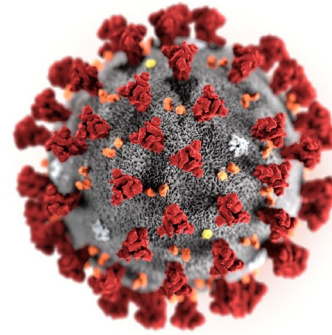
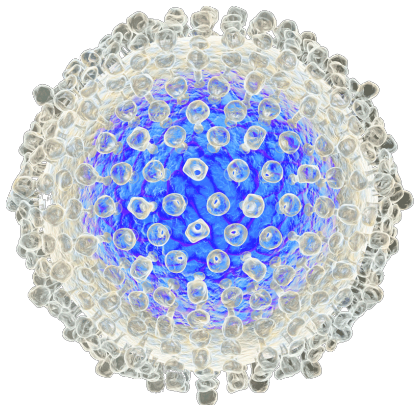


ALIGOS
THERAPEUTICS



Lawrence M. Blatt, Ph.D.
Chairman, CEO
& Co-Founder

Corporate Presentation
September 2021



Disclosures

This presentation contains forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995. All statements other than statements of historical facts contained in this presentation, including statements regarding our future results of operations and financial position, business strategy, prospective drugs and drug candidates, the potential scope, progress, results and costs of developing our drug candidates or any other future drug candidates, the potential market size and size of the potential patient populations for our drug candidates, the timing and likelihood of success of obtaining drug approvals, ability to maintain existing, and establish new, strategic collaborations, licensing or other arrangements, plans and objectives of management for future operations, the scope of protection we are able to establish and maintain for intellectual property rights covering our drug candidates, and future results of anticipated drugs and drug candidates, and the impact of developments related to the COVID-19 pandemic are forward-looking statements. These statements involve known and unknown risks, uncertainties and other important factors that may cause our actual results, performance or achievements to be materially different from any future results, performance or achievements expressed or implied by the forward-looking statements. Because forward-looking statements are inherently subject to risks and uncertainties, some of which cannot be predicted or quantified and some of which are beyond our control, you should not rely on these forward-looking statements as predictions of future events. The events and circumstances reflected in our forward-looking statements may not be achieved or occur and actual results could differ materially from those projected in the forward-looking statements. Except as required by applicable law, we do not plan to publicly update or revise any forward-looking statements contained herein, whether as a result of any new information, future events, changed circumstances or otherwise. For a further description of the risks and uncertainties that could cause actual results to differ from those anticipated in these forward-looking statements, as well as risks relating to the business of Aligos Therapeutics in general, see Aligos' Quarterly Report on Form 10-Q filed with the Securities and Exchange Commission on August 5, 2021, and its future periodic reports to be filed with the Securities and Exchange Commission. Except as required by law, Aligos Therapeutics undertakes no obligation to update any forward-looking statements to reflect new information, events or circumstances, or to reflect the occurrence of unanticipated events.

Except where otherwise indicated, the information contained in this presentation speaks as of the date hereof or as of the date at which such information is expressed to be stated, as applicable, and such information may express preliminary estimated, unaudited results which shall be subject to audit or other year-end adjustments and such audited or adjusted results may materially differ from those contained in this presentation.

This presentation concerns drug candidates, some of which are undergoing nonclinical studies and others of which are under clinical investigation, and all of which have not yet been approved for marketing by the U.S. Food and Drug Administration. These drug candidates are currently limited by federal law to investigational use, and no representation is made as to their safety or effectiveness for the purposes for which they are being investigated.

Aligos Therapeutics Highlights

OUR MISSION

Develop novel therapeutics to address unmet medical needs in viral and liver diseases

OUR PLATFORM

Oligonucleotides
Small molecules
Leverage complementary modalities to broaden the range of therapeutic targets we can address

OUR STRATEGY

Develop pharmacologically optimized drug candidates against clinically-validated targets
Pursue combination regimens designed to achieve improved treatment outcomes

OUR TEAM

Decades of drug discovery and development experience
Proven track record of success in the areas of viral infections and liver diseases

OUR GOAL

Become a world leader in antiviral therapies for CHB and create targeted therapeutics for NASH

Our Platforms, Capabilities and Collaborations

Oligonucleotide Modalities

Small Molecule Chemistry

Intellectual Property

Chemistry, Manufacturing
and Controls

Biological Sciences

Translational Safety
Sciences

Clinical Sciences and
Operations

ALIGOS
THERAPEUTICS

Emory University
CAM IP & Collaboration

Luxna Biotech
3rd Generation XNA Nucleotides

AM Chemicals
Proprietary GalNAc

Merck (MSD) NASH
Collaboration

Rega / CD3
Collaboration COVID Protease

Agency for Innovation and
Entrepreneurship (VLAIO)

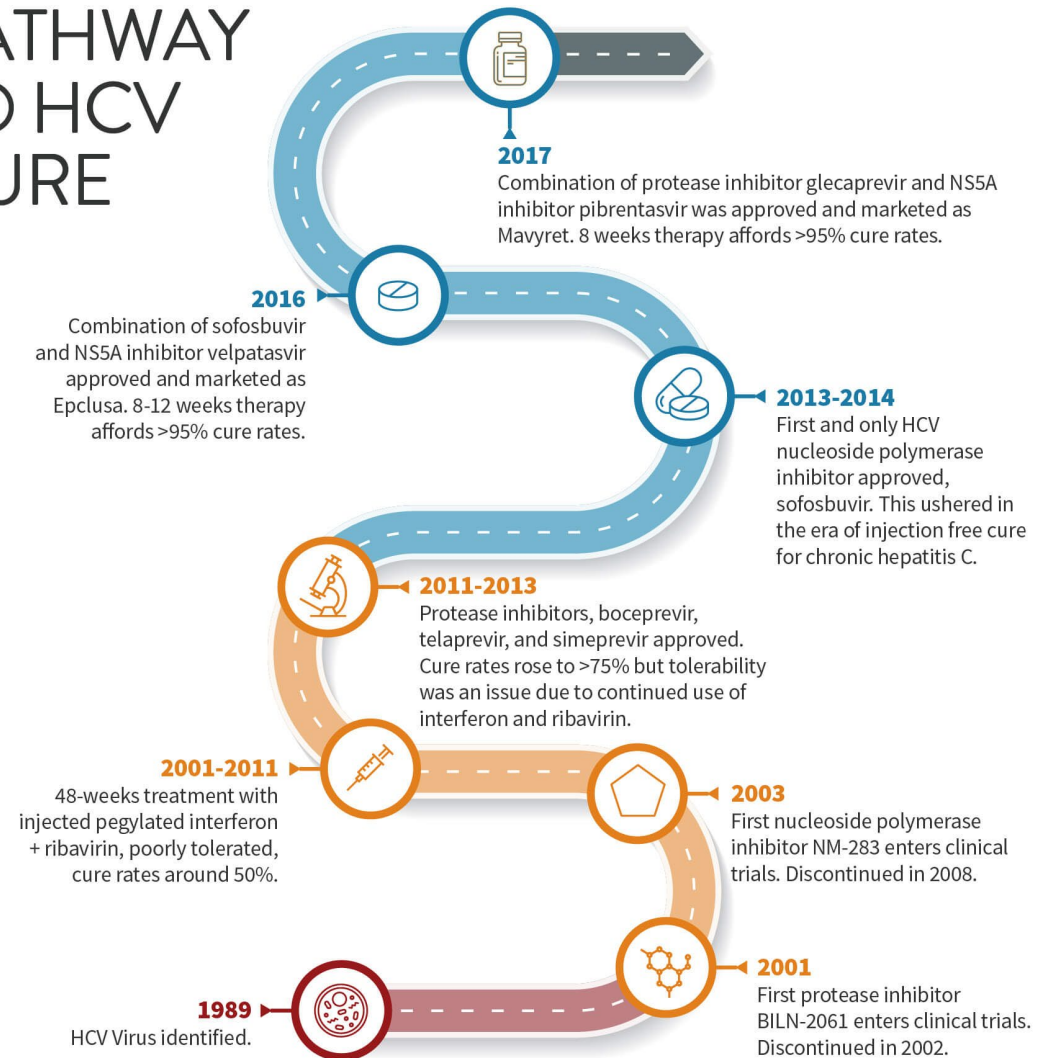
Enabling efficient drug discovery in pursuit of optimized combination regimens

A Lesson from Chronic Hepatitis C

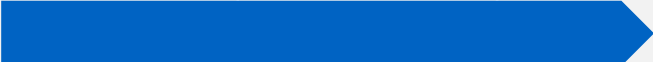
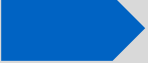
From First Movers to Best-in-Class Regimens

- Efficacy → Safety → Convenience
- First in class entrants
 - Demonstrated mechanistic proof of principle
 - Several did not gain approval due to poor pharmacology
- Late entrants with improved pharmacology attained significant market share
- Aligos is pursuing potentially best-in-class molecules for CHB where clinical validation has been demonstrated

PATHWAY TO HCV CURE

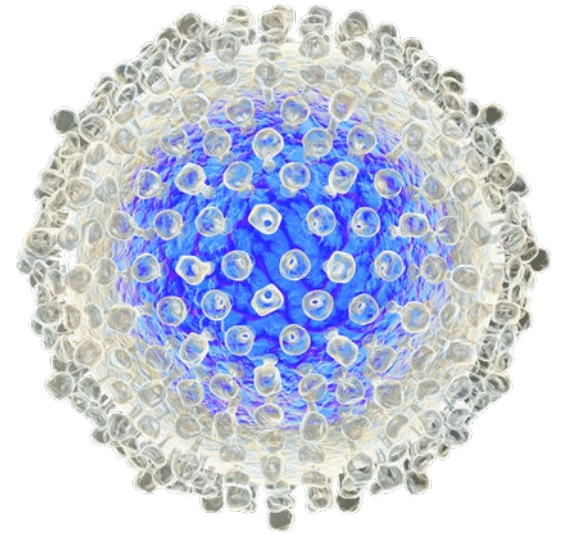


Our Pipeline

Candidate	Indication	MOA	Discovery	Nonclinical	Phase 1	Phase 2	Phase 3	Anticipated Milestones
ALG-010133	CHB	STOPS™						Initial Phase 1 Data
ALG-000184	CHB	CAM						Initial Phase 1 Data
ALG-020572	CHB	ASO						Phase 1 Start in 2H 2021
ALG-125755	CHB	siRNA						Phase 1 Start in 1H 2022
ALG-055009	NASH	THR-β Agonist						Phase 1 Start in 2H 2021
Discovery	Coronavirus	Multiple						—
Discovery	Liver Diseases	Multiple						—
Discovery	NASH	Undisclosed						—

LEGEND  Oligonucleotides  Small Molecules  Multiple Modalities

Chronic Hepatitis B



CHB Epidemiology & Our Portfolio

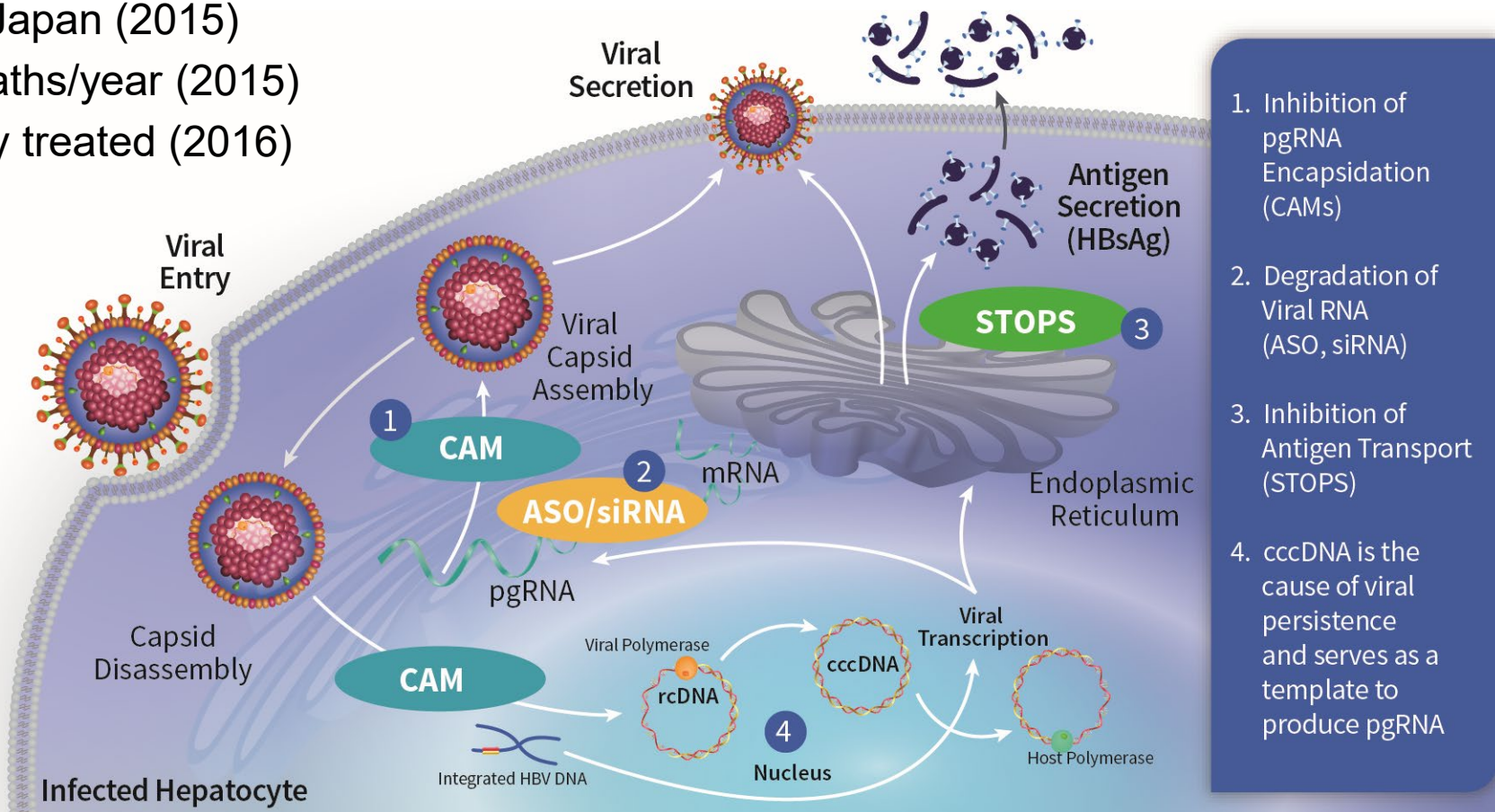
- CHB Epidemiology* - Most common chronic viral infection in the world

- >290 million carriers worldwide (2020)
- Affects ~8 million in US, EU, Japan (2015)
- Responsible for ~900,000 deaths/year (2015)
- Only ~2% of patients currently treated (2016)

- Our Portfolio

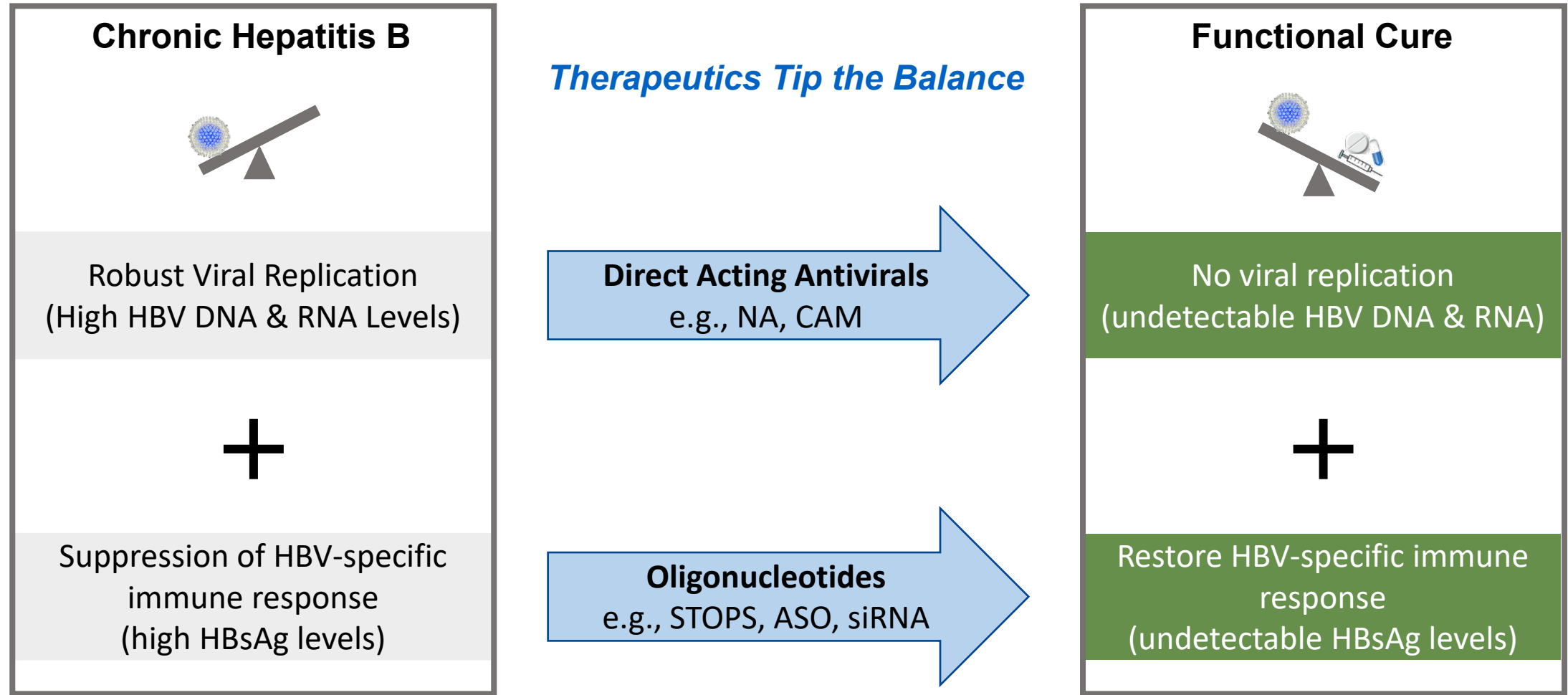
- Potentially best-in-class drug candidates against validated targets with potential for high functional cure rates

- › CAM
- › Oligonucleotides
 - STOPSTTM
 - ASO
 - siRNA

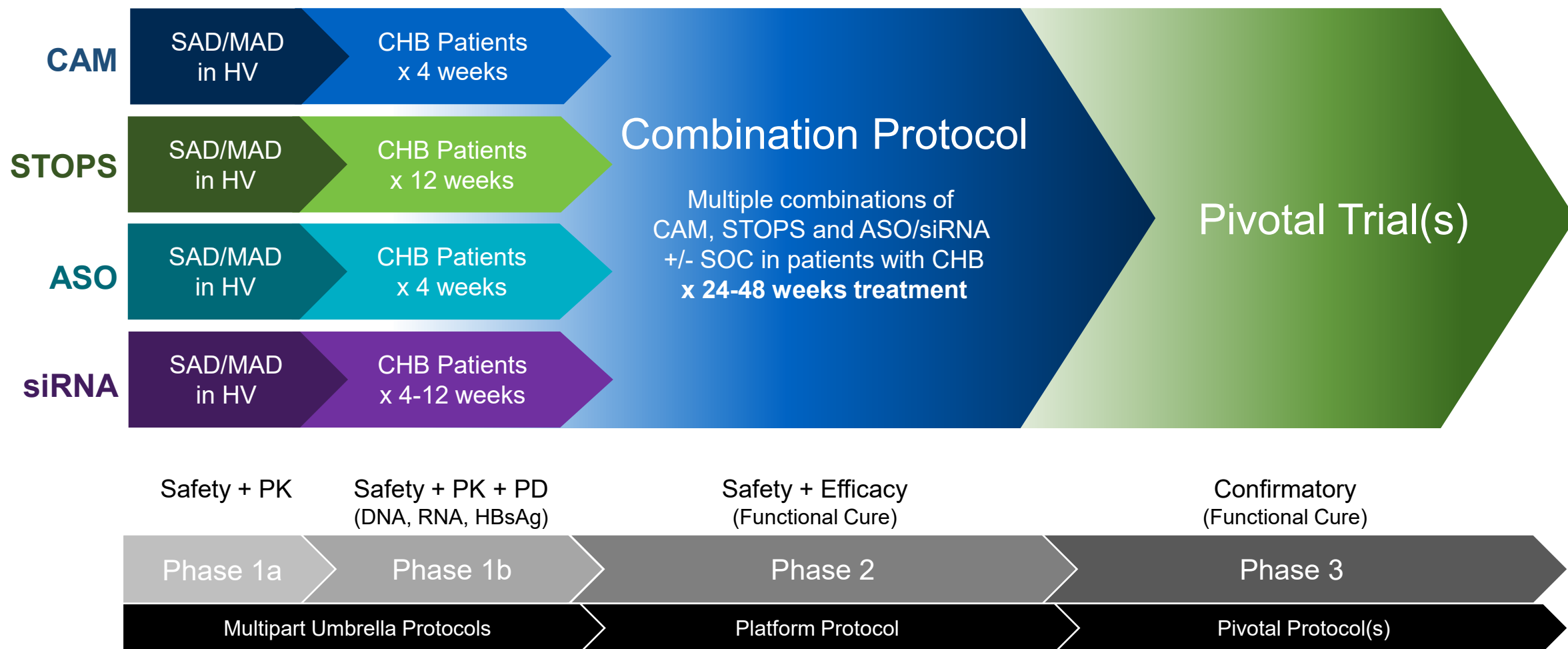


Chronic Hepatitis B and Functional Cure

Outcome Determined by Balance Between Virus and Host Immune Response



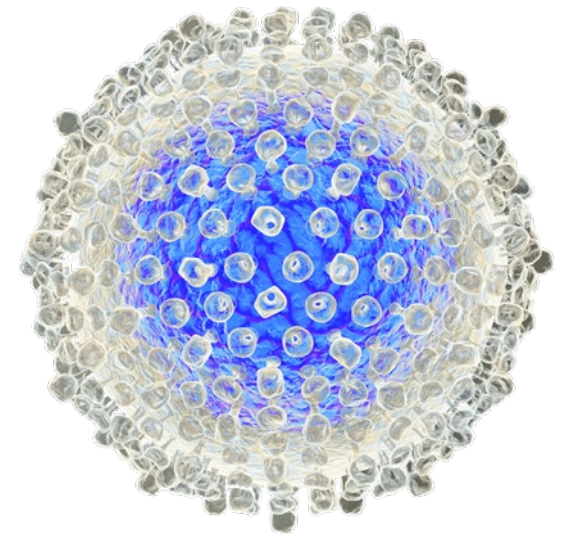
Our CHB Clinical Development Strategy



STOPS

ALG-010133, a novel, chemically optimized poly-AC oligonucleotide analog

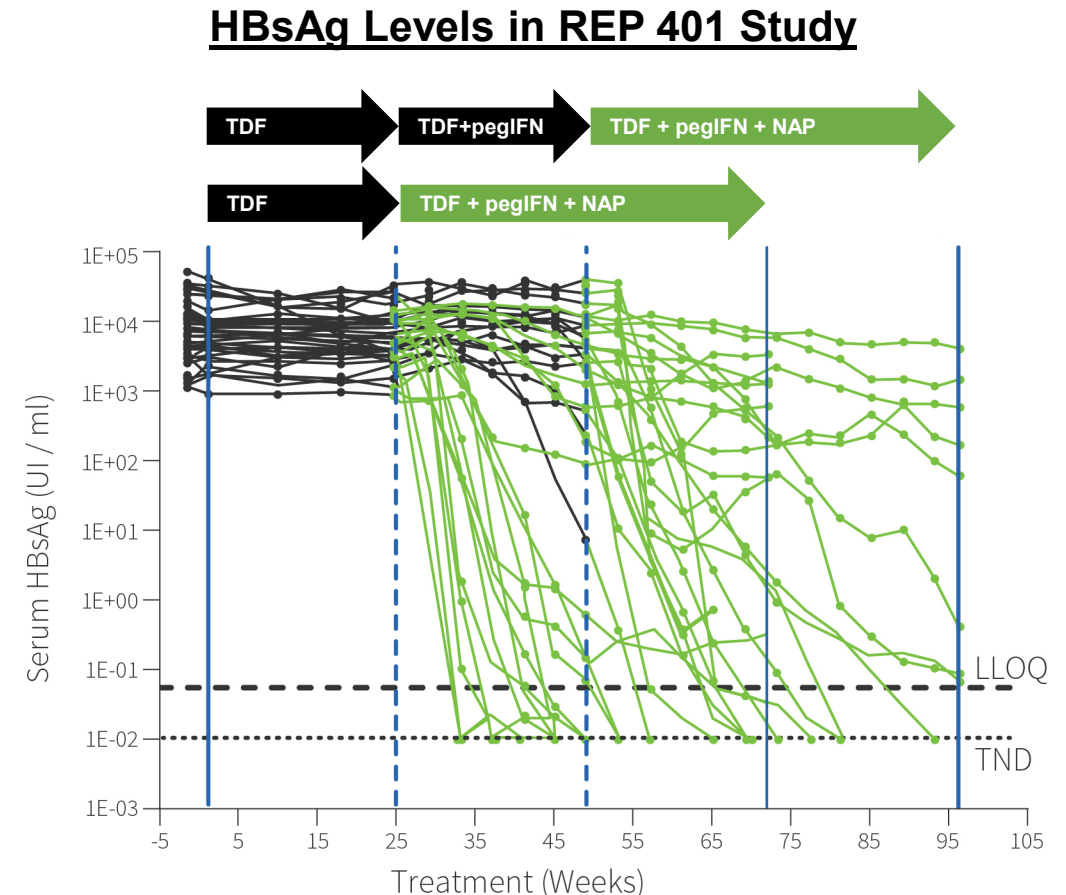
(STOPS = S-antigen Transport inhibiting Oligonucleotide Polymers)



Poly AC Oligonucleotides Have Clinical Validation

Functional Cures Observed in Combination Studies

- Replicor 401 Study (Figure*)
 - Treatment naïve, HBeAg negative
 - NAP REP 2139-Mg or REP 2165-Mg
 - › NAP = Nucleic Acid Polymer
 - 250 mg Q-weekly, 2-hr IV administration
 - Triple combination
 - › With tenofovir and pegylated-IFN
- Reported a ~39% functional cure rate**



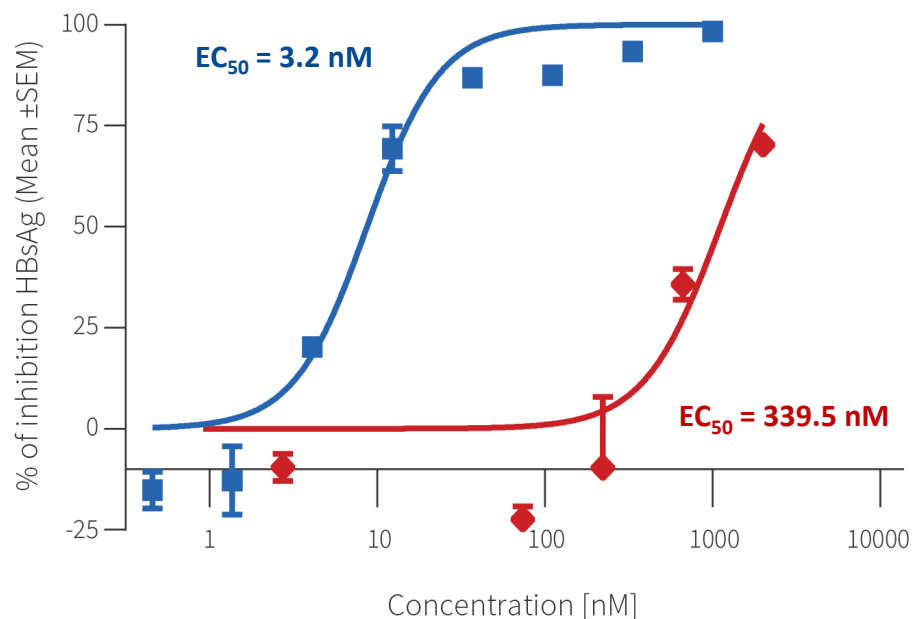
Treatment results in multiple $\log_{10}$ IU/mL reductions of HBsAg and substantially higher rates of functional cure

ALG-010133

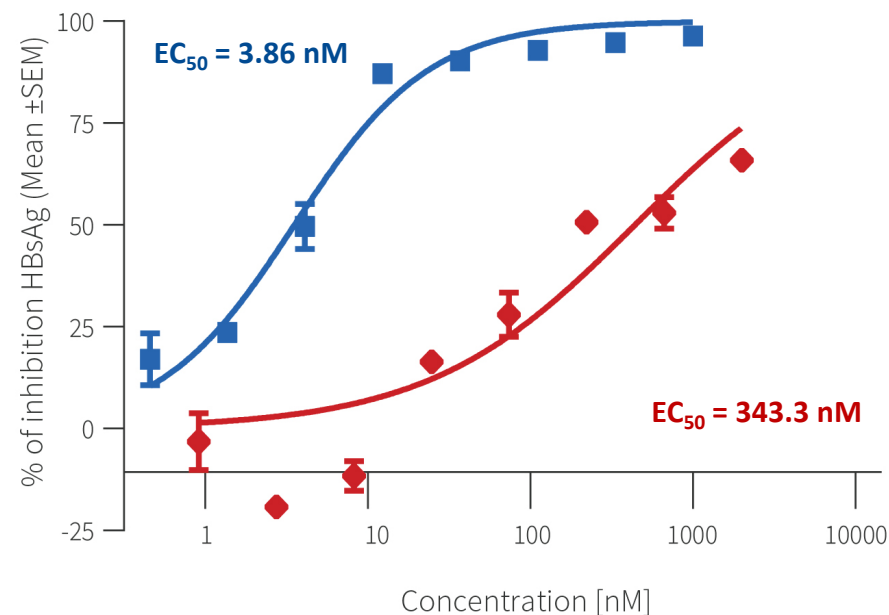
Our Lead STOPS Molecule

Significantly greater potency* vs. reference Poly-AC oligonucleotide**

Dose-response curves for ALG-010133 and ALG-010004 induced inhibition of HBsAg in HepG2-NTCP



Dose-response curves for ALG-010133 and ALG-010004 induced inhibition of HBsAg in HepG2.2.15



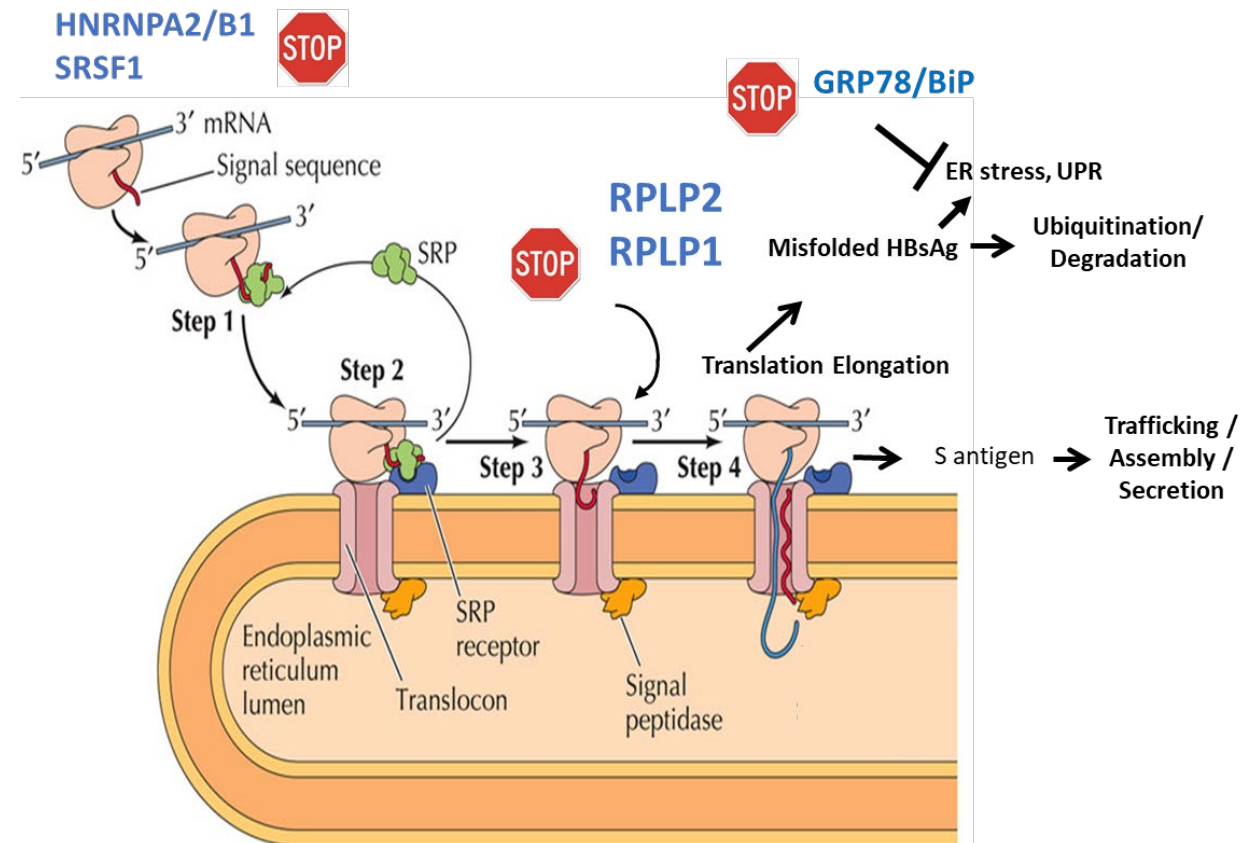
LEGEND ■ ALG-010133 ◆ ALG-010004

~100-Fold improvement in potency enables efficient subcutaneous administration

STOPS

Mechanism of Action to Inhibit HBsAg Secretion*

- STOPS affinity chromatography identified ~20 cellular binding proteins
- Five of these proteins are required for optimal HBsAg production based on siRNA knockdown experiments in HepG2.2.15 cells
 - SRSF1, HNRNPA2/B1, RPLP2 and RPLP1, GRP78
- All five proteins have previously been reported to function in other viral infections
 - HIV, CMV and flaviviruses
- STOPS molecules have a complex mechanism of action
 - By sequestration of proteins involved in RNA processing, STOPS molecules decrease the production of HBsAg
 - STOPS molecules sequester proteins required to translate and properly fold HBsAg, which increases ubiquitination and intracellular degradation

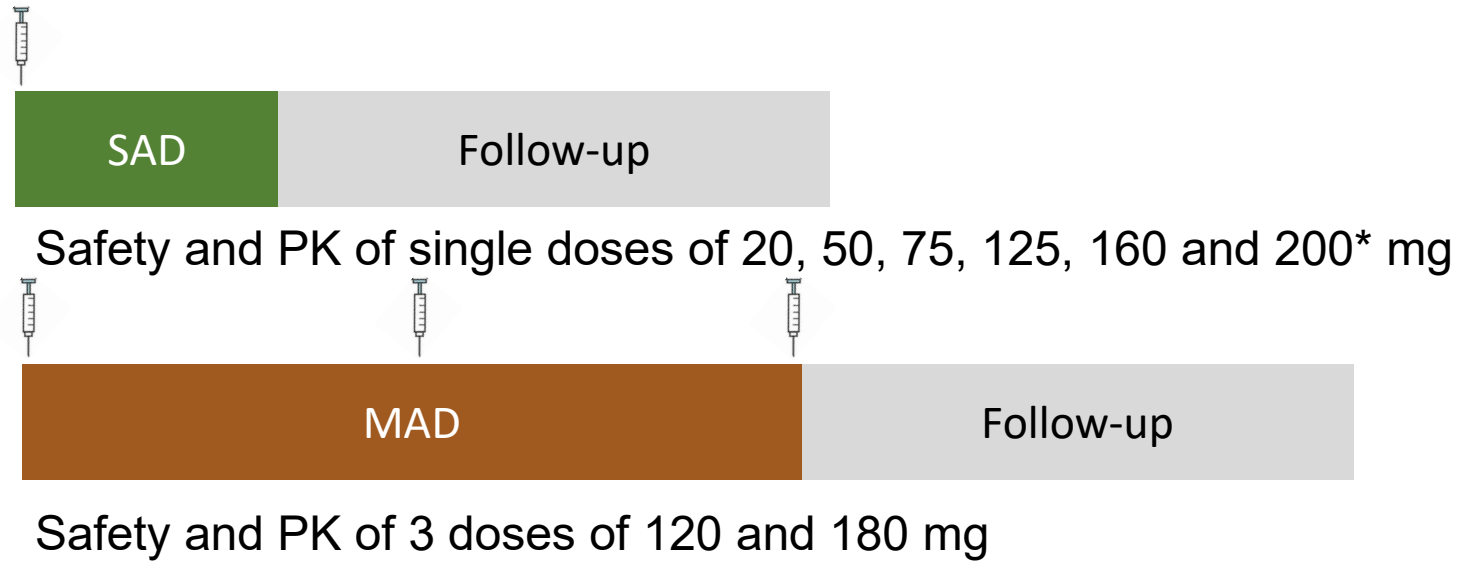


ALG-010133 Phase 1 Study in HV

Study Design

Single/Multiple Ascending Dose (SAD/MAD) study in HV

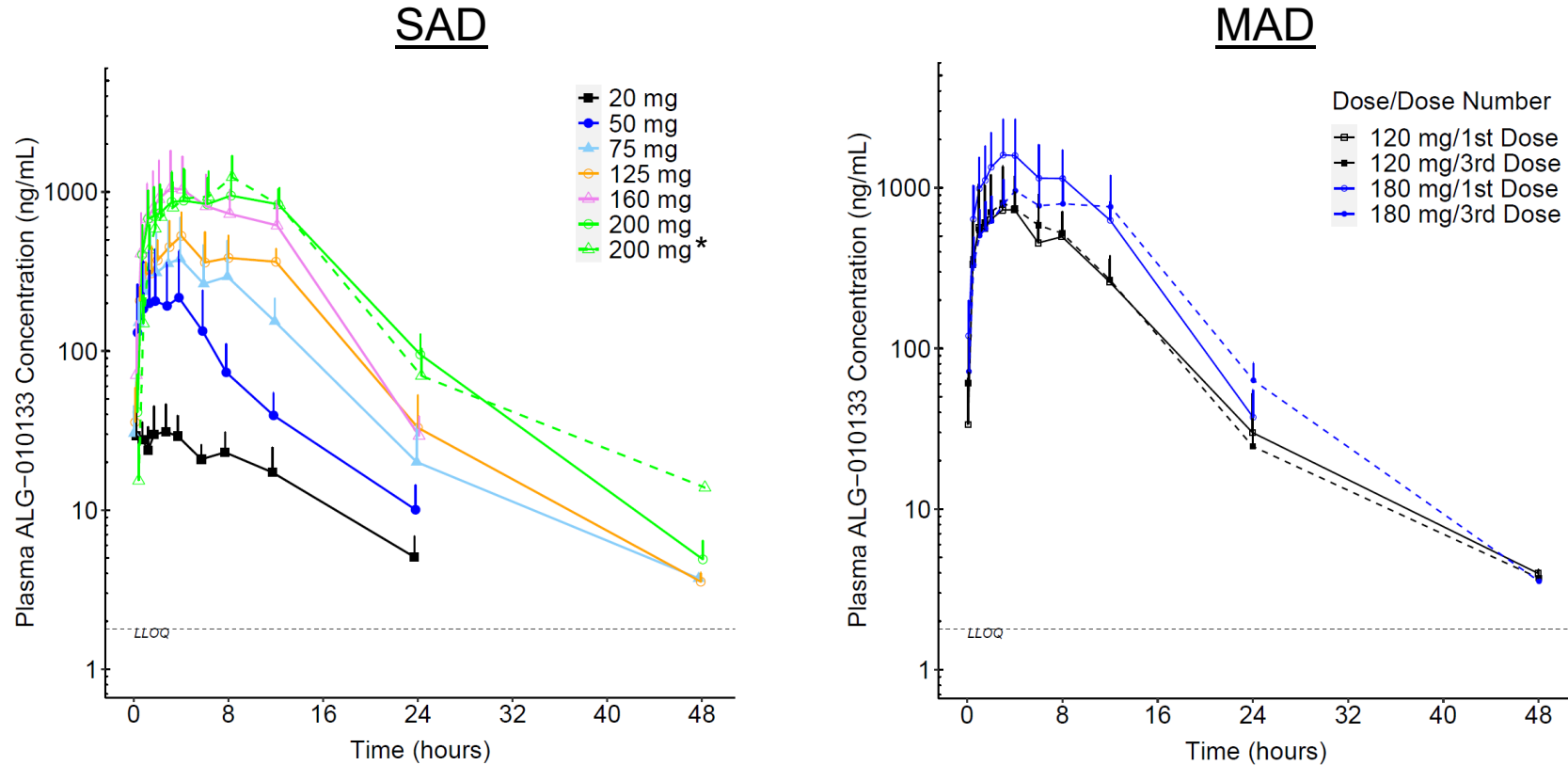
N=72; n=8 per Cohort, n=6 ALG-010133, n=2 Placebo
Single (SAD) or three weekly subcutaneous (SC) doses (MAD)



Dosing in HV complete

ALG-010133 Phase 1 Study in HV

PK Data



Doses ≥ 125 mg approach linearity; low-to-moderate intersubject variability; no accumulation
Doses ≥ 120 mg anticipated to have antiviral activity

ALG-010133 Phase 1 Study in HV Safety

- No serious adverse events (SAEs)
- Treatment emergent adverse events (TEAEs)
 - No TEAEs led to study drug discontinuation
 - Injection site reactions (ISRs)
 - › Occurred in ~19% of ALG-010133-treated subjects
 - › Generally characterized by localized erythema that was mild to moderate in severity and resolved over time. One SAD ISR was considered severe based on surface area criteria ($>100 \text{ cm}^2$)
- No clinically concerning laboratory, ECG, vital sign or physical examination findings

Single, multiple ALG-010133 doses generally well tolerated in HV
Prophylactic topical steroids are being used in CHB subjects to potentially mitigate ISRs

CHB Studies

Regulatory Guidance for Advancing Antiviral Drugs

- Phase 1a – Safety / PK in healthy volunteers
- Phase 1b – Safety / PK and biological response (e.g., HBsAg, DNA) in CHB patients. Studies evaluate a range of doses as monotherapy for short durations
 - Demonstrate biological response
 - Unlikely to lead to clinical response (functional cure)
 - Minimizes risk of drug resistance
 - Enables selection of dose(s) for each drug for Phase 2 combination studies
- Phase 2/3 – Combine selected dose(s) with other MOAs and measure clinical response (functional cure) and overall risk-benefit profile

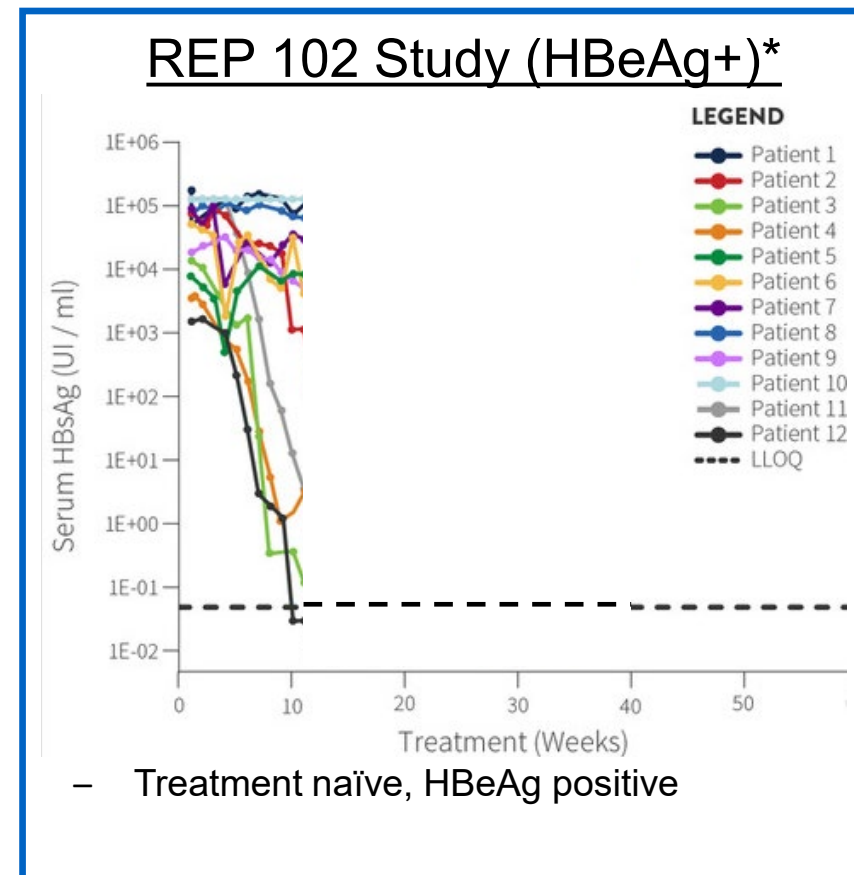
Aligos STOPS, CAM programs currently in Phase 1b

Seeking to demonstrate biologic response by evaluating a range of doses as monotherapy for the biomarker of interest

Poly AC Oligonucleotides Have Clinical Validation

Significant HBsAg Reductions

- Phase 1 NAP study (REP 102)
 - REP 2139-Ca (IV) monotherapy x ≥ 20 weeks
 - Highly variable HBsAg reductions
 - HBsAg reduction at Week 10**
 - › Ranged from +0.1 to -4.7 $\log_{10}$ IU/mL
 - › Median $\log_{10}$ reduction: 0.4 IU/mL
 - › Mean (SD) $\log_{10}$ reduction: 1.65 (1.97) IU/mL
 - No functional cures

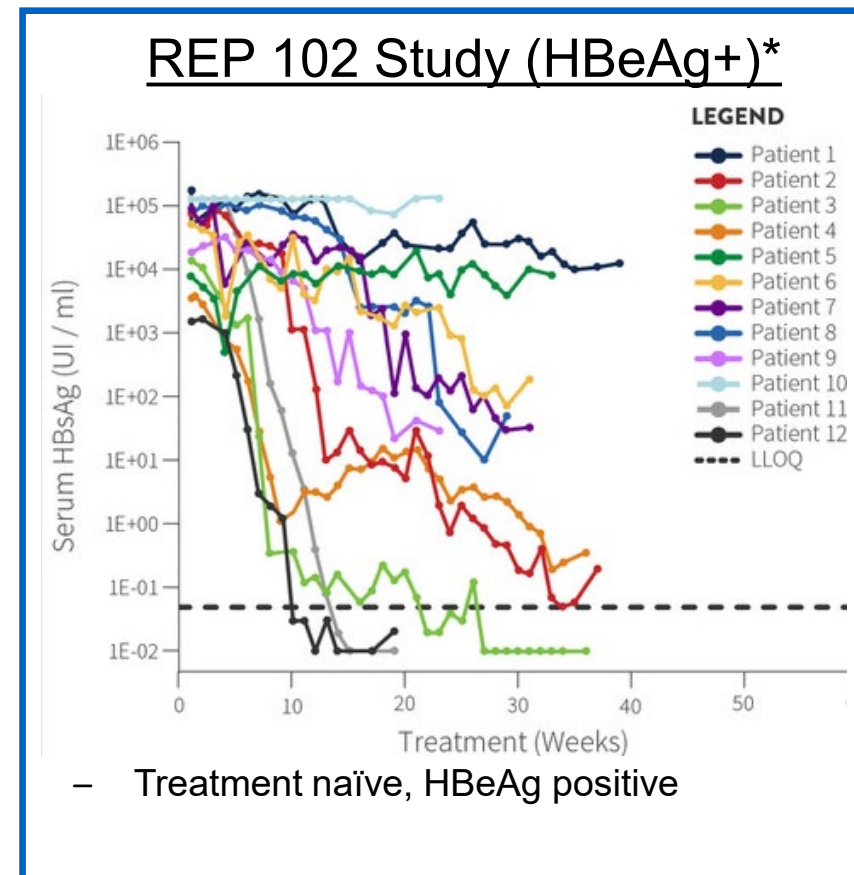


REP-2139 monotherapy x 10 weeks: mean $\sim 1.65 \log_{10}$ IU/mL HBsAg reduction with considerable heterogeneity

Poly AC Oligonucleotides Have Clinical Validation

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 - › Mean (SD) $\log_{10}$ reduction: 1.65 (1.97) IU/mL
 - Delayed HBsAg response in significant proportion of subjects



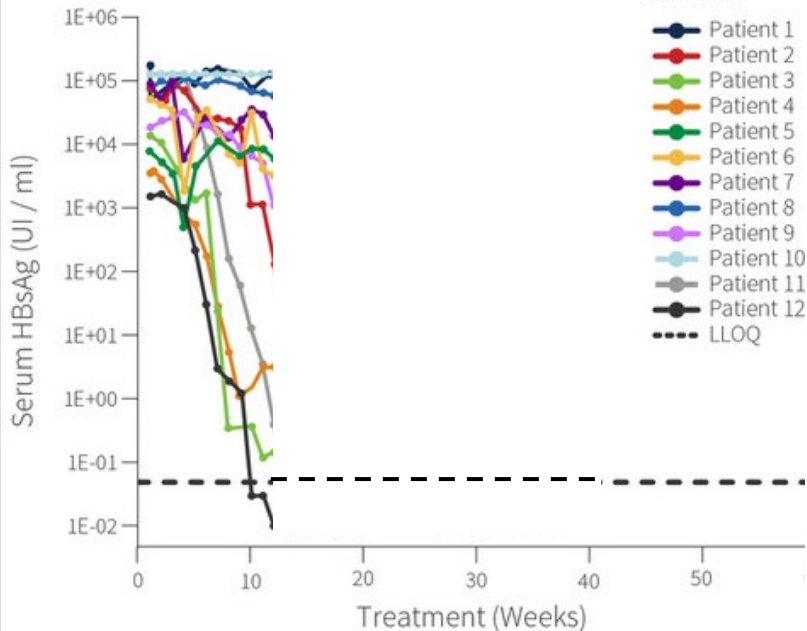
REP-2139 monotherapy x ≥ 20 weeks: mean 3.4 $\log_{10}$ IU/mL HBsAg reduction with considerable heterogeneity

Oligonucleotides HBsAg Reductions Over Time

NAPS

REP 2139

Weekly 500 mg SC doses x 10 weeks

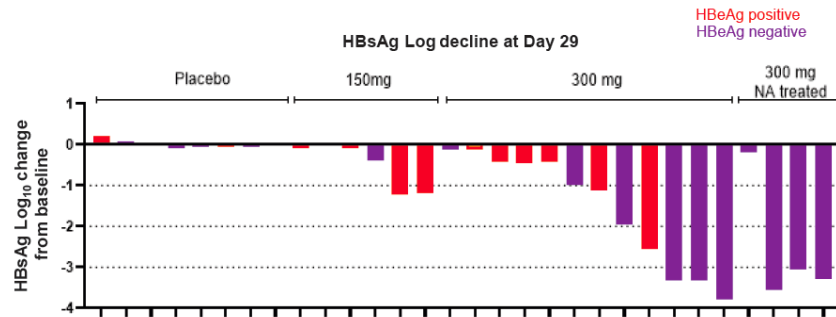


Mean HBsAg reduction: $1.65 \pm 2.0 \log_{10}$ IU/mL

ASO

GSK3228836

7 doses across 29 days (Days 1,4,8,11,15, 22, 29)

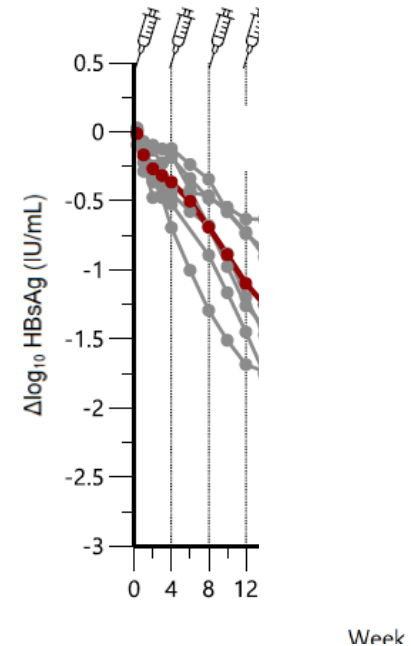


Mean HBsAg reduction: 1.6 ± 1.4 (naïve) – 2.0 ± 1.8 (NA) $\log_{10}$ IU/mL

siRNA

AB-729

AB729 60 mg Q4 x 12 weeks



Mean HBsAg reduction: $\sim 1.1 \log_{10}$ IU/mL

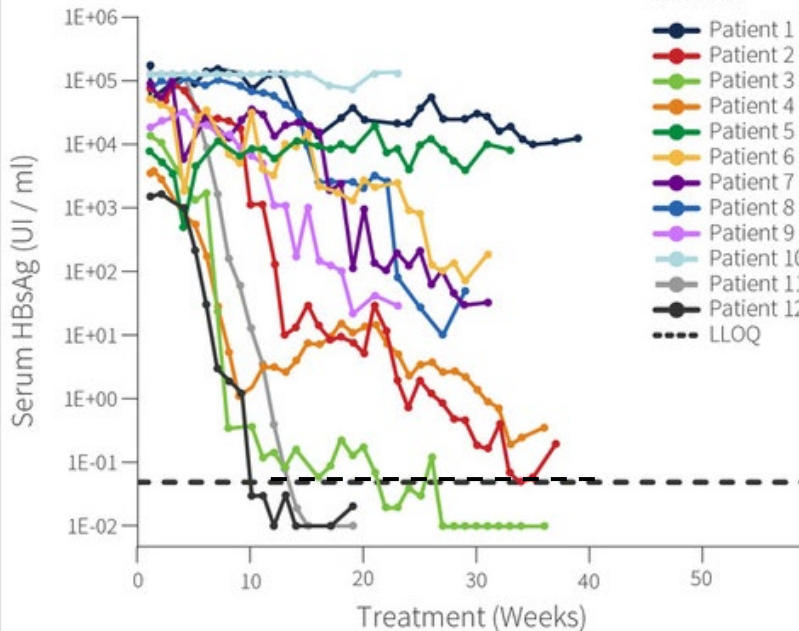
12 weeks is sufficient time to establish proof of concept (HBsAg reduction) for an oligonucleotide
Oligonucleotides with evidence of activity typically observe $\geq 1.0 \log_{10}$ IU/mL reduction within 12 weeks

Oligonucleotides HBsAg Reductions Over Time

NAPS

REP 2139

Weekly 500 mg SC doses x ≥ 20 weeks

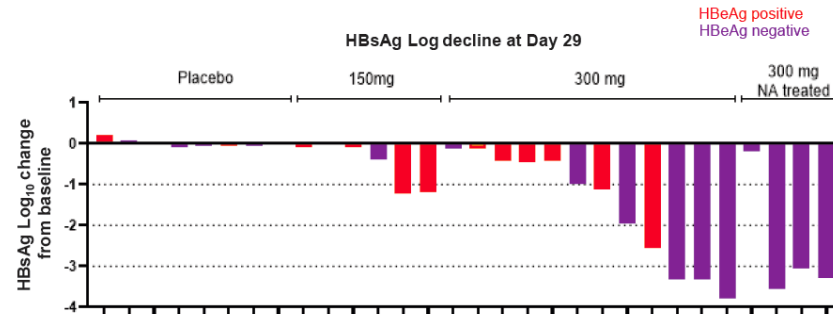


Mean HBsAg reduction: $3.4 \pm 2.3 \log_{10}$ IU/mL

ASO

GSK3228836

7 doses across 29 days (Days 1,4,8,11,15, 22, 29)

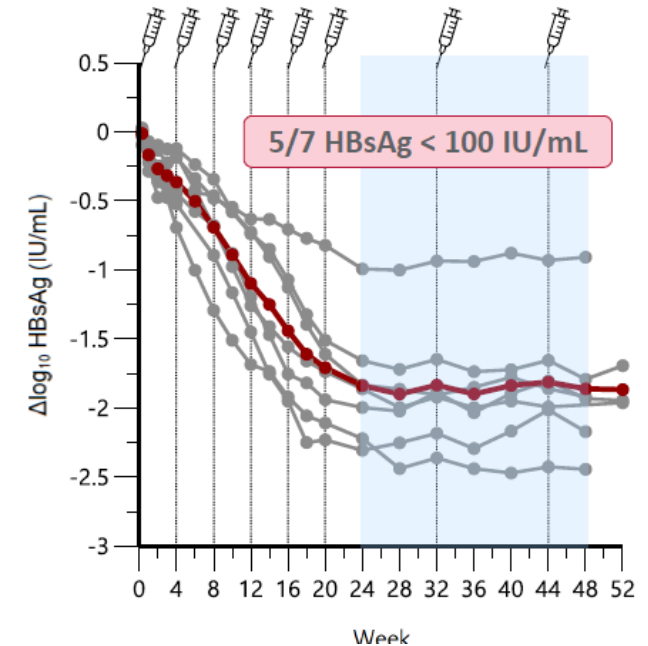


No Data beyond 29 days

siRNA

AB-729

AB729 60 mg Q4-12W x 48 weeks



Mean HBsAg reduction: $1.9 \log_{10}$ IU/mL

Additional HBsAg declines

NAPS: $\sim 1.8 \log_{10}$ IU/mL (beyond 1st 10 weeks); ASO: no data; siRNA: $\sim 0.8 \log_{10}$ IU/mL (beyond 1st 12 weeks)

ALG-010133

Phase 1 Study in CHB Subjects

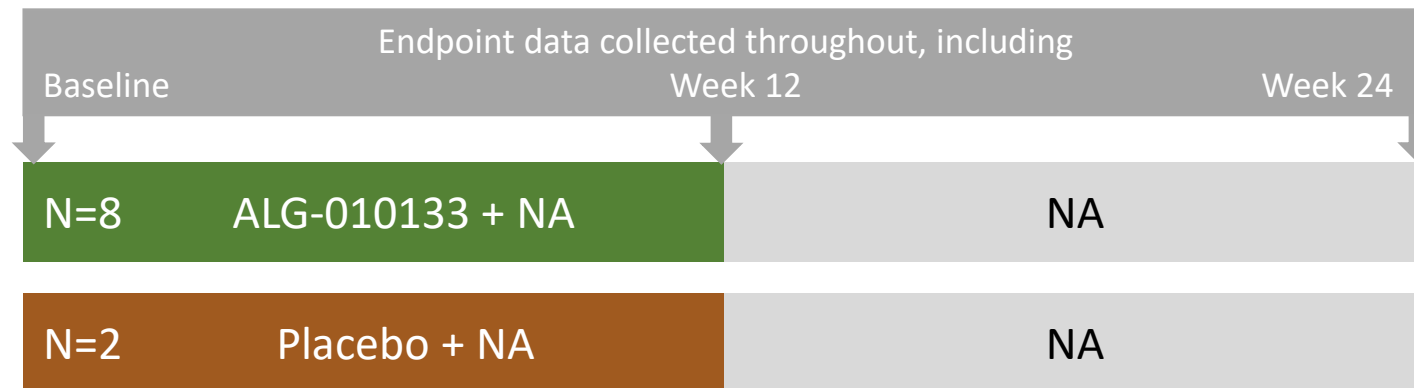
Multiple Doses in CHB Subjects

NA Suppressed (DNA < LLOQ), HBeAg neg or pos, HBsAg >1000 IU/mL

Up to 6 cohorts (n=10 per Cohort; n=8 ALG-010133, n=2 Placebo)

Up to 12 weekly doses

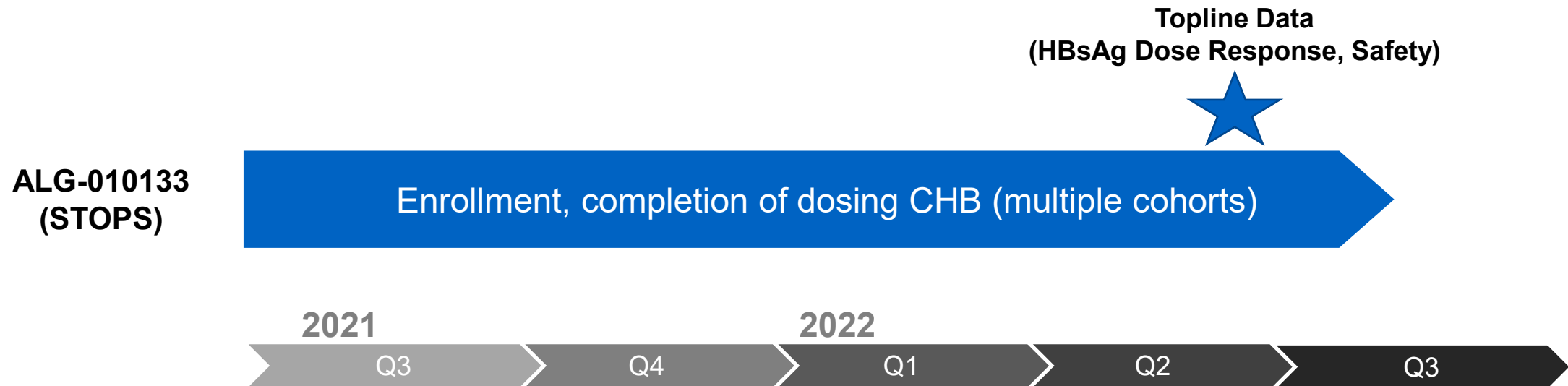
Endpoints: PK, safety, antiviral activity (e.g., HBsAg)



1st cohort (120 mg) fully enrolled, dosing
2nd cohort (200 mg) enrolling, dosing
Safety, viral kinetic data (multiple cohorts) available H1 2022
Target mean HBsAg reduction: $\geq 1 \log_{10}$ IU/mL

ALG-010133

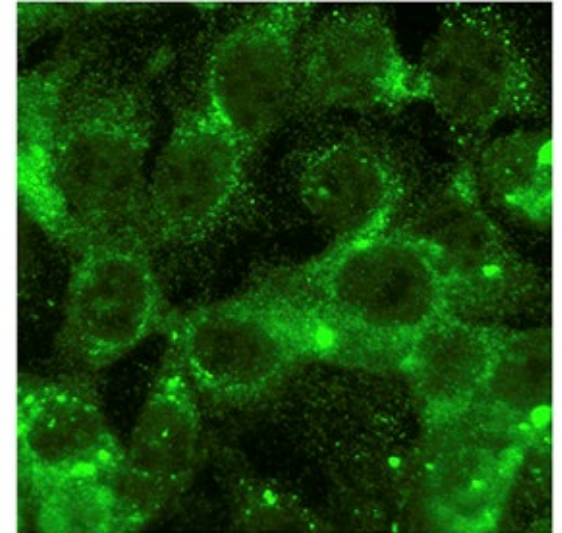
Development Timelines*



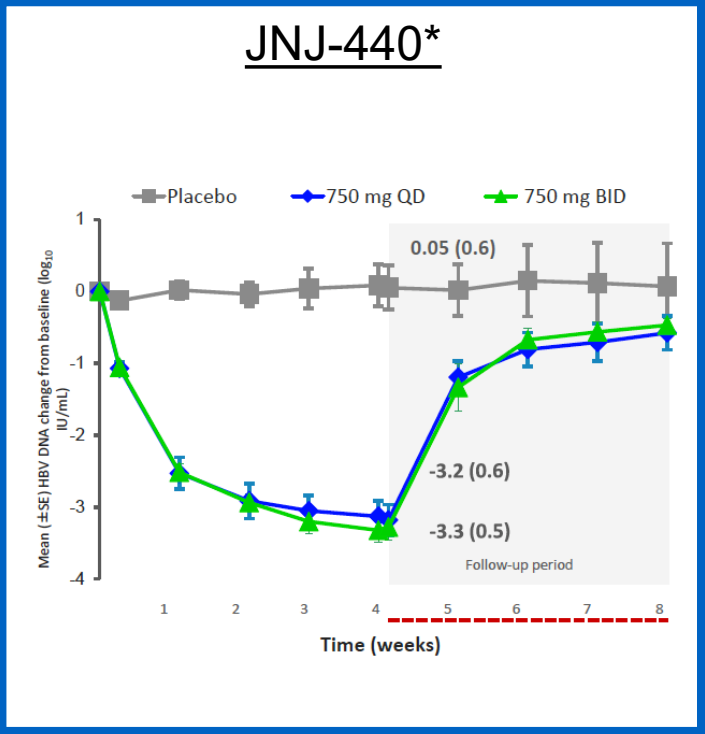
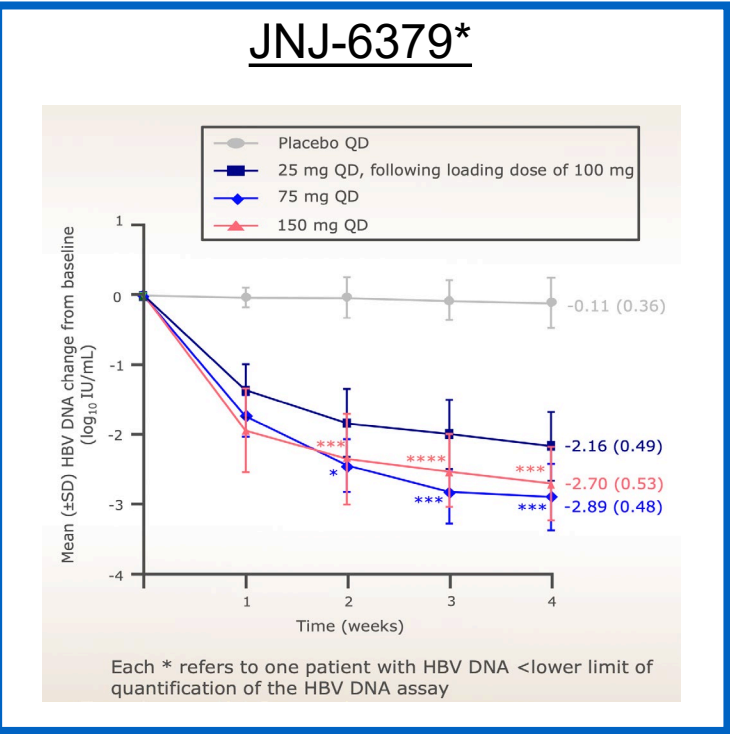
Topline data anticipated Q2 2022

CAM

ALG-000184, a prodrug of ALG-001075



Class-II CAMs Have Clinical Validation



ABI-731

		Change from Baseline Log ₁₀ HBV DNA (IU/mL) At Day 28			
Patients		HBeAg Pos		HBeAg Neg	
Dose (mg)	N	Mean (Range)		N	Mean (Range)
100 mg	6	1.3 (0.8 - 1.7)		4	2.2 (0.7 - 3.6)
200 mg	5	1.9 (1.0 - 2.6)		5	2.4 (1.5 - 3.8)
300 mg	6	2.9 (1.8 - 3.9)		3*	2.5* (0.8 - 4.1)

*Excludes subject with known resistance at baseline

Class-II CAMs result in ~2.5-3 log₁₀ IU/mL reductions in DNA at Day 28 in treatment naïve HBeAg negative CHB patients

ALG-000184 Demonstrates ~10 to 300-Fold Improvement in In Vitro Potency vs. Other Known CAMs in Clinical Development

Compound	Current Status	HBV DNA reduction (EC ₅₀ nM)	Cell Type
Assembly ABI-H0731	Phase 2	172	AD38
Assembly ABI-H2158	Phase 2	22	AD38
Assembly ABI-H3733	Phase 1	5	AD38
Janssen JNJ-6379	Phase 2	54	HepG2.117
Janssen JNJ-0440	Completed Phase 1	12	HepG2.117
Enanta EDP-514	Phase 1	17	HepG2.115
Arbutus AB-836	Phase 1	10	HepDE19
Aligos ALG-000184	Phase 1	0.63	HepG2.117
		0.53	HepG2.2.15

ALG-000184

Phase 1 Study in HVs

SAD/MAD in HVs

N=48; n=8 per Cohort, n=6 ALG-000184, n=2 Placebo
Up to 7 oral daily doses

SAD

Follow-up

Safety and PK of single doses of 40, 100, 250 and 500 mg

MAD

Follow-up

Safety and PK of 7 doses of 150 and 250 mg

Results

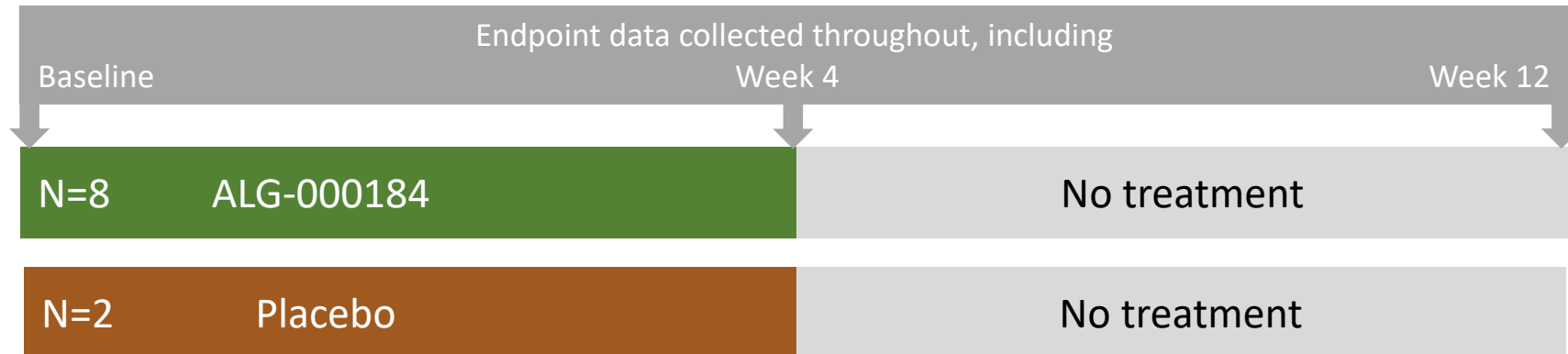
Safety: all doses well tolerated – no SAEs, TEAEs leading to discontinuation
PK: linear; no food effect; no ethnic differences; doses ≥ 100 mg anticipated to have antiviral activity

ALG-000184

Phase 1 Study in CHB Subjects

Multiple Doses in Currently Not Treated/Treatment Naïve CHB Subjects

HBV DNA > 2000 IU/mL, HBeAg Negative
Up to 6 cohorts (n=10 per Cohort, n=8 ALG-000184, n=2 Placebo)
28 daily oral doses
Endpoints: PK, safety, antiviral activity (e.g., DNA, RNA)



ALG-000184 CHB Cohort 1 (100 mg)

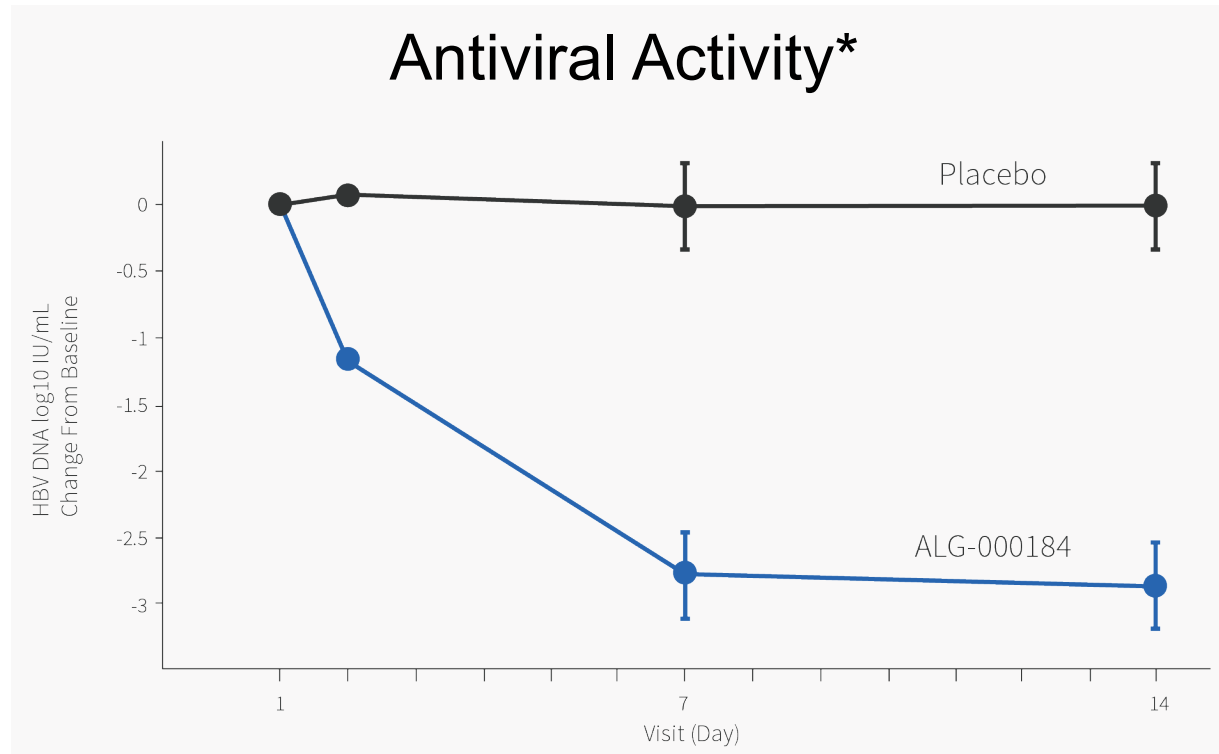
Safety, PK Data

- Safety
 - No serious adverse events
 - No TEAEs leading to discontinuation
 - All TEAEs were mild except one case of moderate back pain (unlikely related to study drug)
 - › Most common TEAEs: back pain, dry mouth & headache (2 subjects each); nausea (3 subjects)
 - No concerning laboratories, EKGs, or vital signs
- PK (Day 1)
 - CHB PK similar to HV PK

100 mg ALG-000184 x 14 days was well tolerated

ALG-000184 CHB Cohort 1 (100 mg) Day 14 Antiviral Activity* Data

Mean (SD) Baseline HBV DNA
4.2 (1.0) log₁₀ IU/mL

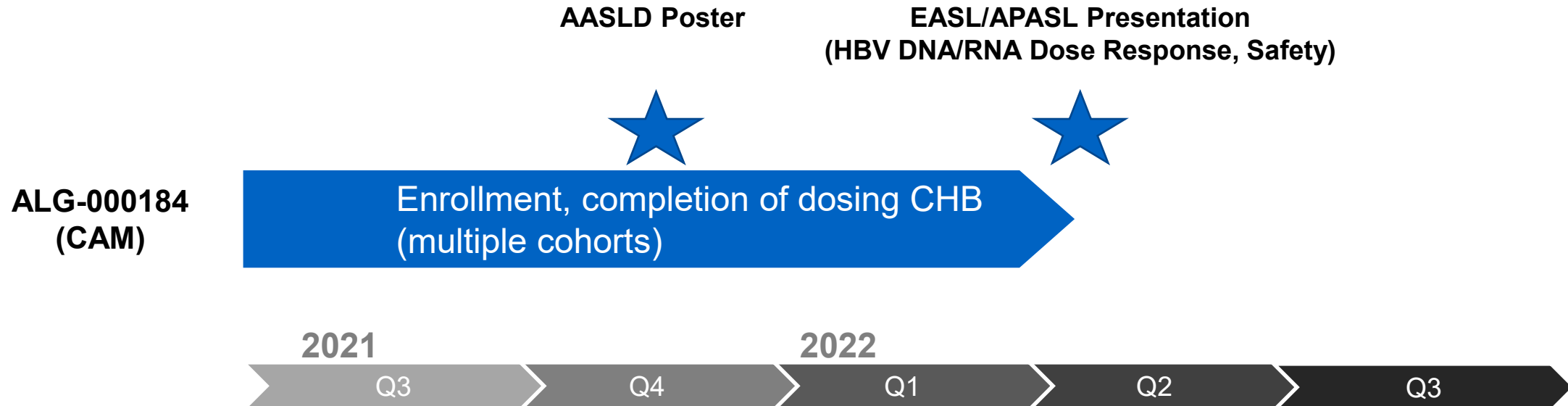


Mean (SEM) HBV DNA reduction: 2.9 (0.2) log₁₀ IU/mL
4 of 8 (50%) ALG-000184-treated subjects <LLOQ

1st cohort (100 mg) has completed dosing, 2nd cohort (50 mg) currently enrolling, dosing
Safety, antiviral activity data through Day 28 from 1st cohort(s) in H2 2021

ALG-000184

Development Timelines*



Topline data targeted for presentation at EASL/APASL (Q2 2022)

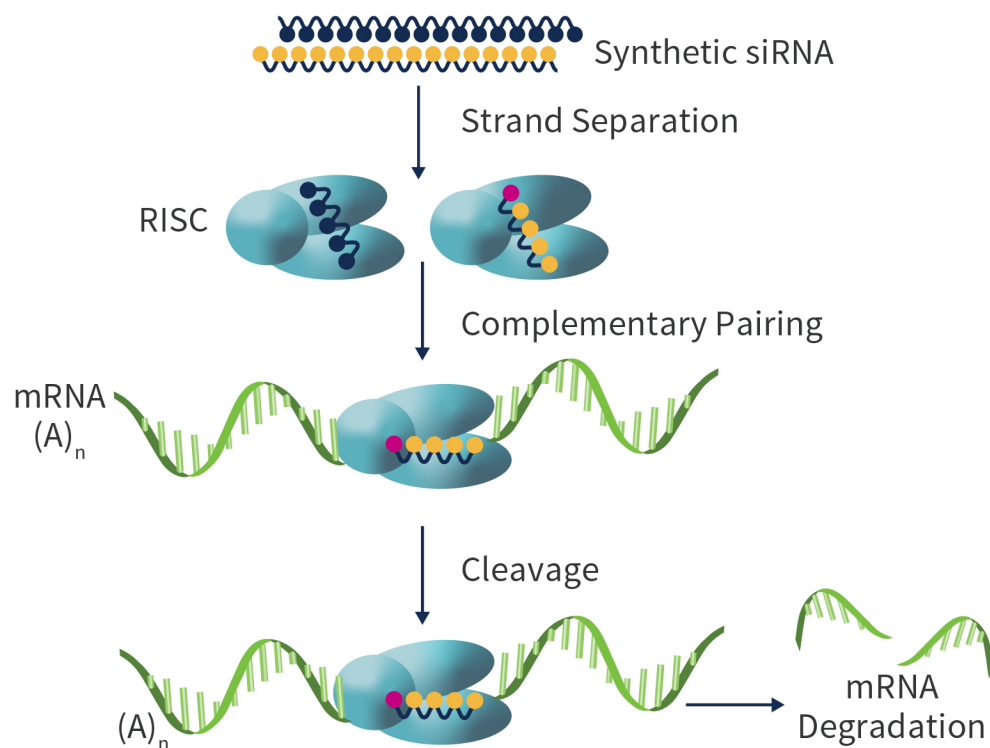
Additional Oligonucleotide Approaches

- ALG-020572 (ASO)
- ALG-125755 (siRNA)

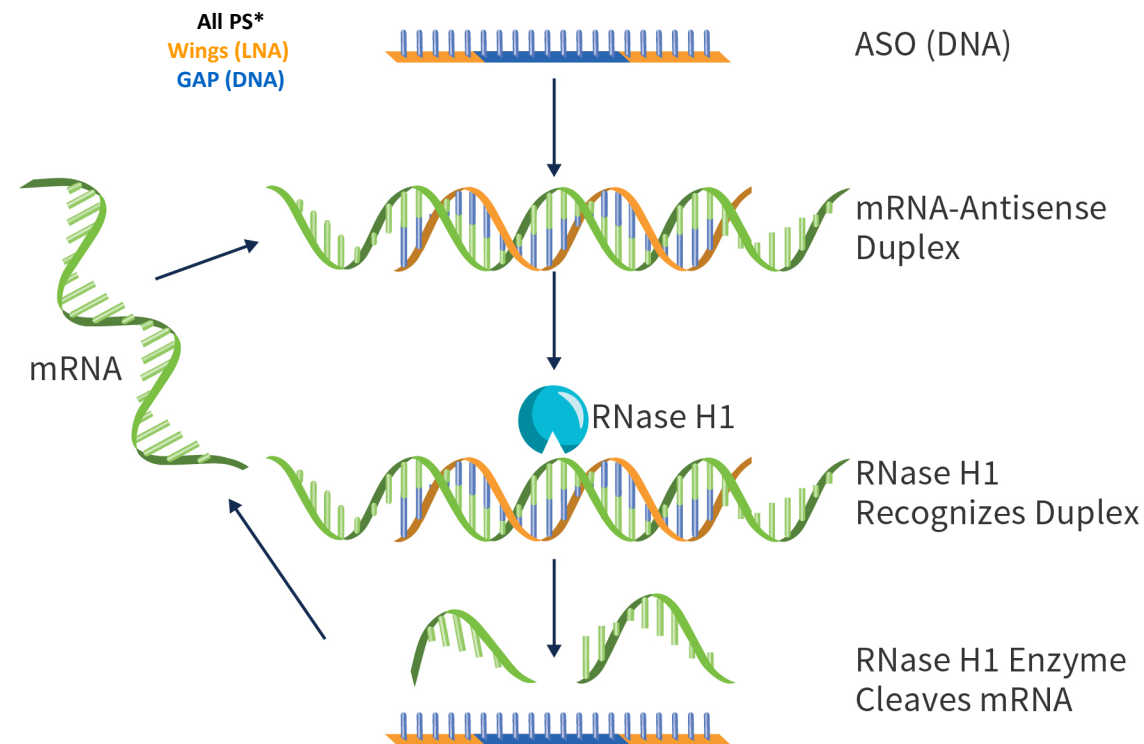


Antisense Oligonucleotides and Small-Interfering RNAs

RNA interference (RNAi)



Antisense oligonucleotides (ASOs)

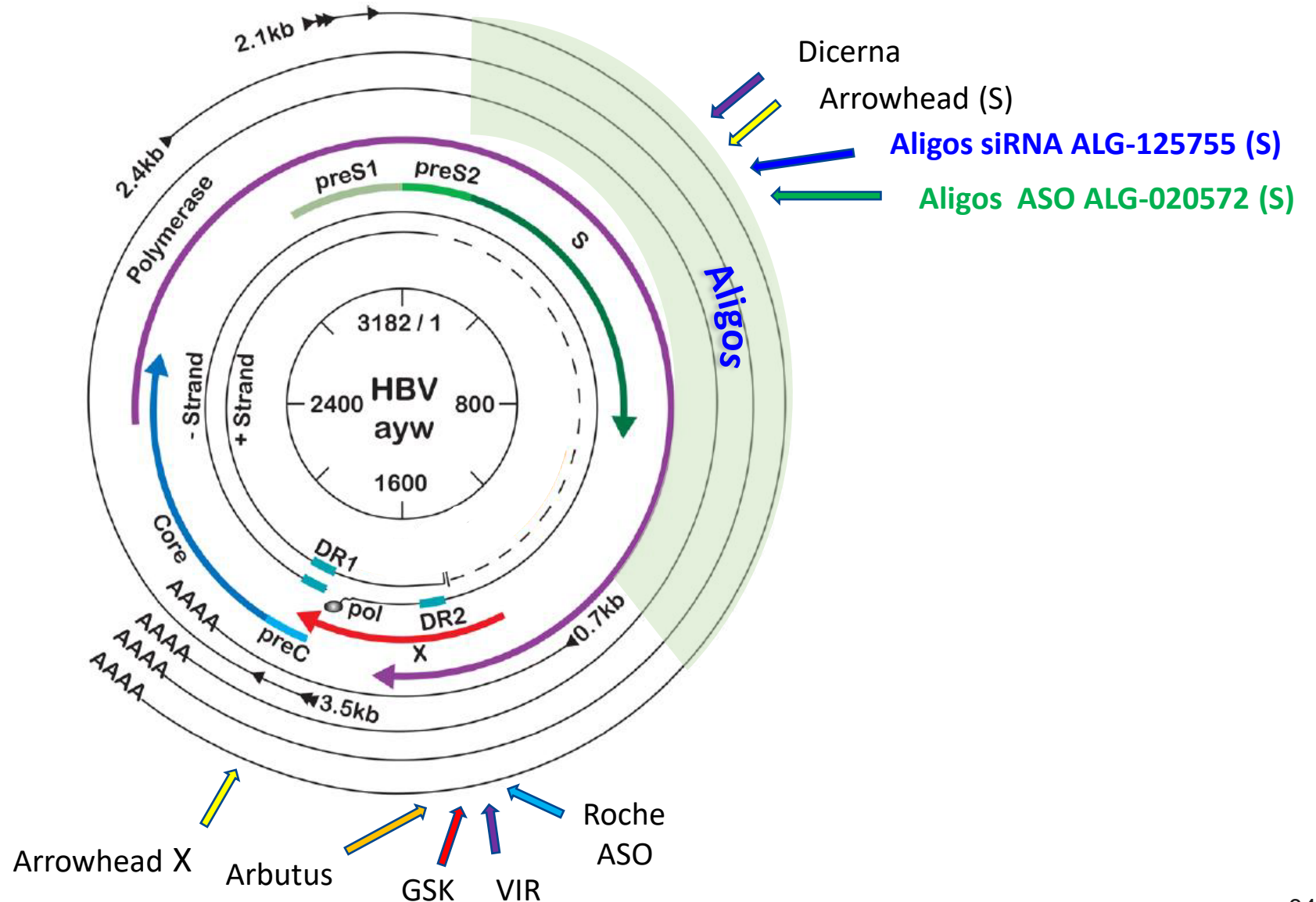


The Aligos team is agnostic to oligonucleotide modality

Map of HBV Trigger Locations

ASO ALG-020572 and siRNA ALG-125755

HBV ASO ALG-020572 (S)
and siRNA ALG-125755 (S)
trigger locations are >100
nucleotides apart



Antisense Oligonucleotide ALG-020572

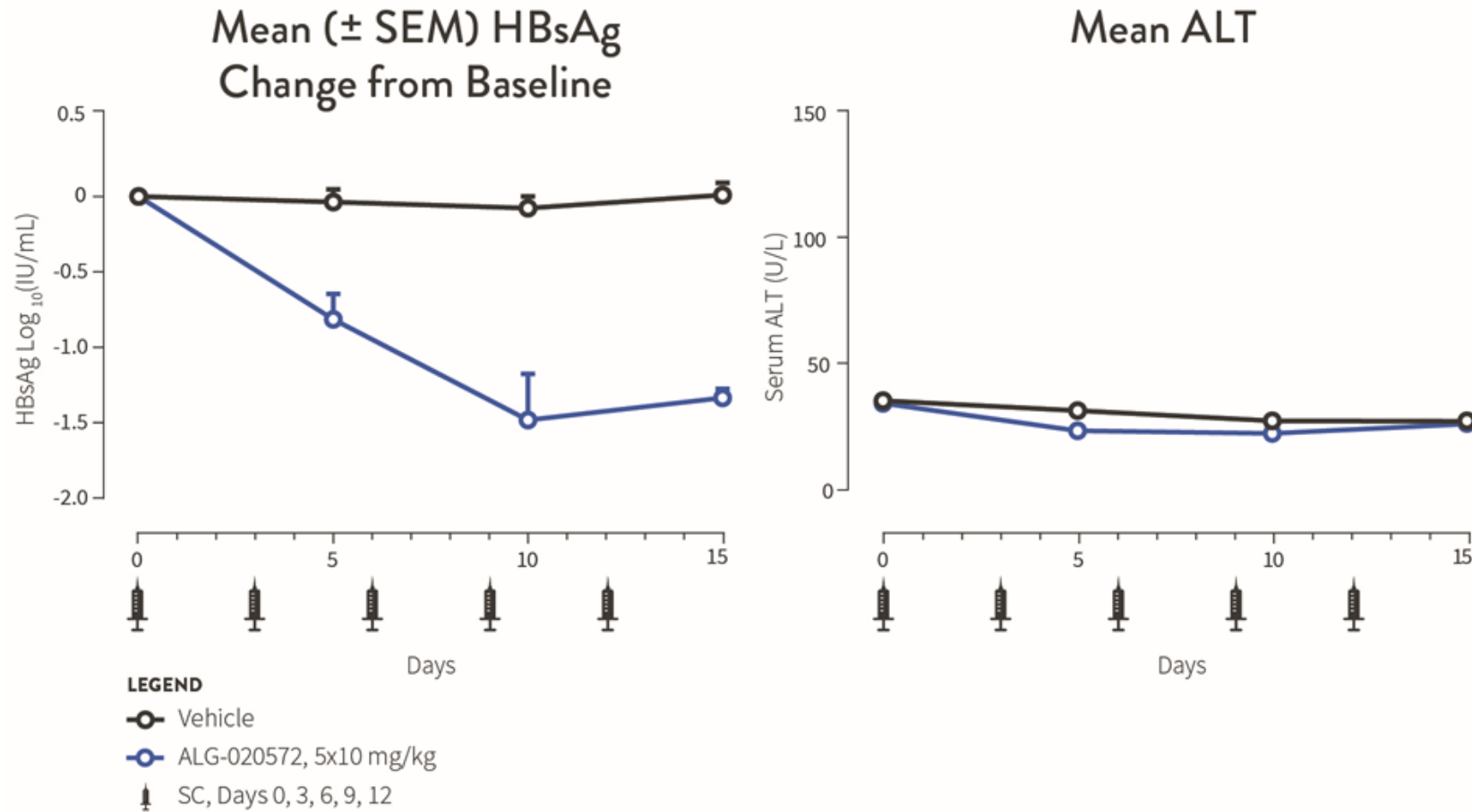
Discovery and Advancement of a Differentiated ASO

- Antisense oligonucleotides have demonstrated clinical validation in CHB infected patients
- We have designed our sequences using our proprietary Luxna Biotech 3rd generation XNA nucleotides to enhance potency, stability and, potentially, safety
 - Our ASO has been conjugated to our proprietary GalNAc ligands for targeted liver delivery
- ALG-020572 demonstrates potent suppression of HBsAg in vivo in the AAV-HBV mouse model of HBV infection
- CTA filed seeking approval for first-in-human clinical studies with ALG-020572

ALG-020572 is differentiated from potential competitors and is progressing towards clinic entry in 2H 2021

ALG-020572

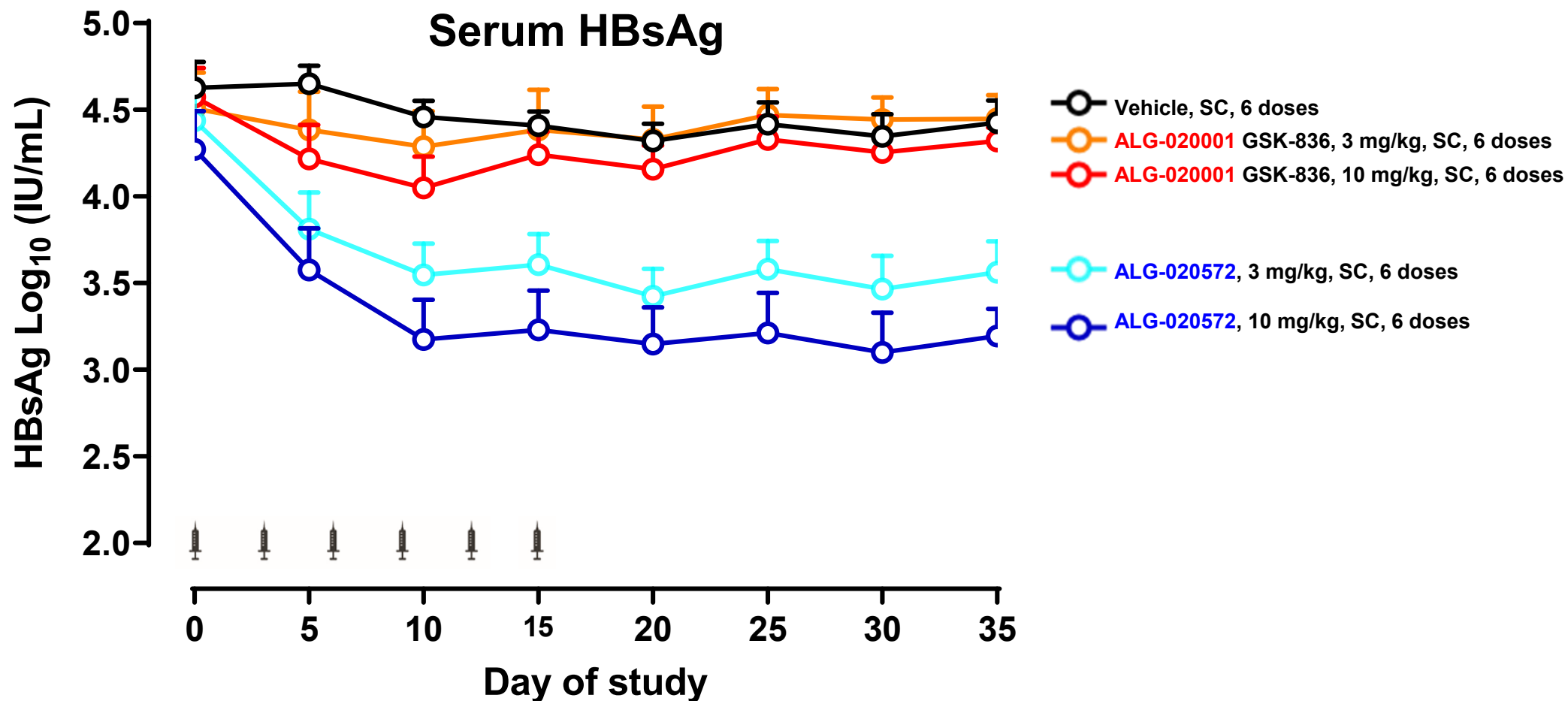
Our ASO Drug Candidate in the AAV-HBV Mouse Model



Potent in vivo activity of ALG-020572 in the AAV-HBV mouse model without ALT elevation

ALG-020572

Our ASO vs. GSK-836 ASO in the AAV-HBV Mouse Model



ALG-020572 demonstrates significantly improved efficacy in vivo vs. a competitor ASO

ALG-020572

Phase 1a/b Clinical Trial Design

Part 1: SAD (HV)

Multiple Cohorts

Each Cohort - 6 Active: 2 Placebo

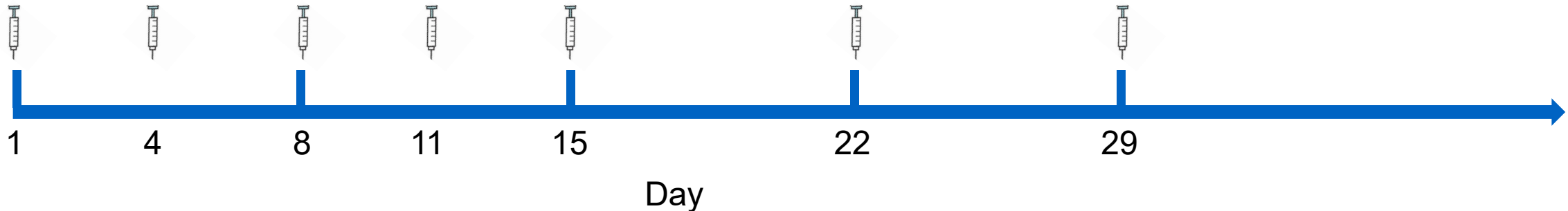
Part 2: VS CHB patients

Multiple Cohorts

Each Cohort - 6 Active: 2 Placebo



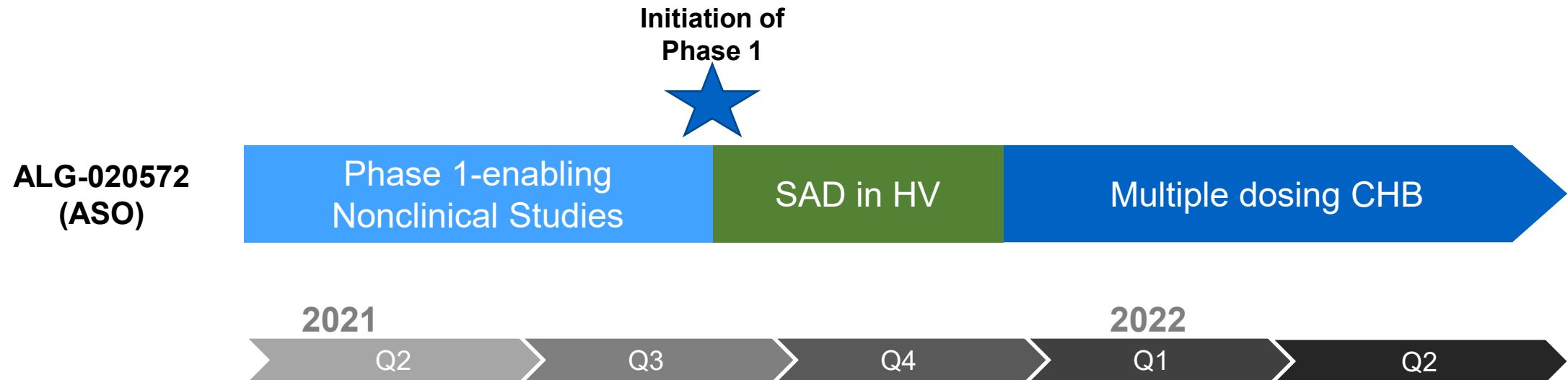
Loading Doses



Phase 1 target mean HBsAg reduction: $\geq 1 \log_{10}$ IU/mL

ALG-020572

Development Timelines*



Dosing in CHB subjects (MAD) anticipated to start late Q4/early Q1

Short Interfering Nucleic Acid ALG-125755

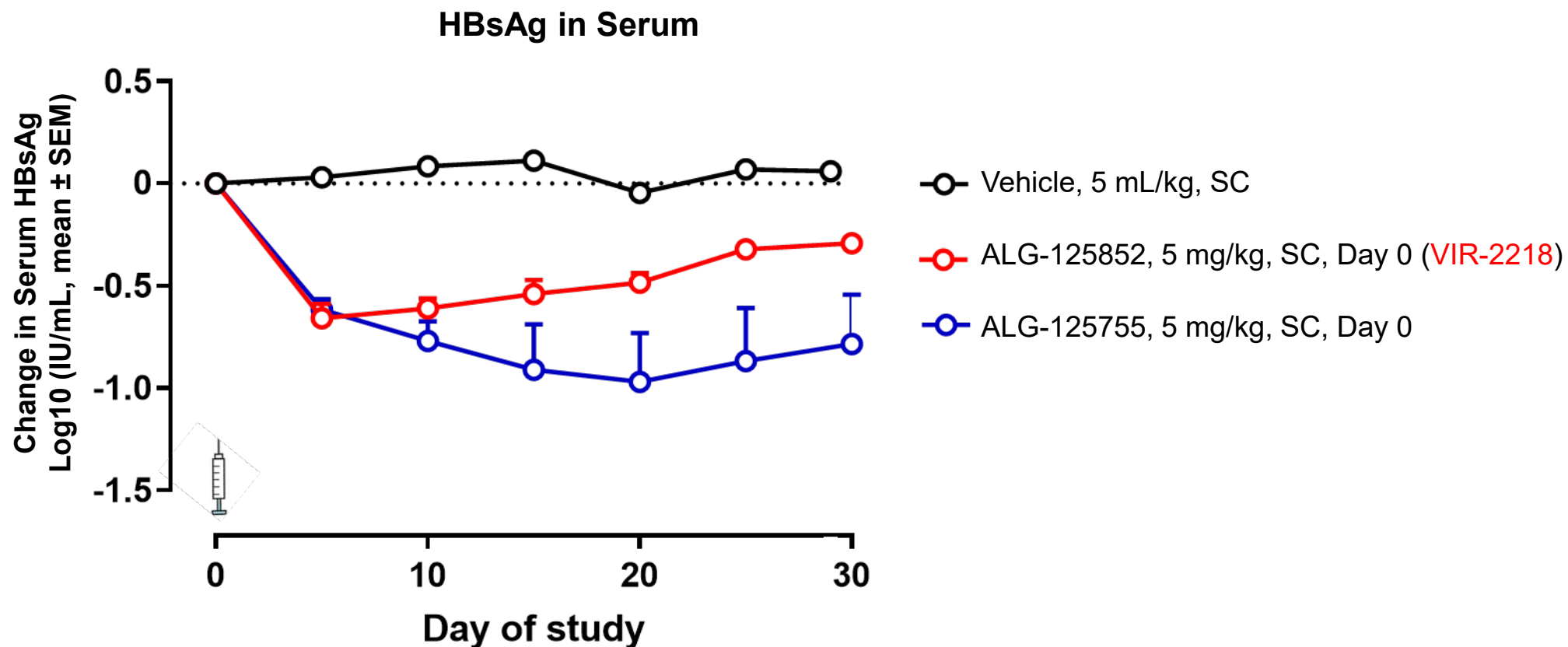
Discovery and Advancement of a Differentiated siRNA

- siRNAs have demonstrated clinical validation in CHB infected patients
- We have designed our siRNA sequences using our proprietary technology and liver targeting conjugation to maximize in vitro and in vivo potency
 - Novel monomers to stabilize antisense strand in the RISC complex
 - Proprietary patterns discovered to increase potency and stability / duration of action
 - Exclusive license to GalNAc technology applicable for liver targeting across oligo modalities
- Our approach to developing siRNAs may have safety, stability and potency advantages over other siRNAs
- We have initiated dose-range-finding studies with our siRNA ALG-125755
 - First-in-human enabling GLP studies are planned for 2H 2021

ALG-125755 is differentiated from potential competitors and is progressing towards clinic entry in 1H 2022

ALG-125755

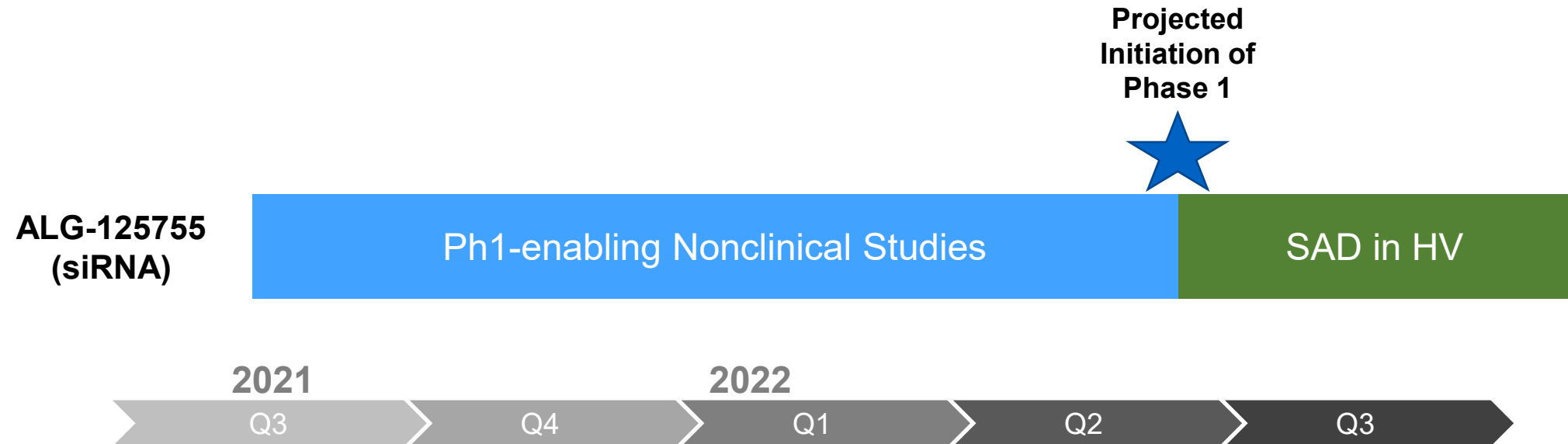
Our siRNA vs. VIR-2218 siRNA in the AAV-HBV Mouse Model



ALG-125755 demonstrates significantly improved efficacy in vivo vs. a competitor siRNA

ALG-125755

Development Timelines*

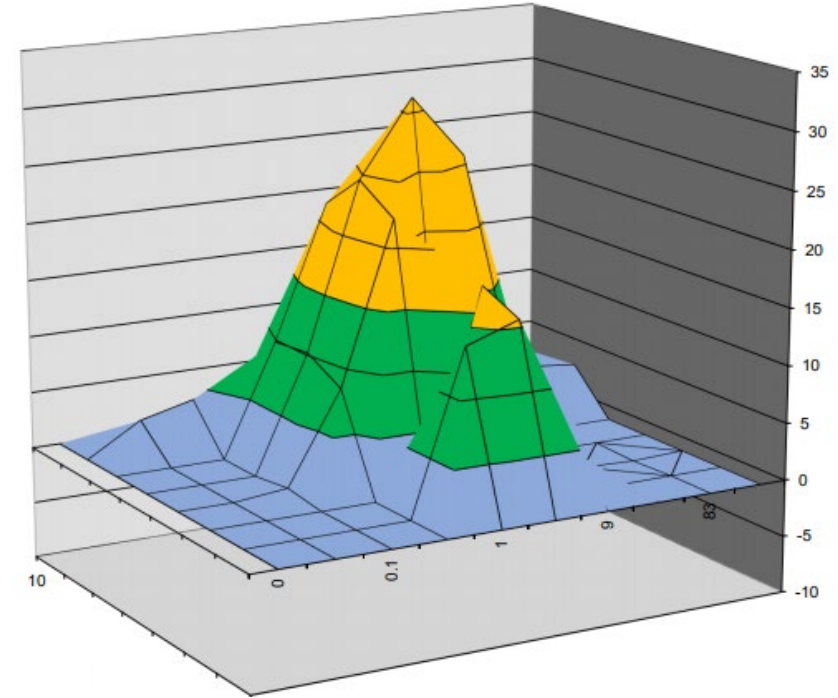


Phase 1 target mean HBsAg reduction: $\geq 1 \log_{10}$ IU/mL

Aligos' Advancing CHB Portfolio

In Vitro Combination Studies in Cell Based Assays

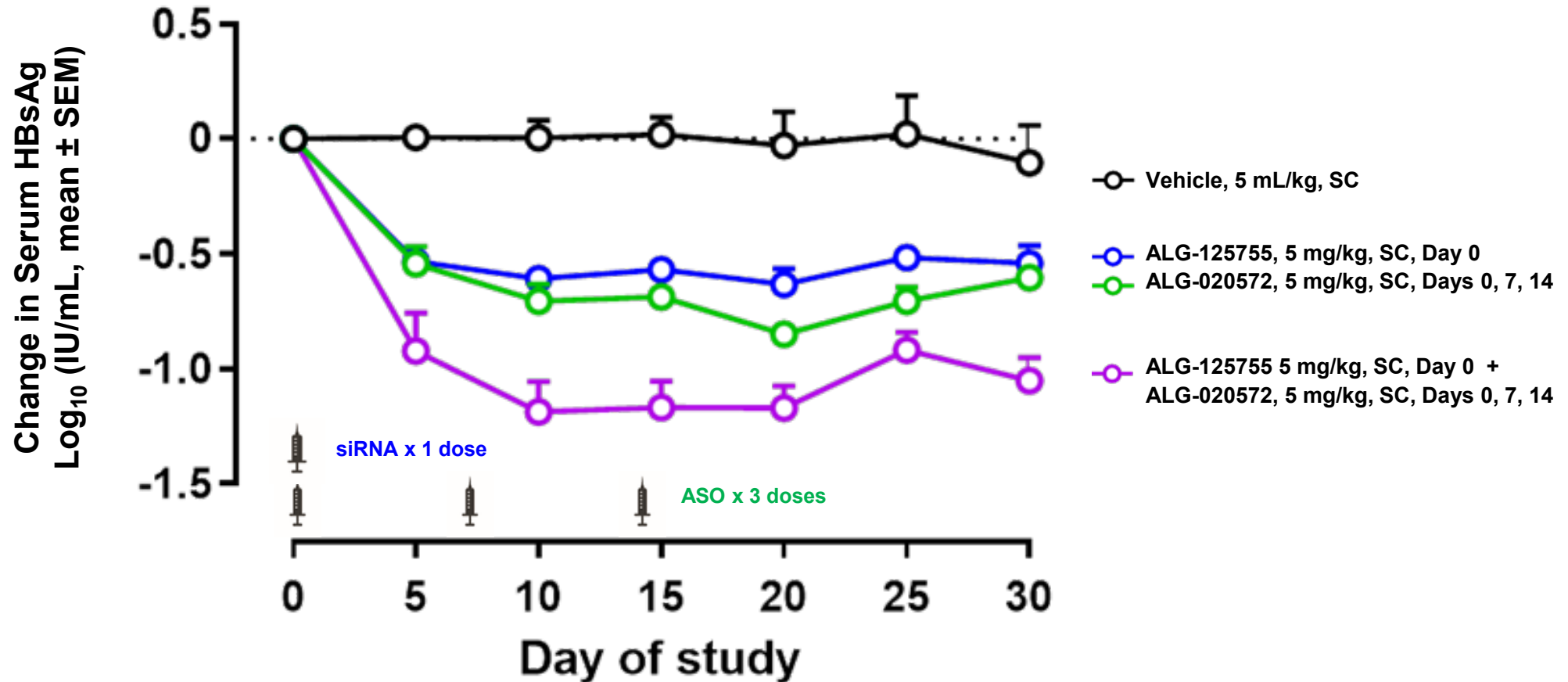
- STOPS ALG-010133 + ASO ALG-020572
(Unconjugated form)
 - Synergistic (HBsAg)
- STOPS ALG-010133 + CAM ALG-000184
(ALG-001075)
 - Synergistic (HBV DNA)
- siRNA ALG-125755 + ASO ALG-020572
(Unconjugated versions)
 - Synergistic (HBsAg)



Aligos' CHB development compounds are synergistic with each other in combination studies

ALG-125755 + ALG-020572

In Vivo Combination Study in the Mouse AAV-HBV Model



The combination of our siRNA and ASO delivers rapid, potent suppression of HBsAg in vivo

NASH

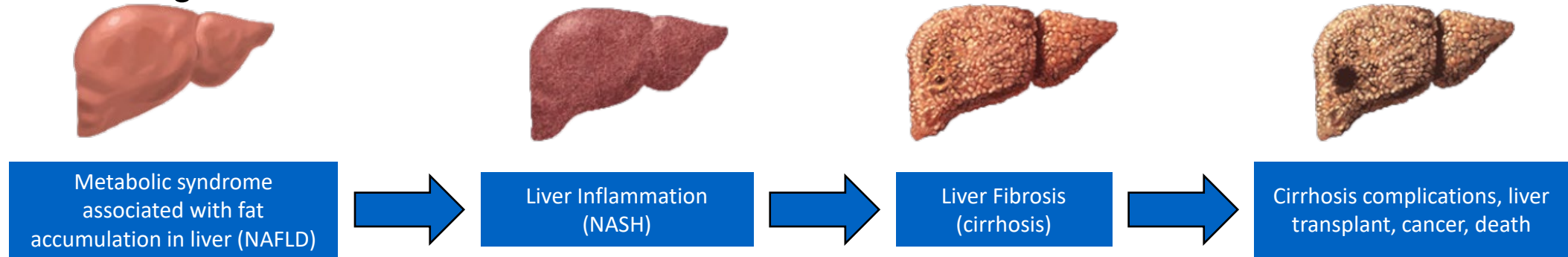
ALG-055009, small molecule THR- β agonist



NAFLD and NASH*

High Prevalence + No Approved Therapy: Large Unmet Need

- Disease Progression



- Incidence

- NAFLD – 25% worldwide
- NASH – 1.5-6.5% (~450 million people) worldwide
 - › Projected to affect 27 million Americans by 2030**

- Impact

- Causes significant morbidity and mortality

- Treatment

- Diet and exercise – effective but difficult to achieve at population level
- Drugs – no approved therapy

THR- β Agonists are Clinically Validated

THR- β Drug [^]	Serum LDL Lowering [*]	Liver Fat Reduction at 12 Weeks [*] (MRI-PDFF)	NASH Resolution Without Fibrosis Worsening [*] at 36 weeks (Liver Biopsy)
Resmetirom ^{**}	17.3% (30 weeks)	22.5%	18.2%
VK-2809 ^{***}	21.8% (12 weeks)	49.2%	—

THR- β agonists have demonstrated beneficial effects on serum lipid levels and liver histology

[^]All dose levels pooled.

^{*}Placebo corrected values.

^{**}In patients with NASH, Harrison, Lancet, 2019. Resmetirom = MGL-3196.

^{***}In patient with NAFLD, hyperlipidemia, Loomba, AASLD, 2018.

LDL: Low Density Lipoprotein, MRI-PDFF: magnetic resonance imaging proton density fat fractionation.

ALG-055009

More Potent and Selective In Vitro than MGL-3196 and VK-2809

Relative THR-α and THR-β Activity in Cell-Based Assays

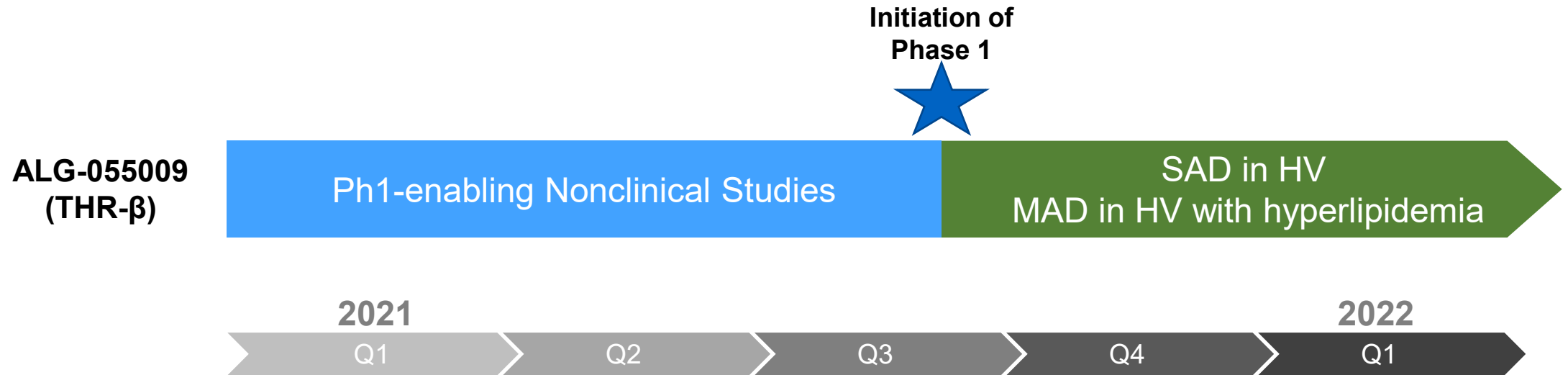
	EC ₅₀ α (nM)	EC ₅₀ β (nM)	Relative THR-β Selectivity (α/β)
T3	14.2	11.6	1.2
MGL-3196	5927	2366	2.5
VK-2809 parent	366	269	1.4
ALG-055009	191	50	3.8

In vitro, ALG-055009 is 5-47x more potent compared to resmetirom (MGL-3196) and 2-3x more selective for THR-β than VK-2809*

High β selectivity and potency may improve risk-benefit profile

ALG-055009

Development Timelines*

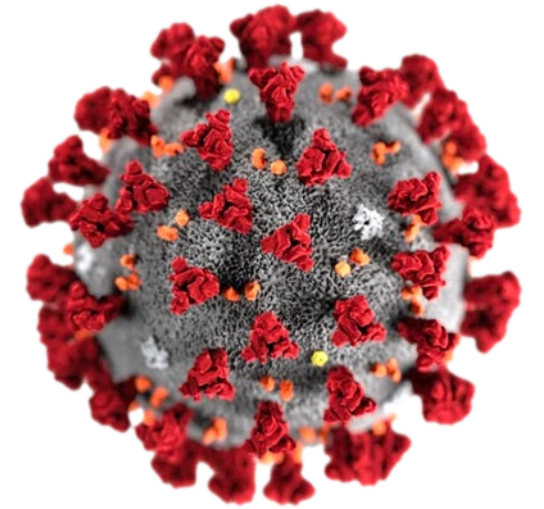


Lipid Reduction Target
Exceed Madrigal/Viking Phase 1 results at Phase 2b/3 dose levels
Mean placebo-corrected LDL-C reduction $\geq 27\%$

Our NASH Strategy

- Advance ALG-055009 through Phase 1 proof of concept study
 - Benchmark safety, PK, anti-lipid data vs. resmetirom & VK2809 Phase 1 data
 - Data expected 2H 2022
- Resmetirom Phase 3 readouts are expected in Q3 2022
- Aligos NASH ALG-055009 program has strong optionality in 2022
 - Continue to advance program on our own into Phase 2
 - If enthusiasm for resmetirom and/or VK-2809 is high and ALG-055009 compares favorably in Phase 1, we expect to seek partnership with a company that is advancing a NASH asset with a complimentary MOA

Coronavirus



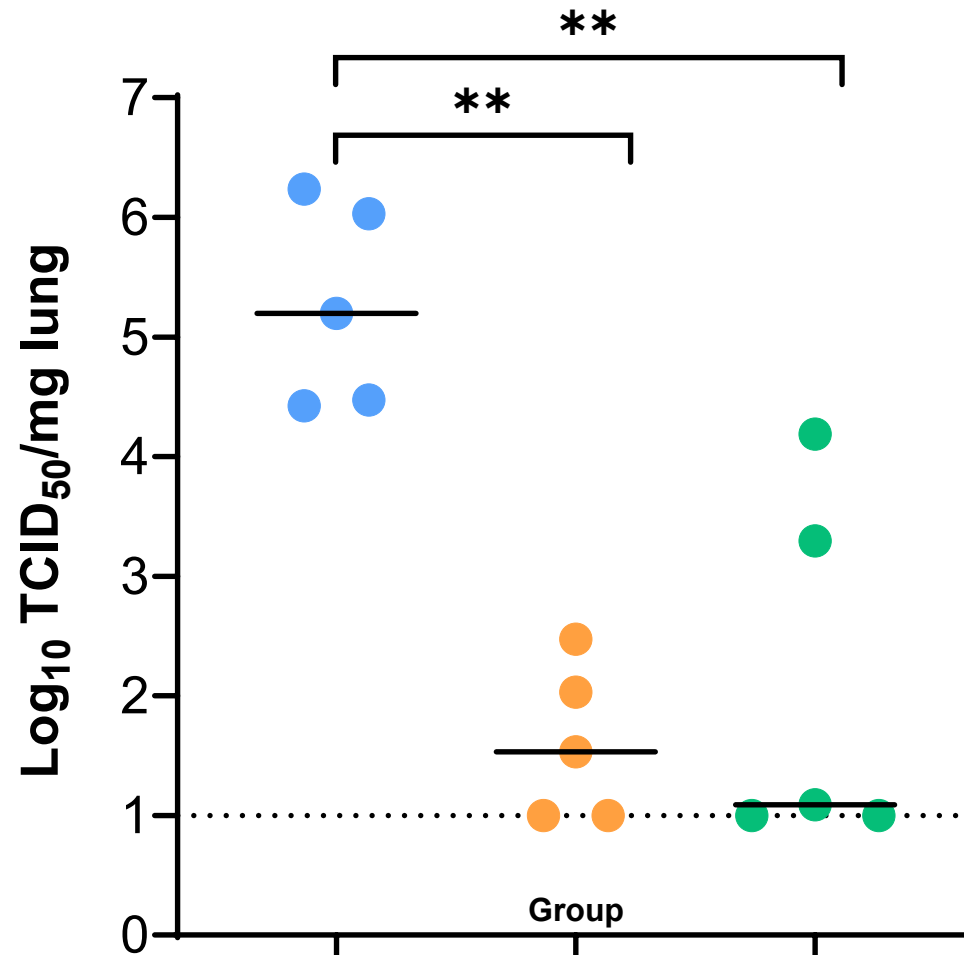
Our Approach to Developing a Broad Coronavirus Treatment

- Despite the recent availability of prophylactic vaccines, a need for therapeutics still exists
 - Resistant variants are emerging and may become dominant
 - Large segments of global population lack access to, or are opposed to vaccination
 - Especially needed in the hospitalized patient population
- Leveraging our virology / chemistry expertise to develop purpose-built drugs with distinct MOAs
 - Protease inhibitors (collaboration with KU Leuven / Rega Institute / CD3)
- We are aiming to develop drug candidates that
 - Are broadly active against a diverse range of coronaviruses with a high barrier to resistance
 - Have a therapeutic window compatible with prophylaxis and treatment
 - Can be combined to prevent emergence of resistance and provide broader strain coverage
- We are prioritizing therapeutics with MOAs that may have activity against potential zoonotic coronavirus transmissions in the future
 - Need for drugs with broad pan-coronavirus activity is essential for the management of future pandemics

ALG-097111

Efficacy in SARS-CoV-2 Hamster Model

Infectious viral loads
*in the lungs of
SARS-CoV-2
infected hamsters at
day 2 post-infection
after 5 doses with
either vehicle or
study drugs*



ALG-097111 SARS-CoV-2 In Vitro

Biochemical IC₅₀ = 7 nM
Cell-based EC₅₀ = 200 nM (A549)

- **RTV + Vehicle**
60% PEG400 in water, subcutaneous(SC) +
ritonavir (50 mg/kg/dose, BID oral gavage)
- **RTV + ALG-097111**
(200 mg/kg/dose, BID, SC) + ritonavir
(50 mg/kg/dose, BID, oral gavage)
- **EIDD-2801**
(200 mg/kg/dose, BID, oral gavage)

ALG-097111 efficiently inhibits viral replication in the lungs of infected hamsters

Aligos Therapeutics Summary

- We are advancing multiple drug candidates with clinically-validated MOAs with the goal of rapidly developing optimized combination regimens
 - CHB
 - › STOPS molecule (ALG-010133) Phase 1 clinical trial ongoing, currently dosing in 2 CHB cohorts (120, 200 mg)
 - › CAM (ALG-000184) Phase 1 clinical trial ongoing, currently dosing in 2nd CHB cohort (50 mg)
 - Well tolerated, demonstrated a mean 2.9 log₁₀ IU/mL HBV DNA reduction (100 mg QD at Day 14)
 - › ASO, siRNA oligonucleotide compounds are planned to advance into the clinic (2H 2021, 1H 2022)
 - NASH
 - › We are advancing our small molecule THR-β agonist, ALG-055009, into clinical development 2H 2021
 - › We have an exclusive oligonucleotide-based License and Research collaboration with Merck
 - Coronavirus
 - › We have identified multiple nM potent coronavirus protease inhibitors
- As of June 30, 2021, our cash balance was \$190.7M*
- As of July 6, 2021, \$77.9M net proceeds from follow-on offering**
- As of August 4, 2021, common stock shares outstanding: 42,608,792

ALIGOS

THERAPEUTICS

