

# Toolan's H-1 Virus (H-1)

## Host species

- natural host: laboratory and wild rats (JACOBY et al. 1979)
- hamsters and other species can be infected experimentally (KILHAM & MARGOLIS 1975, National Research Council 1991)
- mouse cells cannot be infected by H-1 (TATTERSALL & COTMORE 1986)

## Properties of the virus

- highly temperature resistant (FASSOLITIS et al. 1985)
- highly resistant at different pH values, desiccation and other environmental conditions (GREENE 1963, TATTERSALL & COTMORE 1986)

## Strain susceptibility

- none

## Organotropism

- viral replication only in mitotically active tissues like, e.g. embryo, intestines, tumours (JACOBY et al. 1979)
- pathogenic for the developing liver and cerebellum (JACOBY & BALL-GOODRICH 1995)

## Clinical disease

- no clinical signs after natural infection (National Research Council 1991)
- fetal and neonatal abnormalities (KILHAM & MARGOLIS 1975)
- cerebellar hypoplasia and chronic ataxia in young animals after experimental infection (MARGOLIS & KILHAM 1975)

## Pathology

- no lesions after natural infection
- experimental malformations of the central nervous system, skeleton, and teeth (KILHAM & MARGOLIS 1975)
- hepatocellular necrosis after partial hepatectomy (RUFFOLO et al. 1966)

## **Morbidity and mortality**

- no clinical signs after natural infection
- mongoloid-like deformations in hamsters experimentally infected as newborns (TOOLAN & LEDINKO 1968)

## **Zoonotic potential**

- none

## **Interference with research**

### **Physiology**

- delayed healing of bone fractures and altered callus formation (KILHAM & MARGOLIS 1975)
- inhibition of lipid formation in rat kidney cells in vitro (SCHUSTER et al. 1991)
- increased abortion rate (KILHAM & MARGOLIS 1969)

### **Pathology**

- hepatocellular necrosis after partial hepatectomy (RUFFOLO et al. 1966)

### **Infectiology**

- viral inclusion bodies in animals bearing larval forms of tapeworms (KILHAM et al. 1970)

### **Cell biology**

- contaminant of permanent human cell lines (HALLAUER et al. 1971)
- infection of human cells is increased after oncogenic transformation (TOOLAN & LEDINKO 1965, DUPRESSOIR et al. 1989, CHEN et al. 1986, ROMMELAERE & CORNELIS 1991)
- human cells naturally or experimentally transformed with DNA tumour viruses are permissive for H-1 infection (FAISST et al. 1989)
- in various human lymphoma-derived cells a persistent infection can occur (FAISST et al. 1990)

### **Teratology**

- fetal deaths and congenital malformation after inoculation into pregnant hamsters (FERM & KILHAM 1964)

### **Infectiology**

- H-1 together with KRV and *C. piliforme* can influence the prevalence rate of Yersinia-induced arthritis in rats (GRIPENBERG-LERCHE & TOIVANEN 1993, 1994)

## Oncology

- greater susceptibility of human oncogenic transformed cells and tumour-derived cell lines than normal untransformed parental cells (CORNELIS et al. 1988, ROMMELAERE & CORNELIS 1991)
- presence of H-1 virus reduces the number of tumours produced by an oncogenic adenovirus in hamsters (TOOLAN & LEDINKO 1968)
- reduced incidence of spontaneous tumours in hamsters experimentally infected at birth (TOOLAN 1967, TOOLAN et al. 1982)
- reduced incidence of chemically induced tumours in experimentally infected hamsters (TOOLAN et al. 1982)
- inhibition of tumour formation in nude mice from a transplanted human tumour and retardation of tumour growth (DUPRESSOIR et al. 1989)

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