

# FOOD ALLERGY IN CHILDREN WITH ECZEMA

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## **Abstract**

### **Introduction:**

It is well documented in the literature that many people with eczema also have a food allergy. A food allergy is defined as an adverse reaction to food, involving the immune system. While the prevalence of true food allergy is difficult to determine, the presence of a positive skin prick test (SPT) indicates the child has been sensitised to that food. The primary aim of this project was to investigate in children with eczema; food sensitisation, most common sensitising foods, clinical reactions to foods, associated symptoms, and family history of allergic disease. The secondary aim was to investigate the natural history of food allergy, in particular peanut allergy. It has been suggested that the prevalence of peanut allergy is increasing, however controversy exists as to the actual reason for this. Numerous studies indicate that most children will grow out of their food allergy, however evidence suggests many children do not grow out of their peanut allergy. Peanut is also the leading cause of food induced anaphylaxis. A final aim of this study is to investigate severe food reactions in children with eczema.

### **Methods:**

Data was collected from medical and dietetic notes of 418 children with eczema aged up to 12 years seen at the RPAH Allergy Unit between 1995 and 1997. Parents and carers were sent a questionnaire to obtain additional information about the child's symptoms, food allergen avoidance and clinical reactions to foods. All information was entered into a confidential database for analysis. To investigate the natural history of peanut allergy, those children with a positive SPT to peanut at first presentation to the clinic were invited back in for a follow up SPT.

### **Results:**

Approximately half of all children with eczema were sensitised to one or more foods. The most common food allergens were egg, peanut, milk, fish, wheat, sesame and soy. The most common symptoms associated with food sensitisation and eczema were asthma and rhinitis. Peanut sensitisation was present in one third of the children with eczema. Peanut sensitisation tends to persist in children longer than other food allergens. There is a trend for food sensitisation to decrease and aero-allergen sensitisation to increase with age. Peanut and milk were the most common foods causing anaphylaxis.

### **Discussion:**

To determine the actual role food allergy plays in eczema is beyond the scope of this study, however results indicate food sensitisation is common. The prevalence of peanut sensitisation is one of concern particularly due to the often severe nature of reactions and the persistence of allergy into adulthood. These children require special care and attention and parents and carers should be educated on how to avoid or reduce the risk of exposure to allergens and suitable procedures in the event of accidental exposure to food allergens.

## **INTRODUCTION**

Why people often have unpleasant reactions to food is still not fully understood. What is known is that many people, including infants and children suffer adverse reactions to food. The term “adverse food reactions” is used to describe the abnormal response exhibited in certain individuals after eating foods that are otherwise tolerated by the majority of people in the community. These reactions appear to be an increasingly common situation in the population (Esteban 1992). Adverse reactions to foods should be categorised as either food allergy or as food intolerance. It is essential to correctly identify the type of adverse reaction for proper management of the problem.

The importance of foods as causes for allergic disease has been the subject of ongoing debate for many years. The confusion and controversy surrounding this area exists as a result of contradictory and conflicting results from clinical studies and from the absence of precise and regulated terms (Esteban 1992, Schwartz 1992). There is a lack of universal acceptance of definitions, diagnosis and management. What is agreed upon is that the incidence and morbidity of allergic diseases such as eczema, asthma and rhinitis are increasing in the population for reasons not fully understood (Bousquet & Michel 1995, Croner 1992, Zeiger, Heller and Mellon, 1995). It is therefore of great importance to conduct research in the area to help in developing strategies to promote allergy prevention.

## **DEFINITIONS**

### **FOOD ALLERGY**

Food Allergy is a term often misused to describe any adverse reaction to foods both immunological and non immunological. Food Allergy or “food hypersensitivity” should only be used to describe an immune reaction as a result of the production of IgE (immunoglobulin E) antibodies after ingestion of proteins in commonly eaten foods such as egg, peanut, milk, fish, soy and wheat and other nuts (Schwartz 1992, Sampson & Metcalfe 1992, Esteban 1992). The incidence of food allergy in children has been estimated between 0.3 and 7.5% in the population (Zeiger 1990)

A true food allergy is easy to recognise. Symptoms are reproducible after ingestion of specific food proteins, often within an hour or less, including itching, burning and swelling around the mouth and throat on contact, abdominal cramps, nausea, vomiting, diarrhoea or urticaria after ingestion. In extreme cases contact with the food can anaphylactic shock (Clarke, McQueen, Samild & Swain 1996, Loblay & Swain 1986, Loblay & Swain 1992)

Skin Prick testing (SPT) or RAST (Radioallergosorbent test) is used to diagnose food allergy in combination with clinical examination and dietary modification (Clarke *et al.* 1996). SPT or RAST are not acceptable as the only way to diagnose food allergy except in the case of severe food-induced anaphylaxis. A negative SPT has a predictive accuracy of 95% therefore is useful, supportive and safe test for excluding food allergy (Caffarelli

*et al.* 1995, Walker 1992). Food sensitisation is determined by a positive SPT result, which indicates the immune system is involved. A positive SPT is predictive of clinical symptoms approximately 50% of the time, as some individuals with a positive SPT have no clinical symptoms when challenged with the sensitising food (Sampson 1983). A positive SPT is considered significant if wheal diameter on skin is  $\geq 3$ mm in diameter. A positive SPT may however be useful to predict a food allergy before the introduction of that food into the diet. (Caffarelli *et al.* 1995).

Conformation of a diagnosis of a true food allergy can be achieved by a double blind placebo controlled food challenge (DBPCFC) sometimes referred to as the “gold standard” (De Weck 1988, 1995, Esteban 1992, Sampson & Metcalfe 1992, Schwartz 1992, Walker 1992). Very few studies have used DBPCFC in selected groups of the population however these studies provide the best estimate of the real prevalence of food allergy in the population. For the purpose of this study food sensitisation (positive SPT) will be investigated. The prevalence of clinical food allergy is beyond the scope of this study as DBPCFC were not conducted. In the case of food-induced anaphylaxis, food challenges are not indicated for safety reasons. The diagnosis is therefore a presumptive one based on clinical history and positive SPT.

Once the diagnosis is made, management of food allergy involves complete avoidance of the relevant food allergens (Loblay & Swain 1992, Scadding & Brostoff 1988). Regular consultation with dietitian is essential to ensure that allergens are eliminated without reducing the nutritional adequacy of the diet. Appropriate alternative foods must be

provided in the diet to replace foods known to cause adverse reactions (Clarke *et al.* 1996).

## FOOD INTOLERANCE

Although IgE mediated allergic reactions are best characterised and easiest to diagnose, other adverse food reactions must not be ignored. In comparison to a food allergy a food intolerance does not involve the immune system. It is a response to the pharmacological effects of predominantly natural chemicals (salicylates, amines and glutamates) found in a wide range of plant foods and other foods. Some food additives (preservatives, flavourings and colourings) may also be involved in food intolerance. Common symptoms include headaches, mouth ulcers, abdominal discomfort, nausea, diarrhoea, hives and behavioural disturbances. The symptoms of food intolerance are dose related, and delayed reactions are common due to the cumulative nature of the chemicals involved. Also the reaction times are variable, leading to a more difficult diagnosis. Allergy and intolerance can also coexist in the same individual (Clarke, McQueen, Samild & Swain 1996, Loblay & Swain 1986, Loblay & Swain 1992)

The most effective method of diagnosing food intolerance is to eliminate all relevant chemicals for a period, and then to systematically reintroduce chemicals that have been eliminated. The reintroduction of specific chemicals, known as challenges, is done by including certain foods into the diet that are known to contain the chemical being tested, or by administering purified chemicals and placebos in capsule form, thereby identifying the particular food compound to which the individual is sensitive. The results of the

challenges enable dietitians to prescribe tailored diets that will avoid adverse reactions to food as much as possible (Loblay & Swain 1986).

Because patients do not always return for follow up, the effectiveness of dietary modification is often unknown to the dietitian. Information indicating dietary outcome of patients whose diets have been modified in response to adverse reactions to food, enables us to refine our understanding of the effectiveness of dietary intervention methods used.

## **LITERATURE REVIEW**

### **FOOD ALLERGY/ SENSITISATION AND ECZEMA**

Food allergic disease can present in patients with an established disease diagnosis such as asthma, rhinitis and eczema. It is believed that a proportion of these patients will have a food allergy as a primary or relevant cause of their chronic disease (Edwards 1995). Eczema (Atopic dermatitis) in children is the chronic disease that has been most extensively investigated with respect to food allergy (Burks, Mallory, Williams and Shirrel, 1988, Edwards 1995, Sampson and McCaskill 1985).

Eczema is a multifaceted disorder affecting 5-6% of children in the population. The child with eczema has thick and rough skin that may exude serous fluid and blood. Itchy lesions may appear on the face and spread to the rest of the body. The consequent scratching associated with eczema accounts for much of the skin damage. (reference). Onset is usually in the first twelve months of life and resolution of symptoms is usually only partial. Recent research suggests that the incidence of eczema is increasing

(Ferguson 1992, Taylor, Wadsworth & Peckhan 1984)

The role of food allergy (IgE-mediated food hypersensitivity) in the pathogenesis of eczema has long been proposed, however this remains unclear (Ring 1988). There is adequate literature to suggest that food allergy is common in children with eczema and that certain foods may irritate eczema and make the distressing skin disease worse (Ferguson 1992). The skin of children with eczema tends to release histamine from the sensitised mast cells when the antigen arrives through the blood stream. In these children, appropriate elimination and challenge protocols have shown approximately 60% responded positively to food challenges with skin, gastrointestinal and respiratory symptoms (Sampson & McCaskill 1992).

The relationship between IgE-mediated food reactions and eczema is determined by the presence of positive SPT to the suspected foods as well as the clinical reaction observed in response to these foods on oral challenge. A direct causal link between food allergy and the eczema is difficult as many foods that provoke a skin symptom during a food challenge do not provoke obvious symptoms when the food is ingested on a regular basis. The onset of chronic eczema may be delayed for up to 24-48 hours and its relationship to food may be unrecognised until the food is withdrawn for one or two weeks and then re-introduced (13). A positive test is indicative that the food identified may be a causative factor in eczema but is not proof. More proof can only come from long term studies in which foods identified by challenge are removed from the diet with clinical improvement

and clinical worsening when they are reintroduced (reference)

The estimated prevalence of food allergy ranges from 33% to 62% in populations of paediatric patients with severe eczema (Sampson and McCaskill 1992, Sampson 1983, 1988, 1991). Sampson & McCaskill investigated 113 patients in a placebo controlled food challenge study. Skin symptoms were observed in 84% of cases and delayed exacerbation of eczema was observed. The most frequent offending foods were egg (42%), peanut (19%), milk (11%), soy (5%) and wheat (5%). When the identified food was eliminated, significant skin improvement was observed.

Burks *et al.* (1988) estimate 33% of children seen at allergy and dermatology clinics with eczema had food allergy contributing to skin symptoms. Moneret-Vautrin (1994) conducted a survey in 35 children with food allergy and found that in 91% of cases there was a previous history of atopic dermatitis either alone or in association with asthma. Upon diagnosis of food allergy, 81% of children also had eczema.

These studies have provided strong evidence for the role of food allergy in the pathogenesis of eczema and it is now recommended that children less than five years of age with eczema should be investigated for food allergy (Schwartz 1992). Food intolerance is also thought to play a role in the pathogenesis of eczema but there is limited research in the area.

## RISK FACTORS FOR ALLERGY

It is believed that both genetic and environmental factors are critical in the development of atopy (Carter 1995, Zeiger 1990), and that a family history of allergy is the strongest predictor of development of allergic disease (Walker 1992). Approximately 25% of the population have allergic disease, the risk of developing allergic disease is increased with positive family history, up to 50% if one parent is allergic and further increased to approximately 75% if both parents are allergic. However, there is still a risk of developing atopic disease even when there is no family history (Bousquet & Michel 1995, Walker 1992, Wolfe 1995).

Any attempt at food allergen avoidance should be accompanied by consideration of both genetic and environmental factors (Carter 1995, Zeiger 1990). An area of uncertainty is the effect of food allergen avoidance in infants of atopic parents. According to Ferguson (1981) parental atopy as well as dietary antigens introduced in infancy contribute to the development of eczema. Some studies suggest that reduced exposure to food and aeroallergens (dust mite, cat dander and rye grass pollen) in early infancy feeding may reduce the incidence and severity risk of IgE atopic disorders such as eczema, food allergy and asthma in later childhood (Hill & Hosking 1993)

## PREVENTION OF ALLERGIC DISEASE

Zeiger *et al.* (1992) investigated the effect of maternal avoidance of allergens on development of allergy. They conducted a study that involved a prophylactic group and control group, the former was either breastfed with mothers excluding foods regarded as highly antigenic from their diets or using hypoallergenic formula feeds. The latter group were fed either breast or bottle but no efforts were made to reduce food and aero-allergen (environmental) exposure. In the first year of life there was a higher incidence of asthma and urticaria in the control group, but differences between groups diminished at two years of age. The cumulative prevalence of food allergy remained lower in the prophylactic group at three and four years of age.

A similar study was conducted by Hide *et al.* (1996). In addition to maternal and infant food allergen avoidance, reduced exposure to house dust mite (*Dermatophagoides pteronyssinus*) was implicated in the prophylactic group. At a one year follow up the prophylactic group showed significantly less total allergy, asthma and eczema than the control group. After two years there was a significantly reduced incidence of food and aero-allergen sensitisation, as assessed by skin prick testing. At a four year follow up, the prophylactic group continued to show a reduced prevalence of atopy including eczema. The findings of this study suggest that a combined approach, involving avoidance of food as well as aero-allergens, leads to a significant reduction of risk of atopic disease. The extent to which food or aero-allergen avoidance alone would produce such benefits is unknown. This issue warrants further study before any definite conclusions can be drawn.

Controversy surrounds the general advice given about allergen avoidance in pregnancy and lactation. Breast fed children can be sensitised by, and later react to foodstuffs contained in breast milk (Ferguson 1981). Breast milk is still considered the preferred means of feeding the child but modification of the maternal diet during lactation may be helpful in reducing the prevalence of atopic eczema and food allergy in infancy. It is generally recommended that common allergens, egg, nuts, cows milk, soy and wheat should be avoided (Wolfe 1995, Schwartz 1992).

If the maternal diet is to be restricted, it is essential the mother receives advice at early stage. Close medical and dietetic supervision must be available to ensure the long-term diet is nutritionally adequate. Avoiding food allergens at an already stressful time may be difficult and cause unnecessary anxiety. For these reasons, maternal dietary modification and reduction in aero-allergen exposure should be reserved for infants at high risk of atopy (Scadding & Brostoff 1988, Vandenplas, Deneyer, Sacre & Loeb 1988, Wolfe 1995).

## PEANUT ALLERGY

There has been a considerable increase in the rate of referrals for food allergy, the most obvious rise has been in the case of peanut and other nut allergies (Ewan 1996, Hourihane, Dean & Warner 1996, Tariq et al 1996). It has been estimated to have increased in prevalence in the USA by 95% over the past ten years. There are a number of

reasons suggested in the literature for this apparent increase in incidence of peanut allergy. It is likely that some of this increase can be attributed to increased awareness and changes in diagnosis in recent years (Taylor *et al.* 1984). Furthermore, the prevalence of atopic diseases such as asthma, rhinitis and eczema are increasing worldwide and the increase of peanut allergy may simply be a reflection of this (Hourihane *et al.* 1996a, Ewan 1996)

Increased peanut allergy may also be attributed to increased consumption of peanut. It is well documented in the literature that the feeding habits of a country, ethnic and local differences dictate the types of food sensitivity. For example in Spain, egg is the most common food allergen and in Scandinavian countries, fish is the most common food allergen. (Reference). The average American consumes approximately 5kg of peanut products per year. Consequently, peanuts are a common food for young children, especially given the popularity of peanut butter as a finger food for young infants (Ewan 1996, Sampson 1996).

Thus, whether the apparent increase is a result of increased peanut consumption in the population or simply a reflection of the general increases in prevalence of atopic disease is not clear, but it is likely to be a combination of both (Hourihane *et al.* 1996, Sampson 1996). With this rising number of individuals at risk for potentially fatal reactions, aggressive intervention in both prevention and treatment is warranted (Sampson 1996).

Peanut sensitisation is reported to be more common in highly atopic infants, irrespective

of peanut exposure. The actual age at which children become sensitised is not known, but it is thought to be decreasing, possibly as a result of increased consumption of peanuts by nursing mothers and by infants and toddlers. A study by Hourihane *et al.* (1996) investigated peanut allergy in a cohort of 622 subjects with reported peanut allergy. They found that 80% of subjects had a reaction on first exposure to peanut. This result and findings of other studies suggest that sensitisation is occurring either in utero, via breast milk and in formula feeds or in foods consumed in early childhood.

In a study by Ewan (1996), a strong association was found with atopy and peanut allergy. The results suggest that peanut and nut allergy is occurring in a sub-population with a strong propensity to develop allergies. This high-risk group could be identified in early childhood. It would seem reasonable to avoid these allergens, particularly peanut and peanut butter during the period when sensitisation is most common, possibly to the age of 5-7 years (Ewan 1996).

These recommendations have not been universally accepted. Contrary to the findings outlined above, Lack & Golding (1996) believe there is limited evidence to suggest that avoiding foods during lactation or early childhood prevents allergic sensitisation to these foods and that avoiding peanut until age seven is extraordinary and unnecessary. Furthermore they have suggested that exposure to peanuts and other food allergens during lactation and childhood may be important in the development of immunological tolerance and may prevent allergic sensitisation to these foods.

## ALLERGY TO OTHER NUTS

It has been estimated that up to up to 50% of self selected and referred subjects with peanut allergy were also sensitised to other nuts (Hourihane *et al.* 1996). According to Ewan (1996) patients allergic to peanuts should also be considered as developing allergy to other tree nuts such as almond, hazelnut, pecan, Brazil, cashew and walnut. In a study of 62 consecutive peanut allergic patients, approximately one third were also allergic to other tree nuts.

## ALLERGY TO SESAME

It has been reported that there is an increase in infantile eczema and anaphylaxis to food associated with sensitisation to sesame seed. Sporik and Hill (1996) reviewed the database of the main referral allergy unit at a paediatric hospital in Melbourne. The number of children sensitised to sesame was higher than the number sensitised to any one tree nut. A large number of children with peanut sensitisation were also sensitised to sesame. In 60% of the cases, sensitisation to sesame occurred before the age of two years. While there is limited data on the true incidence of sesame sensitisation, severe reactions have been reported and it may be more common than previously thought (Sporik and Hill 1996).

## THE NATURAL HISTORY OF FOOD ALLERGY

The natural history of food allergy has long been the subject of anecdotes about children “outgrowing” their allergy, however a lack of evidence and prospective systematic studies that document these opinions has made it difficult to give clear cut guidelines for management and prevention (Bock 1981, Ewan 1996). It is generally believed that most, but not all children will grow out of their clinical reactions after a few years but unfortunately some children retain their sensitivity to foods throughout their life.

Children have a higher incidence of food allergy than adults reflecting the early sensitisation to food allergens (Esteban 1992). Most longitudinal studies of the natural history of food allergy have concentrated on egg and milk allergy in very young children. Knowledge of the natural history of peanut allergy is limited due to limited studies in the area (Bock & Atkins 1989, Ewan 1996). Allergies to peanuts, nuts and shellfish tend to continue to be lifelong and are usually more severe than allergic responses to other foods. This is recognised by the number of adults in the population with food allergy to peanut and very few with food allergy to egg and cow's milk (Bock & Atkins 1989, Ewan 1996 & Gold, Sussman, Loubser and Binkley 1997).

According to Bock & Sampson (1994), sensitisation rates do not fall equally with age for

all allergens. Chandra, Gill & Kumari (1993) investigated the natural history of food allergy in 480 children with suspected cow's milk allergy. Over five years the presence of positive oral challenge declined from 49.2% to 4% for egg, 38.8% to 1.3% for cows milk, and from 25% to 14.2% for peanut.

Furthermore it is suggested that children who are diagnosed with food sensitivity at an older age tend not outgrow their problem. In a study by Bock (1982), skin prick Tests were conducted on subjects over a period of several years and sensitisation was found to persist, even in those subjects who could consume the sensitising food without symptoms (Bock 1982).

#### SEVERE FOOD REACTIONS AND ANAPHYLAXIS

It has been suggested that the frequency of fatal and near fatal food-induced anaphylaxis has risen over the past several years (Sampson, Mendelson and Rosen 1992). A further aim of this study is to investigate the incidence and associated symptoms of food-induced anaphylaxis in children with eczema.

Anaphylaxis refers to a systemic allergic reaction affecting multiple body systems. Clinical signs usually occur within minutes of ingesting the food. Itching of lips and palate and sensation of the throat tightening is followed by urticaria and angioedema, stomach cramps and diarrhoea, vomiting, shortness of breath, wheeze, drop in blood pressure, and shock (Moneret-Vautrin 1994, Sampson *et al.* 1992). These reactions can be life threatening and in the event of accidental exposure to food allergen adrenaline is

required immediately.

In non-allergic individuals, peanut is considered a healthy, wholesome source of protein and energy, however for the allergic child, peanut can be the cause of severe symptoms. Peanut allergy is thought to be the leading cause life threatening food-induced anaphylaxis. For this reason, the increase in peanut allergy is the most worrisome food allergy and warrants further investigation (Sampson 1990, Schwartz 1992).

Some exquisitely allergic individuals have reported reactions to minute amounts of peanut, some by contact with a person who has recently ingested peanuts or being in the vicinity of a peanut, even severe reactions to inhalation of cooking fumes have been reported. For these highly sensitive individuals complete avoidance is almost impossible without being socially restricting (Dawe & Ferguson 1996, Gold *et al.* 1997, Sampson 1992, Scadding & Brostoff 1988, Yunginger 1988, 1992).

A study by Yunginger (1992) described seven cases of fatal anaphylaxis to food allergy in adults over a period of 16 months. Factors believed to have lead to fatal outcome included the patient's denial of symptoms, and reliance of oral antihistamines alone for treatment. Fatalities most often occurred away from home and were associated with delayed or non-use of adrenaline (Gold *et al.* 1997).

Similar findings were reported by Assem, Gelder, Spiro, Baderman & Armstrong (1990) when they investigated severe anaphylaxis induced by peanuts in four patients. All were aware of their allergies but could not avoid the allergen and the food was ingested away from home. The accidental ingestion of peanut antigen in a disguised form was described in a patient who died after eating icing that contained almond and another who had near fatal anaphylaxis after thinking she was eating a beef-burger that was actually a vegetarian burger containing peanut. (Donovan & Peters 1990).

Sampson *et al.* (1992) investigated severe food-induced anaphylactic reactions in children and adults aged among 2-16 years. In the total six fatal reactions, peanut was the leading cause followed by other nuts and egg. All patients were considered highly atopic with current asthma, rhinitis and eczema. None of the patients were aware that the allergen was in the food they ate. A study by Bock & Atkins (1989) found that 50% of children with a history of peanut allergy had accidentally ingested some form of peanut a year before the survey. Most parents did not appreciate the potential severity of allergic reactions to foods. Adrenaline had been prescribed for three of the six children with fatal reactions, but was not available at the time of their reaction.

Avoidance of allergens is essential, and this requires constant care and attention and appropriate education of parents and carers as well as secondary carers where the risk of accidental exposure may be increased (Gold *et al.* 1997). While many parents are fully aware of their child's peanut allergy, they are often accidentally exposed to peanut, and

many parents and children live in fear of having further reactions (Bock & Atkins 1989, Steinman 1996). There is limited literature reporting fatal and near-fatal food-induced anaphylactic reactions therefore it is difficult to estimate the prevalence of such reactions.

## **AIMS**

The purpose of this study is to investigate adverse food reactions in children with eczema with emphasis on food allergy. A parallel study by Narelle Greenlees using the same eczema population will focus on food intolerance. Where food allergy and intolerance coexist in the same individual they will be included in both results.

For the purpose of this study we aim to investigate the natural history of peanut allergy. The often severe reactions associated with accidental exposure to peanut, make it a worthwhile area to investigate further. Prognostic information about the clinical course of food sensitivity is of great use for allergy specialists and dietitians so they can give parents and carers accurate information about avoidance treatment in case of accidental exposure.

### ***Specific aims are to investigate the;***

1. Symptoms associated with eczema and food sensitisation
2. Family history of allergy
3. Feeding practices of children with eczema and food sensitisation
4. Prevalence of food sensitisation (IgE-positive skin prick test to one or more food allergens) in children with eczema

5. The natural history of food and aero-allergy (especially peanut)
  - Age of sensitisation to common food and aero-allergens
6. Clinical food reactions
  - Frequency and nature of severe food allergic reactions
  - Anaphylaxis in children with eczema

## **METHODS**

### **ETHICAL APPROVAL**

Ethical approval was obtained from the Ethics Review committee of the Central Area Health service. An ethics application was also submitted to the University of Wollongong.

### **RECRUITMENT**

A cohort of 418 children (0-12 years) patients both male and female seen by Dr. Velencia Soutter (Paediatrician) Allergy Consulting Rooms, Royal Prince Alfred Hospital (RPAH) presenting with eczema and possible food related symptoms were included in the study. The subjects were selected from a total cohort of 1200 patients seen at the Allergy Clinic seen between 1995 and 1997. Figure 1 illustrates the methodology in a flow diagram.

### **DATA COLLECTION (Part one)**

Preliminary information was gathered from the case history and dietetic notes of children reviewed by Dr Soutter between 1995 and 1997. This information was recorded on data information sheets used as a summary of information and children were divided into four

groups;

1. Children with eczema & food sensitisation
2. Children with eczema and food intolerance
3. Children with both
4. Children with no reported food reactions

For the purpose of this study attention will be paid to group one, children with food sensitisation and eczema. A parallel study by Narelle Greenlees will focus on the other groups. Where data is relevant to both studies combined results will be included.

#### THE QUESTIONNAIRE (Part 2)

Parents and carers of children with eczema were sent a package inviting them to participate in the study containing :

1. Information letter outlining the aims and procedures of the study  
(Appendix 1)
2. Food Allergy and Intolerance Questionnaire (Appendix 2)
3. Connor's Rating Scale questionnaire for children > 3 years\*
4. (Appendix 3)
5. Postage Paid envelope for return of questionnaire if they decided to participate.

\*Connor's Rating Scale for parents was not analysed in this particular study, however other research in the same population investigating behaviour and adverse food reactions

is currently taking place.

### FOLLOW UP STUDY (PART 3)

Once all questionnaires had been sent out with information about the study, parents and carers of children with a positive SPT to peanut (n=139), who had not been seen in the past 6 months were telephoned. They were invited to bring their child/children into the RPAH Allergy Consulting Rooms for a follow up SPT to detect food sensitisation to peanut and/or other food and inhalant allergens.

#### **Skin prick tests (SPT)**

Follow up SPT were performed on 29 children to detect specific IgE antibodies. Each child was tested with the standard children's set including egg white, cow's milk, fish, peanut, wheat, soy, sesame and sunflower, aero-allergens including dust mite, cat dander, rye grass pollen, alternaria and aspergillus (common moulds). Peanut was routinely tested and in some but not all children other nuts were tested including Brazil, hazelnut, walnut, cashew, pecan and almond. Histamine was used as the positive control and saline as the negative control. Additional allergens, particularly other foods were added depending on the child's history. The wheal diameter was recorded at 10-15 minutes. A mean wheal diameter at least 3mm after 10-15 minutes was taken as a positive result.

However reactions less than 3mm were also measured and recorded. The initial SPT results of all children and family members were obtained from medical notes and entered into a data base.

### **Follow up consult**

Parents and Carers were given the opportunity to discuss any problems or concerns regarding their child's allergies with Dr Velencia Soutter (Paediatrician). In the case of severely peanut allergic children attention was paid to educating parents about appropriate emergency procedures in case of accidental ingestion of peanut and discussion of foods which may contain hidden peanut.

The results of the follow up SPT were discussed and compared with previous SPT results. Parents were given a summary of their child's SPT results and given the opportunity to discuss the results further with specialist dietitians Dr. Anne Swain and Dorothy Callender. If necessary, appropriate recommendations were made by the specialist dietitian on elimination of offending allergens while maintaining good nutritional status.

Consultation with both Dr Soutter and the dietitian were free of charge. Travel reimbursement was available if necessary. This was particularly important for those travelling large distances to participate in this study.

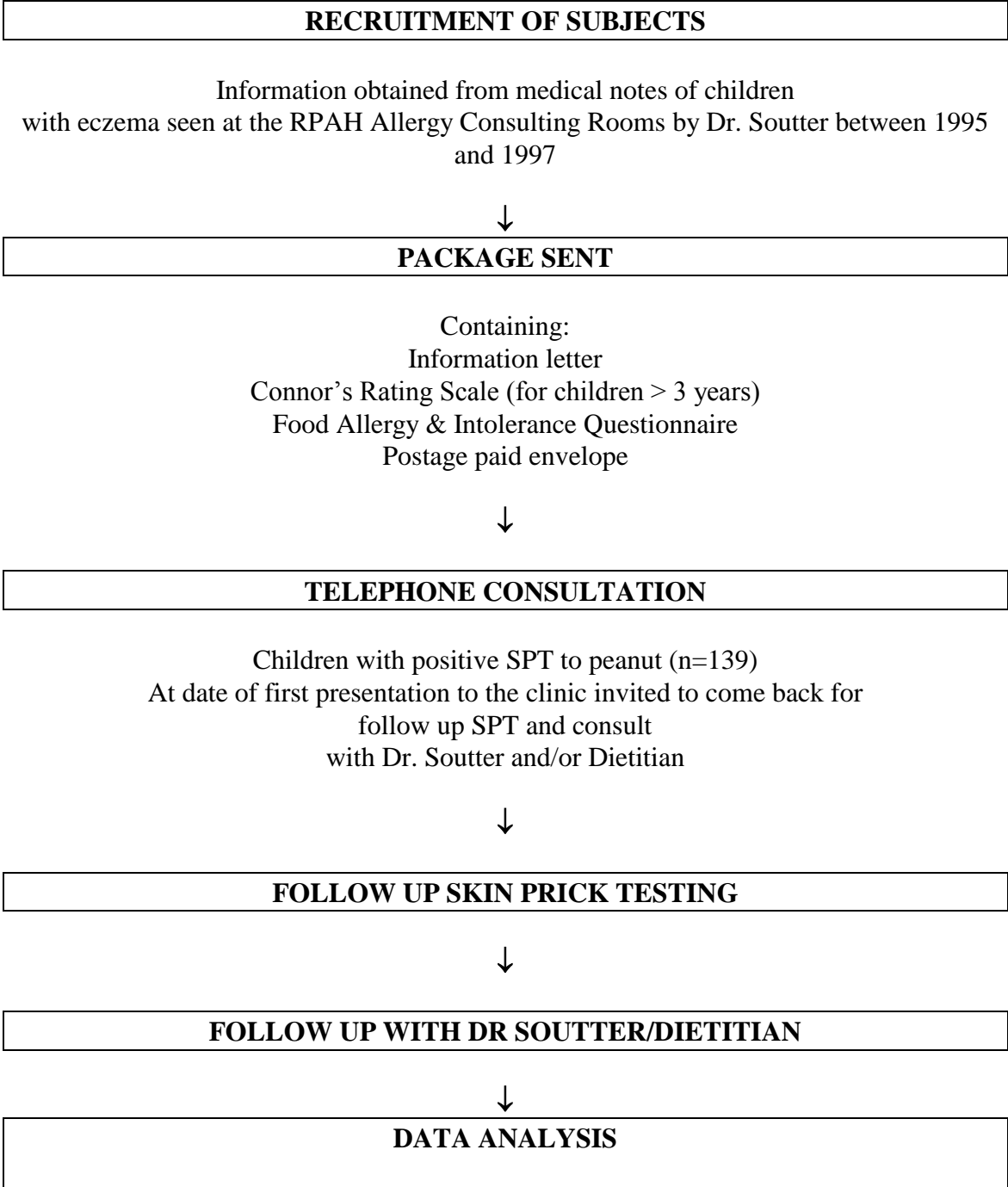
### **Follow up Response**

Parents and carers were telephoned;

- 33 agreed to children had follow up SPT during September & October, four were booked in but did not turn up or cancelled (n=29)
- 38 were lost to contact either not home on two or more occasions, or moved and not able to be contacted.
- 30 already had follow up SPT at clinic in last 6 months and were also included in study
- 38 were not contacted due to time restraints of the study.

### **STATISTICAL ANALYSIS**

Descriptive statistics including mean, standard deviation, and range were obtained using Word For Windows, Excel 6.0 statistical package.



*Figure 1: Flow Diagram of Study Methodology*

## RESULTS

### *SUBJECTS*

#### Gender

In the total sample population of 418 children with eczema, there were more males 58.4% (N= 244) than females 41.6% (N= 174).

#### Age of Presentation to Allergy Clinic

*Table 1: Age of presentation to the RPAH allergy Unit for children with eczema*

Age in Months	Total (418)	Male (n=244)	Female (n=174)
0-6	24	9	15
6.1-12	58	34	24
12.1-18	49	26	23
18.1-24	32	19	13
24.1-36	46	22	24
36.1-60	87	55	32
> 60	122	79	43

#### Age Of Onset of Eczema

The age of onset of eczema was documented in medical notes of 69 children. The mean age of onset in months was 8.35 (SD 11.10, range 1-72 months or 0.08-6 years).

#### Feeding Practises

Seventy five percent of mothers who had recorded feeding practises had breastfed their

child. The average age of weaning was 8 months (Range 1 week to .....)

***SYMPTOMS ASSOCIATED WITH ECZEMA***

At presentation to the clinic, the child’s symptoms were recorded in medical notes. These were entered into the database for analysis. All children in the sample population had eczema or a history of eczema. A total of 42% of children with eczema also had rhinitis and 37% had asthma/wheeze. Anaphylaxis was reported in 23 children. A summary of associated symptoms is presented in Table 2.

***Table 2: Symptoms associated with eczema in total study population***

Total Symptoms	Children n= 418	% of Total Population
<b>Eczema</b>	418	100
<b>Rhinitis</b>	174	42
<b>Asthma/wheeze</b>	153	37
<b>GIT (loose stools)</b>	81	19
<b>Urticaria</b>	68	16
<b>Behaviour Irritability</b>	41	10
<b>Sleep Disturbance</b>	36	9
<b>Angioedema</b>	28	7
<b>Anaphylaxis</b>	23	6



### ***FAMILY HISTORY OF ALLERGY***

In the total eczema population (n=418) 130 mothers and 79 fathers had positive SPT to one or more allergens. In the food sensitisation group (n=202), 91 mothers and 52 fathers had a positive SPT to one or more allergens.

### ***FOOD SENSITISATION (IgE)***

In 418 children with eczema, 202 children (48%) were sensitised to one or more food allergens. Forty per cent (n=166) had a significant positive SPT which was greater than or equal to 3mm in wheal diameter. This is presented in table 3.

***Table 3: Food Sensitisation and Eczema***

<b>Total Eczema Population</b>	<b>Food Sensitisation to one or more food allergens</b>	<b>% Of Total</b>
n= 418	n=202	48
	n=166*	40*

\* Total number of children with a significant SPT result  $\geq$  3mm wheal diameter

### ***ASSOCIATED SYMPTOMS IN CHILDREN WITH FOOD SENSITISATION***

Other symptoms diagnosed by Paediatrician Dr. Velencia Soutter of children presenting to the clinic with eczema and food sensitisation were recorded in the medical notes. The most common symptoms in 202 children with food sensitisation were rhinitis (40%) and asthma (40%) presented in Table 4.

***Table 4: Symptoms Associated with Food Sensitisation***

<b>Total Symptoms</b>	<b>Children n= 202</b>	<b>% of total food sensitised population</b>
<b>Eczema</b>	202	100
<b>Rhinitis</b>	81	40
<b>Asthma/wheeze</b>	80	40
<b>Urticaria</b>	39	19
<b>GIT (loose stools)</b>	30	15
<b>Anaphylaxis</b>	22	11
<b>Angioedema</b>	21	10
<b>Sleep Disturbance</b>	17	8
<b>Behaviour Irritability</b>	14	7

***RESPONSE TO ALLERGEN AVOIDENCE***

Question 13 in the questionnaire asked parents and carers if their child’s symptoms had improved since coming to the clinic. After presentation these children avoided allergens which involved eliminating all of those foods which the child had a significant SPT result to or a known clinical reaction to that food when ingesting it. Twenty one responses to this question are presented in Table 10.

***Table 5: Symptoms after allergen avoidance since presenting to the clinic.***

<b>Improvement on Allergy</b>	<b>Number of Children</b>	<b>% of Total</b>
<b>Avoidance Diet</b>	<b>(n = 21)</b>	
Same	3	14
A little better	2	10
Much better	12	57
Completely well	4	19

## ***FOOD ALLERGENS***

The most common food allergens in 202 children with food sensitisation are presented in Table 5. The most common food allergens were egg and peanut. Thirty five percent of the total eczema population were sensitised to egg, 33% to peanut and 23% to cows milk. These three food allergens contributed to 62% of all positive SPT to foods. Significant food sensitisation to sesame was more common than soy.

***Table 5 : Most common food allergens in 202 children with food sensitisation***

<b>ECZEMA- 418 children</b>		
<b>Food</b>	<b>All +ve SPT (≥ 1mm wheal)</b>	<b>+ve SPT (sig) (≥ 3mm wheal)</b>
<b>Egg</b>	145 (35%)	122 (29%)
<b>Peanut</b>	139 (33%)	107 (26%)
<b>Cows Milk</b>	98 (23%)	57 (14%)
<b>Fish</b>	61 (15%)	29 (7%)
<b>Wheat</b>	54 (13%)	31 (7%)
<b>Soy</b>	50 (12%)	20 (5%)
<b>Sesame</b>	47 (11%)	27 (6%)
<b>Sunflower</b>	22 (5%)	9 (2%)

*Sensitisation to other tree nuts*

Those children who were suspected of having reactions to other nuts or by parents request were tested for sensitisation to other nuts including almond, cashew, hazelnut, Brazil, pecan and walnut. A total of 32 children were sensitised to other nuts, many of those to more than one nut. Almond was the most common nut followed by cashew and hazelnut (Table 7)

***Table 7: Sensitisation to other tree nuts***

<b>Other Tree Nuts</b>	<b>Number of Children sensitised (n=32)</b>
Almond	18
Cashew	12
Hazelnut	12
Brazil	7
Pecan	4
Walnut	4
<b>Total</b>	<b>57</b>

### ***SIZE OF SPT RESULT***

The mean was calculated for all positive SPT results (sensitisation) and for SPT results that had a diameter greater than or equal to 3mm (significant). The average size of the wheal diameter was largest for the peanut allergen in all SPT.

***Table 8: Mean Size of SPT for 8 common food allergens***

<b>Food Allergen</b>	<b>Mean Wheal Size of SPT</b>	
	<b>= to &gt; 3mm in diameter (significant)</b>	<b>= to &gt; 1mm (sensitised)</b>
Peanut	5.90 (SD 3.34) range 3-17	4.87 (SD* 3.43) range 1-17
Egg	5.49 (SD 2.65 ) 3-18	4.84 (SD 2.8) 1-18
Milk	5.07 (SD 2.26) range 3-10	3.69 (SD 2.32) range 1-10
Wheat	4.77 (SD 2.9) range 3-14	3.22 (SD 2.40) range 1-14
Sesame	4.41 (SD 2.09) range 3-11	3.15 (SD 2.10) range 1- 11

Fish	3.93 (SD 2.8) range 3-14	2.82 (SD 1.91) range 1- 14
Sunflower	4.33 (SD1.65) range 3-8	2.68 (SD1.61) range 1-8
Soy	3.75 (SD 1.37) range 3-8	2.54 (SD 1.35) range 1- 8

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### ***FOOD SENSITISATION VS CLINICAL REACTIONS***

From information collected in the case files and dietetic notes it was possible to estimate how many children with a positive SPT had a reaction to food when it was ingested and the clinical symptoms associated with it.

Careful history of food reactions was recorded and analysed and it was found that in the total 139 children sensitised to peanut, 44 had actually reacted when ingesting or coming into contact with peanut (32%). The most common allergic reaction was angioedema (n=10, 23%) followed by anaphylaxis (n=6, 14%), other rash (n=3, 7%), and other (urticaria, eczema and vomiting, n=3, 7%). In 22 of these reactions (50%) the symptoms associated with reaction were unknown or not recorded. One child had a history of anaphylaxis to peanut with a SPT result of only 1mm.

**NATURAL HISTORY OF ALLERGY**

***Table 9: Age of sensitisation to food and aero-allergens in children with eczema.***

ECZEMA – 418 children								
<b>AGE (Months)</b>	<b>&lt;6</b>	<b>6-12</b>	<b>12-18</b>	<b>18-24</b>	<b>24-36</b>	<b>36-60</b>	<b>60-84</b>	<b>&gt;84</b>
<b>Number of children</b>	<b>n= 10</b>	<b>n= 47</b>	<b>n= 32</b>	<b>n=24</b>	<b>n=34</b>	<b>n= 54</b>	<b>n=32</b>	<b>n=38</b>
<b>Allergen</b>								
<b>Egg</b>	90%	77%	66%	50%	53%	52%	28%	24%
<b>Peanut</b>	60%	72%	56%	54%	47%	39%	44%	39%
<b>Milk</b>	60%	47%	41%	50%	50%	20%	31%	13%
<b>Soy</b>	40%	13%	16%	13%	24%	11%	25%	16%
<b>Fish</b>	20%	15%	9%	21%	32%	22%	31%	11%
<b>Wheat</b>	10%	28%	16%	13%	26%	20%	22%	13%
<b>D p</b>	10%	36%	69%	75%	79%	87%	88%	79%
<b>Cat</b>	0	21%	38%	29%	32%	56%	63%	55%
<b>Rye</b>	0	2%	9%	13%	18%	37%	56%	53%

As illustrated in Table 5, food sensitisation decreases and dust mite sensitisation increases

as children get older. Peanut is the most common food allergen in children aged 5 years or more. Egg sensitisation in the < six month group is present in 90% whereas in the 5-7 year old group it is present in 24%. Dust mite sensitisation is most common at age 18-24 months and does not change much after this. Figure one represents age specific sensitisation rates for food allergens egg, milk and peanut in children up to seven years old. Figure 3 represents age specific sensitisation rates for aero-allergens dust mite, cat and rye grass pollen.

***SERIOUS FOOD INDUCED REACTIONS***

Question 21 in the questionnaire (see appendix 3) asked parents or carers if their child had suffered any serious reactions to foods since being seen at the clinic. For those children who had positive SPT to one or more food allergens at presentation, 24 were reported to have had serious allergic reactions to foods. A total of 55 allergic reactions were reported, and the average number of reactions for each child was calculated (mean 2.29, range 1-6). The age of reaction ranged from 1.75 to seven years. Clinical presentation of these reactions can be seen in Table 10.

***Table 10: Serious Reactions reported in 24 children since last presentation to clinic.***

<b>Food Induced Allergic Reaction</b> <b>in 24 children</b>	<b>Frequency of Symptom</b> <b>(N=55)</b>
Angioedema	14
Anaphylaxis	12

Contact reaction	10
Asthma	9
Croup	5
Vomiting/nausea	3
Urticaria	2

One child had three separate instances of angioedema as a reaction to the fumes of fish cooking, being near peanuts and also being kissed by someone who had recently eaten peanut butter caused the eyes to water and the face to swell. Seven of the 24 children were reported to have needed hospital treatment after coming into contact with the food allergen.

***FOOD INDUCED ANAPHYLAXIS***

In the total sample of eczema children, detailed information was available on 23 children with a history of food induced anaphylaxis either before or after leaving the clinic. Areas of investigation included food trigger, presence of multiple food allergy and food intolerance, size of SPT to offending food and presence and severity of asthma.

***Table 11: Associations with Anaphylaxis in 23 children***

<b>Associated Symptom</b>	<b>Number of children (n=23)</b>	<b>% of Total</b>
Multiple food Allergy*	22	96%

Food intolerance**	18	78%
Asthma	18	78%
Dust mite allergy	14	61%

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*\*positive SPT to more than one food allergen*

*\*\* food intolerance diagnosed by paediatrician/dietitian by elimination diet and challenge*

As seen in Table 11, 22 (96%) children had other food allergy apart from that which caused anaphylaxis. Eight of the 18 children with a history of asthma had severe asthma. Food intolerance was also present in 18 (78%) of children with a history of food induced anaphylaxis. Dust mite allergy was present in 14 children.

### ***FOODS CAUSING ANAPHYLAXIS***

***Table 12: Foods causing 29 instances of anaphylaxis in 23 children***

<b>Offending food</b>	<b>Number of reactions</b>	<b>% of total reactions</b>
Peanut	9	31%
Milk	8	28%

Egg	6	21%
Other nuts	5	17%
Fish	1	3%
	<b>29</b>	<b>100</b>

As illustrated in Table 12, peanut was the most common cause of anaphylaxis accounting for 31% of all reactions. One child had a history of anaphylaxis to three foods egg, peanut and milk. Another child had a history of four anaphylactic reactions to other nuts and peanuts, three of which were very severe. This makes the total number of anaphylactic reactions to foods in 23 children equal to 29. Two children were accidentally exposed to nuts at Christmas time. One child had anaphylaxis to cows milk given inadvertently while in hospital, and needed adrenaline to start breathing.

## **DISCUSSION**

### **SYMPTOMS ASSOCIATED WITH ECZEMA**

The prevalence and morbidity of allergic disease is thought to be increasing in the population (Esteban 1996, Zeiger *et al.* 1995). Furthermore, food allergy often presents in patients with established allergic disease diagnosis such as asthma, rhinitis and eczema. (Edwards 1995). It was not surprising to find that a large proportion of the children with eczema also had other symptoms at presentation to the clinic. Asthma/wheeze and rhinitis were equally as common in these children reported in 40% of children with food sensitisation. Other symptoms such as angioedema, urticaria and gastrointestinal symptoms are commonly associated with food allergic disease (Loblay & Swain 1992).

## FAMILY HISTORY

These symptoms were also present in ...% of mothers and ...% of fathers supporting the importance of family history in development of atopic disease. Percentage with allergy (positive SPT).

## FEEDING PRACTISES OF CHILDREN WITH ECZEMA

Seventy five per cent of the children in this eczema population were breastfed, for an average of eight months. Interestingly the average onset of eczema in this study population was also eight months, ranging from one month to six years. It is likely that in some of these children, onset of eczema is occurring at a time when the infant diet is expanding and new foods are introduced into the diet. In some children who had earlier onset of eczema, before weaning, it may be suggested that maternal diet during lactation contributed to the eczema, although the extent is unknown. Exposure to aero allergens may also be contributing to eczema in infancy and the age of onset of eczema may also be related to increased exposure to environmental allergens.

## FOOD SENSITISATION AND ECZEMA

The actual role of food sensitisation in the aetiology of eczema is beyond the scope of this study, however results indicate that food sensitisation is common in children with eczema. The prevalence of food allergy in paediatric patients with eczema is estimated between 33 and 62% (Sampson & McCaskill 1985, Sampson 1991, Burks *et al.* 1988). In

this study, 48% of children were sensitised to one or more food allergens, 40% with a significant SPT result.

The cornerstone of management of food allergy after a correct diagnosis is made is the avoidance of identified food allergens. Sampson & McCaskill (1992) found a significant improvement in skin symptoms after food allergens were eliminated from the diet in children with eczema. In this study, symptoms improved in 18/21 children, reported by parents and carers who returned the questionnaire. Fifty seven per cent said their child was much better and symptoms had significantly improved since avoiding allergens and 19% children were completely well as a result. The finding may suggest that the elimination of these allergens in the diet has led to a significant improvement in symptoms, likely eczema, however the low response rate to this question makes it difficult to determine the real impact allergen avoidance on improving eczema. To determine whether food allergy causes or exacerbates eczema can only be resolved if suspected or offending foods are challenged in DBPCFC.

### **Food Allergens**

It is well known that egg, peanut and milk are the most common food allergens in children (Schwartz 1992, Sampson & Metcalfe 1992). In this study population a large proportion of the children with eczema had a positive SPT to egg (35%) and peanut (33%) and milk (23%). Other common food allergens include fish, wheat, sesame and soy.

Studies suggest the increases of peanut consumption in the western world has lead to an increase in the prevalence of peanut allergy ( Hourihane *et al.* 1996, Tariq *et al.* 1996 ) Others suggest that this may be just a reflection of the overall worldwide increases in atopic disease (Ewan 1996, Sampson 1996). Controversy exists around the actual cause of this increase. The findings of this study suggest that peanut sensitisation is very common as one in four children with eczema had a significant SPT to peanut.

### **Sensitisation to other nuts**

Children with peanut allergy are at risk of developing allergy to other tree nuts and they should be considered in the management and prevention of food allergy (Ewan 1996, Hourihane *et al.* 1996). It is difficult to determine the actual prevalence of sensitisation to other nuts in this study group the standard battery of foods tested does not contain other nuts and these are only tested for if requested by parents or paediatrician suspects allergy to other nuts from a careful history. Thirty two children had positive SPT to other nuts, many children sensitised to more than one type of nut. The most common nut allergens other than peanut, were almond, cashew and hazelnut. Hazelnut spread and cashew nut paste are readily available and considered an alternative to other nuts and an enjoyable spread for children, maybe even used as a weaning food. Children with a history of peanut allergy should also be tested for other nuts before recommending these foods. There is also potential for very sensitive peanut allergic children to be accidentally exposed to peanuts as they are sometimes disguised as more expensive nuts. Further study with DBPCFC would provide the best estimate of the true prevalence of allergy to other nuts in these children.

## **Sesame sensitisation**

Sesame sensitisation, while not as common as peanut sensitisation, may be increasing and can be severe in some children (Sporik & Hill 1996). In nearly all studies, sesame is never mentioned as a food that children are allergic to. In accordance with the findings of Sporik & Hill (1996), the number of children sensitised to sesame was higher than the number sensitised to any one tree nut (excluding peanut). The number of children in this study sensitised to sesame was also very similar to the number sensitised to fish and wheat, yet it is rarely mentioned as a common food allergen. Interestingly, there were more children with a significant SPT result to sesame than to soy. Soy is often associated with food allergy in the literature and the perception of many parents and carers in the community.

The feeding habits of a country reflect the types of food individuals are allergic to. The diverse marketplace and the increased availability of a variety of different ethnic foods in recent times in Australia, such as Middle eastern Tahini and Homous and sesame oil used in Asian cooking, may contribute to increased consumption of sesame. The sometimes severe nature of sesame allergy makes it worthwhile investigating and may be suggested that all children suspected of a food allergy, especially those with peanut allergy should also be tested for sesame. In highly allergic families maternal avoidance of sesame, similar to peanut may be indicated. Further study is warranted before any definite recommendations can be made.

## NATURAL HISTORY OF ALLERGY

### **Age specific food sensitisation rates**

There is a trend for food sensitisation decreases and aero sensitisation increases as children get older. It also appears that sensitivity to all food allergens is much less common in older children (over the age of five) than in younger children. According to Bock & Sampson (1994), sensitisation rates do not fall at equal rates for all food allergens. Peanut allergy does not appear to follow the same trend as egg and milk in these children. As seen in Figure 1, there appears to be a slower decrease in peanut sensitisation as children get older. In children aged 7 years and over, peanut is the most common food allergen (39%) much higher than the percentage of children sensitised to egg or milk. Peanut sensitisation is present in 60% of children tested at age six months or less and decreases to 39% in children aged over seven years of age. In comparison, sensitisation to egg in the six month or less group was present in 90% and falls to 24% and sensitisation for milk falls from 60% to 13% respectively in children over seven years of age.

It is interesting that in children less than six months old, six were sensitised to peanut and sensitisation was most likely transferred via breast milk, as it is unlikely a child of that age would have already eaten peanuts, unless peanut butter was given as a weaning food. There may be other means of food sensitisation, possibly by mouth contact with parents after eating peanut butter and kissing a child, or by cross contamination of utensils which

may have come in contact with the offending food. It is difficult to make any definite conclusions, these are only suggestions and warrant further study.

### **Aero-allergen sensitisation**

It can be suggested that aero-allergen sensitisation becomes more common than food allergen sensitisation as children get older. Eighty eight per cent of children tested aged between 5-7 years were sensitised to dust mite and only 28% to egg. There tends to be a large increase in dust mite sensitisation from six months to 2 years and then it tends to plateau and there is no significant increase from 2 years onwards. It may be suggested that if a child is not sensitised to dust mite by age three, it is unlikely they will be. Cat and rye grass pollen sensitisation tends to increase at a later age than dust mite and peaks around 5 years old.

One of the limitations is that the actual age of sensitisation is not known. The results are recorded from the actual time of first SPT at presentation. This can only estimate the actual age when the child became sensitised to the food or aero-allergen. It would be impossible to determine the actual age of sensitisation. A longitudinal study which investigated sensitisation to food and aero-allergens in the same children followed up at regular time periods from birth to the age of seven years would give a more accurate indication of age specific sensitisation rates for foods and aeroallergens.

A dual approach to prevention of allergy in children is suggested by Hide et al 1996. Avoiding as much as possible food and aero-allergens in breastfeeding and infancy may

help to reduce the risk of allergic disease. Eczema is likely to be irritated by both food and aero-allergens and management needs to consider both factors. Our results indicate that sensitisation to food and aero-allergens is common in children with eczema. Especially in those children with asthma, eczema and food allergy who are risk of anaphylaxis, it may be beneficial to minimise household levels of dust mite and exposure to other aero-allergens such as cats in infancy to reduce risk of sensitisation.

### **Peanut Sensitisation**

The natural history of peanut sensitisation was investigated in a group of children who were sensitised to peanut at first presentation. These children were followed up 6 months or longer after their first SPT and retested. The average time between first and second test was 20 months and ranged from 6 months to ..... years. No significant change over time was found. This may suggest that many children with peanut sensitisation may not grow out of it.

Limitations may be the length of time between first and second test was not enough to show any significant change and maybe these children need to be tested 3 years after first test for a true indication of the nature of peanut allergy. Another limitation is that oral challenges to peanut were not performed for two reasons, time limitations and lack of adequate supervision. It is likely that parents of highly allergic children are not going to be willing to challenge their child with a peanut if there is a history of serious reactions. For this reason it is also difficult to estimate if those children sensitised to peanut can now tolerate the food. They may have a positive SPT but not actually react when peanut

is ingested. Although some children may not have obvious reactions to peanut when they ingest, small amounts of the peanut may aggravate eczema. It is very difficult, due to the delayed onset of eczema in many cases to determine a direct causal link. It can be suggested that peanut allergy may persist as children get older and that many children do not outgrow their sensitivity to peanut. Further longitudinal study is warranted.

Time restraints meant that although 139 children were sensitised to peanut, time allowed only 53 of those to come back for follow up. It was apparent that of those parents and carers contacted, almost all of them were very keen to bring their child in for a follow up SPT and dietetic consult. Many of these children require constant care and attention and it seems that many parents of these children are highly motivated, and want to keep up to date with their child's progress. If time allowed, a larger sample size would have strengthened the results.

#### CLINICAL REACTIONS TO FOODS

The most obvious limitation in this study is the lack of DBPCFC. It is difficult to determine the true prevalence of true food allergy in the sample population, as oral challenges to foods were not performed. Oral food challenges need to be undertaken with adequate medical supervision especially in highly allergic children with a history of severe food reactions. Challenges to confirm peanut allergy in children with a positive result to SPT were not taken so we cannot comment on the sensitivity of such testing for peanut. Many parents would be apprehensive about exposing their child to food allergen and the symptoms it may provoke and therefore not be involved in the study.

It is estimated that 50% of patients with positive SPT will have a positive reaction when challenged with that food (Cafarelli et al. 1995). While no oral challenges were performed, careful history of food reactions and confirmation by SPT indicates similar findings to those of other researchers. In the total number of children with significant peanut and egg sensitisation, 41% had a clinical history of food reactions to egg and 39% to the peanut, the most common symptom being angioedema for both foods. The clinical history of food reactions was based on reports from parents and carers. It may have been difficult for them to attribute the reaction to a specific food therefore SPT are used to confirm their suspected food allergy. If these children were challenged with suspected foods in a double blind challenge procedure, more accurate results would be obtained.

### **Severe food reactions**

Symptoms associated with food allergy include angioedema, asthma, and in extreme cases, anaphylaxis. From the questionnaire sent to parents and carers it was found that 24 children had reported serious food reactions since leaving the clinic, many of those having serious reactions on more than one occasion. Angioedema and anaphylaxis were the two most common serious reactions reported by parents.

These findings suggest that no matter how careful patients are, absolute avoidance can be difficult to achieve which is reflected in the results where 24 children had multiple serious reactions to foods since being seen at the allergy clinic. It seems that a group of

highly sensitive children are having multiple reactions to foods. These reactions may be attributed to hidden allergens, and inadequate or no labelling of foods (Steinman 1996), ignorance in the general public by relatives, carers, caterers who fail to confirm nut free products. Another reason for the number of children having reactions could be that people may not be aware of the severity of the allergy and it is only until the child actually has a reaction that they realise. It may only be after a severe reactions that parents become strict with allergen avoidance. Appropriate education of parents and carers as well as secondary carers, where the risk of accidental exposure may be increased, is essential (Gold *et al.* 1997).

Another limitation of this study is the low response rate to the questionnaire also makes it difficult to determine the true prevalence of serious food reactions. If more questionnaires were returned the actual incidence of severe food reactions may be higher than reported. On the contrary, the parents of children with severe food allergy may be a more motivated group who are regularly seeking advice and follow up and therefore more inclined to return the questionnaire. If this is the case the actual incidence of severe food reactions reported may be a good indication of true prevalence in the sample.

### **Anaphylaxis**

In extreme cases, contact with food can lead to anaphylaxis. The incidence of food induced anaphylaxis is thought to have increased in recent years (Sampson *et al.* 1992).

Eleven per cent of children with eczema and food sensitisation had a history of

anaphylaxis. These reactions are occurring in a very sensitive group of individuals nearly always with asthma, eczema and multiple food allergy seem to be at risk for anaphylaxis reactions. The most common foods causing anaphylaxis in 29 reported cases were peanut (31%), milk (28%), egg(21%), other nuts (17%) and fish (3%). These foods are potentially more of a concern than other food allergens such as wheat and soy.

This result suggests that it may be worthwhile to avoid peanut butter and peanut or other nuts during pregnancy and lactation in mothers with a family history of atopy. The suitability of peanut butter as a weaning food may also be disputed in children with eczema. Peanut butter should not be introduced into the diet of these children until at least five years, if possible longer, especially in those children with asthma and other atopic disorders that may put them at increased risk of having severe food reactions.

A very interesting finding results strongly suggest that food intolerance may play a significant role in severe food reactions. Children with both a food allergy and intolerance may be at increased risk of anaphylaxis and therefore dietary management of both is indicated. This an area which needs further investigation.

The incidence of food sensitisation and severe food reactions is reported in a very select sample population. Care should be taken when extrapolating the prevalence of allergy to normal population. This is a specific group of children with a known atopic disease and therefore at higher risk of food allergy. Care needs to be taken in interpreting these results as over-diagnosis of food allergy can result in people eliminating foods from their child's

diet based on the assumption they have a food allergy.

Children with allergy need individual treatment as each child will have a different level of sensitivity. The management of allergy and the appropriate diet for the allergic child should be discussed with a specialist dietitian and regular follow up is recommended. This ensures the child is obtaining adequate nutrition at various important stages in the growth cycle. Regular follow up is recommended so parents know alternative foods to give their child, and whether or not they have outgrown their allergy.

Adrenaline is the only drug that should be used in an emergency and parents and carers should be given an “auto-injector” and taught how to use it. It should be kept in locations that are easily accessible to parents and carers (Gold *et al.* 1997). According to Sampson *et al.* 1992 all children should be kept under medical supervision for three to four hours after anaphylaxis reaction has occurred.

Parents and carers of allergic children should be given advice on “hidden” allergens in foods and label reading, when they go out and how to avoid accidents in social situations when the children are away from home and at increased risk of exposure. The use of oral antihistamines before at risk occasions such as parties is indicated to dampen the severity of the reaction if there is accidental exposure. Other secondary carers including family members, grandparents, daycare and school personnel should be aware these children need special attention (Yunginger 1992). It is often only after an accident occurs they realise the severity of the allergy. Meanwhile the child has had to unnecessarily had to suffer due

to ignorance of others and failure to appreciate the potential severity of the allergy.

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