NGM Biopharmaceuticals, Inc. Corporate Overview

Novel Biology. Powerful Medicines. Transformative Impact.

NASDAQ: NGM

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 “anticipated,” “believe,” “continue,” “can,” “could,” “estimate,” “expect,” “intend,” “may,” “plan,” “potential” “predict,” “preliminary,” “should,” “will” or the negative of these terms or other similar expressions. These statements include those related to potential indications for product candidates in NGM’s clinical and preclinical pipeline; the planned development, timing, enrollment and results of NGM’s clinical trials, including the announcement of topline data and other data from the Phase 2 clinical study of aldafermin (NGM282) in patients with NASH and the initiation of trials of NGM313 and NGM217; NGM’s option to participate in the economic return of NGM313 or ability to receive milestone payments or royalties from NGM313; the potential complementarity of aldafermin and NGM313; the continued development and potential dosing of NGM621; the safety, tolerability and efficacy of NGM’s product candidates; and NGM’s expectation of potential value driving catalysts. 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 these slides and the accompanying presentation. 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 September 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 **four 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

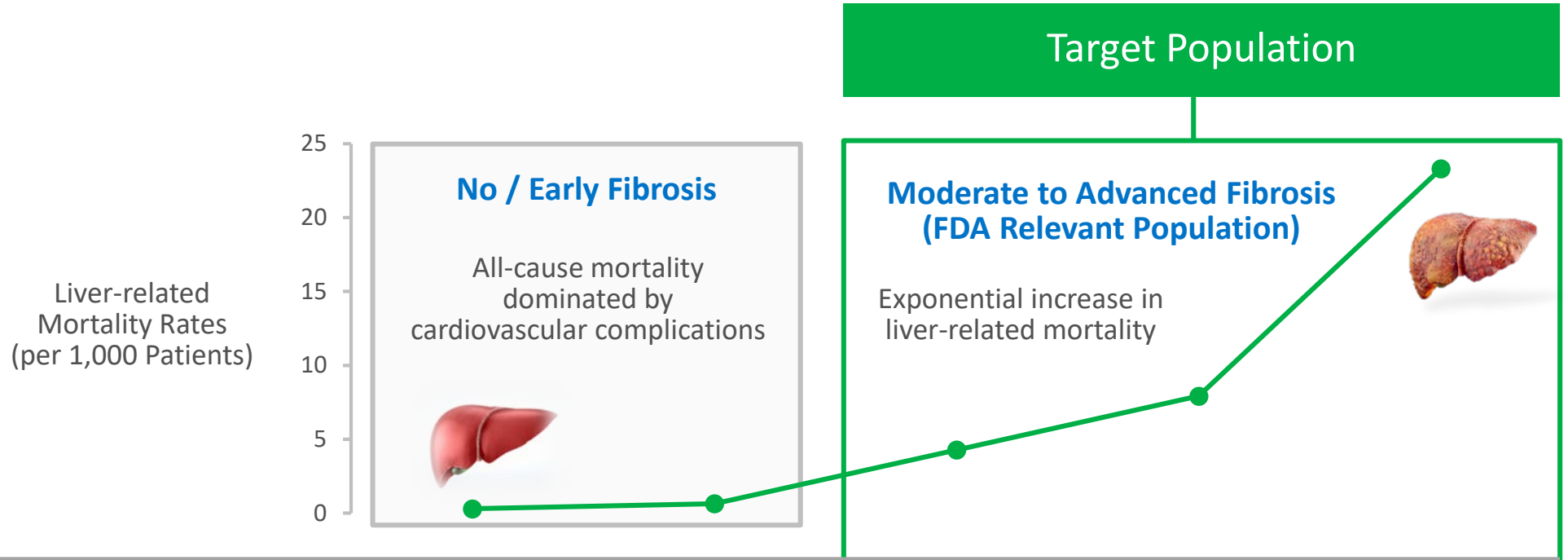
Our Expansive Pipeline

6 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 Inhibitory Antibody (Long Acting)	Dry Age-Related Macular Degeneration (AMD)	Phase 1	NGM Bio; MERCK: Option
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.

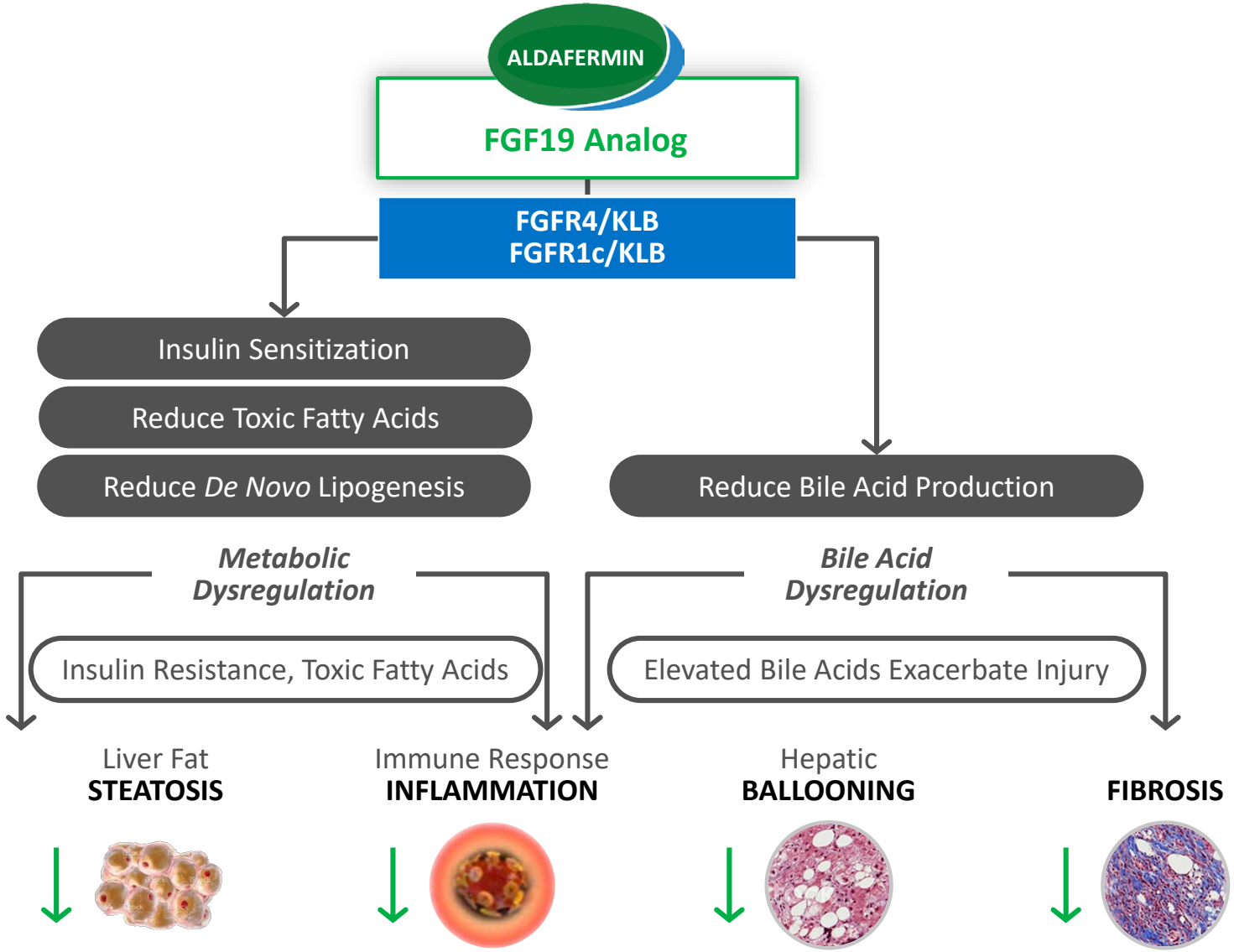
[1] *Canbay et al, Visc Med 2016, 32: 234-238.*

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

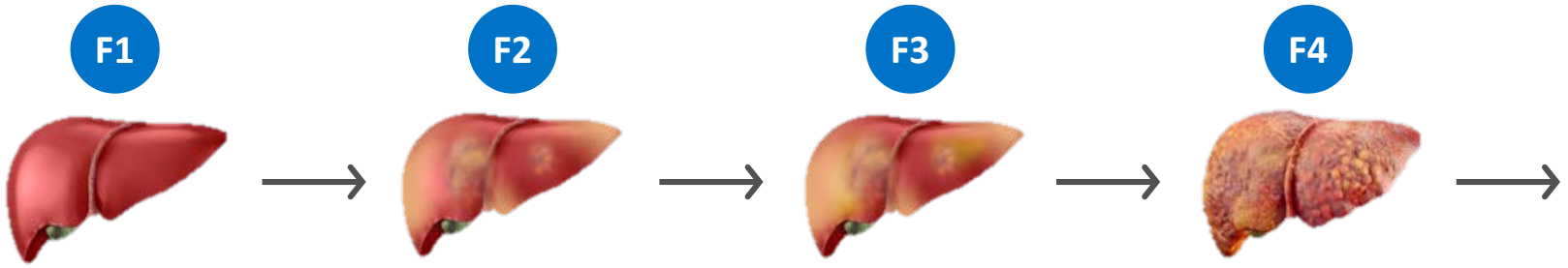
Competitive Landscape

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

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 topline data 1Q20)
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)

HISTOLOGY (Biopsy)

NON-INVASIVE MEASURES

KEY ENDPOINTS/ Assessment Measures

STEATOSIS

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

INFLAMMATION

BALLOONING

ALT/AST (Biomarkers)

FIBROSIS

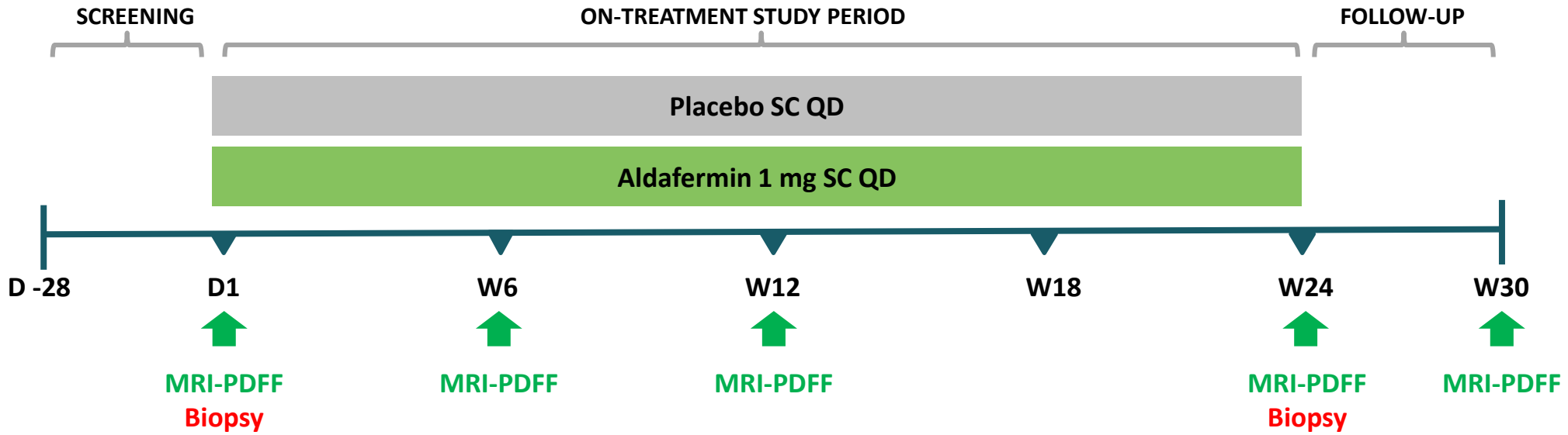
ELF/PRO-C3 (Biomarkers)

BILE ACIDS

C4[1] (Biomarker)

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

Cohort 4: A 24-Week, Double-Blind, Placebo-Controlled Multi-Center Ph2 Study of Aldafermin in Patients with Biopsy-Proven NASH



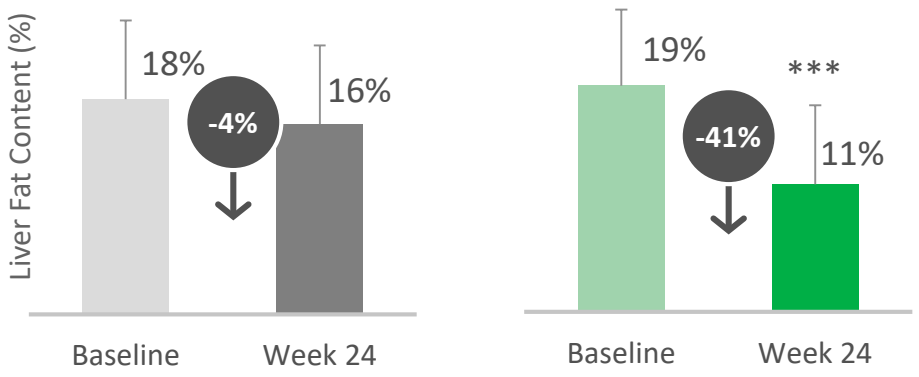
- 78 subjects randomized 2:1 to aldafermin 1 mg or placebo
- Key inclusion criteria include:
  - Biopsy confirmed NASH with NAS ≥4 (1 point in each component); stage 2 or 3 liver fibrosis (F2 or F3 by NASH CRN criteria);
  - Absolute liver fat content (LFC) ≥8% by MRI-PDFF;
  - ALT ≥ 19 IU/L in females, ALT ≥ 30 IU/L in males
- **Primary endpoint**: change from baseline in absolute LFC (as measured by MRI-PDFF) in subjects with histologically confirmed NASH after 24 weeks of treatment
- A pre-specified **interim analysis on MRI-PDFF and select biomarkers** was conducted when **38 subjects** completed Week 24 procedures
- Rosuvastatin (ROS 20 mg) started at W2 if LDL-C rise of 10 mg/dL observed
  - ROS dose titrated up to 40 mg at W4 to W8 if LDL-C remains above baseline

Summary of Phase 2 Data: Liver Fat Content

MEASURE/ BIOMARKER

24-Week Cohort 4 IA

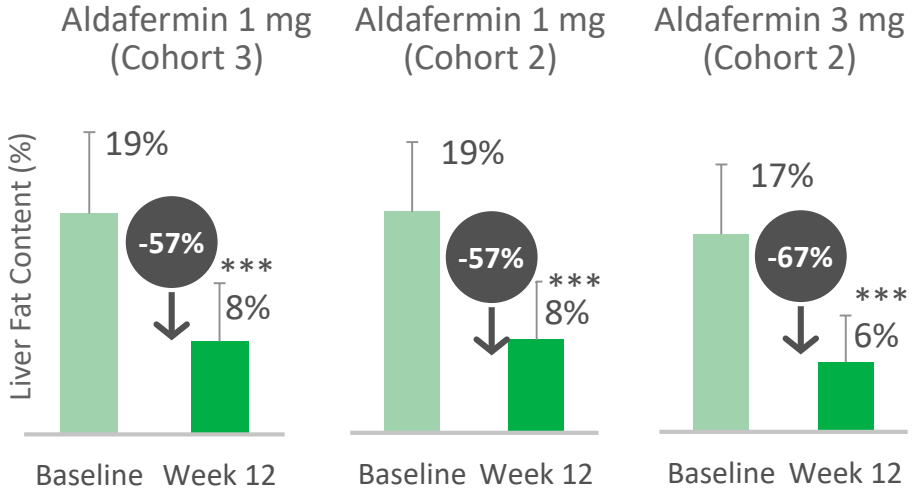
Liver Fat Content by MRI-PDFF[1]



17% (Placebo) and 72% (aldafermin 1 mg) of Subjects Achieved ≥ 30% Relative LFC Reduction at Week 24

Previous Cohorts

Liver Fat Content by MRI-PDFF[1]



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

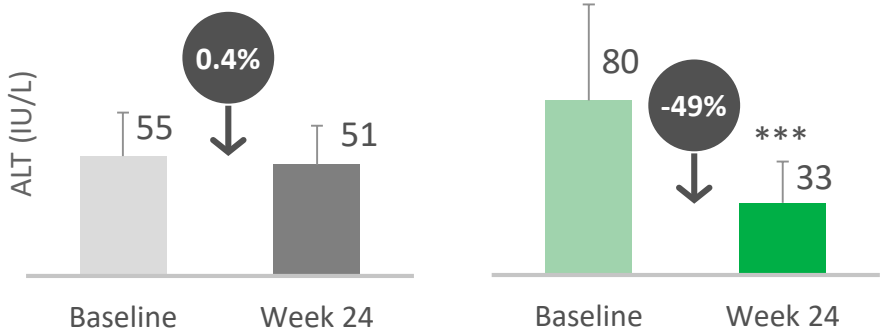
[1] *Relative values are calculated as mean change from baseline*
Cohort 4 interim analysis - Preliminary data; Cohorts 2-3 preliminary data

Summary of Phase 2 Data: ALT

MEASURE/ BIOMARKER

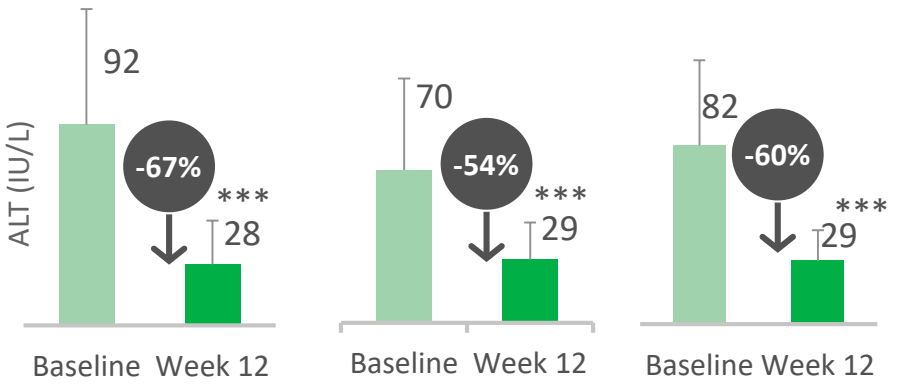
24-Week Cohort 4 IA

ALT (IU/L)[1]



Previous Cohorts

ALT (IU/L)[1]



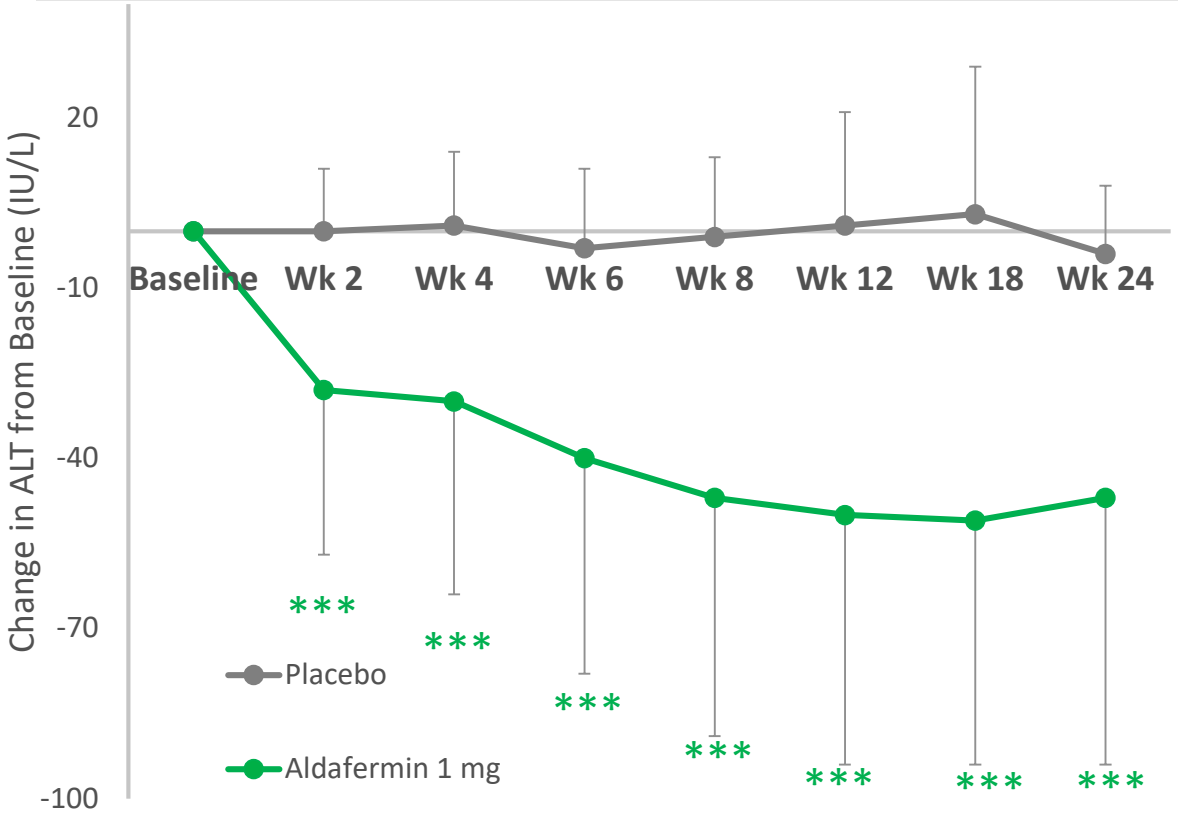
***P<0.001, **P<0.01, *P<0.05 vs baseline

[1] *Relative values are calculated as mean change from baseline*
Cohort 4 interim analysis - Preliminary data; Cohorts 2-3 preliminary data

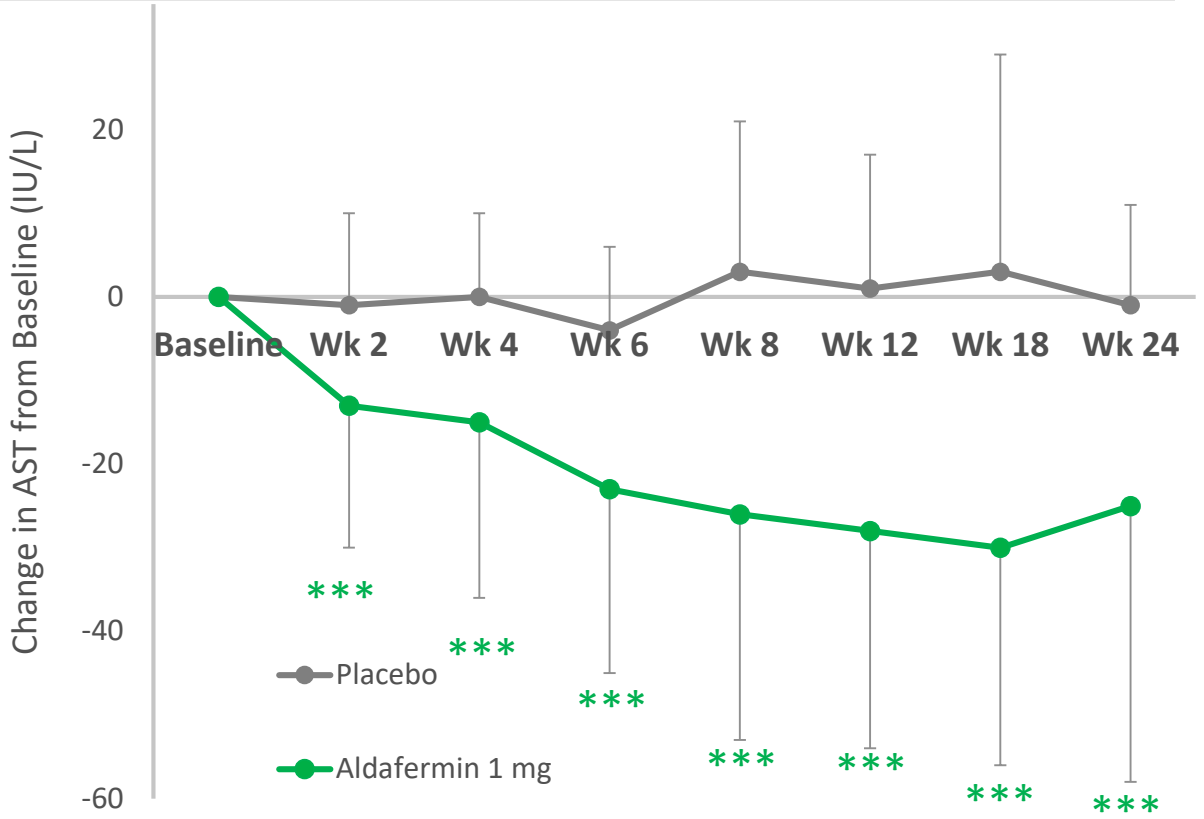
Rapid and Sustained Decreases in ALT and AST with Aldafermin

INFLAMMATION

ALT (IU/L)



AST (IU/L)



Cohort 4 interim analysis; Preliminary data

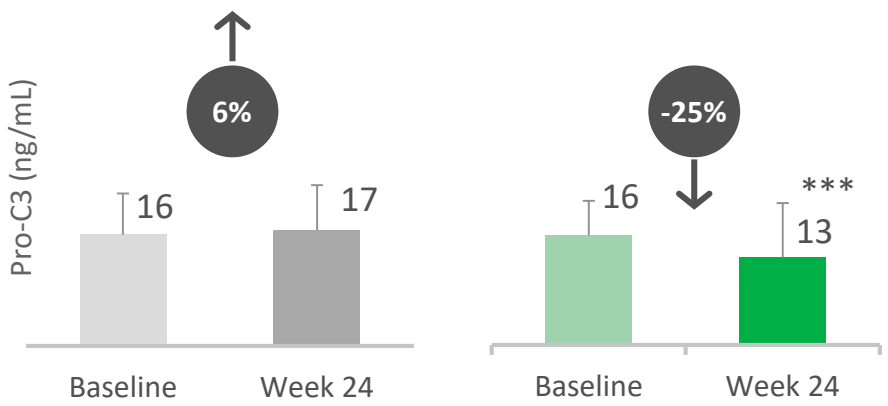
****P<0.001 vs. baseline (LS mean)*

Summary of Phase 2 Data: PRO-C3

MEASURE/ BIOMARKER

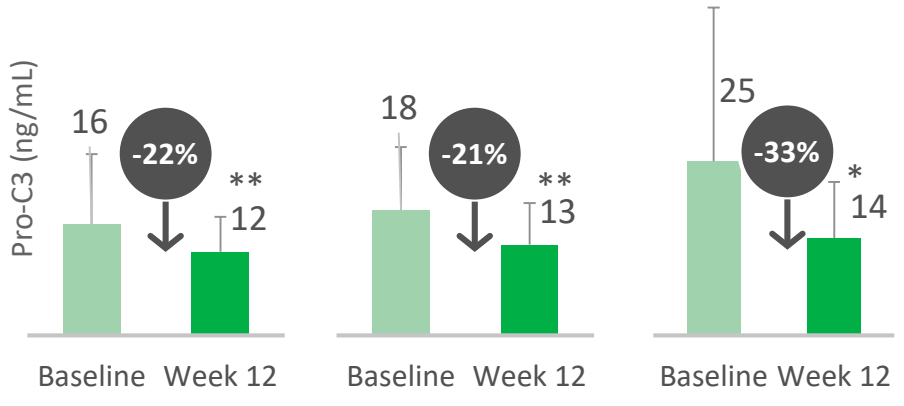
24-Week IA

PRO-C3 (ng/mL)[1]



Previous Cohorts

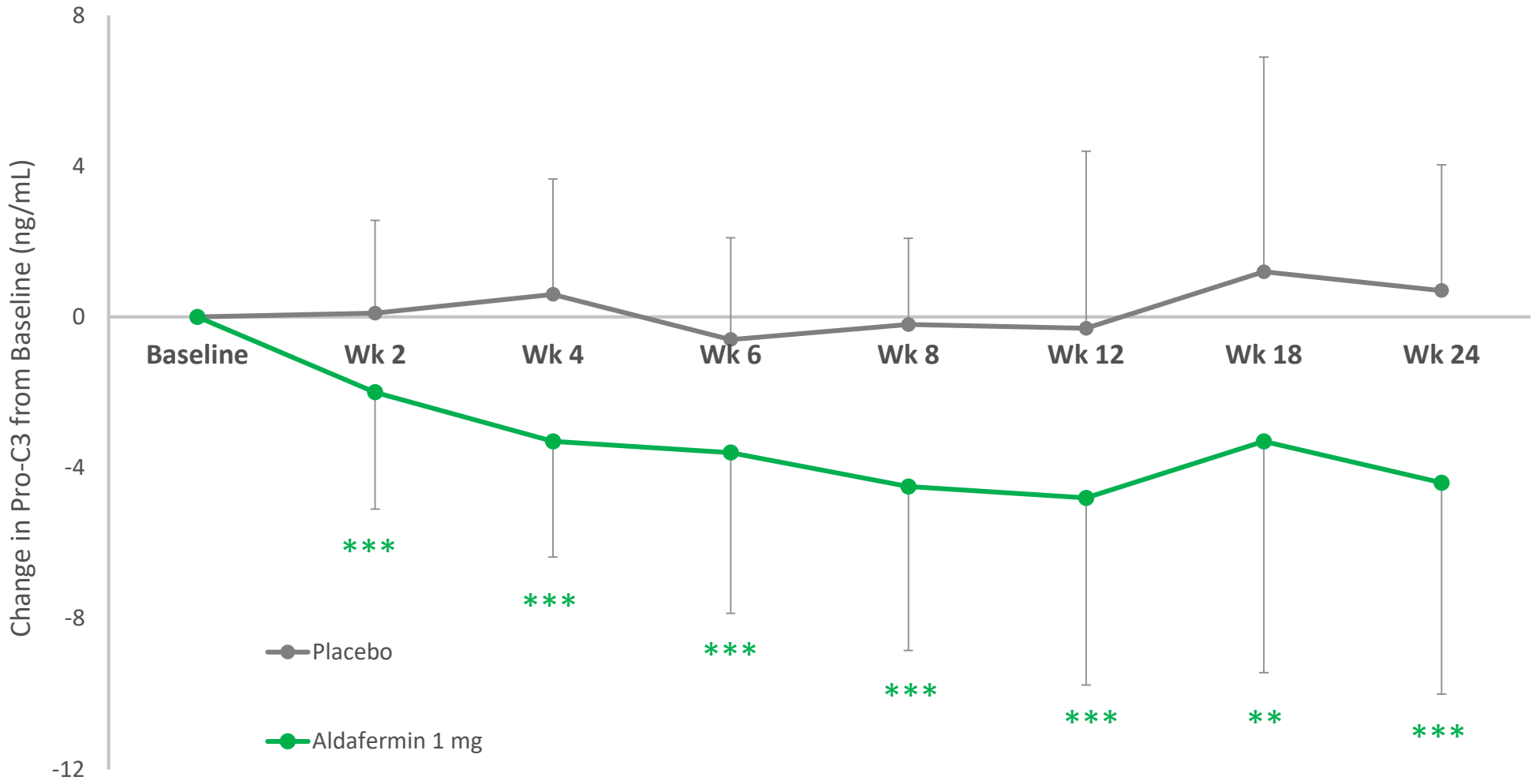
PRO-C3 (ng/mL)[1]



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

[1] *Relative values are calculated as mean change from baseline*
Cohort 4 interim analysis - Preliminary data; Cohorts 2-3 preliminary data

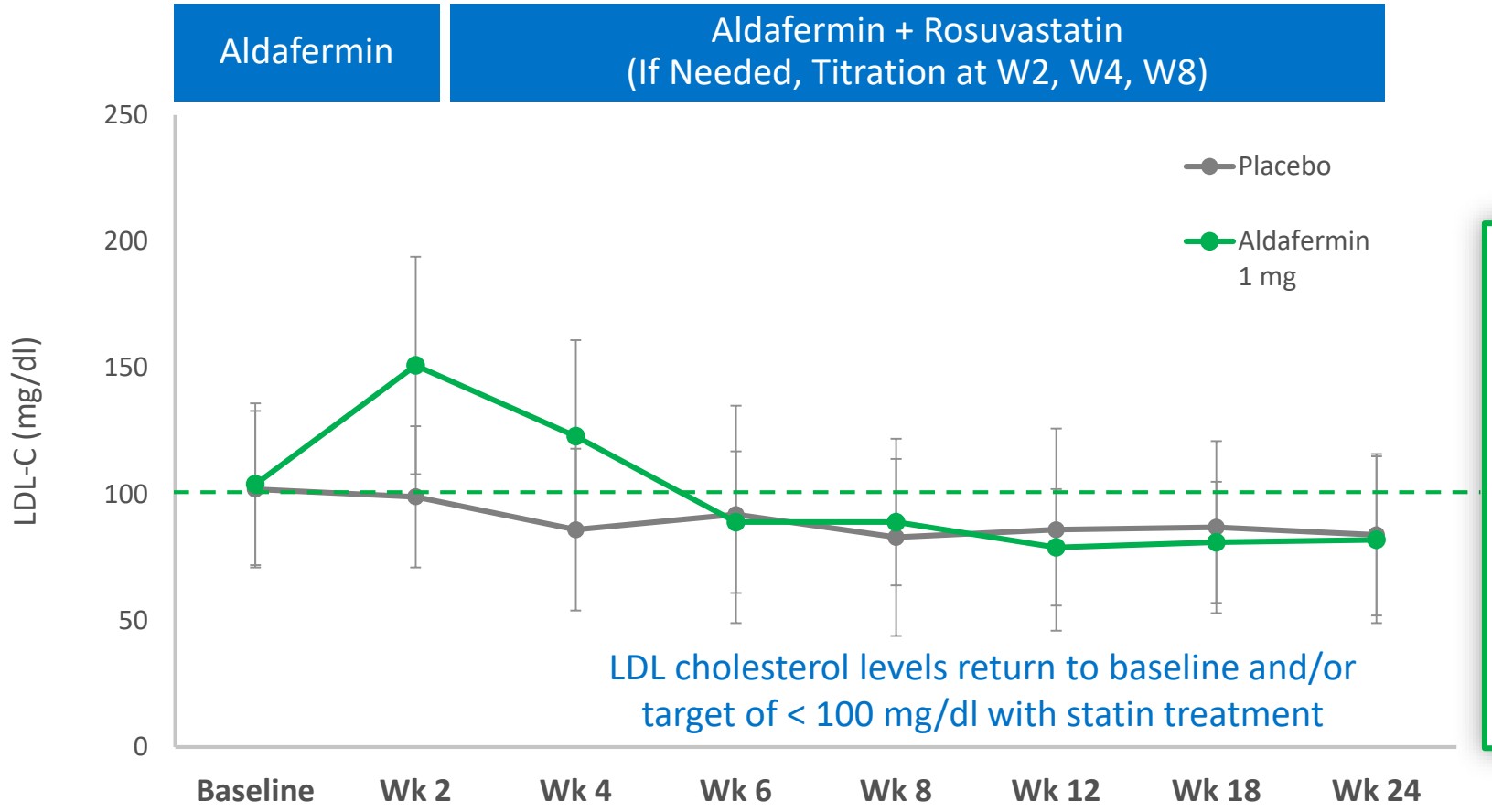
Rapid and Sustained Statistically Significant Reduction in PRO-C3 as Early as Week 2



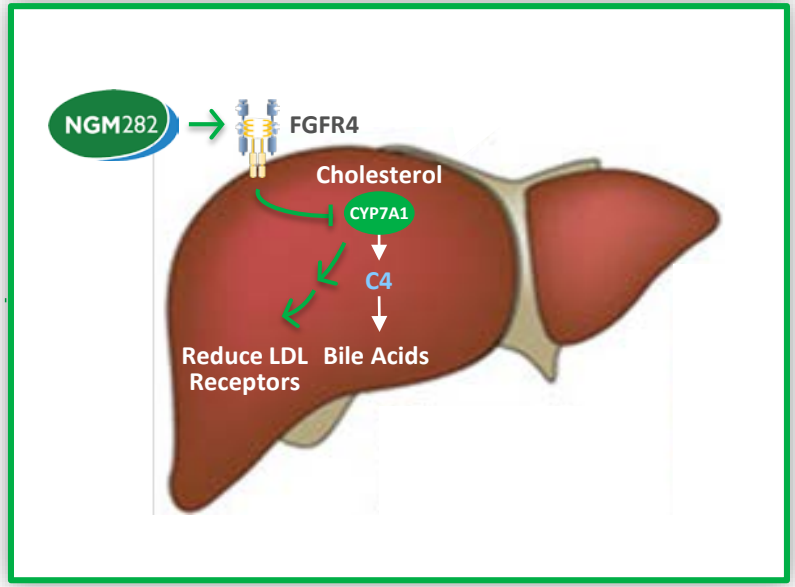
****P<0.001, **P<0.01 vs. baseline (LS mean)*

Cohort 4 interim analysis; Preliminary data

Cohort 4 Interim Analysis:
LDL-C 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



Preliminary results; Cohort 4 Interim analysis

Cohort 4 Interim Analysis: Adverse Event Summary

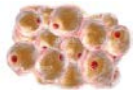
TEAE Classification	Placebo (N=13)	Aldafermin 1.0 mg (N=25)
Any TEAE	11 (84.6%)	22 (88.0%)
TEAE Leading to Drug Withdrawal	1 (7.7%)	0 (0%)
Serious TEAE	2 (15.4%)	0 (0%)
Drug-Related TEAE	8 (61.5%)	13 (52.0%)
TEAE Leading to Death	0 (0 %)	0 (0%)

MedDRA Preferred Term	Placebo (N=13)	Aldafermin 1.0 mg (N=25)
Diarrhea	1 (7.7%)	7 (28%)
Headache	5 (38.5%)	3 (12%)
Nausea	4 (30.8%)	3 (12%)
Arthralgia	0 (0%)	3 (12%)
Diabetes Mellitus	2 (15.4%)	2 (8%)
Influenza like Illness	2 (15.4%)	1 (4%)
Loose stools	2 (15.4%)	1 (4%)
Pruritus	2 (15.4%)	1 (4%)
Hypertension	2 (15.4%)	1 (4%)
Frequent bowel movements	0 (0%)	1 (4%)
Increased frequency of defecation	0 (0%)	1 (4%)
Peripheral Edema	2 (15.4%)	0 (0%)
Fatigue	2 (15.4%)	0 (0%)

Cohort 4 interim analysis; Preliminary data

Consistent Robust Reduction to Near Normal Levels Across Key Biomarkers of Disease

Mean relative reduction across Cohorts 1-4[1]
(1 mg and 3 mg doses of aldafermin, 12W and 24W)



STEATOSIS

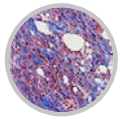
Liver Fat Content by MRI-PDFF

41% – 67%

INFLAMMATION

ALT

43% – 67%



FIBROSIS

PRO-C3

16% – 33%

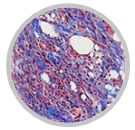
Absolute level (EOT)	**6-11%**	**29-33 IU/L**	**12-14 ng/mL**

NEAR NORMAL LEVELS ACHIEVED WITH ALDAFERMIN TREATMENT

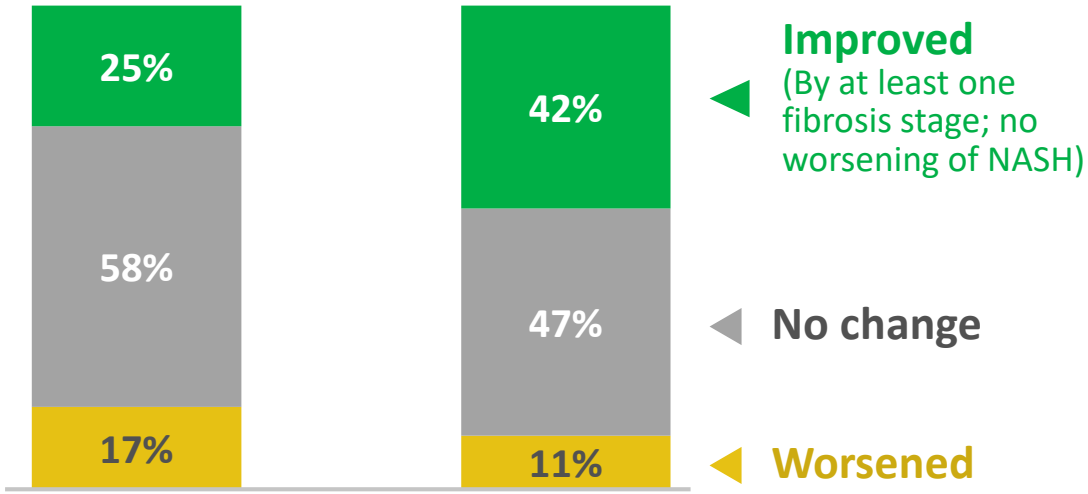
[1] Relative changes are calculated as mean change from baseline at end of treatment (EOT)
Cohorts 1-3 and Cohort 4 interim analysis - Preliminary data

Rapid Regression of Fibrosis at Week 12

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 two-stage fibrosis improvement

Patient Population

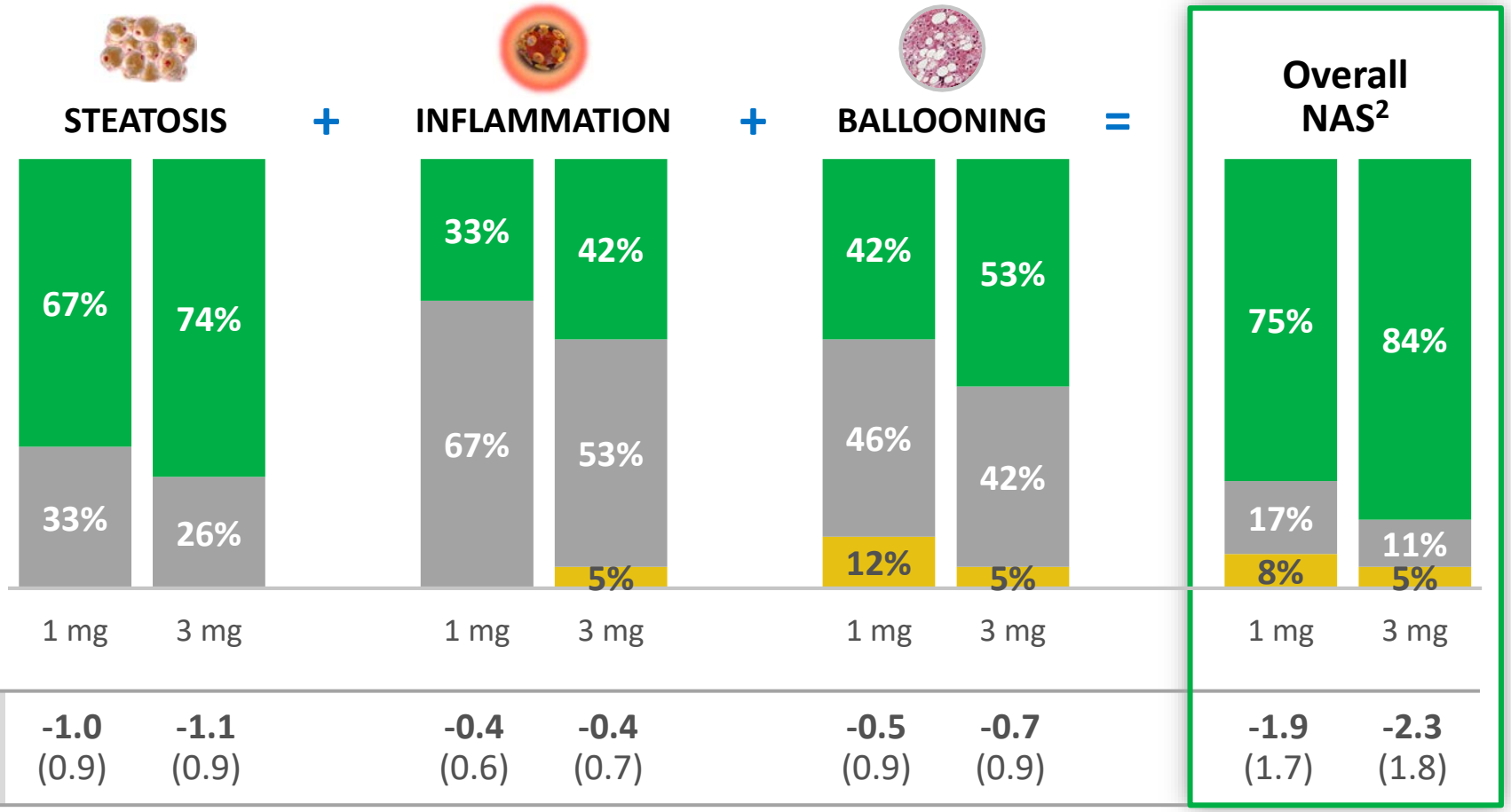
- Over 80% of patients in 3 mg Cohort 2 and 1 mg Cohort 3 had F2/F3 fibrosis at baseline

[1] *Preliminary data, Cohorts 2-3*

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)

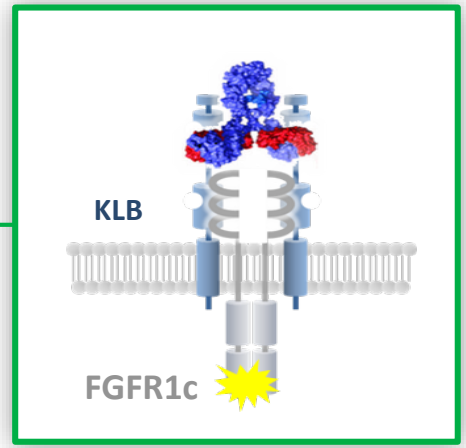
1 *Preliminary data, Cohorts 2-3*
2 *NAS: NAFLD Activity Score*

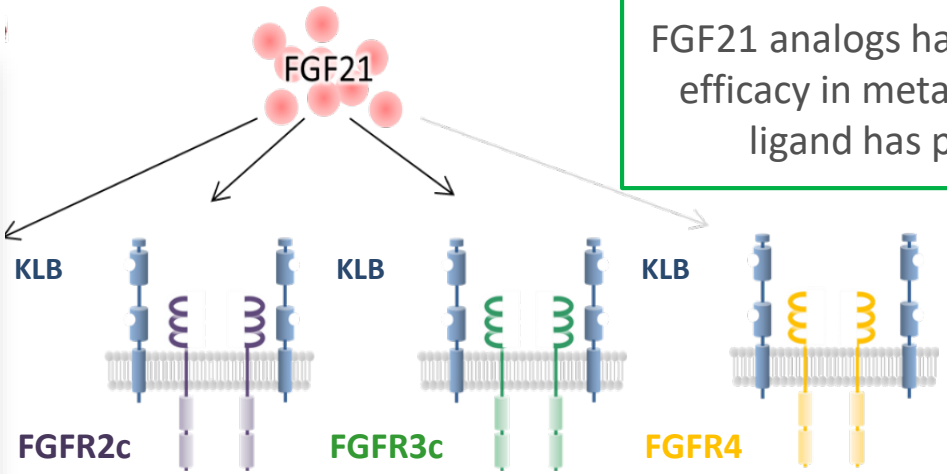
Aldafermin Development Plan

	PHASE 2 – COHORT 4	PHASE 2b (ALPINE 2/3)	PHASE 2b – COMPENSATED CIRRHOTICS (ALPINE 4)	PHASE 3 PROGRAM
Status	**Ongoing** (Topline histology anticipated 1Q20)	**Ongoing**	**In Planning**	**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**

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

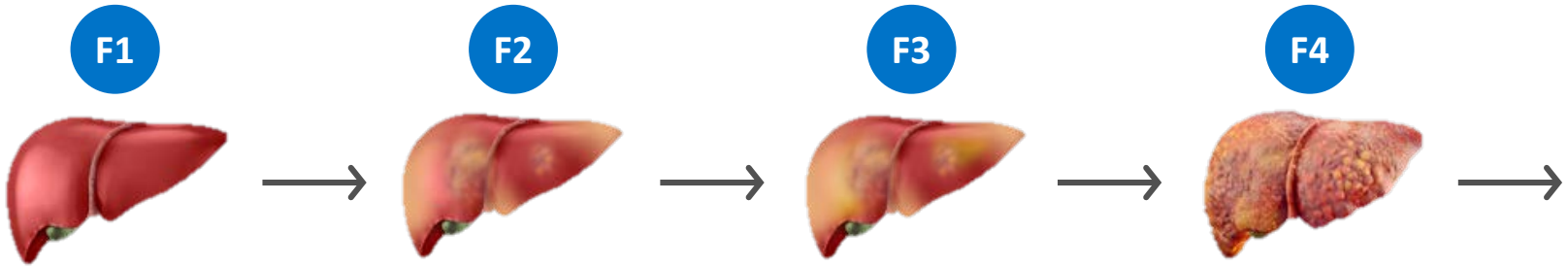
Competitive Landscape

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

Beyond NASH, an Expansive Pipeline in Other Indications

6 Development Programs

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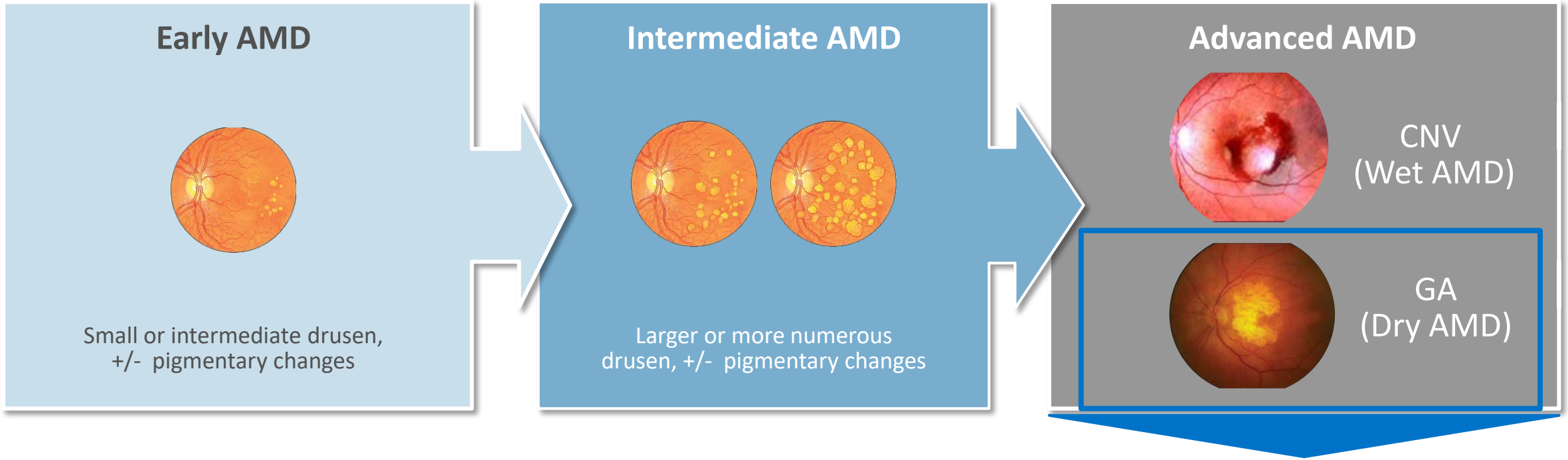
NGM313 (MK-3655)	FGFR1c/KLB Agonistic Antibody (Once Monthly)	NASH, Type 2 Diabetes	Phase 1b	MERCK: Licensed; NGM Bio
NGM120	GFRAL Antagonistic Antibody (Long Acting)	Cancer Anorexia/Cachexia Syndrome (CACS)	Phase 1	NGM Bio; MERCK: Option
NGM217	Undisclosed (Long Acting)	Diabetes	Phase 1	NGM Bio; MERCK: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

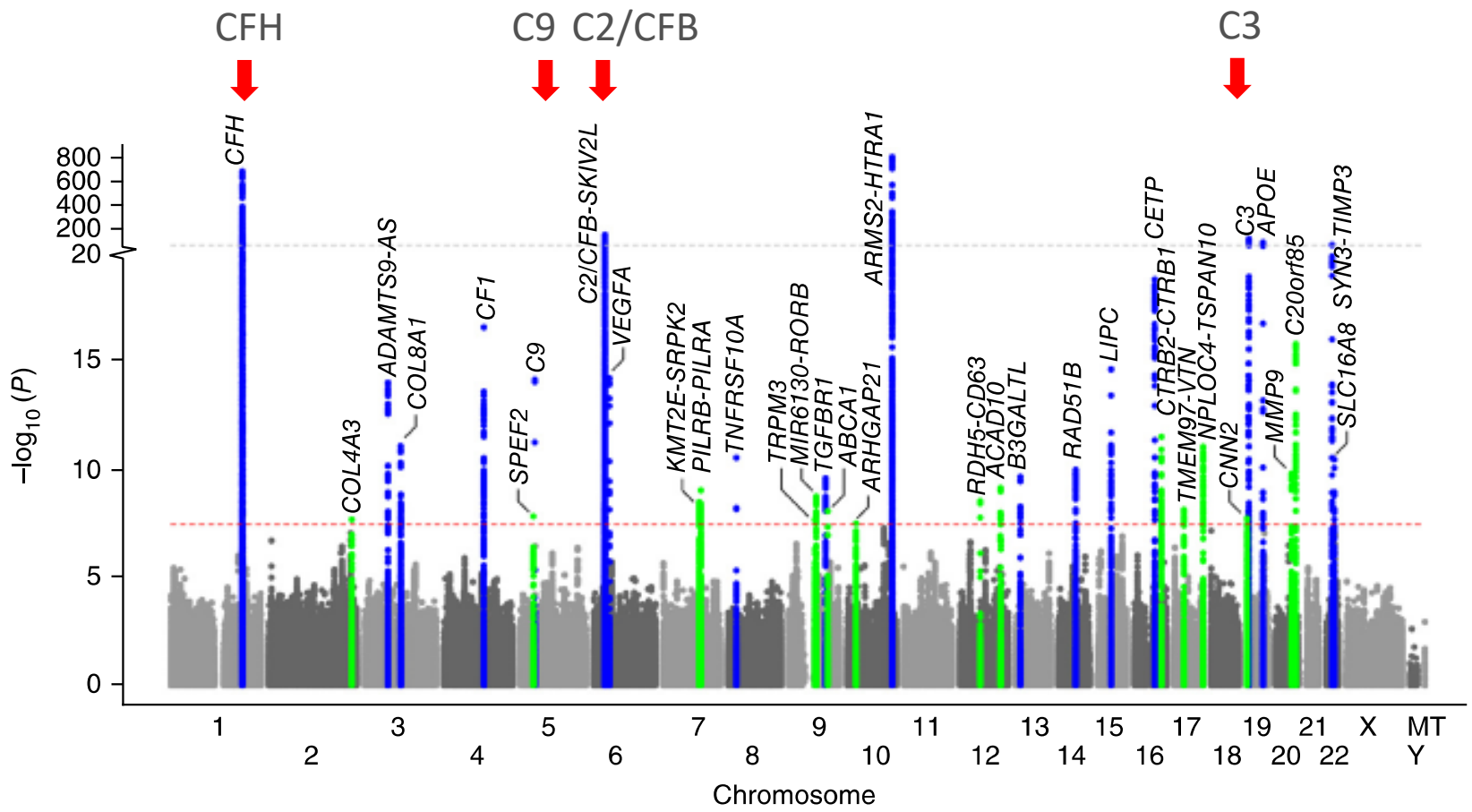
Geographic Atrophy (GA) is an Advanced Form of AMD



- 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

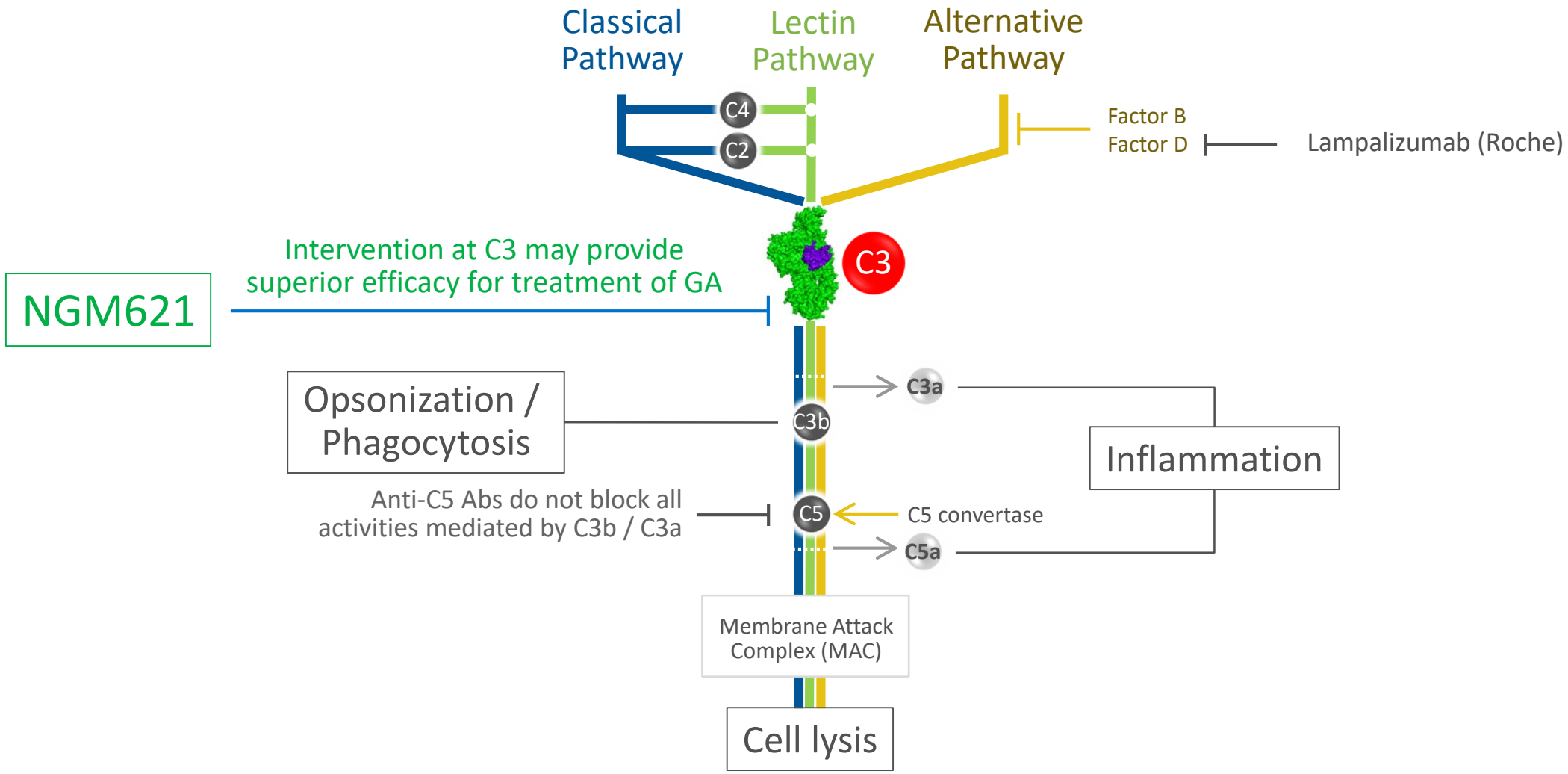
Complement Activation is Associated with Development of Advanced AMD



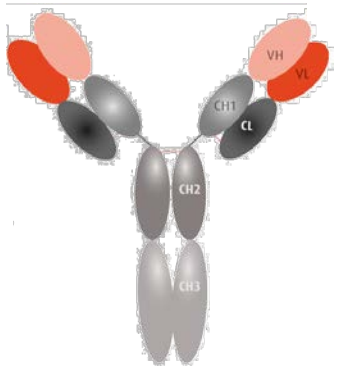
Variants in the complement pathway account for the majority of the known genetic risk for AMD

Fritsche et al. Nat Genet 2016

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

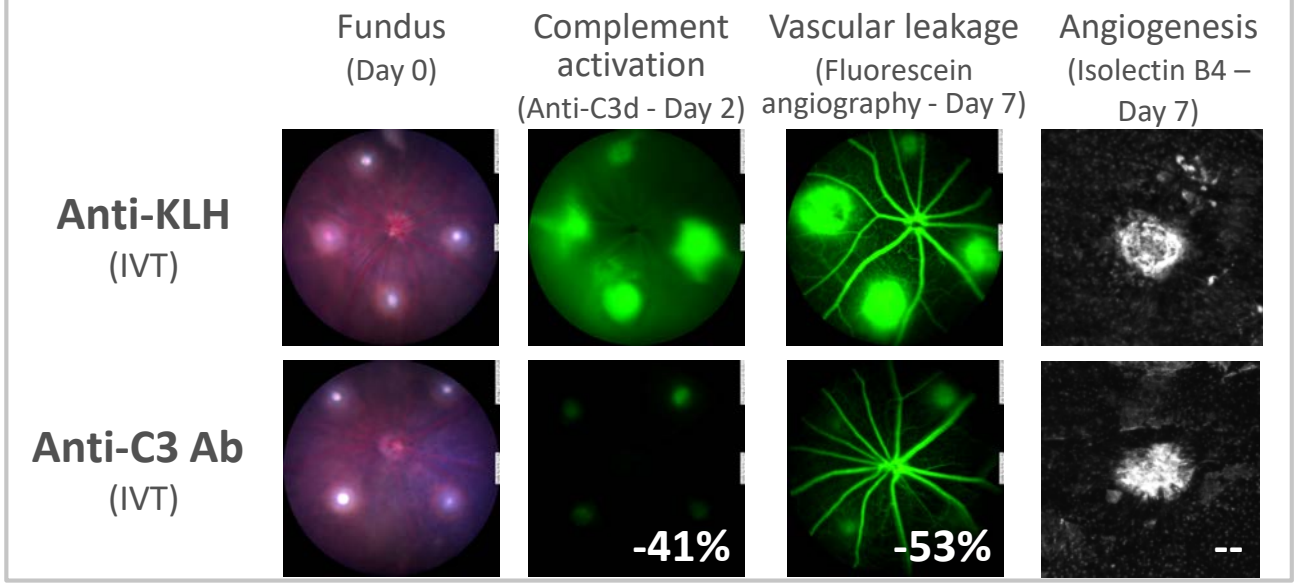


NGM621: A Potent Anti-Complement C3 Antibody

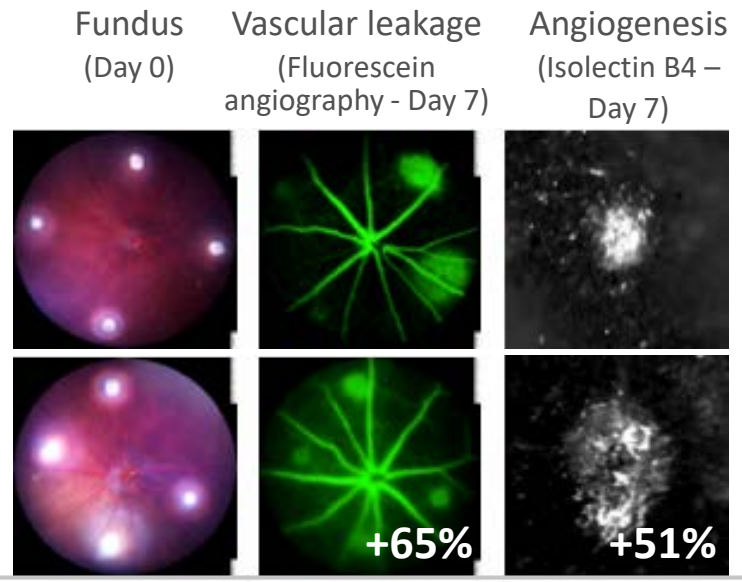


- Antibody that has high binding affinity for human C3 (K_D < 1 nM)
- Potent inhibition of both classical and alternative pathways of complement activation (IC_{50} ~ 5-6 nM)
- Potential for QM (monthly) or Q2M (EOM) intravitreal dosing

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

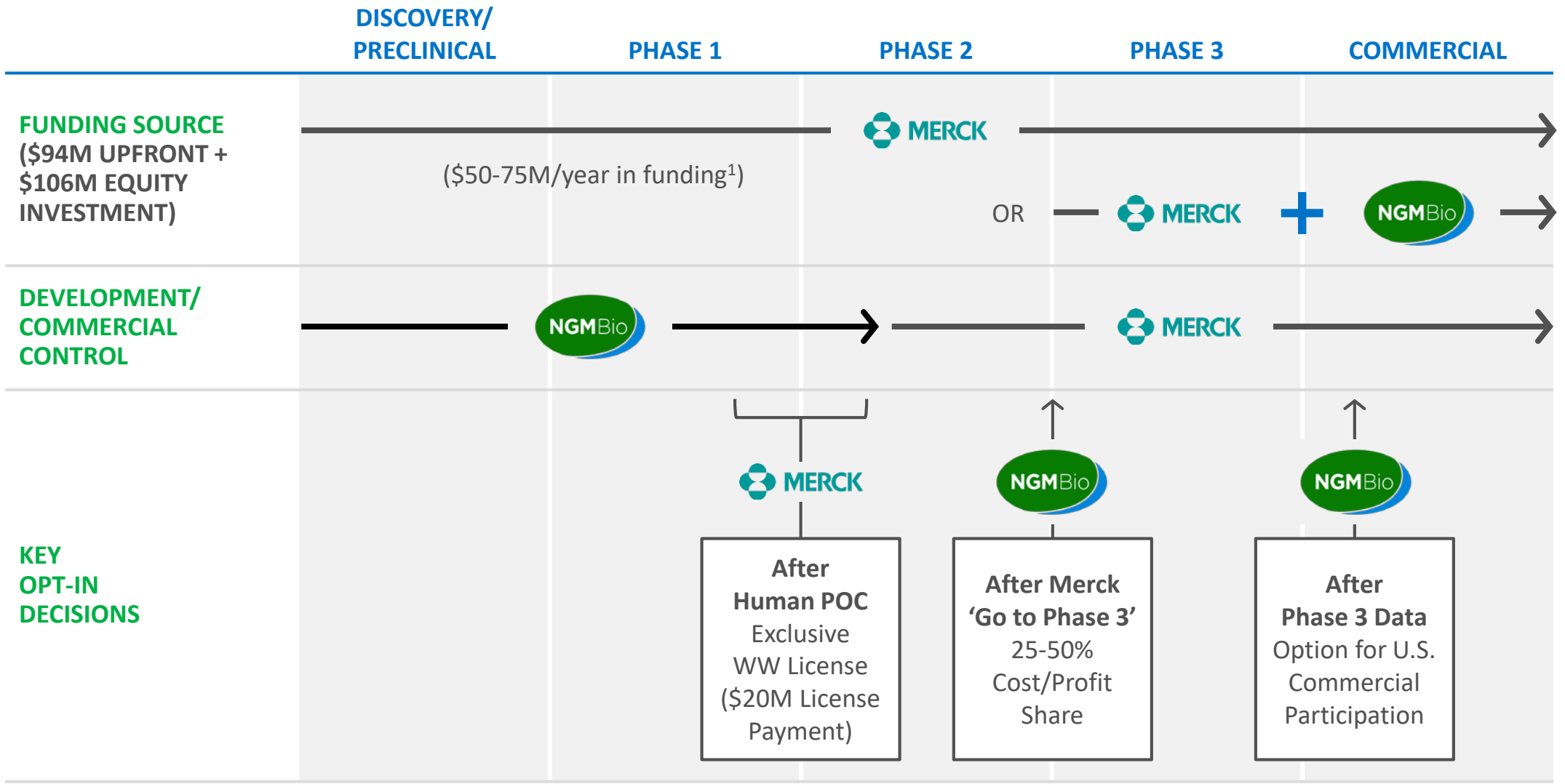


NGM621 Development

- 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 provide us with the equivalent value in in-kind services for such activities.*

Momentum with Potential Value-Driving Catalysts

4Q18

✓ **NGM313** Licensed to Merck

1H19

✓ **MERCK** Extended collaboration to March 2022 at same levels of R&D funding + up to $20M in 2021-22[1]

✓ **Aldafermin** Phase 2b NASH (F2/F3) – ALPINE 2/3 trial initiation (May 2019)

2H19

✓ **NGM621** Phase 1 initiation

✓ **Aldafermin** Interim Phase 2 NASH data (non-invasive measures) from Cohort 4

- **NGM120** Planned Phase 1a/1b initiation

2020

- **Aldafermin** Phase 2 Cohort 4 topline biopsy data
- **NGM313** Planned Phase 2b NASH (F2/F3) initiation
- **Aldafermin** ALPINE 2/3 data
- **NGM217** Planned Phase 1b/2a initiation

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NGM Biopharmaceuticals, Inc. Corporate Overview

Novel Biology. Powerful Medicines. Transformative Impact.

NASDAQ: NGM

Next Generation Medicines