Novel Biology. Powerful Medicines. Transformative Impact.

NGM Biopharmaceuticals, Inc.

CORPORATE OVERVIEW

FEBRUARY 2020

NASDAQ: NGM

Safe Harbor Statement

This presentation contains forward-looking statements, including, but not limited to, statements regarding potential indications for, and planned development of, product candidates in NGM's pipeline, including aldafermin (NGM282); the planned timing of initiation, enrollment and results of NGM's clinical trials; the potential activity, complementarity, safety, tolerability and efficacy of NGM's product candidates, including aldafermin and specifically including its differentiation and the potential benefits of extended treatment with aldafermin; NGM's option to participate in the economic return of any programs licensed by Merck; NGM's expectation of potential value-driving catalysts and the timing thereof; and any other statements other than statements of historical facts. Because such statements deal with future events and are based on NGM's current plans, objectives, estimates and expectations, they are subject to significant risks and uncertainties and actual results and achievements and the timing of events could differ materially from those described in or implied by the statements herein. Such risks and uncertainties include, without limitation, those associated with the costly and time-consuming pharmaceutical product development process and the uncertainty of clinical success, including risk related to failures or delays in initiating, enrolling or completing clinical trials; the risk that results obtained in NGM's clinical trials to date may not be indicative of results obtained in ongoing or future trials, including the risk that ongoing or future studies show that aldafermin is not a tolerable or effective treatment for NASH patients; seeking and maintaining protection of intellectual property; NGM's reliance on third party manufacturers and delays or problems in the manufacture of product candidates; and other risks and uncertainties affecting NGM and its development programs, including those described under the caption "Risk Factors" and elsewhere in NGM's quarterly report on Form 10-Q for the quarter ended September 30, 2019 and future filings and reports of NGM with the Securities and Exchange Commission. The forward-looking statements contained herein are made only as of the date hereof or as of the dates indicated in the forward-looking statements, even if they are subsequently made available by NGM on its website or otherwise. NGM undertakes no obligation to update or supplement any forward-looking statements after the date hereof, or to update the reasons why actual results may differ or 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; **Licensed by Merck**

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

Strategic collaboration with Merck – **up to $75M/yr. R&D support[1]** 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 12-18 months

[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.*
T2D: type 2 diabetes

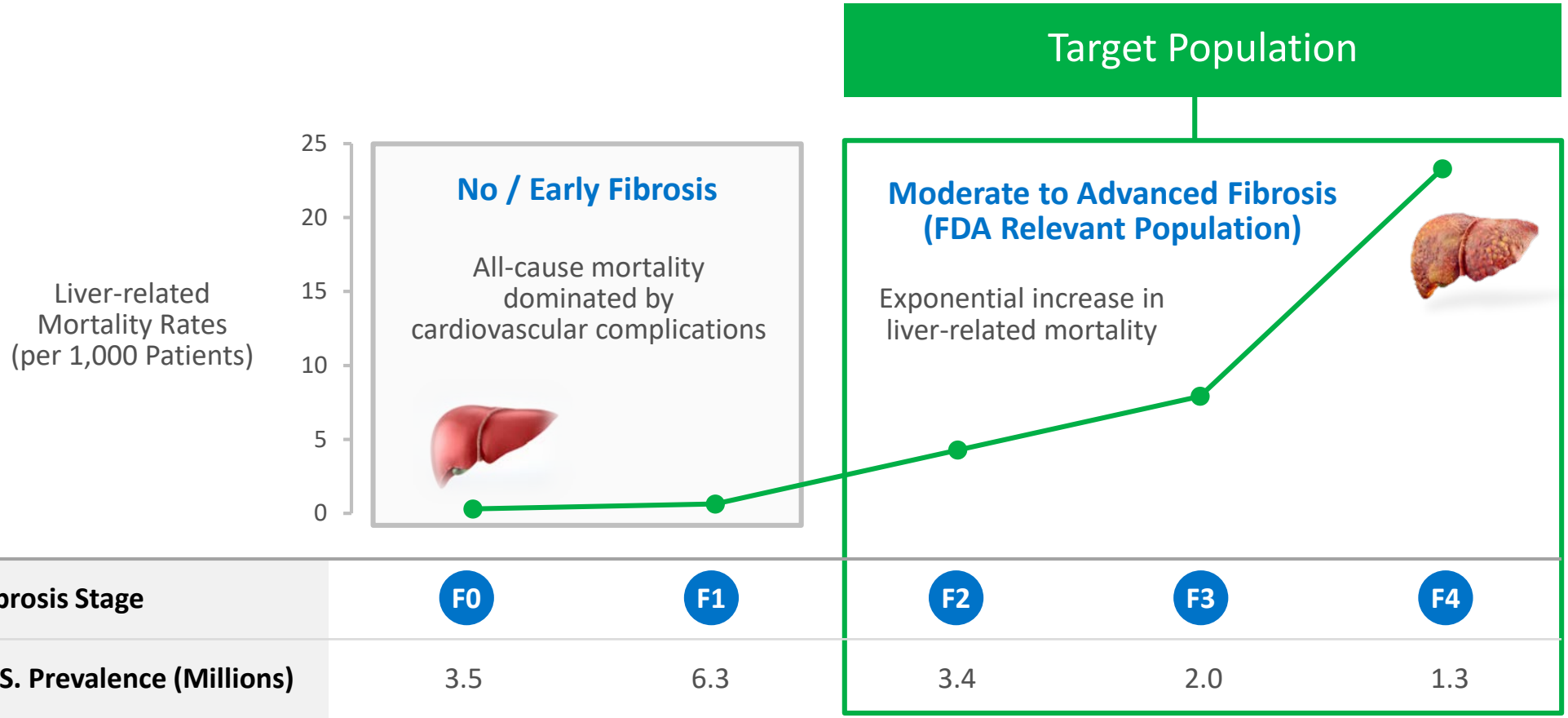
Our Expansive Pipeline

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, Cancer Anorexia/Cachexia Syndrome (CACS)	Phase 1a/1b	NGM Bio; MERCK: Option
NGM217	Undisclosed (Long Acting)	Diabetes	Phase 1	NGM Bio; MERCK: Option
NGM621	Complement C3 Inhibitory Antibody (Long Acting)	Dry AMD / Geographic Atrophy	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; GFRAL: glial-cell-derived neurotrophic factor receptor alpha-like; GDF15: growth differentiation factor 15

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				

Decompensated cirrhosis patients estimated to account for majority of direct US healthcare cost burden of NASH[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.

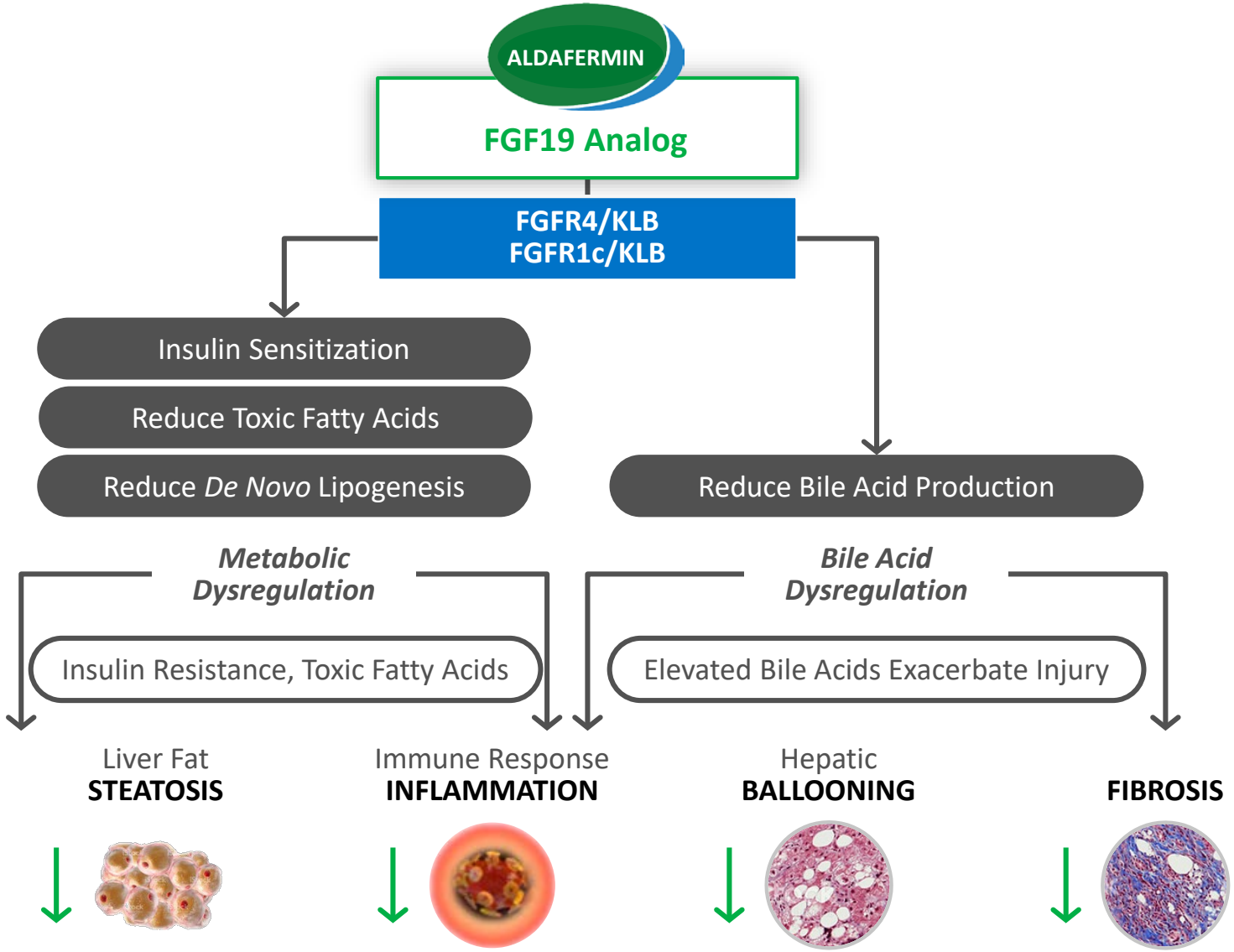
[1] H Razavi, Paris NASH meeting July 5, 2018 presentation "The value proposition of NASH therapy on the burden of disease related to obesity"

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

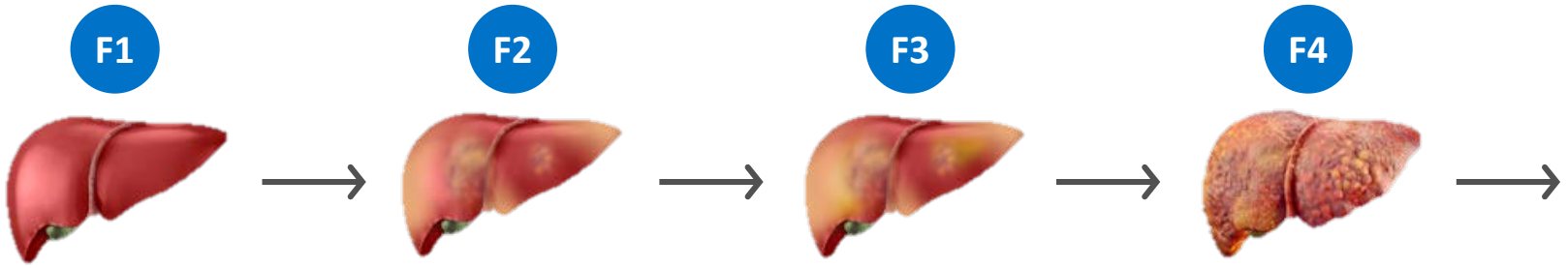
Competitive Landscape

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)	**Preliminary Topline Results** (More details planned for EASL 2020)
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 (53)

HISTOLOGY (Biopsy)

NON-INVASIVE MEASURES

KEY ENDPOINTS/ Assessment Measures

STEATOSIS

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

INFLAMMATION

BALLOONING

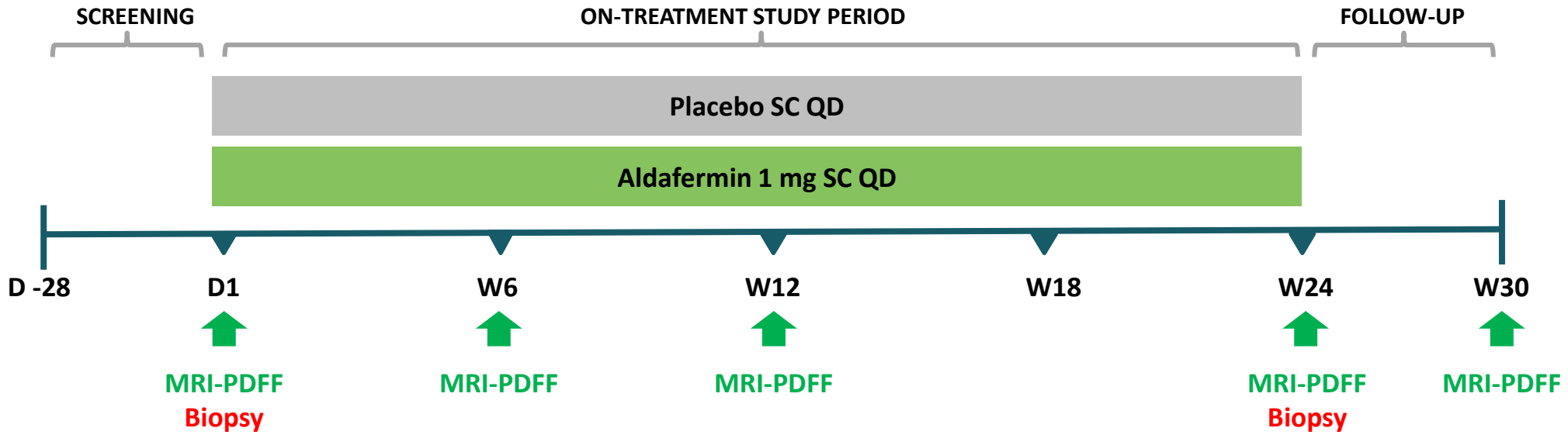
ALT/AST (Biomarkers)

FIBROSIS

ELF/PRO-C3 (Biomarkers)

MRI-PDFF: magnetic resonance imaging-estimated proton density fat fraction; ALT: alanine aminotransferase; AST: aspartate aminotransferase; ELF: Enhanced Liver Fibrosis score; PRO-C3: exploratory biomarker of fibrogenesis

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



Safety population	N=78	Aldafermin : placebo (2:1)
Efficacy population	N=77	At least one dose and valid post-dose efficacy value
Liver histology population	N=72	Valid, non-missing biopsy at baseline and W24

- 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) at W24
- **Secondary and exploratory endpoints** include ALT, AST, biomarkers of fibrosis and effect on liver histology at W24
- Over-encapsulated rosuvastatin (ROS 20 mg) started at W2 if low-density lipoprotein cholesterol (LDL-C) rise of 10 mg/dL observed

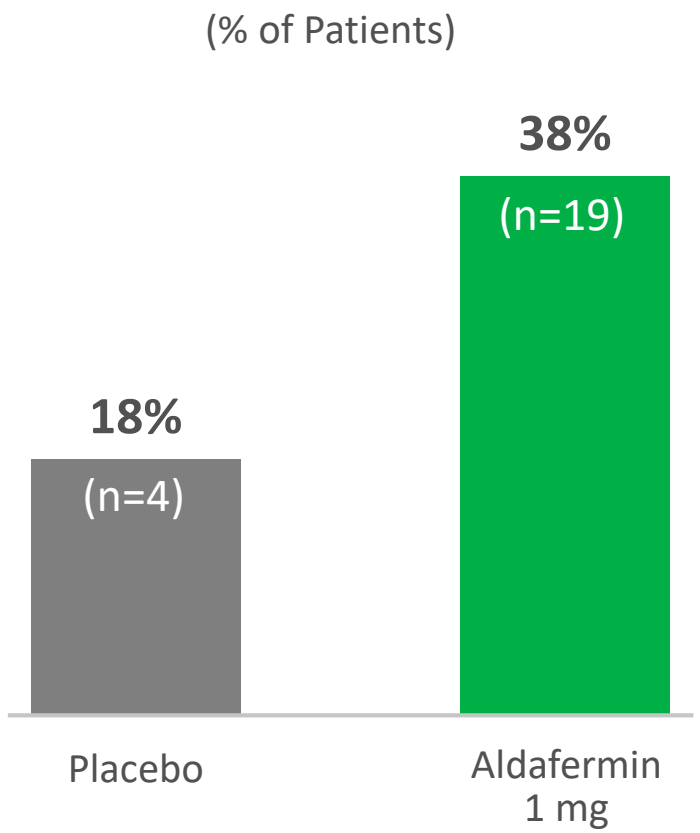
Patient Baseline Demographics and Characteristics (Cohort 4 Efficacy Population)

Parameters Mean (SD)	Placebo (n=25)	Aldafermin 1 mg (n=52)
Age (years)	54.1 (9.7)	53.0 (12.1)
Sex (Male/Female)	9 / 16	27 / 25
Weight (kg)	102.5 (29.7)	100.1 (21.0)
BMI (kg/m^2)	36.8 (9.0)	35.8 (6.4)
Waist (cm)	114.3 (17.0)	111.9 (15.4)
Type 2 Diabetes, n (%)	16 (64%)	31 (60%)
NAFLD Activity Score (NAS)	5.4 (1.1)	5.7 (1.1)
Fibrosis stage (F2 / F3)[1]	13 / 9	27 / 23
Liver Fat Content (% by MRI-PDFF)	18.5 (6.8)	18.0 (5.9)
Alanine aminotransferase, ALT (IU/L)	55.1 (29.6)	73.3 (39.6)
Aspartate aminotransferase, AST (IU/L)	44.3 (23.7)	54.5 (27.4)
HDL-C (mg/dL)	34.5 (16.7)	31.7 (12.5)
LDL-C (mg/dL)	95.0 (31.6)	95.1 (31.0)
Triglycerides (mg/dL)	167.7 (119.2)	194.2 (164.3)
Pro-C3 (ng/mL)	17.1 (7.0)	17.5 (8.4)

[1] *Liver histology population (aldafermin n=50; placebo n=22)*

Cohort 4: Rapid and Sustained Improvement in Fibrosis

Fibrosis Improvement ≥1 Stage with No Worsening of NASH[1] at W24



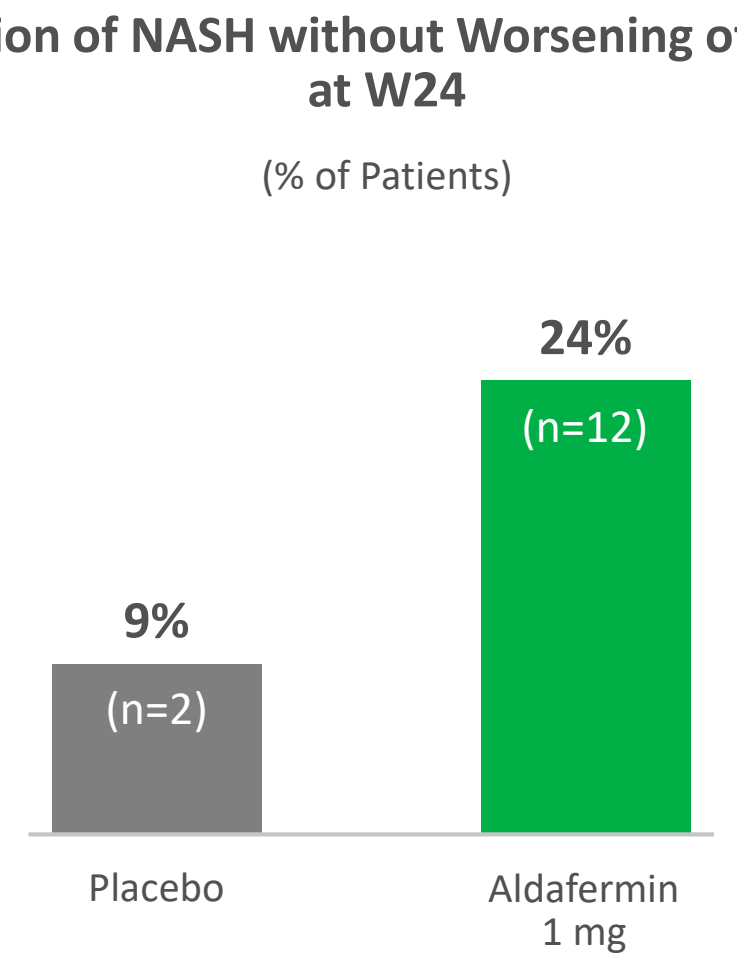
Biopsy Reads

- Both baseline and W24 liver biopsies were centrally read by the same NASH-CRN pathologist
- Baseline biopsies were not re-read at end of study
- All biopsies were read blinded to treatment assignment and patient

Liver Histology Population (n=50 aldafermin vs. n=22 placebo)

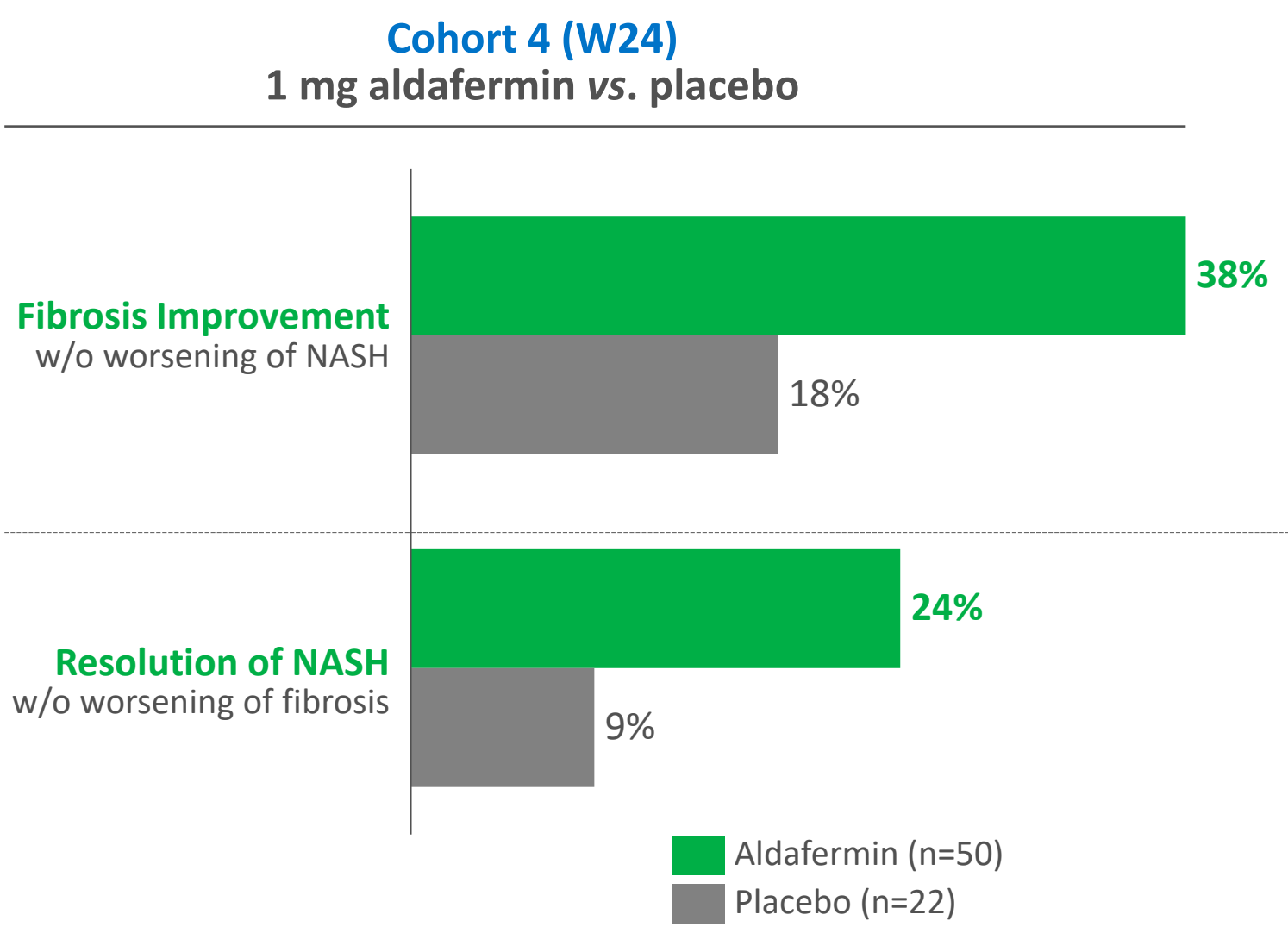
[1] Cohort 4 preliminary topline data; Defined as patients who have an improvement in liver fibrosis by ≥1 stage with no worsening of NASH (no worsening of steatosis, lobular inflammation or hepatocyte ballooning grade) from baseline to W24 (not powered for statistical significance)

Cohort 4: Additional Benefit in Resolution of NASH



Liver Histology Population (n=50 aldafermin vs. n=22 placebo)
[1] Cohort 4 preliminary topline data; Defined as subjects having a NAS score of 0 or 1 for inflammation and 0 for ballooning, with no worsening of fibrosis (no progression of NASH CRN fibrosis stage) from baseline to W24 (not powered for statistical significance)

Potential Amplification of Fibrosis Improvement and Resolution of NASH with Longer Treatment Duration



Cohort 3 (W12) 1 mg aldafermin	Cohort 2 (W12) 3 mg aldafermin
25%	42%
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13%	11%
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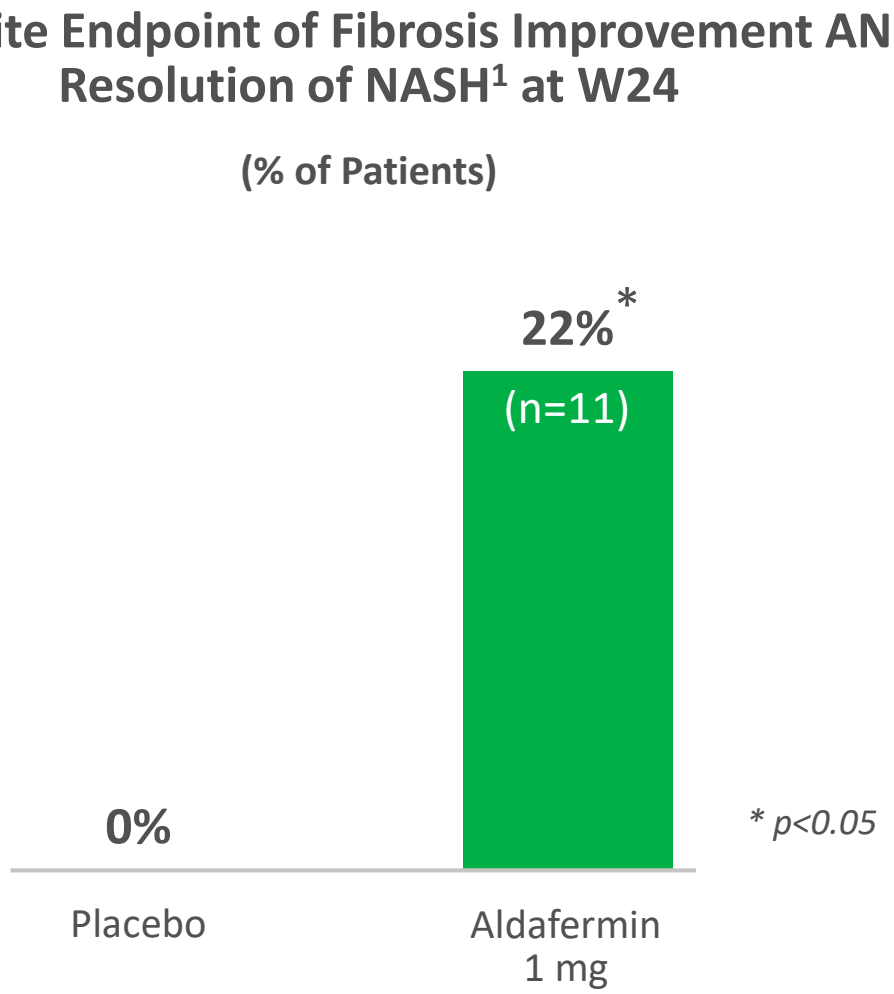
Cohorts 2-3 preliminary data; Cohort 4 preliminary topline data (endpoints not powered for statistical significance); % of patients achieving endpoint

Cohort 4: Statistically Significant Proportion of Patients Achieved Both Fibrosis Improvement AND Resolution of NASH

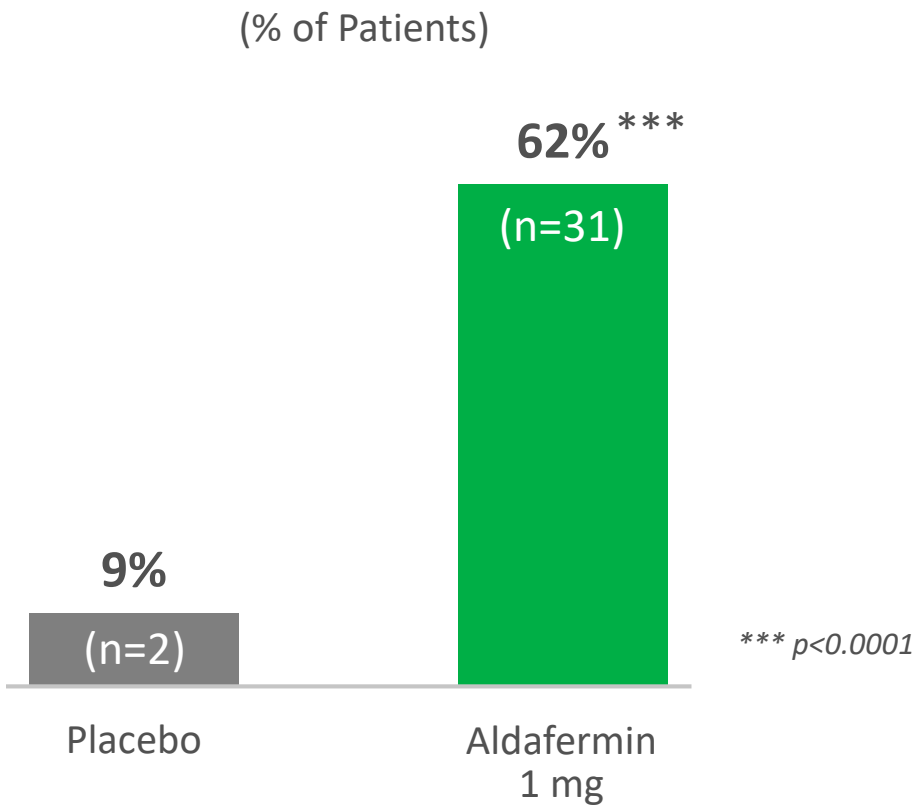


Liver Histology Population (n=50 aldafermin vs. n=22 placebo)

[1] Cohort 4 preliminary topline data; Defined as patients who have an improvement in liver fibrosis by ≥1 stage with no worsening in NASH AND have a NAS score of 0 or 1 for inflammation and 0 for ballooning without worsening of fibrosis at W24 (not powered for statistical significance)

Cohort 4: Statistically Significant Proportion of Patients Achieved NAS Reduction of ≥ 2 Points

Improvement of NAS by ≥ 2 Points without Worsening of Fibrosis[1] at W24

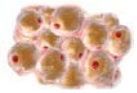


Statistically significant improvements in each NAS component of:

- Steatosis
- Lobular Inflammation
- Ballooning

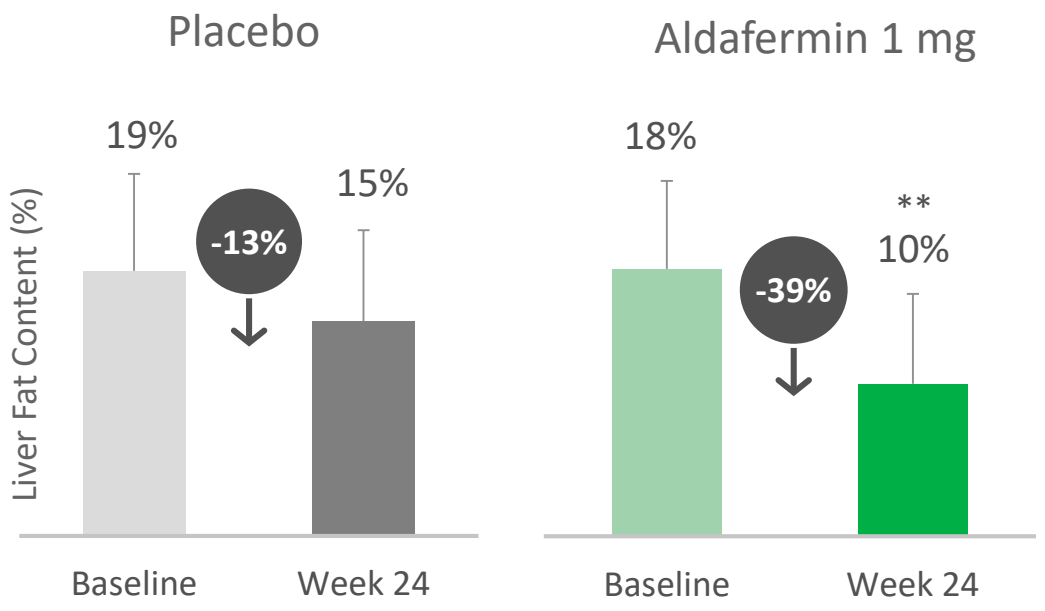
Liver Histology Population (n=50 aldafermin vs. n=22 placebo)
[1] Cohort 4 preliminary topline data; endpoint not powered for statistical significance

Cohort 4 Primary Endpoint Met: Statistically Significant Reduction in Absolute Liver Fat Content (LFC)



Cohort 4 (24W)

LFC by MRI-PDFF[1]



- 68% of aldafermin patients achieved ≥ 5% absolute LFC reduction *vs*. 24% placebo
- 66% of aldafermin patients achieved ≥ 30% relative LFC reduction *vs*. 29% placebo
- Consistent response on LFC across Cohorts 1-4

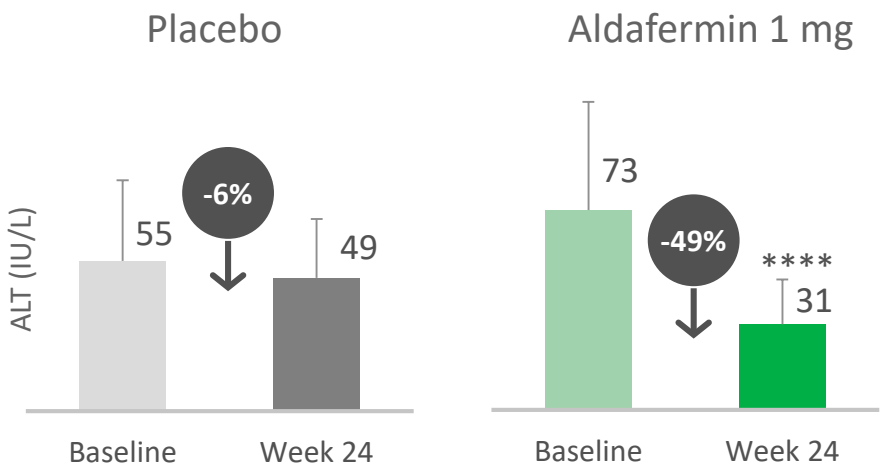
**P<0.01 *vs*. placebo

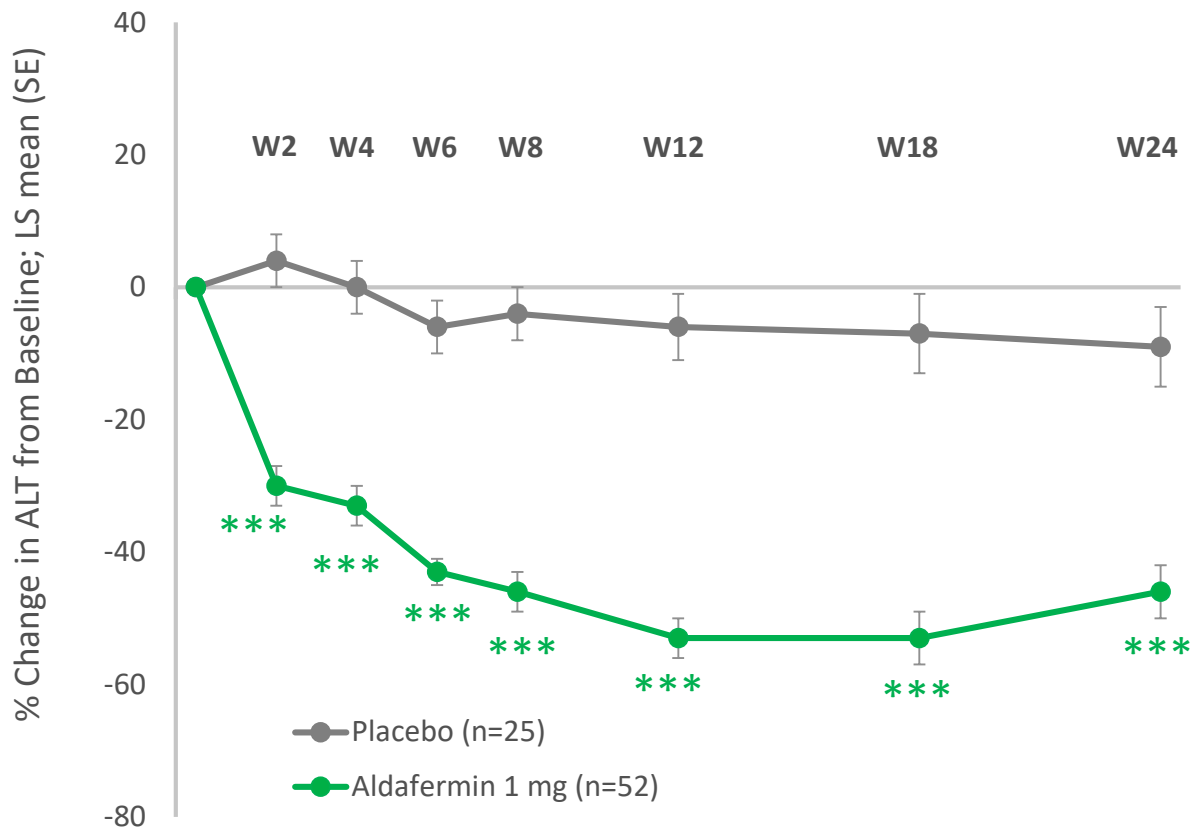
[1] *Cohort 4 preliminary topline data; Relative values are calculated as mean change from baseline*

Cohort 4: Rapid and Sustained Decrease in ALT to Near Normal Levels with Aldafermin

Cohort 4 (24W)

ALT (IU/L)[1]





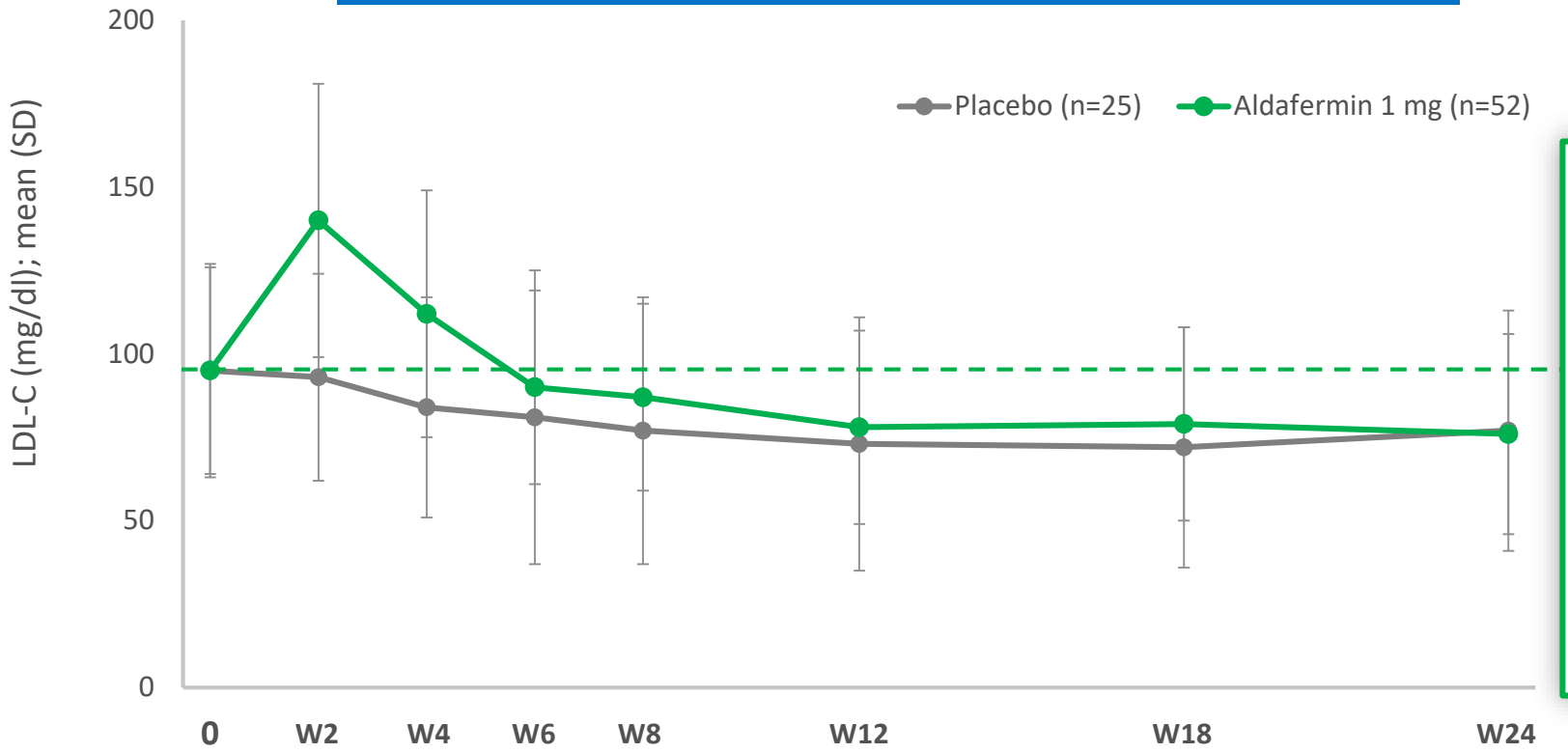
Statistically significant reductions *vs.* placebo also observed with AST and PRO-C3

****P<0.0001, ***P<0.001 *vs.* placebo

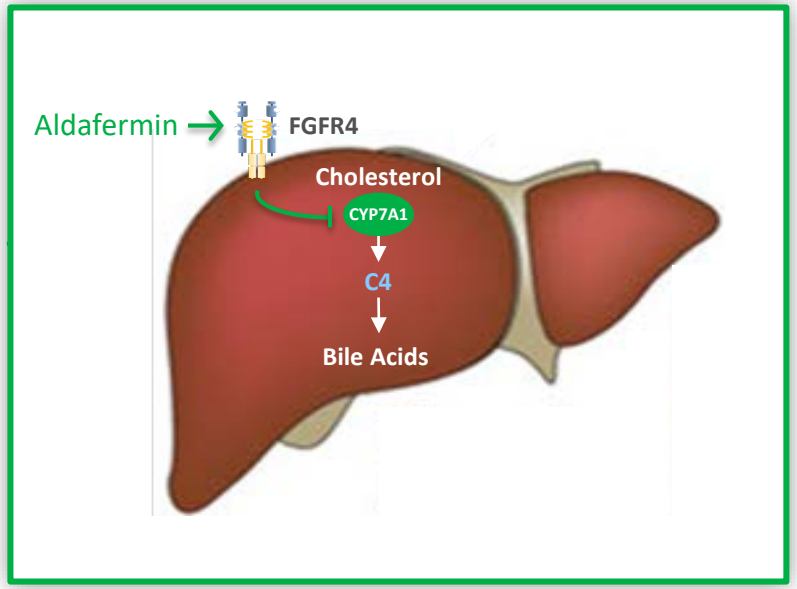
[1] *Cohort 4 preliminary topline data; Relative values are calculated as mean change from baseline*

Cohort 4:
LDL-C Changes Effectively Managed with Statin Therapy

Add Rosuvastatin to Placebo or Aldafermin as Needed (If Needed, Titration at W2, W4, W8)



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



- Mean LDL-C levels returned to ~77 mg/dL for both placebo and aldafermin arms
- Statistically significant reduction in triglycerides observed as early at W2 and sustained through W24

Cohort 4 preliminary topline data
C4 = 7α-hydroxyl-4-cholesten-3-one; CYP7A1: cholesterol 7 alpha-hydroxylase

Cohort 4: Aldafermin Generally Well Tolerated and Most Common Adverse Events Comparable to Placebo

TEAE Classification	Placebo (N=25)	Aldafermin 1 mg (N=53)
Any TEAE	22 (88.0%)	46 (86.8%)
TEAE Leading to Drug Withdrawal	1 (4.0%)	0
Serious Adverse Event (SAE)[1]	3 (12.0%)	2 (3.8%)
Drug-Related TEAE	11 (44.0%)	27 (50.9%)
TEAE Leading to Death	0	0

Most Common (>10%) Adverse Events	Placebo (N=25)	Aldafermin 1 mg (N=53)
Diarrhea	6 (24.0%)	15 (28.3%)
Headache	9 (36.0%)	7 (13.2%)
Abdominal Distension	3 (12.0%)	7 (13.2%)
Nausea	6 (24.0%)	5 (9.4.%)
Fatigue	4 (16%)	3 (5.7%)
Diabetes Mellitus	5 (20.0%)	2 (3.8%)
Peripheral Edema	3 (12.0%)	2 (3.8%)

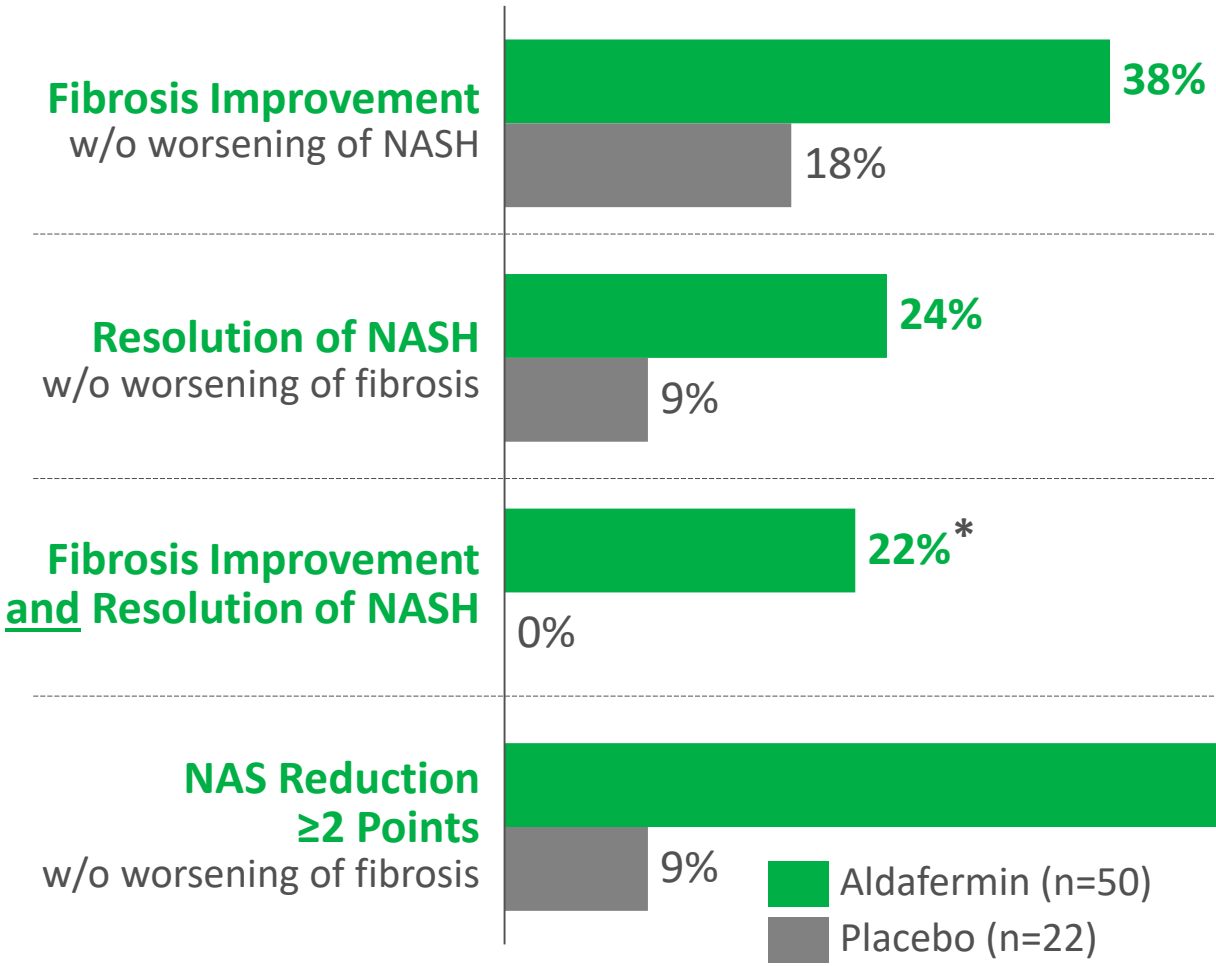
- All SAEs were deemed to be not related to treatment by site investigator

- Pruritus (4% aldafermin *vs*. 8% placebo)
- Injection site bruising (6% aldafermin *vs*. 0% placebo)

Cohort 4 preliminary topline data

1 SAEs: Placebo (mental status changes; appendicitis; anxiety); Aldafermin (rectal bleeding; post-biopsy bleeding)

Phase 2 Data Supports Aldafermin's Potential as Differentiated Monotherapy for Treatment of NASH with Established Fibrosis



Cohort 3 (W12) 1 mg aldafermin	Cohort 2 (W12) 3 mg aldafermin
25%	42%
--	--
13%	11%
--	--

*** p<0.0001
* p<0.05

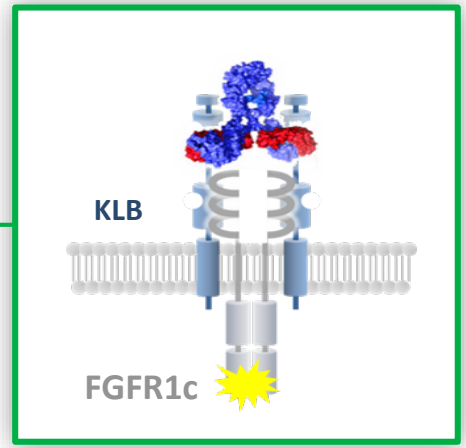
Cohorts 2-3 preliminary data; Cohort 4 preliminary topline results; % of patients achieving endpoint

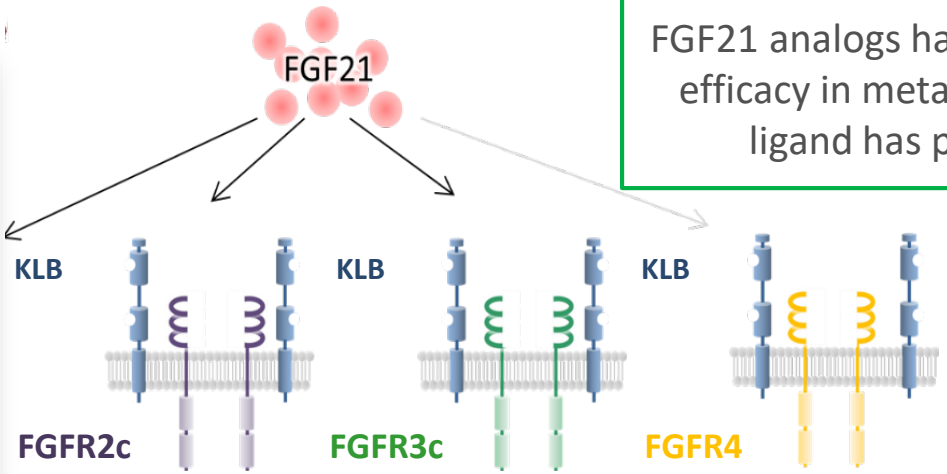
Summary of Aldafermin Cohort 4 Preliminary Topline Results

- **Meaningful improvements in histology regulatory endpoints**: fibrosis improvement, resolution of NASH and the composite endpoint requiring achievement of both
- Aldafermin's differentiated **rapid dual anti-fibrotic and metabolic effect** is evidenced by the significant improvements observed as early as two weeks
- Cohort 4 data suggest that the histological effects we observed at 12 weeks are **sustained and potentially amplified with extended treatment**
- **Favorable tolerability profile**: most common adverse events occurred with similar frequency in placebo and aldafermin arms

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

NGM313



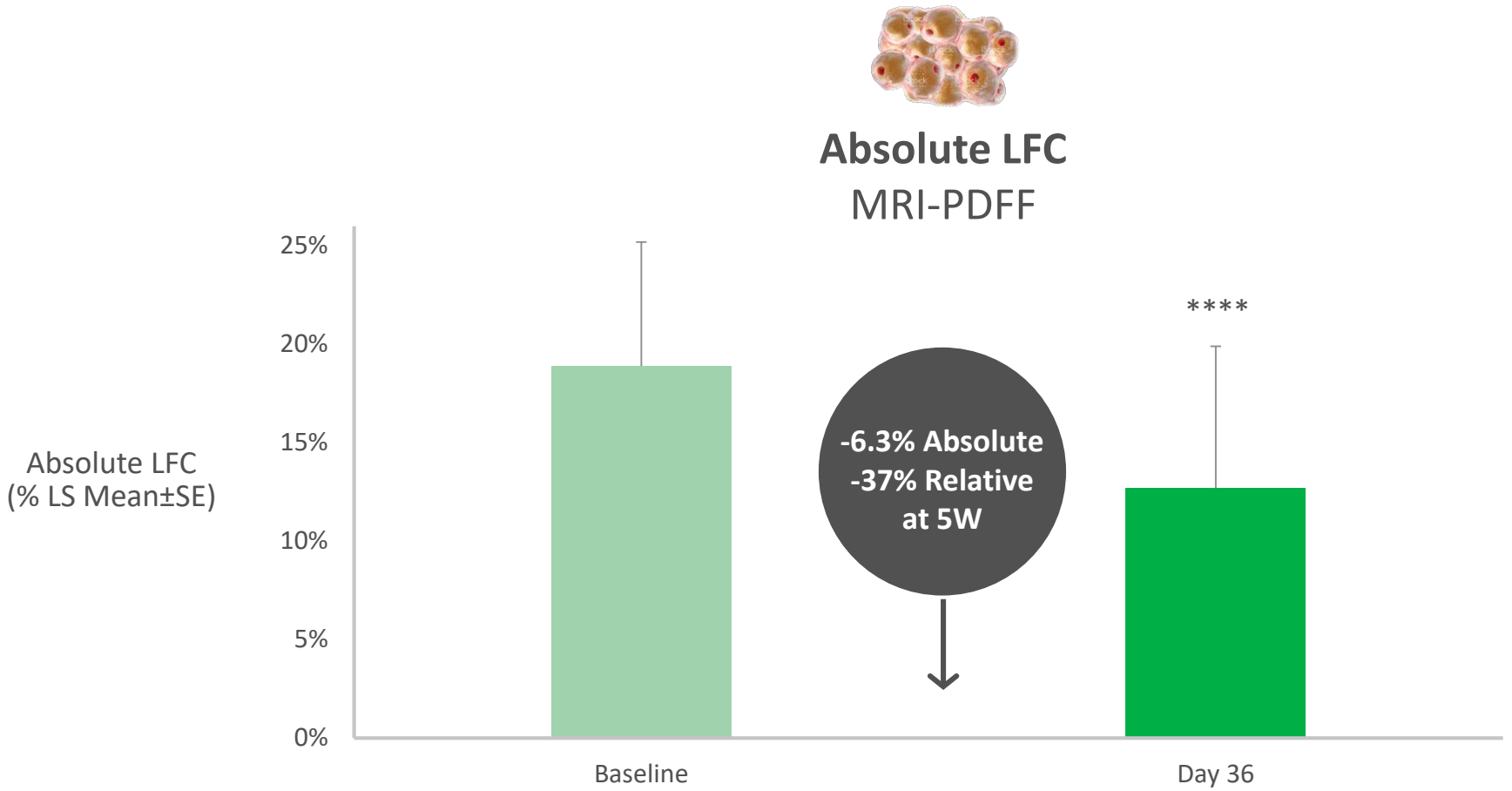


FGF21 analogs have demonstrated variable clinical efficacy in metabolic syndrome patients; native ligand has potential for safety liabilities

- 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



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

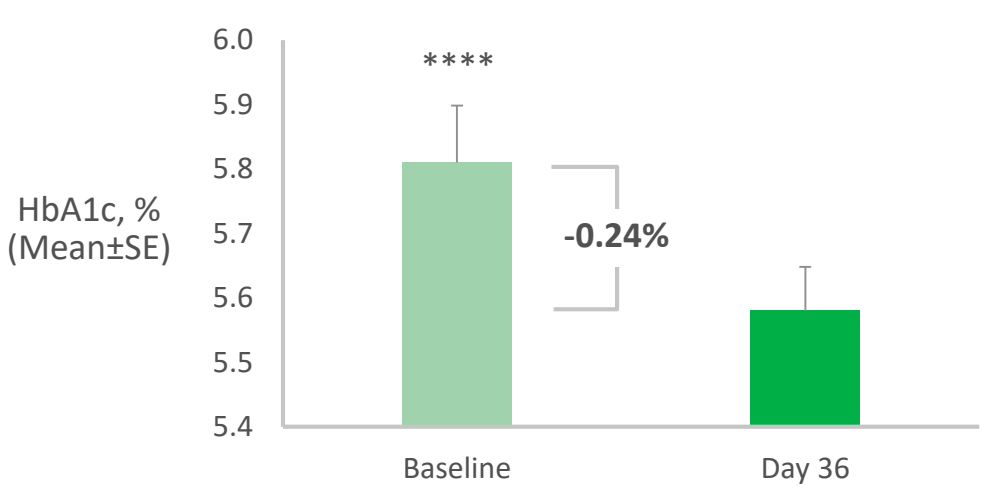
*****p<0.0001*

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

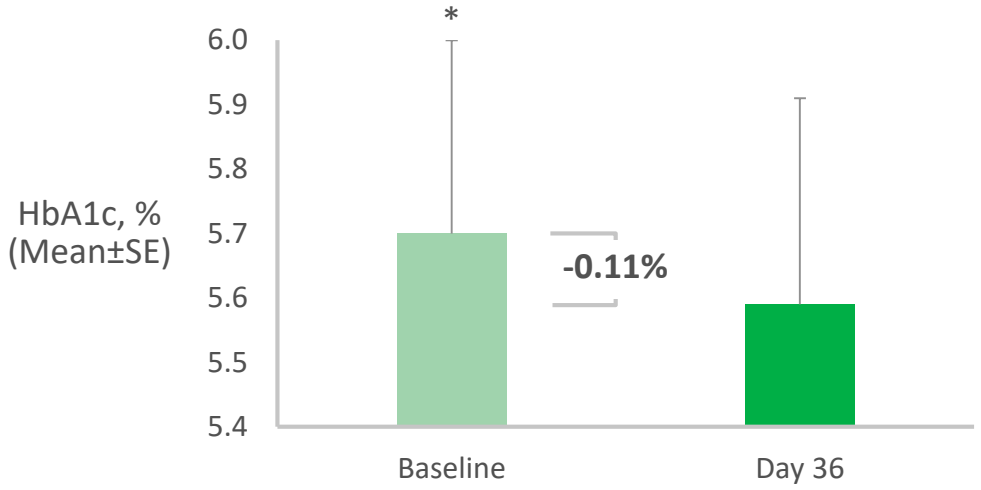
Phase 1b Study in Obese, Insulin Resistant Subjects with NAFLD

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

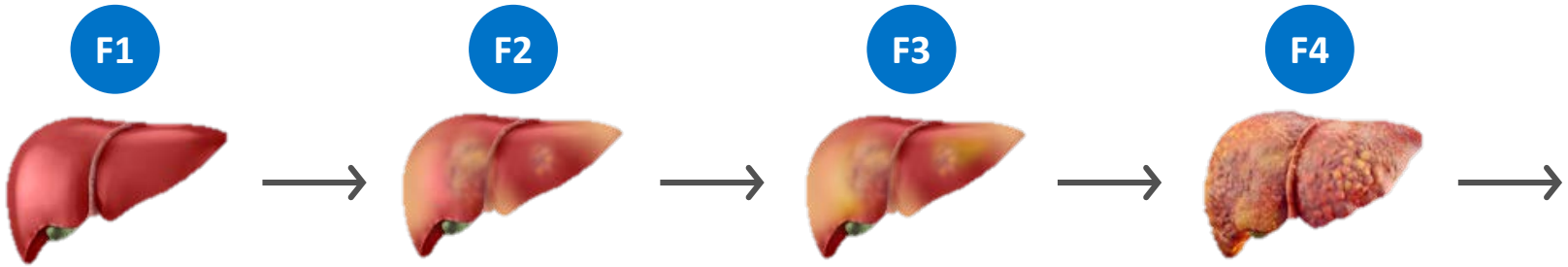
Competitive Landscape

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

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6.3M	3.4M	2.0M	1.3M

Target Patient Populations

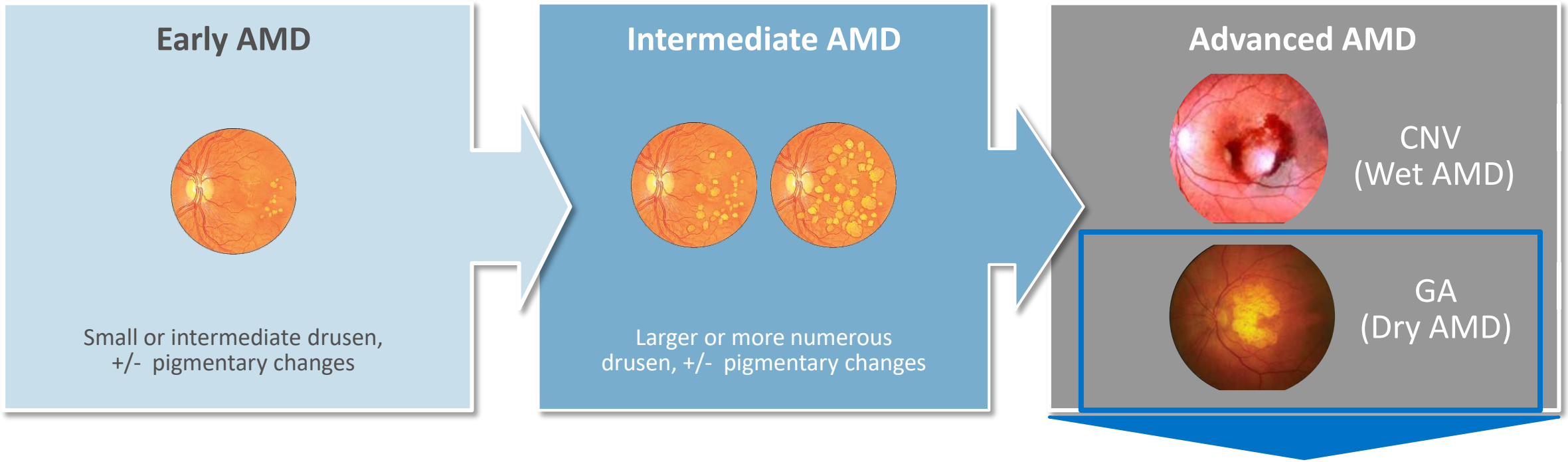
Complementary Treatments for NASH

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	
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, Cancer Anorexia/Cachexia Syndrome (CACS)	Phase 1a/1b	NGM Bio	MERCK Option
NGM217	Undisclosed (Long Acting)	Diabetes	Phase 1	NGM Bio	MERCK Option
NGM621	Complement C3 Inhibitory Antibody (Long Acting)	Dry AMD / Geographic Atrophy	Phase 1	NGM Bio	MERCK Option
NGM395	GDF15 Analog (Long Acting)	Metabolic	Preclinical	NGM Bio	Wholly-Owned

Geographic Atrophy (GA) is an Advanced Form of AMD

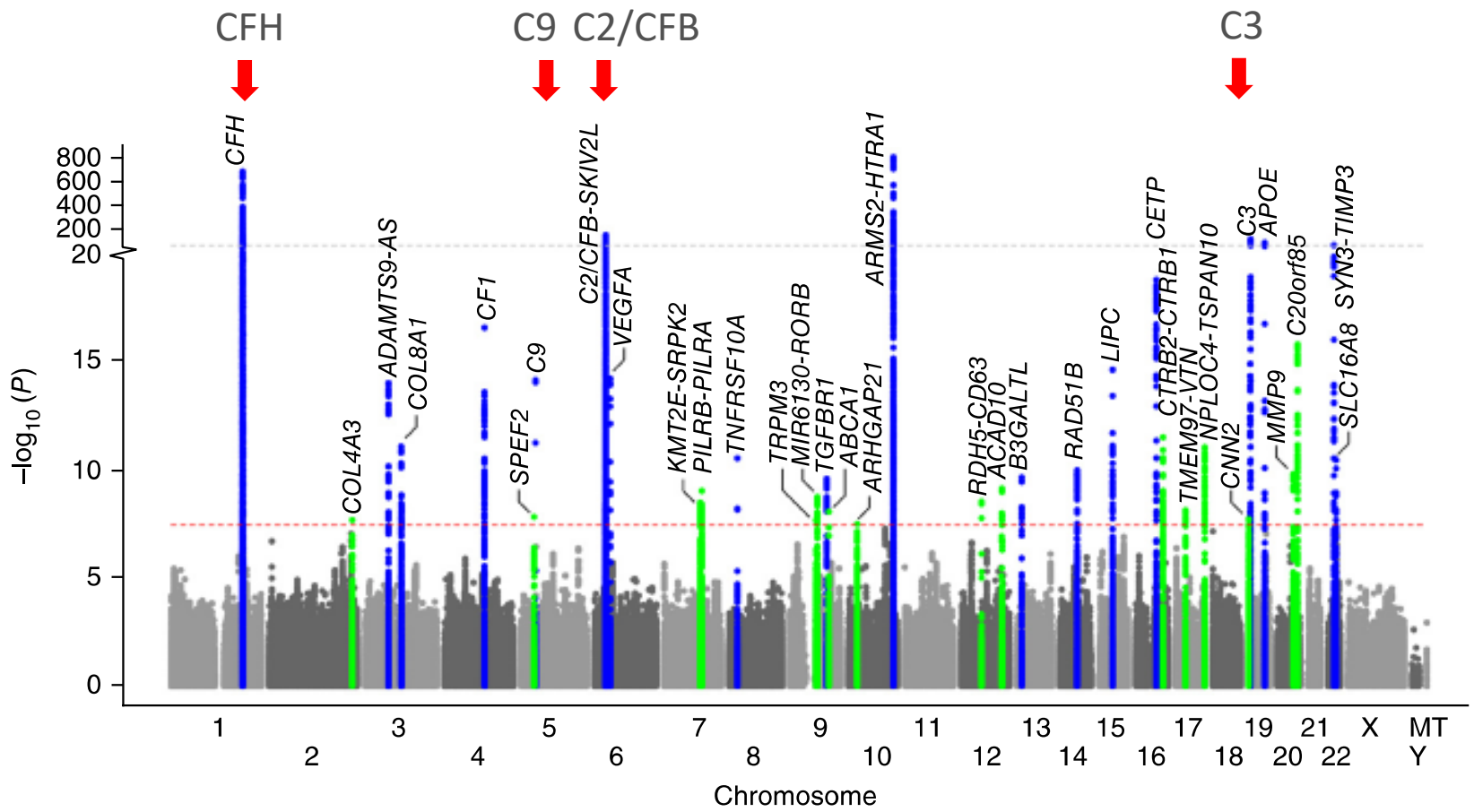


- Geographic atrophy (GA) is the dry form of advanced AMD, characterized by progressive and irreversible loss of photoreceptors, retinal pigment epithelium (RPE) and choriocapillaris
- GA is typically bilateral and lesion enlargement results into irreversible blindness
- GA affects ~5 million people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

CNV = choroidal neovascularization

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

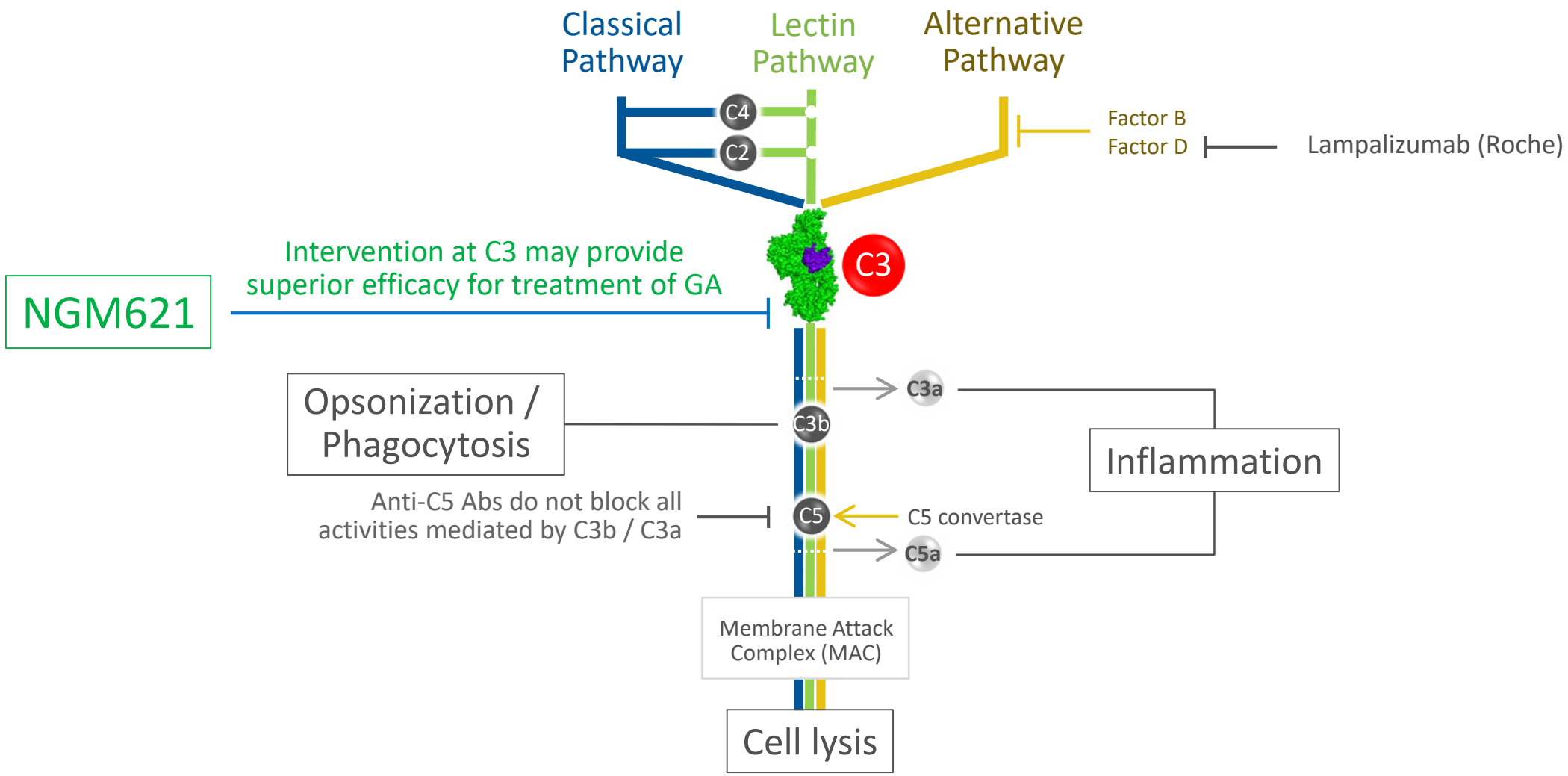
Complement Activation is Associated with Development of Advanced AMD



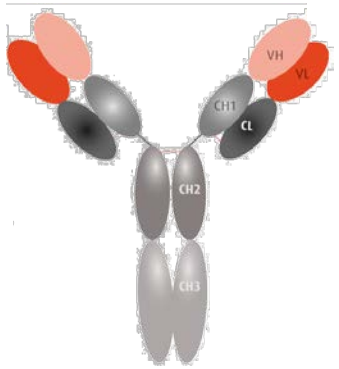
Variants in the complement pathway account for the majority of the known genetic risk for AMD

Fritsche et al. Nat Genet 2016

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

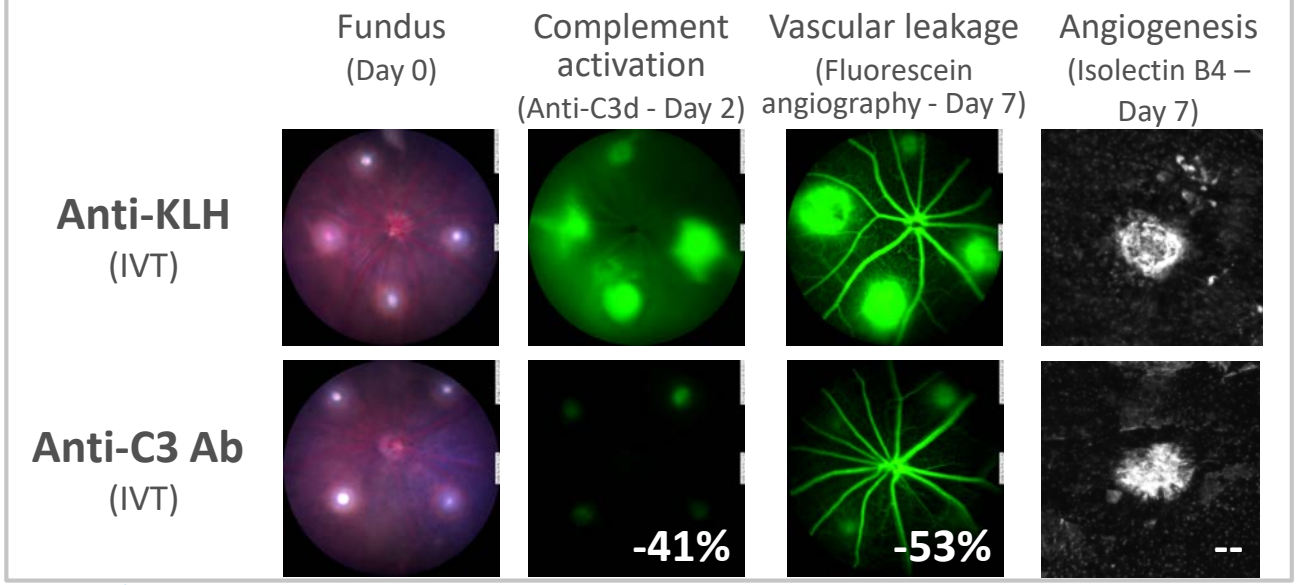


NGM621: A Potent Anti-Complement C3 Antibody

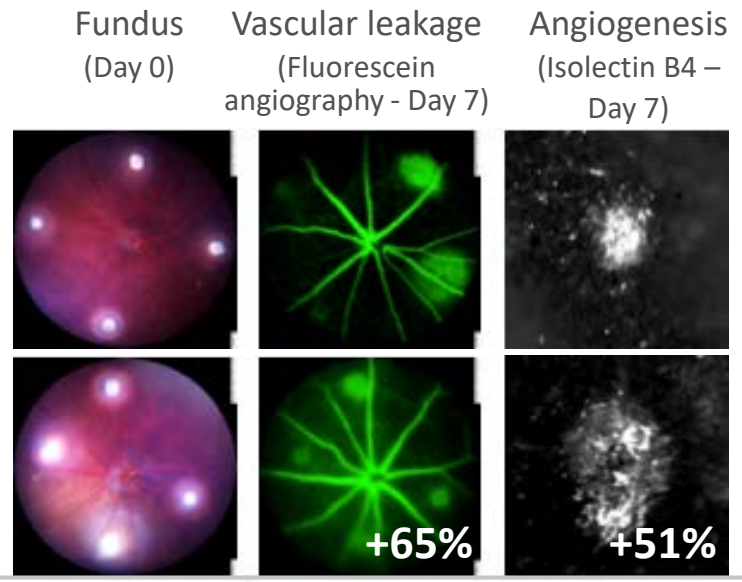


- Antibody that has high binding affinity for human C3 (K_D < 1 nM)
- Potent inhibition of both classical and alternative pathways of complement activation (IC_{50} ~ 5-6 nM)
- Potential for QM (monthly) or Q2M (EOM) intravitreal dosing

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



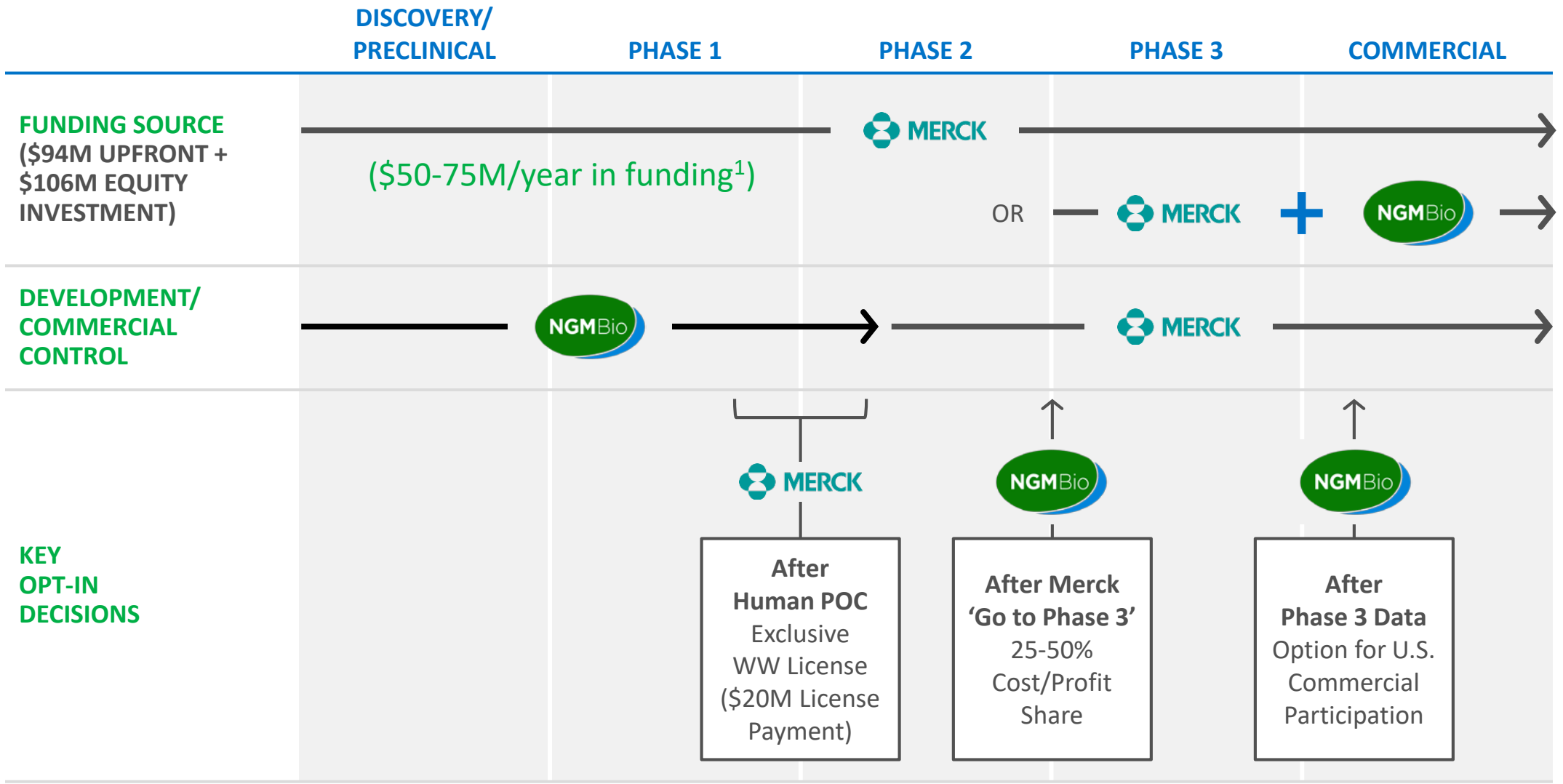
Antibody is preferred modality; PEG-40kD exacerbates laser injury-induced CNV in mice



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.*

3Q19 Financial Results[1]

STATEMENT OF OPERATIONS (In thousands, unaudited)	THREE MONTHS ENDED SEP 30, 2019	NINE MONTHS ENDED SEP 30, 2019
RELATED PARTY REVENUE	$21,568	$72,461
RESEARCH AND DEVELOPMENT EXPENSES	$28,953	$87,299
GENERAL AND ADMINISTRATIVE EXPENSES	$5,612	$17,208
TOTAL OPERATING EXPENSES	$34,565	$104,507
LOSS FROM OPERATIONS	**($10,917)**	**($26,854)**

BALANCE SHEET	SEP 30, 2019 (unaudited)	DEC 31, 2018 (audited)
CASH, CASH EQUIVALENTS AND SHORT-TERM MARKETABLE SECURITIES	**$356.6M**	**$206.6M**

[1] *See the Company's Quarterly Report on Form 10-Q for the quarter ended September 30, 2019 for more complete financial information*

Multiple Potential Value-Driving Catalysts in 2020

Product Candidate	Potential Indications	Targeted 2020 Milestones	Targeted Timing
ALDAFERMIN	NASH F2/F3	Phase 2 Cohort 4 biopsy data	1Q20 ☑
ALDAFERMIN	NASH F4	ALPINE 4 FPI	1H20
ALDAFERMIN	NASH F2/F3	ALPINE 2/3 topline data	1H21
NGM313 (MK-3655)	NASH F2/F3	Phase 2b FPI (Merck)	2H20
NGM120	Cancer/CACS	Phase 1a/1b FPI	1Q20 ☑
NGM217	Diabetes	Phase 1b/2a FPI	2H20
NGM621	Dry AMD/GA	Phase 1 safety & tolerability data	2H20
NGM621	Dry AMD/GA	Phase 2 FPI	2H20
NGM395	Metabolic	Phase 1 FPI	1H20

FPI = first patient in; GA = geographic atrophy

NASDAQ: NGM

Novel Biology.
Powerful Medicines.
Transformative Impact.