

REVIEW

What do asthmatics have to fear from food and additive allergy?

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Summary

International studies report marked increases in the prevalence of food allergy, along with increases in hospital admissions and emergency presentations for severe allergic reactions due to foods. The prevalence of self-reported food allergy is common, but generally exceeds that which can be verified from challenge studies, although nut allergies appear to be an important exception to this rule. Studies examining food allergy deaths suggest that those who die of food allergy usually have co-existent asthma. Adolescents and young adults are at most risk, and adrenaline auto-injectors are sub-optimally used. Food chemical sensitivity is very commonly reported but not usually verified by challenge testing. However, the exception to this is sulphite sensitivity, which can elicit reproducible reactions in some. The increasing prevalence of severe food allergies and awareness of its risk in those with asthma demands an especially rigorous approach to the diagnosis and management of co-existent food allergy and asthma, especially in young people who appear to be at most risk from death from severe food allergy.

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Introduction

Hypersensitivity reactions to food are perceived to be very common, with over 20% of children and adults reporting food allergy in some studies [1, 2]. People with asthma are five times more likely to report adverse food reactions than people without asthma [3]. However, international asthma guidelines state that food is not considered an important trigger of asthma attacks except in those with documented IgE-mediated food sensitivity [4], where a link between anaphylaxis mortality and asthma is well established [5–8]. Hence, is community perception different to reality? Given that the reported prevalence of symptomatic food allergy greatly exceeds confirmed IgE-specific food allergy, is food chemical sensitivity important in asthma? This article will attempt to define the links between food allergy and food additive hypersensitivity and asthma. In doing so, we would seek to provide evidence for those caring for individuals with concurrent food allergy and asthma to enable the construction of a risk assessment and appropriate management strategy to keep those with food allergies and asthma alive and well.

Definition

International consensus definitions of allergy designate 'hypersensitivity' as a 'reproducible illness on exposure to

an environmental substance at a dose tolerated by normal persons'. Hypersensitivity reactions can be further classified as mediated by either immunological or non-immunological mechanisms [9]. Immunologically mediated reactions are designated 'food allergy' and those food reactions due to IgE are defined clinically as 'IgE-mediated food allergy'. Some well-described food hypersensitivity syndromes such as coeliac disease would therefore be classified as a 'food allergy' but not mediated by IgE. Similarly, some of the food hypersensitivities generally diagnosed in children such as food-protein-induced enterocolitis syndromes and allergic eosinophilic oesophagitis are classified as food allergy, but the immunological mediation may be by both IgE- and T-cell-dependent mechanisms [10]. Food hypersensitivity mediated through non-immunological mechanism is referred to as non-allergic food hypersensitivity and would encompass food additive hypersensitivity [9]. The links between food allergies and asthma are most strongly drawn in the setting of IgE-dependent food allergy (Fig. 1).

Prevalence – food allergy

The prevalence of self-report in food allergy in adults is usually overestimated and highlights the need for wider allergy education in the community. Rona et al. [1] performed a meta-analysis including 51 studies of food

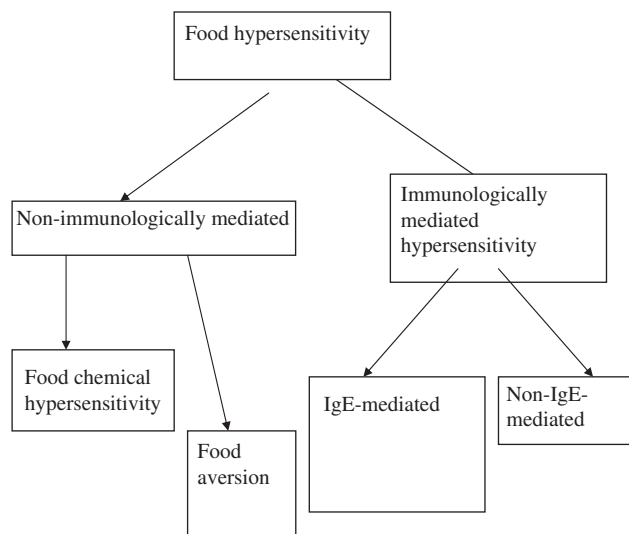


Fig. 1. A classification of food sensitivity. Adapted from Johansson et al. [9]

allergy prevalence. There was marked heterogeneity in the studies due to the country of origin and the age of participants but the mean prevalence of self-reported food allergy was 12% in both adults and children. The reported prevalence of allergy to most foods was consistently higher than objective markers so that the prevalence of self-reported milk allergy was 17% compared with an estimated prevalence of 0–3% in food challenge studies. However, the discrepancy between reported and immunologically diagnosed food allergy was not documented for all allergens and the self-reported prevalence of peanut allergy was <2%, correlating far more closely with the prevalence documented on skin prick test (SPT) or challenge testing, which revealed a mean prevalence of peanut allergy of approximately 1%. Similarly, self-report of tree nut allergy has been examined in a systematic review and shown a prevalence of self-reported sensitivity of up to 4.1% and objective challenge test confirmation of 4.3% [11].

There has been a rising prevalence of food allergy in the population over the last 10 years. Mullins [12] identified an increasing prevalence of food allergy in his community-based allergy practice in children aged 0–5 years between 1995 and 2006. Further, the number of children with food-associated anaphylaxis increased sevenfold during this decade. Poulos et al. [13] reviewed Australian hospital data on admissions and deaths for anaphylaxis, angioedema and urticaria between 1993 and 2005. In the 0–4 age group, the anaphylaxis hospitalizations increased from 4.1 to 19.7 per 100 000 over the study period. This rise in presentations was attributed to food-related anaphylaxis.

Specifically, there is a marked increase in the prevalence of peanut allergy over the past 20 years with cohort studies demonstrating peanut allergy prevalence rates of 1–2%, double that of previous cohorts [14]. Because allergy to peanuts is one of the major causes of anaphy-

laxis deaths, increases in the prevalence of nut allergy is a major cause for concern.

Cause – food allergy and sensitization

The co-existence of food allergy and asthma in atopic patients is seen as a manifestation of the 'atopic march' for which the pathogenesis is still not completely understood. A sequence of allergic diseases beginning with atopic dermatitis in infancy is followed closely by food sensitization and in some, food allergy. Later, aeroallergen sensitization often develops with the onset of rhinitis followed by asthma. This pattern of atopic sensitization was highlighted in a 22 year follow-up of an at-risk (of atopy) birth cohort [15]. Thirty-three percent of the cohort developed a food sensitization with 12/13 occurring by year 2. Food sensitizations waned over time such that only one patient remained sensitized at age 22. Aeroallergen sensitization occurred throughout childhood and tended to be permanent. Food sensitization predicted aeroallergen sensitization.

International cohorts reveal that eczema in the first 6 months of life is associated with an increased risk of egg, milk and peanut allergies in childhood [16]. Food sensitization and allergy in infancy are predictive of adult asthma. Rhodes et al. [17] performed skin testing on 100 infants with high-risk atopic parents under 1 year of age and prospectively followed them until the age of 22. A positive SPT to hen's egg, cow's milk or both was independently predictive of adult asthma [odds ratio (OR) 10.7, CI 2.1–55.1].

The role of diet as a risk factor for the development of both food allergy and asthma has been explored. Omega-3-fatty acid supplementation has been shown to improve asthma control in some studies; however, there is no evidence that supplementation can prevent the development of food allergies [18]. A Mediterranean diet may protect from asthma, but is not associated with protection from food allergy [19]. Prospective studies of vitamin D and allergy and asthma onset are awaited but no clear links have been shown in the onset of asthma and food allergy [18].

A further potential link between food allergy and asthma is oesophageal reflux and the common use of antacid medication to increase gastric pH. Elevated gastric pH is likely to cause delayed breakdown of food allergens and retained allergenicity, and has been associated with increased levels of IgE to common foods. Antacid therapy may also decrease the threshold of food consumption, which can elicit an allergic reaction by preventing the breakdown of foods and the consequent retention of allergenicity [20]. Conversely, gastro-oesophageal reflux is considered an important risk factor for the exacerbation of asthma symptoms, being found in approximately 40% of those with severe asthma, although studies of the use of antacid treatments for severe asthma do not suggest that this is a useful therapeutic option [21].

Reasons for the observed increases in food allergy prevalence are not established but environmental causes are likely to be implicated. Increased exposure to allergenic foods is one possibility, for example peanut and fish allergens, which are relatively resistant to digestion and proteolysis [22]. Alternately cutaneous exposure to food allergens such as peanut oil is currently being explored as a potential factor [23]. The increase of prevalence in food allergies has been predominantly reported in developed nations suggesting that changes in gut microflora may influence sensitization and the development of food allergies and trials of probiotics and pre-biotics are currently underway to elucidate this. The role of other environmental influences such as gastric acid suppression and vitamin D deficiency in altering allergen processing and immune responses has also been postulated [24]. Recently, the co-existence of obesity and food allergy has been described suggesting a link between these two concurrent epidemics [25]. However, despite the discovery of these influences, the factors that determine the acquisition of food allergy, and as importantly, the development of tolerance to foods are not well established and are an important task of current research in this area.

Morbidity

Food allergy is a risk factor for asthma morbidity including hospitalization. Roberts et al. [26] undertook a case-controlled study of 19 children (1–16 years) ventilated for an exacerbation of asthma. All subjects undertook a questionnaire, spirometry, skin testing and RAST testing. Food allergy was independently associated with life-threatening asthma with an OR of 5.89. Similarly, Miles et al. [27] used a case-control study design of 29 adults with poorly controlled asthma and a well-matched group with stable asthma. Atopic status was assessed to 19 common aero and food allergens with specific IgE measurements. Atopic sensitization to food (and aeroallergens) was a risk factor for brittle or sub-optimally controlled asthma.

Berns S et al. [28] identified 203 asthmatic patients in an outpatient clinic and examined the association between food allergy and asthma morbidity. Twenty-two percent of these patients were identified as having a history consistent with IgE-mediated food allergy. Patients with allergies to more than one food (7/22) had increased asthma hospitalizations, ED visits and use of oral steroids. Jarmillo et al. [29] investigated the association of food sensitization to egg, milk, peanut and shrimp, and asthma in 8203 US participants. Food sensitization was significantly more prevalent in those with asthma (27.5% vs. 14.9%). For those with a high-level sensitization determined by the specific IgE level corresponding to a 95% likelihood of clinical food allergy, the OR for a recent emergency room visit was 7.0 and that for current asthma was 4.4.

The underlying severity of a patient's asthma is important in predicting the severity of allergic food reactions. Summers et al. [30] analysed 1094 patients assessed and treated in an allergy centre between 1992 and 2004 with peanut and tree nut allergy. Life-threatening bronchospasm was more likely to occur in those patients with severe asthma (relative risk of 6.8) compared with those patients with mild asthma (relative risk of 2.7).

Mortality

Asthma has been identified as a frequent co-morbidity in fatal food allergy and studies of anaphylaxis death can provide lessons for the management of individuals with severe food allergy. Bock and Munoz-Furlong [5] analysed 32 anaphylactic fatalities reported to a national registry in the United States and subsequently an additional 31 fatalities between 2001 and 2006 [6]. In the first study, 96% (24/25) of fatalities had a past history of asthma. Peanuts and tree nuts were identified as the likely food trigger in 30/32 (94%) instances. Most fatalities had a known past history of food allergy.

Pumphrey and Gowland [7], in a UK study of 48 anaphylaxis deaths between 1999 and 2006, supports these findings showing that 43/48 took daily treatment for asthma and that 10 of the asthmatics had a current exacerbation in the lead up to their catastrophic reaction to food. Another important observation made by Pumphrey was that over 50% of the deaths occurred in patients whose previous reaction to foods was considered mild and that anaphylaxis deaths were more likely to occur with 'social eating': that is eating away from home.

Food-induced anaphylaxis is also more likely to manifest as acute or life-threatening asthma. Pumphrey [8] analysed 214 deaths associated with anaphylaxis in the United Kingdom between 1992 and 2001. For food anaphylaxis, the peak age for fatal anaphylaxis was 17–27 and was observed mainly in atopic females. Anaphylaxis usually occurred 25–35 min after the offending food.

Regrettably, the failure to administer adrenaline in a timely manner is also a feature of anaphylaxis deaths [6, 8]. Recent studies of survivors of anaphylaxis confirm that people with anaphylaxis are reluctant to use adrenaline despite reporting of systemic symptoms of anaphylaxis, highlighting a need for clear action plans for dealing with anaphylaxis [31]. In this study, however, those with asthma were more likely to use adrenaline than those without. Just as importantly, one quarter of study participants did not have an adrenaline auto-injector to use despite describing systemic symptoms and a past history of food allergies.

Taken together, these studies indicate important factors in the co-occurrence of food allergy and anaphylaxis deaths [5–8, 32]. The ages of those who died of food-induced anaphylaxis are consistently in the adolescent

and young adult age group. Moreover, the location of fatal-food-related anaphylaxis is not usually in the person's own home, suggesting that 'social eating' [7] is a particular time of risk.

Physiological explanations have also been found for the reasons why people with asthma and co-existent food allergies are at a greater risk for death. Cardiac manifestations of anaphylaxis are a major cause of anaphylaxis death [7]. Mast cells exist in the myocardium and are likely responsible for low cardiac output states, which can complicate a severe allergic episode [33]. Moreover in allergic individuals, mast cells are likely to be activated by the presence of elevated levels of allergen-specific IgE, rendering the mast cells more sensitive to a systemic allergic event. In the lung, a comparison of the bronchial histology of those with fatal asthma with those with mild asthma reveals greater numbers of the anaphylatoxin receptors C3aR and C5aR in the lungs of those with fatal asthma [34]. This would render those individuals who have severe or near-fatal asthma more sensitive to the effects of systemic anaphylaxis than their peers with milder asthma, providing a reason for a greater risk of anaphylaxis-related death in those with severe or unstable asthma.

Age and risk-taking behaviour

That fatal anaphylaxis to food allergens is more likely to occur in young adults and adolescents might be attributed, in part, to risk-taking behaviour [35]. An internet-based questionnaire including 174 patients with a history of anaphylaxis highlighted this problem [35]. Only 61% of participants 'always' carried their auto-injectable adrenalin device. Forty-two percent of participants reported 'willingness' to eat a food labelled 'may contain' an allergen. Pumphrey [8] described the case fatality of a 16-year-old girl with a known nut allergy that ate a chocolate cake in the belief her adrenalin auto-injector would be protective.

Other factors important in the management of food allergy and asthma in young people is likely to be the developmental context of young people as they move through the process of adolescence [36]. Central to the successful negotiation of adolescence is the acquisition of independence from the family environment and the development of peer relationships, each of which will necessitate embarking on an independent social life. This is likely to include social eating at places away from the relative safety of family influences.

Risk-taking behaviour in adolescents and young adults requires a careful psychological assessment. There is a relationship between risk-taking behaviour and depression. Non-adherence to asthma treatment and asthma morbidity are also closely correlated to depression. Risk-taking behaviours in the patient with food allergy and asthma range from non-adherence to asthma treatment, cigarette smok-

ing, failure to carry an adrenalin auto-injector to eating foods containing known allergens [37].

Therefore for adolescents with severe food allergies to be safe, they must be competent to manage their food avoidance plans, be able to communicate their plans to others and be competent to administer their emergency treatment. Individuals with asthma and food allergies are at particular risk and clinicians need to direct special vigilance to this group and recognize both the higher risks and the unique challenges inherent in the normal development of young people with food allergies and asthma.

Quality of life impact with food allergy

Food allergy has a dramatic impact on the quality of life (QOL) of patients and their parents and families. Bollinger et al. [38] surveyed the parents of 87 children in Baltimore with food allergies. Half of the caregivers reported that food allergy affected the social activities of families. Further, 34% reported that food allergies had an impact on school attendance. Of concern, 10% of patients were home schooled because of these food-related anxieties. In a similar study, Primeau et al. [39] compared the psychological burden of peanut allergy in 153 children and 37 adults with sufferers of rheumatological disease. The parents of children with peanut allergy reported more impairment of daily activity and more impairment of aspects of Impact on Family Questionnaire.

Sicherer et al. [40] surveyed 253 parents of children with food allergies. He confirmed the negative impact of food allergies as measured by general health perception, emotional impact on the parent and limitations on family activities. The QOL of families with children was further significantly and adversely affected when the children had more than two food allergies. Finally, the additional co-morbidity of asthma also contributed to the global health perception of these families. Therefore, it would appear that concomitant asthma and food allergy places a particular burden on families in addition to the substantial burden conferred by either condition alone.

There appears to be a variable impact on the individual members of a family with a child/sibling with peanut allergy. Mothers of children with a food allergy appear to be more affected than fathers with a significantly poorer psychological and physical QOL. Children with peanut allergy also have more impairment in QOL than their siblings [41]. Food allergy in an Asian paediatric population also predicted impaired parental QOL. Reduced QOL was associated with the current food avoidance, food allergy to more than three allergens and specific food allergens including peanut, egg and cows milk [42].

Adults with a history of food allergy also report a significant impairment in their QOL. Le et al. [43] surveyed 82 patients with a history suggestive of IgE-mediated allergy to peanuts, tree nuts or fruit. Seventy-two percent

of patients with nut allergy and 62% of those with fruit allergy reported that the food allergy influences their daily life considerably. Flokstra-de Blok et al. [44] have recently developed and validated a food allergy-specific health-related QOL questionnaire (FAQLQ) for children, adolescents and adults. This instrument has been developed to address the complex disease-specific issues associated with food allergy. The most important overall issue for adult patients with food allergy when validating this tool (FAQLQ-AF) was the constant vigilance required as to what they were eating.

Food-dependent exercise-induced anaphylaxis

Food-dependent exercise-induced anaphylaxis is an important and potentially life-threatening allergy. Specific foods ingested before exercise trigger anaphylaxis. The mechanism remains incompletely explained but most patients have an evidence of sensitization to the implicated food allergen with common foods involved including wheat, celery, hazelnut and soy. Palosuo et al. [45] examined 18 patients with wheat-dependent exercise-induced anaphylaxis. All 18 patients had elevated IgE levels to a γ -like gliadin allergen and 13/18 had elevated levels to α -gliadin.

The subjects were followed-up between 2 and 84 months after dietary advice. Importantly, a strict gluten- and wheat-free diet when adhered to was protective against further episodes of anaphylaxis during exercise. Two patients avoided wheat only before exercise and experienced one to three milder urticarial reactions annually.

Oral allergy syndrome

The oral allergy syndrome, often known as the food-pollen syndrome, presents with local oro-pharyngeal symptoms after exposure to certain fruits in patients with seasonal rhinitis and sensitization to one or more aero-allergens, particularly birch pollen [46]. The syndrome represents a cross-reactivity of pollen-specific IgE to particular fruits and food allergens. Birch-sensitive patients commonly react to apple, celery, carrots and hazelnut. The apple allergen Mal d 1 has over 60% homology with the birch allergen, Bet v 1 [47]. There is little consensus among allergists regarding the management of these patients and whether food avoidance should be prescribed or whether allergen immunotherapy is effective [48]. Food-pollen reactions often only occur to the raw fruits and vegetables and are mild, associated only with local symptoms. However, systemic reactions have occasionally been reported, which suggests that this group of patients, while usually not at risk of a severe reaction, should be carefully assessed especially those with asthma so that the risk of a severe reaction can be accurately assessed.

Prevention of food allergy

Considerable controversy exists regarding dietary interventions to prevent food allergy as earlier recommendations for the delayed introduction of allergenic foods have evidently not been associated with a decline in food allergy prevalence. Current recommendations for the prevention of food allergies are therefore based on concepts of inducing tolerance to ingested foods and a lack of evidence to support specific recommendations. There is evidence that breastfeeding for at least 4 months is associated with a reduced prevalence of wheezing and dermatitis in high-risk infants [49]. In infants with a strong family history of allergic disease who are not breastfed, the use of a partially hydrolysed cow's milk formula in the first few months may be associated with a lower risk for the development of allergic disease. However, the evidence regarding solid feeding of potentially allergenic foods remains controversial. Current recommendations suggest the introduction of solid foods at 4–6 months of age. There is no evidence to recommend further delays in introducing potentially allergenic foods. Evidence from prospective carefully conducted trials is awaited to further inform advice on this matter [50].

Food additives

Despite popular opinion strongly linking asthma to exposure to food additives, there is scant evidence to support this. There is a vast number of food additives used commercially and only a few have been definitively linked to asthma. The gold standard test for identifying food chemical intolerance is a blinded placebo-controlled food chemical challenge after a strict elimination diet [51]. Sulphites and monosodium glutamate (MSG) are often identified as potential catalysts for asthmatic reactions.

Woods et al. [52] undertook a double-blind MSG food challenge in 12 subjects with clinically proven asthma and perceived MSG-induced bronchospasm. Participants were challenged on three separate challenge days to 1 g MSG, 5 g MSG and to placebo. No definite immediate or late asthmatic reactions occurred. These results were further confirmed by Woessner et al. [53] who challenged 100 asthmatic subjects to 2.5 g of MSG in a single-blinded placebo-controlled challenge. Thirty of the asthmatic subjects had a history suggestive of MSG sensitivity, the so-called 'Oriental restaurant asthma attacks'. There was no evidence of MSG-induced bronchospasm in any of the asthmatic patients. The authors concluded that we should maintain a 'healthy scepticism' in patients with a history of MSG-exacerbated bronchospasm. Williams and Woessner [54] further reviewed the evidence for MSG as a trigger for asthma. In these published studies using rigorous methodology including blinded placebo-controlled challenges, there was no association identified between MSG and asthma.

There have been more convincing evidences of asthmatic reactions to sulphiting agents. Sulphiting agents are important in the commercial food industry and have a number of important roles. Sulphites added as preservatives delay bacterial contamination of foods. Sulphites are also used in the alcohol industry to prevent undesirable fermentation.

Stevenson and Simon [55] reported four cases of metabisulphite-induced bronchospasm. These asthmatic patients had a past history of asthmatic reactions during ingestion of restaurant food and drinks. Interestingly, they all described asthmatic symptoms triggered by irritant fumes and smog. Skin testing to sulphites was negative. Single-blinded challenges to oral potassium metabisulphite was performed to an increasing amount of sulphites from 1 to 50 mg. Acute bronchospasm was observed in all four patients at doses between 10 and 50 mg.

Simon et al. [56] went on to assess the incidence of ingested metabisulphite sensitivity in a population of asthmatics without a suggestive history. Sixty-one patients were challenged to increasing doses of metabisulphite from 10 to 200 mg and were monitored for bronchospasm with spirometry. Five patients (8.2%) reacted to metabisulphite. These results suggest the sulphite intolerance represents an important risk factor in asthmatic patients.

Vally et al. [57] investigated the role of sulphites in asthmatics with a history of wine-sensitive asthma. Interestingly, in 10 asthmatics that underwent a single-blind high-sulphite wine challenge, only two demonstrated evidence of bronchospasm with a fall of 15% or more in lung function. More recently, Vally et al. [58] assessed whether bronchial hyperresponsiveness (BHR) is a more sensitive marker in self-reported wine-sensitive asthma. None of the eight patients investigated demonstrated a clinically significant reduction change in BHR following challenge with either high- or low-sulphite wine. Three patients had significant increases in BHR with the low-sulphite wine and one of these also reacted with the high-sulphite wine. Vally concluded that sulphites appear to play only a small role in patients reporting wine-sensitive asthma.

Tartrazine has been identified as a possible trigger of asthma. Tartrazine is a coal tar-derived food dye used widely in the food industry. Mathison et al. [59] performed a double-blinded placebo challenge of tartrazine in doses up to 75 mg in over 100 patients. Tartrazine did not cause bronchospasm in any of these patients. Arden [60] confirmed these results in a Cochrane review of six studies. Tartrazine avoidance or challenge had no significant effect on asthma, suggesting that this agent is an unlikely asthma trigger.

The management of food-induced anaphylaxis and asthma

People with asthma are an important subset of patients with food allergy because of a higher risk of morbidity and mortality. This group should be identified and referred

to a specialist asthma and food allergy unit. They require careful specialist assessment and ongoing education.

The cornerstones of management of food allergy are avoidance of the offending food together with an anaphylaxis management plan. Accurate diagnosis of the food or foods causing adverse food reactions is critical to appropriate food avoidance advice. The diagnosis of food allergies requires careful history taking and confirmation of putative allergens with specific testing for IgE, and in some instances food challenge may be required in order to accurately identify the food or foods to be avoided. This allows for appropriate dietary advice focused on accurate avoidance of the offending allergen as well as the delivery of precise information and education to the patient and family. Nut and peanut allergies are particularly associated with severe anaphylaxis, and hence particular attention should be paid to the management of those with peanut and tree nut allergy. Despite careful avoidance, exposure to likely suspect foods is common with 14% of peanut-allergic individuals being exposed over 2 years [61]. Once patients are aware of what foods to avoid and how to avoid them, individuals with food allergy should be encouraged to notify restaurants or other eating venues in advance of attending to reduce the risk of inadvertent food allergen exposure [62]. Patients are advised to 'touch-test' foods prepared by others by rubbing the food on their lip before eating it.

Patients at risk of anaphylaxis will require an adrenaline auto-injector device with a management plan for both the patient and family. To be independent, young people will need to be educated and confident in the use of their auto-injector device. It is noteworthy that such education is frequently not effective in the first instance and requires repetition to ensure the correct delivery of an adrenaline auto-injector [63]. As evidence also exists that unstable asthma is associated with severe food allergy, excellent asthma control should be rigorously maintained in patients with asthma and food allergies. Unstable asthma appears to be a risk factor for severe anaphylaxis so that particular care needs to be observed by concurrent food allergy and asthma sufferers at these times. Furthermore, clinicians should particularly focus on adolescents and young adults as they appear to be the age group at most risk of death from anaphylaxis.

Conclusion

The co-existence of food allergy and asthma is an important risk factor for anaphylaxis mortality. Patients with these conditions should be managed in a specialist allergy unit to ensure accurate diagnosis, optimal avoidance advice and ongoing education in the use of an action plan for anaphylaxis and asthma. It is critical that the underlying asthma be actively managed and be under 'control' to reduce the likelihood of fatal food-related anaphylaxis.

Young adults and adolescents with asthma and food allergy demand particular attention. They require active and ongoing education. Importantly, they should have their adrenalin self-injecting pen with them at all times for accidental exposures. This group also needs to be warned against risk taking. Non-allergic food hypersensitivity appears to be of lesser importance in exacerbating asthma. Sulphites are the most likely implicated food additives and should be considered in the appropriate clinical context.

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