Allergy review

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UOM

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Allergy

An ever increasing problem world wide

Atopy and Allergy

- Atopy : a personal/familial tendency to become sensitised and produce Ig E antibodies in response to ordinary exposure to allergens usually proteins. They MAY develop eczema , asthma or Rhinoconjunctivitis.

- Allergy is a HYPERSENSITIVITY REACTION initiated by immunological mechanisms. It can be antibody or cell mediated. The antibody is typically Ig E . IgE mediated disease is typical of asthma and allergic rhinitis. Food and drug allergy may also involve Ig G and T-cell dependent mechanisms.

Topics

- Basic mechanisms
- Food allergies
- Drug allergies
- Urticaria and Angioedema
- Natural History and prevention

What do patients present with ?

- Rashes (rougeurs,boutons , la, zébillition)
- Swellings (bosses, enflures)
- Oral irritation/burning
- Suspect specific/a variety of foods
- Rarely suspect medicines (eg ACE –I), unless immediate reactions
- Respiratory symptoms (not covered today)
- Want “ tests “ to diagnose the problem
- What is the most important diagnostic tool in allergy diagnosis ?

Diagnosis of allergy

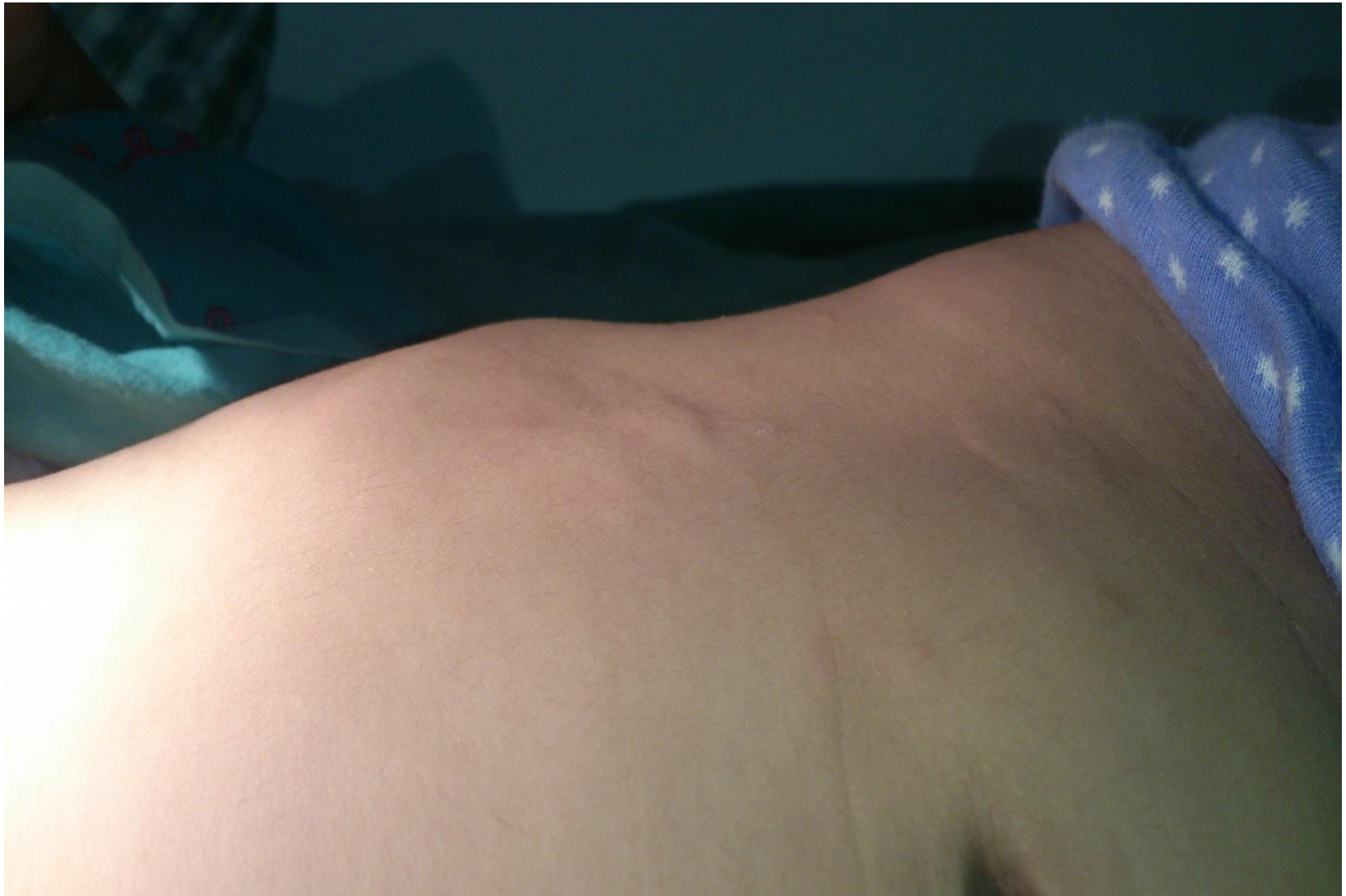
- Which of the following is the first line tool to diagnose allergy ?

1. Skin prick tests
2. Intradermal testing
3. Phadiatop (Immunocap)
4. Component resolved diagnostics (ISAC)
5. None of the above

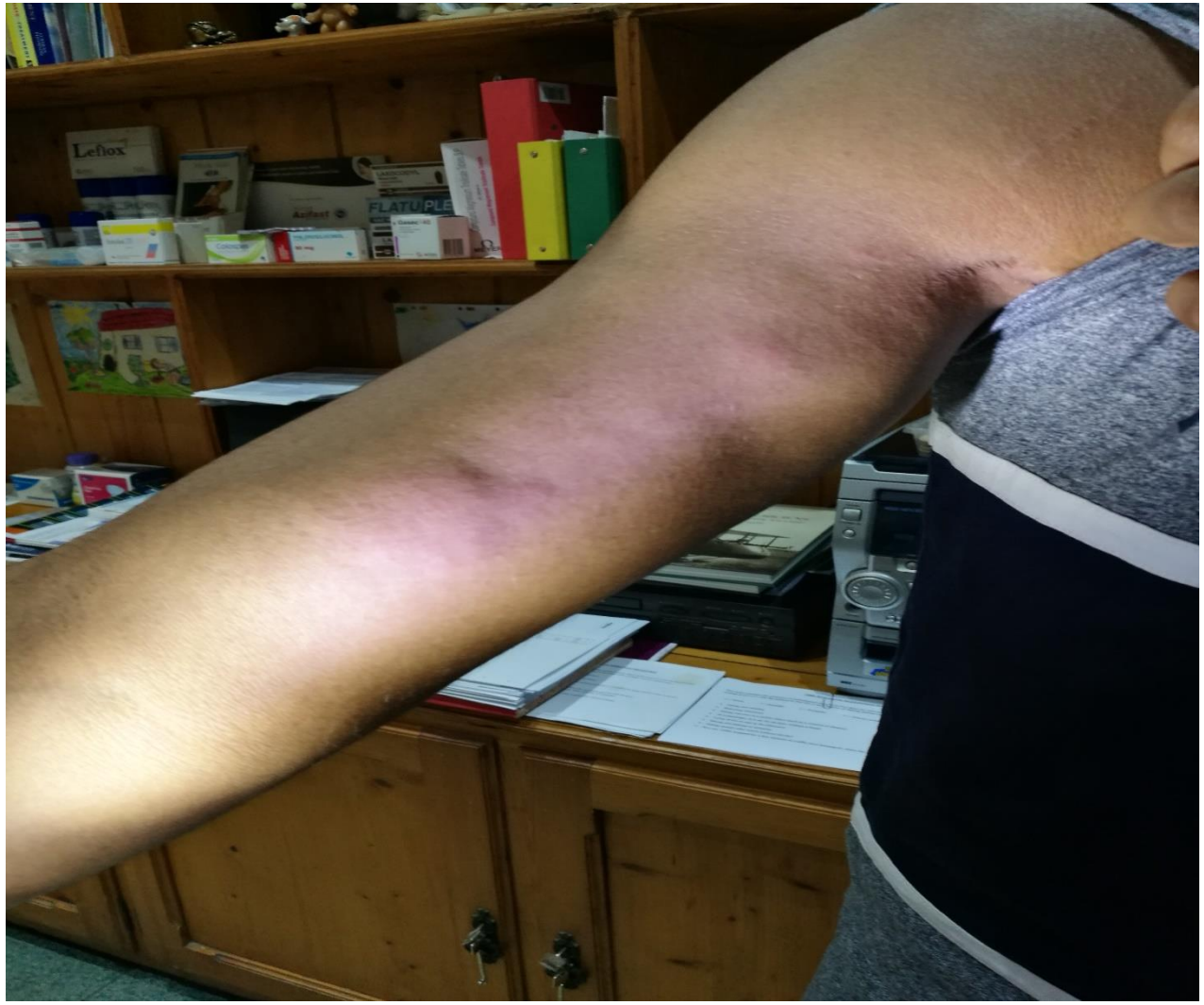
Answer

Detailed history taking !

Cephalosporin Urticaria



Angioedema



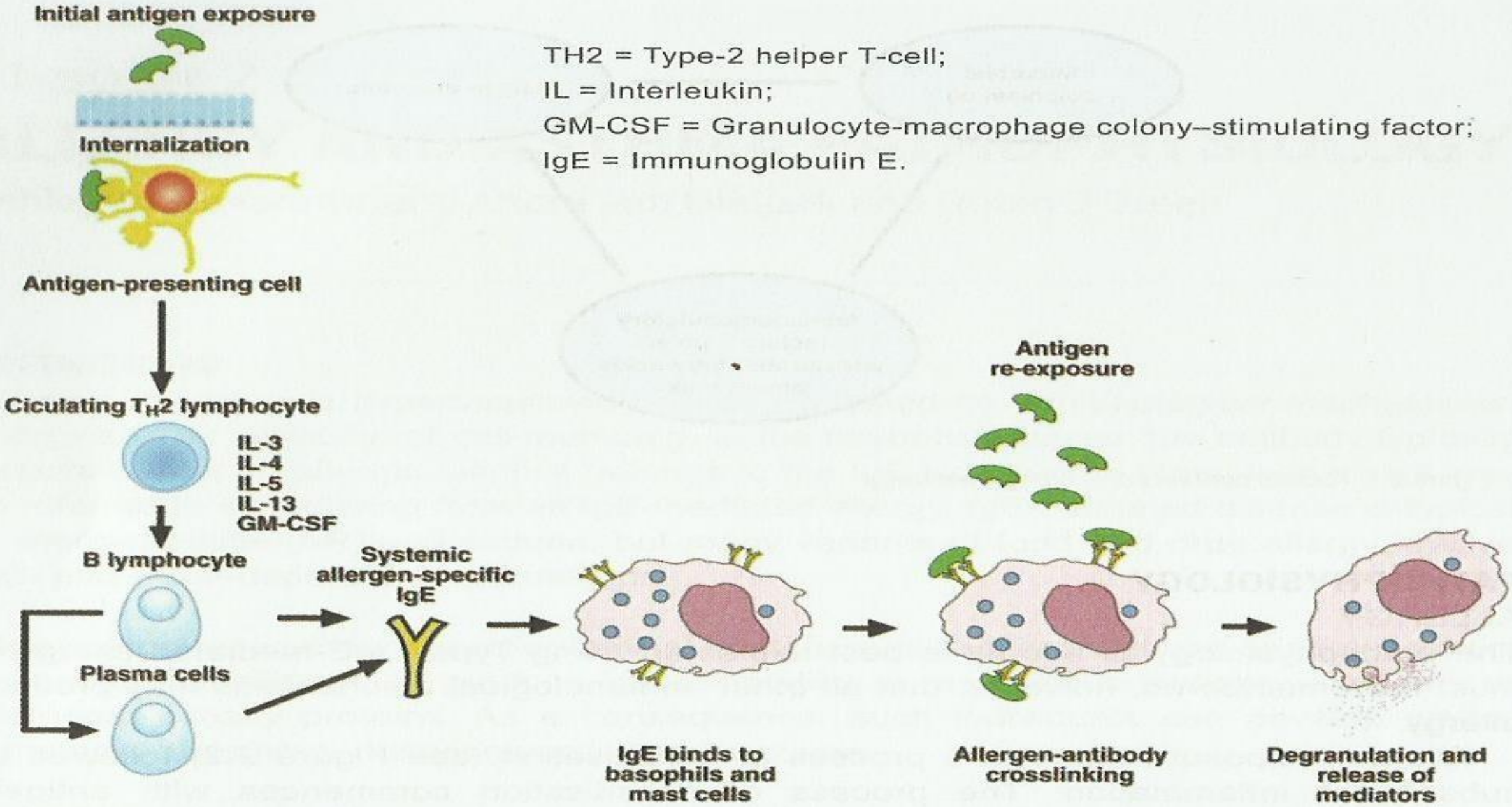
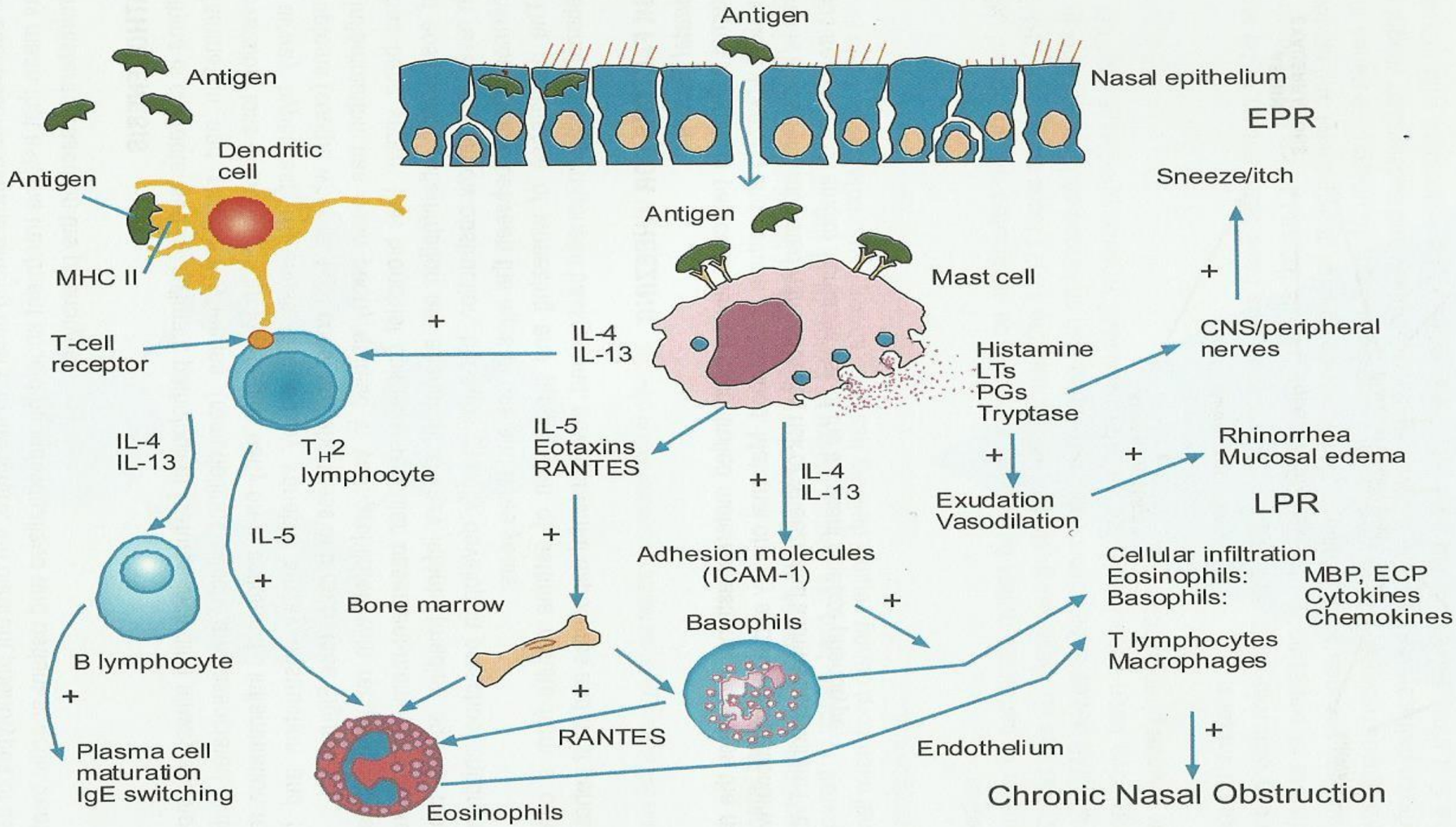


Figure 2.2: Type-1 hypersensitivity mechanism


Antigen
Antigen
Nasal epithelium
EPR
Dendritic cell
Antigen
Sneeze/itch
Antigen
MHC II
Mast cell
+
CNS/peripheral nerves
T-cell receptor
+
IL-4
IL-13
Histamine
LTs
PGs
Tryptase
IL-5
Eotaxins
RANTES
IL-4
IL-13
+
+
Rhinorrhea
Mucosal edema
IL-4
IL-13
T_H2 lymphocyte
+
Exudation
Vasodilation
LPR
IL-5
+
+
Adhesion molecules
(ICAM-1)
Cellular infiltration
Eosinophils: MBP, ECP
Basophils: Cytokines
Chemokines
Bone marrow
Basophils
+
T lymphocytes
Macrophages
B lymphocyte
+
+
RANTES
Endothelium
+
Plasma cell
maturation
IgE switching
Eosinophils
Chronic Nasal Obstruction

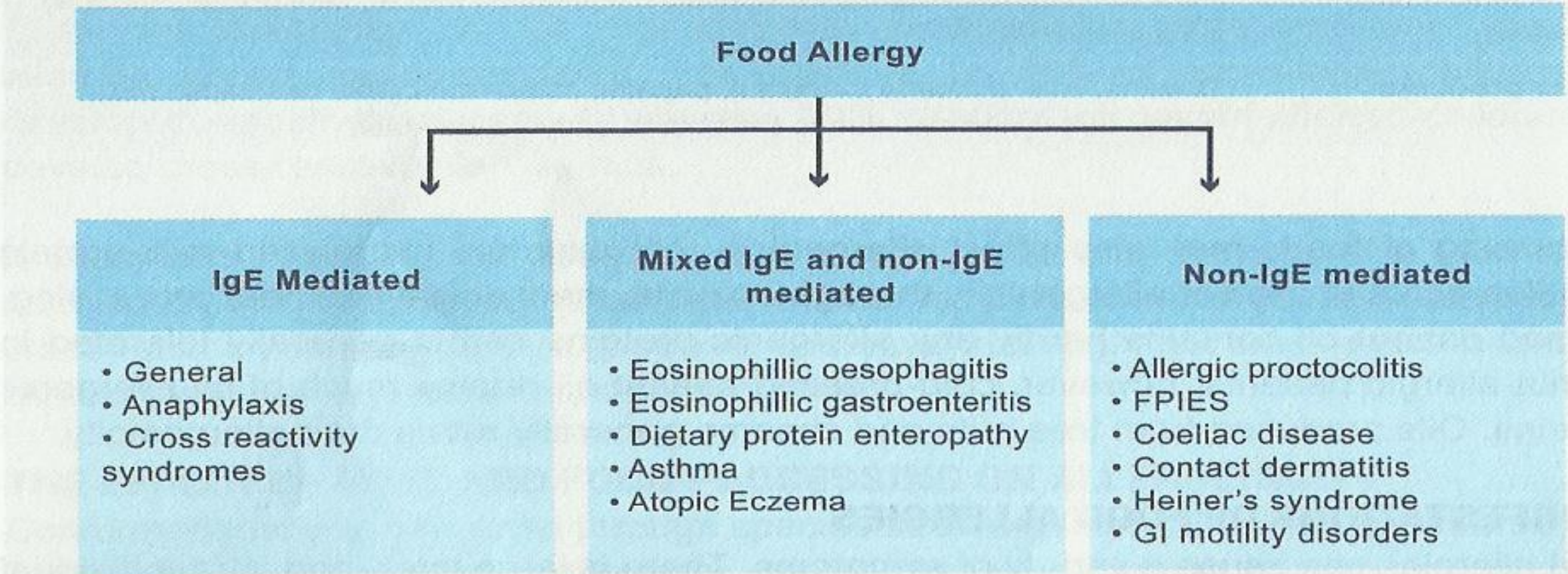


Figure 9.2: Overview of clinical manifestations of food allergy.

Is it truly food allergy ?

- Pineapple, strawberries : non Ig E histamine release- lip and mouth irritation
- Scrombroid fish poisoning : Histidine ⟹ Histamine
- Tyramine in old cheeses
- Theobromine in chocolate
- FREY's syndrome- auriculotemporal nerve syndrome of gustatory sweating and flushing

IgE-MEDIATED ALLERGIC REACTIONS

TABLE 9.2: SUMMARY OF IGE-MEDIATED FOOD-ALLERGIC REACTIONS

SYSTEM	MANIFESTATIONS
SKIN (produced by ingestion or skin contact with the food allergen)	• Urticaria and/or angioedema; • Pruritis, erythma and flushing; • Immediate worsening of eczema; • Morbilliform rashes and erythema after skin contact to fruit and vegetables such as tomato, citrus and berries; • Acute-localised urticaria after contact with food (e.g. seafood, eggs), especially by chefs and food handlers.
GASTROINTESTINAL	• Swelling, tingling and itching of lips and mouth; • Nausea and/or vomiting; • Abdominal pain, cramp, or colic; • Diarrhoea; • Oral-allergy syndrome
RESPIRATORY (can be produced by ingestion or inhalation of the food allergen)	• Rhinitis (sneezing, nasal blockage, itching of nose and throat, conjunctivitis); • Wheeze, tight chest or cough can be a sign of a severe allergic reaction (anaphylaxis). Wheeze seldom occurs in isolation and is usually associated with urticaria and or angioedema; • Swelling of larynx can produce stridor.
MULTI-SYSTEM/SYSTEMIC	• Anaphylaxis.
BEHAVIOURAL	• Behavioural changes may be noted as the first sign of an allergic reaction. Adults describe a 'feeling of impending doom'. Children may

TABLE 9.3: SIGNIFICANT QUESTIONS IN THE ALLERGY HISTORY

QUESTIONS	SIGNIFICANCE
What food allergen is thought to be causing the reaction?	• Is the allergen typical for age? For example, allergies to cow's milk and egg are common in young children but rare in adulthood. • In breast-fed infants food allergens may be transmitted via human milk.
Timing of the reaction post exposure?	• IgE-mediated allergic reactions usually occur within 20 minutes of the ingestion and always within two hours thereof. • Non-IgE-mediated immune reactions are typically more delayed in onset.
Description of allergic symptoms?	• If symptoms are not typical of an immediate onset IgE-mediated reaction then a *differential diagnosis* must be considered. Common confusing scenarios include: – Patients who experience peri-oral erythema and irritation provoked by contact with irritants such as raw tomato, citrus and berries. – Symptoms due to the Oral Allergy Syndrome (Pollen Food syndrome). – Chronic urticaria (where food allergy is a rare cause). – Disorders with symptoms which mimic anaphylaxis (e.g. anxiety disorders, vasovagal attacks, mastocytosis, hereditary angioedema and vocal cord dysfunction). • In some children food allergy may masquerade as a food aversion or refusal, but this may also be behavioural due to oral tactile aversions, or odour sensitivity.
Any other foods the patient may be allergic to or tends to 'dislike'?	• Ask specifically about foods causing co- and cross-reactivity with the index food. • Ask if a child is able to consume age-appropriate quantities of specific foods (e.g. a 5-year-old should be able to consume a whole egg/full glass of milk before being labelled as truly tolerant to the food).
Route of allergen exposure?	• A proportion of patients will react after skin contact (e.g. kiss contact or inhalation).
Is there a prior history of tolerance to the food (and cross-reacting allergens)?	• It is rare (but possible) to have a history of tolerance to a food prior to developing an allergy (e.g. peanut allergy usually presents on first known exposure). • Clinical reactivity may be influenced by the amount of food allergen eaten, fat content, and route of allergen exposure. • Processing of the food is important (e.g. egg allergic children will commonly tolerate baked egg but react to foods containing undercooked egg protein).
Concomitant disease, and in particular, allergic disease?	• The majority of children with food allergy will have eczema, and at least 25% will go on to develop additional food allergies. • Food-allergic infants are at risk for the development of asthma; and asthma is a risk factor for more severe food-induced allergic reactions.
Associated factors, for example asthma, medication use (especially ACEI, β-blockers and salicylates), alcohol intake and/or exercise?	• All are factors which may increase the severity of allergic reactions.

Which of the following is true ?

- Cows Milk Protein Allergy (CMPA) will resolve in > 70 % patients before adulthood.
- An amino acid milk formula is the first line treatment
- Baked milk products should be encouraged if tolerated
- Only a minority of peanut allergic patients will resolve
- A patient whose mouth itches with oranges is likely to be latex allergic
- A patient who has anaphylaxis at midnight should not receive rituximab

Cow’s milk protein allergy (CMPA)

- At time of cow’s milk being introduced in the infant diet
- Often associated with eczema
- More severe if also asthmatic
- 2 – 7.5 % in early childhood
- Ig E mediated cf: FPIES
- Skin Prick Testing or blood Fx 5 screen and then specific testing for milk

SPT reagents

Skin prick testing

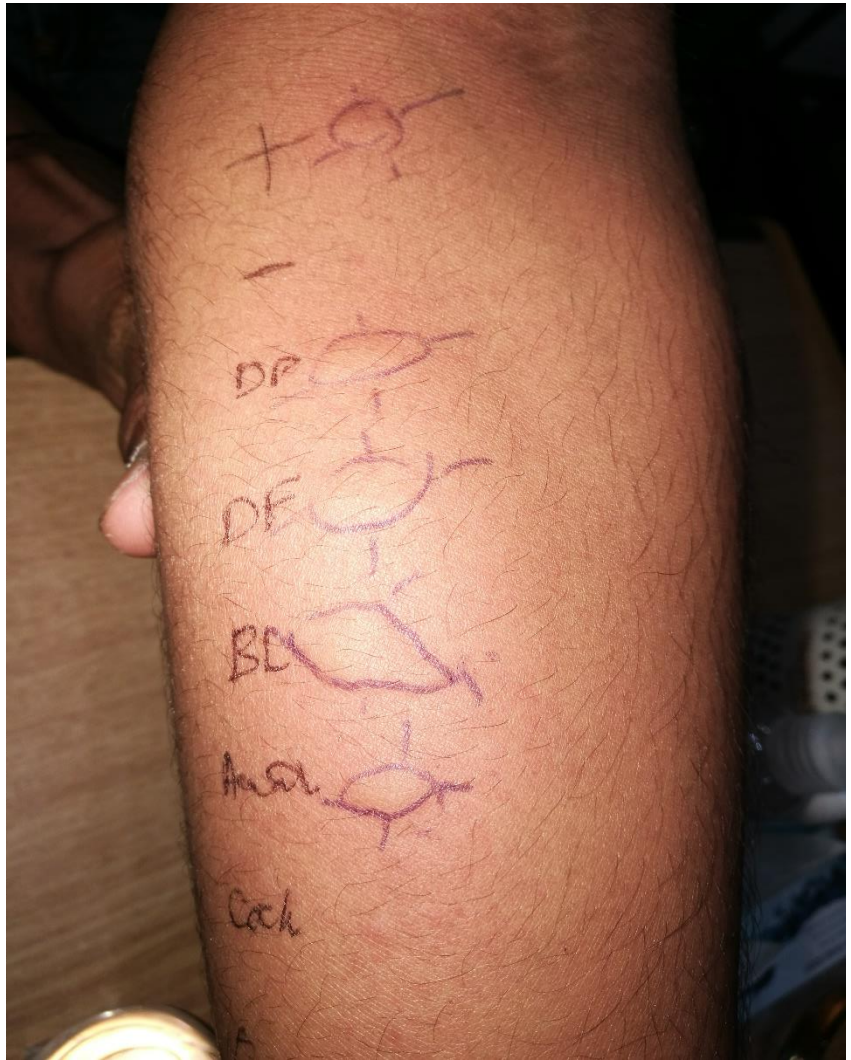


TABLE 9.4: 95% POSITIVE PREDICTIVE VALUES FOR COMMON FOOD ALLERGENS	
≥95% SPECIFIC-IGE LEVEL (KU/L) POSITIVE PREDICTIVE VALUES*	
Egg	7
Infants ≤2 years	2
Milk	15
Infants ≤2 years	5
Peanut	15
Tree Nuts	15
Fish	20
≥95% SPTS (WHEAL DIAMETER IN MM) POSITIVE PREDICTIVE VALUES**	
Milk	8
Infants ≤2 years	6
Egg	7
Infants ≤2 years	5
Peanut	8
Infants ≤2 years	4

** ImmunoCAP® (Sampson 2001)*
***SPTs performed with commercial extracts (Sporik 2000)*

CMPA

- 50 % tolerant by age 8 yr, 80 % by age 16 yr
- Soya milk tolerated by 90 % but test for soy allergy first
- If not , Extensively Hydrolised Milk Formulas (EHF)
- A minority will require Amino Acid Formulae (AAF)
- Never other non-bovine milk eg Goat, mare (X- reactive)

- If tolerated (80 %) allow and encourage BAKED MILK products

Egg allergy

- Egg allergy (0.5 – 2.5 %)
- Egg white (ovomucoid) most allergenic
- Tolerance in 85 % by age 7 yrs
- Baked egg- biscuit, cake, cupcakes- tolerated by 80 %
- Vaccines produced in eggs potentially a problem if severe allergy - Influenza, Yellow ,fever , rabies. Assess risk-Benefit ratio
- MMR vaccine grown in Chick fibroblasts- no danger

Peanut allergy

- Tends to be lifelong in 75 %
- One of the leading causes of death from food allergy
- 30 % Co allergic to tree nuts and sesame
- Countries where introduced early in diet – low prevalence
- Israel, Mauritius
- LEAP study confirms this

Wheat

- Wheat dependent exercise-induced anaphylaxis (Ω - 5 gliadin)
- Occurs during exercise within 2 hrs of wheat containing foods
- Often missed because of time interval

Oral allergy syndrome = Pollen Food Syndrome

- Due to class 2 allergens- easily digestible- so oral cavity only
- Itching, burning lips and oral cavity; no other symptoms
- Commoner in adolescents and adults
- Occurs when individuals sensitised to grass or tree pollen are exposed to certain fruits/veg.
- X – sensitivity because of shared allergens (profilins, PR-10)
- Grass pollen X-reacts with melon, tomato,orange, peach,celery
- Latex-Fruit syndrome : avocado, banana, kiwi, papaya, peach, apricot,chestnut (enzyme chitinase)

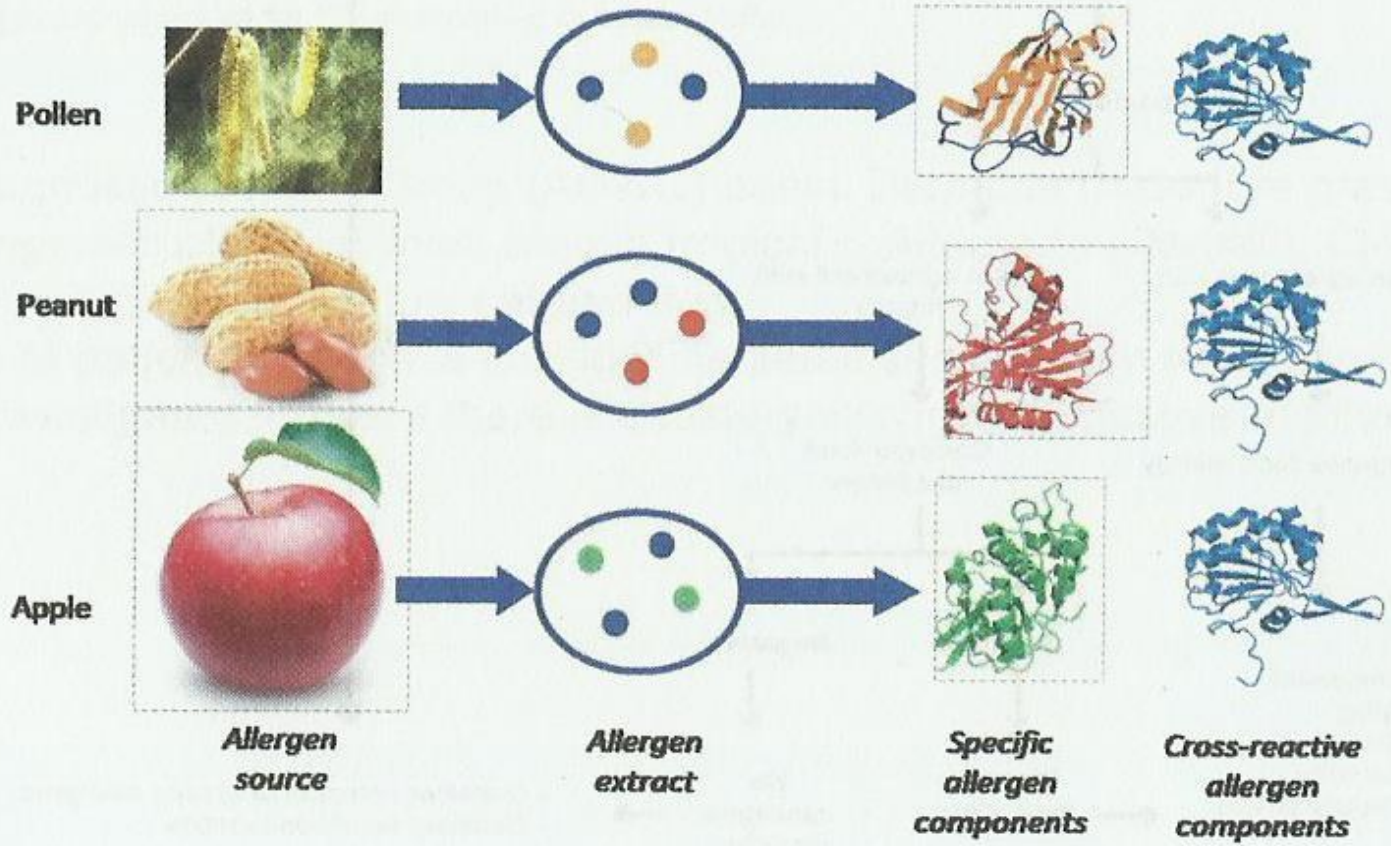


Figure 26.1: Illustration of specific and cross-reactive allergen components

TABLE 9.1: EXAMPLES OF CROSS-REACTING FOOD ALLERGENS

FOOD ALLERGEN	CROSS-REACTING FOODS
Peanuts	Tree nuts, Legumes (e.g. green peas, lentils, soya), sesame, lupin
Specific closely related tree nuts	*Anacardiaceae* family co-sensitisation: pistachio and cashew nut, and to a lesser degree with Brazil nut *Juglandaceae* family co-sensitisation: walnut and pecan nut. *Proteaceae* and *Betulaceae* family co-sensitisation: hazelnut and macadamia
Sesame	Peanut, possibly other seeds (e.g. poppy)
Finfish	Other finfish
Shellfish	Other shellfish
Molluscs	Other molluscs
House-dust mite	Crustaceans and molluscs – due to a similar muscle-protein tropomyosin
Wheat	*Poaceae* grains (e.g. barley, oat and rye). Seldom to *Festucoideae* grains (e.g. rice, corn)
Cow's milk	Goat's milk. Cross-reactivity between cow's milk and soya is significant only in non-IgE-mediated food allergy
Tree pollen (e.g. birch tree)	Fruit and vegetables (e.g. apple, peach, pear, cherry, hazelnut, carrot, raw potato, kiwi, banana, tomato) This causes PFS due to conserved functional proteins such as profilin
Grass pollen	Fruit and vegetables (e.g. raw tomato) – causing PFS
Mugwort pollen	Celery, apple, peanut, kiwi fruit, carrot, parsley, spices (fennel, coriander, aniseed, cumin), causing PFS
Weed pollen (e.g. ragweed)	Fruit and vegetables (e.g. watermelon, honeydew melon, cantaloupe, banana) – causing PFS
Latex products	Banana, kiwi, avocado, chestnut, papaya, pitted fruits. This can cause the 'Latex fruit syndrome' due to the shared enzyme chitinase

Midnight anaphylaxis (2009)

- Delayed (3 hours after meal) anaphylaxis to red meat, usually BEEF
- Antibodies to galactose -α -1,3 galactose (α-gal)
- Polysaccharide, major blood group substance in non-primates
- Ixodus Tick bites cause humans to become sensitised
- Diagnosis : Ig E antibodies to beef (lamb) and α-gal
- Also causes Immediate hypersensitivity to CETUXIMAB
- Monoclonal antibody used for cancer Rx
- Cetuximab has an α- gal side chain

& CLINICAL IMMUNOLOGY

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Basics and beyond

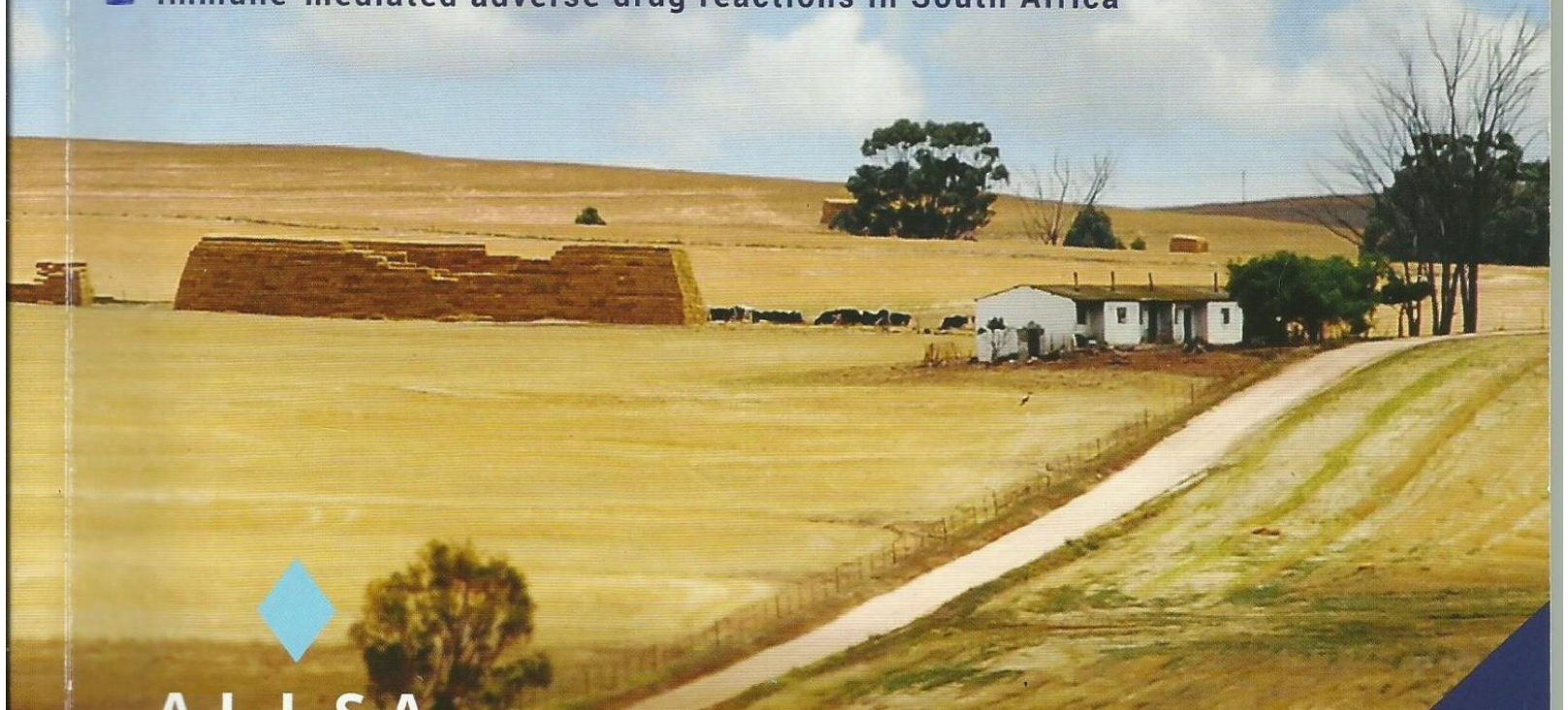
The interaction between respiratory allergies and infection

Peanut allergy: changing epidemiology and management

Allergy prevention: Something old, something new, something borrowed, something blue

Gluten-related disorders: making sense of Coeliac Disease and other reactions to wheat and gluten

Immune-mediated adverse drug reactions in South Africa



ALLSA

Which of the following are true ?

- There is 15 % X-reactivity between penicillin allergy and cephalosporin allergy
- At least 50 % of patients who say they are penicillin allergic are truly so
- Allergy to one cephalosporin contraindicates all other cephalosporins
- Cephalosporin allergic patients can safely be given penicillin
- It is mandatory to do an intradermal skin test before administering a parenteral antibiotic

Drug allergies

- Still very common practice to do cutaneous drug allergy testing before antibiotic administration
- Prick test, intradermal or subcutaneous ?
- Irritant drugs- Cipro, clarithro
- If NO History of previous reaction no need to skin test
- If + ve history, Dangerous to do intradermal test on a ward ; refer for SPT in fully controlled environment or blood tests

Prophylactic antibiotics before surgery

- Important to ask about antibiotic allergy before admission
- Best to administer when patient still awake
- Can then report early symptoms of allergy eg itch
- Otherwise may be detected quite late
- Bronchospasm, shock state

Penicillin Allergy

- Commonest reaction is non specific maculopapular rash not associated with immunological memory
- Only 1 in 20 people labelled as penicillin allergic have true Ig E mediated hypersensitivity requiring avoidance
- Do SPT using standard penicilloyl reagents and ampicillin +/- cephalosporin
- In vitro tests (IgE, CAST test) if very severe reaction
- 1/3 of truly penicillin allergic individuals can have negative SPT
- Always do provocation test even if all results negative

Penicillin and cephalosporins

- Only 2 % X- reactivity between penicillin and G2, G3 C-sporins; even less for G 4
- If proven penicillin allergy, do SPT with cephalosporin
- If negative, give 1/10 of cephalosporin dose initially to be safe. < 1 % chance of a reaction
- In patient allergic to a particular cephalosporin give a c-sporin with a different side chain; SPT and test dose first
- If patient is cephalosporin allergic and needs penicillin, SPT/IDT and test dose if negative

Which of the following are true ?

- > 90 % of aspirin sensitive patients are also NSAID sensitive.
- The majority of NSAID sensitive patients have Ig E mediated allergy
- Patients with Chronic Spontaneous Urticaria are at increased risk of reacting to NSAIDS
- NSAIDS are absolutely contraindicated in patients with asthma
- A patient who reacted to diclofenac can safely be given celecoxib

TABLE IV: KEY INFORMATION REQUIRED TO COLLECT WHEN ASSESSING OR REFERRING A PATIENT WITH SUSPECTED DRUG ALLERGY

1. Detailed description of the reaction:
   - Clinical features, sequence, duration and severity;
   - Treatment required and outcome of therapy;
   - Witness description:
   - Photographs.
2. Timing of symptoms in relation to drug administration.
3. Has the patient had the suspected drug before this course of treatment?
   a. How long had the drug(s) been taken before reaction?
   b. *When was/were the drug(s) stopped?*
   c. What was the impact of stopping?
   d. Has the patient tolerated drugs of the same class or other preparations with the same parent drug, e.g. generics?
5. Illness for which the suspected drug was being taken, i.e. underlying disease which may have caused symptoms; alternative treatment options which may be able to be utilised.
6. List of all drugs taken at time of the reaction (including over-the-counter medicines) **NB!** In assessment of anaphylaxis during general anaesthesia the anaesthetic charts should be looked at for a detailed list of drugs given.
7. Previous history of other drug reaction, allergies and co-morbidities.

TABLE II: DRUGS CAUSING THE MAJORITY OF ADVERSE REACTIONS REFERRED TO ALLERGY CLINICS

ANTIBIOTICS
• Penicillins and other Beta-Lactams
• Non-Beta Lactam antibiotics, e.g. tetracyclines, macrolides
REACTIONS DURING GENERAL ANAESTHESIA DUE TO*
• Neuromuscular blockers
• Anaesthetic agents, e.g. propofol
• Exposure to latex or cleaning solutions, e.g. chlorhexidine or iodine
ACE inhibitors
Aspirin and other NSAIDS
Local anaesthetics, e.g. lignocaine
Radio-contrast media
Others including: insulin, opiates, vaccines, heparin, plasma expanders

**Peri-operative antibiotics still a major cause of drug reactions during general anaesthesia*

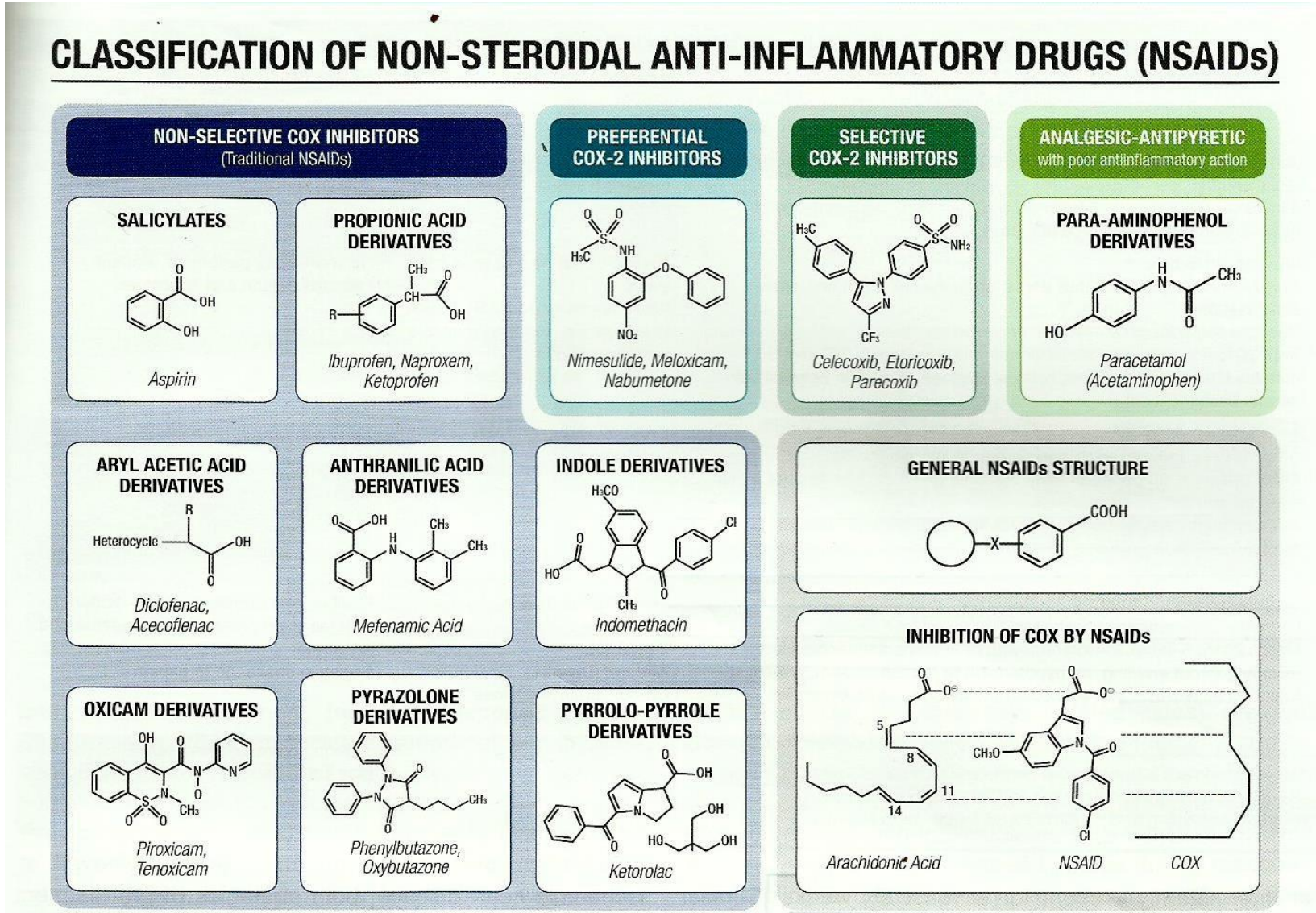


Figure 1: Classification and chemical structure of NSAIDs

TABLE II: CLASSIFICATION OF HYPERSENSITIVITY TO NSAIDS

GROUP	TIMING OF REACTION	CLINICAL SYMPTOMS	CROSS-REACTIVITY AMONG NSAID CLASS	ASSOCIATED/ UNDERLYING DISEASE	PUTATIVE MECHANISM
A	Multiple NSAID-exacerbated urticaria/angioedema in patients with underlying cutaneous disease (NECD)				
	Acute	Urticaria/angioedema	Yes	Chronic spontaneous urticaria	COX-1 inhibition
B	Multiple NSAID-induced urticaria/angioedema in otherwise asymptomatic patients (NIUA)				
	Acute	Urticaria/angioedema	Yes	None	Likely COX-1 inhibition
C	Single NSAID-induced anaphylactic reactions (SNIUAA)				
	Acute	Anaphylaxis, Urticaria/angioedema	No	Atopy	IgE-mediated
D	Aspirin- or NSAID-exacerbated respiratory disease (AERD or NERD)				
	Acute	Rhinitis, nasal congestion, bronchoconstriction, asthma exacerbation	Yes	Asthma/ rhinosinusitis/ nasal polyps	COX-1 inhibition
E	Delayed reactions to NSAIDs (SNIDR)				
	Delayed (>24hrs)	Varied: maculopapular drug eruptions, fixed drug eruptions, bullous skin reactions, maculopapular drug eruptions	Single or cross-reactive	None	Varied: T-cell-mediated, cytotoxic T-cells, NK cells, other

TABLE V: *IN VIVO* AND *IN VITRO* DIAGNOSTIC TESTING AVAILABLE IN SA FOR THE ASSESSMENT OF DRUG HYPERSENSITIVITY REACTIONS

DIAGNOSTIC TEST	AVAILABILITY IN SA	MAJOR UTILITY	COMMENTS
Tryptase	Widespread	Confirmation of anaphylaxis (Cannot distinguish IgE- from Non IgE-mediated mast cell degranulation).	Requires sampling immediately after reaction, at 2 hours (up to 6 hours still useful) and then a baseline for comparison (> 24 hours after reaction or at follow-up).
Skin prick tests (SPTs)	Limited to clinics where resuscitation equipment and experienced staff are available.	Provides evidence of clinical sensitisation to drug. Useful for beta-lactams, and neuromuscular blockers.	Useful when parent drug or metabolite causing reaction is known and used in testing. Predictive value/utility undetermined with drugs where relevant immunogen is unknown, e.g. clindamycin. Parenteral or commercial preparations should be used skin prick testing.
Intradermal testing (IDT)	Limited to clinics where resuscitation equipment and experienced staff are available.	Improves the sensitivity of SPT for same agents where SPTs are reliable, e.g. beta-lactams.	Require extensive experience to perform and interpret. Non-irritant drug concentrations must be known and matching patient controls should be used to confirm non-irritating drug concentrations to reduce risk of false positives.
Serum specific IgE testing	Widespread although available drugs vary between major SA laboratories.	Useful to confirm sensitivity in severe reactions where SPT/IDT testing may be dangerous.	Not available for many drugs. Uncertain sensitivity and specificity; mainly useful if positive to confirm suspected culprit drug and prevent the need for further testing in severe anaphylactic reactions or to add further confidence when negative to progress to drug challenge test.
Cellular Antigen Stimulation Testing (CAST) ELISA	ADCRU (performs testing on samples referred to Pathcare and NHLS services)	Adjunctive *in vitro* testing in severe reactions encompassing IgE and non IgE mechanisms of reactions.	Measures leukotrienes after peripheral blood cell stimulation with drug. Not validated in large prospective studies with many drugs despite commercial availability. Useful for testing of drug excipients and specific drug formulation. Expert knowledge of local laboratory experience required for appropriate test interpretation.
FlowCAST	Ampath	Adjunctive *in vitro* testing in severe reactions encompassing IgE and non IgE mechanisms of reactions.	Measures basophil activation (CD63) markers after peripheral blood cell stimulation with drug. Useful for testing of drug excipients and specific drug formulation.
Patch testing	A number of dermatologists and dermatology clinics offer testing.	Useful for investigating Type IV, usually cutaneous drug reactions, e.g. rifampicin.	Experience required to prepare patches with appropriate drug concentrations and interpret the results at 24, 48 and 96 hours.

Clinical criteria for Anaphylaxis

- 1. Acute illness-skin / mucosa and airway or hypotension

- 2. Two or more of the following after exposure to known allergen
  - Skin/mucosa
  - Airway
  - gastrointestinal
  - hypotension

- 3. Hypotension alone after exposure to known allergen

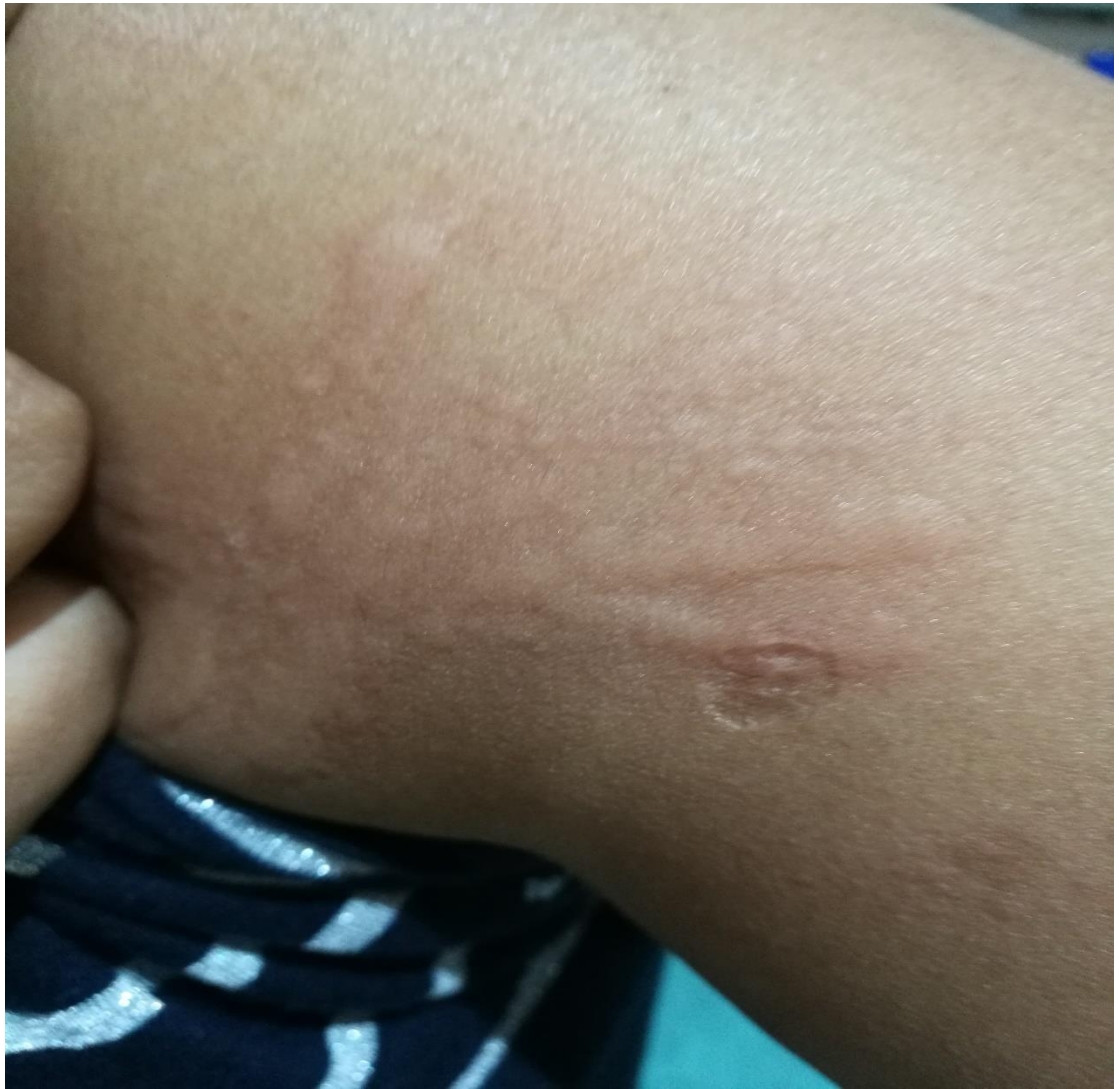
Anaphylaxis

- Charles Richet, 1902 Injected dog with sea anemone toxin to protect it. On reinjection 3 weeks later fatal reaction. Pro-phylaxis (protection) had failed, so called it a-phylaxis (without protection).
- Later changed to ANAPHYLAXIS because sounded nicer
- Essential message : adrenaline, adrenaline , adrenaline !!!
- Epipen – how to use
- Bracelets, dog tags

Epipen


NO MEDICINE
NO NEEDLE
TRAINER
FOR PRACTICE ONLY
See Trainer Insert for full instructions.
Lot 5500027
EpiPen
To reset Trainer:
1 Replace blue safety release
2 Pinch sides push down
Prdt. Code: 500-00
MS:50000:LBB:R1

Guess what ?

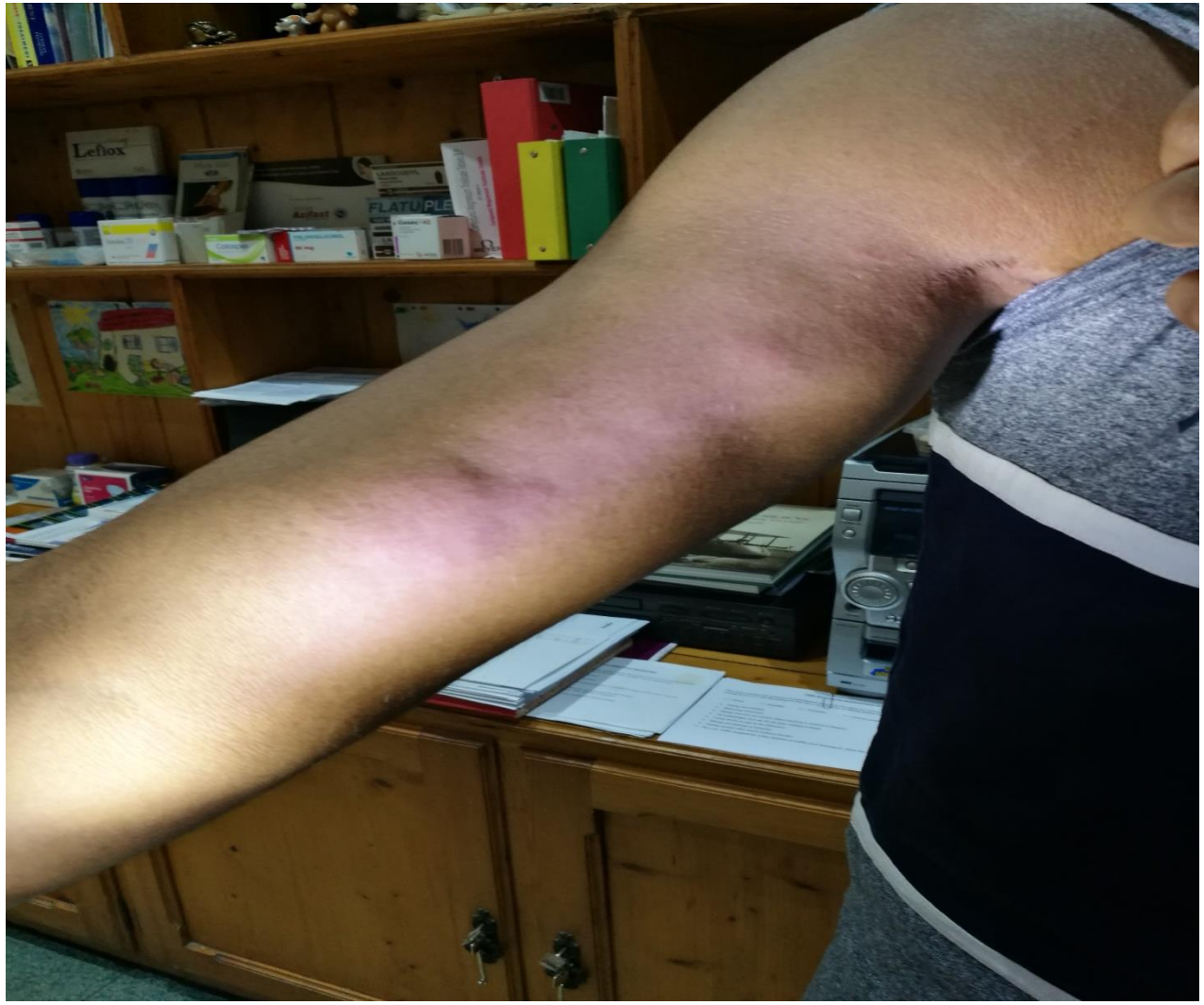


Stress urticaria

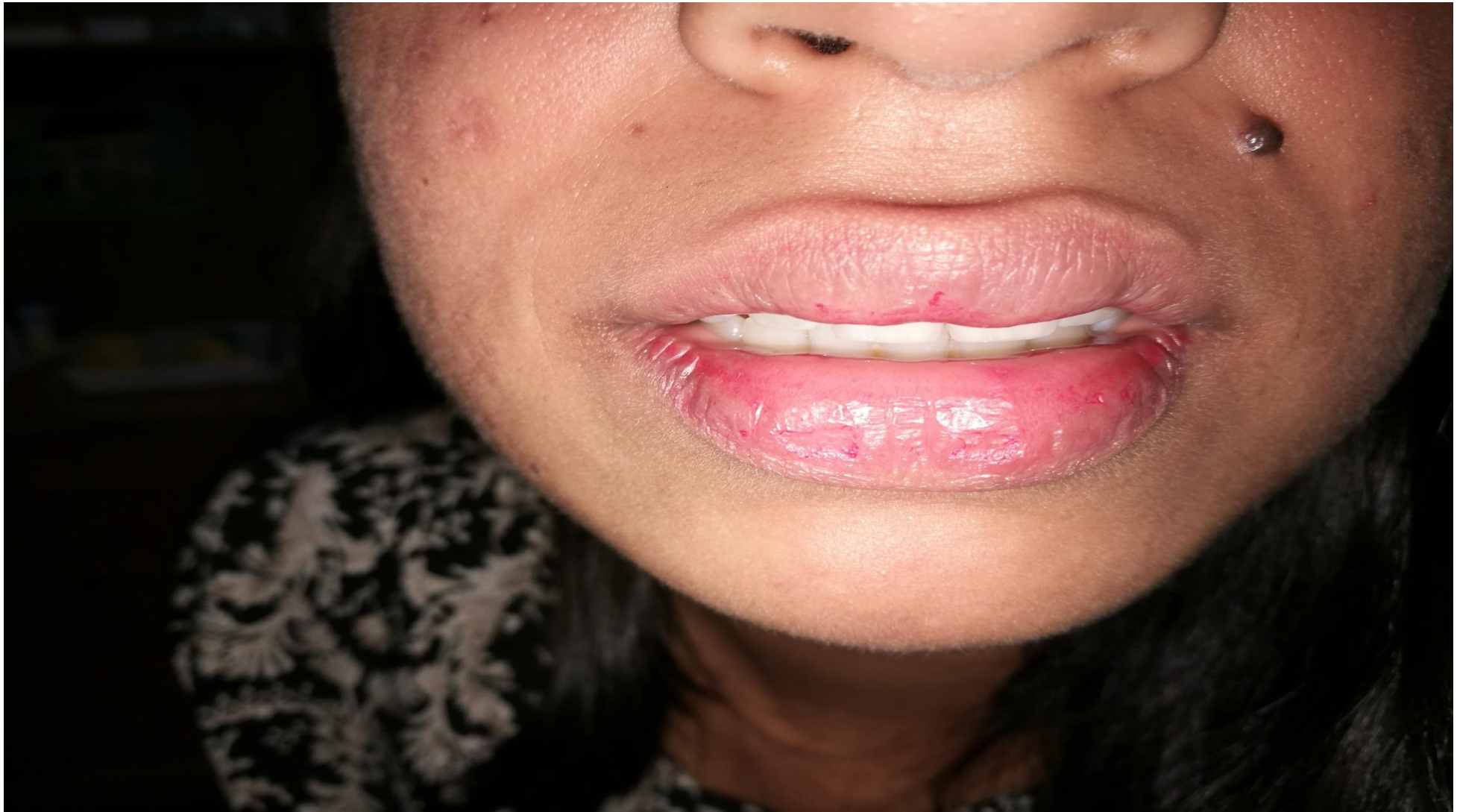
- MENTAL STRESS is one of the commonest triggers of urticaria angioedema in Mauritius
- Screen for obvious drug allergy (ACE inhibitors) and systemic disease, including MALIGNANCY , THYROID DISEASE and AUTOIMMUNITY
- Autoantibodies to Ig E and Ig E receptor on mast cells may play a role

- Detected by autologous serum inoculation test

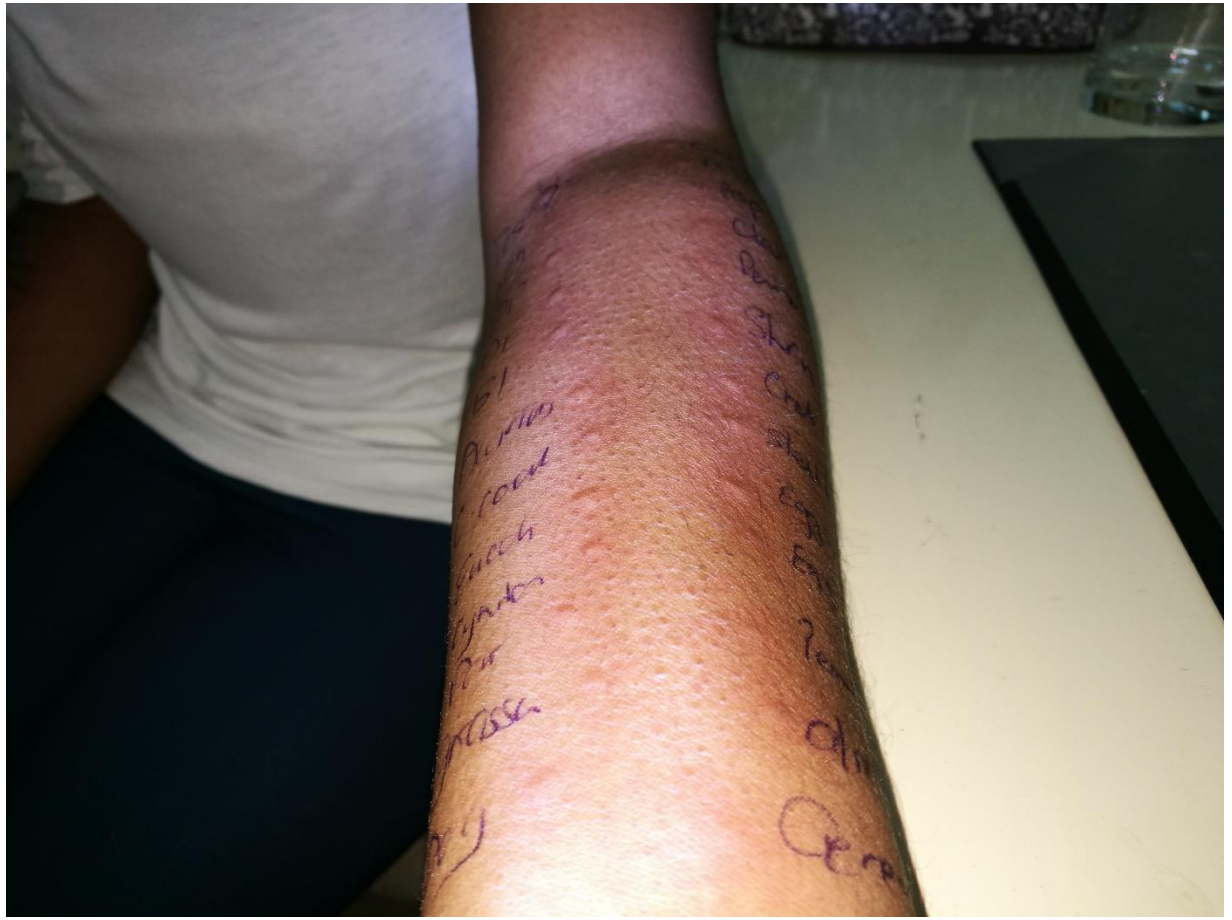
Angioedema



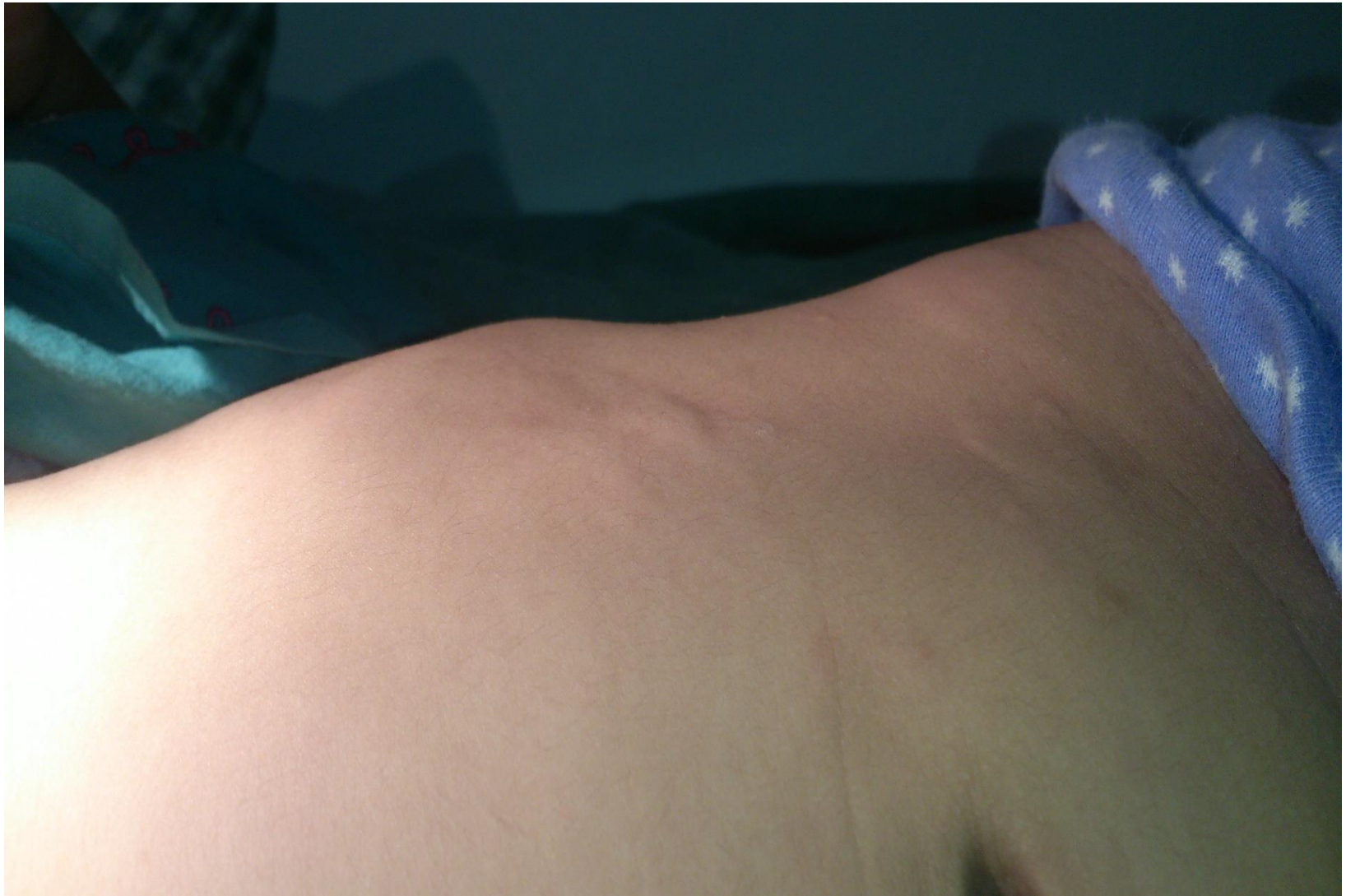
Angioedema lips



Dermatographism

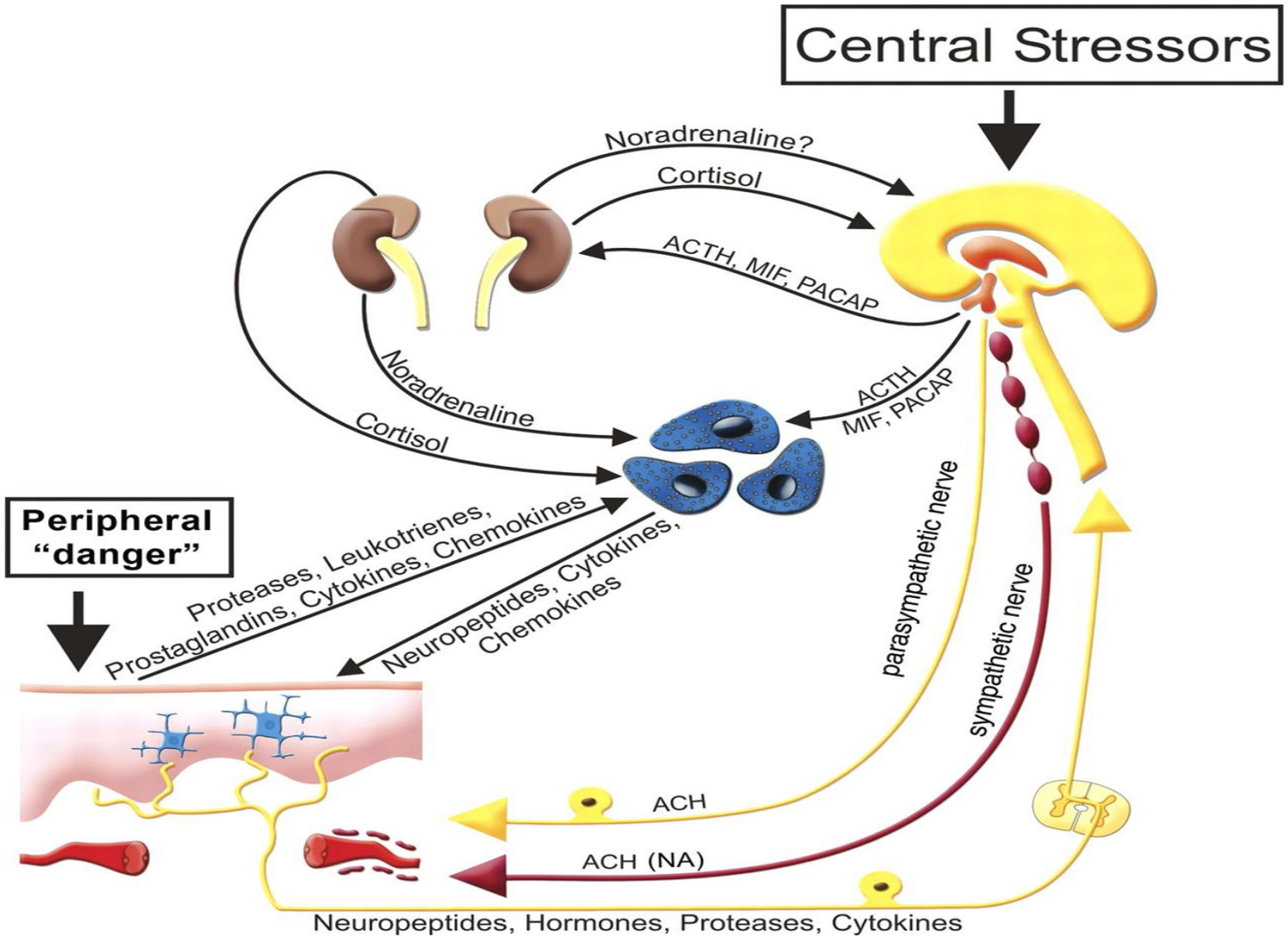


Cephalosporin Urticaria

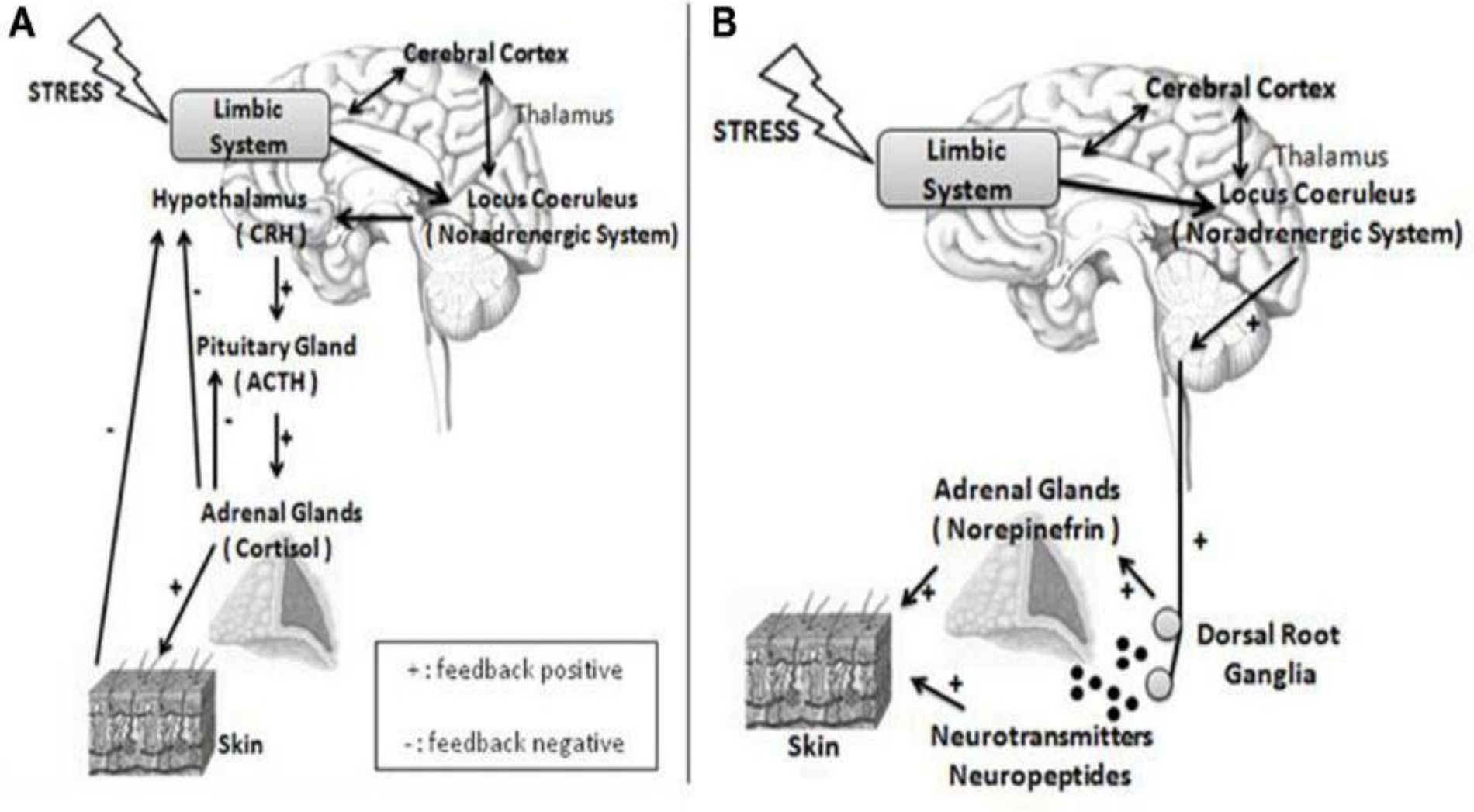


Goji Berries

Brain-Skin interaction



Stress, Limbic system and skin



The following are proven allergy reduction strategies

- Avoidance of peanuts and fish in the last 6 months of pregnancy in mothers who are allergic to these foods
- Avoidance of antibiotics in the last trimester of pregnancy
- Avoidance of paracetamol in the infant
- Delaying the introduction of allergenic foods till age 1 year
- Avoidance of tobacco smoke exposure in pregnancy and infancy

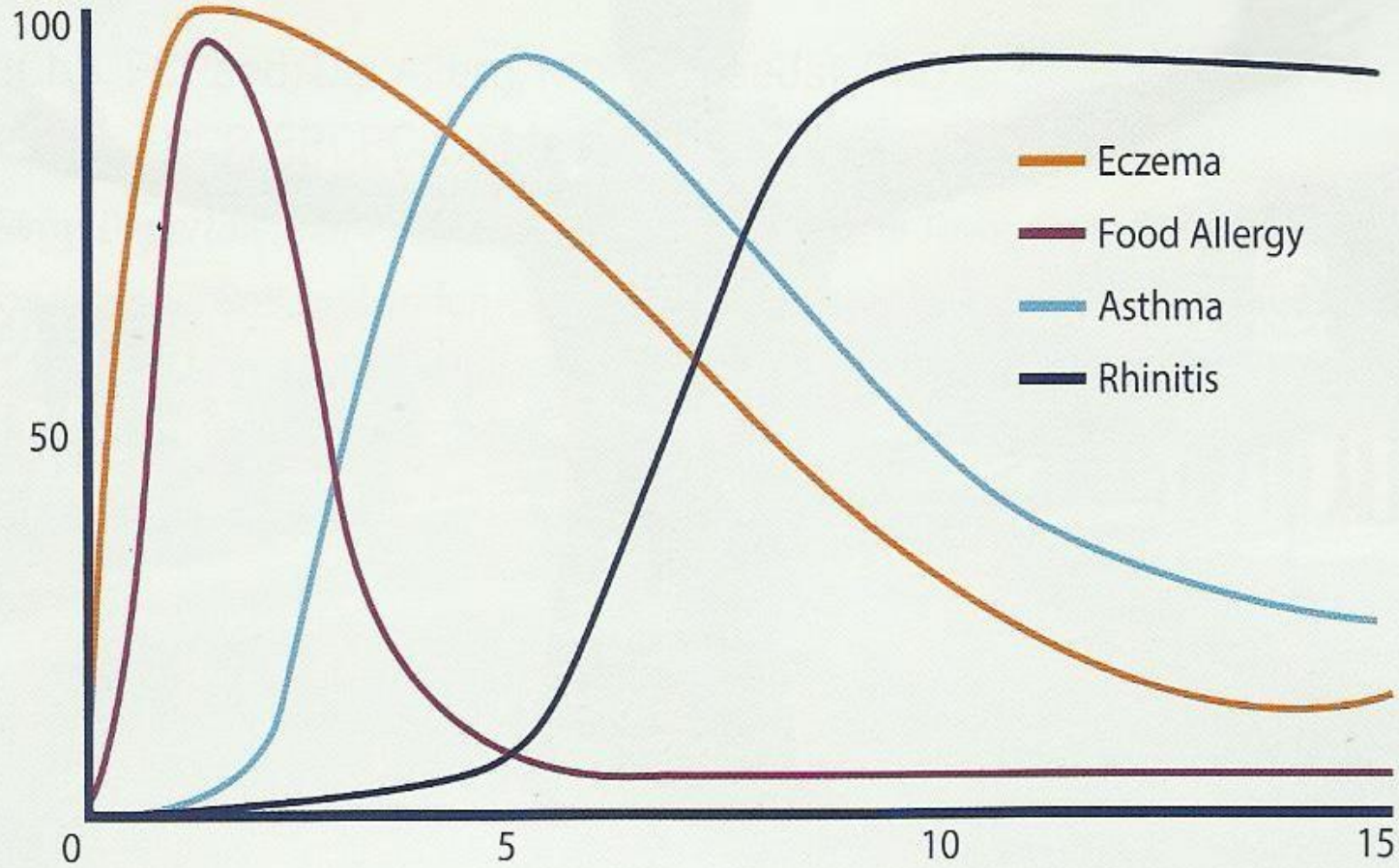


Figure 1: Diagramatic representation of the 'atopic march' from birth to adolescence

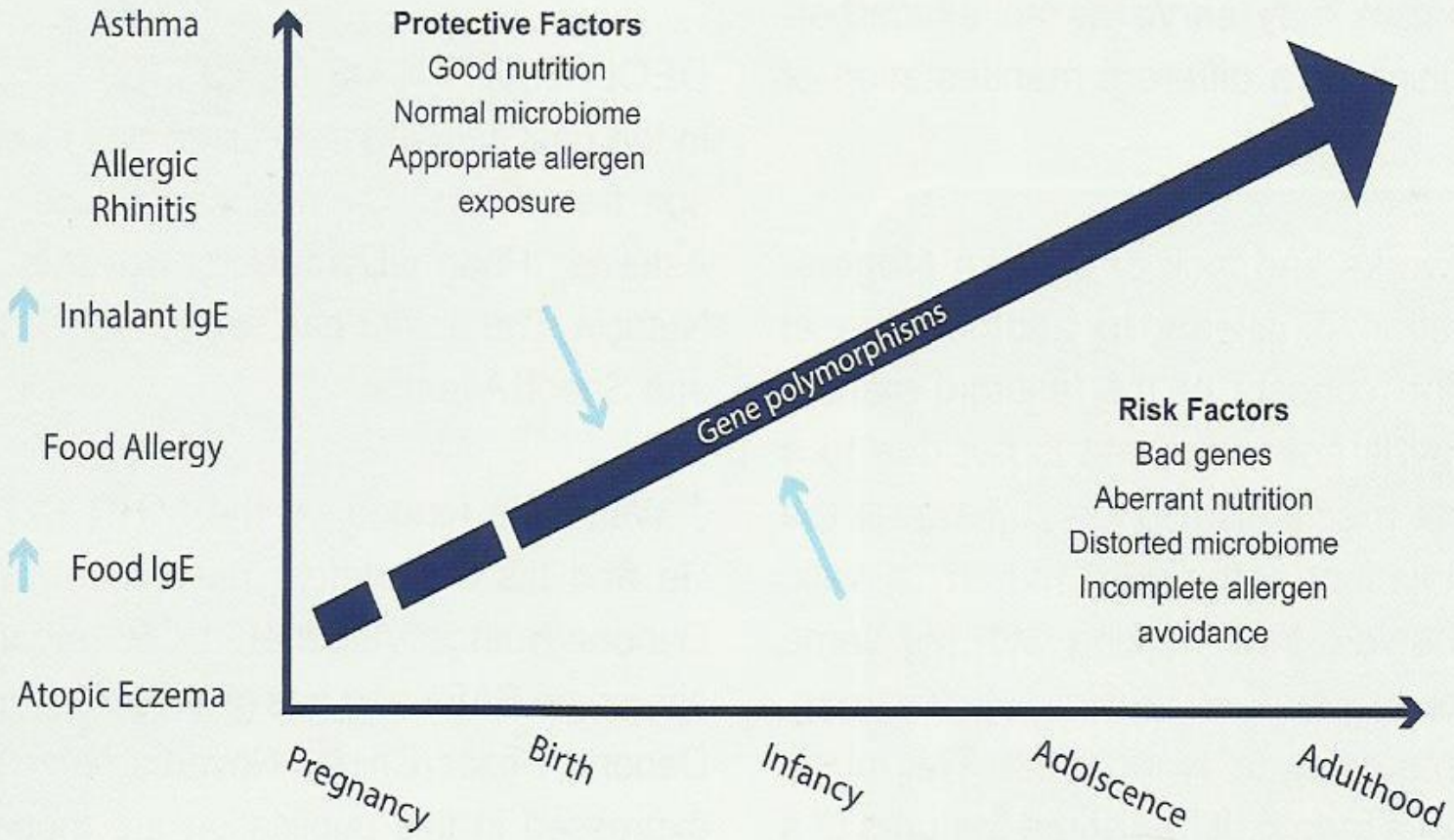


Figure 3: Protective and risk factors contributing to the evolution of allergic diseases across the life course

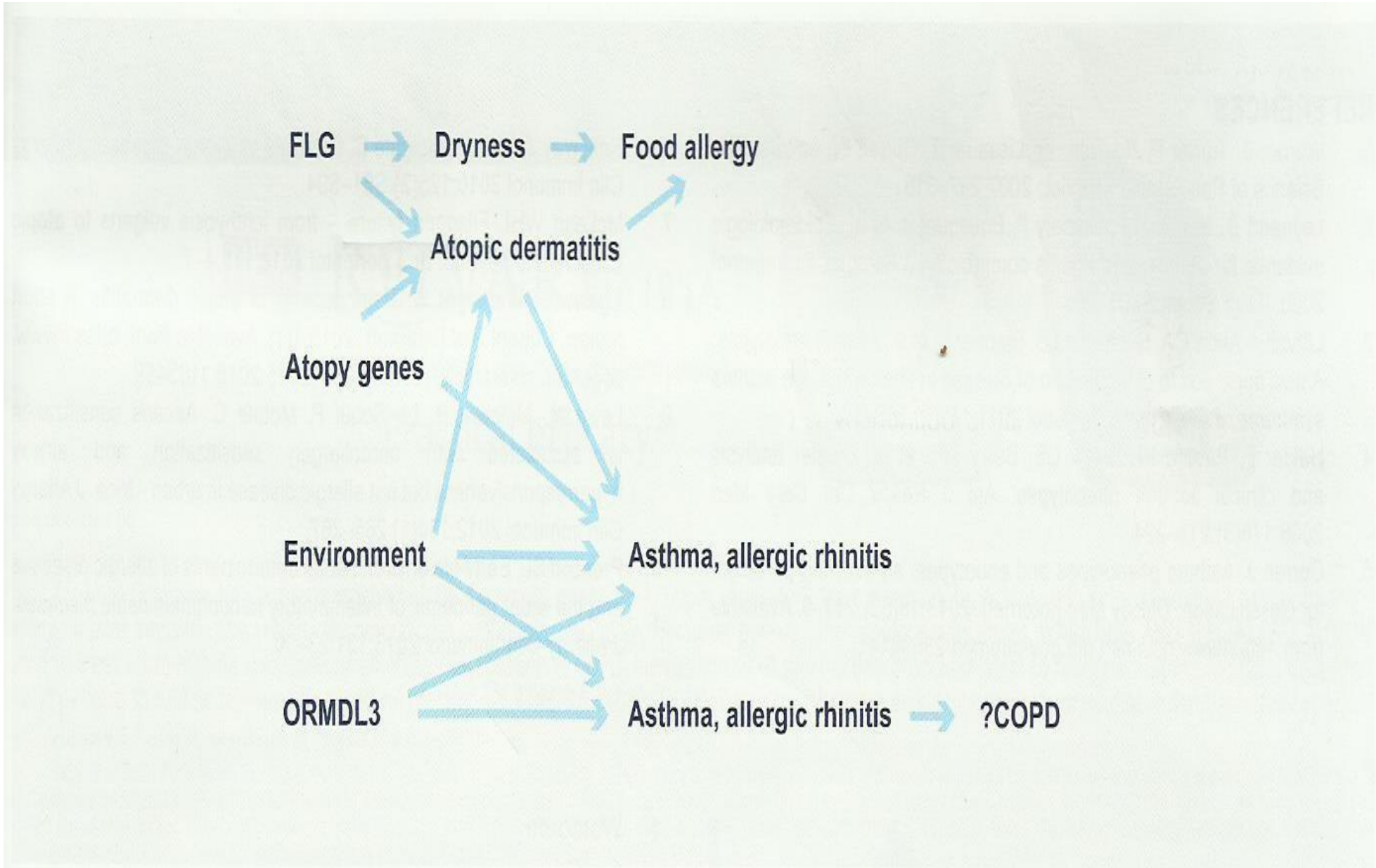


Figure 4: The Atopic Tango: Interaction between genetic, environmental factors and other allergic 'diseases' in allergy

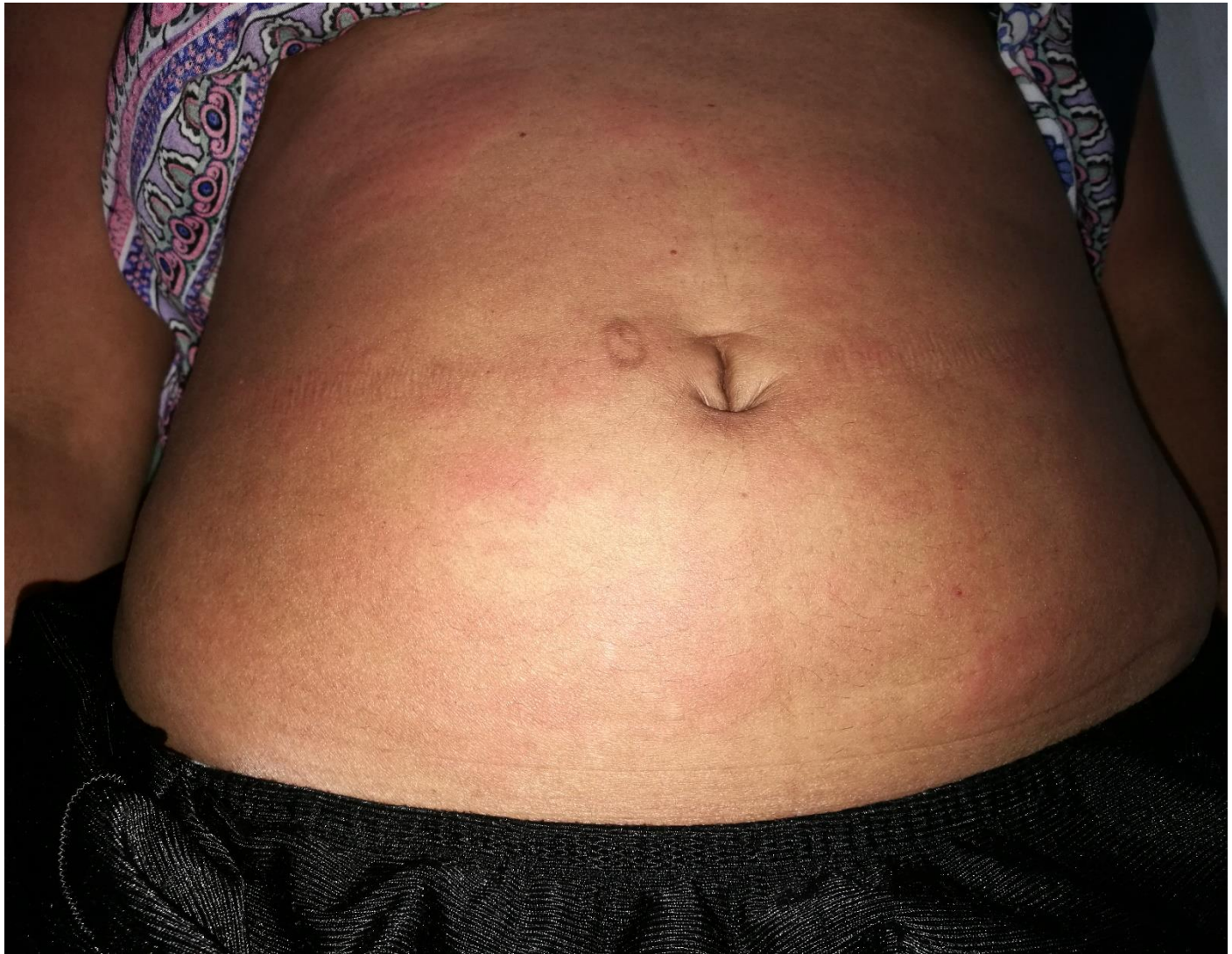
TABLE 28.1: SUMMARY OF ALLERGY-PREVENTION STRATEGIES

- Maternal avoidance of allergenic foods is not recommended during pregnancy.
- There is inconclusive evidence for peanut avoidance during pregnancy.
- A generally healthy diet is recommended during pregnancy, with as much dietary diversity as possible.
- No special diet is required for the lactating mother (except if the infant is already showing manifestations of particular allergies).
- There is no clear evidence to support the use of supplements such as probiotics, fish-oil supplements or vitamin D during pregnancy and lactation.
- Exclusive breastfeeding for up to 6 months is recommended for its general beneficial properties; however, its role in the protection against food allergy risk remains unclear. Ideally, there should be an overlap between breastfeeding and solids introduction.
- Introduction of complementary foods is recommended between four and six months of age irrespective of atopic heredity.
- There is no evidence to delay introduction of allergenic solids such as dairy, wheat, egg and peanut beyond six months of age (an exception is the child who is already showing signs of allergies: such children need a thorough allergy assessment to guide introduction of allergenic solids).
- There is evidence that earlier introduction of peanut in high-risk patients can reduce peanut allergy.
- The infant should have as diverse a diet as possible in the first year of life.
- There is some evidence that partially or extensively hydrolysed formulae may provide protection against food allergy or eczema.
- There is no evidence for use of soya milk (or milk of other mammalian origin such as goat's milk) as an allergy-prevention strategy.
- There is insufficient evidence to recommend supplementations including probiotics, fish oil or vitamin D to the infant as an allergy-prevention strategy.
- Optimising skin-barrier function through nonallergenic moisturisers from an early age and decreasing the use of drying soaps and detergents may reduce the risk of atopic dermatitis.
- Avoidance of excessive maternal antibiotics during the latter half of pregnancy, antibiotics in the infant, and paracetamol in the infant may be protective against development of asthma.
- Excessive maternal psychological stress during pregnancy should be avoided.
- Prenatal and passive exposure to tobacco smoke is an important risk factor in the development of asthma and should be strongly discouraged.

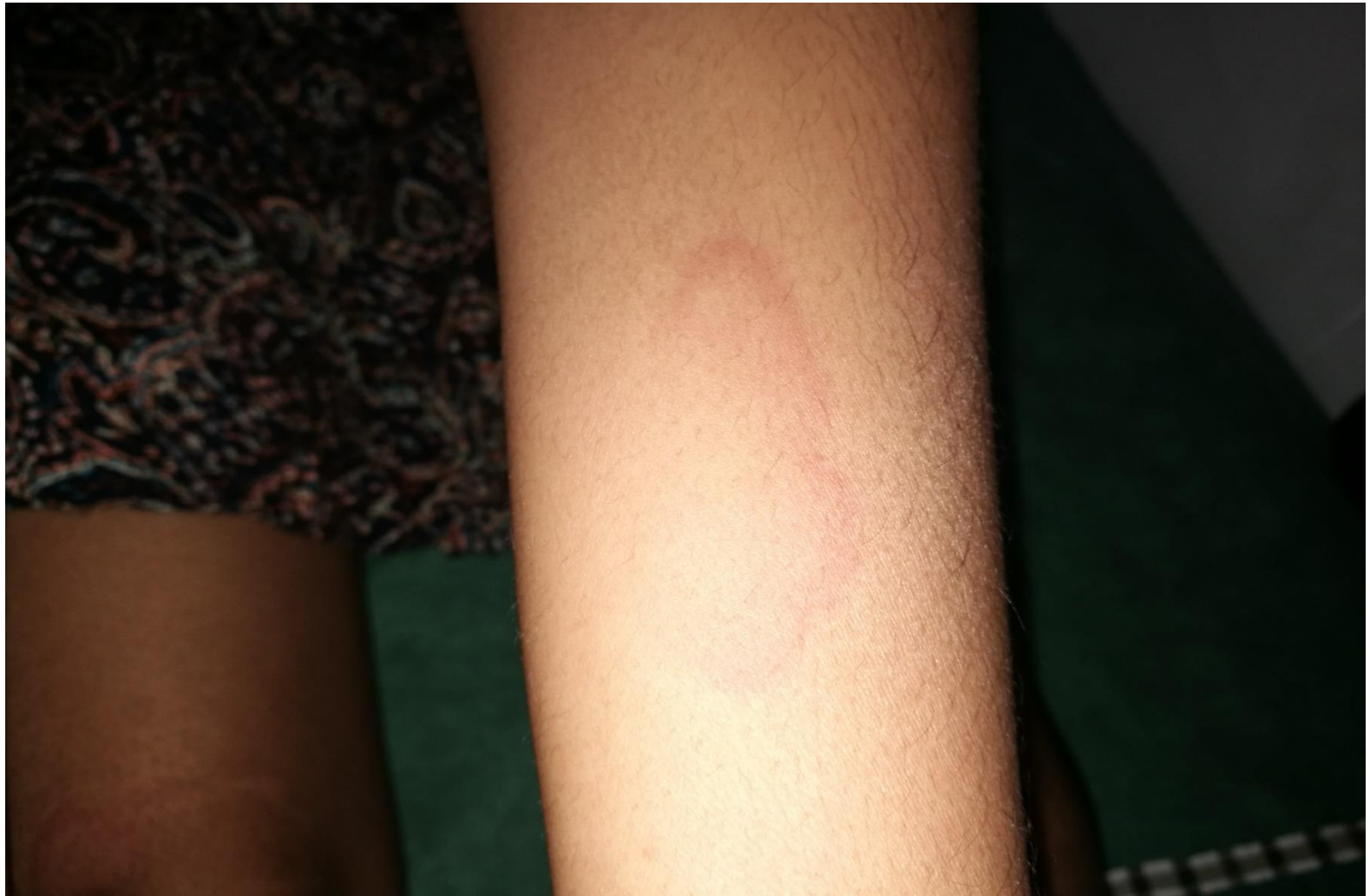
Thank You

- Think about PREVENTION
- Accurate diagnosis- think allergy and intelligent testing.
- Avoid giving bad advice- “arrête manze la chair, arrête tou protein, etc. “
- Know how to recognize and treat ANAPHYLAXIS. If in doubt, ADRENALINE !

NSAID Urticaria



Exam angioedema



OTHER FOODS

TABLE 9.6: OTHER COMMON ALLERGENIC FOODS

FOOD	TYPICAL AGE OF ONSET	COMMENT	PERSISTENCE LIKELY
TREE NUTS	Infancy	Tree-nut allergy can also develop later in life, or as part of the pollen–fruit syndrome.	Yes
SESAME SEED	Early childhood	Most reactions occur to sesame concentrates (e.g. hummus and tahini). Reactions to sesame oil and loose seeds (e.g. in bread) are rare but possible.	Yes
OTHER SEEDS (MUSTARD, POPPY, LINSEED, FLAXSEED)	Early childhood	Mustard-seed allergy is common in France.	Yes
FINFISH	Early childhood	Aerosolised fish-induced reactions are common.	Yes
SHELLFISH	Later childhood		Yes
MOLLUSCS	Later childhood		Yes
KIWI	Early childhood	Cross-reactivity with golden kiwi is high. Commonly associated with sensitivity to banana and avocado. Cross-reactivity with other tropical fruit (e.g. passion, dragon, star fruit) is not well studied.	No
OTHER FRUIT (PITTED FRUITS, MELONS)	Common in early infancy as contact allergen, then in older children as part of OAS	The seeds of fruit may prove allergenic to those with seed-storage protein allergies (e.g. citrus seeds/pip allergy in those with cashew-nut allergy).	No
LEGUMES/PULSES (SOYA, PEA, LUPIN, CHICK-PEA, BEAN)	Early childhood	A common allergy in children of South Asian descent, termed the 'Asian Allergy Syndrome'.	No

Skin Prick Test- dermatographism