VIKING THERAPEUTICS

Corporate Presentation

January 2021

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.

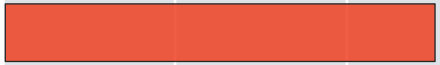
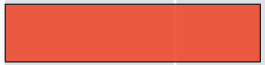
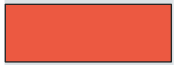
The forward-looking statements included in this presentation represent our views as of the date of this presentation. We anticipate that subsequent events and developments will cause our views to change. While we may elect to update these forward-looking statements in the future, we specifically disclaim 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 Programs: 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 (TRβ) agonist
  - In vivo data show improvement in key biomarkers
  - Phase 1 studies underway
- Other Pipeline Programs: Musculoskeletal and metabolic disorders

Pipeline Overview

Development Programs	Indication	Stage of Development				Status
		Preclin	Phase 1	Phase 2	Phase 3	
VK2809 (TRβ agonist)	*NASH*					Phase 2b VOYAGE trial ongoing
VK0214 (TRβ agonist)	*X-ALD*					Phase 1 studies ongoing
Other Programs						
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

VIKING THERAPEUTICS

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: Phase 1 ongoing

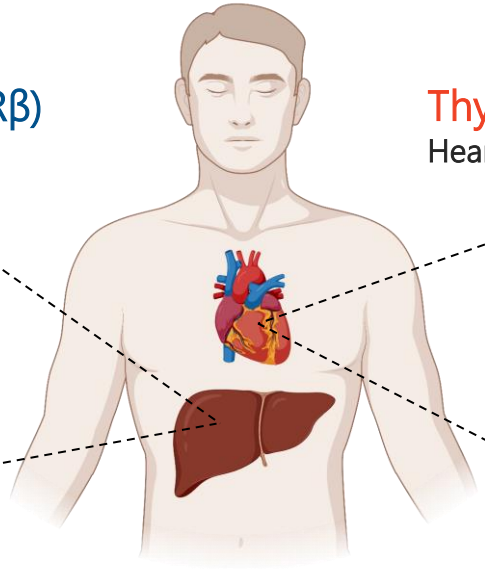
Thyroid Hormone Receptor Overview

Nuclear hormone receptors: 2 main types

Thyroid hormone receptor beta (TRβ)
Liver

Positive effects

- Regulates lipid metabolism
- Reduces LDL-C, triglycerides, atherogenic proteins
- Improves metabolic control



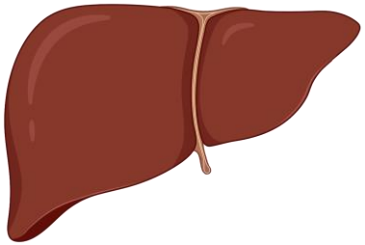
Thyroid hormone receptor alpha (TRα)
Heart, skeletal muscle

Negative effects

- Proarrhythmic potential
- Elevates heart rate
- Bone/cartilage effects

Therapeutic goal, lipid setting: Beta receptor selectivity, minimize alpha effects

Thyroid Receptor β Agonists for NAFLD and NASH

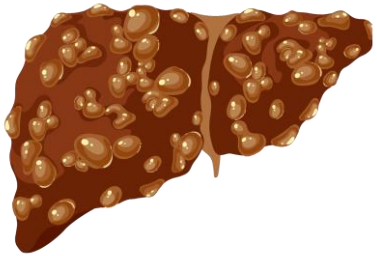


NASH Progression

Accumulation of fatty acids, triglycerides; NAFLD

Oxidative stress, inflammatory response

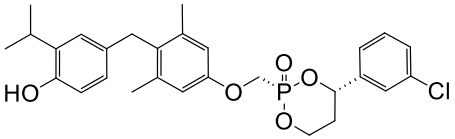
NASH: Steatosis, ballooning, hepatocyte damage



- 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

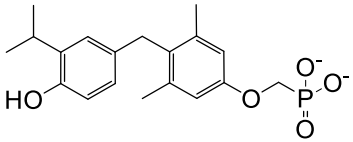
VK2809: Unique Liver-Targeted Characteristics



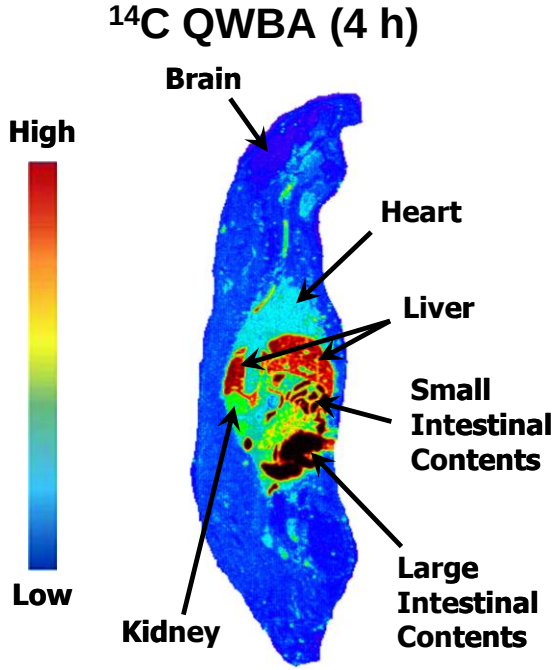
VK2809, Novel Prodrug

Following oral dosing:

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



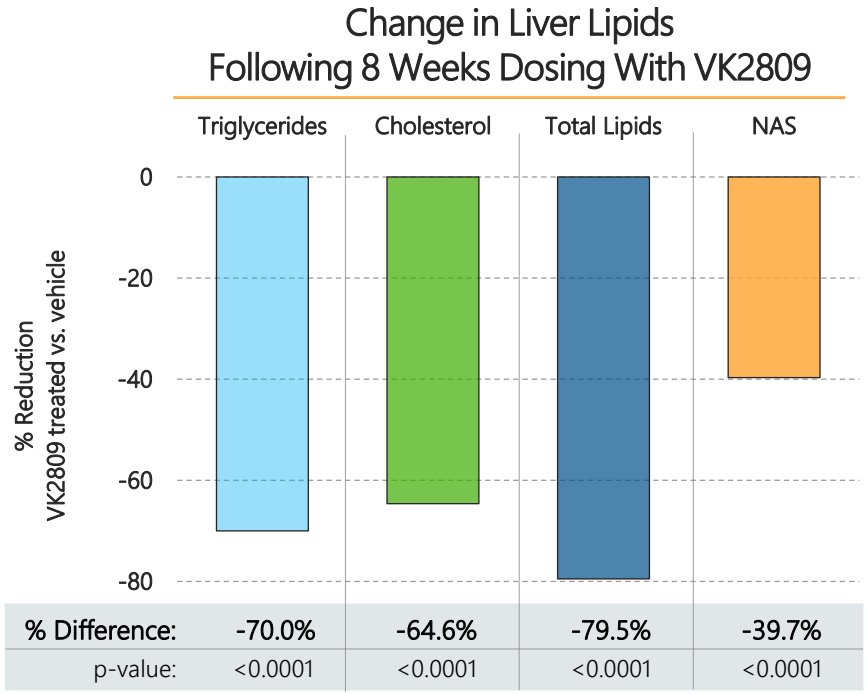
VK2809A, Potent TRβ Agonist, 2.2 nM Ki



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

VK2809 Significantly Reduces Steatosis in Diet-Induced NASH

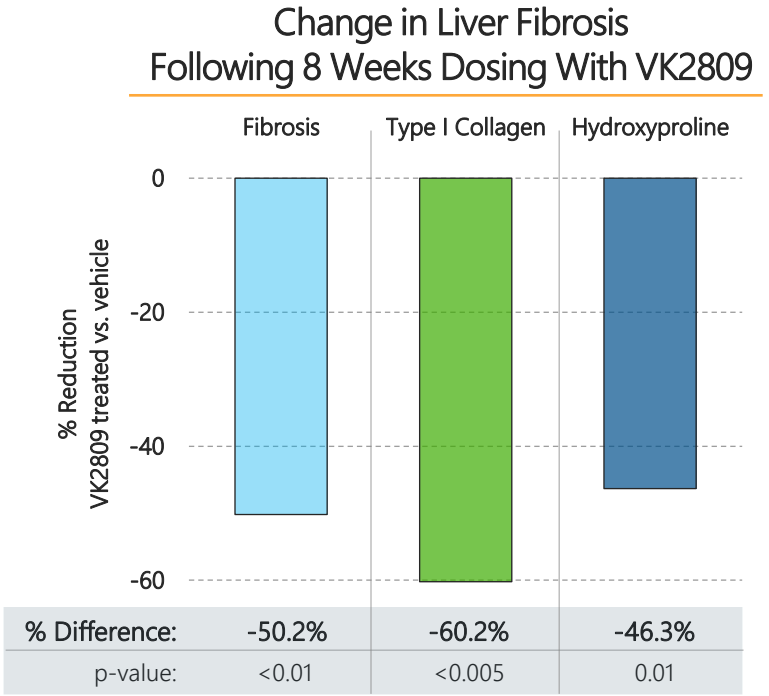
- Evaluation in biopsy-confirmed diet-induced NASH model
  - Rodent model designed to reflect progression of disease in humans
  - Animals biopsied pre-study; only those with NASH *and* fibrosis selected
  - VK2809 dosed once-daily for 8 weeks



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

VK2809 Improves Fibrosis in Diet-Induced NASH Model

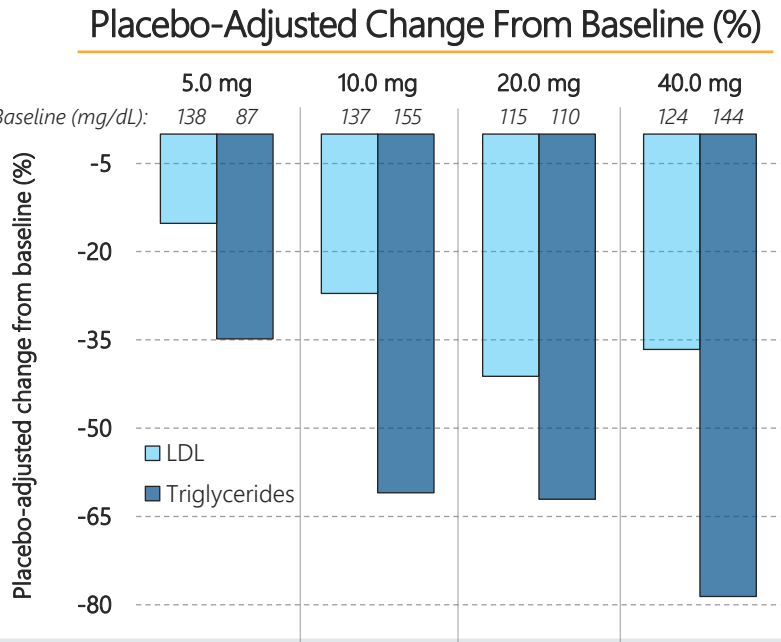
- Significant reductions in fibrosis, collagen, hydroxyproline after 8 weeks
- Supports thesis that selective TRβ activation produces broad metabolic benefits



VK2809 significantly improved NASH and fibrosis in this model

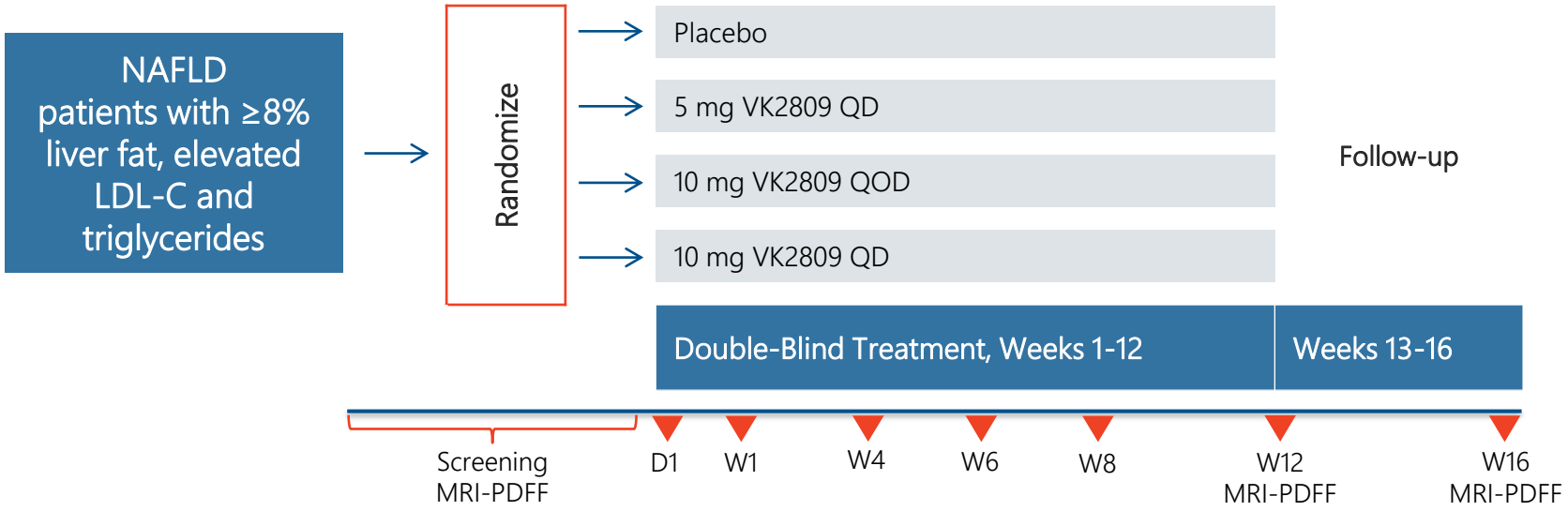
VK2809 Early Clinical Highlights: 14-Day Phase 1b Study

- 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



	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

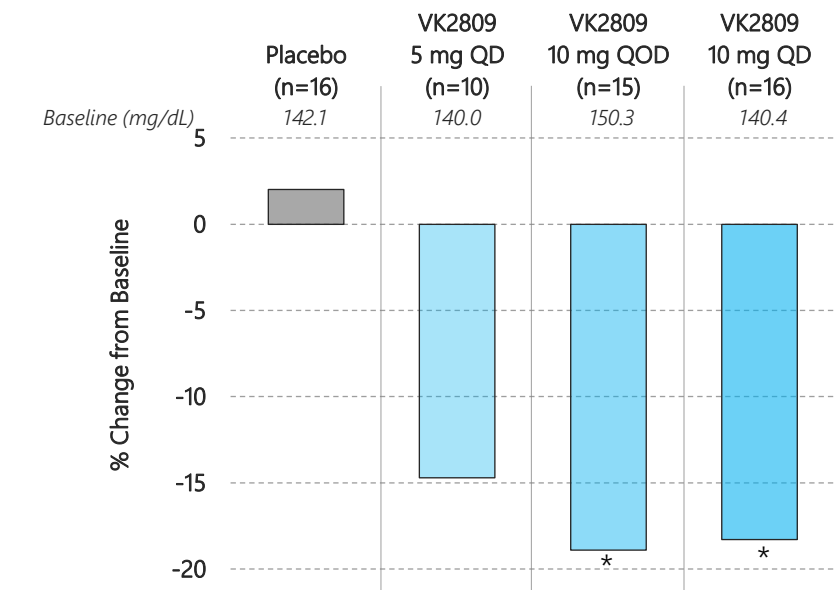


- Multi-arm, dose-ranging, 12 week Phase 2a trial
  - **Primary endpoint:** Change in LDL-C vs. placebo
  - **Secondary endpoint:** Change in liver fat by MRI-PDFF
  - **Exploratory endpoints:** Changes in atherogenic proteins

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

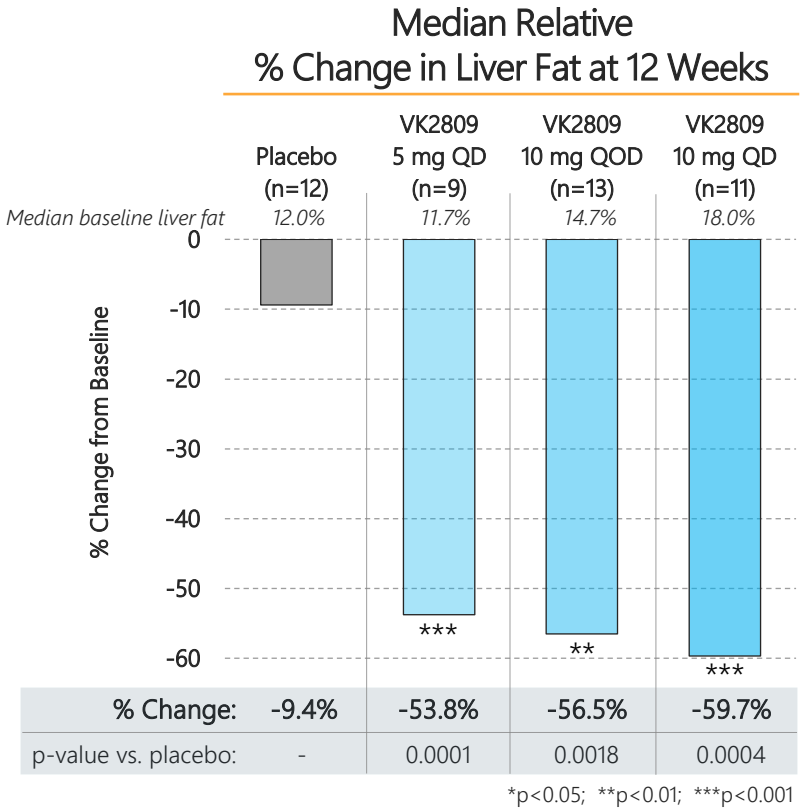


	Placebo (n=16)	VK2809 5 mg QD (n=10)	VK2809 10 mg QOD (n=15)	VK2809 10 mg QD (n=16)
% Change:	2.0%	-14.7%	-18.9%	-18.3%
p-value vs. placebo:	-	0.080	0.034	0.025

* p<0.05

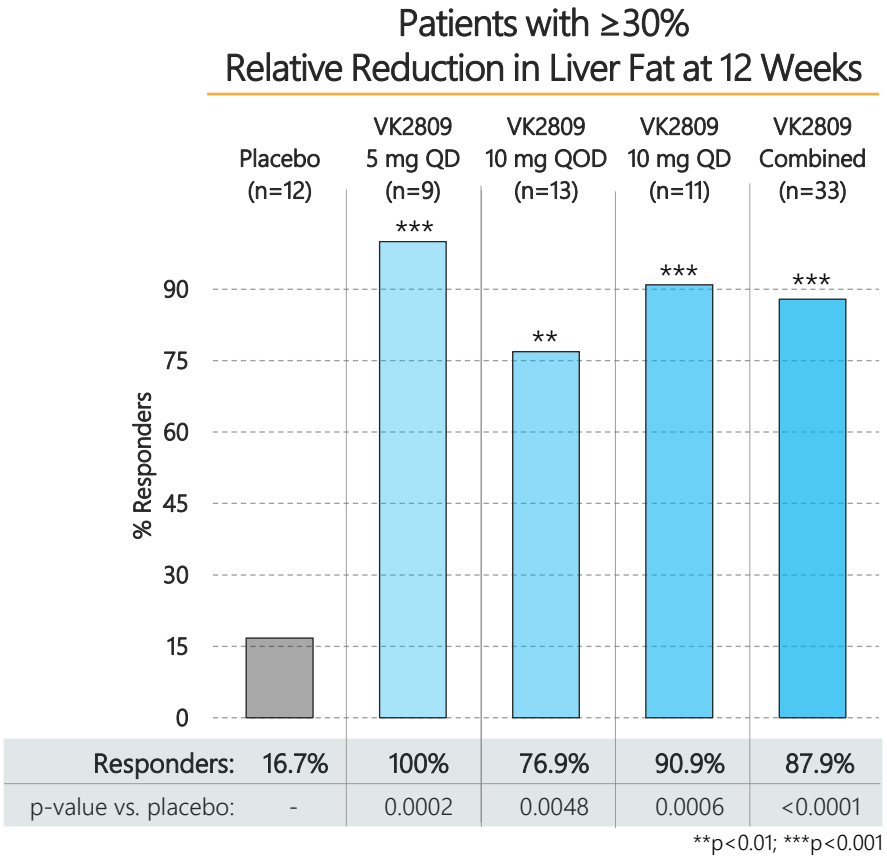
VK2809 Produced Significant Relative Reductions in Liver Fat

- 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%



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]



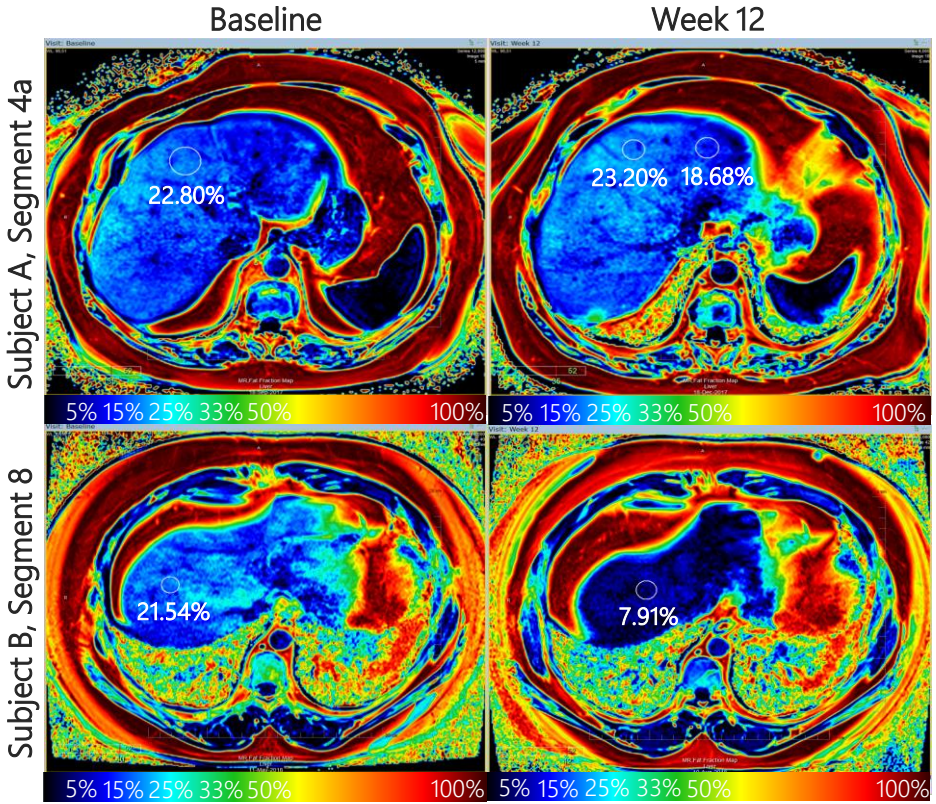
1) *Ther. Adv. Gastroenterol., 9(5), 692-701, (2016).*

Representative Fat Reduction, VK2809 and Placebo Subject

- **Placebo**: Minimal change to liver color, fat content
- **VK2809**: Dramatic change in liver shade, indicating reduced fat

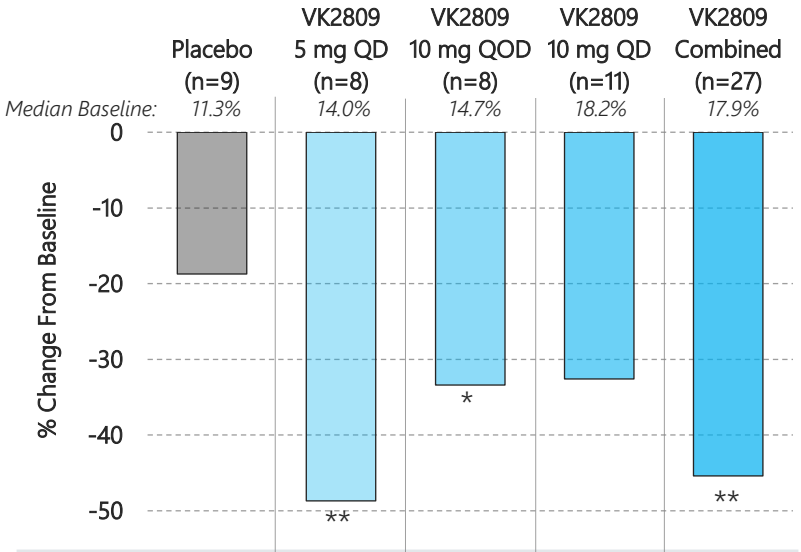
Overall Mean Hepatic Fat Values

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%



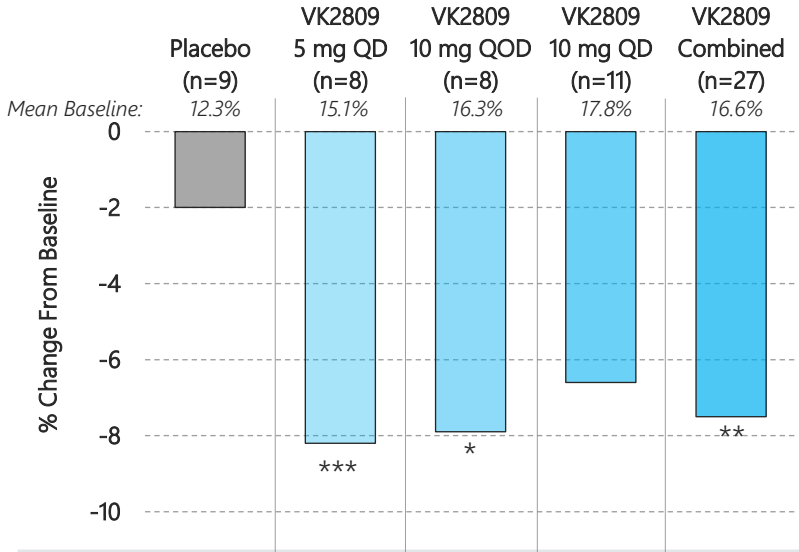
Durable Liver Fat Reduction Observed at 16 Weeks

Median Relative % Change in Liver Fat at 16 Weeks



	Placebo (n=9)	VK2809 5 mg QD (n=8)	VK2809 10 mg QOD (n=8)	VK2809 10 mg QD (n=11)	VK2809 Combined (n=27)
% Change:	-18.7%	-48.7%	-33.4%	-32.6%	-45.4%
p-value:	-	0.0016	0.0206	0.1519	0.0053

Mean Absolute % Change in Liver Fat at 16 Weeks



	Placebo (n=9)	VK2809 5 mg QD (n=8)	VK2809 10 mg QOD (n=8)	VK2809 10 mg QD (n=11)	VK2809 Combined (n=27)
% Change:	-2.0%	-8.2%	-7.9%	-6.6%	-7.5%
p-value:	-	0.0006	0.0360	0.0674	0.0027

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

Significant reduction in MRI-PDFF maintained 4 weeks after last dose

VK2809-201: Encouraging Safety Profile Through 12 Weeks

• No SAEs observed	No SAEs reported in completed clinical studies 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

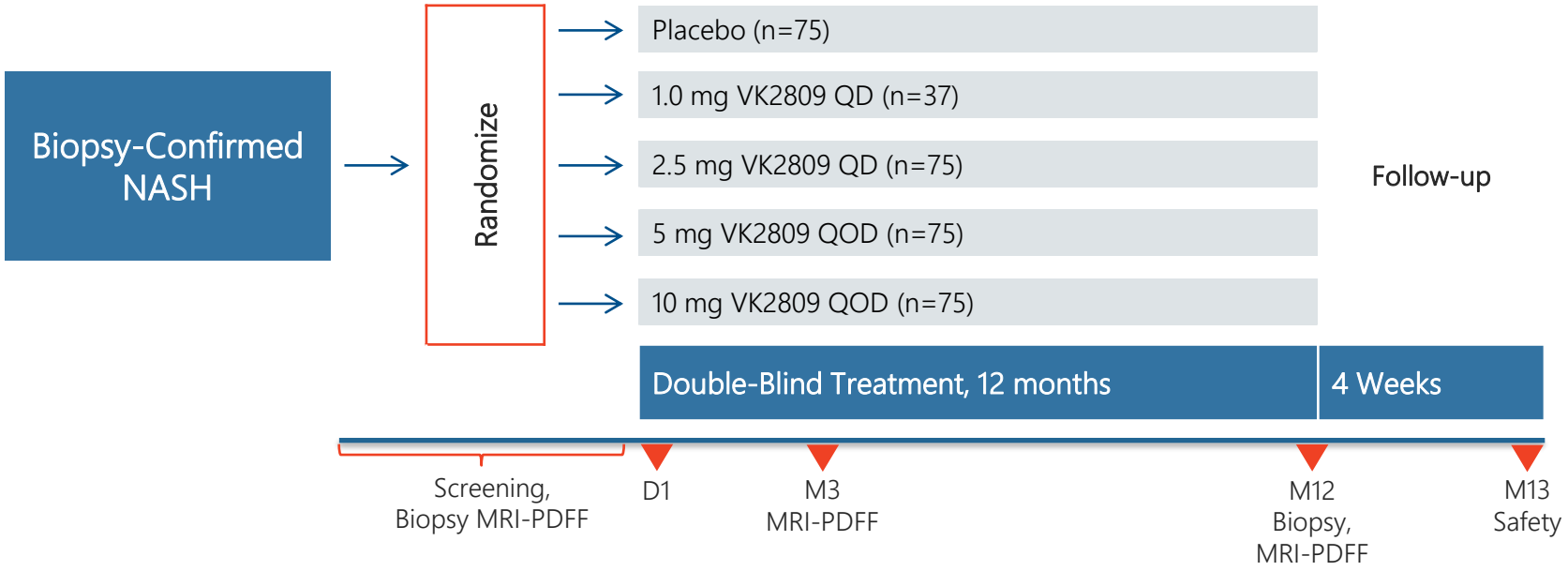
VK2809: Phase 2a Summary and Conclusions

• VK2809 produced robust reduction in liver fat on MRI-PDFF in NAFLD patients after 12 weeks of dosing	88% of patients receiving VK2809 experienced ≥30% reduction in liver fat content, including all patients receiving 5 mg doses; 70% experienced reductions ≥50%
• Consistent effects observed across common NASH risk factors	Elevated ALT, BMI, hypertension, Hispanic ethnicity
• Liver fat reduction maintained 4 weeks post-dosing	70% of VK2809 patients remained responders 28 days after conclusion of dosing
• 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 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
- Consistent efficacy demonstrated across key high-risk subsets
- 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

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.)

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

Rare Disease Program

VK0214

X-Linked Adrenoleukodystrophy

VK0214 for X-ALD

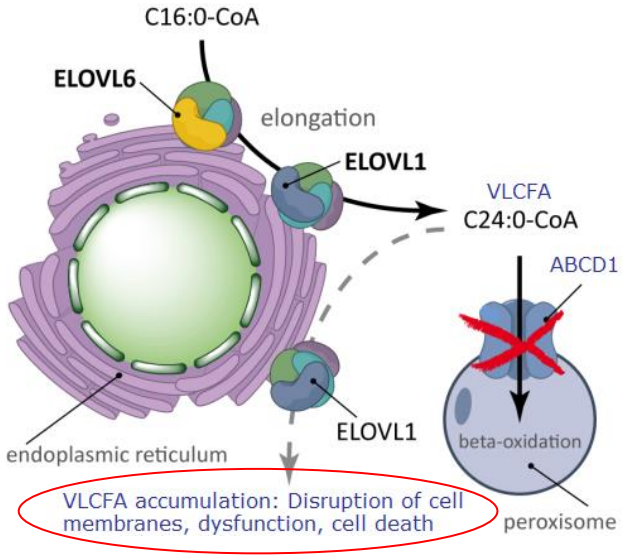
- X-Linked adrenoleukodystrophy (X-ALD)
  - Orphan neurodegenerative disorder
  - X-linked: Carried by females, primarily manifesting in males
  - No cure, no approved therapy
- Most severe form: Cerebral ALD
  - Rapidly progressive inflammatory demyelination; disruption of BBB
  - Affects ~35% before 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

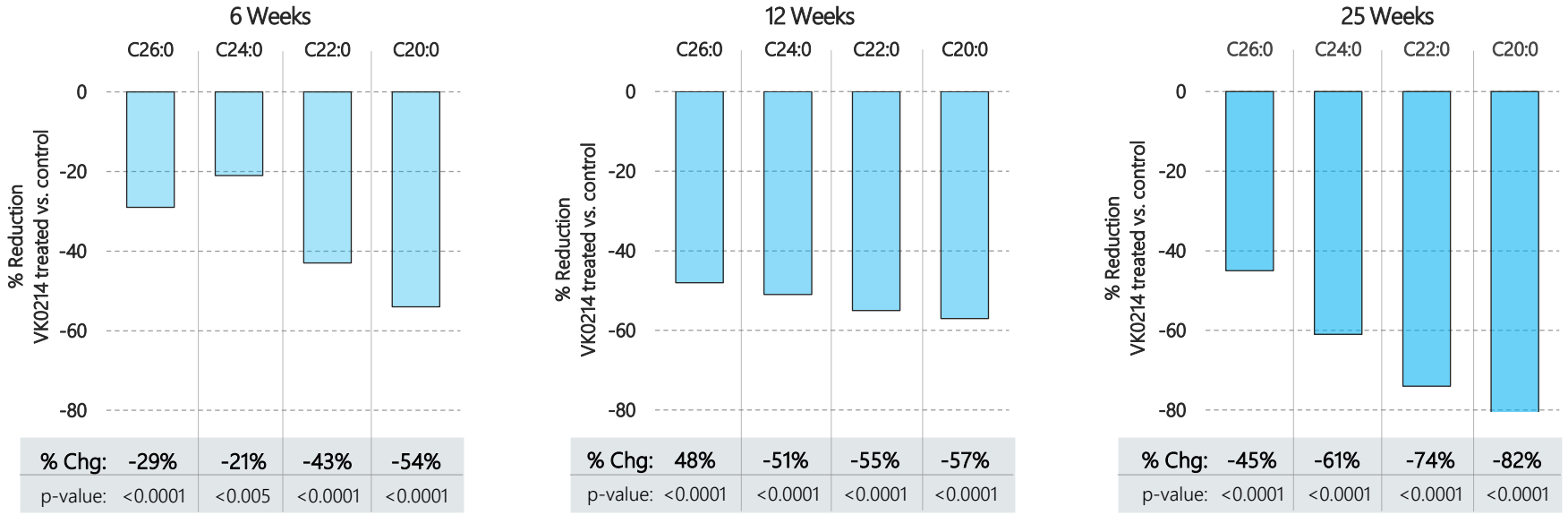
TRβ Agonists: Stimulate expression of compensatory transporters ABCD2, 3; may mitigate VLCFA elevation

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 exposure0

Reductions in Plasma VLCFA-LPC, ABCD1 Knockout Model



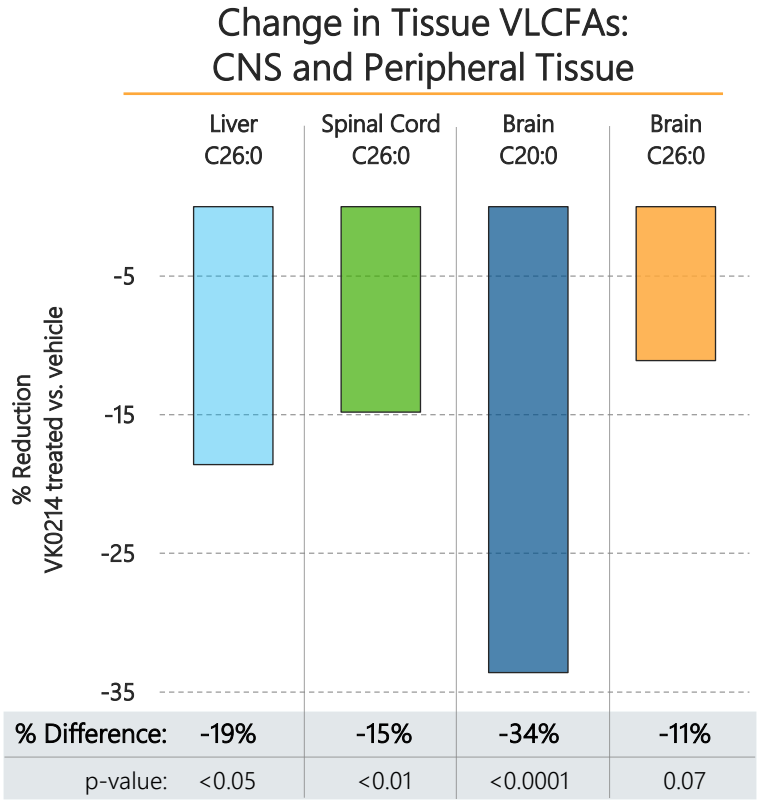
	C26:0	C24:0	C22:0	C20:0
% Chg:	-29%	-21%	-43%	-54%
p-value:	<0.0001	<0.005	<0.0001	<0.0001

	C26:0	C24:0	C22:0	C20:0
% Chg:	48%	-51%	-55%	-57%
p-value:	<0.0001	<0.0001	<0.0001	<0.0001

	C26:0	C24:0	C22:0	C20:0
% 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



VK0214 Highlights and Current Status

- Potential to be best in-class oral, small molecule TRβ therapeutic for X-ALD
- Encouraging efficacy with rapid (6 weeks) and progressive (up to 25 weeks) VLCFA reductions in plasma, brain, spinal cord and liver
- VK0214 has received Orphan Drug status from the FDA
- Phase 1 study ongoing in healthy volunteers
- Phase 1b proof-of-concept study in X-ALD patients planned for 2021

Financial Summary

- Capital structure and summary financials

Capital Structure [1]	In '000s
Shares outstanding	72,921
Options, RSUs	4,002
Warrants	5,525
Total shares, options, RSUs, warrants	82,448

Financials	September 30, 2020 ($'000s)
Cash burn YTD 3Q 2020	$20,334
Cash and ST Investments	$255,304

Notes: 1) As of September 30, 2020

Investment Highlights

- Focused on novel therapeutics for metabolic and endocrine diseases
  - Clinical programs demonstrate best-in-class efficacy data
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