Encephalitozoon cuniculi

*Encephalitozoon cuniculi* is a single-cell, microsporidial organism. Microsporidia are a diverse group of organisms and more than 1200 species have been identified. They parasitise a range of vertebrate and invertebrate hosts. Microsporidia lack mitochondria and rely on a host cell for energy. They produce large numbers of spores that contain a coiled polar tube. Classification of microsporidia within the animal kingdom has proved difficult. Many early papers describe microsporidia as protozoa but papers that are more recent classify them as atypical fungi. Chitin is present in the cell wall.

There has been an upsurge of interest in microsporidia in recent years because several species can cause disease in humans, especially in people that are immunocompromised by HIV infection or anti-rejection drugs following organ transplantation. Medication to treat cancer can also be immunosuppressive. *Encephalitozoon* is only one genus of microsporidia and it contains several species, including *Encephalitozoon cuniculi*. The most common microsporidial infections in humans are due to *Encephalitozoon bieneusi* and *Encephalitozoon intestinalis*. Both species have been found worldwide, mainly in HIV-infected patients with chronic diarrhoea but also in immunocompetent persons with acute, self-limited diarrhoea. Disease due to *Encephalitozoon cuniculi* and *Encephalitozoon hellem* are rare but have been diagnosed in humans.

**Strains of Encephalitozoon cuniculi**

At least three strains of *E. cuniculi* have been identified. There is no morphological difference between the strains. They are differentiated by differences in their DNA. Serological testing does not differentiate between them. It is not known how species specific the strains are or whether they can transfer freely between different hosts.

**Strain I** is associated with rabbits.

**Strain II** is associated with rodents although it has been isolated from a gyrfalcon

**Strain III** has been reported in dogs, tamarin colonies from zoos, lemmings, swine, birds and humans.

Others strains have been identified in a bearded dragon and a Gyr falcon.

**Spore resistance**

Spores of *E. cuniculi* are highly resistant in the environment and can survive several months under humid conditions. Direct contact with infected animals or humans is not required for survival and water may be contaminated by spores. A study of drinking water in Spain found contamination by *E. cuniculi* amongst other microsporidia. Common disinfectants are effective in killing spores.

**Encephalitozoon cuniculi in other species**

Although rabbits are the species that are most likely to show clinical signs of encephalitozoonosis, there are reports of illness in horses, foxes, dogs, rats, muskrats, hamsters, groundshrews, pigs, sheep, cats, mice, monkeys, lemmings, bearded dragons, cats and a snow leopard.

Myocarditis, encephalitis, cataracts, placentitis and abortion are among diseases that have been linked with *E. cuniculi* infection in these species. In guinea pigs, encephalitis and nephritis have been reported and, in dogs, encephalitozoonosis has been associated with fading puppy syndrome.
INFECTIONIOUS DISEASES: *E. cuniculi*, myxomatosis, RHD and parasites

**Zoonotic potential of *E. cuniculi***

In immunosuppressed people, especially those with HIV, microsporidiosis can be a real problem and *E. cuniculi* is just one of the microsporidia that can cause disease. Apart from a single case of a brain abscess, encephalitozoonosis in immunocompetent people has not been described. There are no human cases of encephalitozoonosis in which an infected rabbit has been confirmed as the source of infection. Conversely, laboratory rabbits have been infected with *E. cuniculi* spores collected from human patients. Even though many articles cite *E. cuniculi* as zoonotic disease that could spread from rabbits to humans, the risk of an immunocompetent human catching the disease from a pet rabbit is negligible.

**Life-cycle of *E. cuniculi***

*E. cuniculi* spores are tiny, oval structures that measure approximately 2.5 x 1.5 µm. Within the spore, there is a coiled polar filament, which can be extruded and anchored to a neighbouring cell. The trigger for activation of the spores is not clear. The changes in pH of intestinal contents is one possibility. After the spore is activated and has extruded its polar tube to penetrate a neighbouring cell, sporoplasm and the spore nucleus are transferred through the polar tube into a vacuole that forms within the host cell. Multiplication of the parasite takes place within the vacuole until it becomes so distended that it forms a bubble like swelling on the host cell, which eventually ruptures to release the spores. These can invade neighbouring cells or if kidney tubule cells are the host cells, cell rupture releases spores into the urine. Cell rupture is associated with an inflammatory response and the development of granulomatous lesions.

Oral ingestion of spores is the usual route of infection. The spores invade the intestinal epithelium and gut associated lymphoid tissue before spreading through the blood stream, either as free spores or in infected monocytes, to other organs including the kidney, brain, liver and heart. The most common sites are the brain and kidney and in infected animals, histologically, spores may be seen in these tissues, usually in association with granulomatous inflammation. Lesions in the brain are most commonly seen in the cerebrum.

**Spread of infection between rabbits**

Spread of *E. cuniculi* infection in rabbits may be vertical or horizontal. Horizontal spread of infection is caused by ingestion of food contaminated with infected urine. The source of infection may be contaminated enclosures or ingestion of food that has been contaminated with a companion’s urine. Vertical spread occurs *in utero*, when spores in an infected dam pass into the lens of a developing embryo.

**Stages of the disease**

1. An acute phase of diseases occurs shortly after infection when the parasite is disseminated around the body. The lung, kidney and liver are the organs that are most affected and, rarely, acute clinical signs may occur.
2. Chronic disease results in granulomatous inflammation that may interfere with organ function. The kidney, brain and heart may be affected. Significant organ damage may take place. The organism may or may not be present in damaged tissue.
3. Latent infection when inactive spores are present in the body. The animal may be asymptomatic although stressful events or disease can trigger an acute episode.
Neurological disease and encephalitozoonosis in rabbits

Neurological disorders are common in pet rabbits, especially vestibular disease and ataxia, and these are often attributed to encephalitozoonosis, although the diagnosis is hard to prove. Concurrent disease and the presence of granulomatous brain lesions in asymptomatic rabbits confuse the diagnosis.

Vestibular disease is the neurological sign that is most commonly associated with *E. cuniculi* and is often attributed acute cell rupture and inflammation. Vestibular disease is usually sudden in onset and may follow a stressful event in the rabbit’s life. Many cases completely or partially resolve with time, whatever the treatment.

Studies have suggested that cerebral disease may be more common than vestibular disease but is more likely to be overlooked because the clinical signs are more subtle. Altered mental status is associated with cerebral disease and is difficult to assess in rabbits. Ataxia, with or without bladder atony and urinary incontinence, is another neurological sign that appears to be linked with encephalitozoon infection. Anecdotally, treatment for encephalitozoonosis often brings about a temporary, or occasionally a permanent, improvement in these cases. However, histopathological studies rarely confirm this link, probably because the spinal cord is seldom examined thoroughly. Trauma and degenerative disc disease are major differential diagnoses for the ataxia and the list is even longer for urinary incontinence.

Renal disease

Renal disease is a feature of *E. cuniculi* infection and results in characteristic scarring of the kidneys. This is a common post mortem finding, and the easiest and cheapest way of confirming infection with *E. cuniculi* although many affected rabbits have shown no obvious signs of encephalitozoonosis or renal disease during life. Acute renal failure due to encephalitozoonosis appears to be rare but chronic renal failure is a common and is associated with nephrolithiasis. Affected rabbits are almost invariably seropositive for *E. cuniculi* although spores are rarely found, perhaps because the clinical signs are due to longstanding tissue damage rather than acute inflammation.

Myocarditis

Myocardial fibrosis is a frequent post-mortem finding in both rabbits with no sign of heart failure and in those with cardiac signs or have died unexpectedly. It is easy to speculate that *E. cuniculi* could be the cause of these lesions. Myocardial lesions are often inflammatory and focal.

Ocular lesions

Cataracts, hypopyon or uveitis can be caused by *E. cuniculi* and rabbits with these signs are invariably seropositive. Ocular lesions are reputed to be due to vertical transmission when the lens is predilection site for the parasite in the foetus. After birth, the lens ruptures, which initiates an inflammatory response. At this point, *E. cuniculi* organisms or their DNA may be found in the lens material, which confirms the diagnosis. This contrasts with renal disease, presumably because the clinical signs are associated with an acute inflammatory response before the parasite has been eliminated.
**INFECTIOUS DISEASES: *E. cuniculi*, myxomatosis, RHD and parasites**

**Diagnosis of encephalitozoonosis**
Definitive diagnosis of *E. cuniculi* as the cause of disease in the live rabbit is difficult. There are many differential diagnoses and even after death, the diagnosis is often presumptive because is based on the presence of characteristic inflammatory lesions rather than the presence of the organism. There are no diagnostic imaging techniques that can provide the diagnosis. Cerebrospinal fluid analysis may or may not be helpful. Serology can show if the animal has been exposed to the parasite but does not prove that it is the cause of the clinical signs that are exhibited. Some laboratories report antibody titre levels although antibody titres do not appear to correlate with organism shedding or severity of lesions found at necropsy. Testing for both IgG and IgM antibodies appears to be more useful but still has limitations.

**Treatment of encephalitozoonosis**
Several medications have been used to treat *E. cuniculi* although it is difficult to prove their efficacy. The subjective opinion of the owner of whether their rabbit 'seems better' may be the only indicator that is available to assess the success of treatment. Prior to 2001, albendazole was the treatment that was recommended because of its success in human patients. Now, in the UK, fenbendazole is used more than albendazole. Although toxicity studies suggest that benzidamazoles are very safe in rabbits, there are reports of haemorrhage and bone marrow suppression in a small number of rabbits, especially if they were overdosed. Albendazole was the main culprit. As a result of concerns about toxicity, some vets recommend treatment protocols based on antibody results and recommend treatment for IgM positive rabbits only with the advice to base further treatment on regular monitoring IgM antibody status. In practice, it often easier, and definitely cheaper, for owners to treat rabbits with fenbendazole without knowing their rabbit's antibody status, in a similar way they worm their dog without knowing whether it is infected or not. In the author's anecdotal and subjective opinion, the incidence of clinical encephalitozoonosis has declined with the advent of Panacur Rabbit and Lapizole, which are two prophylactic fenbendazole products that are sold in the UK.

Other products that are used to treat *E. cuniculi* in rabbits include oxytetracycline and pyrimethamine and ponazuril although their efficacy is questionable. Lufenuron has also been suggested as a treatment because it is a chitin synthetase inhibitor.

Some vets recommend no treatment at all for *E. cuniculi*, especially for vestibular disease, because they believe the syndrome is idiopathic and self-limiting, as it is in elderly dogs. This could be the case. It is true that many cases of vestibular disease resolve with or without treatment and a few cases continue to deteriorate despite treatment.

Corticosteroids are often used for acute neurological signs associated with *E. cuniculi*. They are indicated to suppress the inflammatory response associated with cell rupture. Recent studies have suggest they are ineffective and there are concerns about effects on the liver if corticosteroids are used as a course rather than a single dose.

In addition to antiparasitic and anti-inflammatory therapy, there are many other palliative options depending on the clinical manifestation. These include antibiotics, analgesics, prokinetics, dietary modification to reduce calcium intake, diuretics and motion sickness remedies.
Myxomatosis

Myxomatosis is a fatal disease of the European rabbit (Oryctolagus cuniculus). It is caused by a poxvirus and is characterised by subcutaneous swellings that exude a mucoid secretion when sectioned (myxomas). Lesions occur around body orifices and on the face especially on the eyelids.

Disease spread

Myxomatosis is mainly spread by biting insects, especially mosquitoes (Aedes and Anopheles spp.) and fleas. Midges (Culicoides spp.) can also transmit infection when the weather conditions are right. Any insect that penetrates the skin can transmit the disease.

In the UK, the European rabbit flea *Spilopsyllus cuniculi* is the main vector. Its life cycle is synchronised with the reproductive status of rabbits. The fleas can be carried on adult rabbits but need to be in the vicinity of a pregnant female in order to become sexually mature and reproduce. An increase in the flea population is synchronised with the birth of baby rabbits and the fleas feed on young rabbits in the nest. This results in heavy flea burden in neonates. Fleas infected with myxomatosis virus can survive in the nest and maintain infectivity throughout the winter. They act as a reservoir of infection for the following year when the young rabbits are born.

In rabbits that live in close proximity with other rabbits, myxomatosis can also be spread directly between rabbits by contact or inhalation. The virus persists in hutches that have been contaminated with fluid from lesions from infected rabbits and will infect unvaccinated rabbits that are put into them. *Cheyletiella parasitovorax* and cat and dog fleas (*Ctenocephalides* spp.) can act as vectors in the spread of disease.

Virus strains

Different strains of the myxoma virus show a variation in virulence. Rabbits infected with highly virulent strains die so quickly that the disease is not transmitted as readily as the less virulent strains. Environmental temperature has an effect on mortality rates with the disease being more lethal at low temperatures. There is a genetic resistance to myxomatosis in some individuals.

In wild rabbits, outbreaks of myxomatosis wax and wane according to the virulence of the strain and the immune status of the native rabbit population. Myxomatosis can occur in hares but infection is rare and usually mild.

Atypical myxomatosis

The term ‘atypical myxomatosis’ is confusing as it has two definitions.

1. Atypical myxomatosis is used to describe a mild non-lethal form of the disease. Some laboratory strains have been produced but in veterinary practice, atypical myxomatosis is usually seen in vaccinated rabbits that have a partial immunity. It presents as isolated crusting skin nodules that regress over a few weeks. The lesions can alarm owners, who worry that they may be neoplastic. The rabbits remain well.

2. Atypical myxomatosis is also used to describe outbreaks in farmed rabbits where the rabbits do not show typical skin lesions but die from respiratory disease instead. This form of myxomatosis is spread by aerosol infection. This syndrome is characterised by a longer incubation period (1-3 weeks) and accompanied by lacrimation and mucopurulent nasal discharge. As there are no skin lesions with this type of infection, the term ‘amyxomatous myxomatosis’ is sometimes used.
Clinical signs of myxomatosis
The most common route of infection is through an insect bite. Replication of the virus takes place at the inoculation site and in the regional lymph node. It is followed by cell associated viraemia and generalised infection throughout the body. The disease starts with a skin lesion, which typically develops 4-5 days after inoculation of the virus and enlarges to become about 3cm in diameter 9-10 days after infection. The rabbit is viraemic, with virus replication taking place throughout the lymphoid system. The eyelids become thickened and eventually the eyes are completely closed by the ninth day with a semipurulent ocular discharge. Secondary swellings develop throughout the body, typically on the nares, lips, eyelids and base of the ears and on the external genitalia and anus. Initially, there is hyperaemia followed by soft swellings that enlarge, harden and become crusty. These lesions then become necrotic and the skin may blacken and die. Eventually (if the rabbit survives) the necrotic tissue falls away and the skin heals although there may be some deformity. In male rabbits that recover, inflammation of the testicles makes them infertile for up to 12 months.

The severity and distribution of the lesions affects the outcome of the disease. It takes approximately 6-8 weeks for the lesions to regress. If the rabbit can survive this period, it may recover but many cases do not. At the outset, it is hard to know which rabbits will survive and which ones will die. Several problems can develop. Lesions around the nose can block the nares and cause severe respiratory distress. Secondary infection and pneumonia are common. The cause of death is not always clear.

Treatment
The rabbit may need nursing for several weeks. The important features appear to be:

- **A warm environment.** Ambient temperature affects the course of the disease with high environmental temperature increasing recovery rate.
- **Antibiotics.** Although antibiotics will not be effective against the virus, they can control secondary infection. Parenteral penicillin is the first choice and may be given daily or long acting products can be given bi-weekly.
- **Topical ointments** to soften and protect skin lesions. Chloromycetin eye ointment is a good choice.
- **Good nursing** i.e. clean bedding that does not stick to the lesions, tempting food and handfeeding. Syringe feeding can be difficult if the rabbit has blocked nostrils. Rabbits that cannot or will not eat on their own should be euthanased.
- **Analgesia** Non-steroidal analgesics are useful. Opioid analgesics due not appear to be effective in ameliorating signs of pain. In a study of the effect of buprenorphine on the course of myxomatosis in laboratory rabbits, there was no difference in survival time.
- **NO corticosteroids.** The use of corticosteroids is contraindicated due to their immunosuppressive effects.

Owners must be aware that nursing a rabbit through myxomatosis can be a harrowing experience and it is not their fault if the rabbit dies. The rabbit’s temperament, immune status and the virulence of the virus strain all play a part in the course of the disease. The chances of survival are small.
Rabbit viral haemorrhagic disease (RHD, VHD)

Rabbit viral haemorrhagic disease (RHD) is a highly infectious lethal disease of rabbits with a high mortality rate. It is caused by a host specific calicivirus with a predilection for hepatocytes causing a necrotising hepatitis, often associated with necrosis of the spleen. Disseminated intravascular coagulation (DIC) produces fibrinous thrombi within small blood vessels in most organs, notably the lungs, heart and kidneys resulting in haemorrhages. Death is due to disseminated intravascular coagulopathy or to liver and/or kidney failure.

RHD has a short incubation period of 3-4 days and the disease can be peracute with animals being found dead within a few hours of eating and behaving normally. Acute cases are quiet, pyrexic with an increased respiratory rate before becoming pallid, shocked and collapsed. Haematuria, haemorrhagic vaginal discharges or foamy haemorrhagic exudate from the nostrils may or may not be seen. Vascular infarcts can occur within the brain and occasionally convulsions or other neurological signs are seen just before death. The 'classic' picture is a dead rabbit in opisthotonus with a haemorrhagic nasal discharge. The occasional rabbit can recover from the acute phase, only to develop jaundice and die a few days later. Very occasionally, a rabbit will survive and recover. There is no treatment for affected rabbits apart from supportive care.

Variants of RHD

'Classic' Rabbit Haemorrhagic Disease (RHD, RHDV, RHD1)
The first outbreak of RHD in China appeared to originate from a colony of Angora rabbits that were imported from Germany. Within a year, there was a loss of over 140 million rabbits. The disease was introduced to Korea by rabbit fur and subsequently spread to other countries in Asia. In Europe, RHD was first diagnosed in Italy in 1986 and by 1988; the disease was reported in many other countries worldwide where it was probably introduced through rabbit meat. In the UK, the first recorded outbreaks of RHD in wild rabbits were recorded in 1994.

Non-pathogenic rabbit calicivirus (RCV)
Although fatalities from RHD were first reported in 1984, retrospective testing showed antibodies to the virus in sera collected before that time. It was proposed that non-pathogenic strains of RHDV (RCV) could have protected rabbits by stimulating antibody production and conferring cross immunity.

New variant (RHDV2)
In 2010, an atypical outbreak of RHD occurred in a rabbitry in France in which 25% of the rabbits that were vaccinated died. Samples from affected rabbits were genetically analysed and the virus that was causing the fatalities was found to be related to, but highly distinct from, the strains of RHD that were isolated from previous outbreaks. This variant is known as RHDV2. There appears to be little or no cross immunity between RHD and RHDV2. The mortality rate is lower with RHDV2 than RHD1, so some rabbits are unaffected and others can recover. The carrier status of these rabbits is not known. The low mortality of RHDV2 may explain why the disease has spread rapidly throughout the UK and other countries. There are outbreaks throughout the country and the disease has killed many pet rabbits. Unlike RHDV, it also appears to affect hares. se is common in the UK and has affected many areas.
Natural immunity to RHDV and RHDV2
Most young rabbits less than 4 weeks of age remain unaffected by the original strain of RHD and develop a life-long immunity if they are exposed to the disease. Unexposed rabbits become increasingly susceptible until 6–10 weeks of age when physiological resistance to the virus disappears. Subsequent exposure to the virus boosts immunity that protects these young rabbits when they reach adulthood. This age immunity does not occur with RHDV2. Young and suckling rabbits may be affected with RHDV2.

Vaccination
Due to the devastating effects of RHD in China, a vaccine was quickly developed from inactivated virus obtained from the liver and spleens of infected rabbits. Although the immunological response to inactivated vaccines (Cylap, Lapinject) was good, in the UK, they have been superseded by a bivalent vaccine that protects against both myxomatosis and RHD (Nobivac Myxo-RHD). It is constructed from a laboratory-attenuated strain of myxoma virus and the capsid protein gene of RHDV. The vaccine is ineffective or only partially effective against RHDV2 so vaccinated rabbits can still succumb to the disease. Vaccines against RHDV2 have been developed other parts of Europe e.g. Filavac or Eravac that are now licensed for use in UK.

Diagnosis
The diagnosis of RHD is usually made at post mortem examination. RHD is suspected in any sudden death especially if more than one rabbit in the household has died. The post-mortem picture may be of a healthy rabbit with non-impacted food in the stomach and hard faecal pellets in the distal colon, suggesting that death was sudden.

Gross post-mortem signs may be minimal or obvious. Suggestive findings include changes in the liver, splenic enlargement and haemorrhages around the body. Histopathology confirms acute hepatocellular necrosis. PCR testing will confirm the diagnosis and identify the variant. Fresh or frozen liver is required. Liver is the most reliable tissue to submit for PCR. False negatives can occur on blood or faeces. There is a possibility of false positives after vaccination.

Parasites
Roundworms.
Several types of roundworms can affect rabbits. Examples include *Trichostrongylus retortaeformis*, *Graphidum strigosum*, and *Obeslicoides, funiculi* and *Passalurus ambiguus*. Most species are found in wild rabbits and have a direct lifecycle. Domestic rabbits are rarely affected because they have no access to contaminated pasture. *Passalurus ambiguus* is sometimes encountered in pet rabbits, because its lifecycle is short, which allows the eggs to become infective quickly so faecal contact can transmit the parasite. The eggs or adult worms can pass out in caecotrophs or hard faeces and transmit infection. *Passalurus ambiguus* is a tiny worm, only 4-11mm in length that inhabits the caecum and colon. It is sometimes called a pinworm or threadworm, which has led to some confusion with the human threadworm that has a different lifecycle. The human threadworm emerges from the anus during the night to lay its eggs, which causes anal irritation. This is the reason for a common misconception among owners that worm infestation always causes anal irritation. It is not true in rabbits.
Tapeworms

Tapeworms are segmented worms with a complicated lifecycle that involves another animal species. In the primary host, the worm anchors itself to the intestinal wall by hooks in the scolex. The scolex then divides and forms segments that mature as they move down the worm. Eventually the segments break off and pass out in the faeces. They look like grains of rice. As the segments disintegrate, eggs are released into the environment and consumed by another species in which they develop into cysts that contain an immature scolex. These cysts remain dormant until the secondary host is eaten by the primary host where the scolex anchors itself in the intestine and forms a new tapeworm.

Rabbits as primary hosts for tapeworms

*Cittotaenia* is the most common species of tapeworm that affects rabbits. It is rare in pets but like many tapeworm, fenbendazole is ineffective as a treatment so it if often *Cittotaenia* that is found in cases of ‘worms’ in rabbits that are still present in rabbits that have been treated with fenbendazole. A free living mite is the secondary host. Rabbits are infected by grazing on contaminated pasture. Praziquantal is effective but if the mites in a garden or grass enclosure have become infected, recurrence can occur.

Rabbits as secondary hosts for tapeworms

Rabbits are the secondary hosts for several species of tapeworm. Foxes are the usual primary host although dogs that have eaten raw rabbit carcasses can also be infected. In the UK, *Taenia serialis* and *Taenia pisiformis* are the most common tapeworms that cause cysts in rabbits. Rabbits are infected by eating plants that have been contaminated by faeces from a fox or dog that has a tapeworm. After the rabbit ingests the tapeworm egg, the parasite can migrate through the body and form a cyst. *Taenia serialis* eggs develop into cysts that form in muscle or tissues under the skin. They can develop into large swellings that contain fluid and many scolices. Any health problems are due to the size of the cyst or where it has formed e.g. behind the eye or in the tongue. *Taenia pisiformis* forms cysts in the abdominal cavity and rarely cause harm although they may be seen during surgery or post-mortem examination.

Worming pet rabbits

No roundworms that affect rabbits pose a health risk to humans. They do not even pose a risk to the rabbit unless the worms are present in large numbers. Regular worming with fenbendazole is not necessary for pet rabbits. They rarely have roundworms and if they do, the worms are obvious and not harmful to the rabbit or their owner although the appearance of the worms may be unpleasant. If *Passalurus* worms are seen in the faeces, it is also worth considering where they came from. *Passalurus* usually comes from the breeding establishment and can spread (via faeces and caecotrophs) between rabbits in the same household. If worms are seen in one rabbit, all the others will need treatment and the environment needs to be cleaned carefully to remove all the faeces with eggs in them. Wild plants are a potential source of roundworm infestation but the rabbit would need to graze the area. Long stems are unlikely to be infected although a plant that is pulled up rather than cut may in some contaminated soil. Plants (including hay) contaminated by dog or fox faeces are the source of tapeworm cysts, but the risk is very small.
**Ectoparasites**

The most common ectoparasites in pet rabbits are *Leporacus gibbus* and *Cheyletiella parasitovorax*. These mites can be found in asymptomatic rabbits. Clinical signs (scaling, pruritis) are usually associated with heavy *Cheyletiella* infestation and invariably linked with a grooming problem. Dental problems, especially incisor problems, obesity and spondylosis are often associated with cheyletiellosis because the rabbit cannot groom and remove the mites and scale from the fur. *Psoroptes cuniculi* is less commonly encountered and tends to be a disease of breeder’s rabbits rather than the individual pet.

Fleas (*Ctenocephalides* spp.) can be transmitted to house rabbits from dogs and cats. They usually cause intense pruritis. Flea allergic dermatitis can occur. Rabbit fleas (*Spilopsyllus cuniculi*) are rarely seen in pet rabbits. Close contact with wild rabbits or their burrows is required. Lice are also unusual.

Selamectin (8-16mg/kg) is a very effective treatment for mites in rabbits. It seems to be more effective than ivermectin, presumably because a single application lasts long enough to kill all stages of the life cycle. It is also effective against fleas although imidacloprid (Advantage) is preferable and carries a product license for use in rabbits. Fipronil (Frontline) should not be used in rabbits. There are reports of fatalities after its use.