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Interferon Alters Transport of Vesicular Stomatitis Virus Glycoprotein. R. Maheshwari, V. Singh, G. Damewood IV, C. Stephensen, C. Oliver*, and R. Friedman, USUHS, Bethesda, MD 20814, U.S.A., *Lab. of Microbiol. and Immunol., NIDR, NIH, Bethesda, MD 20205, U.S.A.

Interferons (IFNs) have an unusual mechanism of action against some membrane associated viruses. The inhibitory effect of IFN is expressed in two major ways: (i) inhibition of virus assembly, release, and budding as seen in most murine leukemia viruses (MLVs), mouse mammary tumor viruses, and bovine parainfluenza viruses; or, (ii) the combination of a small decrease in the production of virus particles and a much greater reduction in the infectivity of released virions as seen in some infections with MLV and vesicular stomatitis (VSV) virus. In IFNβ-treated mouse L_B cells, VSV glycoprotein (G) does not efficiently localize on the plasma membrane from which site it is normally incorporated into budding VSV particles. Double-label immunofluorescense studies indicated that almost all of the VSV G protein was plasma membrane-associated during the logarthmic phase of virus replication. In contrast, treatment with IFN resulted in inhibition of VSV-G transport, so that the G remained associated with the Golgi complex (GC). In both IFN-treated and control cells, G was resistant to treatment with the enzyme endo-β-N-acetylglucosamine H indicating that the bulk of the G had moved to the trans compartment of the GC.

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Recombinant Alpha and Beta Interferons Inhibit Epidemic Isolates of Acute Hemorrhagic Conjunctivitis. [redacted], and M. Yin-Murphy. Department of Ophthalmology, [redacted] University Medical School, Shreveport, LA and Department of Microbiology, National University of Singapore, Singapore.

The antiviral activities of recombinant and natural human alpha and beta interferon (IFN) against epidemic isolates of CA24 was investigated in human conjunctival cells. All CA24 isolates were sensitive to the antiviral activity of natural and recombinant alpha and beta IFN, however, CA24 isolated late in the epidemic were inhibited more by alpha IFN subtypes A and C ($\geq$0.5 log10 reduction) than natural alpha IFN. Recombinant IFNs were equally or more inhibitory than natural IFN except for alpha IFN J, which was less effective in inhibiting CA24 cytopathogenesis and replication than natural IFN and required a longer period ot time to initiate an antiviral state against CA24. Interestingly, inhibition of cell death by IFN was reduced when cells were infected with $\geq$0.3-0.5 CA24/cell. Most importantly, CA24 was inhibited when 1000 units of IFN was applied as early as 2 hr before infection. Our results indicate that IFN sensitivity of CA24 epidemic isolates was relatively stable during the course of this AHC epidemic but the relative IFN sensitivity of each CA24 was dependent upon multiplicity of infection and the CA24 isolate. Also, they suggest that some recombinant IFNs may be equally or more effective than natural IFN in preventing and/or inhibiting disease and spread of virus during AHC epidemics.