Restoring Balance. Renewing Life.

Corporate Presentation

September 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; the Phase 2b HARMONY and SYMMETRY studies, including expected timing to complete enrollment, report preliminary results, and other related milestones; the availability of a new drug product formulation to support Phase 3 clinical trials; risks related to the competitive landscape; expectations regarding the Company’s use of capital, expenses and other future financial results; 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 (EFX): Highly Differentiated, Potentially Best-in-Class NASH Medicine	• Human FGF21 analog addresses all core aspects of NASH pathology • Engineered for optimal activity and convenient once-weekly dosing • We believe Phase 2a BALANCED study results in biopsy-confirmed NASH patients among strongest data in field for both F1-F3 and cirrhotic (F4) patients • Generally well-tolerated
Regulatory Status	• EFX designated as a Priority Medicine (PRIME) based on strength of Phase 2a data • Plan to pursue marketing approval in 2 distinct patient populations: F2/F3 & F4 NASH
Milestones: Recent & Expected Near-Term	• Dosed first patient in Phase 2b HARMONY study in F2/F3 patients in March 2021 • Initiated Phase 2b SYMMETRY study in cirrhotic (F4) patients in July 2021 • Preliminary results of Phase 2b HARMONY study expected in 3Q'22 • Release newly-formulated drug product for Phase 3 use expected in 1H'23
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

Kitty Yale | 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

Tim Rolph, D.Phil | Co-Founder & Chief Scientific Officer

- 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 | Co-Founder & COO

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

William White | 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

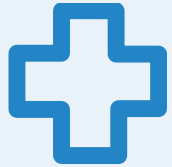
NASH: A Serious and Debilitating Multi-System Disease

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**

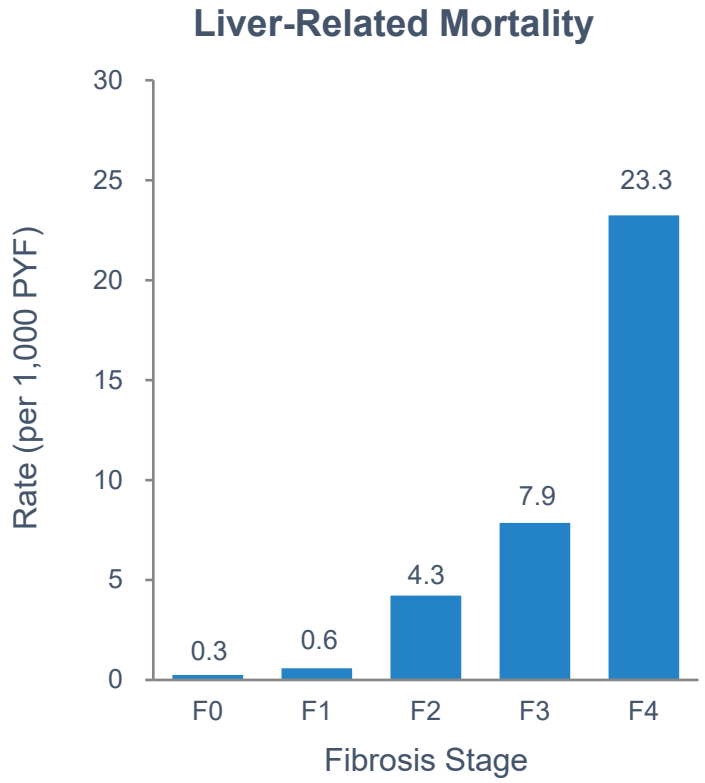


NASH is a **leading cause of liver transplantation** in the US and Europe

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

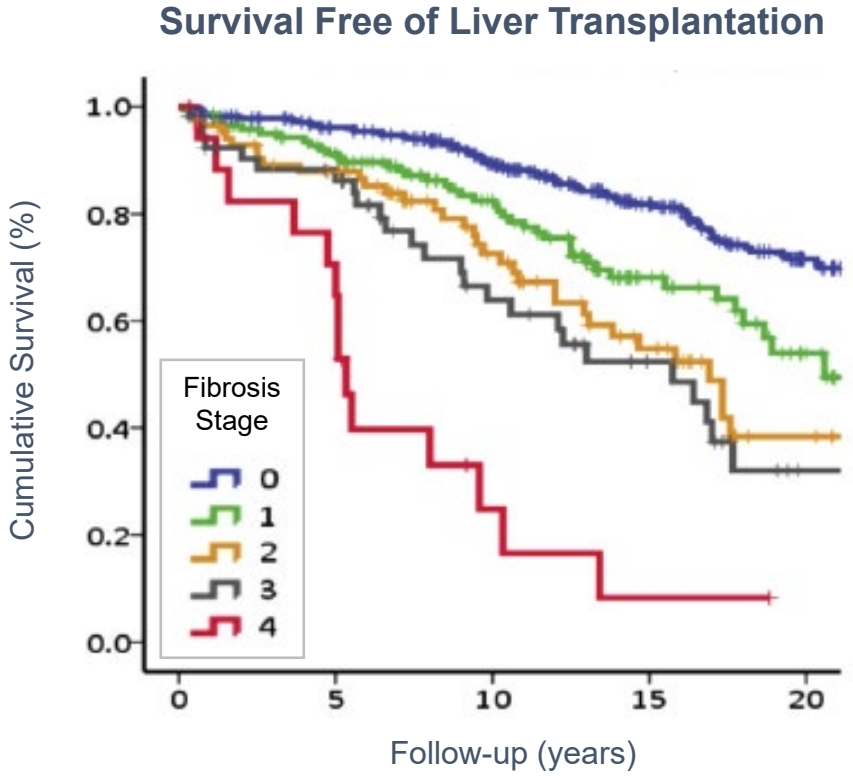
Cirrhotic NASH Patients Have Poor Prognosis

Liver-related mortality rate increases substantially from F3 to F4 fibrosis



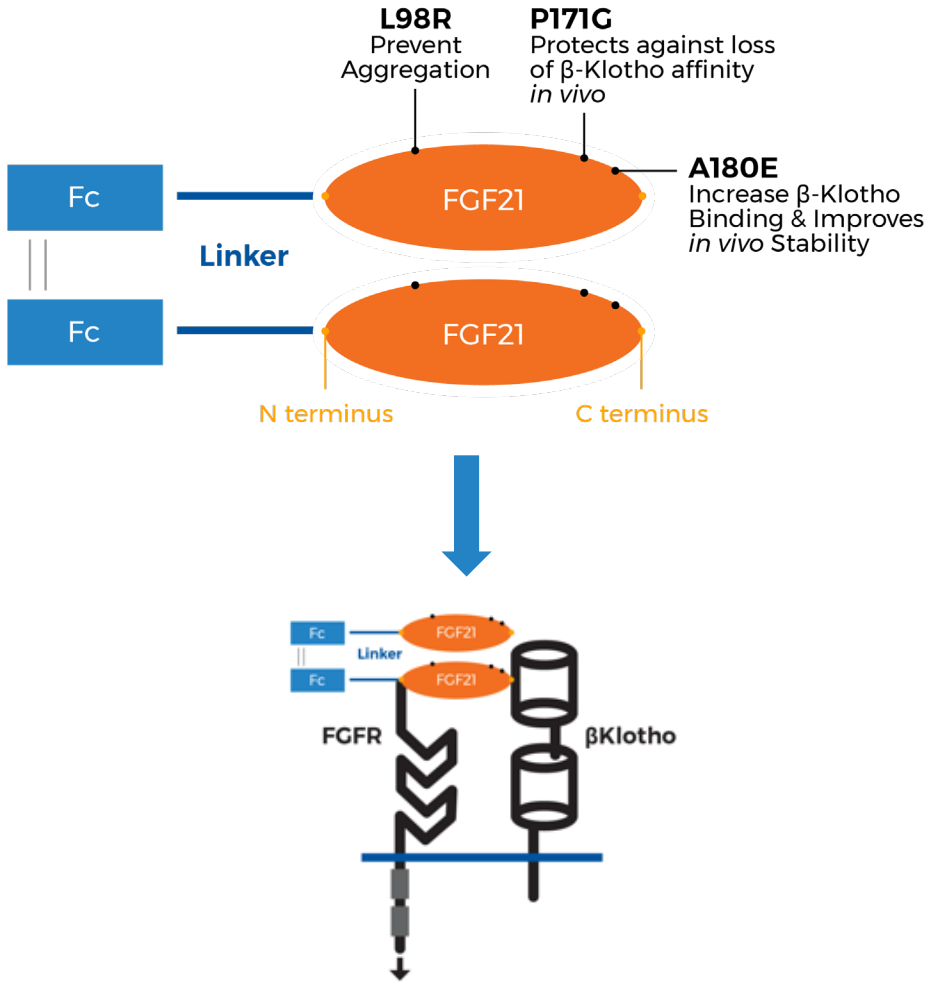
Dulai, PS et al. (2017) Hepatology 65:1557-65

~60% 5-year mortality for F4 NASH patients absent transplant



Angulo, P et al. (2015) Gastroenterology 149:389-397

EFX Engineering Potentially Optimal for NASH Efficacy, With Convenient Once-weekly Dosing



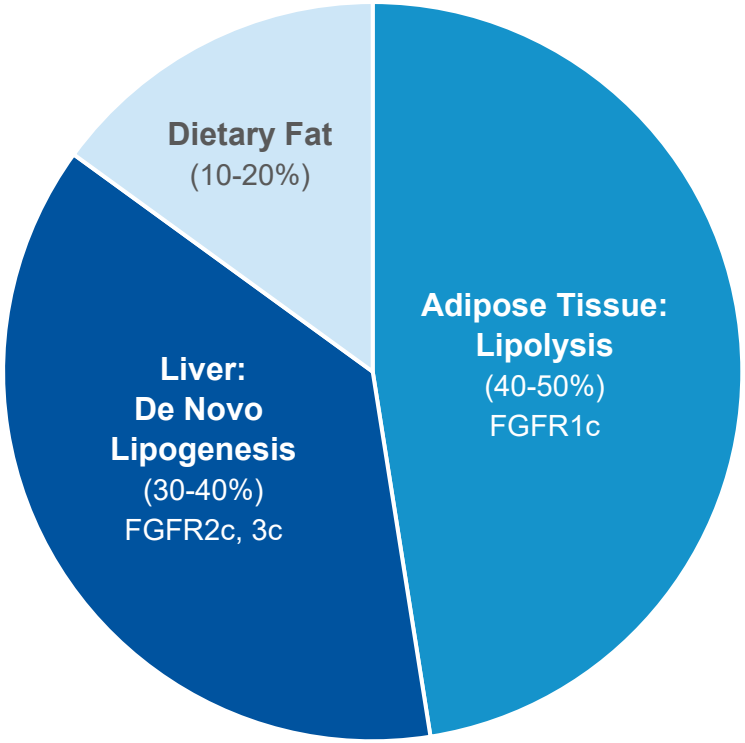
Key attributes

- 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



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

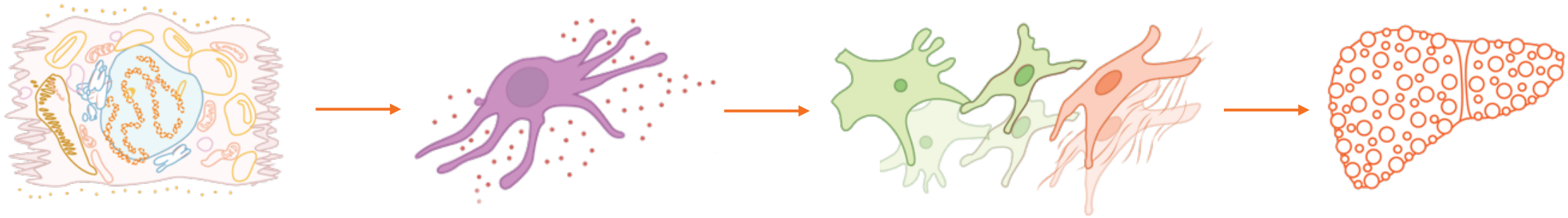
Pathogenesis of Fibrosis

Hepatocyte Stress & Death

Kupffer Cell Activation

Myofibroblast Differentiation

Fibrogenesis

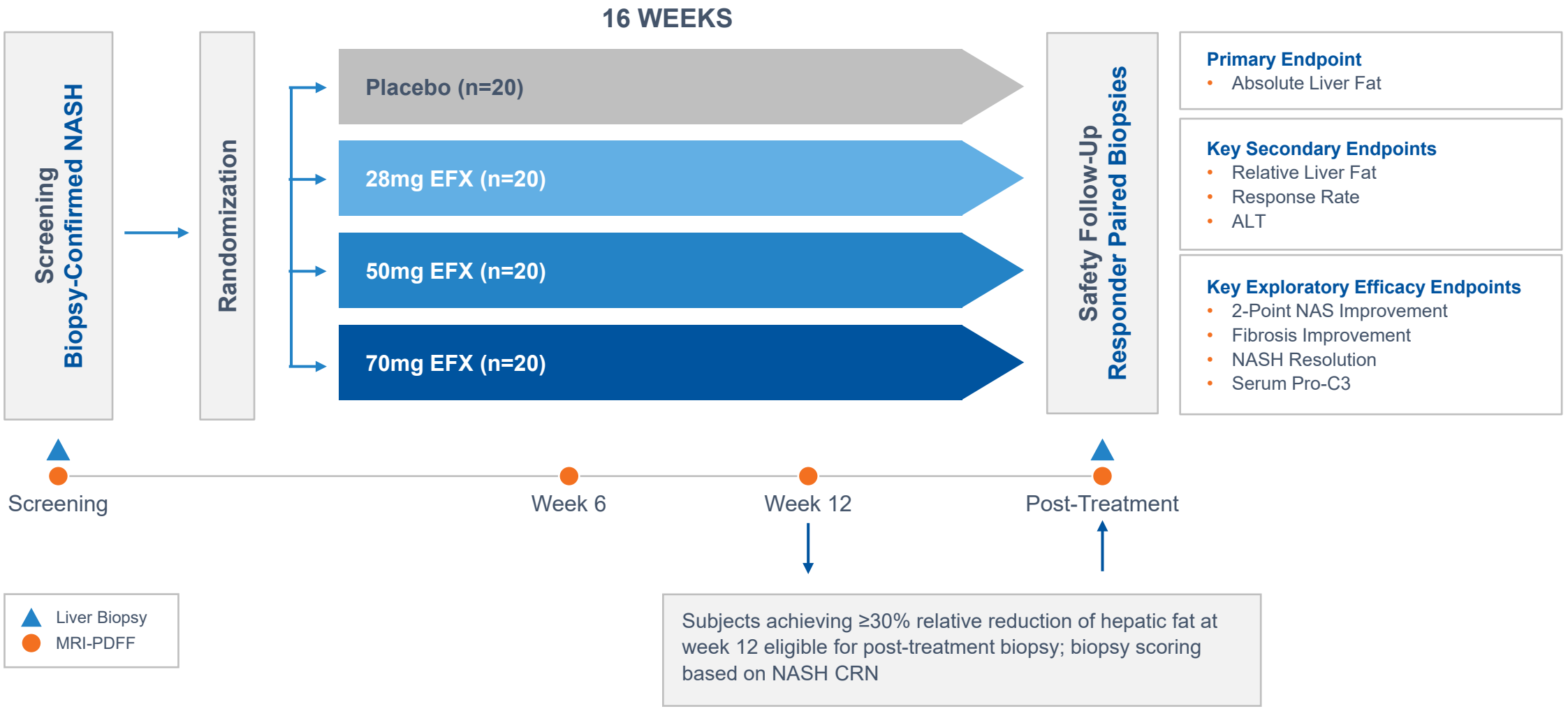


EFX Anti-Fibrotic Activity

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*

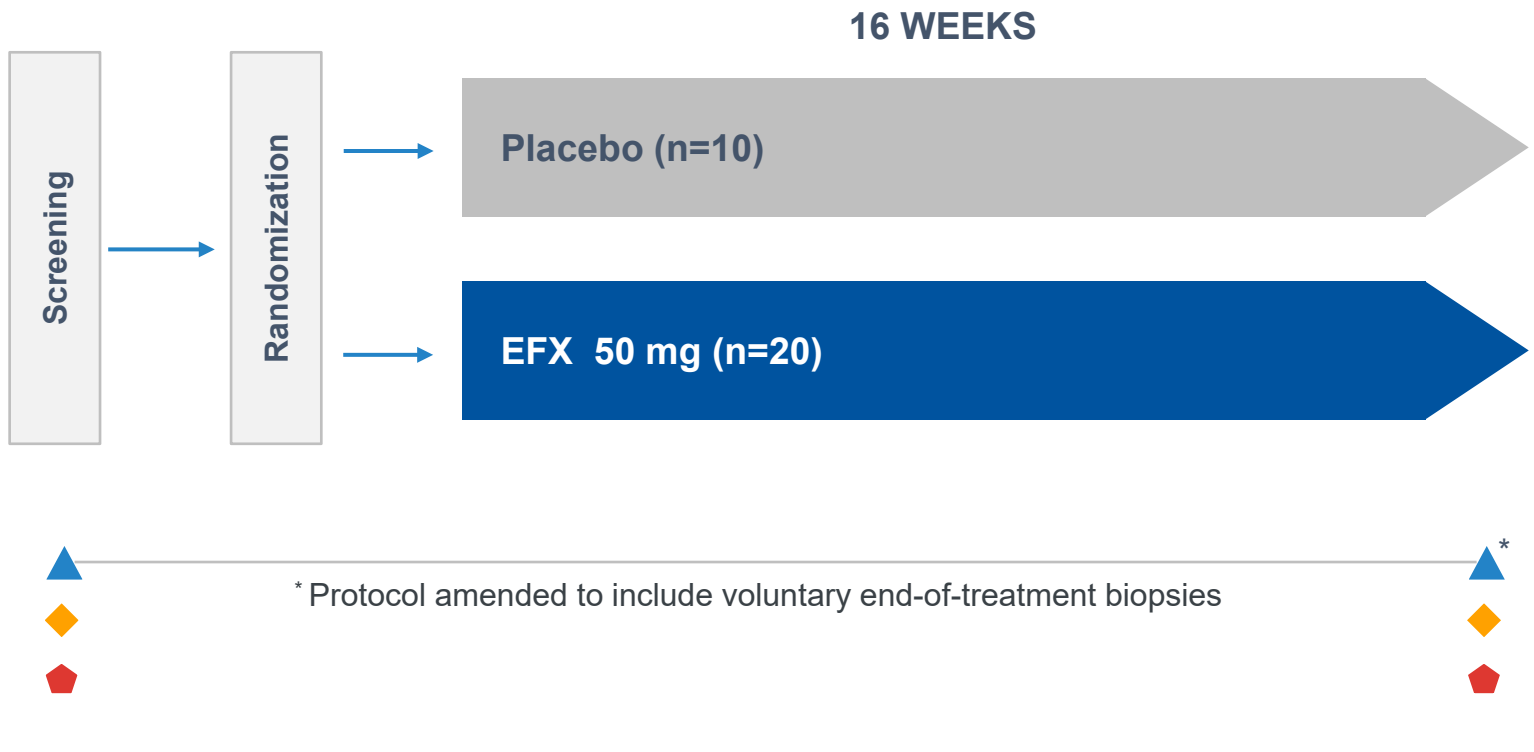
Phase 2a Trial (Balanced) Design (F1-F3 NASH)



Phase 2a Expansion Cohort C Trial Design (F4 NASH)

BALANCED study included an expansion cohort, Cohort C, of patients with compensated cirrhosis (F4), Child-Pugh Class A

Primary Endpoint
Safety & tolerability

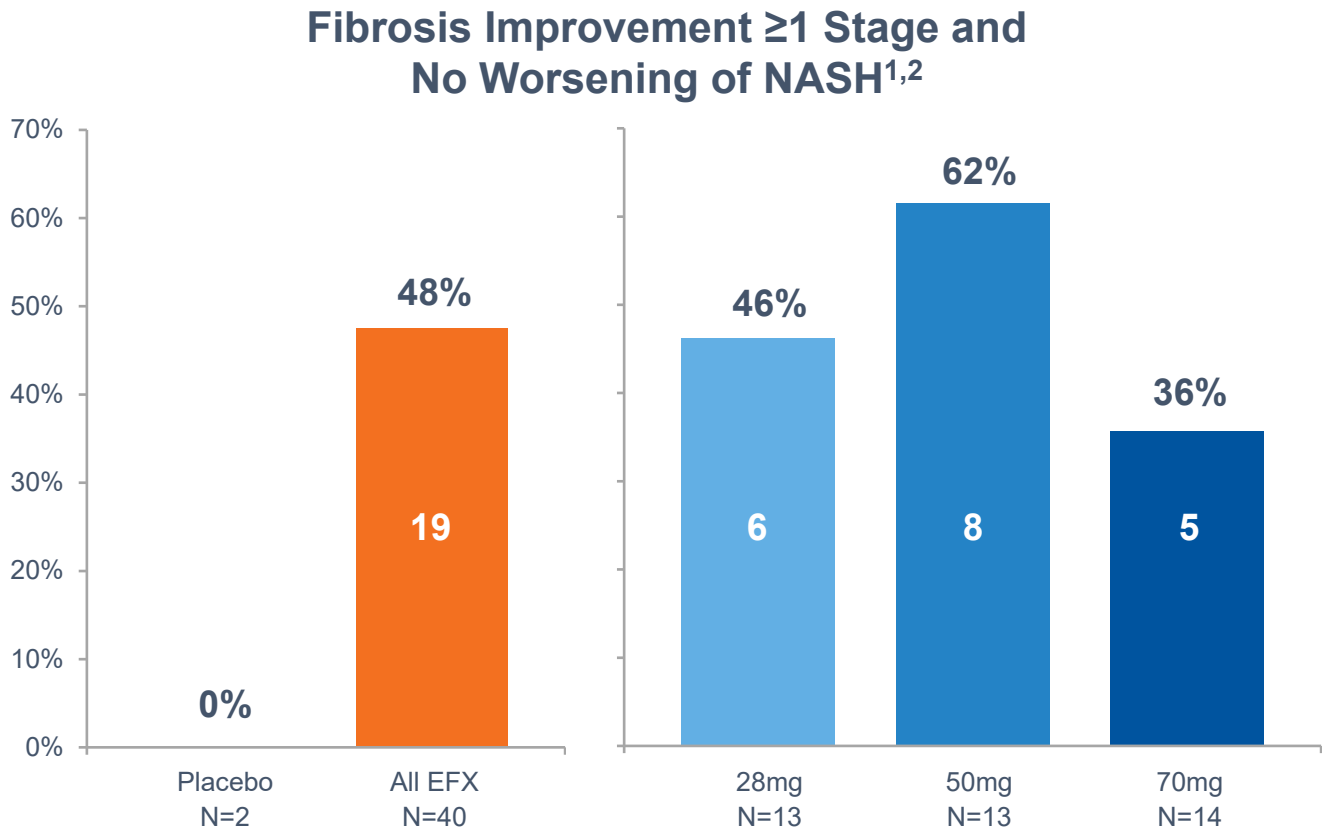


Baseline Demographics: Main Study & Cohort C

	BALANCED Main Study[a]				Cohort C[b]	
Parameter Mean	**Placebo (N=21)**	**EFX 28mg (N=19)**	**EFX 50mg (N=20)**	**EFX 70mg (N=20)**	**Placebo (N=10)**	**EFX 50mg (N=20)**
Age (Years)	52	50	53	53	57.1	61.1
Sex (Male/Female)	6/15	9/10	10/10	9/11	7/3	4/16
Weight (kg)	99.6	108.2	103.6	103.1	119.1	97.9
NAFLD Activity Score (NAS) (range)	5.1 (4 to 7)	5.6 (4 to 7)	5.1 (3 to 7)	5.6 (5 to 7)	3.4[c] (1 to 6)	4.2[c] (1 to 7)
Alanine Aminotransferase (ALT) (U/L)	50.7	62.5	53.4	56.8	32.7	31.7
Aspartate Aminotransferase (AST) (U/L)	38.6	41.1	35.4	44.6	28.9	31.4
% Type 2 Diabetes	67	37	50	50	50	50
HbA1c (%)	6.5	6.2	6.4	6.2	6.5	6.1
Triglycerides (mg/dL)	208	176	177	180	122	135
ELF Score	9.4	9.5	9.5	9.6	9.7	10.4
Pro-C3 (µg/L)	16.1	19.2	16.2	17.2	22.6	25.6
Liver Stiffness (kPA)	11.9	12.5	11.3	12.4	25.8	22.1

[a] Full Analysis Set, F1-F3 (all subjects randomized into the BALANCED main study); b Full Analysis Set, F4 (all subjects randomized into BALANCED Cohort C [except where otherwise noted]); c Liver Biopsy Analysis Set, F4 (all Cohort C subjects confirmed by central reader as F4 at baseline with Week 16 liver biopsy results)

High Rates of Fibrosis Improvement After 16 Weeks Across All Dose Groups (F1-F3 NASH)



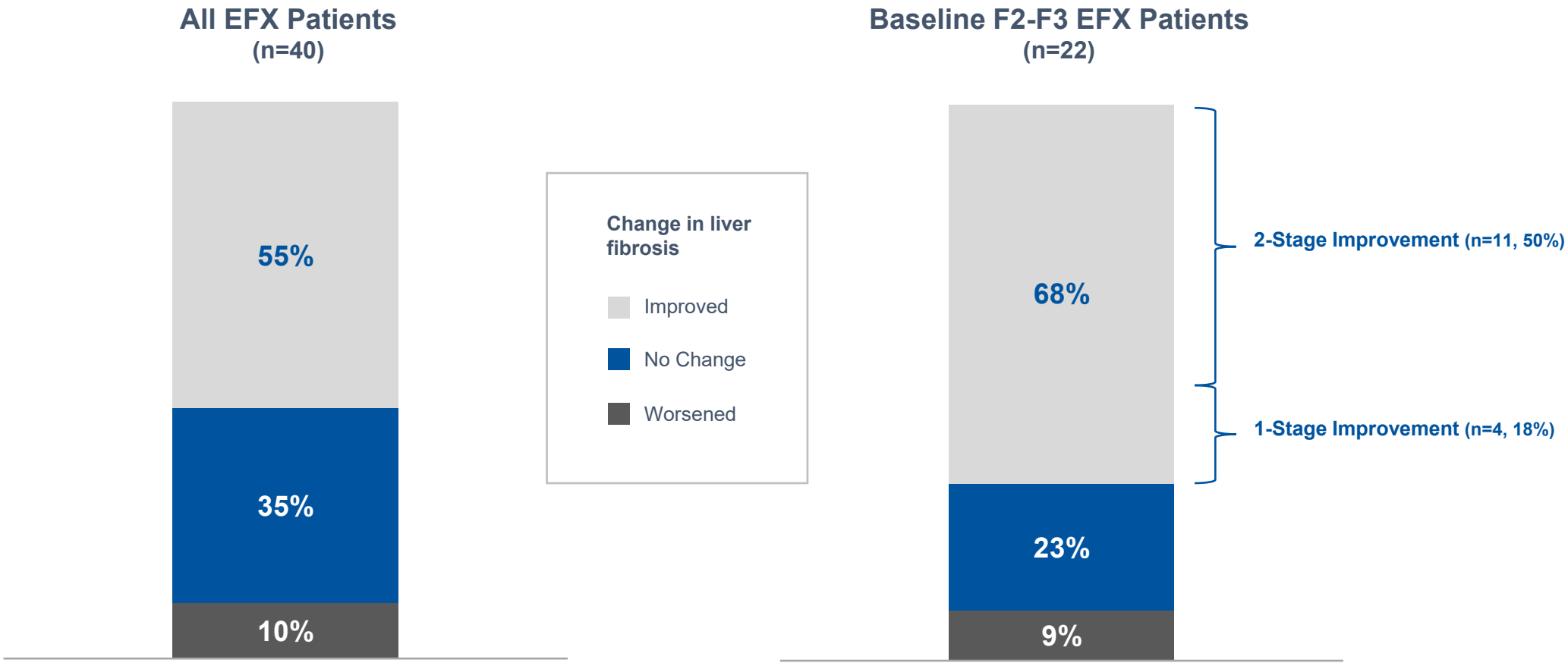
[1] Improvement in liver fibrosis greater than or equal to one stage and no worsening of NASH (defined as no increase in NAS for ballooning, inflammation, or steatosis)
[2] Secondary and exploratory histological endpoints were not powered for statistical significance

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

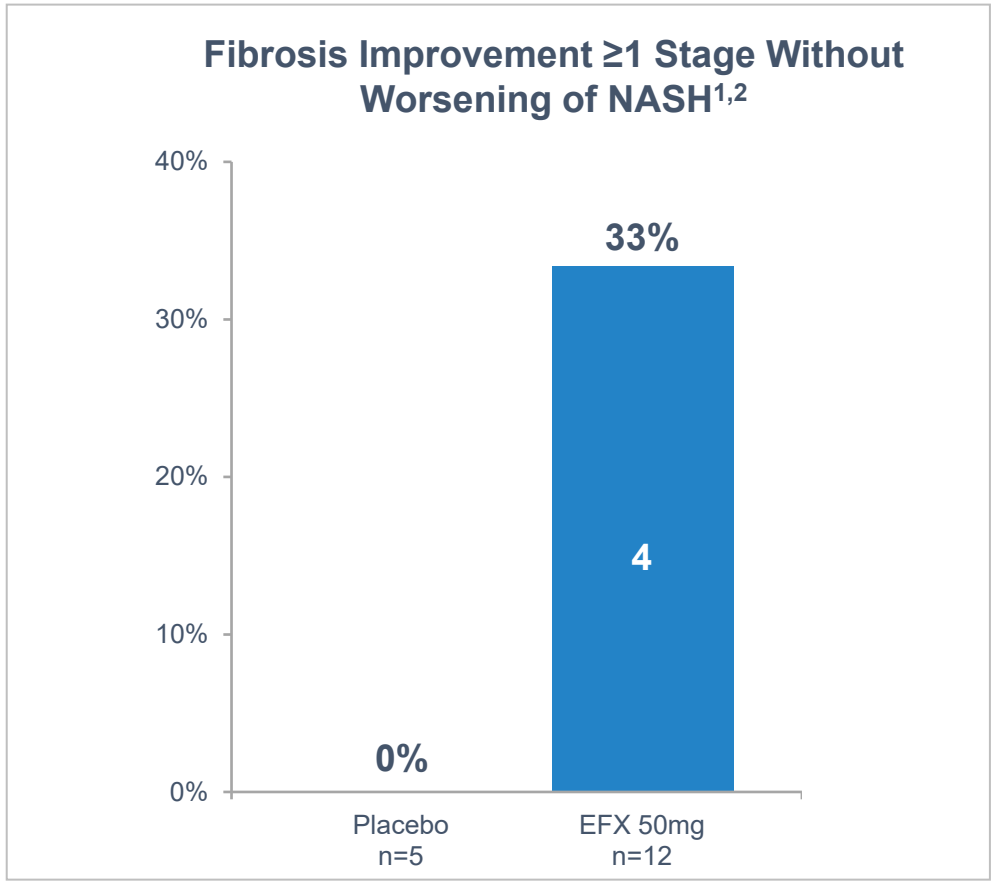
Source Data: Liver Biopsy Evaluable Analysis Set, F1-F3 (all BALANCED main study responders who had baseline and end-of-treatment liver biopsy results)

Half of F2-F3 Patients Achieved 2-Stage Fibrosis Improvement



Source Data: Liver Biopsy Evaluable Analysis Set, F1-F3

High Rate of Fibrosis Improvement After Only16 Weeks Among Cirrhotic Patients (F4 NASH)



Biopsy Reading

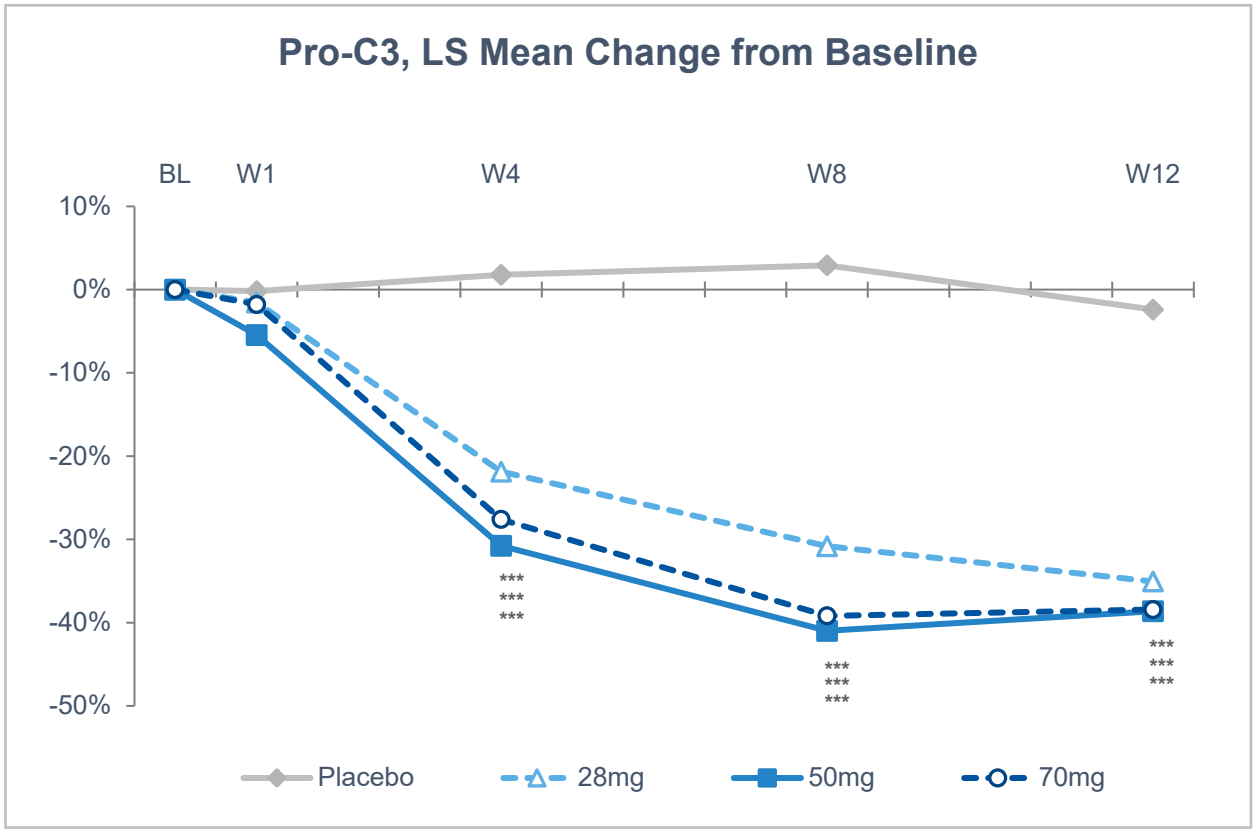
- All baseline and end-of-treatment biopsies were read by a single NASH-CRN pathologist
- All biopsies were read blinded to both treatment assignment and patient
- Baseline biopsies were read independently of end-of-treatment biopsies, in random fashion and not paired

Baseline Biopsy Timing

- Mean time from historical biopsy to patient screening = 6 months

[1] No increase in NAS for ballooning, inflammation, or steatosis; [2] Study not powered to assess statistical significance of changes in histological endpoints
Source Data: Liver Biopsy Analysis Set, F4; *Topline preliminary data;*

Rapid Improvements in Fibrosis Biomarkers Consistent with Histological Improvements (F1-F3 NASH)



*** 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

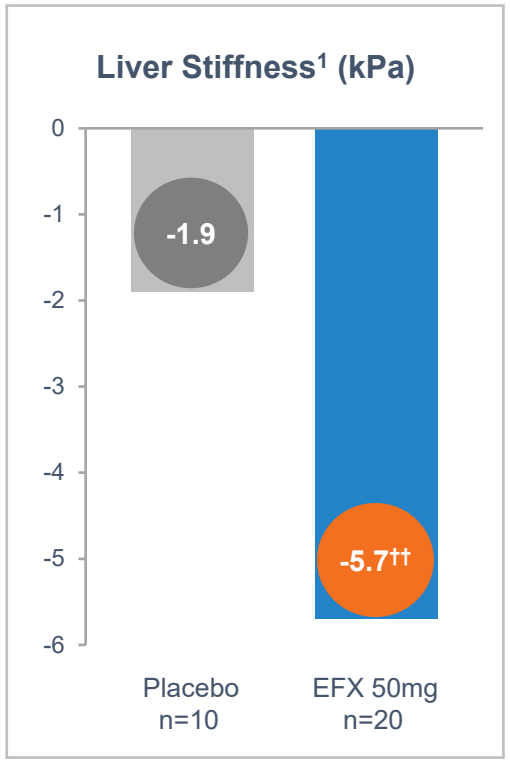
Dose Group	Baseline	Δ Week 12
Placebo	9.4	0.0
28mg	9.5	-0.7***
50mg	9.5	-0.8***
70mg	9.6	-0.4*

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

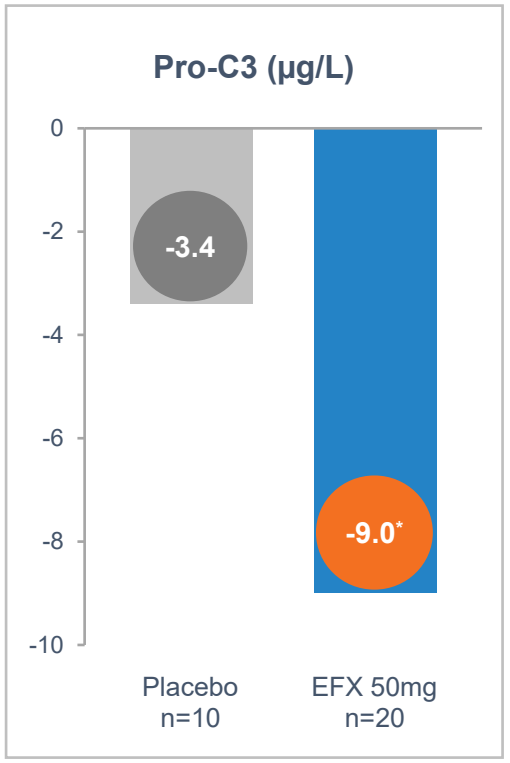
Source Data: Full Analysis Set, F1-F3

Improvements in Fibrosis Biomarkers in Cirrhotic NASH Patients Support Histology Results (F4 NASH)

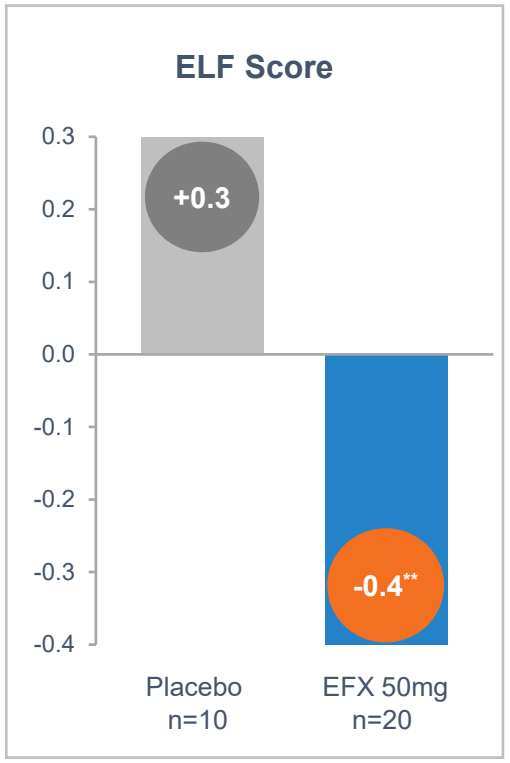
LS Mean Change From Baseline to Week 16



[1] Measured by FibroScan
†† p<0.01, versus baseline (ANCOVA)



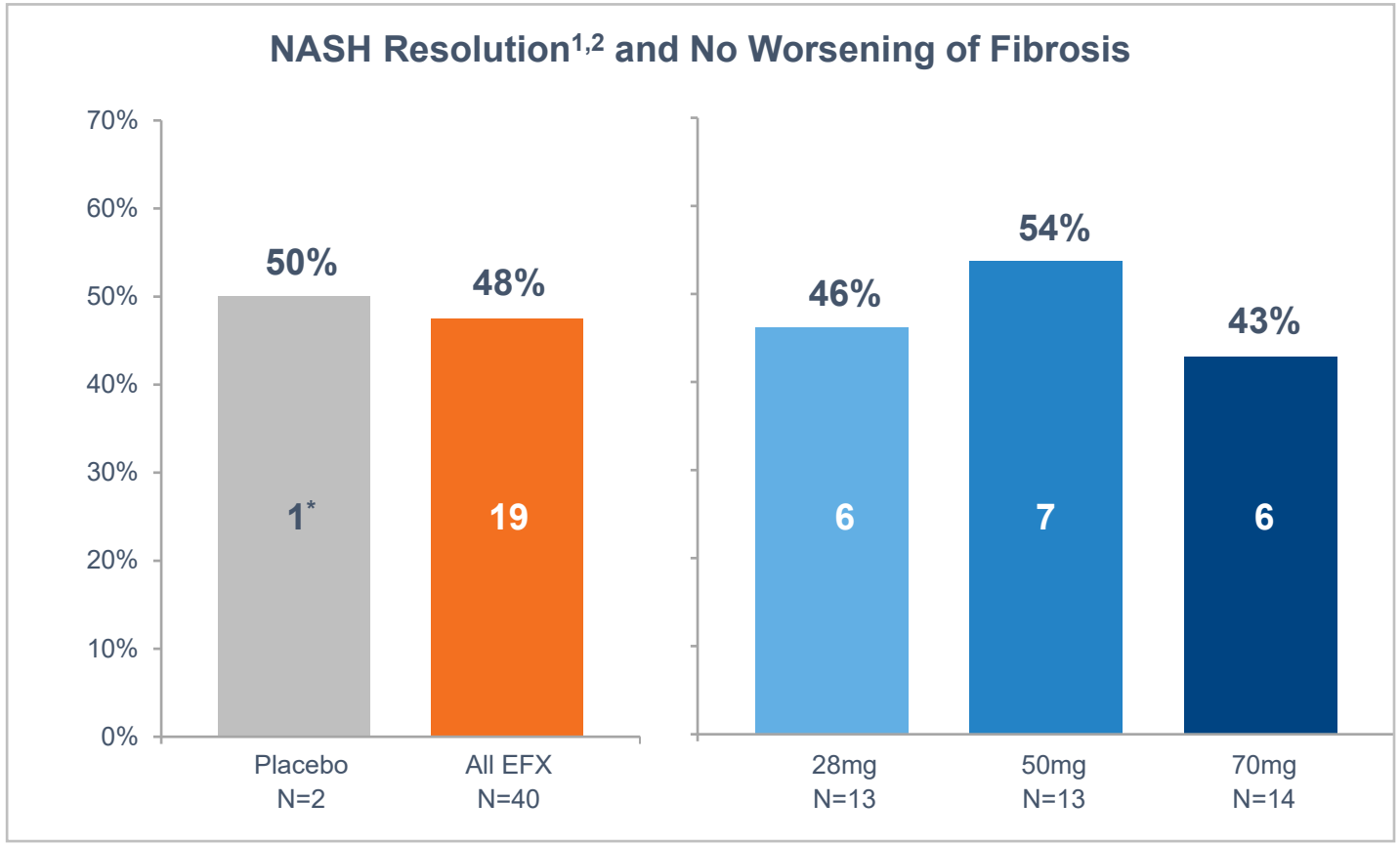
* p<0.05, versus placebo (ANCOVA)



** p<0.01, versus placebo (ANCOVA)

Source Data: Biomarker Analysis Set, F4 (all Cohort C subjects with baseline and interpretable on-study measure of ELF or pro-C3, respectively); Liver Stiffness Analysis Set, F4 (all Cohort C subjects with baseline and interpretable on-study measure of Liver Stiffness); Topline preliminary data

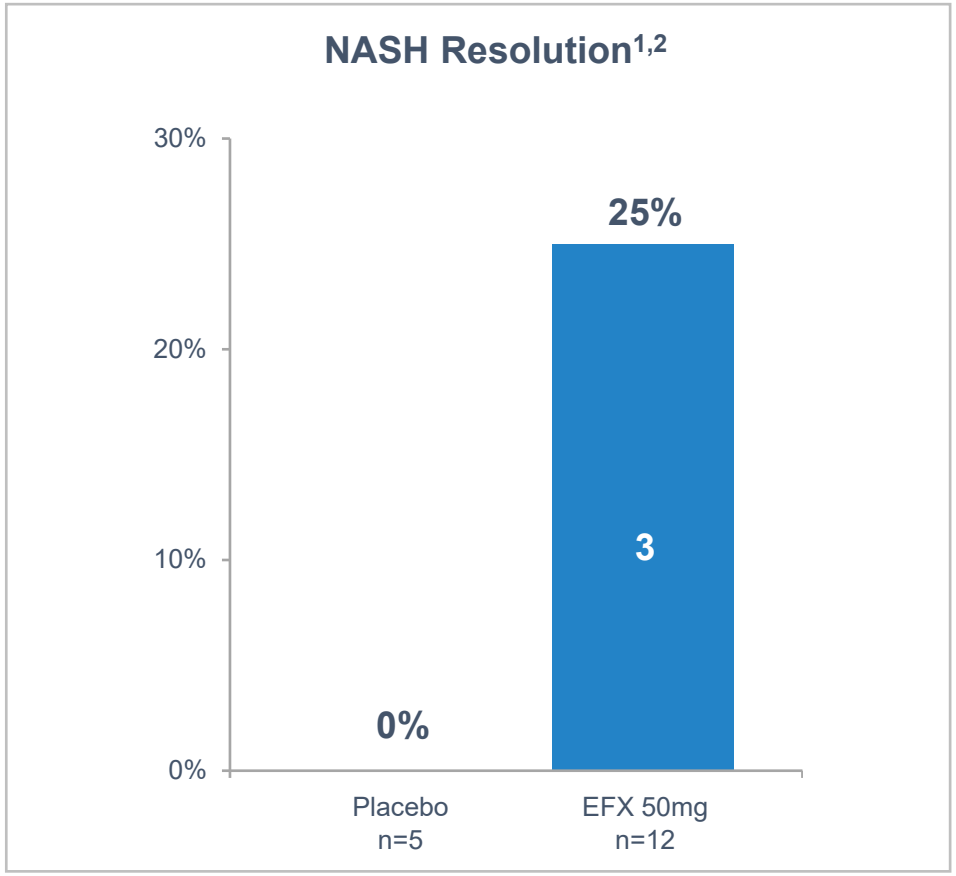
High Response Rates on NASH Resolution After 16 Weeks Across All Dose Groups (F1-F3 NASH)



[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, F1-F3

NASH Resolution Also Observed in Cirrhotic Patients (F4 NASH)



[1] NAS score of 0 or 1 for lobular inflammation and a score of 0 for ballooning
[2] Study not powered to assess statistical significance of histological endpoints

Change in NAS among Subjects Achieving NASH Resolution

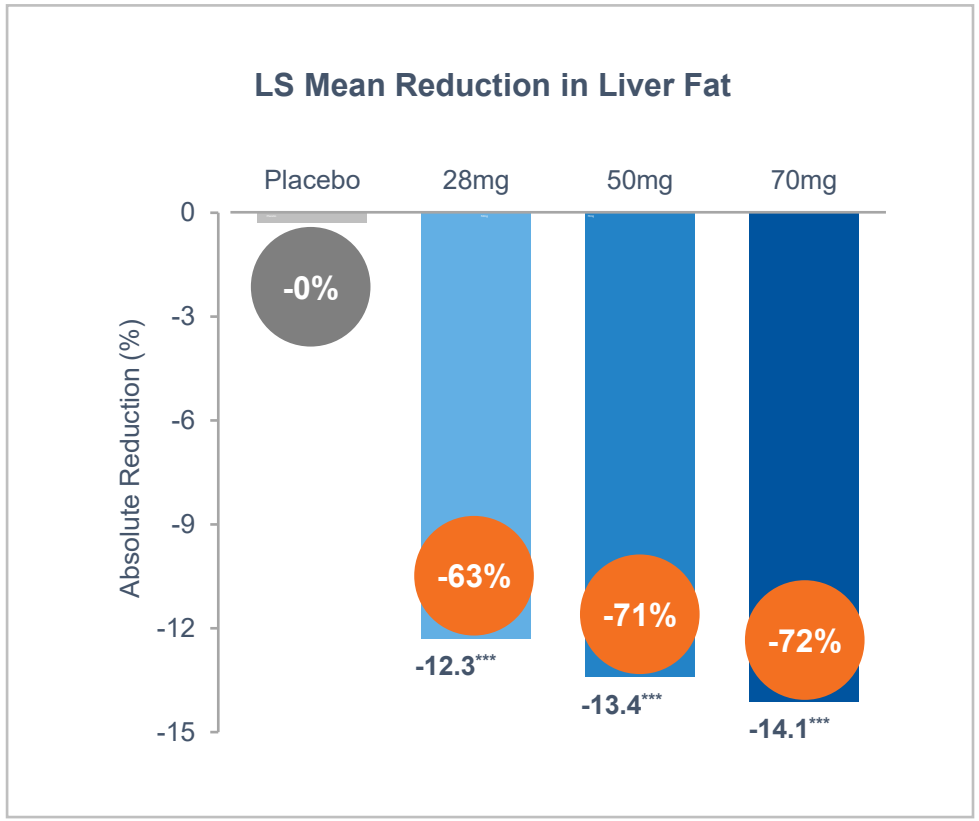
EFX Subject	Baseline NAS	Week 16 NAS
A	7	1
B	3	1
C	6	1

Proportion of Subjects with ≥2 Point NAS Reduction

Placebo	EFX 50mg
1 (20%)	7 (58%)

Source Data: Liver Biopsy Analysis Set; *Topline preliminary data*

Substantial Reductions in Liver Fat at Week 12 Across All Dose Groups (F1-F3 NASH)



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

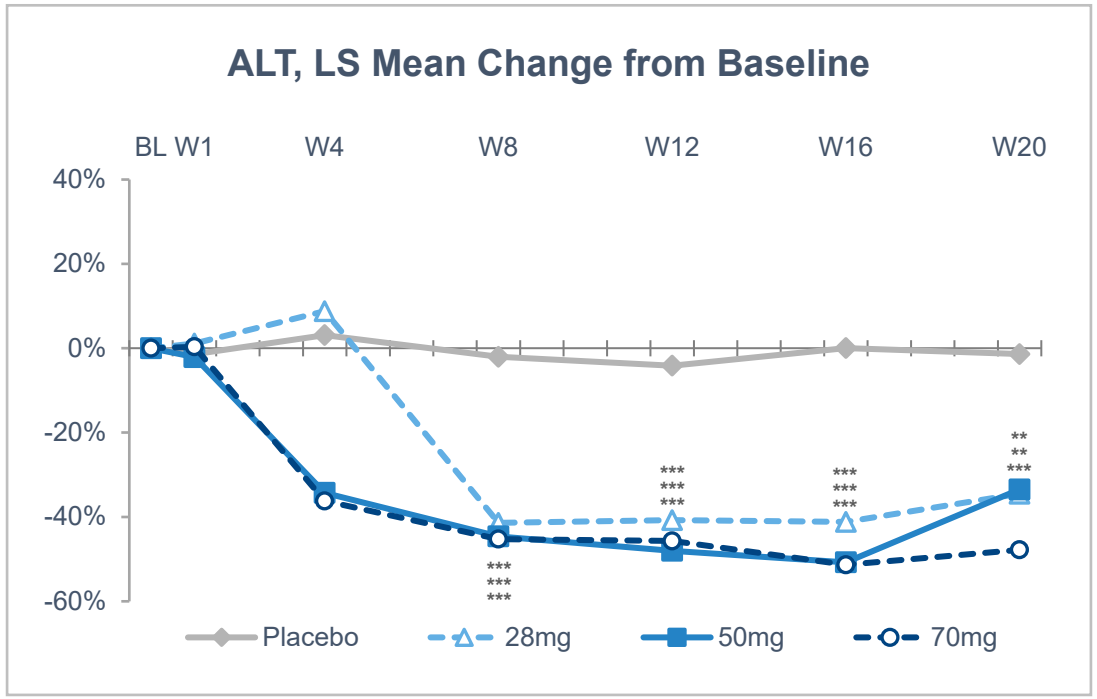
Proportion of Patients Achieving Fat Reduction Thresholds

Endpoint	Placebo (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%***

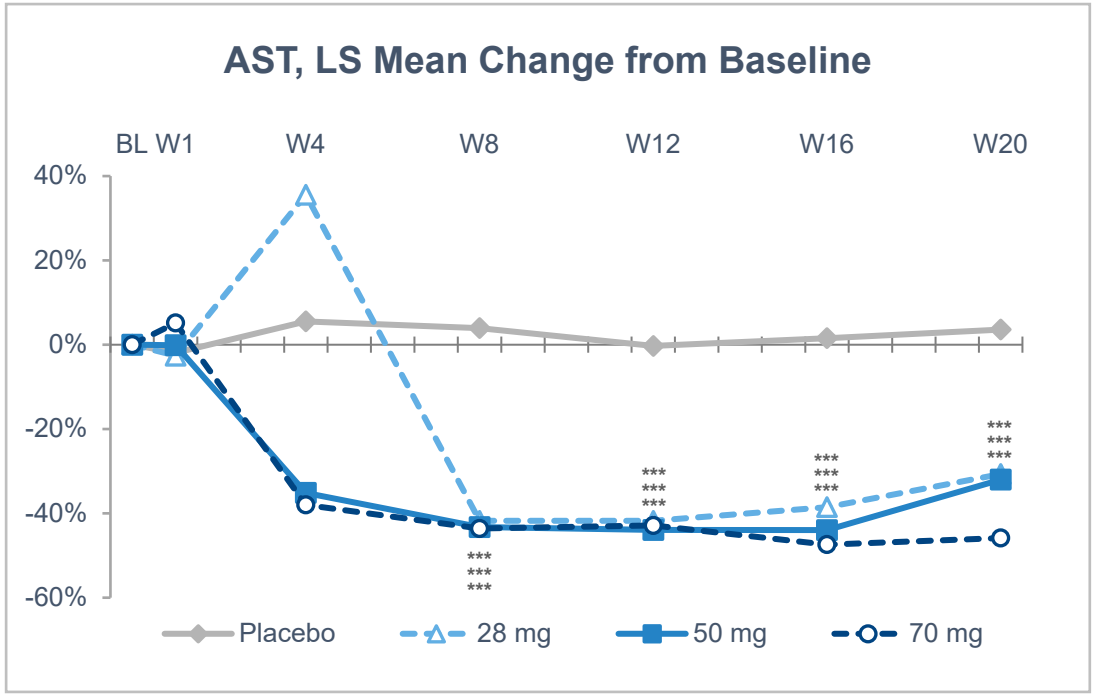
* p<0.05, ** p<0.01, *** p<0.001, versus placebo (ANCOVA)

Source Data: Full Analysis Set, F1-F3

Substantial Reductions in Markers of Liver Injury After 16 Weeks of Treatment (F1-F3 NASH)



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

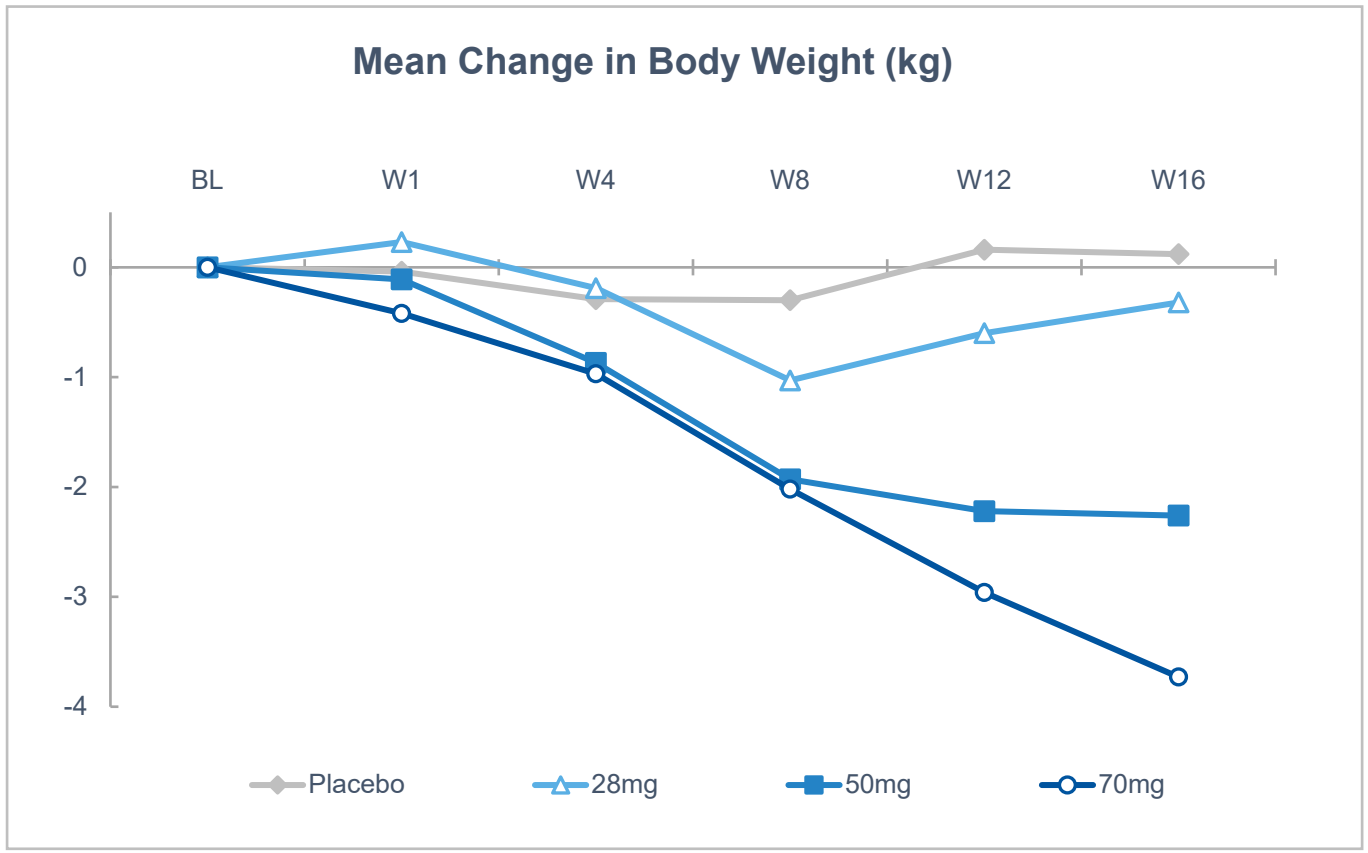


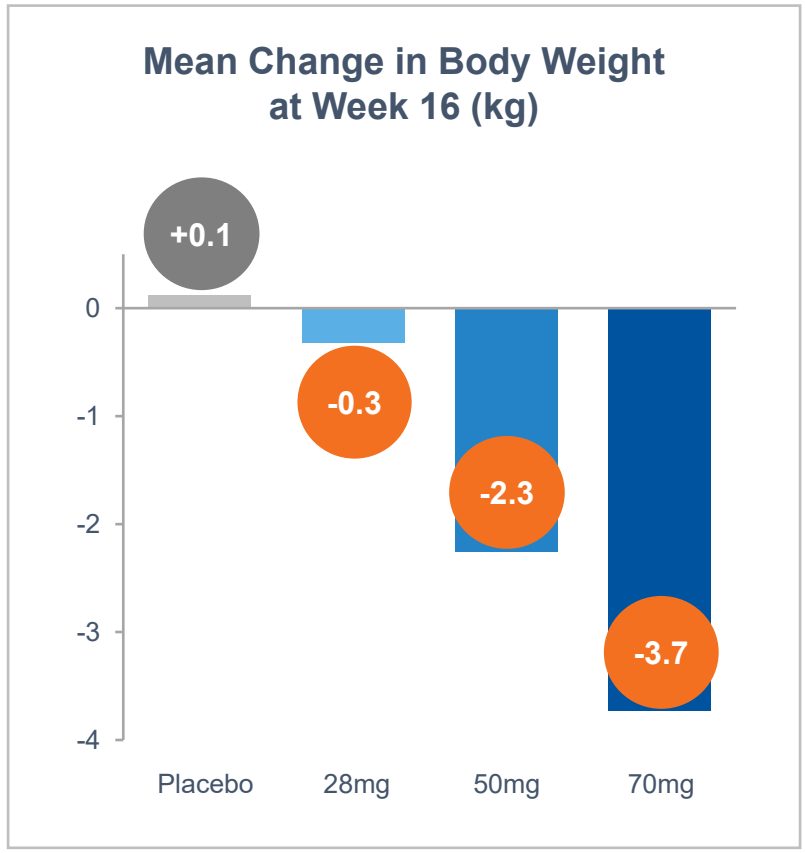
*** p<0.001, versus placebo (MMRM)

Similar dose-related improvements observed for GGT & ALP

Source Data: Full Analysis Set, F1-F3

Weight Loss Observed For All Dose Groups (F1-F3 NASH)

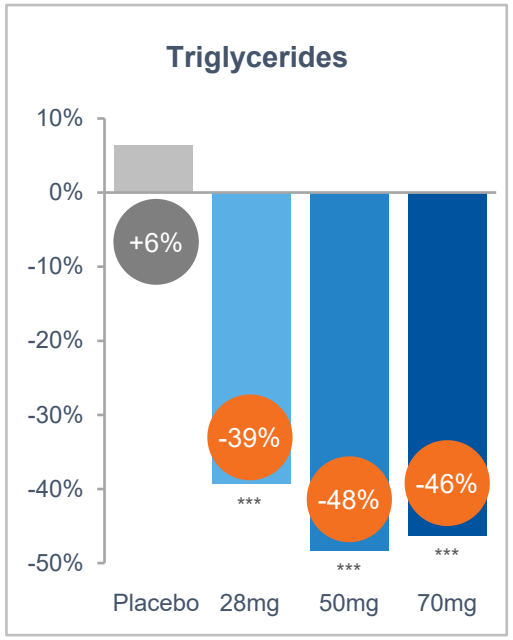




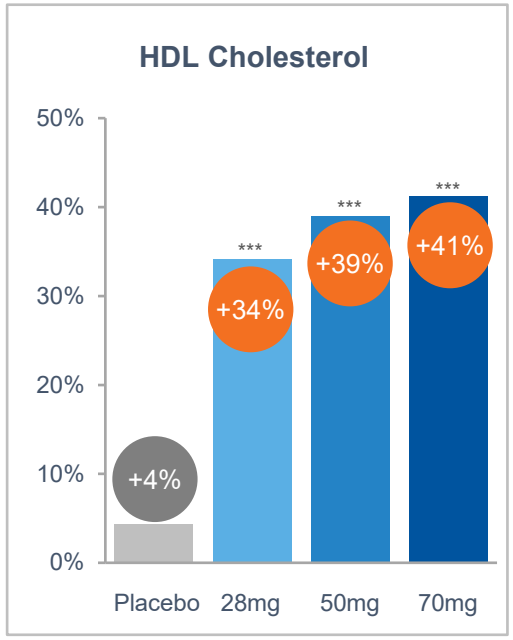
Source Data: Full Analysis Set, F1-F3

Improved Lipoprotein Profile (F1-F3 NASH)

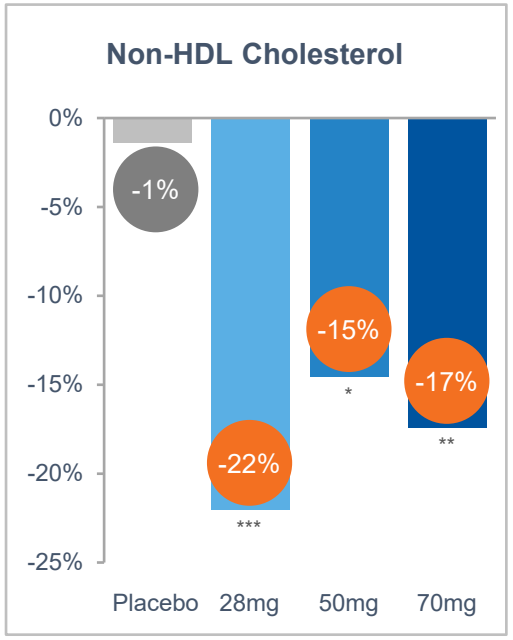
LS Mean Change From Baseline to Week 16 (%)



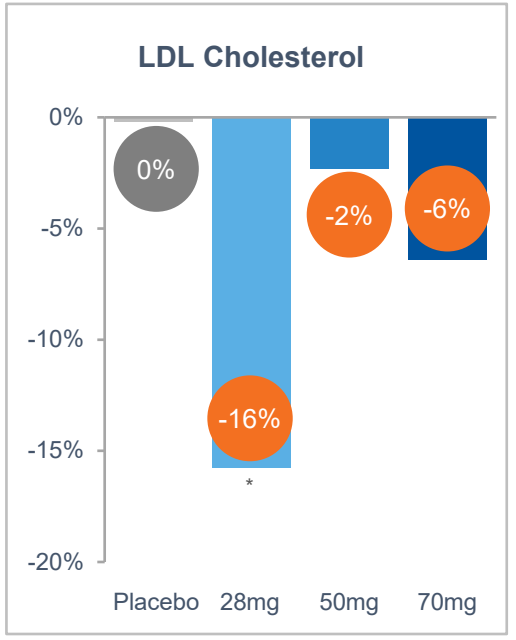
*** p<0.001, versus placebo (ANCOVA)



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



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



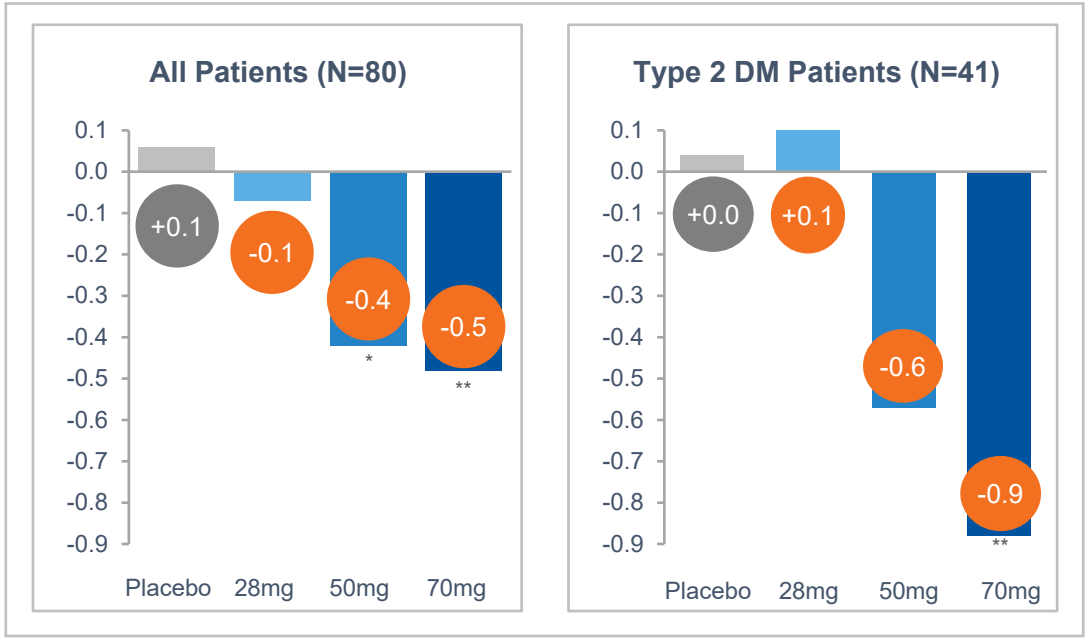
* p<0.05, versus placebo (ANCOVA)

Source Data: Full Analysis Set, F1-F3

Clinically Meaningful Improvements in Glycemic Control After 16 Weeks (F1-F3 NASH)

LS Mean Change From Baseline to Week 16 (%)

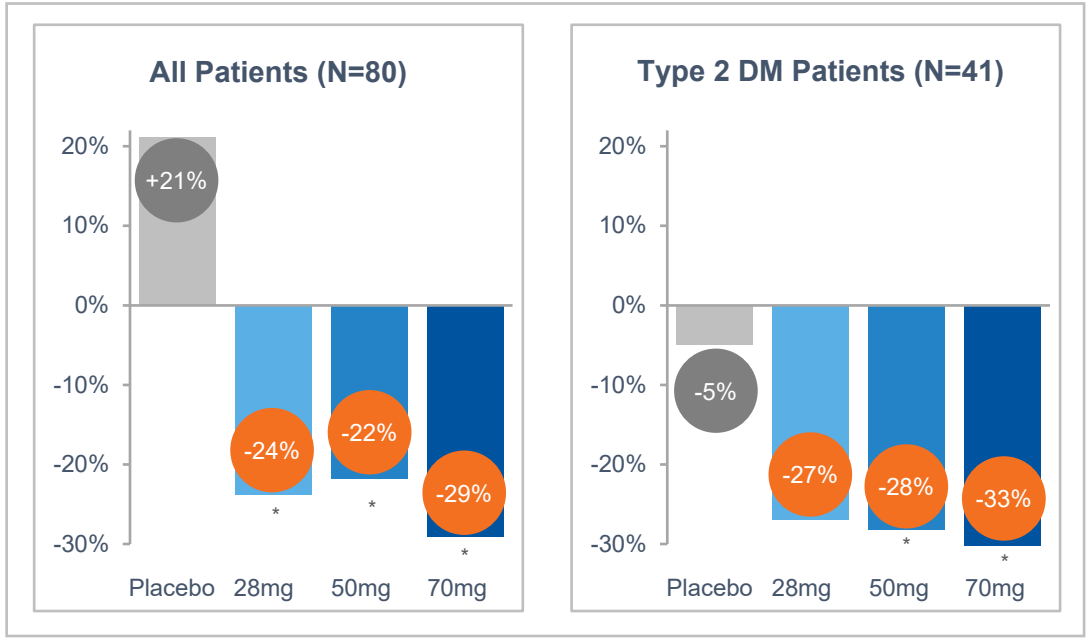
HbA1c[1]



[1] Absolute change from baseline, %

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

C-Peptide[2]



[2] Relative percent change from baseline

* p<0.05, versus placebo (ANCOVA)

Source Data: Full Analysis Set, F1-F3

Interpreting the Rapid Reversal of Fibrosis Observed in NASH Patients Treated with EFX

- Fibrosis reversal in cirrhotic patients (F4), two-stage improvement of fibrosis in F2/F3 patients, and corroborating non-invasive markers of fibrosis improvement likely reflects direct anti-fibrotic activity
- Fibrosis reversal is especially advantageous for cirrhotic patients who face high risk of mortality and severe morbidity

- Addressing underlying NASH disease drivers may indirectly contribute to fibrosis reversal for F1-F3 patients with adequate time for the liver to regenerate
- Redress of the underlying NASH disease drivers is necessary to sustain fibrosis reversal in F1-F4
- Supports broader metabolic improvements

Drug-related Treatment-Emergent Adverse Events (TEAE) (F1-F3 NASH)

Most Frequent (>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%)	9 (45%)
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, F1-F3 (all BALANCED main study subjects who received at least one dose of study drug

Tolerability Overview (F4 NASH)

Most Frequent (>15%) Drug-Related AEs	Placebo (N=10)	EFX 50mg (N=17)
Diarrhea	1 (10%)	7 (41%)
Nausea	1 (10%)	5 (29%)
Injection site reaction	0	5 (29%)
Injection site erythema	0	4 (24%)
Headache	0	3 (18%)
TEAE/SAE Disposition	**Placebo**	**EFX 50mg**
Study Discontinuations	1[a]	1[b]
Serious Adverse Events (SAE)	1[c]	0
Deaths	0	0

[a] *Withdrawal of consent*
[b] *abdominal distension, constipation, diarrhea, pruritus*
[c] *pulmonary embolism*

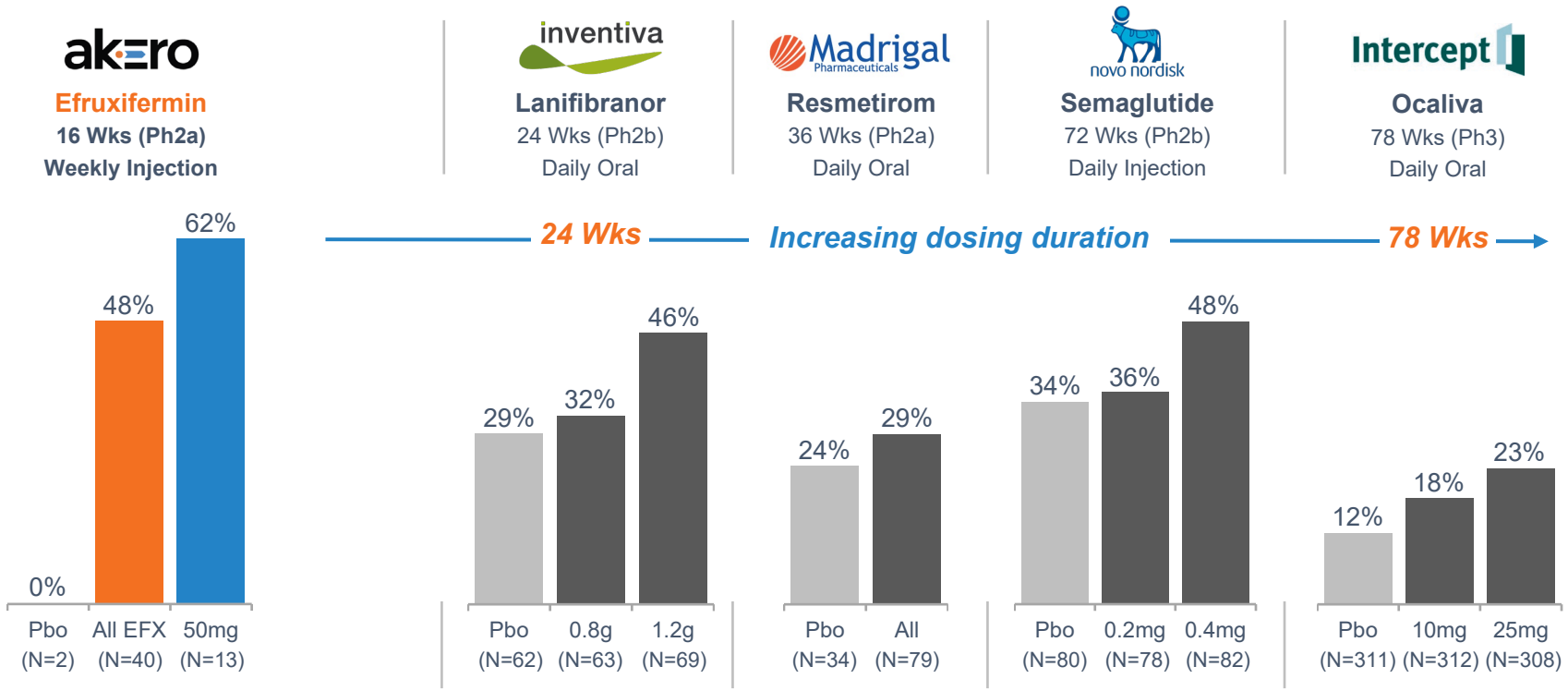
Key Observations

- Encouraging tolerability given population with more advanced disease
- All injection site AEs Grade 1
- No reports of tremor

Source Data: Safety Set, F4 (all Cohort C subjects confirmed by central reader as F4 at baseline who received at least one dose of study drug); *Topline preliminary data*

NASH Development Landscape: Fibrosis Improvement (F1-F3)

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



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)

EFX Anticipated Path to Phase 3: Parallel Phase 2b Trials

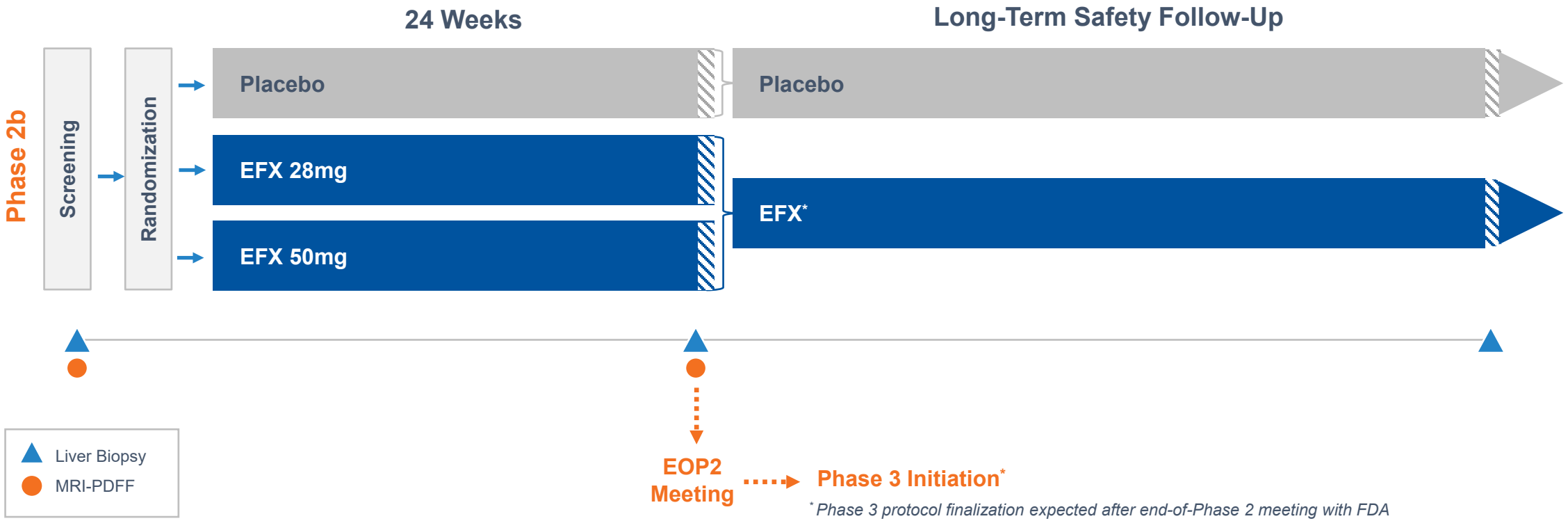
	BALANCED	Cohort C (Expansion of BALANCED)	HARMONY	SYMMETRY
Status	**Completed** (Readout Jun'20)	**Completed** (Readout Mar'21)	**Readout expected in 3Q'22**	**Initiated Jul'21**
Duration	16 Weeks	16 Weeks	24 Weeks	36 Weeks
EFX Arms	28, 50, 70mg	50mg	28, 50mg	28, 50mg
Placebo-Controlled	✓	✓	✓	✓

HARMONY Trial Design: Non-Cirrhotic NASH (F2/F3)

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
- MRI-PDFF
- Liver Injury Markers



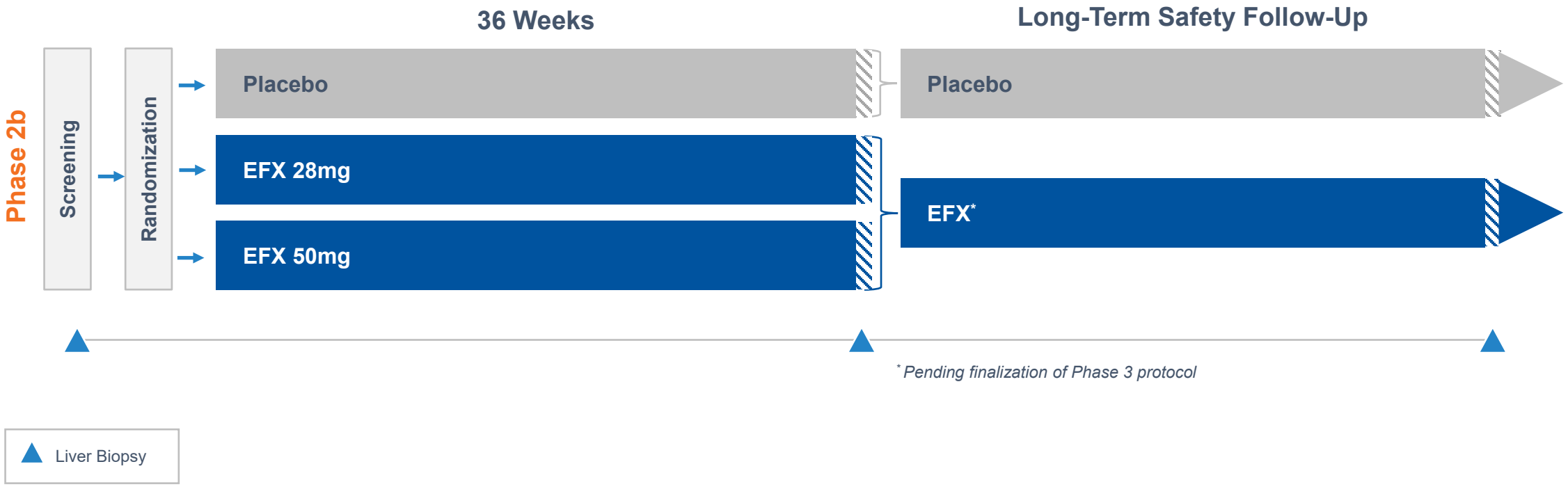
*Phase 3 protocol finalization expected after end-of-Phase 2 meeting with FDA

SYMMETRY Trial Design: Cirrhotic NASH (F4)

Key Inclusion Criteria
- F4 NASH

Phase 2b Primary Endpoint
- Fibrosis Improvement

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



* *Pending finalization of Phase 3 protocol*

Strong Financial Position

akero

COMPLETED UPSIZED IPO
June 20, 2019

~$106M
Raised in aggregate gross proceeds

$16/share
Priced upsized IPO at top of marketing range

COMPLETED UPSIZED FOLLOW-ON OFFERING
July 10, 2020

~$216M
Raised in aggregate gross proceeds

$36/share
Priced upsized follow-on offering at top of marketing range

CASH[1] ON HAND
As of June 30, 2021

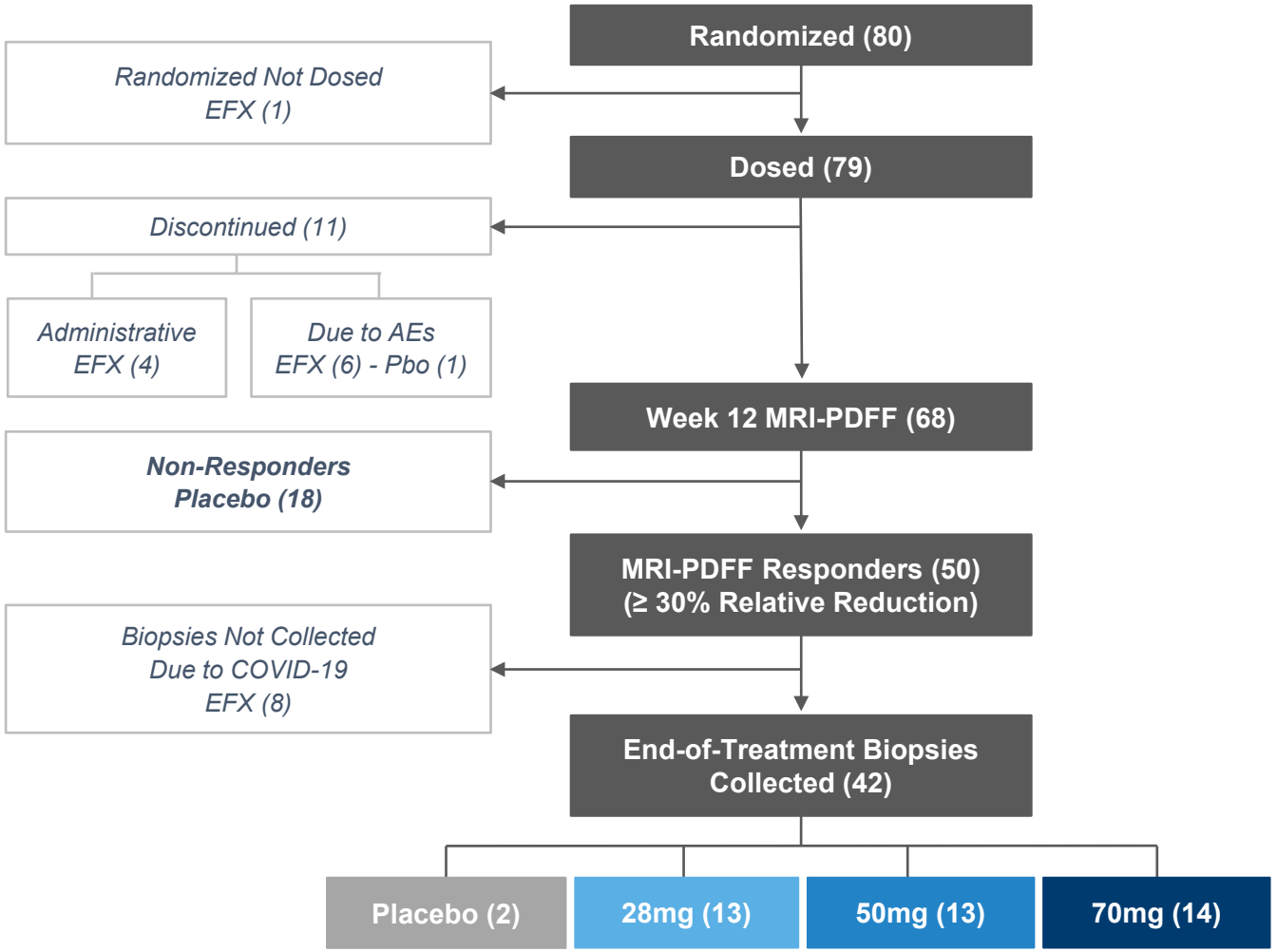
~$230M

[1] Cash, cash equivalents and short-term marketable securities

Current cash, cash equivalents and marketable securities are expected to be sufficient to fund current operating plan into the third quarter of 2023

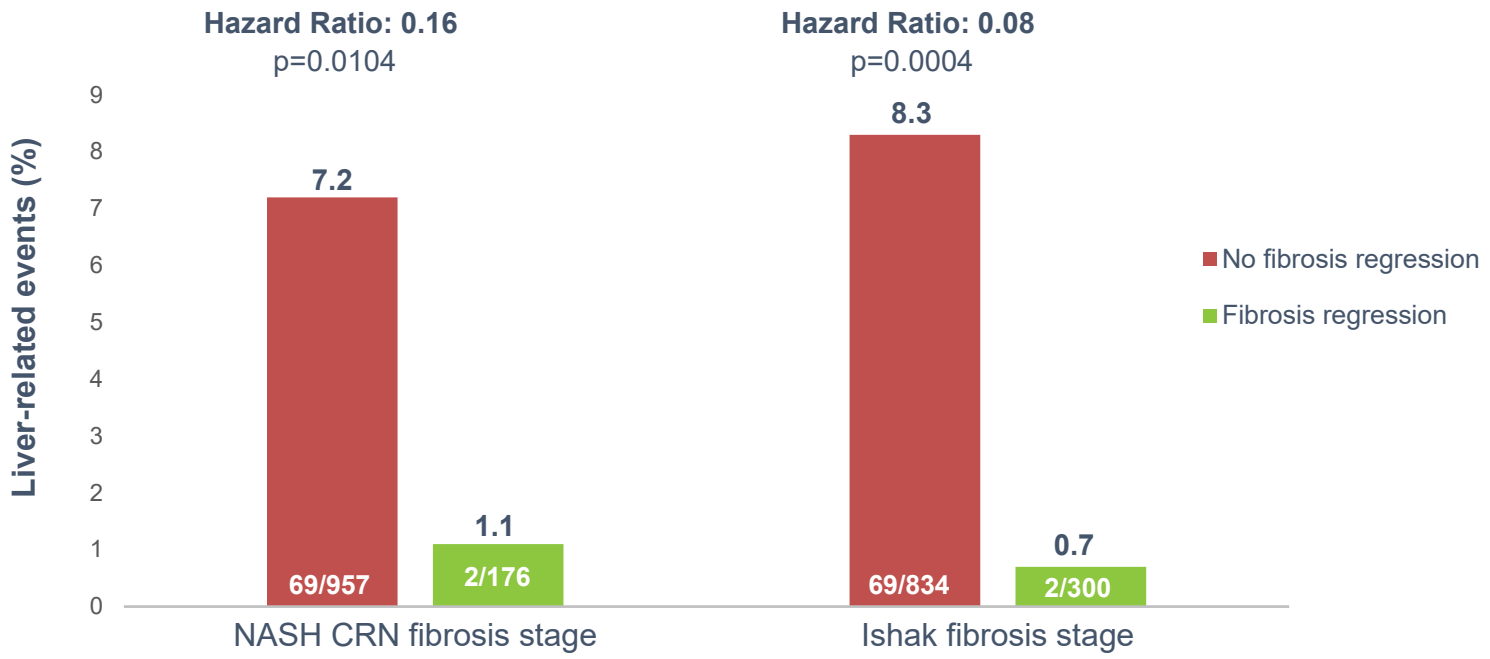
Backup Slides

Patient Disposition (Balanced Main Study)



Cirrhosis Regression is Associated with Improved Clinical Outcomes

Pooled treatment groups from STELLAR 4 and simtuzumab cirrhosis study



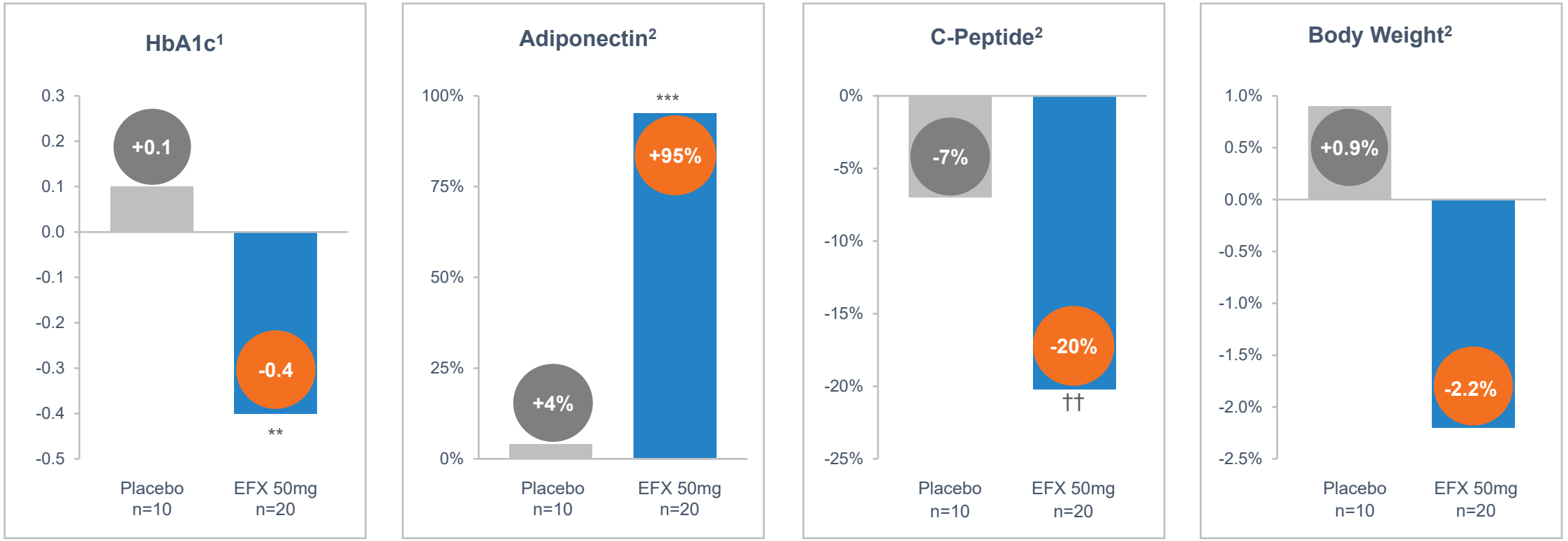
Cirrhosis regression observed in 16% of patients (treatment and placebo groups) over 48 weeks

Sanyal A, et al. AASLD TLMdX2020. #90

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.

Improved Glycemic Control; Trend Toward Weight Loss (F4 NASH)

LS Mean Change From Baseline to Week 16 (%)



[1] Absolute change from baseline, %
** p<0.01, versus placebo (ANCOVA)

[2] Relative percent change from baseline
*** p<0.001, versus placebo (ANCOVA)

[2] Relative percent change from baseline
†† p<0.01, versus baseline (ANCOVA)

[2] Relative percent change from baseline

Source Data: Full Analysis Set; *Topline preliminary data*

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

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

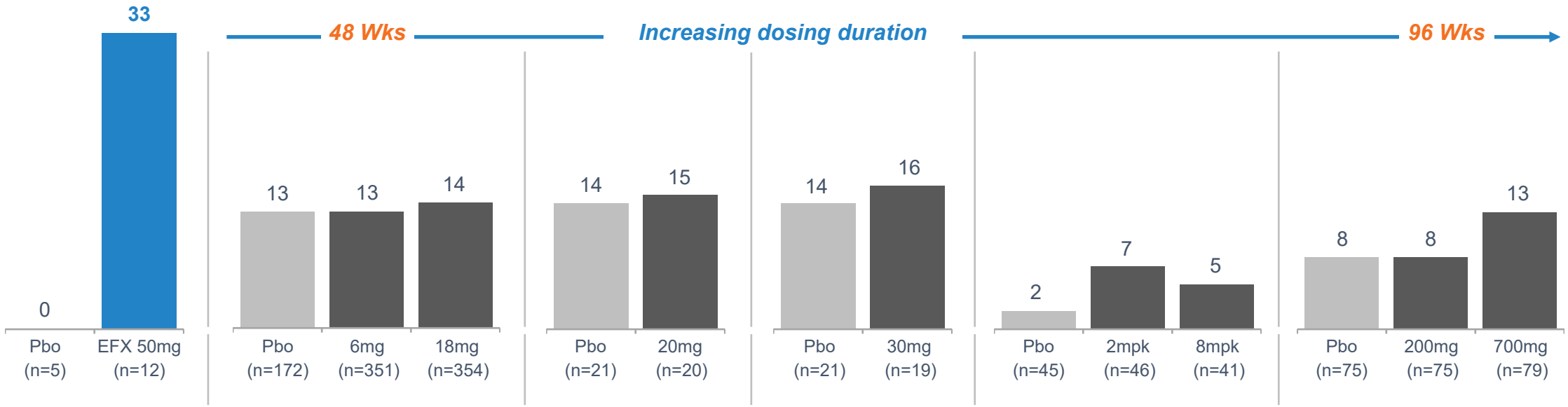
FGF21 Development Landscape: Summary

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*		

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

Cohort C Results In Context: Fibrosis Improvement* (F4)

Fc-FGF21	ASK-1	ACC	FXR	GAL-3	LOXL2
Efruxifermin	Selonsertib	Firsocostat	Cilofexor	Belapectin	Simtuzumab
16 Wks (Ph2a)	48 Wks (Ph3)	48 Wks (Ph2b)	48 Wks (Ph2b)	52 Wks (Ph2b)	96 Wks (Ph2b)
SC Injection	Oral	Oral	Oral	Oral	IV infusion



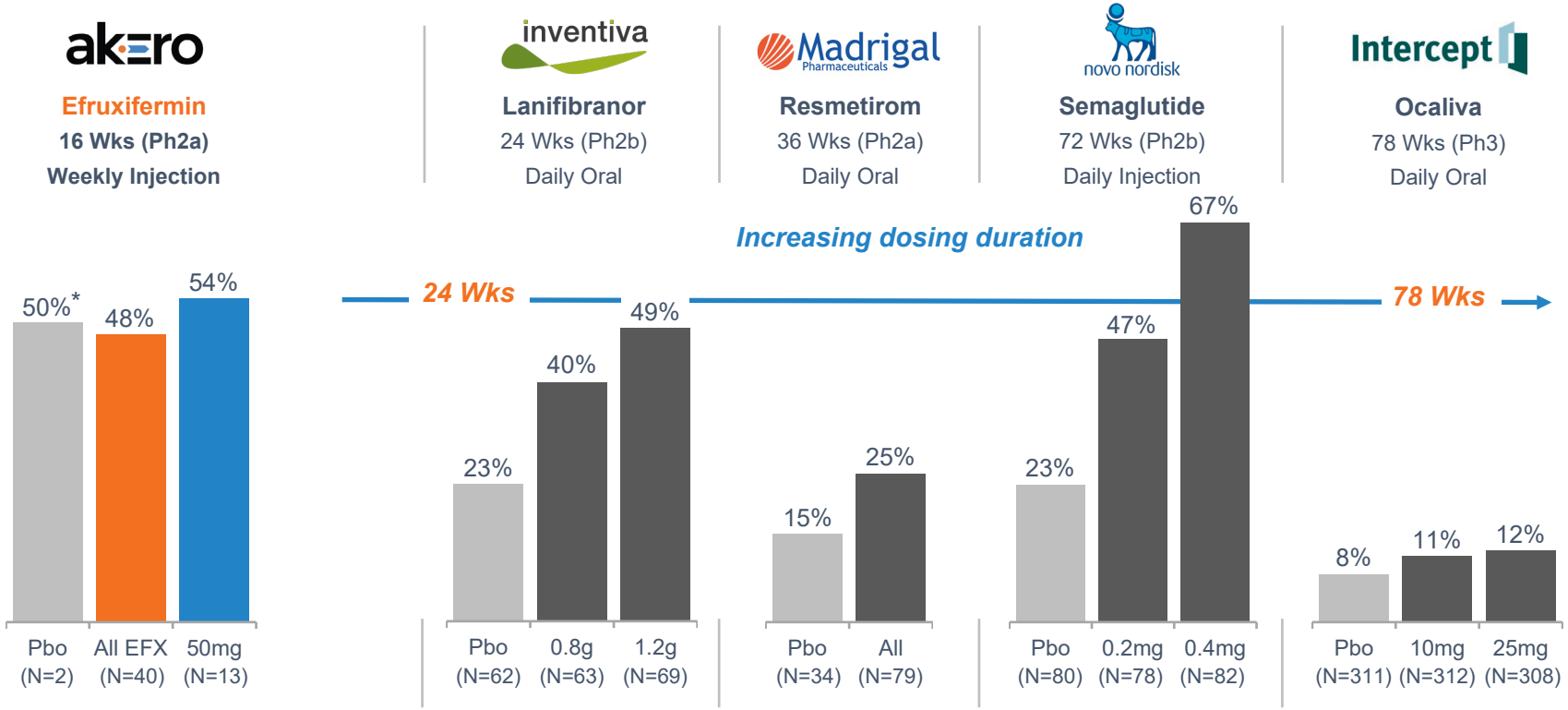
** Results from all publicly reported NASH studies for single agents in F4 patients reporting either ≥ 1-stage fibrosis improvement (belapectin and simtuzumab) or ≥ 1-stage fibrosis improvement and no worsening of NASH (EFX, selonsertib, firsocostat and cilofexor); numerical values represent percent responders*

Harrison, SH et al. (2020) J Hepatol 73(1):26-39; Loomba, R et al. (2020) Hepatol 73(2):625-43; Chalasani, N et al. (2020), Gastro 158:1334–45; Harrison, SH et al. (2018) Gastro 155:1140-53

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.

NASH Development Landscape: NASH Resolution (F1-F3)

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)

NASDAQ: AKRO

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