

## **Food allergy and Eczema – how are they related and implications for management.**

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Up to 80% of patients with eczema are sensitized to one or more of the common food or inhalant allergens. The role of allergy in eczema however remains contentious. Central to the debate is whether allergic sensitization actually causes eczema or whether this sensitization is as a secondary consequence of a damaged skin barrier and not integral to the development of eczematous lesions. Those doctors supportive of the role of allergy in eczema will investigate and recommend dietary and inhalant avoidance whilst many respected practitioners will do the opposite and explain that allergy testing is meaningless and allergen avoidance unhelpful. This confusion has led to the widespread use of alternative medicine as patients seek answers to their questions with up to two thirds of patients attending a paediatric allergy clinic in central London having undergone assessment by an alternative practitioner. In a survey of children with eczema in the UK, 75% had removed foods from their diet usually without the support of a medical practitioner or dietitian. This practice raises concerns as the risk of calcium and vitamin deficiency is increased in children placed on diets during periods of rapid growth without adequate nutritional support.

### **Skin barrier and eczema:**

Recent research has shown that damage to the skin barrier is a primary event and occurs before allergic sensitization in early infancy. The number and risk of allergic sensitizations (positive allergy tests) increases with increased skin barrier disruption and worsening eczema with those young children with widespread inflammation and itch being at greatest risk. This therefore supports the view that skin barrier disruption is the primary event and allergic sensitization a secondary event.

Further research has shown that damage to the skin barrier is under genetic regulation with a number of genes being identified for eczema. Of these genes, a gene called filaggrin (filament aggregating gene), has emerged as the most important gene determining the onset of eczema in up to one half of patients with moderate to severe eczema. Filaggrin is an important hydrating protein within the outer layers of the skin allowing it to retain water and produce natural moisturizing factor. When absent, the skin dehydrates with increased water loss from its surface. The skin barrier breaks down and becomes "leaky" allowing for the penetration of allergens and microbes. Patients carrying the filaggrin gene have abnormal markings on the palms of their hands called "palmar hyperlinearity". These patients are at increased risk for more persistent eczema and are more likely to be allergic to food and inhalant allergens. The risk of peanut allergy is increased as is the risk of asthma.

Thus a genetic defect associated with eczema has also been shown to increase the risk of food allergy and asthma. This observed progression of symptoms from early onset eczema and food allergy to the later onset of asthma and/or hayfever is often referred to as "the allergic march".

### **Food allergy and eczema:**

The association of food allergy with eczema is well established although often still questioned. Studies in multiple centers over the past 20 years have shown irrefutably the existence of food allergy in one third of children with moderately severe eczema and two thirds with severe disease. Food allergens have been shown to cause immediate reactions in patients with eczema including skin rashes

but also more severe symptoms such as breathing difficulties. In approximately half of these patients the initial immediate reaction is followed by eczema at usual eczema sites over the next 2-3 days. The immediate component of this reaction may be absent in those with truly delayed reactions or missed in patients with established skin inflammation, where the initial flare or skin rash may not be obvious.

The risk of food allergy is greatest in those infants who develop troublesome eczema in the first few months of life, which persists despite the frequent application of emollients and topical steroids. This holds true even for exclusively breast fed infants, where the risk of being food allergic may range from 30-60% in those infants with widespread active eczema. The risk of being food allergic is further increased if gut symptoms (ie reflux, frequent loose stools, constipation, bloody stools or poor weight gain) are present. In children with mild intermittent eczema that is treatment responsive, food allergy is much less likely.

The most frequent foods to cause allergic reactions are cows milk, eggs and nuts. These foods, together with wheat, soya, sesame, fish, shellfish, kiwi and pulses account for 90% of all food allergic reactions in the UK. Allergy to milk, egg, wheat and soya occur more frequently in younger children whilst allergy to nuts, sesame, fish and shellfish are found more frequently in older children although can occur at any age.

Foods often mistaken for being allergenic include acidic foods ie oranges, satsumas, lemons or foods high in natural histamines ie tomatoes and strawberries. These foods commonly provoke skin rashes at the site of contact eg around the mouth leading to irritation and a secondary flare in eczema. These reactions are usually transient and disappear, as the child grows older.

Not all food allergic reactions are immediate in nature. Certain foods (milk, soya, wheat, eggs) can trigger delayed reactions in the skin and gut, causing symptoms within 6-72 hours of ingestion. These reactions can be difficult to detect and rely on recognising certain symptom patterns. The risk of delayed reactions to foods greatly decreases after the age of 5 years.

In children with eczema and food allergy, the correct identification and avoidance of food allergens can have a significant impact on their eczema and quality of life. Underscoring the importance of allergy, the NICE guideline for eczema published in 2007 suggests that all infants < 6months of age with moderate to severe eczema should be offered a trial of a hypoallergenic formula to establish whether milk allergy could be triggering their eczema. In breast feeding mothers a trial of dietary avoidance of milk and egg is recommended whilst supplementing the maternal diet with calcium. [guidance.nice.org.uk/CG57](http://guidance.nice.org.uk/CG57)

Dietary avoidance is most effective when instituted early, before a child develops chronic thickened skin inflammation. It is however never too late to institute dietary antigen avoidance in children particularly in those with associated gut symptoms. The avoidance of foods in children should always be carried out under the supervision of a registered paediatric dietitian. It is never a substitute for good skin care and regular standard treatments of eczema are given together with antigen avoidance. Effective dietary avoidance has the benefit of allowing for reduction in topical steroids and skin barrier repair in some but not all children.

### **Allergy testing and Eczema**

The difficulties arise when interpreting allergy tests as these are often falsely positive or negative. It is possible to be food allergic and test negative on allergy testing or food tolerant and yet test positive. Crucial to the evaluation of food allergy in patients with eczema is a thorough understanding of food allergy and the performance of allergy tests. This usually requires evaluation by an allergy specialist.

A new national quality standard for eczema Sept 2013 has recommended that all infants and children with moderate or severe eczema < 5 years are referred for specialist allergy testing and evaluation. [publications.nice.org.uk/atopic-eczema-in-children-qs44](http://publications.nice.org.uk/atopic-eczema-in-children-qs44). It is hoped that this quality standard will raise awareness amongst practitioners regarding the importance of early identification and management of food and inhalant allergies in children with established persistent eczema.

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