

Epidemiology of Food Allergy

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KEYWORDS

• Epidemiology • Food allergy • Prevalence • Incidence

Food allergy (FA) is perceived to be a common problem, especially during childhood. Accurate assessment of incidence and prevalence of FA has been difficult to establish, however, due to lack of universally accepted diagnostic criteria; hence, reported prevalence varies widely across the globe. Research has shown that perceived FA is 10 times higher than what can be confirmed by appropriate tests; hence, studies that rely on self-reports tend to show higher prevalence. The prevalence also depends on which foods or food additives were included and the type of adverse reaction considered. FA is defined as an immune-mediated adverse reaction to foods, and, if there is evidence of allergic sensitization (presence of immunoglobulin E) to a food, it is called IgE-mediated FA. Although many foods have been reported to cause IgE-mediated FA, most studies have focused on 4 common food groups. These are cow's milk, hen's egg, peanut/tree nuts, and fish/shellfish. There may be a true variation in the prevalence of FA in different regions of the world and a likely increase in prevalence has been observed in recent decades. This cannot be stated with confidence, however, without the use of consistent methodology and diagnostic criteria.

REPORTED FOOD ALLERGY

The natural history of FA is such that it is common in early childhood and becomes less common with age. Data from the United States showed that the annual incidence of doctor-diagnosed FA decreased from an average of 4.7% per year during the first 2 years of life to an average of 1.2% for the fifth and sixth years of age.¹ Thus, age is a major factor in determining FA prevalence. Even allowing for that, however, there

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remains considerable variation in the prevalence of FA in different populations. This is true for overall FA and also for allergy to individual foods. For example, maternally reported or doctor-diagnosed FA in early childhood was 6% in the United States² but only 1.7% in Israel.³ A study from Germany, assessing doctor-diagnosed FA prospectively from 1 to 6 years of age, reported this to be 4.6% at age 1, 6.6% at age 2, and 3.9% from ages 3 to 6.¹ US data from the National Health Interview Survey in 2007 showed reported FA during childhood to be 3.9%.⁴

No two studies of FA prevalence have used the same methodology. Hence, data from the same geographic region often report widely varying prevalences. In France, Penard-Morand and colleagues⁵ reported the prevalence of FA in children ages 9 to 11 to be 2.1%. In the same year, Roehr and colleagues⁶ estimated FA prevalence to be 6.7% in children ages 2 to 14. In Germany, overall prevalence of reported FA was 20.8% in 1537 subjects,⁷ whereas a telephone survey of 4477 adults in the United States showed self-reported FA to be 9.1%.⁸ An overall view of FA prevalence was estimated by a meta-analysis of 51 studies. Self-reported FA varied between 3% and 35%, whereas confirmed FA was on average 1% to 10.8% in 6 studies using oral food challenges.⁹ Only part of this variation reflects true variation in prevalence; the remainder likely relates to variation in how questions were formulated and which foods were included.

ALLERGIC SENSITIZATION

Allergic sensitization is defined by the presence of IgE antibodies to a specific food allergen. This may be assessed on skin prick test (SPT) or specific IgE as demonstrated in the serum using an immunoassay. The mere presence of IgE, however, does not necessarily mean that the subject will clinically react to the food. Thus, sensitization is often higher than true clinical FA. There is a direct correlation between the degree of sensitization and the likelihood of clinical FA. Previous studies have suggested various cutoffs, which might reliably predict FA while acknowledging that this may vary with the food and the age of the person. It has been suggested that specific IgE between 0.35 and 2 kilounits of allergen per litre (kU/L) indicates a low probability, between 0.7 and 5 kU/L moderate probability, and a high IgE level of greater than 5 kU/L a high probability of FA reaction.¹⁰ Oral food challenge, however, may still be required in some cases to establish or exclude the diagnosis of FA.

In a birth cohort study from Germany, the prevalence of food sensitization was reported to be 9.2% in children age 2 and 11.7% in children age 6, using the ImmunoCAP ($f \times 5$) (Phadia, Upsala, Sweden), which measures specific IgE to one or more of 6 common foods (cow's milk, hen's egg, peanut, wheat, cod fish, and soy).¹ In one of the largest multicenter studies, sera from 4522 individuals living in 13 countries were tested for IgE to at least one food allergen. Prevalence of sensitization to any of the 24 food allergens ranged from 7.7% in Reykjavik (Iceland) to 24.6% in Portland (Oregon, United States), with an overall prevalence of 16.2%.¹¹

The natural history of food sensitization mirrors that of clinically reported allergy. In a large unselected study ($n = 8203$), the overall prevalence of food sensitization in the United States was 16.8%. The highest prevalence (28%) was observed in children 1 to 5 years old and declined steadily with age to 13.0% in adults. The prevalence of milk (22%) and egg (13.9%) sensitization was again highest in children ages 1 to 5. Peanut sensitization was most common in older children and young adults (6–19 years, 10.7%; 20–39 years, 8.7%).¹⁰ Spontaneous remission of peanut and tree nut allergy is uncommon.¹² Savage and colleagues¹³ recently reported that 50% of children with soy allergy outgrow their allergy by age 7.

INCREASING PREVALENCE OF FOOD ALLERGY

There is some support in the literature of increasing FA prevalence. The number of hospital discharges with a diagnosis of FA among children (<18 years) increased from approximately 2000 to 10,000 between 2004 and 2006, respectively.⁴ A recent report from Boston suggests that food-induced anaphylaxis has been increasing in recent years, and, in addition to the classical 4 to 5 major foods, other food groups are increasingly associated with anaphylactic reactions. The report showed that the number of visits to a pediatric emergency department for food-induced anaphylaxis more than doubled from 14.9 per 10,000 visits in 2001 to 38.0 per 10,000 visits in 2006.¹⁴

COMMON FOOD ALLERGENS

Milk

Cow's milk protein allergy (CMA) is an immunologic reaction to the protein in cow's milk and can be IgE mediated, non-IgE mediated, or a mixture of both.¹⁵ CMA is seen in both children^{16,17} and adults.¹⁸ The pattern of IgE-mediated and non-IgE-mediated allergies and the proportion of children suffering from IgE-mediated versus non-IgE-mediated CMA also seems to vary between countries, with higher rates of non-IgE-mediated allergy seen on the Isle of Wight (United Kingdom)¹⁷ and in Denmark¹⁹ than in Spain²⁰ and Australia.²¹ Symptoms to cows' milk can also occur as a result of lactase deficiency (ie, lactose intolerance), which is a nonallergic food hypersensitivity reaction, because this indicates an inability or reduced ability to digest the carbohydrate in lactose.^{22,23}

CMA usually develops in the first year of life²⁴ and is one of the most common allergies seen in childhood, with a documented prevalence of between 0.3% and 3.5% in young children under 5 years and less than 1% in older children (**Table 1**).

In adults, however, CMA is reported as rare, with less than 0.5% of adults suffering from cow's milk allergy (**Table 2**). The majority of data regarding CMA in adults report only sensitization data, which vary between 0.1% and 2.3%, depending on the population studied (see **Table 2**). In Australia, Woods and colleagues³³ found that only 0.7% of atopic adults had a positive SPT to cow's milk proteins and none of those patients reported symptoms associated with the consumption of cows' milk. In this population, however, a total of 4.8% reported adverse reactions when consuming dairy products but were not sensitized to cow's milk proteins.

Resolution of CMA

Several studies have looked at the remission rate of CMA. In an unselected Danish population studied by Host and Halken,¹⁶ the overall prognosis of CMA, including both IgE-mediated and non-IgE-mediated CMA, was good, with a total recovery of 22 of 39 (56%) children at 1 year, 30 of 39 (77%) at 2 years, and 34 of 39 (87%) at 3 years. Venter and colleagues¹⁷ reported a remission rate of 80% by age 3 in an unselected population studied in the United Kingdom. The natural history of CMA was also investigated in two centers that routinely investigated children with CMA for development of tolerance: approximately 45% to 50% of children at 1 year, 60% to 75% of children at 2 years, 85% to 90% of children at 3 years, 92% of children at 5 and 10 years, and 97% of children at 15 years of age developed tolerance.^{34,35}

A recent study in the United States¹⁸ challenged these high rates of clinical tolerance, which particularly may not be relevant for children seen in tertiary referral centers, because only 5% were tolerant at age 4 and 21% at age 8.

Table 1
Prevalence of cow's milk allergy in children

	Children (Age, y)	Prevalence of Sensitization (%)	Prevalence of Allergy (%)	Method of Diagnosis
United States				
United States (2004) Sampson ²⁵	Not specified		2.5	Not specified
United States (2009) Branum and Lukacs ⁴	<18	12 2 (>90th percentile)	NA	NA
Europe				
France (2005) Rance et al ²⁶	2–14	NA	1.1	Reported cumulative incidence
France (2005) Rance et al ²⁶	2–5	NA	1.0	Reported
France (2005) Rance et al ²⁶	6–10	NA	0.6	Reported
France (2005) Rance et al ²⁶	11–14	NA	0.2	Reported
Norway (2001) Eggesbo et al ²⁷	2.5	NA	1.2	Food challenge
United Kingdom (2008) Venter et al ¹⁷	1	0.3	1.8	Food challenge
United Kingdom (2008) Venter et al ¹⁷	2	0.5	1.2	Food challenge
United Kingdom (2008) Venter et al ¹⁷	3	0.5	0.45	Food challenge
United Kingdom (2008) Venter et al ¹⁷	By 3	NA	2.3	Food challenge
United Kingdom (2008) Venter et al ²⁸	6	0.4	0.25	Food challenge
United Kingdom (2005) Pereira et al ²⁹	11	0.29	0.3	Food challenge
United Kingdom (2005) Pereira et al ²⁹	15	0.3	0.2	Food challenge
Denmark (1990) Host and Halken ¹⁶	By 3	1.1	2.2	Food challenge
Denmark (2005) Osterballe et al ¹⁹	3	NA	0.6	Food challenge
Rest of World				
Australia (1997) Hill et al ²¹	2	4.1 (Cohort of infants from high-risk families)	2.02	Estimated/calculate
China (2010) Hu et al ³⁰	0–2	1999: 3.2 2009: 6.2	1999: 1.6 2009: 3.5	Food challenge
Israel (2002) Dalal et al ³	0–2	NA	0.3 IgE mediated	History and sensitization

Abbreviation: NA, not available.

Table 2
Prevalence of milk allergy in adults and whole populations

	Adults (Age, y)	Prevalence of Sensitization	Prevalence of Allergy (%)	Method of Diagnosis	All Ages Sensitized (%)	All Ages Prevalence (%)
United States						
United States (2004) Sampson ²⁵	Not specified	—	0.3	Not specified	—	NA
United States (2009) Liu et al ¹⁰	NA	2.3 ^{a,11}	NA	NA	1–85 y: 5.7 sensitized	NA
United States (2007) Vierk et al ⁸	18→60	—	1.1	Self-reported doctor diagnosed	—	—
Europe						
France	20–40	0.1 ^{a,11}	NA	NA	NA	NA
Norway	20–40	1.3 ^{a,11}	NA	NA	NA	NA
United Kingdom	20–40	0.5 ^{a,11}	NA	NA	NA	NA
Denmark (2009) Osterballe et al ³¹	22	—	0.1	Food challenges	NA	NA
Denmark (2009) Osterballe et al ¹⁹	Parents of a cohort of 3 y olds	—	0.3	Food challenges	NA	NA
Germany (2004) Zuberbier et al ³²	20–40	1.3 ¹¹	NA	NA	NA (small sample)	4.5 of n = 104 with positive challenges (IgE mediated) 15.9 of n = 44 with positive challenges (non-IgE mediated)
Rest of World						
Australia (2002) Woods et al ³³	26–50	0.7 (Sensitized and tolerating milk)	4.8	Reported	NA	NA
	20–40	1.1 ^{a,11}	NA	NA	NA	NA

^a Random sample of adults who formed part of the European Community Respiratory Health Survey.

Several factors have been identified as playing a role in the development of tolerance, including (1) lower levels of cow's milk-specific serum IgE in the first 2 years of life than in those who remain allergic (median 1.8 kU/L vs 19.0 kU/L; $P < .001$)¹⁸; (2) absence of asthma or allergic rhinitis and never having been formula fed¹⁸; (3) the mechanisms involved in CMA, because individuals with delayed reactions have been shown to develop tolerance sooner than those with immediate reactions (In a Finnish study, 64% of those children with delayed symptoms developed tolerance at age 2, 92% at age 3, and 96% at age 4 vs 31% of those with immediate reactions developing tolerance at age 2, 53% at age 3, and 63% at age 4, respectively.)³⁶; and (4) IgE sensitization to cow's milk in the first year of life, associated with an increased risk of persisting CMA by age 3.³⁷

Egg Allergy

Egg allergy is, alongside milk, one of the most prevalent FAs in children. The estimated prevalence varies between 0.5% and 5% in early childhood (**Table 3**). Limited data available suggest that egg allergy is much less common in older children and adults, with an estimated prevalence in both cases of less than 0.5% (see **Table 3**; **Table 4**).

Resolution of Egg Allergy

In general, the prognosis for children with egg allergy is good.³⁹ Approximately half of egg-allergic children are tolerant by age 3 and 66% by age 5.^{40,41} Egg allergy is considered mainly IgE mediated, although some non-IgE-mediated reactions, mainly related to eczema, have been reported.^{17,42}

As with CMA, development of clinical tolerance may show reduced or delayed patterns when studying populations referred to tertiary centers. Retrospective data from the United States indicate that in these populations, only 11% of subjects develop clinical tolerance by age 4, 26% of subjects by age 6, 53% of subjects by age 10, and 82% of subjects by age 16.¹³

Risk factors identified for the persistence of egg allergy include (1) high levels of egg-specific serum IgE at diagnosis, (2) the presence of atopic disease, and (3) the presence of other FAs.¹³

Although several articles have reported peanut allergy recurring, only one case of egg allergy recurrence in adulthood after a period of tolerance (of approximately 23 years) has been reported. In this case, a 30-year old pregnant woman presented with egg allergy at the end of her pregnancy, which was characterized with immediate severe reactions and anaphylaxis.⁴³

Wheat

Many people believe that they have a wheat allergy or intolerance, but research suggests that in reality, this is confirmed in only a few cases. In the United Kingdom, in a large nationwide survey conducted in 1994, 20.4% of people reported that they had an allergy to any food, of which 0.9% reported being allergic to wheat.⁴⁴ A more recent consumer survey in 2009 reported that 4.5% of the population then considered themselves wheat allergic or wheat intolerant.⁴⁵ The limited data in the literature however, suggest a prevalence of less than 0.5% to 1% (**Tables 5 and 6**). Studying the prevalence of wheat allergy is further complicated by deciphering the underlying mechanism involved and whether or not the wheat-induced adverse reactions are indeed caused by IgE immune-mediated reactions or non-IgE immune-mediated reactions, wheat intolerance to non-allergic food hypersensitivity,⁴⁶ or an inability to digest the poorly absorbed carbohydrates in wheat.⁴⁶ In addition,

Table 3
Prevalence of egg allergy in children

	Children (Age, y)	Prevalence of Sensitization (%)	Prevalence of Allergy (%)	Method of Diagnosis
United States				
United States (2004) Sampson ²⁵	Not specified	—	1.3	Not specified
United States (2009) Branum and Lukacs ⁴	<18	7 Sensitized 2.2 Sensitized (>90th percentile)	NA	NA
Europe				
France (2005) Rance et al ²⁶	2–14	NA	0.8	Reported cumulative incidence
France (2005) Rance et al ²⁶	2–5	—	0.7	Reported
France (2005) Rance et al ²⁶	6–10	—	0.8	Reported
France (2005) Rance et al ²⁶	11–14	—	0.2	Reported
Norway (2001) Eggesbo et al ³⁸	2.5	NA	2.6	Food challenges
United Kingdom (2008) Venter et al ¹⁷	1	1.8	1.8	Food challenges
United Kingdom (2008) Venter et al ¹⁷	2	2.1	1.2	Food challenges
United Kingdom (2008) Venter et al ¹⁷	3	1.4	0.45	Food challenges
United Kingdom (2008) Venter et al ¹⁷	By 3	—	2.3	Food challenges
United Kingdom (2008) Venter et al ¹⁷	6	0.9	0.25	Food challenges
United Kingdom (2005) Pereira et al ²⁹	11	0.29	0.26	Food challenges
United Kingdom (2005) Pereira et al ²⁹	15	0.15	0.13	Food challenges
Denmark (2005) Osterballe et al ¹⁹	3	—	1.6	Food challenges
Rest of World				
Australia (2007) Hill et al ²¹	2	16.4 (Cohort of infants from high-risk families)	3.2	Estimated/calculated
China (2010) Hu et al ³⁰	0–2	1999: 7.3 2010: 15.5	2.9 (1999) 5.0 (2009)	Food challenges
Israel (2002) Dalal et al ³	0–2	—	0.5	History and SPT

Table 4
Prevalence of egg allergy in adults and whole populations

	Adults (Age, y)	Prevalence of Sensitization (%)	Prevalence of Allergy (%)	Method of Diagnosis	All Ages Sensitization (%)	All Ages Prevalence
United States						
United States (2004) Sampson ²⁵	Not specified NA	— 0 ^{a,11}	0.2 NA	Not specified NA	NA 1–85 y: 3.9 sensitized ¹⁰	NA NA
United States (2007) Vierk et al ⁸	18–>60	—	0.3	Self-reported doctor diagnosed	—	—
Europe						
France	20–40	0.1 ^{a,11}	NA	NA	NA	NA
Norway	20–40	1.5 ^{a,11}	NA	NA	NA	NA
United Kingdom	20–40	0.5 ^{a,11}	NA	NA	NA	NA
Denmark (2005) Osterballe et al ¹⁹	Parents of 3-y-old cohort	NA	0.1	Food challenges	NA	NA
Denmark (2009) Osterballe et al ³¹	22	—	0	Food challenges	NA	NA
Germany (2004) Zuberbier et al ³²	20–40	0.3 ¹¹	NA	NA	NA (small sample)	2.9% of n = 104 with positive challenges (IgE mediated)
Rest of World						
Australia (2002) Woods et al ³³	26–50	1.8 (Sensitized and 0.2 not tolerating egg)	1.3% (0.2% sensitised and not tolerating wheat + 1.1% not sensitised and not tolerating wheat)	History	NA	NA
	20–40	0.5 ^{a,11}	NA	NA	NA	NA

^a Random sample of adults who formed part of the European Community Respiratory Health Survey.

Table 5
Prevalence of wheat allergy in children

	Children (Age, y)	Prevalence of Sensitization (%)	Prevalence of Diagnosed Food Allergy (%)	Method of Diagnosis
United States				
—	NA	NA	NA	NA
Europe				
United Kingdom (2008) Venter ¹⁷	1	0	0.3	Food challenges
United Kingdom (2008) Venter et al ¹⁷	2	0.2	0.3	Food challenges
United Kingdom (2008) Venter et al ¹⁷	3	0	0.2	Food challenges
United Kingdom (2008) Venter et al ¹⁷	By 3	—	0.3	Food challenges
United Kingdom (2006) Venter et al ²⁸	6	0.4	0.4	Food challenges
United Kingdom (2005) Pereira et al ²⁹	11	0.57	0.0	Food challenges
United Kingdom (2005) Pereira et al ²⁹	15	1.2	0.1	Food challenges
Denmark (2005) Osterballe et al ¹⁹	3	NA	0	Food challenges
France (2005) Rance et al ²⁶	2–14	NA	0.18	Reported
Rest of World				
Australia (1997) Hill et al ²¹	2	NA	0.15	Estimated/calculated
China (2010) Hu et al ³⁰	0–2	1999: 0.3 2009: 0.5	NA	NA

Table 6
Prevalence of wheat allergy in adults and whole populations

	Adults (Age, y)	Prevalence of Sensitization (%)	Prevalence of Wheat Allergy (%)	Method of Diagnosis	All Ages Sensitization (%)	All Ages Prevalence
United States						
United States	20–40	6.1 ^{a,11}	NA	NA	NA	NA
United States (2007) Vierk et al ⁸	18–>60	NA	0.4	Self-reported doctor diagnosed	NA	NA
Europe						
United Kingdom	20–40	3.9 ^{a,11}	NA	NA	NA	NA
Denmark (2005) Osterballe et al ¹⁹	Parents of 3-y-olds cohort	NA	0	Food challenges	NA	NA
Denmark (2009) Osterballe et al ³¹	22	—	0	Food challenges	NA	NA
Germany (2004) Zuberbier et al ³²	20–40	6.0 ^{a,11}	—	—	NA (small sample)	14.9% Allergic to “flour” of n = 104 with positive challenges (IgE mediated) 6.8% Allergic to “flour” of n = 44 with positive challenges (non-IgE mediated/intolerance)
France	20–40	5.5 ¹¹	NA	NA	NA	NA
Rest of World						
Australia (2002) Woods et al ³³	26–50	2.2 (Sensitized and tolerating wheat)	1.3	Reported	—	—
Australia	20–40	7.9 ^{a,11}	NA	NA	NA	NA

^a Random sample of adults who formed part of the European Community Respiratory Health Survey.

wheat-induced symptoms may also indicate the presence of celiac disease (a non-IgE immune-mediated reaction to gluten).⁴⁷

Resolution of Wheat Allergy

Little is known about the natural history of wheat allergy, with only two studies available in the literature. A recent study from Finland⁴⁸ performed in children ($n = 28$) between 6 and 75 months of age, showed clinical tolerance in 59%, 69%, 84%, and 96% by ages 4, 6, 10, and 16 years, respectively. Both IgE-mediated and non-IgE-mediated symptoms were experienced by the children, presenting as skin symptoms (likely IgE mediated) and gut-related symptoms (likely non-IgE mediated). In a study from the United States,⁴⁹ 103 children with a history of reactions to wheat and sensitization to wheat were followed and the rates of tolerance noted were 29% by 4 years, 56% by 8 years, and 65% by 12 years. The median age of resolution of wheat allergy was approximately 6.5 years in this population. In both studies, only a minority of patients experienced ongoing wheat allergy into adolescence.

Factors that affected the development of clinical tolerance include (1) sensitization to gliadin with an SPT wheal of greater than or equal to 5 mm at the time of the diagnostic challenge, associated with a slower course of recovery from wheat allergy ($P = .019$),⁴⁸ and (2) higher wheat serum IgE levels, associated with poorer outcomes, although many children outgrew wheat allergy with even the highest levels of wheat-specific IgE.⁴⁹

Fish and Shellfish

A wide range of seafood has been implicated in food-allergic responses, but the species identified usually reflects their availability and consumption. Regarding fish, cod, tuna, salmon, trout, and plaice have been involved in food-allergic reactions in the United States, United Kingdom, and Europe. Less well known species, such as sea urchin roe, boiled razor-shellclam, krill, bonita, yellowtail, saurel, skipper, pomfret, hilsa, and bhetki, are often eaten in other countries, such as Spain,^{50,51} Japan,⁵² and India.⁵³ The prevalence of fish allergy is low in children ($\leq 0.2\%$ [Table 7]) and adults ($\leq 0.5\%$ [Table 8]).

Although the prevalence of shellfish allergy is still lower in children ($\leq 0.5\%$ [Table 9]) than adults ($\leq 2.5\%$ [Table 10]), it is slightly higher than fish allergy.

Seafood allergy is usually only associated with immediate symptoms related to an IgE-mediated food hypersensitivity response and is not normally implicated in delayed reactions. Few of the children sensitized to cod fish were diagnosed with a clinical allergy to fish in a UK study, with the majority of the children consuming cod fish on a regular basis.¹⁷ In this cohort, however, fish accounted for 11.4% of reported reactions, whereas shellfish accounted for only 3.8% of reactions.²⁹ A Danish study of a cohort of 3-year-old children and their older siblings reported they were unable to confirm any reported reactions to cod fish or shrimp in these children, although the actual prevalence of the same FAs in their parents was reported to be 0.2% to cod and 0.3% to shrimp (see Tables 7–10).¹⁹

Results from a US telephone survey suggest a prevalence of 0.2% for fish allergy in children and 0.5% in adults. Prevalence of an allergy to shellfish was 0.5% in children and 2.5% in adults. A more recent study suggests, however, that the prevalence of shellfish allergy is less than 0.3% for doctor-diagnosed allergy in adults. The reported fish allergy was higher than before (0.6% vs 0.5%).^{8,54}

Seafood allergy is thought to be lifelong. Some studies and case reports suggest, however, that remission may be possible. Solensky⁵⁶ reported a case of resolution of fish allergy in an adult with a history of fish anaphylaxis and, in a study of 32 children

Table 7
Prevalence of fish allergy in children

	Children (Age, y)	Prevalence of Sensitization (%)	Prevalence of Diagnosed Food Allergy (%)	Method of Diagnosis
United States and Canada				
United States (2004) Sampson ²⁵	Not specified	NA	0.1	Not specified
United States (2004) Sicherer et al ⁵⁴	0–17	NA	0.2	Reported
Canada (2010) Ben-Shoshan et al ⁵⁵	>18	NA	0.18	Self-reported doctor diagnosed
Europe				
United Kingdom (2008) Venter et al ¹⁷	1	0.3	0.1	Food challenges
United Kingdom (2008) Venter et al ¹⁷	2	0.5	0	Food challenges
United Kingdom (2008) Venter et al ¹⁷	3	0.5	0	Food challenges
United Kingdom (2008) Venter et al ¹⁷	By 3		0.1	Food challenges
United Kingdom (2006) Venter et al ²⁸	6	1.0	0	Food challenges
United Kingdom (2005) Pereira et al ²⁹	11	1.3	0.0	Food challenges
United Kingdom (2005) Pereira et al ²⁹	15	1.4	0.1	Food challenges
Denmark (2005) Osterballe et al ¹⁹	3	NA	0	Food challenges
Rest of World				
Australia (1997) Hill et al ²¹	2	NA	0.07	Estimated/calculated
China (2010) Hu et al ³⁰	0–2	1999: 0.3 2009: 0.75	NA	NA
Israel (2002) Dalal et al ³	0–2	—	0.1	History and SPT

Table 8

Prevalence of fish allergy in adults and whole populations

	Adults (Age, y)	Prevalence of Sensitization (%)	Prevalence of Fish Allergy (%)	Method of Diagnosis	All Ages Prevalence
United States					
United States (2004) Sampson ²⁵	Not specified	—	0.4	Not specified	NA
United States	20–40	0 ^{a,10}	NA	NA	NA
United States (2004) Sicherer et al ⁵⁴	18–>61	NA	0.5	Reported	NA
United States (2007) Vierk et al ⁸	18–>60	NA	0.6	Self-reported doctor diagnosed	NA
Canada (2010) Ben-Shoshan et al ⁵⁵	>18	NA	0.56	Self-reported doctor diagnosed	NA
Europe					
United Kingdom	20–40	0.2 ¹⁰	NA	NA	NA
Denmark (2005) Osterballe et al ¹⁹	Parents of 3 y olds	NA	0.2	Food challenges	NA
Denmark (2009) Osterballe et al ³¹	22	NA	0.1	Food challenges	NA
Germany (2004) Zuberbier et al ³²	20–40	0.9 ^{a,10}	NA	NA	Food challenges 0–79 years: 0
France	20–40	0 ¹⁰	NA	NA	NA
Rest of World					
Australia	20–40	0 ^{a,10}	NA	NA	NA

^a Random sample of adults who formed part of the European Community Respiratory Health Survey.

Table 9
Prevalence of shellfish allergy in children

	Children (Age, y)	Prevalence of Sensitization (%)	Prevalence of Diagnosed Food Allergy (%)	Method of Diagnosis
United Kingdom and Canada				
United Kingdom (2004) Sampson ²⁵	Not specified	NA	0.1	Not specified
United Kingdom (2004) Sicherer et al ⁵⁴	0–17	NA	0.5	Reported
United Kingdom (2009) Branum and Lukacs ⁴	<18	5.2	NA	NA
Canada (2010) Ben-Shoshan et al ⁵⁵	>18	NA	0.5	Self-reported doctor diagnosed
Europe				
United Kingdom (2008) Venter et al ¹⁷	1	0	0	Food challenges
United Kingdom (2008) Venter et al ¹⁷	2	0	0	Food challenges
United Kingdom (2008) Venter et al ¹⁷	3	0	0	Food challenges
United Kingdom (2008) Venter et al ¹⁷	By 3		0	Food challenges
United Kingdom (2008) Venter et al ²⁸	6	0	0.0	Food challenges
United Kingdom (2005) Pereira et al ²⁹	11	0.14	0.13	Food challenges
United Kingdom (2005) Pereira et al ²⁹	15	1.5	1.3	Food challenges
Denmark (2005) Osterballe et al ¹⁹	3	NA	0	Food challenges
France (2005) Rance et al ²⁶	2–14	NA	1.4	Reported cumulative incidence
France (2005) Rance et al ²⁶	2–5	NA	0.2	Reported
France (2005) Rance et al ²⁶	6–10	NA	1.8	
France (2005) Rance et al ²⁶	11–14	NA	1.2	
Rest of World				
Australia	2	NA	NA	NA
China (2010) Hu et al ³⁰	0–2	1999: 0 2009: 0.3	NA	NA

Table 10
Prevalence of shellfish allergy in adults and whole populations

	Adults (Age, y)	Prevalence of Sensitization (%)	Prevalence of Shell Fish Allergy (%)	Method of Diagnosis	All Ages Prevalence (%)
United States and Canada					
United States (2004) Sampson ²⁵	Not specified	NA	2	—	Sensitization: 5.9 Allergy: 0.99 ¹⁰
United States	20–40	0 (Shrimp) ^{a,11}	NA	NA	NA
United States (2004) Sicherer et al ⁵⁴	18–>61	NA	2.5	Reported	NA
United States (2007) Vierk et al ⁸	18–>60	NA	0.3	Self-reported doctor diagnosed	NA
Canada (2010) Ben-Shoshan et al ⁵⁵	>18	NA	1.69	Self-reported doctor diagnosed	NA
Europe					
United Kingdom	20–40	6.2 (Shrimp) ^{a,11}	NA	NA	NA
Denmark (2005) Osterballe et al ¹⁹	Parents of 3 y olds	NA	0.3 (Shrimp)	Food challenge	NA
Denmark (2009) Osterballe et al ³¹	22	NA	0.2 (Shrimp)	Food challenge	NA
Germany	20–40	4.4 (Shrimp) ^{a,11}	NA	NA	0–79: 0 ³²
France	20–40	7.0 (Shrimp) ^{a,11}	NA	NA	NA
Rest of World					
Australia (2002) Woods et al ³³	26–50	2002: 3.7 (0.9 Sensitized and not tolerating shrimp)	4.2 Reported a problem with ingesting shrimp (0.9 was sensitized and 3.3 not sensitized)	Reported	NA

^a Random sample of adults who formed part of the European Community Respiratory Health Survey.

with fish allergy, 5 seemed to lose their allergy.⁵⁷ In contrast, this has yet to be reported for those who are allergic to shellfish, with no change in specific IgE antibody levels to shrimp reported in 11 shrimp-allergic subjects over a 2-year period.⁵⁸

Peanut Allergy

Peanut allergy is the most common cause of food-induced anaphylaxis. In 1999, Sicherer and colleagues⁵⁹ reported that approximately 0.6% of the general population suffer from peanut allergy. Using the same methodology (random digit dialing), they reassessed the prevalence in 2002⁶⁰ and 2008.⁶¹ There was a small increase in prevalence to 0.8% in 2008. Ben-Shoshan and colleagues⁵⁵ reported recently a 1% peanut and 1.22% tree nut allergy prevalence in Canada. As shown in **Table 11**, other studies indicate prevalences that vary from 0.06% (Israel) to 5.9% (Sweden).

Table 11 Prevalence of reported allergy to peanut				
Country	Age Assessed (y)	N	Prevalence (%)	First Author
North America				
United States (2007)	18–85	4477	0.5	Vierk et al ⁸
United States (1999)	1–65	12,032	0.6	Sicherer et al ⁵⁹
United States (2003)	1–65	13,493	0.6	Sicherer et al ⁶⁰
United States (2010)	1–65	13,534	0.8	Sicherer et al ⁶¹
United States (2010)	1–85	8203	1.3	Liu et al ¹⁰
Canada (2010)	1–65	3613	1.0	Ben-Shoshan et al ⁵⁵
Canada (2003)	7	4254	1.5	Kagan et al ⁶²
Europe				
France (2005)	9–11	6672	0.3	Penard-Morand et al ⁵
France (2005)	2–14	2716	0.7	Rance et al ²⁶
Germany (2001)	25–74	1537	2.1	Schafer et al ⁷
Sweden (2004)	13–21	1451	5.9	Marklund et al ⁶³
United Kingdom (1996)	4	1218	0.5	Tariq et al ⁶⁴
United Kingdom (2002)	3–4	1273	1.0	Grundy et al ⁶⁵
United Kingdom (2005)	11	775	1.8	Pereira et al ²⁹
United Kingdom (2006)	6	798	1.9	Venter et al ²⁸
United Kingdom (2005)	15	757	2.5	Pereira et al ²⁹
United Kingdom (2008)	4–18	3943	1.9	Du Toit et al ⁶⁶
Rest of the World				
Australia (2002)	20–45	1141	0.6	Woods et al ³³
Israel (2002)	0–2	9040	0.06	Dalal et al ³
Israel (2008)	4–18	4657	0.2	Du Toit et al ⁶⁶
Singapore (2010)	4–6	4390	3.6	Shek et al ⁶⁷
Singapore (2010)	14–16	6450	1.18	Shek et al ⁶⁷
Philippines (2010)	14–16	11,322	1.29	Shek et al ⁶⁷
United Kingdom (2005)	15	649	2.6	Pereira et al ²⁹
United Kingdom (2002)	3–4	1246	3.3	Grundy et al ⁶⁵
United Kingdom (2005)	11	699	3.7	Pereira et al ²⁹

In a large US study, overall peanut sensitization in children and adults was 7.6%.¹⁰ In the EuroPrevall study, peanut sensitization varied from 0.8% in Norway to 9.3% in the United States, with an overall prevalence of 2.6%.¹¹ Various other studies have reported prevalences of peanut sensitization between 1% and 11% in Europe (Table 12).

Peanut allergy often starts in early childhood but a late-onset group has also been identified in whom peanut allergy occurs for the first time in early adult life.⁶⁸ A small proportion (20%) of children with peanut allergy outgrow their allergy by adolescence or early adult life and occasionally a relapse may occur.⁶⁹ A low level of peanut-specific IgE or negative SPT indicates this possibility. In one study, however, both children and adults were shown clinically reactive to peanut despite negative skin test.⁶⁸

Tree Nut Allergy

The prevalence of tree nut allergy in the United States has not changed much since 1997 and remained at approximately 0.5% (Table 13). The prevalence among children in European countries varied from 0.2% in France to 1.4% in the United Kingdom. In adolescents the prevalence was higher (2.2% to 4.1%) and in adults possibly even more so, with a reported 8.6% prevalence in one German study.

In a recent meta-analysis, the prevalence of perceived reactions to any tree nuts ranged from 0.03% to 1.4%, and the prevalence of sensitization was 0.02% to 0.7%.⁷¹ The EuroPrevall study reported a sensitization to walnut in 2.2% and to hazel nut in 7.8% of 4220 adults.¹¹

Allergy to tree nuts may start in childhood or occur for the first time in adult life. This allergy is usually persistent but approximately 10% of children grow out of tree nut allergy and among those with a low specific IgE (<5 kU/L), nearly 50% grow out.^{69,72}

Allergy to Plant Food

Few studies have assessed allergy to foods, such as fruits, vegetables, legumes (except peanut), cereals, and meat. In children, the reported prevalence of food allergy to fruit or vegetables was 0.8% (56 of 6672).⁵ In adults, however, perceived FA may be relatively high, as indicated by a study from Germany. This study showed a prevalence of reported allergy to vegetable of 3% and to fruits 6%.⁷ Allergic symptoms to various fruits and some vegetables may be due to cross-reactive proteins in pollens, which is termed pollen-fruit syndrome or oral allergy syndrome. In this syndrome, IgE antibodies to pollen proteins react against similar proteins present in fruits and

Table 12
Prevalence of sensitization to peanut

Country	Age Assessed (y)	N	Sensitized (%)	First Author
North America				
United States (2010)	1–85	8203	7.6	Liu et al ¹⁰
Europe				
Germany (2001)	25–74	4178	11.1	Schafer et al ⁷
France (2005)	9–11	6672	1.1	Penard-Morand et al ⁵
Sweden (2010)	2	1082	2.1	Schnabel et al ¹
Sweden (2010)	6	1982	5.2	Schnabel et al ¹
United Kingdom (1996)	4	981	1.1	Tariq et al ⁶⁴
United Kingdom (2008)	3	642	2.0	Venter et al ¹⁷
United Kingdom (2006)	6	700	2.6	Venter et al ²⁸

Table 13 Tree nut allergy prevalence studies					
	Age (y)	N	Reported Prevalence (%)	Sensitized (SPT or Specific IgE) (%)	History plus Sensitized (%)
North America					
United States (1999) Sicherer et al ⁵⁹	1–65	12,032	0.5	—	—
United States (2003) Sicherer et al ⁶⁰	1–65	13,493	0.6	—	—
United States (2010) Sicherer et al ⁶¹	1–65	13,534	0.6	—	—
United States (2007) Vierk et al ⁸	18–85	4477	0.5	—	—
Canada (2010) Ben-Shoshan et al ⁵⁵	1–65	3613	1.22	1	—
Europe					
France (2005) Penard-Morand et al ⁵	7–9	6672	0.2	—	—
France (2005) Rance et al ²⁶	2–14	2716	0.7	—	—
Germany (2001) Schafer et al ⁷	25–74	1537	8.5	17.8	—
Sweden (2004) Marklund et al ⁶³	13–21	1451	4.1	—	—
United Kingdom (1996) Tariq et al ⁶⁴	4	1218	—	0.2	0.2
United Kingdom (2008) Venter et al ¹⁷	3	891	—	—	0.7
United Kingdom (2006) Venter et al ⁷⁰	6	798	1.4	—	0.4
United Kingdom (2005) Pereira et al ²⁹	11	775	1.2	—	—
United Kingdom (2005) Pereira et al ²⁹	15	757	2.2	—	—
Rest of the World					
Israel (2002) Dalal et al ³	0–2	9040	0.03	—	0.02
Singapore (2010) Shek et al ⁶⁷	4–6	4339	3.41	—	—
	14–16	6436	0.81	—	—
Philippines (2010) Shek et al ⁶⁷	14–16	11,071	0.72	—	—

vegetables, causing oral symptoms on ingestion. In a study from Sweden, 142 of 1451 (9.8%) adolescents ages 13 to 14 had oral allergy syndrome-like symptoms, such as itching and swelling of the lips and oral cavity.⁶³ Those sensitized to fruit and vegetable are often also sensitized to birch pollen (Table 14). In another study, however, the prevalence of self-reported FA to fruits and vegetables was 10% in early childhood and increased to 33% in the population above age 30.⁷³

In a recent systemic review,^{71,74} the prevalence of perceived allergy to any fruit varied from 0.4% to 11.5%. In adults, reported allergy to individual fruits was less

Table 14
Allergic sensitization to common plant origin foods in a multicenter study

Food	Sensitized (Specific IgE) (%)	Sensitized (Excluding Birch Positive) (%)
Apple	4.2	2.0
Peach	5.4	2.8
Melon	1.6	1.0
Tomato	3.3	2.3
Carrot	3.6	2.0
Celery	3.5	1.8
Sesame	3.7	2.8
Poppy seed	1.8	1.4
Sunflower	2.1	1.8
Mustard	0.9	0.8
Banana	2.5	2.4
Kiwifruit	3.6	2.7
Wheat	4.6	3.4
Buckwheat	2.8	2.2
Rice	2.9	2.2
Corn	3.3	2.6
Soya	2.1	1.4

Data from Burney P, Summers C, Chinn S, et al. Prevalence and distribution of sensitization to foods in the European Community Respiratory Health Survey: a EuroPrevall analysis. *Allergy* 2010; 65:1182–8.

than 1% in all studies and similarly, the prevalence of sensitization (assessed by SPT) to specific fruits was well below 1%. Overall, for fruits and vegetables, the prevalence based on perception was generally higher than that based on sensitization, but for wheat and soy, sensitization was higher. The prevalence of challenge-proved FA to fruits ranged from 0.1% to 4.3%.

SUMMARY

FA is commonly reported, but there is a large discrepancy between reported and diagnosed FA. The true prevalence of FA in adults particularly needs further investigation. In children, the prevalence of FAs depends on the food studied and the country involved. It seems that there may be pockets of higher and lower prevalence across the world and FA may be increasing in some countries, such as the United States. Many foods are reported to cause symptoms of FA, but only 6 to 7 foods and ingredients form the core components of FAs, although the number of foods increases dramatically if plant FAs are included.

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