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Viral diseases of the rabbit

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This article is designed to be a practical reference for veterinarians who see rabbits in private practice. We have not attempted to thoroughly cover major aspects of husbandry, biology, or nonviral disease; the reader is referred to other references for these purposes [1–4]. We have emphasized naturally-occurring viral infections that cause clinically significant disease. This emphasis comes at the expense of discussing viral pathology and alterations in physiology that may be important in research settings but have less clinical impact in a practice setting.

This paper is subdivided into sections based on the organ system that is affected predominantly by infection. We recognize that this format may oversimplify the multi-systemic effects of viral diseases but believe that the approach will be helpful for easy reference and discussion of the most common clinical presentations. Each section includes a brief overview of the viral agent, host susceptibility, disease transmission, clinical signs, diagnosis, control, and prognosis. Important differential diagnoses for disease presentation also are described when appropriate.

Treatment of viral disease centers on supportive care; in some cases, the prognosis may be grave. We recommend that practitioners make use of their clinical judgment when dealing with a sick rabbit, including aggressive treatment (eg, encouraging gastric motility), meeting the patient's fluid needs, and removing nonessential stressors. In a multiple rabbit household or in a rabbitry, control measures to minimize the contagious nature of viral disease must be implemented. In rare instances, it may be appropriate to discuss the possibility of zoonotic disease transmission.

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Viral diseases of the neurologic system

Viral diseases that affect the central nervous system of rabbits rarely have been reported; however, rabies and herpes simplex infections have been associated with neurologic disease in this species [5–7].

Rabies

Between 2000 and 2002, eight laboratory-confirmed cases of rabies in rabbits (*Oryctolagus cuniculus*) were reported to the Centers for Disease Control and Prevention. All reports occurred in states in which rabies is enzootic in raccoons [8–10]. In the few clinical case reports that documented rabies infection of the domestic rabbit (*O cuniculus*), a paralytic form of the disease was observed principally. In these cases, infected rabbits had a history of being housed in an outdoor situation and had confirmed or potential exposure to wildlife [5,11–13]. Initial signs of disease in infected rabbits have been nonspecific, including anorexia and lethargy; in one case, these were the only signs that were observed [12]. As the disease progresses, neurologic signs, including head tremors, blindness, and ascending paralysis, have been described; the affected rabbit often dies within 3 or 4 days [5,11–13].

Rabies is diagnosed in animals by means of postmortem direct immunofluorescent antibody testing of brain tissue by state or local health departments [14]. Although rabies is rare in rabbits, they are susceptible to infection and initial signs of infection may be nonspecific in nature. Rabies infection should be considered as a rule-out in rabbits who demonstrate symptoms of a rapidly progressing neurologic disease with a history of outdoor housing or possible exposure to wildlife or other potentially rabid animals. No approved rabies vaccination for use in rabbits is marketed in the United States [5]. Clients who keep rabbits outdoors should be cautioned to protect their animals from potential exposure to feral animals.

Herpes virus hominus

Naturally-occurring disease that is associated with herpes virus hominus infection has been reported in the domestic rabbit; there have been two reports of clinical disease associated with this virus. In both cases, a person who had a herpetic lesion (eg, cold sore) was known to have close contact with the infected animals [6,7]. Infected rabbits exhibited signs of anorexia and neurologic abnormalities, including restlessness, circling, and tonic-clonic spasms. In both cases, neurologic disease progressed to lateral recumbency at which point the animals were euthanized. On histopathology, a nonsuppurative meningoencephalitis with neural cell necrosis and intranuclear inclusion bodies was observed. The agent involved in the infection was classified as herpes virus hominus subcategory herpes simplex virus by polymerase chain reaction (PCR) and immunohistochemistry.

Viral diseases of the integument

Viral infections of the integument are more common in the rabbit than in most species. The viruses that are associated with skin disease in the rabbit belong to the pox and papova virus families.

Myxoma virus

Myxoma virus is a leporipoxvirus of the Poxviridae family [15]. In 1896, myxoma virus infection was documented in European rabbits that were located in Uruguay. This outbreak was associated with high mortality and numerous mucinous skin masses on affected animals [16]. In 1928, myxoma virus was first recognized in North America when an outbreak of fatal disease was observed in California [17].

Myxoma virus is transmitted principally by arthropod vectors, mosquitoes, and fleas. Rabbits of the genus *Sylvilagus* serve as the natural host for myxoma virus; in them, myxomatosis produces a benign skin disease that is associated with the development of local skin tumors that resemble fibromas at the site of viral inoculation by way of bite by mosquito or other blood-sucking insect [18]. In the European hare (*Lepus europaeus*), myxoma virus infection rarely has been associated with disease. Infection in the European or domesticated rabbit (*O cuniculus*) has been associated with systemic disease with mortality rates of up to 100% [19]; however, the presentation of disease depends greatly upon the strain of virus and the particular species of rabbit [20,21]. Typical skin lesions that are associated with disease in the domestic rabbit include edema of the eyelids, ears, nose, anus, and genitals; blepharoconjunctivitis; hemorrhage; and nodules on the ears, head, body, and legs at the site of infection that may become congested or necrotic [17,19,22–24]. Histologically, the skin lesions consist of the proliferation of stellate (myxoma) mesenchymal cells surrounded by a mucinous matrix. In addition, endothelial cell proliferation, intracytoplasmic inclusions in various cell types, and epidermal cell hyperplasia or degeneration in advanced lesions may be observed [19,22,24,25].

For a thorough description of the systemic disease that is associated with myxoma virus refer to the section that details viral multi-systemic diseases.

Rabbit (Shope) fibroma virus

Rabbit fibroma virus is a member of the Poxviridae family and is related closely antigenically to myxoma virus [26]. Rabbit fibroma virus was first identified in an Eastern cottontail rabbit (*S floridanus*) in 1932 [27]. This species was shown to serve as the natural host for the virus, but other species of cottontail rabbits and European rabbits (*O cuniculus*) have proven to be susceptible as well [19,27]. Initially, this virus was considered to be a benign skin disease of cottontail rabbits; however, it since has been associated with an epizootic of disease in domestic rabbits that resulted in high morbidity

and mortality in newborn animals. Neonatal infection was associated with systemic disease that was characterized by lethargy, poor body condition, and death [28].

Rabbit fibroma virus infection has been recognized in cottontail rabbits in the United States and Canada [18]. The natural cycle of infection is not understood completely, but biting arthropods may serve as the primary means for virus transmission [29–31].

Infection in European rabbits (*O cuniculus*) is characterized by flat, freely movable, subcutaneous tumors that primarily are located along the legs, feet, ears, muzzle, and around the eyes (Fig. 1). These tumors may measure several centimeters in diameter and resolve spontaneously within a few months. Microscopic examination of the skin lesions reveal proliferation of mesenchymal cells which become stellate or ovoid, multifocal areas of necrosis, and mononuclear and polymorphonuclear infiltrates [28,32–34].

Diagnosis of rabbit fibroma virus is based upon clinical presentation and histopathologic examination of biopsied masses. Differentiation of rabbit fibroma virus from infection with myxomatosis and papillomatosis in the domestic rabbit should be accomplished readily based upon characteristic gross lesions [17]. Control of disease may be undertaken by limiting



Fig. 1. Rabbit (Shope) fibroma virus. Photograph of a Rex rabbit that presented with classic multi-focal subcutaneous masses located around the eyes, digits, and in the genital region subsequent to rabbit fibroma viral infection. (Courtesy of Craig Franklin, DVM, PhD, DACLAM, University of Missouri, Columbia, Missouri).

exposure to arthropod vectors. Because tumors regress spontaneously, treatment, in general, is not necessary [35].

Cottontail rabbit (Shope) papillomavirus

The cottontail rabbit (Shope) papillomavirus was the first oncogenic virus to be identified in mammals. In 1933, this virus was found to be associated with the appearance of wartlike tumors in the cottontail rabbit (*S floridanus*) [36]. Although the cottontail rabbit serves as the natural host, this virus is transmissible to domestic rabbits (*O cuniculus*) and was responsible for a spontaneous outbreak of papillomatosis in domestic rabbits from southern California [37,38].

Cottontail papillomavirus has been associated with disease in the cottontail rabbit population in the midwestern United States and in domestic rabbits in California [38,39]. This virus may spread by direct contact; however, the principal mode of transmission occurs through arthropod vectors. The rabbit tick (*Haemaphysalis leporis-palustris*) is suspected to be the most common mode of transmission in the cottontail rabbit; however, experimental transmission of this virus also was demonstrated by mosquitoes and reduviid bugs [40,41]. In the domestic rabbit, the predominant location of papillomas along hairless areas of the ears and eyelids suggests that transmission from the cottontail rabbit to the domestic rabbit may occur primarily through the mosquito [38].

In the cottontail rabbit, infection is characterized by wartlike protuberances that are located predominantly along the neck, shoulders, and abdomen. At the sites of infection, these warts start as red, raised areas that become papillomas and eventually may develop into large, keratinized horny growths. These growths may vanish within a few months or they may become neoplastic in nature and be replaced by squamous cell carcinomas [36,42]. In the domestic rabbit, spontaneous infection was characterized by horny protuberances that were located most commonly along the ears and eyelids (Fig. 2) [38]. Experimental infection of the domestic rabbit revealed a lesser degree of papilloma regression than was observed with the cottontail rabbit and a high rate of carcinoma development following infection [43].

Cottontail papillomavirus may be diagnosed based upon characteristic clinical presentation and histopathologic examination. In areas where infection is endemic, natural infection may be controlled through control of the arthropod vector. Treatment may be accomplished through surgical removal of the papilloma [35].

Viral diseases of the gastrointestinal system

Gastrointestinal disease in rabbits is common and accounts for a significant economic loss. Viral diseases that affect the gastrointestinal system of rabbits include papillomavirus, parvovirus, rotavirus, and enteric



Fig. 2. Cottontail rabbit (Shope) papillomavirus, papillomatosis. Photograph of a domestic rabbit that presented with an approximately 1.5-cm keratinized horny protuberance located along the dorsal surface of the ear pinna secondary to cottontail rabbit papillomavirus. (Courtesy of Craig Franklin).

corona virus. Prominent nonviral causes of gastrointestinal disease include colibacillosis, coccidiosis, and enterotoxemia [4]. Coinfection by viral and nonviral pathogens is common and often cause more severe clinical signs than the viral pathogen alone would induce.

Papillomavirus

Oral papillomatosis in the rabbit is caused by a papillomavirus in the Papovaviridae family and is not related antigenically to the Shope papilloma virus [44]. The prevalence of oral papillomatosis in the rabbit has been described as “infrequent.” The estimated prevalence has been reported to be between 5% and 33% [44–47]. Infection has been noted most frequently in animals between the ages of 2 months and 2 years [44,46,47].

Infection with oral papillomavirus is characterized by the development of small (1–2 mm) papillomas that usually occur on the ventral aspect of the tongue, although other locations on the tongue and in the oral cavity have been reported [46–48]. One report described lesions that were as large as 10 mm behind the mandibular incisors [45]. Gross lesions have been noted as soon as 14 days after experimental infection and reach their maximal size after approximately 1 month; most lesions begin to regress spontaneously [45,46,48]. Experimental infection only has been demonstrated when the rabbits were inoculated following artificial damage to the epithelium of the tongue and oral cavity [48]. This suggests that rough or hard food, chewing on rough cage bars, or malocclusion may predispose animals to infection with the oral papillomavirus. Animals that were exposed previously to oral papillomavirus (seropositive) were resistant to reinfection [46]. The duration of this immunity is not known.

Microscopically, oral papillomavirus lesions consist of proliferating epithelial cells with hyperkeratosis, with or without a fibrovascular stalk.

Cells at the junction of the stratum spongiosum and the papilloma have abundant, clear cytoplasm with eccentric nuclei and basophilic intranuclear inclusions [44,46,48].

Affected animals show few clinical signs and the disease usually is diagnosed incidentally upon finding the characteristic papillomas on the tongue or in the oral cavity during preparation for surgery or during necropsy [45,47]. The absence of obvious clinical signs may account, in part, for the variation in reported prevalence.

There are few differential diagnoses for oral papillomas in the rabbit; however, one case report noted a sialoceles (ranula) on necropsy that was mistaken for an oral papillomavirus lesion [49]. The diagnosis of rabbit oral papillomatosis usually is based upon demonstration of the characteristic papillomatous lesion in the oral cavity. Because the lesions are transient and clinical signs usually are not associated with the disease, treatment or removal of oral papillomas is unnecessary in most cases. Although natural transmission to other species has not been noted, hamsters developed fibromas following experimental infection with rabbit oral papilloma virus [46].

Parvovirus

Just as in many other species, parvovirus infection in rabbits was shown to produce clinical disease [50,51]. Lapine parvovirus (LPV) was first isolated from rabbits in Japan in 1977. This investigation found antibodies to LPV in 46.7% of 90 commercial rabbits that were tested [52]. A similar study that was conducted in the United States using rabbits (*O cuniculus*) that were held in a laboratory setting demonstrated that 75% of 46 animals were seropositive for LPV [51].

Although the percentage of animals that is seropositive is high, the clinical signs that are caused by the disease are mild. Following experimental inoculation, clinical signs included listlessness and anorexia 4 to 6 days postinoculation. Virus was recovered from the liver, pancreas, spleen, small intestine, cecum, mesenteric lymph nodes, and feces for 14 days following inoculation. Microscopically, the virus caused mild to moderate catarrhal enteritis with hyperemia, exfoliation of enterocytes, and luminal fluid accumulation [50].

Given the short duration and minimal clinical signs, diagnosis of parvoviral disease in rabbits may prove to be difficult in a clinical setting. Isolation or demonstration of viral particles from the feces is the primary diagnostic method. To the authors' knowledge, a serologic test is not commercially available. Treatment of uncomplicated parvoviral infection is supportive.

Rotavirus

Rotaviral infections were shown to cause diarrhea in a wide variety of host species. Rotaviruses are a member of the Reoviridae family. Initially,

rotavirus was isolated from rabbits that had diarrhea in England in 1976 [53]. Since that time, rabbit rotavirus has been studied in depth and has been used in modeling the immune response and in developing vaccines for human rotaviral infection [54]. Studies showed that the rotaviral strains that infect rabbits are group A serotype 3, subgroup I and II rotaviruses [55,56]. These serotypes are common among a variety of species, including humans and cattle [56,57].

Although the true incidence of rotaviral disease in rabbits is not known, antibodies were demonstrated in 29% of 17 wild cottontail rabbits (*S. floridanus*) in Ontario, 52% of 27 snowshoe hares (*L. americanus*) in the Yukon, and 98% of 91 commercially-raised New Zealand White rabbits (*O. cuniculus*) from two rabbitries in Ontario [58]. The presence of rotaviral antibody in rabbits was shown to be age-dependent. More than 90% of neonates (<1 month), weanlings (2–3 months), young adults (3–4 months), and breeding adults (>5 months) had antibodies to rotavirus, whereas only 25% to 69% of preweanling (1–2 months) animals had antibodies to rotavirus [59,60]. This corresponds with other reports that show the peak prevalences of rotaviral disease in endemically-infected rabbitries is in animals that are between 36 and 42 days old [61]. This suggests that animals become infected after maternal antibodies wane. In naïve and experimentally-inoculated colonies, 1- to 2-week-old animals were the most severely affected [62,63]. Following experimental infection, animals between 1 and 2 weeks old were the only age group that showed clinical signs. Fecal viral shedding is similar for all age groups and tapers around 10 days after experimental infection [63].

The most common clinical signs in rabbits that have rotaviral disease are diarrhea and dehydration [53,62,64,65]; however, rotaviral infections may be clinically silent, with the affected animals shedding virus in their feces [53,54,60,66]. Experimental infections with rotavirus that was isolated from the feces of animals that had severe diarrhea resulted in only mild clinical signs [63–65]. This suggests that other factors may be involved when rotavirus is isolated in outbreaks of severe diarrhea and a high mortality rate. This is supported by a study that demonstrated more severe diarrhea and an increased mortality rate when animals were coinfecting with *Escherichia coli* [65]. On necropsy, animals that are infected with rotavirus have a distended, fluid-filled small intestine and cecum [58,64]. Histopathologic findings spanned the jejunum and the ileum; lesions included shortened, fused villi with attenuated epithelium and slight to moderate increases in crypt depth [62,64].

A clinical diagnosis may be made based upon clinical signs in the proper age group or viral isolation from the feces of animals that have clinical signs. An antigen capture ELISA, such as those used in rotaviral diagnosis in cattle [67], also may be useful given the similarity in serotypes. Differential diagnoses include coccidiosis, Tyzzer's disease (*Clostridium piliforme*), clostridial enteritis/toxemia, colibacillosis (*E. coli*), and cecal dysbiosis (mucoid enteropathy) [4].

Treatment of an individual animal relies upon fluid and electrolyte replacement, either orally or subcutaneously. Treatment in a rabbitry might include discontinuing breeding for 4 to 6 weeks to stop the influx of naïve animals and to allow infected animals adequate time to mount an immune response and stop shedding the virus [18]. After an individual animal has recovered, it is resistant to subsequent infection [54,66,67].

Coronavirus

Coronaviral infections in rabbits have two manifestations: (1) enteritis and (2) pleural effusion disease and cardiomyopathy [68]. Cardiomyopathy was reported following coronaviral experimental infection in a laboratory setting; however, there are no reports to suggest that this occurs as sequelae to naturally-occurring infection [2,69–71]. Coronavirus was linked first to diarrhea in rabbits in Canada in 1980 when viral particles that were consistent with the Coronaviridae family were noted on electron microscopic evaluation of fecal matter from rabbits that presented with diarrhea [72]. In a survey of rabbitries from the northwestern United States and southwestern Canada, the prevalence of antibodies to rabbit enteric coronavirus (using a canine coronavirus test) ranged from 3% to 33%. Rabbitries with a high prevalence of diarrhea were more likely to have antibody titers to rabbit enteric coronavirus (RECV). The cross-reactivity with canine coronavirus (type I coronavirus) and lack of cross-reactivity with mouse hepatitis virus (type II coronavirus) suggest that RECV is a type I coronavirus [73]. Viral particles have been isolated from the feces of experimentally-infected rabbits as long as 29 days postinfection [74].

Clinical signs of RECV infection in rabbits include watery diarrhea, abdominal distention, anorexia, and sudden death [68,72,74]. Rabbits that are between 3 and 10 weeks old are affected most commonly [72]. Experimentally infected animals have not shown sudden death as a clinical sign; this suggests that like rotaviral infections, concurrent infections with other organisms may exacerbate clinical signs. Experimental infection has revealed that gross lesions (eg, congested small intestines and fluid cecal contents) may be noted 6 hours to 3 days postinoculation. On histopathologic examination of the small intestine, necrosis of the villous epithelial cells has been noted within 6 hours of experimental infection. By 48 hours postinoculation, small intestinal villous blunting, crypt hypertrophy, complete M-cell necrosis, and necrosis of villous epithelial cells overlying the gut-associated lymphoid tissue was noted. In addition, RECV may result in subclinical infections [74].

Diagnosis may be made by finding viral particles in the feces of rabbits that have diarrhea or characteristic lesions on necropsy and histologic evaluation of the gastrointestinal tract. A commercially-available serologic assay for RECV is not known to the authors but it may be possible to

detect RECV by using a commercial canine coronavirus test [67]. In addition, use of serology may be of limited immediate utility since animals that develop diarrhea are unlikely to have mounted an antibody response to RECV.

Treatment of individual animals includes isolation from unaffected rabbits and supportive therapy. Management changes in the face of an outbreak (eg, delayed weaning, diet changes, broad spectrum antibiotic treatment, vaccination) have not been successful in preventing morbidity and mortality [75].

Viral multi-systemic diseases

Rabbit caliciviral disease

Rabbit viral hemorrhagic disease (RHD) is related closely to the European brown hare syndrome (EBHS); both diseases are caused by related, but antigenetically distinct, caliciviruses [2,76,77]. RHD is an extremely contagious disease and is the only rabbit disease that is reportable to the U.S. Department of Agriculture. Disease was first reported in China in 1984 and is now endemic in New Zealand and Australia. Outbreaks of disease in American domestic rabbits were documented recently [78,79]. A recent outbreak in Illinois resulted in the euthanasia of more than 4800 rabbits to stop the spread of disease [78]. If RHD is suspected, the state and federal veterinarian should be contacted immediately and quarantine should be instituted.

Confirmation of disease can be performed with PCR of tissue extracts, hemagglutination assay, and ELISA; however, histopathologic examination of the liver and spleen also may serve as a means of diagnosis [78,80]. Clinical signs may include nonspecific illness, fever, convulsions, morbidity of more than 30%, and mortality of 90% or more in affected domestic (*O cuniculus*) rabbit populations. Disseminated intravascular coagulopathy plays a prominent role in disease pathology; gross findings at necropsy may include blood-stained nasal discharge, hepatomegaly, splenomegaly, and hemorrhage on surfaces, including the pericardium and intestine [2,81]. EBHS has been reported to affect wild and farmed hares (*L timidus* and *L europaeus*) in many European countries; similar to RHD, outbreaks of explosive disease are possible with similar clinical signs and prevalences of mortality in hares that are affected with EBHS [82,83]. Experimental infection of domestic rabbits with EBHS calicivirus did not result in clinical disease and did not protect animals from subsequent challenge with RHD virus [84]. Outbreaks of RHD in domestic rabbits may occur rapidly owing to an incubation time of only 1 to 2 days and death in 2 to 3 days; fomites, direct contact, aerosols, insect vectors, and carcasses serve as means of spreading disease [2,85].

Herpesvirus

Sudden death in Canadian rabbitries (*O cuniculus*) has been attributed to a herpeslike viral infection. Antemortem signs of disease may be subtle or absent but gross necropsy findings include hydropericardium and multifocal hemorrhages in the skin, abdominal viscera, and heart. Although the prevalence and pathogenesis of this virus is unclear, histopathologic lesions, including intranuclear inclusions that are consistent with herpes virus and isolation of herpesviral particles from lesioned tissues, were found. These findings led some investigators to conclude that herpeslike viral infection was responsible for the outbreak [2].

In wild populations of rabbits, including the eastern cottontail (*S floridans*), serologic surveys demonstrated a low (<4%) prevalence of serum antibodies to *Herpesvirus sylvilagus* [86]. The clinical significance of this is unclear; however, in rare cases, unexplained death in cottontails may be attributed to herpesvirus [87]. Experimental inoculation of pregnant cottontail rabbits demonstrated that this virus is not transmitted across the placenta [88]. Recent studies demonstrated that experimental inoculation of cottontails with *H sylvilagus* resulted in the development of lymphoproliferative lesions that were similar to lesions noted following Epstein-Barr virus infection in humans [89,90].

Myxomatosis

Myxoma virus in its natural hosts (*S brasiliensis* or *S bachmani*) causes only a benign fibroma. In the European or domestic rabbit (*O cuniculus*), however, it can cause a fulminant disease that is known as myxomatosis (or the amyxomatous form of myxomatosis). After its initial introduction into European rabbit populations in Europe and Australia, the virus killed nearly 100% of infected animals [91]; however, the combination of selection for resistant hosts and attenuated strains of virus has greatly reduced the prevalence of mortality due to disease. A study that was performed in Spain demonstrated endemic myxomatosis in wild European rabbit populations, but found no evidence of adult mortality that was associated with this prevalence [92]. A recent study in which naïve European rabbits were infected experimentally with several strains of myxoma virus provided additional evidence of the variable virulence of different strains [93]. Experimental infection of European rabbits with an attenuated form of myxoma virus resulted in transient systemic illness with orchitis and epididymitis; animals recovered from infection and demonstrated normal fertility by 60 to 90 days following infection [94]. There are occasional outbreaks of disease in domestic rabbits in the United States (most commonly California) and Mexico which are believed to represent accidental transmission from the brush rabbit (*S brushmani*) by way of an insect vector [95].

Prognosis and presence or absence of clinical signs vary tremendously with host and viral strain. Clinical signs may be consistent with nonspecific illness and hypothermia (unlike the fever associated with RHD); gross pathology of dead animals may range from edema of the eyelids, conjunctiva, and anogenital region to extensive lung lesions (associated with secondary bacterial infections) [2,25,93]. Diagnosis may be performed through PCR of tissue extracts, gross or microscopic lesions, or inoculation of suspected infected tissue into susceptible rabbits [2]. Treatment consists of supportive care and prognosis varies significantly with strain and host. Vaccine development is on-going but no vaccine is marketed in the United States [96,97]. Clients in California or other areas with increased risk of disease, should be counseled to control potential exposure to insect (flea and mosquito) vectors.

Summary

Viral disease in the rabbit is encountered infrequently by the clinical practitioner; however, several viral diseases were reported to occur in this species. Viral diseases that are described in the rabbit primarily may affect the integument, gastrointestinal tract or, central nervous system or may be multi-systemic in nature. Rabbit viral diseases range from oral papillomatosis, with benign clinical signs, to rabbit hemorrhagic disease and myxomatosis, which may result in significant clinical disease and mortality. The wild rabbit may serve as a reservoir for disease transmission for many of these viral agents. In general, treatment of viral disease in the rabbit is supportive in nature.

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