



# Manifestations of Food Allergy

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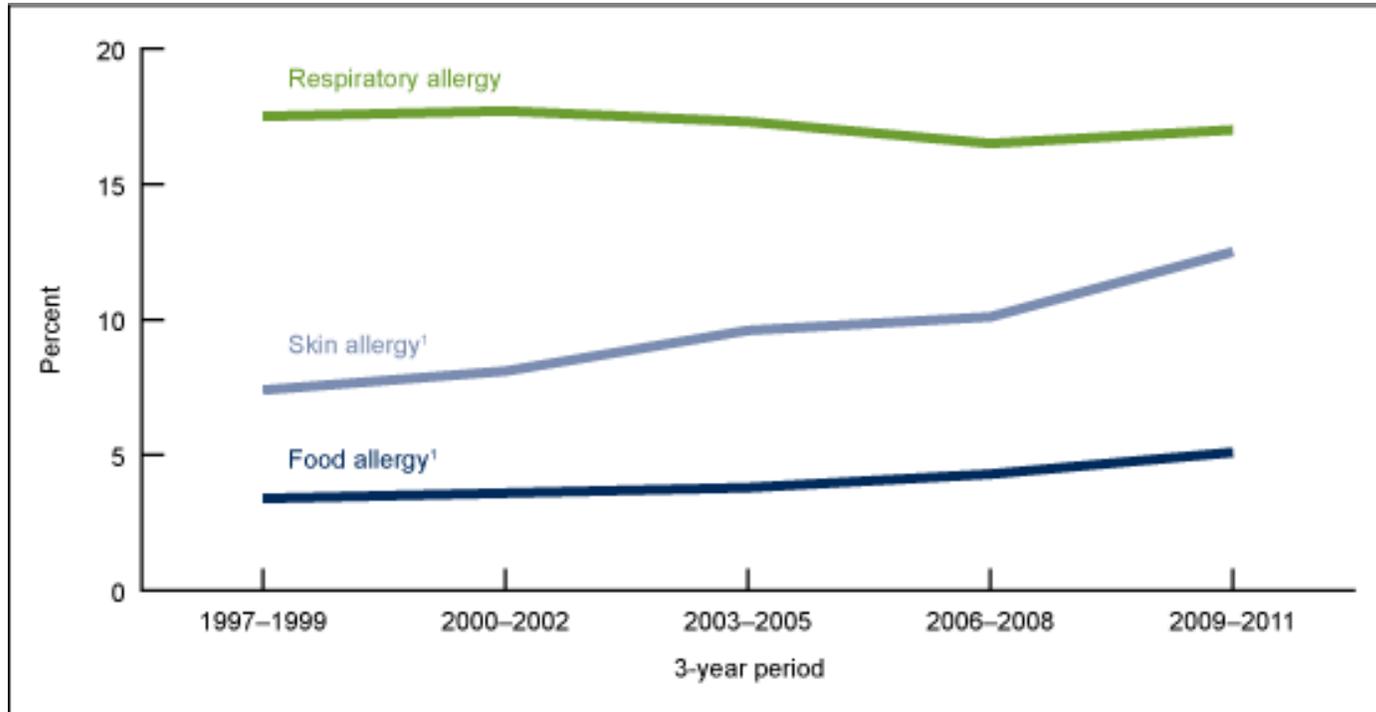
FAAP, FACAAI, FAAAAI

Pediatric Allergy Immunology

Mississippi Center for Advanced Medicine

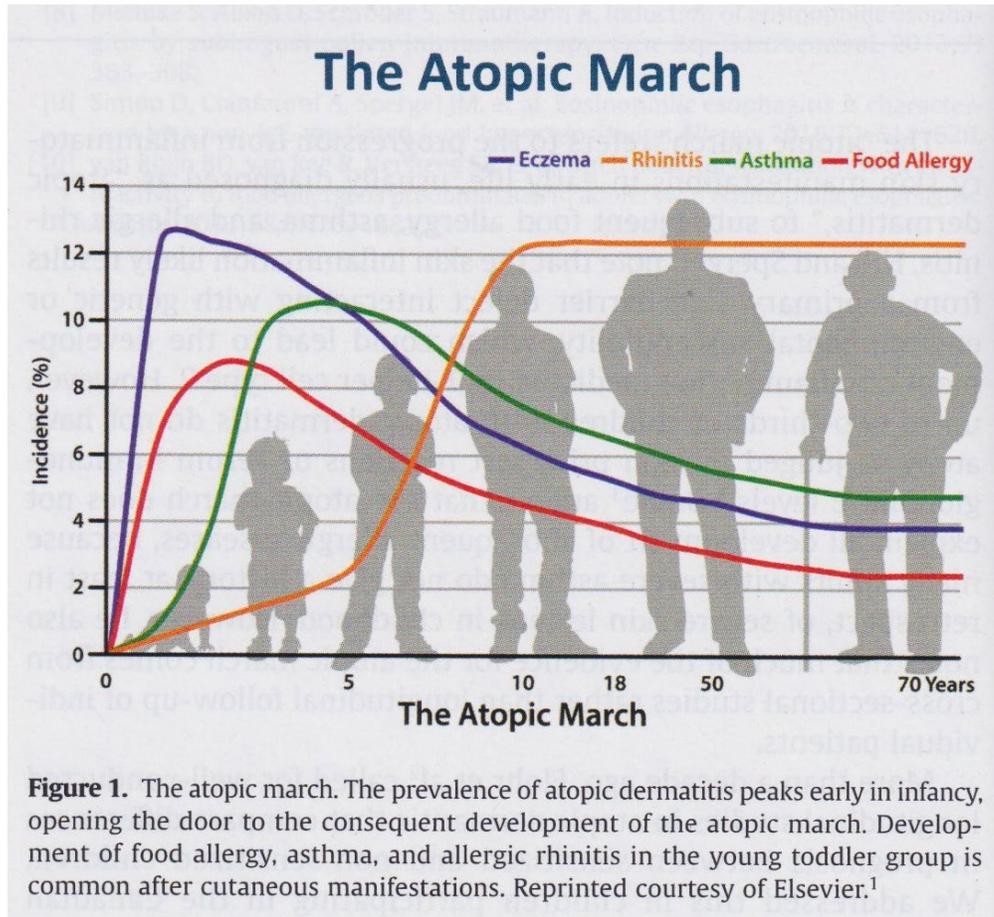


# Food allergy and skin allergy increased in US children age 0–17 years from 1997–2011.

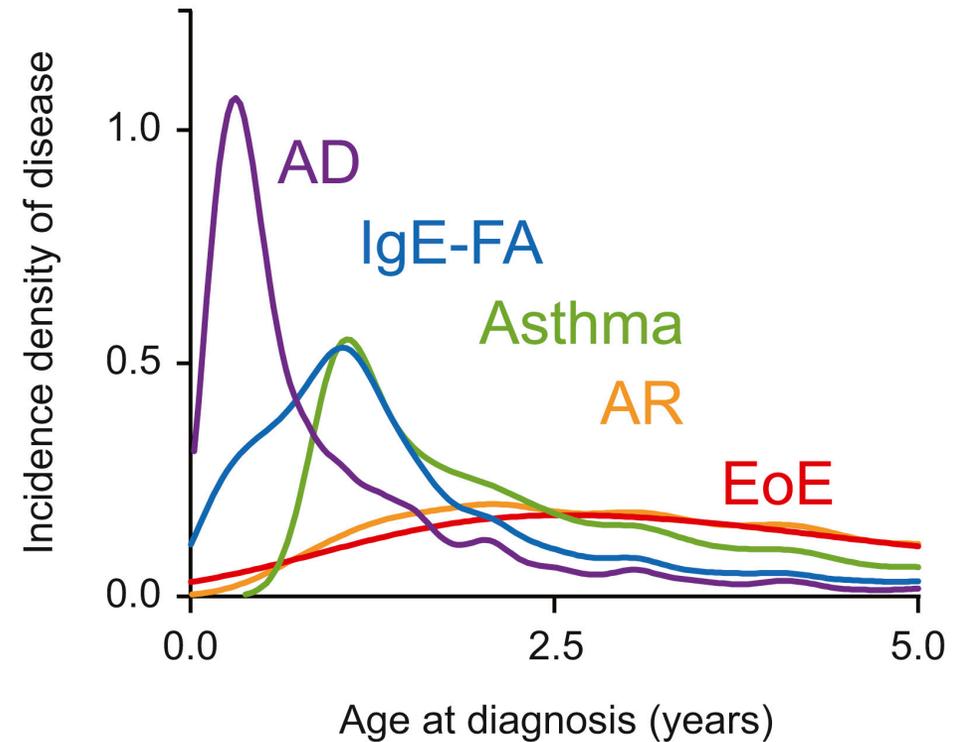


- Among children aged 0–17 years, the prevalence of food allergies increased from 3.4% in 1997–1999 to 5.1% in 2009–2011.
- The prevalence of skin allergies increased from 7.4% in 1997–1999 to 12.5% in 2009–2011.
- The prevalence of respiratory allergy (asthma and allergic rhinitis) did not change from 1997–1999 to 2009–2011, yet respiratory allergy remained the most common type of allergy among children throughout this period (17.0% in 2009–2011).

# The Atopic March

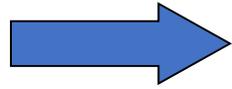


## The allergic march



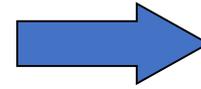
# Immunologic (Allergic) Adverse Food Reactions

## IgE-Mediated



- Systemic (Anaphylaxis)
- Oral Allergy Syndrome
- Immediate gastrointestinal allergy
- Asthma/rhinitis
- Urticaria
- Morbilliform rashes and flushing
- Contact urticaria

## Mixed IgE/Non IgE



- Eosinophilic esophagitis (EoE)
- Eosinophilic gastritis
- Eosinophilic gastroenteritis
- Atopic dermatitis

## Non-IgE Mediated Cell-Mediated

- Food Protein-Induced Enterocolitis
- Food Protein-Induced Enteropathy
- Food Protein-Induced Proctocolitis
- Dermatitis herpetiformis
- Contact dermatitis

# Evaluation: History & Physical Exam

- History: most important
  - Symptoms, timing, reproducibility, treatment and outcome
  - Concurrent exercise, medications
- Diet details / symptom diary
  - Subject to recall
  - “Hidden” ingredient(s) may be overlooked
- Physical exam: assess for other allergic and alternative disorders
- Identify general mechanism
  - Allergy vs intolerance
  - IgE vs non-IgE mediated



# Evaluation of Food Allergy

- Suspect IgE-mediated:
  - Panels/broad screening should NOT be done without supporting history because of high rate of false positives.
  - Skin prick tests (prick with fresh food if pollen-food syndrome)
  - *In vitro* tests for food-specific IgE
  - Oral food challenge
- Suspect non-IgE-mediated, consider:
  - Biopsy of gut, skin
- Suspect non-immune, consider referral for:
  - Hydrogen breath test
  - Sweat test
  - Endoscopy

Food Protein Induced Proctocolitis

Food Protein Induced Enterocolitis

Eosinophilic Esophagitis

# Food Protein Induced Proctocolitis

# Food Protein-Induced Allergic Proctocolitis (FPIAP)

- Painless rectal bleeding, usually during the first weeks/months of life.
- Breast-fed or formula fed infants.
- No weight loss. Thriving infants.
- Rectal bleeding resolves within 48-72 hours of food elimination.
- Usually resolves by age: 12 months.
- Food reintroduction at home.

# Clinical features of FPIAP (95 breast-fed infants)

Initial Presentation	Frequency (%)
Blood-tinged stools	100
Pain during defecation	22
Diarrhea/loose stools	4
Failure to thrive	0

Endoscopic findings	Frequency (%)
Focal erythema or erosions	100
Lymphoid nodular hyperplasia	48

Positive Response to dietary protein elimination	Frequency (%)
Cow's milk	65
Egg	19
Corn	6
Soy	3
Two of the above	5
Not identified	12
Response to L-amino acid formula only	4

# FPIAP Management

- Formula fed infants

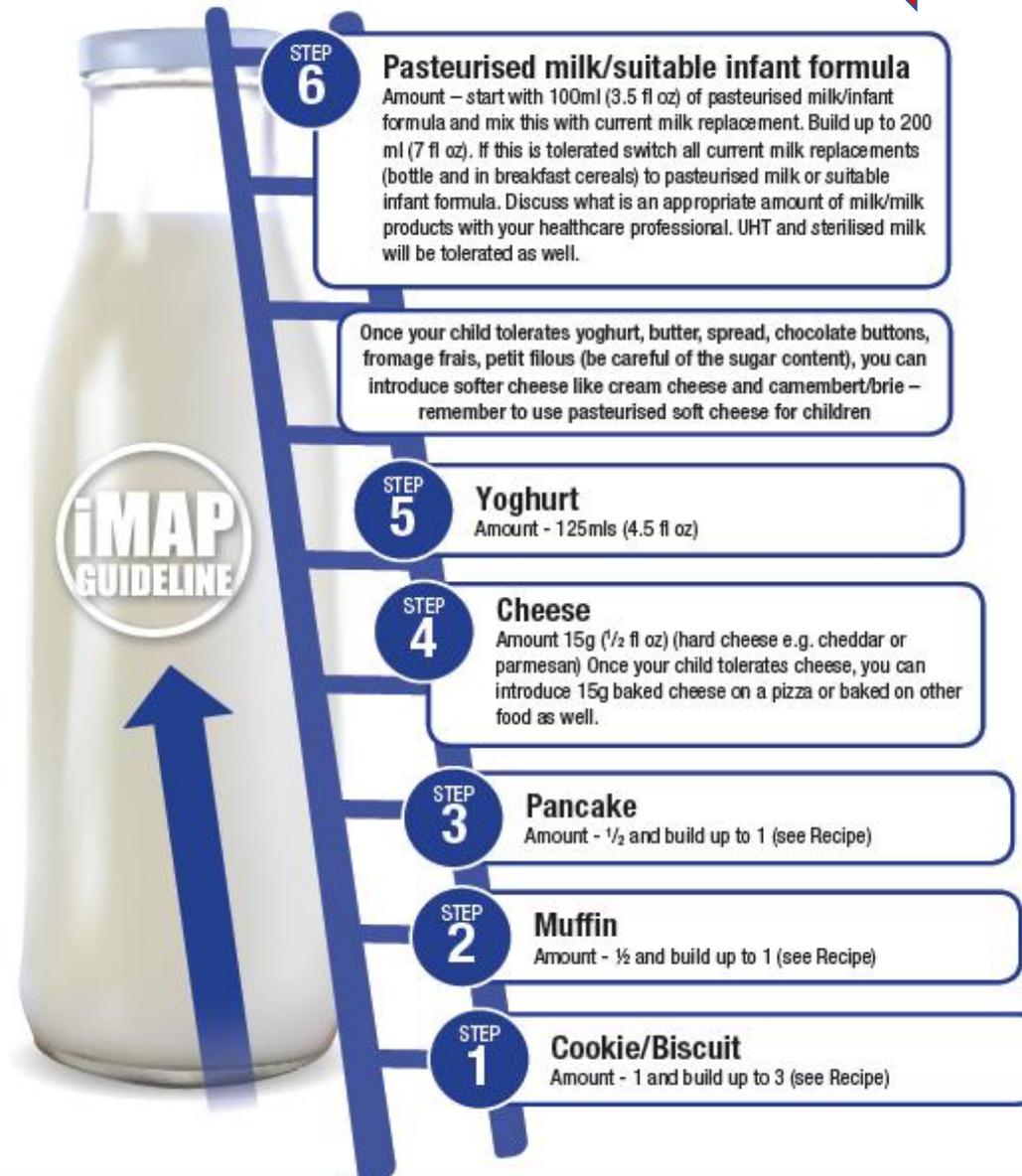
1. Alternative formula: partial hydrolyzed, elemental formula or amino acid formula.
2. Normal timing of solid food introduction.
3. Reintroduce food AT HOME (around 12 months).

- Breast-fed infants

1. Eliminate food trigger from maternal diet.
2. Can use hypoallergenic formula (partial hydrolyzed, elemental diet, amino acid).
3. Normal timing of solid food introduction.
4. Reintroduce food AT HOME (around 12 months).

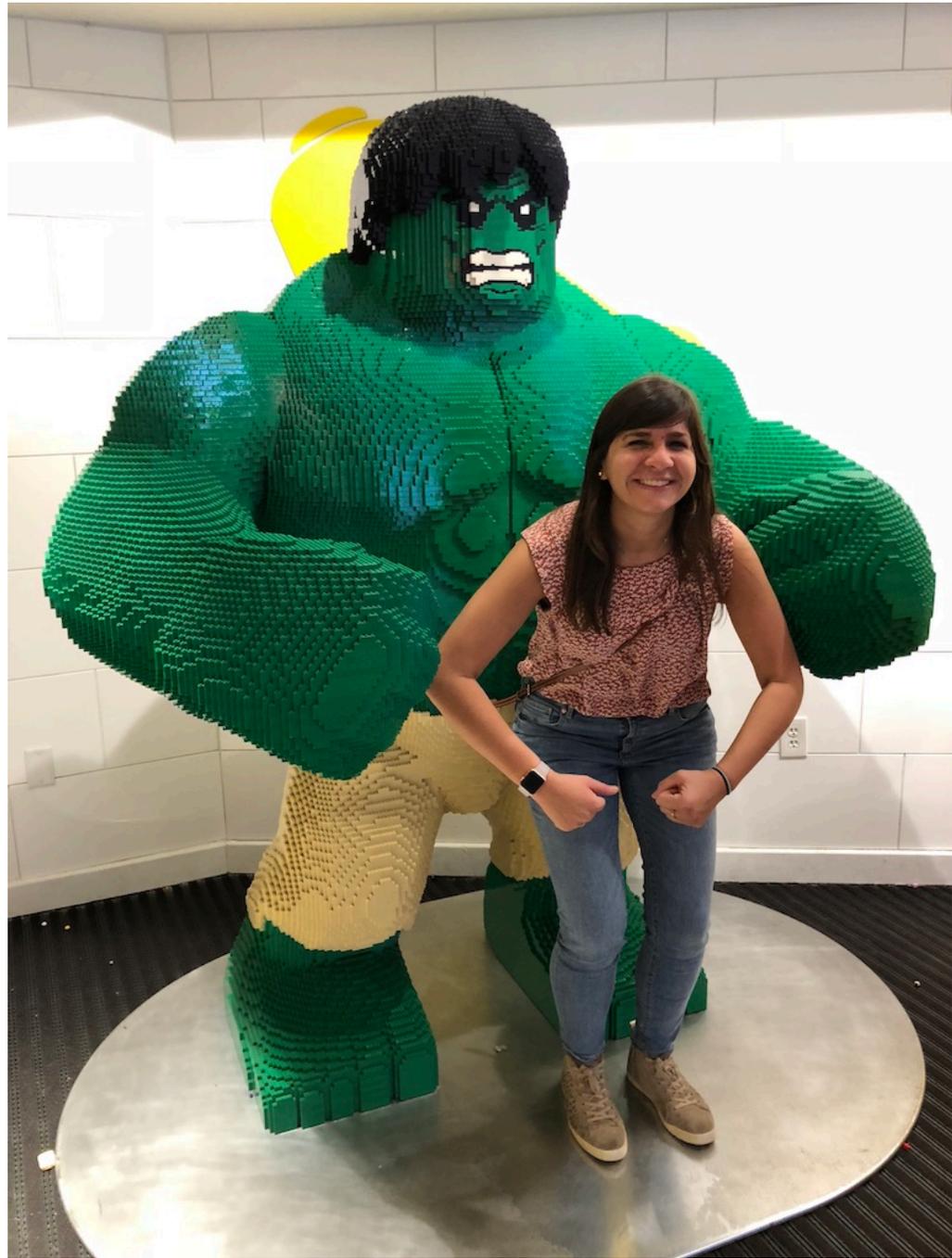
# THE iMAP MILK LADDER

To be used only in children with Mild to Moderate Non-IgE Cow's Milk Allergy  
Under the supervision of a healthcare professional  
PLEASE SEE THE ACCOMPANYING RECIPE INFORMATION



# Milk Ladder

- The Milk Ladder is an evidence-based guideline for the home reintroduction of milk and dairy products into the diets of infants and children with mild to moderate cow's milk allergy.
  - It is a plan to re-introduce milk products gradually and in stages, starting with foods that contain only a small amount of well-cooked milk and progressing towards un-cooked dairy products and fresh milk.
  - It is called the 'Milk Ladder' because completing each of the 6 steps in the plan is like climbing a rung on a ladder towards being able to fully tolerate milk and dairy products
- Who should use the Milk Ladder?
    - The Milk Ladder can only be followed by infants and children with a mild to moderate cow's milk allergy known as a 'non-IgE mediated' milk allergy.  
It is not suitable for children with a milk allergy that results in severe or immediate type allergic reactions known as an 'IgE mediated' milk allergy.



# Food Protein Induced Enterocolitis (FPIES)

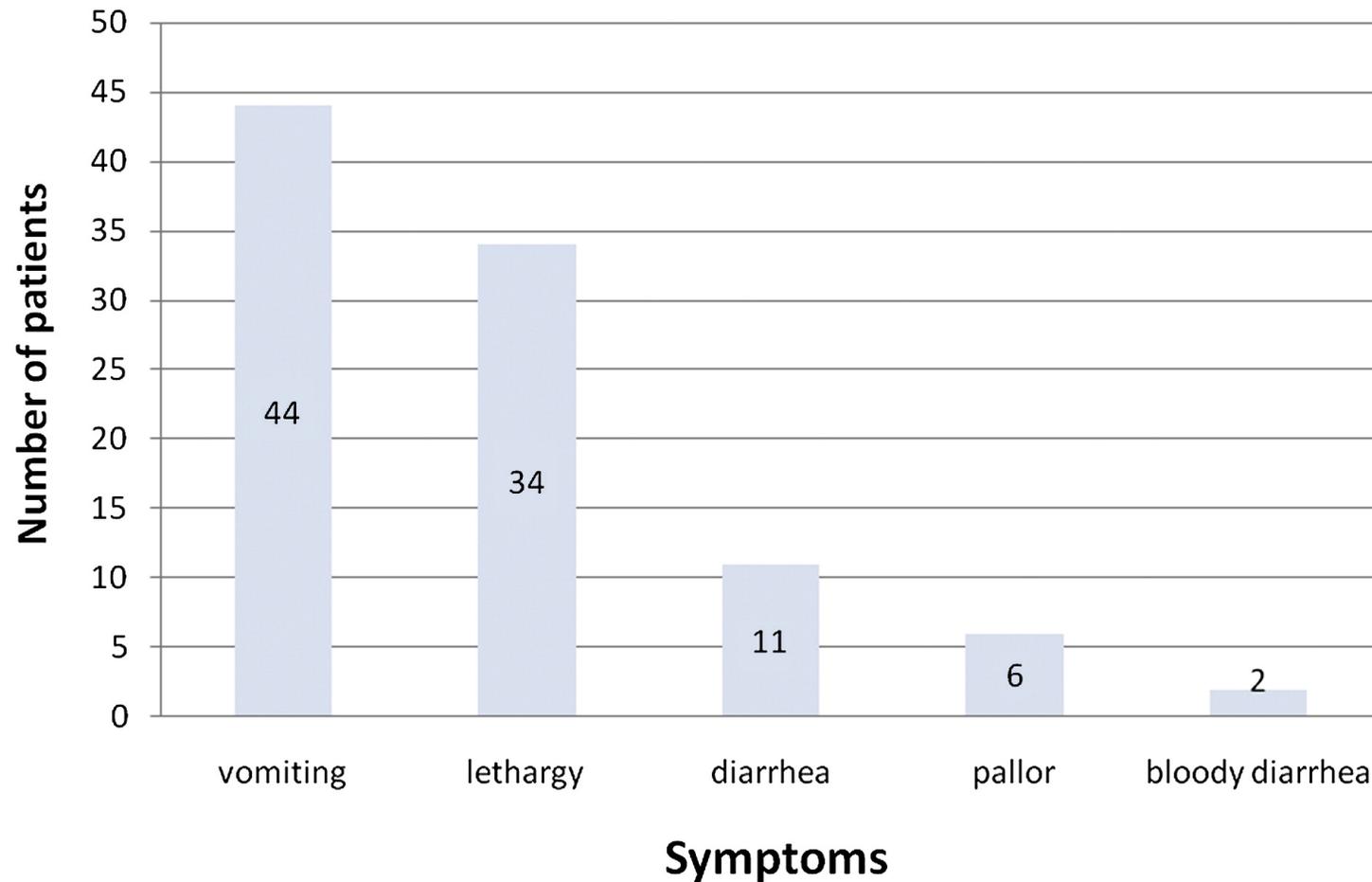
# FPIES

- Typical onset during the first year of life.
  - Reports of older children and adults (seafood: shrimp and fish).
- Symptoms can trigger severe dehydration, hypovolemia, metabolic acidosis and intubation. So far, no fatalities.
- Poorly recognized (acute FPIES can be confused with sepsis).
- Usually with negative IgE specific test
  - Atypical FPIES can have positive IgE
- No definite biomarkers and pathophysiology.
- Traumatic experience and high burden to family members
- Usually outgrown by age 3-5 years

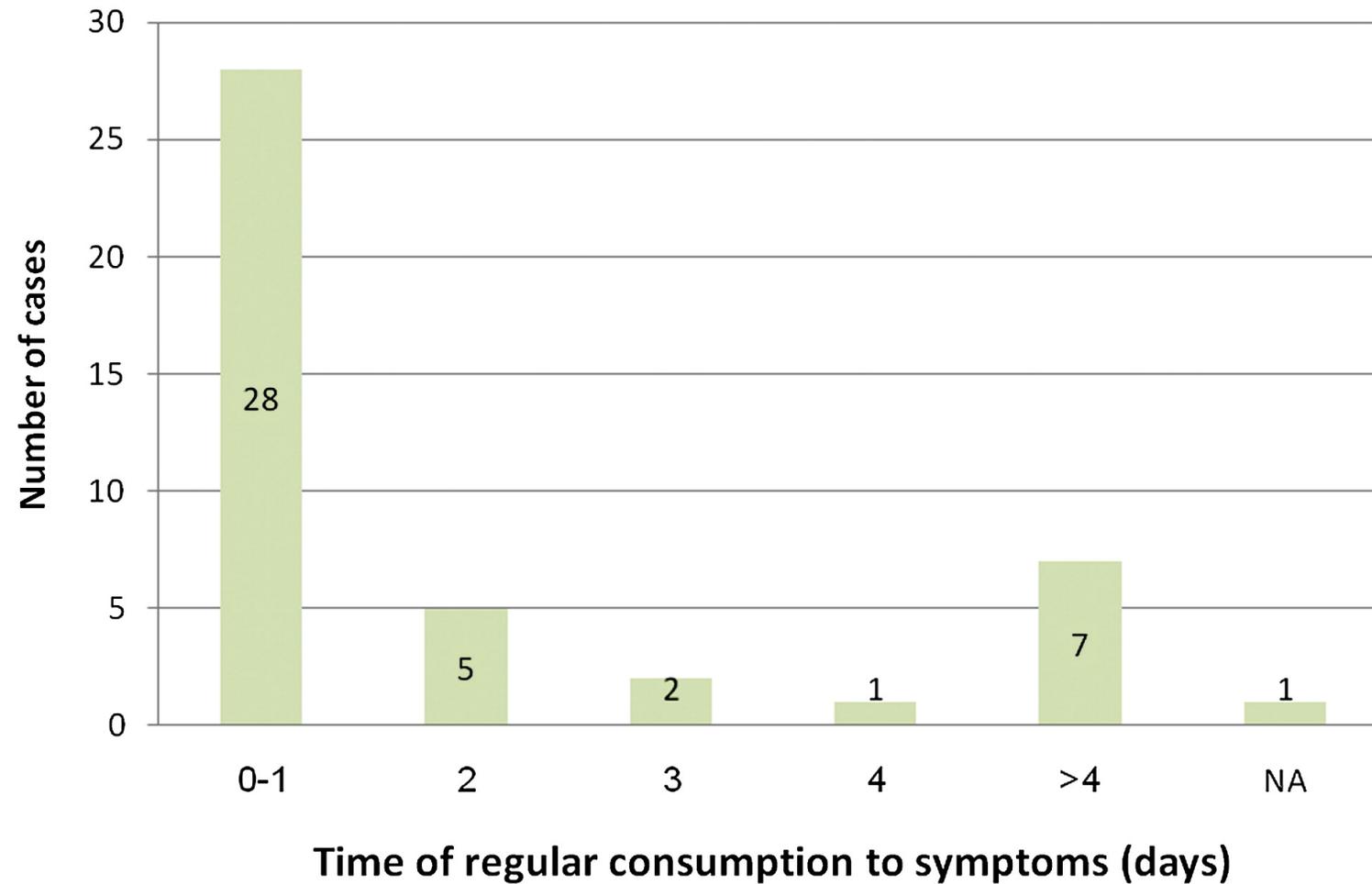
# FPIES prevalence in the United States

- A cross-sectional, population-based survey (10/2015 - 9/2016)
- 53,575 US households included (US population: 327.2 million)
- Primary outcome: lifetime prevalence of physician-diagnosed FPIES.
- Participants were asked “*Has your child **ever been diagnosed by a physician with food protein-induced enterocolitis syndrome (FPIES)?***”
- 375,000 Children with FPIES (0.51% of US Pediatric Population)
- 550,000 Adults with FPIES (0.22% of US Adult population)

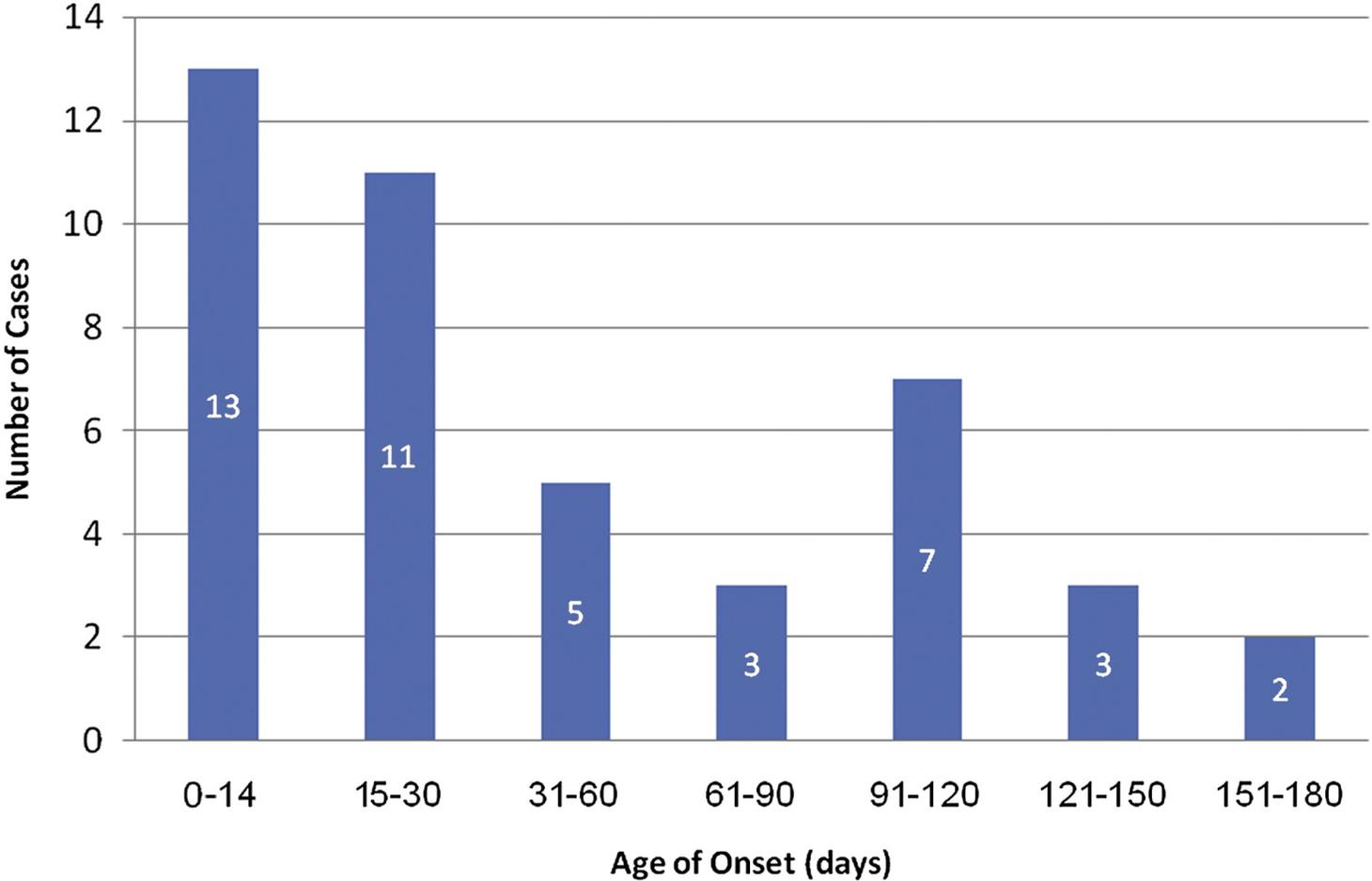
# Clinical characteristic of patients with FPIES



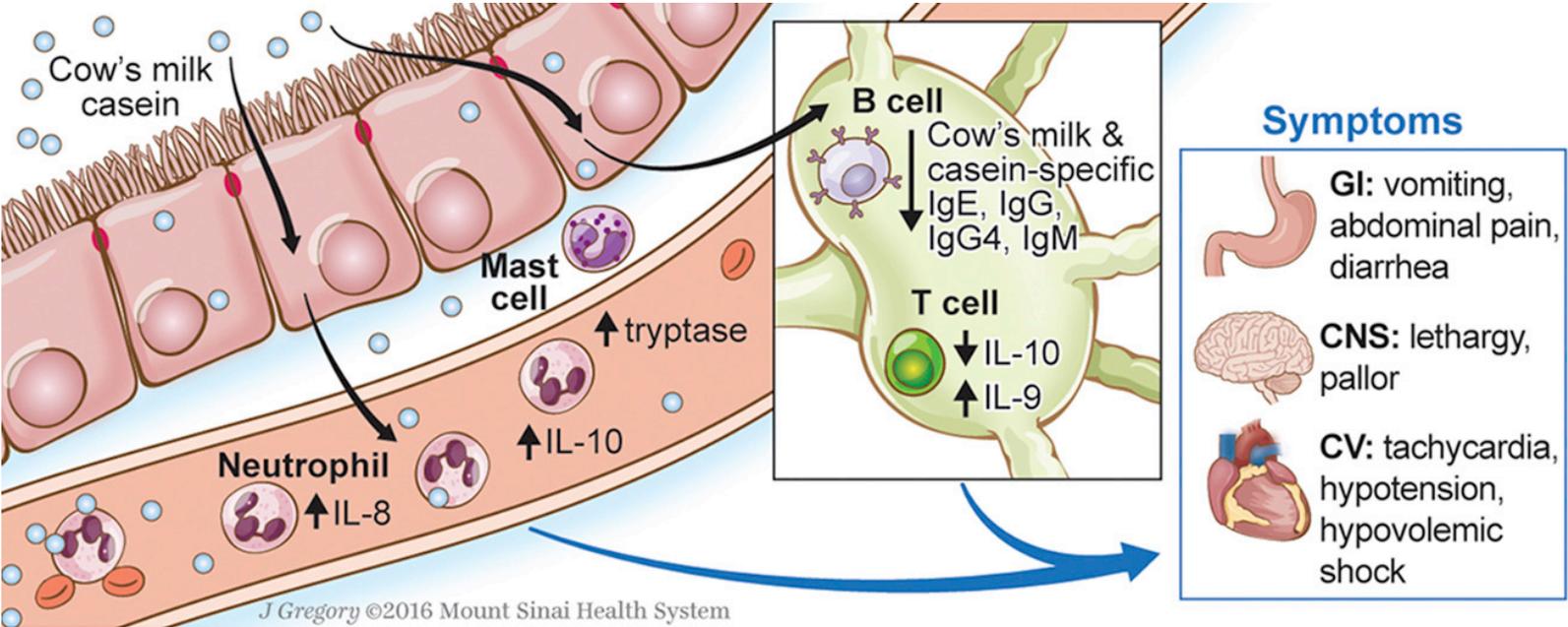
# Days of Consumption of Cow's Milk Protein before the development of symptoms



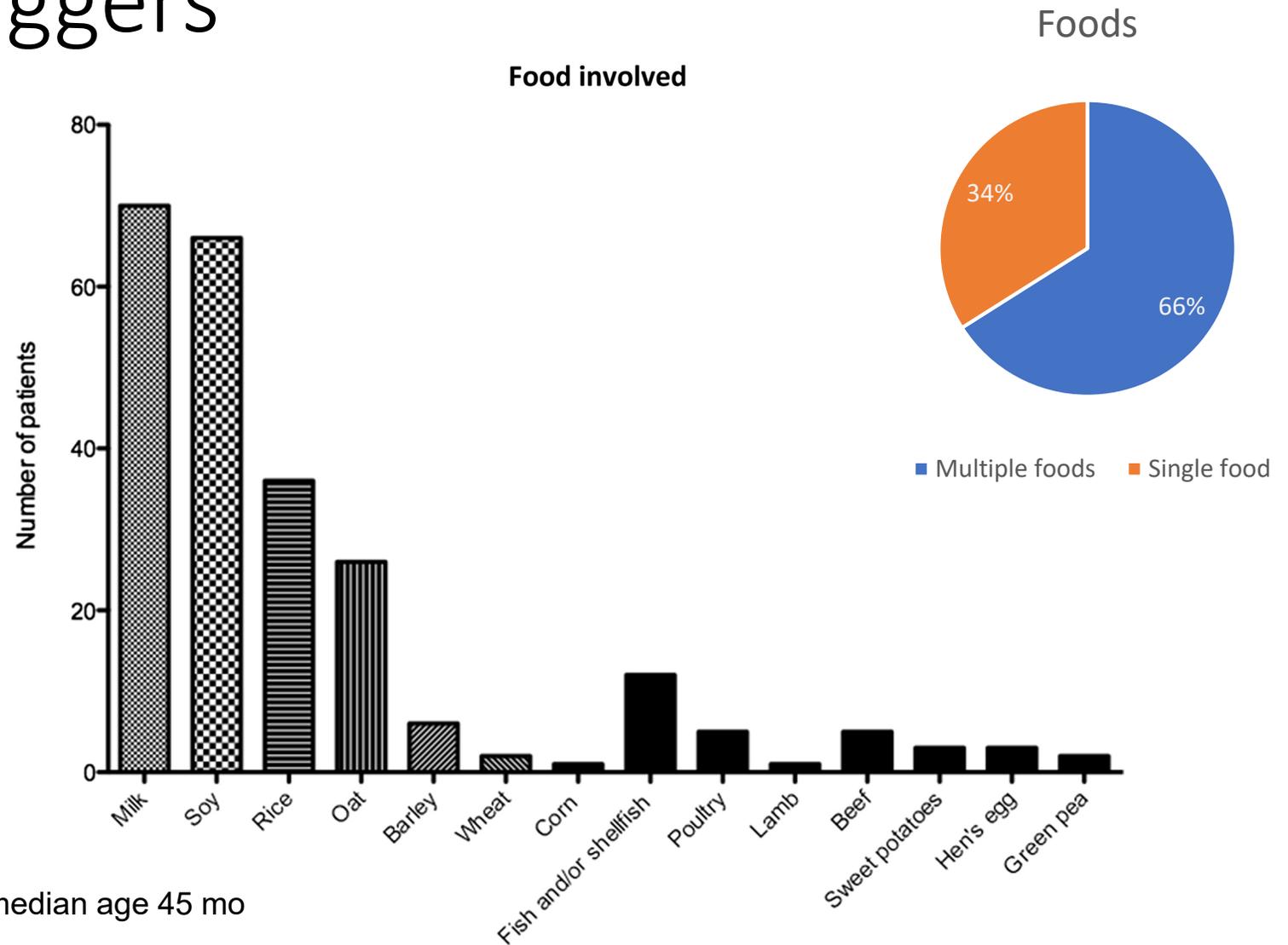
# Age distribution for the onset of FPIES.



*Humoral and cellular responses to casein in patients with food protein–induced enterocolitis to cow's milk*



# FPIES food triggers

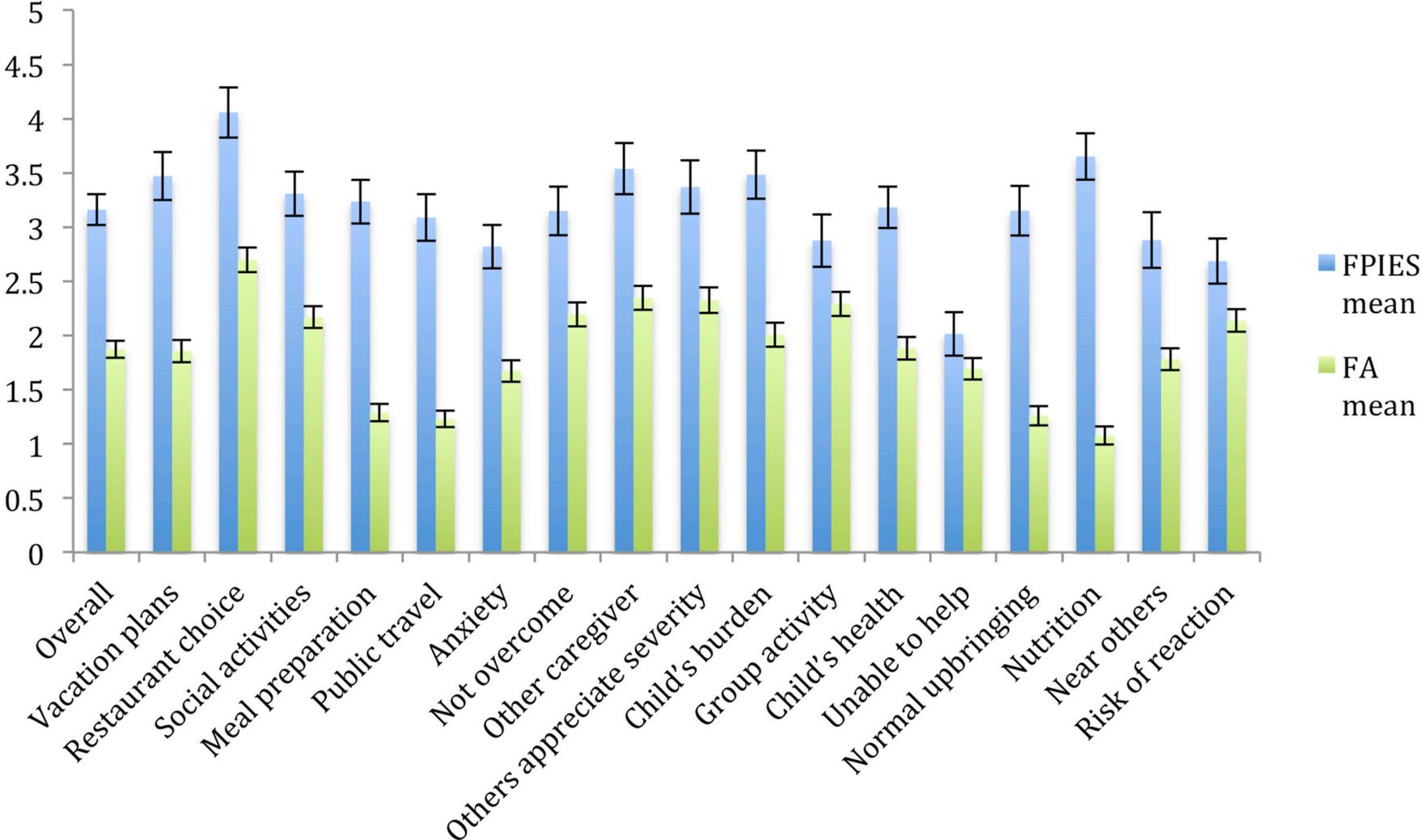


N=160, diagnosis confirmed by OFC in 30%, median age 45 mo

# FPIES in adolescent and adults

- Gleich et al; J Allergy Clinical Immunol Prac 2015 Nov
  - Thirty eight adults with allergy to shrimp
- Gonzales-Delgado P, et al JACI in Practice 2018
  - Spain: Fish, bivalves, cephalods, fish, and crustaceans.

*A validated index to measure health-related quality of life in patients with food protein–induced enterocolitis syndrome*



# FPIES

- Approximately 30 percent of infants with FPIES develop atopic diseases:
  - Atopic dermatitis (25 to 65 percent), asthma (3 to 20 percent), or allergic rhinitis (20 percent).
  - Thirty nine percent of the children with FPIES had concurrent sensitization (positive IgE test) to other foods.
- Family history of atopic diseases is present in 40 to 80 percent of patients, including a family history of food allergy in approximately 20 percent

# Clinical Phenotyping of FPIES (International Consensus Guidelines J Allergy Clin Immunol 2017;139:1111-26)

FPIES Subtypes	Defining features
Age of onset	
Early	Younger than age 9 months
Late	Older than age 9 months
Severity	
Mild to Moderate	Repetitive emesis with or without diarrhea, pallor, mild lethargy
Severe	Repetitive projectile emesis with or without diarrhea, pallor, lethargy, dehydration, hypotension, shock, methemoglobinemia, metabolic acidosis
Timing and duration of symptoms	
Acute	Occurs with intermittent food exposures, emesis starts usually within 1-4 h, accompanied by lethargy and pallor; diarrhea can follow within 24 hours, with usual onset of 5-10 h. Usual resolution of symptoms within 24 h after elimination of the food from diet. Growth is normal, and child is asymptomatic during food trigger elimination
Chronic	Occurs with daily ingestion of the food (eg, feeding with CM- or soy-based formula in an infant); symptoms include intermittent emesis, chronic diarrhea, poor weight gain, or FTT. Infants with chronic FPIES usually return to their usual state of health within 3-10 d of switching to a hypoallergenic formula, although in severe cases temporary bowel rest and IV fluids might be necessary. Subsequent feeding of the offending food after a period of avoidance results in acute symptoms
IgE positivity	
Classic	Food specific, IgE negative
Atypical	Food specific, IgE positive

# Diagnostic Criteria for Acute FPIES

**1** Major criterion



**≥3** Minor criteria

Vomiting in 1-4 hrs  
& absence of skin  
and respiratory sx

>1 episode to the same food

Repetitive emesis to another food

Lethargy

Pallor

Emergency room visit

Need for intravenous fluids

Diarrhea in 24 hours (5-10 hours)

Hypotension

Hypothermia

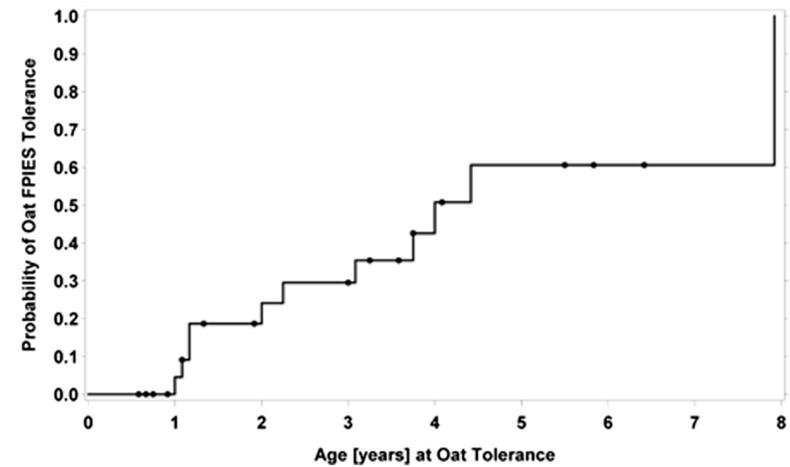
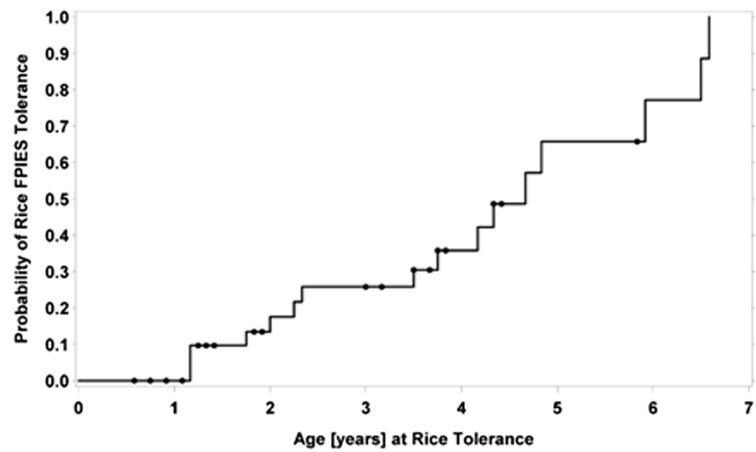
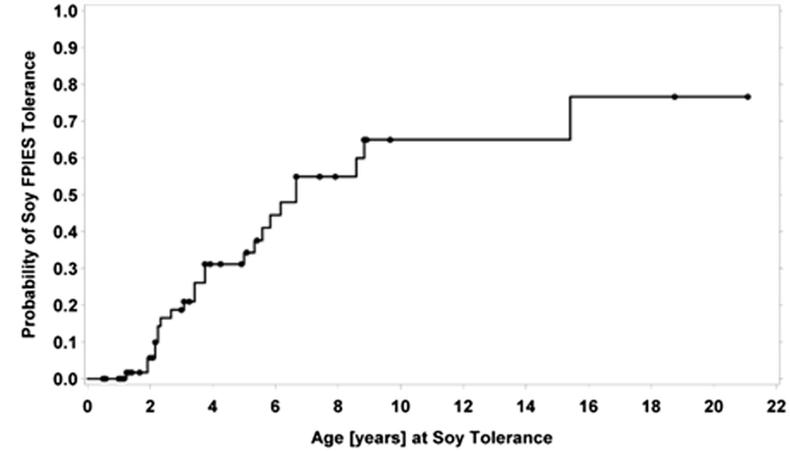
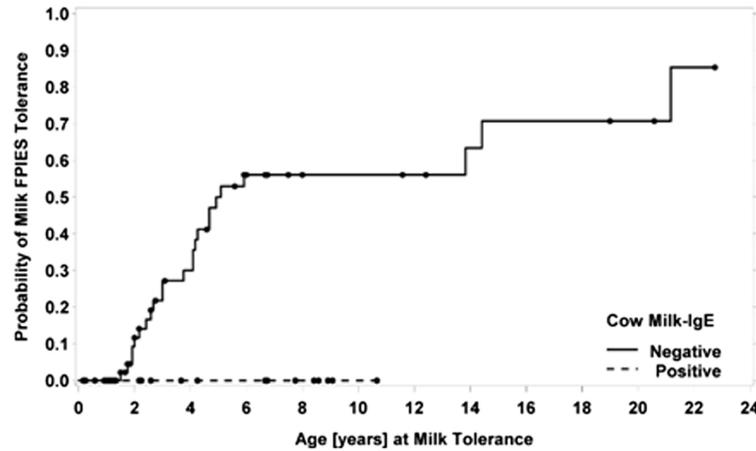
# Chronic FPIES

- Onset: first 1-3 months of life
- Usually to milk and soy
- Watery diarrhea, mucous, and sometimes bloody stools
- Intermittent emesis
- Failure to thrive
- Labs: low albumin and total protein, anemia, elevated WBC/neutrophils, and platelets
- Clinical presentation: dehydration, hypovolemic shock, and metabolic acidosis
- Symptom resolution after stopping the offending food: days to weeks
- Subsequent feeding of the offending food after a period of avoidance results in acute symptoms

# Atypical FPIES

- Some patients with FPIES have mixed immunologic reactions to the food(s) causing their FPIES symptoms.
- Up to 25 percent of infants and children fulfilling the clinical diagnostic criteria for FPIES have or develop IgE antibodies to the trigger food.
- 1 in 3 progress to immediate IgE- CMA
- In one study, for example, 7 of 17 children with milk-induced FPIES who developed milk IgE positivity progressed to immediate-allergic reactions ranging from mild symptoms to anaphylaxis.
- They tend to have a more protracted course of FPIES and have the potential for developing symptoms of IgE-mediated allergy (eg, anaphylaxis) in addition to FPIES.
- 2/3 remain FPIES.

# Clinical features and resolution of food protein–induced enterocolitis syndrome: 10-year experience



# FPIES Challenge Protocol

- Under physician supervision (inpatient/outpatient)
- High risk
- Food challenge:
  - Gradual over 30 minutes: 3 equal doses or a single dose of food protein: 0.06 - 0.6 g/kg (no more than 3 grams of food protein).
- Fifty percent of positive challenges will require fluid resuscitation.
  - IV access
- If no reaction:
  - Discharge after 4-6 hours.
  - Post-reaction in 6 hours and tolerating PO.

# Interpretation of FPIES Challenge

- Major Criteria

- One major criteria

- Vomiting in 1-4 hours and absence of skin and respiratory symptoms

- Minor Criteria

- Two major criteria

- Pallor
    - Diarrhea
    - Hypotension
    - Lethargy
    - Hypothermia
    - Increase absolute neutrophil count (>1,500)

# FPIES Management

- Avoid food trigger
  - No need for avoidance of traces or foods with “may contain” labels
  - Avoid baked milk/egg
  - Don’t restrict maternal diet unless the infant is symptomatic
  - CM/soy FPIES: hypoallergenic diet: elemental formula or amino acid formula (40%)
- Timely introduction of solids
- Periodic food challenges (every 12-24 months)
- Food emergency plan
  - Manage FPIES emergencies
- Dietary Nutrition Consult
- Treat acute episodes

# Empiric guidelines for selecting weaning foods in infants with FPIES

Ages and Stages	Lower Risk Foods	Moderate Risk foods	High Risk Foods
<p><b>4-6 months (as per AAP, CoN)</b>            If developmentally appropriate and safe and nutritious foods are available:            Begin with smooth, thin purees and progress to thicker purees            Choose foods that are high in iron            Add vegetables and fruits</p>	<p>Vegetables            Broccoli cauliflower, parsnip, turnip, pumpkin</p>	<p>Squash, carrot, white potato, green bean (legume)</p>	<p>Sweet potato, green pea (legume)</p>
<p><b>6 months (as per WHO)</b>            Complementary feeding should begin no later that 6 months of age:            In the BF infant, high iron foods or supplemental iron (1 mg/kg/d) are suggested by 6 months of age            Continue to expand variety of fruits, vegetables, legumes, grains, and other foods as tolerated</p>	<p>Fruits            Blueberries, strawberries, plum, watermelon, peach, avocado</p>	<p>Apple, pear, orange</p>	<p>Banana</p>
<p><b>8 months of age or when developmentally appropriate</b>            Offer soft cooked and bite and dissolve textures from around 8 m of age or as tolerated</p>	<p>High Iron foods            Lamb, fortified quinoa cereal, millet</p>	<p>Beef, fortified grits and corn cereal, wheat (whole wheat and fortified), fortified barley ceereal</p>	<p>Higher iron foods: fortified infant rice and oat cereals</p>
<p><b>12 months of age or when developmentally appropriate</b>            Offer modified tolerated foods from the family table            chopped meats, soft cooked vegetables, grain and fruits</p>	<p>Other            Tree nuts and seed butters (sesame, sunflower, etc)            Thinned with water or infant puree for appropriate infant texture and to prevent choking</p>	<p>Peanut, other legumes (other that green pea)</p>	<p>Milk, soy, poultry, egg, fish</p>

# Acute Management FPIES

1. Place a peripheral intravenous line and administer normal saline bolus, 20 mL/kg rapidly; repeat as needed to correct hypotension
2. If age 6 mo and older: administer intravenous ondansetron, 0.15 mg/kg/dose; maximum, 16 mg/dose
3. If placement of intravenous line is delayed because of difficult access and age is 6 mo or older, administer ondansetron intramuscular, 0.15 mg/kg/dose; maximum, 16 mg/dose
4. Consider administering intravenous methylprednisolone, 1 mg/kg; maximum 60-80 mg/dose
5. Monitor and correct acid base and electrolyte abnormalities
6. Correct methemoglobinemia, if present
7. Monitor vital signs
8. Discharge after 4-6 h from the onset of a reaction when the patient is back to baseline and is tolerating oral fluids
9. Transfer the patient to the emergency department or intensive care unit for further management in case of persistent or severe hypotension, shock, extreme lethargy, respiratory distress

**TABLE I.** Comparison of FPIES, FPIAP, and FPE

	FPIES	FPIAP	FPE
Age at onset	Dependent on age of exposure to antigen; usually 1 d to 1 y; might be older in case of solid foods, such as chicken, eggs, and seafood	Days to 6 mo, usually 1-4 wk; later onset in older children has been reported to CM, egg, and wheat	Dependent on age of exposure to antigen; CM and soy up to 2 y
Food proteins implicated			
Less common	CM, soy, rice, oat, egg	CM, soy	CM, soy
Most common	Multiple other food proteins have been described	Wheat, egg	Wheat, egg
React to $\geq 2$ different foods	Up to 35%; in the United States up to 40% react to both CM and soy	Up to 20% might react to CM and soy or multiple foods	Rare
Transition to IgE positivity	Up to 35 %, especially in patients with CM-induced FPIES	None reported in infants; in older children up to 19% have detectable CM-specific IgE	None reported
Feeding at the time of onset	Formula or breast milk in solid food-induced FPIES	Breast milk or CM or soy formula	CM or soy formula
Atopic background	Variable	Variable	Variable
Family history of atopy	40% to 70%	Up to 25%	Unknown
Personal history of atopy	Up to 30%	Up to 20%	22%
Symptoms			
Emesis	Prominent, repetitive	Absent	Intermittent
Diarrhea	Severe in patients with chronic FPIES	Mild	Moderate
Bloody stools	Severe in patients with chronic FPIES	Prominent	Rare
Edema	Severe in patients with chronic FPIES	Mild, infrequent	Moderate
Shock	15%	Absent	Absent
FTT	Moderate-to-severe in patients with chronic FPIES	Absent	Moderate
Hypothermia	Present (<25%)	Absent	Absent
Laboratory findings			
Anemia	Moderate	Mild, infrequent	Moderate
Hypoalbuminemia	Acute	Mild, infrequent	Moderate
Methemoglobinemia	Might be present	Absent	Absent
Acidemia	Might be present	Absent	Absent
Malabsorption <sup>†</sup>	Absent	Absent	Present
Leukocytosis with neutrophilia	Prominent	Absent	Absent
Thrombocytosis	Moderate	Mild	Absent
Allergy evaluation			
Food skin prick test*	Can be positive in 4% to 30%	Negative	Negative
Serum food allergen IgE*	Can be positive in 4% to 30%	Negative	Negative
Total IgE	Normal or increased	Normal or increased	Normal
Peripheral blood eosinophilia	Absent	Occasional	Absent
Biopsy findings in infants with chronic symptoms			
Villous atrophy	Patchy, variable	Absent*	Variable <sup>†</sup>
Colitis	Prominent; rectal ulceration	Focal	Absent
Mucosal erosions	Occasional	Occasional, linear	Absent
LNH	Absent	Common	Duodenum and colon
Eosinophils	Prominent; cryptal abscesses	Prominent; cryptal abscesses; >60 eosinophils per 10 high-power fields in the lamina propria strongly suggest FPIAP	Few Increased IEL numbers
Supervised OFC	Vomiting, lethargy, pallor in 1-3 h; diarrhea in 5-8 h	Usually not necessary; visible or occult fecal blood in 12 h to several days	Usually not necessary; Vomiting and/or diarrhea in 40-72 h
Treatment	Food elimination; symptoms clear within hours in patients with acute FPIES and in 3-10 d in patients with chronic FPIES; 80% respond to hydrolysate; soy formula can be introduced under supervision; rechallenge in 12-24 mo	Food elimination from the maternal diet or hypoallergenic formula, about 10% might need elemental formula Food reintroduction after 12 mo	Food elimination, symptoms clear in 1-3 wk, rechallenge and biopsy in 1-2 y
Natural history	Varies by population, CM tends to resolve by age 3-5 y; rice-induced FPIES, 50% outgrow by age 5 y	Majority resolve by age 12 mo	Most cases resolve in 24-36 mo



# Eosinophilic Esophagitis

# Esophageal Eosinophilic Esophagitis

- EoE was first described by Dobbins et al in 1977 in a patient with eosinophilic gastroenteritis and a normal pH study. (*Gastroenterology* 1977;72(6):1312-6).
- Landres et al in 1978 reported the first case of eosinophilic infiltration isolated to the esophagus with associated esophageal dysfunction (*Gastroenterology* 1978;74(6):1298-301)

# Esophageal Esophagitis

- In 1993, Attwood et al retrospectively compared patients with esophageal eosinophilia who had normal and abnormal 24-hour pH studies (*Dig Dis Sci 1993;38(1):109-16*)
- In 1995, Kelly et al reported evidence that food proteins were the cause of non-GERD trigger of esophageal eosinophilia by using amino acid based elemental diet (*Gastroenterology 1995;109(1)1503-12*)

# EoE

(American College of Gastroenterology)

- Symptoms related to esophageal dysfunction.
- Eosinophil predominant inflammation of esophageal biopsy (>15 eosinophils per high power field)
- Secondary causes of esophageal eosinophilia have been excluded.
- A response to treatment (dietary elimination; topical glucocorticoids) supports the diagnosis but is not required.

# Clinicopathologic Diagnosis

- Pathology: Peak eosinophils  $\geq 15$  per hpf
- Typical Histologic Features
  - Basal Zone Hyperplasia
  - Pan-Esophagitis
  - Residual Eosinophilia on Proton Pump Inhibitors
- Multiple biopsy specimens are recommended

# Eosinophilic Esophagitis

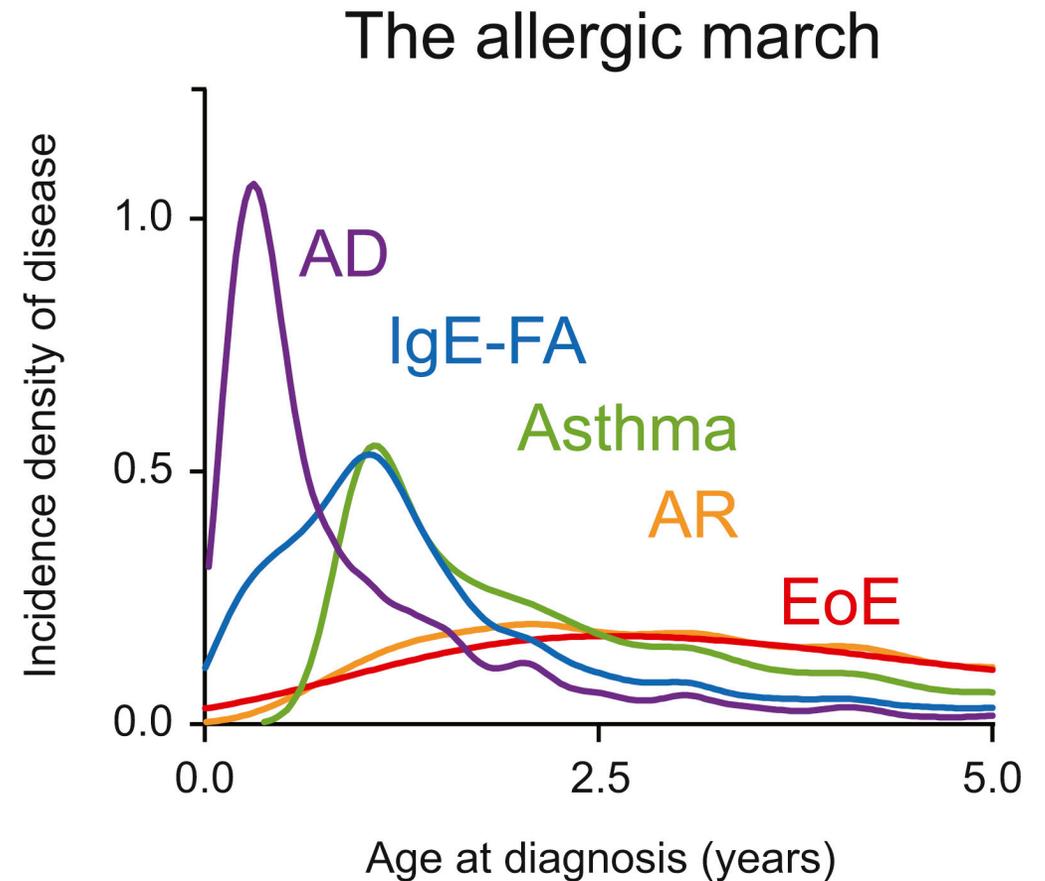
- EoE is global
  - Mostly in North and South America, Western and Eastern Europe, and Australia.
  - Fewer cases in Asia and the Middle East
  - No case reported in India or Sub-Saharan Africa.
- Incidence: 1 new case per 10,000 per year.
- Prevalence increase until 35-45 years of age then decreases.

# Epidemiology

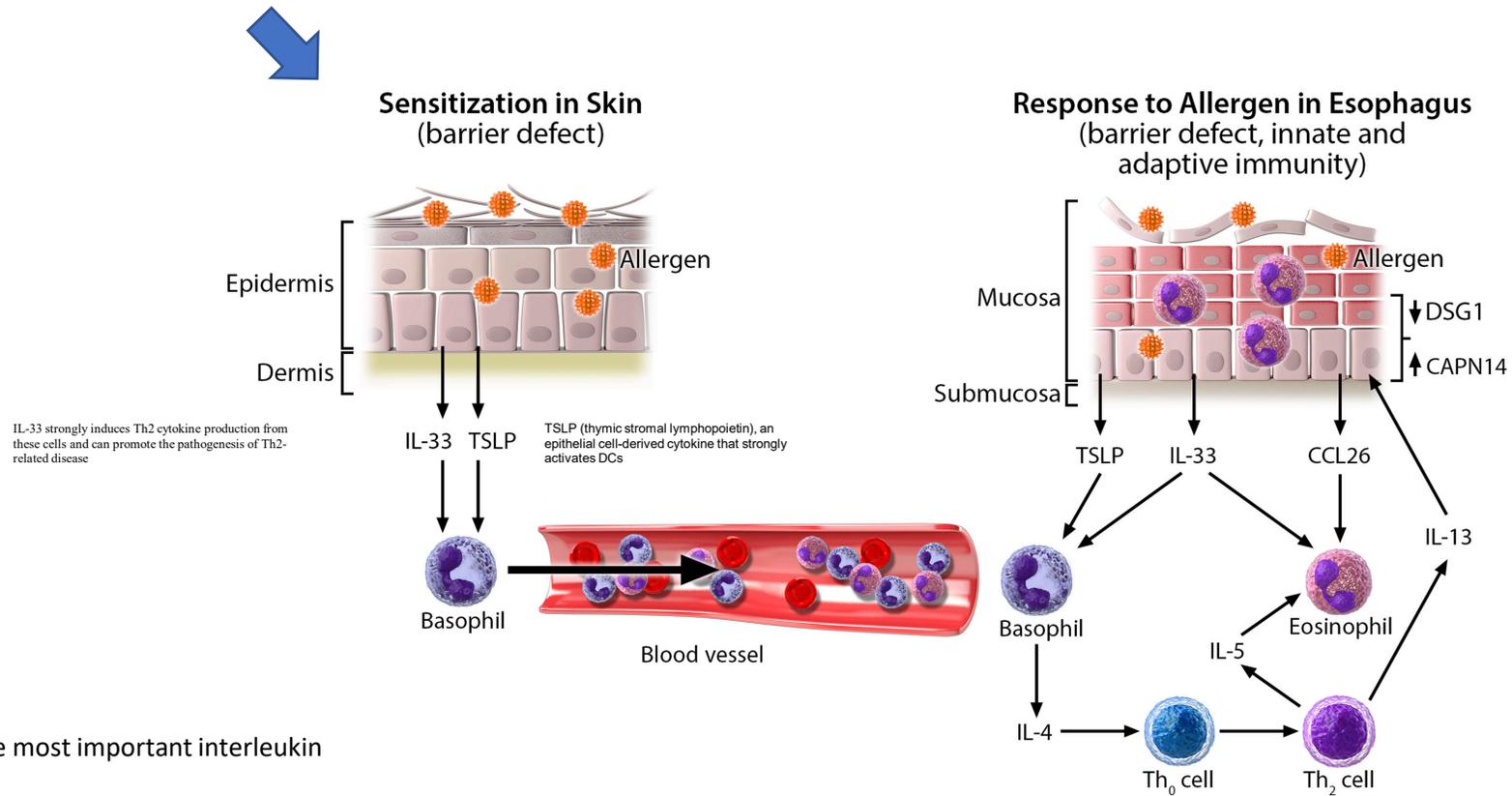
- **Male predominance** (different from asthma)
- Some patients can have a familial form in 5-10% of patients.
- Seasonal variations.
- Evidence of seasonal variability.
  - Varies by climate zone (more EoE in arid and cold climates).

# Clinical Features

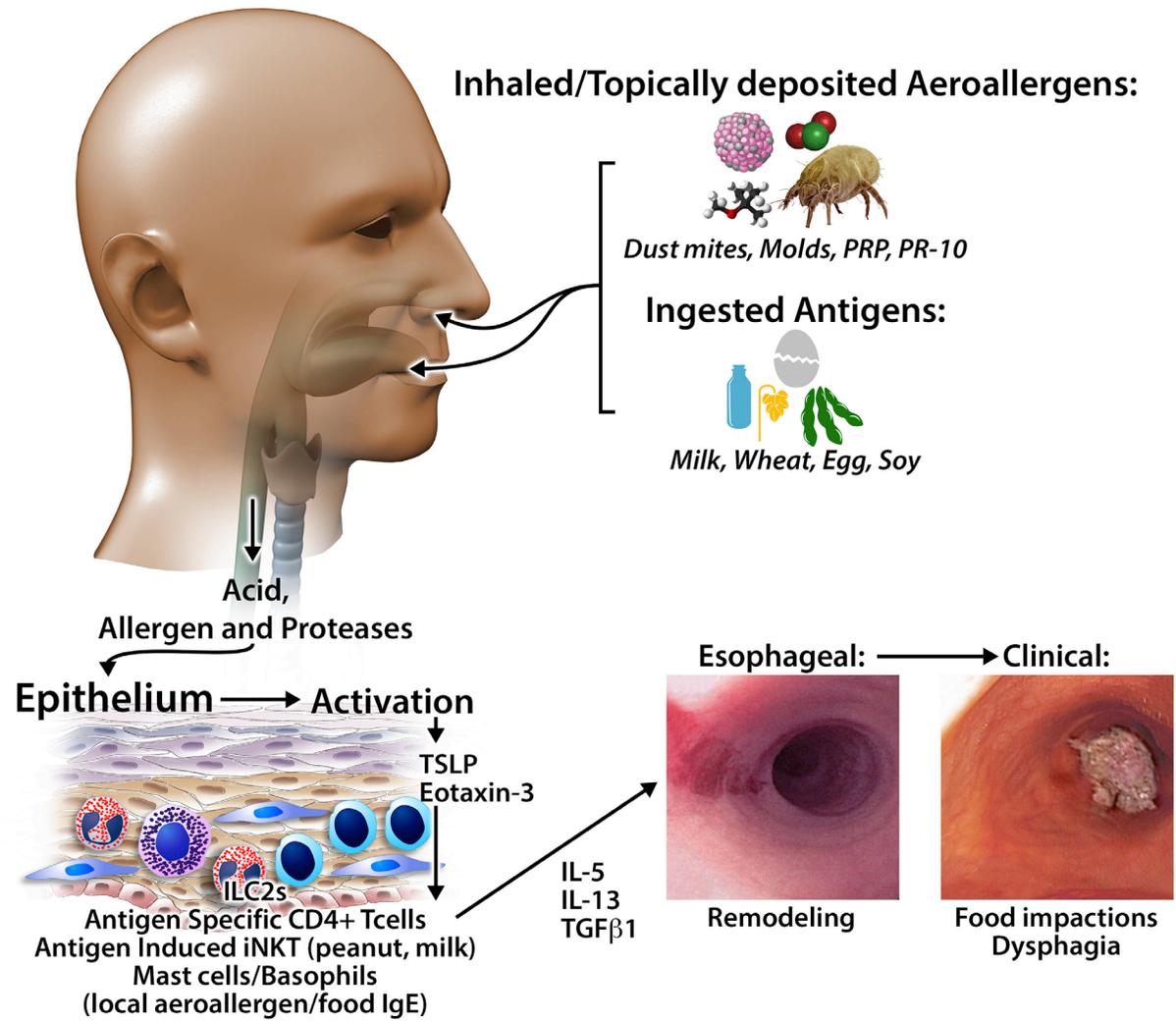
- EoE is associated with atopic diseases:
  - Up to 70% of patients have asthma
  - Four to seventy five percent have allergic rhinitis
  - Five to forty three percent have food allergies with anaphylaxis
  - Eighty percent have sensitization to foods.



Impaired skin barrier integrity, such as that caused by injury or *FLG* mutations, allows for increased allergen penetration through the epithelium, causing allergic sensitization and release of IL-33 and TSLP from the epithelium



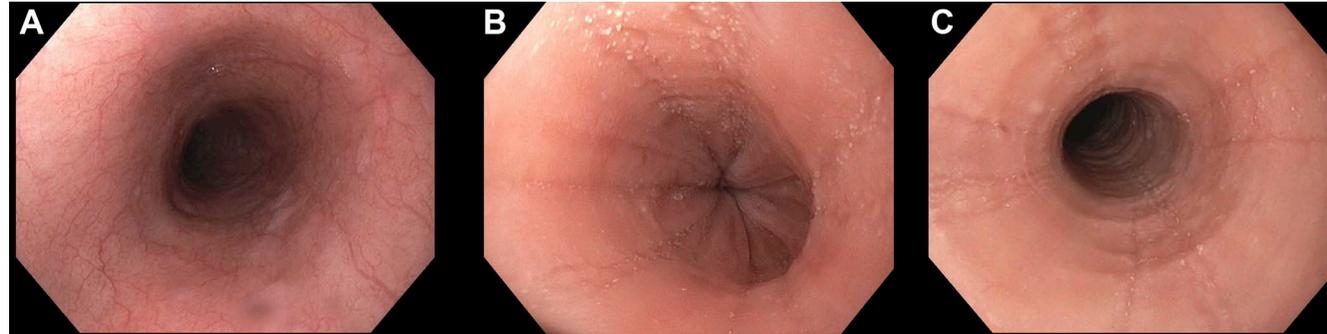
- Interleukin (IL)-13 is the most important interleukin involved.
  - IL-13: increases production of eotaxin-3 and fibroblast induce periostin.
- IL-5 is required (Th-2 response).
- Eosinophils
  - Produces TGF- $\beta$ 1: increases gene expression of collage and fibronectin in the esophagus.



# EoE Clinical Presentation

- Infants and toddlers
  - Difficulty feeding (gagging, choking, refusal of food, and vomiting)
- Children
  - Vomiting, regurgitation, nausea, epigastric or abdominal pain, chest pain, water brash, globus, or decreased appetite.
- Adults
  - Solid food dysphagia (most common)
  - Heartburn, chest pain, drinking copious amount of water after each bite, crushing or avoiding pills, etc.
  - Adults with food impaction: 46-63% have EoE.

A patient with edema, exudates, and furrows.



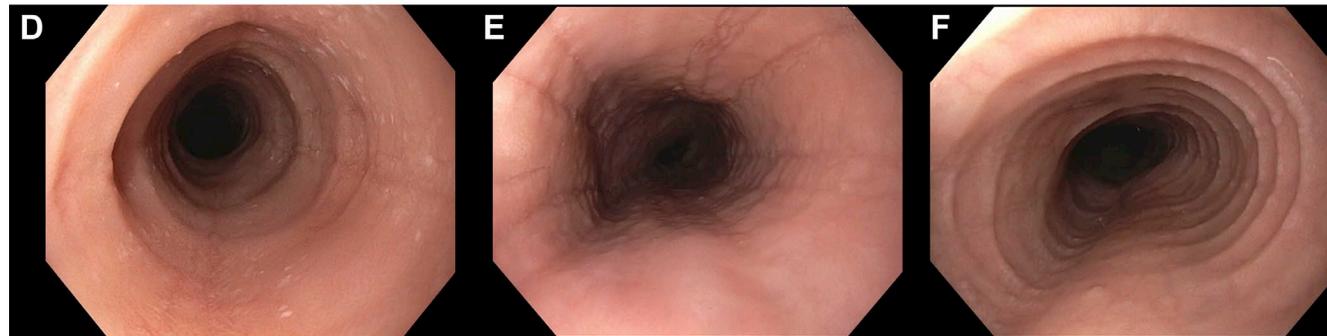
E0 R0 E0 F0 S0

E1 R0 E2 F1 S0

E1 R1 E1 F2 S1

A patient with suspected EoE, but with a normal endoscopy.

A patient with edema, rings, exudates, furrows, and a stricture.



E1 R2 E2 F1 S1

E1 R0 E0 F2 S0

E1 R2 E1 F1 S0

A patient with edema, rings, exudates, furrows, and a stricture

A patient with edema, rings, exudates, and furrows

A patient with edema and furrows

# EoE: foods

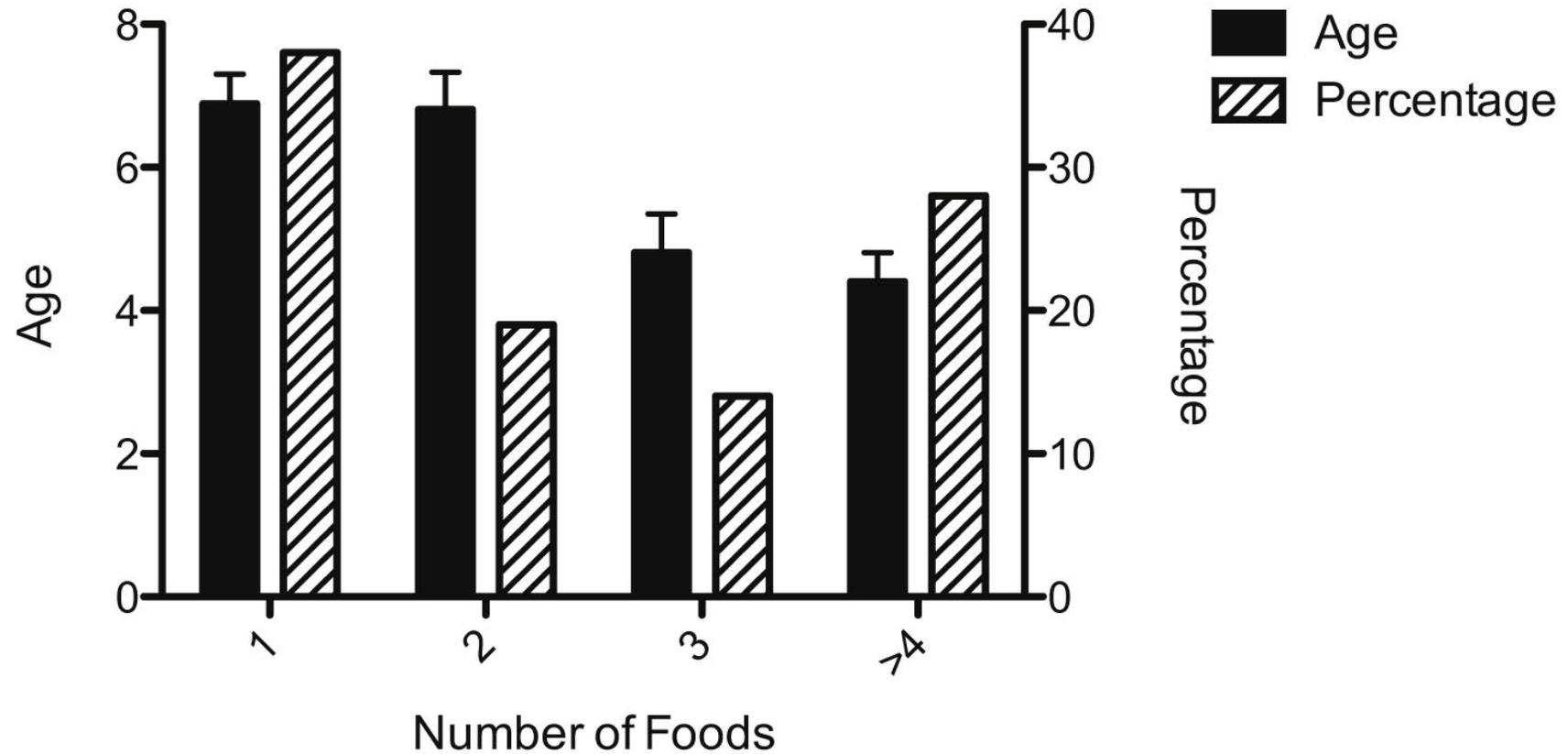
**TABLE II.** Most common food combinations in patients with EoE

<b>Food</b>	<b>No. of subjects</b>
Milk	78
Milk, meats*	24
Milk, egg, wheat, soy	20
Milk, soy	15
Grains*	13
Milk, egg, wheat, meats	11
Egg, wheat	10
Milk, egg	8
Milk, egg, wheat	8
Egg	8
Soy	7
Wheat	5

Food combinations were identified based on biopsy results. Each food is identified by a change in esophageal biopsy results from normal (0 eosinophils/hpf) to greater than 15 eosinophils/hpf when 1 food was added. Milk was seen in 7, egg in 13, wheat in 11, and soy in 10 other food combinations, respectively.

\**Grains*, Rice, oat, or wheat; *meats*, beef, chicken, turkey, or pork.

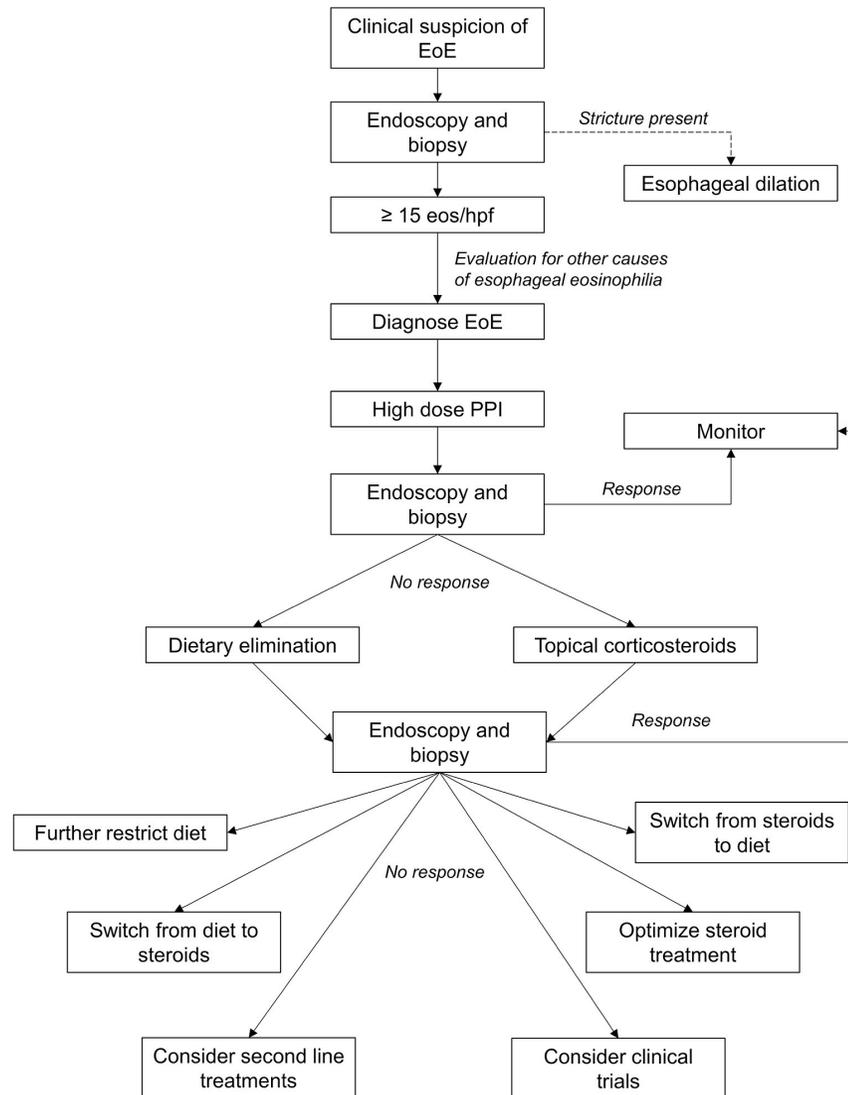
# EoE and Food Allergies



# EoE

- Role of aeroallergens:
  - Case reports documented spontaneous recurrence and remission of EoE during pollen season.<sup>1</sup>
  - One case report of a pediatric patient with DM sensitization and difficult EoE that was treated with AIT.<sup>2</sup>
  - Remember that EoE patients have a higher rates of aeroallergen sensitization than the general population.

# Algorithm for the diagnosis and management of eosinophilic esophagitis (EoE).



# EoE Treatment Options

EoE Diagnosis and Educate family on options

Dilation

Pharmacology Therapy

Nutrition  
Therapy

Topical/Systemic  
Steroids

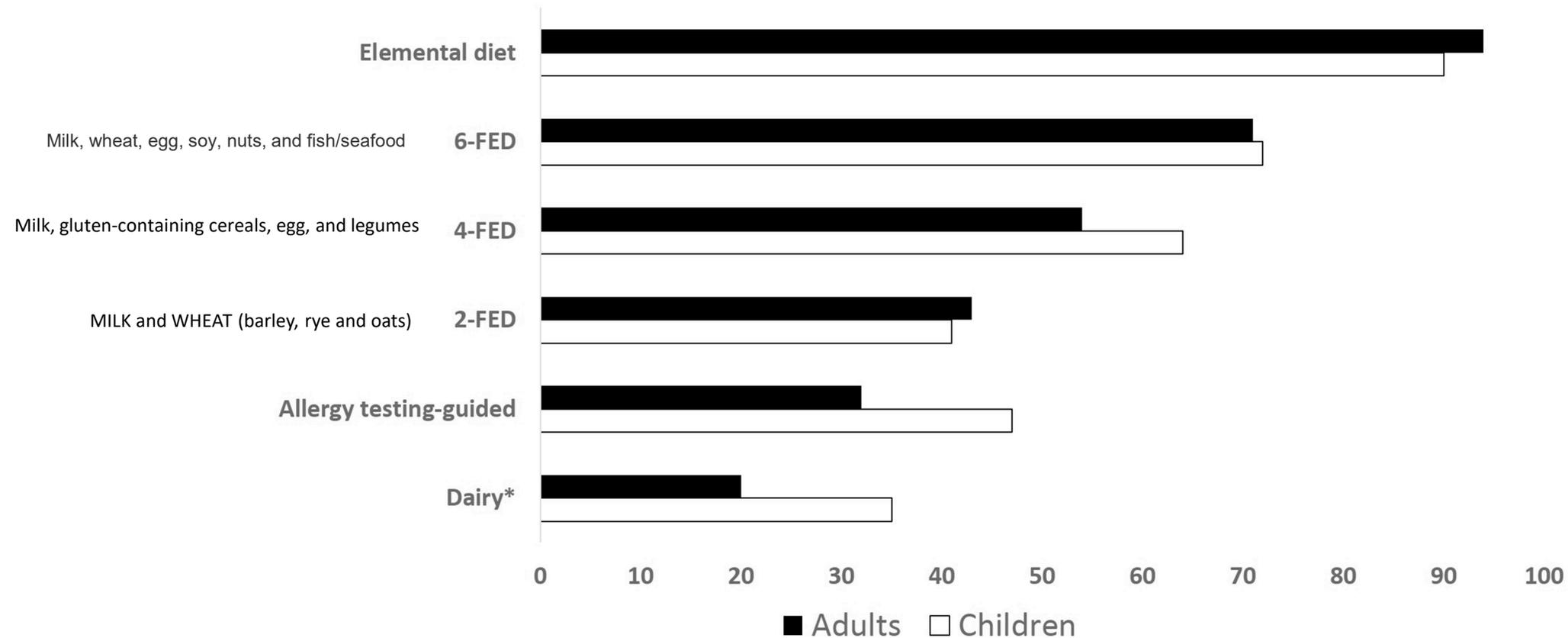
Biological

Combination of  
Elimination Diet  
and Topical  
Steroid

Elimination diet

Elemental Diet

# Histologic Remission Rates by diets



Least allergenic

Most allergenic

(Add several foods over a period of time)

(Add one food over a period of time)

A	B	C	D
<p><b>Vegetables (non-legume)</b> Carrots, squash (all types, sweet potato, white potato, string beans, broccoli, lettuce, beets, asparagus, cauliflower, brussel sprouts</p> <p><b>Fruit (non-citrus, non-tropical)</b> Apple, pear, peaches, plum, apricot, nectarine, grape, raisins</p> <p><b>Vegetables</b> Tomatoes, celery, cucumber, onion, garlic, any other vegetables</p>	<p><b>Citrus fruits</b> Orange, grapefruit, lemon, lime</p> <p><b>Tropical fruits</b> Banana, kiwi, pineapple, mango, papaya, guava, avocado</p> <p><b>Melons</b> Honeydew, cantaloupe, watermelon</p> <p><b>Berries</b> Strawberry, blueberry, raspberry, cherry, cranberry</p> <p><b>Grains</b> Rice, millet, quinoa</p>	<p><b>Legumes</b> Lima beans, chickpeas, white/black/red beans</p> <p><b>Grains</b> Oat, barley, rye, other grains</p> <p><b>Meat*</b> Lamb, chicken, turkey, pork</p> <p><b>*Progress from well-cooked to rarer</b></p>	<p><b>Fish/shellfish</b></p> <p><b>Corn</b></p> <p><b>Peas</b></p> <p><b>Peanut</b></p> <p><b>Wheat</b></p> <p><b>Beef</b></p> <p><b>Soy</b></p> <p><b>Egg</b></p> <p><b>Milk</b></p>

Figure 4. Dietary reintroduction of food allergens. Modified with permission from Spergel and Shuker.<sup>154</sup>

# EoE Management

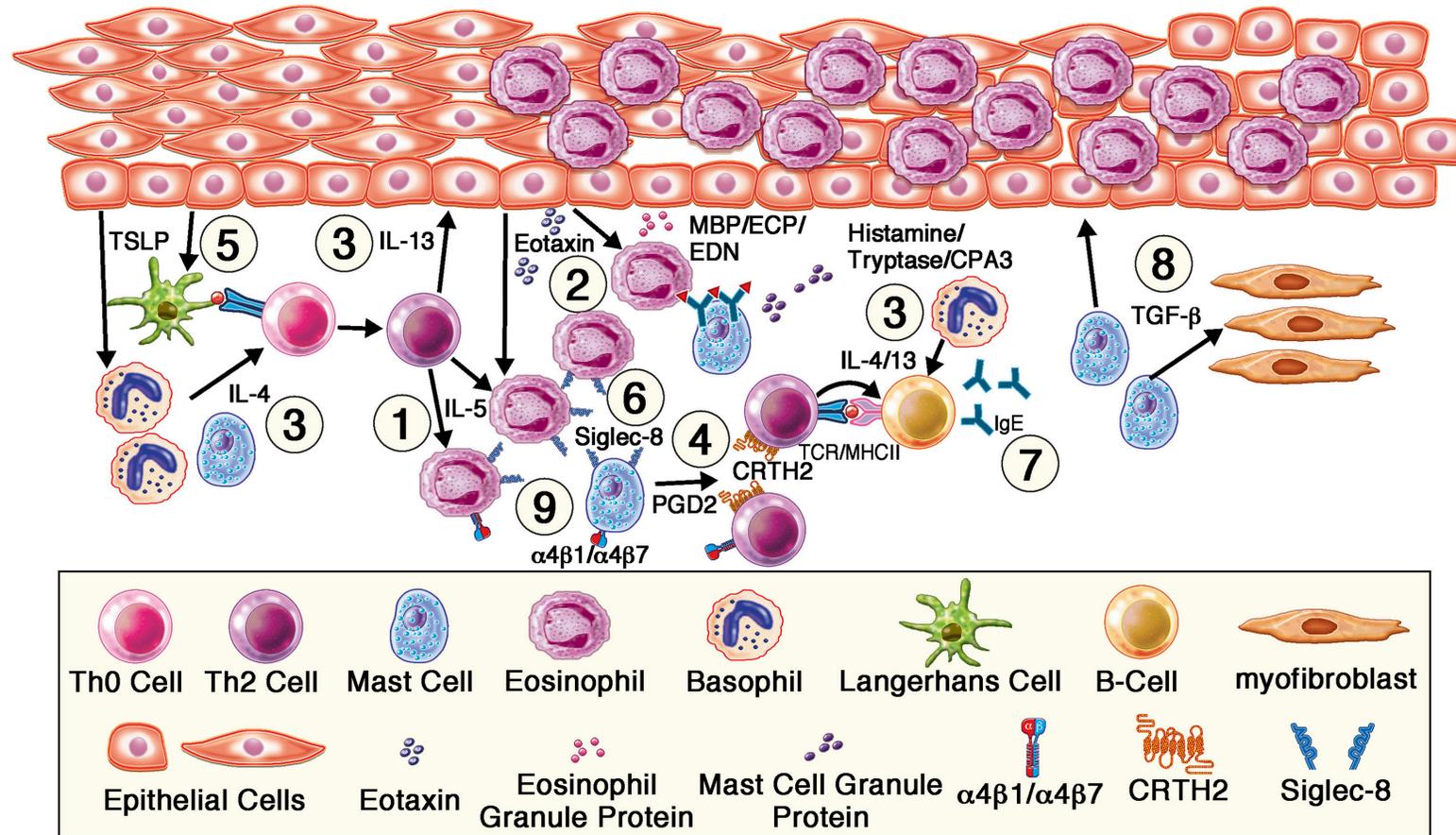
- Maintenance Therapy
  - It is a CHRONIC disease.
  - Will need long term treatment (topical steroids).
  - Dietary elimination:
    - Foods are reintroduced one by one with repeat endoscopy with biopsy to prove retained inflammatory control.
    - Foods are avoid for 6-8 weeks and then reintroduced for about 8 weeks before repeat endoscopy
    - In children, it has been reported that only 8% developed tolerance to some of all of their avoided foods over 1-14 years <sup>1</sup>

<sup>1</sup>Spergel et al, J Pediatr Gastroenterol Nutr 2009;48(1): 30-36

# Topical Steroids

- Corticosteroids induce histologic remission in 50-90% of patients. They must be continued indefinitely to maintain disease remission
- Topical steroid
  - Initial dosing for 6-8 weeks:
    - Flovent 220 mcg: Children: 880-1760 mcg/day. Adolescent and adults: 1760 mcg/day.
    - Budesonide slurries: children: 1 mg per day. Adolescent: 2 mg per day
  - Maintenance:
    - Flovent: children: 440-880 mcg/d. Adolescent: 880-1760 mcg/d
    - Budesonide maintenance: children 0.5 mg/day. Adolescent: 1 mg/day.

# Biologicals



# How to monitor EoE ?

- Esophageal EndoFlip procedure
  - A balloon catheter that is advanced orally down the esophagus to measure the actual diameter and distensibility of the esophagus.
- Esophageal String Test:
  - Measure esophageal inflammation (EoE biomarker panel: levels of eosinophil-derived granule proteins (MBP1, EDN, ECP, EPX, CLC/Gal-10)).
- Transnasal Endoscopy:
  - Using transnasal endoscopy in unsedated children appeared effective and safe while reducing costs.
- Cytosponge:
  - The Cytosponge™ Cell Collection Device (Cytosponge) is intended to collect surface cells from the esophagus. The device consists of a swallowable capsule, which dissolves in the body cavity, releasing a self-expandable sponge. The sponge is then retrieved from the esophagus using an attached cord. During the retrieval process, the sponge collects cells from the most superficial layer of the esophageal mucosa.
- Confocal Video Endoscopy





S. Urbain

