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THERAPEUTICS

Corporate Presentation

November 2019

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 best-in-class drugs for metabolic and endocrine diseases

- Two Phase 2 programs supported by positive clinical data

Metabolic Disease Program: VK2809 for NASH

- Novel, selective thyroid receptor-β (TRβ) agonist
- Phase 2 results demonstrate significant reduction in liver fat content, lipids

Rare Disease Program: VK0214 for X-ALD

- Second selective thyroid receptor-β agonist
- In vivo data show improvement in key biomarkers

Musculoskeletal Program: VK5211 for hip fracture recovery

- Non-steroidal selective androgen receptor modulator (SARM)
- Phase 2 results demonstrate significant increases in lean body mass

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, 1H20
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

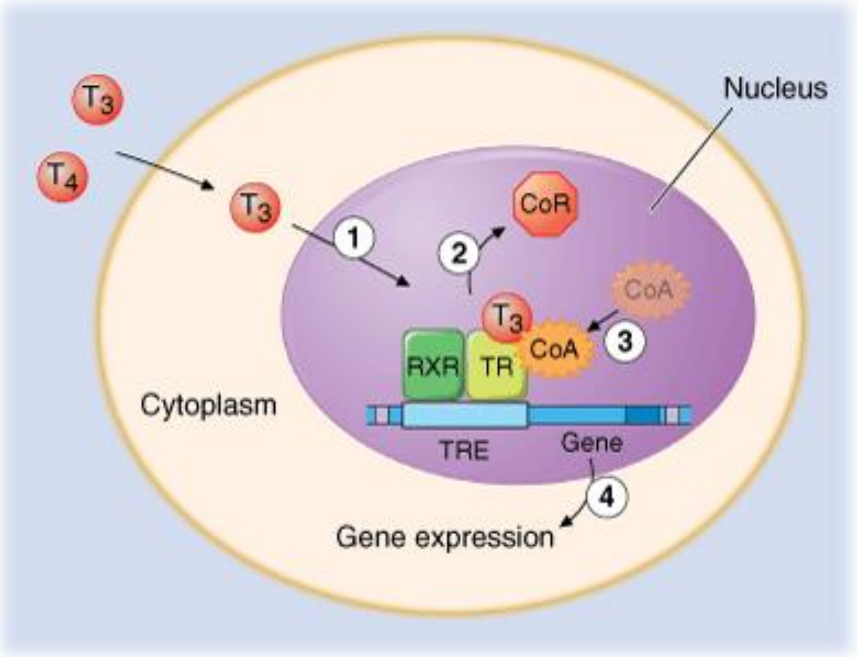
VK2809: Selective Thyroid Receptor-β Agonist

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: Successfully completed Phase 2 trial in NAFLD and hypercholesterolemia
  - VK0214: Preclinical, 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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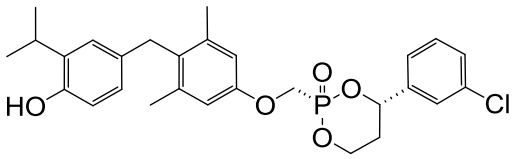
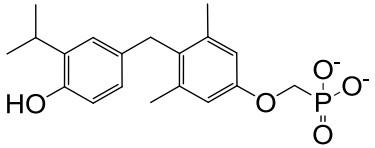
VK2809: Selective Liver-Targeted TRβ Agonist



VK2809, Novel Prodrug

Following oral dosing:

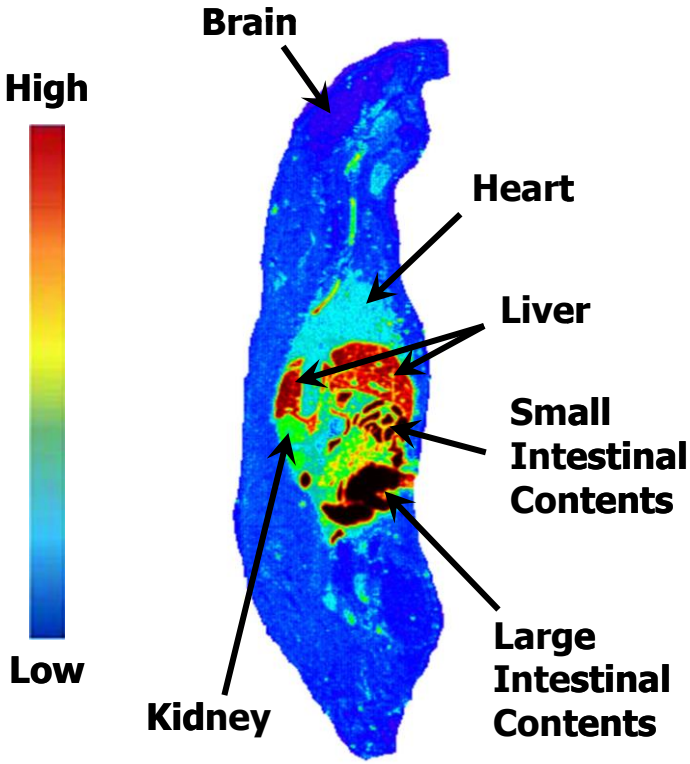
- Cyp3A4-mediated cleavage of prodrug
- 3A4 primarily expressed in liver
- Results in targeted delivery of drug to liver



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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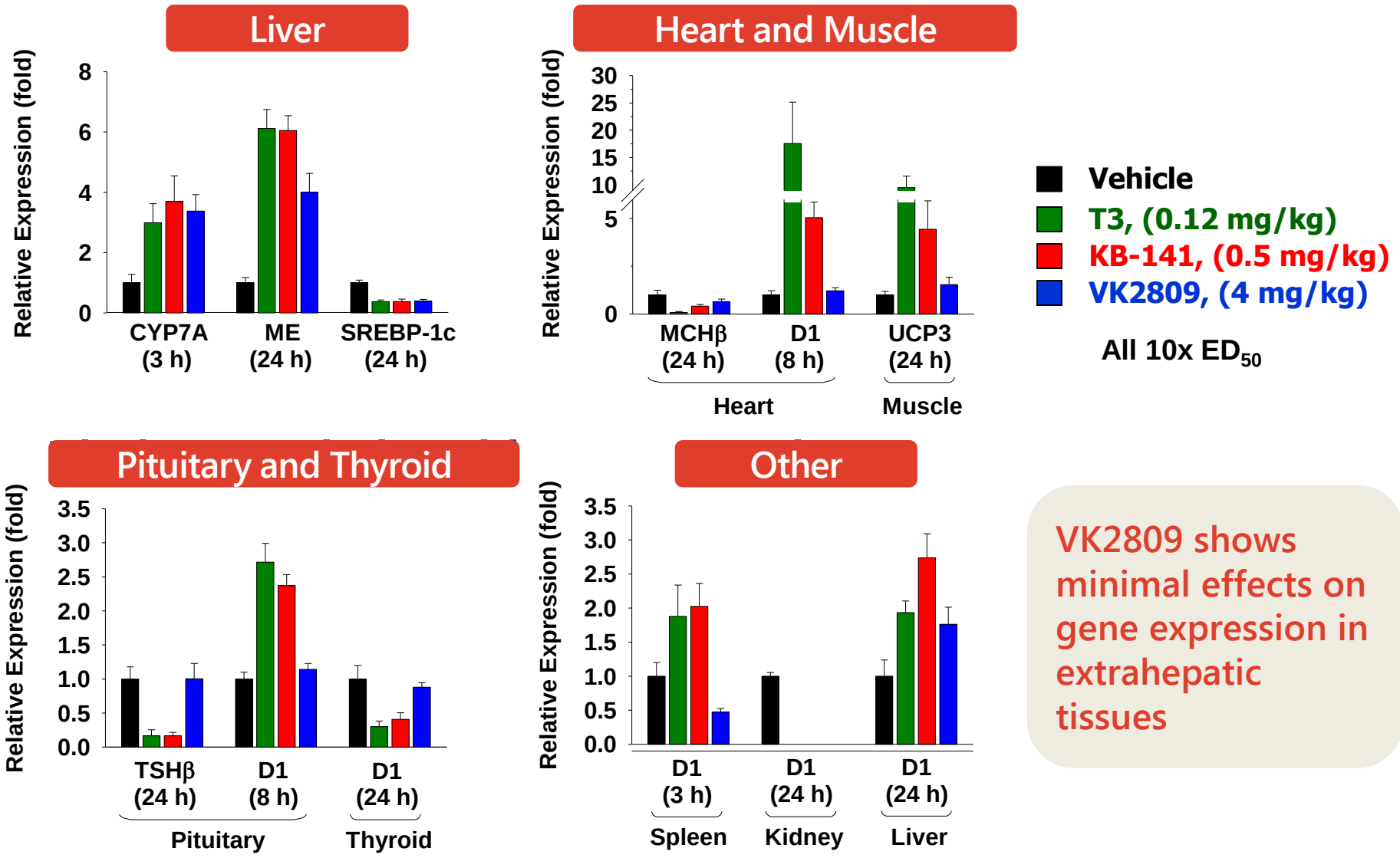
VK2809: Liver-Selective Transcriptional Effects



VK2809 shows minimal effects on gene expression in extrahepatic tissues

1) *Proc. Nat. Acad. Sci.*, 104(39), 15490-15495, (2007).

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Thyroid Receptor β Agonists for NAFLD and NASH

Development of NASH:

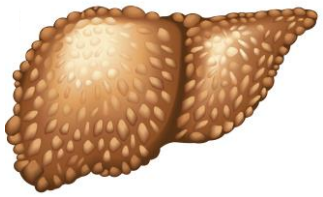
Healthy liver

Accumulation of fatty acids, triglycerides

Oxidative stress, inflammatory response

NASH: Steatosis, ballooning, hepatocyte damage

NAFLD Progression



- Cirrhosis
- HCC
- Liver failure

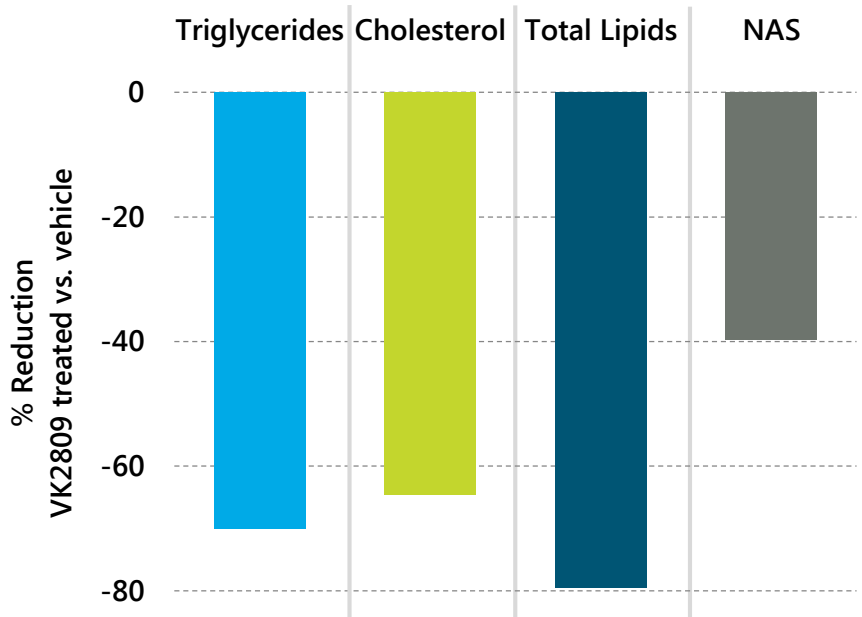
- β-Receptor: Key role in lipid metabolism; systemic and liver-specific effects
- Receptor localized to liver, limited ex-hepatic expression
- In vivo evidence suggests β-activation provides anti-fibrotic benefits
- Clinical data indicate a correlation between reduced liver fat, improvement in NAS

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

VK2809 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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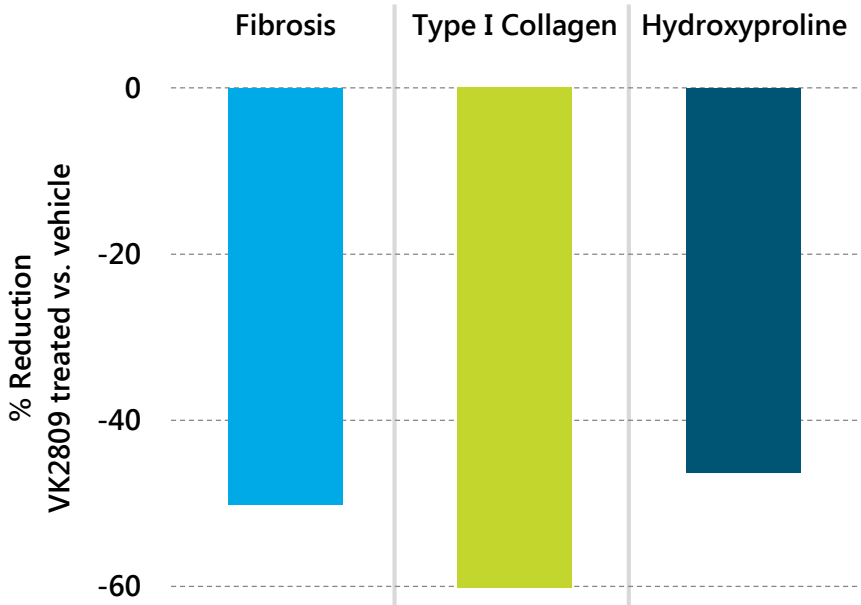
VK2809 Improves Fibrosis in Diet-Induced NASH Model

- 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



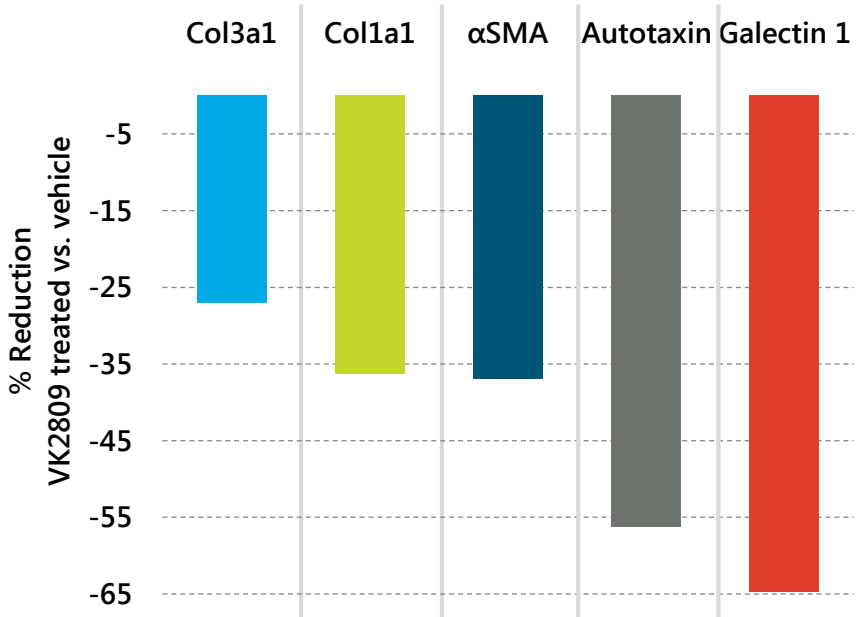
	Fibrosis	Type I Collagen	Hydroxyproline
% Difference:	-50.2%	-60.2%	-46.3%
p-value:	<0.01	<0.005	0.01

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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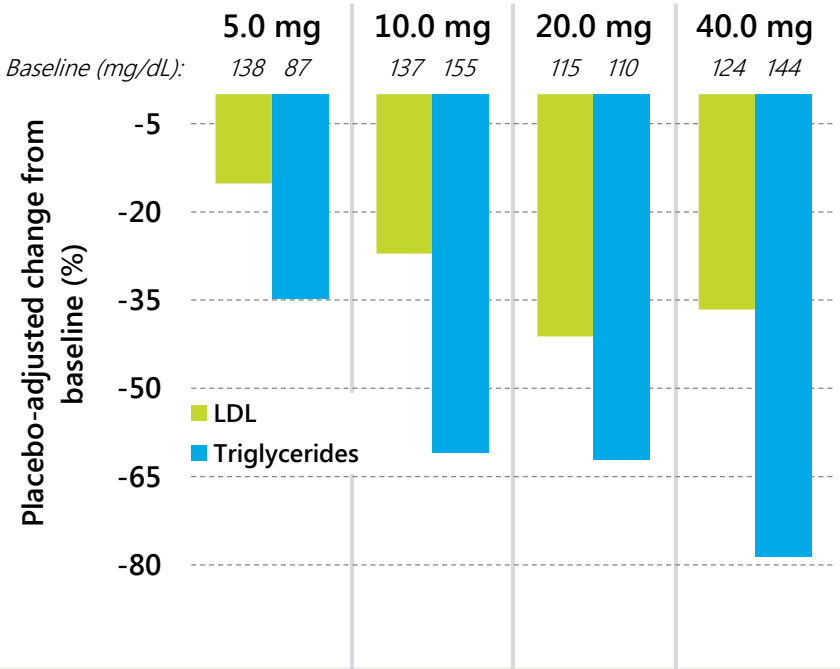
VK2809 Clinical Highlights: 14-Day Phase 1b Study

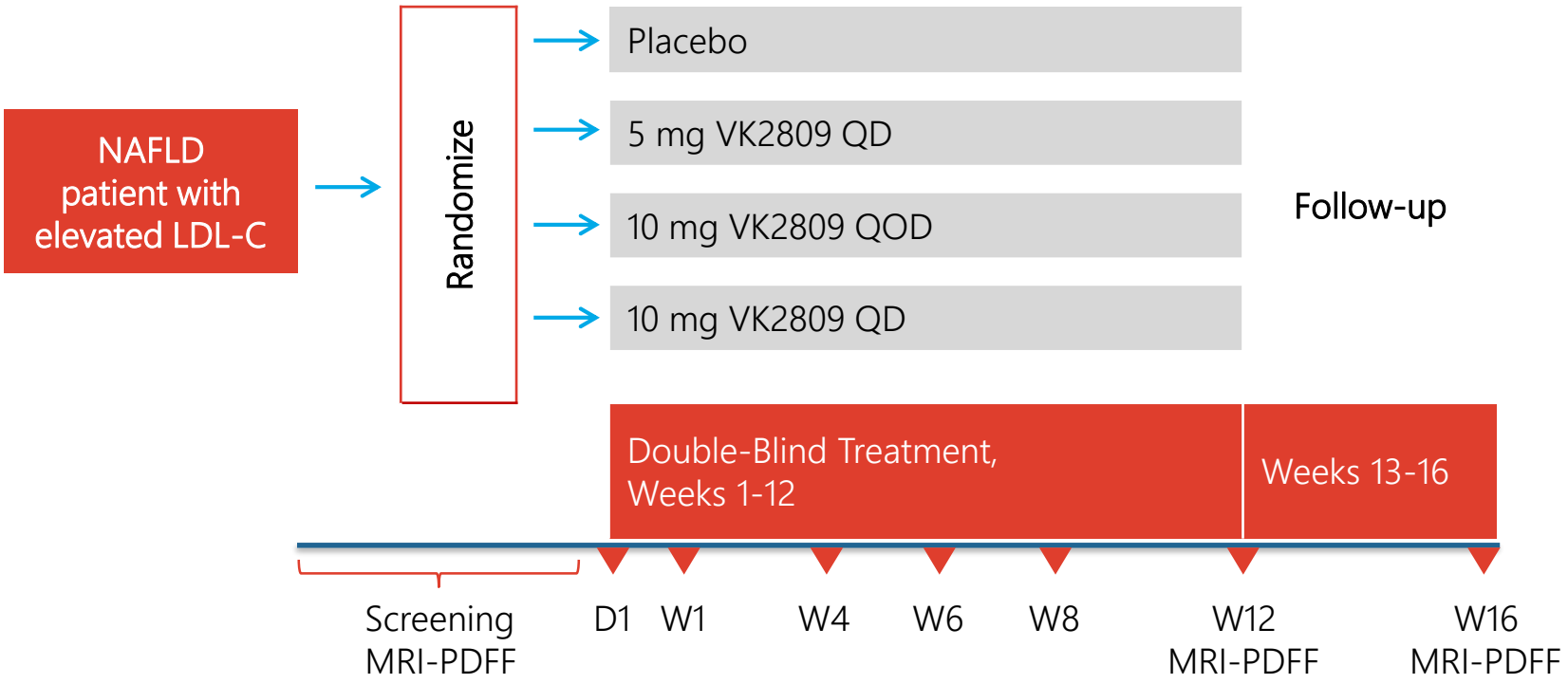
- Placebo-controlled trial (n=56), mild hypercholesterolemia
- Results: clinically, statistically significant reductions in LDL and triglycerides
- Encouraging safety and tolerability, no SAEs
- Results supported a proof-of-concept study in patients with NAFLD and elevated LDL-C

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 2 Study Design

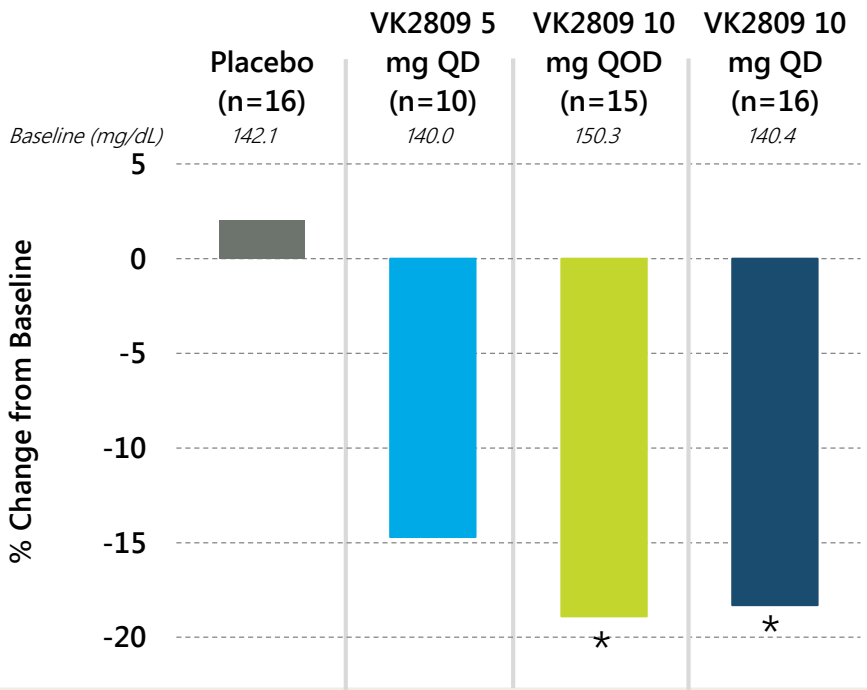


- Multi-arm, dose-ranging, 12 week Phase 2 trial
  - Target enrollment: 20 patients per arm
  - **Primary endpoint:** Change in LDL-C vs. placebo
  - **Secondary endpoint:** Change in liver fat by MRI-PDFF
  - **Exploratory endpoints:** Changes in atherogenic proteins

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

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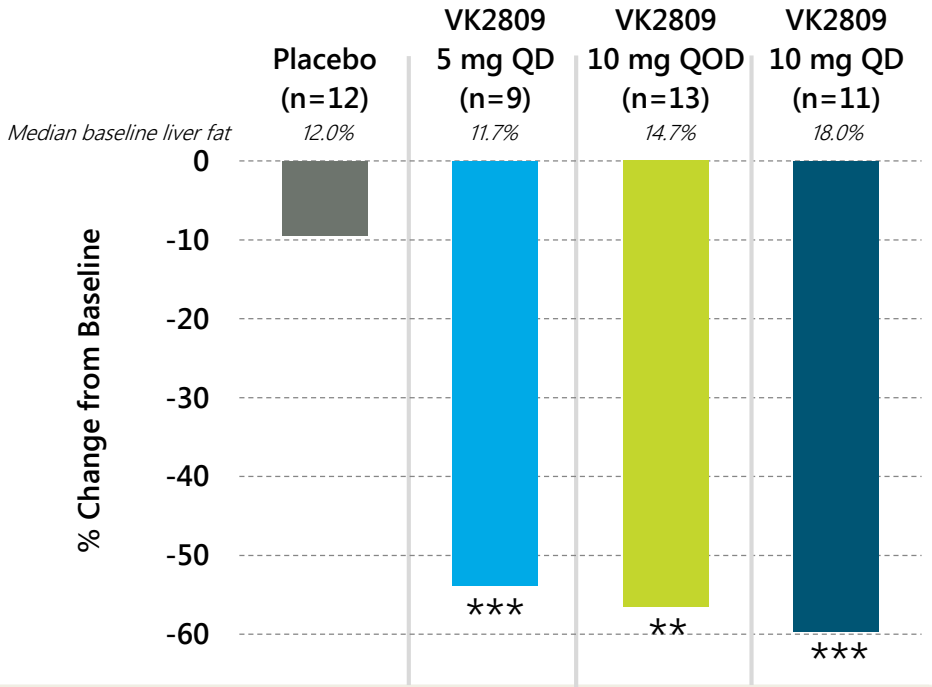
VK2809 Produced Significant Relative Reductions in Liver Fat

- 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



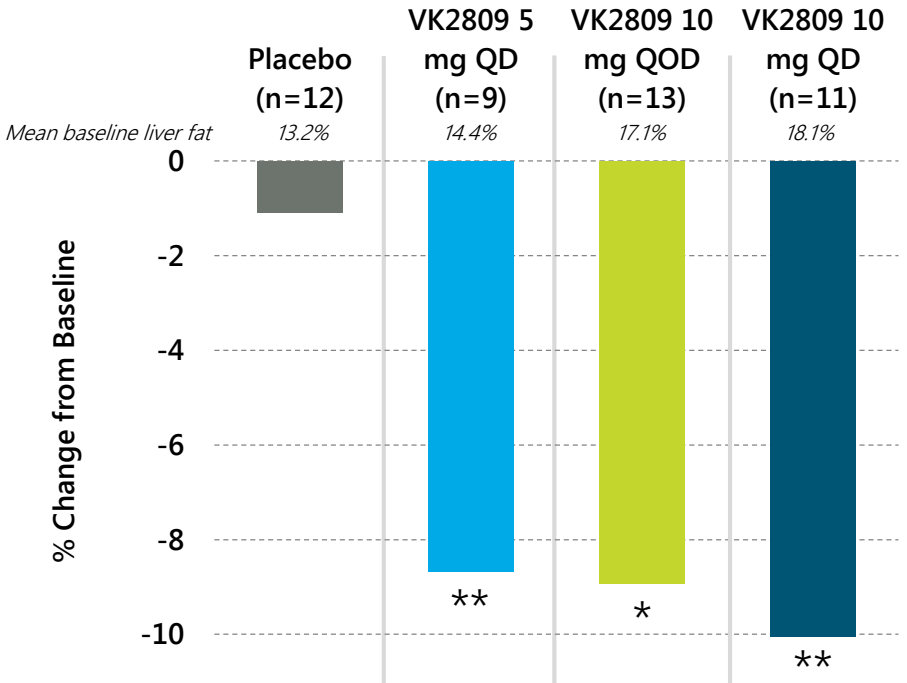
	Placebo (n=12)	VK2809 5 mg QD (n=9)	VK2809 10 mg QOD (n=13)	VK2809 10 mg QD (n=11)
% Change	-9.4%	-53.8%	-56.5%	-59.7%
p-value vs. placebo	-	0.0001	0.0018	0.0004

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

VK2809 Produced Significant Absolute Reductions in Liver Fat

- Significant Absolute Reductions from Baseline in Liver Fat by MRI-PDFF
- Maximal reductions at Week 12
  - Placebo: -6%
  - 5 mg QD: -18%
  - 10 mg QOD: -21%
  - 10 mg QD: -19%

Mean Absolute % Change in Liver Fat at 12 Weeks

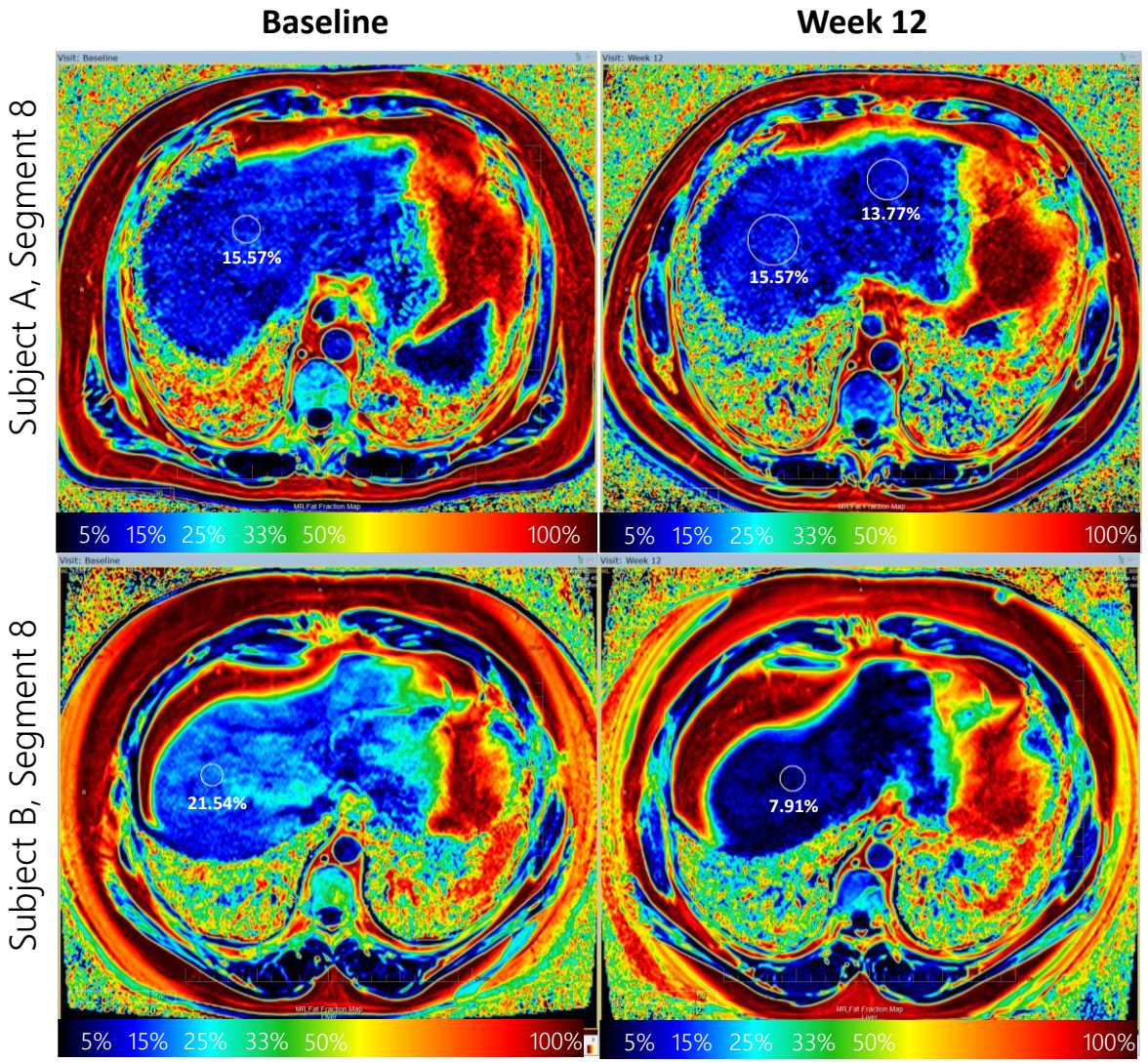


	Placebo (n=12)	VK2809 5 mg QD (n=9)	VK2809 10 mg QOD (n=13)	VK2809 10 mg QD (n=11)
% Change (SD)	-1.1% (2.8)	-8.7% (4.8)	-8.9% (6.2)	-10.6% (5.2)
p-value vs. placebo	-	0.0014	0.013	0.0030

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

Representative Fat Reduction, VK2809 and Placebo Subject

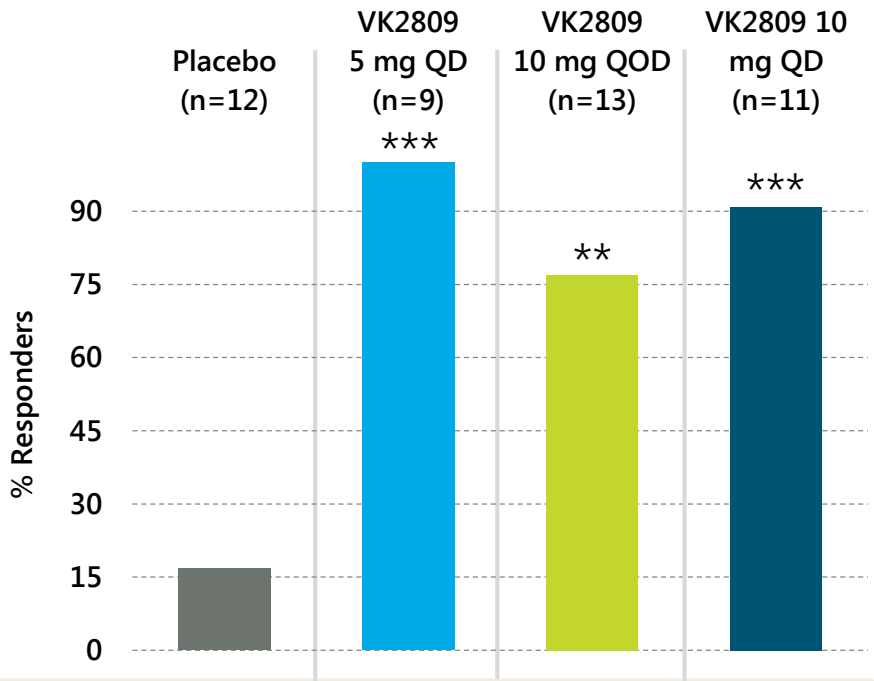
Overall Mean Hepatic Fat Values				
Subject, Dose	Baseline	Week 12	Absolute Change	Relative Change
Subject A Placebo	15.7%	15.6%	-0.04%	-0.3%
Subject B 10 mg QD	24.6%	6.0%	-18.6%	-75.6%



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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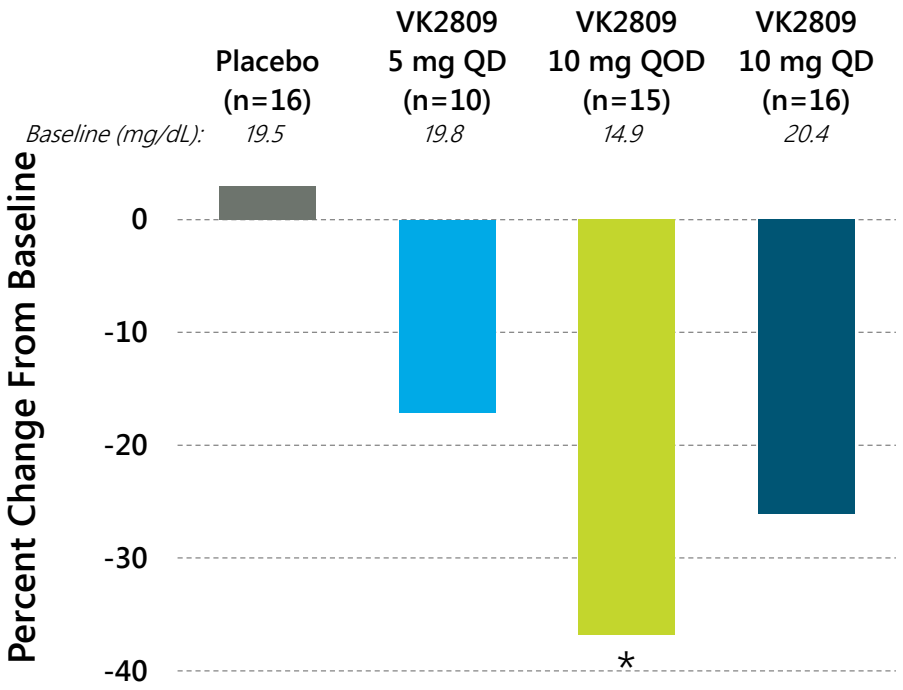
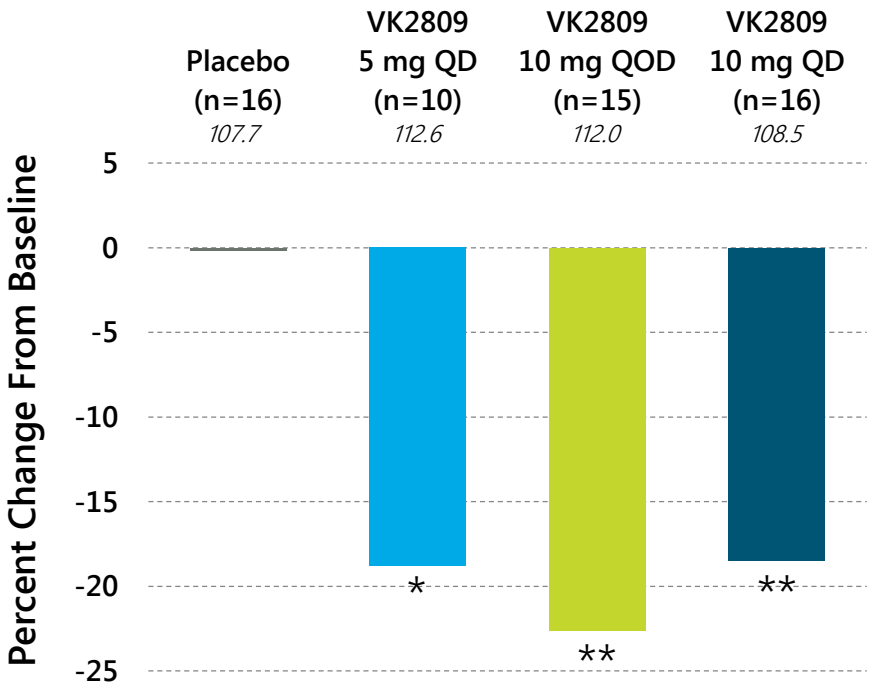
VK2809 Improved Atherogenic Protein Levels at 12 Weeks

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: Summary of Safety and Adverse Events

	Placebo	VK2809 5 mg QD	VK2809 10 mg QOD	VK2809 10 mg QD	All VK2809
Randomized (n)	17	10	16	16	42
Number (%) of subjects completing study	11 (65)	9 (90)	11 (69)	12 (75)	32 (76)
Number (%) of subjects with treatment-emergent AEs (TEAEs)	9 (53)	6 (60)	10 (63)	10 (63)	26 (62)
Number of Serious TEAEs	0	0	0	0	0
Number (%) of subjects discontinued due to AE	2 (12)	1 (10)	3 (19)	1 (6)	5 (12)
Number (%) of subjects with CV-related TEAEs	2 (12)	0	3 (19)	0	3 (7)
Number (%) of subjects with diarrhea or nausea	3 (18)	1 (10)	0	2 (13)	3 (7)

- Encouraging safety and tolerability, no dose-related trends
- No serious adverse events observed in any arm
- Excellent GI tolerability

VK2809-201: Safety Summary

- Mean ALT, AST levels in VK2809-treated subjects were 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 at Week 12
- No clinically meaningful changes among VK2809-treated patients relative to placebo-treated patients for:
  - Thyroid hormones (fT4, tT3, TSH)
  - Cardiovascular safety markers (troponin, CK-MB, NT proBNP)
  - Vital signs (systolic BP, diastolic BP, heart rate, weight)

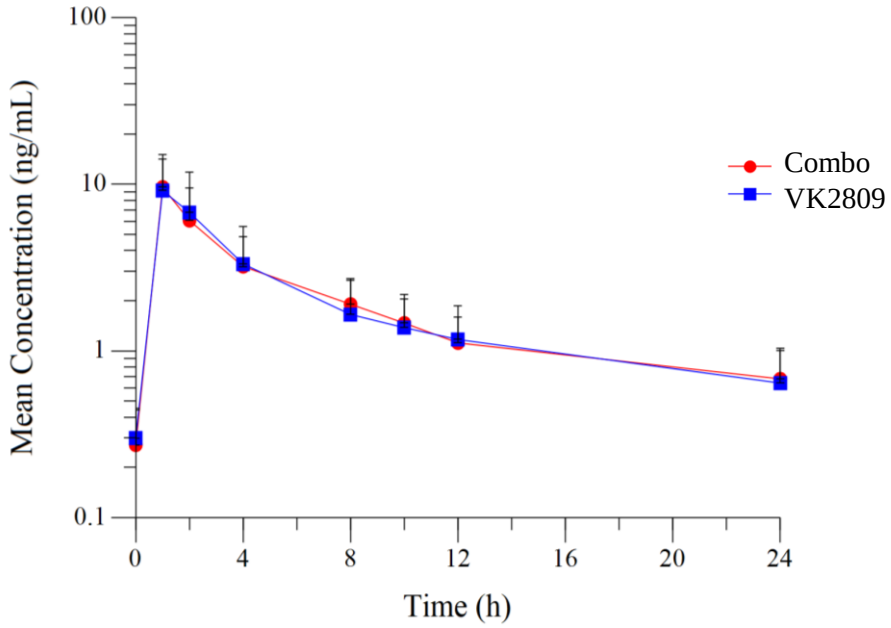
VK2809-201: Study Summary and Conclusions

- 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 at 12 weeks, including all patients receiving 5 mg daily doses
  - 70% of VK2809 treated patients experienced liver fat reductions ≥50%
- Week 16 results maintain statistically significant liver fat reduction despite no Tx for 4 weeks
- VK2809 produced significant reduction in LDL-C, triglycerides, Apo B, and Lp(a), suggesting potential long-term CV benefit
- VK2809 was safe in this 12-week Phase 2 study; No SAEs observed
- VK2809 was well-tolerated in this 12-week Phase 2 study
  - Diarrhea, nausea rates higher in placebo (18% vs. 7% for combined VK2809 cohorts)

VK2809-Atorvastatin DDI Study Shows Minimal Interaction

- Three-way crossover study in healthy volunteers
- No significant changes to PK profile of VK2809, VK2809A, atorvastatin, or atorvastatin metabolites
- Data confirm prior results in rabbits, dogs, primates
- Supports co-administration with statin therapies in future studies

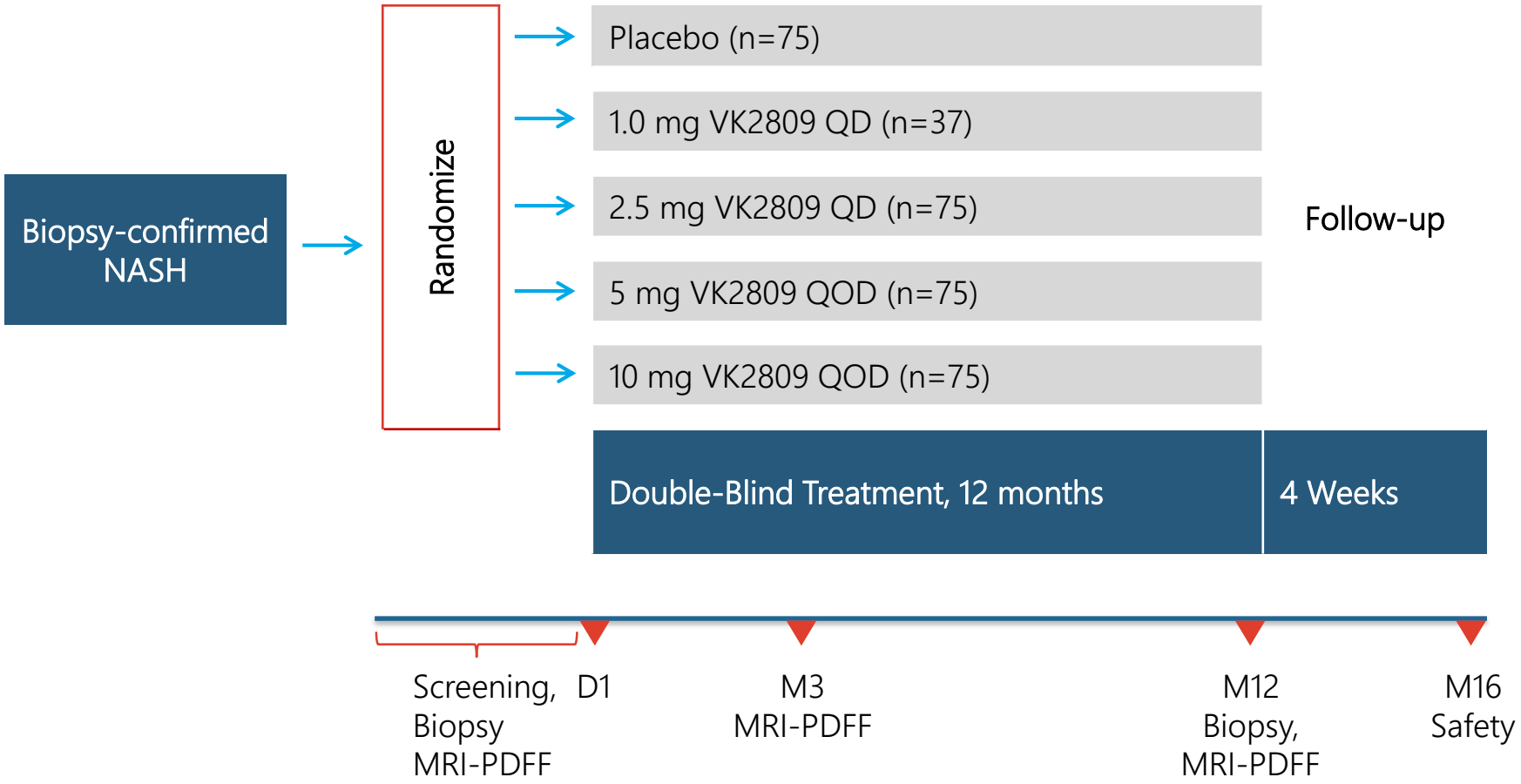
Mean VK2809 Concentration in the Presence and Absence of Atorvastatin



VK2809: Clinical Summary and Current Status

- Multiple Phase 1 studies demonstrated robust lipid-lowering effects
- 12-Week Phase 2 study demonstrated potent liver fat reduction
- No drug-drug interaction upon co-administration with atorvastatin
- IND filed with GI division in 4Q19
- VOYAGE 12-month Phase 2b NASH study underway

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.)

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Rare Disease Program

VK0214

X-Linked Adrenoleukodystrophy

VK0214 for X-ALD

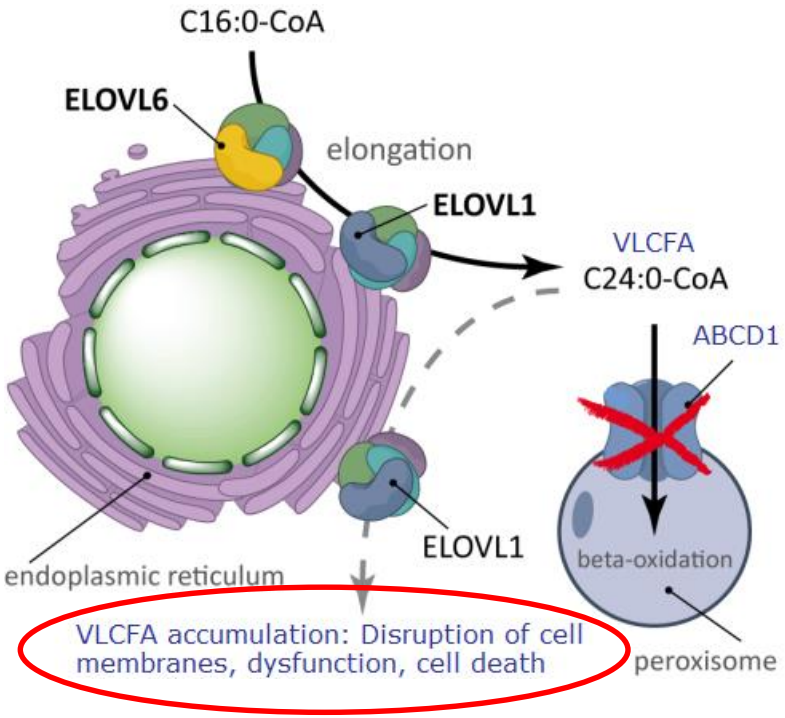
- X-Linked adrenoleukodystrophy (X-ALD)
  - Orphan neurodegenerative disorder
  - X-linked: Carried by females, primarily manifesting in males
  - No cure, no approved therapy
- Most severe form: Cerebral ALD
  - Rapidly progressive inflammatory demyelination; disruption of BBB
  - Affects ~35% before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

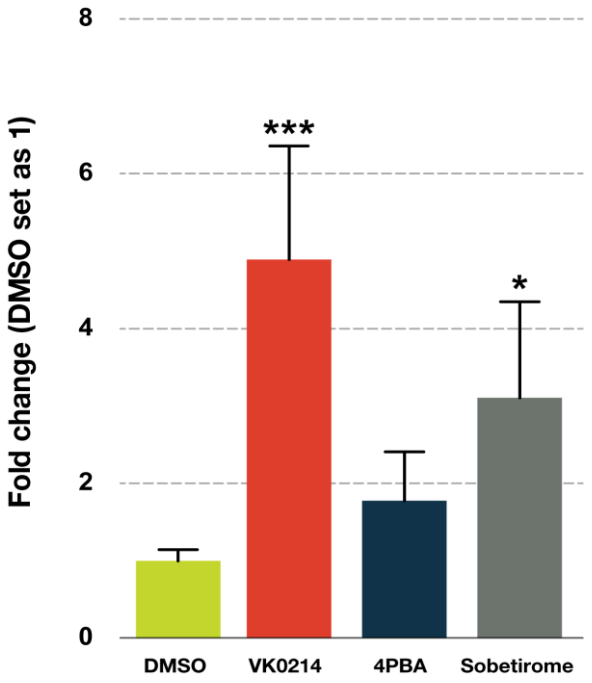
TRβ Agonists: Stimulate expression of compensatory transporters ABCD2, 3; may mitigate VLCFA elevation

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

Strong Rationale for TRβ Role in X-ALD

- Alternative VLCFA transporters ABCD2,-3 induced by TRβ receptor
  - Mechanistically compensate for ABCD1 deficiency
- Over-expression of ABCD2 corrects VLCFA elevation *in vitro* and *in vivo*
- *In vitro* POC established in X-ALD fibroblasts

VK0214: Potent ABCD2 induction in X-ALD fibroblasts



VK0214 successfully induces ABCD2 expression in human X-ALD cells

1. *Eur. J. Cell Biol.* **87**, 933-945, 2008
2. *J. Steroid Biochem. Mol. Biol.,* **116**, 37-43, 2009
3. Short call oral presentation 6, 84th American Thyroid Association, November 1, 2014.

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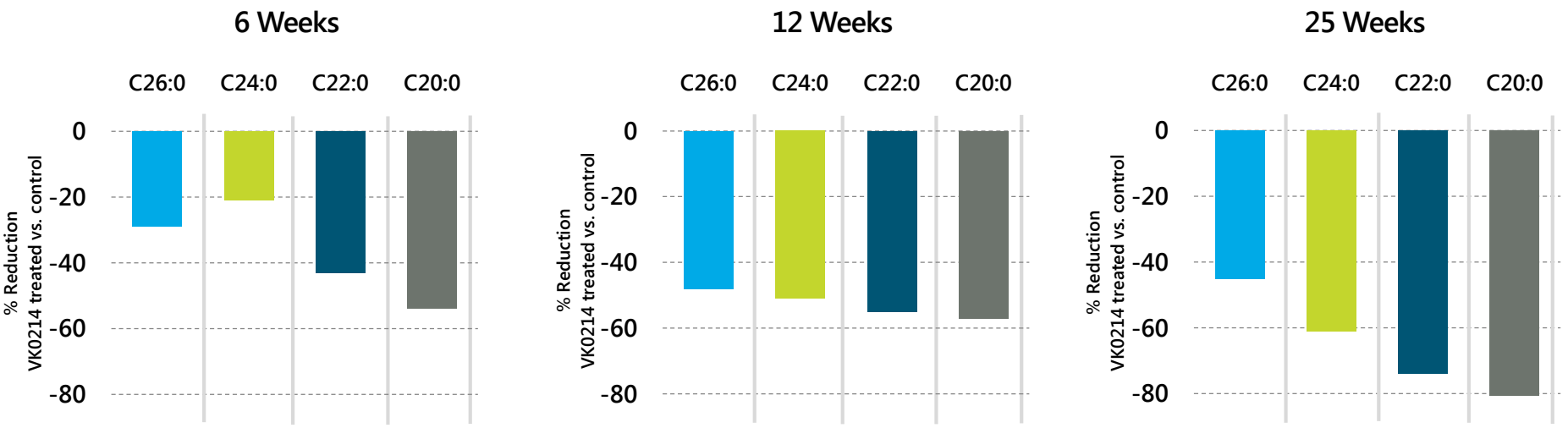
VK0214: *In Vivo* Proof-of-Concept Data, ABCD1 KO Mouse

- 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

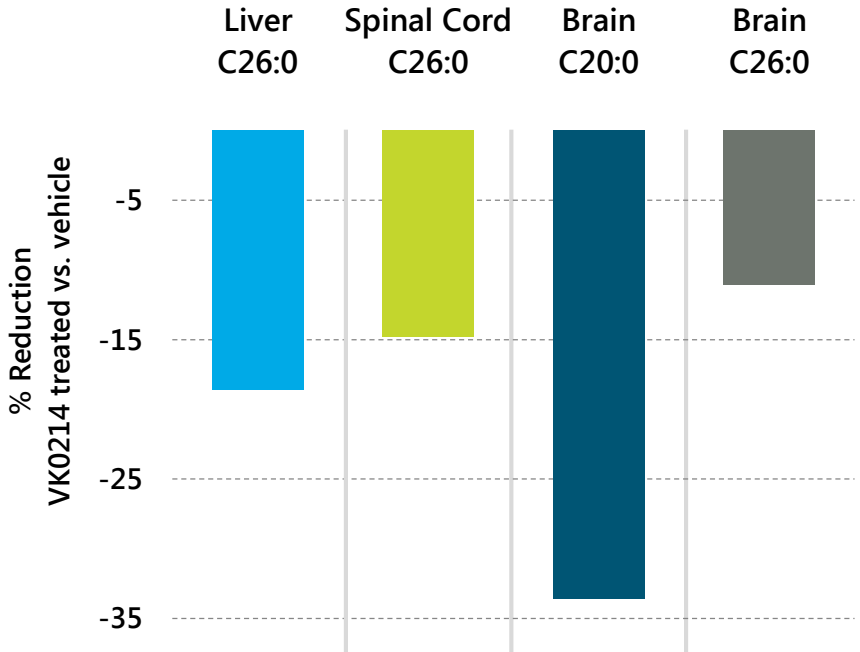


	C26:0	C24:0	C22:0	C20:0
% Chg:	-29%	-21%	-43%	-54%
p-value:	<0.0001	<0.005	<0.0001	<0.0001
% Chg:	-48%	-51%	-55%	-57%
p-value:	<0.0001	<0.0001	<0.0001	<0.0001
% Chg:	-45%	-61%	-74%	-82%
p-value:	<0.0001	<0.0001	<0.0001	<0.0001

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 1H20

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

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Financial Summary

- Capital structure and summary financials

Capital Structure[1]	In '000s
Shares outstanding	72,256
Options, RSUs	2,891
Warrants	5,979
Total shares, options, RSUs, warrants	81,126

Financials	Sept 30, 2019 ($'000s)
Cash burn 3Q YTD 2019	$13,009
Cash and ST Investments	$288,072

Notes: 1) As of September 30, 2019.

Investment Highlights

Focused on best-in-class drugs for metabolic and endocrine diseases

- Two Phase 2 programs supported by positive clinical data

Metabolic Disease Program: VK2809 for NASH

- Novel, selective thyroid receptor-β (TRβ) agonist
- Phase 2 results demonstrate significant reduction in liver fat content, lipids

Rare Disease Program: VK0214 for X-ALD

- Second selective thyroid receptor-β agonist
- In vivo data show improvement in key biomarkers

Musculoskeletal Program: VK5211 for hip fracture recovery

- Non-steroidal selective androgen receptor modulator (SARM)
- Phase 2 results demonstrate significant increases in lean body mass

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Musculoskeletal Program

VK5211: Selective Androgen Receptor Modulator

Hip Fracture

Musculoskeletal Program: VK5211 for Hip Fracture

- **Novel Selective Androgen Receptor Modulator (SARM), for hip fracture**
  - VK5211 is a potentially best-in-class small molecule SARM
- **Promising efficacy signals to date**
  - Significant improvements in lean body mass in animals and humans
- **Target indication: Rehabilitation post-hip fracture**
  - Population rapidly loses muscle, bone, function
  - >300,000 patients per year, U.S.; expect continued growth
  - Market opportunity potentially exceeds $1B annually
- **Potential benefits in other fractures, joint replacements, and muscle wasting disorders**

VK5211: Potential Therapeutic Benefits of SARMs

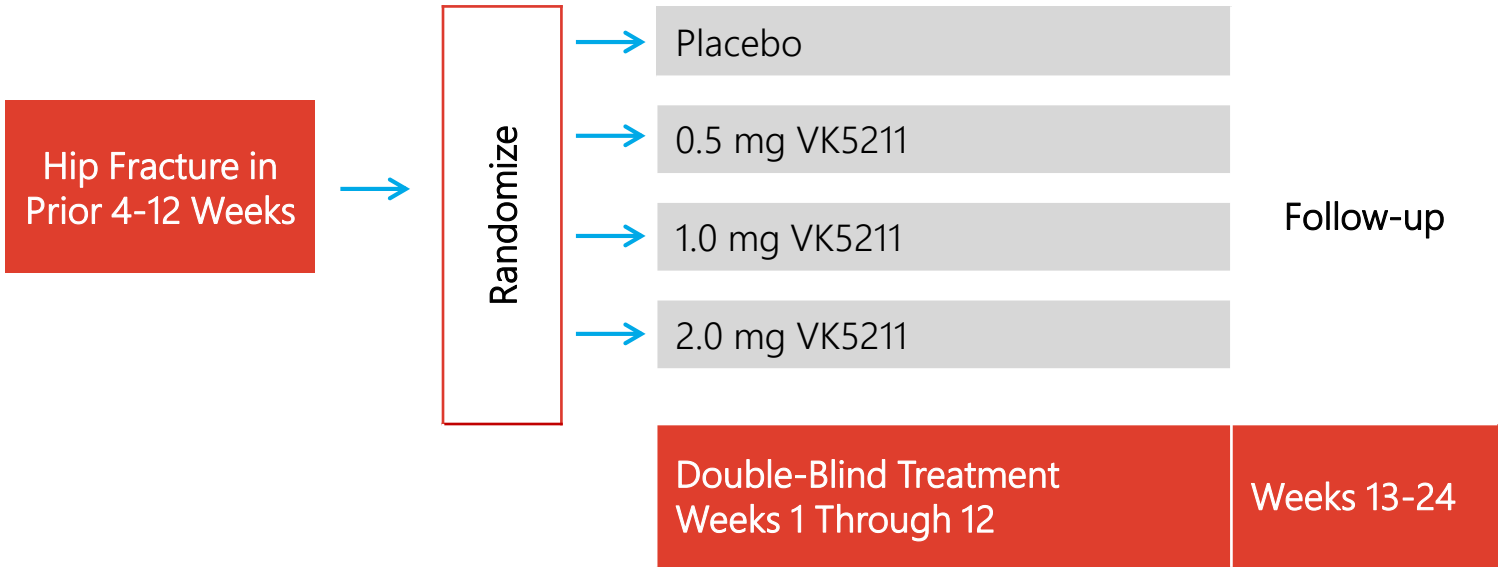
- ↑ Muscle Mass
- ↑ Muscle Strength

- ↑ Bone Formation
- ↑ Bone Strength
- ↓ Bone Resorption

- ↑ Cognition
- ↑ Libido
- ↑ Energy

Goal: Retain beneficial properties of natural androgens with fewer side effects relative to anabolic steroids

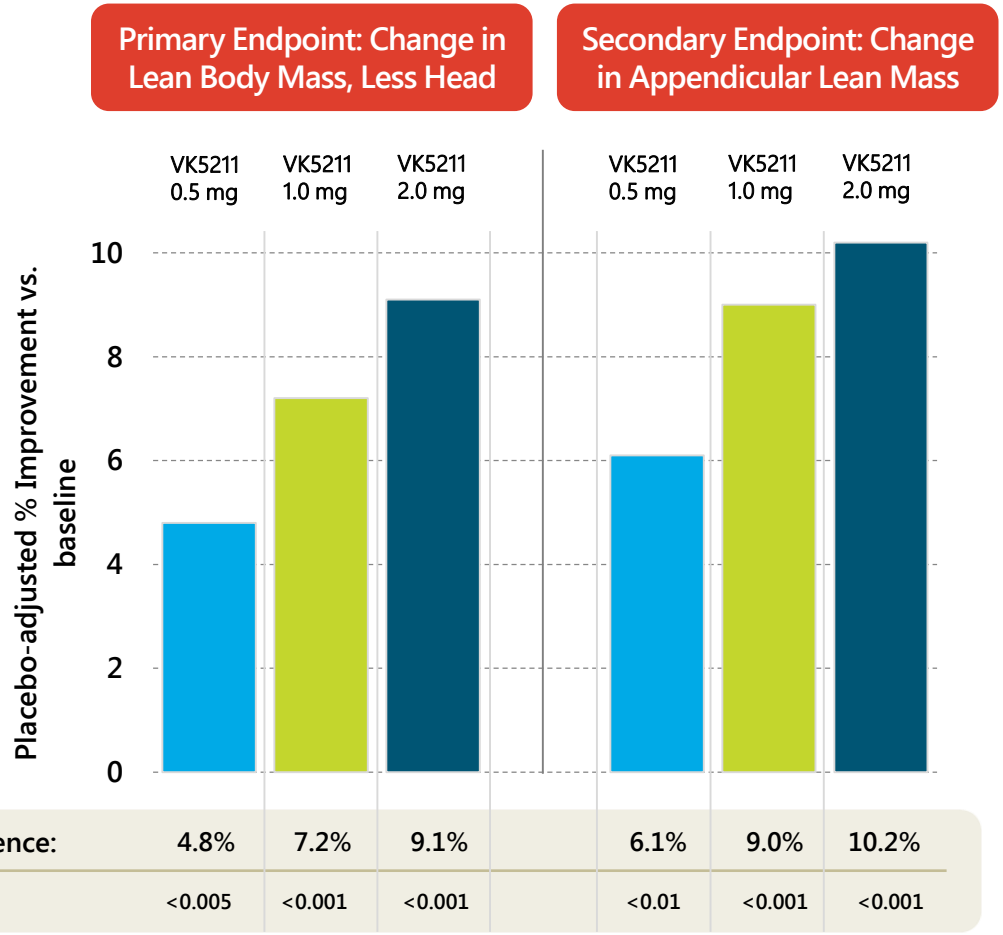
VK5211: Phase 2 Trial, Hip Fracture



- **12-week Phase 2 trial in 108 patients**
  - Once-daily oral dosing
  - Primary endpoint: Change in lean body mass
  - Secondary and exploratory endpoints: Change in appendicular lean mass, total lean body mass, BMD, functional status, ADL, QOL

Phase 2 Results Show Significant Increases in Lean Body Mass

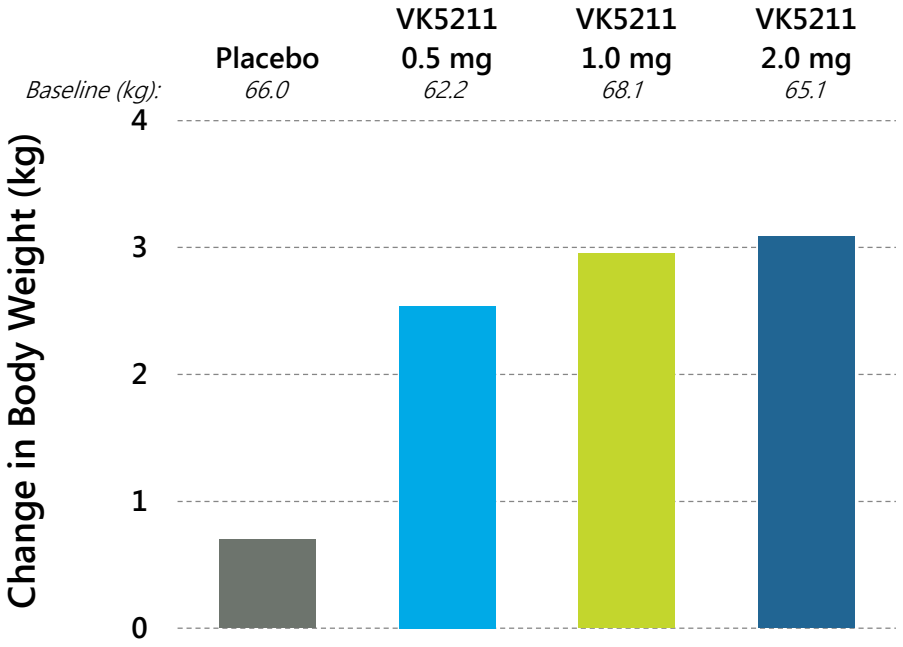
- Significant increases in LBM and appendicular lean mass following 12 weeks of daily dosing
- Consistent dose response observed across primary, secondary efficacy measures
- Encouraging safety and tolerability, no drug-related SAEs



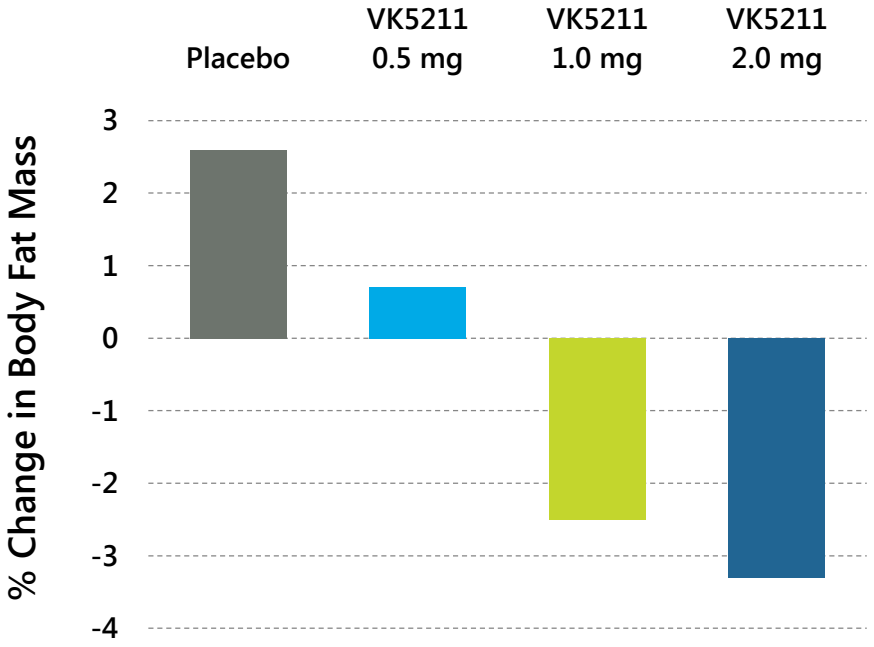
VK5211 produced robust dose-dependent effects on primary and all secondary measures of lean body mass

VK5211-201: Change in Body Composition at 12 Weeks

Change in Mean Body Weight, at Week 12



Change in Mean Fat Mass, at Week 12



Change	0.70 kg	2.54 kg	2.95 kg	3.09 kg	2.9%	0.7%	-2.5%	-3.3%
p-value	-	NS	NS	NS	-	0.24	0.11	0.01

- Dose-dependent increase in mean body weight
- Dose-dependent decrease in mean fat mass; significant at high dose

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VK5211 Phase 2 Study: Overall Safety and Adverse Events

- Encouraging safety and tolerability
- Frequency of reported adverse events demonstrates no dose-relationship
- No drug-related SAEs
- Next steps
  - Exploring collaboration and licensing opportunities
  - Plan for next steps with partner

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Corporate Presentation