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): **September 5, 2019**

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

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

170 Harbor Way, 3rd Floor **South San Francisco, CA**	**94080**
(Address of principal executive offices)	(Zip Code)

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

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

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

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

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

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

o 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 x

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. o

Item 5.02. Departure of Directors or Certain Officers; Election of Directors; Appointment of Certain Officers; Compensatory Arrangements of Certain Officers.

To fill an existing vacancy on the Audit Committee of the Board of Directors (the "**Audit Committee**") of Akero Therapeutics, Inc. (the "**Company**"), effective as of September 5, 2019, the Company's Board of Directors appointed a current independent director, Dr. Kevin Bitterman, to serve as a member of the Audit Committee. The Board of Directors has determined that Dr. Bitterman meets the requirements for independence and financial literacy of audit committee members under the applicable listing standards of The Nasdaq Stock Market LLC and the Securities Exchange Act of 1934, as amended.

Item 7.01. Regulation FD Disclosure.

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 attached to this Current Report on Form 8-K as Exhibit 99.1. The Company undertakes no obligation to update, supplement or amend the materials attached hereto as Exhibit 99.1.

The information contained in Item 7.01 in this Current Report on Form 8-K (including Exhibit 99.1) shall not be deemed "filed" for purposes of Section 18 of the Securities Exchange Act of 1934 (the "**Exchange Act**") or otherwise subject to the liabilities of that section, nor shall it be deemed incorporated by reference in any filing under the Securities Act of 1933 or the Exchange Act, except as expressly set forth by specific reference in such a filing.

Item 9.01. Financial Statements and Exhibits.

(d) Exhibits

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

SIGNATURES

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

Date: September 6, 2019

AKERO THERAPEUTICS, INC.

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

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RESTORING METABOLIC BALANCE. TRANSFORMING LIVES.

Developing medicines to reverse the course of serious metabolic diseases.

CORPORATE PRESENTATION

Safe Harbor

This presentation has been prepared by Akero Therapeutics, Inc. ("we," "us," "our," "Akero" or the "Company") and is made for informational purposes only and does not constitute an offer to sell or a solicitation of an offer to buy securities, nor shall there be any sale of any securities in any state or jurisdiction in which such offer, solicitation or sale would be unlawful prior to registration or qualification under the securities laws of any such state or jurisdiction. The information set forth herein does not purport to be complete or to contain all of the information you may desire. Statements contained herein are made as of the date of this presentation unless stated otherwise, and neither this presentation, nor any sale of securities, shall under any circumstances create an implication that the information contained herein is correct as of any time after such date or that information will be updated or revised to reflect information that subsequently becomes available or changes occurring after the date hereof.

This presentation may contain "forward-looking statements" 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, our development plans, our preclinical and clinical results and other future conditions. 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 applicable law, we do not plan to publicly update or revise any forward-looking statements contained herein, whether as a result of any new information, future events, changed circumstances or otherwise. Although we believe the expectations reflected in such forward-looking statements are reasonable, we can give no assurance that such expectations will prove to be correct. Accordingly, readers are cautioned not to place undue reliance on these forward-looking statements. No representations or warranties (expressed or implied) are made about the accuracy of any such forward-looking statements.

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.

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

Precedented Path in NASH

- FGF analogs observed to reduce liver fat and reverse fibrosis in NASH patients
- AKR-001 expected to (a) have **metabolic & anti-fibrotic** effects and (b) 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 assessment (MRI-PDFF)

2Q20

Ph2a clinical trial repeat liver biopsy assessments

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LEADERSHIP

Andrew Cheng, MD, PhD
President & CEO
- 19 years at Gilead
- Chief Medical Officer & HIV Division Head
- Major role in 11 NDA/MAA approvals

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

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

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)

Kitty Yale
EVP & Chief Development Officer
- Over 25 years Gilead, Roche, Pfizer
- VP Gilead Worldwide Clinical Operations
- Major role in 8 global approvals NDA, MAA , JNDA and CFDA

EXTENSIVE EXPERIENCE IN
- Drug discovery
- Development
- Commercialization

INVOLVED IN 20+ MEDICINE APPROVALS

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STRONG FINANCIAL POSITION

COMPLETED UPSIZED IPO *June 20, 2019*	Q2 EARNINGS UPDATE *June 30, 2019*	FUNDED THROUGH 2022
~$106M Raised in aggregate gross proceeds	~$163M Cash and cash equivalents	Phase 2b Projected completion by EOY 2022
$16 Price upsized IPO at top of marketing range	$7.6M Q2 operating expenses	Phase 2a To be completed in 1H 2020

NASH
A SERIOUS AND DEBILITATING CONDITION

NASH is fueled by rise in obesity and diabetes

A GROWING HEALTH EPIDEMIC

An estimated **17 million Americans** have NASH

The US NASH patient population is expected to **grow over 50% to 27 million 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

FGF21 TARGETS
ALL CORE STAGES OF NASH PATHOGENESIS

NASH PATHOGENESIS	Excessive Caloric Burdening of Liver	Excessive Liver Fat, Fat Oxidation	Hepatocyte cell stress, injury, death	Inflammation	Fibrosis
FGF21 INTERVENTION	Improve insulin sensitivity Redirect calories away from liver without weight gain	Reduce all three sources of liver fat Reduce de novo lipogenesis Reduce fat oxidation Improve lipoproteins	Reduce oxidative stress & lipotoxicity Reduce ER stress and cumulative mitochondrial damage Suppress cell death	Suppress activation of pro-inflammatory Kupffer cells	Suppress activation of collagen-producing hepatic stellate cells

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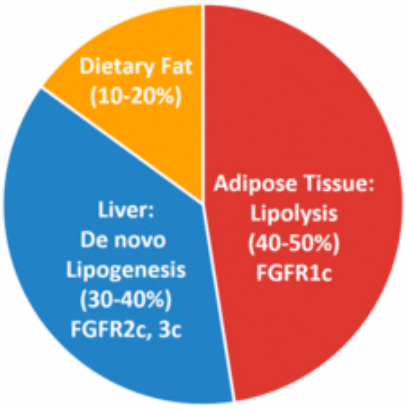
UNDERSTANDING THE SOURCES OF LIVER FAT
AND RELEVANT FGF RECEPTORS

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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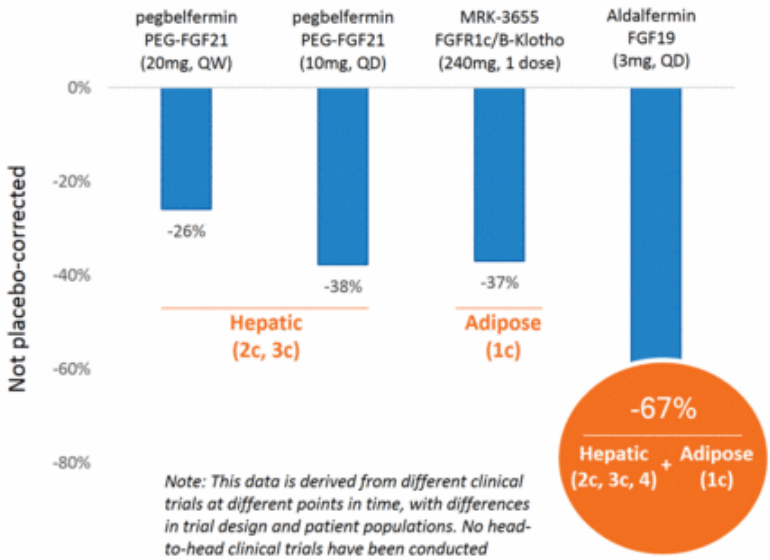
IMPACT OF BALANCED FGFR AGONISM ON FAT REDUCTION

Relative Fat Reduction (MRI-PDFF)



Note: This data is 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

Liver fat reduction of 67% (MRI-PDFF)
largest reported among all candidates that we know to be targeting FGFRs

Balanced potency at FGFR1c, 2c, 3c
appears to drive liver fat reduction

FGFR4 appears unnecessary
for glucose sensitivity & 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

POTENTIAL FOR IMPROVED LIVER HISTOLOGY
WITHIN 12 WEEKS

Mean histology change from baseline at W12 (% of patients)	aldafermin 3 mg (n=19)
Fibrosis ≥ 1 stage reduction	42%
NAS ≥ 1 point reduction	84%
Steatosis ≥ 1 point improvement	74%
Lobular inflammation ≥ 1 point improvement	42%
Hepatocellular Ballooning ≥ 1 point improvement	53%

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

aldafermin (NGM282) appeared to improve 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

Underscores potential for AKR-001

- Balanced agonism of FGR1c, 2c and 3c
- Reduction in hepatocyte stress and apoptosis
- Direct and indirect anti-fibrotic effects

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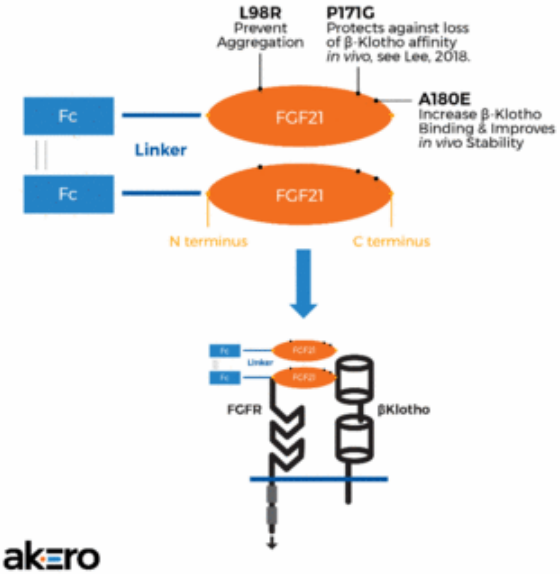
AKR-001 ENGINEERING
POTENTIALLY OPTIMAL FOR NASH EFFICACY



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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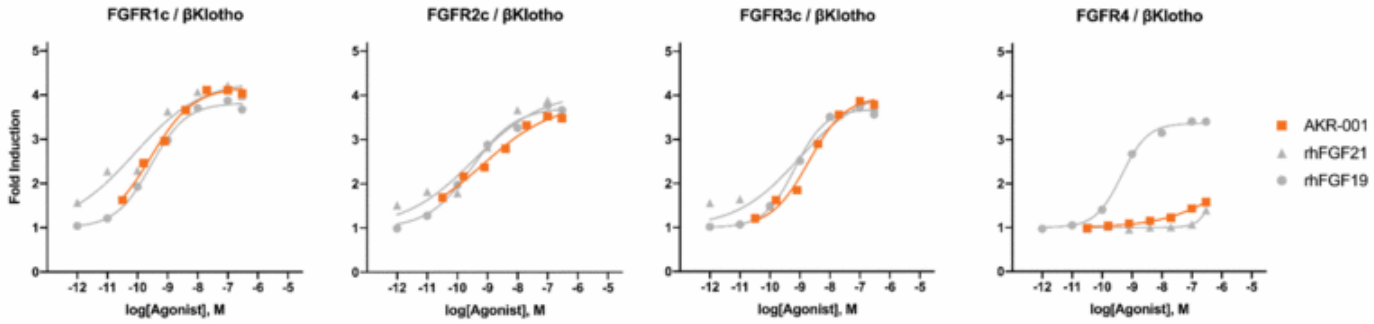
AKR-001 REPRODUCED BALANCED *IN VITRO* POTENCY

AKR-001, rhFGF21, and rhFGF19 Activation of FGFR-Dependent Intracellular Signaling in L6 Cells



Pharmacology of other FGF21 analogs has not been disclosed for individual receptors

Stanislaus, S *et al.* (2017) *Endocrinology* 158(5):1314-1327

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PROMISING DATA
FROM PHASE 1 CLINICAL TRIALS

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

- **Improved Lipoproteins**
- **Improved Insulin Sensitivity**
- **Weight Neutral**

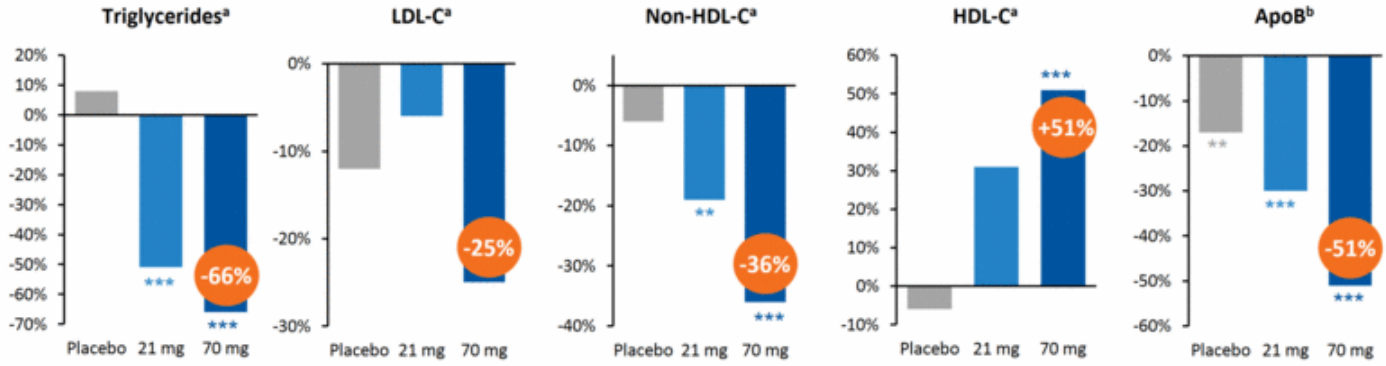
And support AKR-001's potential, in NASH patients, to be

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

ANTI-FIBROTIC
Suppress inflammation
Reverse fibrosis

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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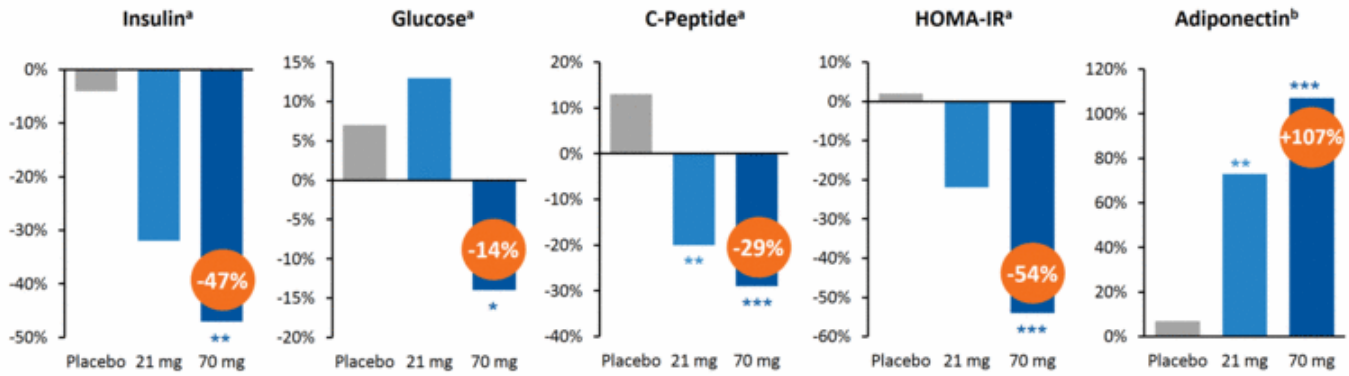
AKR-001 **IMPROVED INSULIN SENSITIVITY**

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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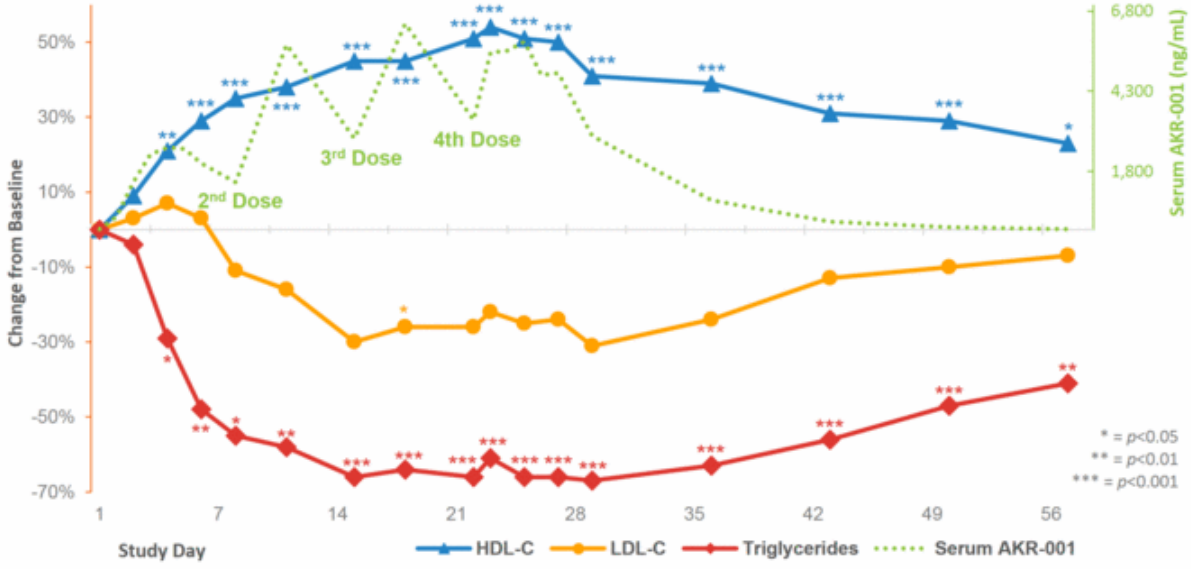
AKR-001 IMPROVED LIPOPROTEINS **RAPIDLY AND DURABLY**

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

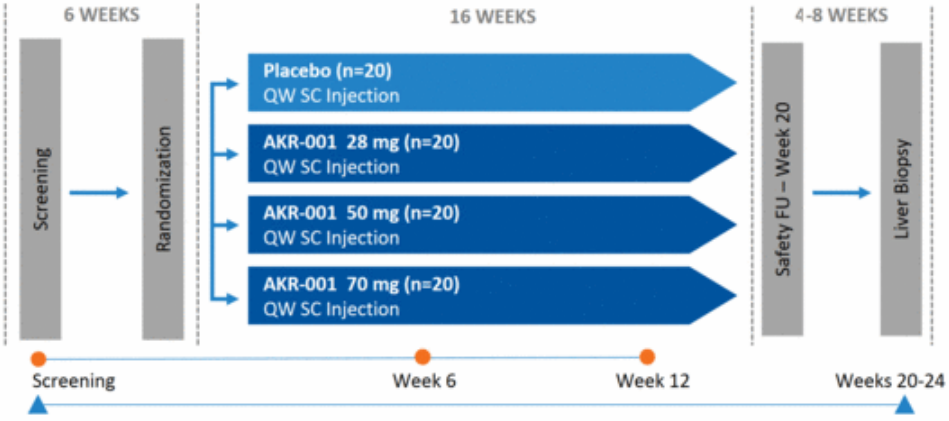


PHASE 2A TRIAL: THE BALANCED STUDY

A Phase 2a multicenter, randomized, double-blind, placebo-controlled, dose-ranging trial to evaluate the safety, tolerability and efficacy of AKR-001 in biopsy-confirmed patients with NASH

NUMBER OF SUBJECTS
About 80

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



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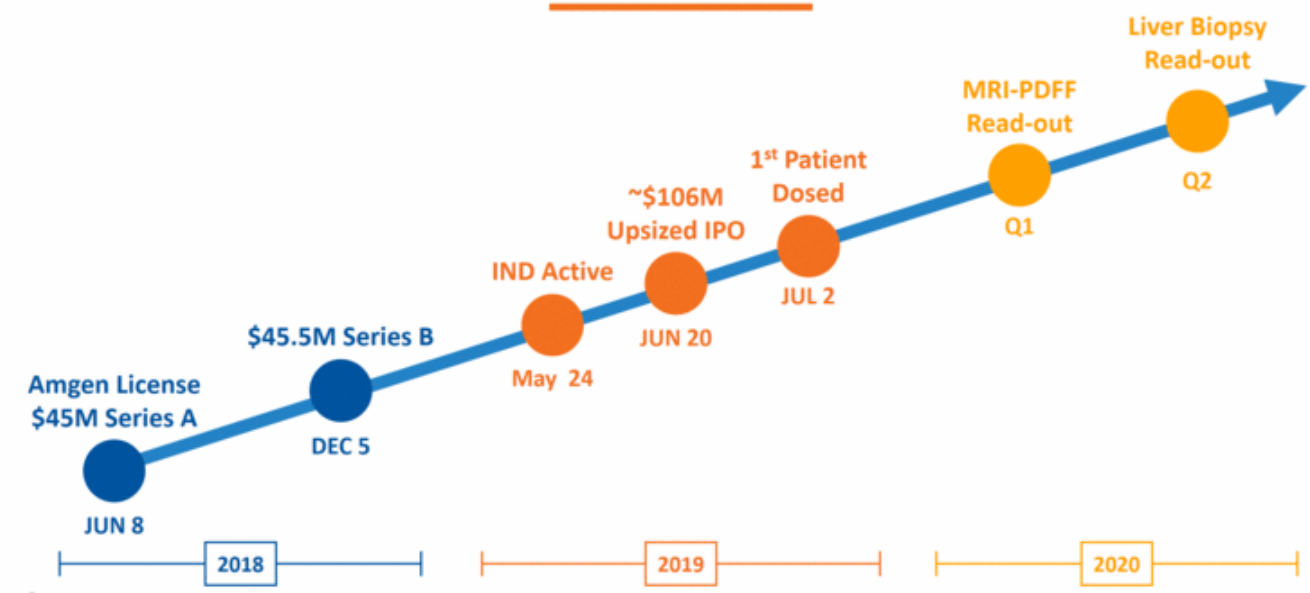
MILESTONES DELIVERED
MILESTONES PROJECTED



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THANK YOU.

CORPORATE PRESENTATION