and action against the microfilariae is rapid. A severe allergic or febrile reaction due to the death of the microfilariae can follow the use of these drugs.



Ascaris lumbricoidesFertilized egg of the roundworm *Ascaris lumbricoides*, the causative agent of ascariasis, magnified at 400x.*CDC/Dr. Mae Melvin*

Like albendazole, mebendazole interferes with glucose uptake and consequently with the production of energy. Mebendazole

accumulates in the intestine and is used for treating large intestinal roundworms (ascarids), hookworm, and whipworm infections. It is well tolerated, but abdominal discomfort and diarrhea can occur in patients with a severe infection.

Piperazine, introduced into human medicine about 1950 and shortly thereafter into veterinary medicine, relaxes the ascarids and pinworms (oxyurids) of humans and domesticated animals so that they are eliminated with the feces. Piperazine has been largely superseded by drugs such as mebendazole and pyrantel pamoate.

pinwormPinworm (*Enterobius vermicularis*).*Walter Dawn*

Pyrantel pamoate causes spastic paralysis of helminth muscle. Most of the drug is not absorbed from the intestinal tract, resulting in high levels in the intestinal lumen. It is a drug of choice in treating pinworm and is an alternative therapy for *Ascaris* infection, hookworm, and trichostrongolosis.

Thiabendazole, which is structurally related to albendazole and mebendazole, is used primarily for the treatment of several nematodes of cattle, horses, and sheep. Dithiazanine is another nematode anthelmintic used in veterinary medicine; it is effective against heartworms and threadworms. Levamisole is used in

the treatment of lungworm infections in cattle. Phenothiazine, introduced in the 1930s, is still used against the wireworm (*Haemonchus contortus*) of sheep and cattle.

Hygromycin is an antibiotic that may also be used as an anthelmintic in the form of a feed additive to eliminate or reduce ascarids, nodular worms (*Oesophagostomum*), and whipworms (*Trichuris*) of swine, and the large roundworms (*Ascaridia*) and cecal worms (*Heterakis*) of poultry.

Trematode Anthelmintics

Praziquantel is the most effective drug in treating infections caused by intestinal, liver, and lung flukes and is the drug of choice in the treatment of schistosomiasis (infections of blood flukes). Praziquantal causes contraction and spastic paralysis of the worm and also damages the membranes of the worm, which activates host defense mechanisms.

liver fluke Liver fluke (*Fasciola hepatica*).*Flukeman*

Ascaris lumbricoides

From Wikipedia, the free encyclopedia

Jump to navigationJump to search

This article is about the organism. For the disease, see Ascariasis.

Ascaris lumbricoides

An adult female *Ascaris* worm
Scientific classification
Kingdom: Animalia
Phylum: Nematoda
Class: Chromadorea

Order:	Ascaridida
Family:	Ascarididae
Genus:	*Ascaris*
Species:	***A. lumbricoides***
Binomial name	
Ascaris lumbricoides Linnaeus, 1758	

Ascaris lumbricoides is the "large roundworm" of humans, growing to a length of up to 35 cm (14 in).[1] It is one of several species of *Ascaris*. An ascarid nematode of the phylum Nematoda, it is the most common parasitic worm in humans. This organism is responsible for the disease ascariasis, a type of helminthiasis and one of the group of neglected tropical diseases. An estimated one-sixth of the human population is infected by *A. lumbricoides* or another roundworm.[2] Ascariasis is prevalent worldwide, especially in tropical and subtropical countries.[3]

It has been proposed that *Ascaris lumbricoides* and *Ascaris suum* (pig roundworm) are the same species.[4]

Contents

Lifecycle[edit]

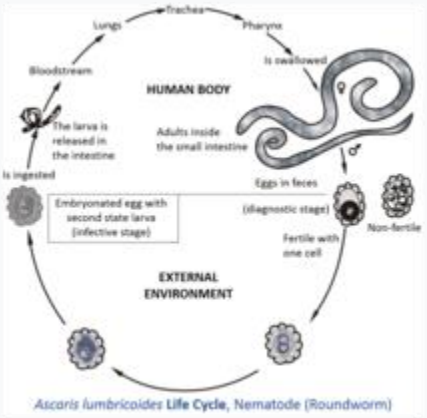


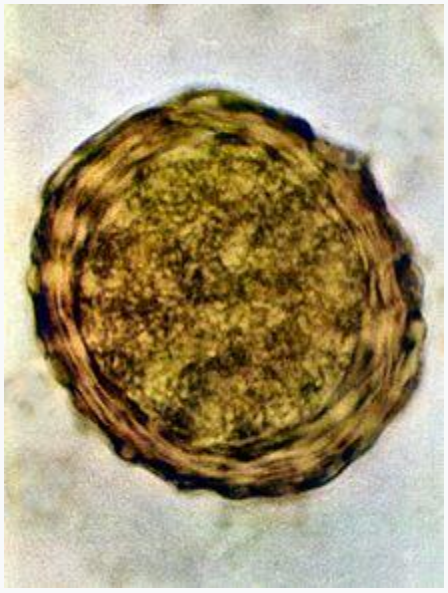
Image showing lifecycle inside and outside of the human body of one fairly well described helminth: *A. lumbricoides*

Ascaris lumbricoides, a roundworm, infects humans via the fecal-oral route. Eggs released by adult females are shed in feces. Unfertilized eggs are often observed in fecal samples but never become infective. Fertilized eggs embryonate and become infective after 18 days to several weeks in soil, depending on the environmental conditions (optimum: moist, warm, shaded soil).[5]. When an embryonated egg is ingested, a Rhabditiform larva hatches then penetrates the wall of the gastrointestinal tract and enters the blood stream. From there, it is carried to the liver and heart, and enters pulmonary circulation to break free in the alveoli, where it grows and molts. In three weeks, the larva passes from the respiratory system to be coughed up, swallowed, and thus reaches the small intestine, where it matures to an adult male or female worm. Fertilization can now occur and the female produces as many as 200,000 eggs per day for 12–18 months. These fertilized eggs become infectious after two weeks in soil; they can persist in soil for 10 years or more.[6]

The eggs have a lipid layer which makes them resistant to the effects of acids and alkalis, as well as other chemicals. This resilience helps to explain why this nematode is such a ubiquitous parasite.[7]

Morphology[edit]

Fertile egg as can be seen in a microscope



Fertile egg in human faeces (detail)

Infertile egg

Ascaris lumbricoides is characterized by its great size. Males are 2–4 mm (0.08–0.2 in) in diameter and 15–31 cm (5.9–12 in) long. The male's posterior end is curved ventrally and has a bluntly pointed tail. Females are 3–6 mm (0.1–0.2 in) wide and 20–49 cm (7.9–19 in) long. The vulva is located in the anterior end and accounts for about one-third of its body length. Uteri may contain up to 27 million eggs at a time, with 200,000 being laid per day. Fertilized eggs are oval to round in shape and are 45–75 µm (0.0018–0.0030 in) long and 35–50 µm (0.0014–0.0020 in) wide with a thick outer shell. Unfertilized eggs measure 88–94 µm (0.0035–0.0037 in) long and 44 µm (0.0017 in) wide.[8]

Epidemiology[edit]

An estimated 1 billion people are infected with *A. lumbricoides* worldwide.[9] While infection occurs throughout most of the world, *A. lumbricoides* infection is most common in sub-Saharan Africa, the Americas, China, and east Asia.[10] Although the prevalence is low in the United States, the infection still exists in southeastern part of the United States due to its temperature and humid climate.[11]

Ascaris lumbricoides eggs are extremely resistant to strong chemicals, desiccation, and low temperatures. The eggs can remain viable in the soil for several months or even years.[8] Eggs of *A. lumbricoides* have been identified in archeological coprolites in the Americas, Europe, Africa, the Middle East, and New Zealand, the oldest ones being more than 24,000 years old.[12]

Infections[edit]

Main article: Ascariasis

Infections with these parasites are more common where sanitation is poor,[13] and raw human feces are used as fertilizer.[14]

Symptoms[edit]

Often, no symptoms are presented with a minor *A. lumbricoides* infection, the inevitable consequence being the e.g. once a year passage of such clearly visible worm(s) on close inspection. In the case of bad infections symptoms commonly include bloody sputum, cough, fever, abdominal discomfort, intestinal ulcer(s), as well as a less commonly missed passing of the quite long worms.[15][16] Ascariasis is the most common cause of Löffler's syndrome worldwide. Accompanying pathological symptoms include pulmonary infiltration, eosinophilia (symptoms of the overabundance of eosinophils in the blood such as asthma and allergic reactions), and a diagnostic symptom is, aside from standard microscopy of stools, radiographic opacities.[17] One study has observed increases in fertility in infected women, in a similar vein to good diet and exercise, but with all of the pathological negatives and discomforts the disease carries with it, varying from host to host and again with diet.[18]

Prevention[edit]

Preventing any fecal-borne disease requires educated hygienic habits/culture and effective fecal treatment systems. This is particularly important with *A. lumbricoides* because its eggs are one of the most difficult pathogens to kill (second only to prions), and the eggs commonly survive 1–3 years. *A. lumbricoides* lives in the intestine where it lays eggs. Infection occurs when the eggs, too small to be seen by the unaided eye, are eaten. The eggs may get onto vegetables when improperly processed human feces of infected people are used as fertilizer for food crops. Infection may occur when food is handled without removing or killing the eggs on the hands, clothes, hair, raw vegetables/fruit, or cooked food that is (re)infected by handlers, containers, etc. Bleach does not readily kill *A. lumbricoides* eggs, but it will remove their sticky film, to allow the eggs to be rinsed away. *A. lumbricoides* eggs can be reduced by hot composting methods, but to completely kill them may require rubbing alcohol, iodine, specialized chemicals, cooking heat, or "unusually" hot composting (for example, over 50 °C (122 °F) for 24 hours[19]).

Details of infection[edit]

Infections happen when a human swallows water or food contaminated with unhatched eggs, which hatch into juveniles in the duodenum. Then they penetrate the mucosa and submucosa and enter venules or lymphatics. Next, they pass through the right heart and into pulmonary circulation. They then break out of the capillaries and enter the air spaces. Acute tissue reaction occurs when several worms get lost during this migration and accumulate in other organs of the body. The juveniles migrate from the lung up the respiratory tract to the pharynx where they are swallowed. They begin producing eggs within 60–65 days of being swallowed. These are produced within the small intestine, where the juveniles mature. It might seem odd that the worms end up in the same place where they began. One hypothesis to account for this behavior is that the migration mimics an intermediate host, which would be required for juveniles of an ancestral form to develop to the third stage. Another possibility is that tissue migration enables faster growth and larger size, which increases reproductive capacity.[20]