

## Virology

**Clinical evaluation of respiratory tract infections due to RSV, Adenovirus, Parainfluenza 1, 2, 3, Influenza A and B and diagnosed within 24 h using an EIA**

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Nasopharyngeal secretions (NPS) collected by a mucus extractor from more than 1000 children with respiratory tract infections (including otitis) and aged from newborn to 10 years were investigated for the presence of viral antigens. The antigens were detected by an enzyme immunoassay (EIA) as originally described by Sarkkinen and coworkers (J. clin. Microbiol. 13 (1981) 2158). Antigens of the following viruses could be found: RSV, Adeno, Parainfluenza 1, 2, 3, Influenza A and B. The study period was one year; an etiological diagnosis could be achieved within 24 h in each instance. Viral antigens could be demonstrated in 25% from all NPS samples studied. RSV infection was diagnosed most frequently; RSV antigens being present in about 46% from all positive specimens, followed by Parainfluenza 3 (28%) and Adenovirus (13%).

Clinically a pronounced association was found between RSV infection and lower respiratory tract disease in particular in infants less than 1 year old. A croup syndrome was often observed during Parainfluenza 3 infection, whereas tonsillitis and pharyngitis most frequently were due to Adenovirus infection.

**Studies on the differential tissue specificity of two variants of the minute virus of mice**

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The prototype strain of the minute virus of mice, MVM(p), grows in fibroblasts. A variant, MVM(i), grows in lymphocytes and is immunosuppressive in vitro. To determine the part of the MVM genome which is responsible for the differential tissue-specificity, we have determined the complete nucleotide sequence of the genome of MVM(i) and compared it to that of MVM(p). We have also constructed hybrid viruses containing complementary parts of the MVM(p) and MVM(i) genomes, and tested their respective tissue-specificities.

Major nucleotide differences are found in the 5' end of the viral genomes. In agreement with this fact, preliminary experiments indicate that the tissue-specific determinants of MVM(p) and MVM(i) are also found in this region.

**Comparison of Sendai virus and influenza virus surface glycoproteins**

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The nucleotide sequences of the Sendai virus (SV) F and HN surface glycoprotein genes were determined. The deduced primary structure of the proteins, when analyzed on a hydropathy plot, showed that the F protein was anchored in the membrane at its C-terminus but that the HN protein was unusually anchored in the opposite orientation. A similar situation exists for the two influenza virus (FLU) surface glycoproteins. Since the two SV and the two FLU glycoproteins share the same three activities (hemagglutinin, neuraminidase, fusion), their aa sequences were compared for homology. Limited but statistically significant homology was detected suggesting that SV and FLU shared common ancestry. The data further suggests that intragenic rearrangement as well as gene concatenation has taken place during this evolution.

**A human homologue to protein Mx of influenza virus resistant mice**

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Protein Mx, a nuclear 75,000 dalton protein, is induced by interferons (IFNs)- $\alpha/\beta$  in mouse cells carrying the influenza virus resistance allele  $Mx^+$ . Treatment of  $Mx^+$  cells with IFN- $\gamma$  fails to induce protein Mx and does not render these cells resistant. Similar proteins involved in host defense against influenza viruses might conceivably exist in other species, including man. Polyclonal and monoclonal antibodies with specificity for protein Mx detected an IFN-induced 80,000-Da protein in peripheral blood lymphocytes and in fibroblasts of healthy human donors. The human protein, like protein Mx, was induced by IFN- $\alpha$  but not by IFN- $\gamma$ . Unlike the mouse protein, it was not localized in the cell nucleus. Its role in antiviral defence in man remains to be established.

**Epidemiologic evidence linking canine distemper virus to multiple sclerosis**

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Multiple sclerosis (MS) is a disease of unknown etiology and pathogenesis, although it is generally thought that one or more infectious agents cause(s) MS, perhaps through an autoimmune pathogenesis. Recent epidemiologic studies show unique MS experiences in a number of relatively isolated communities, and together with the unusual worldwide prevalence of MS, supports the possibility that one exogenous factor may be primarily responsible for causing MS. While this factor has not been identified, the canine distemper virus (CDV)-MS hypothesis has predicted or explained the MS experiences in the Orkney and Faeroe Islands, Iceland, Newfoundland, and Sitka, Alaska. No other factor adequately explains even one such MS experience. Thus at the present time CDV appears to be the leading candidate agent in initiating MS.

**Macromolecular synthesis in hepatitis A virus-infected cells**

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Infection of MRC-5 (diploid human embryonic lung) cells by hepatitis A virus (HAV) leads to a persistent infection. In such cells virus replication does not cause cytopathology and, apparently, cellular viability is not impaired. The course of HAV replication in MRC-5 cells is known to be characterized by three distinct phases: 1) a short phase of active virus production, 2) a phase during which only noninfectious hepatitis A antigen (HAAg) is synthesized, and 3) a persistent phase with only little virus-specific metabolic activity. Infectious HAV and viral antigen mostly remain cell associated. Pulse-label experiments with nucleic acid and protein precursors confirmed that neither cellular DNA- and RNA- nor cellular protein synthesis is significantly disturbed by infection with HAV. According to the results of similar experiments in the presence of actinomycin D, significant levels of the synthesis of genomic viral RNA and of viral mRNA are confined to phase 1 only. Whether the increased production of non-infectious HAAg during phase 2 is due to a continuing selective synthesis of viral mRNA or only reflects a prolonged half-life time of such molecules is presently under investigation.