Enanta Pharmaceuticals

Great Chemistry Cures

Corporate Presentation

November 16, 2026

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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
- **$194.5 in cash** at September 30, 2026

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

MAVYRET® glecaprevir/pibrentasvir 100 mg/40 mg tablets

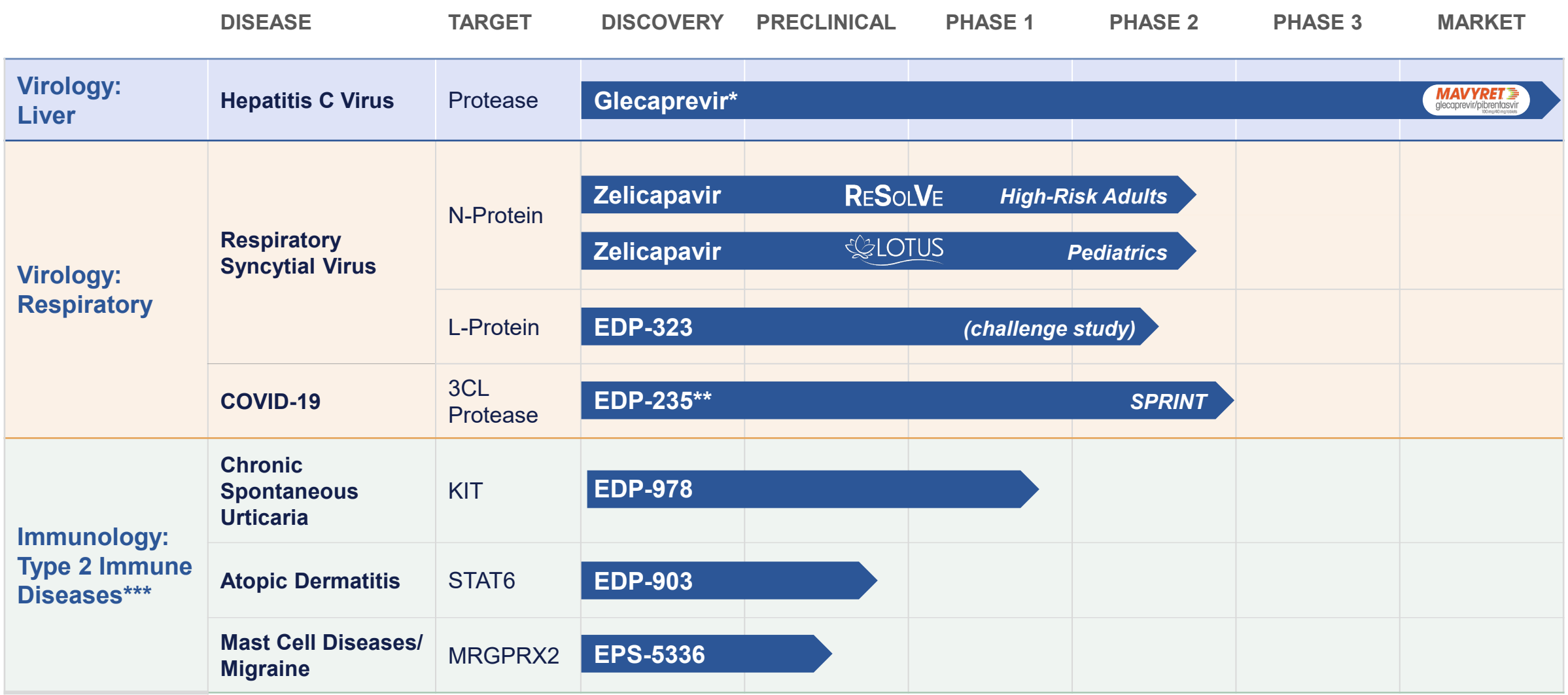
MAVIRET™ glecaprevir/pibrentasvir

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), COPD, Eosinophilic Esophagitis (EoE); Prurigo Nodularis (PN), Migraine and others.

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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	Hospitalizations	Deaths
up to 6.8M	up to 370K	up to 24K

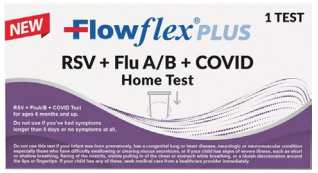
Populations at higher risk for severe illness

- 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+

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Multi-Billion-Dollar Opportunity for an RSV antiviral

2035 Global Forecast Across Pediatrics & High-Risk Adults

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U.S. ADDRESSABLE POPULATION

Up to 4M

patients with RSV[1]

Pediatrics Age <3 years	**~1.5M**
HR3 adults CHF, COPD, age ≥75 years	**~1.2M**
Additional high-risk adults CKD, IC, T2DM, oncology	**~1.3M**

GLOBAL PEAK SALES POTENTIAL[2]

Pediatrics and all high-risk adults

~$2.6–3.5B

Pediatrics + HR3 adults

~$1.8–2.4B

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; 8(3): 284-286; Hsiao A et al. *Pediatrics*. 2025; 156(2): e2024069510; ABRYSVO USPI Adults: Landi SN et al. *JAMA Netw Open*. 2024; 7(11): e2446010. Horn EK et al. *Influenza Other Respir Viruses*. 2025;19(12): e70181; McLaughlin JM et al. *Open Forum Infect Dis*. 2022; 9(7): ofac300. 2. Peak sales forecast (2035); Based on primary market research and Enanta internal modeling that accounts for diagnosis, treatment and prescription fill rates.

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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 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

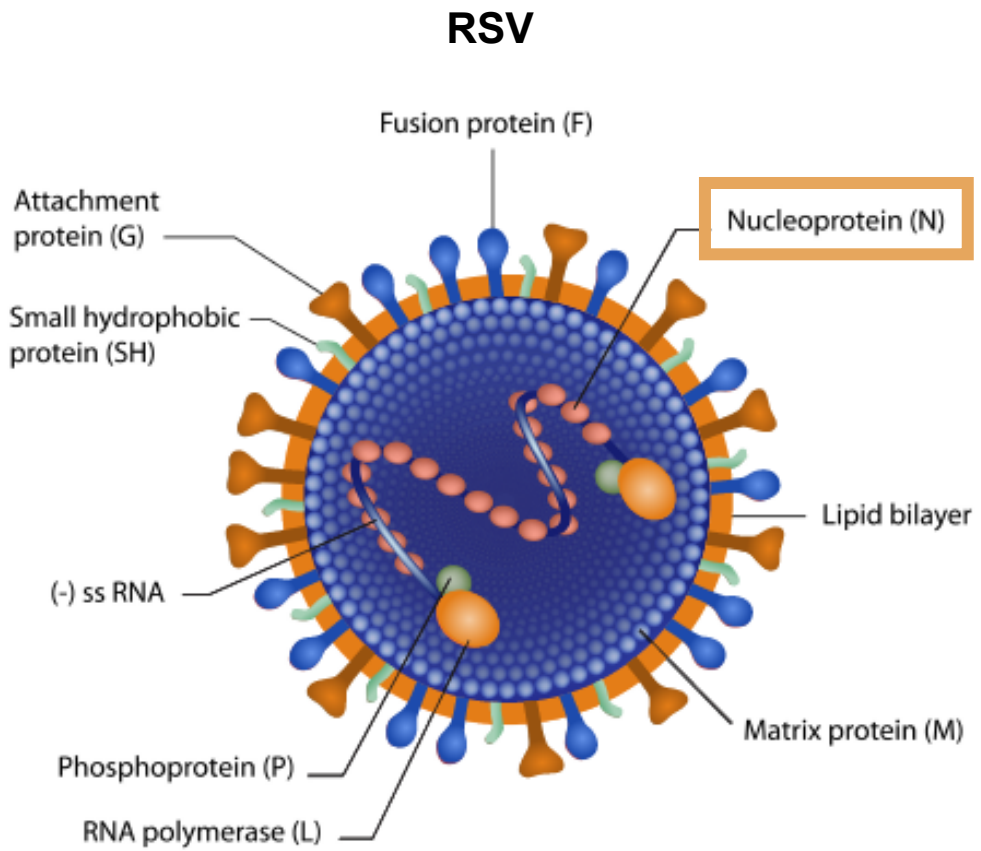
*A clinically meaningful improvement in time to complete resolution of all 13 RSV symptoms and a lower hospitalization rate was observed for zelicapavir compared to placebo. No effect was observed on the time to resolution of the lower respiratory tract disease subset of four symptoms to mild, which was the primary endpoint.

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

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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



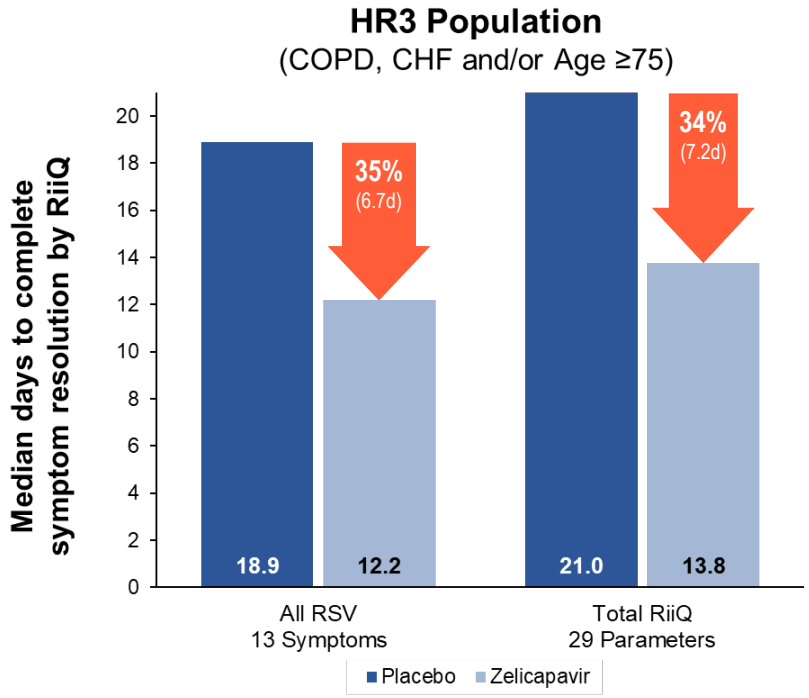
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Zelicapavir High-Risk Adult Program:
Phase 2b Study Summary

- Demonstrated compelling results on multiple clinically meaningful endpoints measuring different aspects of RSV disease*
  - ✓ Up to one week (35%) improvement in complete RiiQ™ symptom resolution
  - ✓ Statistically significant 2-day improvement in Patient Global Impression of Severity (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

*No effect was observed on the time to resolution of the lower respiratory tract disease subset of four symptoms to mild, which was the primary endpoint.

RiiQ: Respiratory Infection Intensity and Impact Questionnaire; COPD: Chronic Obstructive Pulmonary Disease; CHF: Congestive Heart Failure

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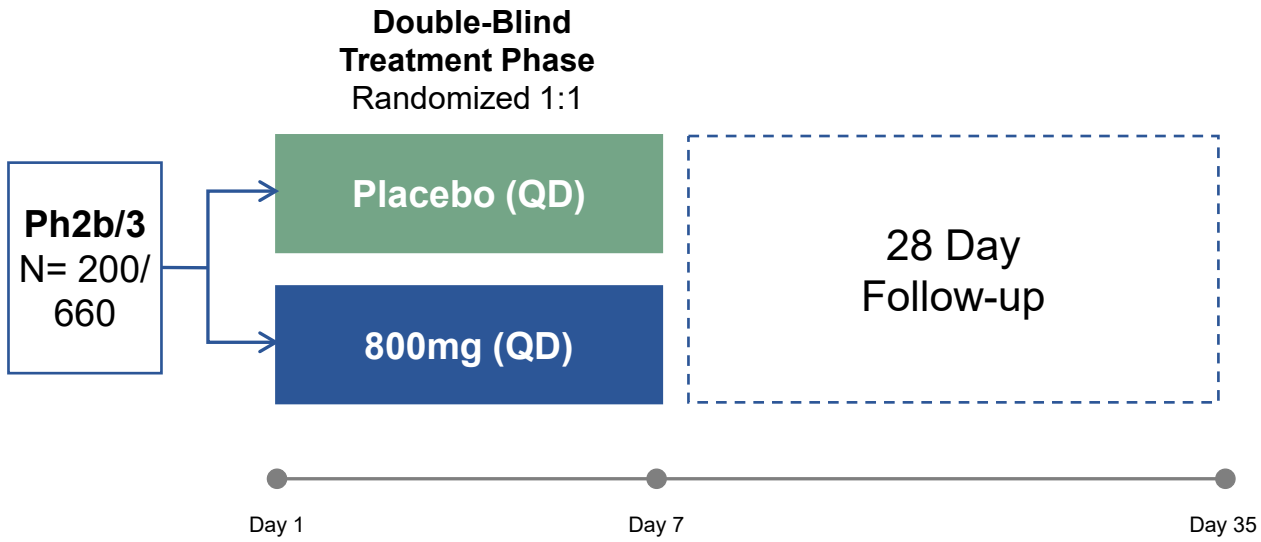
Zelicapavir High-Risk Adult (HR3) Program:
Phase 2b/3 Global Study Design

RESOLVE

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 initiated 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

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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 time to complete symptom resolution by 1.6 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 © 2026 Enanta Pharmaceuticals, Inc. All rights reserved.

Zelicapavir Pediatric Program:
Phase 2b Study Design

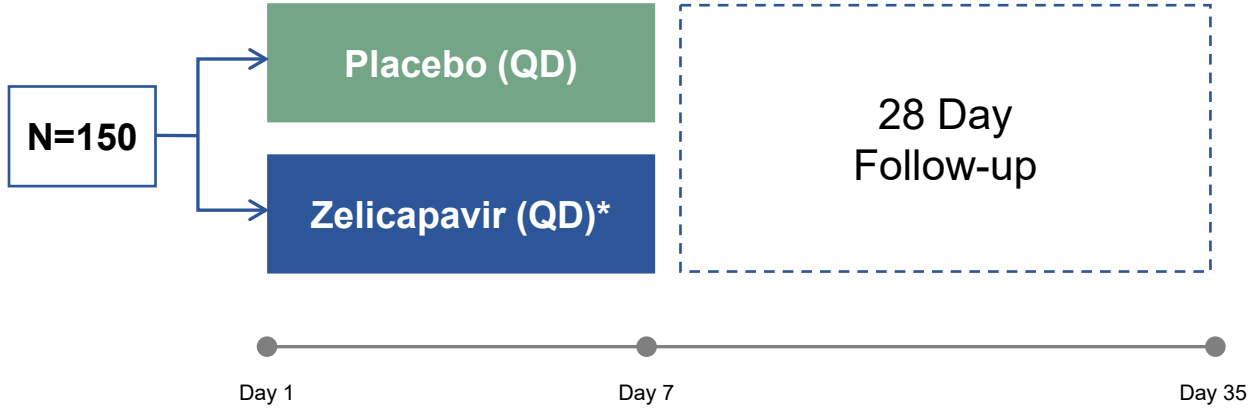
LOTUS

Trial Design

Double-Blind Treatment Phase
Randomized 1:1

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

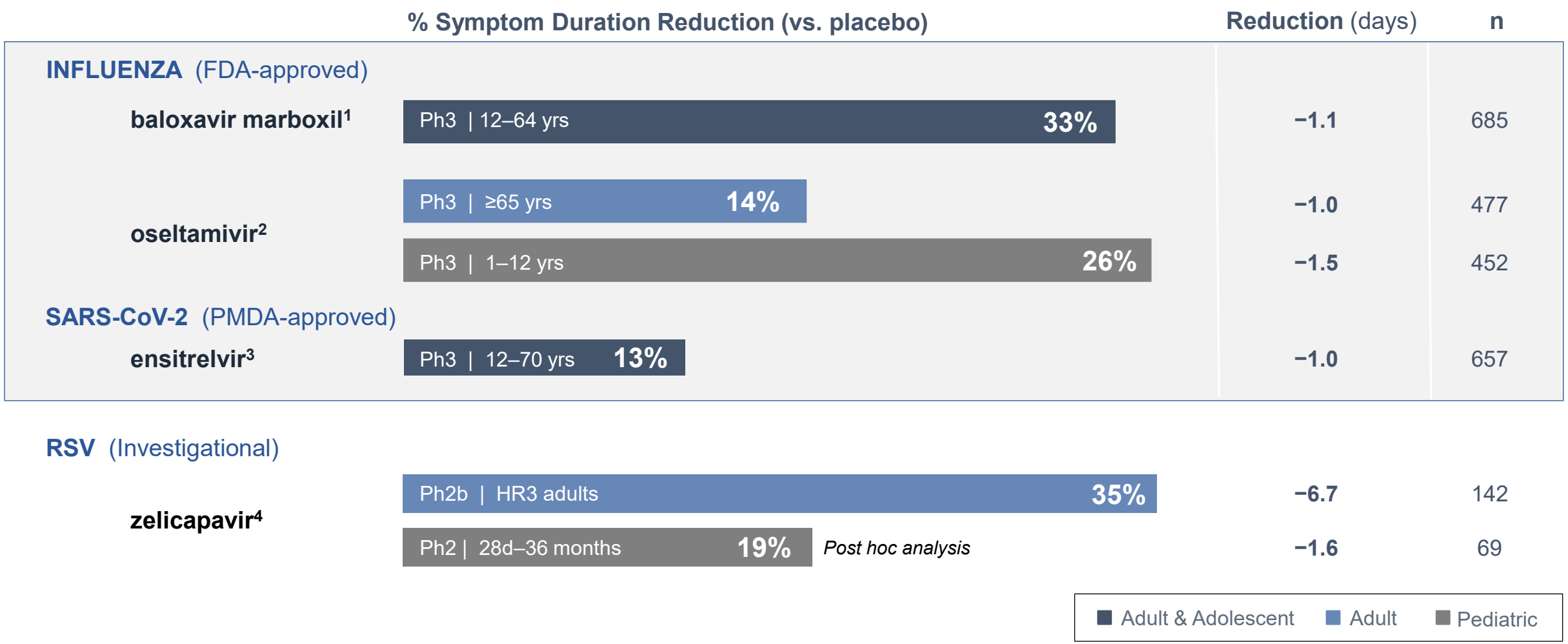
Safety, virology, additional clinical efficacy measures

Enrollment completed in 4Q 2026; Phase 2b topline data expected in 1Q 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 © 2026 Enanta Pharmaceuticals, Inc. All rights reserved.

Approved Respiratory Antivirals Reduce Symptom Duration by 13% to 33%



Cross-trial descriptive comparison. Approved antivirals limited to those with symptom reduction primary endpoint. Endpoints: influenza = symptom alleviation; SARS-CoV-2 = resolution of symptoms not present at baseline; RSV = complete symptom resolution. HR3 = Patients with Congestive Heart Failure, Chronic Obstructive Pulmonary Disease, or age ≥75 yrs

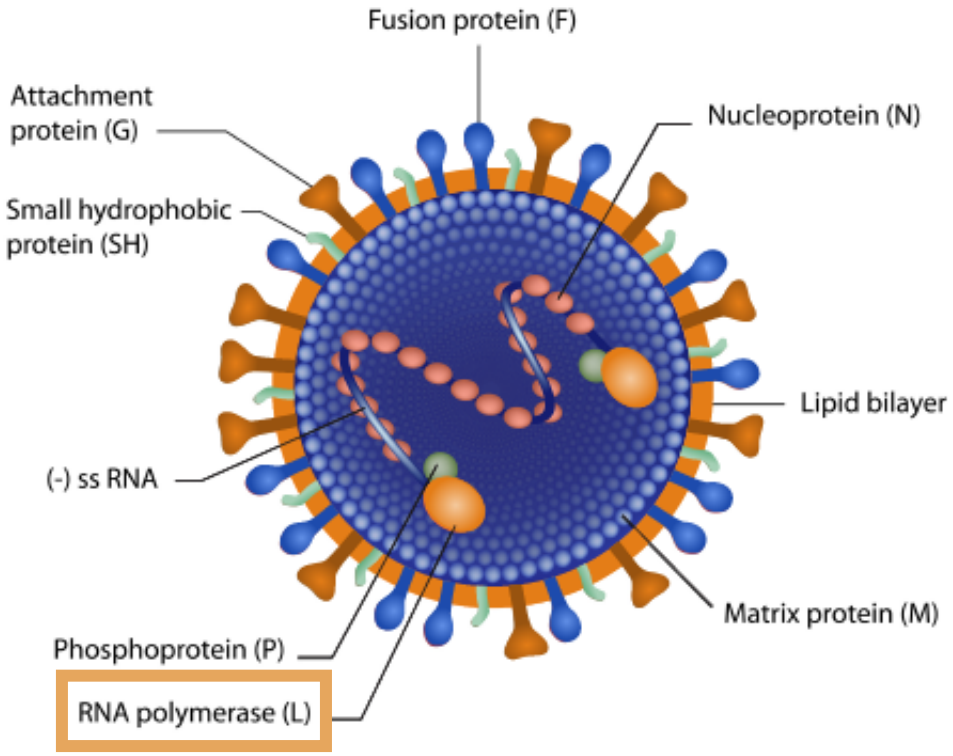
1. Hayden FG et al., NEJM 2018 2. Dobson J et al., Lancet 2015 & Whitley RJ et al., Pediatr Infect Dis J 2021 3. Kegg: ensitrelvir product information 4. IDWeek 2025 Pediatric Poster & IDWeek 2026 High-Risk Adult Poster

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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

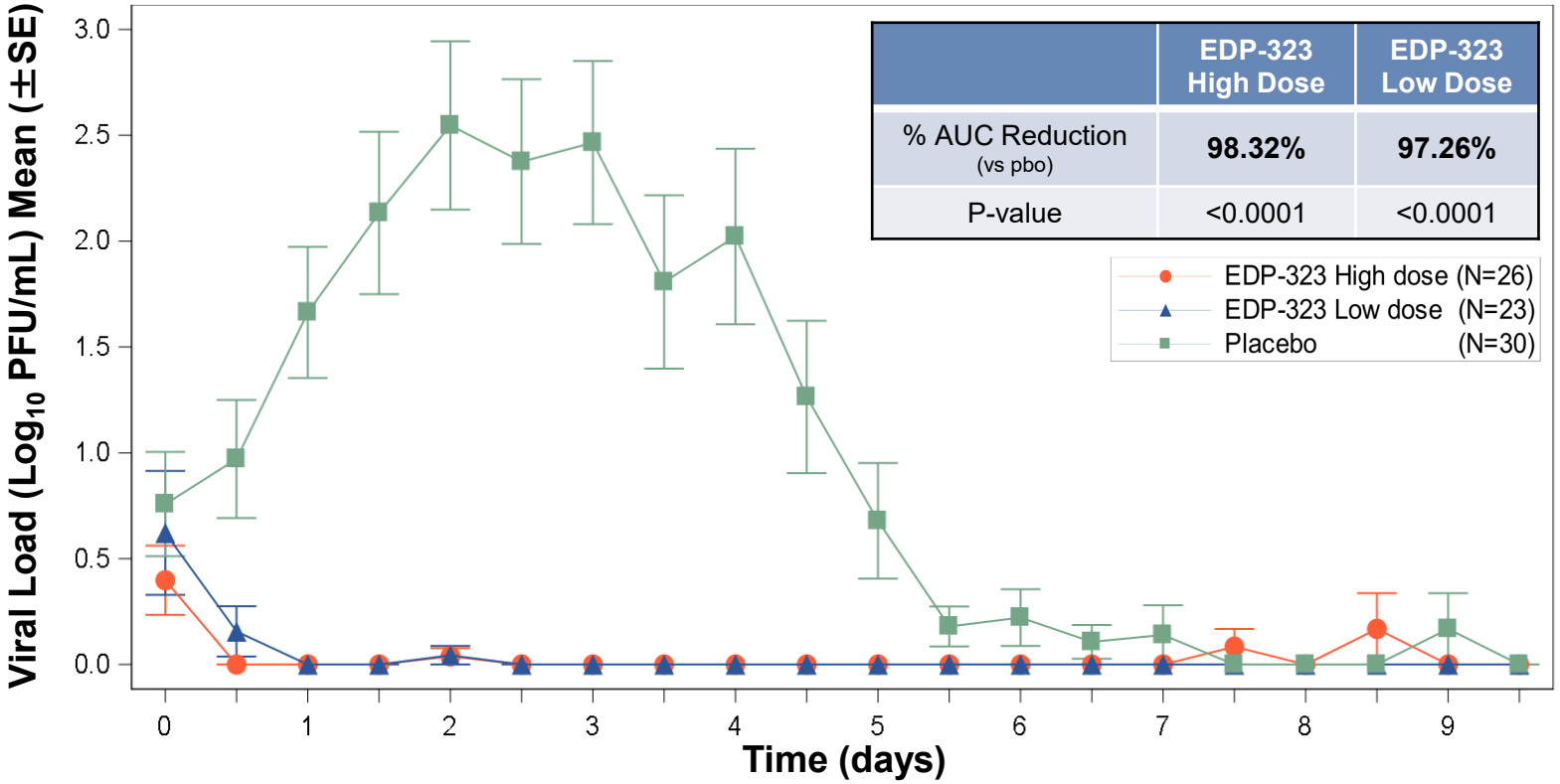
RSV



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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

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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

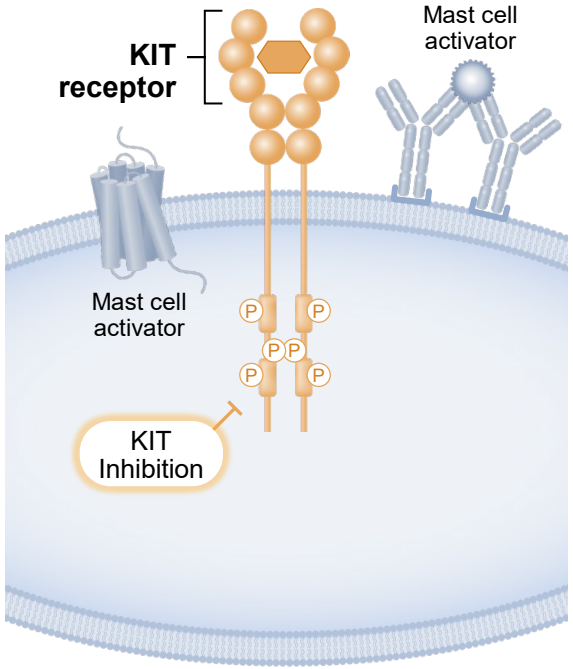
Strong Target Rationale	Clinical, genetic, or pathway validation to link target to disease
Clear Means to Differentiation	Yet to be drugged, drugged by injectables or suboptimal orals
Expedited Clinical Path to POC	Predictive biomarkers or early clinical signals derisk development
Significant Unmet Need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

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Immunology Portfolio:
Multiple Targets Addressing Type 2 Immune Diseases

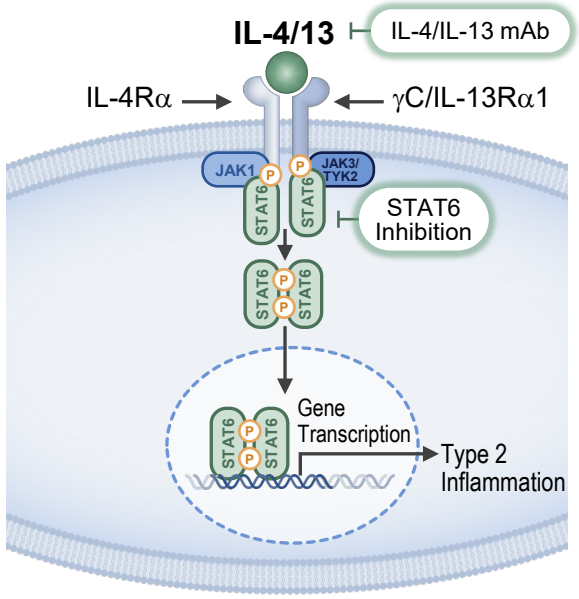
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KIT



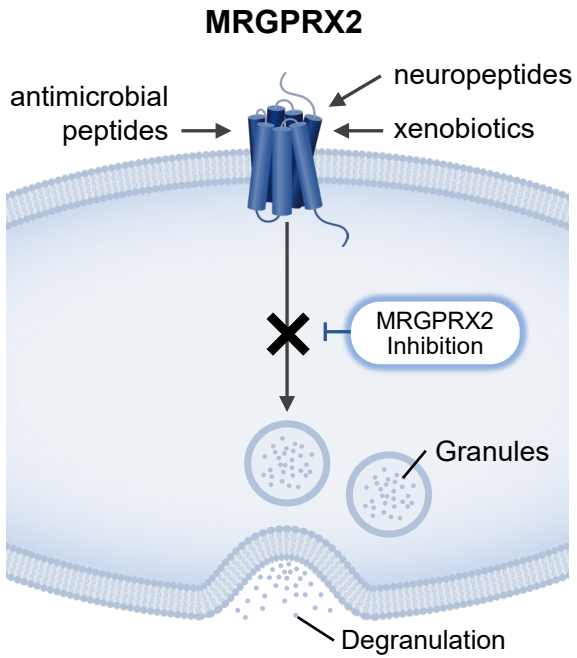
KIT Inhibition blocks cell survival signal resulting in apoptosis and cell death

STAT6



STAT6 Inhibition impedes type 2 inflammation

MRGPRX2



MRGPRX2 Inhibition prevents activation and degranulation in mast cells

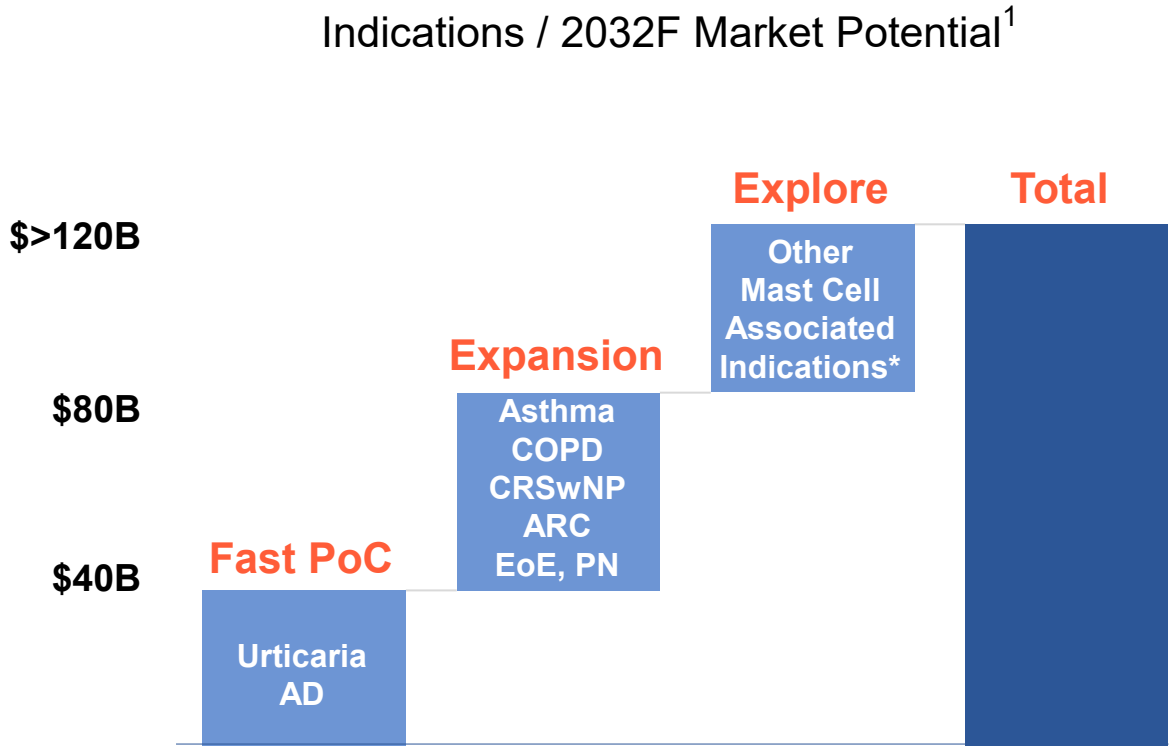
Potential Indications: Atopic Dermatitis, Asthma, CSU, CIndU, COPD, EoE, PN, CRSwNP, Migraine, Others

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

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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
1. Evaluate Pharma, © Evaluate Ltd, September 2026, www.evaluate.com

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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[1,3,4]

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



Angioedema



1. Kaplan A et al. *Allergy*. 2023. 2. Yosipovitch et al. *Dermatol Ther*. 2023. 3. Bernstein JA et al. *Dermatol Ther (Heidelb)*. 2025. 4. H1 antihistamines up to 4x standard dose.

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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

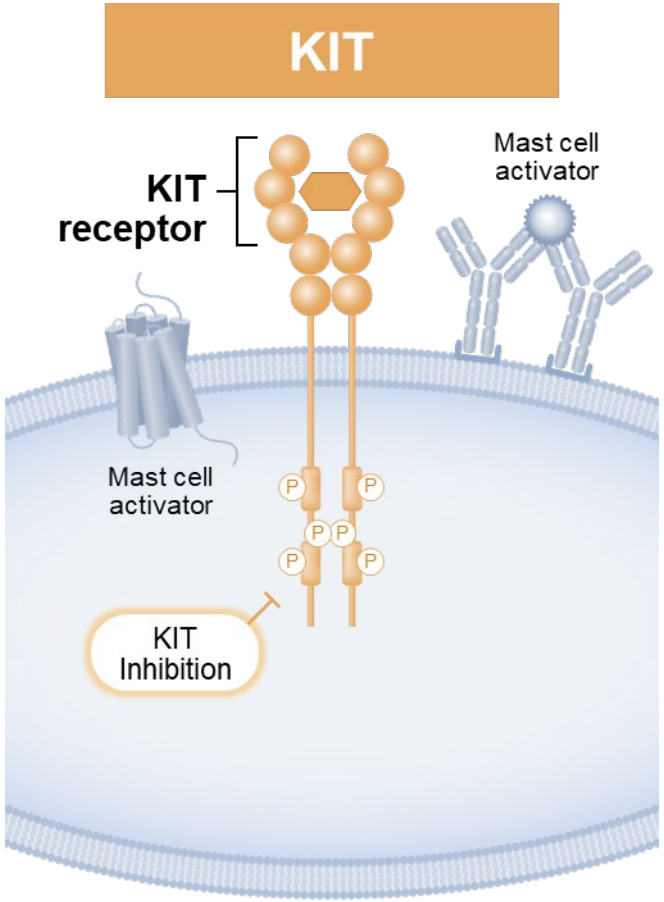
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Immunology: KIT Inhibitor

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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 (CSU, CIndU), asthma 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 with anti-KIT mAb



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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 1Q 2027

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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

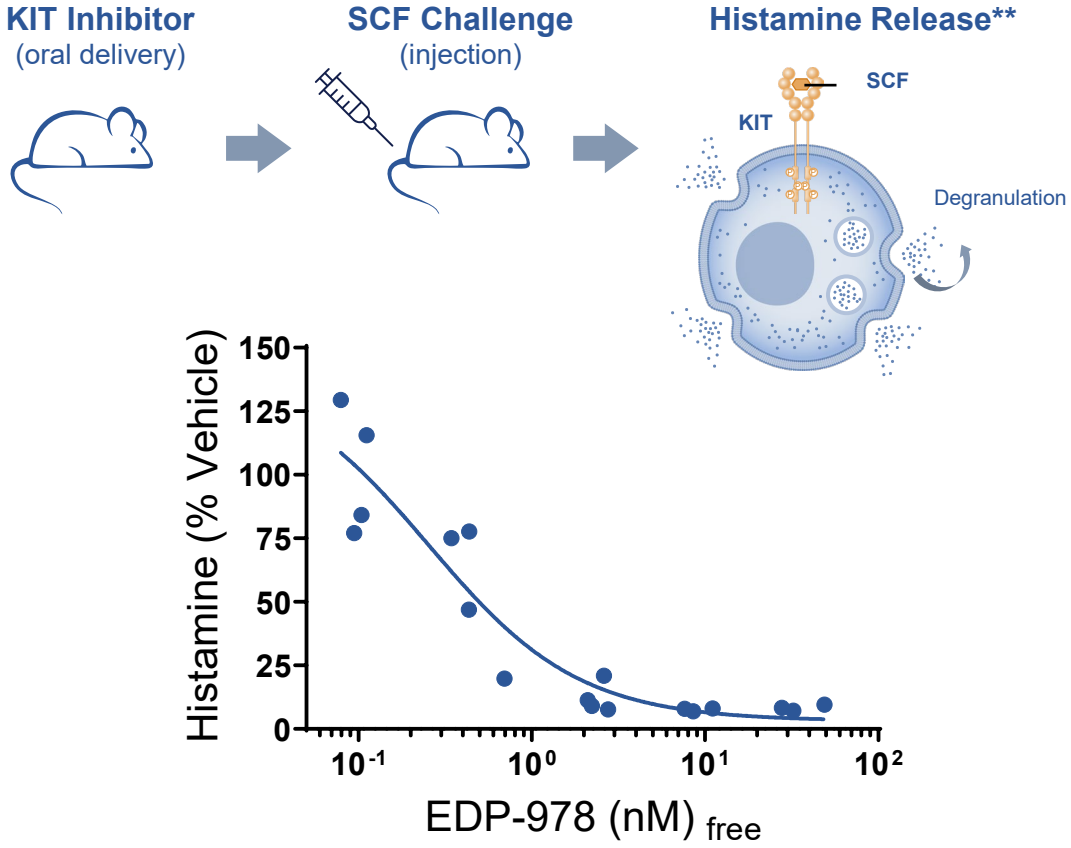
- Greater than 200-fold selectivity for KIT over other KIT family members

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

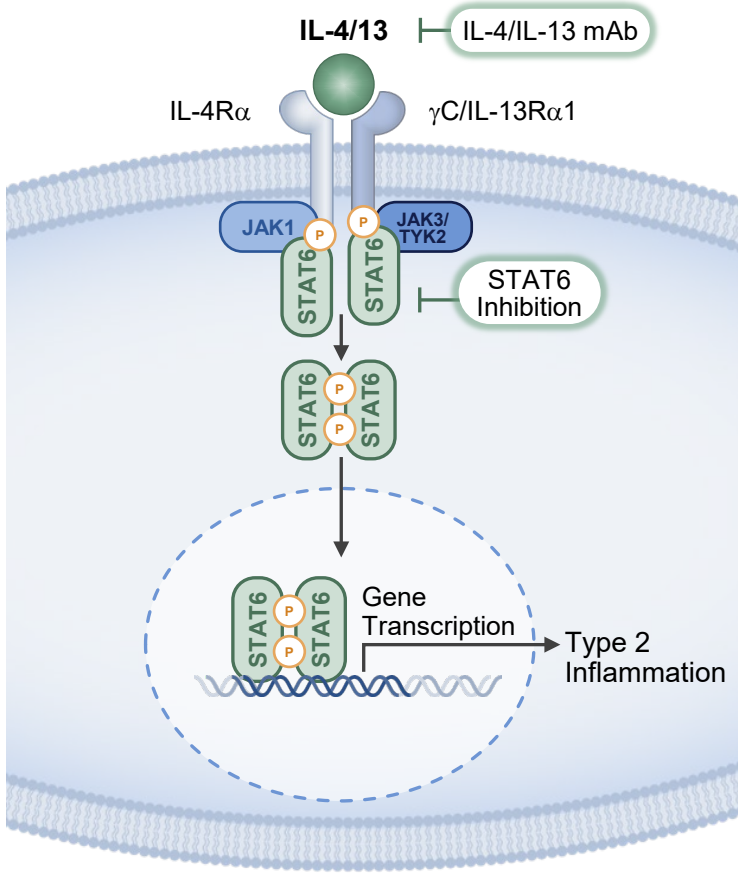
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Immunology: STAT6 Inhibitor

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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.

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

- EDP-903 selected as clinical 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)

- IND-enabling studies complete; Drug manufacturing ongoing with goal of IND filing in 2Q 2027

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EDP-903 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)	EDP-903	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

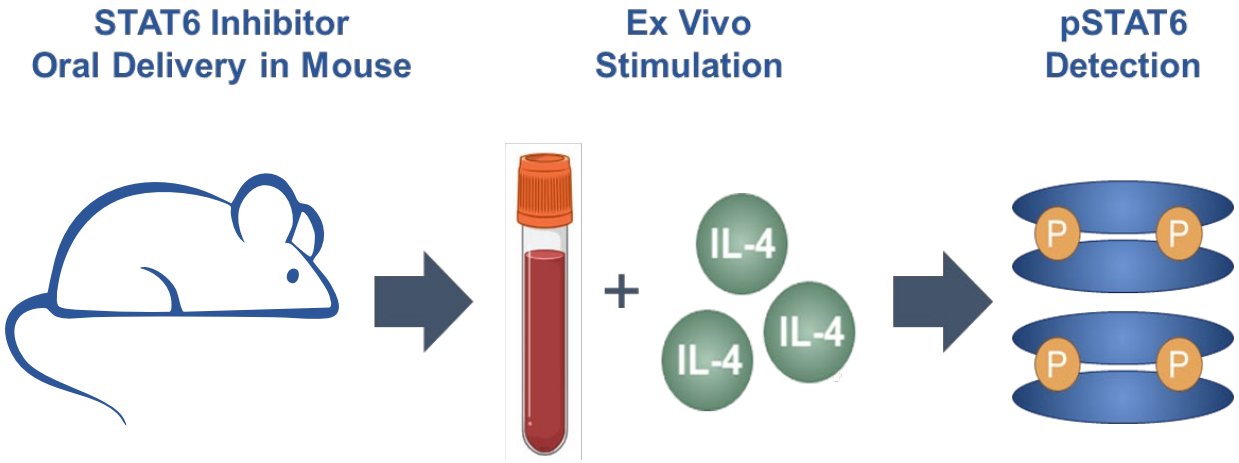
Selectivity in hPBMCs	EDP-903	Upadacitinib
	EC_{50} (nM)	
pSTAT1	>5000	1
pSTAT2	>5000	8
pSTAT3	>5000	24
pSTAT4	>5000	195
pSTAT5	>5000	16
pSTAT6	4	10

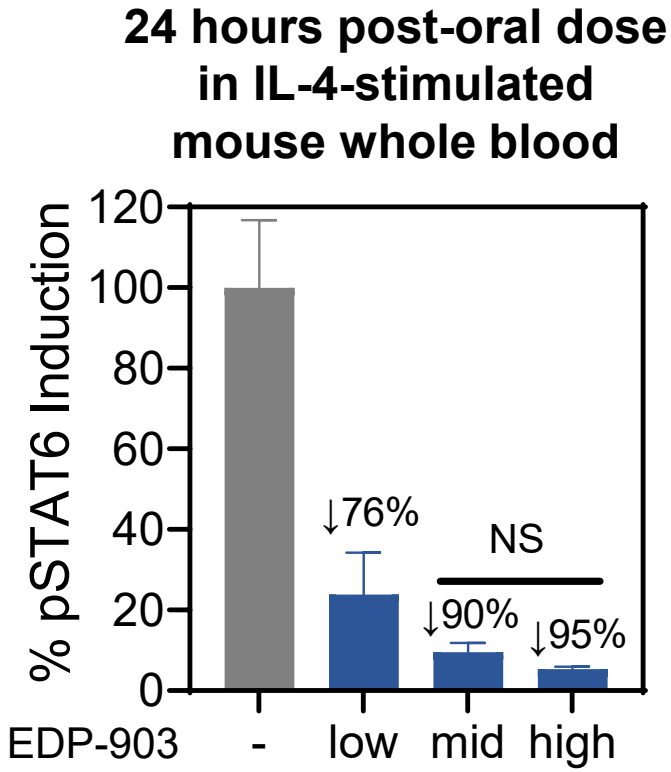
Upadacitinib: JAK Inhibitor ; hPBMC: Human Peripheral Blood Mononuclear Cells ; pSTAT6: Phosphorylated STAT6; TARC: Thymus and Activation-regulated Chemokine

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EDP-903 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





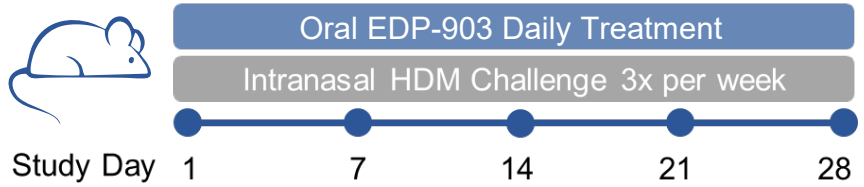
NS=not statistically different

pSTAT6: Phosphorylated STAT6

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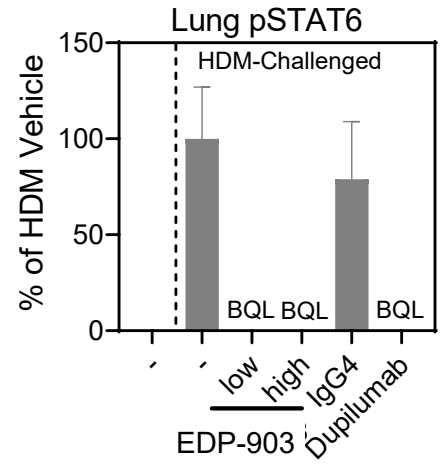
EDP-903 Shows Comparable Efficacy to Dupilumab in a House Dust Mite (HDM) Challenge Asthma Model

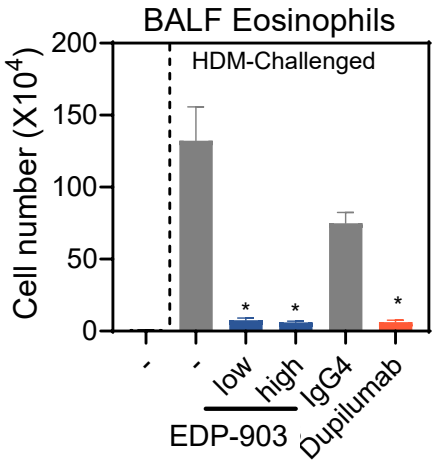
- HDM model in transgenic mice expressing human IL-4/IL-4Rα triggers allergic airway inflammation that mimics key aspects of asthma

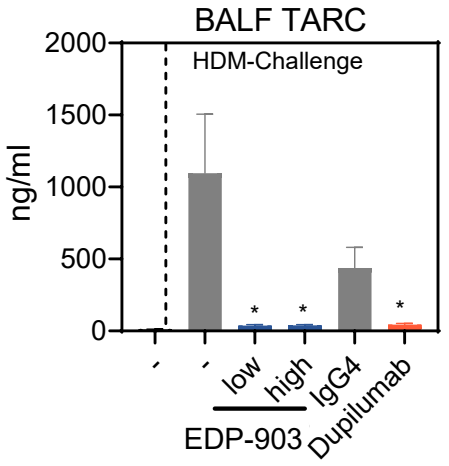


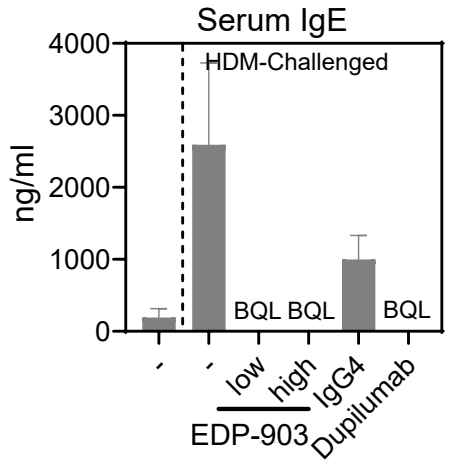
EDP-903 efficacy comparable to dupilumab

- >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









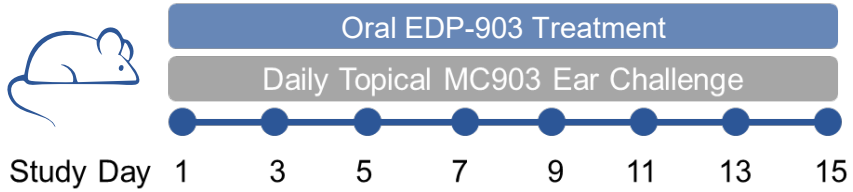
*p<0.05 vs Corresponding Vehicle

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

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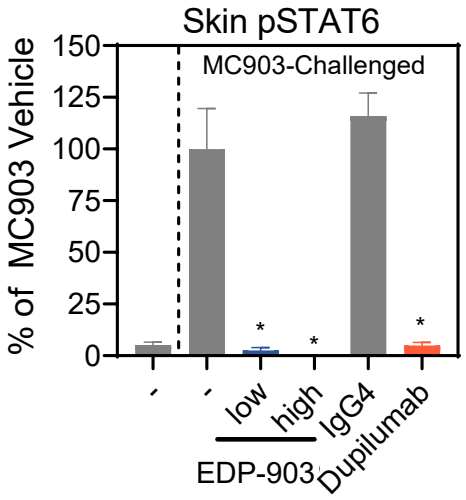
EDP-903 Demonstrates Comparable Efficacy to Dupilumab in a MC903-Driven Atopic Dermatitis Mouse Model

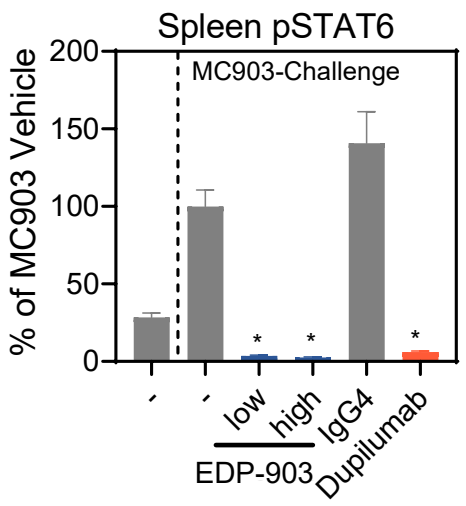
- MC903 model in transgenic mice expressing human IL-4/IL-4Rα triggers TSLP-mediated systemic inflammation with increased B-cell activity

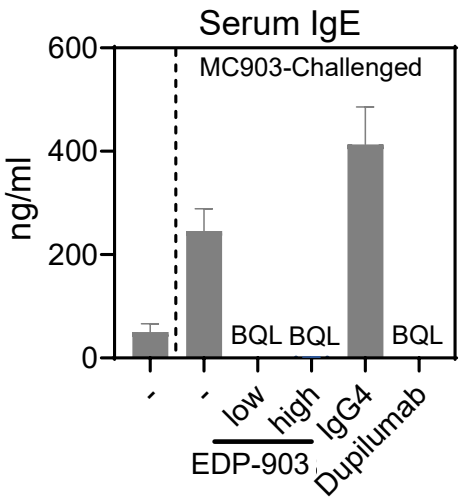


EDP-903 efficacy comparable to dupilumab

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







*p<0.05 vs Corresponding Vehicle

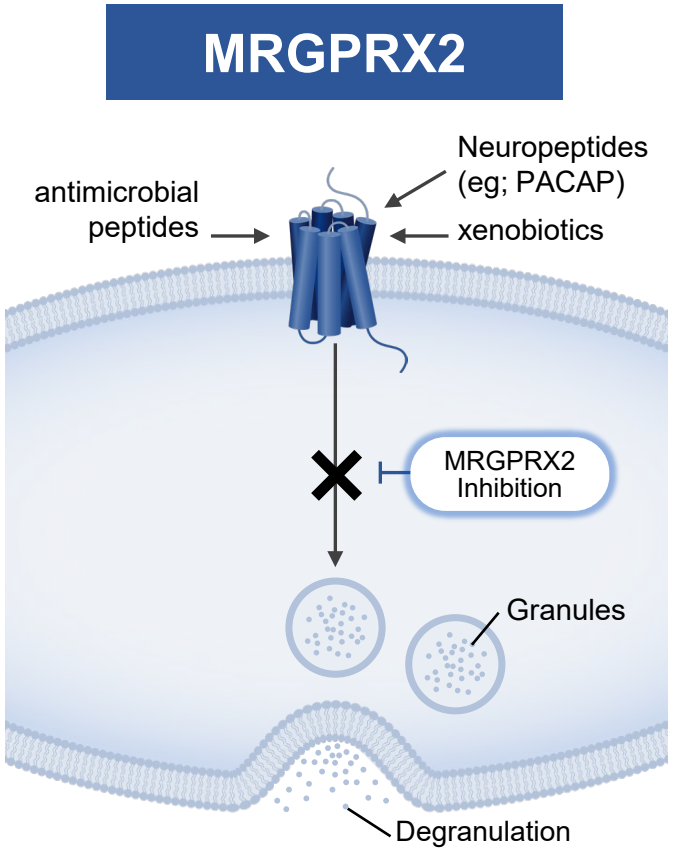
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Immunology: MRGPRX2 Inhibitor

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MRGPRX2 Inhibitors Offer Potential in Migraine and Other Mast Cell Associated Diseases

- **MRGPRX2**: non-canonical GPCR expressed on mast cells
- **Mast Cells:** involved in the pathology of multiple diseases
  - **Th2 Diseases**: urticaria, asthma, AD and others
    - MRGPRX2 agonism leads to degranulation and release of inflammation mediating components
  - **Migraine**: neurogenic inflammation driven by crosstalk between sensory neurons and mast cells
    - Blocking PACAP, but not its receptor (PAC1), has shown efficacy in a Ph2 study (mAb)
    - MRGPRX2 is candidate receptor for PACAP in migraine
- **MRGPRX2 Inhibitors:** potential for strong efficacy with a good safety profile
  - Th2 diseases: non-depleting mast cell modulation
  - Migraine: inhibits signaling from multiple neuropeptides involved in pathology



MRGPRX2 Inhibition prevents activation and degranulation in mast cells

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

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

- **EPS-5336 selected as development candidate**
  - Potent inhibitor of MRGPRX2 signaling
    - Subnanomolar potency in cellular assays
    - Noncompetitive inhibition
    - Prevents skin mast cell activation in mouse model
    - Inhibits MRGPRX2-induced degranulation in migraine-relevant meningeal mast cells
  - Inhibits degranulation induced by multiple ligands
  - Highly selective for MRGPRX2 versus other GPCRs
  - Favorable *in vitro* and *in vivo* ADME properties supporting once daily dosing

- Initiated IND-enabling activities with goal of IND filing in 4Q 2027

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EPS-5336 Potently Inhibits MRGPRX2 Signaling *In Vitro* and *In Vivo*

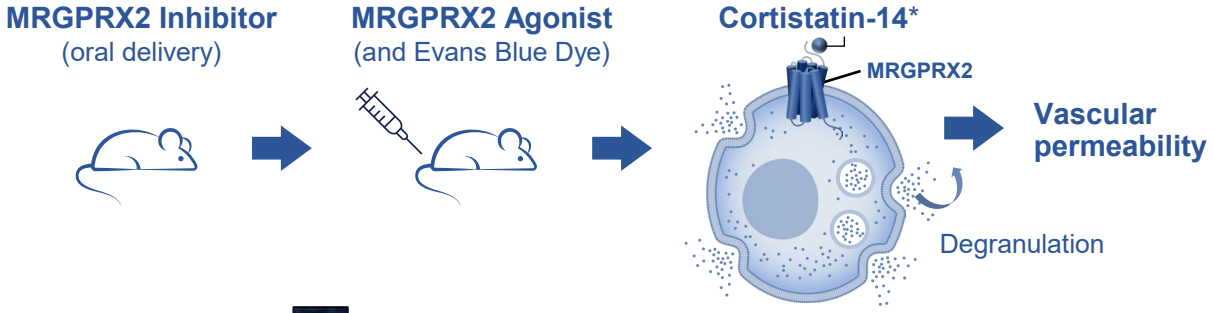
- Potent nanomolar inhibition in cellular assays with sub-nanomolar EC_{50}

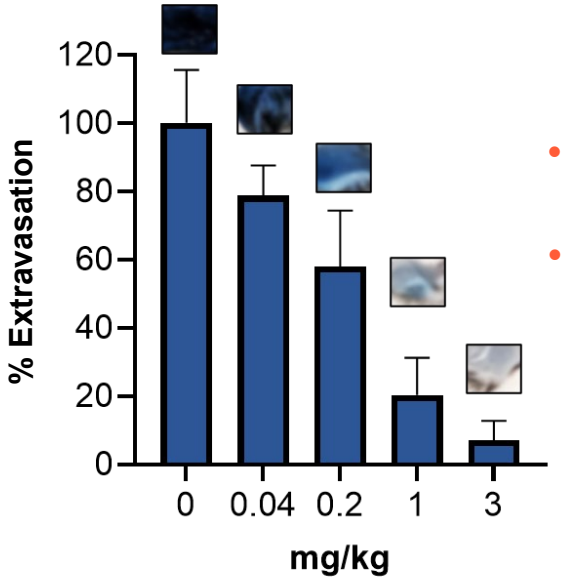
Cellular Assay	EC_{50} (nM)
LAD2 Cells (β-hexosaminidase release)	0.21
LAD2 Cells (Histamine Release)	0.27
Primary Human Skin Mast Cells (β-hexosaminidase release)	0.60

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

- Preferred **non-competitive** mechanism of inhibition
- Selective for MRGPRX2 over a broad GPCR panel with no inhibition ≤ 10 µM

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



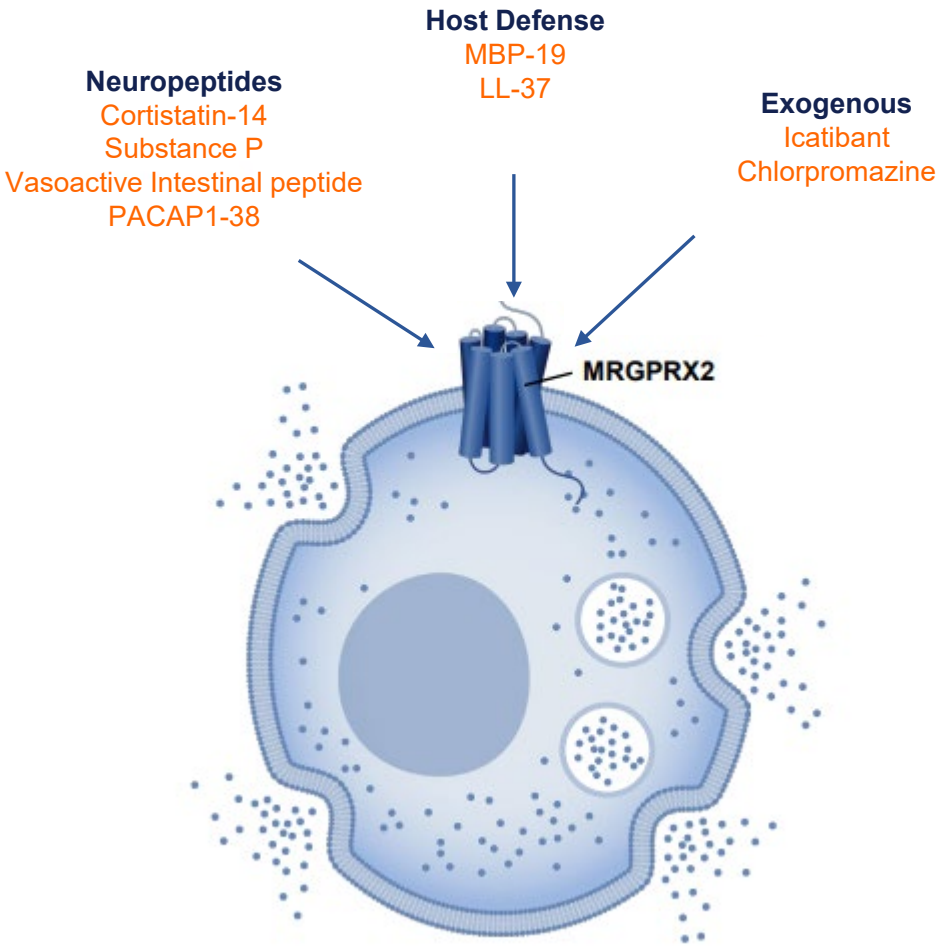


- In vivo free drug IC_{50}: 0.20 - 0.45 nM
- Robust *in vitro – in vivo* correlation

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

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EPS-5336 Inhibits Degranulation Induced by Multiple Endogenous and Exogenous MRGPRX2 Ligands in LAD2 Cells

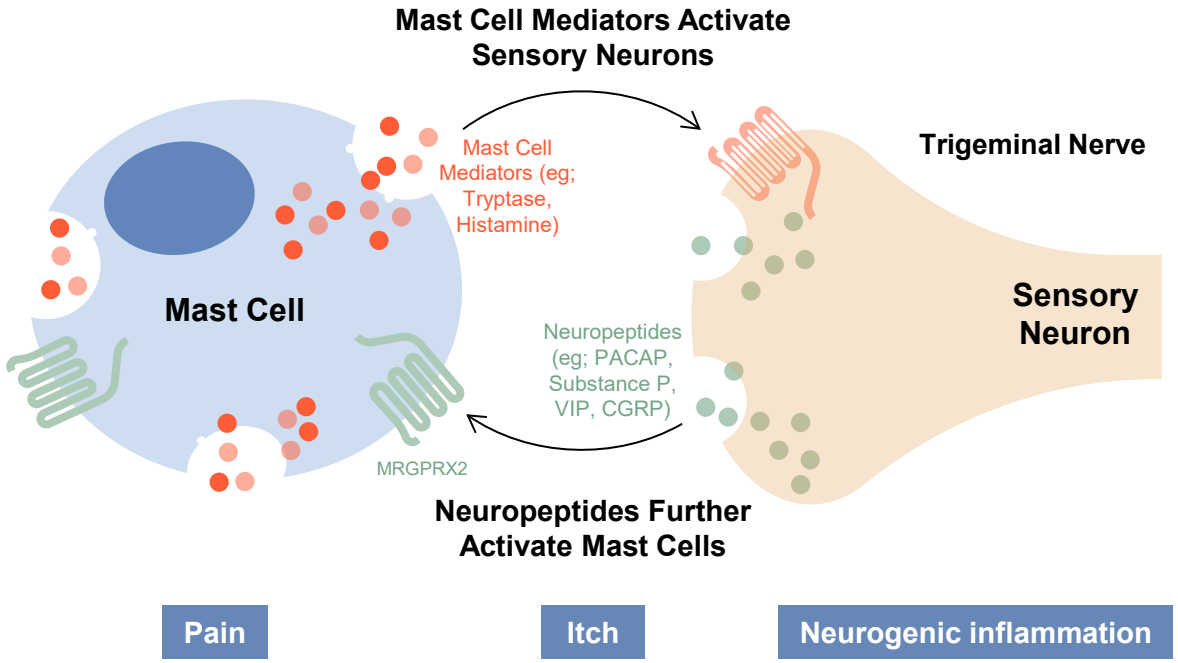


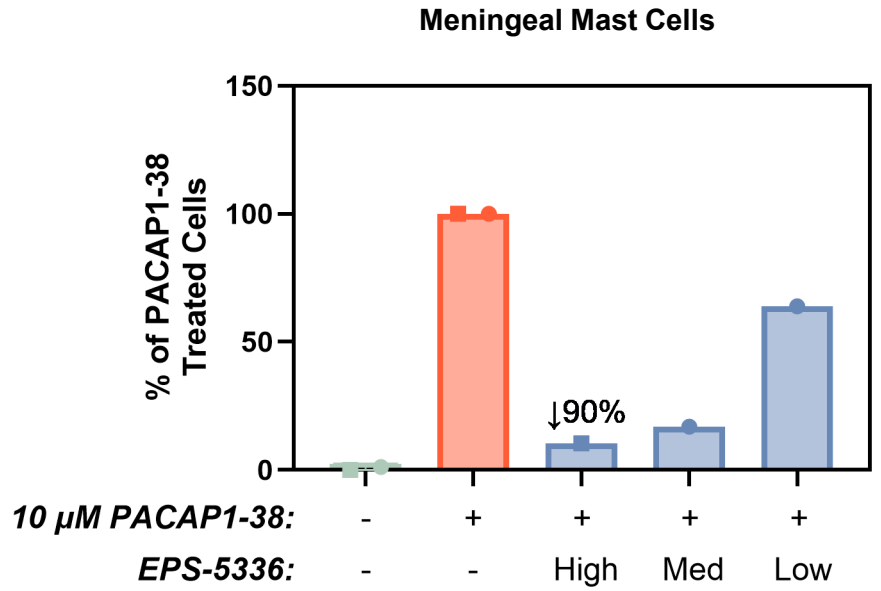
- Potent inhibition of degranulation in LAD2 cells across endogenous and exogenous MRGPRX2 ligands

Category	Ligand	EPS-5336 EC_{50} [nM]
Neuropeptides/Peptide Hormones	Cortistatin-14	**0.21**
	Substance P	**0.20**
	Vasoactive Intestinal Peptide	**0.08**
	PACAP1-38	**0.33**
Antimicrobial/Host Defense	MBP-19	**0.17**
	LL-37	**0.21**
Exogenous	Icatibant	**0.09**
	Chlorpromazine	**0.09**

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EPS-5336 Inhibits MRGPRX2-Induced Degranulation in Migraine-Relevant Meningeal Mast Cells





Data from 2 independent post mortem meningeal donors

- Migraine pain results from the activation of trigeminal neurons that innervate the meninges
  - Sensory neurons release neuropeptides (eg; PACAP, VIP, CGRP) which activate mast cells through MRGPRX2
- Cross talk between mast cells and trigeminal neurons through X2 propagate an inflammatory and pain cascade

- EPS-5336 exhibits potent inhibition of PACAP induced degranulation in human meningeal mast cells

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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 (*now 1Q 2027*)

STAT6 Program

- ☐ File IND for EDP-903 in 2H 2026 (*now 2Q 2027*)

MRGPRX2 Program

- ✓ Nominate development candidate in 2H 2026

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