PEANUT ALLERGY

Open University: Short Course

Janice Joneja, Ph.D., RD 2011

Peanut Allergy

Janice M. Joneja, Ph.D., RD
2011

Peanuts are one of the most frequently cited causes of anaphylactic reactions in Western countries. Although all food allergies have the potential to induce anaphylaxis, some foods are more likely than others to cause potentially life-threatening reactions. Peanuts, tree nuts, fish, and shellfish are the foods most frequently identified as causes of anaphylaxis, and of these, peanuts seem to prompt the greatest concern.

The prevalence of peanut allergy in the Western world has been estimated to be between 1 in 10,000 and up to 1 in 200 in various populations and seems to have increased during the last decade. The incidence of peanut allergy was reported to have doubled in American children less than 5 years of age in the five years between 1998 and 2003 (Sampson 2003). One study reported that allergy to peanuts represents 28% of food allergies in children, develops before one year of age in 46% of cases, and under 15 years of age in 93% (Moneret-Vautrin et al 1998). Interestingly, the incidence of peanut allergy in other countries differs from that in the USA, and evidence suggests that peanut allergy in people moving from a part of the world with low levels of peanut allergy to America, who adopt the American diet and lifestyle, quickly assumes the same incidence as in the indigenous population.

The reasons for the severity of allergic reactions to peanuts, and the high incidence of the allergy in some countries and not others is presently largely unknown. This course will examine our current understanding about the mechanisms responsible for hypersensitivity reactions to peanut, why peanuts are so highly allergenic, and discuss strategies for the management of peanut allergy.

Incidence of Peanut Allergy and Anaphylaxis

The true incidence of peanut allergy in any population is largely a matter of speculation, based on questionnaires and occasional epidemiological studies. There is, however, data about fatalities arising from food allergy, which provides definite information about the incidence of fatal anaphylactic reactions to specific foods. For example, a 2001 report of 32 fatal cases of anaphylactic reactions to foods reported to a national registry in the USA, peanut was identified as the responsible food in 14 (67%) and possible cause in a further 6 (55%). Tree nuts were identified as the cause in 7 (33%) and possible cause in a further 3 (27%) of cases. The other 2 cases were thought to be due to milk and fish (1 [19%] each). Most of the victims were adolescents or young adults; all but one were known to have food allergy before the fatal event; and all but one were known to have asthma. In this report, peanuts and tree nuts accounted for more than 90% of the fatalities (Bock et al 2001).

In contrast, clinical allergy to peanut is far less prevalent in China, despite the high rate of peanut consumption. Allergy to all foods reportedly affects only 3.4% – 5.0% of the residents in Beijing, Guangdong, and the Sheng-Li Oil Fields, with fish, shrimp, crab and seaweed, but not peanut, being the major allergens. In 29 children aged 2 to 12 years with diagnosed food allergy in the Chinese population studied, none had signs of clinical allergy to peanut, although 2% of them were skin-test positive to peanut. Interestingly, the Chinese-American population living in the USA had an incidence of peanut allergy similar to that of the general US population (Beyer et al 2001).

In Australia, the risk of a fatal reaction to food, particularly in pre-school children is remote: an estimated one fatality in 30 years in the under 5-year-old population and 2 deaths in 10 years in the entire child population. Statistics seem to indicate that the proportion of Australian children at risk of a severe peanut reaction is only 0.25% even in a high-risk sector of the population. Skin-prick testing for peanut allergy in 456 Tasmanian children aged 7-8 years was negative in all, indicating that in this population the risk for peanut allergy was less than 0.2% (Kemp 2005).

Reports from the UK, indicate that fatal anaphylactic reactions to foods are rare. Of 20 fatal anaphylactic reactions reported each year to a national registry from 1998 to 2000, 5 were due to food, and none were caused by peanuts or nuts. Furthermore, peanuts and nuts did not seem to play a significant role even in non-fatal anaphylactic reactions (Pumphrey 2000).

A recent report from Germany indicated that of 103 cases of anaphylaxis reported by physician questionnaire in 2004, foods were the most frequent cause of the reaction (57%), followed by insect stings (13%) and immunotherapy injections (12%). Peanuts and tree nuts were the foods most frequently causing the reactions (Mehl et al 2005).

The prevalence of peanut allergy was determined to be between 1.5% and 1.76% of children in kindergarten through Grade 3 in randomly selected schools in Montreal, Canada. The study encompassed a questionnaire, followed by skin-prick tests, measurement of peanut-specific IgE, and oral peanut challenge tests in a response population of 4339 out of 7768 children surveyed (Kagan et al 2003).

These few examples indicate the wide disparity in anaphylactic reactions to peanuts in diverse populations throughout the world. Undoubtedly, the incidence of peanut allergy in the USA is higher than any other country. Various suggestions have been put forward to account for this observation, including differences in the varieties of peanuts consumed in different places, differences in the processing methods of peanuts offered in the retail market, and differences in the genetic predisposition of the population. These points are discussed further in this course (see below).

Symptoms Associated with Peanut Allergy

Symptoms reported to be due to peanut allergy include urticaria (hives), angioedema (tissue swelling, especially of the face), wheezing, asthma, nausea, vomiting, nasal congestion, itching, allergic conjunctivitis (itchy, watery eyes), and anaphylaxis. Contact dermatitis and hives from direct peanut contact have also been reported. In addition, eczema (atopic dermatitis) is often reported as a symptom of peanut allergy.

A 2003 review (Al-Muhsen et al 2003) indicates that the first allergic reaction to peanuts usually appears between 14 and 24 months of age, and commonly occurs at home.

The organ systems affected are:

- Skin (hives, reddening, tissue swelling, especially of the face)
- Respiratory tract (wheezing, noisy breathing, cough, breathing difficulty, throat tightening, nasal congestion)
- Gastrointestinal tract (vomiting, diarrhea, abdominal pain)
- Cardiovascular system (drop in blood pressure, irregular heart rate, cardiac arrest)

According to a voluntary registry in the USA (Sicherer et al 2001):

- 50% of peanut allergic children have symptoms in 1 organ system
- 30% have symptoms in 2 systems
- 10 – 15% in 3 systems
- 1% in 4 systems

Primary Sensitization to Peanut

In more than 70% of children with peanut allergy, symptoms develop at their first known exposure to peanuts. Because IgE-mediated allergic reactions require an initial exposure to an allergen to induce sensitization, and this primary exposure is characteristically symptom-free, it seems logical to assume that children who develop symptoms on the first observed contact with the allergen must have been exposed by an earlier, unknown event. Possible suggested routes of exposure include:

- Fetal exposure to peanut protein ingested by the mother during pregnancy
- Exposure to peanut protein in mother's breast milk
- Through the skin from peanut oils in topical creams
- Accidentally from siblings, caretakers and other unnoticed sources in the home or elsewhere

Except for accidental contact or ingestion, none of these routes of exposure have strong scientific validation, but preliminary evidence has been provided for the following:

- Although there is evidence for the presence of peanut protein, and blood cells (T cell lymphocytes) that can respond to antigens in amniotic fluid, there is no strong evidence to suggest that peanuts in the maternal diet during pregnancy can sensitize the fetus in utero (Hayday and Shannon 2003). Small quantities of all foods as part of a balanced diet should promote tolerance rather than sensitization to potential allergens (Wennergren 2009). However, some authorities recommend that during pregnancy the mother refrains from consuming *large* quantities of peanuts to reduce the risk of allergic sensitization of their baby (Sicherer et al 2010).

- Evidence for the passage of peanut protein from mother's diet into her breast milk during lactation was provided by a study using a very sensitive assay for peanut allergens in breast milk. Samples of breast milk were tested for the presence of peanut protein at various times after consumption of dry, roasted peanuts by a group of volunteers. The two major peanut allergens associated with anaphylaxis were detected in breast milk within one to three hours after ingestion in approximately 50% of the volunteers (Vadas et al 2001).

The authors suggest that exposure to peanut protein during breast-feeding might be a route for sensitization to peanut for at-risk infants.

However, there is a difference of opinion amongst clinicians and researchers regarding the response of the infant's immune systems to potentially allergenic proteins both in utero and in breast milk. Exposure to low doses of food proteins has been shown to tolerize the infant to allergens by "educating" the immune system to recognize the protein as safe. It is this process of immunological tolerization that allows us to consume foods without our immune systems mounting a defense against the animals and plants that we consume as food, all of which are distinct from, and foreign to, the human body. Exposure to peanuts and other food allergens during pregnancy, lactation and early childhood may be important in the development of immunological tolerance and may prevent allergic sensitization to these foods (López-Expósito et al 2009; Wellergren 2009).

Consequently, the concentration of peanut protein, the timing of the child's exposure to it, and the frequency with which the infant encounters it may lead to either allergic sensitization or to tolerization. In some cases, exposure to peanut protein in breast milk may actually protect against later development of peanut allergy. Therefore, at this stage in our knowledge, it would not be prudent to suggest that all lactating women avoid peanut products during breastfeeding. While this may protect some children from peanut sensitization, it may predispose other children to acquiring peanut allergy by preventing the process of tolerization (Lack et al 2003).

The possibility that the first exposure to peanuts may be through topically applied peanut oil-containing skin creams used to treat rashes over joints and in skin creases in young babies has been suggested by a number of studies (Lack et al 2003). It is recommended that any skin creams containing peanut oil should not be used on babies because of the risk for sensitization to peanut allergens through this route.

Introduction of Peanuts

For many years, the prevailing maxim for prevention of food allergy in at-risk infants was to reduce allergic sensitization by avoiding exposure to highly allergenic foods until the baby's immune and digestive systems were sufficiently developed to cope with the allergen. This included avoidance during pregnancy, lactation and introduction of solid foods. The first consensus document on the introduction of solid foods for the food-allergic infant was published in July 2006 by the Adverse Reactions to Foods Committee of the American College of Allergy, Asthma and Immunology (Fiocchi et al 2006). They recommended that introduction of the multiple allergens in solid foods to the allergic infant is preferably delayed until after 6 months of age. Until this age the authors suggest that the infant's immature digestive tract and immune system may increase the risk of sensitization and development of allergy. Furthermore, it was recommended that the most highly allergenic foods should not be introduced until after one year of age or later. Specific times of introduction were suggested as: cow's milk at 12 months; egg at 24 months; peanut, tree nut and fish at 3 years.

This thinking dramatically changed when it was realized that in spite of these restrictions, the incidence of childhood food allergy had significantly increased during the years these directives had been implemented. A ground-breaking study by DuToit et al in 2008 strongly suggested that early introduction of peanut during infancy, rather than avoidance, will prevent the development of peanut allergy. The authors demonstrated that Jewish children in the UK have a prevalence of peanut allergy that is 10-fold higher than that of Jewish children in Israel (prevalence in the UK was 1.85%; prevalence in Israel was 0.17% ($P < .001$). This difference was not accounted for by differences in atopy, social class, genetic background, or peanut allergenicity. The important indicator of outcome was the fact that Israeli infants consume peanut in high quantities in the first year of life, whereas UK infants avoid peanuts. Peanut is introduced earlier and is eaten more frequently and in larger quantities in Israel than in the UK. The median monthly consumption of peanut in Israeli infants aged 8 to 14 months is 7.1 g of peanut protein, and it is 0 g in the UK ($P < .001$). The median number of times peanut is eaten per month was 8 in Israel and 0 in the UK ($P < .0001$).

The more recent AAP paper, published in 2008 (Greer et al 2008) states, "*..the evidence…does not allow one to conclude that there is a strong relationship between the timing of the introduction of complementary foods and development of atopic disease"*.

Diagnosis of Peanut Allergy

Diagnosis of peanut allergy includes four major steps (Roberts et al 2005):

1. A medical history of allergy, and appearance of symptoms (see above) after exposure to peanut.
2. Positive skin tests for peanut
3. Presence of peanut-specific IgE in blood measured by a radioallergosorbent test (RAST)

4. Where necessary for confirmation, a positive challenge test in which symptoms develop after the patient consumes peanut in a supervised, controlled setting. This test is usually not carried out if there is a risk for a life-threatening anaphylactic reaction.

Threshold Doses of Peanut Provoking Allergy

Several studies have attempted to determine the smallest amount of peanut protein that is likely to trigger an allergic reaction in a peanut-allergic person. However, because there are so many variables associated with peanut allergy, it is very difficult to specify a dose of peanut that would elicit symptoms in all peanut-allergic people, or to define a dose that would be safe for all peanut-sensitive individuals. Variables in individual responses to peanut, include:

- Individual differences in the dose of peanut protein required to elicit symptoms among peanut allergic subjects: some people report symptoms after consuming peanut protein doses as low as 100 micrograms, while others do not develop symptoms until they have consumed 1 gram or more
- Differences in the type of symptoms elicited in different individuals, especially with regard to subjective (felt and reported by the patient, but not visible to the observer) and objective (visible and measurable) symptoms
- Variability in symptoms within the same individual on different occasions

One typical study that demonstrates the wide variability in the dose of peanut eliciting symptoms in different individuals was reported in 2002 (Wensing et al 2002). Twenty-six adult patients with a convincing history of peanut-related symptoms, presence of peanut-specific IgE in their blood, and a positive skin prick test response to peanut underwent double-blind, placebo-controlled food challenges with increasing doses of peanut. Threshold doses for allergic reactions ranged from a dose as low as 100 µg up to 1 gm of peanut protein. Fifty percent of the study population had an allergic reaction after ingestion of 3 mg of peanut protein.

Other studies have suggested that contact or inhalation of peanut protein may be sufficient to elicit a potentially life-threatening anaphylactic reaction, so usually it is wise for peanut-allergic people to avoid even the smallest quantity of peanut in foods, or in their environment (Sicherer et al 1999).

Peanut Anaphylaxis from Kissing

The possibility that anaphylaxis can be triggered by accidental contact with a minute amount of peanut in another person's saliva was suggested by the case of the November 2005 death of a Canadian teenager. It was at first thought that the 15-year old girl's death was caused by a kiss from her boyfriend, who had consumed a peanut butter sandwich

several hours before; the girl died four days later in hospital. The story made headlines in news media throughout the world. Subsequently, however, the result of an autopsy proved that the cause of death was not related to the peanut allergy, nor to the kiss. Apparently, the girl had asthma, and autopsy revealed that at the time of death she was suffering from a lack of oxygen to the brain, which caused serious damage, as a consequence of an asthma attack (CTV News 2006). The fear that the kiss might have been the cause of the reaction, however, prompted an immediate preliminary study of the saliva of 10 people who had eaten peanut. The peanut allergen was detectable in a majority of the subjects after eating but left the saliva after several hours. The authors of the study advised people with peanut allergies to make sure that their partners brush their teeth and then wait a number of hours before kissing, especially if the kissing is going to be passionate.

Allergenicity of Peanut Oil

Most of the oils available to the public for consumption are highly refined. The oil is subjected to physical and chemical methods of purification, such as degumming, refining, bleaching, and deodorization. It is then referred to as refined oil, and ingredients other than oil from the plant source are usually undetectable. This is the case with refined peanut oil. Until recently it was thought that refined peanut oil was completely free from peanut protein, and since an allergic reaction requires the presence of the allergenic protein, pure oil was considered to be incapable of eliciting an allergic response. However, modern extremely sensitive techniques allow detection of much smaller quantities of protein than previously, and it is now recognized that refined oil does indeed contain sufficient peanut allergen to elicit a reaction in highly sensitive individuals (Olszewski et al 1998). Nevertheless, all but the most highly sensitive peanut allergic individuals can consume the refined oil with impunity. However, if an oil is used to cook peanuts, peanut protein will be detectable in the previously pure oil, which would then be a great hazard to people with peanut allergy. The reuse of oil is common in some homes and in fast food outlets, so these sources of contamination with allergenic protein must constantly be avoided by individuals at risk for anaphylaxis (Hourihane et al 1997).

In contract, *crude* peanut oil does not go through the complex process of refining and may contain sufficient peanut protein to cause an allergic reaction in a peanut-sensitive individual. Cold-pressed oils by definition are not refined and therefore are potential sources of allergenic protein. Crude peanut oil is strongly flavoured and is often used in Oriental and East Indian cooking. As much as 3.3 micrograms of peanut protein per mL of oil has been measured in crude oils, which is quite enough to trigger an anaphylactic reaction in peanut-allergic individuals (Hoffman et al 1994).

In the interests of safety, individuals who are anaphylactic to peanut, nuts, or a grain are usually cautioned to avoid the oil derived from the allergenic plant because of the danger of the inclusion of traces of plant protein in the oil. Such individuals are particularly cautioned against use of cold-pressed oils from the plant food to which they are allergic.

Botanical Classification of Peanut

Peanut (*Arachis hypogea*) belongs to the biological family *Leguminosae* or 'legumes', which include peas, beans, soy, lentils, vegetable gums as acacia and tragacanth, and licorice.
Peanuts are unrelated botanically to nuts that grow on trees, and most people are able to eat a variety of tree nuts, such as walnuts, pecans, Brazil nuts, almonds, cashew nuts, hazelnuts (filberts), macadamia nuts, coconut, etc. without difficulty.

Nuts belong to a wide array of different biological families, for example:

- Walnuts and pecans belong to the *Judanglacea* family, which also include the hickory nut
- Almond is of the *Drupacea* family which includes peaches, apricots, plums, nectarine and cherries
- Cashews and pistachios belong to the *Anacardiacea* family group, which also includes mango
- Hazelnut (filbert) is part of the *Betulaceae* family, to which Birch trees belong
- Chestnuts belong to the *Fagaceae* family, which includes Beech trees
- Coconut belongs to the *Arecaceae* family group which also includes dates and palm trees
- Brazil nuts belong to the *Lecythidaceae*, a family of tropical trees which include the anchovy pear (*Grias cauliflora*) (a West Indian species with edible fruit used for pickles), and several lumber trees of South America.
- Macadamia nuts belong to a tropical plant family called the *Proteaceae* which includes the cone flower and pincushion tree

An allergy to nuts should be distinguished from an allergy to peanut and other legumes, otherwise the diet can become stressful and cumbersome if all traces all tree nuts are being avoided in addition to every trace of peanuts. However, because tree nuts are also highly allergenic foods, they are also frequent causes of strong allergic reactions and anaphylaxis. In addition, often no differentiation is made in the marketing of peanuts and nuts, and the two are often found together in "nut mixtures". Publications originating in the United Kingdom often include peanut when discussing nut allergy, and many people do not realize that in the strict definition of the term, peanuts are not the same as tree nuts. Furthermore, tree nuts easily become contaminated with peanuts, for example, when nuts and peanuts are sold in bulk, a utensil used to handle nuts has often been previously used with peanuts without cleaning in between. In the manufacture of candies, confectioneries and ice cream, there is frequent cross-contamination between nuts of different species and peanuts, so a person with severe peanut allergy is advised to avoid any product containing "nuts" because of the danger of encountering peanuts inadvertently.

"Mandalona" nut is one of the names given to a manufactured product made from deflavored, decolored peanut meal that is pressed into molds, reflavored and colored and sold as a cheaper substitute for tree nuts such as almonds, pecans, and walnuts. Persons with peanut allergy must be cautious when consuming any food that may contain such a product.

Peanut Allergens

Peanuts contain many different proteins, each with its own distinct structure. Several of these are allergenic, and can trigger IgE antibodies – each antibody molecule being specific to its inciting allergen. The term "peanut protein" may thus include some or all of the known peanut allergens.

The major peanut protein allergens have been characterized and named (Ref: Allergome). These include:

i. Ara h1
ii. Ara h2 (5 subtypes)
iii. Ara h3
iv. Ara h4
v. Ara h5
vi. Ara h6
vii. Ara h7
viii. Ara h8
ix. Ara h Agglutinin
x. Ara h LTP
xi. Ara h Oleosin
xii. Ara h TI

The relative quantity of each specific allergen will affect individuals differently when they are sensitized to one more than another. The assumption that all of the known allergens in a specific food are of equal importance to each subject may not hold true. Some of these allergens are more frequently detected as triggers of peanut-specific IgE in peanut-allergic individuals than others. For example, in a recent study, Ara h2 was recognized most frequently as the causative allergen in all tests for symptom-triggering, and induced a reaction at relatively low concentrations, whereas Ara h1 and Ara h3 were recognized less frequently and reacted only at 100-fold higher concentrations than Ara h2. The authors conclude that for their patient group Ara h2 was the most important peanut allergen, that a few of the other proteins may be important allergens, but less frequently, and at higher concentrations (Koppelman, Wensing et al 2004).

In another study (Becker et al 2001) the authors calculated the prevalence of sensitization to every single allergen in a population of 40 patients sensitized to peanut. Their results indicated that in addition to Ara h1 (prevalence 65%) and Ara h2 (prevalence 85%), Ara h4 (53%) was also a major allergen, while Ara h5 (13%), Ara h6 (38%) and Ara h7 (43%) were minor allergens. Interestingly, although Ara h6 was considered a minor allergen, the authors found that sensitization to Ara h6 was associated with more severe clinical symptoms than most of the other allergens.

Future research will undoubtedly reveal which of the allergenic proteins in peanuts are most important to a specific population, and which are affected by various processing methods in the manufacture of foods that include peanuts. This information may then explain why certain populations are at risk for peanut allergy and why others are

relatively safe. Furthermore, improved understanding of the molecular structure of the major peanut allergens and the peanut-specific immune response will eventually lead to effective methods for diagnosis, therapy, and possibly prevention strategies for peanut allergy (Scurlock and Burks 2004).

Effects of Cooking Methods on Peanut Allergenicity

Roasting of peanuts has been demonstrated to increase their allergenicity, compared to the same variety of peanut when processed by other methods (Maleki et al 2000). This means that roasted peanuts, which is the form in which most peanuts and peanut butter is consumed in the United Sates because of the improved flavor and taste, is more likely to trigger a severe anaphylactic reaction than the fried or boiled peanut, which is the form in which people in countries such as China consume the food (Beyer et al 2001). It is a documented fact that the incidence of peanut allergy in China is virtually non-existent, whereas in the USA about 3 million people report being allergic to peanuts.

It appears that the main factor influencing the increase the allergenicity of peanut during roasting is due to the higher temperature reached in roasting in comparison to frying or boiling. Peanut proteins, like many highly allergenic proteins in foods, are not degraded at high temperatures, and retain their potential to trigger an allergic reaction even after cooking. However, the excessively high temperatures achieved during roasting has the effects of changing the protein to a more insoluble form compared to the protein in the raw state, or when it is subjected to only slightly elevated temperatures. The increase in insolubility means that less of the protein will move into solution in the digestive tract, and will consequently be more protected from digestive enzymes and destruction by gastric acid than a more soluble form would be. This results in undegraded peanut protein being available as a continuous source of major allergens in the digestive tract long after the same protein in a more soluble form would have been removed from the system (Kopper et al 2005).

In addition, roasting, compared to boiling and frying, altered the binding of peanut specific IgE antibodies to the individual allergens Ara h1, Ara h2, and Ara h3, even though the amounts of each of the allergens were similar in all the peanut samples tested. This indicates that the method of heating the proteins significantly affects the interaction between the allergen and the antibodies produced by the allergic individual, and therefore the likelihood of the individual developing symptoms, and probably the severity of the symptoms in a subsequent reaction (Beyer et al 2001; Mondoulet et al 2005).

Other factors that might affect the allergenicity of peanut proteins are thought to include the type and variety of the peanut (of which there are about 14,000), the conditions under which the peanut has been grown, and the degree of maturation of the peanuts (Chung et al 2003).

Fat Content and Peanut Allergenicity

Another factor that may affect the allergenicity of peanut is the fat content of the food in which the peanut is included as an ingredient. A 2003 study of six people with a known peanut allergy demonstrated that the reactivity of four of the subjects was significantly increased when the peanut was included in a lower-fat meal compared to when the food had a high fat content (Grimshaw et al 2003). The subjects reacted to a dose equivalent to 23 times less peanut with the low-fat recipe compared to the higher fat meal.
This study emphasises the probability that a number of factors have a significant effect on the response of individuals to peanuts in their diets, and that the level of fat is one such factor to be considered in assessing the risk of certain foods to food-allergic consumers.

Cross-Reactivity of Peanuts with Other Legumes

A common problem faced by peanut-allergic people is whether they should avoid all legumes, for example soy, chick peas, lentils, beans, peas, licorice, and all other members of the *Leguminoceae* family, to which peanut belongs. In laboratory experiments, cross-reactivity between peanut and soy, for example, is quite frequent, but in clinical trials, the cross-reactivity is quite uncommon; one study reports only 2 out of 41 peanut-allergic patients reacted mildly to other members of the legume family (Bernhisel-Broadbent et al 1989).

Previously, the incidence of soy allergy was reported to be low compared to other highly allergenic foods, but recent studies seem to suggest that the prevalence of soy allergy is increasing (Sicherer et al 2000). It has been suggested that the increase in the incidence of soy allergy, especially in children, could be the result of exposure to the allergenic protein, in the form of soy-based infant formulas, in early infancy when the child is at highest risk for allergic sensitization. In addition, recent research suggests that an association between peanut and soy allergy could arise from cross-sensitization from soy-based infant formulae, Soy and peanuts contain a similar allergen, and it is thought that exposure to the soy allergen in infant formula could prime the child's immune system to respond to the peanut allergen, even when he or she shows no signs of allergy to soy (Lack et al 2003). As a result, the child could exhibit allergic symptoms on an apparent first exposure to the peanut.

Some researchers consider that there is under-reporting of anaphylactic reactions to other legumes, such as soy. A report of life-threatening anaphylactic reactions to foods from 1993 to 1996 included six reactions to soy, and four fatal cases of anaphylaxis, probably due to soy, in asthmatic peanut-allergic patients with no previously known allergy to soy (Foucard et al 1999). Other legumes that have been linked to peanut allergy include chick peas (garbanzo beans), lentils, and lupine flour (Moneret-Vautrin et al 1999)

Although many members of the legume family can be allergenic and trigger allergic symptoms in individuals sensitized to them, there is no evidence to support the thinking that peanut-allergic individuals should avoid all legumes. Allergy to multiple foods is

usually due to reactions to botanically unrelated, highly allergenic foods, such as peanut, egg, milk, shellfish, fish and tree nuts. Highly allergic individuals are most likely to react to the most highly allergenic foods for their age group. Therefore, allergy to specific foods is usually a result of independent sensitization to each individual food allergen.

Prognosis and Management of Peanut Allergy

In spite of the high incidence of peanut allergy in some Western countries, and the potential for severe anaphylactic reactions to the food, peanut allergy, like many early food allergies, can be outgrown. In 2001 pediatric allergists in the U.S. reported that about 21.5 per cent of children will eventually outgrow their peanut allergy (Skolnick et al 2001), and those with a mild peanut allergy, as determined by the level of peanut-specific IgE in their blood, have a 50% chance of outgrowing the allergy (Fleischer et al 2003). In contrast, only about 9% of patients are reported to outgrow their allergy to tree nuts (Fleischer et al 2005).

Studies indicate that when there is no longer any evidence of symptoms developing after a child has consumed peanuts, it is preferable for that child to *eat peanuts regularly*, rather than avoid them, in order to maintain tolerance to the peanut. Children who outgrow peanut allergy are at risk for recurrence, but the risk has been shown to be significantly higher for those who continue to avoid peanuts after resolution of their symptoms (Fleischer et al 2004).

The Peanut-Safe Environment

Currently there is strong pressure for public places such as schools, hospitals and airliners to be "peanut-free" in an attempt to protect vulnerable individuals, especially children and young adults, from accidental exposure to peanuts. However, declaring an environment as "peanut-free" in itself could be a mistake, since when people assume that the area is free from contamination, they may relax their guard, and thus be at risk for inadvertent exposure to the allergen. "Peanut-safe" indicates that although people using the facility are requested to avoid bringing peanuts into the area, and special precautions are in place to reduce the possibility of exposure of the at-risk population, there must be constant vigilance on the part of everyone to maintain the environment in a peanut-safe condition.

Important measures for a peanut-safe environment should include:

- Ensuring that all personnel in a peanut-safe facility are well-informed about the dangers to the peanut-allergic person of accidental contamination
- Clear strategies for maintaining the facility in a peanut-safe condition, with strict rules about cleaning

- Informing everyone entering the facility to avoid introducing peanuts into the area, and the reasons for the restrictions
- Education of the peanut-allergic person concerning avoidance of their own exposure to peanuts, including:
    - Avoidance of foods likely to contain, or be contaminated by, peanuts
    - Being aware of all terms on food labels that would indicate the possible presence of peanut
    - Carrying an Epipen of injectable adrenalin, and being familiar with its use in case of accidental exposure and an allergic reaction
    - Wearing a MedicAlert tag or bracelet in case of loss of consciousness in an allergic reaction
- Informing all staff in the facility about emergency procedures should anyone in the area develop symptoms and require medical treatment. Such information should include:
    - Familiarity with the use of the Epipen where appropriate
    - Contacting key care-givers such as parents and guardians of children, and the person's doctor or other health provider
    - Instructions for transporting the individual to the nearest hospital in the quickest way possible

The Peanut-Safe Life-Style

The person who is allergic to peanuts must avoid peanuts in any and every form as long as he or she develops symptoms after consuming the food.

It is important that all sources of peanut should be carefully avoided if there is even a moderate risk of an anaphylactic reaction to it. However, there is no evidence that even a severe allergy to peanut requires avoidance of all other legumes. Avoidance of legumes such as soy, lentils, dried peas and beans is only necessary when allergy to the individual foods has been identified. It is also unnecessary to avoid tree nuts, which are botanically unrelated to peanuts, unless the individual has an allergy to them. However, because of the risk of contamination of tree nuts, especially nut mixtures, to contain, or to be contaminated by peanuts, a person who has demonstrated allergy to peanuts is usually advised to avoid nuts of all types in the interests of safety.

Peanuts are widely used in the food industry owing to their nutritive value and to their taste. Many manufactured foods contain peanuts, resulting in an increased risk for inadvertent ingestion of peanuts by allergic individuals. Additionally, contamination of intended peanut-free products with traces of peanuts during the manufacturing process has sometimes resulted in several fatal and near-fatal allergic reactions. It is essential for the peanut-allergic individual to be familiar with all the terms on a product label that could indicate the presence of peanut (See Table 1).

Note on peanut oil

Although highly refined peanut oil contains barely detectable amounts of peanut protein, and should be safe for consumption by most peanut-allergic individuals, there is no guarantee that any peanut oil is *completely* free from peanut protein. A person anaphylactic to peanut is strongly advised to avoid all types of peanut oil entirely. Cold-pressed oils (also labelled “pure-pressed”, “expeller pressed” or “unrefined”) in particular should be avoided.

Important nutrients in peanut

Peanuts supply niacin, magnesium, vitamin E, manganese, pantothenic acid, chromium and in smaller amounts vitamin B6, folacin, copper and biotin. These nutrients are easily replaced by including meat, whole grains, legumes and vegetable oils in the diet, and therefore a diet completely free from peanuts should not pose any nutritional risk to the average consumer. However, strict vegans may need to eat tree nuts and seeds in order to obtain adequate quantities of high-grade proteins, so this population is strongly advised to undergo a careful investigation of their allergic reactivity to tree nuts and seeds, so that they can be assured that these nutritious foods can be consumed in safety.

Table 1

Terms on Food Labels that Indicate the Presence of Peanut Protein

(Joneja 2003)

Ingredients Indicating the Presence of Peanut		
• Peanut protein	• Mandalona nuts	• Goober nuts
• Hydrolysed peanut protein	• Artificial nuts	• Goober peas
• Peanut oil	• Nu-nuts flavored nuts	
• Cold-pressed peanut oil	• Beer nuts	
• Peanut butter	• Mixed nuts	
• Peanut flour		

Products that May Contain Peanut		
• Marzipan (almond paste)	• Baked goods	• Vegetable oil
• Prepared soups	• Cookies	• Hydrogenated vegetable oil
• Dried soup mixes	• Candies	• Vegetable shortening
• Chili	• Chocolate bars	
• Egg rolls	• Prepared and frozen desserts	
• Thai dishes	• Ice cream with nuts	
• Chinese dishes	• Chocolate ice cream	
• Satay sauces	• Cheese balls and other cheese products coated with nuts	

The Peanut Allergic Baby

Protein from peanuts in the mother's diet can pass into her breast milk and cause allergic symptoms in the breast-fed baby (Vadas et al 2001). If the breast-fed infant is allergic to peanut protein, the elimination of all peanut and peanut containing products from the mother's diet should be beneficial. If peanut elimination only partially eases the infant's distress, carefully kept exposure diaries by the mother, may isolate other possible dietary or medication irritants. Use of skin creams containing peanut oil, or vegetable oil from undeclared sources, should be carefully avoided

The Peanut–Free Diet

Table 2. Foods Allowed/Restricted for a Peanut-Free Diet
(Joneja 2003)

Type of Food	Foods Allowed	Foods Restricted
Milk and Milk Products	♦ Milk ♦ Cream ♦ Plain yogurt ♦ Buttermilk ♦ Ice cream made with allowed ingredients ♦ Plain cheese ♦ Sour cream ♦ Quark ♦ Dips made with allowed ingredients	♦ Milk-based desserts and confectioneries (e.g. ice cream) containing peanuts or nuts ♦ Chocolate ice cream or other milk-based confectioneries unless labeled "peanut-free" ♦ Cheese foods (e.g. slices, dips, spreads, cheese balls) containing nuts or undisclosed ingredients
Breads and Cereals	♦ Any breads, buns, or baked goods that are known to be free from peanut and peanut oil ♦ Plain cooked grains ♦ Plain oatmeal ♦ Regular Cream of Wheat® ♦ Ready to eat cereals without added oil or nuts ♦ Homemade granola without peanut ♦ Dried pasta	♦ Commercial or homemade baked goods made with peanut oil or peanuts ♦ Baked goods made with undisclosed sources of "nuts", oil, or shortening ♦ Baking mixes ♦ Ready-to-eat cereals with added oils and nuts, such as granola
Vegetables	♦ All pure vegetables and their juices	♦ Vegetable dishes with sauces containing peanuts, peanut oil or unknown nuts or oils ♦ Salads with dressings containing unknown oil or nuts ♦ Vegetables canned in undisclosed oils

Type of Food	Foods Allowed	Foods Restricted
Fruit	♦ All pure fruit and fruit juices	♦ Fruit dishes containing peanuts or nuts ♦ Fruit dishes made with oil or shortening of unknown origin
Meat, Poultry, and Fish	♦ All pure fresh or frozen meat, poultry or fish ♦ Fish canned in broth, water or non-peanut oils	♦ Meat, poultry or fish dishes made with peanut or undisclosed nuts or oils ♦ Fish canned in undisclosed oils ♦ Chinese dishes ♦ Thai dishes ♦ Egg rolls ♦ Commercial chilli ♦ Vegetarian burgers unless labelled "peanut-free" ♦ Peanut protein
Eggs	♦ All without restricted ingredients	♦ Egg dishes prepared with oils or nuts from unknown sources ♦ Egg rolls
Legumes	♦ All pure legumes except peanut ♦ Tofu	♦ Peanut and peanut products including: - Artificial nuts - Goober nuts - Goober peas - Hydrolyzed peanut protein - Mandalona nuts - Mixed nuts - Peanut butter - Peanut flour - Peanut oil - Peanut protein ♦ Legume dishes containing peanut, or oils, or nuts from undisclosed sources

Type of Food	Foods Allowed	Foods Restricted
Nuts and Seeds	♦ All packaged plain, pure nuts and seeds ♦ All pure nut and seed oils and their butters, such as - sesame tahini - almond butter - almond paste - cashew butter	♦ Mixed nuts ♦ Mandalona nuts ♦ Artificial nuts ♦ Nuts or oils of undisclosed origin ♦ Goober nuts ♦ Goober peas
Fats and Oils	♦ Butter ♦ Cream ♦ Pure vegetable, nut, or seed oil with source specified (except peanut) ♦ Lard ♦ Meat drippings ♦ Gravy made with meat drippings	♦ Peanut oil ♦ Salad dressings that list "oil" without revealing source ♦ Margarine, unless source of all oils is revealed and is peanut-free
Spices and Herbs	♦ All pure herbs and spices ♦ Blends of herbs and spices, without added oils	♦ Seasoning packets with undisclosed ingredients ♦ Vegetables such as garlic or sun-dried tomatoes packed in oil, unless source of oil is disclosed and is peanut-free
Sweets and Sweeteners	♦ Plain sugar ♦ Honey, ♦ Molasses ♦ Maple syrup ♦ Corn syrup ♦ Pure chocolate ♦ Pure cocoa ♦ Artificial sweeteners ♦ Homemade cookies and candies with allowed ingredients	♦ Chocolates with unknown ingredients ♦ Chocolate bars ♦ Marzipan (almond paste) ♦ Cookies and candies ♦ Any confectionery containing nuts unless specified to be peanut-free

References

Allergome: A database of allergenic molecules
http://www.allergome.org/script/search_step2.php

Al-Muhsen S, Clarke AE, Kagan R. Peanut allergy: an overview. Canadian Medical Association Journal 2003;168:1279-1285
http://www.cmaj.ca/cgi/reprint/168/10/1279 (complete article)

Bernhisel-Broadbent J, Taylor S, Sampson HA. Cross-allergenicity in the legume botanical family in children with food hypersensitivity. II. Laboratory correlates. J Allergy Clin Immunol 1989;84(5 Pt 1):701-709
http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=90038088&dopt=Citation

Beyer K, Morrow E, Li X-M, Bardina L, Bannon GA, Burks AW, Sampson HA. Effects of cooking methods on peanut allergenicity. J Allergy Clin Immunol 2001;107(6):1077-1081
http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=11398088&dopt=Abstract

Bock SA, Munoz-Furlong A, Sampson HA. Fatalities due to anaphylactic reactions to foods. J Allergy Clin Immunol 2001;107(1):191-193
http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=11150011&dopt=Abstract

Chung SY, Butts CL, Maleki SJ, Champagne ET. Linking peanut allergenicity to the process of maturation, curing and roasting. J Agric Food Chem 2003;51(15):4273-4277
http://pubs.acs.org/cgi-bin/abstract.cgi/jafcau/2003/51/i15/abs/jf021212d.html

CTV News:
Teenager with peanut allergy dies after a kiss. Sat. Nov. 26 2005
Lack of oxygen, not peanut butter, killed girl: Mon. Mar. 6 2006
http://www.ctv.ca/servlet/ArticleNews/story/CTVNews/20060306/teen_peanutbutter_060306/20060306/

Du Toit G1, Katz Y, Sasieni P, Mesher D, Maleki SJ, Fisher HR, Fox AT, Turcanu V, Amir T, Zadik-Mnuhin G, Cohen A, Livne I, Lack GJ. Early consumption of peanuts in infancy is associated with a low prevalence of peanut allergy. Allergy Clin Immunol. 2008 Nov;122(5):984-91.
http://www.jacionline.org/article/S0091-6749(08)01698-9/abstract

Fiocchi A, Assa'ad A, Bahna S. for the Adverse Reactions to Foods Committee of the American College of Allergy, Asthma and Immunology. Food allergy and the introduction of solid foods to infants: a consensus document. Annals of Allergy, Asthma & Immunology July 2006;97(1):0–21 http://www.sciencedirect.com/science/article/pii/S1081120610613646

Fleischer DM, Conover-Walker MK, Christie L, Burks AW, Wood RA. The natural progression of peanut allergy: Resolution and the possibility of recurrence. J Allergy Clin Immunol; 2003;112(1):183-189 http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?db=PubMed&cmd=Retrieve&list_uids=12847497&dopt=Citation

Fleischer DM, Conover-Walker MK, Christie L, Burks AW, Wood RA. Peanut allergy: Recurrence and its management. J Allergy Clin Immunol 2004;114(5):1195-1201 http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=15536431&dopt=Citation

Fleischer DM, Conover-Walker MK, Matsui EC, Wood RA. The natural history of tree nut allergy. J Allergy Clin Immunol 2005;116(5):1087-1093 http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=16275381&dopt=Citation

Foucard T, Malmheden Yman I. A study on severe food reactions in Sweden – is soy protein an underestimated cause of food anaphylaxis? Allergy 1999;54:261-265 http://www.blackwell-synergy.com/links/doi/10.1034/j.1398-9995.1999.00924.x/abs/

Greer FR, Sicherer SH, Burks AW, and the Committee on Nutrition and Section on Allergy and Immunology Effects of Early Nutritional Interventions on the Development of Atopic Disease in Infants and Children: The Role of Maternal Dietary Restriction, Breastfeeding, Timing of Introduction of Complementary Foods, and Hydrolyzed Formulas. Pediatrics 2008;121(1):183-191 http://pediatrics.aappublications.org/content/121/1/183.full.pdf

Grimshaw KE, King RM, Nordlee JA, Hefle SL, Warner JO, Hourihane JO. Presentation of allergen in different food preparations affects the nature of the allergic reaction – a case series. Clin Exp Allergy 2003;33(11):1581-1585 http://www.blackwell-synergy.com/doi/abs/10.1046/j.1365-2222.2003.01795.x;jsessionid=cH4ctdqJt1wdYE_RBN?journalCode=cea

Hayday K, Shannon S. Are early exposures linked with childhood peanut allergy? The Journal of Family Practice 2003;52(7) On-line journal http://www.jfponline.com/Pages.asp?AID=1503

Hoffman DR, Collins-Williams C. Cold-pressed oils may contain peanut allergen. J Allergy Clin Immunol 1994;93:801-802
http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=8163791&dopt=Abstract

Hourihane JO'B, Bedwani SJ, Dean TP, Warner JO. Randomised, double-blind, cross-over challenge study of allergenicity of peanut oils in subjects allergic to peanuts. BMJ 1997;314:1084-1088 http://bmj.bmjjournals.com/cgi/content/full/314/7087/1084

Joneja JMV. Dealing with Food Allergies. Bull Publishing Company, Boulder, Colorado 2003. 189-194 ISBN 0-923521-64-X
https://www.bullpub.com/catalog/dealing-with-food-allergies

Joneja JMV. Dealing with Food Allergies in Babies and Children. Bull Publishing Company, Boulder, Colorado.. October 2007. ISBN 978-1933503-05-9
https://www.bullpub.com/catalog/dealing-with-food-allergies-in-babies-and-children

Kagan RS, Joseph L, Dufresne C, Gray-Donald K, Turnbull E, Pierre YS, Clarke AE. Prevalence of peanut allergy in primary-school children in Montreal, Canada. J Allergy Clin Immunol 2003;112(6):1223-1228
http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=14657887&dopt=Citation

Kemp **A.** Severe peanut allergy in Australian children, The Medical Journal of Australia MJA 2005; 183 (5): 277
http://www.mja.com.au/public/issues/183_05_050905/letters_050905_fm-3.html

Koppelman SJ, Wensing M, Ertmann M, Knulst AC, Knol EF. Relevance of Ara h1, Ara h2 and Ara h3 in peanut-allergic patients, as determined by immunoglobulin E Western blotting, basophil-histamine release and intracutaneous testing: Ara h2 is the most important peanut allergen. Clin Exper Allergy 2004;34(4):583-590
http://www.blackwell-synergy.com/links/doi/10.1111/j.1365-2222.2004.1923.x/abs/

Kopper RA, Odum NJ, Sen M, Helm, RM, Stanley JS, Burks AW. Peanut protein allergens: the effect of roasting on solubility and allergenicity. Int Arch Allergy Immunol 2005;136(1):16-22
http://content.karger.com/ProdukteDB/produkte.asp?Aktion=ShowFulltext&ProduktNr=224161&Ausgabe=230625&ArtikelNr=82580

Lack G, Fox D, Northstone K, Golding J. Factors associated with the development of peanut allergy in childhood. New Eng J Med 2003;348(11):977-985
http://content.nejm.org/cgi/content/abstract/348/11/977

López-Expósito I, Song Y, Järvinen KM, Srivastava K, Li XM. Maternal peanut exposure during pregnancy and lactation reduces peanut allergy risk in offspring. J Allergy Clin Immunol. 2009 Nov;124(5):1039-1046. http://www.ncbi.nlm.nih.gov/pubmed/19895992

Maleki SJ, Hurlburt BK. Structural and functional alterations in major peanut allergens caused by thermal processing. J AOAC Int 2004;87(6):1475-1479 http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=15675461&dopt=Citation

Mehl A, Wahn U, Niggemann B. Anaphylactic reactions in children – a questionnaire-based survey in Germany. Allergy 2005;60(11):1440-1445 http://www.blackwell-synergy.com/doi/abs/10.1111/j.1398-9995.2005.00909.x

Mondoulet L, Paty E, Drumare MF, Ah-Leung S, Scheinmann P, Willemot RM, Wal JM, Bernard H. Influence of thermal processing on the allergenicity of peanut proteins. J Agric Food Chem 2005;53(11):4547-4553 http://pubs.acs.org/cgi-bin/abstract.cgi/jafcau/2005/53/i11/abs/jf050091p.html

Moneret-Vautrin DA, Rance F, Kanny G, Olsewski A, Gueant JL, Dutau G, Guerin L. Food allergy to peanuts in France-evaluation of 142 observations. Clinical & Experimental Allergy 1998;28(9):1113-1119 http://www.ingentaconnect.com/content/bsc/cea/1998/00000028/00000009/art00370

Moneret-Vautrin DA, Guerin L, Kanny G, Flabbee J, Fremont S, Morisset M. Cross-allergenicity of peanut and lupine: the risk of lupine allergy in patients allergic to peanuts. J Allergy Clin Immunol 1999;104(4 Pt 1):883-888 http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=10518837&dopt=Citation

Olszewski A, Pons L, Moutete F, Aimone-Gastin I, Kanny G, Moneret-Vautrin DA, Gueant JL. Isolation and characterization of proteic allergens in refined peanut oil. Clinical & Experimental Allergy 1998;28(7):850-859 http://www.ingentaconnect.com/content/bsc/cea/1998/00000028/00000007/art00325

Pumphrey RS. Lessons for management of anaphylaxis from a study of fatal reactions. Clin Exper Allergy 2000;30(8):1144-1150 http://www.blackwell-synergy.com/doi/abs/10.1046/j.1365-2222.2000.00864.x

Roberts G, Lack G. Diagnosing peanut allergy with skin prick and specific IgE testing. J Allergy Clin Immunol 2005;115(6):1291-1296 http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=15940149&dopt=Citation

Sampson HA. Food allergy. J Allergy Clin Immunol 2003;111(2 Suppl):S540-547
http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=12592300&query_hl=15&itool=pubmed_docsum

Scurlock AM, Burks AW. Peanut allergenicity Ann Allergy Asthma Immunol 2004;93(5 Suppl 3):S12-18
http://www.ingentaconnect.com/content/acaai/aaai/2004/00000093/A00305s3/art00003

Sicherer SH, Furlong TJ, DeSimone J, Sampson HA. Self-reported allergic reactions to peanut on commercial airliners. J Allergy Clin Immunol 1999;104(1):25-27
http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=10400859&dopt=Abstract

Sicherer SH, Sampson HA, Burks AW. Peanut and soy allergy: a clinical and therapeutic dilemma. Allergy 2000;55:515-521
http://www.blackwell-synergy.com/links/doi/10.1034/j.1398-9995.2000.00119.x/abs/

Sicherer SH, Furlong TJ, Minoz-Furlong A, Burks AW, Sampson HA. A voluntary registry for peanut and tree nut allergy: characteristics of the first 5149 registrants. J Allergy Clin Immunol 2001;108(1):128-132
http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=11447394&dopt=Citation

Sicherer SH, Wood RA, Stablein D, Lindblad R, Burks AW, Liu AH, Jones SM, Fleischer DM, Leung DY, Sampson HA.
Maternal consumption of peanut during pregnancy is associated with peanut sensitization in atopic infants. J Allergy Clin Immunol 2010 Dec;126(6):1191-1197
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3018157/

Skolnick HS, Conover-Walker MK, Koerner CB, Sampson HA, Burks W, Wood RA.
The natural history of peanut allergy. J Allergy Clin Immunol 2001;107(2):367-374
http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=11174206&dopt=Abstract

Tariq, S. M., Stevens, M., Matthews, S., Ridout, S., Twiselton, R., and Hide, D. W.
Cohort study of peanut and tree nut sensitisation by age of 4 years. Br. Med. J. 1996;**313**, 514-517 http://bmj.bmjjournals.com/cgi/content/full/313/7056/514 (complete article)

Vadas P, Wai Y, Burks W, Perelman B. Detection of peanut allergens in breast milk of lactating women. JAMA 2001;285(13):1746-1748
http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=11277829&dopt=Abstract

Wennergren G. Acta Paediatr. 2009 Jul;98(7):1085-1087 What if it is the other way around? Early introduction of peanut and fish seems to be better than avoidance. http://onlinelibrary.wiley.com/doi/10.1111/j.1651-2227.2009.01342.x/abstract;jsessionid=B3C7D70B73B52C97372B7C6EF2708CAB.f01t01

Wensing M, Penninks AH, Hefle SL, Koppelman SJ, Bruijnzeel-Koomen CA, Knulst AC. The distribution of individual threshold doses eliciting allergic reactions in a population with peanut allergy. J Allergy Clin Immunol 2002;110(6):915-920 http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=12464959&dopt=Citation

Glossary of Terms

Term	Definition
Immunoglobulin E (IgE)	Abbreviated IgE. A class of immunoglobulins that includes the antibodies elicited by an allergic substance (allergen). A person who has an allergy usually has elevated blood levels of IgE. IgE antibodies attack and engage the invading army of allergens
IgE mediated hypersensitivity	Immunological hypersensitivity due to formation of excessive amounts of immunoglobulin E (IgE) in response to otherwise innocuous antigens (allergens) in the environment or in food. Another name for allergy.
Total IgE level	The quantity of IgE antibodies in a subject's serum, without reference to any specific antigen to which the IgE may be formed
Allergen-specific IgE	IgE antibodies formed in response to a specific allergen. These are the antibodies that are detected in allergy tests such as RAST and skin tests. When a person eats the allergenic food, antigens (allergens) in the food couple with the allergen-specific IgE, which is attached to specialized cells, called mast cells. The allergen-IgE coupling causes the release of specialized chemicals stored within the mast cells, called inflammatory mediators. The release of inflammatory mediators causes symptoms typical of allergy when they react with body cells and tissues (see Allergic disease)
Allergic sensitization	Presence of IgE antibodies against environmental and/or food antigens.
Immunologic tolerance	Usually applied to foods. The immune system recognizes that the food is foreign to the body, but as a result of a complex interplay of antigen recognition and response, immunological defence systems remain dormant and the foreign antigens are deemed to be safe. This process allows us to eat foods, which of course originate from sources entirely foreign to the body, without developing symptoms, except in the case of allergy.
Allergic diseases	Disorders caused by exposure to specific antigens (allergens) in people who are sensitized to them. Includes allergic asthma, allergic rhinoconjunctivitis (hayfever), atopic dermatitis/eczema, food allergy, hypersensivity to drugs, allergy to insect stings

Atopy/atopic	A term applied to people exhibiting symptoms of allergic disease
Anaphylaxis	A severe and potentially life-threatening allergic reaction. Symptoms can include hives, swelling (especially of the lips and face), difficulty breathing (either because of swelling in the throat or an asthmatic reaction), vomiting, diarrhea, cramping and a fall in blood pressure
Skin testing	The skin test is a method of measuring the patient's level of IgE antibodies to specific allergens. Using diluted solutions of specific allergens, the physician either injects the patient with the solutions (intradermal test), or scratches (scratch test), or pricks the skin with a sharp lancet (prick test) through the allergen drop. A positive reaction appears as a small raised area (wheal) surrounded by a flat red area (flare) on the skin (the wheal and flare reaction). The size of the reaction, measured in millimetres (mm), is usually considered to be a rough estimate of the level of IgE in the patient's body formed against the allergen in the test reagent. A positive reaction to the skin test, especially with food allergens, does not always mean that the patient is will develop symptoms when the food is eaten.
RAST	RAST is an abbreviation for RadioAllergoSorbent Test, a trademark of Pharmacia Diagnostics, which originated the test. RAST is a laboratory test used to detect IgE antibodies to specific allergens in a sample of blood. The aim with RAST, as with skin tests, is to check for allergic sensitivity to specific antigens. In the test, the sample of blood is mixed with substances known to trigger allergies. The test measures the level of IgE allergy antibodies to specific antigens in the blood which are present if there is an allergic reaction.
Food Challenge test	Subjects consume the test food under controlled conditions and their response is monitored for onset of allergy symptoms.