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Expert Information

From the Working Group on Hygiene

Implication of infectious agents on results of animal experiments

Mouse Hepatitis Virus

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Contents

Mouse Hepatitis Virus / Murine Hepatitis Virus (MHV)

Background

- First reported in 1949 as JHM strain (named after J. Howard Mueller at Harvard University) of the mouse hepatitis virus[1,2]
- Family *Coronaviridae*, genus *Coronavirus*[1,3]
- Enveloped single-stranded RNA virus, with a corona of surface projections (spikes)[1,3]
- Antigenetically related to rat coronaviruses and other coronaviruses of pigs, cattle and humans[3]
- Different strains or isolates of MHV have been described, of which the following have been studied most extensively and are considered the prototype strains: MHV 1, -2, -3, -4 (=JHM); A59 and S 1[1]; MHV-3 is regarded as the most virulent.[3]
- MHV mutants rapidly and new sub strains are constantly evolving.[3]
- Among the three major structural proteins of MHV, nucleoprotein N is moderately and membrane glycoprotein M highly conserved among MHV strains, whereas spike glycoprotein S is very variable.[4]
- The external domain of glycoprotein S is subdivided and the S1 domain contains hypervariable regions that contribute to antigenic variation, tissue tropism, and virulence; it binds to specific host cell receptors, induces neutralizing antibodies, binds antibodies and solicits cell-mediated immunity.[1]

Prevalence

- In the 1980th the prevalence of MHV in laboratory mice worldwide was 80%.[1]
- In a study conducted in 2009 by one of the leading commercial rodent diagnostic laboratories, 1.57% of the mouse serum samples submitted from institutions in North America were positive for MHV antibodies.[5]
- In Europe, prevalence rates in mouse colonies in 2009 ranged from 3.25% [5] to 5.5%.[6]

Host species

- Natural host of the virus is the mouse (*Mus musculus*).[1,3]
- MHV can be found in wild and laboratory mice.[3,7]
- MHV is one of the most prevalent viral pathogens in research mouse colonies.[3]

Properties

- MHV might remain infective in mouse colonies for several days, at low humidity (20% rH) or low temperature (4°C) on surfaces even for weeks.[3]
- MHV is extremely contagious; it is shed in feces and nasopharyngeal secretions and appears to be transmitted via direct contact, aerosol and fomites.[1,3]
- MHV is a common contaminant of transplantable tumors and cell lines.[1,3]
- MHV is sufficiently transferred to sentinel mice via soiled bedding,[8,9] especially the enterotropic virus strains.[2,3]
- Persistent transmission of MHV from MHV-seropositive transgenic mice for over two years was described[8]

- Another study reported the persistent transmission of MHV from tumor necrosis factor (TNF) knockout mice B.6129S1-Tnf^{tm1Lj}(TNF-/-) over a period of five month; the virus was transferred to nude sentinels by direct contact and also in some cases to immunocompetent mice after only 24 h of direct contact.[10]
- Transmission of MHV-Y-inoculated BALB/c mice to sentinels was described after one-day contact with the mice or one-week contact with soiled bedding.[11]

Susceptibility

- Wild and laboratory mice[3,7]
- All ages and strains are susceptible to active infection, but disease is mainly age related (enterotropic strains in neonatal mice can cause severe enterocolitis with high mortality whereas adult mice develop minimal lesions).[3]
- Differences depending on mice strains, e.g. BALB/c mice highly susceptible to enterotropic MHV, developing early and severe disease, while SJL mice more resistant (to certain virus strains)[3,12,13]
- The resistance in SJL mice is not caused by receptor dysfunction.[14]
- Mouse genotypes susceptible to disease caused by one MHV strain may be resistant to disease caused by another virus strain.[3]
- Recovered mice are resistant to reinfection with the same but remain susceptible to infection with another MHV strain.[3]
- Immunodeficient mice such as Foxn1^{nu} or Prkdc^{scid} mice cannot clear the virus.[3]
- Other genetically modified mouse strains with deficits in antiviral response may develop persistent MHV infection.[3,8]
- Suckling rats were susceptible for experimental infection with MHV including seroconversion, but did not develop clinical disease.[3,15]
- Also, deer mice (*Peromycus maniculatus*) showed seroconversion but no clinical disease after experimental infection.[3]

Organotropism

- Two biotypes, based on primary organotropism: enterotropic strains and respiratory (polytropic) strains; also, intermediate forms exist[3,16]
- Respiratory tropism: initial infection of nasal mucosa, may disseminate via blood and lymphatic strains to a variety of other organs (polytropic)[3]
- Secondary replication in vascular epithelium and parenchymal tissues, causing disease of brain, liver, lymphoid organs, bone marrow and others[3]
- Respiratory (polytropic) strains include MHV-1, MHV-2, MHV-3, A59, S and JHM.[3]
- Some MHV-strains show a strong neurotropism with demyelinating or nondemyelinating effects; demyelinating virus strains are e.g. MHV-A59 and JHM.[17,18,19]
- Enterotropic strains: selective infection of intestinal mucosa with no or minimal dissemination to other organs such as mesenteric lymph nodes or liver[3]; virus strains related to enteric disease pattern are e.g. MHV-D or Y[1]
- Homberger et al. found the enterotropic MHV-biotypes to be predominant in contemporary laboratory mouse colonies.[16]

Clinical disease

- Most MHV infections in immunocompetent mice are subclinical.[1,3]
- Clinical disease can occur when the virus is introduced to a naïve population and can spready rapidly. [3]
- Clinical signs depend on virus and mouse strains and are most evident in infant mice.[3]
- Clinical signs can include diarrhea, poor growth, lassitude and death.[3]
- Flaccid paralysis of hindlimbs, convulsions and circling are possible neurologic signs caused by some virus strains.[3]
- Subclinical infection can be activated by experimental procedure such as thymectomy, whole body irradiation, treatment with chemotherapeutic agents, halothane anaesthesia or by coinfection with other pathogens (e.g. K-virus, *Eperythrozoon coccoides*).[3]
- Duration of infection and disease manifestation vary depending on mouse and virus strain.[3,12]
- Polytropic and enterotropic MHV infections are self-limiting in immunocompetent mice; elimination of virus begins ca. 1 week and is complete approx. 3-4 weeks after infection.[3]
- Humoral and cellular immunity needed; especially functional T-cells required for immune defence[3]
- A study of Compton et al. in B cell-deficient (μMT) and T cell-deficient (Tcrβδ^{-}) mice supported the concept that B cells promote clearance of enterotropic MHV-Y strains from intestinal mucosa and that T cells are required to prevent dissemination of enterotropic MHV strains from gastrointestinal tract.[20]
- Athymic mice can develop severe generalized disease with emaciation leading to debility and death (wasting disease) with diarrhea as the leading clinical sign in cases with enterotropic MHV strains.[1]

Pathology

- In most natural infections gross lesions are not present or transient.[3]
- Gross findings in neonates with clinical signs include dehydration, emaciation, empty stomach (in contrast to EDIM)[3]
- Furthermore, distended intestine filled with watery to mucoid yellowish contents can be found, haemorrhage or rupture of the intestine can occur.[3]
- BALB mice experimentally inoculated with MHV at the age of 24 h developed severe necrotizing enterocolitis.[12]
- Necrotic foci of liver and thymus involution can be seen in susceptible mice depending on virus strain.[3]
- Lesions in the liver can be combined with jaundice and haemorrhagic peritoneal exsudate; compensatory splenomegaly possible.[3]
- Experimental infection of C57BL/6 mice with MHV-A59 and recombinant virus (MHV-inf-1) caused acute hepatitis, acute meningoencephalitis, and chronic demyelination similar to the human disease multiple sclerosis.[21]

Morbidity and mortality

- Depending on virus and mouse strain, see also above under Clinical disease

- Mortality can reach 100% in infant mice infected with virulent enterotropic MHV strains.[1,3]
- Infection with virulent polytropic strains is often rapidly fatal in immunodeficient mice (e.g. Foxn1^{nu} or Prkdc^{scid}).[3]
- Experimental infection with high doses of MHV-A59 and recombinant virus (MHV-inf-1) caused high mortality rates up to 100% in 4-week-old C57BL/6 mice.[21]

Zoonotic potential

- No data

Interference with research

Oncology

- Contamination of transplantable tumors[22]
- Abnormal tumor invasion pattern, abnormal tumor passage intervals, spontaneous regression or oncolysis of normally stable tumors[23,24,25,26,27]
- Rejection of human xenografts[28]
- Altered response to chemical carcinogens[29]

Teratology

- No data

Infectiology / Interactions with other infectious agents

- Reduced susceptibility for other viral infections (PVM, Sendai)[30]
- Potentiation of subclinical MHV infections by urethane and methylformamide[24], halothane[31], transplantation of tumors[32]
- Enhances resistance to *Salmonella typhimurium* in mice by inducing suppression of bacterial growth[33]
- *Helicobacter hepaticus* appeared to reduce the severity of MHV-induced lesions during the acute phase of (experimental) infection in gamma interferon-deficient mice.[34]
- Can increase susceptibility to other infectious agents (and vice versa) such as *Eperythrozoon coccoides*, K-virus, *Schistosoma mansoni*[1]
- Alters the course of concurrent viral infections due to pneumonia virus of mice or Sendai virus; diminishes the production of interferon in response to infection with sendai virus[1,30,35]

Immunology

- MHV has the capacity to grow in several types of immune cells.[36]
- Immunodepression and immunostimulation depending on the time of infection[35] MHV replicates in macrophages and with or without lysis in both B and T lymphocytes.[37,38,39]
- Enhanced and suppressed macrophage function[40,41] and dysfunction of T and B cells[42,43,44,45] was observed.
- Activation of natural killer (NK) cells and alteration of the interferon responsiveness of infected mice[35,46]

- Reduced levels of cytokines, interleukins and gamma interferon in spleen cells[44] Recovered mice have complete or partial protection against T cell dysfunctions when re-infected with different strains of MHV.[47]
- Macrophage dysfunctions continue in MHV-recovered mice.[40]
- MHV infection can durably modify unrelated T cell responses that are initiated at the time of infection.[48]
- Permanent decrease of skin graft rejection and T cell dependent antibody responses after recovering from MHV-A59 infection[49]
- Enhancement of concomitant autoimmune reactions[50]

Toxicology

- No data

Physiology

- Alteration of liver enzyme levels, patterns of protein synthesis and other biochemical markers[29,51]
- Induces production of alpha-fetoprotein[52] and increase of iron uptake[53]
- Changes in peripheral blood such as anemia, thrombocytopenia, leukopenia and increased monocyte procoagulant activity[54,52]
- Decrease of the incidence of diabetes in non-obese diabetic mice[55]

Cell biology

- No data

Assisted reproductive technology

- Persistent contamination of embryonic stem cells without diminishing their pluripotency[56]

Special considerations

- Relatively resistant to repeated freezing and thawing, heating and acid pH[1,3]
- Sensitive to drying and disinfectants[3]
- All known MHV strains cross react in serological tests such as ELISA and IFA; the antibody response can depend on the mouse strain and genotype; for example, DBA/2 mice produce less antibodies than C57BL/6; the latter are, therefore, good sentinels regarding MHV detection.[3]
- Antibody titres are detectable from ca. 2 weeks after initial infection and remain high for at least 6 month.[3]
- Antibodies are transferred to pups in utero and via milk.[4]
- Maternal antibodies against MHV (IgG) may be present in pups up to the age of 10 weeks.[3,4]
- The best means of MHV control is to prevent its entry into a facility; if infection occurs, caesarean rederivation or embryo transfer can be used, cross-fostering was also reported.[3,57]
- A review from 2001 describes that inoculation of mice with most neurotropic strains of MHV results in an immune response-mediated demyelinating disease that serves as an excellent animal model for the human disease multiple sclerosis; it was also shown

that either virus-specific $CD4^+$ or $CD8^+$ T cells are able to mediate the demyelination; there is an association between viral persistence in the CNS and chronic immune stimulation, which ultimately results in neuropathology.[13]

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