UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
Washington, D.C. 20549

FORM 8-K

CURRENT REPORT
Pursuant to Section 13 or 15(d) of the
Securities Exchange Act of 1934

Date of Report (Date of earliest event reported): **June 20, 2018**

ASSEMBLY BIOSCIENCES, INC.
(Exact name of registrant as specified in its charter)

Delaware	**001-35005**	**20-8729264**
(State or other jurisdiction of incorporation)	(Commission File Number)	(I.R.S. Employer Identification No.)

11711 N. Meridian St., Suite 310
Carmel, Indiana 46032
(Address of principal executive offices, including zip code)

(317) 210-9311
(Registrant's telephone number, including area code)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions:

☐ Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)

☐ Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)

☐ Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))

☐ Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 or Rule 12b-2 of the Securities Exchange Act of 1934.

Emerging growth company ☐

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act. ☐

Item 7.01 Regulation FD Disclosure.

Assembly Biosciences, Inc. (the "Company") is furnishing a corporate presentation, attached as Exhibit 99.1 to this Current Report on Form 8-K, that the Company intends to use from time to time in meetings with investors and others beginning on June 20, 2018. The corporate presentation will also be available on the Company's website at https://investor.assemblybio.com/events-presentations.

The information in this Item 7.01 and Exhibit 99.1 attached hereto is furnished and shall not be deemed "filed" for purposes of Section 18 of the Securities Exchange Act of 1934, as amended (the "Exchange Act"), or otherwise subject to the liabilities of that section, nor shall it be deemed incorporated by reference in any filing under the Securities Act of 1933, as amended, or the Exchange Act, except as expressly set forth by specific reference in such filing.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits:

The following exhibit relating to Item 7.01 shall be deemed furnished and not filed.

Exhibit No.	Description
99.1	Assembly Biosciences, Inc. Corporate Presentation dated June 20, 2018.

EXHIBIT INDEX

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SIGNATURES

Pursuant to the requirements of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

Date: June 20, 2018

Assembly Biosciences, Inc.

By: /s/ Derek A. Small
Derek A. Small
President and Chief Executive Officer

R&D Day
Hepatitis B Program

June 20, 2018

NASDAQ: ASMB

CAUTIONARY NOTE REGARDING FORWARD-LOOKING STATEMENTS

The information in this presentation contains estimates and other forward-looking statements regarding future events, including statements about the clinical and therapeutic potential of our HBV-cure program, the timing of the initiation of and availability of data from our ongoing and planned clinical trials in our HBV-cure program, and the plans, strategies, milestones, and intentions related to our HBV-cure and Microbiome programs. Certain forward-looking statements may be identified by reference to a future period or periods or by use of forward-looking terminology such as "forecast," "believe," "planned," "initiate," "potential," "anticipated," or "expected." Such forward-looking statements, which we intend to be covered by the safe harbor provisions contained in Section 27A of the Securities Act of 1933, as amended, and Section 21E of the Securities Exchange Act of 1934, as amended, are just predictions and are subject to risks and uncertainties that could cause the actual events or results to differ materially. These risks and uncertainties include, among others: outcomes of nonclinical testing and clinical trials are uncertain; results of earlier nonclinical studies and clinical trials may not be predictive of future clinical trial results; the components, timing, patient enrollment and completion rates, cost and results of clinical trials and other development activities involving our product candidates; the duration and results of regulatory review of those product candidates by the FDA and foreign regulatory authorities; our estimates regarding our capital requirements, and our need for future capital; and the possible impairment of, or inability to obtain, intellectual property rights and the costs of obtaining such rights from third parties. These and other potential risks and uncertainties that could cause actual results to differ from the results predicted are more fully detailed under the heading "Risk Factors" in our Annual Report on Form 10-K for the year ended December 31, 2017, and our Quarterly Report on Form 10-Q for the quarter ended March 31, 2018, each filed with the Securities and Exchange Commission. It is not possible for Assembly Biosciences management to predict all risks nor can we assess the impact of all factors on our business or the extent to which any factor, or combination of factors, may cause actual results to differ materially from those contained in any forward-looking statements we may make. This presentation also contains estimates and other statistical data made by independent parties and by us relating to market potential. These estimates involve a number of assumptions and limitations, and you are cautioned not to give undue weight to such estimates. In light of these risks, uncertainties and assumptions, the forward-looking events and circumstances discussed in this presentation may not occur and actual results could differ materially and adversely from those anticipated. Except as required by law, we assume no obligation to update publicly any forward-looking statements, whether as a result of new information, future events, or otherwise.

ASSEMBLY BIOSCIENCES OVERVIEW

	HBV Cure 	Microbiome 
 **Unmet Patient Need**	**No cure** for the **>250 million patients** with chronic HBV	The gut microbiome is **essential** to human health, yet there are **no approved** microbiome therapies
 **Innovation**	Core inhibitors designed to **break the life cycle** of HBV	**Targeted delivery of oral, synthetic, live bio-therapeutics** designed to address the diseases associated with the gut microbiome

HBV = hepatitis B virus.

DEVELOPMENT PROGRAMS FOCUSED ON LARGE PATIENT POPULATIONS WITH HIGH UNMET NEED

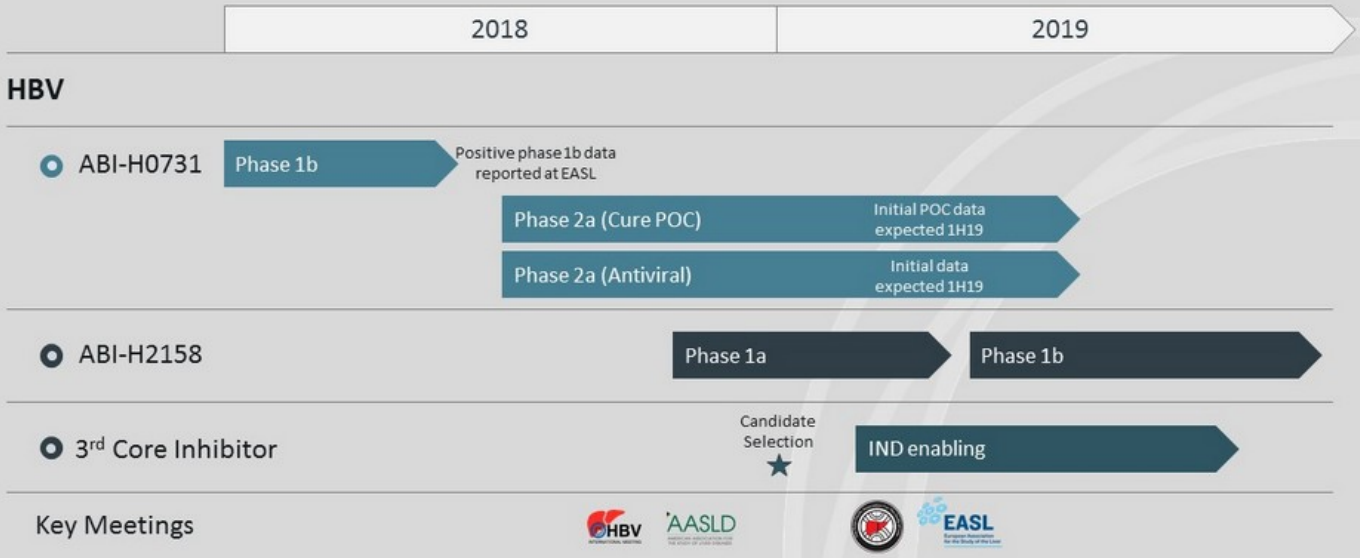
Drug Candidate (Mechanism/Indication)	Discovery & Lead Op/Selection	IND Enabling	Phase 1a	Phase 1b	Phase 2	Phase 3	NDA/BLA	Worldwide Rights
HBV Cure								
ABI-H0731 (Core Inhibitor)								
ABI-H2158 (Core Inhibitor)								
Third Core Inhibitor								
Microbiome								
ABI-M201 (Ulcerative Colitis)								Allergan
ABI-M301 (Crohn's Disease)								Allergan
Irritable Bowel Syndrome								Allergan
NASH								
Immuno-oncology								

BLA = Biologic License Application; IND = Investigational New Drug Application; NASH = nonalcoholic steatohepatitis; NDA = New Drug Application.

SIGNIFICANT PROGRESS WITH EXCITING NEW DEVELOPMENTS

- Positive Phase 1b data presented at EASL on ABI-H0731
  - These data and some of our preclinical data showing that Core Inhibitors are effective antivirals with distinct mechanisms from standard of care nucleoside analogs
- On track to initiate two Phase 2 clinical trials for ABI-H0731 this summer – data is expected 1H 2019
- ABI-H0731 composition of matter patent has been allowed by USPTO
  - Our HBV program has over 14 patents filed or pending in the US and over 100 filings in other major geographies with patent terms through 2035-2038, and additional filings in process
  - Each of our Core Inhibitor programs are novel, and chemically distinct

UPCOMING HBV MILESTONES



EASL = European Association for the Study of the Liver; POC = proof of concept.

HBV DISEASE PROGRESSION

~ 2 people per minute will die from complications associated with HBV

Chronic Infection

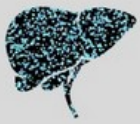
>250 million chronically infected worldwide

8% diagnosed

<1% receive treatment

1%-3% of those receiving treatment with current options achieve functional cure

Cirrhosis/HCC



20%-30%

Surgery, chemotherapy, and liver transplant

Death

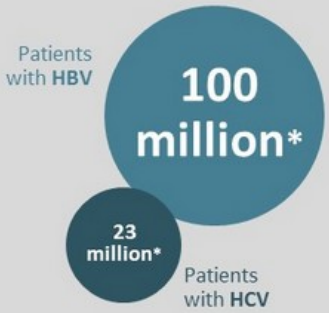
~1 million people/year

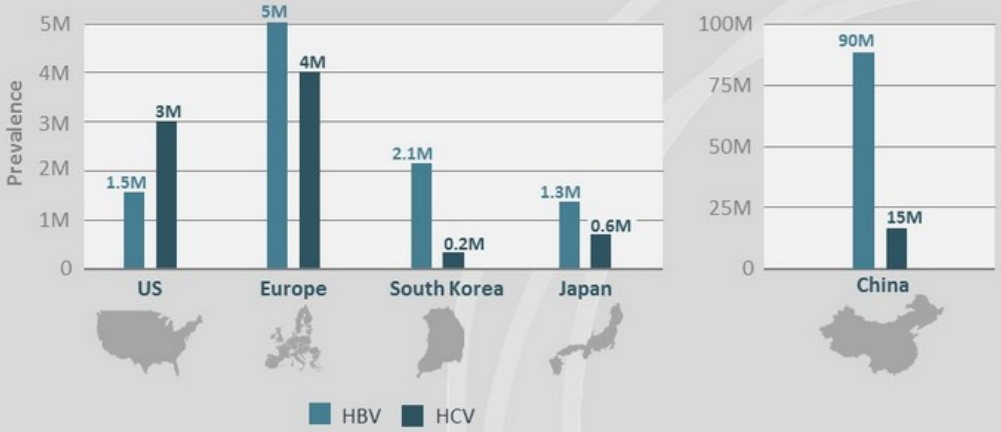
2 people/minute

HCC = hepatocellular carcinoma.
Source: World Health Organization; Hepatitis B Foundation.

MORE HBV PATIENTS THAN HCV PATIENTS IN THE MAJOR GEOGRAPHIES

More total Hepatitis B patients in top 4 major geographies than Hepatitis C patients; this is not including China, which is even worse





*US, Europe, South Korea, Japan, China.
HCV = hepatitis C virus.
WHO and ECDC.

ASSEMBLY CHINA ESTABLISHED TO RAPIDLY DEVELOP PROGRAMS IN CHINA

HBV is a **public health epidemic in China** – the government has made HBV one of their highest public health priorities

- China has made significant advances in regulatory, IP and business policies over the last few years
- Assembly China efforts began in 2015
  - ASMB China headquarters in Shanghai (Zhangjiang Hi-Tech Park)
  - Launching a regulatory office in Beijing
- Currently establishing a dedicated team for our China business to develop our programs as a domestic Chinese entity

DEVELOPING A POTENTIAL CURE FOR HEPATITIS B

Cure is achievable

but currently at very low rates

Core Inhibitors

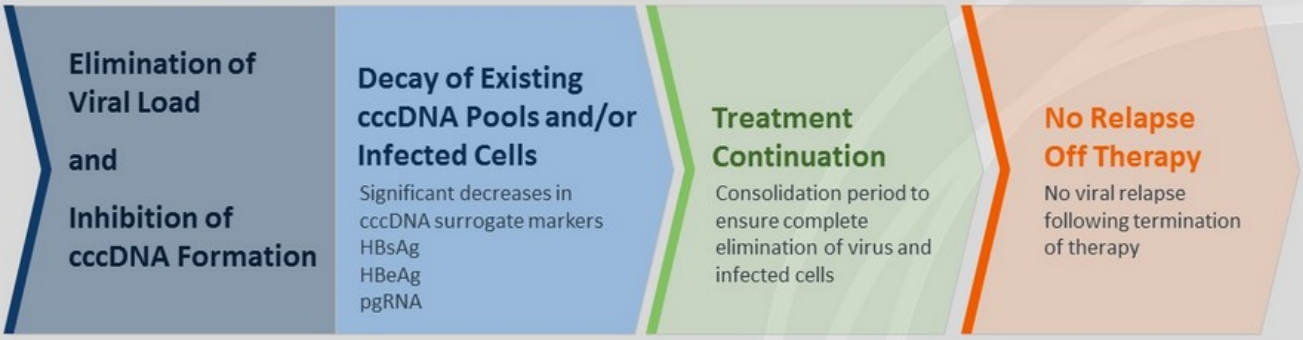
We believe backbone of **curative therapy**

Novel mechanism designed to **break the HBV life cycle**

Assembly Biosciences has a **deep pipeline of potent core inhibitors**

HBV CURE: CLINICAL COMPONENTS

Expected Treatment Components to Achieve Cure



THOUGHT WE HAD A POTENTIAL CURE OVER 10 YEARS AGO

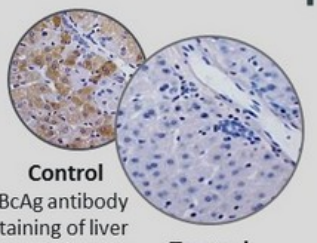
ETV MONOTHERAPY CURED CHRONICALLY INFECTED WOODCHUCKS

CLEAR EVIDENCE OF CURE WITH
ETV (0.5 mg/kg) daily/weekly

WHBV DNA
UNDETECTABLE
in <2 MONTHS

cccDNA levels
REDUCED
>4 LOGS

Prevented HCC and
EXTENDED
life of infected ANIMALS



Control
HBcAg antibody staining of liver biopsies

Treated

mean WHBsAg levels
REDUCED
by **91%**

NO Viral
24 Relapses
MONTHS
post treatment

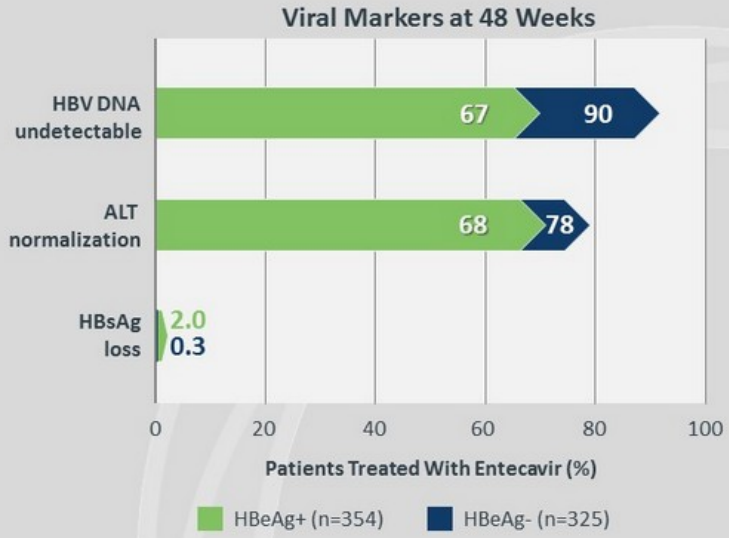
Unfortunately, this is not what happens in HBV patients, despite prolonged therapy

ETV = entecavir; HBcAg = hepatitis B core antigen; WHBsAg = woodchuck hepatitis B surface antigen; WHBV = woodchuck hepatitis B virus.
Colonno, et al. *JID.* 2001;184:1236-1245

SO WHAT HAPPENED IN PATIENTS? VIRUS NOT FULLY SUPPRESSED

SOC NUCS ARE POTENT ANTIVIRALS AND REDUCE VIRAL LOAD

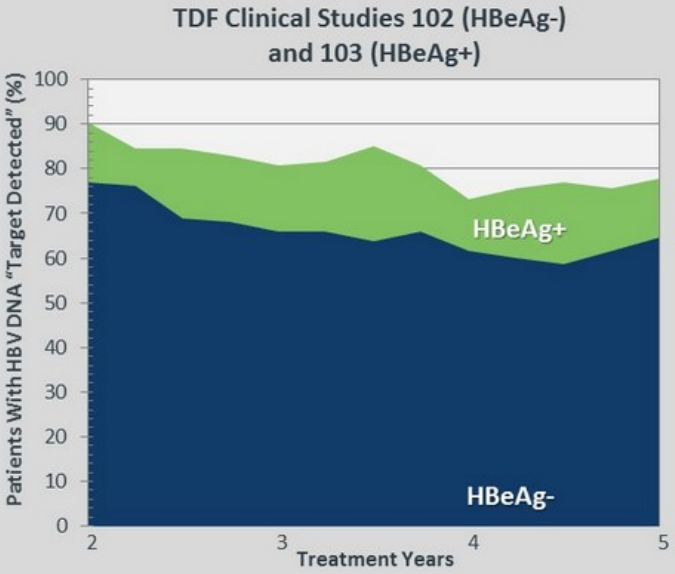
- Preferred SOC nucs are ETV, TFV, and TAF
- Exhibit potent antiviral efficacy
- Rapidly reduce viral DNA to lower limit of detection in virtually all patients
- **Viral suppression maintained indefinitely with continued therapy**



ALT = alanine aminotransferase; HBeAg = hepatitis B e antigen; HBV = hepatitis B virus; nuc = nucleos(t)ide inhibitor; SOC = standard of care; TAF = tenofovir alafenamide; TFV = tenofovir.

1. Chang T-T, et al. *N Engl J Med*. 2006;354:1001-1010. 2. Lai C-L, et al. *N Engl J Med*. 2006;354:1011-20.

...BUT THEY FAIL TO FULLY SUPPRESS VIRAL REPLICATION



- Reductions in HBsAg alone is insufficient, as the immune system fails to eliminate low-level persistent infection
- **Numerous long-term Nuc-treated patients with low HBsAg levels continue to have detectable HBV DNA and fail to seroconvert**

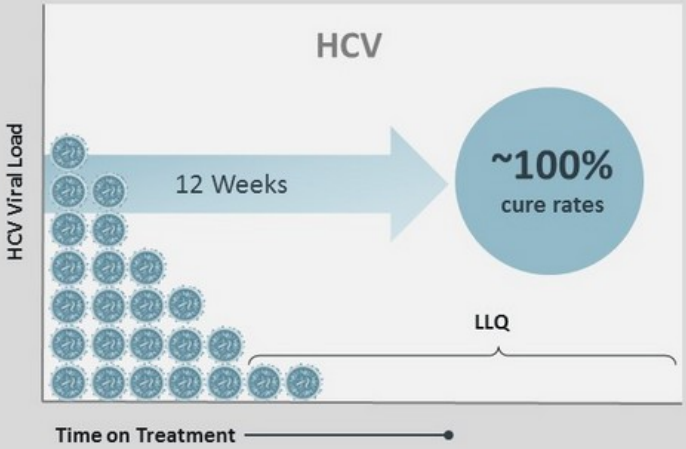
HBeAg Pos. Patient	Treatment	Treatment Years	HBsAg IU/mL	HBV DNA (copy/mL)
003	ADV/LVD	5	**6.6**	**1,530**
016	LVD/ADV/IFN	13	**3.8**	**1,040**
019	LVD/ADV/ETV/TDF	6.5	**4.7**	**1,840**
024	IFN	1	**0.6**	**188**

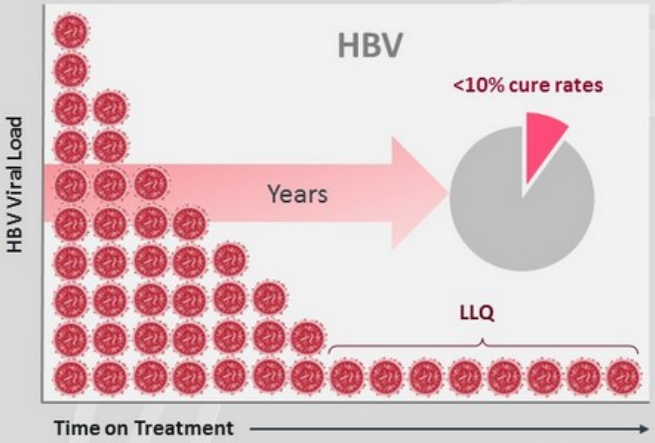
- **Cure is not possible if viral infection persists**

ADV = adefovir dipivoxil; IFN = interferon; LVD = ledipasvir; TDF = tenofovir disoproxil fumarate.

1. Marcellin P, et al. AASLD Poster 1861, 2014. 2. Huang, Q, et al. collaborative study in progress. 2018.

LACK OF FULL VIRAL SUPPRESION MAY BE REASON FOR LOW HBV CURE RATES



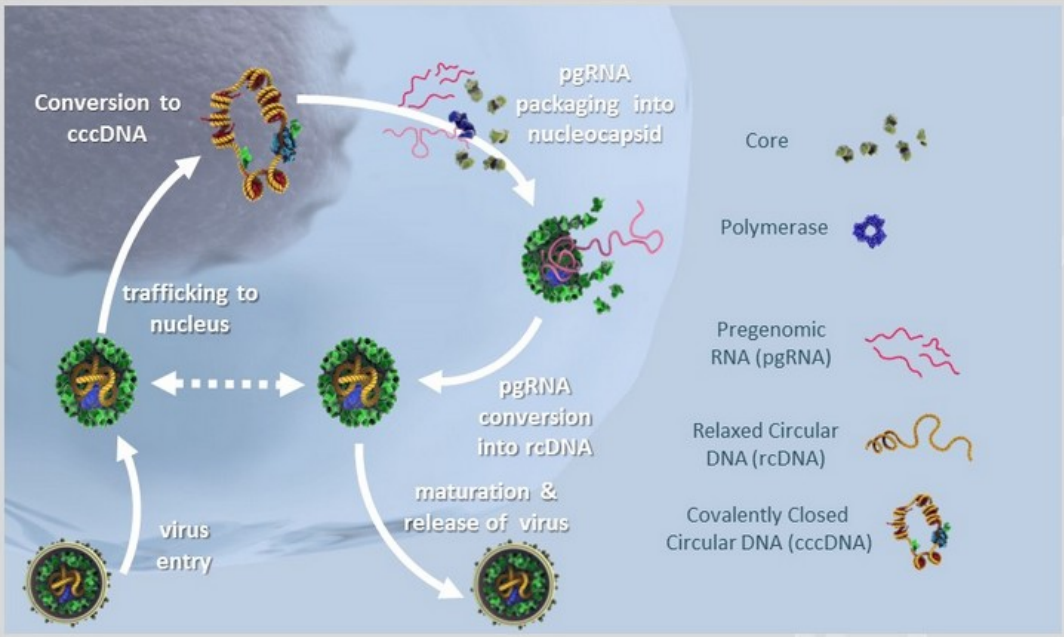


To improve cure rates…must eliminate residual virus

HCV = hepatitis C virus; LLQ = lower limit of quantification.

HOW CAN THERAPY BE IMPROVED?

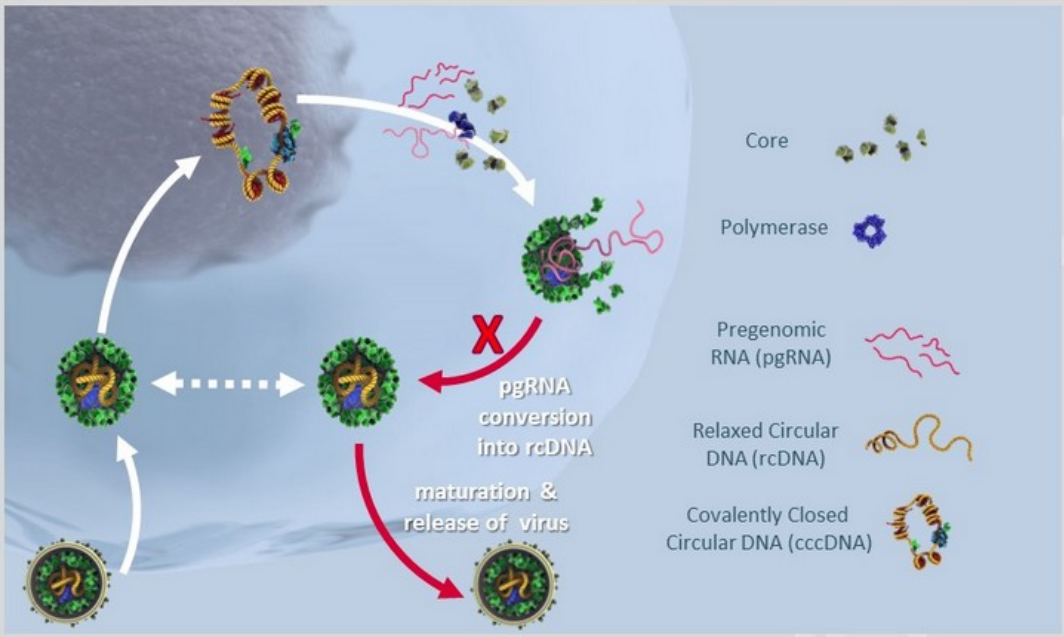
CRITICAL ELEMENTS OF HBV LIFE CYCLE



Core and polymerase proteins play critical roles in HBV life cycle

- Trafficking of nucleocapsid to nucleus
- Establishment of cccDNA
- Packaging of pgRNA into nucleocapsids
- Conversion of pgRNA into rcDNA

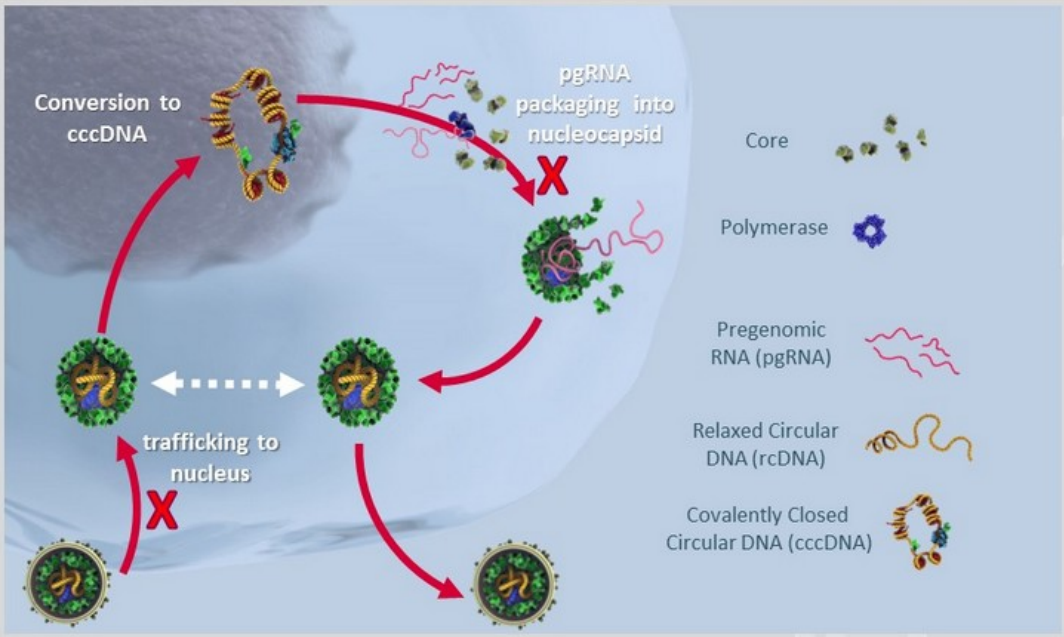
NUCS REDUCE VIRUS LEVELS BUT FAIL TO PREVENT cccDNA ESTABLISHMENT



Polymerase inhibition

- Prevents conversion of pgRNA to rcDNA
- Does not eliminate 100% of virus
- Has no effect on incoming virus
- Has a minimal effect on cccDNA pool

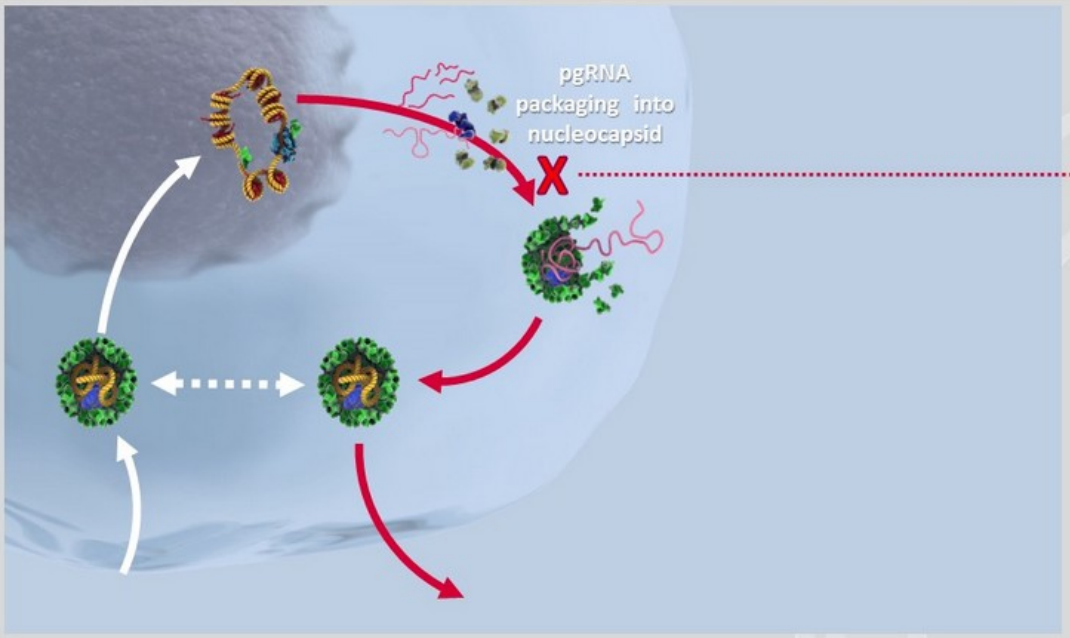
CORE INHIBITORS BLOCK VIRAL REPLICATION AND cccDNA ESTABLISHMENT

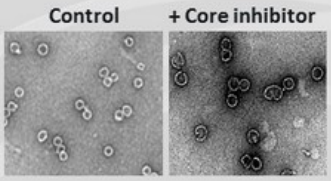


Core inhibition

- Inhibits both production of new virus and trafficking of incoming nucleocapsid to nucleus
- Unlike nucs, blocks establishment of cccDNA
- Has potential to be additive or synergistic with polymerase inhibition

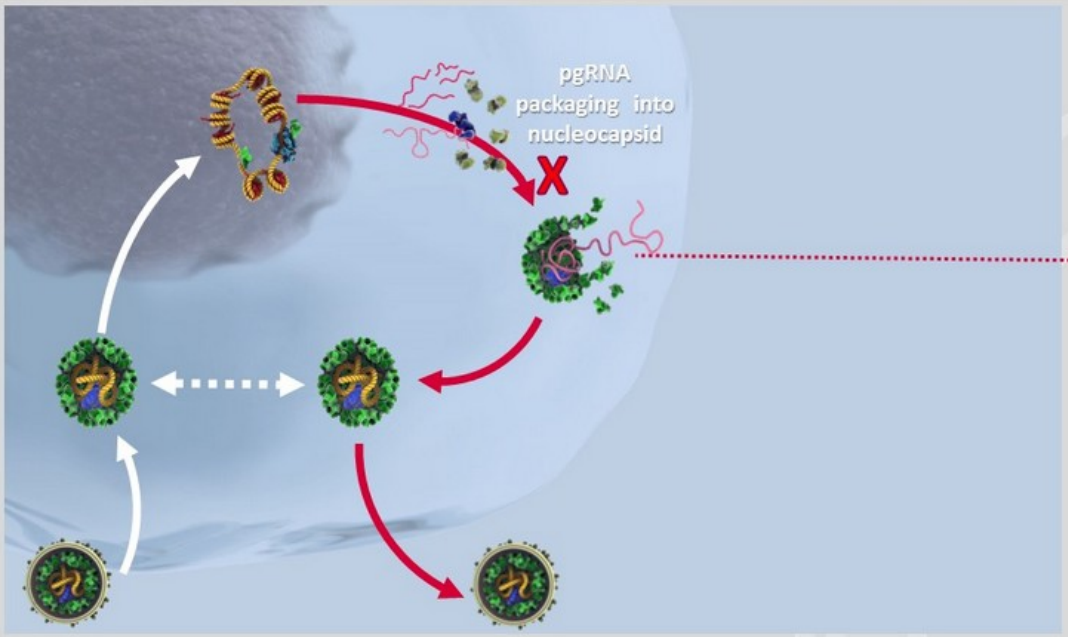
CORE INHIBITORS BLOCK FORMATION OF FUNCTIONAL NUCLEOCAPSIDS

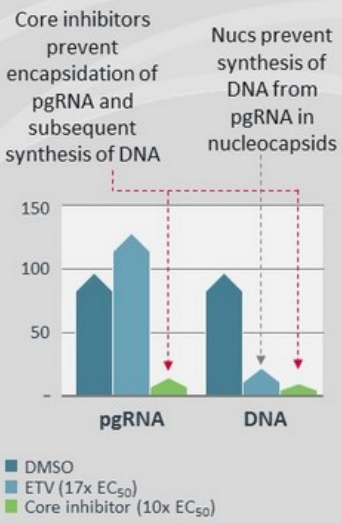




Addition of core inhibitor causes formation of aberrant capsids that are larger, cracked, and asymmetrical

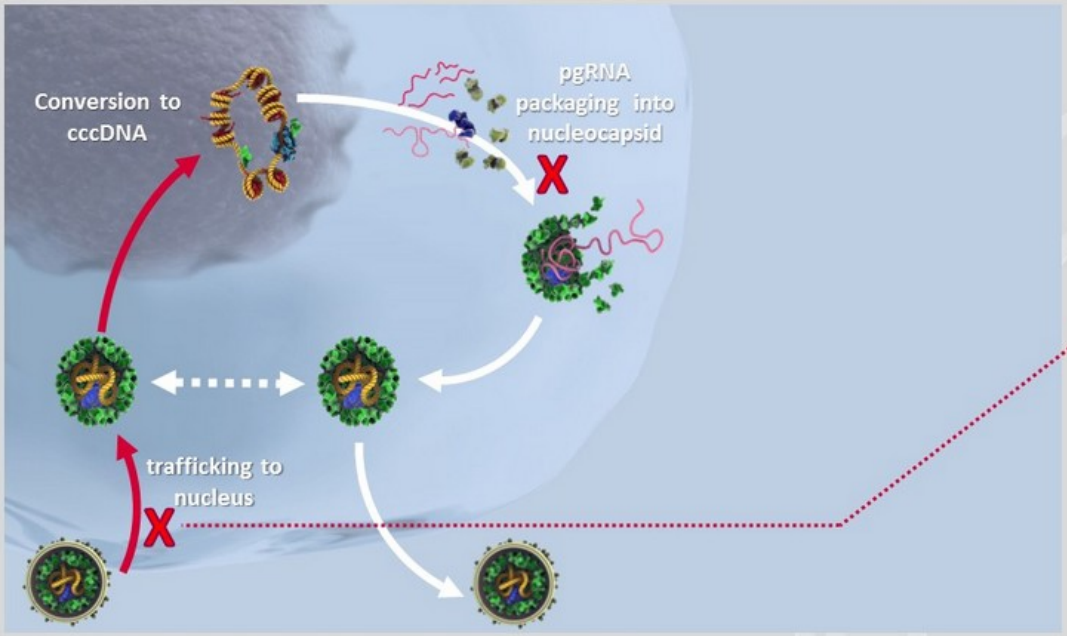
CORE INHIBITORS BLOCK ENCAPSIDATION OF pgRNA



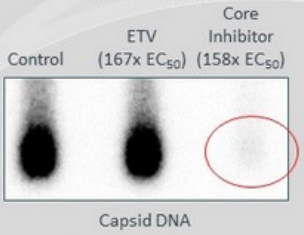


Huang Q, et al. AASLD Poster 922 2017.

CORE INHIBITORS CAUSE PREMATURE MELTING OF NUCLEOCAPSIDS



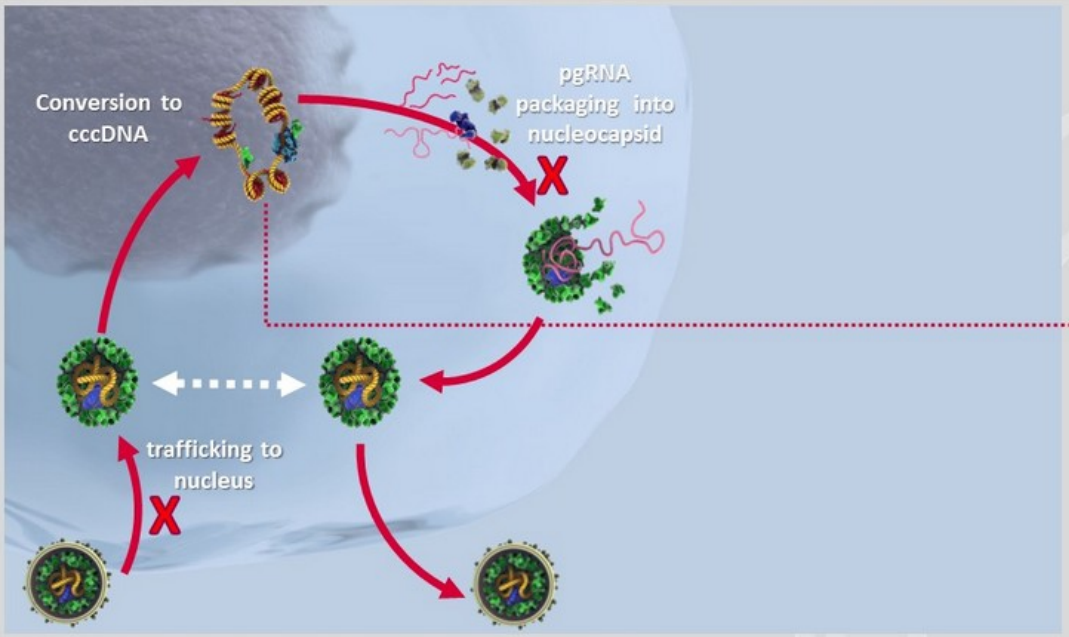
Southern Blot of HBV DNA From HepG2-NTCP Cells 3 Hours After Infection



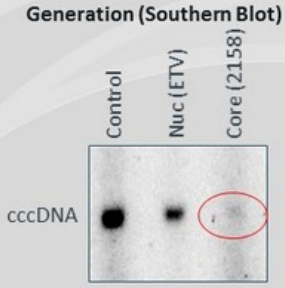
Core inhibitors cause premature melting of existing nucleocapsids and prevent trafficking of rcDNA to nucleus

1. Cai, D, et al. Intl HBV Mtg Poster P-140. 2017; 2. Huang Q, et al. AASLD Poster 922 2017.

CORE INHIBITORS BLOCK GENERATION OF cccDNA



Core Inhibitors Block cccDNA Generation (Southern Blot)

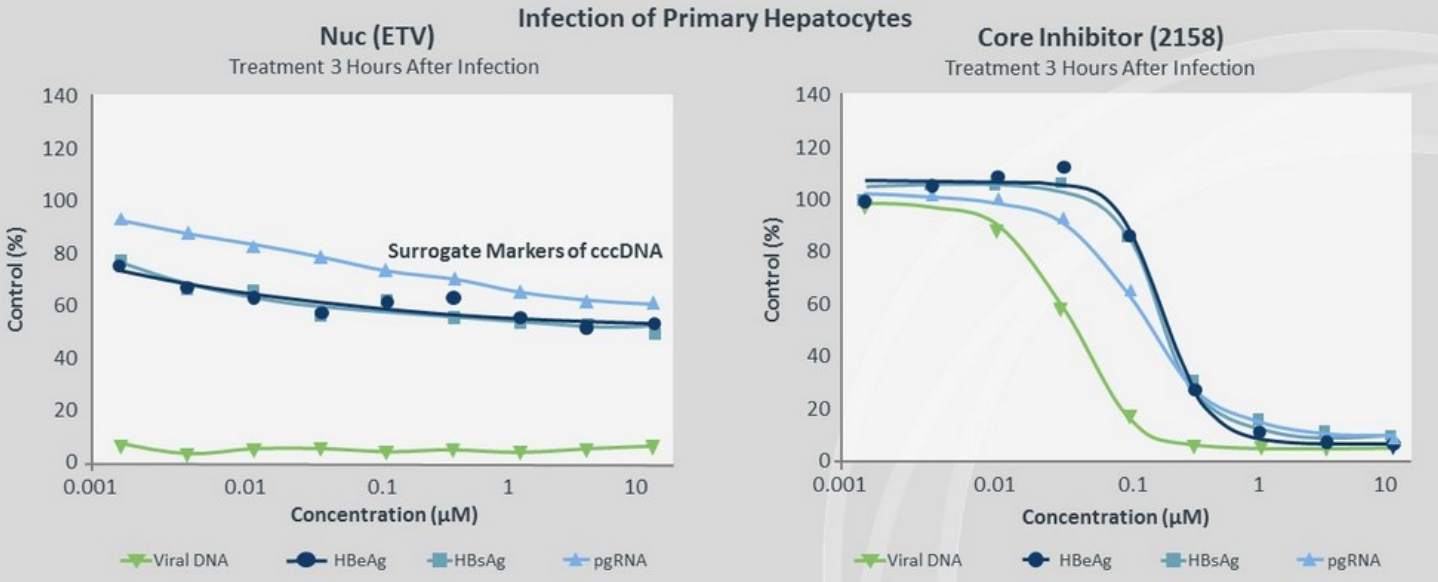


ETV >10,000x EC_{50}
2158 50x EC_{50}

Core inhibitors prevent cccDNA generation and formation

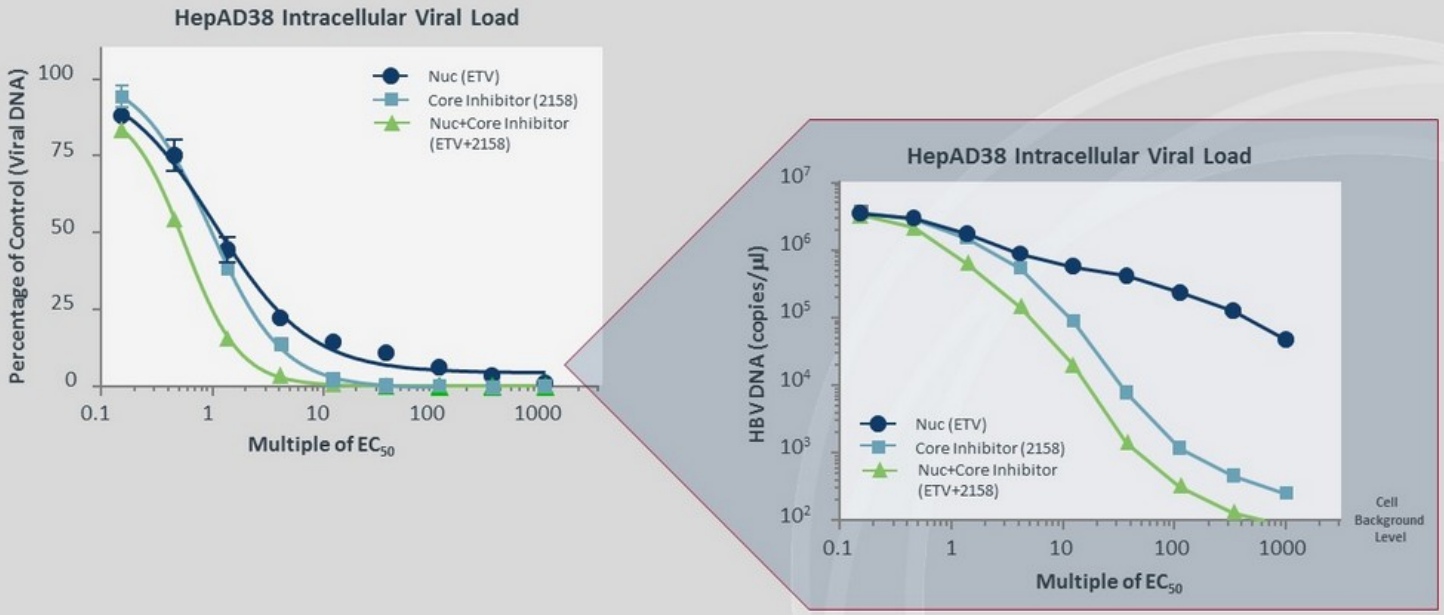
1. Cai, D, et al. Intl HBV Mtg Poster P-140. 2017; 2. Huang Q, et al. AASLD Poster 922 2017.

CORE INHIBITORS REDUCE KEY SURROGATE MARKERS FOR cccDNA



Huang Q, et al. AASLD Poster 922 2017.

BOTTOM LINE…CORE INHIBITORS ARE MORE EFFECTIVE ANTIVIRALS



HOW QUICKLY DO cccDNA AND INFECTED CELLS TURN OVER?

cccDNA BIOSYNTHESIS STUDY

Strategy/Approach

- Resistance emerges rapidly in lamivudine- and telbivudine-treated HBV patients
- Obtain longitudinal clinical samples (paired plasma and biopsy)
- Establish and validate isolation methodologies
- Follow resistance signature mutations as genetic markers of cccDNA turnover, confirm pgRNA sequences reflect cccDNA sequences

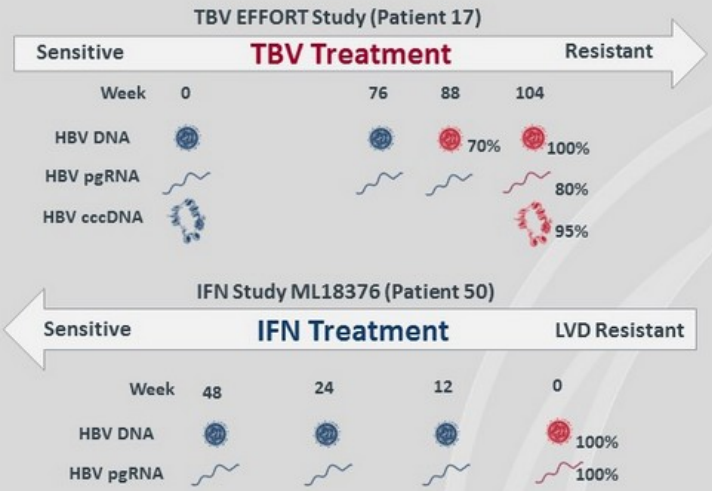
Objectives

- Confirm that genetic source of resistance is cccDNA
- Determine the timeframe required to turn over existing cccDNA populations in patients
- Determine if there are inactive subpopulations of cccDNA

Huang, et al. AASLD Poster 1503. 2017.

RAPID TURNOVER OF cccDNA IN AS LITTLE AS 12-16 WEEKS

HBV-Infected Hepatocyte

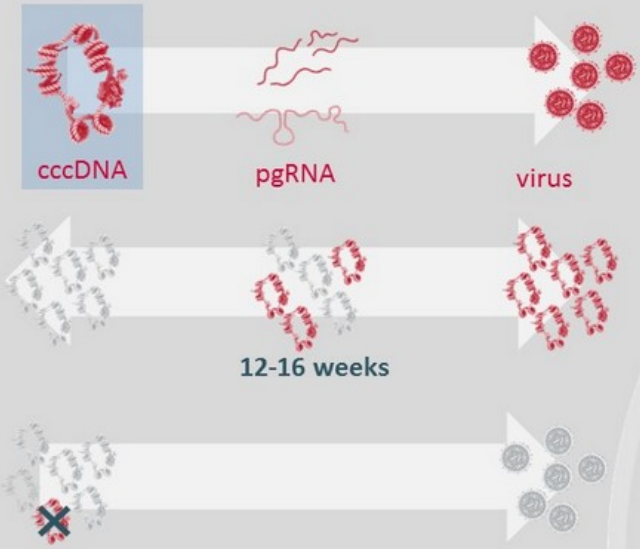


HBV^{R}-Infected Hepatocyte

Virus, pgRNA and cccDNA populations can be completely replaced in as little as 12 weeks

IFN = interferon; TBV = telbivudine.
Huang, et al. AASLD Poster 1503 2017.

SUMMARY RESULTS FROM ONGOING cccDNA BIOSYNTHESIS STUDY



1. Genetic source of resistance shown to be cccDNA

2. Turnover of cccDNA from sensitive to resistant and from resistant to sensitive occurs in 12-16 weeks…suggesting relatively rapid turnover of both pgRNA and cccDNA pools or infected cells

3. No evidence of an inactive subpopulation of cccDNA, as resistance mutations were observed in the entire population of cccDNA

Huang, et al. AASLD Poster 1503. 2017.

PIPELINE OF POTENT CORE INHIBITORS

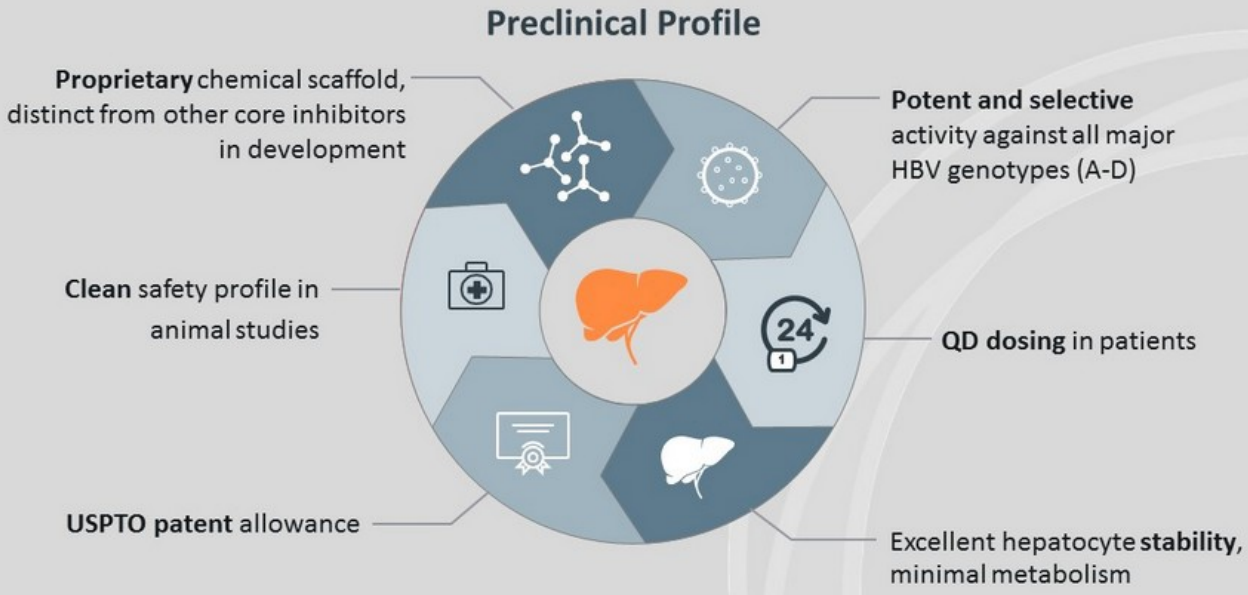
ASMB HBV CORE INHIBITOR PROGRAM PORTFOLIO

Novel Molecules With Distinct Chemical Scaffolds Discovered at Assembly Biosciences

Drug Candidate	Discovery	Lead Op/ Selection	IND Enabling	Phase 1a	Phase 1b	Phase 2	Phase 3	NDA/BLA	Worldwide Rights
ABI-H0731									
ABI-H2158									
Third Core Inhibitor									

BLA = Biologic License Application; IND = Investigational New Drug Application; NDA = New Drug Application.

ABI-H0731 IS THE LEAD CANDIDATE FROM A PIPELINE OF UNIQUE CORE INHIBITORS



USPTO = United States Patent and Trademark Office; QD = once daily.

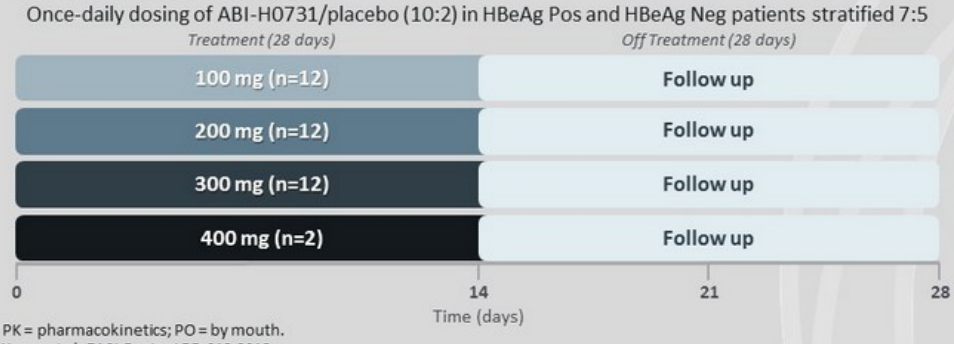
ABI-H0731
PHASE 1 STUDY DESIGNS

Study ABI-H0731-102

Once-daily dosing of ABI-H0731/placebo (10:2) in healthy volunteers

Study ABI-H0731-101(B)

Once-daily dosing of ABI-H0731/placebo (10:2) in HBeAg Pos and HBeAg Neg patients stratified 7:5



PK = pharmacokinetics; PO = by mouth.
Yuen, et al. EASL Poster LBP-012 2018.

Objectives

Primary

- Dose-related safety and tolerability

Secondary

- Steady state human PK
- Dose-related antiviral efficacy
- HBsAg and HBeAg levels
- Pre-existing and emergent resistance

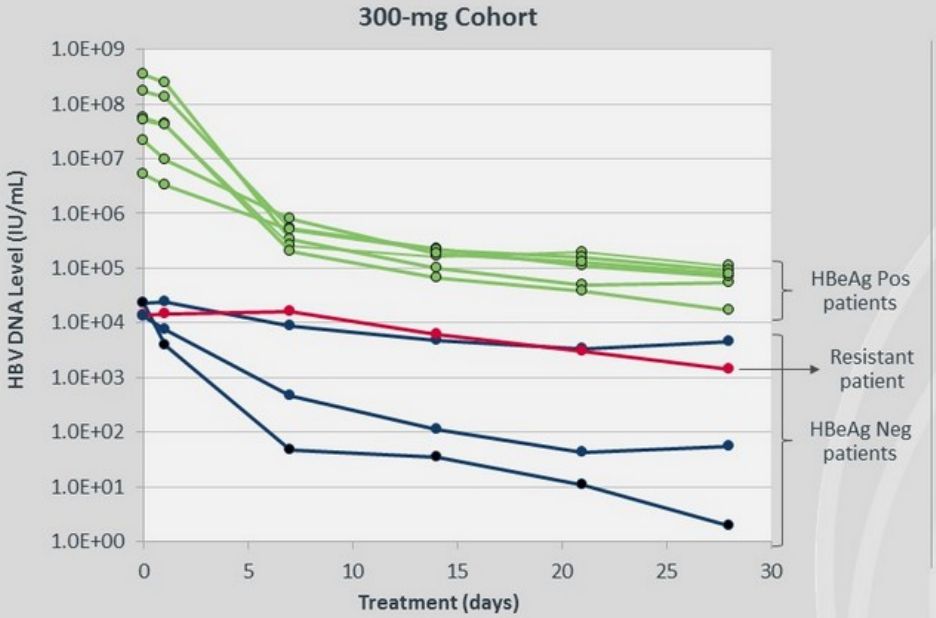
ABI-H0731
PHASE 1 – CLINICAL SAFETY SUMMARY

Dose (n)	100 mg (20)	200 mg (20)	300 mg (20)	400 mg (2)	Placebo (12)
Grade 1 AEs	24	15	12	4	9
Grade 2 AEs	0	0	0	0	0
Grade 3 AEs	0	0	0	1	0

- Generally well tolerated, with **no SAEs reported** and **no dose-limiting toxicities**
- **AEs not dose dependent**
- **All TEAEs were grade 1** (mild), except for a single subject with a grade 3 rash
  - Occurred in an Asian male, 46 years of age, HBeAg-, 400-mg patient on day 10
  - Deemed probably related to study drug
  - Rash resolved rapidly following treatment discontinuation without additional medical intervention

AE = adverse event; SAE = serious adverse event; TEAE = treatment-emergent adverse event.
Interim Data - Yuen, et al. EASL Poster LBP-012 2018.

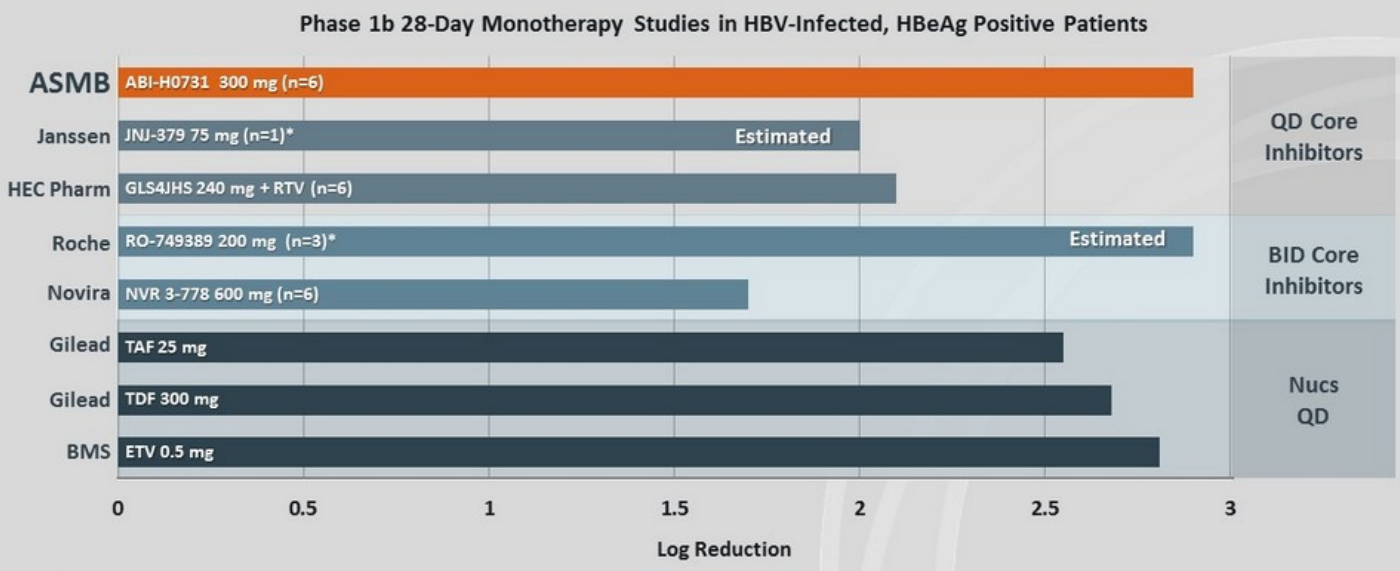
PHASE 1 STUDY – HBV DNA LEVELS IN TREATED HBV PATIENTS



- Steady state exposures achieved in ≤5 days, ~2-fold accumulation observed over 28 days
- Efficacious at all dose levels evaluated
- Mean maximal HBV DNA reduction of 2.8 logs observed in 300-mg cohort
- Individual patients achieved maximal declines of up to 4 logs
- One patient harbored T109M resistance mutation at baseline but still experienced a 1 log decline
- **The 300-mg dose was selected for evaluation in the upcoming phase 2a studies**

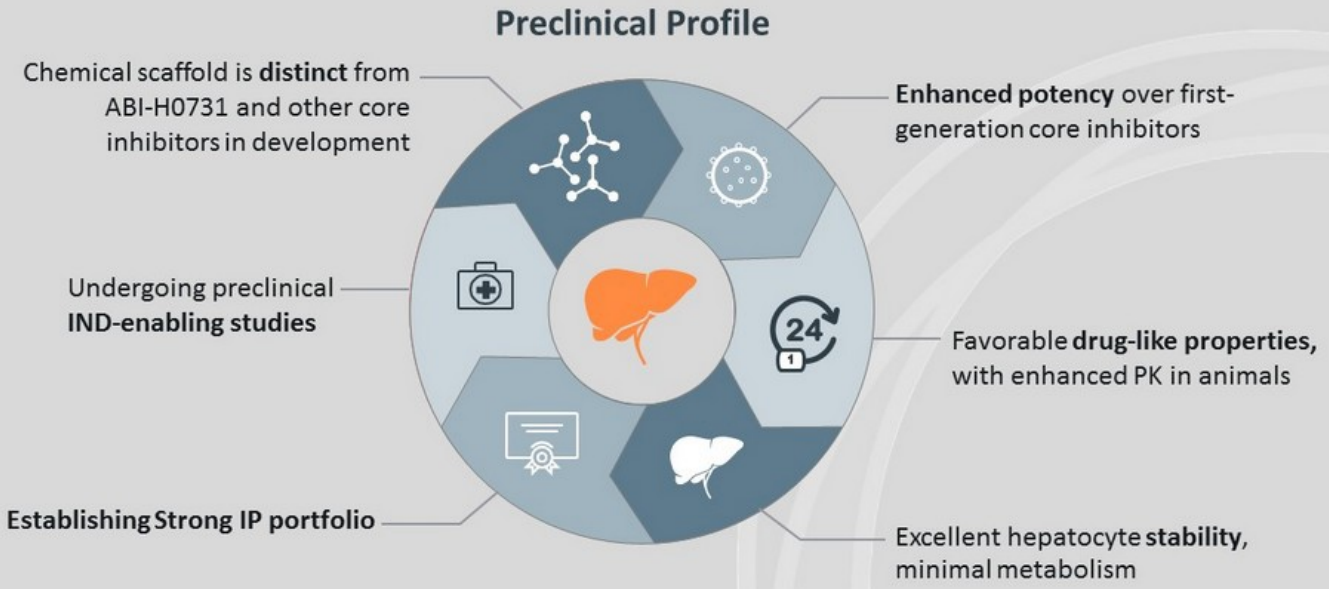
Yuen, et al. EASL Poster LBP-012 2018.

731 IS AS POTENT AS ANY THERAPY FOR HBV



*Estimated.
BID = twice a day; POC = proof of concept; QD = once a day; RTV = ritonavir.
1. Yuen, et al. AASLD Poster LB-10 2015. 2. Ding, et al. AASLD Poster 920 2017. 3. Zoulim, et al. AASLD Poster LB-15. 2017. 4. Gane, et al. EASL Presentation 2018. 5. Yuen, et al. EASL Poster LBP-012 2018. 6. De Man, et al. *Hepatology*. Vol 34 2001. 7. Agarwal J. *Hepatology*. 2015;vol 62.

ABI-H2158 – SECOND-GENERATION CORE INHIBITOR ADVANCING TO PHASE 1 STUDIES



IP = intellectual property.

ENHANCED PROPERTIES OF ABI-H2158

HBV Infection of Primary Human Hepatocytes

Viral Marker	EC_{50} (nM)		Fold Improvement
	ABI-H0731	ABI-H2158	
Viral DNA	154	41	**4**
HBeAg	2,210	204	**11**
HBsAg	3,000	216	**14**
pgRNA	1,840	160	**12**

- Both compounds are highly stable and effective in human hepatocytes
- **ABI-H2158 exhibits >11-fold enhanced potency in reducing surrogate markers of cccDNA**

PK Parameters at 30-mg/kg Dose

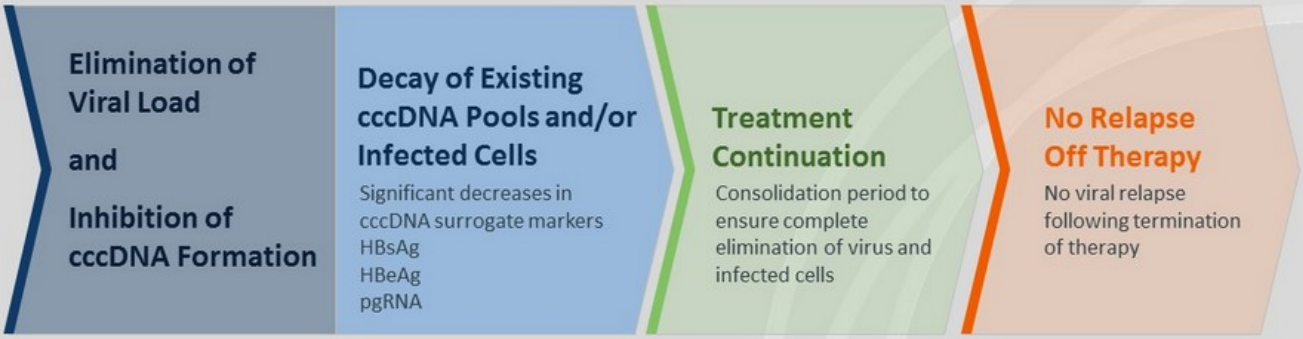
Animal Species	ABI-H0731	ABI-H2158	ABI-H0731	ABI-H2158
	C_{max} (µg/mL)	C_{max} (µg/mL)	AUC_{inf} (µg*hr/mL)	AUC_{inf} (µg*hr/mL)
Rat	6.4	24.8	62.1	445
Monkey	3.2	45.4	45.2	328
Dog	2.1	49.9	3.9	763

- Both compounds exhibit high bioavailability and terminal half-life supportive of QD human dosing
- **ABI-H2158 exhibits 4- to 24-fold increase in C_{max} and 7 to 195-fold increase in AUC_{inf} when dosed at 30 mg/kg in animals**

AUC_{inf} = area under the curve from zero to infinity; C_{max} = maximum concentration.

HBV CURE: CLINICAL COMPONENTS

Expected Treatment Components to Achieve Cure



ASMB CORE INHIBITOR PROGRAM SUMMARY

Core Inhibitors: Disrupt viral replication at multiple steps AND inhibit the generation of new cccDNA

ASMB Portfolio: Derived from multiple distinct and proprietary chemical scaffolds, exhibit balance of potency AND favorable drug-like properties

ABI-H0731
First-Generation Candidate

- Favorable safety and PK properties enabling QD dosing in patients
- Potent antiviral efficacy observed in phase 1b study in chronically infected HBV patients
- Dose level of 300 mg selected for phase 2a study (start mid-year)

ABI-H2158
Second-Generation Candidate

- Exhibits enhanced potency and PK properties, while retaining favorable drug-like properties
- Completing IND-enabling studies
- Phase 1a clinical trial expected to initiate in Q4 2018

Future combinations of core inhibitors and Nucs should result in


More rapid and deeper reduction in viral levels (eradication)


Decrease cccDNA levels


INCREASE CURE RATES

ABI-H0731 WILL BE AMONG THE FIRST CORE INHIBITORS EXPLORED IN AN HBV CURE PROGRAM

CORE INHIBITOR ROADMAP FOR FUNCTIONAL CURE

cccDNA = covalently closed circular DNA; ETV = entecavir; HBV = hepatitis B virus; nuc = nucleos(t)ide inhibitor; SVR = sustained viral response.

ABI-H0731 WILL BE AMONG THE FIRST CORE INHIBITORS EXPLORED IN AN HBV CURE PROGRAM

Phase 2a

300 mg 731 + 0.5 mg ETV

Goal: Significant improvement in viral load reduction over ETV-only arm

CORE INHIBITOR ROADMAP FOR FUNCTIONAL CURE

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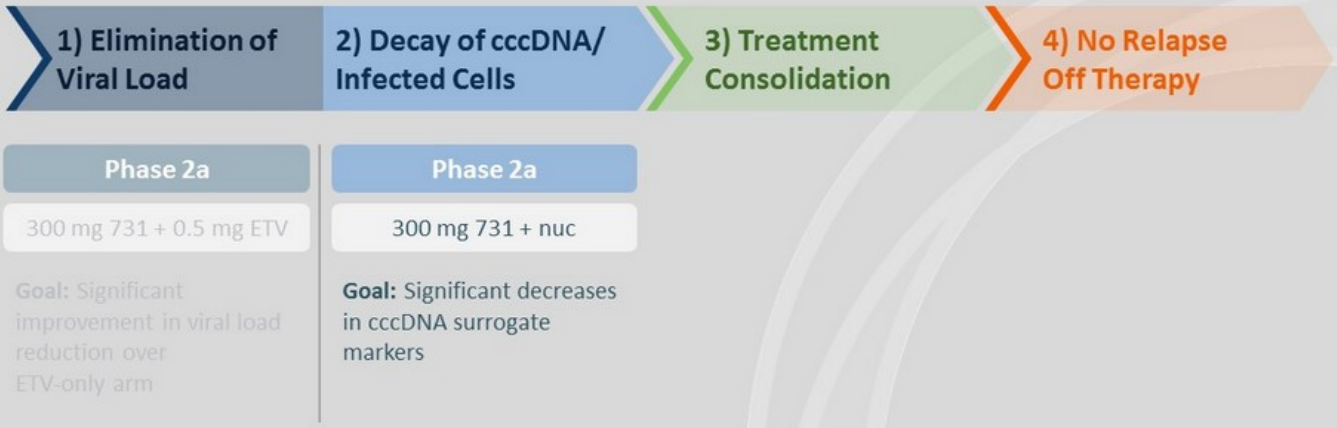

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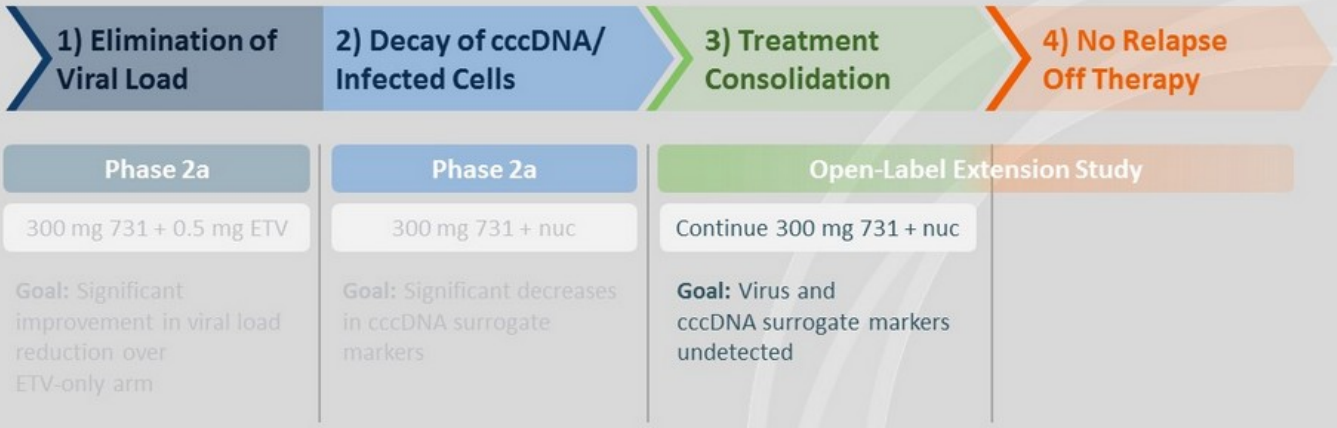

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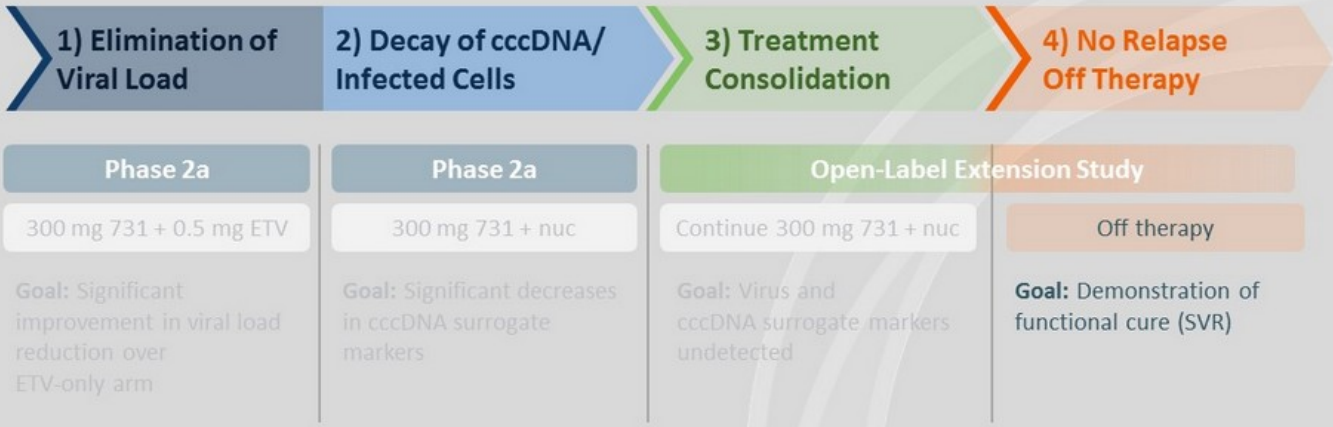

CORE INHIBITOR ROADMAP FOR FUNCTIONAL CURE

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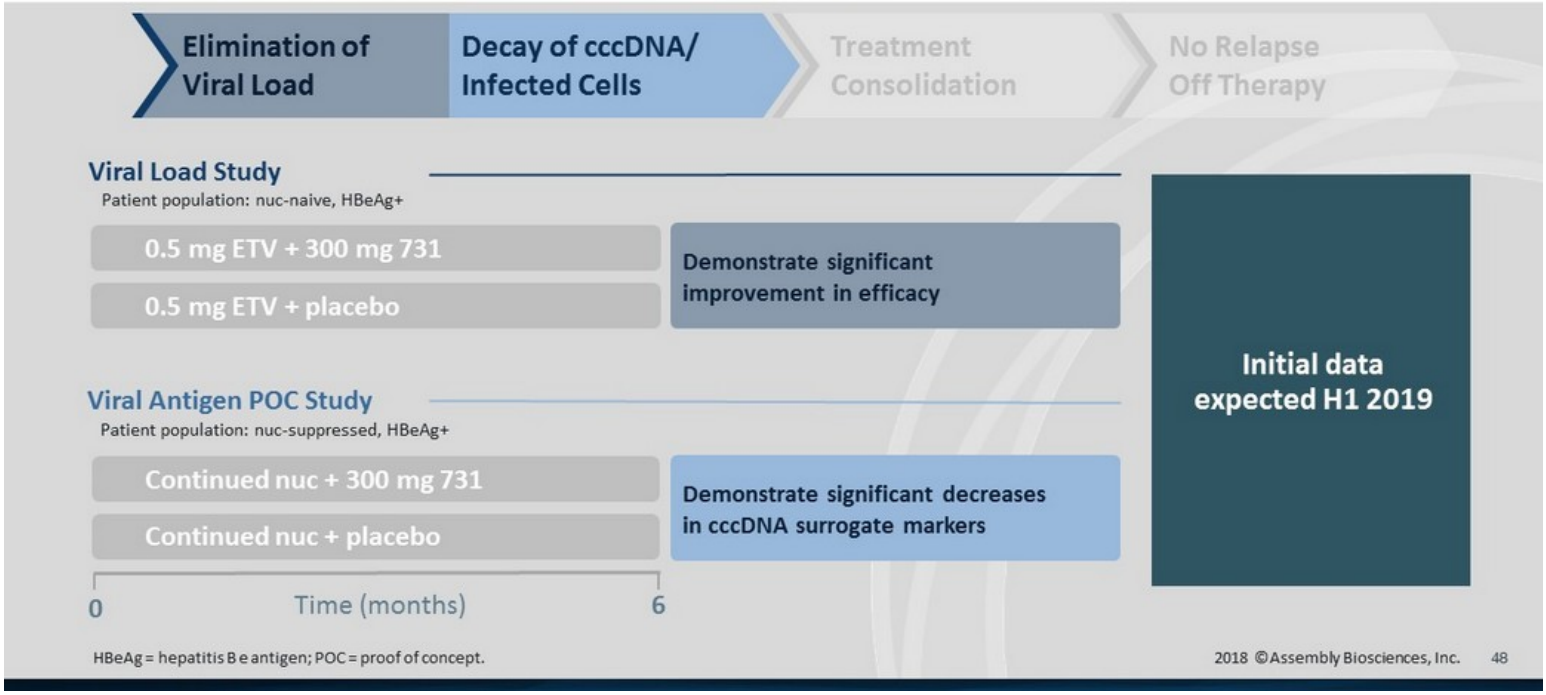
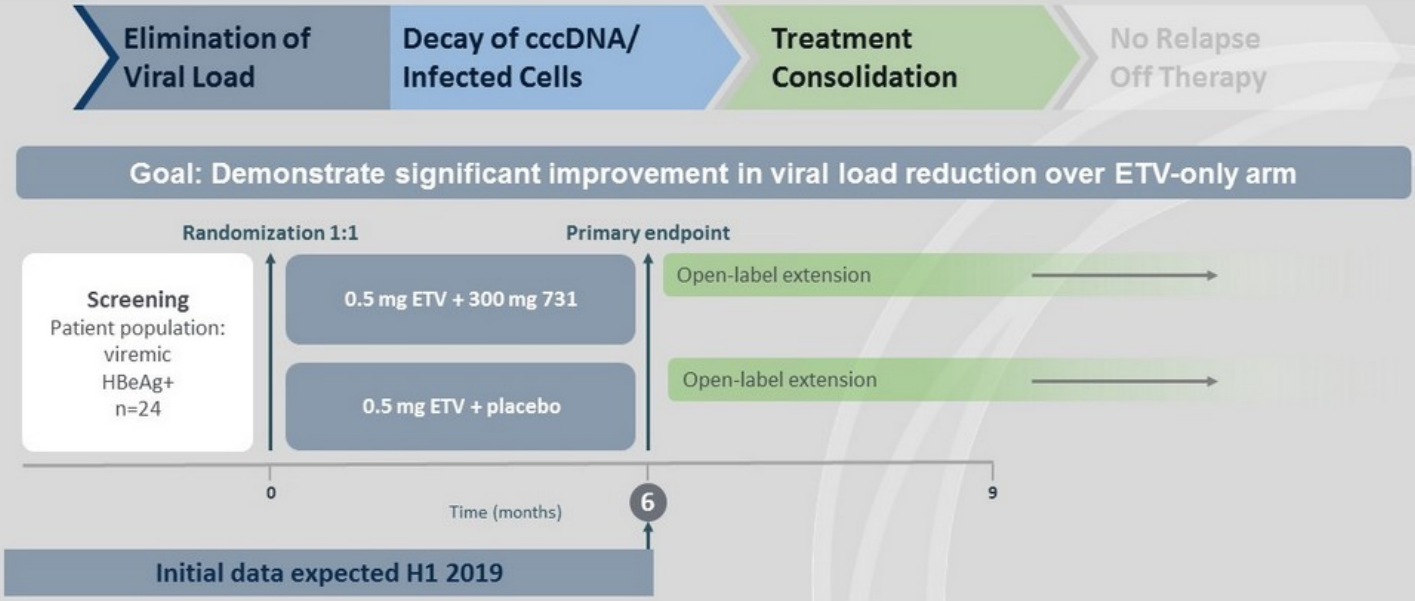
ABI-H0731 PHASE 2A STRATEGY AND DESIGNS

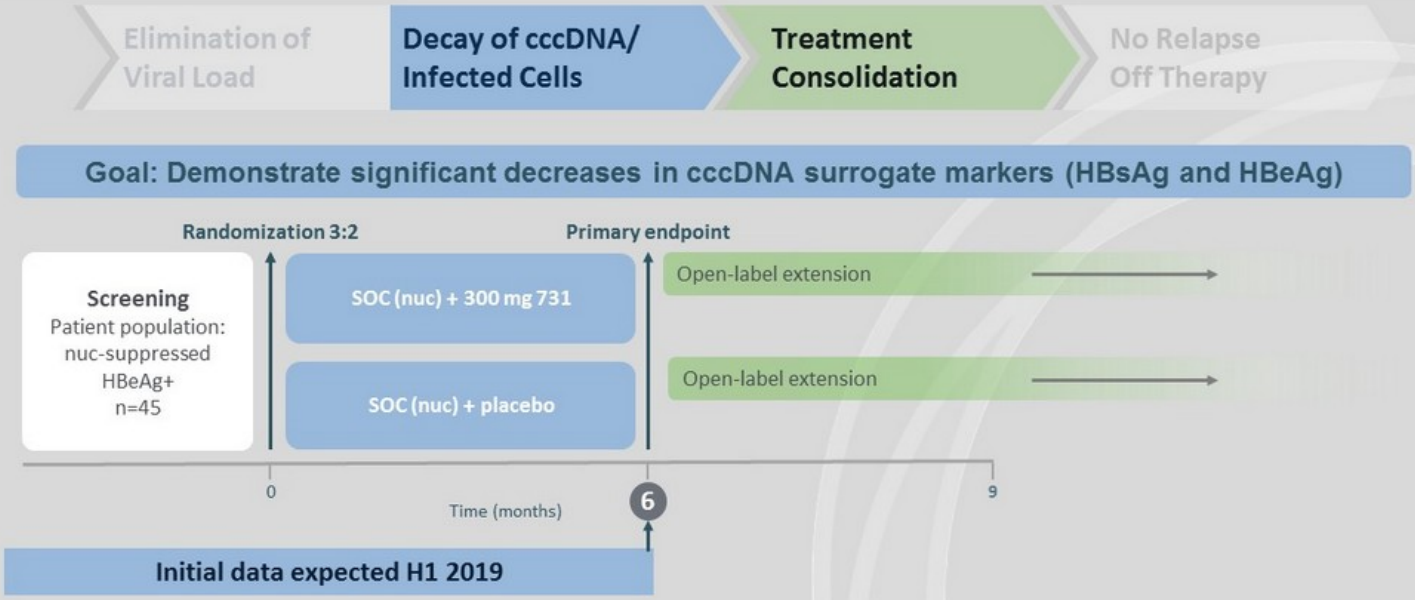


HBeAg = hepatitis B e antigen; POC = proof of concept.

ABI-H0731 VIRAL LOAD STUDY: COMBO THERAPY INHIBITS HBV LIFE CYCLE MORE COMPLETELY



ABI-H0731 VIRAL ANTIGEN POC STUDY: COMBO THERAPY DRIVES cccDNA LOSS



SOC = standard of care.

ABI-H0731 OPEN-LABEL EXTENSION STUDY
TEST OF CURE!

Goal: CONSOLIDATION = Eliminate the last vestiges of replicating virus while continuing to block new cccDNA and allow time for old cccDNA to decay

LLQ = lower limit of quantification.

ABI-H0731 OPEN-LABEL EXTENSION STUDY
TEST OF CURE!

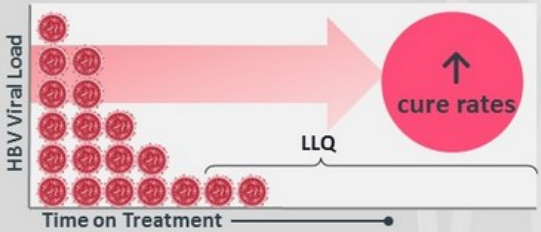
Elimination of Viral Load → Decay of cccDNA/ Infected Cells → Treatment Consolidation → No Relapse Off Therapy

Goal: CONSOLIDATION = Eliminate the last vestiges of replicating virus while continuing to block new cccDNA and allow time for old cccDNA to decay

Viral Load Study

Viral Antigen POC Study

Open-label 300 mg 731 + nuc



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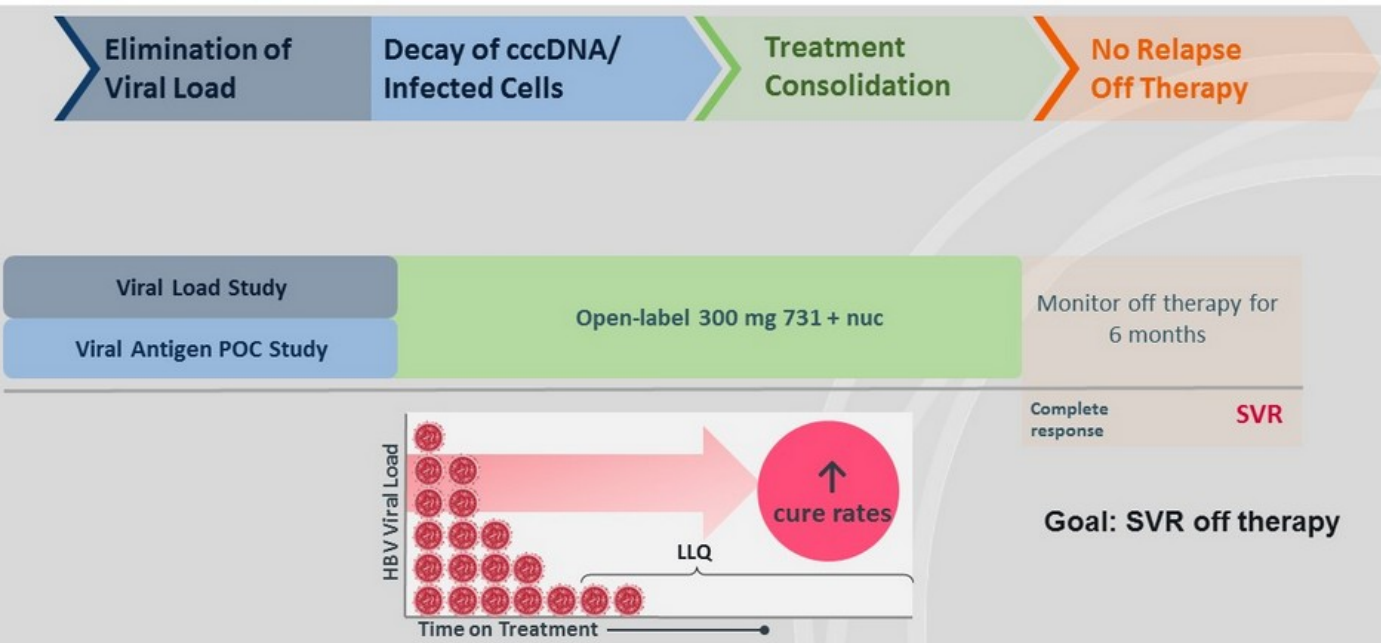
TEST OF CURE!



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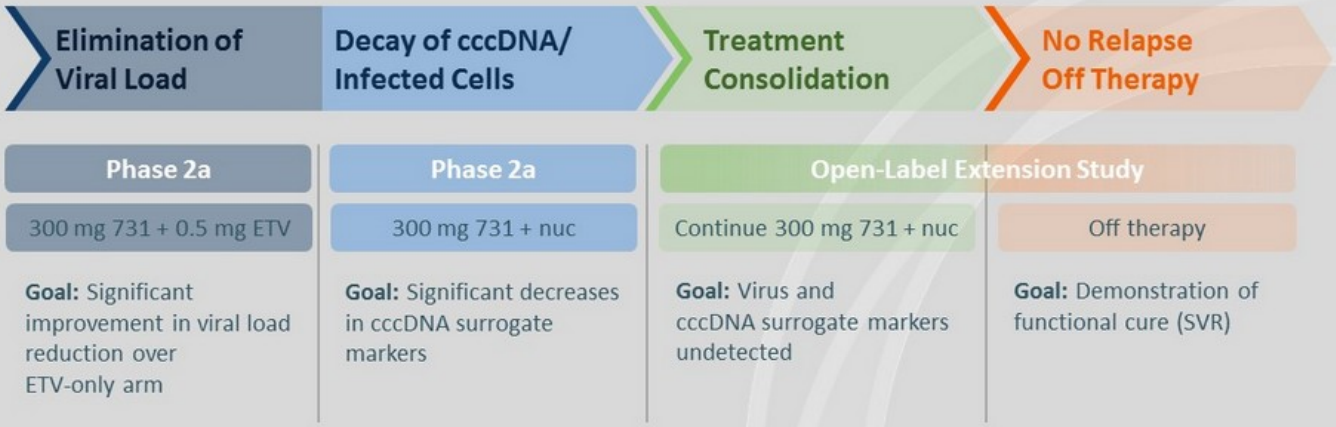
CORE INHIBITOR ROADMAP FOR FUNCTIONAL CURE

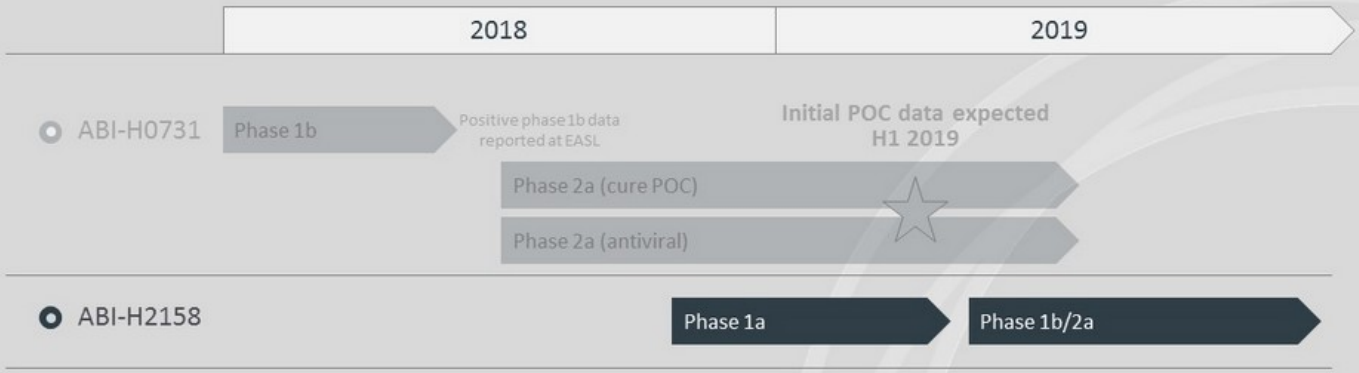


INITIAL DATA EXPECTED H1 2019

EXPECTED CLINICAL TIMELINES AND MILESTONES

EXPECTED CLINICAL TIMELINES AND MILESTONES

EXPECTED CLINICAL TIMELINES AND MILESTONES



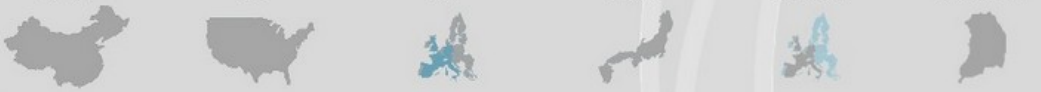
EXPECTED CLINICAL TIMELINES AND MILESTONES

CHRONIC HEPATITIS B: TODAY

SALES IN 2017 for chronic HBV:

$2.5 BILLION
even with **non-curative**, mostly **generic** agents

China	US	EU5	Japan	Rest of EU	South Korea
$963 MILLION	$621 MILLION	$375 MILLION	$237 MILLION	$172 MILLION	$157 MILLION



HBV = hepatitis B virus.
IQVIA.

CHRONIC HEPATITIS B: TOMORROW

SALES IN HBV
in the next decade

forecasted to grow
dramatically

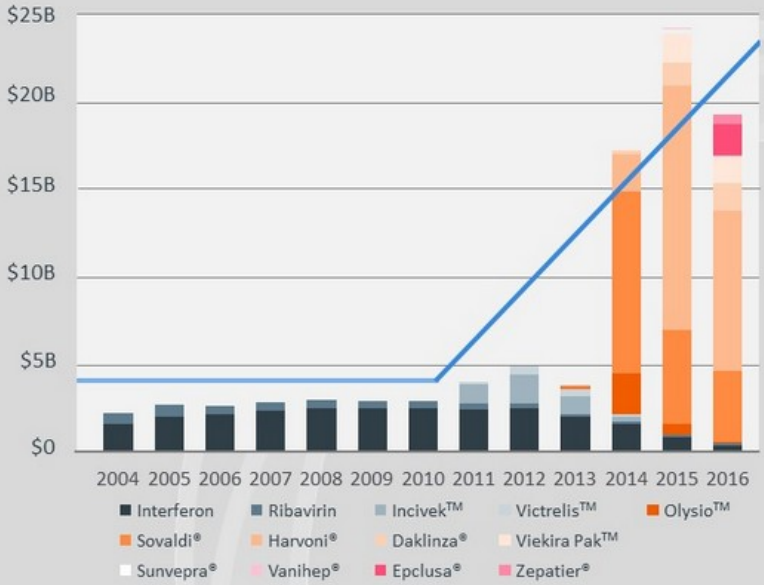
WHY?

- The prevalence of chronic HBV
- The crucial role of improved cure rates
  - As cure rates improve, diagnoses and treatment rates expected to increase
  - The HCV experience as analog

HCV = hepatitis C virus.
Data on file.

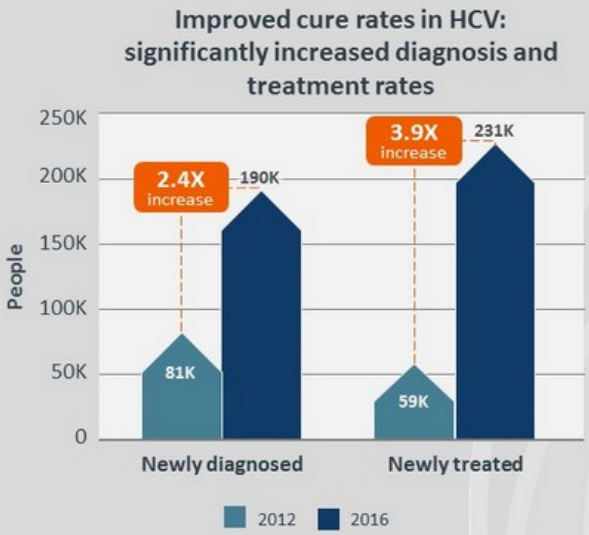
THE HISTORY OF HCV THERAPY

- In 2010, sales of the treatment for HCV (interferon and ribavirin) were $2.8 billion
  - SVR was ~50%
- The first generation of DAAs launched in 2011 and brought SVR to ~75%
- But patients were warehoused in anticipation of even better DAAs



DAA = direct acting antiviral; SVR = sustained viral response.
Evaluate Ltd. 2017.

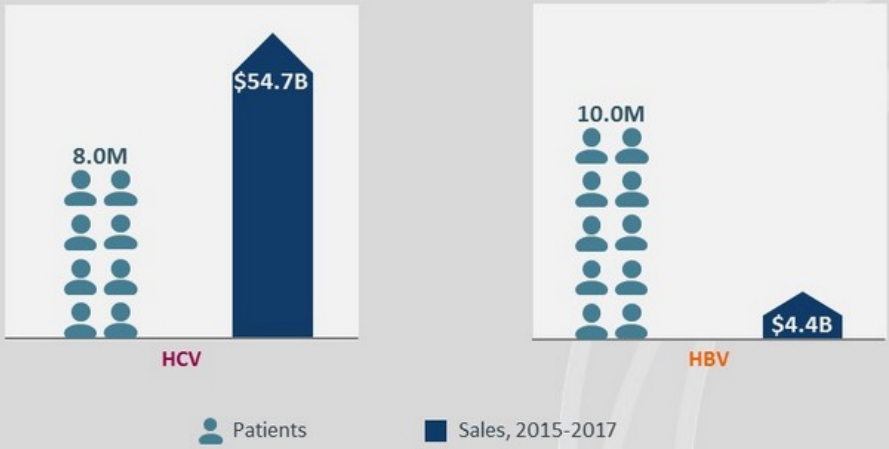
THE HCV EXPERIENCE...



1. Gilead Sciences, Inc. Q2 2017 earnings results. 2. Gilead Sciences, Inc. Q4 2016 earnings results. 3. Healthcare Analytics.

LACK OF A CURE HAS LIMITED THE UPTAKE OF TREATMENT

Although there are more HBV patients than HCV patients in the developed countries, treatment has lagged



CDC, ECDC, IMS, Evaluate Ltd.

TREATMENT COULD ULTIMATELY LOOK SIMILAR

		Cure	Tolerability	Duration		Cure	Tolerability	Duration
HCV	Pre-2011 (pre-DAAs)	~50%	Very poor	48 weeks	Today	~100%	Very good	12 weeks
HBV	Today	~10%	Very good	Lifetime	Goal	Significantly better	Very good	Defined duration

HBV BY THE NUMBERS

$2.5 BILLION

The market in 2017 for **nucs used in HBV** in the US, Europe, China, Japan, and Korea

>250 million

people with chronic HBV

1 %

estimated number of **CHRONIC HBV patients** currently treated in these markets

2x HBV-induced deaths compared to HCV-induced deaths

HBV is the cause of an enormous **burden of disease** (cirrhosis, HCC) in healthcare systems

HCC = hepatocellular carcinoma; nuc = nucleos(t)ide inhibitor.
Data on file.

CLOSING REMARKS

assembly
biosciences