JOURNAL OF FOOD QUALITY & HAZARDS CONTROL
SHAHID SADOUGHI UNIVERSITY OF MEDICAL SCIENCES & HEALTH SERVICES

Journal of Food Quality and Hazards Control 2 (2015) 1-5

Overview of Relationship of Allergic Asthma with Diet and Food Intake

R. Garcia[1], S.S. Athari[2,3*]

1. Center for Protein Studies, Faculty of Biology, University of Havana, Cuba
2. Department of Immunology, Faculty of Medical Sciences, Tarbiat Modares University, Tehran, Iran
3. Health Policy Research Center, Shiraz University of Medical Sciences, Shiraz, Iran

Article type
Review article

Keywords
Asthma
Diet
Antioxidants
Food Hypersensitivity

Received: 25 Sep 2014
Revised: 3 Nov 2014
Accepted: 11 Dec 2014

Abstract

Allergic asthma is the most common acute inflammatory airway disorder and their incidence population has exploded in the last years, being estimated that over 300 million people worldwide have the condition. Here, we present a brief overview of relationship of allergic asthma with diet and food intake. Several studies have shown that most asthma cases are due to an allergic immune response to environmental allergens. Diet affects strongly the development of asthma because the systemic inflammatory mediators are affected by a high fat diet and obesity. Thus, obesity has effect on airway function and trigger inflammation pathways. There are also strong associations between asthma and dietary antioxidants, such as vitamins, and they may protect the respiratory system from inflammation and allergic reactions. An increased prevalence of food allergy has been also observed in the last years and this problem could be a major risk factor for severe asthma. According to the findings of many researchers, specific diets could help controlling asthma, therefore therapeutic strategies for allergic asthma suggest the modification of diet, which could include antioxidant-rich foods, the avoidance of allergic food, low saturated fat intake and maintenance of weight in the healthy range.

Introduction

Allergic asthma is one of the main world health problems. It is estimated that 300 million people worldwide have allergic asthma. Asthma is the most common chronic inflammatory airway disorder with variable and reversible airflow limitation. This disorder is the major cause of respiratory morbidity and mortality. The majority of asthma cases represent the fact that an allergic immune response occurs to environmental allergens. Allergic asthma is characterized by airway inflammation, wheezing, coughing and breathlessness (Athari, 2013a; Athari, 2013b; Athari and Athari, 2014; Hasanloei and Athari, 2014).

Immune responses can be affected by diet and obesity. High fat diet has been linked to metabolic syndrome and also immune function changes. Bad diet and obesity could decrease lung function therefore increase incidence and prevalence of asthma. Several epidemiological studies have suggested that the dietary pattern associated with western cultures, is detrimental to respiratory function and health. Intake fast-foods, high in fat and low in antioxidant content increase asthma risk. Fresh fruit and vegetables consumption were associated with decreased risk of asthma. Cytokine balance and T cell subsets are altered in obesity that it could increase prevalence of asthma. This induces pro-inflammatory state and alters cytokine levels (Athari, 2013c; Peat and Li, 1999; Vries and Howie, 2009).

Therefore, to improve public health and reduce the costs that health care causes, it would be essential to undertake longitudinal, epidemiological studies to investigate effects of diet on the development of asthma. Allergic asthma which is observed in world population is mainly due to

*Corresponding author
E-mail: ss.athari@gmail.com

[Downloaded from jfqhc.ssu.ac.ir on 2026-11-09]

increased exposure to aeroallergens and westernized diet (Athari, 2013d; Mihrshahi et al., 2002). Thus, focusing on dietary explanations as one of the main causative factors for control of asthma is interested.

Effects of food vitamins and antioxidants on allergic asthma

The rising prevalence of asthma has been suggested to be linked to a falling dietary intake of antioxidants leading to an increased vulnerability of the pulmonary airways to reactive oxygen species. Moreover, an association between allergic asthma and dietary intake antioxidants such as vitamin C, vitamin E, some carotenoids, and selenium has been proposed (Fogarty et al., 2003; Schwartz and Weiss, 1994; Shahar et al., 1994). This is supported by several epidemiological studies in adults and children showing beneficial associations between dietary antioxidants and parameters of asthma.

In most people, dietary antioxidant intake of vitamins such as vitamin C and vitamin E has positively effect on the lung ventilatory function. Beneficial associations have been also demonstrated between dietary vitamin A intake and ventilatory function and respiratory symptoms (Mito et al., 2002; Shahar et al., 1994).

Airways inflammation processes, for example in asthma attack, can increase endogenously produced oxidants. Thus, antioxidants play an important role for protection of lung pathogenesis against oxidative stress and have also protective actions against inhaled toxic substances. Oxidant stress could lead to activation of two pivotal inflammatory regulators and nuclear factor kB (NFκB) gene expression. Antioxidants in the diet could be used as a scavenger of reactive oxygen, thereby with inhibiting an NFκB-mediated pathway, antioxidants could inhibit innate immune response.

There is evidence of a relationship between safe food intake and lung function. Selenium is also an important antioxidant that protects cells against oxidative damage and, together with vitamin E protects the respiratory system from inflammation and allergic reactions. Selenium is the potential factor to predispose toward development of asthma by affecting airway development and promoting Th2 differentiation and allergic sensitization (Bowler and Crapo, 2002; Caramori and Papi, 2004; Hu and Cassano, 2000; Pearson et al., 2004).

Moreover, asthma was found to be less in adults who consumed more apples that it may be result from a protective effect of flavonoids, such as anthocyanins, and phloridzin and its derivatives (Shaheen et al., 2001). According to the mentioned authors, flavonoids could reduce asthma inflammation through antioxidant, antiallergic, and antiinflammatory properties, although an antioxidant activity of nonflavonoid phenolic compounds found in apples is also possible.

On the other hand, lycopene, a vegetal pigment found in tomatoes and other fruits and vegetables, may have a protective effect on asthma by reducing the bronchoconstriction in the airways of asthmatics and decreasing eosinophilic infiltrate and mucus production in the lungs. Safe antioxidants of the food, when consumed in combination with the other essential nutrients in the food, have the most potent effects in these disorders (Caramori and Papi, 2004; Greene, 1995; Pearson et al., 2004; Picado et al., 2001).

The pathophysiological features of allergic asthma are thought to result from the aberrant expansion of $CD4^+$ T cells producing different cytokines. Consequently and in agreement with the positive effect formerly mentioned for the antioxidant vitamins, vitamin E has the ability to inhibit circulating pro-inflammatory mediators and cytokines and also reduces the production of TNF-α, IL-1β, IL-6 and IL-8. Moreover, lycopene and β-carotene could inhibit NFκB, although lycopene can inhibit the oxidative damage more efficiently (Hatch, 1995).

Despite the positive antioxidant effect of dietary vitamins, early infant multivitamin supplementation has been associated with increased risk for food allergy and asthma (Milner et al., 2004). According to Moreno-Macias and Romieu (2014), there have been some longitudinal studies of maternal or child dietary or vitamin/supplement antioxidant intake and development of asthma/allergy. Thus, the potential benefits and risks of vitamin supplements might be considered in those situations in which vulnerable populations have deficiency or reduced access to dietary antioxidants, or high exposure to environmental sources of oxidants (Moreno-Macias and Romieu, 2014).

Obesity and diet relationship with asthma

Several studies have reported a relationship between asthma and obesity, both of which are having a dramatic increase in prevalence (Alam et al., 2007; Williams et al., 2013). The pathogenesis of their linkage is complex; obesity causes metabolic, mechanical, and immunological changes that can affect the beginning of airways inflammation. Obesity is associated with weak response to standard controller medications and therefore, the treatment of asthma in obesity can be challenging process.

Obesity affects lung health causing respiratory muscle weakness, reduced exercise ability and dyspnea. The mechanisms leading to these effects are not only mechanical or anatomical, but also related to inflammatory responses (Nieman et al., 1999; Raatz et al., 2001).

Obesity and a high fat diet promote an innate immune inflammatory response that may lead to airway inflammation and asthma in susceptible individuals. Dietary fat and obesity predispose the activation of inflammation pathways, with an increase of the circulating levels of various pro-inflammatory mediators. The systemic inflammatory medi-

[Downloaded from jfqhc.ssu.ac.ir on 2026-11-09]

ators that are affected by a high fat diet influence airway function and followed alteration in asthma (Alam et al., 2007; Bourgeois et al., 1983; Lundback et al., 1991; Mihrshahi et al., 2003; Oddy et al., 2004; Schwartz and Weiss, 1990). Several hours after consumption of a high fat containing-meal, the concentrations of IL-6 -as well as those of soluble IL-6 receptor in serum- are elevated. It has been reported that the level of IL-6 in plasma is a clinically relevant marker of innate immune activation in asthma (Alam et al., 2007; Calder, 2003; Nieman et al., 1999; Raatz et al., 2001). The circulating TNF-α levels are also increased in obesity allergic asthma and may be involved in asthma occurrence as well, since TNF-α-deficient obese mice have decreased airway reactivity compared to other obese mice (Bourgeois et al., 1983; Lundback et al., 1991; Mihrshahi et al., 2003; Williams et al., 2013). Other mediators such as IL-8, and plasminogen activator inhibitor-1, are also elaborated by adipose tissue, and they could also have direct and indirect effects on the airway (Pradeepan et al., 2013).

Moreover, adipokines are likely to play a role in the development of lung disease in obesity, thus being involved in the progression of asthma in obese individuals. Adipokine leptin levels are increased in subjects with asthma and impaired lung function and it have been shown to increase airway reactivity even in lean mice (Shore et al., 2005). The airway epithelial cells bear receptors for leptin, and visceral leptin levels are significantly related to airway responsiveness in obesity. Several studies show that leptin is involved in lung development and fibrotic responses suggesting a role for leptin in airway remodeling (Jain et al., 2011; Kirwin et al., 2006).

Summarizing, obesity leads to several changes in normal lung physiology and immune function, having an important impact on the pathogenesis of lung disease in this population. Therefore, approaches combining pharmacologic and non-pharmacologic therapies including weight loss, dietary interventions or exercise, are an alternative for this population.

Food allergy and asthma

Food allergy is one of the main public health problem worldwide, and its prevalence appears to be rising. There is a strong relationship between food allergy and asthma, and thus, an increased prevalence of food allergy risk categories is observed in individuals with diagnosed asthma. Moreover, food allergies could be a major risk factor for severe asthma and life-threatening asthma episodes. They both are atopic diseases and the former may be a risk factor for life-threatening asthma and contrariwise.

According to literature, the usual allergenic foods are eggs, cow´s milk, soy, wheat, nuts, fish, shrimp and other shellfishes. There is an association between food sensitivity and the development and exacerbation of asthma that could even be mortal (Chahine and Bahna, 2010; Prescott et al., 2010; Sicherer and Sampson, 2010). In addition to allergenic foods, additives could be trigger for asthma. Nowadays, thousands of chemical compounds, such as monosodium glutamate, tartrazine, nitrite and sulphites are commonly added to foods with the aim of enhancing their flavor or acting as preservatives. However, some of these food additives seem to be a problem for some asthmatic patients (Bischoff and Crowe, 2005; Bock, 1992; Sicherer and Sampson, 2006).

The link between food sensitization, asthma, and the real prevalence of food allergy in the asthmatic population is unknown, but food sensitization is a marker for more severe asthma morbidity and asthma is the most common respiratory symptom of food-induced anaphylaxis. Three different mechanisms of food-induced allergic asthma have been proposed: 1) Small particles of ingested food may be inhaled into the airway during mastication or with gastroesophageal reflux. Therefore, food allergens would be able to stimulate airway directly; 2) Allergenic proteins are potentially absorbed from the gut and could reach the lungs via the circulation system; 3) Allergenic proteins may indirectly act on the lower respiratory tract, via inflammatory mediators released from the skin or gastrointestinal tract. These mediators circulate in the blood system and have an inflammatory effect in the lung and the body (Bischoff and Crowe, 2005; Bock, 1992; Kattan et al., 2006; Lowe et al., 2007; Novembre et al., 1988; Sampson, 2001; Sicherer and Sampson, 2006).

Food allergy in contrast to food intolerance, is an immunological hypersensitivity reaction (IgE- or non IgE-mediated) that may be an important factor in asthmatic patients. Many food allergens have been characterized at a molecular level, which allows understanding the immunopathogenesis of food allergy and might lead to diagnostic and therapeutic approaches (Breiteneder et al., 1995; Costa et al., 2014; Leung et al., 1998; Pahr et al., 2012).

However, currently management of food allergies consists of identifying the allergic food or additive, and avoiding direct and indirect exposures to them or initiating a therapy in case of an unintended ingestion (Björkstén, 2005; Costa et al., 2014).

Conclusion

Various dietary antioxidant deficiencies increase susceptibility to oxidative stress, inflammation and asthma. Low vitamins intake has been associated with a greater decline in lung function in response to allergic triggers. The ideal therapeutic strategy for allergic asthma may be modification of dietary intake, including antioxidant-rich foods, avoidance of allergic food, low saturated fat intake and maintenance of weight in the healthy range.

[Downloaded from jfqhc.ssu.ac.ir on 2026-11-09]

Conflicts of interest

None declared.

References

Alam I., Lewis K., Stephens J.W., Baxter J.N. (2007). Obesity, metabolic syndrome and sleep apnoea: all pro-inflammatory states. *Obesity Reviews*. 8: 119-127.

Athari S.S. (2013a). Best treatment for allergic asthma with traditional herbal medicine: a brief report. *Pharmacology*. 82: 586-599.

Athari S.S. (2013b). Immune response shifting of asthma in aging. *Middle-East Journal of Scientific Research*. 13: 489-498.

Athari S.S. (2013c). Inflammation, asthma and tumor. *Bulletin of Environment, Pharmacology and Life Sciences*. 2: 98-100.

Athari S.S. (2013d). Traditional medicine for asthma. *Advances in Biological Research*. 7: 112-113.

Athari S.S., Athari S.M. (2014). The importance of eosinophil, platelet and dendritic cell in asthma. *Asian Pacific Journal of Tropical Disease*. 4: S41-S47.

Bischoff S., Crowe S.E. (2005). Gastrointestinal food allergy: new insights into pathophysiology and clinical perspectives. *Gastroenterology*. 128: 1089-1113.

Björkstén B. (2005). Genetic and environmental risk factors for the development of food allergy. *Current Opinion in Allergy and Clinical Immunology*. 5: 249-253.

Bock S.A. (1992). Respiratory reactions induced by food challenges in children with pulmonary disease. *Pediatric Allergy and Immunology*. 3: 188-194.

Bourgeois F., Alexiu A., Lemonnter D. (1983). Dietary-induced obesity: effect of dietary fats on adipose tissue cellularity in mice. *British Journal of Nutrition*. 49: 17-26.

Bowler R.P., Crapo J.D. (2002). Oxidative stress in allergic respiratory diseases. *Journal of Allergy and Clinical Immunology*. 110: 349-356.

Breiteneder H., Hoffmann-Sommergruber K., O'Riordain G., Susani M., Ahorn H., Ebner C., Kraft D., Scheiner O. (1995). Molecular characterization of Api g 1,the major allergen of celery (*Apium graveolens*), and its immunological and structural relationships to a group of 17-kDa tree pollen allergens. *European Journal of Biochemistry*. 233: 484-489.

Calder P.C. (2003). Polyunsaturated fatty acids and cytokine profiles: a clue to the changing prevalence of atopy? *Clinical and Experimental Allergy*. 33: 412-415.

Caramori G., Papi A. (2004). Oxidants and asthma. *Thorax*. 59: 170-173.

Chahine B.G., Bahna S.L. (2010). The role of the gut mucosal immunity in the development of tolerance versus development of allergy to food. *Current Opinion in Allergy and Clinical Immunology*. 10: 394-399.

Costa J., Carrapatoso I., Oliveira M.B., Mafra I. (2014). Walnut allergens: molecular characterization, detection and clinical relevance. *Clinical and Experimental Allergy*. 44: 319-341.

Fogarty A., Lewis S.A., Scrivener S.L., Antoniak M., Pacey S., Pringle M., Britton J. (2003). Oral magnesium and vitamin C supplements in asthma: a parallel group randomized placebo-controlled trial. *Clinical and Experimental Allergy*. 33: 1355-1359.

Greene L.S. (1995). Asthma and oxidant stress: nutritional, environmental, and genetic risk factors. *Journal of the American College of Nutrition*. 14: 317-324.

Hasanloei M.A.V., Athari S.S. (2014). Proper Care of Allergic Asthma before Hospitalization. *Journal of Allergy and Therapy*. 5: 161.

Hatch G.E. (1995). Asthma, inhaled oxidants, and dietary antioxidants. *The American Journal of Clinical Nutrition*. 61: 625S-630S.

Hu G., Cassano P.A. (2000). Antioxidant nutrients and pulmonary function: the third National Health and Nutrition Examination Survey (NHANES III). *American Journal of Epidemiology*. 151: 975-981.

Jain M., Budinger G.R., Lo A., Urich D., Rivera S.E., Ghosh A.K., Gonzalez A., Chiarella S.E., Marks K., Donnelly H.K., Soberanes S., Varga J., Radigan K.A., Chandel N.S., Mutlu G.M. (2011). Leptin promotes fibroproliferative acute respiratory distress syndrome by inhibiti ng peroxisome proliferator-activated receptor-γ. *American Journal of Respiratory and Critical Care Medicine*. 183: 1490-1498.

Kattan J.D., Srivastava K.D., SampsonH.A., Li X. (2006). Pharmacologic and immunologic effects of individual herbs of food allergy herbal formula 2 in a murine model of peanut allergy. *Journal of Allergy and Clinical Immunology*. 117: S34.

Kirwin S.M., Bhandari V., Dimatteo D., Barone C., Johnson L., Paul S., Spitzer A.R., Chander A., Hassink S.G., Funanage V.L. (2006). Leptin enhances lung maturity in the fetal rat. *Pediatric Research*. 60: 200-204.

Leung P.S., Chen Y.C., Gershwin M.E., Wong S.H., Kwan H.S., Chu K.H.(1998).Identification and molecular characterization of *Cha -rybdis feriatus* tropomyosin, the major crab allergen. *Journal of Allergy and Clinical Immunology*. 102: 847-852.

Lowe A.J., Hosking C.S., Bennett C.M., Carlin J.B., Abramson M.J., Hill D.J., Dharmage S.C. (2007). Skin prick test can identify eczematous infants at risk of asthma and allergic rhinitis. *Clinical and Experimental Allergy*. 37: 1624-1631.

Lundback B., Nystrom L., Rosenhall L., Stjernberg N. (1991). Obstructive lung disease in northern Sweden: respiratory symptoms assessed in a postal survey. *European Respiratory Journal*. 4: 257-266.

Mihrshahi S., Peat J.K., Marks G.B., Mellis C.M., Tovey E.R., Webb K., Leeder S.R. (2003). Eighteen-month outcomes of house dust mite avoidance and dietary fatty acid modification in the Childhood Asthma Prevention Study (CAPS). *Journal of Allergy and Clinical Immunology*. 111: 162-168.

Mihrshahi S., Vukasin N., Forbes S., Wainwright C., Krause W., Ampon R., Peat J. (2002). Are you busy for the next 5 years? Recruitment in the Childhood Asthma Prevention Study (CAPS). *Respirology*. 7: 147-151.

Milner J.D., Stein D.M., McCarter R., Moon R.Y. (2004). Early infant multivitamin supplementation is associated with increased risk for food allergy and asthma. *Pediatrics*. 114: 27-32.

Mito N., Kitada C., Hosoda T., Sato K. (2002). Effect of diet-induced obesity on ovalbumin-specific immune response in a murine asthma model. *Metabolism*. 51: 1241-1246.

Moreno-Macias H., Romieu I. (2014). Effects of antioxidant supplements and nutrients on patients with asthma and allergies. *Journal of Allergy and Clinical Immunology*. 133: 1237-1244.

Nieman D.C., Henson D.A., Nehlsen-Cannarella S.L., Ekkens M., Utter A.C., Butterworth D.E., Fagoaga O.R. (1999). Influence of obesity on immune function. *Journal of The American Dietetic Association*. 99: 294-299.

Novembre E., Martino M.D., Vierucci A. (1988). Foods and respiratory allergy. *Journal of Allergy and Clinical Immunology*. 81: 1059-1065.

Oddy W.H., De Klerk N.H., Kendall G.E., Mihrshahi S., Peat J.K. (2004). Ratio of omega-6 to omega-3 fatty acids and childhood asthma. *Journal of Asthma*. 41: 319-326.

Pahr S., Constantin C., Mari A., Scheiblhofer S., Thalhamer J., Ebner C., Vrtala S., Mittermann I., Valenta R. (2012). Molecular characterization of wheat allergens specifically recognized by patients suffering from wheat-inducedrespiratory allergy. *Clinical and Experimental Allergy*. 42: 597-609.

Pearson P.J.K., Lewis S.A., Britton J., Fogarty A. (2004). Vitamin E supplements in asthma: a parallel group randomised placebo controlled trial. *Thorax*. 59: 652-656.

Peat J.K., Li J. (1999). Reversing the trend: reducing the prevalence of asthma. *Journal of Allergy and Clinical Immunology*. 103: 1-10.

Picado C., Deulofeu R., Lleonart R., Agusti M., Mullol J., Torra M., Quintó L. (2001). Dietary micronutrients/antioxidants and their relationship with bronchial asthma severity. *Allergy*. 56: 43-49.

Pradeepan S., Garrison G., Dixon A.E. (2013). Obesity in asthma: approaches to treatment. *Current Allergy and Asthma Reports*. 13: 434-442.

[Downloaded from jfqhc.ssu.ac.ir on 2026-11-09]

Prescott S.L., Bouygue G.R., Videky D., Fiocchi A. (2010). Avoidance or exposure to foods in prevention and treatment of food allergy? *Current Opinion in Allergy and Clinical Immunology*. 10: 258-266.

Raatz S.K., Bibus D., Thomas W., Kris-Etherton P. (2001). Total fat intake modifies plasma fatty acid composition in humans. *The Journal of Nutrition*. 131: 231-234.

Sampson H.A. (2001). Utility of food-specific IgE concentrations in predicting symptomatic food allergy. *Journal of Allergy and Clinical Immunology*. 107: 891-896.

Schwartz J., Weiss S.T. (1990). Dietary factors and their relation to respiratory symptoms the second national health and nutritin examination survey. *American Journal of Epidemiology*.132:67-76.

Schwartz J., Weiss S.T. (1994). Relationship between dietary vitamin C intake and pulmonary function in the First National Health and Nutrition Examination Survey (NHANES I). *The American Journal of Clinical Nutrition*. 59: 110-114.

Shahar E., Folsom A.R., Melnick S.L., Tockman M.S., Comstock G.W., Gennaro V., Szklo M. (1994). Dietary n-3 polyunsaturated fatty acids and smoking-related chronic obstructive pulmonary disease. *New England Journal of Medicine*. 331: 228-233.

Shaheen S.O., Sterne J.A., Thompson R.L., Songhurst C.E., Margetts B.M., Burney P.G. (2001). Dietary antioxidants and asthma in adults: population-based case-control study. *American Journal of Respiratory and Critical Care Medicine*. 164: 1823-1828.

Shore S.A., Schwartzman I.N., Mellema M.S., Flynt L., Imrich A., Johnston R.A. (2005). Effect of leptin on allergic airway responses in mice. *Journal of Allergy and Clinical Immunology*. 115: 103-109.

Sicherer S.H., Sampson H.A. (2006). Food allergy. *Journal of Allergy and Clinical Immunology*. 117: S470-S475.

Sicherer S.H., Sampson H.A. (2010). Food allergy. *Journal of Allergy and Clinical Immunology*. 125: S116-S125.

Vries A., Howie S.E.M. (2009). Diet and asthma—Can you change what you or your children are by changing what you eat? *Pharmacology and Therapeutics*. 122: 78-82.

Williams A.S., Chen L., Kasahara D.I., Si H., Wurmbrand A.P., Shore S.A. (2013). Obesity and airway responsiveness: role of TNFR2. *Pulmonary Pharmacology and Therapeutics*. 26: 444-454.

[Downloaded from jfqhc.ssu.ac.ir on 2026-11-09]