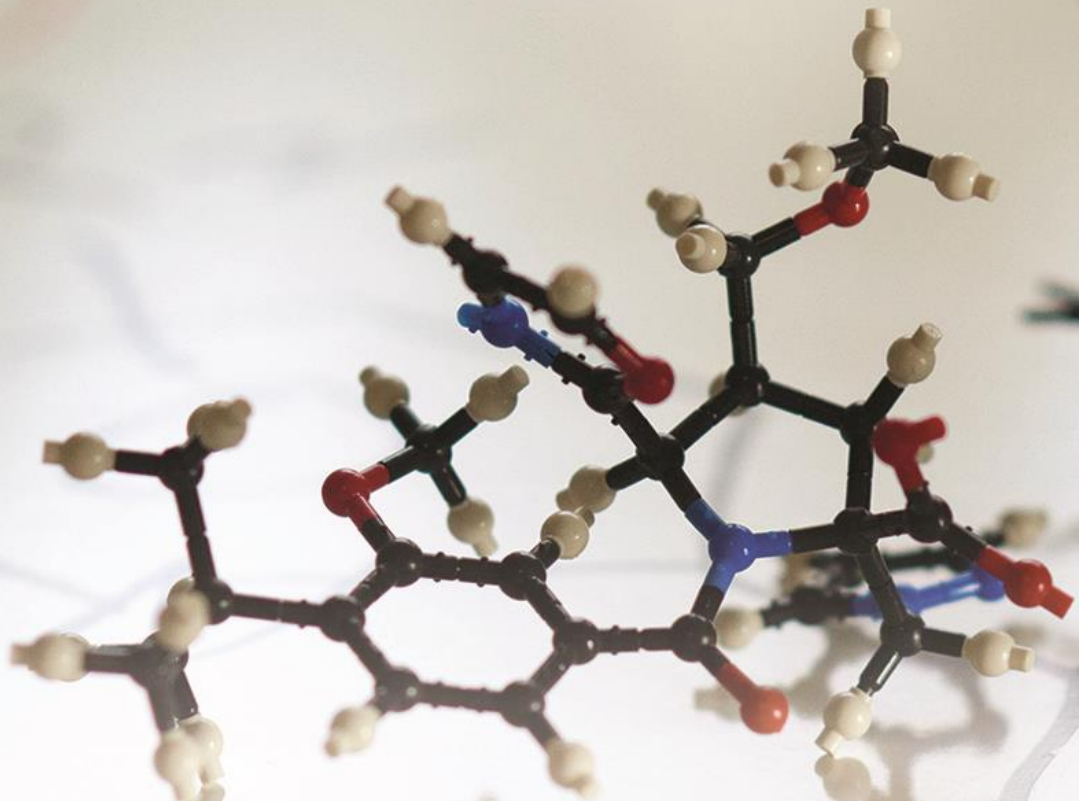
Corporate Presentation

January 7, 2019



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
- Two partnered products marketed in AbbVie's HCV regimens:
  - Glecaprevir – HCV protease inhibitor in MAVYRET™/MAVIRET™
  - Paritaprevir – HCV protease inhibitor in VIEKIRA* regimens
  - Fiscal 4Q18 royalties on HCV regimens: $67 million
- Three clinical-stage programs in areas of high unmet medical need:
  - RSV: Phase 2 human challenge study ongoing
  - NASH: Phase 2 "ARGON-1" study ongoing
  - PBC: Phase 2 "INTREPID" study ongoing
- Ongoing R&D programs in NASH/PBC, HBV and RSV
- Strong balance sheet to fund clinical programs and other R&D efforts
  - Approx. $325.1M in cash at 9/30/18

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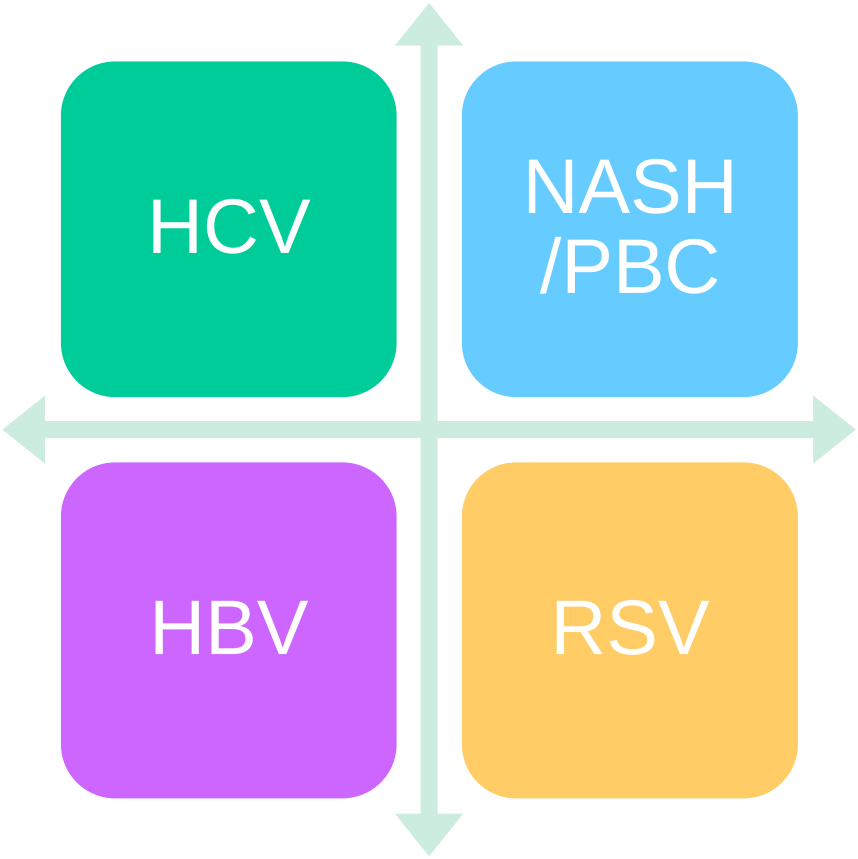
*VIEKIRA regimens include AbbVie's marketed HCV regimens of VIEKIRA PAK® and VIEKIRAX™

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	Preclin	Phase 1	Phase 2	Phase 3	Market
HCV	Protease Inhibitor	glecaprevir – containing pan-genotypic 2-DAA combo					MAVYRET glecaprevir/pibrentasvir
HCV	Protease Inhibitor	paritaprevir – containing regimens					
NASH	FXR Agonist	EDP-305	Ph2 “ARGON-1”				
PBC	FXR Agonist	EDP-305	Ph2 “INTREPID”				
RSV	N-protein Inhibitor	EDP-938	Ph2 Challenge Study				
HBV	Core Inhibitor	EDP-514					
NASH	FXR Agonist Follow-on						
NASH	Undisclosed						

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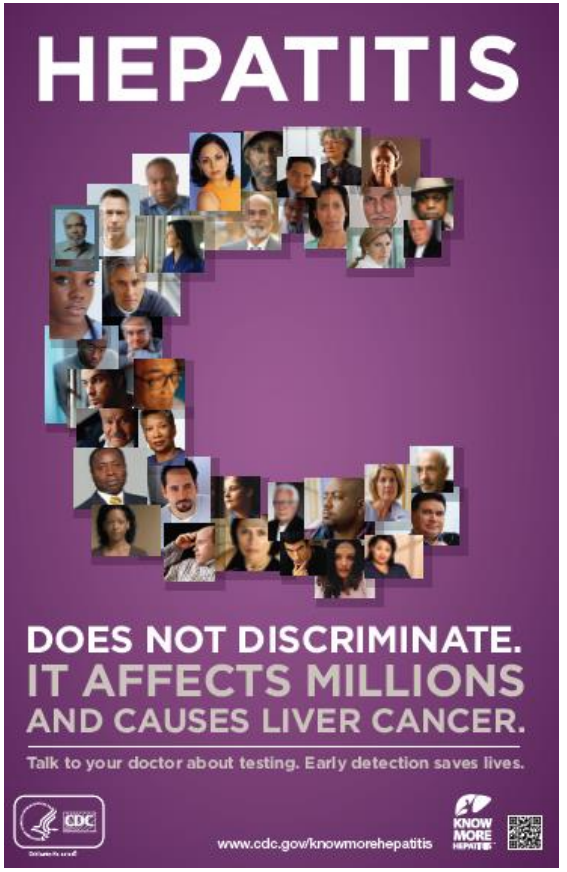
HCV Market

- Market for HCV therapies:
  - Approx. $12.5B for 2017
- Prevalence of chronic infection
  - Globally: ~ 71M infections, ~ 400K deaths*
  - US: ~ 2.7 to 3.9M (CDC)
  - Europe: ~ 14M** to 15M***
  - Japan: ~ 1.5M to 2M****



Source: www.cdc.gov

* WHO http://www.who.int/mediacentre/factsheets/fs164/en/

** Hepatitis C in the WHO European Region – Fact Sheet. http://www.euro.who.int/__data/assets/pdf_file/0010/283357/fact-sheet-en-hep-c-edited.pdf

*** EASL and HIV in Europe, "The Number of People Living with Viral Hepatitis is Increasing", Press release Oct. 22, 2015. available from: http://newsite.hiveurope.eu/Portals/0/Newsletters/HiE_late%20present_Press%20release_new_2015OCT_final.pdf

**** Kohnodai Hospital. National Center for Global Health and Medicine [cited 20 February 2013]. Available from:http://www.ncgm.go.jp/center/forpatient_hcv.html

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Glecaprevir–
A Pan-genotypic HCV Protease Inhibitor

- Glecaprevir: the protease inhibitor in AbbVie's MAVYRET™*

  - RBV-free, once-daily, fixed-dose combination (2-DAA)

- MAVYRET treats the majority of patients today (treatment naïve/non-cirrhotic) in only 8-weeks

- Also treats patients with specific challenges:
  - compensated cirrhosis
  - severe chronic kidney disease
  - PI or NS5A treatment failures

- Marketed by AbbVie (U.S., EU, Japan & other countries globally)

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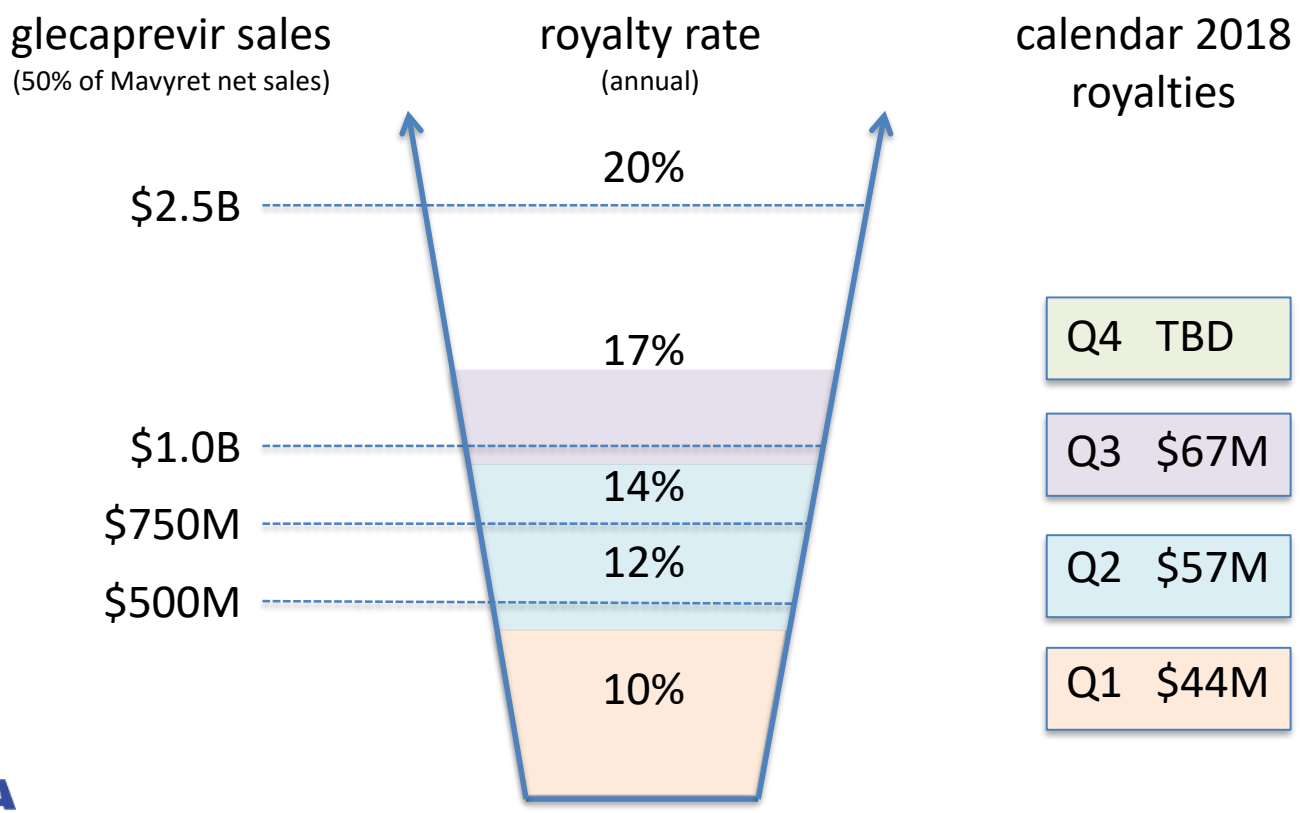
*sold as MAVYRET™ in the U.S., MAVIRET™ outside the U.S.

Glecaprevir– The Pan-genotypic HCV Protease Inhibitor in AbbVie's MAVYRET™

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



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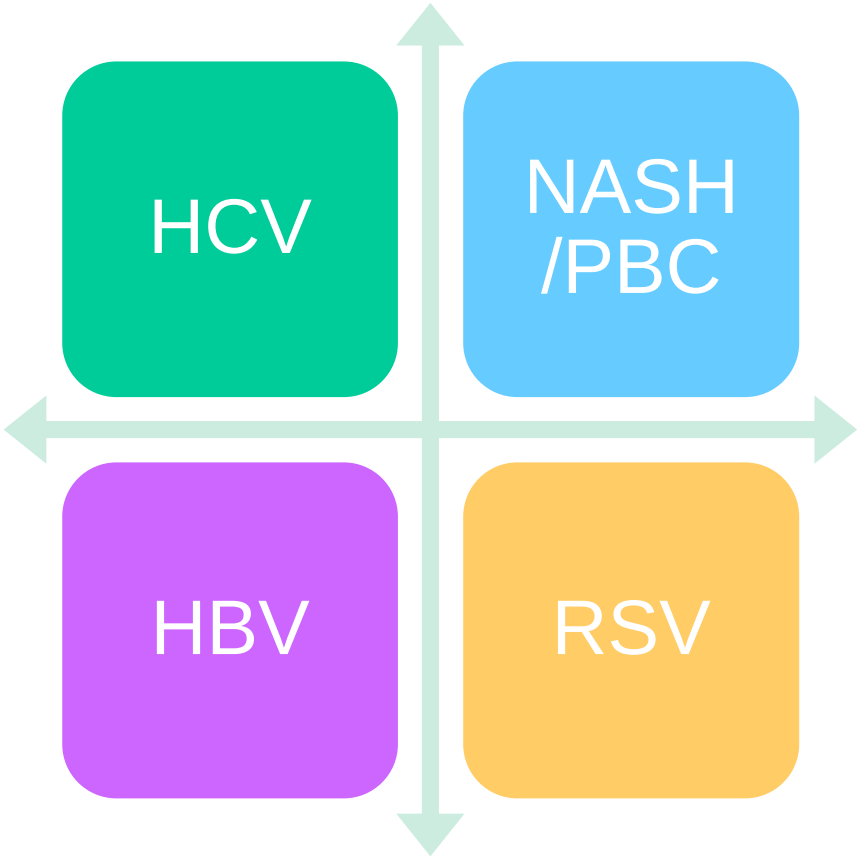
* Enanta also receives royalties on paritaprevir sales (30% of Viekira 3DAA sales, same tiers)

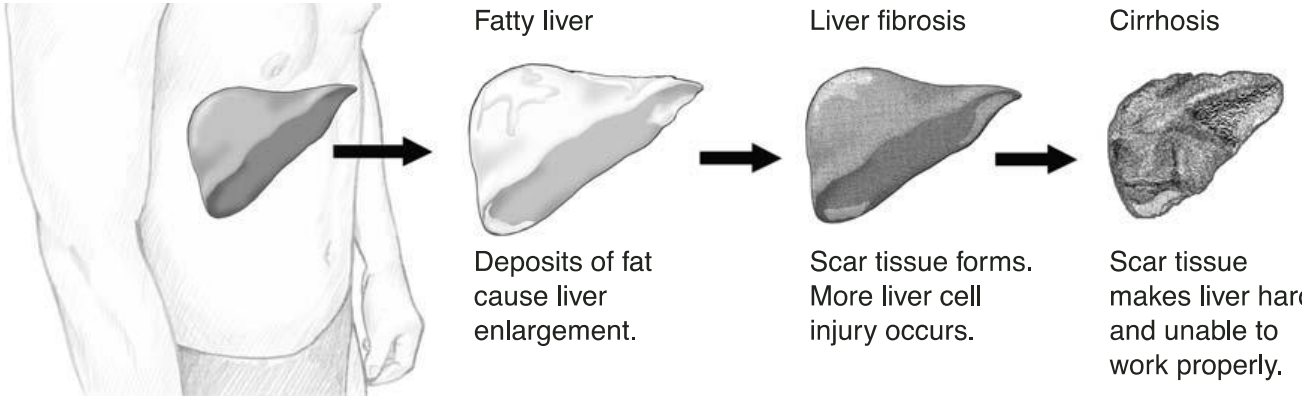
Virology & Liver Disease Focus Areas



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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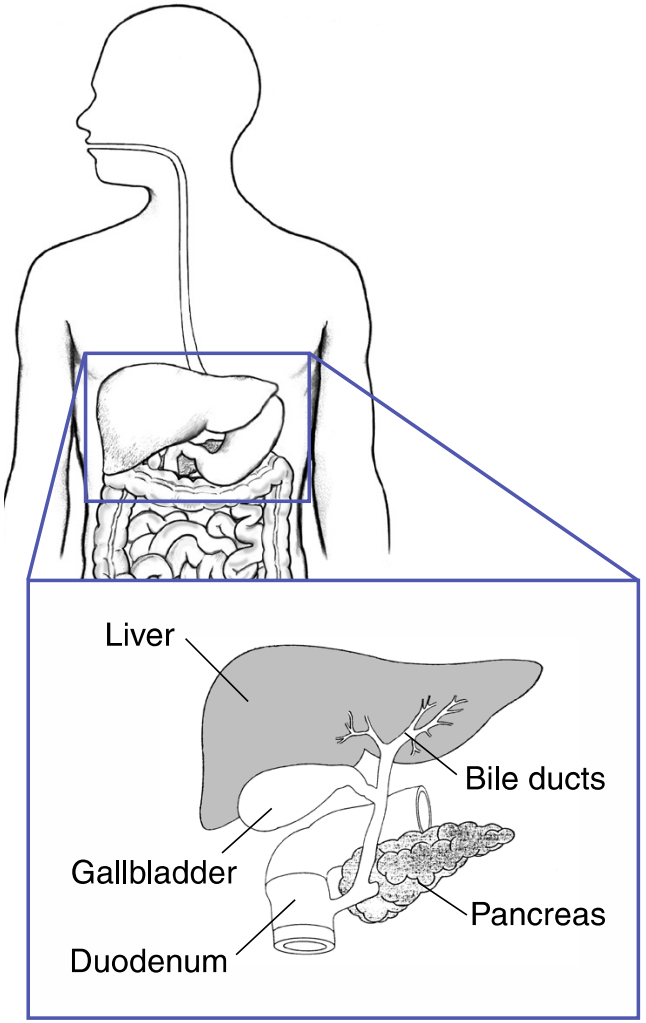
Primary Biliary Cholangitis (PBC)

- PBC is a chronic inflammatory liver disease
- Slowly destroys bile ducts, causing bile to remain in the liver
- Leads to liver cell damage, cirrhosis, and potential liver failure, liver transplantation, or hepatocellular carcinoma



NIDDK

NASH and PBC Potential Markets

NASH

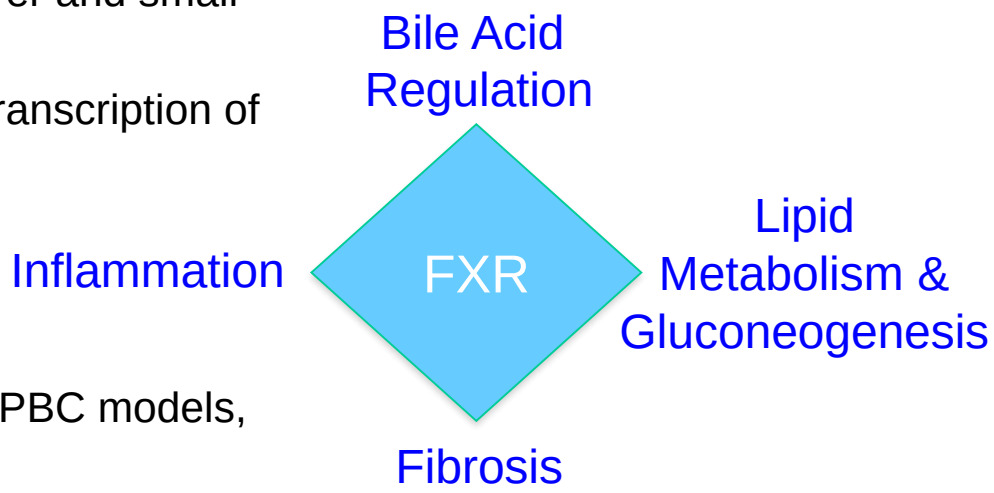
- Currently no approved therapies
- U.S. prevalence estimated to be 3%-5% (~9 to15 million)
  - 20% of whom likely to develop cirrhosis (Rinella, Hepatology, 2011)
- Patient pool size may rival HCV
- Prevalence of NASH likely to increase due to increase in underlying causes, e.g. obesity

PBC

- Estimated U.S. incidence: 4.5 cases for women and 0.7 cases for men per 100,000 population
- Two approved PBC therapies:
  - Ursodiol (ursodeoxycholic acid or UDCA); only effective in 50% of patients
  - OCALIVA®, (OCA) in combination with UDCA in adults with an inadequate response to UDCA or as monotherapy in adults unable to tolerate UDCA

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

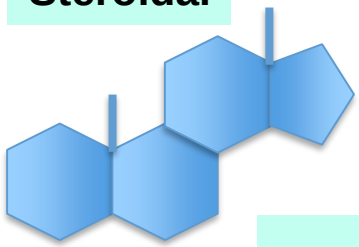
- FXR
  - nuclear receptor
  - main regulator of bile acid levels in liver and small intestine
  - responds to bile acids by regulating transcription of key enzymes and transporters
- FXR agonist preclinical PoC
  - ameliorate pathologies in NASH and PBC models, including an effect on fibrosis
- Clinical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



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

Classification of FXR Agonists – Four fundamental types (with variations)

Steroidal



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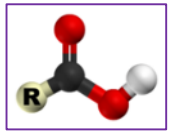
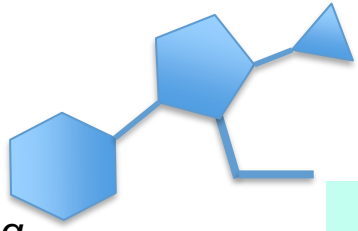


carboxylic acid
non-carboxylic acid

Non-steroidal



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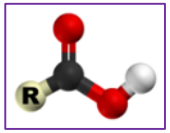


e.g.

carboxylic acid
non-carboxylic acid

FXR Agonists		Example
Steroidal carboxylic acid	**S-CA**	OCA, bile acids
Steroidal non-carboxylic acid	**S-NCA**	Enanta compounds
Non-steroidal carboxylic acid	**NS-CA**	Enanta compounds, GS-9674, LJN452
Non-steroidal non-carboxylic acid	**NS-NCA**	Enanta compounds

FXR Agonist EDP-305: Introduction

- EDP-305: 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

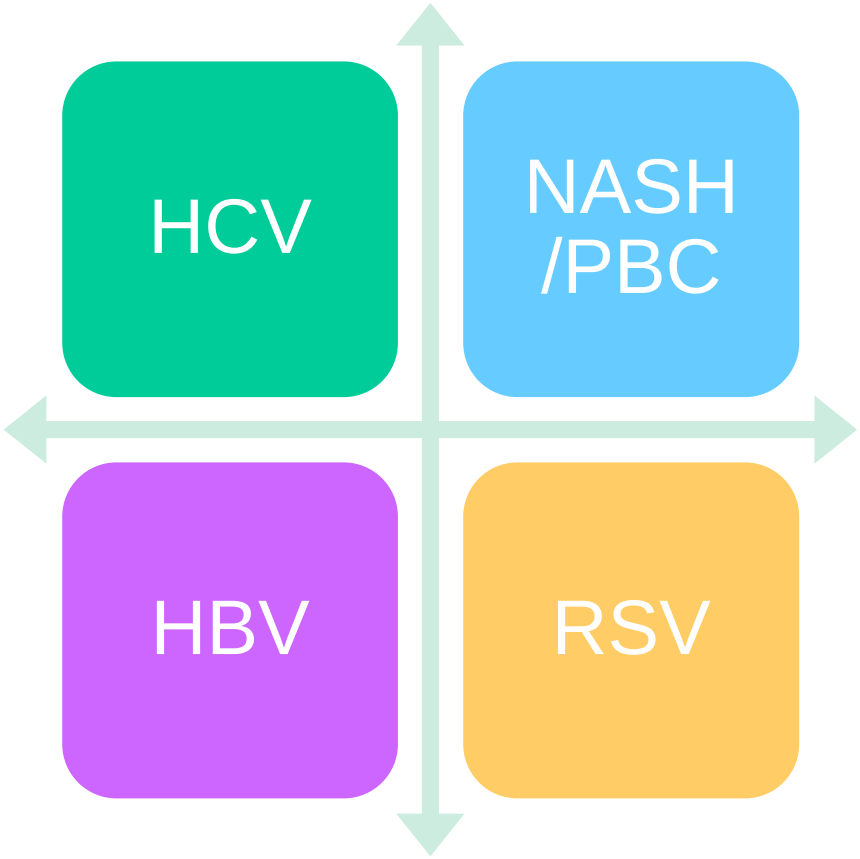
EDP-305 Phase 1 Study

- Double-blind, placebo-controlled, Phase 1a/b study
- Healthy adults, and adults with presumptive NAFLD (“PN”)
  - PN were obese, with or without pre-diabetes or type 2 diabetes mellitus, mean BMI= 32
- Oral suspension EDP-305 or placebo, dosed once daily
  - Total N=146 subjects (n=110 EDP305, n=36 pbo)
  - SAD, n=50, 6 cohorts at 1, 5, 10, 20, 40 and 80 mg
  - MAD, n=48 healthy and n=48 PN, 6 cohorts at 0.5, 1, 2.5, 5, 10, and 20 mg for 14 days
- Safety, tolerability, PK, and proof of target engagement support progression to Ph2 with once daily dosing

FXR Agonist EDP-305: Ph2 Studies

- Fast Track Designation granted by FDA for PBC and for NASH with fibrosis
- Two Ph 2 studies ongoing:
  - “ARGON-1” (NASH) and “INTREPID” (PBC)
  - 12 week dose ranging, randomized, double-blind, placebo-controlled
  - Evaluate safety, tolerability, PK, and efficacy (ALP reduction in PBC and ALT reduction in NASH)
  - New tablet formulation at 1 and 2.5 mg (~2X greater exposure than Ph1 suspension formulation)

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: Enanta's First Clinical-Stage Compound for RSV

- EDP-938 is the only N-inhibitor under clinical evaluation
  - Non-Fusion approach directly targets viral replication
- Strong 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 1 results:
  - Safe and well tolerated, no SAEs, AEs were mild
  - At Phase 2 doses, mean trough levels30x higher than EC90 of EDP-938 against RSV-infected human cells
- Phase 2 human challenge study ongoing

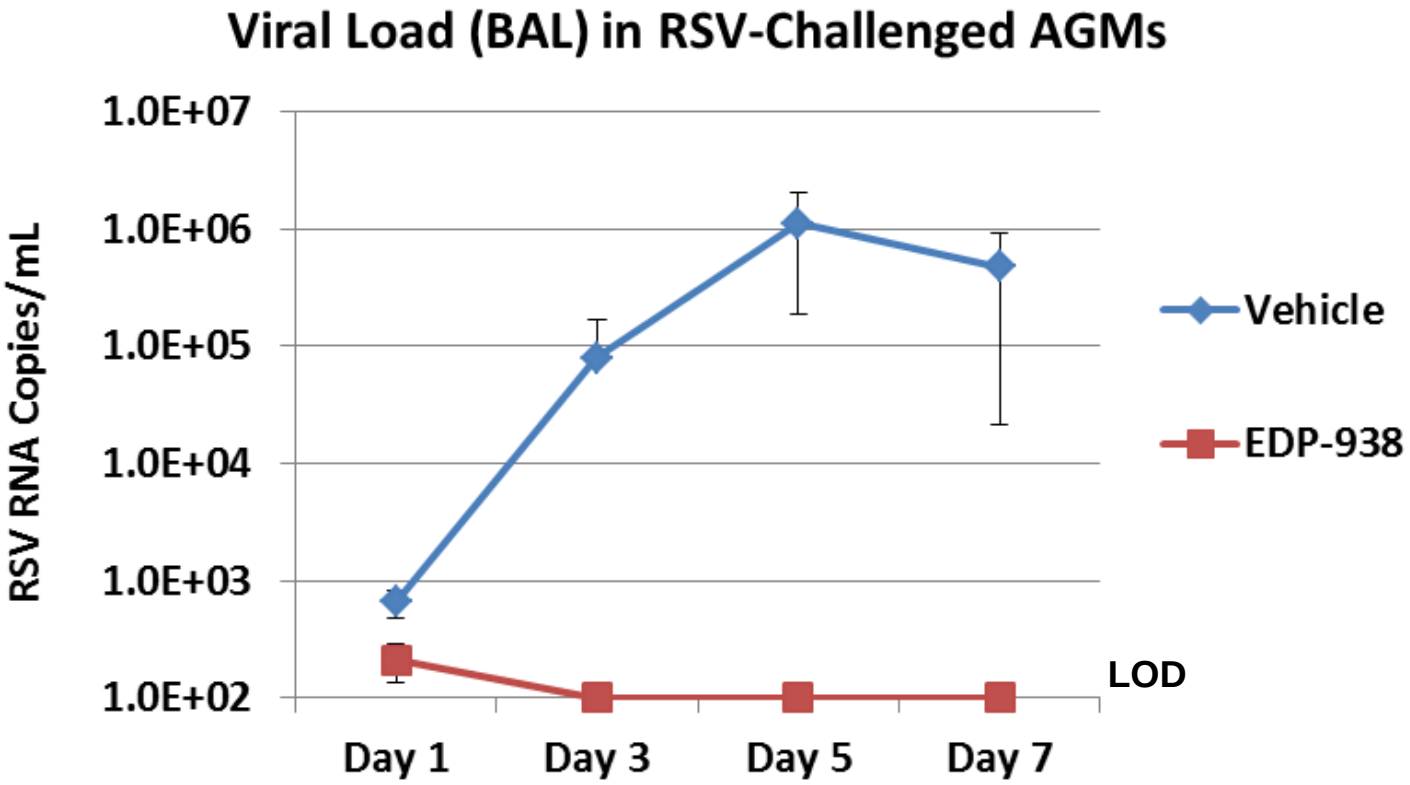
EDP-938 Presents a High Barrier to Resistance and No Cross-Resistance to Other RSV Inhibitors

Compounds	wt RSV EC_{50} (nM)	Drug Resistant (^{R}) Virus					
		EDP-938^R EC_{50} (nM)	Fold Change	AZ-27^R EC_{50} (nM)	Fold Change	GS-5806^R EC_{50} (nM)	Fold Change
EDP-938 (N inhibitor)	53 ± 5	250 ± 53	5	68 ± 8	1	<100	< 2
AZ-27 (L inhibitor)	19 ± 2	29 ± 5	2	>20,000	>1,060	5 ± 1	0.3
GS-5806 (F inhibitor)	5 ± 0.4	2 ± 0.6	0.4	6 ± 0.3	1	>200,000	>40,000

- Resistant virus only selected with EDP-938 starting at low drug concentration (1xEC_{50}) followed by slow increase to 16xEC_{50} after multiple passages
  - selection with higher drug concentration results in elimination of virus rather than development of resistance
- Low level of resistance (fold increase in EC_{50}) with EDP-938 compared to fusion (F) or L inhibitors
- No cross-resistance between EDP-938 and other RSV inhibitors

EDP-938 Dramatically Reduces Viral Load in BAL (Bronchoalveolar Lavage) Fluid

Viral loads in EDP-938 treated animals were below the limit of detection (LOD) on days 3, 5 and 7



100 mg/kg BID of EDP-938 or vehicle control was given 24h prior to infection (day -1), on the day of infection (day 0), and for days 1-4

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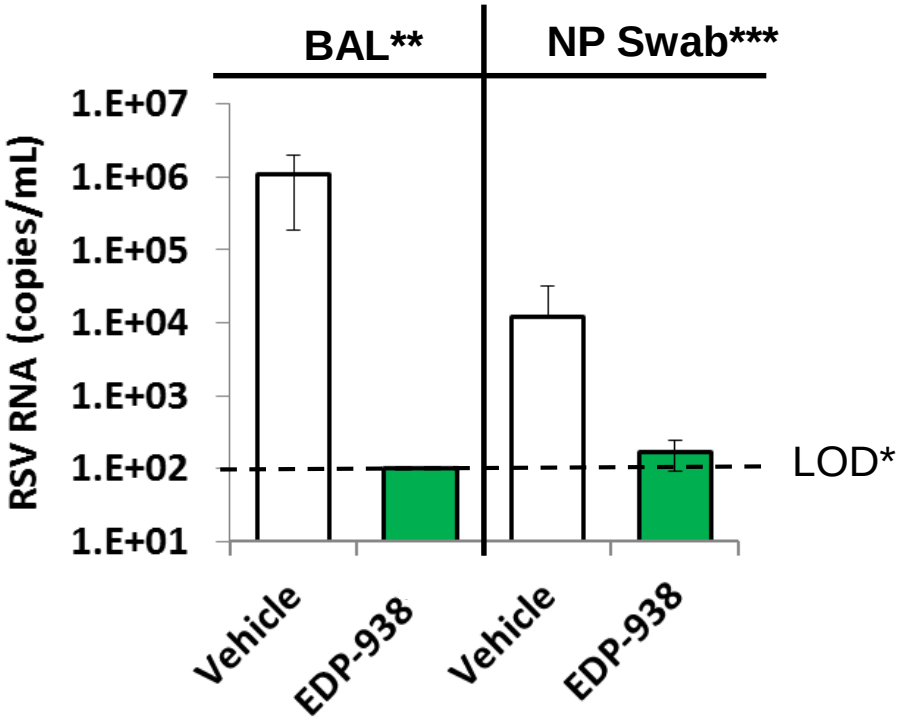
EDP-938 *vs.* ALS-8176: Efficacy at the End of Treatment (Day 5) in AGMs



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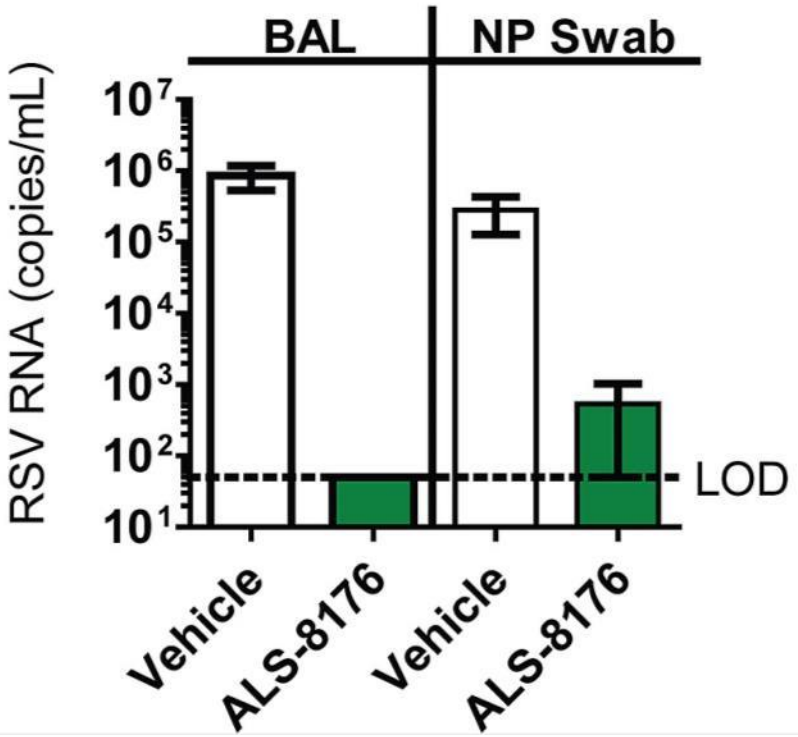

Loading dose of 200 mg/kg ALS-8176 given 24h prior to infection, followed by 50 mg/kg BID on the day of infection, and for 4 additional days.(Deval *et. al., PLoS Pathogens 2015)*

* LOD (limit of detection)
** BAL (bronchoalveolar lavage fluid)
*** NP (nasopharyngeal) Swab

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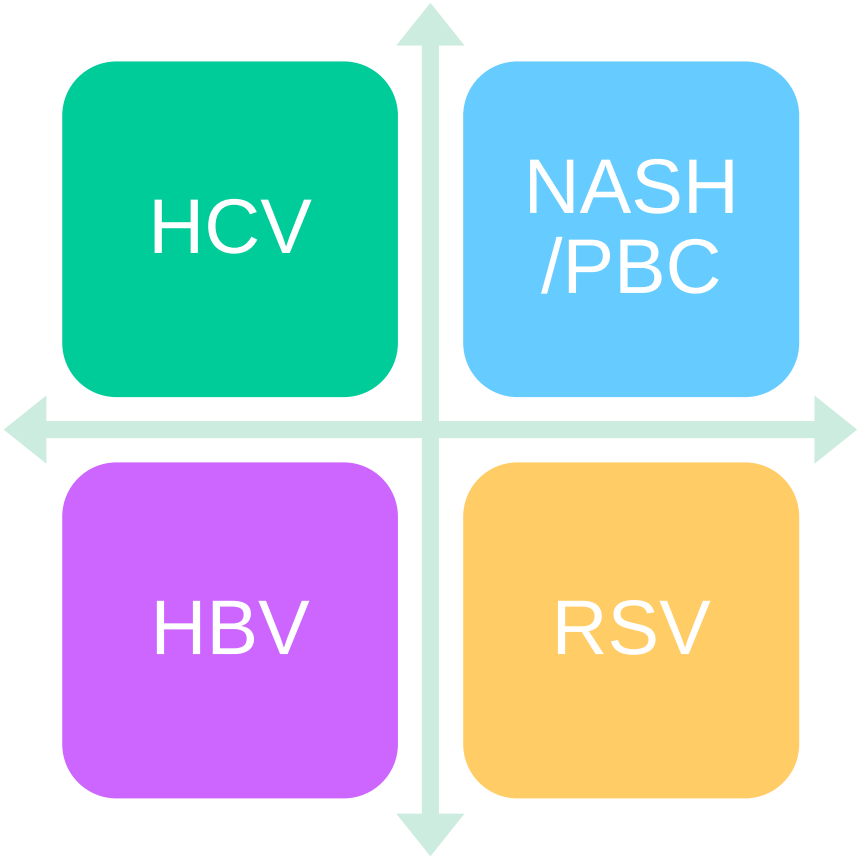
EDP-938: RSV Summary

- Phase 2a Human Challenge study ongoing
  - Randomized, double-blind placebo-controlled trial in healthy adult volunteers infected with attenuated RSV virus to assess efficacy and dose selection for future trials
  - N=114; dosed for 5 days (placebo, 600mg QD, 600 mg BID w/ 500mg loading dose
  - Primary and secondary endpoints include changes in viral load and symptoms
- Topline Phase 2a data in mid-2019
- Future Phase 2 studies will focus on both adult and infant populations
- Regulatory path for clinical studies greatly aided by recent draft guidance from FDA
- Focused path to commercialization may allow “go alone” opportunity for Enanta

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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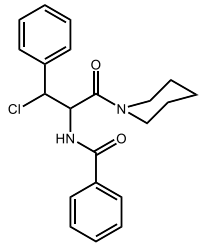
Sources: WHO, CDC, Datamonitor

Core inhibitors: Introduction

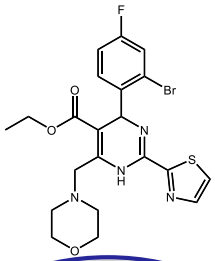
(also called capsid assembly modulators, core protein allosteric modulators, capsid inhibitors)

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

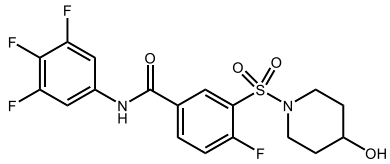
Phenylpropemides (AT-130)



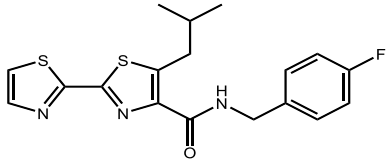
HAPs (GLS4)

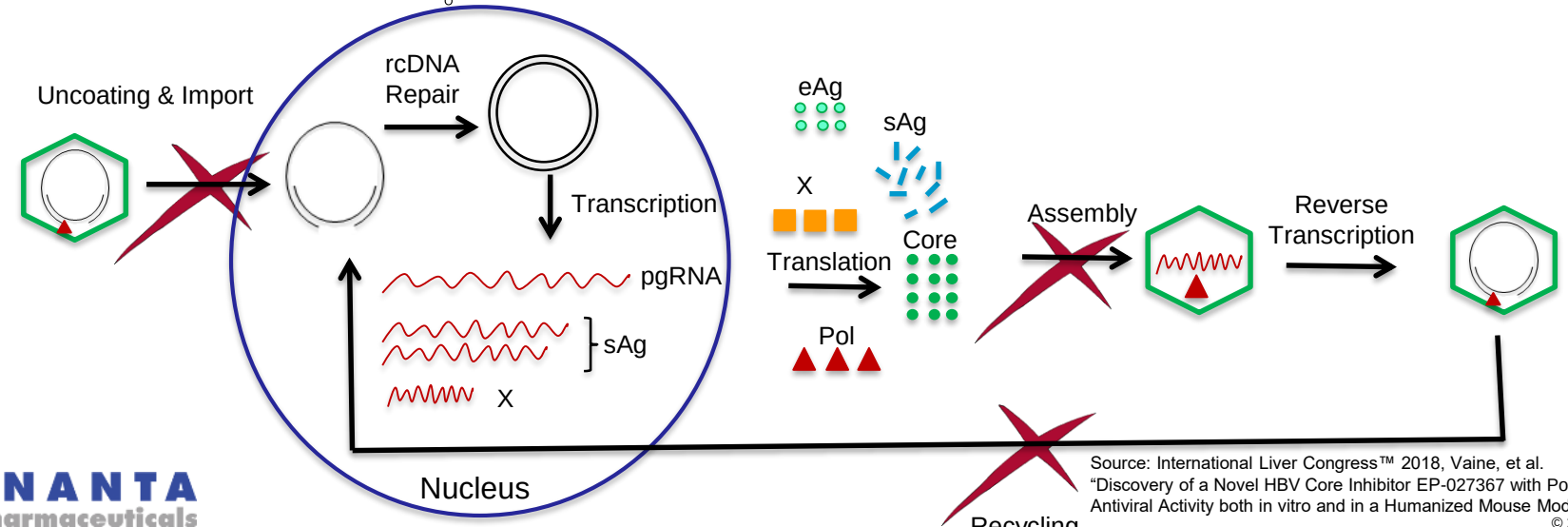


Sulfamoylbenzamides (SBA-R01)



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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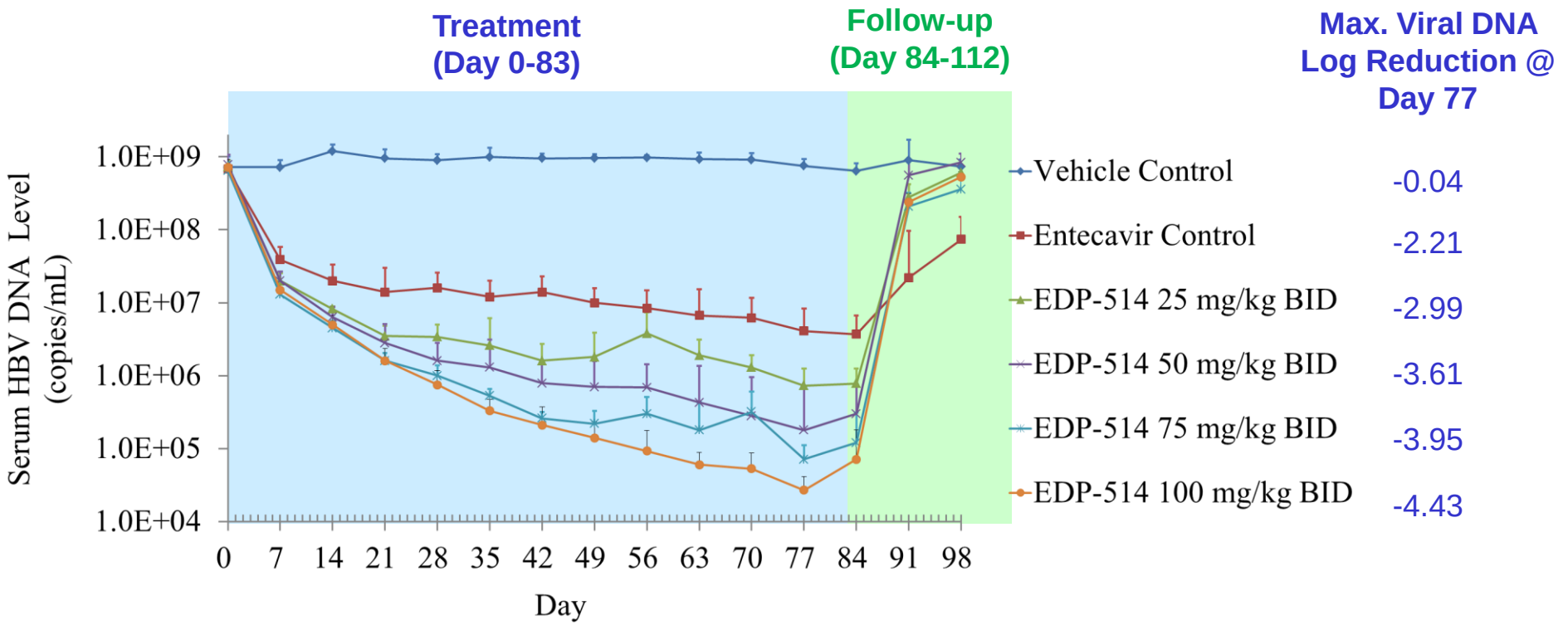
EDP-514 is Efficacious in the Humanized Liver Mouse Model

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



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HBV Core Inhibitor EDP-514 Summary

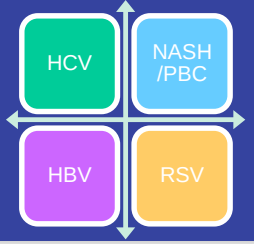
- A novel core inhibitor that displays potent anti-HBV activity at multiple points in the HBV lifecycle
- *In vitro*:
  - Potent anti-HBV activity in HBV expressing stable cells lines
  - Capable of preventing the establishment of cccDNA
  - Potent pan-genotypic activity
- *In vivo*:
  - Favorable tolerability and pharmacokinetic profile
  - Over 4-log reduction in HBV viral titers with 12 weeks of treatment in a chimeric liver mouse model
- Ph1 start targeted for 2H19

Financial Highlights

($ In millions)	Fiscal Year Ended Sept. 30, 2018	Fiscal 4Q18
Total Revenues	206.6*	$67.2
R&D Expenses	$94.9	$26.9
G&A Expenses	$23.4	$5.8
Net Income	$71.9	$27.4
EPS (per diluted share)	$3.48	$1.30
Balance Sheet		
Cash, Cash Equivalents and Marketable Securities	$325.1	$325.1

* Includes $15M milestone payment from AbbVie for reimbursement approval of MAVIRET™ in Japan

Key Catalysts



- Ongoing double-digit HCV royalties from glecaprevir (MAVYRET™)
- RSV program:
  - Phase 2a human challenge study data in calendar mid-2019
- FXR agonist EDP-305 for NASH / PBC:
  - Phase 2 data in NASH in 3Q19
  - Identify follow-on FXR clinical candidate for NASH in 2019
  - Advance non-FXR compounds for NASH
  - Continued PBC enrollment in 2019
- HBV program
  - Initiate Phase 1 with Core Inhibitor EDP-514 in 2H19

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