Mouse Hepatitis Virus

Host species

- mouse

Organotropism

- polytropic strains: liver, brain, lymphoid tissue, (other organs)
- enterotropic strains: intestine, lymphoid tissue

Clinical disease

- inapparent in immunocompetent adults
- diarrhea and death in neonates (epizootic infection)
- wasting disease in immunodeficient mice

Pathology

- polytropic strains: acute necrosis and syncytia formation in liver, spleen and lymphoid tissue; necrotizing encephalitis with demyelinisation and syncytia formation
- enterotropic strains: villus attenuation, enteroytic syncytia and mucosa necrosis of the terminal small intestine, the cecum and the ascending colon

Morbidity and mortality

- usually 100% of animals are infected
- mortality close to 100% in neonates (all virus strains, epizootic infection) and in immunodeficient mice (polytropic strains)
- mortality 0% (or very low) in all other cases

Interference with research

Oncology

- Contamination of transplantable tumors (Nicklas et al., 1993)
• abnormal tumor invasion pattern, abnormal tumor passage intervals, spontaneous regression or oncolysis of normally stable tumors (Akimaru et al., 1981; Braunsteiner and Friend, 1954; Fox et al., 1977; Manaker et al., 1961; Nelson, 1959)
• rejection of human xenografts (Kyriazis et al. 1979)
• altered response to chemical carcinogens (Barthold, 1986a)

**Infectiology**

• Confusion about origin of virus isolates: Tettang (Smith et al., 1983), multiple sclerosis (Gerdes et al., 1981), puffinosis (Nuttall and Harrap, 1982)
• Reduced susceptibility for viral infections (PVM, Sendai) (Carrano et al., 1984)
• Potentiation of subclinical MHV infections by urethane and methylformamide (Braunsteiner and Friend, 1954), halothane (Moudgil, 1973), transplantation of tumors (Barthold, 1986b), concurrent infection with Eperythrozoon coccoides (Kraft, 1982)
• enhances resistance to Salmonella typhimurium in mice by inducing suppression of bacterial growth (Fallon et al., 1991).

**Immunology**

• immunodepression and immunostimulation depending on the time of infection (Virelizier et al., 1976).
• MHV replicates in macrophages and with or without lysis in both B and T lymphocytes (Bang and Warwick, 1960; de Souza and Smith, 1991; Lamontage et al., 1989).
• enhanced and suppressed macrophage function (Boorman et al., 1982; Dempsey et al., 1986) and dysfunction of T and B cells (Casebolt et al., 1987; Cook-Mills et al., 1992; de Souza et al., 1991; Smith et al., 1991).
• activation of natural killer (NK) cells and alteration of the interferon responsiveness of infected mice (Schindler et al., 1982; Virelizier et al., 1976).
• reduced levels of cytokines, interleukins and gamma interferon in spleen cells (de Souza et al., 1991).
• recovered mice have complete or partial protection against T cell dysfunctions when re-infected with different strains of MHV (Smith et al., 1992).
• macrophage dysfunctions continue in MHV-recovered mice (Boorman et al., 1982)
• MHV infection can durably modify unrelated T cell responses that are initiated at the time of infection (Coutelier et al., 1991).
• permanent decrease of skin graft rejection and T cell dependent antibody responses after recovering from MHV-A59 infection (Cray et al., 1993).
• enhancement of concomitant autoimmune reactions (Lardans et al., 1996)

**Physiology**

• alteration of liver enzyme levels, patterns of protein synthesis and other biochemical markers (Barthold, 1986a; Lucchiari et al., 1992; Piazza, 1969)
• induction of alpha-fetoprotein (Kiuchi et al., 1974) and increase of iron uptake (Tiensiwakul and Husain, 1979)
• changes in peripheral blood such as anemia, thrombocytopenia, leukopenia and increased monocyte procoagulant activity (Levy et al., 1981; Piazza et al., 1965).
• decrease of the incidence of diabetes in non-obese diabetic mice (Wilberz et al., 1991)

Reproductive technology
• persistent contamination of embryonic stem (ES) cells without diminishing their pluripotency (Okumura et al., 1996)

References


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