Zoonotic Diseases

(also known as zoonoses)

- Zoonoses are caused by infections that are shared between animals and people.
- Bacterial, viral, parasitic or fungal infections.
- More than 6 out of every 10 known infectious diseases in people are spread from animals.
- 3 out of every 4 new or emerging infectious diseases in people are spread from animals.

Cases of infections reported in the Czech Republic in 2013-2017

	2013	2014	2015	2016	2017
Campylobacteriosis	18 389	20 903	21 102	24 291	24 508
Salmonelosis	10 280	13 633	12 739	11 912	11 779
Lyme disease	4 646	3 743	2 913	4 694	3 939
Hepatitis E	218	299	412	339	344
Tularemia	36	49	59	59	51
Listeriosis	35	37	34	46	30
Leptospirosis	7	37	17	18	21
Taeniasis	30	18	6	5	6
Ehrlichiosis	8	6	2	6	4
Leishmaniasis	2	0	1	3	2
Psittacosis	1	0	0	2	1
Echinococcosis	2	6	3	4	1
Brucellosis	0	0	0	1	1
Q - fever	0	0	1	2	**0**

Why Are Zoonoses a Public Health Concern?

- The number and types of zoonotic diseases are increasing as we can **travel** in less time than it takes for disease symptoms to develop after exposure (incubation period).
- Human manipulation of the environment (e.g., **climate change**) is helping some zoonotic disease vectors thrive.
- Overuse of antibiotics has made some of these diseases harder to treat (**antibiotic resistance**).
- Some organisms that cause zoonoses could be used for **bioterrorism**.
- **Vaccines** are not available to protect humans against many zoonoses.
- **Treatments** may not exist or be readily available.

How do germs spread between animals and people?

Direct contact: with body fluids of an infected animal - petting or touching animals, and bites or scratches.

Indirect contact: with areas where animals live or objects that have been contaminated with germs - aquarium tank water, pet habitats, chicken coops, plants, and soil, as well as pet food and water dishes.

Vector-borne: ticks, mosquitos or fleas.

Foodborne: Eating or drinking something unsafe (such as unpasteurized milk, undercooked meat or eggs, or raw that are contaminated with feces from an infected animal).

Who is at a higher risk of serious illness from zoonotic diseases?

Anyone can become sick from a zoonotic disease, including healthy people.

Some people are more likely than others to get really sick, and even die, from infection. These groups of people include:

- Children younger than 5
- Adults older than 65
- People with weakened immune systems
- Pregnant women

Prevention

Keep hands clean.

Prevent bites from mosquitoes, ticks, and fleas.

Avoid bites and scratches from animals.

Vaccinate yourself.

Enteric Zoonotic Diseases.

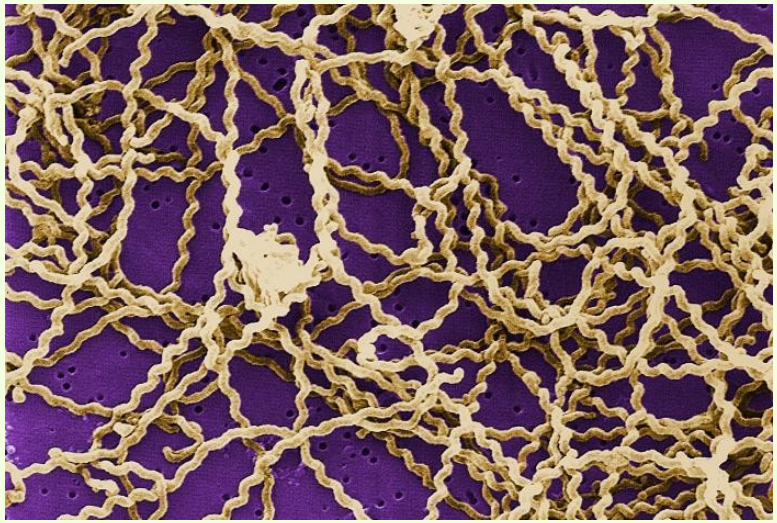
Mostly Salmonella and Campylobacter, but Escherichia coli too.

Leptospirosis

Infectious disease of humans and animals that is caused by pathogenic spirochetes of the genus *Leptospira*.

It is considered the most common zoonosis in the world.

The most important reservoirs are rodents, and rats are the most common source worldwide.



Leptospirosis

Leptospira species infect a wide range of animals, including mammals, birds, amphibians, and reptiles. Humans are considered accidental hosts.

The organism is typically transmitted via exposure of mucous membranes or abraded skin to the soil or fresh water contaminated with the urine of an animal that is a chronic carrier.

- **Occupational exposure** probably accounts for 30-50% of human cases (farm workers, veterinarians, plumbers, workers in the fishing industry and sewer workers).
- **Sporting events** that involve fresh water or hiking.
- **Flooding**

Leptospirosis

The traditional system divided the genus into 2 species: the pathogenic *Leptospira interrogans* and the nonpathogenic *Leptospira biflexa.*

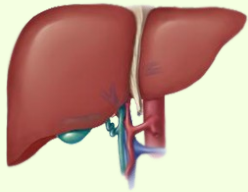
The current classification system is based on DNA homology and recognizes the heterogeneity of the classic leptospires, dividing *L interrogans* and *L biflexa* into **20** named **species**. Within these species, leptospires are **further grouped by serogroups**, and strains on the basis of microscopic agglutination testing (MAT).

Although certain species (eg, *L interrogans*) have a classic association with Weil disease, knowledge of the species type does not necessarily help predict disease severity.

Leptospirosis

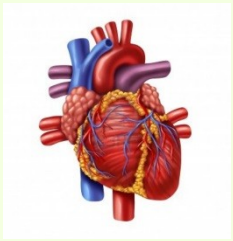
- In 90% of cases, leptospirosis manifests **as an acute febrile illness** with a biphasic course and an excellent prognosis. Nonspecific signs and symptoms of leptospirosis (eg, fever, headache, nausea, vomiting) are **often confused with viral illness**.
- In 10% of cases, the presentation is more dramatic, and the infection has an overall case fatality rate of 5%-10%. Known as **Weil disease** or icteric leptospirosis, the classic definition of this form of leptospirosis includes **fever, jaundice, renal failure, and hemorrhage**. Other organ systems (ie, pulmonary system, cardiac system, central nervous system) are also frequently involved.

Leptospirosis



- The incubation period is usually 5-14 days but has been described from 72 hours to a month or more.
- Mediators induced by the leptospire are the suspected causes of the disease's various manifestations (endotoxin, hemolysin and lipase as possible sources of pathogenicity).

- The most consistent pathologic finding in leptospirosis is **vasculitis** of capillaries and resulting loss of red blood cells and fluid through enlarged junctions and fenestrae, which cause secondary tissue injury, probably accounts for many of the clinical findings.



Leptospirosis - laboratory

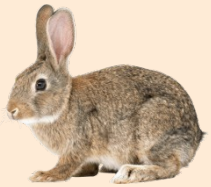
- Elevated **ESR**, **CRP** and **WBC** (3,000-26,000 x 109/L)
- Significant **anemia** due to pulmonary and gastrointestinal hemorrhage can occur
- The **platelet count** may be diminished and **coagulation times** may be prolonged as a component of disseminated intravascular coagulation (DIC)
- Levels of serum **urea and creatinine** may be profoundly elevated in the anuric or oliguric phase
- Serum **creatine kinase** levels are often elevated in patients with muscular involvement.
- Serum **bilirubin** levels (disproportional to transaminases) elevate as part of the obstructive disease due to capillaritis in the liver.

Leptospirosis

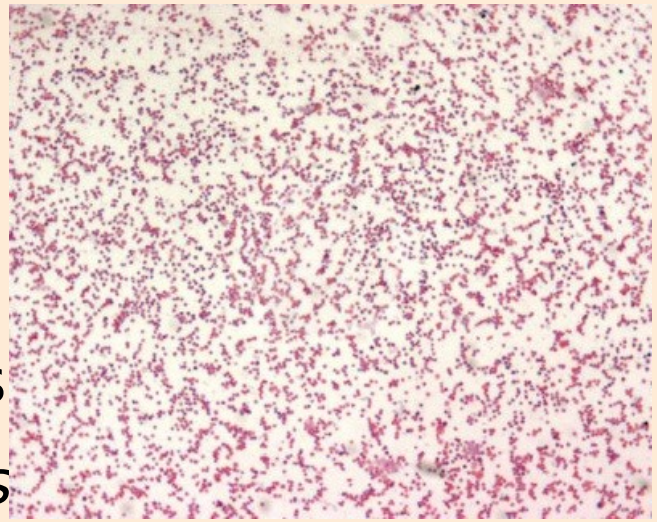
- Culture times for *Leptospira* are long and recovery rates are low.
- The criterion standard for serologic identification of leptospires, **microscopic agglutination testing** (MAT), is available only at reference laboratories. **Paired acute and convalescent serum specimens** can provide delayed confirmation of the diagnosis.
- Blood and CSF may produce positive PCR or cultures during the first 7-10 days of symptoms.

Leptospirosis - treatment

- **Treatment is begun empirically** in patients with a exposure history and compatible symptoms.
- In uncomplicated infections that do not require hospitalization, oral **doxycycline** has been shown to decrease duration of fever and most symptoms.
- In hospitalized patients (Weil disease), **penicillin G** / **3.g ceph.** are the treatment of choice + management of renal, hepatic, hematologic, and central nervous system complications.
- **Corticosteroid** therapy is controversial. Treatment with high-dose methylprednisolone (30 mg/kg/d, not to exceed 1500 mg) has been used successfully to treat patients with leptospiral renal failure and in the



Tularemia

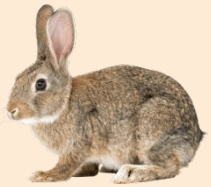


Disease of animals and humans caus

by the bacterium *Francisella tularens*

Humans can become infected through several routes:

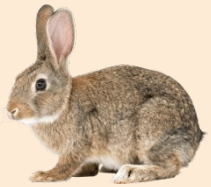
- Skin contact with infected animals (In particular, rodents, most often rabbits, but many other animals have also been known to become ill with tularemia.)
- Ingestion of contaminated water
- Tick bites
- Inhalation of contaminated aerosols or agricultural dusts



Tularemia

Two main serologically identical **biovars**, differing primarily in geographic distribution, fermentation reactions, and virulence.

- ***F tularensis* biovar *tularensis***
  - is found predominantly in North America
  - an extremely virulent organism, as few as 10-50 organisms may result in disease if inhaled or injected intradermally
- ***F tularensis* biovar *holarctica***
  - is found primarily in Europe and Asia
  - less-virulent subspecies



Tularemia

The incubation period for tularemia depends on the size of the inoculum, but ranges from 1-21 days (average 2-6 days).

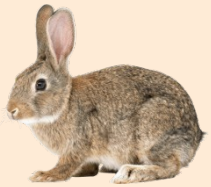
1) acute inflammatory response at entry point, necrosis

2) phagocytosis, ability to survive in infected cells is the main virulence factor

3) IC dissemination into lymphatic system

4) **A**) focal necrosis or granulomas formation

B) "cytokine storm" causing capillary leakage, tissue injury, and lethal organ failure



Tularemia

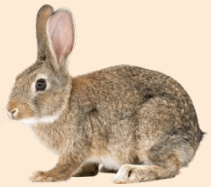
The signs and symptoms of tularemia vary **depending on how the bacteria enter the body**.

Illness ranges from mild to life-threatening.

All forms are accompanied by fever.

Main forms of this disease are listed below:

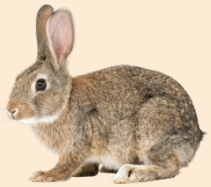
- **Ulceroglandular**
- **Glandular**
- **Oculoglandular**
- **Oropharyngeal**
- **Pneumonic**
- **Typhoidal**



Ulceroglandular form

This is the most common form of tularemia and usually occurs following a tick bite or after handling of an infected animal.

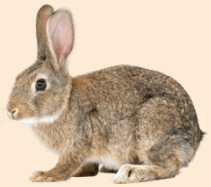
A skin ulcer appears at the site where the bacteria entered the body. The **ulcer** is accompanied by **swelling of regional lymph glands**, usually in the armpit or groin.



Glandular form

Similar to ulceroglandular tularemia but without an ulcer.

Also generally acquired through the bite of an infected tick or from handling sick or dead animals.

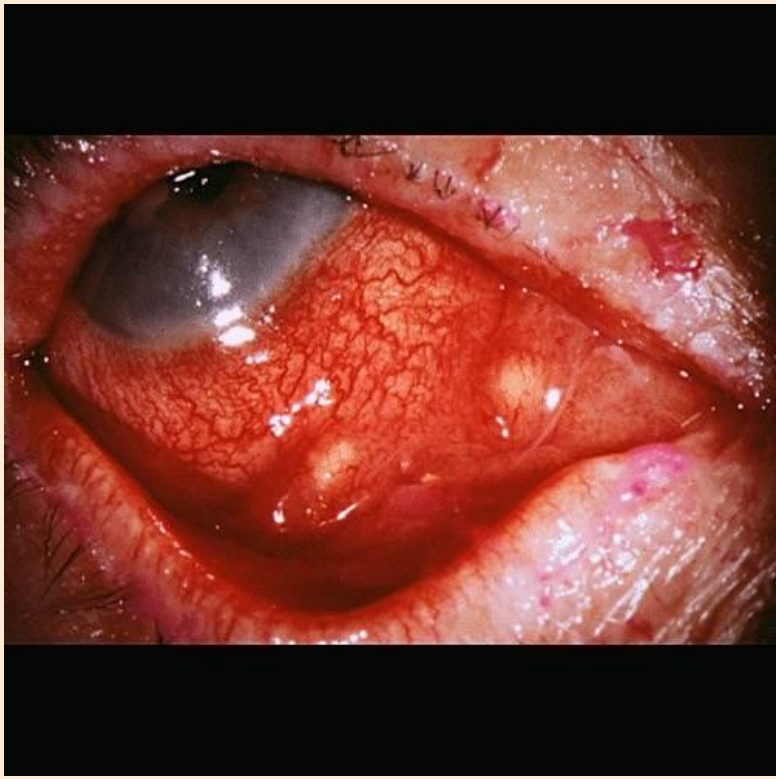


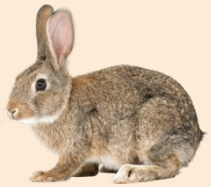
Oculoglandular form

This form occurs when the bacteria enter through the eye. This can occur when a person is butchering an infected animal and touches his or her eyes.

Symptoms include irritation and **unilateral conjunctivitis** and **swelling of lymph glands in front of the ear**.

- corneal ulceration, photophobia, lacrimation, lid edema, vision loss (rare).



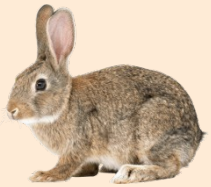


Oropharyngeal form

This form results from eating or drinking contaminated food or water.

Patients with orophyangeal tularemia may have sore throat, mouth **ulcers**, **tonsillitis**, and **swelling of lymph glands in the neck**.

- Stomatitis and exudative pharyngitis or tonsillitis; abdominal pain, nausea, and vomiting; diarrhea; gastrointestinal bleeding.

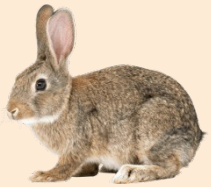


Pneumonic form

This is the most serious form of tularemia.

This form results from breathing dusts or aerosols containing the organism. It can also occur when other forms of tularemia (e.g. ulceroglandular) are left untreated and the bacteria spread through the bloodstream to the lungs.

- Dry cough, dyspnea, and pleuritic-type chest pain.



Tularemia

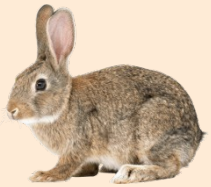
Intestinal form

- Abdominal pain, vomiting, and diarrhea

Typhoidal form

This form is characterized by **any combination of the general symptoms** (without the localizing symptoms of other syndromes)

- Fever, chills, myalgias, malaise, and weight loss.



Tularemia

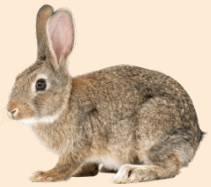
The diagnosis of tularemia is usually based on serology results (latex agglutination or ELISA).

… approx. 3 weeks…

An agglutination titer greater than 1:160 is considered presumptively positive, and treatment may be started if this result is obtained. A second titer, demonstrating a 4-fold increase after 2 weeks, confirms the diagnosis.

Indirect fluorescent antibody testing

PCR



Tularemia - treatment

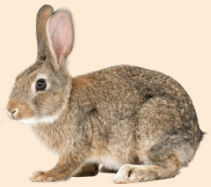
Gentamicin is considered the drug of choice in systemic forms.

Amikacin, fluoroquinolones and rifampicin were used successfully, but have limited clinical experience and in vitro data.

While chloramphenicol and **doxycycline** are clinically useful, relapse rates of up to 50% have been reported in patients treated with these agents (in US).

F. tularensis is naturally resistant to penicillins and first-generation cephalosporins.

Ceftriaxone has been examined - although it was



Tularemia

Surgical care is not needed in tularemia management unless an ulcerative lesion develops a superinfection and requires debridement or drainage is required for empyema or a fluctuant lymph node.

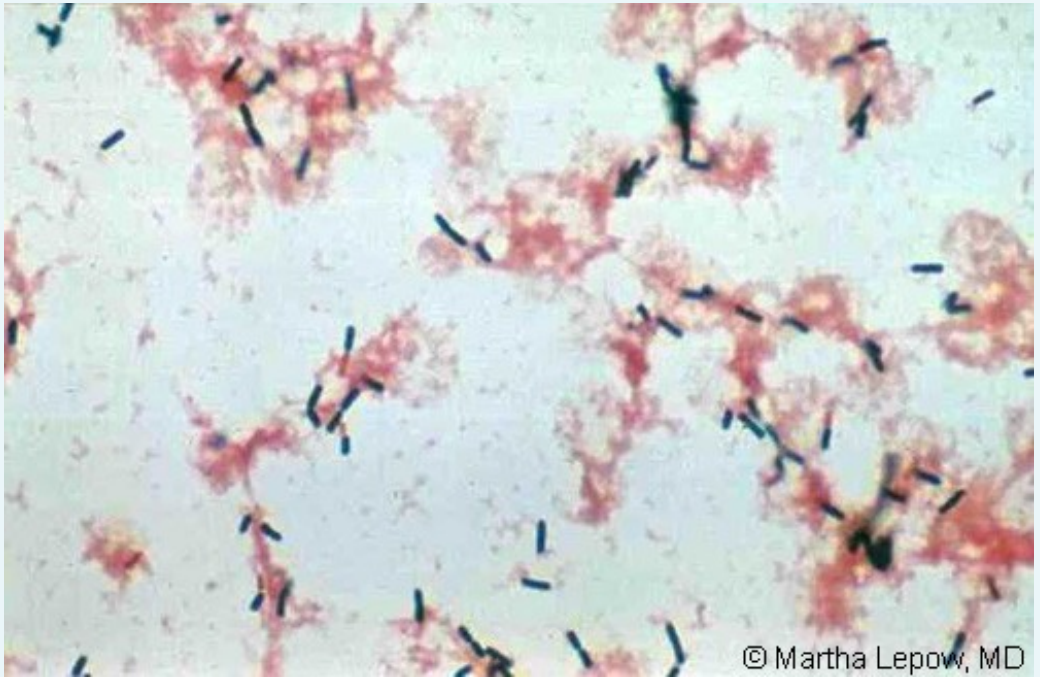
No tularemia **vaccine** is currently available.

A vaccine based on a live strain of the bacterium was previously available but is no longer produced because of concerns about unknown attenuation, safety, and production.

Listeria (Listeriosis)

Usually caused **by eating food** contaminated with the bacterium *Listeria monocytogenes* (**G+**), (which is ubiquitous in the environment).

The infection is most likely to sicken pregnant women and their newborns, adults aged 65 or older and immunocompromised.



Listeriosis

People with normal immune systems rarely develop invasive infection. However, they may experience a **self-limited acute febrile gastroenteritis** following high-dose *Listeria* exposure.

Incubation period is mostly 1-2 days.

Rarely diagnosed outside of outbreak settings.

Listeriosis - pregnancy

Many pregnant women can carry *Listeria* **asymptomatically** in their GI tract or vagina.

Pregnant women typically experience only fever and other **flu-like symptoms**, such as fatigue and muscle aches.

However, infections during pregnancy can rarely lead to chorioamnionitis, premature labor, spontaneous abortion, or life-threatening infection of the newborn.

Listeriosis – neonatal infection

Early-onset neonatal listeriosis (day 0-5) is usually acquired through transplacental transmission.

- usually associated with sepsis or meningitis

The sources of **late-onset listeriosis** (day 6-28) are less clear; they may involve exposure during delivery or nosocomial exposure.

- may present with purulent meningitis

Some neonates develop **granulomatosis infantiseptica**, disseminated rash with small pale nodules (microabscesses or granulomas) involving

Listeriosis – neonatal infection

Listeriosis presents in the same manner as other more common neonatal pathogens, such as group B streptococci and Escherichia coli:

- Respiratory distress - tachypnea, grunting, apnea
- Temperature instability
- Poor feeding
- Lethargy/irritability
- Seizures
- Granulomatosis infantisepticum

Listeriosis – children

Beyond the neonatal period, most children with *Listeria* infections have an underlying immunodeficiency.

Meningitis is developed more often.

Listeriosis – immunocompromised and aged 65 or older

Self-limited **gastrointestinal tract** or a more **severe CNS infection**, **bacteremia**, or a localized infection such as **monoarticular septic arthritis**.

- *Listeria* has a predilection for the brain parenchyma, especially the brain stem, and the meninges.
- Mental status changes are common.
- Seizures, both focal and generalized, occur in at least 25% of patients.
- Cranial nerve deficits may be present.
- Strokelike syndromes with hemiplegia may occur.

Listeriosis - diagnosis

Because *Listeria* cannot be detected by routine stool culture, febrile gastroenteritis from *Listeria* infection is rarely diagnosed outside of outbreak settings…

- **Blood culture**
- **Microscopy and culture of cerebrospinal fluid**

Laboratory results that show diphtheroids should prompt heightened awareness for the possibility of *Listeria* infection, particularly in

Listeriosis - treatment

Intravenous antibiotics must be started **immediately** when the diagnosis is suspected or confirmed.

Ampicillin is generally considered the preferred agent, but other agents may be acceptable (ie co-trimoxazole).

Gentamicin is added frequently for synergy with ampicillin.

Bacteremia should be treated **for 2 weeks.**

Longer courses may be required in the immunocompromised. (endocarditis, for 4-6 weeks;

Listeriosis prevention

Listeriosis may be part of a larger epidemic.

Year	Location	Source
2014	United States	Prepackaged caramel apples
2011	United States	Cantaloupe
2007	Massachusetts	Milk
2003	United Kingdom	Sandwiches
2002	United States	Delicatessen turkey breast
Aug 1998 to Jan 1999	United States	Hot dogs, deli meats
1997	Italy	Corn
1997	Sweden	Rainbow trout
1995	Switzerland	Soft cheese
1994	Illinois	Chocolate milk

Listeriosis prevention

- Avoid eating **soft cheese** (brie, blue-veined, …) unless it is labeled as made with pasteurized milk.
- Do not eat raw or lightly cooked **sprouts** of any kind.
- Eat cut **melon** right away or refrigerate it.

- Avoid eating hot dogs, lunch meats, refrigerated pâté or **meat** spreads, fermented or dry sausages unless they are heated to an internal temperature of 74°C just before serving.

- Do not eat refrigerated smoked **seafood** unless it is canned or shelf-stable or it is

Rabies

Viral **disease of mammals** most often transmitted through the bite of a rabid animal.

There are 10 viruses in the rabies serogroup, most of which only rarely cause human disease.

The genus Lyssavirus, includes the classic rabies virus, Mokola virus, Duvenhage virus, Obodhiang virus, Kotonkan virus, Rochambeau virus, European bat Lyssavirus types 1 and 2, and Australian bat Lyssavirus.

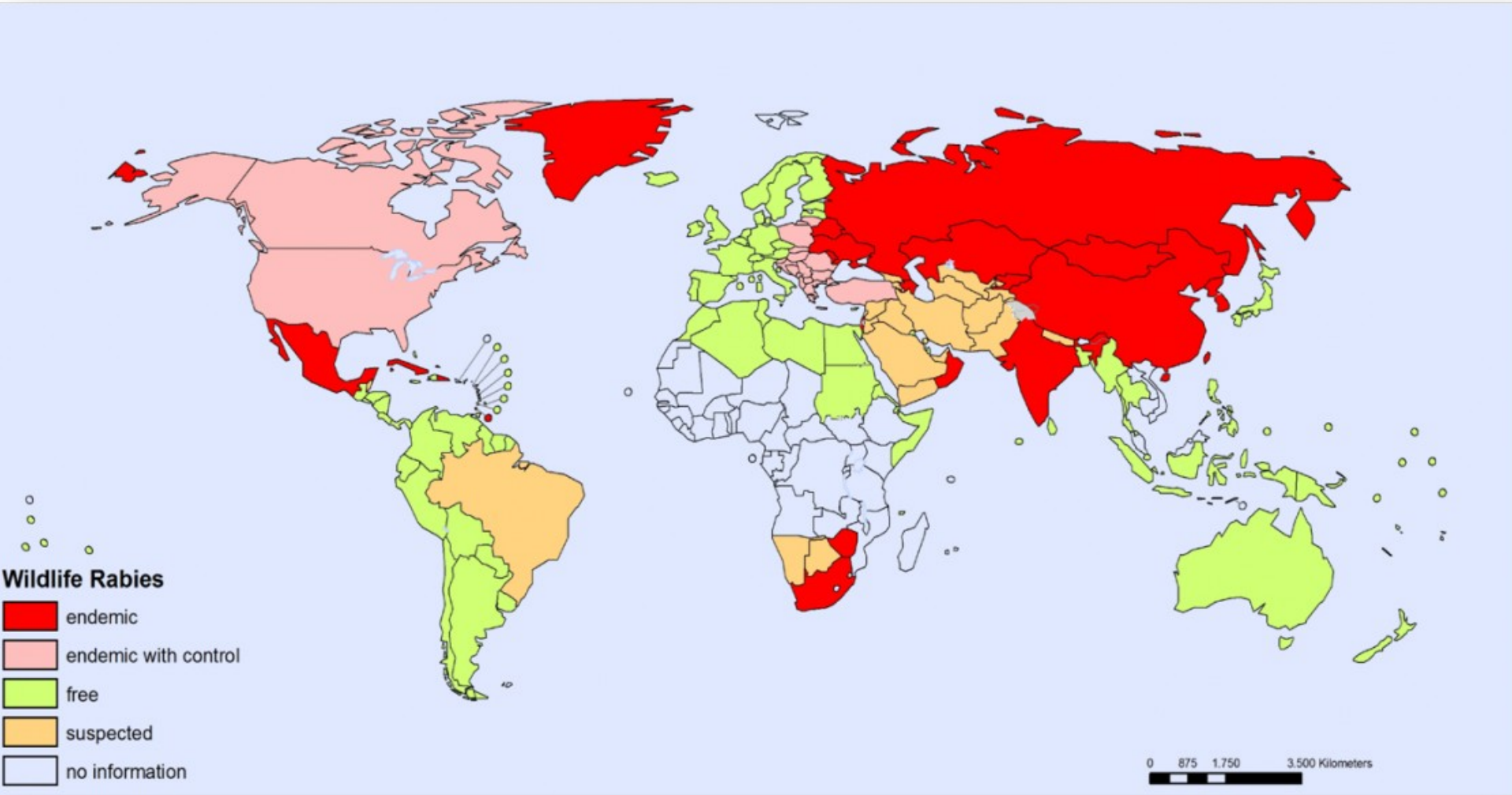
Rabies

Unvaccinated dogs are the major reservoir for rabies worldwide.

In Europe mostly foxes, bats and wolves in Eastern Europe.

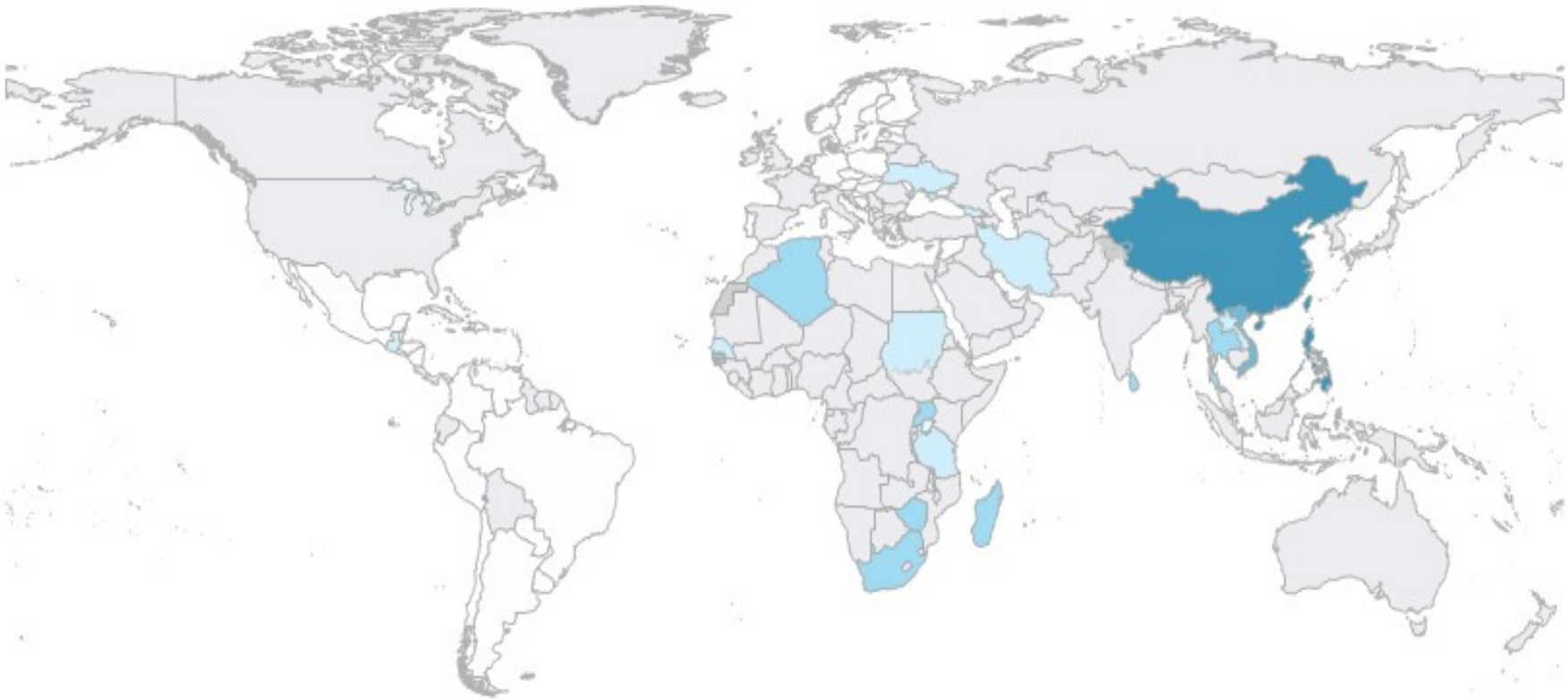
www.who-rabies-bulletin.org

Occurrence of wildlife mediated rabies



www.who-rabies-bulletin.org

Reported number of human rabies deaths, 2014



http://apps.who.int/neglected_diseases/ntddata/rabies/rabies.html

Rabies

Rabies is a highly neurotropic virus that evades immune surveillance by its sequestration in the nervous system.

Upon inoculation, it **enters the peripheral nerves**.

A prolonged incubation follows, the length of which depends on the size of the inoculum and its proximity to the CNS.

The virus travels along axons **to** enter the **spinal ganglion and** then spreads into the **CNS**.

Thereafter, the virus spreads to the periphery and **salivary glands**.

Rabies

Rabies does not cause cytotoxicity.

Death occurs from global neurologic and organ dysfunction.

- The virion bind neurotransmitter receptors in the synaptic space. Thus, its action is neurotoxic, rather than direct damage.

Rabies – incubation period

The average duration of incubation is 20-90 days, depends on the distance between entry point and CNS.

In more than 90% of cases, incubation is less than 1 year.

In most cases, there are no symptoms until the virus reaches CNS.

Rabies - prodromal period

The **virus enters the CNS**. The duration of this period is 2-10 days.

Paresthesia, pain, or intense itching at the inoculation site is pathognomonic for rabies and occurs in 50% of cases.

- Malaise
- Headaches
- Fever or chills
- Pharyngitis
- Anorexia , nausea, emesis or diarrhea
- Anxiety, agitation or depression
- Insomnia

may be present.

Rabies - acute neurologic period

The duration is 2-7 days and ends up with a coma and death.

Symptoms include **muscle fasciculations and focal or generalized convulsions.**

Patients may die immediately because of cardiorespiratory arrest or may progress to paralysis.

Rabies - acute neurologic period

Furious episodes lasting less than 5 minutes may develop

- agitation, restlessness, biting, confusion or hallucinations
- episodes may be triggered by visual, auditory, or tactile stimuli or may be spontaneous.

20 % of patients do not develop the furious form (**paralytic rabies)**

- fever and headache are prominent.

Rabies - diagnosis

History of suspected virus exposure:

- The nature of the interaction with the animal (humans may not understand what is provocative to a wild animal)
- Strange animal behavior (eg, nocturnal animal out during the daytime)
- Vaccination status of the animal for rabies
- Availability of the animal for testing

Rabies - diagnosis

Intra-vitam diagnosis in suspect human patients is based on detecting virus or viral **RNA in saliva**, in innervated **skin biopsy** samples or **hair follicles**.

Serological assays **are not** suitable for diagnosis.

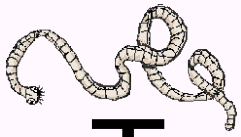
Brain tissue is the preferred specimen for **post-mortem** diagnosis in both humans and animals.

Rabies - prevention

Pre-exposure prophylaxis - recommended to anyone at increased risk for exposure to the rabies virus

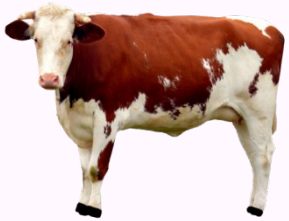
Post-exposure treatment

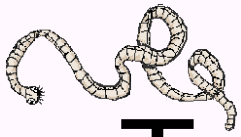
1) Any bite wound or scratches should immediately be flushed with rinsing water as first-aid procedure! Thorough washing for a minimum of 15 minutes with soap and water, detergent, povidone iodine will drastically reduce infectivity of the virus.
2) Administration of a rabies vaccine / and the rabies immunoglobulin in cases of transdermal bites or scratches, bleeding or contamination of



Taenia Infection (tapewoms)

- Of the 32 recognized species of *Taenia,* only *Taenia solium* and *Taenia saginata* are medically important.
- Approximately 50 million people worldwide are infected and approx. 50 000 people die annually of cysticercosis.
- ***T saginata*** is common in cattle-breeding regions. Areas with the highest prevalence are central Asia, the Near East, and central and eastern Africa.
- ***T solium*** is endemic in Central and South America, Southeast Asia, India, the Philippines, Africa, Eastern Europe, and China.





Taenia Infection - taeniasis

- Adult tapeworms live in the **human small intestine**.
- Humans pass mature **eggs in feces**; these eggs contaminate pastures and barnyards, where cattle and pigs ingest them.
- Upon reaching the alimentary canal of **infected animals**, the larvae are released, penetrate the gut wall, enter the circulation and **encyst in muscular tissue** (cysticerci).
- Humans develop a taeniasis by eating raw or undercooked beef or pork.

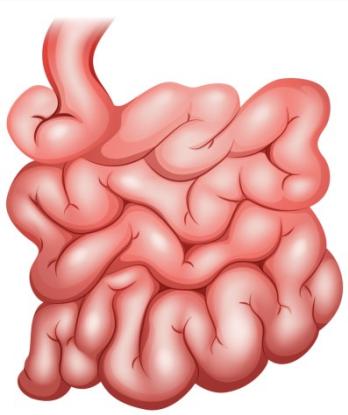
Taenia Infection - cysticercosis

Humans can also act as an intermediate host **for *T solium*.**

Cysticercosis results from human ingestion of *T solium* eggs through fecal contamination (or autoinfection). The cysticerci may develop in any organ, and their effects depend entirely on the location of the cysticerci.

Cysticercosis caused by *T saginata* is rare.

Taeniasis symptoms



Most individuals are asymptomatic or have mild complaints.

The most common complaint is passage of proglottids, which is associated with slight discomfort.

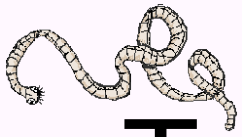
Other symptoms include the following:

- Colicky abdominal pain (more common in children)
- Nausea
- Loss of appetite or increased appetite
- Constipation or diarrhea
- Weakness, headache, dizziness, fever

Cysticercosis and neurocysticercosis

The cysticerci are most often located in subcutaneous and intermuscular tissues, followed by the eye and then the brain. Most patients have more than one cyst.

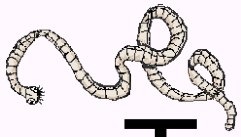
1. **Neurocysticercosis**: 3 possible syndromes
   - Convulsions and/or seizures
   - Intracranial hypertension: symptoms include headache, nausea, vomiting, vertigo, and papilledema.
   - Psychiatric disturbances: behavioral changes and learning disabilities.
2. **Muscular and dermatologic cysticercosis**: most patients are asymptomatic, subcutaneous nodules may be evident.



Taenia Infection - diagnosis

Intestinal taeniasis

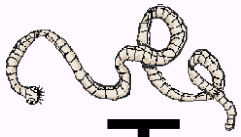
- CBC count detects eosinophilia in no more than 45% of patients.
- Examine 3 consecutive stool samples (direct and concentrated stool preparations) from patients and contacts.



Taenia Infection - diagnosis

Neurocysticercosis

- Examine stool samples as described above.
- CSF findings are abnormal in 50-90%
  - Protein levels are usually ↑, glucose levels are usually mildly ↓, predominantly mononuclear pleocytosis is common (<300/µL).
- ELISA or immunoblot on both CSF and serum
- Imaging Studies
  - Plain X-ray of the chest, neck, arms, and thighs can depict calcified cysticerci (takes >3 years to occur).
  - CT or MRI of the brain.
- Funduscopic examination
- Biopsy of subcutaneous nodules



Taenia Infection - treatment

If adult tapeworms are detected in the stools, anthelmintic therapy usually suffices.

Asymptomatic cysticercosis requires no treatment.

Treatment for symptomatic neurocysticercosis is challenging

- albendazole , praziquantel or both + high-dose glucocorticosteroids + anticonvulsants.

Ocular, ventricular, and spinal lesions may require surgical treatment because treatment with anthelmintic drugs can provoke irreversible drug-induced inflammation.

Taenia Infection

Following treatment, patients should carefully examine stools for proglottid elimination during the next 5 weeks for *T solium* infection and for 3 months for *T saginata* infection.

Individuals should avoid eating inadequately cooked beef or pork in endemic regions.

Toxocariasis

An infection caused by the **ingestion of larvae** of the dog roundworm *Toxocara canis*

or

the cat roundworm *Toxocara cati*.

The soil of parks and playgrounds is commonly contaminated with the eggs of *T canis,* and infection may cause human disease.
In humans, toxocariasis is considered an aberrant infection because **humans are incidental hosts**, as the parasites cannot successfully reach full

Toxocariasis

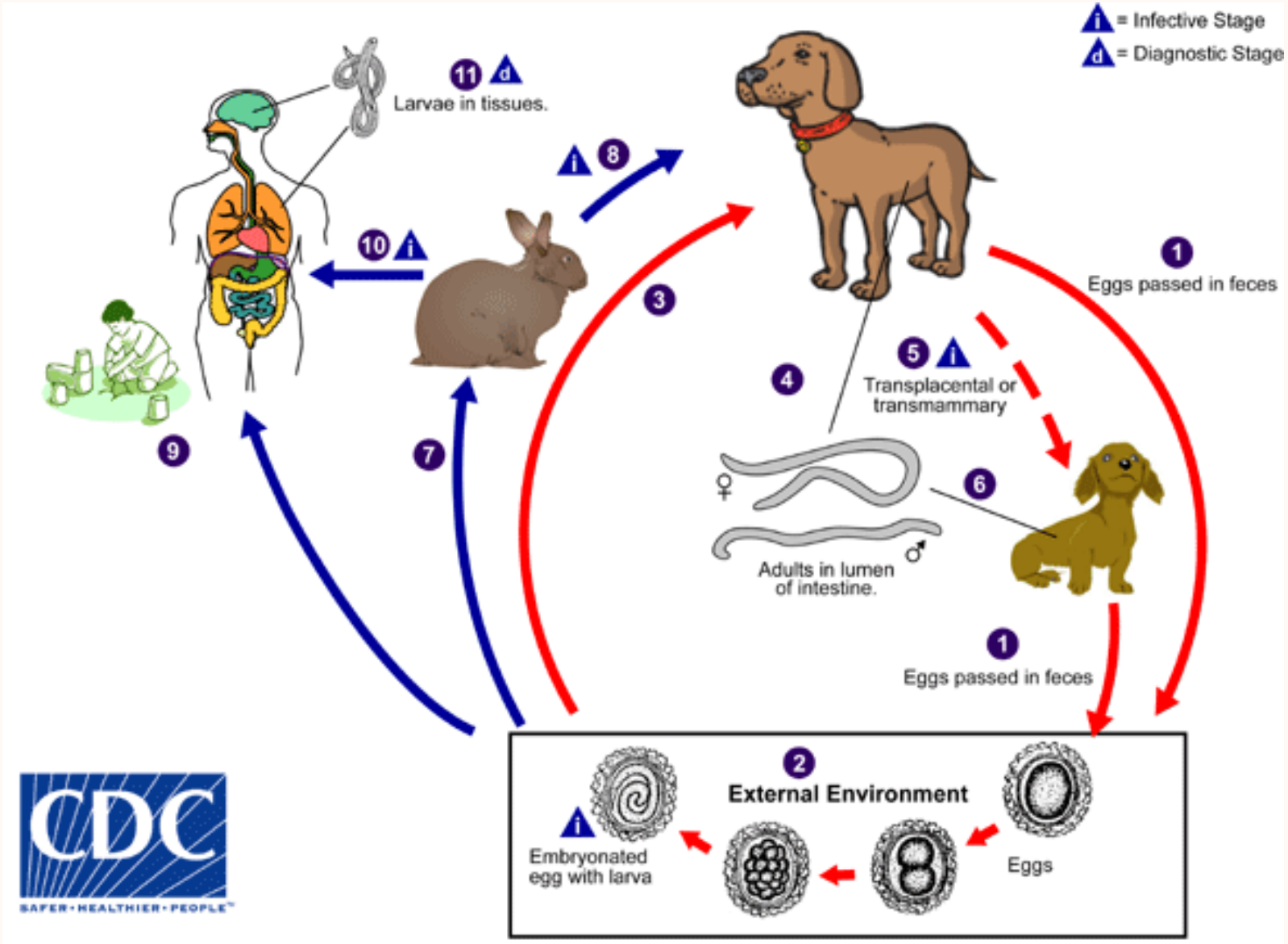
Adult worms live in the small intestine of dogs. Almost all puppies are infected at, or soon after, birth.

The soil is commonly contaminated with the eggs and humans typically ingest the eggs via oral contact with contaminated hands.

Once introduced into the human intestine, the eggs decorticate, releasing the larvae (0.5 mm x 0.02 mm).

The larvae penetrate the bowel wall and migrate through vessels to the muscles, liver, and lung and sometimes to the eye and brain.

Toxocariasis



Toxocariasis

Disease severity depends not only on the number of larvae ingested but also on the degree of the allergic reaction.

The most common characteristic is chronic **eosinophilia, without any symptoms**.

Other typical findings follow according to the involved organs.

The **two main clinical presentations** of toxocariasis are visceral larva migrans (**VLM**) and ocular larva migrans (**OLM**).

Visceral larva migrans

VLM is caused by the migration of larvae through the internal organs of humans and the resulting inflammatory reaction.

Syndrome is characterized by **bouts of fever, coughing, wheezing, lymphadenitis and hepatomegaly**.

Fatigue, anorexia, weight loss, **pneumonia**, fever, cough, bronchospasm, **abdominal pain**, headaches, **rashes**, and, occasionally, **seizures** may develop.

Severe cases can rarely lead to myocarditis or respiratory failure.

Ocular larva migrans

OLM is caused by migration of larva into the posterior segment of the eye.

Patients may present with **decreased vision, red eye,** or **leukokoria** (white appearance of the pupil).

Granulomas and chorioretinitis can be observed in the retina, especially at the macula. Unilateral visual loss, retinal fibrosis, retinoblastoma, and retinal detachment occur.

Tends to occur in older children and young adults

Toxocariasis - diagnosis

Diagnosis is based on **clinical** and serologic **findings**.

Laboratory: leukocytosis, anemia, eosinophilia, hypergammaglobulinemia IgM, and positive *Toxocara* titers.

Imaging studies in toxocariasis (VLM and OLM) depend on the body location of the disease.

Stool examination for *Toxocara* larvae is not a helpful diagnostic test for toxocariasis.

Toxocariasis - treatment

Most patients recover **without** therapy.

Treatment with **anthelmintic** agents is indicated **for** severe **complications**, such as for patients with cerebral, cardiac, ocular and/or pulmonary involvement.

Anthelmintic treatment can induce an increased systemic inflammatory reaction, as such corticosteroids are sometimes used in conjunction with or without anthelminthic therapy.

Toxocariasis - treatment

Drug	Dose and duration
Albendazole (Zentel)	400 mg by mouth twice a day for five days (both adult and pediatric dosage)
Mebendazole (Vermox)	100-200 mg by mouth twice a day for five days (both adult and pediatric dosage)

Quiz

- http://www.prognosisapp.com/cases/play/M140_en/Muddy
- http://www.prognosisapp.com/cases/play/M54_en/Skin-Deep
- http://www.prognosisapp.com/cases/play/M250_en/Invaded
- http://www.prognosisapp.com/cases/play/M133_en/Feathers
- http://www.prognosisapp.com/cases/play/M147_en/Zoonotic
- http://www.prognosisapp.com/cases/play/M143_en/Infested