



**Enanta**  
Pharmaceuticals  
**Great Chemistry Cures**

## **Corporate Presentation**

May 11, 2026



# Forward Looking Statements Disclaimer

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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



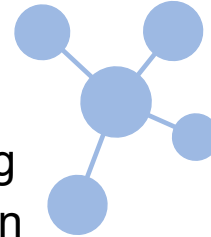
Clinical-stage biotechnology company dedicated to creating **small molecule drugs for virology & immunology indications**

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



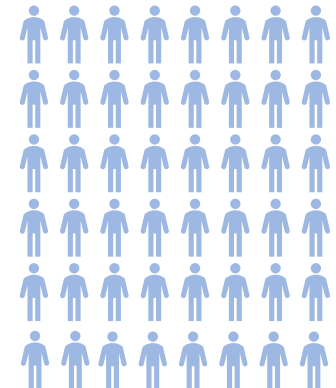
**viekira pak**<sup>®</sup>  
ombitasvir, paritaprevir and ritonavir tablets; dasabuvir tablets

**MAVYRET**<sup>™</sup>  
glecaprevir/pibrentasvir  
100 mg/40 mg tablets


**MAVIRET**<sup>™</sup>  
glecaprevir/pibrentasvir

**2** products approved with **abbvie**

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



# Enanta Pipeline

	DISEASE	TARGET	DISCOVERY	PRECLINICAL	PHASE 1	PHASE 2	PHASE 3	MARKET
<b>Virology: Liver</b>	<b>Hepatitis C Virus</b>	Protease	<b>Glecaprevir*</b>					
<b>Virology: Respiratory</b>	<b>Respiratory Syncytial Virus</b>	N-Protein	<b>Zelicapavir</b>			<i>High-Risk Adults</i>		
			<b>Zelicapavir</b>			<i>Pediatrics</i>		
		L-Protein	<b>EDP-323</b>			<i>(challenge study)</i>		
	<b>COVID-19</b>	3CL Protease	<b>EDP-235**</b>			<i>SPRINT</i>		
<b>Immunology: Type 2 Immune Diseases***</b>	<b>Chronic Spontaneous Urticaria (CSU)</b>	KIT	<b>EDP-978</b>					
	<b>Atopic Dermatitis (AD)</b>	STAT6	<b>EPS-3903</b>					
	<b>CSU/AD</b>	MRGPRX2	<b></b>					

\*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

# 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<sup>1</sup> (2024 - 2025 U.S.)



### 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<sup>2</sup>) to <50% (flu<sup>3</sup>)
- 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



1. CDC Preliminary Estimates of RSV Burden for 2024-2025; for period: 10/1/24-9/27/25 2. Terlizzi EP et al. *NCHS Data Brief*. 2020; Age 60+ 3. CDC Influenza Vaccination Coverage, Adults 65+

# RSV Development Goal:

## Treatment for Patients at High-Risk for Severe RSV Infection

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  $\geq 65$  years

Chronic heart or lung disease  
(e.g. COPD, CHF, asthma)

✓ Positive Phase 2b Results

### Zelicapavir Pediatric Phase 2 Study

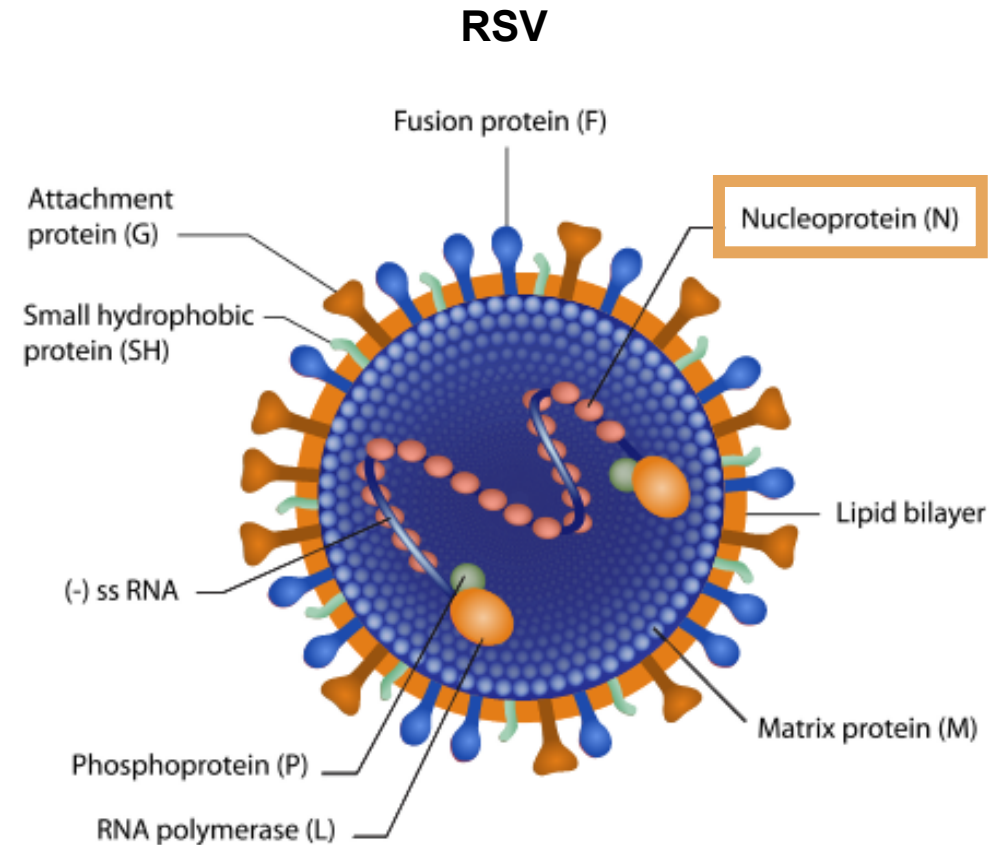
Infants and young children

✓ 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

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

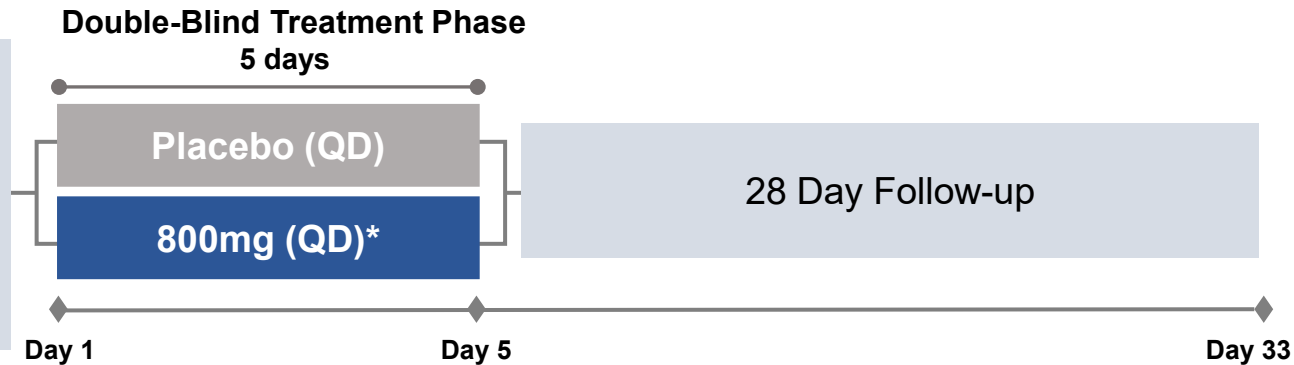
- Only N-inhibitor in clinical development for RSV
  - Replication inhibitor: shuts down the production of new virions (vs. fusion inhibitors which block viral entry)
- Granted Fast Track designation by the FDA
- Strong preclinical profile
  - Nanomolar potency against RSV-A and RSV-B
  - Antiviral potency 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
  - Well-tolerated in more than 700 people dosed



# Zelicapavir Phase 2b High-Risk Adult Study: Design & Objectives

## 186 adults with at least one of the following:

- COPD
- Congestive heart failure
- Asthma
- Age  $\geq 65$



- Patients enrolled within 72 hours of symptom onset
- Proportion of patients aged 65-74 years or those with asthma capped at 20% of the total population
- ▶ **HR3 = ~80% of the population with CHF, COPD, or age  $\geq 75$**

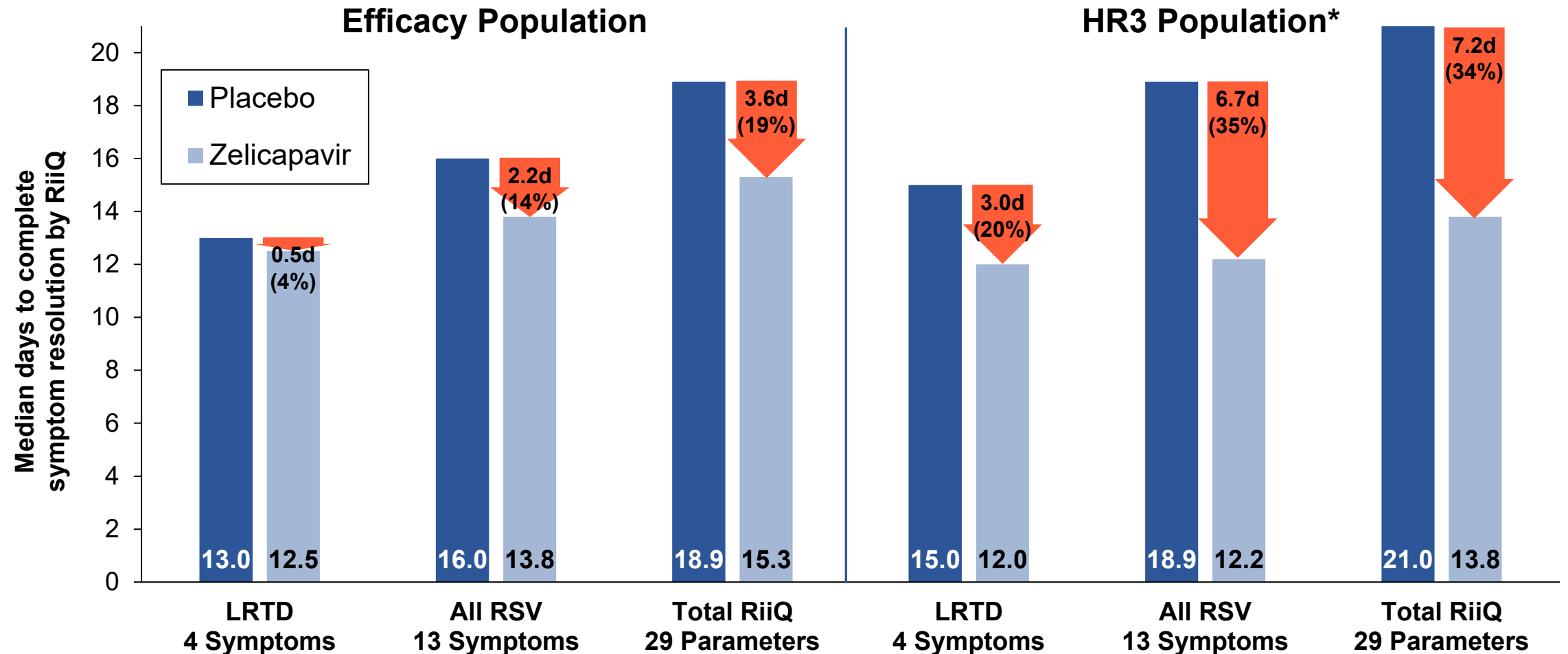
- **Primary Objective:**  
Time to resolution\*\* of RSV LRTD symptoms (RiiQ™ symptom scale)
  - Shortness of breath, wheezing, coughing, coughing up phlegm
- **Secondary Objectives:**  
PROs, MAVs, virology, antibiotic use, bronchodilator use, corticosteroid use, hospitalization, ICU, mechanical ventilation, all cause mortality, PK & safety

First proof-of-concept Phase 2 high-risk adult outpatient study with positive clinical signal

\*Equivalent to 600mg suspension dosage form used in challenge study; \*\*Resolution: all symptoms mild or absent

COPD: chronic obstructive pulmonary disease; LRTD: lower respiratory tract disease; PROs: patient reported outcomes; MAVs: medically attended visits; ICU: intensive care unit; PK: pharmacokinetics; QD: once-daily

# Zelicapavir Phase 2b High-Risk Adult Study: Faster Time to Complete Symptom Resolution Across Multiple RiiQ™ Measures



\*HR3 Population: Patients with CHF, COPD, or age  $\geq 75$ ; LRTD: lower respiratory tract disease

# Zelicapavir Phase 2b High-Risk Adult Study: Symptom Summary for Zelicapavir Compared to Placebo

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## Symptoms Measured by RiiQ™

- Faster time to **complete** resolution of all RSV symptoms to **absent**
  - About a week reduction in the HR3 population (for all 13 RSV symptoms and total RiiQ)
- No effect on time to resolution of RSV symptoms to **mild**, including primary endpoint (time to resolution of LRTD subset of symptoms to mild in the efficacy population)
- Improvement in RSV 13-symptom score\* in HR3 population
  - Day 9 (p=0.0403) and Day 14 (p=0.0247)

## Symptom Resolution Measured by PGI-S

- 2-day faster median time to improvement in efficacy (p=0.0446) and HR3 population (p=0.0465)

\*post-hoc analysis

RiiQ: Respiratory Infection Intensity and Impact Questionnaire; PGI-S: Patient Global Impression of Severity; HR3 Population: Patients with CHF, COPD, or age  $\geq 75$ ; LRTD: lower respiratory tract disease

# Zelicapavir Phase 2b High-Risk Adult Study: Hospitalization and Death Endpoints

- Lower hospitalization rate for patients treated with zelicapavir

	Placebo	Zelicapavir
All-cause-hospitalizations	5.0% (3/60)	1.7% (2/115)
RSV-associated hospitalizations*		
• Investigator attribution	5.0% (3/60)	0% (0/115)
• Post-hoc attribution	5.0% (3/60)	0.9% (1/115)

- One death on placebo; no deaths on zelicapavir

\*72yo man on zelicapavir was hospitalized on study day 21 for influenza infection after RSV symptoms had completely resolved on day 11; Investigator and Enanta deemed hospitalization not related to RSV

\*74yo woman on zelicapavir was hospitalized on study day 7 for infective exacerbation of asthma; Investigator deemed hospitalization not related to RSV; Enanta deemed hospitalization related to RSV

# Zelicapavir Phase 2b High-Risk Adult Study: Virology Results for Zelicapavir Compared to Placebo

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- Larger viral load decline at the end of treatment (Day 5)
  - Efficacy Population: 0.6 log
  - HR3 population: 0.7 log
- Greater proportion of patients with undetectable viral load at the end of treatment
  - Efficacy Population: 23.5% (27/115) vs 10.0% (6/60) (p=0.0198)
  - HR3 Population: 23.9% (22/92) vs 10.0% (5/50) (p=0.0292)
- Faster median time to undetectable viral load
  - Efficacy Population: 4 days
  - HR3 Population: 5 days

# Zelicapavir Phase 2b High-Risk Adult Study:

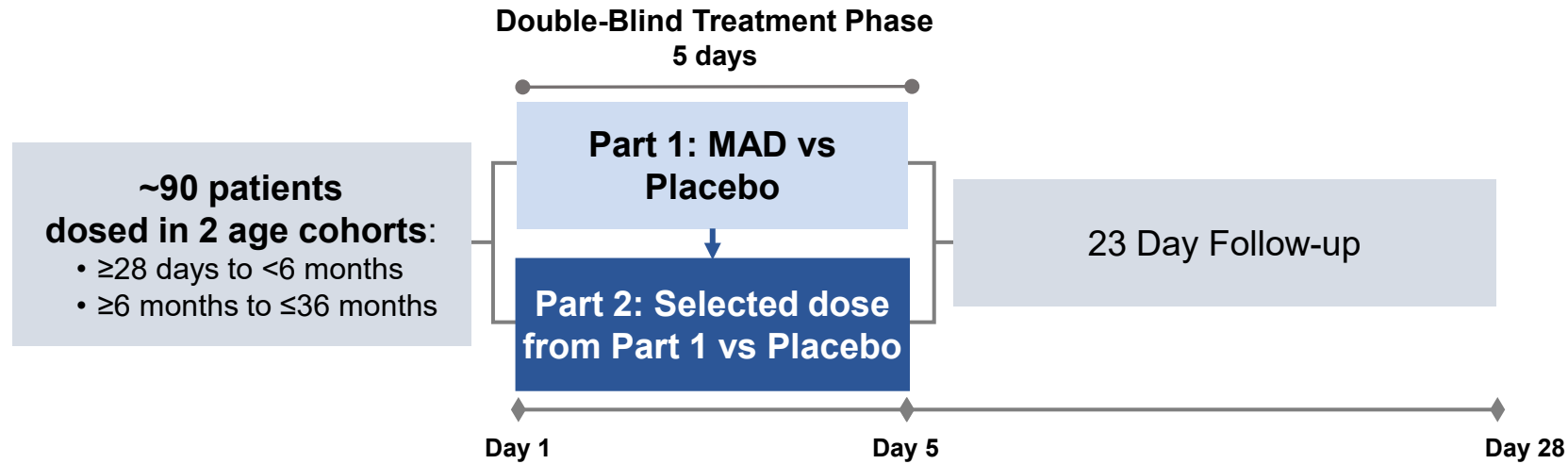
## Conclusions

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- Zelicapavir demonstrated compelling results on multiple clinically meaningful and potential registrational endpoints measuring different aspects of RSV disease
  - ✓ Up to one week improvement in complete RiiQ symptom resolution
  - ✓ Statistically significant improvement in PGI-S
  - ✓ Lower hospitalization rate
- Robust antiviral effect
- Well-tolerated, with a favorable safety profile

Data support advancement of zelicapavir into Phase 3 study of high-risk adults

# Zelicapavir Phase 2 Pediatric Study: Design & Objectives



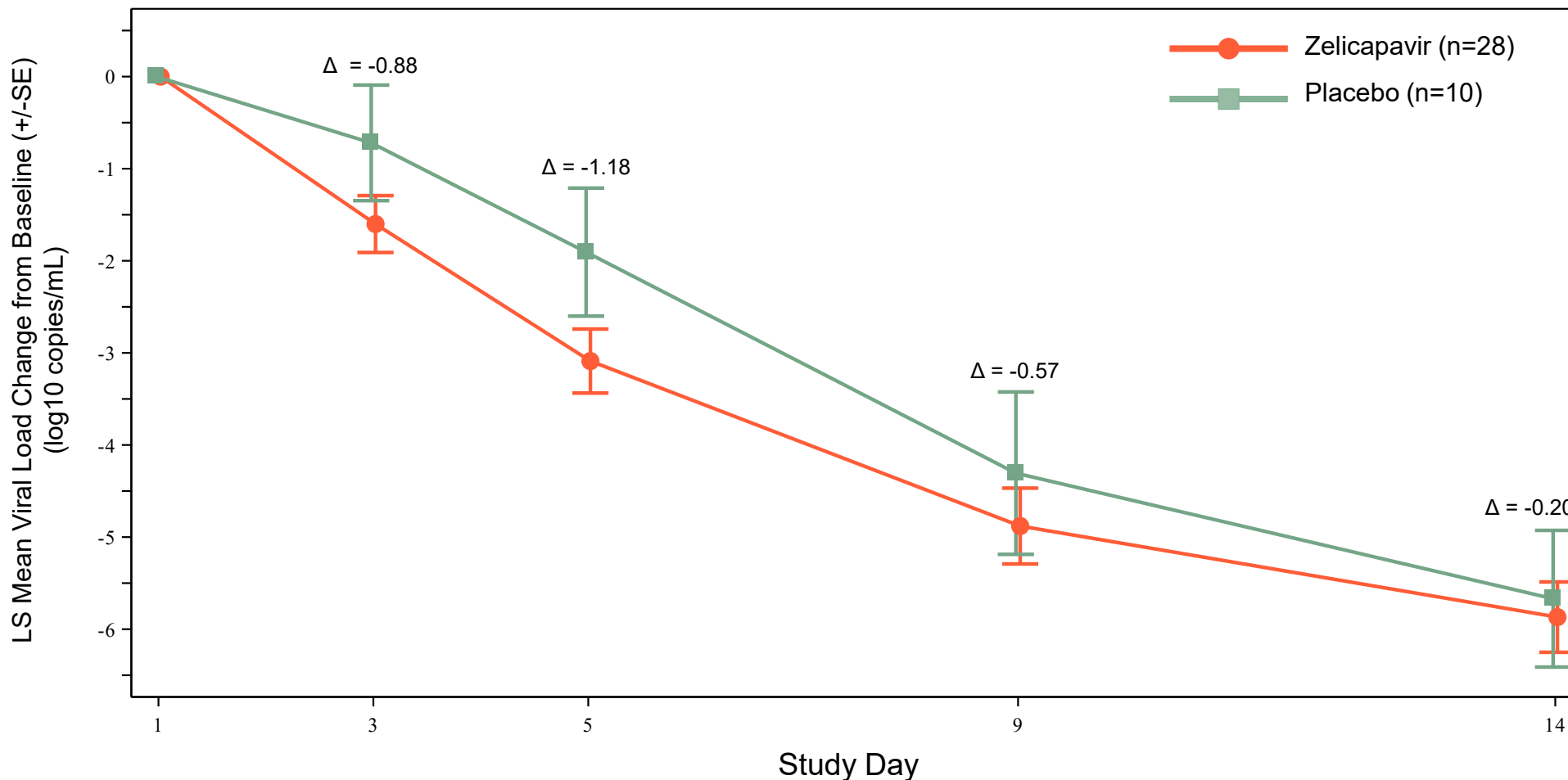
## Primary Objectives of Study

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

- First zelicapavir pediatric study: safety and dose selection
- Signal finding in different patient populations to inform a potential registration-enabling trial
  - Age: ≥28 days to <6 months and ≥6 months to ≤36 months
  - Time from symptom onset to treatment
  - Hospitalized or outpatient

# Zelicapavir Phase 2 Pediatric Study: Prespecified mITT-3 Population: RSV PCR Viral Load

- Viral load decline of 0.88 log at Day 3 and 1.18 log at Day 5



# Zelicapavir Phase 2 Pediatric Study: RSV Signs/Symptoms by ReSViNET

- In a post-hoc analysis, zelicapavir reduced the time to complete symptom resolution by 1.6 days and sustained resolution by 3.7 days compared to placebo

Symptom Resolution (ReSViNET)	Zelicapavir (n=69)	Placebo (n=27)	Difference
Median time to symptom resolution <sup>1</sup> (protocol-defined) (95% CI)	3.0 days (2.0-3.8)	2.7 days (1.0-4.1)	0.3 days
Median time to complete resolution <sup>2</sup> (post hoc) (95% CI)	7.0 days (5.9-8.0)	8.6 days (5.0-12.0)	-1.6 days
Median time to sustained resolution <sup>3</sup> (post hoc) (95% CI)	7.0 days (6.0-8.2)	10.7 days (5.2-13.0)	-3.7 days

ReSViNET: Respiratory Syncytial Virus Network.

1. All 7 symptoms were mild or absent; 2. All 7 symptoms were absent and patient was not hospitalized; 3. All 7 symptoms were absent, remained absent, and patient was not hospitalized.

# Zelicapavir Phase 2 Pediatric Study: Conclusions

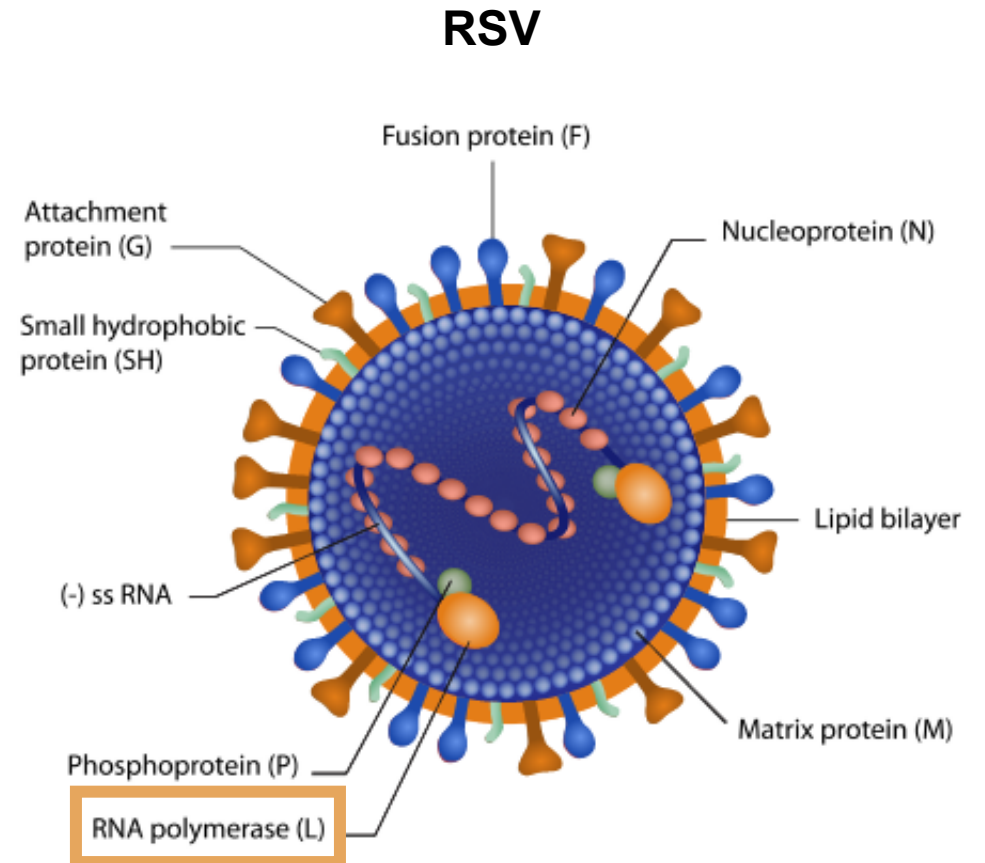
- Well-tolerated, with favorable safety profile
  - No adverse events leading to treatment discontinuation or study withdrawal
- Antiviral effect observed for the primary and secondary virology endpoints in overall population
- Viral load decline of 1.4 log at the end of treatment in Part 2
- Viral load 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
  - 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

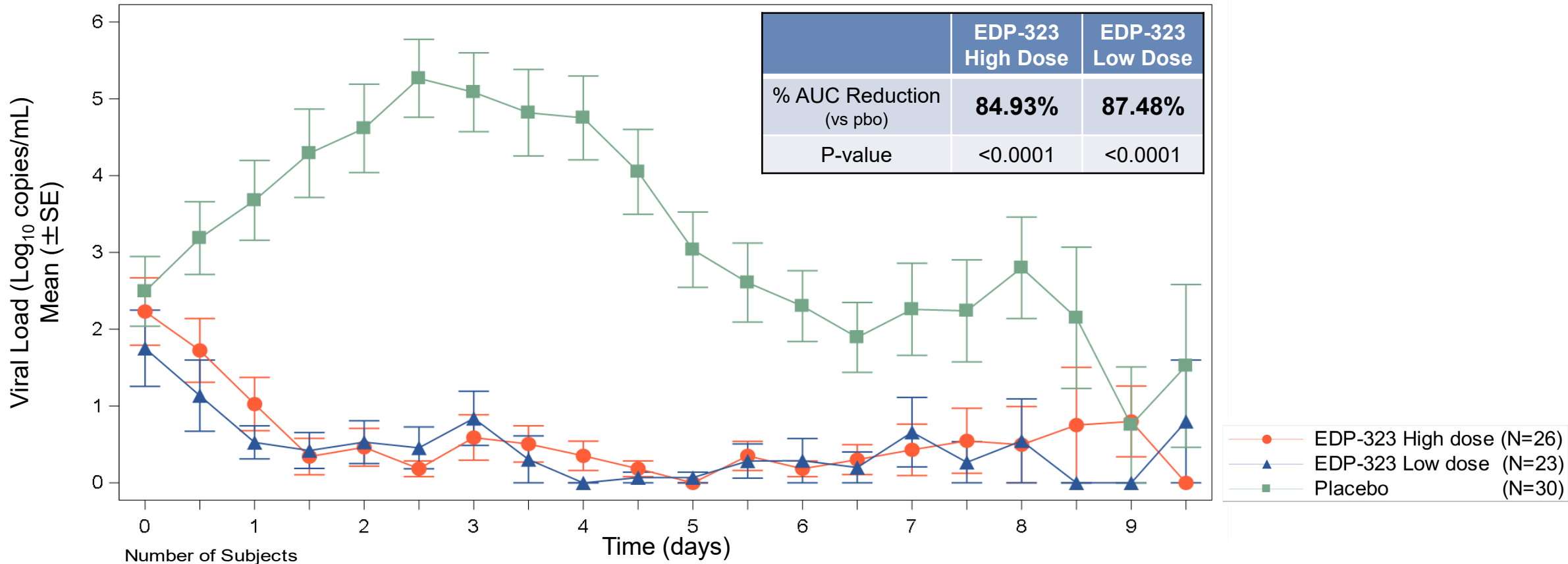
- Direct-acting antiviral targeting the L-protein
  - Replication inhibitor: shuts down the production of new virions (vs. fusion inhibitors which block viral entry)
- Granted Fast Track designation by the FDA
- Sub-nanomolar potency against RSV-A and B
- Protects mice in a dose-dependent manner from 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



# EDP-323: Robust Antiviral Effect in Human Challenge Model

Primary Efficacy Endpoint: 85-87% ↓ in Viral Load AUC by qRT-PCR

- Highly statistically significant reductions in Viral Load AUC measured by qRT-PCR compared to placebo
  - No statistically significant difference between the two EDP-323 dosing regimens; 600mg and 200mg (with 600mg loading dose)



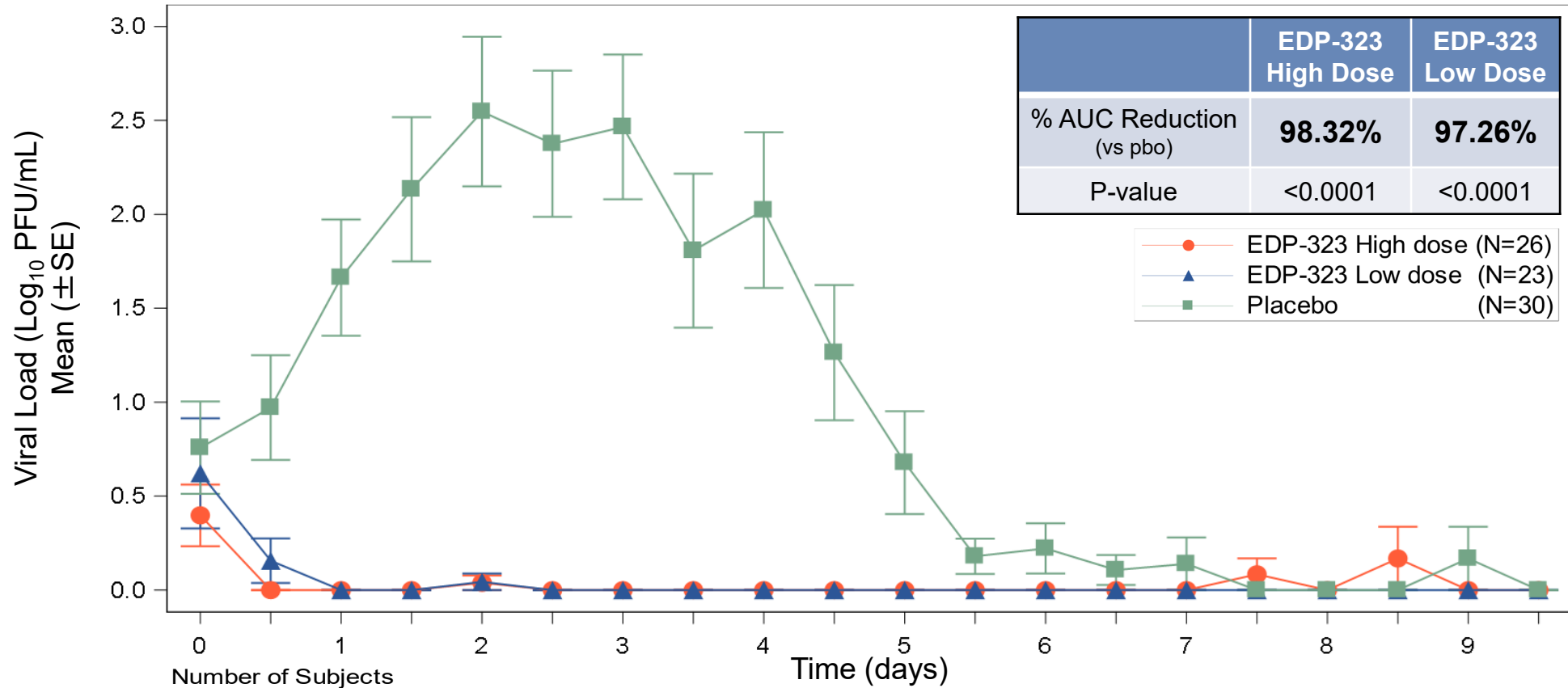
	0	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20					
EDP-323 High dose	26	26	26	26	26	26	26	26	26	26	26	26	26	26	26	26	26	26	26	26	15	12	10	6	4	1
EDP-323 Low dose	23	23	23	23	23	23	23	23	23	23	23	23	23	23	23	23	23	23	23	23	17	16	10	5	4	2
Placebo	30	30	30	30	30	30	30	30	30	30	30	30	30	30	30	30	30	30	30	30	19	18	11	8	6	4

VL AUC: viral load area under the curve (hours x log10 copies/mL); qRT-PCR: quantitative reverse transcriptase polymerase chain reaction; SE: standard error

# EDP-323: Robust Antiviral Effect in Human Challenge Model

## Secondary Efficacy Endpoint: 97-98% ↓ in VL AUC by Viral Culture

- Highly statistically significant reductions in infectious VL AUC measured by quantitative culture compared to placebo
  - No statistically significant difference between the two EDP-323 dosing regimens; 600mg and 200mg (with 600mg loading dose)



	0	1	2	3	4	5	6	7	8	9
EDP-323 High dose	26	26	26	26	26	26	26	26	15	12
EDP-323 Low dose	23	23	23	23	23	23	23	23	17	16
Placebo	30	30	30	30	30	30	30	30	19	18

VL AUC: viral load area under the curve (hours x log10 copies/mL); PFU: plaque forming units; SE: standard error

# EDP-323 Phase 2a RSV Challenge Study:

## Conclusions

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- 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%

Data support further clinical development of EDP-323

# Immunology

# 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

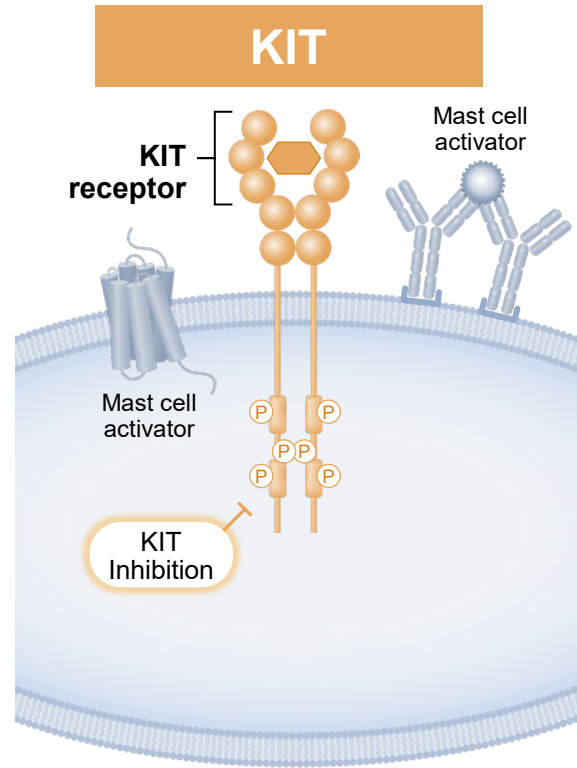
Significant Unmet Need



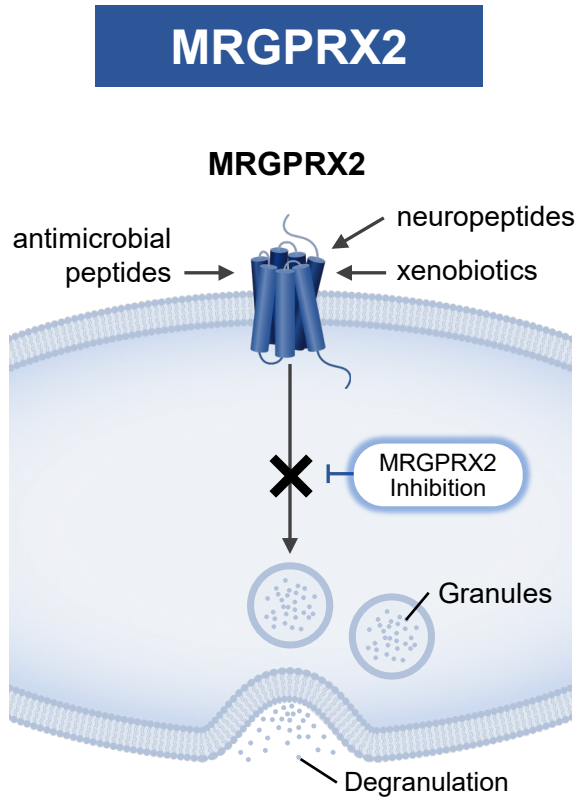
Differentiated value proposition;  
Large market opportunity

# Immunology Portfolio:

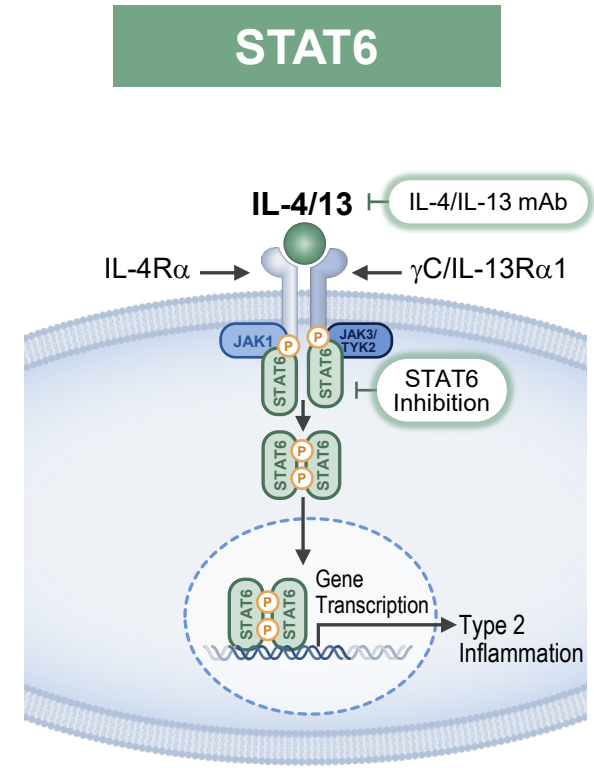
## Multiple Targets Addressing Type 2 Immune Diseases



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



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



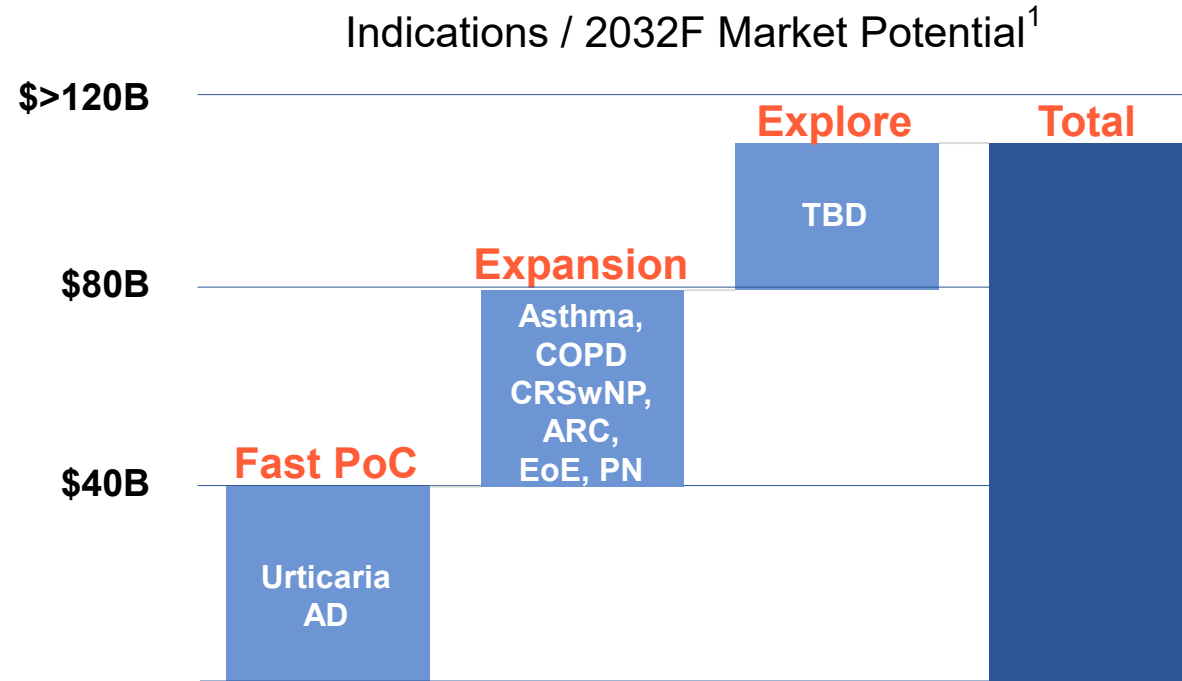
**STAT6** Inhibition impedes type 2 inflammation

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

# 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 Rhinconjunctivitis; COPD: Chronic Obstructive Pulmonary Disease; CRSwNP: Chronic Rhinosinusitis with Nasal Polyps; EoE: Eosinophilic Esophagitis; PN: Prurigo Nodularis

Source: 1. Evaluate Pharma, © Evaluate Ltd, September 2025, [www.evaluate.com](http://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<sup>1</sup>
  - Quality of life impacts beyond the skin: sleep disturbances, fatigue, irritability, anxiety and depression<sup>2</sup>
- Affects ~0.5-1% of the global population<sup>1</sup>
- Substantial unmet need for efficacious oral agent
  - ~50% not controlled with antihistamines<sup>1,3</sup>

Hives Itch Erythema



Angioedema



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

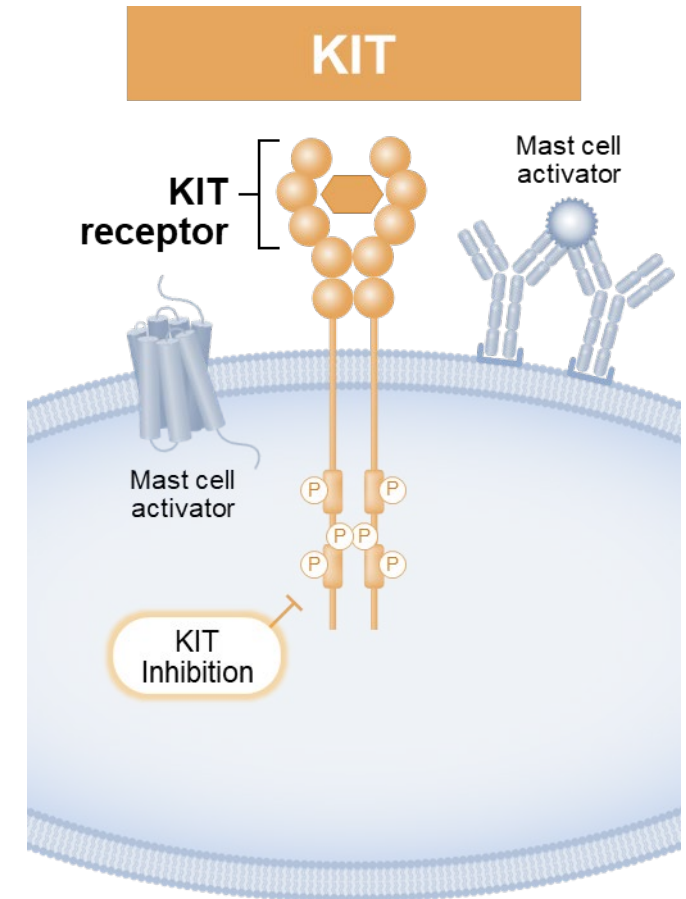
- Chronic dermatological disease characterized by dry, red, inflamed, irritated and itchy skin
  - Driven by Th2 immune dysregulation<sup>1</sup>
  - Quality of life impacts beyond the skin: limited lifestyle, avoidance of social interaction and impacted activities<sup>1</sup>
  
- 7.3% of U.S. adults, ~40% have moderate-severe symptoms<sup>2</sup>
  
- Significant need for efficacious and safe oral agents
  - Market dominated by mAb (IL-4/13)
  - Oral JAKi use <10%<sup>3</sup>
    - Boxed warning: serious infections, mortality, malignancy, major adverse cardiac events, thrombosis<sup>4</sup>



# Immunology: KIT Inhibitor

# 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

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- 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

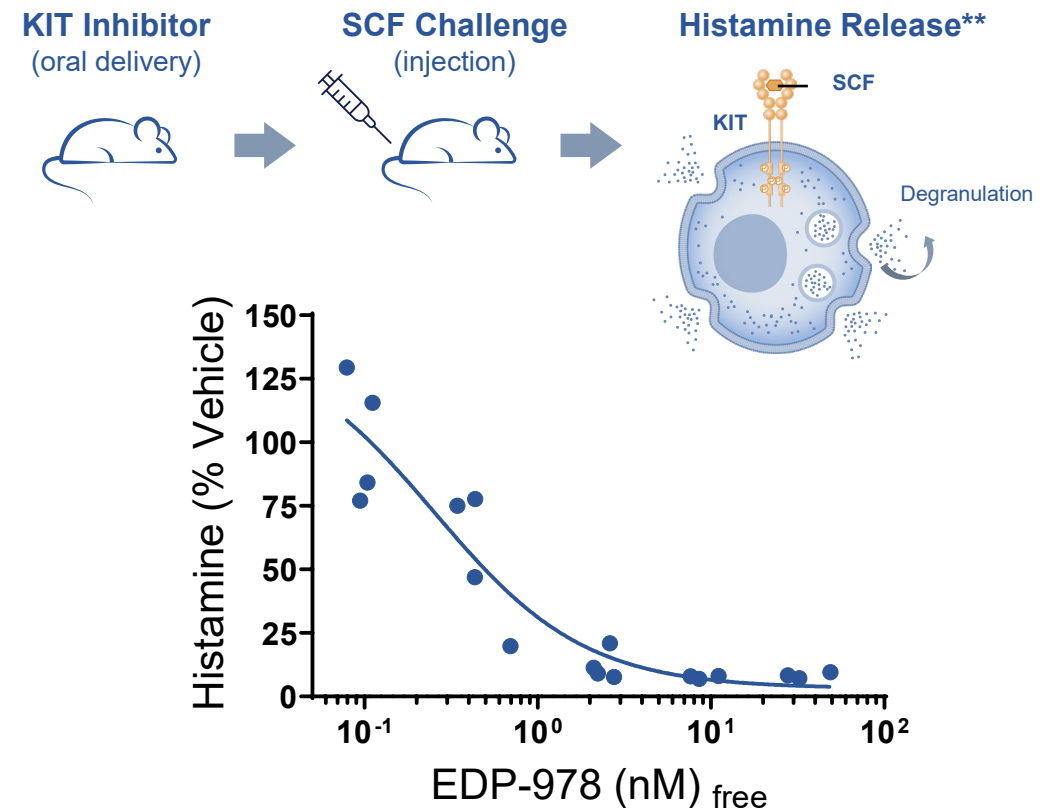
# EDP-978 Exhibits Potent Inhibition of KIT *In Vitro* and *In Vivo*

- Nanomolar inhibition in cellular assays with an EC<sub>50</sub> of **1.6 – 3.4nM**

Binding or Cellular Assay*	EC <sub>50</sub> (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 <sup>+</sup> Derived Mast Cell Degranulation	3.4

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

- Inhibits SCF-mediated histamine release in mice with an EC<sub>50</sub> (free drug) of **0.25nM**



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

# EDP-978 Demonstrates Good Selectivity for KIT

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

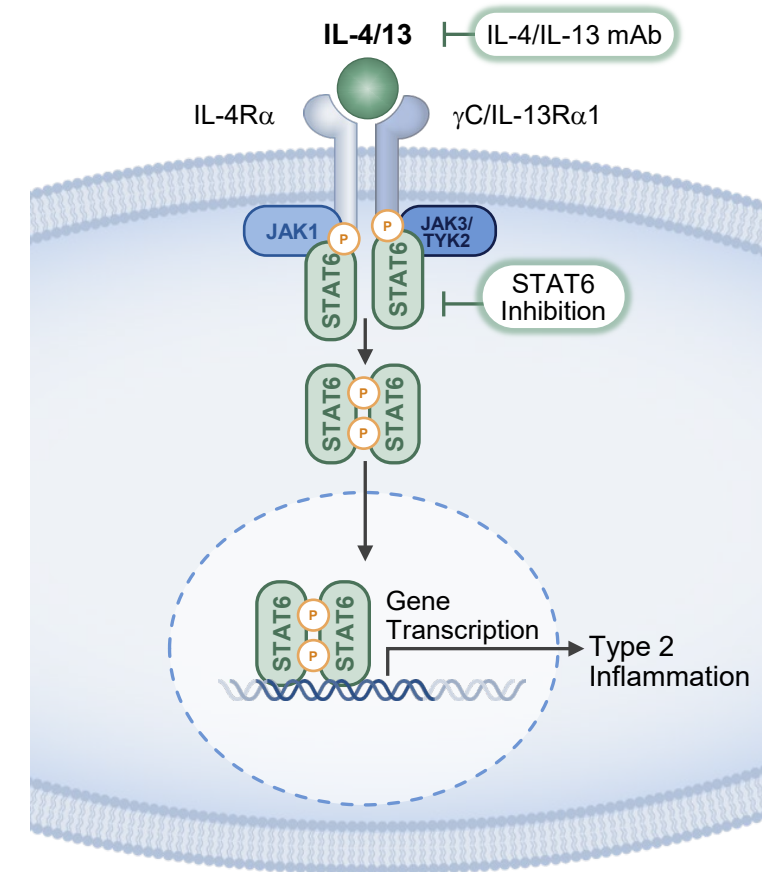
Selectivity	EDP-978
S(10) @ 1 $\mu$ M	0.02
CSF1R*	>200x
PDGFR $\alpha$ *	>900x
PDGFR $\beta$ *	>800x
FLT3 Kd	>30,000x

\*Cellular selectivity calculated as ratio of EC<sub>50</sub> in kinase dependent Ba/F3 cells

# Immunology: STAT6 Inhibitor

# 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<sup>1</sup>
  - STAT6 loss-of-function protects against type 2 high asthma<sup>2</sup>
  - 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



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

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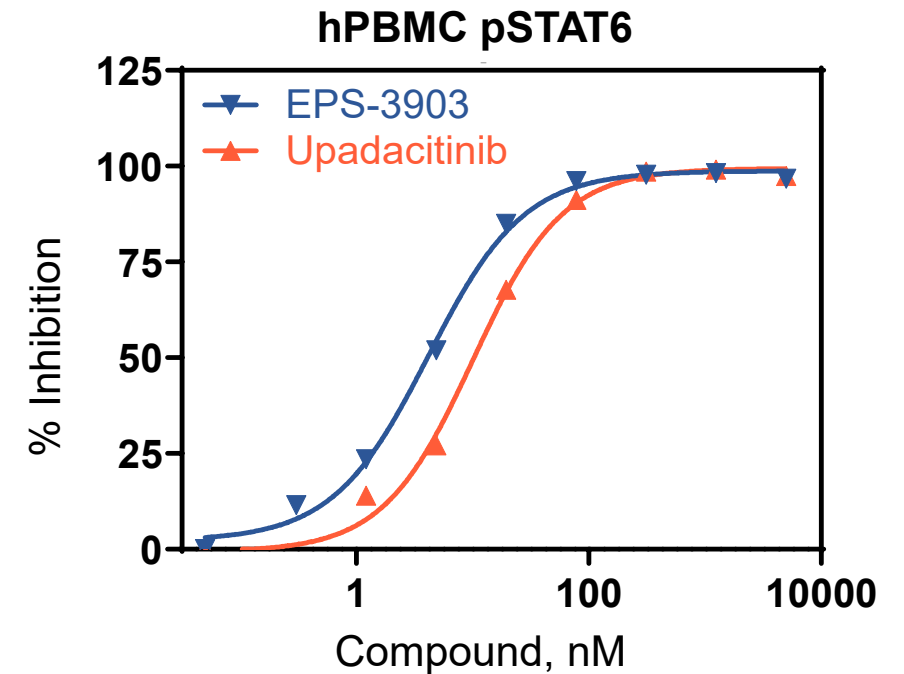
- EPS-3903 selected as development candidate
  - Favorable *in vitro* and *in vivo* ADME properties supporting once daily dosing
  - Inhibits STAT6 with nanomolar potency in both binding and cellular assays
  - Highly selective for STAT6 versus other STATs
  - Rapid, continuous and complete (>90%) inhibition 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

- EPS-3903 exhibits nanomolar inhibition in biochemical ( $K_d = 0.4 \text{ nM}$ ) 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 $EC_{50}$ (nM)	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  
Upadacitinib: JAK Inhibitor



# EPS-3903 Displays Good Selectivity

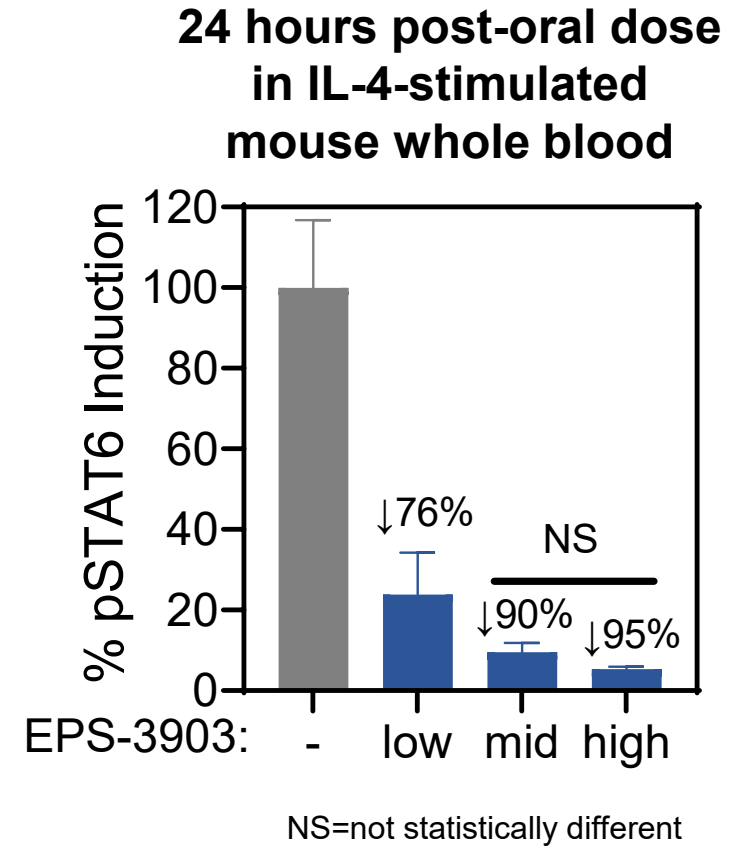
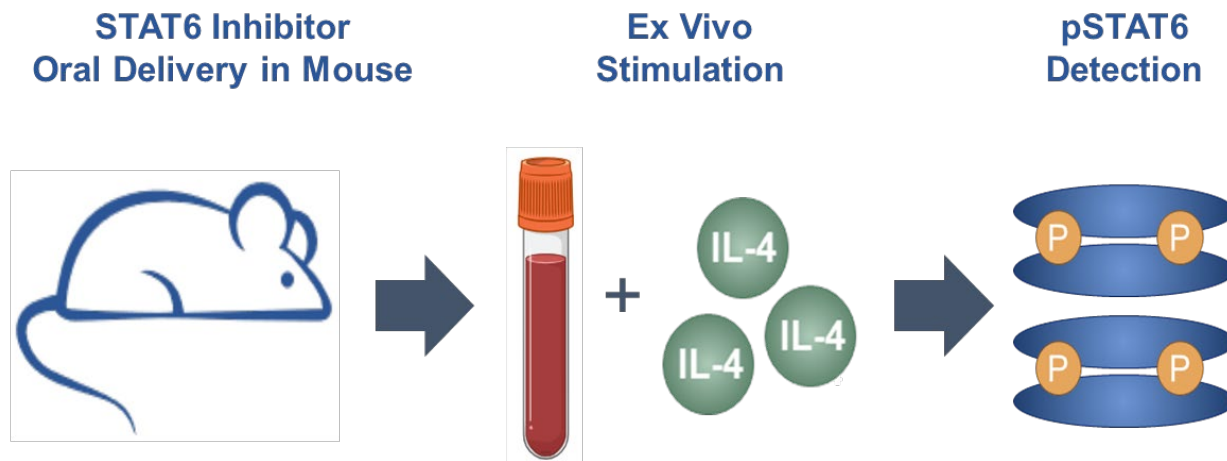
- No inhibition of other STATs observed in human PBMCs
- Greater than 1000x biochemical selectivity for STAT6 over other STATs
- Selectivity much improved compared to JAK inhibitors

**Selectivity in hPBMCs EC<sub>50</sub> (nM)**

	pSTAT1	pSTAT2	pSTAT3	pSTAT4	pSTAT5	pSTAT6
EPS-3903	>5000	>5000	>5000	>5000	>5000	4
Upadacitinib (JAK Inhibitor)	1	8	24	195	16	10

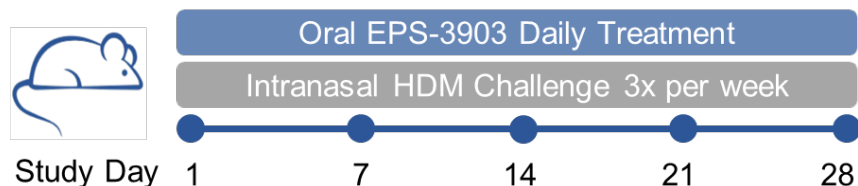
# EPS-3903 Results in Complete Inhibition of pSTAT6 in Mouse Model

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



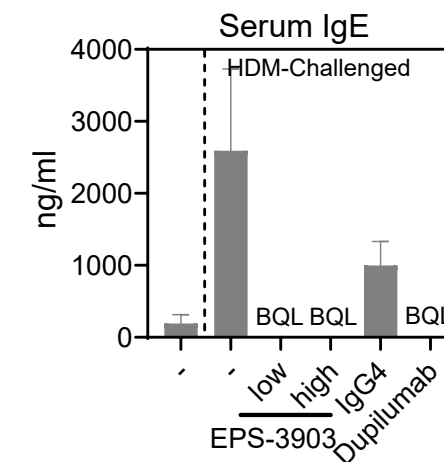
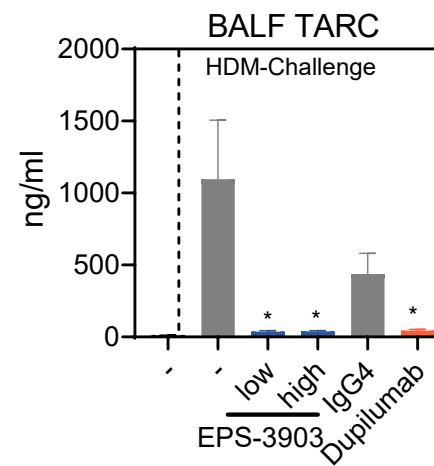
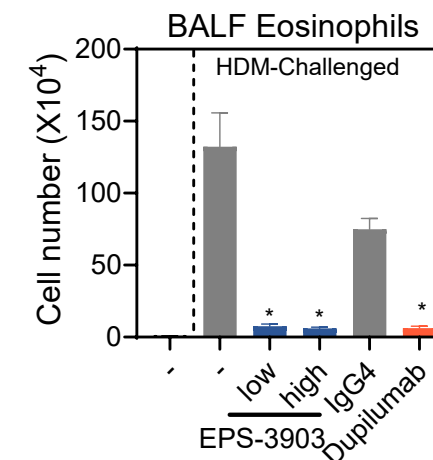
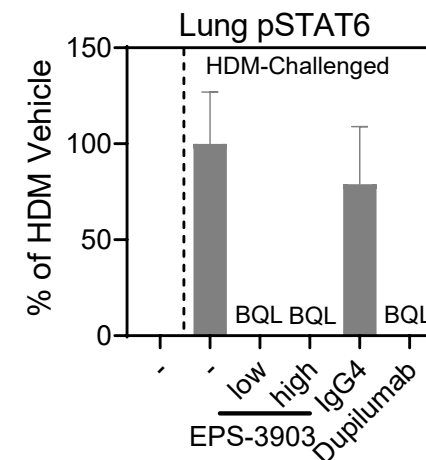
# EPS-3903 Shows Comparable Efficacy to Dupilumab in a House Dust Mite (HDM) Challenge Asthma Model

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



## EPS-3903 efficacy comparable to dupilumab

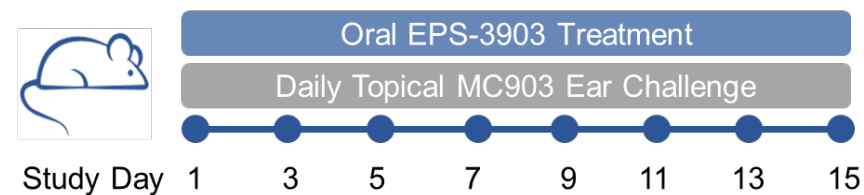
- >90% inhibition of pSTAT6 in lung
- Decreased type 2 inflammation
  - BALF Eosinophils, TARC, Serum IgE
  - Improved histological scoring
    - Reduced bronchiolar hyperplasia, inflammatory infiltrates, airway wall thickness



\*p<0.05 vs Corresponding Vehicle

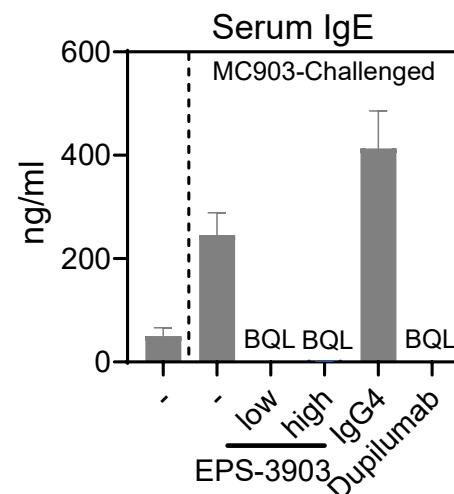
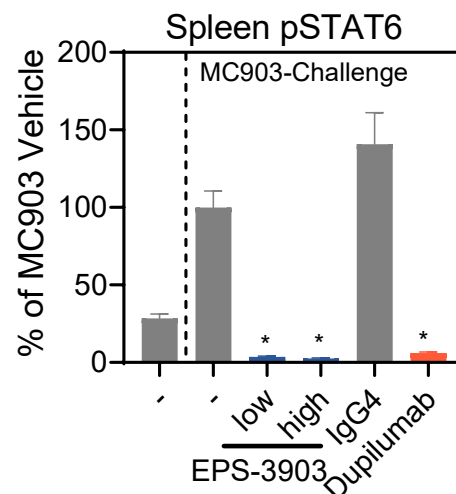
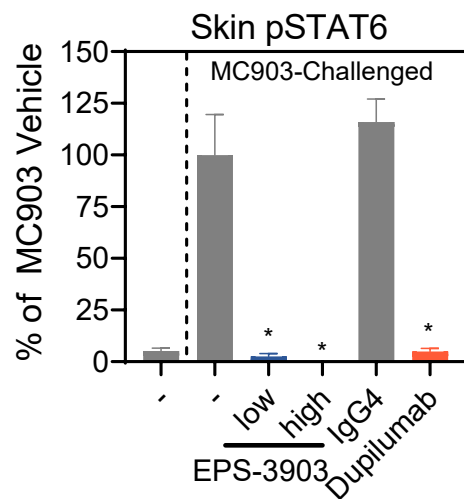
# EPS-3903 Demonstrates Comparable Efficacy to Dupilumab in a MC903-Driven Atopic Dermatitis Mouse Model

- MC903 model in transgenic mice expressing human IL-4/IL-4R $\alpha$  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

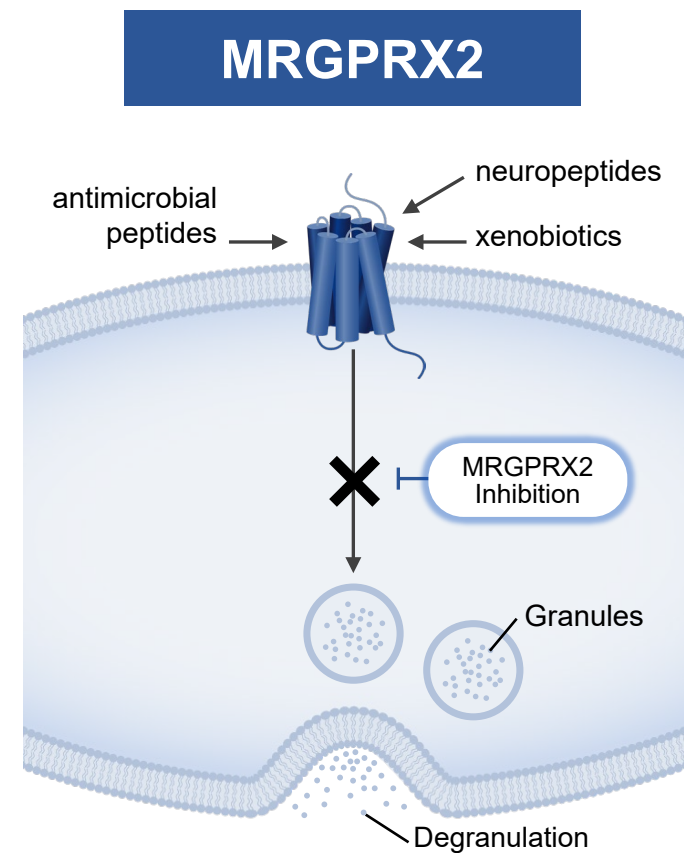


\*p<0.05 vs Corresponding Vehicle

# Immunology: MRGPRX2 Inhibitor

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

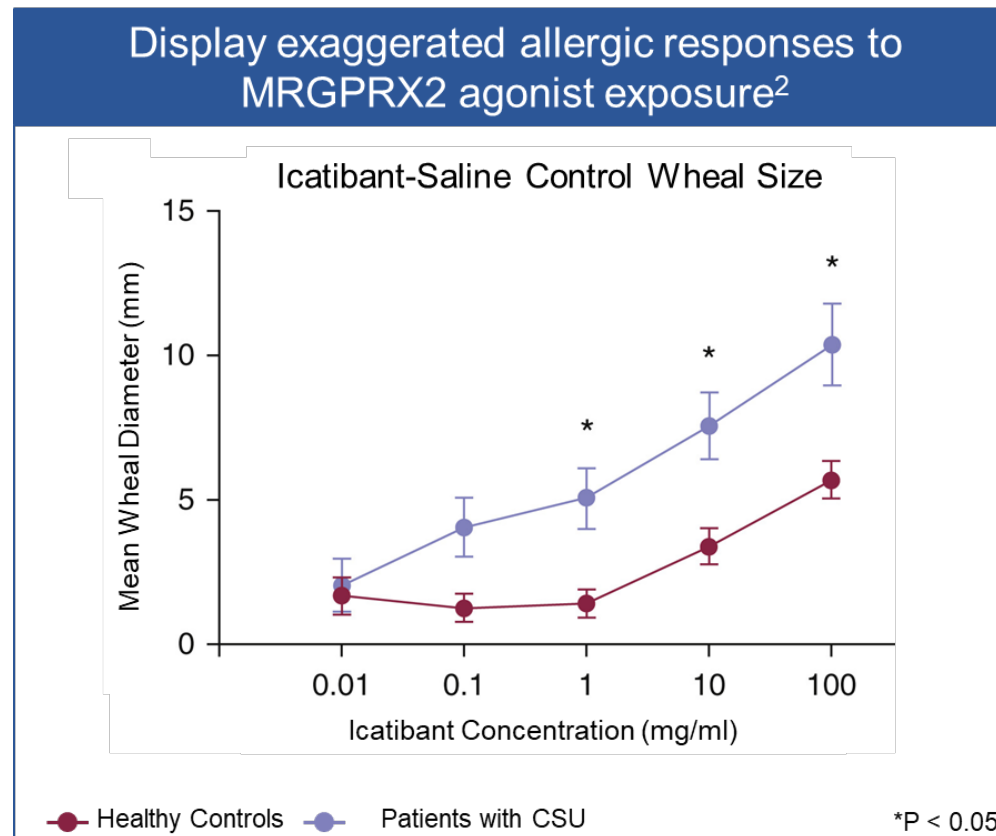
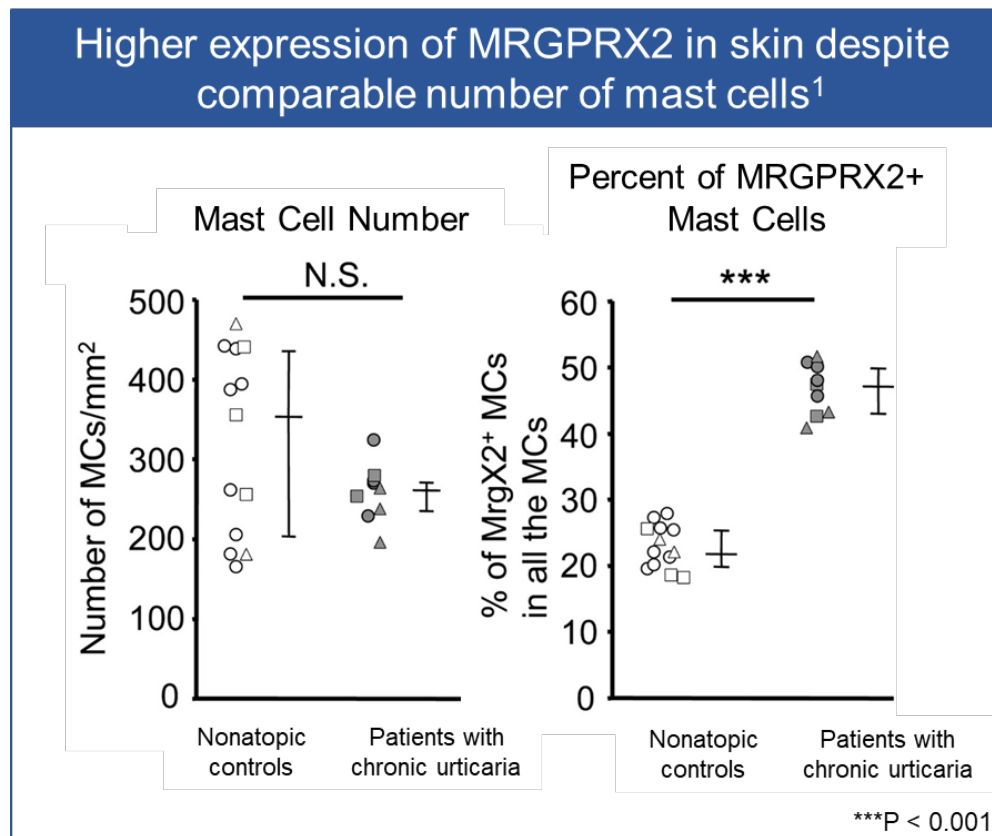
- **Mast Cells:** primary driver of inflammation in skin, and implicated in multiple allergic diseases
  - Urticaria, asthma, prurigo nodularis (PN) and others
- **MRGPRX2:** non-canonical GPCR expressed predominantly on mast cells
  - Agonism leads to degranulation and release of inflammation mediating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



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

# MRGPRX2: Evidence for a Key Role in Chronic Spontaneous Urticaria

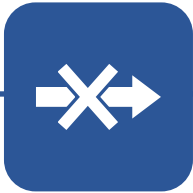
- Patients with CSU have more MRGPRX2 expressing mast cells and a greater response to MRGPRX2 agonist



# Goal of Oral MRGPRX2 Inhibitor Program:

## Develop Best-in-Class Treatment for Mast Cell Mediated Diseases

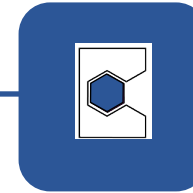
- Novel, oral inhibitors of MRGPRX2 being optimized in discovery stage



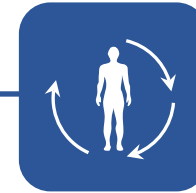
Inhibits  
MRGPRX2  
with nanomolar  
potency in cellular  
assays



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

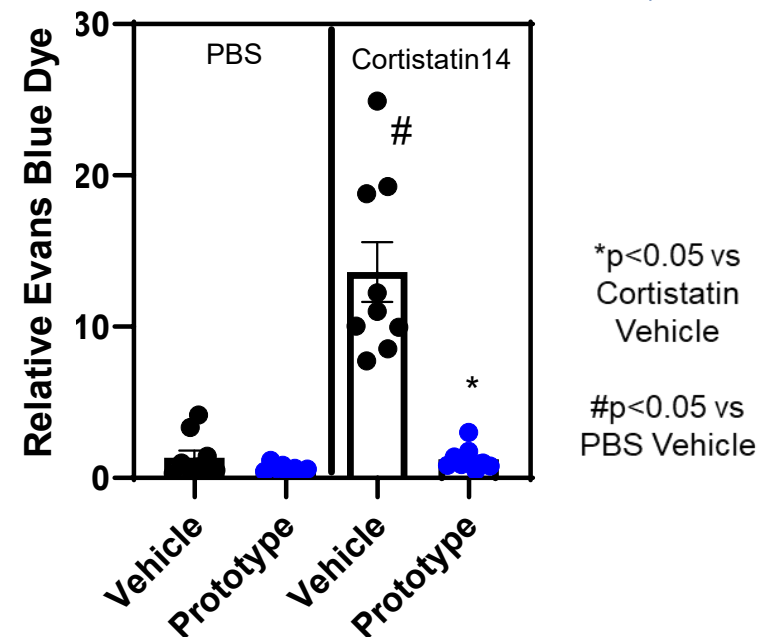
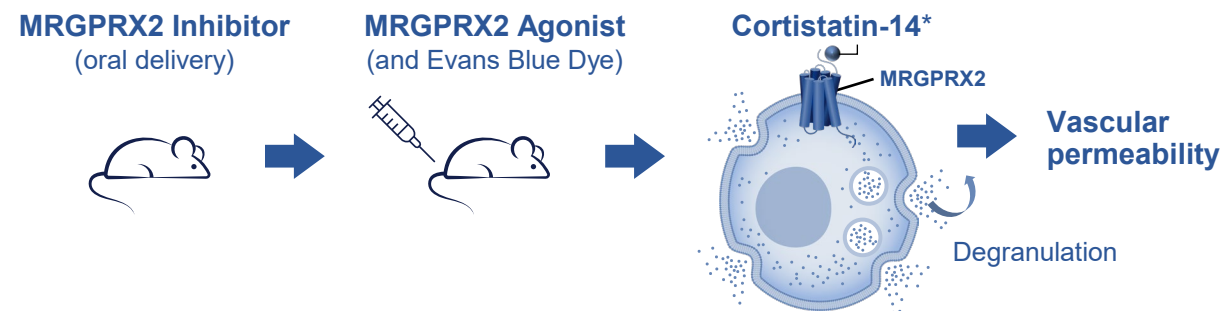
# Prototype MRGPRX2 Inhibitor Exhibits Potent Inhibition *In Vitro* and *In Vivo*

- Nanomolar inhibition in cellular assays with an EC<sub>50</sub> of **1 – 2nM**

Cellular Assay	EC <sub>50</sub> (nM)
LAD2 Cells (β-hexosaminidase release)	1.7
LAD2 Cells (Histamine Release)	2.1
Primary Human Skin Mast Cells (β-hexosaminidase release)	1.2

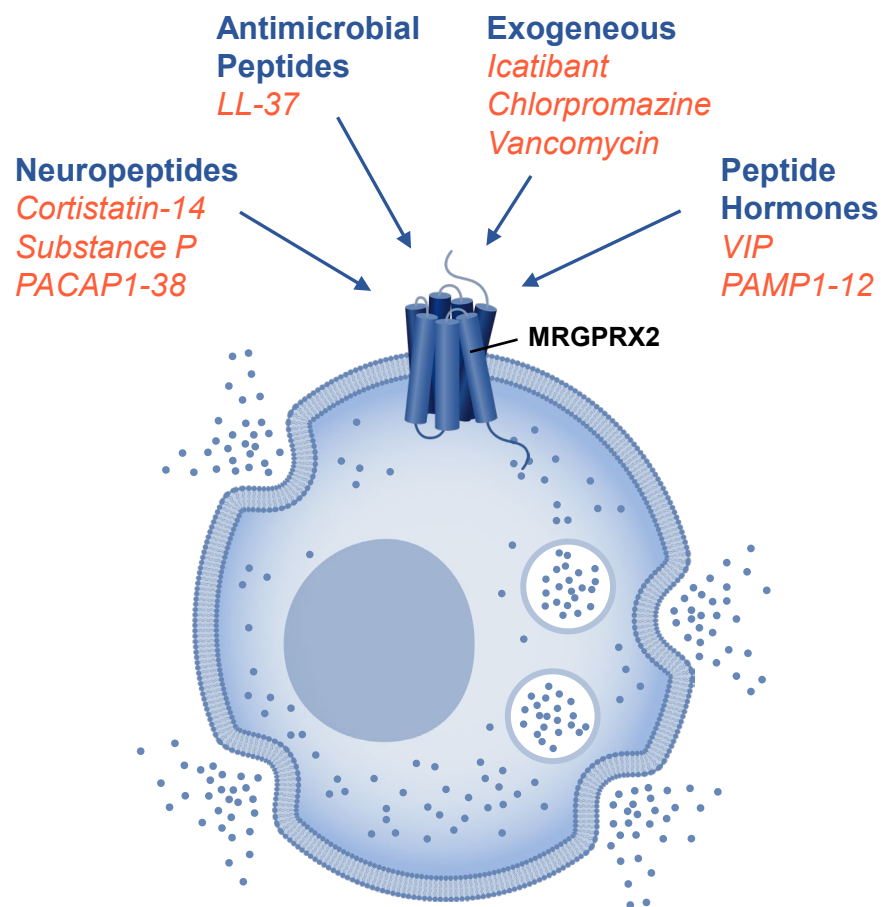
Cells stimulated with MRGPRX2 agonist Cortistatin-14  
LAD2 Cells: human mast cell line capable of histamine release (degranulation)

- Prevents skin mast cell activation in humanized MRGPRX2 mice<sup>1</sup>



# Prototype MRGPRX2 Inhibitor Demonstrates Potent Activity Across Multiple Agonists

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



Agonist	$\beta$ -hexosaminidase EC <sub>50</sub> (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

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## Virology

### Respiratory Syncytial Virus

- Align with FDA on adult Phase 3 design and registration path in 2Q 2026
- Prepare for a Phase 3 study of zelicapavir in high-risk adults in 2H 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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