NGM Biopharmaceuticals, Inc. Corporate Overview

Novel Biology. Powerful Medicines. Transformative Impact.

NGMBio

Next Generation Medicines

Safe Harbor Statement

This presentation contains forward-looking statements within the meaning of the “safe harbor” provisions of the Private Securities Litigation Reform Act of 1995, as amended. These forward-looking statements involve substantial risks and uncertainties. All statements other than statements of historical facts contained in this presentation, including statements regarding our future financial condition, results of operations, business strategy and plans, expected near-term milestones, and objectives of management for future operations, as well as statements regarding industry trends, are forward-looking statements. In some cases, you can identify forward-looking statements by terminology such as “anticipate,” “believe,” “continue,” “could,” “estimate,” “expect,” “intend,” “may,” “plan,” “potentially” “predict,” “should,” “will” or the negative of these terms or other similar expressions. These statements include those related to NGM’s advancement of its clinical and preclinical pipeline; the timing, enrollment and results of NGM’s clinical trials, including the continued enrollment and announcement of preliminary results of the Phase 2 clinical study of aldafermin (NGM282) in patients with NASH; NGM’s option to participate in the economic return of NGM313 or ability to receive milestone payments or royalties from NGM313; the continued development of NGM621; the safety, tolerability and efficacy of NGM’s product candidates; NGM’s ability to fund its clinical programs and NGM’s financial outlook. Because such statements deal with future events and are based on NGM’s current expectations, they are subject to various risks and uncertainties and actual results, performance or achievements of NGM could differ materially from those described in or implied by the statements in this press release. These forward-looking statements are subject to risks and uncertainties, including those discussed in the sections titled "Risk Factors" and "Management's Discussion and Analysis of Financial Condition and Results of Operations" in quarterly report on Form 10-Q for the quarter ended June 30, 2019 and other filings that we make from time to time with the Securities and Exchange Commission. Except as required by law, NGM assumes no obligation to update these forward-looking statements after the date of this presentation, or to update the reasons if actual results differ materially from those anticipated in the forward-looking statements.

Company Highlights

Aldafermin (NGM282)
Wholly-owned, Phase 2b product candidate for treatment of **NASH** (non-alcoholic steatohepatitis)

NGM313 (MK-3655)
Insulin sensitizer for treatment of **NASH** and T2D; Completed Ph1b and **licensed by Merck**

Pipeline of **five additional product candidates** in cardio-metabolic, oncologic and ophthalmic diseases

Strategic collaboration with Merck providing robust R&D support and **NGM option on future Merck late-stage programs**

Experienced team with highly productive R&D engine generating on average **~1 development candidate/year**

Multiple **key milestones** and potential **value driving catalysts** expected in the next 18 months

Highly Experienced Team with Proven First-in-Class Drug Development Track Record

MANAGEMENT

William J. Rieflin
Executive Chairman

David J. Woodhouse, Ph.D.
Chief Executive Officer and
Acting Chief Financial Officer

Aetna Wun Trombley, Ph.D.
President and Chief Operating Officer

Jin-Long Chen, Ph.D.
Founder
Chief Scientific Officer

Alex DePaoli, M.D.
Senior Vice President
Chief Translational Officer

Hsiao D. Lieu, M.D.
Senior Vice President
Chief Medical Officer

Wenyan (David) Shen, Ph.D.
Senior Vice President, Biologics

Hui Tian, Ph.D.
Senior Vice President, Research

EXTENSIVE PRIOR EXPERIENCE

MERCK
Genentech
AMGEN
NOVARTIS
Tularik
Lilly

NGMBio

prolia (denosumab) injection
myalept (metreleptin) for injection
Effient (prasugrel) tablets
taltz (ixekizumab)
Lexiscan
Repatha
Humulin

Key Roles in Prior Drug Approvals

BOARD OF DIRECTORS

William J. Rieflin
Executive Chairman, NGM Bio

David V. Goeddel, Ph.D.
Lead Independent Director
Managing Partner, The Column Group

Jin-Long Chen, Ph.D.
Founder, CSO NGM Bio

Suzanne Sawochka Hooper
Former Exec. VP and GC, Jazz Pharmaceuticals

Mark Leschly
Managing Partner, Rho Capital Partners

David Schnell, M.D.
Managing Director, Prospect Venture Partners

Peter Svennilson
Managing Partner, The Column Group

McHenry (Mac) T. Tichenor, Jr
Managing Partner, Tichenor Ventures, LLC

David J. Woodhouse, Ph.D.
CEO and Acting CFO, NGM Bio

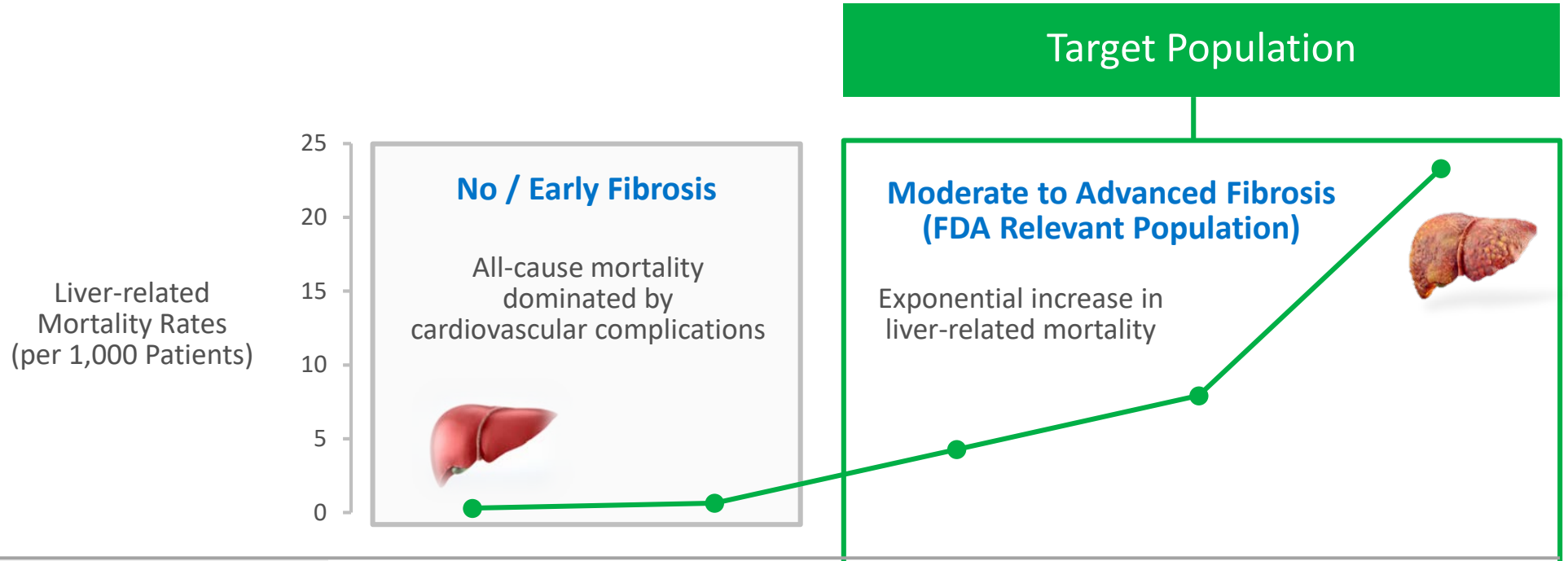
Our Expansive Pipeline

7 Development Programs

PRODUCT CANDIDATE	PRODUCT DESCRIPTION (DOSING FREQUENCY)	POTENTIAL INDICATIONS	STAGE OF DEVELOPMENT	WORLDWIDE COMMERCIAL RIGHTS
Aldafermin (NGM282)	FGF19 Analog (Once Daily)	NASH	Phase 2b	NGM Bio: Wholly-Owned
NGM313 (MK-3655)	FGFR1c/KLB Agonistic Antibody (Once Monthly)	NASH, Type 2 Diabetes	Phase 1b	MERCK: Licensed; NGM Bio
NGM120	GFRAL Antagonistic Antibody (Long Acting)	Cancer Anorexia/Cachexia Syndrome (CACS)	Phase 1	NGM Bio; MERCK: Option
NGM217	Undisclosed (Long Acting)	Diabetes	Phase 1	NGM Bio; MERCK: Option
NGM621	Complement C3 Antagonistic Antibody (Long Acting)	Dry Age-Related Macular Degeneration (AMD)	Phase 1	NGM Bio; MERCK: Option
NGM386	GDF15 Analog (Once Daily)	Metabolic	Phase 1	NGM Bio: Wholly-Owned
NGM395	GDF15 Analog (Long Acting)	Metabolic	Preclinical	NGM Bio: Wholly-Owned

FGF19: fibroblast growth factor 19; FGFR1c/KLB: fibroblast growth factor receptor 1c/beta-klotho; GDF15: growth differentiation factor 15; GFRAL: glial-cell-derived neurotrophic factor receptor alpha-like

Improving Fibrosis Leads to Better Outcomes for NASH Patients



Fibrosis Stage	F0	F1	F2	F3	F4
U.S. Prevalence (Millions)	3.5	6.3	3.4	2.0	1.3
Drivers of Mortality	Cardiovascular Disease		Liver Disease		
Time to Progression	Approximately One Fibrosis Stage Worsening Every 7 Years				

NASH expected to be the #1 cause of liver transplant by 2020[1]

Sources: Dulai et al, Hepatology 2017, 65(5):1557-1565; Singh et al, Clin Gastroenterol Hepatol. 2015, 13(4): 643–654; Estes et al, Hepatology 2018, 67(1): 123-133.

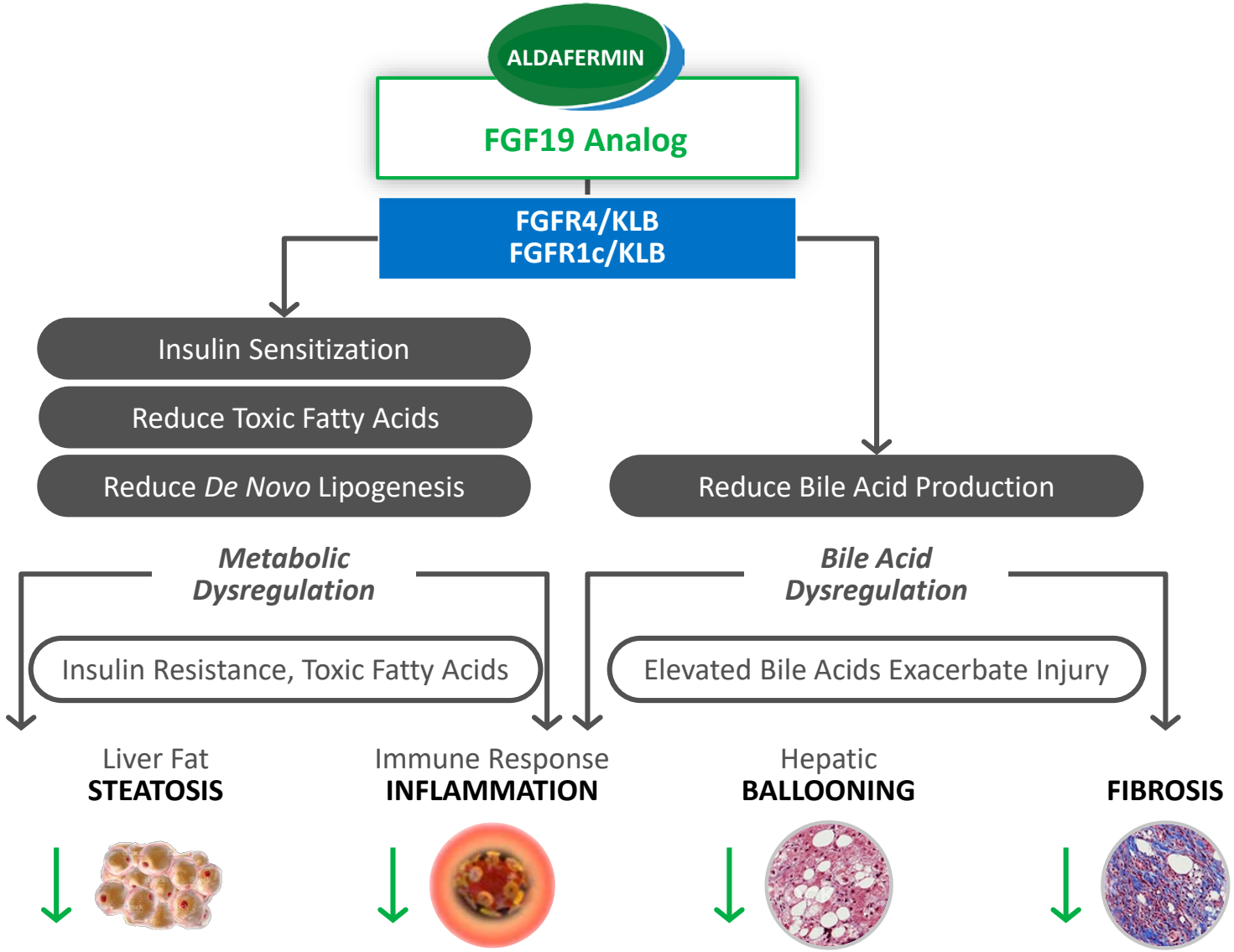
[1] *Canbay et al, Visc Med 2016, 32: 234-238.*

Aldafermin Impacts the Key Drivers of NASH Pathogenesis

ALDAFERMIN (NGM282)
Actions on Implicated Disease Drivers

Implicated Disease Drivers

Resulting Impact on Disease Progression in the Liver



Unlike Many Other Drugs in Development, Aldafermin Targets Fibrosis Reversal and Cirrhosis Prevention in Advanced NASH Patients

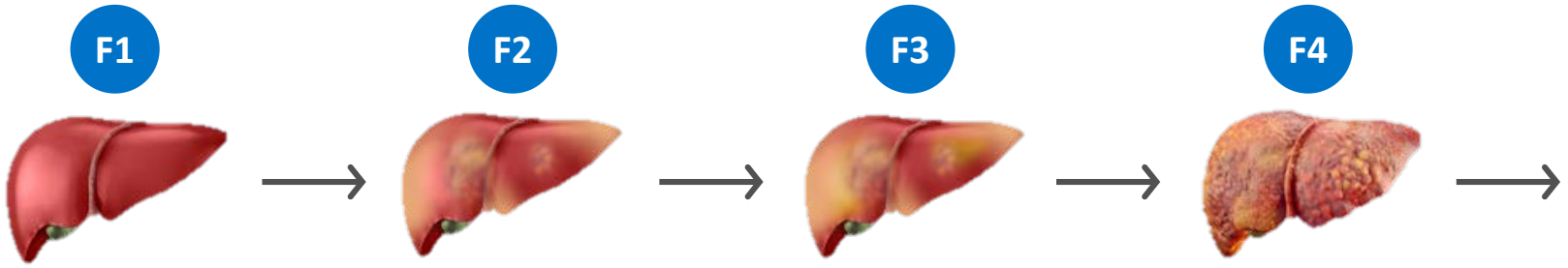
Competitive Landscape

Metabolic Drugs (Many)

Drugs with Metabolic **and** Anti-fibrotic Effect (Limited)

Like Type 2 Diabetes (T2D), NASH Can Support Multiple Drug Classes

NASH Patient Segmentation



Liver Transplant/ Liver-related Mortality

US Prevalence

F1	F2	F3	F4
6.3M	3.4M	2.0M	1.3M

Target Patient Populations

Phase 2 data suggests aldafermin may:

- Rapidly reverse fibrosis
- Prevent cirrhosis
- Prevent decompensation, liver events

Aldafermin: Single Agent with Metabolic and Anti-Fibrotic Efficacy

- ☑ Only FGF19 agonist in clinical development for NASH → **potent activity on FGFR1c and FGFR4**
- ☑ Significant reduction **across all non-invasive measures at 12W**: MRI-PDFF, ALT/AST, fibrosis biomarkers (ELF, PRO-C3) and bile acid synthesis (C4; Phase 2, Cohorts 1-3)
- ☑ **Rapid reversal of fibrosis** (≥ 1 stage) **in 42% of patients at 12W** (3 mg dose)
- ☑ **Improvements across all other histological measures of NASH at 12W** (% of patients with ≥ 1 stage improvement; 3 mg dose): steatosis (74%), inflammation (42%) and ballooning (53%)
- ☐ Durability of effect on non-invasive measures at 24W (Interim analysis, 2H19)
- ☐ Confirmation of effect on fibrosis/NASH histology at 24W in placebo-controlled trial (early 2020)

NASH Phase 2 Program Provides Foundation for Late Stage Development of Aldafermin

Clinical Data in Presentation

	COHORT 1	COHORT 2	COHORT 3	COHORT 4
Status	**Completed** (Published, *The Lancet* 2018)	**Completed** (Presented, EASL 2018)	**Completed** (Presented, AASLD 2018)	**Ongoing** (Anticipated interim data 2H19)
Duration	12 Weeks			24 Weeks
Aldafermin Dose (# Patients)	Placebo (27); 3 mg (27); 6 mg (28)	0.3 mg (23); 1 mg (21)	3 mg (22); 1 mg (28)	Placebo (~25); 1 mg (~50)

KEY ENDPOINTS/ Assessment Measures[1]

HISTOLOGY (Biopsy)

NON-INVASIVE MEASURES

STEATOSIS

% Liver Fat Content (LFC) (MRI-PDFF)

INFLAMMATION

BALLOONING

ALT/AST (Biomarkers)

FIBROSIS

ELF/PRO-C3 (Biomarkers)

BILE ACIDS

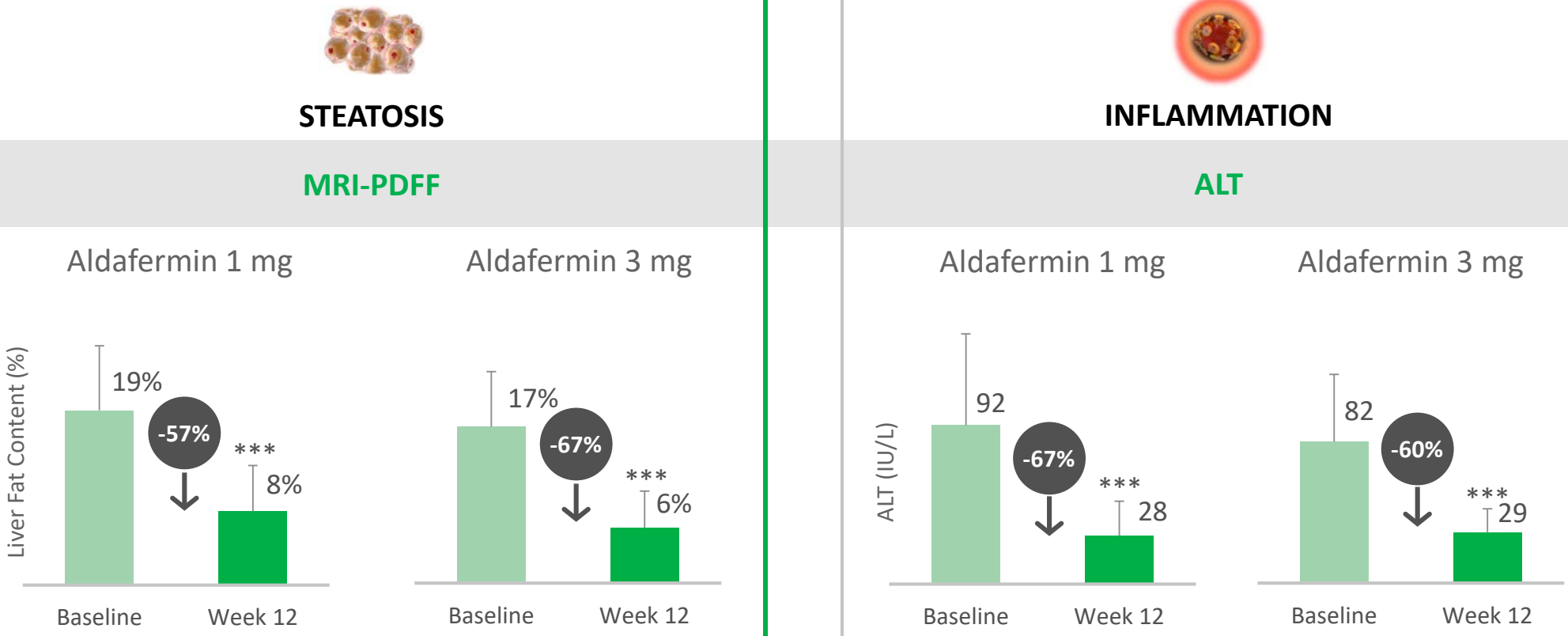
C4[1] (Biomarker)

[1] *C4: 7α-hydroxyl-4-cholesten-3-one*

Key Primary and Secondary Endpoints Achieved by Week 12 (MRI-PDFF and ALT)

Markers of Hepatic Steatosis and Inflammation

MEASURE/ BIOMARKER	STEATOSIS	INFLAMMATION
	MRI-PDFF	ALT



92% (1 mg) and 100% (3 mg) of Subjects Achieved Primary Phase 2 Endpoint (≥ 5% Absolute LFC Reduction by Week 12)

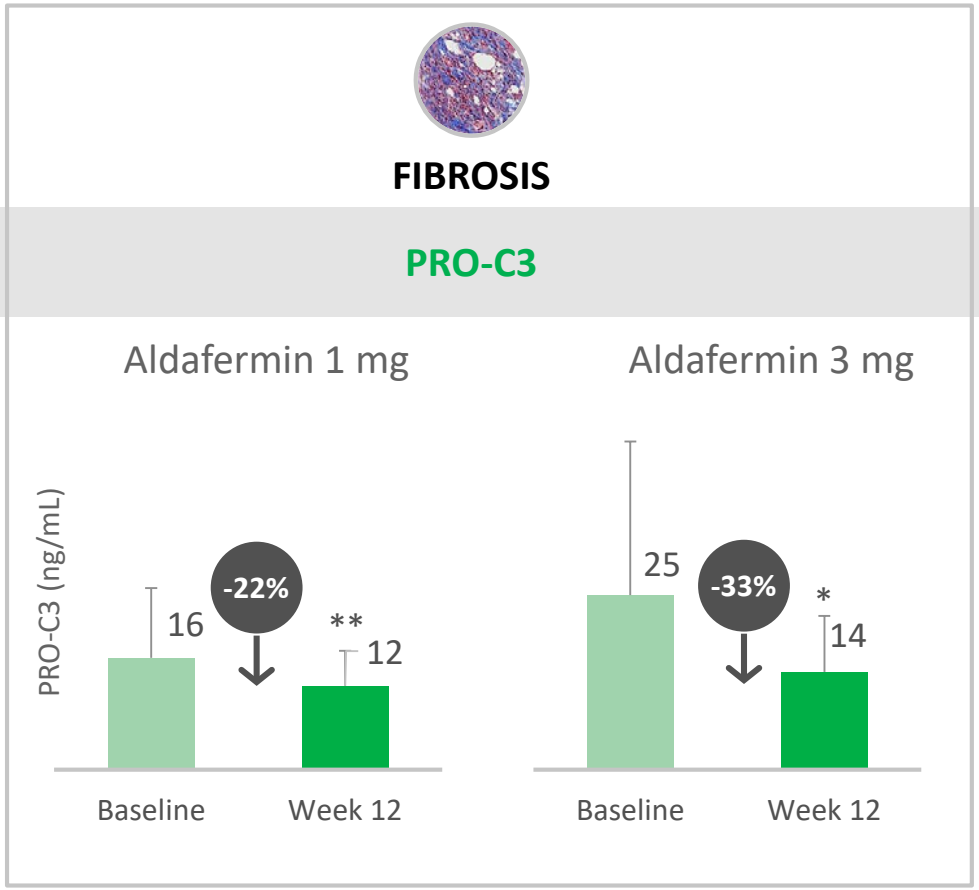
****P<0.001, **P<0.01, *P<0.05 vs. baseline*

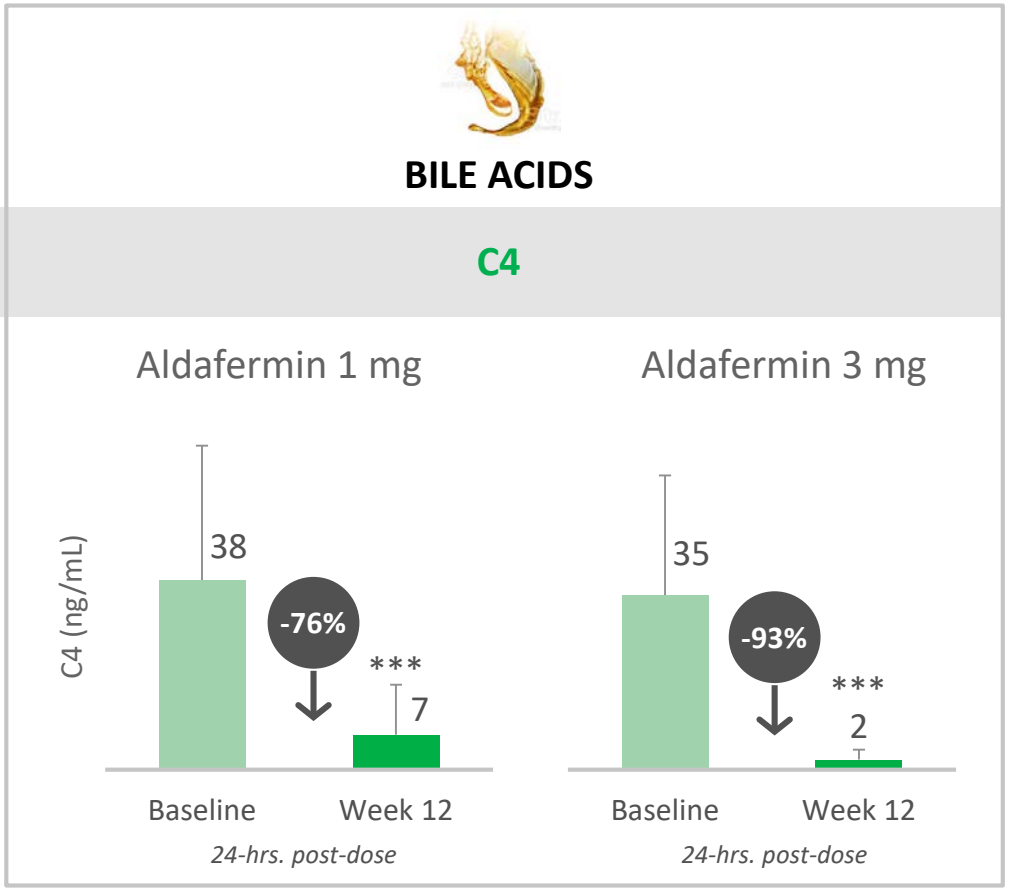
Source: Aldafermin 1 mg (Cohort 3) and 3 mg (Cohort 2) biopsy cohorts; Preliminary data

Key Primary and Secondary Endpoints Achieved by Week 12 (PRO-C3 and C4)

Markers of Fibrogenesis and Bile Acid Synthesis

MEASURE/ BIOMARKER	FIBROSIS	BILE ACIDS
	PRO-C3	C4





****P<0.001, **P<0.01, *P<0.05 vs. baseline*

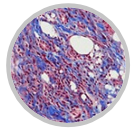
C4 = 7alpha-hydroxyl-4-cholesten-3-one

Source: Aldafermin 1 mg (Cohort 3) and 3 mg (Cohort 2) biopsy cohorts; Preliminary data

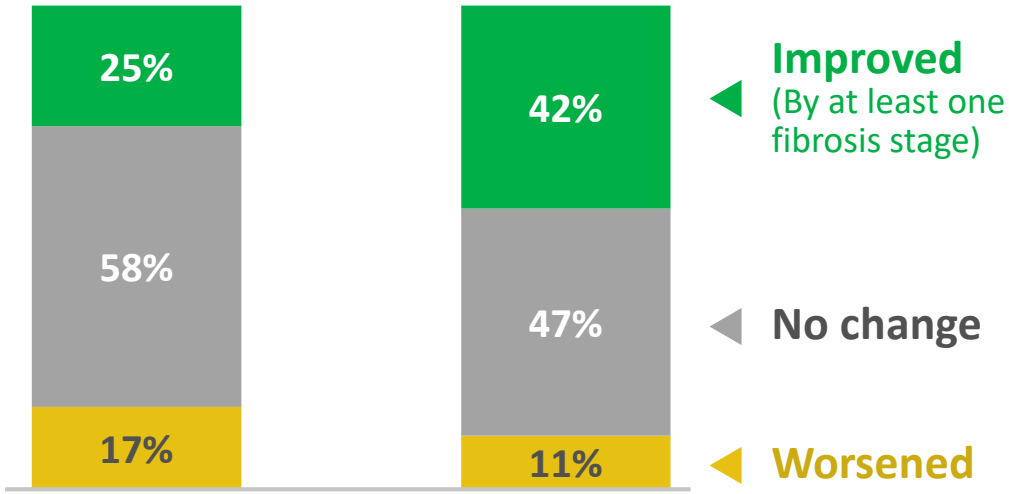
Rapid Regression of Fibrosis at Week 12 in Patients Treated with Aldafermin

Fibrosis Histological Response at Week 12[1]

(% of Patients; n=19 in 3 mg dose of Cohort 2, n=24 in 1 mg Cohort 3)



FIBROSIS



	1 mg	3 mg
Mean change at week 12 (SD)	**-0.1** (0.7)	**-0.5** (0.9)

Improvements

- All of the patients experiencing improvements were F2 or worse at baseline
- Across the two cohorts, four subjects had a 2-stage fibrosis improvement

Patient Population

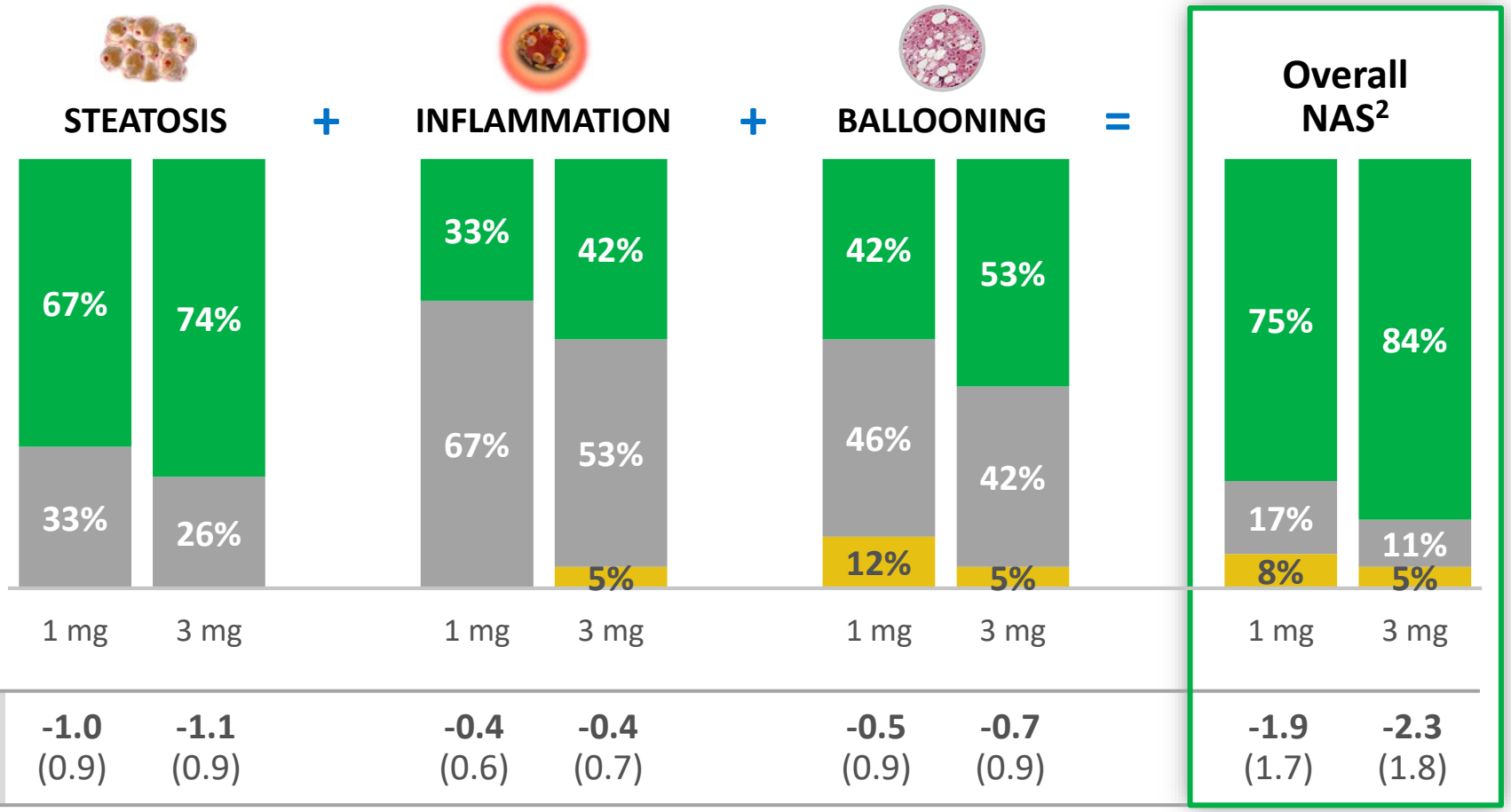
- Over 80% of patients in 3 mg Cohort 2 and Cohort 3 had F2/F3 fibrosis at baseline

[1] *Preliminary data*

Exploratory Endpoints Achieved: All NASH Histological Parameters Improved at Week 12

NAS Histological Response at Week 12[1,2]

(% of Patients; n=19 in 3 mg dose of Cohort 2, n=24 in 1 mg Cohort 3)



Mean change at week 12 (SD)	**-1.0** (0.9)	**-1.1** (0.9)		**-0.4** (0.6)	**-0.4** (0.7)		**-0.5** (0.9)	**-0.7** (0.9)		**-1.9** (1.7)	**-2.3** (1.8)

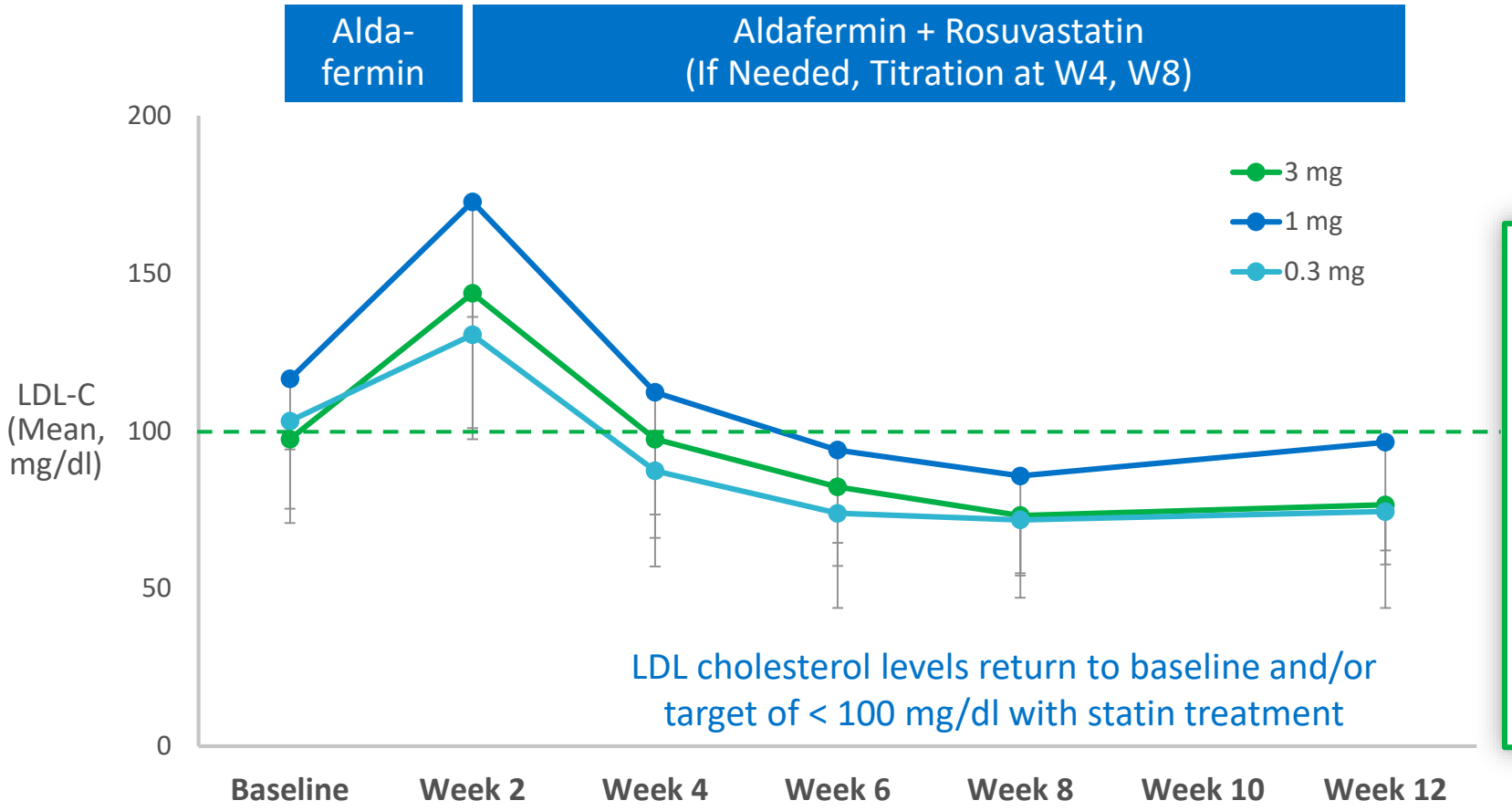
1 *Preliminary data*
2 *NAS: NAFLD Activity Score*

Favorable Tolerability Profile

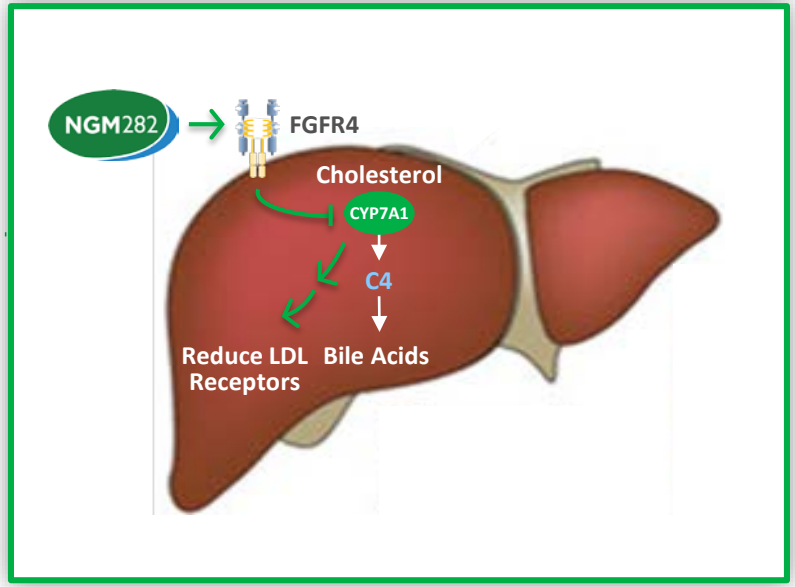
- Over 400 subjects have been dosed with aldafermin and the drug was well-tolerated
- Preliminary data indicates that there were no tolerability signals identified in the NASH population
- Preliminary safety data from the Phase 2 NASH study:
  - Cohort 1: a single serious adverse event (SAE) of acute pancreatitis was reported and assessed as possibly related to study drug
  - Cohort 2: seven SAEs (none of which were considered related to study drug) were reported in five subjects
  - Cohort 3: a single SAE (kidney mass) was reported, which was not considered related to study drug
- The most common adverse events in cohorts 1, 2 and 3 were increased stool frequency, loose stools, nausea and injection site erythema, with the majority being mild

Source: Phase 2 NASH Study, Cohorts 1-3

Cholesterol Changes Effectively Managed with Statin Therapy



LDL-C elevation is a direct effect of FGF19's potent inhibition of the classical bile acid synthesis pathway



Serum triglyceride levels decreased and HDL-C elevated with aldafermin treatment

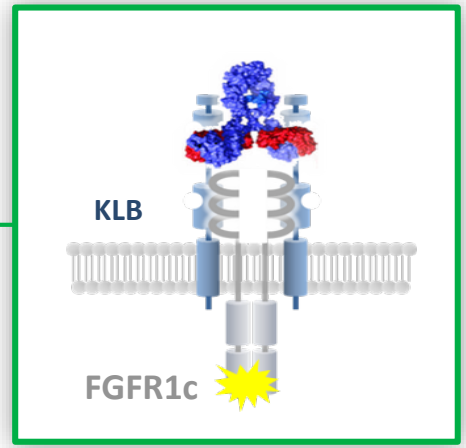
Source: Phase 2 NASH Study, Cohort 2

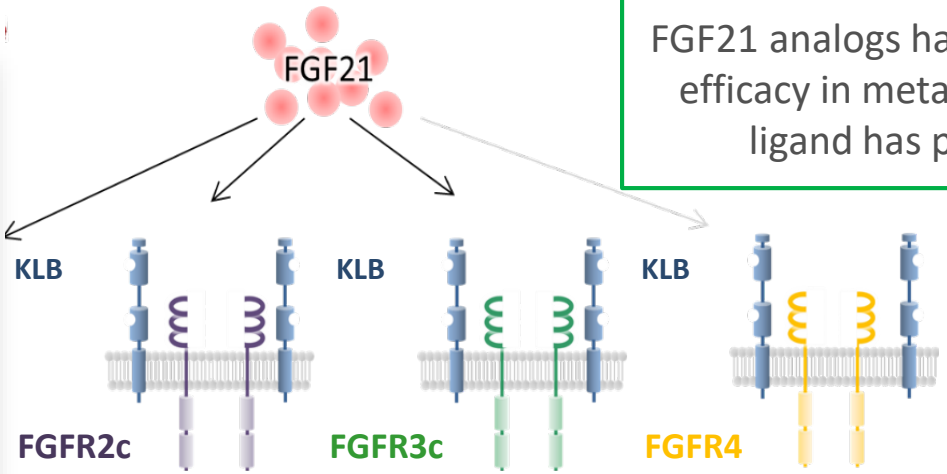
Aldafermin Development Plan

	PHASE 2 – COHORT 4	PHASE 2b (ALPINE 2/3)	PHASE 2b – COMPENSATED CIRRHOTICS (ALPINE 4)	PHASE 3 PROGRAM
Status	**Ongoing** (Interim data of non-invasive measures anticipated 2H19)	**Ongoing**	**To Initiate 2H19**	**To Initiate Planning with Phase 2 Cohort 4 Data**
Duration	24 Weeks	24 Weeks	TBD	TBD
Aldafermin Dose (# Patients)	Placebo (~25) 1 mg (~50)	Placebo (~40) 0.3 mg, 1 mg, 3 mg (~40 per dose level)	Placebo Dose Levels TBD	Placebo Single Dose Level TBD

NGM313 (MK-3655) for the Treatment of NASH and Type 2 Diabetes

NGM313



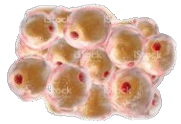


FGF21 analogs have demonstrated variable clinical efficacy in metabolic syndrome patients; native ligand has potential for safety liabilities

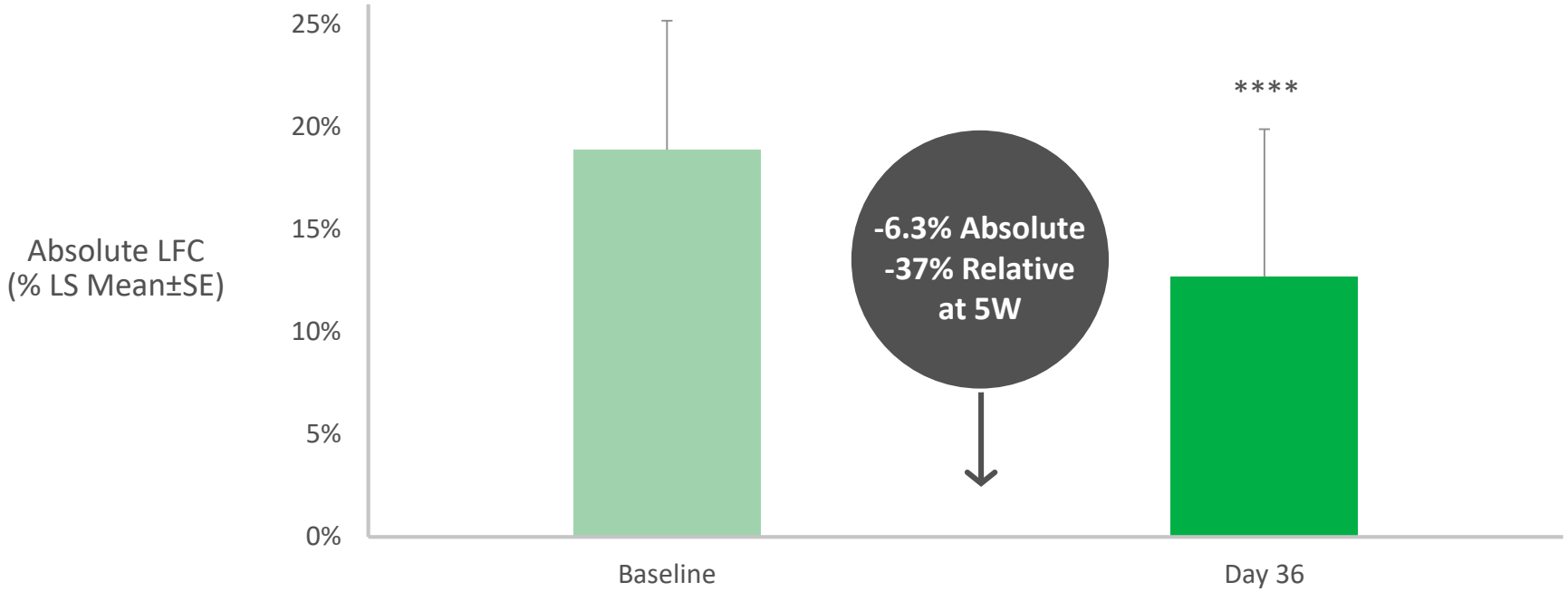
- Agonistic antibody that selectively activates FGFR1c / KLB to regulate energy metabolism
- Potential to be once-monthly injectable insulin sensitizer for treatment of NASH and T2D
- Completed Phase 1 SAD/MAD study in obese, insulin resistant subjects and Phase 1b study in subjects with NAFLD
- Single dose of NGM313 resulted in **significant reductions in liver fat content and improvement in metabolic markers** based on preliminary data from a Phase 1b study in obese, insulin resistant subjects with NAFLD **after five weeks**
- Well-tolerated across Phase 1 and Phase 1b studies
- **Merck exercised its option and licensed NGM313 and other FGFR1c/KLB agonists in 4Q18**

Significant Reduction in LFC After 5 Weeks Following Single Dose of NGM313 (MK-3655)

Phase 1b Study in Obese, Insulin Resistant Subjects with NAFLD



Absolute LFC
MRI-PDFF



Pioglitazone 45 mg

(Positive Control)

- 4.0% absolute (25% relative) LFC reduction at 5W

NGM313

Statistically Significant Improvements In:

- ALT and AST
- Triglycerides
- HDL-C, LDL-C
- PRO-C3

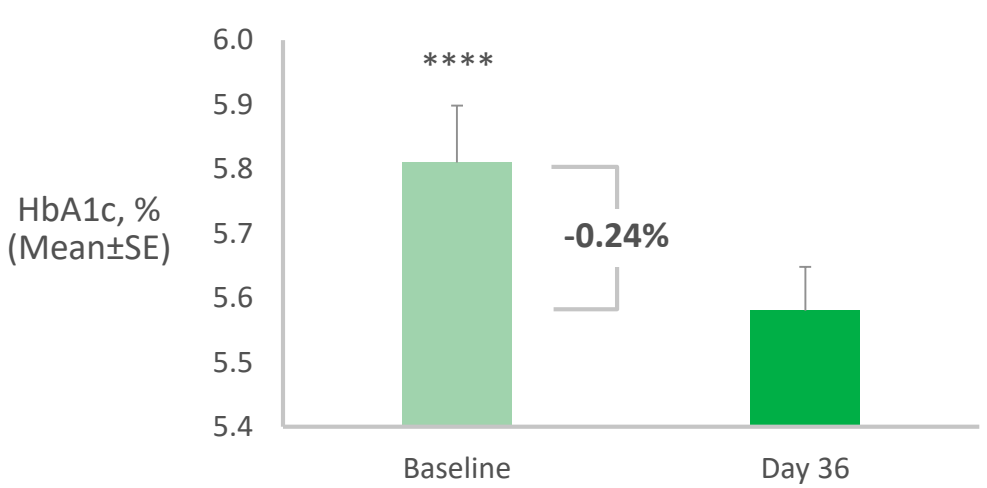
*****p<0.0001*

Rapid Reduction in HbA1c Without Hypoglycemia After Single Dose of NGM313 (MK-3655)

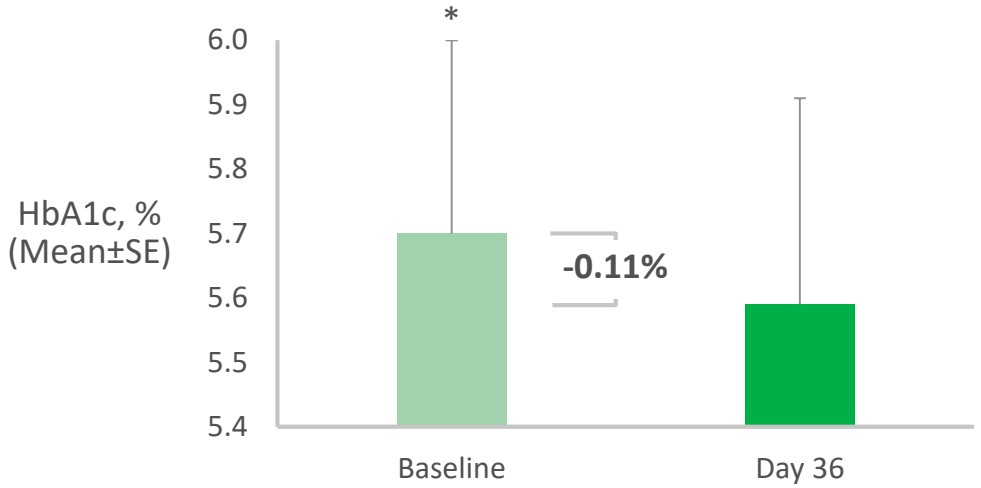
Phase 1b Study in Obese, Insulin Resistant Subjects with NAFLD

HbA1c

NGM313, 240 mg
(n=17)



Pioglitazone, 45 mg
(n=8)



Reduction in HbA1c observed in insulin-resistant, non-diabetic patients supports promise of NGM313 to potentially improve glucose control in patients with T2D

NGM313 increases body weight by 1.6 kg (no edema or fluid retention) *vs*. 2.4 kg increase with pioglitazone at day 36

** p<0.05; ****p<0.0001*

NGM313 (MK-3655) has Potential to Complement Aldafermin by Targeting NASH Population with T2D

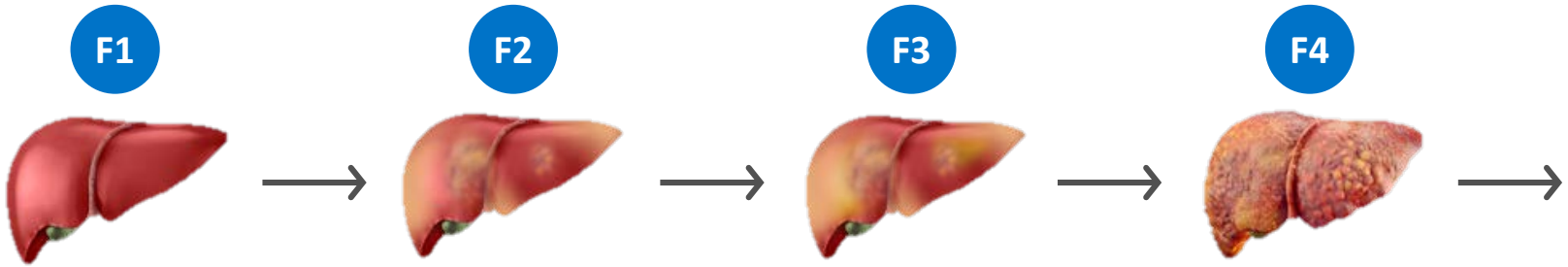
Competitive Landscape

Metabolic Drugs (Many)

Drugs with Metabolic **and** Anti-fibrotic Effect (Limited)

Like T2D, NASH Can Support Multiple Drug Classes

NASH Patient Segmentation



Liver Transplant/ Liver-related Mortality

US Prevalence

F1	F2	F3	F4
6.3M	3.4M	2.0M	1.3M

Target Patient Populations

Complementary Treatments for NASH

NGM313 (MK-3655) Next Steps

PHASE 1b

Completed

Presented preliminary data at AASLD 2018, EASL 2019 and ADA 2019

- Merck exercised its option to license the program in 4Q18
- NGM received a $20M payment in connection with the option exercise

PHASE 2b

Merck to Initiate in 2020

Ph2b study to evaluate the effect of MK-3655 on liver histology and glucose control in NASH F2/F3 patients with or without diabetes

- **Merck to fund all Phase 2 development costs**
- **NGM retains an option, when program has advanced to Phase 3, to participate in up to 50% of the economic return of the program**
- **If NGM does not exercise its option, NGM is eligible for development and commercial milestone payments and tiered royalties ranging from low double digit to mid-teen percentage rates on product sales**

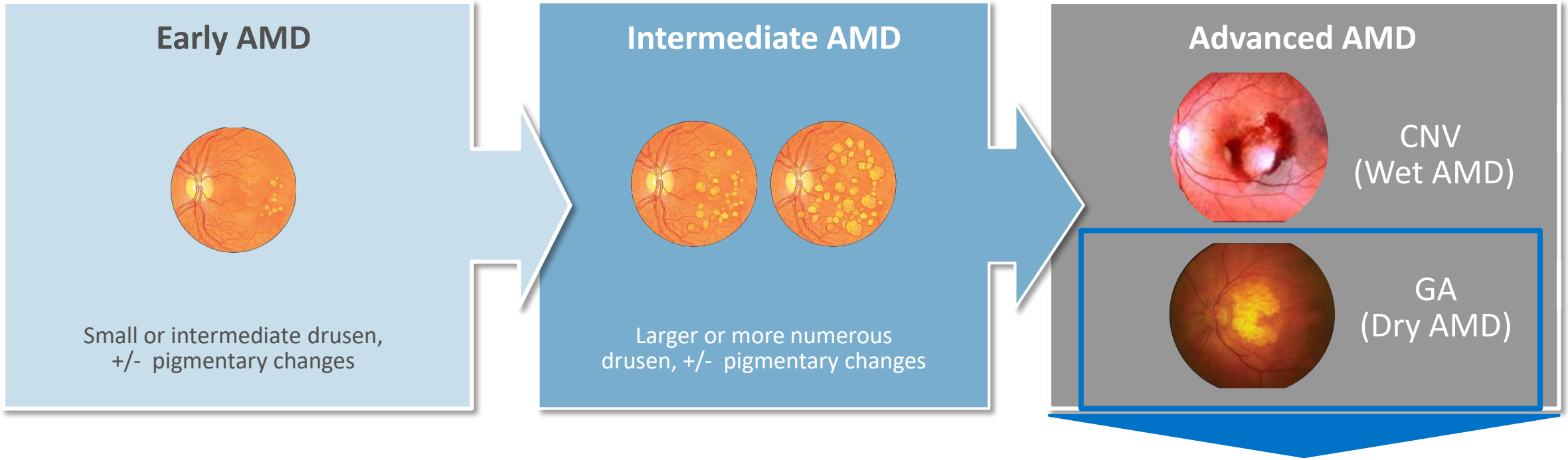
Beyond NASH, an Expansive Pipeline in Other Indications

7 Development Programs

PRODUCT CANDIDATE	PRODUCT DESCRIPTION (DOSING FREQUENCY)	POTENTIAL INDICATIONS	STAGE OF DEVELOPMENT	WORLDWIDE COMMERCIAL RIGHTS
Aldafermin (NGM282)	FGF19 Analog (Once Daily)	NASH	Phase 2b	NGM Bio: Wholly-Owned
NGM313 (MK-3655)	FGFR1c/KLB Agonistic Antibody (Once Monthly)	NASH, Type 2 Diabetes	Phase 1b	MERCK: Licensed; NGM Bio
NGM120	GFRAL Antagonistic Antibody (Long Acting)	Cancer Anorexia/Cachexia Syndrome (CACS)	Phase 1	NGM Bio; MERCK: Option
NGM217	Undisclosed (Long Acting)	Diabetes	Phase 1	NGM Bio; MERCK: Option
NGM621	Complement C3 Antagonistic Antibody (Long Acting)	Dry Age-Related Macular Degeneration (AMD)	Phase 1	NGM Bio; MERCK: Option
NGM386	GDF15 Analog (Once Daily)	Metabolic	Phase 1	NGM Bio: Wholly-Owned
NGM395	GDF15 Analog (Long Acting)	Metabolic	Preclinical	NGM Bio: Wholly-Owned

FGF19: fibroblast growth factor 19; FGFR1c/KLB: fibroblast growth factor receptor 1c/beta-klotho; GDF15: growth differentiation factor 15; GFRAL: glial-cell-derived neurotrophic factor receptor alpha-like

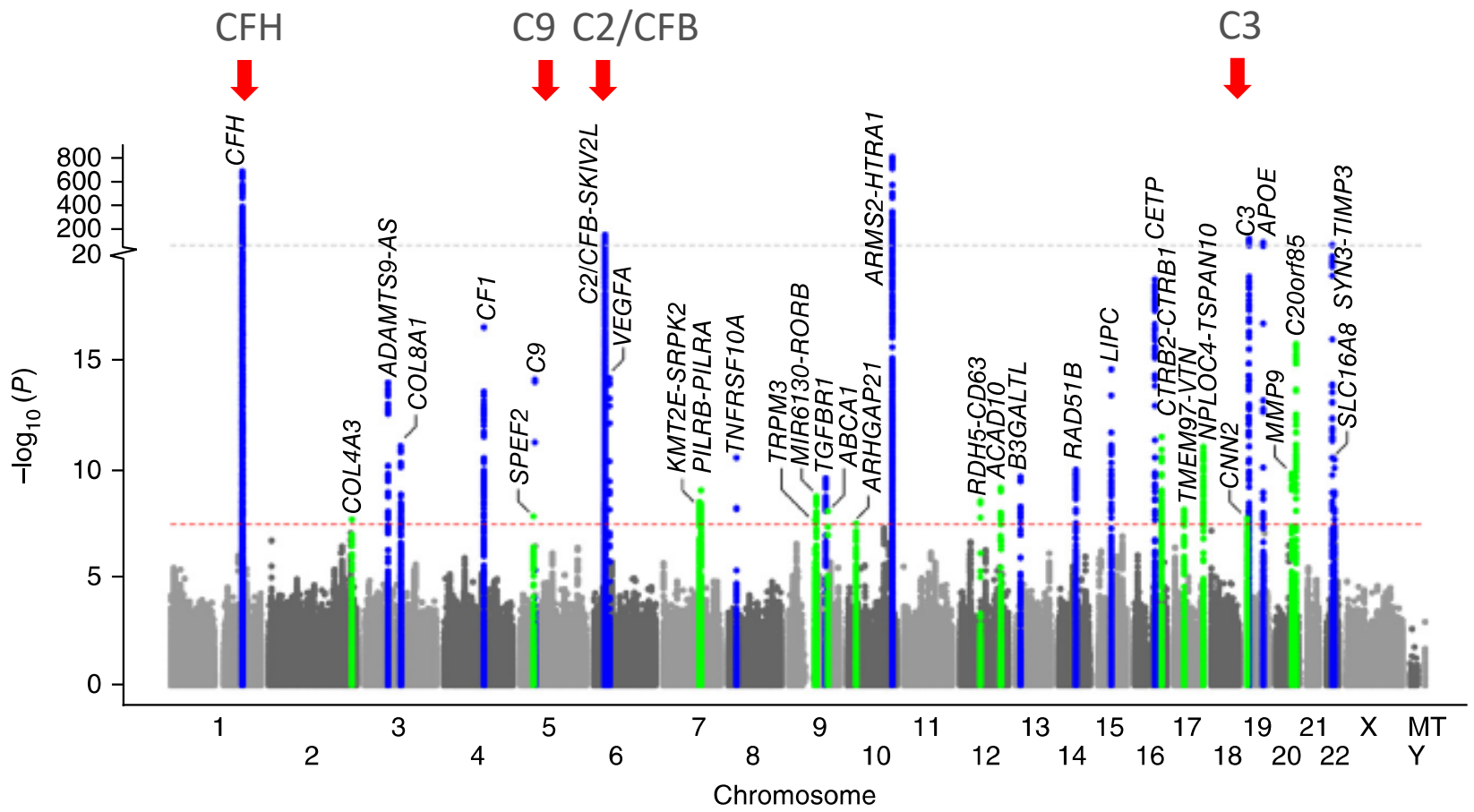
Geographic Atrophy (GA) is an Advanced Form of AMD



- Geographic atrophy (GA) is the dry form of advanced AMD, characterized by progressive and irreversible loss of photoreceptors, retinal pigment epithelium (RPE) and choriocapillaris
- GA is typically bilateral and lesion enlargement results into irreversible blindness
- GA affects ~5 million people globally and ~ 1 million people in the US
- Currently no approved treatment for GA

Fleckenstein et al, Ophthalmology 2018, 125(3): 369-390; Friedman et al, Arch Ophthalmol. 2004, 122: 564-572

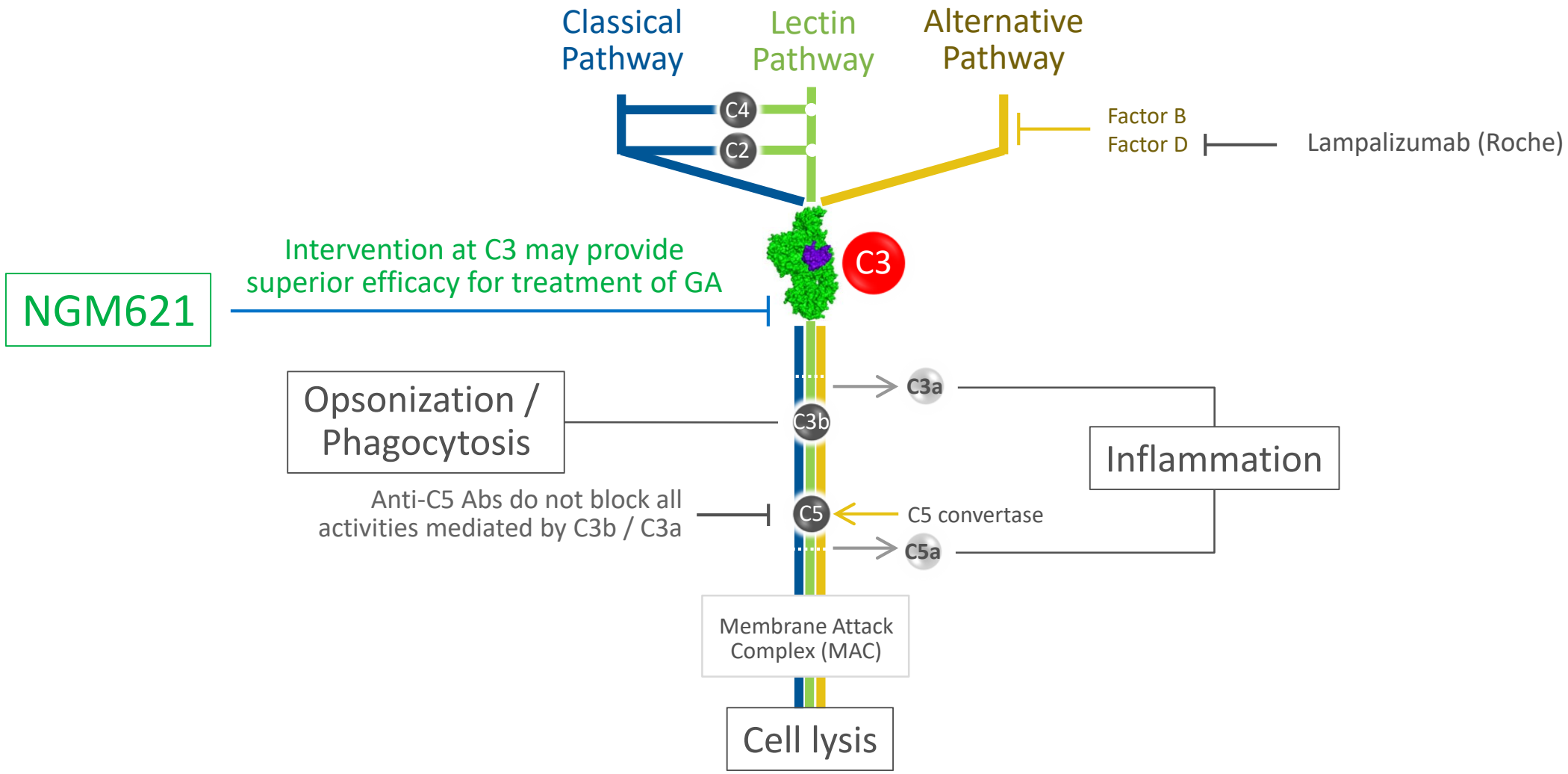
Complement Activation is Associated with Development of Advanced AMD



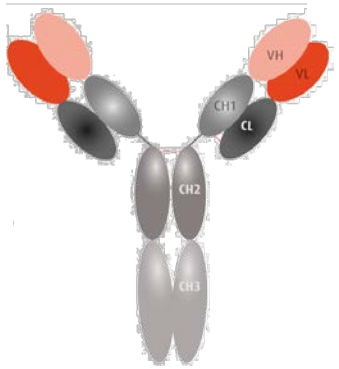
Variants in the complement pathway account for the majority of the known genetic risk for AMD

Fritsche et al. Nat Genet 2016

NGM621 Targets Complement C3, Blocking All Three Pathways of Complement Activation

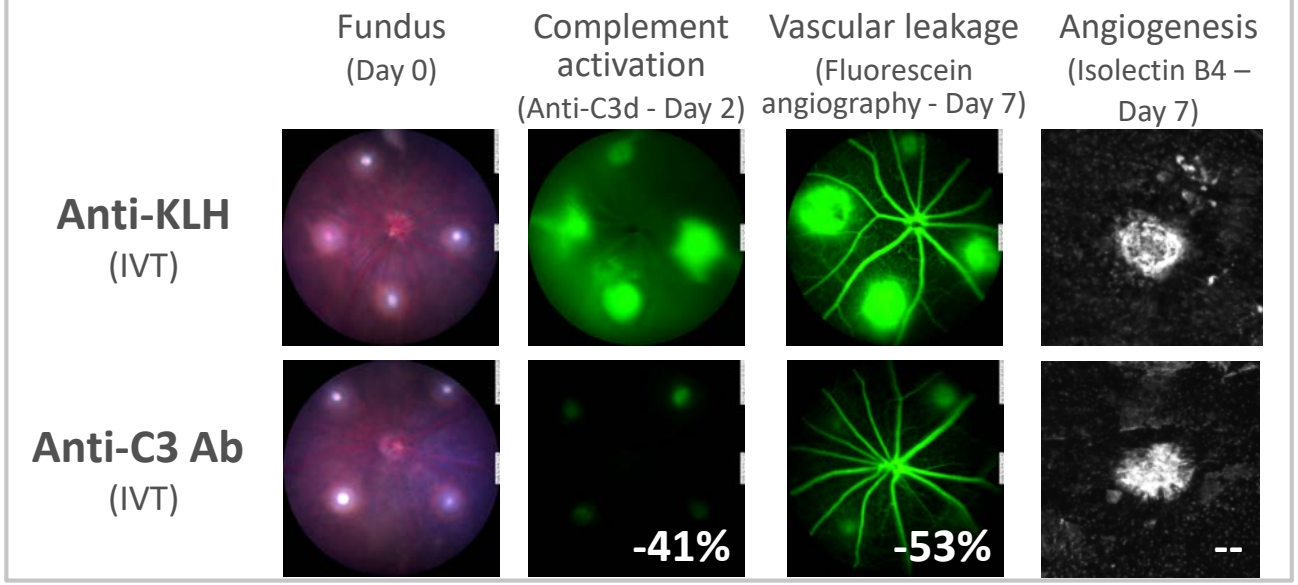


NGM621: A Potent Anti-Complement C3 Antibody

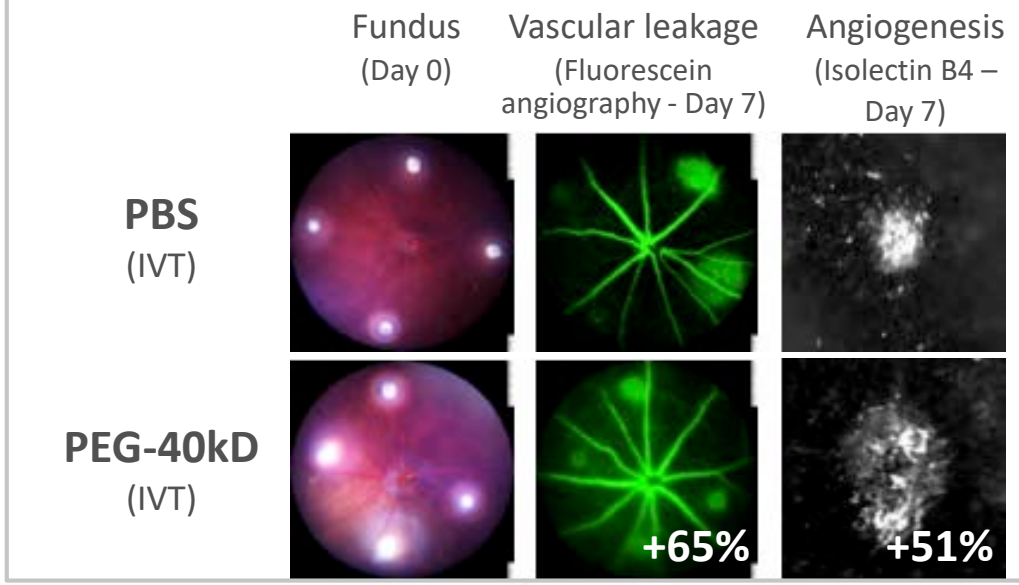


- Antibody that has high binding affinity for human C3 (K_D < 1 nM)
- Potent inhibition of both classical and alternative pathways of complement activation (IC_{50} ~ 5-6 nM)
- Potential for QM (monthly) or Q2M (EOM) intravitreal dosing

Anti-C3 Ab reduces vascular leakage in laser injury-induced CNV in mice



Antibody is preferred modality; PEG-40kD exacerbates laser injury-induced CNV in mice

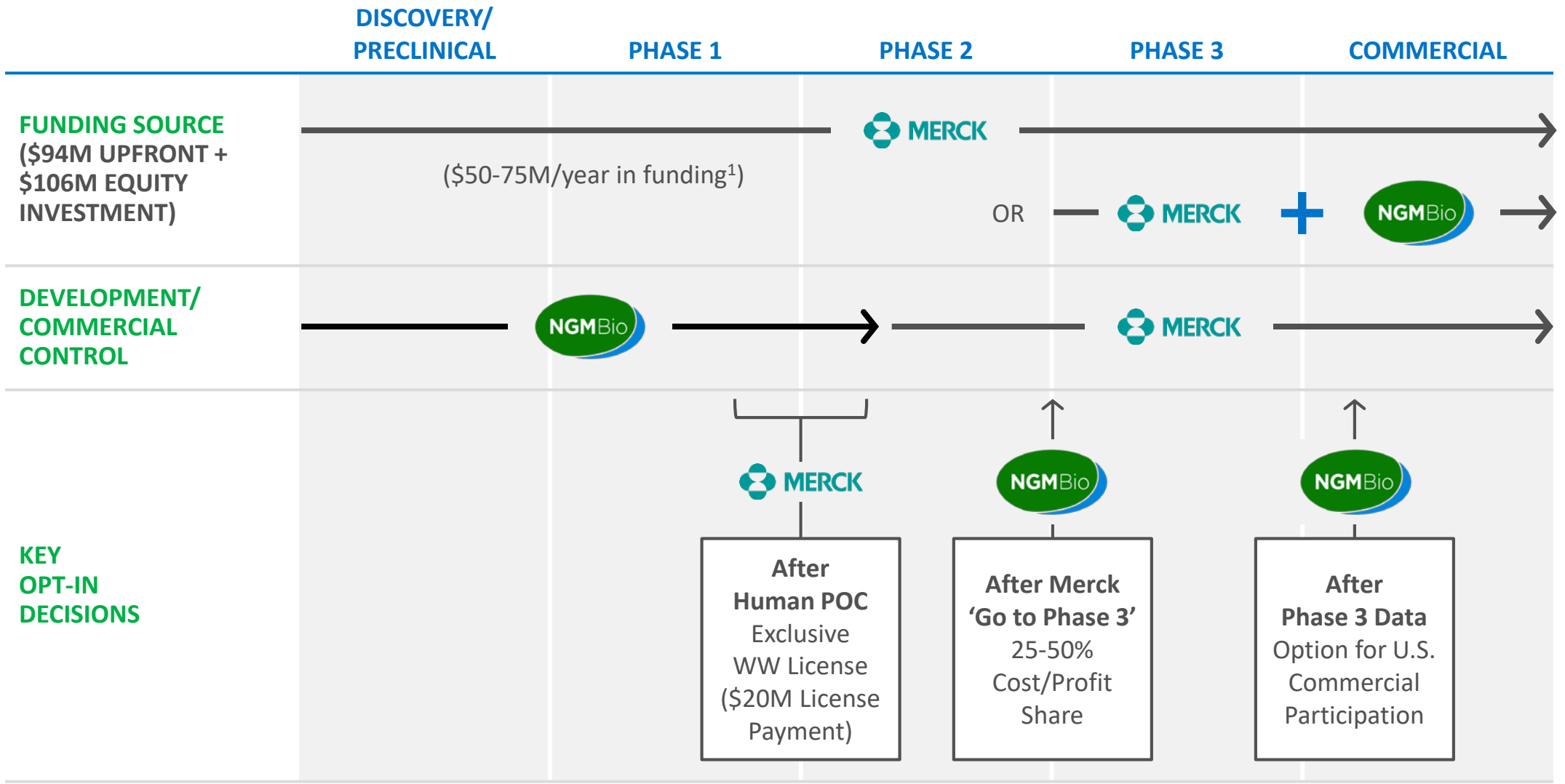


NGM621 Development

Initiated Phase 1 open-label single dose and multiple dose study in patients with GA

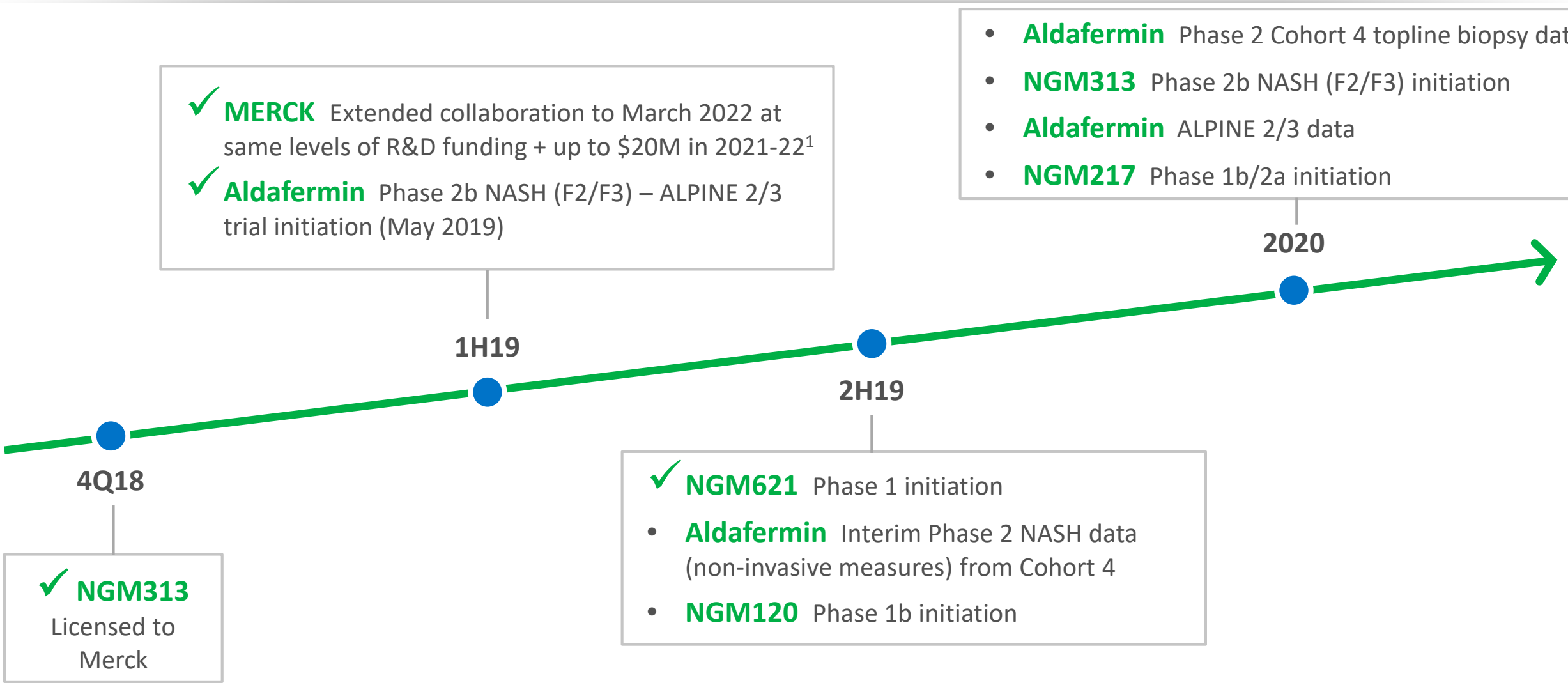
- Primary objective to evaluate the safety, tolerability and pharmacokinetics of intravitreal injection(s) of single and multiple doses of NGM621
  - Estimated enrollment of ~24 patients with GA secondary to AMD
- Study enables a potential Phase 2 POC study in GA
- Favorable tolerability profile observed from 5W GLP toxicology study in monkey
- Program is subject to Merck option to license the program

Our Merck Collaboration: Growth-Accelerating Partnership



[1] *Merck has committed to provide R&D reimbursement of up to $50 million per year. If our R&D expenses exceed $50 million in a given year, Merck can either reimburse up to an additional $25 million for use in funding IND-enabling or later-staged activities or to provide us with the equivalent value in in-kind services for such activities.*

Momentum with Potential Value-Driving Catalysts



[1] *Merck has committed to provide R&D reimbursement of up to $50 million per year; if our R&D expenses exceed $50 million in a given year, Merck can either reimburse up to an additional $25 million for use in funding IND-enabling or later-staged activities or to provide us with the equivalent value in in-kind services for such activities. In lieu of a $20 million extension fee payable to NGM, Merck will make additional payments totaling $20 million in R&D funding from Jan 2021-Mar 2022.*

NGM Biopharmaceuticals, Inc. Corporate Overview

Novel Biology. Powerful Medicines. Transformative Impact.

NGMBio

Next Generation Medicines