



Enanta
Pharmaceuticals
Great Chemistry Cures

Corporate Presentation

May 6, 2024

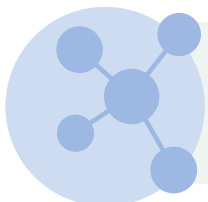


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A Proven Approach to Drug Discovery



Using a proven, chemistry-driven approach to develop best-in-class small molecule drugs for virology and immunology indications

Robust Pipeline

Virology:

- Phase 2 in pediatric patients with RSV (RSVPEDs)
- Phase 2 in high-risk adults with RSV (RSVHR)
- Phase 2 challenge study with second RSV candidate
- Phase 2 in COVID-19 complete (SPRINT)
- Phase 1b in HBV patients complete

Immunology: New program in Chronic Spontaneous Urticaria

Proven Track Record of Success

Glecaprevir: HCV protease inhibitor in MAVYRET®/MAVIRET®

Strong Balance Sheet

Strong balance sheet and royalties to support robust pipeline
\$300M in cash at March 31, 2024

Virology

Immunology

HCV

RSV

CSU

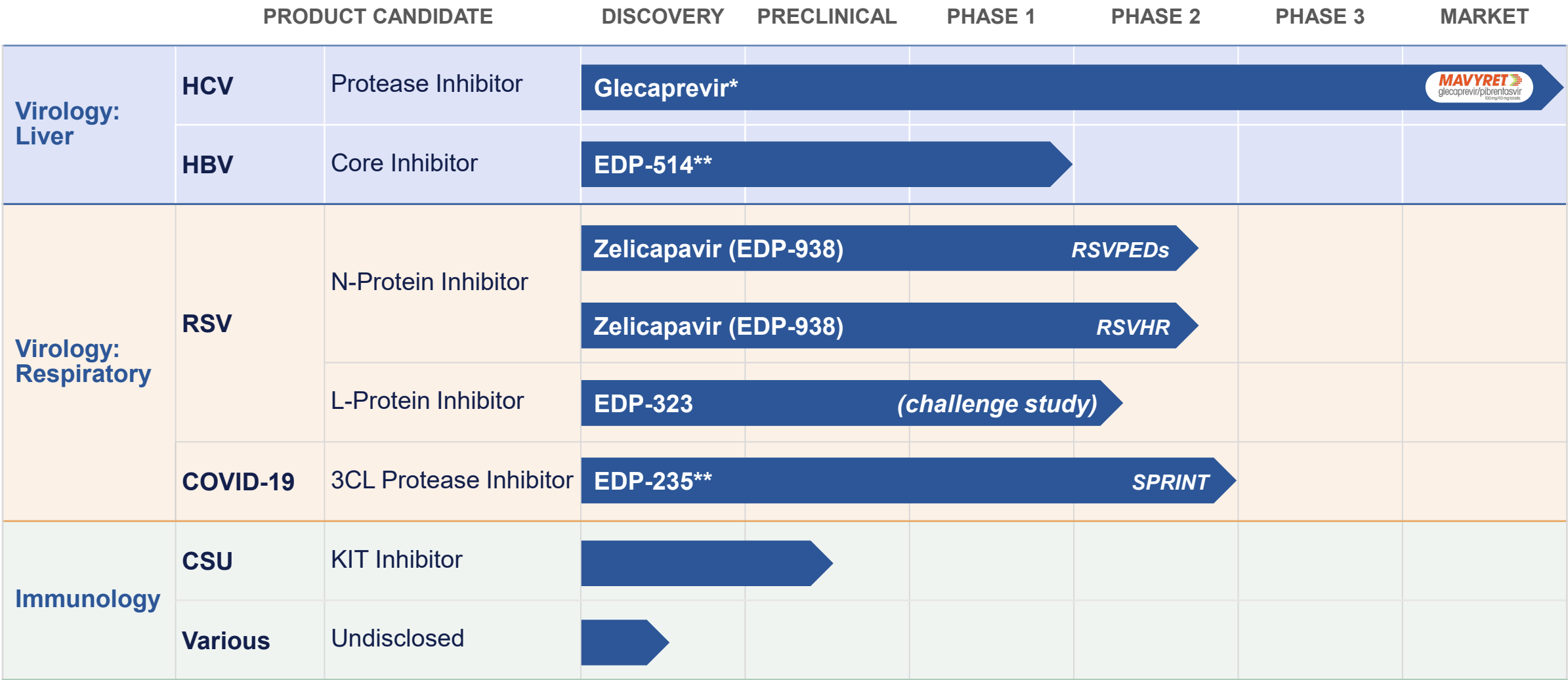
HBV

COVID-19

Our Therapeutic Focus

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

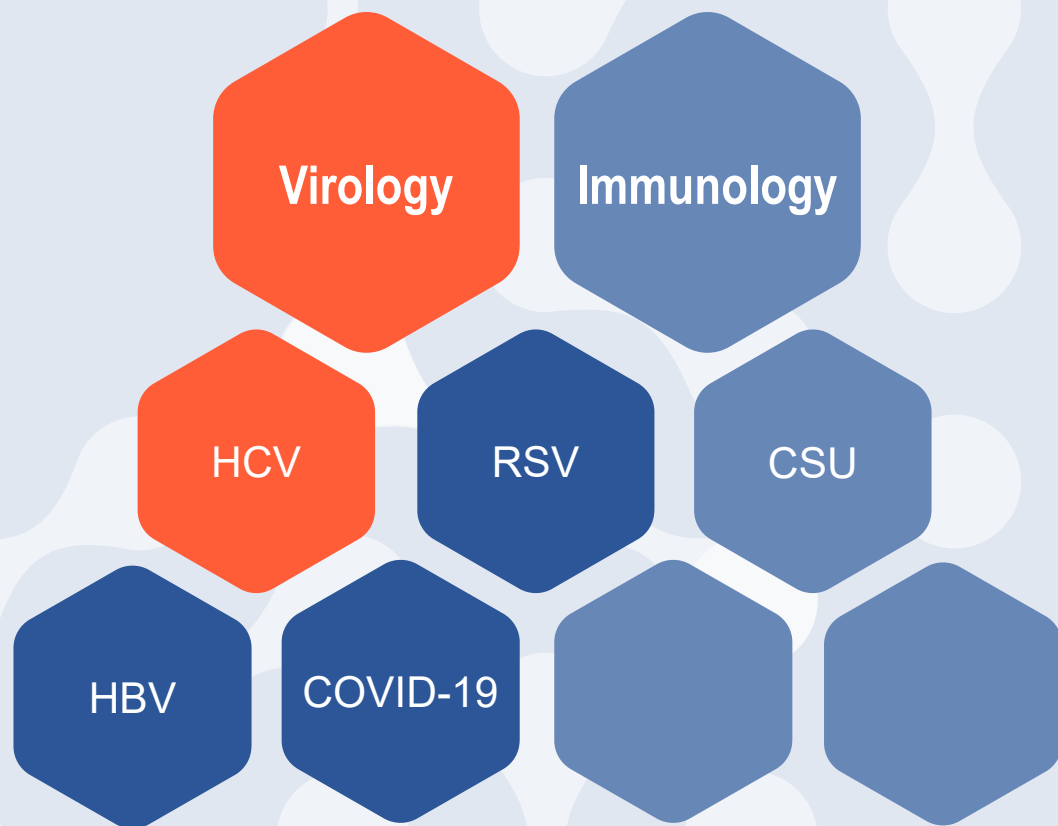
Enanta Pipeline



*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.

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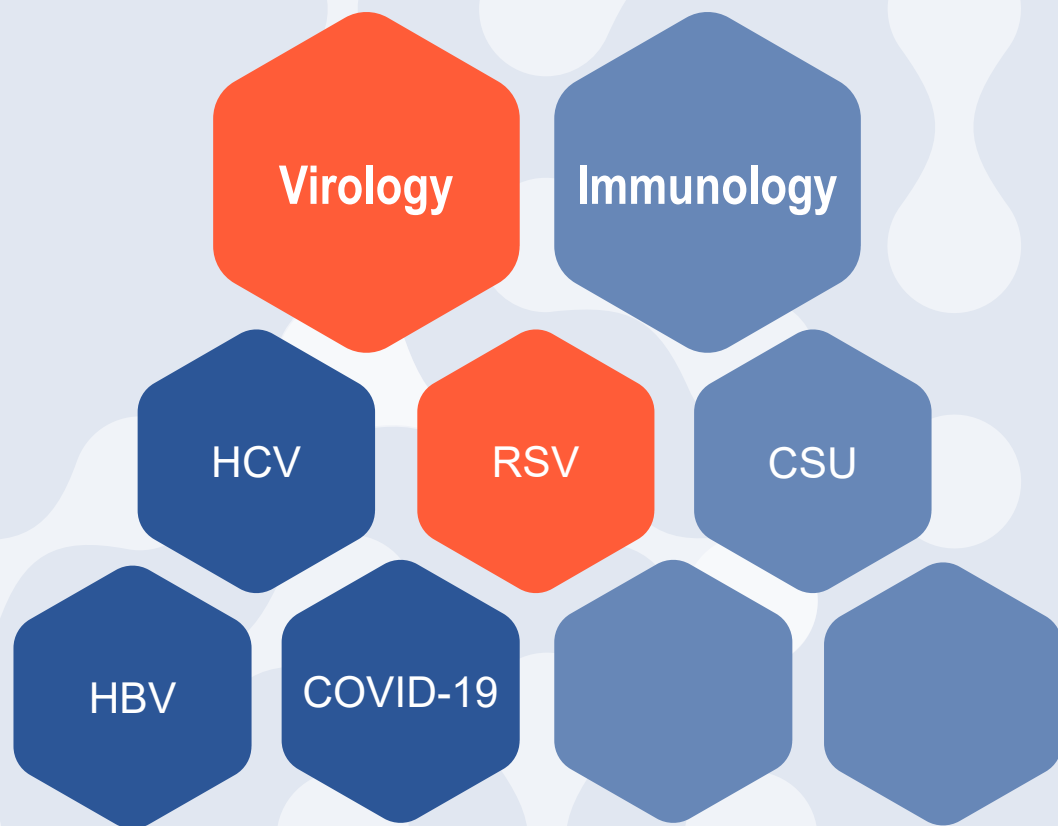
Glecaprevir: Our Licensed Protease Inhibitor for Hepatitis C Virus

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

- Glecaprevir (licensed to AbbVie) is key ingredient in MAVYRET®
- Fiscal 2024 GAAP Royalty Revenue (in millions)

Q1	Q2	Q3	Q4	Total
\$18.0	\$17.1	\$--	\$--	\$35.1
- Effective Q4 2023, Enanta sold 54.5% of future royalty cash payments for \$200M cash payment
- After royalty sale, Enanta will:
 - continue to record 100% of royalty revenue earned in its GAAP reporting*
 - receive only 45.5% of the royalty cash payments

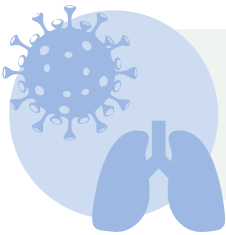
* Royalty sale is accounted for as debt because royalty buyer gets up to a total cap of 1.42 x the \$200M sale price



Our Therapeutic Focus

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

Respiratory Syncytial Virus (RSV)



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

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 ¹	Adults > 65 years ²
33M global cases	
3M global hospitalizations	177K U.S. hospitalizations
101K global deaths	14K U.S. deaths

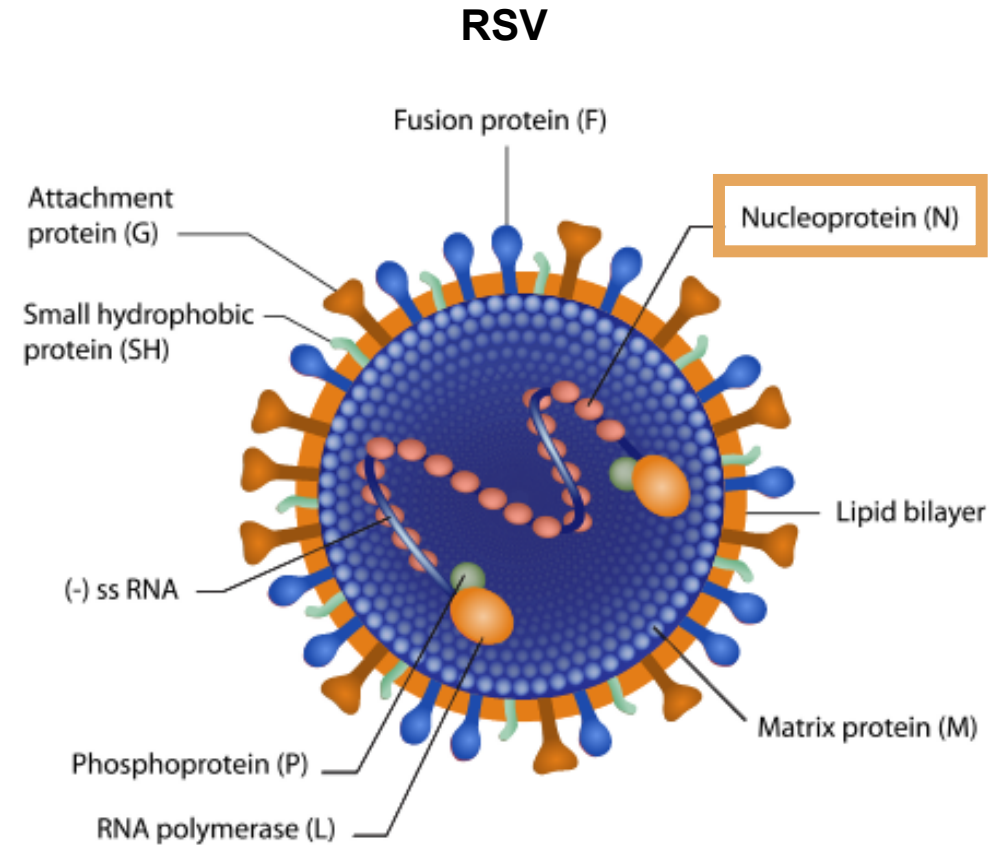
Significant unmet need for antiviral treatment despite availability of prophylaxis:

- Adoption of prophylactic approaches with shared clinical decision-making likely to be sub-optimal
 - Peak adoption of universally recommended vaccines for elderly range from ~35% (shingles³) to ~55% (flu⁴)
- 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

Sources: 1. [https://www.thelancet.com/journals/lancet/article/PIIS0140-6736\(22\)00478-0/fulltext](https://www.thelancet.com/journals/lancet/article/PIIS0140-6736(22)00478-0/fulltext) 2. <https://pubmed.ncbi.nlm.nih.gov/15858184/>
 3. <https://www.cdc.gov/nchs/products/databriefs/db370.htm>: 33.4% in 2016 & 34.5% in 2018 for Age 60+
 4. <https://www.cdc.gov/flu/fluview/dashboard/vaccination-coverage-adults-65-over.htm>: 54% 2021-2022; 58% 2020-2021; 56% 2019-2020 for age 65+

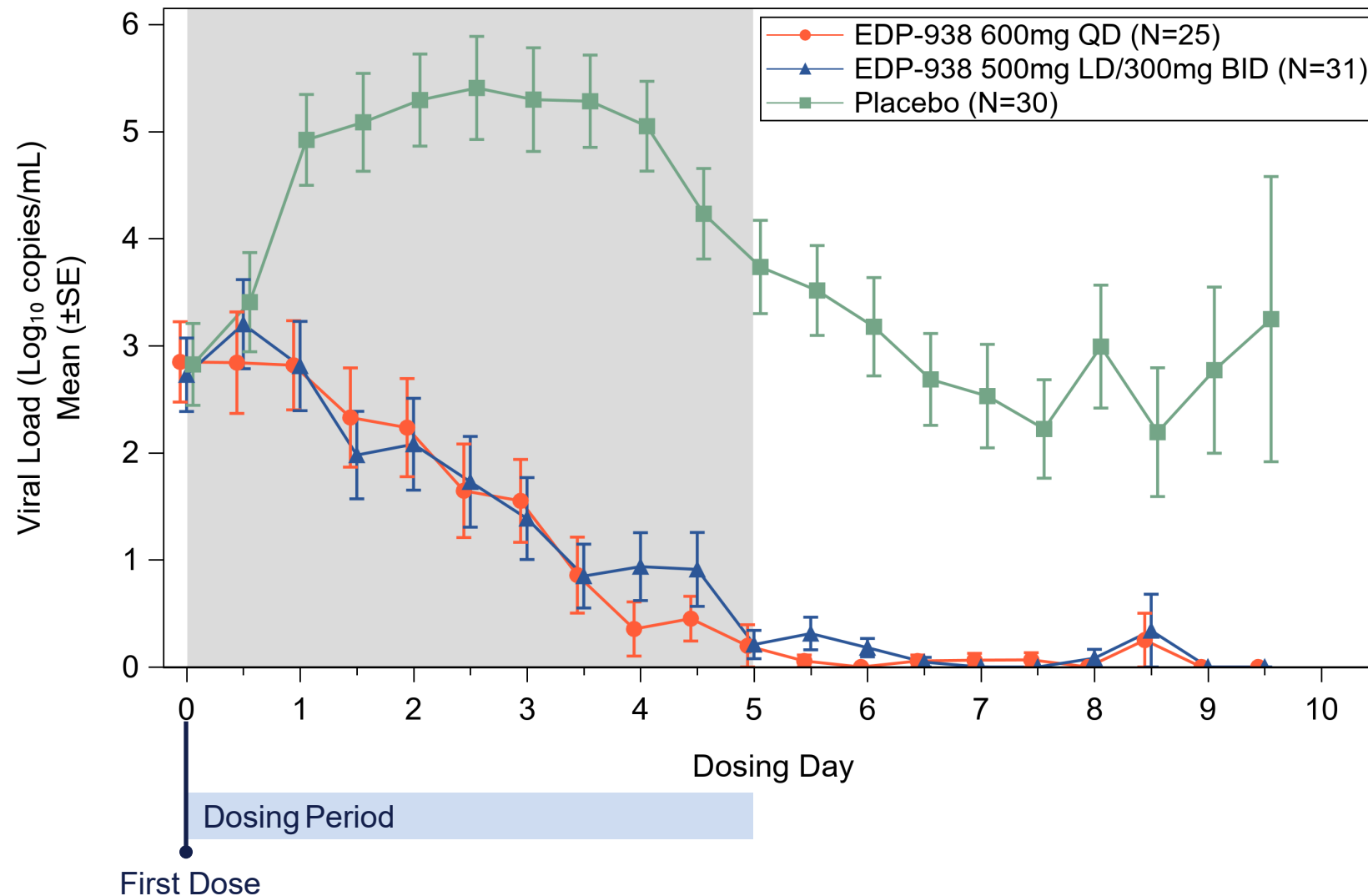
Zelicapavir (EDP-938): N-Protein Inhibitor for RSV

- Zelicapavir is the only N-inhibitor in clinical development
 - Directly targets viral replication vs 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
 - Synergy with other drug mechanisms (e.g. fusion and L-inhibitors)
 - No cross-resistance with other mechanisms



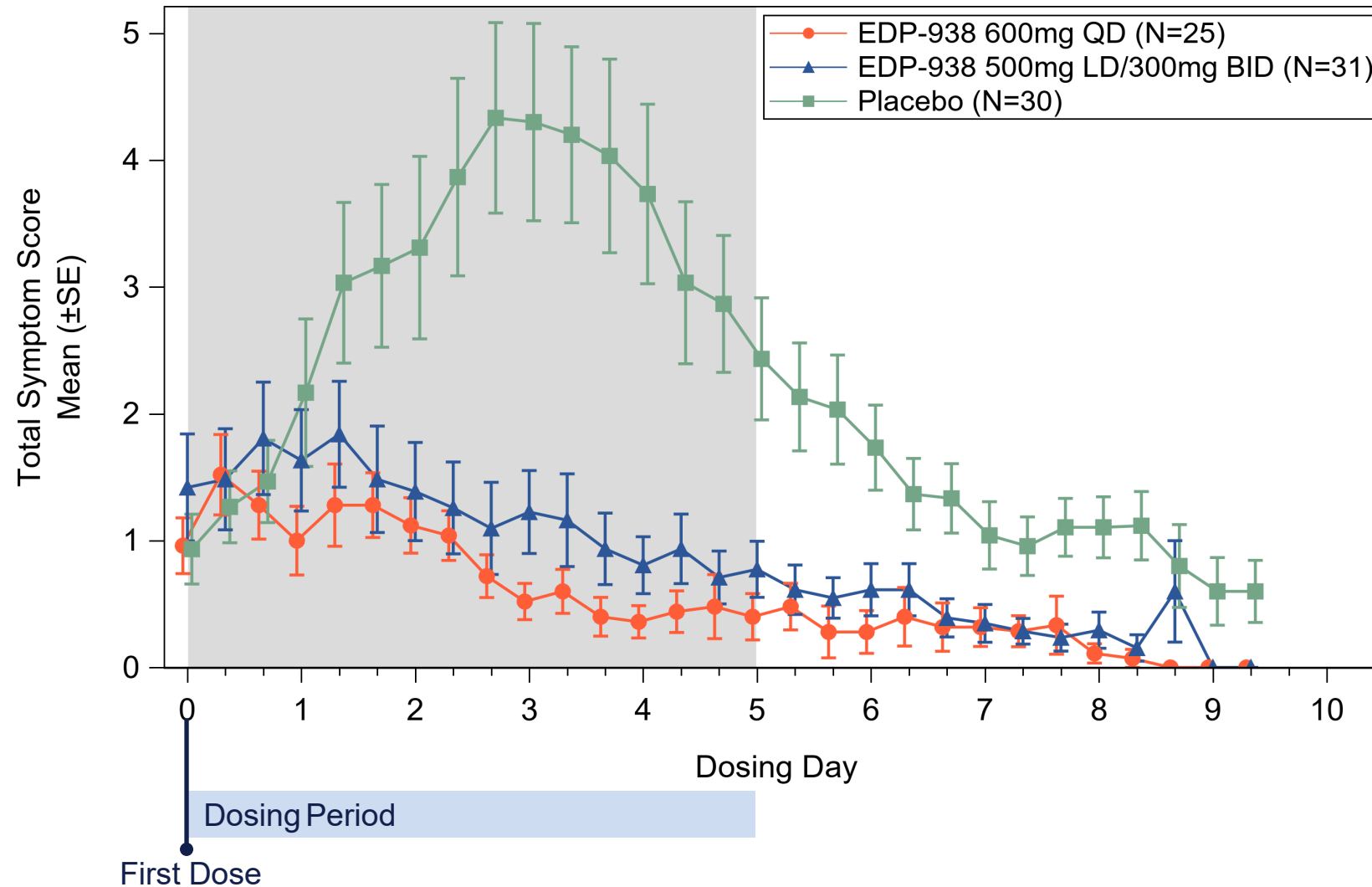
Zelicapavir: Robust Antiviral Effect in Human Challenge Model

Rapid and Sustained Reduction in Viral Load in Both Active Arms Compared to Placebo (71%, 74% ↓ AUC; $P < 0.001$)






Zelicapavir: Robust Symptom Reduction in Human Challenge Model

Rapid and Sustained Attenuation of RSV Symptoms in Both Active Arms Compared to Placebo (68%, 74% ↓ AUC; $P < 0.001$)



Zelicapavir: Potential to be the Leading Antiviral Treatment for RSV

				
Properties		Zelicapavir/ EDP-938	Ziresovir/ AK0529	Sisunatovir/ PF-07923568 ¹
Mechanism		N inhibitor	Fusion inhibitor	Fusion inhibitor
Pre-clinical Effectiveness After Infection		Yes	No	No
Clinical Efficacy (challenge study ⁴)	Viral Load Reduction ²	75% (p<0.001)	n/a	55% (p=0.007)
	Symptoms ³	71% (p<0.02)	n/a	71% (p=0.018)
Resistance Barrier		High	Low	Low
Dosing Frequency		5 days; QD	5 days; BID	5 days; BID
Stage of Development		Two Global Phase 2 (Peds, HR Adults)	Regulatory Review – China ⁵ (Peds)	Phase 2/3 (HR Adults)

Only includes compounds in development with clinical data in patients

Sources: 1. [DeVincenzo et al](#), 2020; AAC; 64(2); 2. % reduction in viral load (VL) area under the curve (AUC) as measured by qPCR; 3. % reduction in total symptom score (TSS) AUC; 4. Data from selected dose moving forward 5. Ark Bio [Press Release](#) December 2022

HR = high risk; HCT = Hematopoietic Cell Transplant Recipients. n/a = not available; challenge study not performed

Zelicapavir: Summary of Data Across Completed Clinical Studies

- Safety Summary
 - Generally safe and well-tolerated
 - Adverse events infrequent, generally mild, and resolved in follow-up
 - Consistent safety profile observed in approximately 500 subjects exposed to date
- Efficacy Summary in Healthy Adults
 - Phase 2a challenge study: highly statistically significant ($p < 0.001$) reduction in both in RSV viral load and clinical symptoms compared to placebo
 - RSVP study in otherwise healthy adults with community-acquired, mild, self-resolving RSV
 - Did not meet primary endpoint of clinical symptom reduction or secondary antiviral endpoints
 - Statistically significant percent of subjects achieved undetectable RSV RNA at end of treatment
 - Although treated within 48 hours of symptom onset, viral load and symptoms had already peaked and were declining, indicating infection resolves quickly in this otherwise healthy population
- Program currently focused on patients at high risk for severe disease with RSV

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



Infants and young children



Elderly

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

Ongoing Phase 2 Clinical Trials: RSVPEDs and RSVHR



~90 patients
Dosed in 2 age cohorts:
>28 days to <6 months
>6 months to <36 months

Double-Blinded Treatment Phase

5 days

Part 1: MAD vs Placebo

Part 2: Selected dose from Part 1 vs Placebo

23 Day Follow-up

Day 1

Day 5

Day 28

- **Primary Objective, Part 1:**
Safety and PK of zelicapavir
- **Primary Objective, Part 2:**
Antiviral activity of zelicapavir



~180 adults with at least one of the following:

- COPD
- Congestive heart failure
- Asthma*
- Age ≥ 65*

Double-Blinded Treatment Phase

5 days

Placebo (QD)

800mg (QD)**

28 Day Follow-up

Day 1

Day 5

Day 33

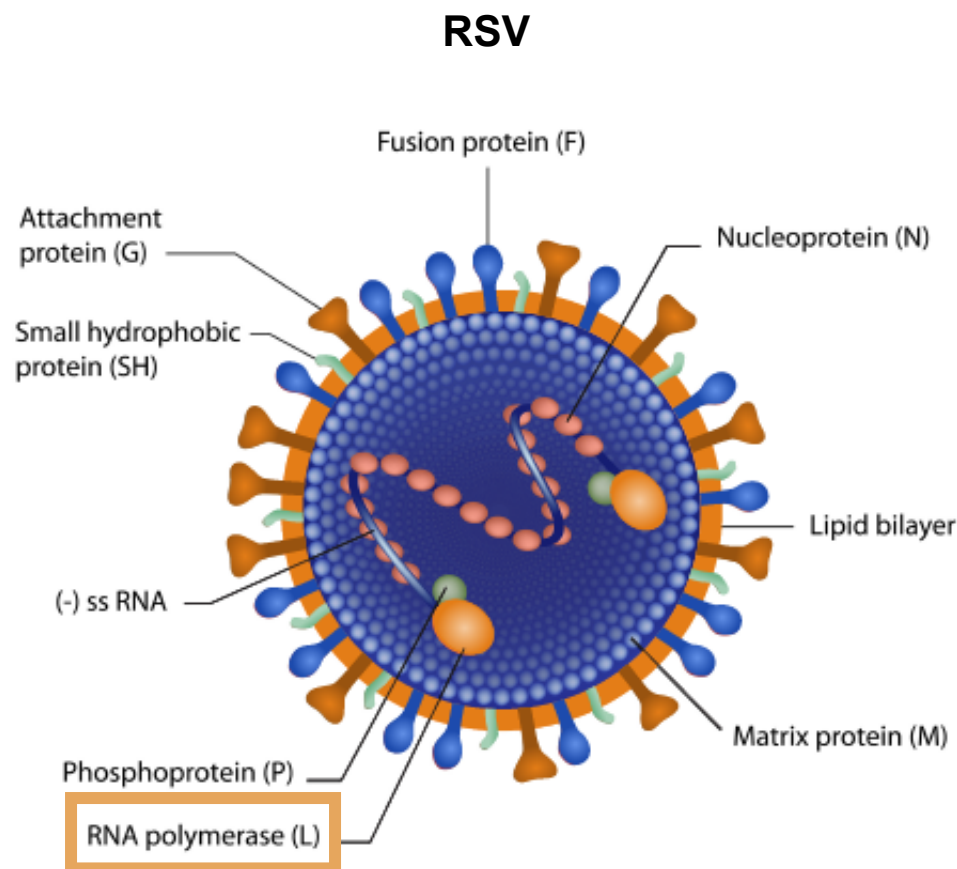
- **Primary Objective:**
Time to resolution of RSV LRTD symptoms as assessed by RiiQ symptom scale through Day 33
- **Secondary Objectives:**
PROs, MAVs, viral load, antibiotic use, bronchodilator use, corticosteroid use, hospitalization, ICU, mechanical ventilation, all cause mortality, PK and safety

*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

EDP-323: RSV L-Protein Inhibitor

- Selective direct-acting antiviral targeting the RSV L-protein
- Granted Fast Track designation by the FDA
- Potential to be used alone or in combination with other mechanisms, such as zelicapavir
 - Additive to synergistic activity with F-, N-, L-inhibitors and ribavirin
 - Not expected to have cross resistance with other mechanisms
- Sub-nanomolar potency against both RSV-A and RSV-B



EDP-323: Potent Inhibitor of RSV Replication *In Vitro* With Excellent Preclinical Pharmacokinetics

- Sub-nanomolar inhibition of RSV replication in a range of cell types
 - Active against both major RSV subtypes

Virus	Cell Type	EC ₉₀ (nM)
RSV-A Long	pHAEC ALI	0.27
RSV-B VR-955	pHAEC ALI	0.33
RSV-A Long	HBEC	0.16

RT-qPCR readout

pHAEC ALI: primary human airway epithelial cells in air-liquid interface culture

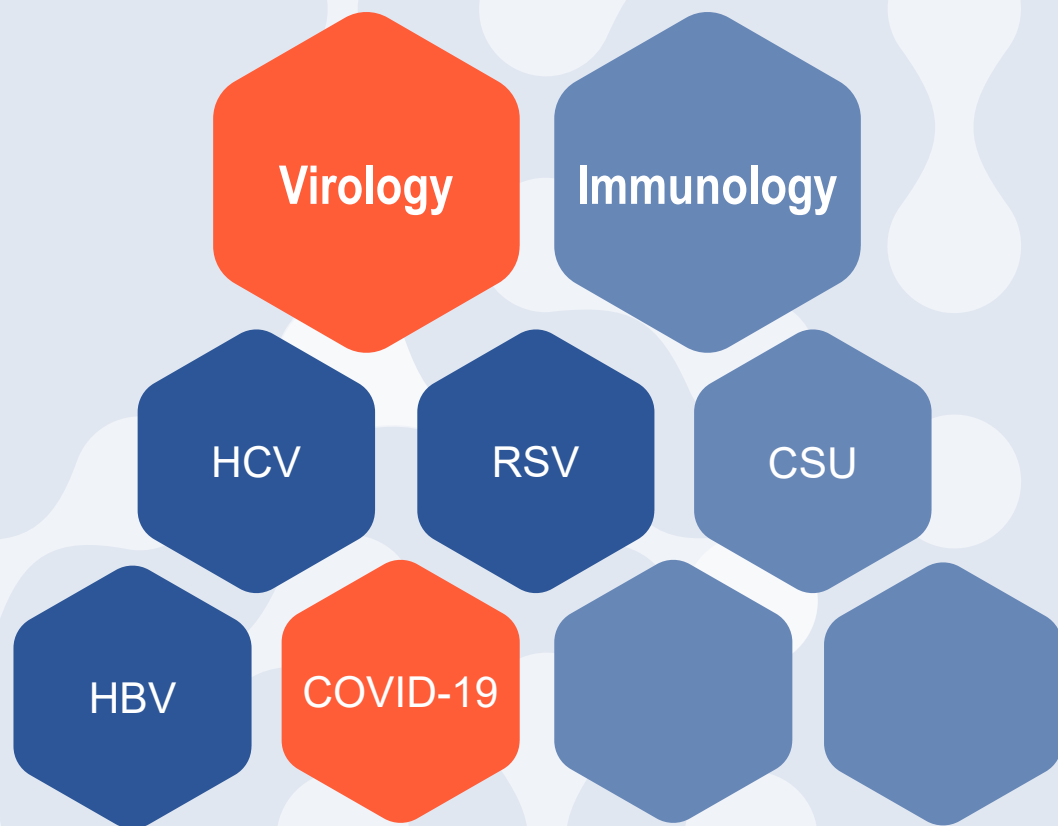
HBEC: human bronchial epithelial cells

50% cytotoxic concentration (CC50) at 5 days = 17,000 – 29,000 nM

- Protects mice in a dose-dependent manner from RSV infection as quantified by both virological and pathological endpoints
- Well-absorbed with good plasma exposure across multiple preclinical species
- High permeability/absorption potential in humans

EDP-323: Positive Phase 1 Results and Next Steps

- Randomized, double-blind, placebo-controlled Phase 1 study in healthy volunteers (n=82)
- Evaluated safety, tolerability and pharmacokinetics of oral single and multiple ascending doses
 - SAD (n=50): 50mg – 800mg and MAD (n=32): 200mg – 800mg once-daily
- Safe and well-tolerated up to 800mg for seven days
 - Most adverse events were mild; no serious or severe adverse events
- Exposure increased with increasing single and multiple dosing up to 600mg with half-life ranging from 11-17 hours, supporting once daily dosing
- All doses achieved target exposures, with strong multiples against both RSV A and B strains
 - Trough plasma concentrations were 11- to 44-fold over protein adjusted EC₉₀ (0.3 nM)
 - No food effect observed
- Phase 2a human challenge study initiated; expect to report data in Q3 2024

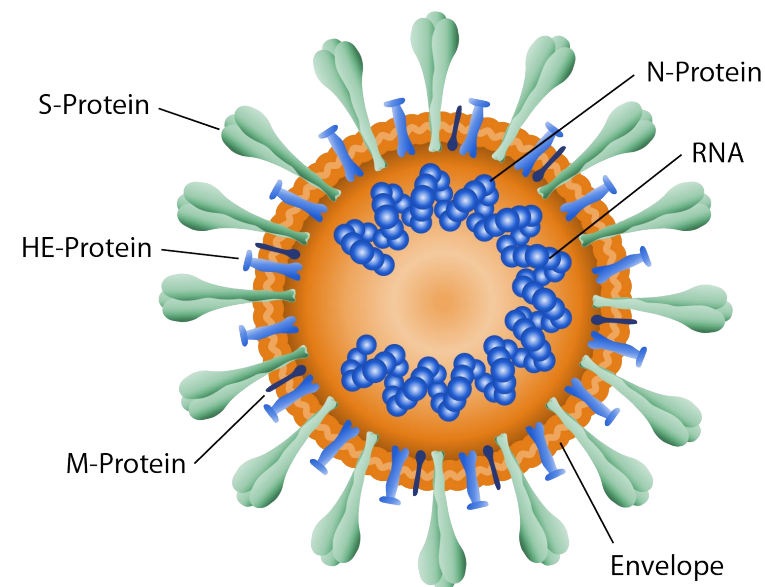


Our Therapeutic Focus

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

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 EC₉₀, without ritonavir boosting
- Phase 2 study (SPRINT) supports advancement to Phase 3



EDP-235 Preclinical Profile Suggests Potential for Best-in-Class Antiviral Treatment for SARS-CoV-2 Infection



Preclinical Properties		EDP-235 ¹	Nirmatrelvir ²	Ensitrelvir ³
Mechanism		Protease	Protease	Protease
Potency (nM) <small>All potency values versus ancestral (A) lineage unless indicated</small>	Enzyme IC ₅₀	5.8	19	13
	Vero Cell EC ₅₀	5.1	75	69 (Delta)
	Vero Cell EC ₉₀	11	155	n/a
Oral Bioavailability ⁴		95%	34 – 50%	97%
Lung Penetration ⁵		4.1	0.8 ⁶	0.7 ⁶
Projected Efficacious Dose		400mg QD	300 mg/100 mg ritonavir BID	375mg (D1)/125 mg (D2-5) QD

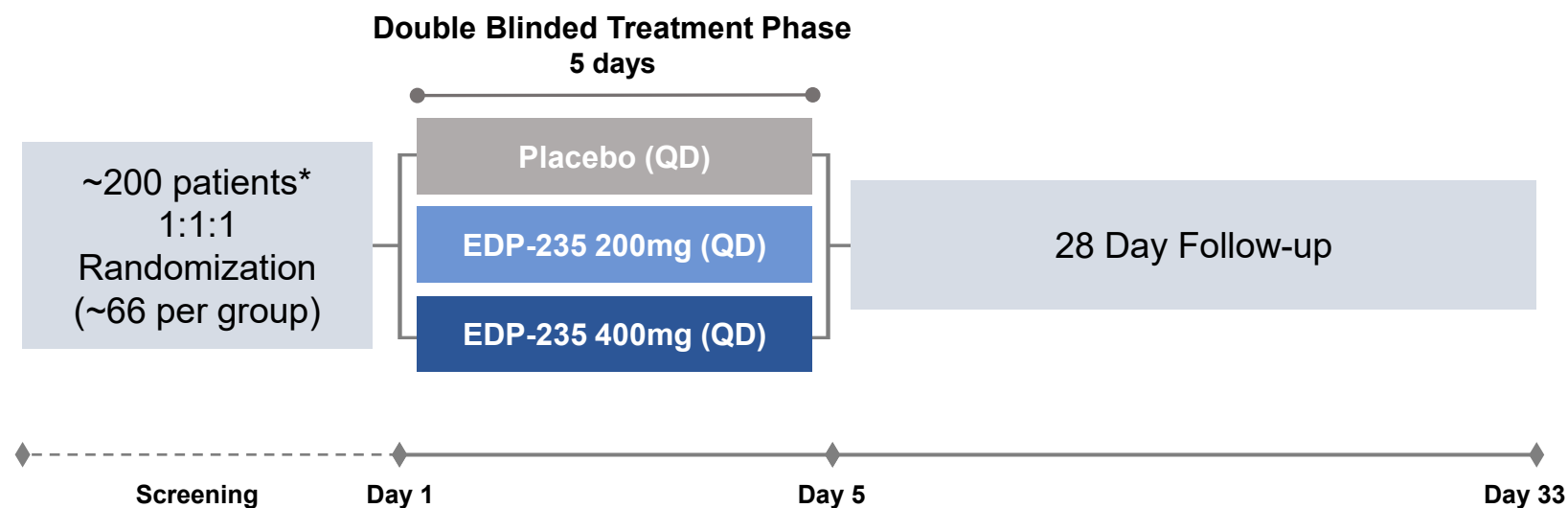
1. Jiang et al., ISIRV Poster #120, Oct 19, 2021
2. Owen et al., *Science* November 2021; Owen et al. ACS Spring 2021 meeting; EUA fact sheet for healthcare providers
3. Tachibana, et al., ISIRV oral presentation, Oct 20, 2021; Unoh, et al., bioRxiv 2022; Sasaki, et al., bioRxiv 2022; Yotsuyanagi, et al., ECCMID oral presentation, Apr 24, 2022
4. Oral bioavailability in rats for EDP-235, nirmatrelvir, and ensitrelvir; in mice for molnupiravir
5. AUC lung to plasma ratio in rats (EDP-235, nirmatrelvir, ensitrelvir), mice (molnupiravir); C12 lung to plasma ratio in humans for AT-527
6. Data for nirmatrelvir and ensitrelvir generated by Enanta

EC₉₀/EC₅₀: a measure of potency, the concentration of drug that results in 90%/50% inhibition of viral replication in cells
IC₅₀: a measure of potency, the concentration of drug that results in 50% inhibition of the protease enzyme

SPRINT: SARS-CoV-2 PRotease INhibitor Treatment

Phase 2 Study for EDP-235 in Non-hospitalized Standard Risk Patients

SPRINT



- **Primary Objective:**
Evaluation of safety and tolerability
- **Secondary Objectives:**
Evaluation of virologic endpoints, clinical symptoms and outcomes, and pharmacokinetics

Eligibility Criteria:

- Non-hospitalized adults who are not at increased risk for developing severe disease
- Initial onset of symptoms within 5 days of randomization
- At least 2 COVID-19 symptoms with one of at least moderate severity
- Have not been vaccinated or infected with SARS-CoV-2 within 90 days of enrollment

SPRINT: Safety and Symptom Summary

Safety

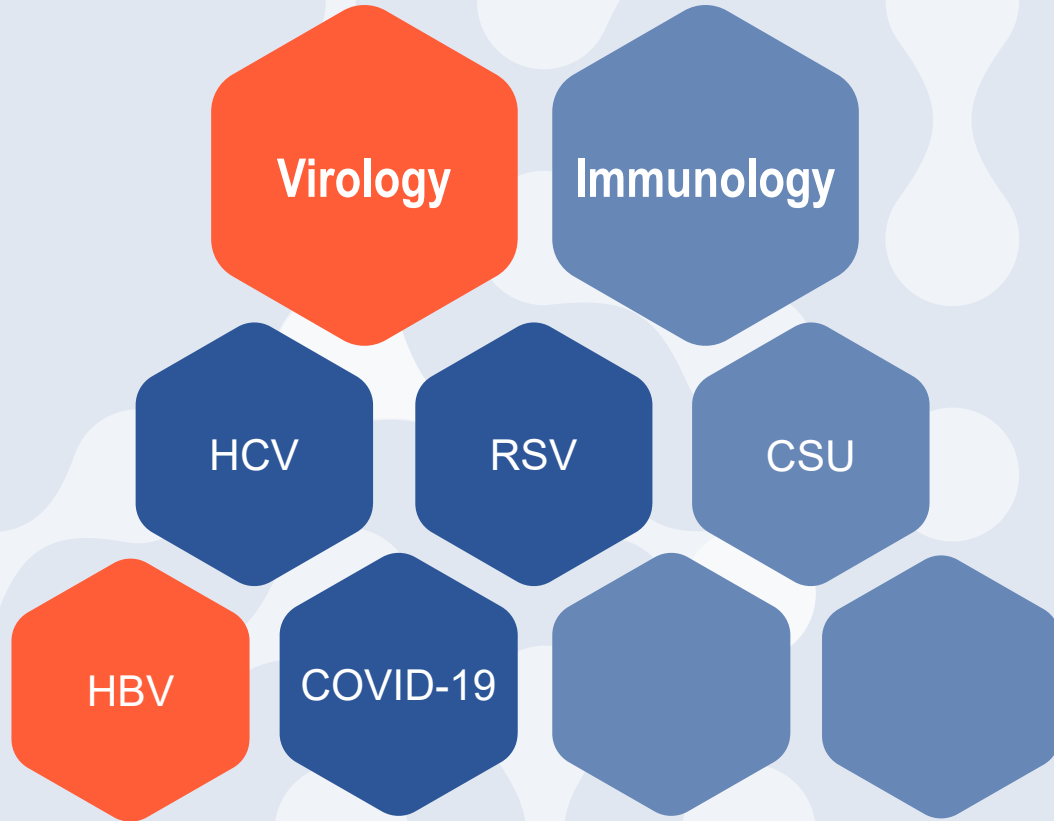
- EDP-235 was generally safe and well-tolerated
- Low frequency of adverse events; most were mild in severity
 - 1.3%, 6.4%, and 2.6% in the EDP-235 200mg, 400mg and placebo arms
- No serious adverse events or discontinuations due to adverse events

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 at all time points
 - Effect enhanced in prespecified population enrolled within 3 days of symptom onset in a subset of 6 symptoms
- No difference in time to 14 symptom improvement for EDP-235 compared with placebo
 - Statistically significant reduction in median time to symptom improvement with EDP-235 400mg vs placebo for subset of 6 symptoms
 - 1-day improvement in full ITT-c population
 - 2-day improvement in patients enrolled within 3 days of symptom onset
 - Median time to improvement was 3 days with EDP-235 vs 5 days with placebo

SPRINT: Virology Summary

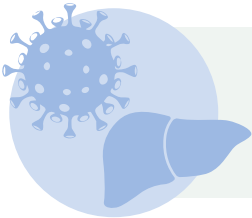
- No difference between treatment arms and placebo for viral RNA decline (ITTc)
- High degree of nucleocapsid seropositivity and rapid decline in viral RNA from nasal swabs in placebo arm indicate a highly immune population that quickly cleared virus from the nose
 - Observed symptom effects suggest that nasal sampling may not accurately reflect the impact of EDP-235 on virus in other tissues
- Additional analyses demonstrate a virologic effect in multiple subsets of patients, with a placebo-adjusted viral load decline at Day 5 in the 400mg group of:
 - 0.4 log: baseline viral load greater than 5 log
 - 0.8 log: nucleocapsid seronegative (suggesting no recent natural infection)
 - 1 log: nucleocapsid seronegative and symptom onset within 3 days



Our Therapeutic Focus

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

Hepatitis B Virus (HBV)



Potentially life-threatening liver infection

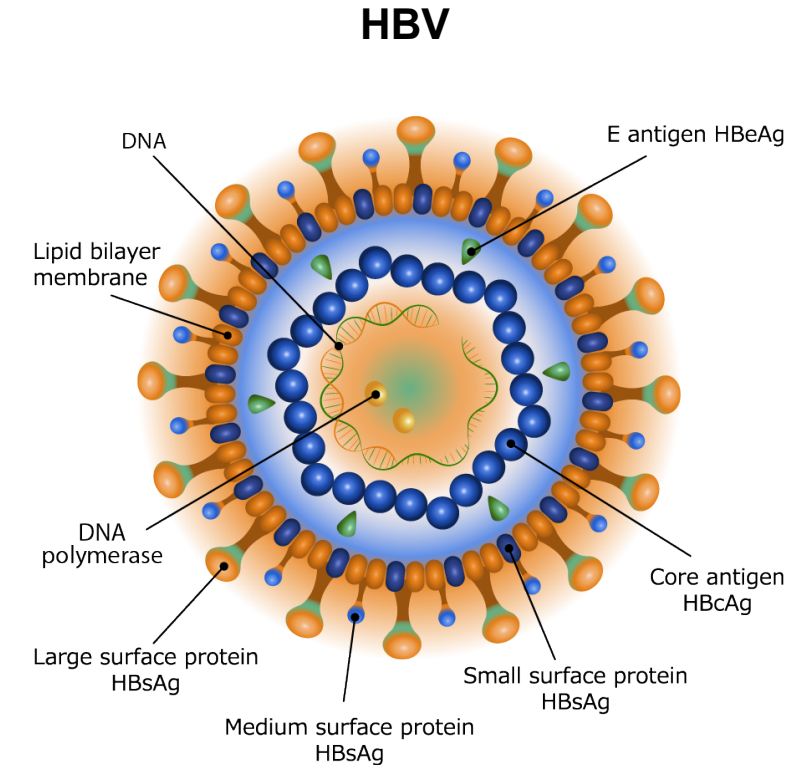
- In 2019, hepatitis B resulted in an estimated 820,000 deaths, mostly from cirrhosis and hepatocellular carcinoma (primary liver cancer)¹
- Current treatments rarely give true cures
 - **Interferon** is ~10% effective, but with side effects²
 - **Reverse-transcriptase inhibitors** effective at reducing viral load, but low cure rates (1% or lower) and treatment for life to improve cirrhosis or HCC outcomes³

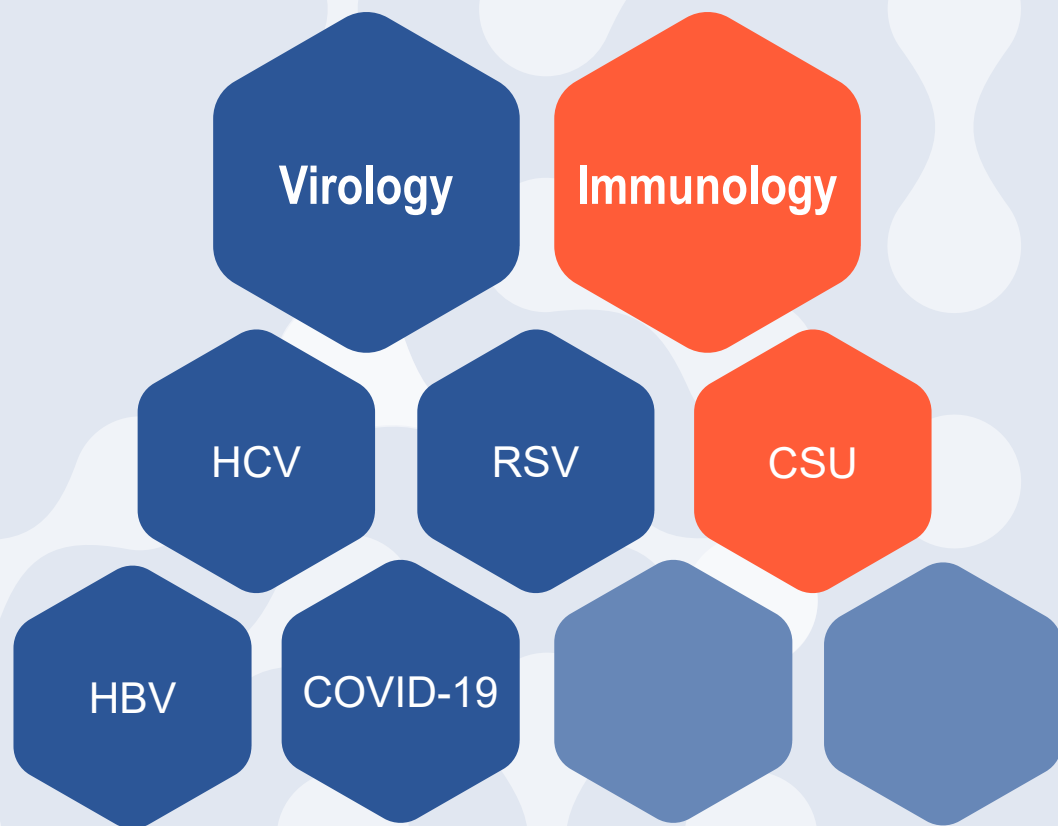
HBV at a Glance	
U.S.	850K – 2M people ⁴
Europe and European Economic Area	~4.7M people ⁵
Worldwide	~290M people ⁶

Sources: 1. <https://www.who.int/news-room/fact-sheets/detail/hepatitis-b> 2. <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5401664/>
3. <https://pubmed.ncbi.nlm.nih.gov/30342034/> 4. <https://jamanetwork.com/journals/jama/fullarticle/2738558>
5. <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5356432/> 6. <https://pubmed.ncbi.nlm.nih.gov/29599078/>

EDP-514: HBV Core Inhibitor

- Oral direct-acting antiviral targeting the HBV core protein
- Granted Fast Track designation by the FDA
- Strong preclinical virologic profile
 - Potent activity in HBV expressing stable cell lines
 - Capable of preventing the establishment of cccDNA
 - Pan-genotypic activity
 - Robust efficacy data in chimeric liver mouse model
- Phase 1 demonstrated good safety and tolerability with PK supportive of once-daily dosing with no food effect
- Two Phase 1b studies in HBV patients demonstrated significant reductions in HBV DNA and RNA after 28 days
 - NUC-suppressed patients: mean reduction in HBV RNA of up to ~1 log compared with 0.2 log in placebo
 - Viremic patients: Mean reductions in HBV DNA of ~3-3.5 logs across dose groups vs 0.2 log in placebo

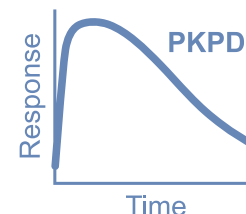
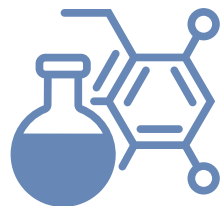




Our Therapeutic Focus

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

Leveraging Enanta's Core Strengths for Immune Driven Diseases



**Expand to
Immune-mediated
Chronic Diseases**

DISCOVERY

Understanding of
disease pathology

DEVELOPMENT

Known clinical path
and markers for
early clinical signal

MARKET OPPORTUNITY

Improved oral treatments
addressing significant
unmet need

**Foundational
Enanta
Capabilities**

STRONG DISCOVERY EXPERTISE

Underlying chemistry
is broadly applicable
across targets

VIROLOGY & IMMUNOLOGY

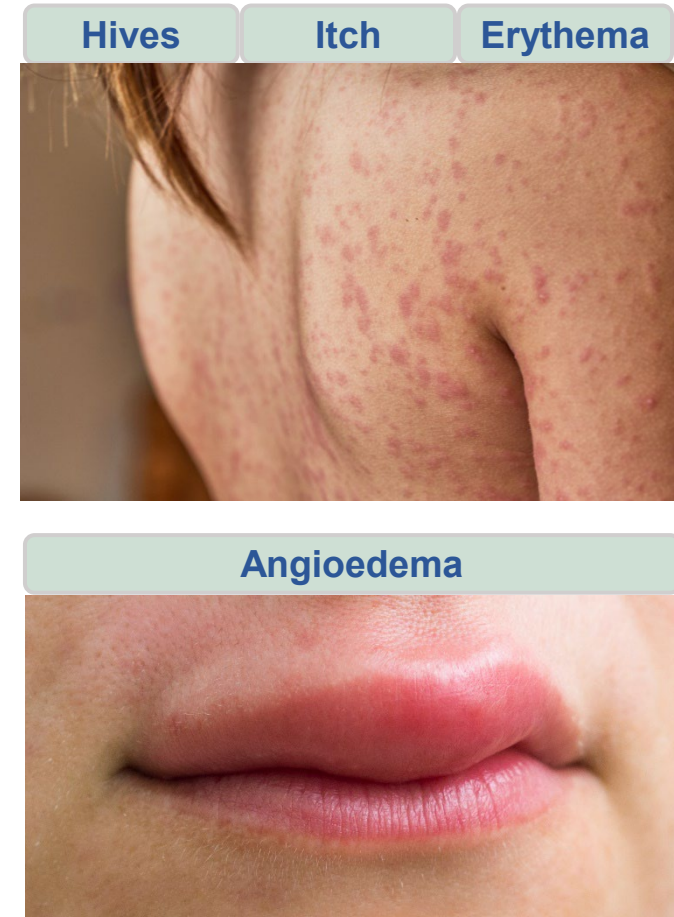
Scientifically
adjacent areas

PROVEN DRUG DISCOVERY PLATFORM

Differentiated small
molecules for established
and novel targets

Chronic Spontaneous Urticaria (CSU)

- **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 (<28%)⁴



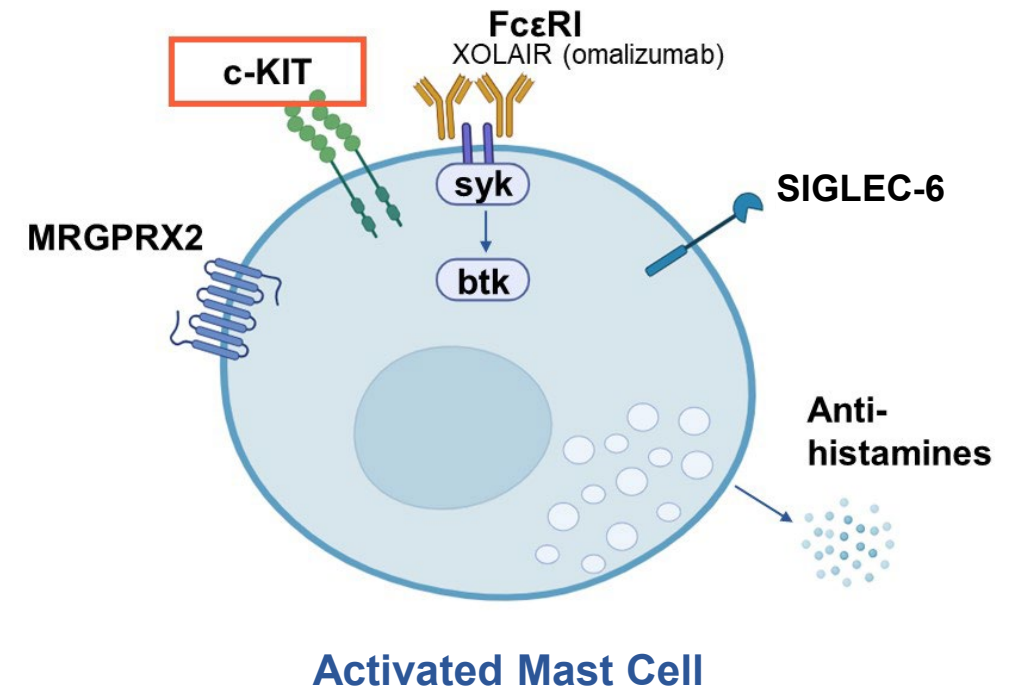
Sources: 1. Maurer M, et al. [Allergy](#). 2011; 66(3): 317–330. 2. Yosipovitch et al. [Dermatol Ther](#). 2023; 13(8): 1647–1660. 3. H₁ antihistamines up to 4x standard dose.

4. Clarivate Treatment Algorithms: Claims Data Analysis - Chronic spontaneous urticaria, February 2023. ©2023 DR/Decision Resources, LLC. All rights reserved. Reproduction, distribution, transmission or publication is prohibited. Reprinted with permission. Clarivate makes no representation or warranty as to the accuracy or completeness of the data ("Clarivate Materials") set forth herein and shall have, and accept, no liability of any kind, whether in contract, tort (including negligence) or otherwise, to any third party arising from or related to use of the Clarivate Materials by Enanta Pharmaceuticals Inc ("Enanta Pharmaceuticals Inc"). Any use which Enanta Pharmaceuticals Inc or a third party makes of the Clarivate Materials, or any reliance on it, or decisions to be made based on it, are the sole responsibilities of Client and such third party. In no way shall any data appearing in the Clarivate Materials amount to any form of prediction of future events or circumstances and no such reliance may be inferred or implied.

KIT Inhibitors: Potential for Best-in-Disease Efficacy

- **Mast cells:** primary driver of inflammation in skin, and implicated in multiple allergic diseases
 - CSU, chronic inducible urticaria (CIndU), asthma, eosinophilic esophagitis (EoE), 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 (eg; signal inhibition) or downstream mediators, but not mast cells themselves
 - Positive proof-of-concept in Phase 2 with anti-KIT mAb

Therapeutic Targets for CSU



KIT Inhibitor Discovery Program

- Novel, **oral**, potent and selective inhibitors of KIT being optimized in preclinical development
- Prototype KIT inhibitors:
 - Potently inhibit KIT activity in both binding and cellular function assays
 - Highly selective for KIT versus other kinases
 - Good *in vitro* and *in vivo* ADME properties
- Targeting selection of a development candidate in Q4 2024

Prototype KIT Inhibitor Demonstrates Potent Activity

- Prototype inhibitor exhibits potent inhibition of KIT in binding and cellular functional assays

	KINOMEScan Kd (nM)	Endogenous Cellular EC ₅₀ (nM)	Engineered Cellular EC ₅₀ (nM)
Prototype Inhibitor	0.3	17	3.4

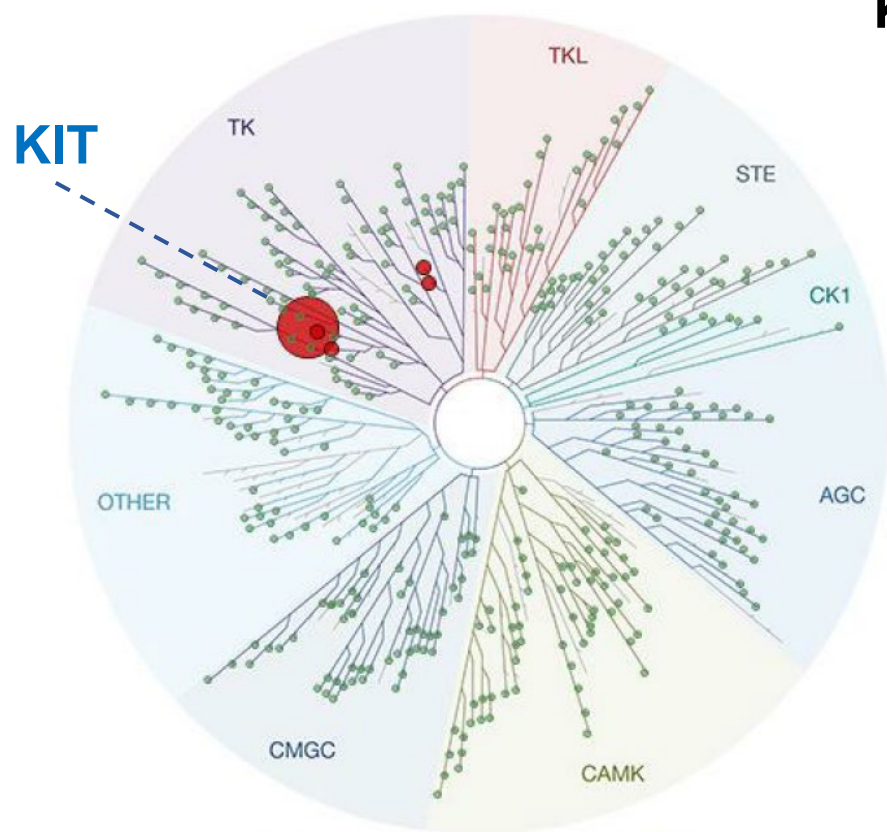
KINOMEScan: high throughput competitive binding assay that measures tagged kinase binding to an immobilized ligand

Endogenous Cellular: Growth inhibition assay using KIT expressing M-07e cells

Engineered Cellular: Growth inhibition assay using KIT expressing Ba/F3 cells

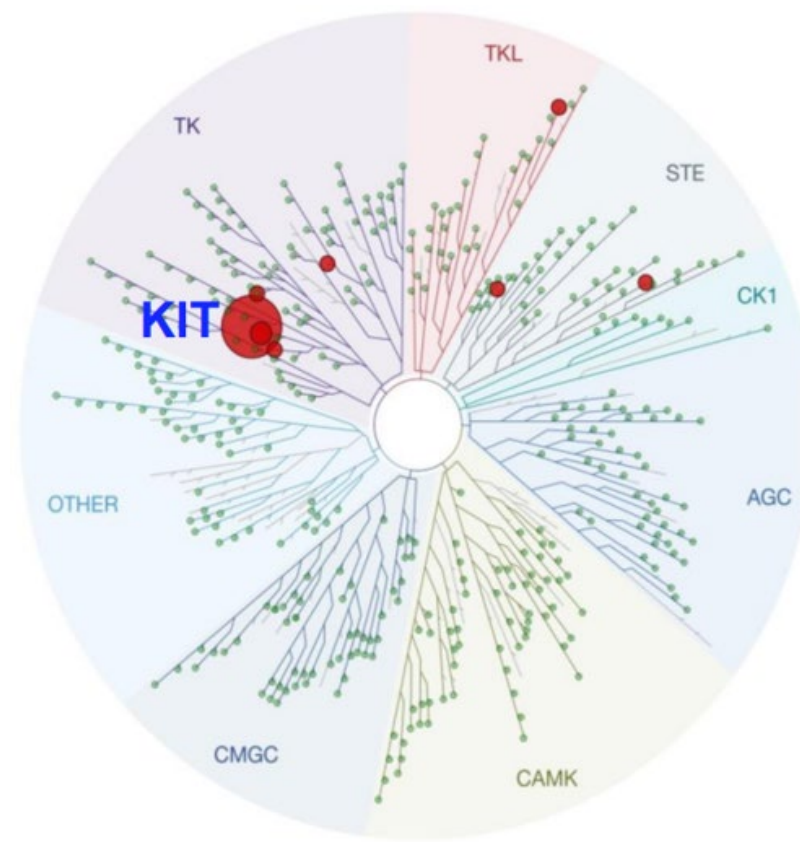
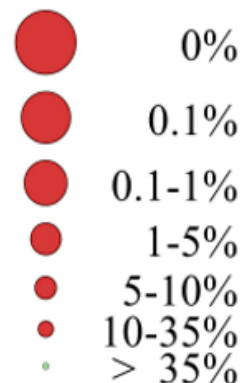
Prototype KIT Inhibitor Shows Greater Selectivity Compared to Other Inhibitors in Development

KINOMEscan at 100 nM



Prototype-001

Percent Control



THB3351¹

Prototype KIT Inhibitor has Favorable ADME Profile

- Good PK profile across multiple preclinical species (dog, mouse and rat)
 - Low potential for off-target penetration (e.g.; brain, testes and ovaries)
- Long half-life and no GSH adducts in human liver microsome incubation
- Low drug-drug interaction potential via CYP enzymes
- Excellent transporter selectivity

2024 Key Catalysts

Virology

Respiratory Syncytial Virus

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

Immunology

Chronic Spontaneous Urticaria

- ☐ Select KIT inhibitor development candidate in Q4 24

New Program

- ☐ Initiate lead optimization

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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