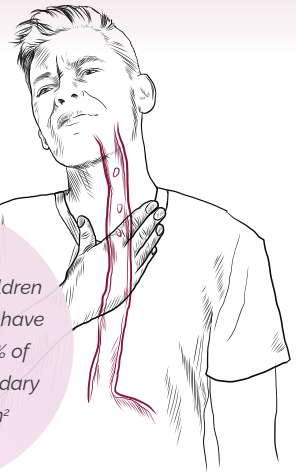
REVOLO BIOTHERAPEUTICS

Analyze. Reset. Revolutionize.

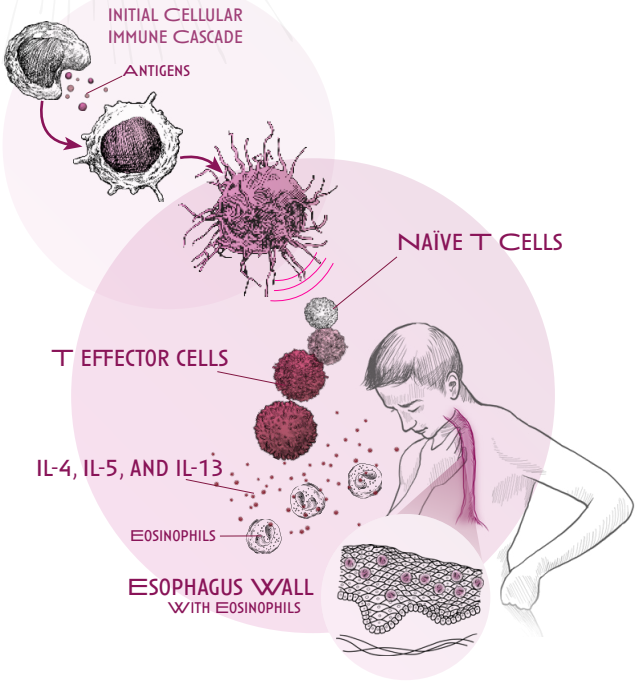
EOSINOPHILIC ESOPHAGITIS

We aim to revolutionize the treatment landscape for eosinophilic esophagitis (EoE)

EoE is a chronic rare allergic inflammatory disease characterized by difficulty swallowing and gastric reflux as a result of the thickening of the esophagus walls. In many cases the patient has difficulty swallowing, and in some cases, food is impacted and requires urgent removal.



Roughly 180,000 children and adults in the US have EoE[1], and up to 80% of patients have secondary allergic condition[2]



WHAT DRIVES EOE?

EoE is caused by an immune response to certain foods and environmental allergens that are unknown to the body, causing the immune system to "overreact".

This leads to an increase in inflammatory cells such as T effector cells and eosinophils in the mucosa that lines the esophagus walls, in addition to the production of inflammatory cytokines **such as interleukin 4, 5 and 13** (IL-4, IL-5 and IL-13).

IL-4, IL-5, and IL-13 are key drivers of the complex pathophysiology of EoE, contributing to immune system overreaction, eosinophil trafficking to the esophagus tissues, as well as esophageal remodeling.

EXISTING THERAPIES

There are no approved therapies for EoE. Currently, patients are forced to control their diet and rely on steroid treatments that target the inflammatory pathway after the immune system has been overactivated, limiting their efficacy.

Treatments prescribed by physicians lead to **chronic exposure** due to frequent dosing and long pharmacokinetic characteristics resulting in **suppression of the immune system**, putting patients at risk of severe life-threatening side effects. Frequently administered oral therapies can also lead to poor compliance.

~50,000[3] patients in the US are unresponsive to available treatments

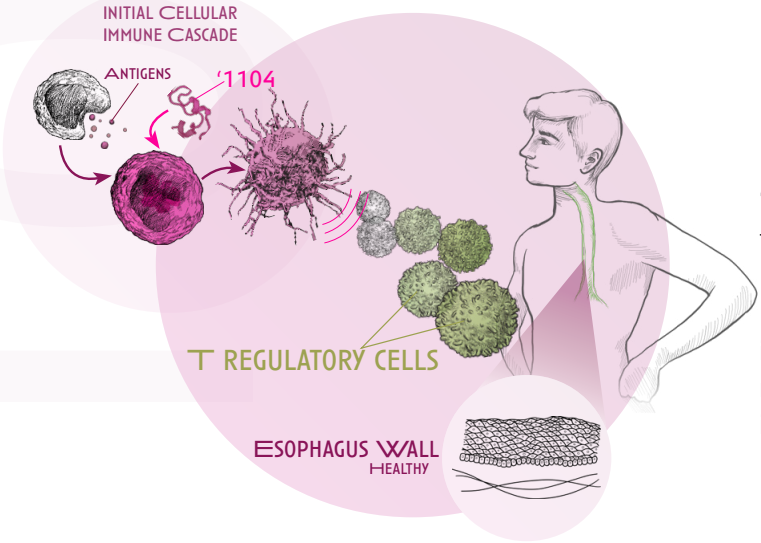
Current therapeutic options do not address the complex physiopathology: While current therapies reduce eosinophil counts, this does not always lead to symptomatic response nor decrease in IL-4, IL-5 or IL-13 levels.

References: 1. Moawad FJ. Eosinophilic esophagitis: incidence and prevalence. Gastrointest Endosc Clin N Am. 2018;28(1):15-25.
2. Wechsler JB, Bryce PJ. Allergic mechanisms in eosinophilic esophagitis. Gastroenterol Clin North Am. 2014;43(2):281-296.
3. Reference to come

‘1104

Resetting the immune system for long-term EoE disease remission.

Revolo’s clinical drug candidate ‘1104 is a peptide derived from a protein involved in resetting the immune system, mTB chaperonin 60.1.



HOW IS ‘1104 DIFFERENT FROM EXISTING THERAPIES?

‘1104 resets the immune system “upstream” or before the inflammatory cascade.

It increases the number of cells that regulate the immune system (T regulatory cells or T regs) and restores its balance, thereby avoiding chronic inflammation without suppressing the immune system.

‘1104 – ADDRESSING BOTH CELLULAR AND CYTOKINE-RELATED INFLAMMATION

REDUCES CELLULAR INFLAMMATION	REDUCES RELEVANT CYTOKINES (IL-4, IL-5, IL-13)	INCREASES EXPRESSION OF A20	ALLERGEN AGNOSTIC	DEMONSTRATED SAFETY AND TOLERABILITY IN TWO PHASE 1 STUDIES
Single dose ‘1104 reduced three key inflammatory cells (eosinophils, neutrophils and lymphocytes) in pre-clinical studies[4-6]	Single dose ‘1104 reduces relevant cytokines involved in the inflammatory cascade[5]	Single dose ‘1104 increased A20, a key regulator of the inflammatory response[6]	‘1104 reduced the cellular inflammation to a range of allergens in pre-clinical studies[4]	with intravenous or subcutaneous administration at clinically relevant doses (total of 112 subjects dosed with ‘1104)[4]

PHASE 2 TRIAL

A PHASE 2 TRIAL for EoE patients is ongoing at multiple clinical sites in the US. Click here to learn more.

Revolo is also testing the applicability of ‘1104 for other allergic diseases through a PHASE 2 allergen sensitivity study.

References: 4. Immune Regulation Ltd. Immune Regulation announces positive results from Phase 1 clinical trial of IRL201104. https://revolobio.com/2019/12/10/immune-regulation-announces-positive-results-from-phase-1-clinical-trial-of-irl201104/. Published December 10, 2019. Accessed Dec 20, 2021. 5. Riffo-Vasquez Y, et al. 2012. “Mycobacterium tuberculosis chaperonin 60.1 Inhibits leukocyte diapedesis in a murine model of allergic lung inflammation.” Am. J. Respir. Cell Mol. Biol.; 47(2):245-52. 6. Riffo-Vasquez Y, et al. 2020. “Modulation of allergic inflammation in the lung by a peptide derived from Mycobacteria tuberculosis chaperonin 60.1.” Clinical & Experimental Allergy; 50(4), 508–519.

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