Allakos

Corporate Presentation

March 2024

Developing Therapeutic Antibodies Targeting Allergic, Inflammatory and Proliferative Disease

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This presentation concerns products that are under clinical investigation, and which have not yet been approved for marketing by the U.S. Food and Drug Administration. It is currently limited by federal law to investigational use, and no representation is made as to its safety or effectiveness for the purposes for which it is being investigated.

Allakos Opportunity

Novel Target

- AK006 (anti-Siglec-6 mAb) selectively inhibits multiple modes of mast cell activation
  - Inhibits IgE-dependent and IgE-independent mast cell activation pathways, including IgE, KIT and MRGPRX2
  - Depletes mast cells by ADCP in the presence of activated macrophages

Significant Need for New Agents

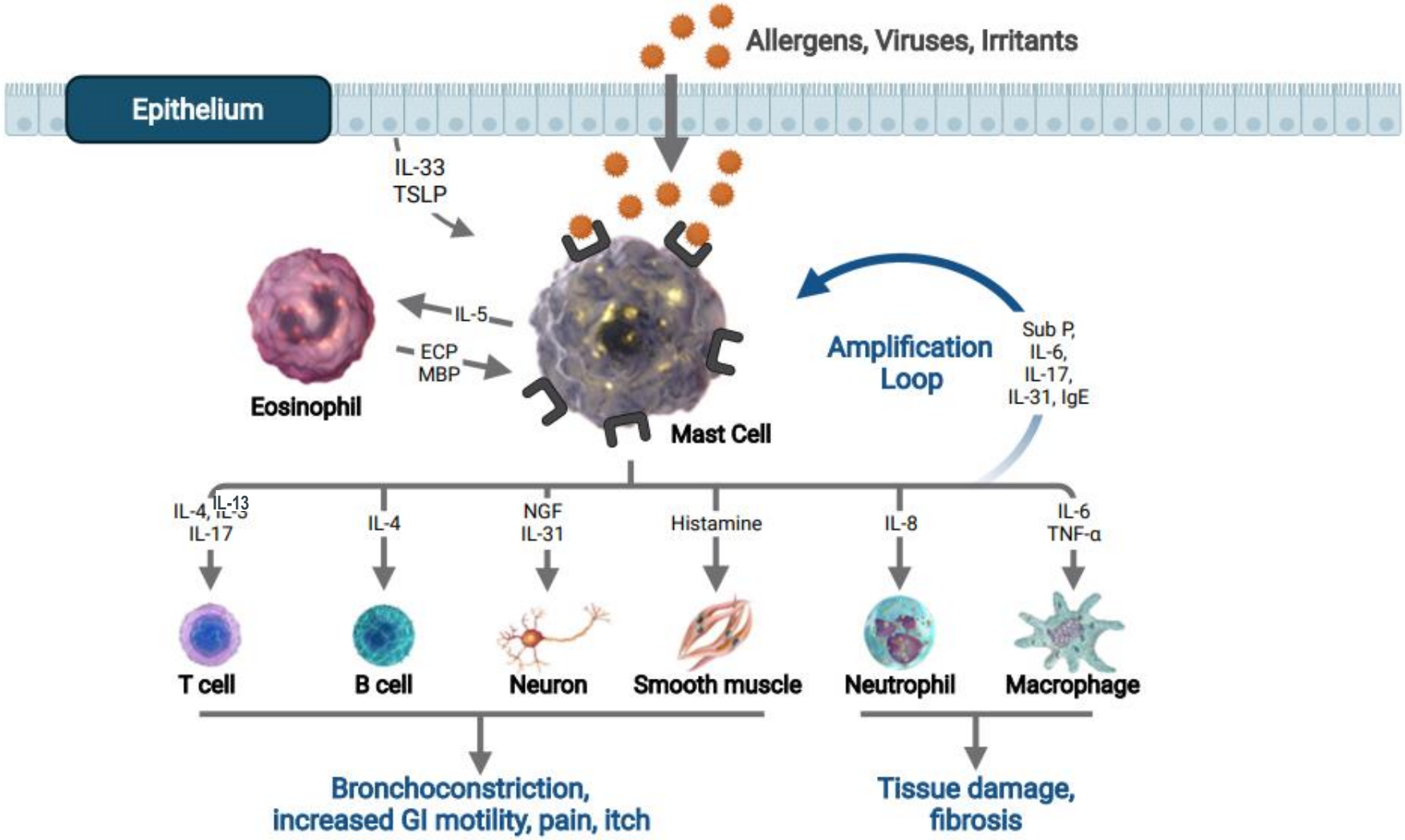
- AK006 has the potential to treat a broad range of mast cell driven diseases
- AK006 to be tested in Chronic Spontaneous Urticaria (CSU) and one additional proof-of-concept study

Upcoming Data Catalysts and Expected Milestones

Milestones

- 2Q24 – Report AK006 single and multiple ascending dose Phase 1 data, including safety PK, PD, and skin biopsy data assessing Siglec-6 target engagement
- YE24 – Report topline Phase 1 data of AK006 in patients with CSU

Mast Cells Are Key Drivers of Inflammatory Disease



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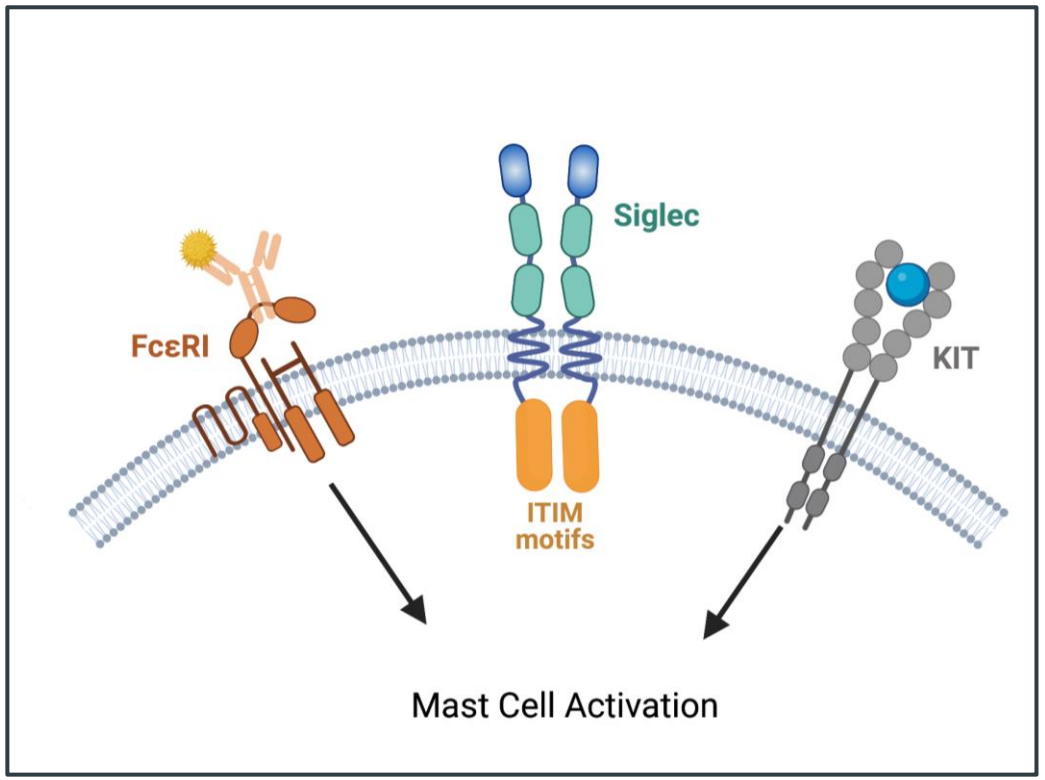
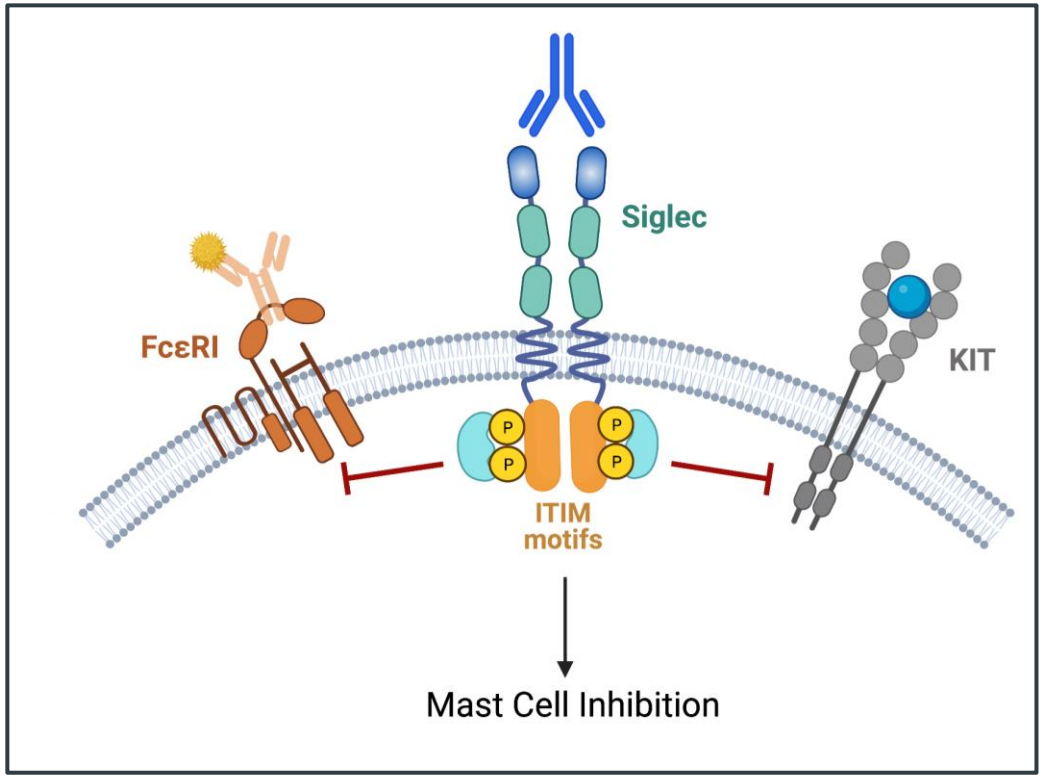
Leveraging the Native Inhibitory Function of Siglecs on Mast Cells

Activated State



Mast cells can be activated by numerous receptors leading to mast cell degranulation and release of histamine, TNFα and other inflammatory mediators

Siglec-Mediated Inhibition



Activation of Siglec-6 with an agonistic antibody activates inhibitor machinery inside cell which attenuates activating signals

SOURCE: O'Sullivan J et al. Seminars in Immunology. 2023 Jul; 4; 69:101799

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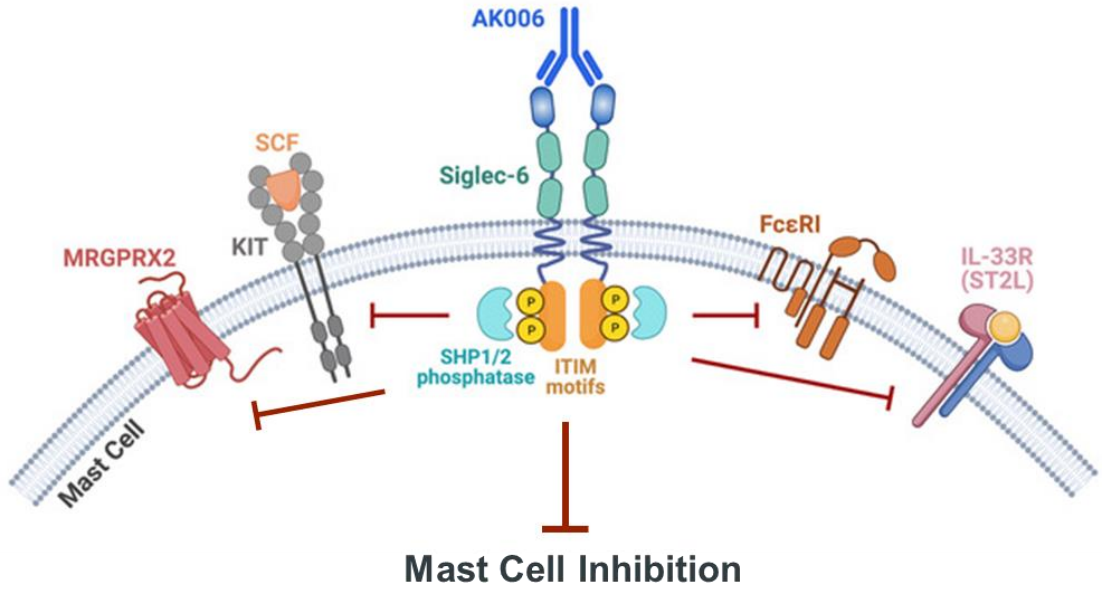
AK006 is Engineered for Deep Mast Cell Inhibition

AK006 (Anti-Siglec-6)

- AK006 is a Siglec-6 mAb that selectively inhibits mast cells and reduces their numbers
  - Engineered for deep mast cell inhibition
  - Reduces mast cells via ADCP in presence of activated macrophages
- AK006 inhibits multiple mast cell activation pathways including IgE, IL-33, KIT, C5a and MRGPRX2
- The Phase 1 study of AK006 consists of single and multiple ascending doses administered in healthy volunteers, followed by a randomized, double-blind, placebo-controlled, CSU cohort



Allakos Pipeline

Program	Indication	Discovery	Preclinical	Phase 1	Phase 2	Phase 3	Milestone
AK006 (Anti-Siglec-6)	Healthy Volunteers & CSU						Expected SAD & MAD results from healthy volunteers in 2Q24; CSU results at YE24
AK068 (Siglec-6/Siglec-8 Bispecific)	Inflammatory Diseases						Ongoing
T Cell Immunomodulatory Receptors	Inflammatory Diseases						Ongoing
AK007 (Anti-Siglec-10)	Immuno-Oncology						Ongoing

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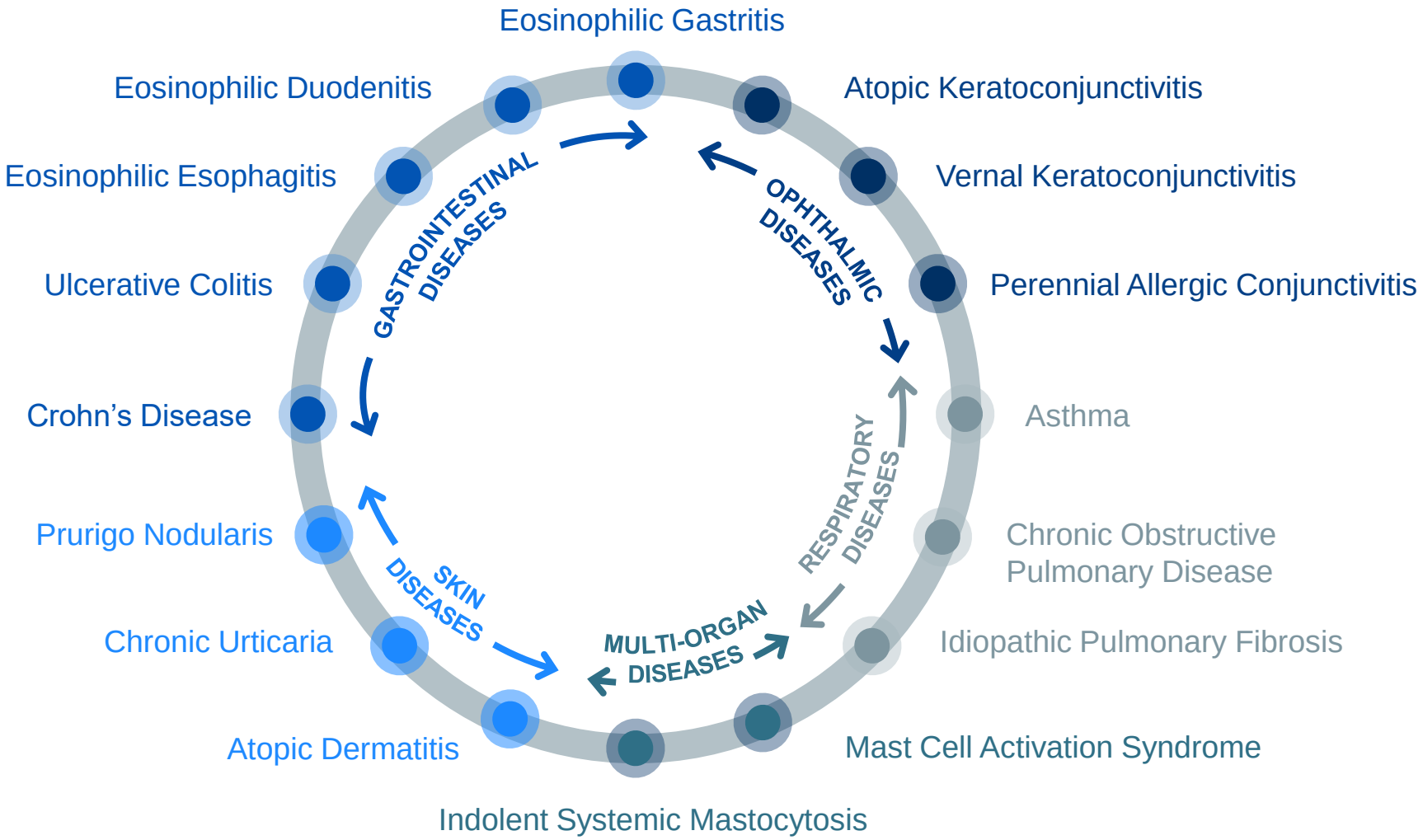
Mast Cells Play a Significant Role in Many Diseases



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AK006: Siglec-6 mAb that Selectively and Potently Inhibits Mast Cells

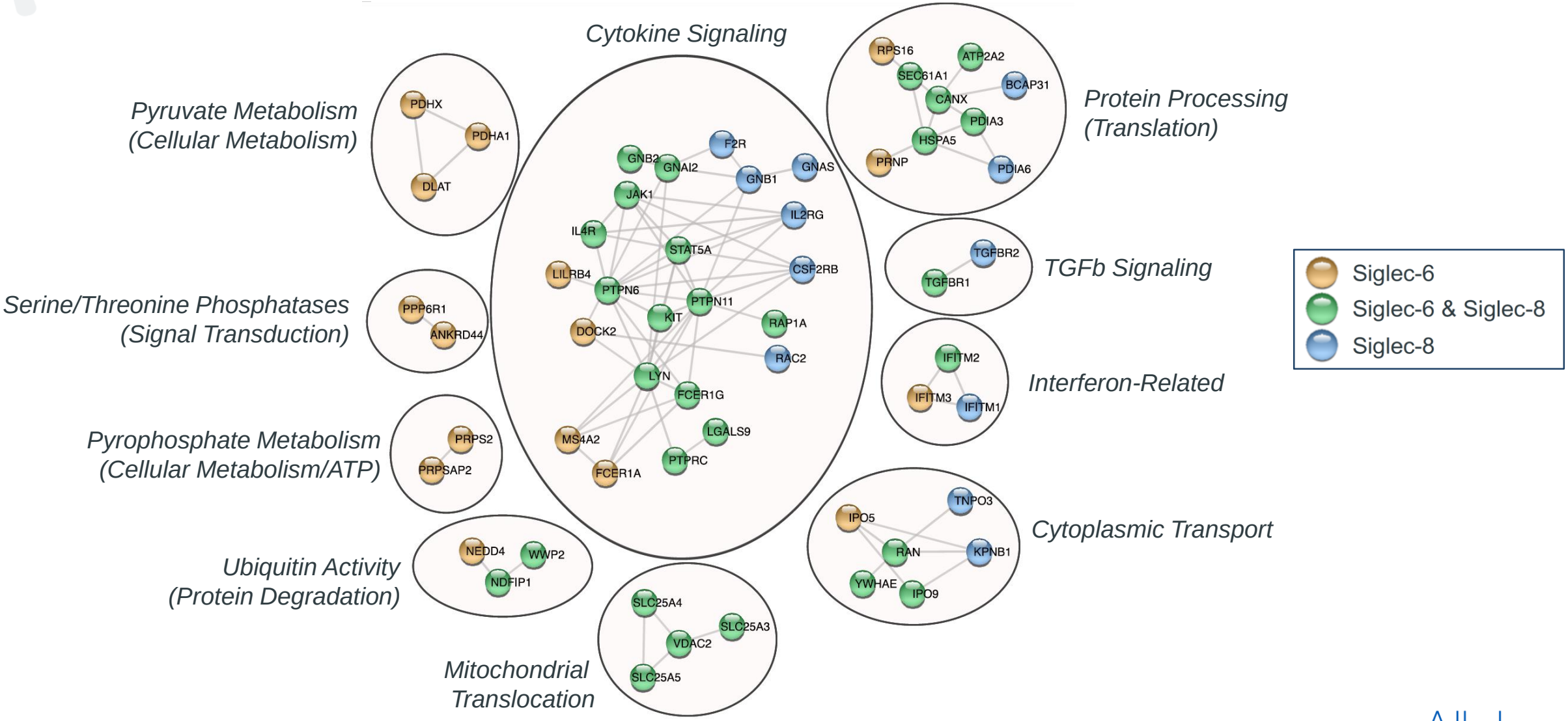
Siglec-6 Biology and AK006

AK006 Targets a Different Receptor Than AK002 with Different Underlying Biology

- **Siglec-6 is a more potent inhibitory receptor than Siglec-8**
  - Siglec-6 regulates more cellular processes than Siglec-8:
    - Signal Transduction
    - Transcription
    - Translation
    - Cellular metabolism
    - Degranulation

- **AK006 has two key attributes**
  - Long residence time on the cell surface which correlates to increased inhibitory activity
  - Antibody Dependent Cellular Phagocytosis (ADCP)

Siglec-6 and Siglec-8 Differentially Interact with Proteins that Regulate Mast Cell Activity



Gray lines represent direct interaction

SOURCE: Korver, W. et al Allergy 2024.

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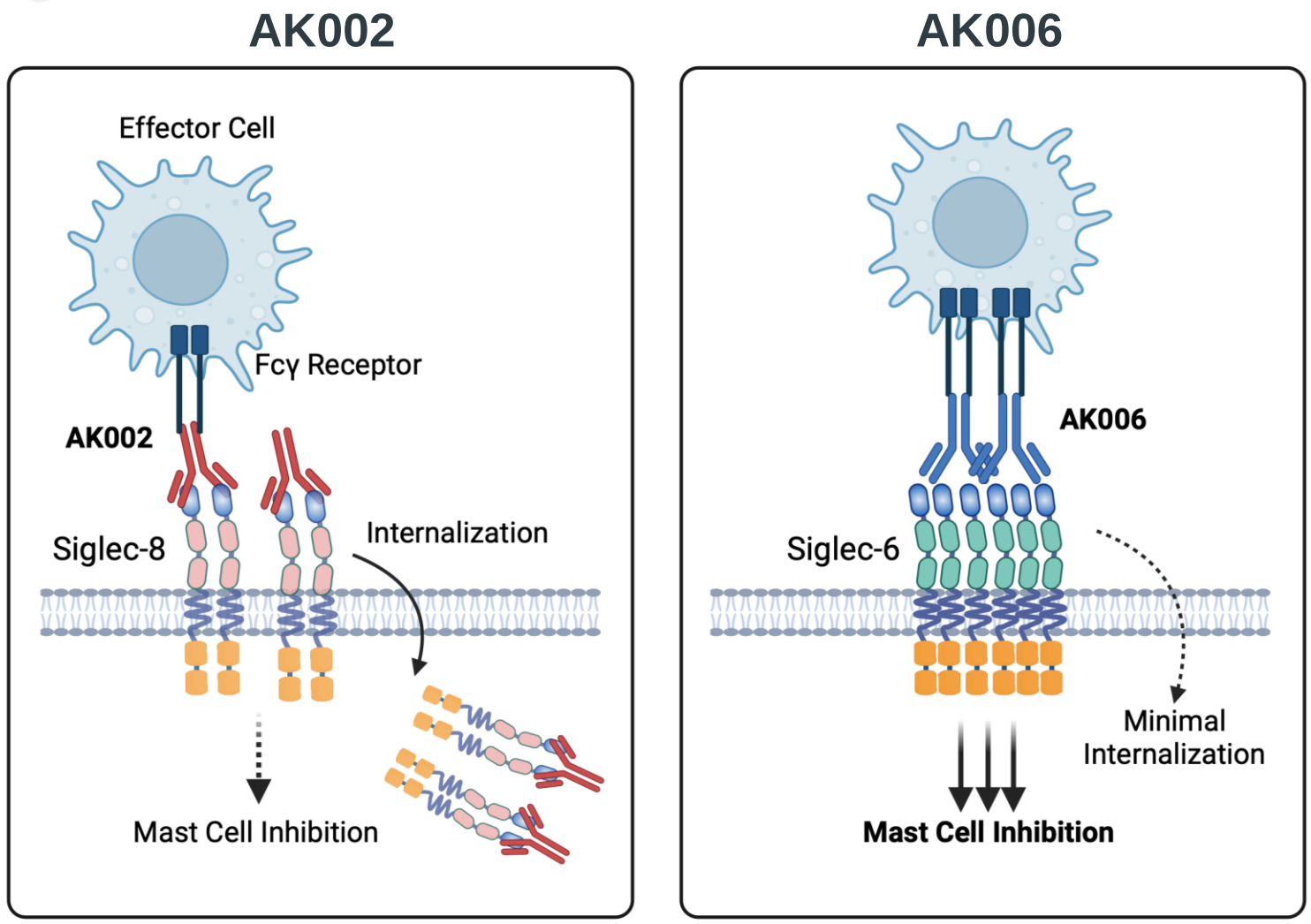
AK006 was Designed to Drive Deep Mast Cell Inhibition



- Mast cell inhibition for AK002 and AK006 requires Fc-Fcγ receptor interaction
- Binding of AK006 leads to minimal receptor internalization, resulting in greater mast cell inhibition
- AK006 displays a high residence time on mast cells which is associated with optimal inhibition
- AK006 induces antibody dependent cellular phagocytosis

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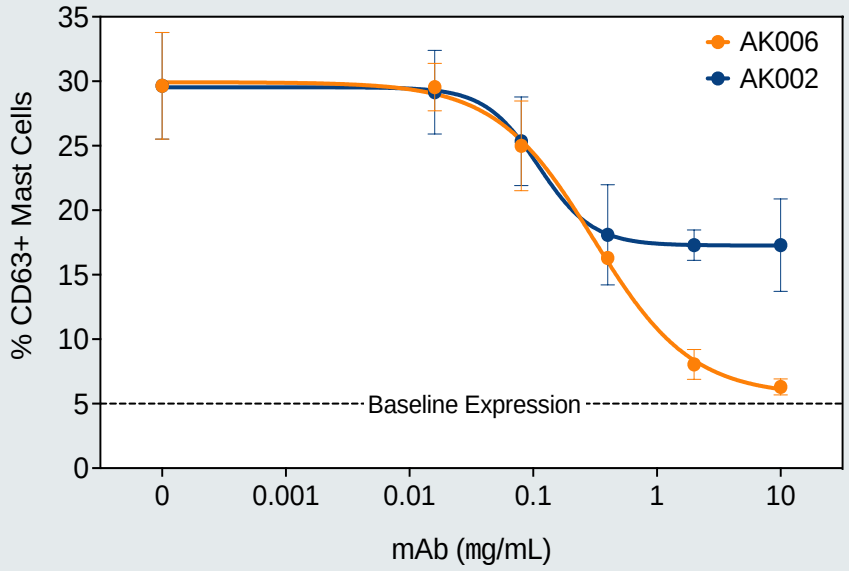
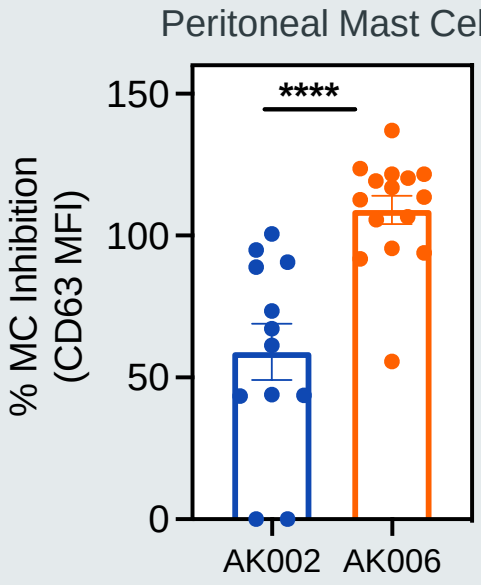
AK006 Displays Greater Mast Cell Inhibition than AK002 in Preclinical Studies

IgE Activation



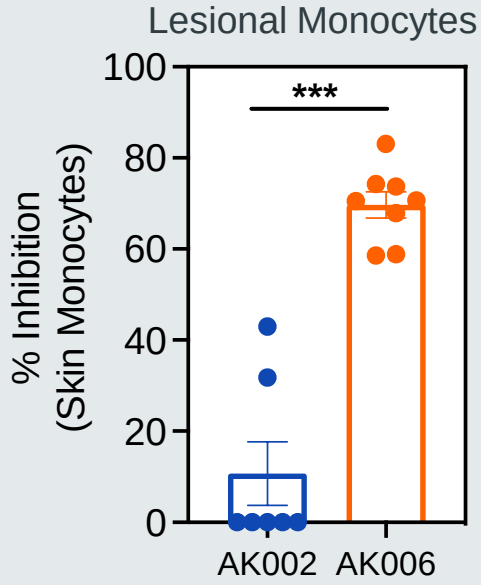
n=3 human donors

KIT Activation



**** $p < 0.0001$, n=8-14 mice/group

MRGPRX2 Activation



*** $p < 0.001$, n=7-8 mice/group

AK006 inhibits IgE-dependent and IgE–independent modes of mast cell activation better than AK002

SOURCE: Korver, W. et al *Allergy* 2024; Benet Z, et al. AAAAI 2024 Presentation.

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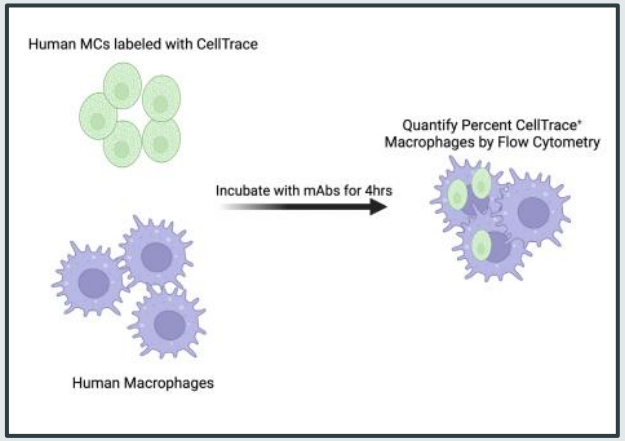
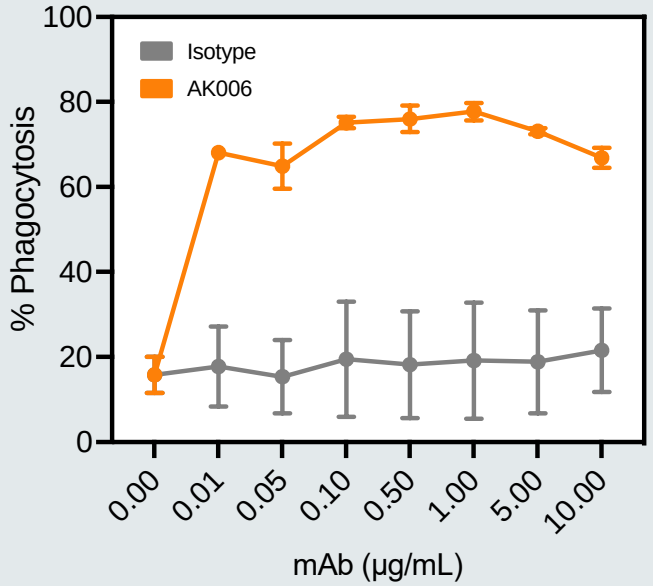
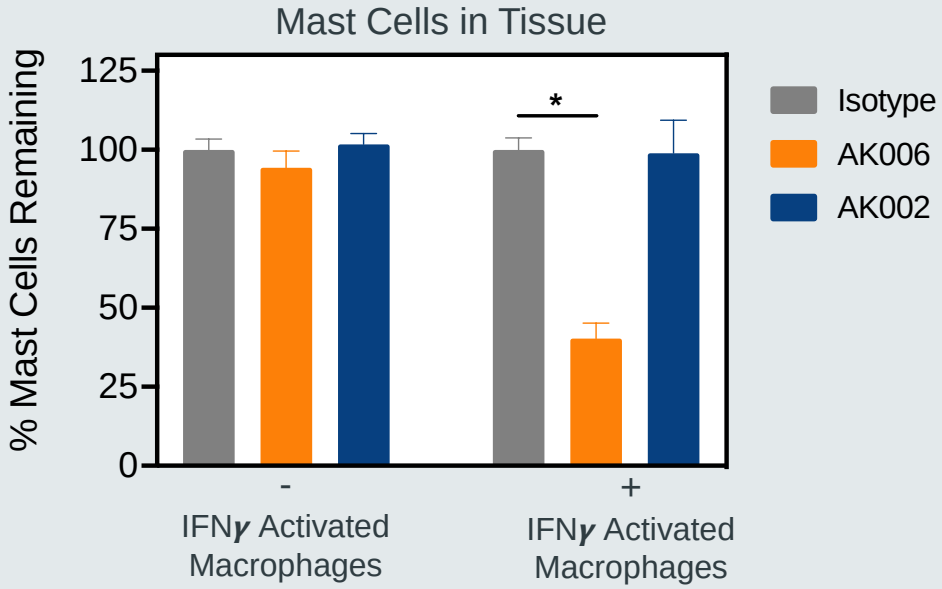
AK006 Reduces Human Mast Cells via Antibody-Dependent Cellular Phagocytosis in Preclinical Studies

In Vitro ADCP Assay





Ex Vivo Human Tissue Mast Cells



* $p < 0.05$; n=3 human donors

In addition to mediating broad inhibition, AK006 can reduce mast cell numbers

SOURCE: Benet Z, et al. AAAAI 2023 Presentation.

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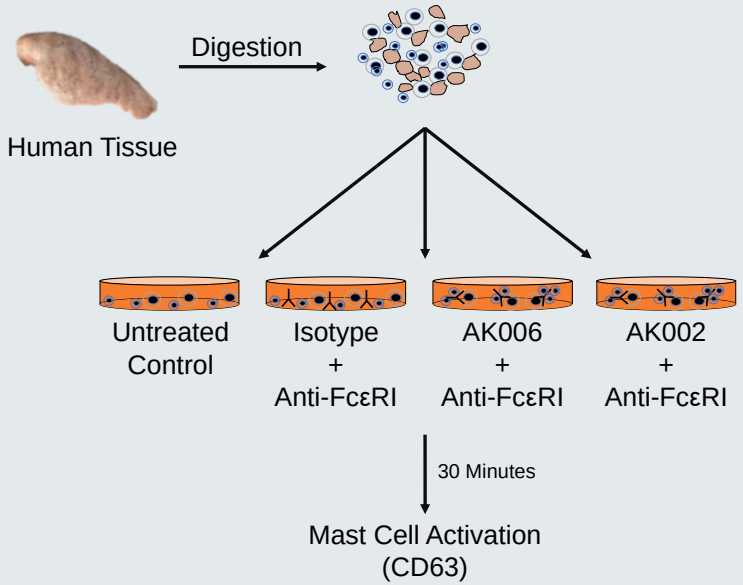
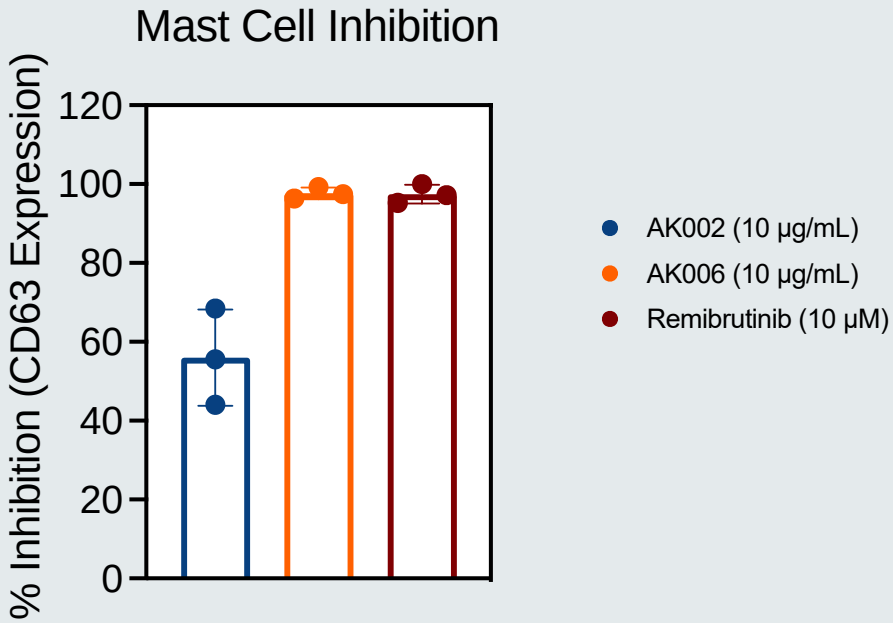
AK006 Inhibits IgE-Mediated Mast Cell Activation Similar to Remibrutinib

Human Mast Cell Activation Assay



IgE-Activated Human Tissue Mast Cells



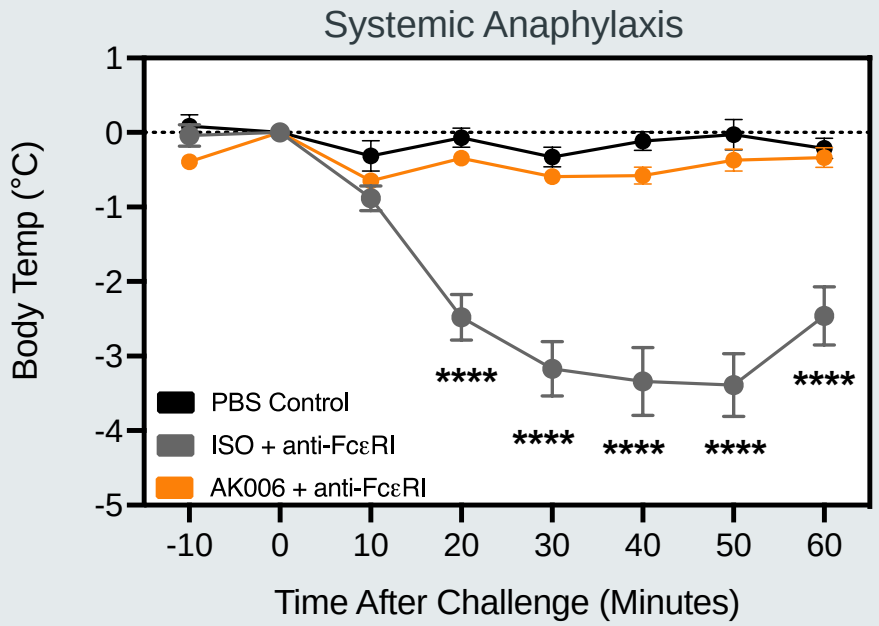
n=3 human donors

AK006 inhibits IgE-mediated mast cell activation

SOURCE: Korver W, et al. EAACI 2023 Presentation.

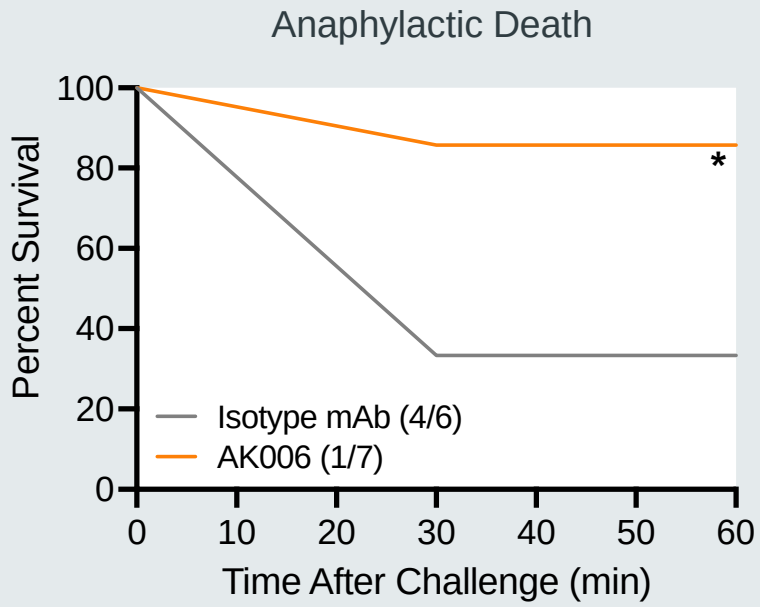
AK006 Protects Against Systemic Anaphylaxis in Humanized Mice

Humanized Mouse Model of Anaphylaxis



**** $p < 0.0001$, n=8-15 mice/group

Humanized Model of Anaphylactic Death



* $p < 0.05$, n=6-7 mice/group

AK006 inhibits IgE-mediated mast cell activation in vivo

SOURCE: Schanin, J et al. Communications Biology 2022

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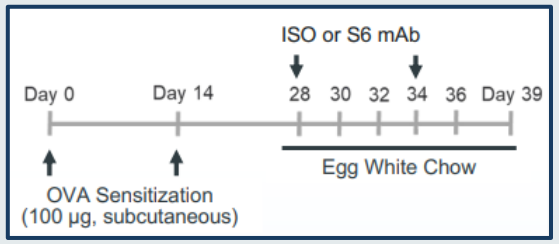
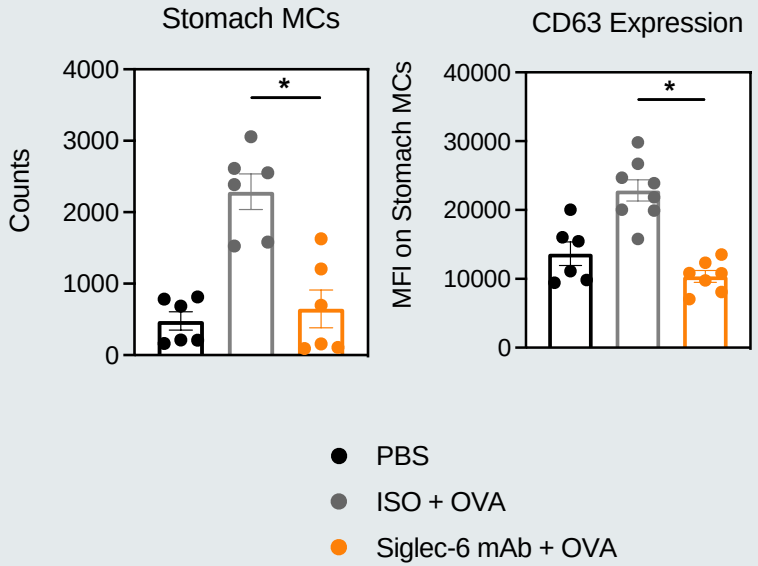
AK006 Resolves Gastric Inflammation in Oral Food Challenge Model

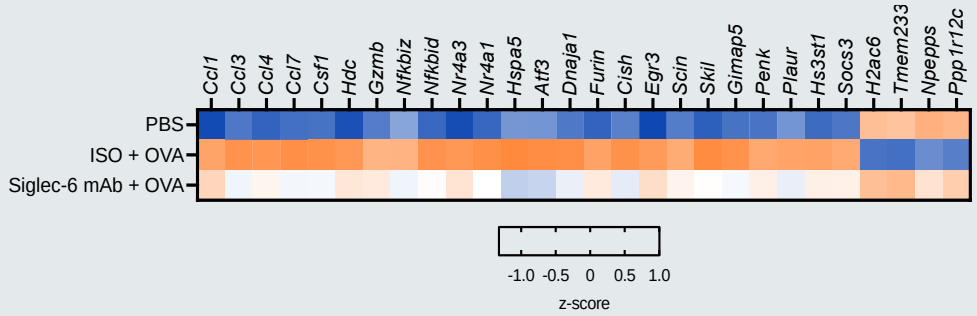
Model of OVA-Induced Allergic Enteritis



Stomach Mast Cells



Transcriptional Profiling of Stomach Mast Cells



* $p < 0.01$, n=6 mice/group

AK006 shows inhibitory and depleting effects in food challenge model

SOURCE: Benet Z, et al. EMBRN 2022 Presentation.

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AK006 for Chronic Spontaneous Urticaria

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AK006 May Inhibit Disease Driving Pathways in Urticaria

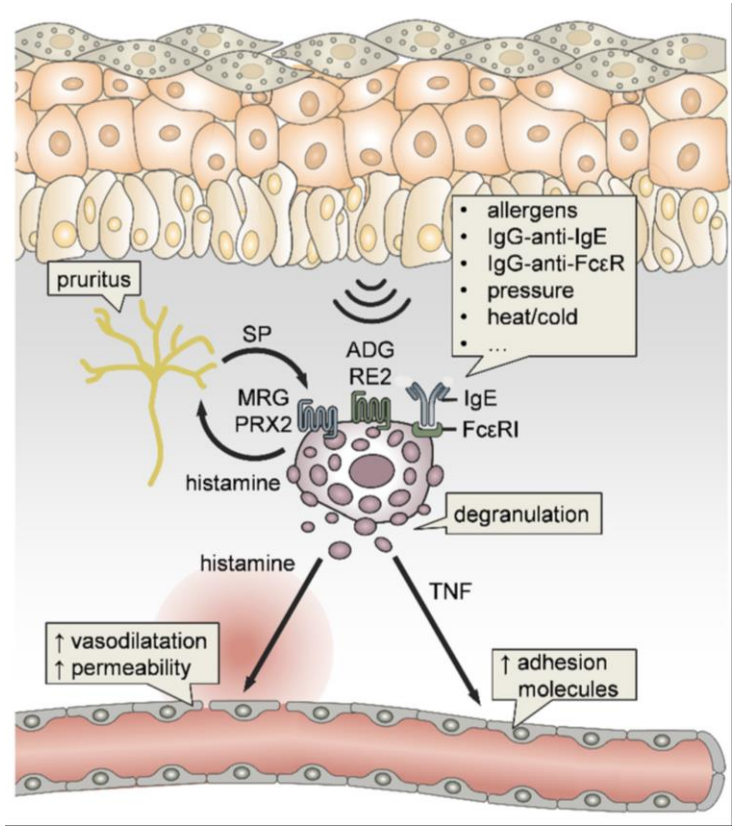
Activated mast cells drive the pathogenesis of urticaria via release of inflammatory mediators resulting in pruritis, vasodilation, and increased vascular permeability

IgE activation of mast cells, from autoantibodies or allergens, has been identified as driving pathogenesis in a proportion of patients with chronic urticaria

IgE-independent mast cell activation, via MRGPRX2 and other mast cell receptors, is also believed to contribute to symptoms

Blocking both IgE activation and IgE-independent mast cell activation could result in improved patient outcomes in CSU

Urticaria



SOURCE: Voss, M.; Int. J. Mol. Sci. 2021, 22, 4589. doi.org/10.3390/ijms22094589; Siiskonen H. Front Cell Neurosci. 2019;13:422. Published 2019 Sep 18. doi:10.3389/fncel.2019.00422

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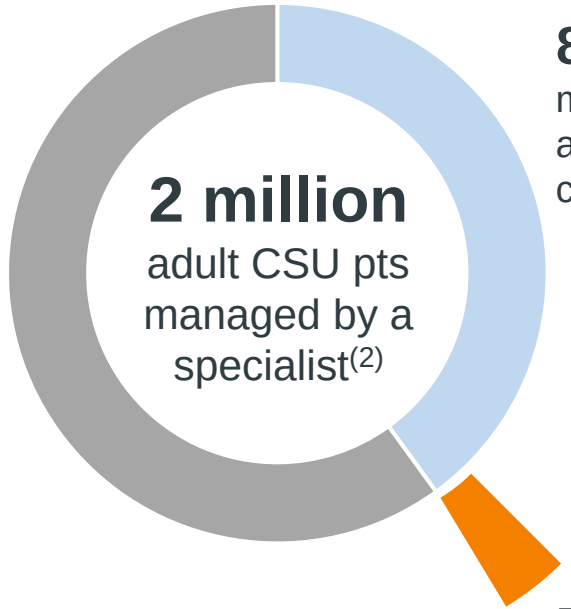
Chronic Spontaneous Urticaria Opportunity

CSU affects up to 3 million adults in the U.S., with 2 million managed by a specialist (allergist or dermatologist)[1,2]

An estimated 800k adults with CSU are biologic-eligible, yet only approximately 10% of eligible patients are currently on a biologic[2,3]

Currently only Xolair is approved for the treatment of antihistamine CSU. Xolair® (omalizumab) global CSU sales were estimated to be $1.6 billion in 2021[4]

Approximately 800k adult CSU patients are eligible for a biologic in the U.S.



1. Maurer M, et al. Allergy. 2011 Mar;66(3):317-30. **2.** Allakos allergist and dermatologist market research survey (N=208), Nov 2021. **3.** Decision Resources Chronic Urticaria Report, Nov 2020.
4. Novartis 2021 annual report and earnings call

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AK006 Phase 1 Study Design

Trial Cohorts

Single Ascending Dose (SAD) and Multiple Ascending Dose (MAD) Cohorts in Healthy Volunteers

- Randomized, double-blind, placebo-controlled
- Intravenous AK006
  - SAD: 5, 20, 80, 240, 720 mg
  - MAD: 80, 240, 720 mg monthly
- Subcutaneous AK006
  - 150 and 720 mg

Planned CSU Cohort

- Randomized, double-blind, placebo-controlled
- Moderate-to-severe antihistamine refractory CSU
  - UAS7 score ≥16 and HSS7 score ≥8 at baseline
- Four doses of AK006 IV given monthly

Endpoints

SAD and MAD Cohort

- Safety and tolerability
- Pharmacokinetics
- Pharmacodynamics
- Target receptor engagement in skin biopsies
- SC Bioavailability

CSU Cohort

- Therapeutic activity assessed by changes in UAS7 at week 14
- Safety and tolerability

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Chronic Spontaneous Urticaria Landscape

Drug Name	MOA	UAS7 Response	Opportunity
Xolair® (omalizumab)	Anti-IgE mAb	Dose Group[1] / 150 mg / 300 mg / Placebo UAS7 / -14.4 (-48%) / -20.8 (-66%) / -8.0 (-26%) UAS7=0 / 15% / 36% / 9%	• >50% of patients continue to have symptoms • Black box for anaphylaxis[1]
Dupixent® (dupilumab)	Anti IL-4/IL-13R mAb	Dose Group[2] / 300 mg / Placebo UAS7 / -20.5 (-65%) / -12.0 (-37%)	• Q2W dosing • No improvement in Xolair failures[3]
Barzolvolimab	Anti KIT mAb	Dose Group[4] / 75 mg Q4W / 150 mg Q4W / 300 mg Q8W / Placebo UAS7 / -17 (-56%) / -23 (-75%) / -24 (-76%) / -10 (-35%) UAS7=0 / 23% / 51% / 38% / 6%	• c-Kit is expressed on hematopoietic stem cells, melanocytes, CNS and germ cells[5]
Remibrutinib	BTK Inhibitor	Dose Group[6] / 25 mg BID / Placebo UAS7 / -20 & -20 (-65% & -65%) / -12 to -14 (-40% to -46%) UAS7=0 / 28% to 31% / 7% to 11%	• BTK is expressed on hematopoietic cells including B cells, myeloid cells, and platelets[7]

SOURCE:1.) Xolair Label; UAS7 scores are calculated change from baseline and percentage change; 2.) Sanofi PR 7/29/21 3.) Sanofi PR 2/18/22 4.) Celldex Presentation 11/6/23 5.) N.F. Russkamp et al. Experimental Hematology 2021;95:31−45 6.) Saini S, et al. ACAAI 2023 Presentation 7.) Garg N et. al. J Clin Med 2022; 11(20):6039

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Lirentelimab: Siglec-8 mAb that Depletes Eosinophils

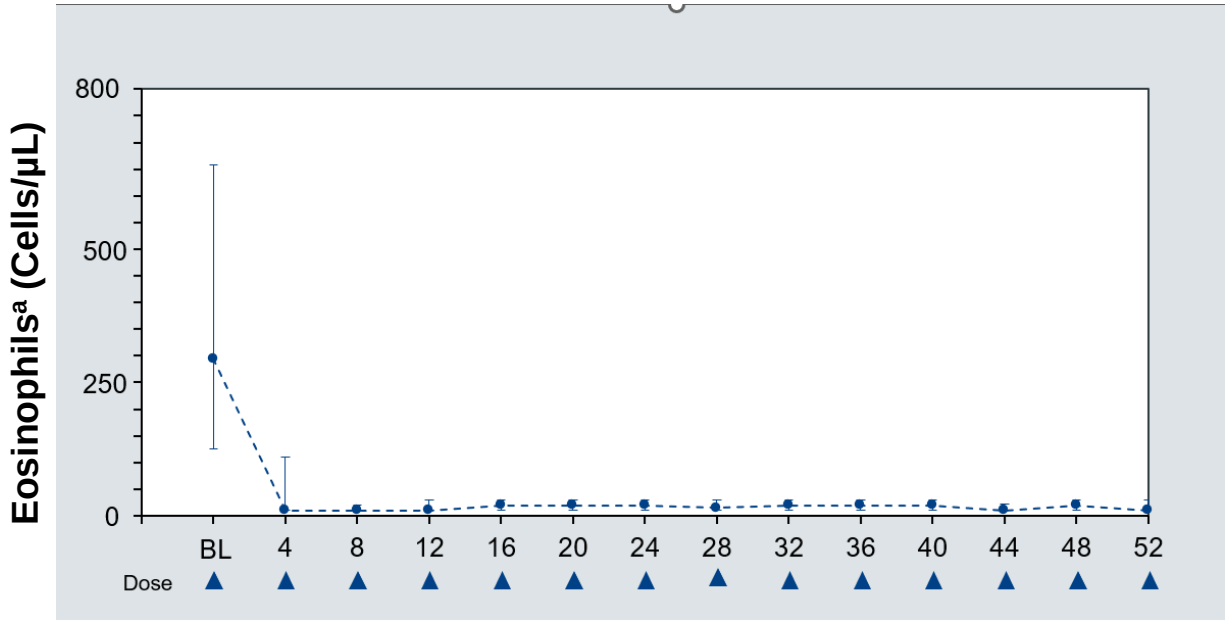
Lirentelimab: Siglec-8 mAb That Depletes Eosinophils

Lirentelimab (Anti-Siglec-8)

- Lirentelimab targets Siglec-8, an inhibitory receptor expressed selectively on mast cells and eosinophils
- Binding of lirentelimab to Siglec-8 results in rapid and sustained depletion of eosinophils via antibody-dependent cellular cytotoxicity (ADCC)
- Eosinophil depletion is detectable at first measurement and sustained through 52 weeks.
- Lirentelimab has been administered in more than 1,000 patients with more than 500 patients that have been treated for six months or longer

Sustained Depletion of Blood Eosinophils

Phase 2 ENIGMA1 Study and Open-Label Extension



SOURCE: Dellon ES, et al. New England Journal of Medicine. 2020;383:1624-1634.
a. Blood eosinophils collected just prior to each infusion
b. Inclusive of Lirentelimab exposure during the open-label portion of the Phase 2 ENIGMA 1 study

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Financial Overview & Key Milestones

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Data Catalysts and Expected Milestones

- **Q2 2024:** Report SAD and MAD safety, pharmacokinetics (PK), and pharmacodynamic (PD) results from the Phase 1 IV AK006 trial in healthy volunteers, including data to confirm Siglec-6 receptor occupancy in skin biopsy samples.
- **Q2 2024:** Initiate the randomized, double-blind, placebo-controlled Phase 1 trial of IV AK006 in patients with chronic spontaneous urticaria (CSU).
- **Q3 2024:** Report subcutaneous (SC) AK006 safety, PK, and PD results from the Phase 1 trial in healthy volunteers, including data to confirm Siglec-6 receptor occupancy in skin biopsy samples.
- **YE 2024:** Report topline data from the Phase 1 trial of IV AK006 in patients with CSU.

Balance Sheet and IP Protection

Cash, Cash Equivalents and Investments in Marketable Securities as of December 31, 2023	$170.8 M
– Estimated 2024 cash used in restructuring (lirentelimab closeout, severance and other costs)	$30 M
– Estimated 2024 cash used in ongoing business operations	$55 to $60 M
Estimated, Cash, Cash Equivalents and Investments in Marketable Securities at year end 2024	$81 to $86 M
Common Shares Outstanding as of December 31, 2023	87.8 M

Allakos expects that the restructuring activities will extend the cash runway into mid-2026

AK006 composition of matter to expire in 2042 without extensions

Planning subcutaneous AK006 for Phase 2 studies

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