VIKING THERAPEUTICS

Corporate Presentation

February 2025

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.

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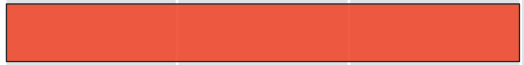
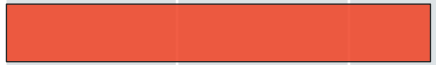
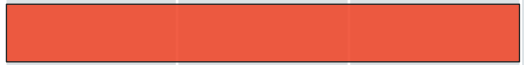
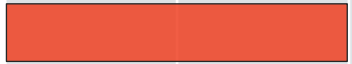
Investment Highlights

- Developing novel therapeutics for metabolic and endocrine diseases
  - Multiple clinical programs demonstrate best-in-class efficacy data
- Metabolic Disease Programs
  - VK2735: GLP-1/GIP dual agonist for obesity
    - VENTURE Phase 2 obesity study successfully achieved primary endpoint; Phase 3 planned 1H25
  - VK2735 Oral: GLP-1/GIP dual agonist for obesity
    - Phase 1 study demonstrated positive PoC, reduction in body weight; Phase 2 underway
  - VK2809: Selective thyroid receptor-β agonist for NASH/MASH
    - VOYAGE Phase 2b trial successfully primary, secondary endpoints; presented at AASLD 4Q24
- Rare Disease Program
  - VK0214: Selective thyroid receptor-β agonist for X-ALD
    - Phase 1b in patients demonstrated PoC in reducing key biomarkers of disease

Pipeline Overview

Development Programs	Indication	Stage of Development	Status
		Preclin / Phase 1 / Phase 2 / Phase 3	
VK2735 Subcutaneous (Dual GLP-1/GIP agonist)	*Obesity*		Phase 3 planned 1H25
VK2735 Oral (Dual GLP-1/GIP agonist)	*Obesity*		Phase 2 underway
VK2809 (TRβ agonist)	*NASH*		Phase 2b VOYAGE trial successfully completed
VK0214 (TRβ agonist)	*X-ALD*		Phase 1b study demonstrated PoC

Near-term events

- VK2735 Injectable: Initiation of Phase 3 studies, 1H25
- VK2735 Oral: Completion of Phase 2 VENTURE-Oral Dosing study, 2H25

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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

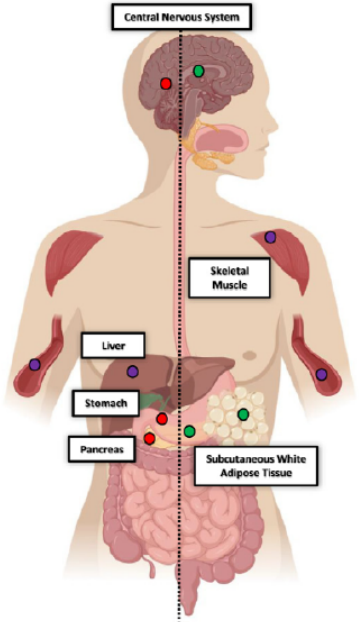
Central Nervous System
- ↑ Satiety
- ↓ Food Intake
- ↑ Nausea
- ↓ Body Weight

Pancreas
- ↑ Insulin
- ↓ Glucagon

Stomach
- ↓ Gastric Emptying

Systemic
- ↓ Hyperglycemia

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



Glucose-Dependent Insulinotropic Polypeptide Receptor

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.

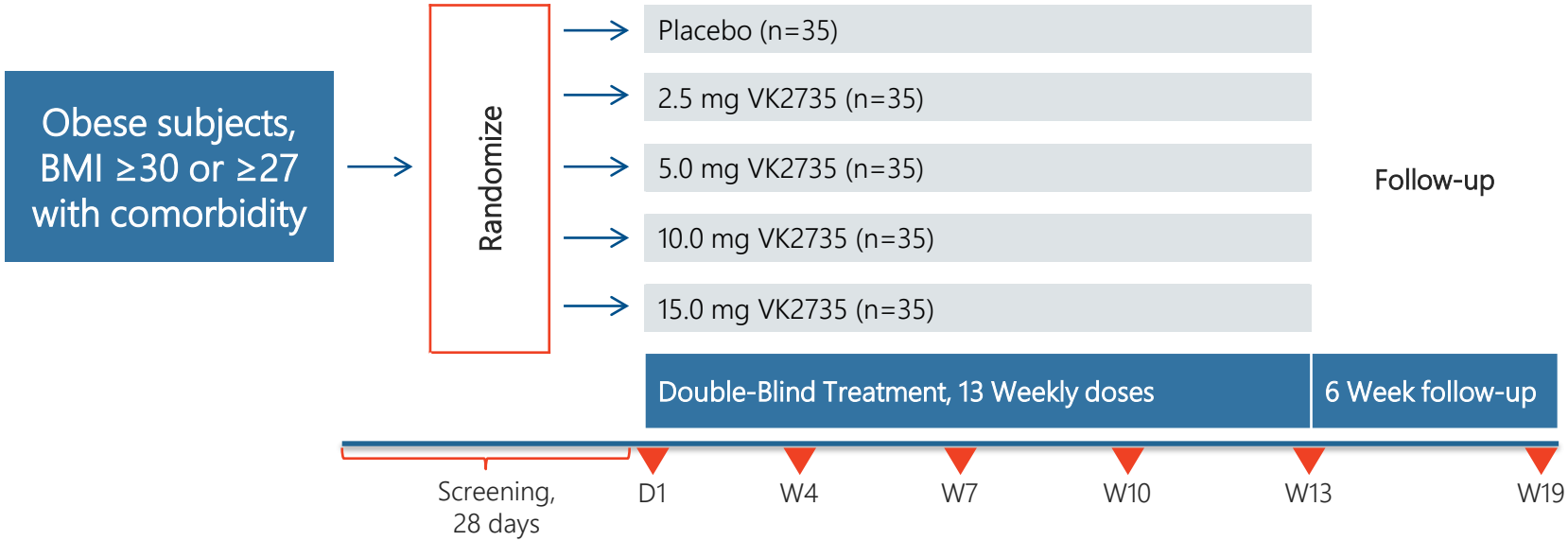
VK2735 Phase 1 Clinical Study Design

- Randomized, placebo-controlled, stacked SAD/MAD study design
- MAD: Weekly doses for 28 days
- Primary objectives: Safety, tolerability
- Exploratory: Body weight, glucose, liver fat

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
- Durable weight loss maintained 21 days after last dose
- Reductions in plasma lipids, liver fat indicate broad metabolic benefits
- PK data suggest excellent exposures from weekly dosing regimen
- Promising safety and tolerability, 98% of AEs mild to moderate

VK2735 VENTURE Phase 2 Obesity Study Design



- Multicenter, parallel cohort, 13-week trial in obese subjects
  - 3-week titration blocks applied at doses ≥5 mg
- Primary endpoint: Percent change in body weight at Week 13 vs. placebo

VK2735 VENTURE Study Demographics

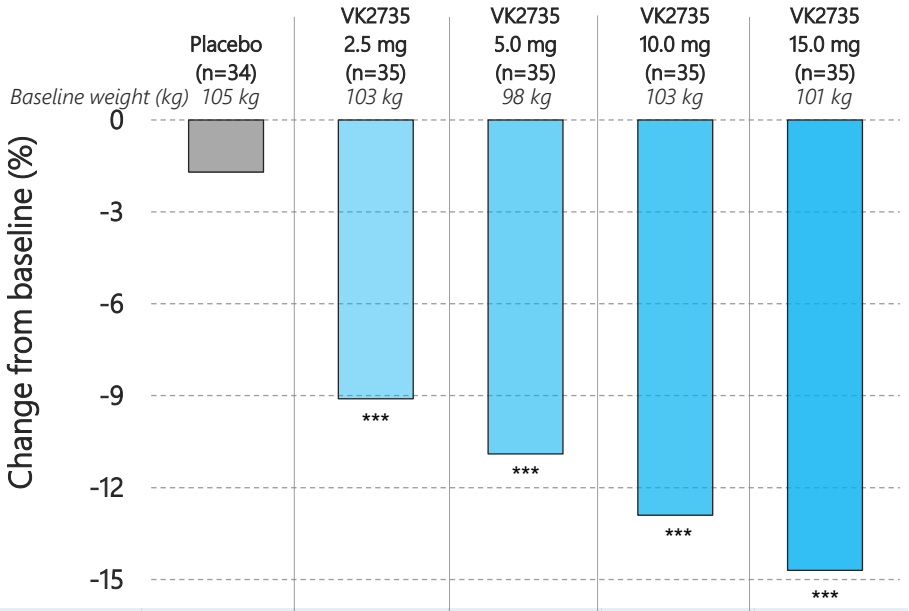
Mean Baseline Characteristics	Placebo (n=34)	2.5 mg (n=35)	5.0 mg (n=35)	10.0 mg (n=35)	15.0 mg (n=35)
Age	48	50	52	47	51
Sex, M:F (%)	18:82	23:77	34:66	34:66	23:77
White (%)	77	80	89	74	80
Weight (kg)	105	103	98	103	101
BMI (kg/m^2)	39	38	36	37	37

- Well-balanced demographics among cohorts
- Gender breakout generally 2:1 to 3:1 women to men
- BMI, weight consistent across Tx arms

VENTURE Study Achieves Primary Endpoint

- Significant reduction in body weight observed after 13 weeks
- Up to approximately 15% reduction from baseline
- Dose dependent effect observed across cohorts

Mean % Change in Body Weight After 13 Weeks

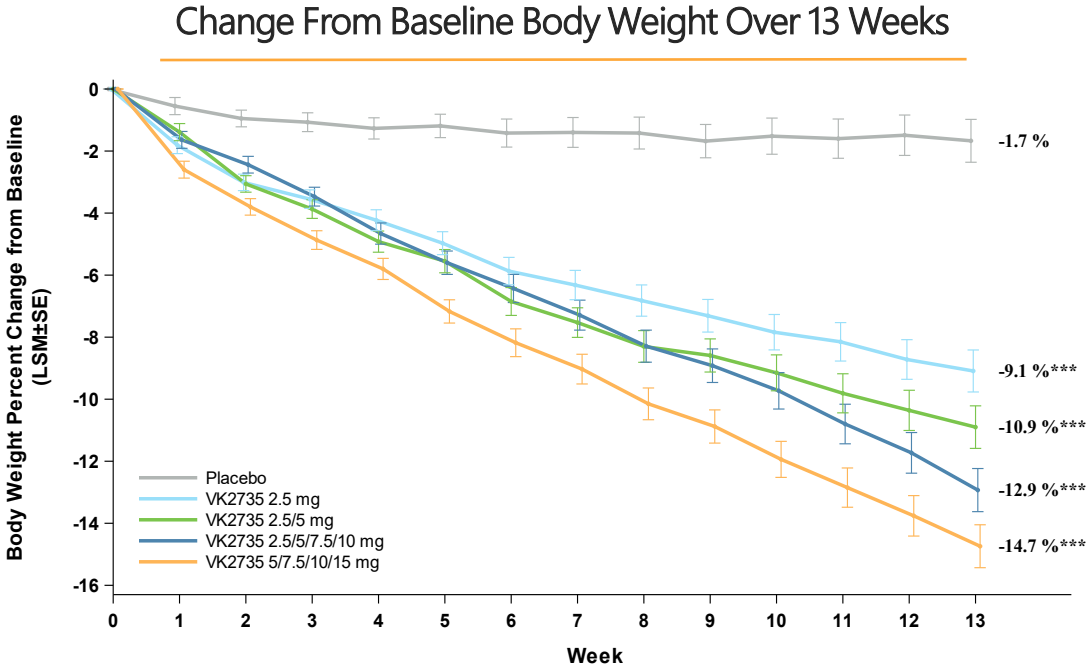


	Placebo (n=34)	VK2735 2.5 mg (n=35)	VK2735 5.0 mg (n=35)	VK2735 10.0 mg (n=35)	VK2735 15.0 mg (n=35)
Baseline weight (kg)	105 kg	103 kg	98 kg	103 kg	101 kg
Percent change	-1.7%	-9.1%	-10.9%	-12.9%	-14.7%
Placebo-adjusted	-	-7.4%	-9.2%	-11.3%	-13.1%
p-value vs. placebo	-	<0.0001	<0.0001	<0.0001	<0.0001

***p<0.0001

VENTURE Phase 2 Results: Rapid, Progressive Weight Loss Observed

- Progressive weight loss observed in all VK2735 dosing cohorts
- All doses statistically significant vs. placebo starting in Week 1 and maintained through Week 13
- Dose dependent effects observed
- No evidence of plateau suggests further body weight reduction possible with continued dosing



Notes: ***p<0.0001. Patients were required to have baseline BMI ≥30 kg/m^2 or BMI≥27 kg/m^2 with at least one weight-related comorbid condition. Patients treated with VK2735 were titrated to final doses as indicated:

2.5 mg cohort = 2.5 x 13 weeks

5 mg cohort = 2.5 mg x 3 wks, 5 mg x 10 wks

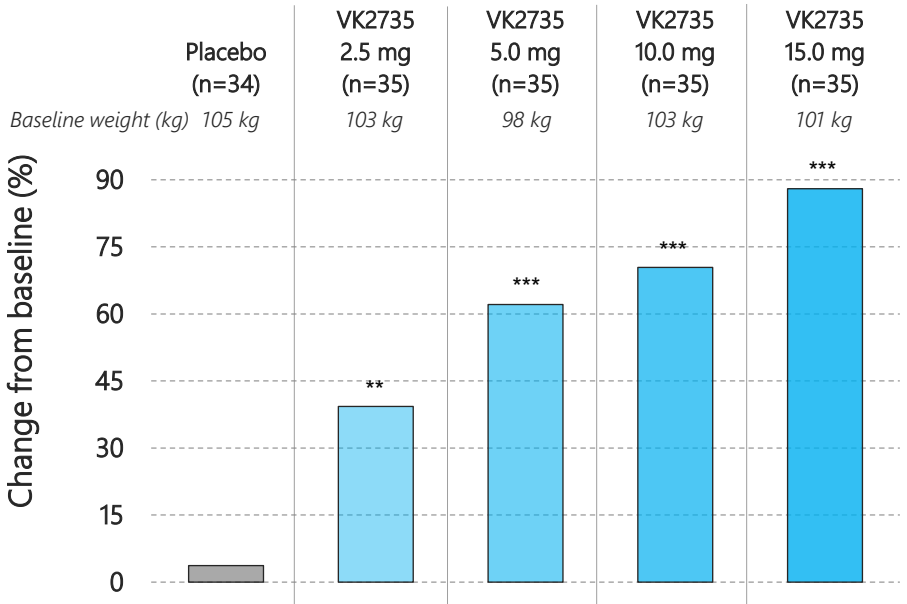
10 mg cohort = 2.5 mg x 3 wks, 5 mg x 3 wks, 7.5 mg x 3 wks, 10 mg x 4 wks

15 mg cohort = 5 mg x 3 wks, 7.5 mg x 3 wks, 10 mg x 3 wks, 15 mg x 4 wks

VENTURE Study Achieves Key Secondary Endpoint

- Up to 88% of patients experienced ≥10% weight loss
- Majority of patients receiving ≥5 mg demonstrated ≥10% weight loss
- Lowest 2.5 mg dosing cohort showed 10x placebo rate

Patients Reporting ≥10% Weight Loss at 13 Weeks

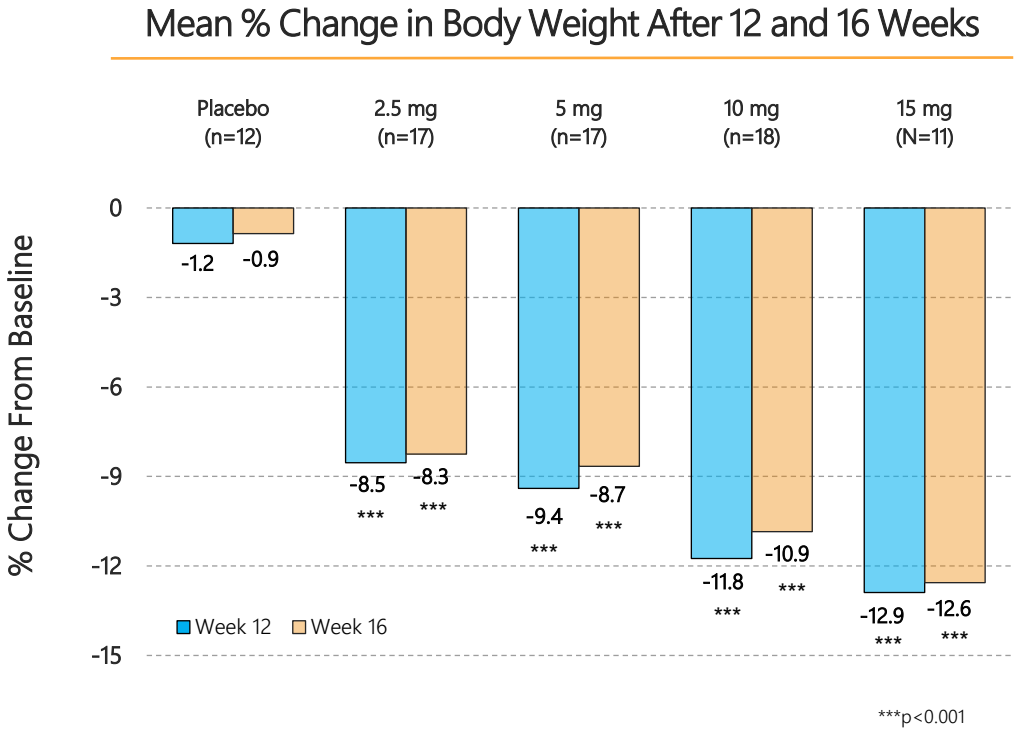


	Placebo (n=34)	VK2735 2.5 mg (n=35)	VK2735 5.0 mg (n=35)	VK2735 10.0 mg (n=35)	VK2735 15.0 mg (n=35)
Baseline weight (kg)	105 kg	103 kg	98 kg	103 kg	101 kg
Percent of patients	3.7%	39.3%	62.1%	70.4%	88.0%
p-value vs. placebo	-	0.0036	0.0002	<0.0001	<0.0001

**p<0.01, ***p<0.001

VENTURE Phase 2 Study: Maintenance Following Last Dose

- Subset of patients who participated in PK assessment
- Week 16 represents 4 weeks from last dose
- Across combined cohorts, 94% of weight loss maintained at Week 16; 83% at week 19
- Suggests monthly dosing regimen may be feasible



VK2735 VENTURE: Maintenance of Weight Loss to Week 19

Proportion of Weight Loss Maintained Following Last Dose, PK subset

Study week	Weeks after last dose	2.5 mg (n=17)	5.0 mg (n=17)	10.0 mg (n=18)	15.0 mg (n=11)	Combined VK2735 arms
16 weeks	4	98%	92%	92%	96%	94%
19 weeks	7	91%	82%	75%	87%	83%

- Majority of weight loss maintained at 4-week and 7-week follow-up visits
- Suggests monthly maintenance dosing may be feasible

VK2735 VENTURE: Shift in Diabetes Status at Week 13

Shift in Diabetes Status From Baseline to Week 13

Parameter[1]	Placebo	2.5 mg	5.0 mg	10.0 mg	15.0 mg	Combined VK2735 arms
Pre-diabetic at baseline[2]	14	21	21	16	16	75
Number shifting to normoglycemic at Week 13 (%)[3]	4 (29%)	17 (81%)	16 (76%)	10 (63%)	15 (94%)	58 (78%)
p-value vs. placebo[4]	-	0.0041	0.0132	0.0813	0.0004	0.0005

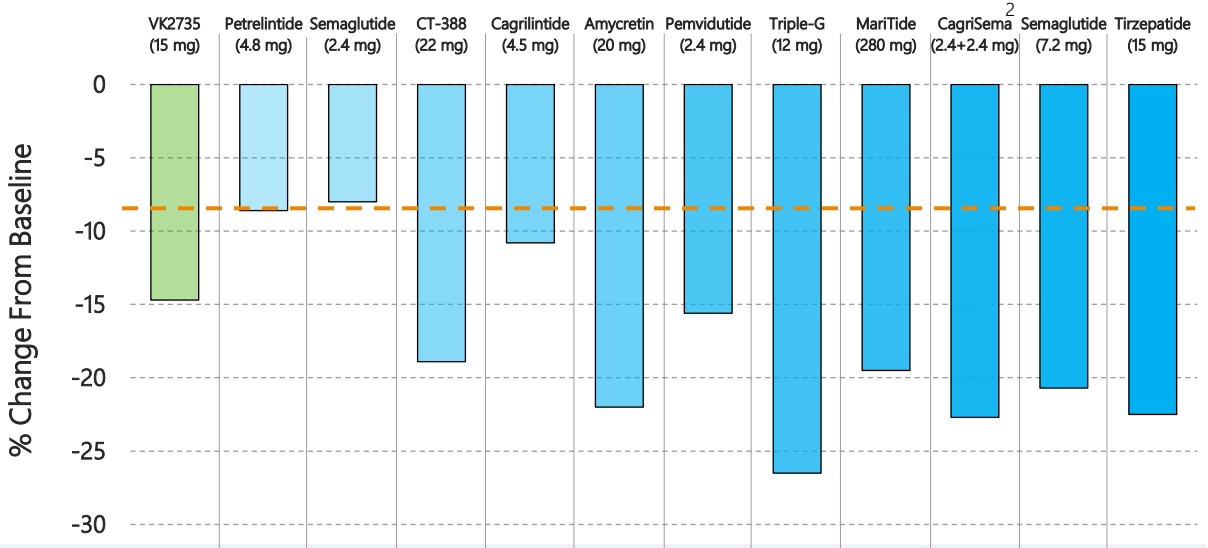
Notes: 1) Observed values, no imputation for missing data. 2) Defined as patients with fasting plasma glucose 100 mg/dL to 125 mg/dL or HbA1c 5.7% to 6.4%. 3) Defined as fasting plasma glucose <100 mg/dL or HbA1c <5.7%. 4) Fisher's exact test.

- Rapid shift from pre-diabetic to normoglycemia over 13 weeks
- Shifts suggest reduced risk of diabetes among patients receiving VK2735

Comparison With Published Data for Other Weight Loss Agents

- VK2735 weight loss appears competitive with other agents despite shorter trial duration
- Longer-term data will be key for determining maximal efficacy

Change in Body Weight Across Competitive Landscape



	VK2735 (15 mg)	Petrelintide (4.8 mg)	Semaglutide (2.4 mg)	CT-388 (22 mg)	Cagrilintide (4.5 mg)	Amycretin (20 mg)	Pemvidutide (2.4 mg)	Triple-G (12 mg)	MariTide (280 mg)	CagriSema[2] (2.4+2.4 mg)	Semaglutide (7.2 mg)	Tirzepatide (15 mg)
% Change in BW:	-14.7%	-8.6%	-8.0%	-18.9%	-10.8%	-22.0%	-15.6%	-26.5%	-20.0%[1]	-22.7%	-20.7%	-22.5%
Duration in Weeks	13	16	20	24	26	36	48	48	52	68	72	72

Notes: Data represent change from baseline. — Indicates approximate tirzepatide 12-week weight loss in Phase 3 Surmount 1 study (~8%).

1: Approximate value as reported by company

2: Reported result assumes all patients adhered to treatment (trial product estimand, regardless of dose), with 57.3% of CagriSema patients on the highest dose (2.4 mg + 2.4 mg) after 68 weeks.

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VENTURE Study Discontinuation Rates Well-Balanced

Number of patients reporting (%)	Placebo (n=35)	VK2735 2.5 mg (n=35)	VK2735 5 mg (n=35)	VK2735 10 mg (n=35)	VK2735 15 mg (n=35)	VK2735 Combined (n=140)
Discontinued treatment early	5 (14%)	2 (6%)	4 (11%)	5 (14%)	7 (20%)	18 (13%)
Discontinued study early	2 (6%)	1 (3%)	1 (3%)	2 (6%)	2 (6%)	6 (4%)
Overall TEAEs	24 (69%)	25 (71%)	31 (89%)	30 (86%)	32 (91%)	118 (84%)
Drug related TEAEs	16 (46%)	21 (60%)	27 (77%)	26 (74%)	30 (86%)	104 (74%)
Drug related TEAEs leading to study discontinuation	0 (0%)	0 (0%)	0 (0%)	0 (0%)	1 (3%)	1 (1%)

Notes: Study safety population, defined as all patients who were randomized and received at least one dose of study drug or placebo.

- Discontinuations well balanced between placebo, VK2735 treatment groups
- Majority (92%) of drug related TEAEs among VK2735 patients mild or moderate
- One VK2735 treated patient experienced SAE of dehydration, probably drug related

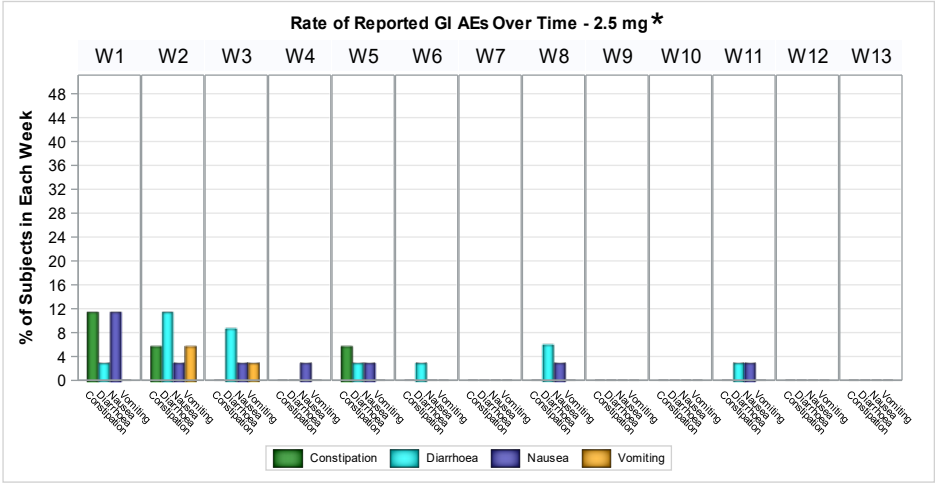
VENTURE Phase 2 Study: GI Tolerability Summary

Common GI related TEAEs Number of patients reporting (%)	Placebo (n=35)	VK2735 2.5 mg (n=35)	VK2735 5 mg (n=35)	VK2735 10 mg (n=35)	VK2735 15 mg (n=35)	VK2735 Combined (n=140)
GERD	1 (3%)	2 (6%)	5 (14%)	4 (11%)	6 (17%)	17 (12%)
Nausea						
Mild	7 (20%)	6 (17%)	11 (31%)	9 (26%)	15 (43%)	41 (29%)
Moderate	0 (0%)	3 (9%)	5 (14%)	4 (11%)	7 (20%)	19 (14%)
Severe	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)
Vomiting	0 (0%)	3 (9%)	6 (17%)	6 (17%)	10 (29%)	25 (18%)
Abdominal pain	1 (3%)	1 (3%)	2 (6%)	1 (3%)	2 (6%)	6 (4%)
Diarrhea	3 (9%)	11 (31%)	6 (17%)	7 (20%)	4 (11%)	28 (20%)
Constipation	4 (11%)	7 (20%)	10 (29%)	9 (26%)	10 (29%)	36 (26%)
Decreased appetite	0 (0%)	2 (6%)	5 (14%)	9 (26%)	6 (17%)	22 (16%)

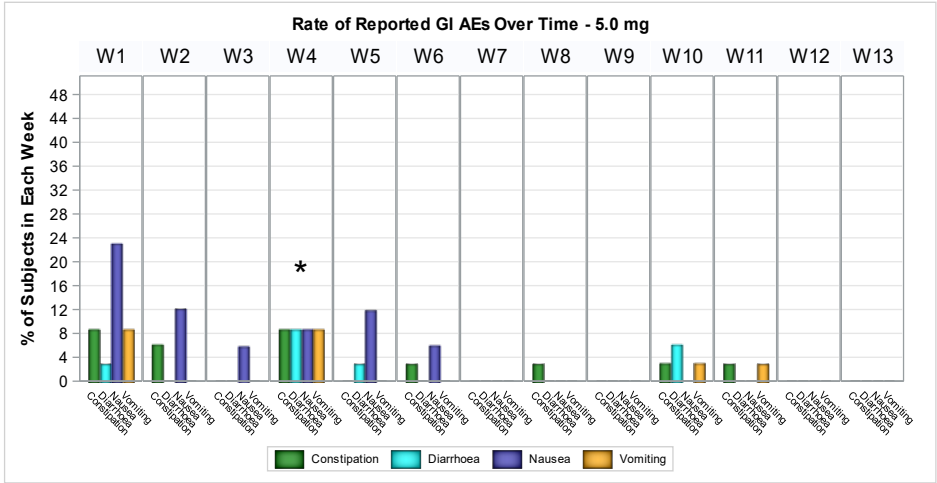
GERD: Gastroesophageal reflux disease.

- Majority (95%) GI specific TEAEs among VK2735 patients mild or moderate

Time Course of GI AEs Through 13 Weeks; 2.5 mg and 5 mg Cohorts



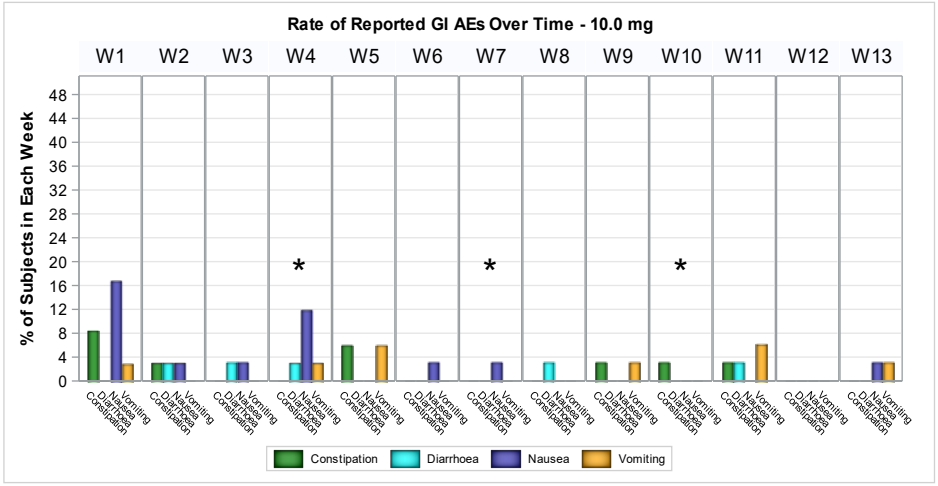
*2.5 mg cohort received fixed 2.5 mg doses for 13 weeks, no titration.



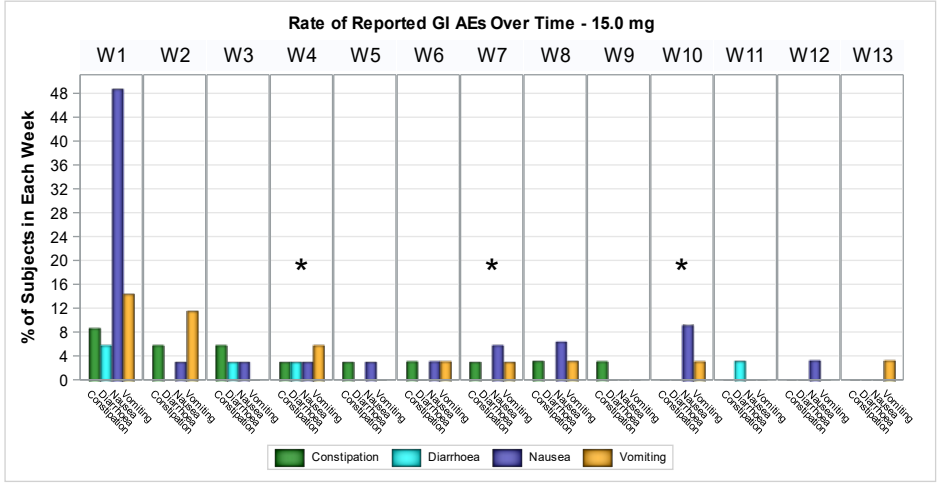
* Denotes up-titration per schedule: 2.5 mg x 3 wks, 5 mg x 10 wks.

- GI AEs most common, expected per GLP-1 mechanism: nausea, vomiting, diarrhea, constipation
- Generally observed early, subside over time

Time Course of GI AEs Through 13 Weeks; 10 mg and 15 mg Cohorts



*Denotes up-titration per schedule: 2.5 mg x 3 wks, 5 mg x 3 wks, 7.5 mg x 3 wks, 10 mg x 4 wks.



*Titration: 5 mg x 3 wks, 7.5 mg x 3 wks, 10 mg x 3 wks, 15 mg x 4 wks.

- GI AEs most common, expected per GLP-1 mechanism: nausea, vomiting, diarrhea, constipation
- Generally observed early, subside over time

VENTURE Phase 2 Study Takeaways

- Up to 14.7% mean weight loss observed after 13 weeks of VK2735 treatment
- Promising tolerability, 92% of all drug related TEAEs mild to moderate
- Durable weight loss observed, >90% of efficacy retained 4 weeks after last dose
- PK suggestive of potential monthly regimen
- Majority of GI-related AEs occur early in treatment, resolve
- Phase 3 trials planned

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VK2735: Oral Formulation

Metabolic Disorders

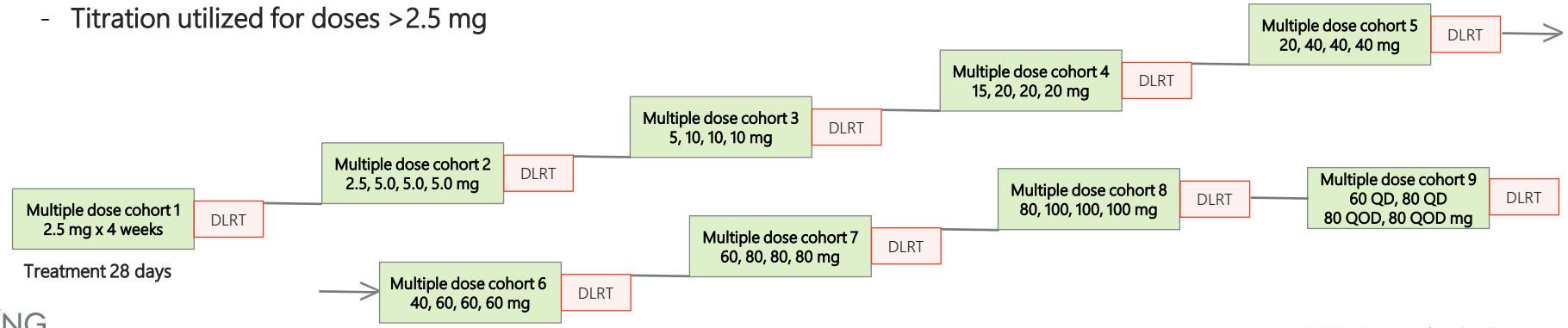
Oral Formulation Overview

- Exploratory work pursued to develop oral formulation of VK2735
- Multiple variations evaluated in multiple species
- Highly iterative process
- Resulted in oral tablet with reproducible exposures
- Tablet formulation progressed into Phase 1 clinical trial
- Ongoing efforts to understand breadth, applicability of oral formulation

VK2735-101 Oral Study

- Phase 1 MAD study design
- Placebo-controlled extension of ongoing trial
- Primary objectives: Safety, tolerability
- Exploratory assessments: Body weight, glucose, lipids after 28 days

First in human study design
- N=8-10 per cohort (~4:1 active:placebo)
- Titration utilized for doses >2.5 mg



DLRT = Dose Level Review Team

VK2735 Oral Study Demographics

Mean Baseline Characteristics	Placebo (n=19)	2.5 mg (n=8)	5 mg (n=7)	10 mg (n=6)	20 mg (n=8)	40 mg (n=8)	60 mg (n=9)	80 mg (n=9)	100 mg (n=9)
Age	38	34	29	35	35	33	44	44	44
Sex, M:F (%)	47:53	63:37	43:57	17:83	63:37	25:75	33:67	56:44	44:56
White (%)	68	75	71	100	88	63	89	100	67
Weight (kg)	99	102	97	97	111	89	108	102	103
BMI (kg/m^2)	36	36	34	36	36	33	37	35	35

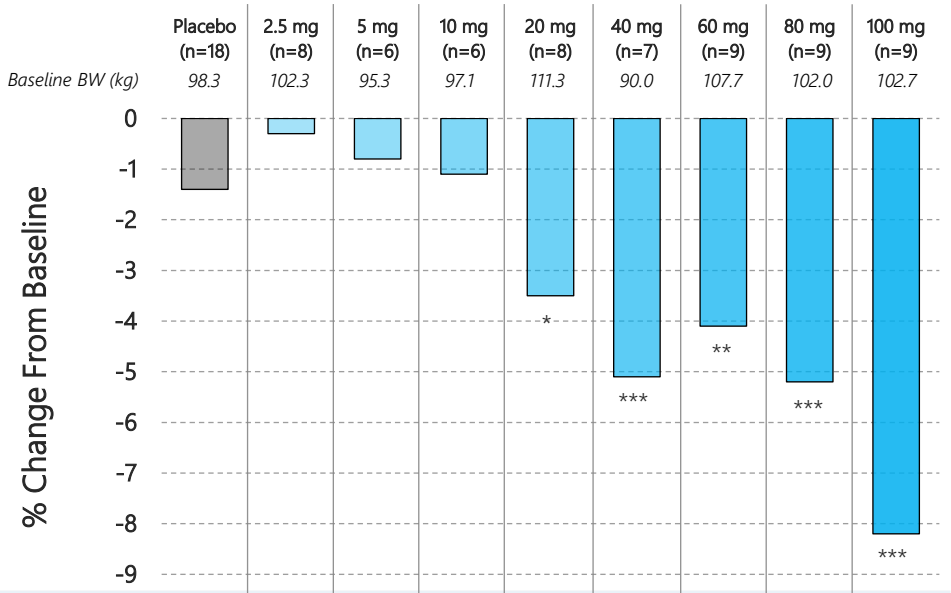
Notes: Safety population, includes all randomized subjects who received at least one dose of study drug or placebo.

- Generally balanced demographics among cohorts
- BMI, weight consistent across Tx arms

Oral VK2735 Phase 1 Results: Weight Change After 28 Days

- Dose dependent reduction in body weight observed across VK2735 dosing cohorts
- Up to approximately 7% placebo-adjusted weight loss at 100 mg

Mean % Change in Body Weight at Day 28

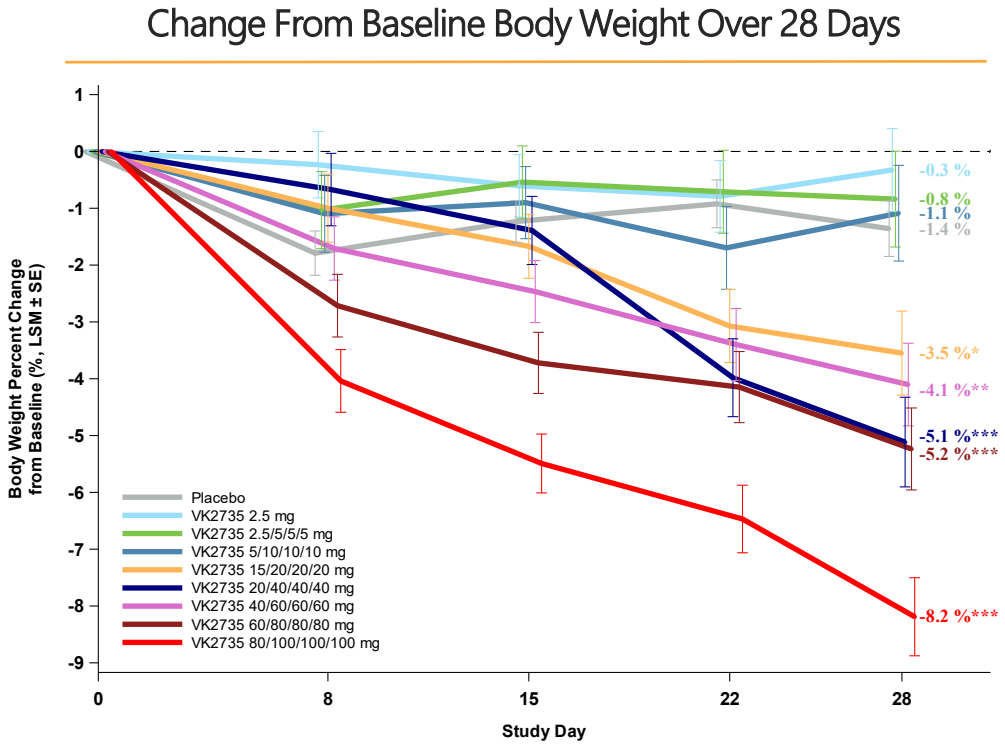


	Placebo (n=18)	2.5 mg (n=8)	5 mg (n=6)	10 mg (n=6)	20 mg (n=8)	40 mg (n=7)	60 mg (n=9)	80 mg (n=9)	100 mg (n=9)
% Change in BW:	-1.4%	-0.3%	-0.8%	-1.1%	-3.5%	-5.1%	-4.1%	-5.2%	-8.2%
Placebo-adjusted:	-	1.0%	0.6%	0.3%	-2.2%	-3.7%	-2.7%	-3.9%	-6.8%
p-value vs placebo:	-	-	-	-	0.017	0.0001	0.0026	<0.0001	<0.0001

Notes: Baseline BMI ≥30 in all subjects.*p<0.05, **p<0.01, ***p<0.001.

VK2735 Oral Phase 1 Results: Progressive Weight Loss Observed

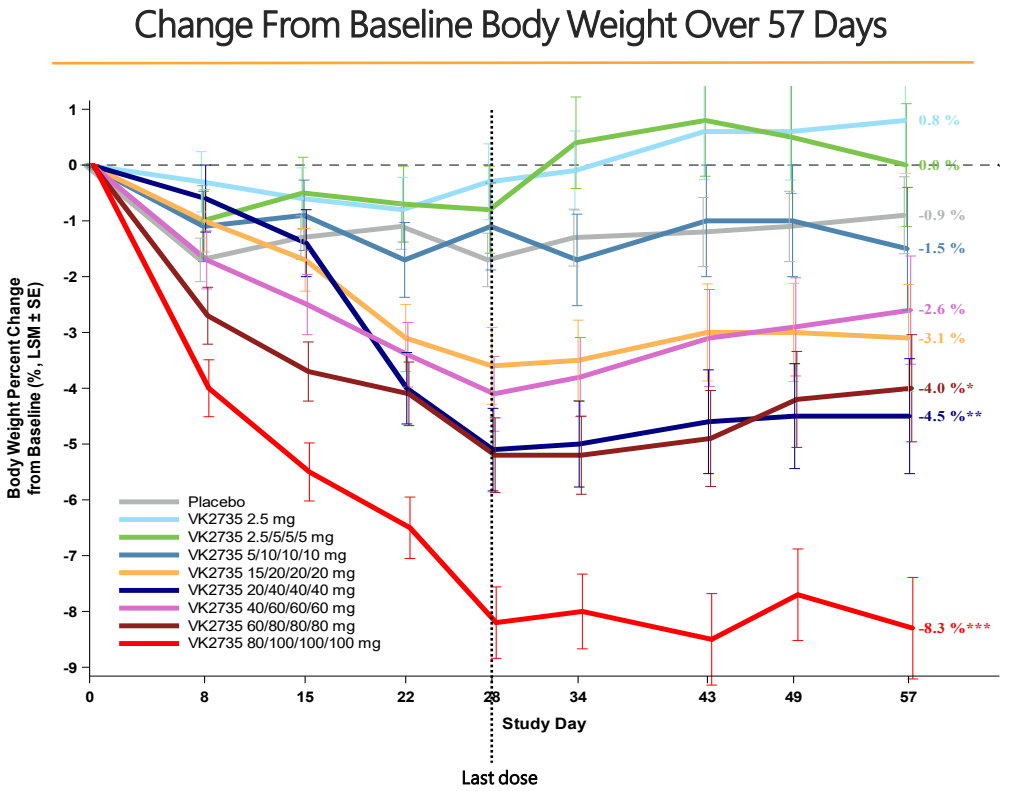
- Overall dose dependent effects among VK2735 cohorts
- Progressive weight loss observed at doses ≥20 mg; no plateau at D28
- Trends suggests further weight reduction possible with longer dosing period



*p<0.05; **p<0.01; ***p<0.001. p-value for comparison of LS mean difference from baseline between treatment and placebo, adjusted for baseline body weight.

VK2735 Oral Phase 1 Results: Weight Loss Through Day 57

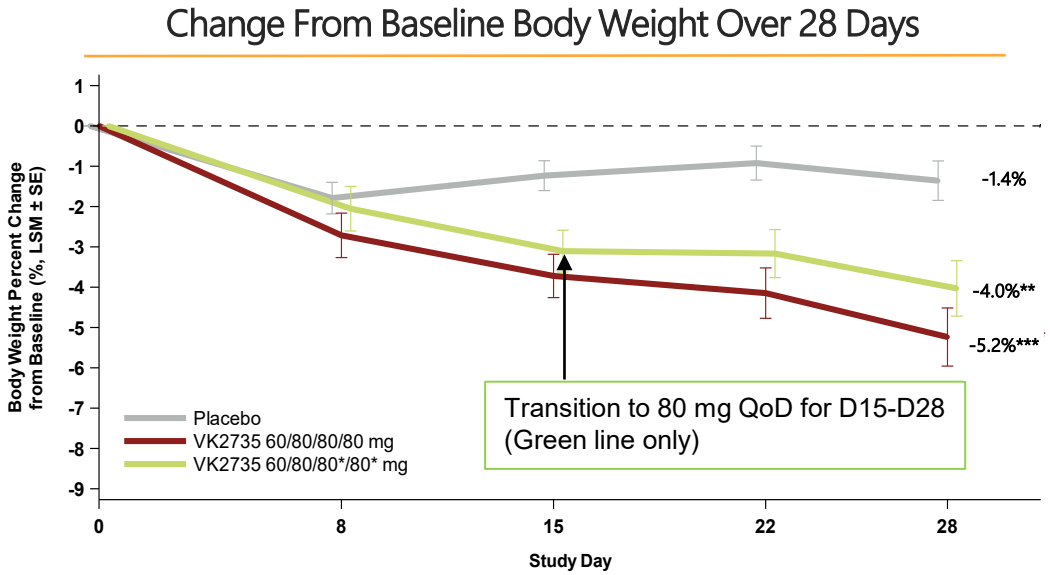
- Weight loss effects largely maintained through Day 57
- 4 Weeks from last study dose
- Suggests maintenance may be feasible at lower doses vs. induction



Notes: Baseline BMI ≥30 in all subjects. *p-value < 0.05, **p-value < 0.01, ***p-value < 0.001. p-value for the comparison of the LS Mean Difference from baseline between treatment and placebo.

VK2735 Oral Phase 1 Results: Exploratory Maintenance Cohort

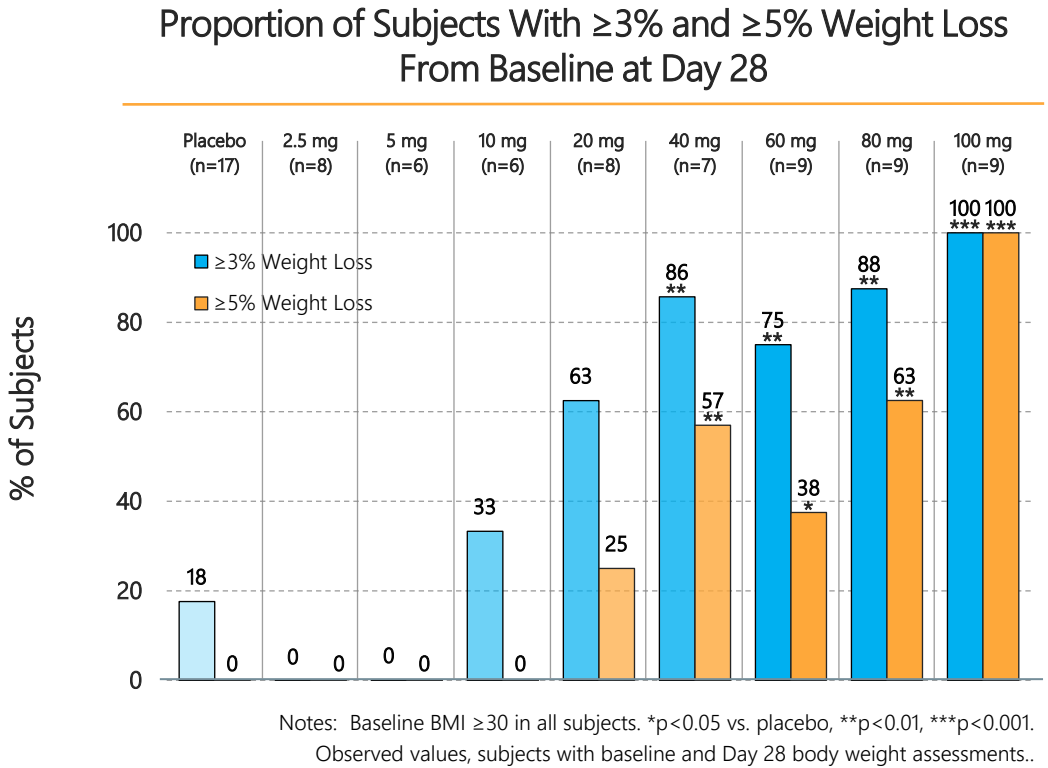
- Exploratory cohort to evaluate higher-lower exposure regimen
- Progressive weight loss maintained despite 50% dose reduction over final 2 weeks
- Suggests low maintenance dose may retain/extend body weight reduction



Comparison between high, low exposure 80 mg regimens; 80 mg QD (red) and 80 mg QoD (green, denoted 80* mg).
80 mg QD (red): 60 mg daily x 1 wk, 80 mg daily x 3 wks.
80 mg QoD (green): 60 mg daily x 1 wk, 80 mg daily x 1 wk, 80 mg QoD x 2 wks.
**p<0.01, ***p<0.001.

Oral VK2735 Phase 1 Results: Subjects with ≥3% and ≥5% Weight Loss

- Dose response shows increased proportion of subjects with 3% and 5% weight loss at higher doses with increasing VK2735 dose
- Potential to improve with higher dose and/or longer dosing period

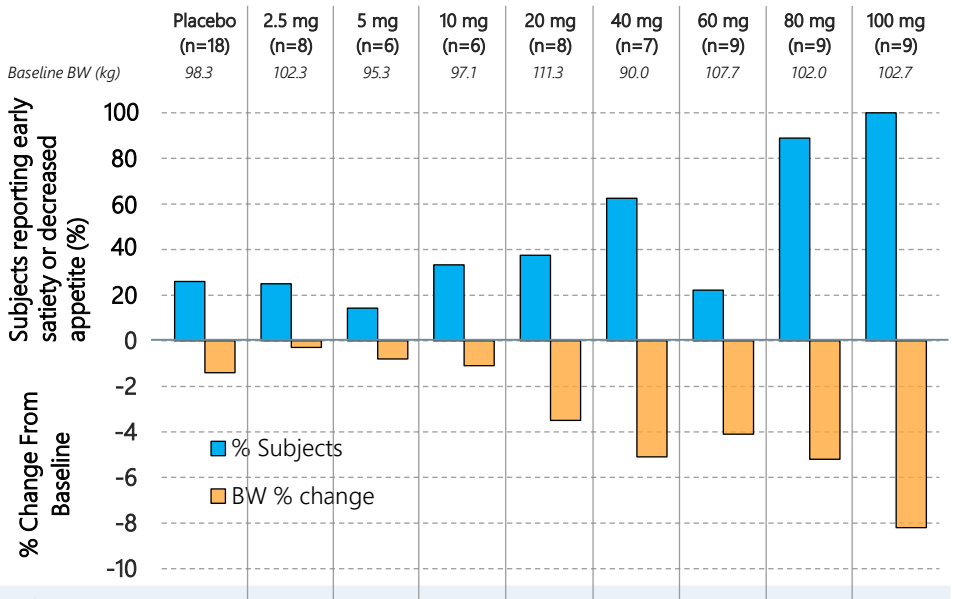


Notes: Baseline BMI ≥30 in all subjects. *p<0.05 vs. placebo, **p<0.01, ***p<0.001.
Observed values, subjects with baseline and Day 28 body weight assessments..

VK2735 Results: Weight Change vs. Early Satiety or Decreased Appetite

- Majority of subjects dosed ≥40 mg reported reduced appetite/increased satiety at Day 28, including all subjects in 100 mg cohort
- Satiety/appetite an established clinical result following GLP-1/GIP activation
- Longer term dosing may demonstrate further reduction in body weight

Change From Baseline Body Weight vs. Satiety



	Placebo (n=18)	2.5 mg (n=8)	5 mg (n=6)	10 mg (n=6)	20 mg (n=8)	40 mg (n=7)	60 mg (n=9)	80 mg (n=9)	100 mg (n=9)
% Subjects:	26%	25%	14%	33%	38%	63%	22%	89%	100%
% Change in BW:	-1.4%	-0.3%	-0.8%	-1.1%	-3.5%	-5.1%	-4.1%	-5.2%	-8.2%

Notes: Baseline BMI ≥30 in all subjects.

VK2735 Oral Phase 1 Study: Adverse Events and Discontinuations

Number of subjects	Placebo (n=19)	VK2735 2.5 mg (n=8)	VK2735 5 mg (n=7)	VK2735 10 mg (n=6)	VK2735 20 mg (n=8)	VK2735 40 mg (n=8)	VK2735 60 mg (n=9)	VK2735 80 mg A (n=9)	VK2735 80 mg B (n=9)	VK2735 100 mg (n=9)
Discontinued study early	2 (11%)	0 (0%)	1 (14%)	0 (0%)	0 (0%)	1 (13%)	1 (11%)	1 (11%)	0 (0%)	0 (0%)
Treatment emergent adverse events, TEAEs	16 (84%)	6 (75%)	6 (86%)	4 (67%)	6 (75%)	7 (88%)	9 (100%)	9 (100%)	8 (89%)	9 (100%)
Drug related TEAEs	11 (58%)	4 (50%)	4 (57%)	3 (50%)	4 (50%)	7 (88%)	6 (67%)	9 (100%)	7 (78%)	9 (100%)
Serious adverse events	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	1 (11%)	0 (0%)	0 (0%)	0 (0%)

Notes: Study safety population, defined as all patients who were randomized and received at least one dose of study drug. Data as of March 18, 2024. Patients treated with VK2735 were titrated to final doses as indicated: 2.5 mg cohort = 2.5 daily x 4 weeks; 5 mg cohort = 2.5 mg daily x 1 wk, 5 mg daily x 3 wks; 10 mg cohort = 5 mg daily x 1 wk, 10 mg daily x 3 wks; 20 mg cohort = 15 mg daily x 1 wk, 20 mg daily x 3 wks; 40 mg cohort = 20 mg daily x 1 wk, 40 mg daily x 3 wks; 40 mg cohort = 60 mg daily x 3 wks; 80 mg A = 60 mg daily x 1 wk, 80 mg daily x 3 wks; 80 mg B = 60 mg daily x 1 wk, 80 mg daily x 1 wk, 80 mg QoD x 2 wks; 100 mg cohort= 80 mg daily x 1 wk, 100 mg daily x 3 wks.

- Discontinuation rates low, balanced across treatment and placebo cohorts
- Majority of observed TEAEs (99%) were reported as mild to moderate

VK2735 Oral Phase 1 Study: GI Tolerability Summary

Common GI related TEAEs Number of subjects reporting (%)	Placebo (n=19)	VK2735 2.5 mg (n=8)	VK2735 5 mg (n=7)	VK2735 10 mg (n=6)	VK2735 20 mg (n=8)	VK2735 40 mg (n=8)	VK2735 60 mg (n=9)	VK2735 80 mg A (n=9)	VK2735 80 mg B (n=9)	VK2735 100 mg (n=9)
GERD	1 (5%)	0 (0%)	0 (0%)	0 (0%)	1 (13%)	0 (0%)	1 (11%)	2 (22%)	0 (0%)	0 (0%)
Nausea										
Mild	2 (11%)	0 (0%)	1 (14%)	0 (0%)	2 (25%)	2 (25%)	2 (22%)	6 (67%)	4 (44%)	6 (67%)
Moderate	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)
Severe	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)
Vomiting	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	1 (11%)	1 (11%)	1 (11%)
Abdominal pain	2 (11%)	0 (0%)	1 (14%)	1 (17%)	0 (0%)	0 (0%)	1 (11%)	0 (0%)	0 (0%)	0 (0%)
Diarrhea	4 (21%)	0 (0%)	0 (0%)	0 (0%)	1 (13%)	0 (0%)	1 (11%)	1 (11%)	1 (11%)	1 (11%)
Constipation	3 (16%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	3 (33%)	2 (22%)	1 (11%)	4 (44%)

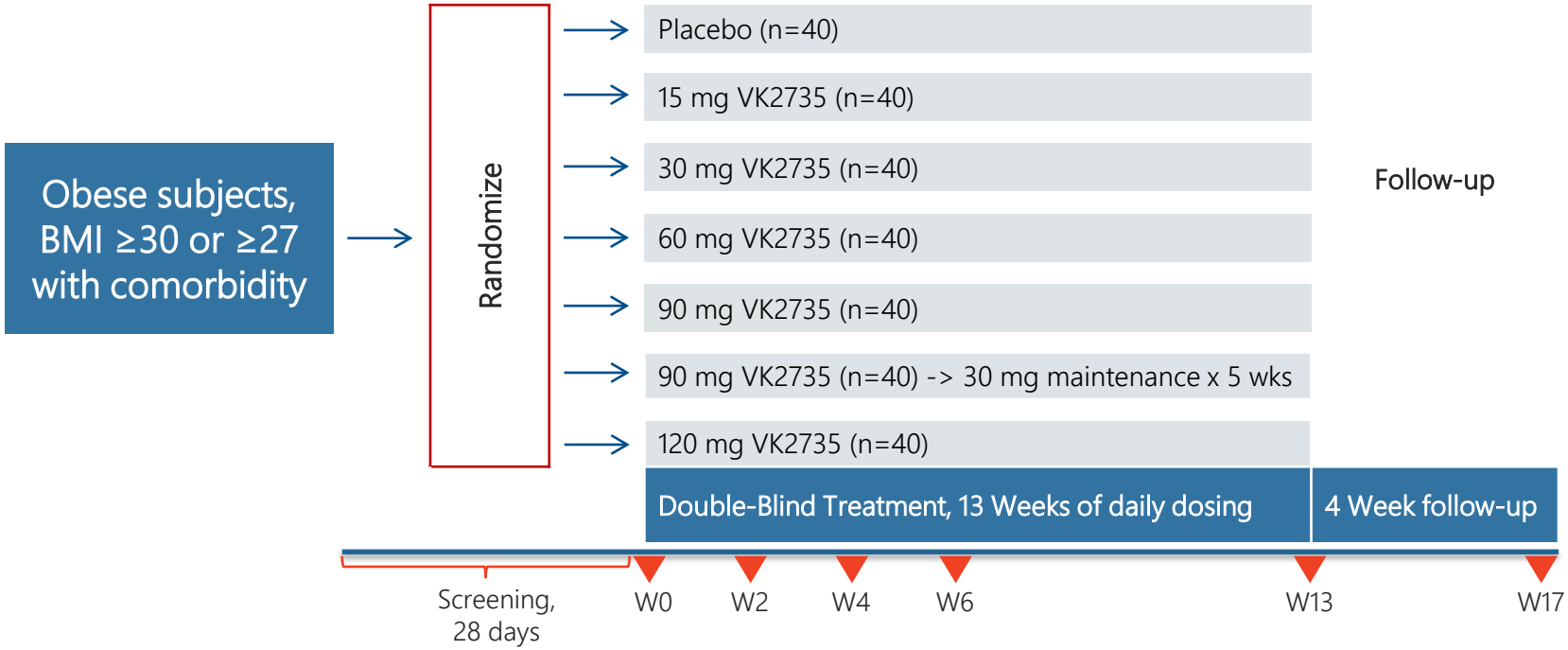
Notes: Safety population, includes all randomized subjects who received at least one dose of study drug or placebo. 80 mg Cohort A = 60 mg x 1 wk, 80 mg x 3 wks; 80 mg Cohort B = 60 mg x 1 wk, 80 mg x 1 wk, 80 mg QoD x 2 wks; GERD: gastroesophageal reflux disease.

- Tolerability continues to be promising; nausea increasing (mild) at higher doses

VK2735 Oral Phase 1 Study Takeaways, Next Steps

- Up to 8.2% reduction in body weight observed after 28 days of oral dosing
- Progressive effect suggests further weight loss possible with longer treatment
- Majority of weight loss maintained 4-weeks following final dose
- Dose-dependent exposures with daily dosing; accumulation likely ongoing at D28
- Excellent tolerability profile through 100 mg dose level; 99% of AEs mild to moderate
- Mild nausea reported at higher doses, likely addressable with slower titration
- Minimal GI AEs; low rates of vomiting, diarrhea, constipation in higher dose cohorts
- Exploratory transition from 80 mg QD to 80 mg QoD suggests feasibility of lower dose maintenance regimens
- VENTURE-Oral Dosing Phase 2 study underway

VENTURE Oral Dosing Phase 2a Study



- Multicenter, parallel cohort, 13-week trial in obese subjects
  - 2-week titration blocks applied at doses ≥60 mg

VIKING THERAPEUTICS

VK2809: Selective Thyroid Receptor-β Agonist

NASH/MASH

Thyroid Hormone Receptor Overview

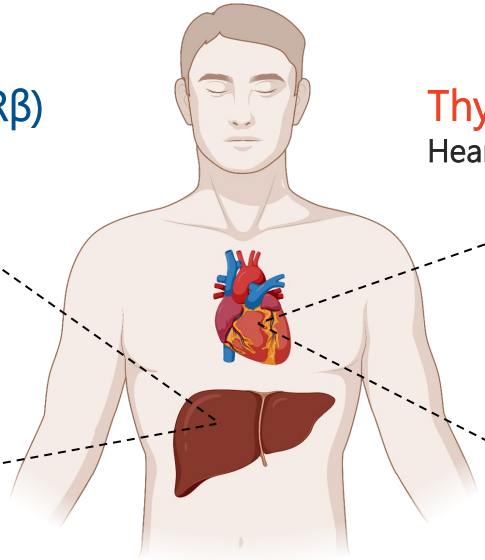
Nuclear hormone receptors: 2 main types

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)

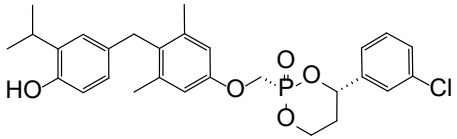
Heart, skeletal muscle

Negative effects

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

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

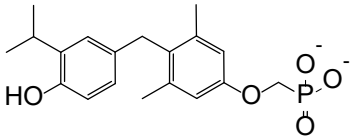
VK2809: Unique Liver-Targeted Characteristics



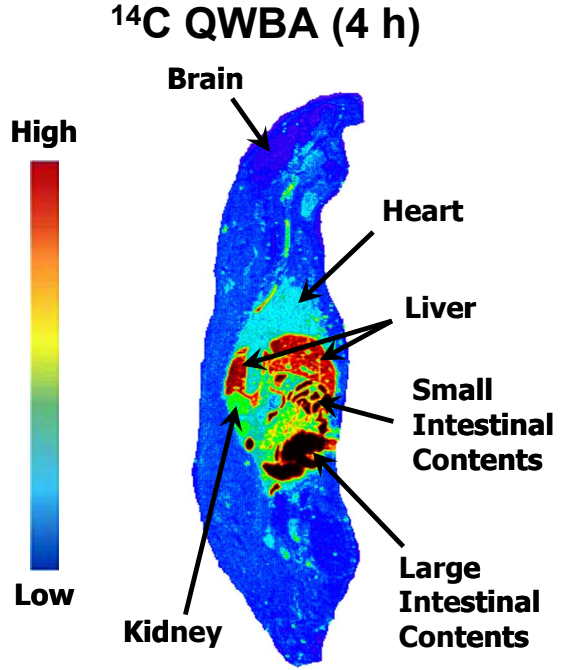
VK2809, Novel Prodrug

Following oral dosing:

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

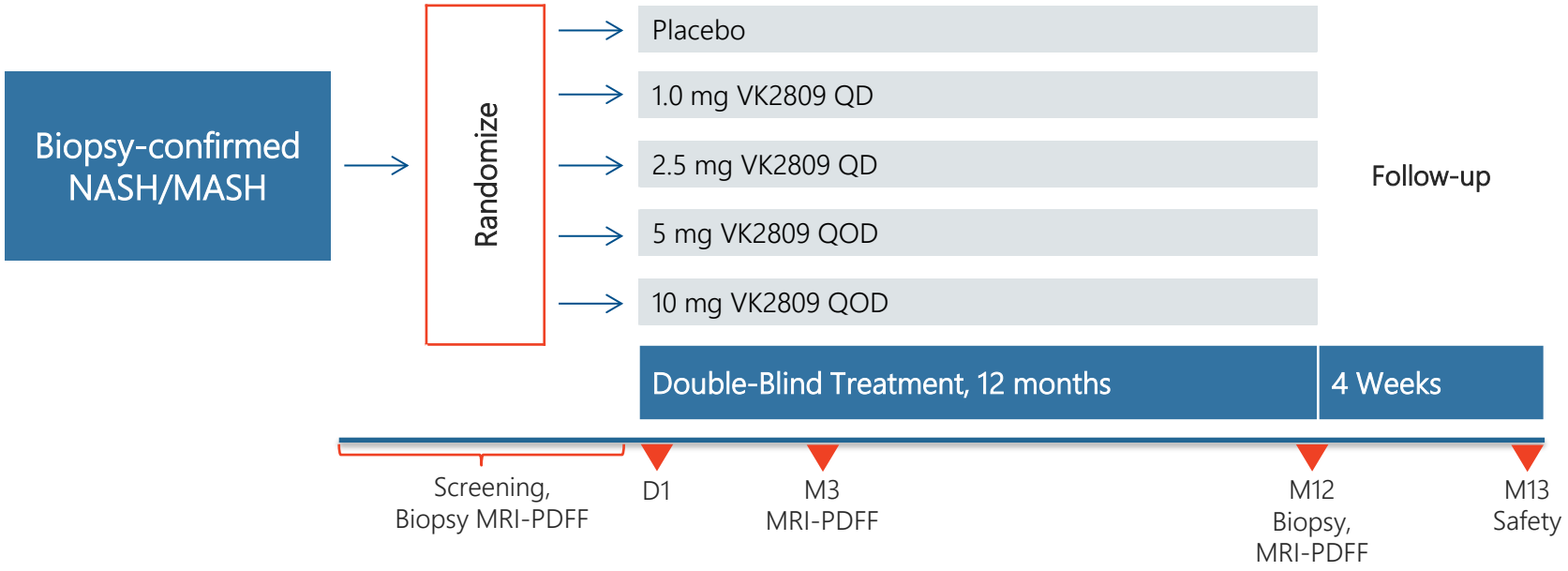


VK2809A, Potent TRβ Agonist, 2.2 nM Ki



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

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

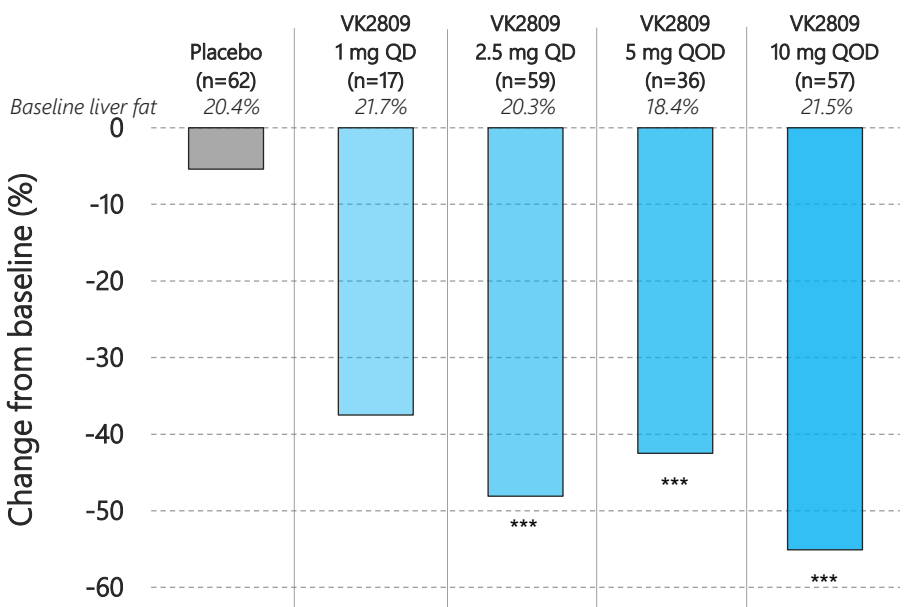


- Multi-arm, dose-ranging, 12-month Phase 2 trial
  - **Primary endpoint:** Change in MRI-PDFF vs. placebo at 3 months
  - **Secondary endpoint:** Change in histology at 12 months (NAS, fibrosis markers, etc.)

VOYAGE Study Achieves Primary Endpoint

- Significant liver fat reduction observed at 12 weeks
- Up to 57% median reduction
- Overall liver fat effect similar to prior 12-week NAFLD study
- Liver fat reductions were sustained or improved through Week 52

Median Relative % Change in Liver Fat at 12 Weeks



	Placebo (n=62)	VK2809 1 mg QD (n=17)	VK2809 2.5 mg QD (n=59)	VK2809 5 mg QOD (n=36)	VK2809 10 mg QOD (n=57)
Percent change	-5.4%	-37.5%	-49.5%	-42.5%	-56.7%
p-value vs. placebo	-	0.075	<0.0001	<0.0001	<0.0001

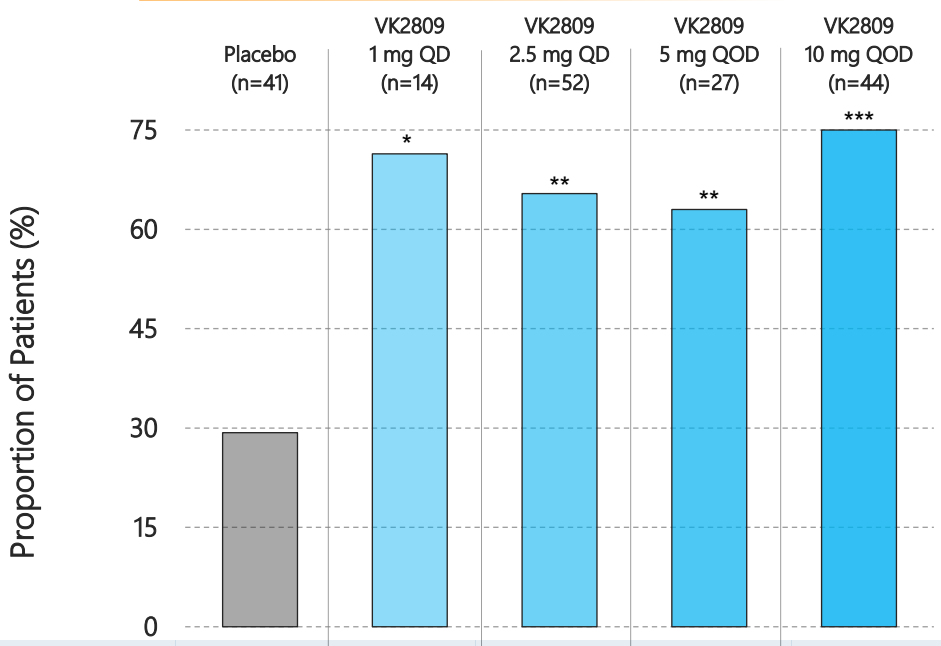
***p<0.001

VK2809 NASH Resolution Observed in up to 75% of Patients

- NASH resolution without worsening of fibrosis[1]
- Key regulatory endpoint

1) Resolution of NASH defined as NAS inflammation score of 0-1, ballooning score of 0.

Patients Demonstrating Resolution of NASH With no Worsening of Fibrosis



	Placebo (n=41)	VK2809 1 mg QD (n=14)	VK2809 2.5 mg QD (n=52)	VK2809 5 mg QOD (n=27)	VK2809 10 mg QOD (n=44)
Proportion of patients	29.3%	71.4%	65.4%	63.0%	75.0%
p-value vs. placebo	-	0.0215	0.0023	0.0091	0.0001

Notes: Includes all patients with baseline and post-baseline MRI, and week 52 biopsy.

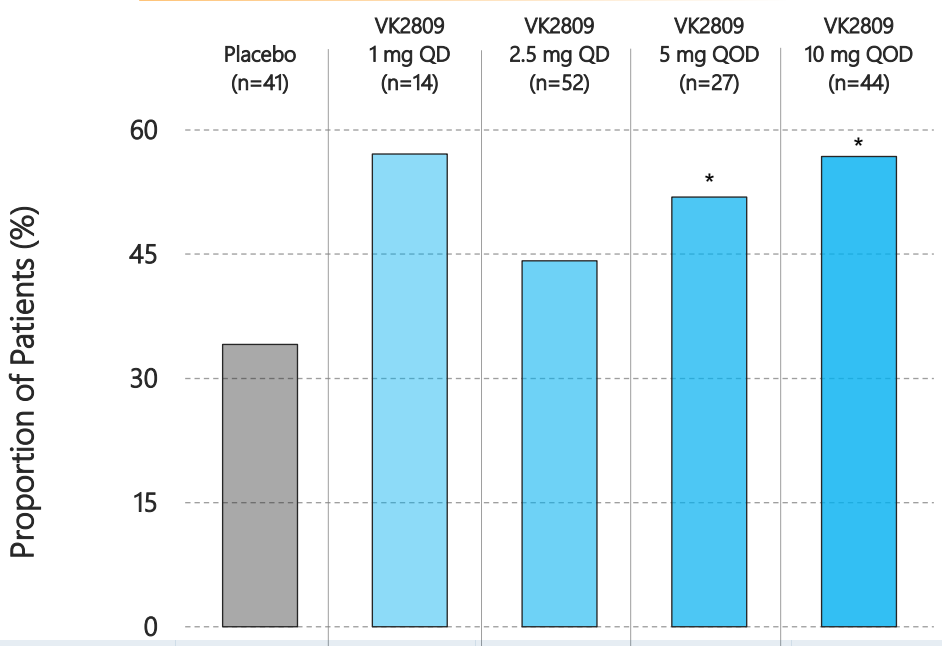
*p<0.05; **p<0.01; ***p<0.001

VK2809 Treatment Improves Fibrosis Stage

- Patients with ≥1-stage improvement in fibrosis, without worsening of NASH[1]
- Key regulatory endpoint

1) No worsening of NASH defined as no increase from baseline in ballooning, inflammation, or steatosis.

Patients Demonstrating ≥1-Stage Fibrosis Improvement, With no Worsening of NASH



	Placebo (n=41)	VK2809 1 mg QD (n=14)	VK2809 2.5 mg QD (n=52)	VK2809 5 mg QOD (n=27)	VK2809 10 mg QOD (n=44)
Proportion of patients	34.1%	57.1%	44.2%	51.9%	56.8%
p-value vs. placebo	-	0.1543	0.4414	0.0304	0.0497

Notes: Includes all patients with baseline and post-baseline MRI, and week 52 biopsy.

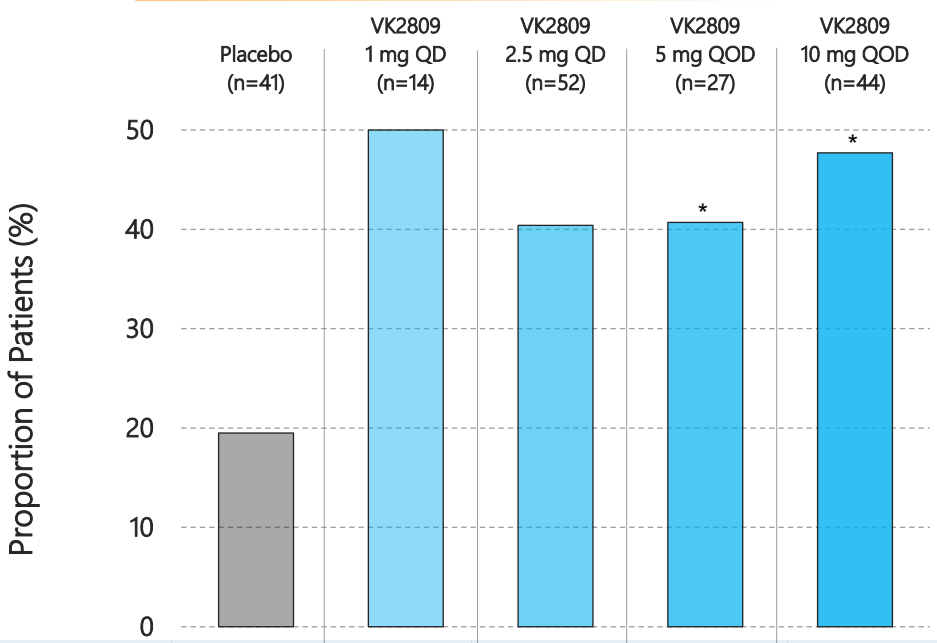
*p<0.05; **p<0.01; ***p<0.001

VOYAGE Demonstrates Fibrosis Improvement and NASH Resolution

- Patients with ≥1-stage improvement in fibrosis AND resolution of NASH[1]

1) Resolution of NASH defined as NAS inflammation score of 0-1, ballooning score of 0.

Patients Demonstrating ≥1-Stage Fibrosis Improvement and Resolution of NASH



	Placebo (n=41)	VK2809 1 mg QD (n=14)	VK2809 2.5 mg QD (n=52)	VK2809 5 mg QOD (n=27)	VK2809 10 mg QOD (n=44)
Proportion of patients	19.5%	50.0%	40.4%	40.7%	47.7%
p-value vs. placebo	-	0.0856	0.0508	0.0206	0.0115

Notes: Includes all patients with baseline and post-baseline MRI, and week 52 biopsy.

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

VK2809 Demonstrates Consistent Safety, Tolerability Profile

Most common AEs to date Number of subjects reporting (%)	Placebo (n=65)	VK2809 1 mg QD (n=17)	VK2809 2.5 mg QD (n=66)	VK2809 5.0 mg QOD (n=37)	VK2809 10.0 mg QOD (n=61)	VK2809 Combined (n=181)
Treatment emergent adverse events, TEAEs	51 (78.5%)	14 (82.4%)	55 (83.3%)	29 (78.4%)	58 (95.1%)	156 (86.2%)
Drug-related TEAEs[1]	22 (33.8%)	7 (41.2%)	13 (19.7%)	9 (24.3%)	24 (39.3%)	53 (29.3%)
TEAEs leading to discontinuation	6 (9.2%)	2 (11.8%)	1 (1.5%)	2 (5.4%)	6 (9.8%)	11 (6.1%)
Drug-related GI adverse events	12 (18.5%)	4 (23.5%)	3 (4.5%)	1 (2.7%)	7 (11.5%)	15 (8.3%)
Nausea	5 (7.7%)	2 (11.8%)	2 (3.0%)	1 (2.7%)	3 (4.9%)	8 (4.4%)
Diarrhea	2 (3.1%)	3 (17.6%)	2 (3.0%)	1 (2.7%)	3 (4.9%)	9 (5.0%)

Notes: Study safety population, defined as all patients who were randomized and received at least one dose of study drug. 1) Deemed by investigator as possibly, probably, or definitely related to study drug.

- Majority of TEAEs (97%) mild or moderate
- Discontinuations due to AEs well balanced between placebo, treatment groups
- GI-related AEs similar to placebo

VK2809 VOYAGE Takeaways

- Achieves primary endpoint demonstrating robust reduction in liver fat at 12 weeks
- Histologic endpoints demonstrate NASH resolution, improvement in fibrosis, and combination of both at 52 weeks
- Significant reductions in plasma lipids LDL-C, triglycerides, Lp(a), ApoB, ApoC-III
- Excellent tolerability, rate of GI-related side effects similar to placebo
- Promising safety, 94% of AEs mild to moderate

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
Reduces liver fat, resolves NASH, improves fibrosis	Potential to be best-in-class, small molecule TRβ therapeutic for NASH/MASH
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

VIKING THERAPEUTICS

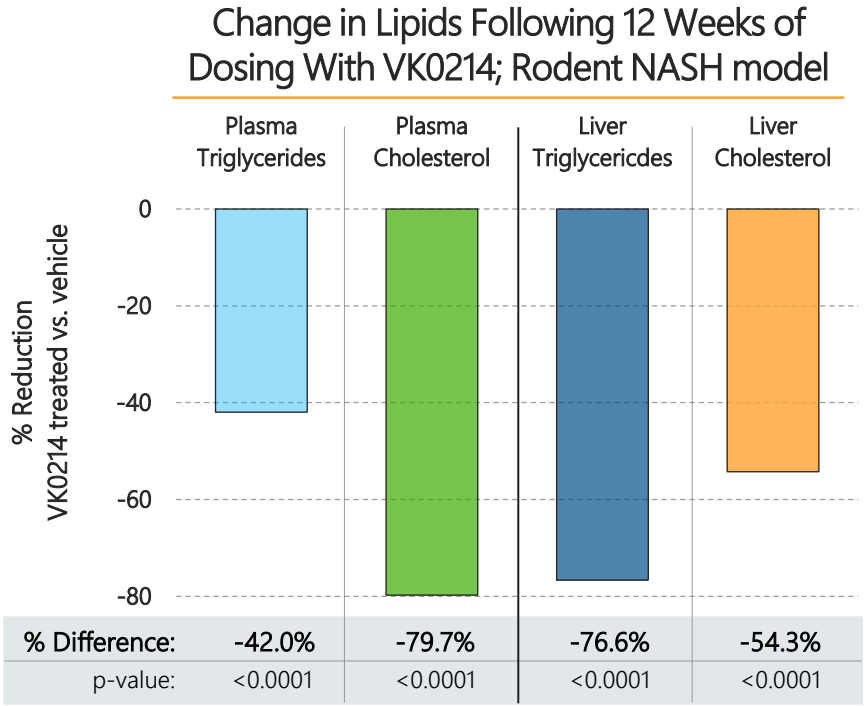
VK0214: Selective Thyroid Receptor-β Agonist

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

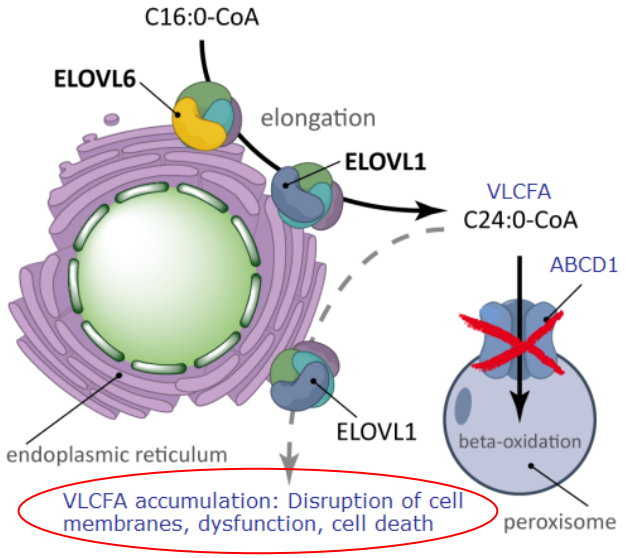


Demonstrates in vitro and vivo efficacy comparable to VK2809

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

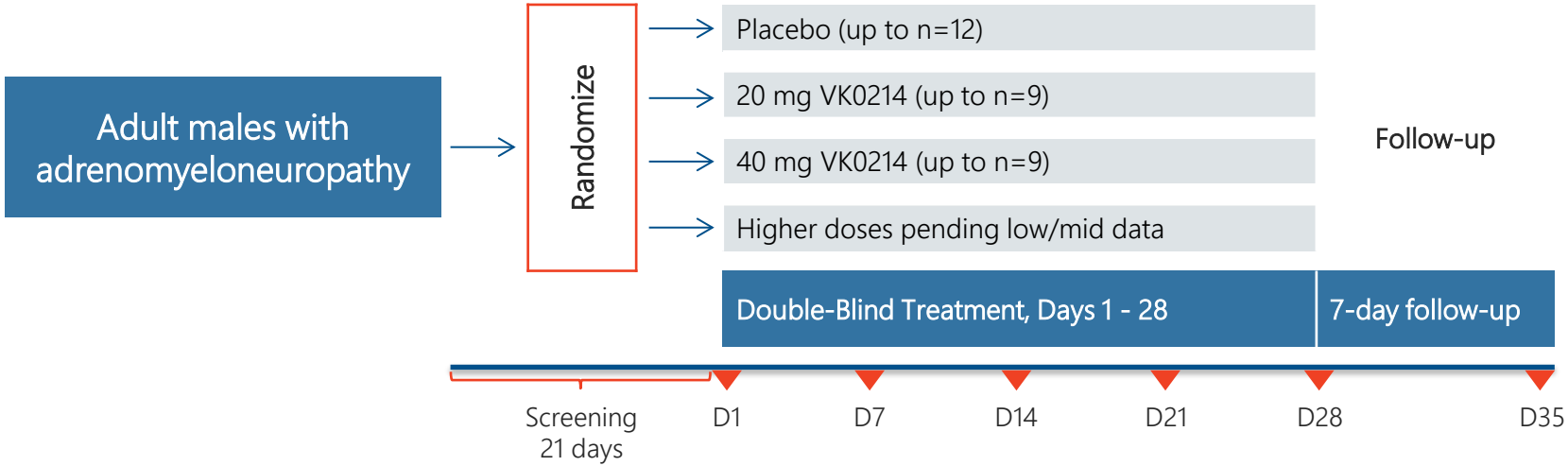
X-ALD: Defective ABCD1 leads to accumulation of VLCFA in tissues

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/.

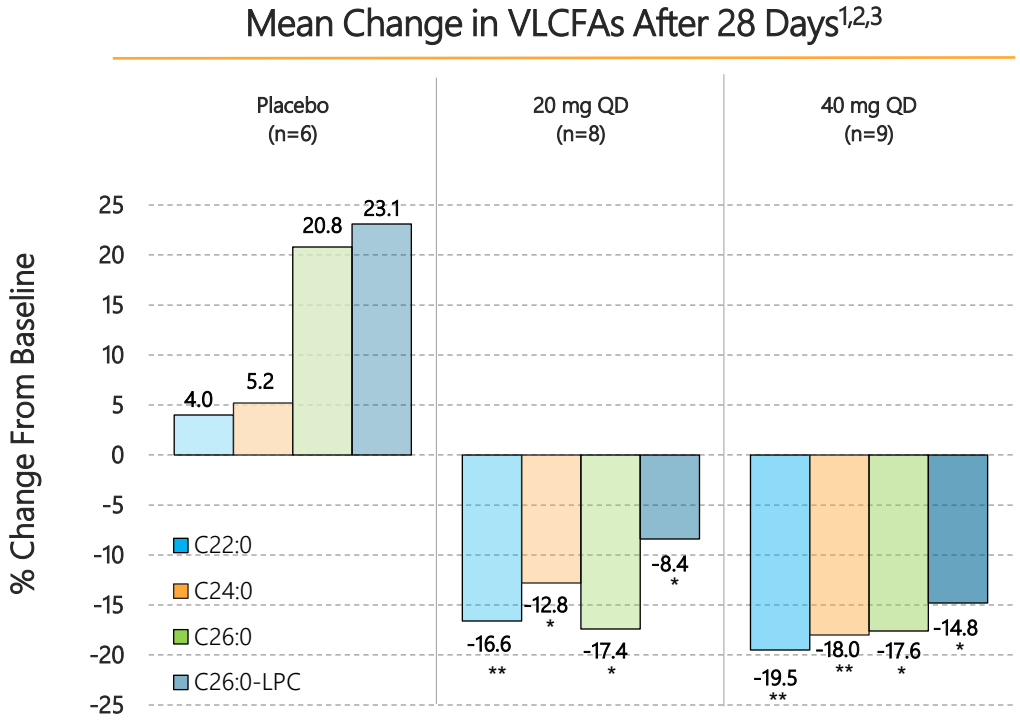
VK0214 Phase 1b Study in Adrenomyeloneuropathy



- 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

VK0214 Phase 1b Results: Significant Reductions of Plasma VLCFAs

- Significant reductions in mean VLCFA levels compared to placebo
- Reductions in mean plasma levels of 26 carbon lysophosphatidyl choline (C26:0-LPC) derivative, a key diagnostic marker



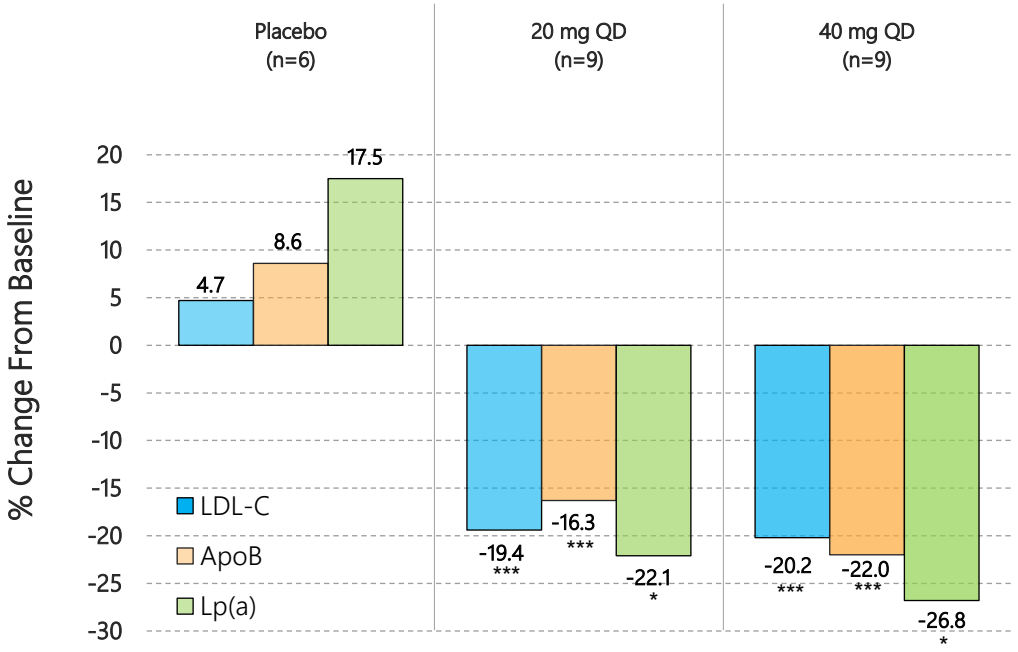
1) Least squares mean change from baseline to Day 28. 2) P-value vs. placebo: Two-sided t-test using mixed model for repeated measures. 3) C26:0-LPC data for 20 mg, 40 mg cohorts include results from n=7, n=8 subjects, respectively.

p-values vs. placebo *p<0.05; **p<0.01; ***p<0.001

VK0214 Phase 1b Results: Significant Reductions of Lipid Markers

- Statistically significant reductions vs. placebo for both doses of VK0214
- Important implications for long-term cardiometabolic benefits

Mean Change in Lipid Markers After 28 Days[1,2]



1) Least squares mean change from baseline to Day 28. 2) P-value vs. placebo: Two-sided t-test using mixed model for repeated measures.

p-values vs. placebo *p<0.05; **p<0.01; ***p<0.001

VK0214 Phase 1b Study: Discontinuations and Adverse Events

Number of subjects reporting (%)	Placebo (n=6)	VK0214 20 mg (n=9)	VK0214 40 mg (n=9)	VK0214 Combined (n=18)
Discontinued treatment early	0 (0%)	1 (11%)	1 (11%)	2 (11%)
Discontinued study early	0 (0%)	1 (11%)	0 (0%)	1 (6%)
Overall TEAEs	3 (50%)	7 (78%)	8 (89%)	15 (83%)
Drug related TEAEs	1 (17%)	5 (56%)	5 (56%)	10 (56%)
Serious adverse events	0 (0%)	0 (0%)	0 (0%)	0 (0%)

Note: Study safety population, defined as all patients who were randomized and received at least one dose of study drug.

- No SAEs reported
- Discontinuation rates low; treatment emergent adverse events (TEAEs) reported as mild to moderate

VK0214 Phase 1b Study: Tolerability Summary

Common GI related TEAEs Number of subjects reporting (%)	Placebo (n=6)	VK0214 20 mg (n=9)	VK0214 40 mg (n=9)	VK0214 Combined (n=18)
All GI disorders				
Mild	2 (33%)	2 (22%)	0 (0%)	2 (11%)
Moderate	0 (%)	0 (%)	0 (%)	0 (%)
Severe	0 (%)	0 (%)	0 (%)	0 (%)
Upper abdominal pain	1 (17%)	0 (0%)	0 (0%)	0 (0%)
Constipation	0 (0%)	1 (11%)	0 (0%)	1 (6%)
Dyspepsia	1 (17%)	0 (0%)	0 (0%)	0 (0%)
Nausea	0 (0%)	1 (11%)	0 (0%)	1 (6%)

Note: Study safety population, defined as all patients who were randomized and received at least one dose of study drug.

- GI adverse events slightly higher among placebo (33%) vs. VK0214 (11%)
- Consistent with VK2809 experience; excellent overall tolerability though small n

Takeaways From VK0214 Phase 1b in AMN Patients Study

- Patients receiving VK0214 demonstrated progressive improvement in plasma levels of very long chain fatty acids (VLCFAs) in the relatively brief treatment period evaluated in this study (28 days)
- VK0214 continued to show benefits on broader plasma lipids, such as LDL-C, important for overall cardiometabolic health
- Consistent with prior clinical results in healthy volunteers, VK0214 was shown to be safe and well-tolerated in this 28-day study

VK0214 – Summary and Current Status

- Potential to be best in-class oral, small molecule TRβ therapeutic for X-ALD
- Encouraging *in vivo* efficacy with rapid (6 weeks) and progressive (up to 25 weeks) VLCFA reductions in plasma, brain, spinal cord and liver
- Phase 1 data in healthy volunteers demonstrated promising safety, lipid-lowering effects
- Phase 1b proof-of-concept study in adults with AMN showed significant VLCFA and lipids reductions in plasma after 28 days of treatment
- VK0214 has received Orphan Drug status from the FDA

Financial Summary

- Capital structure and summary financials

Capital Structure	December 31, 2024 ('000s)
Shares outstanding	111,574
Options, RSUs	7,455
Total shares, options, RSUs	119,029

Financials	December 31, 2024 ('000s)
Cash burn YTD	$199,312
Cash and ST Investments	$902,612

Investment Highlights

- Developing novel therapeutics for metabolic and endocrine diseases
  - Multiple clinical programs demonstrate best-in-class efficacy data
- Metabolic Disease Programs
  - VK2735: GLP-1/GIP dual agonist for obesity
    - VENTURE Phase 2 obesity study successfully achieved primary endpoint; Phase 3 planned 1H25
  - VK2735 Oral: GLP-1/GIP dual agonist for obesity
    - Phase 1 study demonstrated positive PoC, reduction in body weight; Phase 2 underway
  - VK2809: Selective thyroid receptor-β agonist for NASH/MASH
    - VOYAGE Phase 2b trial successfully primary, secondary endpoints; presented at AASLD 4Q24
- Rare Disease Program
  - VK0214: Selective thyroid receptor-β agonist for X-ALD
    - Phase 1b in patients demonstrated PoC in reducing key biomarkers of disease

VIKING THERAPEUTICS

Corporate Presentation

February 2025