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“Global Outbreaks: Interferons as 1st Responders”

For many re-emerging and newly emerging global outbreaks of virus infections, there are no vaccines or antivirals available. Each antiviral developed to target a specific viral pathogen will inevitably lead to the emergence of resistant strains. This is evident for H5N1 avian flu, where Tamiflu-resistant strains have emerged. Moreover, the time from identification of a new/altered virus to development of the appropriate vaccine, may take up to 6 months, when populations will be unprotected during a pandemic. Accordingly, a complementary strategy is to develop broad spectrum antivirals that are not pathogen-specific, but enhance the host immune response to infection – regardless of the virus. Type I interferons present as ideal candidate broad spectrum antivirals. Data will be presented that demonstrate both the direct antiviral effects of interferons against any and all viruses and also their influence on the immune response to virus infection. These results led to the examination of the antiviral effects of interferons against the SARS coronavirus during the outbreak in 2003 and also against influenza A strains, notably the recent H1N1 pandemic strain. During the Ebola outbreak in West Africa there were no approved antiviral drugs for the treatment of Ebola virus disease. Based on our *in vitro* evidence of antiviral activity of interferon (IFN)- β activity against Ebola virus, we conducted a single arm clinical study in Guinea to evaluate the safety and therapeutic efficacy of IFN- β treatment for EVD during the recent outbreak. When compared to supportive care only, IFN- β treatment facilitated viral clearance from the blood and appeared associated with earlier resolution of disease symptoms. Survival, calculated from the date of consent for those in the trial and date of admission to the treatment unit from those in the control cohort, to the date of death, was 19% for those receiving supportive care only, compared to 67% for those receiving supportive care plus IFN- β . Given the differences in baseline blood viremia between the control cohort and the IFN- treated cohort, additional controls were included for a subset analysis, from other treatment units in Guinea, matched with the IFN-treated patients based on age and baseline blood viremia. Subset analyses using this expanded control cohort indicated that patients without IFN- β treatment were more likely to die than those treated. Viewed altogether the results suggest a rationale for further clinical evaluation of IFN- β .

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