
SPRING BANK
Driven by a novel pharmaceutical
platform focused on
SELECTIVE IMMUNOMODULATION
spring bank
pharmaceuticals

Forward Looking Statements

This presentation includes forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995. Forward-looking statements include, among other things, statements, other than historical facts, regarding: the progress, scope, duration or results of clinical trials and preclinical studies of inarigivir soproxil ("inarigivir"), SB 9225, SB 11285 or any of our other product candidates or programs, such as the size, design, population, conduct, cost, objective or endpoints of any clinical trial, or the timing for initiation or completion of or availability of results from any clinical trial (including our Phase 2 clinical trial of inarigivir in patients with chronic Hepatitis B virus); the potential benefits that may be derived from any of our product candidates; our future operations, financial position, revenues, costs, expenses, uses of cash, capital requirements or our need for additional financing; or our strategies, goals, milestones, prospects, beliefs, intentions, plans, expectations, forecasts or objectives. Words such as "anticipates," "believes," "plans," "expects," "projects," "future," "intends," "may," "will," "should," "could," "estimates," "predicts," "potential," "continue," "guidance," and similar expressions sometimes identify forward-looking statements. Any forward-looking statement involves known and unknown risks, uncertainties and other factors that may cause our actual results, levels of activity, performance or achievements to differ materially from those expressed or implied by such forward-looking statement, and, therefore, you are cautioned not to place undue reliance on any forward-looking statement. These factors include, but are not limited to: whether our cash resources will be sufficient to fund our continuing operations for the period anticipated; the components, timing, costs and results of our clinical trials, preclinical studies and other development activities involving our product candidates; whether certain top-line results from our clinical trials materially change as more information becomes available; whether results obtained in preclinical studies and clinical trials will be indicative of results obtained in future clinical trials; whether inarigivir, SB 9225, SB 11285 and any of our other product candidates will advance through the clinical trial process on a timely basis and receive approval from the United States Food and Drug Administration or equivalent foreign regulatory agencies; and whether, if inarigivir, SB 9225, SB 11285 or any of our other product candidates obtain regulatory approval, it will be successfully distributed and marketed. These and other risks and uncertainties that we face are described in our most recent Annual Report on Form 10-K, filed with the Securities and Exchange Commission (SEC) on March 11, 2019, and in other filings that we make with the SEC from time to time.

All forward-looking statements speak only as of March 11, 2019 and should not be relied upon as representing our views as of any other date. We specifically disclaim any obligation to update any forward-looking statement, except as required by applicable law. All trademarks, service marks, trade names, logos and brand names identified in this presentation are the properties of their respective owners.

This presentation also contains estimates and other statistical data generated by independent parties and by us relating to market size and statistics. These estimates involve a number of assumptions and limitations, and you are cautioned not to give undue weight to such estimates.

Spring Bank (NASDAQ: SBPH) Investment Opportunity

- **Expanding into the broadest clinical development program in HBV in 2019**
  - Recently-issued FDA Draft Guidance provides a clear development pathway opportunity for inarigivir & SB 9225
- **SB 9225 (inarigivir + tenofovir disoproxil fumarate) progressing towards Phase III initiation in 2020**
- **Gilead clinical collaboration currently in Phase 2 with multiple cohorts**
  - Inarigivir is part of an expanding HBV development strategy with Gilead
- **SB 11285 on track to be the first IV administered STING agonist to enter the clinic in 2019**
- **Currently funded into Q2 2021 with over $64M* and numerous data readouts in the next 12–18 months**

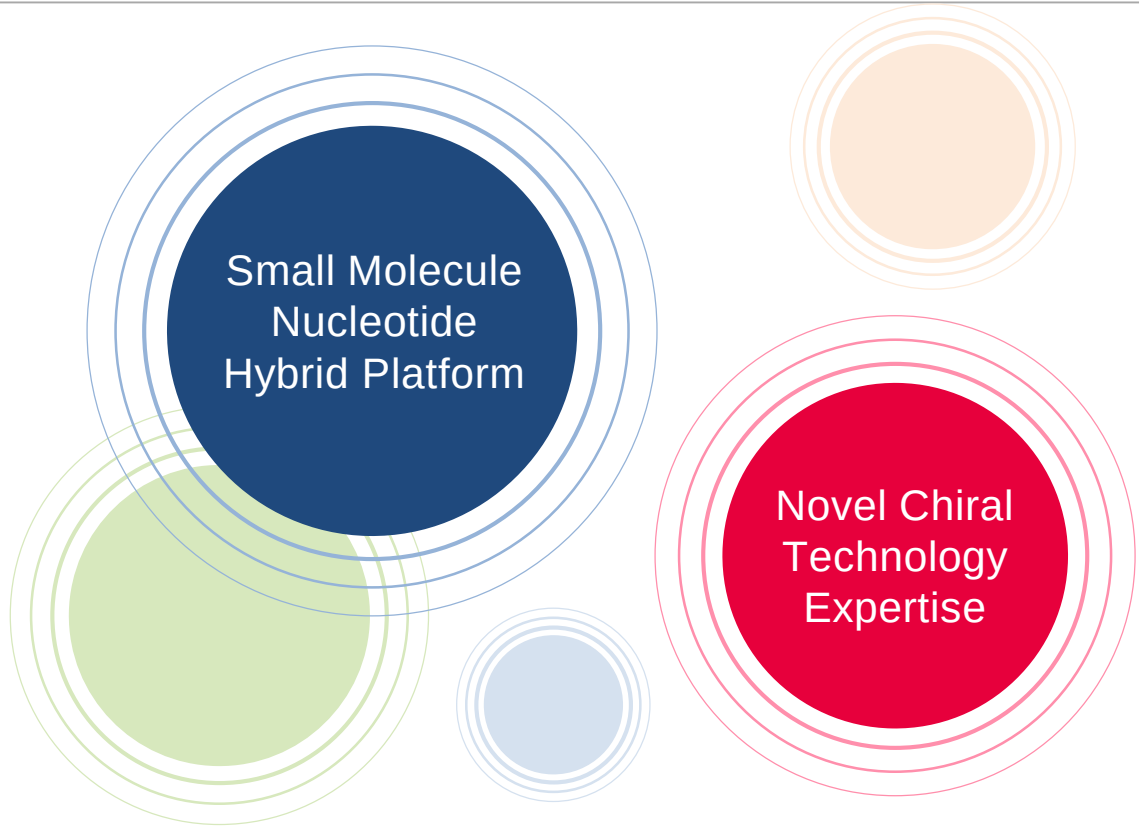
*As of December 31, 2018.

SBPH Corporate Focus and Strategy

Platform focused on immunomodulatory targets based on endogenous nucleotide ligands

Rapid evolution of nucleotide scaffolds into development candidate small molecule nucleotide hybrids

Reduces discovery cycle and potentially shortens development time



Differentiated Pipeline Designed To Meet Significant Unmet Needs

Therapeutic Areas	Compound	Discovery/Lead Optimization	Preclinical	Phase 1	Phase 2	Phase 3
HBV	**Inarigivir**					
	Monotherapy					
	Co-Administration with Gilead's Vemlidy®		*Funded by Gilead*			
	Co-Administration with NUCs					
	SB 9225 (inarigivir + tenofovir disoproxil fumarate) fixed-dose combination					
	HBx-gene antagonist					
Cancers	**Second-Generation STING Agonists**					
	SB 11285 (intravenous, intratumoral)					
	Multiple analogs (nanoparticle, neoadjuvant and ADC development)					
	RIG-I agonist					
Inflammatory Diseases	**STING antagonist**					

Vemlidy®: tenofovir alafenamide (TAF) 25mg.

Chronic HBV: A Global Problem

Chronic HBV infection prevalence (approximate)

~257MM

GLOBALLY

~17MM

US AND EUROPE

Estimated Deaths

~900,000

(IN 2017)

HBV NUC global therapeutics sales

$2.1B

(IN 2017)

LOW TREATMENT RATES OF CHRONIC HBV PATIENTS

- Estimated 10-15% in the US and Europe
- Estimated 1% in the Asia-Pacific region

LOW FUNCTIONAL CURE RATES WITH CURRENT STANDARD OF CARE

*Approximate.

An Opportunity to Change the Chronic HBV Paradigm

Elevating the Functional Cure Rate

A meaningful new therapy will need:

1. A good safety & tolerability profile
2. Ease of administration
3. ACHIEVE finite course of treatment leading to functional cure rates >8-10%*

- HBV is complex and heterogenous
- Combinatorial approach will be required
- Immunomodulation will need to be the backbone

"Combination of antiviral and immune modulatory therapies will likely be needed to achieve functional hepatitis B virus cure."

– Lok A, Zoulim F, Dusheiko G, Ghany MG. Hepatitis B cure: from discovery to regulatory approval. *J Hepatol.* 2017;67:847-861.

*Represents the approximate number of patients that achieved HBsAg clearance after 48 weeks of treatment with αIFN + Viread® HBsAg, hepatitis B surface antigen; HBV, hepatitis B virus; IFN, interferon.

Inarigivir

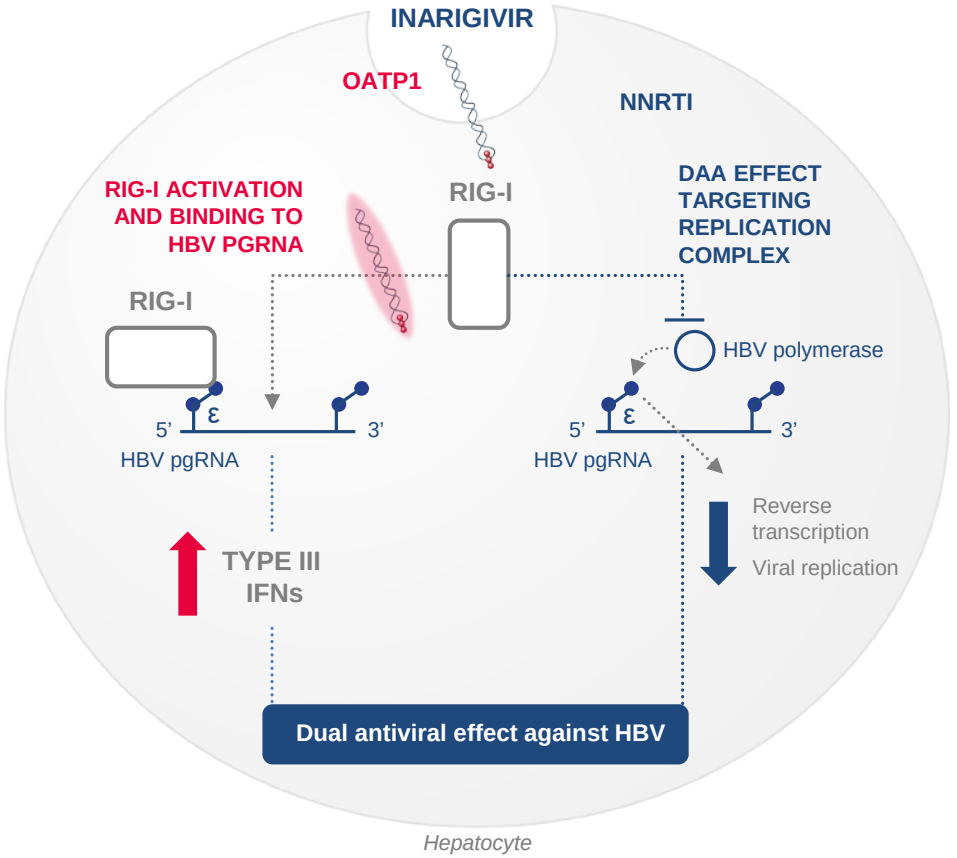
A Novel, Oral, Selective Immunomodulator With a Dual Mechanism of Action

- Potent RIG-I agonist
- Orally bioavailable, liver-selective immunomodulator
- Demonstrated dose response in HBV DNA and HBV RNA
- Only oral drug to show significant HBsAg reduction
- Favorable safety profile in over 100 subjects to date with no SAEs
- Deep collaboration with Gilead for co-administration studies with “NUCs”

Inarigivir: A Novel and Oral Selective Immunomodulator with a Dual Mechanism of Action

INARIGIVIR is a RIG–I AGONIST which is designed to:

- **Restore hepatic selective innate and adaptive immune response** stimulating the production of type I and III IFNs
- Inhibit the HBV replication complex via a direct acting anti-viral effect
- Result in significant anti-HBV activity with reduction in HBV DNA, HBV RNA, HBsAg and cccDNA



Inarigivir: Selective Hepatic Immunomodulation and Direct Acting Antiviral Activity

PRE-CLINICAL STUDIES

- Host mediated pan antiviral agent against RNA viruses
- Inarigivir binds to CARDs and regulatory domain of RIG-I with activation of IRF-3 and hepato-selective innate immune response
- Inarigivir effective against NUC- and capsid-resistant HBV variants
- Inarigivir up-regulates intra-hepatic RIG-I, activates intra-hepatic ISGs and suppresses HBsAg,
  HBV DNA, HBV RNA, and cccDNA in the woodchuck model
- Inarigivir acts as a non-nucleotide reverse transcriptase inhibitor (NNRTI) within the nucleocapsid replication complex

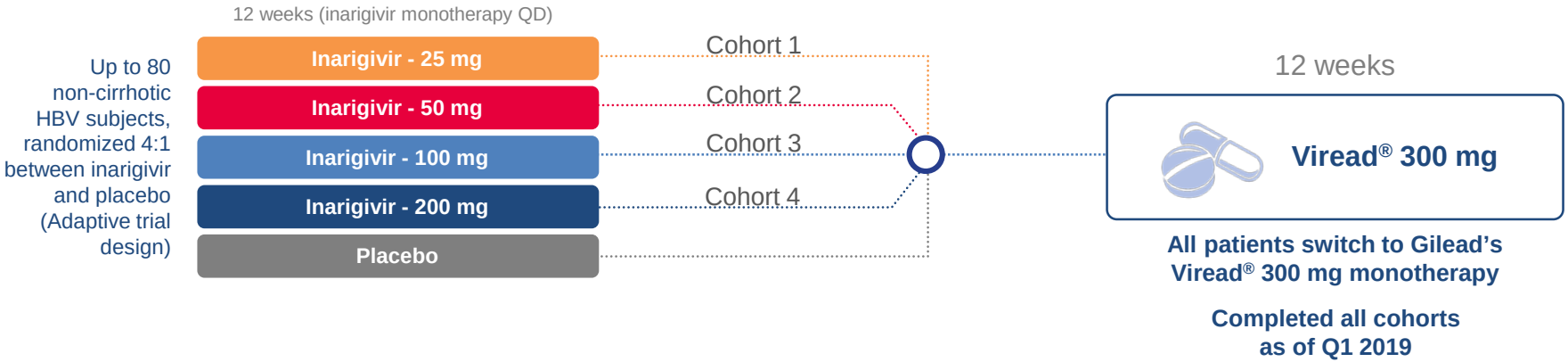
Inarigivir: Selective Hepatic Immunomodulation and Direct Acting Antiviral Activity (Cont.)

CLINICAL STUDIES

- Inarigivir demonstrates potent antiviral activity against HCV with response proportional to ISG activation and IL-28b status
- Preliminary data shows inarigivir responses in HBV associated with activation of ISGs in PBMCs
- Inarigivir activates B-cell neutralizing HBsAb response in responder patients

ACHIEVE Phase 2 Dose Escalation Study

Clinical trial collaboration with Gilead to evaluate inarigivir followed by Viread® 300 mg



PRIMARY ENDPOINT

Safety and antiviral activity at 12 weeks

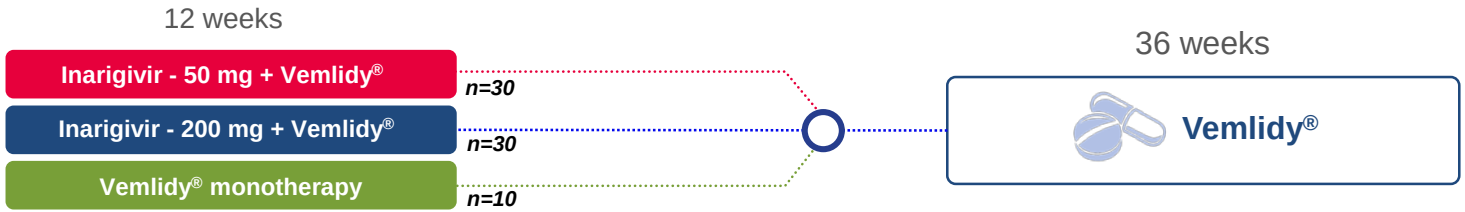
SECONDARY ENDPOINT

PK, change in serum HBV DNA, HBsAg, HBV RNA and HBeAg from baseline to weeks 6, 12, 14, 16, and 24

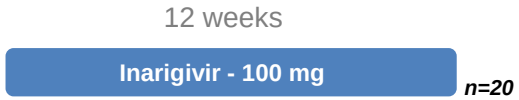
Global HBV Clinical Collaboration With Gilead

Expanded Gilead Phase 2 HBV Study

Inarigivir co-administered with Vemlidy® (tenofovir alafenamide) 25 mg in ***naïve patients***



Inarigivir monotherapy in ***virally suppressed patients***



Patients remain on current oral antiviral therapy

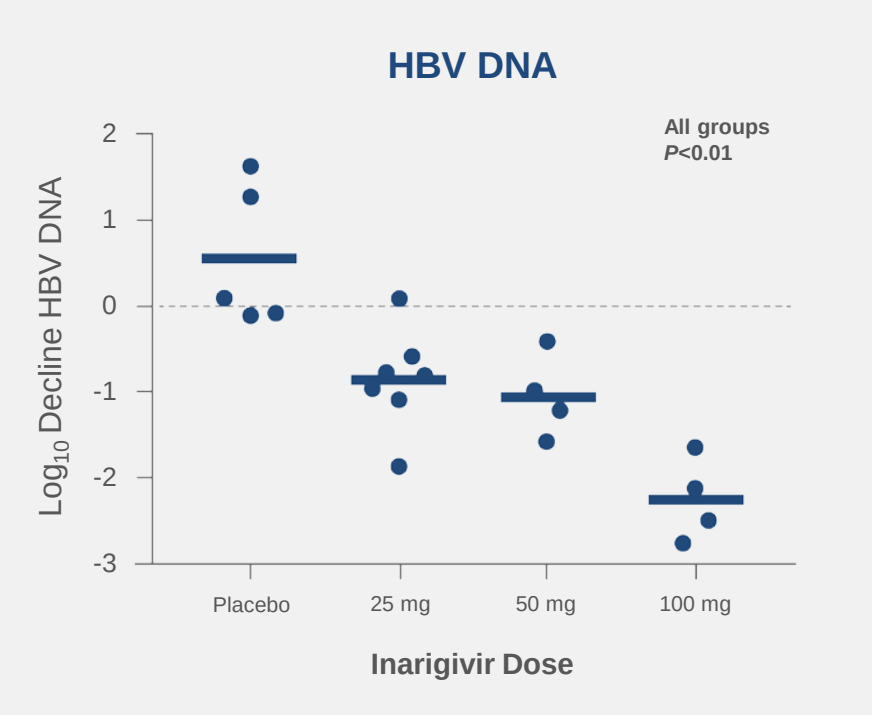
Initiated by Gilead in 1H 2018
Executed and funded by Gilead

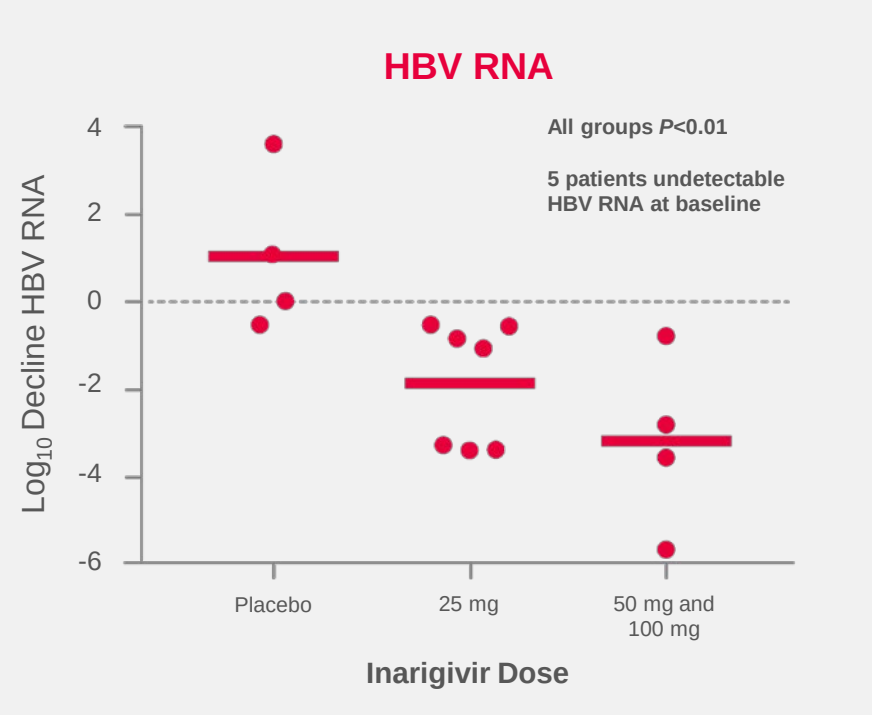
ACHIEVE Trial: Representative Demographics of Global "Real World" HBV Patient Population

			Cohort 1		Cohort 2		Cohort 3	
		Placebo	25 mg HBeAg +ve	25 mg HBeAg -ve	50 mg HBeAg +ve	50 mg HBeAg -ve	100 mg HBeAg +ve	100 mg HBeAg -ve
Number		11	9	7	11	5	13	4
Mean Age		40	37	43	36	47	34	46
Gender M:F		7:4	5:5	3:3	9:2	5:0	7:6	3:1
Mean Baseline ALT		69	82	75	75	65	75	90
Mean Baseline HBV DNA log_{10}		6.20	7.86	5.69	7.79	4.55	8.20	5.95
Genotype	A	1		1				
	B	6	4	3	3	3	4	3
	C	4	5	1	7	1	8	1
	D			2	1		1	

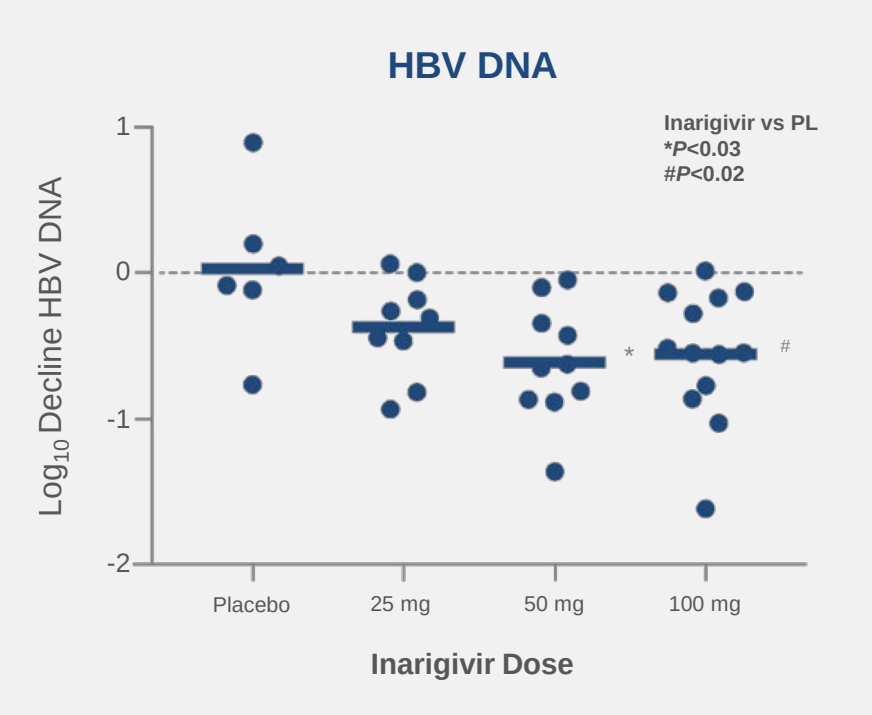
In cohort 2 (50mg), two patients (1 HbeAg +ve and 1 HBeAg –ve) withdrew at day 1 and day 14 from patient choice

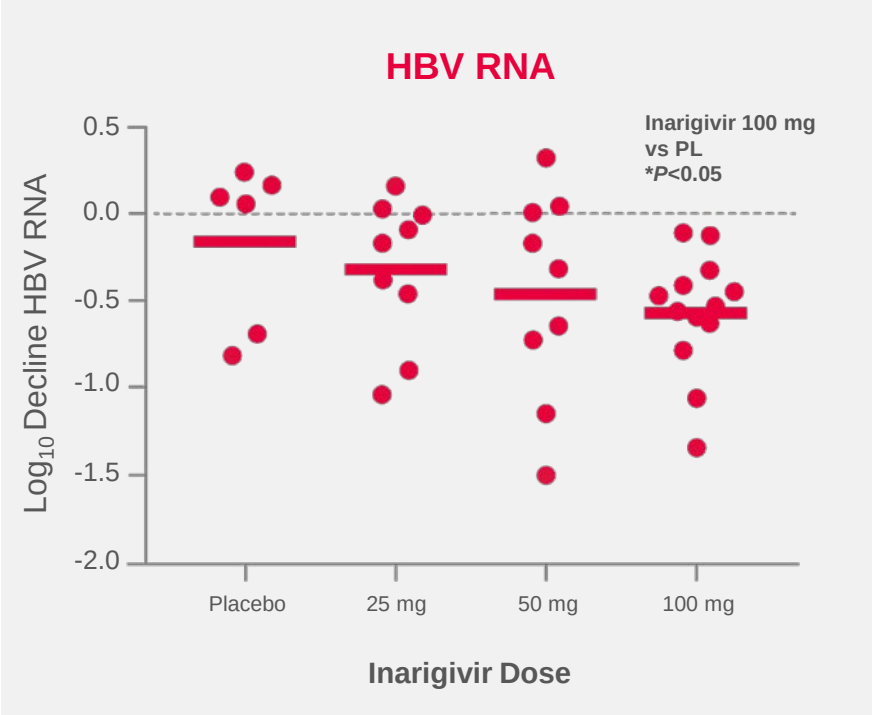
Inarigivir Demonstrates a Continuing Positive Dose Response in HBeAg -ve Patients at Week 12





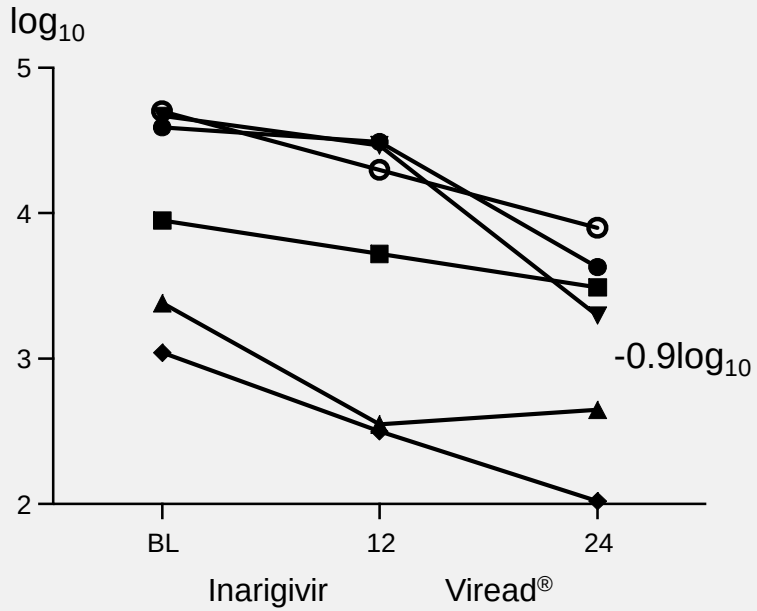
Inarigivir Demonstrates a Continuing Positive Dose Response in HBeAg +ve Patients at Week 12



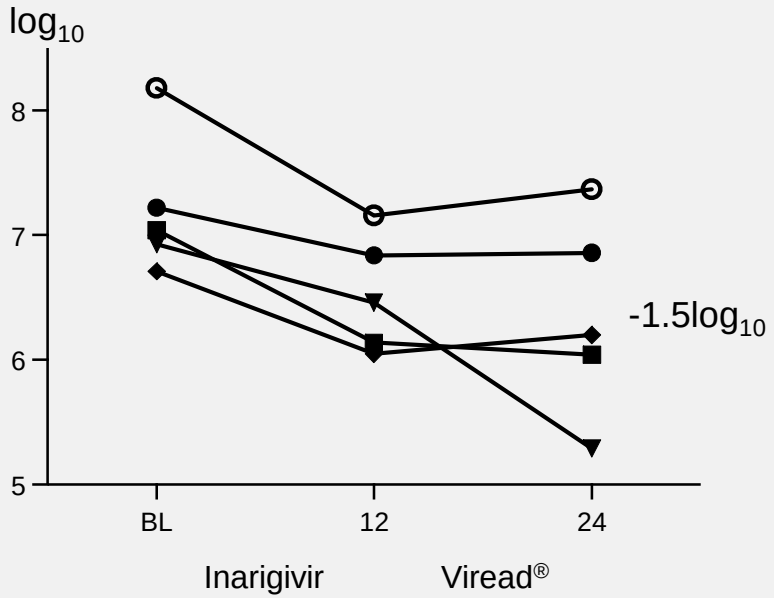


Inarigivir Targets cccDNA in HBeAg +ve Patients

HBsAg decline in HBeAg +ve responders



HBV RNA decline in HBeAg +ve responders

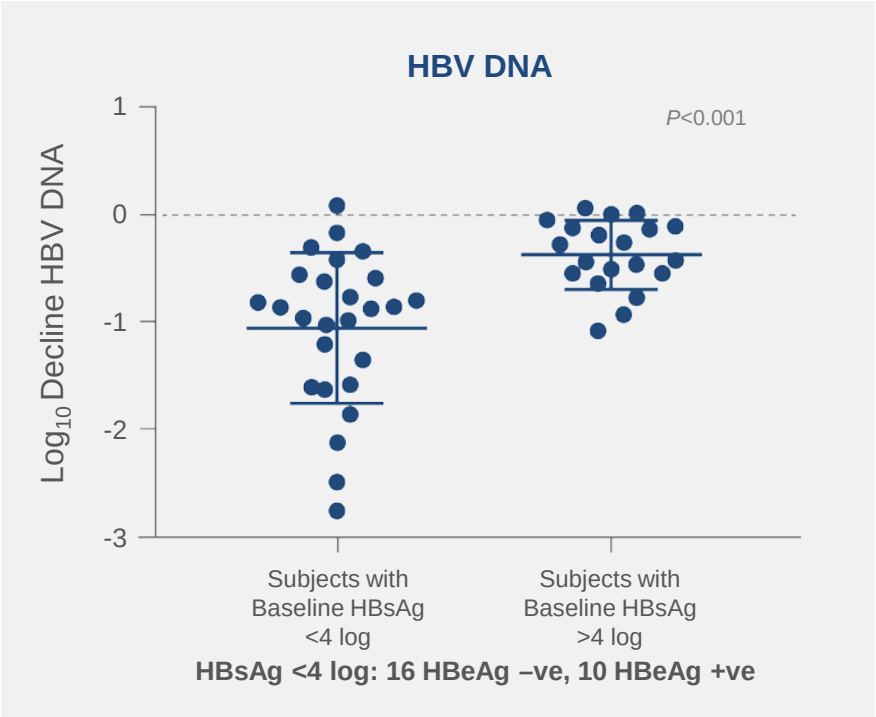


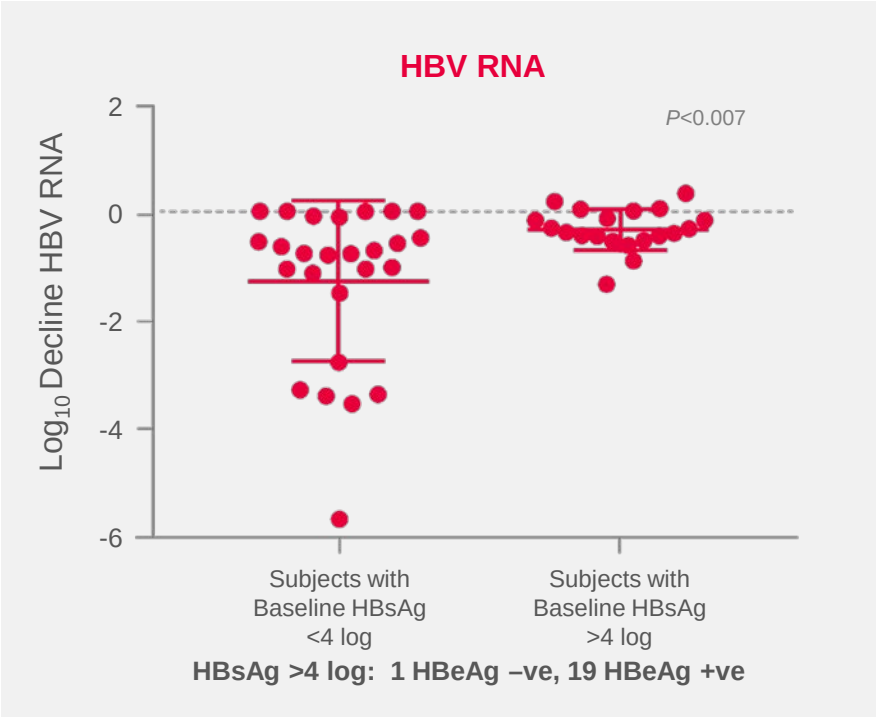
Summary of ACHIEVE Phase 2 Data on Quantitative HBsAg (Cohorts 1-3)

13 of 47 (28%) patients experienced 0.5 log_{10} reduction on inarigivir alone or at 24 weeks after Viread® switch

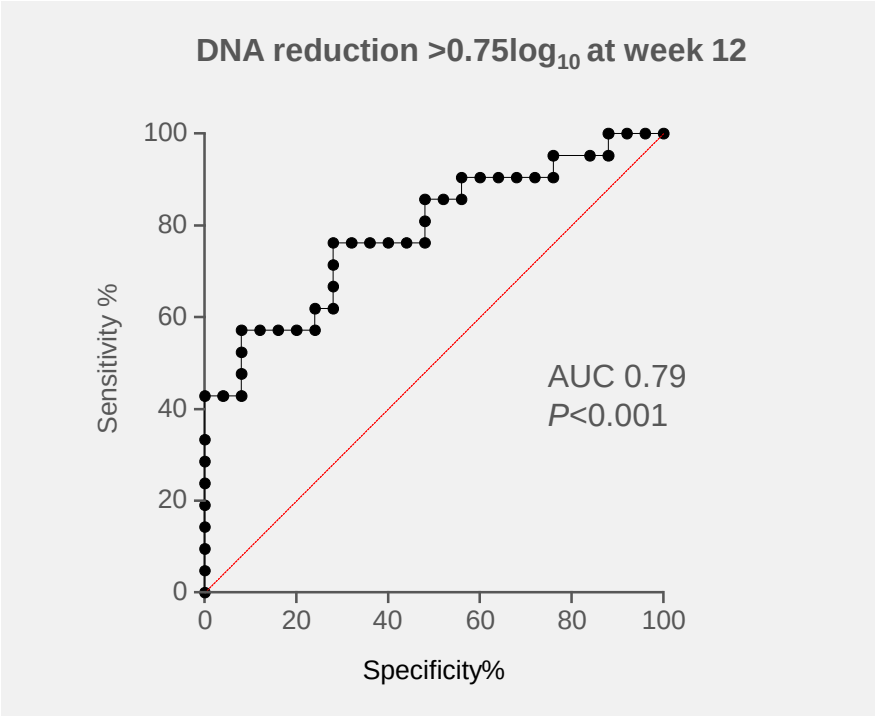
- Mean and median HBsAg reduction 0.8 log_{10} (range 0.5 – 1.4 log_{10}) in 13 responders
- Effect on HBsAg seen at all doses in both monotherapy and after Viread® switch
- HBsAg response seen in 6 HBeAg -ve and 7 HBeAg +ve patients across all genotypes
- HBsAg response associated with declines in HBV DNA and HBV RNA
- HBsAg reduction can be associated with “immune flares” in HBeAg –ve patients on inarigivir monotherapy

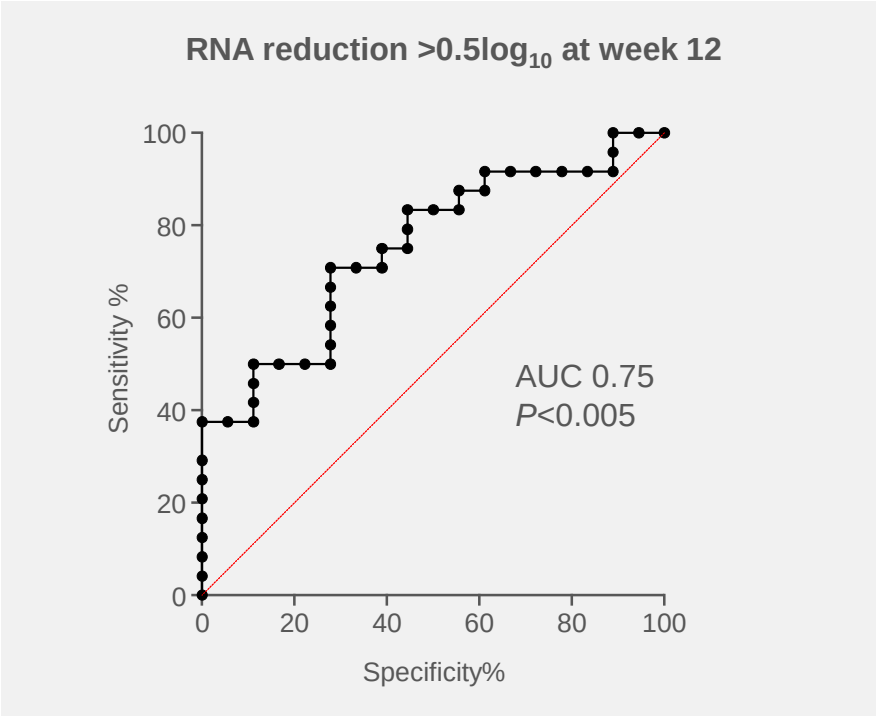
All 3 ACHIEVE Cohorts: Baseline HBsAg Predicts Response of Both HBV DNA & HBV RNA to Inarigivir





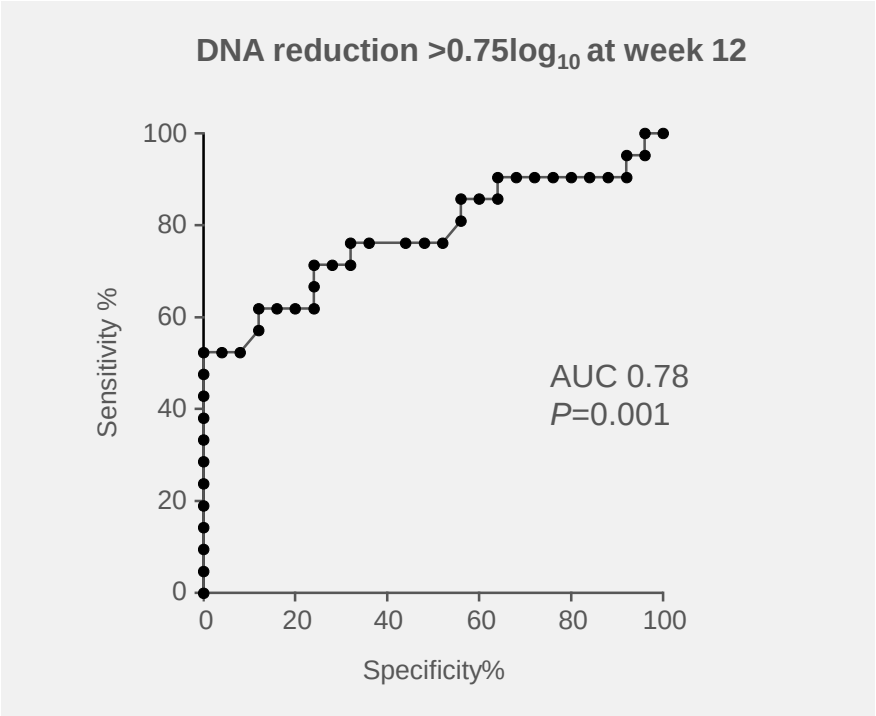
ACHIEVE Trial: Baseline Serum IP-10 Predictor of HBV DNA & HBV RNA Reduction by Inarigivir

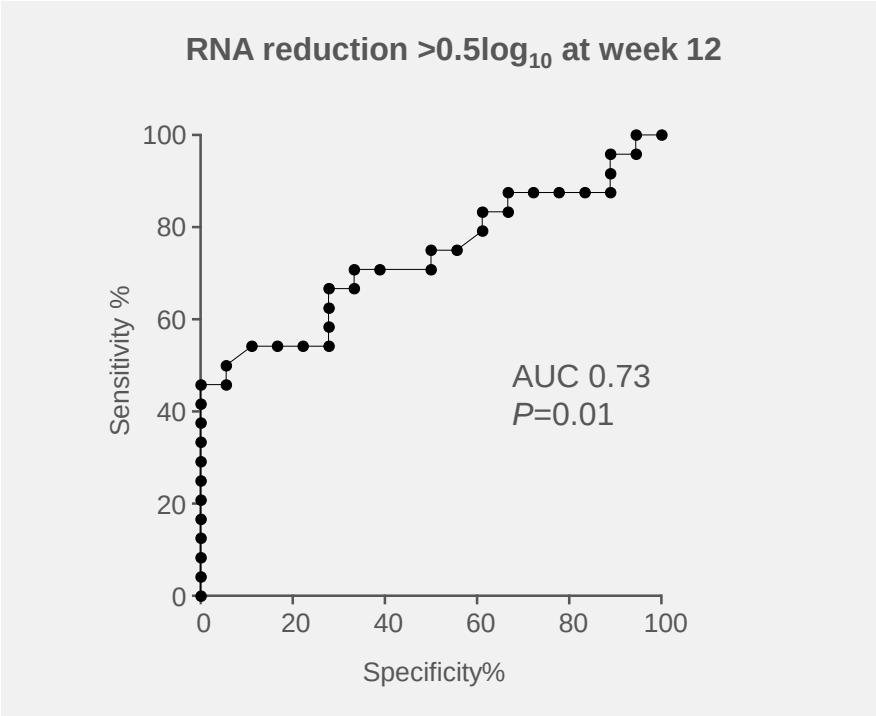




IP-10 >350ng/L, Sensitivity 60%, Specificity 80%

ACHIEVE Trial: Reduction in Serum IP-10 at Week 12 Predictor of HBV DNA & HBV RNA Response





IP-10 decline >110ng/L, Sensitivity 60%, Specificity 88%

Effect of Oral Inarigivir (25, 50, and 100mg) on HBsAg

Only oral drug candidate to show meaningful effect on HBsAg in clinical studies

Effect on HBsAg seen at all doses in both monotherapy and after TDF switch

Overall, 13 of 47 (28%) patients experienced a 0.5 log_{10} reduction on inarigivir alone or at 24 weeks after TDF switch

HBsAg response seen in 6 HBeAg -ve and 7 HBeAg +ve patients across all genotypes

Mean HBsAg reduction 0.8 log_{10} (range 0.5 – 1.4 log_{10}) in 13 responder patients

Effect of Oral Approved or Investigational HBV Drugs on HBsAg*

Inarigivir

Week 12 or 24 mean reduction: 0.8 $\log_{10}$ in responder population

- No other oral drug has shown this level of decline

13 of 47 (28%) patients achieved at least a 0.5 $\log_{10}$ reduction, superior to any other oral HBV drug in development

TLR-7 (Vesatolimod 4 mg)

Week 12 mean reduction:
0.05 $\log_{10}$

No patient >0.5 $\log_{10}$ reduction

TDF / TAF

Week 48 mean reduction:
HBeAg +ve 0.3 $\log_{10}$
HBeAg –ve 0.017 $\log_{10}$

<1% HBsAg loss

cPAM/CAPSID

No effect reported on HBsAg at week 4

*Comparisons are not based on head-to-head studies and therefore conclusions should not be drawn about comparative effect.

ACHIEVE Trial Safety Profile

- ALT flares >200 IU/ml seen in 6 patients on inarigivir and 3 patients on placebo
- Inarigivir-related flares associated with reduction in HBV DNA and HBsAg occurred within 4 weeks of treatment
- No changes in bilirubin, INR or albumin seen with flares
- Per protocol dose reduction in 6 patients, 1 dose discontinuation for ALT >400 IU/ml
- 1 Grade 3 transient hypertriglyceridemia not sustained on retesting
- 1 hospitalization for knee pain, likely unrelated to inarigivir (cohort 4)
- No flu-like symptoms and no other interferon-like side effects to date

Highlights of ACHIEVE Trial to Date

- 28% of patients met pre-defined responder criteria with a mean / median HBsAg decline 0.8$\log_{10}$
- Dose dependent response from 25mg to 100mg inarigivir monotherapy on HBV DNA and HBV RNA
- Response predicted by HBsAg baseline levels and IP-10 in both HBeAg +ve and HBeAg -ve patients
- Evidence of cccDNA targeting
- Strong safety and tolerability profile

Spring Bank Interpretation of FDA Draft Guidance on HBV Drug Development

- HBsAg loss (functional cure) remains major goal for Spring Bank with greatest potential for clinical uptake and benefit
- HBV DNA sustained off treatment suppression (suppressive cure) is a strong opportunity for additional benefit in both treatment-naïve and NUC-suppressed patients who do not have HBsAg loss and is most likely achievable with an immune activator such as inarigivir
- Different approaches acceptable for treatment-naïve and NUC-suppressed patients and also HBeAg +ve and -ve patients
- Safety database of >1,000 patients will be required with long term follow up to demonstrate maintenance of response

Inarigivir Development Plan: 2019 – 2020

- Differential approach to inarigivir treatment for treatment-naïve and NUC-suppressed HBV patients
- Clinical trial designs responsive to new FDA guidance for HBV with clinical endpoints to include both sustained suppression of HBV DNA and loss of HBsAg
- Initial focus on combination of inarigivir with NUC, but potential for novel combinations in 2019, including triple therapy
- Planned Clinical Trials
  - CATALYST 1 and CATALYST 2 (Q2 2019 initiation)
    - Includes SUPPRESS & SHOCK and STOP & SHOCK cohorts
  - Liver Biopsy MOA study (Q1 2019 initiation)
- Potential for Phase 3 initiation in 2020

Inarigivir Development Plan Accounts for HBV Heterogeneity

HBeAg - ve

- **70-80% of chronic HBV patients**
- **Dominant population in US & EU**
- **Older age group**
- **Lower viral burden**

HBeAg + ve

- **20-30% of chronic HBV patients**
- **Younger population**
- **High viral burden**

POTENTIAL MARKET ENTRY

~17 Million infected with HBV in US and EU

~12-15% TREATMENT RATES **~10-12%**

NUC-Suppressed

Inarigivir + existing oral anti-viral
"Suppress & Shock"
OR
Inarigivir monotherapy
"Stop & Shock"

POTENTIAL EXPANSION OPPORTUNITY

Naïve or new to treatment

Opportunity to **increase treatment rates** with improved functional cure

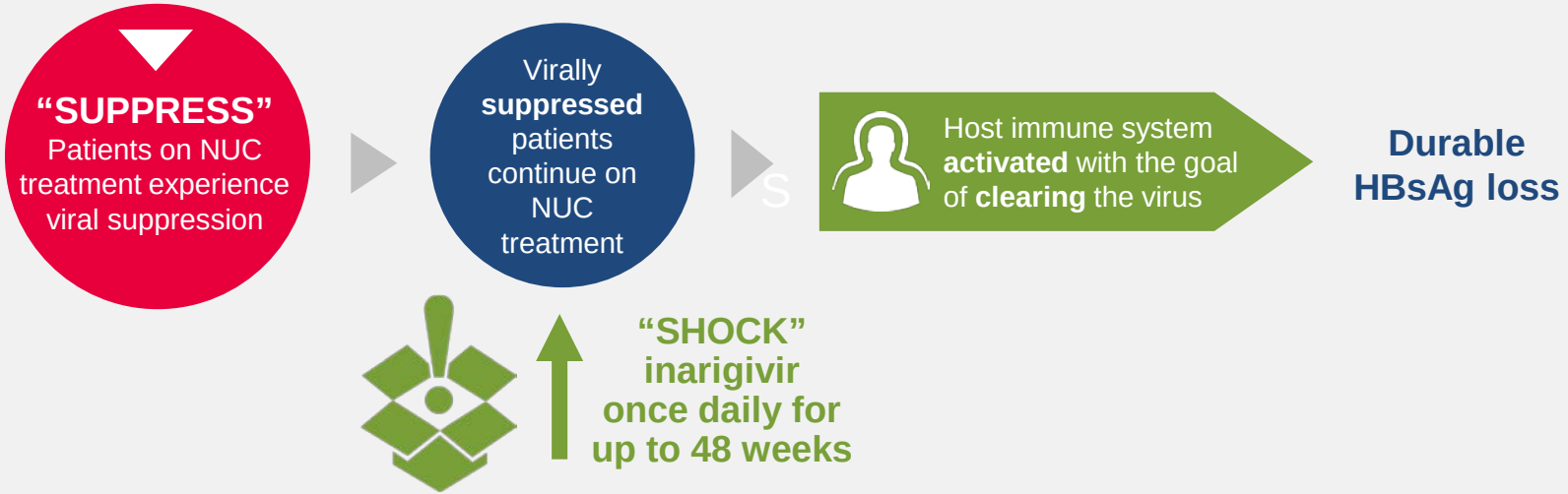
SB 9225
(inarigivir + TDF)

SB 9225 + different MOA

- Further expand HBV population eligible for functional cure
- Potentially reduce treatment duration for other HBV MOAs

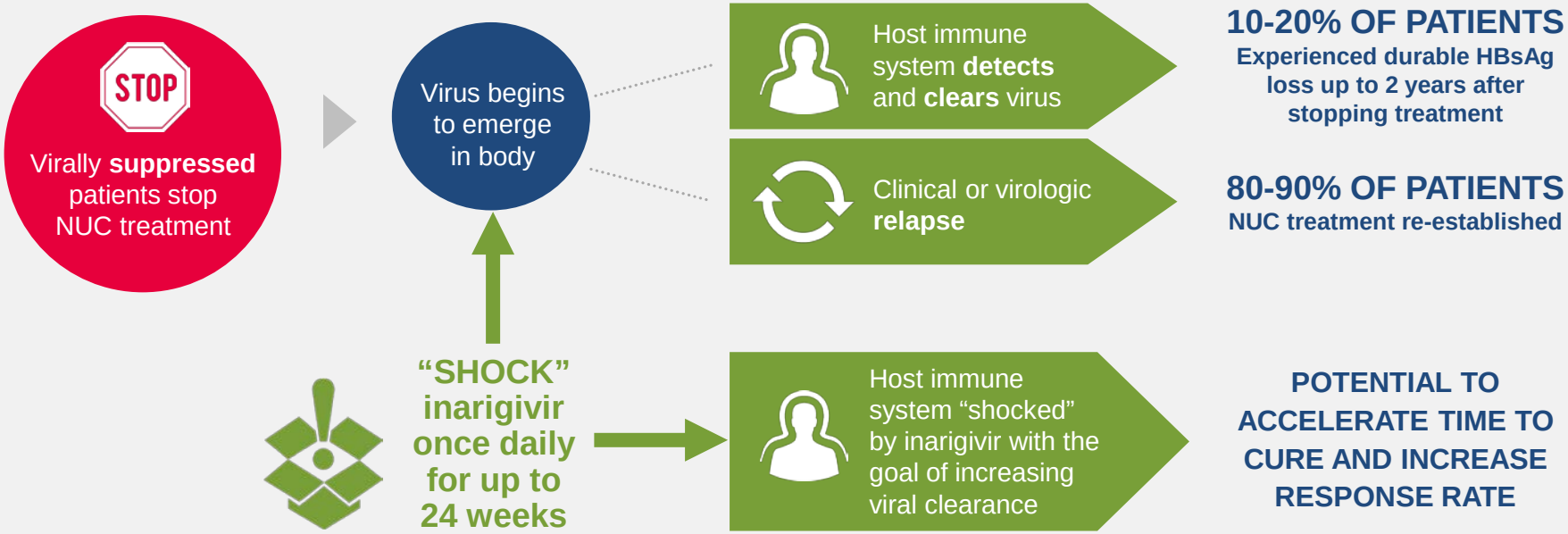
“Suppress & Shock” Adding Inarigivir in Virally Suppressed Patients

Inarigivir immunomodulation anticipated to be studied in a Phase 2b/3 trials for its ability to promote HBsAg loss in NUC-suppressed patients



“Stop & Shock” – Inarigivir Monotherapy

Inarigivir monotherapy anticipated to be studied in a Phase 2b/3 trial for its potential to improve current functional cure rate (10-20%) and reduce relapse rates following cessation of SOC therapies



Planned CATALYST 1 Trial – Key Features and Outcomes

- 60 HBeAg +ve and -ve, non-cirrhotic, treatment-naïve patients
- 3 cohorts (response-guided)
  - Inarigivir 400mg daily monotherapy for 12 weeks, followed by co-administration with Vemlidy® 25mg for 12 weeks (n=20)
  - Inarigivir 400mg three times a week for 12 weeks, followed by co-administration with Vemlidy® 25mg for 12 weeks (n=20)
  - Inarigivir 400mg daily + Vemlidy® 25mg daily for 24 weeks (n=20)
- Pre-defined responder patients at week 24 may continue to receive additional 24 weeks of treatment
- Potential to see functional cure
- End points
  - HBV DNA, HBV RNA
  - HBeAg loss, HBsAg reduction/loss
- ***When combined with data from Gilead's trial of inarigivir + Vemlidy®, will inform Phase 3 treatment-naïve strategy for SB 9225 (inarigivir + tenofovir disoproxil fumarate fixed-dose combination)***

Planned CATALYST 2 Trial – Key Features and Outcomes

- 60 HBeAg -ve, non-cirrhotic, NUC-suppressed patients
- COHORT 1
  - “Stop and Shock” – Stop NUC, inarigivir 400mg daily for 24 weeks (n=20)
  - End points: ALT flare, fine needle aspirations (FNA) of liver for intra-hepatic immunology, HBsAg loss
- COHORT 2 (response-guided, up to 48 weeks of treatment)
  - “Suppress and Shock” Design (n=40)
  - Specialized intra-hepatic virology and immunology using liver FNA
  - End points: HBsAg reduction/loss
- ***Will inform potential Phase 3 treatment for NUC-suppressed patients in 2020***

SB 9225 – Fixed-Dose Combination for Naïve HBV

- SB 9225 (inarigivir + TDF)
- Planned Phase 3 program in US, EU and ASIA in 2020
- Newly diagnosed chronic HBV patients
- SB 9225 once-daily vs. NUC alone
- Primary endpoints: HBV DNA & durable HBsAg loss

Novel Combination Opportunities for Inarigivir in 2019

- siRNA and anti-sense strategy
  - Rational design based on efficacy of inarigivir with low HBsAg level
  - Can utilize pulse dosing strategy
  - HBsAg loss as endpoint
- NUC / CpAM combination
  - Can increase efficacy or reduce therapy duration
  - Pre-clinical data suggests potential additive DAA and immunomodulatory benefits
- Dual immunomodulator
  - Combine innate immune activation of inarigivir with adaptive / T-cell strategies
  - Inarigivir with either TLR8 or PD-1 inhibitors attractive combinations

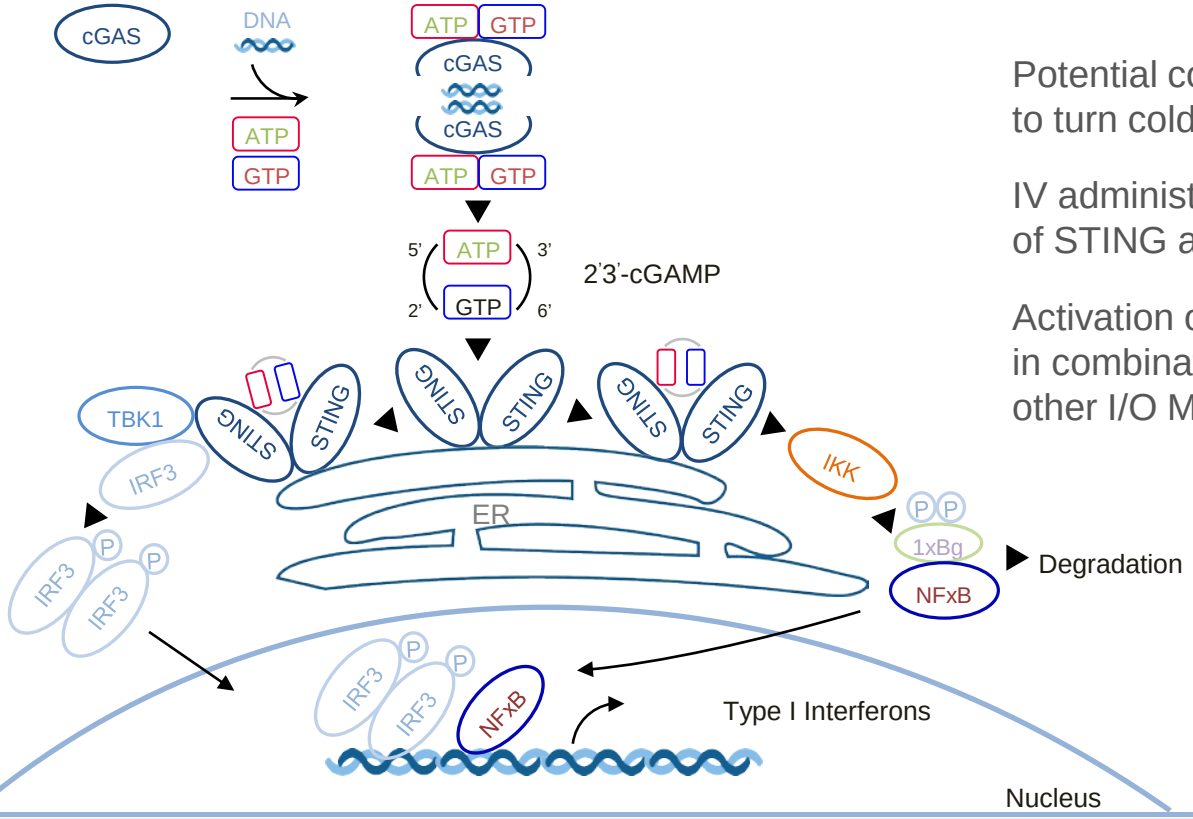
LEAD COMPOUND

SB 11285

A NOVEL SYNTHETIC

STING AGONIST

STING Agonist – Potent Activator of Innate Immune Response

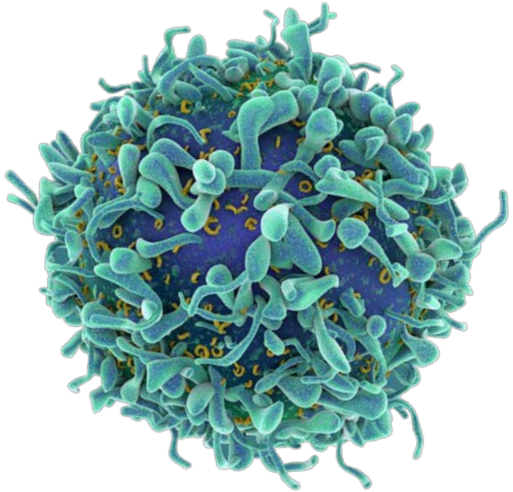


Potential component of I/O strategies to turn cold tumors “hot”

IV administration could be next frontier of STING agonist development

Activation of Type I IFN could enhance efficacy in combination with checkpoint inhibitors or other I/O MOAs

Spring Bank's Second-Generation STING Agonist Platform



Differentiated cyclic dinucleotide

Potentially first IV STING agonist to enter clinic

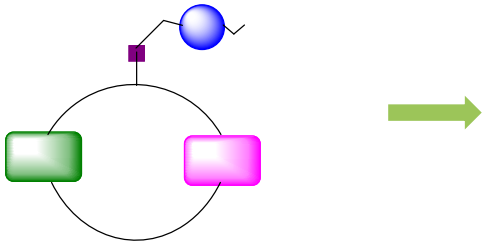
Observed to turn "cold" tumors "hot" in preclinical studies

Shown to be highly potent & efficacious across multiple preclinical cancer models with associated abscopal and tumor memory responses

Distinctive chemistry allows for potential nanoparticle formulation and conjugation with ADCs for targeted delivery

Unique chemistry allows for "self assembly" could enhance immune cell recruitment via IV administration

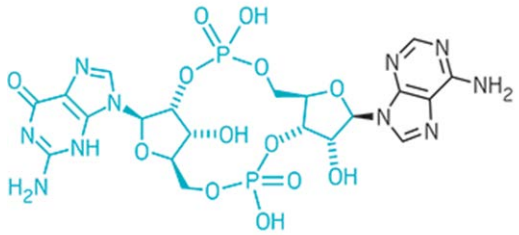
Spring Bank's Second-Generation STING Agonist Platform

Spring Bank's STING agonists	• More potent • Cell permeable • Flexible dosing routes
Demonstrated single agent potent activity across tumor models by I.V., I.P. & I.T.	• In vitro - enters cells without permeabilizing agents • In vivo - induces immune cell infiltration • Leads to a "hot" immune phenotype at tumor site • Induces immune memory
Synthetic dinucleotide scaffolds 	• Optimized base and linker compositions • Proprietary chemical modifications for differentiated chemistry • Unique chemistry allows site specific conjugation for tumor targeting • Enables nanoparticle formulation for enhanced immune cell uptake

Cell permeability critical for uptake of agonist into DCs and immune cells at TME and periphery

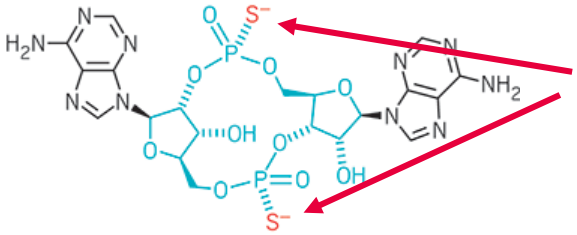
Systemic delivery may facilitate trafficking of newly activated CD8+T cells from periphery into the tumor site

First Generation STING Agonists – Poor Cellular Permeability



2’3’-cGAMP

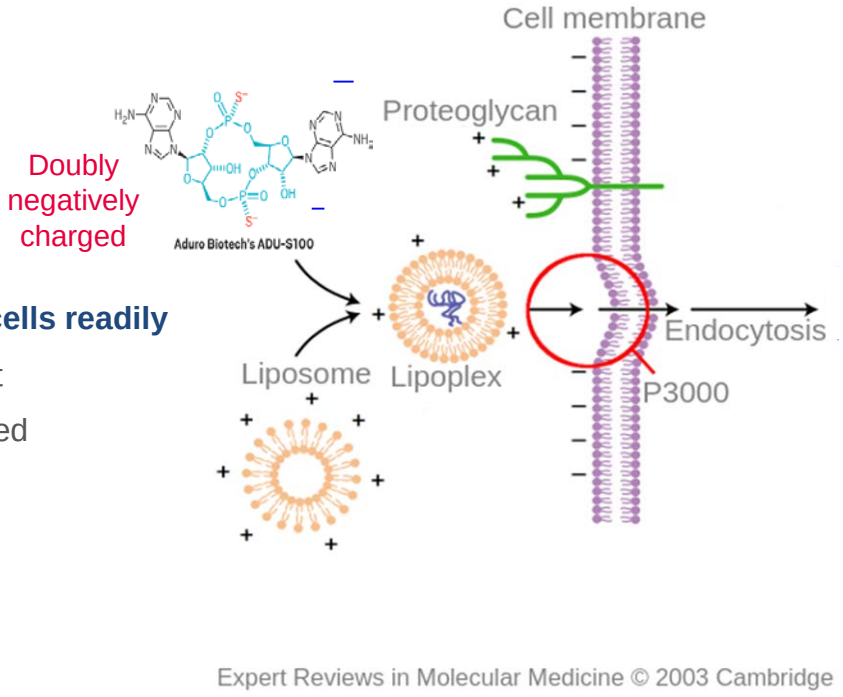
- Natural STING Ligand
- Cyclic di-nucleotide
- Guanine/Adenine
- Lack lipophilicity
- Nuclease labile



Aduro Biotech's ADU-S100

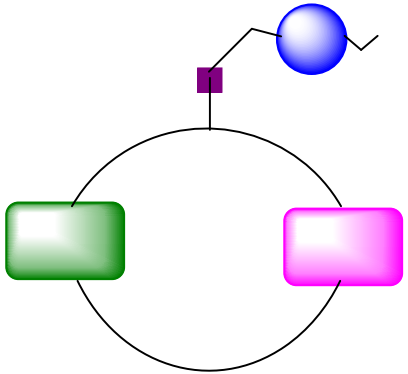
Does not enter cells readily

- Synthetic STING agonist
- Doubly negatively charged
- Adenine/Adenine
- 2’3’ Linkage
- Lack lipophilicity
- IT administration



SB 11285 – Second-Generation STING Agonist Candidate

SB 11285 is efficiently engineered



Structure Imparts Improved Drug-Like Properties

- ✓ Enhanced lipophilicity
- ✓ Greatly enhanced cellular permeability
  - ✓ Demonstrates potency without lipofectamine
- ✓ Safe and efficacious systemic delivery
- ✓ Enables regio-specific conjugation capability

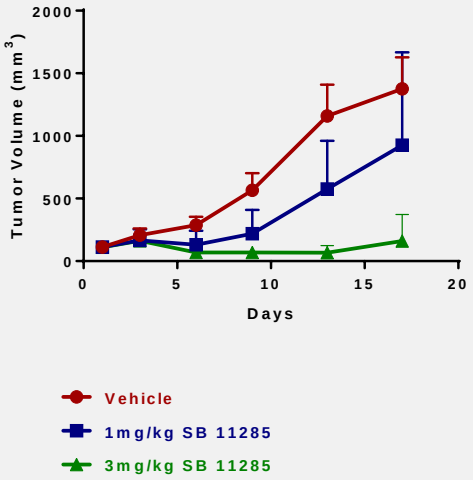
Cell permeability critical for uptake of agonist into DCs and immune cells at TME and periphery.

Systemic delivery may facilitate trafficking of newly activated CD8+T cells from periphery into the tumor site.

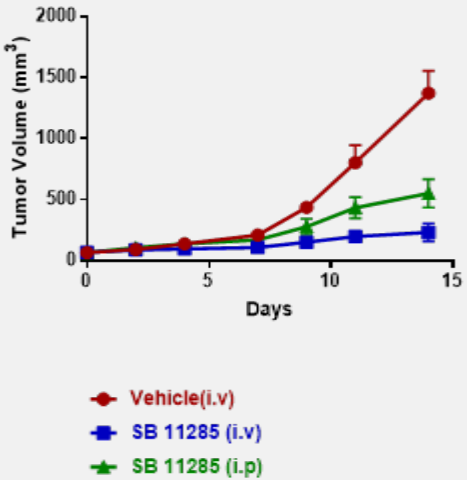
SB 11285 Significantly Inhibited Tumor Growth in Relevant Oncology Models

Efficacy in relevant oncology animal models observed with intravenous (IV), intraperitoneal (IP) and intratumoral (IT) delivery

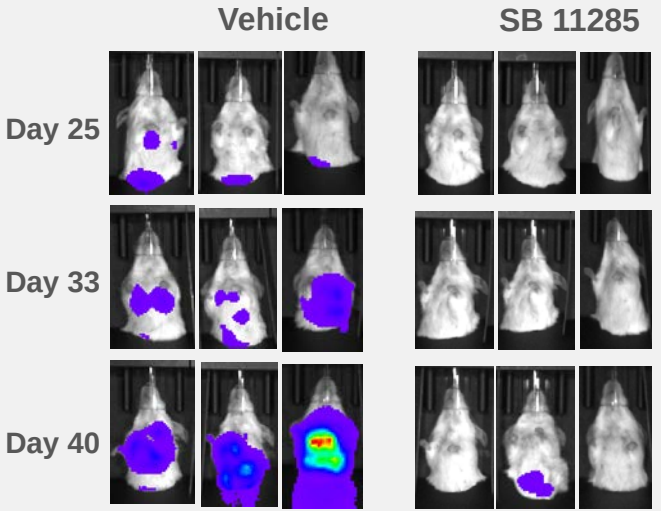
CT26 Colon Cancer (IV)



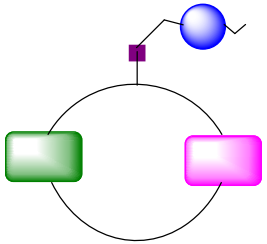
B16 Melanoma (IV)



4T1 Metastatic Breast Cancer (IP)



Highlights of Spring Bank's Second-Generation STING Agonists



Potential to make immunotherapy available to a much wider patient population

Spring Bank's Second-Generation STING Agonists:

- Designed using intelligent chemistry to enable efficient cellular delivery
- Key missing component of current clinical programs
- Possibility to treat tumor types that are not accessible by I.T.

- I.V. and I.T. administered SB 11285 lead to tumor regression in multiple preclinical models
  - Melanoma, Breast, Colorectal, Glioblastoma, AML

Turns cold tumors hot

- Induces a local concentration of cytokines for activation of Antigen Presenting Cells
- Facilitates trafficking of increased numbers of CD8 CTL cells to tumor site
- Reduces population of Treg cells at tumor site

Distinctive chemistry

- Better tolerated - reduced induction of proinflammatory cytokines
- Enable systemic delivery to facilitate trafficking of activated CD8+T cells from periphery into tumor site
- Enables nanoparticle formulation
- Facilitate conjugation with antibodies and targeting agents

SB 11285 Development Plan

- FDA IND submission Q2 2019
- Phase 1/2 IV SB 11285 studies as monotherapy with early transition to combination therapy in locally invasive and metastatic solid tumors
  - Includes patients with PD-1/PDL-1 failure
- IT SB 11285 study in Hepatocellular Carcinoma (HCC)/solid tumors

The Path Forward for Spring Bank

Potential for Multiple Catalysts

Upcoming 2019 Milestones

1H 2019

Initiate liver biopsy study

Report 12 and 24 week dosing from the final cohort (200mg) of the ACHIEVE Phase 2 trial

Initiate global inarigivir CATALYST 1 and 2 trials (including SUPPRESS/STOP & SHOCK cohorts)

Submit IND for SB 11285

2H 2019

Report initial data from first inarigivir + NUC combination cohorts (GILD data)

Initiate Phase 1/2 clinical trial(s) with SB 11285

Report initial data from liver biopsy study

Initiate Phase 2 trial with inarigivir and novel MOA (triple combination)

Spring Bank Pharmaceuticals, Inc.

A Focus on Simplicity, Safety, and Selectivity

Unique in-house platform

Focused on small molecule nucleotide hybrid immunomodulatory molecules

World class expertise in HBV

Deep clinical collaboration in HBV with Gilead

Orally administered inarigivir has clinically shown potent antiviral activity in HBV

Favorable safety profile to date with no related SAEs observed

SB 9225

(inarigivir + TDF)

Simplifies combination therapy

Next-generation STING agonist program:

Lead candidate SB 11285 anticipated to enter clinic in multiple cancers in 2019

Anticipate **multiple data points for potential valuation enhancements** in the next 12–18 months