Transforming the Care of Cardiovascular Disease Through Single-course Gene Editing Medicines

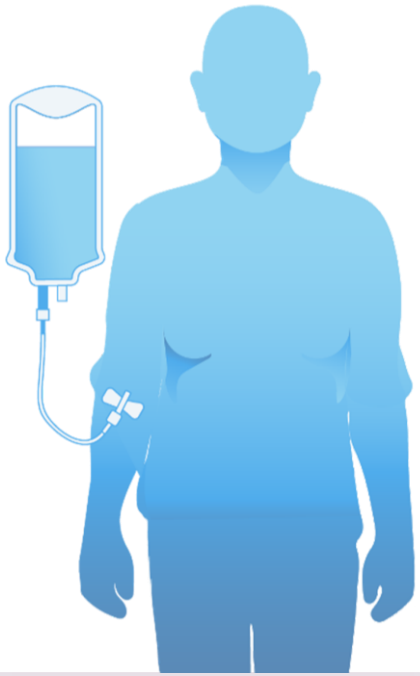
MAY 2025

Forward looking statements and disclaimers

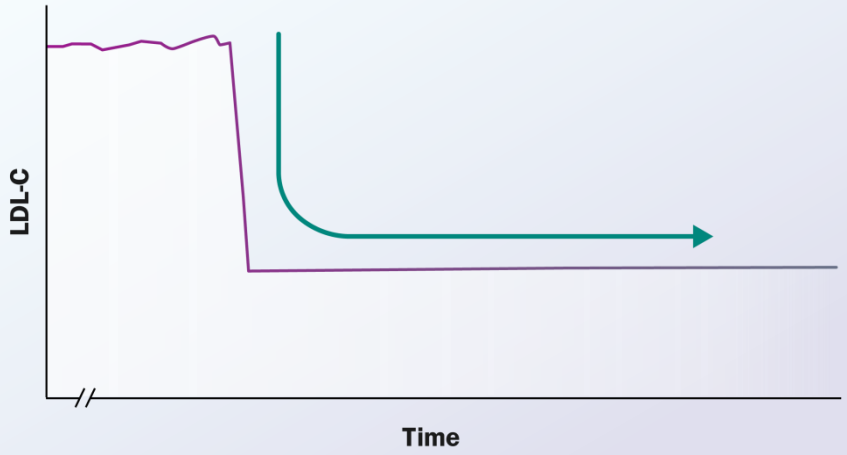
This presentation contains “forward-looking statements” within the meaning of the Private Securities Litigation Reform Act of 1995 that involve substantial risks and uncertainties, including statements regarding the Company’s ongoing Heart-2 clinical trial; the timing and availability of data for the Heart-2 trial and timing for initiation of a Phase 2 clinical trial for VERVE-102; the timing for delivery of the opt-in package and of Eli Lilly and Company’s decision for the PCSK9 program; the timing of updates for the ANGPTL3 program; the Company’s strategic plans and prospects; the potential advantages and therapeutic potential of the Company’s programs; market opportunity estimates or projections; and the period over which the Company believes that its existing cash, cash equivalents and marketable securities will be sufficient to fund its operating expenses. All statements, other than statements of historical facts, contained in this presentation, including statements regarding the Company’s strategy, future operations, future financial position, prospects, plans and objectives of management, are forward-looking statements. The words “anticipate,” “believe,” “continue,” “could,” “estimate,” “expect,” “intend,” “may,” “plan,” “potential,” “predict,” “project,” “should,” “target,” “will,” “would” and similar expressions are intended to identify forward-looking statements, although not all forward-looking statements contain these identifying words. Any forward-looking statements are based on management’s current expectations of future events and are subject to a number of risks and uncertainties that could cause actual results to differ materially and adversely from those set forth in, or implied by, such forward-looking statements. These risks and uncertainties include, but are not limited to, risks associated with the Company’s limited operating history; the Company’s ability to timely submit and receive approvals of regulatory applications for its product candidates; advance its product candidates in preclinical studies and clinical trials; initiate, enroll and complete its ongoing and future clinical trials on the timeline expected or at all; correctly estimate the potential patient population and/or market for the Company’s product candidates; replicate in clinical trials positive results found in preclinical studies and/or earlier-stage clinical trials of VERVE-101, VERVE-102 and VERVE-201; advance the development of its product candidates under the timelines it anticipates in preclinical studies and in current and future clinical trials; obtain, maintain or protect intellectual property rights related to its product candidates; manage expenses; and raise the substantial additional capital needed to achieve its business objectives. For a discussion of other risks and uncertainties, and other important factors, any of which could cause the Company’s actual results to differ from those contained in the forward-looking statements, see the “Risk Factors” section, as well as discussions of potential risks, uncertainties and other important factors, in the Company’s most recent filings with the Securities and Exchange Commission and in other filings that the Company makes with the Securities and Exchange Commission in the future. In addition, the forward-looking statements included in this presentation represent the Company’s views as of the date hereof and should not be relied upon as representing the Company’s views as of any date subsequent to the date hereof. The Company anticipates that subsequent events and developments will cause the Company’s views to change. However, while the Company may elect to update these forward-looking statements at some point in the future, the Company specifically disclaims any obligation to do so.

Verve's vision: a one dose future to address chronic disease

Single IV Infusion of Verve Product



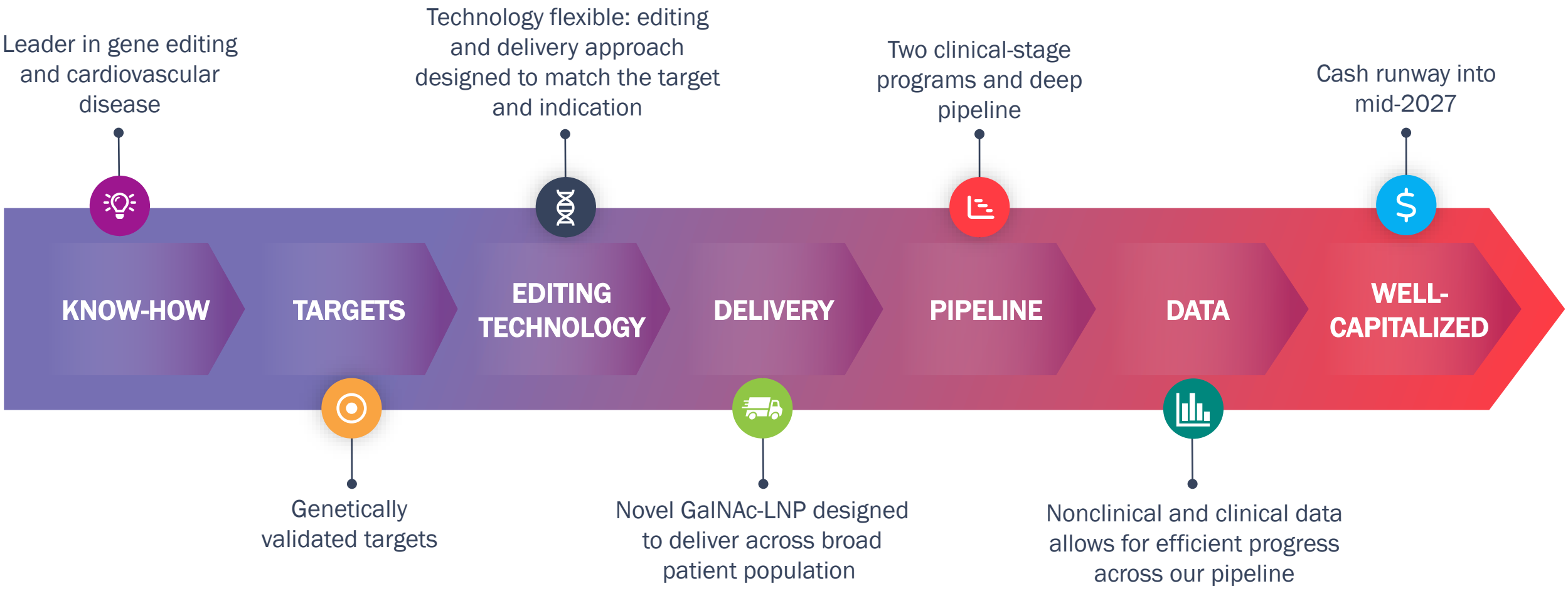
Lifelong Blood Cholesterol Reduction



VERVE MISSION

Deliver enduring efficacy to patients living with or at high risk for ASCVD

Transforming the treatment of cardiovascular disease (CVD) from chronic care to once-and-done



ASCVD and HeFH:
highly prevalent diseases with profound need for improved treatments

Atherosclerotic Cardiovascular Disease (ASCVD)

What is ASCVD?

Build-up of cholesterol-driven deposits in artery walls that restricts healthy blood flow and can cause heart disease, stroke, and peripheral vascular disease

>30M

Patients with ASCVD not at LDL-C goal in U.S. + EU[1,2]

~75%

ASCVD patients are not at LDL-C goal[2]

ASCVD is the leading cause of death in the world

Heterozygous Familial Hypercholesterolemia (HeFH)

What is HeFH?

Inherited disease characterized by high levels of LDL-C [LDL-C ≥ 190 mg/dL] that frequently results in early-onset ASCVD

>3M

Patients with HeFH in U.S. + EU[3]

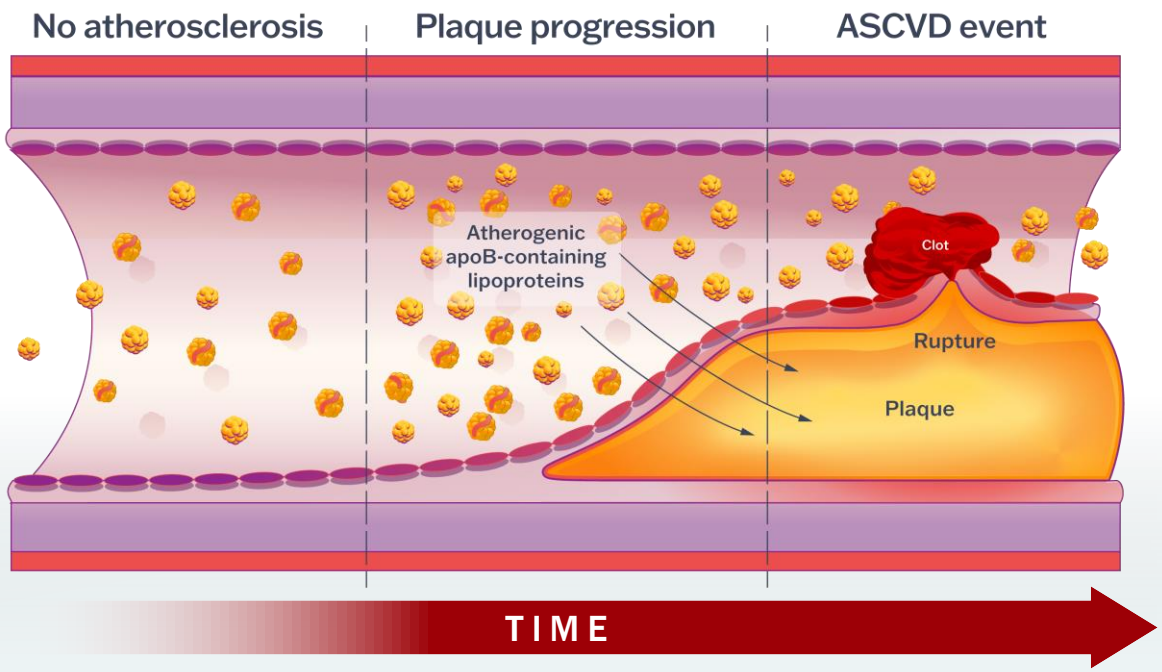
97%

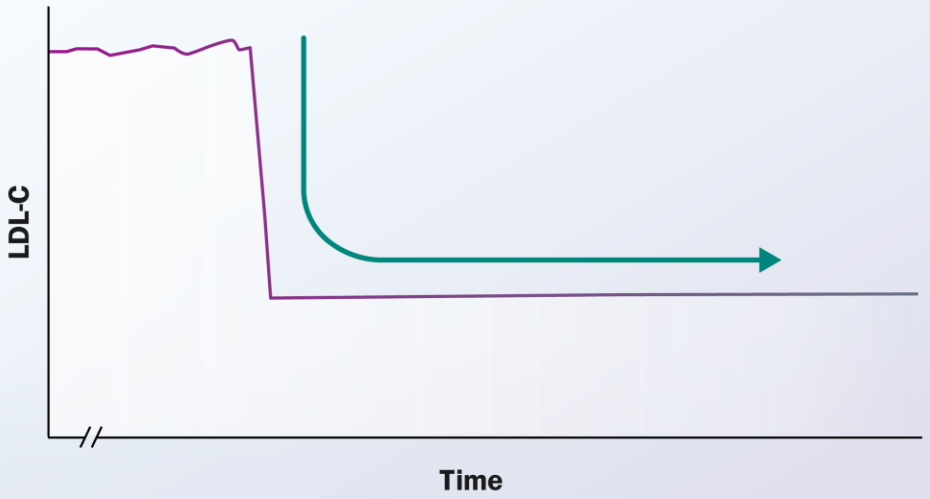
HeFH patients are not at LDL-C goal[4]

HeFH is the most prevalent genetic disease in humans

1. Ray KK et al. European Journal of Preventive Cardiology 2021. 2. Gu J et al. Am J Prev Cardiol 2022. 3. de Ferranti et al. Circulation 2016. 4. Vallejo-Vaz et al. Lancet 2021
LDL-C, low-density lipoprotein cholesterol

Treatment and prevention of ASCVD:
keep blood cholesterol as low as possible for as long as possible

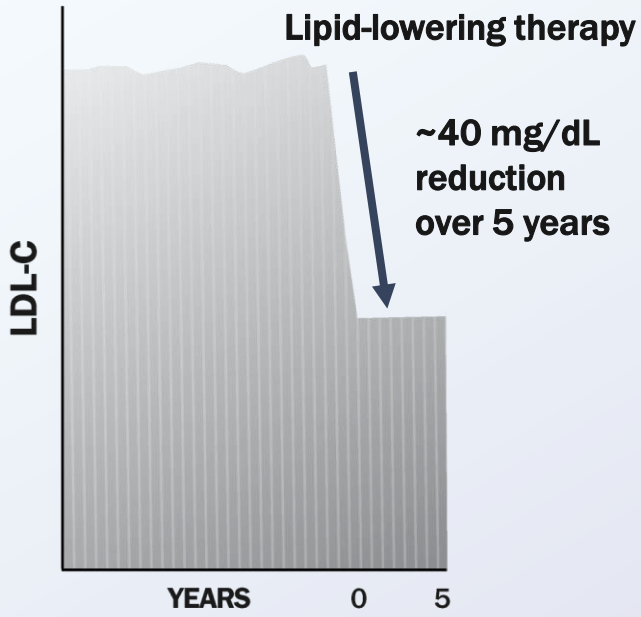




apoB, apolipoprotein B; LDL-C, low-density lipoprotein cholesterol

Significant unmet need: today's chronic care approach leads to transient LDL-C reduction and inadequate efficacy

Today's Approach:
Transient LDL-C Reduction, Inadequate Efficacy



21% Major CVD Event Reduction[1]

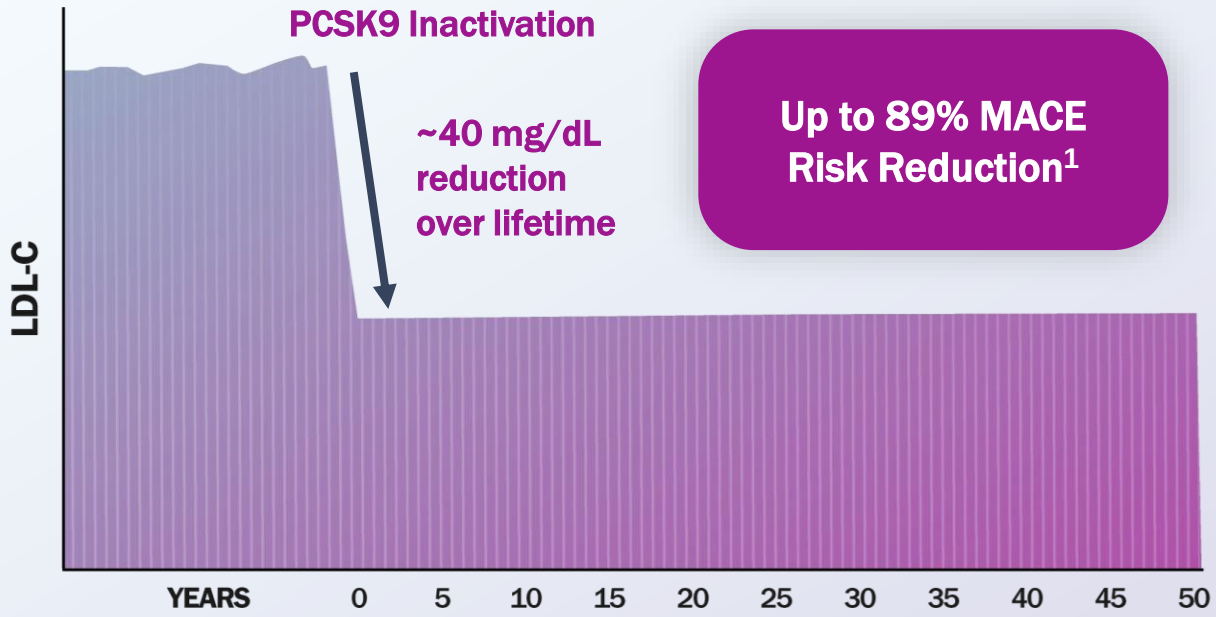
Learnings from lipid lowering trials:*
5 years of consistent LDL-C lowering reduces major cardiovascular event risk by 21%

Efficacy is Compromised by Frequent Discontinuation

	PCSK9 siRNA	PCSK9 mAb
Dosing	Injection every 6 months	Injection every 2 weeks
1-year Patient Discontinuation Rate[2]	~1 in 5	~1 in 2
Estimated Real-world LDL-C Reduction at 1 year[3]	- 35%	- 23%

*In 28 trials evaluating efficacy and safety of statin therapy. 1. Cholesterol Treatment Trialists' (CTT) Collaboration. Lancet 2019. 2. Popadic et al. Journal of Clinical Lipidology 2024. 3. Rosenson et al. 2025 ACC. Poster #12209
LDL-C, low-density lipoprotein cholesterol; CVD, cardiovascular disease; PCSK9, proprotein convertase subtilisin/kexin type 9; siRNA, small interfering RNA; mAb, monoclonal antibody

Solution: a treatment approach that can provide **enduring efficacy**; 40 mg/dL reduction over a lifetime = dramatic ASCVD risk reduction



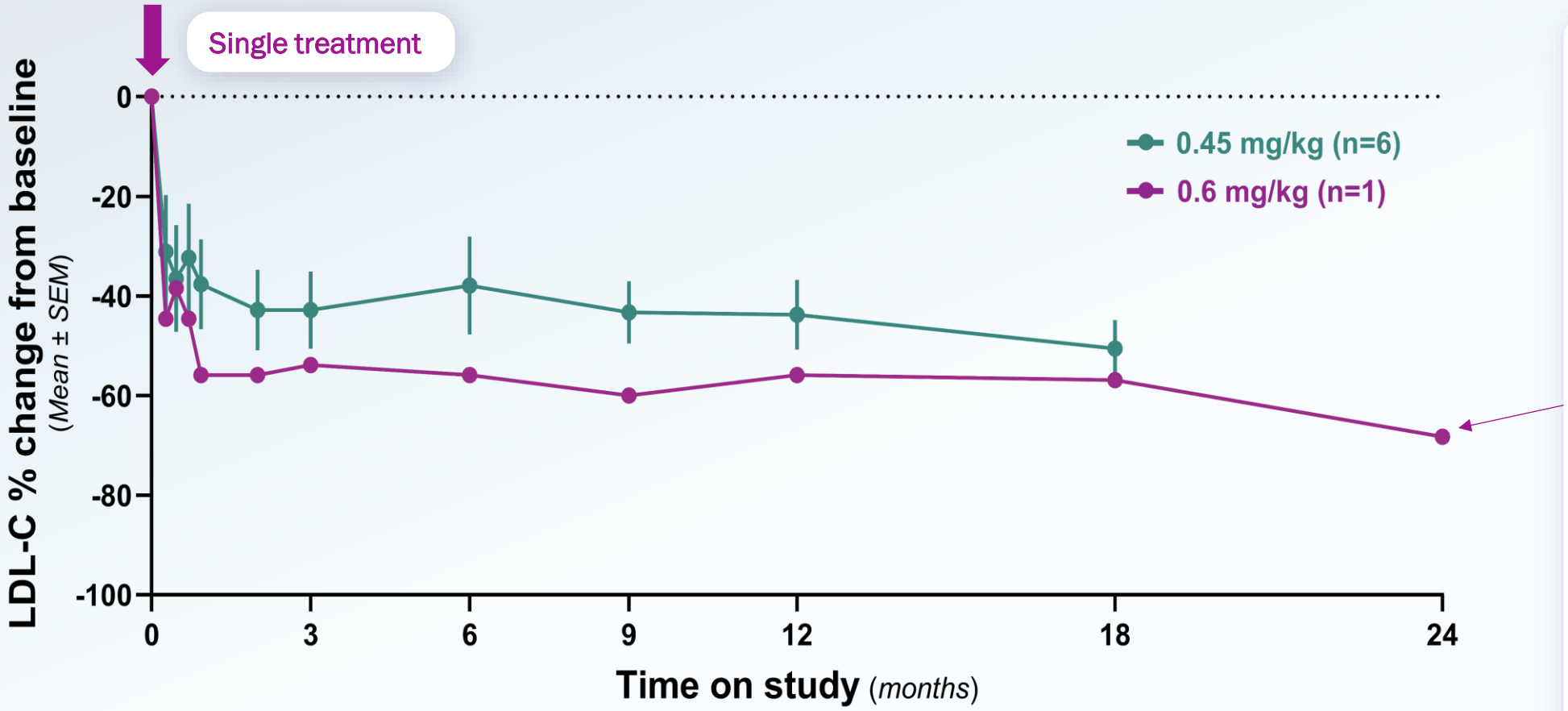
Lifetime of lowered LDL-C <u>safely</u> reduces MACE risk by up to 89%

Benefit of LDL-C lowering accrues over time and is maximized when started early and durably maintained

1. Cohen et al. N Engl J Med 2006

LDL-C, low-density lipoprotein cholesterol; PCSK9, proprotein convertase subtilisin/kexin type 9; MACE, major adverse cardiac events

VERVE-101 data provides proof of concept that base editing mechanism may deliver enduring efficacy: 2 years after treatment, time-averaged LDL-C reduction of 58% for single participant



Patient diagnosed with HeFH who had suffered heart attack before age 30

Post VERVE-101 treatment, has been at LDL-C goal for 2 years

Data from parent Heart-1 study as of February 27, 2025. Data from long-term follow-up study as of March 26, 2025. Data are from ongoing studies with open databases that have not been fully cleaned. Participants in 0.45 mg/kg cohort have variable duration of follow up, with n=2 at 18 months and n=4 at 12 months. Select time points were impacted for two participants in the 0.45 mg/kg cohort due to changes in background lipid-lowering therapy.
LDL-C, low-density lipoprotein cholesterol; SEM, standard error of the mean

Verve is advancing a pipeline of *in vivo* gene editing programs designed to lower cholesterol lifelong after a single treatment

TARGET	INDICATION	TECHNOLOGY	RESEARCH	IND-ENABLING	CLINICAL
PCSK9[1] (VERVE-102)	Heterozygous familial hypercholesterolemia	Base Editor (GalNAc-LNP)			
	ASCVD				
ANGPTL3[1] (VERVE-201)	Homozygous familial hypercholesterolemia	Base Editor (GalNAc-LNP)			
	Refractory hypercholesterolemia				
LPA[2] (VERVE-301)	ASCVD patients with high blood Lp(a)	Novel Editor (GalNAc-LNP)			
Undisclosed	Undisclosed ASCVD	Novel Editor			
Undisclosed	Undisclosed liver disease	Novel Editor			

1. Under collaboration with Eli Lilly and Company; Verve-led development, with Lilly option for cost and US margin share.
2. Under collaboration with Eli Lilly and Company; Verve-led development through Phase 1, with Verve option for cost and margin share.

Verve's pipeline of gene editing programs is designed to address distinct groups of patients with ASCVD

All atherosclerotic cardiovascular disease (ASCVD) *~51M in US/EU*

VERVE-102	VERVE-102	VERVE-201	VERVE-201	VERVE-301
HeFH[2]	ASCVD not at LDL-C goal[1,3]	HoFH[4]	Refractory-hypercholesterolemia[5] *(ASCVD not at LDL-C goal on maximum standard of care)*	Elevated Lp(a)[6]
> 3M in US/EU	**> 30M** in US/EU	**> 3,000** in US/EU	**~4M** in US/EU	**~13M** in US/EU (~25% ASCVD)

ASCVD, atherosclerotic cardiovascular disease; HeFH, heterozygous familial hypercholesterolemia; HoFH, homozygous familial hypercholesterolemia; Lp(a), lipoprotein(a)

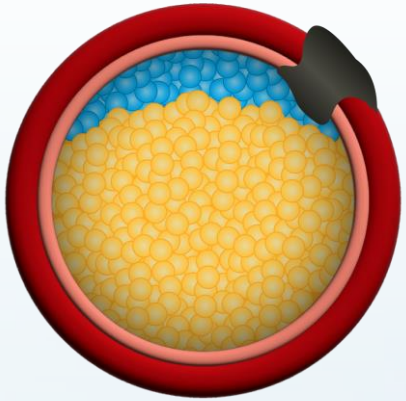
1. Gu J et al. Am J Prev Cardiol 2022;10: 100336; Ray KK et al. European Journal of Preventive Cardiology 2021; 28: 1279–1289; Townsend et al. Nature Reviews Cardiology 2022; 19: 133-143; 2. de Ferranti et al. Circulation 2016;133: 1067-72; 3. Gu J et al., Am J Prev Cardiol 2022;10: 100336; Ray KK et al., European Journal of Preventive Cardiology 2021;28: 1279–1289; 4. Cuchel M et al. Eur Heart J 2023; 44: 2277-2291; 5. O'Donoghue ML et al. Circulation 2022; 146: 1109-1119; 6. Nissen SE et al. Open Heart 2022;9:e002060.

The three cholesterol drivers of atherosclerosis — LDL, TRL, and Lp(a) — are addressed by VERVE-102 (PCSK9), VERVE-201 (ANGPTL3), and VERVE-301 (LPA)

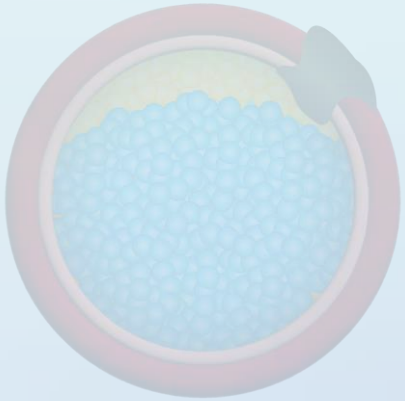
LDL, low-density lipoprotein; TRL, triglyceride-rich lipoprotein; Lp(a), lipoprotein(a); PCSK9, proprotein convertase subtilisin/kexin type 9; ANGPTL3, angiopoietin-like protein 3

PCSK9 program: VERVE-102

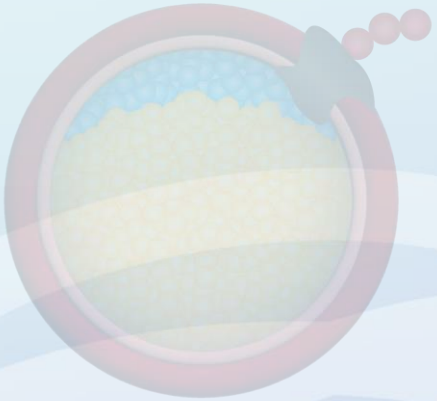
LDL



TRL



Lp(a)



VERVE-102 (PCSK9)

VERVE-201 (ANGPTL3)

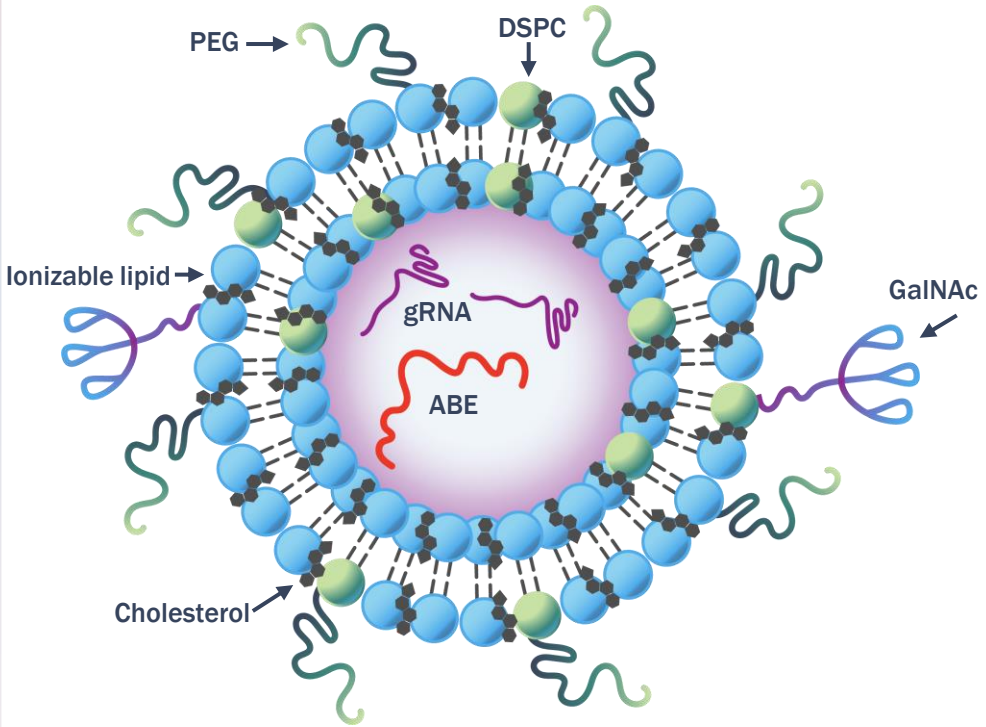
VERVE-301 (LPA)

Cholesterol Triglyceride *Apolipoprotein B* *Apolipoprotein(a)*

LDL, low-density lipoprotein; PCSK9, proprotein convertase subtilisin/kexin type 9

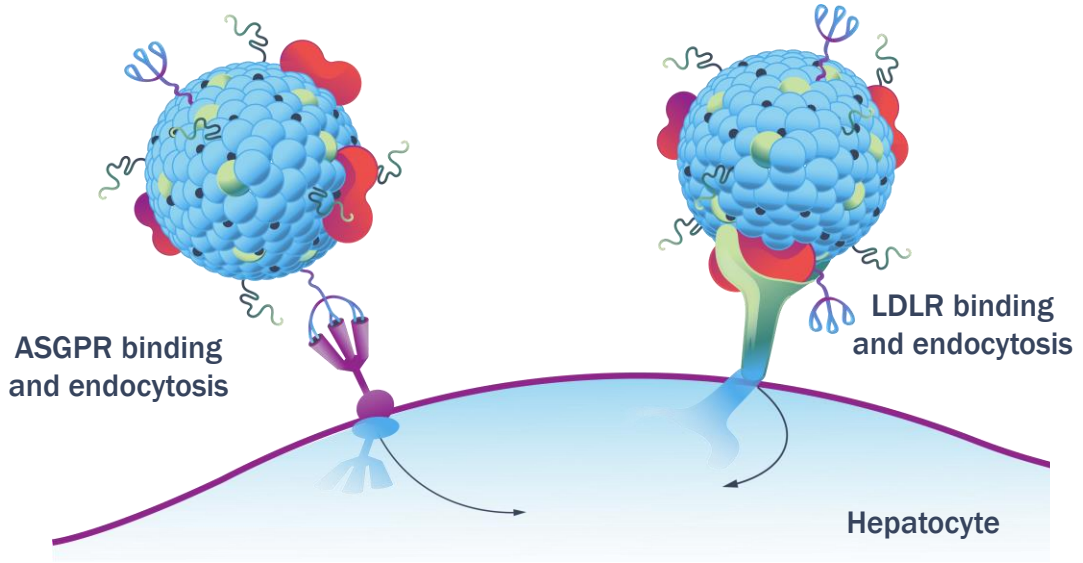
VERVE-102 is an investigational *in vivo* base editing medicine that is delivered by a GalNAc-LNP and is designed to inactivate *PCSK9*

VERVE-102



Role of the GalNAc Ligand

After IV infusion of the GalNAc-LNP, VERVE-102 enters hepatocytes through LDLR or ASGPR



LDLR, low-density lipoprotein receptor; LNP, lipid nanoparticle; PEG, polyethylene glycol; DSPC, 1,2-distearoyl-sn-glycero-3-phosphocholine; gRNA, guide RNA; ABE, adenine base editor; IV, intravenous; ASGPR, asialoglycoprotein receptor

Heart-2: Phase 1b clinical trial designed to evaluate safety and tolerability of VERVE-102 (PCSK9)

Population: HeFH and/or premature coronary artery disease patients who require additional LDL-C lowering

heart-2

Intervention:
Single dose administered as a 2- to 4-hour peripheral intravenous infusion[1]

Phase 1b
Single ascending dose, n = 3 – 9 per cohort

- 0.3 mg/kg
- 0.45 mg/kg
- 0.6 mg/kg
- 0.7 mg/kg

Phase 2*
Expect first patient dosed H2 2025

Doses selected based on findings from Phase 1

Objectives:

- Primary: evaluate safety and tolerability
- Secondary: measure changes in blood PCSK9 and LDL-C levels

* Subject to regulatory clearance. [1]Pre-medication with dexamethasone and antihistamines.
HeFH, heterozygous familial hypercholesterolemia; LDL-C, low-density lipoprotein cholesterol; PCSK9, proprotein convertase subtilisin/kexin type 9. NCT06164730

Initial update: safety and pharmacodynamic data from 14 participants across first three cohorts of Heart-2 trial

Population: HeFH and/or premature coronary artery disease patients who require additional LDL-C lowering

heart-2

Intervention:
Single dose administered as a 2- to 4-hour peripheral intravenous infusion[1]

Phase 1b
Single ascending dose, n = 3 – 9 per cohort

- 0.3 mg/kg (n=4)
- 0.45 mg/kg (n=6)
- 0.6 mg/kg (n=4)
- 0.7 mg/kg

Initial data from first 14 participants dosed across first three cohorts with ≥ 28 days of follow up

Phase 2*
Expect first patient dosed H2 2025

Doses selected based on findings from Phase 1

Objectives:

- Primary: evaluate safety and tolerability
- Secondary: measure changes in blood PCSK9 and LDL-C levels

As of March 13, 2025, data cut off date
*Subject to regulatory clearance. [1]Pre-medication with dexamethasone and antihistamines.
HeFH, heterozygous familial hypercholesterolemia; LDL-C, low-density lipoprotein cholesterol; PCSK9, proprotein convertase subtilisin/kexin type 9. NCT06164730

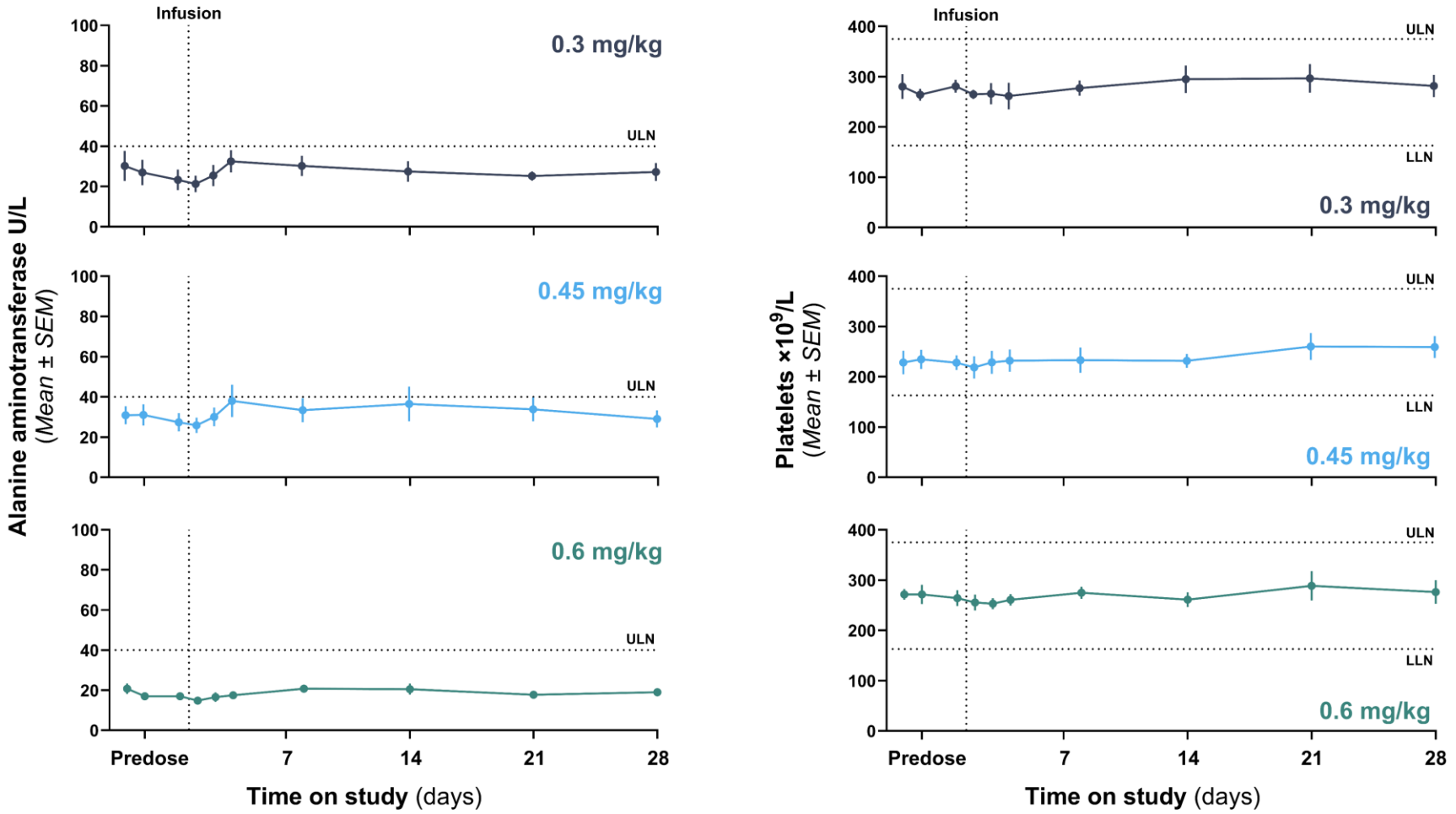
verve THERAPEUTICS

Clinical safety: VERVE-102 was well-tolerated across all dose levels

- No treatment-related SAEs and no DLTs
- No clinically significant laboratory abnormalities
- No cardiovascular events
- Across all 14 participants, there was one infusion-related reaction (IRR)
  - Single Grade 2 IRR in a participant dosed at 0.6 mg/kg (transient symptoms that resolved with acetaminophen)

As of March 13, 2025, data cut off date; data are from an ongoing study with an open database and have not been fully cleaned.
SAE, serious adverse event; DLT, dose-limiting toxicity

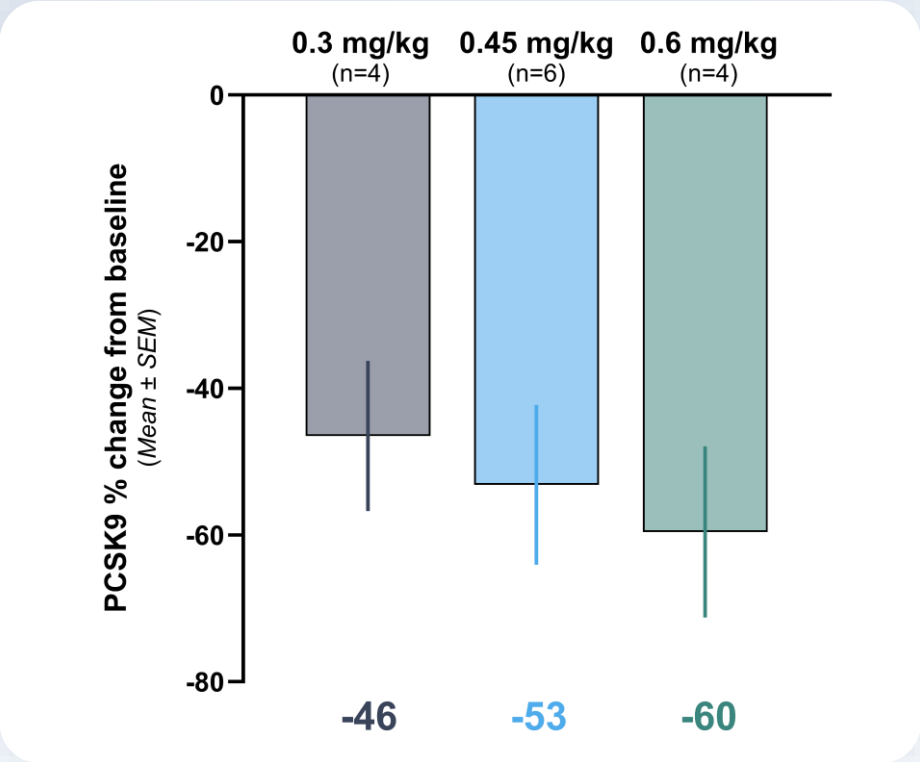
Laboratory assessment: no clinically significant changes in alanine aminotransferase (ALT) or platelets at any dose level following VERVE-102 infusion



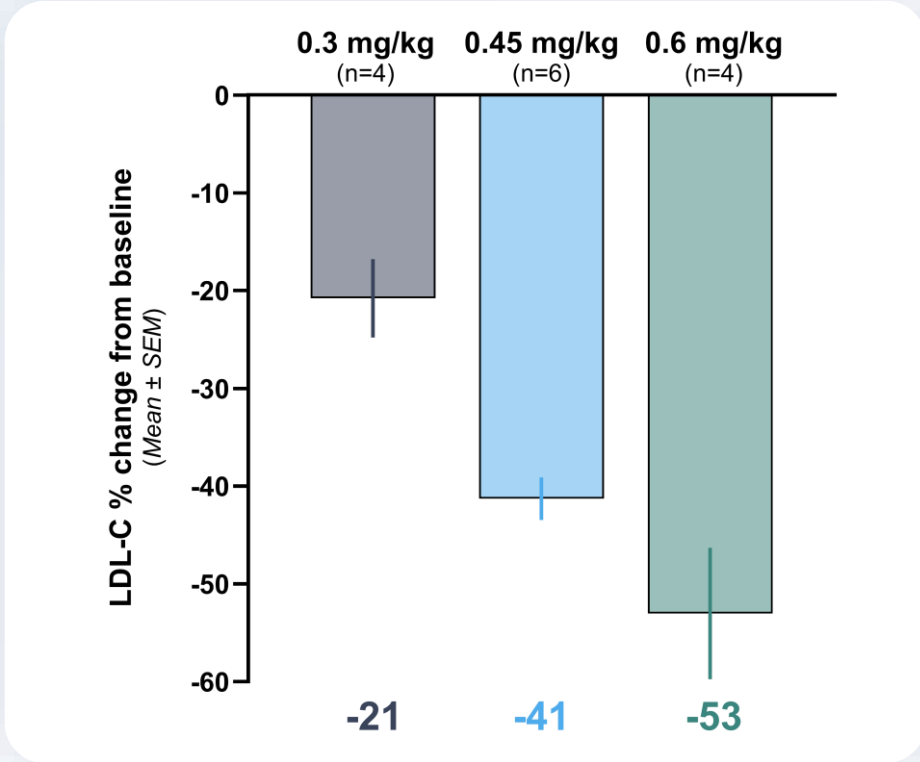
As of March 13, 2025, data cut off date; predose values include screening visits and day of infusion before dosing; ULN indicated for ALT is value for males (female ULN is 33 U/L).
LLN, lower limit of normal; SEM, standard error of the mean; ULN, upper limit of normal

Single infusion of VERVE-102 led to dose-dependent reductions in blood PCSK9 protein and blood LDL-C

PCSK9 protein reduction:
mean reduction of 60% observed in highest weight-based dose group



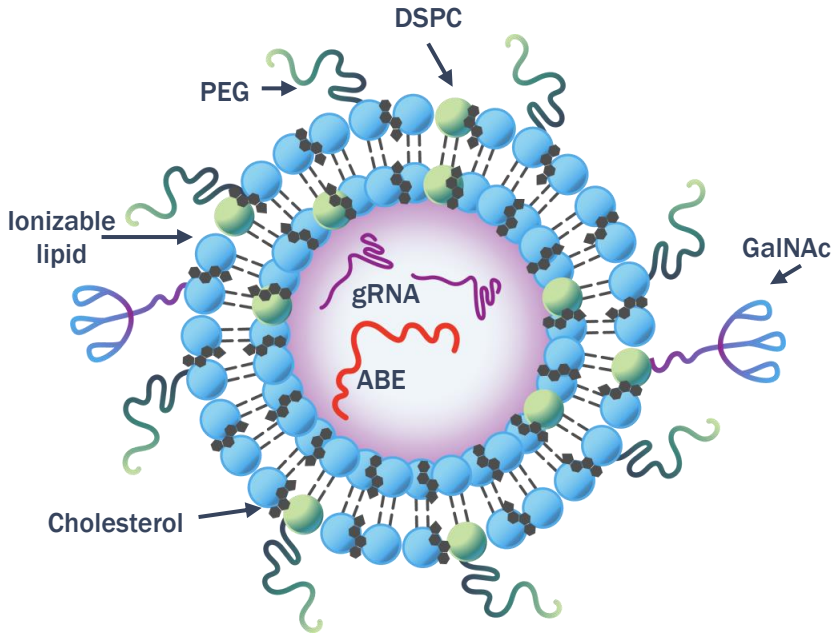
LDL-C reduction:
mean reduction of 53% observed in highest weight-based dose group



As of March 13, 2025. Data are from an ongoing study with an open database and have not been fully cleaned. Percent change from baseline uses time-averaged values from Day 28 through last available follow-up. Day 28 value was used in participants where the Day 28 was the last timepoint. Single observation in 2 participants censored due to changes in background lipid-lowering therapy.
PCSK9, proprotein convertase subtilisin/kexin type 9; SEM, standard error of the mean, LDL-C, low-density lipoprotein cholesterol

Fixed dose groups: Verve evaluated data by total RNA dose, in line with the field's shift towards fixed doses

VERVE-102



Total RNA Dose

Total RNA dose refers to the total weight (in mg) of the RNA in VERVE-102

In any given weight-based cohort, there is a range of total RNA doses administered

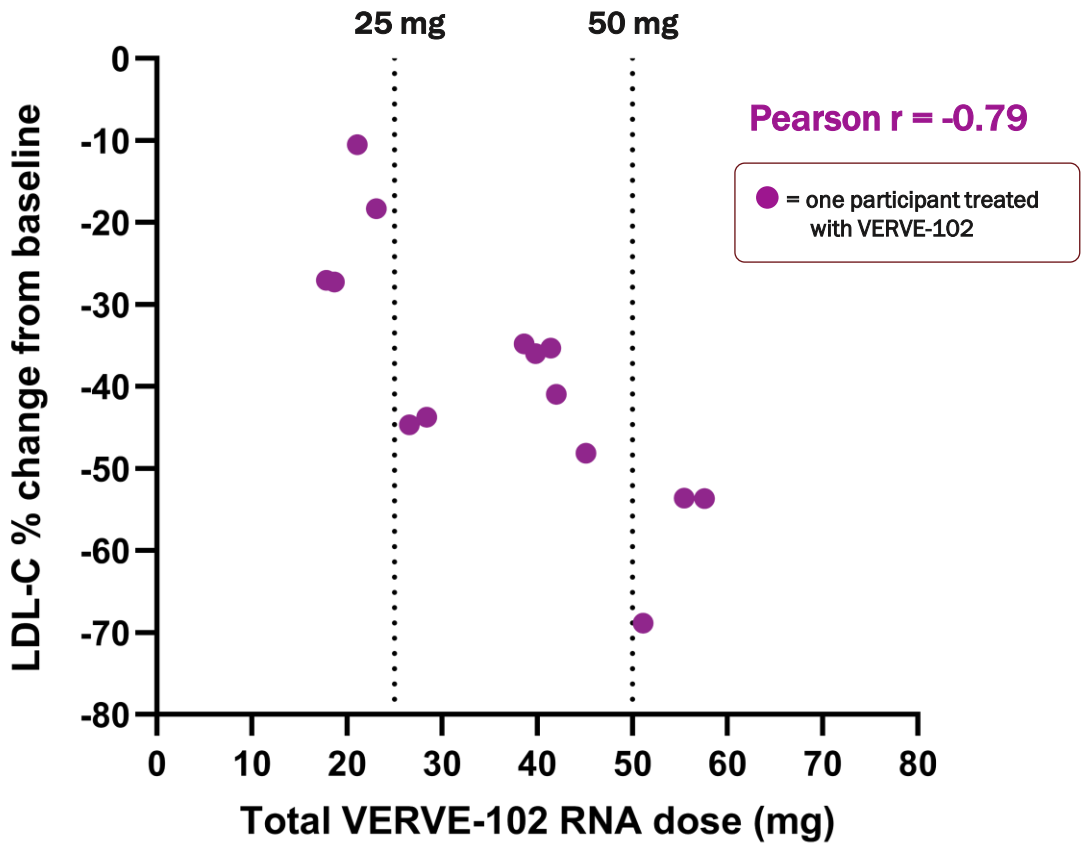
E.g., at 0.6 mg/kg dose:

60 kg Person Receives:	100 kg Person Receives:
36 mg total RNA	60 mg total RNA

RNA, ribonucleic acid; PEG, polyethylene glycol; DSPC, 1,2-distearoyl-sn-glycero-3-phosphocholine; ABE, adenine base editor; gRNA, guide RNA

LDL-C by total RNA dose, per participant: across 14 participants, strong dose-dependent response observed with near-linear relationship

Maximum LDL-C reduction of 69%



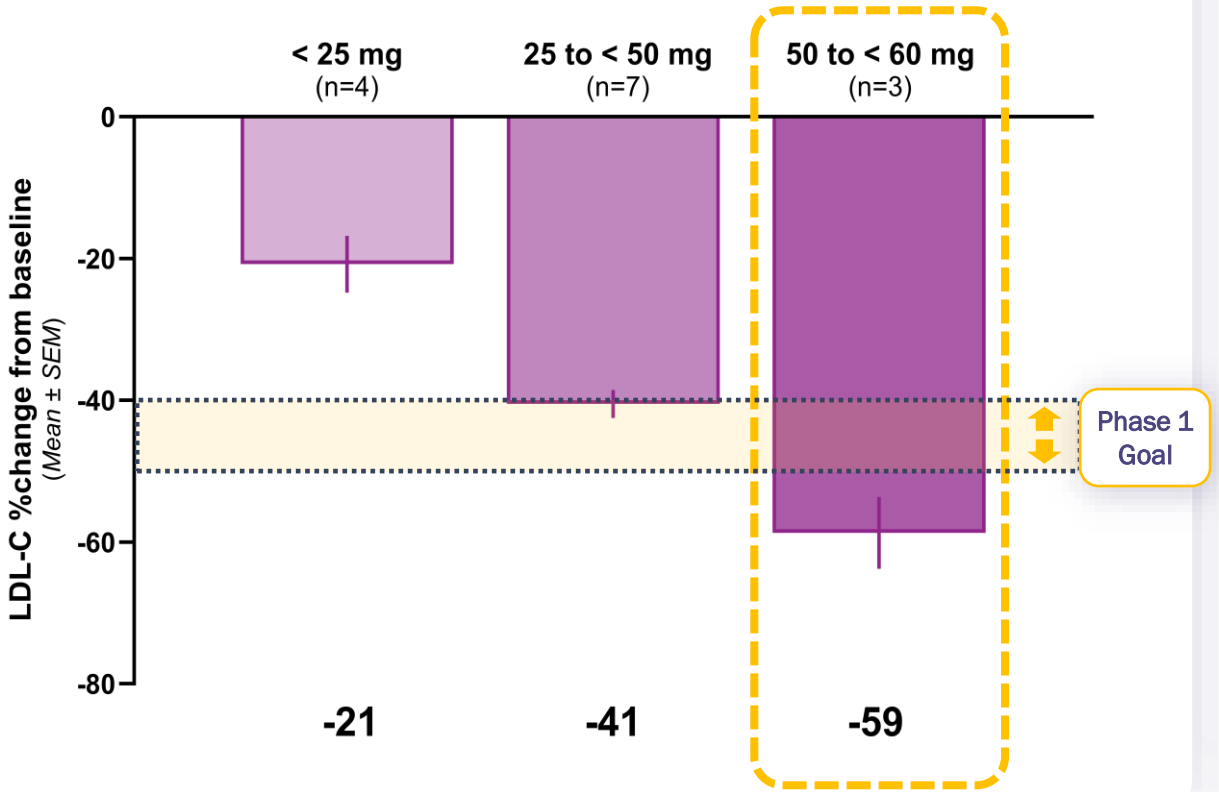
Total RNA Ranges:

- < 25 mg
- 25 – < 50 mg
- 50 – <60 mg

As of March 13, 2025. Data are from an ongoing study with an open database and have not been fully cleaned. Percent change from baseline uses time-averaged values from Day 28 through last available follow-up. Day 28 value was used in participants where the Day 28 was the last timepoint. Single observation in 2 participants censored due to changes in background lipid-lowering therapy.
RNA, ribonucleic acid, LDL-C, low-density lipoprotein cholesterol

LDL-C: mean reduction of 59% in blood LDL-C observed in highest total RNA dose group of VERVE-102

All participants who received ≥ 50 mg achieved > 50% LDL-C reduction from baseline



VERVE-102 dose range	< 25 mg	25 - < 50 mg	50 - < 60 mg
Participants (n)	4	7	3
Mean total RNA dose	20 mg	37 mg	55 mg
Mean LDL-C % reduction from baseline	-21%	-41%	-59%

As of March 13, 2025. Data are from an ongoing study with an open database and have not been fully cleaned. Percent change from baseline uses time-averaged values from Day 28 through last available follow-up. Day 28 value was used in participants where the Day 28 was the last timepoint. Single observation in 2 participants censored due to changes in background lipid-lowering therapy.
LDL-C, low-density lipoprotein cholesterol; RNA, ribonucleic acid; SEM, standard error of the mean

Takeaways from the initial data of the Heart-2 clinical trial

- VERVE-102 was well-tolerated at all dose levels with proprietary GalNAc-LNP delivery platform
  - No treatment-related SAEs and no DLTs
  - No clinically significant laboratory abnormalities and no cardiovascular events
  - One infusion-related reaction across 14 participants treated
- Strong dose-dependent response with total RNA dose identified as a key driver of pharmacodynamics
- At total RNA dose ≥ 50 mg, each participant achieved LDL-C reductions from baseline > 50%, with mean LDL-C reduction of 59% and a maximum reduction of 69% in a single participant

As of March 13, 2025, data cut off date; data are from an ongoing study with an open database and have not been fully cleaned.
SAE, serious adverse events; DLT, dose-limiting toxicity, LNP, lipid nanoparticle; RNA, ribonucleic acid; LDL-C, low-density lipoprotein cholesterol

Next steps for VERVE-102: expect first patient to be dosed in Phase 2 in H2 2025[1]

- Enrollment ongoing in the 0.7 mg/kg cohort in the Heart-2 clinical trial
- Plan to disclose final data from dose escalation portion of the Heart-2 clinical trial in H2 2025
- Anticipate delivery of opt-in package to Lilly in H2 with potential decision by year-end 2025
- Expect first patient to be dosed in Phase 2 trial in H2 2025[1]

Data are from an ongoing study with an open database and have not been fully cleaned.
1. Subject to regulatory clearance. PD, pharmacodynamic

Beyond the data: VERVE-102 program poised for success

SUCCESS FACTORS FOR VERVE-102

Time to market

- Phase 1 enrollment pace has exceeded expectations
- 5 CTAs & U.S. IND expand global footprint for Phase 2 onwards
- FDA Fast Track designation received

Capital

- Cash runway estimated into mid-2027, sufficient to support completion of the Phase 2 trial
- Lilly decision for opt-in at end-of-year; opt-in would defray costs and extend runway further

Manufacturing

- Consistent supply established for Phase 1 and 2 trials; transferring processes to commercial-scale CDMOs
- Low cost of goods expected given precedent for analogous at-scale RNA / LNP products

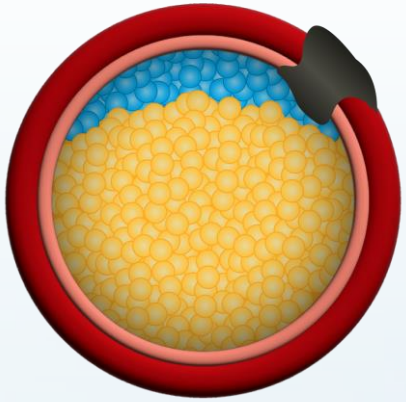
Commercial

- WW PCSK9 market of ~$4B; growing at ~40% year-over-year
- Strong enthusiasm for VERVE-102 amongst surveyed patients and HCPs[1,2]

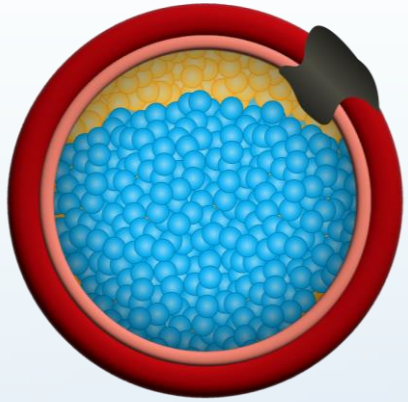
Subject to regulatory clearance. 1. HCP Survey; HCP Interviews; ClearView Analysis; 2. Patient preferences survey conducted by polling firm Morning Consult; LifeSci Capital
CTA, clinical trial applications; IND, investigational new drug application; CDMO, contract development and manufacturing organization; RNA, ribonucleic acid; LNP, lipid nanoparticle; WW, worldwide; PCSK9, proprotein convertase subtilisin/kexin type 9; HCP, healthcare practitioner

ANGPTL3 program: VERVE-201

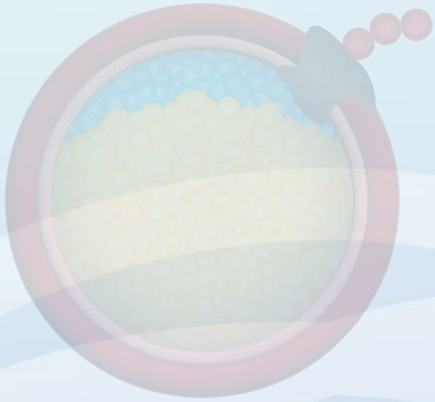
LDL



TRL



Lp(a)



VERVE-102 (PCSK9)

VERVE-201 (ANGPTL3)

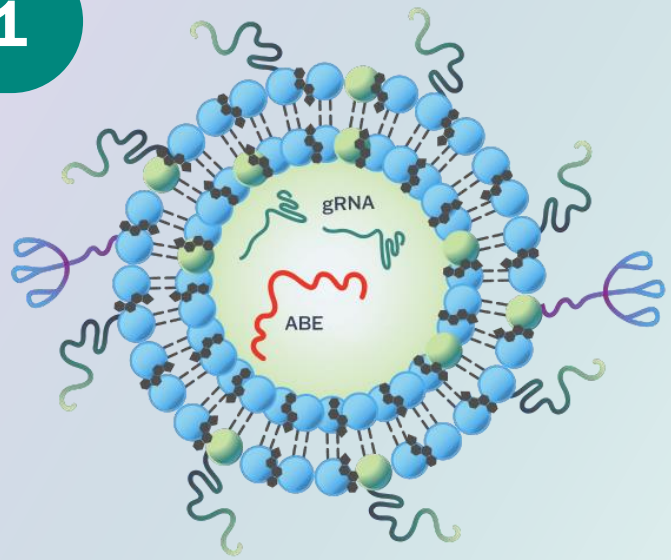
VERVE-301 (LPA)

Cholesterol Triglyceride *Apolipoprotein B* *Apolipoprotein(a)*

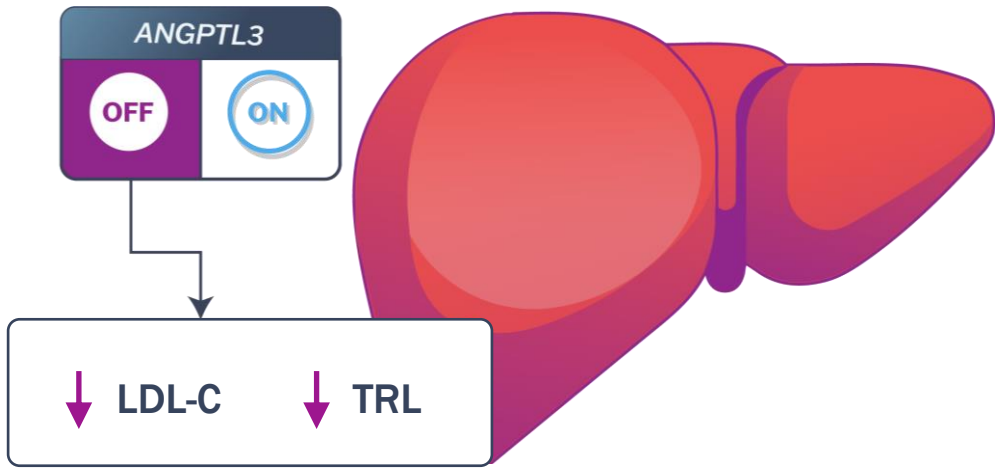
ANGPTL3, angiopoietin-like protein 3; LDL, low-density lipoprotein; TRL, triglyceride rich lipoprotein

Inactivation of hepatic *ANGPTL3* is expected to lower circulating LDL-C and triglyceride concentrations

VERVE-201



- Genetic and pharmacologic validation of target
- ANGPTL3 protein produced almost exclusively in the liver
- Mechanism of LDL-C lowering is fully independent of functional LDLR



***ANGPTL3* inactivation by introducing premature stop codon**

ANGPTL3, angiopoietin-like protein 3; ABE; adenine base editor; gRNA: guide RNA; LDL-C: low-density lipoprotein cholesterol; mRNA, messenger RNA; LDLR, low-density lipoprotein receptor; TRL, triglyceride-rich lipoprotein

ANGPTL3 inactivation has the potential to treat a broad range of lipid disorders that have a large unmet need

Homozygous FH[1]

LDL-C ≥ 500 mg/dL

> 3,000

Refractory Hypercholesterolemia[2]

(ASCVD patients not at LDL-C goal on max standard-of-care)

~4 million

Severe HTG[3]

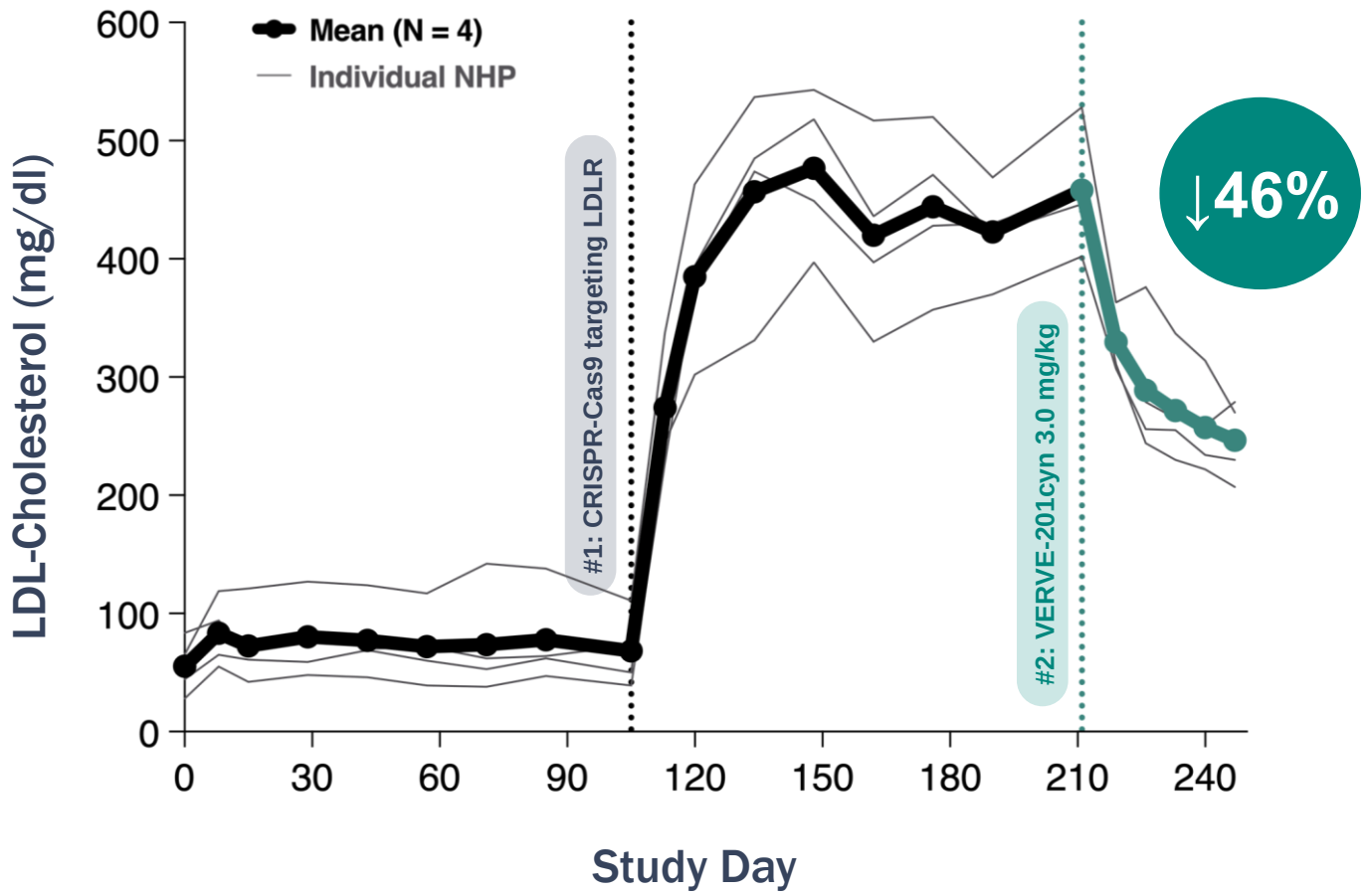
TG ≥ 500 mg/dL

> 10 million

PREVALENCE (U.S. + EU)

1. Cuchel M et al. Eur Heart J 2023;44: 2277-2291. 2. O'Donoghue ML et al. Circulation 2022; 146: 1109-1119. 3. Christian J et al. American Journal of Cardiology 2011; 107: 891–897.
ANGPTL3, angiopoietin-like protein 3; ASCVD, atherosclerotic cardiovascular disease; FH, familial hypercholesterolemia; HTG, hypertriglyceridemia; LDL-C: low-density lipoprotein cholesterol; TG, triglycerides

In LDLR-deficient NHPs treated with VERVE-201cyn, 46% mean decrease in LDL-C observed (458 to 247 mg/dL)



Dosed with VERVE-201cyn

- Mean LDL-C decreased from 458 to 247 mg/dL
- VERVE-201cyn in NHPs:
  - **46%** decrease in LDL-C
  - **54%** decrease in TG

LDLR, low-density lipoprotein receptor; NHP, non-human primates; LDL-C, low-density lipoprotein cholesterol; TG, triglycerides

1. Method previously described in Kasiewicz et al. Nat Comm 2023;14: 2776.

Pulse-1: Phase 1b clinical trial designed to evaluate safety and tolerability of VERVE-201 (ANGPTL3); anticipated program update in 2H 2025

pulse-1

Population:

- Males and females (age 18 to 70)
- Refractory hypercholesterolemia
- Require additional LDL-C lowering despite maximally tolerated standard of care therapies, potentially including PCSK9 inhibitors

Single Ascending Dose:

- Three to nine participants per cohort receive a single dose; adaptive design

Trial Endpoints:

- Primary: evaluate safety and tolerability
- Secondary: changes in blood ANGPTL3 and LDL-C levels

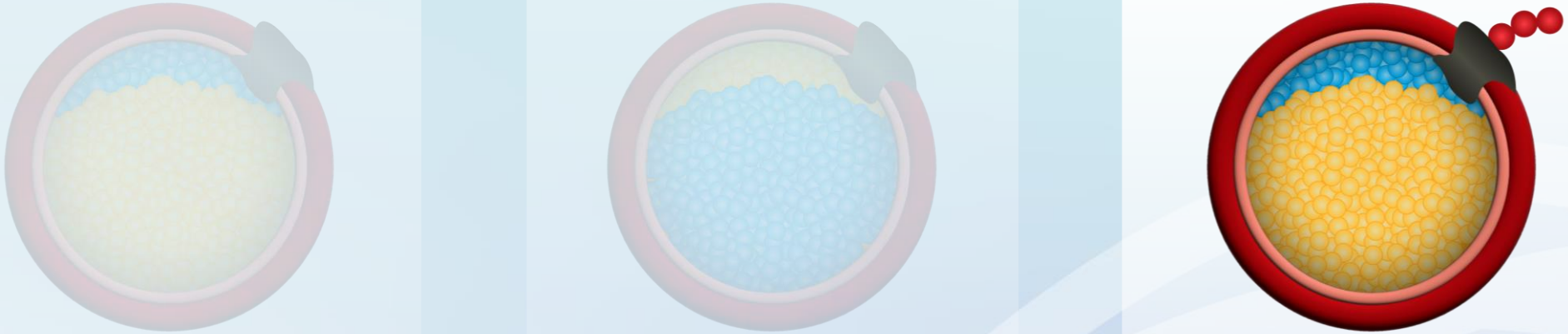
ANGPTL3, angiopoietin-like protein 3; LDL-C, low-density lipoprotein cholesterol; PCSK9, proprotein convertase subtilisin/kexin type 9
Clinical trial registration: NCT06451770
Women of childbearing potential are excluded from the study.

Lp(a) program: VERVE-301

LDL

TRL

Lp(a)



VERVE-102 (PCSK9)

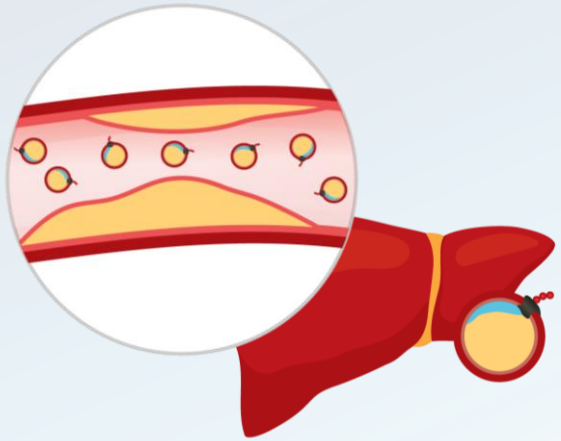
VERVE-201 (ANGPTL3)

VERVE-301 (LPA)

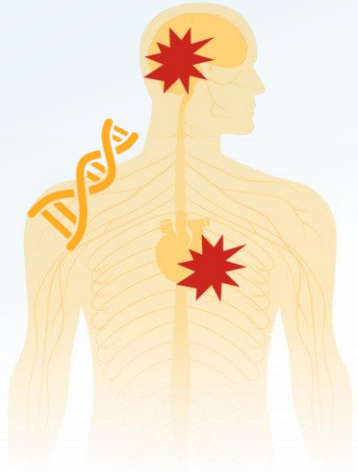
Cholesterol Triglyceride *Apolipoprotein B* *Apolipoprotein(a)*

Lp(a), lipoprotein(a)

Lipoprotein(a), or Lp(a), is a major area of unmet need



Lp(a) is an LDL-like particle with apolipoprotein(a) covalently linked to apolipoprotein B; produced in the liver and circulates in the blood



Lp(a) is a genetically validated, independent **risk factor for ASCVD, ischemic stroke, thrombosis, and aortic stenosis**

~13M people in the U.S. and EU have **elevated Lp(a)[1]**

Lp(a)

~25% of ASCVD patients with Lp(a) > 125 nmol/L (~ 50 mg/dL)[1]

Currently no therapies approved for the treatment of elevated Lp(a)

Lp(a), lipoprotein(a); LDL, low-density lipoprotein; ASCVD, atherosclerotic cardiovascular disease

1. Nissen SE et al. Open Heart 2022;9:e002060.

VERVE-301: targeting the *LPA* gene to address a major independent risk factor for ASCVD

NOMINATION OF DEVELOPMENT CANDIDATE

Milestone payment received from Eli Lilly

Designed to durably inactivate the *LPA* gene in the liver with novel, *in vivo* gene editing technology

Delivered by Verve's GalNAc-LNP

DC selection based on acceptable off-target profile, dose-response profile, and apo(a) reduction

LPA, lipoprotein(a); ASCVD, atherosclerotic cardiovascular disease; DC, development candidate

Focused execution in 2024; milestone-rich 2025

2024 ACHIEVED MILESTONES

PCSK9 PROGRAM

- ✓ Potent and durable LDL-C lowering with PCSK9 editing approach
- ✓ Dose escalation of VERVE-102 (using proprietary GalNAc-LNP liver delivery technology) with no clinically significant lab abnormalities

ANGPTL3 PROGRAM

- ✓ First patient dosed with VERVE-201

2025 ANTICIPATED MILESTONES

PCSK9 PROGRAM

- ✓ Initial Phase 1b data for VERVE-102 (2Q 2025)
- Final data for dose escalation portion of the Phase 1b for VERVE-102 (2H 2025)
- Deliver opt-in package to Lilly (2H 2025)
- Initiate Phase 2 clinical trial (2H 2025)[1]

ANGPTL3 PROGRAM

- Program update for VERVE-201 (2H 2025)

LPA PROGRAM

- ✓ DC nomination of VERVE-301

PCSK9, proprotein convertase subtilisin/kexin type 9; LDL-C, low-density lipoprotein cholesterol; ANGPTL3, angiopoietin-like protein 3; DC, development candidate; LPA, lipoprotein(a)
1. Subject to regulatory clearance