Chinese Formula for Anti-viral properties and offsetting the flu

Caution: Unfortunately, The FDA does not regulate herbal medicine rendering the average consumer unaware of certain American and European manufacturers with proper testing and proven track records in place. Always consult your herbal specialist prior to purchasing an herbal medicine – for this and many other reasons.

• Format: Abstract

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Mechanism by which ma-xing-shi-gan-tang inhibits the entry of influenza virus.

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ETHNOPHARMACOLOGICAL RELEVANCE:

Ma-xing-shi-gan-tang (MXSGT, aka maxing shigan powder), a Chinese herbal decoction, has been used for the treatment of the common cold, fever, and influenza virus infections. However, the underlying mechanisms of its activity against the influenza virus are not fully understood. In this study, we examined the antiviral effects of MXSGT in influenza-virus-infected MDCK cells and their underlying mechanisms, including the damage of the viral surface ultrastructure and the consequent inhibition of viral entry.

MATERIALS AND METHODS:

The antiviral activity of nontoxic concentrations of MXSGT against influenza virus A/WSN/33 was examined by assaying (neutralization assay) its inhibition of the virus-induced cytopathic effects. The mode of MXSGT action was first examined with a time-of-addition assay of synchronized infections, followed by viral attachment and penetration assays. Viral endocytosis was evaluated with attachment and penetration assays. We also performed assays related to the inhibition of viral entry, such as neuraminidase activity, hemagglutinin activity, and phosphoinositide-3-kinase (PI3K)/AKT phosphorylation assays. The inhibition of viral replication was demonstrated by quantitative real-time PCR, immunoblotting, and immunofluorescence microscopy. The surface ultrastructure of the MXSGT-treated virus was revealed by atomic force microscopy.

RESULTS:

MXSGT exhibited an EC(50) of 0.83±0.41mg/ml against influenza virus A/WSN/33 (H1N1), with broad-spectrum inhibitory activity against different strains of human influenza A viruses, including clinical oseltamivir-resistant isolates and an H1N1pdm strain. The synthesis of both viral RNA and protein was profoundly inhibited when the cells were treated with MXSGT. The time-of-addition

assay demonstrated that MXSGT blocks the virus entry phase. This was confirmed with attachment and penetration assays, in which MXSGT showed similar inhibitory potencies (IC(50) of 0.58±0.07 and 0.47±0.08mg/ml). High-resolution images and quantitative measurements made with atomic force microscopy confirmed that the viral surface structure was disrupted by MXSGT. We also established that viral entry, regulated by the PI3K/AKT signaling pathway, was abolished by MXSGT.

CONCLUSIONS:

Our results give scientific support to the use of MXSGT in the treatment of influenza virus infections. MXSGT has potential utility in the management of seasonal pandemics of influenza virus infections, like other clinically available drugs.

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