akero

A Global Disease, A Pioneering Treatment

Akero Therapeutics, Inc.

Corporate Presentation

September 2020

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; 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

Histological Improvements

- Response rates for all efruxifermin (EFX) treated subjects who achieved at least a 30% liver fat reduction and had end-of-treatment biopsies (N=40):
  - **48%** fibrosis improvement ≥1 stage and no worsening of NAS
  - **48%** NASH resolution and no worsening of fibrosis
  - **28%** fibrosis improvement ≥2 stage
  - **28%** for combination of fibrosis improvement ≥1 stage and NASH resolution

Safety & Tolerability

- EFX was generally well-tolerated (N=79) with no discontinuations due to treatment-emergent adverse events (TEAEs) in 50mg dose group
- Most frequent TEAEs were transient mild/moderate gastrointestinal events
- No treatment- or dose-related effects on blood pressure, heart rate, or bone mineral density

Improved Glycemic Control

- Significant improvements in HbA1c, HOMA-IR, C-Peptide, and Adiponectin

Weight Loss

- Reductions seen across all groups

Improved Dyslipidemia

- Significant improvements in triglycerides, HDL, and non-HDL cholesterol across all dose groups

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

akero

NASH: A SERIOUS AND DEBILITATING MULTI-SYSTEM DISEASE

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

A GROWING HEALTH EPIDEMIC

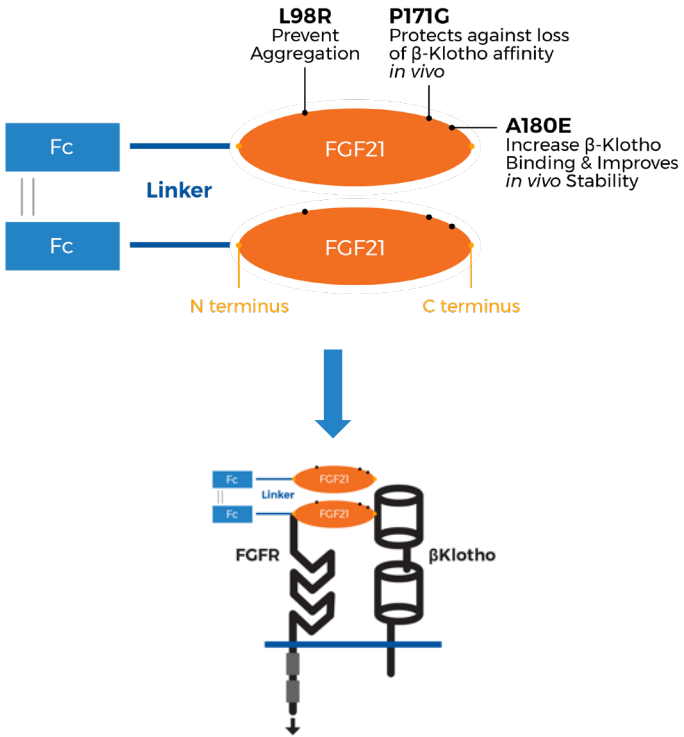
An estimated **17 million Americans** have NASH, with expectation that population will **grow >50% by 2030**

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

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

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

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



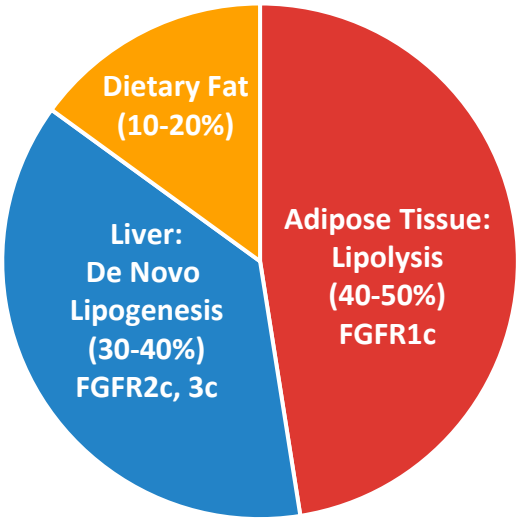
Key attributes

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

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

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

Sources of Fat Flowing into and Through Liver for NASH Patients



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

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

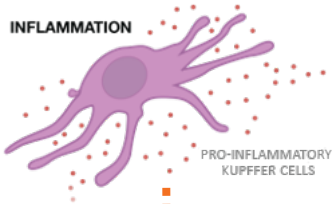
Source of Liver Fat	FGF Receptor	FGF21 Activity
Lipolysis	FGFR1c	✓
De Novo Lipogenesis	FGFR2c FGFR3c	✓

EFX DIRECT AND INDIRECT ANTI-FIBROTIC EFFECTS

Direct Effect

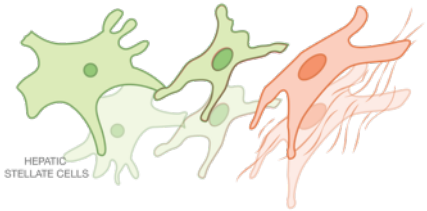
Indirect Effect

Kupffer Cell Activation

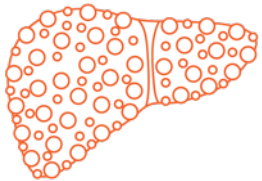


Hepatocyte Stress & Death

EFX



Myofibroblast Differentiation



Fibrosis

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

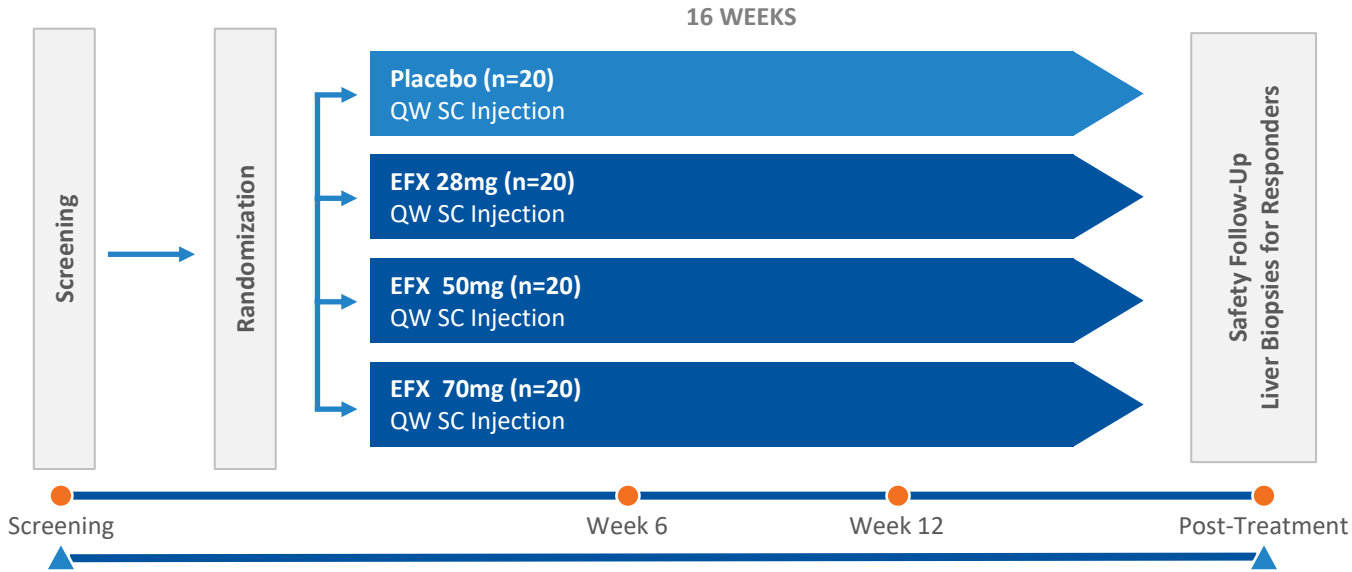
**Cited literature available on company website*

BALANCED STUDY TRIAL DESIGN

KEY INCLUSION CRITERIA
- F1-F3 NASH
- NAS ≥4
- Liver fat ≥10%

PRIMARY ENDPOINT
Absolute change from baseline in hepatic fat fraction (MRI-PDFF) at Week 12

PAIRED BIOPSIES
Subjects achieving ≥30% relative reduction of hepatic fat at week 12 are eligible for post-treatment biopsy



KEY SECONDARY EFFICACY ENDPOINTS
- Relative Liver Fat
- Response Rate
- ALT

KEY EXPLORATORY EFFICACY ENDPOINTS
- 2-Point NAS Improvement
- Fibrosis Improvement
- NASH Resolution
- Serum Pro-C3

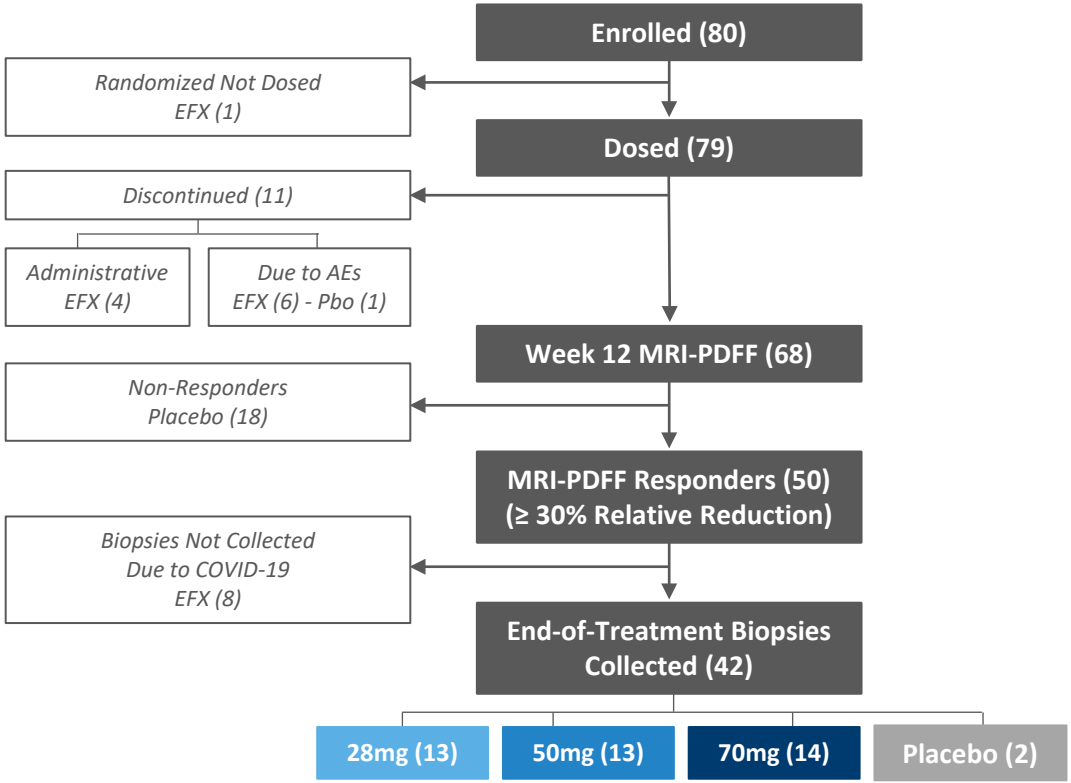
BASELINE DEMOGRAPHICS

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

Analysis Set	N	Definition
Full Analysis Set (FAS)	N=80	All subjects who were randomized into the study
Safety Set (SS)	N=79	All subjects who received at least one dose of study drug.
MRI-PDFF Evaluable Analysis Set (MAS)	N=68	All FAS subjects who have Baseline and Week 12 hepatic fat fraction assessed by MRI-PDFF
Liver Biopsy Evaluable Analysis Set (BAS)	N=42	All responders who have Baseline and end-of-treatment liver biopsy results

Source Data: FAS

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%

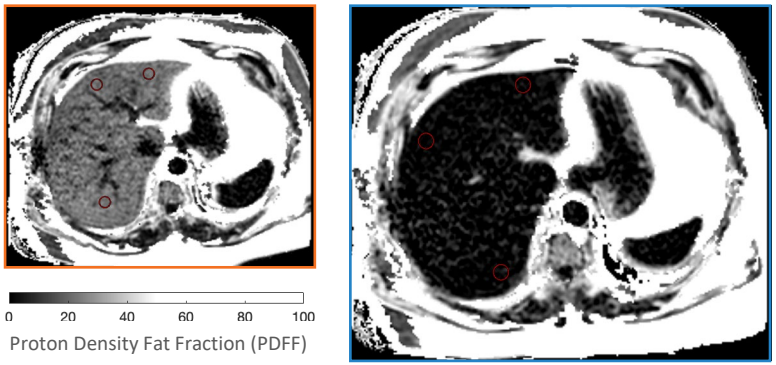
[1]Source Data: FAS; [2]Source Data: MAS

SUBSTANTIAL NORMALIZATION OF LIVER FAT AT WEEK 12

MRI-PDFF Images for Patient 207-012 (50mg EFX)

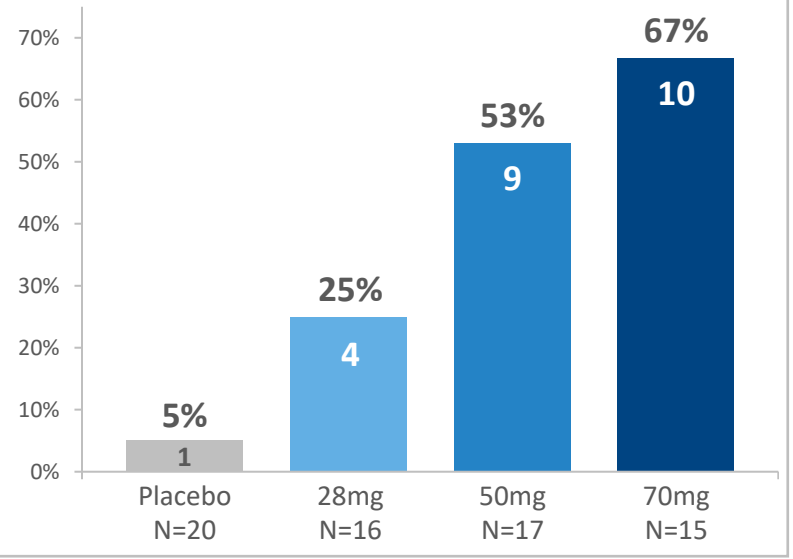
Baseline
22.2% Liver Fat

Week 12
2.0% Liver Fat (↓90%)



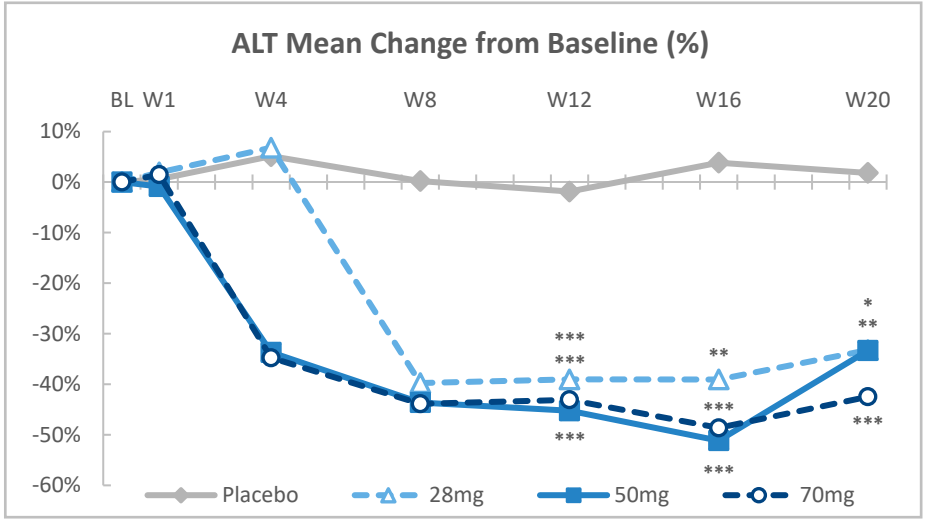
Analyzed with Liver*MultiScan*

Proportion of Patients Achieving Normalized Liver Fat (≤5% absolute liver fat content at Week 12)[1]



[1]Source Data: MAS

REDUCTION IN HEPATOCYTE STRESS AND COLLAGEN SYNTHESIS ACROSS ALL DOSE GROUPS



Similar dose-related improvements observed for AST, GGT, ALP

* p<0.05, ** p<0.01, *** p<0.001, versus placebo
(statistical significance tested only at Weeks 12, 16 and 20)

Serum Pro-C3

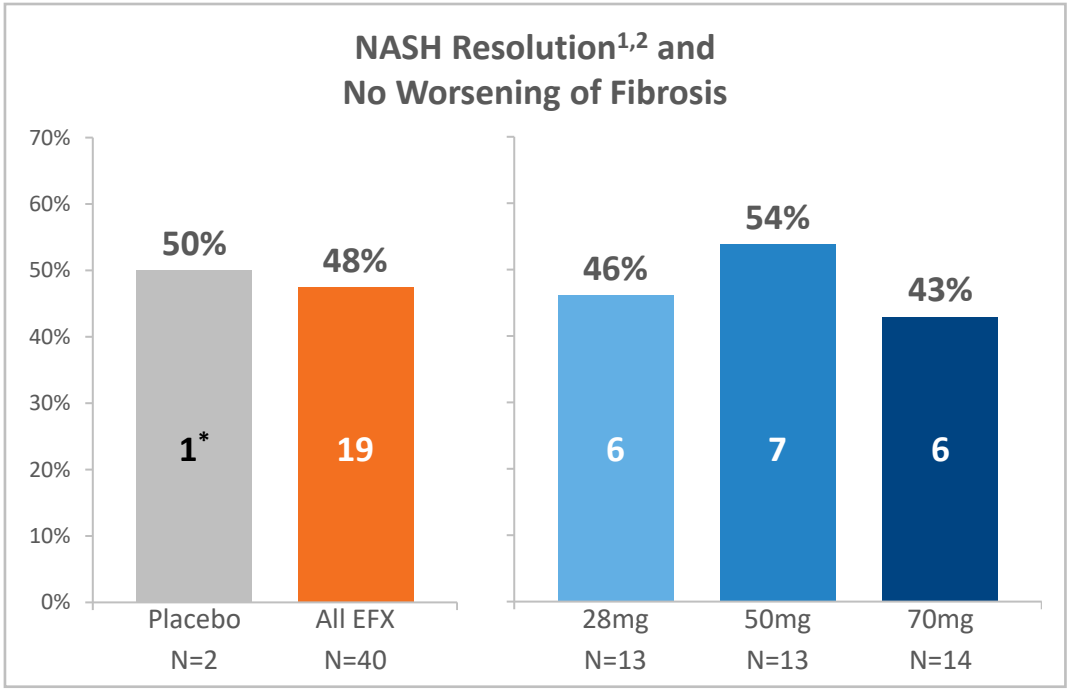
Mean Change from Baseline to Week 16

Placebo	+4%
28mg	-34%***
50mg	-27%**
70mg	-32%***

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

Source Data: FAS; ANCOVA LS Mean

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



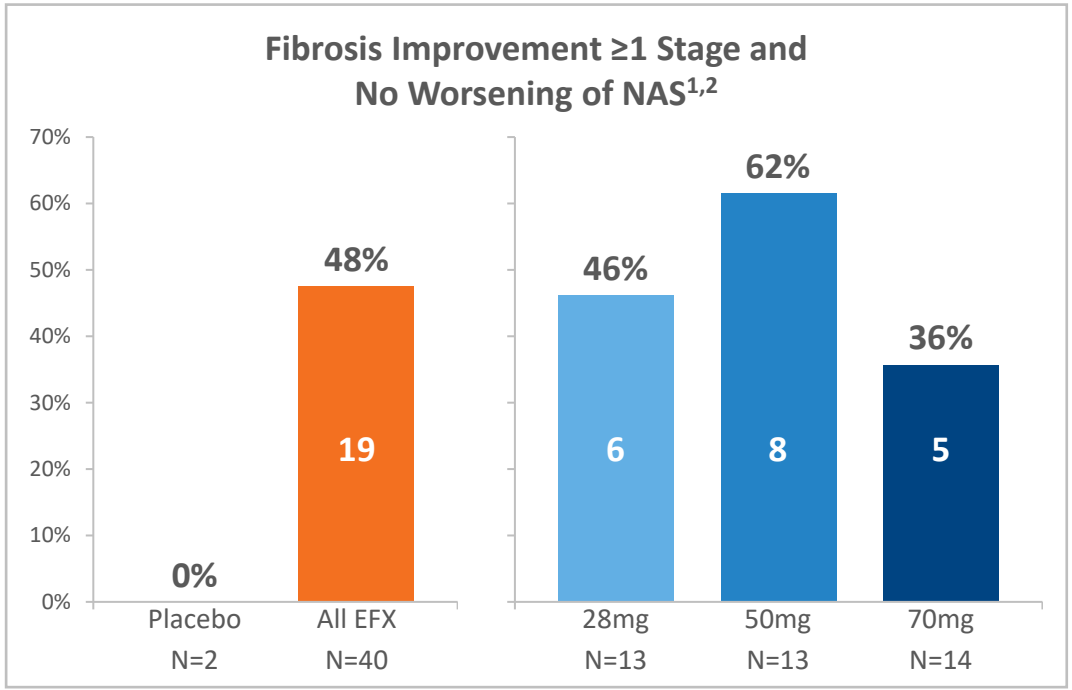
Biopsy Reading

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

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

Source Data: BAS

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

Source Data: BAS

SAFETY OVERVIEW

Treatment-Emergent Adverse Event (TEAE) Classification	Placebo (N=21)	EFX 28mg (N=19)	EFX 50mg (N=19)	EFX 70mg (N=20)
TEAE Leading to Death	0	0	0	0
TEAE Leading to Discontinuation	1[a]	2[b]	0	4[c]
Serious Adverse Event (SAE)	0	1[d]	0	1

[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: SS

DRUG-RELATED TREATMENT-EMERGENT ADVERSE EVENTS (TEAEs)

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%)	19 (33%)	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%)

*Across EFX dose groups

Gastrointestinal Adverse Events:

- Majority were transient, Grade 1, with on-drug resolution
- Often single episodes
- Overall frequency decreased over treatment period
- No study discontinuations due to diarrhea

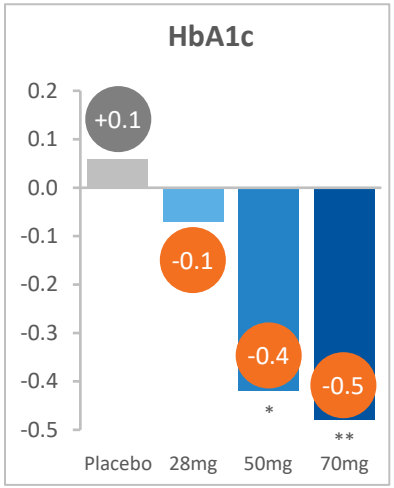
No Treatment-Related Effects On:

- Heart Rate
- Systolic Blood Pressure
- Diastolic Blood Pressure
- Bone mineral density

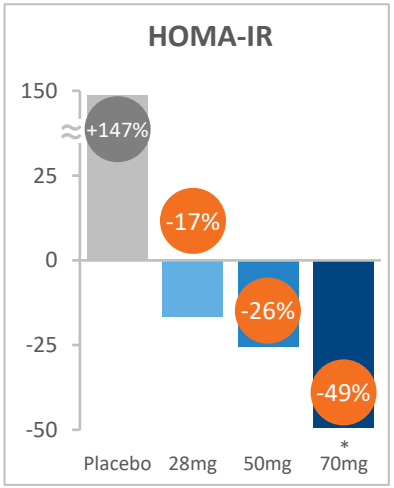
Source Data: SS

CLINICALLY MEANINGFUL IMPROVEMENTS IN GLYCEMIC CONTROL AFTER 16 WEEKS

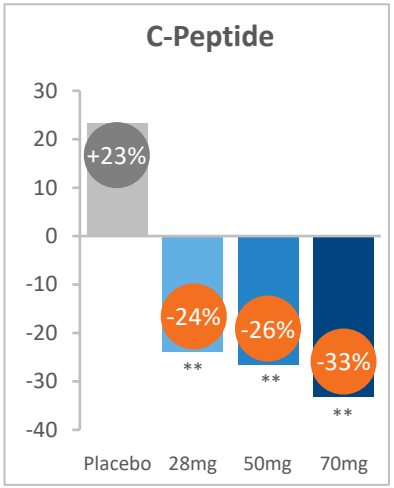
Mean Change From Baseline to Week 16 (%)[1]



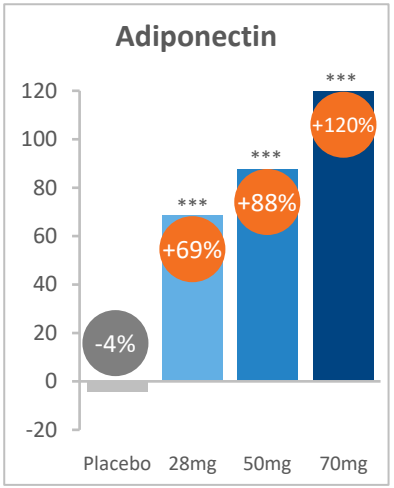
* p<0.05, ** p<0.01, versus placebo



* p<0.05, versus placebo



** p<0.01, versus placebo

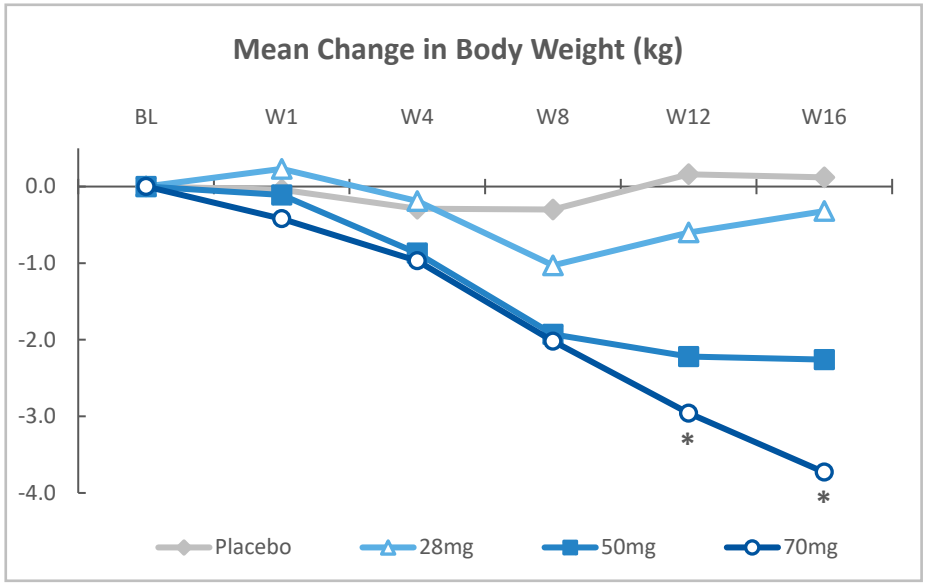


*** p<0.001, versus placebo

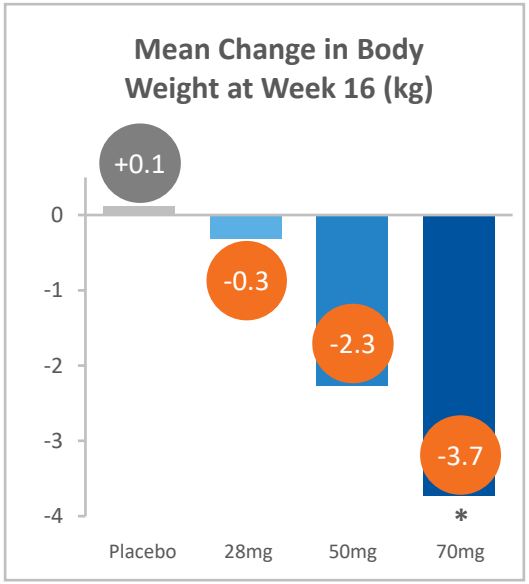
[1] HbA1c is presented in absolute percent change from baseline, whereas HOMA-IR, C-Peptide, and Adiponectin are presented in relative percent change from baseline

Source Data: FAS; ANCOVA LS Mean

WEIGHT LOSSES OBSERVED FOR ALL DOSE GROUPS: FIRST REPORT OF SIGNIFICANT WEIGHT LOSS FOR FGF21 CLASS



* p<0.05, versus placebo
(statistical significance tested only at Weeks 12 and 16)

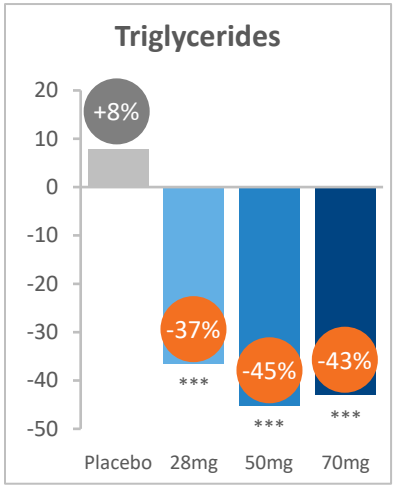


* p<0.05, versus placebo

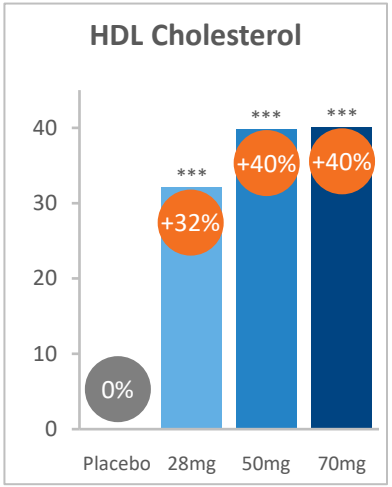
Source Data: FAS; ANCOVA LS Mean

IMPROVED LIPOPROTEIN PROFILE FOR CARDIOVASCULAR HEALTH

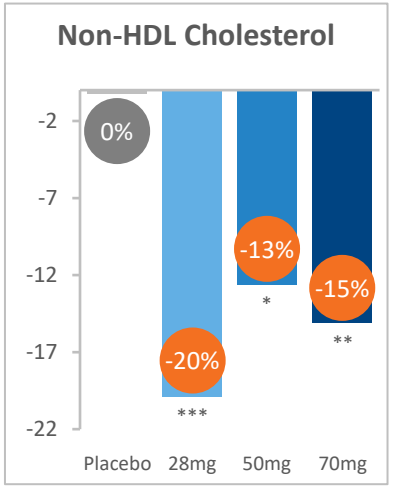
Mean Change From Baseline to Week 16 (%)



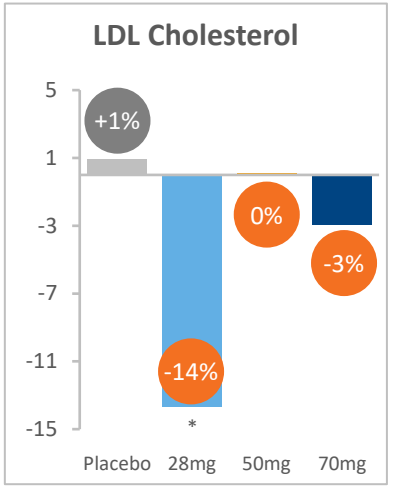
*** p<0.001, versus placebo



*** p<0.001, versus placebo



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

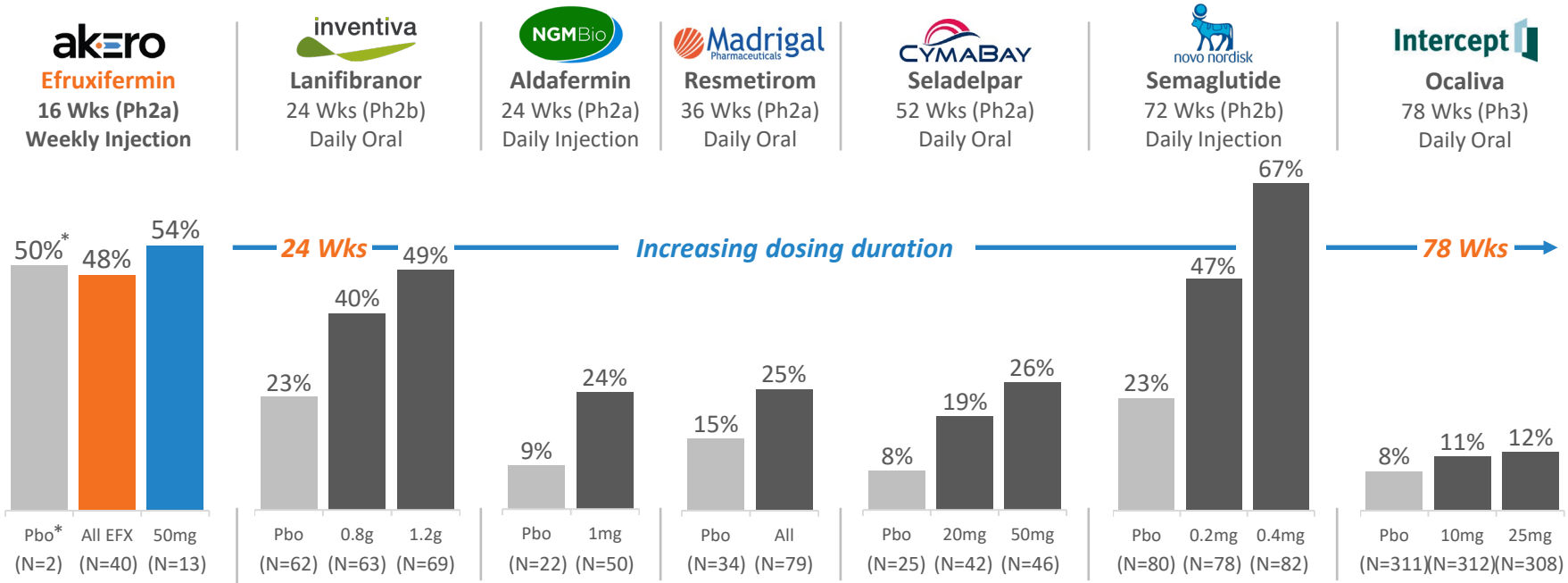


* p<0.05, versus placebo

Source Data: FAS; ANCOVA LS Mean

DEVELOPMENT LANDSCAPE: NASH RESOLUTION

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



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

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

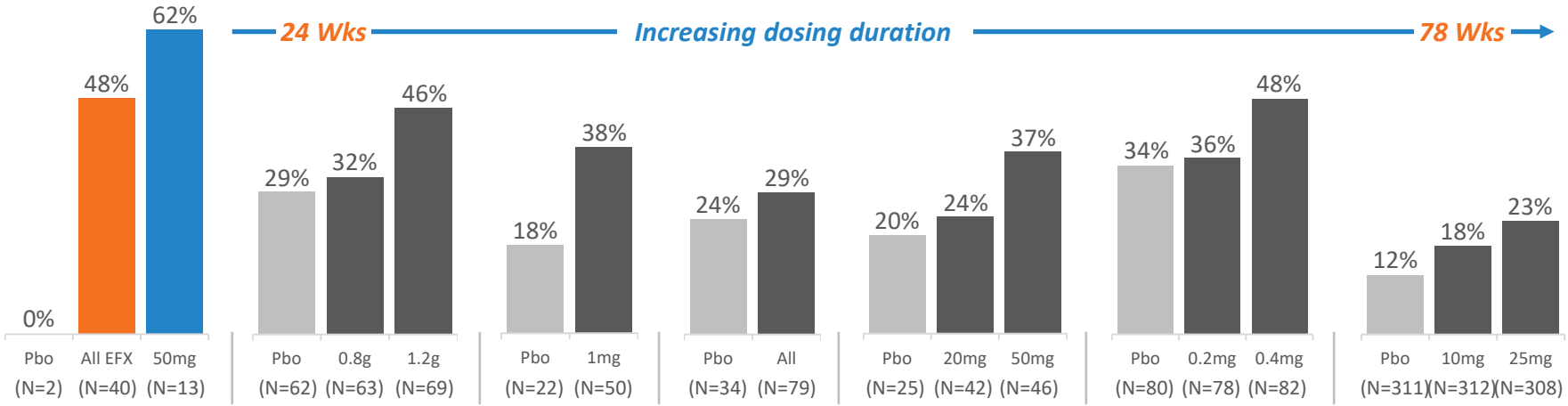
Inventiva (2020) June 16 Corporate Presentation; NGM Bio (2020) June 3 Corporate Presentation; Harrison, S et al. (2019) Lancet 394(10213):2012-24; CymaBay (2020) March 12 Press Release; Novo Nordisk (2020) June 19 R&D Investor Presentation; Younossi Z et al. (2019) Lancet 394(10215):2184-96. All trademarks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

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

DEVELOPMENT LANDSCAPE: FIBROSIS IMPROVEMENT

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

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

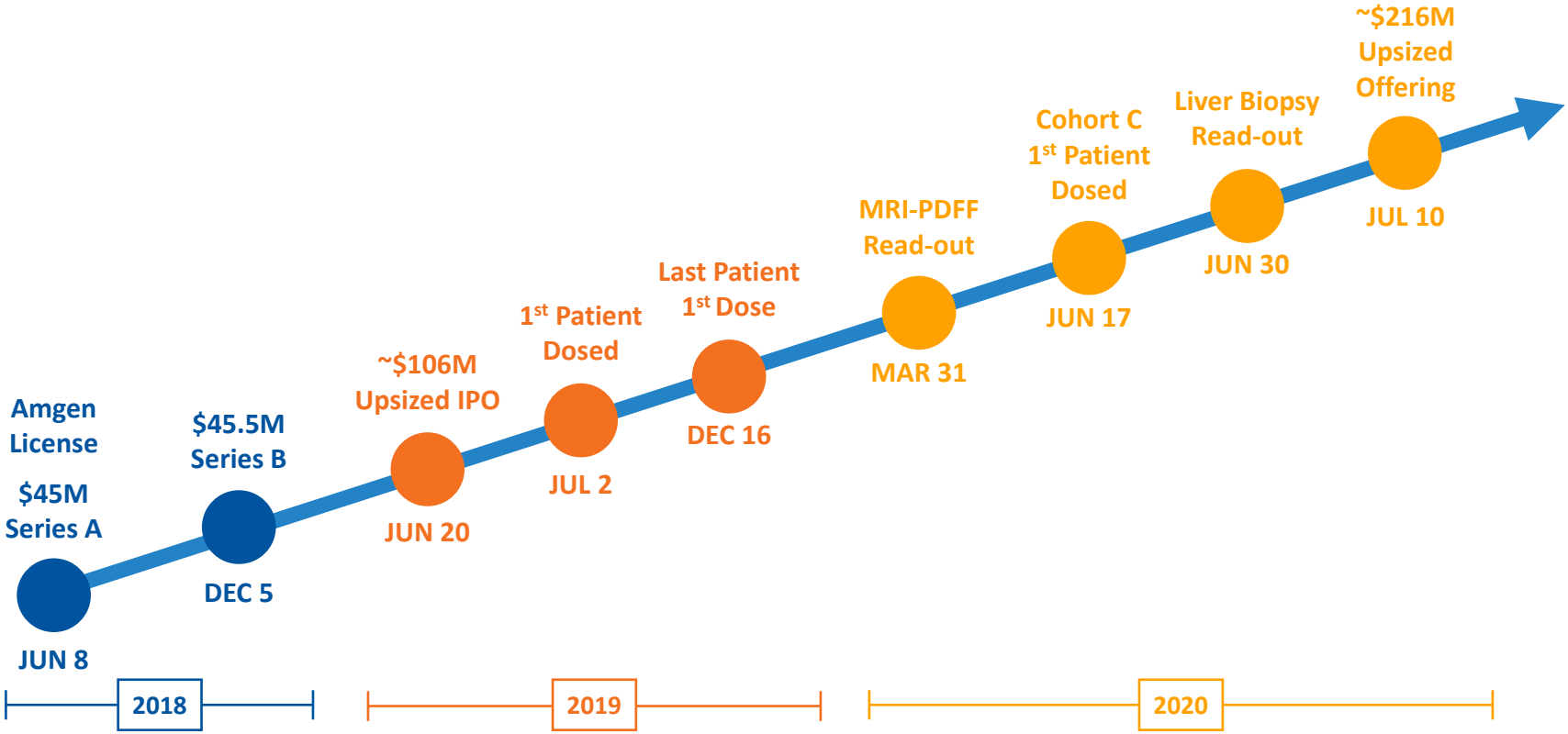


Note: These data are derived from different clinical trials at different points in time, with differences in trial design and patient populations. No head-to-head clinical trials have been conducted.

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

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

MILESTONES PROJECTED
MILESTONES DELIVERED



STRONG FINANCIAL POSITION

COMPLETED UPSIZED IPO

June 20, 2019

~$106M

Raised in aggregate gross proceeds

$16

Priced upsized IPO at top of marketing range

COMPLETED UPSIZED FOLLOW-ON OFFERING

July 10, 2020

~$216M

Raised in aggregate gross proceeds

$36

Priced upsized offering at top of marketing range

CASH ON HAND

July 10, 2020

~$306M*

cash, cash equivalents and short-term marketable securities

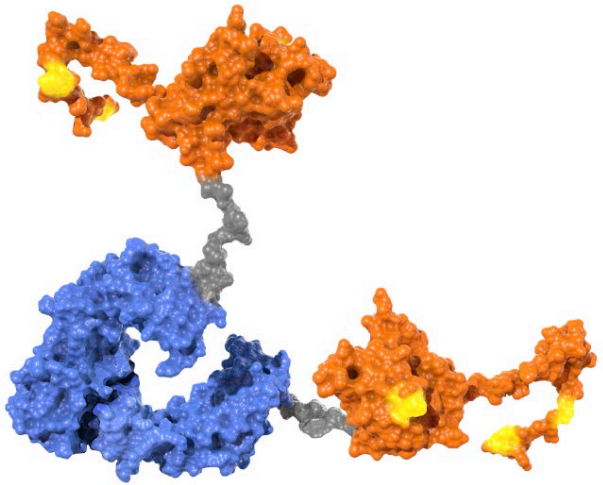
Not audited, reviewed, or compiled by our independent registered public accounting firm.*

** 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.*

akero

EFRUXIFERMIN:
POTENTIALLY FOUNDATIONAL NASH MONOTHERAPY

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



akero

akero

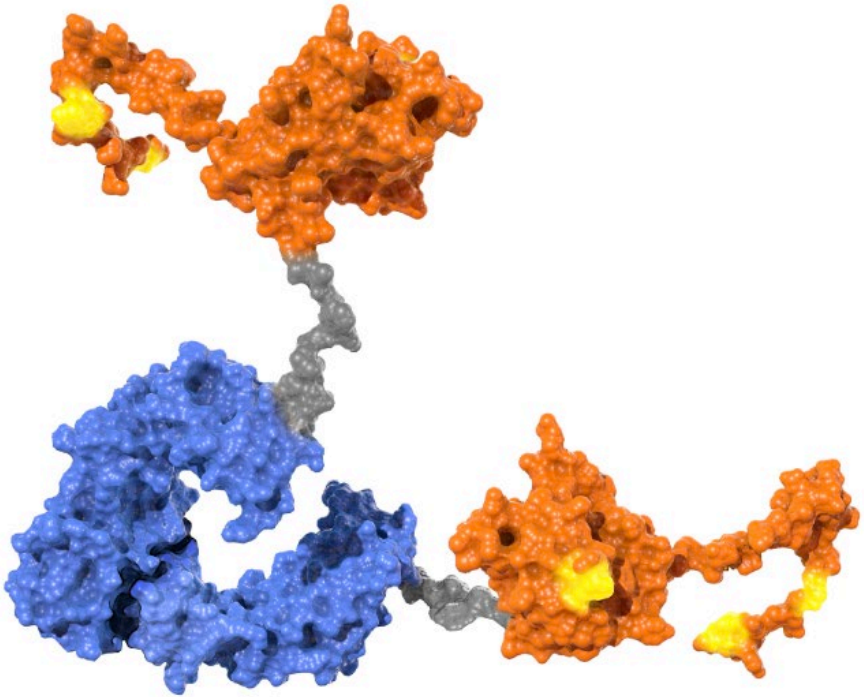
A Global Disease, A Pioneering Treatment

NASDAQ: AKRO

akero

BACKUP SLIDES

INTRODUCING EFRUXIFERMIN



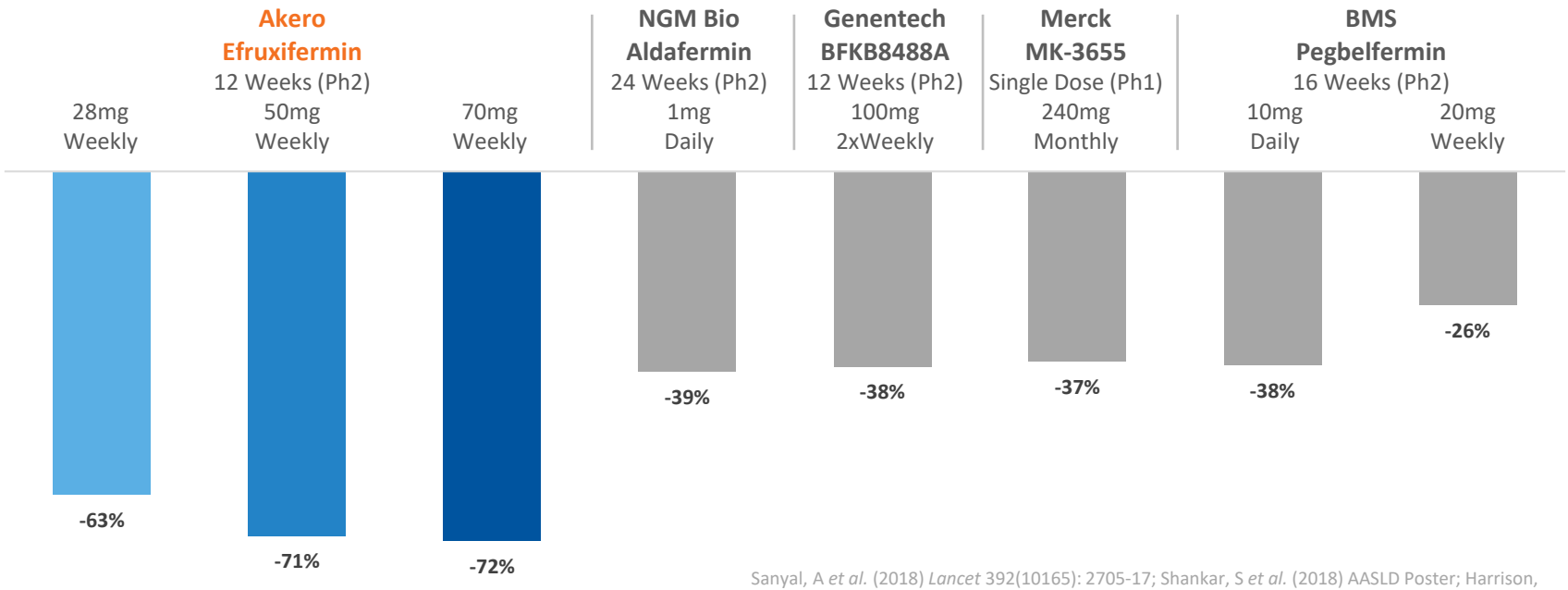
Efruxifermin
(EFX)

ē-FRUX-i-FER-min

(Formerly AKR-001)

DEVELOPMENT LANDSCAPE FOR RELATIVE REDUCTION IN LIVER FAT

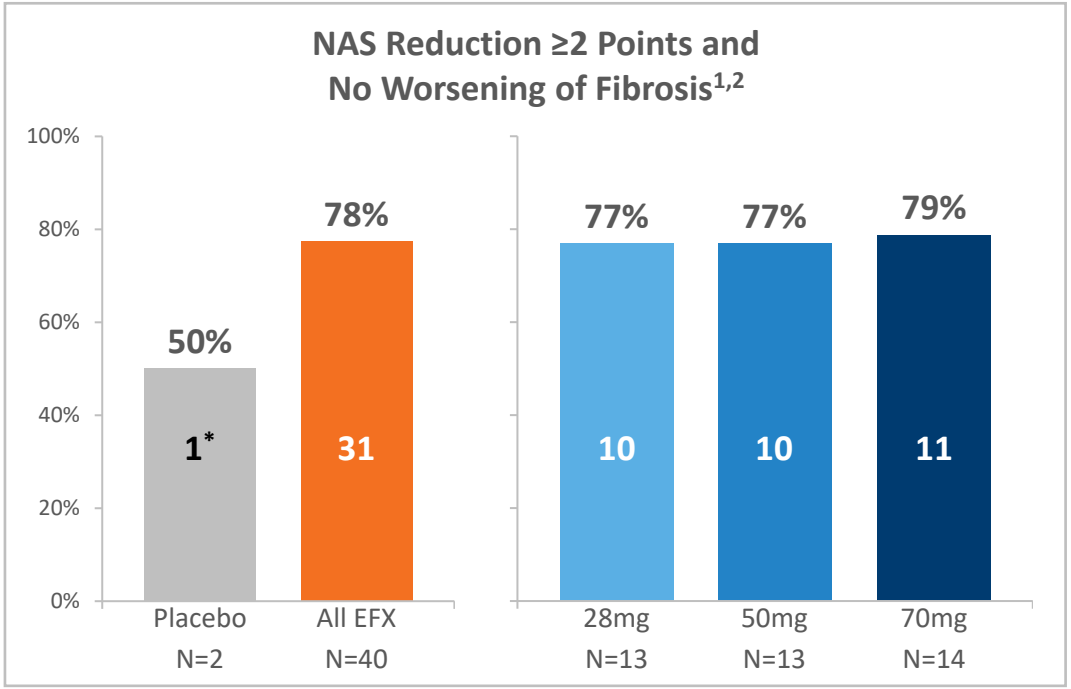
Relative Fat Reduction (MRI-PDFF) of FGFs



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, A *et al.* (2018) *Lancet* 392(10165): 2705-17; Shankar, S *et al.* (2018) AASLD Poster; Harrison, S *et al.* (2018) EASL Presentation; Ge, H *et al.* (2014) *J Biol Chem* 289(44): 30470-80; Yu, X *et al.* (2013) *PLOS ONE* 8(7): e66923; Wu, X *et al.* (2013) *J Lipid Res* 54(2): 325-32; Wu, A-L *et al.* (2013) *PLOS ONE 6(3): e17868;* Wu, X *et al.* (2009) *Proc Natl Acad Sci* 106(34): 14379-84; Huang, X *et al.* (2007) *Diabetes* 56(10): 2501-10; Kunder et al. (2019) AASLD Poster; NGM (2019) October 7 Corporate Presentation.

CONSISTENT IMPROVEMENT IN STEATOHEPATITIS



[1] Endpoint recommended by FDA for Phase 2 clinical trials in NASH (F1-F3)
[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)

Mean NAS Reduction

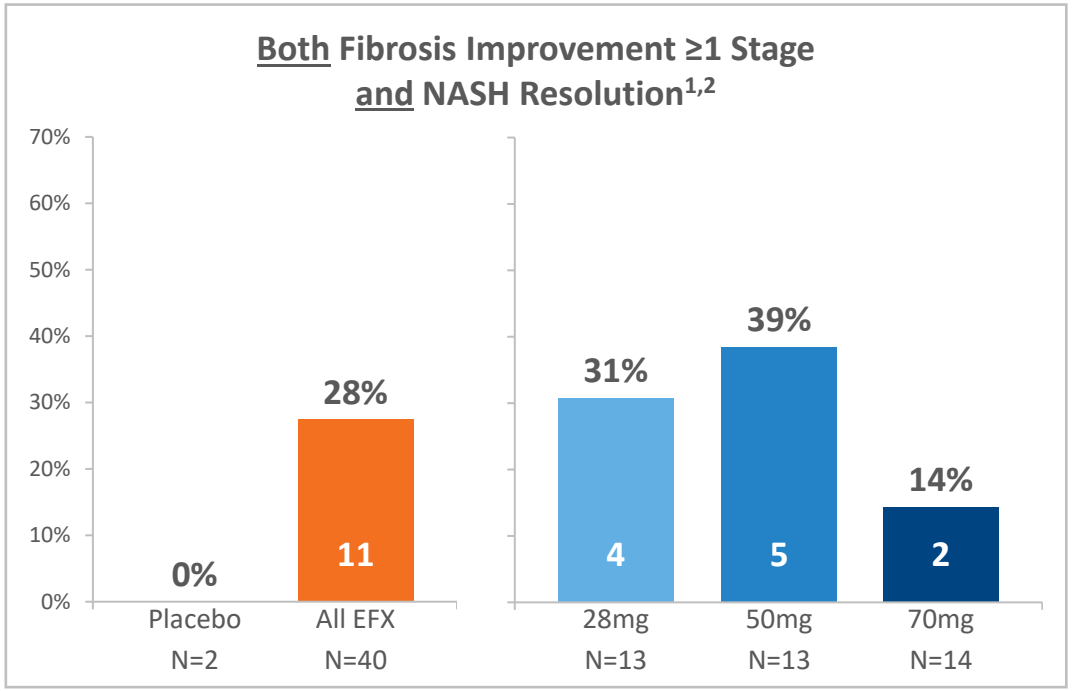
Change in NAS from Baseline after 16 weeks of dosing

Placebo	-2.5
28mg	-2.9
50mg	-3.1
70mg	-3.6

The placebo arm was enriched for NAS endpoints because only 10% of placebo patients met the MRI-PDFF responder definition and had an end-of-treatment biopsy

Source Data: BAS

ENCOURAGING RESPONSE RATES FOR BOTH FIBROSIS IMPROVEMENT AND NASH RESOLUTION AFTER 16 WEEKS



[1] Subjects who achieve a NAS score of 0 or 1 for lobular inflammation and a score of 0 for ballooning AND Improvement in liver fibrosis greater than or equal to one stage

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

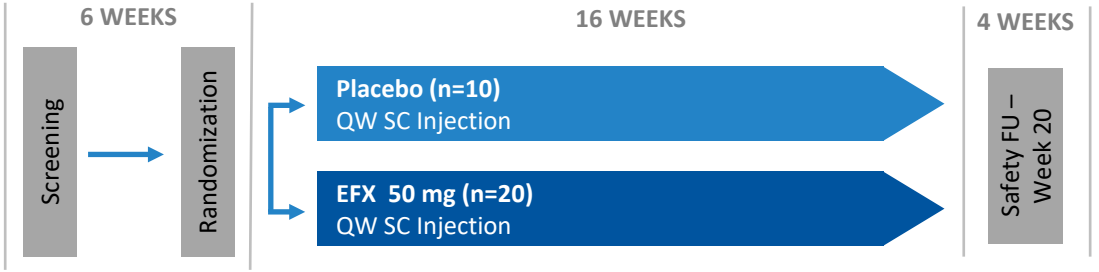
Source Data: BAS

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; the first patient was dosed on June 17, 2020

NUMBER OF SUBJECTS
30

PRIMARY ENDPOINT
Safety and tolerability



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