Journal of Nutritional Disorders & Therapy

Food Summit 2020

Vitamin A deficiency Promotes Inflammation by Induction of Type 2 Cytokines in Experimental Ovalbumininduced Asthma Murine Model

Dong Li

Abstract

Asthma is a major threat in public health as it affects more than 300 million people worldwide. Asthma, especial allergic allergen-induced asthma, is associated with airway eosinophilic inflammation and increased serum IgE level. Although there have been extensive experimental and human studies about the mechanisms of asthmatic airway inflammation inflammation and remodeling, more work remains required to completely know it. At the present, it is generally believed that the type 2 helper T cell (Th2) cytokines (including interleukin (IL)-4, IL-5, and IL-13), also known as type 2 cytokines since the major source of them are Th2 cells and type 2 innate lymphoid cells, play an essential role in the pathogenesis of asthma.

Statement of the Problem: vitamin A (VA) deficiency is one among the foremost common malnutrition conditions. Topical intelligences presented that VA shows an imperative role in the immune balance, lack of VA could result in enchanted type 2 immune response characterized by increased type 2 cytokines production and type 2 innate lymphoid cells infiltration and activation. Type 2 resistant answers shows defensive part in anti-infection, but shows pathological role in asthmatic disease. Methodology & Theoretical Alignment: In command to examine the role of VA in the asthmatic disease, we used ovalbumin-induced asthma murine model, and observed the pathological changes between mouse received VA-deficient and -sufficient diets. We also measured the type 2 cytokine expressions to reveal the potential mechanism. Findings: Our results showed that VA deficiency exacerbates ovalbumin-induced lung inflammation via induction of the sort 2 cytokine productions. Conclusion & Significance: VA deficiency, or malnutrition in further extent, may contribute to the increasing prevalence of asthma.

Given the well-established contribution of mal-nutrition to dysfunctions of system , we reasoned that VA deficiency (VAI) could also be exacerbate ovalbumin-induced asthma in mice. to guage the effect of VA in asthma, we gave male C57 mouse normal diet or VA-deficient diet from day 0; we then sensitized the mice with ovalbumin plus adjuvant or PBS on day 14; the mice were challenged with ovalbumin or PBS for 3 times on days 22, 23, and 24; and eventually , all the mice we sacrificed 48 h after the last challenge. Mice that received a VAI diet alone didn't develop lung inflammation, but in ovalbumin-sensitized and ovalbumin-challenged groups, mice from VAI group had significantly more severe lung inflammation compared to mouse with a traditional diet. VAI groups were also with higher IgE serum levels (2.98 μg/ml) compared with control groups (1.95 μg/ml) after ovalbumin challenges.

Data reported during this work reveal a previously unrecognized effect and mechanism by which VAI exacerbates ovalbumin-induced asthma in mice. VA had significant effect on the immune reaction during this asthma mouse model. VAI mice had more severe lung inflammation, more neutrophil and eosinophil infiltration to the airway, and more type 2 cytokines and IgE expressions. Although vitamin A deficiency itself doesn't induce asthmatic reaction, we found that IL-5 and IL-13 expressions were slightly enhanced, although the differences weren't significant. Nevertheless, VAI mice had significantly higher level of type 2 cytokines compared to regulate mice after challenged by ovalbumin. And neutralizing of IL-5 and IL-13 abolished the exacerbation.

Vitamin A deficiency had been considered to be linked with asthmatic diseases, serum vitamin A level was lower in patients with asthma than in healthy controls, and therefore the serum concentration of asthma was also negatively correlated with disease severity. But, the underline mechanisms are largely unknown; previous studies showed conflicted results. Ex vivo

Dong Li
Jilin University, China

studies showed that vitamin A and its metabolite retinoic acid are essential for the proliferation and cyto-toxicity of T cells, and vitamin A is required for the polarization of type 2 helper T cells. Yet, other in vivo studies reported that vitamin A is required for the sort 1 and three ILCs but not type 2 ILCs. These findings suggested that vitamin A may need different effects on helper cells from innate and adaptive immune systems. This work showed that VAI mice had increased type 2 cytokine productions, especially IL-5 and IL-13. Type 2 cytokines can enhance the IgE production from B cell via affecting the isotope switch. And, IL-5 could recruit and activate eosinophil to cause eosinophilic inflammation. Both of them are key factors of asthma. But, the precise sources of those cytokines are still needed to be investigated further; type 2 innate lymphoid cells could be one among the main contributors of those cytokines. Nevertheless, malnutrition conditions like vitamin A deficiency have sort of effects on the immune system; there are still more researches to be required so as to completely know it.

Asthmatic diseases are public health concerns especially in westernized countries, due to their associated morbidity and burdens on the health system, and therefore the prevalence of asthma has dramatically increased in westernized countries since 1970s. it's been hypothesized that these increases are due to the changing of diets. it's been well established that malnutrition is one among the most reasons for dysfunctions of system . Recent reports showed that vitamin A level is negatively linked with the danger of developing asthma. Our work provided further evidence that vitamin A play an important role in asthmatic diseases and revealed that the mechanism involved the improved production of type 2 cytokines. These findings may provide novel strategy for the prevention and coverings of asthmatic diseases.

Dong Li
Jilin University, China