VIKING
THERAPEUTICS

Corporate Presentation

June 2020

Forward-Looking Statements

This presentation contains statements about our future expectations, plans and prospects that constitute forward-looking statements for purposes of the safe harbor provisions of the Private Securities Litigation Reform Act of 1995. Actual results may differ materially from those indicated by these forward-looking statements as a result of various important factors, including risks relating to: both our and our collaborators' ability to successfully research, obtain regulatory approvals for, develop and commercialize products based upon our technologies; our ability to obtain and maintain proprietary protection for our technologies and product candidates; our reliance on third parties to manufacture our preclinical and clinical drug supplies; competitive pressures; our ability to obtain and maintain strategic collaborations; compliance with our in-license agreements; our ability to successfully execute on, and receive favorable results from, our proprietary drug development efforts; market acceptance of our drug candidates; retaining members of our senior management; and our ability to raise additional funds to finance our operations.

The forward-looking statements included in this presentation represent our views as of the date of this presentation. We anticipate that subsequent events and developments will cause our views to change. While we may elect to update these forward-looking statements in the future, we specifically disclaim any obligation to do so. These forward-looking statements should not be relied upon as representing our views as of any date subsequent to the date of this presentation.

For more information regarding risks and uncertainties that could affect the results of our operations or financial condition review our filings with the Securities and Exchange Commission (in particular, our most recent Annual Report on Form 10-K and any subsequently filed Quarterly Reports on Form 10-Q).

Investment Highlights

- **Focused on novel therapeutics for metabolic and endocrine diseases**
  - Clinical programs demonstrate best-in-class efficacy data
- **Metabolic Disease Program: VK2809 for NASH**
  - Novel, selective thyroid receptor-β (TRβ) agonist
  - Phase 2a results demonstrate significant reduction in liver fat content, lipids
  - Phase 2b VOYAGE trial ongoing
- **Rare Disease Program: VK0214 for X-ALD**
  - Novel, selective thyroid receptor-β agonist
  - In vivo data show improvement in key biomarkers
  - Clinical development to commence 2020
- **Other Pipeline Programs: Musculoskeletal and metabolic disorders**

Pipeline Overview

Product Candidates	Indication	Development Stage				Status
Development Programs		Preclin	Phase 1	Phase 2	Phase 3	
VK2809 (TRβ agonist)	*NASH*					Phase 2b VOYAGE trial ongoing
VK0214 (TRβ agonist)	*X-ALD*					IND planned, 2020
Other Programs		Preclin	Phase 1	Phase 2	Phase 3	
VK5211 (SARM)	*Hip fracture, muscle wasting*					Phase 2 completed
VK0612 (FBPase inhibitor)	*Type 2 Diabetes*					Phase 2a completed
VK1430 (DGAT-1 inhibitor)	*Hypertriglyceridemia, NASH*					Preclinical

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Metabolic Disease Program

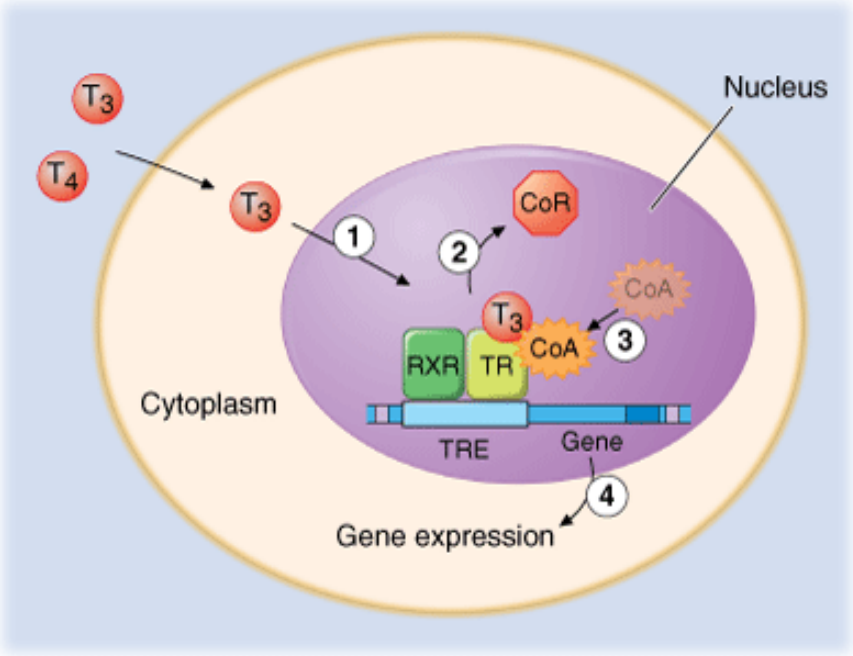
VK2809: Selective Thyroid Receptor-β Agonist

Liver Disorders

Metabolic Disease Program: Selective Thyroid-β Agonists

- **Proprietary platform for small molecule thyroid hormone mimetics**
  - Highly tissue and receptor selective
  - Produce potent lipid reductions in animals and humans
  - Unique chemical scaffolds, expected wider safety window vs. other approaches
- **Biological profiles suggest potential benefit in multiple indications**
  - Broad: NASH, hypercholesterolemia, dyslipidemia
  - Rare: X-linked adrenoleukodystrophy (X-ALD), other
- **Lead molecules VK2809, VK0214**
  - Oral, once-daily formulations
  - VK2809: Phase 2b ongoing, biopsy-confirmed NASH
  - VK0214: IND-enabling studies in progress, X-ALD

Thyroid Receptor Overview



Key steps in receptor activation: Endogenous thyroid hormone T3 crosses mitochondrial membrane (1) binding thyroid receptor TR, dissociating co-repressor CoR (2). Subsequent binding of co-activator CoA (3) results in altered gene expression (4). RXR: retinoid X receptor; TRE: thyroid response element.

- **Nuclear hormone receptors**
- **Two major subtypes**
  - **Thyroid beta receptor:** Liver, brain; modulates cholesterol, triglyceride levels
  - **Thyroid alpha receptor:** Cardiac tissue, modulates heart rate, contraction

Therapeutic goal, lipid setting: β-receptor selectivity; minimize alpha-effects

Graphic: *Harrison's Principles of Internal Medicine,* 17th Edition, Chapter 335, Fig 335-4, copyright McGraw-Hill, 2008.

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Thyroid Receptor β Agonists for NAFLD and NASH

Development of NASH:

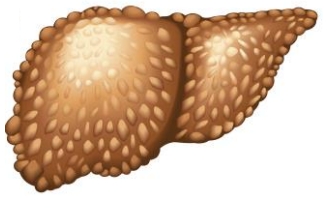
Healthy liver

Accumulation of fatty acids, triglycerides

Oxidative stress, inflammatory response

NASH: Steatosis, ballooning, hepatocyte damage

NAFLD Progression

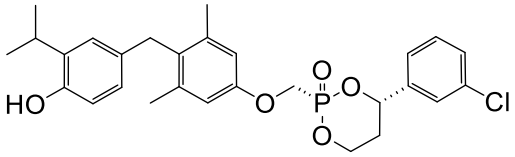


- Cirrhosis
- HCC
- Liver failure

- β-Receptor: Key role in lipid metabolism; systemic and liver-specific effects
- Receptor localized to liver, limited ex-hepatic expression
- In vivo evidence suggests β-activation provides anti-fibrotic benefits
- Clinical data indicate correlation between reduced liver fat, improvement in NAS

An agent that reduces liver fat, improves systemic lipids, and antagonizes fibrotic signaling could provide multi-pronged benefits in NASH

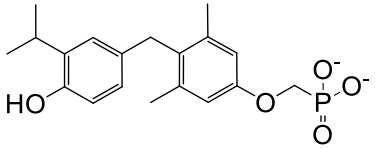
VK2809: Selective Liver-Targeted TRβ Agonist



VK2809, Novel Prodrug

Following oral dosing:

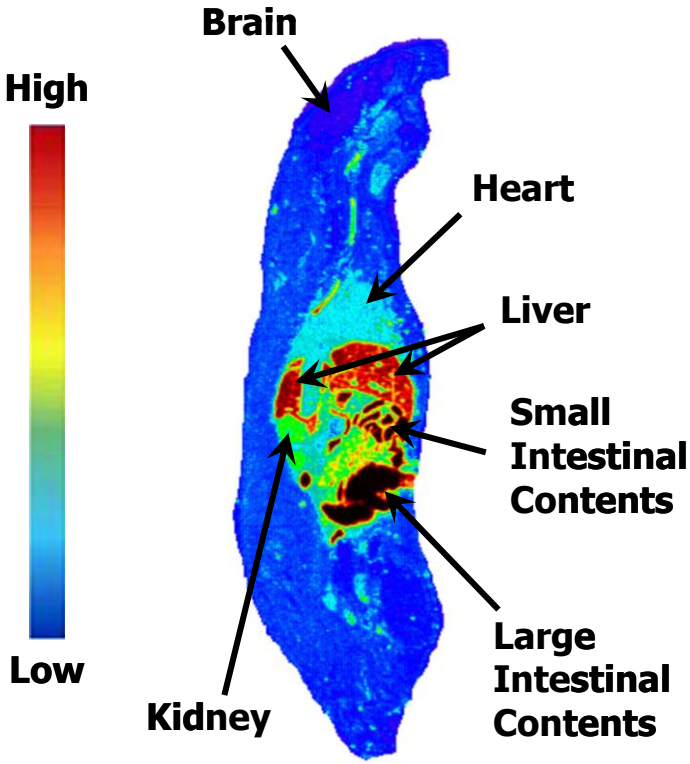
- Cyp3A4-mediated cleavage of prodrug
- 3A4 primarily expressed in liver
- Results in targeted delivery of drug to liver



VK2809A, Potent TRβ Agonist

- 2 nM Ki at TRβ
- 17:1 Selective for β:α

Drug Accumulation by ^{14}C QWBA (4 h)



SD rat, 5mg/kg dose; >30x anticipated human doses

Selective activation, differentiated chemistry lends VK2809 liver selectivity; reduces risk of systemic effects

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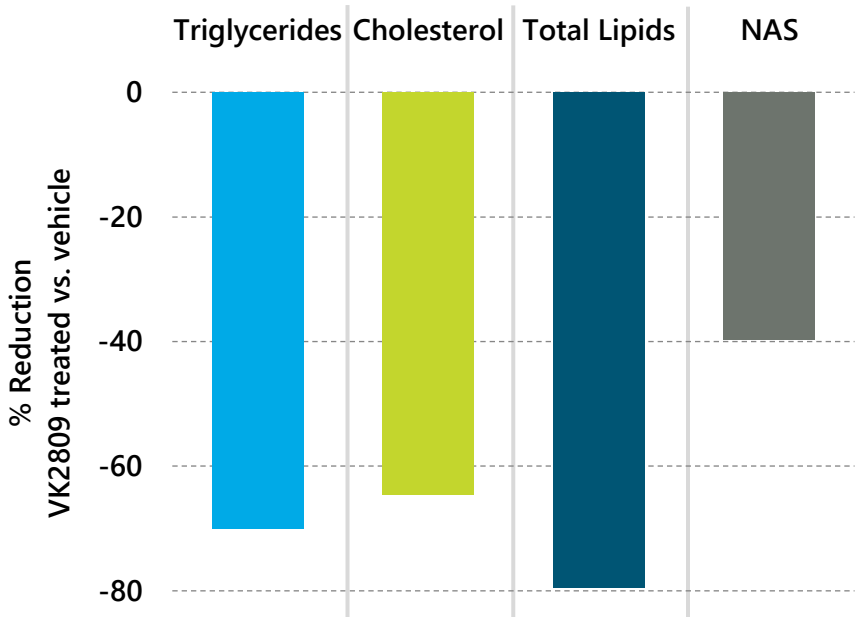
VK2809 Significantly Reduces Steatosis in Diet-Induced NASH

- Evaluation in biopsy-confirmed diet-induced NASH model
  - Rodent model designed to reflect progression of disease in humans
  - Animals biopsied pre-study; only those with NASH *and* fibrosis selected
  - VK2809 dosed once-daily for 8 weeks

Change in Liver Lipids Following 8 Weeks Dosing With VK2809



	Triglycerides	Cholesterol	Total Lipids	NAS
% Difference:	-70.0%	-64.6%	-79.5%	-39.7%
p-value:	<0.0001	<0.0001	<0.0001	<0.0001

Treatment with VK2809 significantly improves lipids, steatosis, NAS at 8 weeks; well-tolerated with no evidence of toxicity

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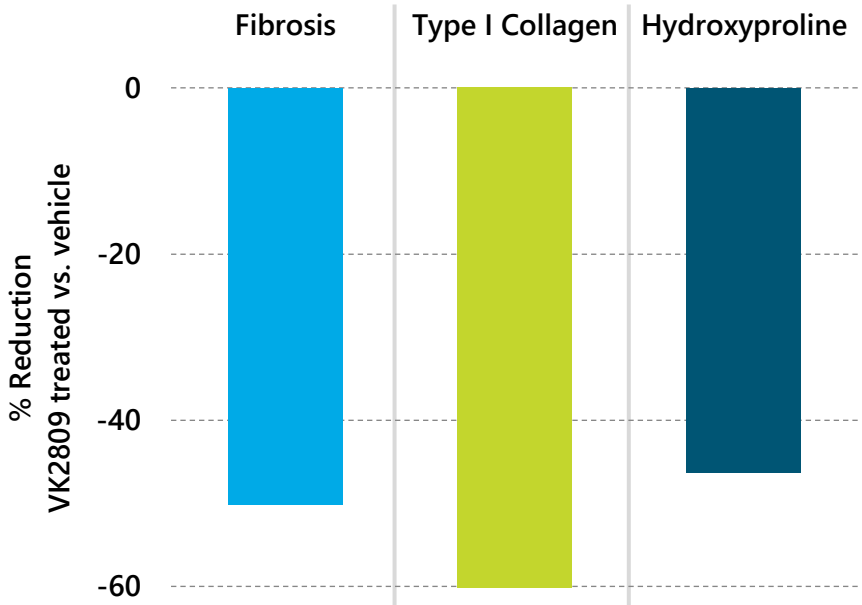
VK2809 Improves Fibrosis in Diet-Induced NASH Model

- Significant reductions in fibrosis, collagen, hydroxyproline after 8 weeks
- Supports thesis that selective TRβ activation produces broad metabolic benefits

Change in Liver Fibrosis Following 8 Weeks Dosing With VK2809



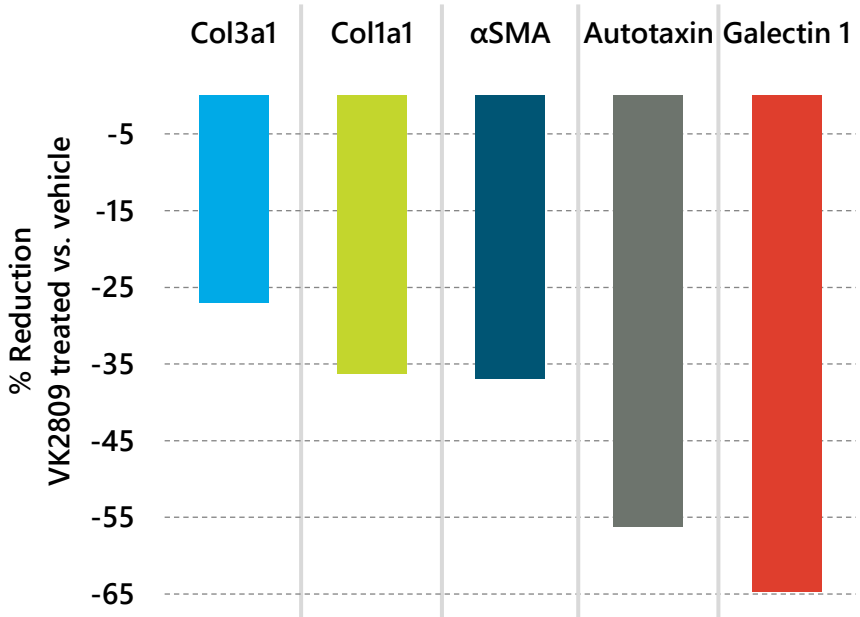
	Fibrosis	Type I Collagen	Hydroxyproline
% Difference:	-50.2%	-60.2%	-46.3%
p-value:	<0.01	<0.005	0.01

VK2809 significantly improved NASH and fibrosis in this model

VK2809: Representative Gene Effects, DIO NASH Model

- VK2809 reduces expression and signaling of key fibrosis drivers
- Gene expression changes align with observed improvement in fibrosis histology
- Improvement in genes associated with lipid metabolism, insulin sensitivity also observed

Change Pro-Fibrogenic Gene Expression Following VK2809 Treatment



	Col3a1	Col1a1	αSMA	Autotaxin	Galectin 1
% Difference	-27.1%	-36.3%	-37.0%	-56.3%	-64.7%
p-value	0.07	<0.05	<0.05	<0.001	<0.001

TRβ mechanism provides broad histologic benefits; improving steatosis, inflammation, fibrosis.

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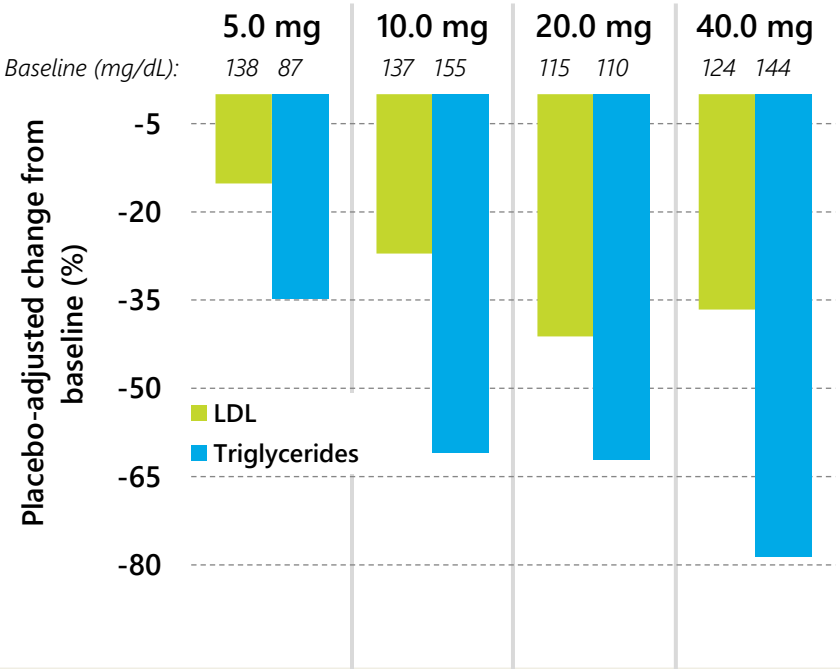
VK2809 Early Clinical Highlights: 14-Day Phase 1b Study

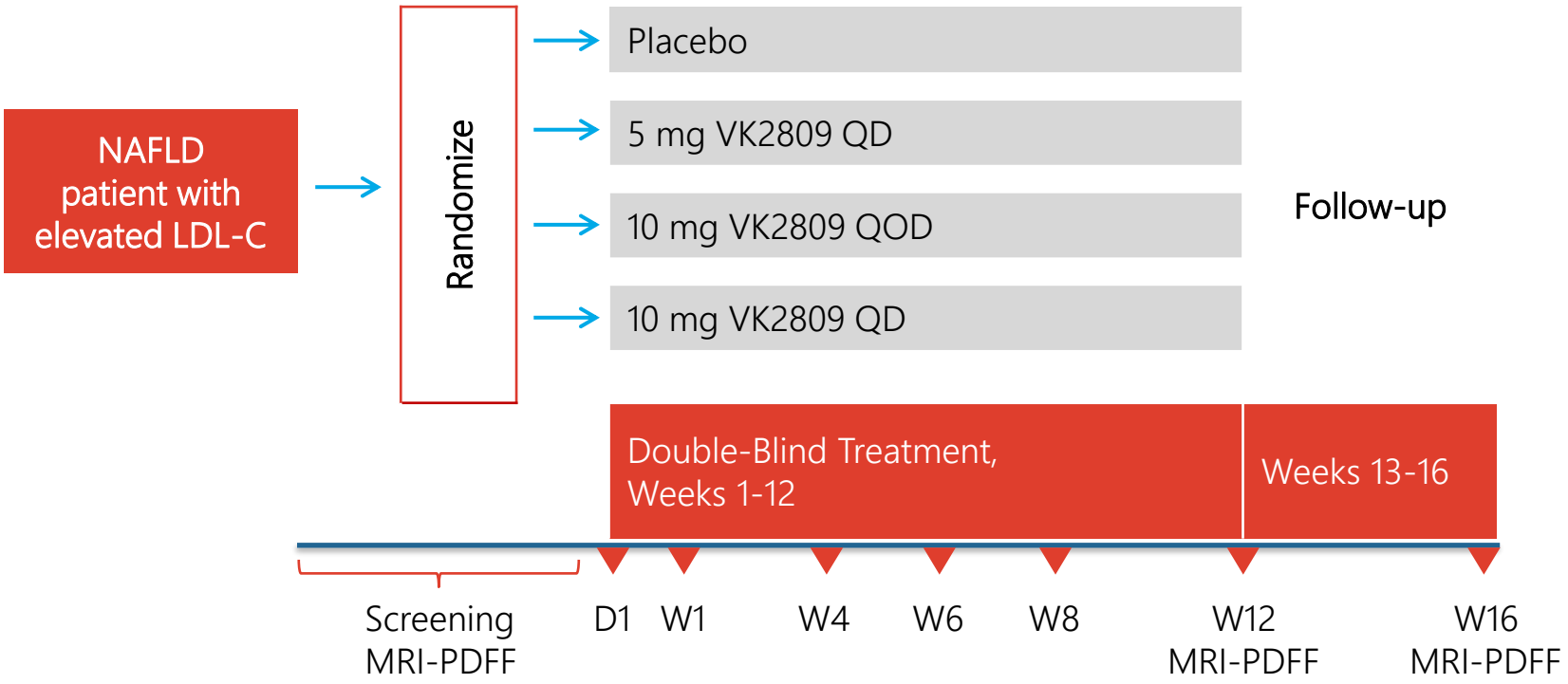
- Placebo-controlled trial (n=56), mild hypercholesterolemia
- Results: clinically, statistically significant reductions in LDL and triglycerides
- Encouraging safety and tolerability, no SAEs
- Results supported a proof-of-concept study in patients with NAFLD and elevated LDL-C

Placebo-Adjusted Change From Baseline (%)



	5.0 mg	10.0 mg	20.0 mg	40.0 mg
Placebo-adjusted reduction, LDL:	-15.2% p=0.026	-27.1% p=0.0003	-41.2% p<0.0001	-36.6% p<0.0001
Placebo-adjusted reduction, triglycerides:	-34.8% p=0.052	-61.0% p=0.0019	-62.1% p=0.0007	-78.6% p=0.0001

VK2809-201: Phase 2a Study Design

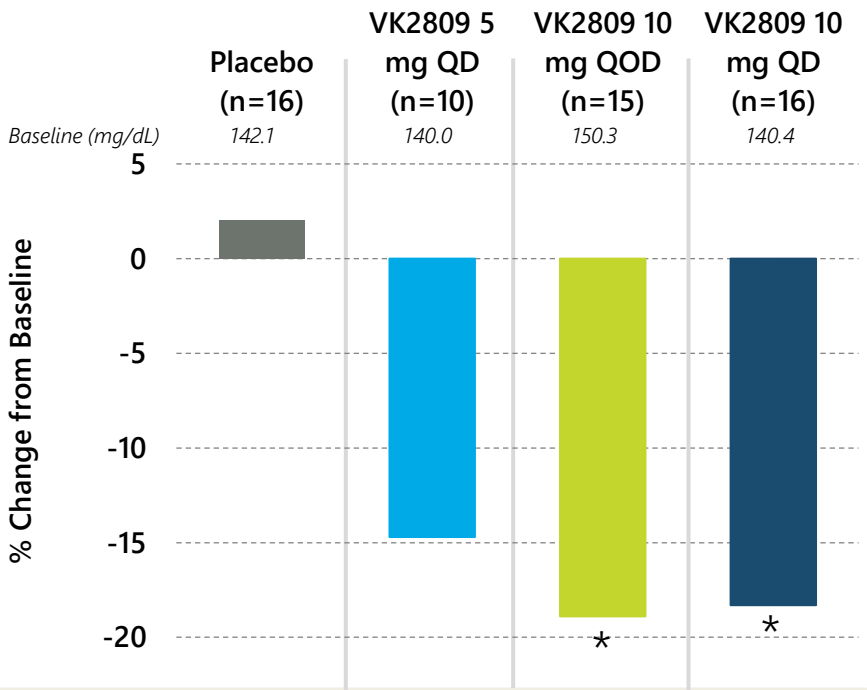


- Multi-arm, dose-ranging, 12 week Phase 2a trial
  - Target enrollment: 20 patients per arm
  - **Primary endpoint:** Change in LDL-C vs. placebo
  - **Secondary endpoint:** Change in liver fat by MRI-PDFF
  - **Exploratory endpoints:** Changes in atherogenic proteins

VK2809 Significantly Reduced LDL-C After 12 Weeks

- All VK2809 cohorts statistically significantly reduced vs. baseline
- Placebo-adjusted change from baseline
  - 5 mg QD: -23.7 mg/dL
  - 10 mg QOD: -27.1 mg/dL
  - 10 mg QD: -28.3 mg/dL

Mean % Change in LDL-C at 12 Weeks



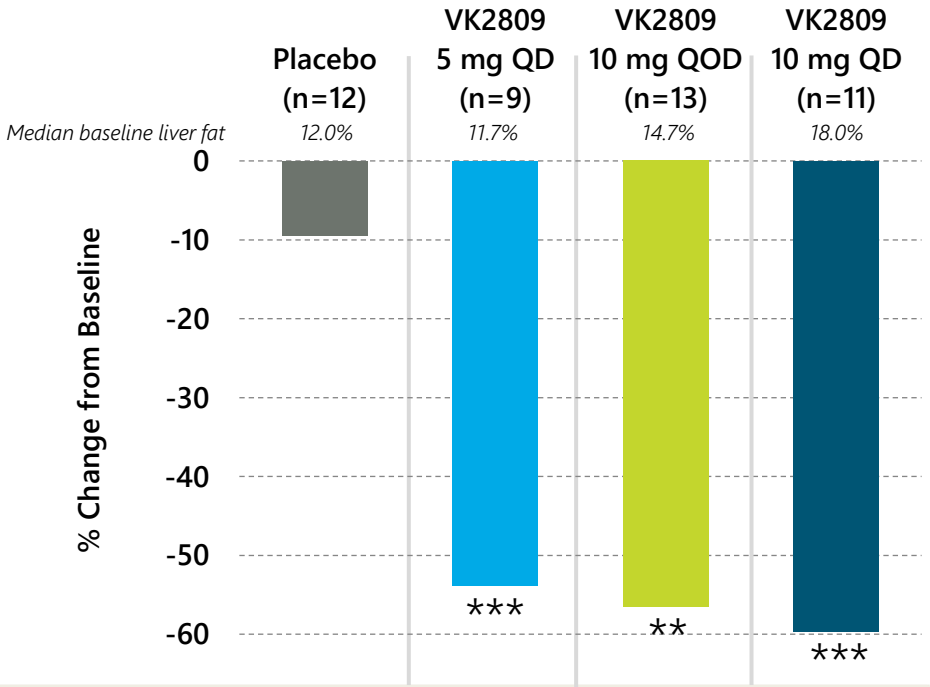
	Placebo (n=16)	VK2809 5 mg QD (n=10)	VK2809 10 mg QOD (n=15)	VK2809 10 mg QD (n=16)
% Change	2.0%	-14.7%	-18.9%	-18.3%
p-value vs. placebo	-	0.080	0.034	0.025

*p<0.05

VK2809 Produced Significant Relative Reductions in Liver Fat

- Significant Relative Reductions from Baseline in Liver Fat by MRI-PDFF
- Maximal reductions at Week 12
  - 5 mg QD: 78%
  - 10 mg QOD: 72%
  - 10 mg QD: 76%

Median Relative % Change in Liver Fat at 12 Weeks

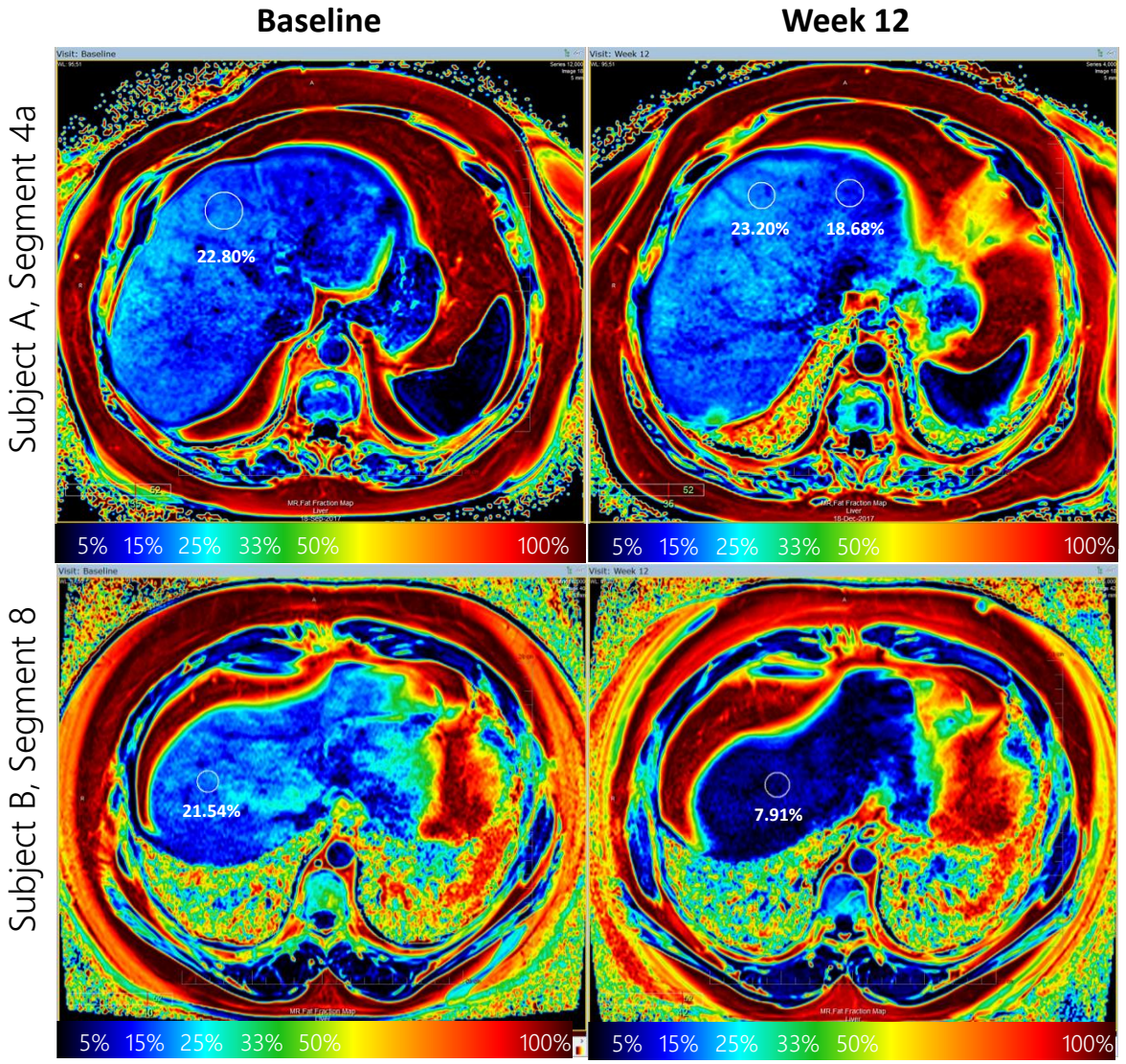


	Placebo (n=12)	VK2809 5 mg QD (n=9)	VK2809 10 mg QOD (n=13)	VK2809 10 mg QD (n=11)
% Change	-9.4%	-53.8%	-56.5%	-59.7%
p-value vs. placebo	-	0.0001	0.0018	0.0004

*p<0.05; **p<0.01; ***p<0.001

Representative Fat Reduction, VK2809 and Placebo Subject

- Upper images (placebo) show minimal change to liver color, fat content
- Lower images (VK2809) demonstrate dramatic change in liver shade, indicating reduced fat



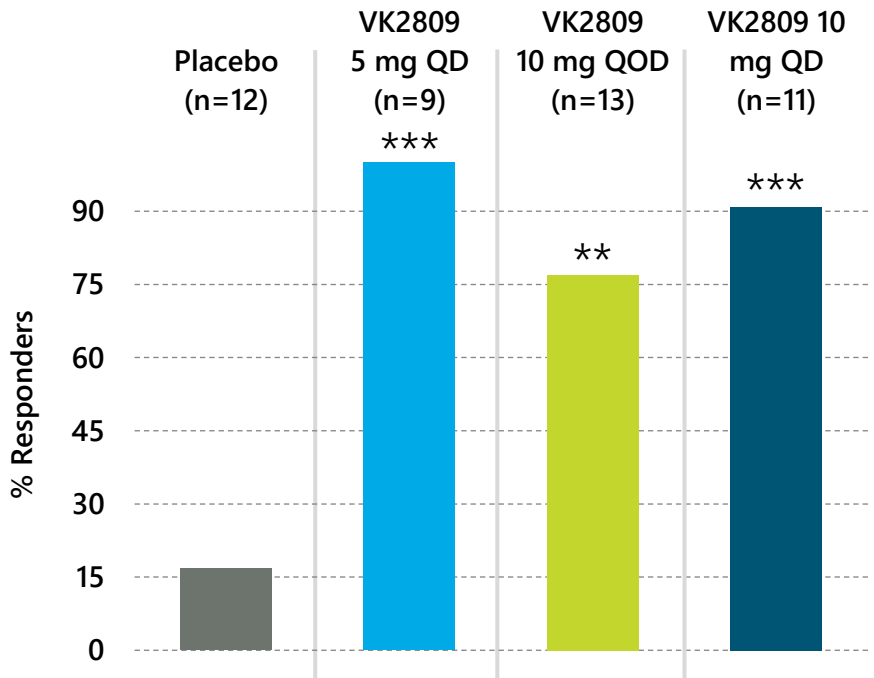
Overall Mean Hepatic Fat Values

Subject, Dose	Baseline	Week 12	Absolute Change	Relative Change
Subject A Placebo	20.3%	22.6%	2.3%	11.4%
Subject B 10 mg QD	24.6%	6.0%	-18.6%	-75.6%

VK2809 Cohorts Demonstrated High Relative Response Rates

- Up to 100% of VK2809 patients experienced response, as defined by ≥30% decrease in liver fat at Week 12
- Combined VK2809 cohorts demonstrated 88% response rate
- 70% of all patients receiving VK2809 demonstrated liver fat reductions ≥50%
- Reduction in liver fat correlated with improved odds of long-term histology benefit[1]

Patients with ≥30% Relative Reduction in Liver Fat at 12 Weeks



	Placebo (n=12)	VK2809 5 mg QD (n=9)	VK2809 10 mg QOD (n=13)	VK2809 10 mg QD (n=11)
Responders	16.7%	100%	76.9%	90.9%
p-value vs. placebo	-	0.0002	0.0048	0.0006

*p<0.05; **p<0.01; ***p<0.001

1) *Ther. Adv. Gastroenterol.*, 9(5), 692-701, (2016).

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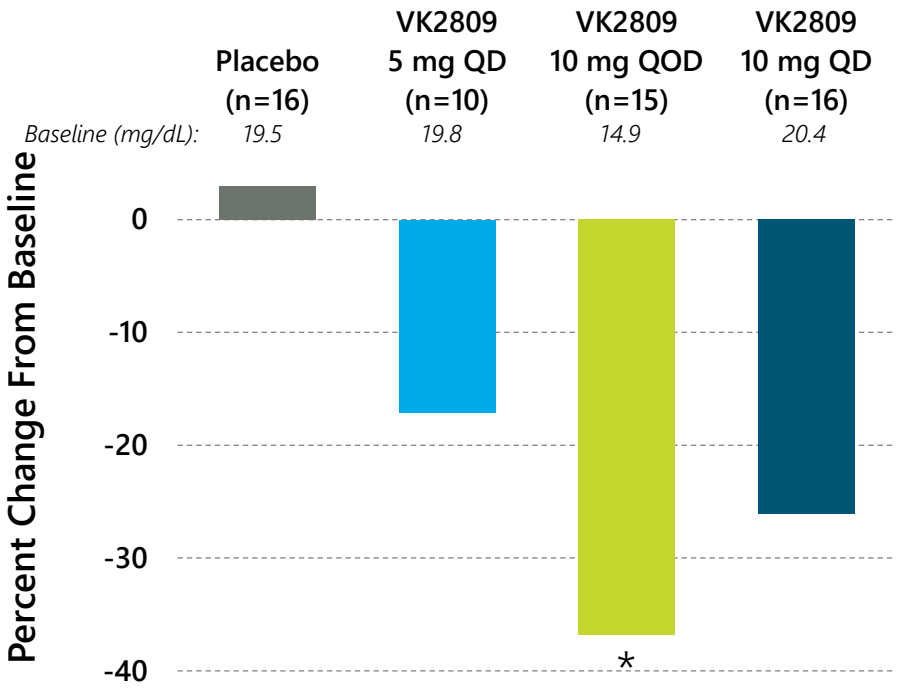
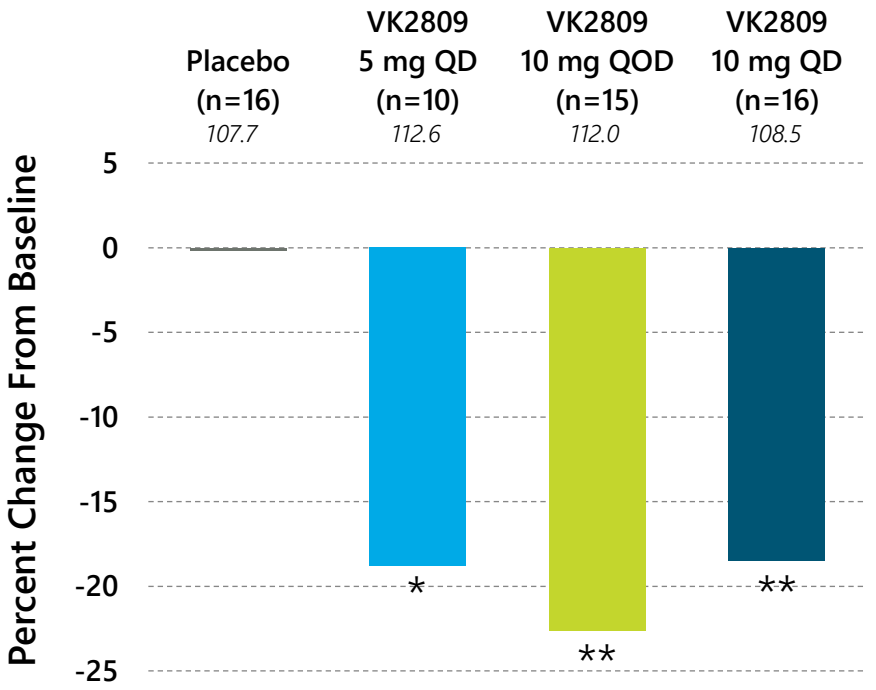
VK2809 Improved Atherogenic Protein Levels at 12 Weeks

Mean Change in Lipoprotein(a) at Week 12



Mean Change in Apolipoprotein B at Week 12



	Placebo (n=16)	VK2809 5 mg QD (n=10)	VK2809 10 mg QOD (n=15)	VK2809 10 mg QD (n=16)
Change	3.0%	-17.1%	-36.8%	-26.1%
p-value	-	0.21	0.048	0.060

	Placebo (n=16)	VK2809 5 mg QD (n=10)	VK2809 10 mg QOD (n=15)	VK2809 10 mg QD (n=16)
Change	-0.2%	-18.8%	-22.6%	-18.5%
p-value	-	0.014	0.0023	0.0081

*p<0.05; **p<0.01; ***p<0.001

- Reductions in Lp(a), ApoB achieved at 12 Weeks
- Suggests potential cardiovascular benefit

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VK2809-201: Encouraging Safety Profile Through 12 Weeks

• No SAEs observed	No SAEs reported in any VK2809 clinical study to date
• Mean ALT, AST levels in VK2809-treated subjects reduced relative to placebo at Week 12	Patients with elevated baseline ALT demonstrated greater improvement relative to placebo at Weeks 12 and 16
• No other liver function tests significantly different from placebo	Direct bilirubin, indirect bilirubin, alkaline phosphatase, INR
• No clinically meaningful changes in other key markers among VK2809-treated patients relative to placebo	Thyroid hormones (fT4, tT3, TSH); Cardiovascular markers (troponin, CK-MB, NT proBNP); Vital signs (BP, heart rate, weight)
• Excellent tolerability	GI and nausea events numerically lower vs. placebo

VK2809: Phase 2a Summary and Conclusions

- VK2809 produced robust reduction in liver fat on MRI-PDFF in NAFLD patients after 12 weeks of oral dosing

88% of patients receiving VK2809 experienced ≥30% reduction in liver fat content, including all patients receiving 5 mg doses

70% experienced liver fat reductions ≥50%

- VK2809 produced significant reduction in plasma lipids, suggesting long-term CV benefit

LDL-C, triglycerides, and atherogenic proteins Apo B, Lp(a)

- VK2809 was safe and well-tolerated in this 12-week Phase 2 study

No SAEs observed, discontinuations well-balanced across cohorts

VK2809 Clinical Highlights and Current Status

- Phase 1 studies demonstrated safety, predictable PK, robust lipid-lowering effects
- 12-Week Phase 2 study demonstrated potent liver fat reduction
- No drug-drug interaction when co-administered with atorvastatin
- Profile supports further development in biopsy-confirmed NASH
- VOYAGE 12-month Phase 2b NASH study initiated 4Q19

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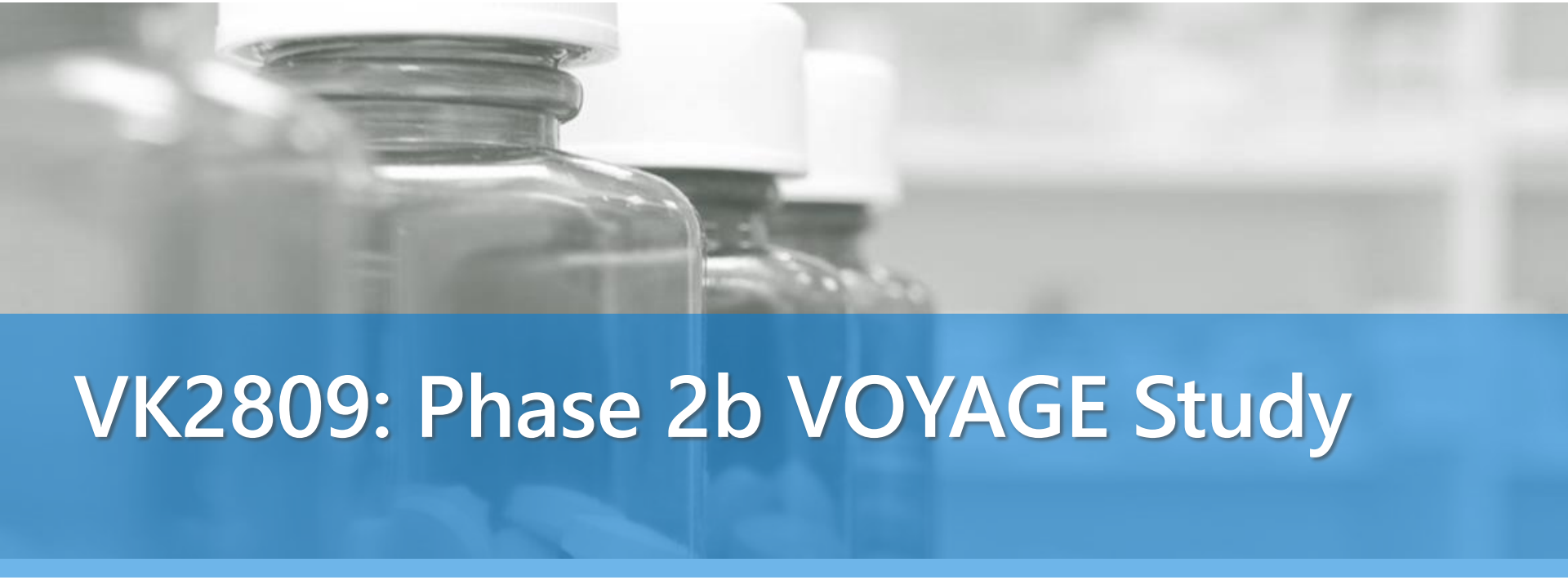
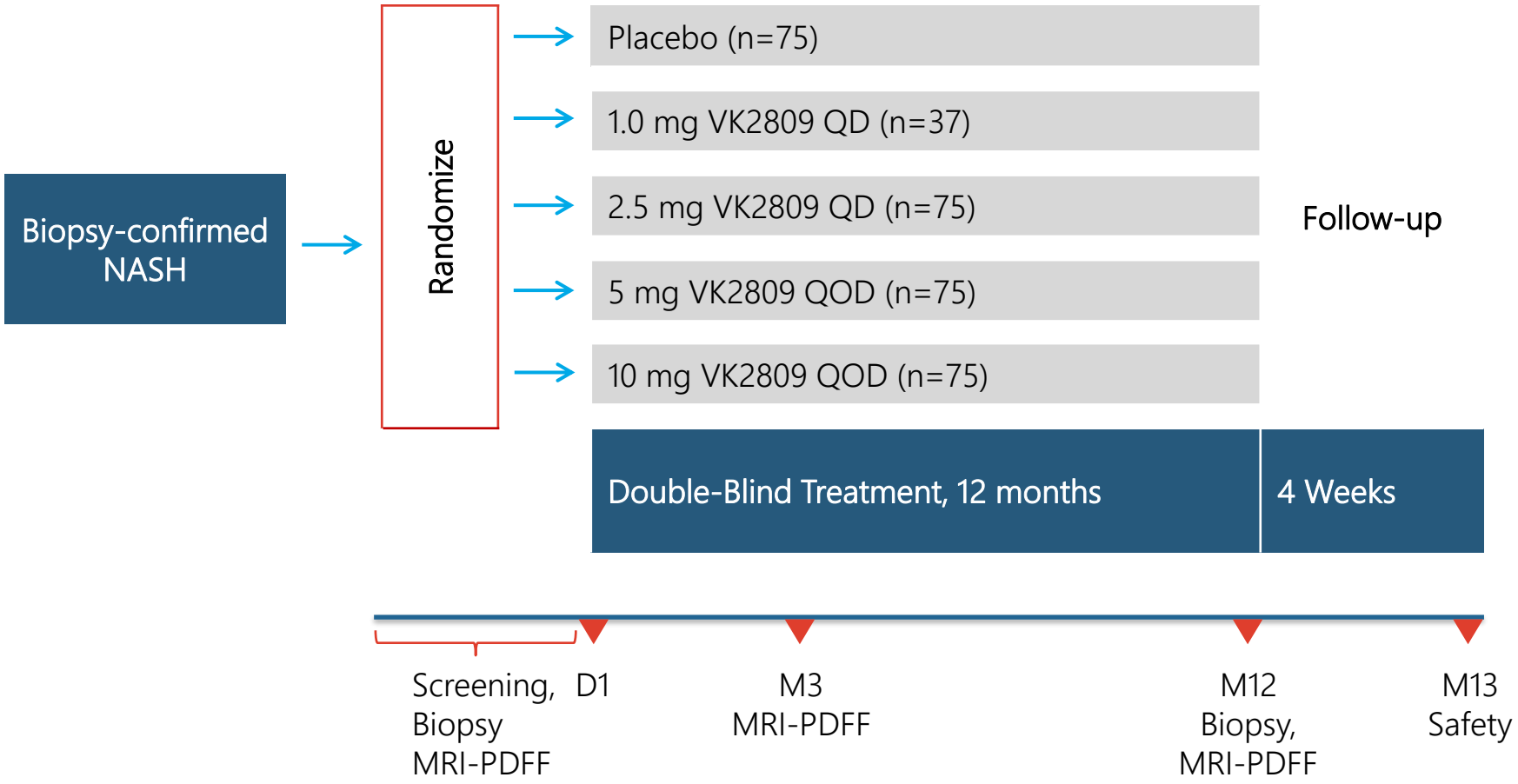


VK2809: Phase 2b VOYAGE Study

VOYAGE Study: 12-Month Phase 2b Study of VK2809



- Multi-arm, dose-ranging, 12-month Phase 2 trial
  - **Primary endpoint:** Change in MRI-PDFF vs. placebo at 3 months
  - **Secondary endpoints:** Change in histology at 12 months (NAS, fibrosis markers, etc.)

VOYAGE Study: 12-Month Phase 2b Study of VK2809

- Key inclusion criteria
  - Biopsy-confirmed NASH with NAS ≥4
  - Liver fat content ≥8%
  - F2-F3 fibrosis, up to 25% F1
- Primary endpoint: Change in liver fat content at week 12
- Secondary, exploratory endpoints: Change in histology at 12 months

Closing Comments: VK2809 Competitive Advantages

- Currently >40 NASH programs in Phase 2 or Phase 3 development
- What differentiates VK2809 from the crowd?

• Orally available	Preferred route of administration for chronic therapy
• Liver-targeted	Reduces risk of undesired effects in other tissues
• Potently reduces liver fat	Weight loss and reduced liver fat correlate with NASH resolution, improved fibrosis markers
• Reduces systemic lipids, may improve overall metabolic profile	Bodes well for potential long-term CV benefit No elevations in other lipids that may require polypharmacy
• Well tolerated	No GI impact, no pruritis or other tolerability issues to date

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Rare Disease Program

VK0214

X-Linked Adrenoleukodystrophy

VK0214 for X-ALD

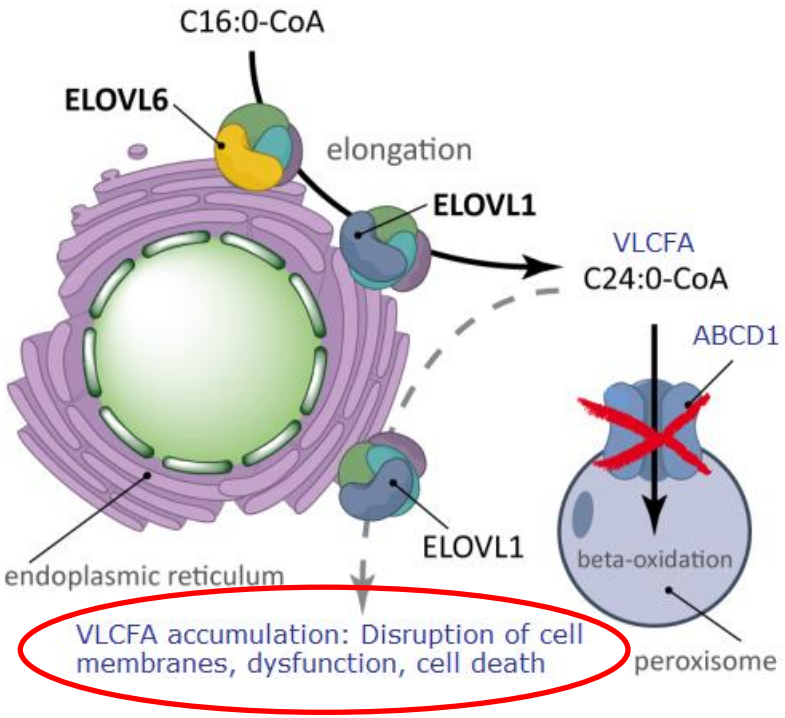
- X-Linked adrenoleukodystrophy (X-ALD)
  - Orphan neurodegenerative disorder
  - X-linked: Carried by females, primarily manifesting in males
  - No cure, no approved therapy
- Most severe form: Cerebral ALD
  - Rapidly progressive inflammatory demyelination; disruption of BBB
  - Affects ~35% before age 12 (CCALD), ~20% between age 20 – 35 (CALD)
  - Deterioration in speech, cognition; vegetative state within 3-5 years
- Most common form: Adrenomyeloneuropathy (AMN)
  - Affects spinal cord, motor neurons; no inflammatory component or brain involvement
  - Affects nearly all adult patients; considered "default" manifestation of ALD
  - Progressive motor impairment; wheelchair confinement, leg paralysis common

(1) *Biochimie*, **98** (2014) 135-142. (2) *Ann. Neurol.* **49**:512-517 (2001). (3) *Biochim. Biophys. Acta* **1822** (2012) 1465-1474. (4) *Orphanet J. Rare Dis.* **7**:51 (2012). (5) *Brain Pathol.* **20**(4): 845-856 (2010).

TRβ: X-Linked Adrenoleukodystrophy

Caused by mutation in gene for the ATP-Binding Cassette transporter D1 (ABCD1)

- Peroxisomal transporter of very long chain fatty acids (VLCFA)



ABCD1: Normal function to transport VLCFA into peroxisome for degradation

X-ALD: Defective ABCD1 leads to accumulation of VLCFA in tissues

High VLCFA levels disrupt cell membranes; inflammatory demyelination in brain tissue; motor neuron deterioration

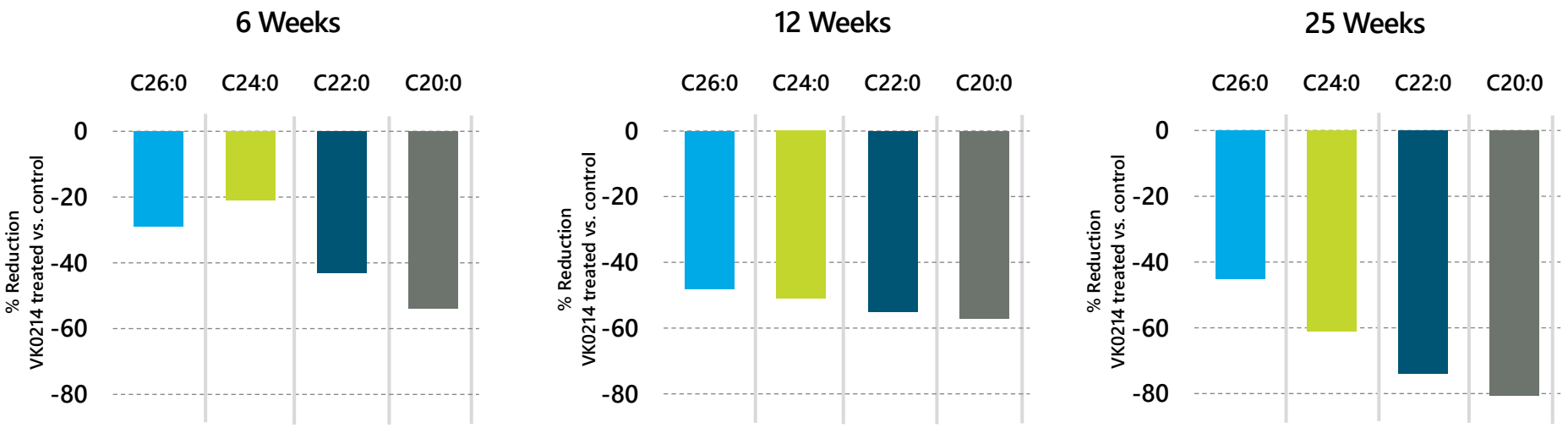
TRβ Agonists: Stimulate expression of compensatory transporters ABCD2, 3; may mitigate VLCFA elevation

Graphic adapted from http://www.x-ald.nl/origin-and-metabolism-of-vlcfa/.

VK0214: *In Vivo* Proof-of-Concept Data, ABCD1 KO Mouse

- ABCD1 Knockout model: Mimics biochemical features of human X-ALD
- VK0214: Durable and progressive reductions in plasma VLCFAs
  - Tissue effects suggest encouraging CNS activity following long-term exposure

Reductions in Plasma VLCFA-LPC, ABCD1 Knockout Model

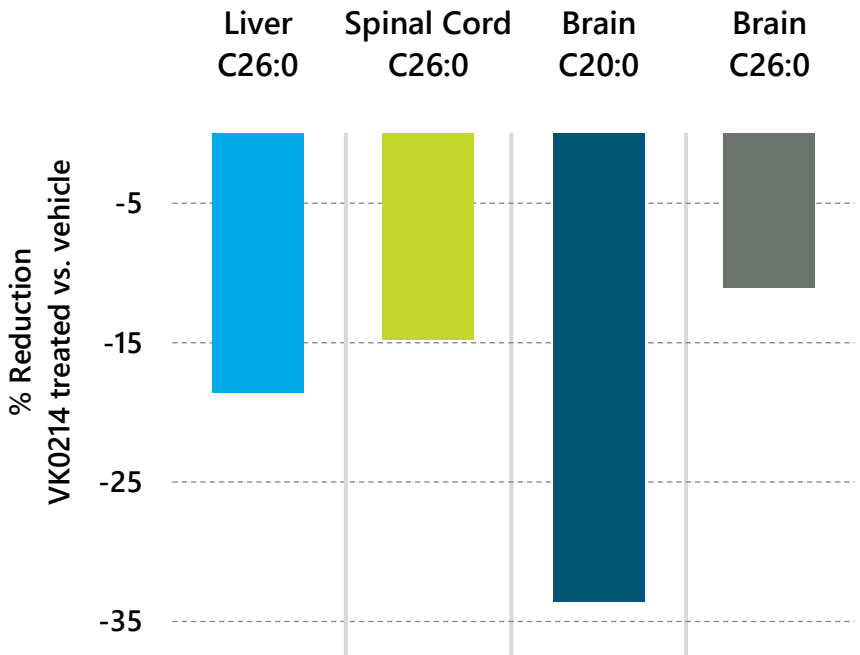


	C26:0	C24:0	C22:0	C20:0
% Chg:	-29%	-21%	-43%	-54%
p-value:	<0.0001	<0.005	<0.0001	<0.0001
% Chg:	-48%	-51%	-55%	-57%
p-value:	<0.0001	<0.0001	<0.0001	<0.0001
% Chg:	-45%	-61%	-74%	-82%
p-value:	<0.0001	<0.0001	<0.0001	<0.0001

VK0214: Reduces VLCFA Levels in Key Tissues

- Significant VLCFA reductions observed in multiple tissues
- Encouraging evidence of CNS activity
- Reductions in multiple VLCFAs consistent with plasma observations
- Suggests potential benefit in both cerebral and AMN forms of X-ALD
- Next steps: IND-filing planned mid-year 2020

Change in Tissue VLCFAs: CNS and Peripheral Tissue



	Liver C26:0	Spinal Cord C26:0	Brain C20:0	Brain C26:0
% Difference:	-19%	-15%	-34%	-11%
p-value:	<0.05	<0.01	<0.0001	0.07

Financial Summary

- Capital structure and summary financials

Capital Structure[1]	In '000s
Shares outstanding	72,563
Options, RSUs	4,198
Warrants	5,736
Total shares, options, RSUs, warrants	82,497

Financials	Mar 31, 2020 ($'000s)
Cash burn 1Q 2020	$6,477
Cash and ST Investments	$269,161

Notes: 1) As of March 31, 2020.

Investment Highlights

- **Focused on novel therapeutics for metabolic and endocrine diseases**
  - Clinical programs demonstrate best-in-class efficacy data
- **Metabolic Disease Program: VK2809 for NASH**
  - Novel, selective thyroid receptor-β (TRβ) agonist
  - Phase 2a results demonstrate significant reduction in liver fat content, lipids
  - Phase 2b VOYAGE trial ongoing
- **Rare Disease Program: VK0214 for X-ALD**
  - Novel, selective thyroid receptor-β agonist
  - In vivo data show improvement in key biomarkers
  - Clinical development to commence 2020
- **Other Pipeline Programs: Musculoskeletal and metabolic disorders**

VIKING
THERAPEUTICS

Corporate Presentation