‘Orphan Parvovirus’ ‘OPV’
Mouse Parvovirus (MPV)
Rat Parvovirus (RPV)

History

- serologic evidence for the existence of additional, antigenetically distinct parvoviruses was found during 1983-1984 in mice and rats
- agents were known as 'orphan' parvoviruses or OPV
- mouse and rat orphan parvoviruses have been identified and characterised and have been renamed mouse parvovirus (MPV) (Ball-Goodrich & Johnson 1994) and rat parvovirus (RPV) (Ball-Goodrich et al. 1998)

Host species

- natural host: laboratory and wild rats (RPV) and mice (MPV)

Properties of the Virus

- all parvoviruses are highly temperature resistant (Fassolitis et al. 1985)
- all parvoviruses are highly resistant to environmental conditions like e.g. desiccation (Tattersall & Cotmore 1986, Yang et al. 1995, Jacoby et al. 1996)
- MPV is distinct from but related to MVM (Ball-Goodrich & Johnson 1994)
- MPV infection persists after seroconversion even in mice inoculated as adults (Smith et al. 1993, Jacoby & Ball-Goodrich 1995)
- viral DNA of RPV is detectable in lymphoid tissues for months (Ueno et al. 1997)

Strain susceptibility

- none

Organotropism

- viral replication in mitotically active tissues like, e.g. gastrointestinal tract, lymphocytes, tumours, tropism for lymphoid cells (McKisic et al 1993, Jacoby et al. 1996, Shek et al. 1998)

- predilection for lymphoid tissues of infant and adult mice (MPV) (Jacoby & Ball-Goodrich 1995) or endothelium and lymphoid tissues of rats (RPV) (Ball-Goodrich et al. 1998)
- MPV detectable in pancreas, spleen, lymph nodes, lungs, intestines, kidneys (Smith et al. 1993, Besselsen et al. 1995)
- RPV detectable in lymph nodes, small intestines, kidneys, spleen, etc. (Ueno et al. 1996, Ball-Goodrich et al. 1998)

Clinical disease

- infection asymptomatic even in infant and severely immunocompromised mice (SCID mice) (Smith et al. 1993, Jacoby et al. 1995) and rats (Jacoby & Ball-Goodrich 1995, Ball-Goodrich et al. 1998)

Pathology

- no pathology or histologic lesions after experimental (i.p., oral) infection (Smith et al. 1993, Jacoby et al. 1995, Ball-Goodrich et al. 1998)

Morbidity and mortality

- infection asymptomatic even in neonatal and infant mice and rats (Smith et al. 1993, Jacoby & Ball-Goodrich 1995, Ball-Goodrich et al. 1998)

Zoonotic potential

- none

Interference with research

Immunology

- MPV first isolated from a CD8+ T cell clone that had lost function and viability (McKisic et al. 1993)
- inhibition of proliferation of CD8+ and CD4+T cell clones stimulated with IL-2 or antigen, but no inhibition of the generation of cytotoxic T cells in mixed lymphocyte cultures (MLC) (McKisic et al. 1993)
- reduced cytolytic capacity of T cells after MPV infection (McKisic et al. 1996)
- MPV diminishes the of proliferation rate o lymphocytes from spleen and popliteal lymph nodes, but augments the proliferative response of cells from mesenterial lymph nodes (McKisic et al. 1996, Jacoby et al. 1996)
- T cell mediated potentiation of rejection of allogeneic skingrafts by MPV infection, induction of rejection of syngeneic skin grafts (McKisic et al 1998)

- RPV infection may modulate immune function (Ball-Goodrich et al. 1998)

Cell biology

- contaminant of cell lines (McKisic et al. 1993)
- infection transplantable tumours (Ball-Goodrich et al. 1998

Oncology

- MPV accelerates tumour allograft rejection (McKisic et al. 1996)
- contamination of transplantable leukaemia cells by RPV (Ball Goodrich et al. 1998)
- milder disease (reduced hepatosplenomegaly) or delayed onset of clinical signs and leukaemia in RPV infected tumour-bearing rats compared to uninfected rats (Jacoby et al. 1996, Ball Goodrich et al. 1998)

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