OPEN ACCESS Freely available online

ISSN: 2155-6121
Journal of
Allergy & Therapy

Perspective

Prophylactic Administration of Astragaloside IV Relieves Recurrent Allergic Atopic Dermatitis

Rijkers Chowhan*

Department of Allergy and Immunology, Lovely Professional University, Gujarat, India

DESCRIPTION

Atopic Dermatitis (AD) is a common allergic inflammatory skin disease with high relapse rates and recurring severity. However, the underlying pathogenesis of AD recurrence is still unclear. Our previous study reported Astragaloside IV (AS-IV) administration in sensitization stage ameliorated the allergic inflammation of AD. In this study, we aimed to evaluate the efficacy of AS-IV which prophylactic applied in remission phase of AD and to investigate the underlying mechanisms

In this study, we compared the efficacy of AS-IV with three commonly applied medicines including Dexamethasone (Dex), Montelukast (Mon), and Loratadine (Lor) in a fluorescein isothiocyanate (FITC)-induced AD relapse (AD-Re) model. By investigating the pharmacokinetics profiles of AS-IV, and its effect on TLRs-mediated signaling pathway, we attempted to unravel the underlying mechanism of AS-IV with prophylactic administration against AD recurrence.

AS-IV has been shown to be effective in relieving allergic asthma. As a representative bioactive compound of YPFS, we reported AS-IV ameliorated allergic inflammation in sensitization stage of AD by inhibiting TSLP and IL-33 production. In this study, we firstly investigated the effect of AS-IV on anti-AD recurrence. Medications in remission phase of AD and re-exposure to allergens in a short time, AS-IV and conventional clinical drugs (Dex, Mon and Lor) alleviated the recurrent inflammation of AD significantly. However, with prolonged drug withdrawal, only AS-IV attenuated ear swelling and inflammatory cell infiltration as well as IgE production in recurrent phase of AD. AS-IV has been reported to down-regulate inflammatory cytokines such as TNF-α, IL-1β, and TGF-β. However, the immune response in AD is skewed towards Th2-mediated pathway and can in turn favour epidermal barrier disruption. Therefore, we then investigated the effects of AS-IV on Th2 cytokines production in AD-Re. Unlike Dex, Mon, and Lor, AS-IV could remarkably damp the IL-4, IL-5, and IL-13 levels of ear homogenates in AD-Re mice, but showed feeble effect on IFN-γ production. These indicated AS-IV could specifically decrease Th2 cytokines production in AD-Re model.

Impairment of epidermal barrier function, for instance, due to deficiency in the structural proteins, has been recognized contributing to AD aetiology and clinical manifestation. We previously found the defects of tight junction protein including occludin and CLDN1 were observed in remission phase of AD. However, AS-IV appeared had barely effects on CLDN1 and occludin (data not shown) expression in AD-Re. In addition, TLRs play a fundamental role in detecting invading pathogens or damage and initiating the innate immune system of mammalian cells. Alterations of TLRs play a potent role in susceptibility to AD. AS-IV presented a wide range of pharmacological effects through multiple pathways, however, most of them are related to TLR4-mediated NF-κB signalling pathways.

Consistently, protein levels of TIRAP and MyD88, the adapter proteins of TLR8 in ear tissue also reduced obviously with AS-IV treatment. Moreover, the downstream transcription factor NF-κB showed a similar trend as TLR8. TLR8 was initially considered to be inactive in mice. Until recently, the potential role of TLR8 in the generation of a critical immune response against bacterial infection and cancer has just begun to be uncovered. Our finding indicated TLR8-mediated NF-κB pathway may also play an important role in AD recurrence pathogenesis. AS-IV administration in remission phase of AD could regulate TLR8-mediated NF-κB pathway to against recurrence.

Correspondence to: Rijkers Chowhan, Department of Allergy and Immunology, Lovely Professional University, Gujarat, India, Email: rlayton56@gmail.com

Received: 04-Apr-2022, Manuscript No. JAT-22-16708; **Editor assigned:** 07-Apr-2022, Pre QC No. JAT-22-16708 (PQ); **Reviewed:** 22-Apr-2022, QC No. JAT-22-16708; **Revised:** 29-Apr-2022, Manuscript No. JAT-22-16708 (R); **Published:** 06-May-2022, DOI: 10.35248/2155-6121.22.13.282.

Citation: Chowhan R (2022) Prophylactic Administration of Astragaloside IV Relieves Recurrent Allergic Atopic Dermatitis. J Allergy Ther. 13:282.