Enanta Pharmaceuticals

Great Chemistry Cures

Corporate Presentation

December 9, 2024

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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A Proven Approach to Drug Discovery

Leveraging our core strength in small molecule drug discovery to develop treatments for high unmet needs

Robust Pipeline

Virology:
- Phase 2 in pediatric patients with RSV complete
- Phase 2 in high-risk adults with RSV ongoing
- Phase 2 challenge study with second RSV candidate complete
- Phase 2 in COVID-19 complete

Immunology:
- Preclinical KIT inhibitor (mast cell driven diseases, e.g., CSU)
- Preclinical STAT6 inhibitor (type 2 immune diseases, e.g., atopic dermatitis)

Proven Track Record of Success

Glecaprevir: HCV protease inhibitor in MAVYRET®/MAVIRET®

Strong Balance Sheet

Strong balance sheet and royalties to support robust pipeline

$248.2M in cash at September 30, 2024

Enanta Pipeline

	DISEASE	TARGET	DISCOVERY	PRECLINICAL	PHASE 1	PHASE 2	PHASE 3	MARKET
Virology: Liver	**Hepatitis C Virus**	Protease	**Glecaprevir***					MAVYRET glecaprevir/pibrentasvir
	Hepatitis B Virus	Core	**EDP-514****					
Virology: Respiratory	**Respiratory Syncytial Virus**	N-Protein	**Zelicapavir (EDP-938)**				***Pediatrics***	
			Zelicapavir (EDP-938)			***High Risk Adults***		
		L-Protein	**EDP-323**			***(challenge study)***		
	COVID-19	3CL Protease	**EDP-235****				***SPRINT***	
Immunology: Type 2 Immune Diseases	**Chronic Spontaneous Urticaria*****	KIT						
	Atopic Dermatitis***	STAT6						

*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), and others.

Virology: Hepatitis C Virus

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Glecaprevir: Our Licensed Protease Inhibitor for Hepatitis C Virus

Marketed Product
MAVYRET® glecaprevir/pibrentasvir 100 mg/40 mg tablets
Regimen 2-DAA (AbbVie)
Enanta Asset Glecaprevir (protease inhibitor)

Fiscal 2024 GAAP Royalty Revenue to ENTA (millions)

Q1	Q2	Q3	Q4	Total
$18.00	$17.05	$17.97	$14.60	**$67.6**

Enanta retains 45.5% of royalty cash payments and continues to record 100% of royalty revenue earned in its GAAP reporting*

* Royalty sale from April 2023 is accounted for as debt under GAAP reporting

Virology: Respiratory Syncytial Virus

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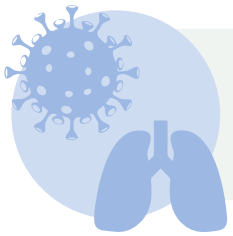
Respiratory Syncytial Virus (RSV)



Causes severe lung infections, including bronchiolitis (infection of small airways in the lungs) and pneumonia. Leading cause of hospitalization in infants[1]. No safe and effective treatments are currently approved.

Populations at higher risk for severe illness:

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

RSV at a Glance

Children < 5 years[2]	Adults > 65 years[3]
33M global cases	
3M global hospitalizations	177K U.S. hospitalizations
101K global deaths	14K U.S. deaths

Significant unmet need for antiviral treatment despite availability of prophylaxis:

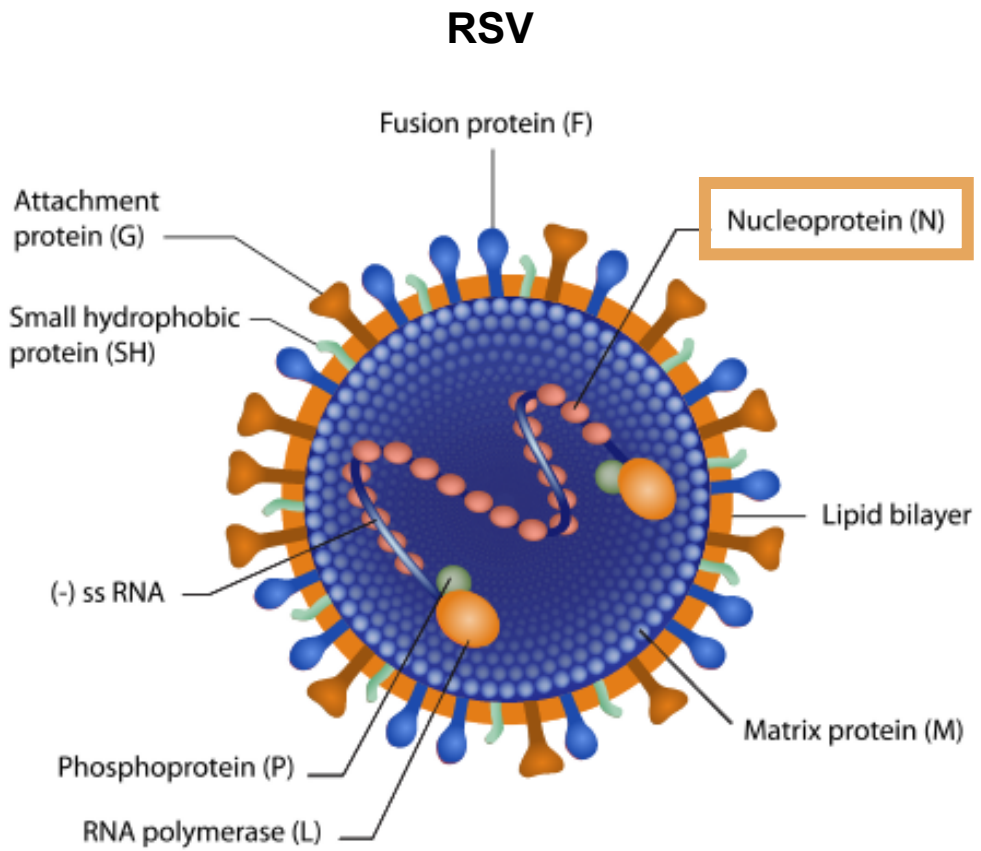
- Adoption of adult vaccines is sub-optimal and not recommended for all FDA-approved patient groups*
  - Peak adoption of vaccines for elderly range from ~35% (shingles[4]) to ~55% (flu[5])
- Pediatric prophylaxis approaches provide passive immunity; will shift first infection to next season
  - Antibody approach has a low barrier to resistance
- Even with adoption, breakthrough infections will still occur

*FDA-approved for adults age ≥60 & 50-59 years who are at increased risk for LRTD caused by RSV[6]
ACIP-recommended for adults age ≥75 years and age 60-74 years at increased risk of severe RSV[7]

Sources: 1. CDC RSV In Infants and Young Children. 2. Li P et al. *The Lancet*. 2022 3. Falsey AR et al. *NEJM*. 2005. 4. Terlizzi EP et al. *NCHS Data Brief*. 2020; Age 60+ 5. CDC Influenza Vaccination Coverage, Adults 65+ 6. FDA-approved labels: Pfizer; GSK & Moderna 7. June 2024 ACIP recommendation

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

- Zelicapavir is currently the 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 observed to date
  - Phase 2a challenge study showed a statistically significant ($p<0.001$) reduction in both viral load and clinical symptoms compared to placebo



Zelicapavir Development Plans:
Treatment for Patients at High-Risk for Severe RSV Infection

High-risk populations have reduced RSV immunity, resulting in a higher and longer duration of viral load and greater disease severity, allowing a bigger window to observe benefit

Goal: Treat patients at high-risk for developing severe infection leading to hospitalization or death, populations with the most significant unmet need

RSVHR

- Age ≥65 years
- Chronic heart or lung disease (e.g. COPD, CHF, asthma)

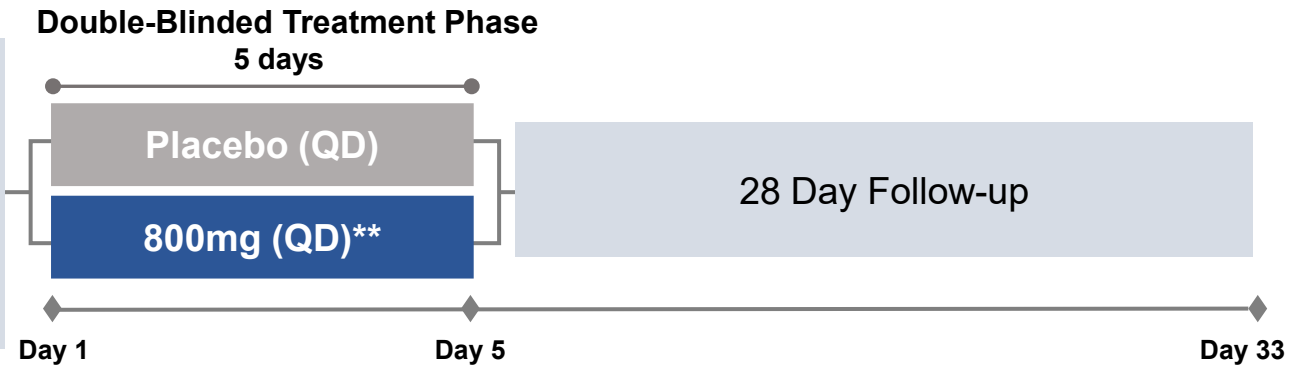
RSVPEDs

- Infants and young children

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

RSVHR

~180 adults with at least one of the following:
- COPD
- Congestive heart failure
- Asthma*
- Age ≥65*



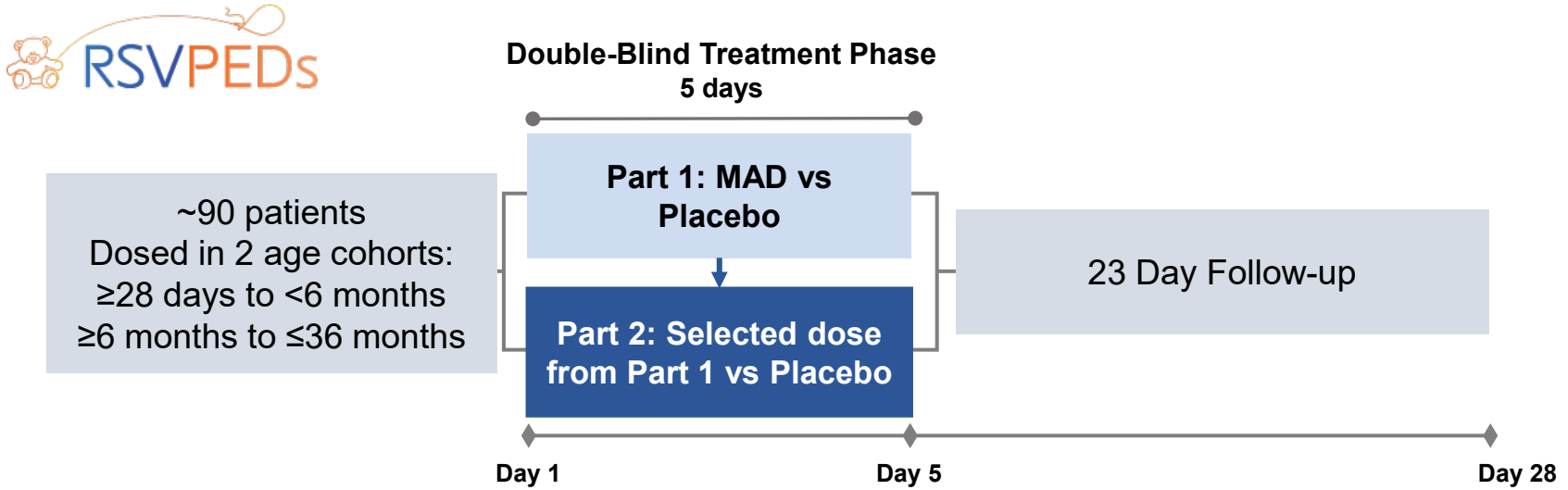
- **Primary Objective:** Time to resolution of RSV LRTD symptoms as assessed by RiiQ symptom scale through Day 33
- **Secondary Objectives:** PROs, MAVs, viral load, antibiotic use, bronchodilator use, corticosteroid use, hospitalization, ICU, mechanical ventilation, all cause mortality, PK and safety

* The total proportion of subjects either 65-74 years of age or patients with asthma combined will be capped at 20%.

** Equivalent to 600mg suspension dosage form used in challenge study

PK: pharmacokinetics; LRTD: lower respiratory tract disease; PROs: patient reported outcomes; MAVs: medically attended visits

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

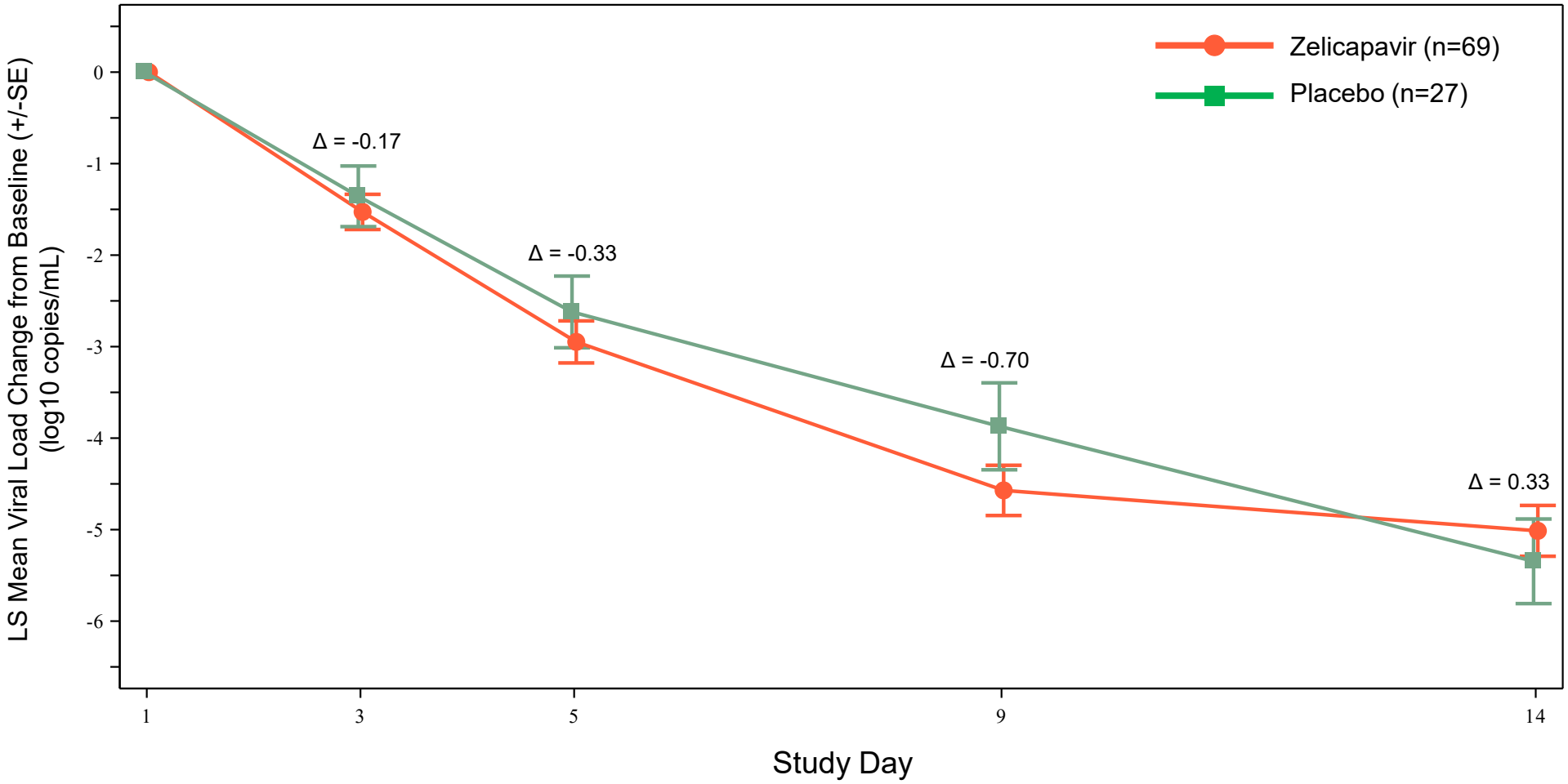
- First zelicapavir pediatric study: safety and dose selection
- Signal finding in different patient populations to inform a potential registration-enabling trial
  - Age: ≥28 days to <6 months and ≥6 months to ≤36 months
  - Time from symptom onset to treatment
  - Hospitalized or outpatient

Zelicapavir Phase 2 Pediatric Study: Virology Summary

- Antiviral effect observed for the primary and secondary virology endpoints in overall population
- Viral load decline in Part 2 of 1.0 log at Day 3 and 1.4 log at Day 5 vs placebo
- Rapid and robust virology effects observed in prespecified subset of patients who were randomized within 3 days of symptom onset (mITT-3)
  - Represents ~40% of the study population (n=38/96)
  - Viral load decline of 0.9 log at Day 3 and 1.2 log at Day 5 vs placebo
  - Greater proportion of patients had undetectable viral load at Days 5 and 9 vs placebo
  - Qualitative improvement in time to undetectable viral load at early timepoints, although median time to undetectable viral load was similar between groups
  - Improvement in AUC of change from baseline for viral load at all timepoints vs placebo
- Results were similar regardless of age or setting of care (outpatient and hospitalized)

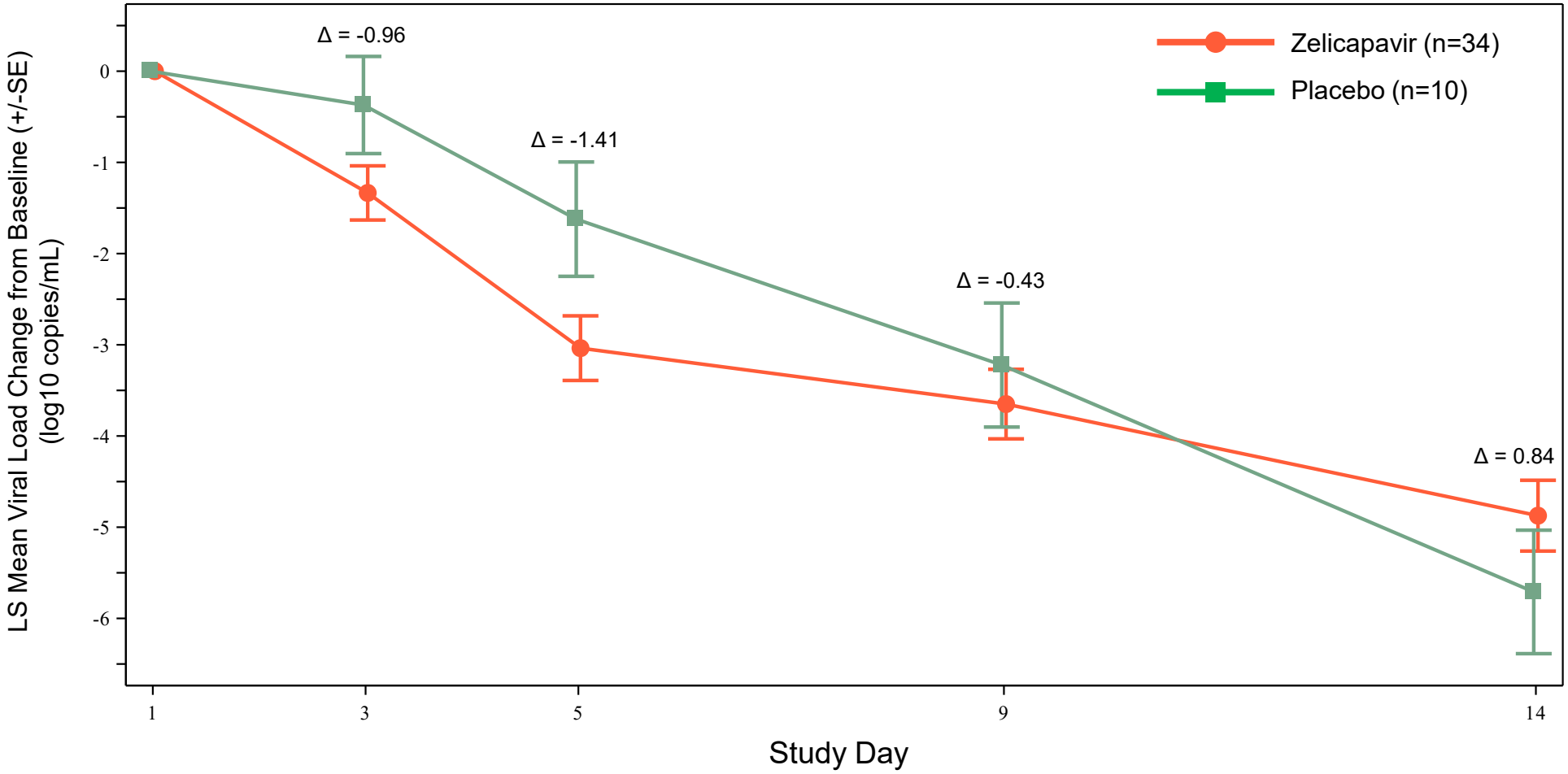
Zelicapavir Phase 2 Pediatric Study: Primary Endpoint – RSV PCR Viral Load for All Patients (Part 1 & 2)

- Trend toward greater viral load decline in patients treated with zelicapavir compared to placebo



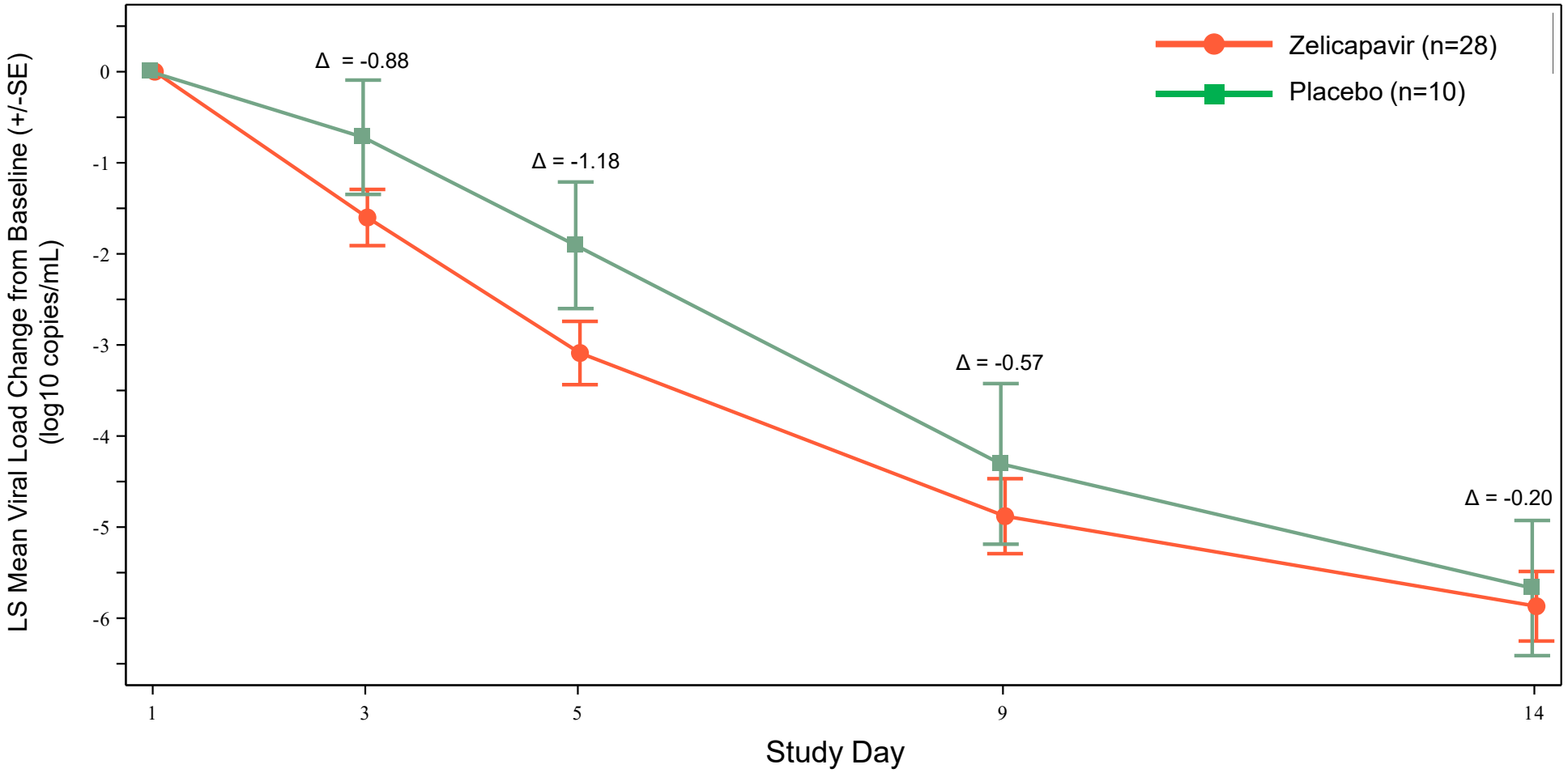
Zelicapavir Phase 2 Pediatric Study: Primary Endpoint of Part 2: RSV PCR Viral Load

- Viral load decline of **0.96 log** at Day 3 and **1.41 log** at Day 5



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



mITT-3: patients randomized within 3 days of symptom onset

Zelicapavir Phase 2 Pediatric Study: Exhibited Favorable Safety Profile in Children

- Adverse events (AEs) were similar between zelicapavir dosing groups and placebo
- No adverse events led to treatment discontinuation or study withdrawal

Description	**Zelicapavir** (pooled) (N=70)	**Placebo** (pooled) (N=26)
Treatment emergent AEs (TEAEs)	28 (40.0%)	13 (50.0%)
Study drug related TEAEs	6 (8.6%)	0 (0.0%)
Grade 3 or higher TEAEs	2 (2.9%)	1 (3.8%)
Serious TEAEs	1 (1.4%)	2 (7.7%)

Zelicapavir Phase 2 Pediatric Study: AEs Occurring in More than One Patient in Any Group

- Adverse events (AEs) were balanced between zelicapavir and placebo
- The two most common AEs in the zelicapavir group were diarrhea and rash

Preferred Term	Zelicapavir (pooled) (N=70)	Placebo (pooled) (N=26)
Diarrhea	7 (10.0%)	1 (3.8%)
Rash	3 (4.3%)	1 (3.8%)
Otitis media acute	2 (2.9%)	1 (3.8%)
Eczema	2 (2.9%)	1 (3.8%)
Thrombocytosis	2 (2.9%)	0 (0%)
Nasopharyngitis	1 (1.4%)	2 (7.7%)

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: No difference in signs/symptoms between groups
  - RESOLVE-P: Trend toward greater signs/symptom reduction with zelicapavir in a small dataset
- Data support further clinical development of zelicapavir

Primary Objectives of Study

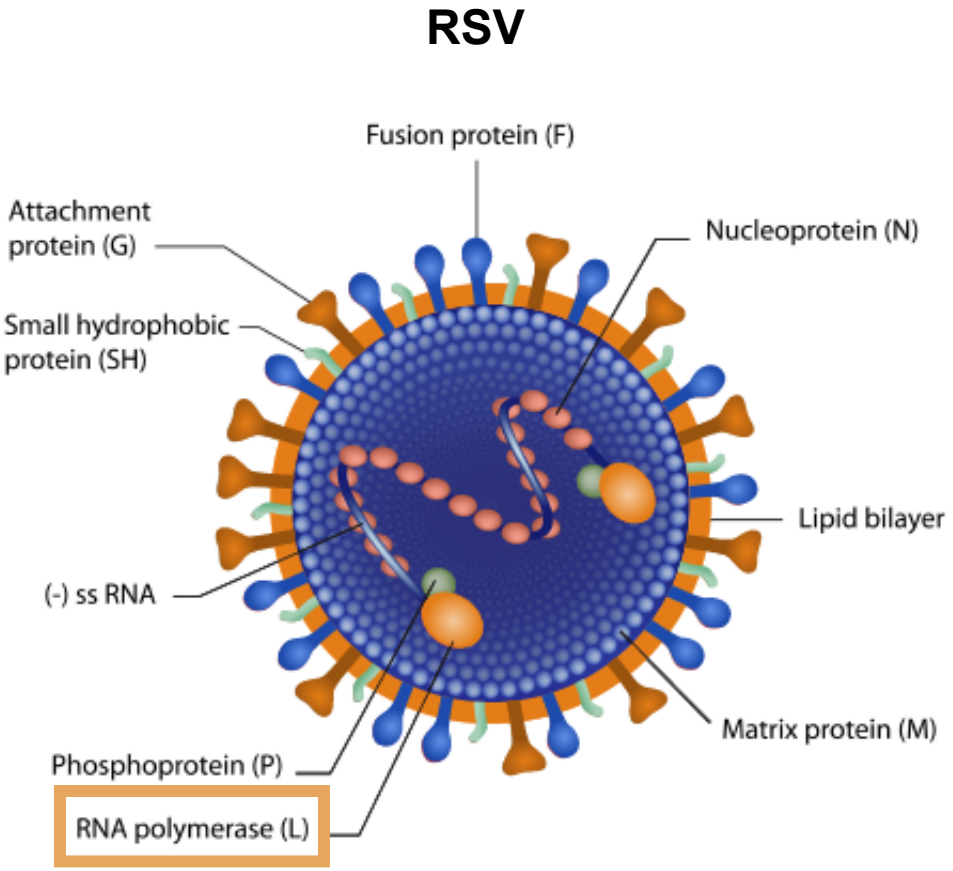
✓ **Overall:** Antiviral activity of zelicapavir across all patients

✓ **Part 1:** Safety and PK

✓ **Part 2:** Antiviral activity

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
- Potential to be used alone or in combination
  - Additive to synergistic activity with zelicapavir
  - No cross resistance expected with other mechanisms
- Sub-nanomolar potency against RSV-A and B
- Protects mice in a dose-dependent manner from RSV infection
- Phase 1 indicated 200mg or 600mg once-daily as potential safe and efficacious doses



EDP-323 Demonstrated Favorable Safety Profile and Achieved Target Exposure Levels in Human Challenge Model

- Favorable safety profile over a 5-day dosing period and through 28 days of follow-up
- No serious or severe AEs and no AEs leading to treatment discontinuation or study withdrawal
- AEs were similar between EDP-323 dosing groups and placebo
- No specific pattern of treatment-emergent AEs was identified

Description	EDP-323 600mg (N=47)	EDP-323 200mg w/ LD* (N=47)	Pooled EDP-323 (N=94)	Placebo (N=47)
Any **treatment emergent** AEs	11 (23.4%)	14 (29.8%)	25 (26.6%)	13 (27.7%)
Any treatment emergent AEs considered **related** to study treatment	1 (2.1%)	1 (2.1%)	2 (2.1%)	0 (0.0%)

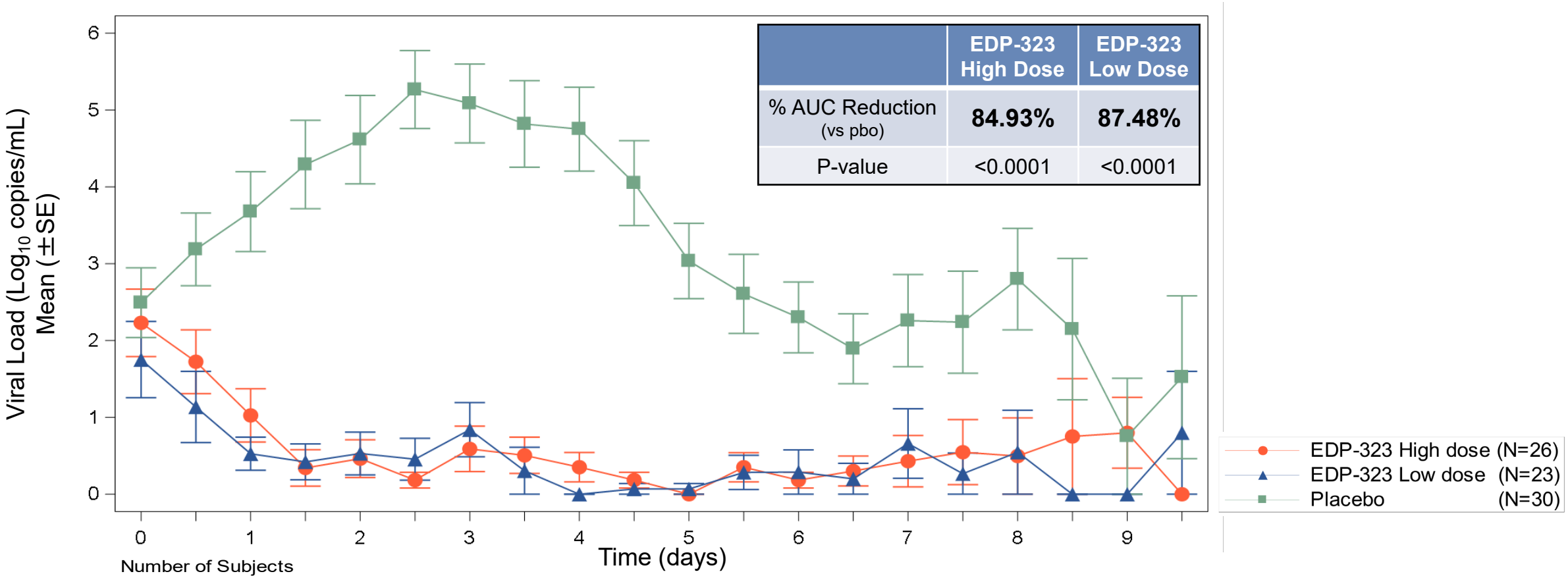
- EDP-323 mean trough plasma concentrations were maintained at 16- to 35-fold above the protein adjusted EC_{90} against both RSV A and B strains

*600mg loading dose (LD) on day one, followed by 200mg on days 2-5
AEs: adverse events; EC_{90}: a measure of potency; the concentration of drug that results in 90% inhibition of viral replication *in vitro*

EDP-323: Robust Antiviral Effect in Human Challenge Model

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

- Highly statistically significant reductions in VL 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)



	EDP-323 High Dose	EDP-323 Low Dose
% AUC Reduction (vs pbo)	**84.93%**	**87.48%**
P-value	<0.0001	<0.0001

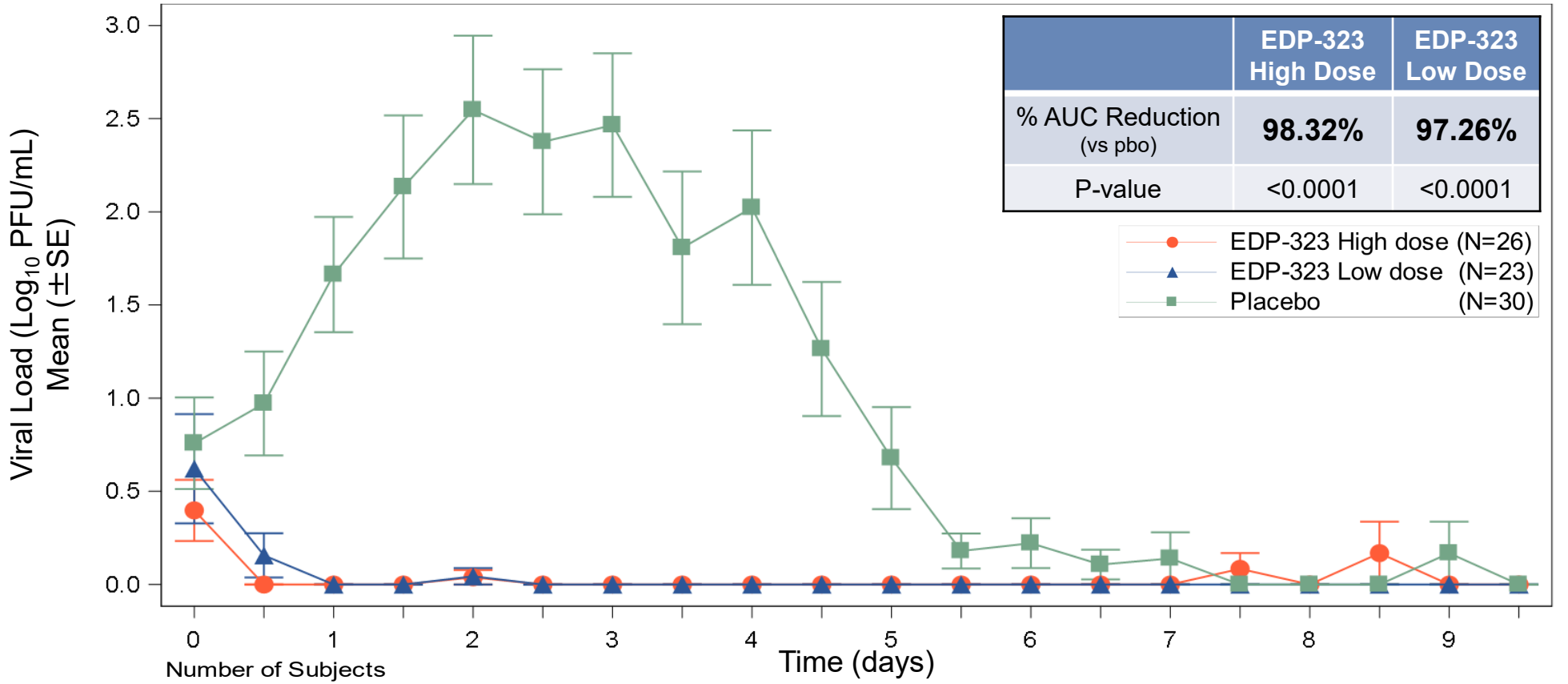
Number of Subjects																			
EDP-323 High dose	26	26	26	26	26	26	26	26	26	26	26	26	26	26	15	12	10	6	4	1
EDP-323 Low dose	23	23	23	23	23	23	23	23	23	23	23	23	23	23	17	16	10	5	4	2
Placebo	30	30	30	30	30	30	30	30	30	30	30	30	30	30	19	18	11	8	6	4

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)



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

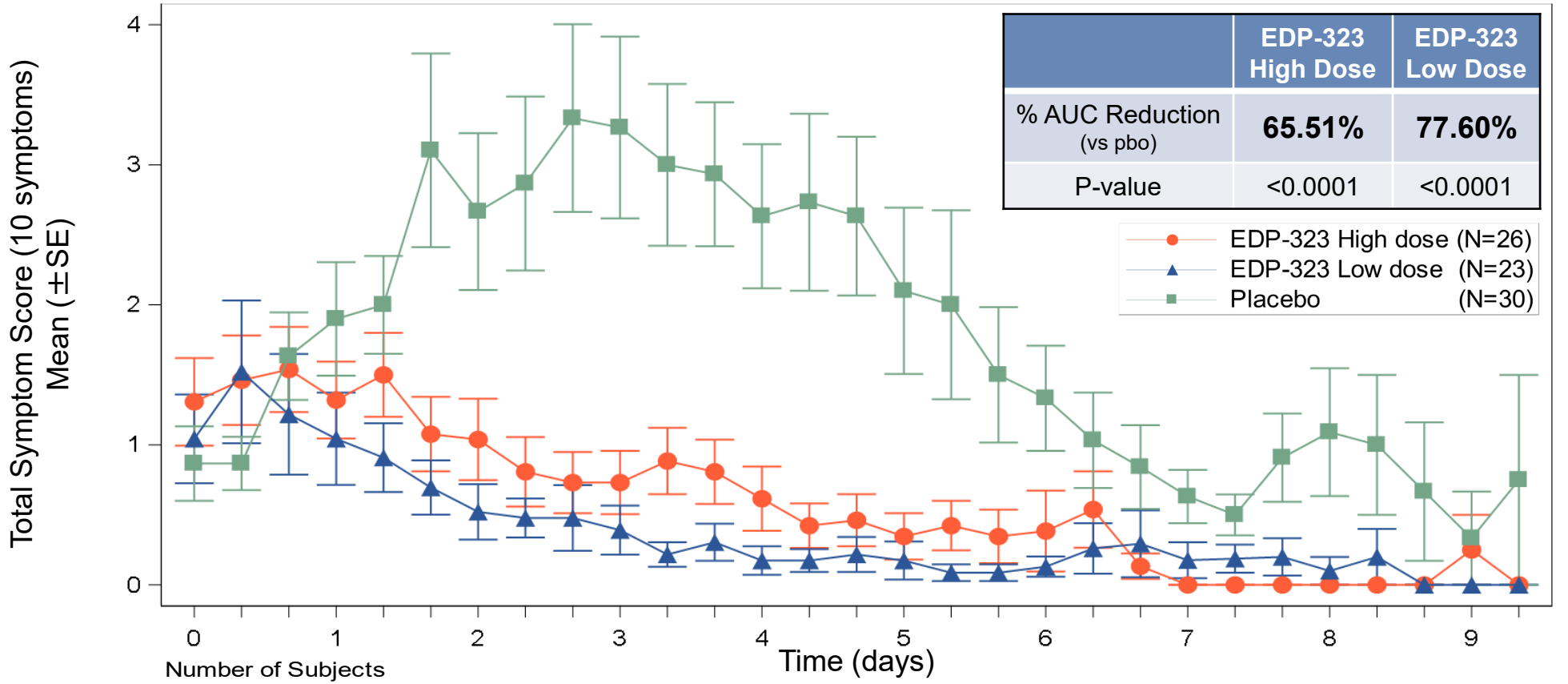
Number of Subjects																			
EDP-323 High dose	26	26	26	26	26	26	26	26	26	26	26	26	26	26	15	12	10	6	4	1
EDP-323 Low dose	23	23	23	23	23	23	23	23	23	23	23	23	23	23	17	16	10	5	4	2
Placebo	30	30	30	30	30	30	30	30	30	30	30	30	30	30	19	18	11	8	6	4

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

EDP-323: Robust Symptom Alleviation in Human Challenge Model

Secondary Efficacy Endpoint: 66-78% Reduction in Symptoms

- Highly statistically significant reductions in TSS AUC in both EDP-323 arms compared to placebo
  - No statistically significant difference between the two EDP-323 dosing regimens; 600mg and 200mg (with 600mg loading dose)



	EDP-323 High Dose	EDP-323 Low Dose
% AUC Reduction (vs pbo)	**65.51%**	**77.60%**
P-value	<0.0001	<0.0001

Number of Subjects

	0	1	2	3	4	5	6	7	8	9
EDP-323 High dose	26	26	26	25	26	26	26	26	26	26	26	26	26	26	26	26	26	26	26	15	15	12	10	10	6	4	4	1
EDP-323 Low dose	23	23	23	23	22	23	23	23	23	23	23	23	23	23	23	23	23	23	23	17	17	16	10	10	5	4	4	2
Placebo	30	30	30	30	30	29	30	30	30	30	30	30	30	30	30	30	30	30	30	19	19	18	11	11	8	6	6	4

TSS AUC: Area Under the Curve over time of Total Clinical Symptoms Score (10 items; hours x score); SE: standard error

Virology: SARS-CoV-2

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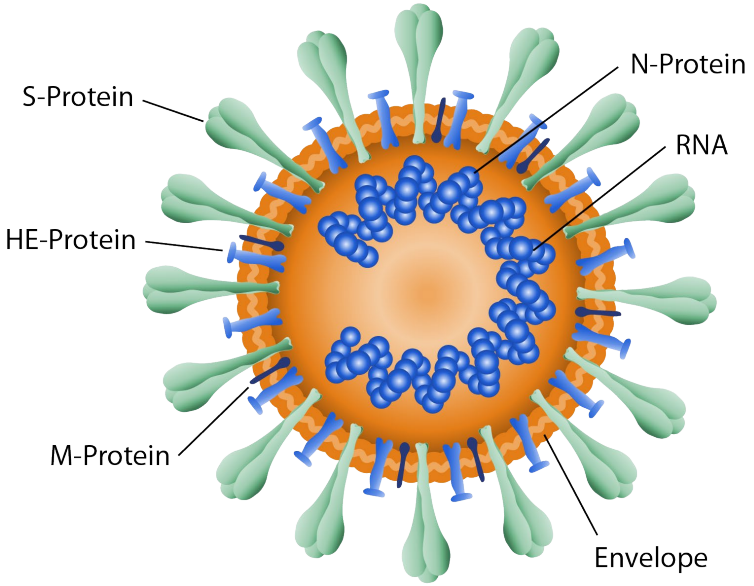
EDP-235: Oral Protease Inhibitor Designed for COVID-19

- Oral antiviral specifically designed to target SARS-CoV-2 3CL protease
- Granted Fast Track designation by the FDA
- Potent and selective inhibition of SARS-CoV-2 3CLpro enzyme
  - Potent inhibition in multiple cellular models
  - Potent against all SARS-CoV-2 variants of concern to date
- Preclinically active against other human coronaviruses
- High barrier to resistance preclinically
- Good target tissue distribution (e.g. lung to plasma AUC ratio >4)
  - Other COVID protease inhibitors lung to plasma AUC ratio <1
- Robust treatment effect and prevention of transmission in ferret model
- Phase 1 supported 200 or 400mg once-daily as safe and efficacious dose
  - Plasma drug levels 7-13x higher than the EC_{90}, without ritonavir boosting
- Phase 2 study (SPRINT) supports advancement to Phase 3



EC_{90}: a measure of potency; the concentration of drug that results in 90% inhibition of viral replication *in vitro*

Phase 2 (*SPRINT*) Results:
Study of EDP-235 in Non-hospitalized Standard Risk Patients

Safety
- EDP-235 was generally safe and well tolerated
- Low frequency of adverse events; most were mild in severity
  - 1.3%, 6.4%, and 2.6% in the EDP-235 200mg, 400mg and placebo arms
- No serious adverse events or discontinuations due to adverse events

Clinical Symptoms
- Statistically significant improvement in total symptom score (TSS) achieved at multiple timepoints for EDP-235 400mg
  - Patients enrolled within 3 days of symptom onset showed a statistically significant improvement in TSS for EDP-235 400mg at all time points
- No difference in time to 14 symptom improvement for EDP-235 compared with placebo
  - EDP-235 400mg significantly reduced duration of 6 symptom subset by 2-days compared to placebo in patients enrolled within 3 days of symptom onset (1-day for ITT-c population; 2-day for patients enrolled within 3 days of symptom onset)

Virology
- No difference between treatment arms and placebo for viral RNA decline
  - Additional analyses demonstrate virologic effect in multiple patient subsets at 400mg: 0.4 log for baseline viral load >5 log, 0.8 log for nucleocapsid negative (suggesting no recent natural infection), and 1 log for nucleocapsid negative and symptom onset within 3 days
- High degree of nucleocapsid positivity & rapid decline in nasal RNA in all study arms indicates a highly immune population

Immunology

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

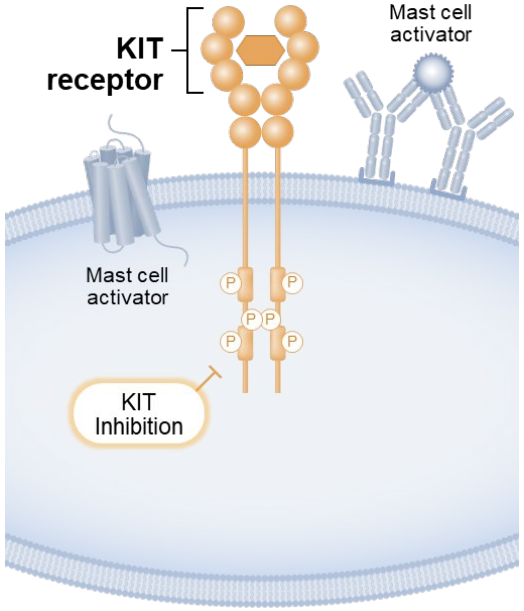
Exploit novel biology to select targets with **potential in multiple indications**

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:
Advancing Multiple Targets in Pipeline

KIT

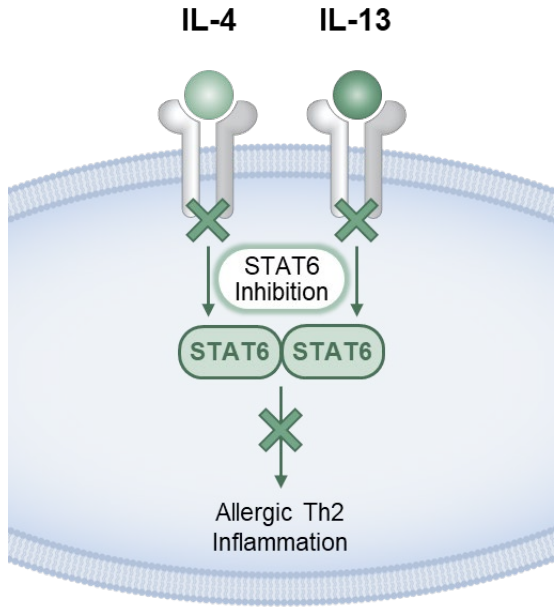


KIT inhibition blocks cell survival signal resulting in apoptosis and cell death

Potential Indications

Atopic Dermatitis
Asthma
CSU
CIndU
COPD
EoE
PN
CRSwNP
Others

STAT6



STAT6 inhibition impedes type 2 inflammation

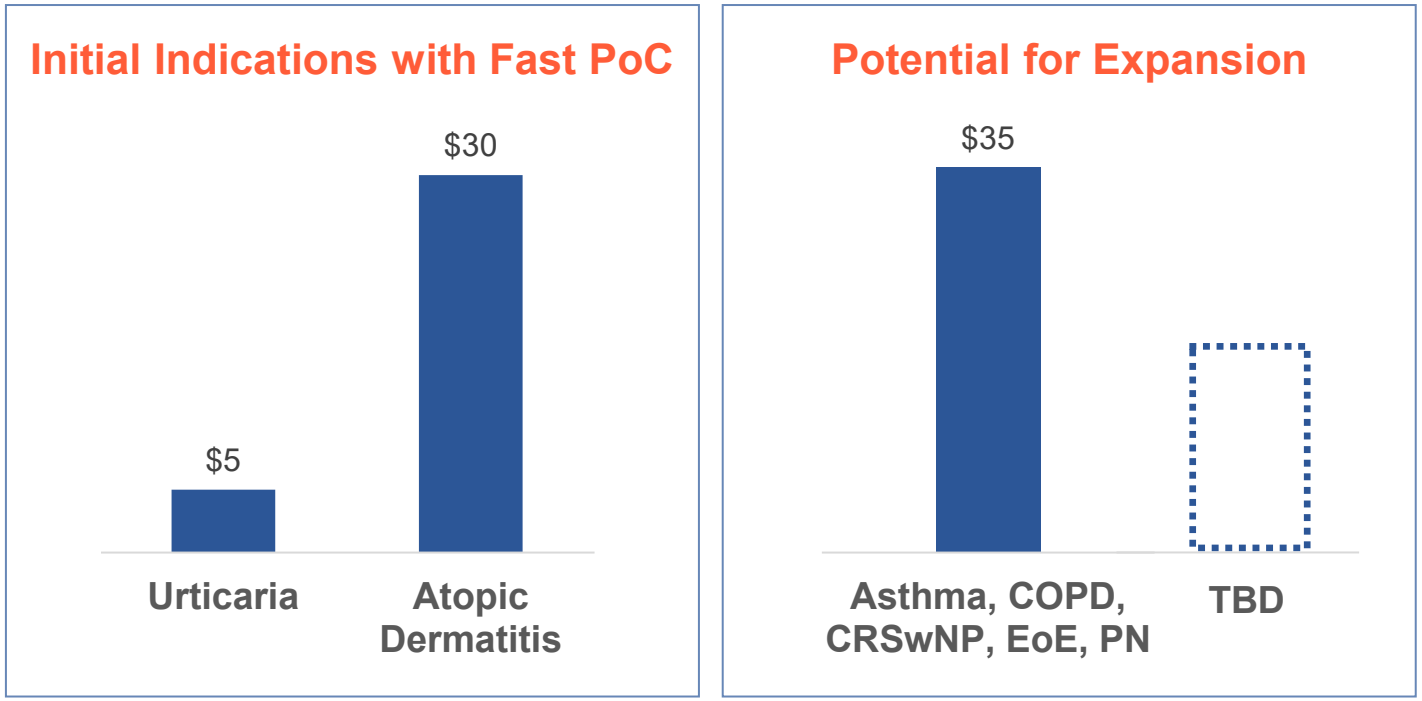
CSU = Chronic Spontaneous Urticaria. CIndU = Cold Inducible Urticaria. COPD = Chronic Obstructive Pulmonary Disease; CRSwNP = Chronic Rhinosinusitis with Nasal Polyps; EoE = Eosinophilic Esophagitis; PN = Prurigo Nodularis

Immunology Portfolio:
Multiple Targets Addressing Type 2 Immune Diseases

Enduring unmet need in multiple diseases driven by Type 2 immune phenotype

- 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

2030F Market Potential[1] ($B)



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Immunology: KIT Inhibitor

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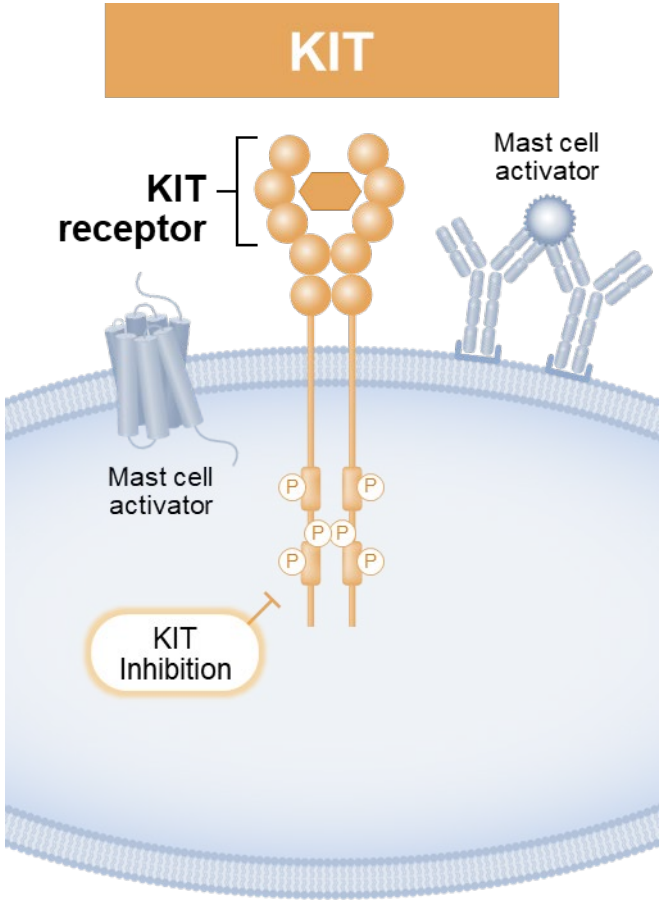
KIT Inhibitors Offer Potential for Differentiated Efficacy

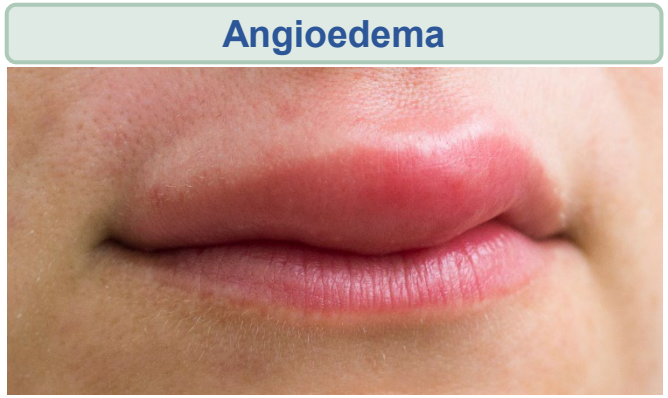
- **Mast cells:** primary driver of inflammation in skin, and implicated in multiple allergic diseases
  - Urticaria, asthma, eosinophilic esophagitis (EoE), and prurigo nodularis (PN)
- **KIT:** well-characterized receptor tyrosine kinase critical for regulating mast cell activity
- **KIT inhibitors:** 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



Chronic Spontaneous Urticaria

KIT Inhibitor Initial Focus for Clinical POC

- Severely debilitating, chronic inflammatory skin disease[1,2]
  - Driven by mast cell activation, triggering release of inflammatory mediators
  - Quality of life impacts beyond the skin: sleep disturbances, fatigue, irritability, anxiety and depression
- Affects ~0.5-1% of the global population[1]
- Substantial unmet need for efficacious oral agent
  - ~50% not controlled with antihistamines[1,3]
  - Minority treated with one indicated biologic (<30%)[4]



Sources: 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. 4. Clarivate Treatment Algorithms: Claims Data Analysis - Chronic spontaneous urticaria, May 2024.

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

- EPS-1421 selected as development candidate
  - Inhibits KIT with nanomolar potency in both binding and cellular assays
  - Highly selective for KIT versus other kinases
  - Good *in vitro* and *in vivo* ADME properties
    - Good PK profile across multiple preclinical species
    - No GSH adducts (or reactive metabolites) detected *in vitro* or *in vivo*
    - Low drug-drug interaction potential via CYP inhibition

EPS-1421 KIT Inhibitor Exhibits Potent Inhibition

Binding and Cell Based Assays

- Potency: Prevents KIT mediated cellular proliferation with EC_{50s} between 2-11 nM

Assay*	EC_{50} (nM)
KINOMEscan Kd	0.8
M-07e Cell Proliferation	11
UT-7 Cell Proliferation	3
KIT Ba/F3 Proliferation	2
$CD34^+$ Derived Mast Cell Degranulation	4
phosphoKIT	2

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

EPS-1421 KIT Inhibitor Demonstrates Good Selectivity

- Selectivity: >500x more selective for KIT over other KIT family members CSF1R, PDGFRα, & PDGFRβ in cell based assays*

Selectivity	EPS-1421
S(10) @ 1 μM	0.015
CSF1R	>500x
PDGFRα	>1300x
PDGFRβ	>1100x
FLT3 Kd	>1800x

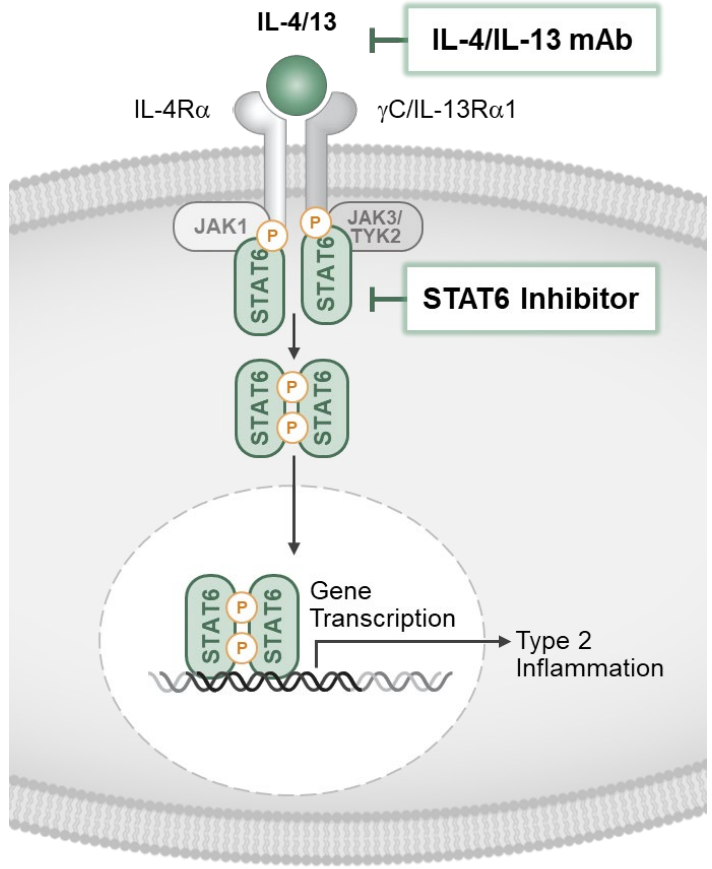
*Cellular selectivity calculated as ratio of EC_{50} in kinase dependent KIT Ba/F3 cells

Immunology: STAT6 Inhibitor

Enanta Pharmaceuticals

STAT6 Inhibitors Offer Potential for an “Oral DUPIXENT®”

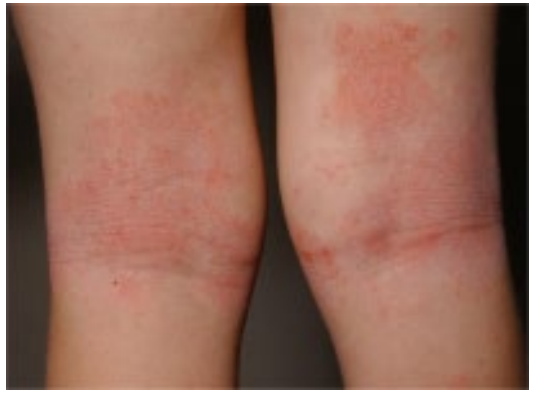
- **Th2 dysregulation** drives allergic and autoimmune diseases including atopic dermatitis and asthma
  - Characterized by IL-4 & IL-13 overproduction
- **STAT6**: transcription factor responsible for IL-4/IL-13 signaling, which drives a Th2 dominant phenotype
  - STAT6 gain-of-function variants result 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

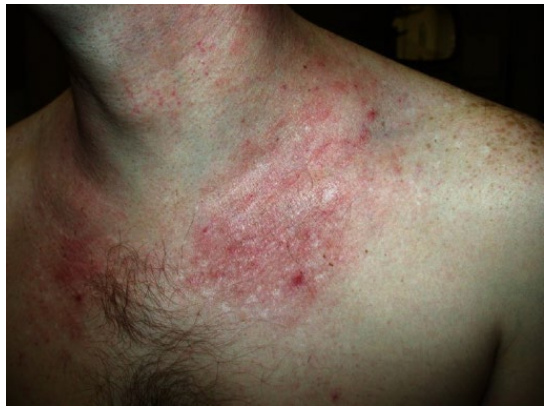


Sources: 1. Suratannon et al. *Journal of Allergy and Clinical Immunology* 2023. 2. Kristjansdottir et al. *Journal of Allergy and Clinical Immunology* 2024.

STAT6 Inhibitor Initial Focus for Clinical POC:
Atopic Dermatitis (AD)

- 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 US adults, ~40% have moderate-severe symptoms[2]
- Significant need for efficacious and safe oral agents
  - Market dominated by mAb (IL-4/13)
  - Oral JAK inhibitors used <10%[3], with boxed warning: serious infections, mortality, malignancy, major adverse cardiac events, thrombosis[4]





Sources: 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, 9/2024, www.evaluate.com 4.Prescribing Information for RINVOQ & CIBINQO

Goal of STAT6 Inhibitor Program:
Discover Novel Potent and Selective Oral STAT6 Inhibitors

- Novel, oral inhibitors of STAT6 being optimized in discovery stage
- Prototype STAT6 inhibitors:
  - Potently inhibit STAT6 activity in both biochemical and cellular assays
  - Highly selective over other STATs in biochemical and cellular assays
  - Demonstrates systemic *in vivo* target engagement after *ex vivo* IL-4 stimulation

2024 Key Catalysts

Virology

Respiratory Syncytial Virus

- ✓ Zelicapavir: Report Phase 2 RSVPEDs data in Q4 24
- ✓ EDP-323: Report Phase 2a challenge study data in Q3 24

Immunology

KIT Inhibition

- ✓ Select KIT inhibitor development candidate in Q4 24 (EPS-1421)

STAT6 Inhibition

- ✓ Initiate lead optimization (STAT6 inhibitor)

Business Development

SARS-CoV-2

- ☐ EDP-235: Pursue partnership for Phase 3 study

Hepatitis B Virus

- ☐ EDP-514: Identify third mechanism and/or out-license

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