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): **September 25, 2017**

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.

On September 25, 2017, Assembly Biosciences, Inc. (the "Company") issued a press release announcing that is has selected a second Core protein Allosteric Modulator (CpAM), ABI-H2158, as a candidate for clinical development and that investigational new drug enabling studies in preparation of a Phase 1a clinical trial are underway. CpAMs are small molecules that directly target and allosterically modulate the hepatitis B virus core (HBc) protein. A copy of the press release is being furnished with this Current Report on Form 8-K as Exhibit 99.1 and is hereby incorporated by reference into this Item 7.01.

In addition, the Company is furnishing a presentation, attached as Exhibit 99.2 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 September 25, 2017. The presentation will also be available on the Company's website at http://investor.assemblybio.com/events.cfm.

The information in this Item 7.01, Exhibit 99.1 and Exhibit 99.2 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 exhibits relating to Item 7.01 shall be deemed furnished and not filed.

Exhibit No.	Description
99.1	Press Release, dated September 25, 2017.
99.2	Assembly Biosciences, Inc. Presentation titled "Targeting HBV Core Protein to Achieve Higher Cure Rates - September 25, 2017.

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: September 25, 2017

Assembly Biosciences, Inc.

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

EXHIBIT INDEX

Exhibit No.	Description
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99.2	Assembly Biosciences, Inc. Presentation titled “Targeting HBV Core Protein to Achieve Higher Cure Rates - September 25, 2017.

Exhibit 99.1

Assembly Biosciences Selects Next-Generation CpAM Candidate for Advancement into Clinical Development

Indianapolis, IN, September 25, 2017 – Assembly Biosciences, Inc. (NASDAQ: ASMB), a clinical-stage biotechnology company advancing a new class of oral therapeutics for the treatment of hepatitis B virus (HBV) infection and novel oral live biotherapeutics for disorders associated with the microbiome, today announced that it has selected a second Core protein Allosteric Modulator (CpAM), ABI-H2158, as a candidate for clinical development and that IND-enabling studies in preparation for initiation of a Phase 1a clincial trial are underway.

Dr. Richard Colonno, Chief Scientific Officer for Assembly's HBV program, who is presenting today at an HBV-focused conference, commented, "Our HBV program aims to increase cure rates by targeting the essential viral Core protein with our direct-acting CpAMs, which have been shown in *in vitro* studies to suppress both viral replication and most importantly, the cccDNA formation associated with viral persistence. In other *in vitro* studies, our next-generation clinical candidate, ABI-H2158, displayed enhanced potency while maintaining the same favorable drug-like (DMPK) characteristics of our first clinical candidate, ABI-H0731."

Assembly's first-generation CpAM, ABI-H0731, completed a Phase 1a study earlier this year and is currently being evaluated in HBV patients in a double blind, placebo-controlled, Phase 1b study. Assembly will present additional data on its CpAM pipeline, including the ABI-H0731 Phase 1a study results, at the Annual Meeting of the American Association for the Study of Liver Diseases (AASLD) in October.

In addition, Dr. Colonno noted, "We are looking forward to to discussing more about the characteristics of our next-generation CpAMs at AASLD, as well as new data that may shed light on the biological half-life of cccDNA that could have implications for our therapeutic approach."

*** Discovery on Target: Targeting HBV** - Boston
Date: September 25, 2017
Panel: Targeting HBV Core Protein to Achieve Higher Cure Rates
Presenter: Richard Colonno, PhD., Chief Scientific Officer, HBV Program, Assembly Biosciences

A copy of Dr. Colonno's presentation will be available in the Events and Presentations section of the Company's website later today, at www.assemblybio.com.

About Assembly Biosciences
Assembly Biosciences, Inc. is a clinical-stage public biotechnology company developing two innovative platform programs: an HBV program advancing a new class of oral therapeutics for the treatment of hepatitis B virus (HBV) infection and a microbiome program developing novel oral live biotherapeutics designed to address diseases associated with the microbiome. Assembly's HBV program is advancing multiple drug candidates with the aim of increasing cure rates in patients with chronic HBV. The company's microbiome program consists of a fully integrated platform that includes a robust strain identification and selection process, methods for strain isolation and growth under current Good Manufacturing Practices and a patent-pending delivery system, GEMICEL®, which allows for targeted oral delivery of live biologic and conventional therapies to the lower gastrointestinal tract. Assembly is developing a robust pipeline of product candidates in multiple disease indications. For more information, visit www.assemblybio.com.

Forward-Looking Statement
The information in this press release contains forward-looking statements regarding future events, including statements about the clinical and therapeutic potential of Assembly's development 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 "designed" or "developing." Assembly intends such forward-looking statements 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. Actual results or developments may differ materially from those projected or implied in these forward-looking statements. More information about the risks and uncertainties faced by Assembly are more fully detailed under the heading "Risk Factors" in Assembly's Annual Report on Form 10-K for the year ended December 31, 2016, and Quarterly Report on Form 10-Q for the quarter ending June 30, 2017 filed with the Securities and Exchange Commission. Except as required by law, Assembly assumes no obligation to update publicly any forward-looking statements, whether as a result of new information, future events or otherwise.

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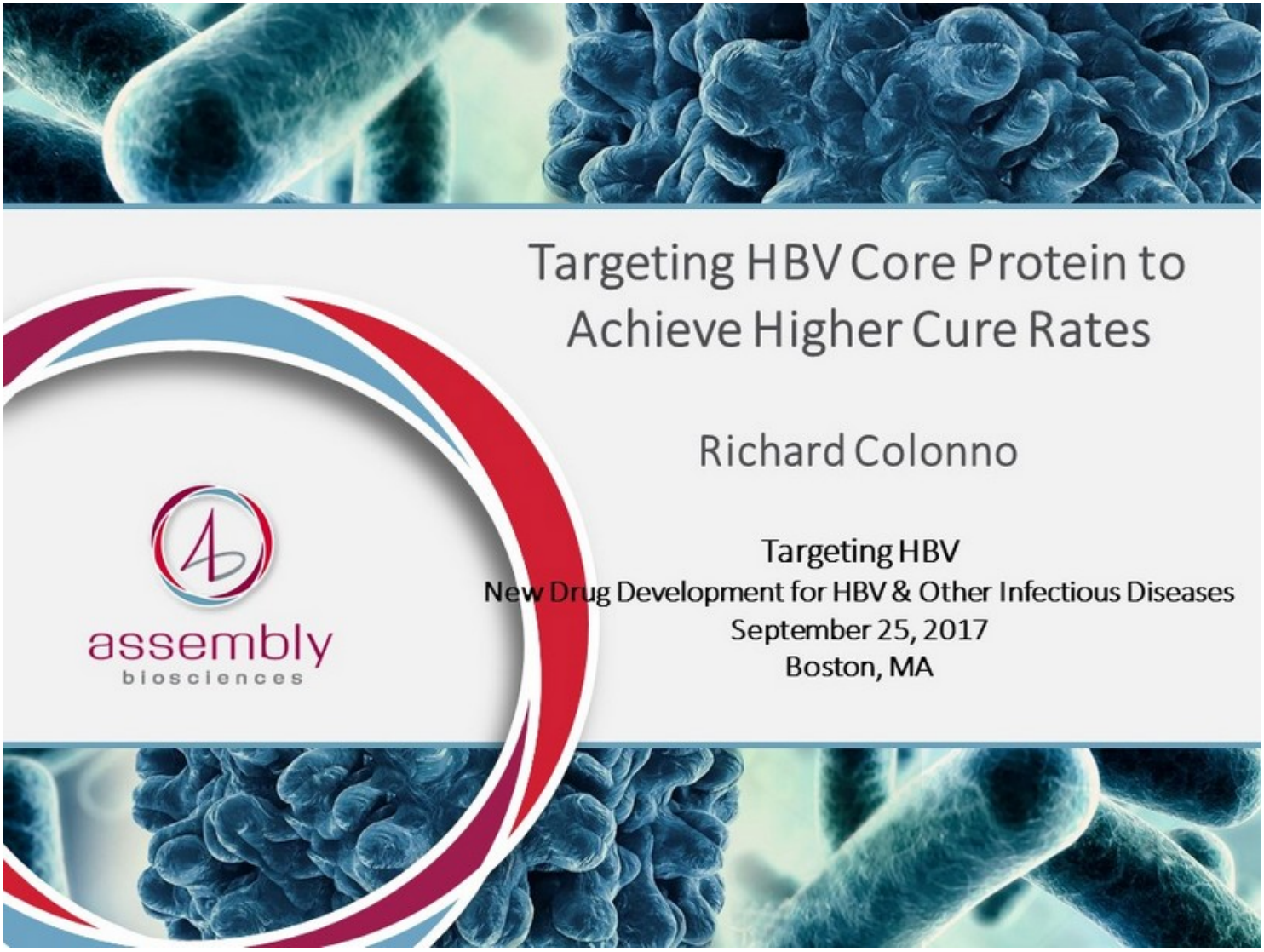
Exhibit 99.2

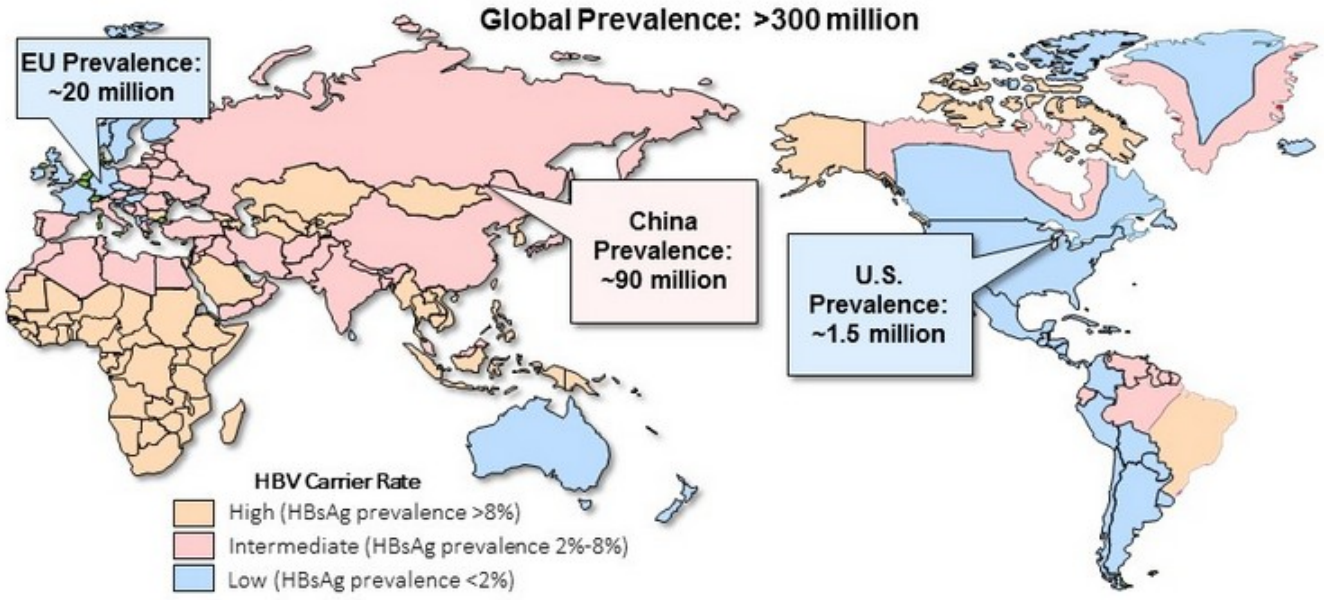

Targeting HBV Core Protein to Achieve Higher Cure Rates
Richard Colonno
assembly biosciences
Targeting HBV
New Drug Development for HBV & Other Infectious Diseases
September 25, 2017
Boston, MA

Cautionary Note Regarding Forward-looking Statements

Some of the information in this presentation contains forward-looking statements regarding future events, including statements about the clinical and therapeutic potential of Assembly's development programs and drug candidates. Certain forward-looking statements may be identified by reference to a future period or periods or by use of forward-looking terminology such as "may," "expected," "should" or "predictive." Assembly intends such forward-looking statements 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. Actual results or developments may differ materially from those projected or implied in these forward-looking statements. More information about the risks and uncertainties faced by Assembly are more fully detailed under the heading "Risk Factors" in Assembly's Annual Report on Form 10-K for the year ended December 31, 2016, and Quarterly Report on Form 10-Q for the quarter ending June 30, 2017 filed with the Securities and Exchange Commission. Except as required by law, Assembly assumes no obligation to update publicly any forward-looking statements, whether as a result of new information, future events or otherwise.

Significant Need Remains for Curative Therapies

- Number of chronically-infected HBV patients exceeds the number of patients infected with HCV (~170M) plus HIV (~37M) combined
- The majority are undiagnosed – often asymptomatic for years
- Chronic HBV infection results in chronic inflammation and progressive liver damage, potentially leading to liver cirrhosis, HCC and death (~1M deaths/year)



Jidong Jia. HBV HCV epidem & therapy 2013
http://www.who.int/hepatitis/en/

Current HBV Therapies

- Currently Approved
  - Nucleoside Analogs: Entecavir, Lamivudine, Telbivudine
  - Nucleotide Analogs: Tenofovir, Adefovir, Tenofovir Alafenamide
  - Interferons (IFN and peg-IFN)
- Entecavir and Tenofovir
  - Safe, highly effective therapies and the current drugs of choice
  - Target the viral polymerase, inhibiting reverse transcription of negative-strand DNA from pgRNA and positive-strand HBV DNA synthesis to generate rcDNA
  - Highly effective reduction and maintenance of HBV DNA at undetectable levels in virtually all treatment-naïve patients
  - HBV DNA undetectability maintained for prolonged periods (years)
  - One pill, once-a-day dosing
  - Very well tolerated, with no meaningful resistance emergence over prolonged treatment periods
  - Unfortunately, cure rates are very low despite prolonged therapy

Deficiencies of Current Approved Therapies

Results at 48 Weeks

HBV Patients	ETV[1,2]	TFV[3]	Peg-IFN[4,5]
HBeAg Positive	N = 354	N = 176	N = 271
HBV DNA Undetectable	67%	76%	25%[a]
HBeAg Seroconversion	21%	21%	27%
ALT Normalization	68%	68%	39%
HBsAg Loss	2.0%	3.2%	2.9%
HBeAg Negative	N = 325	N = 250	N = 177
HBV DNA Undetectable	90%	93%	63%[a]
ALT Normalization	78%	76%	38%
HBsAg Loss	0.3%	0%	0.6%[b]

[a] HBV DNA <400 copies/mL; [b] 72 weeks

Table courtesy of Geoff Dusheiko

[1] Chang T-T, et al. N Engl J Med 2006:354:1001-10

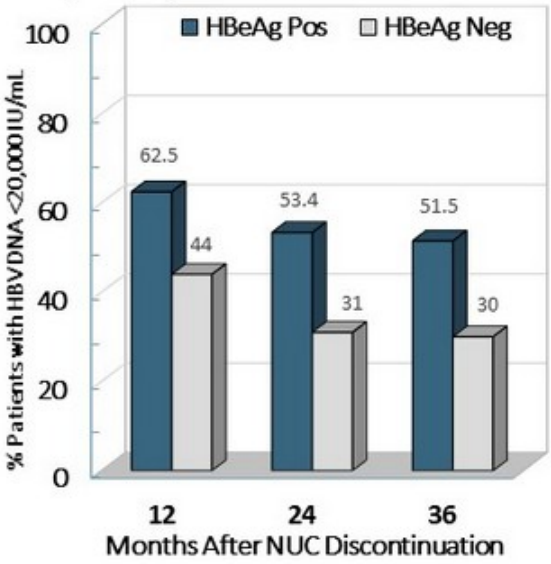
[2] Lai C-L, et al. N Engl J Med 2006:354:1011-20

[3] Marcellin P, et al. N Engl J Med 2008:359:2442-55

[4] Lau GKK, et al. N Engl J Med 2005:352:2682-95

[5] Marcellin P, et al. N Engl J Med 2004:351:1206-1

Virologic Relapse After Nuc Discontinuation



HBeAg Positive Patients

14 studies, 733 initially HBeAg positive

Pooled HBsAg loss: 1%

HBeAg Negative Patients

17 studies, 967 HBeAg negative

Pooled HBsAg loss: 1.7%

Papatheodoridis G. et al, Hepatology 2016

Thought We Had A Chance for Cure 12 Years Ago

Woodchuck Hepatitis B Virus Model

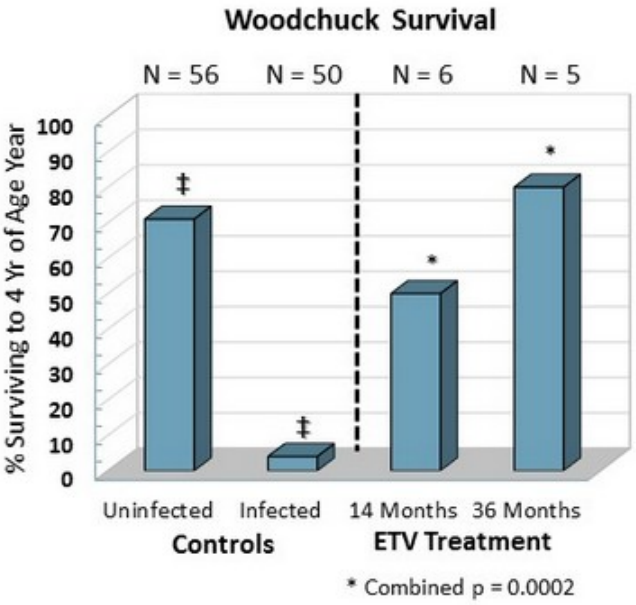
- WHBV infection at 3 days of age results in a carrier state with life-long viremia
- Chronically-infected animals mimic the HBV carrier state in man (viral pathogenesis & development of HCC)
- Infected woodchucks have a >90% chance of dying of HCC within 4 years
- Predictive model for toxicity and effectiveness of antivirals in man
- ETV potency against WHBV equivalent to HBV

Long-term treatment study conducted to determine if prolonged ETV therapy could cure woodchucks?

Colonno, et al. JID 2001;184:1236-45

Long-Term Woodchuck Study: Cure and Survival

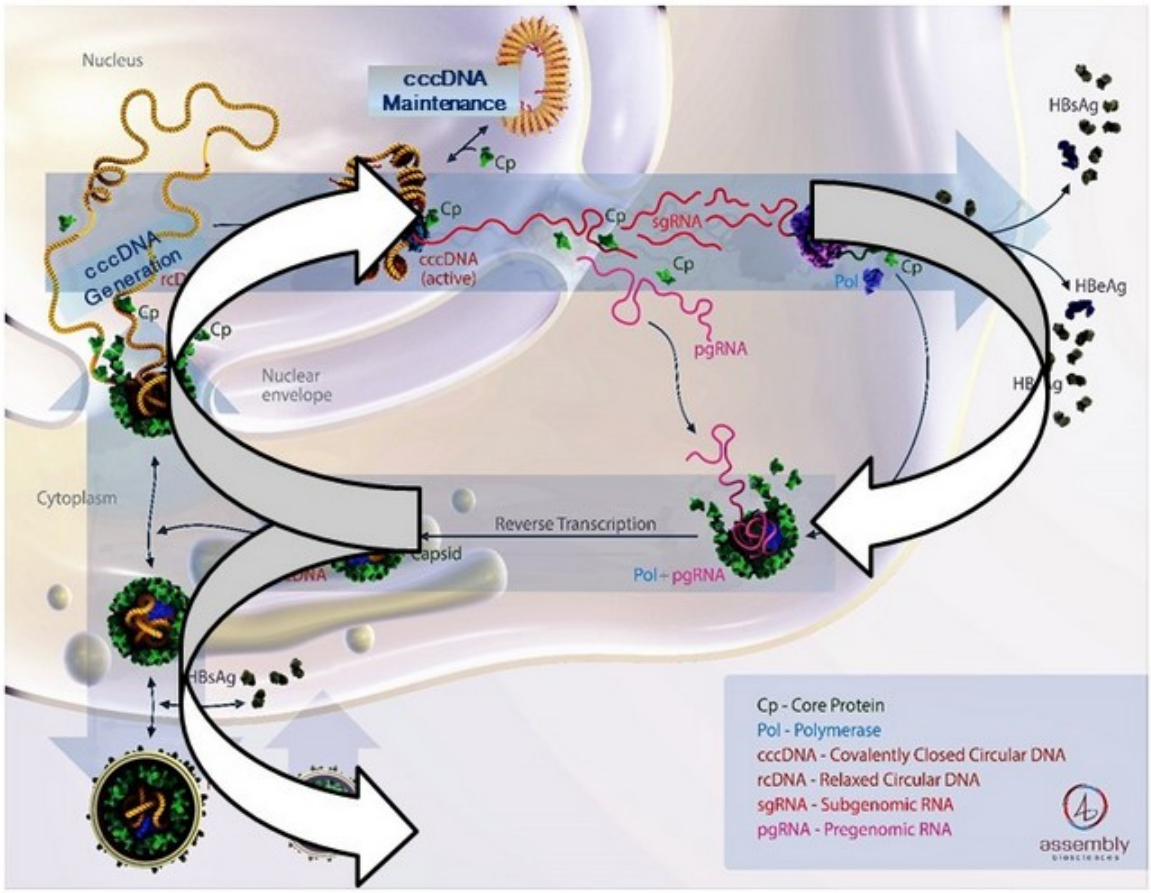
- ETV Treatment (0.5 mg/kg)
  - Two months of daily treatment
  - 12 or 34 months of weekly treatment
- Sustained suppression (≥ 8 logs) of viral DNA levels for 1-3 years – no rebounds or evidence of resistance
- cccDNA levels reduced >4 logs
- WHBsAg levels reduced 91% at Week 96
- Survival in ETV-treated animals significantly improved over historical controls
- Clear evidence that ETV can cure woodchucks based on multiple parameters



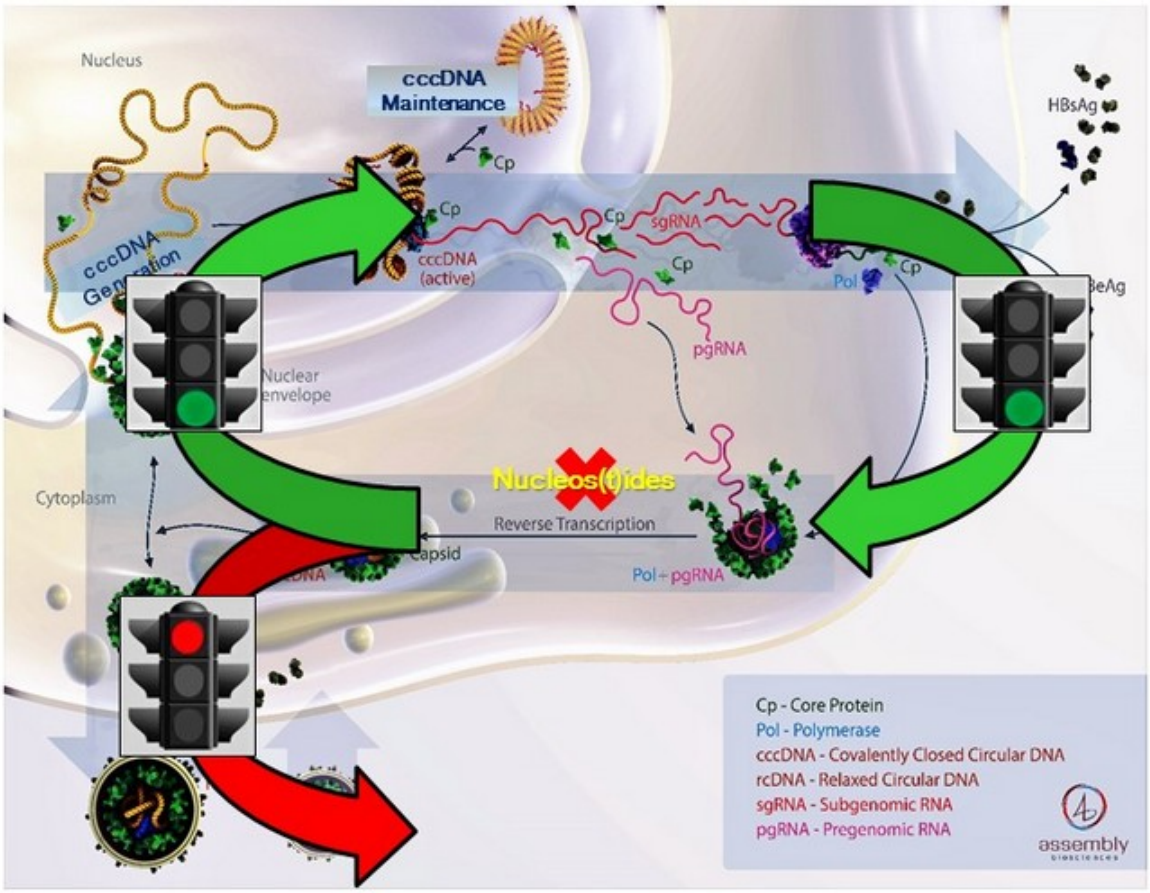
Unfortunately, this is not what happens in HBV patients with prolonged therapy

‡Historical control. Tennant, et al. Viral Hepatitis and Liver Disease 1988: 462-464
Colonno, et al. JID 2001;184:1236-45

HBV Life Cycle: Complex and Poorly Understood



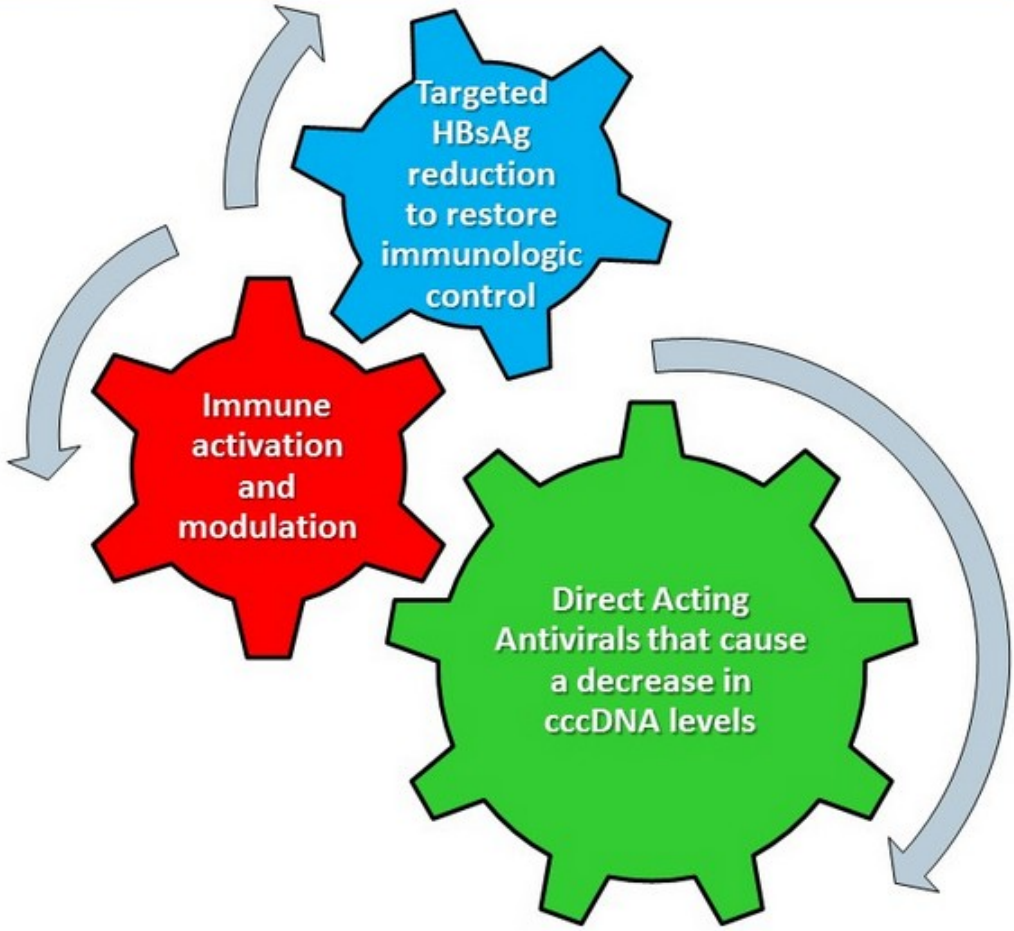
HBV Life Cycle: Failure of Nucs to Inhibit cccDNA



Aspirational Objectives for Clinical Cure in Humans

- We want what we achieved in woodchucks!
- Must cause depletion of cccDNA pools
  - Inhibit generation of new cccDNA
  - Direct silencing or elimination of existing cccDNA (more challenging)
- Decrease HBsAg levels to restore/enhance immune response
- Treatment of less than 2 years
  - Convenient dosing (QD?) and low pill burden
  - Excellent safety profile, with minimal side effects
- Sustained remission off therapy
  - Viral DNA replication remains undetectable
  - Elimination of cccDNA reservoirs
- Clinical efficacy
  - HBsAg loss and ideally, seroconversion
  - Reversal of liver damage, lack of hepatic inflammation
  - Significant reduction in the risk of future HCC development

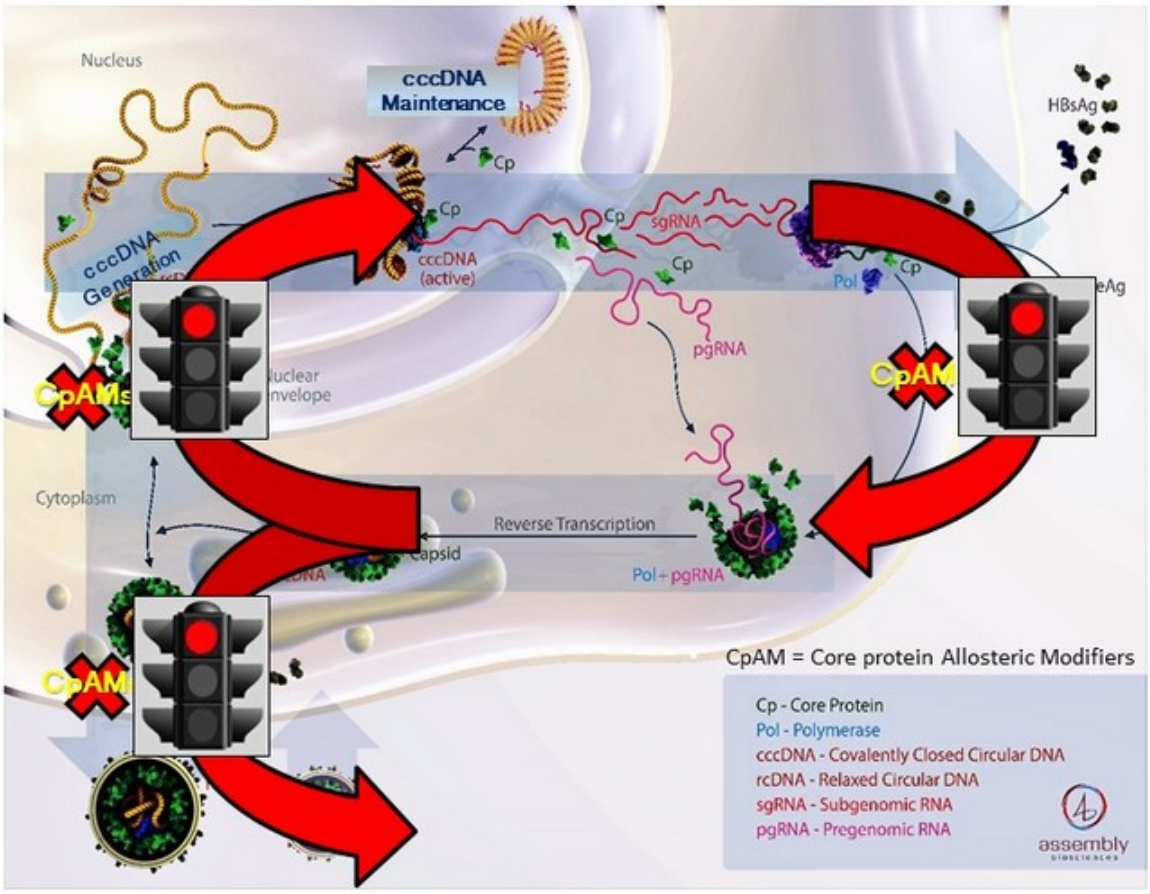
Strategic Approaches Being Pursued for Cure



Assembly Biosciences Focused on Targeting Core Protein to Decrease cccDNA Levels

assembly
biosciences

ASMB Focused on Inhibitors of Core Protein

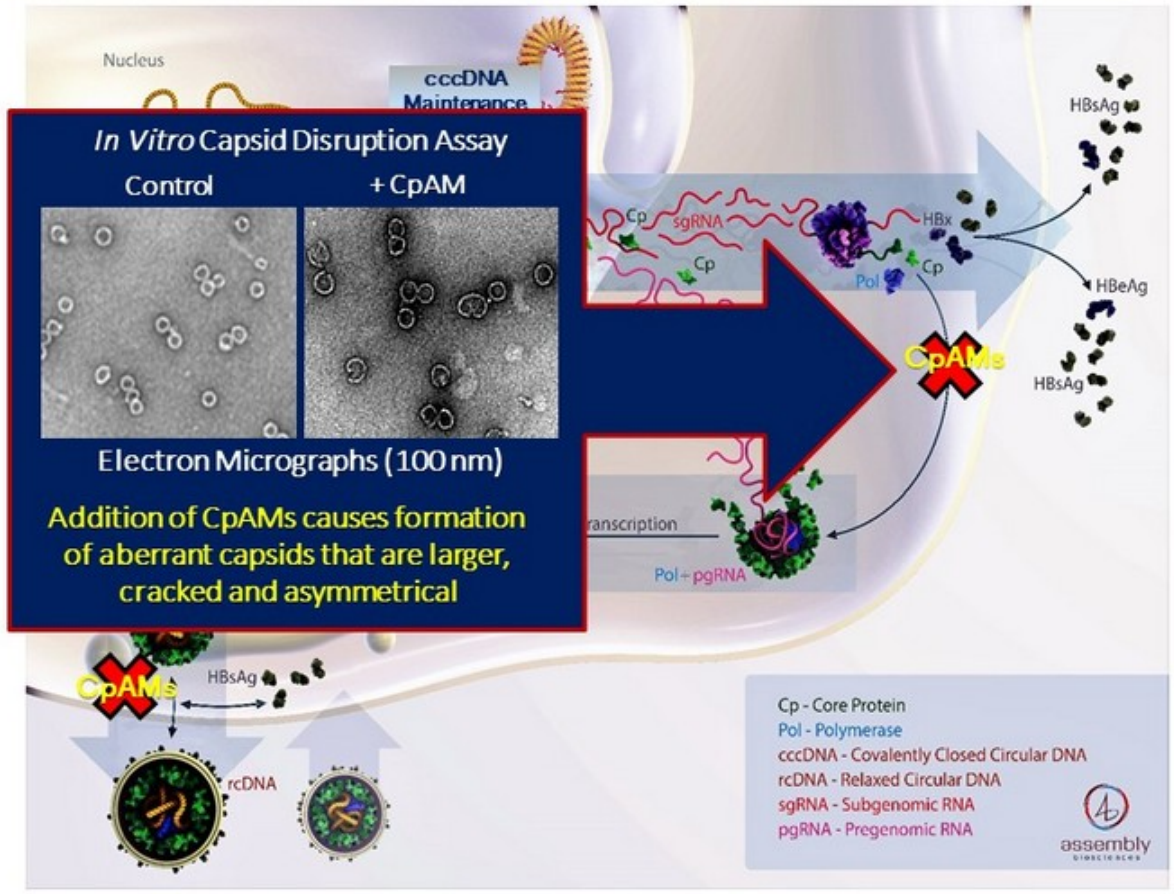


CpAMs Inhibit HBV Life Cycle at Several Steps

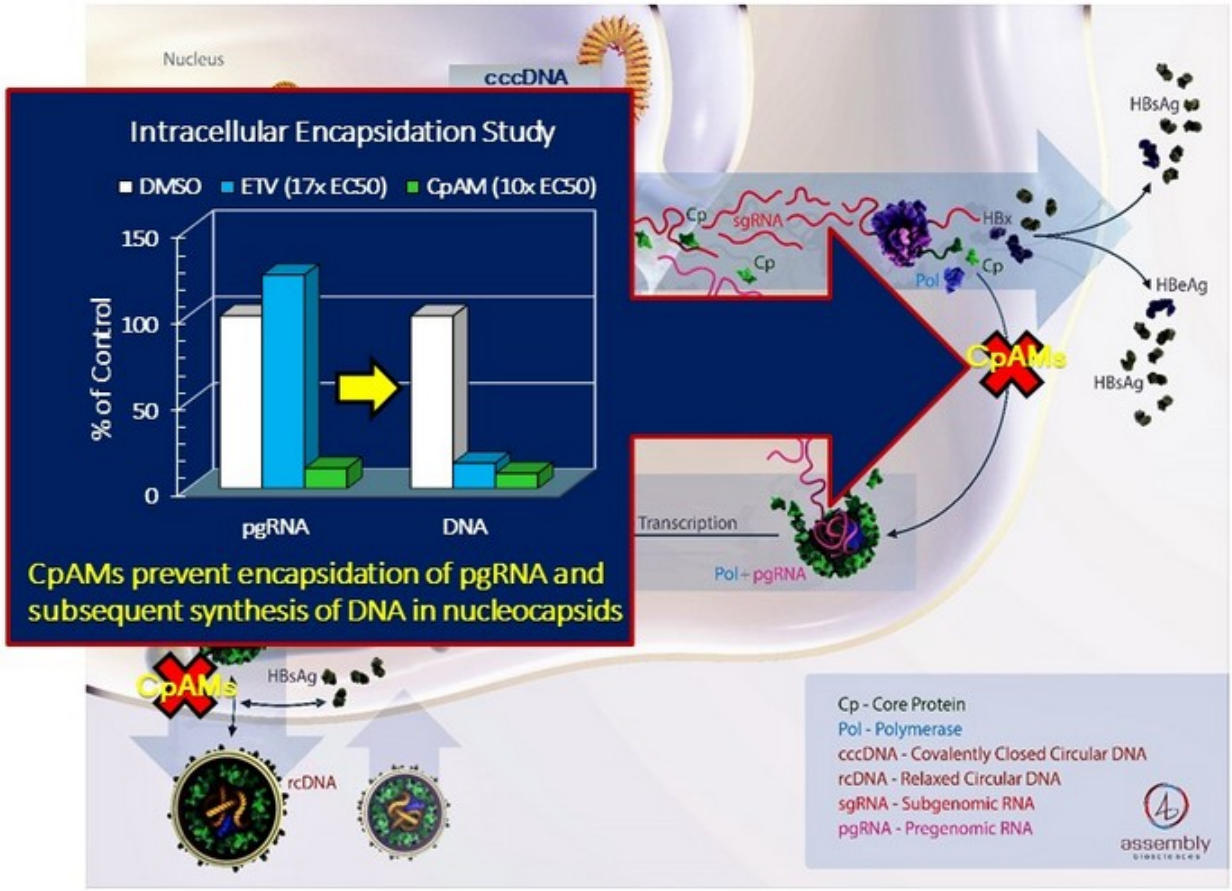
- CpAMs (Core protein Allosteric Modifiers) target Core protein and inhibit key functional steps required for to cccDNA generation
- Bind at dimer-dimer interface and disrupt the orderly folding of Core protein dimers into functional nucleocapsids, generating empty aberrant capsids
- Prevent encapsidation of Pol and pgRNA, a pre-requisite for RT activity and generation of rcDNA
- Prevent maturation of infectious viral particles
- Prevent trafficking of encapsidated rcDNA to nucleus for conversion to cccDNA
- Alter phosphorylation levels of Core protein, and may also lead to Core protein elimination in infected cells
- Ability to disrupt existing nucleocapsids

Because their distinct inhibitory mechanism(s), CpAMs and Nucs together should exhibit enhanced antiviral potency and have the potential to reduce levels of both cccDNA and HBsAg

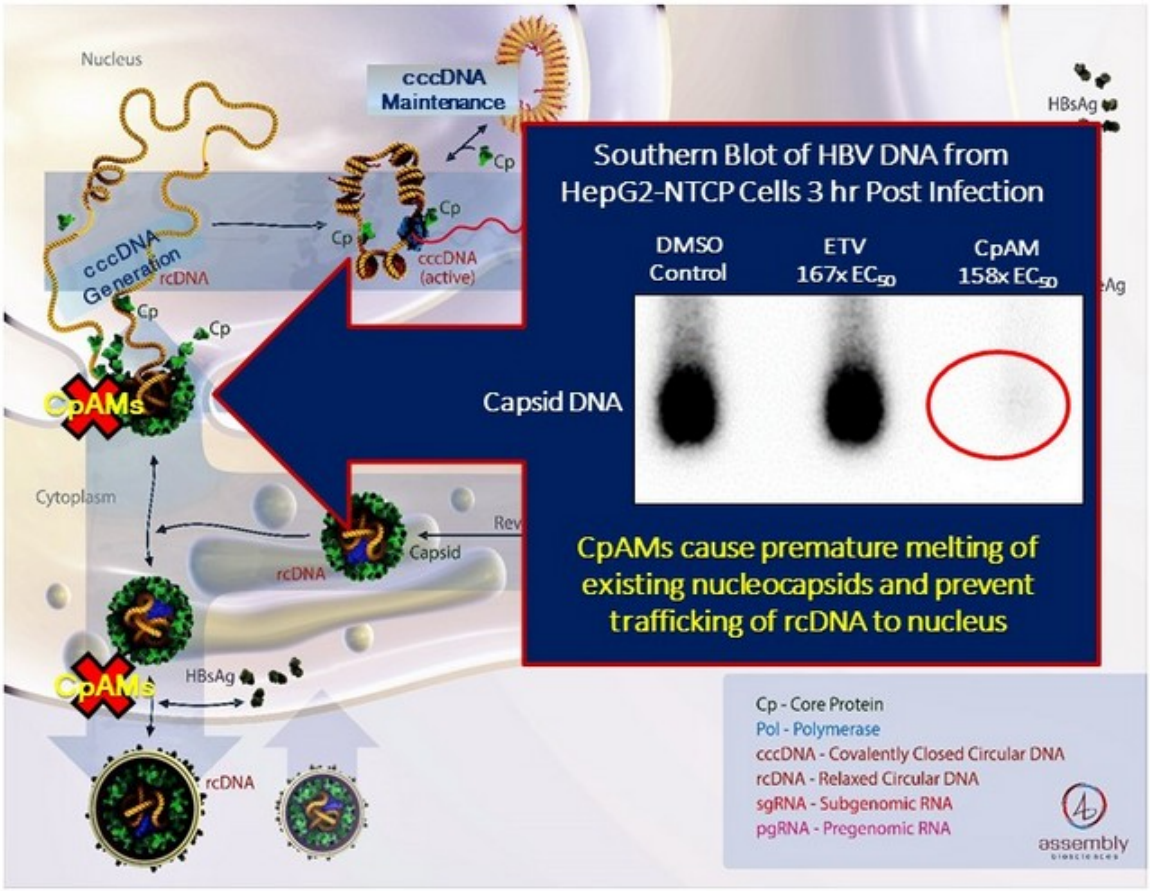
Antiviral Effects of CpAMs



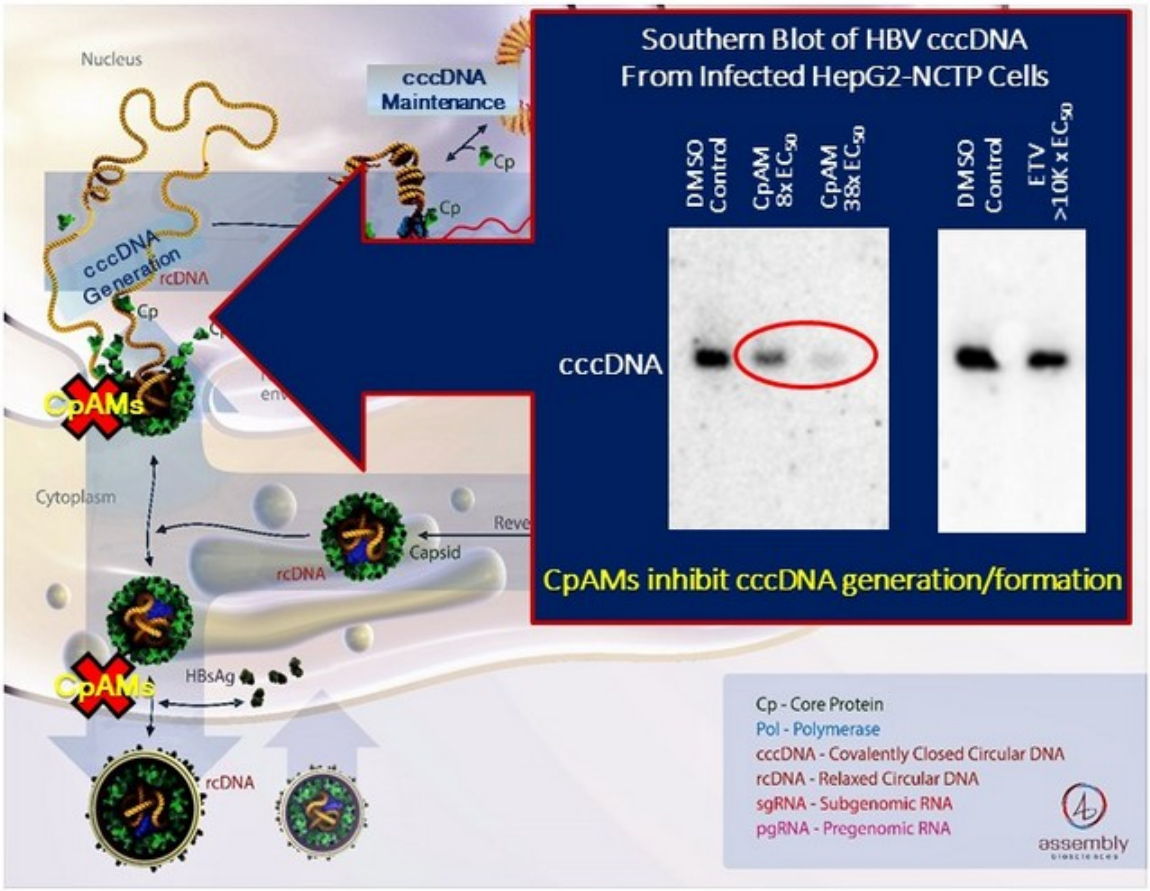
Antiviral Effects of CpAMs



Antiviral Effects of CpAMs

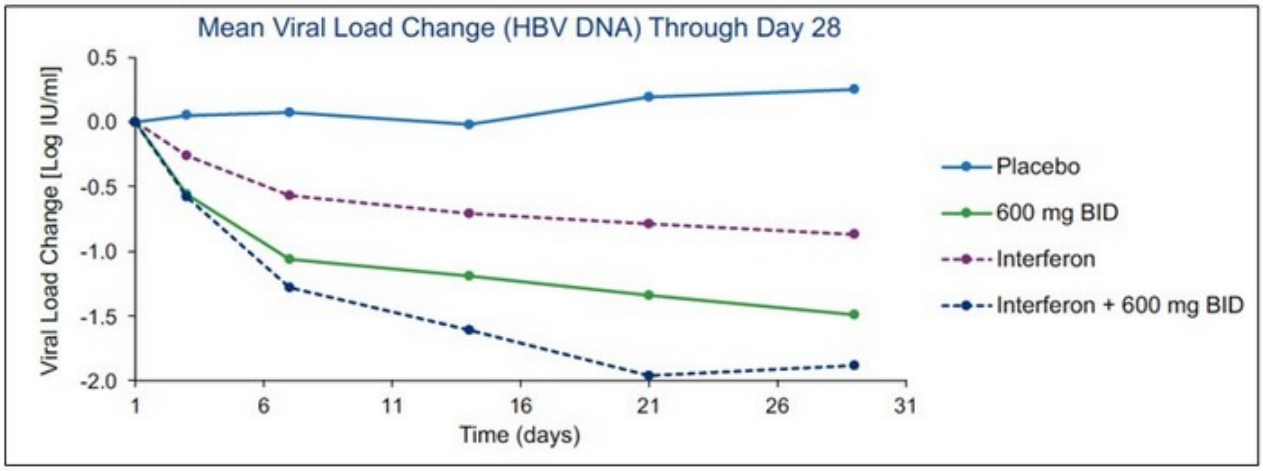


Antiviral Effects of CpAMs



Clinical POC of CpAMs as Effective HBV Antivirals

NVR 3-778 Phase 1b 28-Day Monotherapy Results in HBV-Infected Patients



- Satisfactory safety at all dose levels, no pattern of treatment-related clinical AEs or lab abnormalities
- Oral doses of 600 mg BD in chronic HBV patients resulted in a potent antiviral response in 28 days
  - Mean 1.72 $\log_{10}$ IU/mL HBV DNA reduction
  - Mean 0.86 $\log_{10}$ copies/ml reduction in serum HBV RNA levels

Lawrence Blatt, J&J R&D Day Presentation 6-17

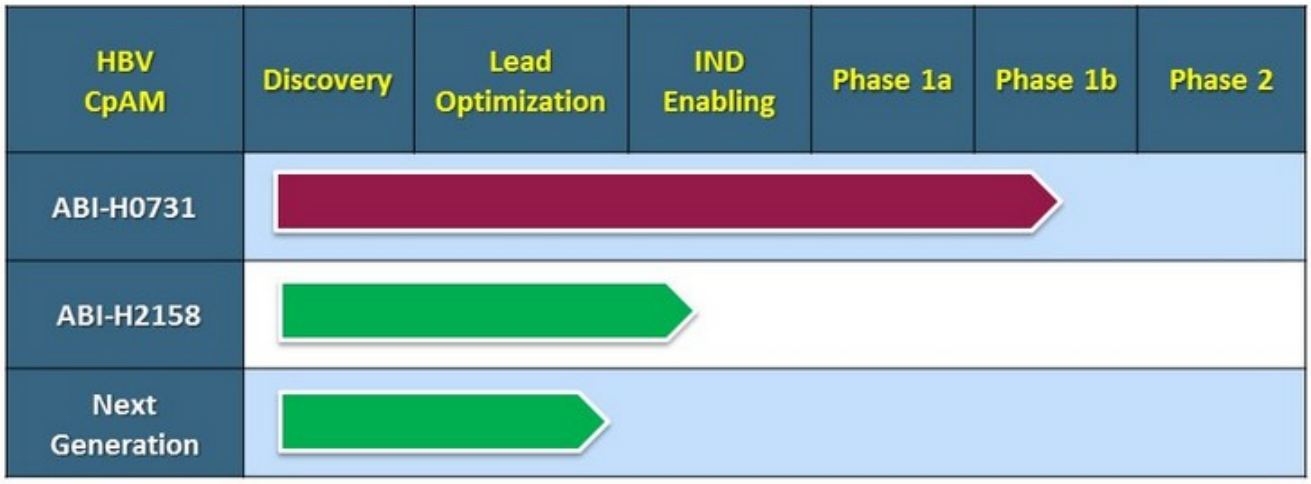
Assembly Biosciences
Establishing A Pipeline of CpAMs

assembly
biosciences

Drugable Properties Will Play an Important Role

- Higher concentrations of CpAMs will be required to decrease cccDNA levels relative to inhibiting viral DNA levels
- Premium will be placed on CpAMs with favorable DMPK properties that are able to achieve and maintain higher effective concentrations in infected hepatocytes
- Convenient oral dosing (frequency and pill burden)
- Metabolic stability in hepatocytes to enable maximal sustained inhibition
- Good PK profile
  - Oral bioavailability, half-life, C_{max} and C_{min}
  - Rapid achievement of steady state
- Controlled and predictable liver:plasma concentrations, minimal accumulation in liver
- Limited drug-drug interactions
- Well tolerated – good safety profile with prolonged dosing

Assembly Pipeline of CpAMs



- ABI-H0731 has completed Phase 1a evaluation, and is currently being studied in HBV patients in a 28-day, double-blind, placebo-controlled Phase 1b study
- ABI-H2158 selected as our next generation CpAM clinical candidate and is currently undergoing IND-enabling studies for initiation of Phase 1a studies
- Plan to identify and select a third CpAM clinical candidate by year end
- All ASMB CpAMs derived from distinct proprietary chemical scaffolds unrelated to previous HAP and Novira-like molecules

ABI-H0731 Preclinical Overview

- Selective targeting of dimer-dimer interface of HBV Core protein, leading to inhibition of cccDNA generation in infected cell assays
- Pangenotypic coverage of HBV genotypes (A – D)
- No significant activity noted against a panel of other viruses or in cytotoxicity assays utilizing a panel of cell types
- Additive to synergistic in combination with Nuc
- Highly favorable DMPK properties
  - High (62-95%) oral bioavailability observed in all animal species tested
  - T½ predictive of human QD dosing
  - Limited accumulation with repeat dosing
  - Enhanced liver concentrations relative to plasma levels, parallel half-lives
  - Highly stable, excretion predominantly as intact compound
- Clean safety profile in a panel of GLP toxicology studies, no organ toxicities identified

ABI-H0731 Clinical Overview

Phase 1a Completed

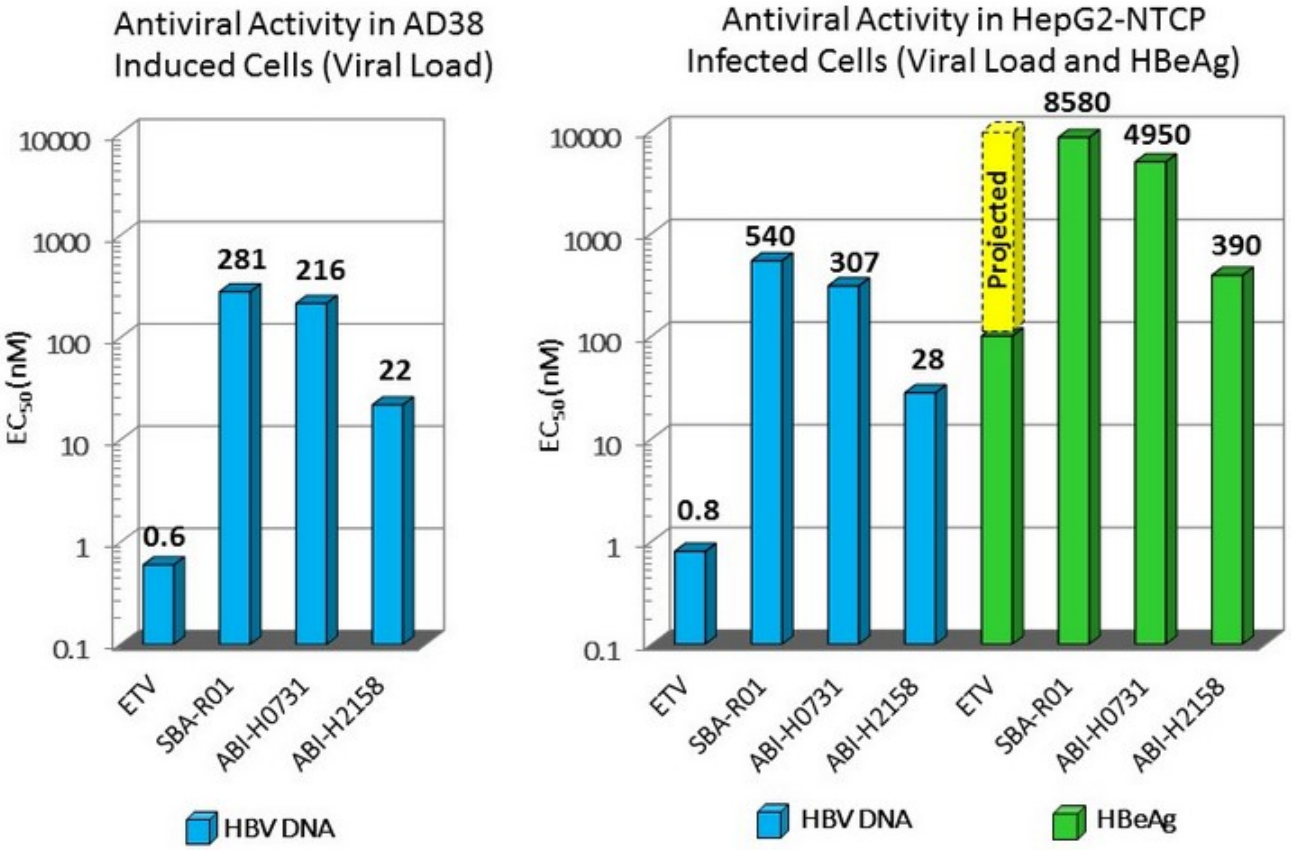
- Single oral doses from 100 to 1,000 mg, multiple doses of 800 mg QD and 800 mg BID x 7 days evaluated
- Favorable PK profile with a half-life consistent with QD dosing
- Well absorbed, with achievement of concentrations believed to be sufficient to suppress viral replication and cccDNA generation
- No SAEs, no clinically significant AEs and no withdrawals due to AEs
- Treatment emergent AEs deemed "possibly related," such as headache and rash, were mild and transient, and only observed at the highest doses
- No clinically significant treatment emergent laboratory abnormalities, vital sign changes or ECG findings

Data to be presented at AASLD, October 2017 in Washington, DC

Phase 1b In Progress

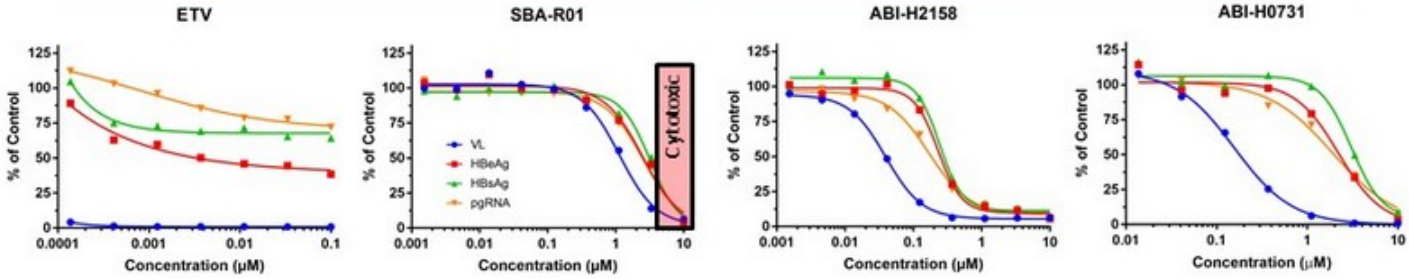
- 28-Day monotherapy dosing in HBV patients

ABI-H2158 - Next Generation CpAM



SBA-R01 is believed to be NVR 3-778 or a very close analog (Pei Y. et al., J. Med. Chem. 2017; 60, 6461–6479)

CpAMs Profiling in Primary Human Hepatocytes



CpAM	HBV DNA EC_{50} (nM)	HBeAg EC_{50} (nM)	HBsAg EC_{50} (nM)	pgRNA EC_{50} (nM)
ETV	<0.1	Est >100	Est >100	Est >100
SBA-R01	1,130	2,502	3,192	2,554
ABI-H0731	154	2,210	3,000	1,840
ABI-H2158	39	230	242	169

- CpAMs reduced viral HBV DNA levels and known surrogate markers for cccDNA (HBeAg, HBsAg and pgRNA)
- ETV was highly effective at inhibiting HBV DNA levels, but exhibited limited effect on cccDNA surrogate markers

Data to be presented at AASLD, October 2017 in Washington, DC

Profile of ASMB Clinical Candidates

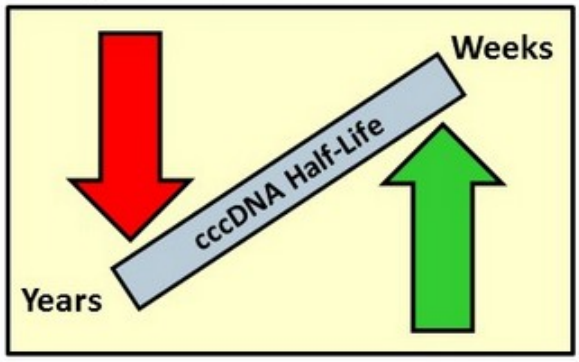
Virology Parameters	SBA-R01	ABI-H0731	ABI-H2158
AD38 VL EC_{50} (nM)	281	170	14
HC9AT HBeAg EC_{50} (nM)	8,580	4,950	390
PHH VL EC_{50} (nM)	1,130	150	23
PHH HBeAg EC_{50} (nM)	2,502	2,210	230
DMPK Parameters			
Human Liver Microsomes (% remaining at 45 min)	100	87	91
CYP Profile (IC_{50})	All > 10 µM	All >10 µM	All ≥10 µM
Protein Binding (%)	98	97	97
Rat PK (1 mg/kg) %F $T_{1/2}$ (hr) C_{max} (ng/mL) Oral AUC_{last} (hr*ng/mL)		95 6.1 162 1,470	50 2.9 536 3,671

- Unclear what potency/exposure levels are required to suppress cccDNA generation
- Liver concentrations of ABI-H0731 projected to be ~25x plasma concentrations
- If needed, ABI-H2158 has superior antiviral potency to ABI-H0731, while maintaining favorable DMPK properties

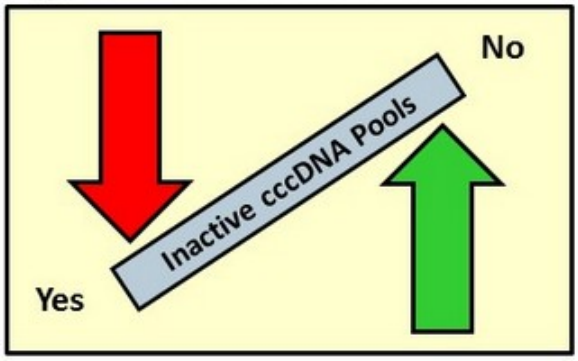
Will CpAM Treatment Result in Higher Cure Rates?

Potential Challenges

Long Intracellular cccDNA Half-life



Significant Pools of Inactive cccDNA



Studies underway using patient samples to gain insight into both topics

- Lamivudine (LVD) and Telbivudine (LdT) treatment results in rapid emergence of resistant variants due to their low barrier to resistance
- LVD/LdT resistance maps to L180M and M204I substitutions in the Pol gene
- Used these genetic markers to monitor the turnover of cccDNA, pgRNA and viral DNA in longitudinal patient samples

cccDNA Studies – Initial Results

- All biological molecules have a half-life, including cccDNA
- Longitudinal study conducted on samples (serum and biopsies) from patients with emerging resistance to LVD and TBV
- Appearance and enrichment of resistant mutations used as a genetic marker in monitoring populations of viral DNA, pgRNA and cccDNA
- Results demonstrated rapid establishment of newly formed cccDNA pools harboring Nuc-resistant mutations
- Significant turnover of wt pgRNA molecules within months suggests that existing cccDNA may decay faster than previously predicted in the absence of any gross inflammation
- Little evidence for the existence and maintenance of substantial pools of inactive wt cccDNA in patient samples

Data to be presented at AASLD, October 2017 in Washington, DC

CpAM Summary

- Mechanism-based studies demonstrated that CpAMs bind to Core protein and disrupt viral replication at multiple steps
- Importantly, and distinct from Nucs, CpAMs appear to block the generation of new cccDNA molecules!
- ASMB's CpAM pipeline consists of candidate compounds selected and optimized from distinct and proprietary chemical series
- While the precise level of intrinsic potency needed for cccDNA inhibition in patients is yet to be established, emphasis has been placed on increasing potency while maintaining favorable DMPK properties
- Lead candidate ABI-H0731 has completed Phase 1a with favorable safety and PK properties predictive of QD dosing in patients, and is currently undergoing evaluation in chronically-infected patients (Phase 1b)
- Second generation candidate ABI-H2158 exhibits enhanced potency while retaining the favorable DMPK properties of ABI-H0731
- Future combinations of CpAMs and Nucs should result in enhanced antiviral activity, have a high resistance barrier and most importantly, decrease cccDNA levels

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