PATHOLOGICAL STUDIES ON MICE INFECTED WITH HEMORRHAGIC FEVER WITH RENAL SYNDROME (HFRS) VIRUS

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Using different mice strains (SCID, nude, BALB/c) with varying immunological competence, the pathogenesis and immunologic effects of HFRS virus were investigated.

Of the different strains infected with the HFRS virus, only the SCID mice died at 35 days postinoculation (PI). The infected mice showed neurological symptoms, such as agitation and hypersensitivity at 21 and 28 days PI, respectively. At 35 days PI, there were ruffled hair coats, depression and wasting among them.

Nude mice exhibited agitation as the only symptom at 35 days PI. BALB/c mice, however, did not exhibit any symptoms.

Immunohistochemically, viral antigens were detectable in the cerebral nerve cells of the SCID mice from 14 days PI. Histologically, eosinophilic intracytoplasmic inclusion bodies were observed in the infected never cells at 21 days PI. From 28 days PI, the distribution of viral antigens extended into the hippocampus, thalamus and cerebral cortex with degeneration and necrosis of the infected never cells. At 35 days PI, viral antigens were observed in the lung, kidney, liver and heart in SCID mice, although no histopathological changes were observed. These results suggest that in HFRS viral-infected SCID mice, the CNS is primarily affected by the virus before spreading to the other viscreral organs.

In nude mice infected with the HFRS virus, the extension of the viral antigens was first observed in the cerebrum at 35 days PI. In the infected BALB/c mice, viral antigens were observed infrequently in the endothelial cells of some blood capillaries, and in the spleen, but did not extend.

The results of this study showed that HFRS viruses infected cells of mature hosts and that preventing HFRS viral infection from extending was dependent on the immunological competence of the host animal.