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Review articles

Parasitic diseases in humans transmitted by vectors

Marcin Cholewiński, Monika Derda, Edward Hadaś

Department of Biology and Medical Parasitology, Poznan University of Medical Sciences, Fredry 10, 61-701 Poznan, Poland

Corresponding author: Monika Derda; e-mail: mderda@ump.edu.pl

ABSTRACT. Despite the considerable progress of medicine, parasitic diseases still pose a great threat to human health and life. Among parasitic diseases, those transmitted by vectors, mainly arthropods, play a particular role. These diseases occur most frequently in the poorest countries and affect a vast part of the human population. They include malaria, babesiosis, trypanosomiasis, leishmaniasis and filariasis. This study presents those vector-transmitted diseases that are responsible for the greatest incidence and mortality of people on a global scale. Attention is focused primarily on diseases transmitted by mosquitoes, flies, Hemiptera and ticks.

Key words: parasitic diseases, vectors

Introduction

Parasites are among the oldest organisms existing in nature, occurring throughout the world. Their presence has been discovered even in fossil sponges millions of years old [1].

Parasitism is a form of antagonistic coexistence of two organisms of which one derives benefits while the second suffers damage. Parasites have developed a whole range of adaptations to their hosts which ensures that they achieve maximum benefits. The host is used as a source of food and also as the living environment of a parasite. These types of interactions usually lead to the onset of pathological states in the host's body. Epidemiologists estimate that at least three-fourths of living organisms are infected by various parasites.

As a result of adaptation to a parasitic mode of life, some organisms present an interesting way of transmission involving the use of various vectors. The vectors are usually arthropods which transmit parasitic organisms from one organism to others, e.g. from animals to people. Parasite transmission occurs mainly through blood sucking by an infected insect or acarine. Parasite transmission may also take place when a vertebrate ingests the infected organism of a transmitter or through wound contamination by insect excretions containing invasive forms of a parasite.

Although the most dangerous diseases caused by parasitic organisms occur in developing countries, they are also a serious health problem in developed countries. In present times increased susceptibility to infection results from numerous factors including a significant increase in international tourism, immigration, import of food products, the lack of public awareness connected with the subject matter of parasites, disregard of basic hygiene principles, the increasing number of people with decreased body resistance, and the growth of civilization diseases. In spite of the rapid development of science and medicine, parasitic diseases are still a very serious problem constantly contributing to disability, death and suffering.

In this article we present vectors and the most important diseases transmitted by them, which are still a very serious health problem, with a significant death toll.

Diseases caused by protozoans

Malaria is an acute or chronic tropical disease caused by protozoans of the genus *Plasmodium*. This protozoan occurs in red blood cells [2]. The hosts of *Plasmodium* are humans and other mammals as well as reptiles and birds. The life cycle of *Plasmodium* spp. is presented in Fig. 1 [3].

Malaria in humans is caused by five species of *Plasmodium*: *Plasmodium falciparum*, occurring mainly in countries with a tropical climate, in particular in Sub-Saharan Africa (south and center of the continent); *Plasmodium vivax*, having the broadest range of occurrence (from the tropical to the moderate zone); *Plasmodium malariae*, occurring sporadically in countries with a

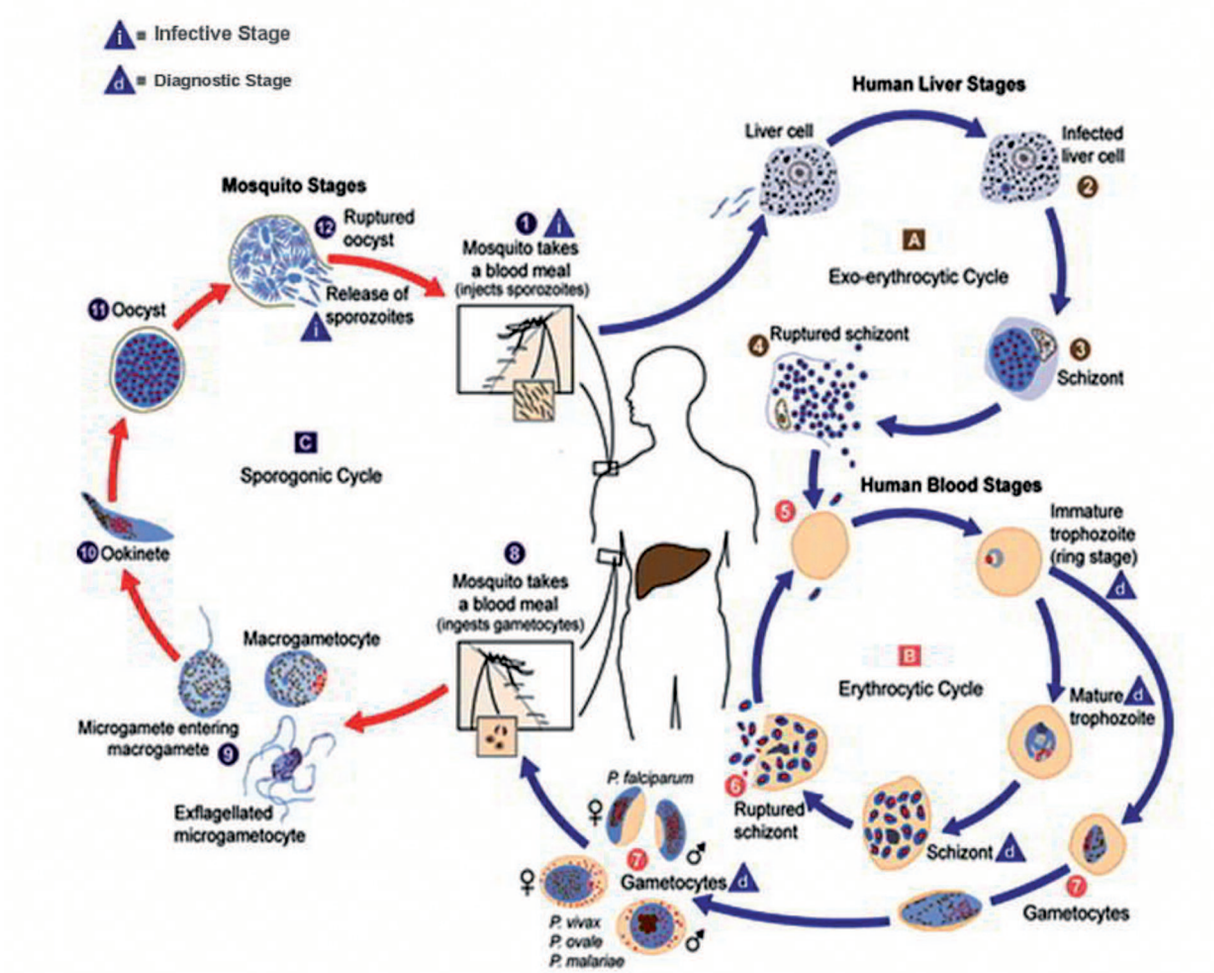

Fig. 1. Life cycle of *Plasmodium* spp. The malaria parasite life cycle involves two hosts. During a blood meal, a malaria-infected female *Anopheles* mosquito inoculates sporozoites into the human host ❶. Sporozoites infect liver cells ❷ and mature into schizonts ❸, which rupture and release merozoites ❹. (Of note, in *P. vivax* and *P. ovale* a dormant stage [hypnozoites] can persist in the liver and cause relapses by invading the bloodstream weeks, or even years later). After this initial replication in the liver (exo-erythrocytic schizogony A), the parasites undergo asexual multiplication in the erythrocytes (erythrocytic schizogony B). Merozoites infect red blood cells ❺. The ring stage trophozoites mature into schizonts, which rupture releasing merozoites ❻. Some parasites differentiate into sexual erythrocytic stages (gametocytes) ❼. Blood stage parasites are responsible for the clinical manifestations of the disease. The gametocytes, male (microgametocytes) and female (macrogametocytes), are ingested by an *Anopheles* mosquito during a blood meal ❽. The parasites' multiplication in the mosquito is known as the sporogonic cycle C. While in the mosquito's stomach, the microgametes penetrate the macrogametes generating zygotes ❾. The zygotes in turn become motile and elongated (ookinetes) ❿, which invade the midgut wall of the mosquito where they develop into oocysts ⓫. The oocysts grow, rupture, and release sporozoites ⓬, which make their way to the mosquito's salivary glands. Inoculation of the sporozoites into a new human host perpetuates the malaria life cycle ❶ [3].

subtropical and tropical climate; *Plasmodium ovale*, occurring in the west of Africa, the Philippines, east of Indonesia and in Papua New Guinea; and *Plasmodium knowlesi*, occurring in south-east Asia, Singapore, Malaysia and Thailand [4]. Among the five species, the most dangerous is *Plasmodium falciparum* [5].

In transmission of malaria approximately 30–40 species of mosquitoes of the genus *Anopheles* fulfill the significant role of vectors [6]. Mosquitoes responsible for spreading the disease breed in various bodies of water; they like puddles and also rice fields. The number of mosquitoes depends to a large extent on the rainfall intensity, temperature and humidity. The most suitable conditions for mosquito development and malaria transmission are the air temperature of 17–33ºC and the mean relative humidity above 60% [7]. Most blood-sucking mosquitoes are crepuscular feeders.

Mosquitoes are characterized by nutritional preferences. Some species prefer human blood (anthropophilic) while others prefer blood of animals (zoophilic) such as cattle or birds. Both play a significant role in transmitting and spreading malaria. Two strongly anthropophilic species of mosquitoes, namely *Anopheles gambiae* and *Anopheles funestus*, are responsible for spreading the highest number of malaria cases in Africa [8]. Both of them transmit a protozoan, *Plasmodium falciparum*, which causes the most severe cases of malaria in people. They are also natural hosts of *Plasmodium ovale* [9] and may transmit *Plasmodium malariae* [10]. Both species are very widely distributed, particularly in the center and in the south of Africa. Other African species transmitting mainly *Plasmodium falciparum* and having great significance in spreading malaria among people include *Anopheles arabiensis* (zoophilic) occurring in the south of Africa and surrounding islands, *Anopheles melas* (zoophilic and anthropophilic) occurring in the western coastal region of Africa, *Anopheles merus* (zoophilic and anthropophilic) living mainly on the southern and eastern coasts of Africa, and *Anopheles moucheti* (anthropophilic) and *Anopheles nili* (anthropophilic) [11,12]. *Anopheles arabiensis*, *Anopheles melas* and *Anopheles moucheti* may also transmit *Plasmodium malariae* [10].

The vast Asiatic continent is home to the following species of mosquitoes connected with spreading malaria among people: *Anopheles stephensi* (zoophilic species) in southern Asia including India and the Persian Gulf, where it is a vector of *P. falciparum*, *P. vivax* and *P. malariae*. *Anopheles culicifacies* (an anthropophilic and zoophilic species) occurs in Iran, Pakistan and India, and transmits *P. falciparum*, *P. vivax* and *P. malariae*. *Anopheles dirus* (a strongly anthropophilic species) occurs in Thailand, Cambodia, Laos, Vietnam and Hainan Islands, and transmits *P. vivax*, *P. falciparum*, *P. knowlesi* and *P. malariae*. *Anopheles epiroticus* (an anthropophilic and zoophilic species) occurs in the Malay Peninsula, Cambodia, Vietnam and Thailand, and transmits *P. falciparum* and *P. vivax*. *Anopheles maculatus* (a strongly zoophilic species) is a vector in the area of Thailand and Taiwan, and transmits *P. falciparum*, *P. vivax* and *P. malariae*; *Anopheles minimus* (a zoophilic and anthropophilic species) is a malaria vector of the northern areas of India and Vietnam, southern China and Taiwan, transmitting mainly *P. vivax*. *Anopheles sinensis* (zoophilic species) is the main vector of *P. vivax* mainly in China and Korea, but it also occurs in Pakistan, Japan, Thailand and Indonesia. Other important vectors of malaria in the Asiatic continent are *A. lesteri* (transmits *P. vivax*), *A. aconitus* (transmits *P. vivax*, *P. falciparum* and *P. malariae*), *A. annularis* (transmits *P. vivax*), *A. balabacensis* (transmits *P. falciparum*), *A. flavirostris* (transmits *P. vivax*), *A. fluviatilis* (transmits *P. falciparum* and *P. malariae*), *A. latens* (transmits *P. knowlesi*), *A. punctulatus* (transmits *P. falciparum*, *P. vivax* and *P. malariae*), *A. farauti* (transmits *P. falciparum*, *P. vivax*, *P. malariae*), *A. koliensis* (transmits *P. falciparum* and *P. vivax*), *A. subpictus* (transmits *P. falciparum* and *P. vivax*), and *A. sundaicus* (transmits *P. falciparum* and *P. vivax*) [10,13–18].

The areas of Papua New Guinea, northern Australia and the Salomon Islands are in turn the regions inhabited first of all by one of the above-listed species of mosquitoes, *Anopheles farauti* (mostly an anthropophilic species), which is the main vector transmitting malaria in these areas.

Mosquitoes transmitting malaria can also be found in North America, South America and Central America. One of the main vectors of this disease in the south of Mexico, in the whole Central America, in the south of South America, in the Caribbean Islands, Texas, Venezuela and Peru is *Anopheles albimanus* (a zoophilic species transmitting *Plasmodium falciparum* and *Plasmodium vivax*) [19]. Other important species transmitting malaria in this region of the world are: *Anopheles darlingi* (a

zoophilic and anthropophilic species transmitting *Plasmodium vivax*, *P. falciparum* and *P. malariae*) occurring in Mexico and in the north of South America; *Anopheles freeborni* (a zoophilic species transmitting *P. falciparum*, *P. vivax* and *P. malariae*) occurring in the west of Canada and in the United States; *Anopheles quadrimaculatus* (a zoophilic species transmitting mainly *P. vivax* and *P. falciparum*, but also *P. malariae*) occurring in the east of the USA, south-east Canada and north-east Mexico; moreover, vectors having significance for transmission of malaria include other species transmitting *Plasmodium falciparum* and *Plasmodium vivax* such as: *Anopheles albitarsis*, *A. aquasalis*, *A. marajoara*, *A. nuneztovari*, and *A. pseudopunctipennis* [10,20–22].

European and Middle Eastern vectors that may have in future great significance for transmitting malaria include *Anopheles atroparvus*, *A. labranchiae*, *A. messeae*, *A. sacharovi*, *A. sergentii*, and *A. superpictus* [22]. It is known that *A. atroparvus*, *A. messae* and *A. sacharovi* transmit *Plasmodium malariae* [10].

In the last years there have been observed more and more cases of infection in people by *Plasmodium knowlesi*, which causes malaria among monkeys, mainly macaques, and occurs in South-East Asia [23]. The species of mosquito responsible for causing malaria in people through this parasite are primarily *Anopheles cracens*, *A. kochi*, *A. hackeri*, and *A. latens* [24–26]. All attack both people and animals.

The manifestations of the disease occur from 7–30 days after becoming infected depending on the species of *Plasmodium*. The first symptoms of infection are shivers and high fever exceeding even 40°C. Next is nausea, severe headache, vomiting, sometimes diarrhea, profuse sweating and then sudden decrease of temperature together with vasodilation. After the acute first episode of inflammation with *Plasmodium* the ill person falls deeply asleep and after waking up returns to normal physical activity. Depending on the species of *Plasmodium* the attacks of fever appear regularly every few days. In the case of *P. vivax* and *P. ovale* the period is 3 days, and such a form of malaria is called a tertian fever; in the case of *P. malariae* it is 4 days (quartan fever), in *P. falciparum* the course of fever is usually irregular and chaotic, while *P. knowlesi* is characterized by daily fever attacks [27]. The disease symptoms are the result of erythrocyte destruction which leads to hemoglobinuria, hepatitis and hemolytic anemia. The additional symptoms of the disease are dyspnea, cough, pain in the spine, muscle pain and neurological manifestations. Unpleasant complications may also occur: rupture of the spleen, severe anemia, kidney failure, hypoglycemia, acute respiratory distress and endocardial fibrosis. In the case of infection with *P. ovale*, *P. vivax* and *P. malariae*, the recurrence of the disease may appear after weeks, months or also years. It is because these three species develop characteristic forms of the parasite existing in liver and called hypnozoites which have significantly slowed down metabolism and are activated sporadically, triggering recurrence of the disease. Malaria caused by the above three species of *Plasmodium* is in its clinical course milder than the one caused by *P. falciparum* or *P. knowlesi*, which do not produce hypnozoites. *P. falciparum* is responsible for the most severe course of malaria; it significantly impairs the performance of numerous internal organs of the body, causing their insufficiency. It causes capillary clogging which leads to the insufficient oxygenation of tissues, deficiency of nutritional substances and impairment of the transport of the metabolic products.

According to the WHO data in the year 2012 approximately 207 million cases of malaria were noted, while 627 thousand people died. The number of cases reported annually is very similar. The number of deaths caused by malaria in Africa represents 90% of all deaths due to the disease worldwide. A huge number of deaths concerns children [28].

Apart the children, increased risk of contracting malaria also concerns pregnant women, people with HIV, and people travelling to areas endangered by malaria.

The number of malaria cases in the world has decreased over the years. Moreover, in the past years countries recognized by the WHO as malaria-free have appeared, among them the United Arab Emirates from 2007, Morocco from 2010, Turkmenistan from 2010 and Armenia from 2011 [29]. Poland was recognized by the WHO as a malaria-free country already in 1963. In spite of that the disease continues to take a toll in the form of deaths and new cases, and it is still a very serious problem in the risk regions.

At present the greatest number of cases of malaria and deaths caused by this disease occurs in central and southern Africa [30]. This disease also occurs in Latin America and to a smaller extent in

Asia and Europe. The prevention of malaria depends primarily on use of insecticides, repellents, mosquito nets and prophylactic drugs. At the moment there is no vaccination against this parasitic disease.

Babesiosis is a parasitic enzootic disease triggered by the protozoan *Babesia* parasitizing erythrocytes of vertebrates, including humans. The intermediate hosts are mammals, and the final host is a tick. The life cycle of *Babesia* spp. is presented in Fig. 2 [3].

The vectors enabling transmission of the parasite are ticks from the family Ixodidae [31]. They prefer woodland and grassy areas, meadows, moors, pastureland and city parks. For the huge number of babesiosis cases in people in Europe, ticks from the species *Ixodes ricinus* are responsible [32]. This is a cosmopolitan species. Its distribution includes Scandinavia, the British Isles, Central Europe, France, Italy, Spain, East Europe, Bosnia and Herzegovina and the Czech Republic. The species strongly associated with the United States is *Ixodes*

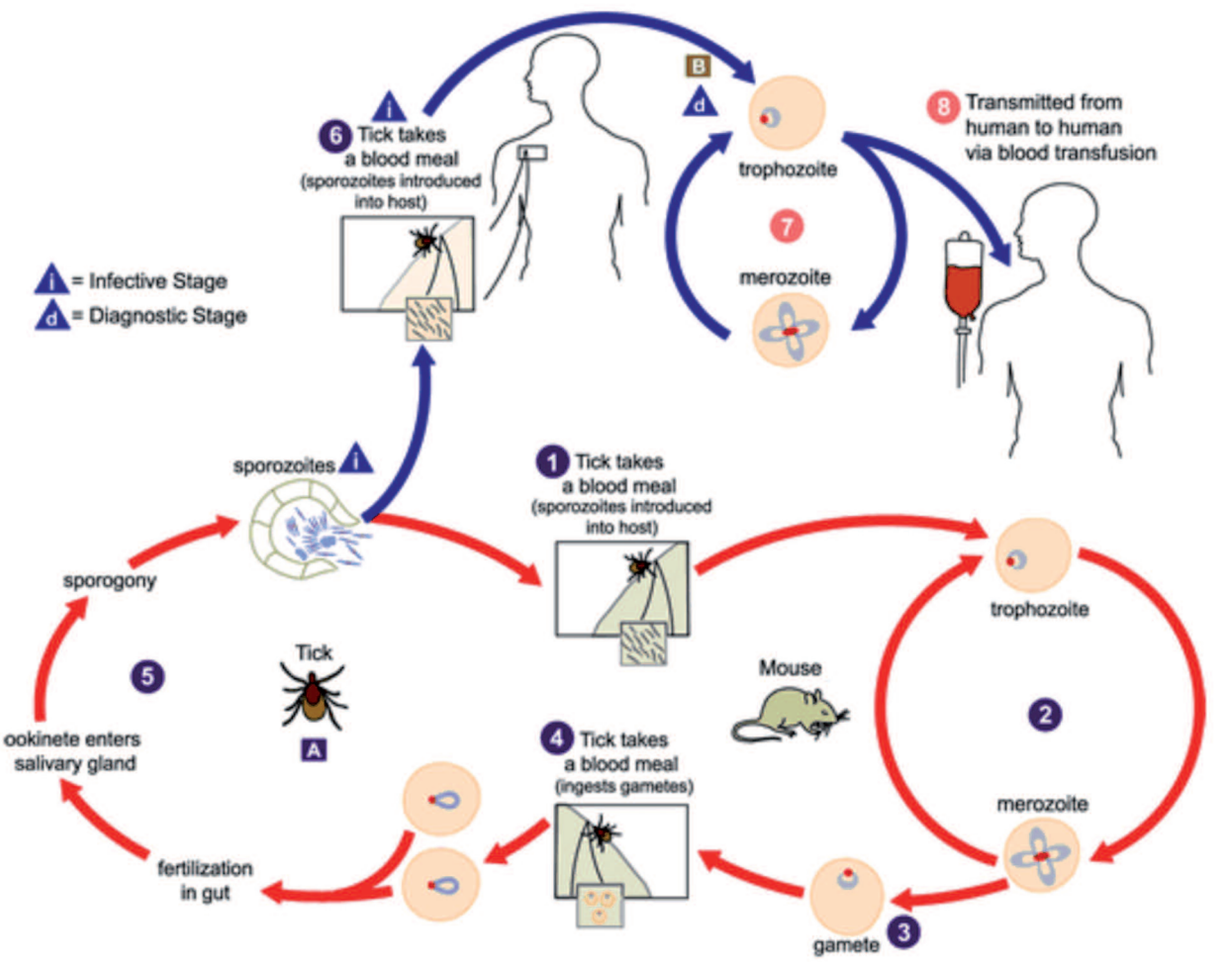

Fig. 2. Life cycle of *Babesia* spp. The *Babesia microti* life cycle involves two hosts, which includes a rodent, primarily the white-footed mouse, *Peromyscus leucopus*, and a tick in the genus, *Ixodes*. During a blood meal, a *Babesia*-infected tick introduces sporozoites into the mouse host 1. Sporozoites enter erythrocytes and undergo asexual reproduction (budding) 2. In the blood, some parasites differentiate into male and female gametes although these cannot be distinguished at the light microscope level 3. The definitive host is the tick. Once ingested by an appropriate tick 4, gametes unite and undergo a sporogonic cycle resulting in sporozoites 5. Transovarial transmission (also known as vertical, or hereditary, transmission) has been documented for „large" *Babesia* spp. but not for the „small" babesiae, such as *B. microti* A.

Humans enter the cycle when bitten by infected ticks. During a blood meal, a *Babesia*-infected tick introduces sporozoites into the human host 6. Sporozoites enter erythrocytes B and undergo asexual replication (budding) 7. Multiplication of the blood stage parasites is responsible for the clinical manifestations of the disease. Humans are, for all practical purposes, dead-end hosts and there is probably little, if any, subsequent transmission that occurs from ticks feeding on infected persons. However, human to human transmission is well recognized to occur through blood transfusions 8 [3].

scapularis, which occurs in the North-East and Central-East areas of the USA [33]. A significant number of cases in the United States is also diagnosed in the areas of New England [34], and the species responsible for the most cases of babesiosis infection in the USA is *Babesia microti* [35]. For example, in 2012 in several states in total approximately 1000 cases of infection with *Babesia microti* were reported, which shows that this protozoan is a relatively significant pathogenic factor in present times and one that requires attention.

In Europe most cases are caused by the species *Babesia divergens* [36]. Infection in a human occurs at the moment when the vector (an infected tick) takes the blood of a vertebrate. Together with the vector's saliva sporozoites enter the human's body, penetrate the red blood cells, proliferate and then cause lysis of erythrocytes and penetrate further ones [36]. Ticks become infected while drawing blood, and then in their organisms in various tissues and cells (various muscles, ovaries and intestinal epithelium) sexual reproduction of the parasite takes place, and then a new generation of sporozoites comes into being which are ready to invade the next hosts.

The clinical manifestations of babesiosis depend mainly on the species of a protozoan. Infections caused by *Babesia divergens* are characterized by more severe and quicker course. They often lead to

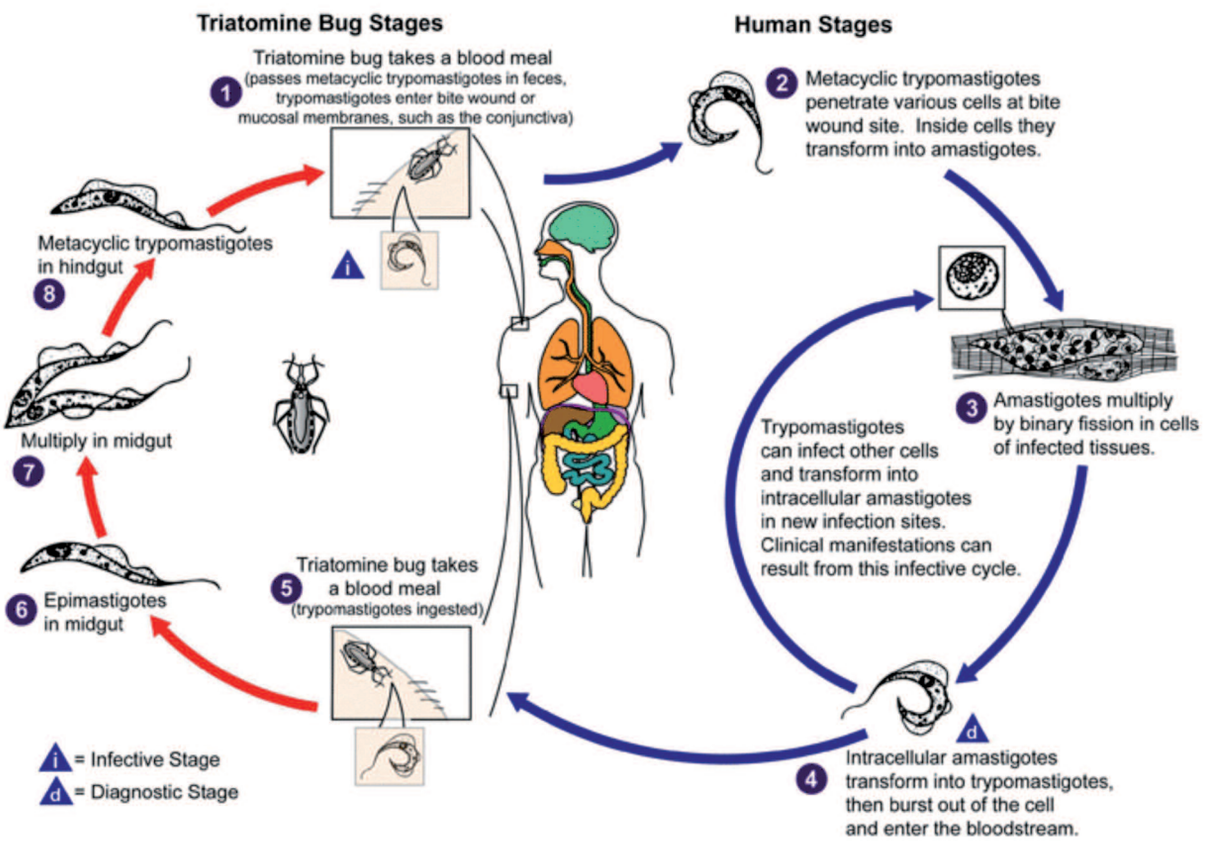

Fig. 3. Life cycle of *Trypanosoma cruzi*. Trypomastigotes enter the host through the wound or through intact mucosal membranes, such as the conjunctiva ❶. Common triatomine vector species for trypanosomiasis belong to the genera *Triatoma*, *Rhodnius*, and *Panstrongylus*. Inside the host, the trypomastigotes invade cells near the site of inoculation, where they differentiate into intracellular amastigotes ❷. The amastigotes multiply by binary fission ❸ and differentiate into trypomastigotes, and then are released into the circulation as bloodstream trypomastigotes ❹. Trypomastigotes infect cells from a variety of tissues and transform into intracellular amastigotes in new infection sites. Clinical manifestations can result from this infective cycle. The bloodstream trypomastigotes do not replicate (different from the African trypanosomes). Replication resumes only when the parasites enter another cell or are ingested by another vector. The „kissing" bug becomes infected by feeding on human or animal blood that contains circulating parasites ❺. The ingested trypomastigotes transform into epimastigotes in the vector's midgut ❻. The parasites multiply and differentiate in the midgut ❼ and differentiate into infective metacyclic trypomastigotes in the hindgut ❽ [3].

death. Invasions by *Babesia microti* are significantly milder in their course and are usually characterized by fever and body weakness which pass on their own. Many people infected with *Babesia* do not experience any signs of disease.

The most frequent symptoms of babesiosis, if they appear at all, develop from one week to a few months and these are: fever, shivering, sweating, headaches, loss of appetite and general fatigue [37].

One can become infected with babesiosis also by blood transfusion. People who are exposed to an acute course of the disease are older persons, persons with a weakened immune system and individuals who have had their spleen removed. The prevention of the disease consists first of all in avoiding areas where ticks occur, especially in warm months, using repellents, and wearing appropriate clothes covering the body. There is no vaccination which would protect against babesiosis.

Chagas disease, also known as American trypanosomiasis, is a tropical parasitic life-threatening disease caused by the trypanosome *Trypanosoma cruzi*, transmitted into people by blood-sucking Hemiptera of the subfamily Triatominae [38] belonging to the genera *Triatoma*, *Panstrongylus* and *Rhodnius*. These insects are called "kissing bugs" because they bite their victims mainly in the mouth area. The disease is spread through contact with the insects' excreta. The life cycle of *Trypanosoma cruzi* is presented in Fig. 3 [3].

This disease occurs mainly in Central and South America, and its range encompasses 21 countries [39]. In recent times it has been more and more often detected in the United States [40].

The most efficient for transmitting *Trypanosoma cruzi* into people are such species of Hemiptera as: *Triatoma infestans*, responsible for the most cases in Bolivia, Argentina, Uruguay, Paraguay, Chile, Brazil and Peru; *Rhodnius prolixus*, occurring in the whole of Central and South America [41]; *Rhodnius pallescans*, being the main vector of Chagas disease in Central America, mainly in Costa Rica and Panama [42]; *Triatoma brasilliensis*, being the main vector in Brazil; *Triatoma dimidata*, occurrence of which spreads from the north of South America (Columbia, Venezuela, Equador and Peru) up to all countries of Central America, and also southern Mexico; *Panstrongylus megistus*, *Triatoma sordida* and *Triatoma pseudomaculata*, occurring in various areas of South America [43], and *Triatoma protracta*, occurring in the south-eastern part of the USA [44].

The insect pierces the skin and defecates close to the arisen wound. Trypanosomes access the body when the human instinctively spreads the insect's excreta across the wound or any other skin damage. It is also possible to become infected via mucosa when rubbing the eyes. The invasive forms, trypomastigotes, which are transmitted with blood, access the body and penetrate the host's cells (of the reticuloendothelial system, neuroglia, skeletal muscles, smooth muscles and also cardiac muscle), in which trypomastigotes turn into amastigotes. The infected cells undergo lysis, and the liberated forms of amastigotes invade the next cells of various tissues of the host or turn into the trypomastigote form and are ingested by Hemiptera while sucking the host's blood. In the body of a hemipteran the parasite gains the ability to infect further hosts through creating appropriate invasive forms.

It is also possible to become infected by the intake of food contaminated with excreta of *Triatoma* [45], and by blood transfusions or transplantations, but these are much rarer situations.

Hemiptera transmitting Chagas disease occur first of all in poor rural areas. They nest mainly in the holes and cracks in walls, and also roofs made of bamboo and sugar cane. They forage at night.

The disease has two stages. During the acute, initial phase, which may last a few months, the number of parasites living in the blood circulation is significant. In most cases the symptoms are mild or non-existent in this phase. If present, they frequently include fever, headaches, enlarged lymph nodes, pallor, muscular pain, difficulty when breathing, swelling, and pain in the abdominal or chest areas. In a small number of people bitten by Hemiptera the first characteristic symptom may be swelling in the bite area or purple swelling of one of the eyelids.

During the second, chronic stage of Chagas disease the parasites colonize first of all the heart and muscles of the alimentary system. 20–40% of infected people suffer from cardiac disorders, and 15–20% from alimentary system disorders, including serious enlargement and dilation of the colon and esophagus [46]. Complications connected with the nervous systems may also appear. Untreated disease may lead in further years to sudden death or serious cardiovascular complications.

Prevention of spreading the American trypanosomiasis consists mainly in controlling vectors, which comprises spraying houses and areas

where Hemiptera live, using mosquito nets, and ensuring personal hygiene and hygiene while preparing and storing meals. The increased inspection of blood during transfusions should be performed, and also organ donors should be checked thoroughly before transplantations in areas of high risk of the disease.

In recent years American trypanosomiasis has appeared in places recognized earlier as disease-free, i.e. in some areas of the Amazon Basin. There are also alarming reports about the recurrence of trypanosomiasis in areas where the vectors' control has so far produced effects, e.g. Chaco region in Bolivia [47].

Combating the disease generates considerable costs, and its complete extermination is practically impossible due to the high number of wild animals constituting a reservoir for *Trypanosoma cruzi*. According to the latest WHO data, approximately 8 million people are currently infected. There is no vaccination against this disease.

Sleeping sickness, also known as African trypanosomiasis, is a dangerous, life-threatening, tropical parasitic disease triggered by trypanosomes belonging to protozoans. Two species of trypanosomes are responsible for development of the disease in people – *Trypanosoma brucei gambiense* and *Trypanosoma brucei rhodesiense* – and the vectors transmitting the parasites are approximately 20 species of tsetse flies (*Glossina*) inhabiting tropical and subtropical Africa. Tsetse flies inhabit fields and rural areas. They like forests, savannas, and riparian areas. They need a specific temperature range of 16–38°C and humidity of 50%–80% to survive and reproduce [48]. These insects bite during the daytime. The life cycle of

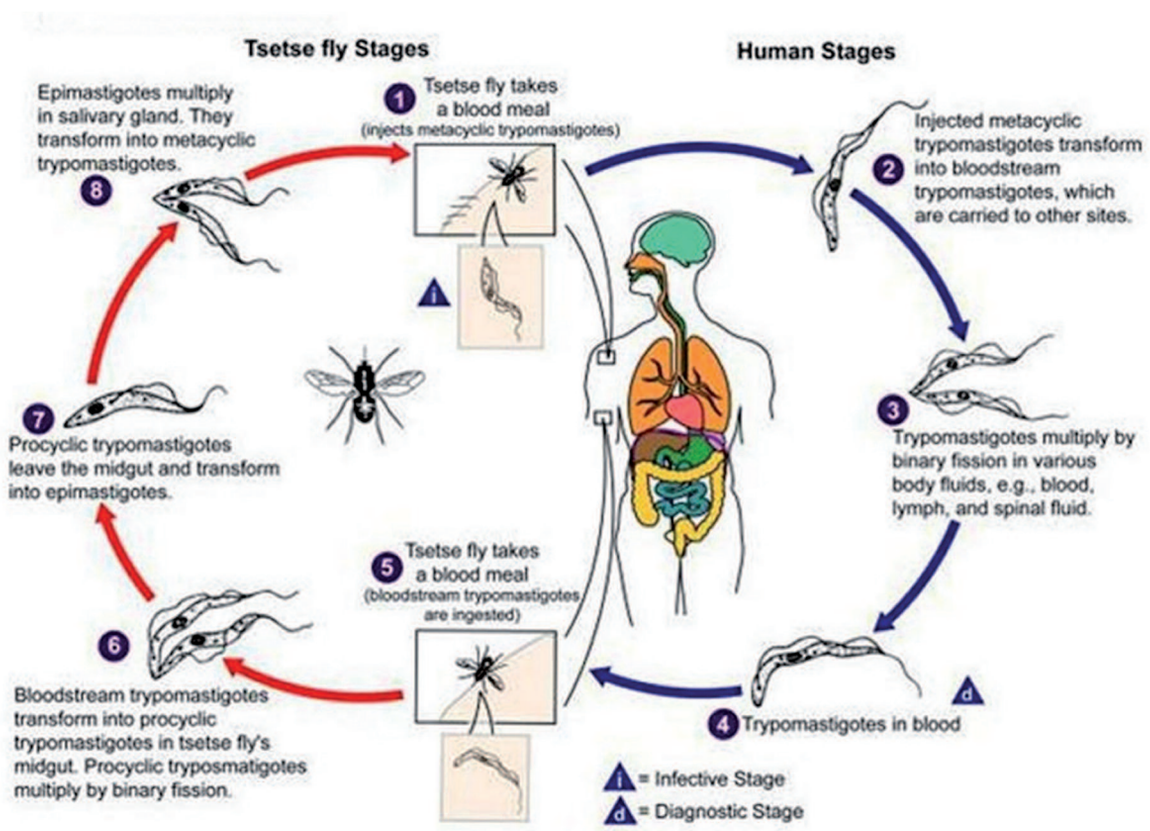

Fig. 4. Life cycle of *Trypanosoma brucei.* During a blood meal on the mammalian host, an infected tsetse fly (genus *Glossina*) injects metacyclic trypomastigotes into skin tissue. The parasites enter the lymphatic system and pass into the bloodstream ❶. Inside the host, they transform into bloodstream trypomastigotes ❷, are carried to other sites throughout the body, reach other blood fluids (e.g., lymph, spinal fluid), and continue the replication by binary fission ❸. The entire life cycle of African trypanosomes is represented by extracellular stages. The tsetse fly becomes infected with bloodstream trypomastigotes when taking a blood meal on an infected mammalian host ❹, ❺). In the fly's midgut, the parasites transform into procyclic trypomastigotes, multiply by binary fission ❻, leave the midgut, and transform into epimastigotes ❼. The epimastigotes reach the fly's salivary glands and continue multiplication by binary fission ❽. The cycle in the fly takes approximately 3 weeks. Humans are the main reservoir for *Trypanosoma brucei gambiense*, but this species can also be found in animals. Wild game animals are the main reservoir of *T. b. rhodesiense* [3].

Trypanosoma brucei is presented in Fig. 4 [3].

Trypanosoma brucei gambiense has been detected in 24 countries of West and Central Africa [49]. According to the most recent data, this species of trypanosome is responsible for 98% of reported cases of sleeping sickness and causes a chronic infection. The species of flies responsible for transmitting this protozoan into people are *Glossina morsitans*, *Glossina palpalis*, *Glossina tachinoides* and *Glossina fuscipes* [48,50].

During the bite by the tsetse fly, parasitic forms – trypomastigotes – which reproduce asexually, enter the blood circulation (of various mammals, including humans) of a host. They can be found in the blood plasma, bone marrow, lymph nodes and cerebrospinal liquid. The vector draws blood together with the parasites. In its body further transformations take place after which the parasite reaches the appropriate stage to infect the hosts.

In African trypanosomiasis, 2 stages can be distinguished. During the first stage the parasites multiply in the subcutaneous tissue, blood and lymph. This stage is accompanied by fever, headache, joints ache and pruritus. In the second stage the parasites cross the blood-brain barrier and infect the central nervous system. Further symptoms such as a change of behavior, disorientation and problems with coordination appear. Finally the infected person falls into a coma which usually finishes with death [48].

A person may be infected for many months, or even years, without the appearance of characteristic symptoms of the disease. When the symptoms are discovered, it is very often too late for any treatment due to the advanced grade of trypanosomiasis development. At first the symptoms are mild: fever, headache, muscle ache and anxiety. Additionally skin itchiness, enlargement of lymph nodes and loss of weight may appear. Then, as a rule after 1-2 years, changes in behavior and frequent sleepiness during the daytime connected with sleeping disorders during the night appear. Partial paralyses may also appear and frequently loss of balance. Untreated infection lasts rarely longer than 6–7 years, killing usually within 3 years [51]. This variety of African trypanosomiasis is called the West African trypanosomiasis and it occurs mainly in Central Africa and in its western areas. This is the most common kind of this disease on the African Continent. Almost 98% of cases have been registered in the Democratic Republic of Congo, Sudan and Angola.

Trypanosoma brucei rhodesiense has been detected in 13 countries of East and West Africa [49]. This protozoan is presently responsible for approximately 2% of reported cases of trypanosomiasis and causes a more severe form of the disease. The species of flies responsible for transmitting this protozoan to people are *Glossina morsitans*, *Glossina swynnertoni*, *Glossina pallipides* and *Glossina fuscipes* [48]. The first symptoms can be noted a few weeks after becoming infected, and the disease progresses very fast. In most patients fever, headache, muscle ache and enlargement of the lymph nodes occur within 1–2 weeks after being bitten by a tsetse fly. In some people a rash appears. After a few weeks of infection the parasite attacks the central nervous system and causes changes in personality and other neurological problems. Death takes place as a rule within a few months [52]. The African trypanosomiasis caused by this parasite is called East African trypanosomiasis and mainly affects the areas of East Africa and South-East Africa, with 90% of cases having been recorded in such countries as Tanzania, Uganda, Malawi and Zambia.

It is also possible to become infected with trypanosomiasis by a contaminated needle or syringe. The parasite may also pass through the placenta, leading to infection of the fetus.

According to WHO data, sleeping sickness occurs in 36 countries of Central and South Africa [53]. Due to the intensified fight against the disease, in 2009 the number of new reported cases dropped below 10 000 for the first time in 50 years. The decreasing tendency continued and in 2012 reached approximately 7500 new cases [54]. It is estimated that the number of current cases is approximately 20 000 per year, whereas the number of people exposed currently to becoming infected is approximately 70 million. The country in which there are the most cases of this disease is the Democratic Republic of Congo. Over 75% of cases of this disease are recorded there, whereas almost all of the diagnosed cases are infections caused by *Trypanosoma brucei gambiense* [48].

Prevention of the disease consists in using insecticides, mosquito nets, repellents, traps and special screens attracting tsetse flies. It is more difficult to combat *Trypanosoma brucei rhodesiense* because this parasite occurs also in many mammals, which constitute a considerable reservoir of this parasite. There is no vaccination protecting against infection with the flagellate of the genus *Trypanosoma*.

Leishmaniases are a group of parasitic diseases dangerous for humans and triggered by over 20 species of flagellate of the genus *Leishmania* [55]. Single-celled parasites are transmitted by vectors belonging to two kinds of Diptera – *Phlebotomus* and *Lutzomyia* – whereas *Phlebotomus* occurs entirely in the Old World (Europe, Asia and Africa) and *Lutzomyia* occurs entirely in the New World (Central America and South America) [56]. Among approximately 800 species of flies of these two genera, approximately 100 play a more or less significant role for transmitting the protozoans of the genus *Leishmania*. These Diptera prefer mainly woodlands. They can be found in great numbers resting in the forest cover, tree hollows, rock cracks and also in caves [57]. They also occur on the African savannas, deserts, and sporadically also in mountain areas. They are found in rodents' burrows. They bite primarily at dusk, more rarely during the daytime. Poverty, lack of hygiene, sleeping outside, malnutrition (in particular the lack of iron, vitamin A and zinc), moving to areas where the flies occur, deforestation (natural environment of the Diptera) and building houses on the verges of forests – all these significantly increase the possibility to become infected with one of the forms of leishmaniasis.

The parasite requires for its development two hosts which are the insects (vectors) and mammals, including humans. The main reservoir of the protozoans are Canidae and rodents. The life cycle of *Leishmania* spp. is presented in Fig. 5 [3].

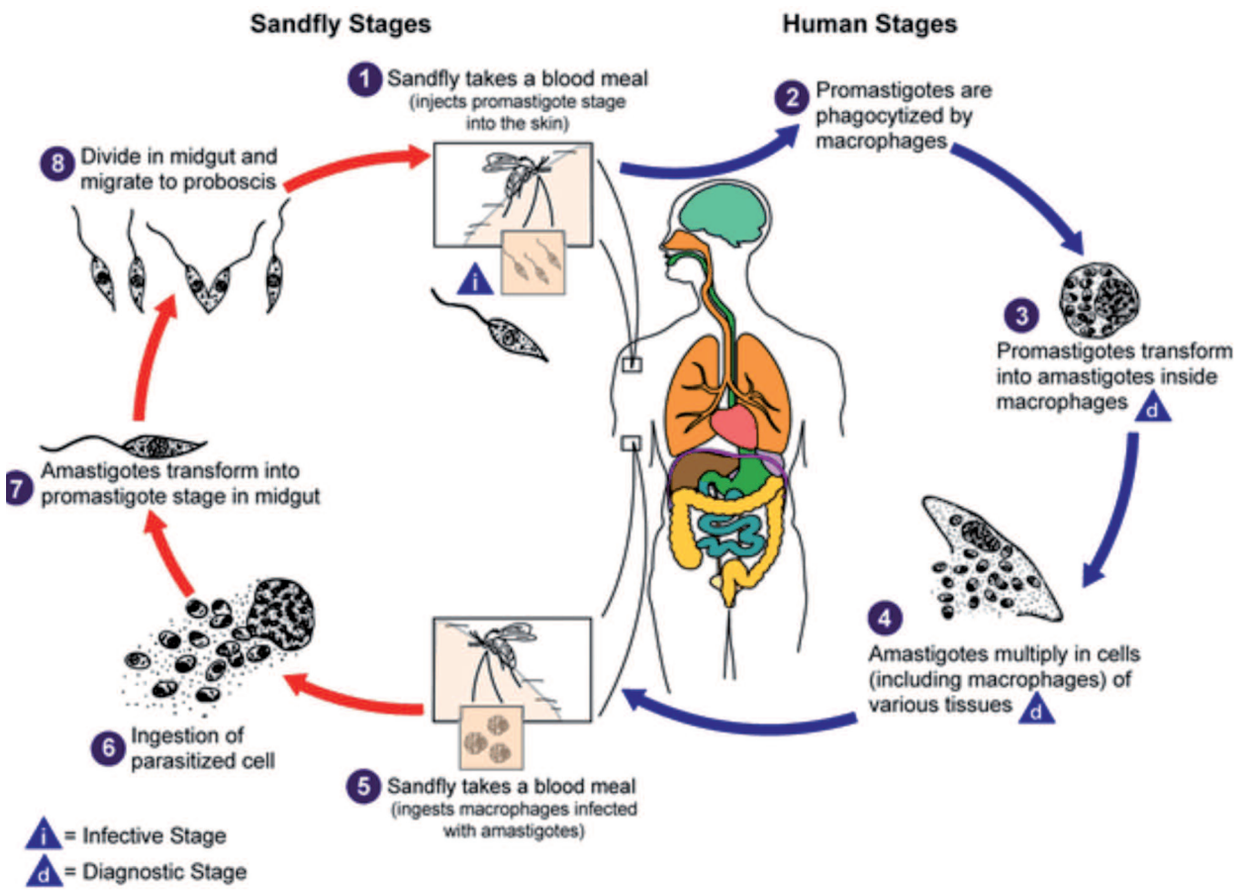

Fig. 5. Life cycle of *Leishmania* spp. Leishmaniasis is transmitted by the bite of infected female phlebotomine sandflies. The sandflies inject the infective stage (i.e., promastigotes) from their proboscis during blood meals❶. Promastigotes that reach the puncture wound are phagocytized by macrophages ❷ and other types of mononuclear phagocytic cells. Progmastigotes transform in these cells into the tissue stage of the parasite (i.e., amastigotes) ❸, which multiply by simple division and proceed to infect other mononuclear phagocytic cells ❹. Parasite, host, and other factors affect whether the infection becomes symptomatic and whether cutaneous or visceral leishmaniasis results. Sandflies become infected by ingesting infected cells during blood meals (❺, ❻). In sandflies, amastigotes transform into promastigotes, develop in the gut ❼ (in the hindgut for leishmanial organisms in the *Viannia* subgenus; in the midgut for organisms in the *Leishmania* subgenus), and migrate to the proboscis ❽ [3].

Leishmaniases are mainly zoonoses, but there are regions in the world where human-vector-human infection occurs. Such a region is for instance India, a country in which the reservoir of the parasite is first of all humans [58].

Infection with the flagellate takes place when the female Diptera bites and sucks the blood of a human. The person's body is entered by invasive forms promastigotes, which are phagocytosed by macrophages and lose their flagellum and transform into amastigotes which reproduce by division. Macrophages become destroyed, and the liberated forms, amastigotes, are phagocytosed by the next macrophages, which can access the reticulo-endothelial system (bone marrow, spleen and liver) through the blood vessels [59]. The infected tissues become seriously damaged. The female Diptera *Phlebotomus* and *Lutzomyia* become infected by the amastigotes during the next blood sucking of the host. Amastigotes transform in the midgut of the insect into promastigotes (invasive for humans) and wander to the insect's throat, causing its blockage. The insect vomits while sucking the host's blood, which enables the parasite to enter the human's body. It is also possible to become infected by squashing the insect and rubbing it in the wound.

The infected person may be affected by one of the three forms of the disease: visceral leishmaniasis (variously called kala-azar, black fever or dum-dum fever), which is the most serious form of the disease; cutaneous leishmaniasis (white leprosy), which is the most common form of the disease; and mucocutaneous leishmaniasis [60].

One of the main vectors of the visceral leishmaniases is the sandfly *Lutzomyia longipalpis* (transmitting mainly *Leishmania infantum*) [61], and it can be found in all countries of Central America and in most countries of South America [62]. Protozoans causing this variety of the disease are most frequently *Leishmania donovani*, *Leishmania infantum*, *Leishmania tropica* and *Leishmania amazonensis* [63]. The following species of sandflies are responsible for the occurrence of this variety of the disease: *Phlebotomus langeroni orientalis* (occurring in Sudan), *P. martini* (Kenya/Ethiopia), *P. syriacus* (Palestinian region), *P. neglectus* (Greece), *P. argentipes* (India, Nepal and Bangladesh), *P. perniciosus* and *P. ariasi* (Morocco), *P. langeroni* (Egypt), *P. alexandri*, *P. chinensis*, *P. longiductus* (China), and *P. orientalis* (the main vector of the northern savannas of the East Africa) [63].

The most cases of death due to the visceral leishmaniases are noted in Brazil and India. When untreated, it leads to death of 80–90% of infected people within 1-2 years. The symptoms start 2–8 months after the bite by the female sandfly. They are characterized by irregular recurrences of fever, profuse sweating, nausea, loss in body weight, spleen and liver enlargement and anemia. Skin may blacken; permanent changes in pigmentation may occur. According to WHO data, every year there are recorded approximately 200 000–400 000 thousand cases of this disease in the world (WHO 2015). Over 90% of cases are recorded in Bangladesh, Brazil, Ethiopia, India and Sudan [63].

One of the main vectors of cutaneous leishmaniasis is the sandfly *Phlebotomus papatasi* (transmitting mainly *Leishmania major*) [65], which occurs mainly in central, northern and eastern Africa, India, southern Europe and Morocco [66]. Protozoans most often causing this variety are *Leishmania donovani*, *Leishmania major* (also transmitted by *Phlebotomus duboscqi*), *Leishmania aethiopica*, *Leishmania mexicana* (also transmitted by *Lutzomyia anthophora*), and *Leishmania brasiliensis* (also transmitted by *Lutzomyia intermedia*) [67]. Moreover, such species of sandfly as *Lutzomyia olmeca* (occurring in Belize and Mexico), *Lutzomyia whitmani* and *Lutzomyia intermedia* (Brazil), *Lutzomyia vallesi* (Venezuela), and *Phlebotomus sergentii* (Palestinian areas) are responsible for the occurrence of this variety of the disease [68].

Cutaneous leishmaniasis causes skin ulcers of various parts of the body, often leaving scars for the whole life. In the places bitten by the female sandfly a bump (sore) appears which enlarges with time, bursts and becomes an open wound. The disease escalates within a few weeks. Significantly more wounds (sores) may appear. The wounds heal usually within a few months, but sometimes they may remain for years. They are painless unless an additional infection develops. The lesions affect mainly the neck, face and legs. According to WHO data, approximately 95% of cases occur in Central and South America, in the Mediterranean Basin, the Middle East and also in Central Asia. Two thirds of cases occur in Afghanistan, Algeria, Brazil, Columbia, Iran and Syria [69]. Every year approximately 1,3 million cases are diagnosed. There has been recently recorded a serious increase in incidence of cutaneous leishmaniasis in South Brazil, which is caused by deforestation and

construction of houses near to the border of woodlands. With regard to deforestation in Brazil, 3 species of sandflies (*Lutzomyia intermedia*, *Lutzomyia whitmani* and *Lutzomyia migonei*) which inhabit primarily forests have started to attack people approaching them [70].

Various species of sandflies of the genus *Lutzomyia* are responsible for cases of the next form of leishmaniasis, i.e. mucocutaneous leishmaniasis. Flagellates most frequently causing this variety of the disease are *Leishmania brasiliensis*, *Leishmania guyanensis* (often transmitted by *Lutzomyia umbratilis*, which is also one of the more important vectors for *Leishmania brasiliensis*), *Leishmania panamensis* (transmitted by *Lutzomyia panamensis*) and *Leishmania peruviana* (transmitted by *Lutzomyia peruensis*) [71].

This variety often leads to partial or entire destruction of the mucous membrane, and deformation of the nose, lips and throat [71]. According to the WHO, almost 90% of cases occur in Bolivia, Brazil and Peru. According to WHO data, approximately 1,5 million new cases are recorded per year, and 20 000 to 30 000 people die [69]. The prevention of leishmaniasis consists mainly in using insecticides, mosquito nets and special nets around beds which the insects cannot penetrate. Similar to other parasitic tropical diseases, there is no vaccination against this disease.

Diseases caused by nematodes

Filariases are a group of human diseases caused by parasitic species of nematodes such as *Wuchereria bancrofti*, *Brugia malayi*, *Brugia timori* (causing lymphatic filariases), *Onchocerca volvulus*, and *Loa loa* (causing cutaneous filariases) [72,73]. In the case of the lymphatic filariases, vectors transmitting invasive larvae of nematodes are a few species of mosquitoes, namely *Anopheles*, *Aedes*, *Culex* and *Mansonia* [74], whereas in the case of the cutaneous filariases, vectors of the larvae of nematodes *Onchocerca volvulus* are black flies of the genus *Simulium*, and the vectors of *Loa loa* larvae are horse-flies of the genus *Chrysops* [75,76].

The life cycles of the three parasites (*W. bancrofti*, *Brugia malayi*, *Brugia timori*) causing lymphatic filariases are almost identical, and the nematodes vary among one other mainly in their morphological features. The intermediate host is a mosquito, while the final host is a human. The human becomes infected during drawing of blood by the infected female mosquito. During the drawing, the invasive larvae of the nematode penetrate the person's blood and lymphatic vessels. They reach the stage of the mature form after a few months. Mature forms, living a few years, inhabit the lymphatic nodes where the females of parasitic nematodes give birth to great number of larvae called microfilariae [77]. These in turn migrate to the blood and lymphatic vessels, appearing in the peripheral blood at night, due to which female mosquitoes – biting mainly after dusk and at night – may draw them for the life cycle of the parasite to proceed. After being drawn with the blood by an appropriate intermediate host, microfilariae develop in a mosquito into invasive forms that are able to infect the next, final hosts. The life cycle of *Wuchereria bancrofti* is presented in Fig. 6 [3].

Infection occurs most often in childhood, and its visible, external symptoms appear later in life, causing temporary or permanent dysfunctions. Many people have no symptoms, but all infected individuals have an impaired immune system, and 40% of them have disorders of kidney function [78]. Lymphatic filariases are also called elephantiases. The name is taken from the symptoms which occur in persons infected with nematodes. The lesions can cause significant enlargement and deformation of parts of the body (legs, genitals, breast and lymphatic nodes) [79]. This is caused by the accumulation of great numbers of larvae in lymphatic nodes and lymphatic vessels, due to which the lymph has great difficulties to flow, it becomes detained in various parts of the body and swelling of cells occurs. These symptoms are accompanied by pain, fever, inability of normal functioning, serious dysfunctions of the body and serious mental trauma.

The main and most common cause of the lymphatic filariases is the nematode *Wuchereria bancrofti* [80], which is responsible for 90% of cases [81]. It is distributed mainly in the countries of tropical and subtropical zones of Africa, and also Asia, as well as South and Central America. The most common vectors of *Wuchereria bancrofti* are mosquitoes of the genus *Anopheles*, among which the main species transmitting the nematode are *Anopheles arabiensis*, *A. bancroftii*, *A. farauti*, *A. funestus*, *A. gambiae*, *A. melas*, *A. merus* and *A. punctulatus* [82–84]. The main vectors of the genus *Culex* for *W. bancrofti* are *C. quinquefasciatus* and *C. pipiens* [85,86]. The main vectors of the genus *Aedes* for *W. bancrofti* are *A. aegypti*, *A. aquasalis*,

A. bellator, *A. cooki*, *A. darlingi*, *A. kochi*, *A. polynesiensis*, *A. pseudoscutellaris*, *A. rotumae*, *A. scapularis* and *A. vigilax* [87]. The main vectors of the genus *Mansonia* for *W. bancrofti* are *Mansonia titillans* and *M. uniformis* [88].

Almost all remaining 10% of the cases of lymphatic filariases are due to the nematode *Brugia malayi*. It is distributed mainly in Asia, mainly in the South of China, India, Indonesia, Thailand, Vietnam, Malaysia, the Philippines and South Korea. The main vectors transmitting this nematode are mosquitoes of the genus *Mansonia*. The species of the genus *Mansonia* most often transmitting the parasitic nematode are *Mansonia annulifera*, *M. indiana*, *M. pudlica*, *M. annulata*, *M. uniformis*, *M. bonneae* and *M. dives* [89].

The third parasite causing lymphatic filariases is *Brugia timori*, responsible for the fewest cases. This parasite occurs only in Indonesia, mainly in Nusa Tenggara. The main vector of this parasite is the mosquito *Anopheles barbirostris*, which multiplies in rice fields and feeds on the hosts' blood at night time [90].

According to the WHO, approximately 120 million people in the world are presently infected by nematodes causing lymphatic filariases, among whom 40 million have visible physical changes on the body, as well as significant dysfunctions of the body. A great risk of becoming ill occurs in particular in 73 countries where the disease is easily spread. Countries in which the disease takes its toll in the form of infected people are Bangladesh, the

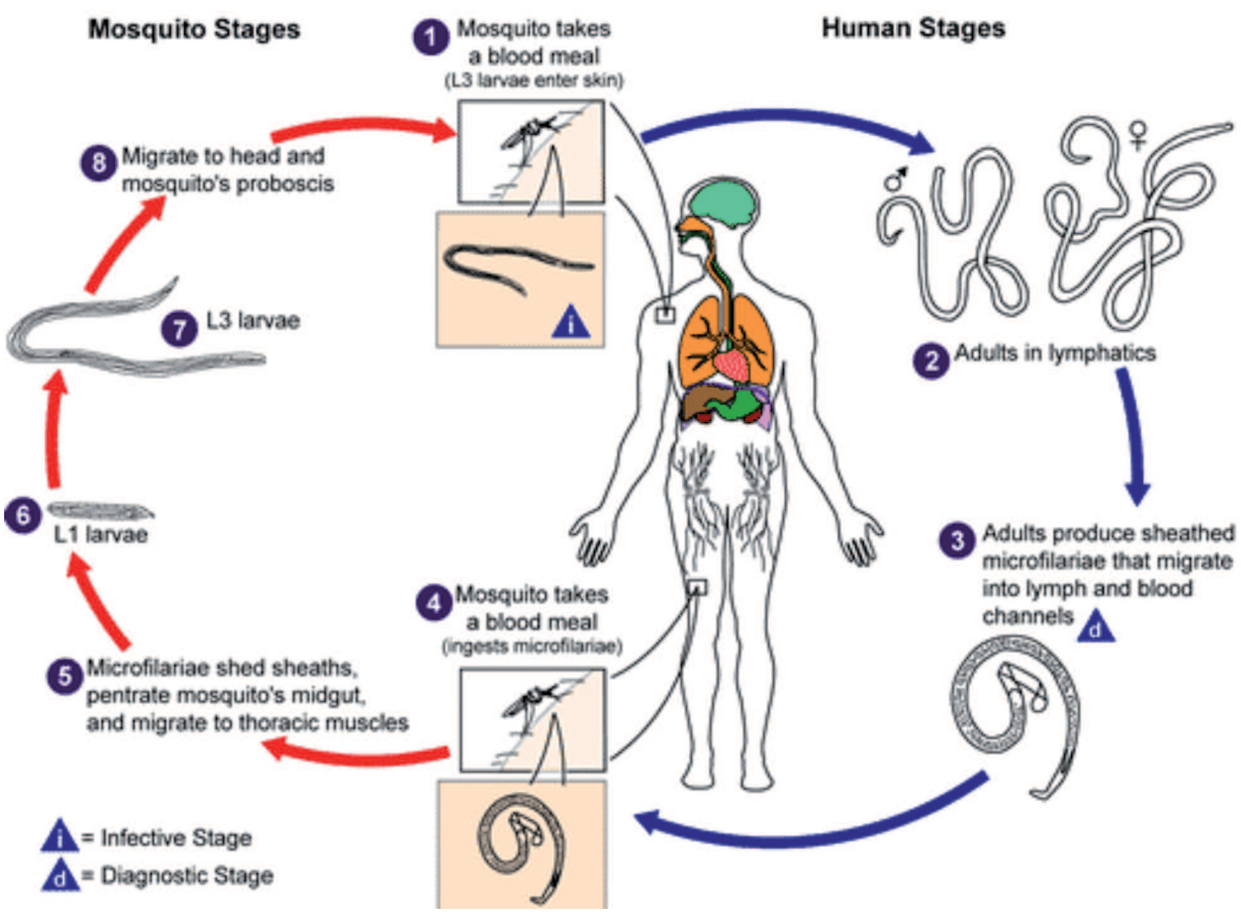

Fig. 6. The life cycle of *Wuchereria bancrofti.* During a blood meal, an infected mosquito introduces third-stage filarial larvae onto the skin of the human host, where they penetrate into the bite wound ❶. They develop in adults that commonly reside in the lymphatics ❷. Adults produce microfilaria, which are sheathed and have nocturnal periodicity, except the South Pacific microfilariae which have the absence of marked periodicity. The microfilariae migrate into lymph and blood channels moving actively through lymph and blood ❸. A mosquito ingests the microfilariae during a blood meal ❹. After ingestion, the microfilariae lose their sheaths and some of them work their way through the wall of the proventriculus and cardiac portion of the mosquito's midgut and reach the thoracic muscles ❺. There the microfilariae develop into first-stage larvae ❻ and subsequently into third-stage infective larvae ❼. The third-stage infective larvae migrate through the hemocoel to the mosquito's prosbocis ❽ and can infect another human when the mosquito takes a blood meal ❶ [3].

Democratic Republic of Congo, Ethiopia, Indonesia, Myanmar, Nigeria, Nepal, the Philippines and Tanzania [91].

Prevention of lymphatic filariases consists in using appropriate repellents and mosquito nets. There is no vaccination preventing this disease. In advanced stages of elephantiasis surgery is frequently performed. Also physical exercise is recommended, enabling the chyle to flow in the vessels of the lymphatic system.

Onchocerciases and loaiases are caused by the parasitic nematodes *Onchocerca volvulus* and *Loa Loa*. They trigger skin filariases. The life cycle of these parasites are presented in Fig. 7 and Fig. 8 [3].

Onchocerca volvulus triggers a dangerous human disease called onchocerciasis or river blindness [92]. Vectors responsible for transmitting onchocerciasis are black flies of the genus *Simulium*. These insects develop by fast flowing streams and rivers.

Simulium species most frequently transmitting *O. volvulus* include the following: *S. damnosum*, distributed in the whole Sub-Saharan Africa and in the Arabian Peninsula, and which is the main vector in Uganda, Benin, Burkina Faso, Cameroon, Ghana, Guinea, the Ivory Cost, Liberia, Mali, Nigeria, the Republic of Niger, Senegal, Sierra Leone, Sudan and Togo; *S. exiguum*, the main vector in Columbia

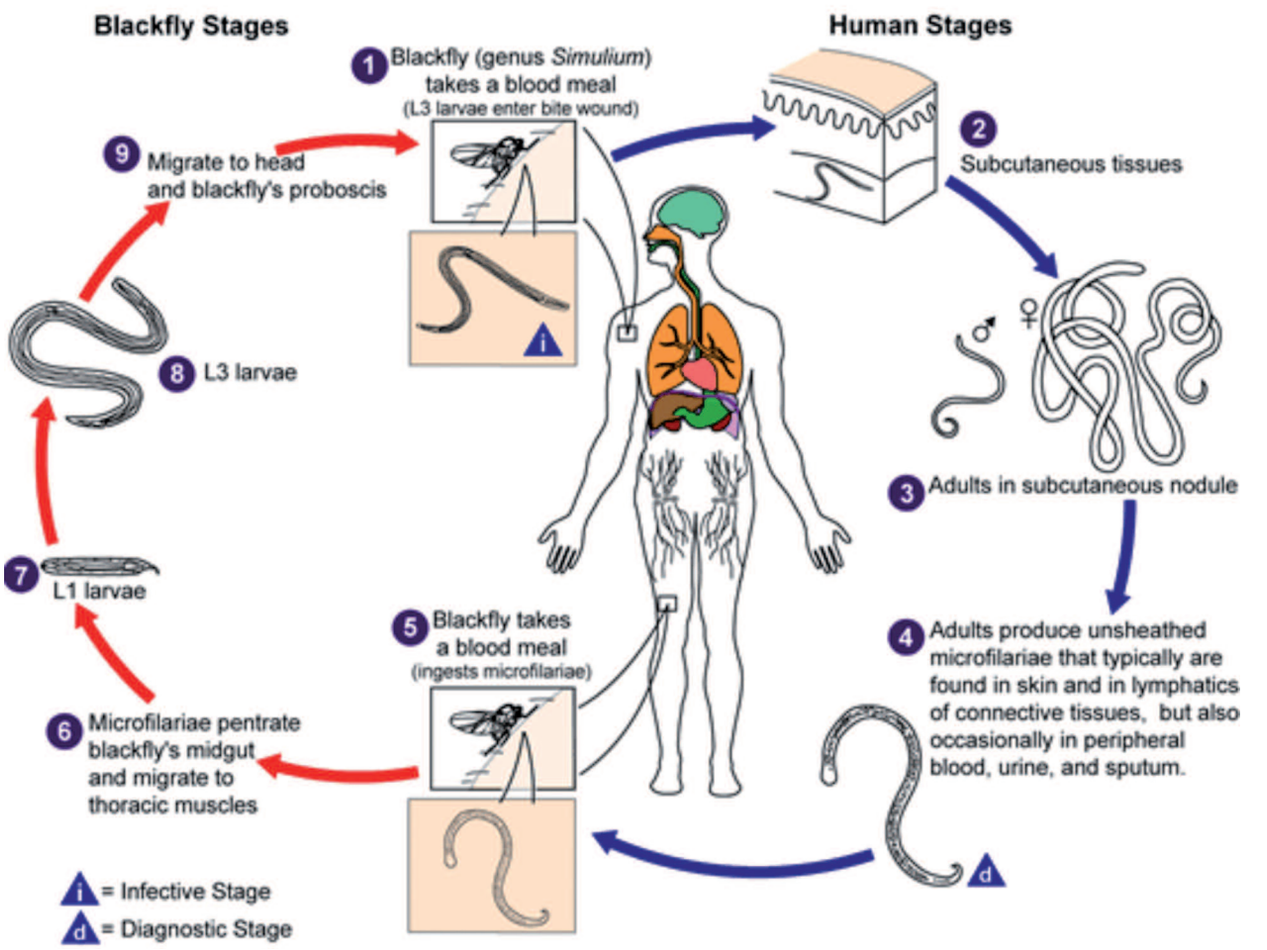

Fig. 7. The life cycle of *Onchocerca volvulus.* During a blood meal, an infected blackfly (genus *Simulium*) introduces third-stage filarial larvae onto the skin of the human host, where they penetrate into the bite wound ❶. In subcutaneous tissues the larvae ❷ develop into adult filariae, which commonly reside in nodules in subcutaneous connective tissues ❸. Adults can live in the nodules for approximately 15 years. Some nodules may contain numerous male and female worms. In the subcutaneous nodules, the female worms are capable of producing microfilariae for approximately 9 years. They are occasionally found in peripheral blood, urine, and sputum but are typically found in the skin and in the lymphatics of connective tissues ❹. A blackfly ingests the microfilariae during a blood meal ❺. After ingestion, the microfilariae migrate from the blackfly's midgut through the hemocoel to the thoracic muscles ❻. There the microfilariae develop into first-stage larvae ❼ and subsequently into third-stage infective larvae ❽. The third-stage infective larvae migrate to the blackfly's proboscis ❾ and can infect another human when the fly takes a blood meal ❶ [3].

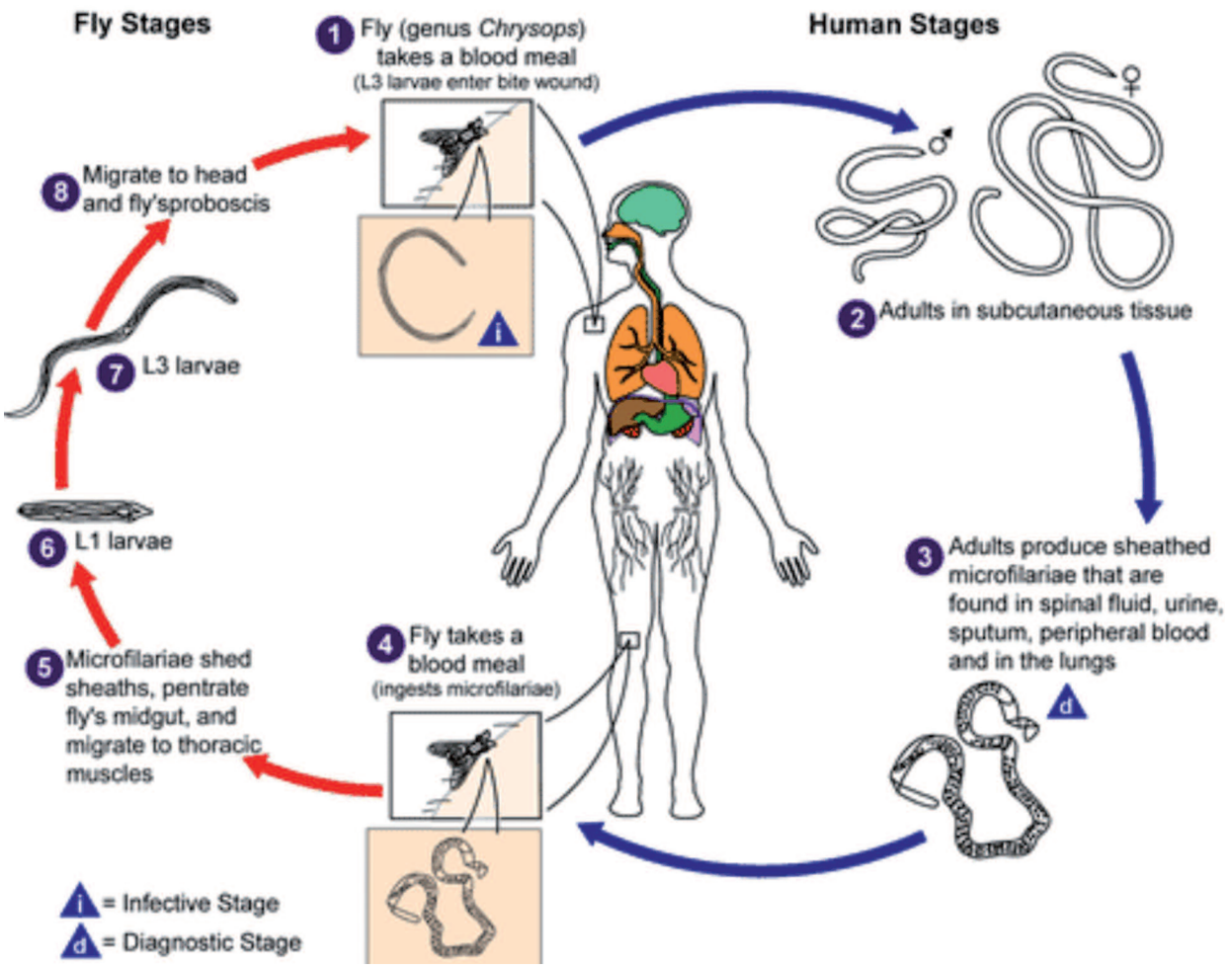

Fig. 8. The life cycle of *Loa loa.* During a blood meal, an infected fly (genus *Chrysops*, day-biting flies) introduces third-stage filarial larvae onto the skin of the human host, where they penetrate into the bite wound ❶. The larvae develop into adults that commonly reside in subcutaneous tissue ❷. Adults produce microfilariae, which are sheathed and have diurnal periodicity. Microfilariae have been recovered from spinal fluids, urine, and sputum. During the day they are found in peripheral blood, but during the noncirculation phase, they are found in the lungs ❸. The fly ingests microfilariae during a blood meal ❹. After ingestion, the microfilariae lose their sheaths and migrate from the fly's midgut through the hemocoel to the thoracic muscles of the arthropod ❺. There the microfilariae develop into first-stage larvae ❻ and subsequently into third-stage infective larvae ❼. The third-stage infective larvae migrate to the fly's proboscis ❽ and can infect another human when the fly takes a blood meal ❶ [3].

and Ecuador, also distributed in Venezuela, Bolivia, Argentina, Brazil, Guatemala, Guiana, Mexico, Panama and Peru; *S. ochraceum*, the main vector in Central America, and in particular in Guatemala, Mexico, also occuring in Columbia, Ecuador, French Guiana, Guadalupe, Peru, Venezuela and in the Virgin Islands; *S. sanctipauli*, distributed in the areas of the Ivory Coast, Ghana, Guinea, Liberia, Mali, Nigeria, Sierra Leone and Togo; *S. sirbanum* and *S. squamosum*, occurring in the areas of Niger, Benin, Burkina Faso, Cameroon, Central African Republic, Ethiopia, Ghana, Ivory Coast, Mali, Nigeria, Sierra Leone, Sudan, Togo and Uganda; *S. thyolense* and *S. woodi*, distributed in Malawi and Tanzania; and *S. yahense*, occurring primarily in the areas of Liberia, Benin, Cameroon, Ghana, Guinea, Ivory Coast, Nigeria, Sierra Leone and Togo [41,93].

Infection occurs when the female *Simulium* sucks human blood. A human is here the final host, and the vector an intermediate host. The insect infected by the nematode pierces the skin, and the larvae of the parasite come out of its mouthparts onto the person's skin, penetrate it and pass into the subcutaneous layer. Within a year they reach the adult stage. Parasites developing in the

subcutaneous layer very often cause the occurrence of subcutaneous lumps which appear most often on the head and torso. Mature female nematodes give birth to larvae (microfilariae) which have the ability to move in the skin and the subcutaneous layer of the whole body. The most dangerous location of the microfilariae for a human is the eye, because they may trigger very serious complications leading often to blindness. During the next blood drawing by an insect, it also draws microfilariae which undergo a series of transformations, reaching a form able to infect the next host.

By moving in the subcutaneous layer and the skin, microfilariae cause skin inflammation. The infected persons experience intensive itchiness in various places of the body; they have wounds, scars, and the skin in some places may be unnaturally droopy due to the loss of flexibility. The wandering larvae after reaching the eyeball very often cause blindness or serious sight impairments [77].

The disease occurs mainly in Africa, South America, Central America, Yemen and Saudi Arabia. According to WHO data, more than 90% of cases of infected people occur in over 30 countries of Sub-Saharan Africa, and also in Latin America in such countries as Brazil, Ecuador, Guatemala, Mexico and Venezuela [94]. It is assessed according to the WHO that approximately 25 million people are currently infected with *O. volvulus*, among whom 300 000 people are blind, and 800 000 have various problems with eyesight. Approximately 125 million people are currently exposed to infections [95].

Prevention of infection with the parasitic nematodes consists principally in using insect repellents and mosquito nets and wearing appropriate clothes protecting people from the vector's bite. There is no vaccination against onchocerciasis.

The next parasite causing cutaneous filariases is *Loa loa*. This nematode triggers a dangerous disease for humans called loaiasis, and the vector responsible for transmitting pathogenic larvae are horse-flies of the genus *Chrysops*. They live in rain forests of central-western Africa, where the disease mainly occurs [96]. They bite during the daytime. Two species of *Chrysops* are responsible for transmitting the disease to people, namely *C. silacea* and *C. dimidata* [94]. Both species occur in the whole of central-western Africa.

Infection of the final host (human) occurs when blood is drawn by an intermediate host (*Chrysops*). Invasive *Loa loa* larvae enter the wound, and next penetrate the blood vessels. After reaching the mature form, the habitat of the mature form is the subcutaneous tissue. The adult individuals frequently move under the skin or in the eye's conjunctiva. Subcutaneous movement is often accompanied by the occurrence of swellings called Calabar swellings [98]. These swellings are usually located near to joints, most often on the arms and legs. In the place of the swelling, unpleasant and intense itchiness may appear. When the parasite is relocating in the conjunctiva, significant congestion, itchiness, pain and hypersensitivity to light may occur. Additional symptoms are pain of muscles and joints, and general fatigue. Rarer but also occurring symptoms of the disease are enlargement of lymph nodes, enlargement and swelling of the scrotum, pneumonia and scarring of the heart. Adult female *Loa loa* are daily able to give birth to thousands of microfilariae which migrate to the blood vessels; they show a specific circadian rhythm, appearing in blood during the daytime, which enables the vectors to ingest them together with the blood of the final host. After drawing microfilariae, the larvae transform in the insect's body into forms able to infect further hosts.

The adult forms of nematodes may survive approximately 15 years in the human body [99], and some people, after many years of being infected, may contract kidney failure.

The highest frequency of infection occurs in Congo, Cameroon, Equatorial Guinea, Gabon and South Chad.

In order to prevent transmission of the disease by vectors, one should wear appropriate clothes to avoid insects' bites; appropriate repellents and mosquito nets should be used, and natural habitats of *Chrysops* horse-flies should be avoided.

Conclusions

It could be supposed that as we live in the 21st century, when the advances of medicine are so great, and our knowledge in this area has constantly been growing, these parasites should not be any problem for us. However, in spite of that, they are among the oldest organisms in the world, they are doing extremely well in the fight for survival, and they still remain in many respects a mystery for us.

The parasites listed in this study are firmly rooted in certain places of our globe (Africa, South America and Asia). They are doing so well because

they can often rely on people's lack of awareness, lack of education, lack of hygiene, lack of money to improve living conditions, on wars and conflicts, on people's willingness to migrate to the natural environments of parasite occurrence, and on people's willingness to destroy natural environments of vectors transmitting parasites, which results in vectors moving to areas not besieged earlier. What is more, the civilizational changes and tourism also contribute to the increased survival of parasitic organisms, and, moreover, their resistance to drugs has increased.

Additionally, the parasites listed in this study have an ally against whom it is difficult to fight. This ally is the insects that are vectors of parasitic diseases. These insects occur in huge numbers, reproduce rapidly, and move quietly. They can very quickly and efficiently transmit a parasite to us, often painlessly and often during our sleep.

Luckily we are not defenseless, and we can combat them in various ways.

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