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 2017 royalties on HCV regimens: $38 million

• Three clinical-stage programs in areas of high unmet medical need:
  - PBC: Phase 2 ongoing
  - NASH: Phase 2 to begin in 1Q18
  - RSV: Phase 1 ongoing

• Ongoing R&D programs in NASH/PBC, HBV and RSV

• Strong balance sheet to fund clinical programs and other R&D efforts
  - Approx. $294M in cash at 9/30/17

*VIEKIRA regimens include AbbVie’s marketed HCV regimens of VIEKIRA PAK®, VIEKIRA XR, TECHNIVIE®, 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

<table>
<thead>
<tr>
<th>Product Candidate</th>
<th>Discovery</th>
<th>Preclin</th>
<th>Phase 1</th>
<th>Phase 2</th>
<th>Phase 3</th>
<th>Market</th>
</tr>
</thead>
<tbody>
<tr>
<td>HCV</td>
<td>Protease Inhibitor</td>
<td></td>
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<tr>
<td></td>
<td>glecaprevir – containing pan-genotypic 2-DAA combo</td>
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</tr>
<tr>
<td>HCV</td>
<td>Protease Inhibitor</td>
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</tr>
<tr>
<td></td>
<td>paritaprevir – containing regimens</td>
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<tr>
<td>PBC</td>
<td>FXR Agonist</td>
<td>EDP-305</td>
<td>Ph2 “INTREPID”</td>
<td></td>
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<td></td>
</tr>
<tr>
<td>NASH</td>
<td>FXR Agonist</td>
<td>EDP-305</td>
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<tr>
<td>RSV</td>
<td>N-protein Inhibitor</td>
<td>EDP-938</td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>HBV</td>
<td>Core Inhibitor</td>
<td></td>
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</tr>
<tr>
<td>NASH</td>
<td>FXR Agonist Follow-on</td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>NASH</td>
<td>Undisclosed</td>
<td></td>
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</tr>
</tbody>
</table>
HCV Market

- Market for HCV therapies:
  - Approx. $19B for 2016

- 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****

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

Source: www.cdc.gov
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)

*sold as MAVYRET™ in the U.S., MAVIRET™ outside the U.S.
## Our HCV Economics

<table>
<thead>
<tr>
<th>Product</th>
<th>Regimen</th>
<th>Enanta Asset</th>
<th>Economics</th>
</tr>
</thead>
<tbody>
<tr>
<td>MAVYRET glecaprevir/pibrentasvir</td>
<td>2-DAA (ABBV)</td>
<td>glecaprevir (PI)</td>
<td>Double-digit royalty on 50% of net sales</td>
</tr>
<tr>
<td>viekira pak, viekira XR</td>
<td>3-DAA (ABBV)</td>
<td>paritaprevir (PI)</td>
<td>Double-digit royalty on 30% of net sales</td>
</tr>
<tr>
<td>technivie</td>
<td>2-DAA (ABBV)</td>
<td>paritaprevir (PI)</td>
<td>Double-digit royalty on 45% of net sales</td>
</tr>
</tbody>
</table>
Virology & Liver Disease Focus Areas

- HCV
- NASH/PBC
- HBV
- RSV
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, progression to NASH dramatically increases risks of cirrhosis, liver failure, and hepatocellular carcinoma

Stages of Liver Injury (NIDDK)

- Fatty liver: Deposits of fat cause liver enlargement.
- Liver fibrosis: Scar tissue forms. More liver cell injury occurs.
- Cirrhosis: Scar tissue makes liver hard and unable to work properly.
Primary Biliary Cholangitis (PBC)

- Bile is a digestive liquid made in the liver that travels through bile ducts to the small intestine, where it helps in digestion.
- PBC is a chronic inflammatory liver disease that slowly destroys bile ducts, causing bile to remain in the liver, leading to liver cell damage and cirrhosis.
- As cirrhosis progresses and liver scar tissue increases, the liver loses its ability to function, leading to potential liver failure, liver transplantation, or hepatocellular carcinoma.
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 is a nuclear receptor and main regulator of bile acid levels in liver and small intestine
- FXR responds to bile acids by regulating transcription of key enzymes and transporters
- FXR agonists have ameliorated a number of the pathologies in NASH and PBC models, including an effect on fibrosis
- Clinical validation has been achieved in NASH and PBC with the FXR agonist 6-ECDCA (OCA)

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

<table>
<thead>
<tr>
<th>Steroidal carboxylic acid</th>
<th>S-CA</th>
<th>OCA, bile acids</th>
</tr>
</thead>
<tbody>
<tr>
<td>Steroidal non-carboxylic acid</td>
<td>S-NCA</td>
<td>Enanta compounds</td>
</tr>
<tr>
<td>Non-steroidal carboxylic acid</td>
<td>NS-CA</td>
<td>Enanta compounds, GS-9674*, LJN452</td>
</tr>
<tr>
<td>Non-steroidal non-carboxylic acid</td>
<td>NS-NCA</td>
<td>Enanta compounds</td>
</tr>
</tbody>
</table>

* 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
  - 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 Reduces Fibrosis Progression by MR Imaging of BDL (bile duct ligation) Rats

Day 0

BDL Surgery

Day 4

FXR Agonist Starts

EDP-305 10, 30 mg/kg QD
OCA 10, 30 mg/kg QD

Day 17 – 18

Image

Study compounds blinded to investigators

- A gadolinium-based collagen targeted probe was used for MR imaging of liver fibrosis
EDP-305 Treated Rats Have Less Fibrosis than BDL Only and OCA Groups

Histological Analysis

BDL rats without drug

EDP-305 30 mg/kg

OCA 10 mg/kg

Collagen Proportional Area

Analysis of Sirius Red Staining
“IImage J open source software”

Biochemical Analysis

Hydroxyproline

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

* P<0.05; ** P<0.01; ***P<0.001
EDP-305 Phase 1: Study Design

- 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 and pharmacokinetics

- Explored specific markers of FXR activity: C4, FGF19
EDP-305 Phase 1: Key Results

• FXR target engagement demonstrated with elevations of FGF19 and diminutions in C4
  - Doses > 1 mg increasing FGF19 and reducing C4 in all subjects
  - PN subjects even more sensitive with effects observed in both parameters at lowest multiple doses of 0.5 and 1 mg

• PK profile suitable for once daily oral dosing
  - Dose proportional increases in exposure were observed
  - Median \( t_{1/2} \) 11-23 hr in healthy and 10-18 hr in PN subjects in MAD
  - Longer \( t_{1/2} \) (2-fold, high end of range) and more drug accumulation (~3 to 4-fold) were observed following the multiple 20 mg dose compared to lower doses
No SAEs reported, generally well tolerated at all doses tested

• Treatment-emergent AEs in ≥ 2 EDP-305 treated subjects in MAD: headache and pruritus in healthy subjects, and constipation and pruritus in PN subjects
  - Pruritus (9% for EDP-305, 3% in placebo), majority were mild or moderate and occurred at MAD 20 mg (no cases below 10 mg). Notably, EDP-305 demonstrated potent engagement of the FXR receptor across the lower dose range where there was no pruritus.
  - Two discontinuations in MAD 20 mg: n=1 transient grade 2 ALT/AST elevation, and n=1 moderate pruritus

• No dose-related changes in lipids in healthy subjects at any dose
• No dose-related changes in lipids in PN subjects except at MAD 20mg (reductions of total cholesterol and HDL, with no concomitant increase in LDL)
• Significant elevations of FGF19 and diminutions in C4 demonstrated potent engagement of the FXR receptor at doses that neither elicit adverse effects on lipids nor result in itch
FXR Agonist EDP-305: Summary

- Phase 1 study results after once-daily oral dosing support further evaluation in NASH and PBC
- Fast Track Designation granted by FDA for PBC and for NASH with fibrosis
- Phase 2 study “INTREPID” in PBC initiated in December 2017
  - 12 week dose ranging, randomized, double-blind, placebo-controlled
  - Evaluates safety, tolerability, PK, and efficacy (alk. phos. reduction)
  - Uses new tablet formulation at 1 and 2.5 mg
  - New tablet formulation results in approximately two-fold greater drug exposure than suspension formulation used in Phase 1 SAD/MAD studies
- Phase 2 study in NASH expected to initiate early 2018
Virology & Liver Disease Focus Areas

- HCV
- NASH/PBC
- HBV
- RSV
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
- Phase1 initiated in December 2017
EDP-938 Presents a High Barrier to Resistance and No Cross-Resistance to Other RSV Inhibitors

<table>
<thead>
<tr>
<th>Compounds</th>
<th>wt RSV EC₅₀ (nM)</th>
<th>Drug Resistant (R) Virus</th>
<th></th>
<th></th>
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<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>EDP-938 R EC₅₀ (nM)</td>
<td>Fold Change</td>
<td>AZ-27 R EC₅₀ (nM)</td>
<td>Fold Change</td>
<td>GS-5806 R EC₅₀ (nM)</td>
<td>Fold Change</td>
<td></td>
</tr>
<tr>
<td>EDP-938</td>
<td>53 ± 5</td>
<td>250 ± 53</td>
<td>5</td>
<td>68 ± 8</td>
<td>&lt;100</td>
<td>250 ± 53</td>
<td>5 &lt; 2</td>
<td></td>
</tr>
<tr>
<td>AZ-27 (L inhibitor)</td>
<td>19 ± 2</td>
<td>29 ± 5</td>
<td>2</td>
<td>&gt;20,000</td>
<td>&gt;1,060</td>
<td>29 ± 5</td>
<td>2 &lt; 2</td>
<td></td>
</tr>
<tr>
<td>GS-5806 (F inhibitor)</td>
<td>5 ± 0.4</td>
<td>2 ± 0.6</td>
<td>0.4</td>
<td>6 ± 0.3</td>
<td>&gt;20,000</td>
<td>2 ± 0.6</td>
<td>0.4 &gt; 2</td>
<td></td>
</tr>
</tbody>
</table>

- Resistant virus can only be selected with EDP-938 starting at low concentration of the drug (1×EC₅₀) followed by a slow increase to 16×EC₅₀ 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)
  - Initiated standard single-ascending dose (SAD) and multiple-ascending dose (MAD) studies to evaluate safety and pharmacokinetics
- Phase 2a Human Challenge Trial anticipated in 2H18
  - 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, provided appropriate safety studies support advancement
- 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

HCV
NASH/PBC
HBV
RSV
• 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

Sources: WHO, CDC, Datamonitor
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
## Financial Highlights

<table>
<thead>
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<th>($ In millions)</th>
<th>Fiscal Year Ended Sept. 30, 2017</th>
<th>Fiscal 4Q17</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total Revenues</td>
<td>102.8*</td>
<td>$75.9*</td>
</tr>
<tr>
<td>R&amp;D Expenses</td>
<td>$57.5</td>
<td>$16.5</td>
</tr>
<tr>
<td>G&amp;A Expenses</td>
<td>$20.7</td>
<td>$5.1</td>
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<tr>
<td>Net Income</td>
<td>$17.7</td>
<td>$36.5</td>
</tr>
<tr>
<td>EPS (per diluted share)</td>
<td>$0.91</td>
<td>$1.86</td>
</tr>
</tbody>
</table>

**Balance Sheet**

| Cash, Cash Equivalents and Marketable Securities | $293.7 | $293.7 |

* Includes $65M in milestone payments from AbbVie for the U.S. and EU commercialization regulatory approval of MAVYRET™ and MAVIRET™, respectively. Does not include $15M milestone payment earned from AbbVie in Nov. 2017 for MAVIRET™ commercialization approval in Japan.
Key Catalysts

- Ongoing double-digit royalties from glecaprevir (MAVIRET™) and paritaprevir (VIEKIRA™)

- FXR agonist EDP-305 for NASH / PBC:
  - Phase 2 start in NASH in early 2018
  - Phase 2 data in PBC and NASH in 2019
  - Advance follow-on FXR compounds & non-FXR compounds for NASH

- RSV program:
  - Complete Phase 1 and advance to Phase 2a human challenge study in 2018

- HBV program:
  - Targeting clinical candidate selection in 2018
Creating Small Molecule Drugs for Viral Infections and Liver Diseases

www.enanta.com