UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
Washington, D.C. 20549

FORM 8-K

CURRENT REPORT
Pursuant to Section 13 or 15(d)
of The Securities Exchange Act of 1934

Date of Report (Date of earliest event reported): April 13, 2021

Akero Therapeutics, Inc.

(Exact name of registrant as specified in its charter)

Delaware	**001-38944**	**81-5266573**
(State or other jurisdiction of incorporation)	**(Commission File Number)**	**(I.R.S. Employer Identification No.)**

601 Gateway Boulevard, Suite 350	
South San Francisco, CA	**94080**
(Address of principal executive offices)	**(Zip Code)**

Registrant's telephone number, including area code (650) 487-6488

Not Applicable
(Former name or former address, if changed since last report)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions:

☐ Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)

☐ Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)

☐ Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))

☐ Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Securities registered pursuant to Section 12(b) of the Act:

Title of each class	Trading symbol(s)	Name of each exchange on which registered
Common Stock, par value $0.0001 per share	AKRO	The Nasdaq Global Select Market

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (§ 230.405 of this chapter) or Rule 12b-2 of the Securities Exchange Act of 1934 (§ 240.12b-2 of this chapter).

Emerging growth company ☒

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act. ☐

Item 8.01. Other Events.

The Company from time to time presents and/or distributes to the investment community at various industry and other conferences slide presentations to provide updates and summaries of its business. A copy of its current corporate slide presentation is being filed herewith as Exhibit 99.1 to this Current Report on Form 8-K and is incorporated herein by reference. The Company undertakes no obligation to update, supplement or amend the materials attached hereto as Exhibit 99.1.

Item 9.01. Financial Statements and Exhibits.

(d) Exhibits

Exhibit No.	Description
99.1	Corporate slide presentation of Akero Therapeutics, Inc.

SIGNATURES

Pursuant to the requirements of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

Date: April 13, 2021

AKERO THERAPEUTICS, INC.

By: /s/ Andrew Cheng
Andrew Cheng, M.D., Ph.D.
President and Chief Executive Officer

Exhibit 99.1

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A Global Disease,
A Pioneering Treatment

Corporate Presentation

April 2021

CORPORATE HIGHLIGHTS

Efruxifermin (EFX): Potential Leading NASH Monotherapy	• 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 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
2021 Milestones (Completed & Upcoming)	• Dosed first patient in Ph2b HARMONY study in F2/F3 patients with 28 and 50mg doses in March • Expect to initiate Ph2b SYMMETRY study in cirrhotic (F4) patients in 2H'21 • Expect to manufacture Phase 3 API in 2H'21
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

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

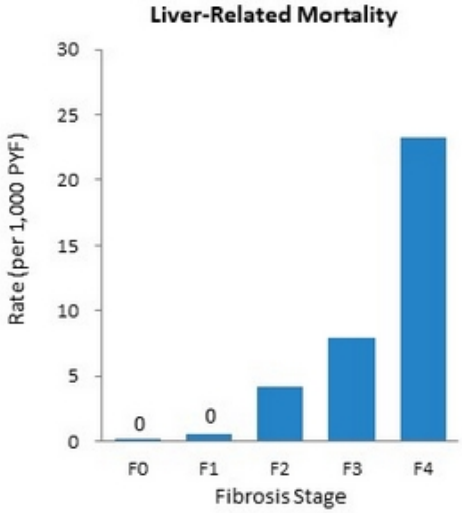
The number of NASH patients with advanced-stage fibrosis and cirrhosis is predicted to **grow to 8 million** in the US by 2030, **an increase of approximately 140% from 2015**

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

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

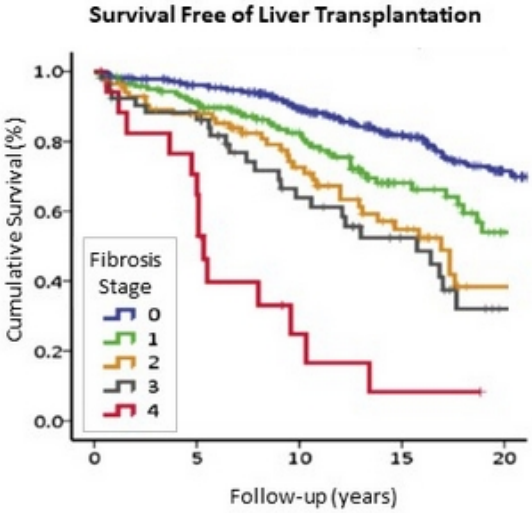
CIRRHOTIC NASH PATIENTS HAVE POOR PROGNOSIS

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



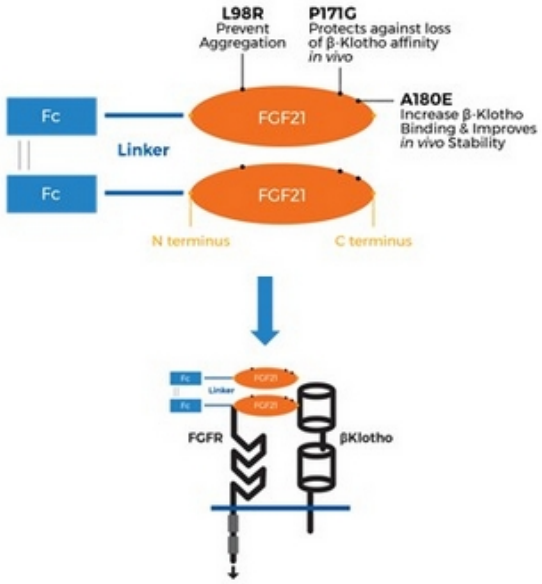
Dulai, PS et al. (2017) Hepatology 65:1557-65

~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



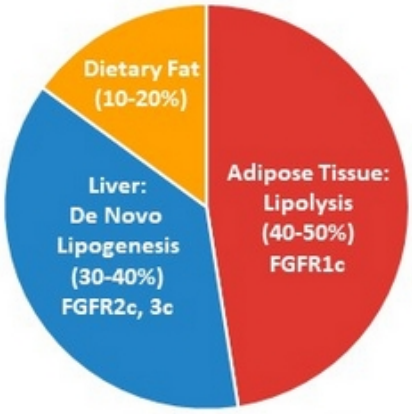
Key attributes

- 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

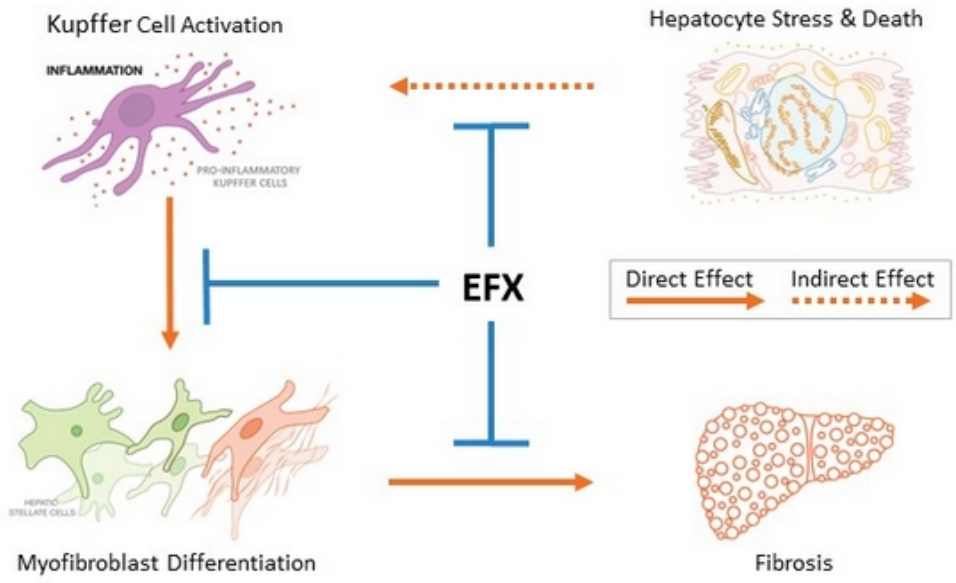


Lambert, JE *et al.* (2014) *Gastroenterology* 146(3): 726-35

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	✓

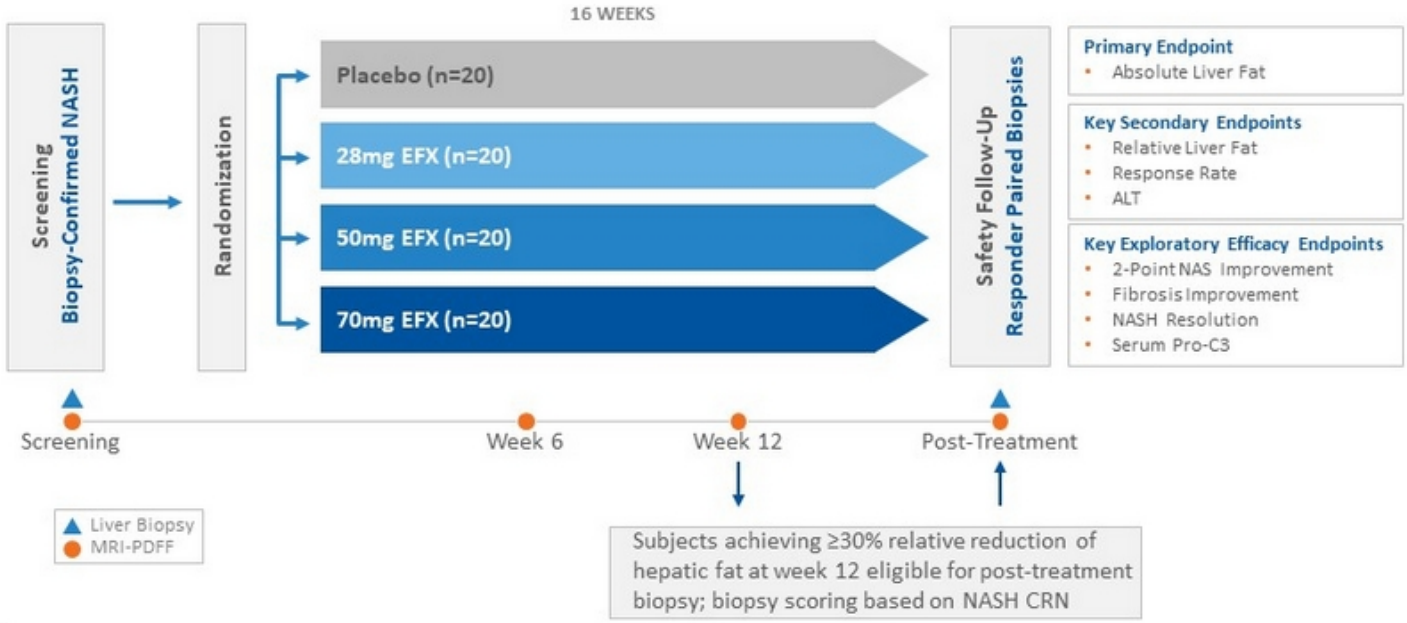
EFX DIRECT AND INDIRECT ANTI-FIBROTIC EFFECTS



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)



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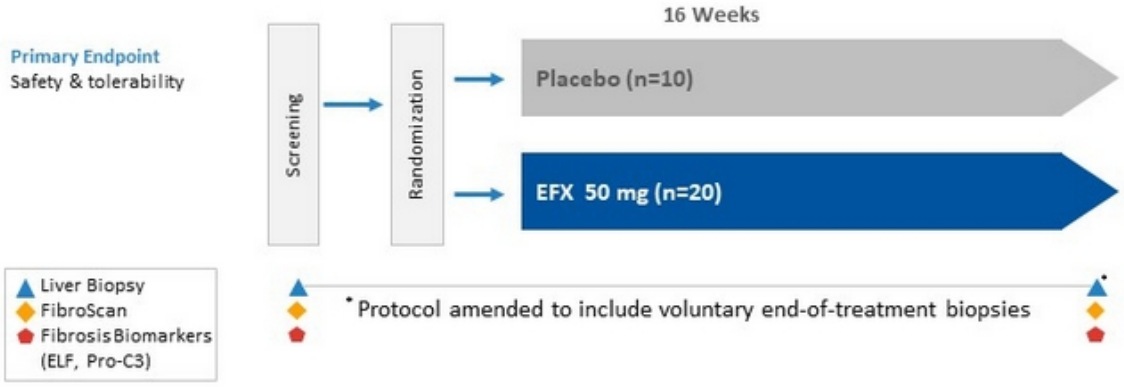
PHASE 2A EXPANSION COHORT C TRIAL DESIGN (F4 NASH)

BALANCED study included an expansion cohort, Cohort C, of patients with compensated cirrhosis (F4), Child-Pugh Class A; results to inform subsequent development in cirrhotic patients

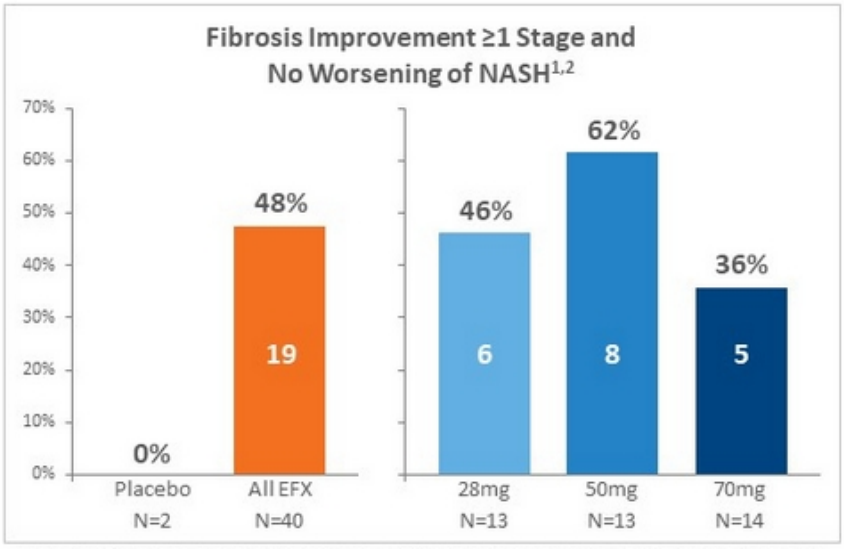


BASELINE DEMOGRAPHICS: MAIN STUDY & COHORT C

	BALANCED Main[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)



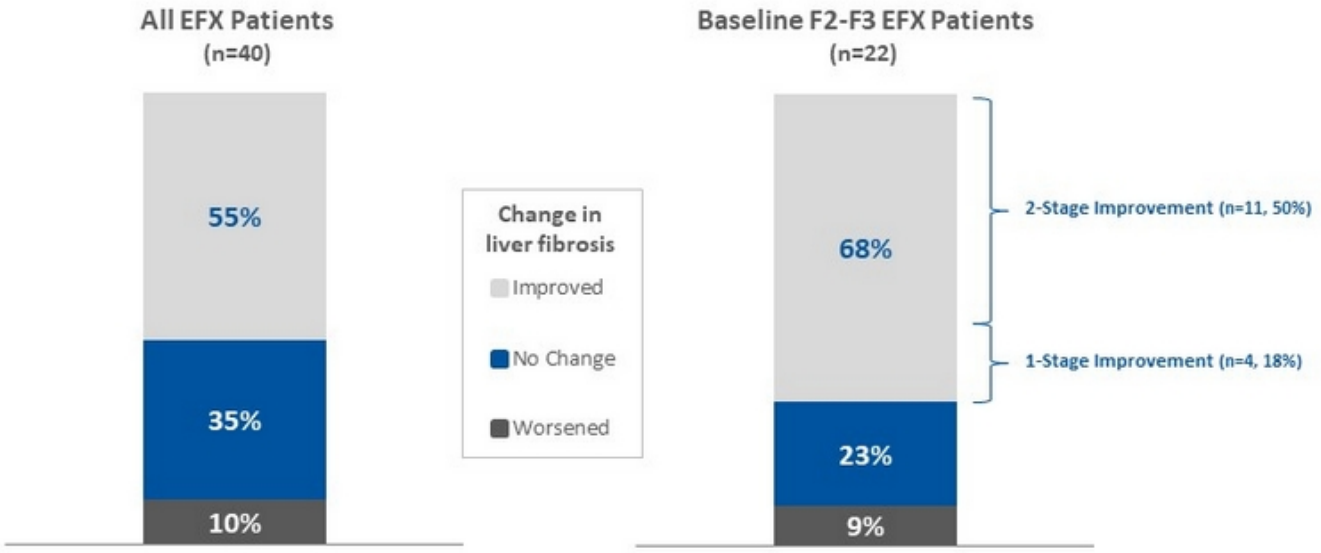
Biopsy Reading

- 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] 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

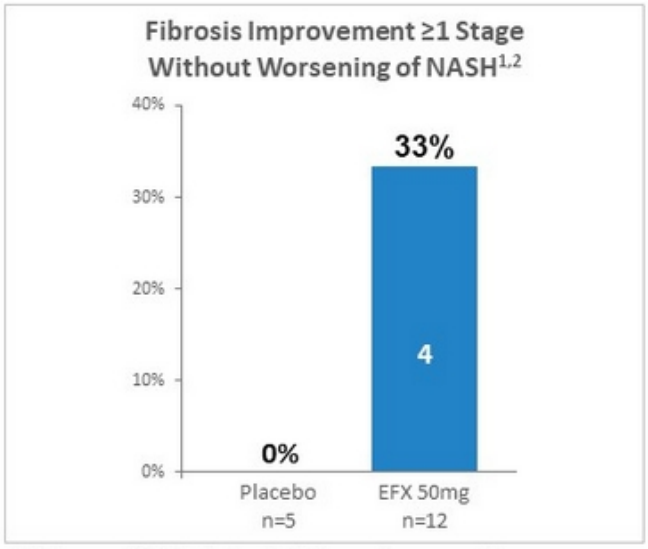
akero Source Data: Liver Biopsy Evaluable Analysis Set, F1-F3 (all BALANCED main study responders who had baseline and end-of-treatment liver biopsy results)

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



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

HIGH RATE OF FIBROSIS IMPROVEMENT AFTER ONLY 16 WEEKS AMONG CIRRHOTIC PATIENTS (F4 NASH)



[1] No increase in NAS for ballooning, inflammation, or steatosis
[2] Study not powered to assess statistical significance of changes in histological endpoints

Biopsy Reading

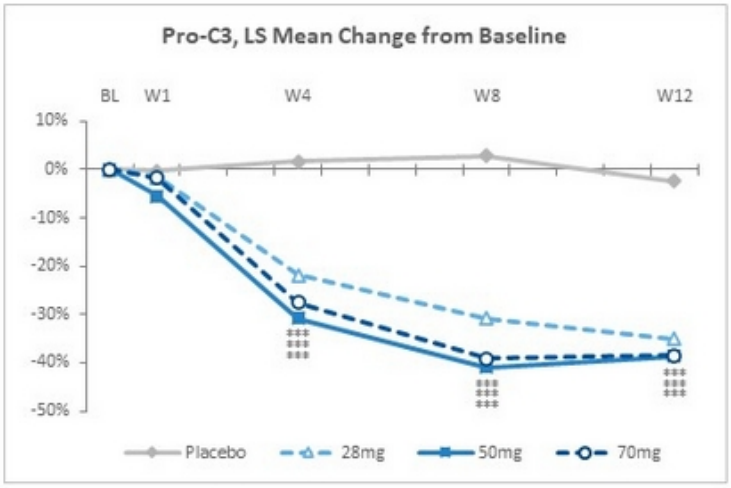
- All baseline and end-of-treatment biopsies were read by a single NASH-CRN pathologist
- All biopsies were read blinded to both treatment assignment and patient
- Baseline biopsies were read independently of end-of-treatment biopsies, in random fashion and not paired

Baseline Biopsy Timing

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

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)

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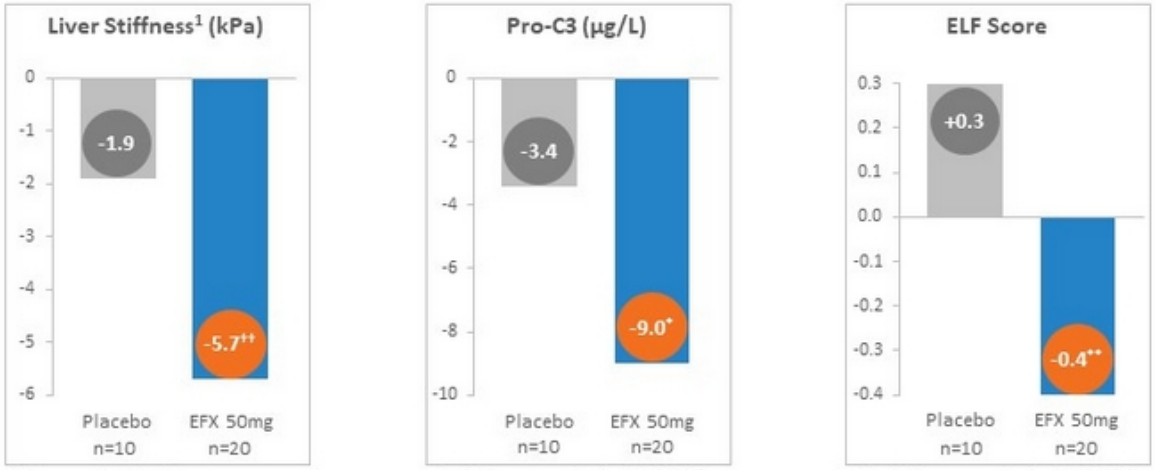
Source Data: Full Analysis Set, F1-F3

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)

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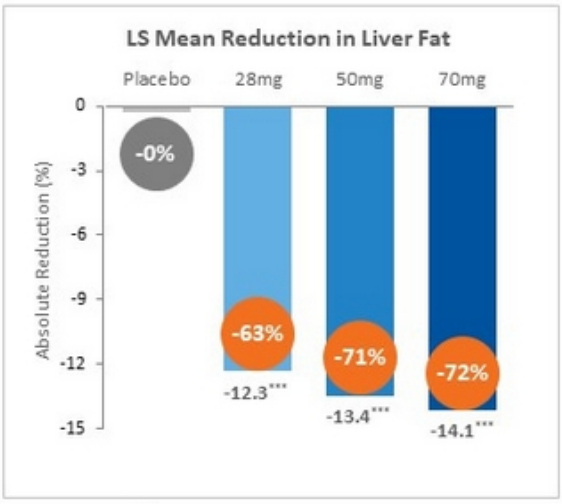
Source Data: Biomarker Analysis Set, F4 (all Cohort C subjects with baseline and interpretable on-study measure of ELF or pro-C3, respectively); Liver Stiffness Analysis Set, F4 (all Cohort C subjects with baseline and interpretable on-study measure of Liver Stiffness); *Topline preliminary data*

INTERPRETING THE RAPID REVERSAL OF FIBROSIS OBSERVED IN NASH PATIENTS TREATED WITH EFX

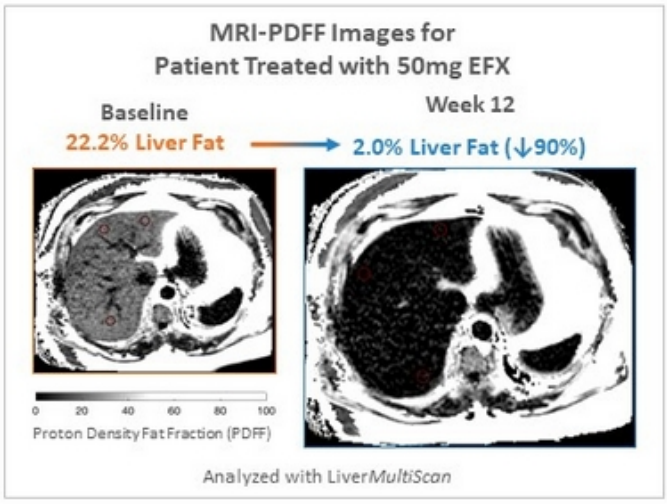
- Fibrosis reversal in cirrhotic patients (F4), two-stage improvement of fibrosis in F2/F3 patients, and corroborating non-invasive markers of fibrosis improvement likely reflects direct anti-fibrotic activity
- Fibrosis reversal is especially advantageous for cirrhotic patients who face high risk of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

SUBSTANTIAL REDUCTIONS IN LIVER FAT AT WEEK 12 ACROSS ALL DOSE GROUPS (F1-F3 NASH)

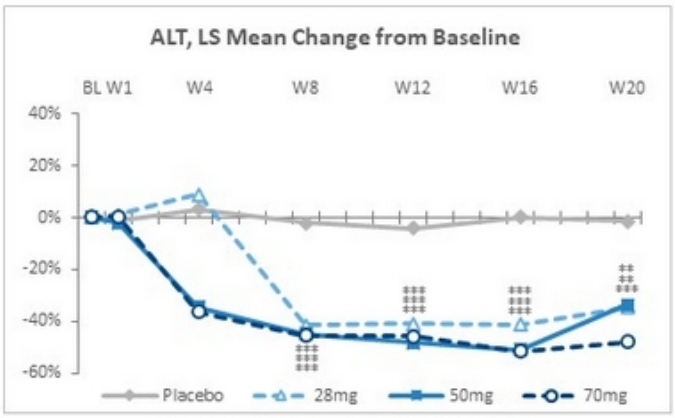


*** p<0.001, versus placebo (ANCOVA)

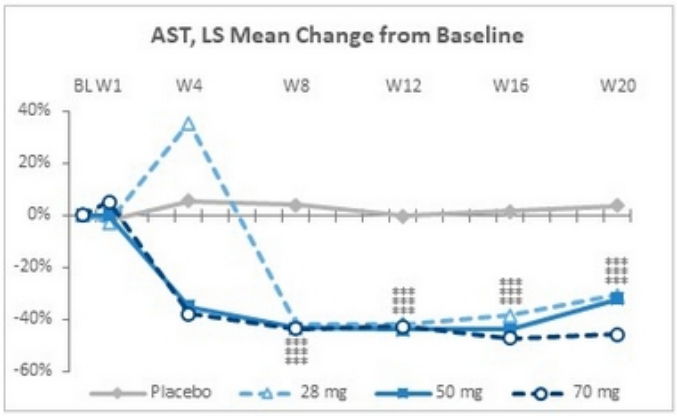


Source Data: Full Analysis Set, F1-F3

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

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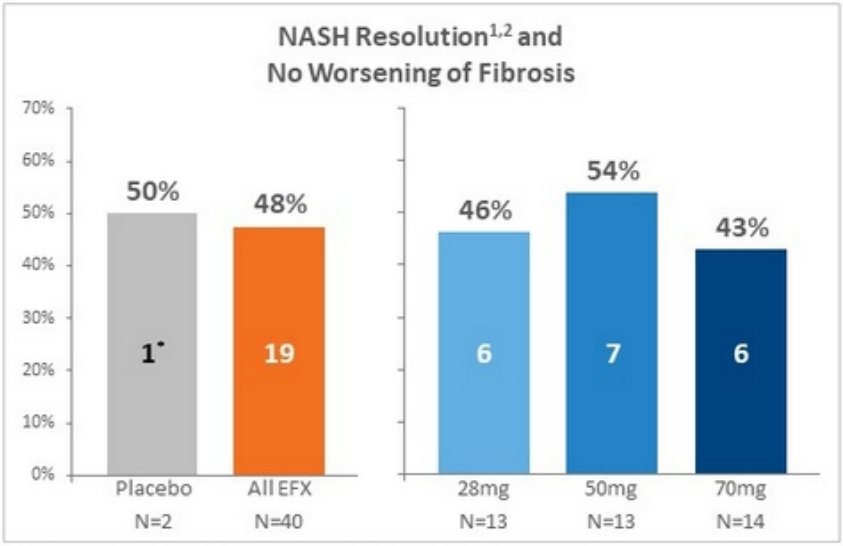
Source Data: Full Analysis Set, F1-F3

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)

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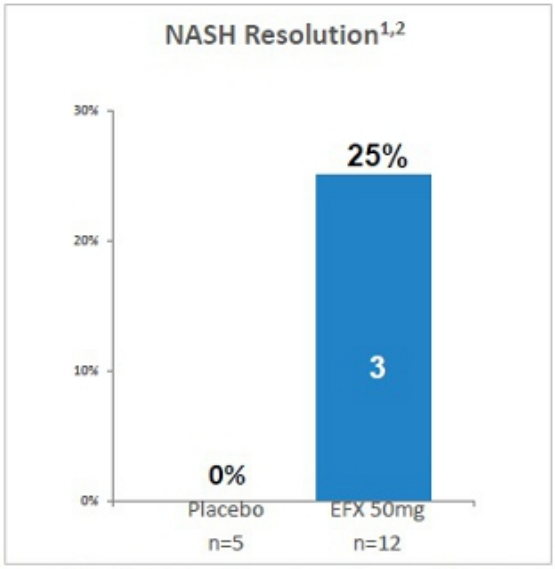
Source Data: Liver Biopsy Evaluable Analysis Set, F1-F3

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*

DRUG-RELATED TREATMENT-EMERGENT ADVERSE EVENTS (TEAE) (F1-F3 NASH)

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%)	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*

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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

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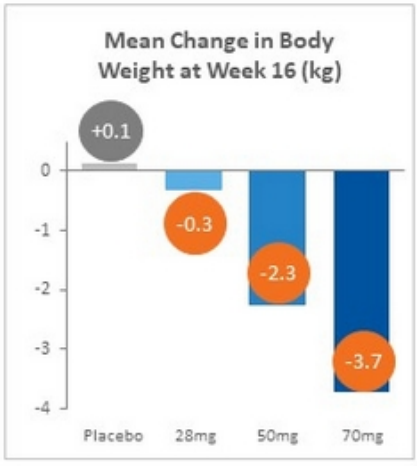
Source Data: Safety Set, F4 (all Cohort C subjects confirmed by central reader as F4 at baseline who received at least one dose of study drug); *Topline preliminary data*

WEIGHT LOSS OBSERVED FOR ALL DOSE GROUPS (F1-F3 NASH)



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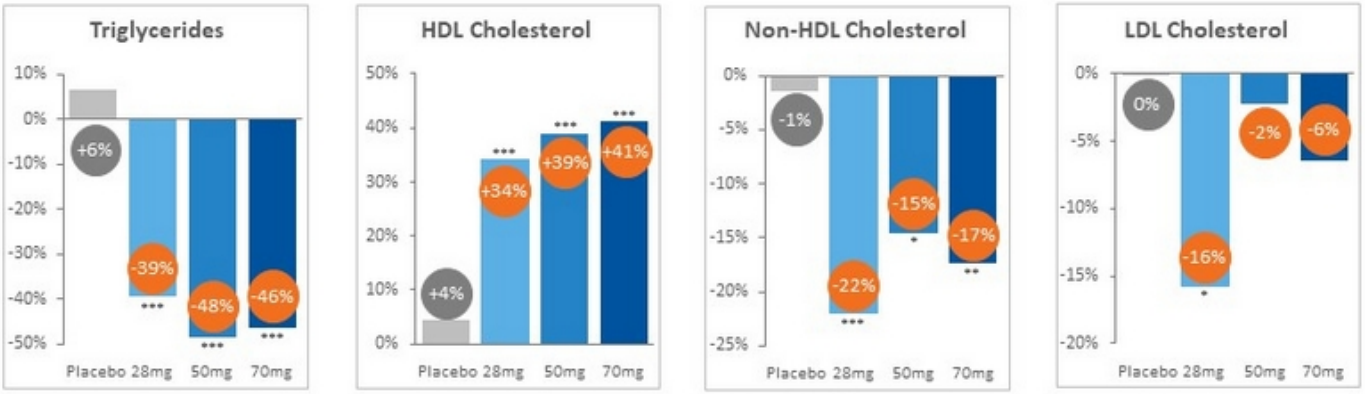
Source Data: Full Analysis Set, F1-F3

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)

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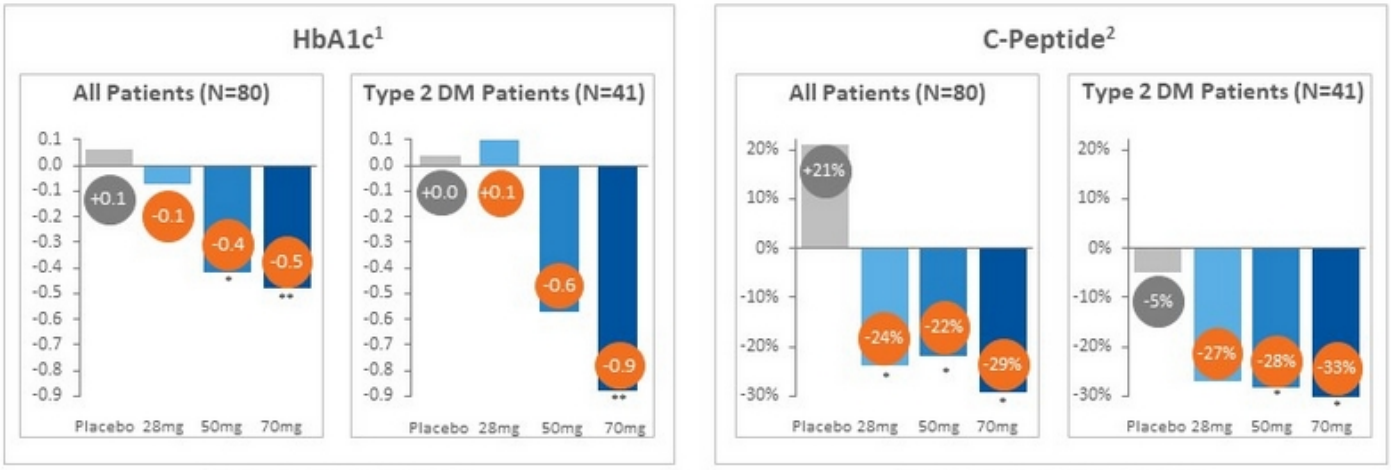
Source Data: Full Analysis Set, F1-F3

CLINICALLY MEANINGFUL IMPROVEMENTS IN GLYCEMIC CONTROL AFTER 16 WEEKS (F1-F3 NASH)

LS Mean Change From Baseline to Week 16 (%)



[1] Absolute change from baseline, %

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

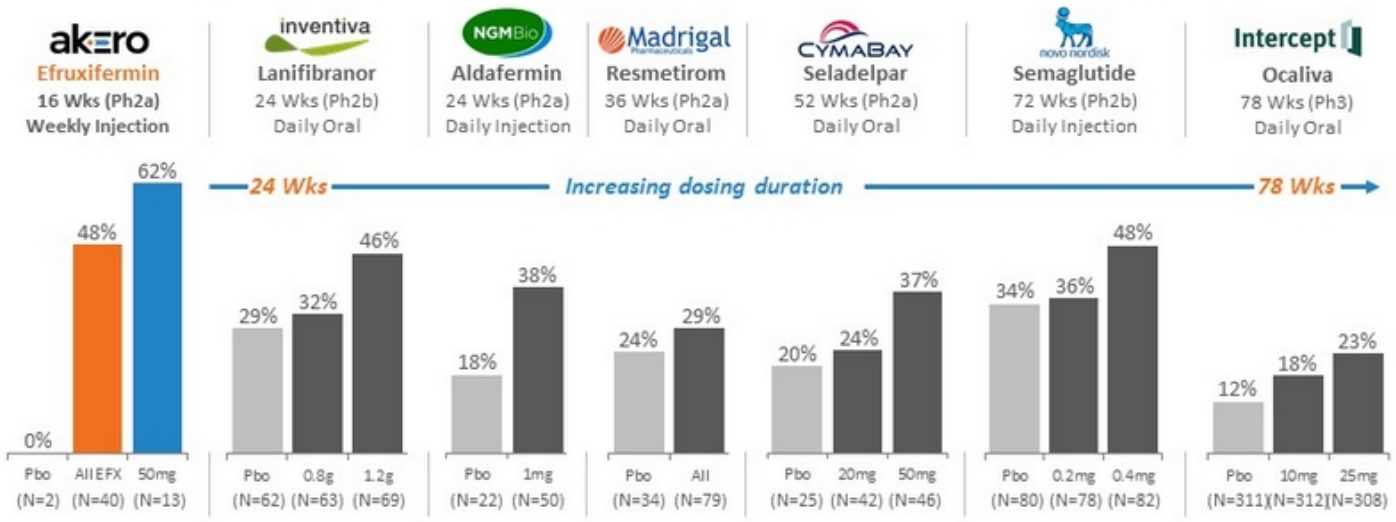
[2] Relative percent change from baseline

* p<0.05, versus placebo (ANCOVA)

Source Data: Full Analysis Set, F1-F3

NASH DEVELOPMENT LANDSCAPE: FIBROSIS IMPROVEMENT (F1-F3)

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

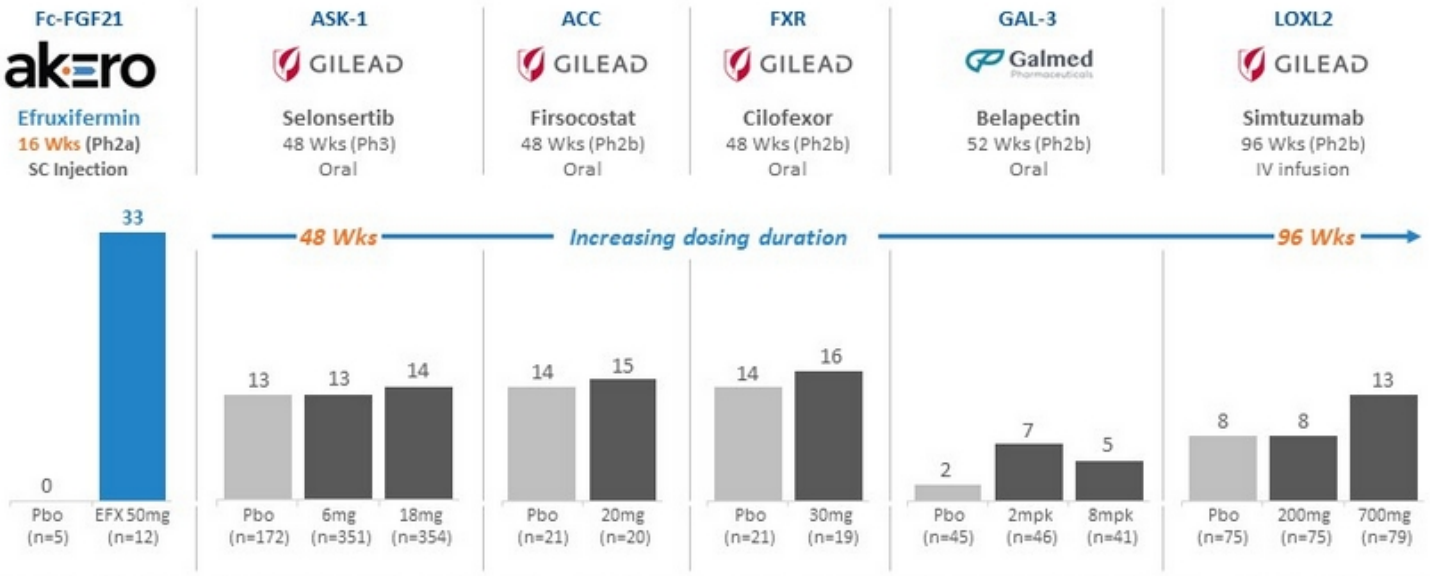


Note: These data are derived from different clinical trials at different points in time, with differences in trial design and patient populations. No head-to-head clinical trials have been conducted.

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)

COHORT C RESULTS IN CONTEXT: FIBROSIS IMPROVEMENT* (F4)



** 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.

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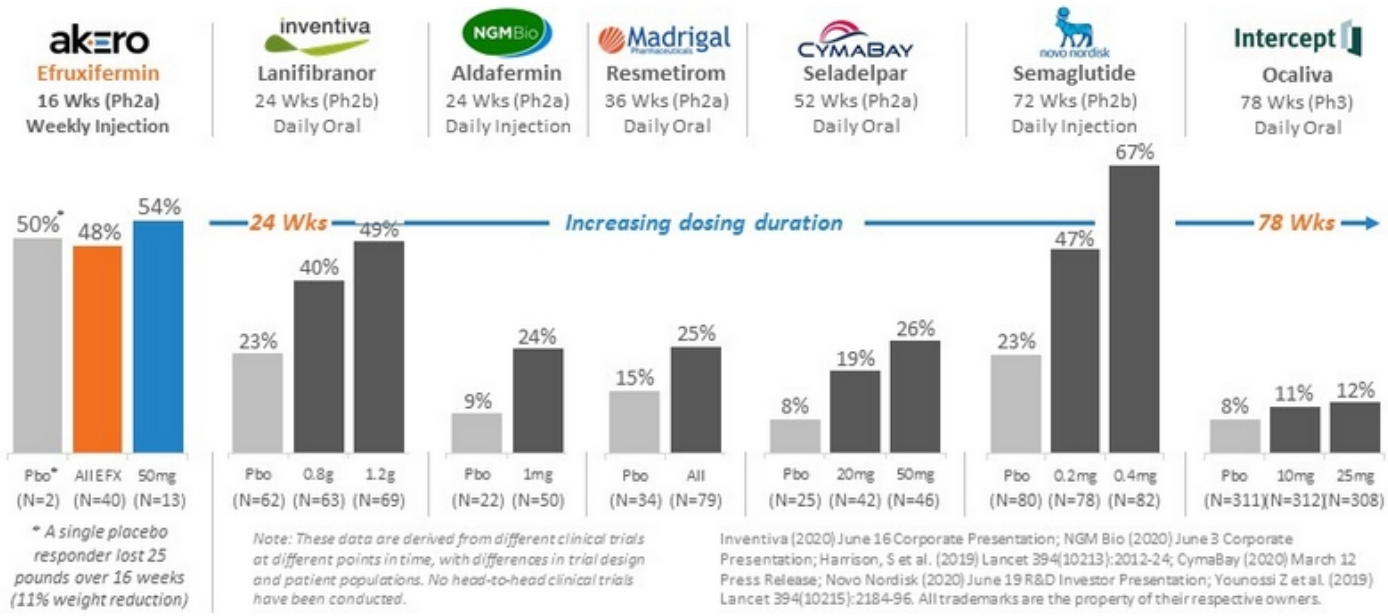
NASH DEVELOPMENT LANDSCAPE: NASH RESOLUTION (F1-F3)

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.

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.

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

NEXT STEPS FOR EFX: PARALLEL 2B TRIALS IN F2/F3 & F4

	BALANCED	Cohort C (Expansion of BALANCED)	HARMONY	SYMMETRY
Status	**Completed** (Readout Jun'20)	**Completed** (Readout Mar'21)	**Ongoing** (Initiated Feb'21)	**Expected to be initiated 2H'21**
Duration	16 Weeks	16 Weeks	24 Weeks	Under review
EFX Arms	28, 50, 70mg	50mg	28, 50mg	Under review
Placebo-Controlled	✓	✓	✓	✓

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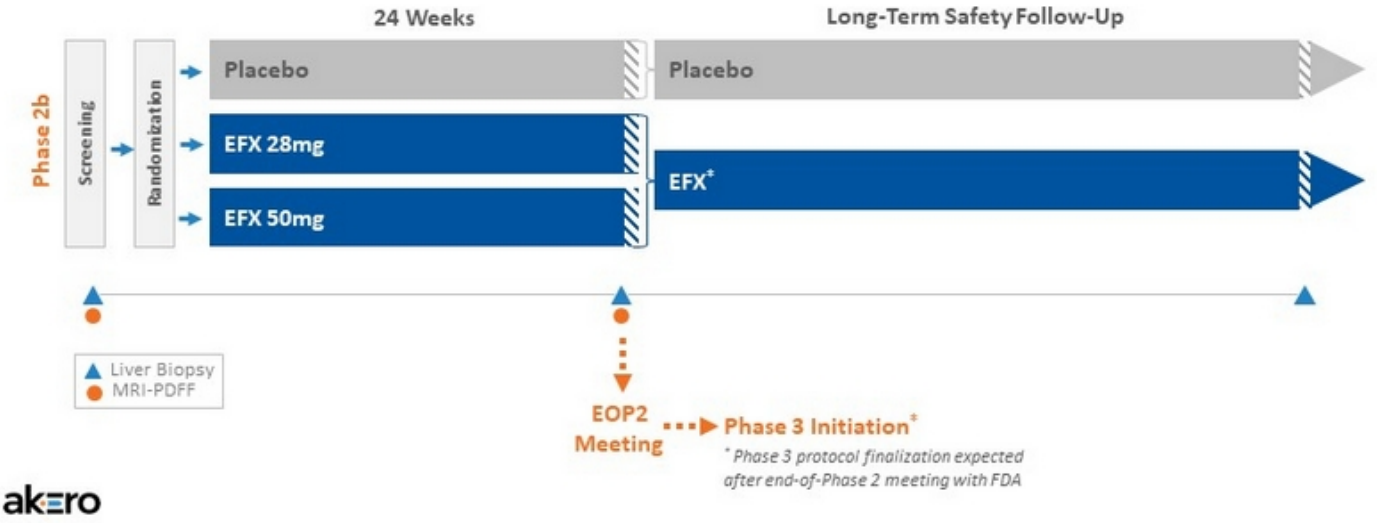
PHASE 2B (F2/F3) TRIAL DESIGN (HARMONY)

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



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STRONG FINANCIAL POSITION

COMPLETED UPSIZED FOLLOW-ON OFFERING
July 10, 2020

~$106M	~$216M	~$268M
Raised in aggregate gross proceeds	Raised in aggregate gross proceeds	

CONSISTENT RECORD OF MILESTONE DELIVERY

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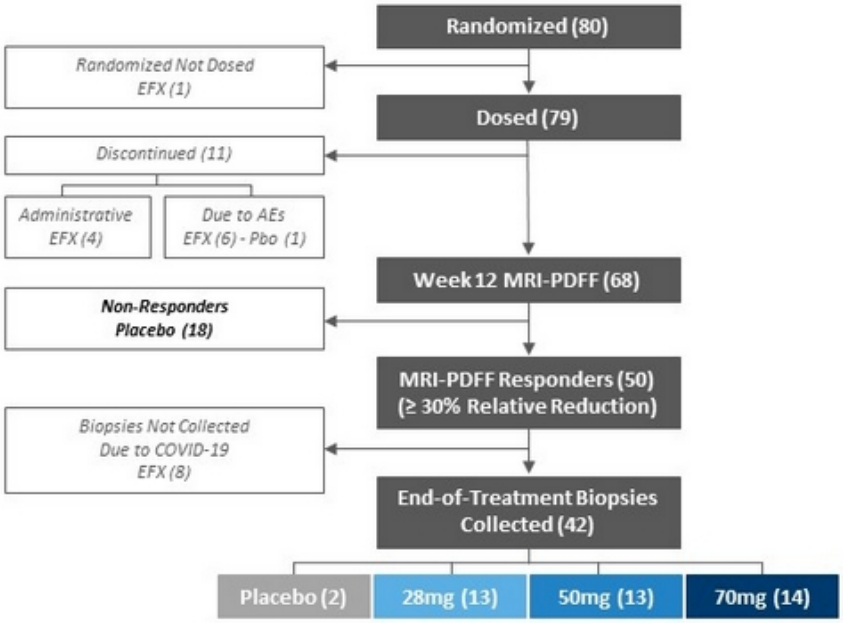
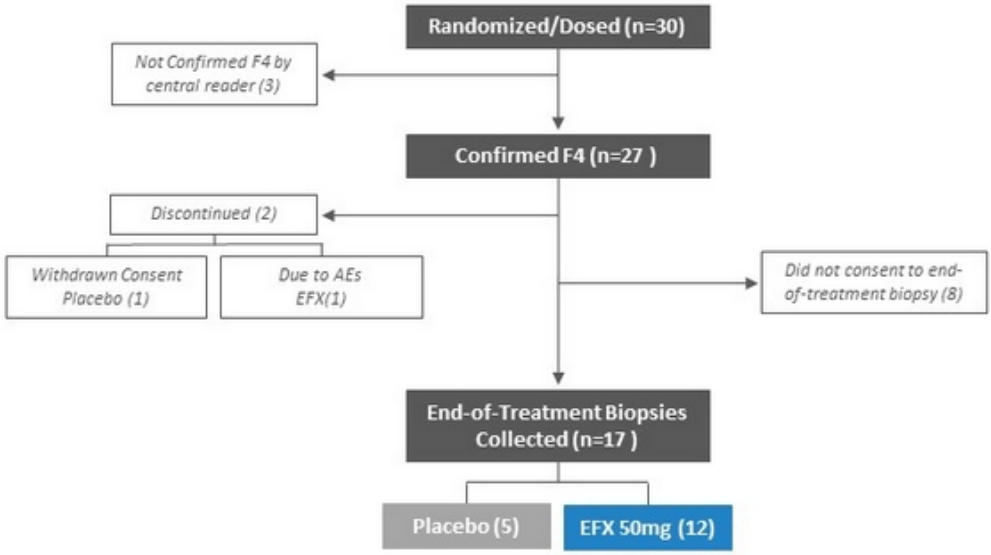
Backup Slides

PATIENT DISPOSITION (BALANCED MAIN STUDY)



PATIENT DISPOSITION (BALANCED COHORT C)



MAGNITUDE OF LIVER FAT REDUCTION (F1-F3 NASH)

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 (all BALANCED main study subjects who had baseline and Week 12 MRI-PDFF

EXPANDING EFX TO CIRRHOTIC PATIENT POPULATION (F4)

Developing Treatments for Cirrhotic (F4) NASH

- FDA draft guidance specific for F4 patients
- Projected ~3.5M US F4 patients in 2030
- FDA: *The goals of treatment for compensated NASH cirrhosis are to halt or slow progression of fibrosis, prevent clinical decompensation, reduce the need for liver transplantation, and improve survival*

Nonalcoholic Steatohepatitis with Compensated Cirrhosis: Developing Drugs for Treatment

Guidance for Industry

DRAFT GUIDANCE

This guidance document is being distributed for comment purposes only.

Comments and suggestions regarding this draft document should be submitted within 60 days of publication in the *Federal Register* of the notice announcing the availability of the draft guidance. Submit electronic comments to https://www.regulations.gov. Submit written comments to the Dockets Management Staff (HFA-305), Food and Drug Administration, 5630 Fishers Lane, Rm. 1061, Rockville, MD 20852. All comments should be identified with the docket number listed in the notice of availability that publishes in the *Federal Register*.

For questions regarding this draft document, contact Frank Anania at 240-402-9725.

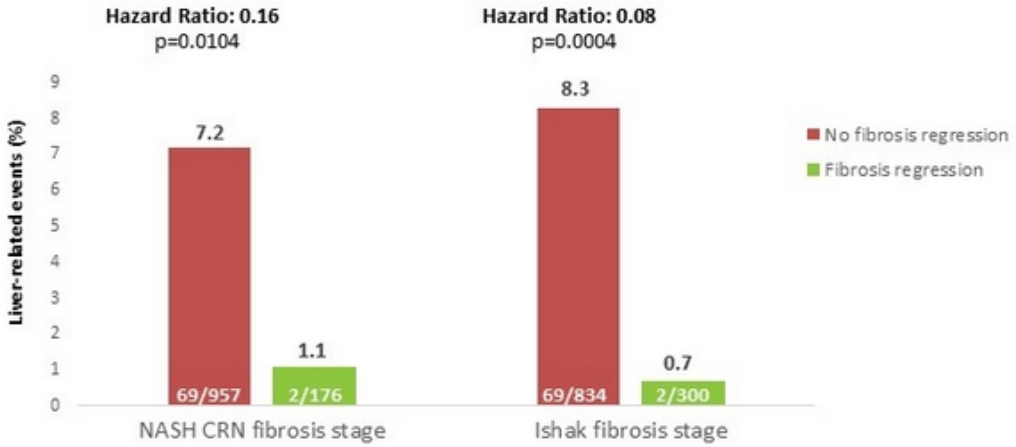
U.S. Department of Health and Human Services
Food and Drug Administration
Center for Drug Evaluation and Research (CDER)

June 2019
Clinical/Medical

247852844fl.docx
3/6/2019

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.

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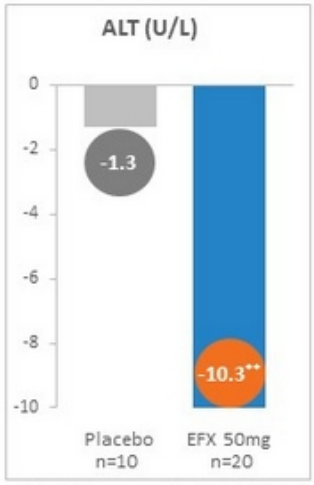
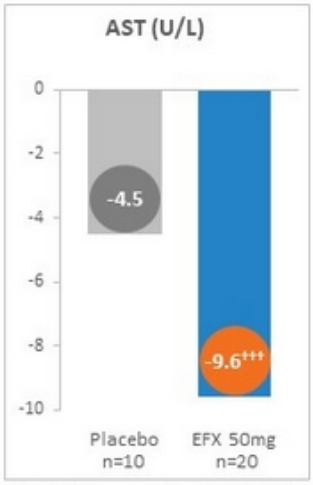
REDUCTIONS IN MARKERS OF LIVER INJURY (F4 NASH)

LS Mean Change from Baseline to Week 16



** p<0.01, versus placebo (ANCOVA)



+++ p<0.001, versus baseline (ANCOVA)

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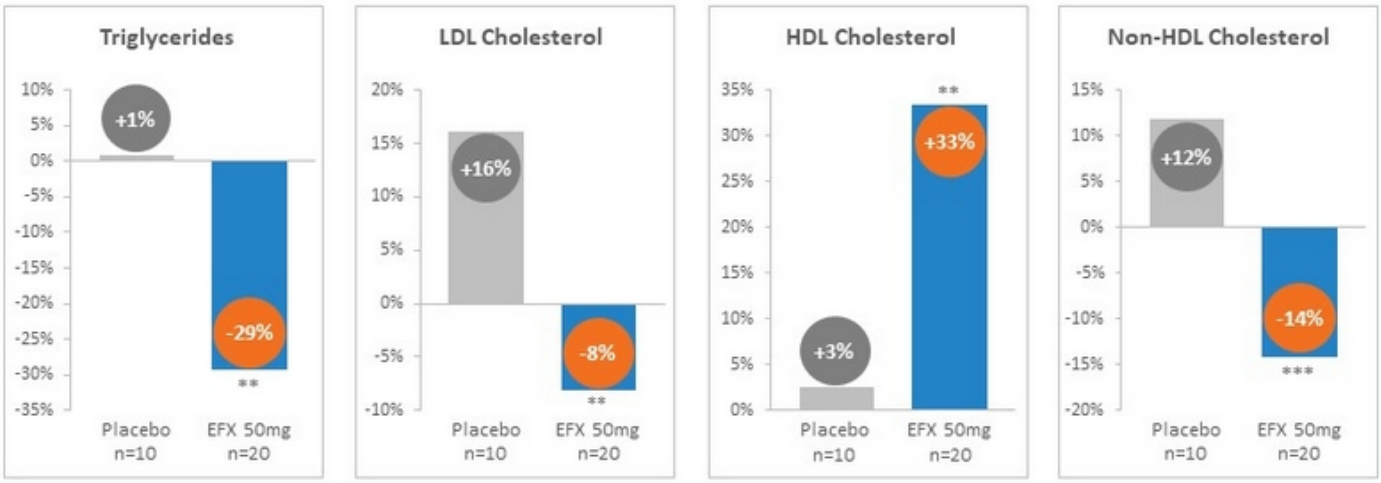
Source Data: Full Analysis Set, F4; *Topline preliminary data*

IMPROVED LIPOPROTEIN PROFILE (F4 NASH)

LS Mean Change From Baseline to Week 16 (%)



** p<0.01, versus placebo (ANCOVA)

** p<0.01, versus placebo (ANCOVA)

** p<0.01, versus placebo (ANCOVA)

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

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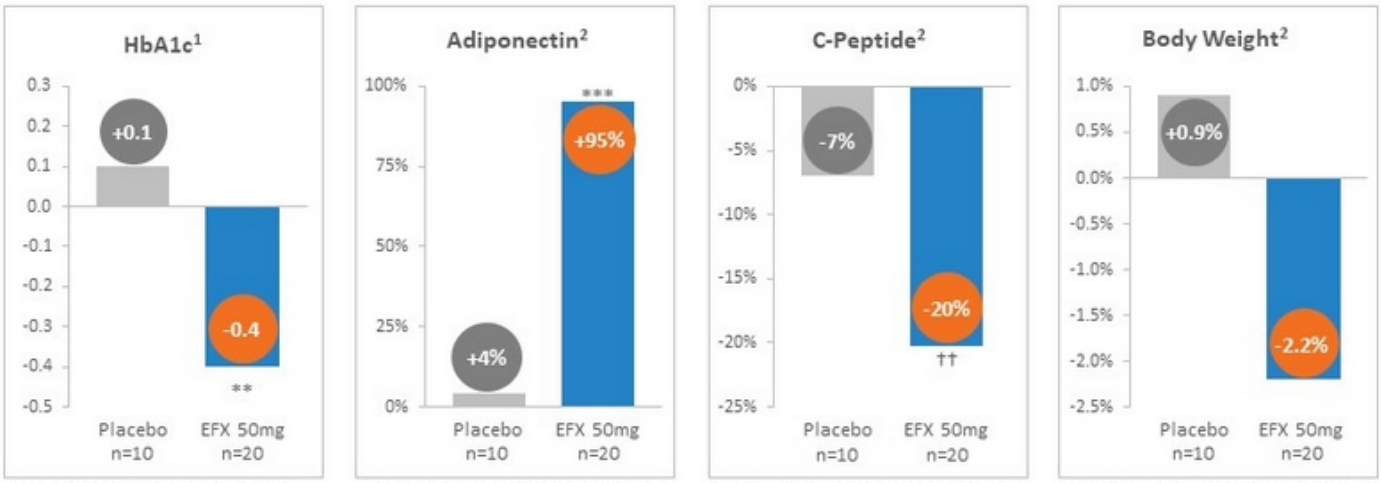
Source Data: Full Analysis Set, F4; *Topline preliminary data*

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

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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

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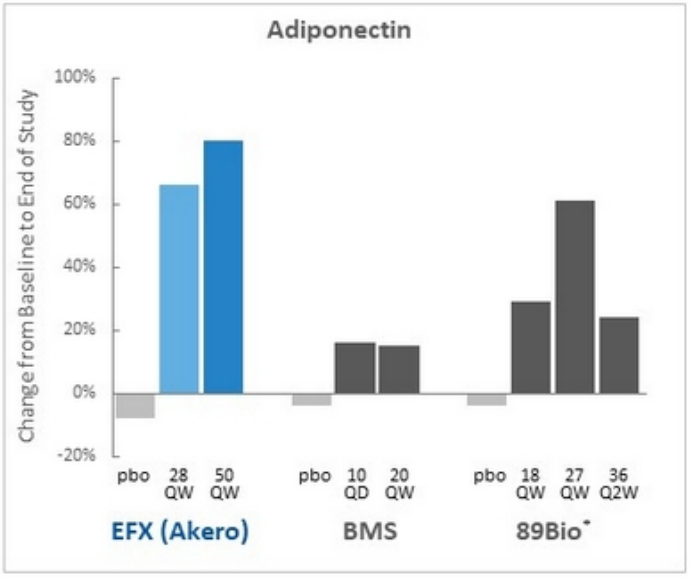
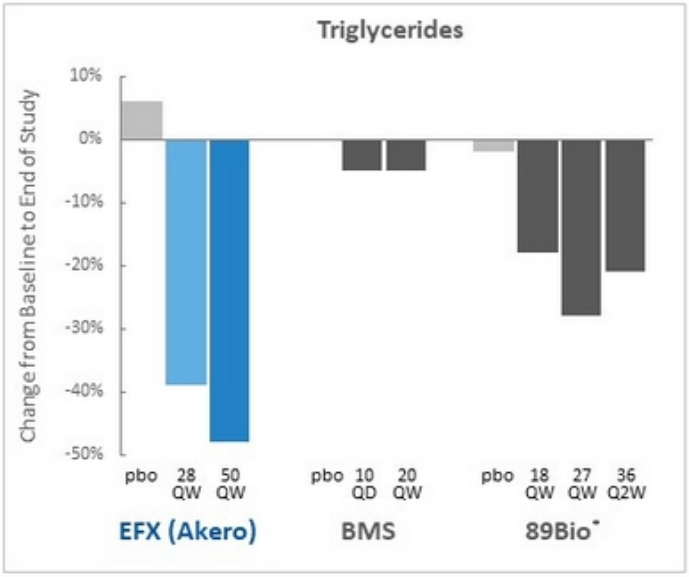
** Subjects with biopsy-confirmed NASH were present only in the 18mg QW & 18 Q2W cohorts (Loomba et al., 2020 AASLD Poster #LP34)*

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

** 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)*

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A Global Disease,
A Pioneering Treatment

NASDAQ: AKRO