Enanta Pharmaceuticals

Great Chemistry Cures

# Corporate Presentation

June 18, 2026

# Forward Looking Statements Disclaimer

This presentation contains forward-looking statements concerning our business, operations and financial performance and condition, as well as our plans, objectives and expectations for our research and development programs, our business and the industry in which we operate. Any statements contained herein that are not statements of historical facts may be deemed to be forward-looking statements. In some cases, you can identify forward-looking statements by terminology such as “aim,” “anticipate,” “assume,” “believe,” “contemplate,” “continue,” “could,” “due,” “estimate,” “expect,” “goal,” “intend,” “may,” “objective,” “plan,” “predict,” “potential,” “positioned,” “seek,” “should,” “target,” “will,” “would,” and other similar expressions that are predictions of or indicate future events and future trends, as well as other comparable terminology. These forward-looking statements include, but are not limited to, statements about overall trends, royalty revenue trends, research and clinical development plans and prospects, liquidity and capital needs and other statements of expectations, beliefs, future plans and strategies, anticipated events or trends, and similar expressions. These forward-looking statements are based on our management’s current expectations, estimates, forecasts and projections about our business and the industry in which we operate and our management’s beliefs and assumptions. These forward-looking statements are not guarantees of future performance or results and involve known and unknown risks, uncertainties and other factors that are in some cases beyond our control. As a result, any or all of our forward-looking statements in this presentation may turn out to be inaccurate.

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# Enanta Overview

Clinical-stage biotechnology company dedicated to creating **small molecule drugs for virology & immunology indications**

Founded: **1995**

Public: **2013**

**WHOLLY OWNED PROGRAMS**

**4** Clinical-stage

**2** Preclinical

All compounds discovered in house, leveraging **deep expertise** in **medicinal chemistry, drug discovery & development**

**STRONG CASH POSITION**

- Ongoing HCV royalties
- **$227M in cash** at March 31, 2026

viekira pak®
ombitasvir, paritaprevir and ritonavir tablets; dasabuvir tablets

MAVYRET® glecaprevir/pibrentasvir 100 mg/40 mg tablets

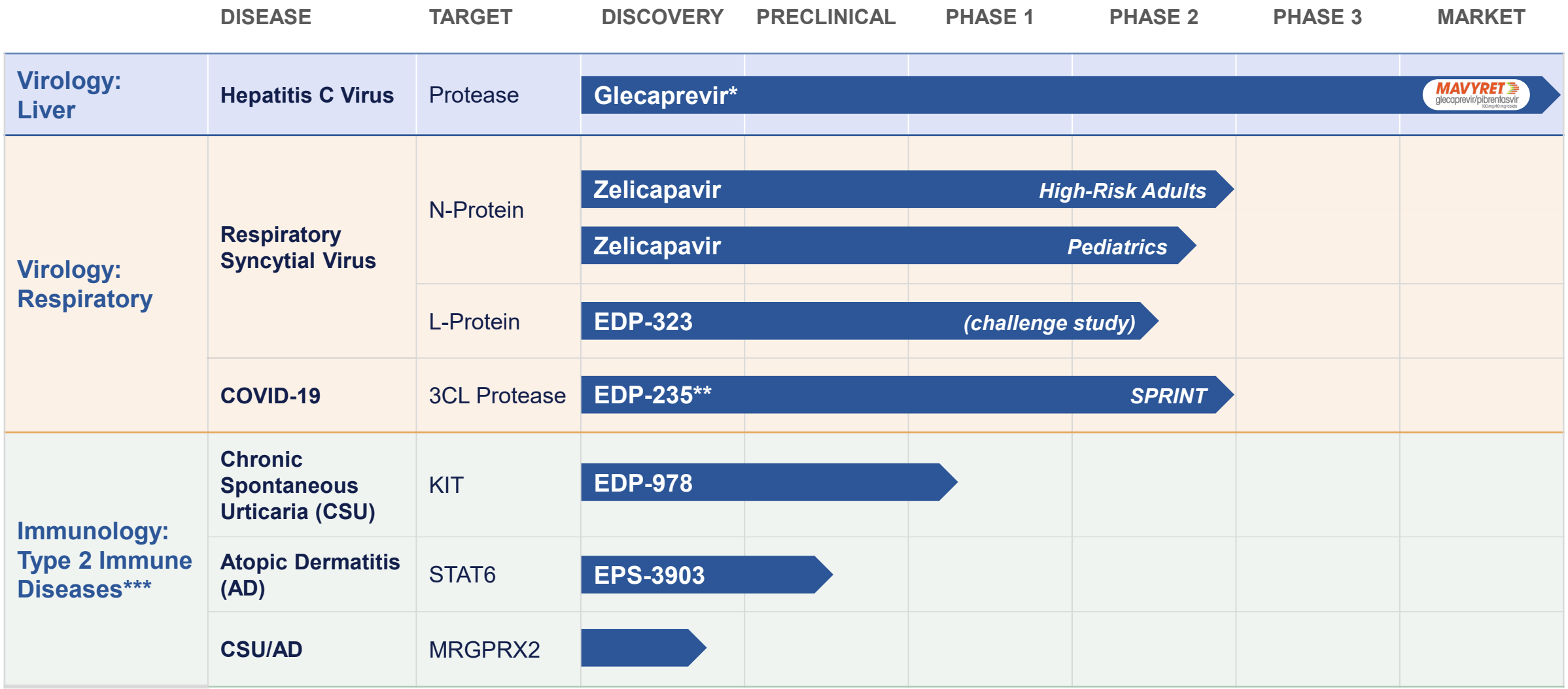
MAVIRET™ glecaprevir/pibrentasvir

**2** products approved with abbvie

**CURED**

**>1 million patients** with Hepatitis C Virus

# Enanta Pipeline




*Fixed-dose antiviral combination contains glecaprevir and AbbVie's NS5A inhibitor, pibrentasvir. Marketed by AbbVie as MAVYRET® (U.S.) and MAVIRET® (ex-U.S.).
**Continued development dependent on a future collaboration.
***Initial indications. Potential future indications include Asthma, Chronic Inducible Urticaria (CIndU), Eosinophilic Esophagitis (EoE); Prurigo Nodularis (PN), Migraine and others.

# Virology: Respiratory Syncytial Virus

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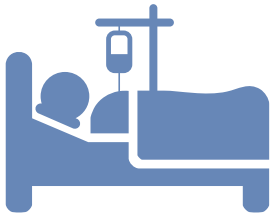
# Respiratory Syncytial Virus (RSV)

Causes severe lung infections, including bronchiolitis (infection of small airways in the lungs) and pneumonia. No safe and effective treatments are currently approved.

## RSV Burden Estimates[1] (2024 - 2025 U.S.)

Outpatient visits

**up to 6.8M**



Hospitalizations

**up to 370K**

Deaths

**up to 24K**

### Populations at higher risk for severe illness

- Pediatrics (infants and young children)
- High-risk adults (e.g.; >65 yrs, COPD, asthma, CHF)
- Immunocompromised (e.g.; HIV, transplant)

### Significant Unmet Need for Antivirals

- Adult vaccines have sub-optimal adoption
  - Not recommended for all FDA-approved patient groups
  - Vaccine adoption for elderly: ~35% (shingles[2]) to <50% (flu[3])
- Pediatric prophylaxis only provides passive immunity; will shift first infection to next season
  - Antibody approach has a low barrier to resistance
- Breakthrough infections can occur despite prophylaxis

### Growing Momentum for RSV Diagnosis, Enabling a Test-to-Treat Model



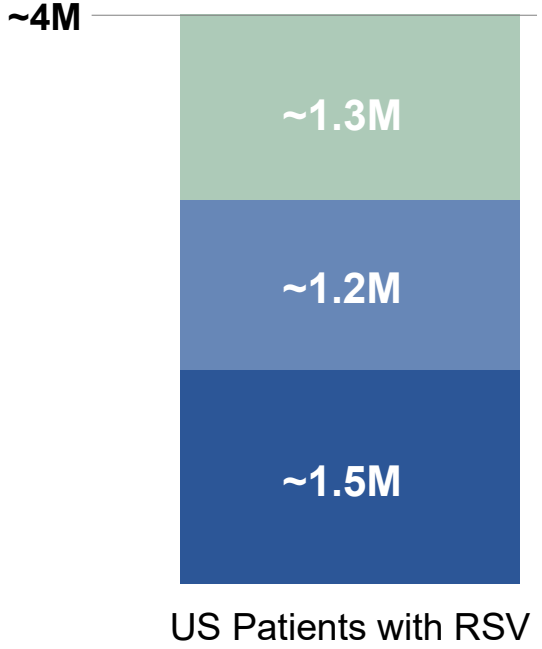
COPD: Chronic Obstructive Pulmonary Disease; CHF: Congestive Heart Failure; HIV: Human Immunodeficiency Virus

1. CDC Preliminary Estimates of RSV Burden for 2024-2025; for period: 9/29/24-9/27/25 2. Terlizzi EP et al. *NCHS Data Brief*. 2020; Age 60+ 3. CDC Influenza Vaccination Coverage, Adults 65+

# Multi-Billion Dollar Global Market Opportunity for an RSV Antiviral

**Up to 4M Addressable Patients with RSV in the United States[1]**




***Additional High-Risk Adults***:
CKD, IC, T2DM, ONC

***HR3 Adults***:
CHF, COPD, Age ≥ 75 yrs

***Pediatrics***:
Age < 3 yrs

***Multi-Billion $ Global Revenue Potential[2]***

***~$2.6B–3.5B***

- Additional High-Risk Adults
- HR3 Adults
- Pediatrics

*~$1.8B–2.4B*

CHF: Congestive Heart Failure, CKD: Chronic Kidney Disease, COPD: Chronic Obstructive Pulmonary Disease, HR: High Risk; IC: Immunocompromised, ONC: Oncology, T2DM: Type 2 Diabetes Mellitus

1. Addressable patients based on outpatient visits adjusted for: anticipated adoption & efficacy of RSV vaccination/prophylaxis; presence of selected comorbidities for adults. Pediatrics: Lively JY et al. *J Pediatr Infect Dis Soc* 2019; Hsiao A et al. *Pediatrics*. 2025; ABRYSVO USPI Adults: Landi SN et al. *JAMA Netw Open*. 2024; Horn EK et al. *Influenza Other Respir Viruses*. 2025; McLaughlin JM et al. *Open Forum Infect Dis*. 2022
2. Peak sales forecast (2035); Based on primary market research and Enanta internal modeling that accounts for diagnosis, treatment and prescription fill rates.

# RSV Development Goal:
## Treatment for Patients at High-Risk for Severe RSV Infection

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**Leading portfolio of RSV replication inhibitors with potential for first-in-disease (zelicapavir) and best-in-disease (EDP-323) treatments and ability for combination**

| | | |
|---|---|---|
| **Zelicapavir High-Risk Adult Phase 2b Study** | Age ≥65 years<br>Chronic heart or lung disease (e.g. COPD, CHF, asthma) | ✓ Positive Phase 2b Results |
| **Zelicapavir Pediatric Phase 2 Study** | Infants and young children | ✓ Positive Phase 2 Results |

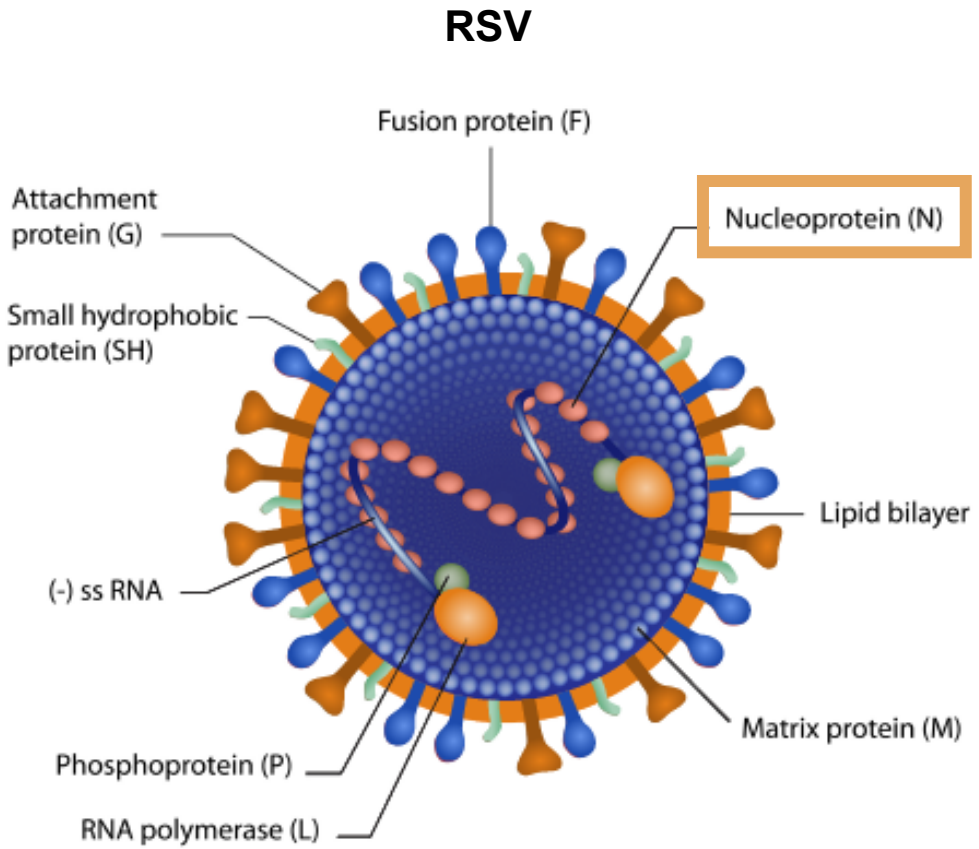
High-risk populations have reduced RSV immunity, resulting in a higher and longer duration of viral load and greater disease severity

COPD: Chronic Obstructive Pulmonary Disease; CHF: Congestive Heart Failure

# Zelicapavir (EDP-938):
## N-Protein Inhibitor for RSV

- Only N-inhibitor in clinical development for RSV
  - Replication inhibitor: shuts down the production of new virions (vs. fusion inhibitors which block viral entry)
- Granted Fast Track designation by the FDA
- Strong preclinical profile
  - Nanomolar potency against RSV-A and RSV-B
  - Antiviral potency across all clinical isolates tested
  - High barrier to resistance
  - Synergistic activity and no cross-resistance with other drug mechanisms (e.g. L-inhibitors)
- Favorable safety and efficacy profile in clinical studies
  - Challenge study showed statistically significant ($p<0.001$) reduction in viral load and clinical symptoms
  - High barrier to the development of clinical resistance
  - Well-tolerated in more than 700 people dosed



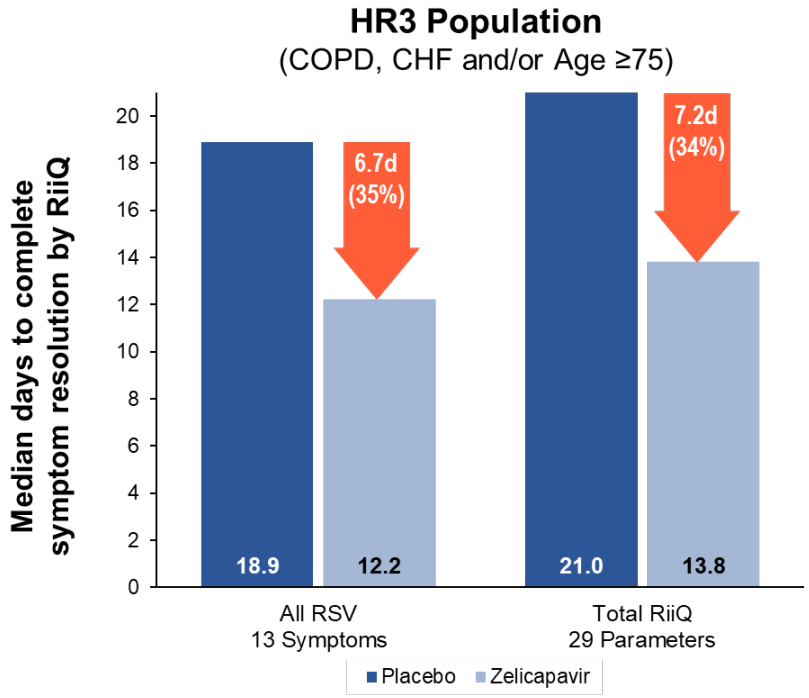

 

# Zelicapavir High-Risk Adult Program:
## Phase 2b Study Summary

- Demonstrated compelling results on multiple clinically meaningful endpoints measuring different aspects of RSV disease
  - ✓ Up to one week improvement in complete RiiQ™ symptom resolution
  - ✓ Statistically significant 2-day improvement in PGI-S
  - ✓ Lower hospitalization rate

| Hospitalizations | Placebo | Zelicapavir |
|---|---|---|
| All-cause | 5.0% (3/60) | 1.7% (2/115) |
| RSV-associated | 5.0% (3/60) | 0% (0/115) |

- Robust antiviral effect
- Well-tolerated, with a favorable safety profile




**Data support advancement of zelicapavir into registrational study of high-risk adults**

RiiQ: Respiratory Infection Intensity and Impact Questionnaire; PGI-S: Patient Global Impression of Severity; COPD: Chronic Obstructive Pulmonary Disease; CHF: Congestive Heart Failure

# Zelicapavir High-Risk Adult (HR3) Program:
## Phase 2b/3 Global Registrational Study Design

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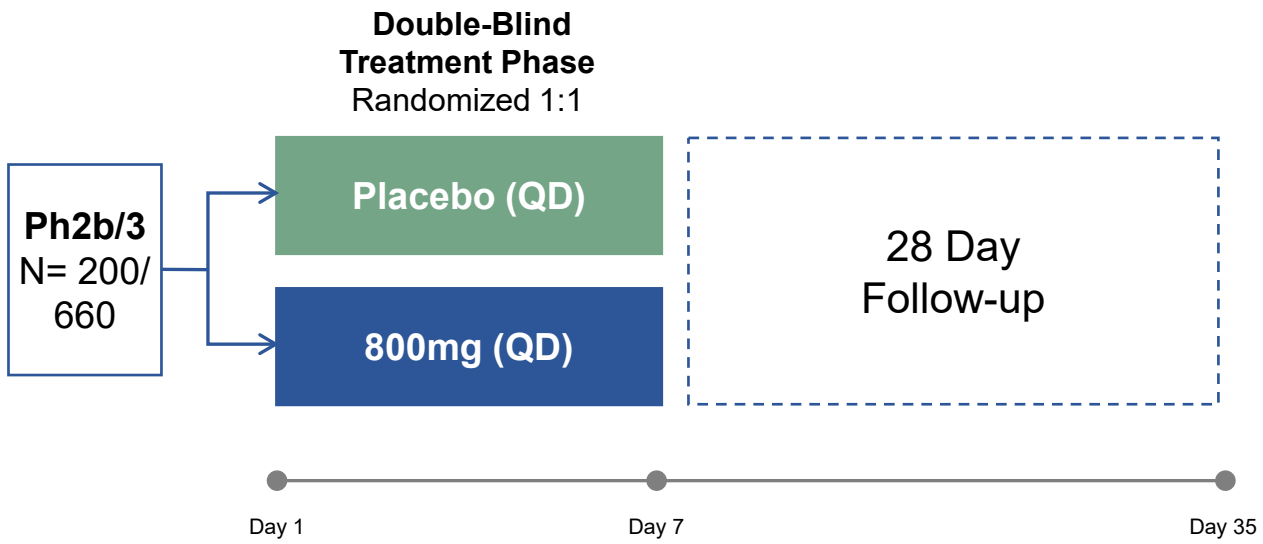
**Trial Design**

### Inclusion Criteria

- Adult outpatients with RSV*
- Symptom onset within 3 days
- At least one of the following:
  - COPD
  - Congestive heart failure
  - Age ≥75




### Primary Objective*

Time to complete resolution of 13 RSV symptoms as measured by RiiQ™

### Secondary Objective

Reduction in hospitalization rate due to RSV

*Other Secondary Objectives:* Additional clinical efficacy measures, virology, safety

**Study to start in 4Q 2026; Phase 2b data in 2027 to confirm endpoints and sizing**

* Patients who have received an RSV vaccine are capped at 10%; Primary endpoint is on MITT of unvaccinated patients (vaccinated patients included in a separate supportive ITT analysis)
COPD: Chronic Obstructive Pulmonary Disease; PGI-S: Patient Global Impression of Severity; QD: Once-daily; RiiQ: Respiratory Infection Intensity and Impact Questionnaire

# Zelicapavir Pediatric Program:
## First-in-Pediatric Phase 2 Study Summary

- Well-tolerated, with favorable safety profile
  - No adverse events leading to treatment discontinuation or study withdrawal
- Antiviral effect observed for the primary and secondary virology endpoints in overall population
- Viral load decline of 1.4 log at the end of treatment in Part 2
- Viral load decline of 1.2 log at Day 5 observed in prespecified subset of patients randomized within 3 days of symptom onset
- RSV Signs/Symptoms
  - ReSViNET: Reduced the time to complete symptom resolution by 1.6 days and 3.7 days
  - RESOLVE-P: Trend toward greater sign/symptom reduction with zelicapavir in a small dataset

**Primary Objectives of Study**

- ✓ **Overall:** Antiviral activity of zelicapavir across all patients
- ✓ **Part 1:** Safety and PK
- ✓ **Part 2:** Antiviral activity

**Data support further clinical development of zelicapavir in pediatrics**

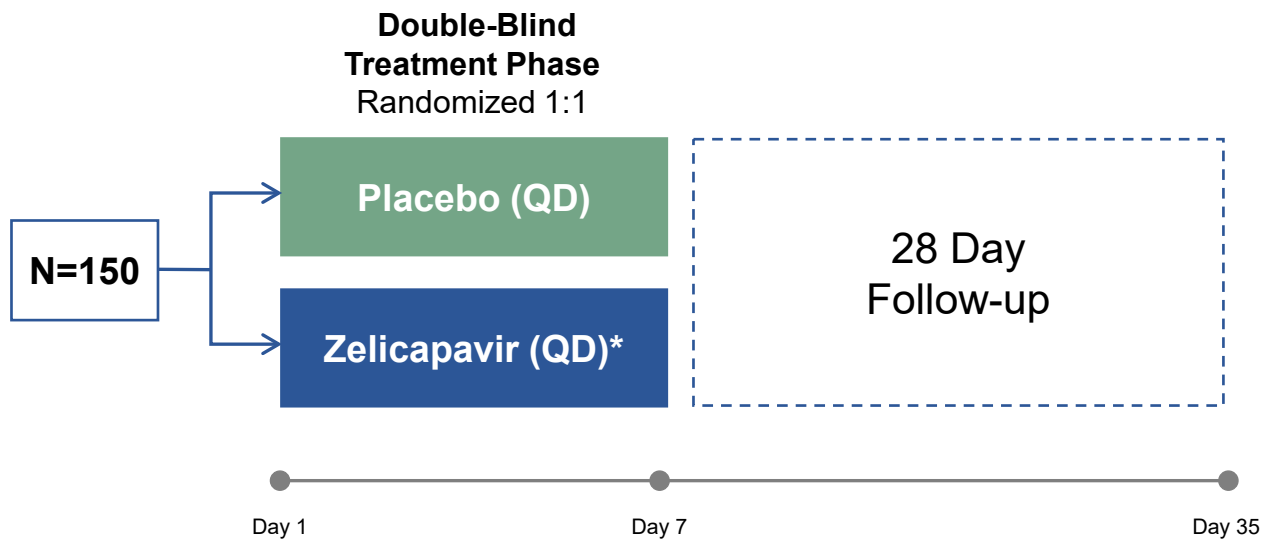
RESOLVE-P: RESpiratory ObservabLE Reported Outcome-Pediatric; Enanta's proprietary ObsRO 

# Zelicapavir Pediatric Program:
## Phase 2b Study Design

Trial Design

**Inclusion Criteria**

- Pediatric inpatients and outpatients with RSV
- Symptom onset within 3 days
- Age 28 days - 36 months




**Primary Objective**

Time to complete symptom resolution as measured by RESOLVE-P

**Secondary Objective**

Safety, virology, additional clinical efficacy measures

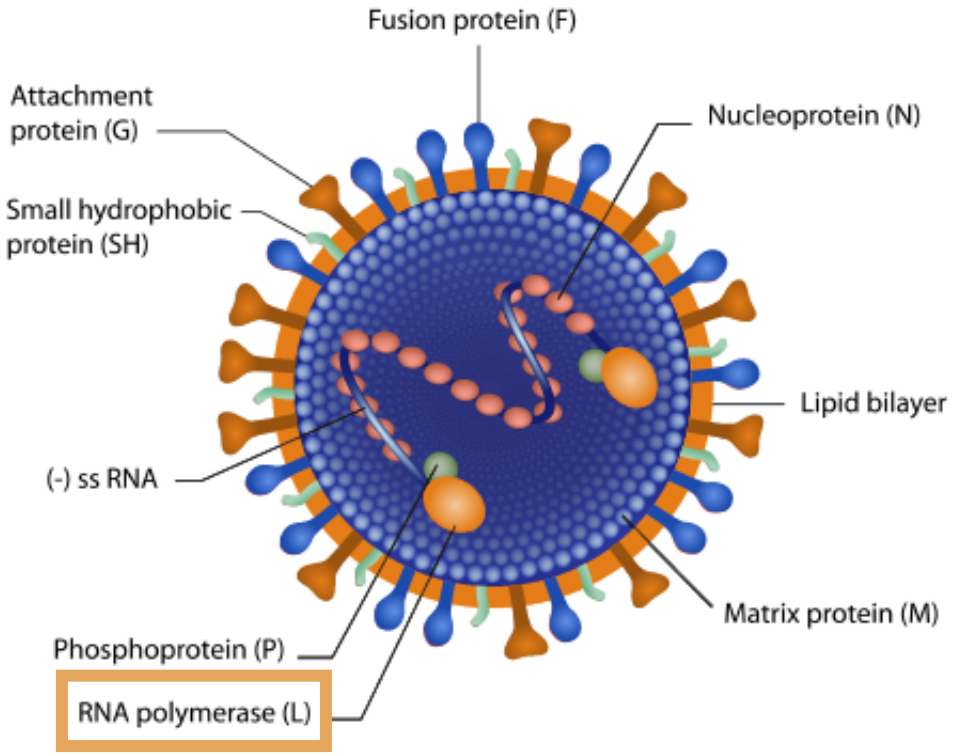
**Study to start in 3Q 2026; Phase 2b topline data in 2027**

*Doses are 5mg/kg for <12 months old; 7.5mg/kg for ≥12 months old
QD: Once-daily; RESOLVE-P: RESpiratory ObservabLE Reported Outcome-Pediatric; Enanta's proprietary ObsRO 

# EDP-323:
## RSV L-Protein Inhibitor

- Direct-acting antiviral targeting the L-protein
  - Replication inhibitor: shuts down the production of new virions (vs. fusion inhibitors which block viral entry)
- Granted Fast Track designation by the FDA
- Sub-nanomolar potency against RSV-A and B
- Protects mice in a dose-dependent manner from RSV infection
- Potential to be used alone or in combination
  - Additive to synergistic activity with zelicapavir
  - No cross resistance expected with other mechanisms
- Phase 1 indicated 200mg or 600mg once-daily as potential safe and efficacious doses

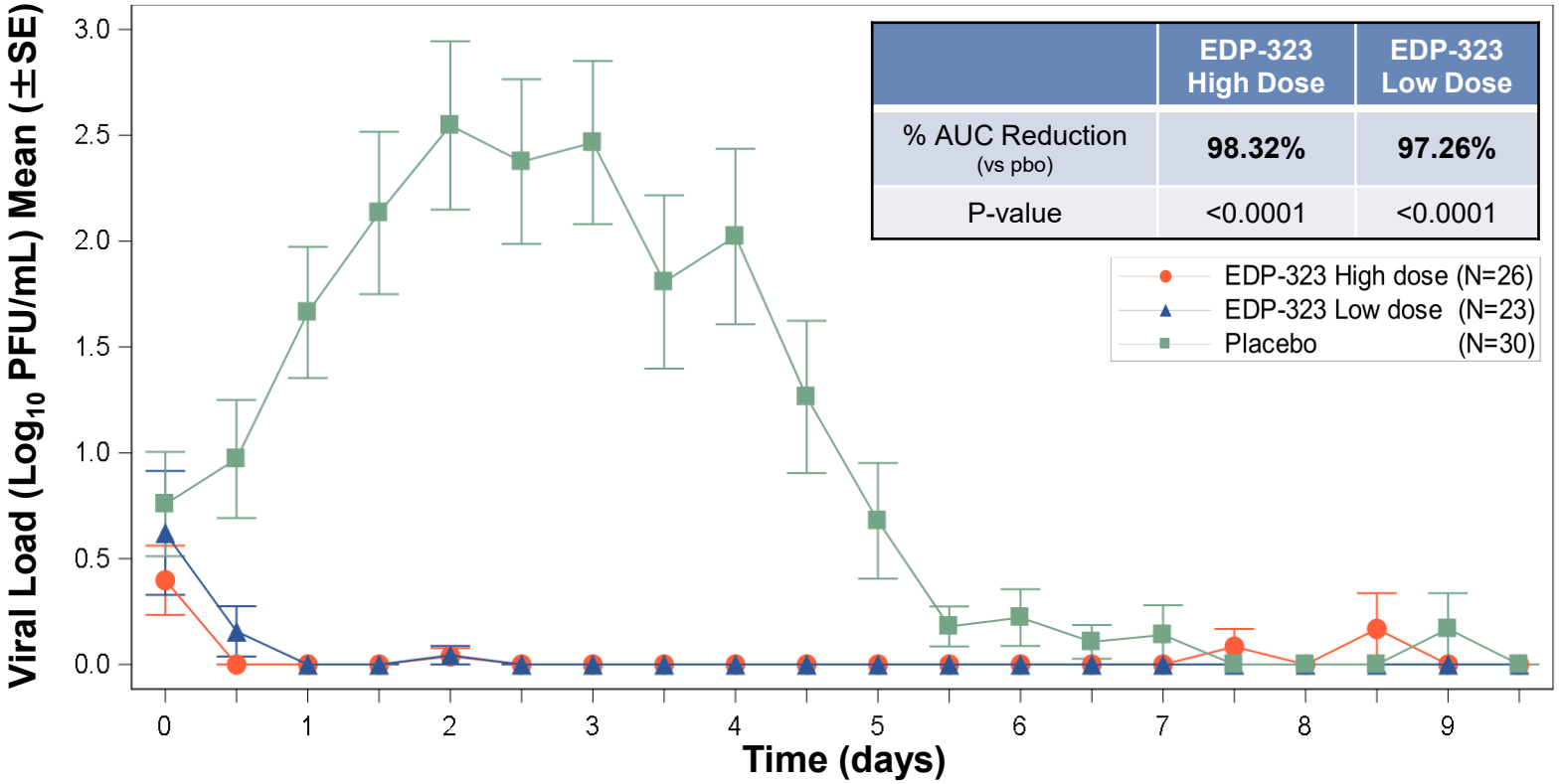
**RSV**



# EDP-323:
## Phase 2a RSV Challenge Study Conclusions

- Well-tolerated with safety profile similar to placebo
- Primary and key secondary efficacy endpoints achieved with high statistical significance at both dose levels compared to placebo:
  - ✓ Reduction in viral load of 85-87%
  - ✓ Reduction in viral culture of 97-98%
  - ✓ Alleviation of clinical symptoms by 66-78%




| | EDP-323 High Dose | EDP-323 Low Dose |
|---|---|---|
| % AUC Reduction (vs pbo) | **98.32%** | **97.26%** |
| P-value | <0.0001 | <0.0001 |

**Data support further clinical development of EDP-323**

VL AUC: Viral Load Area Under the Curve (hours x log10 copies/mL); PFU: Plaque Forming Units; SE: Standard Error

# Immunology

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# Building Enanta's Immunology Portfolio:
## Focusing on Best-in-Disease Treatments

Exploit novel biology to select targets with **potential in multiple indications**

Leverage innovative chemistry to develop **best-in-class** small molecule drugs

| | |
|---|---|
| Strong Target Rationale | Clinical, genetic, or pathway validation to link target to disease |
| Clear Means to Differentiation | Yet to be drugged, drugged by injectables or suboptimal orals |
| Expedited Clinical Path to POC | Predictive biomarkers or early clinical signals derisk development |
| Significant Unmet Need | Differentiated value proposition; Large market opportunity |

 

# Immunology Portfolio:
## Multiple Targets Addressing Type 2 Immune Diseases

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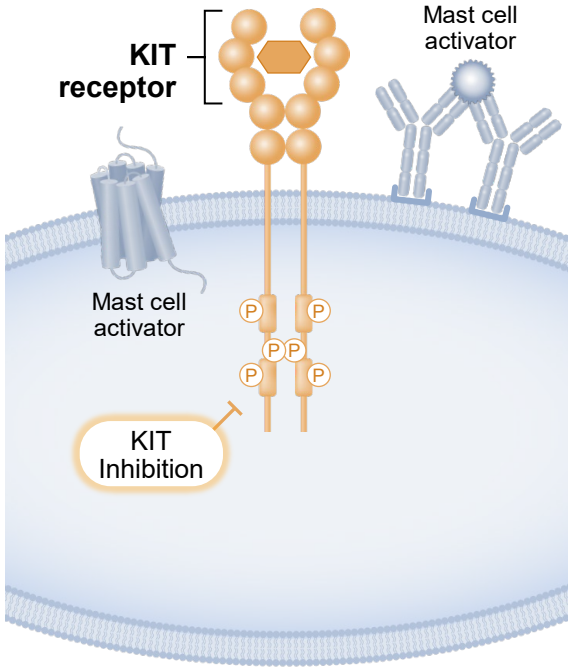
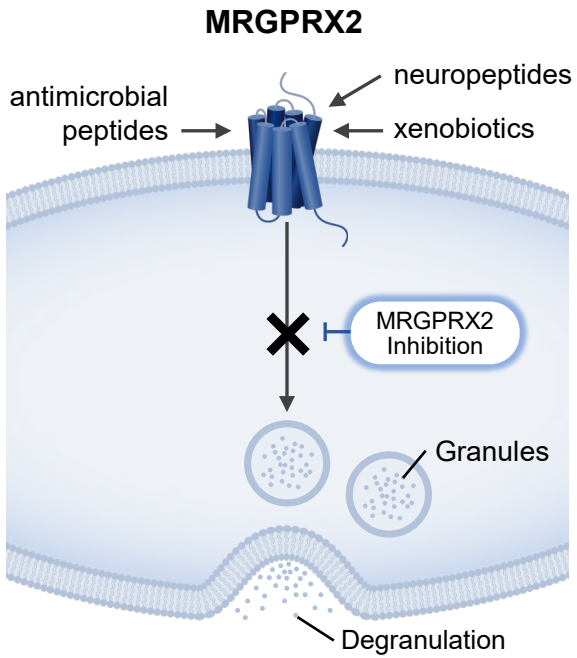
### KIT



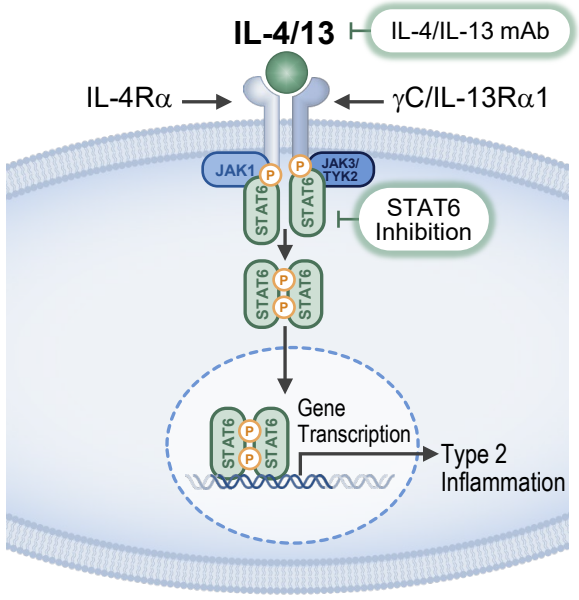

**KIT** Inhibition blocks cell survival signal resulting in apoptosis and cell death

### MRGPRX2




**MRGPRX2** Inhibition prevents activation and degranulation in mast cells

### STAT6




**STAT6** Inhibition impedes type 2 inflammation

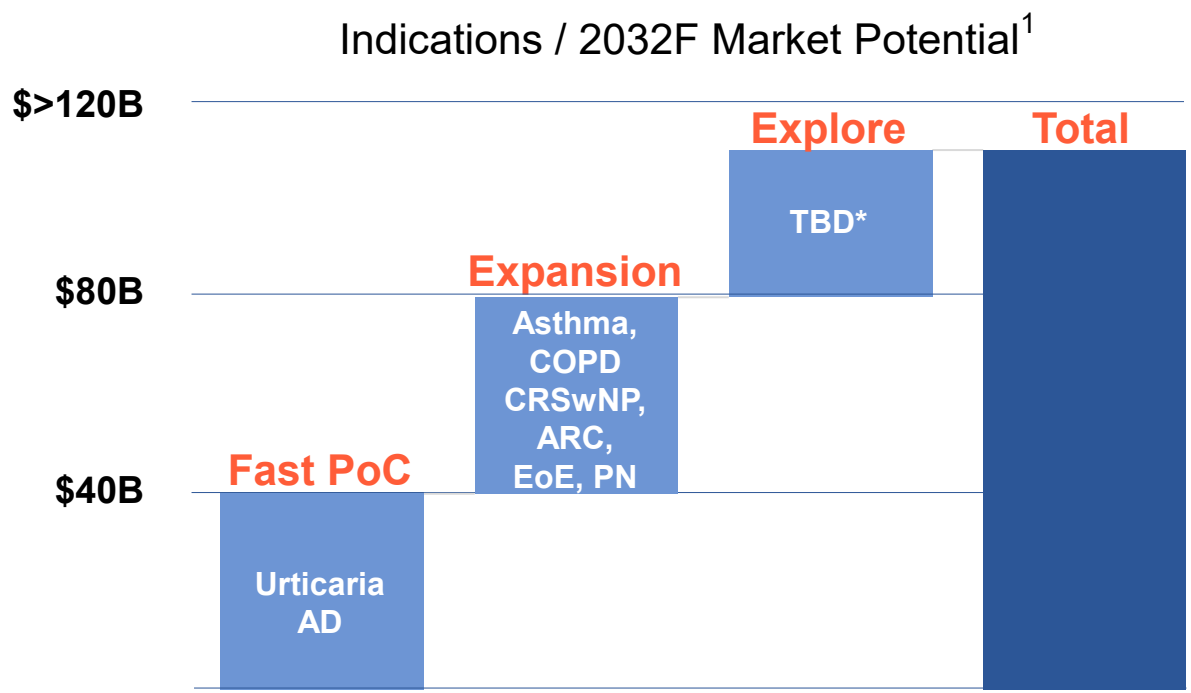
**Potential Indications:** Atopic Dermatitis, Asthma, CSU, CIndU, COPD, EoE, PN, CRSwNP, Migraine, Others

CSU: Chronic Spontaneous Urticaria; CIndU: Chronic Inducible Urticaria, CRSwNP: Chronic Rhinosinusitis with Nasal Polyps; EoE: Eosinophilic Esophagitis, PN: Prurigo Nodularis

# Immunology Portfolio:
## Addressing unmet need in multiple Type 2 immune-driven diseases

- Type 2 inflammation is characterized by overproduction of IL-4, IL-5, IL-13, and IgE which recruits and activates Th2 CD4 T cells, B cells, mast cells, eosinophils, and basophils
- Potential to treat **broad patient populations** across numerous disease areas




AD: Atopic Dermatitis; ARC: Allergic Rhinoconjunctivitis; COPD: Chronic Obstructive Pulmonary Disease; CRSwNP: Chronic Rhinosinusitis with Nasal Polyps; EoE: Eosinophilic Esophagitis; PN: Prurigo Nodularis
* Includes expansion into GI and neurology indications thought to be associated with mast cells
Source: 1. Evaluate Pharma, © Evaluate Ltd, September 2025, www.evaluate.com

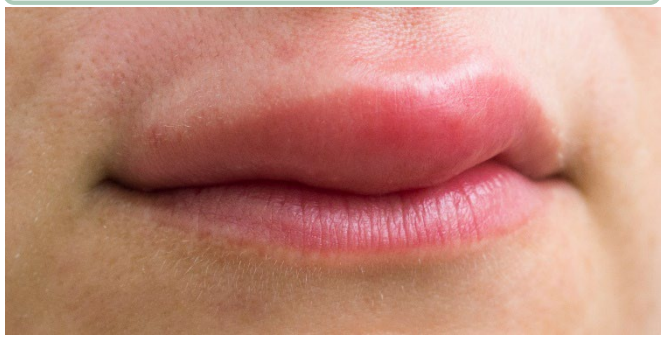
 

# Chronic Spontaneous Urticaria (CSU):
## KIT and MRGPRX2 Inhibitor Initial Focus for Clinical Proof of Concept

- Severely debilitating, chronic inflammatory skin disease
  - Driven by mast cell activation, triggering release of inflammatory mediators[1]
  - Quality of life impacts beyond the skin: sleep disturbances, fatigue, irritability, anxiety and depression[2]
- Affects ~0.5-1% of the global population[1]
- Substantial unmet need for efficacious oral agent
  - ~50% not controlled with antihistamines[1,3]

**Hives** | **Itch** | **Erythema**

**Angioedema**

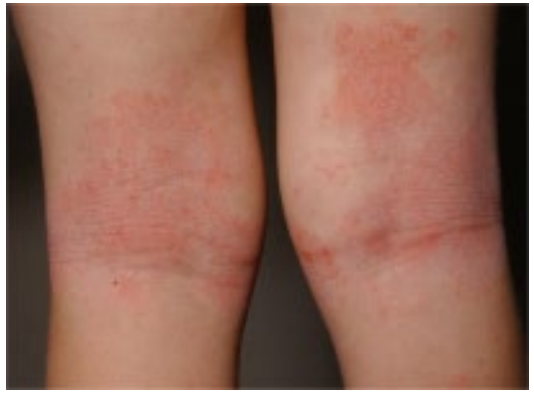


1. Kaplan A et al. *Allergy*. 2023; 78(2): 389–401. 2. Yosipovitch et al. *Dermatol Ther*. 2023; 13(8): 1647–1660. 3. H1 antihistamines up to 4x standard dose.

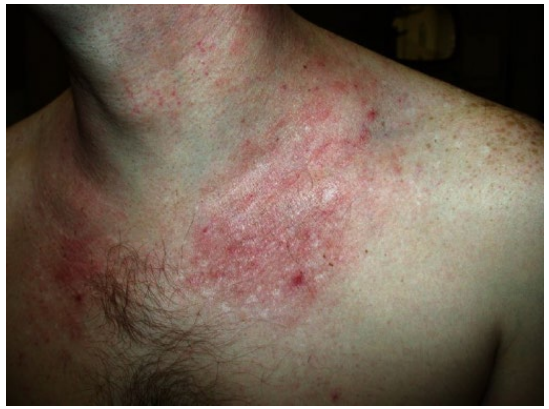
# Atopic Dermatitis (AD):
## STAT6 Inhibitor Initial Focus for Clinical Proof of Concept

- Chronic dermatological disease characterized by dry, red, inflamed, irritated and itchy skin
  - Driven by Th2 immune dysregulation[1]
  - Quality of life impacts beyond the skin: limited lifestyle, avoidance of social interaction and impacted activities[1]



- 7.3% of U.S. adults, ~40% have moderate-severe symptoms[2]

- Significant need for efficacious and safe oral agents
  - Market dominated by mAb (IL-4/13)
  - Oral JAKi use <10%[3]
    - Boxed warning: serious infections, mortality, malignancy, major adverse cardiac events, thrombosis[4]



1. Silverberg JI et al. *Annals of Allergy, Asthma & Immunol*. 2018. 2. Asthma and Allergy Foundation of America: https://aafa.org/asthma-allergy-research/our-research/atopic-dermatitis-in-america/. 3. Market share ($) from Evaluate Pharma, © Evaluate Ltd, September 2024, www.evaluate.com 4.Prescribing Information for RINVOQ & CIBINQO

# Immunology: KIT Inhibitor

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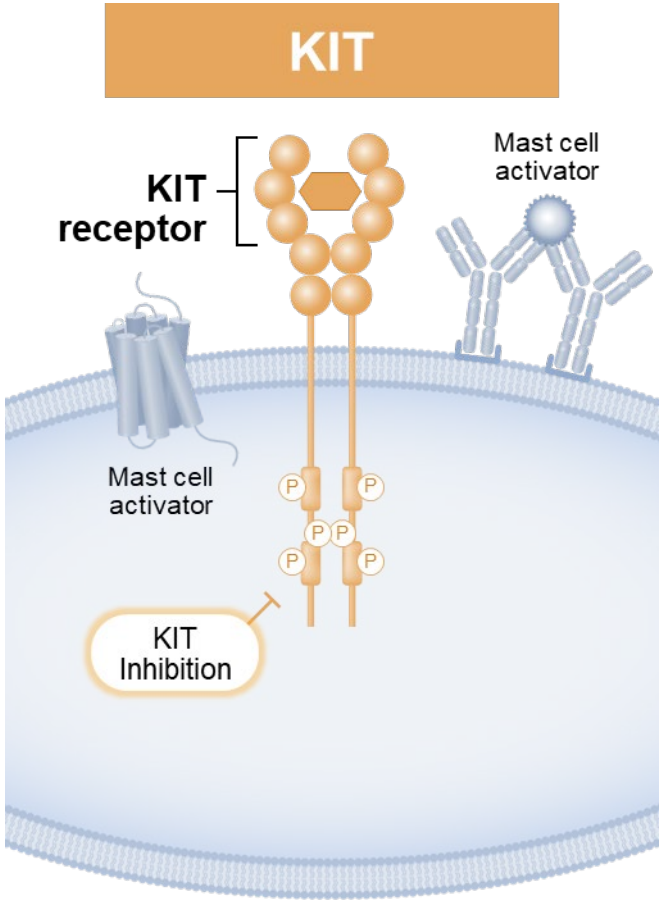
# KIT Inhibitors Offer Potential for Differentiated Efficacy

- **Mast cells:** primary driver of inflammation in skin, and implicated in multiple allergic diseases
  - Urticaria, asthma, prurigo nodularis (PN) and others
- **KIT:** well-characterized receptor tyrosine kinase critical for regulating mast cell activity
- **KIT inhibitors:** potential for best-in-disease efficacy
  - Directly reduces quantity of mast cells through apoptosis and depletion, addressing key disease driver
  - Current therapy reduces mast cell activator levels or downstream mediators, but not mast cells directly
  - Positive proof-of-concept in urticaria and PN with anti-KIT mAb



# Goal of Oral KIT Inhibitor Program:
## Develop Best-in-Class Treatment for Mast Cell Mediated Diseases

- EDP-978 selected as clinical candidate
  - Inhibits KIT with nanomolar potency in both binding and cellular assays
  - Demonstrates sub-nanomolar activity *in vivo*
  - Highly selective for KIT versus other kinases
  - Good *in vitro* and *in vivo* ADME properties
    - Well-absorbed with good plasma exposure across preclinical species
    - No GSH adducts (or reactive metabolites) detected *in vitro* or *in vivo*, minimizing metabolic/safety issues
    - Low drug-drug interaction potential via CYP inhibition or induction
    - Excellent metabolic stability with once-daily dosing potential in human

- Topline Phase 1 data targeted for 4Q 2026

# EDP-978 Exhibits Potent Inhibition and Good Selectivity of KIT *In Vitro* and *In Vivo*

- Nanomolar inhibition in cellular assays with an $EC_{50}$ of **1.6 – 3.4nM**

| Binding or Cellular Assay* | $EC_{50}$ (nM) |
|---|---|
| KINOMEscan Kd | 0.3 |
| M-07e Phosphorylated KIT | 1.6 |
| UT-7 Cell Proliferation | 2.8 |
| KIT Ba/F3 Cell Proliferation | 3.3 |
| $CD34^+$ Derived Mast Cell Degranulation | 3.4 |

*M-07e and UT-7 cells endogenously express KIT; Ba/F3 cells are engineered to express KIT

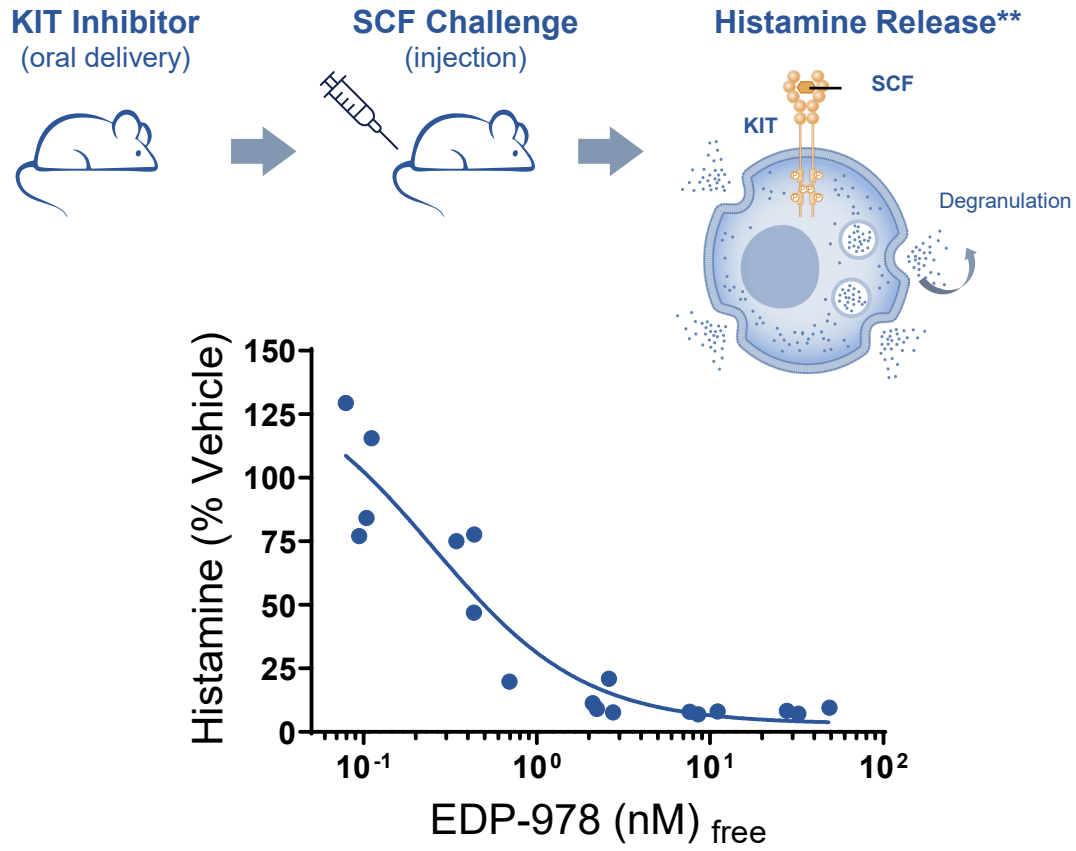
- Greater than 200-fold selectivity for KIT over other KIT family members

| Selectivity | EDP-978 |
|---|---|
| S(10) @ 1 µM | 0.02 |
| CSF1R* | >200x |
| PDGFRα* | >900x |
| PDGFRβ* | >800x |
| FLT3 Kd | >30,000x |

*Cellular selectivity calculated as ratio of $EC_{50}$ in kinase dependent Ba/F3 cells

S(10) @ 1 µM: selectivity score at a concentration of 1 µM; CSF1R: colony stimulating factor 1 receptor; PDGFRA/B: platelet-derived growth factor receptor alpha/beta; FLT3: FMS-like tyrosine kinase 3

- Inhibits SCF-mediated histamine release in mice with an $EC_{50}$ (free drug) of **0.25nM**




**KIT binding to its ligand SCF (Stem Cell Factor) *in vivo* triggers mast cell degranulation with histamine release

# Immunology: STAT6 Inhibitor

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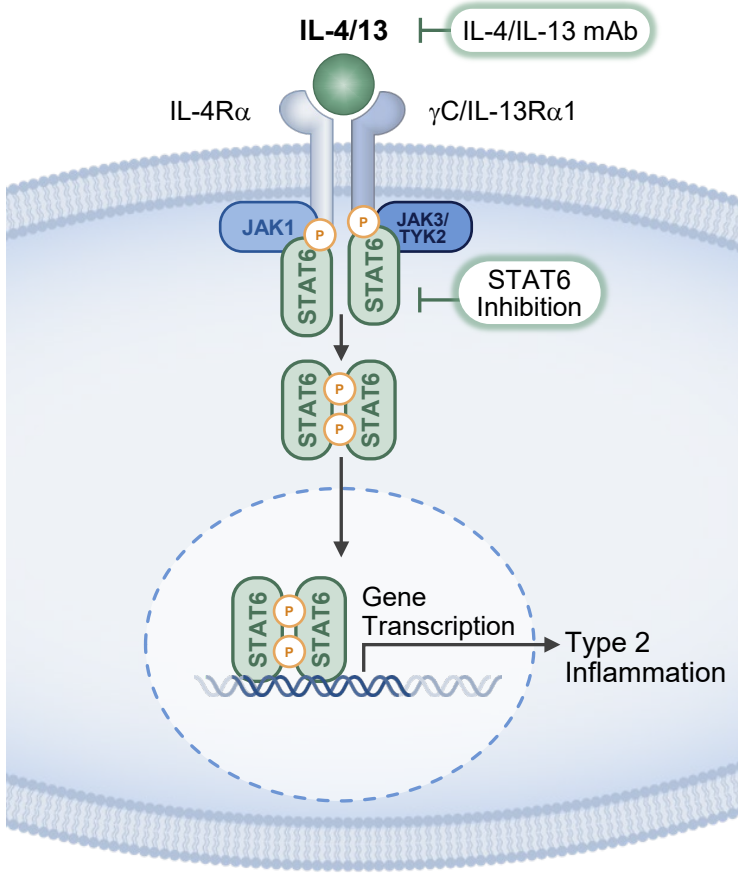
# STAT6 Inhibitors Offer Potential for an "Oral DUPIXENT®"

- **Th2 dysregulation** drives allergic and autoimmune diseases including atopic dermatitis and asthma
  - Characterized by IL-4 & IL-13 overproduction
- **STAT6**: transcription factor responsible for IL-4/IL-13 signaling, which drives a Th2 dominant phenotype
  - STAT6 gain-of-function variants result in severe atopic dermatitis[1]
  - STAT6 loss-of-function protects against type 2 high asthma[2]
  - Inhibition of the IL-4/13 pathway is clinically validated
- **STAT6 inhibitors**: potential for an "oral DUPIXENT"
  - Blocks IL-4/13 signaling pathway
  - Reduces inflammation in Th2 driven preclinical models
  - No oral therapy selectively targeting IL-4/13 pathway available




1. Suratannon et al. *J Allergy Clin Immunol*. 2023. 2. Kristjansdottir et al. *J Allergy Clin Immunol*. 2024.

# Goal of Oral STAT6 Inhibitor Program:
## Develop Best-in-Class Oral STAT6 Inhibitors

- EPS-3903 selected as development candidate
  - Favorable *in vitro* and *in vivo* ADME properties supporting once daily dosing
  - Inhibits STAT6 with nanomolar potency in both binding and cellular assays
  - Highly selective for STAT6 versus other STATs
  - Rapid, continuous and complete (>90%) inhibition of pSTAT6 after oral dosing in mice
  - *In vivo* efficacy comparable to dupilumab or an anti-mouse IL-4/IL-13 antibody in multiple disease models of asthma (ovalbumin, house dust mite) and atopic dermatitis (MC903)

- Initiated IND-enabling activities with goal of IND filing in 2H 2026

# EPS-3903 Demonstrates Potent Activity & Good Selectivity

- Nanomolar inhibition in biochemical (Kd = 0.4 nM) and cellular assays
  - Inhibits phosphorylation of STAT6 and STAT6-mediated cellular proliferation
  - Prevents production of STAT6-driven biomarkers of type 2 inflammation

| Cellular Assay (IL-4/IL-13 Induced) | EPS-3903 | Upadacitinib |
|---|---|---|
| | $EC_{50}$ (nM) | |
| pSTAT6 (hPBMC) | 4 | 10 |
| Cell Proliferation (TF-1) | 8 | 16 |
| TARC (hPBMC) | 16 | 20 |
| Periostin (primary bronchial cells) | 3 | 11 |

TF-1: human erythroleukemic cell line, dependent on STAT6 signaling for cell growth

- No inhibition of other STATs observed in human PBMCs
- >1000x biochemical selectivity for STAT6 over other STATs
- Selectivity much improved compared to JAK inhibitors

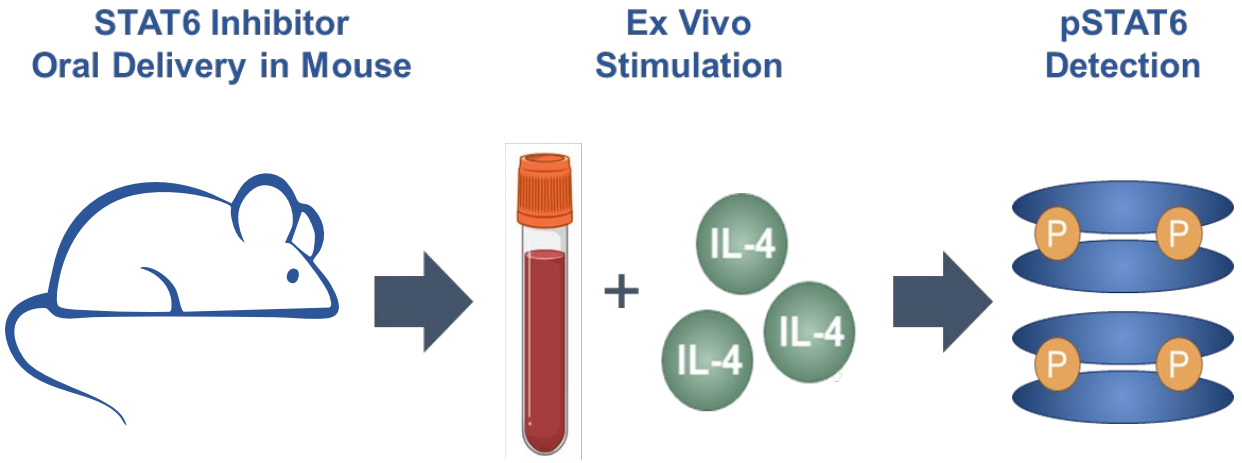
| Selectivity in hPBMCs | EPS-3903 | Upadacitinib |
|---|---|---|
| | $EC_{50}$ (nM) | |
| pSTAT1 | >5000 | 1 |
| pSTAT2 | >5000 | 8 |
| pSTAT3 | >5000 | 24 |
| pSTAT4 | >5000 | 195 |
| pSTAT5 | >5000 | 16 |
| pSTAT6 | 4 | 10 |

Upadacitinib: JAK Inhibitor ; hPBMC: Human Peripheral Blood Mononuclear Cells ; pSTAT6: Phosphorylated STAT6; TARC: Thymus and Activation-regulated Chemokine

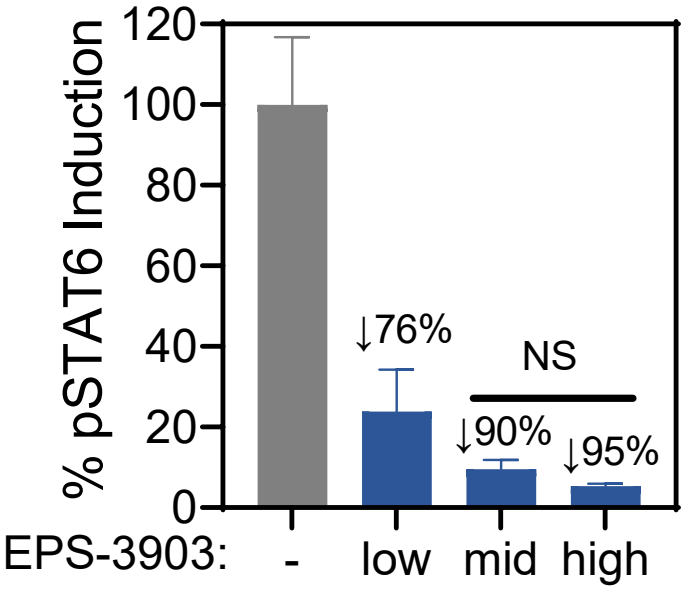
 

# EPS-3903 Results in Complete Inhibition of pSTAT6 in Mouse Model

- *In vivo* STAT6 target engagement after a single oral dose
- Rapid, complete and continuous (>90%) inhibition of phosphorylated STAT6 sustained for 24 hours




**24 hours post-oral dose in IL-4-stimulated mouse whole blood**



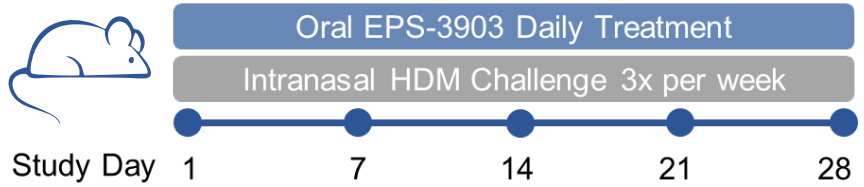

NS=not statistically different

pSTAT6: Phosphorylated STAT6

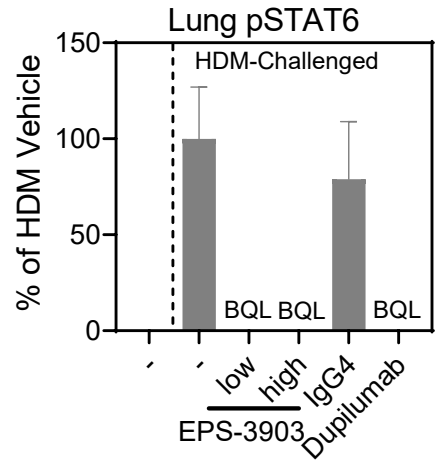
# EPS-3903 Shows Comparable Efficacy to Dupilumab in a House Dust Mite (HDM) Challenge Asthma Model

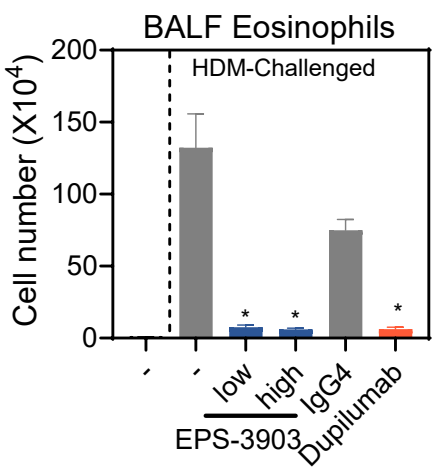
- HDM model in transgenic mice expressing human IL-4/IL-4Rα triggers allergic airway inflammation that mimics key aspects of asthma

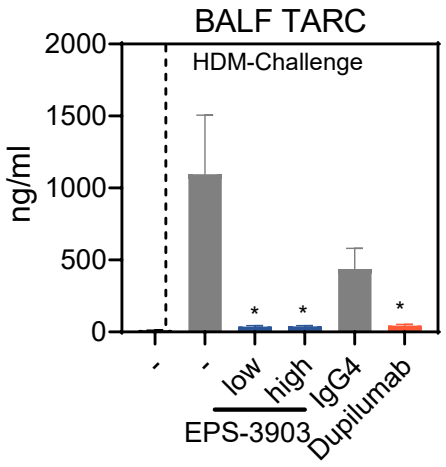


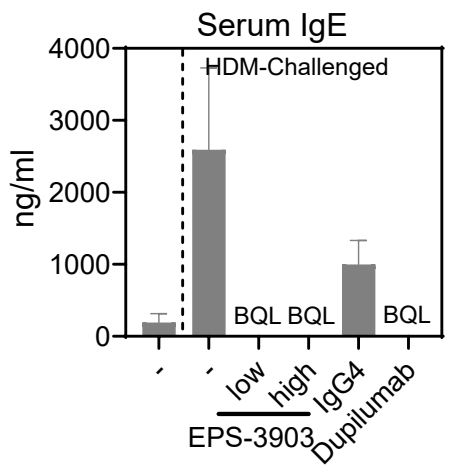

## EPS-3903 efficacy comparable to dupilumab

- >90% inhibition of pSTAT6 in lung
- Decreased type 2 inflammation
  - BALF Eosinophils, TARC, Serum IgE
  - Improved histological scoring
    - Reduced bronchiolar hyperplasia, inflammatory infiltrates, airway wall thickness



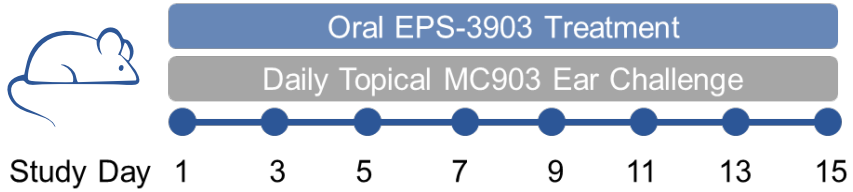

*p<0.05 vs Corresponding Vehicle

pSTAT6: Phosphorylated STAT6; BALF: Bronchioalveolar Lavage Fluid; TARC: Thymus and Activation-regulated Chemokine; BQL: Below Quantification Limit

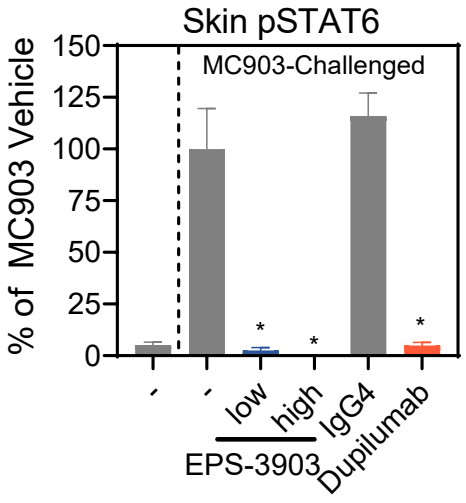
# EPS-3903 Demonstrates Comparable Efficacy to Dupilumab in a MC903-Driven Atopic Dermatitis Mouse Model

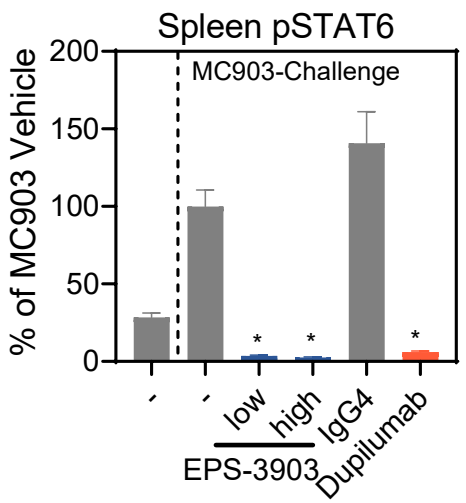
- MC903 model in transgenic mice expressing human IL-4/IL-4Rα triggers TSLP-mediated systemic inflammation with increased B-cell activity

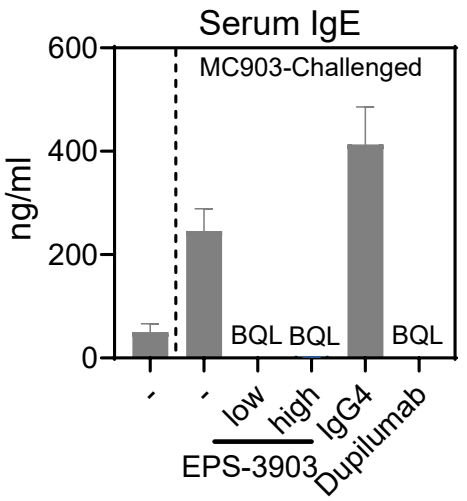



**EPS-3903 efficacy comparable to dupilumab**

- >90% inhibition of pSTAT6 in skin and spleen
- Robust decrease in serum IgE




*p<0.05 vs Corresponding Vehicle

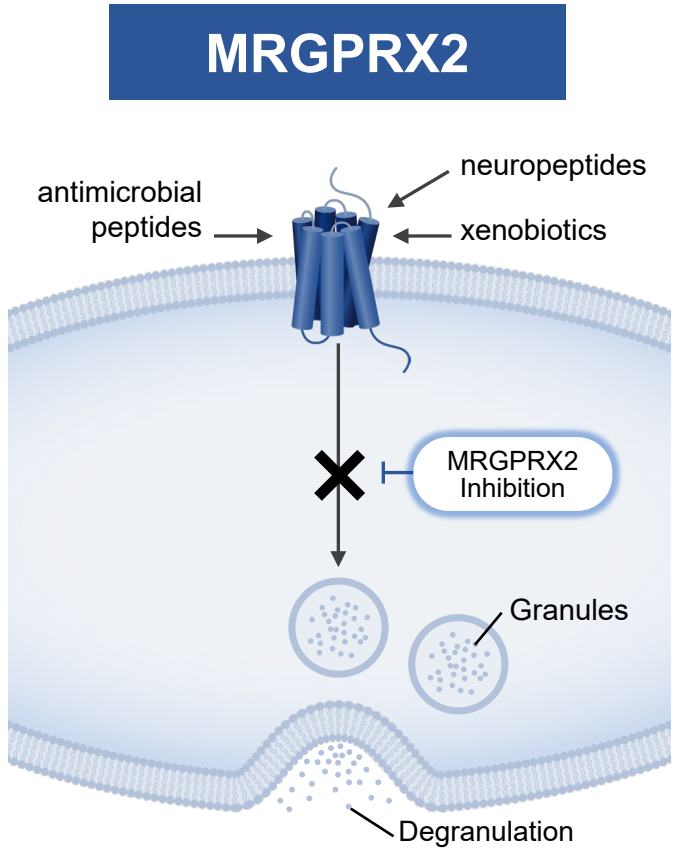
 

# Immunology: MRGPRX2 Inhibitor

Enanta Pharmaceuticals

# MRGPRX2 Inhibitors Offer Potential for Strong Efficacy and Best-in-Disease Safety Profile

- **Mast Cells:** primary driver of inflammation in skin, and implicated in multiple allergic diseases
  - Urticaria, asthma, prurigo nodularis (PN) and others
- **MRGPRX2:** non-canonical GPCR expressed predominantly on mast cells
  - Agonism leads to degranulation and release of inflammation mediating components
  - CSU patients have more MRGPRX2 expressing mast cells & a greater response to X2 agonist
- **MRGPRX2 Inhibitors:** potential for strong efficacy with best-in-disease safety profile
  - Non-depleting mast cell modulation
  - Includes IgE-independent pathways



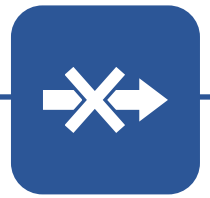

**MRGPRX2** Inhibition prevents activation and degranulation in mast cells

MRGPRX2: Mas-related G-protein Coupled Receptor Member X2

# Goal of Oral MRGPRX2 Inhibitor Program:
## Develop Best-in-Class Treatment for Mast Cell Mediated Diseases

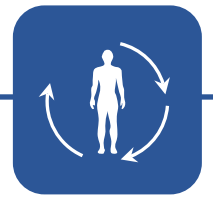
- Novel, oral inhibitors of MRGPRX2 being optimized in discovery stage



Inhibits MRGPRX2 with nanomolar potency in cellular assays

Prevents skin mast cell activation in mouse model

Highly selective for MRGPRX2 versus other GPCRs



Favorable *in vitro* and *in vivo* ADME properties supporting once daily dosing

- Prototypes being optimized with goal of selecting development candidate in 2H 2026

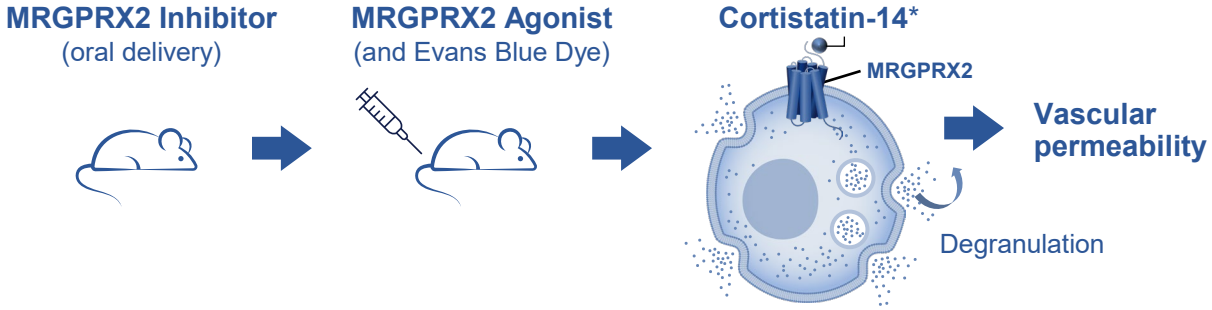
# Prototype MRGRPX2 Inhibitor Exhibits Potent Inhibition *In Vitro* and *In Vivo*

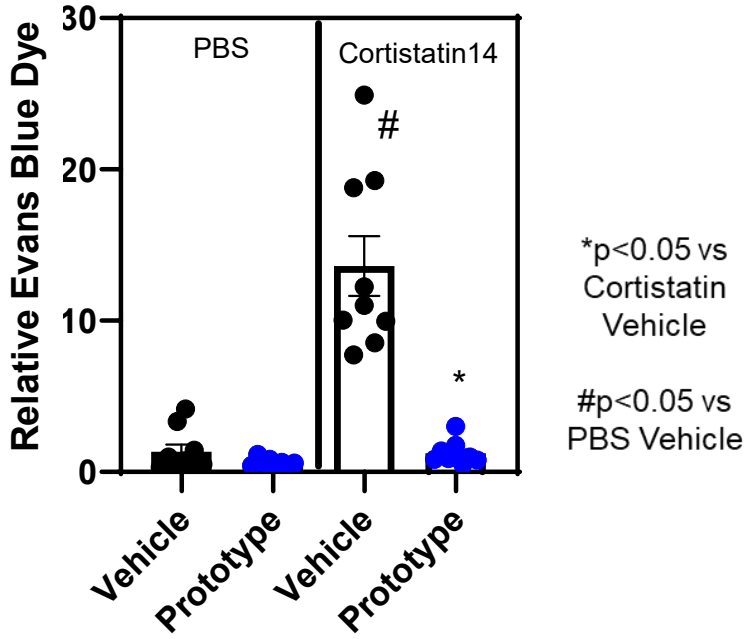
- Nanomolar inhibition in cellular assays with an $EC_{50}$ of **1 – 2nM**

| Cellular Assay | $EC_{50}$ (nM) |
|---|---|
| LAD2 Cells (β-hexosaminidase release) | 1.7 |
| LAD2 Cells (Histamine Release) | 2.1 |
| Primary Human Skin Mast Cells (β-hexosaminidase release) | 1.2 |

Cells stimulated with MRGPRX2 agonist Cortistatin-14
LAD2 Cells: human mast cell line capable of histamine release (degranulation)

- Prevents skin mast cell activation in humanized MRGPRX2 mice[1]



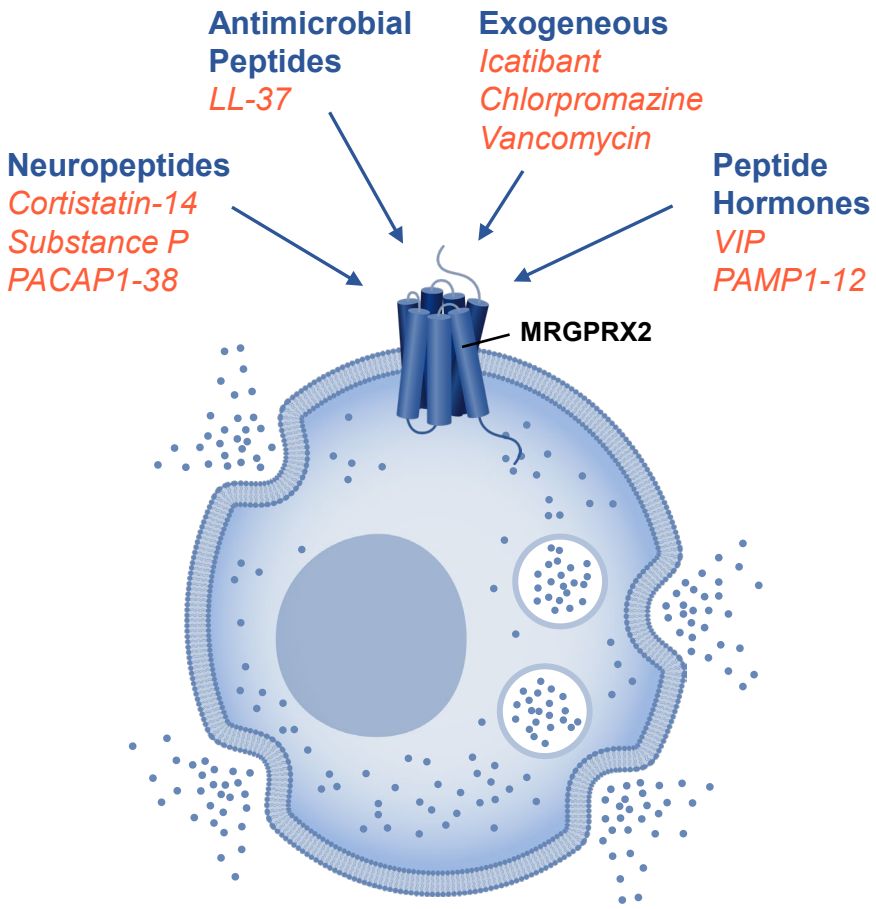

*p<0.05 vs Cortistatin Vehicle

#p<0.05 vs PBS Vehicle

1. Cortistatin-14 binding to MRGPRX2 triggers mast cell degranulation resulting in vascular permeability, allowing blue dye to leak into tissues

# Prototype MRGRPX2 Inhibitor Demonstrates Potent Activity Across Multiple Agonists




- Potent inhibition of degranulation in LAD2 cells across endogenous and exogenous MRGPRX2 ligands

| Agonist | β-hexosaminidase $EC_{50}$ (nM) |
|---|---|
| Cortistatin-14 | 1.7 |
| Vasoactive intestinal peptide | 0.6 |
| PACAP1-38 | 1.9 |
| Substance P | 1.0 |
| LL37 | 2.3 |
| Icatibant | 0.7 |
| Chlorpromazine | 0.7 |

# 2026 Key Catalysts

## Virology

### Respiratory Syncytial Virus

- ✓ FDA End-of-Phase 2 meeting on adult pivotal study design & registration path in 2Q 2026
- ☐ Initiate Phase 2b study of zelicapavir in pediatric patients in 3Q 2026
- ☐ Initiate Phase 2b/3 study of zelicapavir in high-risk adults in 4Q 2026
- ☐ Explore business development opportunities for RSV assets

## Immunology

### KIT Program

- ✓ File IND for EDP-978 in 1Q 2026
- ☐ Report Phase 1 data for EDP-978 in 4Q 2026

### STAT6 Program

- ☐ File IND for EPS-3903 in 2H 2026

### MRGPRX2 Program

- ☐ Nominate development candidate in 2H 2026

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