CREATING SMALL MOLECULE DRUGS
FOR VIRAL INFECTIONS AND LIVER DISEASES

Corporate Overview
January 11, 2021
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 research and development programs, our business 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. These forward-looking statements include, but are not limited to, statements about overall trends, royalty revenue trends, research and clinical development plans and prospects, liquidity and capital needs and other statements of expectations, beliefs, future plans and strategies, anticipated events or trends, and similar expressions. These forward-looking statements are 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 results and involve known and unknown risks, uncertainties and other factors that are in some cases beyond our control. As a result, any or all of our forward-looking statements in this presentation may turn out to be inaccurate.

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A Unique Approach to Drug Discovery

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

Robust Clinical Stage Pipeline

RSV: Phase 2b in adult patients (RSVP)
Phase 2b in adult stem cell transplant patients (RSVPTx)

NASH: Phase 2b (ARGON-2)
Phase 1b (Follow-on FXR agonist)

HBV: Two Phase 1b studies (core inhibitor)

Proven Track Record of Success

Glecaprevir – HCV protease inhibitor in MAVYRET®/MAVIRET®
$122M in fiscal 2020 royalties on HCV regimens

Strong Balance Sheet

Strong balance sheet and royalties to fund robust pipeline
$419M in cash at 9/30/20
Leveraging our core strength in Hepatitis C to become a leader in oral treatments for *viral* infections and *liver* diseases

Several new therapeutic areas with goal of building multiple approaches in each
## Enanta Pipeline

<table>
<thead>
<tr>
<th>PRODUCT CANDIDATE</th>
<th>DISCOVERY</th>
<th>PRECLINICAL</th>
<th>PHASE 1</th>
<th>PHASE 2</th>
<th>PHASE 3</th>
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<tbody>
<tr>
<td><strong>Virology</strong></td>
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<td>Glecaprevir-containing pan-genotypic 2-DAA combo</td>
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<td>N-Protein Inhibitor</td>
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<td>EDP-938</td>
<td>RSVPEDs in Q1 2021</td>
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<td>NUC-suppressed HBV patients</td>
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<td>EDP-514</td>
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<td>FXR Agonist</td>
<td>Viremic HBV patients</td>
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<td>EDP-721</td>
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<td>RNA Destabilizer</td>
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<td>ARGON-2</td>
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<td>COVID-19</td>
<td>SARS-CoV-2 Inhibitor</td>
<td>EDP-305</td>
<td>ARGON-2</td>
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<td><strong>Non-viral Liver Disease</strong></td>
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<td>NASH</td>
<td>FXR Agonist</td>
<td>EDP-297</td>
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<td></td>
<td>EDP-305</td>
<td>ARGON-2</td>
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<td></td>
<td>EDP-297</td>
<td>ARGON-2</td>
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<td><strong>Discovery or Preclinical</strong></td>
<td>RSV, HBV, NASH, other</td>
<td>EDP-305</td>
<td>ARGON-2</td>
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</tbody>
</table>

*Fixed-dose combination contains glecaprevir and AbbVie's NS5A inhibitor, pibrentasvir. Marketed as MAVYRET (U.S.) and MAVIRET (ex-U.S.)*
Leveraging our core strength in Hepatitis C to become a leader in oral treatments for viral infections and liver diseases.

Several new therapeutic areas with goal of building multiple approaches in each
Respiratory Syncytial Virus (RSV)

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

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)

### RSV at a Glance

<table>
<thead>
<tr>
<th>Children &lt; 5 years</th>
<th>Adults &gt; 65 years</th>
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<tbody>
<tr>
<td>33M global cases</td>
<td>177K US hospitalizations</td>
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<tr>
<td>3M global hospitalizations</td>
<td>120K global deaths</td>
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<tr>
<td>2.1M US outpatient visits</td>
<td>14K US deaths</td>
</tr>
</tbody>
</table>

EDP-938: N-Protein Inhibitor for RSV

• EDP-938 is the only N-inhibitor under clinical evaluation
  – Non-fusion approach directly targets viral replication vs. entry
  – Granted Fast Track Designation by FDA

• 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 virus variants resistant to other mechanisms
  – Robust *in vivo* efficacy data
EDP-938 Development Program

Phase 1 Results
- Safe and well tolerated, no SAEs, AEs were mild
- At Phase 2 doses mean trough levels 30x higher than EC90 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
  - Primary endpoint: Reduction in area under the curve (AUC) viral load in the intent-to-treat-infected population (ITT-I)
  - Secondary endpoint: Reduction in Total Symptom Score (TSS)
- 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
Robust Antiviral Effect
Rapid and Sustained Reduction in Viral Load in Both Active Arms Compared to Placebo (71%, 74% ↓ AUC; P<0.001)
Robust Symptom Reduction

Rapid and Sustained Attenuation of RSV Symptoms in Both Active Arms Compared to Placebo (68%, 74% ↓ AUC; P<0.001)
**Primary Objective:**
Effect of EDP-938 on progression of RSV infection by assessment of clinical symptoms measured over the 14-day study period

**Secondary Objective:**
Antiviral efficacy, safety, and PK of EDP-938

*Equivalent to 600mg suspension dosage form used in challenge study*
Enanta is Prepared for When RSV Returns

• Current RSV season in Northern Hemisphere has not begun due to continuing COVID-19 mitigation measures

• Modeling predicts large future outbreaks of RSV¹
  – Caused by an increase in susceptible RSV population, resulting from ongoing mitigation measures
  – Assuming measures are reduced, a peak in infections are projected for the next RSV seasons

• Enanta is ready to move quickly upon RSV re-emergence
  – Number of clinical sites more than doubled with expansion in EU and Asia-Pacific (total of ~150 sites globally)

• Updated guidance will be provided once RSV becomes prevalent again

Two Additional Phase 2 Clinical Trials: RSVTx and RSVPEDs

**RSVPEDs**

Planned for Q1 2021

- ~90 subjects
  - Age 28 days – 24 months
  - Dosed in 4 age cohorts

**Primary Objective, Part 2:**
Antiviral activity of EDP-938

**Secondary Objectives:**
Viral load, progression to respiratory failure or all-cause mortality, PRO, PK, & safety

**RSVTx**

Initiated in Q4 2020

- ~200 adult HCT recipients
  - Age 18 – 75 years

**Primary Objective:**
Effect of EDP-938 on development of LRTC in HCT subjects with acute RSV URTI

**Secondary Objectives:**
Viral load, progression to respiratory failure or all-cause mortality, PRO, PK, & safety

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LRTC: lower respiratory tract complication; HCR: hematopoietic cell transplant; URTI: upper respiratory tract infection; PRO: patient reported outcomes; PK: pharmacokinetics
RSV L-Protein Inhibitor

- Enanta’s newest RSV program
- RSV L-protein is a viral RNA-dependent RNA polymerase that contains multiple enzyme activities required for RSV replication
- Novel RSV L-protein inhibitor leads have nanomolar potency against RSV-A and RSV-B
- Not expected to have cross resistance to other classes of inhibitors
  - Potential to be used alone or in combination with other RSV mechanisms, such as EDP-938
Human Metapneumovirus (hMPV)

Important cause of respiratory tract infections (RTIs), particularly in children, the elderly and immunocompromised individuals

- Paramyxovirus closely related to RSV
  - hMPV replication dependent on several viral proteins that form a multiprotein complex in cells
  - Multiple potential targets for hMPV drug discovery
- No approved vaccine or therapeutics available
- Enanta nanomolar hMPV inhibitor leads under active optimization

**hMPV at a Glance**

<table>
<thead>
<tr>
<th>Source</th>
<th>Details</th>
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<tbody>
<tr>
<td>Serious respiratory infections can occur in children under 5 years old</td>
<td></td>
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<tr>
<td>Second most common cause of lower RTIs in children (behind RSV)</td>
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<tr>
<td>Reinfection with hMPV occurs throughout life</td>
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</tr>
</tbody>
</table>

Sources: Epidemiology of Human Metapneumovirus; Jeffrey S. Kahn; *Clinical Microbiology Reviews* Jul 2006, 19 (3) 546-557; DOI: 10.1128/CMR.00014-06; [https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1539100/](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1539100/)
SARS-CoV-2 (COVID-19)

Caused by respiratory infection of a new highly pathogenic coronavirus, SARS-CoV-2

- SARS-CoV-2 belongs to coronaviridae, a family of enveloped RNA viruses that includes SARS-CoV and MERS-CoV
- Despite vaccine and therapeutic progress, need an oral treatment for those infected with SARS-CoV-2, and potentially for mutated virus, or for future coronaviruses
- Enanta is leveraging years of antiviral drug discovery expertise to identify direct-acting antivirals
  - Discovery efforts on multiple targets utilizing a combination of screening and drug design
  - Potent molecules currently undergoing lead optimization
Our Therapeutic Focus

Leveraging our core strength in Hepatitis C to become a leader in oral treatments for viral infections and liver diseases

Several new therapeutic areas with goal of building multiple approaches in each

- RSV
- hMPV
- COVID-19
- HBV
- HCV
- NASH
Hepatitis B Virus (HBV)

Potentially life-threatening liver infection caused by the hepatitis B virus

Current treatments rarely give true cures

- **Interferon** is ~10% effective, but with side effects
- **Reverse-transcriptase inhibitors** effective at reducing viral load, but low cure rates (1% or lower) and treatment for life to improve cirrhosis or hepatocellular carcinoma (HCC) outcomes

<table>
<thead>
<tr>
<th>HBV at a Glance</th>
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<tbody>
<tr>
<td><strong>US</strong></td>
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<tr>
<td><strong>US, Japan, Major EU Populations</strong></td>
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<tr>
<td><strong>Worldwide</strong></td>
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</tbody>
</table>

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 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 cell 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
EDP-514 is Efficacious in the Humanized Liver Mouse Model

- uPA/SCID mice were infected with genotype C HBV and dosed with EDP-514 for 12 weeks

![Graph showing serum HBV DNA levels over time for different treatment groups.](image-url)
# EDP-514 Clinical Development Program

## Phase 1 Study

### Part 1 Complete
- Healthy volunteer SAD/MAD study evaluated safety, tolerability and PK
- Generally safe and well tolerated over dose range for up to 14 days
  - All reported TEAEs of mild severity
- PK profile supportive of once daily dosing with no food effect

### Part 2 Ongoing
- NUC-suppressed patients
- Evaluating safety, tolerability, PK and antiviral activity over 28 days
- 24 patients randomized to receive one of three multiple ascending doses of EDP-514 or placebo
- Preliminary data anticipated in 2Q 2021

## Phase 1b Study

### Ongoing
- Viremic chronic HBV subjects not currently on therapy
- Evaluating the safety, tolerability, PK and antiviral activity over 28 days
- 24 patients randomized to receive one of three multiple ascending doses of EDP-514 or placebo
- Preliminary data anticipated in 2Q 2021
EDP-721: HBV RNA Destabilizer

- Small molecules that cause destabilization and ultimate degradation of HBV RNAs
  - Result in reduction of HBsAg and other viral proteins in whole cell systems and animal models
  - Potential to complement or replace injectable (siRNA/ASO) with oral agents

- EDP-721 is an oral small molecule HBV RNA destabilizer with a robust preclinical profile
  - Sub-nanomolar potency \textit{in vitro}; dose dependent HBsAg reductions \textit{in vivo}
  - HBV pan-genomic activity; additive to synergistic activity with nucleosides and core inhibitors

- EDP-721 in combination with other agents may lead to a functional cure
  - High levels of HBsAg suppress immune responses through multiple mechanisms
  - EDP-721 reduces HBsAg derived from both integrated viral DNA and cccDNA
  - Sustained loss of HBsAg is regarded as a core component of a functional cure for HBV
Combination Regimen: Potential for Functional Cure

- Combination of multiple antiviral agents can block different points in the HBV life cycle
- Potential to drive rapid and deep suppression of viral replication (EDP-514 + NUC) and suppression of sAg production (EDP-721)
- All-oral regimen of EDP-514, EDP-721, NUC has potential to lead to a functional cure for HBV
Leveraging our core strength in Hepatitis C to become a leader in oral treatments for *viral* infections and *liver* diseases

Several new therapeutic areas with goal of building multiple approaches in each
Non-Alcoholic Steatohepatitis (NASH)

Leading cause of liver disease in western countries

- Associated with metabolic syndrome, diabetes and hypertension
- Dramatically increases risk of cirrhosis, liver failure and hepatocellular carcinoma
- By 2030 NASH will be the most frequent reason for liver transplants in the U.S.

NASH at a Glance

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<tr>
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<tbody>
<tr>
<td>US</td>
<td>2.5 – 16.3M people</td>
</tr>
<tr>
<td>Worldwide</td>
<td>115M people</td>
</tr>
<tr>
<td>Worldwide in 2030</td>
<td>357M people</td>
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</table>

Sources: American Liver Foundation, GlobalLiver.org
EDP-305: A Potent FXR Receptor Agonist

**Farnesoid X Receptor (FXR)**
- Nuclear hormone 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 agonists ameliorate pathologies in NASH models, including fibrosis
- Clinical validation of FXR agonist in NASH with 6-ECDCA (OCA)

**EDP-305**
- Non-bile acid
- Designed to take advantage of increased binding interactions with the receptor
- Highly selective for FXR vs other nuclear receptors
- Potent and differentiated effects on FXR-dependent gene expression vs OCA in preclinical models
- Robust efficacy seen in multiple fibrosis and NASH models
- Granted Fast Track Designation by FDA
EDP-305 ARGON-1 Study Summary

Endpoints Met at Week 12 Using 2.5 mg Dose

Primary Endpoint:
ALT change

Secondary Endpoint:
Liver fat by MRI-PDFF

- Strong target engagement as shown by decrease in C4, and increase in FGF-19 and ALP
- Robust reduction in marker of liver injury (GGT)
- Generally safe for up to 12 weeks
  - Majority of treatment emergent adverse events were mild to moderate
  - Incidence of treatment discontinuation due to pruritus: 1.8% for 1 mg and 20.8% for 2.5 mg
  - Associated with small numeric absolute changes in lipids
EDP-305: ARGON-2 Phase 2b Study

- Includes 12-week internal interim analysis by mid-2021 (to generate dose information more quickly for potential combinations), as well as a prespecified powered IA when ~40 of subjects reach week 72 biopsy
- Two doses selected to provide strong target engagement and a balanced profile in terms of efficacy and tolerability
  - 1.5 mg dose: designed to demonstrate stronger biomarker signals of efficacy than seen at 1.0 mg
  - 2.0 mg dose: designed to demonstrate less pruritus than seen at 2.5 mg

Primary Endpoint:
Improvement of fibrosis without worsening of NASH and/or NASH resolution without worsening of fibrosis
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

• Initiated Phase 1 study in 3Q 2020; data expected in 2Q 2021
## EDP-297: Highly Potent with Excellent Target Tissue Distribution

<table>
<thead>
<tr>
<th>Compound</th>
<th>Mode</th>
<th>FXR FL Activation EC$_{50}$ (nM)</th>
<th>Dose (mouse, po)</th>
<th>Intestine / Plasma</th>
<th>Liver / Plasma @ 4 hrs ~ Tmax</th>
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<tbody>
<tr>
<td>OCA</td>
<td>Bile Acid</td>
<td>130$^1$</td>
<td>10 mg/kg</td>
<td>160</td>
<td>26</td>
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<tr>
<td>cilofexor</td>
<td>Non-Bile Acid</td>
<td>41$^2$</td>
<td>1 mg/kg</td>
<td>0.6</td>
<td>0.9</td>
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<td>1 mg/kg</td>
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<td>EDP-297$^4$</td>
<td>Non-Bile Acid</td>
<td>&lt;0.1</td>
<td>1 mg/kg</td>
<td>265</td>
<td>75</td>
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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
Leveraging our core strength in Hepatitis C to become a leader in oral treatments for viral infections and liver diseases

Several new therapeutic areas with goal of building multiple approaches in each
# Glecaprevir – Our Licensed Protease Inhibitor for Hepatitis C Virus

<table>
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<tr>
<th>Product</th>
<th>Regimen</th>
<th>Enanta Asset</th>
<th>Economics*</th>
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<tbody>
<tr>
<td>Mavyret</td>
<td>2-DAA (ABBV)</td>
<td>glecaprevir (PI)</td>
<td>Double-digit royalty on 50% of net sales</td>
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<table>
<thead>
<tr>
<th>Royalty Rate (annual)</th>
<th>Glecaprevir Sales (50% of Mavyret net sales)</th>
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<tbody>
<tr>
<td>20%</td>
<td>$2.5B</td>
</tr>
<tr>
<td>17%</td>
<td>$1.0B</td>
</tr>
<tr>
<td>14%</td>
<td>$750M</td>
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<tr>
<td>12%</td>
<td>$500M</td>
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<tr>
<td>10%</td>
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## Calendar 2020 HCV Royalties

- **Q4** $TBD
- **Q3** $23.6M
- **Q2** $18.7M
- **Q1** $27.6M

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

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<td>Total Revenues</td>
<td>$122.5</td>
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<td>R&amp;D Expenses</td>
<td>$136.8</td>
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<td>G&amp;A Expenses</td>
<td>$27.4</td>
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<tr>
<td>Net Income (Loss)</td>
<td>$(36.2)</td>
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<td>Net Income (Loss) per Diluted Common Share</td>
<td>$(1.81)</td>
<td>$(1.46)</td>
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Balance Sheet

| Cash, Cash Equivalents and Marketable Securities | $419.3 | $419.3 |
Key Catalysts 2021

Virology

RSV N-Inhibitor EDP-938
- Initiated RSVTx in 4Q 2020
- Initiate RSVPEDs in Q1 2021
- Resume recruitment for RSVP when RSV returns

hMPV, SARS-CoV-2 and RSV L-inhibitor
- Nominate two clinical development candidates

HBV: Core Inhibitor EDP-514 and RNA Destabilizer EDP-721
- EDP-514 Phase 1b in viremic HBV patients; preliminary data anticipated in 2Q 2021
- EDP-514 Phase 1b in NUC-suppressed HBV patients; preliminary data anticipated in 2Q 2021
- Initiate Phase 1 with EDP-721 in mid-2021

NASH

FXR Agonists EDP-305 and EDP-297
- ARGON-2 Phase 2b in NASH ongoing; 12-week interim analysis in mid-2021
- Phase 1 with EDP-297 (follow-on FXR) ongoing with data expected in 2Q 2021
- Advance non-FXR compounds for NASH