VIKING THERAPEUTICS

Corporate Presentation

April 2023

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.

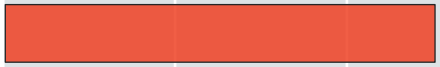
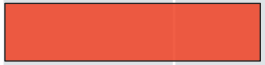
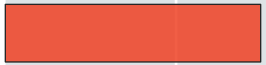
The forward-looking statements included in this presentation represent our views as of the date of this presentation. We anticipate that subsequent events and developments will cause our views to change. While we may elect to update these forward-looking statements in the future, we specifically disclaim 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

- Developing novel therapeutics for metabolic and endocrine diseases
  - Multiple clinical programs demonstrate best-in-class efficacy data
- Metabolic Disease Programs
  - VK2809: Novel, selective thyroid receptor-β agonist for NASH and lipid disorders
    - Phase 2b VOYAGE trial in biopsy-confirmed NASH ongoing
  - VK2735: Novel GLP-1/GIP dual agonist for obesity
    - Phase 2 obesity study planned for mid-2023
  - VK2735 Oral: GLP-1/GIP dual agonist for metabolic disorders
    - Phase 1 study ongoing
- Rare Disease Program
  - VK0214: Novel, selective thyroid receptor-β agonist for X-ALD
    - Phase 1b in patients ongoing

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
VK2735 (Dual GLP-1/GIP agonist)	*Obesity*					Phase 2 planned for mid-2023
VK2735 Oral (Dual GLP-1/GIP agonist)	*Metabolic disorders*					Phase 1 ongoing
VK0214 (TRβ agonist)	*X-ALD*					Phase 1b ongoing

Potential data events expected over next 12-18 months

- VK2809: Phase 2b data, 12-week MRI-PDFF and biopsy results in NASH
- VK2735: Phase 2 data, obesity
- VK2735 Oral: Phase 1 data in healthy subjects
- VK0214: Topline data, Phase 1b study in X-linked adrenoleukodystrophy

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VK2809: Selective Thyroid Receptor-β Agonist

NASH

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
  - Large markets: NASH, hypercholesterolemia, dyslipidemia
  - Rare diseases: X-linked adrenoleukodystrophy (X-ALD), other
- Lead program VK2809
  - Oral formulation
  - Shown to be safe and well-tolerated in 8 completed clinical studies
  - Phase 2b VOYAGE trial ongoing

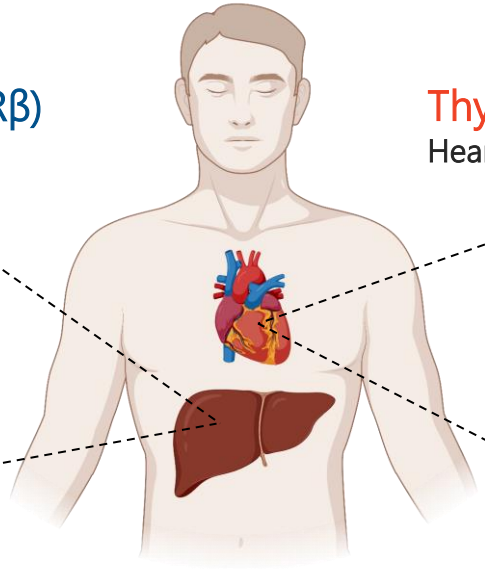
Thyroid Hormone Receptor Overview

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



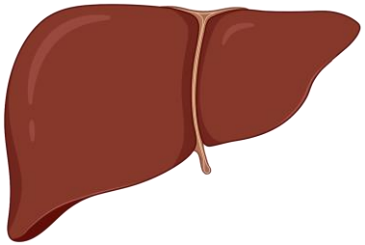
Thyroid hormone receptor alpha (TRα)
Heart, skeletal muscle

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

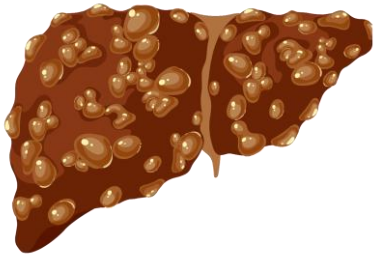


NASH Progression

Accumulation of fatty acids, triglycerides; NAFLD

Oxidative stress, inflammatory response

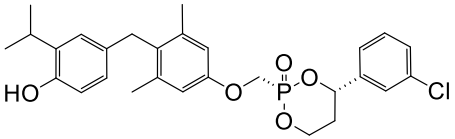
NASH: Steatosis, ballooning, hepatocyte damage



- 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

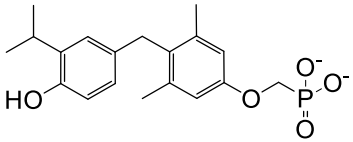
VK2809: Unique Liver-Targeted Characteristics



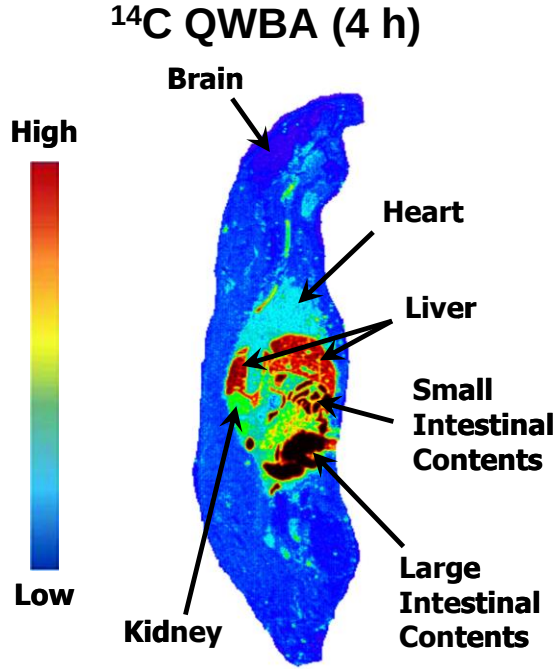
VK2809, Novel Prodrug

Following oral dosing:

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

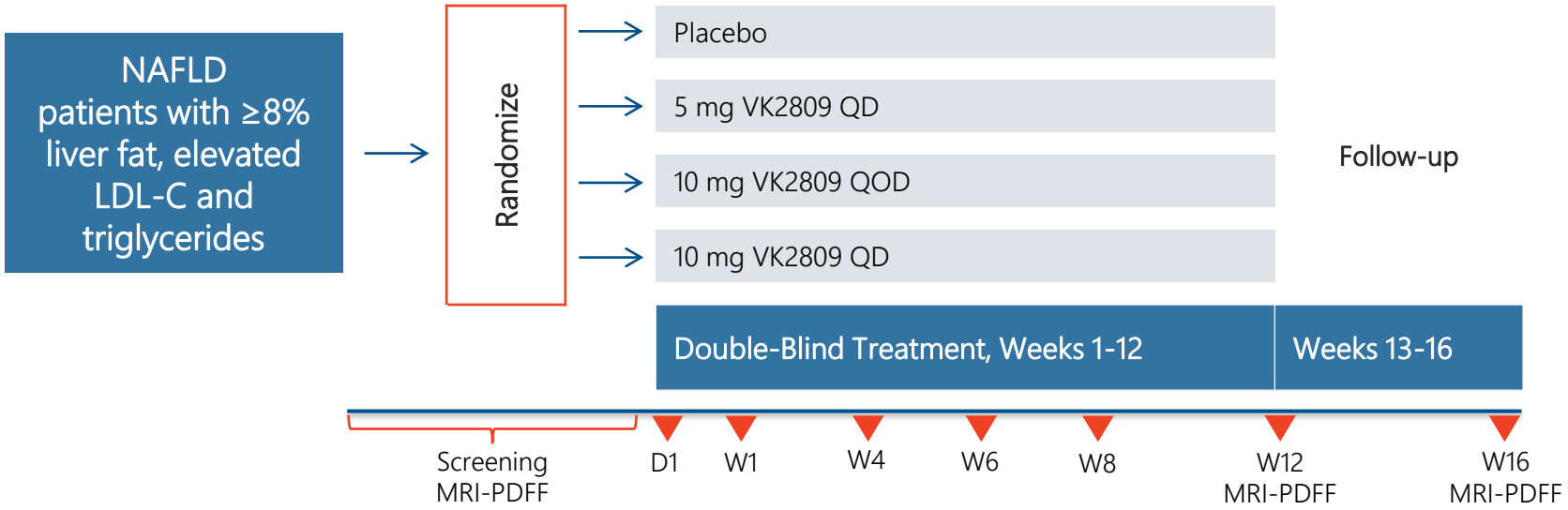


VK2809A, Potent TRβ Agonist, 2.2 nM Ki



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

VK2809-201: Phase 2a Study Design

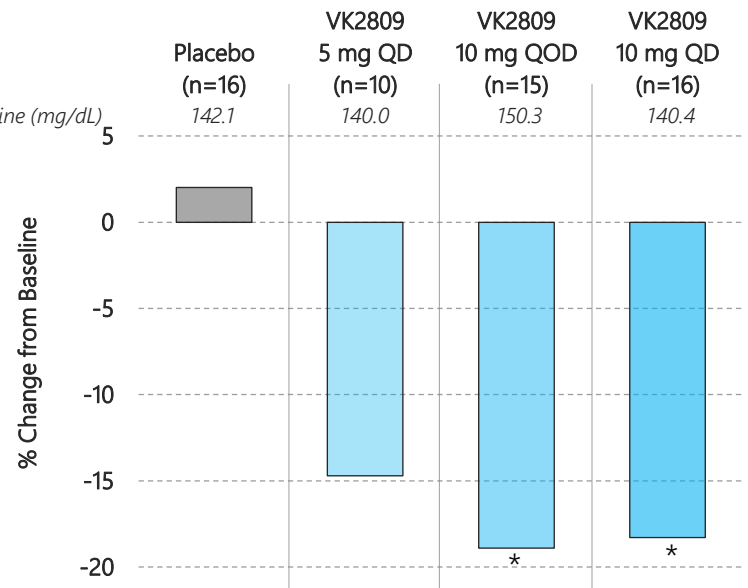


- Multi-arm, dose-ranging, 12 week Phase 2a trial
  - **Primary endpoint:** Change in LDL-C vs. placebo
  - **Secondary endpoint:** Change in liver fat by MRI-PDFF
  - **Exploratory endpoints:** Changes in atherogenic proteins

VK2809 Significantly Reduced LDL-C at 12 Weeks

- Statistically significant reductions in all VK2809 cohorts 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

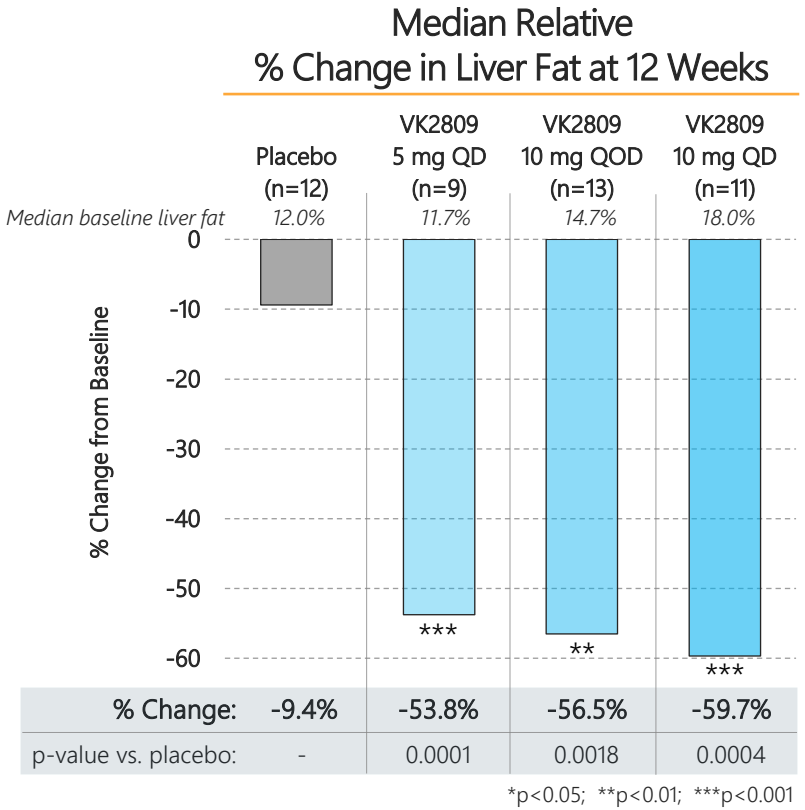


	Placebo (n=16)	VK2809 5 mg QD (n=10)	VK2809 10 mg QOD (n=15)	VK2809 10 mg QD (n=16)
% Change:	2.0%	-14.7%	-18.9%	-18.3%
p-value vs. placebo:	-	0.080	0.034	0.025

* p<0.05

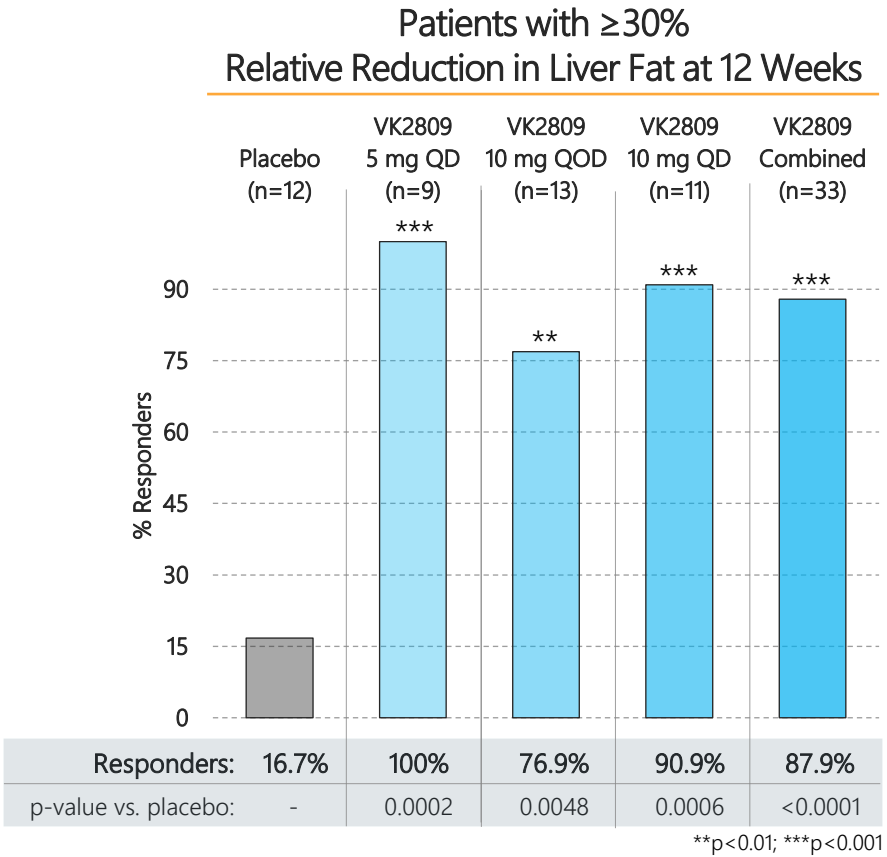
VK2809 Produced Significant Relative Reductions in Liver Fat

- 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).*

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

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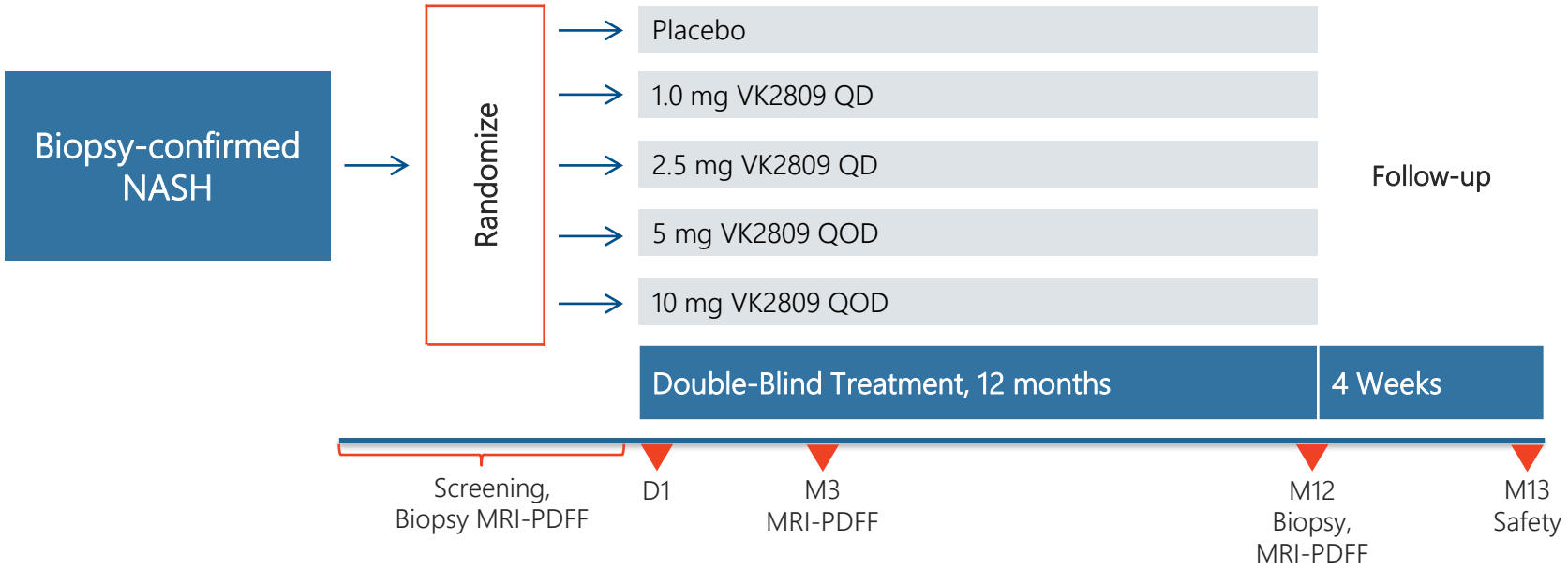
VK2809: Phase 2b VOYAGE Study

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 endpoint:** Change in histology at 12 months (NAS, fibrosis markers, etc.)

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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VK2735: Dual GLP-1/GIP Receptor Agonist

Metabolic Disorders

GLP-1/GIP Dual Agonists for Metabolic Disorders

- Peptides secreted by intestines after meals
- Complementary tissue distribution and activities
- Stimulate insulin production, induce satiety
- Therapeutic benefits in obesity, NASH, diabetes

GLP-1/GIP Receptor Co-Activation and Downstream Effects

Glucagon-Like Peptide 1 Receptor | **Glucose-Dependent Insulinotropic Polypeptide Receptor**

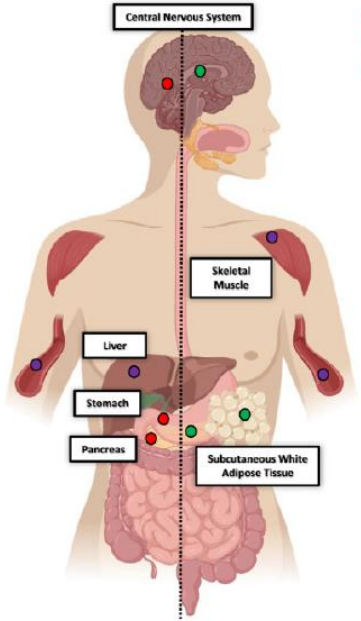
Central Nervous System
- ↑ Satiety
- ↓ Food Intake
- ↑ Nausea
- ↓ Body Weight

Pancreas
- ↑ Insulin
- ↓ Glucagon

Stomach
- ↓ Gastric Emptying

Systemic
- ↓ Hyperglycemia

Liver
- ↑ Insulin Sensitivity
- ↓ Hepatic Glucose Production
- ↓ Ectopic Lipid Accumulation



Central Nervous System
- ↓ Food Intake
- ↓ Nausea
- ↓ Body Weight

Pancreas
- ↑ Insulin
- ↑ Glucagon

Subcutaneous White Adipose Tissue
- ↑ Insulin Sensitivity
- ↑ Lipid Buffering Capacity
- ↑ Blood Flow
- ↑ Storage Capacity
- ↓ Proinflammatory Immune Cell Infiltration

Systemic
- ↓ Hyperglycemia
- ↓ Dietary Triglyceride

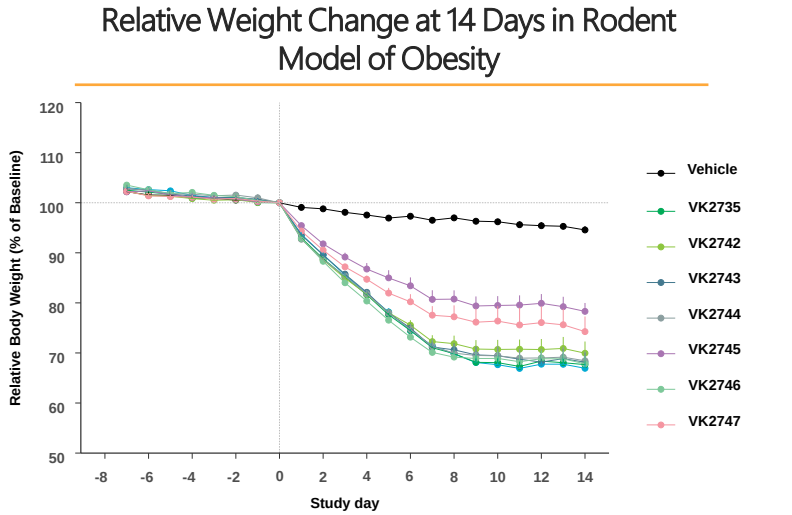
Skeletal Muscle
- ↑ Insulin Sensitivity
- ↑ Metabolic Flexibility
- ↓ Ectopic Lipid Accumulation

GLP-1 Receptor Activity
GIP Receptor Activity
Indirect Activities

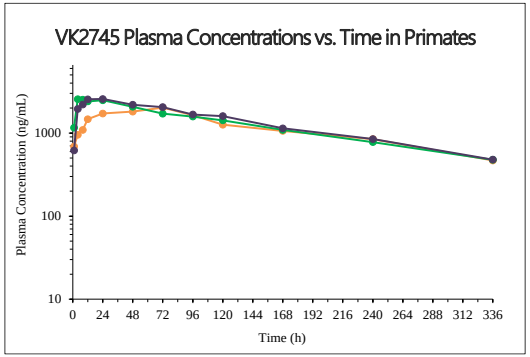
Graphics: Trends in Endocrinology and Metabolism 2020, 31(6), 410-421.

Novel Peptide-Based Dual Agonists

- Potent binding (<500nM) observed at human GLP-1 and GIP receptors
- Variable GIP activity
- Robust weight loss observed in rodent models
- Predictable PK; $T_{1/2}$ 2 – 7 days in primates; variable exposures
- VK2735 selected for further development

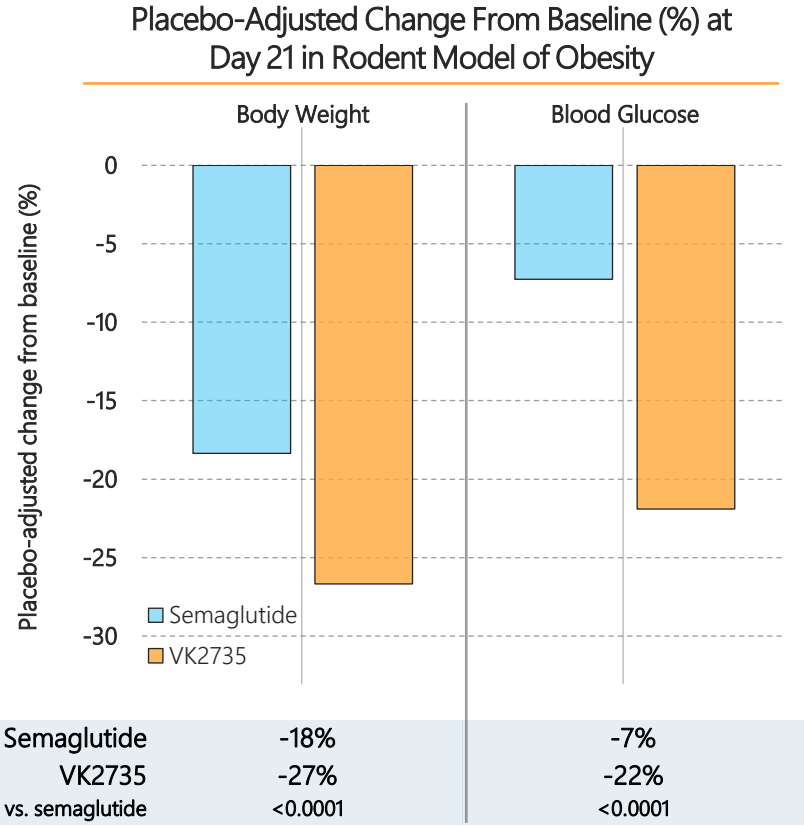


Representative PK profile: Generally well-behaved with extended $T_{1/2}$



VK2735: Novel Dual Agonist of GLP-1 and GIP Receptors

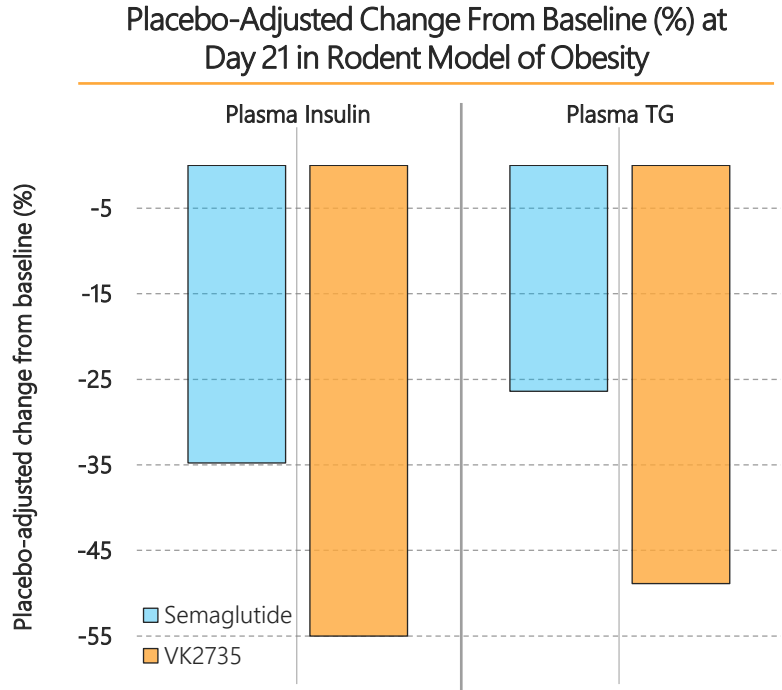
- GLP-1 receptor activity similar to known agonist semaglutide (<300nM)
- GLP-1/GIP binding ratio <1
- Robust reduction in body weight, blood glucose in rodent models
- Data support additive benefit of GIP-agonist activity on top of GLP-1 activation



Semaglutide	-18%	-7%
VK2735	-27%	-22%
p-value vs. semaglutide	<0.0001	<0.0001

VK2735 Glucose, Lipid Effects Exceed GLP-1 Mono-Agonists

- VK2735 demonstrates broad improvements vs. GLP-1 mono-agonist at same dose level
- Improved insulin sensitivity, plasma lipids after 21 days of dosing in rodent model
- Supports additive GIP-mediated insulin secretion on top of GLP-1 effect



Semaglutide	-35%	-26%
VK2735	-55%	-49%
p-value vs. semaglutide	0.1499	0.0034

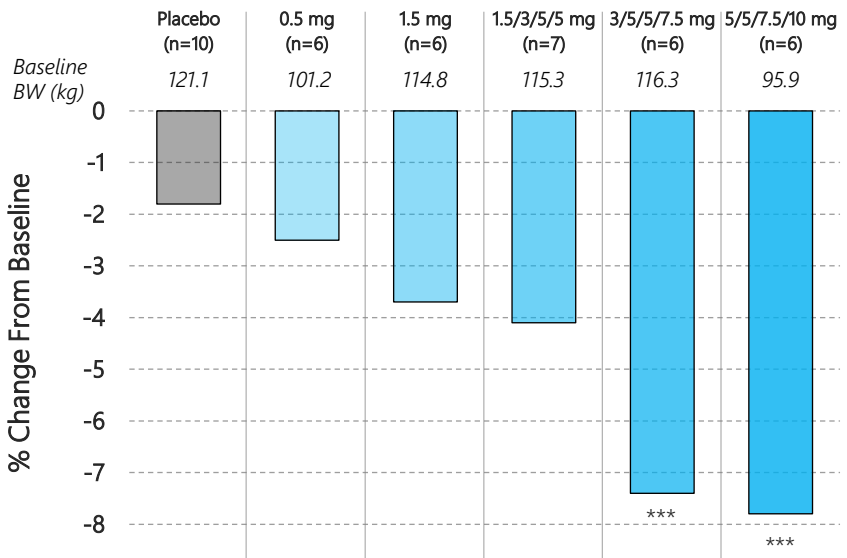
VK2735 Phase 1 Clinical Study Design

- Randomized, placebo-controlled, stacked SAD/MAD study design
- SAD portion: Escalating single doses in healthy subjects
- MAD study: 28 Days of weekly dosing in healthy subjects with BMI ≥30 kg/m^2
- Primary objectives: Safety, tolerability
- Exploratory: Changes in body weight, glucose, liver fat

VK2735 Phase 1 MAD Results: Weight Change After 28 Days

- Reduction in body weight observed in all VK2735 dosing cohorts
- Dose dependent effect observed across VK2735 cohorts
- Significant reduction vs. placebo observed at higher VK2735 doses

Mean % Change in Body Weight at Day 29



% Change in BW:	-1.8%	-2.5%	-3.7%	-4.1%	-7.4%	-7.8%
Placebo-adjusted:	-	-0.7%	-1.9%	-2.3%	-5.6%	-6.0%
p-value vs placebo	-	0.64	0.17	0.09	0.0003	0.0002

Notes: Baseline BMI ≥30 in all MAD subjects. ***p<0.001

VK2735 Phase 1 MAD Study: GI Tolerability Summary

Most common AEs to date Number of subjects reporting (%)	Placebo (n=10)	0.5 mg (n=6)	1.5 mg (n=6)	1.5/3/5/5 mg (n=7)	3/5/5/7.5 mg (n=6)	5/5/7.5/10 mg (n=6)
GERD	1 (10%)	0 (0%)	0 (0%)	3 (43%)	4 (67%)	1 (17%)
Nausea	5 (50%)	2 (33%)	4 (67%)	5 (71%)	5 (83%)	2 (33%)
Vomiting	1 (10%)	2 (33%)	0 (0%)	2 (29%)	1 (17%)	1 (17%)
Abdominal pain	1 (10%)	0 (0%)	1 (17%)	3 (43%)	4 (67%)	2 (33%)
Diarrhea	3 (30%)	1 (17%)	1 (17%)	2 (29%)	0 (0%)	0 (0%)
Constipation	0 (0%)	1 (17%)	1 (17%)	2 (29%)	1 (17%)	0 (0%)

GERD: Gastroesophageal reflux disease.

- Majority of all reported AEs (98%) mild or moderate
- Mechanism-based mild (89%) to moderate (11%) nausea observed
- No discontinuations related to GI adverse events

VK2735 Phase 1 Study Takeaways

- Encouraging early profile observed in healthy subjects with BMI ≥30
- Dose-dependent improvement in weight loss of up to 7.8% (6.0% placebo-adjusted) reported after 28 days
- PK data suggest excellent exposures from weekly dosing regimen
- Promising safety and tolerability, 98% of AEs mild to moderate
- Expected observation of mild to moderate nausea appears manageable

VK2735 Next Steps

- Phase 1 results support further study in Phase 2 trial in patients with obesity
- Study planned for mid-2023
- Novel oral tablet formulation demonstrates promising exposures in vivo
- Phase 1 study with oral formulation underway

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VK0214: Selective Thyroid Receptor-β Agonist

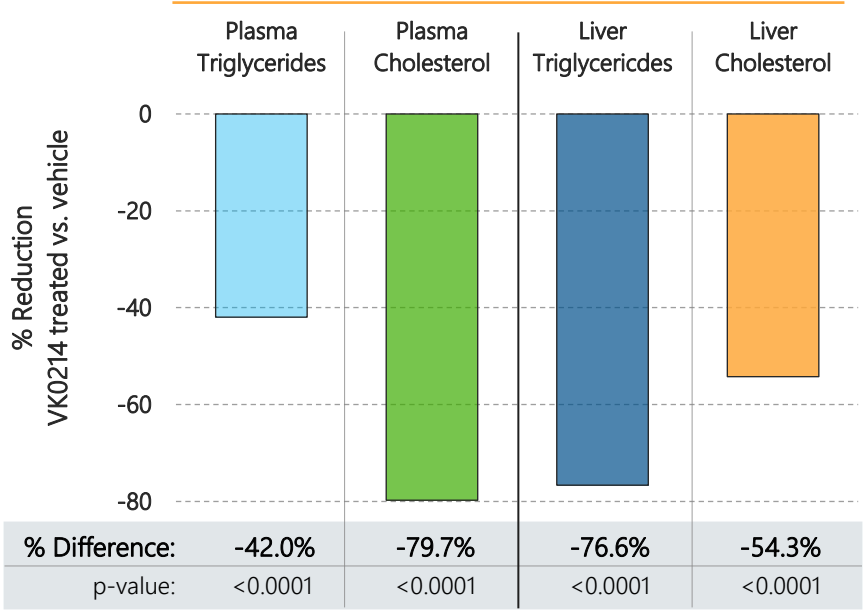
X-Linked Adrenoleukodystrophy

VK0214: Summary Profile

VK0214

- Potent small molecule thyroid receptor agonist
- 8 nM Ki at TRβ receptor
- >20:1 selective for β:α
- Oral formulation, once-daily dosing
- Robust lipid lowering effects in multiple models

Change in Lipids Following 12 Weeks of Dosing With VK0214; Rodent NASH model



	Plasma Triglycerides	Plasma Cholesterol	Liver Triglycericdes	Liver Cholesterol
% Difference:	-42.0%	-79.7%	-76.6%	-54.3%
p-value:	<0.0001	<0.0001	<0.0001	<0.0001

Demonstrates in vitro and vivo efficacy comparable to VK2809

VK0214 for X-ALD

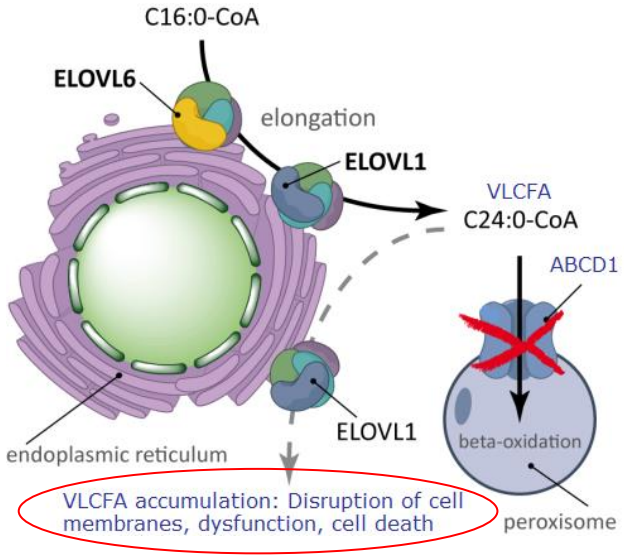
- X-Linked adrenoleukodystrophy (X-ALD)
  - Orphan neurodegenerative disorder
  - X-linked: Carried by females, primarily manifesting in males
- Most severe form: Cerebral ALD
  - Rapidly progressive inflammatory demyelination; disruption of BBB
  - Affects ~35% before age 12 (CCALD), ~20% between age 20 – 35 (CALD)
  - Deterioration in speech, cognition; vegetative state within 3-5 years
- Most common form: Adrenomyeloneuropathy (AMN)
  - Affects spinal cord, motor neurons
  - 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 and 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

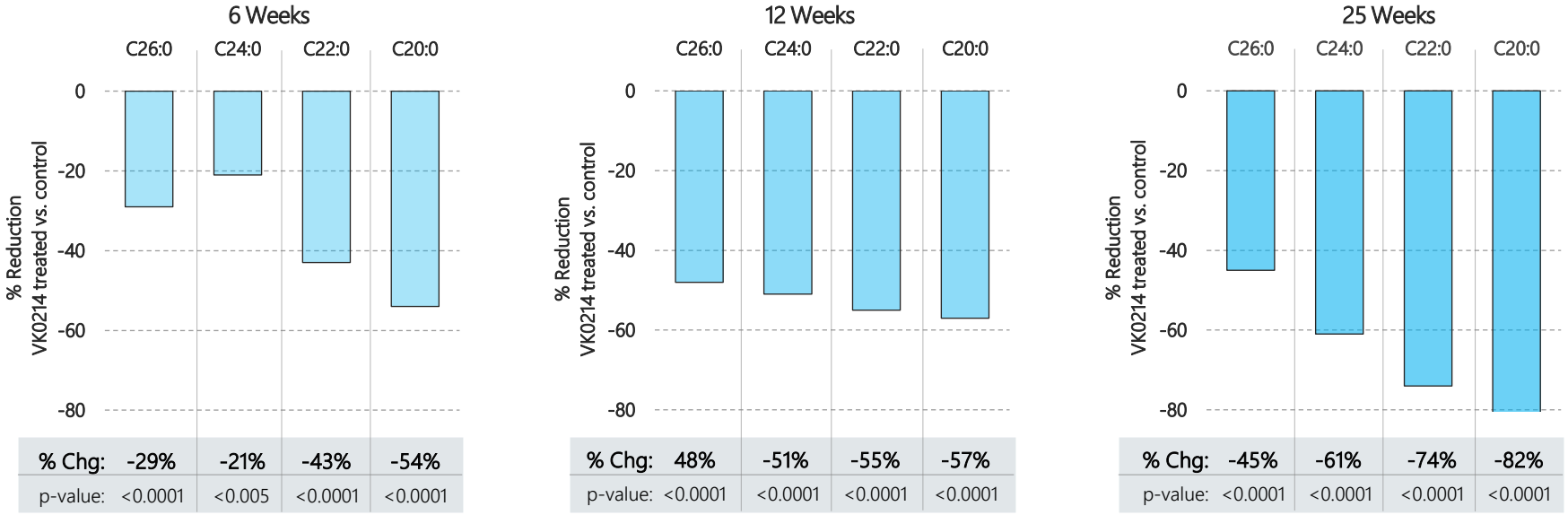
TRβ Agonists: Stimulate expression of compensatory transporters ABCD2, 3; may mitigate VLCFA elevation

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

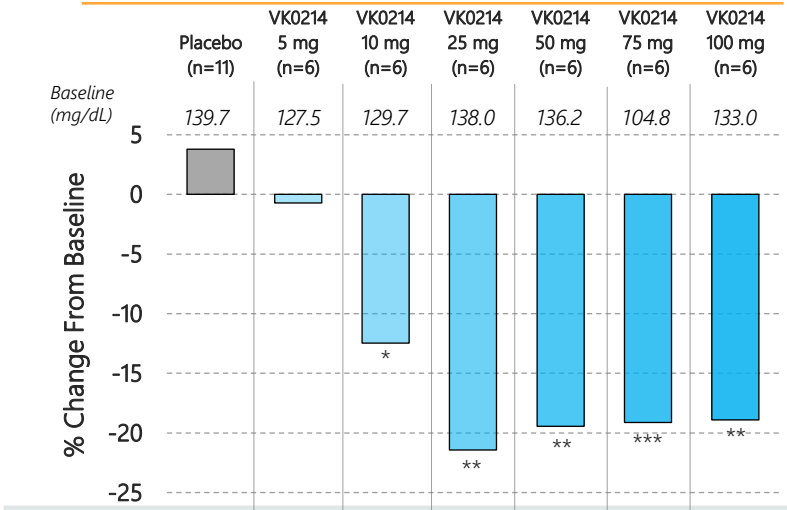
Reductions in Plasma VLCFA-LPC, ABCD1 Knockout Model



VK0214 Phase 1 Results: LDL-C Reduction Observed After 14 Days

- Reduction in LDL-C similar to observations with VK2809
- Initial effect observed @ ~10 mg
- Data to date indicate a ~20% reduction from baseline

Mean % Change in LDL-C at Day 14



	Placebo (n=11)	VK0214 5 mg (n=6)	VK0214 10 mg (n=6)	VK0214 25 mg (n=6)	VK0214 50 mg (n=6)	VK0214 75 mg (n=6)	VK0214 100 mg (n=6)
Baseline (mg/dL)	*139.7*	*127.5*	*129.7*	*138.0*	*136.2*	*104.8*	*133.0*
% Change:	3.8%	-0.7%	-12.5%	-21.4%	-19.5%	-19.1%	-18.9%
p-value:	-	0.2827	0.0230	0.0040	0.0060	0.0003	0.0049

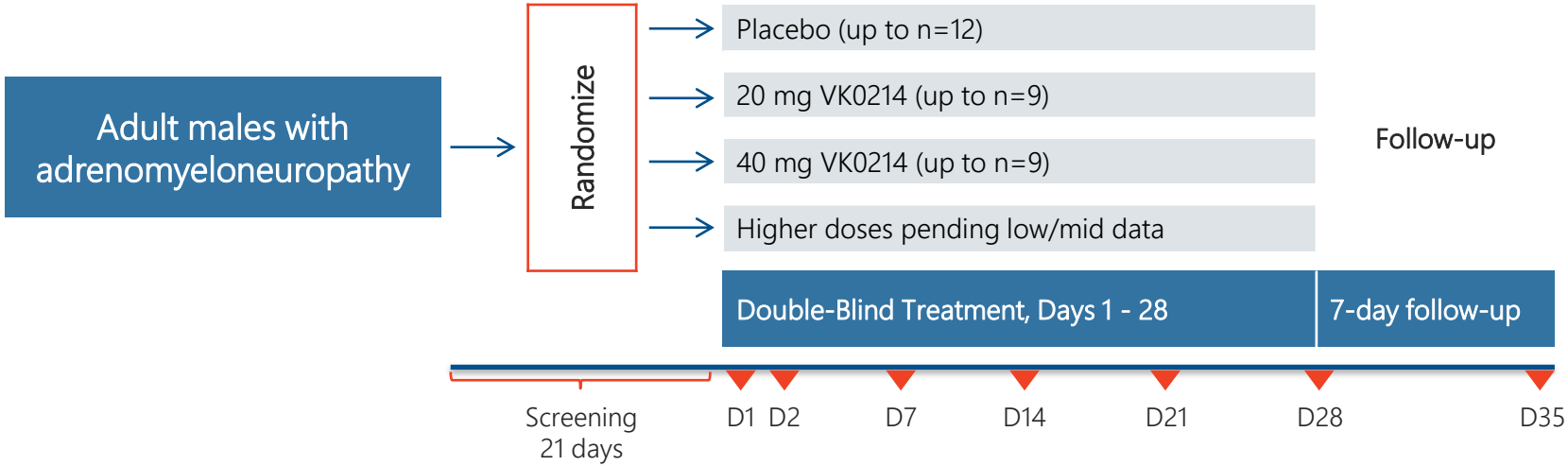
*p<0.05; **p<0.01; ***p<0.001

Magnitude of LDL-C reductions are consistent with TRβ agonist mechanism

Takeaways From VK0214 Phase 1 SAD/MAD Study

- Encouraging overall safety and tolerability, no SAEs observed
- No meaningful impact to vital signs, cardiovascular parameters, thyroid axis
- Attractive PK profile; predictable exposures with once-daily dosing
- Preliminary lipid data suggest similar efficacy to VK2809
- Results support further study in adrenomyeloneuropathy (AMN) patients

VK0214 Phase 1b Study in Adrenomyeloneuropathy



- Multicenter, parallel cohort, 28-day Phase 1b trial in adrenomyeloneuropathy
  - Higher doses may be explored pending review of initial cohorts
- Safety, tolerability, change in VLCFAs in male patients with AMN

Financial Summary

- Capital structure and summary financials

Capital Structure [1]	In '000s
Shares outstanding	78,419
Options, RSUs	6,924
Warrants	0
Total shares, options, RSUs, warrants	85,343

Financials	Dec. 31, 2022 ($'000s)
Cash burn 2022	$46,618
Cash and ST Investments	$155,485

Notes: 1) As of December 31, 2022

Investment Highlights

- Developing novel therapeutics for metabolic and endocrine diseases
  - Multiple clinical programs demonstrate best-in-class efficacy data
- Metabolic Disease Programs
  - VK2809: Novel, selective thyroid receptor-β agonist for NASH and lipid disorders
    - Phase 2b VOYAGE trial in biopsy-confirmed NASH ongoing
  - VK2735: Novel GLP-1/GIP dual agonist for obesity
    - Phase 2 obesity study planned for mid-2023
  - VK2735 Oral: GLP-1/GIP dual agonist for metabolic disorders
    - Phase 1 study ongoing
- Rare Disease Program
  - VK0214: Novel, selective thyroid receptor-β agonist for X-ALD
    - Phase 1b in patients ongoing

VIKING THERAPEUTICS

Corporate Presentation

April 2023