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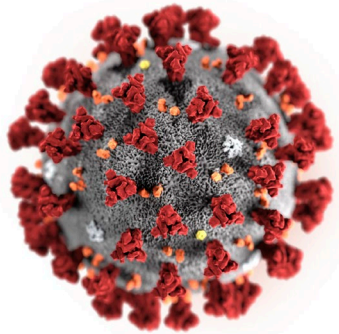

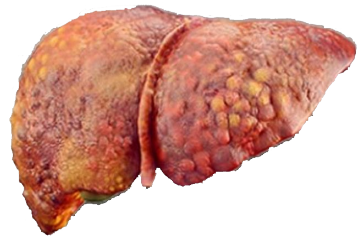

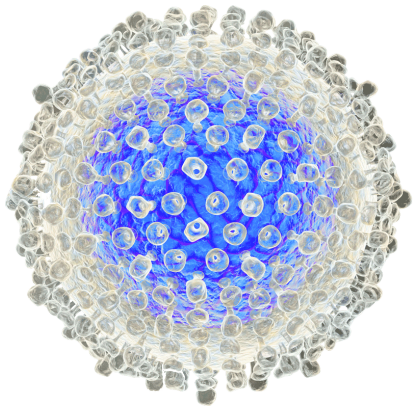

Lawrence M. Blatt, Ph.D.
Chairman, CEO & Co-Founder

Corporate Presentation
January 24th, 2023

Disclosures

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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 Milestones/Data Readouts Anticipated in 2023

Candidate	Indication	MOA	2023 Clinical Trial Timelines and Data Readouts
			Q1 → Q2 → Q3 → Q4
ALG-055009	NASH	THR-β Agonist	Phase 1 → Phase 2 Enabling Activities (GLP Toxicology, Drug Product Supply) ★ CTA
Oligonucleotides (MERCK)	NASH	Multiple	Preclinical Activities
ALG-097558	Covid-19	Protease Inhibitor	Phase 1 Enabling Activities → SAD/MAD in HV → Phase 2
ALG-000184	CHB	CAM-E	Up to 48 Weeks Dosing in CHB: ★ APASL (12 weeks CHB); ★ EASL (≥12 weeks CHB); ★ AASLD (≥12 weeks CHB)
ALG-125755	CHB	siRNA	Single, Multiple Dosing in CHB: ★ APASL (SAD in HV); ★ EASL (SAD in CHB); ★ AASLD (SAD in CHB)

All timelines are approximate and subject to change based on enrollment and operational considerations.
Presentations at conferences are subject to abstract approvals.

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AASLD = American Association for the Study of Liver Diseases; APASL = Asian Pacific Association for the Study of the Liver; CAM-E = capsid assembly modulator (empty); CHB = chronic hepatitis B; Covid-19 = coronavirus disease of 2019 (SARS-CoV-2); CTA = clinical trial application; EASL = European Association for the Study of the Liver; GLP = good laboratory practice; HV = healthy volunteers; MAD = multiple ascending doses; MOA = mechanism of action; NASH = nonalcoholic steatohepatitis; PK = Pharmacokinetics; SAD = single ascending doses; siRNA = small interfering ribonucleic acid (RNAi); THR-β = thyroid hormone receptor beta.

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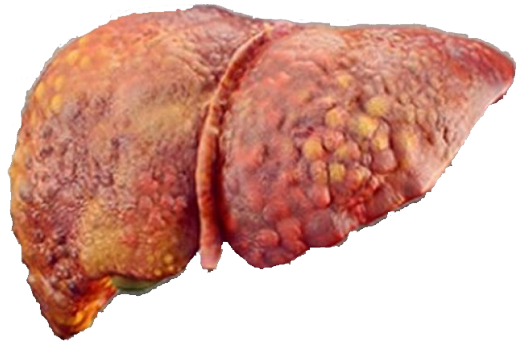
NASH

ALG-055009, small molecule THR-β agonist



ALG-055009
Summary

- Discovered by Aligos – issued US patent expires 2040
- More β-selective, >50 fold more potent than resmetirom
- Phase 1
  - Safety – well tolerated without clinical safety signals
  - PK – favorable profile (linear, low variability) that is differentiated vs. resmetirom
    - More uniform exposures may lead to more consistent efficacy and safety
  - Biomarkers – generally dose proportional
    - Increases in SHBG
    - Decreases in lipids
- Phase 2 preparation ongoing
  - Phase 2 enabling activities (e.g., GLP toxicology, Phase 2 drug supply) – 2023
  - CTA/IND filing Q4 2023

ALG-055009 is differentiated vs. resmetirom with favorable PK, which may improve risk-benefit profile
Phase 2 filing planned in Q4 2023

Characteristics of Commercially Successful Drugs

- Two main types of commercially successful drugs
  - First to market
  - Best in market (seldom the first)

- Top Selling Drugs

Drug Name	Cumulative Value	Timing to Enter Market
Atorvastatin (Lipitor)	$150B	5
Adalimumab (Humira)	$109B	3
Clopidogrel (Plavix)	$84B	2

ALG-055009 unlikely to be the first THR-β in the market, but may be better than resmetirom

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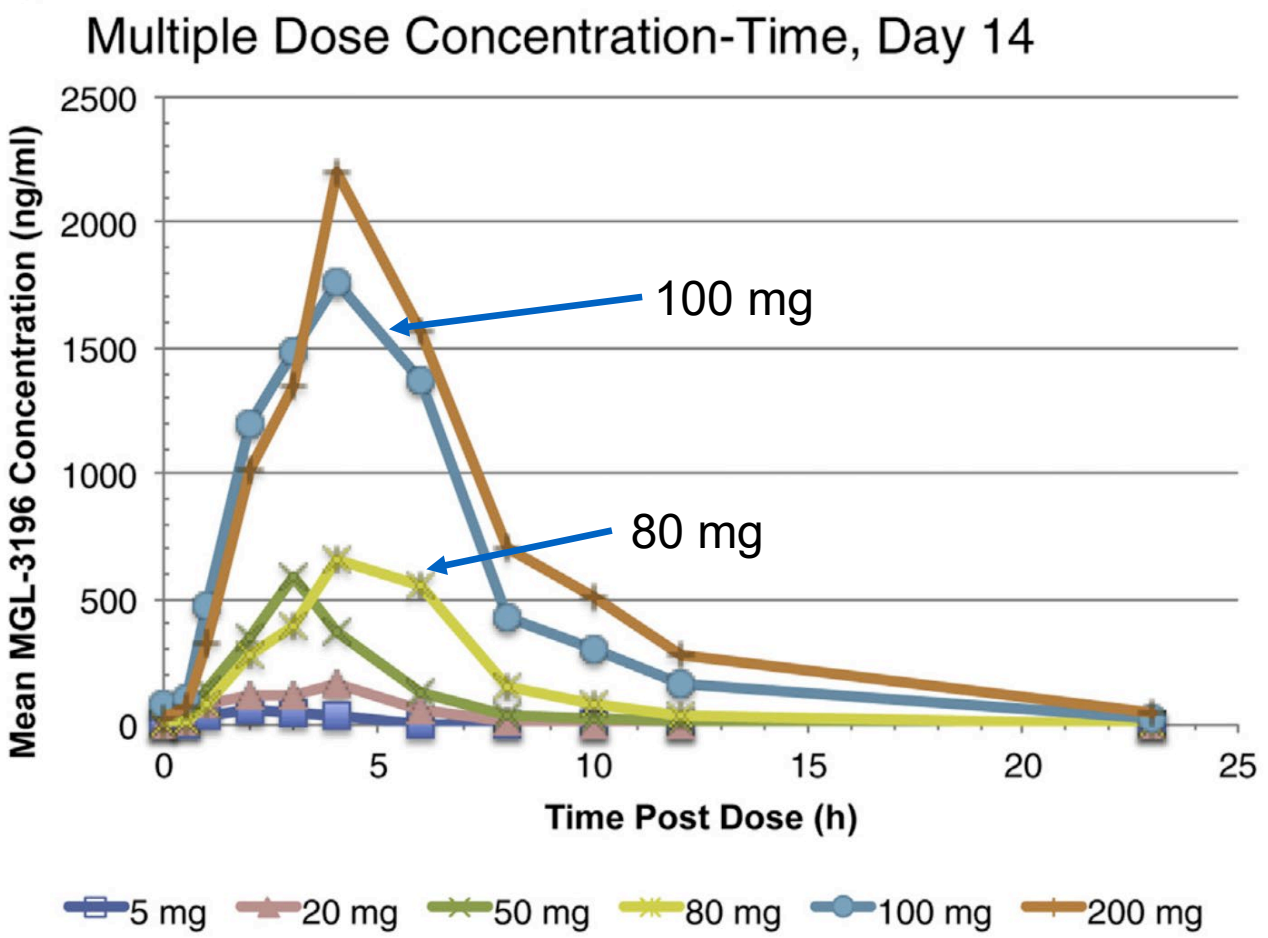
Brumley, Kiplinger, Dec 5, 2017.

Resmetirom Phase 1 MAD Data
Nonlinear PK



A 25% increase in dose (80 mg →100 mg) results in a ~300% increase in exposure

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MAD data at Day 14 from Taub et al, Atherosclerosis, 2013.

Resmetirom Phase 2 Data
MRI-PDFF

- Study design
  - 125 NASH subjects randomized in a 2:1 ratio to resmetirom or placebo x 36 weeks
  - Starting dose (4 weeks) 80 mg. Dose titrated at Week 4 based on Week 2 PK
    - 53% of resmetirom subjects had their dose titrated up (100 mg) or down (40-60 mg)
  - Endpoints: MRI-PDFF at 12 weeks (primary), biopsy at 36 weeks (secondary)

- Efficacy

MRI-PDFF Parameter (Week 12)	Placebo (N=38)	Resmetirom	
		Low Exposure (N=34)	High Exposure (N=44)
% Change from Baseline (standard error)	-10.4% (4.3)	-24.1% (4.4)	-39.7% (3.9)
% of Subjects with ≥30% Fat Reductions	18.4%	41.2%	75.0%

Resmetirom exposure-responsively improved MRI-PDFF

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MRI-PDFF = Magnetic Resonance Imaging Proton Density Fat Fraction.
Harrison et al., Lancet 2019.

Resmetirom Phase 2 Data
Liver Biopsy

MRI-PDFF de-fatting correlated with histologic improvement

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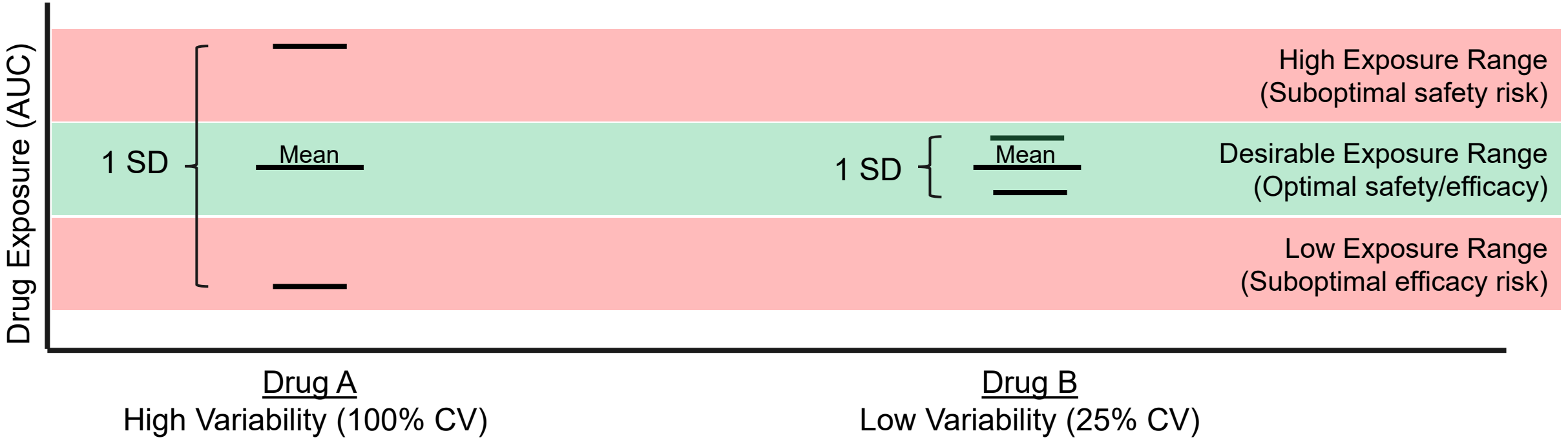
NR = Non-Responder.
Image adapted from Loomba et al, EASL 2020.

Hypothetical Impact of PK Variability on Efficacy/Safety



Two drugs with same mean could have different risk-benefit profiles due to different PK profiles

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CV = coefficient of variation; SD = standard deviation.

ALG-055009
More Potent and Selective In Vitro than Resmetirom 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)
ALG-055009	**191**	**50**	**3.8**
Resmetirom	5927	2366	2.5
VK-2809 Parent	366	269	1.4
T3	14.2	11.6	1.2

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

High β selectivity and potency may improve risk-benefit profile

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*Data collected in HEK293T cells.

ALG-055009
Best In Class Potential vs. 1st Generation THR-β Agonists

Parameter		VK-2809	Resmetirom	ALG-055009
Efficacy	28 day mouse DIO TC, LDL lowering	28%	28%	Up to 44%
	Percent of patients with ≥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	β Selectivity (α/β)*	1.4	2.5	3.8
	Potential cardiosafety (i.e., α) liability	Yes	No	No
	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. resmetirom and enhanced efficacy/safety vs. VK-2809

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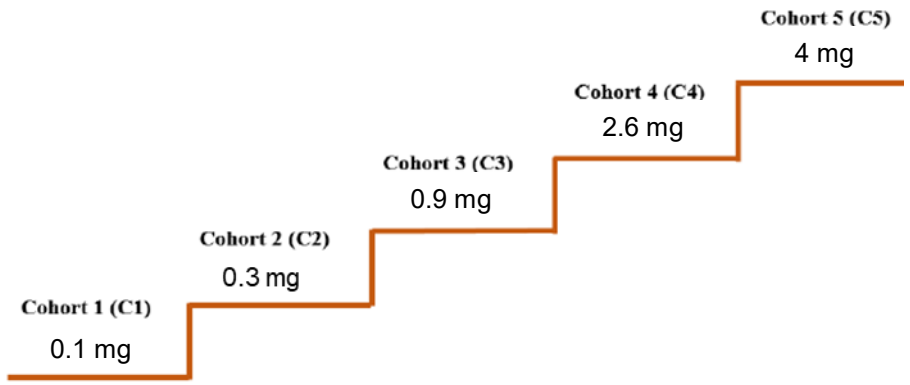
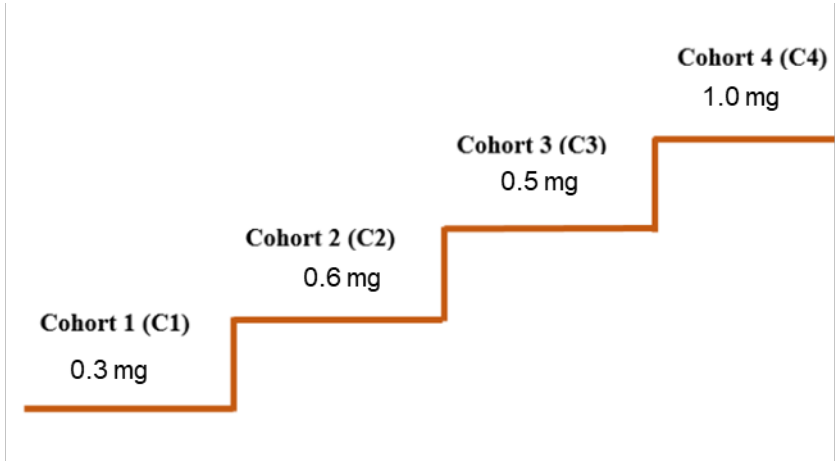
Abbreviations: AUC = area under the curve, BRCP = breast cancer resistance protein, Cmax = maximum concentration, CYP = cytochrome P450, DILI = drug induced liver injury, DIO = diet induced obesity; GSH = glutathione, LDL = low density lipoprotein, MRI-PDFF = magnetic resonance imaging – proton density fat fraction; OATP = organic anion transporting polypeptide, TC = total cholesterol,
* ALG-055009 clinical data from Charfi et al., EASL 2022. Competitor data derived from in-house studies.
**Same pro-drug as Pradefovir (pro-drugged adefovir). Pradefovir, but not adefovir, found carcinogenic in mice and rats (http://media.corporate-ir.net/media_files/IROL/89/89839/PipelineAug07.pdf).

ALG-055009 Phase 1
Study Design

Part 1: Single Ascending Dose (SAD)
N=up to 64 HV; n=8 per Cohort, n=6 ALG-055009 and n=2 Placebo



Part 2: Multiple Ascending Dose (MAD) – Dosing PO QD × 14 Days
N=up to 80 Subjects with Hyperlipidemia; n=10 per Cohort, n=8 ALG-055009 and n=2 Placebo



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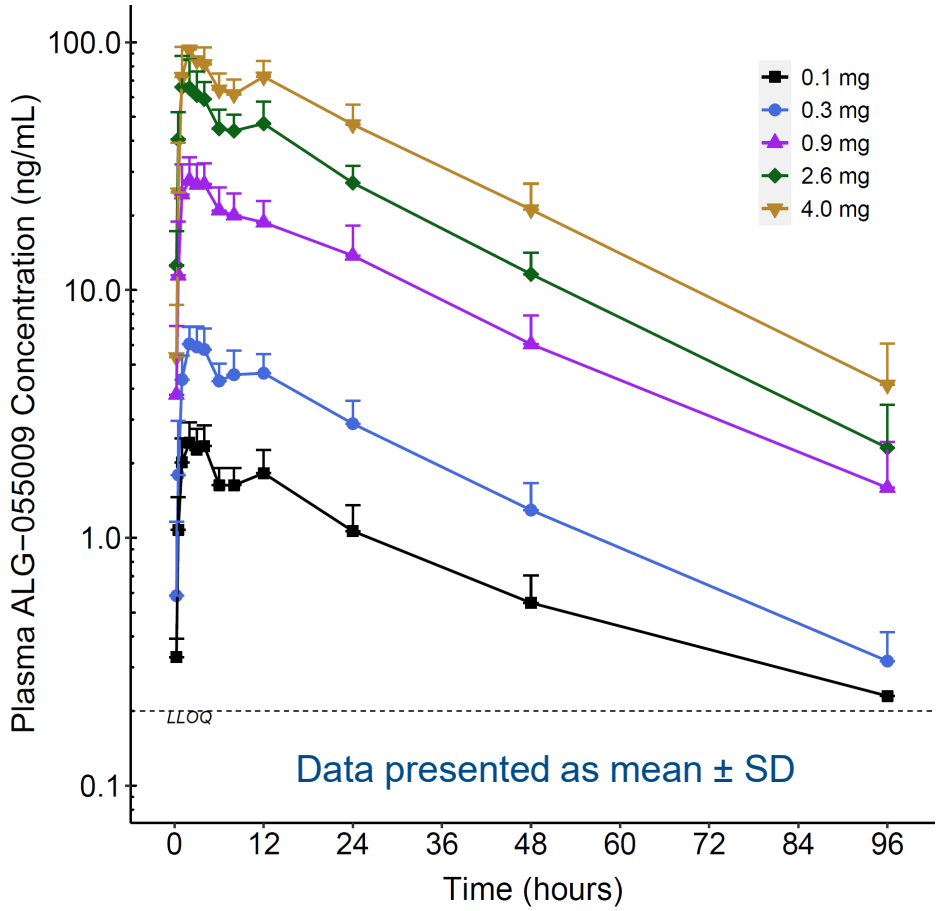
Study ALG-055009-301
Single Ascending Dose - PK, Safety, Biomarkers

- Oral doses evaluated: 0.1, 0.3, 0.9, 2.6, 4.0 mg
- PK
  - Dose proportional, with low variability
  - $t_{1/2}$ = 20-24 hours (supports once daily (QD) dosing)
- Safety: well tolerated
  - No serious adverse events (SAEs), premature discontinuations, Grade ≥3 treatment emergent adverse events (TEAEs), or clinical hyper/hypothyroidism
- Biomarkers
  - Expected thyromimetic effects observed



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

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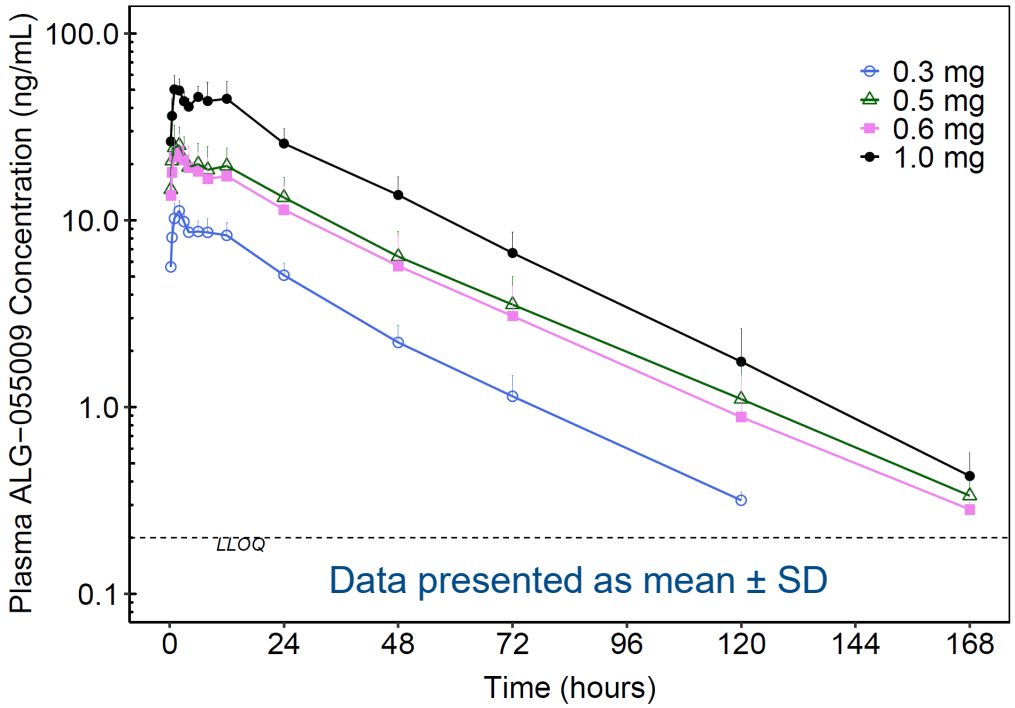
Study ALG-055009-301
Multiple Ascending Dose - PK, Safety

- Oral doses evaluated: 0.3, 0.5, 0.6, and 1.0 mg QD x 14 days
- PK: favorable profile (dose-proportional, low variability (≤27%), 2x accumulation)
- Safety: well tolerated, no clinical evidence of hypo or hyperthyroidism
  - No SAEs, discontinuations
  - All TEAEs Grade ≤2
  - No concerning labs, ECGs, vital signs, physical examinations



Multiple doses (≤1 mg) well tolerated with favorable PK

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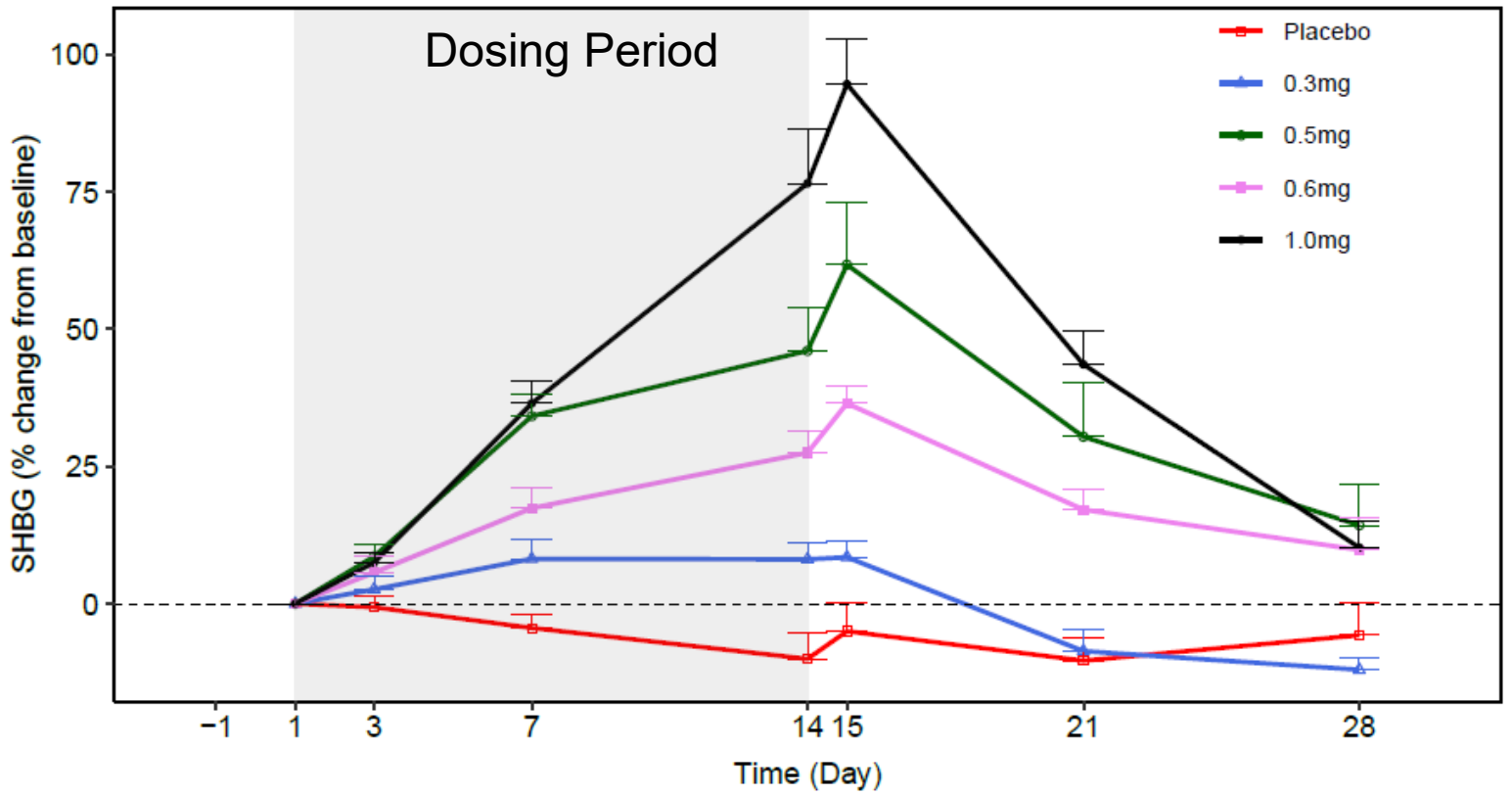
Multiple Ascending Dose - Biomarkers
Expected Thyromimetic Effects Observed



Dose proportional increases in SHBG

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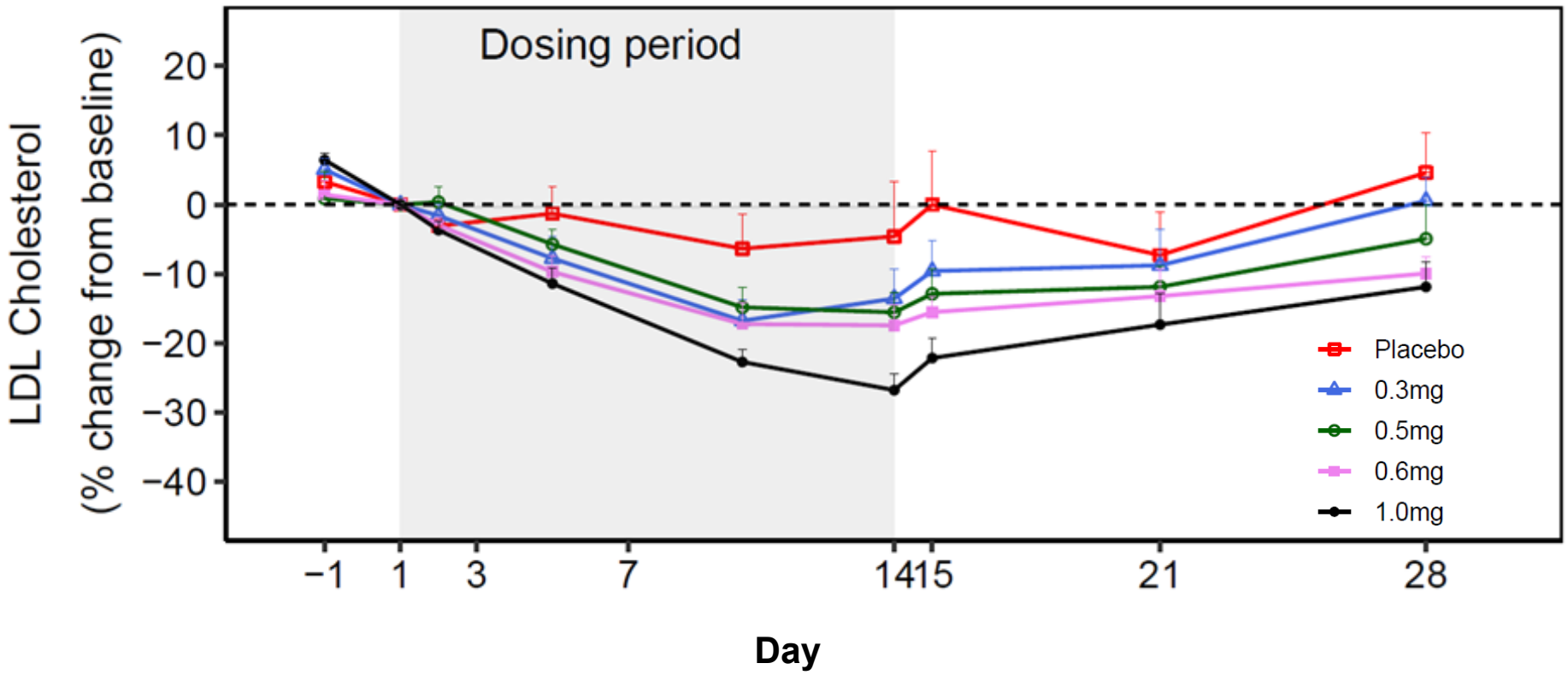
Charfi et al., AASLD 2022.

Multiple Ascending Dose - Biomarkers
Expected Thyromimetic Effects Observed



Dose responsive reductions in lipids (e.g., LDL, Apo-B, Triglycerides)

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Charfi et al., AASLD 2022.

ALG-055009
Summary

- Discovered by Aligos – issued US patent expires 2040
- More β-selective, >50 fold more potent than resmetirom
- Phase 1
  - Safety – well tolerated without clinical safety signals
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    - More uniform exposures may lead to more consistent efficacy and safety
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  - Phase 2 enabling activities (e.g., GLP toxicology, Phase 2 drug supply) – 2023
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ALG-055009 is differentiated vs. resmetirom with favorable PK, which may improve risk-benefit profile
Phase 2 filing planned in Q4 2023

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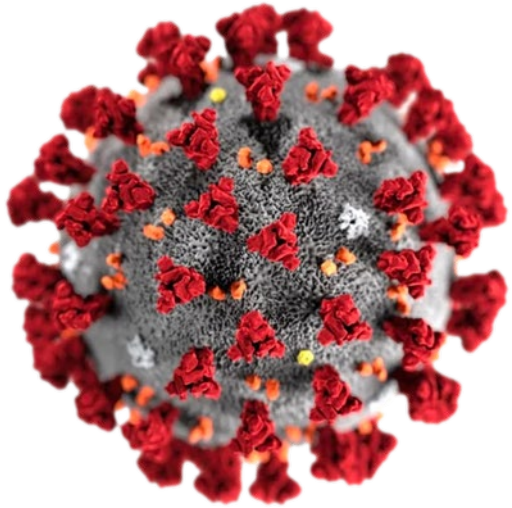
Coronavirus

- 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
  - New variants are continuously emerging
  - Large segments of global population lack access to, or are opposed to, vaccination
  - Especially needed to prevent hospitalization in high-risk groups where standard of care is contraindicated

- 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
  - 6-27 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

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ALG-097558
Superior Biochemical Potency Against SARS-CoV-2

SARS-CoV-2 3CLpro	IC_{50} (nM)[1]	Hillslope	K_i (nM)
ALG-097558	**0.26**	**1.99[2]**	**0.074**
Nirmatrelvir	2.92	0.91	2.03
PBI-0451	3.6	1.74	3.4
Ensitrelvir	4.0	1.31	2.6

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

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[1]The assays were performed with 0.3 nM 3CLpro.
[2]High hillslope (>1.5) is indicative of enzyme titration.

ALG-097558 Superior Biochemical Potency Against 3CLpro Derived from SARS-CoV-2, MERS and Other Human CoV's

	IC_{50} (μM)				
Virus	**SARS-CoV-2 (Wuhan)**	**MERS**	**HKU-1**	**229E**	**NL63**
Enzyme Concentration (nM)	**25**	**100**	**12.5**	**50**	**50**
ALG-097558	**0.009**	**0.106**	**0.008**	**0.033**	**0.049**
Nirmatrelvir	0.021	0.422	0.038	0.185	0.558
PBI-451	0.039	0.432	0.054	0.153	0.228
Ensitrelvir	0.014	4.35	0.015	4.96	>5
GC376	0.016	0.274	0.014	0.082	0.132

Experimentally, higher enzyme concentrations required for MERS, 229E, and NL63, resulting in increased IC_{50}s for all compounds vs. SARS-CoV-2 or HKU-1

ALG-097558 inhibits 3CLpro from SARS-CoV-2, MERS and other human CoV's
ALG-097558 is more active than PF-07321332, PBI-0451 and ensitrelvir across all CoV's tested

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ALG-097558
Superior Cell-Based Potency Against SARS-CoV-2 and Variants

Virus	Variant/Cell line	EC_{50} (μM)			
		PBI-0451	Ensitrelvir	Nirmatrelvir	ALG-097558
SARS-CoV-2	03021/2020 [1]	n.d.	n.d.	0.114	0.012
	B.1.1.7 (alpha) [2]	0.038	0.022	0.106	0.011
	B.1.617.2 (delta) [2]	0.126	0.141	0.217	0.013
	B.1.1.529 (omicron) [1]	0.152	0.123	0.069	0.008
	BA.2 [1]	0.137	0.035	0.045	0.007
	BA.5 [1]	0.215	n.d.	0.075	0.013
SARS-CoV-1 [1]		0.297	0.150	0.148	0.022
OC43 (β-hCoV) [3]		0.168	0.135	0.047	0.008
229E (α-hCoV) [4]		0.281	6.30	0.502	0.017

MERS testing pending; bioinformatics predicts retained activity against BA.2.12.1, BA.3, BA.4, BQ.1, BQ1.1, BF.1

ALG-097558 demonstrates pan-coronavirus antiviral activity
ALG-097558 is more active than nirmatrelvir, PBI-0451 and ensitrelvir across all CoV's tested

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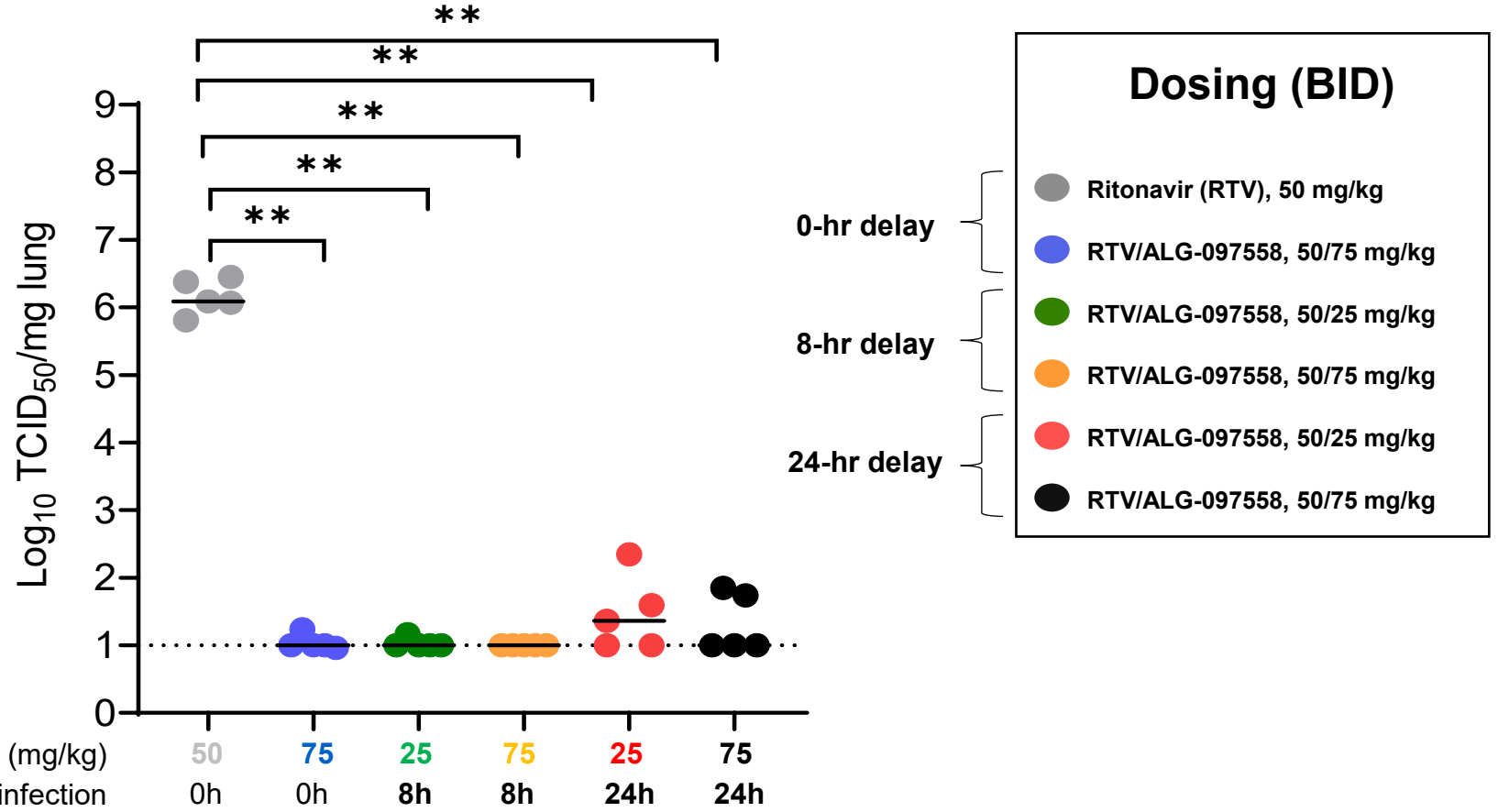
Aligos data. Cell lines used (1) VeroE6 (in presence of 2 μM of P-glycoprotein inhibitor CP-100356; (2) A549-ACE2-TMPRSS2; (3) HeLa; (4) Huh-7. No cytotoxicity was detected for ALG-097558 at concentrations up to 100 μM.

ALG-097558
Oral Therapeutic Treatment in the SARS-CoV-2 Hamster Model

Amount of Infectious Virus on Day 4



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

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5 Hamsters / group, intranasal infection, B.1.617.2 (1 x 10^4 $TCID_{50}$).
Treatment started before or after infection as indicated, administered by oral gavage BID over 3-days.
RTV administered 1 h before the ALG cpd, RTV and ALG-097558 formulated as a solution in 43% Ethanol + 27% PG in water.
Sacrifice and assessment of infectious virus in lung at day 4.

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)
ALG-097558 (Aligos)	3 (n=3)	0.11-0.14 (9-fold)
Nirmatrelvir (Pfizer)	66 (n=6)	3.5-3.7 (38-fold)
PBI-0451 (Pardes)	>65 (n=2)	7.6-8.4 (25-fold)
Ensitrelvir (Shionogi)	>67 (n=2)	15-16 (59-fold)

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

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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 Nirmatrelvir (Pfizer)
  - 6-27 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 240-380 mg BID without ritonavir
- Timelines: Phase 1 enabling activities ongoing, HV dosing starts H1 2023

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

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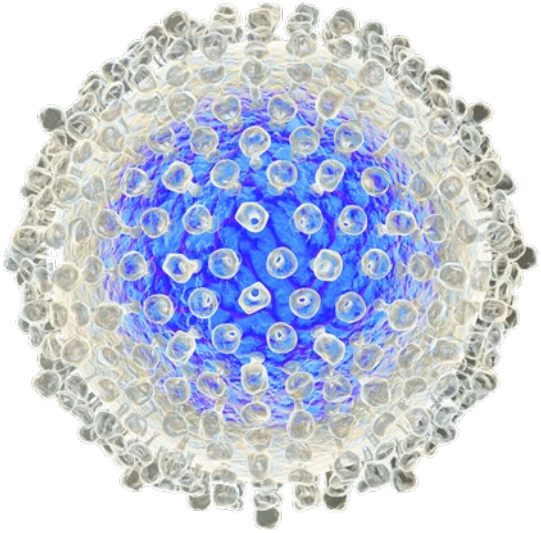
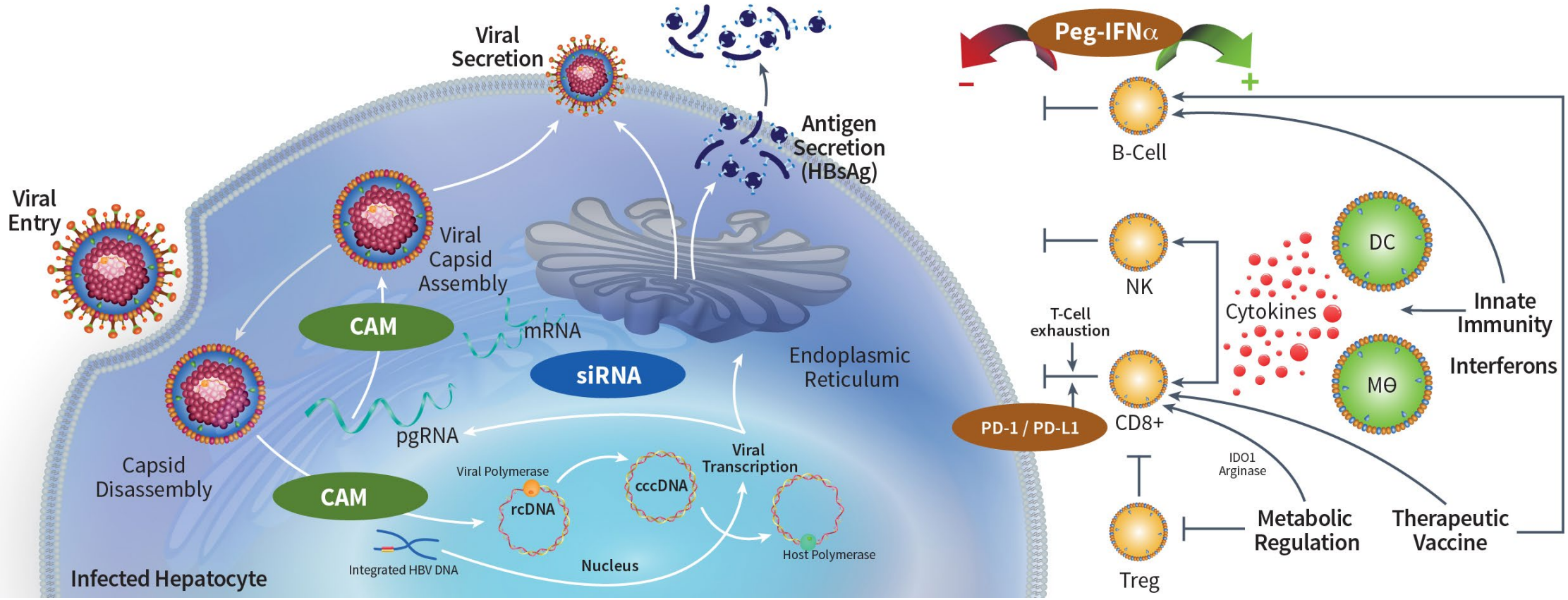
Chronic Hepatitis B



Therapeutic Approaches to CHB Functional Cure

Inhibit Viral Replication	Lower Antigen Burden	Boost Immune Response



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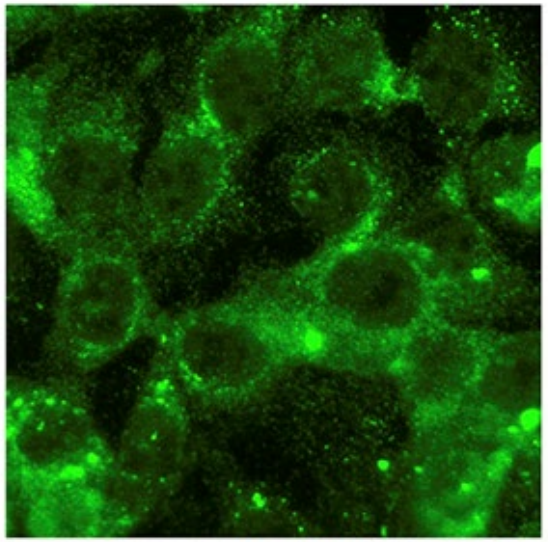
Inhibiting Viral Replication

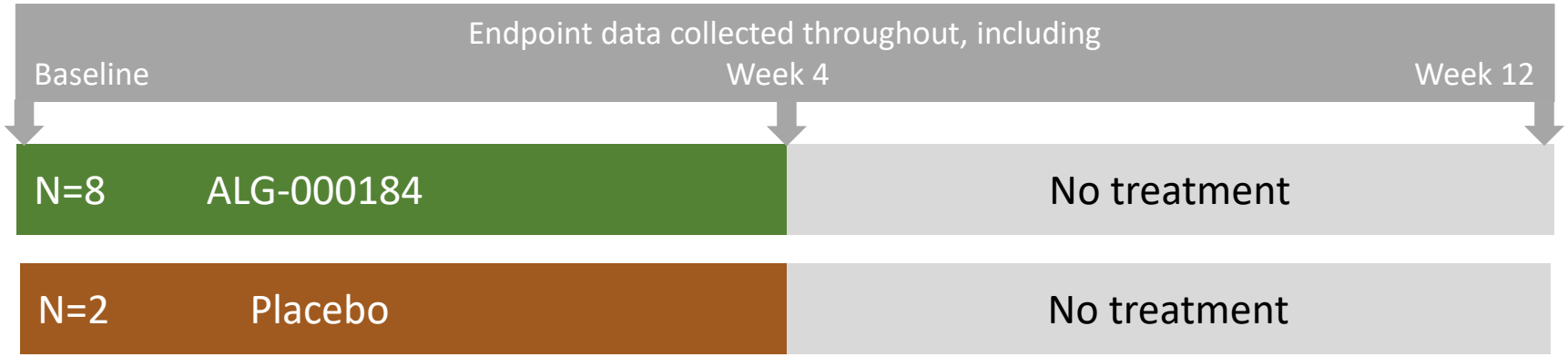
- ALG-000184 (CAM-E)



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)



Parts 1-2 (complete): SAD/MAD in HV – good safety, PK
Part 3: ALG-000184 mono-rx for 28 days in HBeAg- (100, 50, 10 mg) or HBeAg+ (100, 300, 10 mg) – good safety, PK

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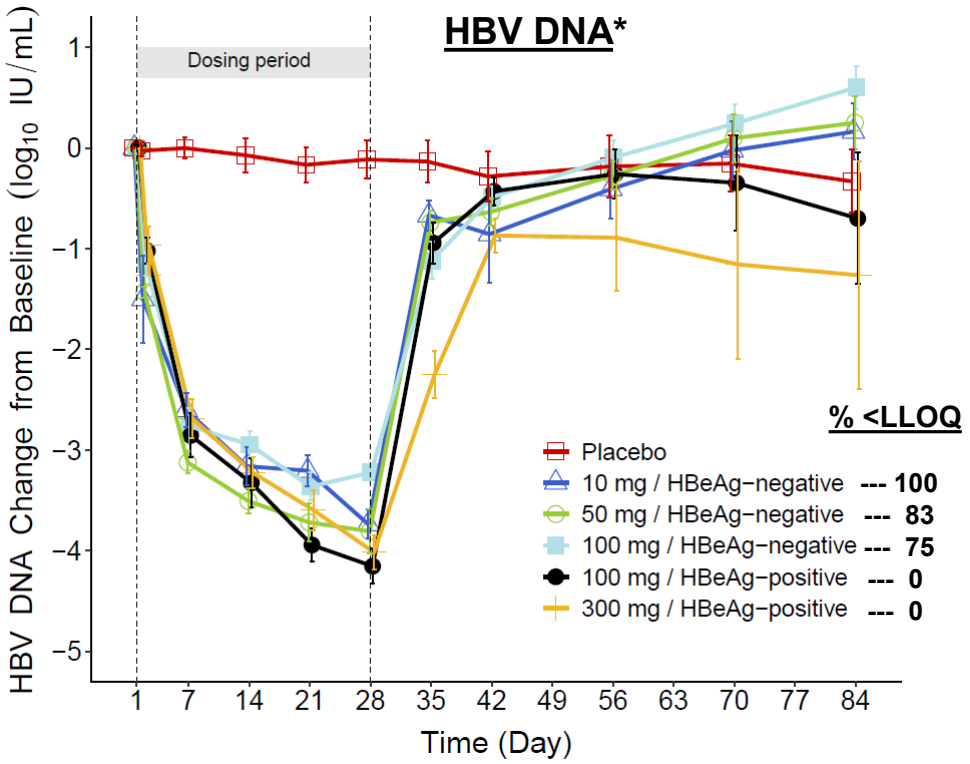
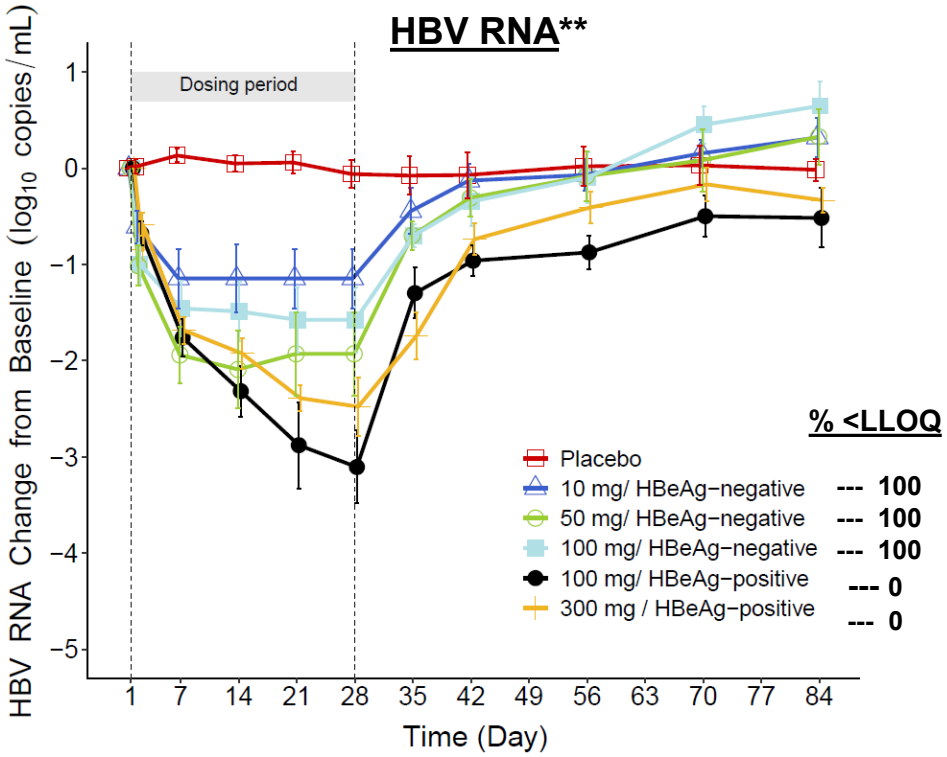
All cohorts complete except 10 mg in HBeAg+ (dosing).
Gane et al., APASL 2021; Gane er al., HBV-TAG 2021; Yuen et al., Poster #SAT-145 EASL 2022; Hou et al., Poster #1329, AASLD 2022.
clinicaltrials.gov: NCT04485663.

ALG-000184-201
Part 3 Cohorts 1-5 Antiviral Activity – HBV DNA and HBV RNA

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 ≥75% and 100% of subjects, respectively
HBeAg+ CHB subjects:	Potent DNA, RNA reductions observed (100 and 300 mg)

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*Roche Cobas® HBV DNA (LLOQ = 10 IU/mL).
**Roche Cobas® assay. The HBV RNA Investigational assay is not approved in any market (LLOQ =10 copies/mL).
Yuen et al., Poster #SAT-145 EASL 2022; Hou et al., Poster #1329, AASLD 2022.

ALG-000184
Antiviral Activity vs. Competitor CAM-Es (HBeAg Negative)

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

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

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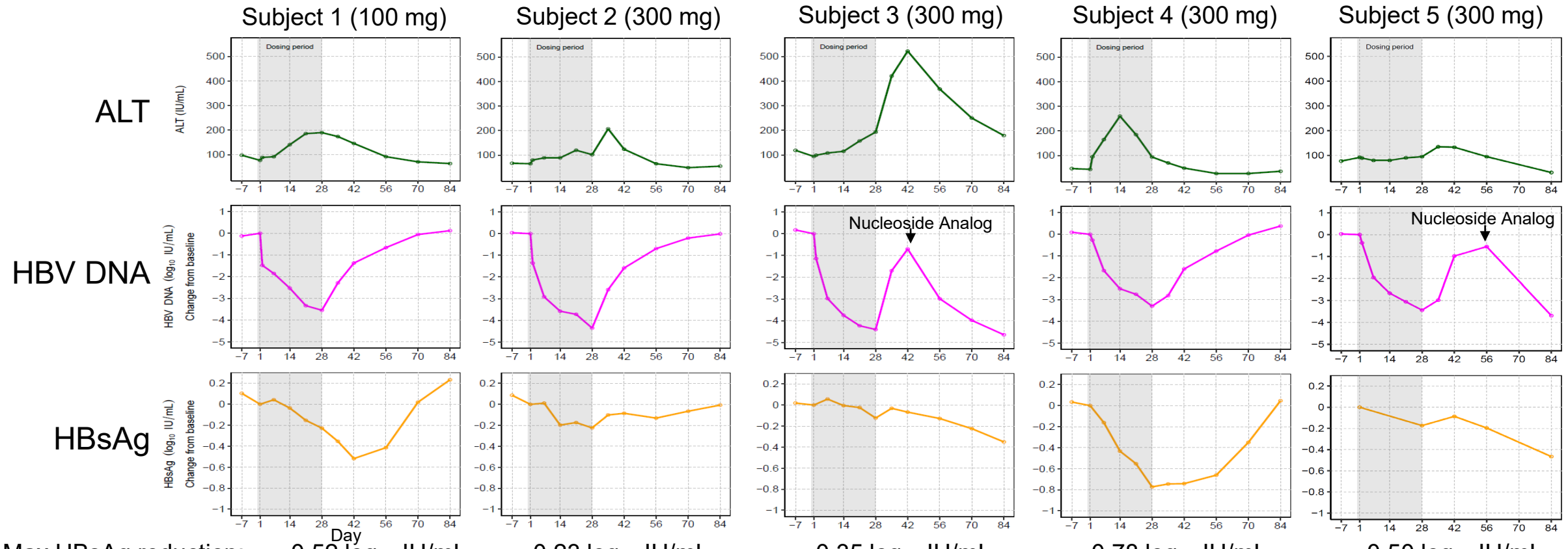
*89% of subjects were HBeAg negative in 250 mg JNJ-6379 arm. **An unknown percentage of subjects dosed with 800 mg EDP-514 were HBeAg negative. N/A – not available; LLOQ (DNA) was ≤20 IU/mL for ABI and JNJ and 10 IU/mL for Aligos.
1. MF Yuen et al. Lancet Gastroenterol Hepatol 2019; 2. MF Yuen AASLD 2018; 3. Zoulim F., et al AASLD 2018; 4. Zoulim F., et al. Gastroenterology 2020; 5. MF Yuen, et al AASLD 2021 (Poster 823). 6. HEP Dart 2021 (preliminary data).

ALG-000184
HBsAg Reductions Observed in 28-Day Monotherapy



Max HBsAg reduction: 0.52 log_{10} IU/mL | 0.23 log_{10} IU/mL | 0.35 log_{10} IU/mL | 0.78 log_{10} IU/mL | 0.50 log_{10} IU/mL

28-day monotherapy at 100 and 300 mg PO QD results in 0.2-0.8 log_{10} IU/mL HBsAg decline in 5 subjects
Best in class activity – no other CAM-E has reported same extent of HBsAg reductions in 28 days
Longer duration cohorts (± entecavir) are currently being enrolled/dosed in Part 4

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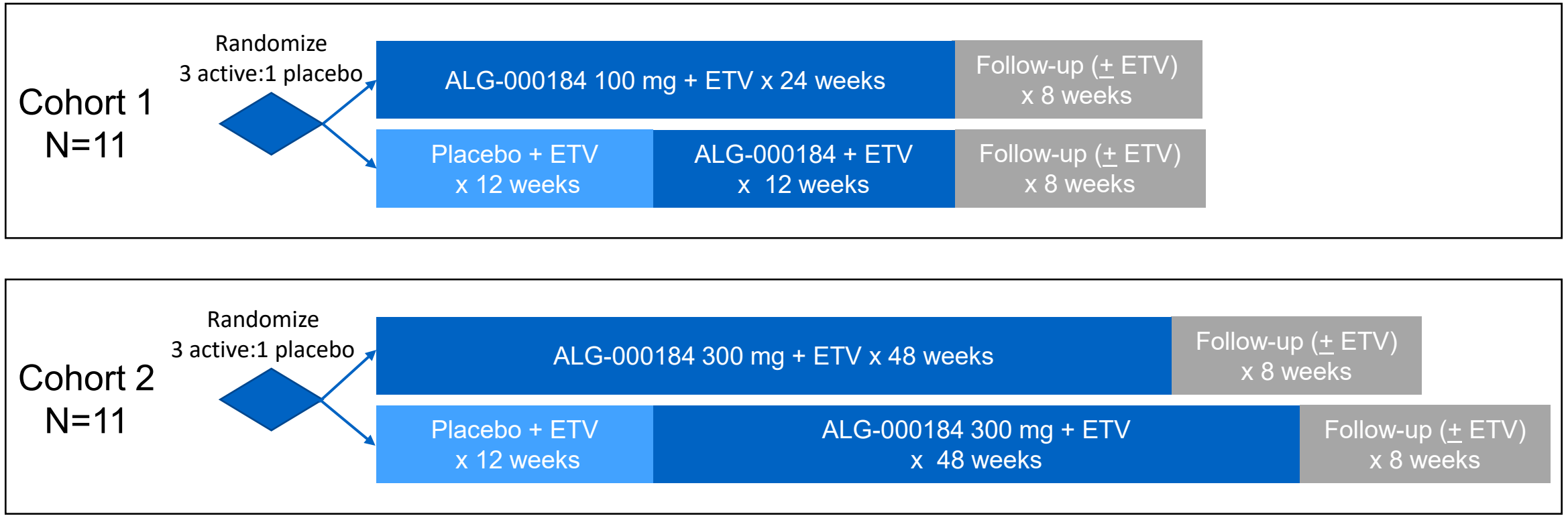
Hou et al., Poster #1329, AASLD 2022.
NCT04536337.

ALG-000184-201
Part 4 Study Design – up to 48 Weeks of Dosing



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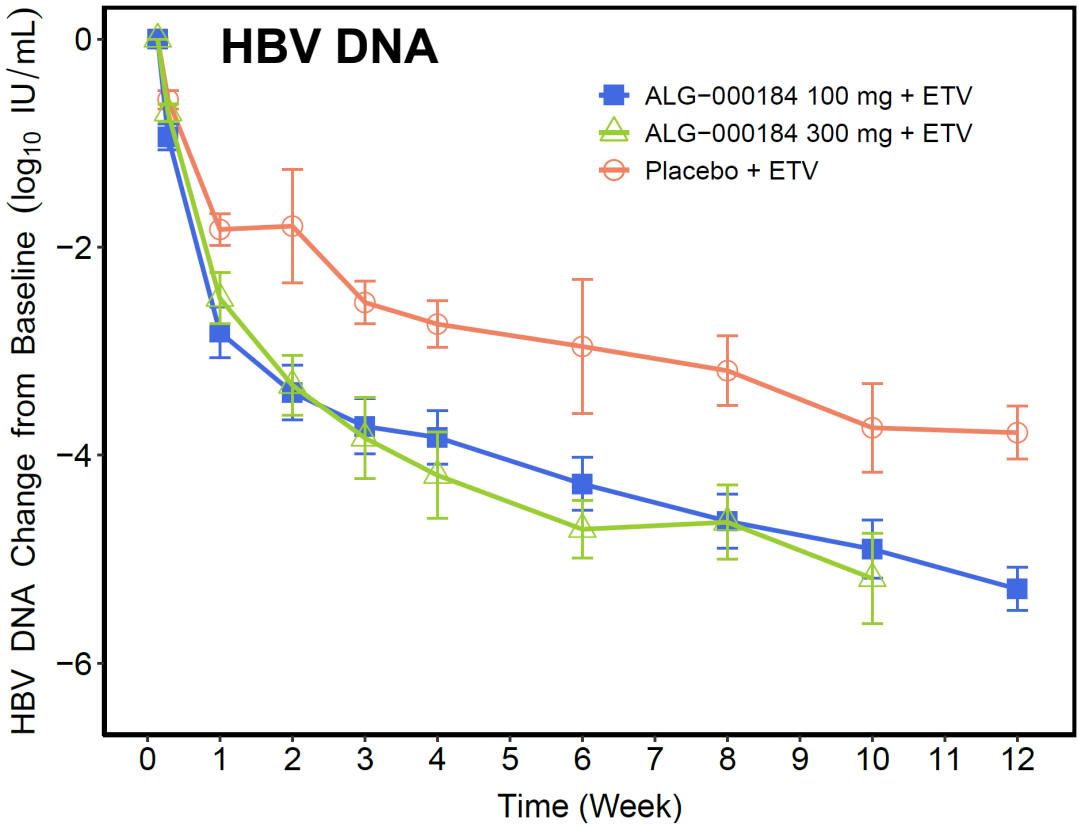
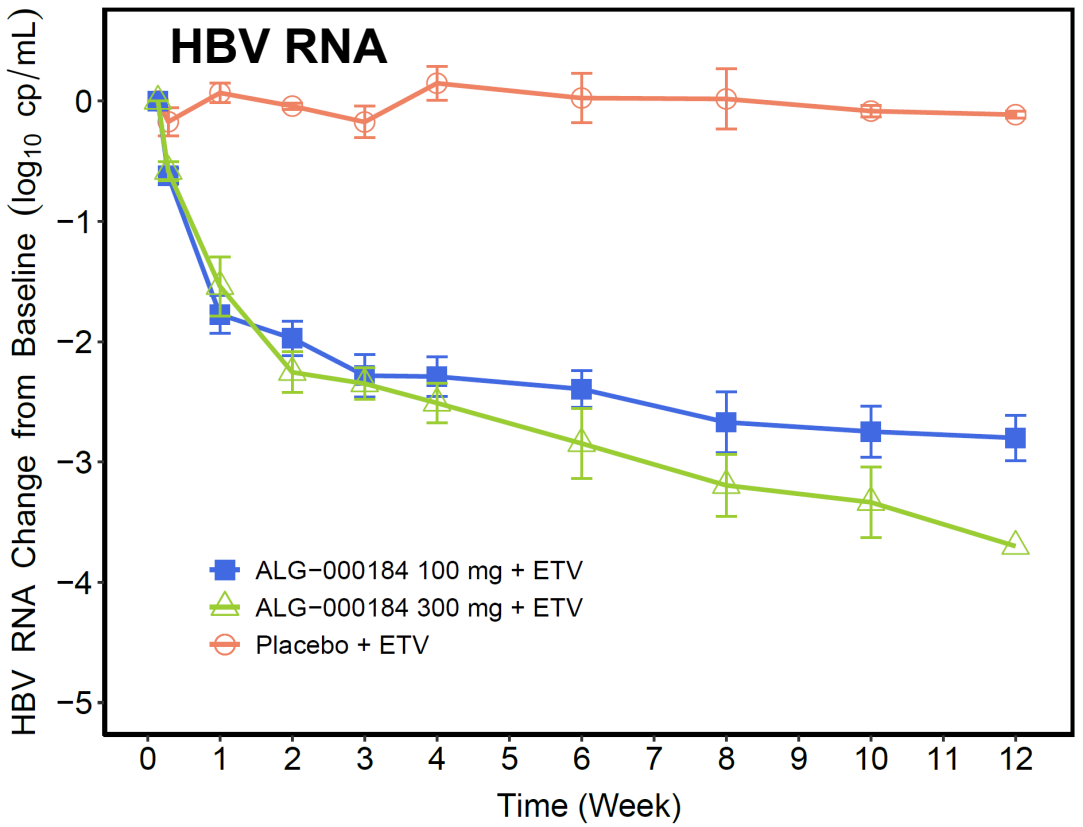
Available preliminary safety, PK, and antiviral activity data as of 23 Dec 2022 are presented.
ETV = entecavir.

ALG-000184-201 (Part 4)
ALG-000184 + ETV Lowers DNA/RNA More Than ETV Alone





Reduction at Week 10	Placebo + ETV, N=3	ALG-000184 100 mg + ETV, N=7	ALG-000184 300 mg + ETV, N=6
HBV DNA, mean (SEM) log_{10} IU/mL	-3.7 (0.43)	-4.9 (0.28)	-5.2 (0.3)
HBV RNA, mean (SEM) log_{10} copies/mL	0.08 (0.05)	-2.7 (0.21)	-3.3 (0.29)

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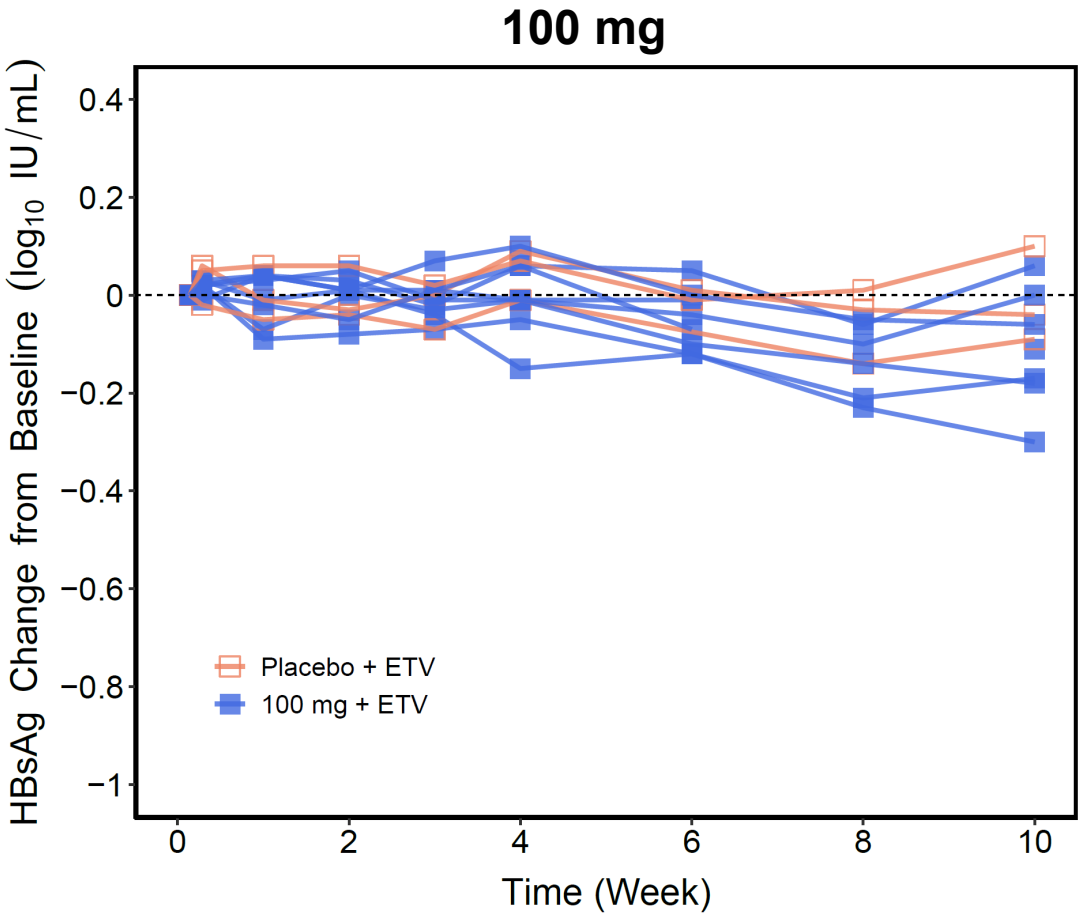
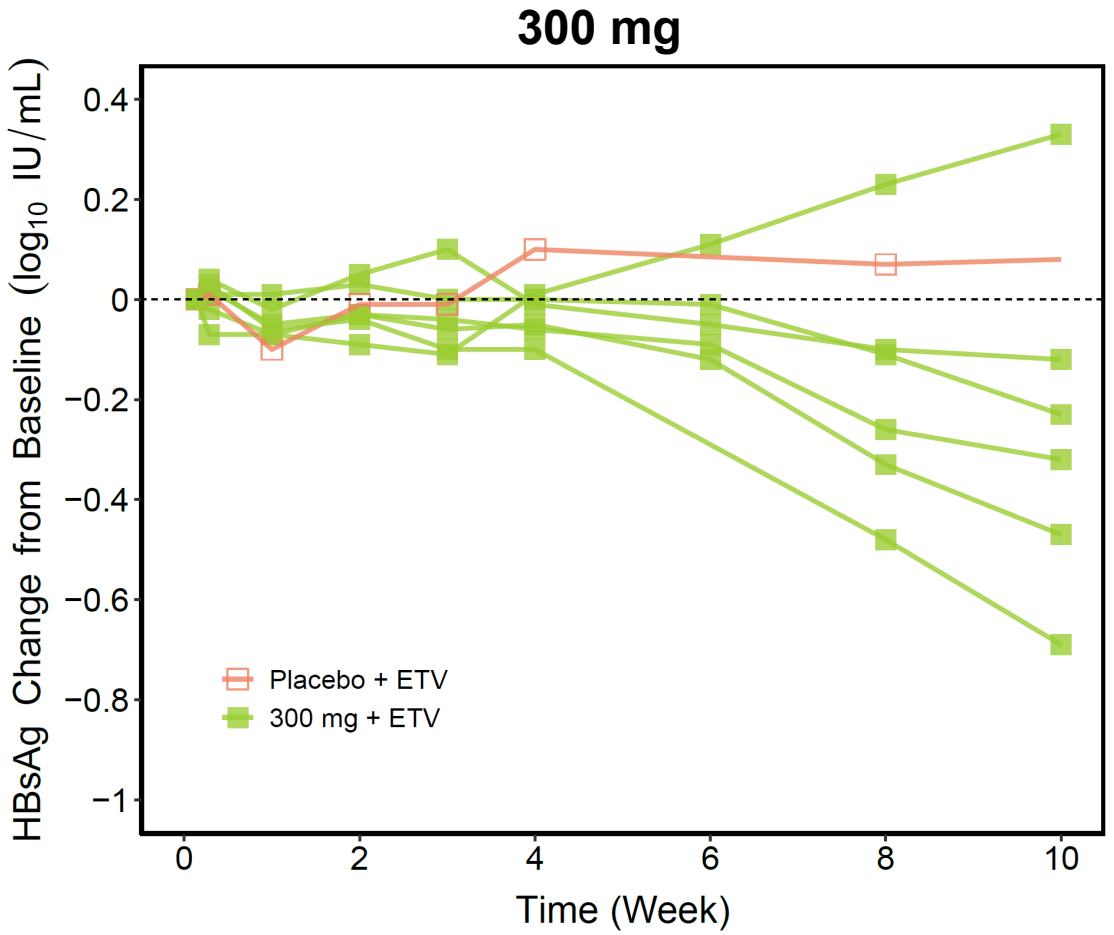
Data on file.

ALG-000184-201 (Part 4)
300 mg ALG-000184 + ETV Lowers HBsAg Over Time





Clear downward trend over 10 weeks for HBsAg among majority of 300 mg ALG-000184 treated subjects

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Data on file.

ALG-000184-201
Ongoing Phase 1b Cohorts

- Enrollment/dosing in multiple longer duration (≤48 weeks) cohorts is ongoing
- Cohorts designed to address several key questions, including
  - Impact of treatment with/without entecavir
  - Antiviral activity in different patient populations (high/normal baseline ALT, HBeAg positive or negative)
- Available cohort data to be presented at APASL, EASL, and AASLD in 2023

Longer dosing duration (≤48 weeks) cohorts currently being enrolled and dosed
Data will be presented at scientific conferences throughout 2023

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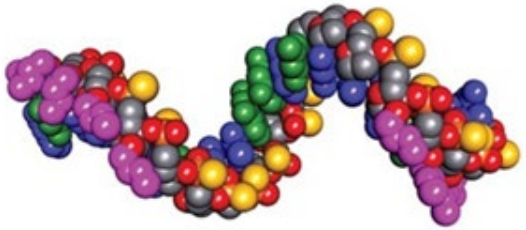
Lowering S-Antigen Burden

- 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

Aligos oligonucleotide know-how and proprietary technologies have resulted in a differentiated siRNA

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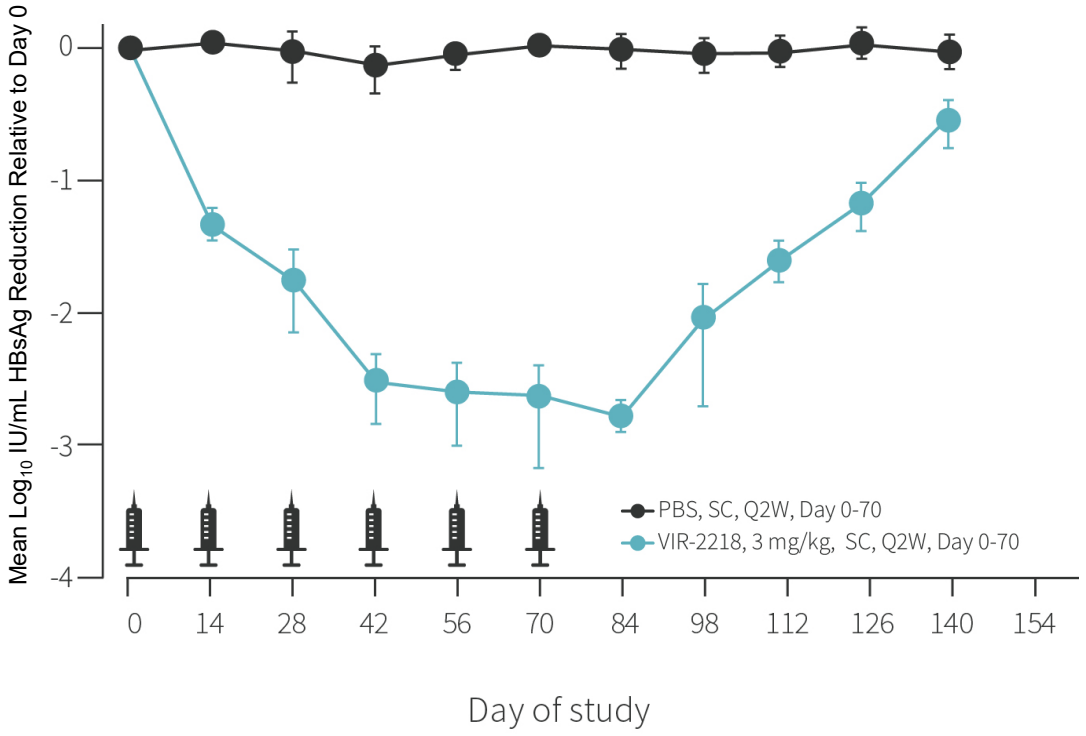
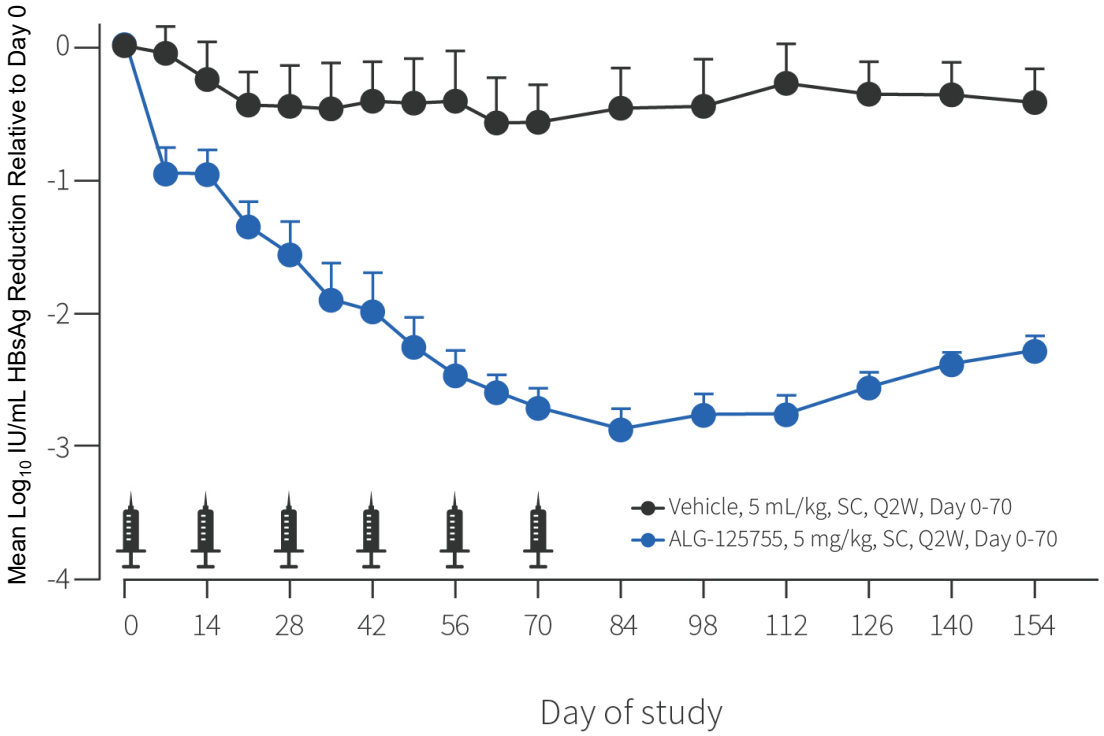
ALG-125755
Repeat Dosing in the AAV-HBV Mouse Model vs. VIR-2218

VIR-2218*



ALG-125755



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

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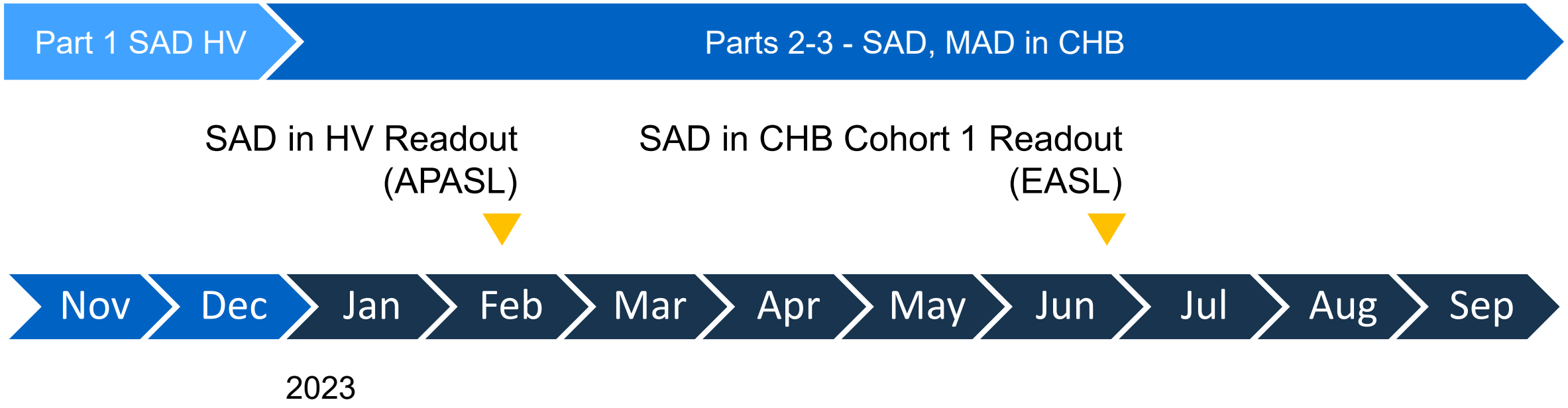
*Data from Anglero-Rodriguez Y. et al., "In Vitro and In Vivo Characterization of VIR-2218, an Investigational RNAi Therapeutic Targeting Hepatitis B Virus." EASL 2020, Poster SAT426.

ALG-125755-501
Study Timelines



SAD in HV: Part 1 Cohorts 1-4 (20 mg, 60 mg, 100 mg and 200 mg) completed dosing
SAD in CHB: Part 2 Cohort 1 enrollment completed (50 mg)

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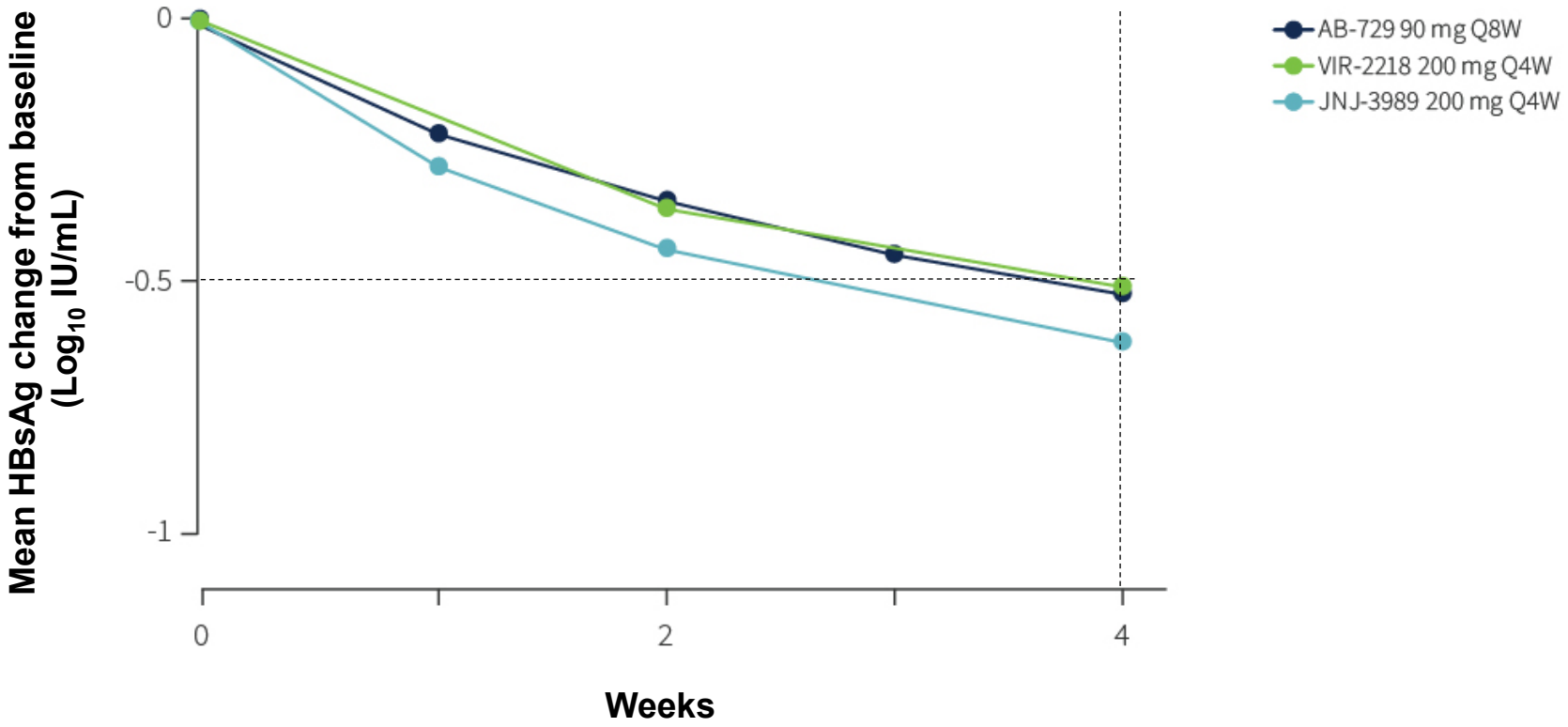
Clinical Experience with siRNAs
HBsAg Reduction After 28 Days in CHB Patients



Consistent reductions of ~0.5 log_{10} IU/mL noted for competitor siRNAs 28 days after a single dose

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Data derived from multiple studies: VIR-2218, Yuen et al., AASLD 2022; AB-729, Yuen et al., AASLD 2020; RG6346, Yuen et al., AASLD 2020; JNJ-3989, Yuen et al., AASLD 2021.

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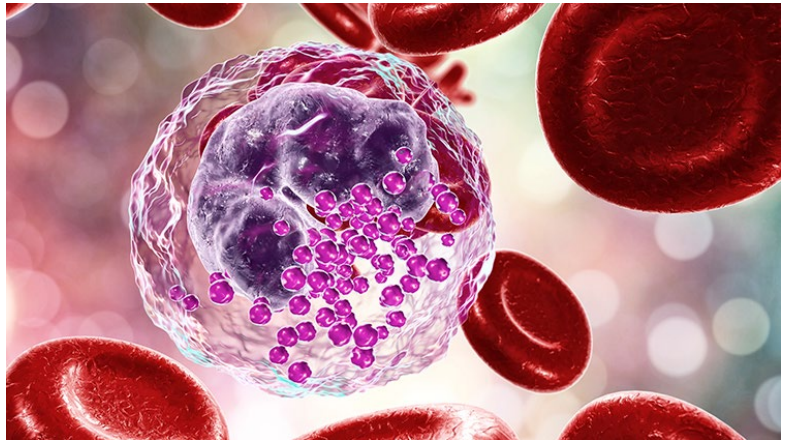
Boosting the Immune Response

- Liver Targeted Oral PD-L1 Inhibitor (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
- Lead compound is a novel liver-targeted small molecule PD-L1 inhibitor
  - Biochemical and cell-based potency established
  - Activation of HBV specific T-cells demonstrated with similar potency as durvalumab
  - Liver targeting achieved with lead compound

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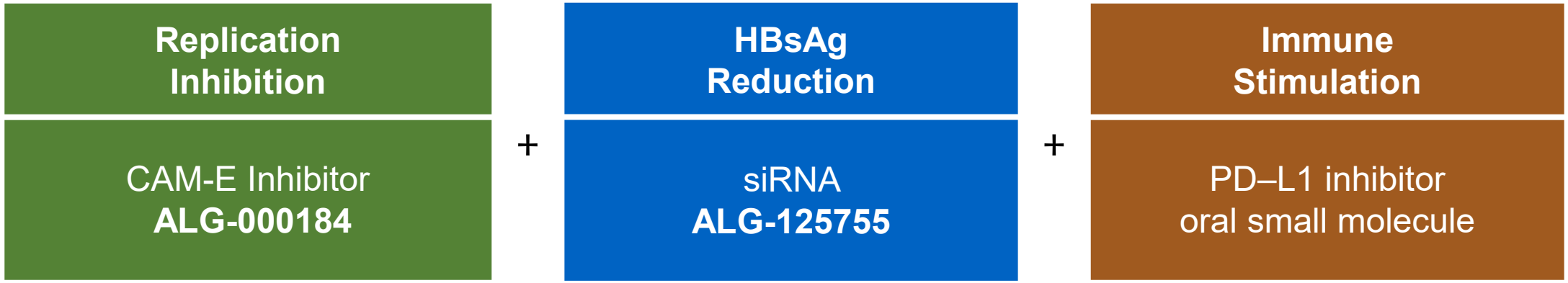
Gane et. al, Anti-PD-1 blockade with nivolumab with and without therapeutic vaccination for virally suppressed chronic hepatitis B: A pilot study. J Hepatol. 2019 Nov;71(5):900-907.

Aligos CHB Portfolio Consists of the Key Pillars Which are Likely Necessary for Enhanced Functional Cure Rates



Executive Summary
Aligos Advancing Multiple Promising Drugs in Areas of Unmet Need

- NASH
  - THR-β agonist (ALG-055009) – more uniform exposure vs. competitor THR-β drugs may lead to more consistent efficacy and safety
  - Merck oligonucleotide collaborations are progressing
- Coronavirus Protease Inhibitor (ALG-097558)
  - On track to complete FIH enabling nonclinical studies and dose in Phase 1 in H1 2023
- CHB – 3 MOAs which, when combined, may increase functional cure rates
  - CAM-E (ALG-000184) - best in class HBsAg, DNA, RNA. Ph1b cohorts (≤48 weeks) ongoing
  - siRNA (ALG-125755) is differentiated in AAV-HBV. Dosing in CHB ongoing
  - PD-L1 liver-targeted, small molecule - advancing towards candidate selection
- As of September 30, 2022; cash balance was $142.3M*; fully diluted common shares: 52,897,859

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*Reflects unrestricted cash, cash equivalents and investments.


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