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Antiviral effect of interferon inducers (florenons) on exoerimental Bunyavirus infections in the Soviet Union

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Antivaral effect of amixin and its derivates were studied in mice infected with Bunyaviruses (Issyk-Kul fever virus,Crimean hemorragic feves virus(CHF), Snow shoew hare virus).Amixin ,125 mg/kg given oraly 24 hrs before the virus inoculation induced IFN synthesis up to 640 u/ml and protected 75% of mice infected with Issyk-Kul virus,55%-CHF virus,40-45% -show shoew hare virus. Decrease the dose at the inducer brought down the IFN response but did not influence on protection effect of the drug. The level of the serum IFN, as well as protective effect of the amixin was less evident at intramuscular injection. Among the amixin derivates 6 different compaunds showed the high antiviral activity.Possible mechanism in action : induction of IFN synthesis and direct activation of IFN system enzymes, and, probably,inhibition of protein synthesis.

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Characterization of a Novel Antiviral Glycoprotein from Human Serum. D.H. Coppenhaver, I.P. Singh, A. Chopra, and S. Baron. Department of Microbiology, University of Texas Medical Branch, Galveston, TX, USA.

UTI-ß is a broadly active viral inhibitor present in normal human serum. Its distinguishing properties include moderate resistance to heat denaturation, lack of species specificity, and action by blocking attachment of viruses to target cells. We have partially purified UTI-ß, and show that the antiviral activity resides in a glycoprotein with a molecular mass of ≈60 kDa. The antiviral activity of UTI-ß is destroyed by $NaIO_4$ oxidation. Treatment with a cocktail of carbohydrases also causes loss of antiviral activity, confirming the presence of essential carbohydrate structure. Treatment with proteinase K decreases the size of the active moiety to <1 kDa, without decreasing its activity. The protease digest of UTI-ß inhibits infection of C81 cells by HIV in preliminary experiments. Like the native molecule, the protease-digestion product of UTI-ß loses its antiviral activity when treated with carbohydrases, suggesting the antiviral activity of UTI-ß resides in its oligosaccharide structure. The protease-released form of UTI-ß bears some similarities to a second broadly active antiviral molecule, UTI-α, which is present in several body secretions and tissue abstracts. These findings show that normal human serum contains a broadly active antiviral glycoprotein in which oligosaccharide structure, but not intact protein structure, is required for antiviral activity.