ZOONOSES

Recognition, Control, and Prevention
DEDICATED TO

PAUL R. SCHURRENBERGER, DVM, MPH

He was a friend and colleague who left us before his time. However, in his time he contributed much to our understanding of the zoonoses.

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When *Outline of the Zoonoses* (Schnurrenberger and Hubbert) was published in 1981, its purpose was stated as follows:

*The primary purpose of this outline is to present the veterinary practitioner with an all-encompassing, concise, desk-top reference to the zoonotic diseases. In daily work, the practitioner holds the key to these diseases through accurate diagnosis and treatment of the patient and counsel to the owner on preventive and control practices. This belief in the importance of the practitioner and the primacy of preventive medicine influenced the formulation of this outline. Although designed for the veterinarian, it should be of value to physicians, nurses, public health officials, wildlife workers, and many others.*

This purpose continues in *Zoonoses: Recognition, Control, and Prevention* with the updated synopses presented in Section IV.

The authors have assembled Sections I, II, and III as a result of needs identified during a collective century of professional experience. During that time, extraordinary changes have occurred in the practice of human and veterinary medicine. However, the diseases, including zoonoses, have not changed—we just understand them better. Much of our newer knowledge of the zoonoses has come from greater insight into their epidemiology and ecology. A major force for improved zoonoses prevention and control has been the introduction and application of economic analysis. Section I presents a historic background, Section II describes current principles, and Section III predicts changes we can expect in the future.
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SECTION I

Introduction
INTRODUCTION TO THE ZOONOSES

Definition

Zoonoses ("zoonosis" is singular) are diseases the agents of which are transmitted between vertebrate animals and people. It is the interaction of agent, host (degree of susceptibility), and the environment they share that determines whether or not transmission of the agent will be successful, leading to infection and, ultimately, occurrence of disease. Carrier hosts, individuals infected without overt signs of disease, are important in the persistence of many zoonotic agents. Vertebrate animals are the reservoirs (where the agent persists in nature) of zoonoses. The agents may be transmitted either directly or indirectly by fomites or vectors. Many diseases are shared by other animals and people, but the reservoir is in the inanimate environment (soil, water), not a vertebrate animal. For example, soil is the reservoir for agents of systemic mycoses (e.g., Blastomyces dermatitidis, Coccidioides immitis, Nocardia asteroides) and many of the mycobacterioses (e.g., Mycobacterium intracellulare, Mycobacterium kansasii). Water is the reservoir for the agents of other mycobacterioses (e.g., Mycobacterium marinum) and free-living pathogenic amoebae such as Naegleria fowleri. Still other diseases clinically similar in animals and people, and once thought to be zoonotic, are now known to be the result of endogenous infection from the individual’s own normal flora. Examples are actinomycosis (e.g., Actinomyces bovis—cattle, Actinomyces israeli—people) and disease caused by Gram-negative anaerobic organisms such as Bacteroides melaninogenicus.
Initial Recognition of the Agents

Clinical features for a few of the zoonoses have been recognized since early history. For example, the signs of encephalitis in dogs with rabies, ringworm in people and animals, *Mycobacterium bovis*-associated scrofula in children, glanders and tetanus in horses and humans, and epidemic urban plague (*Yersinia pestis*) have been described for many centuries.\(^{24,16,23,24}\) Confirmation of the specific etiology for all zoonoses, however, awaited Leeuwenhoek’s invention of the microscope in the late 1600s and the scientific discoveries that have followed to date.\(^{9,19}\)

All major microbial and parasitic categories, from viruses to helminths, include some zoonotic agents. It is not surprising, therefore, that the larger parasites were the first to be examined with the primitive microscopes. In 1758, Linnaeus (the father of scientific classification) included descriptions of two cestodes (*Dipylidium caninum* and *Diphyllobothrium latum*), a trematode (*Fasciola hepatica*), and two nematodes (*Ascaris lumbricoides*, essentially identical to *A. suum*, and *Dracunculus medinensis*) in *Systema Naturae*, 10th edition. Although earlier reports exist, such as the description of *Fusciflua hepatica* by Jehan de Brie in 1379 (perhaps the earliest description of a parasite), organized classification/description began with Linnaeus. By 1800, most of the cestodes had been described and, by the 1870s, so had most of the trematodes.\(^{5,6,30}\) Although Bilharz had described *Schistosoma haematobium* in 1851, the zoonotic schistosomes were described a little later, *S. japonicum* in 1904, *S. mansoni* in 1907.\(^{36}\) Most of the roundworm species were first described during the mid- to late 1800s, although descriptions of several *Thelazia* species first appeared between 1910 and 1930 and it was not until 1935 that *Angiostrongylus cantonensis* (rat lung worm) was first described.\(^{13,17}\) From 1900 to 1910, numerous methods were described to concentrate parasite ova in feces by sedimentation, filtration, and/or flotation before microscopic examination. With only slight improvements since, these are the most widely used parasitologic methods today.\(^{4,14}\) The zoonotic species of most genera of protozoa (*Babesia, Entamoeba, Giardia, Pneumocystis, Toxoplasma, Trypanosoma*) were first described between 1885 and 1915. The plasmodia of primates, however, were much later, descriptions first appearing in the 1930s to 1960s.\(^{35}\) Clarification of species among the coccidia and their role as zoonoses, especially *Besnoitia, Cryptosporidium, Hammondia*, and *Sarcocystis*, has been even more recent, continuing to the present.

The mycotic etiology of ringworm was first described microscopically by David Gruby in the 1840s.\(^{5}\) Raimond Sabouraud began using culture medium in the 1890s. The cat as reservoir of human *Microsporum canis* infection was identified in 1902. Some early descriptions of agents of systemic mycoses around 1900 suggested they were protozoa. Soil was first reported as the
reservoir of a systemic mycosis, *Coccidioides immitis*, in 1932. Although the agents of the systemic mycoses are capable of producing disease when transmitted from an infected host to a susceptible host, these occurrences are uncommon, usually involving accidental inoculation of infectious material. The resulting disease is also atypical, appearing as a regional lymphangitis rather than a pneumonia and subsequent generalized infection. Inasmuch as these infections always involve transmission from an individual previously exposed to infection from an inanimate reservoir in nature, the agents of the systemic mycoses are not considered zoonotic because there is no animate reservoir. Beginning with *Coccidioides immitis* in 1932, soil has been identified as the reservoir for all of the agents of the systemic mycoses.

Although there were earlier microscopic observations of *bacterial zoonotic agents*, such as the spirochaete causing tickborne relapsing fever seen in blood (1873), it was the isolation of *Bacillus anthracis* by Robert Koch in 1877 that really opened the door to their description.\(^9\)\(^{19}\) Use of aniline dyes to stain bacteria (and other organisms) in wet preparations began in the 1870s. In 1877, Koch reported the first observations of stained bacteria (*B. anthracis*) in a dried film. Hans Gram introduced decolorizing and counterstaining, i.e. the Gram stain, in 1884. Koch's early efforts at producing pure cultures involved serial dilutions in liquid medium. He introduced the first solid medium, the cut surface of a potato, in 1881. The Petri dish (glass, of course, not disposable plastic) was introduced in 1887. By 1890, most zoonotic bacteria had been described, with reports continuing to appear until 1916, when the first description of *Leptospira* was published. There was a hiatus from this time until the late 1940s (with the exception of the report of *Listeria monocytogenes* in 1924), when a few more descriptions began to emerge—*Pasteurella pneumotropica* (1948), *Yersinia enterocolitica* (1949), *Vibrio parahaemolyticus* (1953), and *Borrelia burgdorferi* (1982).\(^3\)\(^{27}\)\(^{31}\)

Within 2 years of the initial isolation of *Pseudomonas pseudomallei* from a human patient in 1911, the agent of melioidosis had been recognized in several animal species in and around the laboratory in Rangoon. In the 1920s, only 1 of more than 20,000 wild rats examined was found infected in Saigon. It was not until the 1960s, however, that soil was identified as the true reservoir and that melioidosis was recognized as not, in fact, a zoonosis. Spread among animals and people essentially does not occur.

In 1909, Howard Ricketts described *Rickettsia rickettsii*, the agent of Rocky Mountain spotted fever.\(^15\) Most of the remaining *zoonotic rickettsiae* were first described in the 1930s, with the agents of Queensland tick typhus (*R. australis*) and rickettsialpox (*R. akari*) first reported in 1946. The first edition of *Bergey's Manual of Determinative Bacteriology* appeared in 1923, and has become the bible of bacterial and rickettsial classification.

In 1798, Edward Jenner demonstrated that people exposed to pox material from cows were later protected against smallpox.\(^11\) And, in 1804,
Zinke showed that normal dogs developed rabies when injected with saliva from rabid dogs. Although these were classic reports of responses to infection, they did not establish the viral nature of the agents involved. In 1898, Loeffler and Frosch passed foot-and-mouth disease serially from animal to animal using a cell-free filtrate, establishing the *filterable agent* (virus).* Rabies virus isolation, as well as Negri's demonstration of the inclusion body, were first reported in 1903. This was followed by isolation of vesicular stomatitis virus, Newcastle disease virus, yellow fever virus, and louping ill virus in 1926, 1927, 1928, and 1929, respectively. Tissue culture techniques for the isolation of viruses were introduced in the late 1920s and use of the chick embryo began in 1931.

Until this time, animal virology involved infecting animals with cell-free material derived from other animals. Guinea pigs, mice, and rabbits have been standard laboratory animals since the 1800s. Intracerebral injection of mice, introduced by R.A. Alexander in 1933 to produce African horse sickness vaccine, was one of the most significant innovations in virology of the time. Max Theiler, using this method, introduced the 17D yellow fever vaccine four years later. The use of ferrets in influenza studies began in the 1930s. With the advent of the electron microscope in 1934, viruses could be seen and described. Most initial reports of the isolation of zoonotic viruses appeared during the 1930s.

Although a lapse in reporting occurred during World War II, considerable new knowledge was acquired regarding zoonoses of military significance. Reporting picked up again during the 1950s, especially isolations of the less common arboviruses. Infant (suckling) mice were already widely used to isolate arboviruses in 1948, when the technique was used in the first isolation of Coxsackie A virus. In 1961, several outbreaks of human hepatitis involving prior contact with chimpanzees and other primates were first reported. Initially, these cases were referred to as simian hepatitis. Within the next decade, however, it became evident that these were undoubtedly all viral hepatitis A infections resulting from exposure to primate shedders rather than a new virus. With the exception of swine vesicular disease (1966), the most recently discovered viral zoonoses share the characteristic of high case fatality rates in people—Argentine hemorrhagic fever (1958), Bolivian hemorrhagic fever (1959), Marburg disease (1967), Lassa fever (1970), Ebola disease (1976), and Korean hemorrhagic fever (1978).

The infectious etiology of cat scratch disease is only now being unraveled with presumptive evidence that the agent is a bacterium, *Rochalimaea henselae.* A clinical relationship to the presence of antibody to simian immunodeficiency virus in laboratory workers has not been established.

**Arthropod vectors** are involved in the transmission of several zoonotic protozoan parasites, bacteria, rickettsiae, and viruses. In 1893, Smith and Kilborne first demonstrated vectorborne transmission of an infectious agent
with tick transmission of Babesia bigemina, the agent of bovine babesiosis. Ogato, in 1896, recovered Yersinia pestis from rat fleas. Ross, in 1898, demonstrated avian malaria parasites in mosquitoes. In 1899, Tanaka proposed the larval red mite as a vector of scrub typhus. Reed and colleagues, in 1900, proved mosquitoborne spread of yellow fever. In 1903, Bruce and colleagues reported transmission of Trypanosoma gambiense by tsetse flies. In 1906, Ricketts and King independently demonstrated tick transmission of the rickettsial agent of Rocky Mountain spotted fever. By the beginning of the 1900s, the concept of vectorborne spread had been established for zoonotic members of all the major groups of infectious agents except fungi.

Application of current technology (not all of which is laboratory-based) in the recognition of agents and vectors involved in zoonotic diseases in people and animals will be presented in later chapters.

**Initial Recognition of the Diseases**

For many zoonoses, the risk of infection for people and animals, as well as the signs of illness, are sufficiently similar that an association between the two has been known for a long time. For instance, outbreaks of eastern and western equine encephalitis affected people and horses at the same time so often over the years that they were associated long before the agents were known. Horses, in fact, were frequently a useful sentinel before vaccination became a widespread practice. Death as a possible outcome of being bitten by a rabid dog is well known in primitive societies. Therefore, when the agents of these diseases were identified in people and animals, it was no surprise that they were the same.

In contrast, connecting disease in people, animal reservoir, and agent has required considerable effort by many investigators to solve the riddles involved. For example, David Bruce observed the agent of undulant fever in the spleen of a fatal human case on the island of Malta in 1886 and isolated it a year later. It was nearly 20 years before the goat as reservoir of Brucella melitensis and raw goat milk as the vehicle were identified. Ten years after Bruce’s discovery (1897), Bernard Bang isolated an agent of bovine abortion, Brucella abortus. It was another 18 years (1925) before this agent was found to also cause undulant fever or human brucellosis. The first description of an outbreak of undulant fever caused by Br. abortus involved college students who drank raw cow’s milk in the dormitory.

Adolf Weil, in the early 1880s, was able to distinguish clinically and epidemiologically a jaundice he observed in sewer workers in Paris from other forms of jaundice. In 1907, Stimson observed the agent in the kidney of a patient believed to have died of yellow fever. In 1916, Inada and
colleagues reported the isolation of the spirochaete and that field rodents were the reservoir. Unfortunately, Hideyo Noguchi confused the signs of yellow fever with those of leptospirosis and mistakenly believed that leptospires were the cause of yellow fever. He spent nearly a decade performing field trials with a vaccine he developed to protect people against yellow fever. Noguchi died of yellow fever in 1928, the year its viral agent was identified. This is an ironic example of a consequence of reporting the etiology of a disease based on insufficient evidence.

In 1912, McCoy and Chapin identified *Francisella tularensis* as the cause of a die-off in ground squirrels similar to plague in Tulare County, California. Although tularemia is widespread in the Northern Hemisphere, oddly it was nearly 30 years after it was first recognized in rodents that the first human case was reported in Tulare County. A major reason is that this locale does not harbor some of the more efficient tick vectors for spread of the agent from rodents to people.

McFadyean and Stockman first reported vibrionic abortion in sheep and cattle, caused by *Vibrio fetus*, in 1913. In 1947, more than 30 years later, Vinzent and colleagues reported the first human case of septicemic vibriosis. They attributed their success in part to the fact that they used a diagnostic laboratory that also did veterinary work. Although isolation of microaerophilic bacterial pathogens under reduced oxygen tension was a common practice in veterinary diagnostic laboratories at this time, it was later that the technique was introduced to medical laboratories. What was once called *Vibrio*, now called *Campylobacter, jejuni* was originally thought to be a commensal of animals—not being of any pathogenic significance. Since the first report by Bokkenheuser in 1970, campylobacteriosis has become recognized as a leading cause of human enteric disease. The principal reason for this increase in recognition has been a change in techniques used in diagnostic enteric bacteriology.

In 1967, the first human cases of Marburg disease were reported in Germany and Yugoslavia among researchers who had worked with the tissues of African green monkeys. Although a few human cases have been reported since in Africa, no reservoir in nature has been identified in spite of intensive searching. Ebola virus disease was first recognized in Zaire and the Sudan in 1976, the former being an extensive nosocomial outbreak. Ebola virus is morphologically identical to Marburg virus but immunologically distinct. The reservoir of Ebola virus, too, has not been identified. Lassa fever was first recognized in Nigeria in 1969 and is caused by a virus related to other viruses known to have rodent reservoirs. It was later found in the African multimammate mouse, *Mastomys natalensis*.

The status of the zoonotic protozoa changed dramatically when Rommel and Heydorn, in 1972, fed beef and pork containing sarcocysts to people, resulting in the shedding of what was for a long time previously called
Isospora hominis. What was thought to be a one-host cycle turned out to be a two-host cycle akin to taeniasis and toxoplasmosis.

Lyme disease was first described clinically in 1977 as a juvenile rheumatoid arthritis (rash and fever syndrome recognized later) in Lyme, Connecticut, following a tick bite. The agent, Borrelia burgdorferi, was reported in 1982. The wildlife reservoir of the spirochaete and its effects on wild and domestic animals have been described more recently.

Human cowpox infections have long been recognized in association with endemic cowpox in cattle. In 1978, the virus was isolated from pox lesions in a cat. In 1989, catpox was observed in a person at the site of a cat scratch. To date, the actual reservoir of catpox/cowpox virus is uncertain inasmuch as it is neither cats nor cows.34

The term emerging microbial threat has been used to characterize newly identified agents and newly recognized diseases in people.20 It is likely that the agents are not newly evolved, but have existed in nature, particularly in animals. The following factors have been associated with the emergence of these threats: changing human demographics and behavior, technology and industry, economic development and land use, international travel and commerce, microbial adaptation and change, and failure of public health measures. Recognizing the presence of a disease in an animal or person, or in a flock, herd, or community, usually is much easier than recognizing the specific factors (including agent) responsible for its occurrence. Means of improving recognition of zoonoses will be presented in later chapters.

### Development of Control and Prevention Strategies

Early religious laws against the eating of pork, whether or not the believers related the prohibition to risk of disease, certainly had a salutary effect in regard to risk of contracting trichinosis and cysticercosis.

During the second plague pandemic, beginning in the 1300s, quarantine (literally, 40 days) was a common practice to protect the susceptible residents of a seaport from whatever evil (plague) was being delivered to them by those on board a ship from another land (often where epidemic plague was known to exist).24 Of course, the ship's rats died along with the crew if plague was aboard. Protecting a susceptible community by quarantine usually was not feasible for other diseases. For one thing, many of the diseases involved carriers who appeared healthy at the time of entry and who were less likely to develop with surety the severe signs of disease such as seen with plague. Therefore, they slipped in with little notice.

At one time, more than 80 major diseases of cattle flourished in East Africa because the cattle there were exposed to ruminants brought by Arab slave traders on their travels to the coast from farther west in Africa or from
across the sea in Asia. Many of these diseases are zoonoses and persist today.

Boiling of milk was an early custom among some societies. Unfortunately, the custom did not always include boiling the milk to make cheese, thereby permitting spread of some of the more important milkborne agents. Commercial pasteurization of milk became widespread in developed countries during the 1920s, virtually eliminating milkborne brucellosis and tuberculosis except on the farm where there were still infected cows and milk was consumed raw, and in villages where raw milk was distributed.

Vaccine and vaccination, the words themselves being derived from vaccinia, the cowpox virus, are synonymous with protection against disease. For nearly 200 years, the vaccine was used to protect against smallpox, and the principal source of this vaccine was the lymph of calves that had been scarified with the virus. The vaccine provided an essential tool in the worldwide eradication of smallpox, so declared officially by the World Health Organization on December 9, 1979 (the last naturally occurring case was in Somalia in October 1977). Zoonoses for which vaccines are widely used to protect people with high risk of exposure include plague, rabies, and yellow fever.

Glanders, a scourge of horses and people since antiquity, caused by Pseudomonas mallei, was eradicated from most of the world by slaughter of animals found positive to the intrapalpebral mallein test. The test was introduced in the early 1900s, and since the 1940s the only remaining endemic areas are in Asia.

A law has existed for nearly a century requiring working dogs used to shepherd livestock to be tested for tapeworms, and successfully treated if any were found before entering the United States. Since the mid-1950s, Iceland and New Zealand have had successful Echinococcus granulosus eradication programs based on test and treatment of dogs. Mycobacterium bovis is quite sensitive to several antibiotics, and drugs have been used cost-effectively to eradicate tuberculosis in cattle herds. Most national bovine tuberculosis eradication programs, however, were in existence long before these drugs were available and are based on the use of intradermal tuberculin tests and slaughter of positive animals. In the United Kingdom and New Zealand, the program has run into difficulty because of infection in wildlife.

Environmental control of arthropod vectors really came of age with the advent of DDT during World War II. Before then, fumigation was used to kill fleas within structures for plague control. Arsenicals were used to dip animals for tick control. Mosquito control involved modification of harborage, such as emptying water from receptacles to eliminate breeding sites for Aedes aegypti and extensive drainage programs for reduction of other vectors.

When people and their livestock lived in small, isolated communities,
they shared relatively few zoonoses. The diseases were principally those for which a significant carrier state persisted in people and/or animals or the disease was sufficiently chronic, e.g., tuberculosis, to provide a continuous source of infection for new susceptibles. Times have changed. The world population has increased roughly fivefold in the last century.\textsuperscript{18,21,22} Between 1880 and 1980, the urban percentage of the United States population increased from 28\% to 74\%.\textsuperscript{7} This shift to urban living is the norm on all continents. During the period 1920 to 1980, the percentage of people actually living on farms declined from 30\% to 2\%. Even though much of the world is now urban, people and their possessions (food, animals) are also more mobile today. In fact, today we have a more complex picture involving relative risk of exposure to zoonotic agents and relative need for planned strategies of zoonoses control and prevention. Explanation of the underlying principles of zoonoses control and prevention, and their current applications, will be presented in later chapters.

### Chronology of Events in Zoonoses

#### Recognition, Control, and Prevention

**Before 1300**
- Ancient descriptions of clinical observations.
- Early religious dietary practices.

**1300-1500**
- Jehan de Brie (1379) described first parasite, \textit{Fasciola hepatica}.
- Quarantining ships from foreign (plague-affected) ports began.

**1501-1700**
- Leeuwenhoek (late 1600s) invented microscope and published first descriptions of microorganisms.

**1701-1800**
- Jenner (1798) demonstrated that cowpox protected against smallpox.
- By 1800, most cestodes had been described.

**1801-1850**
- Zinke demonstrated rabies transmission by saliva in dogs (1804).
- Gruby (1840s) described mycotic etiology of ringworm.

**1851-1900**
- By 1870s, most trematodes had been described.
- Koch (1877) isolated \textit{Bacillus anthracis}.
- Gram stain introduced (1884).
- Petri dish introduced (1887).
- By 1890, most zoonotic bacteria had been described.
Tick (vector) transmission of *Babesia* demonstrated (1893).

**1901-1950**

Rabies virus isolated and Negri body described (1903).
Goats demonstrated to be the reservoir of *Brucella melitensis* and goat milk a source of human infection (1905).
Ricketts (1909) described agent of Rocky Mountain spotted fever.
Commercial milk pasteurization introduced (1920s).
Electron microscope invented (1934).
17D yellow fever vaccine introduced (1937).
In 1930s, most zoonotic rickettsiae and viruses isolated.
Vector control with DDT introduced (1940s).

**Since 1950**

Smallpox officially eradicated worldwide (1979).

### Table 1.1. List of zoonotic agents

<table>
<thead>
<tr>
<th>Agent</th>
<th>Disease in Humans (Class*)</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Armillifer armillatus</em></td>
<td>Pentastomid infection (U)*</td>
</tr>
<tr>
<td><em>Armillifer grandis</em></td>
<td>Pentastomid infection (U)</td>
</tr>
<tr>
<td><em>Armillifer moniliformis</em></td>
<td>Pentastomid infection (U)</td>
</tr>
<tr>
<td><em>Linguatula serrata</em></td>
<td>Pentastomid infection (U)</td>
</tr>
</tbody>
</table>

**Pentastomids**

<p>| <em>Ancylostoma braziliense</em>      | Cutaneous larva migrans (5) |
| <em>Ancylostoma caninum</em>          | Cutaneous larva migrans (5) |
| <em>Angiostrongylus cantonensis</em>  | Parasitic meningo-encephalitis (5) |
| <em>Angiostrongylus costaricensis</em>| Parasitic meningo-encephalitis (5) |
| <em>Anisakis marina</em>              | Anisakiasis (5)              |
| <em>Ascaris suum</em>                 | Ascariasis (5)               |
| <em>Baylisascaris procyonis</em>      | Visceral larva migrans (5)   |
| <em>Brugia malayi</em>                | Filariaisis (U)              |
| <em>Bunostomum phlebotomum</em>       | Cutaneous larva migrans (5)  |
| <em>Capillaria aerophila</em>         | Capillariasis (5)            |
| <em>Capillaria hepatica</em>          | Capillariasis (5)            |
| <em>Capillaria philippinensis</em>    | Capillariasis (5)            |
| <em>Dioctophyma renale</em>           | Giant kidney worm (U)        |
| <em>Dipetalonema perstans</em>        | Filariaisis (U)              |
| <em>Dipetalonema strepiocerca</em>    | Filariaisis (U)              |
| <em>Dirofilaria immitis</em>          | Filariaisis (U)              |
| <em>Dirofilaria repens</em>           | Filariaisis (U)              |
| <em>Dirofilaria tenuis</em>           | Filariaisis (U)              |
| <em>Dracunculus insignis</em>         | Dracunculiis (5)             |
| <em>Dracunculus medinensis</em>       | Dracunculiis (5)             |
| <em>Gnathostoma spinigerum</em>       | Cutaneous and visceral larva migrans (5) |</p>
<table>
<thead>
<tr>
<th>Agent</th>
<th>Disease in Humans (Class*)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Nematodes</strong></td>
<td></td>
</tr>
<tr>
<td><em>Haemonchus contortus</em></td>
<td>Trichostrongyliasis (U)</td>
</tr>
<tr>
<td><em>Loa loa</em></td>
<td>Filariasis/Loiasis (S)</td>
</tr>
<tr>
<td><em>Onchocerca cervicalis</em></td>
<td>Filariasis/Onchocerciasis (S)</td>
</tr>
<tr>
<td><em>Onchocerca volvulus</em></td>
<td>Filariasis/Onchocerciasis (S)</td>
</tr>
<tr>
<td><em>Ostertagia spp.</em></td>
<td>Trichostrongyliasis (U)</td>
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<tr>
<td><em>Strongyloides fulleborni</em></td>
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</tr>
<tr>
<td><em>Strongyloides myopotami</em></td>
<td>Strongyloidiasis (S)</td>
</tr>
<tr>
<td><em>Strongyloides procyonis</em></td>
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<tr>
<td><em>Strongyloides ransomi</em></td>
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<td><em>Strongyloides ratti</em></td>
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<td><em>Strongyloides stercoralis</em></td>
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<td><em>Strongyloides westeri</em></td>
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<td><em>Thelazia callipaeda</em></td>
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<td><em>Toxocara canis</em></td>
<td>Visceral larva migrans (S)</td>
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<td><em>Toxocara cati</em></td>
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<td><em>Trichinella spiralis</em></td>
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<td><em>Trichostrongylus spp.</em></td>
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<tr>
<td><em>Uncinaria stylocephala</em></td>
<td>Cutaneous larva migrans (S)</td>
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<td><strong>Trematodes</strong></td>
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<td><em>Amphimerus pseudoofelineus</em></td>
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<tr>
<td><em>Clinostomum complanatum</em></td>
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<td><em>Clonorchis sinensis</em></td>
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<td><em>Dicrocoelium dendriticum</em></td>
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<td><em>Dicrocoelium hastes</em></td>
<td>Dicrocoeliasis (U)</td>
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<td>Echinostomiasis (U)</td>
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<td><em>Echinostoma lindoense</em></td>
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<td><em>Echinostoma revolutum</em></td>
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<td><em>Echinococcus granulosus</em></td>
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<tr>
<td>Agent</td>
<td>Disease in Humans (Class*)</td>
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<td><strong>Echinococcus multilocularis</strong></td>
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<td><strong>Echinococcus vogeli</strong></td>
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<td><strong>Hymenolepis diminuta</strong></td>
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<td><strong>Spirometra thelli</strong></td>
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<td>Tapeworm-sheep/goat (U)</td>
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<td><strong>Taenia ovis</strong></td>
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<td><strong>Sarcocystis suihominis</strong></td>
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<td>Trypanosomiasis, African (3B)</td>
</tr>
<tr>
<td>var. gambiens**</td>
<td>Trypanosomiasis, African (3B)</td>
</tr>
<tr>
<td>var. rhodesiense**</td>
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</tr>
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