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Small mammal virology

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This article is intended to provide information on clinically relevant viral diseases of various small mammal species encountered in veterinary practice, including mice, rats, guinea pigs, hamsters, gerbils, chinchillas, prairie dogs, hedgehogs, and sugar gliders. The number of known viral diseases varies enormously with each species, and the majority of viral infections in small mammals are asymptomatic. When researching viral infections of small mammals, one should be aware that much of the literature discusses experimental infections that may result in clinical signs and lesions that do not occur with natural infections. For details of virus biology and experimental studies involving small mammal viruses, the reader is referred to a number of excellent textbooks (see Further Readings).

Most viral infections are transient and asymptomatic. Certain predisposing factors may influence the development of clinical signs due to viral infections. The immune status of the animal is important because very young (<2 weeks old), aged, and other immunocompromised animals are at increased risk of developing clinical disease. Some diseases manifest at weaning age with the waning of maternally derived passive immunity and the relatively naïve juvenile immune system. Ongoing infections (whether bacterial or viral) predispose an animal to developing additional infections, viral or otherwise. Additionally, good husbandry is vital to small mammal health, and suboptimal husbandry can influence susceptibility to viral diseases. High ammonia levels caused by infrequent cage cleanings or overcrowding, high humidity, and poor nutrition contribute to poor health and the development of clinical viral infections. Stress factors such as

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shipping and temperature fluctuations may also influence the development of clinical disease.

Because viral infections are often transient, treatment is generally confined to supportive care if the disease is not complicated by bacterial infections. If secondary bacterial infections are suspected, antibiotic therapy may also be appropriate, but careful consideration should be made when choosing antibiotics for certain antibiotic-sensitive species (eg, guinea pigs). In colony or group-housed settings, viral infections may be very contagious and difficult to eliminate if naïve animals are added or produced through breeding. In these cases, a stop breeding or “burnout” program may be effective to eliminate viral infections. In these programs, breeding is stopped and no new additional animals are added (closed colony) for 6 to 8 weeks. This allows infected animals to develop a sufficient immune response to clear infections and ultimately eliminate the virus from the colony. Quarantine of potentially exposed animals and culling of carriers may also be warranted.

Diagnosis of viral infections is often presumptive and based on clinical signs if present. Serologic testing for most rodent viral agents is available at commercial laboratories, but is often not practical for diagnosis in individual animals because antibodies develop as disease is waning or after signs have abated and the virus has been cleared. However, serologic testing may be warranted in colony settings if exposed animals need to be identified. Additionally, polymerase chain reaction (PCR)-based tests have been developed for the diagnosis of many small mammal infections.

Clinical signs in small mammals

Rodents and other small mammals are active and curious animals, especially at night. Diseased animals are inactive and exhibit hunched postures and poorly groomed, ruffled hair coats (Fig. 1). This collection of clinical signs is referred to as *sick rodent syndrome*. Diseased rats may also accumulate crusty, brick red, porphyrin secretions from the Harderian gland around the eyes, nose, and forepaws. This accumulation of “red tears” is termed *chromodacryorrhea*. Labored breathing is another sign of poor health and may indicate respiratory disease or systemic disease such as bacterial septicemia or trauma.

Viral infections of rats

Respiratory disease in rats

“Respiratory disease of indeterminate nature” is a common complaint among rat breeders and fanciers. Respiratory diseases are often multifactorial, involving pathogens such as *Mycoplasma pulmonis*, cilia-associated respiratory bacillus, rat coronaviruses, and paramyxoviruses. Viral infections

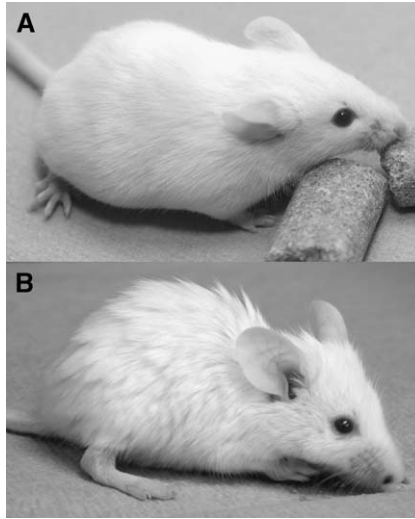


Fig. 1. Photograph of (A) a healthy mouse demonstrating normal groomed haircoat and (B) a mouse exhibiting signs of “sick mouse syndrome,” including rough hair coat and hunched posture.

alone rarely result in respiratory disease, but may contribute to the establishment of more clinically relevant bacterial infections.

Rat coronaviruses, including the often-mentioned variant sialodacryoadenitis virus (SDAV), are capable of inducing salivary and lacrimal gland (especially Harderian gland) disease or respiratory disease [1]. Disease presentations range from subclinical to overt. Early clinical signs may include blepharospasm and epiphora, but often the most profound sign is intermandibular swelling due to edema and inflammation of the submandibular salivary glands. Exophthalmus and chromodacryorrhea result from swelling of the Harderian gland, and unilateral or bilateral glaucoma/megaloglobus, hyphema, and corneal ulceration can occur secondarily. SDAV infection may potentiate clinical disease caused by *M pulmonis* or cilia-associated respiratory bacillus infections. Additionally, there are reports that SDAV infection may be associated with clinically significant reproductive disorders or decreased litter production, but this relationship remains unclear [2–4].

Pneumonia virus of mice and Sendai virus are paramyxoviruses that, as single agents, cause asymptomatic transient infections in rats. However, either virus may contribute to the severity of disease associated with bacterial respiratory pathogens, most notably *M pulmonis*.

Enteric disease in rats

Infectious diarrhea of infant rats, caused by an atypical rotavirus, was identified as the causative agent of a spontaneous outbreak of diarrhea in

suckling rats in 1984 [5]. Rats orally inoculated with rotavirus isolated from the spontaneous outbreak produced signs similar to those of natural infection, including diarrhea of 5 to 6 days' duration, growth retardation, and drying and flaking of the skin. Rats more than 2 weeks of age were generally considered resistant to clinical disease [5]. This disease has been experimentally replicated numerous times; however, no additional spontaneous outbreaks have been reported, thus its significance in the pet rat population is unknown.

Systemic disease in rats

Infections with Kilham's rat virus, a parvovirus, are most often asymptomatic. However, clinical disease has been reported on two occasions. In 1966, Kilham and Margolis [6] reported fetal resorption in pregnant females, runting and hepatitis in suckling rats, and the development of ataxia and cerebellar hypoplasia in a juvenile rat assumed to be infected at birth. In 1983, Coleman and colleagues [7] reported death, dyspnea, ruffled haircoats, hunched posture, lethargy, muscular weakness, weight loss, dehydration, swollen abdomens, and cyanotic scrotums in a naïve colony of rats that became naturally infected with Kilham's rat virus. At necropsy, hemorrhage and necrosis of the brain, testes, and epididymides were observed.

Other viral infections of rats

Rats have been reported to seroconvert to a number of other viruses; however, these infections rarely, if ever, result in the development of clinical disease. These include rat parvovirus, rat minute virus, H-1 virus, rat cardiovirus, reovirus, and adenovirus.

Viral infections of mice

Dermal disease in mice

Ectromelia virus is the causative agent of mousepox. The prevalence of this virus is very low in North America [8], but when present can spread rapidly with severe consequences. The disease was termed *infectious ectromelia* because of the high incidence of limb amputation due to self-trauma in surviving mice. Clinical signs in adult mice include foot swelling, lethargy, depression, and sudden death [9], with pocks occurring most commonly on the face, muzzle, feet, or abdomen [10]. Gross pathologic changes may include small intestinal mucosal erosions, necrosis and sloughing of distal portions of the tail and limbs, or massive splenic, lymph node, thymic, and hepatic necrosis [9]. Although the prognosis for mousepox is variable, mice generally recover completely from infection and do not serve as carriers [8].

Enteric disease in mice

Mouse hepatitis virus includes a number of strains of coronaviruses that vary in pathogenicity, organ tropism, and clinical disease presentation. Infections are most often asymptomatic, but can also manifest as diarrhea, sick rodent syndrome, decreased reproductive indices, and death [11]. Gross lesions include flaccid intestines with watery intestinal contents and multifocal hepatic necrosis (Fig. 2). All ages and strains of mice are susceptible to infection, but the severity of disease is inversely correlated to age. Neonatal mice (<7 days of age) can develop marked necrotizing enterocolitis with high mortality. Adults develop minimal lesions but are equally susceptible to viral infection, propagation, and shedding [12].

Naturally occurring clinical infections due to mouse rotavirus, the causative agent of epizootic diarrhea of infant mice, have not been reported since 1948 [12–14]. Those reports described high infant mortality with yellow feces staining the perineum, dehydration, cyanosis, and fine, dry scales appearing over the shoulders and dorsum [14]. There is controversy over whether the original outbreaks involved solely rotavirus infection or represented multifactorial disease, because many clinical signs—such as high infant mortality—have not been observed in more recent mouse rotavirus infections.

Respiratory disease in mice

Infection with Sendai virus, a labile but highly contagious parainfluenza virus, is usually clinically inapparent. There have been several reports of clinical disease in mice due to Sendai virus [15–18]; however, these reports are more than 20 years old, and it is possible that multiple factors contributed to the occurrence of clinical disease in these populations. Sendai

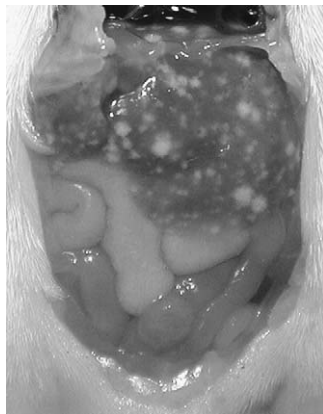


Fig. 2. Photograph of mouse infected with mouse hepatitis virus demonstrating multifocal hepatic necrosis and flaccid, fluid-content filled intestines.

virus infection will often predispose mice to clinical respiratory disease when complicated by *M pulmonis*. Antibiotic treatment may be helpful in such mixed infections.

Systemic disease in mice

Because of its polytropic nature, ectromelia virus can affect multiple systems in mice, causing conjunctivitis, blepharitis, and visceral lesions in addition to the more obvious dermal signs described previously. Polytropic strains of mouse hepatitis virus may also result in systemic disease because of their propensity to replicate in endothelium; however, this disease presentation rarely occurs in natural infections.

Neoplastic disease in mice

Murine leukemia viruses and murine mammary tumor viruses are oncogenic retroviruses of mice. Most retroviruses of mice are incorporated directly into the genome as proviruses and are transmitted genetically from generation to generation. There is no effective treatment for leukemia, lymphoma, and mammary tumors in mice. Most mammary tumors in mice are adenocarcinomas [19]. Mammary tumors can be surgically excised but are likely to recur with a poor prognosis.

Other viral infections in mice

Mice have been reported to seroconvert to a number of other viruses; however, these infections rarely result in the development of clinical disease. These include mouse parvovirus, mice minute virus, mouse poliovirus (Theiler's murine encephalomyelitis virus), pneumonia virus of mice, reovirus, adenovirus, polyoma virus, and cytomegalovirus.

Viral infections of guinea pigs

Respiratory disease in guinea pigs

Although there are viral causes of respiratory disease in guinea pigs, infections from other causes should be ruled out first, because they occur more frequently. For example, respiratory disease due to a bacterial infection is more common and may indicate an underlying stressor such as poor husbandry or nutritional management.

Adenovirus-induced respiratory disease with low morbidity and high mortality has been documented in guinea pigs [20–24]. Clinical signs range from those of pneumonia, including dyspnea, tachypnea, nasal discharge, rough hair coat, weight loss, and a hunched posture to acute death with no clinical signs. Postmortem evaluation of guinea pigs that die may reveal pulmonary consolidation and emphysema with petechiae, while histologic

examination may reveal a necrotizing bronchitis and bronchiolitis with basophilic intranuclear inclusion bodies. Currently, there is no commercially available serologic test for adenoviral infection in guinea pigs because the agent is difficult to grow in culture. However, a PCR assay has been described [25].

It should be noted that there are reports in the literature describing infection with adenoviruses in guinea pigs without any concurrent clinical signs [25,26]. In addition, in experimental infection of guinea pigs with adenovirus-infected crude suspensions of lung tissue, only newborn guinea pigs (2–3 days old) displayed any clinical signs [27], suggesting other factors such as altered immune status are needed to induce adenoviral respiratory disease.

Reproductive disease in guinea pigs

Natural infection with cytomegalovirus in guinea pigs is usually subclinical; overt disease is rare [28]. The true prevalence of infection is unknown and likely to be low. However, fetal death and sow mortality have been reported in association with cytomegaloviral infection [29]. Sows may be depressed at presentation and die 2 to 3 days later, and they may have late abortions, stillbirths, or fetal death shortly after birth. Other clinical signs may include weight loss and lymphadenopathy. The virus may be transmitted transplacentally or by way of infected saliva or urine and can persist in the guinea pig as a latent infection for years [30]. An immunocompromised state, pregnancy, or host factors may play a role in the severity of clinical signs [31].

Neoplastic disease in guinea pigs

Based on ultrastructural examination of affected tissues, type C retroviral infections have been associated with leukemia and lymphoma in the guinea pig. Clinical signs included lymphadenopathy and a white blood cell count from 25,000 to 250,000/mm³ [32]. Lymphoblastic cells were the predominant type and in some animals, infiltrated the liver and spleen, causing enlargement. The disease is often fatal within 2 to 5 weeks. Chemotherapy may be attempted, but there are no published reports of efficacy. The actual role of viral infections in naturally occurring guinea pig leukemia remains unknown. Several cases of naturally occurring leukemia were described in 1980 [33]; however, the role of viral infections in these cases was not determined.

Other viral infections in guinea pigs

There are rare reports of other guinea pig viral infections. These are usually subclinical in natural infection, produce clinical signs only in experimental infection, or the cause–effect relationship is still undetermined.

Although these agents are mentioned briefly here, clinicians should use caution when considering them as potential causes for disease. For example, although poliovirus infection has been suggested to cause hind limb lameness in pet shop guinea pigs, the clinical signs resolved with vitamin C treatment, thus complicating the association of poliovirus and disease [34]. Seroconversion to paramyxoviruses (caviid parainfluenza virus 3, Sendai virus, pneumonia virus of mice, simian virus 5) has been described in guinea pigs. No clinical signs have been seen with natural infections; however, histologic evidence of mild pneumonia has been identified in infected guinea pigs [35,36]. There is one report of a coronavirus-like particle causing a wasting syndrome in 3- to 4-week-old guinea pigs in which no other cause of disease could be identified [37]. Clinical signs included anorexia, weight loss, diarrhea, and death.

Viral infections of Syrian hamsters

Like other rodents, spontaneous viral infections that result in clinical signs are uncommon in the hamster. A discussion of hamster viral infections also must include lymphocytic choriomeningitis virus (LCMV). The latter is most important because of its zoonotic disease causing potential.

Systemic disease in hamsters

LCMV is an RNA virus of the arenavirus group. Natural infections are uncommon and asymptomatic; however, this virus can infect humans, and many reported human cases have been associated with infected hamsters, including pets (see the review by Parker [38]). This may be because infected hamsters shed larger amounts of virus in their urine and may develop a more persistent viruria when compared with other rodents [38,39].

The manifestation of disease in hamsters experimentally infected with LCMV may give some insight into natural disease progression [39]. Disease manifestation varies with virus and hamster strain and the age of inoculation [40,41]. Experimental in utero or perinatal infections produce a subclinical persistent infection or a chronic, progressive wasting disease associated with immune complex glomerulonephritis, vasculitis, and multi-organ inflammation. Approximately half of these infected hamsters cleared the infection. Of note, similar signs were seen in pups born to viremic mothers, which conceivably could occur in a natural setting. When infected as young adults, hamsters developed viruria and viremia for up to 6 months, but did not exhibit clinical signs [39].

Because of the zoonotic potential of LCMV, screening of breeding colonies supplying the pet trade may be warranted. Diagnosis of asymptomatic infections is based on detection of serum antibody to LCMV by way of ELISA. PCR analysis of tissues can also be used to confirm persistent or acute infections [42]. All hamsters that are infected

with LCMV or are at risk for infection should be euthanatized, and their cages and environment should be thoroughly disinfected.

Neoplastic disease in hamsters

Hamster polyomavirus is a DNA oncogenic virus that induces two neoplastic syndromes in hamsters depending on the age of the animal at the time of infection. Young hamsters develop a multicentric lymphoma involving mesenteric lymph nodes and abdominal viscera [43]. The other neoplastic syndrome occurs in adult hamsters and in endemically infected colonies, where viral infection results in trichoepitheliomas (Fig. 3) [30]. A presumptive diagnosis can be made on gross findings of epitheliomas or lymphomas. Confirmation requires histopathologic examination or PCR analysis of neoplastic tissue or kidney [43].

Musculoskeletal disease in hamsters

Recently, a case of naturally occurring spontaneous disease due to hamster parvovirus infection was described [44]. The disease manifested as decreased litter sizes with clinical signs in 2- to 4-week-old hamsters. Signs included dome-shaped crania, potbellied appearance, testicular atrophy and discoloration, malformation, and loss of incisor teeth. These signs were reproduced with experimental infection studies, and higher-dose experimental infections yielded a multisystemic hemorrhagic disease. A presumptive diagnosis may be made based on clinical signs and gross lesions.



Fig. 3. Photograph of adult hamster infected with hamster polyomavirus demonstrating multiple trichoepitheliomas. (Courtesy of Dr. Joe Simmons, DVM, PhD, West Point, Pennsylvania).

Confirmation requires identification of parvovirus through PCR analysis [44,45].

Respiratory disease in hamsters

Sendai virus and pneumonia virus of mice are serologically distinct RNA viruses of the paramyxoviridae family. Incidence of seroconversion to these viruses is high. However, clinical disease and gross and histologic lesions have not been reported in naturally occurring infections, although rhinitis, bronchitis, and neonatal deaths have been reported with experimental infections [46,47].

Hamsters may also seroconvert to a number of viral pathogens of rodents. These include reovirus-3 and simian virus 5 [30,38]. The significance of these findings is poorly understood and clinical signs associated with seroconversion have not been reported. Adenoviral [48] and cytomegaloviral [49] inclusions have also been identified in hamsters, but to date, have not been associated with clinical disease.

Viral infections of chinchillas

Neurologic disease in chinchillas

Only two cases of naturally occurring viral infection in the chinchilla have been reported in the literature [50,51]; both are thought to be due to herpes or a herpes-like virus. The first report describes an adult female chinchilla that presented with a history of acute death. Necrotic foci with inclusion bodies were found in the spleen, pineal body, adrenal glands, and medulla of the brain. Electron microscopy was highly suggestive of a herpes-like viral infection based on morphology of the virus and distribution of lesions. In the second case, a 1-year-old male chinchilla with conjunctivitis, uveitis, and bilateral mydriasis for 2 weeks began to show neurologic signs, including disorientation, seizures, and biting at the cage bars, leading to atonic recumbancy. The chinchilla was treated with antibiotics and electrolytes but was euthanized after 3 weeks because of a poor prognosis. Significant pathologic findings included a unilateral purulent rhinitis, keratouveitis, retinopathy, optical neuritis, nonsuppurative meningitis, and polioencephalitis with neuronal necrosis. Intranuclear inclusion bodies were found in many areas of the brain and the nasal cavity. Electron microscopy, immunohistochemical staining, virus isolation, and nucleic acid sequence analysis confirmed an infection with human herpes simplex virus type 1, a common virus carried by humans causing oral or labial lesions that can lead to encephalitis. The authors proposed a primary ocular infection in the chinchilla potentially from contact to a human with a herpetic lesion that subsequently spread to the central nervous system. Although this route of transmission could not be confirmed, this raises the question of whether chinchillas can serve as reservoirs for herpes simplex virus.

Viral infections of prairie dogs

Systemic disease in prairie dogs

Until recently, there were no reports in the literature describing naturally occurring viral infections in prairie dogs. However, in May and June of 2003, an outbreak of monkeypox infection that spread from infected prairie dogs to humans occurred for the first time in the United States affecting multiple Midwestern states. Prairie dogs were exposed to monkeypox when housed in the same pet store as an infected Gambian giant rat and dormice [52] that were imported from Ghana. Animal-to-animal transmission of monkeypox was likely from respiratory and mucocutaneous routes [53]. Clinical signs of monkeypox infection in prairie dogs include anorexia, wasting, sneezing, coughing, lymphadenopathy, conjunctivitis, ocular and nasal discharge, papular skin lesions, tongue ulceration, and death [53,54].

Monkeypox is a member of the orthopoxvirus group that includes smallpox virus (variola) and vaccinia virus (the virus used in the smallpox vaccine). This viral disease occurs mostly in Africa, and many animal species are capable of being infected, including captive African monkeys (in which the virus was first recognized). In humans, clinical signs of monkeypox are similar to those of smallpox but are usually milder and include vesiculopustular skin lesions, fever, headache, muscle aches, lymphadenopathy, and lethargy [54]. Transmission from animals to people can occur via bite wounds, scratches, or exposure to infected blood, body fluids, or skin lesions. In people, mortality rates are reported to be from 1% to 10%; however, no human deaths occurred in the United States outbreak. The smallpox vaccine may be protective against monkeypox. No further cases of monkeypox in humans have been reported since June 22, 2003 [54].

There are no specific treatments for prairie dogs infected with monkeypox. If a case of monkeypox is suspected in an animal, precautions should be taken to prevent potential human exposure, including prior communication with the veterinary clinic before admitting the animal. Owners should isolate the potentially infected animal and wash hands, clothing, and any materials that have come in contact with the animal with household detergents or bleach. State or local health departments should be notified, and sample submission for diagnostic testing must be coordinated with state health departments. The Centers for Disease Control and Prevention (CDC) recommend that veterinarians should not perform biopsies or necropsies of animals with suspected monkeypox infection. Animals with suspected or confirmed infection with monkeypox should be humanely euthanized. The CDC and the US Food and Drug Administration currently prohibit any sale, interstate movement, or release into the environment of prairie dogs within the United States. More information and guidelines can be found on the CDC Web site (www.cdc.gov/ncidod/monkeypox/index.htm).

Viral infections of hedgehogs

Neoplastic disease in hedgehogs

Retroviruses have been linked to multicentric skeletal sarcomas in African pygmy hedgehogs [55]. Retroviruses are often incorporated directly into the genome as proviruses, rendering attempts for diagnosis impractical. There are no established protocols for treatment of neoplasia in hedgehogs.

Systemic disease in hedgehogs

There has been one report of herpes simplex virus infection in an African pygmy hedgehog that was undergoing corticosteroid therapy for a prolapsed intervertebral disc [56]. Clinical signs were confined to anorexia. On necropsy, the liver was stippled with punctate off-white foci.

Viral infections of gerbils and sugar gliders

No naturally occurring viral diseases have been reported in gerbils or sugar gliders to date.

Viral zoonoses of small mammals

LCMV can infect many species, including the small mammals discussed in this article. If LCMV infection is suspected in a small mammal, those in contact with the potentially infected animal should seek medical attention immediately. Humans may become infected with LCMV from bite wounds or exposure to contaminated feces or urine, and may experience flu-like symptoms or rarely nonsuppurative meningitis as a result (see www.cdc.gov/ncidod/dvrd/spb/mnpages/dispages/lcmv.htm for more information regarding LCMV infection in people). Hamsters are the most common source of human exposure, most likely because they maintain a more persistent viremia than other mammals. However, it must be stressed that natural infection in hamsters and other small mammals is uncommon, and most recent human LCMV exposures have been linked to exposure in a laboratory setting either while handling experimentally infected immunocompromised research mice or through bench accidents.

Several species of wild rodents serve as reservoirs for hantavirus infection and are therefore considered a potential zoonotic hazard to humans. However, the likelihood of pet rodents acquiring this infection is extremely low. Simmons and Riley [36] offer an excellent review of hantavirus infections in rodents.

Rabies has been documented in rodents and other small mammals, but these numbers account for less than 1% of reported rabies cases in the United States and involve only feral rodents (<http://www.cdc.gov/ncidod/>

[dvr/d/rabies/Epidemiology/Epidemiology.htm](#)). To illustrate this fact, between the years 1985 and 1994, there were 4 reported rabies cases in mice and rats, and these numbers were far surpassed by reports of the disease in feral woodchucks, raccoons, and rabbits [57]. Transmission of rabies to small mammals is extremely unlikely, because captive-bred animals have minimal exposure to the disease; furthermore, in the wild, when a small mammal comes in contact with a rabies-infected predator, the small mammal is more likely to be consumed than to have the disease transmitted during the encounter. Thus, rabies should be considered of very minimal concern to owners of the small mammals covered in this report.

Summary

Most viral infections in small mammals are transient and rarely produce clinical signs. When clinical signs do appear, they are often of a multifactorial etiology such as respiratory infection with Sendai virus and the bacteria *M pulmonis* in rodents. Diagnosis is generally made based on clinical signs, while therapy involves treatment for concurrent bacterial infections and supportive care. Small mammals may carry zoonotic viruses such as LCMV, but natural infections are uncommon. Viral diseases are rare (or largely unknown) for hedgehogs, chinchillas, and prairie dogs, while no known naturally occurring, clinically relevant viral diseases exist for gerbils and sugar gliders. This article is intended to aid the clinician in identifying viral infections in small mammals and to help determine the significance each virus has during clinical disease.

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Further readings

- Baker DG. Natural pathogens of laboratory animals. Washington, DC: ASM Press; 2003.
- Committee on infectious disease of mice and rats. Infectious diseases of mice and rats. Washington, DC: National Academy Press; 1991.
- Fox JG, Anderson LC, Loew FM, Quimby FW. Laboratory animal medicine. 2nd ed. Amsterdam: Academic Press; 2002.
- Harkness JE, Wagner JE. The biology and medicine of rabbit and rodents. Baltimore (MD): Lea & Febiger; 1995.
- Percy DH, Barthold SW. Pathology of laboratory rodents & rabbits. 2nd ed. Ames (IA): Iowa State University Press; 2001.
- Quesenberry KE, Carpenter JW. Ferrets, rabbits, and rodents, clinical medicine and surgery. 2nd ed. St. Louis (MO): Saunders; 2004.

References

- [1] Percy DH, Barthold SW. Rat. Viral infections. In: Percy DH, Barthold SW, editors. Pathology of laboratory rodents & rabbits. 2nd ed. Ames (IA): Iowa State University Press; 2001. p. 108–20.
- [2] Macy JD Jr, Weir EC, Barthold SW. Reproductive abnormalities associated with a coronavirus infection in rats. *Lab Anim Sci* 1996;46:129–32.
- [3] Utsumi K, Maeda K, Yokota Y, et al. Reproductive disorders in female rats infected with sialodacryoadenitis virus. *Jikken Dobutsu* 1991;40:361–5.
- [4] Utsumi K, Yokota Y, Ishikawa T, et al. Reproductive disorders in female SHR rats infected with sialodacryoadenitis virus. *Adv Exp Med Biol* 1990;276:525–32.
- [5] Vonderfecht SL, Huber AC, Eiden J, et al. Infectious diarrhea of infant rats produced by a rotavirus-like agent. *J Virol* 1984;52:94–8.
- [6] Kilham L, Margolis G. Spontaneous hepatitis and cerebellar “hypoplasia” in suckling rats due to congenital infections with rat virus. *Am J Pathol* 1966;49:457–75.
- [7] Coleman GL, Jacoby RO, Bhatt PN, et al. Naturally occurring lethal parvovirus infection of juvenile and young-adult rats. *Vet Pathol* 1983;20:49–56.
- [8] Percy DH, Barthold SW. Mouse/viral infections. In: Percy DH, Barthold SW, editors. Pathology of laboratory rodents & rabbits. 2nd ed. Ames (IA): Iowa State University Press; 2001. p. 23–5.
- [9] Committee on Infectious Disease of Mice and Rats. Lindsey JR, Boorman GA, Collin MJ Jr, et al. (Multiple systems). In: Infectious diseases of mice and rats. Washington, DC: National Academy Press; 1991. p. 236–9.
- [10] Fenner F. Mousepox (infectious ectromelia): past, present, and future. *Lab Anim Sci* 1981; 31:553–9.
- [11] Homberger FR, Zhang L, Barthold SW. Prevalence of enterotropic and polytropic mouse hepatitis virus in enzootically infected mouse colonies. *Lab Anim Sci* 1998;48:50–4.
- [12] Committee on Infectious Disease of Mice and Rats. Lindsey JR, Boorman GA, Collin MJ Jr, et al. (Digestive system). In: Infectious diseases of mice and rats. Washington, DC: National Academy Press; 1991. p. 102–17.
- [13] Cheever FS, Mueller JH. Epidemic diarrheal disease of suckling mice. III. The effect of strain, litter, and season upon the incidence of disease. *J Exp Med* 1948;88:309–16.
- [14] Cheever FS, Mueller JH. Epidemic diarrheal disease of suckling mice. I. Manifestations, epidemiology, and attempts to transmit the disease. *J Exp Med* 1947;85:405–16.
- [15] Zurcher C, Burek JD, Van Nunen MC, et al. A naturally occurring epizootic caused by Sendai virus in breeding and aging rodent colonies. I. Infection in the mouse. *Lab Anim Sci* 1977;27:955–62.
- [16] Bhatt PN, Jonas AM. An epizootic of Sendai infection with mortality in a barrier-maintained mouse colony. *Am J Epidemiol* 1974;100:222–9.
- [17] Parker JC, Reynolds RK. Natural history of Sendai virus infection in mice. *Am J Epidemiol* 1968;88:112–25.
- [18] Itoh T, Kagiya N, Iwai H, et al. Sendai virus infection in a small mouse breeding colony. *Nippon Juigaku Zasshi* 1978;40:615–8.
- [19] Donnelly TM. Disease problems of small rodents. In: Quesenberry KE, Carpenter JW, editors. Ferrets, rabbits, and rodents: clinical medicine and surgery. 2nd ed. St. Louis (MO): Saunders; 2004. p. 299–315.
- [20] Brennecke LH, Dreier TM, Stokes WS. Naturally occurring virus-associated respiratory disease in two guinea pigs. *Vet Pathol* 1983;20:488–91.
- [21] Naumann S, Kunstyr I, Langer I, et al. Lethal pneumonia in guinea pigs associated with a virus. *Lab Anim* 1981;15:235–42.
- [22] Harris IE, Portas BH, Goydich W. Adenoviral bronchopneumonia of guinea pigs. *Aust Vet J* 1985;62:317.

- [23] Feldman SH, Richardson JA, Clubb FJ Jr. Necrotizing viral bronchopneumonia in guinea pigs. *Lab Anim Sci* 1990;40:82–3.
- [24] Eckhoff G, Mann P, Gaillard E, et al. Naturally developing virus-induced lethal pneumonia in two guinea pigs (*Cavia porcellus*). *Contemp Top Lab Anim Sci* 1998;37:54–7.
- [25] Butz N, Ossent P, Homberger FR. Pathogenesis of guinea pig adenovirus infection. *Lab Anim Sci* 1999;49:600–4.
- [26] Crippa L, Giusti AM, Sironi G, et al. Asymptomatic adenoviral respiratory tract infection in guinea pigs. *Lab Anim Sci* 1997;47:197–9.
- [27] Kunstyr I, Maess J, Naumann S, et al. Adenovirus pneumonia in guinea pigs: an experimental reproduction of the disease. *Lab Anim* 1984;18:55–60.
- [28] Van Hoosier GL Jr, Giddens WE Jr, Gillett CS, et al. Disseminated cytomegalovirus disease in the guinea pig. *Lab Anim Sci* 1985;35:81–4.
- [29] Motzel SL, Wagner JE. Diagnostic exercise: fetal death in guinea pigs. *Lab Anim Sci* 1989;39:342–4.
- [30] Percy DH, Barthold SW. Guinea pig/viral infections. In: Percy DH, Barthold SW, editors. *Pathology of laboratory rodents & rabbits*. 2nd ed. Ames (IA): Iowa State University Press; 2001. p. 213–6.
- [31] Harkness JE, Murray KA, Wagner JE. Biology and diseases of guinea pigs. In: Fox JG, Anderson LC, Loew FM, editors. *Laboratory animal medicine*. 2nd ed. Amsterdam: Academic Press; 2002. p. 203–47.
- [32] Van Hoosier GL Jr, Robinette LR. Viral and chlamydial diseases. In: Wagner JE, Manning PJ, editors. *The biology of the guinea pig*. New York: Academic Press; 1976. p. 137–52.
- [33] Hong CC, Liu PI, Poon KC. Naturally occurring lymphoblastic leukemia in guinea pigs. *Lab Anim Sci* 1980;30:222–6.
- [34] Hansen AK, Thomsen P, Jensen HJ. A serological indication of the existence of a guinea pig poliovirus. *Lab Anim* 1997;31:212–8.
- [35] Blomqvist GA, Martin K, Morein B. Transmission pattern of parainfluenza 3 virus in guinea pig breeding herds. *Contemp Top Lab Anim Sci* 2002;41:53–7.
- [36] Simmons JH, Riley LK. Hantaviruses: an overview. *Comp Med* 2002;52:97–110.
- [37] Jaax GP, Jaax NK, Petrali JP, et al. Coronavirus-like virions associated with a wasting syndrome in guinea pigs. *Lab Anim Sci* 1990;40:375–8.
- [38] Parker JC. Viral diseases. In: Van Hoosier GLJ, McPherson CW, editors. *Laboratory hamsters*. Orlando (FL): Academic Press; 1987. p. 95–111.
- [39] Parker JC, Igel HJ, Reynolds RK, et al. Lymphocytic choriomeningitis virus infection in fetal, newborn, and young adult Syrian hamsters (*Mesocricetus auratus*). *Infect Immun* 1976;13:967–81.
- [40] Genovesi EV, Peters CJ. Susceptibility of inbred Syrian golden hamsters (*Mesocricetus auratus*) to lethal disease by lymphocytic choriomeningitis virus. *Proc Soc Exp Biol Med* 1987;185:250–61.
- [41] Genovesi EV, Johnson AJ, Peters CJ. Susceptibility and resistance of inbred strains of Syrian golden hamsters (*Mesocricetus auratus*) to wasting disease caused by lymphocytic choriomeningitis virus: pathogenesis of lethal and non-lethal infections. *J Gen Virol* 1988;69:2209–20.
- [42] Besselsen DG, Wagner AM, Loganbill JK. Detection of lymphocytic choriomeningitis virus by use of fluorogenic nuclease reverse transcriptase-polymerase chain reaction analysis. *Comp Med* 2003;53:65–9.
- [43] Simmons JH, Riley LK, Franklin CL, et al. Hamster polyomavirus infection in a pet Syrian hamster (*Mesocricetus auratus*). *Vet Pathol* 2001;38:441–6.
- [44] Besselsen DG, Gibson SV, Besch-Williford CL, et al. Natural and experimentally induced infection of Syrian hamsters with a newly recognized parvovirus. *Lab Anim Sci* 1999;49:308–12.

- [45] Besselsen DG, Besch-Williford CL, Pintel DJ, et al. Detection of newly recognized rodent parvoviruses by PCR. *J Clin Microbiol* 1995;33:2859–63.
- [46] Profeta ML, Lief FS, Plotkin SA. Enzootic sendai infection in laboratory hamsters. *Am J Epidemiol* 1969;89:316–24.
- [47] Percy DH, Barthold SW. Hamster/viral infections. In: Percy DH, Barthold SW, editors. *Pathology of laboratory rodents & rabbits*. 2nd ed. Ames (IA): Iowa State University Press; 2001. p. 169–74.
- [48] Gibson SV, Rottinghaus AA, Wagner JE, et al. Naturally acquired enteric adenovirus infection in Syrian hamsters (*Mesocricetus auratus*). *Am J Vet Res* 1990;51:143–7.
- [49] Lussier G. Murine cytomegalovirus (MCMV). *Adv Vet Sci Comp Med* 1975;19:223–47.
- [50] Wohlsein P, Thiele A, Fehr M, et al. Spontaneous human herpes virus type 1 infection in a chinchilla (*Chinchilla laniger f. dom.*). *Acta Neuropathol (Berl)* 2002;104:674–8.
- [51] Goudas P, Giltoy JS. Spontaneous herpes-like viral infection in a chinchilla (*Chinchilla laniger*). *Wildl Dis* 1970;6:175–9.
- [52] Centers for Disease Control and Prevention. Update: multistate outbreak of monkeypox—Illinois, Indiana, Kansas, Missouri, Ohio, and Wisconsin, 2003. *MMWR* 2003;52:642–6.
- [53] Guarner J, Johnson BJ, Paddock CD, et al. Monkeypox transmission and pathogenesis in prairie dogs. *Emerg Infect Dis* 2004;10:426–31.
- [54] Reed KD, Melski JW, Graham MB, et al. The detection of monkeypox in humans in the Western Hemisphere. *N Engl J Med* 2004;350:342–50.
- [55] Peauroi JR, Lowenstine LJ, Munn RJ, et al. Multicentric skeletal sarcomas associated with probable retrovirus particles in two African hedgehogs (*Atelerix albiventris*). *Vet Pathol* 1994;31:481–4.
- [56] Allison N, Chang TC, Steele KE, Hilliard JK. Fatal herpes simplex infection in a pygmy African hedgehog (*Atelerix albiventris*). *J Comp Pathol* 2002;126:76–8.
- [57] Childs JE, Colby L, Krebs JW, et al. Surveillance and spatiotemporal associations of rabies in rodents and lagomorphs in the United States, 1985–1994. *J Wildl Dis* 1997;33:20–7.