

**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): October 15, 2020

Akero Therapeutics, Inc.
(Exact name of registrant as specified in its charter)

Delaware
(State or other jurisdiction
of incorporation)

001-38944
(Commission
File Number)

81-5266573
(I.R.S. Employer
Identification No.)

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

94080
(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 Akeru Therapeutics, Inc.
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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: October 15, 2020

AKERO THERAPEUTICS, INC.

By: /s/ Andrew Cheng

Andrew Cheng, M.D., Ph.D.

President and Chief Executive Officer



akero

**A Global Disease,
A Pioneering Treatment**
Akero Therapeutics, Inc.
Corporate Presentation

October 2020

This presentation may contain “forward-looking statements” of Akeo Therapeutics, Inc. (“we,” “us,” “our,” “Akeo” 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; 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.



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 NASH Comorbidities

- Improved HbA1c
- Reduction in triglycerides
- No LDL-C increase
- Weight loss

Improved Histology

- 48% fibrosis improvement ≥ 1 stage and no worsening of NASH
- 48% NASH resolution and no worsening of fibrosis

Safety & Tolerability

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

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



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



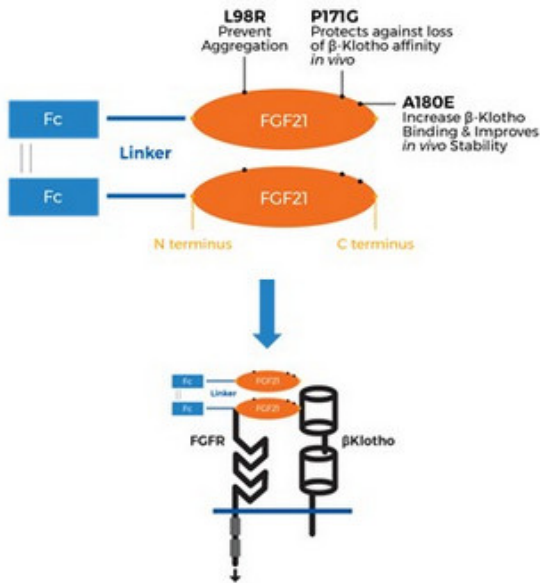
NASH is a **leading cause of liver transplantation** in the US and Europe



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



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



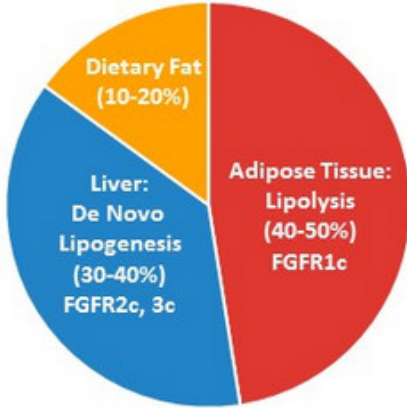
Key attributes

- ✓ 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; Kharitonov, 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



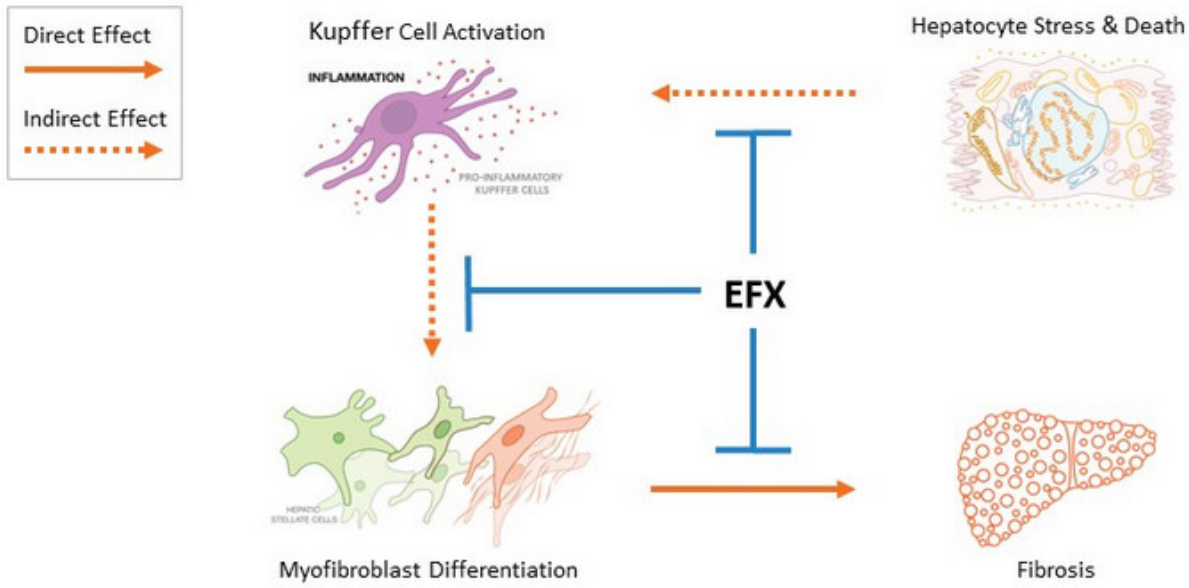
Lambert, JE et al. (2014) *Gastroenterology* 146(3): 726-35

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

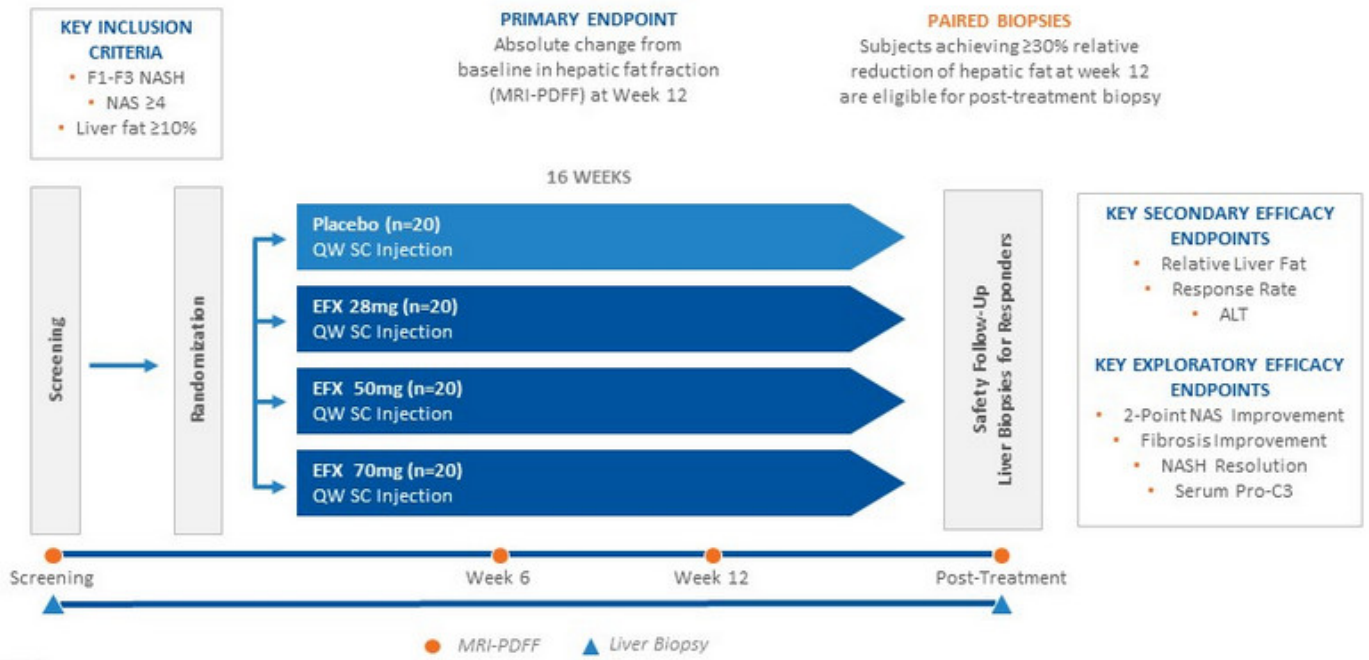
Source of Liver Fat	FGF Receptor	FGF21 Activity
Lipolysis	FGFR1c	✓
De Novo Lipogenesis	FGFR2c FGFR3c	✓



EFX DIRECT AND INDIRECT ANTI-FIBROTIC EFFECTS



BALANCED STUDY TRIAL DESIGN

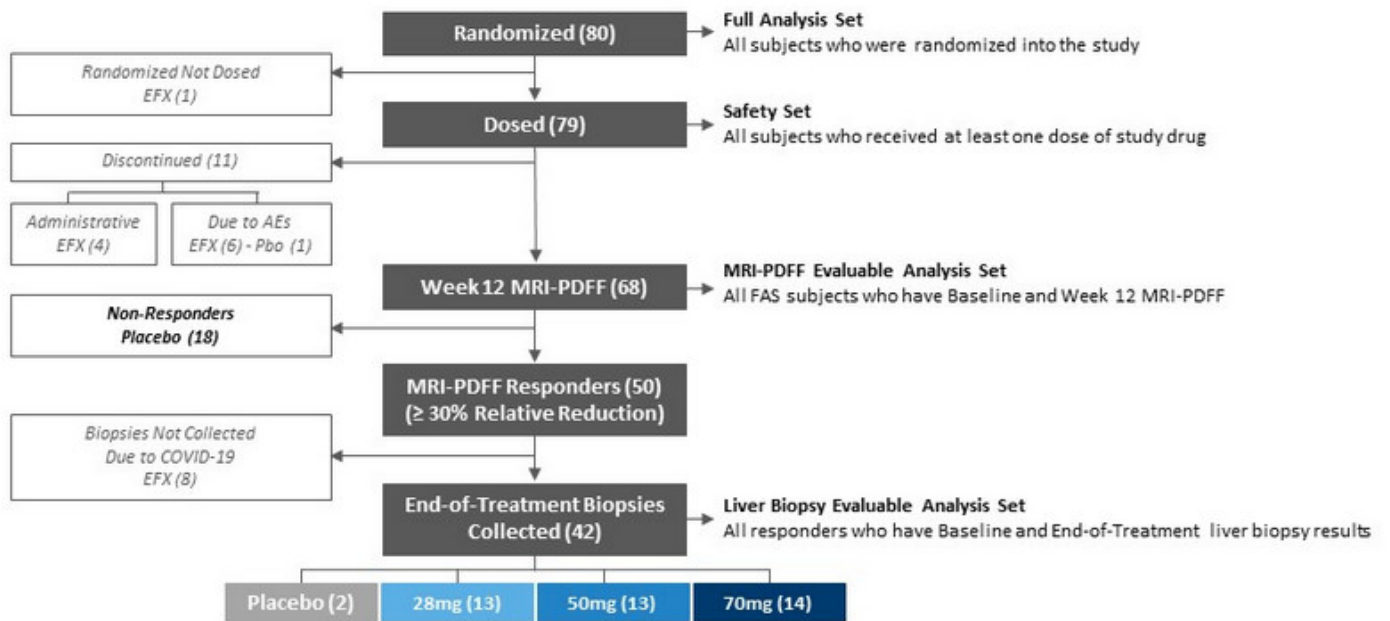




BASELINE DEMOGRAPHICS

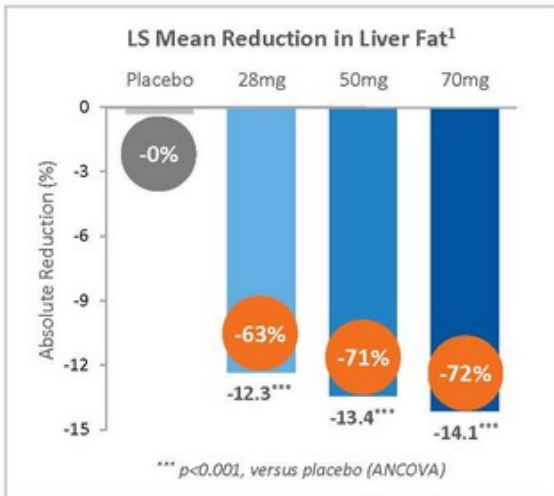
Parameter Mean	Placebo (N=21)	EFX 28mg (N=19)	EFX 50mg (N=20)	EFX 70mg (N=20)
Age (Years)	52	50	53	53
Sex (Male/Female)	6/15	9/10	10/10	9/11
Weight (kg)	99.6	108.2	103.6	103.1
BMI (kg/m ²)	37.6	38.8	36.7	37.2
Liver Fat Content (% by MRI-PDFF)	19.3	21.4	18.3	19.4
NAFLD Activity Score (NAS)	5.1	5.6	5.1	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

PATIENT DISPOSITION





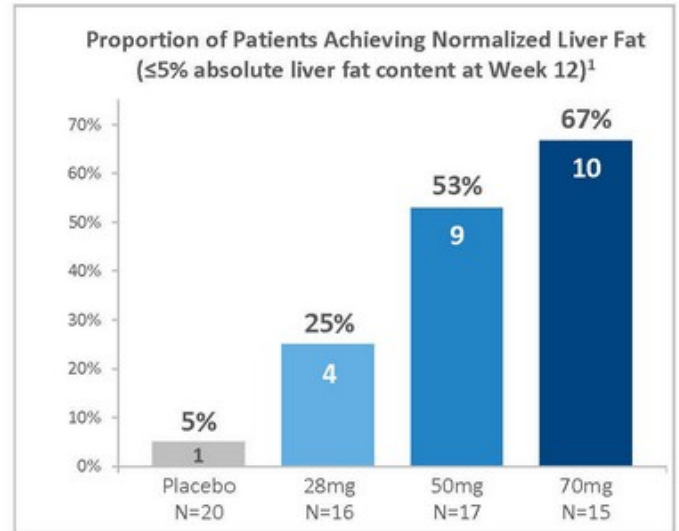
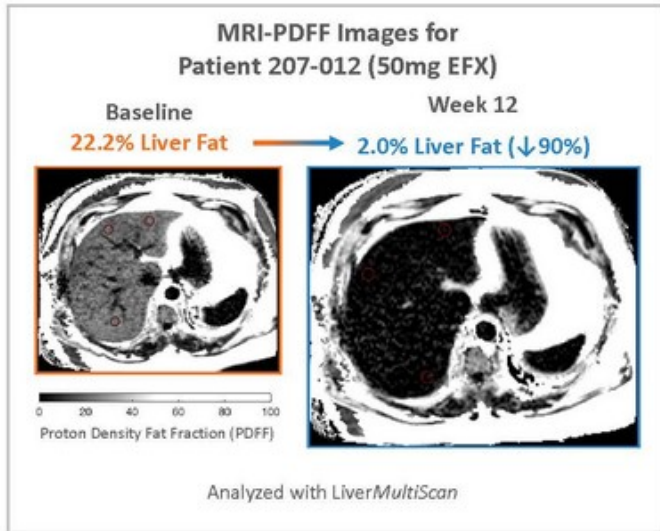
SUBSTANTIAL REDUCTIONS IN LIVER FAT AT WEEK 12 ACROSS ALL DOSE GROUPS



**Proportion of Patients Achieving
≥30% Relative Reduction in Liver Fat
(MRI-PDFF responder)²**

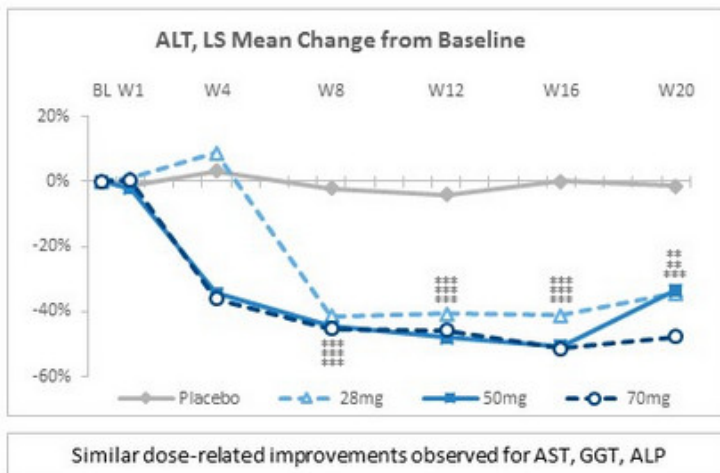
Placebo (N=20)	EFX 28mg (N=16)	EFX 50mg (N=17)	EFX 70mg (N=15)
10%	100%	100%	100%

SUBSTANTIAL NORMALIZATION OF LIVER FAT AT WEEK 12





REDUCTION IN HEPATOCYTE STRESS AND COLLAGEN SYNTHESIS ACROSS ALL DOSE GROUPS



Pro-C3, LS Mean (ug/L)

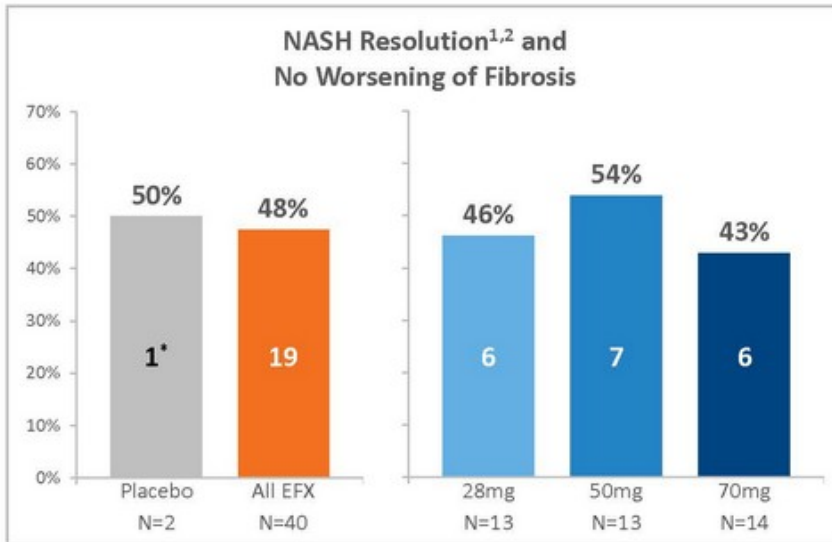
Dose Group	Baseline	Δ Week 12
Placebo	16.1	-1.5
28mg	19.2	-6.1***
50mg	16.2	-5.9***
70mg	17.2	-6.7***

* p<0.05, ** p<0.01, *** p<0.001, versus placebo

Source Data: Full Analysis Set; MMRM (ALT), ANCOVA (Pro-C3)



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

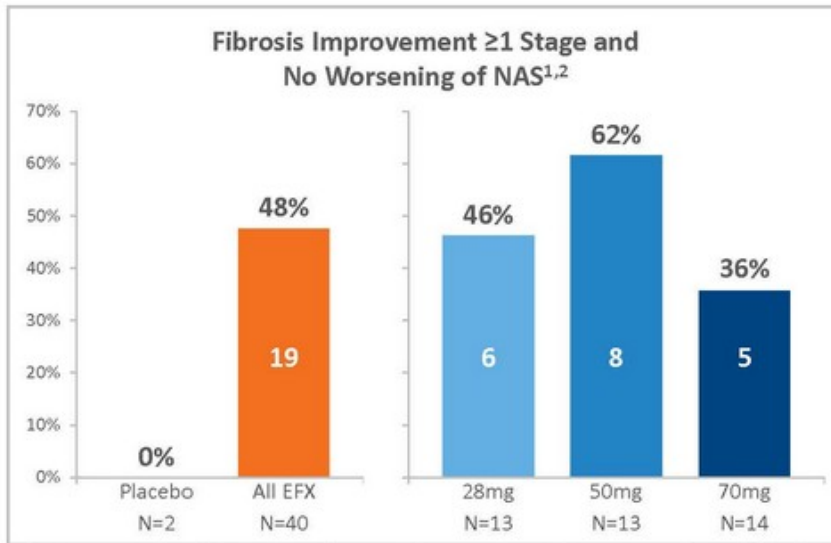
¹ NAS score of 0 or 1 for lobular inflammation and a score of 0 for ballooning

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)



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



≥ 2 -Stage Improvement in Fibrosis

11 of 40 EFX patients (28%) had a ≥ 2 -stage improvement

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

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

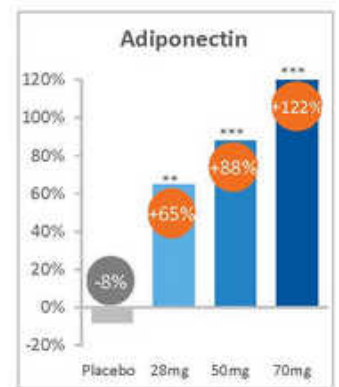
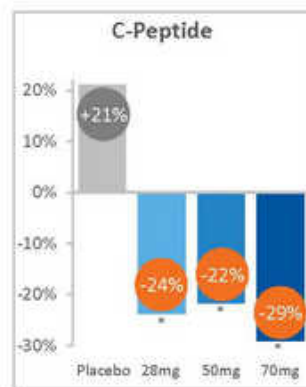
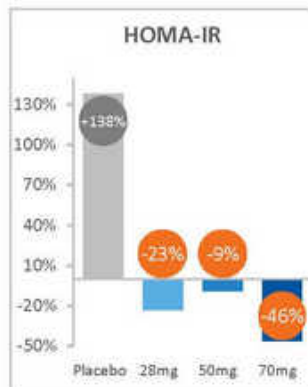
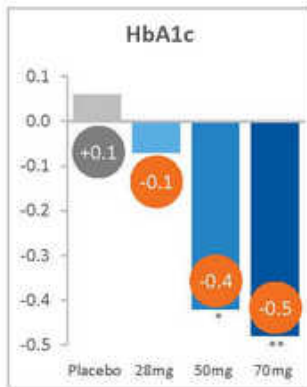
[†] Across EFX dose groups

^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



CLINICALLY MEANINGFUL IMPROVEMENTS IN GLYCEMIC CONTROL AFTER 16 WEEKS

LS Mean Change From Baseline to Week 16 (%)¹



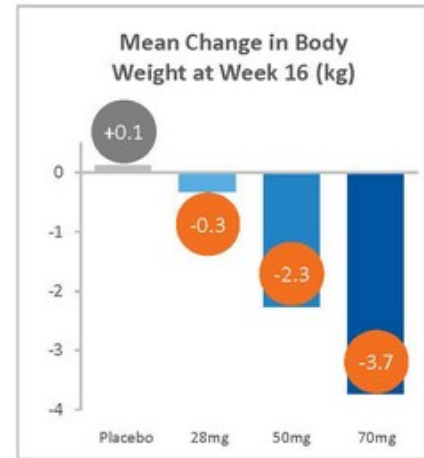
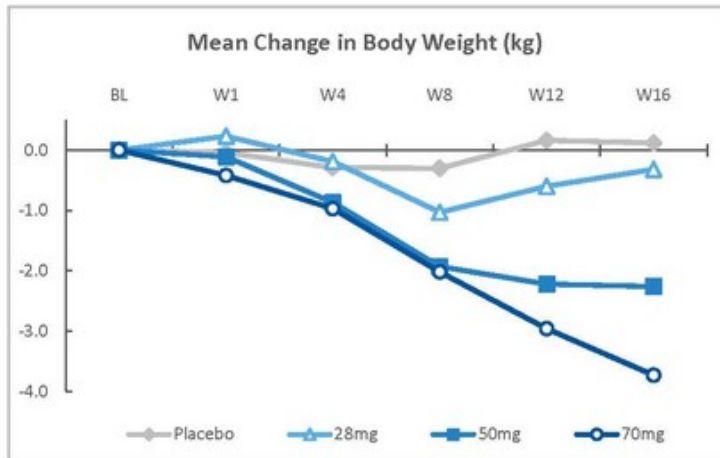
¹ HbA1c is presented in absolute percent change from baseline, whereas HOMA-IR, C-Peptide, and Adiponectin are presented in relative percent change from baseline

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

Source Data: Full Analysis Set



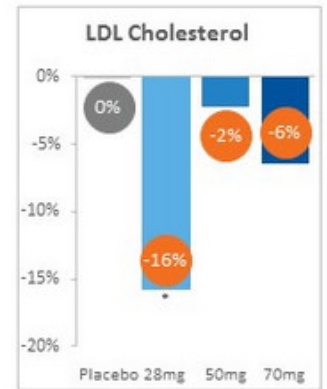
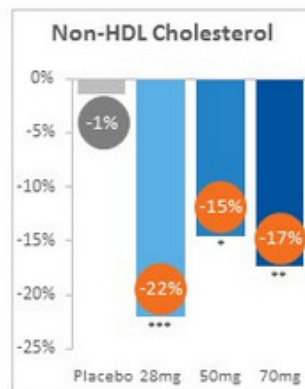
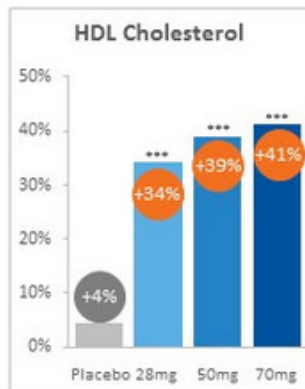
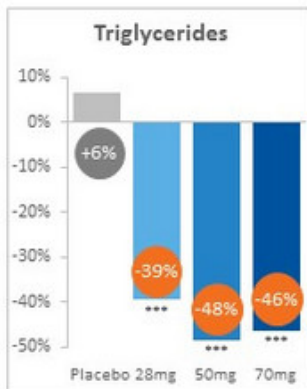
WEIGHT LOSSES OBSERVED FOR ALL DOSE GROUPS





IMPROVED LIPOPROTEIN PROFILE FOR CARDIOVASCULAR HEALTH

LS Mean Change From Baseline to Week 16 (%)



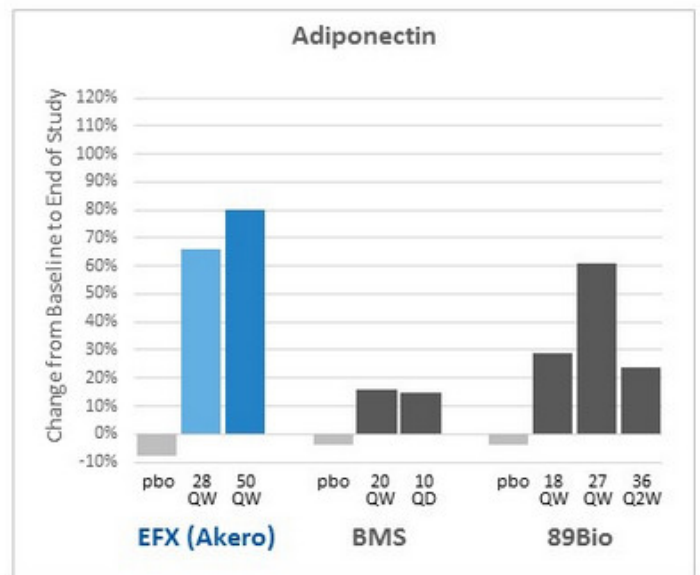
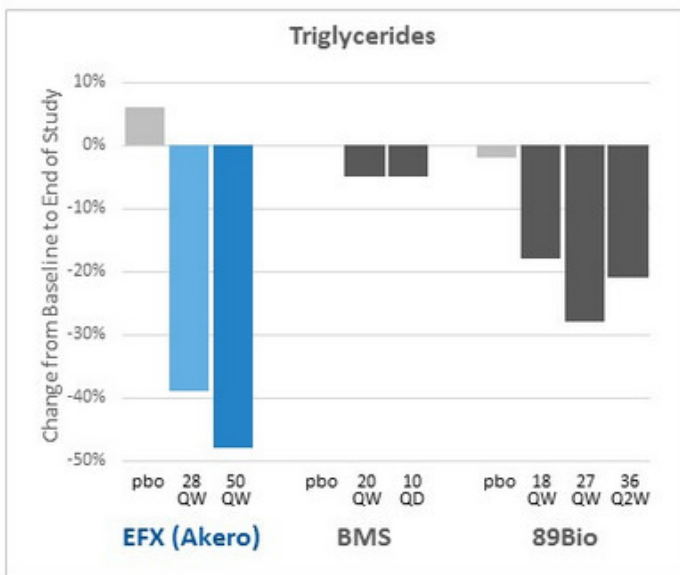
FGF21 DEVELOPMENT LANDSCAPE

Noninvasive Measures: Percent Change From Baseline to End of Study	Akeru (EFX) 16 weeks			BMS (Pegbelfermin) 16 weeks			89Bio (BIO89-100) 12 weeks			
	pbo	28 QW	50 QW	pbo	20 QW	10 QD	pbo	18 QW	27 QW	36 Q2W
Dose										
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



MARKERS OF 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



FGF21 DEVELOPMENT LANDSCAPE: SUMMARY

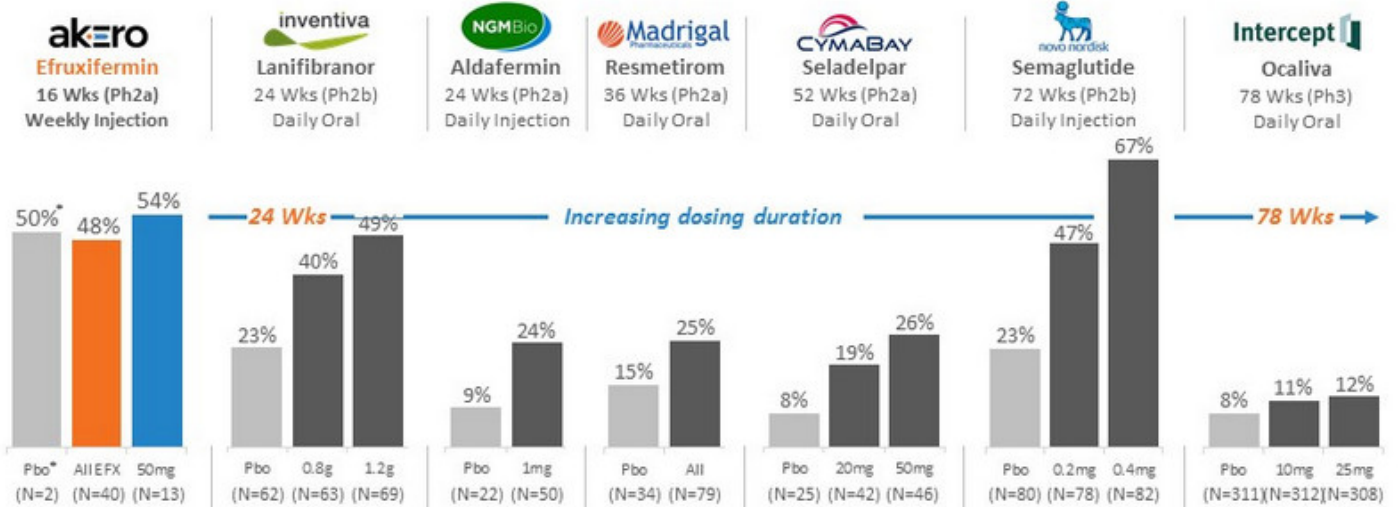
Consideration	Fc-FGF21 Fusion Protein (Akeru)	Pegylated FGF21 (BMS or 89Bio)
Patient Population: NASH diagnosed only by biopsy; NASH patients have more advanced disease and worse comorbidities	Biopsy-confirmed NASH	BMS: biopsy-confirmed NASH; 89Bio: ~20% biopsy-confirmed NASH
Histology: Fibrosis only 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



NASH DEVELOPMENT LANDSCAPE: NASH RESOLUTION

Proportion of Subjects with Resolution of NASH and No Worsening of Fibrosis¹



* 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.

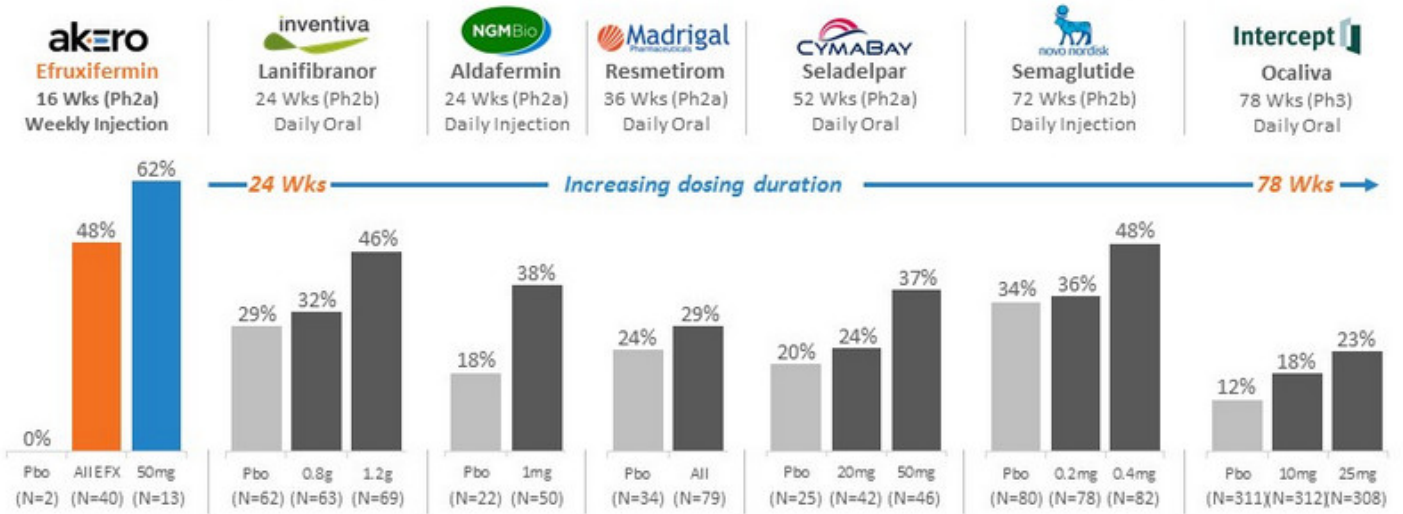


¹ FDA Guidance for Industry: Noncirrhotic Nonalcoholic Steatohepatitis With Liver Fibrosis: Developing Drugs for Treatment (2018)



NASH DEVELOPMENT LANDSCAPE: FIBROSIS IMPROVEMENT

Proportion of Subjects with ≥ 1 Stage Improvement in Fibrosis and No Worsening of NAS¹



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.

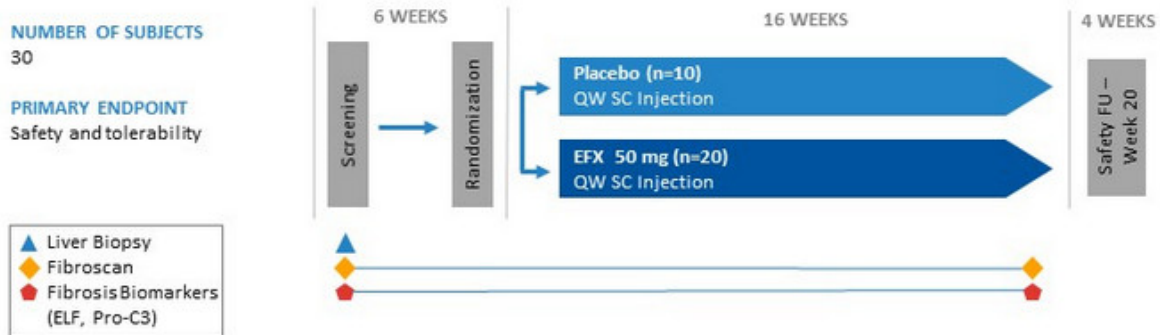
Inventiva (2020) June 16 Corporate Presentation; NGM Bio (2020) June 3 Corporate Presentation; Harrison, S et al. (2019) Lancet 394(10213):2012-24; Cymabay (2020) March 12 Press Release; Novo Nordisk (2020) June 19 R&D Investor Presentation; Younossi Z et al. (2019) Lancet 394(10215):2184-96. All trademarks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



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

F4 COHORT EXPANSION (COHORT C)

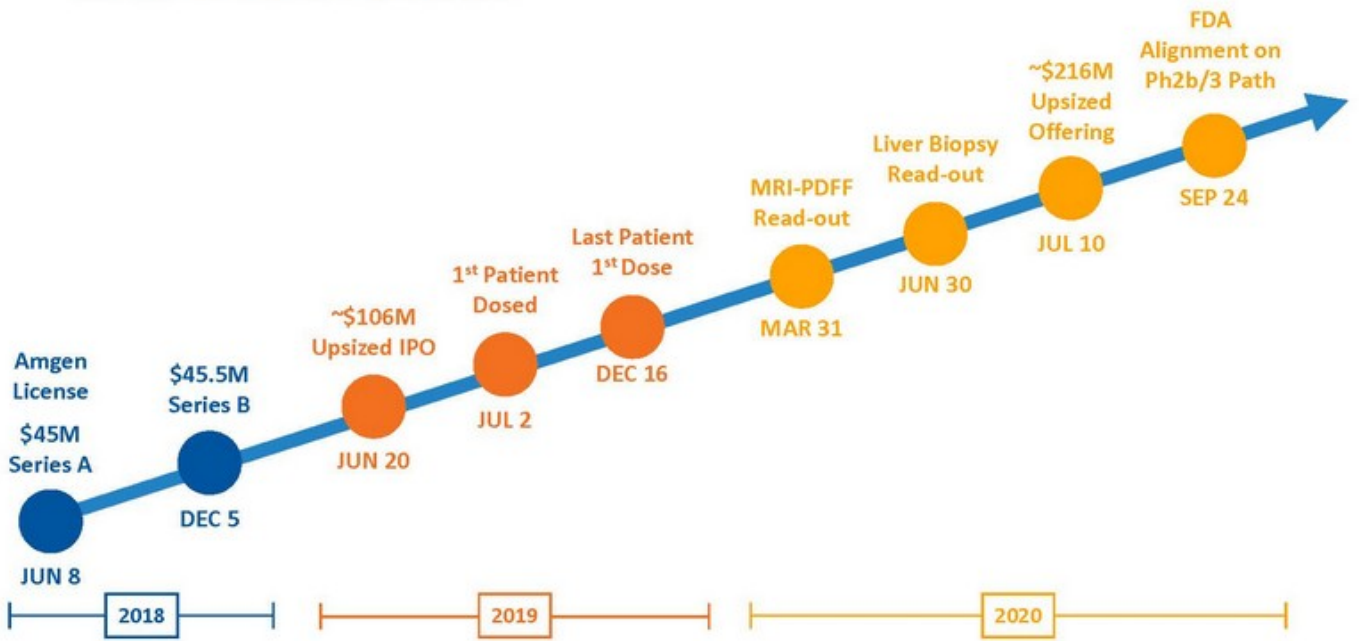
Screening of an additional cohort of patients with compensated cirrhosis (F4), Child-Pugh Class A, began on May 7, 2020; enrollment was completed on September 30, 2020



Selection of 50 mg dose based on PK-PD modeling of Phase 1b data, results of BALANCED main study, and availability of drug product



MILESTONES PROJECTED MILESTONES DELIVERED





STRONG FINANCIAL POSITION

COMPLETED UPSIZED FOLLOW-ON OFFERING

July 10, 2020

~\$106M

Raised in aggregate
gross proceeds

~\$216M

Raised in aggregate
gross proceeds

~\$306M*

cash, cash equivalents and short-
term marketable securities

** As of July 10, 2020, we had approximately \$305.6 million of cash, cash equivalents and short-term marketable securities. These amounts have not been audited, reviewed, or compiled by our independent registered public accounting firm. Our actual cash, cash equivalents and short-term marketable securities as of July 10, 2020 may differ from these amounts after we complete our comprehensive accounting procedures for the three months ended September 30, 2020.*



EFRUXIFERMIN: POTENTIALLY FOUNDATIONAL NASH MONOTHERAPY

- ✓ Substantial fibrosis improvement
- ✓ Substantial reductions in liver fat
 - Confirmed by NASH resolution
- ✓ Ameliorated dyslipidemia
 - No LDL cholesterol increase
- ✓ Improved glycemc control
- ✓ Weight loss across all dose groups
- ✓ Large, sustained reductions in ALT
- ✓ Few discontinuations due to AEs

