charles river

RESEARCH MODELS

Murine Chapparvovirus (MuCPV), aka Mouse Kidney Parvovirus (MKPV)

Classification

ssDNA virus, nonenveloped

Family

Parvoviridae

Genus

Provisionally named *Chapparvovirus* by ICTV[1], genetically and antigenically distinct from *Protoparvovirus* (MVM, MPV).

Affected species

Wild and laboratory mice are affected.

Frequency

It is moderately prevalent in laboratory and wild mice[2-4].

Transmission

MuCPV is likely transmitted through both feces and urine[2].

Clinical Signs and Lesions

Infection by MuCPV results in intranuclear inclusion bodies in renal tubular epithelial cells with variable incidence based on immune status[2]. Intranuclear inclusion bodies of mouse renal tubular epithelium (inclusion body nephropathy, IBN) have been anecdotally observed by veterinary pathologists for more than 40 years[2,5]. Their occurrence has been reported as low in both immunocompetent and immunocompromised mice[6,7]. Clinical disease has only been reported in immunocompromised models[2].

Clinical disease from MuCPV has not been described in immunocompetent mice, although histopathology has infrequently revealed intranuclear inclusion bodies of renal tubular epithelial cells[7]. Adult immunodeficient mice may develop chronic renal disease indicated by shrunken, pale, and pitted kidneys. Infection of immunocompromised mice varies based on level immune dysfunction and ranges from mild IBN to intranuclear inclusions, tubular degeneration, and necrosis. Infection may also progress onto chronic renal failure with accompanying renal fibrosis. Mild, subclinical IBN has been reported in athymic nude mice with limited immune dysfunction. The most severe pathology progressing to renal failure has been associated with the presence of increased MuCPV viral DNA in severely immunocompromised models such as Rag-/-, SCID-/-, and NSG models[2].

Diagnosis

Diagnosis of MuCPV is usually made using PCR performed on the kidney, feces, and environmental samples.
The limited literature describing infection and disease progression indicating the ability to detect infection may be delayed. Kidneys show a persistent presence of infection by 61 days in an endemically infected colony while consistent viremia was delayed until approximately 100 days of life[2].

Additionally, a survey of wild mice showed that the prevalence of infection by MuCPV increased with age. Only 5% of juveniles were infected, compared to 62% of adult mice[3].

Experience indicates PCR testing of environmental samples such as open-top caging environment and exhaust air dust (EAD®) of appropriate racks, feces, and kidneys are good sample types to determine the detection of MuCPV. PCR is a reliable method of detection in samples from older, breeder age mice. Urine may also be submitted from immunodeficient mice. Testing young, soiled bedding sentinels may not reliably detect the virus.

MuCPV is highly divergent from previously known mouse parvoviruses (protoparvoviruses). It is not detected by currently available serological and PCR tests for mouse parvovirus (MPV) and minute virus of mice (MVM)[2,8]. Serological assays specific to MuCPV have not been reported.

Interference with Research

Parvoviruses replicate in cells undergoing active division; therefore, their presence results in modification of cellular physiology. Mouse parvovirus-1 (MPV-1) infection has been associated with modification of immune function in mice, although previously identified rodent parvoviruses, such as MPV-1, have been clinically silent. The effects of MuCPV infection have not been fully described. Infection by MuCPV has shown to induce changes in rental tubular epithelial cells infrequently in immunocompetent mouse models while infection in severely immunocompetent models may progress to chronic renal failure, morbidity, and mortality[1-3].

Prevention and Treatment

MuCPV is a newly identified virus and pathobiology of the agent hasn't been fully described. Previously known mouse parvoviruses (e.g., MPV) have been shown to contaminate animal biological products such as cell lines, transplantable tumors, and blood products, which have been shown to transmit the virus. Since MuCPV has recently been identified, biological sources for transmission of the virus have not been fully determined. The limited work with MuCPV has shown it can reside in both kidney and liver tissues. It is also reasonable to assume that additional tissues, as well as transplantable tumors from infected mice, may be a source of transmission. Cell lines, transplantable tumors, and other biological products should be tested for MuCPV using PCR. Wild mice can serve as a reservoir for MuCPV, so access of wild rodents to mouse colonies and their supplies must be prevented[3].

Parvoviruses are generally persistent and environmentally stable. Contamination of shared equipment, supplies, and surfaces must be addressed in any effort to eliminate them. Microisolation housing, strict aseptic husbandry, and experimental procedures are critical in limiting the spread of parvoviruses within facilities. Aggressive chemical decontamination using detergents and oxidizing disinfectants is advised, along with autoclaving or cold sterilization of materials in direct contact animals.

Response to identification of infected animals will depend on their value, possibility of replacement, research, and ability to maintain replacement animals free from infection in the future. If the virus needs to be eliminated from a research colony, depopulation, thorough cleaning and sanitization of all aspects of the animal room, and restocking are recommended. Rederivation by embryo transfer has been described in the literature[2]. Anecdotal evidence suggests hysterectomy rederivation should be effective in removing MuCPV from infected mouse lines.

References

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