



Attenuation of allergic airways inflammation by an extract of *Hymenocardia acida*

Submitted by claire.leroy on Wed, 04/29/2015 - 10:37

Titre Attenuation of allergic airways inflammation by an extract of *Hymenocardia acida*

Type de publication Article de revue

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Editeur Academic Journals

Type Article scientifique dans une revue à comité de lecture

Année 2014

Langue Anglais

Date Nov. 2014

Numéro 3

Pagination 15-24

Volume 5

Titre de la revue Journal of Physiology and Pathophysiology

ISSN 2141-260X

Mots-clés asthma [9], *hymenocardia acida* [10], Mice [11], Prévention [12]

Tracheal hyperresponsiveness, airway mucus production and bronchoalveolar inflammation are the major components of asthma. Here, we aim to investigate the role in the control of asthma of a bioactive plant extracted from *Hymenocardia acida* in a physiological and pathophysiological model. The effect of *H. acida* crude extract (HACE) on total cellular components of bronchoalveolar (BAL) fluids was performed on ovalbumin (OVA) and lipopolysaccharide (LPS)-challenged Swiss mice for induction of allergic asthma and airways inflammation, respectively. Mice were pretreated with 0.9% sodium chloride (NaCl), HACE (oral doses at 100 mg/kg/body weight) for a week and then by intranasal instillation with OVA (0.5 mg/ml) + aluminium hydroxyde (20 mg/ml), during three days after intraperitoneally sensitization or with LPS (0.4 mg/ml) for a day (OVA or LPS + HACE). The BAL cells were collected in a mixed solution (0.9% NaCl and 2.6 mm Ethylenediaminetetraacetic acid EDTA) one day after the last challenge and total cells were numbered in a Neubauer chamber. The HACE: (i) significantly inhibited the airways inflammation induced by a single intranasal instillation of LPS or allergic asthma on mice challenged with 3 consecutive days intranasal instillation of OVA in comparison to control mice only instilled with 0.9% sterile. NaCl : (ii) significantly impaired the increased levels of total cells in OVA and LPS-treated mice, without changing the basal cellularity after NaCl or HACE treatment; (iii) and significantly inhibit hydroxyl radicals and superoxide anions production. Taken together, these results suggest that HACE exposure induces a marked reduction of cellular component in the BAL fluid, which is only partially lymphocytes dependent.

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Titre abrégé J. Physiol. Pathophysiol.

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