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By lowering Dendritic Cell-Derived Th2 Cell Responses, lactoferrin Improves Ovaalbumin-Persuade Asthma in Mice

Shang Mu Chen*

Department of Pulmonary and Critical Care Medicine, Chia-Yi Christian Hospital, Chiayi 600, Taiwan

Introduction

More than 300 million people worldwide suffer from asthma, a dangerous respiratory condition. Tachypnea, a cough, and chest pain are some of the respiratory symptoms of asthma, which are caused by chronic airway inflammation, expiratory airflow constriction, and airway hyperresponsiveness (AHR). The causes of asthma are multifaceted and linked to the interactions of genes and surroundings. Allergy sensitization, dendritic cell activation, and cellular immunity are asthmatic immune responses that play a role in mediating inflammatory cells and mediators in the respiratory tracts. In addition to causing mast cells, eosinophils, and basophils to release mediators of allergic inflammation, allergens also cause IgE to be secreted. Due to its great degree of heterogeneity, asthma is challenging to diagnose clinically. Despite the development of numerous medications and non-pharmacological treatment options, there are still many asthma people with poorly managed symptoms or whose symptoms keep becoming worse. Therefore, there is still a pressing need for the creation of novel therapeutic strategies.

Based on immune-inflammatory pathways, asthma endotypes are classified as type-2 high (T2), type-2 low (non-T2), or a combination of T2 and non-T2. The production of Th2-related cytokines, eosinophilic airway inflammation, and circulating IgE release are all associated with allergic asthma, which is often categorised as a T2 immune-mediated disease. Following allergen sensitization, DCs absorb allergens and give naive T cells short peptides [1].

Description

We showed that LF has protective benefits on asthma in this study. Additionally, we showed that LF affected DC maturation and reduced the intensity of Th2 immune responses in allergic asthma. Asthma-related lung inflammation and damage, as well as AHR, may be effectively reduced by LF. These findings suggested that LF might act as a supplement to prevent lung damage from asthma and as an extra agent to lessen the severity of asthma. Millions of individuals throughout the world suffer from asthma, a chronic respiratory illness. In clinical practise, asthma is still difficult to treat and manage. There are hazards of constricted airflow limitation, exacerbations, hospitalisation, and death in over 10% and 2.5% of adults and children, respectively, who suffer from severe asthma. Here, we demonstrated how LF protects against asthma. OVA-induced AHR, pulmonary inflammation, Th2-related cytokines, and OVA-specific IgG1 and IgE production were all reduced in mice when LF was given orally [2].

***Address for Correspondence:** Shang Mu Chenm, Department of Pulmonary and Critical Care Medicine, Chia-Yi Christian Hospital, Chiayi 600, Taiwan; E-mail: Shangmuchen25@gmail.com*

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Additionally, we discovered that LF might inhibit DC maturation and OVA-specific Th2 cell responses in asthma that was caused by OVA. Bronchoconstriction, respiratory inflammation, airway remodelling, and AHR are the pulmonary symptoms of asthma. The most significant asthmatic symptom, AHR induces pulmonary tissue inflammation, respiratory mucus secretion, and airway smooth muscle development and constriction. Penh has been used to calculate pulmonary function changes. Mast cells, type-2 innate lymphoid cells (ILC2s), basophils, eosinophils, alternative activated macrophages (AAMs), DCs, and Th2 cells are all involved in allergen-induced asthma, a complex illness of the airways. Here, we demonstrated that LF therapy could lower the amount of cells associated with asthma in the airways. In addition, we demonstrated that lung tissue and proinflammatory cell infiltration were reduced in the groups that received LF treatment. Asthma is linked to airway goblet cell hyperplasia, which is regarded as a pathologic trait of mild, moderate, and severe asthma [3].

We discovered that the LF-treated groups showed a reduction in the goblet cell hyperplasia caused by OVA. Bronchial hyperresponsiveness (BHR) and the secretion of antibodies occur in T2 asthma as a result of the Th2 cytokines IL-4, IL-5, IL-9, IL-13, and IL-25 as well as the acute proinflammatory cytokines TNF-, IL-1, IL-6, and IL-8. We showed that the release of these asthma-related cytokines and antibodies was decreased by LF therapy. Th1-related cytokines (IFN- and IL-12), Th2-related cytokines (IL-4, IL-5, and IL-13), Th9-related cytokines (IL-9), Th17-related cytokines (IL-17A and F), and Treg-related cytokines are all produced by the stimulated naive T cells as they differentiate into Th1, Th2, Th9, Th17, and regulatory T (Treg) cells. cytokines (TGF- and IL-10) and subsequently cause asthmatic airway inflammation. The synthesis of IgE, eosinophil buildup, mast cell proliferation, mucus hypersecretion, and AHR are therefore increased as a result of these cytokines [4].

There are three different immunopathologies associated with asthma: eosinophilic asthma (allergic and nonallergic eosinophilic inflammation), noneosinophilic asthma (paucigranulocytic and type 1 and type 17 neutrophilic inflammation), and mixed granulocytic asthma (mixed granulocytic inflammation). Allergen-induced respiratory inflammation caused by DC-Th2-cells is known as allergic eosinophilic inflammation. Mast cells and ILC2-cell-induced respiratory inflammation caused by pollution or bacteria is known as nonallergic eosinophilic inflammation. Paucigranulocytic is oxidative stress- or pollution-induced BHR in mast cells. Type 1 and type 17 neutrophilic inflammation, respectively, are respiratory inflammations brought on by oxidative stress, pathogens, or DC-Th1 and Th17 cells. We showed that LF therapy in our asthmatic animal model reduced OVA-induced AHR by preventing Th2-type immunological reactions. Myeloid dendritic cells (mDCs) deliver antigens to Th0 in T2 asthma [5].

Conclusion

In the present study, our results showed that LF exerted protective effects in a BALB/c mouse model of OVA-induced allergic asthma, ameliorating AHR as well as lung inflammation and damage, reducing the expression of Th2 cytokines and the secretion of allergen-specific antibodies, influencing the functions of DCs, and decreasing the level of Th2 immune responses. Numerous research have examined the advantages of LF for conditions like anti-inflammation, damage to human nasal epithelial cells, and airway hyperresponsiveness induced by allergens. Our research shows that giving LF to mice orally can reduce the severity of asthma brought on by OVA. This result

suggests that LF could be easily consumed by the body to control asthma. Additionally, the LF purification from milk protein manufacturing process is an established and affordable one. A suitable chemical for airway care may be LF. These findings imply that LF might be a different therapeutic approach for the treatment of asthma.

Acknowledgement

None.

Conflict of Interest

None.

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