MED439
KING SAUD UNIVERSITY

pediatric
439 Teamwork

Revised by The Academic Leader
Homoud Algadheb

Common pediatric allergies

objectives:

- Epidemiology of common pediatric allergic diseases
- Clinical presentation of allergies e.g. allergic rhinitis, atopic eczema, sinusitis, food allergies
- Age-specific presentations of allergies in infants and children
- Genetic background and environment as risk factor in developing allergies
- Mediators produced by inflammatory cells and their role in manifestations of clinical signs / symptoms of allergies
- IgE mediated allergic conditions
- Role of IgG in allergy
- Cell mediated allergic condition
- Early and late phase allergic response
- Basis of allergic response and role of inflammatory mediators e.g. leukotriene in treatment of allergic conditions
- Role of skin prick test and RAST in the diagnosis of allergy
- Co-existence of allergic rhinitis and asthma
- Common food allergies
- Clinical presentation of food allergy and food intolerance
- Pathophysiology of Type I and Type II food allergy.

Done by: Bader alrayes

Revised by: Saud Alrsheed

Team Leader: Saud Alrsheed

Special thanks to team 437 & Faisal alsaif

Notes

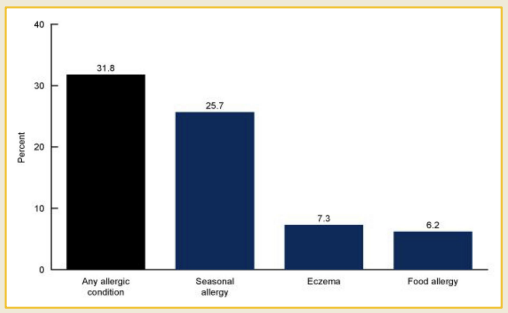
Important

Book

Common Pediatric Allergies

Prevalence

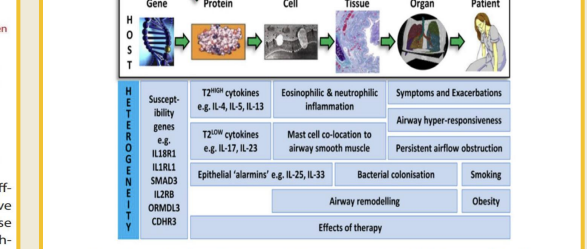
Note:
Adults were considered to have any allergic condition if they were diagnosed with one or more of three selected conditions: seasonal allergy, eczema, or food allergy. Categories for each allergic condition were not mutually exclusive. Estimates are based on household interviews of a sample of the U.S. civilian noninstitutionalized population.



Prevalence of atopy and asthma in primary school children in australia:

- Asthma diagnosed 31.0%
- Hay Fever (aka Allergic rhinitis) 38.4%
- Eczema 24.8%

- ❖ Allergic Disease is the 5th leading chronic disease among all ages (when we talk about allergies, we mean allergic manifestations in general, whether they were: Eczema, Food allergy, Angioedema, Urticaria, asthma, Sinusitis, Allergic conjunctivitis and so on)
- ❖ 3rd common chronic disease among children under 18 years old; up to one child in three is affected
- ❖ Trends indicate that by 2025, half of all europeans may be suffering from an allergy
- ❖ 20-30% of total Indian population suffer from on or other allergic condition
- ❖ Prevalence of Asthma, Rhinitis and Eczema in Saudi Arabia: (graph)
- ❖ Asthma Prevalence among saudi children <5 years of age was 24 %



Middle Bar indicates physicians' diagnosed asthma
Red Bar indicates highly suspected asthma

The Hygiene Hypothesis

- It's an observational hypothesis not an interventional one.
- Th-2 is an indication of allergy, while Th-1 is non-allergic, there's a balance between the two, and exposure to microbial products early in life (e.g. children who live in farms) will make the balance in favor of Th-1 and this helps the immune system shift to the non-allergic immune response
- Probiotics and Prebiotics are also factors that switch the immune system towards the non-allergic response.
  - Studies show that early introduction of probiotics and prebiotics will prevent the immune system from switching to an allergic response
  - an example of this is that علب الحليب الجاهز اللي تعطى للأطفال Contains probiotics and prebiotics, among other things

The hygiene hypothesis suggests that early exposure to microbial products will switch off allergic responses and thereby prevent allergic disease. Certainly epidemiological studies comparing populations who have or have not had such exposure support the hypothesis strongly in relation to seasonal rhinoconjunctivitis but perhaps rather less clearly in relation to asthma. The hypothesis becomes even more compelling when studying children who were born and lived on farms. In such circumstances, frequent exposure to the stables during pregnancy and postnatally with or without drinking farm milk (presumably unpasteurized) resulted in dramatic decreases in prevalence rates for asthma, rhinitis, and allergic sensitization. To what extent this might or might not be a consequence of exposure to endotoxin remains to be established by intervention studies.

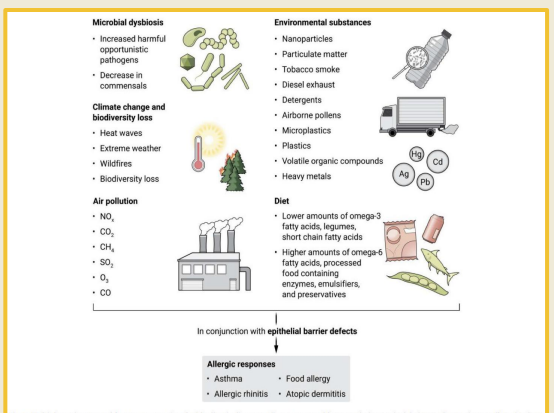


Fig. 3. Multiple environmental factors are associated with allergic diseases. Allergy-associated factors include microbial dysbiosis, climate change effects, biodiversity loss, dietary changes, and increased exposure to pollutants. Some of the dietary choices indicated in the figure, such as foods rich in legumes, short-chain fatty acids, and omega-3 fatty acids, promote health and may prevent allergic disease. The effects of these factors are exacerbated by epithelial barrier defects, which drive asthma, allergic rhinitis, atopic dermatitis, and food allergy.

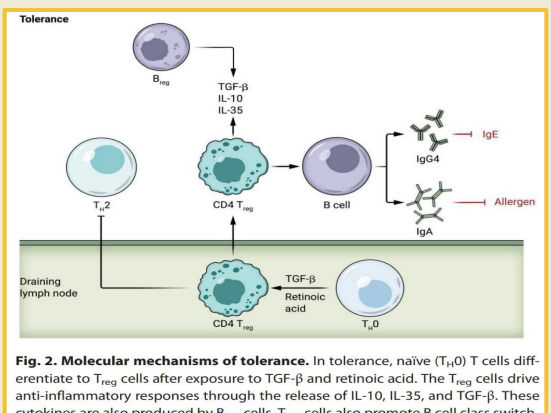
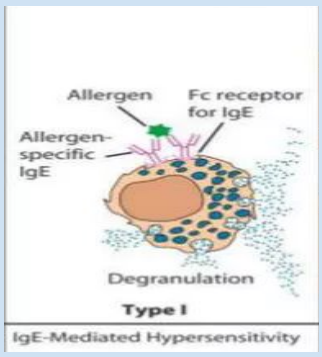
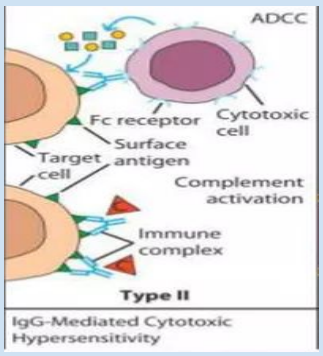
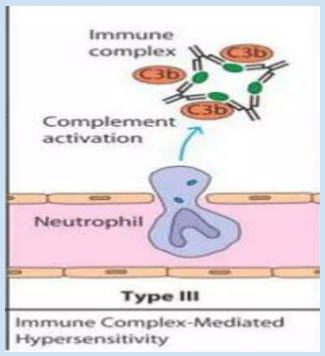
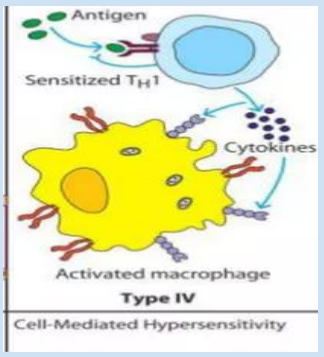


Fig. 2. Molecular mechanisms of tolerance. In tolerance, naïve (T_H0) T cells differentiate to T_{reg} cells after exposure to TGF-β and retinoic acid. The T_{reg} cells drive anti-inflammatory responses through the release of IL-10, IL-35, and TGF-β. These cytokines are also produced by B_{reg} cells. T_{reg} cells also promote B cell class switching to IgA and IgG4, both of which promote tolerance by binding allergens or blocking IgE activation.

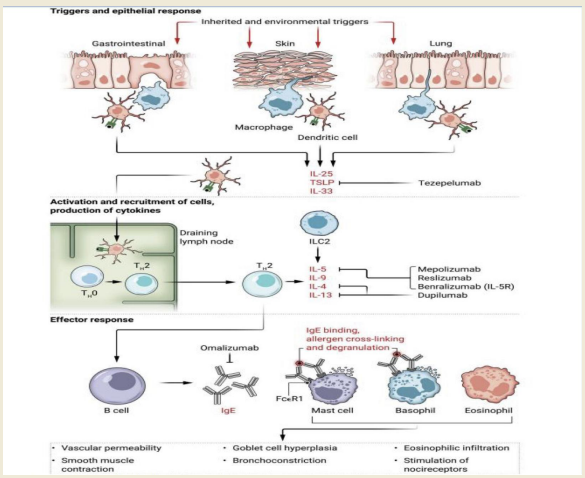
Figure 1. Scales of disease of asthma with examples of disease heterogeneity at all levels from gene through to patient.
Scales of disease of asthma with examples of disease heterogeneity at all levels from gene through to patient.

Hypersensitivity

Gell and Coombs Classification of hypersensitivities:

			
Type I	Type II	Type III	Type IV
IgE Mediated	IgG/IgM Mediated	IgG Mediated With immigration of inflammatory cells from circulation into tissue	T-cell
Classic reaction starts with: Allergen⟶ IgE⟶ Fc receptor	RBC lysis	Immune complex disease	Delayed type Hypersensitivity

❖ **Figure: Molecular mechanisms of IgE-mediated allergic disease:** Epithelial defects enhance entry of antigen, infectious agents, and other stressors into the lung, skin, or gastrointestinal tract. This is followed by a release of proinflammatory epithelium-derived cytokines such as IL-25, IL-33, and thymic stromal lymphopoietin (TSLP). These cytokines enable dendritic and other cells to prime naïve (T_H0) cells to become T_H2-polarized. T_H2 cells release proinflammatory cytokines, such as IL-4, IL-5, IL-9, and IL-13. The epithelium-derived cytokines also activate type 2 innate lymphoid cells (ILC2s), which also release IL-5 and IL-13. These cytokines favor B cell isotype class switching to IgE, which binds to the surface of effector cells such as mast cells and basophils through the high-affinity IgE receptor FcεR1, leading to sensitization. IL-5 favors eosinophilia in the tissues and circulation. In sensitized individuals, subsequent exposure to allergens causes cross-linking of IgE bound to FcεR1 receptors on sensitized mast cells and basophils, leading to the release of preformed and de novo-synthesized proinflammatory mediators, such as histamine, prostaglandins, leukotrienes, cytokines, and others. These lead to symptoms of bronchoconstriction, enhanced vascular permeability, smooth muscle contraction, and eosinophilic infiltration.



Allergens

What causes the body to react?

- Any protein can be recognized by the immune system as a foreign substance: starting from vaccines to plants, foods, insects, animal danders. In theory, there's nothing that cannot cause an unwanted reaction by the immune system (only water is the except- since it's not a protein)

TABLE 16-1 COMMON ALLERGENS ASSOCIATED WITH TYPE I HYPERSENSITIVITY

Proteins	*Foods*
Foreign serum	Nuts
Vaccines	Seafood
	Eggs
Plant pollens	Peas, beans
Rye grass	Milk
Ragweed	
Timothy grass	*Insect products*
Birch trees	Bee venom
	Wasp venom
Drugs	Ant venom
Penicillin	Cockroach calyx
Sulfonamides	Dust mites
Local anesthetics	
Salicylates	*Mold spores*
	Animal hair and dander

Drug reactions

Drug reactions can be ANY type of hypersensitivity

- Type 1,2,3 are usually related to oral +injectable administrations
- Type 4 mostly happens with oral administrations
- The reaction is not necessarily to the active ingredient of the drug, it could be one of the other components

TABLE 16-5 Penicillin-induced hypersensitive reactions

	Type of reaction	Antibody or lymphocytes induced	Clinical manifestations
Humoral and systemic	I	IgE	Urticaria, systemic anaphylaxis
	II	IgM, IgG	Hemolytic anemia
	III	IgG	Serum sickness, glomerulonephritis
(Cellular) and Local	IV	T_{DTH} cells	Contact dermatitis

Hypersensitivity

Fc ε receptors (FcεR)

FC = Fragment crystallizable

FcεR1:

- high affinity IgE receptor found on Mast cells, basophils, and activated eosinophils
- Allergen Binding to IgE attached to FcεR1 triggers release of granules from cell.

FcεR1 triggers release of mediators:

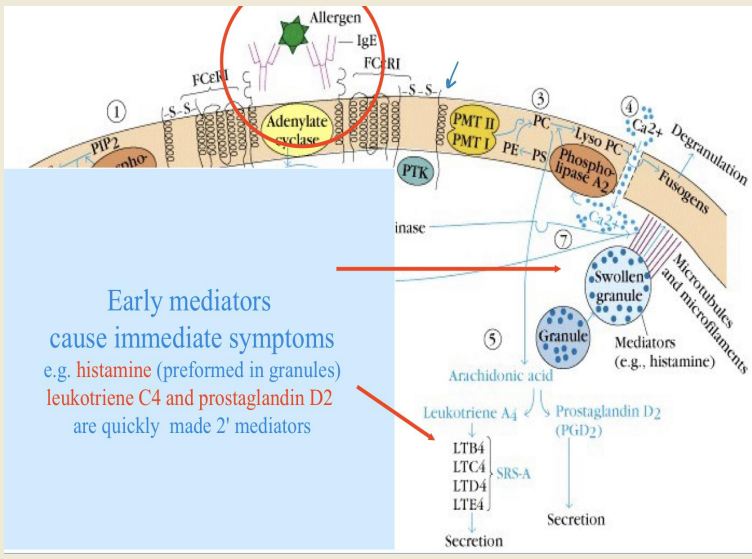
When IgE binds to the allergen on the second exposure, there will be a massive release of inflammatory mediators (imp term in allergy; they're involved in the dx, lab tests, & treatment as some medications are pinpointed against certain mediators)

- Leukotrienes are one of the inflammatory mediators & are studied in detail to produce medications that antagonize them & block them. Common medications include: Singulair, Montair

In the future, molecular biology will evolve to let you know what are the proteins that cause this allergic reaction. let's take egg allergy as an ex:

- In the past, we used to say that there is an allergy from the yellow part of the egg, & an allergy from the white part.
- Now, we know that the yellow part has 6 different proteins (some more important than others, some start causing the allergic reactions at earlier age than others, & some proteins cause long lasting allergy while others may have a non long-lasting course

عشان كذا احيانا بعض الأطفال اللي عندهم حساسية بيض يتحسسون من البيض المسلوق بس اللي في الكيك ما يسبب لهم حساسية لان نوع البروتين هذا يصير له denaturation إذا تجهز بهالطريقة



Mediators of Type I hypersensitivity

Immediate effects

- Histamine: (main cause of the reaction)
  - constriction of smooth muscles (bronchoconstriction = wheezing, constriction of intestine = cramps-diarrhea)
  - Vasodilation with increased fluid into tissues causing increased swelling or fluid in mucosa.
  - Activates enzymes for tissue breakdown

- leukotrienes
- Prostaglandins

Primary mediators

Pre-formed in mediators in granules

- Histamine
- Cytokines TNF-a, IL-1, IL-6
- Chemoattractants for neutrophils and eosinophils
- Enzymes
  - (tryptase, chymase, cathepsin)
  - Changes in connective tissue matrix. tissue breakdown

Mediators of Type I Hypersensitivity

Secondary mediators

Mediators formed After activation

- Leukotrienes
- Prostaglandins
- Th2 Cytokines- IL-4, IL-5, IL-13, GM-CSF

Continuation of sensitization cycle

- Mast cell controls immediate response.
- Eosinophils and neutrophils drive late or chronic response
- More IgE production further driven by activated mast cells, basophils, Eosinophils
- Eosinophils Play a key role in late phase reaction
- eosinophils make
  - enzymes
  - cytokines (IL-3, IL-5, GM-CSF)
  - Lipid mediators (LTC4, LTD4, PAF)
- Eosinophils can provide CD40L and IL-4 for B-cell activation

localized anaphylaxis

Target organ responds to direct contact with allergen. (can happen anywhere in the body: skin, GIT….)

- Digestive tract contact results in vomiting, cramping, diarrhea
- Skin sensitivity usually reddened inflamed area resulting in itching
- Airway sensitivity results in sneezing and rhinitis OR wheezing and asthma

Systemic anaphylaxis

- Systemic Vasodilation and smooth muscle contraction leading to severe bronchoconstriction, edema and shock
- Similar to systemic inflammation

Delayed Type Hypersensitivity

- What we saw previously was all IgE mediated
- Here the main players are cells (no humeral reaction)

Th1 cells and macrophages

- DTH response is from:
  - Th1 cells release cytokines to activate macrophages causing inflammation and tissue damage
  - continued macrophages activation can cause chronic inflammation resulting in tissue lesions, scarring and granuloma formation (e.g. of cell mediated reaction in all of us : BCG immunization, ends in a scar on our left arm)

- Delayed is relative because DTH response arise 24-72 hours after exposure rather than within minutes (diagnosed with Patch test, left for 24-72 hours)

Stages of Type IV DTH

Th-1 is the main player of the delayed type hypersensitivity reaction

1

Sensitization stage:

- Memory Th1 cells against DTH antigens are generated by dendritic cells during the sensitization stage
- These Th1 cells can activate macrophages and trigger inflammatory response

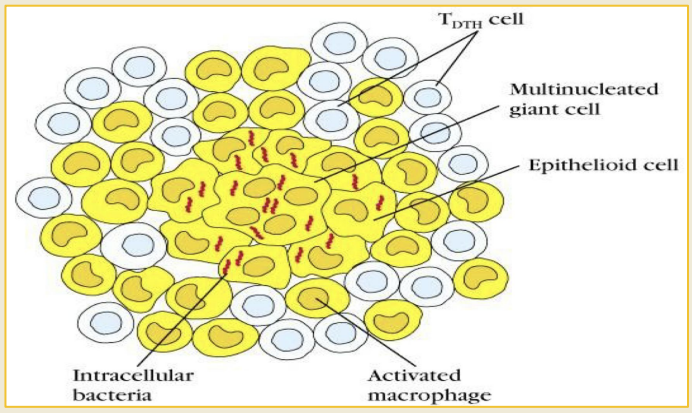
2

Effector Stage:

- Secondary contact yields what we call DTH
- Th1 memory cells are activated and produce cytokines.
  - INF-γ, TNF-α and TNF-β which cause tissue destruction and inflammation
  - IL-2 that activates T cells and CTLs
  - Chemokines for macrophages recruitment
  - IL-3, GM-CSF for increased monocyte/macrophage
- Upon secondary exposure to antigen
  - inflamed area becomes red and fluid filled, can form lesion (from tissue damage there is activation of clotting cascades and tissue repair
    - continued exposure to antigen can cause chronic inflammation and result in granuloma formation

Granuloma formation from DTH mediated by chronic inflammation

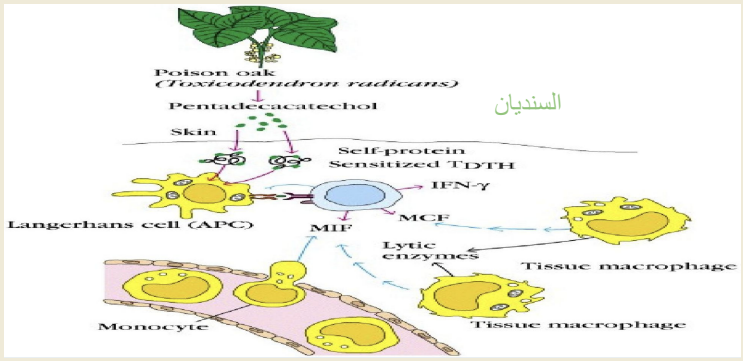
As seen in chronic eczema where they develop lichenification



Delayed Type Hypersensitivity

Contact dermatitis

- Type IV DTH
- The response to poison oak is a classic type IV
  - small molecules act as haptens and complex with skin proteins to be taken up by APCs and presented to Th1 cell to get sensitization
  - During secondary exposure Th1 memory cells become activated to cause DTH



poison oak contain an allergen that causes allergy to the workers that interact with poison oak ⟶ DTH ⟶ Granuloma formation

- ❖ DTH is a type of immune response classified by Th1 and macrophages activation that result in tissue damage.
- ❖ DTH can be the result of chronic infection or exposure to some antigens
  - ❖ Hair dye causes DTH, you will see that the box instructs the person to test the dye and then do a complete hair dye 24 hours later (ideally: you wait 72 hours).
  - ❖ Other examples of DTH include PPD test, or contact dermatitis from nickel or leather.
    - ➢ someone can wear nickel-containing product, then remove it. After a while they'll notice that the area which was in contact with nickel is red and harsh. After a while, it might even become dark and brown

TABLE 14-3 INTRACELLULAR PATHOGENS AND CONTACT ANTIGENS THAT INDUCE DELAYED-TYPE HYPERSENSITIVITY

Intracellular bacteria	Intracellular viruses
Mycobacterium tuberculosis	Herpes simplex virus
Mycobacterium leprae	Variola (smallpox)
Listeria monocytogenes	Measles virus
Brucella abortus	Contact antigens
Intracellular fungi	Picrylchloride
Pneumocystis carinii	Hair dyes
Candida albicans	Nickel salts
Histoplasma capsulatum	Poison ivy
Cryptococcus neoformans	Poison oak
Intracellular parasites	
Leishmania sp.	

Respiratory allergens: (think of them as indoor or outdoor allergens)

- Bermuda grass الحشيش النجيل (في الاستراحات و ملاعب الكورة)
- Rye
- Atriplex (Rughl) عشبة برية
- Amaranthus SP (an ornament plant) سندار, عرف الديك, قطيفة
  - Amaranthus viridis
  - celosia argentea
- Salsola spp (herm)
- Chenopodium album (etra)
- Prosopis
- Cats
- Dogs
- Cockroaches
- Dust Mites عث الخبار

Allergic Rhinitis

Health effects of Allergic Rhinitis

- Social inconvenience
- Sleep disturbances/obstruction
- Learning difficulties
- Impaired maxillary growth
- Dental Problems
- Infection: nose and sinuses
- Comorbidities: conjunctivitis, asthma, rhinosinusitis, otitis media

History

The history is the most important element in the evaluation of allergy, key features of the history are:

- Worsening of the symptoms on exposure to aeroallergens (aggravating factors)
- seasonal variation in symptoms related to pollination of trees, grasses and weeds
- A family history of atopic disease
- An environmental history assessing exposure to indoor and outdoor allergens and the presence of associated allergic condition

Family History

- Because allergic rhinitis has a significant genetic component, a positive family history for atopy makes the diagnosis more likely
- A greater risk of allergic rhinitis exists if both parents are atopic than if one parent is atopic
- However, the cause of allergic rhinitis appears to be multifactorial, and a person with no family history of allergic rhinitis can develop allergic rhinitis
- If mother or father have allergies: 50% of their children will have allergies
- If mother AND father: 70%
- if mother and father and one of the siblings: 75%

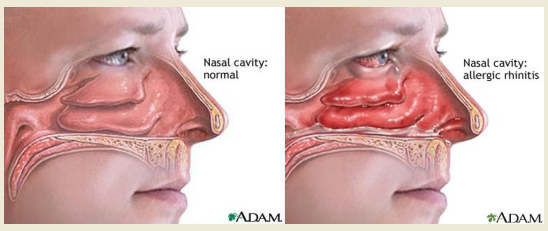
Diagnosis of allergic rhinitis

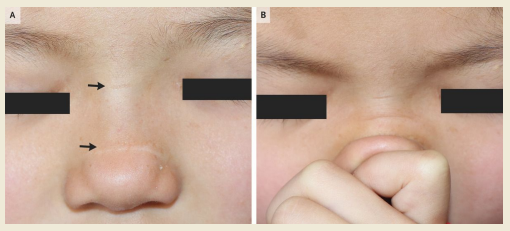
- History and symptoms of recurrent or persistent rhinitis and/or associated health effects
- Signs of atopy and recurrent or persistent rhinitis
- Demonstration of IgE allergy
- Exclusion of other causes of rhinitis

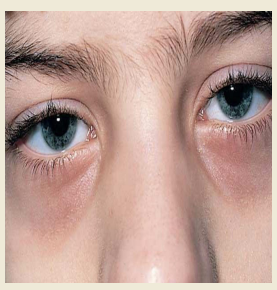
Signs and symptoms

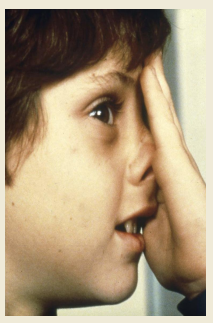
IgE mediated !!

- Rhinorrhoea
- Nasal blockage
- Postnasal drip
- Itchiness
- Sneezing
- Associated health effects
- Enlargement of nasal turbinates (may block the orifice of the nasal sinuses leading to rhinorrhea, sinusitis, headache, etc)
- Allergic salute sign and transverse nasal crease
- Associated skin manifestations (atopic dermatitis)
- Allergic shiners (هالات تحت العين)





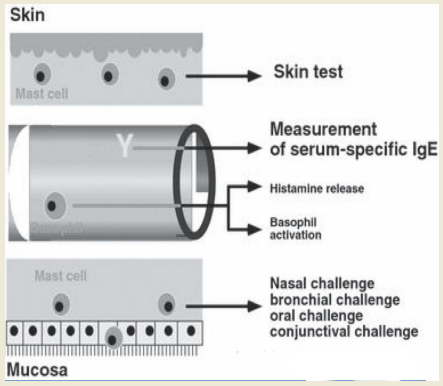




Allergic Rhinitis

Workup of patients with allergic rhinitis

- Blood eosinophils 250-400 cell/mm3 (can also be elevated in eczema and parasitic infections)
- Nasal eosinophils >15/100 cells (to insure its respiratory allergies)
- SPT (skin prick test is the golden standard to identify the allergen)
- Nasal provocation test (nasal allergen challenge)
- Rhinometry
- Total IgE (if high, you will suspect allergic rhinitis, so you have to know the type of allergen by doing **specific IgE**, [against what? grass? legumes? etc])
- RAST
- Peripheral blood eosinophilia >400/ul IS common but 50% of Pts have no eosinophilia on any one occasion
- Nasal eosinophils ⟶ Wrights stain >15/100 cells (SIGNIFICANT)



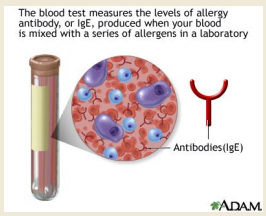
Demonstration of IgE allergy

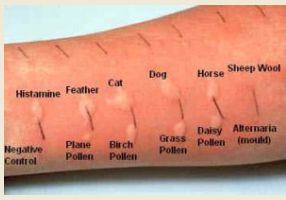
Skin prick test (SPT):

- The most important ancillary test to confirm the diagnosis of allergy is the skin test, which is the gold standard in this regard. the skin test result must be interpreted in the light of the history to determine the importance of a +ve test.
- Produces wheel (swellings) and erythema (surrounding redness)
- positive result when wheal >3mm(standard +ve result, Textbook: >4 mm) more than control
- 80% of +ve skin test give +ve RAST
- And 50% give +ve challenge
- Panel of test antigens appropriate to the locality and season and history should be used (tells you two things 1- diagnosis, 2- to plan appropriate management)

Recording and scoring of skin test results

- Skin test reactions to allergens are normally evaluated 15-20 minutes after the test have been placed, when the reactions are typically maximal. the best method to record the results of skin tests is to measure the greatest diameter of the wheal and flare in millimeters and record these results for all tests and for the positive and negative controls.
- after measurement, the result of a test can be easily recorded as, for example, 5/21, meaning that the wheal was 5 mm in greatest diameter and the flare was 21 mm in diameter. ay epicutaneous test that produces a wheal at least 3 mm larger than the wheal of the negative control with a larger surrounding flare is normally considered positive for the presence of allergen-specific IgE.





Management

1- avoid environmental exposure

2- Immunotherapy

Box 16.2 Range of treatment for allergic rhinoconjunctivitis

- Second-generation non-sedating antihistamines (used topically or systemically)
- Topical corticosteroid nasal or eye preparations (the latter under specialist ophthalmology supervision)
- Cromoglycate eye drops
- Leukotriene receptor antagonists, e.g. montelukast
- Nasal decongestants (use for no more than 7–10 days due to risk of rebound effect)
- Allergen immunotherapy – sublingual or subcutaneous (limited by anaphylaxis risk)
- Systemic corticosteroids should not be used due to the risk of adverse effects

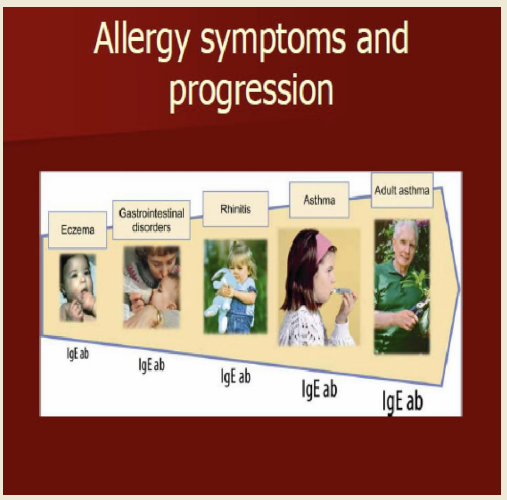
Allergic Dermatitis and Atopic eczema

- Anyone with allergic rhinitis **will** have atopic dermatitis to some degree (could be mild dryness to full eczema)
  - The drier the skin the more sensitive it is.

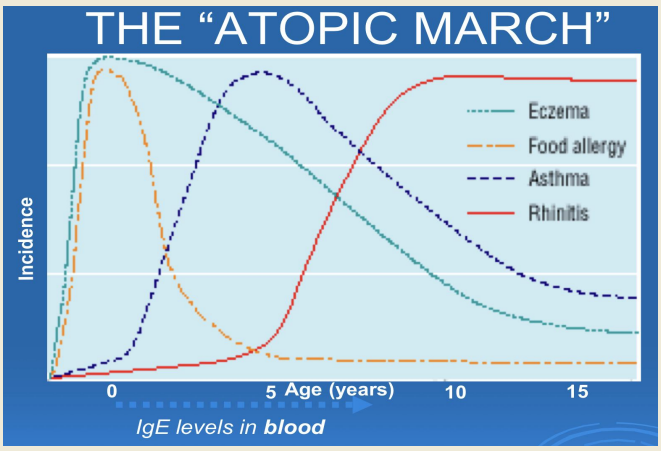
- **Filaggrin Gene** mutation have been identified as the key factor for eczema due to the impairment of skin barrier.
  - filaggrin gene mutation predispose to food allergy, hay fever, asthma and eczema

Allergic children develop individual allergic disorders at different ages:

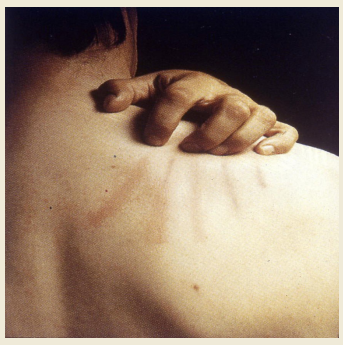
- Eczema and food allergy usually develop in infancy
- Allergic rhinitis, conjunctivitis and asthma being most often in preschool and primary school years.
- Rhinitis and conjunctivitis may precede the development of asthma
- the presence of eczema or food allergy in infancy is predictive of asthma and allergic rhinitis in later life.

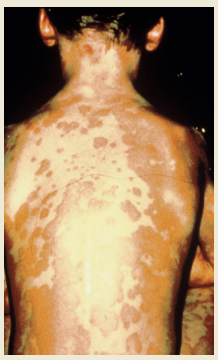


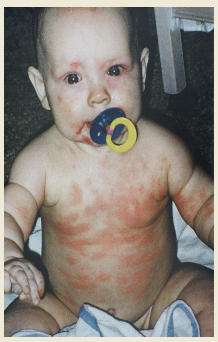
Allergic disorders often overlap. many children with food allergy have eczema, and up to 80% of children with asthma have rhinitis.

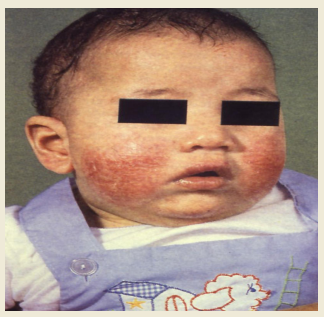


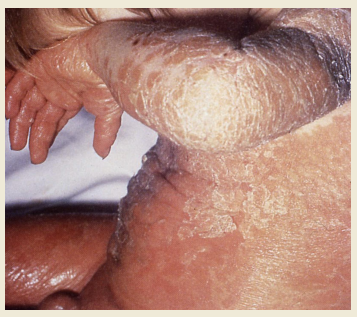
Allergic dermatitis is in most cases the first manifestation of the atopic disposition

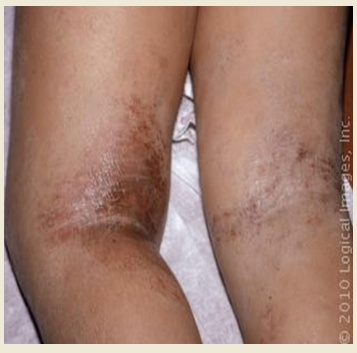


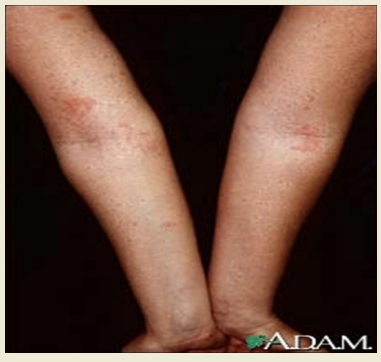


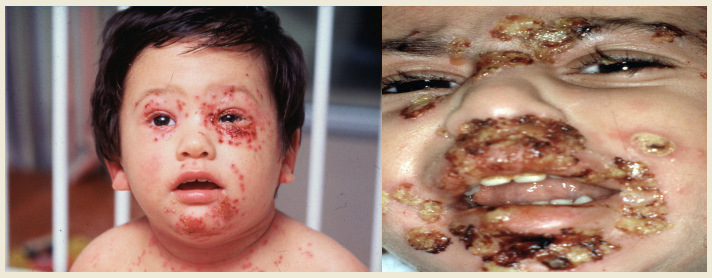








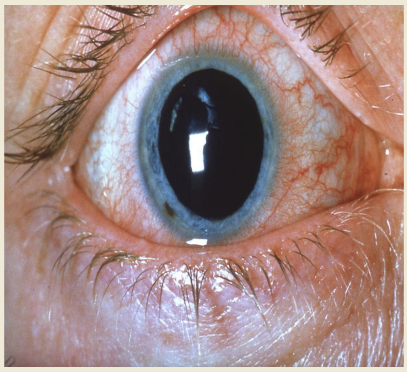


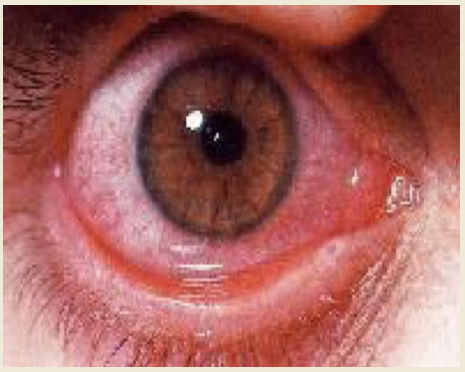


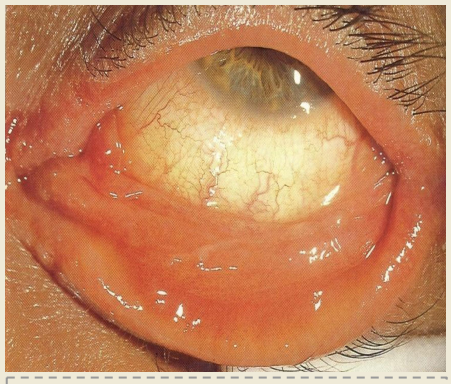
As the patient scratches, it exposes the skin to secondary bacterial (staph) or viral (herpetic) infection, thats why its better to keep the skin moist

Allergic Conjunctivitis

- Conjunctivitis is part of allergy because the conjunctivitis is also a mucous membrane(like the nose) so it also prone to it







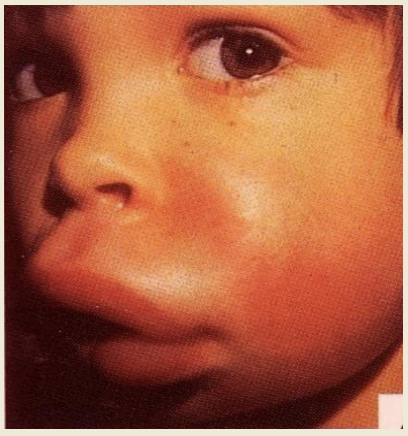
Vernal keratoconjunctivitis: pt can't see because conjunctiva encroaches to the cornea

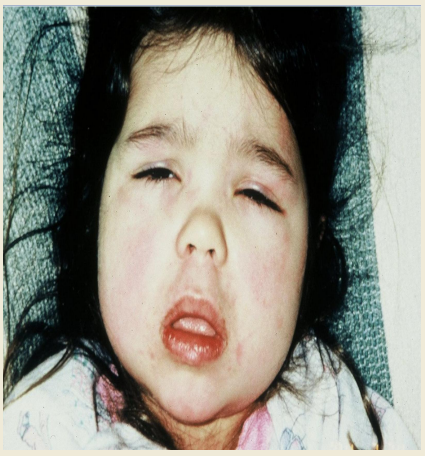
Urticaria and Angioedema

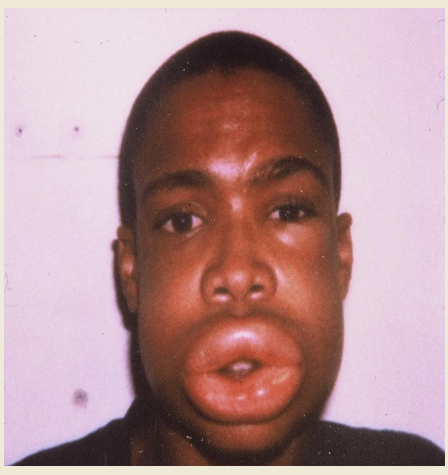
- As we said, theoretically. the site of the allergic reaction can happen anywhere (soft tissues, legs,etc) but the skin, GI and respiratory system are the most common places

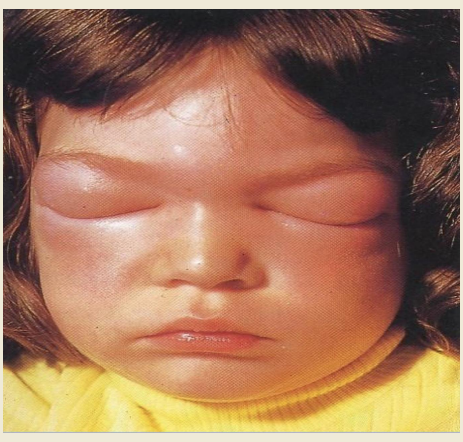
Types of Urticaria/angioedema

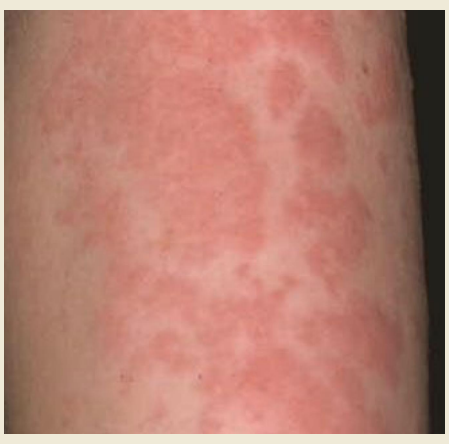
- Acute urticaria/angioedema
  - lasts less than 8 weeks
- Chronic urticaria/angioedema
  - lasts more than 8 weeks
  - idiopathic, autoimmune
  - urticarial vasculitis
- C1 inhibitor deficiency angioedema (an enzyme deficiency not related to allergies)











Food Allergy

Classical (Class 1) Food Allergens

Peanut	Ara h1, Ara h2, Ara h3	
Cow's milk	Caseins a, b, k	Bos d8
	B-Lactoglobulin	Bos d5
	A-Lactalbumin	Bos d4
	Bovine serum albumin	Bos d6
Eggs	Ovomucoid	Gal d1
	Ovalbumin	Gal d2
Shrimp	Tropomyosin	Pen a1
Codfish	Parvalbumin	Gad c1
Lipid transfer proteins		
	Apple	Mal d1, Mal d4
	Peach	Pru p1, Pru p2, Pru p3
	Hazelnut	Cor a1, Cor a2

As mentioned, the evolution of molecular biology is fast. Milk allergies used to be classified in 2 types, allergy to whey or caseins. Now, we know that there are 3 types of caseins (a, b, k) while whey (lactoglobulin a or b, each having 3 different types → 6 possible allergens in total giving different clinical presentations, age of onset and reaction to being cooked)

Adverse reactions to food

IgE-Mediated → **Non-IgE Mediated**

- Oral allergy syndrome (cross reactivity with other allergens)
- Anaphylaxis
- Urticaria

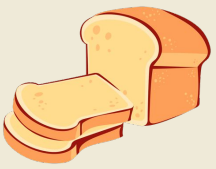
- Eosinophilic esophagitis
- Eosinophilic gastritis
- Eosinophilic gastroenteritis
- Atopic dermatitis

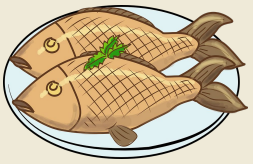
- Protein-induced enterocolitis
- Protein-induced enteropathy
- Eosinophilic proctitis
- Dermatitis herpetiformis

Incidence of allergy to specific foods

➢ **In young children: 90% caused by:**

- Milk (casein, whey)
- Egg
- Peanut
- Tree nuts
- Wheat (gluten)
- Soy
- Shellfish



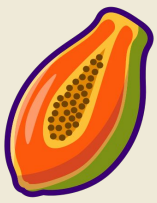


➢ **In adults: 85% caused by:**

- Peanut
- Fish
- Shellfish
- Tree nuts

➢ **Increasing incidence of allergy to "exotic foods" such as:**

- Kiwi
- Papaya
- Seeds: sesame, grape, poppy
- Grains: psyllium



Food Allergy

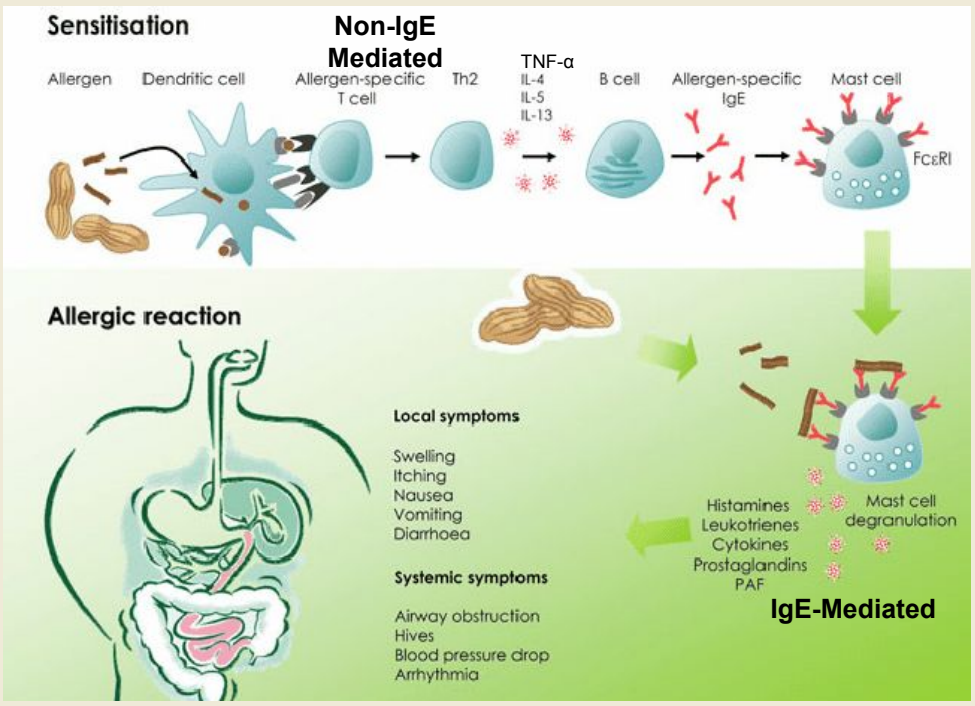
Food allergy prevalence in specific disorders

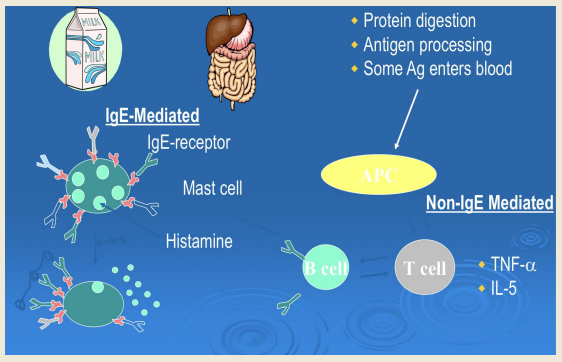
Disorder	Food allergy prevalence
Anaphylaxis	35-55%
Oral allergy syndrome	25-75% in pollen allergic
Atopic dermatitis	37% in children (rare in adults)
Urticaria	20% in acute (rare in chronic)
Asthma	5-6% in asthmatic or food allergic children
Chronic Rhinitis	Rare

Prevalence of clinical cross reactivity among food “families”

Food allergy	Prevalence of allergy to >1 food in family
Fish	30-100%
Tree nut	15-40%
Grain	25%
Legume	5%
Any	11%

Pathophysiology: immune mechanisms





Oral Allergy Syndrome (OAS)

- ❖ OAS refers to clinical symptoms in the mucosa of the mouth and throat that:
- ➢ Result from direct contact with a food allergen
- ➢ in an individual who also exhibits allergy to inhaled allergens
- ➢ Usually pollens (pollinosis) are the primary allergens
- ➢ Pollens usually trigger rhinitis or asthma in these subjects
- ➢ OAS is acquired, transient, and usually pt also has inhalant/respiratory allergies

Case scenario:
Someone eats shrimp salad and feels numbness or itching of the lips and throat, so he stops and eats something else and the symptoms disappear. this is OAS

Characteristics of OAS

- Inhaled pollen allergens sensitise tissues of the upper respiratory tract
- Tissues of the respiratory tract are adjacent to oral tissues, and the mucosa is continuous
- Sensitisation of one leads to sensitisation of the other
- First described in 1942 in patients allergic to birch pollen who experience oral symptoms when eating apple and hazelnut
- OAS symptoms are mild in contrast to primary food allergens and occur in oral tissues

Allergens

- Pollens and foods that cause OAS are usually botanically unrelated
- several types of plant proteins with specific functions have been identified as being responsible for OAS:
  - Profilins
  - Pathogenesis-related proteins
  - Lipid transfer proteins. pan-allergen (found in all animal +plant kingdom)

- Profilins are associated with reproductive functions
- Pathogenesis-related proteins tend to be expressed when the tree is under “stress” (e.g. growing in a polluted area)
- Lipid transfer proteins induce IgE antibodies. resistant to heat, gastric acid and digestive enzymes

Oral Allergy Syndrome (OAS)

Associated Foods

- Foods most frequently associated with OAS are mainly fruits, a few vegetables, and nuts
- The food cause symptoms in the oral cavity and local tissue immediately on contact:
  - Swelling
  - Throat tightening
  - Tingling
  - Itching
  - “blistering”

Cross-Reactivity

- Occurs most frequently in people allergic to certain weeds and tree pollens e.g.
  - Ragweed pollen
  - Mugwort pollen
  - Grass pollen

Cross-Reacting allergens

➢ **Birch pollen (also: mugwort, and grass pollen) with:**

- Apple
- Kiwi fruit
- Orange
- Melon
- Carrot
- Watermelon
- Potato
- Tomato
- Peanut
- Hazelnut
- Celery
- fennel
- Stone fruits (apricot, peach, nectarine, plum, cherry)

➢ **Ragweed pollen with:**

- Banana
- honeydew
- Cantaloupe
- zucchini (courgette)
- Cucumber
- Watermelon
- Other melons

Latex allergy

- Allergy to latex is thought to start as a Type IV (contact) hypersensitivity reaction
- Most urinary catheters are made from latex, so patients with latex allergy who need urinary catheter has to be careful otherwise it might lead to allergic urethritis
- Contact is with a 30 Kd protein, usually through:
  - Abraded (non-intact) skin
  - Mucus membrane
  - Exposed tissue (e.g. during surgery)

Related foods:

Foods that have been shown to contain a similar 30 Kd antigen include:

- Avocado
- Banana
- Kiwi fruit
- Fig
- Tomato
- Pineapple
- Passion fruit
- Celery
- Peanut
- Tree nuts
- Chestnut
- Citrus fruit
- Grapes
- Papaya

❖ Most kids with food allergies grow out of it as their immune system matures