R&D Day
Hepatitis B Program

June 20, 2018

NASDAQ: ASMB

assembly biosciences

Derek Small

President and Chief Executive Officer

assembly biosciences

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.

AGENDA

11:30AM – 11:50AM	**Opening Remarks** Derek Small Chief Executive Officer
11:50AM – 12:15PM	**Douglas Dieterich, MD** Professor of Medicine Division of Liver Diseases Director Institute for Liver Medicine Mount Sinai School of Medicine
12:15PM – 12:40PM	**Jörg Petersen, MD, PhD** Professor of Medicine and Head of the Liver Unit IFI Institute for Interdisciplinary Medicine Asklepios Klinik St. George, University of Hamburg
12:40PM – 1:30PM	**R&D Overview and Clinical Data Hepatitis B Program** Richard Colonno, PhD EVP & Chief Scientific Officer of Virology Operations
1:30PM – 1:45PM	**Clinical Development Hepatitis B Program** Uri Lopatin, MD Chief Medical Officer
1:45PM – 2:00PM	**Commercial Perspectives on Hepatitis B** JP Benya Vice President, Commercial
2:00PM – 2:25PM	**Management and KOL Q&A Session** Speakers: Dr. Dieterich, Dr. Petersen, Derek Small, Dr. Richard Colonno, Dr. Uri Lopatin, JP Benya

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

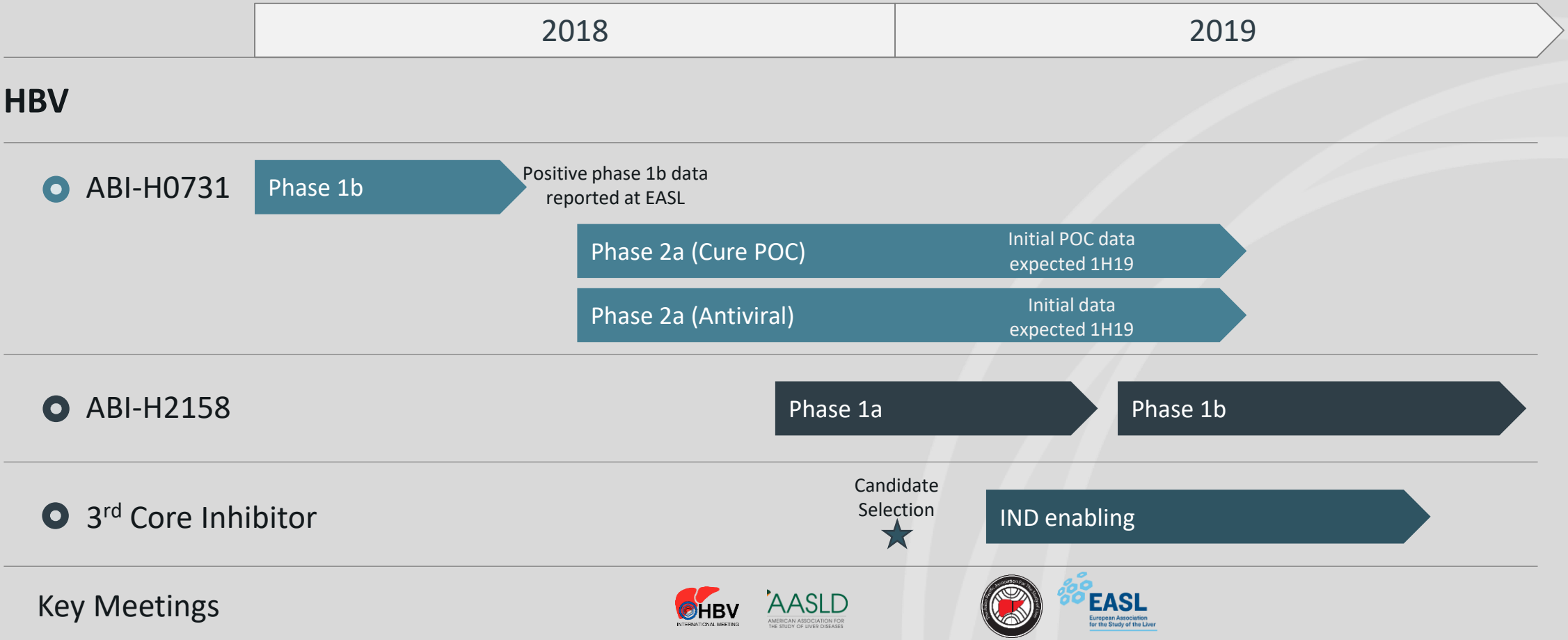
Drug Candidate (Mechanism/Indication)	Discovery & Lead Op/Selection	IND Enabling	Phase 1a	Phase 1b	Phase 2	Phase 3	NDA/BLA	Worldwide Rights
HBV Cure								
ABI-H0731 (Core Inhibitor)								
ABI-H2158 (Core Inhibitor)								
Third Core Inhibitor								
Microbiome								
ABI-M201 (Ulcerative Colitis)								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

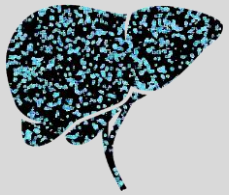
>250 million chronically infected worldwide

8% diagnosed

<1% receive treatment

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

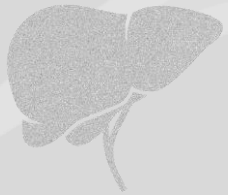
Cirrhosis/HCC



20%-30%

Surgery, chemotherapy, and liver transplant

Death



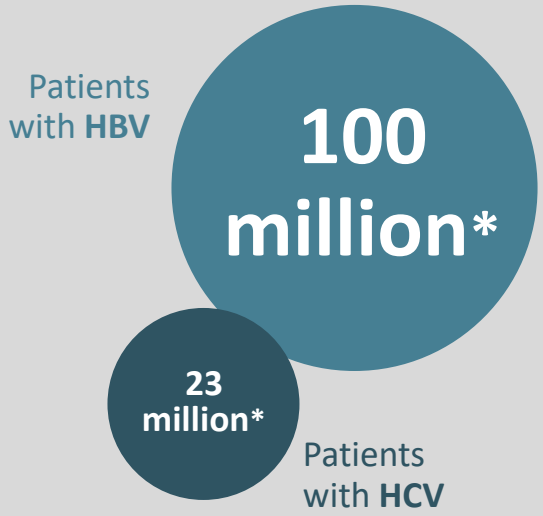
~1 million people/year

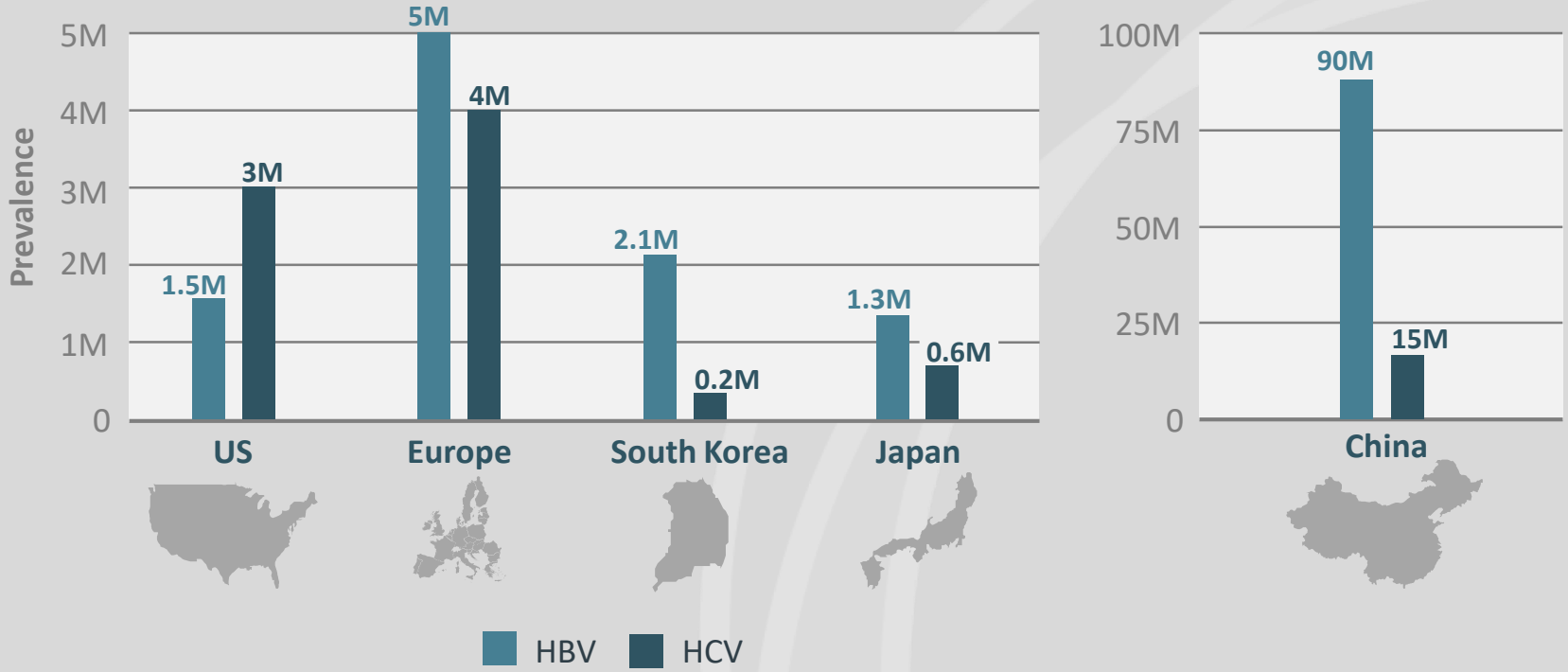
2 people/minute

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

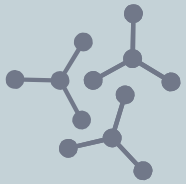
- China has made significant advances in regulatory, IP and business policies over the last few years
- Assembly China efforts began in 2015
  - ASMB China headquarters in Shanghai (Zhangjiang Hi-Tech Park)
  - Launching a regulatory office in Beijing
- Currently establishing a dedicated team for our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

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DOUGLAS DIETERICH, MD

Professor of Medicine
Division of Liver Diseases
Director Institute for Liver Medicine
Mount Sinai School of Medicine

Icahn School of Medicine at Mount Sinai

Mount Sinai Institute for Liver Medicine

DISCLOSURES

Grant/research support: Abbvie, Gilead, Merck

Clinical studies: Abbvie, Gilead, Merck

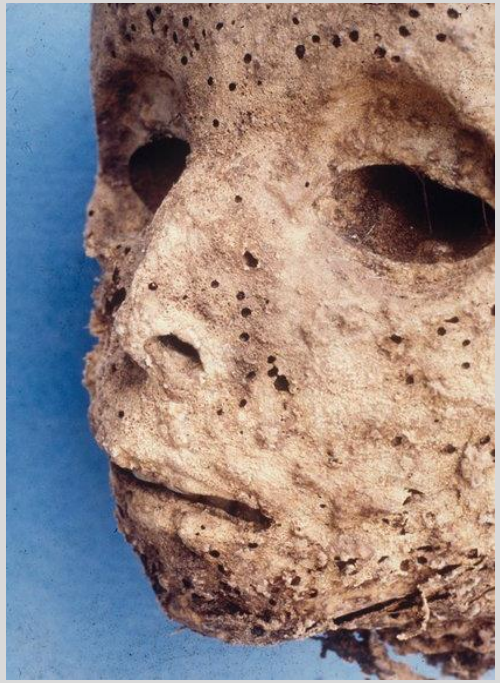
Consultant/advisor: Abbvie, Arbutus, Assembly, Gilead, Intercept, Merck

Sponsored lectures: Abbvie, Gilead, Intercept, Merck

HOW OLD IS HBV?

The Paradox of HBV Evolution As Revealed From a 16th Century Mummy

Zoe Patterson Ross, Jennifer Klunk, Gino Fornaciari, Valentina Giuffra, Sebastian Duchêne, Ana T. Duggan, Debi Poinar, Mark W. Douglas, John-Sebastian Eden, Edward C. Holmes , Hendrik N. Poinar



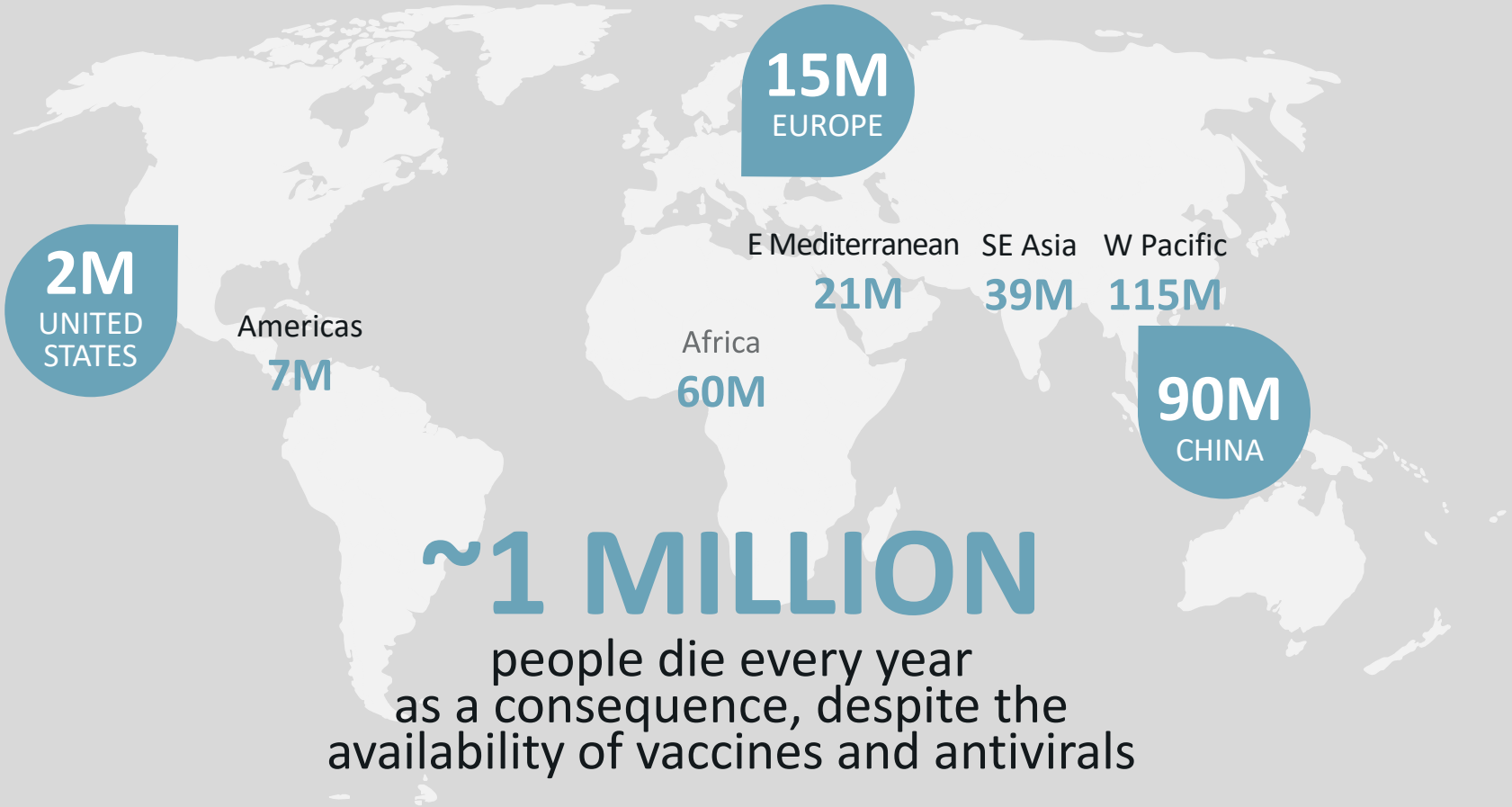
- 16th century Italian mummy
- Previously thought to have smallpox
- No variola DNA identified, but HBV DNA identified
- Phylogenetically closely related to present-day genotype D
- HBV genotypes diversified long before the 1500s
- HBV evolution evades molecular clock to date its origin

HBV = hepatitis B virus.

PLOS Pathogens. http://journals.plos.org/plospathogens/article?id=10.1371/journal.ppat.1006750. January 4, 2018.

TREMENDOUS MEDICAL NEED

257 million people are chronically infected with HBV globally



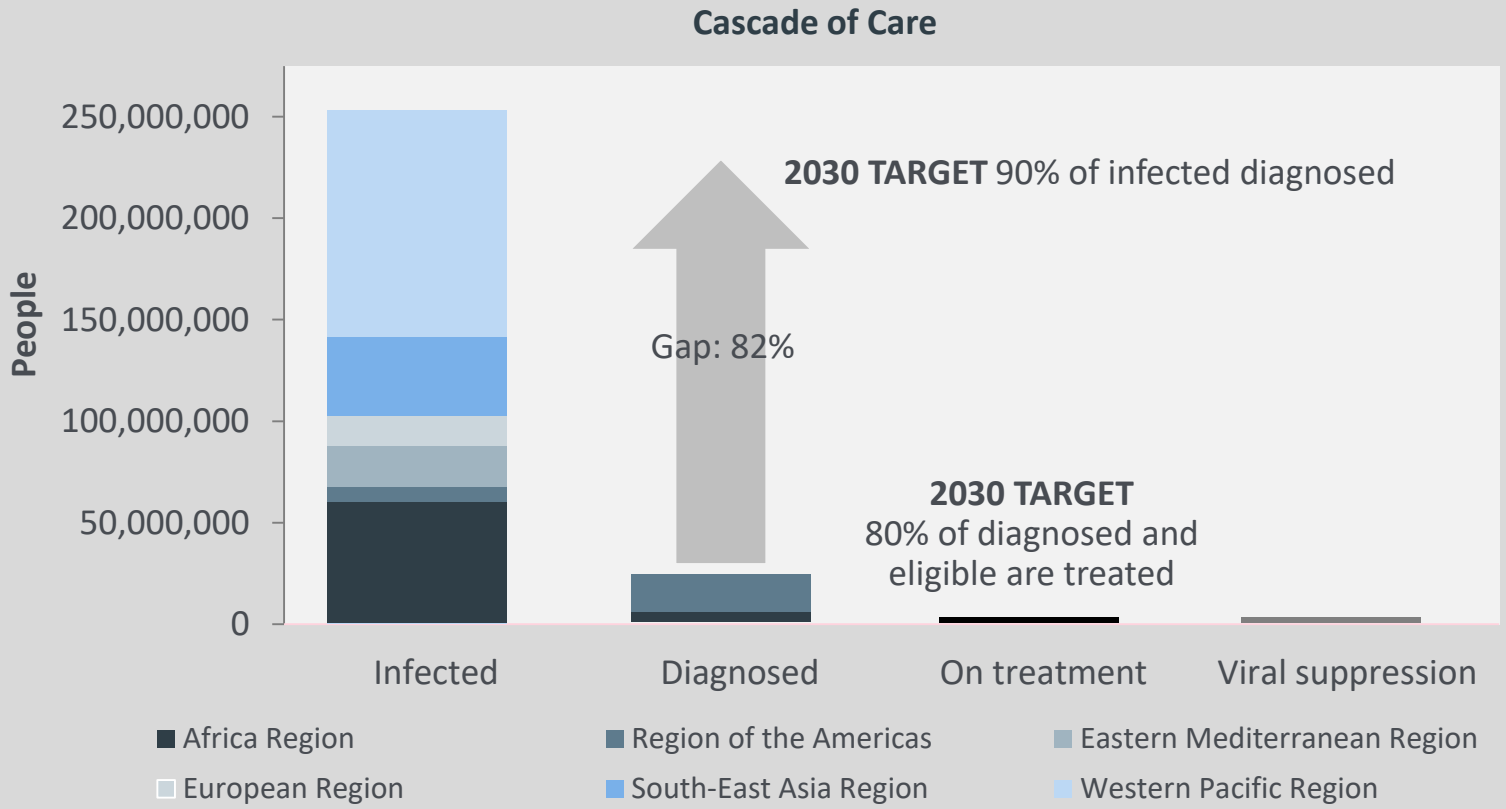
~1 MILLION

people die every year
as a consequence, despite the
availability of vaccines and antivirals

Hepatitis B. WHO (2017). http://www.who.int/mediacentre/factsheets/fs204/en/.

FEW PATIENTS SEEKING TREATMENT – WHY?

HBV cascade: only 9% of 257 million people are diagnosed



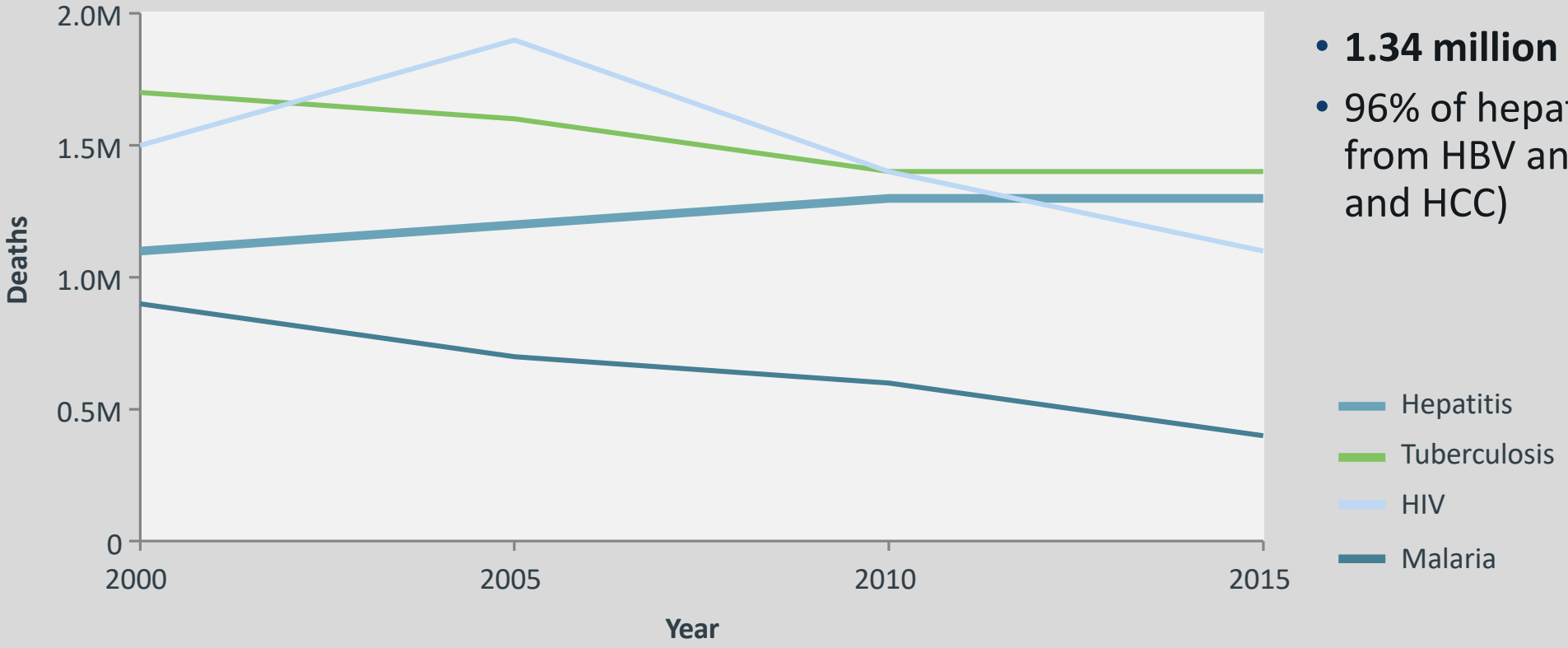
1.7 MILLION ON TREATMENT IN 2015

- Not interested in starting a lifelong therapy, even though we know this decreases probability of serious liver complications
- Do not feel sick so do not want to start treatment
- Asking physicians for when an HCV-like cure option for HBV will be available

HCV = hepatitis C virus.
Hepatitis B. WHO (Center for Disease Analysis).

HBV REMAINS A SERIOUS DISEASE

Mortality is increasing in hepatitis A, B, E, and C



- **1.34 million deaths in 2015**
- 96% of hepatitis deaths are from HBV and HCV (cirrhosis and HCC)

HCC = hepatocellular carcinoma.
Hepatitis B. WHO (Center for Disease Analysis).

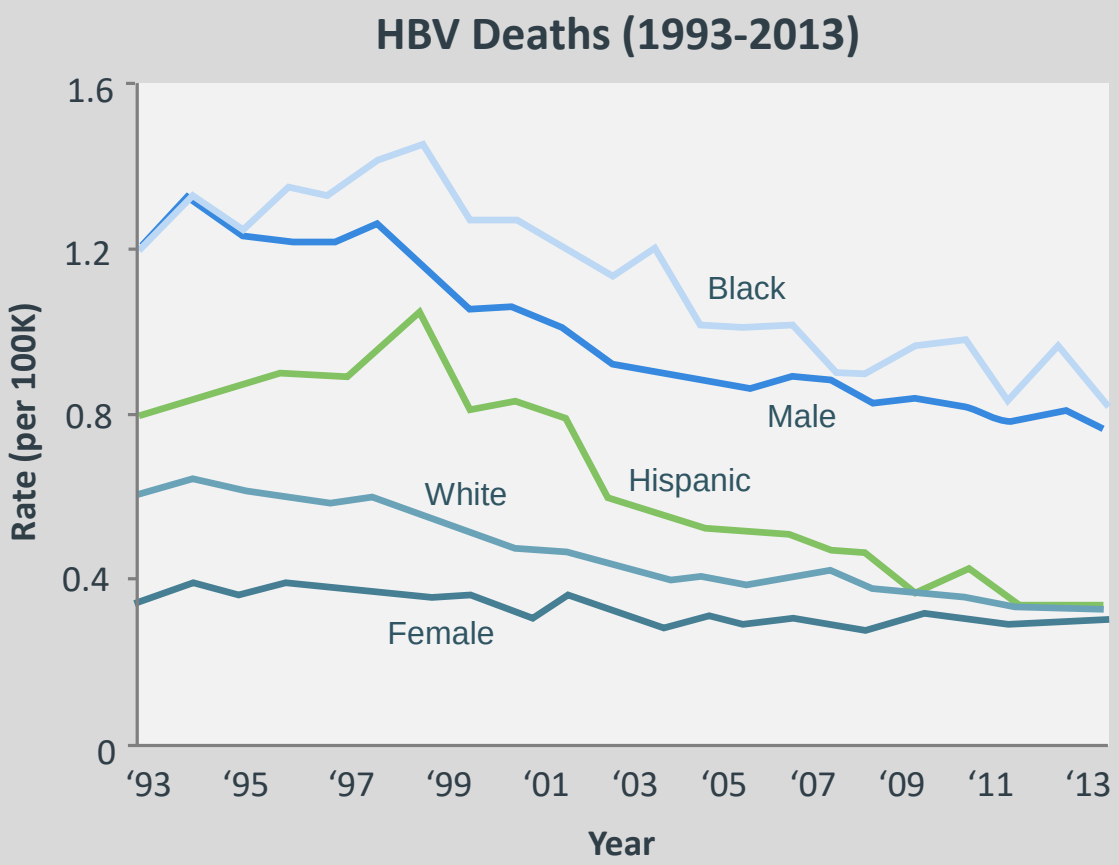
BURDEN OF HBV IN THE US POPULATION

National databases

- National Ambulatory Care Survey (1992-2012)
- Healthcare Cost and Utilization Project National Inpatient Sample (1993-2013)
- Vital Statistics: Multiple Cause-of-Death Data (1993-2014)

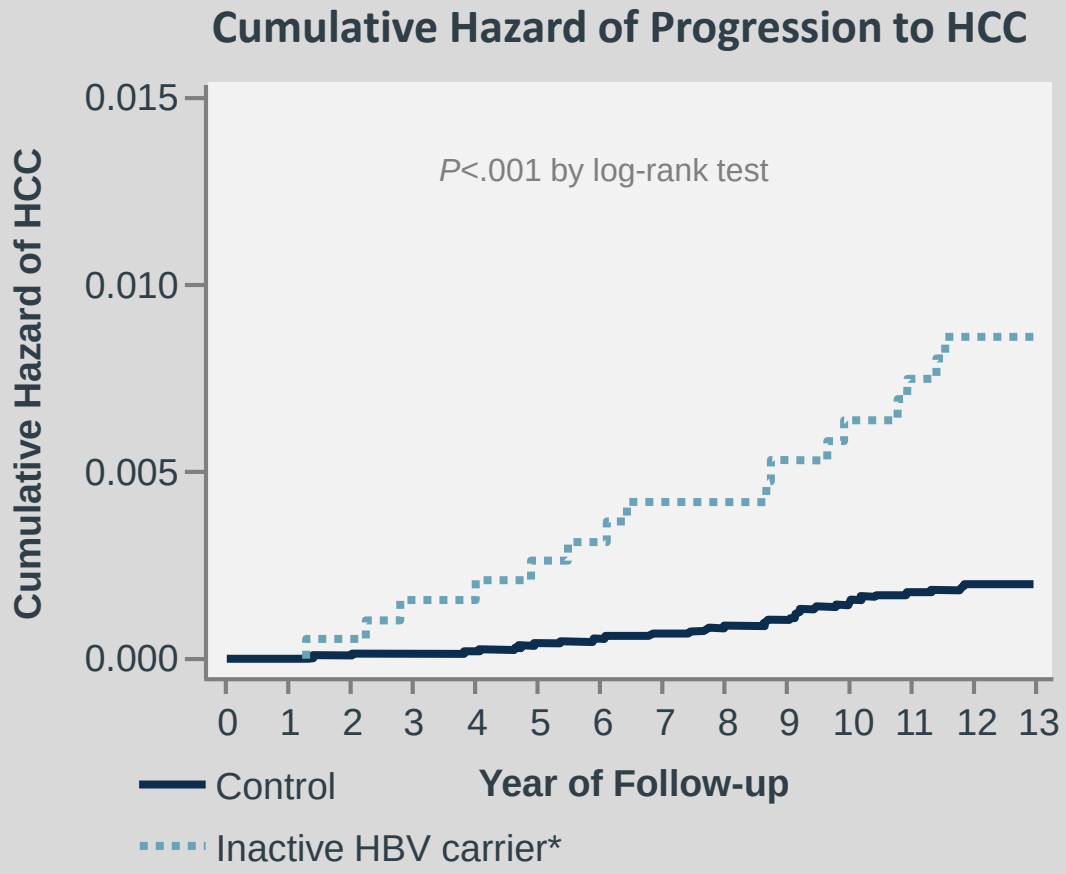
HBV deaths (2014)

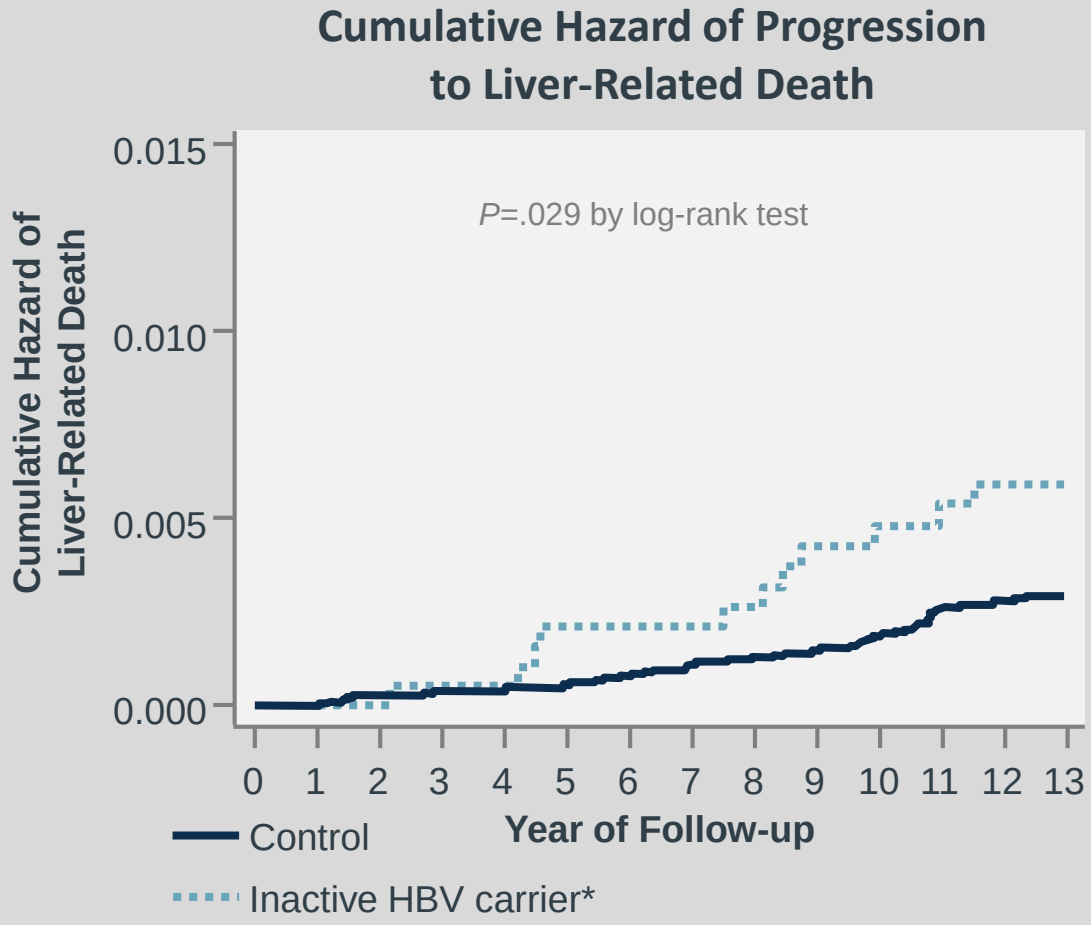
- 2000 deaths (mortality rates decline among all sex, race, and ethnic groups)
- Higher mortality rates (per 100K)
  - Men vs women: 0.7 vs 0.3
  - Black vs white: 0.8 vs 0.3
  - Non-Hispanic vs Hispanic: 0.5 vs 0.3



Unalp-Arida A, et al. *Hepatology*. 2016;64(suppl S1):391A. Abstract 793.

REVEAL: PROGRESSION TO HCC AND LIVER-RELATED DEATH IN INACTIVE HBV CARRIERS*

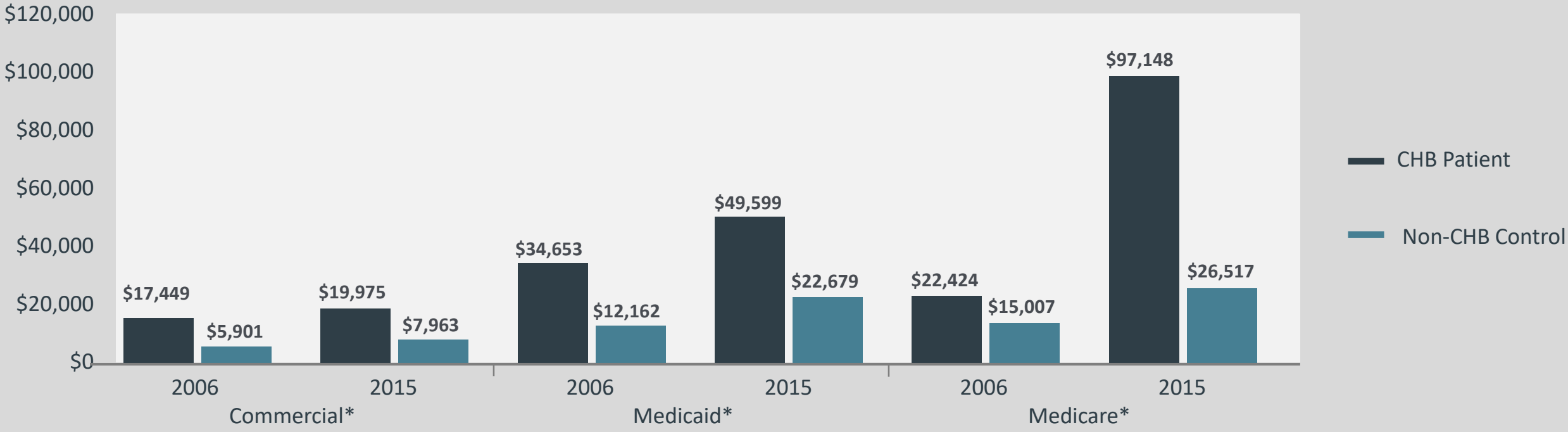




*Inactive carriers defined as HBV DNA <10,000 copies/mL and ALT <45 U/L.
ALT = alanine transaminase.
Chen JD, et al. *Gastroenterology*. 2010;138(5):1747-1754.

SIGNIFICANT COST OF HBV INFECTION

All-Cause Annual per Patient Total Healthcare Costs Among CHB Patients and Non-CHB Controls Over Time (2006 – 2015)



The total all-cause healthcare costs among patients with CHB were significantly higher than non-CHB controls across all payers and years (all *P*<.05). Notably, in 2015, Medicare patients with CHB had total all-cause healthcare costs, which were nearly 4-fold higher than Medicare non-CHB controls.

**P*<.05 for all comparisons of patients with CHB vs non-CHB controls.
CHB = chronic hepatitis B.

IS AN HBV CURE POSSIBLE?

Are we on the cusp of a paradigm shift in HBV treatment?

Current Paradigm

- Indefinite treatment
- Poor off-Rx response
- Reduces overall mortality
- Reduces but does not eliminate the risk of HCC
- Potent nucs suppress viral replication but **cannot cure the disease**

New Paradigm

- Finite treatment duration
- Sustained off-Rx response shift toward endpoint of true immune control and HBsAg seroconversion
- No increased risk of mortality and HCC
- New HBV treatments with increased chance of curing disease

HBsAg = hepatitis B s antigen.

RECENT EXCITING DEVELOPMENTS

- cccDNA half-life deserves to be revisited as prior reports were based on overly optimistic expectations from nucs
- For the first time, new direct-acting antivirals that target different parts of the HBV life cycle are available
- Core inhibitors from Assembly Biosciences, JNJ and Roche showed antiviral potency similar to ETV, TDF, and TAF in monotherapy 28-day studies

cccDNA = covalently closed circular DNA; ETV = entecavir; nuc = nucleos(t)ide inhibitor; TAF = tenofovir disoproxil fumarate; TDF = tenofovir disoproxil fumarate.

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MANAGEMENT OF HBV IN 2018: LOOKING TO THE FUTURE

Jörg Petersen, MD, PhD
Professor of Medicine and Head of the Liver Unit
IFI Institute for Interdisciplinary Medicine
Asklepios Klinik St. George, University of Hamburg

DISCLOSURES

Grant/research support: Bristol-Myers Squibb, Novartis, Roche

Clinical studies: AbbVie, Arrowhead, Bristol-Myers Squibb, Eisai, Falk, Gilead Sciences, Hepatera, Hologic, Intercept, Janssen, Merck Sharp & Dohme, Roche, Siemens, Vertex

Consultant/advisor: Abbott, AbbVie, Arrowhead, Assembly Biosciences, Bristol-Myers Squibb, ContraVir, Gilead Sciences, GlaxoSmithKline, Janssen, Kedrion, Merck Sharp & Dohme, Novira, Roche

Sponsored lectures: Abbott, Bristol-Myers Squibb, Boehringer Ingelheim, Gilead Sciences, Janssen, Kedrion, Merck Sharp & Dohme, Merz, Novartis, Roche

GLOBALLY, AN ESTIMATED 250 MILLION PEOPLE ARE INFECTED WITH HBV

It is estimated that 15 million people in Europe are living with HBV

Region (Prevalence of HBsAg [%])

- African region (3.0)
- Region of the Americas (0.2)
- Eastern Mediterranean region (1.6)
- European region (0.4)
- Southeast Asian region (0.7)
- Western Pacific region (0.9)

HBsAg = hepatitis B s antigen; HBV = hepatitis B virus.

WHO. Global hepatitis report 2017. Available at: http://apps.who.int/iris/bitstream/10665/255016/1/9789241565455-eng.pdf?ua=1 (accessed November 2017).

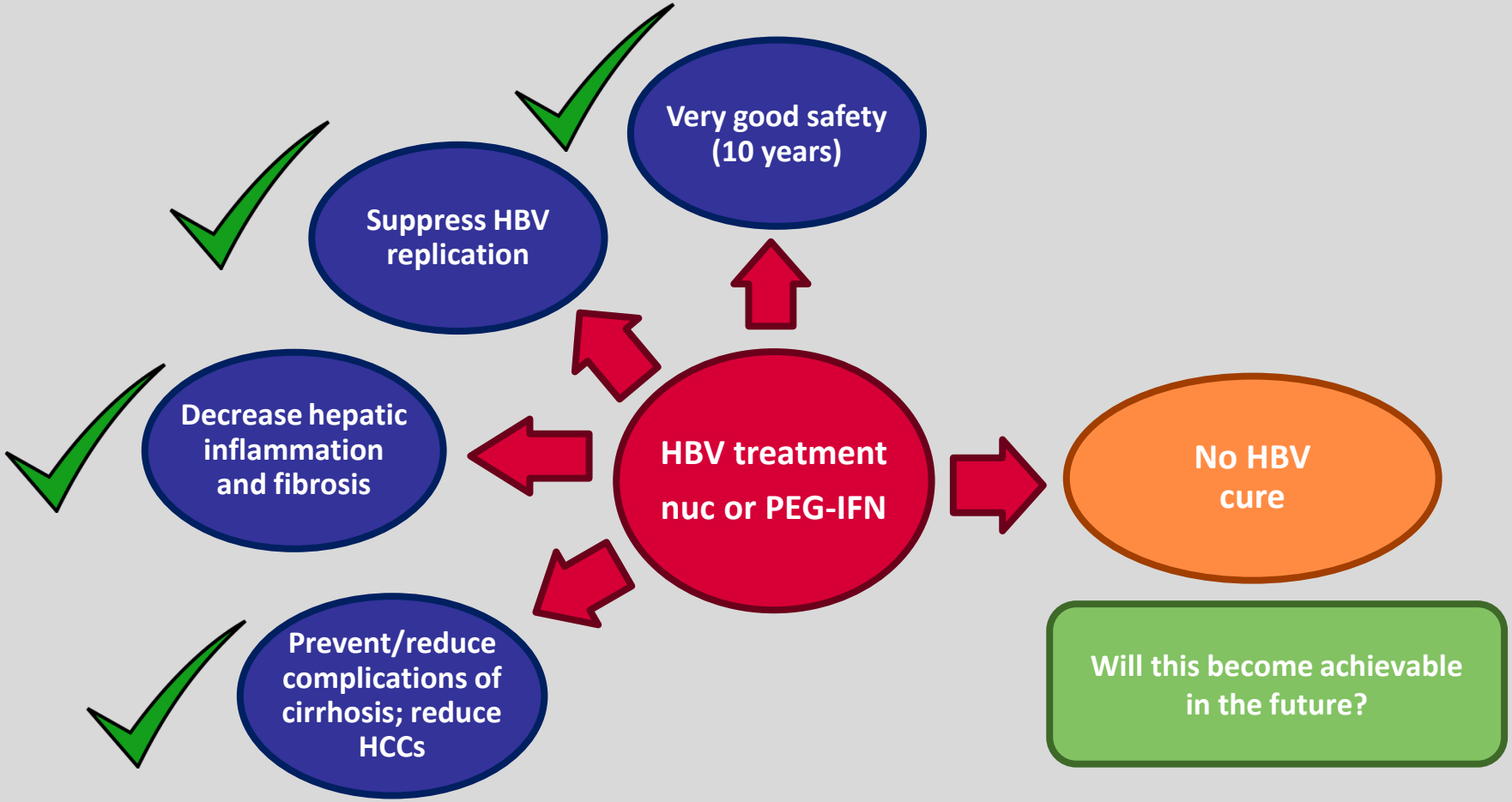
MIGRATION STATUS AND HBV INFECTION

	The Netherlands	Germany	Finland	Denmark	Sweden	UK
Foreign born/non-citizens in total population (%), 2003[32]	10.1/4.2	12.5/8.9	2.5/1.7	6.8/5.0	12.0/5.3	8.3/4.5
Percent of three largest migrant groups in foreign born 2001–03[6]	Turkey 11.1, Suriname 11.0, Morocco 9.5	Turkey 26.1, Italy 8.3, Former Yugoslavia 8.1	Russia 23.9, Sweden 18.8, Estonia 6.2	Turkey 9.3, Germany 6.8, Iraq 6.0	Finland 18.8, Former Yugoslavia 7.1, Iraq 5.4	Ireland 10.9, India 9.6, Pakistan 6.6
HBsAg+ in three largest migrant groups (%) (2010)[31]	4.0	4.0	4.6	4.0	4.0	4.0
HBsAg+ in general population (%) 2005–07[30]	0.3	0.7	0.23	0.01	0.03	0.3
Under-reporting estimate (%)	60[33]	80[34]	No info	50[30]	No info	25[30]
Immigrant indicator/link to population statistic[a]	Country of birth of the infected and the mother/No	No/No	Country of birth/Yes	Migrant or country of origin/Yes	Country of birth/Yes	Ethnic group (acute HBV)/No

a: Source: European Centre for Disease Prevention and Control[30] and expert interview.

Chu JJQ et al, European J Public Heath 2012

HBV: FROM A TREATABLE DISEASE TO A CURABLE DISEASE?



HCC = hepatocellular carcinoma; nuc = nucleos(t)ide inhibitor; PEG-IFN = pegylated interferon.

1. Liaw YF, et al. *N Engl J Med.* 2004;351:1521-1531. 2. Marcellin P, et al. *Lancet.* 2013;381:468-475. 3. Dandri M, Petersen. *J Clin Infect Dis*. 2016;62:281-288.

HBV: FROM A TREATABLE DISEASE TO A CURABLE DISEASE?

Challenges to Overcome in Both Diseases

HCV

- No suppressive therapy, and older therapies were both ineffective (<50%) and poorly tolerated
- Multiple enzymatic targets
- RNA life cycle with short half-life
- Cure possible without immune modulators or flares

HBV

- Well tolerated; very effective; suppressive therapy only
  - Very low cure rate (<10%)
- Single enzymatic target
- Intranuclear DNA life cycle with unknown half-life
- Immune control
  - Common in adults; rare in infants
  - Flares

CONSIDERATIONS FOR CURRENT TREATMENT OPTIONS

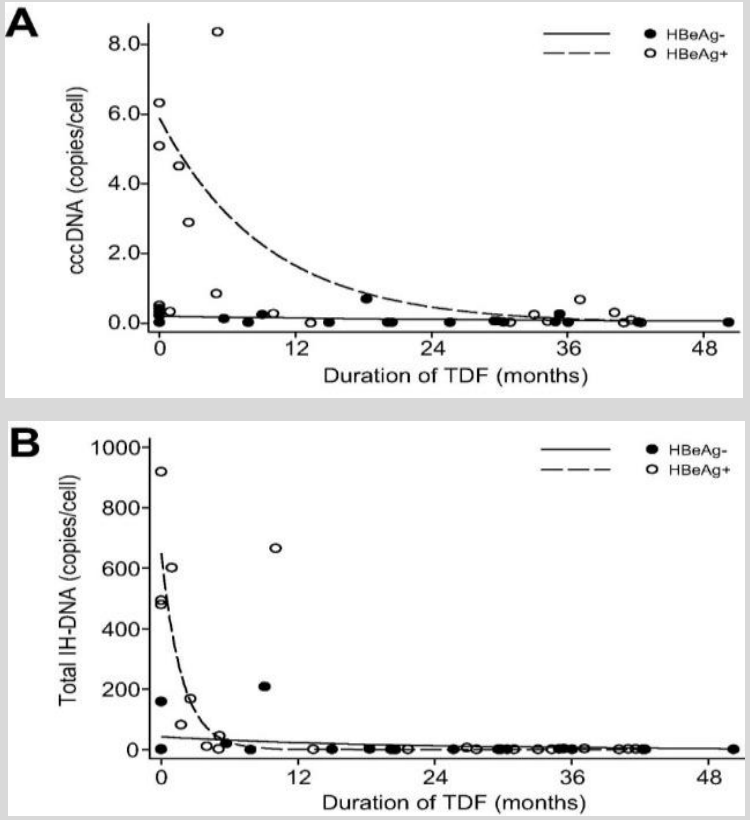
HBV remains an area of medicine that can be improved

- **Current treatments are not curative**
  - HBV DNA not fully suppressed
  - cccDNA establishment not affected
- **Definition of *cure***
  - Functional cure vs eradication of the virus
- **Endpoint simplification is needed**
  - HBsAg loss as endpoint of CHB therapy
  - Phase 3 requirements: endpoints and biomarkers
- **Presentations at EASL 2018 meeting in Paris**
  - New therapeutic targets and possible combination therapies
  - Similar to HCV in 2008
  - Large pharmaceutical companies navigating back toward HBV with new drug combinations

cccDNA = covalently closed circular DNA; CHB = chronic hepatitis B; EASL = European Association for the Study of the Liver.

WHY DO OUR CURRENT TREATMENT OPTIONS NOT RESULT IN COMPLETE CURE?

HBV DNA is not fully suppressed

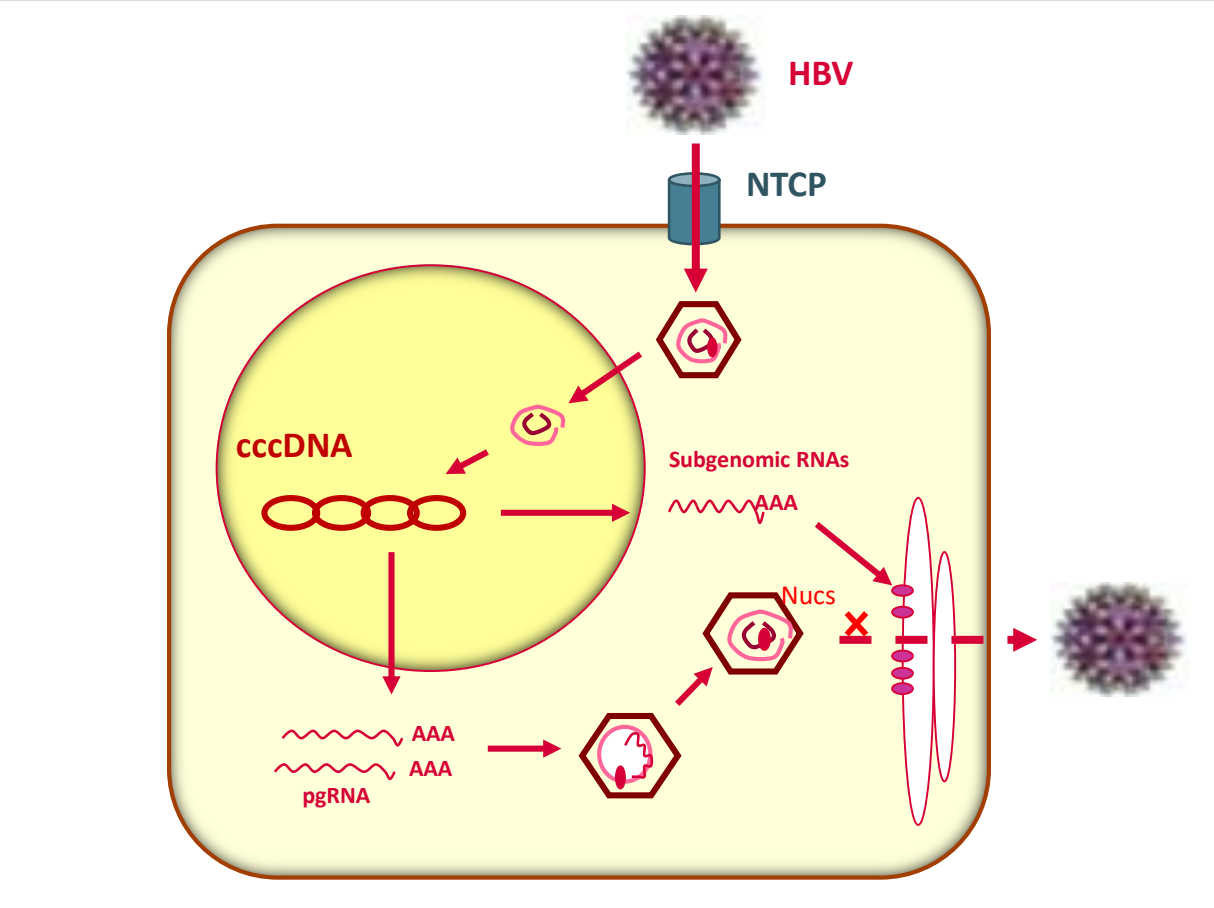


- Several recent studies demonstrated persistence of HBV DNA or intrahepatic HBV DNA synthesis during nuc treatment, although qPCR showed undetectable DNA levels
- No prevention of cccDNA synthesis from incoming virus/replenishment
- Combination nuc + CpAMs (siRNA): expect to improve inhibition of viral genomes within hepatocytes

CpAM = core protein allosteric modulator; HBcrAg = hepatitis B core-related antigen; qPCR = quantitative polymerase chain reaction; siRNA = small interfering RNA. Boyd A, et al. *J Hepatol.* 2016;65:683-691; Petersen J, personal opinion.

WHY DO OUR CURRENT TREATMENT OPTIONS NOT RESULT IN COMPLETE CURE?

cccDNA establishment is not affected

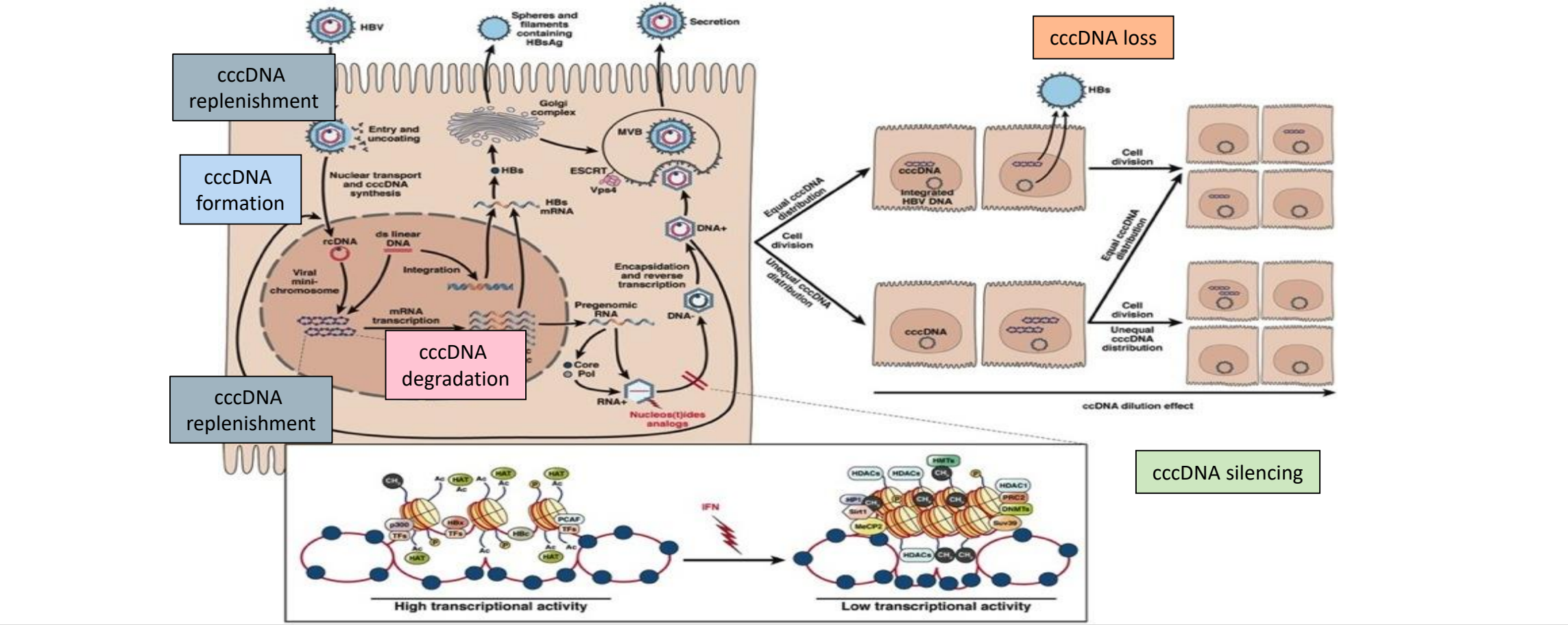


Key role of cccDNA

- Nucs do not block establishment
- Established cccDNA not affected by nucs
- Established cccDNA not efficiently affected by PEG-IFN
- **New therapies must have potential to affect cccDNA**

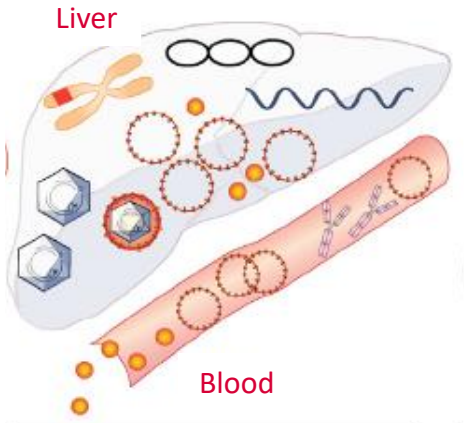
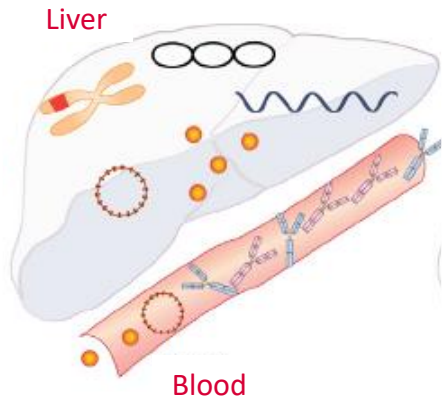
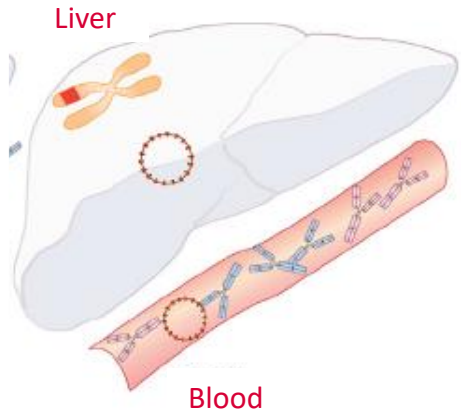
1. Dandri M, Petersen J. Hepatitis B virus DNA clearance: killing for curing? *Hepatology.* 2005;42:1453-1458. 2. Allweiss, et al. *Gut.* 2017.

TARGETING cccDNA: THE VIRAL MINICHROMOSOME



1. Zoulim, et al. *Clin Gastroenterol Hepatol.* 2013. 2. Lucifora, et al. *Science.* 2014. 3. Belloni, et al. *JCI.* 2012. 4. Koeniger, et al. *PNAS.* 2014. 5. Durantel, Zoulim. *J Hepatol.* 2016.

SEVERAL DEFINITIONS OF *CURE* ARE USED

Achieved with nucs	Achieved with IFN-based and nuc therapy in a very small proportion of patients	Currently unachievable
		
Nuc-induced virus suppression (HBeAg seroconversion = 20%)	**Decreased viral RNA and protein synthesis,** HBsAg loss +/- seroconversion	Elimination of virus and cccDNA (but persistence of integrated viral DNA)

Virus **suppression** → **Functional cure (no eradication)** → Complete cure

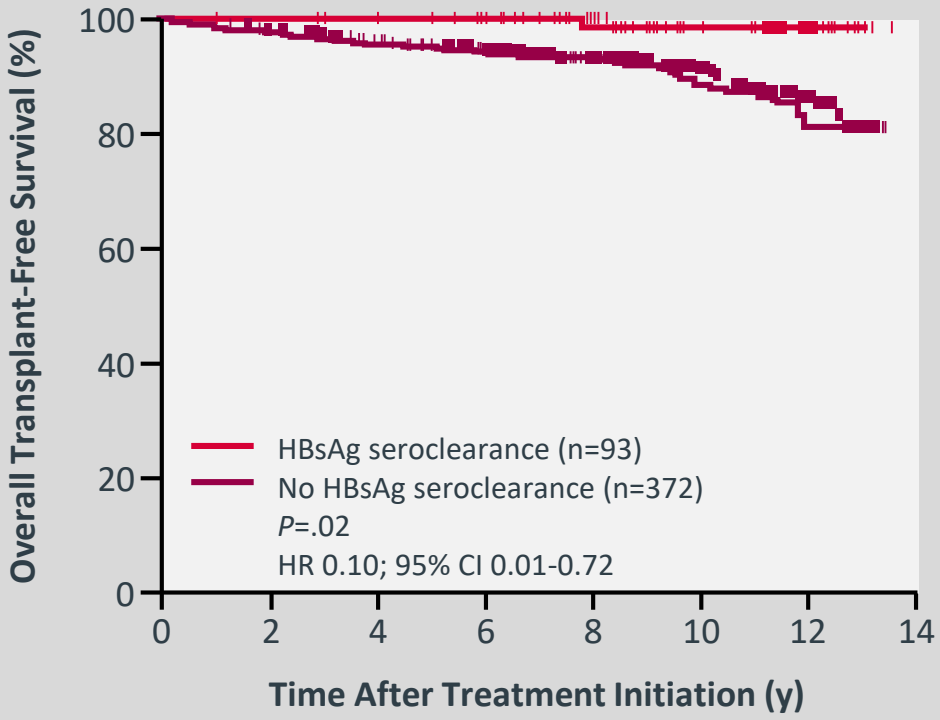
HBeAg = hepatitis B e antigen; IFN = interferon.

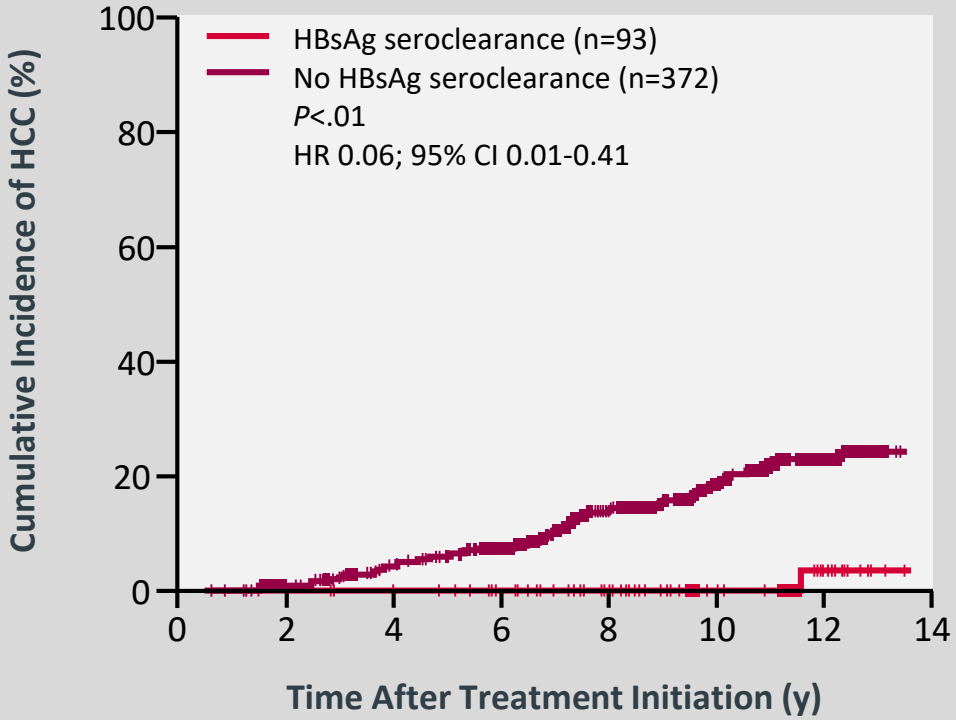
Modified from Durantel D, et al. *J Hepatol.* 2016;64:S117-S131.

WHY IS FUNCTIONAL HBV CURE IMPORTANT?

If you cannot eradicate HBV, inactivate the disease as much as possible

HBsAg loss improves survival and lowers HCC incidence in patients who are currently on oral antiviral therapy





CI = confidence interval; HR = hazard ratio.
Kim GA, et al. *Gut.* 2014;63:1325-1332.

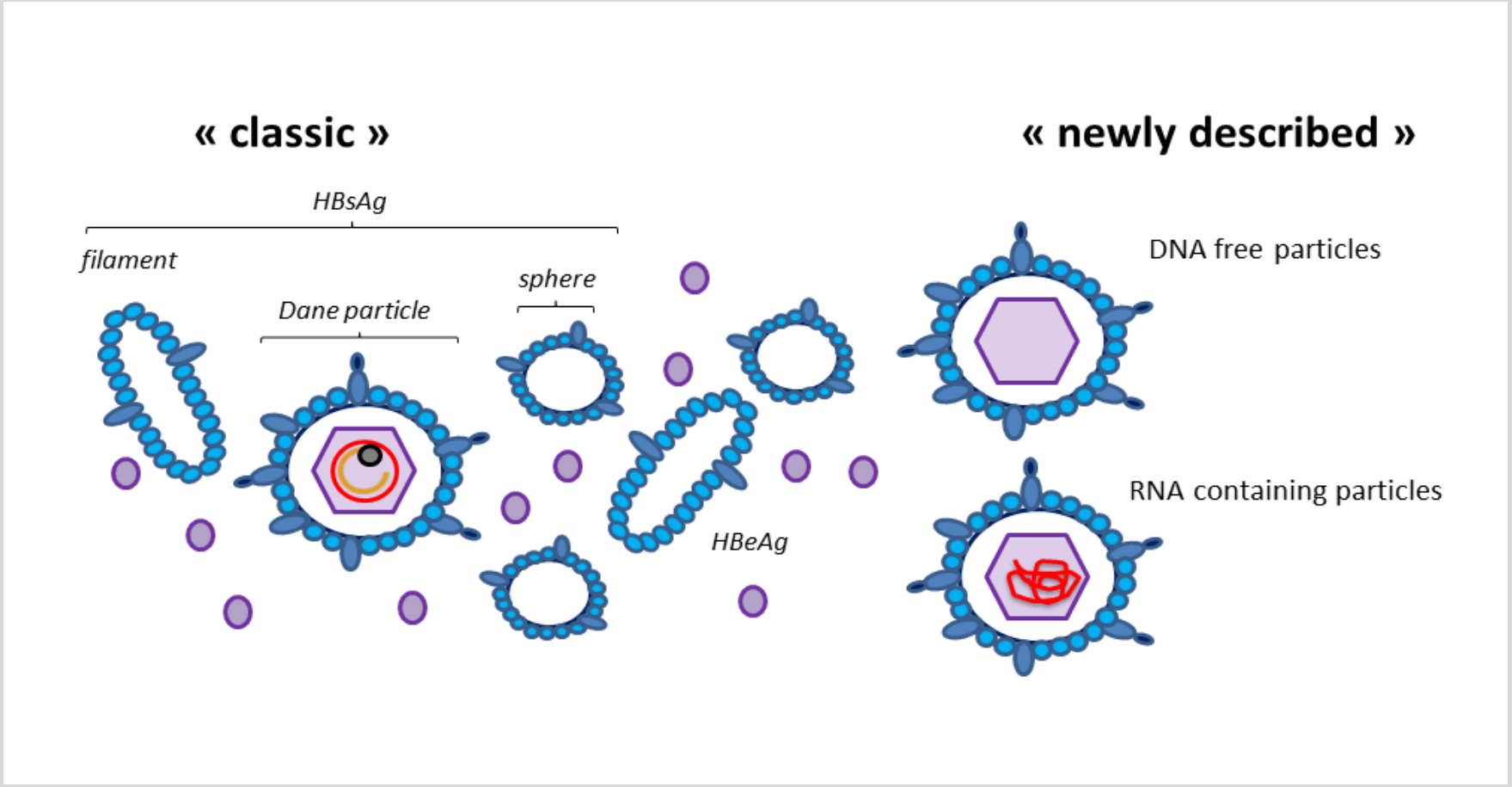
SIMPLIFICATION OF HBV TREATMENT ENDPOINTS

- Current consensus: The ultimate goal would be achievement of HBsAg loss with a finite course of oral antiviral (combination) therapy
- Phase 2 endpoints vs Phase 3 endpoints
- HBsAg decline as a phase 2 endpoint?
- HBsAg loss (or low stable plateau?), with or without anti-HBs seroconversion, may be an optimal endpoint
- Hypothesis: 24 weeks of combination treatment inducing >5% HBsAg loss would be considered significant
- cccDNA half-life discussion nice to have, but let's see results of combination studies first!

(New) data will drive (re)evaluation of (old) dogma

HBs = hepatitis B surface.
Petersen J, personal opinion.

(NEW) BIOMARKERS NEEDED: CIRCULATING VIRAL ANTIGENS AND PARTICLES



(Novel) HBV serum markers

- qHBsAg (established)
- HBcrAg
- Circulating viral RNAs

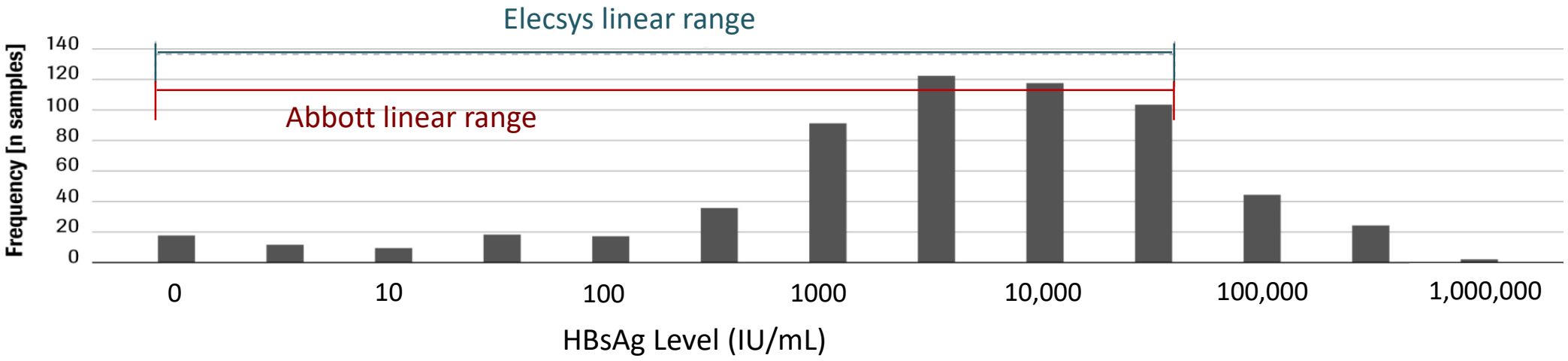
qHBsAg = quantitative hepatitis B surface antigen.

Adapted from Lucifora J, Protzer U. Hepatitis B virus X protein: a key regulator of the virus life cycle. Available at: http://cdn.intechweb.org/pdfs/29526.pdf (accessed November 2017).

QUANTITATIVE HBsAg TESTS: TOTAL HBsAg

Abbott linear range 0.05-250 IU/mL

Elecsys® linear range 0.05-52,000 IU/mL



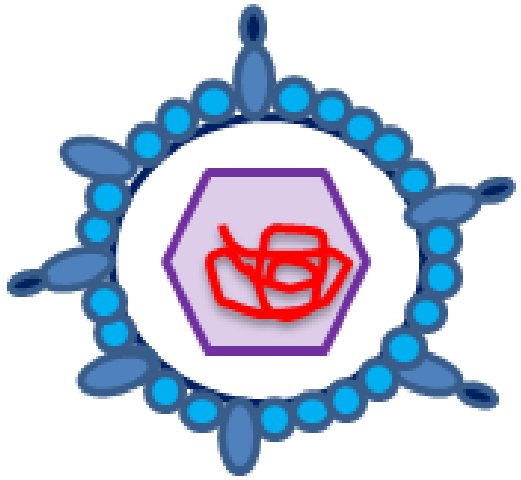
Linear ranges covering clinically relevant HBsAg levels

1. Zacher BJ, et al. *Clin Vaccine Immunol.* 2011;18:1943-1950. 2. Package insert. Elecsys HBsAg II quant.

HBV RNA (pgRNA) IN CIRCULATING VIRAL PARTICLES

- First described in 1996 in the serum of infected patients
- Can be released into serum as enveloped 3.5 kb pgRNA-containing virions
- Amount of circulating pgRNA strongly correlates with the amount of pgRNA present in the whole liver and with transcriptionally active cccDNA in humanized mice
- Marker to study the transcriptional activity of cccDNA

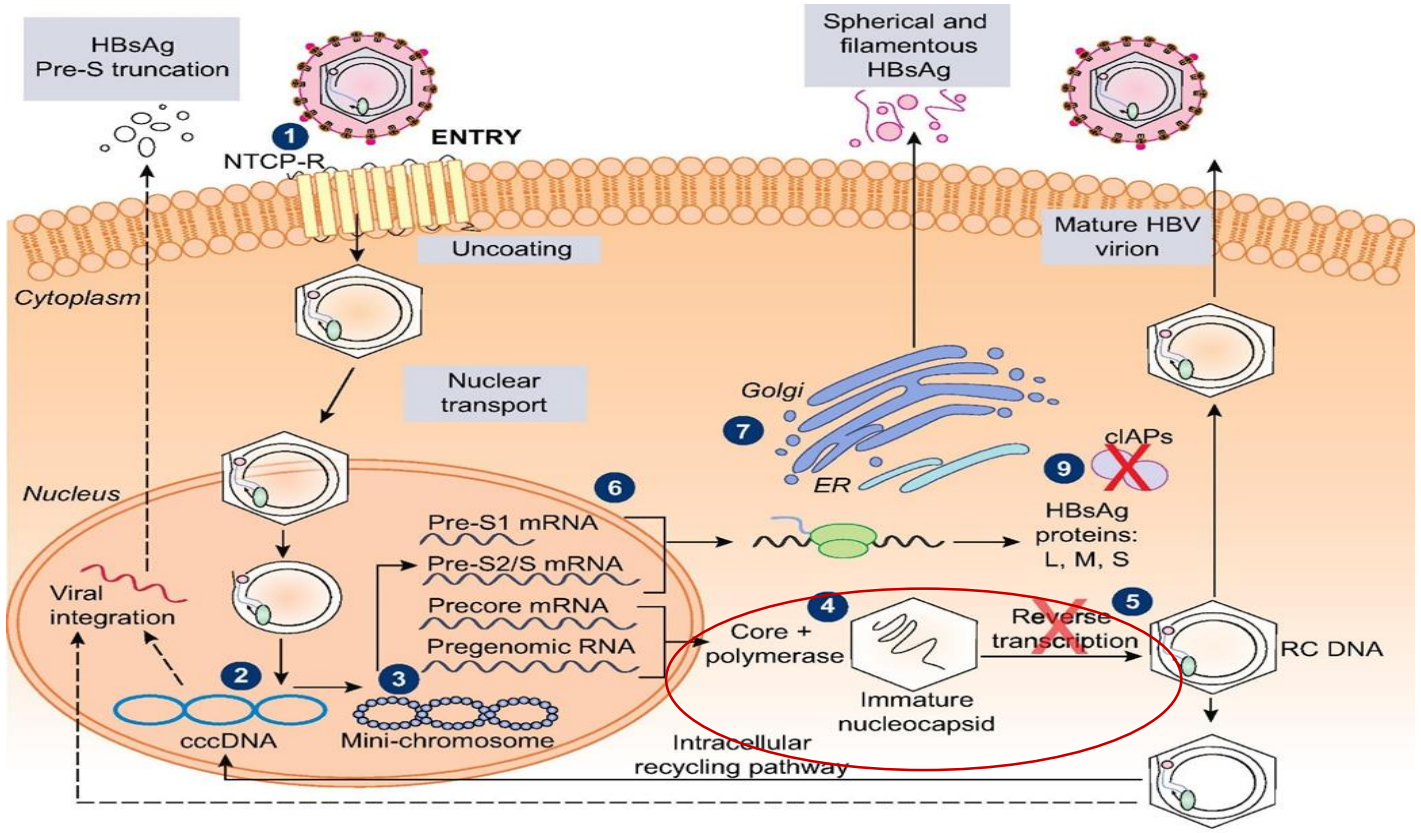
No commercially available test yet, but “performance of an automated prototype assay for the detection and quantification of hepatitis B pregenomic RNA in chronic HBV patients receiving nucleo(t)side analogue therapy”



RNA-containing particles

1. Wang J, et al. *J Hepatol.* 2016;65:700-710. 2. Giersch K, et al. *J Hepatol*. 2017;66:460-462. 3. Butler K, et al. *AASLD.* 2017;Poster#1915.

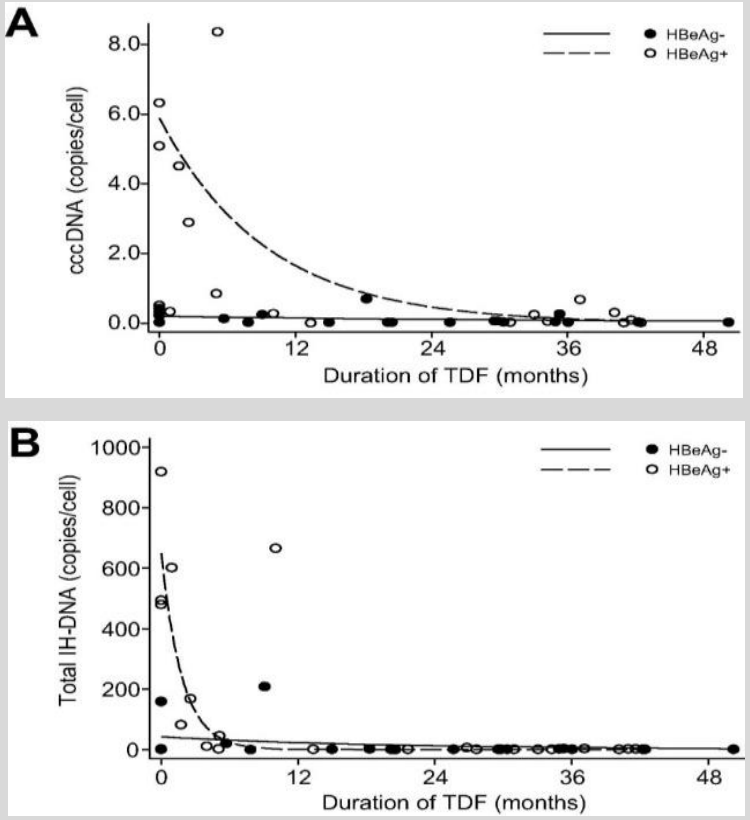
HOW TO USE THESE MARKERS?



Petersen J, et al. *J Hepatol.* 2016;65:835-848.

WHY DO OUR CURRENT TREATMENT OPTIONS NOT RESULT IN COMPLETE CURE?

HBV DNA is not fully suppressed

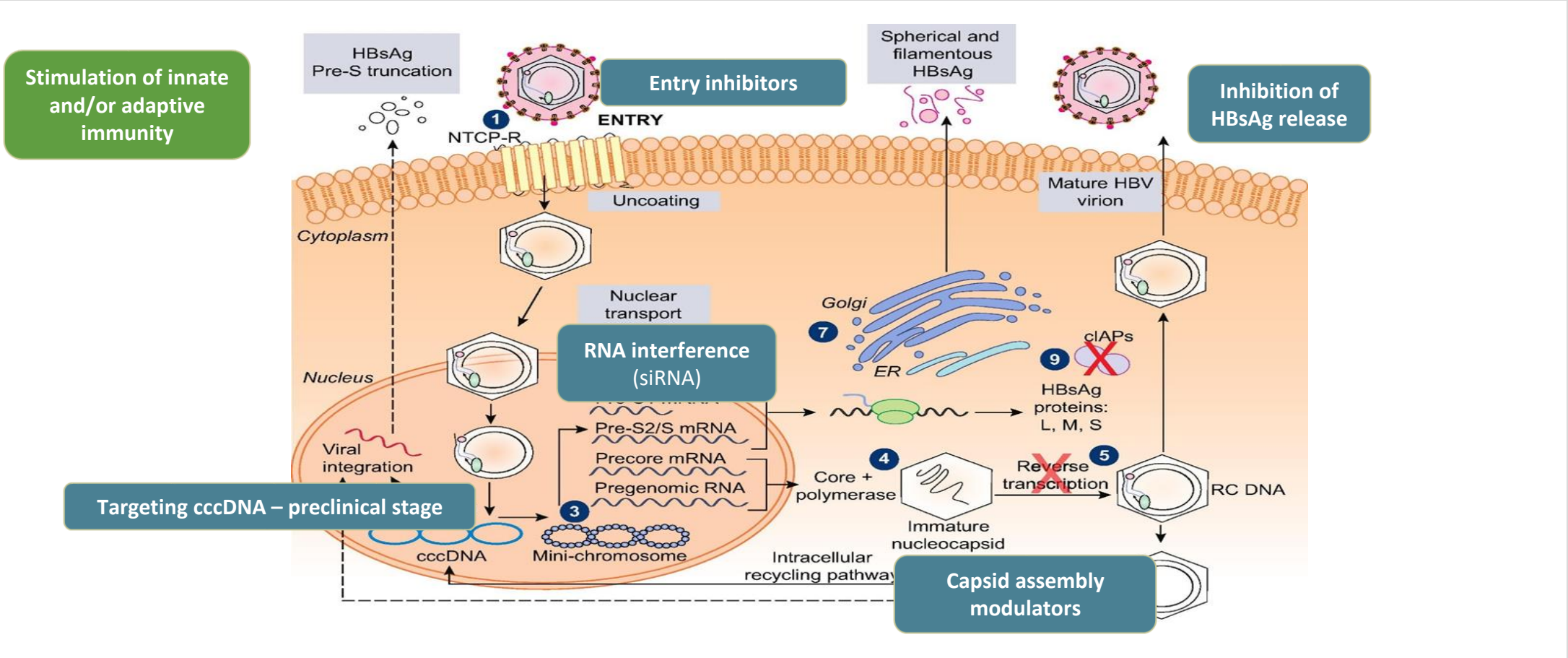


- Several recent studies demonstrated persistence of HBV DNA or intrahepatic HBV DNA synthesis during nuc treatment, although qPCR showed undetectable DNA levels
- No prevention of cccDNA synthesis from incoming virus/replenishment
- Combination nuc + CpAMs (siRNA): expect to improve inhibition of viral genomes within hepatocytes

How to measure efficacy if biopsies unavailable? pgRNA? HBcrAg?

1. Boyd A, et al. *J Hepatol.* 2016;65:683-691. 2. Petersen J, personal opinion.

NEW HBV TARGETS: WHICH PATHWAYS OR APPROACHES MIGHT WE TAKE?



Petersen J, et al. *J Hepatol.* 2016;65:835-848.

CORE PROTEIN – ATTRACTIVE TARGET – ORAL THERAPY

Highly conserved

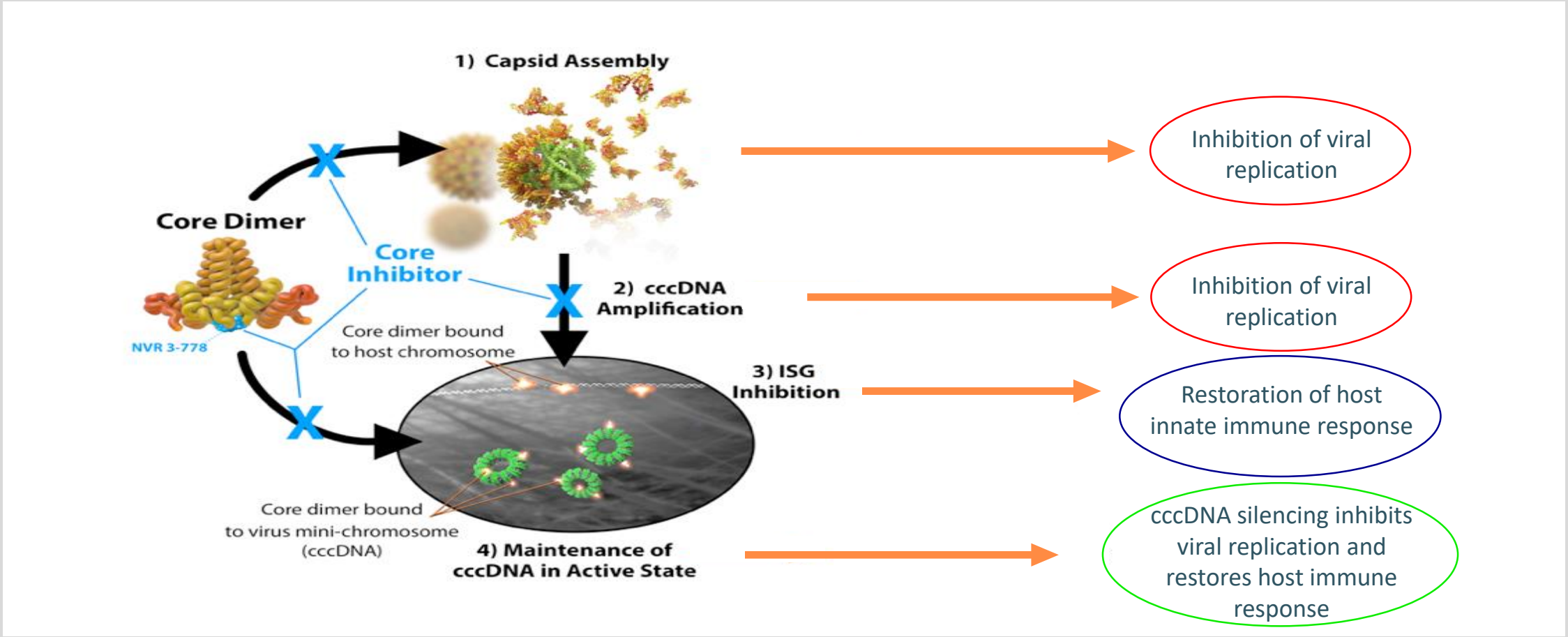
A lot of functions

- Transport to the nucleus
- Uncoating of HBV DNA
- Packaging
- Capsid assembly
- Modulate reverse transcription
- Interacts with sAg
- May also modulate cccDNA and export viral RNA

All allosteric regulation…1 molecule that affects any function will affect them all!

sAg = surface antigen.

TARGETING THE HBV CORE PROTEIN WITH CORE INHIBITORS



1. Billioud, et al. *Antiviral Res*. 2011. 2. Klumpp K, et al. *PNAS*. 2015. 3. Venkatakrishnan B, et al. *J Virol*. 2016.

SUMMARY

- Currently, only a small proportion of patients will achieve functional cure (HBsAg loss); complete virological cure is still not possible
- Knowledge of the HBV replication cycle has resulted in identification of new agents that may have the potential to (functionally) cure more HBV patients in the future
- Today's view is that combining new treatment targets appears to be necessary to achieve higher rates of functional cure in the future
- Reasonable aim: >5% HBsAg loss within 24 weeks of all oral combination therapy
- Demonstration of the long-term safety profile of all new treatment targets is of utmost importance (part of the dilemma: safe nucs but life-long therapy for most of patients)
- With new therapies comes need to proof success of treatment – HBV RNA as biomarker

THANK YOU FOR YOUR ATTENTION

Jörg Petersen, MD, PhD
Professor of Medicine and Head of the Liver Unit
IFI Institute for Interdisciplinary Medicine
Asklepios Klinik St. George, University of Hamburg

AGENDA

11:30AM – 11:50AM	**Opening Remarks** Derek Small Chief Executive Officer
11:50AM – 12:15PM	**Douglas Dieterich, MD** Professor of Medicine Division of Liver Diseases Director Institute for Liver Medicine Mount Sinai School of Medicine
12:15PM – 12:40PM	**Jörg Petersen, MD, PhD** Professor of Medicine and Head of the Liver Unit IFI Institute for Interdisciplinary Medicine Asklepios Klinik St. George, University of Hamburg
12:40PM – 1:30PM	**R&D Overview and Clinical Data Hepatitis B Program** Richard Colonno, PhD EVP & Chief Scientific Officer of Virology Operations
1:30PM – 1:45PM	**Clinical Development Hepatitis B Program** Uri Lopatin, MD Chief Medical Officer
1:45PM – 2:00PM	**Commercial Perspectives on Hepatitis B** JP Benya Vice President, Commercial
2:00PM – 2:25PM	**Management and KOL Q&A Session** Speakers: Dr. Dieterich, Dr. Petersen, Derek Small, Dr. Richard Colonno, Dr. Uri Lopatin, JP Benya

RICHARD COLONNO, PhD

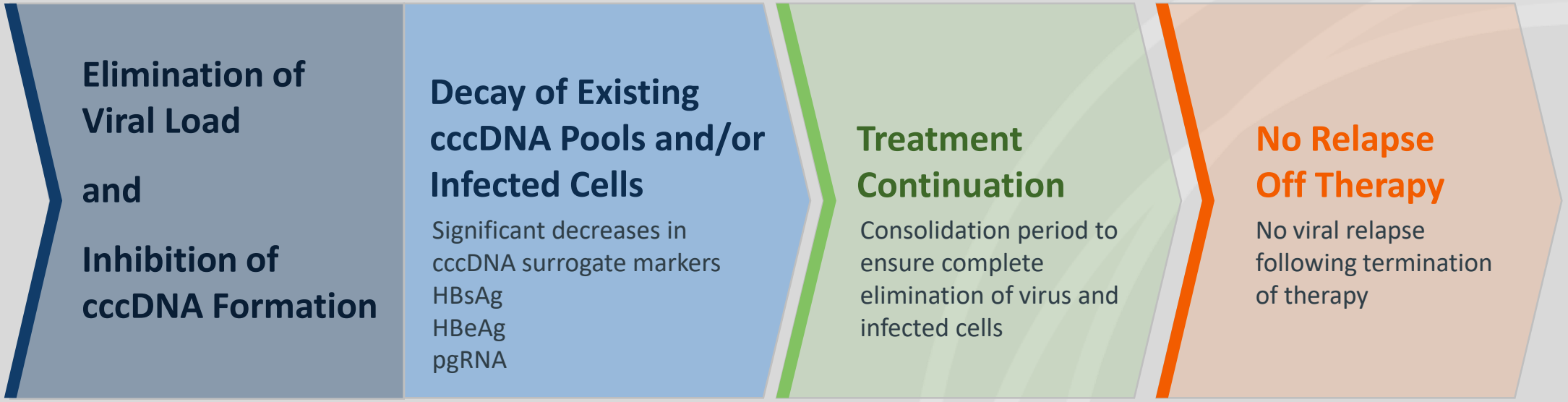
Executive Vice President and
Chief Scientific Officer of Virology Operations

Prior: Merck, Bristol-Myers Squibb (Baraclude®, Reyataz®) and Presidio

assembly biosciences

HBV CURE: CLINICAL COMPONENTS

Expected Treatment Components to Achieve Cure



THOUGHT WE HAD A POTENTIAL CURE OVER 10 YEARS AGO

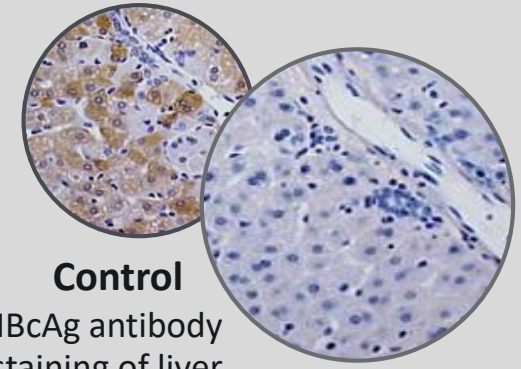
ETV MONOTHERAPY CURED CHRONICALLY INFECTED WOODCHUCKS

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

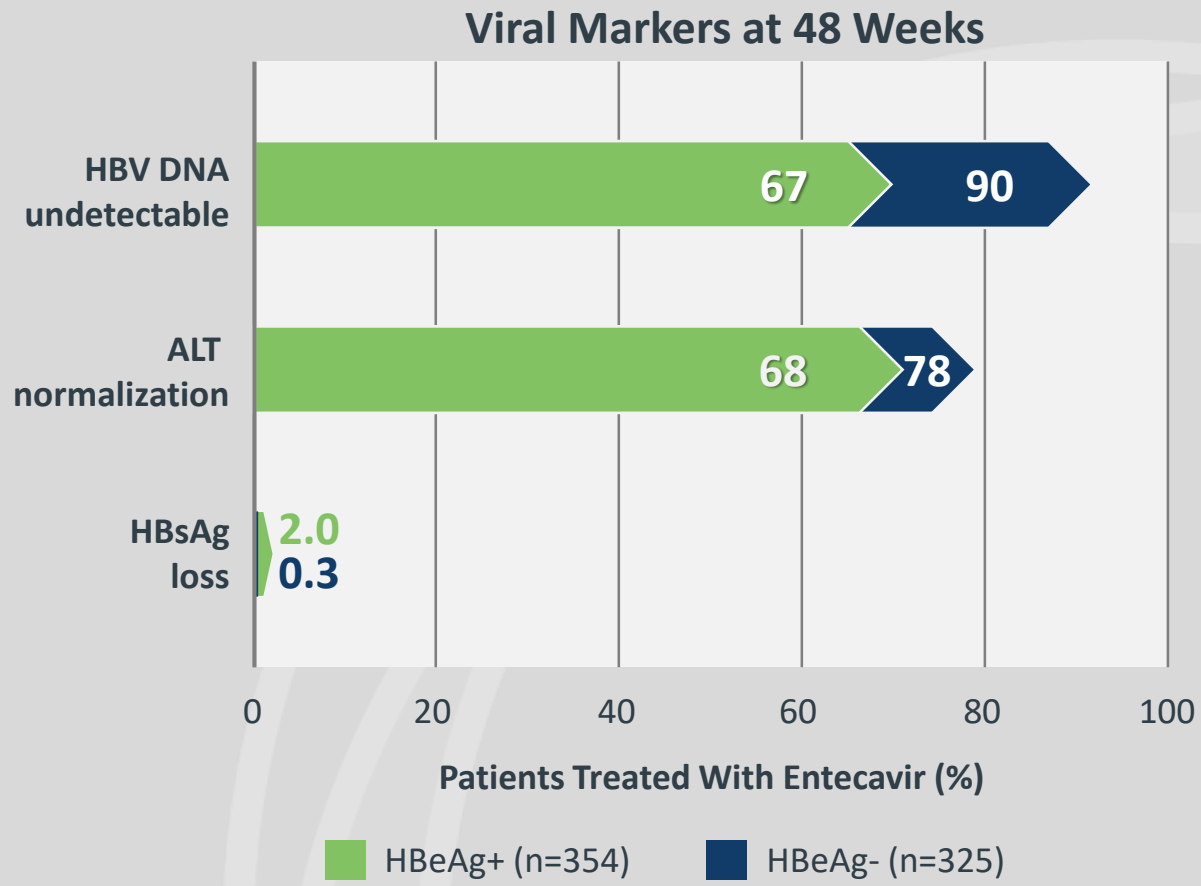
Unfortunately, this is not what happens in HBV patients, despite prolonged therapy

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

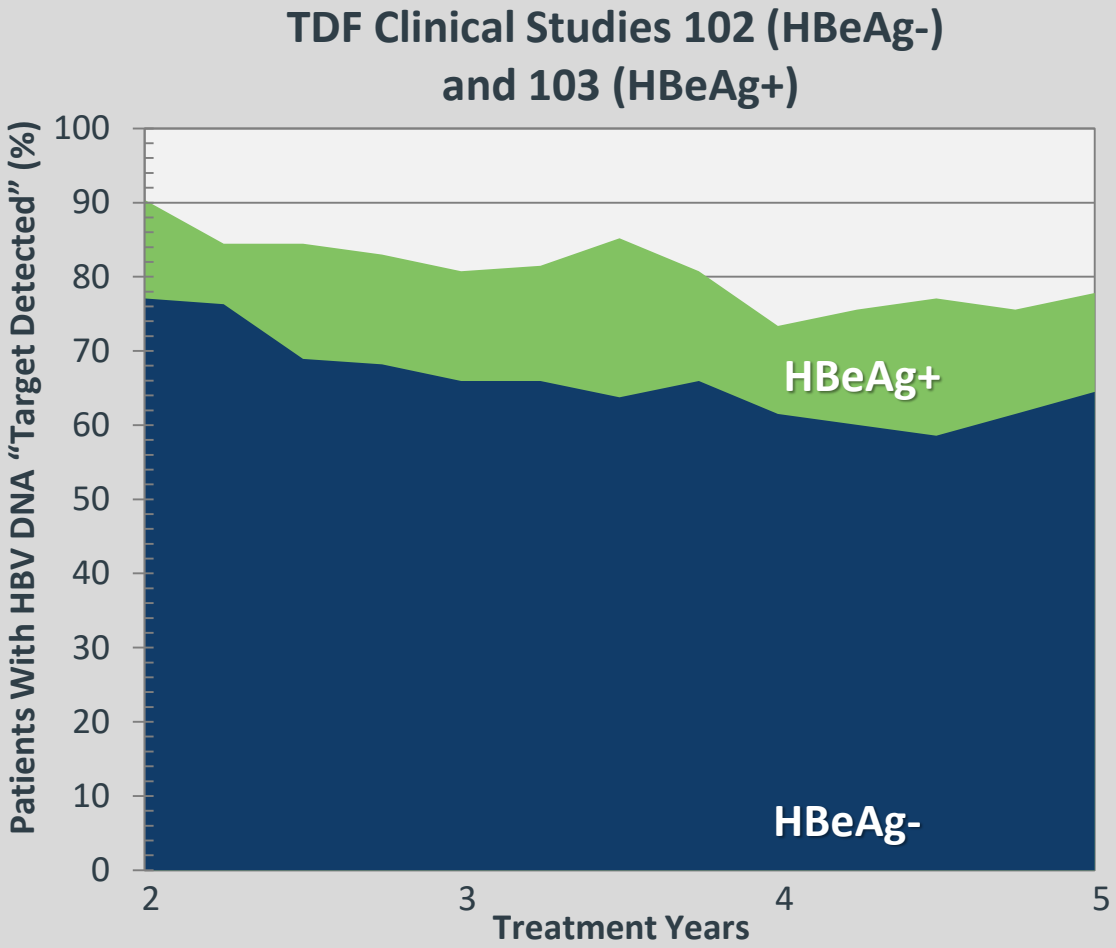
- Preferred SOC nucs are ETV, TFV, and TAF
- Exhibit potent antiviral efficacy
- Rapidly reduce viral DNA to lower limit of detection in virtually all patients
- **Viral suppression maintained indefinitely with continued therapy**



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**

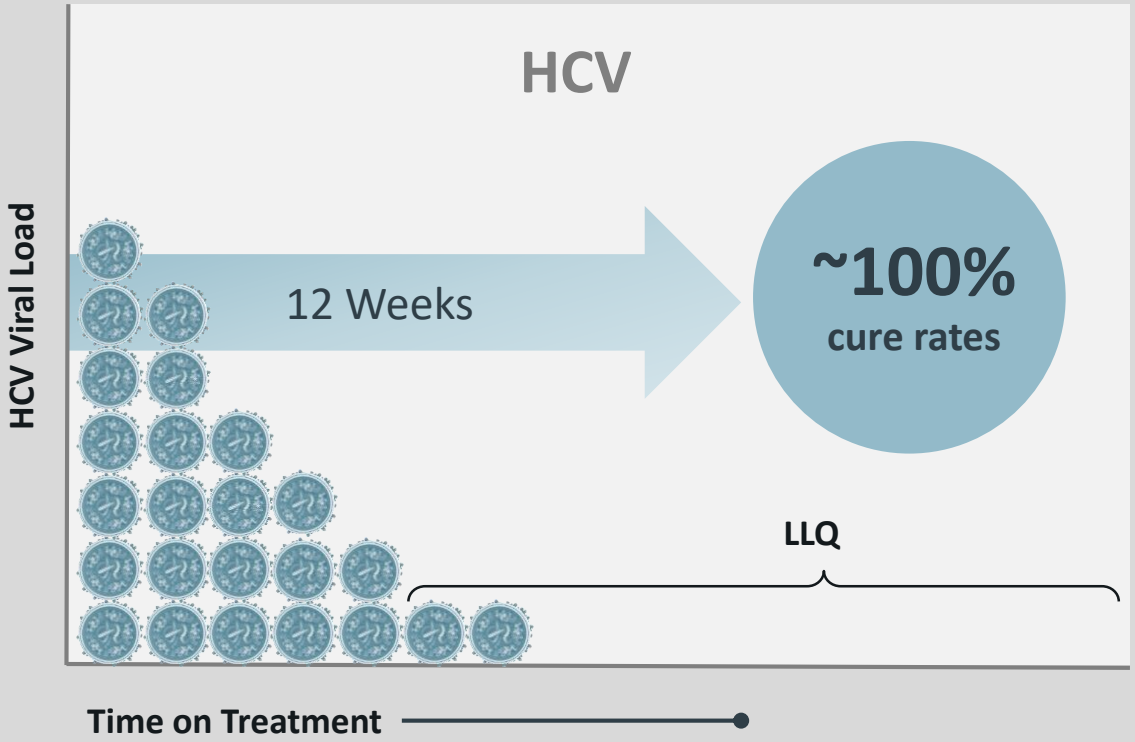
HBeAg Pos. Patient	Treatment	Treatment Years	HBsAg IU/mL	HBV DNA (copy/mL)
003	ADV/LVD	5	**6.6**	**1,530**
016	LVD/ADV/IFN	13	**3.8**	**1,040**
019	LVD/ADV/ETV/TDF	6.5	**4.7**	**1,840**
024	IFN	1	**0.6**	**188**

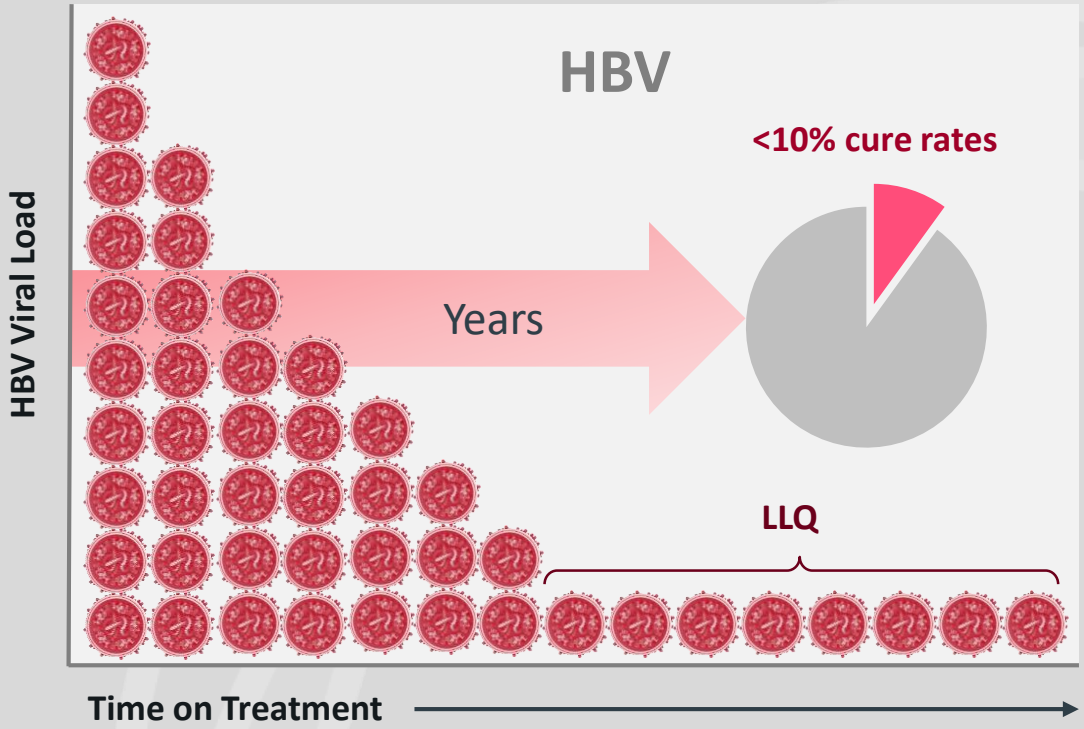
- **Cure is not possible if viral infection persists**

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



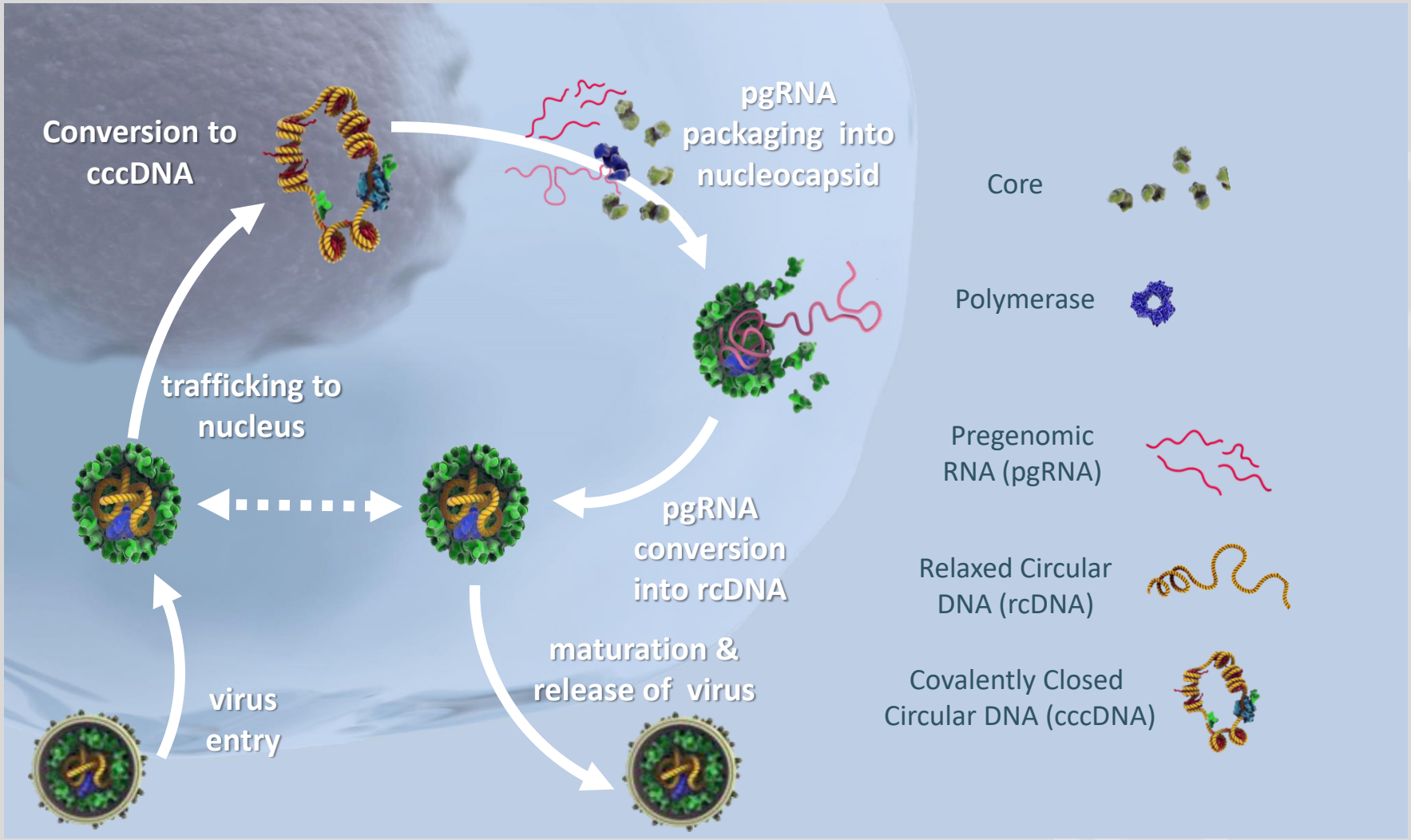


To improve cure rates…must eliminate residual virus

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

HOW CAN THERAPY BE IMPROVED?

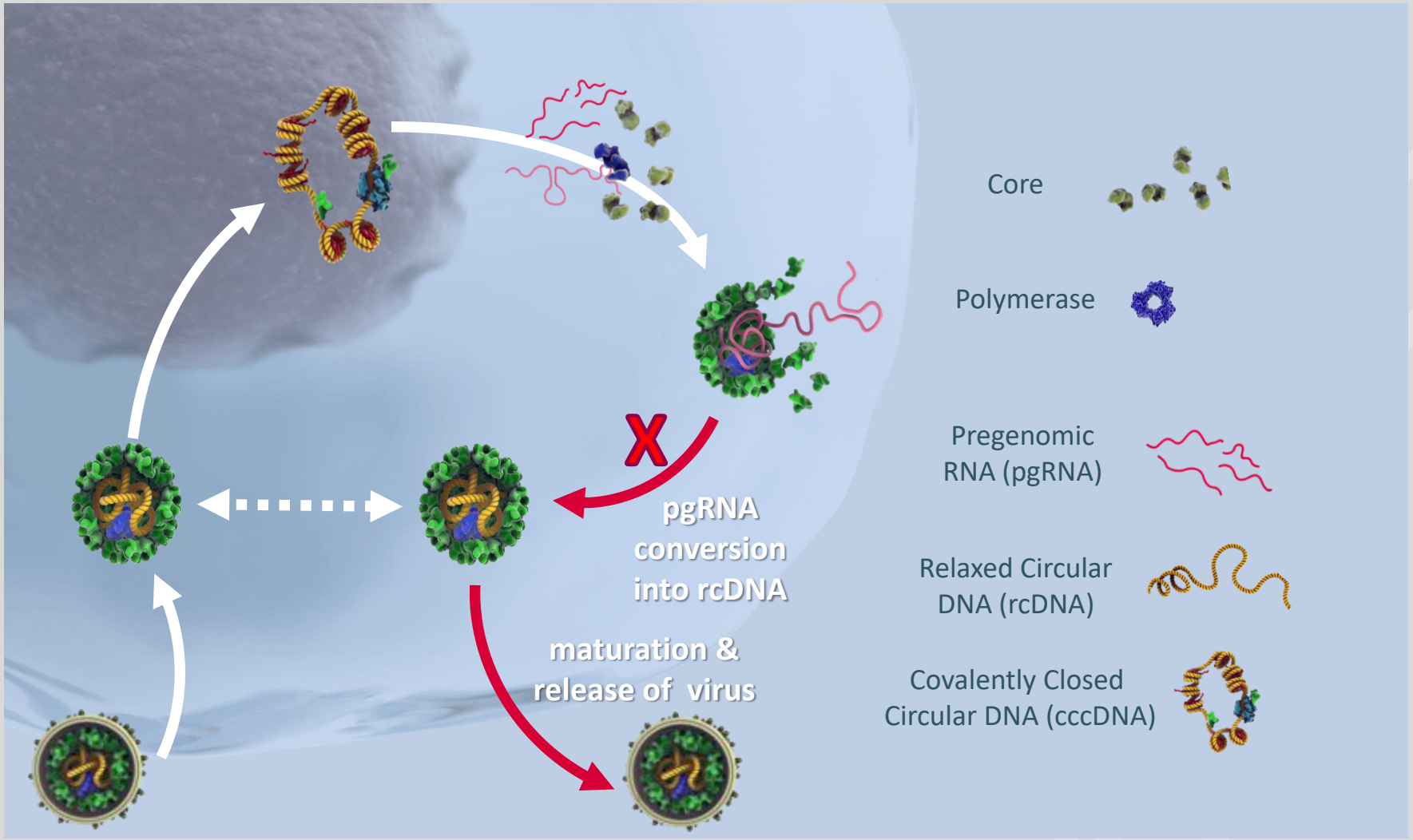
CRITICAL ELEMENTS OF HBV LIFE CYCLE



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

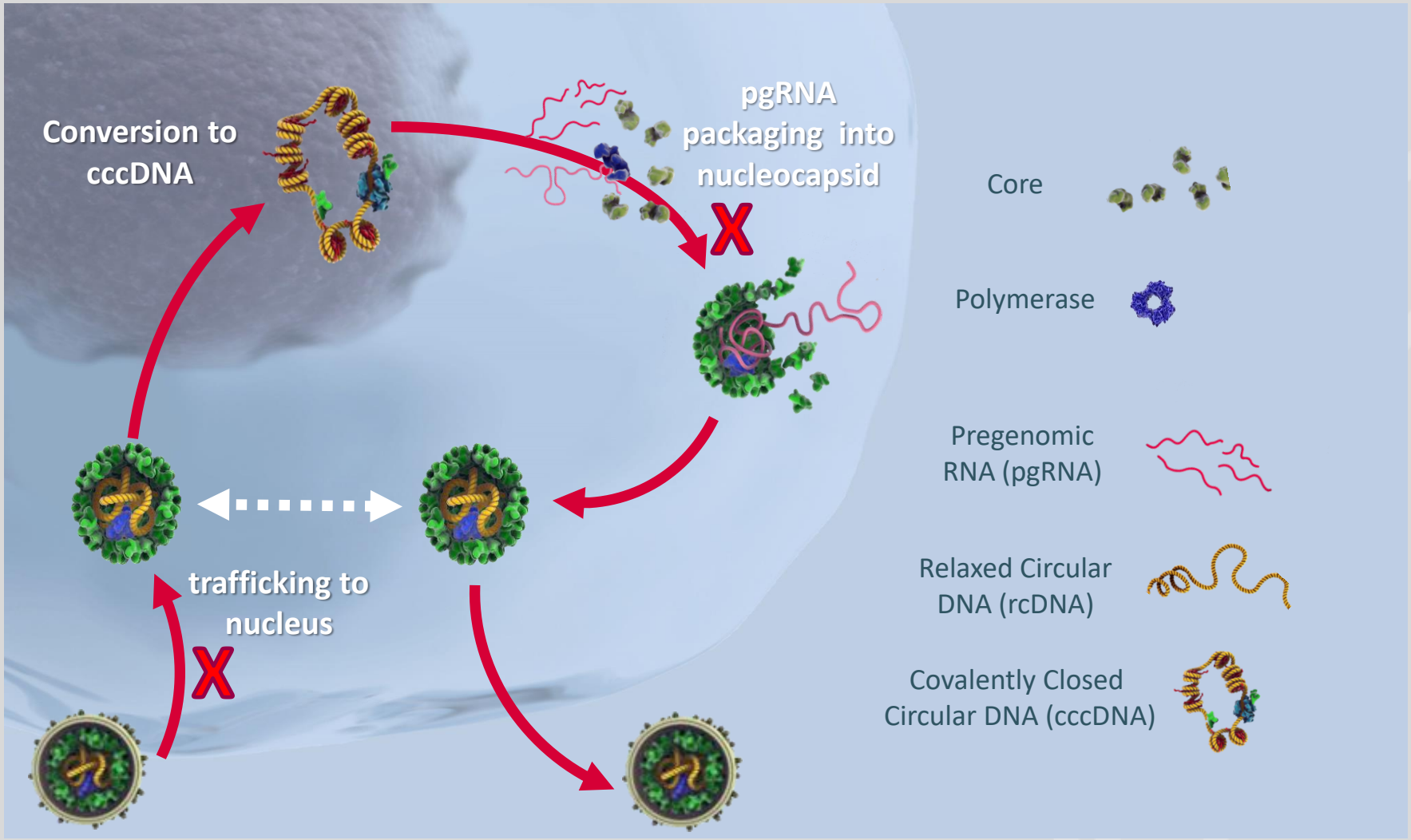
NUCS REDUCE VIRUS LEVELS BUT FAIL TO PREVENT cccDNA ESTABLISHMENT



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

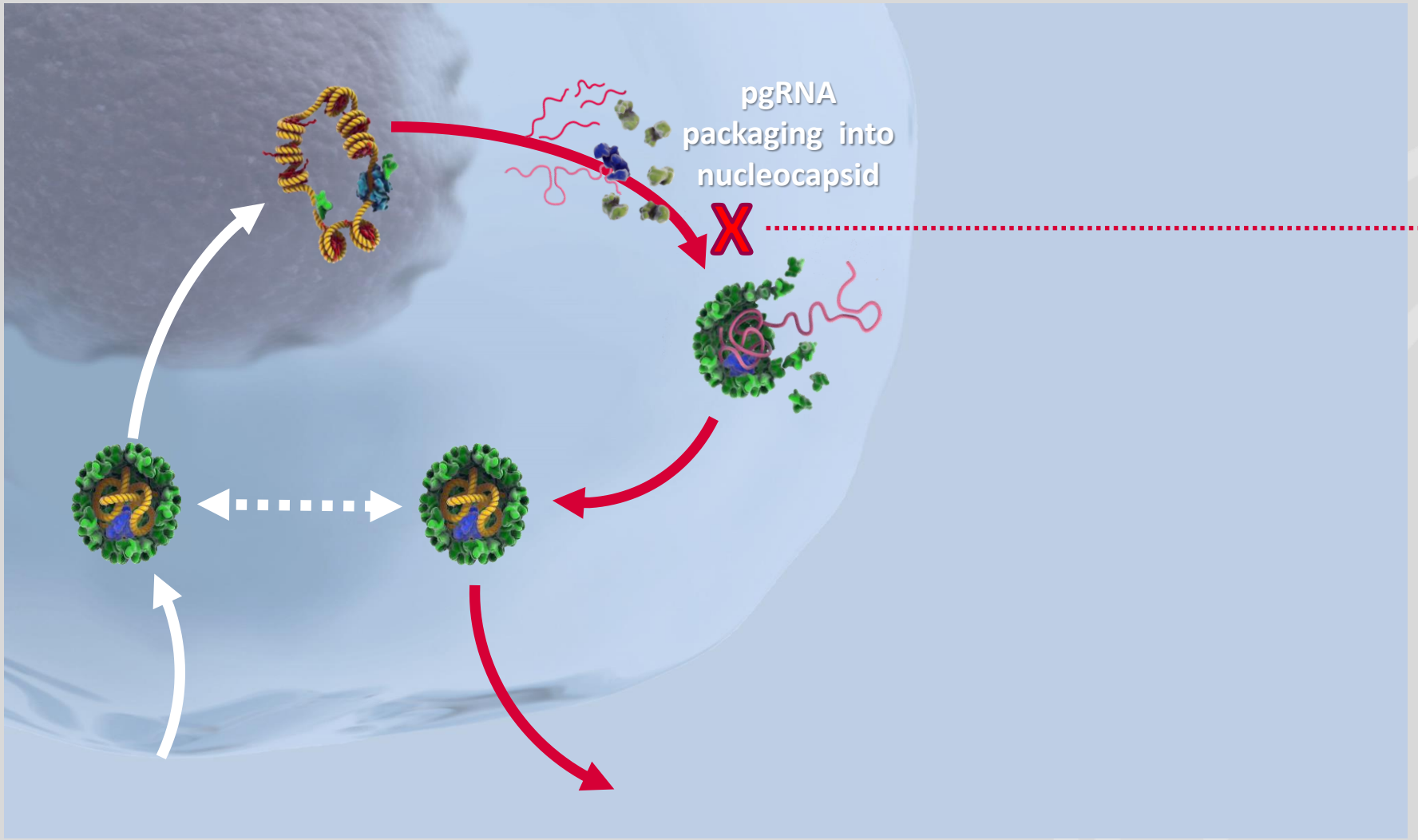
CORE INHIBITORS BLOCK VIRAL REPLICATION AND cccDNA ESTABLISHMENT

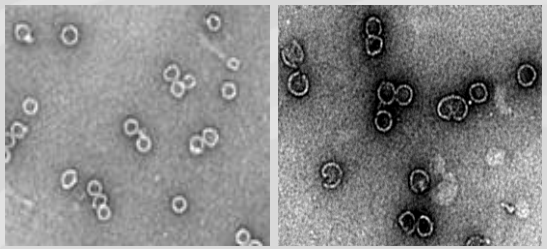


Core inhibition

- 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

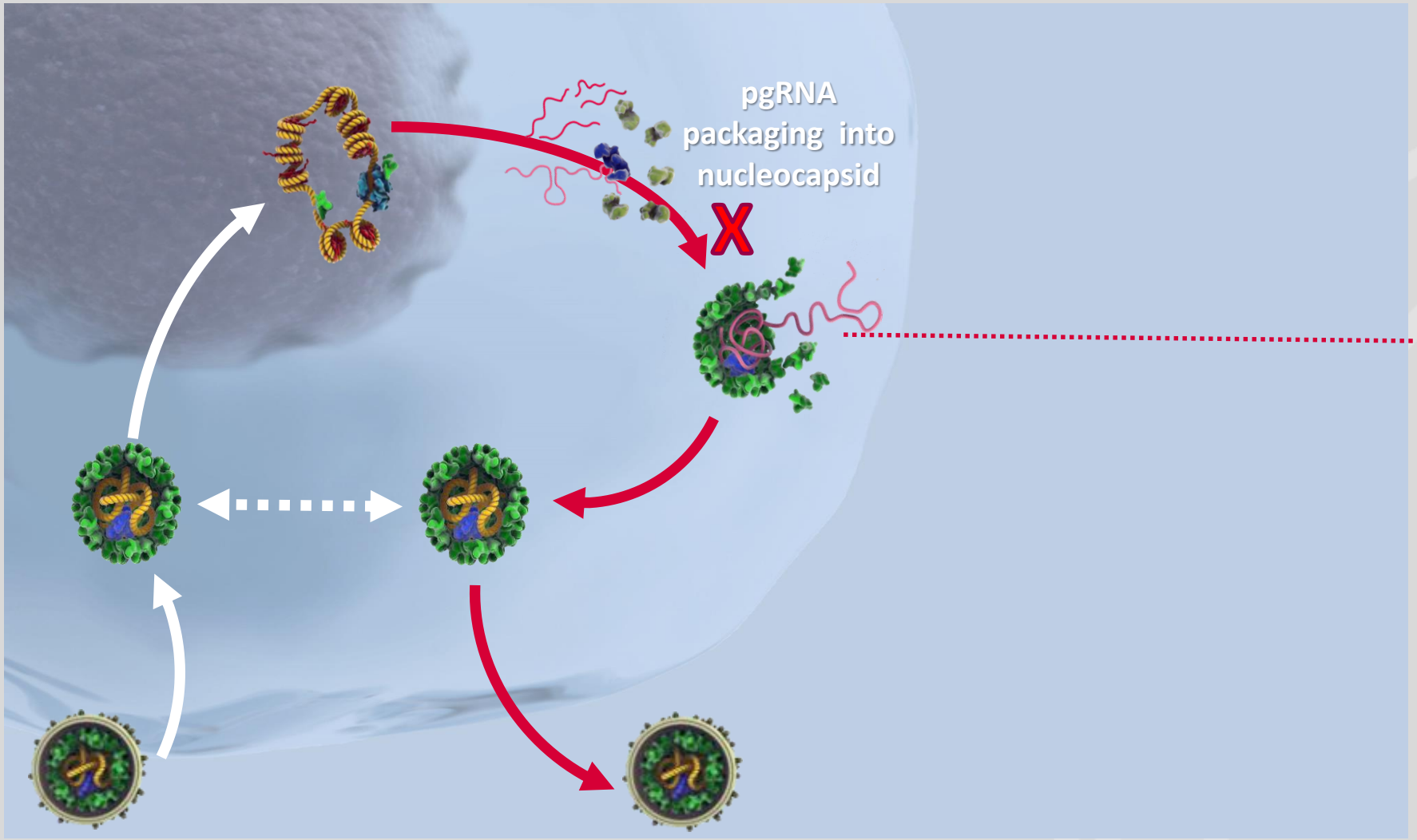
CORE INHIBITORS BLOCK FORMATION OF FUNCTIONAL NUCLEOCAPSIDS

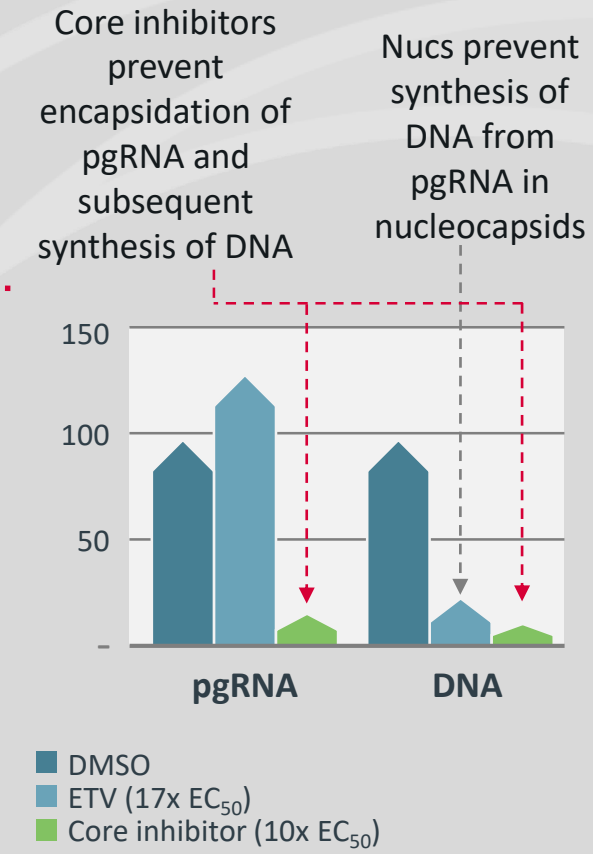




Addition of core inhibitor causes formation of aberrant capsids that are larger, cracked, and asymmetrical

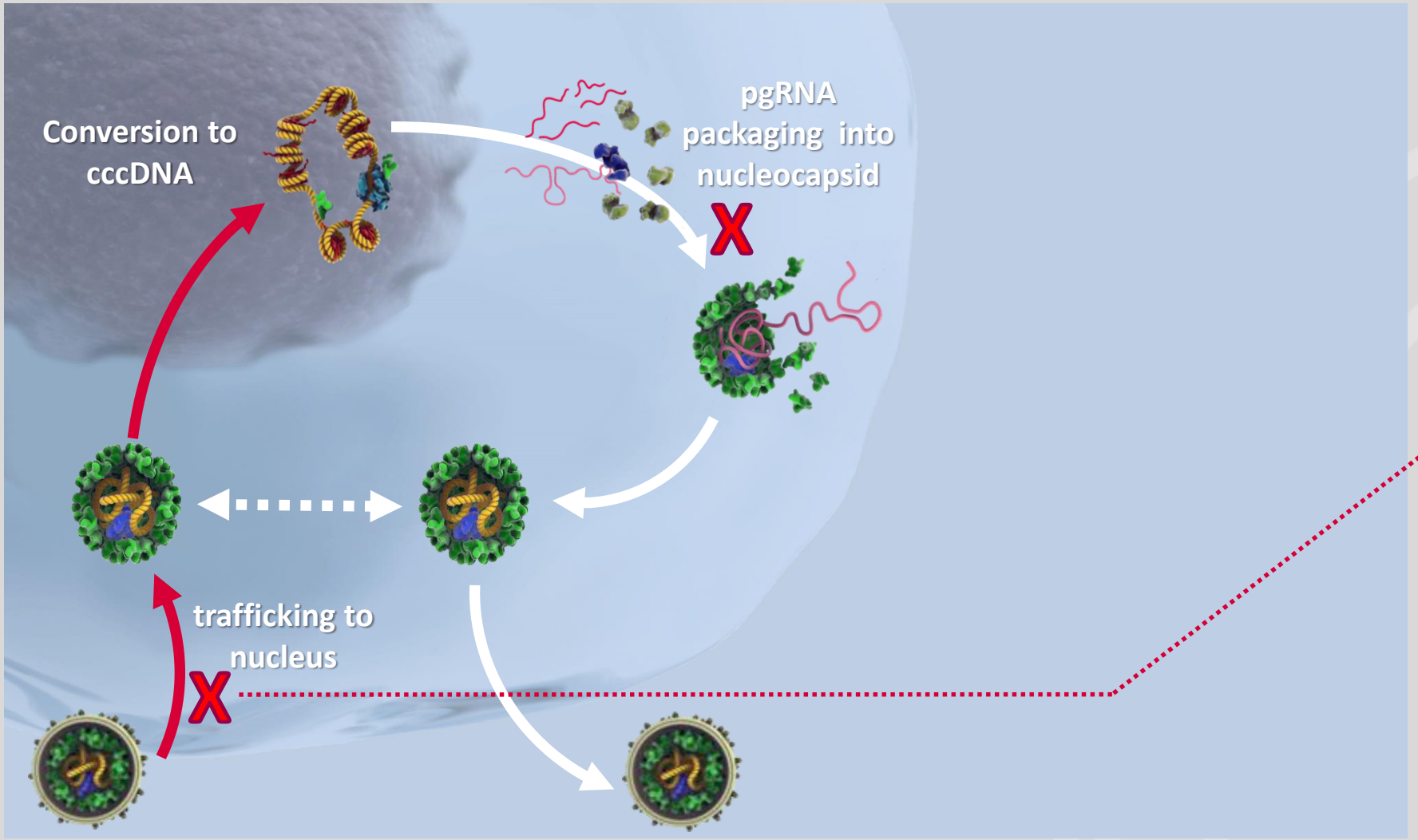
CORE INHIBITORS BLOCK ENCAPSIDATION OF pgRNA



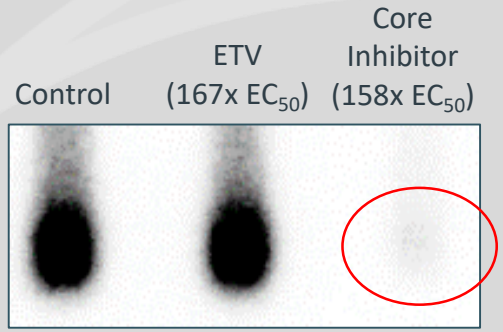


Huang Q, et al. AASLD Poster 922 2017.

CORE INHIBITORS CAUSE PREMATURE MELTING OF NUCLEOCAPSIDS



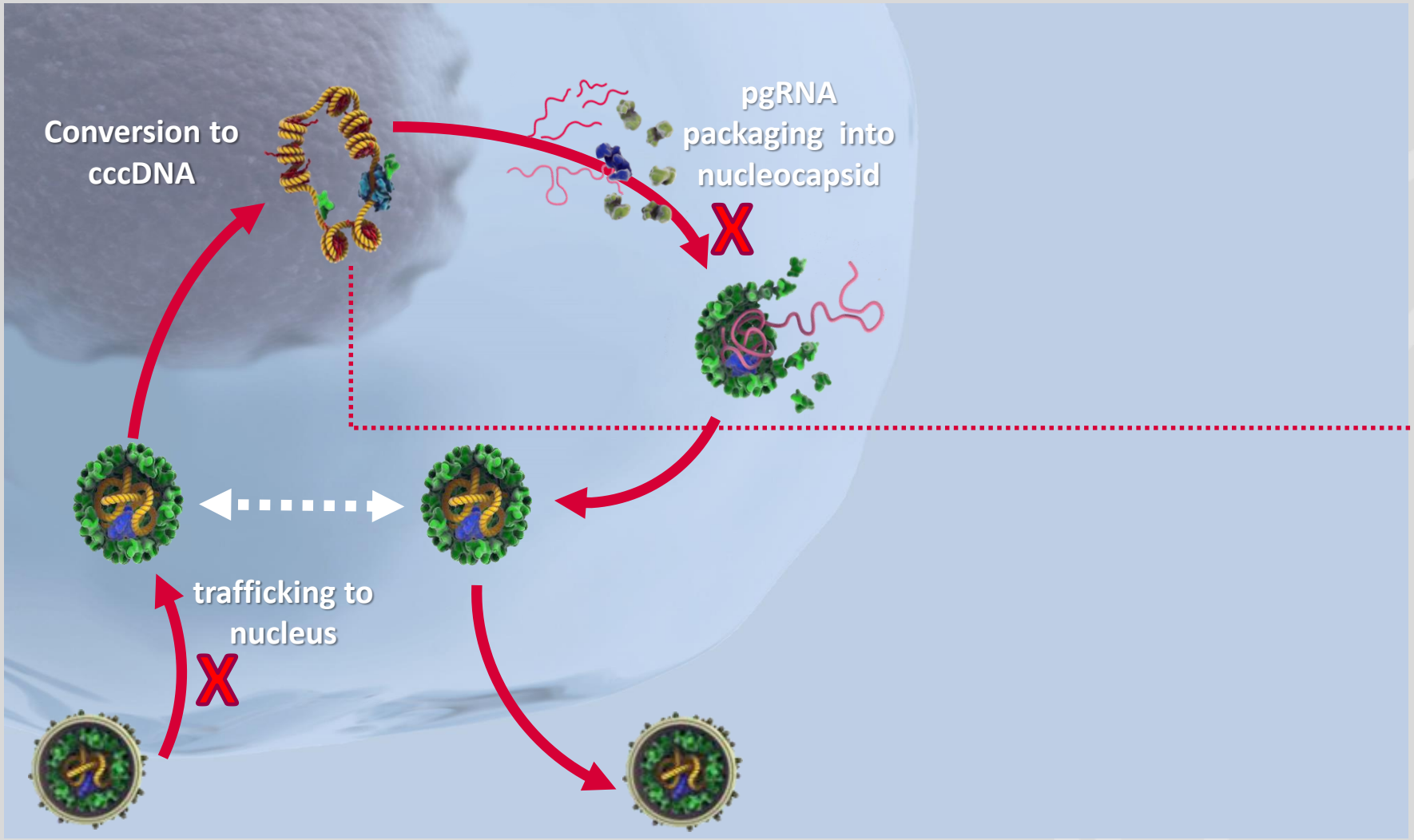
Southern Blot of HBV DNA From HepG2-NTCP Cells 3 Hours After Infection



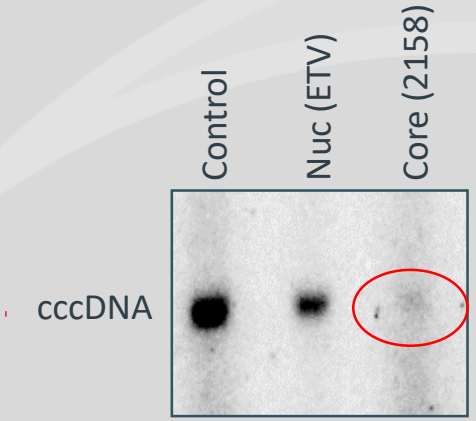
Core inhibitors cause premature melting of existing nucleocapsids and prevent trafficking of rcDNA to nucleus

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)

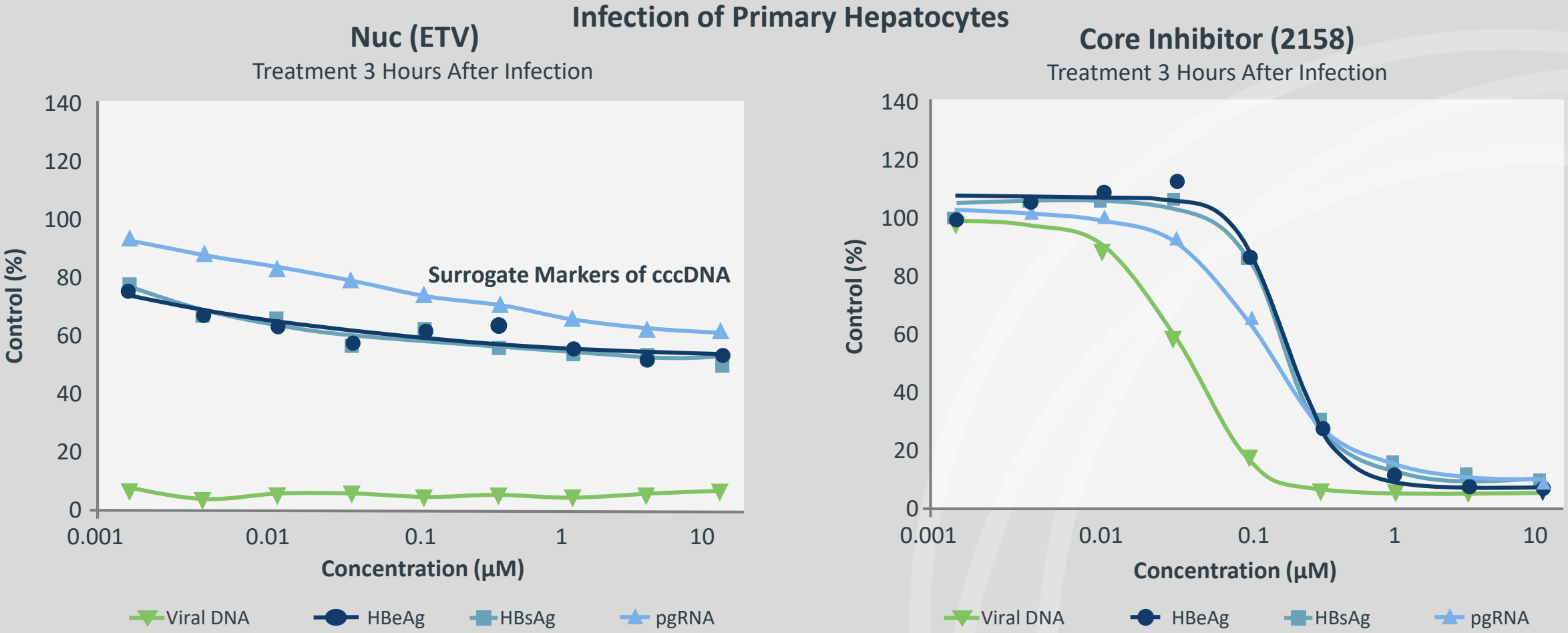


ETV >10,000x EC_{50}
2158 50x EC_{50}

Core inhibitors prevent cccDNA generation and formation

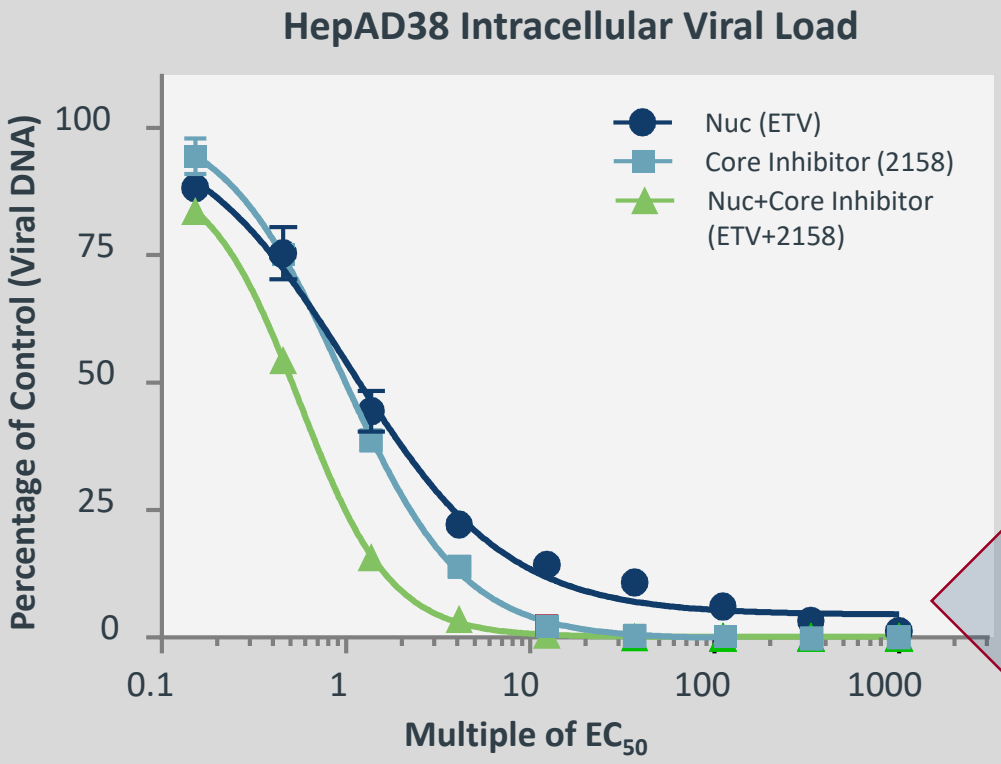
1. Cai, D, et al. Intl HBV Mtg Poster P-140. 2017; 2. Huang Q, et al. AASLD Poster 922 2017.

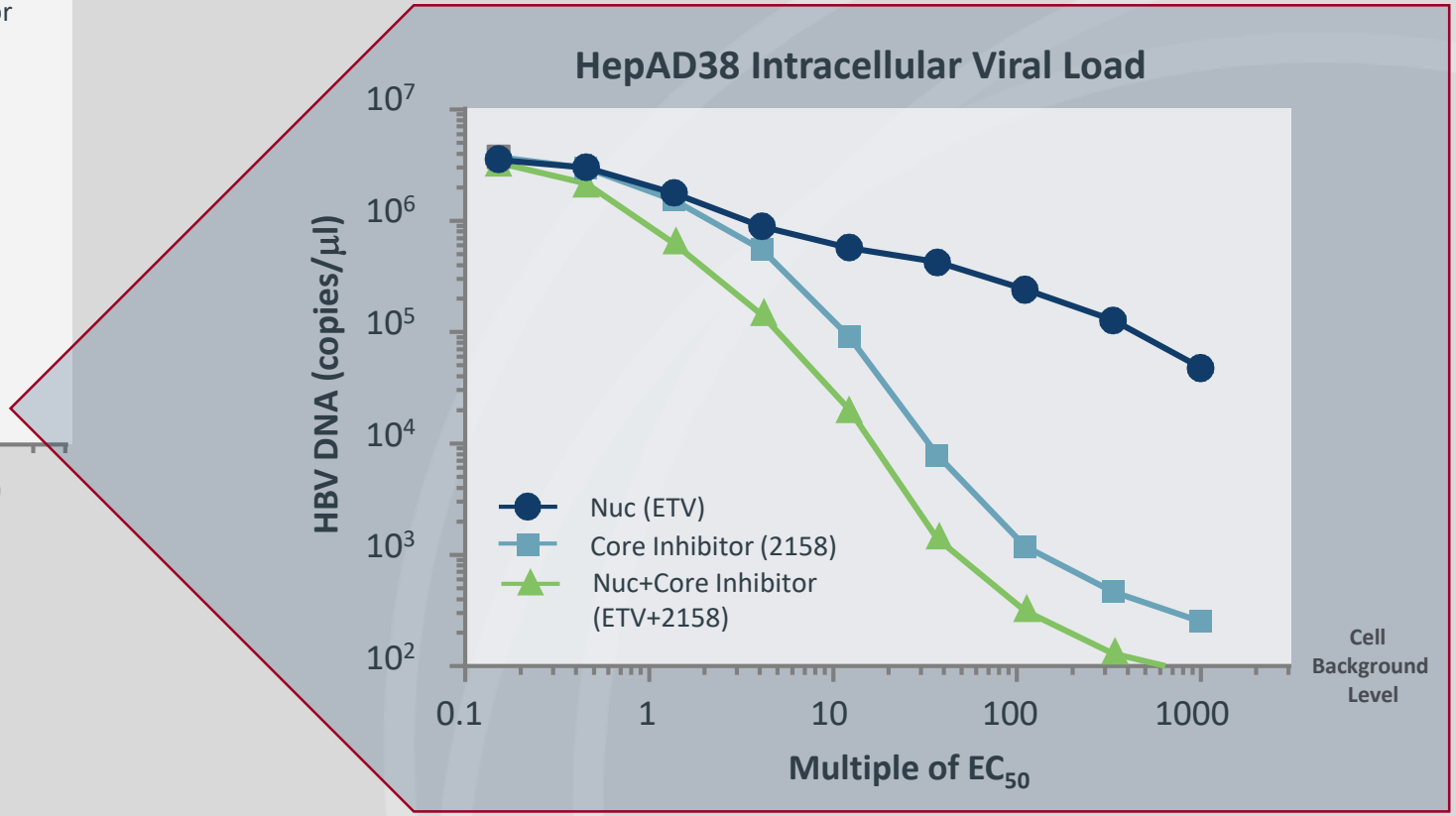
CORE INHIBITORS REDUCE KEY SURROGATE MARKERS FOR cccDNA



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

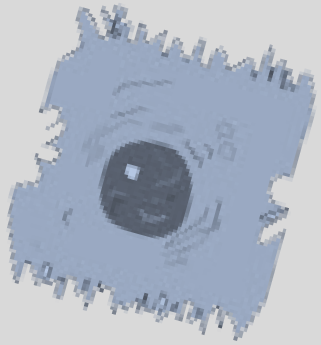
Confirm that genetic source of resistance is cccDNA

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

TBV EFFORT Study (Patient 17)

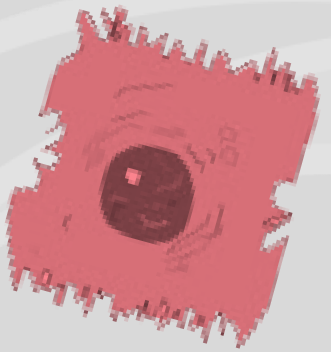
Sensitive — **TBV Treatment** — Resistant

Week	0	76	88	104
HBV DNA			70%	100%
HBV pgRNA				80%
HBV cccDNA				95%

IFN Study ML18376 (Patient 50)

Sensitive — **IFN Treatment** — LVD Resistant

Week	48	24	12	0
HBV DNA				100%
HBV pgRNA				100%



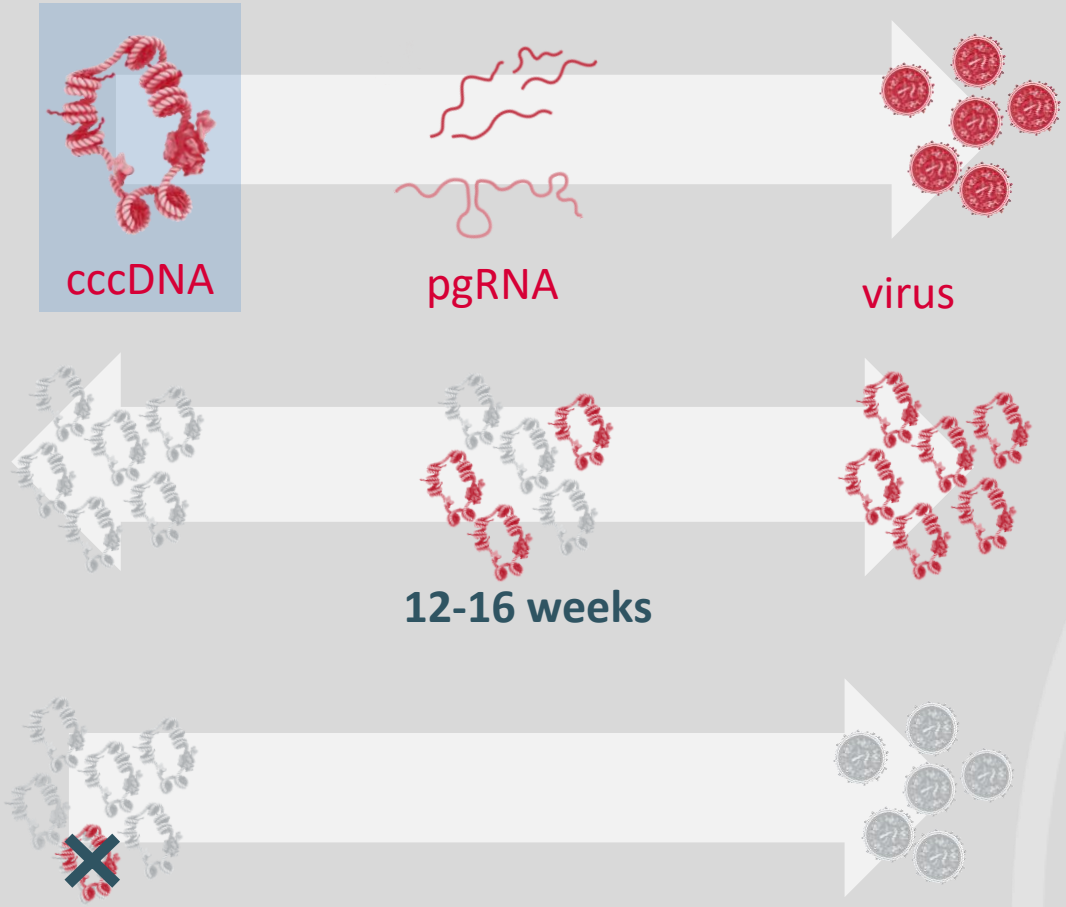
HBV^{R}-Infected Hepatocyte

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.

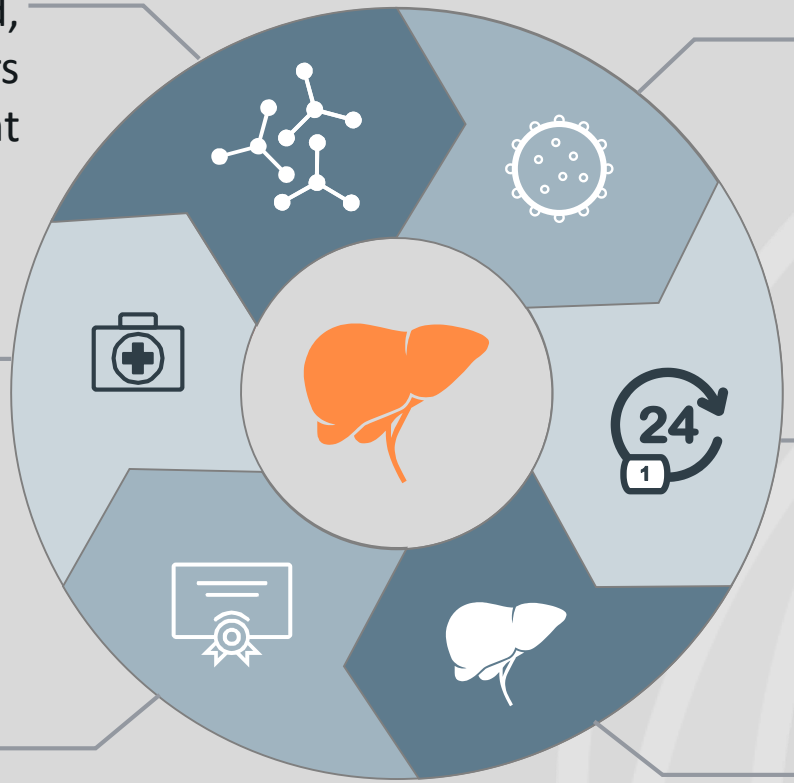
ABI-H0731 IS THE LEAD CANDIDATE FROM A PIPELINE OF UNIQUE CORE INHIBITORS

Preclinical Profile

Proprietary chemical scaffold, distinct from other core inhibitors in development

Clean safety profile in animal studies

USPTO patent allowance



Potent and selective activity against all major HBV genotypes (A-D)

QD dosing in patients

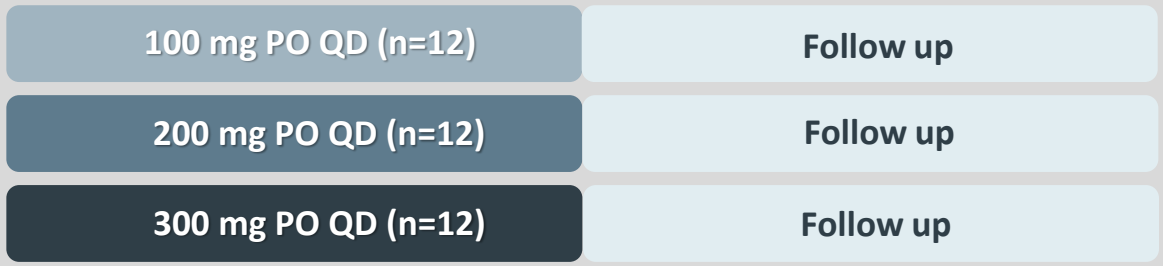
Excellent hepatocyte **stability**, minimal metabolism

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

ABI-H0731
PHASE 1 STUDY DESIGNS

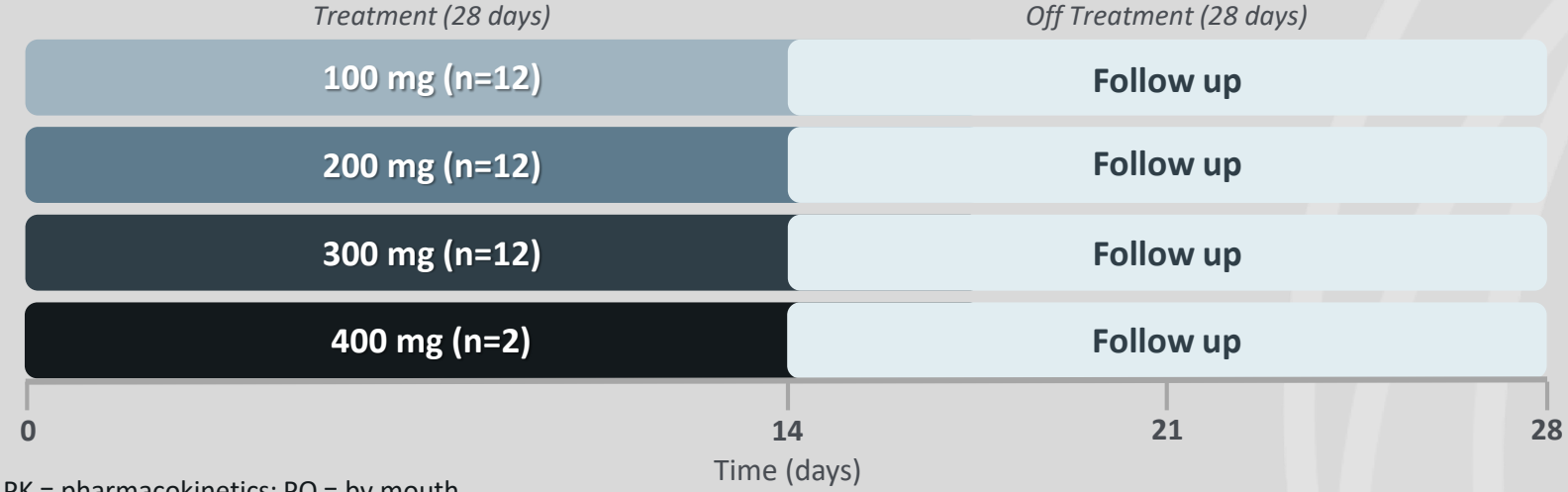
Study ABI-H0731-102

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

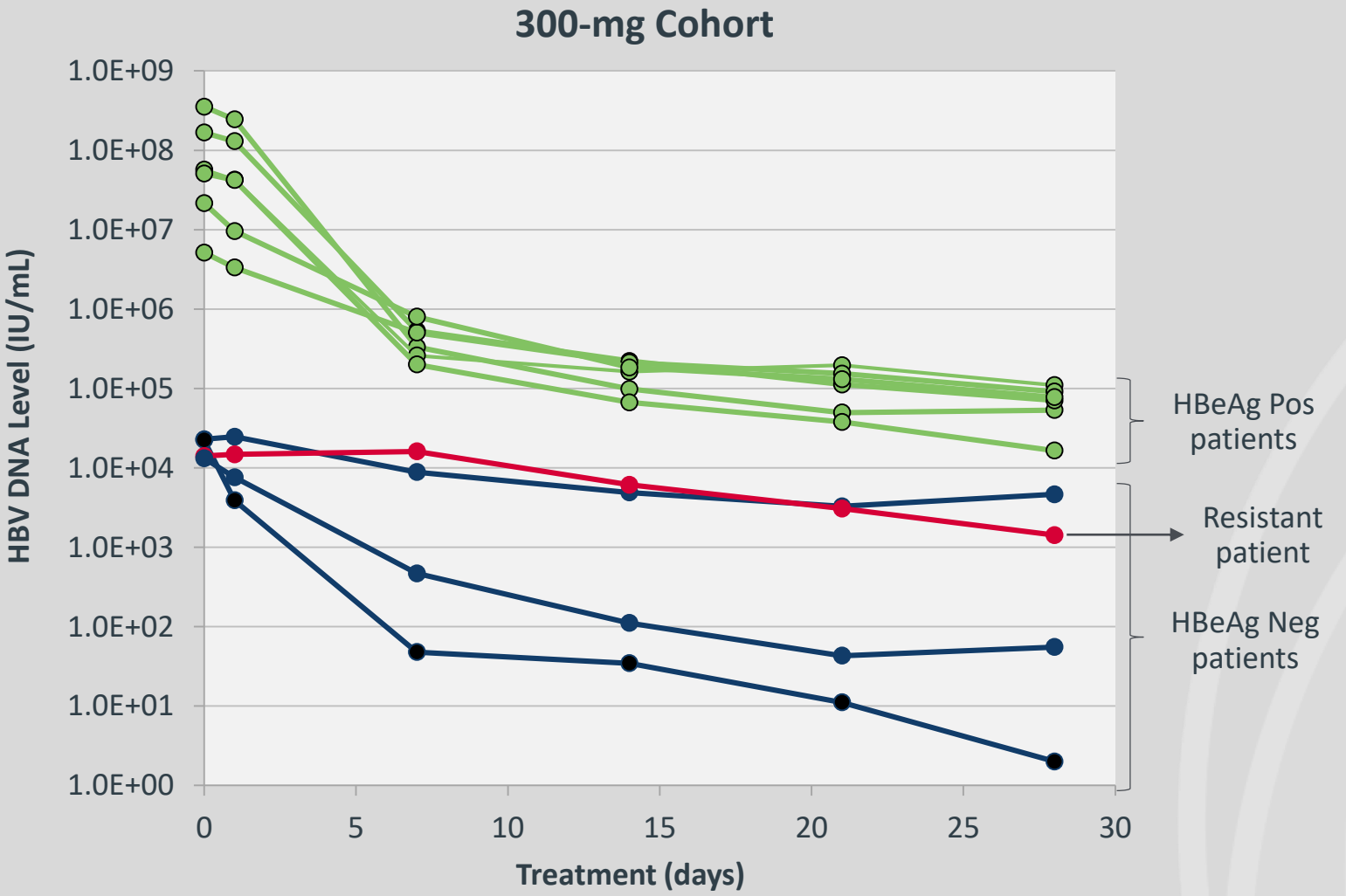
ABI-H0731
PHASE 1 – CLINICAL SAFETY SUMMARY

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.

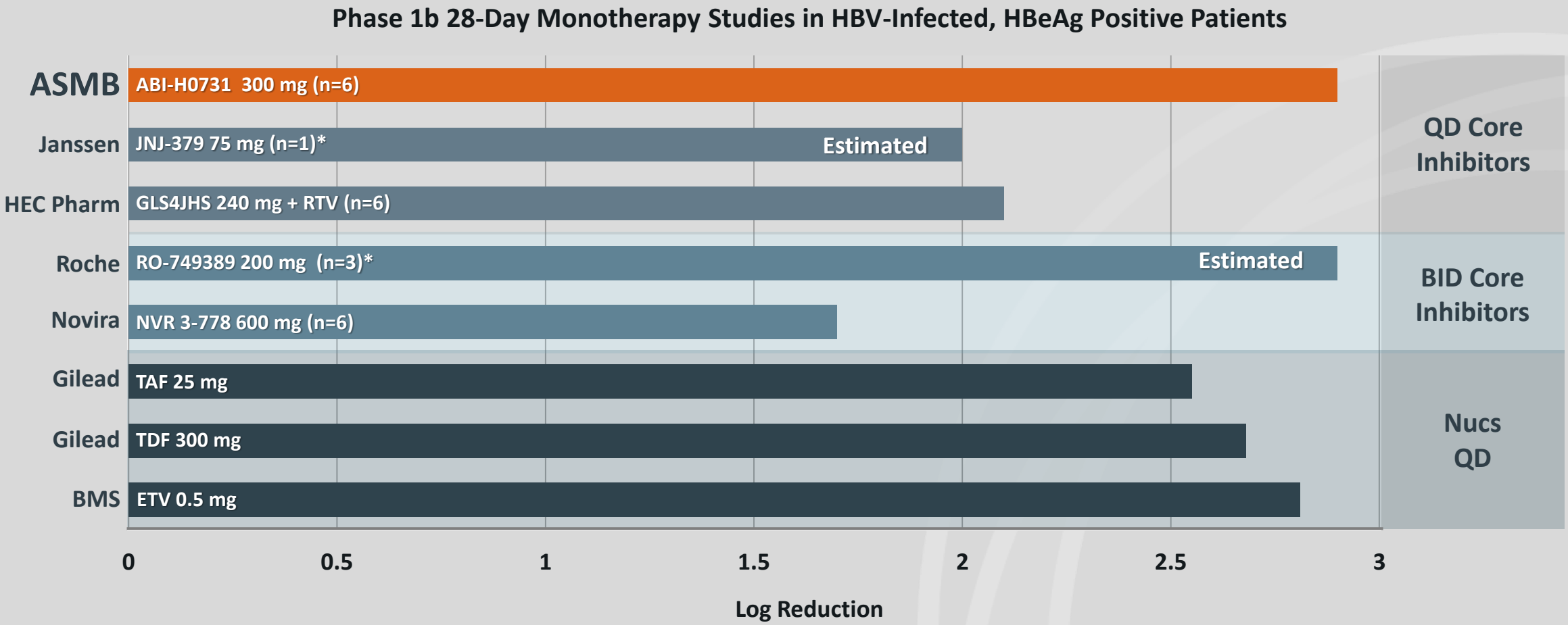
PHASE 1 STUDY – HBV DNA LEVELS IN TREATED HBV PATIENTS



- 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.

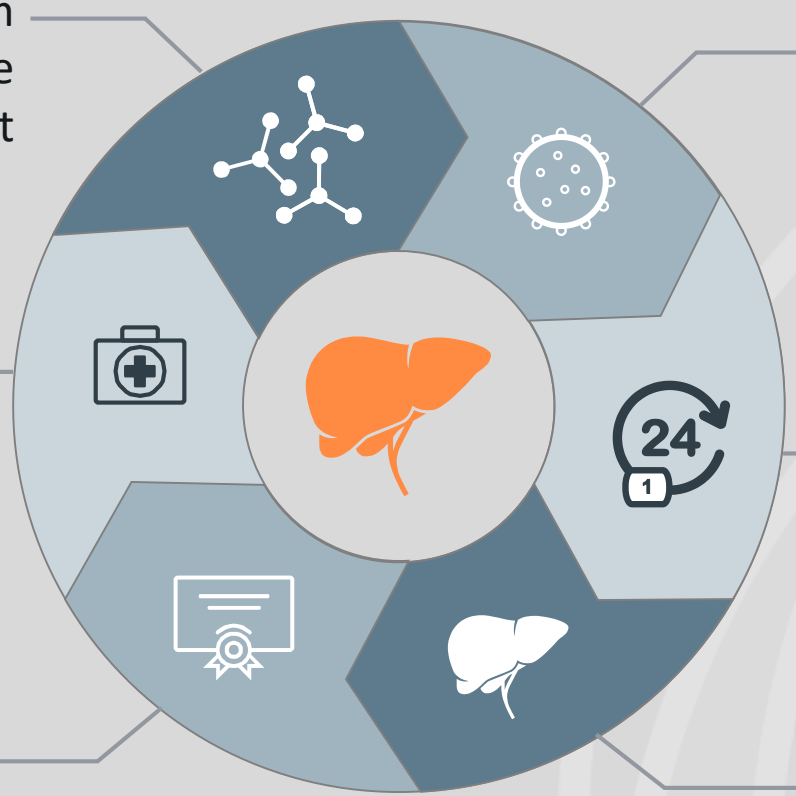
1. Yuen, et al. AASLD Poster LB-10 2015. 2. Ding, et al. AASLD Poster 920 2017. 3. Zoulim, et al. AASLD Poster LB-15. 2017. 4. Gane, et al. EASL Presentation 2018. 5. Yuen, et al. EASL Poster LBP-012 2018. 6. De Man, et al. *Hepatology*. Vol 34 2001. 7. Agarwal J. *Hepatology.* 2015;vol 62.

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

Preclinical Profile

Chemical scaffold is **distinct** from ABI-H0731 and other core inhibitors in development

Enhanced potency over first-generation core inhibitors



Undergoing preclinical **IND-enabling studies**

Favorable **drug-like properties,** with enhanced PK in animals

Establishing Strong IP portfolio

Excellent hepatocyte **stability**, minimal metabolism

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

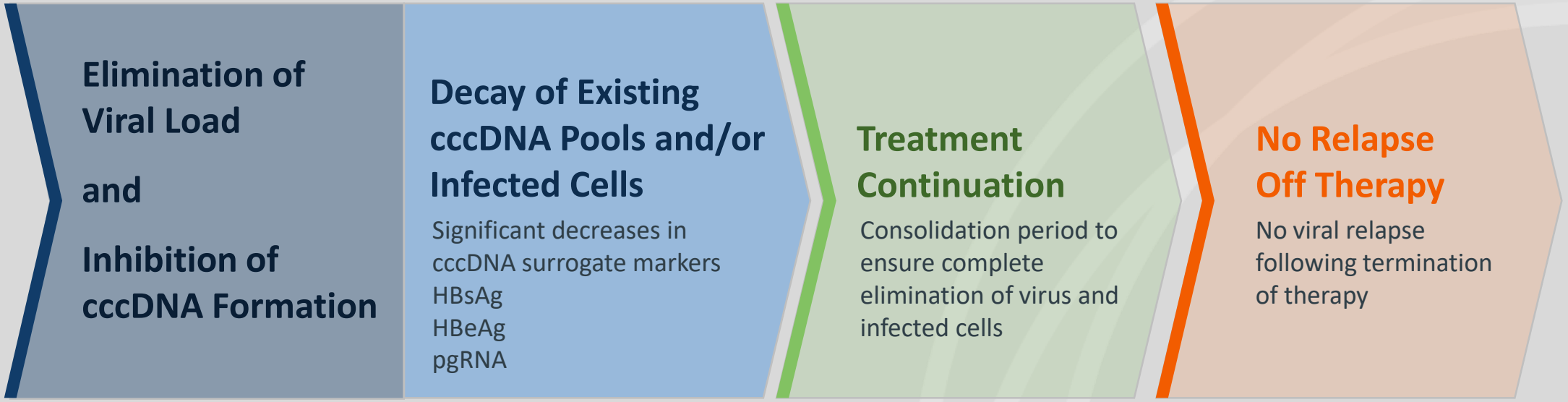
Animal Species	ABI-H0731	ABI-H2158	ABI-H0731	ABI-H2158
	C_{max} (µg/mL)	C_{max} (µg/mL)	AUC_{inf} (µg*hr/mL)	AUC_{inf} (µg*hr/mL)
Rat	6.4	24.8	62.1	445
Monkey	3.2	45.4	45.2	328
Dog	2.1	49.9	3.9	763

- 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

AGENDA

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URI LOPATIN, MD

Chief Medical Officer, Assembly Biosciences

Prior: Schering Plough, Roche, Gilead

assembly biosciences

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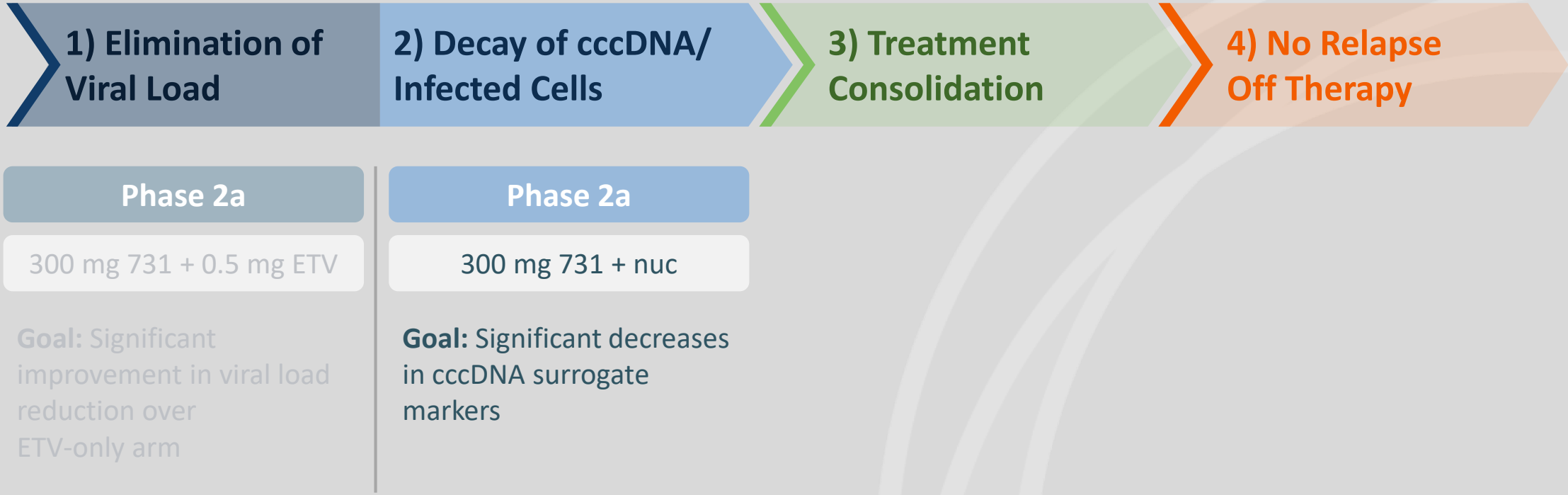
ABI-H0731 WILL BE AMONG THE FIRST CORE INHIBITORS EXPLORED IN AN HBV CURE PROGRAM



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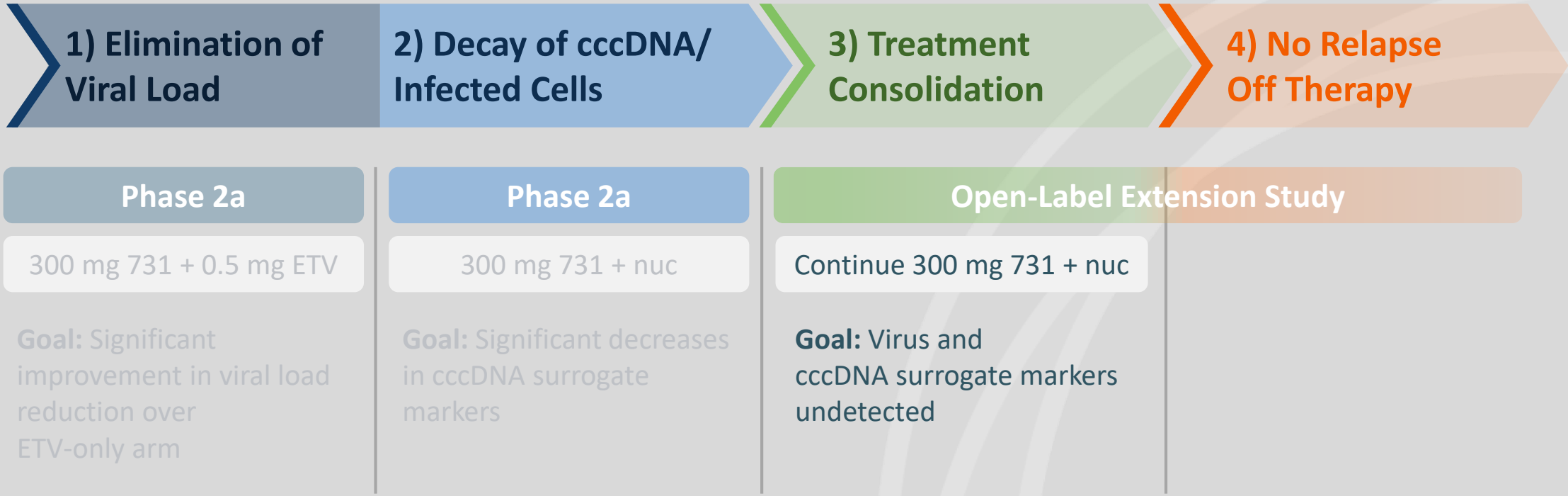

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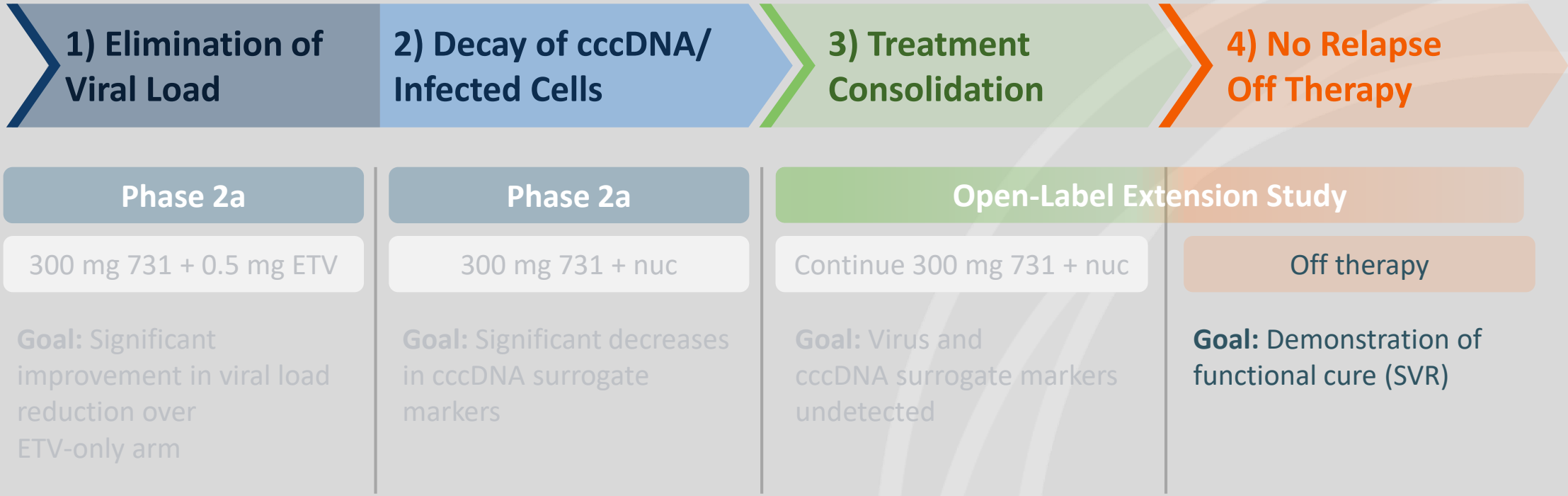


CORE INHIBITOR ROADMAP FOR FUNCTIONAL CURE

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

ABI-H0731 PHASE 2A STRATEGY AND DESIGNS

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

Viral Load Study

Patient population: nuc-naive, HBeAg+

- 0.5 mg ETV + 300 mg 731
- 0.5 mg ETV + placebo

Demonstrate significant improvement in efficacy

Viral Antigen POC Study

Patient population: nuc-suppressed, HBeAg+

- Continued nuc + 300 mg 731
- Continued nuc + placebo

Demonstrate significant decreases in cccDNA surrogate markers

0 — Time (months) — 6

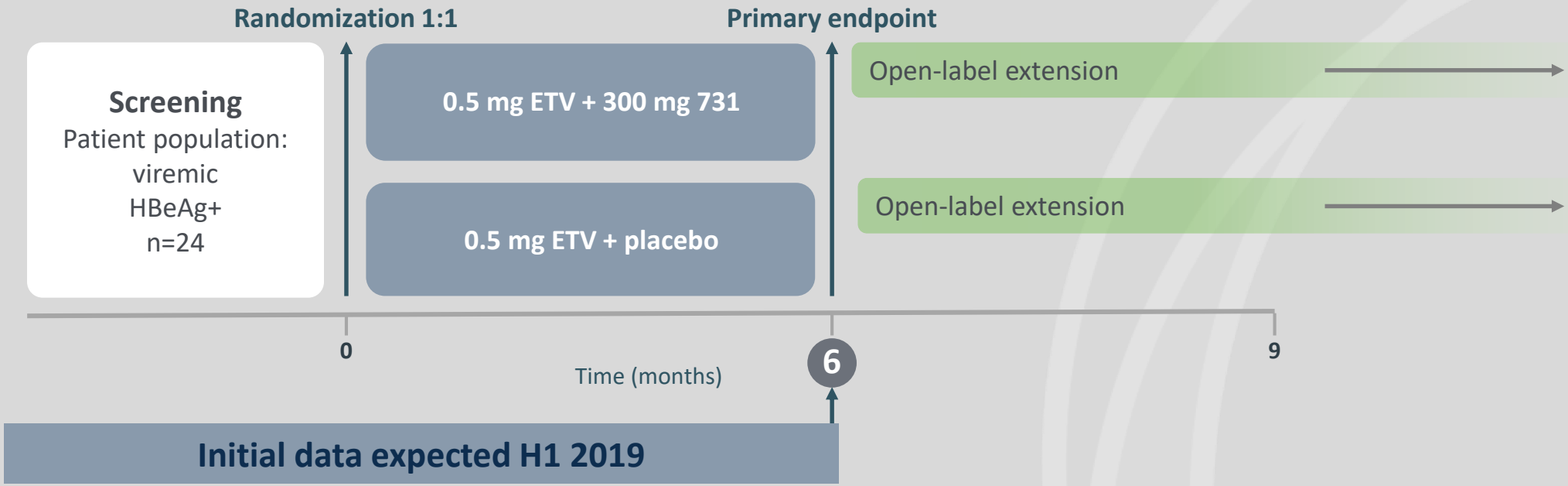
Initial data expected H1 2019

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

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

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

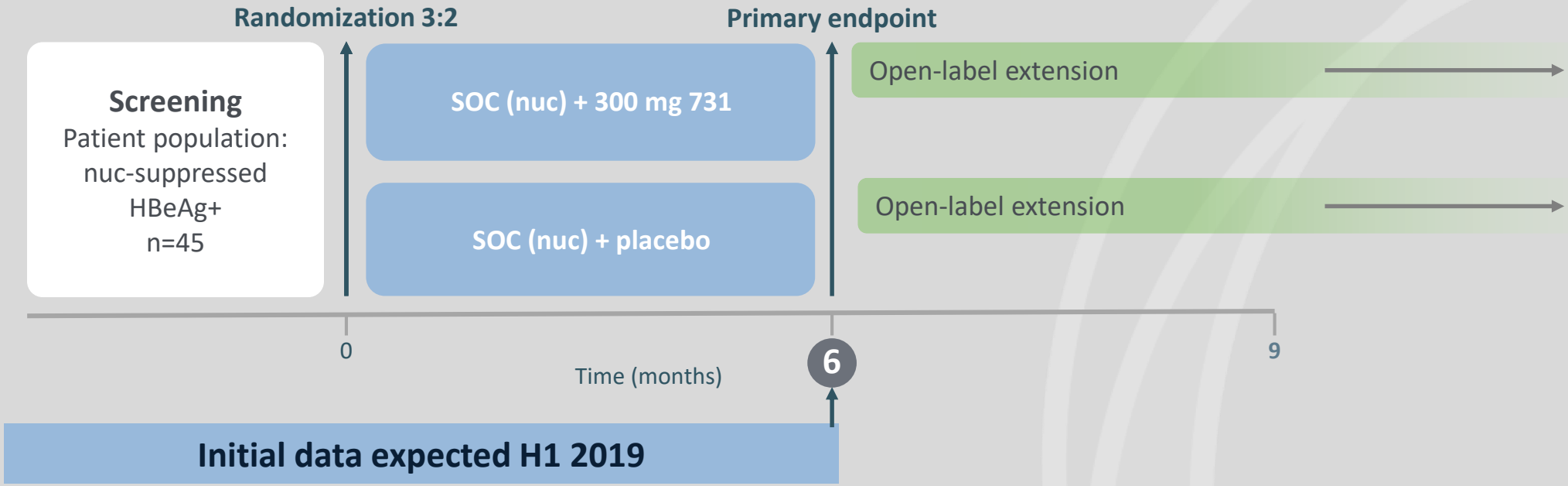
Goal: Demonstrate significant improvement in viral load reduction over ETV-only arm



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

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

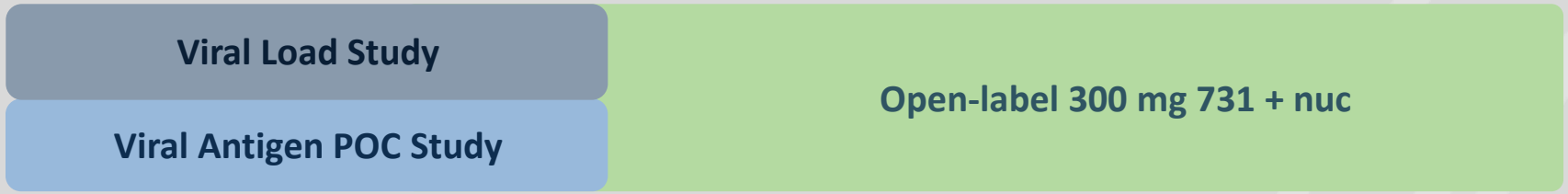
Goal: Demonstrate significant decreases in cccDNA surrogate markers (HBsAg and HBeAg)



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



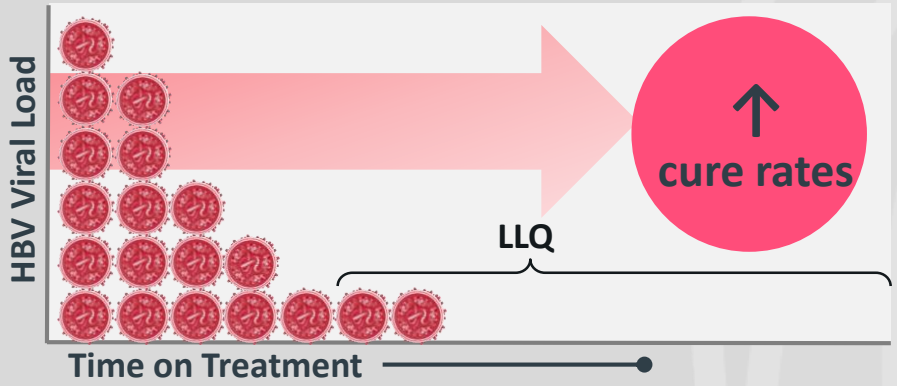
LLQ = lower limit of quantification.

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	Open-label 300 mg 731 + nuc
Viral Antigen POC Study	



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

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

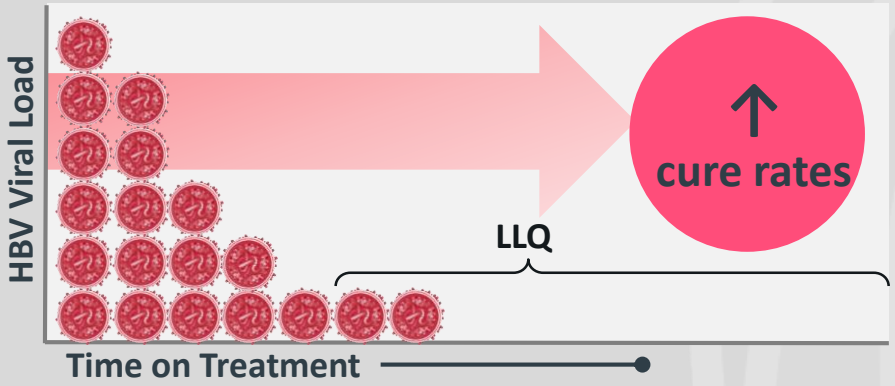
Viral Load Study

Viral Antigen POC Study

Open-label 300 mg 731 + nuc

Monitor off therapy for 6 months

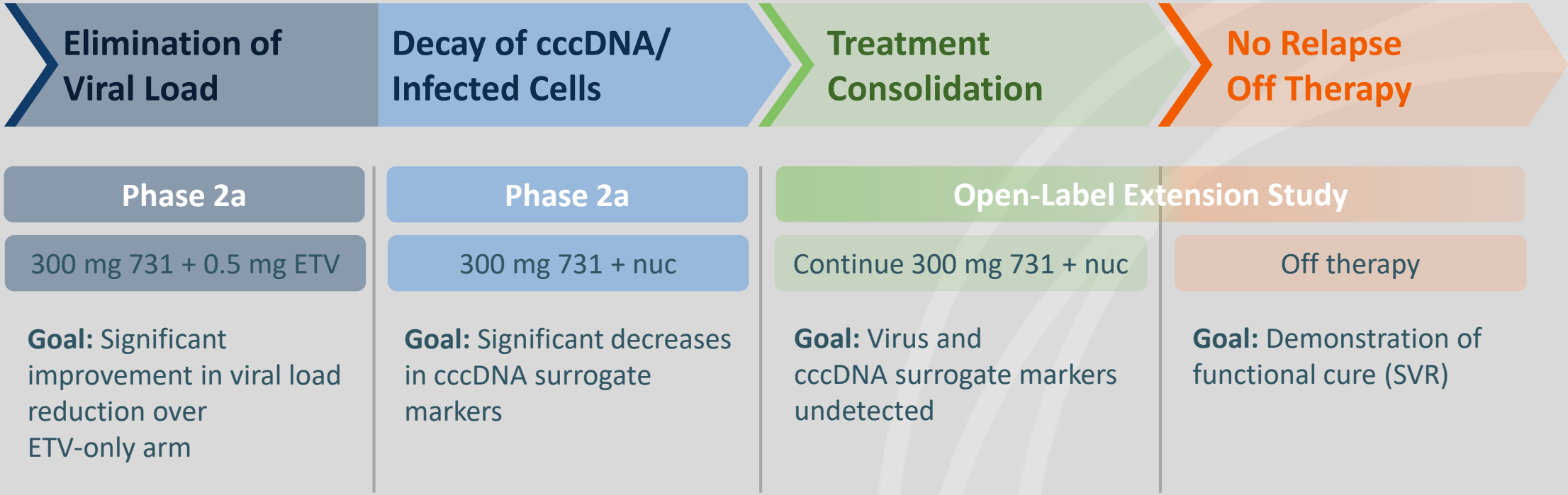
Complete response — **SVR**



Goal: SVR off therapy

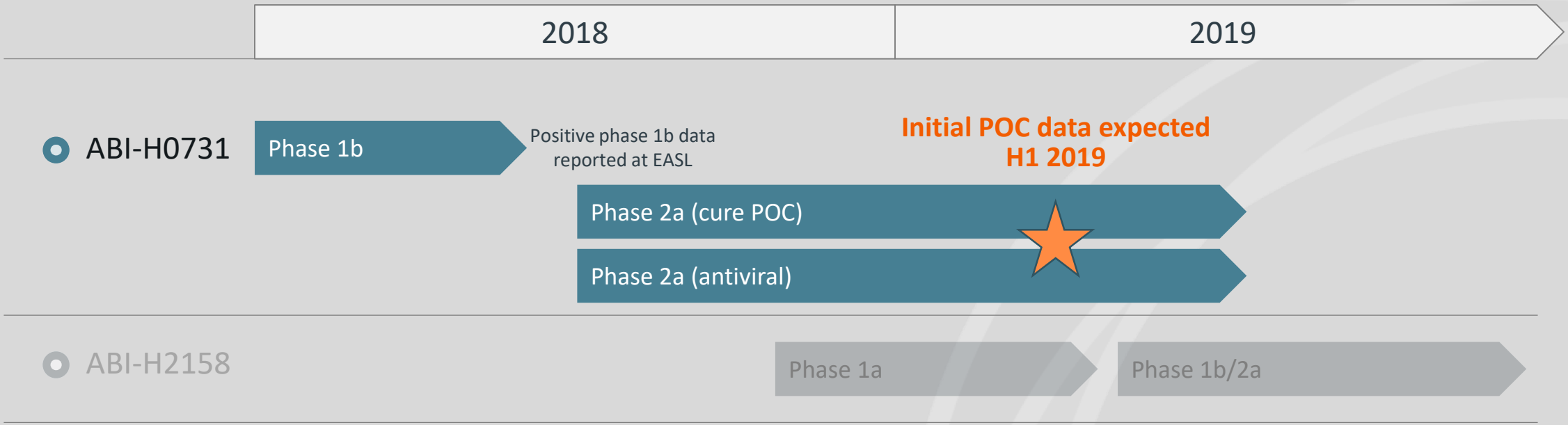
LLQ = lower limit of quantification.

CORE INHIBITOR ROADMAP FOR FUNCTIONAL CURE



INITIAL DATA EXPECTED H1 2019

EXPECTED CLINICAL TIMELINES AND MILESTONES



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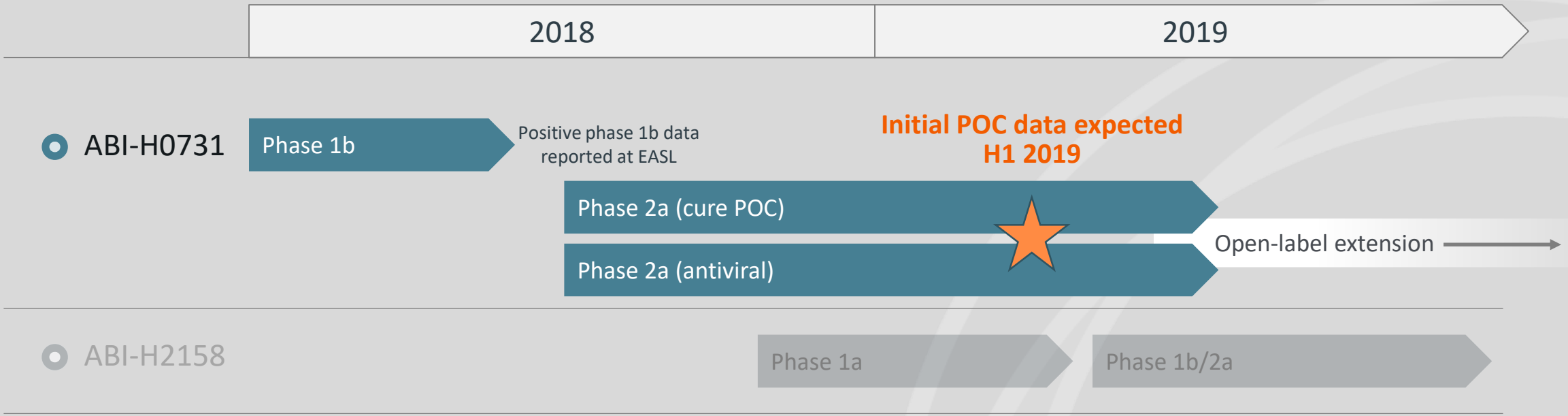

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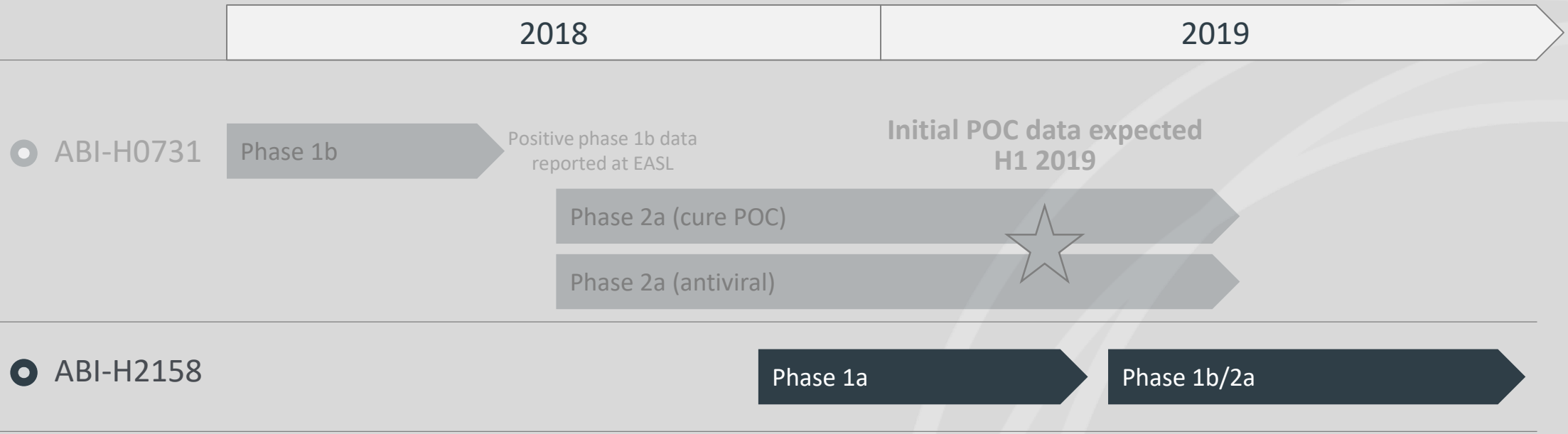

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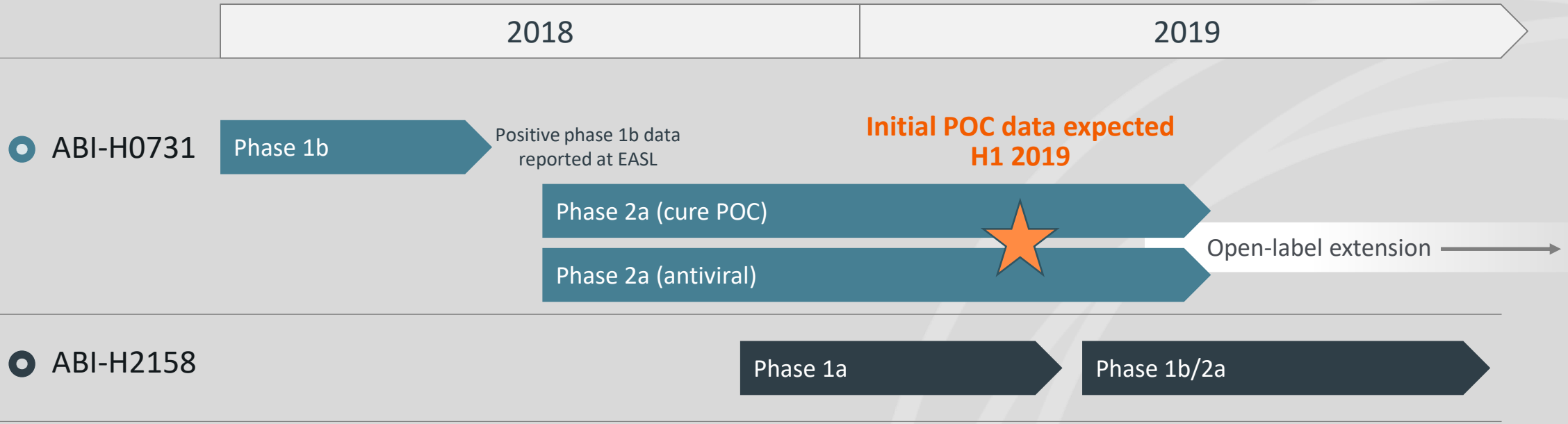

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JP BENYA

Vice President, Commercial

Commercial Perspectives on Hepatitis B

assembly biosciences

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

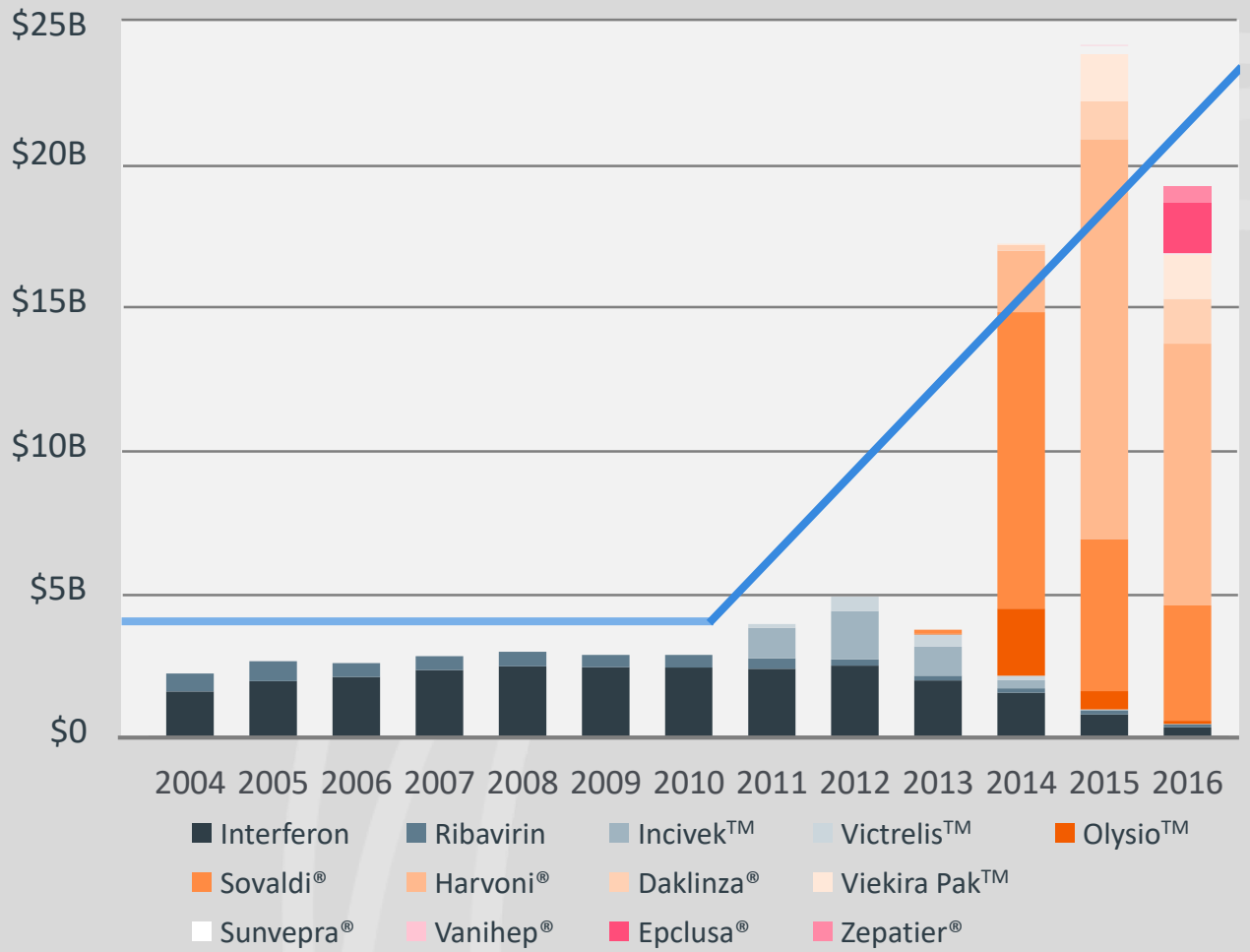
WHY?

- 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.

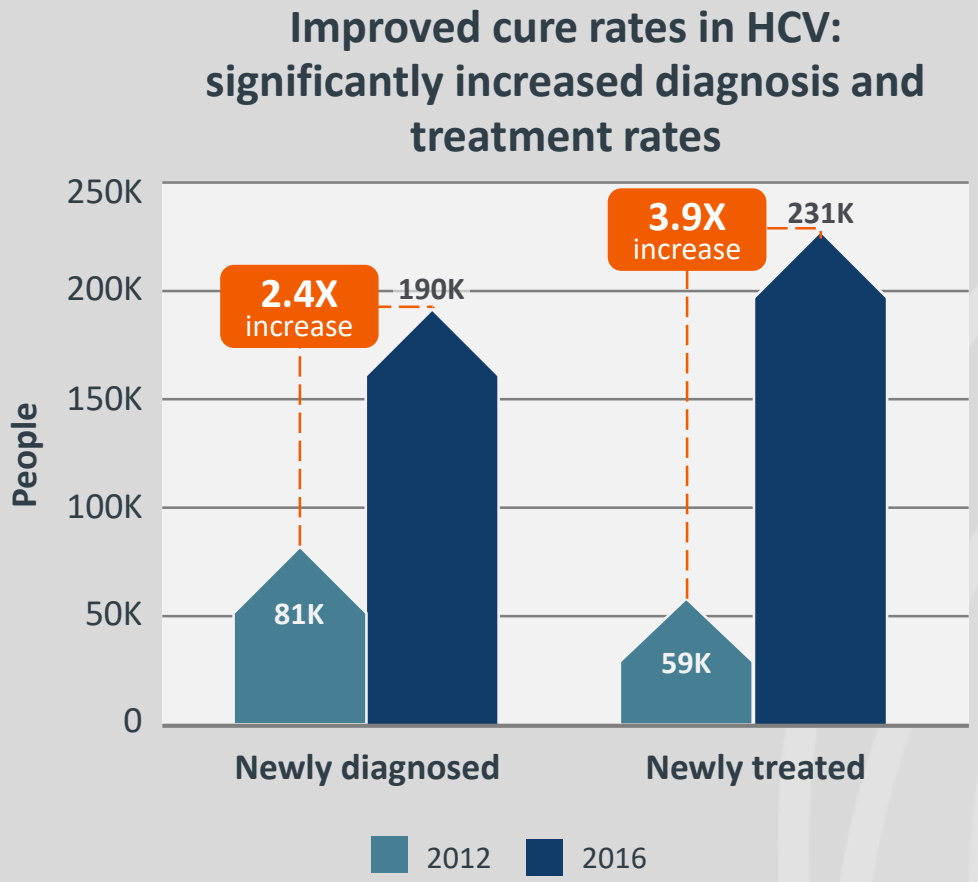
THE HISTORY OF HCV THERAPY

- 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.

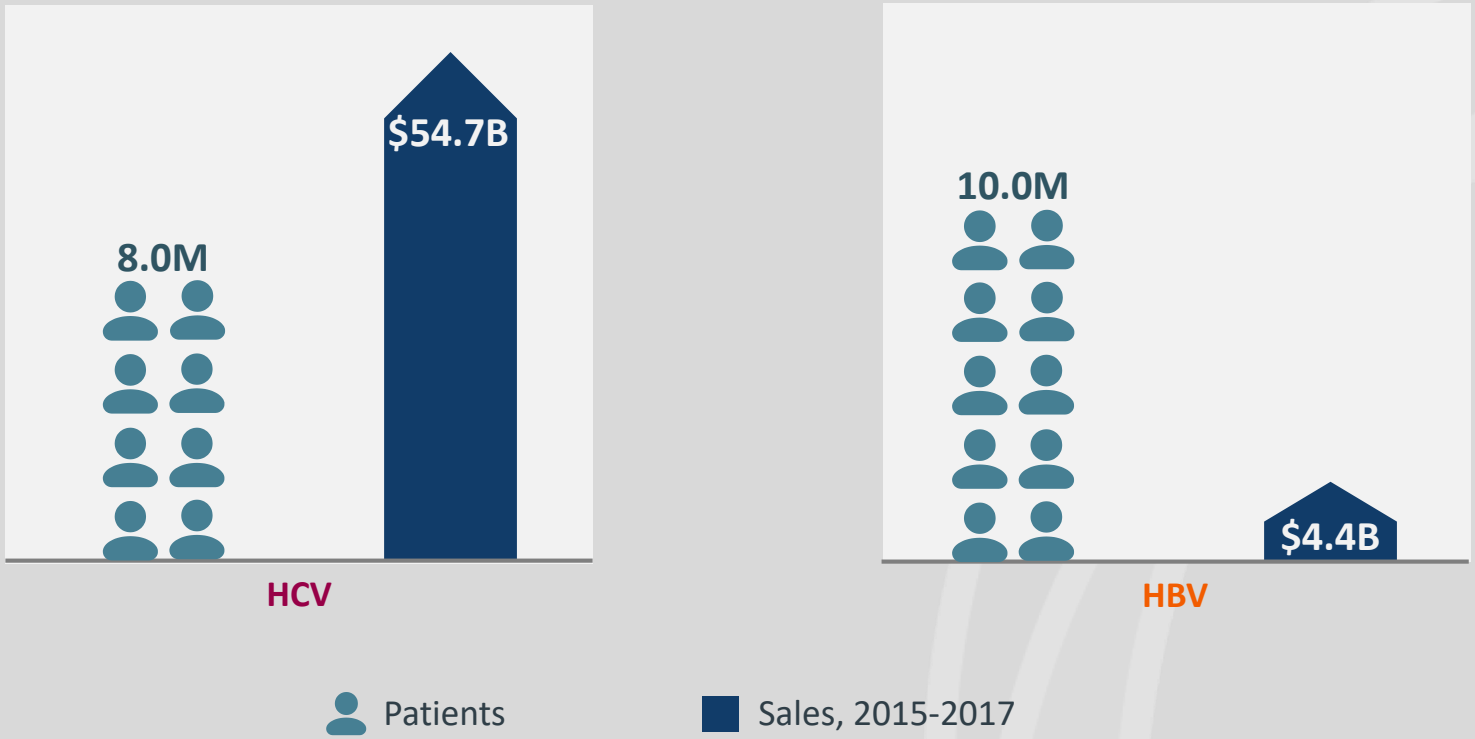
THE HCV EXPERIENCE…



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.

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MANAGEMENT AND KOL Q&A SESSION

Speakers:

Dr. Douglas Dieterich

Dr. Jörg Petersen

Dr. Richard Colonno

Dr. Uri Lopatin

JP Benya

Moderator:

Derek Small

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CLOSING REMARKS

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