Eosinophilic esophagitis is driven primarily by Type 2 inflammation, characterized by epithelial barrier dysfunction and immune dysregulation\textsuperscript{1-3}

**TYPE 2 INFLAMMATION IN EOSINOPHILIC ESOPHAGITIS ENCOMPASSES\textsuperscript{4}:

- Both adaptive and innate cell types
  Th2 cells, ILC2 cells, mast cells, basophils, and eosinophils
- Key Type 2 cytokines
  IL-4, IL-13, and IL-5

Type 2 inflammation in eosinophilic esophagitis is a result of interactions among triggers, the epithelium, and the immune system\textsuperscript{4,5}

**3 COMPONENTS OF TYPE 2 INFLAMMATION**

<table>
<thead>
<tr>
<th>ENVIRONMENTAL FACTORS</th>
<th>HOST FACTORS</th>
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- **Allergic\textsuperscript{*}**
  - IgE
  - B cell
  - Mast cell
  - Basophil

- **Epithelial Barrier Dysfunction**
  - Allergens
  - Can lead to irreversible tissue remodeling

- **Eosinophilic**
  - (activation and trafficking to the epithelium)
  - Eosinophils

\textsuperscript{*}EoE is primarily a non-IgE-mediated disease.\textsuperscript{6}

EOS, eosinophil; ILC2, type 2 innate lymphoid cells.
Learn to recognize the signs of Type 2 inflammation in eosinophilic esophagitis\textsuperscript{2,7,8}

Look for all the signs of eosinophilic esophagitis and coexisting Type 2 inflammatory diseases when evaluating your patients\textsuperscript{2,6,9-13}:

**Symptoms**
- Dysphagia
- Food impaction
- Chest pain (noncardiac)

**Endoscopic findings**
- Rings
- Exudates
- Furrows
- Edema
- Strictures

**Histologic findings**
- Eosinophilic count $\geq 15$ EOS/HPF histology/HE stain

**Coexisting Type 2 inflammatory disease** (present in ~75% of patients with eosinophilic esophagitis)
- Allergic rhinitis
- Asthma
- Atopic dermatitis
- CRSwNP
- IgE-mediated food allergy

**Understand the impact of Type 2 inflammation on symptoms of eosinophilic esophagitis**

CRSwNP, chronic rhinosinusitis with nasal polyposis; EOS/HPF, eosinophils per high-power field; HE, hematoxylin and eosin.
IL-4, IL-13, and IL-5 are key drivers of Type 2 inflammation in eosinophilic esophagitis\textsuperscript{4,7-9,14-16}

- Eosinophilic esophagitis is a heterogeneous disease with a complex pathophysiology
- Immune cells such as eosinophils, mast cells, basophils, and B cells also participate in the inflammatory response of eosinophilic esophagitis

**MAST CELL AND BASOPHIL INFLAMMATION**
- IL-4 and IL-13 contribute to the activation of mast cells and basophils
- Leads to cellular activation and the degranulation of several inflammatory mediators

**EPITHELIAL BARRIER DYSFUNCTION**
- Decreased levels of epithelial junction proteins
- Increased access to allergens and pathogens across the epithelial barrier
- Increased epithelial permeability and barrier disruption

**REMODELING AND FIBROSIS**
- Remodeling and fibrosis, such as strictures, rings, or furrows
- Increased smooth muscle contraction

**EOSINOPHILIC INFLAMMATION**
- Eosinophil trafficking to tissues
- Contributes to esophageal remodeling
- Eosinophil differentiation in the bone marrow

**Type 2 Inflammation**

**Recognize the role of Type 2 inflammation as a driver of disease in patients with eosinophilic esophagitis**

TSLP, thymic stromal lymphopoietin.
IL-4, IL-13, and IL-5 are key mediators of Type 2 inflammation in eosinophilic esophagitis\textsuperscript{4,7-9,14,17}

**TYPE 2 INFLAMMATION**

- Th0 cell differentiation to Th2
- Effect on mast cells and basophils
- Increased barrier disruption
- Fibrosis, tissue remodeling, and increased smooth muscle contraction
- Increased endothelial permeability

IL-4

IL-13

IL-5

Eosinophils trafficking to tissues

Eosinophil differentiation in bone marrow

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**EoE AND THE ROLE OF TYPE 2 INFLAMMATION IN ITS PATHOGENESIS**

References: