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MX1 requirements for influenza A virus restriction

Contenu

Type 1 interferons (IFNs) are produced by infected cells upon detection of pathogenic agents and are the first line of defence against viral infections. IFNs induce the expression of hundreds of IFN-stimulated genes (ISGs), both in infected and neighbouring cells. The products of these ISGs in turn induce in cells a potent antiviral state, capable of limiting viral replication. The dynamin-like, high-molecular weight GTPases MX1 and MX2 play a significant role in the IFN-induced inhibition of viral replication. Human MX1(or MxA) is a restriction factor of broad antiviral activity, able to inhibit influenza A virus (FLUAV) and a great diversity of RNA and DNA viruses at different stages of their life cycles. Human MX2 (or MxB) is notably able to inhibit HIV-1 and herpes viruses. Although the antiviral activity of human MX1 has been studied extensively, the molecular mechanism of action remains largely unsolved. MX1 and MX2 are 63% identical at the amino acid level, share a similar domain organization and their crystal structures are almost practically superimposable. Taking advantage of chimeras between MX1 and MX2 in which their different domains have been swapped, as well as point mutants, we have notably identified a new motif required for influenza A restriction by MX1. Importantly, some MX1/MX2 chimeric proteins are highly active against influenza A viruses but not in the context of minireplicon assays. Additional ongoing efforts to better characterize MX1's requirement for influenza A restriction and mechanism of action will be presented.

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Innate Immunity

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Type de contribution : Paper presentation