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A Global Disease,
A Pioneering Treatment

Corporate Presentation

September 2021

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

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NASH: A SERIOUS AND DEBILITATING MULTI-SYSTEM DISEASE

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

A GROWING HEALTH EPIDEMIC

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

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

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

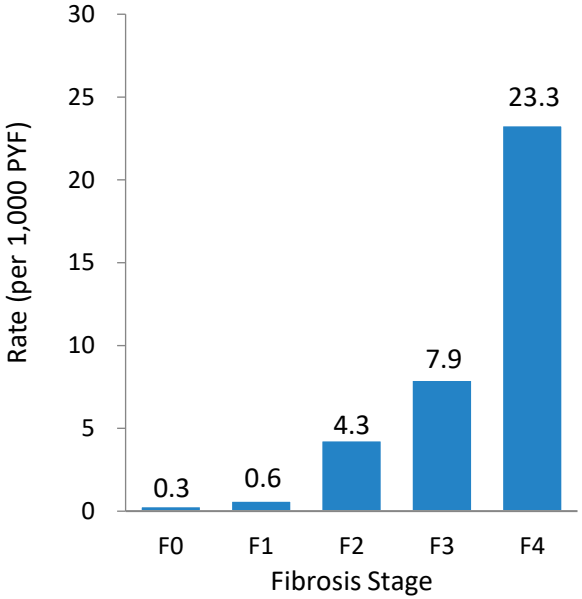
The **leading cause of death** for NASH patients is cardiovascular disease

CIRRHOTIC NASH PATIENTS HAVE POOR PROGNOSIS

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

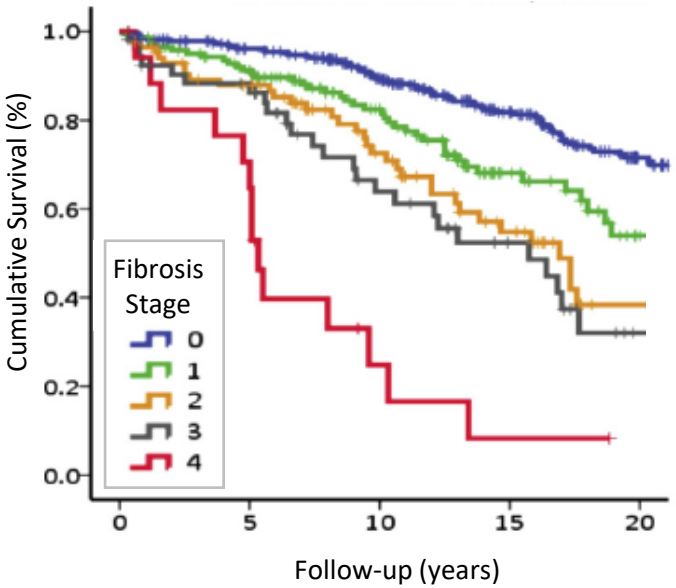
~60% 5-year mortality for F4 NASH patients absent transplant

Liver-Related Mortality



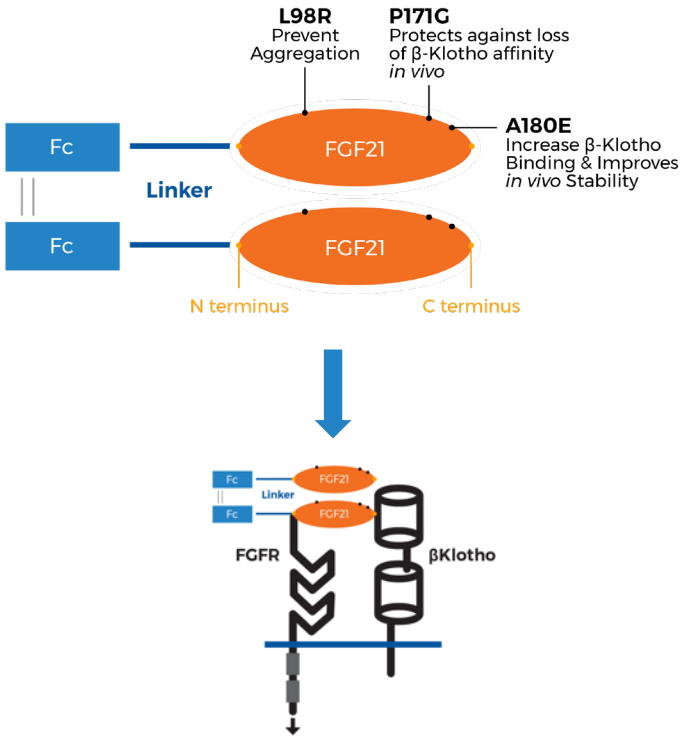
Dulai, PS et al. (2017) Hepatology 65:1557-65

Survival Free of Liver Transplantation



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

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



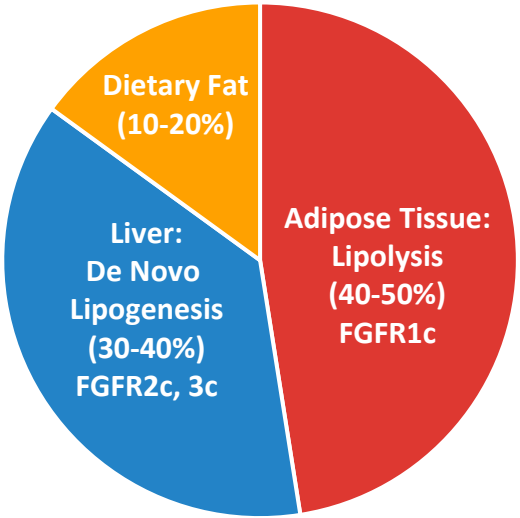
Key attributes

- 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

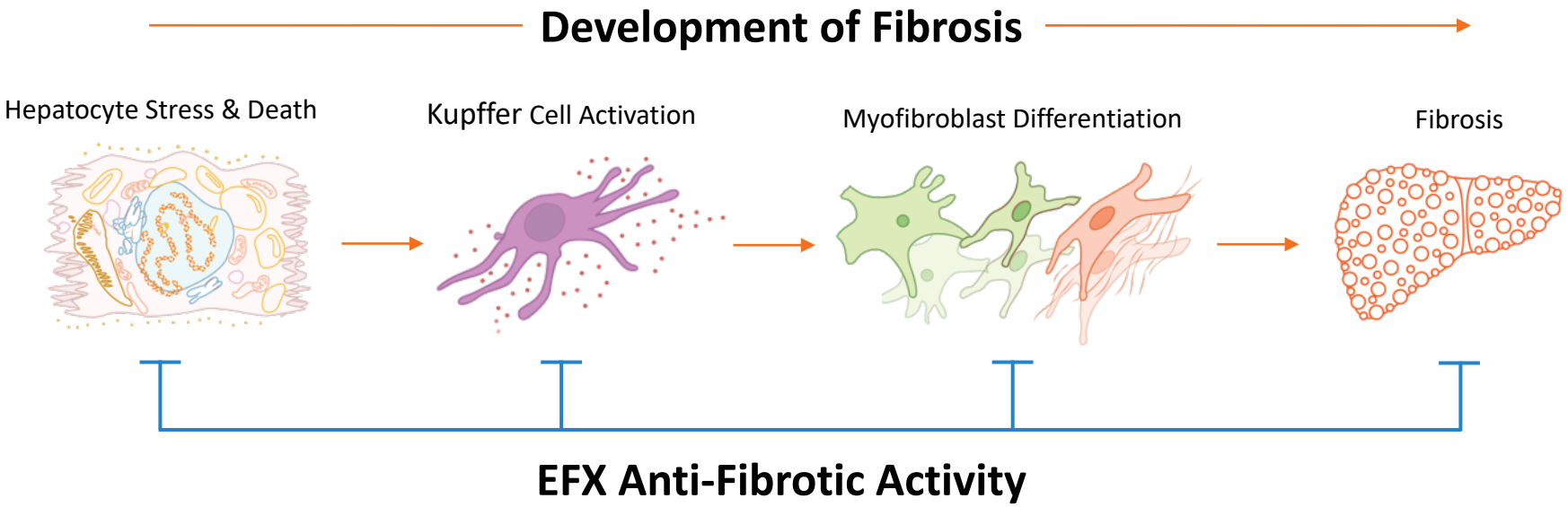


Lambert, JE *et al.* (2014) *Gastroenterology* 146(3): 726-35

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	✓

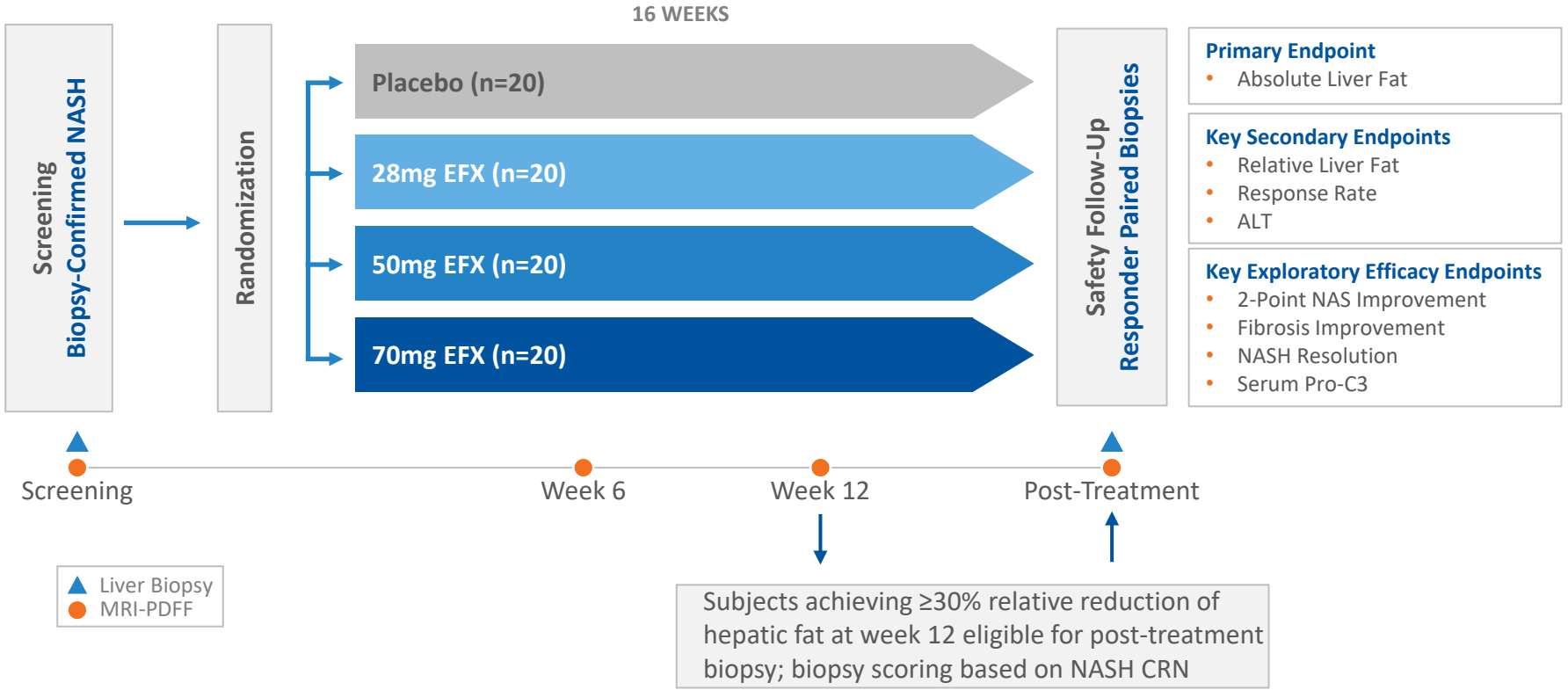
EFX DIRECT AND INDIRECT ANTI-FIBROTIC EFFECTS



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

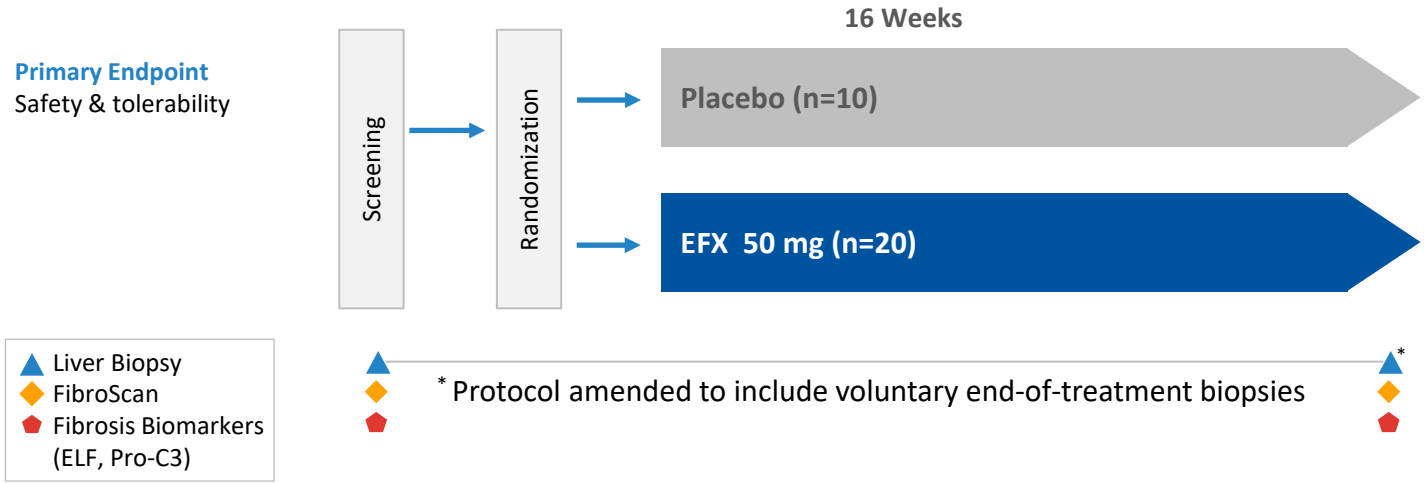
**Cited literature available on company website*

PHASE 2A TRIAL (BALANCED) DESIGN (F1-F3 NASH)



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 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)

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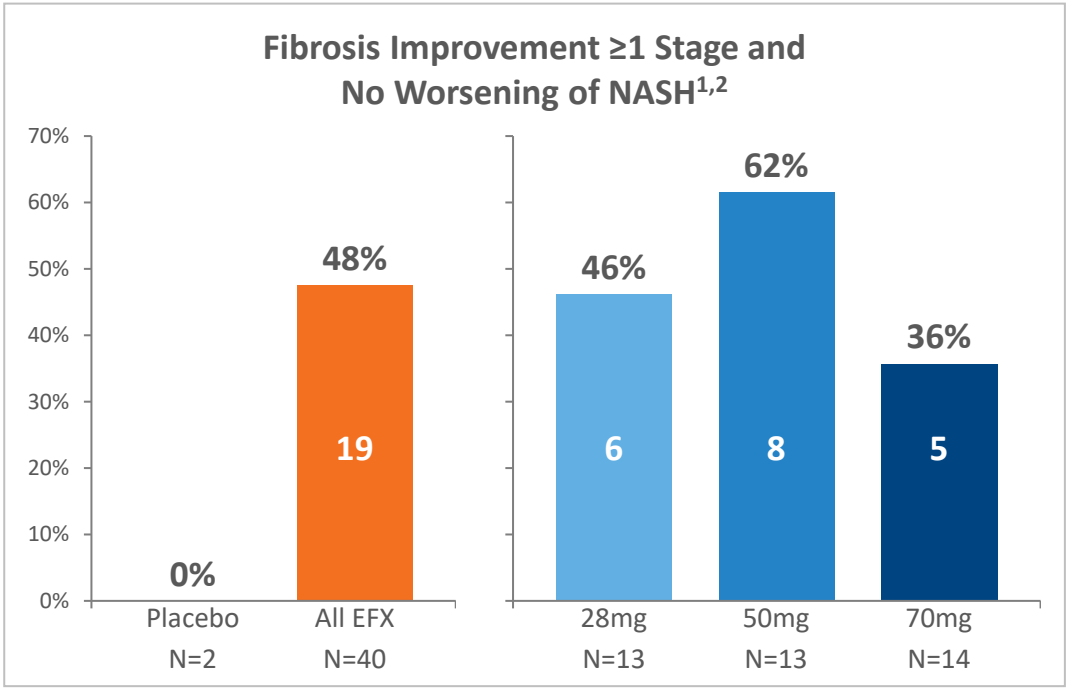
HIGH RATES OF FIBROSIS IMPROVEMENT AFTER 16 WEEKS ACROSS ALL DOSE GROUPS (F1-F3 NASH)



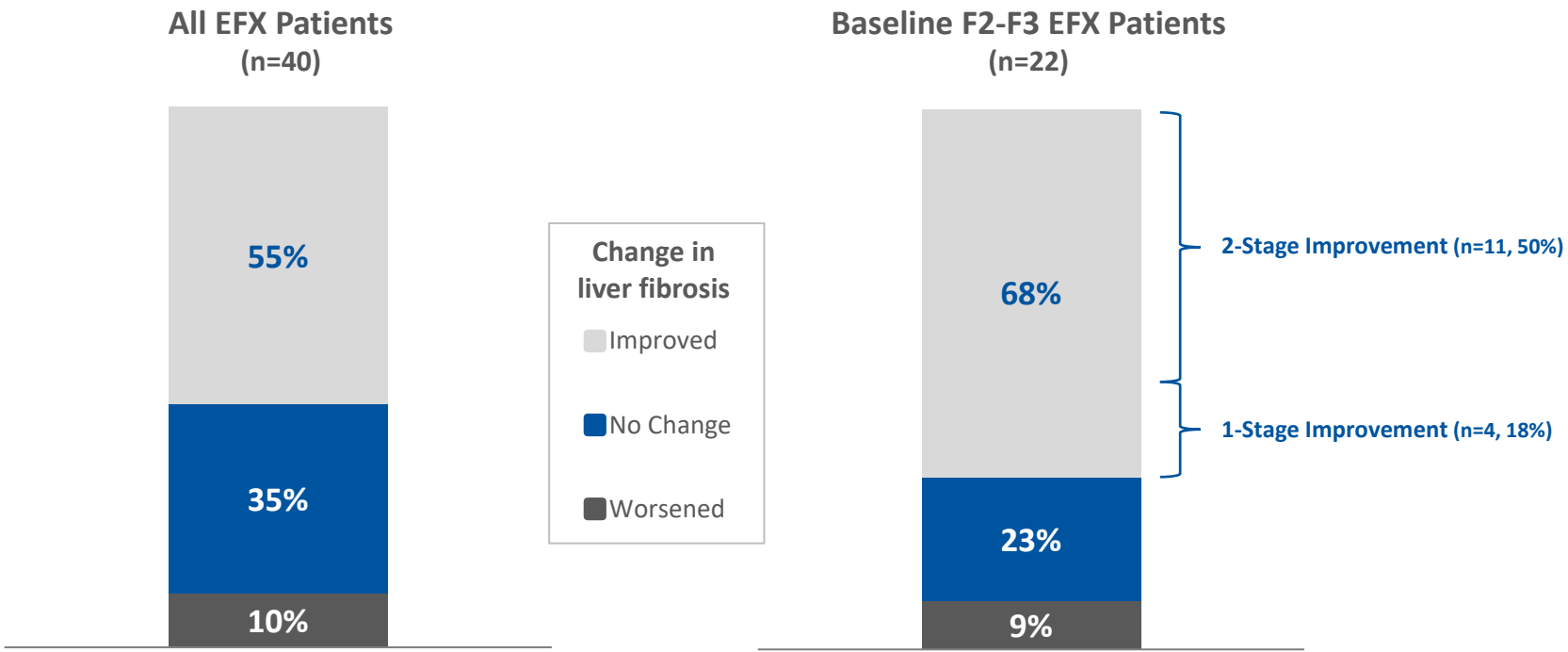
Biopsy Reading

- 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

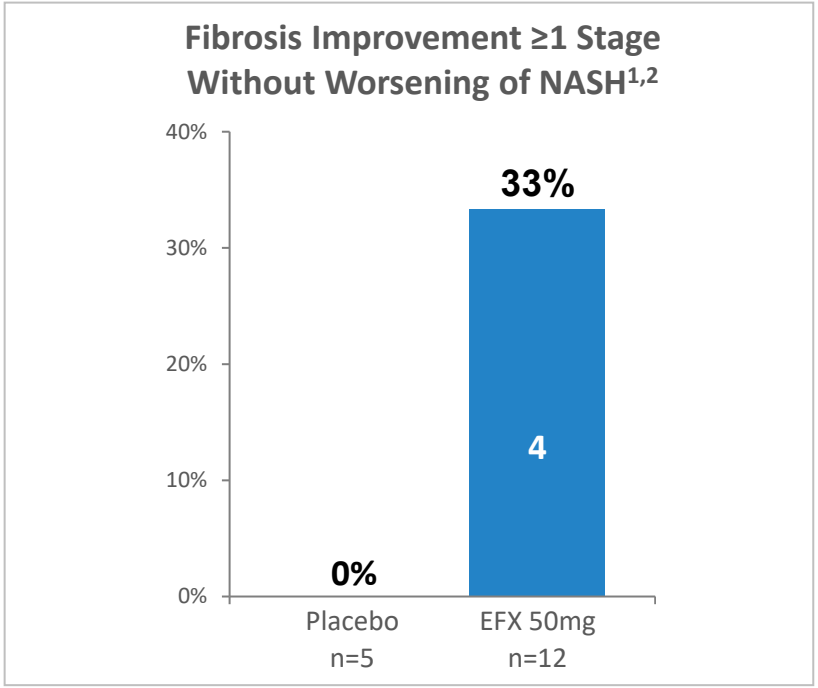
Source Data: Liver Biopsy Evaluable Analysis Set, F1-F3 (all BALANCED main study responders who had baseline and end-of-treatment liver biopsy results)

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

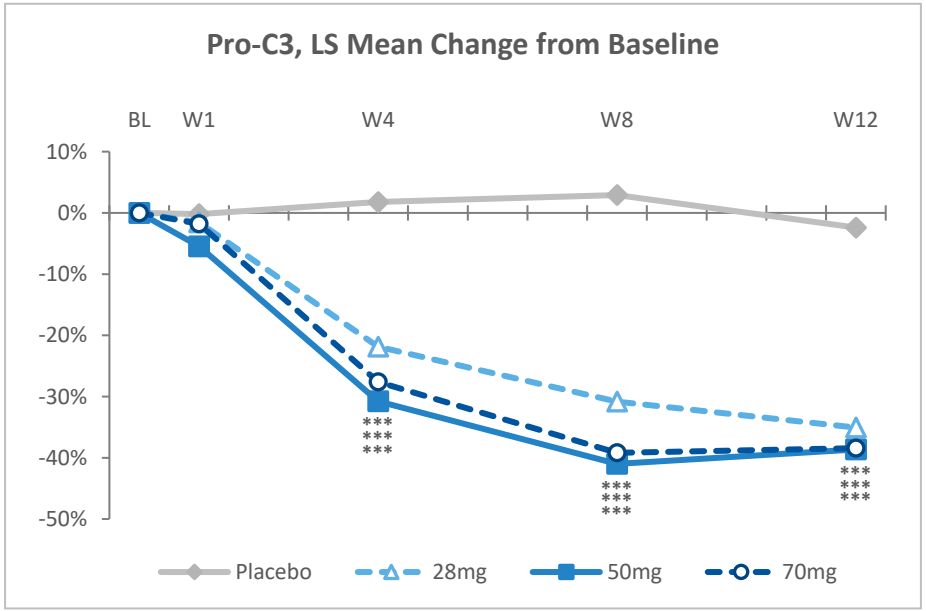
- All baseline and end-of-treatment biopsies were read by a single NASH-CRN pathologist
- All biopsies were read blinded to both treatment assignment and patient
- Baseline biopsies were read independently of end-of-treatment biopsies, in random fashion and not paired

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

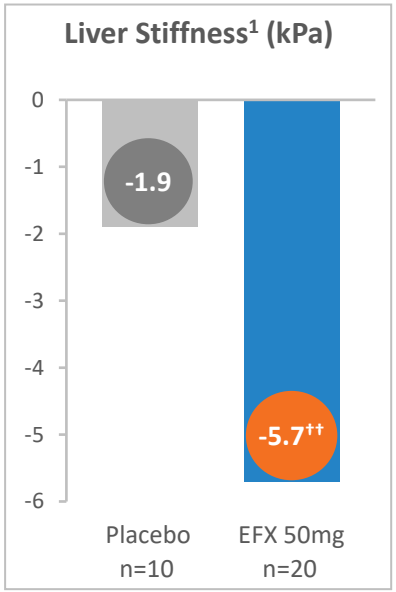
Dose Group	Baseline	Δ Week 12
Placebo	9.4	0.0
28mg	9.5	-0.7***
50mg	9.5	-0.8***
70mg	9.6	-0.4*

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

Source Data: Full Analysis Set, F1-F3

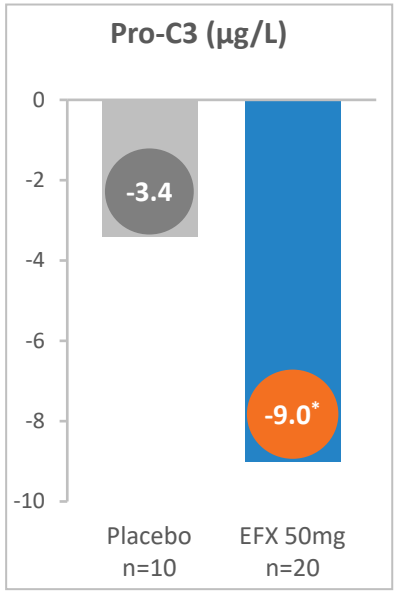
IMPROVEMENTS IN FIBROSIS BIOMARKERS IN CIRRHOTIC NASH PATIENTS SUPPORT HISTOLOGY RESULTS (F4 NASH)

LS Mean Change From Baseline to Week 16

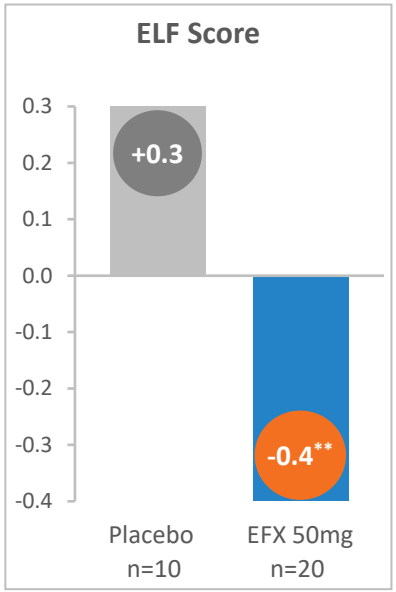


[1] Measured by FibroScan

†† p<0.01, versus baseline (ANCOVA)



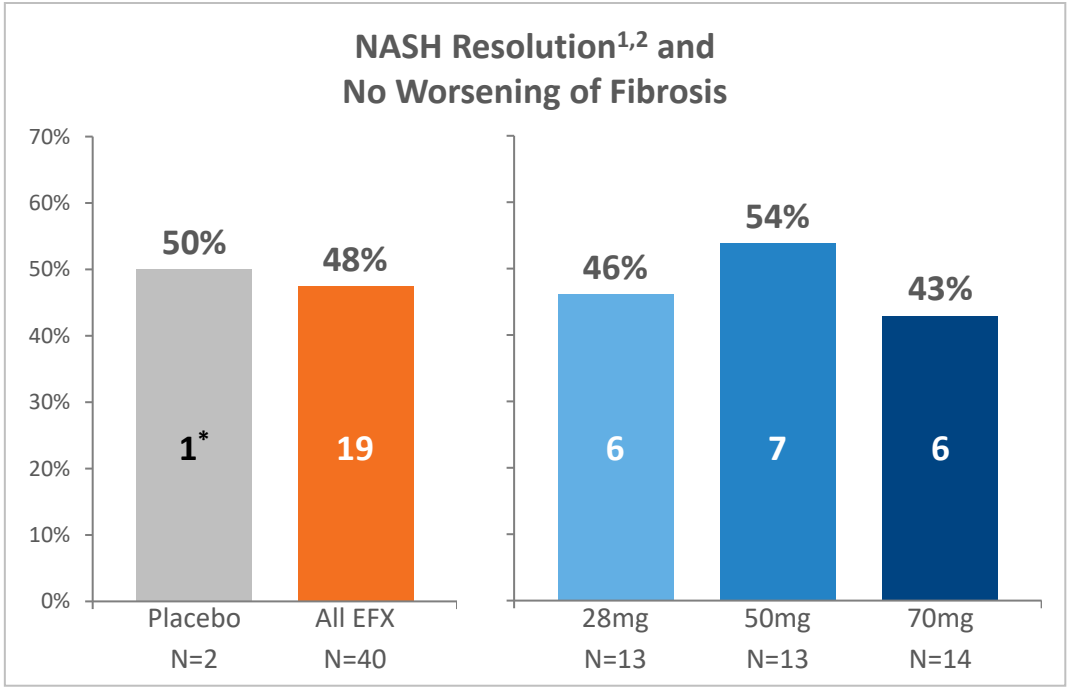
* p<0.05, versus placebo (ANCOVA)



** 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*

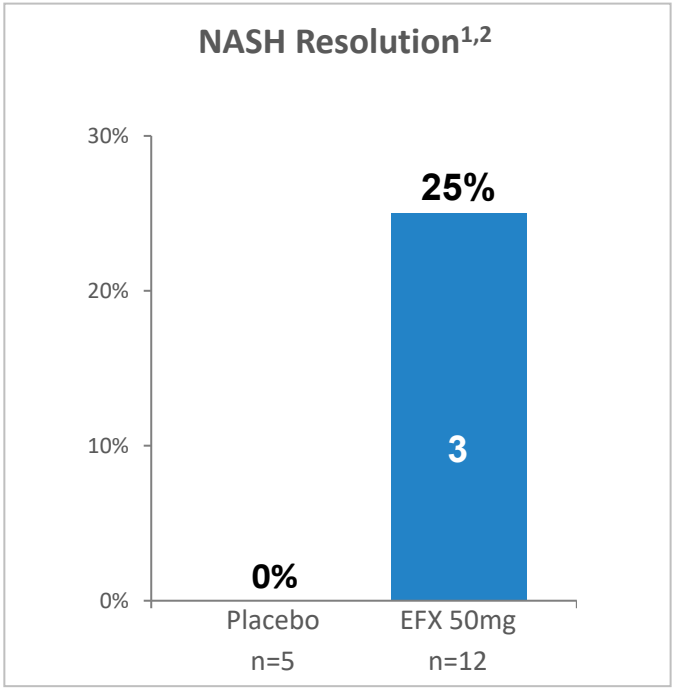
HIGH RESPONSE RATES ON NASH RESOLUTION AFTER 16 WEEKS ACROSS ALL DOSE GROUPS (F1-F3 NASH)



[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

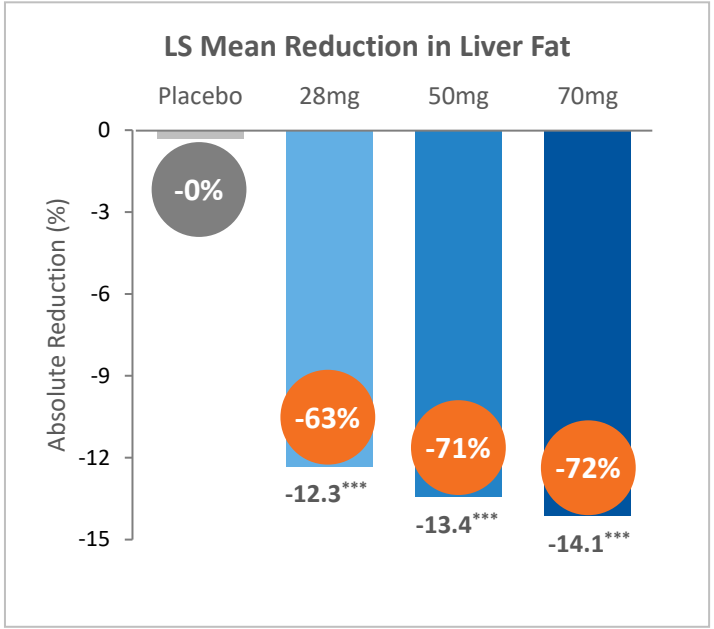
EFX Subject	Baseline NAS	Week 16 NAS
A	7	1
B	3	1
C	6	1

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)

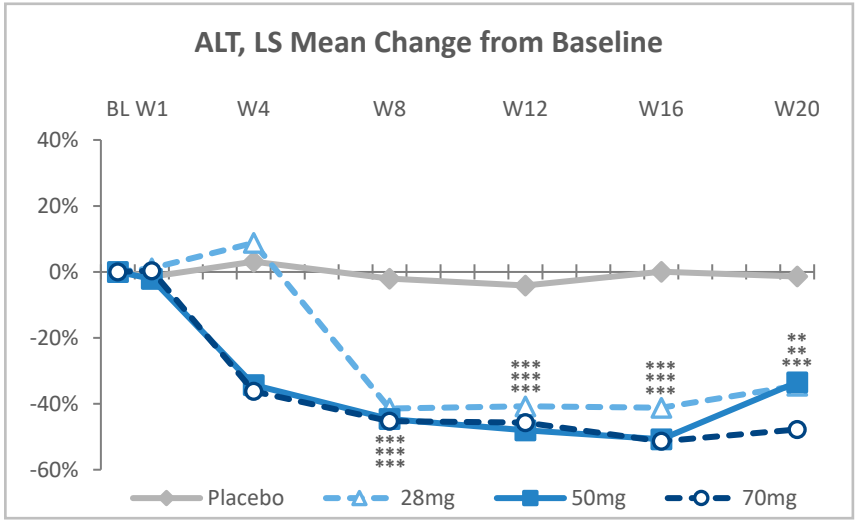
Proportion of Patients Achieving Fat Reduction Thresholds

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%***

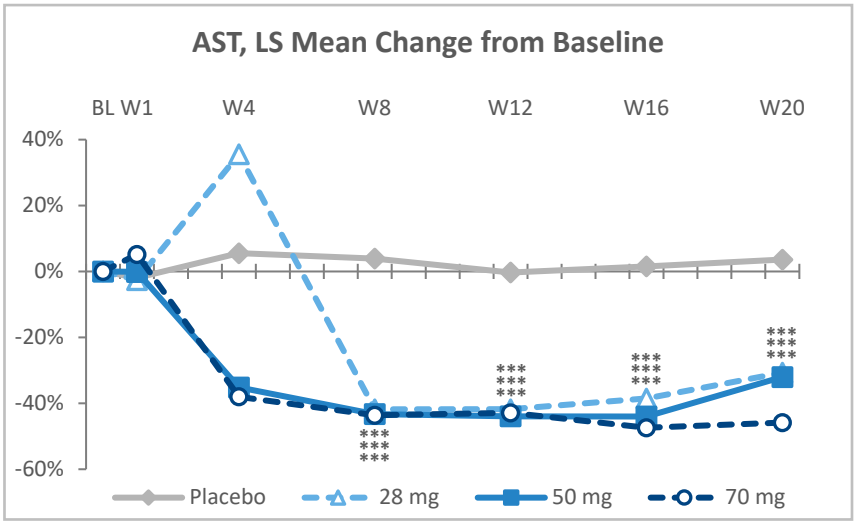
* p<0.05, ** p<0.01, *** p<0.001, versus placebo (ANCOVA)

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)

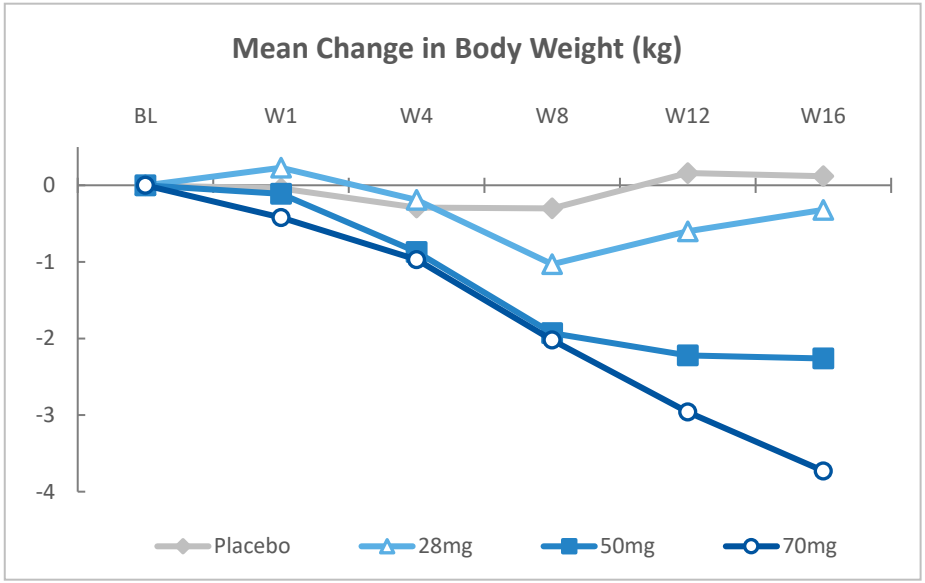


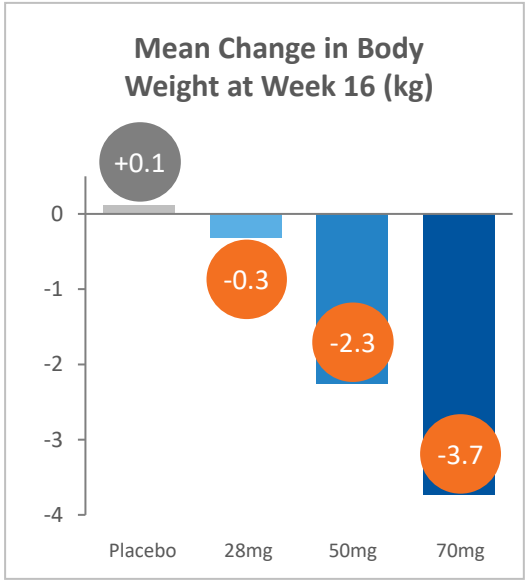
*** p<0.001, versus placebo (MMRM)

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)

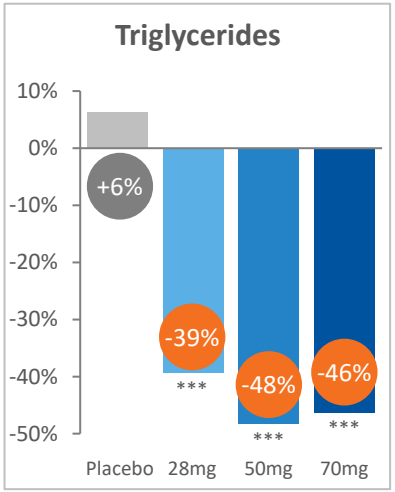




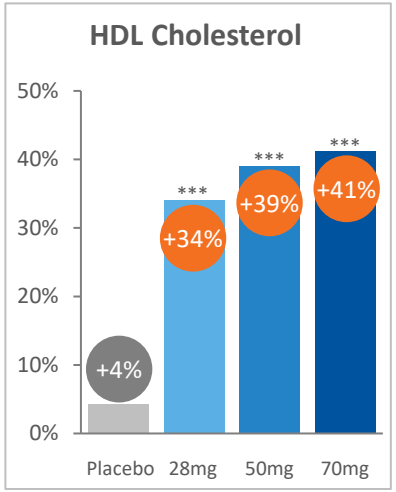
Source Data: Full Analysis Set, F1-F3

IMPROVED LIPOPROTEIN PROFILE (F1-F3 NASH)

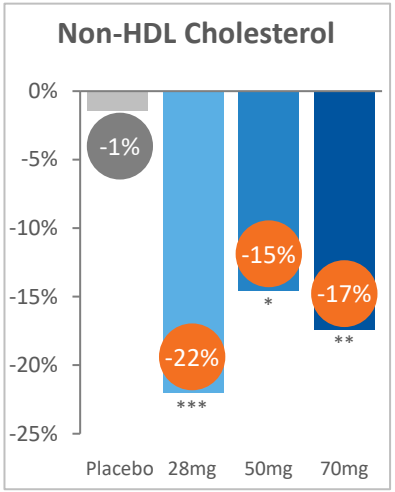
LS Mean Change From Baseline to Week 16 (%)



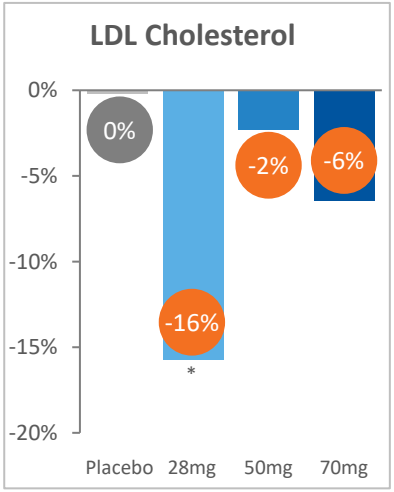
*** p<0.001, versus placebo (ANCOVA)



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



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

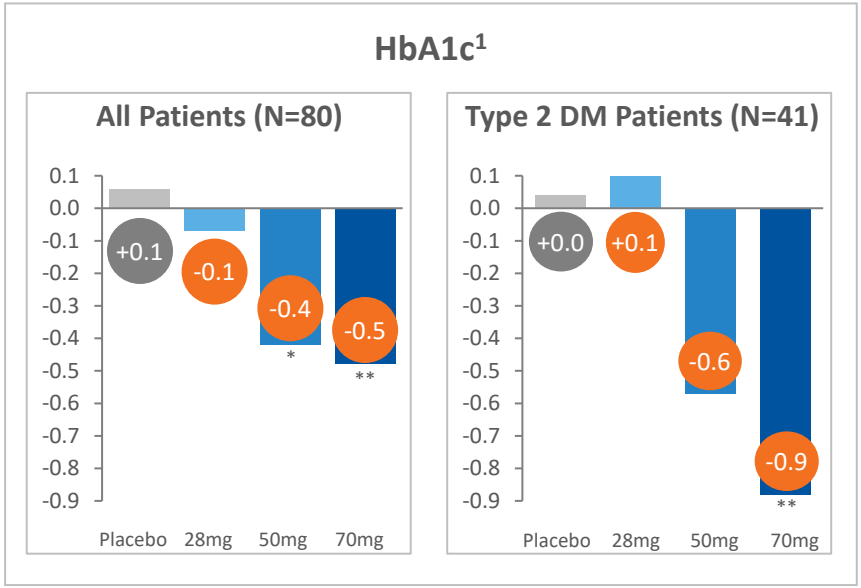


* p<0.05, versus placebo (ANCOVA)

Source Data: Full Analysis Set, F1-F3

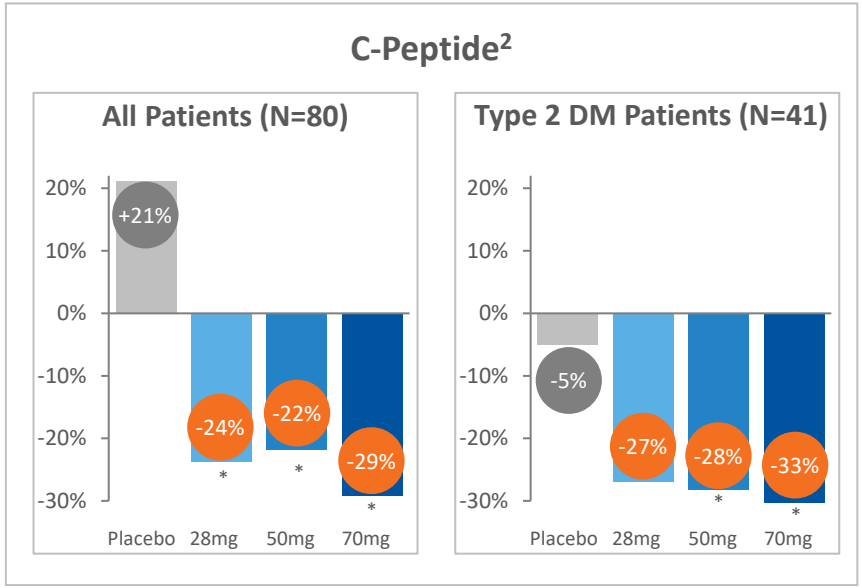
CLINICALLY MEANINGFUL IMPROVEMENTS IN GLYCEMIC CONTROL AFTER 16 WEEKS (F1-F3 NASH)

LS Mean Change From Baseline to Week 16 (%)



[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

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*

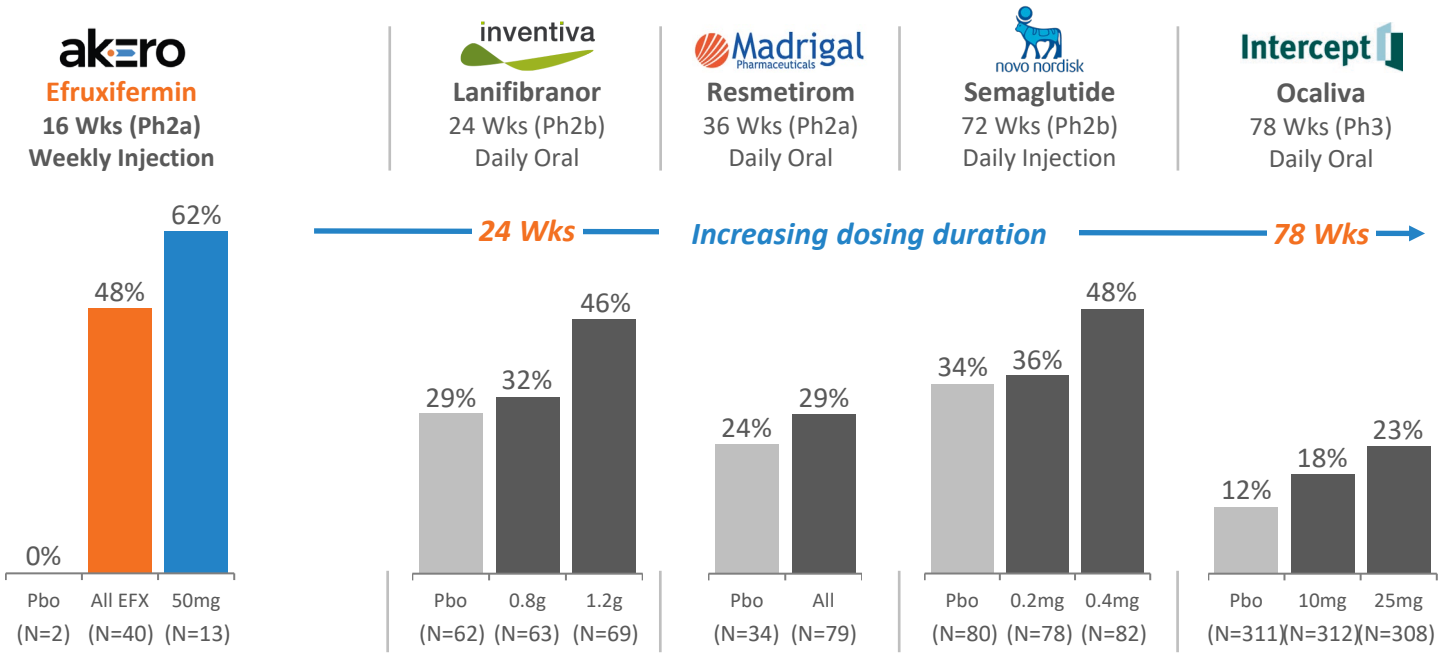
Key Observations

- 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]

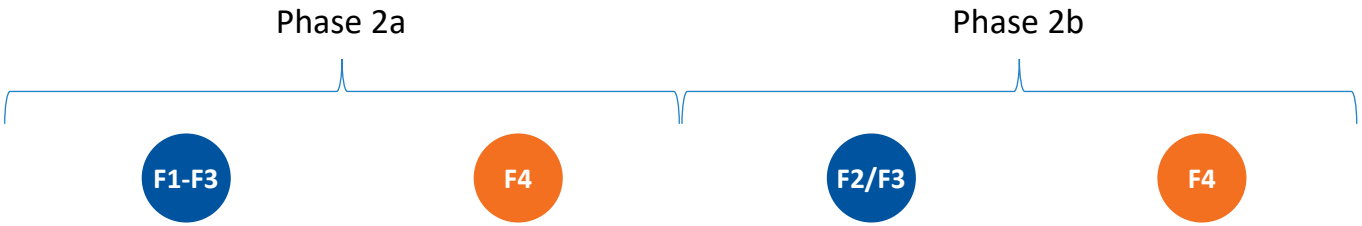


Note: These data are derived from different clinical trials at different points in time, with differences in trial design and patient populations. No head-to-head clinical trials have been conducted.

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



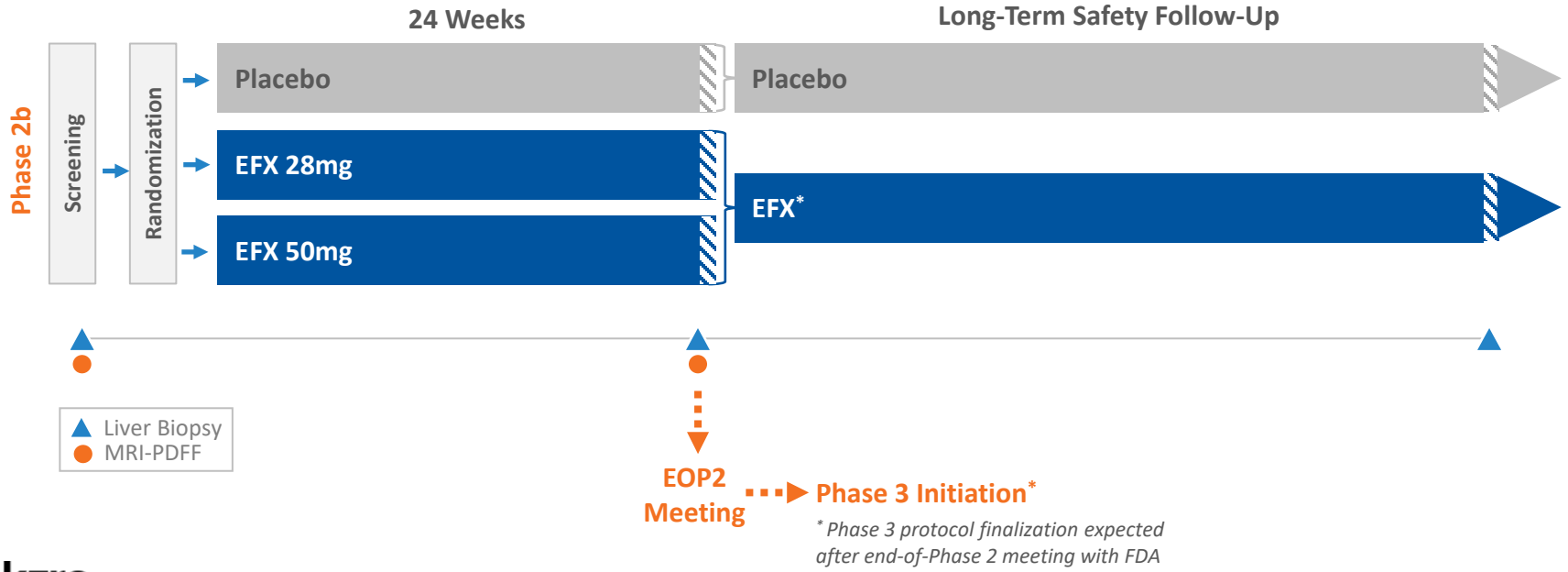
	BALANCED	Cohort C (Expansion of BALANCED)	HARMONY	SYMMETRY
Status	**Completed** (Readout Jun'20)	**Completed** (Readout Mar'21)	**Readout expected in 3Q'22**	**Initiated Jul'21**
Duration	16 Weeks	16 Weeks	24 Weeks	36 Weeks
EFX Arms	28, 50, 70mg	50mg	28, 50mg	28, 50mg
Placebo-Controlled	✓	✓	✓	✓

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

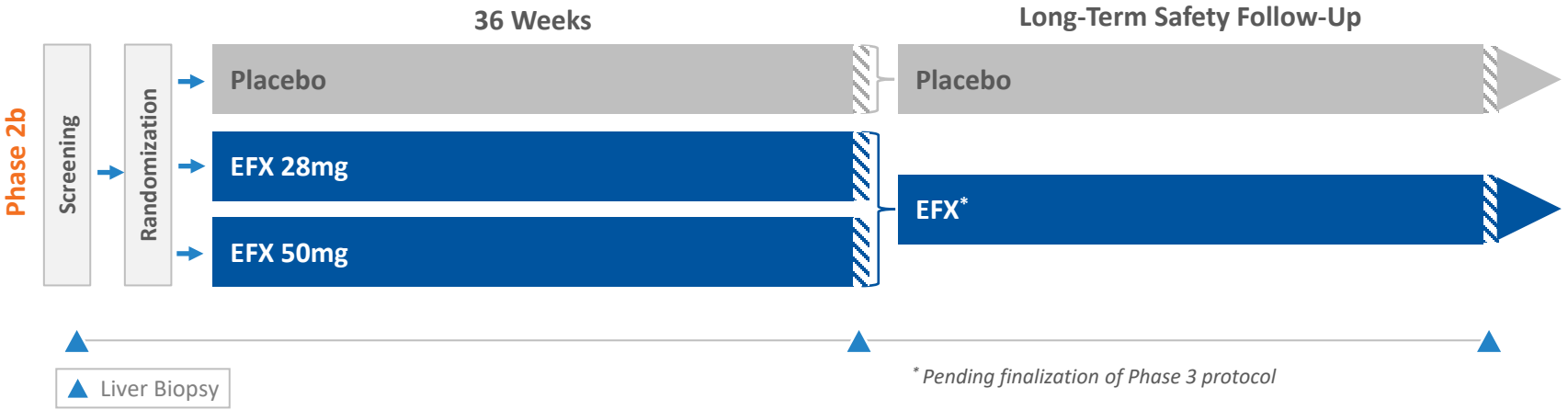


SYMMETRY TRIAL DESIGN: CIRRHOTIC NASH (F4)

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*

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STRONG FINANCIAL POSITION

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

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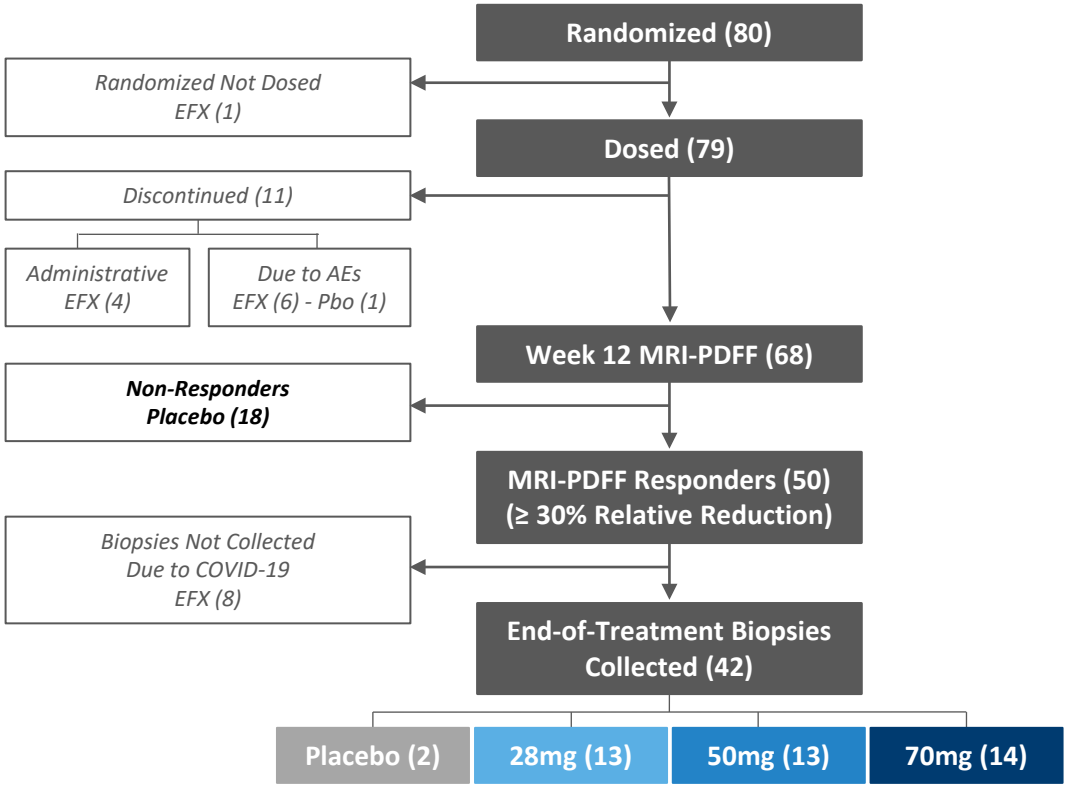
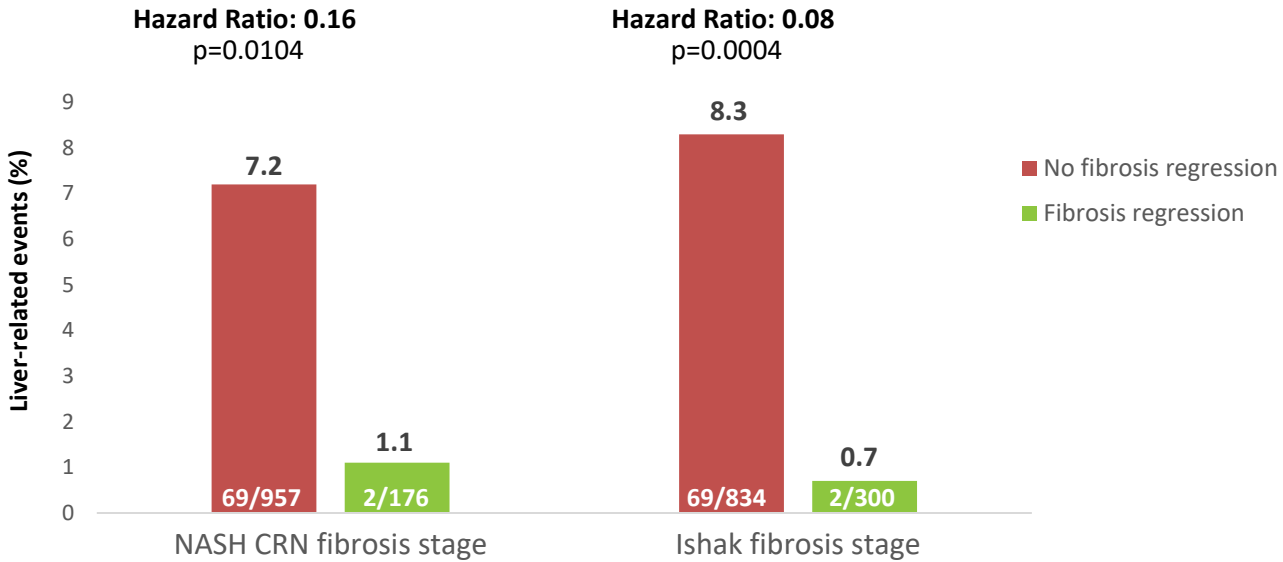
Backup Slides

PATIENT DISPOSITION (BALANCED MAIN STUDY)



CIRRHOSIS REGRESSION IS ASSOCIATED WITH IMPROVED CLINICAL OUTCOMES

Pooled treatment groups from STELLAR 4 and simtuzumab cirrhosis study



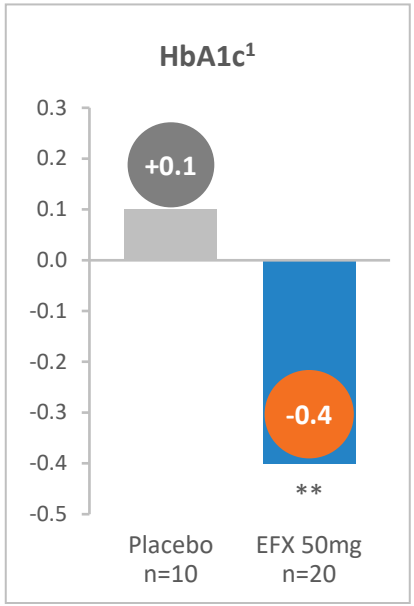
Cirrhosis regression observed in 16% of patients (treatment and placebo groups) over 48 weeks

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.

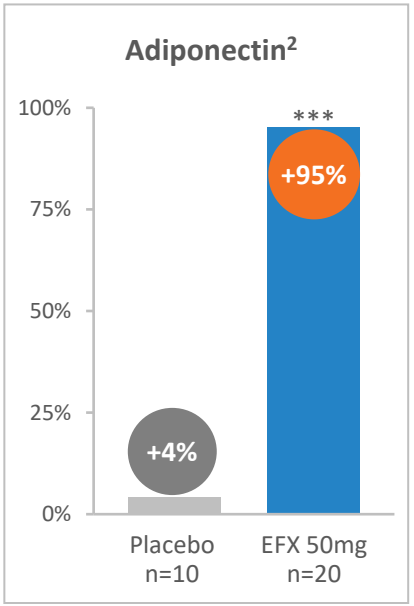
IMPROVED GLYCEMIC CONTROL; TREND TOWARD WEIGHT LOSS (F4 NASH)

LS Mean Change From Baseline to Week 16 (%)



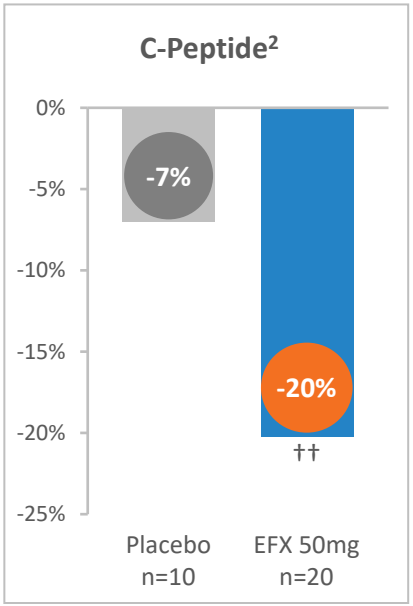
[1] Absolute change from baseline, %

** p<0.01, versus placebo (ANCOVA)



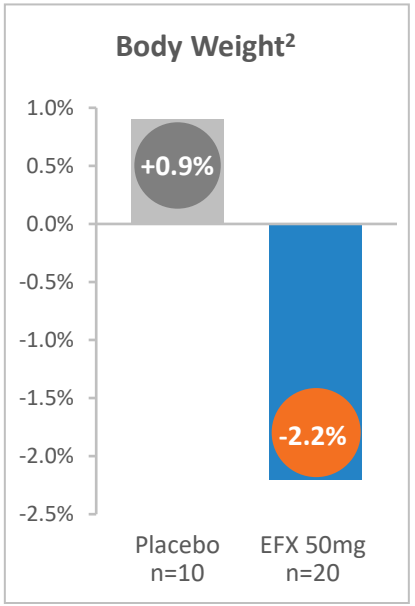
[2] Relative percent change from baseline

*** 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

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

FGF21 DEVELOPMENT LANDSCAPE: SUMMARY

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

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

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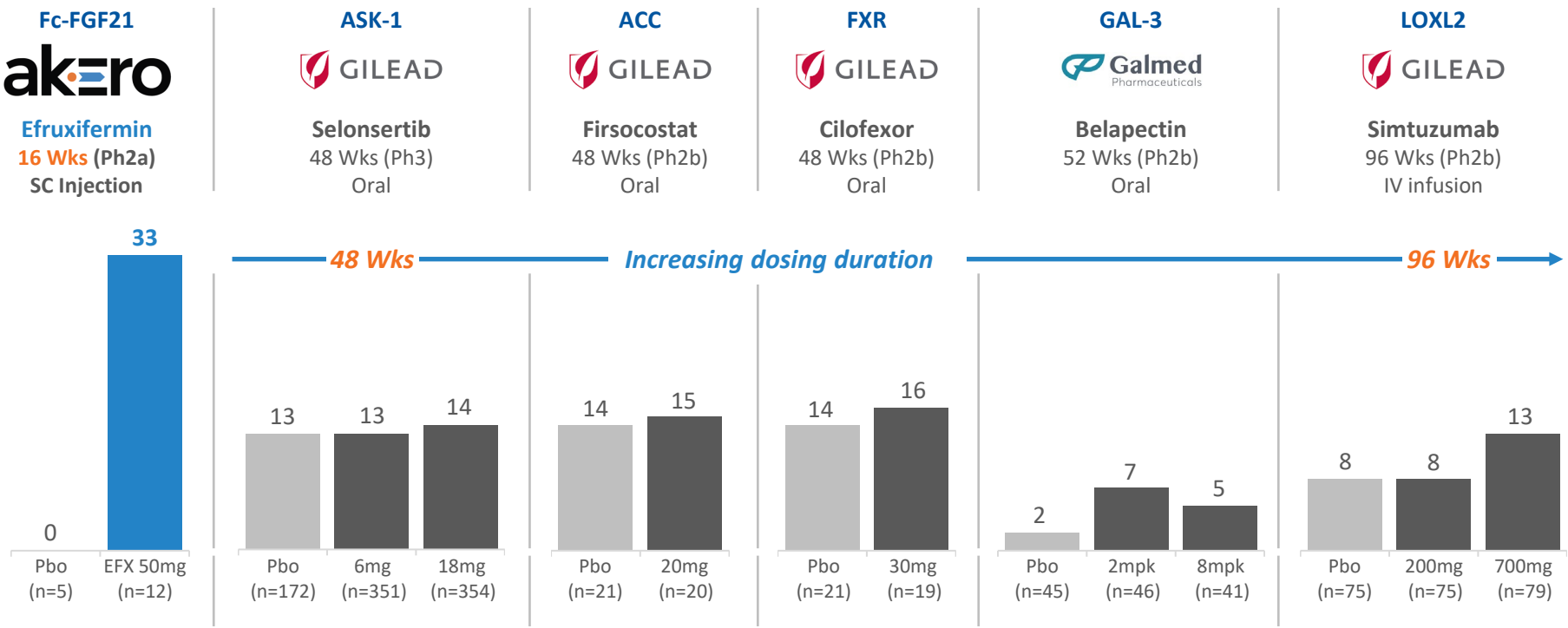
* *Subjects with biopsy-confirmed NASH were present only in the 18mg QW & 18 Q2W cohorts (Loomba et al., 2020 AASLD Poster #LP34)*

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



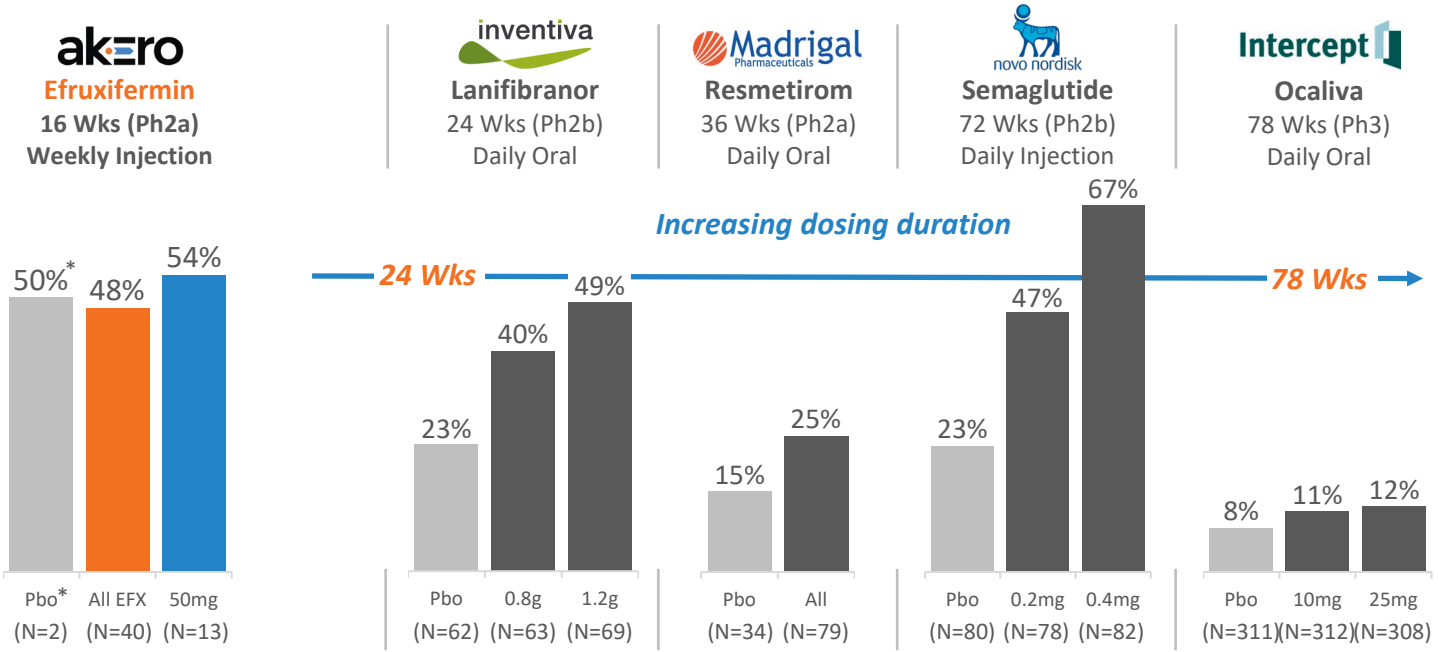
** Results from all publicly reported NASH studies for single agents in F4 patients reporting either ≥ 1-stage fibrosis improvement (belapectin and simtuzumab) or ≥ 1-stage fibrosis improvement and no worsening of NASH (EFX, selonsertib, firsocostat and cilofexor); numerical values represent percent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

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.

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

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A Global Disease, A Pioneering Treatment

NASDAQ: AKRO