Novel Biology. Powerful Medicines. Transformative Impact.

NGM Biopharmaceuticals, Inc.

ALDAFERMIN PHASE 2 COHORT 4 TOPLINE RESULTS

NASDAQ: NGM

Safe Harbor Statement

This presentation contains forward-looking statements, including, but not limited to, statements related to the safety, tolerability and potential efficacy of aldafermin, including its potential to reverse liver disease and act as a monotherapy; the therapeutic potential, effect and differentiation of aldafermin; potential benefits of extended treatment with aldafermin and its potential role in the future treatment landscape for NASH; NGM's expectations as to the endpoints that would potentially be supportive of accelerated approval; NGM furthering its aldafermin development program and advancing aldafermin into pivotal studies; the design, initiation, enrollment and availability of data for NGM's clinical trials and the timing thereof; NGM's expectation of potential value-driving catalysts in 2020 and the timing thereof; opportunities for enhancing shareholder value 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 in this presentation. These risks and uncertainties include, without limitation, risks and uncertainties associated with the costly and time-consuming pharmaceutical product development process and the uncertainty of clinical success, including risks related to failure or delays in successfully enrolling or completing clinical studies, the risk that the results obtained to date in NGM's clinical trials may not be indicative of results obtained in pivotal or other late-stage trials, and the risk that NGM's ongoing or future clinical studies in humans may show that aldafermin is not a tolerable and effective treatment for NASH patients; the time-consuming and uncertain regulatory approval process; NGM's reliance on third-party manufacturers for aldafermin; the sufficiency of NGM's cash resources and need for additional capital; and other risks and uncertainties affecting NGM and its development programs, including those described under the caption "Risk Factors" in NGM's quarterly report on Form 10-Q for the quarter ended September 30, 2019 and future filings and reports that NGM makes from time to time with the United States Securities and Exchange Commission. The forward-looking statements contained in this presentation 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 of this presentation, or to update the reasons why actual results may differ or differ materially from those anticipated in the forward-looking statements.

Summary of Aldafermin (NGM282)
Cohort 4 Preliminary Topline Results

24 Week Study of 1 mg Aldafermin *vs*. Placebo

- **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

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

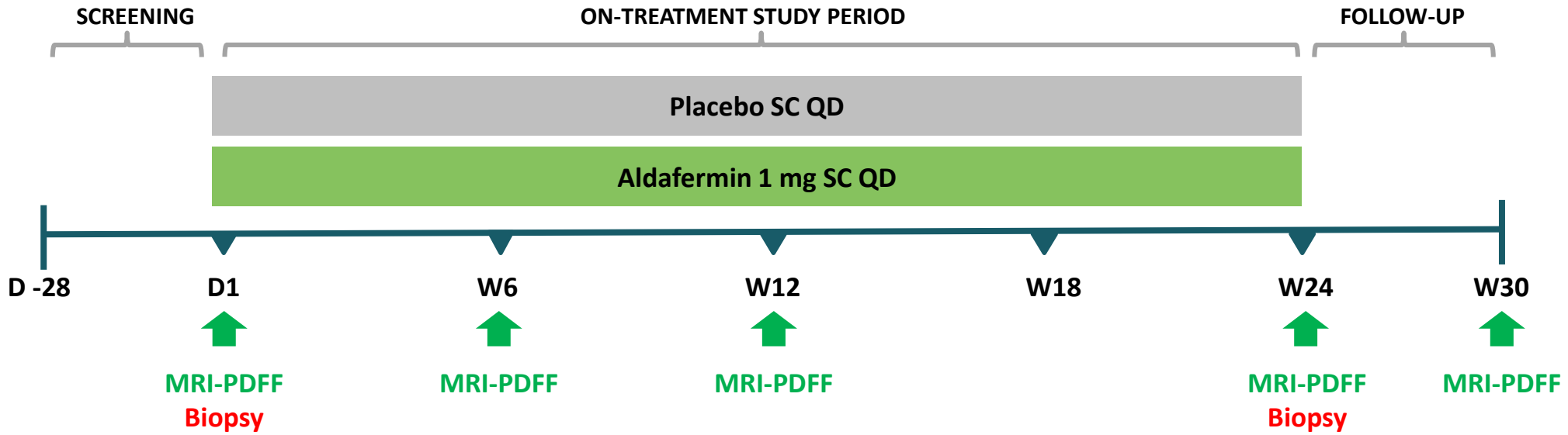
ALT/AST (Biomarkers)

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

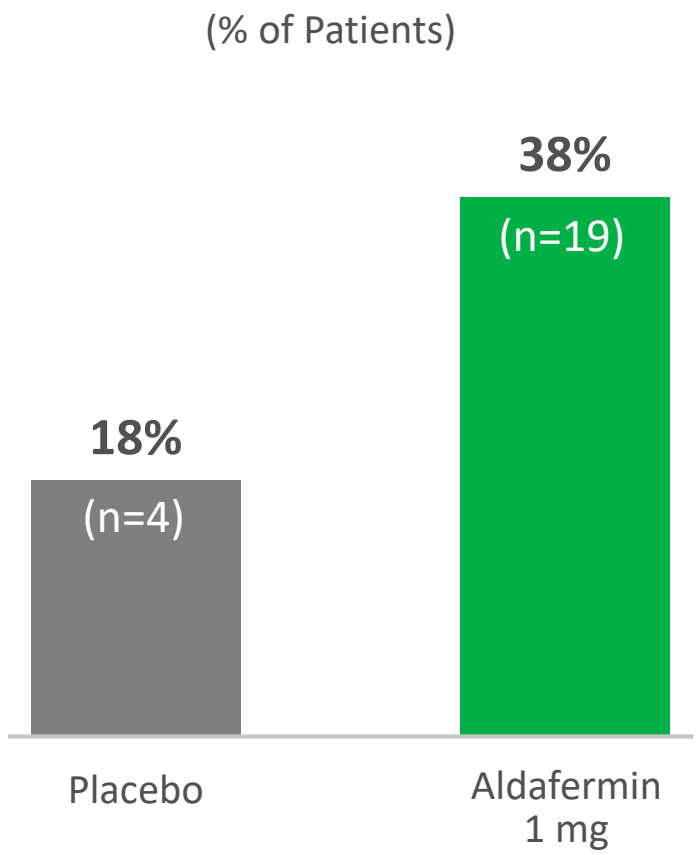
Patient Baseline Demographics and Characteristics (Cohort 4 Efficacy Population)

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)*

Rapid and Sustained Improvement in Fibrosis

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



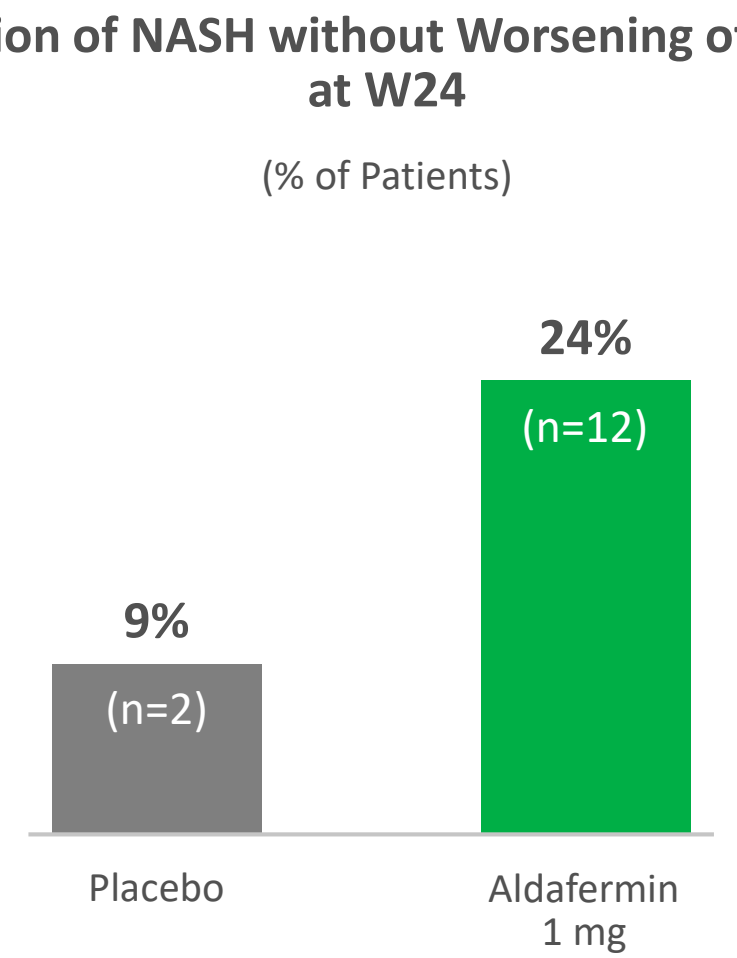
Biopsy Reads

- 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)

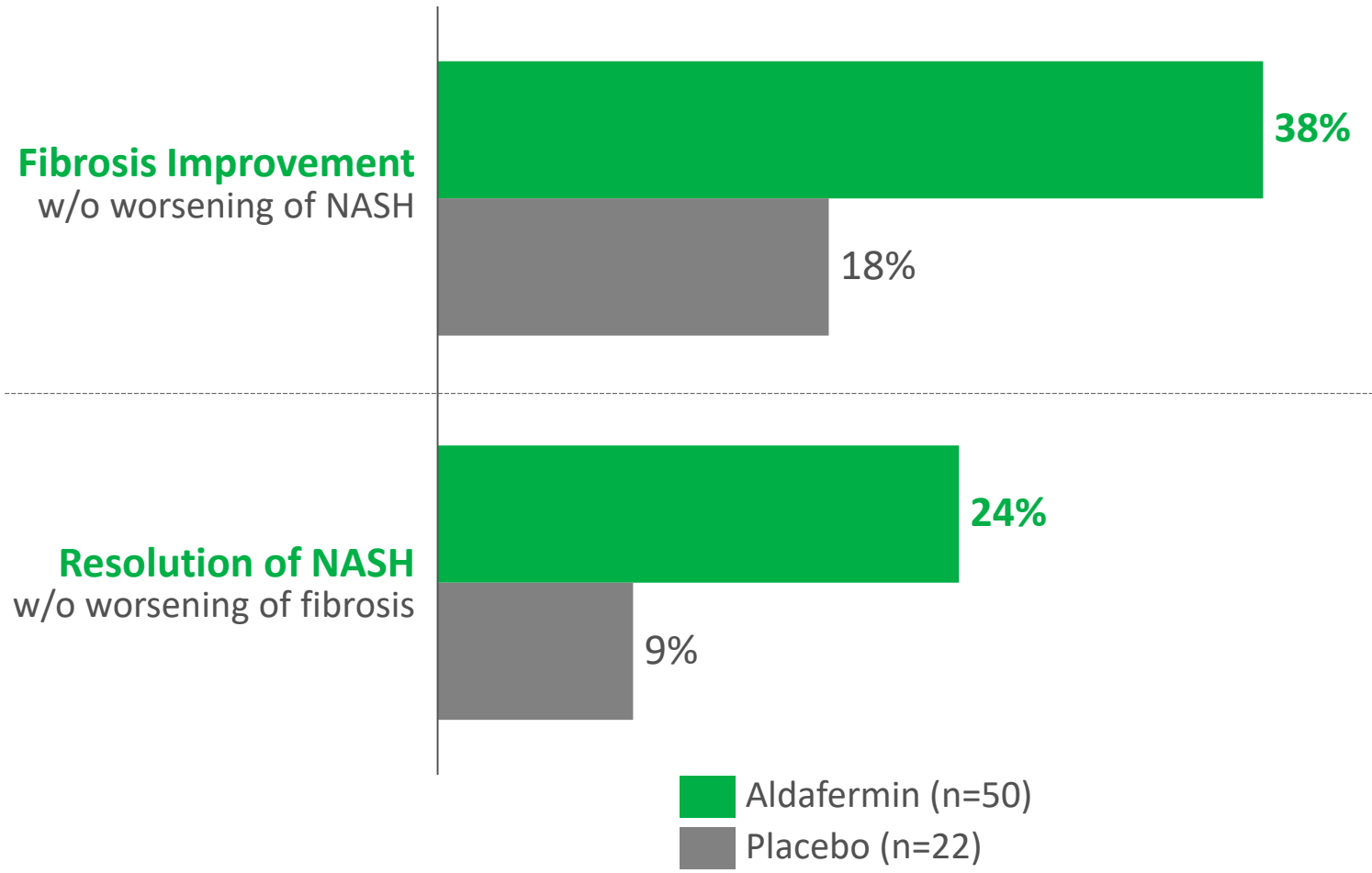
Additional Benefit in Resolution of NASH



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)

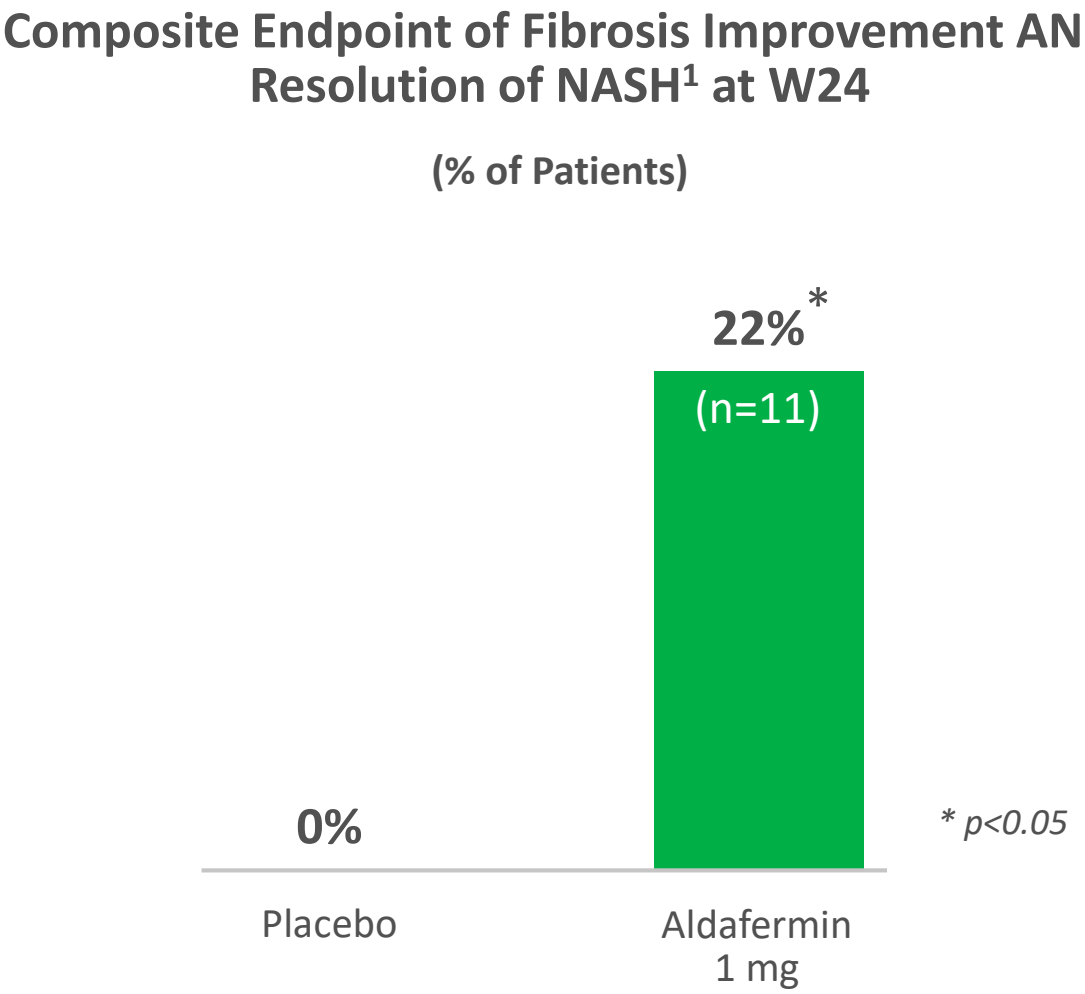
Potential Amplification of Fibrosis Improvement and Resolution of NASH with Longer Treatment Duration



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

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

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

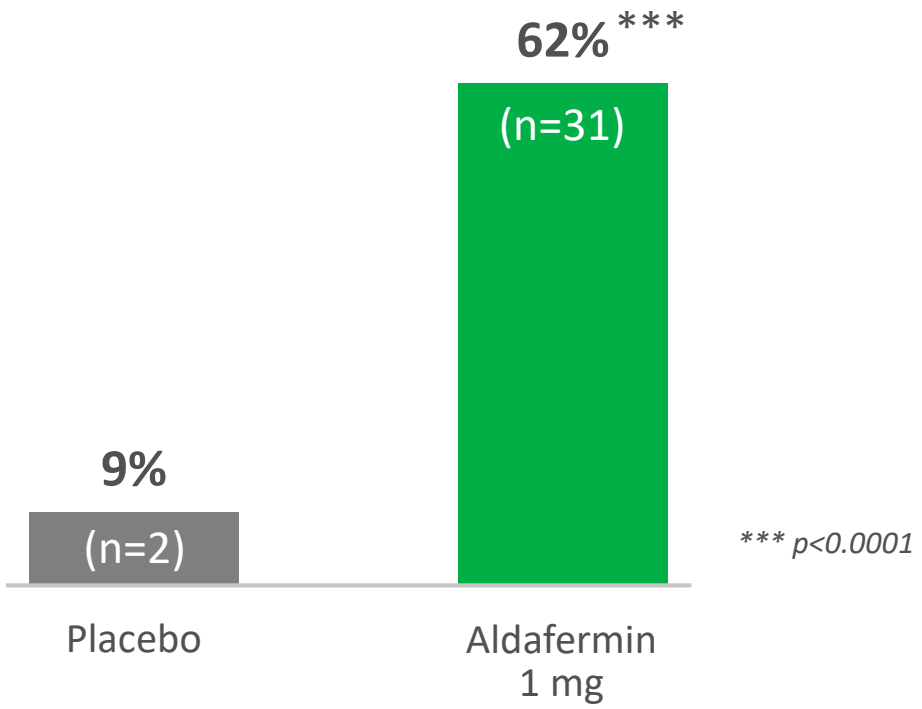


Liver Histology Population (n=50 aldafermin vs. n=22 placebo)

[1] Cohort 4 preliminary topline data; Defined as patients who have an improvement in liver fibrosis by ≥1 stage AND have a NAS score of 0 or 1 for inflammation and 0 for ballooning at W24 (not powered for statistical significance)

Statistically Significant Proportion of Patients Achieved NAS Reduction of ≥ 2 Points

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

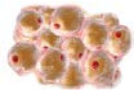


Statistically significant improvements in each NAS component of:

- Steatosis
- Lobular Inflammation
- Ballooning

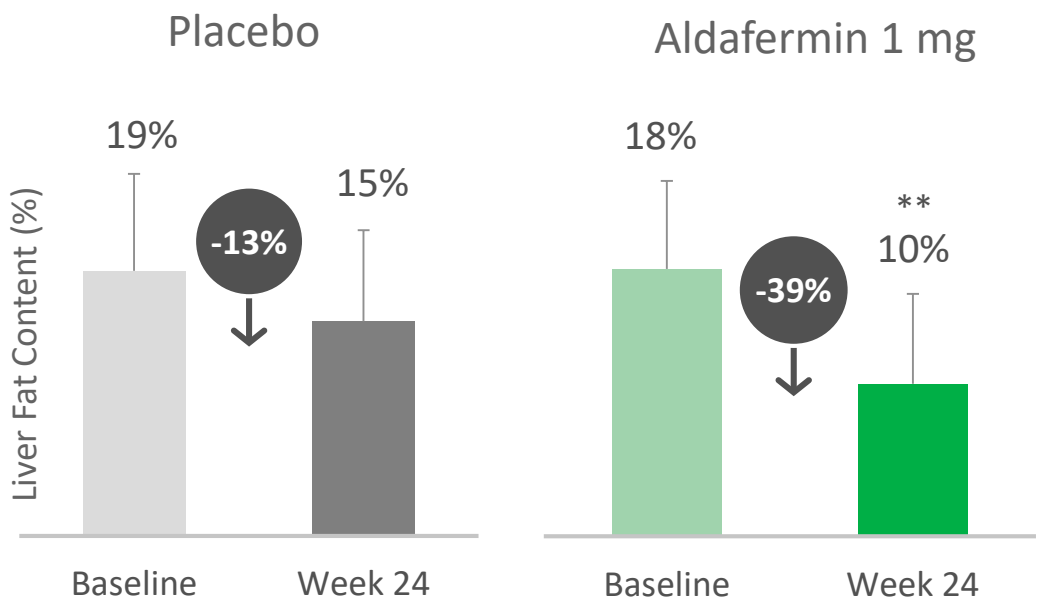
Liver Histology Population (n=50 aldafermin vs. n=22 placebo)
[1] Cohort 4 preliminary topline data; endpoint not powered for statistical significance

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

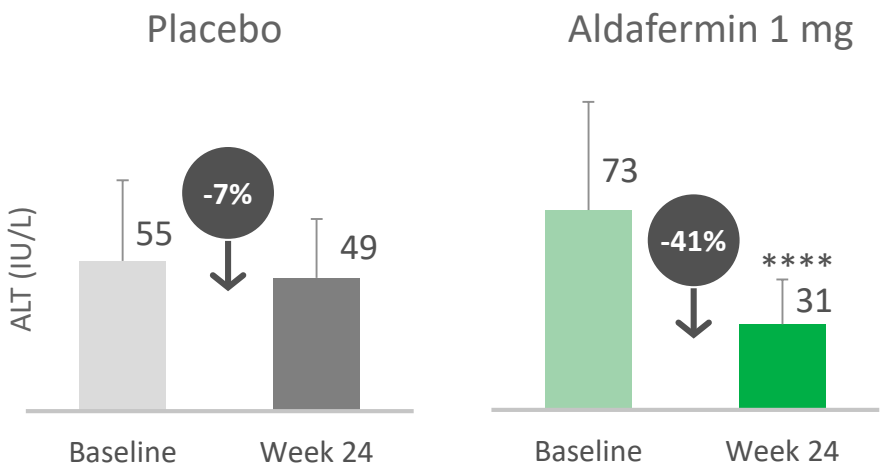
**P<0.01 *vs*. placebo

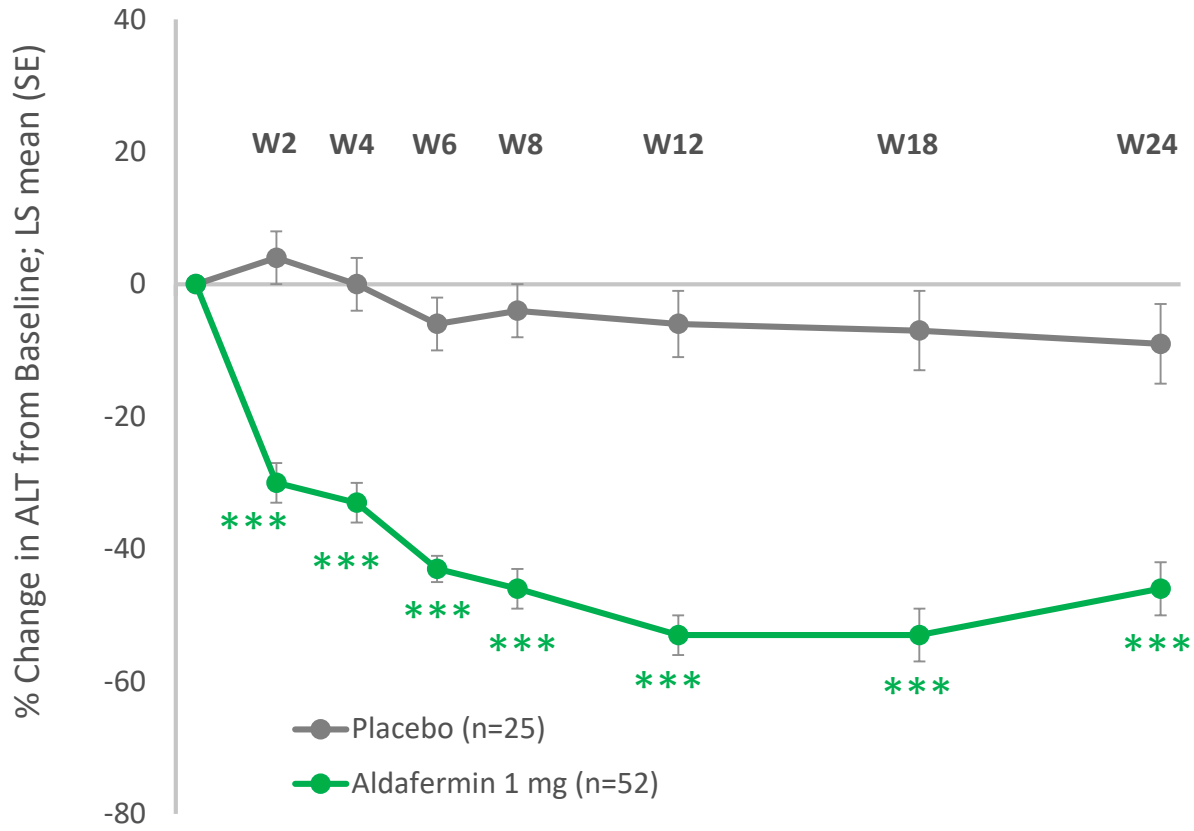
[1] *Cohort 4 preliminary topline data; Relative values are calculated as mean change from baseline*

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

Cohort 4 (24W)

ALT (IU/L)[1]





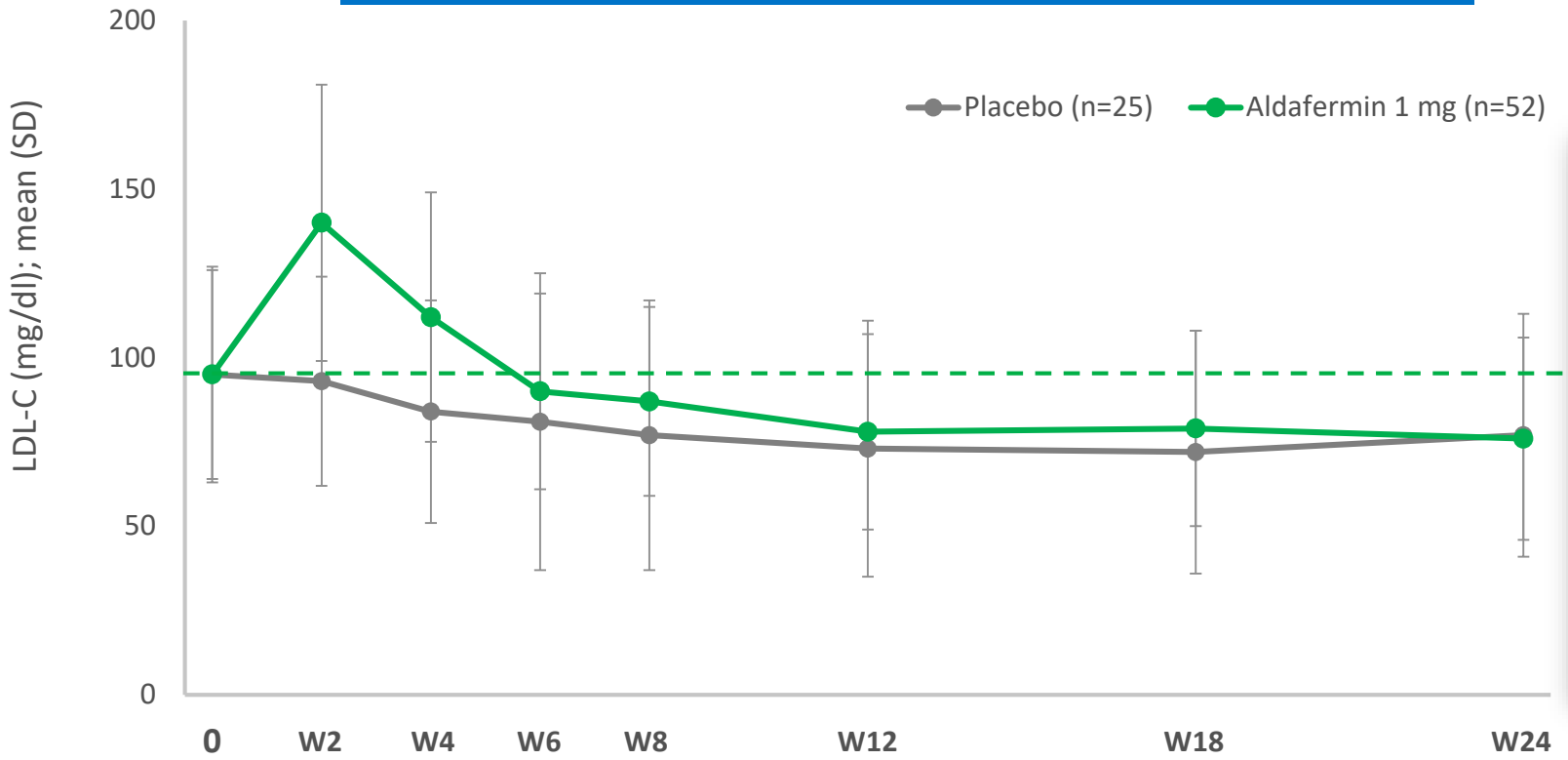
Statistically significant reductions *vs.* placebo also observed with AST and PRO-C3

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

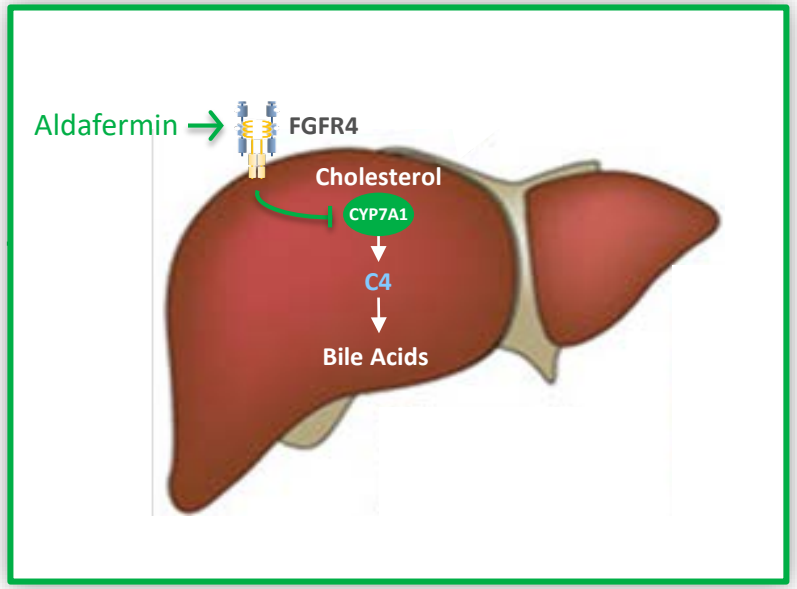
[1] *Cohort 4 preliminary topline data; Relative values are calculated as mean change from baseline*

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

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%)

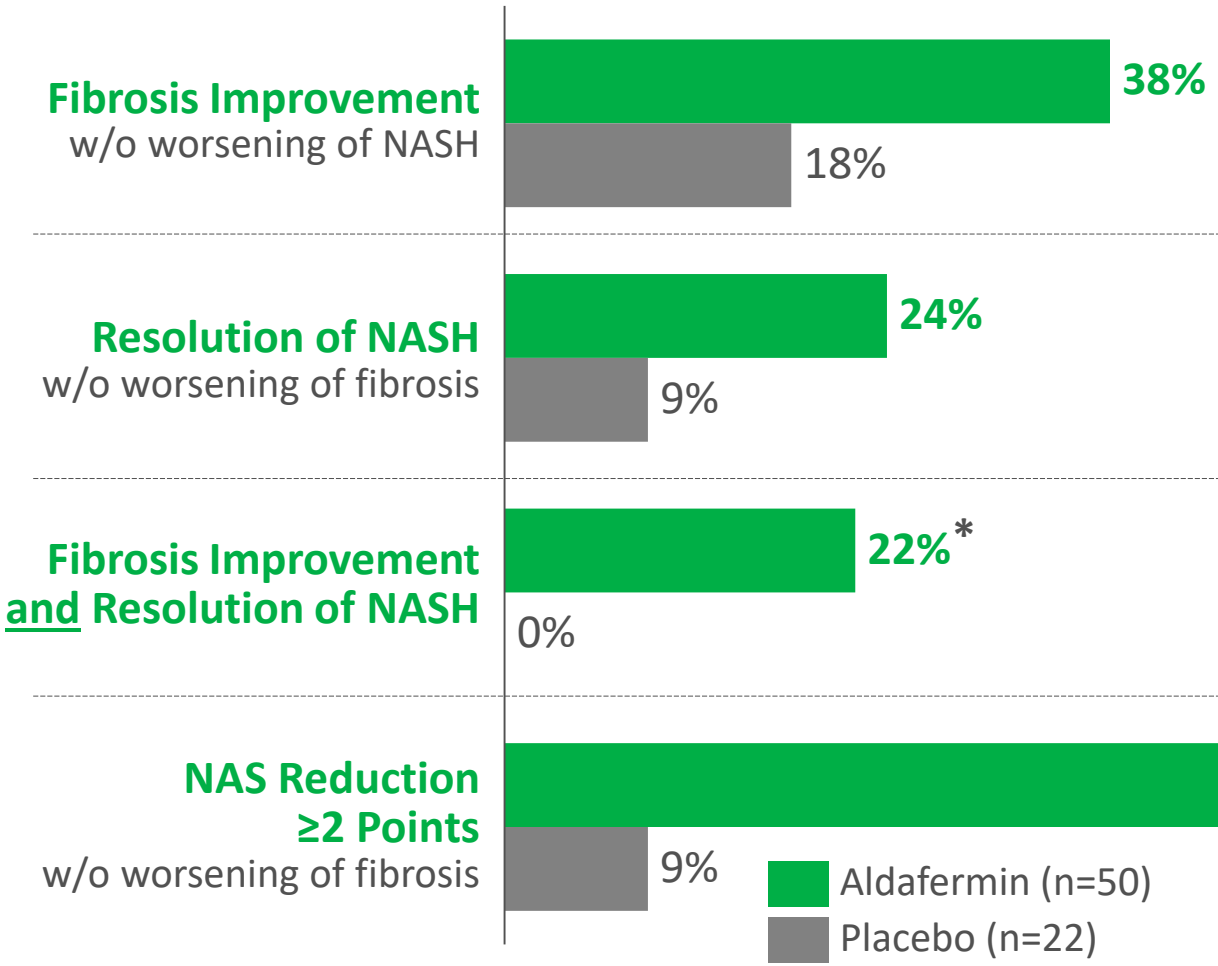
- All SAEs were deemed to be not related to treatment by site investigator

- 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)

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

Multiple Potential Value-Driving Catalysts in 2020

	Product Candidate	Potential Indications	Target Milestones	Target Timing
Wholly-owned	**ALDAFERMIN**	NASH F2/F3	Phase 2 Cohort 4 biopsy data	1Q20 ☑
	ALDAFERMIN	NASH F4	Phase 2b ALPINE 4 FPI	1H20
	ALDAFERMIN	NASH F2/F3	Phase 2b ALPINE 2/3 topline data	1H21
	NGM395	Metabolic	Phase 1 FPI	1H20
Merck collaboration	**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

FPI = first patient in; AMD = age-related macular degeneration; GA = geographic atrophy; CACS = cancer anorexia cachexia syndrome

NASDAQ: NGM

Novel Biology.
Powerful Medicines.
Transformative Impact.