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RESTORING METABOLIC BALANCE.
TRANSFORMING LIVES.

Developing medicines to reverse the course
of serious metabolic diseases.

CORPORATE PRESENTATION

SAFE HARBOR

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AKERO
BUILDING VALUE ON A STRONG FOUNDATION

Clinical-Stage FGF Analog

- AKR-001, Fc-FGF21 fusion protein, licensed from **Amgen**
- **3-4 day half-life** for QW dosing
- Robust effects on lipoproteins and markers of insulin sensitivity in 83 T2D patients in two Phase 1 trials
- Well tolerated
- Currently in Phase 2a

Validated Path in NASH

- FGF analogs reduce liver fat; reverse fibrosis in NASH patients
- Phase 1 demonstrated AKR-001's **metabolic effects; anticipated to have anti-fibrotic effects** and target both liver & adipose tissue
- With balanced receptor activity, AKR-001 has potential to be leading FGF analog

Key Upcoming Milestones

1Q20

Ph2a clinical trial primary endpoint (MRI-PDFF)

2Q20

Ph2a clinical trial repeat liver biopsy assessments

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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**

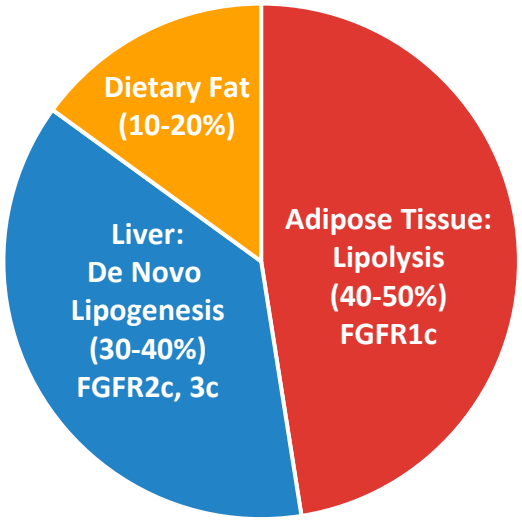
The number of NASH patients with advanced-stage fibrosis and cirrhosis is predicted to **grow to 8 million** in the US by 2030, **an increase of approximately 140% from 2015**

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

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

FGF21 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	✓

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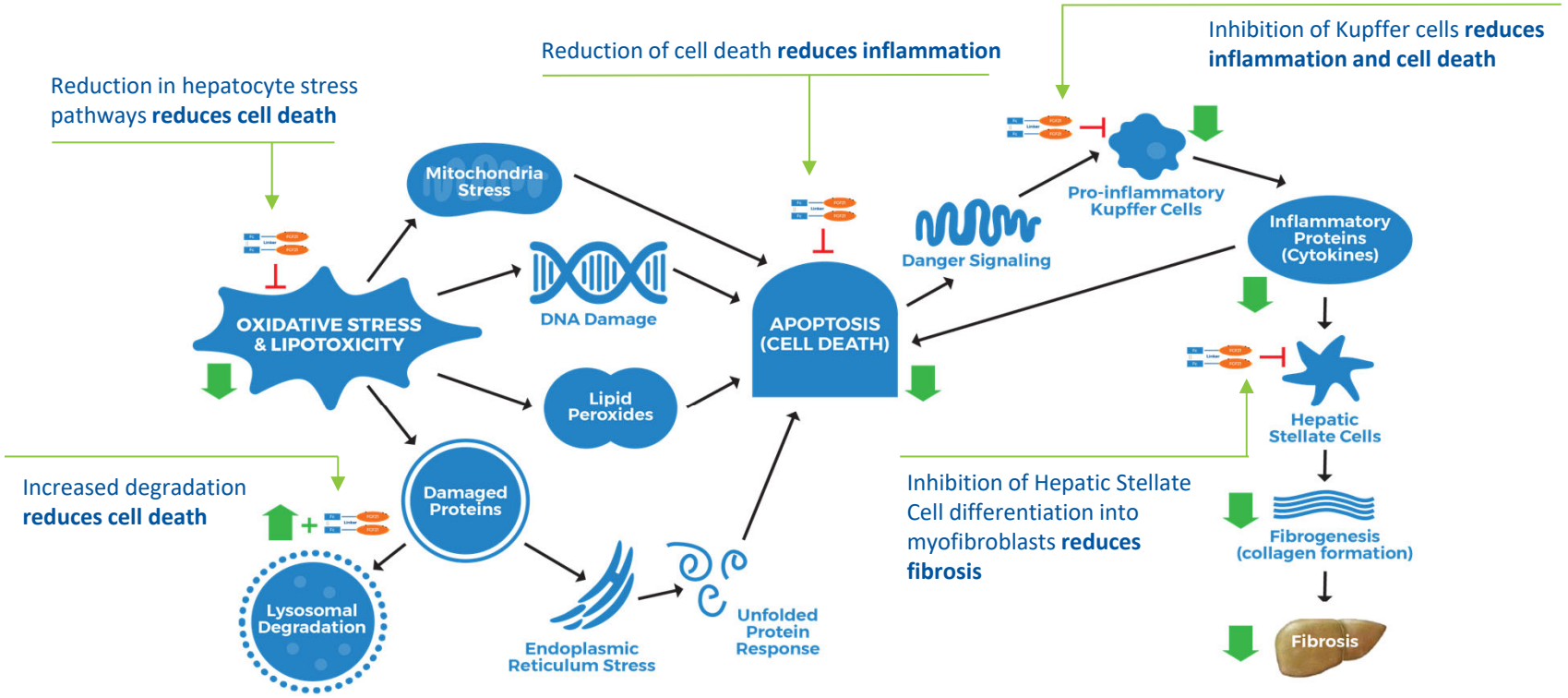
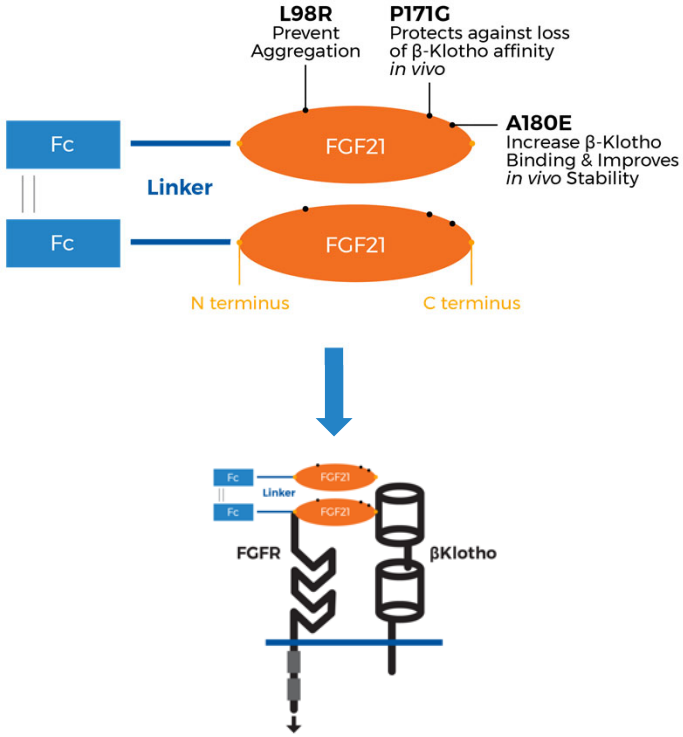
FGF21 PROTECTS HEPATOCYTES AND MITIGATES INFLAMMATION & FIBROSIS



AKR-001 ENGINEERING POTENTIALLY OPTIMAL FOR NASH EFFICACY, WITH CONVENIENT ONCE-WEEKLY DOSING



Key attributes

Amgen-designed 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

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AKR-001 RESTORED METABOLIC BALANCE AND IMPROVED MARKERS OF CARDIOVASCULAR RISK IN PHASE 1

Two Phase 1 clinical trials of AKR-001 studying 83 patients with type 2 diabetes showed

- **Improved Lipoproteins**
- **Improved Insulin Sensitivity**
- **Weight Neutrality**
- **Favorable Tolerability**

And support AKR-001's potential, in NASH patients, to be

METABOLIC
Redirect calories away from the liver
Reduce liver fat
Reduce hepatocellular stress and cell death

ANTI-FIBROTIC
Suppress inflammation
Reverse fibrosis

CARDIOVASCULAR DISEASE IS LEADING CAUSE OF DEATH IN PEOPLE WITH NASH

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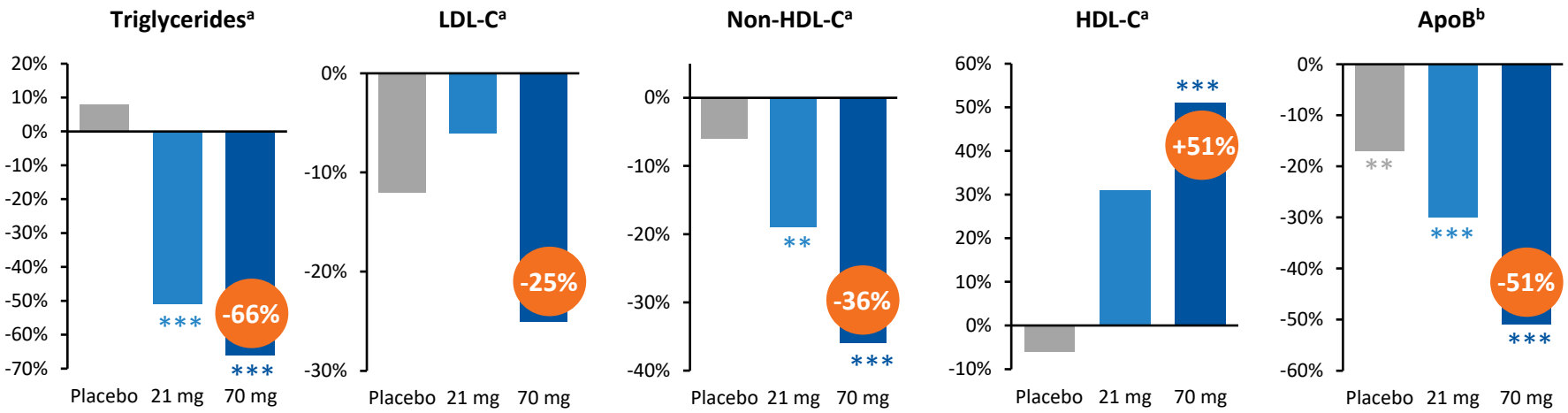
AKR-001 IMPROVED LIPOPROTEIN PROFILE

AKR-001's significant improvements in lipoproteins and reduction in adipose lipolysis are consistent with effective agonism of FGFR2c and 3c in the liver and FGFR1c in adipose tissue



Percent Change From Baseline, Placebo, 21 mg QW and 70 mg QW [a] Day 25; [b] Day 29 ** = $p<0.01$; *** = $p<0.001$

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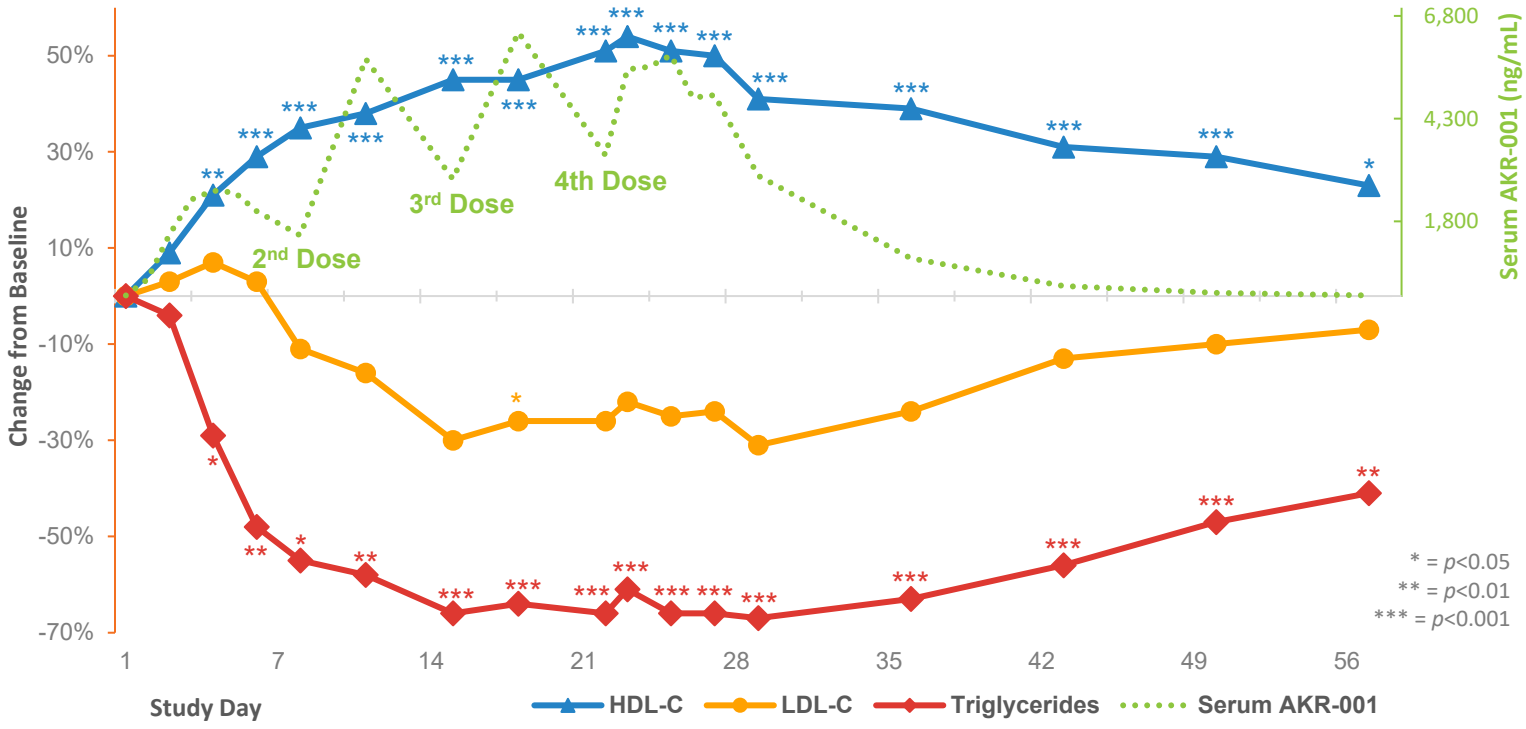
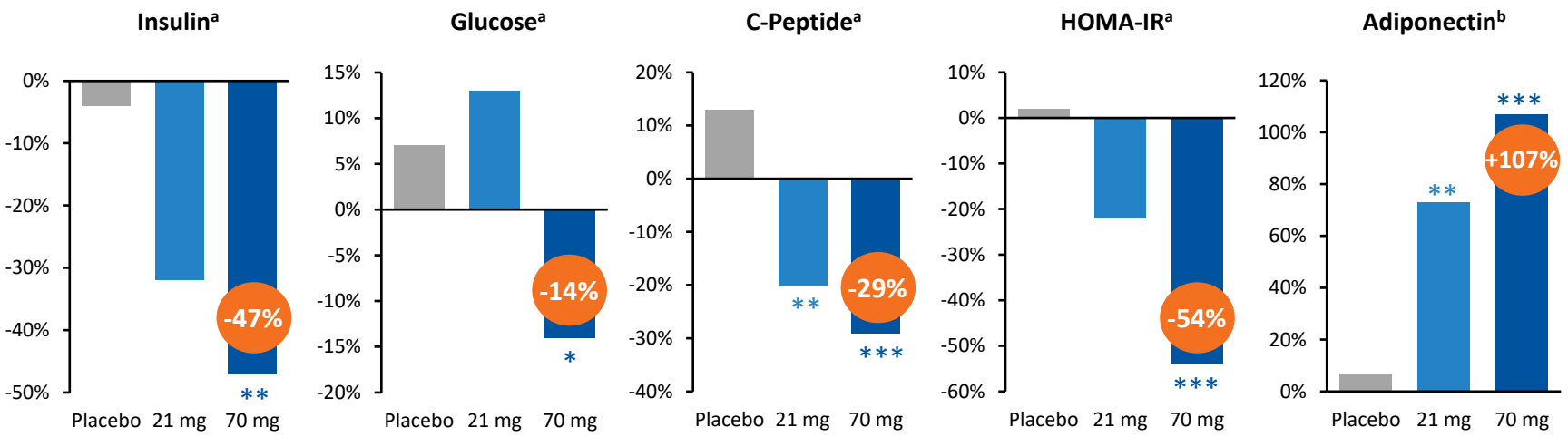
AKR-001 IMPROVED LIPOPROTEINS RAPIDLY AND DURABLY

AKR-001 70 mg QW effects on lipoproteins in Phase 1b clinical trial



AKR-001 IMPROVED INSULIN SENSITIVITY

AKR-001's significant improvement in insulin sensitivity is consistent with effective FGFR1c agonism



Percent Change From Baseline, Placebo, 21 mg QW and 70 mg QW [a] Day 25; [b] Day 29 * = $p<0.05$; ** = $p<0.01$; *** = $p<0.001$

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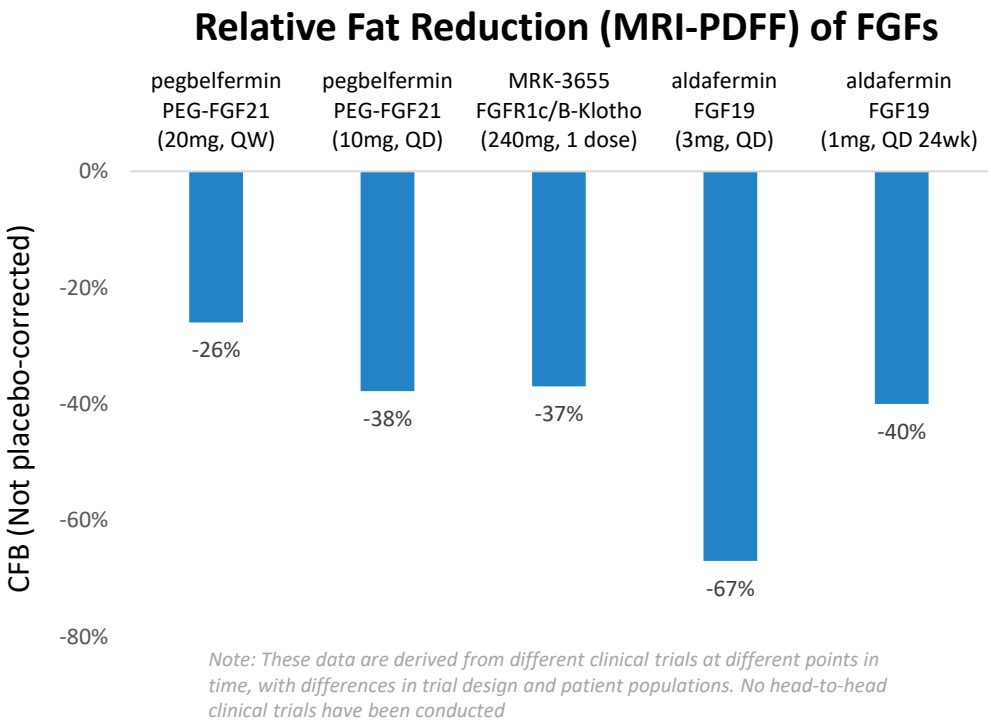
HISTORICAL DATA VALIDATE POTENTIAL FOR BALANCED FGFR AGONISM TO REDUCE LIVER FAT



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

Balanced potency at FGFR1c, 2c, 3c has potential to yield maximum liver fat reduction

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; NGM (2019) October 7 Corporate Presentation.

POSITIVE DATA FOR FGFR AGONIST IN LIVER HISTOLOGY UNDERSCORES POTENTIAL FOR AKR-001

AKR-001 is a balanced agonist of FGFR1c, 2c and 3c, with potential for reduction in hepatocyte stress and apoptosis as well as direct and indirect anti-fibrotic effects

Mean histology change from baseline at W12 (% of patients)	aldafermin 3 mg (n=19)
Fibrosis ≥ 1 stage reduction	**42%**
NAS ≥ 1 point reduction	**84%**

Shankar, S *et al.* (2018) AASLD Poster

Historical improvements in biopsy-based measures in NASH patients after 12 weeks

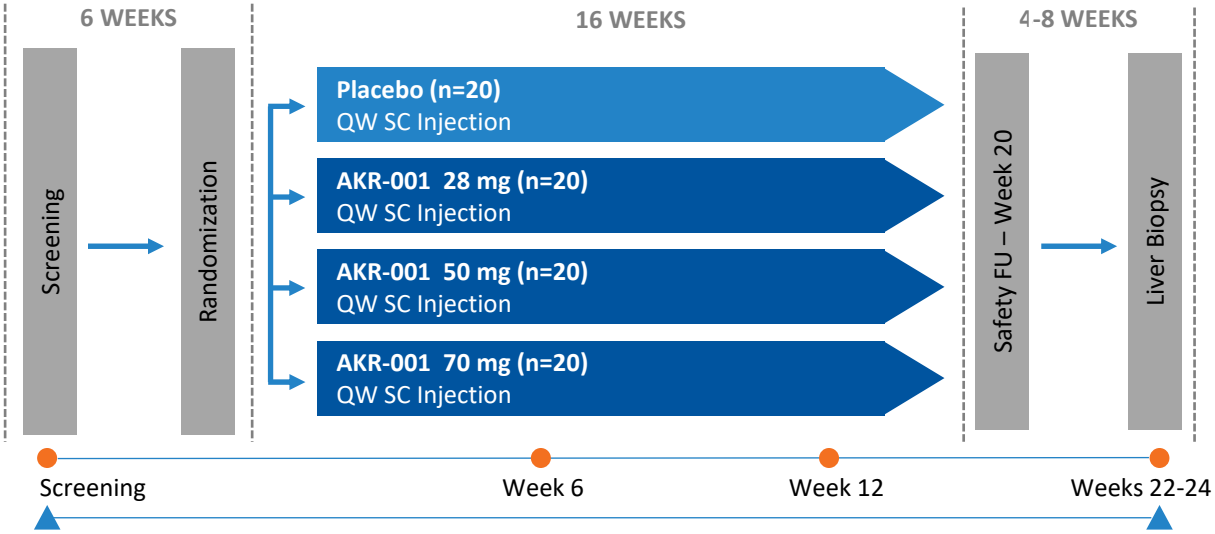
- Underscores the capacity of β-Klotho-dependent FGFR agonism to improve NASH and reverse fibrosis
- May open up opportunity to accelerate path to registrational trials, via early demonstration of histological improvement

PHASE 2A TRIAL: THE BALANCED STUDY (MAIN STUDY)

Multiple endpoints, including liver fat reduction as well as paired biopsies, designed to provide proof of concept that Akero's FGF21 approach combats major hallmarks of NASH

NUMBER OF SUBJECTS
80

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

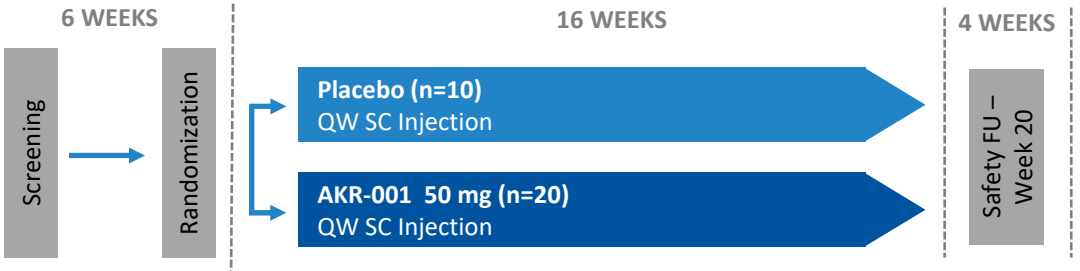


F4 COHORT EXPANSION (COHORT C)

The BALANCED study is being expanded to include a cohort of patients with compensated cirrhosis (F4), Child-Pugh Class A

NUMBER OF SUBJECTS
30

PRIMARY ENDPOINT
Safety and tolerability



- Liver Biopsy
- Fibroscan
- Fibrosis Biomarkers (ELF, Pro-C3)

Selection of 50 mg dose based on PK-PD Modeling of Phase 1b data and availability of drug product

EXTENSIVE DEVELOPMENT, 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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STRONG FINANCIAL POSITION

COMPLETED UPSIZED IPO

June 20, 2019

~$106M
Raised in aggregate gross proceeds

$16
Price upsized IPO at top of marketing range

Q3 EARNINGS UPDATE

September 30, 2019

~$148M
Cash and cash equivalents

~$14M
Q3 operating expenses

SIGNIFICANT NEAR TERM MILESTONES

Phase 2a
Main Study projected completion 1H 2020

Phase 2b
Projected initiation 1H 2021

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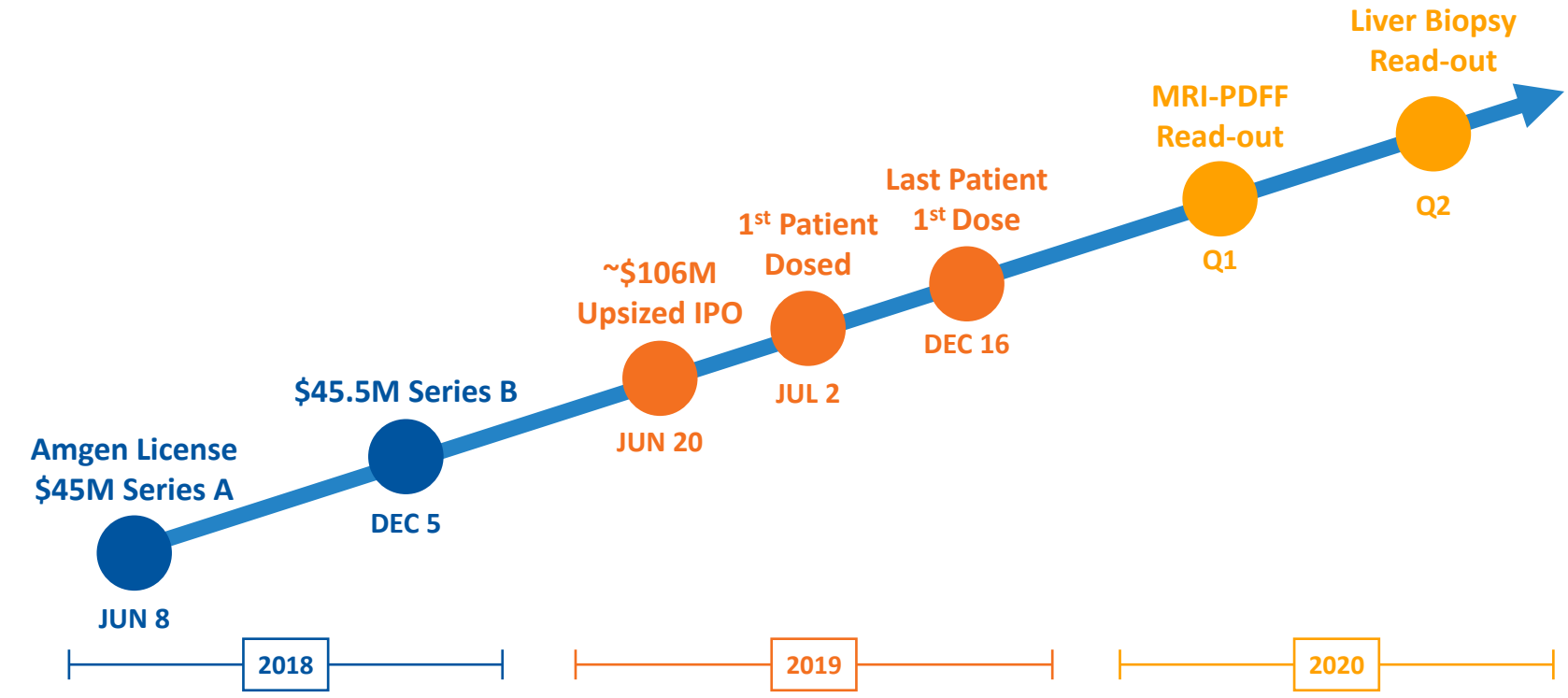
MILESTONES PROJECTED
MILESTONES DELIVERED



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THANK YOU.

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