



Significance of nutrition in respiratory allergic diseases: a review of scientific knowledge

IVANA SABOLIĆ PIPINIĆ
JELENA MACAN
BOŽICA KANCELJAK-MACAN

Institute for Medical Research
and Occupational Health
Unit for Occupational Health
and Environmental Medicine
Ksaverska cesta 2
10000 Zagreb, Croatia

Correspondence:

Ivana Sabolić Pipinić
Unit for Occupational Health
and Environmental Medicine
Institute for Medical Research
and Occupational Health
Ksaverska cesta 2
10000 Zagreb, Croatia
E-mail: ivanasp@imi.hr

Abbreviations:

PUFA – polyunsaturated fatty acids
AA – arachidonic acid
EPA – eicosapentaenoic acid
LT – leukotriene
PG – prostaglandin

Key words: antioxidants, flavonoids,
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fatty acids, trace elements, vitamins

Abstract

A significant increase in the occurrence of allergic respiratory diseases has been perceived in the world in the last several decades. One of the presumed causes of this phenomenon is the contemporary altered dietary pattern. Many studies investigated the possible inflammatory or anti-inflammatory effects of various nutrients in the pathophysiology of respiratory allergies. Recent epidemiological studies reported an association between a high intake of ω -6 and a low intake of ω -3 polyunsaturated fatty acids (PUFA-s) with the elevated prevalence of respiratory allergies. ω -6 PUFA-s are precursors of strong inflammatory mediators which promote allergic inflammation, whereas metabolites of ω -3 PUFA-s are weak inflammatory mediators. However clinical trials of ω -3 supplementation failed to show substantial beneficial results. A high dietary intake of salt could have a bronchoconstrictive effect in the pathogenesis of asthma, whereas magnesium is considered to act bronchodilatively, yet most research in asthmatic patients has not proven these proposed impacts. Antioxidants, such as vitamin C, E, A, flavonoides, isoflavones and some trace elements (selenium, zinc, manganese, copper) are relevant in diminishing oxidative stress. Despite epidemiological and some clinical evidence of a protective antioxidant effect against the development of respiratory allergies, interventional studies mainly have not confirmed significant benefit of antioxidant supplementation therapy. Due to a large amount of controversial scientific evidence regarding the possibility of preventing and treating respiratory allergies with a modified diet, there is a need for further research in this field.

INTRODUCTION

Over the past 40 years there has been a dramatic increase in the prevalence of allergic respiratory disease around the world (1). This phenomenon, particularly present in industrial Western countries, is considered to be the result of many environmental and lifestyle changes of human population. Environmental changes include higher outdoor (SO_2 , O_3 , NO_x) and indoor (cigarette smoke) air pollution. Additionally, the increased development of respiratory allergic diseases could partly be a result of the lack of early childhood infections due to vaccination, increase in hygiene, improvement in nutrition, overuse of antibiotics and decrease in family size (2). Recent findings emphasize that lifestyle changes related to nutritional composition and dietary habits could be strongly implicated in the observed elevated prevalence of allergic asthma, allergic rhinitis and hay fever (1). The term Western diet reflects a dietary pattern in developed countries which has extensively

altered in the composition of nutritional products in the last five decades. Nutritional Western diet consists of elevated consumption of refined sugars, fats and salt and a reduced consumption of fresh fruit, vegetables, fresh fish and red meat (3). Fresh fruit and vegetables are main sources of vitamins, minerals and fiber content, while fresh fish and red meat are important sources of antioxidants such as selenium, zinc and copper (1, 3, 4). It is presumed that an adequate diet rich in products that contain substances with a beneficial impact on human health could inhibit, arrest or even reverse the chain of events in the pathophysiology of respiratory allergic diseases. Substances with a protective effect against the risk of respiratory allergy development are supposed to have either anti-inflammatory or antioxidative characteristics. ω -3 polyunsaturated fatty acids and some trace elements such as magnesium have been reported to have anti-inflammatory characteristics, whereas antioxidative protective effects are attributed to vitamins A, C, E, flavonoids, isoflavones and some trace elements (e.g. zinc, manganese, selenium) (Table 1). A diet deficient in the mentioned protective substances and/or a high dietary intake of proinflammatory and prooxidative substances could possibly increase individual susceptibility to adverse environmental exposures such as allergens, infectious agents and air pollution (4, 5, 6). Likewise, it has been speculated in a number of scientific publications that a high dietary intake of proinflammatory ω -6 polyunsaturated acids or a consumption of products containing prooxidative agents, e.g. excessive levels of iron or copper, increase the risk of developing allergic respiratory diseases (2).

This review will elaborate the dietary factors important for the cause and prevention of respiratory allergy and the possible aspects of therapy with dietary manipulation in allergic respiratory diseases. Scientific results, reviewed in this article, will be mostly based on epidemiological studies such as cross-sectional, case-control and longitudinal studies and on the reports of clinical studies among patients with allergic respiratory disorders.

Polyunsaturated fatty acids: ω -6 versus ω -3

Numerous scientific reports have assumed that the consumption of essential polyunsaturated fatty acids is related to the occurrence and severity of asthma, allergic rhinitis and hay fever. Essential polyunsaturated fatty acids (PUFA-s) cannot be synthesized in the human organism and, since they are the compounds of phospholipid membranes (7), they must be derived from nutrition. Today's Western diet contains less amounts of food products rich in saturated fat (lard, dairy fats) than before, and they have been mostly replaced by food substances abundant in ω -6 PUFA-s (vegetable oils, margarine, meat). This dietary shift, intended to reduce cardiovascular diseases, simultaneously led to a decreased consumption of food products rich in ω -3 PUFA-s (oily fish, leafy vegetables) which are believed to have a beneficial impact on human health (8, 9, 10). ω -3 PUFA-s are reported to

have anti-inflammatory biological actions, whereas ω -6 PUFA-s generally have pro-inflammatory actions and therefore it is speculated that a modification in the amount of these PUFA-s in nutrition could modulate the inflammatory process in allergic respiratory diseases. It has been emphasized that the appropriate ratio of ω -6/ ω -3 PUFA-s, and not the absolute higher intake of ω -3 PUFA-s could have beneficial impact on human health (11, 12). Throughout the years researchers estimated that the diet on which human beings evolved and their genetic patterns were established had the ratio of ω -6/ ω -3 PUFA-s approximately 1/1 (11) whereas the ratio of ω -6/ ω -3 PUFA-s in a typical Western diet is presumed to be 15/1 or even higher (12). Such extremely different ratios of ω -6 and ω -3 PUFA-s of the past and present dietary trends and the inappropriate amounts ω -6 PUFA-s in today's nutrition are considered to be responsible for the higher occurrence and development of respiratory allergic diseases in our era (13). Furthermore, a recent review on the importance of the appropriate ratio of ω -6/ ω -3 PUFA-s in chronic diseases indicated eligible effects of the 5/1 ratio on patients with asthma, whereas a ratio of 10/1 had adverse consequences (12).

ω -6 and ω -3 PUFA-s, integrated in the phospholipid cell membranes, can be converted by various inflammatory enzymes into metabolites with diverging inflammatory potentials (Figure 1). Most commonly present ω -6 PUFA in nutrition is linoleic acid (margarine, vegetable oils). The consumed linoleic acid is desaturated and elongated by enzymes into arachidonic acid (AA). AA is a substrate for pro-inflammatory enzymes, cyclooxygenases and lipoxygenases, which convert AA into various highly active inflammatory mediators, such as 4-series leukotrienes (LT₄) and 2-series prostaglandins (PG₂) (7, 14). In the pathophysiology of allergic diseases, especially asthma, LT₄ play a major role by increasing vascular permeability, inducing chemotaxis of leukocytes, accelerating the production of reactive oxygen species and various cytokines, promoting bronchoconstriction and mucus secretion (15). A specific prostaglandin PGE₂ as a proinflammatory mediator induces the activity of an inflammatory enzyme cyclooxygenase-2 and the production of a pro-inflammatory cytokine IL-6 (16). Some research indicates that PGE₂ has immunomodulatory properties on T- lymphocytes by promoting the T_H2 phenotype which plays a dominant role in the pathophysiology of allergic diseases (8, 9, 17). However, there is some opposing evidence about PGE₂ suggesting, additionally, its antiinflammatory and bronchodilative properties in the lung (18). Conversely, α -linolenic acid, the most common ω -3 PUFA in the diet, is converted into eicosapentaenoic acid (EPA) by the same series of enzymes (desaturases and elongases) that are in charge of the synthesis of AA from linoleic acid. The products of EPA's enzymatic conversion are 3-series prostaglandins (PG₃), 5-series leukotrienes (LT₅) and other inflammatory mediators which possess low biological activity (7, 14). The chemotactic potency of LT₅ is 10–100 times lower than of LT₄ (15) while the bronchoconstrictive potency of LT₅ is two

TABLE 1

Nutrients associated with allergic respiratory diseases*

Nutrients	Dietary sources	Mechanisms of effect
ω -6 PUFA	margarine, sunflower oil, corn oil	– pro-inflammatory effect: increases LT_4 and PG_2 production
ω -3 PUFA	oily fish, shellfish, leafy vegetables	– anti-inflammatory effect: decreases LT_4 and PG_2 production
Sodium	salt	– bronchoconstrictive effect
Magnesium	nuts, cereal grains, legumes, seafood	– bronchodilative effect – anti-inflammatory effect
Zinc	cereal grains, dairy products, animal products	– antioxidant cofactor of SOD
Copper	seafood, nuts, cereals, legumes, dried fruits	– antioxidant cofactor of SOD – in high quantities pro-oxidative effect
Manganese	nuts, shellfish, soya, dark chocolate	– antioxidant cofactor of SOD
Selenium	seafood, animal products	– antioxidant cofactor of glutathion peroxidase – detoxification of toxic metals – DNA repair – immunomodulatory effect – antiinflammatory effect
Vitamin C	fresh fruits and vegetables	– antioxidant – regenerates oxidized vitamin E – anti-inflammatory effect – bronchodilative effect
Vitamin E	eggs, vegetables, vegetable and seed oils	– antioxidant – immunomodulatory effect
Vitamin A & carotenoids	liver, egg yolk, milk fat, fish oils, red, orange, green, and yellow fruits and vegetables	– antioxidants
Flavonoids	apples, pears, red wine, green, white and black tea, dark chocolate	– antioxidants – chelators of toxic metals – immunomodulatory effect – anti-inflammatory effect
Isoflavones	soya, legumes	– antioxidants – antiinflammatory effect

PUFA, polyunsaturated fatty acid; LT_4 , leukotriene 4; PG_2 , prostaglandin 2; SOD, superoxid-dismutase; DNA, deoxyribonucleic acid. *Modified according to (4).

times lower than of LT_4 (19). In addition, prostaglandin PGE_3 much less induces cyclooxygenase-2 expression and IL-6 secretion compared to PGE_2 (16). Since α -linolenic acid and linoleic acid compete for the same enzymatic substrate positions, it is evident that α -linolenic acid competitively inhibits AA production and ultimately reduces the synthesis of strong inflammatory mediators of AA conversion. In addition to that, EPA, the α -linolenic metabolite, competes with AA as a substrate for inflammatory enzymes and consequently directly inhibits the generation of AA metabolites (7, 13).

There is evidence that PUFA-s can also modulate the activity of a transcription factor that plays a major role in

various inflammatory signaling pathways, nuclear factor κB (NF- κB). It controls the expression of a variety of genes which encode inflammatory mediators such as cytokines, chemokines, adhesion molecules and inducible effector enzymes (e.g. cyclooxygenase-2) (20). It is presumed that NF- κB is an important factor in the pathophysiology of asthma (13) since there is some evidence that macrophages of induced sputum and bronchial epithelial cells from stable asthmatic patients have an increased activity of NF- κB in comparison to healthy subjects (21). It has been reported that ω -3 PUFA-s inhibit NF- κB activity directly by decreasing the degradation of its inhibitory unit I κB (22, 23) and indirectly by decreas-

ing the production of AA metabolites which have been demonstrated to activate NF- κ B (24). In contrast, experimental studies did not show increased activity of NF- κ B in the presence of EPA (24), and there is some evidence that EPA decreases the endotoxin induced activation of NF- κ B in human monocytes (23). These observations could explain EPA's ability to reduce gene expression of cyclooxygenases and inflammatory mediators via inhibition of the proinflammatory transcription factor NF- κ B. Increased dietary intake of ω -3 rich products could therefore result in increased concentrations of these fatty acids in phospholipid cellular membranes and consequently, through the described mechanisms of the reduced inflammatory mediator production, lead to a beneficial, antiinflammatory outcome (25). Epidemiological and clinical studies confirmed these properties of PUFA-s, indicating that serum IgE levels are positively associated with ω -6 PUFA-s and negatively with serum EPA levels (26). Fish oil supplementation rich in ω -3 PUFAs has been shown to decrease the production of PGE₂ (27), LTB₄ (28), and other pro-inflammatory mediators (29) and to induce the production of LTB₅ in inflammatory cells (30). Likewise, another study demonstrated that high daily EPA doses increase LT₅ generation and reduce AA, LT₄ and PGE₂ generation by polymorphonuclear and mononuclear leukocytes in asthmatic patients (31).

The effect of ω -6 and ω -3 PUFA intake on the incidence, prevalence and severity of respiratory allergic diseases has been evaluated in a number of epidemiological and interventional studies with controversial results. Many epidemiological studies, mostly cross-sectional and case-control studies, reported positive association between a high ω -6 PUFA intake and a low ω -3 PUFA intake, and the prevalence, incidence or the severity of asthma (9, 32, 33), and allergic rhinitis (34, 35, 36) in children (37) and adults. On the other hand, a number of population studies reported no association between increased consumption of ω -3 and the risk of allergic respiratory disease (38, 39, 40).

A significant protective role of ω -3 PUFA-s in the incidence and severity of respiratory allergy symptoms was reported in a prospective study only for children (41). Therefore, there is a presumption that increased ω -3 PUFA intake in early childhood could lead to a protective effect of ω -3 PUFAs which extends into later life (41). This observation could encourage the recommendation for increased consumption of products rich in ω -3 in early childhood.

A number of reported clinical trials regarding the effect of ω -3 PUFA-s on the severity of allergic respiratory disorders has also had controversial results. There are studies with disappointing outcomes and no evidence for recommendation of ω -3 PUFA-s as a dietary supplement (42, 43, 44). These studies failed to show clinical benefit of ω -3 PUFA or EPA supplementation, but they were able to show a substantial suppression of neutrophil chemotaxis and leukotrien production in treated patients (42, 43). On the contrary, some studies found the evidence of a clinical benefit in asthmatic patients following

ω -3 PUFA supplementation due to the positive effect on lung function, decreased bronchial hyperreactivity, decreased concentrations of airway inflammation markers in exhaled air, and decreased reported daytime wheeze (45, 46, 47). The studies of Okamoto (45, 46) also suggest that asthmatic patients differently manifest susceptibility for a positive ω -3 effect which was speculated to be genetically predisposed (12). This assumption emerged from the observation that ω -3 PUFA supplementation differently modified the leukotrien profile among asthmatic patients (46).

Electrolytes: Sodium & Magnesium

Certain substances in nutrition that are implicated in the development of allergic respiratory diseases, particularly asthma, are sodium and magnesium. These minerals have opposing effects on the airway smooth muscle cells and it has been postulated that a diet rich in sodium and low in magnesium could be a risk factor for airway allergic diseases (2, 6, 17).

Sodium. It appears that individuals with airway hyperresponsiveness have an increased sodium influx into airway smooth muscle cells resulting in their sustained hyperpolarization. This increased level of hyperpolarization could contribute to the airway hyperreactivity to specific antigen stimuli in asthma (17, 49). There is evidence that a diet rich in sodium could predispose the development of allergic airway hyperreactivity (50, 51, 52), yet research has not provided conclusive proof that sodium restriction improves asthma (17, 53, 54).

Magnesium. Magnesium maintains a stable electric potential across cell membranes and inhibits colinergic neuromuscular transmission and therefore could have a direct relaxing effect on bronchial smooth muscle, producing airway dilatation (17, 55, 56). Magnesium also has antiinflammatory and indirect bronchodilative properties by stabilizing mast cells and T-lymphocytes, and stimulating the generation of nitric oxide and prostacyclin (56). Reported population studies provide evidence of a protective role of magnesium against asthma (6) but placebo-controlled trials of magnesium supplementation have not confirmed beneficial results (57, 58).

Antioxidants

Oxidative stress is considered to play a substantial role in the pathophysiology of respiratory allergic diseases (2, 59). Oxidative stress arises when the production of reactive oxygen species (ROS) exceeds the capacity for their elimination. Reactive oxygen species such as superoxide radical (O₂⁻), hydroxyl radical (OH[•]) and hydrogen peroxide (H₂O₂) contain oxygen atoms with one or more unpaired electrons, generated by either endogenous or exogenous sources. Endogenous production of ROS is mostly derived from aerobic metabolism or inflammation in the organism. Exogenous ROS (NO_x, O₃) emerge in the tissue of the respiratory system from polluted air, e.g. cigarette smoke. ROS promote the inflammatory process by inducing the release of proinflammatory me-

diators (cytokines, chemokines, eicosanoid metabolites) and activating gene expression of primal inflammatory regulators such as nuclear factor κ B (60).

Oxidative stress is mitigated by enzymatic and non-enzymatic scavenging mechanisms of ROS. Enzymatic antioxidant defense consists of three main groups of enzymes: 1) superoxide dismutases (SOD), 2) catalases (CAT), 3) glutathion peroxidases. Some of these antioxidative enzymes, specifically superoxide dismutases and glutathion peroxidases, need cofactors for their activity. Zinc (Zn), copper (Cu) and manganese (Mn) are cofactors of two types of superoxide dismutases, Cu/Zn-SOD and Mn-SOD, while selenium (Se) is the cofactor of glutathion peroxidases (2). These cofactors are derived from various nutritional products in the diet. Additional elimination of ROS is accomplished by extracellular non-enzymatic antioxidants which includes vitamins C, E, A and carotenoids together with flavonoids and isoflavones (2, 4, 5). The above mentioned antioxidative agents (cofactors, vitamins, flavonoids and isoflavones) diminish oxidative stress either through common mechanisms or unique functions which include anti-inflammatory properties. Since these enzymatic and non-enzymatic antioxidants are mostly derived from various nutritional substances, the possibility of modifying oxidative stress and by it allergic inflammation with a particular dietary intake has been widely studied.

Enzymatic antioxidants

Zinc, copper and manganese. Superoxid dismutases (Cu/Zn-SOD and Mn-SOD) are intracellular or extracellular scavengers of ROS, and a dietary deficiency in their cofactors, Zn, Cu or Mn could diminish SOD activity and therefore decrease antioxidant protection. Zinc, besides its antioxidant properties as a cofactor, has the ability to displace redox active iron which promotes oxidative stress from macromolecular binding sites (2, 61). Therefore a high dietary intake of iron and a low intake of zinc could enhance oxidative damage and possibly increase the risk of allergic respiratory diseases. Furthermore, there is evidence that zinc has specific anti-inflammatory properties due to the findings that a Zn deficiency could be a trigger for NF- κ B activation (62). Copper, in spite of its antioxidative role as an enzyme cofactor, is a powerful redox active transition metal and therefore high intake of Cu (contaminated drinking water) should be considered as a possible risk for asthma (2). One study evaluated plasma Se, Mn, Cu, and Zn concentrations in asthmatic patients and healthy subjects, reporting lower levels of only Mn in asthmatic subjects compared to healthy subjects (63). To our knowledge, dietary supplementation of these trace elements in patients with allergic respiratory diseases has not been studied except for one study concerning the supplementation of Zn in asthmatic subjects which showed no beneficial results (64).

Selenium. Selenium, as a cofactor of glutathion peroxidase, achieves its antioxidative effects by reducing lipid peroxidation, scavenging free radicals, detoxicating toxic metals and regenerating damaged DNA (4). It ap-

pears that selenium also exhibits immunomodulatory properties by promoting T_H1 lymphocyte cytokine secretion and possibly inhibiting the secretion of T_H2 cytokines (65). Additionally, selenium expresses anti-inflammatory and antiallergic properties by inhibiting the activation of the nuclear factor κ B (66), and modulating arachidonic acid metabolism which includes reducing the synthesis of leukotrien₄ (2). A recent study suggested that serum Se levels and the prevalence of atopy had an inverse association (67). Additionally, other epidemiological studies reported lower serum levels of Se and glutathion peroxidase activity in asthmatic subjects compared to non-asthmatic subjects (4, 63, 68) and also reported that a higher dietary selenium intake was related to a lower prevalence of asthma (69). Even some clinical trials indicated beneficial properties of Se in asthmatic patients reporting that supplementation of Se resulted in the improvement of asthma symptoms (70) and in the reduction of corticosteroid therapeutic dose (71).

Non-enzymatic antioxidants

Vitamin C. Vitamin C, a water soluble vitamin, is an abundant antioxidant in the respiratory tract lining fluid where it majorly contributes to the endogenous and exogenous elimination of ROS (72). Vitamin C has a prominent role in the regeneration of membrane-bound oxidized vitamin E (73). It has been reported that vitamin C may be useful for prevention of lung oxidant injury not only as an oxidant scavenger but also as an inhibitor of polymorphonuclear leukocyte influx to the pulmonary tissue (74). Additionally, vitamin C appears to prevent the secretion of histamine by leukocytes and increase its degradation (75). The antagonistic effects of vitamin C on prostaglandin F_{2a}-induced bronchoconstriction have been reported in an experimental study (17). There is ample published literature on vitamin C and allergic respiratory diseases. Most population studies (cross-sectional and case-control) that evaluated the relation between different values of vitamin C intake or serum ascorbic acid levels with parameters of respiratory function or reported respiratory symptoms showed a beneficial effect of vitamin C on the prevalence and severity of symptoms in asthma (76, 77) and indicated that plasma and leukocyte vitamin C concentrations are significantly lower in individuals with asthma than in healthy subjects (78). However, the only prospective study found no effect of vitamin C on the incidence of asthma (40). Clinical trials have shown inconsistent results regarding the effect of vitamin C on the severity of respiratory allergic diseases. These studies usually involved a small group of subjects and short term administration of vitamin C. There are studies reporting that ascorbic acid supplementation in asthmatic patients reduces airway hyperactivity (79) or decreases the severity of asthmatic attacks (80) and in combination with vitamin E protects against ozone-induced bronchoconstriction (81, 82). Effective vitamin C therapy has been reported in patients with perennial allergic rhinitis (83, 84). However, the evidence

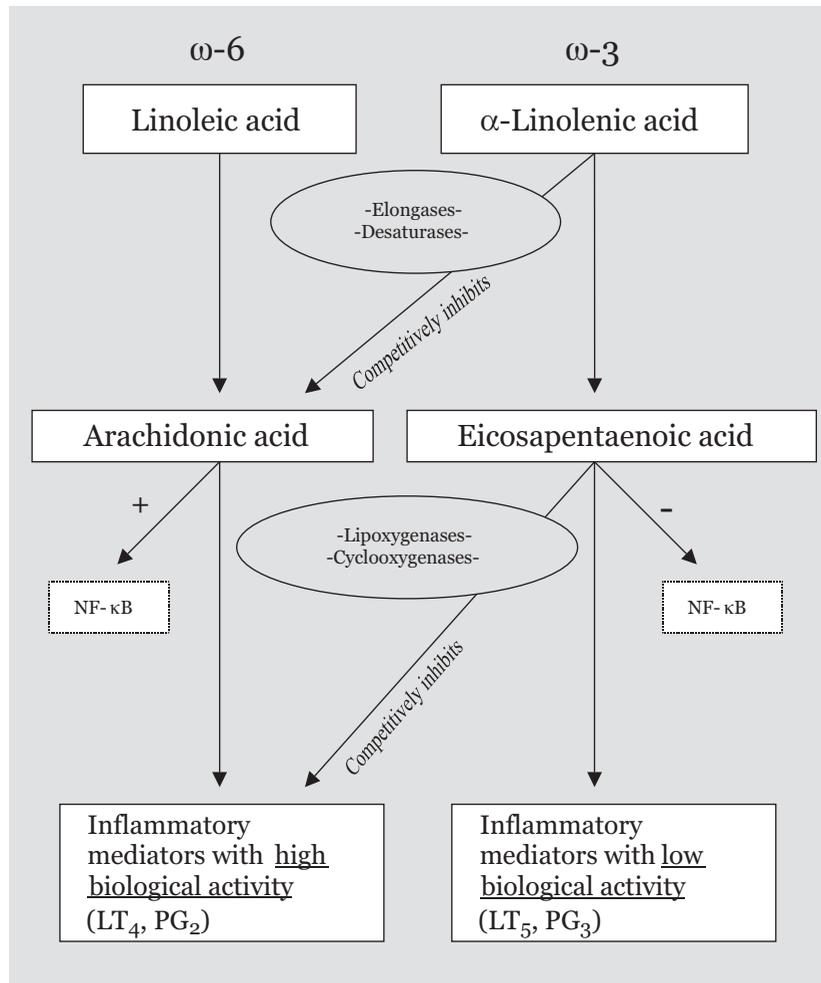


Figure 1. Metabolism of ω -6 and ω -3 PUFA-s and biosynthesis of inflammatory mediators. Legend: »+« indicates activation; »−« indicates inhibition.

of positive effects of vitamin C in asthmatic patients has not been verified in majority of studies (57, 80, 85).

Vitamin E. Vitamin E is a lipid soluble vitamin and a potent dietary antioxidant in the pathophysiology of allergic respiratory diseases (2, 4). Antioxidative mechanism of vitamin E is achieved by protecting lipid cell membranes against oxidative stress. In addition, vitamin E appears to have immunomodulatory effects promoting T_H1 differentiation (86, 87, 88) and antiallergic properties since there is evidence that a high intake of vitamin E is inversely associated with allergic sensitization and IgE serum levels (88, 89). Epidemiological studies reported that vitamin E intake is associated with a reduced asthma incidence and prevalence (40, 77) and in the combination with vitamin C appears to protect against ozone induced asthma (81, 82). However, supplementation of vitamin E in asthmatic patients did not reveal any clinical benefit (90).

Vitamin A & carotenoids. Vitamin A and carotenoids are lipid soluble antioxidants which diminish lipid peroxidation and scavenge superoxide radicals (4). In epidemiological studies dietary vitamin A and carotenoid

intake and serum retinol levels were positively associated with ventilatory function of asthma patients (91) and inversely correlated with the prevalence of adult asthma (92) and the severity of asthma symptoms (93, 94). Additionally, high plasma carotenoid concentration is suggested to have a protective effect on allergic rhinitis in adulthood (95). However, clinical trials investigating the effect of vitamin A on allergic respiratory diseases have been scarce and reported limited beneficial results (96).

Flavonoids & isoflavones. Flavonoids are hydrosoluble scavengers of ROS and lipid peroxy radicals (4). Their antioxidative role is also reflected in inhibiting prooxidative enzymes and acting as chelators of toxic metals (4, 97). In addition to antioxidative effects, flavonoids are supposed to be immunomodulatory and antiallergic agents. Immunomodulatory properties are exhibited by the promotion of T_H1 lymphocyte differentiation (98, 99) whereas antiinflammatory effects are achieved by the inhibition of nuclear factor κ B activity (100). There is evidence that flavonoids have protective effects on the incidence and prevalence of asthma (97, 101). Epidemiological studies have showed that high intake of apples and pears lowered the risk of asthma, while red wine consumption

was negatively associated with asthma severity. The protective effects in apples, pears and red wine were prescribed to flavonoids which are abundant in these products (69, 101, 102). However, it should be regarded that the protective effect of fresh fruits reported in these epidemiological studies could actually be provided from the combining effect of various antioxidative agents that are abundant in these nutritional products (69).

Recent research on isoflavones, known for their estrogenic characteristics, reported antioxidative and anti-inflammatory properties (103) which could explain their ability to reduce the prevalence and severity of respiratory allergic symptoms found in epidemiological studies (104, 105).

CONCLUSION

Currently, there is a significant amount of epidemiological evidence supporting the idea of a preventive effect of ω -3 PUFA-s, magnesium and antioxidants against the development of allergic respiratory diseases in adults. However, controversial results of ω -3 or antioxidant therapy and disappointing results of magnesium supplementation in the conducted clinical trials show a possible complex association of these dietary nutrients and allergic respiratory disorders. There is lacking evidence to recommend ω -3 PUFA-s as adjuvant therapy except in identified genetically predisposed individuals who could possibly benefit from such supplementation. On the other hand, disappointing results of antioxidant therapy in clinical trials could be interpreted by the presumption that the protective effect of antioxidants emerges from their combination in nutrition, and therefore supplementation of one specific antioxidant would be pointless. Based on the reported scientific data in this review we can conclude that, due to controversial evidence, the concept of preventing and treating respiratory allergies with a modified diet needs further clarification and investigation.

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