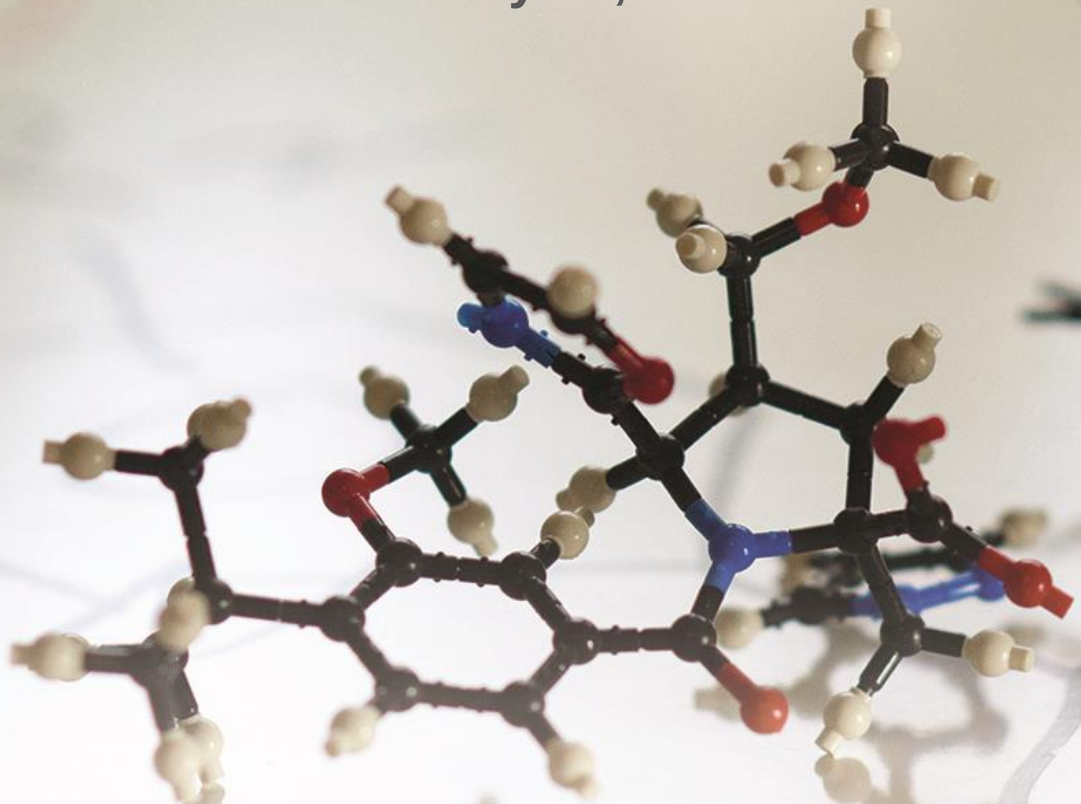
38th Annual J.P. Morgan Healthcare Conference

January 13, 2020



ENANTA Pharmaceuticals

Creating Small Molecule Drugs for Viral Infections and Liver Diseases

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 business prospects 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. All are forward-looking statements 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 development and involve known and unknown risks, uncertainties and other factors that are in some cases beyond our control. These risks and uncertainties include the following: (i) our financial prospects for the next several years are dependent upon the development and commercializing efforts of AbbVie for combination therapies for HCV incorporating paritaprevir, or glecaprevir, (ii) competition for these therapies, and (iii) the length, uncertainty and expense of discovering and developing new therapeutics for other diseases. As a result, any or all of our forward-looking statements in this presentation may turn out to be inaccurate.

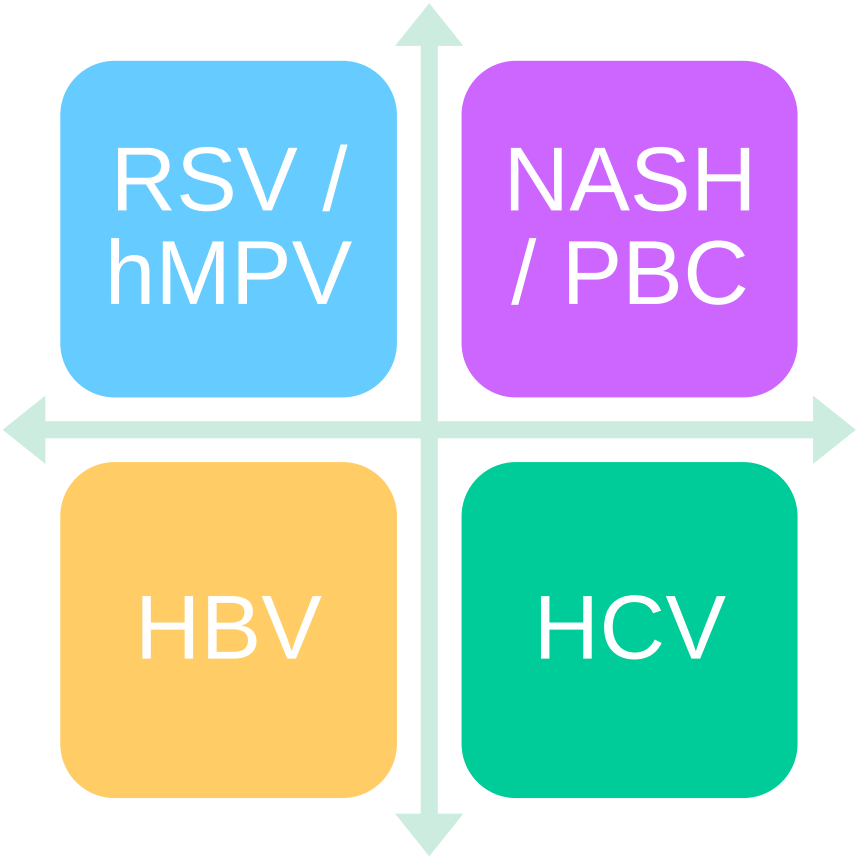
Please refer to these and other risk factors described or referred to in "Risk Factors" in Enanta's most recent Form 10-K, and other periodic reports filed with the Securities and Exchange Commission. Enanta cautions investors not to place undue reliance on the forward-looking statements contained in this presentation. These statements speak only as of the date of this presentation, and Enanta undertakes no obligation to update or revise these statements, except as may be required by law.

Investment Highlights

- Virology & liver disease-focused biotech company
- Four clinical-stage programs in areas of high unmet medical need:
  - RSV: Phase 2b "RSVP" study initiated
  - NASH: Phase 2a "ARGON-1" study completed
  - PBC: Phase 2 "INTREPID" study ongoing
  - HBV: Phase 1 study ongoing
- Discovery / preclinical programs in NASH, HBV, RSV, hMPV and other areas
- Partnered product marketed in AbbVie's HCV regimen:
  - Glecaprevir – HCV protease inhibitor in MAVYRET™/MAVIRET™
  - $205M in fiscal 2019 royalties on HCV regimens
- Strong balance sheet and royalties fund R&D programs
  - $400M in cash at fiscal year end 9/30/19

Our Therapeutic Focus

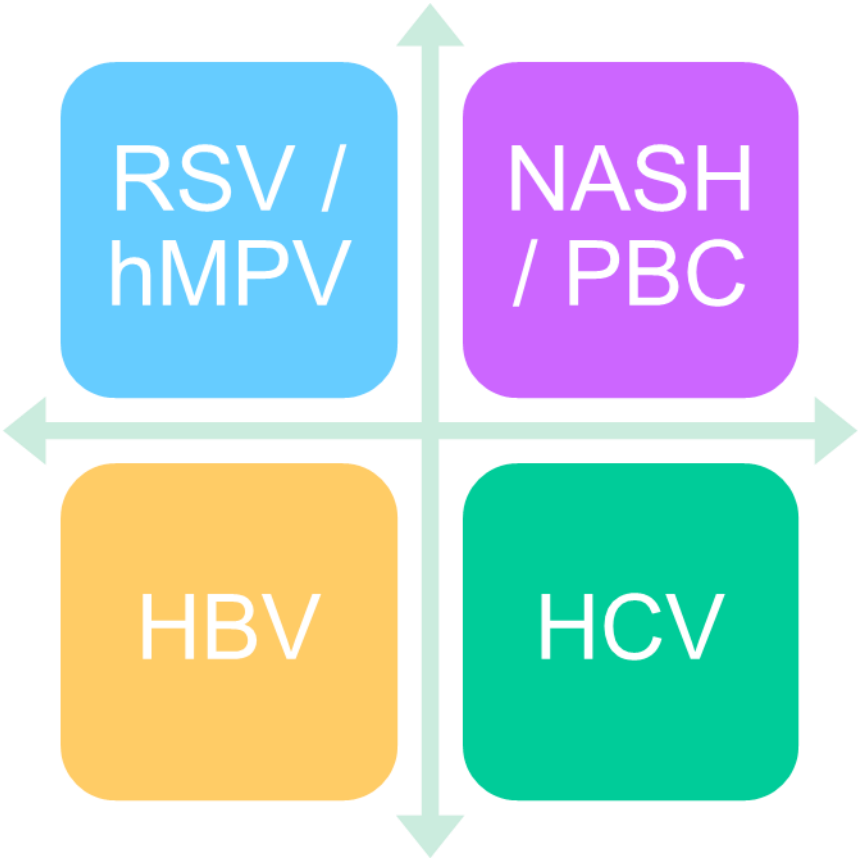
- Leverage our core strength in HCV to become a leader in Viral and Liver diseases
- Multiple new therapeutic areas with goal of building multiple approaches in each



Broad Virology and Liver Disease Pipeline

Product Candidate		Discovery	Preclinical	Phase 1	Phase 2	Phase 3	Market
HCV	Protease Inhibitor	glecaprevir – containing pan-genotypic 2-DAA combo					MAVYRET glecaprevir/pibrentasvir
RSV	N-protein Inhibitor	EDP-938		Ph 2b	RSVP		
NASH	FXR Agonist	EDP-305		Ph 2a	ARGON-1		
PBC	FXR Agonist	EDP-305		Ph 2	INTREPID		
HBV	Core Inhibitor	EDP-514		Ph 1			
NASH	FXR Agonist Follow-on	EDP-297					
hMPV	Inhibitor						
RSV, HBV, NASH, other Discovery or Preclinical							

Virology & Liver Disease Focus Areas



Respiratory Syncytial Virus (RSV)

- Negative-sense, single-stranded RNA virus of family Pneumoviridae
- Can cause severe lung infections, including bronchiolitis (infection of small airways in the lungs) and pneumonia (an infection of the lungs)
- Higher risk populations for severe illness include:
  - Premature babies
  - Older adults, especially those 65 years and older
  - People with chronic lung disease or certain heart problems
  - People with weakened immune systems (e.g. HIV, organ transplant, chemotherapy)
- Each year in U.S.:
  - > 57,000 children below age 5 are hospitalized for RSV
  - ~ 177,000 older adults are hospitalized, and about 14,000 die
- No safe and effective treatments

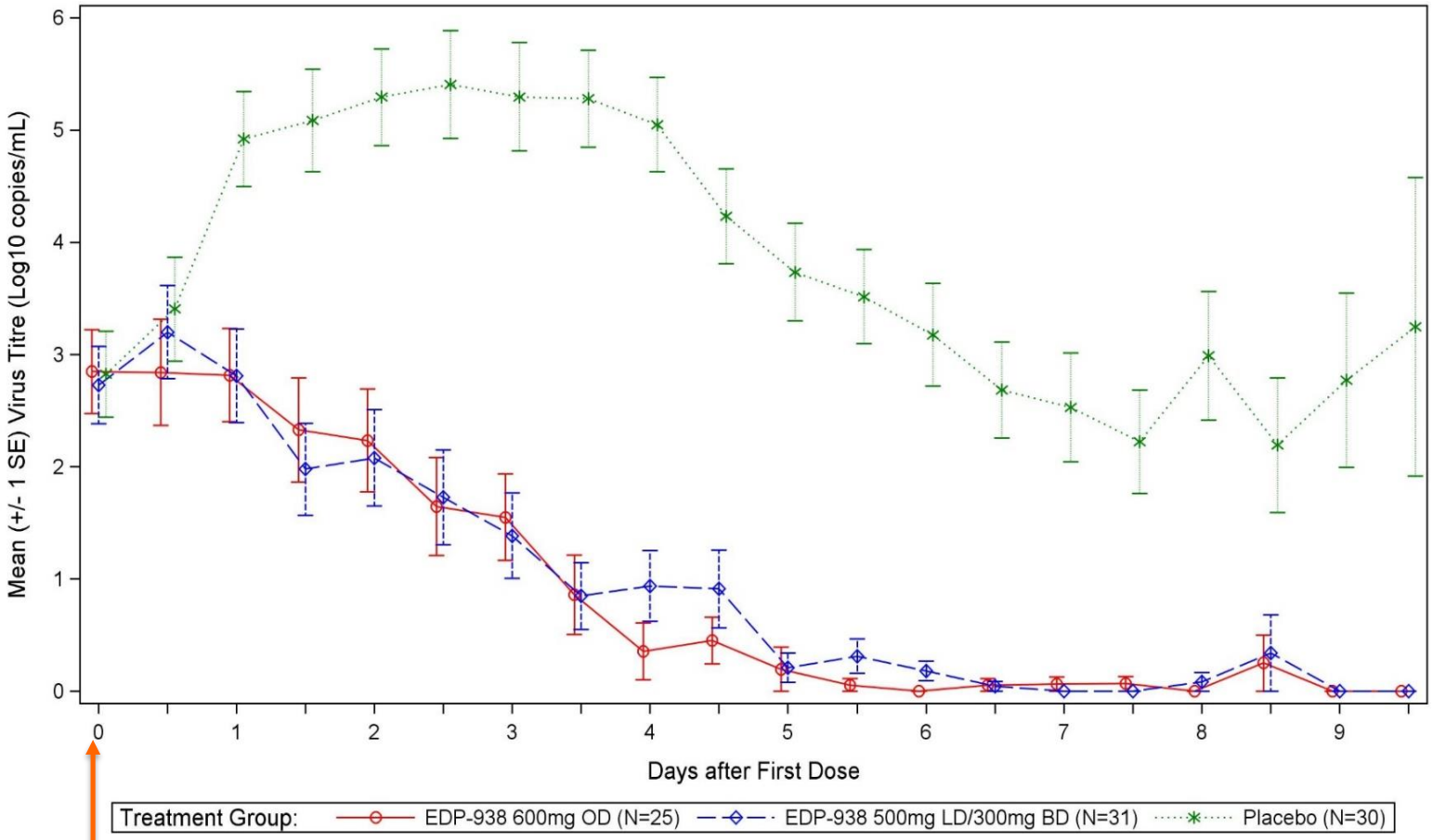
Source: CDC

EDP-938: N-Protein Inhibitor for RSV

- EDP-938 is the only N-inhibitor under clinical evaluation
  - Non-Fusion approach directly targets viral replication
- Strong preclinical virological profile:
  - Nanomolar inhibitor of both RSV-A and RSV-B activity
  - Maintained antiviral potency across all clinical isolates tested
  - Demonstrated high-barrier to resistance *in vitro*
  - Synergy with other drug mechanisms (*e.g.* fusion and L inhibitors)
  - Active against resistant virus from other mechanisms
  - Robust *in vivo* efficacy data
- Phase 2a human challenge study met primary and key secondary efficacy endpoints
- Phase 2b “RSVP” study in adult outpatients initiated
- Additional Ph2 RSV studies in pediatric patients and adult transplant patients targeted to begin 4Q 2020

Robust Antiviral Effect

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



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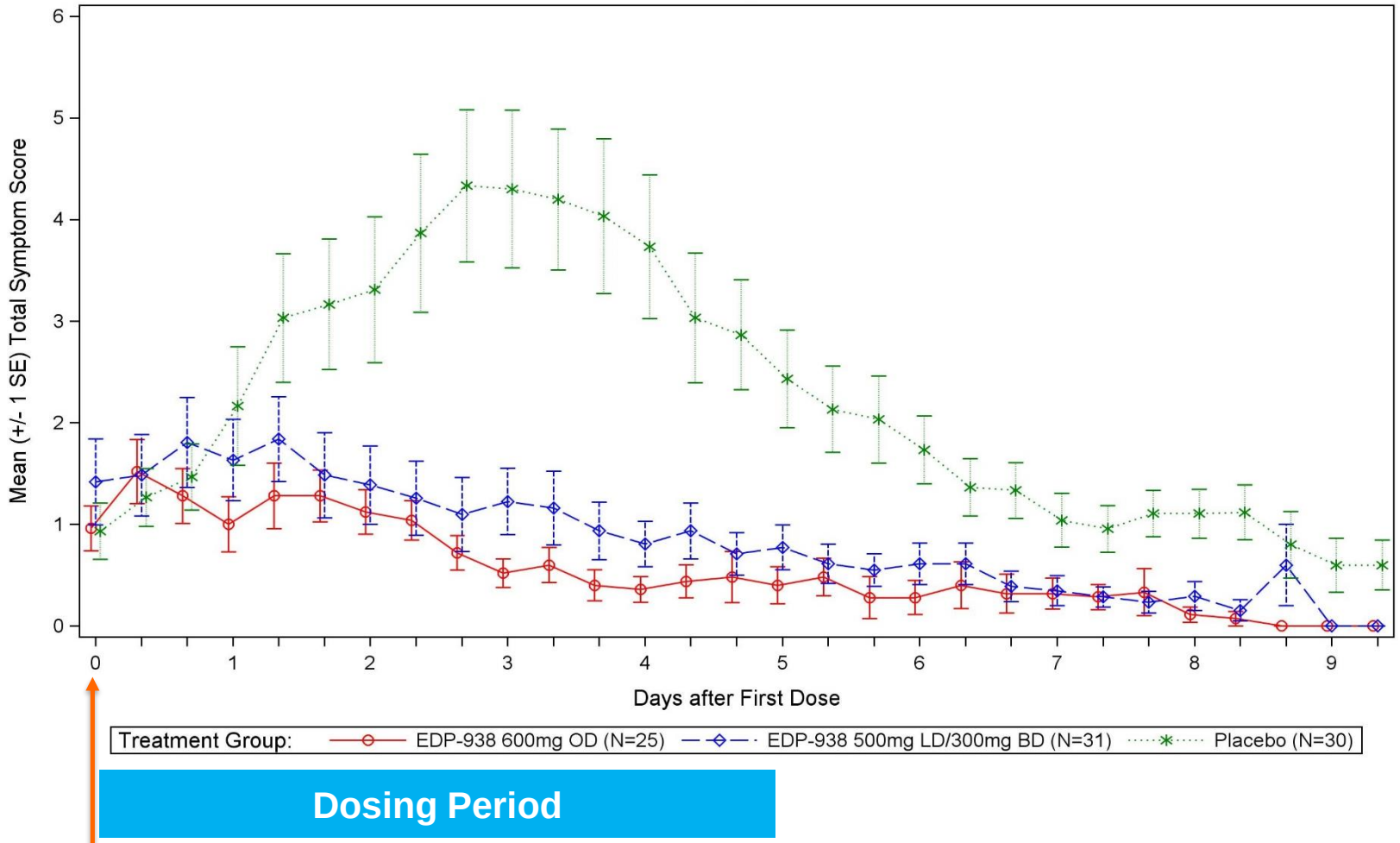
EDP-938 Shows a Rapid and Sustained Attenuation of RSV Symptoms in Both Active Arms Compared to Placebo (68%, 74%↓ AUC; P<0.001)



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Summary: EDP-938, A Highly Efficacious and Safe RSV N-Inhibitor

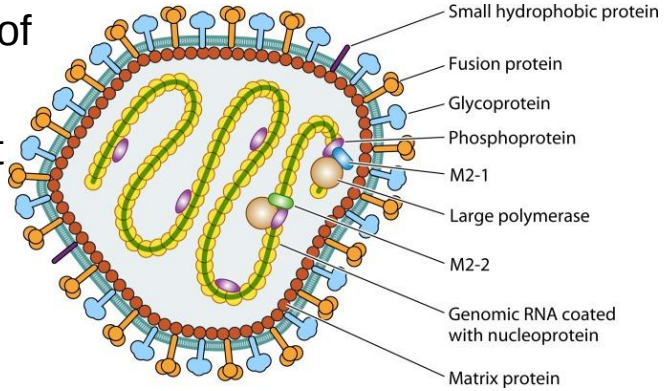
- Phase 1 results:
  - Safe and well tolerated, no SAEs, AEs were mild
  - At Phase 2 doses, mean trough levels 30x higher than EC_{90} of EDP-938 against RSV-infected human cells
- Phase 2a Challenge Study Results:
  - Primary and Key Secondary Efficacy Endpoints were achieved ($p<0.001$) at both dose levels after 5 days of dosing
  - EDP-938 mean C_{trough} concentrations were approximately >20-40x higher than EC_{90}
  - Well tolerated with safety profiles similar to placebo
  - Consistent profile observed in >250 subjects exposed to EDP-938 for up to 7 days in Phase 1 and Phase 2a

"RSVP" – a Phase 2b Study in Adult Outpatients with RSV

- RSVP Study Design:
  - Randomized, double-blind, placebo-controlled in approximately 70 subjects, up to the age of 75 years
  - 800mg EDP 938 or placebo for 5 days
    - comparable to 600mg suspension dosage form used in challenge study
  - Subjects will be followed for a total of 14 days
  - Primary Objective: to evaluate the effect of EDP-938 on progression of RSV infection by assessment of clinical symptoms measured over the course of the 14-day study observation period
- Goal: Topline data 3Q 2020 assuming completion in one RSV season in northern hemisphere

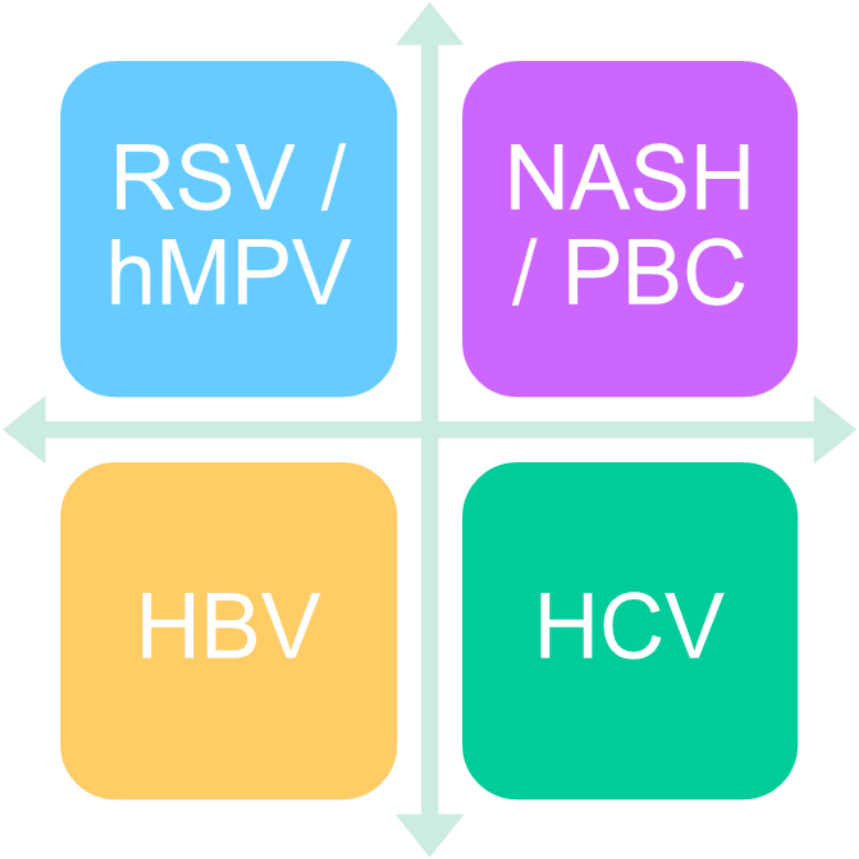
Human Metapneumovirus (hMPV)

- Paramyxovirus closely related to RSV
  - Has the ability to cause mild to severe disease in people of all ages
  - hMPV replication dependent on several viral proteins that form a multiprotein complex in cells
    - Multiple potential targets for hMPV drug discovery



- Important cause of respiratory tract infections (RTIs), particularly in children, the elderly, & immunocompromised individuals
  - 2nd most common cause of lower RTIs in children (behind RSV)
  - Reinfection with hMPV occurs throughout life
- No approved vaccine or therapeutics available
- Enanta nanomolar hMPV inibitor leads under active optimization

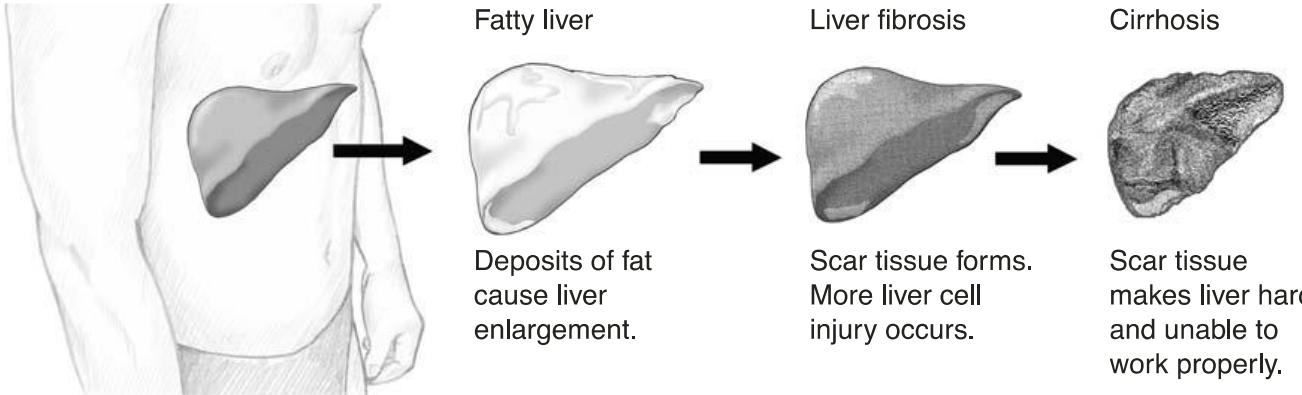
Virology & Liver Disease Focus Areas



Non-Alcoholic Fatty Liver Disease (NAFLD) and Non-Alcoholic Steatohepatitis (NASH)

- Number one cause of liver disease in Western Countries
- NAFLD: excessive fat (triglyceride) accumulation in the liver (steatosis)
- A subgroup of NAFLD patients has liver cell injury and inflammation in addition to excessive fat (steatohepatitis), *i.e.* NASH
- NASH is associated with the metabolic syndrome – diseases related to type 2 diabetes, insulin resistance, obesity, hyperlipidemia, and hypertension
- While NAFLD does not correlate with short-term morbidity or mortality, but progression to NASH dramatically increases risks of cirrhosis, liver failure, and hepatocellular carcinoma

Stages of Liver Injury
(NIDDK)



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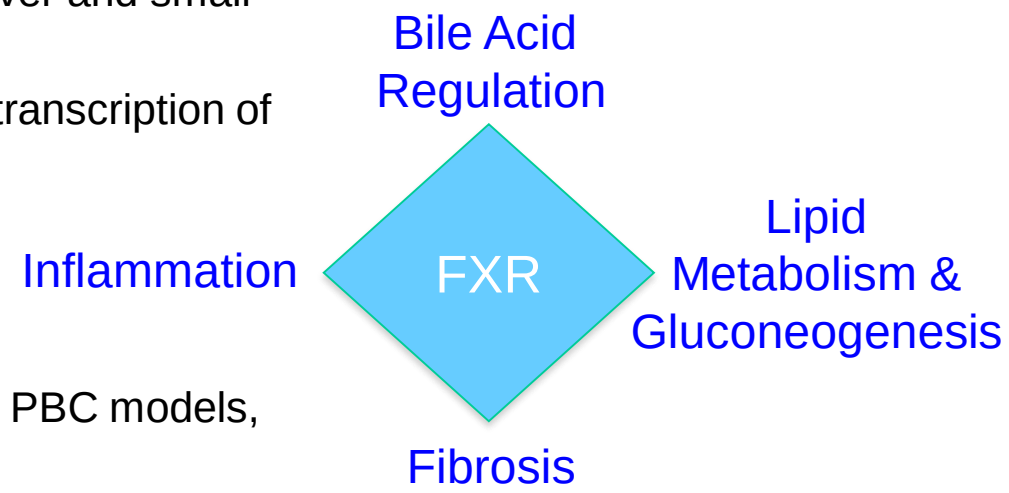
Enanta's Approach to NASH and PBC– Agonists of Farnesoid X Receptor (FXR)

- FXR
  - nuclear receptor
  - main regulator of bile acid levels in liver and small intestine
  - responds to bile acids by regulating transcription of key enzymes and transporters
- FXR agonist preclinical PoC
  - ameliorate pathologies in NASH and PBC models, including an effect on fibrosis
- Clinical validation of FXR agonist in NASH and PBC with 6-ECDCA (OCA)



Source: Matsubara Mol Cell Endocrinol 2013; Neuschwander-Tetri et al, Lancet, 2014

FXR Agonist EDP-305: Introduction

- EDP-305: Non-bile Acid
  - Steroidal non-carboxylic acid, modified with additional non-steroidal binding element to enhance potency
- Potent FXR receptor agonist activity *vs* OCA
- Highly selective for FXR *vs* other nuclear receptors
  - and *vs* TGR5 receptor
- Potent and differentiated effects on FXR-dependent gene expression *vs* OCA
  - *e.g.* Shp, Cyp7a1, Bsep, Fgf15/FGF19
  - human hepatocytes and *in vivo* mouse model
- Efficacy in multiple NASH models
  - STAM™ mouse NASH model and dietary-induced NASH (DIN) mouse model
  - Improvement in hepatocyte ballooning and overall NAFLD Activity Score vs OCA
- Reduced liver fibrosis in rodent models
  - Mdr2-/-, MCD, CDAHFD, thioacetamide, and bile duct ligation models

FXR Agonist EDP-305: Phase 2 Studies

- Fast Track Designation granted by FDA for PBC and for NASH with fibrosis
- Phase 2 studies in NASH and PBC
  - ARGON-1 study in NASH complete
  - INTREPID study in PBC ongoing (data targeted for 2Q 2020)
  - Design: 12-week dose ranging, randomized, double-blind, placebo-controlled
  - Evaluate safety, tolerability, PK, and efficacy (ALP reduction in PBC and ALT reduction in NASH)
- Positive Ph2a ARGON-1 results warrant further studies in NASH (Ph2b ARGON-2)

Summary of EDP-305 ARGON-1 Study

- Primary (ALT change) and key secondary (liver fat by MRI-PDFF) endpoints were met at week 12 using 2.5mg dose
- Strong target engagement as shown by reductions in C4, and increases in FGF-19 and ALP
- Robust reduction in marker of liver injury (GGT)
- Generally safe for up to 12 weeks
  - majority of TEAEs were mild to moderate
  - incidence of treatment discontinuation due to pruritus was 1.8% for 1mg and 20.8% for 2.5mg
  - associated with small numeric absolute changes in lipids

EDP-305 Next Steps: ARGON-2, a Phase 2b NASH Study

- Initiate Ph2b NASH study in 2Q 2020:
  - Randomized, placebo-controlled in biopsy-proven NASH patients (~ 340)
  - 72-week treatment duration
  - includes a 12-week interim analysis (to generate dose information more quickly for potential combinations)
  - Primary endpoint: Improvement of fibrosis without worsening of NASH and/or NASH resolution without worsening of fibrosis
- Two doses selected to provide strong target engagement and a balanced profile in terms of efficacy & tolerability:
  - 1.5 mg dose: designed to demonstrate stronger biomarker signals of efficacy than seen at 1.0mg
  - 2.0 mg dose: designed to demonstrate less pruritus than seen at 2.5mg

EDP-297 – A Potent and Differentiated Follow-on FXR Agonist

- EDP-297 preclinical profile shows:
  - high target-tissue distribution (liver and intestine) *vs* plasma and skin
  - potency greater than that published on any FXR agonist in clinical development today
- A highly potent *and* highly targeted FXR agonist may allow for lower doses and reduced drug levels at non-targeted tissues
  - potential to reduce pruritus unless pruritus is FXR-mediated by FXR receptors in liver or intestine
- Planned phase 1 study initiating mid-calendar 2020 and data expected in 1H 2021

EDP-297 is a Highly Potent FXR Agonist with Excellent Target Tissue Distribution

Compound		FXR FL Activation EC_{50} (nM)	Dose (mouse, *po*)	Intestine / Plasma @ 4 hrs ~ Tmax	Liver / Plasma @ 4 hrs ~ Tmax
OCA	Bile Acid	130 [1]	10 mg/kg	160	26
cilofexor	Non-Bile Acid	41 [2]	1 mg/kg	0.6	0.9
EDP-305	Non-Bile Acid	8	1 mg/kg	7	15
tropifexor	Non-Bile Acid	0.4 [3]	1 mg/kg	0.8	8
EDP-297 [4]	Non-Bile Acid	**<0.1**	1 mg/kg	**265**	**75**

Enanta data except where noted

1. EC_{50} = 99 nM reported by Intercept
2. Gilead data. Trauner *et al Hepatology* 2019
3. EC_{50} = 0.26 nM reported by Novartis. Tully *et al J. Med. Chem.,* 2019
4. **EDP-297 is undetectable in mouse skin**

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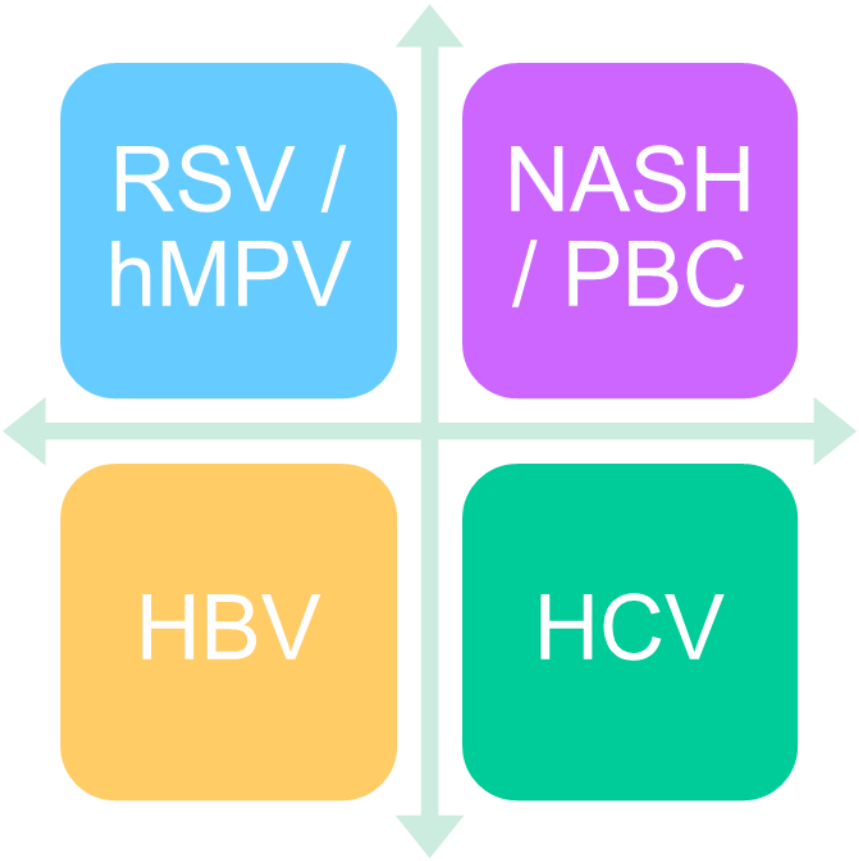
Virology & Liver Disease Focus Areas



HBV Background

- Potentially life-threatening liver infection caused by the hepatitis B virus
- Current treatments rarely give true cures
  - **Interferon** gives better results (~10%), but with side effects
  - **RT inhibitors** very effective at reducing viral load, but offer very low cure rates (1% or lower) and must be taken for life to improve cirrhosis or HCC outcomes
- Prevalence estimates
  - US: ~850,000 - 2 million
  - US + Japan + major EU populations: ~4.9 million
  - Worldwide: ~250 million
- Estimated 15-25% of patients with chronic HBV infection will develop chronic liver diseases including cirrhosis, HCC, or liver decompensation

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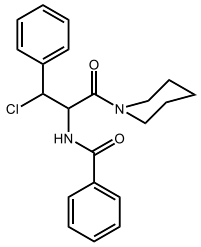
Sources: WHO, CDC, Datamonitor

Core inhibitors: Introduction

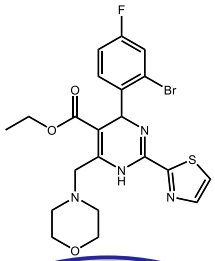
(also called capsid assembly modulators, core protein allosteric modulators, capsid inhibitors)

- Novel class of replication inhibitor
- Act at multiple steps in HBV lifecycle
  - prevent proper uncoating, nuclear import, assembly, and recycling

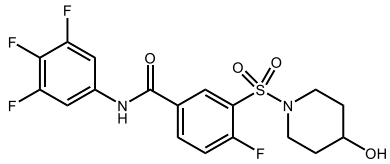
Phenylpropemides (AT-130)



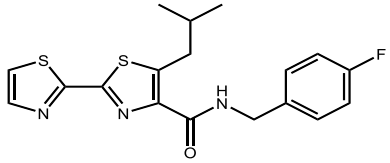
HAPs (GLS4)

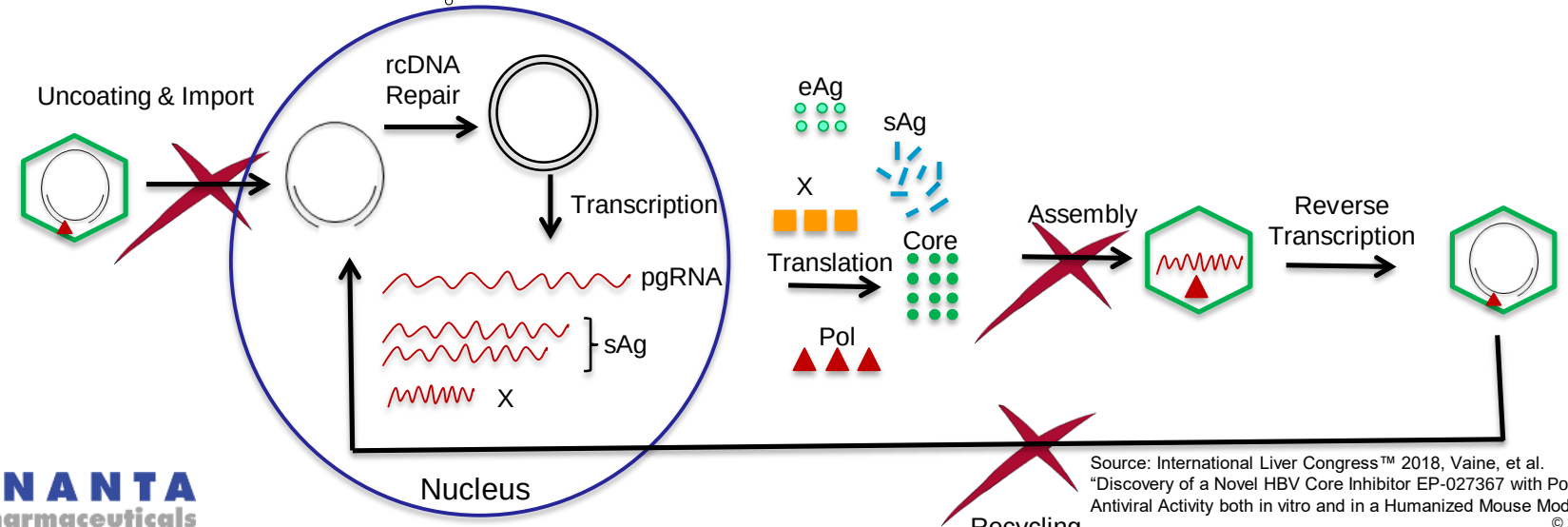


Sulfamoylbenzamides (SBA-R01)



Isothiafludine (NZ-4)





Source: International Liver Congress™ 2018, Vaine, et al. "Discovery of a Novel HBV Core Inhibitor EP-027367 with Potent Antiviral Activity both in vitro and in a Humanized Mouse Model"

Core Inhibitor EDP-514 is a Potent Inhibitor of HBV Replication

- EDP-514 is active in multiple HBV stable cell lines

	HBV Stable Cell Line EC_{50} (nM)		
	HepAD38	HepDE19	HepG2.2.15
Intracellular Viral DNA	18	27	17
Encapsidated pgRNA	25	3	5
HBeAg	20	34	>500*

* In HepG2.2.15 cells, HBeAg is transcribed off transgene and is not dependent on viral replication

Viral DNA measured by qPCR
Encapsidated pgRNA measured by modified pulldown and qPCR
HBeAg measured by commercial ELISA kit

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EDP-514 Prevents *de novo* Formation of cccDNA in Primary Human Hepatocytes

- EDP-514 prevents cccDNA establishment when present at early time points in infection (HBsAg as surrogate marker)

Compound	HBsAg EC_{50} (nM)		HBV DNA EC_{50} (nM)	
	d0 Addition	d3 Addition	d0 Addition	d3 Addition
EDP-514	35	>1000	10	6
Entecavir	>1000	>1000	0.25	0.21

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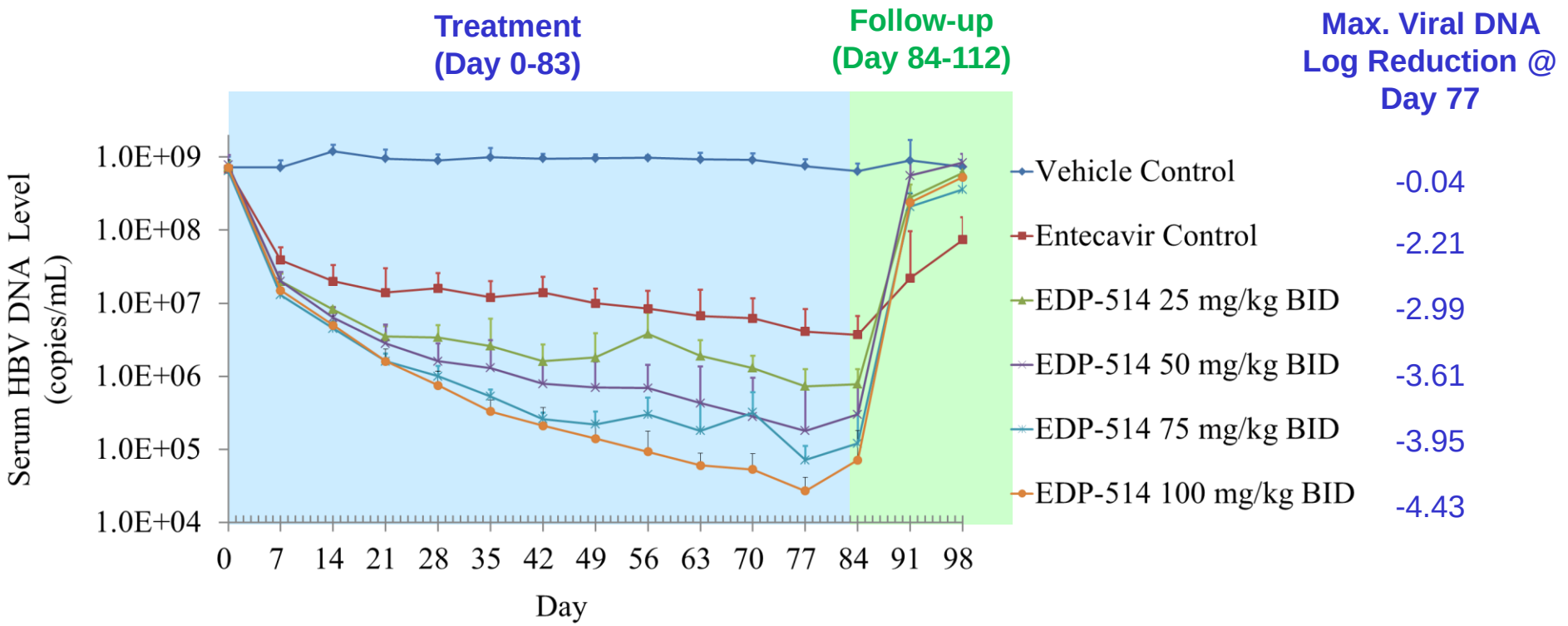
EDP-514 is Efficacious in the Humanized Liver Mouse Model

- uPA/SCID mice were infected with genotype C HBV and subsequently treated with EDP-514 BID at indicated doses for 12 weeks



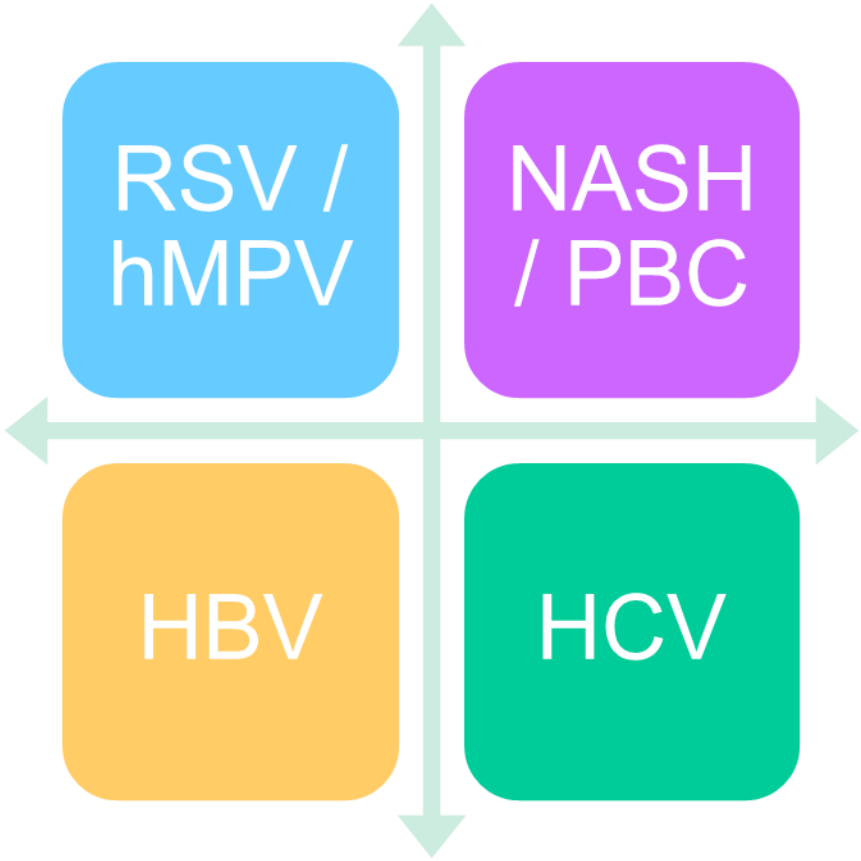
HBV Core Inhibitor EDP-514 Summary

- A novel core inhibitor that displays potent anti-HBV activity at multiple points in the HBV lifecycle
- *In vitro*:
  - Potent anti-HBV activity in HBV expressing stable cells lines
  - Capable of preventing the establishment of cccDNA
  - Potent pan-genotypic activity
- *In vivo*:
  - Favorable tolerability and pharmacokinetic profile
  - Over 4-log reduction in HBV viral titers with 12 weeks of treatment in a chimeric liver mouse model
- Fast Track designation by FDA

EDP-514 Phase 1 Development

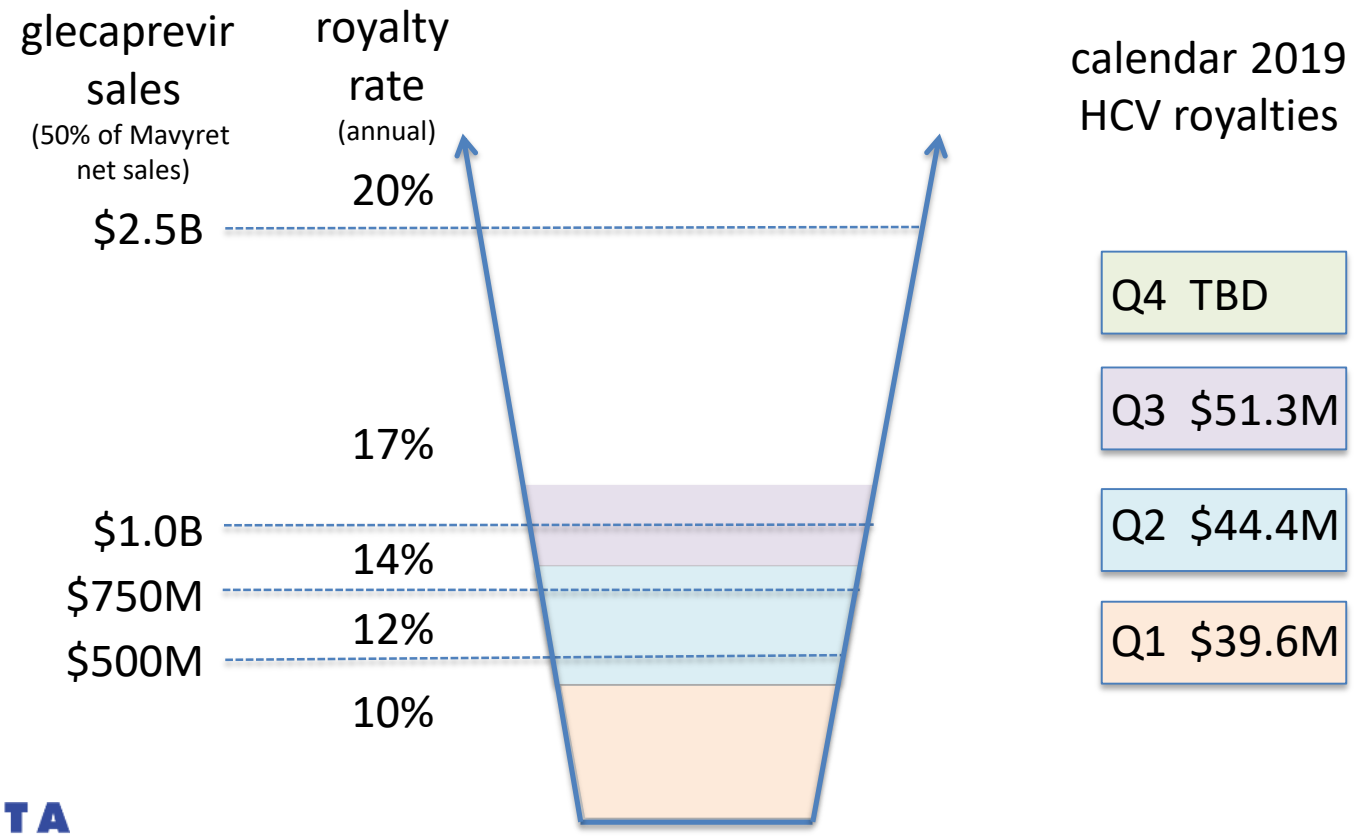
- Phase 1 Ongoing
  - Part 1: Evaluate safety, tolerability PK of SAD/ MAD doses in healthy volunteers; Data expected in 1Q 2020
  - Part 2: Assess safety and antiviral activity in NUC-suppressed patients; To be initiated in 1Q 2020
- Additional Phase 1b study in viremic HBV patients is expected to initiate in 2Q 2020

Virology & Liver Disease Focus Areas



Glecaprevir– Our Licensed Protease Inhibitor for Hepatitis C Virus

Product	Regimen	Enanta Asset	Economics*
MAVYRET glecaprevir/pibrentasvir 100 mg/40 mg tablets	2-DAA (ABBV)	glecaprevir (PI)	Double-digit royalty on 50% of net sales

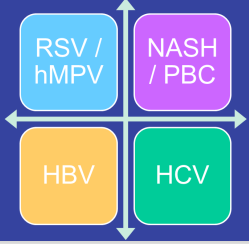


* Enanta also receives royalties on paritaprevir sales (30% of Viekira 3DAA sales, same tiers)

Financial Highlights

($ In millions)	Fiscal Year Ended Sept. 30, 2019	Fiscal 4Q19
Total Revenues	205.2	$51.3
R&D Expenses	$142.2	$38.7
G&A Expenses	$26.2	$6.2
Net Income	$46.4	$9.2
EPS (per diluted share)	$2.21	$0.44
Balance Sheet		
Cash, Cash Equivalents and Marketable Securities	$400.2	$400.2

Key Catalysts and Funding



- RSV N-inhibitor EDP-938 and hMPV Inhibitor Leads
  - Goal: Data from RSVP Phase 2b adult outpatient study 3Q 2020
    - if enrollment can be completed in one RSV season in northern hemisphere
  - Initiate additional Phase 2 RSV studies in pediatrics and transplant 4Q 2020
  - Optimize nanomolar hMPV inhibitor leads
- NASH / PBC: FXR Agonists EDP-305 and EDP-297
  - Initiate ARGON-2 Phase 2b in NASH by early 2Q 2020
  - Phase 2 data from INTREPID study for PBC in 2Q 2020
  - Initiate Phase 1 for EDP-297 (follow-on FXR) in mid-2020
  - Advance non-FXR compounds for NASH
- HBV: Core Inhibitor EDP-514
  - Phase 1 data in healthy volunteers in 1Q 2020
  - Initiate Phase 1b in nuc-suppressed HBV patients in 1Q 2020
  - Initiate Phase 1b in viremic HBV patients in 2Q 2020
- Funding by double-digit HCV royalties from glecaprevir (MAVYRET™)

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Creating Small Molecule Drugs for Viral Infections and Liver Diseases

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