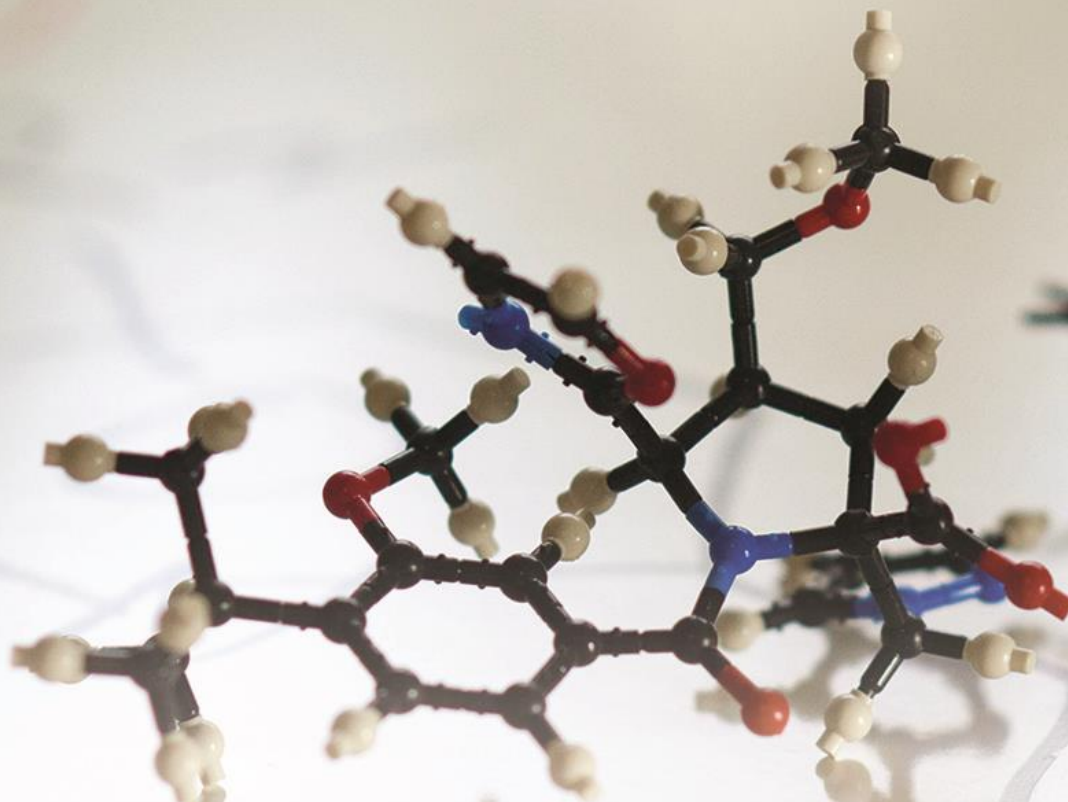


Corporate Presentation

SEPTEMBER, 2018



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.

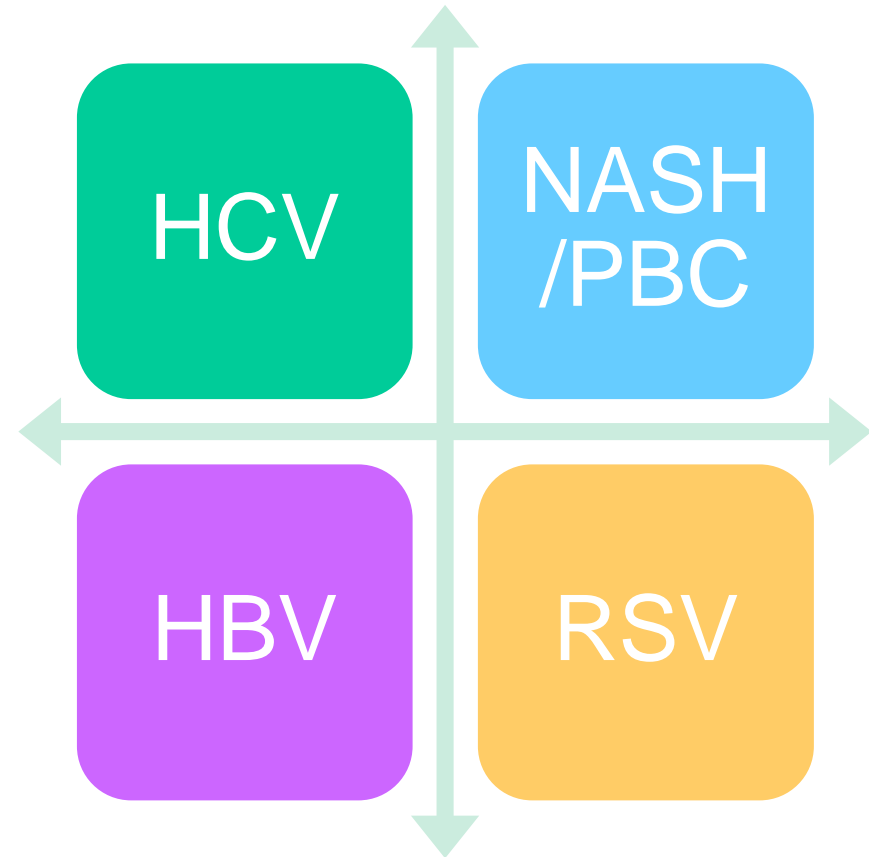
Please refer to these and other risk factors described or referred to in “Risk Factors” in Enanta’s most recent Form 10-K, and other periodic reports filed with the Securities and Exchange Commission. Enanta cautions investors not to place undue reliance on the forward-looking statements contained in 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 3Q18 royalties on HCV regimens: \$57 million
- Three clinical-stage programs in areas of high unmet medical need:
 - NASH: Phase 2 “ARGON-1” study ongoing
 - PBC: Phase 2 “INTREPID” study ongoing
 - RSV: Phase 1 dosing complete
- Ongoing R&D programs in NASH/PBC, HBV and RSV
- Strong balance sheet to fund clinical programs and other R&D efforts
 - Approx. \$295.5M in cash at 6/30/18

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						
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						
HBV	Core Inhibitor							
NASH	FXR Agonist Follow-on							
NASH	Undisclosed							



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****

HEPATITIS

**DOES NOT DISCRIMINATE.
IT AFFECTS MILLIONS
AND CAUSES LIVER CANCER.**

Talk to your doctor about testing. Early detection saves lives.

 www.cdc.gov/knowmorehepatitis 

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

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)

MAVYRET™
glecaprevir/pibrentasvir
100 mg/40 mg tablets

Our HCV Economics

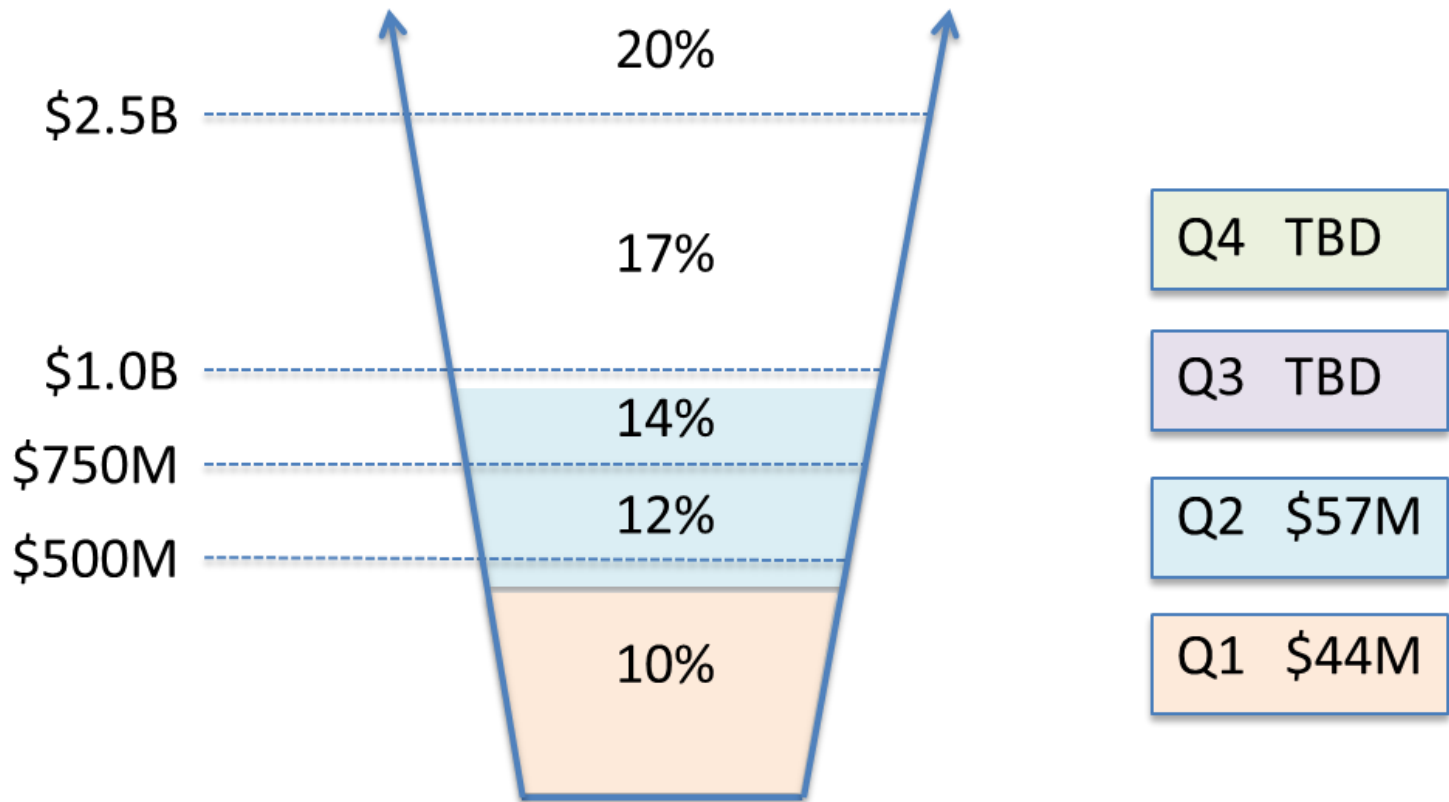
Product	Regimen	Enanta Asset	Economics
 <p>MAVYRET glecaprevir/pibrentasvir 100 mg/40 mg tablets</p>	2-DAA (ABBV)	glecaprevir (PI)	Double-digit royalty on 50% of net sales
 <p>viekira pak[®] ombitasvir, paritaprevir and ritonavir tablets; dasabuvir tablets</p>	3-DAA (ABBV)	paritaprevir (PI)	Double-digit royalty on 30% of net sales
 <p>viekirax[™] ombitasvir / paritaprevir / ritonavir tablets</p>	2-DAA (ABBV)	paritaprevir (PI)	Double-digit royalty on 45% of net sales

Our HCV Royalties

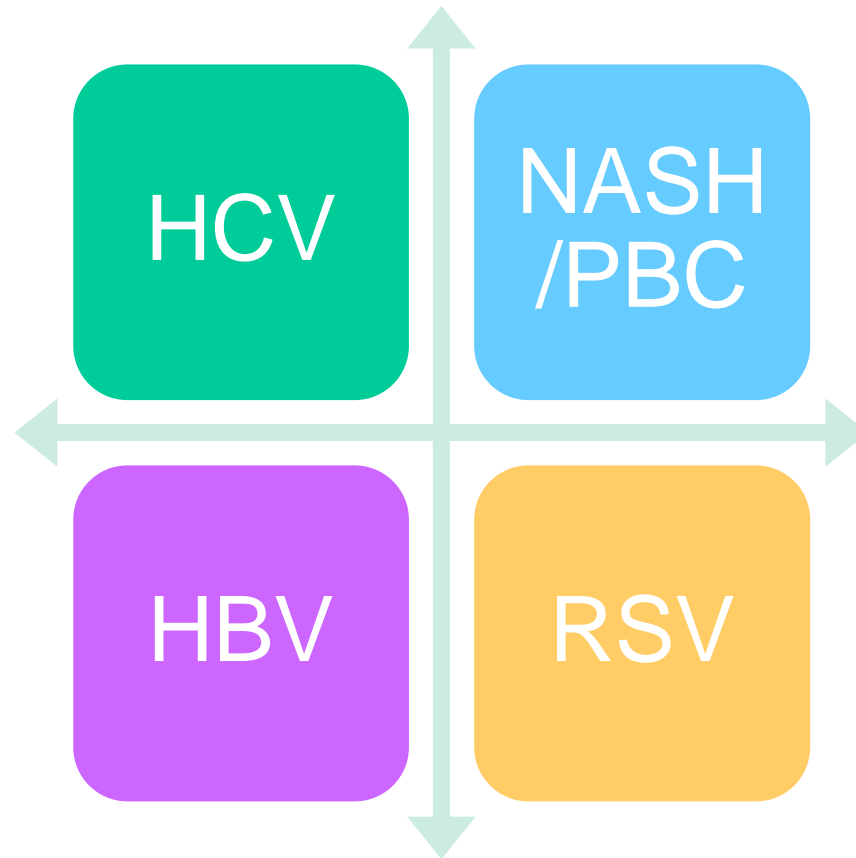
glecaprevir sales
(50% of Mavyret net sales)

royalty rate
(annual)

calendar 2018
royalties



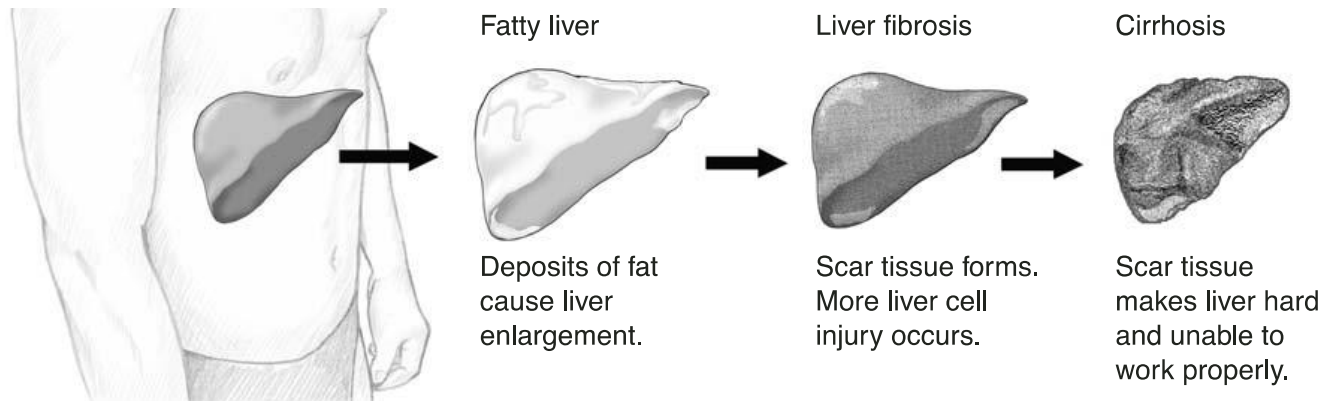
Virology & Liver Disease Focus Areas



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

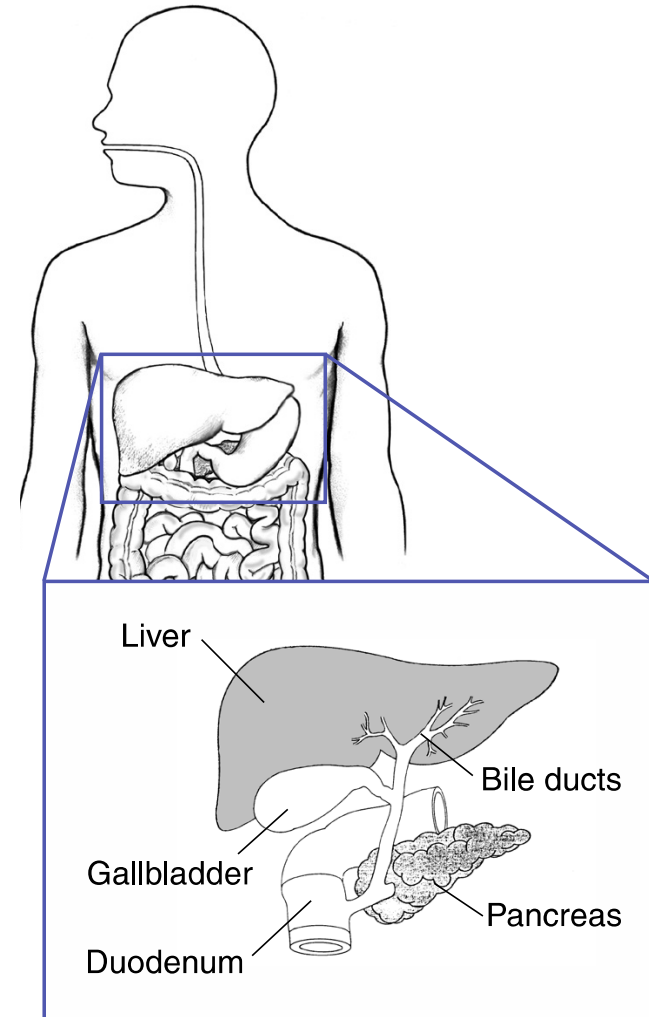
- Number one cause of liver disease in Western Countries
- NAFLD: **excessive fat (triglyceride)** accumulation in the liver (steatosis)
- A subgroup of NAFLD patients has **liver cell injury and inflammation** in addition to **excessive fat** (steatohepatitis), *i.e.* NASH
- NASH is associated with 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)



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
- US prevalence estimated to be 3%-5% (~9 to 15 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 US 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
- Significant potential add-on value beyond NASH

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

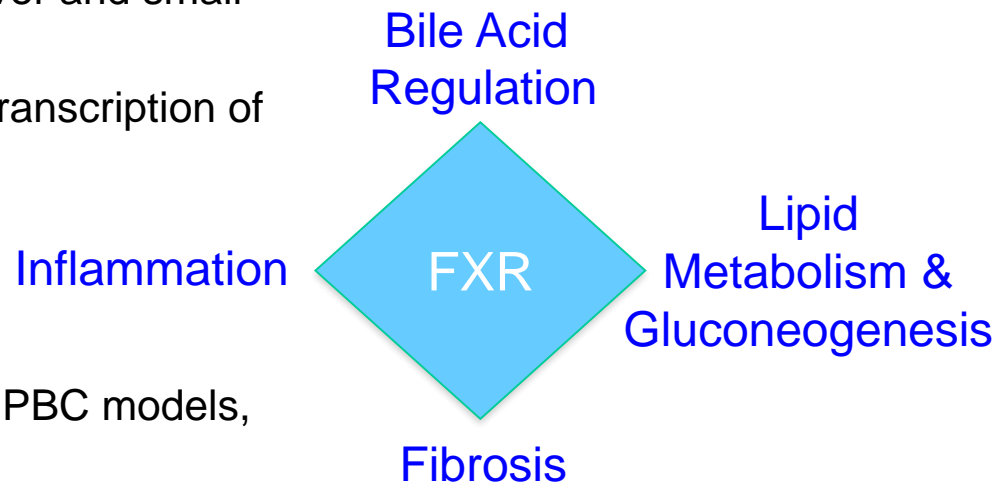
- FXR

- 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

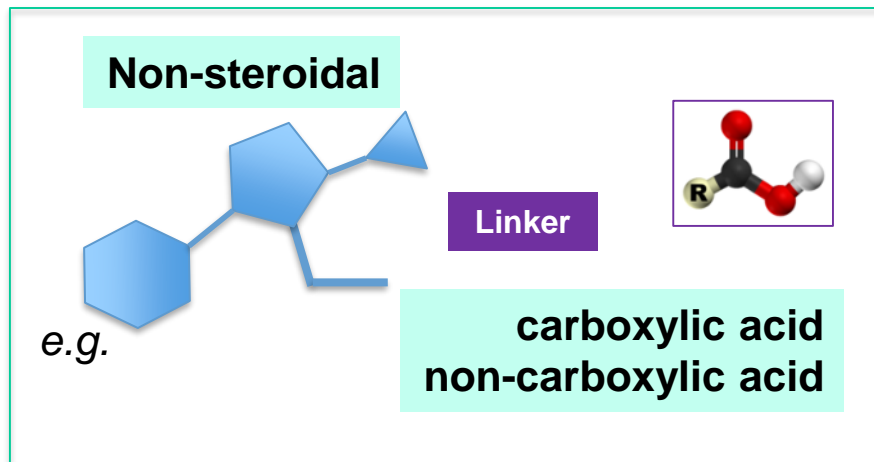
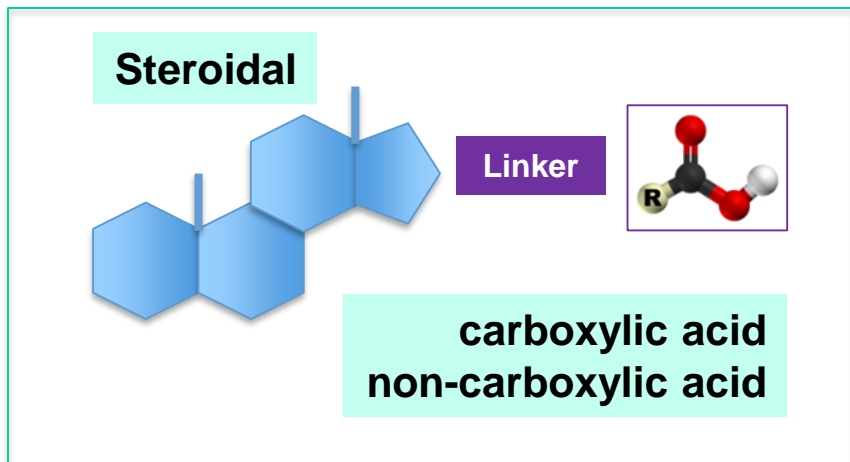
- FXR agonist preclinical PoC

- 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)



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



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

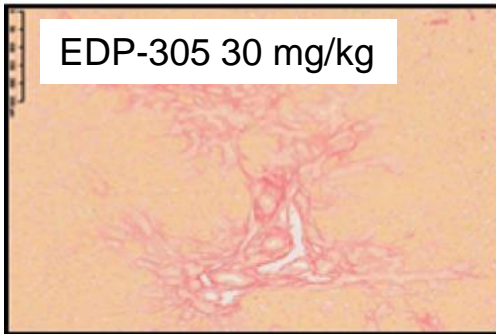
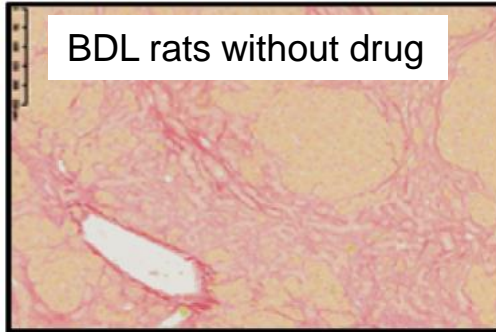
* Exact structure not disclosed

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
 - STAMTM 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

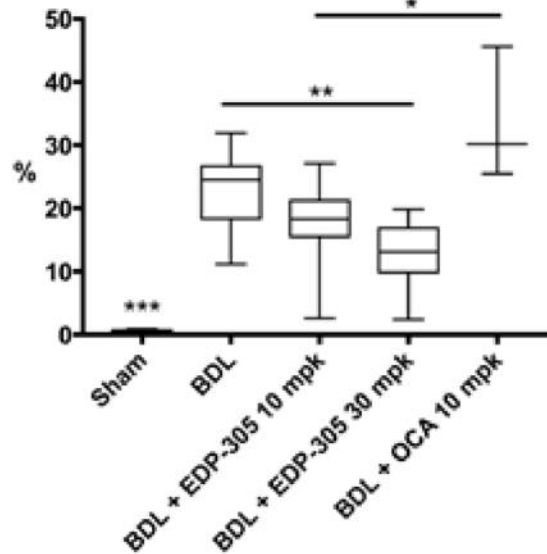
EDP-305 Treated Rats Have Less Fibrosis than BDL Only and OCA Groups

Histological Analysis



Collagen Proportional Area

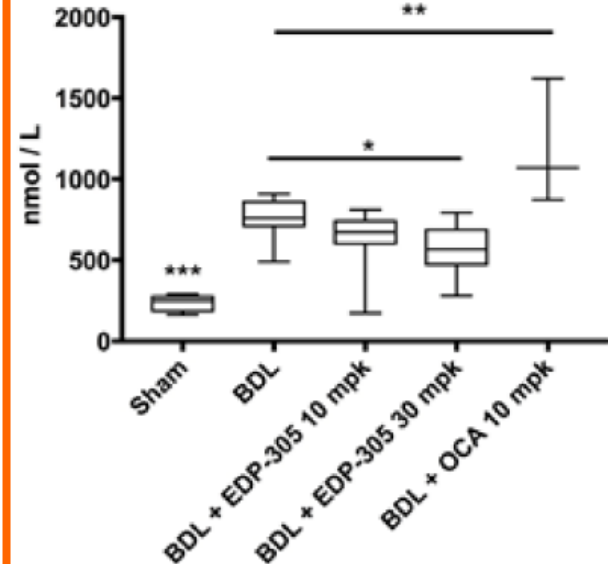
Analysis of Sirius Red Staining
"Image J open source software"



* P<0.05; ** P<0.01; ***P<0.001
OCA dosed at 10 mg/kg, toxicity at 30 mg/kg

Biochemical Analysis

Hydroxyproline



* P<0.05; ** P<0.01; ***P<0.001

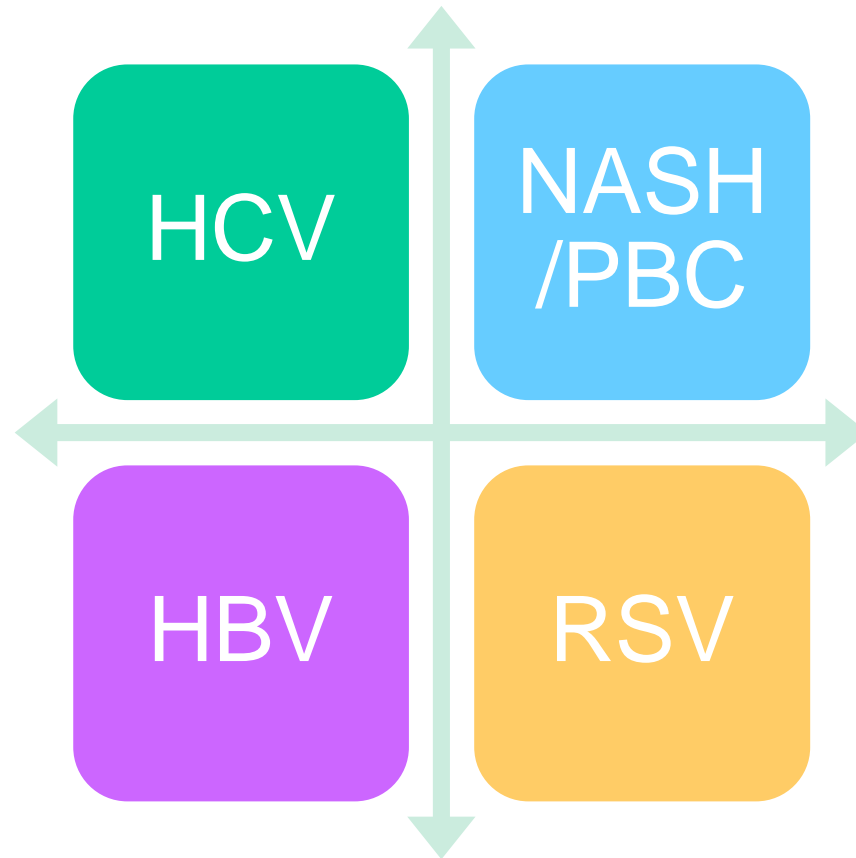
EDP-305 Phase 1 Study

- 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:
 - “INTREPID” (PBC) and ARGON-1 (NASH)
 - 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 Paramyxoviridae
- 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

- Non-Fusion approach directly targets virus replication
 - N-protein inhibitor
- 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 dosing complete

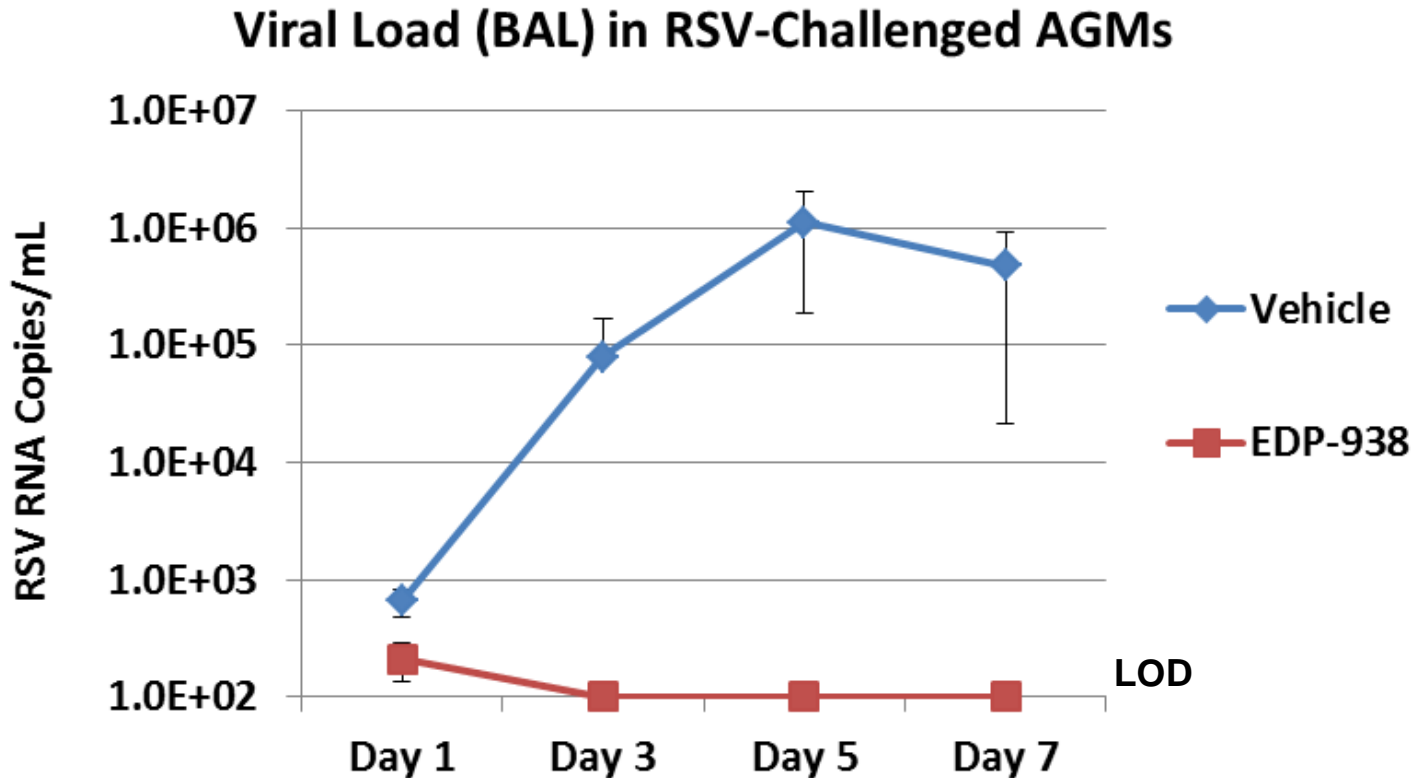
EDP-938 Presents a High Barrier to Resistance and No Cross-Resistance to Other RSV Inhibitors

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	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 can only be selected with EDP-938 starting at low concentration of the drug (1xEC₅₀) followed by a slow increase to 16xEC₅₀ after multiple passages
- Selection with higher concentration of the drug results in elimination of the virus rather than development of resistance
- The level of resistance (fold increase in EC₅₀) with EDP-938 is much lower compared to those with fusion and L inhibitors
- There is no cross-resistance between EDP-938 and other RSV inhibitors

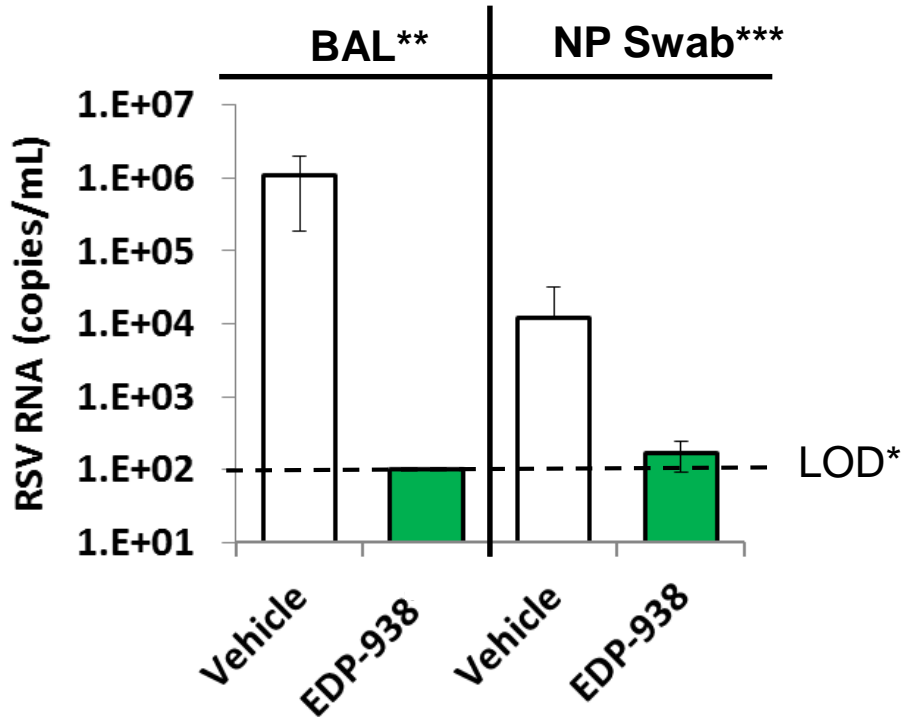
EDP-938 Dramatically Reduces Viral Load in BAL (Bronchoalveolar Lavage) Fluid

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

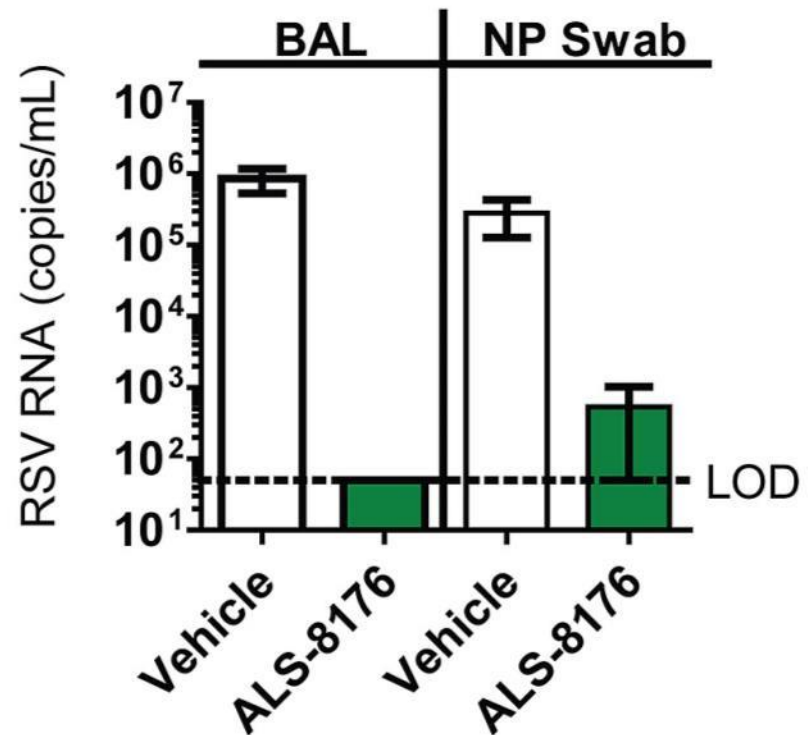


100 mg/kg BID of EDP-938 or vehicle control was given 24h prior to infection (day -1), on the day of infection (day 0), and for days 1-4

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



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



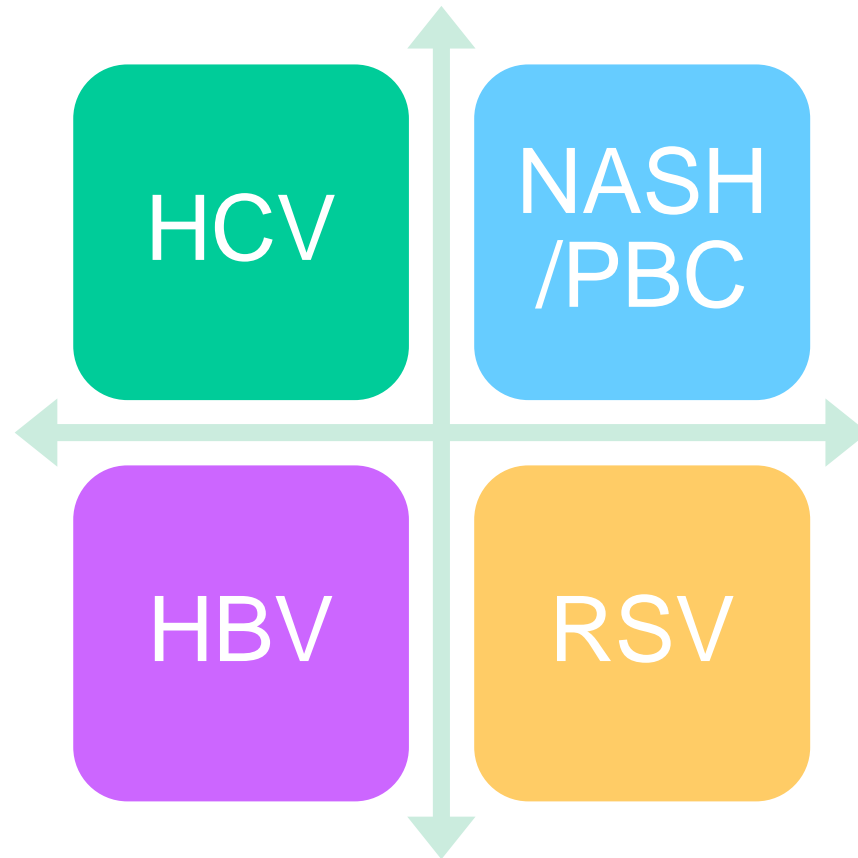
Loading dose of 200 mg/kg ALS-8176 given 24h prior to infection, followed by 50 mg/kg BID on the day of infection, and for 4 additional days. (Deval *et. al.*, *PLoS Pathogens* 2015)

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

EDP-938 RSV Summary

- EDP-938 is the only N-inhibitor under clinical evaluation (Phase 1)
 - Preliminary Phase 1 results demonstrate EDP-938 was generally safe and well tolerated
 - Final data to be presented at the 11th International Respiratory Syncytial Virus Symposium, Oct. 31 – Nov. 4, 2018
- Phase 2a Human Challenge Trial start anticipated in 4Q18
 - Randomized, double-blind placebo-controlled trial in healthy adult volunteers infected with attenuated RSV virus to assess efficacy and dose selection for future trials
- 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



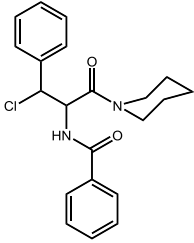
HBV Program: Summary

- Emphasis on developing “best-in-class” core inhibitor
 - Also exploring additional mechanisms with goal of a functional cure
- Enanta core inhibitors currently in advanced stages of pre-clinical lead optimization
 - Clinical validation for mechanism established by Novira and JNJ
- **Goal:** Clinical candidate selection in 2018

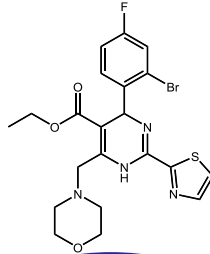
Core inhibitors: Introduction

- Core inhibitors (capsid assembly modulators, core protein allosteric modulators, capsid inhibitors) are a novel class of replication inhibitor that have been shown to act at multiple steps in the HBV lifecycle
 - Prevents proper uncoating, nuclear import, assembly, and recycling

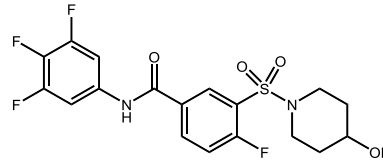
Phenylpropemides
(AT-130)



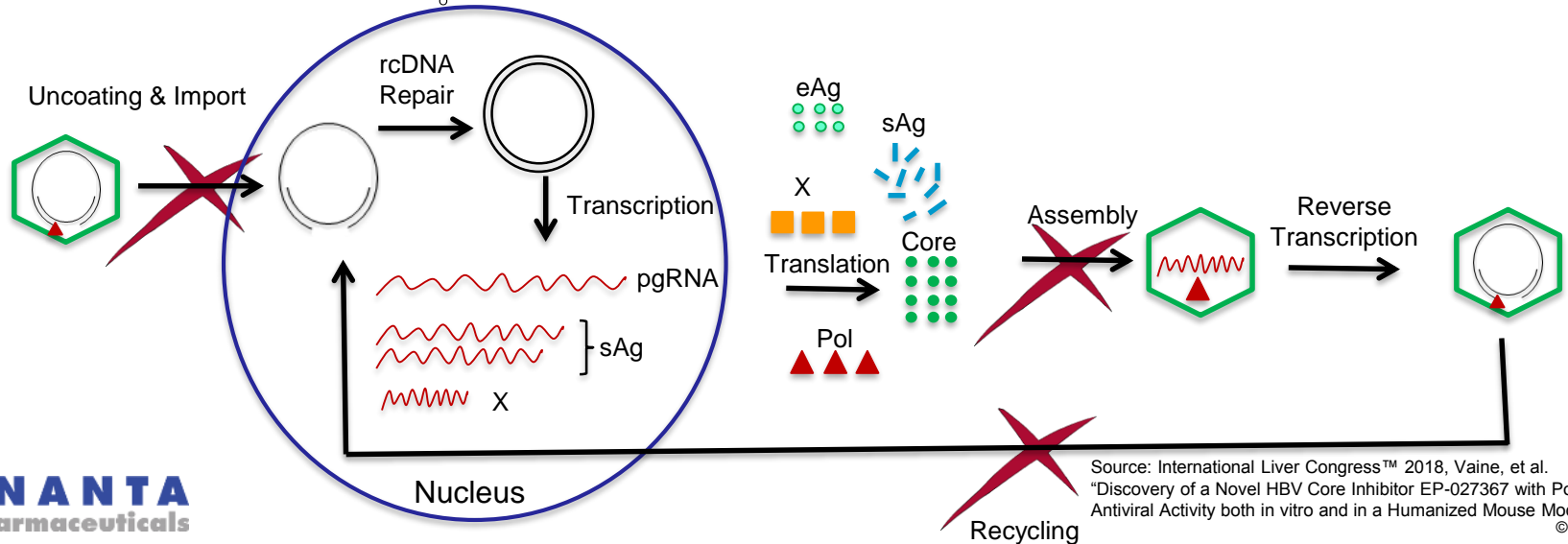
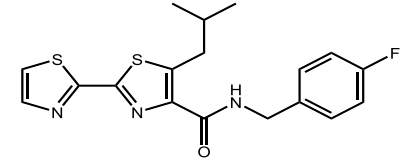
HAPs
(GLS4)



Sulfamoylbenzamides
(SBA-R01)



Isothiafludine
(NZ-4)



Prototype Core Inhibitor EP-027367 is a Potent Inhibitor of HBV Replication

- EP-027367 is active in multiple HBV stable cell lines

	HBV Stable Cell Line EC ₅₀ (nM)		
	HepAD38	HepDE19	HepG2.2.15
Encapsidated pgRNA	32	53	28
Intracellular Viral DNA	24	40	20
Secreted Viral DNA	34	54	30
HBeAg	57	47	>200
HBsAg	>200	>200	>200

Encapsidated pgRNA measured by modified pulldown and qPCR

Viral DNA measured by qPCR

HBeAg & HBsAg measured by commercial ELISA kit

Prototype Core Inhibitor EP-027367 vs. Competitors: In Vitro Potency

	HBV Intracellular Viral DNA EC ₅₀ (nM)		
	HepAD38	HepDE19	HepG2.2.15
EP-027367 ¹	24	40	20
NVR 3-778 ²			240
JNJ-379 ³			69
ABI-H0731 ⁴	253 (172)		
ABI-H2158 ⁵	23		
AB-423 ^{4,5}		262/340	146
AB-506 ⁵		40	40
RG-7049389 ¹			6.1

Source of data: ¹EASL 2018, ²AASLD 2014, ³AASLD 2016, ⁴EASL 2016, ⁵AASLD 2017

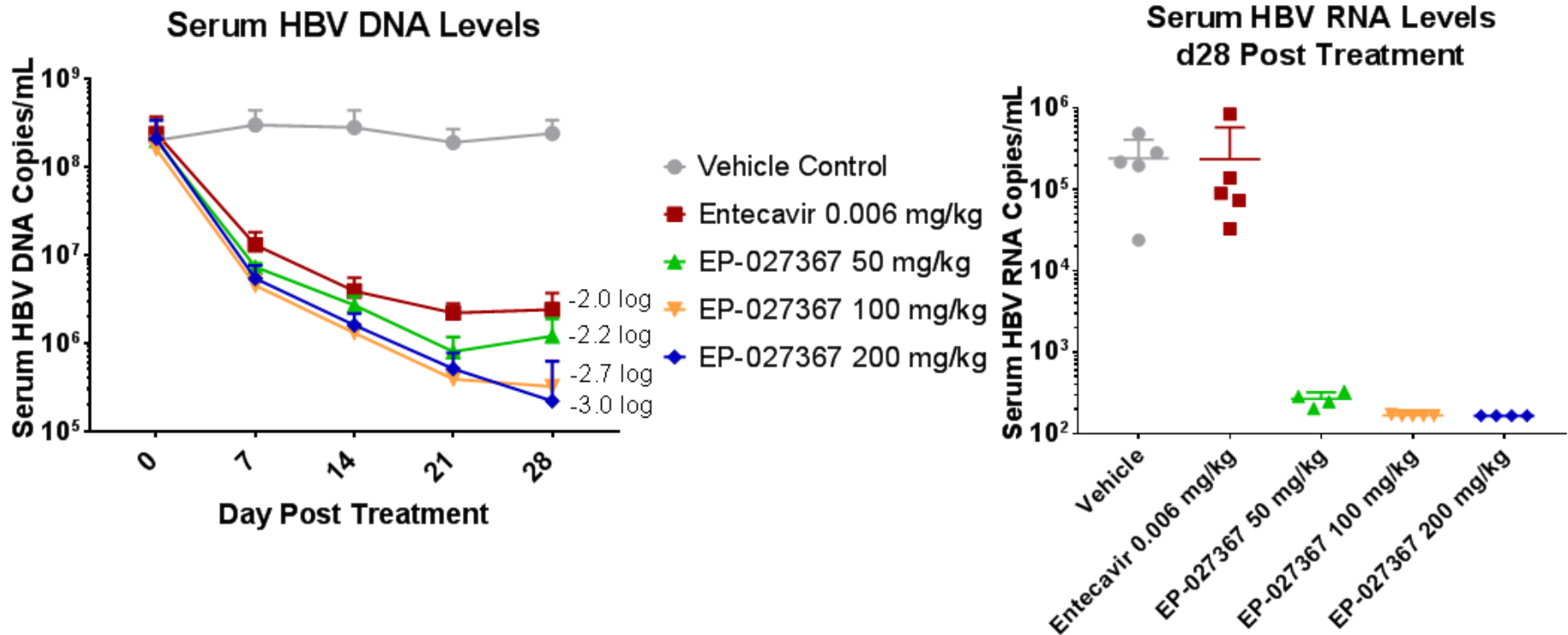
Prototype Core Inhibitor EP-027367 vs. Competitors: Genotype Coverage

Genotype (GT)	EC ₅₀ (nM)						
	EP-027367 ¹	NVR3-778 ²	JNJ-379 (median) ³	ABI-H0731 ^{3,4}	ABI-H2158 ⁴	AB-423 ^{4,5}	AB-506 ⁴
GT-A	11	380	20	113/39	0.6	57/89	8/23
GT-B	9	200	12	86/67	11	39/91	17/20
GT-C	7	340	11	88/31	7.1	52/55	15/9
GT-D	12	430	23	142		195	40
GT-E	10	580	33				
GT-F	34	560	10				
GT-G	24	250	11				
GT-H	21	580	19				

Source of data: ¹EASL 2018, ²AASLD 2015, ³AASLD 2016, ⁴AASLD 2017, ⁵EASL 2016

EP-027367 is Efficacious in a uPA/SCID Humanized Liver Mouse Model

- uPA/SCID mice were infected with genotype C HBV and subsequently treated with EP-027367 BID at indicated doses for 28 days



- EP-027367 is the first reported core inhibitor to achieve a 3-log reduction in circulating HBV DNA and RNA levels as a monotherapy

Prototype EP-027367 vs. Competitors uPA/SCID Humanized Liver Mouse Model

Compound	Dose	HBV DNA Reduction from Baseline after 4 Weeks
EP-027367	50 mg/kg BID, PO	2.2 log
	100 mg/kg BID, PO	2.7 log
	200 mg/kg BID, PO	3.0 log
NVR3-378 ¹	405 mg/kg BID, PO	~2 log
AB-423 ²	100 mg/kg BID, PO	~1 log
JNJ-632 ³	200 mg/kg QD, SC	2.77 log

¹ Klumpp et al. EASL 2015

² Mani et al. EASL 2016

³ Berke et al. 2016 International HBV Meeting

HBV Core Inhibitor Summary

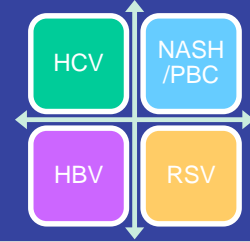
- A novel core inhibitor that displays potent anti-HBV activity at multiple points in the HBV lifecycle
- *In vitro* EP-027367 demonstrated:
 - Potent anti-HBV activity in HBV expressing stable cells lines
 - Capable of preventing the establishment of cccDNA
 - Pan-genotypic activity
 - Activity against known nucleos(t)ide resistant variants
 - Additive to synergistic activity with nucleoside analogs and other core inhibitors
- *In vivo* EP-027367 demonstrated:
 - Favorable tolerability and pharmacokinetic profile
 - Up to 3 log reduction in HBV viral titers with 4 weeks of treatment in a chimeric liver mouse model
- *In vitro* and *in vivo* profiling of EP-027367 and other Enanta core inhibitors ongoing with goal of nominating finalist clinical candidate in 2018

Financial Highlights

(\$ In millions)	Fiscal Year Ended Sept. 30, 2017	Fiscal 3Q18
Total Revenues	102.8*	\$57.3
R&D Expenses	\$57.5	\$28.5
G&A Expenses	\$20.7	\$6.1
Net Income	\$17.7	\$20.3
EPS (per diluted share)	\$0.91	\$0.97
Balance Sheet		
Cash, Cash Equivalents and Marketable Securities	\$293.7	\$295.5

* Includes \$65M in milestone payments from AbbVie for the U.S. and EU commercialization regulatory approval of MAVYRET™ and MAVIRET™, respectively

Key Catalysts



- Ongoing double-digit royalties from glecaprevir (MAVYRET™) and paritaprevir (VIEKIRA™)
- RSV program:
 - Final Phase 1 data in calendar 4Q18
 - Advance to Phase 2a human challenge study in calendar 4Q18
- FXR agonist EDP-305 for NASH / PBC:
 - Phase 2 data in PBC and NASH in 2019
 - Advance follow-on FXR compounds & non-FXR compounds for NASH
- HBV program:
 - Targeting clinical candidate selection in 2018

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