ALIGOS
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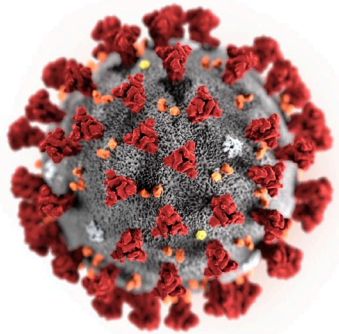
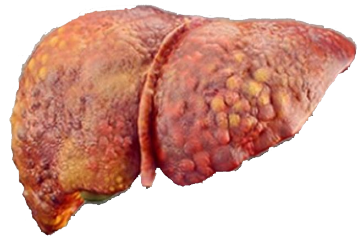
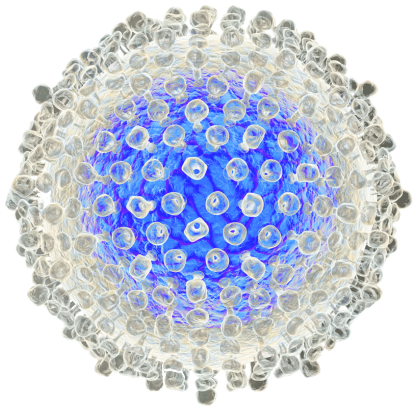






Lawrence M. Blatt, Ph.D.
CEO & Co-Founder

JP Morgan 39th Annual Healthcare Conference

January 11-14, 2021

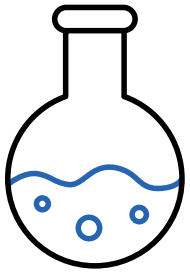
Disclosures

This presentation contains forward-looking statements. 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, 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' prospectus filed with the Securities and Exchange Commission on October 19, 2020, 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.

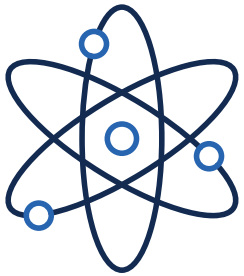
This presentation concerns drug candidates, some of which are undergoing nonclinical studies and others of which are under clinical investigation, and all of which have not yet been approved for marketing by the U.S. Food and Drug Administration. These drug candidates are currently limited by federal 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 Platforms

Proprietary oligonucleotide and small molecule platforms

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 is to become a world leader in the development of targeted, antiviral therapies for CHB and COVID-19 and to leverage our expertise in liver diseases to create targeted therapeutics for NASH

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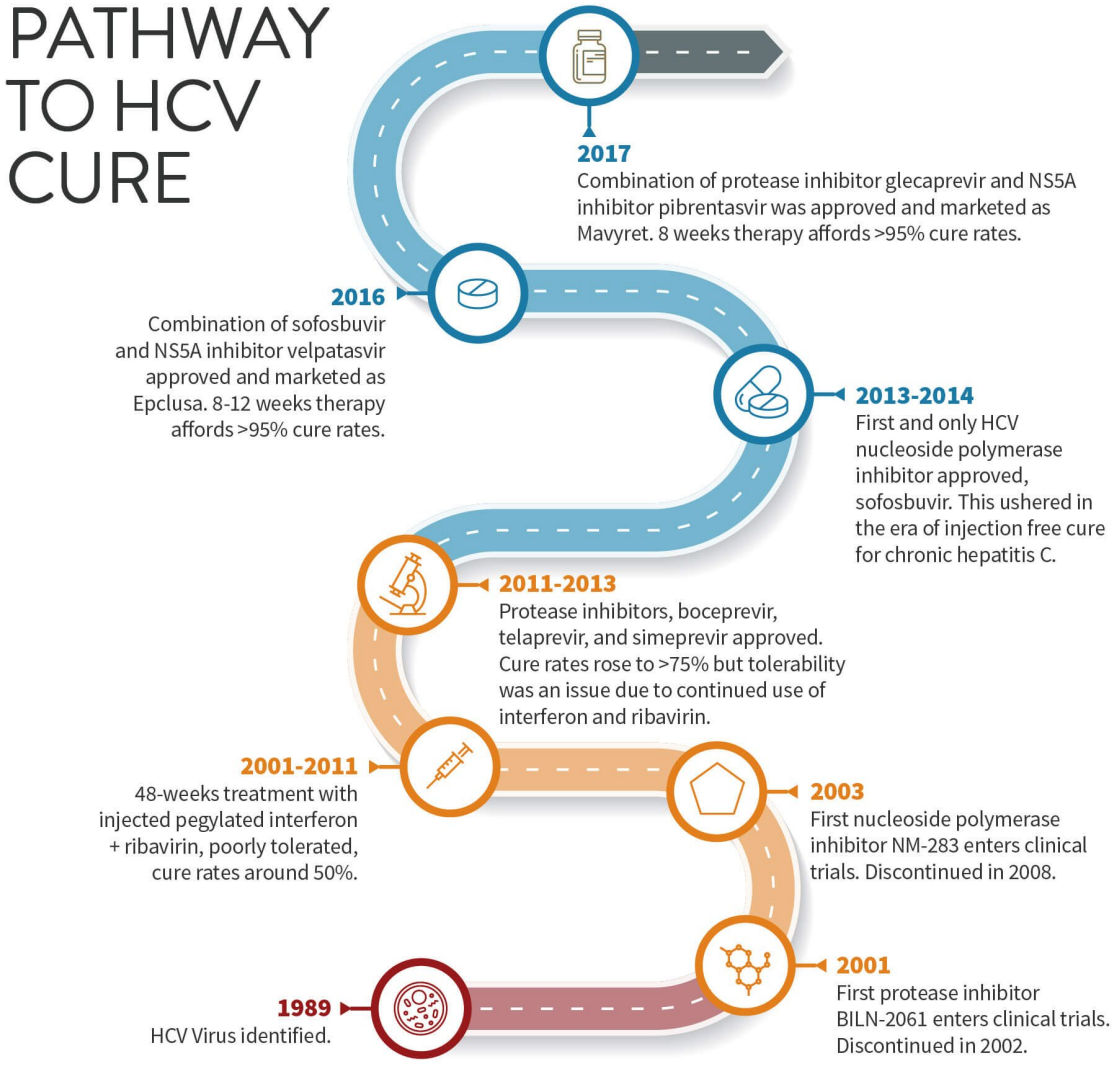
Our Platforms, Capabilities and Collaborations

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
- Standard of care treatment until 2011
  - Pegylated interferon + ribavirin
  - 48-weeks, ~50% cure rate, poorly tolerated
- Protease inhibitors
  - First clinical entrant – BILN-2061 (2001)
  - First approvals were boceprevir and telaprevir (2011)
  - Simeprevir was third (2013)
- Nucleos(t)ide analogs
  - First clinical entrant – NM283 (2003)
    - Followed by balapiravir (2004) and IDX-184 (2010)
    - Only approved compound, sofosbuvir (2013)
- Current standard of care includes NS5A inhibitors
  - Cure in 8-12 weeks, >95% cure rate
  - Epclusa: sofosbuvir + velpatasvir (Gilead, 2016)
  - Mavyret: glecaprevir + pibrentasvir (AbbVie, 2017)



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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
ALG-125755	CHB	siRNA						Phase 1 Start
ALG-055009	NASH	THR-β Agonist						Phase 1 Start
Discovery	Coronavirus	Multiple						—
Discovery	Liver Diseases	Multiple						—
Discovery	NASH	Undisclosed		MERCK				—

LEGEND Oligonucleotides Small Molecules Multiple Modalities

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ASO = antisense oligonucleotide; CAM = capsid assembly modulator; CHB = Chronic Hepatitis B; MOA = Mechanism of Action; NASH = nonalcoholic steatohepatitis; siRNA = small interfering ribonucleic acid (RNA); STOPS = S-antigen Transport-inhibiting Oligonucleotide Polymers; THR-β = thyroid hormone receptor beta.

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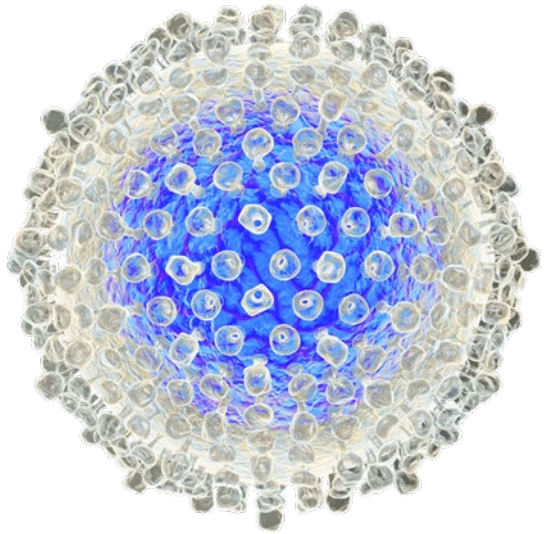
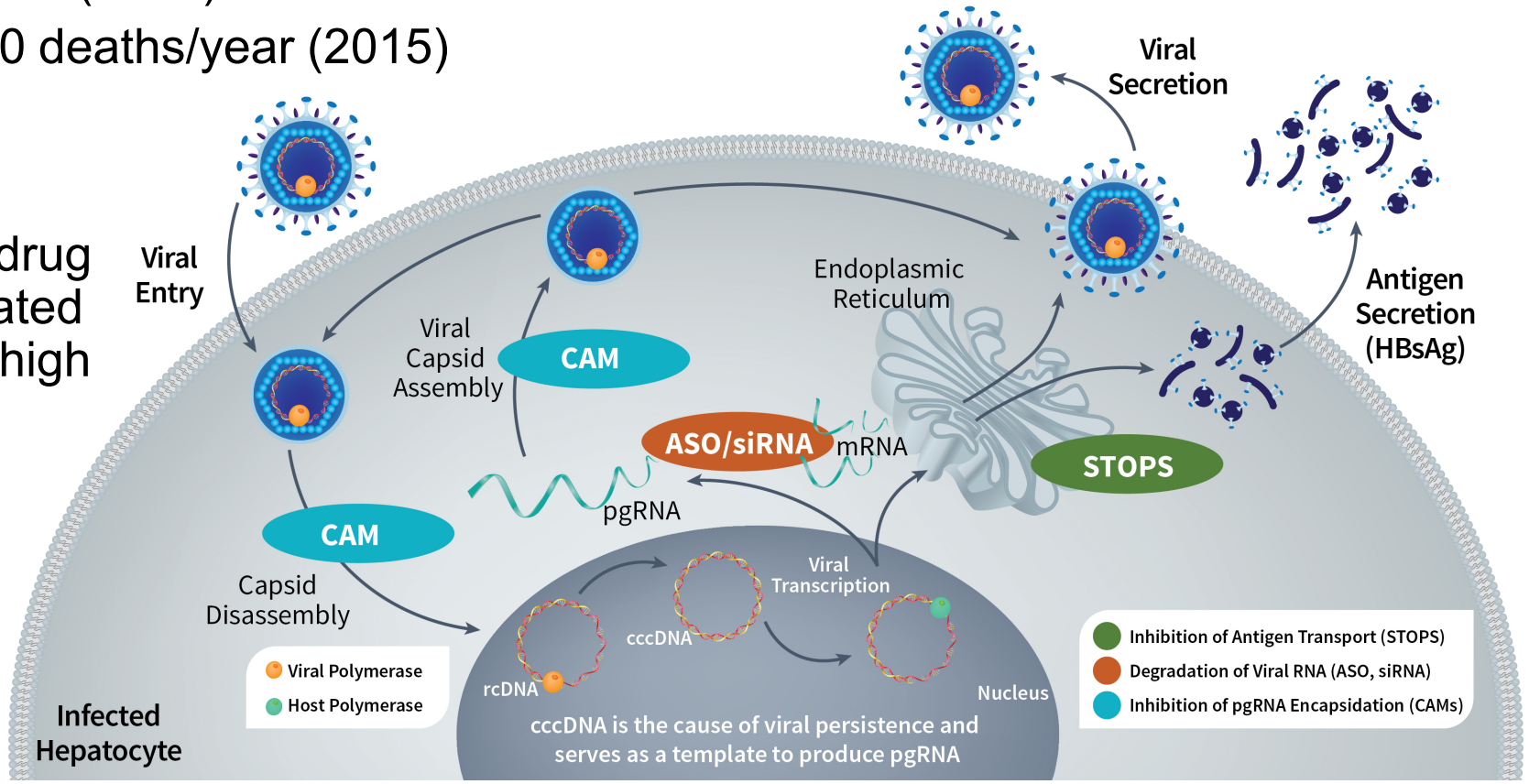
Chronic Hepatitis B



CHB Epidemiology & Our Portfolio

- CHB Epidemiology* - Most common chronic viral infection in world
  - >290 million carriers worldwide (2020)
  - ~8 million in US, EU, Japan (2015)
  - Responsible for ~900,000 deaths/year (2015)

- Our Portfolio
  - Potentially best-in-class drug candidates against validated targets with potential for high functional cure rates
    - › STOPS™
    - › CAM
    - › Oligonucleotides
      - ASO
      - siRNA



*Hepatitis B Foundation (hepb.org), World Health Organization Global Hepatitis 2017 Report (who.int, euro.who.int), accessed August 2020.

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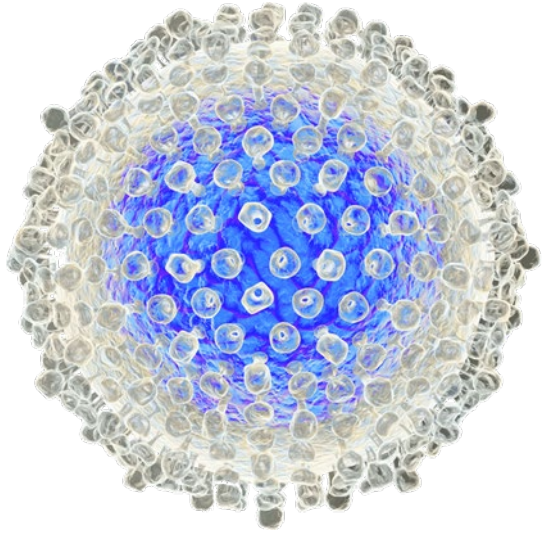
STOPS

- ALG-010133, a novel, chemically optimized poly-AC oligonucleotide analog
- Completed healthy volunteer portion of Phase 1 and transitioning into CHB patients
- Presentation accepted for APASL 2021

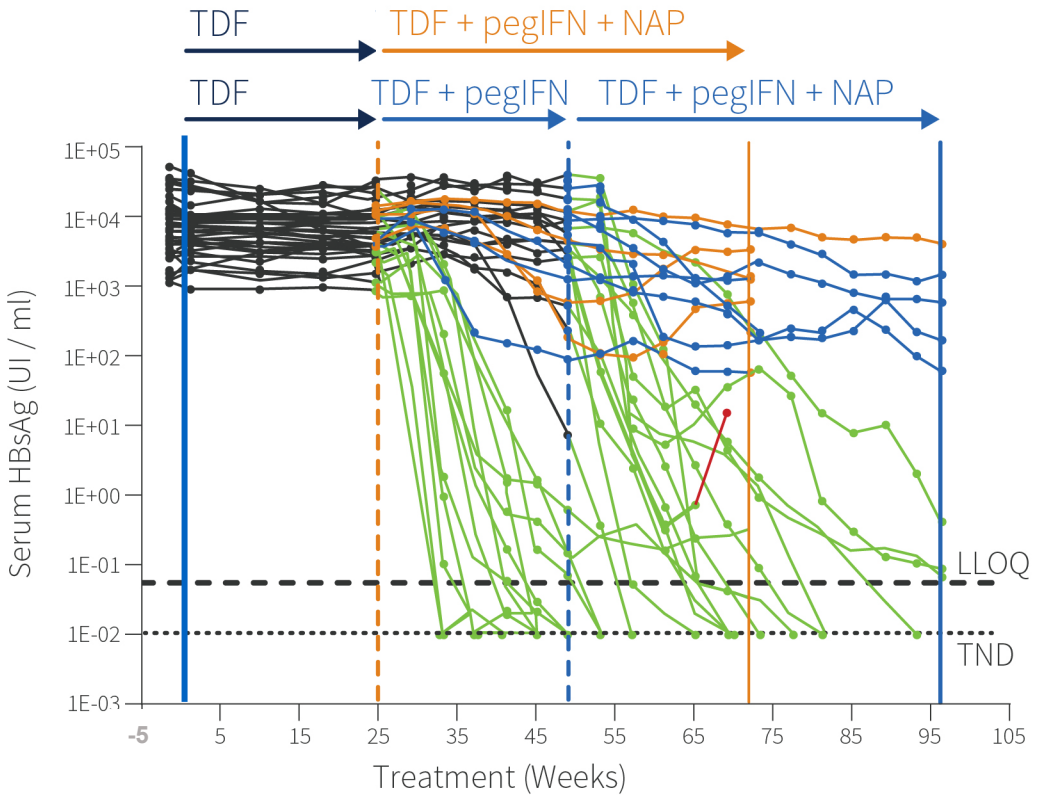


Poly AC Oligonucleotides Have Clinical Validation

- Replicor 102 Study*
  - REP 2139-Ca Monotherapy
  - 500 mg Q-weekly, 2-hr IV administration
  - Significant reduction in HBsAg observed

- Replicor 401 Study (Figure**)
  - REP 2139-Mg or REP 2165-Mg
  - 250 mg Q-weekly, 2-hr IV administration
  - Triple combination
    - With tenofovir and pegylated-IFN
  - Reported a ~39% functional cure rate***

HBsAg Reduction in REP 401 Study



Treatment results in multiple log_{10} reductions of HBsAg and higher rates of functional cure

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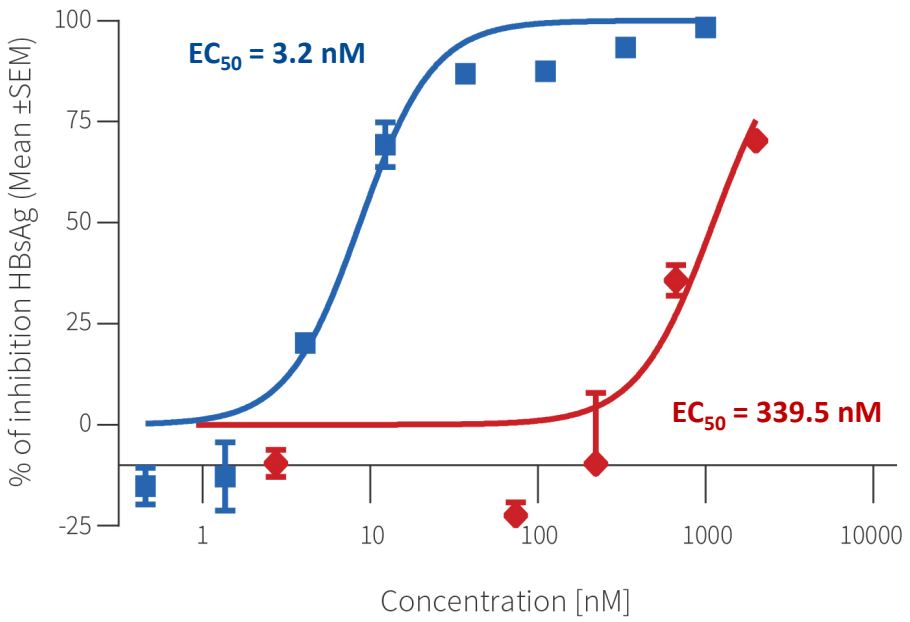
*REP 102 Study; Al-Mahtab et. al., PLOS One, June 3, 2016.
**Figure adapted from Bazinet M. et. al. OP-02 EASL 2019 and Bazinet M. et. al. EASL 2020, LBP-033.
***Bazinet M., et. al., Gastroenterology 2020 (158) 2180.

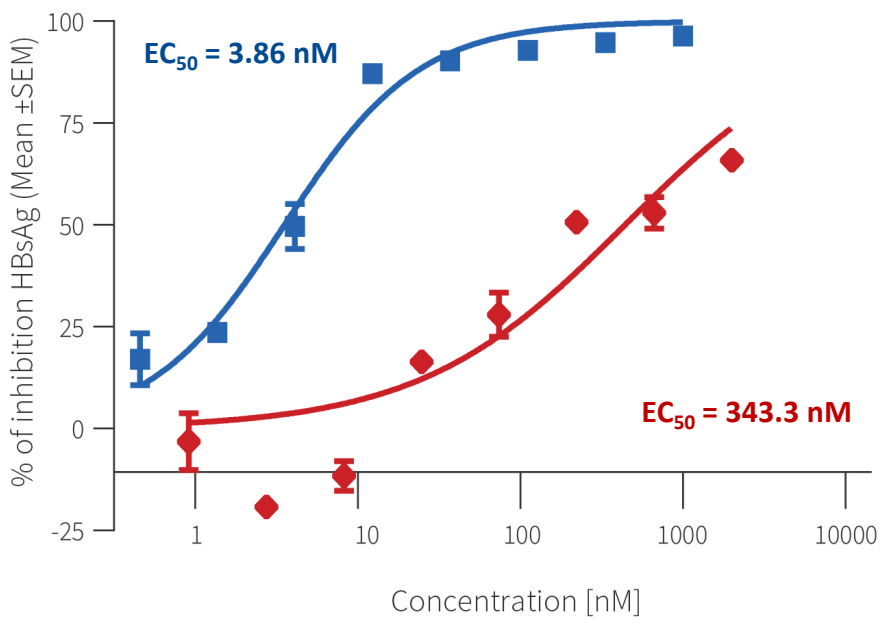
ALG-010133
Our Lead STOPS Molecule

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

Similar potency improvement also seen in primary human hepatic cells

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*ALG-010004 has an identical oligonucleotide sequence as REP 2139. A. Vaillant, Antiviral Research 133 (2016) 32.

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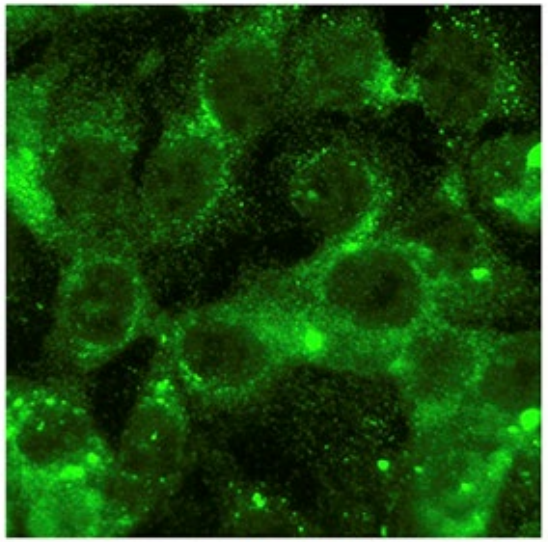
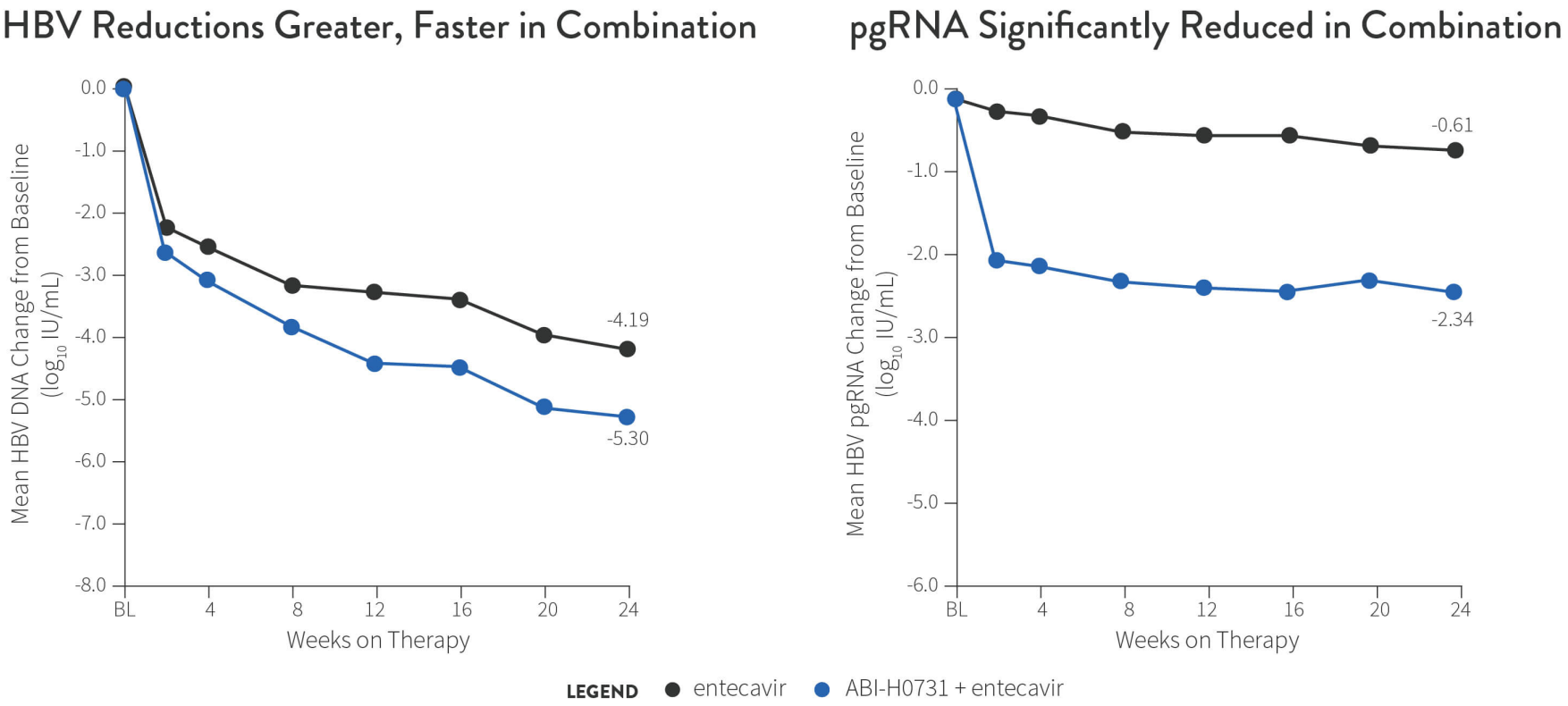
CAM

- ALG-000184, a prodrug of ALG-001075
- Currently dosing in healthy volunteers
- Presentation accepted for APASL 2021



CAMs Have Demonstrated Clinical Anti-HBV Activity

Assembly ABI-H0731 at 300 mg QD in HBeAg (+) treatment naïve CHB patients in combination with the nucleoside analog entecavir



Unlike nucleoside analogues when used in monotherapy, reductions in HBV pgRNA were also observed

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ABI-H0731 = Vebicorvir.

Modified from Assembly Biosciences, Corporate Presentation, September 2020; https://clinicaltrials.gov/ct2/show/NCT03780543.

Our CAM Demonstrates Superior 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	HepG2.2.15
Aligos ALG-000184	**Phase 1**	**0.63**	**HepG2.117**
		0.53	**HepG2.2.15**

With the exception of ALG-000184, data was sourced from publicly available literature, posters and presentations.
ALG-000184 data was generated by Aligos on the parent compound ALG-001075.

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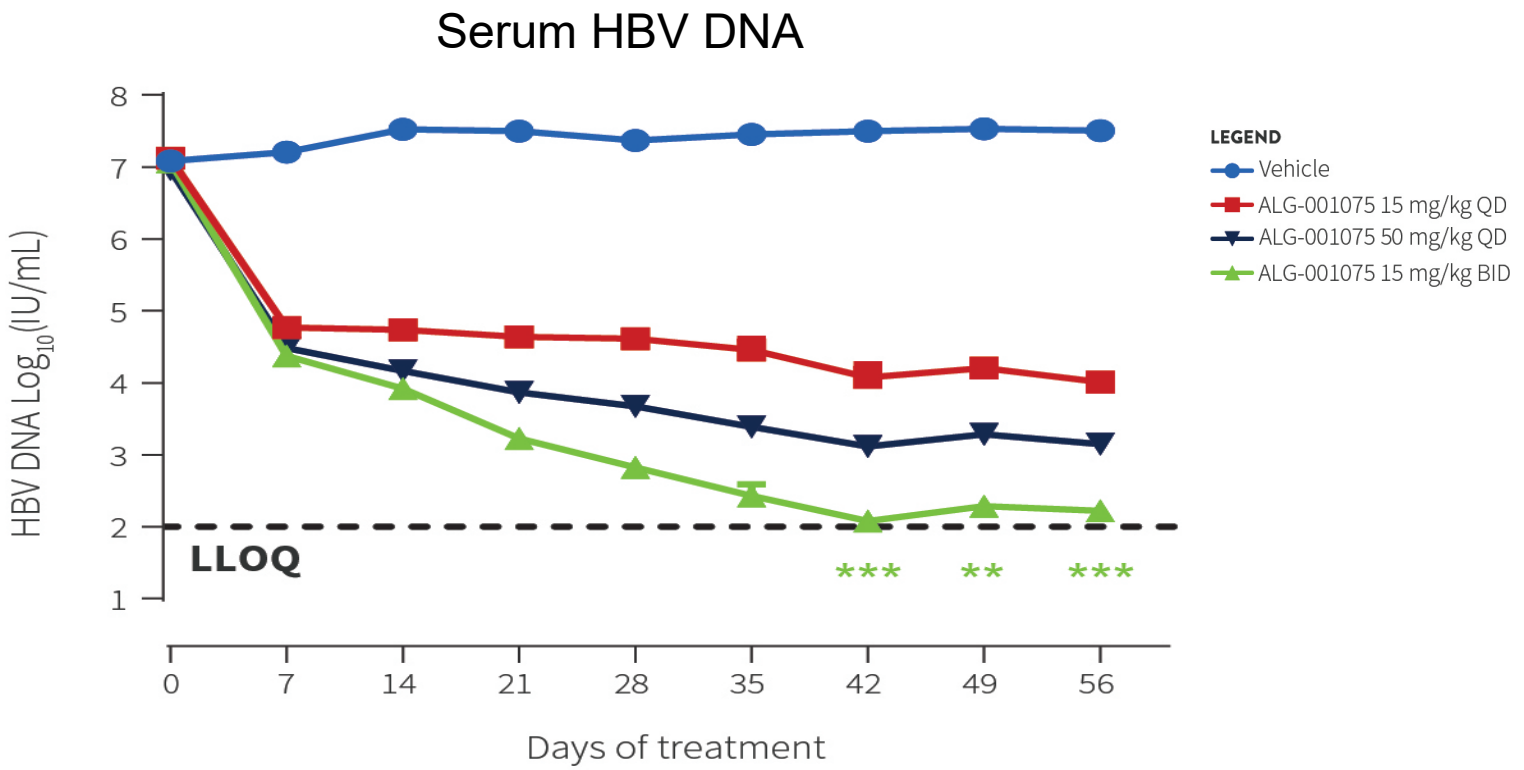
ALG-001075
Potent In Vivo Activity in the AAV-HBV Mouse Model



Dose dependent reductions in HBV DNA observed across treatment groups
>5 $\log_{10}$ IU/mL reduction in HBV DNA relative to placebo in the 15 mg/kg BID treatment group

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HBV DNA levels in AAV-HBV infected mice when dosed orally for 56 days. Values represent means ±SEM from six animals per group. Number of stars represent the number of mice with HBV DNA levels reaching the lower limit of quantification (LLOQ) at that time point.

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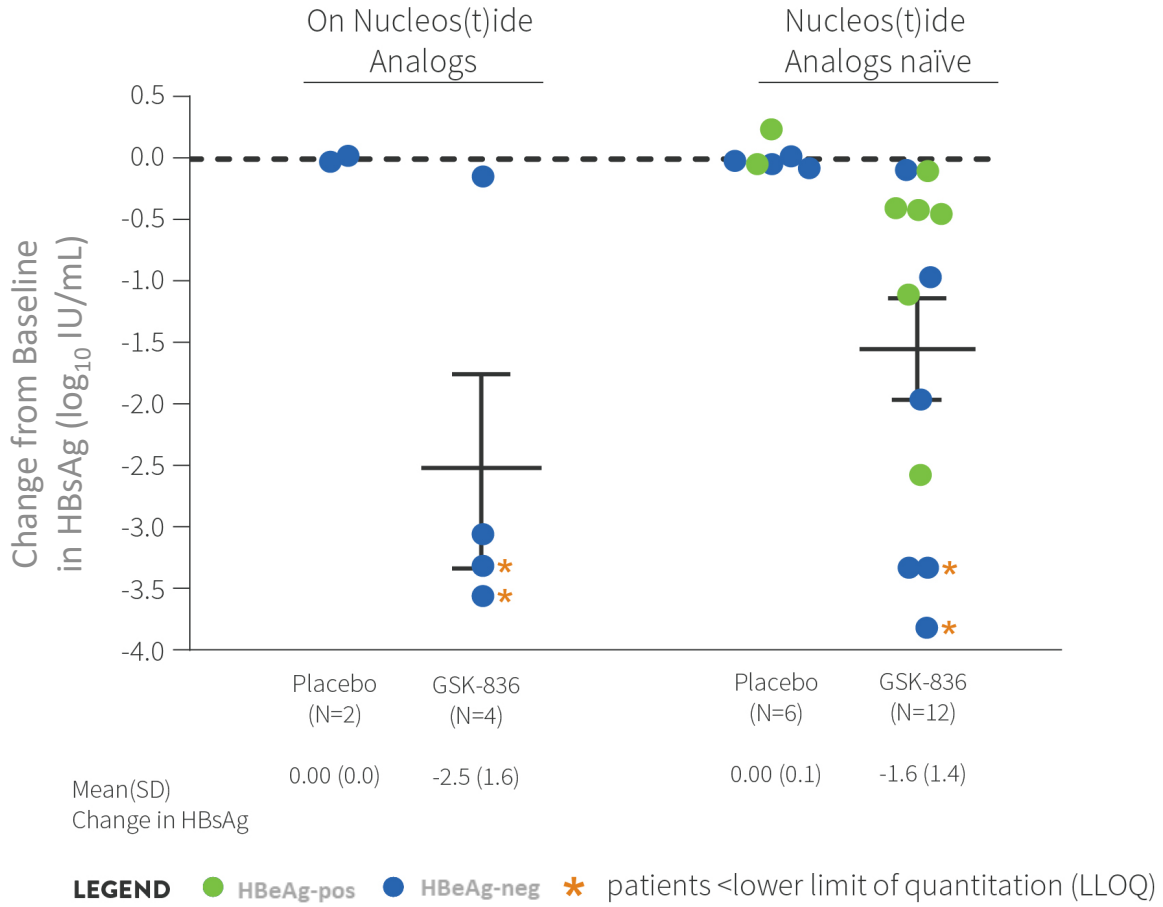
Oligonucleotide Approaches

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

ASOs Have Demonstrated Clinical Anti-HBV Activity



HBsAg reductions at Day 29 among nucleos(t)ide analog (NA) naïve and treated CHB patients receiving six doses of 300 mg GSK-3228836 or placebo over 22 days

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Modified from Yuen et al., EASL dILC2020. #AS067.

ALG-020572
Discovery of our ASO Drug Candidate

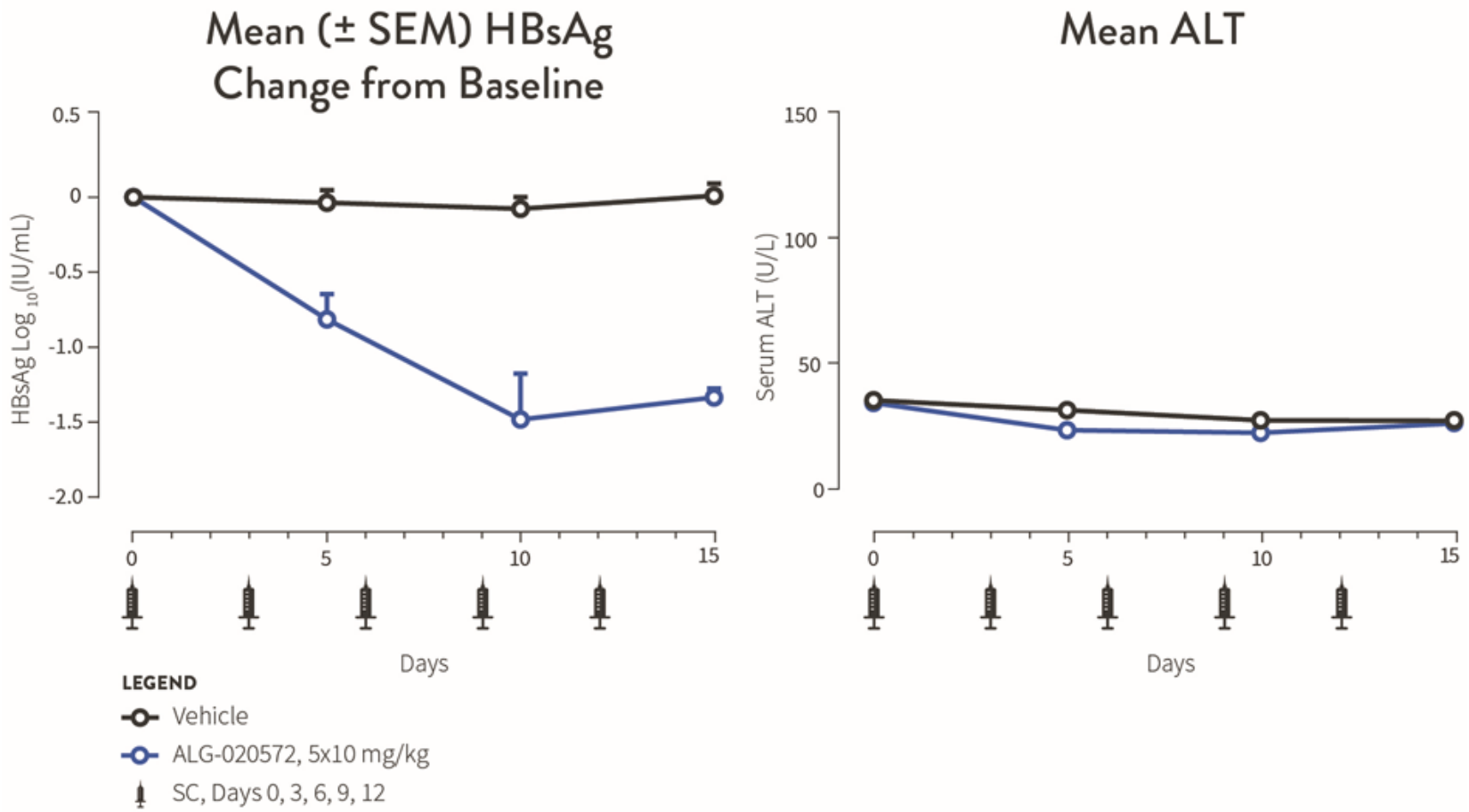
- We have utilized our bioinformatics approach in the selection and optimization of novel ASO constructs

- We optimized our sequences using our proprietary Luxna Biotech chemistries to potentially enhance in vitro potency, stability and safety
  - Luxna xeno nucleic acids (XNA) are 3rd generation bridged nucleic acids (BNA) that may improve nuclease resistance and reduce hepatotoxicity

- Potent ASOs were conjugated to our proprietary AM Chemicals GalNAc ligands for targeted liver delivery

- This approach has led to the selection of our ASO drug candidate ALG-020572

ALG-020572
Our ASO Drug Candidate in the Mouse AAV-HBV Model



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

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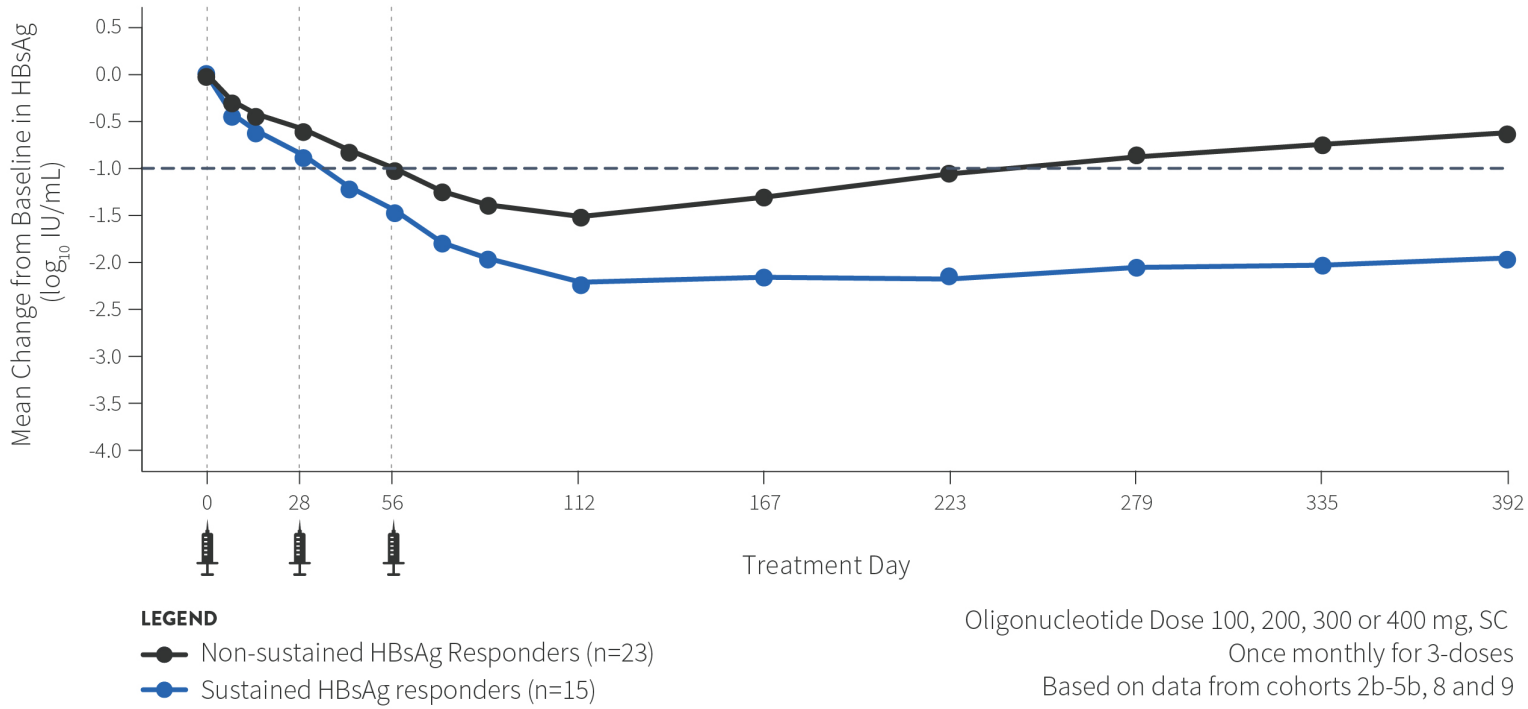
siRNAs Have Demonstrated Clinical Anti-HBV Activity

JNJ-3989, a GalNAc conjugated, dual (X + S) trigger siRNA



JNJ-3989 in combination with a nucleos(t)ide analog demonstrated durable HBsAg reductions following three monthly doses

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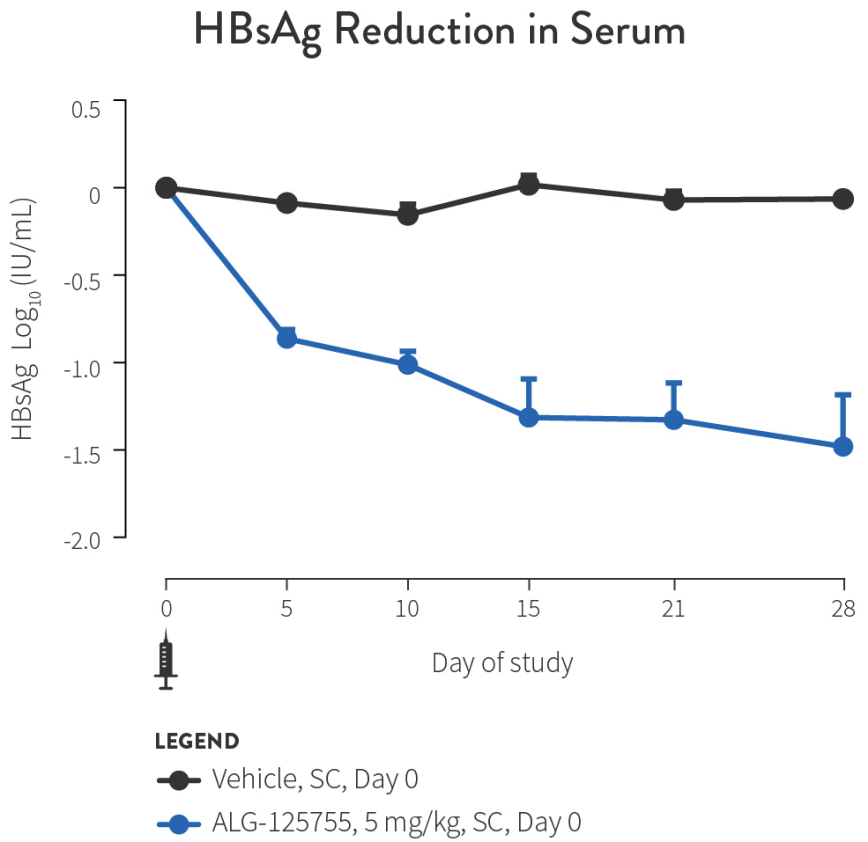
Modified from Gane E, et al. EASL dILC2020. #AS068.

ALG-125755
Discovery of our siRNA Drug Candidate

- We have utilized our bioinformatics approach in the selection and optimization of novel siRNA target sequences
- We optimized siRNA sequences using our proprietary technology including chemical modifications and liver targeting conjugation to maximize in vivo potency
- Our approach to developing siRNAs may have safety, stability and potency advantages over ASOs and other siRNAs
- ALG-125755 has optimized chemistry enabling potency and durability in nonclinical animal models of CHB

ALG-125755
siRNA Drug Candidate in the Mouse AAV-HBV Model



Potent and sustained antiviral activity following a single dose through 28-days of monitoring

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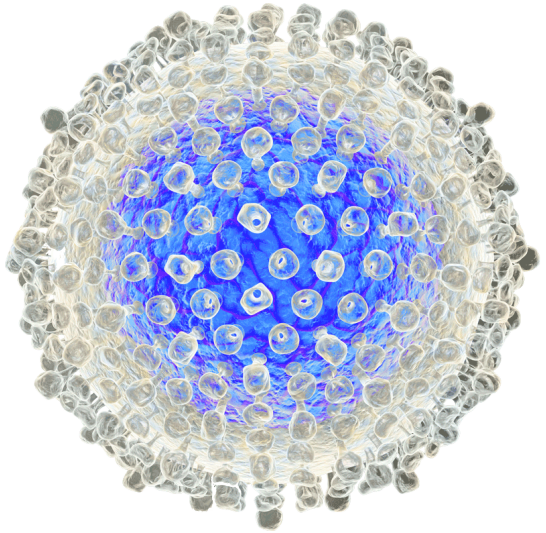
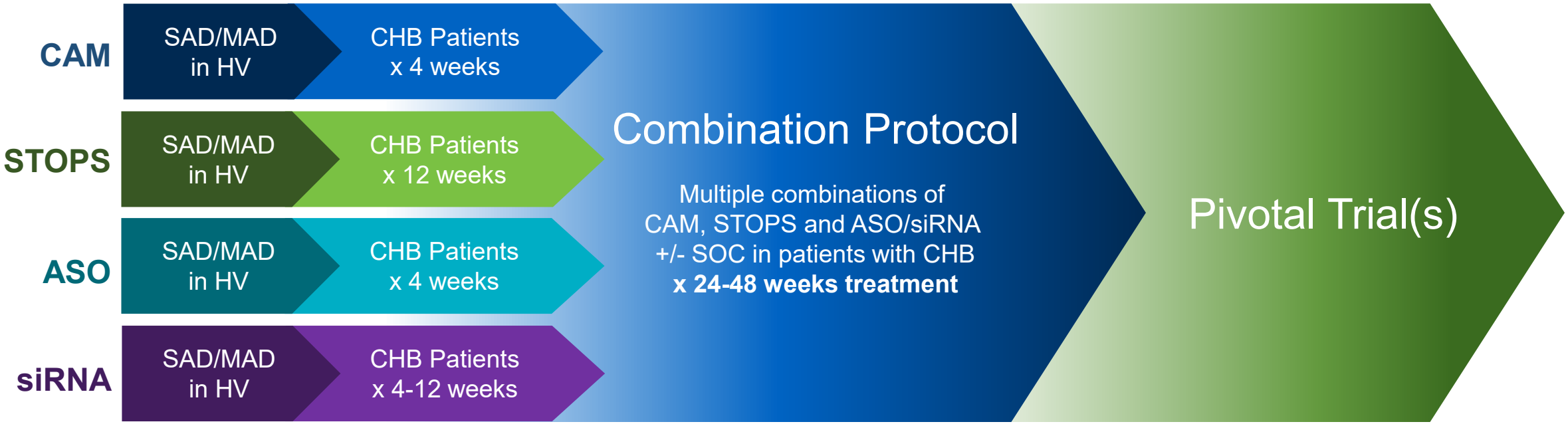
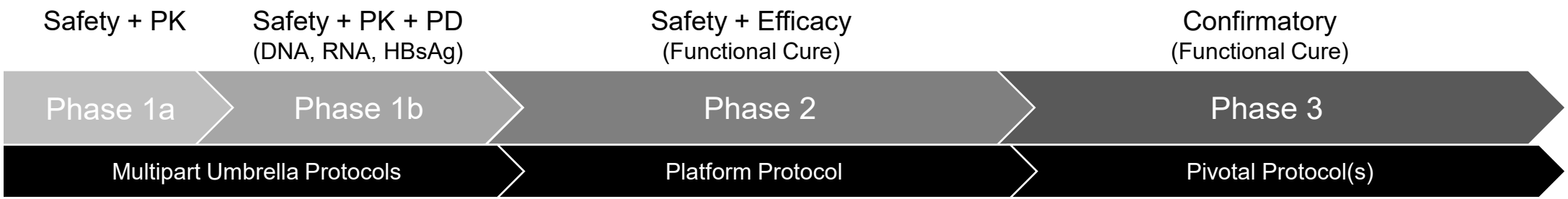
Chronic Hepatitis B

Clinical Development Combination Strategy



Our CHB Clinical Development Strategy





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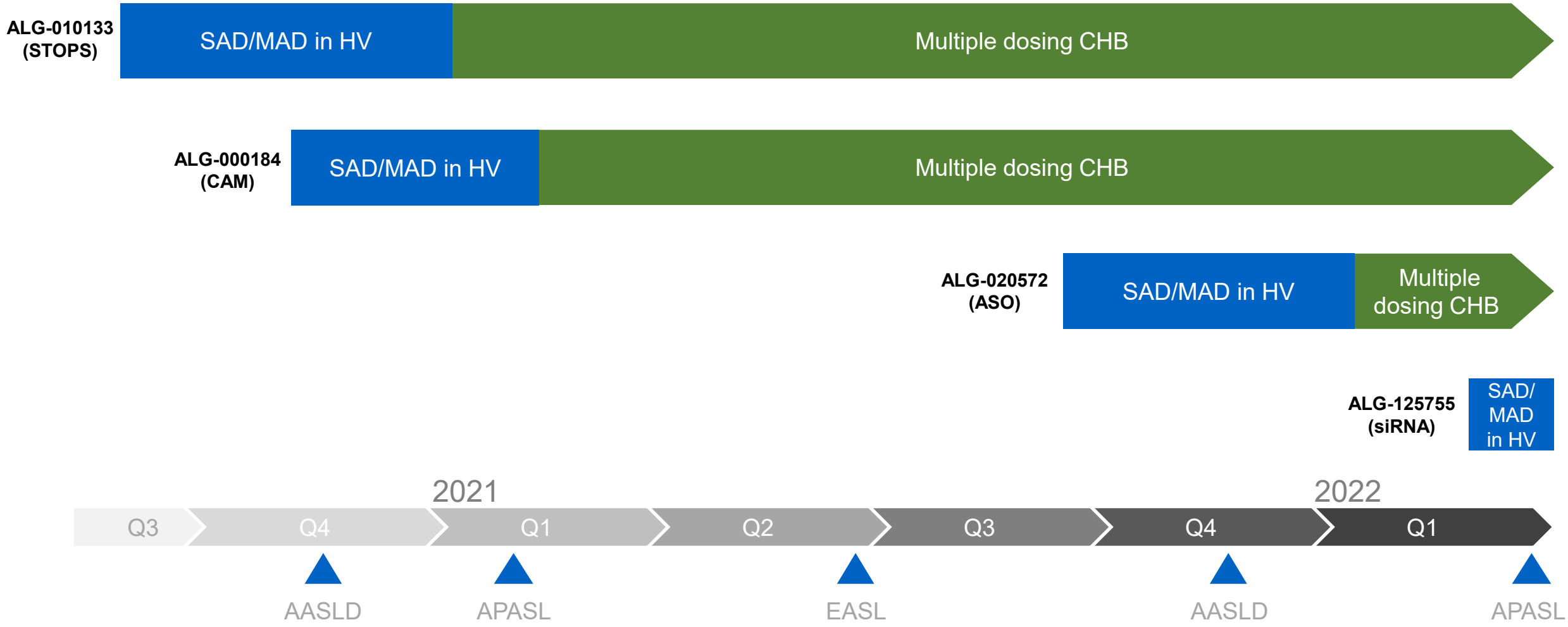
Aligos CHB Clinical Development Timelines
Phase 1 Target Timelines*



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SAD – Single ascending doses; MAD- Multiple ascending doses; HV – Healthy volunteers; CHB – Chronic hepatitis B
*Timelines are approximate and subject to change based on regulatory approvals.

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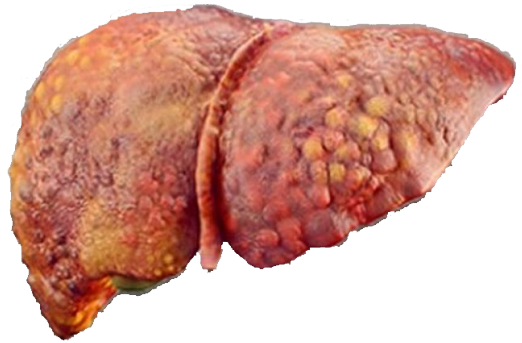
NASH

- ALG-055009, small molecule THR-β agonist
- CTA filing planned for 2H 2021



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.

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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

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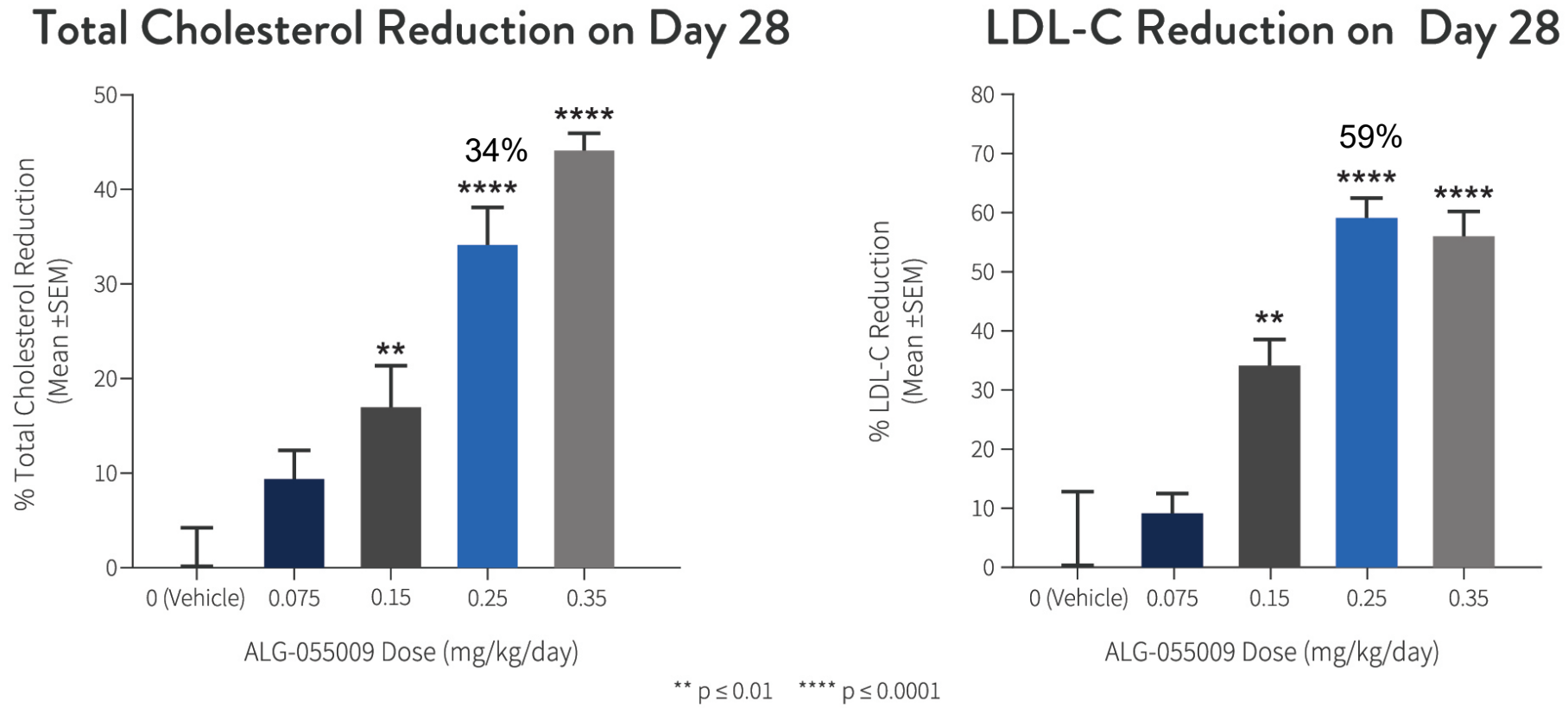
*Data collected in HEK293T cells.

ALG-055009
Dose-Related Effect on Serum Lipids in the DIO Mouse Model



0.25 mg/kg ALG-055009 resulted in greater serum lipid reductions in the DIO model compared to what has been previously reported for VK-2809 and resmetirom at exposures equivalent to Phase 2b/3 clinical doses without a thyroid hormone safety signal

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DIO = Diet Induced Obesity. Kelly et.al. 2014, Erion et. al. 2007, Taub et. al. 2013, Harrison et. al. 2019.

Our NASH Strategy

- Advance our lead THR-β drug, ALG-055009
  - Phase 1 proof of concept study planned for 2021-2022
    - › Provide clinical confirmation of the safety, PK and efficacy advantages identified in nonclinical studies
    - › Data readouts expected to begin in 2022

- Madrigal Phase 3 and VK-2809 Phase 2b readouts are expected in late 2021 through early 2022
  - We believe that enthusiasm for the THR-β MOA is likely to be high following these data

- If enthusiasm for the MOA is high, we plan to partner ALG-055009 with a NASH company with a complementary MOA

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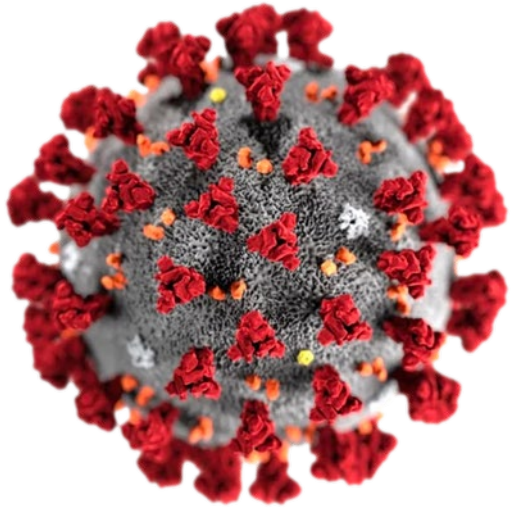
Coronavirus



Our Approach to Developing a Broad Coronavirus Treatment

- We are leveraging our virology and chemistry expertise to develop purpose-built drugs with distinct MOAs
  - Coronavirus protease inhibitors in collaboration with CD3/Rega at KU Leuven
    - Initial formulation will be developed for the IV route for use in hospitalized patients
  - Oligonucleotides
    - Inhaled (outpatient)

- We are aiming to develop drug candidates that
  - Are broadly active against diverse coronaviruses
  - Have 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 regimens with MOAs that may offer therapeutic benefit for potential zoonotic coronavirus transmissions in the future

Potent and Selective Coronavirus Protease Inhibitors Identified

- ALG-097111 is one of our most advanced compounds and exhibits promising drug-like properties
  - In contrast to other known SARS-CoV-2 protease inhibitors, Aligos compounds are designed to be highly selective over host protease cathepsin L
  - Single digit nM potency vs. SARS-CoV-2 protease
  - Excellent cell-based potency vs. multiple coronaviruses
  - Strong synergy demonstrated in combination with remdesivir
- Candidate selection for clinical development projected for mid-2021

Assay System	ALG-097111	PF-00835231	GC-376
SARS-CoV-2 3CLp IC_{50} (SAMDI)/Ki(FRET) (μM)	0.007 / 0.001	0.005 / 0.00025	0.025
Cathepsin L IC_{50} (μM)	>10	0.155	<0.0005
SARS-CoV-2 Huh-7 EC_{50}/CC_{50} (μM)	2.1 / >100	2.1 / >10	0.105 / >100
OC43(HeLa) EC_{50}/CC_{50} (μM)	0.123 / >100	0.127 / >100	0.390 / >100

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Aligos Therapeutics Summary

- We have assembled a world-class team with a proven track record of success
- Strong management team supported by top tier investors
  - $100M* Series A in September 2018, $125M** Series B in December 2019 / October 2020
  - $167.2M gross proceeds from our IPO in October 2020 and the underwriters exercise of its over-allotment option
- 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
    - CAM (ALG-000184) Phase 1 clinical trial ongoing
    - ASO and siRNA oligonucleotide compounds are planned to advance into the clinic (2H 2021 / 1H 2022)
  - NASH
    - We are advancing small molecule ALG-055009 into clinical development and plan to partner after Phase 1
    - We have entered into an exclusive oligonucleotide-based License and Research collaboration with Merck (up to $458M)
  - Coronavirus
    - We have identified multiple nM potent coronavirus protease inhibitors

ALIGOS THERAPEUTICS

*Series A total included $5M in convertible notes from angel investors.
**$125M closed in two tranches; $85M in December 2019 and $40M in October 2020.


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THERAPEUTICS