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A Global Disease, A Pioneering Treatment

Akero Therapeutics, Inc.

Corporate Presentation

January 2021

SAFE HARBOR

This presentation may contain “forward-looking statements” of Akero Therapeutics, Inc. (“we,” “us,” “our,” “Akero” or the “Company”) within the meaning of the Private Securities Litigation Reform Act of 1995 relating to our business, operations, and financial conditions, including but not limited to current beliefs, expectations and assumptions regarding: the future of our business; future plans and strategies, including our expectations around the therapeutic potential and clinical benefits of Efruxifermin; our development plans for Efruxifermin, including our belief in the unique potential of Efruxifermin as a foundational NASH therapy; our preclinical and clinical results, including our top-line safety/tolerability, laboratory measures and paired biopsy data from our Phase 2a BALANCED study; the potential benefits resulting from the PRIME designation of EFX; expectations regarding the design, implementation, timing and success of the Phase 2b study and its results; the timing of Phase 3 and its results; the expected timing to complete Cohort C and the collection of voluntary end-of-treatment biopsies; risks related to the competitive landscape; and the potential impact of COVID-19 on strategy, our employees, supply chain, future operations and clinical trials. Words such as, but not limited to, “look forward to,” “believe,” “expect,” “anticipate,” “estimate,” “intend,” “plan,” “would,” “should” and “could,” and similar expressions or words, identify forward-looking statements. New risks and uncertainties may emerge from time to time, and it is not possible to predict all risks and uncertainties. Except as required by law, we assume no obligation to update these forward looking statements publicly, or to update the reasons actual results could differ materially from those anticipated in the forward-looking statements, even if new information becomes available in the future. For a discussion of these and other risks and uncertainties, and other important factors, any of which could cause our actual results to differ from those contained in the forward-looking statements, see the section entitled “Risk Factors” in our most recent annual report on Form 10-K filed with the Securities and Exchange Commission, as well as discussions of potential risks, uncertainties, and other important factors in our other filings with the Securities and Exchange Commission. All information in this presentation is as of the date hereof, and we undertake no duty to update this information unless required by law. Certain information

Certain information contained in this presentation relates to or is based on studies, publications, surveys and other data obtained from third-party sources and the Company’s own internal estimates and research. While the Company believes these third-party sources to be reliable as of the date of this presentation, it has not independently verified, and makes no representation as to the adequacy, fairness, accuracy or completeness of, any information obtained from third-party sources. In addition, all of the market data included in this presentation involves a number of assumptions and limitations, and there can be no guarantee as to the accuracy or reliability of such assumptions. Finally, while we believe our own internal research is reliable, such research has not been verified by any independent source.

CORPORATE HIGHLIGHTS

Efruxifermin: Potential Leading NASH Monotherapy

- Human FGF21 analog addresses all core aspects of NASH pathology
- Engineered for optimal activity and convenient once-weekly dosing
- Phase 2a BALANCED study results among strongest data in field: liver fat reduction coupled with improvements in histology, lipoproteins and glycemic control
- Generally well-tolerated

Key Support from Regulators

- Written guidance from FDA provides that Phase 2b protocol is safe to proceed
- EFX designated as a Priority Medicine (PRIME) based on strength of Phase 2a data

Expected 1H'21 Milestones

- Initiation of Ph2b trial (HARMONY) with 28 and 50mg doses
- Readout from BALANCED study cohort in cirrhotic NASH patients

Experienced Team

- Involved in 20+ FDA approvals
- Extensive experience in drug discovery, development and commercialization

EXTENSIVE DEVELOPMENT AND COMMERCIALIZATION EXPERIENCE INVOLVED IN 20+ MEDICINE APPROVALS

Andrew Cheng, MD, PhD | President & CEO

- 19 years at Gilead
- Chief Medical Officer & HIV Division Head
- Major role in 11 NDA/MAA approvals

Tim Rolph, D.Phil | Founder & CSO

- Over 30 years at Pfizer & Glaxo
- CSO of Pfizer's cardiovascular and metabolic disease unit
- Head of Groton & UK Discovery Research, Pfizer
- Major role in discovery and early clinical evaluation of two medicines: Selzentry (HIV) and Steglatro (Diabetes)

Jonathan Young, PhD, JD | Founder, EVP & COO

- Over 15 years in biotechnology product development, law and regulatory policy
- General Counsel and VP Policy, Braeburn
- Partner and General Counsel, FoxKiser

Kitty Yale | EVP & Chief Development Officer

- Over 25 years at Gilead, Roche, Pfizer
- VP, Gilead Worldwide Clinical Operations
- Major role in 8 global approvals NDA, MAA, JNDA and CFDA

William White | EVP, CFO & Head of Corporate Development

- 18 years in life sciences investment banking at Goldman Sachs, Citigroup and Deutsche Bank
- Most recently, Head of US Life Sciences Investment Banking at Deutsche Bank
- Advised on more than $70bn in M&A and $25bn in financing transactions

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NASH: A SERIOUS AND DEBILITATING MULTI-SYSTEM DISEASE

NASH epidemic fueled by rise in obesity and diabetes
No treatments currently available

A GROWING HEALTH EPIDEMIC

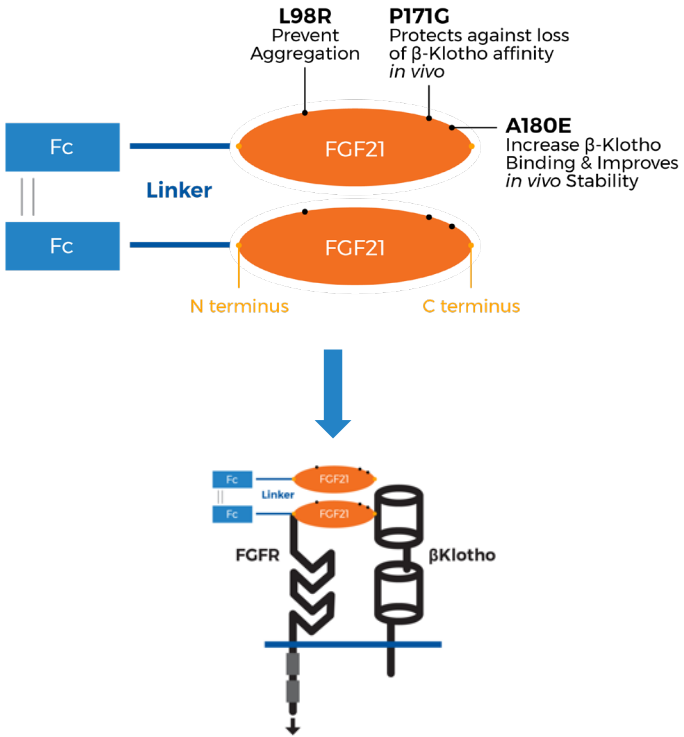
An estimated **17 million Americans** have NASH, with expectation that population will **grow >50% by 2030**

The number of NASH patients with advanced-stage fibrosis and cirrhosis is predicted to **grow to 8 million** in the US by 2030, **an increase of approximately 140% from 2015**

NASH is a **leading cause of liver transplantation** in the US and Europe

The **leading cause of death** for NASH patients is cardiovascular disease

EFX ENGINEERING POTENTIALLY OPTIMAL FOR NASH EFFICACY, WITH CONVENIENT ONCE-WEEKLY DOSING



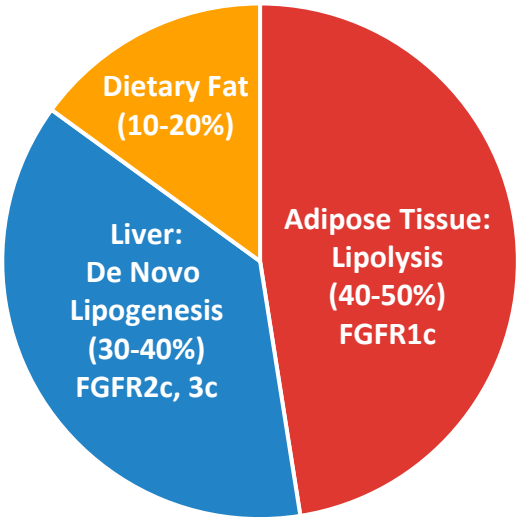
Key attributes

- Akero proprietary Fc-FGF21, Point mutations
- Increases half-life from < **2 hours** to **3-4 days**
- **High affinity** for β-Klotho
- Better translation to **human** pharmacology
- **Balanced potency** at FGFR1c, 2c, 3c
- **Inactive** at FGFR4

Stanislaus, S *et al.* (2017) *Endocrinology* 158(5): 1314-27; Lee, S *et al.* (2018) *Nature* 553: 501-505; Kharitonenkov, A *et al.* (2007) Endocrinology 148(2)774-781

EFX ACTS ON TWO MAJOR SOURCES OF LIVER FAT WITH POTENTIAL FOR OPTIMAL REDUCTION

Sources of Fat Flowing into and Through Liver for NASH Patients



Lambert, JE *et al.* (2014) *Gastroenterology* 146(3): 726-35

Acting on both hepatic and peripheral sources of liver fat is key to optimizing liver fat reduction

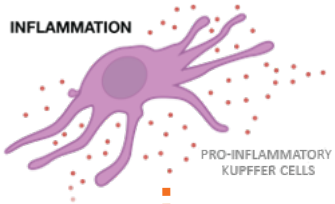
Source of Liver Fat	FGF Receptor	EFX Activity
Lipolysis	FGFR1c	✓
De Novo Lipogenesis	FGFR2c FGFR3c	✓

EFX DIRECT AND INDIRECT ANTI-FIBROTIC EFFECTS

Direct Effect

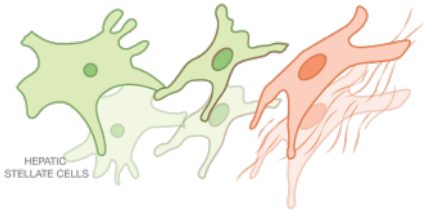
Indirect Effect

Kupffer Cell Activation

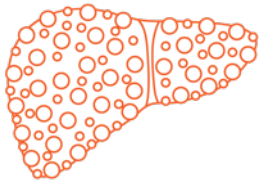


Hepatocyte Stress & Death

EFX



Myofibroblast Differentiation

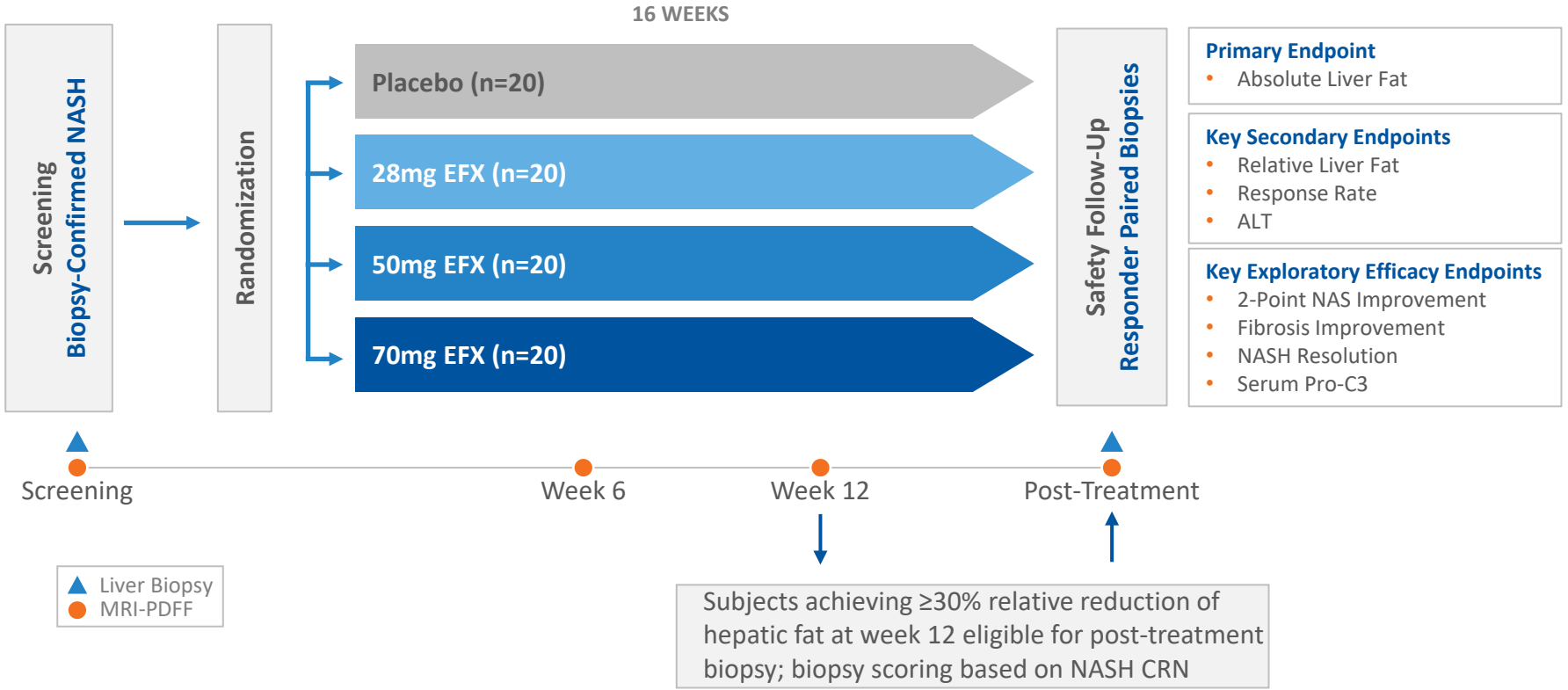


Fibrosis

Bao, L *et al.* (2018) *Br J Pharmacol* 175:3379-3393; Fisher, FM *et al.* (2014) *Gastroenterology* 147:1073-1083.e6; Jimenez, V *et al.* (2018) *EMBO Mol Med* 10:e8791; Lee, JH *et al.* (2016) *Am J Transl Res* 8:4750-4763; Sanyal, A *et al.* (2018) *Lancet* 392:2705-2717; Le, CT *et al.* (2018) *PLOS one* 13:e0192146; Xu, P *et al.* (2016) *Toxicol Appl Pharmacol* 290:43-53; Yu, Y *et al.* (2016) *Int Immunopharmacol* 38:144-152

**Cited literature available on company website*

PHASE 2A TRIAL (BALANCED) DESIGN

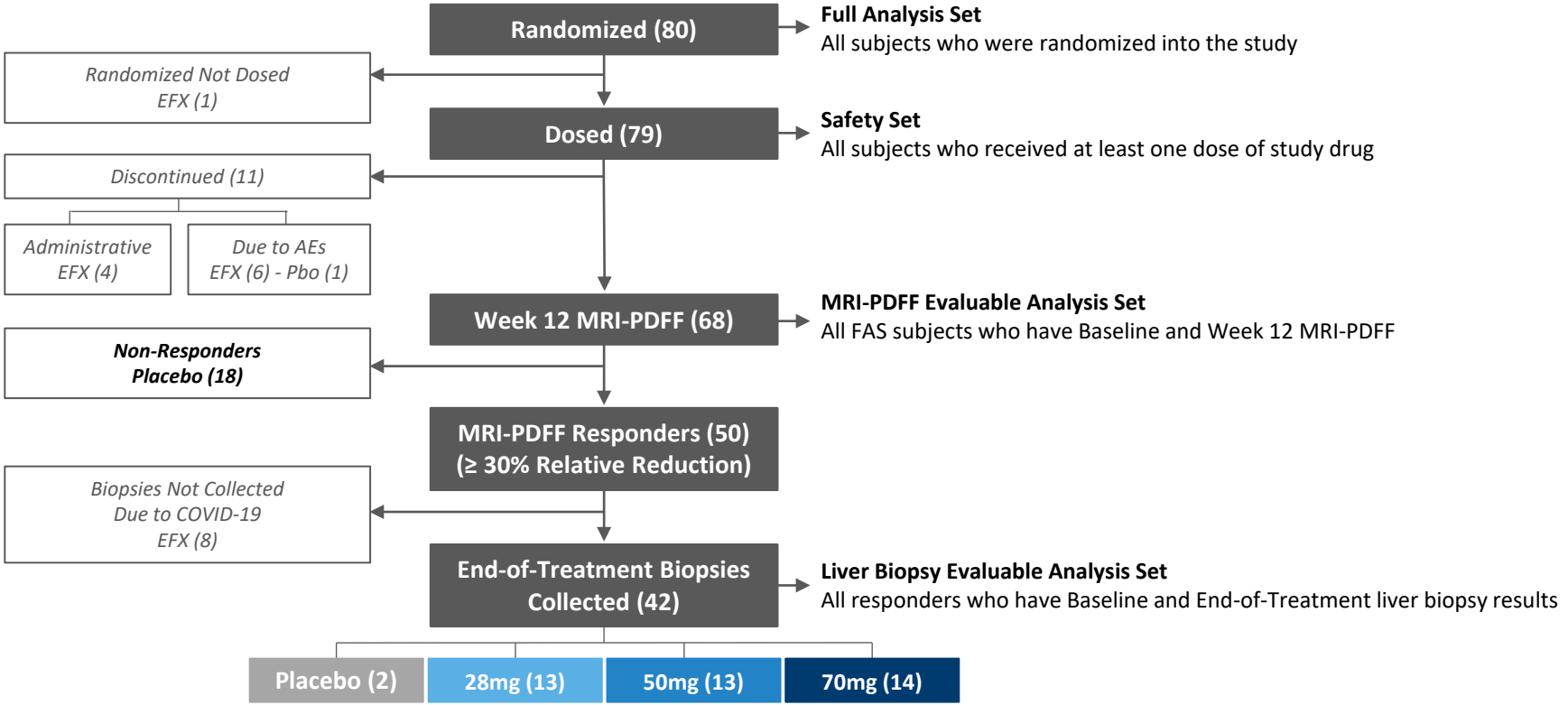


BASELINE DEMOGRAPHICS

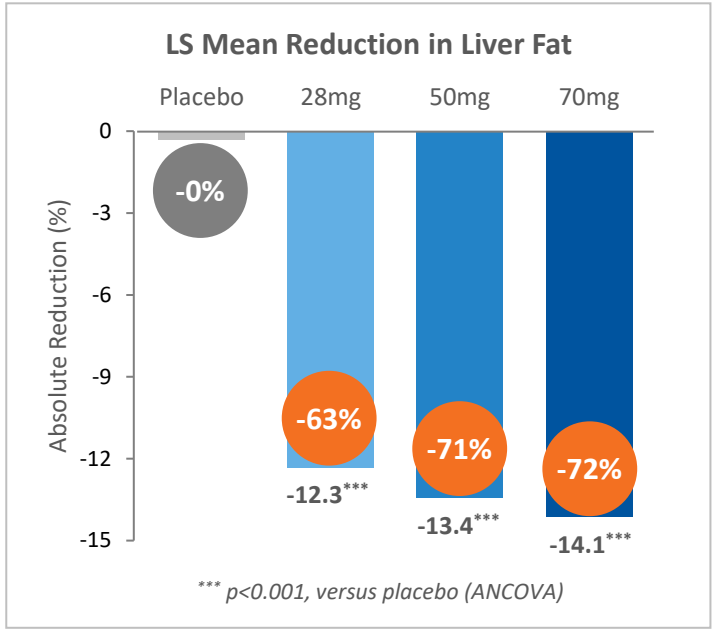
Parameter Mean	Placebo (N=21)	EFX 28mg (N=19)	EFX 50mg (N=20)	EFX 70mg (N=20)
Age (Years)	52	50	53	53
Sex (Male/Female)	6/15	9/10	10/10	9/11
Weight (kg)	99.6	108.2	103.6	103.1
BMI (kg/m^2)	37.6	38.8	36.7	37.2
Liver Fat Content (% by MRI-PDFF)	19.3	21.4	18.3	19.4
NAFLD Activity Score (NAS)	5.1	5.6	5.1	5.6
Fibrosis Stage (% F2-F3)	62	63	65	65
Alanine Aminotransferase (ALT) (U/L)	50.7	62.5	53.4	56.8
Aspartate Aminotransferase (AST) (U/L)	38.6	41.1	35.4	44.6
% Type 2 Diabetes	67	37	50	50

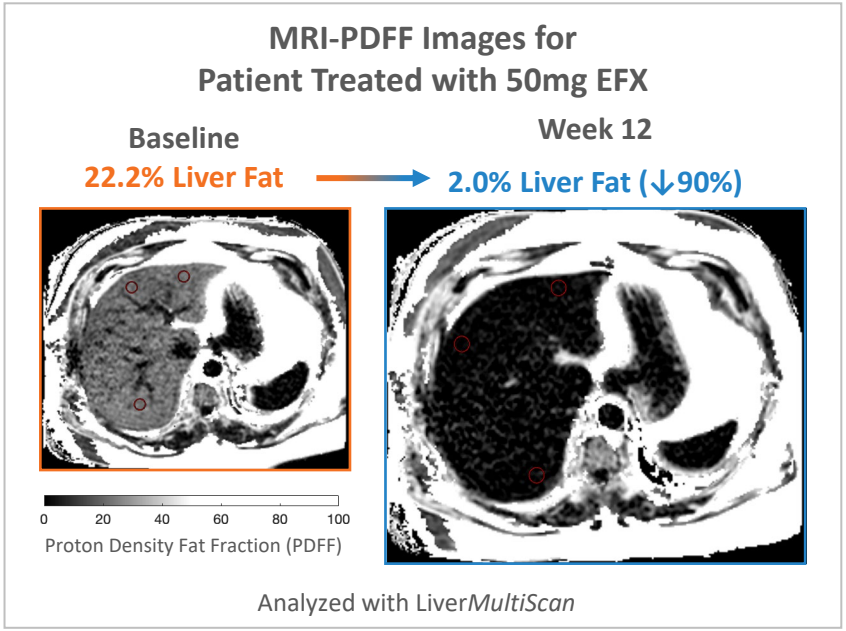
Source Data: Full Analysis Set

PATIENT DISPOSITION



SUBSTANTIAL REDUCTIONS IN LIVER FAT AT WEEK 12 ACROSS ALL DOSE GROUPS





* p<0.05, ** p<0.01, *** p<0.001 versus placebo (ANCOVA)

Source Data: Full Analysis Set

MAGNITUDE OF LIVER FAT REDUCTION

Proportion of Patients Achieving Fat Reduction Thresholds at Week 12

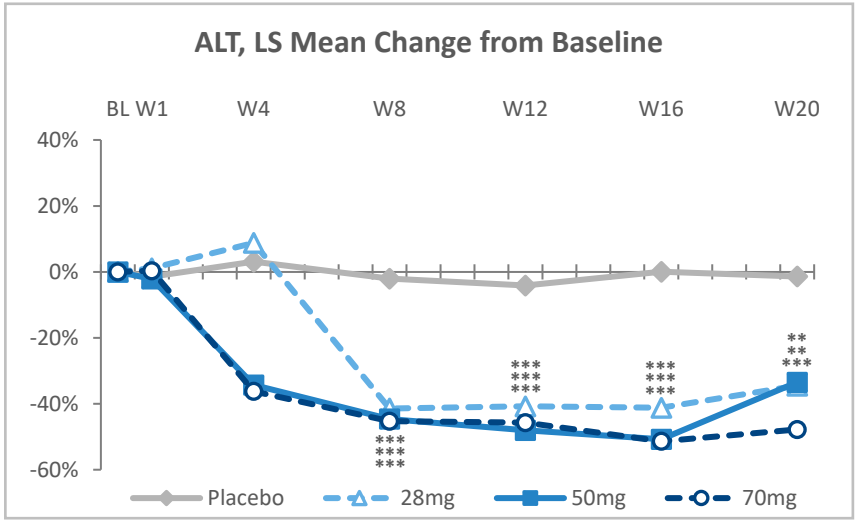
Endpoint	Placebo[1] (N=20)	28mg (N=16)	50mg (N=17)	70mg (N=15)
Relative Reduction in Liver Fat				
≥30%	10%	100%**	100%***	100%***
≥50%	5%	69%**	100%***	93%***
≥70%	5%	50%*	53%**	80%***
Normalization of Liver Fat Content				
≤5%	5%	25%*	53%**	67%***

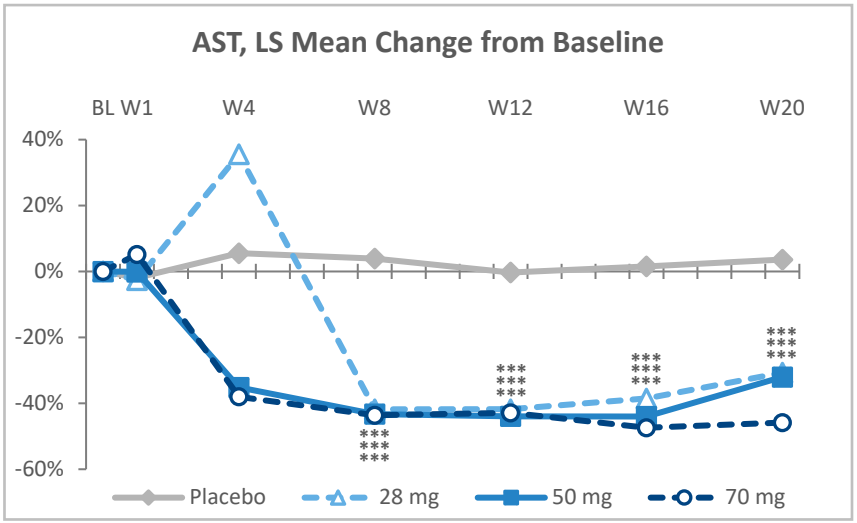
[1] A single placebo responder lost 25 pounds over 16 weeks (11% weight reduction)

* p<0.05, ** p<0.01, *** p<0.001 versus placebo (ANCOVA)

Source Data: MRI-PDFF Evaluable Analysis Set

SUBSTANTIAL REDUCTIONS IN MARKERS OF LIVER INJURY AFTER 16 WEEKS OF TREATMENT



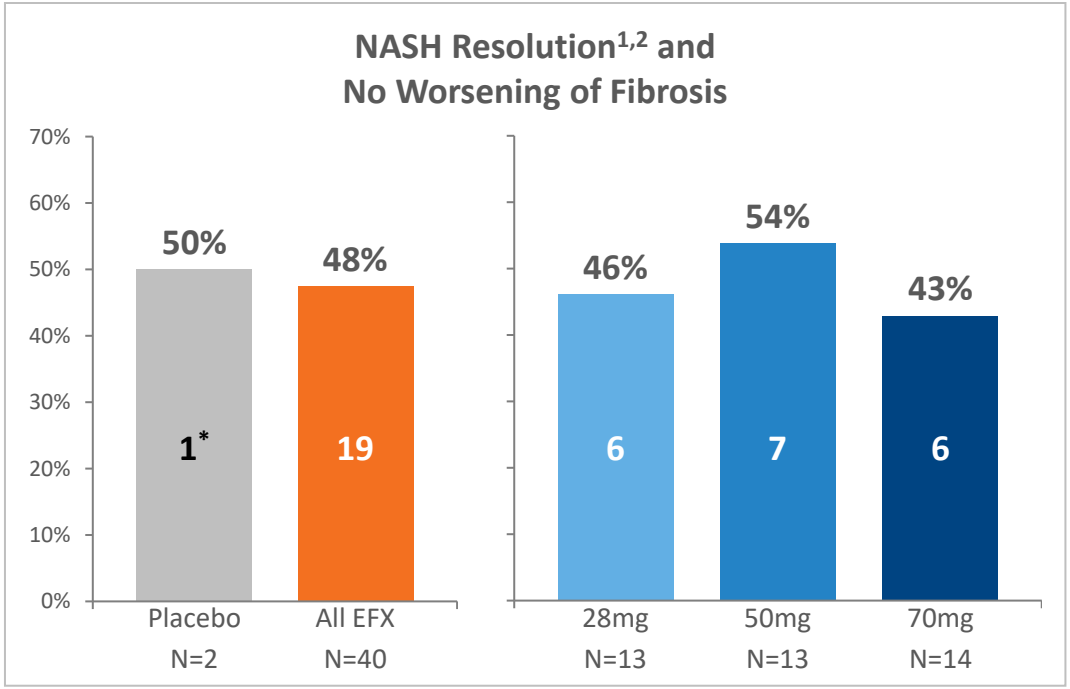


Similar dose-related improvements observed for GGT & ALP

** p<0.01, *** p<0.001, versus placebo (MMRM)

Source Data: Full Analysis Set

HIGH RESPONSE RATES ON NASH RESOLUTION AFTER 16 WEEKS ACROSS ALL DOSE GROUPS



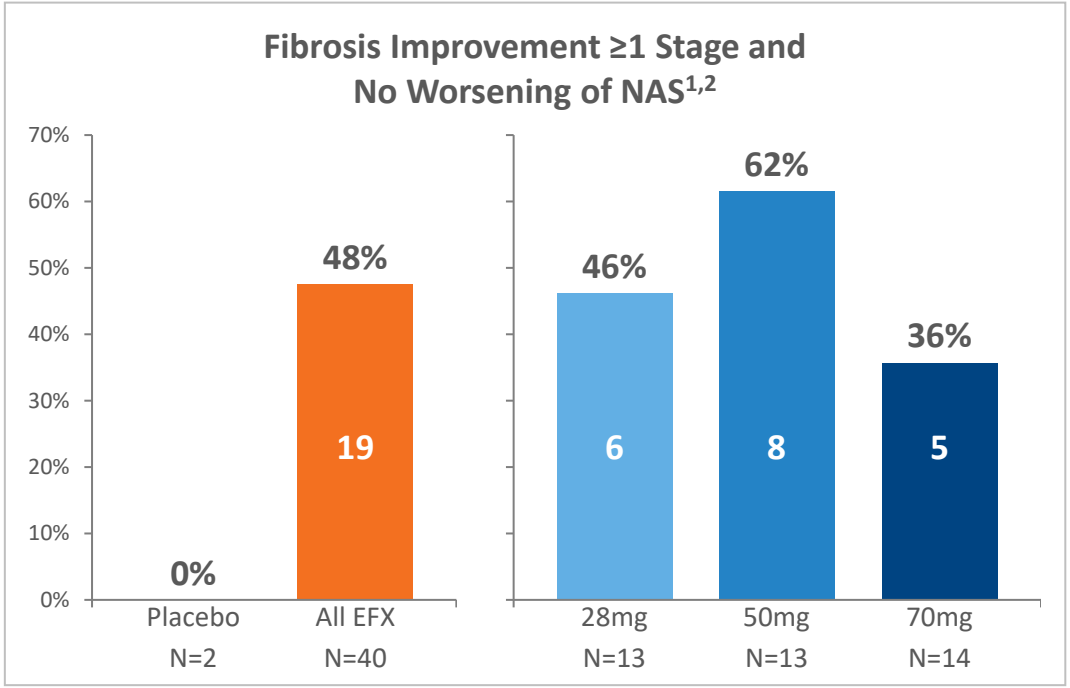
Biopsy Reading

- All baseline and end-of-treatment biopsies were centrally read by a single NASH-CRN pathologist
- Baseline biopsies were not re-read with end-of-treatment biopsies
- All biopsies were read blinded to both treatment assignment and patient

[1] NAS score of 0 or 1 for lobular inflammation and a score of 0 for ballooning
[2] Secondary and exploratory histological endpoints were not powered for statistical significance
* A single placebo responder lost 25 pounds over 16 weeks (11% weight reduction)

Source Data: Liver Biopsy Evaluable Analysis Set

HIGH RATES OF FIBROSIS IMPROVEMENT AFTER 16 WEEKS ACROSS ALL TREATED PATIENTS

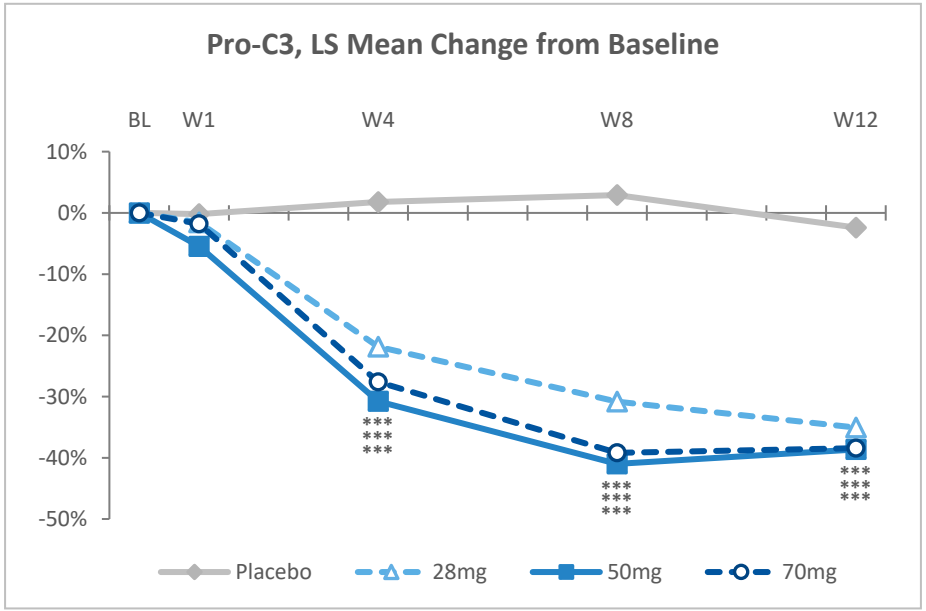


[1] Improvement in liver fibrosis greater than or equal to one stage and no worsening of NASH (defined as no increase in NAS for ballooning, inflammation, or steatosis)

[2] Secondary and exploratory histological endpoints were not powered for statistical significance

Source Data: Liver Biopsy Evaluable Analysis Set

RAPID IMPROVEMENTS IN FIBROSIS BIOMARKERS CONSISTENT WITH HISTOLOGICAL IMPROVEMENTS



*** p<0.001, versus placebo (MMRM)

Pro-C3, LS Mean (ug/L)

Dose Group	Baseline	Δ Week 12
Placebo	16.1	-1.5
28mg	19.2	-6.1***
50mg	16.2	-5.9***
70mg	17.2	-6.7***

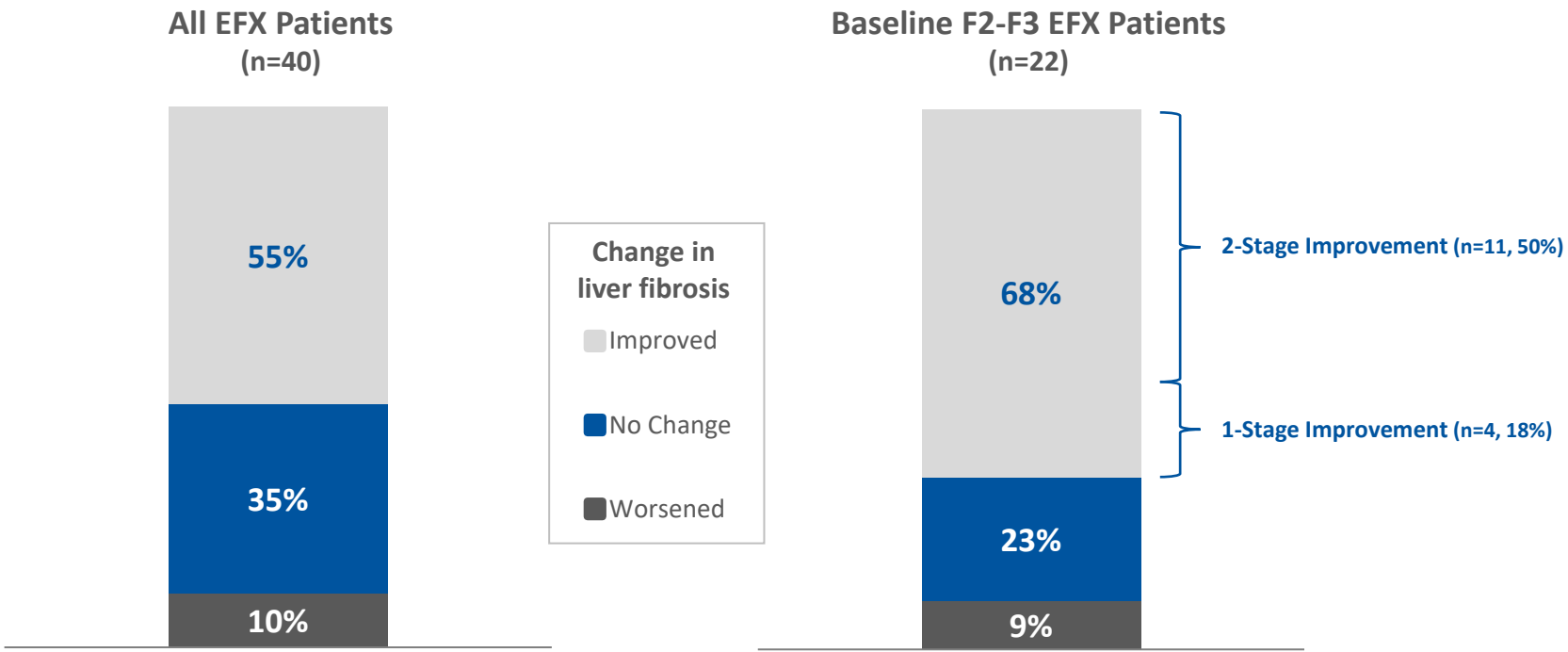
Enhanced Liver Fibrosis (ELF) Score, LS Mean

Dose Group	Baseline	Δ Week 12
Placebo	9.4	0.0
28mg	9.5	-0.7***
50mg	9.5	-0.8***
70mg	9.6	-0.4*

* p<0.05, *** p<0.001 versus placebo (ANCOVA)

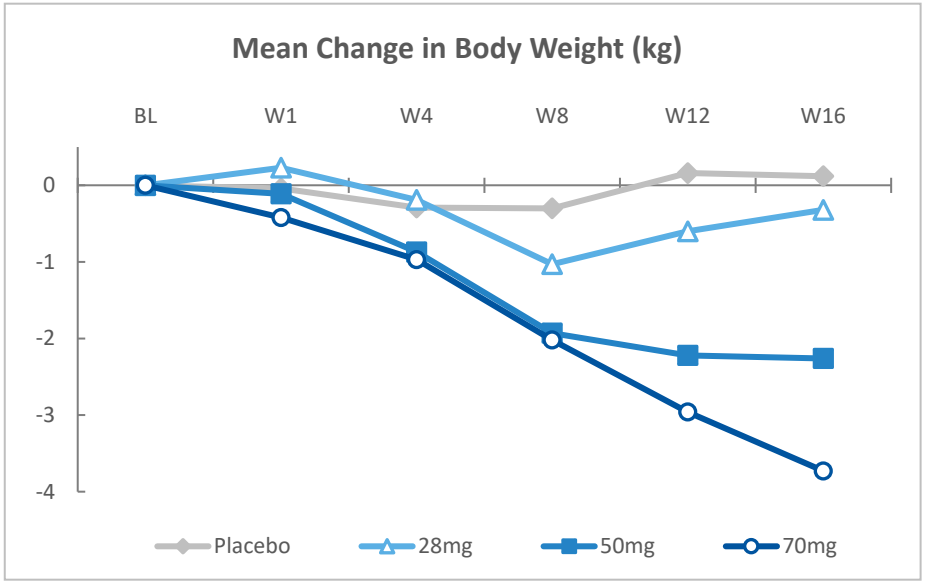
Source Data: Full Analysis Set

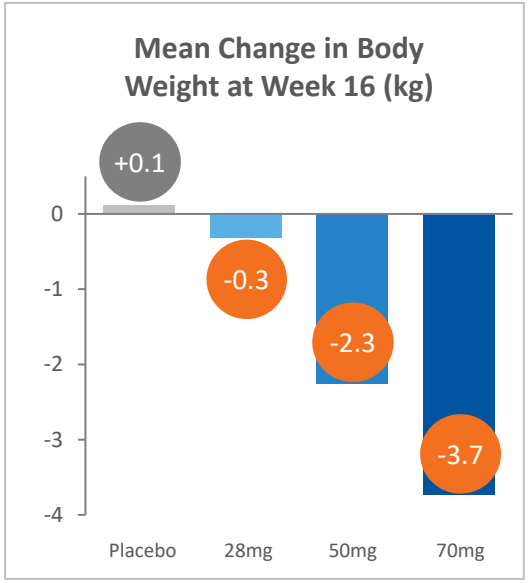
FIBROSIS IMPROVEMENT IN F2-F3 PATIENTS: HALF OF PATIENTS IMPROVED 2 STAGES



Source Data: Liver Biopsy Evaluable Analysis Set

WEIGHT LOSS OBSERVED FOR ALL DOSE GROUPS

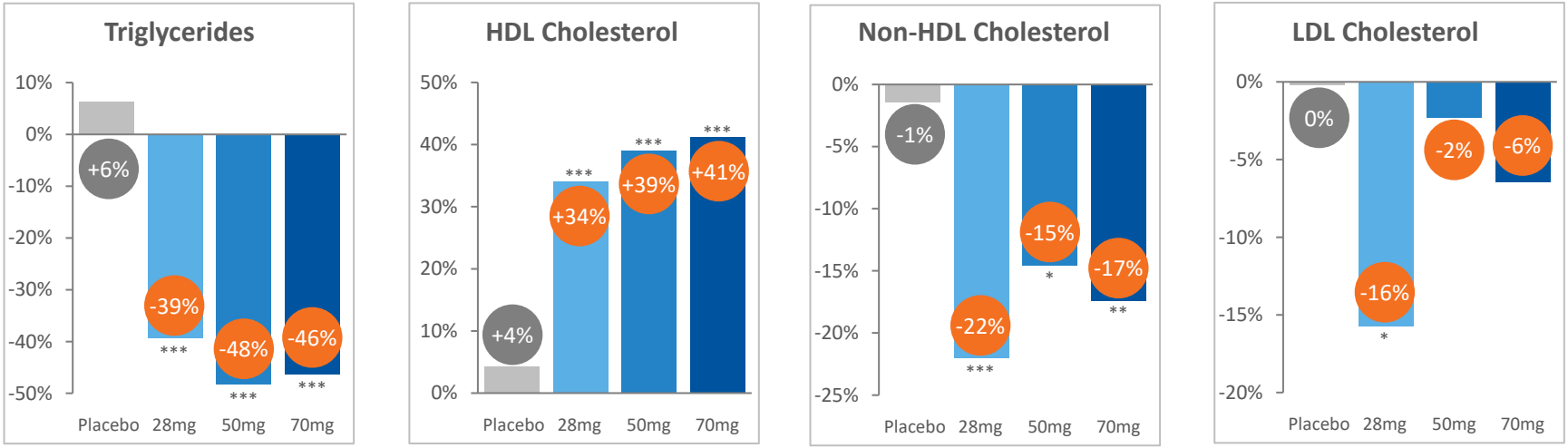




Source Data: Full Analysis Set

IMPROVED LIPOPROTEIN PROFILE

LS Mean Change From Baseline to Week 16 (%)

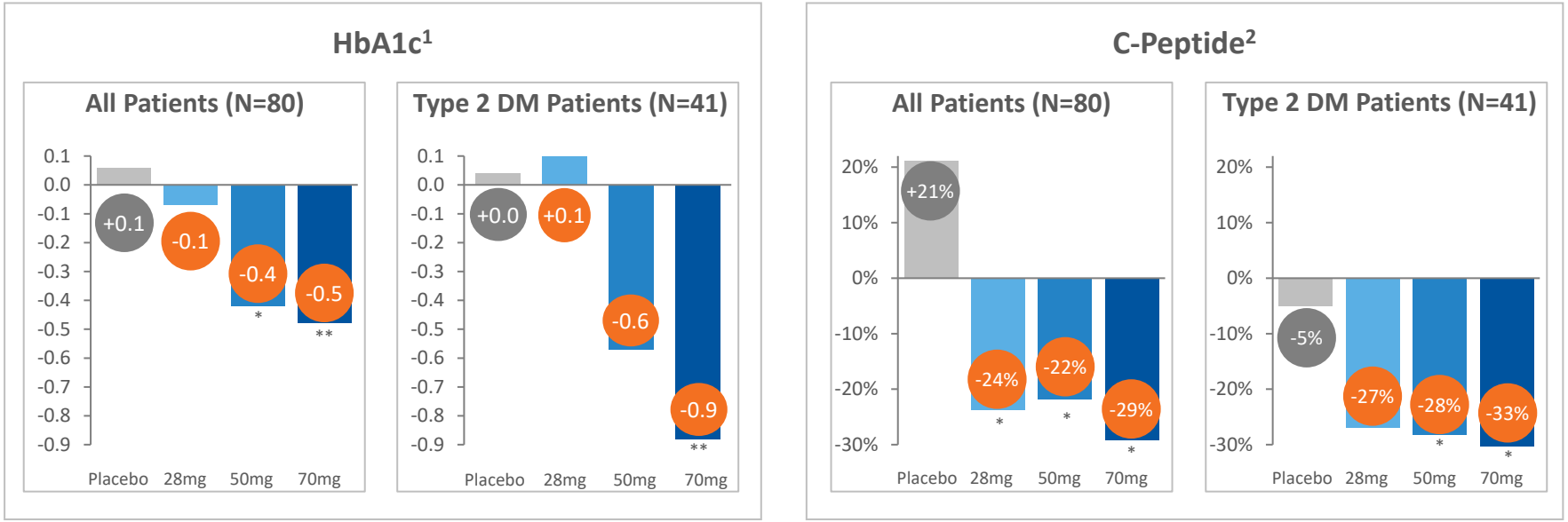


* p<0.05, ** p<0.01, *** p<0.001 versus placebo (ANCOVA)

Source Data: Full Analysis Set

CLINICALLY MEANINGFUL IMPROVEMENTS IN GLYCEMIC CONTROL AFTER 16 WEEKS

LS Mean Change From Baseline to Week 16 (%)



[1] Absolute change from baseline, %

[2] Relative percent change from baseline

* p<0.05, ** p<0.01, versus placebo (ANCOVA)

Source Data: Full Analysis Set

DRUG-RELATED TREATMENT-EMERGENT ADVERSE EVENTS (TEAE)

Most Common (>10%) Drug-Related AEs*	Placebo (N=21)	All EFX (N=58)	EFX 28mg (N=19)	EFX 50mg (N=19)	EFX 70mg (N=20)
Diarrhea	2 (10%)	21 (36%)	5 (26%)	10 (53%)	6 (30%)
Nausea	0 (0%)	20 (34%)	6 (32%)	4 (21%)	10 (50%)
Increased appetite	1 (5%)	13 (22%)	4 (21%)	4 (21%)	5 (25%)
Vomiting	0 (0%)	9 (16%)	5 (26%)	2 (11%)	2 (10%)
Frequent bowel movements	0 (0%)	8 (14%)	3 (16%)	2 (11%)	3 (15%)
Abdominal pain	0 (0%)	7 (12%)	1 (5%)	3 (16%)	3 (15%)
Injection site erythema	0 (0%)	7 (12%)	2 (11%)	0 (0%)	5 (25%)
Injection site reaction	0 (0%)	6 (10%)	2 (11%)	1 (5%)	3 (15%)
Fatigue	2 (10%)	6 (10%)	0 (0%)	1 (5%)	5 (25%)
TEAE/SAE Disposition	**Placebo**	**All EFX**	**28mg**	**50mg**	**70mg**
TEAE Leading to Death	0	0	0	0	0
TEAE Leading to Discontinuation	1[a]	6	2[b]	0	4[c]
Serious Adverse Event (SAE)	0	2	1[d]	0	1

*Across EFX dose groups

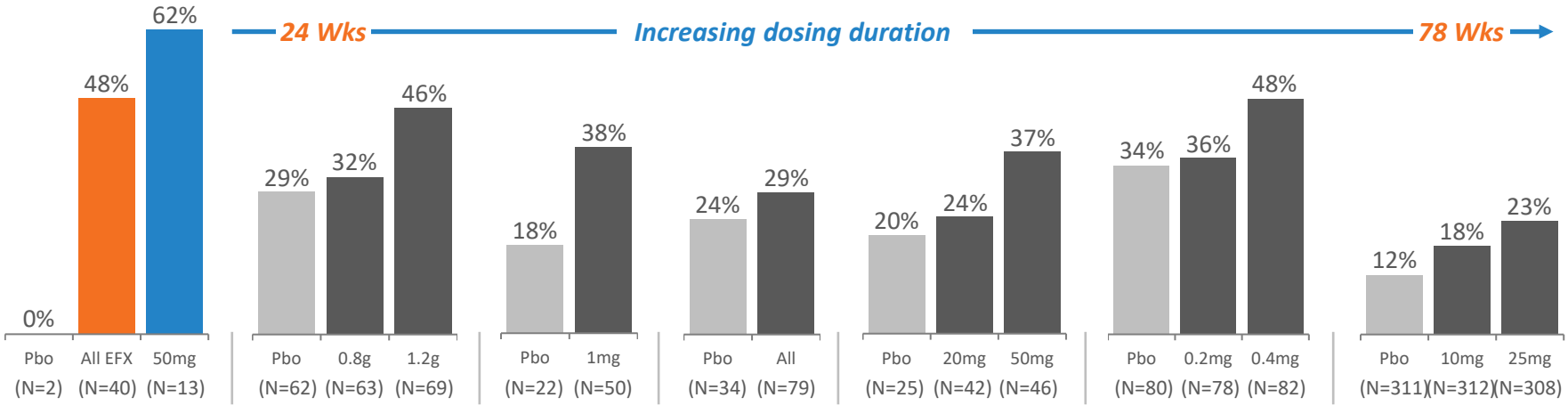
[a] *Muscular Weakness & Myalgia;* [b] *Nausea, Vomiting & Dysgeusia; Panic Attack and Anxiety-Linked Tremor;*
[c] *Dysphagia (Not Drug Related); Acute Pancreatitis (also an SAE); Vomiting; Fatigue & Nausea;* [d] *Related to pre-dosing liver biopsy*

Source Data: Safety Set

NASH DEVELOPMENT LANDSCAPE: FIBROSIS IMPROVEMENT

Proportion of Subjects with ≥1 Stage Improvement in Fibrosis and No Worsening of NAS[1]

akero	inventiva	NGM Bio	Madrigal Pharmaceuticals	CymaBay	novo nordisk	Intercept
Efruxifermin	**Lanifibranor**	**Aldafermin**	**Resmetirom**	**Seladelpar**	**Semaglutide**	**Ocaliva**
16 Wks (Ph2a)	24 Wks (Ph2b)	24 Wks (Ph2a)	36 Wks (Ph2a)	52 Wks (Ph2a)	72 Wks (Ph2b)	78 Wks (Ph3)
Weekly Injection	Daily Oral	Daily Injection	Daily Oral	Daily Oral	Daily Injection	Daily Oral



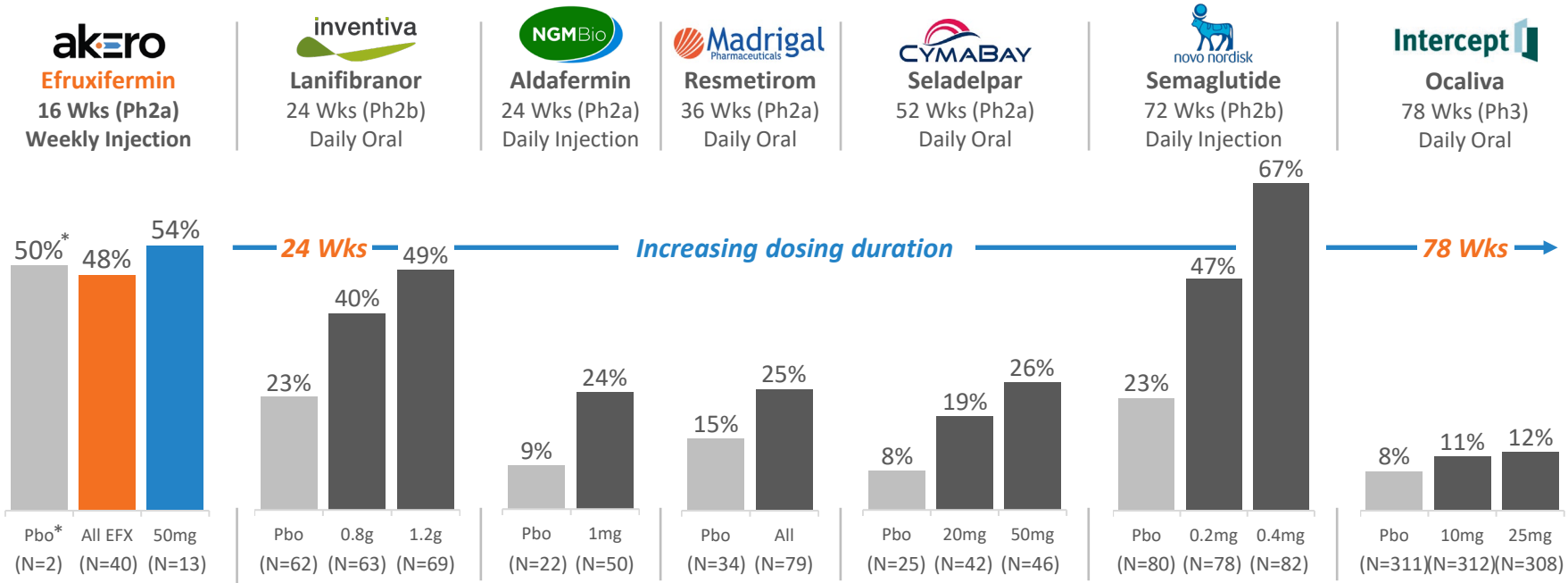
Note: These data are derived from different clinical trials at different points in time, with differences in trial design and patient populations. No head-to-head clinical trials have been conducted.

Inventiva (2020) June 16 Corporate Presentation; NGM Bio (2020) June 3 Corporate Presentation; Harrison, S et al. (2019) Lancet 394(10213):2012-24; CymaBay (2020) March 12 Press Release; Novo Nordisk (2020) June 19 R&D Investor Presentation; Younossi Z et al. (2019) Lancet 394(10215):2184-96. All trademarks are the property of their respective owners.

[1] FDA Guidance for Industry: Noncirrhotic Nonalcoholic Steatohepatitis With Liver Fibrosis: Developing Drugs for Treatment (2018)

NASH DEVELOPMENT LANDSCAPE: NASH RESOLUTION

Proportion of Subjects with Resolution of NASH and No Worsening of Fibrosis[1]



** A single placebo responder lost 25 pounds over 16 weeks (11% weight reduction)*

Note: These data are derived from different clinical trials at different points in time, with differences in trial design and patient populations. No head-to-head clinical trials have been conducted.

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[1] FDA Guidance for Industry: Noncirrhotic Nonalcoholic Steatohepatitis With Liver Fibrosis: Developing Drugs for Treatment (2018)

FGF21 DEVELOPMENT LANDSCAPE

Noninvasive Measures: Percent Change From Baseline to End of Study	Akero (EFX) 16 weeks			BMS (Pegbelfermin) 16 weeks			89Bio (BIO89-100) 12 weeks			
Dose	pbo	28 QW	50 QW	pbo	20 QW	10 QD	pbo	18 QW	27 QW	36 Q2W
Patient Population	Biopsy-confirmed NASH			Biopsy-confirmed NASH			80% NAFLD; 20% biopsy-confirmed NASH*			
≥1 Stage Fibrosis Improvement and No Worsening of NASH, % of Subjects	0%	46%	62%	No end-of-study biopsy			No end-of-study biopsy			
MRI-PDFF, % relative reduction	0	-63	-71	-6	-26	-38	+10	-36	-60	-50
ALT	0	-41	-51	-5	-22	-33	-4	-27	-44	-40
Triglycerides	+6	-39	-48	0	-5	-5	-2	-18	-28	-21
HDL-C	+4	+34	+39	-2	+12	+13	+2	+9	+3	+10
LDL-C	0	-16	-2	+1	+1	-11	+1	+3	-16	-4
Adiponectin	-8	+65	+80	-4	+16	+15	-4	+29	+61	+24
% HbA1c, absolute change	+0.1	-0.1	-0.4	NR			0	+0.1	-0.3	+0.5

Note: These data are derived from different clinical trials at different points in time, with differences in trial design and patient populations. No head-to-head clinical trials have been conducted.

NR, not reported

Sanyal et al (2019) Lancet;
89Bio October 5 Corporate Presentation

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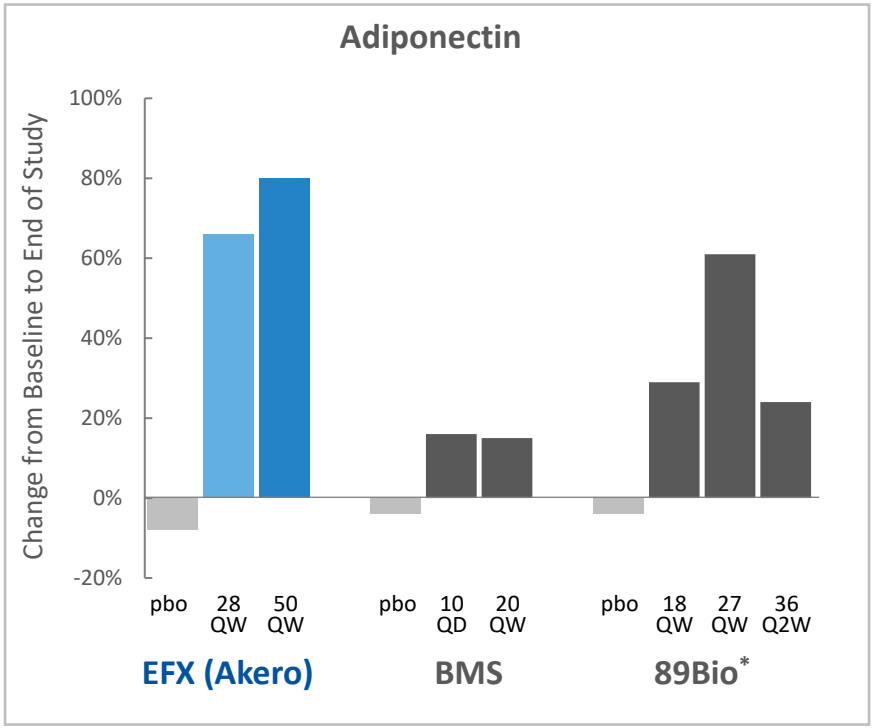
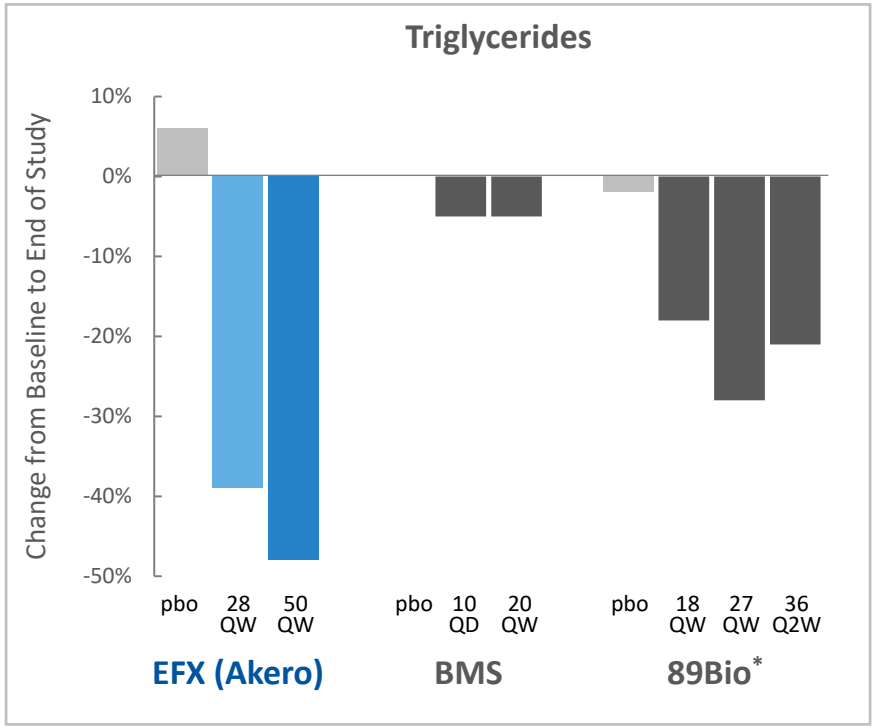
* *Subjects with biopsy-confirmed NASH were present only in the 18mg QW & 18 Q2W cohorts (Loomba et al., 2020 AASLD Poster #LP34)*

PERIPHERAL FGFR1c ACTIVATION





Note: These data are derived from different clinical trials at different points in time, with differences in trial design and patient populations. No head-to-head clinical trials have been conducted.

Sanyal et al (2019) Lancet;
89Bio October 5 Corporate Presentation

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* *Subjects with biopsy-confirmed NASH were present only in the 18mg QW & 18 Q2W cohorts (Loomba et al., 2020 AASLD Poster #LP34)*

FGF21 DEVELOPMENT LANDSCAPE: SUMMARY

Consideration	Fc-FGF21 Fusion Protein (Akero)	Pegylated FGF21 (BMS or 89Bio)
Patient Population: NASH diagnosed only by biopsy; NASH patients have more advanced disease and worse comorbidities	Biopsy-confirmed NASH	BMS: biopsy-confirmed NASH; 89Bio: ~20% biopsy-confirmed NASH*
Histology: Fibrosis only histological endpoint correlated with liver outcomes	Demonstrated fibrosis improvement by histology	BMS: histology data pending 89Bio: no histology data
Liver Fat Reduction: Max reduction requires inhibition of both hepatic fat synthesis and adipose tissue lipolysis	71% (50mg QW)	BMS: 38% (10mg QD) 89Bio: 60% (27mg QW*)
Liver Enzymes (LFTs): Reductions indicate improved liver health	Large reductions in LFTs; Consistent dose response	BMS/89Bio: Smaller effects on LFTs
Lipids: Cardiovascular risk #1 cause of mortality for NASH patients. Reflects liver and adipose effects	Robust and consistent TG and HDL-C effects	BMS/89Bio: Smaller effects on TG and HDL-C
Glycemic Control: Improvement in HbA1c mediated by peripheral insulin sensitization; 50% NASH patients diabetic	Significant decrease in HbA1c	BMS: HbA1c not reported 89Bio: no significant change in HbA1c
Safety & Tolerability: Baseline patient population influences profile; Mild GI events common for FGF21 in NASH patients	In line with FGF21 class	BMS: In line with FGF21 class 89Bio: ~80% NAFLD*

***EFX** delivered numerically largest effects, a clear dose response, with maximal or near-maximal effect at 50mg QW*

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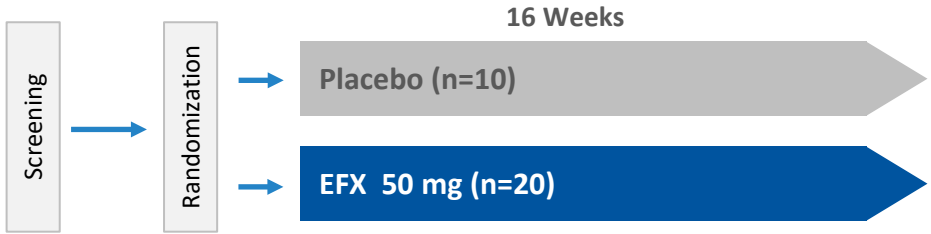
* *Subjects with biopsy-confirmed NASH were present only in the 18mg QW & 18 Q2W cohorts (Loomba et al., 2020 AASLD Poster #LP34)*

BALANCED F4 COHORT EXPANSION (COHORT C) TRIAL DESIGN

BALANCED study expanded to include cohort of patients with compensated cirrhosis (F4), Child-Pugh Class A; results expected to inform long-term development in cirrhotic patients

Number of Subjects
30

Primary Endpoint
Safety & tolerability



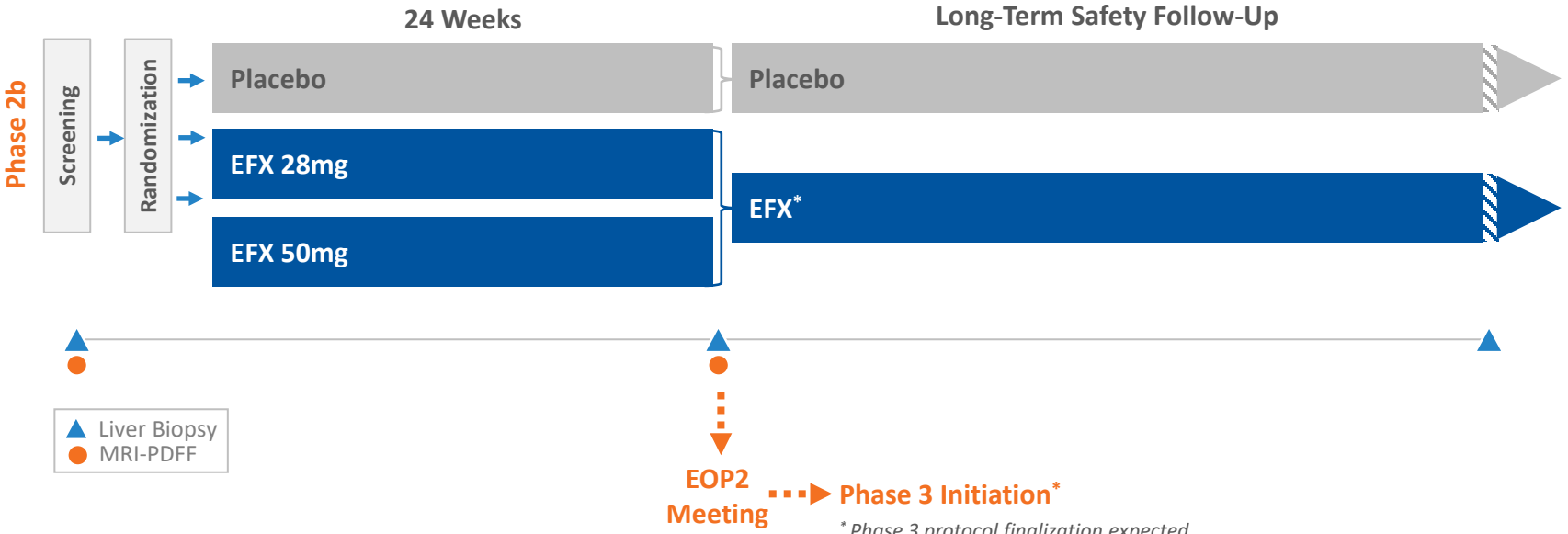
Data readout with biopsy results anticipated in 1H'21

PHASE 2B TRIAL (HARMONY) DESIGN

Key Inclusion Criteria
- F2-3 NASH
- NAS ≥4
- Liver fat ≥8%

Phase 2b Primary Endpoint
- Fibrosis Improvement

Key Secondary Efficacy Endpoints
- NASH Resolution
- Fibrosis Markers
- Lipoproteins
- Glycemic Control
- Weight Change



*Phase 3 protocol finalization expected after end-of-Phase 2 meeting with FDA

STRONG FINANCIAL POSITION

COMPLETED UPSIZED IPO

June 20, 2019

~$106M

Raised in aggregate gross proceeds

$16

Priced upsized IPO at top of marketing range

COMPLETED UPSIZED FOLLOW-ON OFFERING

July 10, 2020

~$216M

Raised in aggregate gross proceeds

$36

Priced upsized offering at top of marketing range

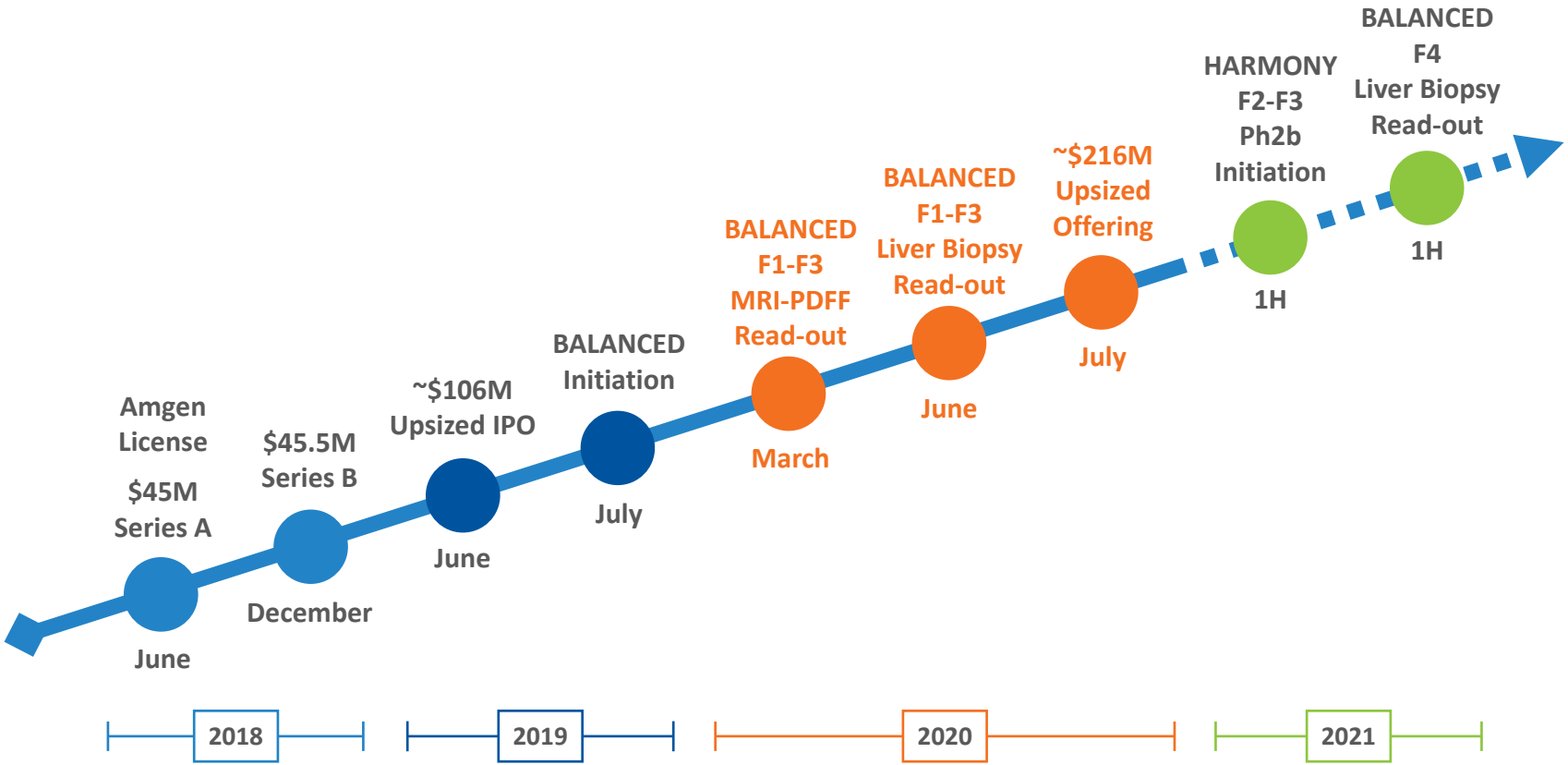
CASH ON HAND

September 30, 2020

~$292M

Cash, cash equivalents and short-term marketable securities

CONSISTENT RECORD OF MILESTONE DELIVERY



EFRUXIFERMIN AFTER 16 WEEKS: POTENTIALLY FOUNDATIONAL NASH MONOTHERAPY

Improved Non-Invasive Markers

- 63-72% relative reduction in liver fat
- ~40% reduction in liver enzymes
- Reduction in ELF and Pro-C3

Improved Histology

- 48% fibrosis improvement ≥1 stage and no worsening of NASH
- 50% two-stage fibrosis improvement in patients with F2-F3 fibrosis at baseline

Improved NASH Comorbidities

- Improved HbA1c and C-peptide
- Reduction in triglycerides
- No LDL-C increase
- Weight loss across all dose groups

Safety & Tolerability

- Generally well-tolerated
- Transient mild/moderate GI events
- No TEAE discontinuations at 50mg

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A Global Disease, A Pioneering Treatment

NASDAQ: AKRO