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Alisol B 23-acetate broadly inhibits coronavirus through blocking virus entry and suppresses proinflammatory T cells responses for COVID-19 treatment

Objectives

Emerging SARS-CoV-2 causes a global health disaster and pandemic. This study aims to test the hypothesis that alisol B 23-acetate could be a viral entry inhibitor with proinflammatory property for COVID-19 treatment.

Methods

SARS-CoV-2 and its variants infected cell lines were applied to evaluate the anti-CoVs activities in vitro. The antiviral and anti-inflammatory effects were further confirmed in the SARS-CoV-2 and its variants challenged hamster and human angiotensin-converting enzyme 2 (ACE2) transgenic mice in vivo. The target of alisol B 23-acetate to ACE2 was analysed using hydrogen/deuterium exchange (HDX) mass spectrometry (MS).

Results

Alisol B 23-acetate had inhibitory effects on different species of coronavirus. HDX-MS study revealed that alisol B 23-acetate had inhibition potency toward ACE2. Alisol B 23-acetate remarkably decreased viral copy, reduced $CD4^+$ T lymphocytes and $CD11b^+$ macrophages infiltration and ameliorated lung damages in the hamster model. In Omicron variant infected human ACE2 transgenic mice, alisol B 23-acetate effectively alleviated viral load in nasal turbinate and reduced proinflammatory cytokines interleukin 17 (IL17) and interferon γ (IFNγ) in peripheral blood. The prophylactic treatment of alisol B 23-acetate by intranasal administration significantly attenuated Omicron viral load in the hamster lung tissues. Moreover, alisol B 23-acetate inhibited proinflammatory responses through mitigating the secretions of IFNγ and IL17 in the cultured human and mice lymphocytes in vitro.

Conclusion

Alisol B 23-acetate could be a promising therapeutic agent for COVID-19 treatment and its underlying mechanisms might be attributed to viral entry inhibition and anti-inflammatory activities.

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