1. Rabies:

- Horses susceptible to wildlife rabies strains in the US (raccoon, skunk, fox and bat strains).

- About 50 cases per year in the USA since 1981 (52 cases in 2000, 51 cases in 2001, 58 cases in 2002).

- Present more frequently with furious form, usually do not attack, but can be very aggressive, dies within a few days. Large number of persons exposed to a rabid horse.

- Vaccination should be performed everywhere where rabies is endemic (both from terrestrial reservoirs or bat reservoir). Horses must be 3 months or older at primary vaccination and must receive an annual booster.
1. **Vesicular stomatitis**: (Rhabdovirus, Vesiculovirus)
   - Mainly horses and cattle susceptible to serotypes Indiana and New Jersey (can also infect pigs, sheep, wildlife). **Found only in the Americas.** VS occurs during warm months in the Southwest of the USA, particularly along riverways and in valleys. VS outbreak in SW USA from May 1998 through January 1999. Contamination by transcutaneous or transmucosal route. Arthropod transmission (*Phlebotomus, Aedes*, etc.)
   - **In horses**: short incubation (2-4 days) with short febrile illness with excessive salivation and blister-like lesions in the mouth and on the dental pad, tongue, lips, nostrils, hooves (lameness). Recover in 1-2 weeks.
   - **In humans**: Rare in humans, mainly found in laboratory personnel. Incubation 1-2 days, flu-like symptoms (fever, headache, myalgia) lasting for a few days.
   - **Prevention**: Wear protective clothing and gloves.

---

**Western Equine Encephalitis**
- First reported in 1847. Large epidemics in California in the 1930s-1950s, Letality of up to 50% in horses. **Human lethality of 3-4%**. **Children, especially < 1 year old, are affected more severely than adults and may be left with permanent sequelae (5 to 30% of young patients).**

Found in North, Central and South America. Most cases in the USA are west of Rocky Mountains & West of the Mississippi.

**Primarily spread by Culex tarsalis**
- Other species of mosquito (e.g. Aedes) and occasionally small wild mammals have been implicated.
- Cycles of wild birds and mosquito interaction allow the virus to remain endemic
- Common in farming areas & irrigated fields.
**VIRAL ZOONOSES**

**Life cycle of WEE**

![Diagram of the life cycle of WEE](image)

### Equine Eastern Encephalitis

- First reported in USA in Massachusetts in horses in 1831. Highly pathogenic to horses. Causes equine epizootics in Eastern USA. **Sequelae and case fatality rate of 30% in EEE cases in humans.**

EEE virus occurs in natural cycles involving birds and *Culiseta melanura*, in swampy areas nearly every year during the warm months. Where or how it survives in the winter is unknown. May be introduced by migratory birds in the spring or it may remain dormant in some yet undiscovered part of its life cycle. With the onset of spring, the virus reappears in the birds (native bird species do not seem to be affected by the virus) and mosquitoes of the swamp.
Clinical Signs in Humans:

**WEE**
- Most asymptomatic
- Mild flu like symptoms, fever, headache, anorexia, frank encephalitis, coma, death
- Mortality rate 3%
- Mild to Serious neurologic deficits in survivors

**EEE**
- More serious than WEE in man.
- 1/3 of all people with clinical encephalitis caused by EEE will die from the disease and many will suffer permanent brain damage requiring permanent institutional care.

Clinical Signs in Horses: WEE, EEE, VEE

- Fever, depression, drowsiness
- Paralysis, circling, dysphagia, photo-sensibility
- Mild to Serious neurologic deficits in survivors
- Can recover but may have brain damage

Mortality Rates
- WEE: 20-40%; EEE: 50-90%; VEE: 50-80%
**EQUINE ENCEPHALITIDES**

*Family: Togaviridae genus: Alphaviruses*  
**Venezuelan Equine Encephalitis:**
- **Equine mortality rates:** 19-83% (only 4-14% in humans) for epidemic variants (serogroups IAB and IC). Enzootic serogroups (ID-IF and II-IV) are generally avirulent for horses.

- **Epizootic strains of VEE virus can infect and be transmitted by a large number of mosquito species.** The natural reservoir host for the epizootic strains is not known. Enzootic strains of VEE virus are maintained in cycles involving forest dwelling rodents and mosquito vectors, mainly *Culex (Melanoconion)* species.

- **A large epizootic (> 200,000 dead horses and several thousand human infections) in South America in 1969 reached Texas in 1971. Controlled by vaccination (live attenuated vaccine). Fall 1995: VEE epidemic in Venezuela and Colombia with an estimated 90,000 human infections.**

**EQUINE ENCEPHALITIDES**

*Family: Flaviviridae*  
**St Louis encephalitis:**
- Mainly seen in humans. Illness ranges in severity from a simple febrile headache to meningoencephalitis, with an overall case-fatality ratio of 5-15%.

- Most common arboviral zoonosis in USA.
- The disease is generally milder in children than in adults, but in those children who do have disease, there is a high rate of encephalitis. The elderly are at highest risk for severe disease and death.

- Birds reservoir, *Culex* spp. mosquitoes vectors. **Causes encephalitis in horses only experimentally.**
Equine Encephalitides

Family: **Flaviviridae**

**West Nile virus:**
- Appeared in 1999 in USA.
- Now present in almost all the continental USA and several Canadian Provinces.

Epidemic in horses with >15,000 cases in 2002 (1/3 horses died). More than 4,000 human cases of West Nile encephalitis in 2002 and >9,300 cases in 2003.

WNV is transmitted principally by *Culex* species mosquitoes, but also can be transmitted by *Aedes, Anopheles*, and other species of mosquitoes.

West Nile re-emerging in Southern France in 2000 after 35 years
### WEST NILE VIRUS OUTBREAKS IN HORSES

<table>
<thead>
<tr>
<th>YEAR</th>
<th>LOCATION</th>
<th># CASES</th>
<th># DEATHS</th>
</tr>
</thead>
<tbody>
<tr>
<td>1960s</td>
<td>Egypt</td>
<td>?</td>
<td>?</td>
</tr>
<tr>
<td>1962</td>
<td>France (Camargue)</td>
<td>about 80</td>
<td>10%</td>
</tr>
<tr>
<td>1996</td>
<td>Morocco</td>
<td>94</td>
<td>42 (44.7%)</td>
</tr>
<tr>
<td>1998</td>
<td>Italy</td>
<td>14</td>
<td>6 (42.8%)</td>
</tr>
<tr>
<td>1999</td>
<td>USA (2 States)</td>
<td>25</td>
<td>9 (36%)</td>
</tr>
<tr>
<td>2000</td>
<td>France</td>
<td>76</td>
<td>21 (27.6%)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>(8.5%; 432/5,107 sero+)</td>
</tr>
</tbody>
</table>

#### Phylogenetic analysis

1662 nt from the PrM, M, E gene with KUN primers

**KUNJIN** (D00246)

**Human** **RUSSIA 1999** (AF317293)

**Cx. pipiens** **ROMANIA 1997** (AF130362)

- **WN 0304 ISRAEL 2000**
- **WN 0303 ISRAEL 2000**

**NEW YORK** (AF194117)

- **Gull ISRAEL 1999**
- **Horse NY 1999** (AF260967)
- **Cx. pipiens Conn. 1999** (AF286518)
- **Human NY 1999** (AF228541)
- **Flamingo NY 1999** (AF196835)

- **WN 0043 ISRAEL 2000**
- **WN 0247 ISRAEL 2000**
WEST NILE VIRUS OUTBREAKS IN HORSES:
Beginning of an epidemic?

<table>
<thead>
<tr>
<th>YEAR</th>
<th>LOCATION</th>
<th># CASES</th>
<th># DEATHS</th>
</tr>
</thead>
<tbody>
<tr>
<td>1999</td>
<td>USA (1 State)</td>
<td>25</td>
<td>9 (36%)</td>
</tr>
<tr>
<td>2000</td>
<td>USA (7 States)</td>
<td>60</td>
<td>23 (38%)</td>
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<tr>
<td>2001</td>
<td>USA (20 States)</td>
<td>752</td>
<td>156/470 (33%)</td>
</tr>
<tr>
<td>2002</td>
<td>USA (40 states)</td>
<td>14,717</td>
<td>1/3 to 1/4</td>
</tr>
<tr>
<td>2003</td>
<td>USA (44 states)</td>
<td>4,636</td>
<td></td>
</tr>
</tbody>
</table>

* Confirmed by USDA
SYMPTOMS ASSOCIATED WITH WEST NILE VIRUS INFECTION IN HORSES

- Usually, asymptomatic. **Clinical signs** (neurological) in about 10% of all infections (Camargue, 1962), to about 20% of all infections (Tuscany, 1998) and to less than 40% of all infections in Long Island, New York, (1999).

- Neurological disease in Horses: Posterior ataxia, proprioception deficits, altered behaviour. Severe cases: Paralysis of the hind legs, recumbency, terminal convulsion and death.
EQUINE ENCEPHALITIDES

**Bunyaviruses:**

**Northern USA/Southern Canada:**
Snowshoe hare virus: rabbits- *Culiseta/Aedes* mosquitoes. MEM in humans, not in horses, but seropositive horses.

**Upper MID WEST-Mid-Atlantic States, Southern USA:**
La Crosse virus: reservoir: squirrels, vector: *Aedes triseriatus*.

**WESTERN USA:**
Cache valley Virus: (transmitted by *Culicoides* and mosquitoes). MEM in horses.
Lockern virus (*Culicoides variipennis*), Western USA. MEM in humans, not in horses, but seropositive horses.
Main Drain virus: Hares, rodents, vector: *Culicoides variipennis*. Encephalitis in a horse in California.

---

**VIRAL ZOONOSES**

**PREVENTION of E. M. E. M.**

- Vaccinate horses annually (WEE, EEE, WNV). Vaccine for VEE in central/South America.
- Avoid exposure to mosquito infested areas.
- Avoid outdoor areas during twilight.
- Use mosquito repellants on skin and clothing.
  - especially those preparations with DEET (N<Diethyl-meta-toluamide)
- Mosquito control, aerial spraying, water drainage
VIRAL ZOONOSES

Influenza viruses (Orthomyxoviruses):

• Horses infected by two different subtypes of influenza viruses: H7N7 (A equi 1) and H3N8 (A equi 2). (The equine influenza-A-2 virus is thought to have originated from an avian (duck) influenza virus).

• Highly severe and contagious disease in horses, sequelae: cardiopathy in race horses. Specific regulations on horse tracks, vaccination required.

• No known direct infection from horses to humans.

• Experimentally, mild flu-like infection in volunteers with A equi 2

• Horses exposed to human Hong Kong H3N2 virus, developed a mild febrile illness.

HENDRA VIRUS (Paramyxoviridae)
HENDRA VIRUS (Paramyxoviridae)

• First described in 1994 in Brisbane (Hendra facility), Australia
• 2 Outbreaks within one month of each other
• In one case 21 horses were infected and 14 died or were euthanized after developing severe respiratory signs.
• 3rd outbreak in 1999 in a 9 y.o. TB mare (near Cairns)
• 3 human cases of disease (trainer, 49 yr-old; stable hand, 40 yr-old; 30 yr old farmer), 2 deaths

Hendra virus, Australia, 1994

Source: C. Brown, 2002
HENDRA VIRUS (Paramyxoviridae)

**CLINICAL SIGNS:**

**HORSES:** Acute respiratory syndrome followed by death in 1-3 days

• Signs include depression, anorexia, fever, tachypnoea, nasal discharge

• Prior to death some horses exhibit head pressing and ataxia with frothy nasal discharge exuded at time of death

**HUMANS:**

**Acute form:** Flu-like symptoms which may progress to pneumonitis, respiratory failure, renal failure, arterial thrombosis and cardiac arrest

**Chronic form:** meningoencephalitis
HENDRA VIRUS (Paramyxoviridae)

Epidemiology:

- 5000 sera from 46 species all negative except in pteropid bats.
- Four species of fruit eating bats (Pteropus sp. = flying foxes: Grey-headed flying fox, black flying fox, little red flying fox and spectacled flying fox) found to have Hendra virus.
- 25% of bats surveyed seropositive
- Horses can get affected by eating material contaminated with the virus e.g. bat urine
- Transmission can also occur by exposure to cat urine

3. Borna Disease (BD)

First described > 200 years ago in southern Germany. Epidemic in horses in 1885 in the town of Borna (Saxony, Germany). Bornaviridae (enveloped RNA). Viral etiology proven by transmission from infected horses to experimental animals (early 1900s). Causes fatal neurologic disease of horses characterized by a disseminated non-purulent meningoencephalomyelitis (MEM). Natural infections in horses, ruminants, rabbits, cats, ostriches. Several epidemics in horses

Horses: "Sad Horse" Disease. Symptomatic infection manifests itself with agitated and aggressive changes, often progressing to inanition and death in several weeks. BDV infection can develop neurologic, cognitive and behavioral changes including hyperactivity, somnolence, apathy, anorexia and depression. Virus transmitted by salival, nasal or conjunctival secretions. Incubation of 4 weeks minimum. Initial phase with non-specific symptoms, then neurologic signs, paralysis & death in 1-3 weeks.

Possible Zoonosis: BDV antibodies found in humans with psychiatric disorders. Virus isolated from CSF and brain in humans.
**BACTERIAL ZOONOSES**

1. **Salmonellosis**

_Salmonella typhimurium_ is the most common serotype isolated from horses. Some other serotypes (i.e. _S. saintpaul_) also reported. **Salmonellosis in horses causes diarrhea, abortion, septicemia and death, especially in foals.** Healthy carriers that can be intermittent shedders or develop symptoms under stress. Several nosocomial outbreaks reported in veterinary teaching hospitals in the USA (especially among horses having surgery for colics). Direct contamination of people from sick horses, including children reported in the USA, France. In humans, symptoms include fever, vomiting, diarrhea, abdominal cramps and dehydration. Identification of the _Salmonella_ type and lysotype from fecal samples is essential to determine the origin of the outbreak. Antibiotic susceptibility to be also tested as _S. typhimurium_ is often multiresistant. Control and prevention by thorough disinfection.

2. **Glanders**

Caused by _Burkholderia (Pseudomonas) mallei_. Once a worldwide disease of equids and humans. It is now very rare and limited to the middle East (Turkey, Iraq), Asia (India, Mongolia), South and Central America. Last human case in the USA was in the 1945. One laboratory worker accidentally contaminated in 2000. Infection usually occur by direct contact. Human-to-human transmission: sexual transmission and taking care of sick patients.
BACTERIAL ZOONOSES

2. Glanders

Horses:

The disease is predominantly chronic in horses, usually acute in donkeys and mules. In the respiratory form, ulcerations of the mucous membrane of the upper respiratory tract or pulmonary nodules are characteristic of the disease. In the cutaneous form, nodules, ulcers with oily exsudate and swollen lymph nodes and vessels are major signs.

*B. mallei* is susceptible to most antibiotics. Sulfadiazine has been found to be an effective in experimental animals and in humans.

Potential biological weapon.

---

BACTERIAL ZOONOSES

2. Glanders caused by *Burkholderia (Pseudomonas) mallei*.

Localized symptoms:

**Pus-forming cutaneous infection**
- nodules
- ulcers with oily exsudate
- swollen lymph node and vessels

**Septicemia** – fatal within 7-10 days

**Chronic suppurative infection**
multiple abscesses in:
- arm & leg muscles
- spleen or liver
BACTERIAL ZOONOSES

2. **Glanders** caused by *Burkholderia (Pseudomonas) mallei*.

Symptoms in Horses: Localized symptoms:

- **Pulmonary infection**
  - Localize in: - lungs
    - mucosa (nose, larynx & trachea)
  - pneumonia or bronchopneumonia
  - pulmonary abscesses
  - pleural effusion

**General symptoms:**
- fever, muscle aches, headache,
- chest pain, muscle tightness,
- excessive tearing of eyes,
- light sensitivity, diarrhea

Treatment: Sulfadiazine, Tetracyclines, Ciprofloxacin, Streptomycin

Pyogenic granulomatus pneumonia in horse

Glanders in a horse
Morve du cheval

Cutaneous abscesses

Colloque ISV, 15 mai 2003
2. **Glanders**

*caused by* *Burkholderia (Pseudomonas) mallei*.

**Symptoms in Humans:**
- Localized symptoms:
  - Pulmonary infection (localized in)
    - lungs
    - mucosa (nose, larynx & trachea)
    - pneumonia or bronchopneumonia
    - pulmonary abscesses
  - pleural effusion
- General symptoms:
  - fever, muscle aches, headache,
  - chest pain, muscle tightness,
  - excessive tearing of eyes,
  - light sensitivity, diarrhea

**Treatment:** Sulfadiazine, Tetracyclines, Ciprofloxacin, Streptomycin

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3. **Melioidosis**

*caused by* *Burkholderia (Pseudomonas) pseudomallei*. *Saprophytic organism in certain soils and waters*, especially in South East Asia, northern Australia and Africa.

Various animals species can be infected, including sheep, goats, horses, swine, monkeys and humans.

Symptoms range from inapparent infection to severe pneumonia and septicemia with high mortality. **More severe in immunocompromised persons, alcoholics, drug addicts.** Lung abscess, pleural effusion, liver and spleen abscesses.

**Outbreak in horses in France in the late 1970s early 1980s with respiratory and digestive symptoms.** Several horsemen seroconverted. No direct contamination from horses to humans. *B. pseudomallei* usually transmitted by cutaneous or respiratory route.

**Treatment:** Trimetoprim-sulfamethoxazole or doxyxcylcine.
4. *Rhodococcus equi*

Originally isolated by Magnusson in 1923 from granulomatous lung infections in young horses, *Rhodococcus (Corynebacterium) equi* remains an important pathogen of foals.

*R. equi* is readily found in soil, especially where domesticated livestock graze. The stool of horses and other animals is the source of soil contamination.

Infection in humans derives from environmental exposure, and the organism may be ubiquitous in soil. While early cases occurred mostly in persons with a history of contact with horses, only 20% to 30% of recent cases can be traced to such contact.  


4. *Rhodococcus equi*

*R. equi* is a rare opportunistic pathogen found in severely compromised patients, and most commonly in recent years, in human immunodeficiency virus (HIV)-infected persons (7 cases between 1977-1986; 67 cases between 1987-1996). Early cases, most in patients receiving immuno suppressant therapy, were more likely to be successfully treated with antimicrobial agents than cases in AIDS patients (1987-1996: 93 cases total, including 34 deaths).

Most often, patients have a slowly progressive granulomatous pneumonia, with lobar infiltrates, frequently developing to cavitating lesions visible on chest x-ray. Other sites of infection include abscesses of the central nervous system, pelvis, and subcutaneous tissue, and lymphadenitis. Cases of lung infection caused by inhalation and cutaneous lesions caused by wound contamination (almost the only *R. equi* infections reported in healthy persons, frequently children) have been documented.
4. **Rhodococcus equi**

Careful and repeated culture and susceptibility testing during treatment is required to discover acquired resistance, in a manner similar to the treatment of mycobacterial infection.

A proposed regimen involves parenteral glycopeptide plus imipenem for at least 3 weeks, followed by an oral combination of rifampin, plus either macrolides or tetracycline.

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5. **Anthrax**

caused by *Bacillus anthracis*. Common bacterium in many parts of the world. Various species infected, including horses. Recent outbreaks in the USA include: outbreak in cattle and horses in Minnesota in June-July 2000; cattle, horses, and bison in North Dakota in August 2000.

**Horses are very susceptible to anthrax and usually develop a septicemic form with high fever, colics, enteritis with hemorrhages, depression and death in 2-4 days.** Anthrax due to insect bite introduction (mechanical transmission) is characterized by localized hot, painful, edematous, and subcutaneous swellings at the bite location that spread to the throat, lower neck, floor of the thorax, abdomen, prepuce, and mammary glands. **Vaccination of horses is highly recommended in endemic areas.** Horses very susceptible to vaccines (need to be monitored and receive antibiotics if have fever). Treatment of choice: penicillin
5. **Anthrax**  
In humans, most common form is cutaneous anthrax. After 2-5 days incubation, a small papule develops and evolves to a vesicle then to an ulcer with a black eschar. Most severe form is after aerosolization of spores and development of pulmonary anthrax. No direct contamination from horses to humans.

Treatment: penicillin, ciprofloxacin.

6. **Brucellosis** caused by *Brucella abortus, B. suis (B. melitensis).*  
Horses are accidental hosts and usually epidemiological cul-de-sac. Infection occurs when horses are sharing pasture or housed with infected cattle or swine. The disease usually manifest itself by a fistulous bursitis (withers). Horses may not show signs for as long as two years post-exposure. May present with late abortion, arthritis, orchitis and suppurative bursitis (fistulous withers). Humans can get infected by exposure to open infected lesions from horses.  
**Disease in humans:** Incubation period: 1-2 months  
Intermittent ‘undulating’ fever  
Headaches, chills, depression, profound weakness  
Arthralgia, myalgia, Weight loss  
Orchitis/epididymitis in men  
**Treatment:** rifampin and doxycycline for at least 6 weeks
7. **Leptospirosis**

caused by *Leptospira* spp.

Horses can be infected by different serovars and usually develop inapparent infection (seroconversion). Can cause fever, abortion in mares, septicemia in foals. Recurrent uveitis (moon blindness) is one of the major sequelae of leptospirosis in horses.

Direct infection of humans from horses has not been proven.

Treatment: Penicillin, Doxycycline, ciprofloxacin.

---

8. **Tuberculosis**

caused by *Mycobacterium bovis*.

Tuberculosis is a very rare disease in horses. They can be infected by *M. bovis* in countries where the disease is endemic and when horses are housed with infected cattle.

could be potentially transmitted to humans.
BACTERIAL ZOONOSES

9. Pasteurellosis and other bite-transmitted zoonoses

Horse bites are often very severe and infection by various infectious organisms such as *Pasteurella multocida* can occur. *Actinobacillus lignieresii* and *A. equuli*-like bacteria were isolated from an infected horse bite in a 22 year old stable foreman and *A. suis* from a 35 year old man who had been attacked by a horse.

10. *Streptococcus equi subsp. zooepidemicus*

*S. equi zooepidemicus* is a commensal of the skin and upper respiratory mucosa of horses. Most common cause of wound infections in horses and causes respiratory tract infections of foals and young horses (strangles). A few report in the literature of people exposed to horses and developed a severe respiratory infection caused by *S. equi zooepidemicus*. One case of meningitis also reported.

PARASITIC ZOONOSES

1. **Trichinosis**

Trichinosis is a food-borne zoonosis. **Large outbreaks with several thousand human cases have occurred in Europe (Since 1975, human trichinellosis has occurred in France (2296 persons in eight outbreaks) and Italy (1030 persons in six outbreaks), following consumption of raw or undercooked horse meat imported from the USA, Mexico, Canada and Eastern Europe.**

**Clinical signs:** Incubation is a few days to weeks after infection. GI signs, nausea, fatigue, and fever. Headaches, cough, eye swelling, aching joints, muscle pains, itchy skin, diarrhea, or constipation follow the first symptoms. For mild to moderate infections, most symptoms subside within a few months. With heavy infections, neurologic symptoms, abortion, cardiac symptoms, and death.

**Diagnosis**

Horse serology not adequate surveillance, antibodies last < 6 months. Cysts mainly found in tongue, diaphragm used for meat inspection

**Treatment**

Albendazole works on intestinal form of parasite but not muscular form. Supportive care.
PARASITIC ZOONOSES

1. **Hydatidosis**
   In Great Britain, two strains of *Echinococcus granulosus equinus* occur, including an equine strain whose life cycle involves horses and dogs. The equine strain infectivity for man. As consumption of horse meat is not traditional in UK, risk of human contamination is very limited.

FUNGAL ZOONOSES

1. ** Dermatophyosis**
   In horses, several dermatophytes cause ringworm: *Trichophyton equinum*, *T. mentagrophytes* and *Microsporum equinum*. *M. Canis* and *M. gypseum* also isolated from horses. Lesions are round, hairless. The skin is thickened and covered with scales.
   - **Diagnosis:** Round, hairless, scaly skin lesions, +/- UV
   - **Treatment:** Griseofulvin or topical fungicides
   - **Prevention:** Disinfection or disposal of grooming tools and bedding from infected animals
2. Dermatophilosis  
Etiologic agent = **Dermatophilus congolensis**

This organism develops characteristic septate filaments with parallel rows of coccoid cells that form motile flagellated zoospores. Infections occur most commonly in horses, cattle and small ruminants, with transmission between animals occurring by release of zoospores from the lesions when they get wet. Transmission may be either direct or via mechanical insect vectors. The skin lesions appear as exudative, crusted areas in which the hairs are cemented together into characteristic "paint-brush" clumps.

1. Dermatophilosis

*Dermatophilus congolensis* is transmitted to humans by direct contact with lesions on animals. Humans develop an exudative dermatitis similar to that seen in animals.

**Treatment**

a. *Remove scabs*: Bath, groom, clip hair  
b. Topicals Bath and soak: **povidone or chlorhexidine shampoos for horses**;  
c. Parenteral antibiotics for generalized disease: **Penicillin, Ceftiofur, TMS**
### FUNGAL ZOONOSES

**3. Sporotrichosis**
- **Caused by** *Sporothrix schenckii*
- Dimorphic fungus, Ubiquitous in soil and vegetation
- Transmission: Direct contact with wounds, soil and plants
  - Cats and other animals (including horses). Inhalation (rare)
- Symptoms: Nodules in skin, nodules may ulcerate, firm and cordlike lymph nodes, may disseminate causing arthritis, meningitis, pneumonitis and other visceral infections; occurs rarely;
- Diagnosis: Culture from unopened lesions
- Treatment: Potassium iodide, *itraconazole*, amphotericin B until lesions resolve
- Prevention: Strict hygiene when handling infected animals. Disinfection with chemicals and hot water

###Protozoal Zoonoses

**4. Cryptosporidiosis/Giardiasis**
- *Cryptosporidium parvum*
- *Giardia duodenalis*
- Fecal-oral transmission
- Waterborne
- Transient diarrhea
- Human, animal genotypes
- Diagnostic: Fecal flotation, AF, DFA, EIA, PCR
- Treatment: Supportive care for *Cryptosporidium*, plus metronidazole for *Giardia*
- Prevention: Proper sanitation
EQUINE ZOONOSES

THE END

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