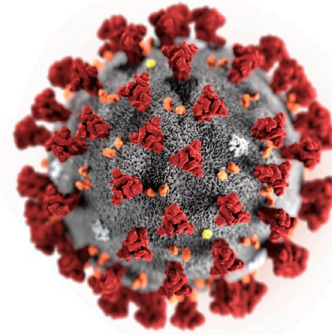




Lawrence M. Blatt, Ph.D.
Chairman, CEO & Co-Founder

Corporate Presentation
September 2022

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 and the ongoing conflict between Ukraine and Russia 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 4, 2022, 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 Development Portfolio

Multiple Data Readouts Planned Throughout 2022

Candidate	Indication	MOA	2022-3 Clinical Trial Timelines* & Data Readouts			
			Q2	Q3	Q4	Q1
ALG-000184	CHB	CAM-II	28 days in CHB	Dosing x 12-weeks in CHB		
					★ AASLD (28 days CHB)	
ALG-125755	CHB	siRNA	Phase 1 enabling nonclinical studies		SAD in HV + CHB	
				★ FIH HV		
ALG-055009	NASH	THR-β Agonist	MAD (hyperlipidemia subjects)		★ AASLD (Lipids, PK, safety)	
ALG-097558	Covid-19	Protease Inhibitor	Phase 1 enabling nonclinical studies			SAD in HV
					CTA Filing ★	

*Timelines are approximate and subject to change based on enrollment and operational considerations.

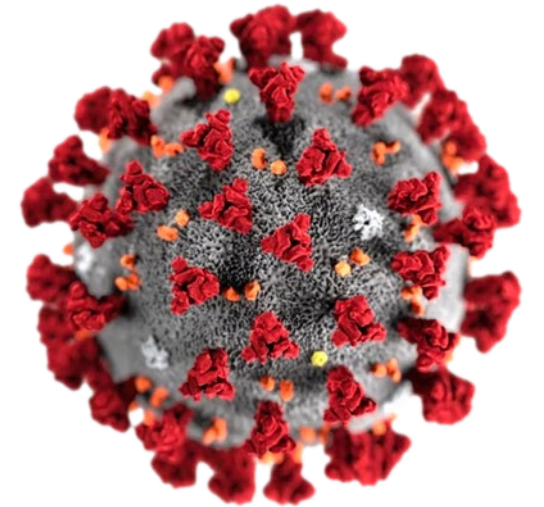
Aligos Discovery Pipeline

Multiple Candidates Advancing Towards Development

Candidate	Indication	MOA	Discovery	Nonclinical	Phase 1	Anticipated Milestones
Discovery	CHB	CAM-I				Compound Nomination
Discovery	CHB	PD-L1				Compound Nomination
Discovery	NASH	Oligonucleotide		 MERCK		Compound Nomination
Discovery	NASH	Oligonucleotide		 MERCK		Compound Nomination
Discovery	Coronavirus	Back-Up Protease Inhibitor				Compound Nomination

Coronavirus

- ALG-097558 (CoV Protease Inhibitor)



ALG-097558

Aligos' Potent COVID-19 Protease Inhibitor

- Despite the availability of prophylactic vaccines, a need for therapeutics still exists
 - Resistant variants are emerging and may become dominant (e.g., Omicron)
 - Large segments of global population lack access to, or are opposed to, vaccination
 - Especially needed in the hospitalized population and to prevent hospitalization in high-risk groups
- Current therapeutics lack sufficient efficacy (molnupiravir, Merck), require ritonavir boosting (nirmatrelvir, Pfizer) or are delivered parenterally (remdesivir, Gilead; mAbs)
- In collaboration with KU Leuven/Rega Institute/CD3, we have identified ALG-097558
 - 7-27 times more potent than nirmatrelvir in both biochemical and cell-based assays
 - Can be dosed orally without the need for ritonavir
 - Broadly active against a diverse range of coronaviruses with a high barrier to resistance
 - Can be combined to prevent emergence of resistance and provide broader strain coverage

ALG-097558 is a potent pan-coronavirus protease inhibitor that does not require ritonavir boosting

ALG-097558

Superior Biochemical Potency Against SARS-CoV-2

SARS-CoV-2 3CLpro	IC ₅₀ (nM) ¹	Hillslope	K _i (nM)
PF-07321332	2.92	0.91	2.03
PBI-0451	3.6	1.74	3.4
S-217622	4.0	1.31	2.6
ALG-097558	0.26	1.99²	0.074

ALG-097558 K_i is 27-46 fold more potent vs. competitors in the 3CLpro biochemical assay

ALG-097558

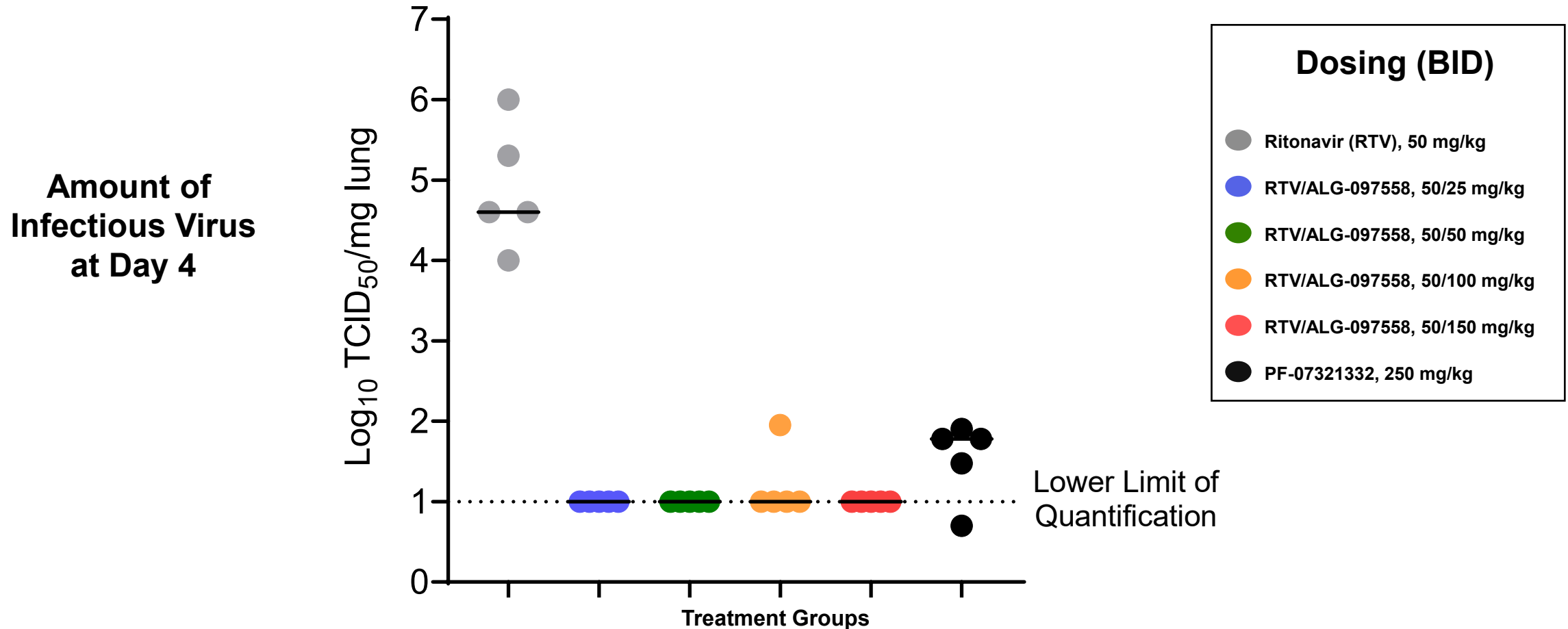
Superior Cell-Based Potency Against SARS-CoV-2 and Variants

	Virus	Variant/Cell line	EC ₅₀ (μM)			
			Pardes PBI-0451	Shionogi S-217622	Pfizer PF-07321332	Aligos ALG-097558
Beta	SARS-CoV-2	03021/2020 Vero E6+CP	n.d.	n.d.	0.116	0.010
		B.1.1.7 (alpha) A549-ACE2-TMPRSS2	0.038	0.023	0.099	0.012
		B.1.617.2 (delta) A549-ACE2-TMPRSS2	0.126	0.141	0.217	0.013
		B.1.1.529 (omicron) VeroE6-GFP+CP	0.136	0.095	0.059	0.008
Alpha	SARS-CoV-1	Vero-E6+CP	0.323	0.154	0.173	0.022
	OC43 (β-hCoV)	HeLa EC ₅₀	0.168	n.d.	0.047	0.009
	229E (α-hCoV)	Huh-7 EC ₅₀	0.281	6.30	0.476	0.017

ALG-097558 demonstrates pan-coronavirus antiviral activity
ALG-097558 is more active than PF-07321332, PBI-0451 and S-217622 across all CoV's tested
At least 7-fold more active than competitors against SARS-CoV-2 Omicron variant

ALG-097558

Oral Prophylactic Treatment in the SARS-CoV-2 Hamster Model

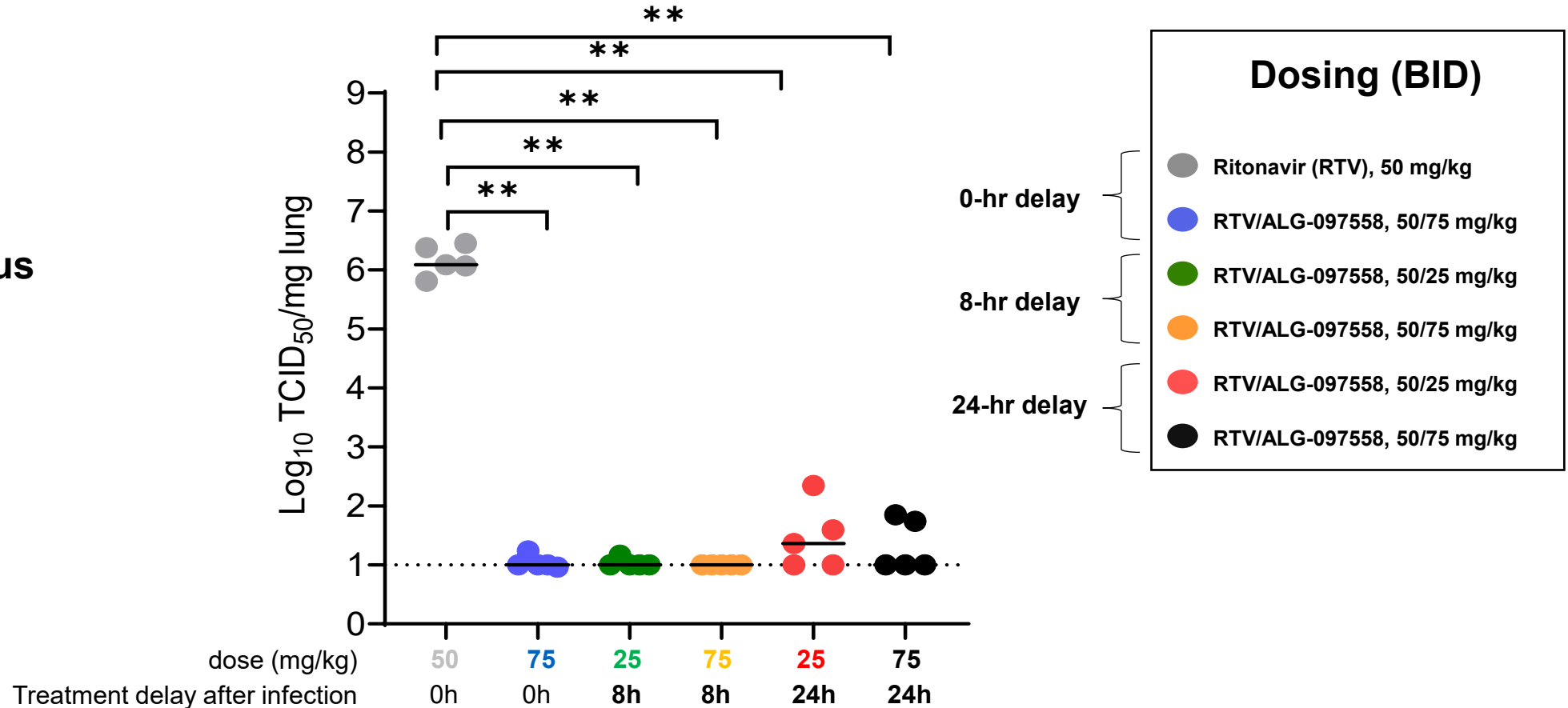


Significant reduction in infectious virus titers after prophylactic treatment with ALG-097558
Use of ritonavir is only needed in the hamster model

ALG-097558

Oral Therapeutic Treatment in the SARS-CoV-2 Hamster Model

Amount of
Infectious Virus
at Day 4



Significant reduction in infectious virus titers after therapeutic treatment with ALG-097558
Use of ritonavir is only needed in the hamster model

ALG-097558

SARS-CoV-2 3CLpro L50F E166A L167F Mutant

- ALG-097558 has the least loss of activity against a triple mutant conferring resistance to other SARS-CoV-2 PIs

Compound Name	Biochemical	VeroE6+CP
	L50F/E166A/L167F (fold change versus WT)	L50F/E166A/L167F EC ₅₀ [μM] (fold change)
PF-07321332 (Pfizer)	66 (n=6)	3.5-3.7 (38-fold)
PBI-0451 (Pardes)	>65 (n=2)	7.6-8.4 (25-fold)
S-217622 (Shionogi)	>67 (n=2)	15-16 (59-fold)
ALG-097558 (Aligos)	3 (n=3)	0.11-0.14 (9-fold)

Potential for ALG-097558 to retain significant potency against resistant mutants

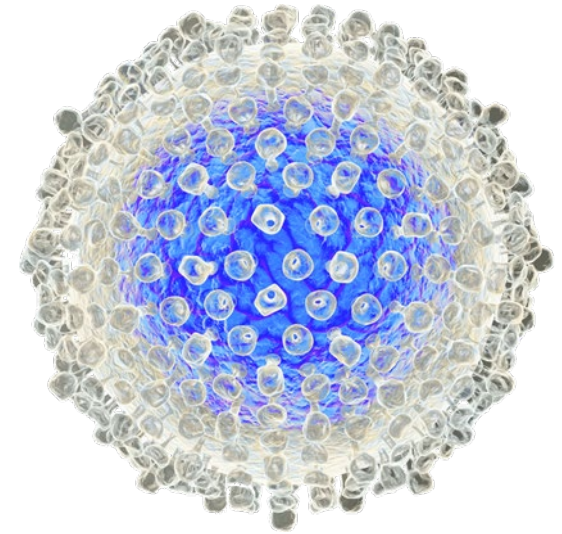
ALG-097558

Coronavirus Protease Inhibitor Drug Candidate

- Potent pan-coronavirus protease inhibitor drug candidate
 - Additional candidates from the series advancing as backup compounds
- Superior preclinical profile versus PF-07321332 (Pfizer)
 - 7-27 fold more potent in biochemical and cell-based assays vs. SARS-CoV-2
 - › 7-fold more potent vs. the Omicron variant (cell-based assay)
 - Greater cellular potency across other coronavirus strains
 - Retains activity against resistant variants
 - Excellent efficacy in the SARS-CoV-2 hamster model
- Potential for more convenient, less complex treatment regimen
 - PK profile in preclinical species predicts a projected human efficacious dose of 350-600 mg BID without ritonavir
- Timelines: Phase 1 enabling nonclinical studies H2 2022, Phase 1 H1 2023

ALG-097558 is a ritonavir-free, highly differentiated, pan-coronavirus protease inhibitor

Chronic Hepatitis B

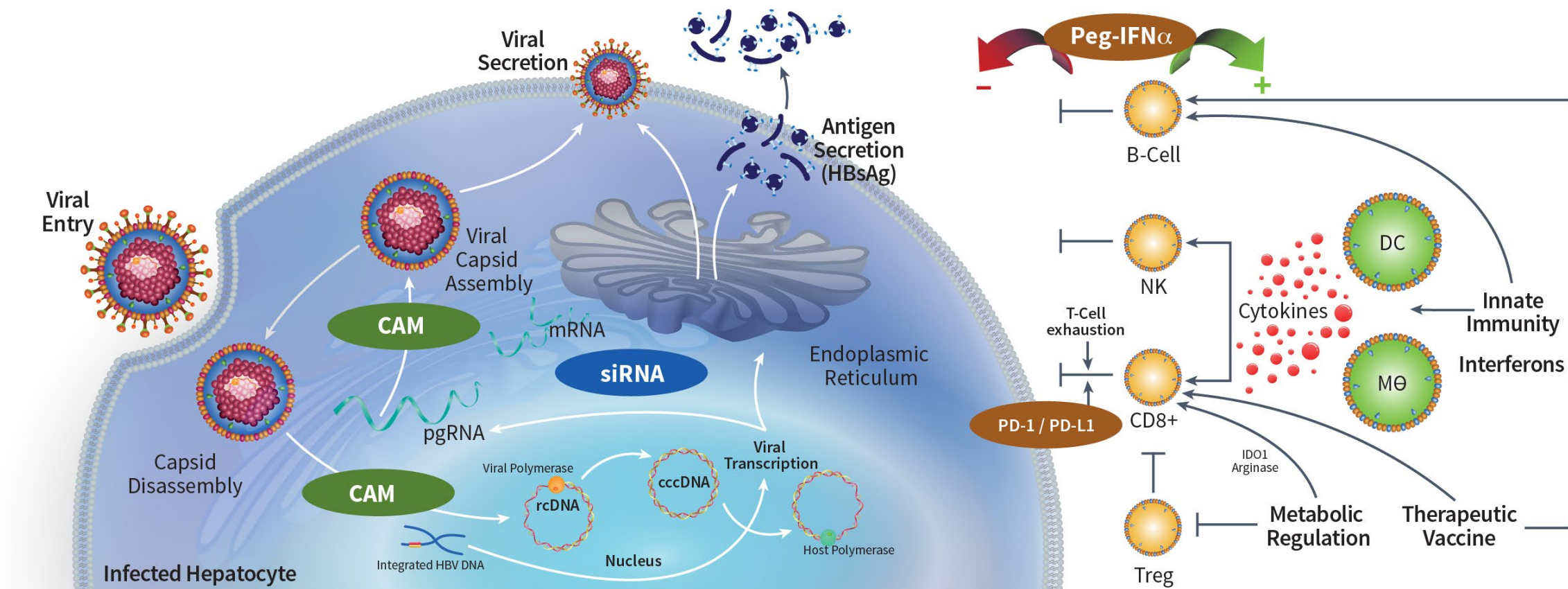


Therapeutic Approaches to CHB Functional Cure

Inhibit Viral Replication

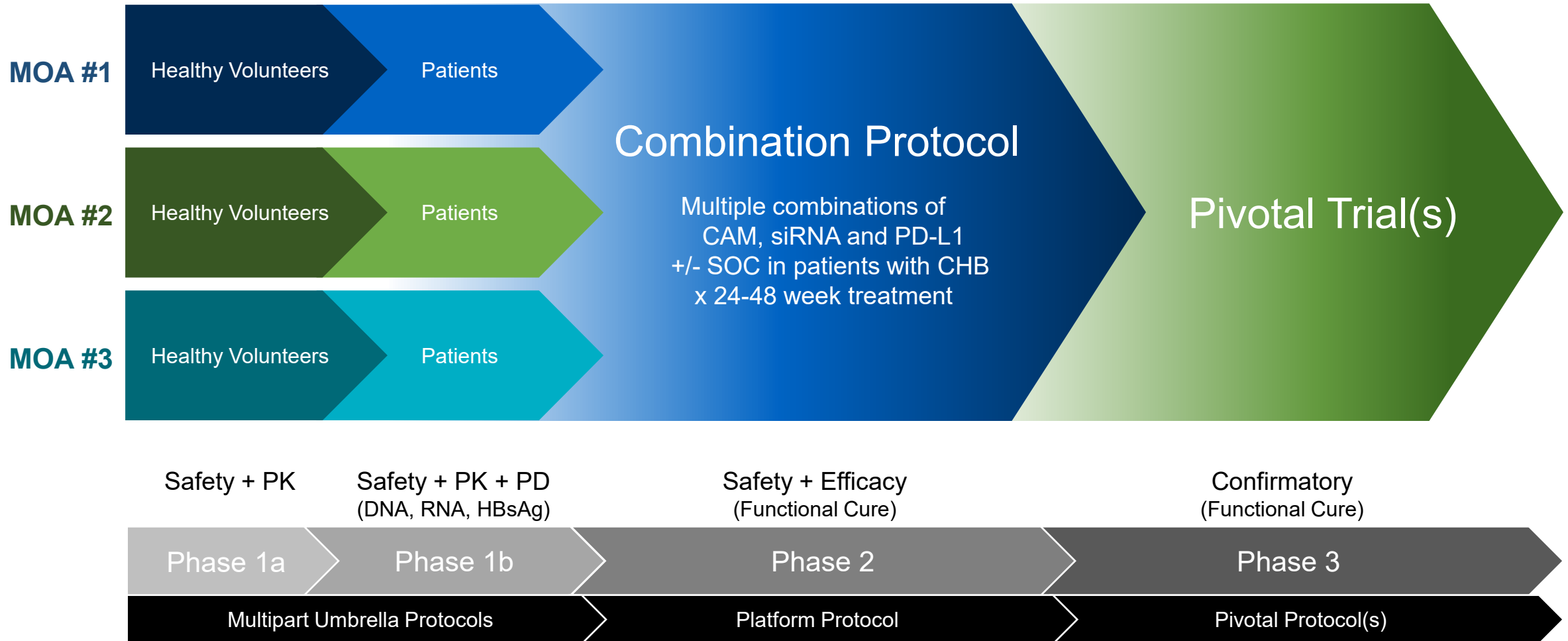
Lower Antigen Burden

Boost Immune Response



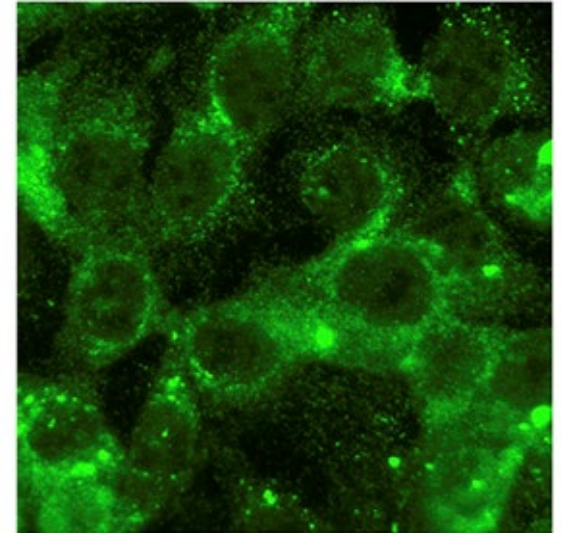
Chronic Hepatitis B

Clinical Strategy for CHB Functional Cure



Inhibiting Viral Replication

- ALG-000184 (Class-II CAM)



ALG-000184

In Vitro Potency vs. Competitor Class-II CAMs

Compound	Current Status	HBV DNA reduction (EC ₅₀ nM)	Cell Type
Vebicorvir	Discontinued	172	AD38
Assembly ABI-H3733	Phase 1	5	AD38
Janssen JNJ-6379	Phase 2	54	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

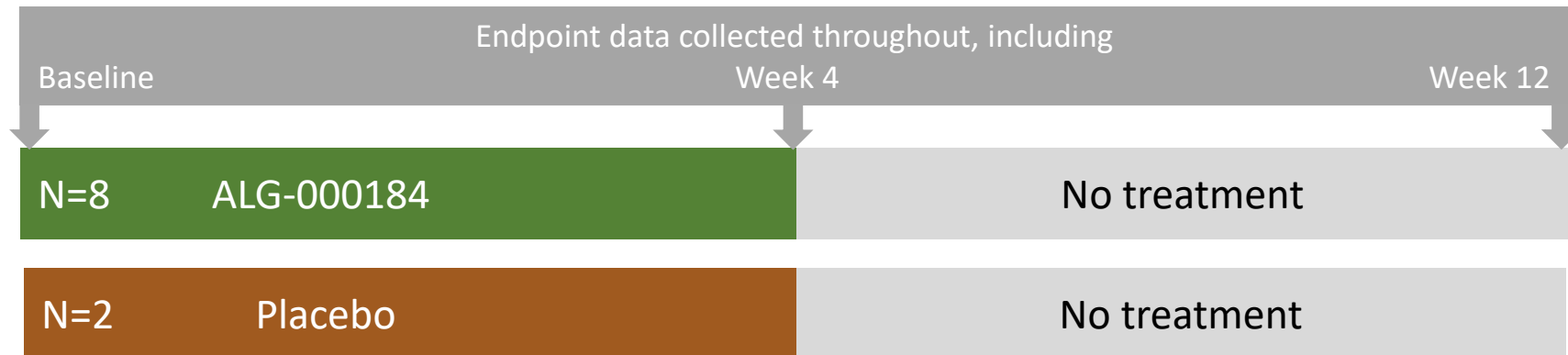
ALG-000184 has ~10 to 300-fold enhanced potency vs. other known CAMs in clinical development

ALG-000184

Phase 1 Study in CHB Subjects

Multiple Doses in Currently Not Treated/Treatment Naïve CHB Subjects

HBV DNA > 2000 IU/mL, HBeAg- or HBeAg+
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)

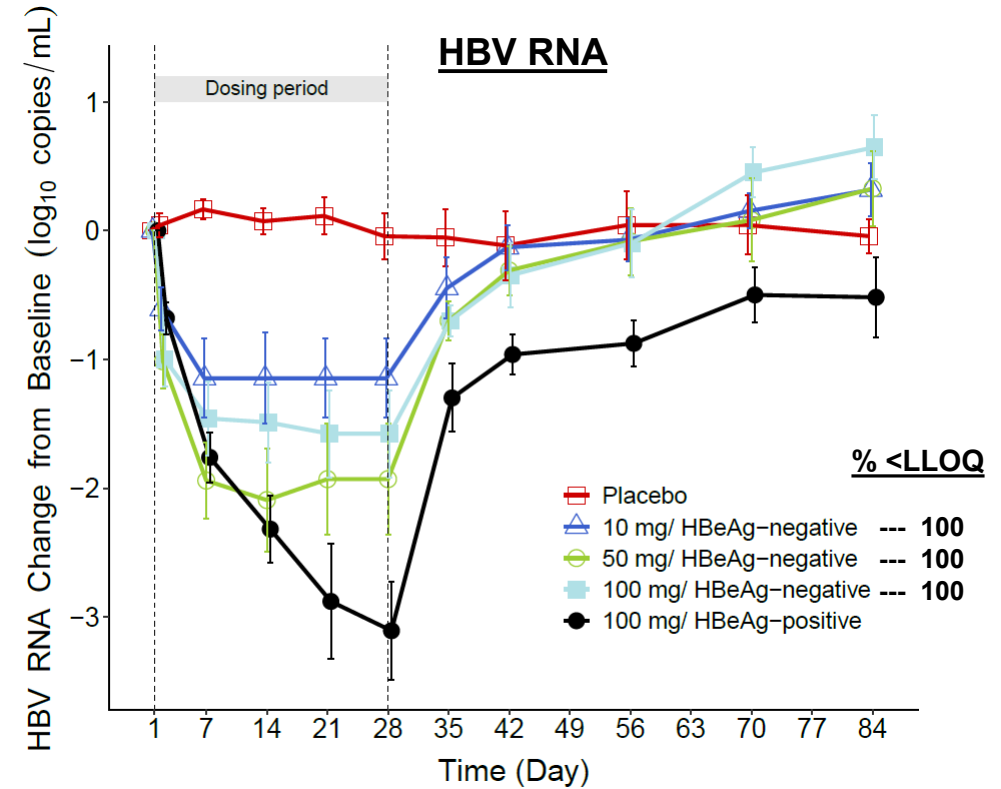
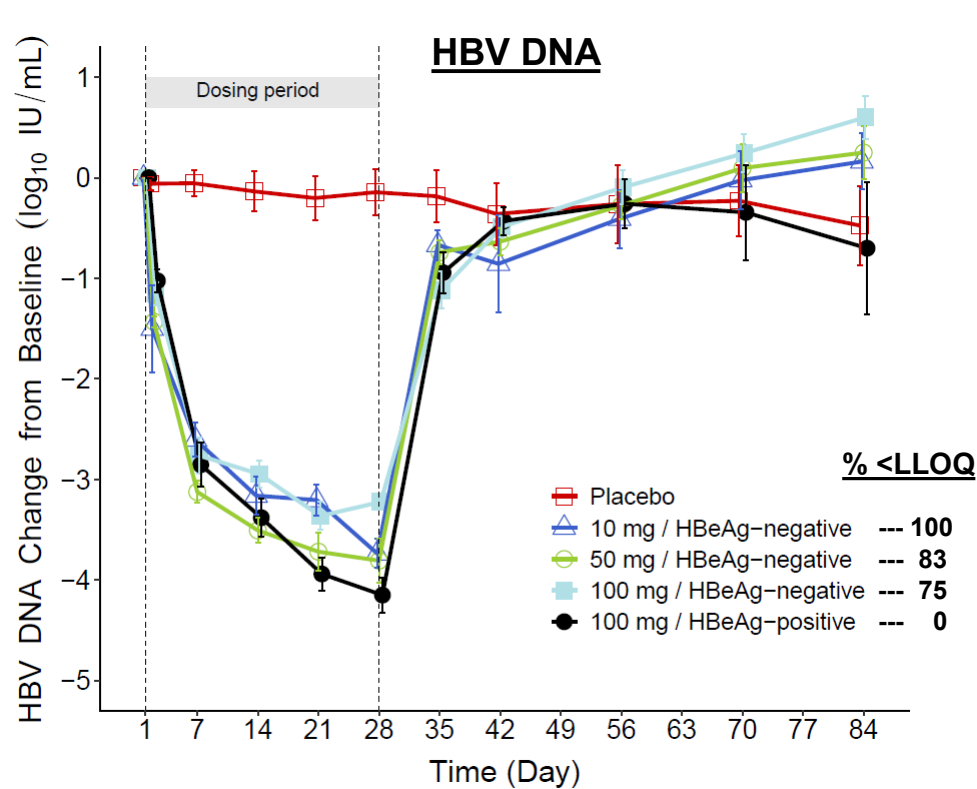


Dosing in cohorts 1 (100 mg, HBeAg-), 2 (50 mg, HBeAg-), 3 (10 mg, HBeAg-), 4 (100 mg, HBeAg+) and 5 (300 mg, HBeAg+) complete

ALG-000184-201

Part 3 Cohorts 1-4 Antiviral Activity Data

Mean (SEM) Serum HBV DNA* and HBV RNA** Levels Change from Baseline Through the End of Study



HBeAg- CHB subjects:

Similar HBV DNA reduction for 10, 50 and 100 mg (~3-4 $\log_{10}$ IU/mL)
DNA, RNA <LLOQ in $\geq 75\%$ and 100% of subjects, respectively

HBeAg+ CHB subjects:

Potent DNA, RNA reductions observed (100 mg)

ALG-000184

Antiviral Activity vs. Competitor CAMs (HBeAg Negative*)

Drug Name	Current Status	Dose	HBV DNA	
			Mean Decline from BL to EOT (Log ₁₀ IU/mL)	% < LLOQ at Day 28
Vebicorvir ^{1,2}	Discontinued	300 mg	2.5	25
JNJ-6379 ^{3,4}	Phase 2	250 mg	2.7	56
EDP-514 ⁵	Phase 1b	200 mg	2.9	N/A
		800 mg	3.4	N/A
AB-836 ⁶	Phase 1	100 mg	3.1	N/A
ALG-000184	Phase 1	10 mg	3.7	100

10 mg ALG-000184 has more potent antiviral activity than competitor CAMs dosed at 100-800 mg

HBeAg+ Chronic Infection (Immune Tolerant)

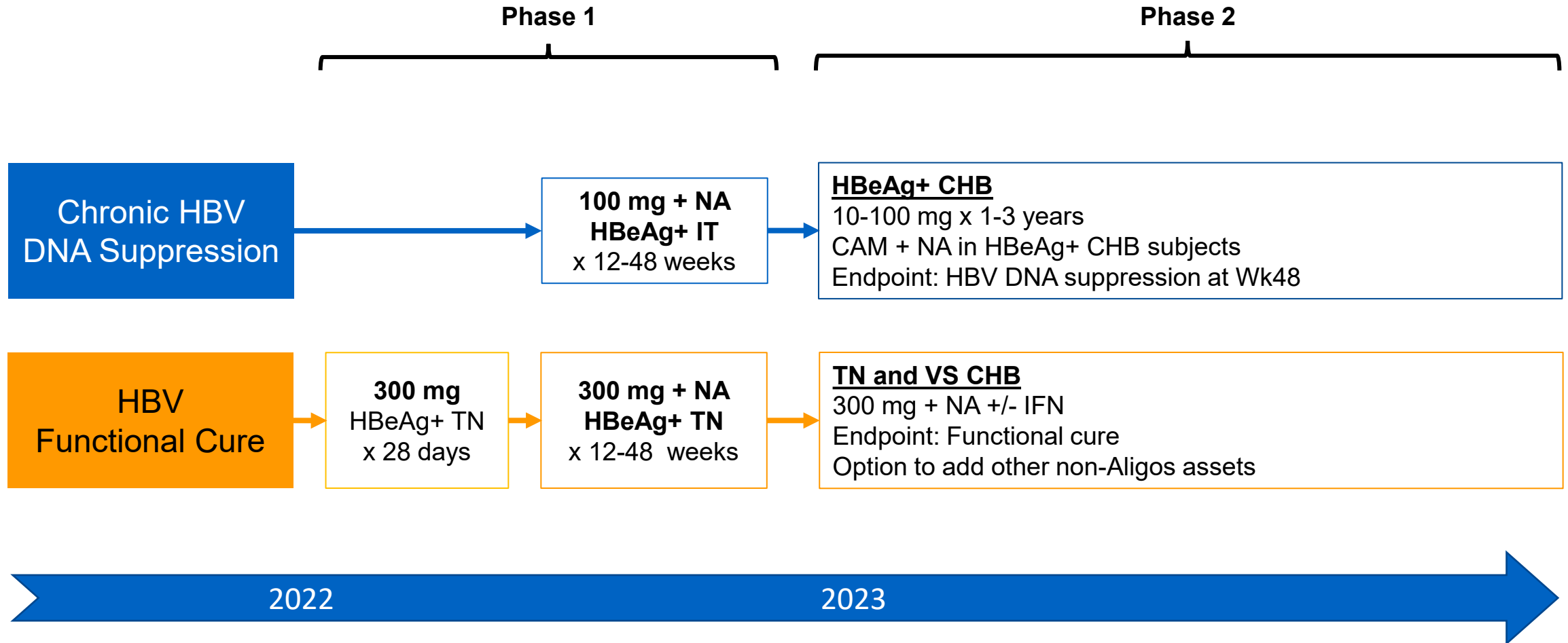
Growing Realization of Unmet Medical Need

- Patients with HBeAg+ chronic infection have high DNA and HBsAg levels
- Baseline HBV DNA levels correlate with HCC, cirrhosis risk*
 - Reducing DNA levels decreases integrations, which are thought to be related to HCC risk**
- Suboptimal DNA suppression, HBeAg loss rates achieved with NA alone***
- NA + CAM therapy shown to result in deeper and faster HBV DNA suppression^
 - ALG-000184 appears to be best in class for DNA/RNA suppression
- Despite current guidelines not recommending treatment, growing consensus that they should be
 - Many KOLs already treat this population
- Large population: estimated to be ~20-30% of all chronic HBV patients^^
- If chronic suppression data are favorable in HBeAg+, opportunity to evaluate suppression in HBeAg-, where subjects on NAs can still have detectable DNA by more sensitive assays^^^

Best in class DNA/RNA suppressive ability of ALG-000184 offers important potential benefits to subjects with HBeAg+ chronic infection

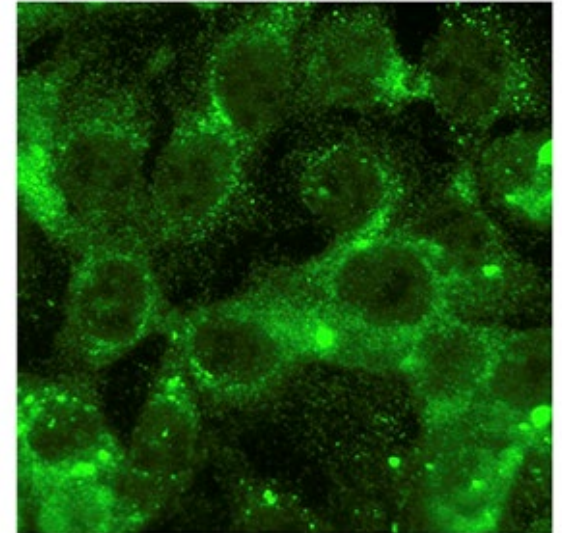
ALG-000184

Phase 1-2 Clinical Development Pathways



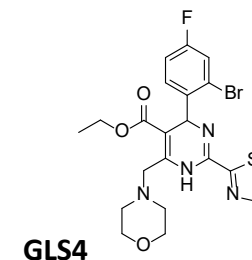
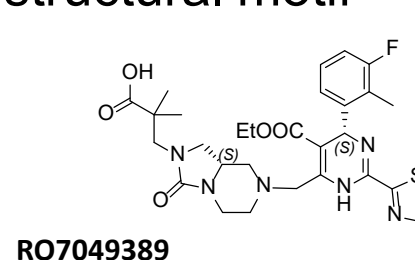
Inhibiting Viral Replication

- Class-I CAM



Class-I Capsid Assembly Modulators

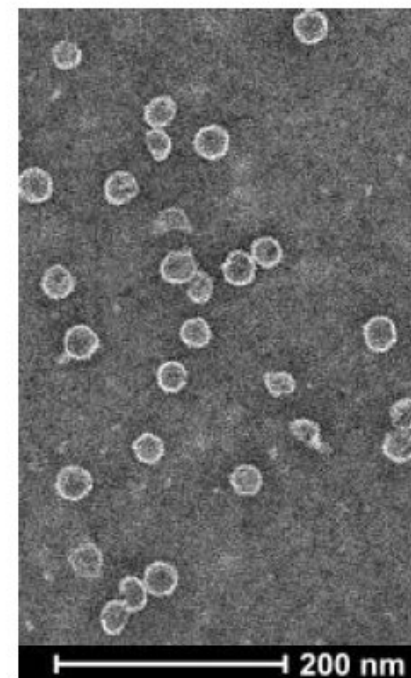
- Class-I capsid assembly modulators are orally bioavailable small molecules that result in large aberrant particles and decrease HBsAg in the AAV-HBV mouse
- Publicly disclosed Class-I CAMs are in the heteroaryl pyrimidine (HAP) class
 - GLS4 and RG7907 are two clinical drug candidates with this structural motif
 - HAPs have several potential drawbacks related to safety
 - › HBc dependent cell death of infected hepatocytes in vitro
 - › ALT flare in animal models in vivo
- Aligos has discovered and optimized multiple non-HAP Class-I CAM series
 - Proof of concept in HBV AAV animal model with ALG-005398
 - › Significant reductions in HBV DNA, HBeAg and HBsAg without observation of ALT increases



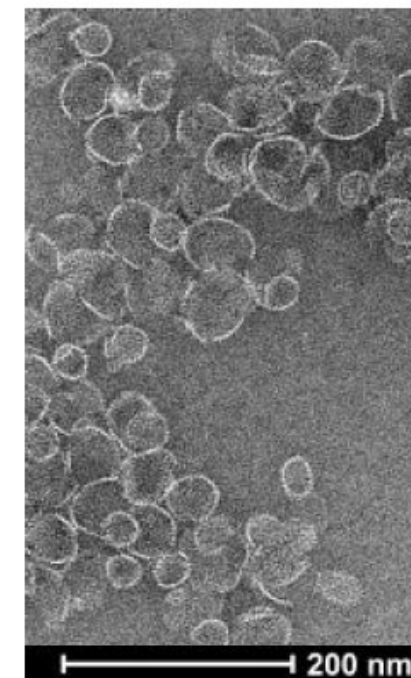
ALG-005398

In Vitro Potent Class-I CAM

- ALG-005398 potency in HepG2.117 cells
 - Reduction in HBV DNA
 - › $EC_{50} = 3.4 \text{ nM}$
 - › $EC_{90} = 10.6 \text{ nM}$
- Demonstration of class-I CAM properties
 - Formation of large aberrant capsids



NaCl
1 M

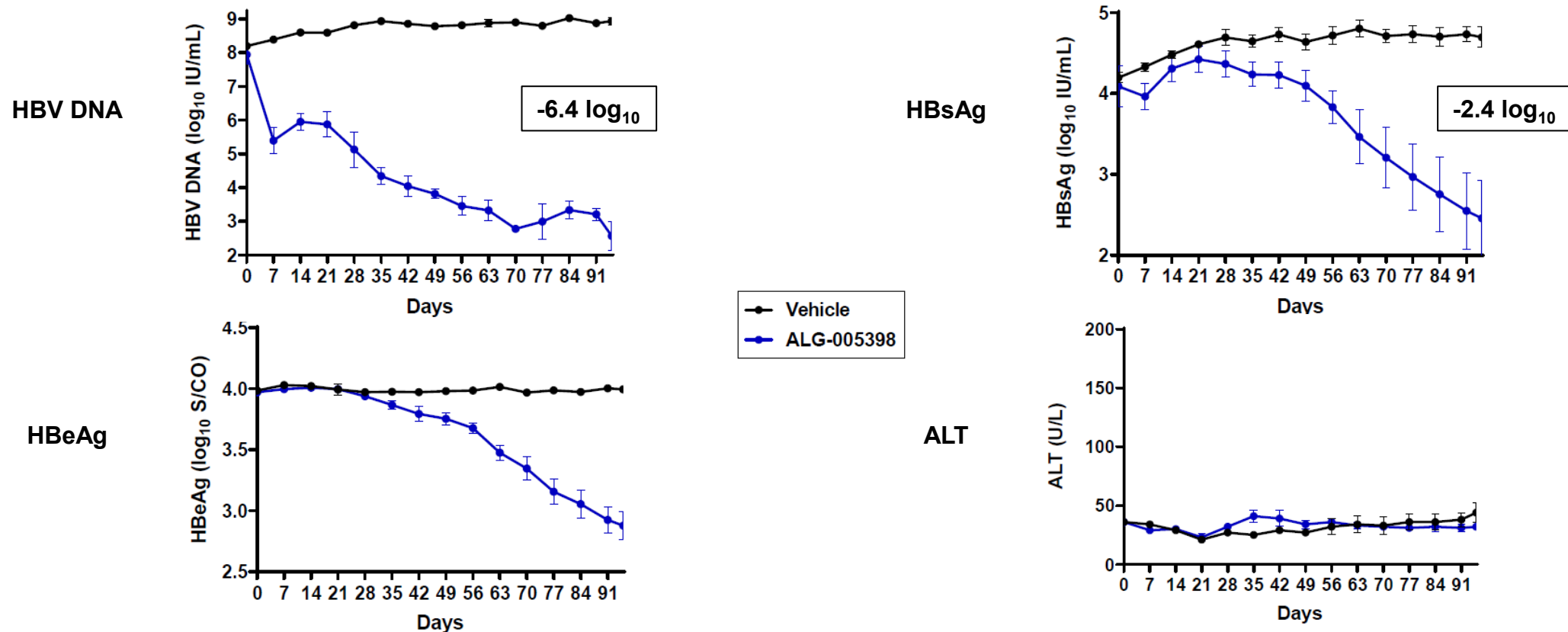


ALG-005398
25 μM

ALG-005398 is a non-HAP Class-I CAM demonstrating excellent in vitro potency

ALG-005398

In Vivo Proof of Concept in the AAV-HBV Mouse Model

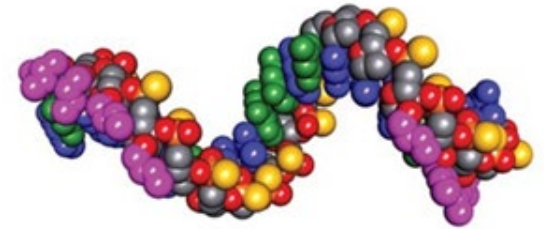


ALG-005398 was dosed at 30 mg/kg/day BID for 96 days

ALG-005398 is a non-HAP Class-I CAM that reduces HBsAg and other antigens in vivo

Lowering S-Antigen Burden

- ALG-125755 (siRNA)



Short Interfering Nucleic Acid ALG-125755

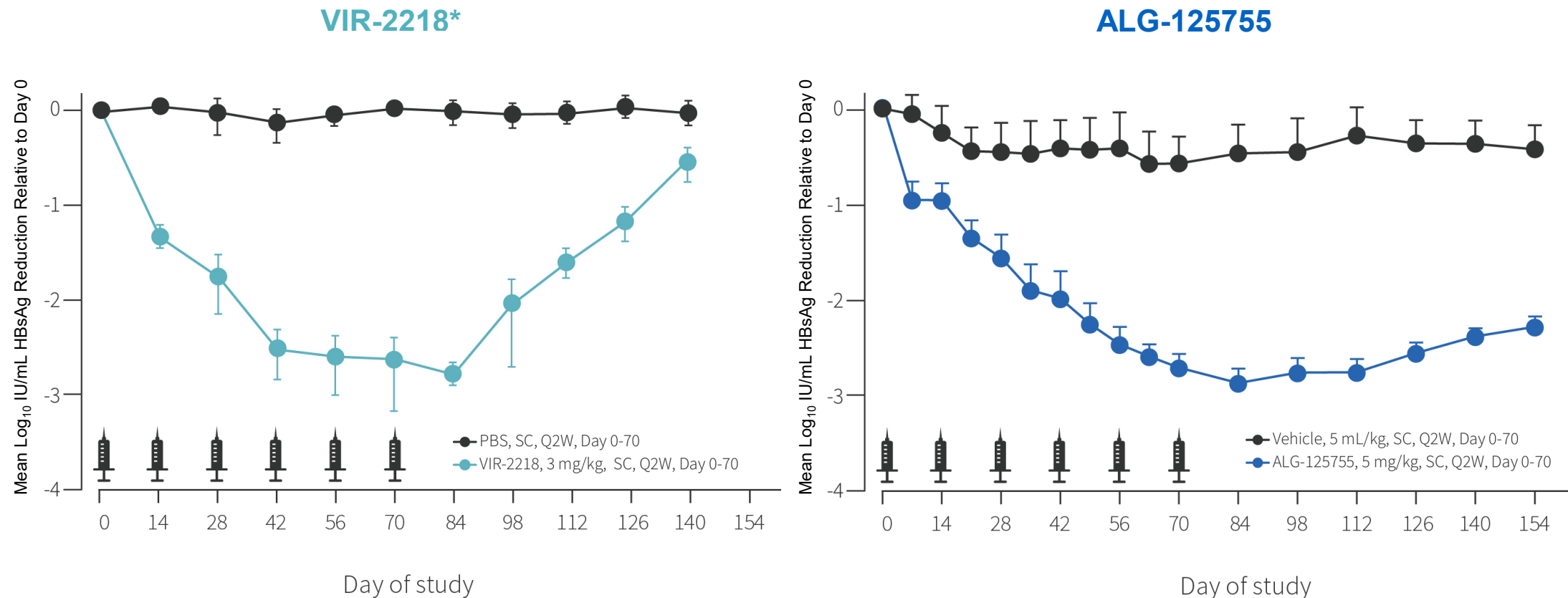
Discovery and Advancement of a Differentiated siRNA

- 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
 - Proprietary patterns discovered to increase potency and stability/duration of action
 - Exclusive license to GalNAc technology applicable for liver targeting across oligo modalities
- Our siRNA approach may have safety, stability and potency advantages vs. competitor siRNAs
- CTA filed 23 August
 - HV dosing to start Q4 2023

ALG-125755 is differentiated from potential competitors. HV dosing to start Q4 2023

ALG-125755

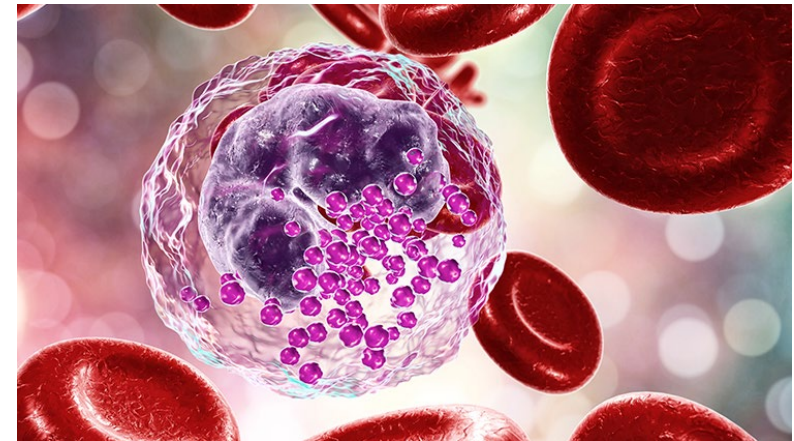
Repeat Dosing in the AAV-HBV Mouse Model vs. VIR-2218



ALG-125755 demonstrates a more sustained reduction in HBsAg vs. competitor siRNAs

Boosting the Immune Response

- ALG-093453 (PD-L1 Small Molecule)



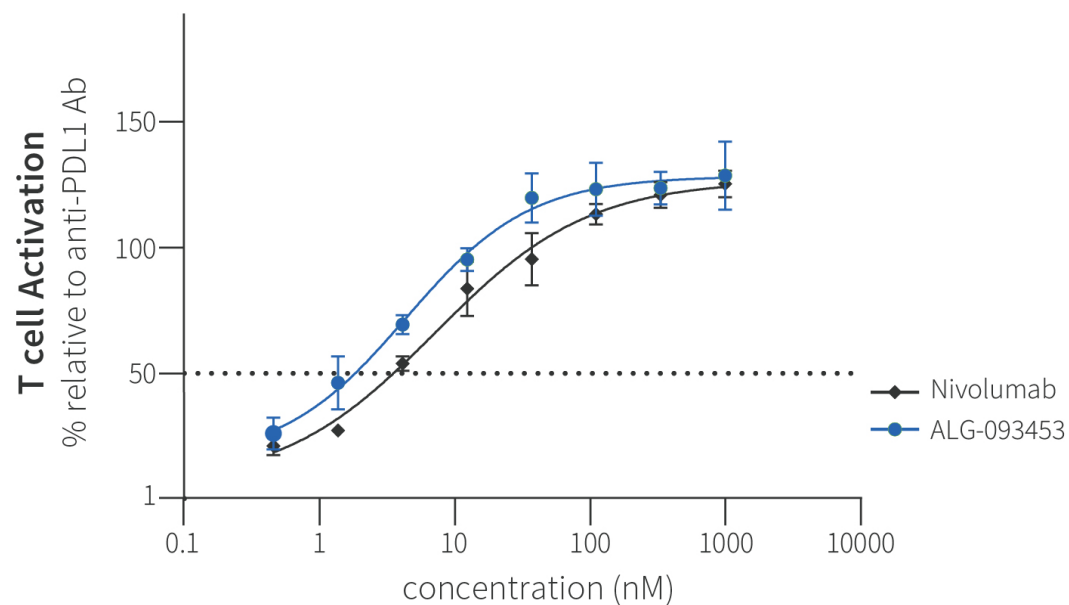
PD-L1 Inhibitors

- Exhaustion of HBV specific T-cells contributes to the persistence of CHB
- Proof of concept in CHB with anti-PD1 antibodies has been established
 - Multiple clinical studies have demonstrated HBsAg reductions in CHB infected patients
- Aligos has discovered several potent series of small molecule PD-L1 inhibitors
 - Potential for oral delivery and targeting to the liver
 - › Liver targeting may avoid the safety liabilities seen with parenterally delivered anti-PD1 antibodies while improving efficacy
- ALG-093453 is a novel small molecule PD-L1 inhibitor
 - Biochemical and cell-based potency established

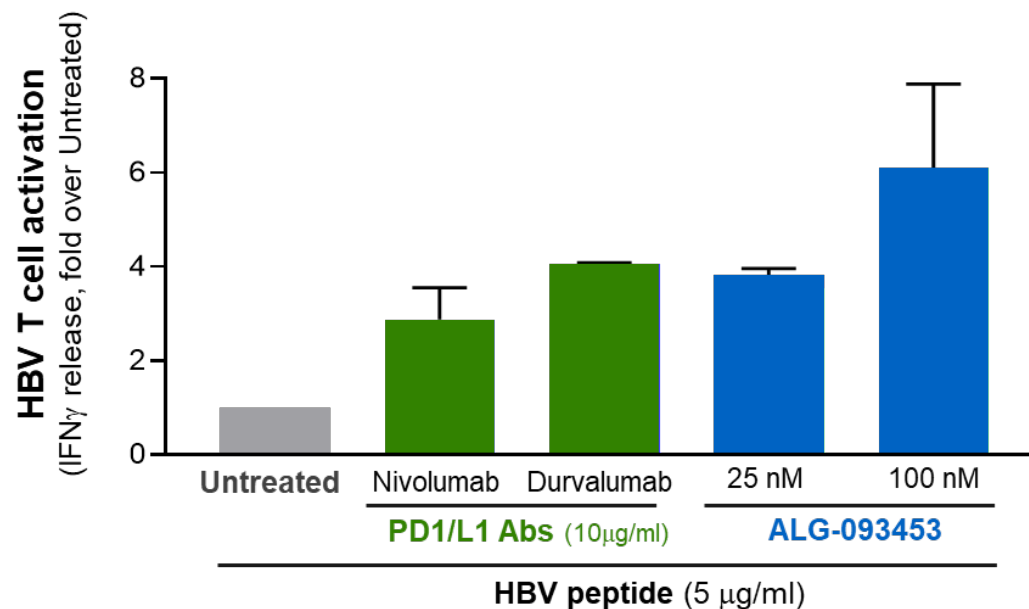
ALG-093453

PD-L1 Inhibitor

T cell Activation
Jurkat-NFAT assay



T cell Activation
PBMC from HBV Positive Donor



ALG-093453 activates HBV specific T cells with low nM potency, similar to nivolumab and durvalumab

NASH

ALG-055009, small molecule THR- β agonist



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

Best In Class Potential vs. 1st Gen THR- β Agonists

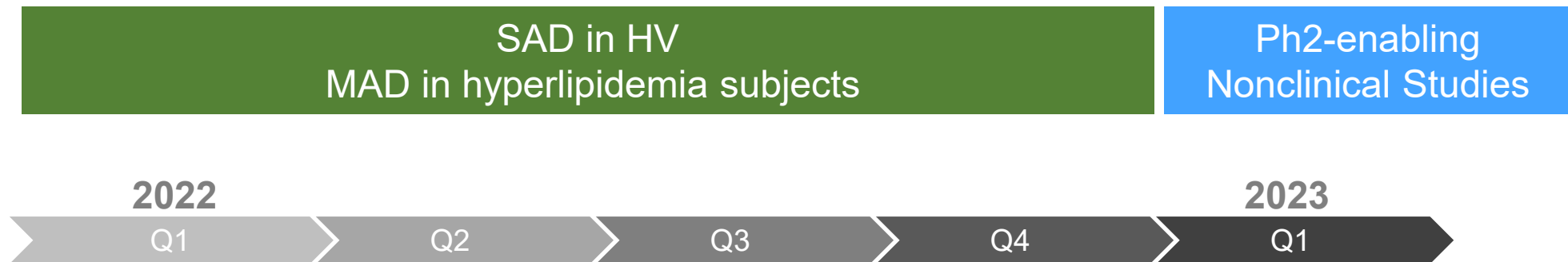
Parameter		VK-2809	MGL-3196	ALG-055009
Efficacy	28 day mouse DIO TC, LDL lowering	28%	28%	Up to 44%
	Percent of patients with $\geq 30\%$ change from baseline in MRI-PDFF (Ph2)	76%-100%	60%	High Expected
	BCRP, OATP substrate	?	Yes	No (NTCP)
	AUC/Cmax interindividual variability	Limited	High	Low
	PK linearity	Yes	No	Yes
Safety	GI side effect Diarrhea/Nausea (%)	-	33% / 14% (7% / 5% placebo)	TBD
	β Selectivity (α/β)*	1.4	2.5	3.8
	Potential cardiosafety (i.e., α) liability	Yes	No	Low risk, high liver/heart ratio
	Reactive metabolites in vitro* (GSH adduct) / Risk for DILI	Yes	No	No
	Possible Prodrug liability	Yes**	No	No
DDI	CYP inhibition $>50\%$ @10 μM *	$>60\%$ for 5 isozymes $>90\%$ for 2C9 & 2C19 CYP3A4-dependent activation	91% for 2C8	No

Potential for enhanced efficacy/PK vs. MGL-3196 and enhanced efficacy/safety vs. VK-2809

ALG-055009

Development Timelines*

ALG-055009
(THR-β)

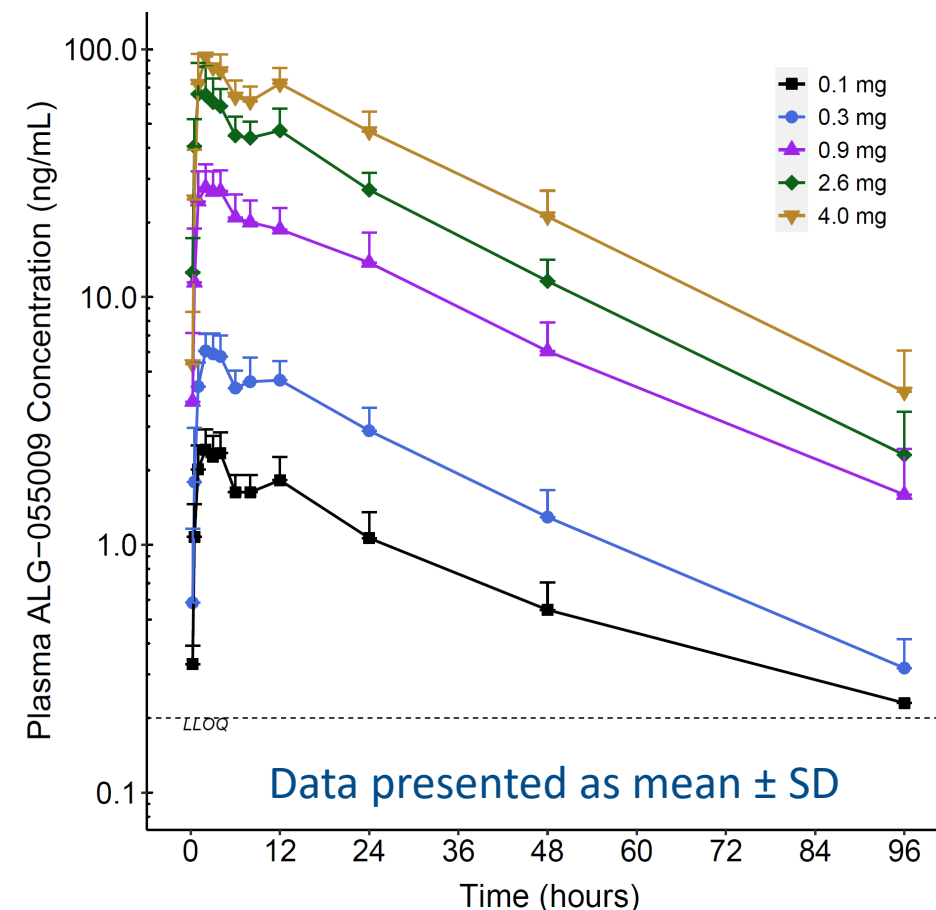


SAD and MAD complete – data to be presented at AASLD 2022

Study ALG-055009-301

SAD Interim Data – Safety, PK

- Study Design
 - Part 1 (SAD) - cohorts randomized to a single oral dose of ALG-055009 (N=6) or placebo (N=2)
 - › Dose levels: 0.1, 0.3, 0.9, 2.6, 4 mg
- Safety: well tolerated
 - No serious adverse events, premature discontinuations, Grade ≥ 3 treatment emergent AEs (TEAEs), or clinical hyper-/hypo-thyroidism
- PK
 - Dose proportional PK with low variability, $t_{1/2}$ = 20-24 hours (supports QD dosing)



Single (≤ 4 mg) ALG-055009 doses well tolerated with favorable PK properties

Study ALG-055009-301

SAD Interim Data - PD

- Expected thyromimetic effects observed: generally dose proportional
 - Increases in Sex Hormone Binding Globulin (target engagement)
 - Decreases in thyroid hormones (TSH, T3, T4)
 - Decreases in lipids (triglycerides, LDL, ApoB, etc.)

Expected thyromimetic effects observed – evidence of anti-lipid activity

ALG-055009

Summary

- More potent, β -selective vs. frontrunner THR- β compounds
- Predictable and dose-proportional PK properties
- Phase 1 proof of concept study
 - SAD in HV – complete
 - › Well tolerated, linear PK (supports QD dosing), expected thyromimetic effects observed
 - MAD (dosing x 14 days in subjects with hyperlipidemia) – dosing in all 4 cohorts complete
 - › Data to be presented at AASLD 2022

ALG-055009 has expected thyromimetic profile with PK that is differentiated vs. Madrigal
Phase 1 SAD/MAD complete; data will be presented at AASLD

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
- Resmetirom Phase 3 readout expected in Q4 2022
- Aligos NASH ALG-055009 program has strong optionality in 2023
 - 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 complementary MOA

Aligos Therapeutics Summary

- We are advancing multiple drug candidates with clinically-validated MOAs with the goal of rapidly developing optimized combination regimens
 - Coronavirus Protease Inhibitor (ALG-097558)
 - › Targeting to complete FIH enabling nonclinical studies and file Phase 1 CTA in Q1 2023
 - Chronic Hepatitis B
 - › CAM-II (ALG-000184) dosing in Phase 1b (up to 12-48 weeks) – best in class data to date
 - › siRNA (ALG-125755) – dosing in HV projected to start in Q4 2022
 - › PD-L1 and CAM-I programs advancing towards candidate selection
 - NASH
 - › THR- β agonist (ALG-055009) SAD and MAD complete; data to be presented at AASLD 2022
 - › Our exclusive oligonucleotide-based License & Research collaborations with Merck are progressing
- As of June 30, 2022; cash balance was \$159.3M*; fully diluted common shares: 51,824,374
- Filed November 4, 2021; Shelf/S-3 Registration (\$400M) and ATM (\$75M)

ALIGOS
THERAPEUTICS

