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Targeting influenza using genetic vaccines

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DNA and RNA based genetic vaccines are now considered as developed. This is lower in cost and effective at the same time which is extensively used for various cancer therapies, ancestral allergies and various infections and chronic diseases such as seasonal flu like bird flu, swine flu, equine flu etc. With all these advantages there are certain limitations for these genetic vaccines which might be the major drawback for its commercial growth and animal trials. The major limitation is delivery of DNA vaccines to the target sites which need to develop carrier molecules for delivering vaccines into immunized individuals. Because of lack of carrier molecules results in low intracellular presence of vaccine encoding as antigen thus low expression level which in turn reduces immune response against the target antigen. By the improvement of DNA delivery technology we can significantly increase effectiveness of genetic vaccines. Various mechanical methods like jet injections, carrier molecule (lipids, proteins etc.), electroporation, micro-needles etc are being used. Other methods are also popular among which most common are peptides, prime-boost with plasmid DNA and monovalent-inactivated vaccines. Using adjuvants of anti-influenza DNA vaccines and PCR generated complementary anti-RNA sequencing to block influenza virus expression are also popular. Researchers are focusing on DNA vaccines and are employing many strategies to fight against influenza.

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Influenza viral manipulation of S1P-metabolizing enzymes: Novel targets for anti-influenza therapy?

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Sphingosine 1-phosphate (S1P)-metabolizing enzymes regulate the level of bioactive sphingolipids and mediate important biological processes. S1P lyase induces the degradation of S1P while sphingosine kinase (SphK) generates S1P from sphingosine. In this study, we investigated the role of S1P lyase and SphK in host defense to influenza virus infection. Importantly, influenza virus infection increased the expression and activation of SphK, whereas the infection reduced the level of S1P lyase. To further study the detailed function of S1P lyase and SphK during influenza virus replication, biochemical, genetic, and pharmacological systems were employed. The results indicate that S1P lyase displayed anti-viral activity by rendering cells resistant to influenza virus infection and viral cytopathogenicity. S1P lyase directly promoted type I IFN signaling pathway, leading to the elevated STAT1/STAT2 activation and heightened expression of interferon-stimulated genes. In contrast to S1P lyase, overexpression of SphK enhanced the production of infectious influenza viruses and the inhibition of SphK activity or expression strongly suppressed the replication of influenza virus, revealing the proviral action of SphK. Further, inhibitors of SphK altered influenza virus-induced cellular signalling pathways that are crucial for efficient viral replication. Collectively, our findings demonstrate that regulation of S1P lyase and SphK could potentially suppress influenza virus propagation and thus these enzymes represent novel cellular targets for the treatment of influenza virus infection.

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