Vol. 20, No. 11 November 1998

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Refereed Peer Review

FOCAL POINT

- Veterinarians must recognize that pets returning to the United States from abroad have the potential to introduce exotic, and often dangerous, pathogens into our country.

KEY FACTS

- Foreign diseases or parasites recovered from pets entering the United States must be reported to state veterinary and public health officials, who will pass pertinent reports on to federal officials as applicable.
- Many pathogens introduced into the United States by imported animals have the potential to become established here.
- *Cochliomyia hominivorax*, once enzootic in the United States but eradicated in the 1950s, is occasionally reintroduced into the country by imported animals.
- Introduction into the United States of *Dermatobia hominis*, which can develop in a wide variety of definitive hosts, could result in serious consequences.

International Travel with Pets. Part II. The Threat of Foreign Pathogens*

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Citizens may live for months to years outside the United States, and they often own dogs or cats that were either acquired in the United States and transported abroad or adopted in a foreign country. Upon the owner's return to this country, dogs, cats, and certain other pets are subject to measures designed to prevent the introduction and spread of rabies and other zoonotic diseases. These pets may be inadvertently infected with foreign diseases or parasites—pathogens with the potential to produce serious consequences within our borders. If diagnosed, these pathogens must be reported to the appropriate state authorities, who in turn will notify the proper federal agencies. Part I of this three-part presentation discussed issues pertinent to leaving and returning to U.S. territorial borders with pets. Parts II and III will review foreign pathogens that have been introduced into various countries by pets. Each pathogen is discussed according to its geographic distribution, clinical presentation, diagnosis, treatment, and zoonotic potential.

Pathogens can spread from completely foreign ecosystems and become established in new geographic locales.[1] Based on recent reports, we have grouped these "invaders" into five major categories (see Categories of Invading Pathogens), each of which is discussed in relation to at least one pathogen. This article describes the first two categories; Part III will describe the final three categories.

*Part I of this three-part presentation appeared in the October 1998 (Vol. 20, No. 10) issue of *Compendium*.

CATEGORY I
Cochliomyia hominivorax

C. hominivorax (the New World or primary screwworm) is an obligatory wound parasite that is native to North and South America. This fly is unusual in that it was once native to the United States but was eradicated in the late 1950s by the release of γ-irradiated, sterile male flies.[2] *C. hominivorax* has often been reintroduced, however, via pets returning to this country and those traveling through our international airports[3–5] and across the borders of other countries.[6,7] Veterinarians must always be on guard to deter reintroduction of this economically important, myiasis-producing parasite.

In the first half of the twentieth century, *C. hominivorax* was enzootic in the southwestern and southeastern United States. The adult fly cannot spend the winter in cold climates; however, summer migrations have occurred as far north as Montana and Minnesota.[8] Although the parasite was eradicated from the United States,[2] the potential for reestablishment is great because of the increase in international travel, particularly air travel.[9] *C. hominivorax* is currently found throughout much of the tropical and subtropical areas of Mexico, Central America, and most of South America.[8]

Categories of Invading Pathogens

I. Pathogens once native to, but eradicated from, the United States[a] that are now native to other countries. These pathogens are occasionally reintroduced into the United States[a] by imported animals; however, because of rigorous control programs, they have not reestablished in this country.[a]
II. Pathogens native to other countries that have been introduced into the United States[a] by imported animals and have the potential to become established.
III. Pathogens native to other countries that have been introduced into the United States[a] by imported animals and have become firmly established.
IV. Pathogens native to other countries that have been introduced into the United States[a] by imported animals but could never become established because a critical intermediate host is lacking within the ecosystem.
V. Pathogens native to other countries that have never been introduced into the United States[a] but have the potential for introduction.

[a]Or other countries.

Clinical Presentation

The adult female *C. hominivorax* never deposits its eggs in carrion but instead lays them at the edge of fresh, uncontaminated wounds of warm-blooded animals. Such wounds are often nothing more than an insignificant puncture wound or tick bite. Within the wound, the larvae (i.e., maggots) penetrate; feed; and rapidly devour fresh, live host tissue. The larvae feed as a group, burrowing deeply into tissue. In time, larvae liquefy the tissue and extend the lesions considerably. The wound emits a pungent odor and a malodorous, oozing liquid. Other secondarily invading flies (e.g., *Musca domestica* and *Lucilia*, *Calliphora*, *Phormia*, and *Sarcophaga* species) that may be attracted to this contaminated wound are referred to as secondary screwworms.[2]

Diagnosis

Third-stage larvae are about 15 mm long and are covered with bands of tiny spines around their body segments. Deeply pigmented tracheal tubes are apparent on the dorsal aspect of the caudal end of the larvae in this most advanced stage of *C. hominivorax* (Figure 1). Once diagnosed in any animal (including humans), *C. hominivorax* must be reported to state and federal authorities to ensure its eradication and elimination from our borders.[10] The introduction of screwworm larvae has the potential to develop into a major animal disease emergency. The response to such emergencies is established by set protocol and regulations.[5]

Treatment and Zoonotic Potential

State and federal veterinarians must lead any treatment and control efforts. Any larvae not submitted for diagnostic examination must be killed and incinerated immediately. Chlorinated hydrocarbon γ-hexachlorocyclohexane and such organophosphates as chlorfenvinphos, diazinon, and bromophos-ethyl have been used to kill *C. hominivorax* larvae as they infest an animal. Synthetic pyrethroids (permethrin, cypermethrin, and cyprothrin) have recently been used as sheep dips.[10] Ivermectin administered at 200 µg/kg to infested animals has been shown effective.[2] State and federal authorities must oversee operations necessary for the elimination of adult flies from the environment.[3–5] Adult male and female flies breed only once during their lifetimes, which is a critical fact that aids in control. If γ-irradiated male flies are released into the environment, they will breed with wild females but no offspring will result.[2]

C. hominivorax remains an economically important fly, especially to the cattle industry. Veterinarians must

be on constant guard to deter its reintroduction. Cattle are not the only affected species; any warm-blooded animal (including humans) is subject to *C. hominivorax* infestation.[2]

CATEGORY II

Chrysomyia bezziana

C. bezziana (the Old World screwworm fly or Bezzi's blowfly) is another obligatory wound parasite that is quite similar in pathology to *C. hominivorax*. This fly is native to countries throughout much of tropical and subtropical Africa, the Indian subcontinent, and southeastern Asia (from southern China in the north to New Guinea in the south).[11] It is widespread in the United Arab Emirates and Oman[12] but is not found in Australia.[13] There has been one report of a possible introduction into the United States in Hawaii in 1971.[14]

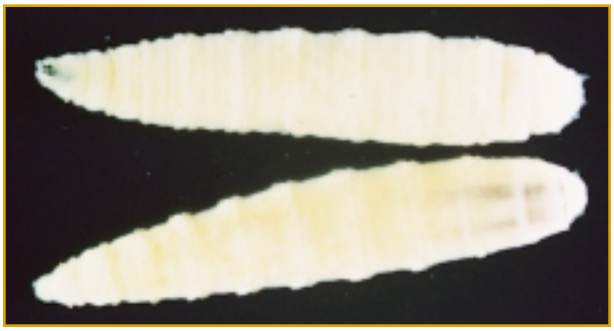

Figure 1—Dorsal and ventral views of the third-stage larvae of *Cochliomyia hominivorax*. Note that the dorsal view of the posterior end reveals the characteristic, deeply pigmented tracheal tubes that are diagnostic for this dipteran larva.

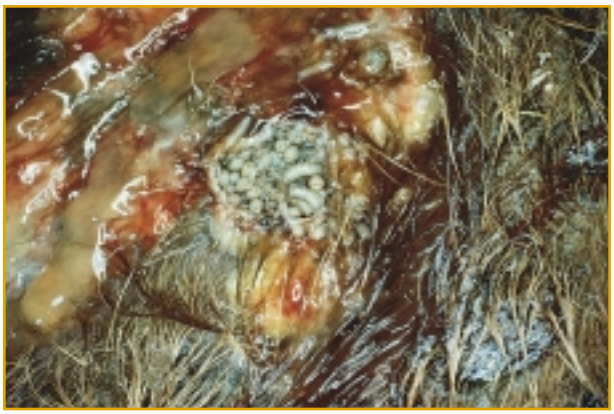

Figure 2—Cutaneous myiasis lesion caused by third-stage larvae of *Chrysomyia bezziana*. (Courtesy of Dr. J. P. Spradbery, CSIRO Division of Entomology, Canberra, Australia.)

Clinical Presentation

As with *C. hominivorax*, no skin wound is too insignificant as a site for *C. bezziana* larval development. The larvae feed en masse, producing a cavernous lesion characterized by progressive liquefactive necrosis and hemorrhage (Figure 2). With further larval development, most infested animals demonstrate a lack of sensation at the site of the wound and the surrounding area. A significant loss in body weight occurs in animals infested with *C. bezziana*.[15]

Diagnosis

Veterinarians are more likely to observe the larval stages (i.e., maggots) within wounds rather than the free-flying adult flies. Adult flies have been reported on livestock vessels and commercial aircraft.[16] Live larvae may be collected from infested animals (Figure 3) and then placed in a screened container partially filled with loose, dry gravel. The larvae should be allowed to pupate, and the emerging adults may then be identified. Descriptive and taxonomic keys are available for both larval and adult *C. bezziana*.[17] As with *C. hominivorax*, state (territorial) and federal animal health officials must be contacted if this pathogen is diagnosed. This fly has the potential to duplicate the distribution of *C. hominivorax* in both North and South America.[11]

Treatment and Zoonotic Potential

As with *C. hominivorax*, adult male and female *C. bezziana* flies breed only once during their lifetimes. *C. bezziana* can therefore also be controlled with the sterile male release technique described for *C. hominivorax*.

Single injections of ivermectin administered to infested cattle at doses of 50, 100, and 200 µg/kg killed 100% of maggots for at least 6, 12, and 14 days, respectively.[13] A similar dosing regimen may be used to treat small animals infested with *C. bezziana*. In established cases, survival of maggots after treatment depended on their age. Maggots 2 days of age or younger were killed, whereas older larvae demonstrated greater tolerance. Residual protection at the 200-µg/kg level lasted from 16 to 20 days. Any larvae that survived treatment failed to mature to adulthood.[13]

Any warm-blooded animal may be parasitized by *C. bezziana*. Infestations of humans with the larvae of *C. bezziana* are very common in endemic areas throughout the world.[17]

Cordylobia anthropophaga

C. anthropophaga (African Tumbu fly, "ver du Cayor," or skin maggot fly) has been introduced into the United States via dogs.[18] Two additional cases have been reported in dogs returning to the United Kingdom[19] and Germany.[20] This non–blood-feeding Dipteran fly is native to African nations south of the Sahara Desert, most commonly those countries bordering the Atlantic Ocean and in eastern and southern Africa.[18] U.S. citizens traveling or residing in sub-Saharan Africa

must understand the dangers of importation of this fly into this country. Depending on the area of introduction (e.g., a tropical or semitropical climate, such as Florida, Hawaii, or Puerto Rico), it may be possible for the fly to become established.[18]

Clinical Presentation

Infestation is characterized by a small, erythematous papule, which appears 2 to 3 days after larval penetration. Within days, the papule enlarges until it becomes a nodule that resembles a boil (furuncle), hence the term *furuncular myiasis.* At the center of the nodule is a pore through which serous fluid oozes. This fluid can be hemorrhagic or purulent and often contains the larva's feces.

Dogs with thin, soft skin seem to be more suitable hosts for larval development than are dogs with thick skin. Preferential sites of infestation are the feet, genitals, tail, and axillary region. In enzootic areas, mild infestations do not produce clinical disease in dogs. Massive infestations may induce marked swelling and edema, especially if larvae are in proximity. Larvae may penetrate deep into tissue, causing considerable damage and eventually host death.[18]

Figure 3A

Figure 3B

Figure 3—(**A**) Third-stage larvae of *Chrysomyia bezziana.* (**B**) Scanning electron photomicrograph of the anterior end of *C. bezziana.* (Courtesy of Dr. J. P. Spradbery, CSIRO Division of Entomology, Canberra, Australia.)

Diagnosis

The presence of furuncular swelling with a central respiratory opening may lead to a tentative diagnosis of cutaneous myiasis caused by *C. anthropophaga.* Larvae may be extracted from the furuncular swelling (Figure 4). If the animal resides in North America, *Cuterebra* species should be included in the differential diagnosis. Diagnosis is often confirmed by either residence in or history of travel to sub-Saharan Africa. Because this fly can possibly survive within tropical or semitropical states or territories, it is imperative that the Centers for Disease Control and Prevention (CDC) be informed of the presence of this foreign pathogen.[18]

Treatment and Zoonotic Potential

Larvae can be removed by coating the breathing pore with a thick viscous compound, such as petrolatum. Clogging the pore causes the larva to become hypoxic. The larva then exits its cavity in search of oxygen. Light pressure at the edge of the lesions also aids in extraction. If these techniques fail, surgical excision may be necessary.[18] Larvae are capable of penetrating any part of the skin surface of humans.[18]

Dermatobia hominis

D. hominis (tropical warble fly, berne, nuche, ver macaque, or torsalo) has never been introduced into the United States in dogs or cats; however, two separate cases have been reported in France[21] and in the Netherlands[22] in dogs returning from Brazil. This Dipteran fly is native to Latin America, from Mexico to northern Argentina.[22] U.S. citizens traveling to or residing in Latin America must understand the dangers of parasitism by and importation of this parasite.

The climatic and biotic factors for most of the United States are favorable to the survival of these flies. If fly larvae enter the United States via dogs or cats during spring or summer, the parasite could establish itself within U.S. borders. Introduction of these flies might result in serious consequences because they are capable of developing in a wide variety of definitive hosts, from humans to both domesticated and wild mammals and birds.

Clinical Presentation

The two cases imported in European dogs exhibited furuncular (boil-like) lesions in the regions of the lateral thorax, feet, and medial side of the hind limb.[21,22]

The furuncular lesion with its central respiratory opening is virtually identical to that of *C. anthropophaga*.

Diagnosis

Diagnosis of *D. hominis* is made by demonstration of typical lesions containing larval stages (Figure 5) and by either residence in or a history of travel to an enzootic area in Latin America. Veterinarians must remember that seasons are reversed for countries and continents south of the equator. If the animal resides in North America, *Cuterebra* species should be included in the differential diagnosis. As with *C. anthropophaga*, it is imperative that the CDC be informed if this foreign pathogen has entered U.S. borders.

Treatment and Zoonotic Potential

The recommended method of extracting larvae from humans is by applying liquid paraffin or petrolatum to the larva's breathing hole. In response to the lack of oxygen, the larva appears at the breathing hole, where it can be extracted with gentle pressure and thumb forceps. Ivermectin (0.2 mg/kg) has been administered to dogs with *D. hominis*; however, the dead larvae produced subcutaneous inflammatory reactions that did not heal well. Likewise, topically administered fenthion (15 mg/kg) has not been proven efficacious in dogs. Topical organophosphate and pyrethrins have been used extensively to treat this condition in South American cattle; however, there is limited information regarding their effectiveness in dogs. Perhaps the safest method is viscous occlusion of the pore, followed by manual pressure and extraction. If this is not successful, surgical removal is an alternative.[22] Humans are suitable definitive hosts for this parasite.[22]

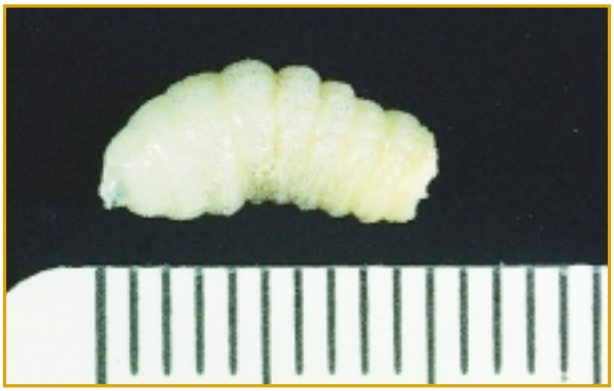

Figure 4A

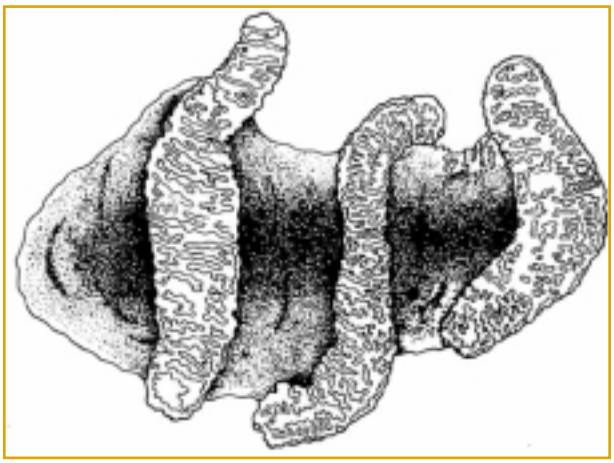

Figure 4B

Figure 4—(**A**) A third-stage larva of *Cordylobia anthropophaga*. Note that the larva is sparsely covered with tiny black spines. (**B**) The unique respiratory spiracular plate diagnostic for this dipteran larva.

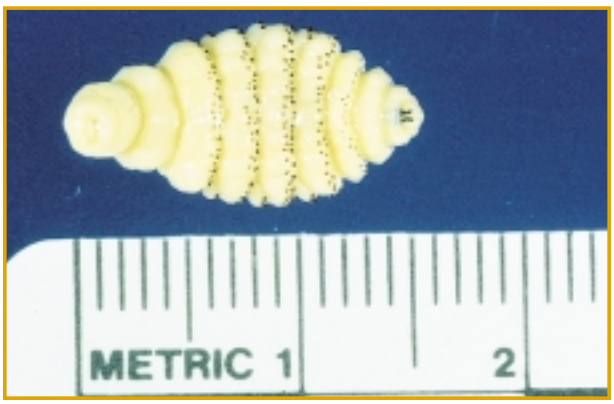

Figure 5—A third-stage larva of *Dermatobia hominis*.

Ixodes holocyclus

I. holocyclus (scrub tick or paralysis tick) is native to the bush and scrub country within a few miles of the eastern coast of Australia. *I. holocyclus* has never been introduced into the United States in dogs or cats; however, an imported case has been reported in New Zealand in a dog returning from Australia.[23] Its natural definitive hosts are the bandicoot, possum, wallaby, kangaroo, koala, and echidna. Domesticated animals (most commonly dogs[23]) are accidental hosts.

Clinical Presentation

The tick's salivary glands produce a potent neurotoxin, which produces an ascending motor paralysis in susceptible hosts. The adult female tick causes most cases of tick paralysis because it produces the greatest amount of neurotoxin. A single female tick can produce enough neurotoxin to kill a large susceptible dog.[23] Repeated exposure usually leads to antitoxic immunity.[24] Most cases of tick paralysis are seen in the late winter or spring but may occur at any time during the warm season.[23] As mentioned previously, veterinarians residing in the northern hemisphere must remember that the seasons are reversed for the "down under" countries and continents.

Diagnosis

Clinical signs of paralysis caused by *I. holocyclus* vary. Early clinical signs include initial liveliness followed by

vague restlessness, altered vocalization, cough, and dysphagia. Affected animals may demonstrate slight incoordination 4 to 7 days after the tick attaches; however, this sign may be delayed for as long as 13 days. Incoordination progresses to an ascending motor paralysis that occurs first in the hindlimbs and advances to the forelimbs. All muscle groups are eventually affected, including the respiratory and cardiac muscles, and death results.[23] Ticks may be found on any part of the body but especially the interdigital spaces and within the external ear canal.[24] The adult tick may be identified by its characteristic mouth parts.[25] A representative of the National Tick Collection[a] will be able to definitively identify ticks suspected of being *I. holocyclus*.

Treatment and Zoonotic Potential

It is important to search for and mark ticks by clipping the haircoat around the tick, then painting the tick with a pyrethroid solution and leaving it attached. Spot treatment with an alcoholic solution of permethrin and a bath with an aqueous solution of permethrin are effective means of treatment. Removing live ticks from a paralyzed patient may worsen symptoms and possibly precipitate an anaphylactic crisis or accelerate paresis. *I. holocyclus* occasionally produces a similar paralytic condition in humans.[23]

Antiserum must be administered and the patient kept quiet. The use of promethazine is controversial because it may increase pulmonary vascular resistance and thus increase mortality. Cardiovascular drugs (e.g., phenoxybenzamine) must be administered and oxygen enrichment or mechanical ventilation used for respiratory support. Cimetadine is commonly used to decrease gastric acid secretion; affected animals may regurgitate because of paralysis of the esophagus.[26]

Ticks may be removed by sliding a small pair of scissors or forceps around the mouth parts, immediately adjacent to the skin, and detaching the tick with an outward jerking motion. The tick must not be squeezed during the extraction process because further toxin may be injected into the tissue from its salivary glands.[25] All animals returning from enzootic areas must be bathed or dipped in an acaricidal solution containing a pyrethroid or permethrin.[26]

Rhipicephalus sanguineus

R. sanguineus (brown or tropical dog tick) is native to North America; it is widely distributed, however, and has been recorded in all continents and some islands between the latitudes 50° North and 35° South.[27] The tick has been occasionally introduced to nations in extreme northern climates[28–30] and island nations, such as the United Kingdom[31,32] and New Zealand.[27,33] Capable of transmitting a wide variety of serious foreign pathogens, *R. sanguineus* serves as vector for *Coxiella burnetti* (Q fever),[32,33] *Rickettsia conorii* (boutonneuse fever),[29,32] and *Ehrlichia canis*[32] as well as an intermediate host for *Babesia canis* (canine babesiosis).[32,33] These ticks enter countries not only via pets, but they have also been known to infest travelers' cars, clothing, and baggage.[32]

Clinical Presentation

Adult ticks are found most often in the ears and between the toes of dogs, whereas larval and nymphal stages are found in the long hair at the back of the neck. This tick can infest both the definitive host and its environment. It has been associated with a wide variety of human and animal pathogens (protozoal, rickettsial, bacterial, and viral), some of which may not be native to the United States or North America.[34]

Diagnosis

This tick is colloquially referred to as the brown dog tick because it has a uniform, brown color. Its basis capituli (i.e., the base of the mouth parts) has a unique, hexagonal shape that is diagnostic for the genus.[2] Ticks may be recovered from infested animals as well as from the host's environment.

Treatment and Zoonotic Potential

A wide variety of on-animal acaricidal solutions are available in the United States and Canada. Veterinarians must remember that kennel environments must also be treated in order to control this parasite.

R. sanguineus will feed on humans and has been shown to transmit certain zoonotic conditions, such as Rocky Mountain spotted fever[34] and boutonneuse fever.[29]

Rickettsia conorii

R. conorii (i.e., boutonneuse fever, Mediterranean spotted fever) is the rickettsial agent that produces tick-borne typhus in northern Africa and the countries surrounding the Mediterranean.[29] It has been reported in southern France, Spain, Italy,[35] Israel, and Turkey and also extends into the Crimea and southeastern Asia.[34] *R. conorii* affects dogs and humans; however, dogs do not show clinical signs. This disease is frequently diagnosed in humans who have recently traveled to enzootic areas with the family dog. Dogs are infected with *R.*

[a]Contact Dr. James Keirans, National Tick Collection, Institute of Arthropodology and Parasitology, Georgia Southern University, PO Box 8056, Statesboro, GA, 30460-8056; 912-681-5564.

sanguineus, the tick that serves as the vector for this rickettsial agent.[30,35–40]

Clinical Presentation

Dogs do not sustain the infection for long and do not show clinical signs. Dogs are important, however, because they bring the infected ticks into intimate contact with humans.[30,35–40]

Diagnosis

Serology may be used to diagnose cases of *R. conorii* in dogs.[36,38] Veterinarians must examine dogs for the presence of the tick vector, *R. sanguineus*. Because *R. sanguineus* is capable of reproducing in the home environment, the household must also be thoroughly examined.

Figure 6—Scanning electron photomicrograph of the anterior sucker of the nasal leech *Myxobdella annandalei.* (Courtesy of Dr. R. Gothe, Institute for Comparative Tropical Medicine and Parasitology, Ludwig Maximilian University, Munich, Germany.)

Treatment and Zoonotic Potential

Treatment in humans is based on the use of tetracyclines,[37,40] such as doxycycline. This rickettsial agent has tremendous zoonotic potential. If the tick vector from the dog bites humans, those humans may become infected with boutonneuse fever. Individuals must not rub their eyes after crushing or removing infected ticks from dogs[29] because infection may also result from contamination of the conjunctiva with infectious tick material.

Dirofilaria repens

D. repens is a filarial parasite that produces nodules in subcutaneous, ocular, and pulmonary tissue in humans.[41–43] *D. repens* is commonly found in the subcutaneous tissue in dogs and cats in areas of Europe, Africa, and Asia[43]; it is the most common filariid of dogs in Nigeria.[2] *D. repens* has never been introduced into the United States in dogs or cats; however, cases have been reported in humans returning from international locales.[41,43] Canine infection with *D. repens* is now a proven zoonosis in Sri Lanka.[42]

Clinical Presentation

Adult *D. repens* live in dogs within deep-seated subcutaneous cysts. The adult worms produce superficial cysts in humans in Sri Lanka; symptoms depend on the infection site.[42]

Diagnosis

Unsheathed microfilariae are 260 to 360 µm in length and may be found within blood and lymphatic fluid of the canine definitive host.[2] Morphologic evaluation of cross- and longitudinal sections of the adult parasite is based on greatest body diameter in microns (175 to 550 µm) and the number (95 to 105), round shape, and interridge distance of the longitudinal ridges on the adult parasite's midbody.[44] Veterinarians should consider history of residence in or travel to an area enzootic for *D. repens* when making a diagnosis.[41–43]

Treatment and Zoonotic Potential

Adult worms must be excised from infected dogs and humans. Microfilariae may be cleared from the canine definitive host with oral ivermectin (150 µg/kg) with retreatments at 6-month intervals or with diethylcarbamazine (1 mg/kg/day increased gradually over 3 days to 6 mg/kg/day in divided doses). The latter treatment modality must be maintained for 21 days.[b] In areas enzootic for *D. repens*, heartworm preventive compounds will probably prove efficacious in preventing infections. In addition, no lesions or associated clinical signs could be detected in dogs after experimental inoculation of infective larvae.[45]

As mentioned previously, *D. repens* can be a zoonotic parasite. Infection can occur in both humans and dogs traveling in endemic areas. Humans, however, are considered to be "dead-end" hosts.[41–43] Because of the myriad of mosquito species in North America, the susceptibility of dogs not protected by a *D. immitis* preventive, and the ability of humans to serve as dead-end hosts, the presence of this parasite in an imported dog or cat must not be taken lightly.

Myxobdella annandalei

M. annandalei, a bloodsucking nasal leech, has been imported into Germany in the nostrils of a dog returning from Nepal (Figure 6).[46] Leeches are found worldwide, always closely associated with an aquatic environment. Most leeches are found in fresh water, but a few

[b]Personal communication: Dissanaike AS, University of Colombo, Rajagiriya, Sri Lanka, 1998.

are found in salt water and there are terrestrial varieties that occur in moist, damp soil. It is important to remember that leeches are hermaphroditic. Male and female organs are located on adjacent body segments; however, self-fertilization is not possible.[47]

Clinical Presentation

In the hills of Nepal, nasal leeches are a common problem in domestic ruminants and buffalo. During the dry season, leeches proliferate in watering sites located 990 to 1800 m above sea level. When animals drink from these watering holes, the young leeches enter their nostrils. After infestation, blood and bloody mucus may be observed around the nostrils. Affected animals sneeze frequently. In the later stages of infestation, leeches may be observed protruding from the nostrils. Animals become greatly agitated and anorexic, and dyspnea often results. Leeches can remain within the nasal passages for as long as 5 months and may grow to a length of 16 cm. Infestation with eight or more leeches often results in anemia.[48]

Diagnosis

When water is misted or aerosolized into the nares of the animal, the leeches will often protrude.[48] An endoscope may be passed up the nares to reveal any remaining leeches.

Treatment and Zoonotic Potential

After leeches are induced to protrude from the nostrils, they should be briefly dipped into a small container filled with an aqueous solution of ivermectin (10 µg/ml). The leeches will gradually become flaccid and fall out of the nostril. Within 3 hours, all leeches are expelled. If the leeches do not fall in response to the application of ivermectin, the solution can be applied in the form of nose drops.[48] Although they will feed on humans, leeches have very limited zoonotic significance.

Trypanosoma evansi (Surra)

T. evansi is native to Africa, South America, and Asia.[49] *T. evansi* causes mammalian trypanosomiasis or surra, which is a Hindi word meaning rotten, resulting in observable clinical signs in camels, horses, and dogs. Subclinical infections have been reported in cattle, buffalo, sheep, and goats; these animals may serve as reservoir hosts for the disease.[2] This form of trypanosomiasis, the most widely distributed form of the disease, occurs throughout the Indian subcontinent, Far East, Near East, northern Africa, Philippines, and Central and South America.

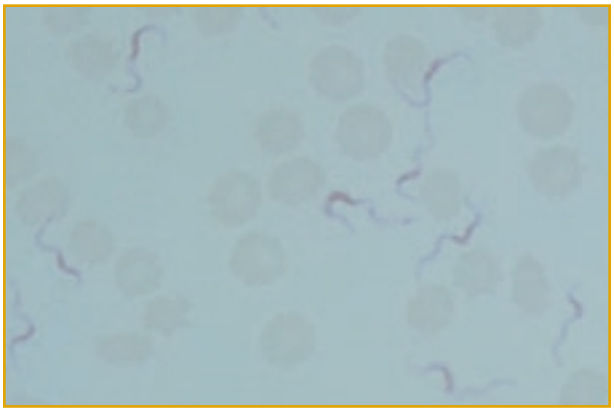

Figure 7—Trypomastigotes of *Trypanosoma evansi* within a canine peripheral blood smear. (Courtesy of Dr. R. J. Slappendel, Utrecht University, Utrecht, The Netherlands.)

Clinical Presentation

Surra in dogs is defined by high morbidity and mortality rates. Infected dogs demonstrate malaise, weight loss, conjunctivitis with corneal clouding, hemolytic anemia, and thrombocytopenia; untreated dogs will die. Some reports indicate neurologic signs in the final stages of the disease.[49]

Diagnosis

Trypomastigotes of *T. evansi* are readily demonstrable in freshly stained peripheral blood smears (Figure 7).[49] The trypomastigote form is known by its anterior flagellum and its prominent undulating membrane.

Treatment and Zoonotic Potential

Subcutaneous administration of berenil at 3.5 to 7.0 mg/kg has been reported to be curative.[49] *T. evansi* is not a zoonotic condition.[2]

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