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Original Article

Food-induced immunoglobulin E-mediated allergic rhinitis

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ABSTRACT

Food allergies are estimated about 1–2% in adults and 8% in children younger than 6 years. Allergic rhinitis is a common disease with a prevalence of 40% among different societies. Although, some foods play a role on exacerbation of allergic rhinitis symptoms, but still there are controversies about the role of diet on incidence or worsening the symptoms. The ongoing research demonstrates a possible relationship between certain foods and their induction of allergic reactions by modulating immunoglobulin E. A total of 100 patients (including both children and adults), between the ages 10 to 60 years, diagnosed with allergic rhinitis were selected for the study. Pregnant females and treated patients with antihistamine were excluded from the study. *In vitro* serum immunoglobulin E (IgE) levels mediated by a combination of food and inhalant allergens were detected by RIDA® Allergy Screen in blood samples. Data were presented as mean, standard deviation and standard error. A statistical analysis was performed by one-way analysis of variance (ANOVA). A p value < 0.05 was considered statistically significant. We reported that 63% of patients with allergic rhinitis were sensitized to common food allergens whereas the rest 37% of patients were not sensitive to any of the food allergens. Similarly, a correlation between the age groups of patients with allergic rhinitis and food allergy were also accomplished. We found the highest response rate for allergic rhinitis and food allergy (53.2%) for the people between aged between 21 years and 40 years. We also demonstrated that females are more prone to mediate allergic rhinitis as induced by food allergies as compare to males (i.e., 66.2% vs. 33.3%). Food allergy is estimated to be 4.5% in adolescents and adults with asthma, rhinitis or both. Rice, citrus fruits, black grams and banana are identified as major allergens for inducing allergic-rhinitis symptoms.

1. Introduction

It is a universally accepted fact that nutrition plays a significant part in diseases, especially in food-induced allergies. Food allergies affect 1–2% of adults and 8% of children younger than 6 years [1–3]. The significance of food allergies increases during early childhood, and it decreases with the age. The following are the significant food allergies in young children: cow's milk (2.5%), eggs (1.3%), peanuts (0.8%), wheat (0.4%), soy (0.4%), nuts (0.2%), fish (0.1%), and shellfish (0.1%). Almost one-half of the children with immunoglobulin E (IgE)-mediated allergy to cow's milk experience sensitivity to other food products [4,5]. Generally, milk-, soy-, egg-, and wheat-related allergies seem to resolve with the adulthood. However, the following allergies seem to persist [1] in adulthood: shellfish (2%), peanuts (0.6%), nuts (0.5%), and fish (0.4%) [4]. Across Estonia, Lithuania, and Russia, the following foods are the most common food allergens: citrus fruits,

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chocolate, apples, hazelnuts, strawberries, fish, tomatoes, eggs, and milk. Allergies associated to nuts, apples, pears, kiwifruit, stone fruits, and carrots are most common in Denmark and Sweden [6]. In the United States, children are most commonly allergic to eggs, cow's milk, peanuts, nuts, soy, wheat, and fish; adults are allergic to shellfish, peanuts, nuts, and fish [7].

In general, adverse food reactions can be divided into two broad categories (i.e., food allergy and food intolerance). A food allergy is due to an abnormal immunologic response following exposure to a food, which may be categorized into either IgE-mediated or non-IgE-mediated process. The IgE-mediated reactions comprise of acute urticaria, angioedema, oral-allergy syndrome, allergic rhinitis (AR), conjunctivitis, and acute asthma. Non-IgE-mediated food allergies present as more subacute and/or chronic symptoms that are typically isolated to the gastrointestinal tract and/or skin. The exclusive non-IgE-mediated food reactions principally include celiac disease, dermatitis herpetiformis, and Heiner syndrome. Both IgE- and non-IgE-mediated mechanisms can be involved into atopic dermatitis and eosinophilic gastrointestinal disorders [8,3]. The second type of adverse food reaction, which is food intolerance, is a nonimmunological reaction to food and could take effect by metabolic deficiencies (e.g., lactase or fructase deficiency), pharmacological intolerance (e.g., caffeine or tyramine in aged cheese), toxicity (e.g., bacteria or contaminants like histamine in scombroid poisoning), or psychological disorders [1,2,8,3,9].

The immune-mediated adverse reactions to food products are caused by IgE, non-IgE-dependent, or mixed mechanism. The immune system's response to allergen exposure can be divided into two phases. The first is immediate hypersensitivity or the early-phase reaction, which occurs within minutes of exposure to the allergen. The second, or late-phase reaction, occurs 4–6 hours after the disappearance of the first-phase symptoms and can last for days or even weeks. Cross-linking of a sufficient number of mast cell/basophil-bound IgE antibodies by allergen initiates a process of intracellular signaling, which leads to degranulation of cells with the release of mediators of inflammation [1]. During the early-phase reaction, chemical mediators released by mast cells, including histamine, mast cell tryptases, prostaglandins, LTC4, LTD4, and LTE4 leukotrienes and thromboxane, produce local tissue responses as the characteristics of an allergic reaction [2,4]. During the late-phase reaction in the lung, cellular infiltration, fibrin deposition, and tissue destruction resulting from the sustained allergic response lead to increased bronchial reactivity, edema, and further inflammatory cell recruitment [2,3]. A general correlation has been found between the rate of cellular infiltration and disease severity.

In spite of the fact that some foods have a role to play on the exacerbation of AR symptoms, there are debates prevailing on the role of diet on the occurrence or worsening of the symptoms. The present study aimed to investigate and identify the relationship of food allergens and AR. Furthermore, this study was undertaken to establish a relationship between sex, age, type of work, and family history of AR and food allergy in tested individuals.

2. Materials and methods

The study groups were selected including all the pediatric as well as adult patients referred for allergic disorders, with suspicion of allergy, to Alborg Laboratory in Jeddah City, Saudi Arabia between October 2013 and July 2014. A total of 100 patients, between the ages 10 years and 60 years, diagnosed with AR, were selected for the study. Patients on antihistamine medication or on long-term steroids, as well as pregnant women, were excluded. A combination of food and inhalant allergens was used for serum IgE *in vitro* tests. The common inhalant panel included the following: *Dermatophagoides pteronyssinus*, *Dermatophagoides farinae*, American cockroach, sheep wool, feather mix, cat epithelia/hair, alfalfa, mesquite, rye grass, desert palm pollens, white goosefoot, Bermuda grass, *Alternaria alternata*, *Penicillium notatum*, *Aspergillus fumigatus*, Timothy grass, mugwort leaf, ambrosia, plantain, and acacia. The common food panel comprises the following: milk, banana, mango, egg white, egg yolk, cereal mix, peanut, gluten, cashew nut, seafood mix, honey, tomato, soya bean, dates, strawberry, and cacao.

Blood was drawn to be tested with RIDA Allergy Screen (R-Biopharm, Darmstadt, Germany). This screening has a high level of efficiency and is highly economical for the diagnostic confirmation of allergies. It provides test methods for the detection of antibodies via an immunoblot with four different panels (panels numbered 1–4) with 20 allergens on each panel. An automated evaluation was undertaken via digital pictures in RIDA X-Screen or RIDA maXi-Screen. The serum IgE results were categorized into zero to six classes according to the severity of allergic reaction to each aeroallergen. Each trial was conducted in triplicate. Data were collected as mean, standard deviation, and standard error, and analyzed by one-way analysis of variance (ANOVA), where $p < 0.05$ was considered statistically significant.

3. Results

A total of 100 cases (i.e., 48 males and 52 females ages between 10 years and 60 years), diagnosed with a history of AR and clinical presentation, underwent specific IgE test with inhalants and food allergens. In this study, 63% of the patients with AR were sensitized to common food allergens, whereas the rest (37%) of patients were not sensitized to any of the food allergens tested. The following allergies associated with inhalant allergens were noted with the higher percentages in the descending order: 52% of the patients were allergic to *D. pteronyssinus*, 47% to *D. farinae*, 36% to American cockroach and *A. fumigatus*, 33% to *P. notatum*, and 32% to cat epithelia/hair. The rates of sensitivity to the other allergens were as follows: 27% to desert palm pollens, 19% to mesquite, 21% to Timothy grass, 17% to Bermuda grass, 15% to rye grass, and 16% to ambrosia. Some patients were sensitive to mugwort leaf (12%), acacia (12%), *A. alternata* (14%), white goosefoot (11%), alfalfa (9%), plantain (7%), and sheep wool (3%; Table 1, Figure 1).

The foodstuffs that were found to mediate hypersensitivity symptoms were cereal mix, peanut, egg, cashew nut, orange, milk, seafood mix, dates, soya bean, tomato,

Table 1
Percentage of inhalant allergens in patients with allergic rhinitis.

Inhalant allergens	%
Dermatophagoides pteronyssinus	52
Dermatophagoides farinae	47
American cockroach	36
Penicillium notatum	33
Aspergillus fumigatus	36
Cat epithelia/hair	32
Desert palm pollens	27
Mesquite	19
Timothy grass	21
Bermuda grass	17
Rye grass	15
Ambrosia	16
Mugwort leaf	12
Acacia	12
Alternaria alternata	14
White goosefoot	11
Alfalfa	9
Plantain	7
Sheep wool	3
Feather mix	1

Table 2
Percentage of food allergens in patients with allergic rhinitis.

Food allergens	%
Egg white	52
Egg yolk	47
Milk	32
Peanut	55
Cashew nut	45
Cereal mix	60
See food mix	23
Banana	10
Mango	12
Strawberry	15
Soya bean	19
Dates	23
Honey	10
Gluten	14
Tomato	17
Orange	37

strawberry, gluten, mango, banana, and honey (Table 2, Figure 2). We observed that 60% of the patients were sensitive to cereal mix, 55% to peanut, 52% to egg white, 47% to egg yolk, 45% to cashew nut, 37% to orange, and 32% to milk. The sensitivity rate for seafood mix and dates was noted to be at 23%. Ten percent of the patients were allergic to banana and honey, while 17% were allergic to tomato, 14% to gluten, 12% to mango, 19% to soya bean, and 15% to strawberry.

Table 3 shows the percentages of age groups for patients with AR and food allergy. According to the three age groups, which included the first-category individuals aged 10–20 years, second category aged 21–40 years, and third category aged 41–60 years, the individuals in the second category were detected with the highest response rates for AR and food allergy [i.e., 53.2% ($p < 0.0001$, Figures 3 and 4)]. This high percentage is due to the fact that this age category represents the largest segment of the society. Similarly, the high percentage in the first age group following the second category represents the school and university students who are often exposed to the allergens (Figures 3 and 4).

The relationship between AR and food allergy and age, gender, family history, and job profile was also taken into consideration. The results revealed that 66.2% of the females appear to develop AR and food allergy, and 58.7% AR only, 33.3% of the males appear to develop AR and food allergy, and 41% AR only.

The percentage of patients with positive or negative family history of AR and food allergy was 78% and 22%, respectively ($p < 0.0001$, Table 4 , Figure 5). The patients with AR alone and family history positive were 75%, whereas 25% patients had a negative family history of AR ($p < 0.0001$, Table 4, Figure 5). Sixty-five percent of the patients representing as workers and 37% as nonworkers were infected with AR and food allergy (Table 4, Figure 5).

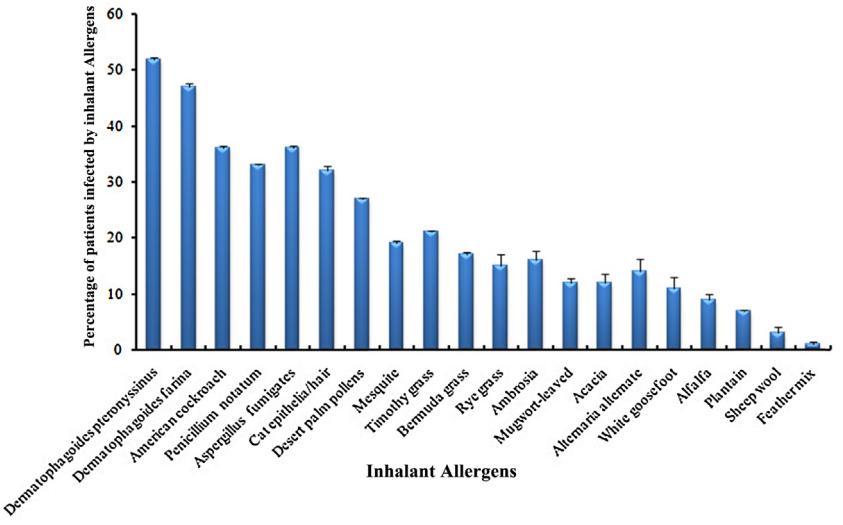

Figure 1. Percentage of infected individuals by inhalant allergens: the *in vitro* serum immunoglobulin levels in blood samples of infected individuals were detected by RIDA Allergy Screen. Each trial was performed in triplicate. The data are presented as mean ± standard error. A p value < 0.05 is considered statistically significant.

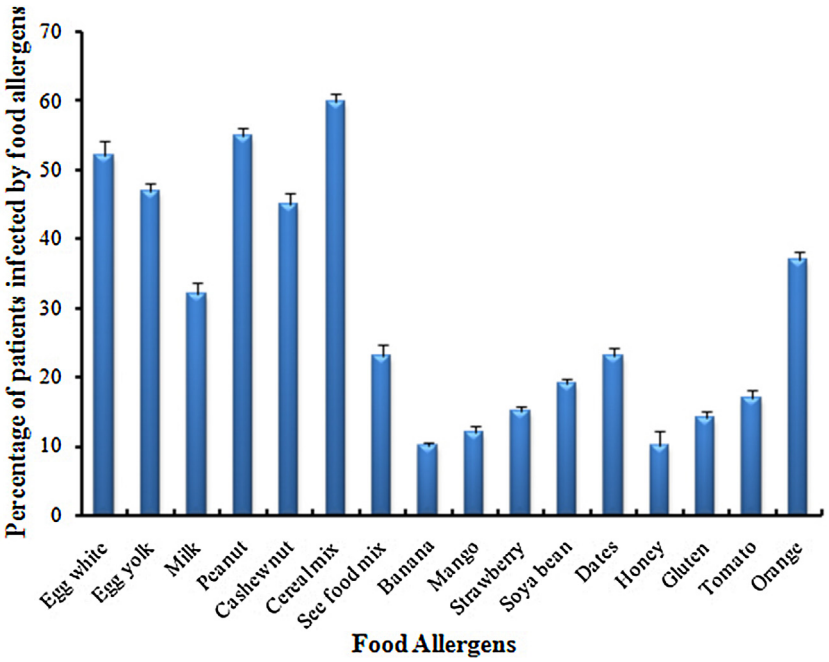

Figure 2. Percentage of infected individuals by food allergens: the *in vitro* serum immunoglobulin levels in blood samples of infected individuals were detected by RIDA Allergy Screen. Each trial was performed in triplicate. The data are presented as mean ± standard error. A p value < 0.05 is considered statistically significant.

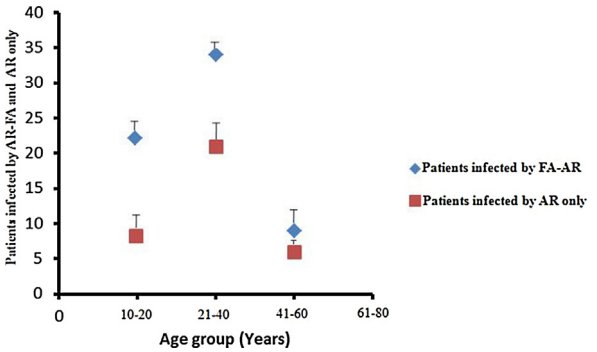

Figure 3. Relationship between the patients' age groups and food-induced allergic rhinitis: each trial was performed in triplicate. The data are presented as mean ± standard error. A p value < 0.0001 is considered statistically significant. AR = allergic rhinitis; FA = food allergy.

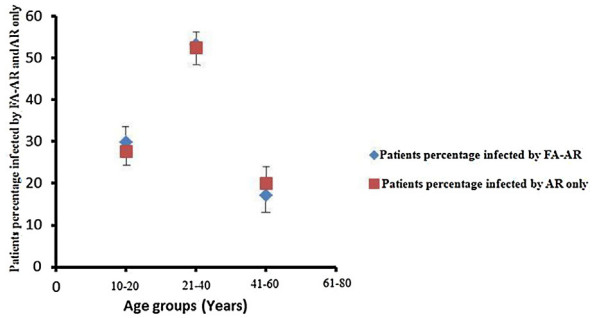

Figure 4. Percentage correlation between the patients' age groups and food-induced allergic rhinitis: each trial was performed in triplicate. The data are presented as mean ± standard error. A p value < 0.0001 is considered statistically significant. AR = allergic rhinitis; FA = food allergy.

Table 3
Percentages of age groups for patients with allergic rhinitis and food allergy.

Age group (y)*	Patients with AR and FA (n = 65)	Patients with only AR (n = 35)
8 (27.6)	22 (29.8)	10–20
21 (52.4)	34 (53.2)	21–40
6 (20)	9 (17)	41–60

Data are presented as n (%).
* p > 0.0001, AR = allergic rhinitis; FA = food allergy

Similarly, 53% workers and 45% nonworkers were found infected with AR only (p < 0.0001, Table 4, Figure 5).

4. Discussion

IgE-mediated reactions produce AR in allergen-sensitive individuals. In AR, IgE-mediated allergic reactions cause the inflammation of the nasal membrane, which is characterized by sneezing, nasal congestion, nasal itching, and rhinorrhea. From 60% to 70% of the infected individuals may accompany the signs and symptoms of eye redness, ocular pruritus, and/or lacrimation [10,11]. The disease itself is not life threatening, but the signs and symptoms adversely affect the individual's quality of life and work, which, in turn, inflicts a significant burden at an individual level and society as a whole [10–13]. Both physical and mental complications may arise due to AR, along with childhood sleep disorders, breathing problems, deteriorating learning skills, and attention deficit and behavioral disturbances in adults [14–18]. The existence of a common correlation between AR and asthma further demonstrates that both conditions are closely related to each other,

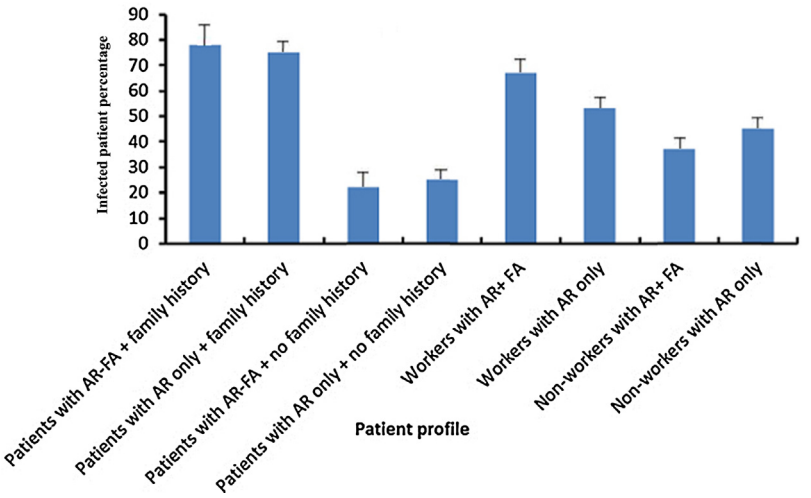

Figure 5. Percentage correlation between the patients' family history of food-induced allergic rhinitis: each trial was performed in triplicate. The data are presented as mean ± standard error. A p value < 0.0001 is considered statistically significant. AR = allergic rhinitis; FA = food allergy.

which subsequently leads to the concept of "one airway, one disease" [19]. Some studies have demonstrated that ~20–50% of AR-infected individuals have clinically proven asthma, whereas > 80% of the patients with allergic asthma express the signs and symptoms of concomitant rhinitis [20–22].

Both acute and chronic types of respiratory-disease symptoms may be induced by food allergies. IgE-mediated symptoms are usually acute reactions, while chronic reactions are modulated by the combined mechanisms of IgE and cellular pathways. It is difficult to predict the inimitable characteristics of food-induced IgE-mediated AR, because the same signs and symptoms may occur in other pathological conditions (e.g., asthma, eczema, oral allergic manifestations, urticaria, and gastrointestinal symptoms) associated with other food allergies. Similarly, a strong correlation in terms of cross-reactions exists between the sensitivity of inhaled pollen proteins and homologous fruit and vegetable proteins [23,24]. One study associated to egg allergies in children delineated that the children who had egg allergy as infants had a significant risk of developing rhinitis and asthma by the time they reached the age of 4 years [25]. In addition, oral food-induced rhinitis posed more challenges during infanthood and early childhood as compared to adulthood [26]. Rhinitis accompanied by rhinorrhea, which occurs due to the post-ingestion of hot, spicy foods and the stimulation of the autonomic nervous system by emotional and psychosomatic factors related to food ingestion could be the cause for nonimmunological rhinitis [24]. Food-induced allergic reactions pose a rare manifestation of asthma, although the food-induced symptoms may often include acute bronchospasm [26]. The patients who are sensitive to post-oral intake of a small amount of a food allergen may culminate into a life-threatening asthma due to the hyperreactivity of the airways. Anaphylaxis and asthmatic reactions may be triggered due to the proteins contained in steam or vapors emitted from cooking food, such as fish [27]. In patients who had a history of refractory asthma, atopic dermatitis, gastroesophageal reflux, and food allergies, asthmatic symptoms induced by foods should be taken into account in such patients [4]. Heiner syndrome is a rare disorder typically induced by cow's milk, the characteristics of which represent recurrent events of pneumonia associated with pulmonary hemosiderosis, gastrointestinal bleeding, iron-deficiency anemia, and failure to thrive in infants [4].

As a matter of fact, AR is a rare manifestation of food allergy [28]. Some patients develop itching and burning sensation in their throat associated with AR symptoms following the ingestion of some fruits and vegetables. These symptoms are collectively referred to as *oral-allergy syndrome* or *pollen-food allergy syndrome*. The prevalence of this syndrome is estimated to be in a range between 5% and 17% in different societies [29,30]. A significant clinical finding regarding this syndrome is the fact that nonvegetated foods play no role in the manifestation of the syndrome; nevertheless, they are still considered as common and important allergens. Therefore, the oral intake of foods, such as cow's milk, eggs, and seafood, has no effect on the pollen-food allergy syndrome. By contrast, some vegetables and fruits could trigger an IgE-mediated food allergy due to its similarities in structure or homology with the pollens, especially during the pollination season. The clinical features of oral allergy are manifested

Table 4
Percentage of patients with family history, no family history, workers, and nonworkers.

	Family history (%)	No family history (%)	Workers (%) *	Nonworkers (%) *
Patients with AR & FA	78	22	67	37
Patients with only AR	75	25	53	45

* p > 0.0001, AR = allergic rhinitis; FA = food allergy.

by burning sensation, pruritus, erythema, and edema of the oropharyngeal mucosa, while in some patients, frequent sneezing, itchy eyes, and runny nose are prevalent [31]. Sycamore pollen and its structurally similar varieties; hazelnuts; peanuts; fruits, such as apples, kiwi, and peaches; and vegetables, such as corn and lettuce, post-ingestion trigger AR symptoms in sensitive persons [32]. AR symptomatology and oral-allergy syndrome are significantly seen in patients hypersensitive to mugwort pollen due to its structural homology with some vegetables, such as cabbage, caraway, parsley, coriander, anise, and carrots, and some spices, like aniseed, pepper, black pepper, onion, garlic, cauliflower, and broccoli [33]. Hypersensitivity to grass and its classes, and the homology of these with other fruits are not studied in detail, but people sensitive to grass are also sensitive to foods, such as potatoes, melons, oranges, tomatoes, and peanuts [34]. The percentage of type I immunologic reactions to foods and oral-allergy-syndrome formation was delineated as syndromic diseases, such as celery–mugwort–spice syndrome, wherein patients exhibit hypersensitivity to black pepper, parsley, carrot, coriander, and anise [35]. Rubber-production industries have a very common problem of polypeptides in latex causing hypersensitivity. Rubber materials, such as wheels, surgical gloves, and different forms of soft rubber, comprise latex, and allergy to polypeptides has been reported. Health care providers are equally exposed to latex. Latex shares homology with some fruit allergens consisting of avocadoes, bananas, peaches, kiwi, potatoes, tomatoes, and chestnuts. Latex-intolerant individuals who consume such foods develop oral-allergy syndrome and AR [36]. Respiratory symptoms and clinical presentation of AR could sometimes be triggered by airborne allergens, such as wheat causing baker's rhinitis or meat causing symptoms to the butcher, or seafood in restaurant workers [37]. Rhinitis symptoms are often led by food reactions at a nonimmunologic standpoint. Therefore, it cannot be delineated as allergy.

Taking into account individuals suffering from gustatory rhinitis, symptoms appear following the ingestion of spicy foods. These symptoms are of a neurogenic pattern, usually manifested by runny nose a few minutes post-ingestion of the foods, but there are no pertinent complaints of nasal congestion, pruritus, or facial pain. It is stated that such reflexes take place due to adrenergic and cholinergic nerve dysfunction [28,38]. Some foods and fruits due to their natural traits cause irritation of the tongue mucosa and throat locally without producing either immunologic or neurologic reactions. For instance, pineapple could cause such symptoms in the mouth and throat. Even though the role of foods and fruits in causing AR is unclear, it is found that rhinitis is clinically manifested by food allergens in a very small percentage of patients. It is demonstrated that 17% of the patients with allergic sensitivity to pollens (i.e., pertaining to trees, weeds, and grass) could have type I allergic reaction to some vegetarian foods comprising of fruits and vegetables. Foods that either act as stimulants or as mediated by neurologic mechanisms can trigger symptoms of rhinitis, which is not counted as an allergic reaction.

5. Conclusions

The concrete evidences suggest that the prevalence of food allergies is unknown, as most of the patient histories are not clinically studied for confirmation. The cases of AR triggered by food are uncommon, as such events may take place along with other symptoms, such as eczema, urticaria, asthma, oral allergic manifestations, and gastrointestinal symptoms. The treatment of food-induced AR is usually topical steroids and antihistamines, but further investigation is not warranted in cases of rhinitis or allergic symptomatology without any specific allergy to food. Nevertheless, the identification of patients who are at risk for life-threatening respiratory-system reaction and those with food allergy mediated by IgE along with anaphylactic risk should be carried out, in order to perform tests and suggest diets accordingly.

Conflicts of interest

The author has no conflict of interest to declare.

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