Allergol Immunopathol (Madr). 2016;44(1):23–31

Allergologia et immunopathologia

Sociedad Española de Inmunología Clínica, Alergología y Asma Pediátrica

www.elsevier.es/ai

ORIGINAL ARTICLE

Amelioration of some immunological disorders caused by the faeces of the dominant true house dust mites in El-Minia Governorate, Egypt

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Received 30 September 2014; accepted 14 January 2015
Available online 13 May 2015

KEYWORDS
HDMs;
TNF-α;
IL-4;
IL-1β;
IL-13;
IgE and ROS

Abstract
Background: House dust mites (HDMs) faeces are the main factor involved in respiratory disorder. The true HDMs, *Dermatophagoides pteronyssinus* and *D. farinae*, detected in the samples collected from the house dust are the most important causes of allergic disorders such as asthma.
Objective: The aim of this investigation was to study the curcuma and karkade amelioration of the allergenic immunological disorder, especially some cytokines, IgE and ROS, caused by the faeces of the dominant true HDM, *D. pteronyssinus* and *D. farinae* in valley and desert houses in EL-Minia Governorate, respectively.
Methods: HDM cultures, faeces isolation, plant extraction and ELISA techniques were used. Male albino rats were classified into control, inhaled, and treated groups.
Results: The present immunological study on the dominant allergenic true HDMs, *D. pteronyssinus* and *D. farinae*, revealed that significantly higher serum levels of TNF-α, IL-1β, IL-4, IL-13 and IgE were found in rats treated with both *D. pteronyssinus* and *D. farinae* faeces than the other groups. In addition, statistical analysis of ROS data showed significant difference between the curcuma- and karkade-treated groups and either the control or the faeces-treated groups ($P < 0.05$).
Conclusions: Some immunological disturbances caused by repeated exposure to the faeces of two dominant allergenic true HDM species (*D. pteronyssinus* and *D. farinae*) in the valley and desert houses could be ameliorated by curcuma and karkade.

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http://dx.doi.org/10.1016/j.aller.2015.01.010

Introduction

Mite faeces are small airborne particles (about 20 μm). They have great allergenic potential due to protein residues, and inhalation of faecal particles is the main way of exposure.[1] Most allergens are biochemically active molecules and include enzymes, enzyme inhibitors, and proteins involved in molecular transport, regulation and cell and tissue structure.[2] The majority of works on mite allergens referred to species of the family Pyroglyphidae.[2] This family has five genera, of which the genus *Dermatophagoides* is the most important one from a medical point of view. The European house dust mite (HDM), *D. pteronyssinus*, and the American HDM, *D. farinae*, are distributed all over the world.[3,4]

The optimal environmental temperature for these mites is 18–27 °C. Proper indoor relative humidity, temperature and enough food are the key factors determining their survival and development.[5] Their major food source is skin scales, hair and wool of pets, fungi, plant pollen and organic debris. The highest HDM densities have been found in bedrooms and living rooms because these indoor spaces usually have large areas covered by textile materials.[4] In recent years, lifestyle changes including central heating systems in homes provide suitable conditions for growth and reproduction of HDMs.[1]

Allergic diseases such as asthma are amongst the most common chronic diseases affecting people in developed countries.[6] Asthma is a chronic and complex inflammatory disease of the airways with symptoms including excess mucus production, wheeze, dyspnoea, cough, fatigue, anxiety, tachycardia, and chest tightness.[7]

Relationships between HDMs and asthma are ambiguous. For example, atopy is not necessarily related to house dust mite allergens and also allergens may not be associated with atopy. Skin-prick test is the routine diagnostic method for atopy-related allergens. The population fraction of asthma attributable to atopy can be calculated when the proportion of people with asthma who have specific IgE or positive skin-prick tests is known.[8]

Many scientific reports suggest that asthma involves the activation of many inflammatory cells like mast cells, macrophages/monocytes, eosinophils, T-helper type-2 lymphocytes (Th2), dendritic cells, basophils, neutrophils and platelets.[9] These cells may also be important sources of mediators in asthma. Cytokines, the main important one of these mediators, can synergise or antagonise the effects of other cytokines and regulated in a complex manner and function via cytokine cascade.[10] The major groups of cytokines are lymphokines, pro-inflammatory cytokines, inhibitory cytokines, growth factors and chemokines. Asthmatic inflammatory cells are involved in the production of different cytokines such as Tumour necrosis factor (TNF)-α, Interleukin (IL)-1β, IL-4 and IL-13.

TNF-α and IL-1β are pro-inflammatory cytokines.[9,10,12] Two major forms of TNF are TNF-α and TNF-β. TNF-α is produced by many cells including macrophages, T-lymphocytes, mast cells, and epithelial cells, but the principal source is the macrophage. The secretion of TNF-α by monocytes/macrophages is greatly enhanced by other cytokines such as IL-1, granulocyte macrophage-colony stimulating factor (GM-CSF) and interferon (IFN)-γ. Devalia et al. showed that human eosinophils are also capable of releasing TNF-α, together with airway epithelial cells.[11] For IL-1, there are also two distinct forms (α and β). The major cellular sources of IL-1 are monocytes, macrophages, neutrophils, eosinophils, mast cells, platelets, lymphocytes, NK cells, endothelial cells, airway smooth muscle cells and vascular smooth muscle cells.[12] Air pollutants like nitrogen dioxide can stimulate epithelial cells express IL-1β while eosinophils can produce IL-1α.[13] A wide variety of stimuli including IL-1 itself TNF-α, GM-CSF, endotoxin and phagocytosis can increase the expression of IL-1 in monocytes/macrophages.[10]

IL-4 and IL-13 are anti-inflammatory lymphokines.[9,10,12] IL-4, a type-2 T-helper cell derived cytokine, is thought to be an upstream cytokine that regulates allergic inflammation by promoting Th2 cell differentiation and IgE synthesis.[14] Synthesis of IL-4 can be induced by stimulation of the antigen receptors on T-lymphocytes and by IgE-Fc receptor cross linking in mast cells and basophils. IL-13 is produced by activated T-lymphocytes, B-lymphocytes and mast cells. In the mouse, almost exclusively Th2 clones express IL-13, however, in humans it can be expressed in both Th1 and Th2 lymphocyte clones.[15]

Immunoglobulin (Ig) E is a class of antibody isotype that has been found only in mammals. It plays an essential role in type I hypersensitivity,[16] which manifests various allergic diseases, such as allergic asthma. Diagnosis of allergy is most often done when a physician reviews a patient's history and finds a positive result for the presence of allergen specific IgE when conducting a skin or blood test.[17]

Oxidative stress plays a role in asthma aetiology due to activation of various inflammatory cells of the respiratory tract such as neutrophils, eosinophils, mast cells and lymphocytes.[18] The continuous exposure of the respiratory tract to environmental oxidants and airway inflammatory cell-generated reactive oxygen species (ROS) creates a high level of oxidative stress in the lung.[7] Many studies have shown that cells involved in an asthmatic inflammatory process have a capacity for producing ROS. Activated eosinophils, neutrophils, monocytes, and macrophages generate superoxide (O_2^-) via a membrane associated NADPH-dependent complex. The subsequent dismutation of O_2^- can result in the formation of hydrogen peroxide (H_2O_2). O_2^- and H_2O_2 are moderate oxidants and both of them are critical in the formation of potent cytotoxic free radicals in biological systems through their interactions with other molecules.[19] This process is involved in asthmatic inflammation; moreover, the concentration of nitric oxide (NO) is increased in airways of asthmatic subjects.[20] In addition to the recruited inflammatory cells, epithelial airway cells are potential sources of ROS production.[21] Several asthma mediators, such as platelet activating factor, chemokines, adhesion molecules, and eosinophilic granule proteins[22] are potential promoters of ROS production. In addition to these endogenous sources, environmental factors linked to asthma, such as air pollutants, are important.[23] Increased production of ROS is deleterious because free radical-induced oxidation of proteins, DNA, and lipids can cause direct tissue damage and evoke cellular responses through the generation of secondary reactive species.[24]