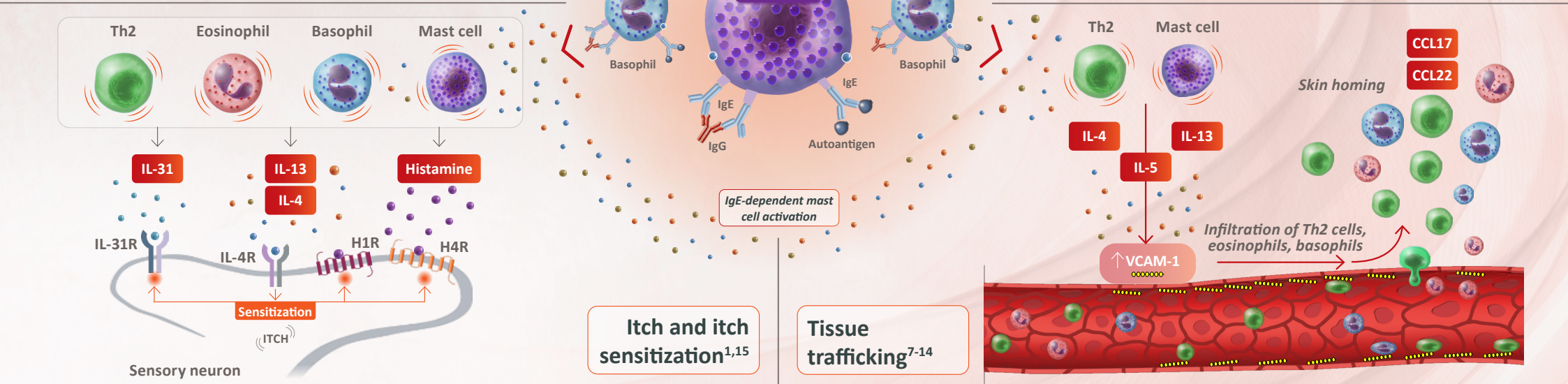




●●●●●●●●●● Indicate secreted mediators (e.g. histamine, IL-4, IL-5, IL-13, IL-31); **CCL**, CC chemokine ligand; **FcεR1**, high-affinity IgE receptor; **H**, histamine; **Ig**, immunoglobulin; **R**, receptor; **Th2**, T helper 2; **VCAM-1**, Vascular cell adhesion protein 1.

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The pathophysiology of CSU is complex and may involve mast-cell degranulation following FcεRI binding, via IgE-dependent and IgE-independent mechanisms.*

Activation and degranulation of **mast cells** and **basophils** lead to the production of **inflammatory mediators**, including histamines, neuropeptides and key type 2 cytokines such as IL-4, IL-5, and IL-13.⁵⁻⁹

Type 2 inflammation promotes IgE synthesis, neuronal sensitization, activation and recruitment of inflammatory cells, contributing to the chronicity of the disease^{1-4, 10-11}

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*These mechanisms may also be characterized as autoimmune (IgG anti-IgE / IgG anti-FcεRI) or autoallergic (IgE to self-antigens).

CSU, chronic spontaneous urticaria; IL, interleukin.

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