

# Leishmaniasis (Cutaneous and Visceral)

*Kala-azar, Black Fever,  
Dumdum Fever, Oriental Sore,  
Tropical Sore, Uta,  
Chiclero Ulcer, Aleppo Boi,  
Pian Bois; Espundia,  
Leishmaniosis*

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## Importance

Leishmaniasis is an important group of vector-borne protozoal diseases that affects both humans and animals. Each species of *Leishmania* can infect multiple hosts, though only some also serve as reservoirs. A few organisms are maintained primarily in humans, but most circulate in animals. Both can cause human disease. The consequences of an infection vary with the species of *Leishmania*, the host's health and other factors, and range from asymptomatic infections or localized skin lesions that heal on their own, to widespread skin lesions, potentially disfiguring mucosal lesions or a life-threatening systemic illness. Clinical cases are seen regularly in humans and dogs and occasionally in other animals including cats, ferrets, livestock, guinea pigs and captive or free-living wildlife. Leishmaniasis can be difficult to prevent, as its sandfly vectors are tiny and can penetrate most mesh screens, though some measures can reduce the incidence of bites. An additional difficulty is that treatment does not always reliably eliminate the organism from the body.

## Etiology

Leishmaniasis can be caused by many species of *Leishmania*, a protozoan parasite in the subfamily Leishmaniinae of the family Trypanosomatidae, order Trypanosomatida. The classification of *Leishmania* is complex and often controversial, with researchers debating the number of valid species (e.g., should *L. infantum* and *L. donovani* be considered separate species) and how they should be grouped into complexes of related organisms such as the *L. tropica* or *L. braziliensis* complex.

The *Leishmania* with mammalian reservoirs belong to one of three subgenera, *Leishmania*, *Viannia* and *Mundinia*. Known pathogens in the subgenus *Leishmania* include *L. major*, *L. tropica*, *L. aethiopica*, *L. donovani*, *L. infantum*, *L. mexicana*, *L. amazonensis*, *L. venezuelensis*, *L. waltoni*, *L. lainsoni*, *L. naiffi* and *L. lindenbergi*, while *L. braziliensis*, *L. panamensis*, *L. guyanensis*, *L. shawi*, *L. peruviana* and *L. colombiensis* are members of *Viannia*. The subgenus *Mundinia* contains *L. enriettii*, *L. macropodum* (formerly "*L. australiensis*"), *L. martiniquensis*, *L. orientalis* and an unnamed *Leishmania* recovered from people in Ghana. Some clinical isolates in this complex were originally described as "*L. siamensis*," which is not considered a valid name. Most were later reclassified as members of *L. martiniquensis*, but a few "*L. siamensis*" in Thailand became the new species *L. orientalis*. Information about the members of *Mundinia*, which have caused relatively few clinical cases and seem to differ somewhat from other *Leishmania* in their epidemiology, is still fairly limited.

The subgenus *Sauroleishmania* contains a group of *Leishmania* that mainly seems to infect reptiles, though a few species (e.g., *L. tarentolae*, *L. adleri*) might occasionally infect mammals. Additional species of *Leishmania* have only been described, to date, in insect vectors or asymptomatic animals.

### **Cutaneous, mucocutaneous and visceral leishmaniasis and their causative agents**

Leishmaniasis in humans is divided into three clinical forms, which are termed visceral, cutaneous and mucocutaneous leishmaniasis, depending on the tissues affected. Collectively, the latter two forms are sometimes called tegumentary leishmaniasis. The form of leishmaniasis seems to be related to the tropism of the causative organism, with some species of *Leishmania* typically causing lesions in the skin and, in some cases, the mucous membranes, while others usually invade the internal organs. The species that tend to cause human visceral leishmaniasis belong to the subgenus *Leishmania*, while organisms causing cutaneous leishmaniasis can be found in *Leishmania*, *Viannia* and *Mundinia*. Whether tropism is primarily a property of the individual species of *Leishmania* or depends on a variety of factors, such as the usual dose of parasites, vector or characteristics of the host response, is unclear. However, occasionally an organism known mainly for causing skin lesions will affect the internal organs, particularly (though not always) in those who are immunocompromised, or an organism normally found in visceral leishmaniasis will only cause skin lesions. Some *Leishmania* species also affect different tissues in different hosts. For example, *L. infantum* usually causes visceral leishmaniasis in humans, but it can cause both cutaneous and visceral lesions in dogs.



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The two organisms responsible for most cases of human visceral leishmaniasis are *L. donovani* and *L. infantum*. At one time, the name *L. chagasi* was used for the organism causing visceral leishmaniasis in the Western Hemisphere and *L. infantum* in the Eastern Hemisphere; however, they are now considered to be the same species. *L. donovani* also contains some organisms previously given individual names, such as *L. archibaldi* and *L. killicki*. Occasional clinical cases have been caused by other species of *Leishmania*, including some that are normally associated with cutaneous leishmaniasis such as *L. tropica*, *L. braziliensis* and *L. amazonensis*. An unnamed *Leishmania* in the subgenus *Sauroleishmania* was apparently detected in some cases of visceral leishmaniasis in China.

*L. tropica*, *L. major* and *L. aethiopica* are the usual agents of cutaneous leishmaniasis in the Eastern Hemisphere, but cases have also been caused by other organisms, such as *L. martiniquensis*, an unnamed member of the *L. enriettii* complex in Ghana and *L. orientalis*. The causative agents in the Western Hemisphere include *L. braziliensis*, *L. panamensis*, *L. guyanensis*, *L. shawi*, *L. peruviana*, *L. mexicana*, *L. amazonensis*, *L. venezuelensis*, *L. waltoni*, *L. lainsoni*, *L. naiffi* and *L. lindenbergi*. The viscerotropic organisms *L. infantum* and *L. donovani* are also found occasionally in skin lesions without visceral involvement. *L. braziliensis* causes most cases of mucocutaneous leishmaniasis in the Western Hemisphere, but other organisms including *L. panamensis*, *L. guyanensis*, *L. amazonensis*, *L. peruviana*, *L. infantum*, *L. donovani*, *L. tropica*, *L. major* and *L. aethiopica* also affect the mucous membranes occasionally. One *Leishmania* of reptiles, *L. adleri*, was reported to cause transient cutaneous lesions in humans.

Most of the organisms that cause leishmaniasis in people, as well as two additional species, *L. macropodum* and *L. enriettii*, have been found in clinical cases in animals. The distinction between cutaneous and visceral syndromes and their associations with specific organisms does not seem to be as clear-cut in animals as humans. For example, *L. infantum* causes both cutaneous and visceral signs in dogs and cats, while mainly being a viscerotropic species in people, and leishmaniasis in equids mainly seems to affect the skin, regardless of the causative organism.

### Species Affected

#### *Leishmania* of mammals

Each species of *Leishmania* has one or more primary reservoir hosts, though it can also infect other species. *L. donovani* and *L. tropica* are maintained in humans, while all other *Leishmania* have animal reservoirs. Most clinical cases have been reported in people and dogs, with occasional reports of illnesses in cats, equids, ferrets, other domestic animals and free-living or captive mammalian wildlife. Leishmaniasis does not seem to be a significant disease in ruminant livestock, though cutaneous lesions have been reported rarely in sheep, goats and cattle.

Individual *Leishmania* species seem to have a broad range of incidental hosts, and information about host susceptibility is likely to be incomplete, as these organisms are not identified beyond the genus in most clinical cases. Dogs are the major reservoir hosts for *L. infantum*, which is thought to be responsible for most clinical cases in this species. Probable wildlife reservoirs of *L. infantum* have also been suggested in some areas, and include various wild canids (e.g., red foxes, *Vulpes vulpes* and South American bush dogs, *Speothos venaticus*), hares (*Lepus* spp.) and rodents. *L. infantum* has also caused clinical cases in cats, equids and pet ferrets, and there are reports of infections or illnesses in free-living or captive wild canids, felids and mustelids, genets (*Geneta geneta*), raccoon dogs (*Nyctereutes procyonoides*), wild rabbits (*Oryctolagus cuniculus*), Bennett's wallabies (*Macropus rufogriseus rufogriseus*), opossums, the lesser anteater (*Tamandua tetradactyla*), Algerian hedgehogs, (*Atelerix algirus*), a seal and some species of bats. While it is possible that cats might help maintain or amplify *L. infantum* in some areas, they are thought to be incapable of acting as the reservoir in the absence of dogs. Proposals for the possible involvement of other domestic animal reservoirs, such as large ruminants, are currently speculative.

In the Eastern Hemisphere, gerbils, jirds and other rodents are known reservoir hosts for *L. major*, while members of the Hyracoidea (hyraxes) maintain *L. aethiopica*. Infections or clinical cases caused by *L. major* have also been reported occasionally in other animals, such as dogs, cats, a least weasel (*Mustela nivalis*), and two hedgehog species (*Atelerix algirus* and *Paraechinus aethiopicus*). Although humans are the usual reservoirs for *L. tropica* and *L. donovani*, there are also reports of these organisms in animals. The rock hyrax (*Procavia capensis*) is implicated as a potential reservoir host for *L. tropica* in Israel, and infections with this organism have been reported occasionally in dogs, cats, golden jackals (*Canis aureus*), foxes, rodents and other species. Serological and/or parasitological evidence suggests the possibility of occasional *L. donovani* infections in dogs, goats, cattle, water buffalo and other domestic animals, as well as wild rats and mongooses.

The organisms that cause cutaneous leishmaniasis in the Western Hemisphere (*L. braziliensis*, *L. panamensis*, *L. guyanensis*, *L. shawi*, *L. peruviana*, *L. mexicana*, *L. amazonensis*, *L. venezuelensis*, *L. waltoni*, *L. lainsoni*, *L. naiffi* and *L. lindenbergi*) are maintained in various sylvatic cycles, often among wildlife in forests. Rodents, sloths (*Bradypus* spp. and *Choloepus* spp.) and marsupials such as opossums are thought to be reservoir hosts for some species, but infections have also reported in a wide variety of other wildlife, some of which might also be reservoirs. Domestic animals are sometimes infected with these species of *Leishmania*, but do not appear to act as reservoir hosts, with the possible exception of a minor role for dogs in maintaining *L. peruviana* in one area of South America. Reports of clinical cases have described *L. peruviana*, *L. mexicana*, *L. colombiensis*, *L.*

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*amazonensis*, *L. braziliensis*, *L. panamensis* and *L. guyanensis* in dogs, and *L. mexicana*, *L. venezuelensis*, *L. braziliensis* and *L. amazonensis* in cats, while *L. braziliensis* has caused lesions in equids.

The members of the subgenus *Mundinia* are incompletely understood. *L. macropodum* is known to cause cutaneous leishmaniasis in kangaroos, wallaroos and wallabies, while *L. martiniquensis* was found in black rats (*Rattus rattus*) and caused a few clinical cases in equids and cattle. *L. enriettii* has only been described from guinea pigs, though experimentally infected hamsters also develop mild skin lesions.

Mammalian *Leishmania* do not seem to be important in other vertebrates, though infections might be possible. One study found antibodies to these organisms in geese and a pheasant (*Phasianus colchicus*), but not chickens or small numbers of Muscovy ducks and guinea fowl. Chickens were not susceptible to experimental infection. There are also a few reports of mammalian *Leishmania*, including *L. infantum*, in various reptiles including lizards and snakes.

### Leishmania of reptiles

*Leishmania* in the subgenus *Sauroleishmania* (e.g., *L. tarentolae*, *L. adleri*) infect various reptiles, such as lizards and snakes. Some of these organisms might rarely be found in mammals. Nucleic acids of *L. tarentolae*, which normally infects geckos and lizards, were detected by PCR in dogs at an animal shelter, and another study reported antibodies to this organism in cats and dogs. An unnamed member of *Sauroleishmania* was apparently found in some cases of visceral leishmaniasis in dogs in China. Mice and hamsters experimentally infected with *L. adleri* did not develop any clinical signs.

### Zoonotic potential

Humans are the main reservoir hosts for *L. tropica* and *L. donovani*. As of 2022, clinical cases have been caused by these species and all other mammalian *Leishmania* except *L. enriettii* and *L. macropodum*. Whether the latter two species are zoonotic is uncertain. One report described antibodies to *L. macropodum* in people, but this could have been caused by cross-reactions with other organisms.

Nucleic acids of *L. tarentolae* were detected by PCR in the blood of some healthy people and the bone marrow of a human mummy, and an unnamed member of the subgenus *Sauroleishmania* was reportedly found in some cases of visceral leishmaniasis in people in China.

### Geographic Distribution

Mammalian *Leishmania* are most prevalent in tropical and sub-tropical regions, but they have been found on every continent except Antarctica. Clinical cases in people are seen mainly in Latin America, Africa, the Middle East, parts of Asia, and the Mediterranean region of Europe.

Many species of *Leishmania* are limited to either the Eastern or Western Hemisphere. Organisms circulating in the Eastern Hemisphere include *L. major* in Africa, the Middle East and parts of Asia; *L. tropica* in the Middle East,

the Mediterranean and parts of Asia; and *L. aethiopica* in Africa. The many species that cause cutaneous leishmaniasis in the Western Hemisphere (*L. braziliensis*, *L. panamensis*, *L. guyanensis*, *L. shawi*, *L. peruviana*, *L. mexicana*, *L. amazonensis*, *L. venezuelensis*, *L. waltoni*, *L. lainsoni*, *L. naiffi* and *L. lindenbergi*) occur mainly in Mexico and Central and South America; however, *L. mexicana* is also endemic in parts of Texas and southern Oklahoma in the U.S. Among the *Mundinia*, *L. enriettii* has been found in South America, *L. macropodum* in Australia, *L. orientalis* in Thailand, and an unnamed member of this subgenus in parts of Africa; while *L. martiniquensis* has been reported from both hemispheres, with clinical cases described in Thailand, Myanmar, Europe, the Caribbean and North America.

*L. donovani* causes human visceral leishmaniasis in Africa and part of Asia (the Indian subcontinent), while *L. infantum* is widely distributed, circulating in Latin America, the Mediterranean, the Middle East and parts of Asia. The latter organism is also found in certain groups of hunting dogs (mainly foxhounds) in the U.S., where vertical transmission seems to be important in maintaining the organism. Other mammals seem to be unaffected by the foxhound-associated *L. infantum* in this area, though there is one report of low antibody titers in a few wild canids in Pennsylvania and North Carolina, either from *Leishmania* or cross-reactivity to other parasites.

Imported cases of leishmaniasis can be seen occasionally in human travelers or animals in areas where *Leishmania* spp. are not endemic. There are also a few reports of cases in people or animals that never left apparently *Leishmania*-free areas in Europe, Asia (e.g., Hong Kong) or North America. Transmission from an imported case via local sandfly populations or other vectors could account for some of these incidents, but other mechanisms (e.g., vertical transmission or contact with the blood of infected animals) are suspected in others.

Members of the subgenus *Sauroleishmania* have been documented in Europe, North Africa, the Middle East and parts of Asia (e.g., China), but could be more widely distributed.

### Transmission

Sandflies in the genera *Phlebotomus* and *Lutzomyia* are the biological vectors for the subgenera *Leishmania* and *Viannia*; however, the vectors for *Mundinia* are unknown, and some evidence suggests that they might be transmitted by biting midges (e.g., *Forcipomyia*, *Culicoides*) rather than sandflies. The *Sauroleishmania* are usually transmitted by sandflies of the genus *Sergentomyia*, which normally feed on reptiles, but mammal-feeding *Phlebotomus* also seems to be a competent vector for some organisms. Some authors have suggested that it might be possible for other arthropods, such as biting midges, ticks or dog fleas, to transmit *Leishmania* mechanically in some circumstances, but this is uncertain.

Sandflies are most likely to be active when it is humid and there is no wind or rain. They are generally most prevalent at dawn, dusk and during the night (especially early



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in the night), but they will bite if disturbed in their hiding places during the day. These include animal burrows, caves, holes in trees, cracks in houses and other relatively cool, humid locations. They are also attracted to light and may enter buildings at night. Only female sandflies can transmit *Leishmania*; the males do not feed on blood. The females take multiple blood meals, which is thought to increase their effectiveness as vectors, and they can be infected from both symptomatic and subclinically infected mammals.

Most sandfly species remain close to where they hatch (adults usually fly < 100 m from their larval sites), though there are a few reports of these insects traveling, or perhaps being transported (e.g., by wind), more than a kilometer. Female sandflies deposit their eggs in humid locations with organic matter, such as cracks or holes in the ground, animal burrows, leaf litter and termite mounds. *Leishmania* does not seem to be transmitted to a new sandfly generation via the eggs (transovarial transmission), but in areas with cold temperatures, the parasite can overwinter in infected mammals.

Vertical transmission is thought to be important for maintaining *L. infantum* among foxhounds in the U.S., and recent evidence suggests it may also be significant among rodents in some endemic areas. Transplacental transmission has been demonstrated in dogs (*L. infantum*), mice (*L. mexicana*, *L. infantum*), hamsters (*L. panamensis*, *L. donovani*) and people (*L. infantum*, *L. donovani*). Human infants can be born infected whether or not the mother was symptomatic. To date, *Leishmania* has not been found in milk; however, organisms were detected in the mammary glands of dogs and one cat. *L. infantum* and/or its nucleic acids have also been found in the semen and vaginal secretions of dogs, and venereal transmission has been shown to be possible in dogs (*L. infantum*), humans (*L. infantum*) and experimentally infected mice.

Other possible, though uncommon, methods of transmission include blood transfusions, as well as needles shared by intravenous drug users. *L. infantum* has occasionally spread between dogs in the same household or kennel in the absence of sandflies, transfusions or other known mechanisms. Case histories suggest that some of these animals might have been infected during a fight, by licking a companion's lesions, or by ingesting blood during a hemorrhage. This hypothesis is supported by the demonstration of oral transmission of *L. infantum* and *L. braziliensis* in experimentally infected hamsters, with both organisms causing lesions in internal organs. *L. infantum* and/or its nucleic acids can also be found sometimes in canine saliva, urine and conjunctival secretions, though whether these secretions and excretions can actually transmit the organism is still uncertain.

## Disinfection

Environmental transmission of *Leishmania* spp., which normally survives only inside a host, is insignificant or absent. If necessary, these organisms can be inactivated by agents such as 1% sodium hypochlorite, 70% ethanol, 0.1%

hand soap, 2% glutaraldehyde or formaldehyde. They are also susceptible to heat of 50-60°C (122-140°F).

## Infections in Animals

### Incubation Period

Animals can remain subclinically infected with *Leishmania* indefinitely, but they may also become ill at any time. Dogs affected by *L. infantum*, including those that acquired the organism *in utero*, have generally developed clinical signs several months to years after exposure. Most of the pups in one vertically infected litter had signs by the age of 2 months, which was considered unusually early.

### Clinical Signs

#### Dogs

The signs of leishmaniasis in dogs are variable and can mimic other illnesses. Some dogs infected with *L. infantum* never develop any clinical signs. Others become mildly to severely ill with skin lesions, visceral signs or both.

Common signs with visceral involvement include lethargy, a decreased appetite, weight loss, anemia, thrombocytopenia, splenomegaly and local or generalized lymphadenopathy. Fever is absent in many cases and can be intermittent if it occurs. One study suggested that, while a few dogs infected with *L. infantum* become severely ill soon after exposure, most symptomatic dogs have only relatively subtle signs such as lymphadenopathy, thrombocytopenia and/or mild non-regenerative anemia, with or without weight loss, during the first 2 years.

Chronic renal disease is common in the later stages. It is sometimes the only abnormality noted, and is often the cause of death. There may also be bleeding disorders, with signs such as epistaxis, hematuria and melena; vomiting and/or chronic diarrhea from involvement of the gastrointestinal tract; osteolytic and osteoproliferative bone lesions; orchitis, chronic prostatitis, autoimmune disorders and cardiovascular signs. Some dogs develop erosive or (more commonly) nonerosive arthritis affecting one or more joints, and chronic polymyositis can cause progressive muscle atrophy. Rare cases with neurological signs (e.g., gait abnormalities, disorientation, seizures, atypical behavior) may be caused by direct involvement of the CNS, peripheral neuropathy or serum hyperviscosity-induced hypoxia. Reproductive losses (abortions, stillbirths) have been reported occasionally, but many congenitally infected pups initially seem to be healthy, and can remain so for months to years.

Skin lesions are common in dogs with visceral disease, but they can also occur alone. One of the most common forms of skin disease in *L. infantum*-infected dogs is a non-pruritic, exfoliative dermatitis mostly seen on the face, ears and/or feet. Some affected dogs may have areas of alopecia with silvery white scales, especially around the eyes. *L. infantum* can also cause nodules, papules, ulcers and/or scabs, and a distinctive dermatitis with solitary to multiple

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papules is associated with this organism in some regions. The latter condition seems to be mild and does not appear to be accompanied by visceral involvement. Atypical skin lesions have included sterile pustular rashes (which may be pruritic), panniculitis, depigmentation, erythema multiforme, digital and nasal hyperkeratosis, and cases that resemble alopecia areata or pemphigus foliaceus. Mucosal involvement is characterized by ulcers, nodules, papules or masses, with or without skin lesions. Tongue nodules appeared to be the only sign of leishmaniasis in a few dogs. Some dogs with cutaneous signs also have abnormally long and brittle nails.

Canine leishmaniasis sometimes affects the eyes, with or without other clinical signs. The most common abnormalities are blepharitis, conjunctivitis, keratitis and anterior uveitis. Some animals may have multiple granulomas on/ in the eyelid margins, nictitating membrane margins, conjunctival limbus, cornea and/or anterior chamber. Sequelae of ocular involvement may include glaucoma, keratoconjunctivitis sicca, corneal pigmentation, iris atrophy, cataracts, retinal detachment, panophthalmitis or phthisis bulbi.

Other species of *Leishmania* seem to cause similar syndromes in dogs, though different types of skin lesions may be more prevalent, and visceral involvement has not been reported with some organisms. Localized ulcers with raised borders, which may persist for months or years, appear to be a common presentation with *L. braziliensis*, while various skin lesions were reported in dogs infected with *L. major*, *L. panamensis*, *L. guyanensis* and some other species. Both visceral and cutaneous signs are seen in dogs in Texas, where *L. mexicana* is endemic, though dogs infected experimentally with this organism only developed skin lesions. *L. colombiensis* and *L. amazonensis* were found in a few cases of visceral leishmaniasis, and visceral, skin and/or mucosal involvement was reported in a few dogs infected with *L. tropica*.

### Cats

Most reports of cats with leishmaniasis have described skin and/or mucosal lesions, with or without visceral involvement, but visceral signs can also occur alone. The most commonly reported skin lesions in this species are localized nodules, papules and chronic crusted or ulcerated lesions, but alopecia, scales, and hemorrhagic pustules or nodules have also been seen. Cutaneous lesions are particularly common on the face (often on the nose, ears, eyelids or lips) and are often single, though multiple or disseminated lesions are also reported. Affected cats may also have regional lymphadenopathy. Nail lesions appear to be rare in cats.

Visceral signs seem to be similar to those in dogs, with various reports describing fever, lymphadenopathy, hepatomegaly, jaundice, splenomegaly, vomiting and/or diarrhea, respiratory signs, anemia, thrombocytopenia, azotemia, hyperproteinemia, polyclonal gammopathy and, in one case, mastitis. Some affected cats had moderate to severe pancytopenia, but whether this was caused by *Leishmania* is uncertain, as some of these animals were also infected with FIV. Ocular lesions, including unilateral or bilateral uveitis,

panophthalmitis, conjunctivitis, conjunctival nodules and blepharitis have also been documented. One cat had a history of abortion. While spontaneous cures have been reported in some cats, leishmaniasis can be fatal, and some cats have relapsed after treatment.

### Equidae

Horses, mules and donkeys sometimes develop skin lesions, particularly on the head, neck, legs and axillary or inguinal regions. The most common lesions are solitary or multiple papules or nodules, which are often ulcerated; however, disseminated skin disease has also been seen. Visceral leishmaniasis has not been documented in equids, but parasites were observed in the bone marrow of one horse in South America, and nucleic acids of *L. braziliensis* were identified in the blood of another animal. Decreased red blood cells and increased serum urea concentrations were reported in one study of *L. infantum*-infected horses with skin lesions in Brazil, though a causative role remains to be demonstrated.

### Other domestic animals

Leishmaniasis has rarely been described in ferrets; however, there are a few reports of animals with skin lesions, which may be accompanied by other signs such as enlargement of the lymph nodes and spleen. Skin lesions and, in some cases, lymphadenopathy were the only signs reported in a few clinically affected sheep, goats and cattle. One pregnant cow infected with *L. martiniquensis* in Germany had multiple ulcerative or plaque-like cutaneous lesions on several parts of the body. It recovered completely after giving birth. Sheep inoculated with *L. donovani* did not have any clinical signs except an elevated temperature. Experimentally infected pigs remained asymptomatic.

*L. enriettii* has been detected in skin lesions, often on the ear, in naturally infected guinea pigs. Cutaneous lesions in experimentally infected guinea pigs first appear as redness and swelling at the inoculation site, but grow rapidly into large, ulcerated, tumor-like masses. Secondary lesions developed on other parts of the body in some studies, but others reported that the lesions did not spread. Some studies, but not others, reported spontaneous healing. *L. enriettii* has also been found in various internal organs of experimentally infected guinea pigs. Experimentally infected hamsters developed non-ulcerated nodules, which healed without treatment.

### Captive wild species and wild animals

The few reported cases of leishmaniasis in free-living or captive wild canids resembled the disease in dogs. Visceral involvement with nonspecific signs (e.g., pale mucous membranes, weight loss) was reported in some nonhuman primates. A lion had clinical signs of colitis and bloody diarrhea, epistaxis, weight loss and ulcers on the footpads, and a captive Eurasian otter (*Lutra lutra*) developed epistaxis and nonspecific signs of illness including lethargy, anorexia and weight loss. Captive Australian marsupials infected with *L. martiniquensis* had

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skin lesions consisting of focal to coalescing areas of thickened skin, or raised, crusted or ulcerative pale nodules. Skin lesions were also found in some rodents, living in the wild, that were infected with members of the *L. mexicana* complex. The lesions, which appeared as swellings with hair loss or ulcers, were most common at the base of the tail, but sometimes also occurred on the ears or toes. Subclinical infections have been reported in many species.

### Post-Mortem Lesions [Click to view images](#)

In addition to the cutaneous and mucosal lesions visible in live animals, there may be signs of cachexia and anemia, regional or generalized lymphadenopathy, enlargement of the liver and/or spleen, and in some cases, hemorrhages in internal organs. Small, light colored nodular foci (granulomas) may also be found in various organs, including the kidney, liver and pancreas. Subclinically infected animals may have no lesions.

### Diagnostic Tests

In sick animals, parasites can sometimes be observed in samples from skin lesions or lymph node, spleen and bone marrow aspirates, using Giemsa, Wright's, Leishman's and other stains. *Leishmania* amastigotes are round to oval, with a round basophilic nucleus and a small rod-like kinetoplast. Histopathology with immunohistochemistry may be helpful when few organisms are present. PCR assays are also useful, and, in addition to other clinical samples, may sometimes detect nucleic acids in conjunctival swabs from dogs. Loop-mediated isothermal amplification (LAMP) assays have also been developed. In addition, it is possible to culture *Leishmania*, though this is rarely used for diagnosis. Culture generally requires 5 to 30 days, and some species can be difficult to isolate. Animal (hamster) inoculation was occasionally employed in the past if leishmaniasis was suspected but the parasite was difficult to find, but it has been generally replaced by PCR.

Most of the commonly-used diagnostic tests, including many PCR assays, identify *Leishmania* only to the level of the genus, and even species-specific PCR tests may sometimes amplify other organisms. The species can be definitively identified by combining PCR with restriction fragment length polymorphism (RFLP) analysis or sequencing, or by multilocus enzyme electrophoresis, MALDI-TOF mass spectrometry and other techniques. These tests are likely to be available only in limited locations, and some require the organism to be cultured.

Serology can be helpful in dogs with visceral involvement, which usually have high antibody titers; however, low titers, or titers in vaccinated dogs can be difficult to interpret, and dogs with localized skin lesions are sometimes seronegative. The most commonly used serological tests in dogs are the indirect fluorescent antibody test (IFA), ELISAs and rapid immunochromatographic assays (rK39 dipstick or strip-test). Other assays have more limited availability or were used more often in the past. Cross-reactivity with other parasites, particularly

*Trypanosoma cruzi*, can be an issue, especially with tests that use crude antigen preparations. Serology also appears to be useful in cats, though it may be more difficult to find tests validated in this species. The delayed hypersensitivity (leishmanin) test used in humans is not employed for diagnostic purposes in animals.

### Treatment

Treatment can produce clinical improvement, but it may not eliminate the parasite and animals sometimes relapse. Some drugs used for leishmaniasis in humans (e.g., pentavalent antimonials, amphotericin B, miltefosine, allopurinol, paromomycin, marbofloxacin) have also been employed successfully in animals, either alone or in combination. Allopurinol has been used long-term to prevent relapses. Pentavalent antimonials are typically unavailable outside endemic regions, though they can sometimes be obtained from government agencies (e.g., the CDC in the U.S.) or other sources. Drug-resistant *Leishmania* can sometimes be an issue. Immunomodulatory agents have sometimes been included in the treatment; however, there is currently no clear evidence for their efficacy.

Other treatments are uncommonly described in animals, but radio-frequency induced heat therapy was successful in two dogs with multiple localized mucocutaneous lesions on the snout, and cryotherapy was effective in a cat with two nodules on its nose. Skin lesions did not return after surgical resection in some animals, including some cats and a number of horses, though others relapsed. Outside endemic regions, euthanasia may be considered to decrease the risk the organism will spread, particularly if a competent sandfly vector exists in the area.

### Control

#### Disease reporting

Veterinarians who encounter or suspect leishmaniasis should follow their national and/or local guidelines for disease reporting. Leishmaniasis in animals may be reportable in some states in the U.S.

#### Prevention

Keeping susceptible animals indoors between dusk and dawn, especially during the warmer months, is likely to reduce their exposure to sandflies. Insecticide-impregnated collars or topical insecticides (spot-on preparations, sprays) are reported to decrease sandfly bites in dogs, and some studies of pets suggest they may be the most effective means of prevention. Some collars also appear to work in cats. Other vector control methods include insecticide sprays; insecticide-treated door and kennel nets and curtains; fans to deter sandflies, which are poor fliers; and habitat modifications to remove or dry out sandfly breeding areas around the home. Adverse effects on the environment or unintended effects such as promoting insecticide resistance can be a factor with some control methods. Untreated mesh does not exclude sandflies unless it is extremely fine.



# Leishmaniasis (cutaneous and visceral)

Commercial *L. infantum* vaccines, which have been reported to decrease the incidence of clinical cases and/or infections, are available for dogs in some countries. However, the efficacy of some vaccines in the field is unclear. Protection is also incomplete, and vaccine manufacturers recommend insect control for vaccinated as well as unvaccinated dogs. Some control programs attempting to reduce the prevalence of *L. infantum* cull infected dogs; however, this is controversial for ethical reasons, and its efficacy is also in doubt. Due to the risk that some offspring will be born infected, it is not considered advisable to breed from infected animals, whether or not they are symptomatic. Dogs or cats used as blood donors in endemic areas should be tested for subclinical *Leishmania* infections.

## Morbidity and Mortality

Exposure to *L. infantum* can be common among dogs in endemic areas, with serological surveys reporting that 2-80% have antibodies, depending on the region, serological test used and specific groups of dogs tested. Seropositive dogs are especially prevalent where the densities of both dogs and sandflies are high. Some dogs bitten by infected sandflies appear to eliminate *L. infantum*, but others remain subclinically infected. Only a small percentage of the latter group seems to become ill. While clinical cases are more likely in dogs that become immunosuppressed, progression to disease is otherwise hard to predict. One study suggested that a high percentage of *L. infantum*-infected dogs will eventually become sick if they have “active” infections, as defined by a combination of high antibody titers, PCR evidence of infection in the bone marrow, and isolation of the organism from lymph nodes. The prognosis for sick dogs is worse in severe cases, and a clinical staging system has been published to assist with treatment considerations and prognosis.

Sporadic cases of leishmaniasis also occur in cats, equids and other species. Studies in cats suggest that they may be frequently exposed to *Leishmania* in some areas, though clinical cases are not reported often. The number of published cases in cats has, however, been increasing, possibly due to increased awareness. About a third to half of the cats that developed clinical leishmaniasis, to date, were co-infected with immunosuppressive viruses (e.g., FIV, FeLV), had other debilitating conditions such as cancer or diabetes, or were on immunosuppressive drugs. However, this disease has also been seen in otherwise healthy cats. Relatively little is known about the prognosis for sick cats, outside case reports.

## Infections in Humans

### Incubation Period

People can carry some species of *Leishmania* asymptotically for long periods or indefinitely. The incubation period ranges from 1-2 weeks to years, but many clinical cases seem to occur within several months of exposure.

## Clinical Signs

Two distinct forms of leishmaniasis, cutaneous and visceral, are seen in humans. Some texts also distinguish a mucocutaneous form, while others consider it to be a subset of cutaneous leishmaniasis. While simultaneous tegumentary (cutaneous, mucosal) and visceral involvement is possible, it is uncommon if the person is healthy.

### Cutaneous and mucocutaneous leishmaniasis

In people, cutaneous leishmaniasis usually begins as one or more erythematous, possibly pruritic, papules, which can develop into a variety of lesions, such as ulcers with raised, indurated margins and hemorrhagic crusts; nodules, which may be smooth or covered in scales; flat plaques; and hyperkeratotic wart-like growths. Unusual forms that mimic other diseases (e.g., erysipelas, psoriasis) are also possible. Some organisms tend to be associated with certain types of skin lesions. *L. major* lesions are often exudative or “wet” and prone to secondary bacterial infections, while those caused by *L. tropica* are more likely to be “dry,” with a central crust. Except in the ear, leishmaniasis ulcers are usually shallow and do not invade subcutaneous tissues, and cutaneous leishmaniasis is often painless unless there are secondary infections or an ulcer lies over a joint.

Many cases of cutaneous leishmaniasis remain localized; however, some people develop secondary lesions on the skin, or occasionally the mucosa, in other parts of the body. When the parasites travel via the lymphatics, the presentation may resemble sporotrichosis. Patients with cutaneous leishmaniasis sometimes have regional lymphadenopathy, and peripheral neuropathy has been reported. The skin lesions often heal spontaneously, but this may take several months to a year or more, depending on the organism, and it may leave permanent scars. Persistent regional lymphadenopathy has occasionally been reported after the skin lesions have healed.

Several uncommon and more severe forms of cutaneous leishmaniasis have also been described. Leishmaniasis recidivans (lupoid leishmaniasis, leishmaniasis recidiva cutis) is an uncommon condition characterized by the development of new skin or mucosal lesions, typically plaques, in and around the edges of a healed skin lesion. Leishmaniasis recidivans tends to be chronic and relapsing, can be difficult to treat, and does not heal without treatment. In disseminated leishmaniasis, a form mostly reported from parts of South America, patients develop hundreds or thousands of lesions and sometimes have systemic signs, such as fever, chills, lethargy and nausea, while the skin lesions are spreading. Another uncommon form called diffuse cutaneous leishmaniasis (DCL) or anergic diffuse cutaneous leishmaniasis, is most often caused by *L. amazonensis* and *L. mexicana*. In patients with DCL, the skin lesions tend not to ulcerate, but appear as nodules, papules and tubercles that spread widely on the skin and can coalesce into large plaques. These lesions may damage deeper tissues and can persist indefinitely. DCL can be incurable in some cases.

# Leishmaniasis (cutaneous and visceral)

Skin lesions in immunosuppressed patients often resemble those in immunocompetent hosts, but the risk of disseminated infections, DCL, mucosal or visceral involvement, and severe or atypical cases is higher.

## Mucosal involvement

Classical mucocutaneous leishmaniasis (espundia) is usually seen in Latin America, where it can be caused by several organisms but especially *L. braziliensis*. It tends to develop within a few years after the skin lesions heal, but it can also be seen while they are still present, or even in people who apparently never had cutaneous leishmaniasis. The first signs are usually erythema and ulcerations at the nares, followed by destructive nasal inflammation with ulcers and nodules, which can spread to involve the septum and, in some cases, the oral cavity, pharynx and/or larynx. Frequent nosebleeds or itching in the nose can be an early sign. The genitalia may also be involved in some instances. Classical mucocutaneous leishmaniasis does not heal spontaneously, and untreated lesions may eventually cause severe facial disfigurement or block the pharynx or larynx.

Several species of *Leishmania* in the Eastern Hemisphere can also affect the mucous membranes, with or without concurrent or previous skin lesions. Some solitary mucosal lesions caused by these organisms may not spread, even when they are untreated for years; others can multiply or later affect the viscera.

## Visceral leishmaniasis

Viscerotropic organisms can cause subclinical infections, self-limited cases with only a few signs such as localized lymphadenopathy, and chronic or fatal disease.

Most clinical cases are insidious and chronic; however, acute illnesses are sometimes seen in travelers from *Leishmania*-free areas, and fulminant disease is possible in people who are immunosuppressed. The most common symptoms of chronic visceral leishmaniasis are a prolonged undulant fever, weight loss, decreased appetite, signs of anemia, and abdominal distension with splenomegaly and hepatomegaly. Some patients may also have lymphadenopathy, a cough, chronic diarrhea, darkening of the skin, edema, bleeding tendencies from thrombocytopenia, increased susceptibility to other infections, and in many prolonged cases, signs of kidney disease. Some of these symptoms, such as darkening of the skin, are more common in some regions than others, or tend to be associated with a specific organism. Particularly in Africa, a primary granuloma sometimes appears on the skin before the systemic signs become evident. CNS signs, peripheral neuropathy and ocular signs (uveitis, retinal hemorrhage) can also be seen but are uncommon. Clinical cases in the immunocompromised can be atypical and may include skin and mucosal lesions, while common signs such as fever and splenomegaly may be less prominent. Cases in congenitally infected infants have mainly been characterized by premature birth, fever and enlargement of the spleen and liver.

Mild visceral leishmaniasis with only a few symptoms (e.g., localized lymphadenopathy) may resolve spontaneously, but most fully symptomatic, untreated cases are thought to eventually be fatal, often from secondary infections and other complications. While spontaneous remissions have generally been considered unlikely in fully symptomatic cases, one report described multiple spontaneous remissions and relapses, over a 2-year-period, in a person infected with *L. infantum*. People with successfully treated infections may continue to carry the parasite, and the disease may recur if they become immunosuppressed.

Some people who recover from visceral leishmaniasis, especially those infected with *L. donovani*, develop post-kala azar dermal leishmaniasis (PKDL). PKDL is characterized by a maculopapular, macular or nodular rash that generally begins on the face, especially around the mouth, but can spread to the neck, torso and extremities, and uncommonly to the nasal or oral mucous membranes, eyelids and/or cornea. Coalescing lesions can result in enlargement of the lips and nose. Occasionally, the lesions may ulcerate, especially in Africa. In Africa, PKDL is common, usually occurs within 6 months of visceral leishmaniasis, and often disappears spontaneously within a year if the mucous membranes are not involved. On the Indian subcontinent, however, this syndrome is not very common, occurs one to many years after visceral leishmaniasis has been cured, and may require prolonged treatment to resolve. Most sources state that that PKDL does not usually regress on its own in the latter region; however, resolution was documented in some untreated patients in Bangladesh, with a median time of 19 months. PKDL has also been reported occasionally in people with no apparent history of visceral leishmaniasis.

## Diagnostic Tests

Leishmaniasis in humans can be diagnosed by direct observation of the parasite, PCR or LAMP assays, immunohistochemistry and/or culture, as in animals. PCR may be particularly valuable early in the visceral form, when parasite numbers are low. In cutaneous leishmaniasis, amastigotes are easiest to detect visually in recent or active lesions, or in cases of diffuse cutaneous leishmaniasis. A latex agglutination test to detect parasite antigens in the urine has been published, and appeared to be particularly useful in immunosuppressed patients.

Serological tests are also employed in the diagnosis of visceral leishmaniasis, though cross-reactivity with the agents of other diseases, such as leprosy, Chagas disease, malaria and schistosomiasis, can sometimes be an issue. Antibodies are often slow to develop and of low titer in cutaneous leishmaniasis; however, serology may be more helpful in chronic skin conditions such as disseminated leishmaniasis or leishmaniasis recidivans, and in the mucocutaneous form.



# Leishmaniasis (cutaneous and visceral)

A delayed hypersensitivity test, the leishmanin skin test (Montenegro skin test), was sometimes useful for diagnosing cutaneous and mucocutaneous leishmaniasis in the past, especially outside endemic areas; however, this test is now uncommon as standardized antigens are unavailable. The leishmanin skin test is often negative in symptomatic visceral leishmaniasis, but reactions can be seen once the disease is cured. It also tends to be negative in the diffuse (anergic) cutaneous form (DCL). The skin test is not as helpful in endemic regions, where a positive reaction may indicate either a current infection or past exposure, including previous asymptomatic visceral or cutaneous infections.

## Treatment

Drugs used to treat leishmaniasis in humans include pentavalent antimonials (e.g., sodium stibogluconate, meglumine antimoniate), amphotericin B (particularly liposomal amphotericin B), paromomycin, miltefosine, pentamidine isothionate, azoles (e.g., ketoconazole, fluconazole, itraconazole), allopurinol and other agents. Resistance to pentavalent antimonials is a major problem in some areas. Significant resistance to other drugs has also been reported in some *Leishmania* species.

Cutaneous leishmaniasis is sometimes treated with systemic drugs, similarly to visceral leishmaniasis; however, these agents may also be administered intralesionally or topically, depending on the infecting species and risk of serious complications. Cryotherapy, thermotherapy, photodynamic therapy, CO<sub>2</sub> laser treatment or curettage have also been employed, either alone or in combination with drugs. Some skin lesions that are improving may simply be observed, if they are caused by relatively benign organisms.

## Prevention

Measures to prevent sandfly bites include using insect repellents such as DEET, covering exposed skin, and staying on higher floors of buildings in the evening and at night, as these insects are poor vertical fliers. Insecticidal sprays or insecticide-impregnated materials (e.g., window curtains) are also employed, and fans may be helpful. Insecticide-treated bed nets can reduce the risk of bites from these insects at night; however, untreated bed nets are not generally helpful, as those with holes small enough to exclude sandflies are likely to be too hot in warm climates. Environmental modifications might reduce sandfly populations around the home, and treatment of infected people, as well as measures to reduce the incidence of *L. infantum* in dogs, are thought to decrease the prevalence of some organisms.

Live vaccines were occasionally used in the past, with inoculation into an inconspicuous site to prevent disfiguring facial lesions, but they are no longer available in most countries, and no new vaccines have been commercialized yet for humans. Leukodepletion significantly reduces or eliminates *Leishmania* in blood transfusions

## Morbidity and Mortality

While asymptomatic infections are thought to be the most common result of exposure to *Leishmania* in healthy people, cutaneous and visceral leishmaniasis are common diseases in endemic regions. The consequences of infection may be influenced by the species of *Leishmania*, host factors (e.g., genetic susceptibility, immunity, general health, age), inoculation site, dose of parasites received and other factors. Immunosuppression from other diseases or drugs, such as TNF-alpha inhibitors and agents to prevent transplant rejection, increases the risk of serious illness or persistent disease, and can make treatment more difficult.

Visceral leishmaniasis caused by *L. donovani* can affect all ages. Its incidence tends to wax and wane, with periods of higher prevalence lasting about 5-15 years and interepidemic periods of roughly 10-30 years, likely due to the patterns of population immunity. On the Indian subcontinent, the ratio of asymptomatic infections to symptomatic cases has been estimated at about 4-10 to 1. Other studies suggest that, in this region, 1-23% of asymptotically infected people develop visceral leishmaniasis within a year, while many others become seronegative and are likely to have eliminated the organism. Healthy adults do not seem to be particularly susceptible to visceral leishmaniasis from *L. infantum*. Asymptomatic infections with this organism are common, and illnesses tend to be seen mainly in young children, or in people who are malnourished or immunosuppressed.

Symptomatic visceral leishmaniasis caused by either *L. donovani* or *L. infantum* can be life-threatening. Estimates of the case fatality rate vary, but are higher in people who are malnourished or have various co-morbidities. HIV infections increase the risk of relapse even when taking highly active antiretroviral therapy (HAART). Other immunosuppressive conditions such as solid organ transplants do not seem to have as much of an effect on relapse rates, but experience is limited. Localized cutaneous leishmaniasis is rarely fatal and often heals spontaneously, though some lesions may last a long time or leave scars. Disseminated cutaneous lesions and mucosal leishmaniasis can be more serious. In particular, the classical mucocutaneous form in South America rarely heals spontaneously, is disfiguring and may occasionally be fatal.

## Internet Resources

[LeishVet. Information for Veterinarians on Leishmaniasis](http://www.leishvet.org/)  
<http://www.leishvet.org/>

[The Merck Manual](#)

[The Merck Veterinary Manual](#)

[World Health Organization](#)

[World Organization for Animal Health \(WOAH\)](#)

[WOAH Manual of Diagnostic Tests and Vaccines for Terrestrial Animals](#)

[WOAH Terrestrial Animal Health Code](#)

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