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

JANUARY 2025

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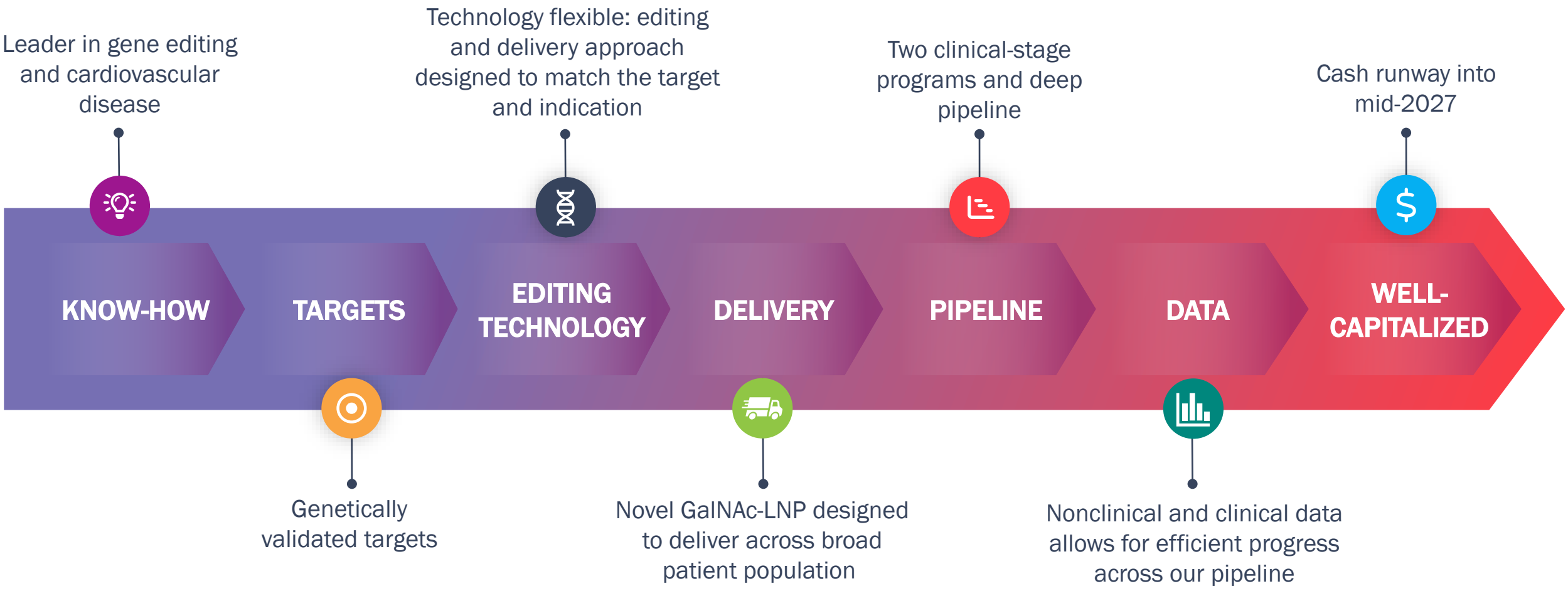
This presentation contains “forward-looking statements” within the meaning of the Private Securities Litigation Reform Act of 1995 that involve substantial risks and uncertainties, including statements regarding the Company’s ongoing Heart-2 clinical trial and Pulse-1 clinical trial; the timing and availability of data for the Heart-2 trial and timing for initiation of a Phase 2 clinical trial for the PCSK9 program; the timing of updates for the PCSK9 and ANGPTL3 programs; the development of VERVE-301; the timing of Eli Lilly and Company’s opt-in decision for the PCSK9 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.

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

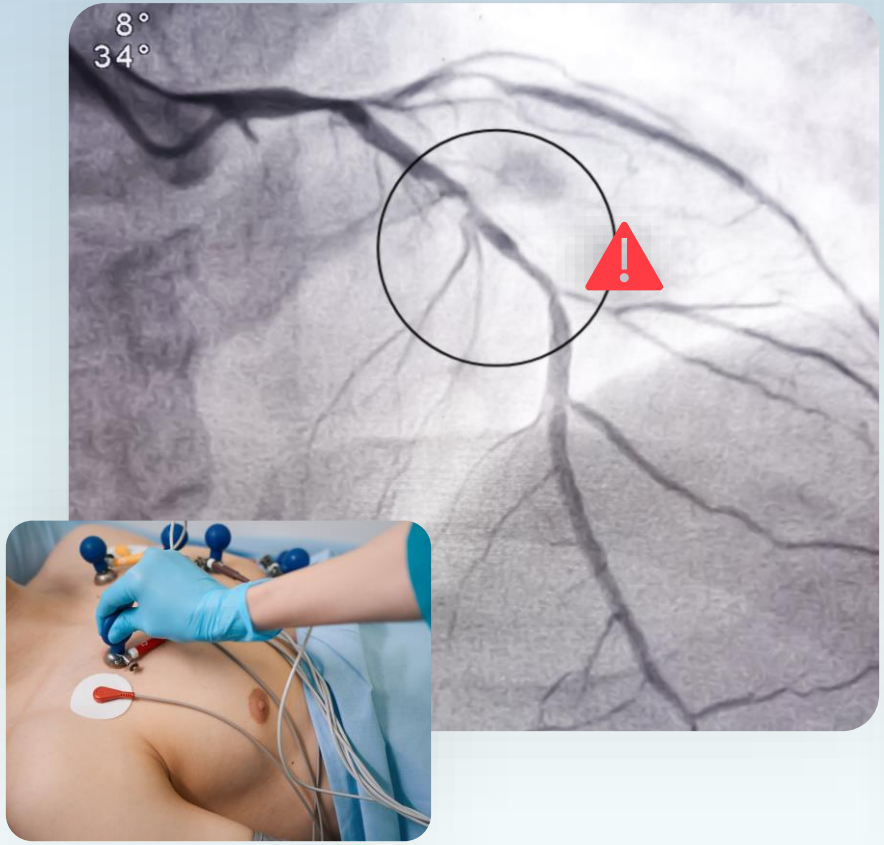


Verve is advancing a pipeline of *in vivo* gene editing programs designed to lower cholesterol lifelong after a single treatment

TARGET	INDICATION	TECHNOLOGY	RESEARCH	IND-ENABLING	CLINICAL
PCSK9 (VERVE-102)	Heterozygous familial hypercholesterolemia ASCVD	Base Editor (GalNAc-LNP)			
PCSK9 (VERVE-101)[1]	Heterozygous familial hypercholesterolemia ASCVD	Base Editor			
ANGPTL3 (VERVE-201)	Homozygous familial hypercholesterolemia Refractory hypercholesterolemia	Base Editor (GalNAc-LNP)			
LPA (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. As of April 2, 2024, Verve has paused enrollment of the Heart-1 Phase 1b trial of VERVE-101 and is prioritizing clinical development of VERVE-102.

Heterozygous familial hypercholesterolemia (HeFH): severe disease with significant unmet need for early, durable lowering of low-density lipoprotein cholesterol (LDL-C)



HeFH is the **most common genetic disease** in humans

>3M Patients in U.S. + EU[1]

Prolonged exposure to high LDL-C **results in early heart attacks and strokes**

50% Of untreated men will develop heart disease before age 50[2]

Diagnosis & treatment often initiated **after significant buildup of cholesterol leading to heart artery blockages**

47 Mean age of diagnosis[3]

Current standard-of-care inadequate to durably reduce heart attack risk

<25% Of treated patients are at LDL-C goal[3]

1. de Ferranti SD et al. Circulation 2016;133: 1067-72. 2. Pećin I et al. European Journal of Preventive Cardiology 2017;24: 1383-1401. 3. Duell P et al. Atherosclerosis 2019;289: 85–93.

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

All atherosclerotic cardiovascular disease (ASCVD) *~54M in US/EU*[1]

VERVE-102 / VERVE-101	VERVE-102 / VERVE-101	VERVE-201	VERVE-201	VERVE-301
HeFH[2]	ASCVD not at LDL-C goal on statin[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	**> 25M** in US/EU	**> 3,000** in US/EU	**> 4M** in US/EU	**> 13M** in US/EU (~25% ASCVD)

For all prevalence numbers contained in this presentation, U.K. population included for purposes of determining patient population, in addition to U.S. and EU.
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)

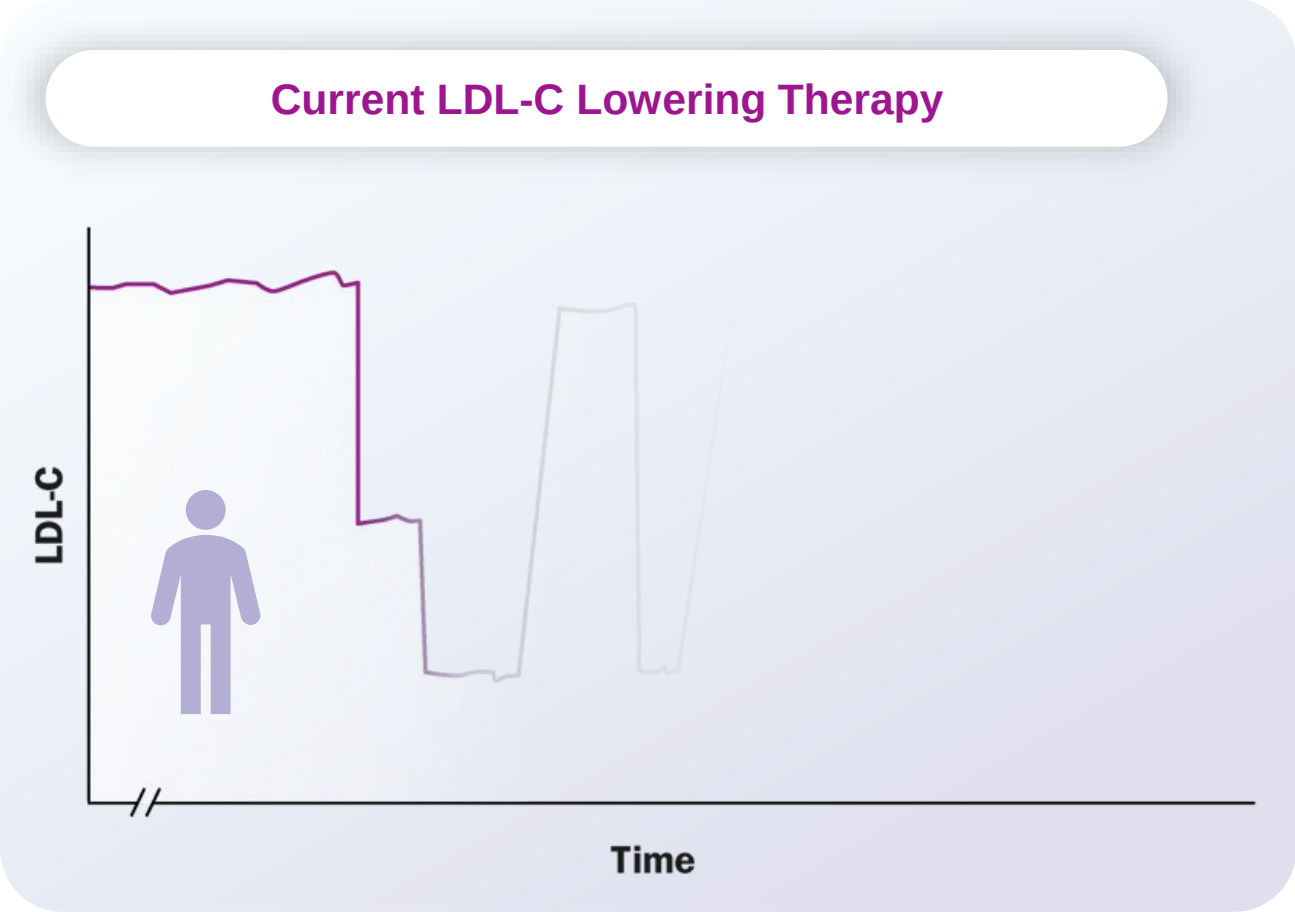
LDL, low-density lipoprotein; TRL, triglyceride-rich lipoprotein; Lp(a), lipoprotein(a)

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

What is the unmet medical need for cholesterol lowering?

Current treatments lower LDL-C by 40–60%; need to be taken regularly and lifelong



LDL-C, low-density lipoprotein cholesterol

What is the unmet medical need for cholesterol lowering?

Requirement for decades of chronic therapy leads to poor real-world LDL-C control

About **50%** of ASCVD patients are **not on a statin**[1]

Only about **2%** of eligible ASCVD patients are **currently on a PCSK9 inhibitor**[2]

Up to **50%** of patients **discontinue** CVD medications **within 12 months**[3,4]

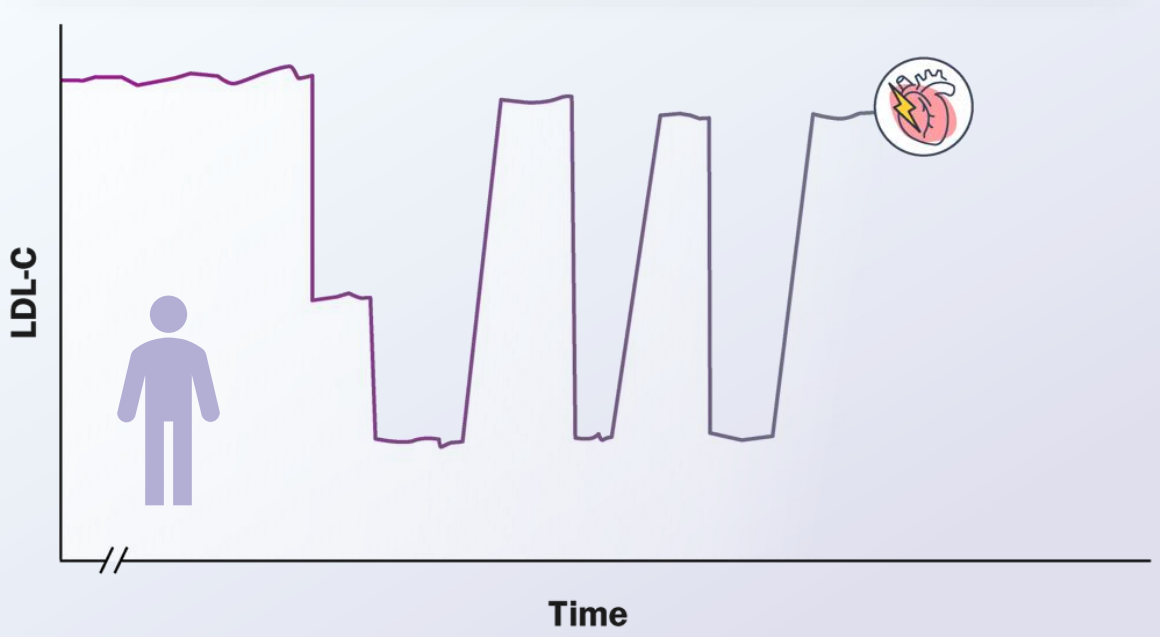
LDL-C, low-density lipoprotein cholesterol; ASCVD, atherosclerotic cardiovascular disease; PCSK9, proprotein convertase subtilisin/kexin type 9; CVD, cardiovascular disease

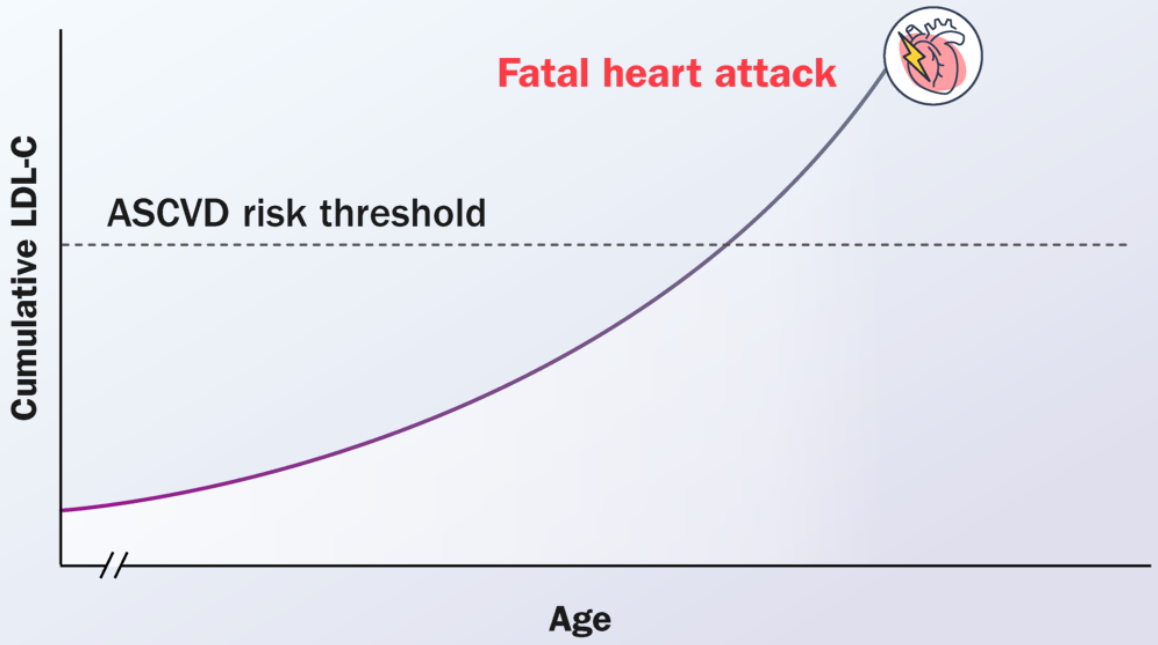
1. Nelson AJ et al. J Am Coll Card 2022;79: 1802–13; 2. Dayoub EJ et al. J Am Heart Assoc 2021;10: e019331; 3. Nelson A et al. Nature Reviews Cardiology 2024. https://doi.org/10.1038/s41569-023-00972-1; 4. Naderi SH et al. Am J Med 2012;125: 882–887.e1.

What is the unmet medical need for cholesterol lowering?

Years of exposure to elevated LDL-C increases the risk for major cardiovascular events

Cumulative exposure to LDL-C (cholesterol*years)



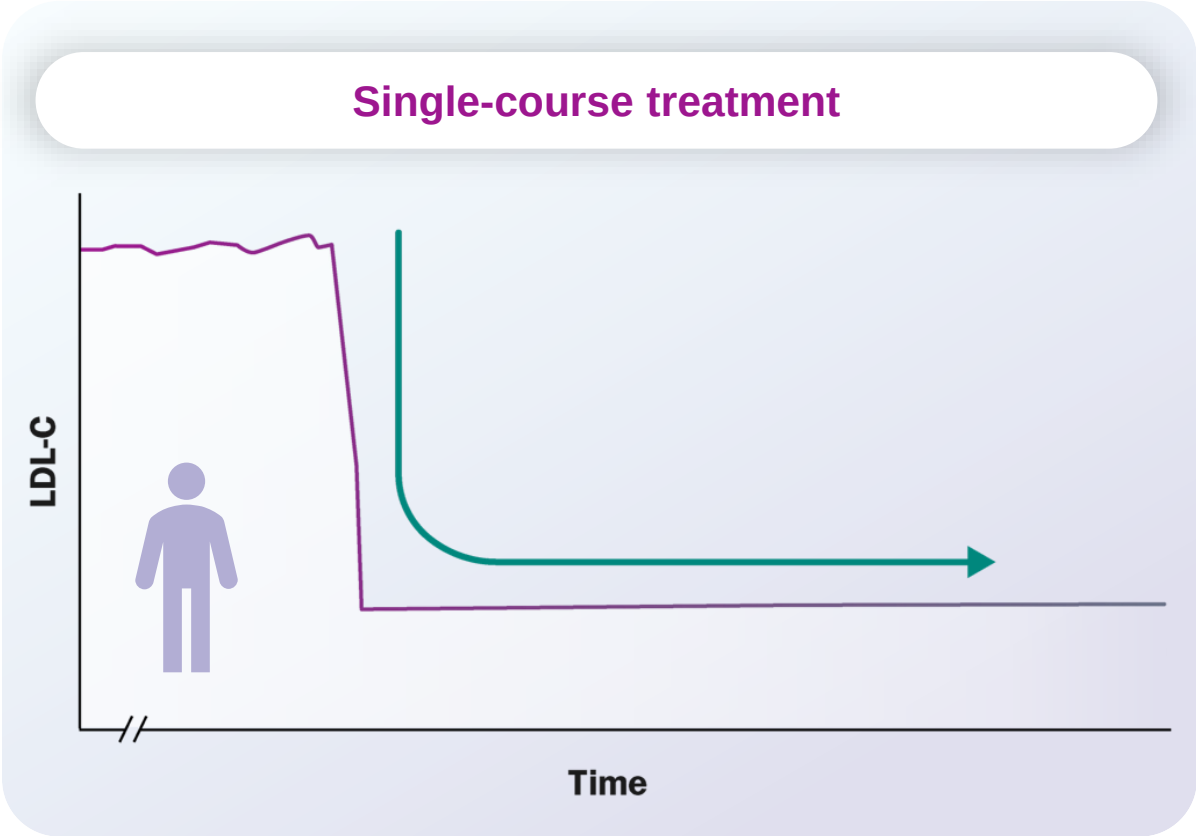


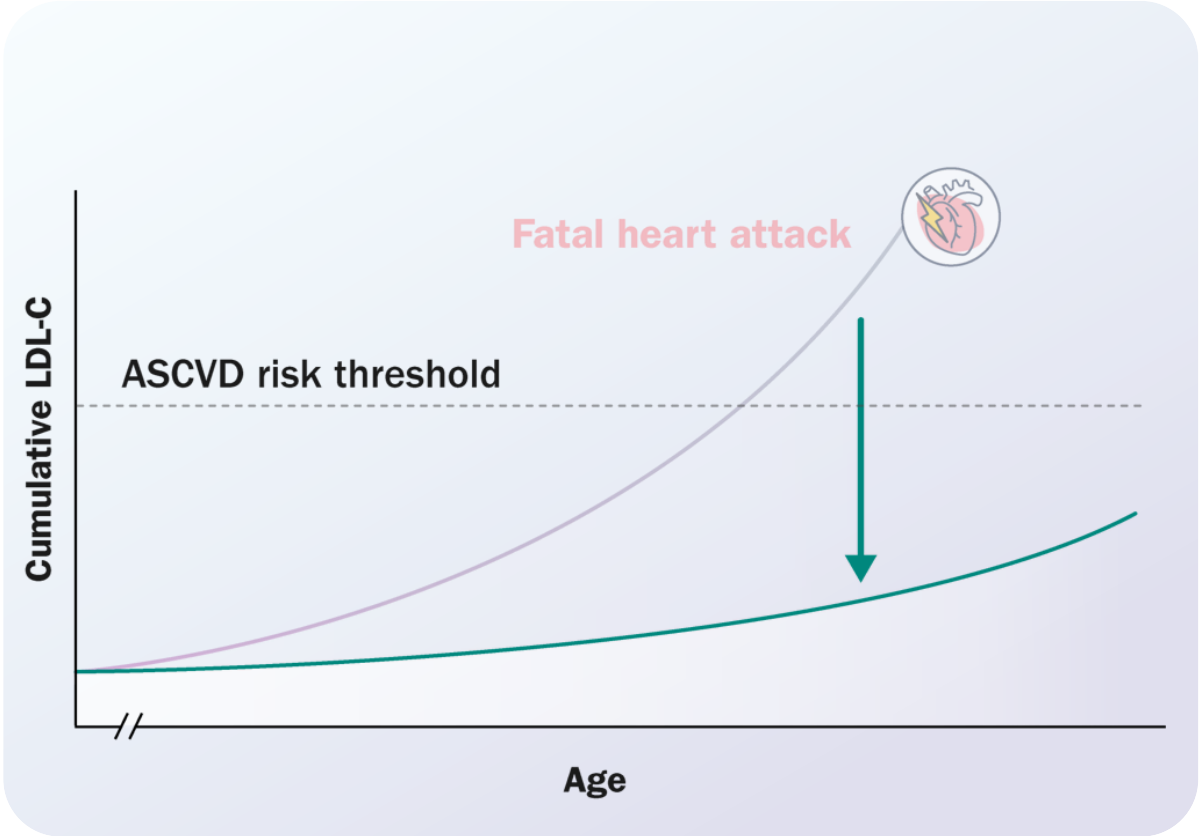
Nonadherence to lipid lowering therapies can increase CVD event risk by more than 40%[1]

LDL-C, low-density lipoprotein cholesterol; ASCVD, atherosclerotic cardiovascular disease; CVD, cardiovascular disease

1. Banislal S et al. J Am Coll Cardiol 2016;68:789-801.

Durably lowering LDL-C meaningfully decreases the risk of ASCVD

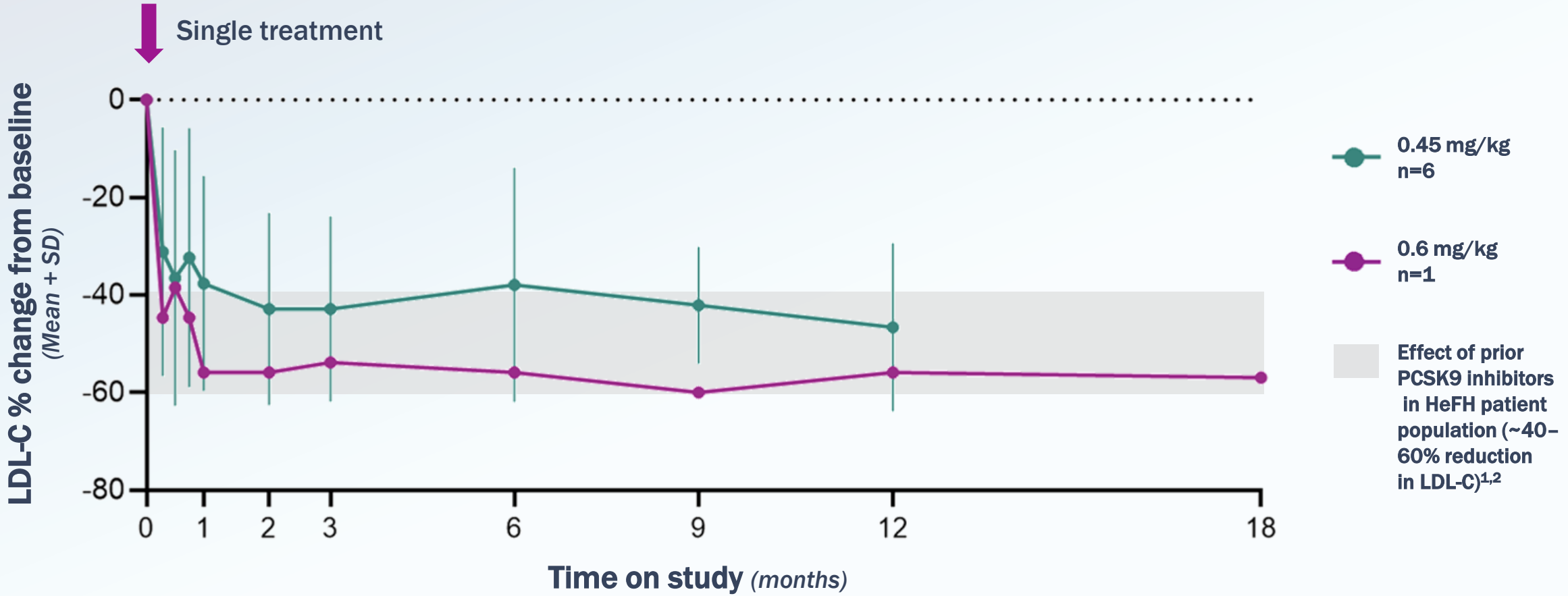




Our goal: one-time treatment, potential for a lifetime of benefit

LDL-C, low-density lipoprotein cholesterol; ASCVD, atherosclerotic cardiovascular disease

Durability in humans: evidence for sustained LDL-C reduction following single VERVE-101 treatment in two higher dose cohorts



LDL-C, low-density lipoprotein cholesterol; SD, standard deviation; HeFH, heterozygous familial hypercholesterolemia; PCSK9, proprotein convertase subtilisin/kexin type 9

As of October 3, 2024. Data are from an ongoing study with an open database and have not been fully cleaned. Participants in 0.45 mg/kg cohort have variable duration of follow up, with n=6 at 6 months and n=3 at 9 months and 12 months.

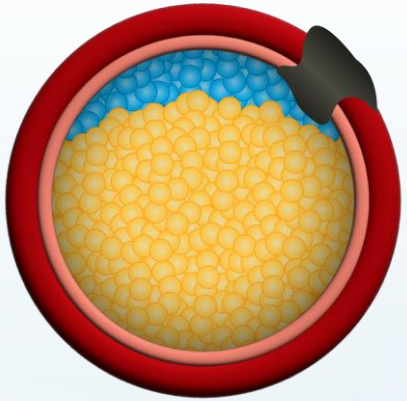
One of the six 0.45 mg/kg participants intensified statin therapy from baseline more than 6 months after VERVE-101 treatment.

1. Raal et al. N Engl J Med 2020;382: 1520-1530; 2. Raal et al. Lancet 2015;385: 331–340.

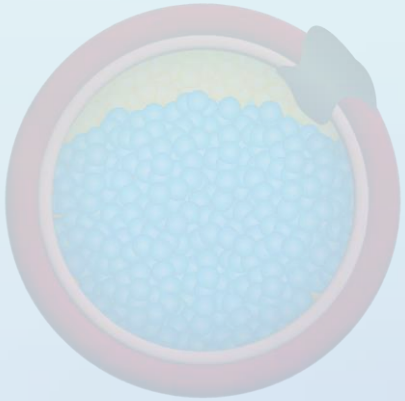
PCSK9 program: VERVE-102

~28M addressable patients (3M HeFH + 25M ASCVD not at LDL-C goal on statin) in the U.S./EU[1, 2, 3]

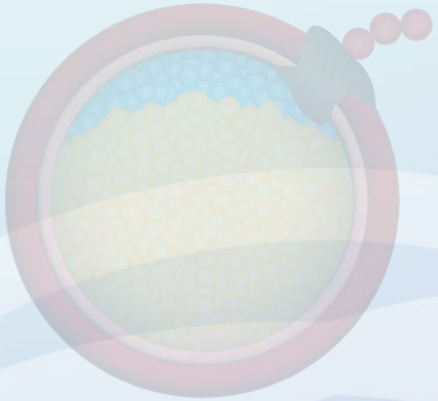
LDL



TRL



Lp(a)



VERVE-102 (PCSK9)

VERVE-201 (ANGPTL3)

VERVE-301 (LPA)

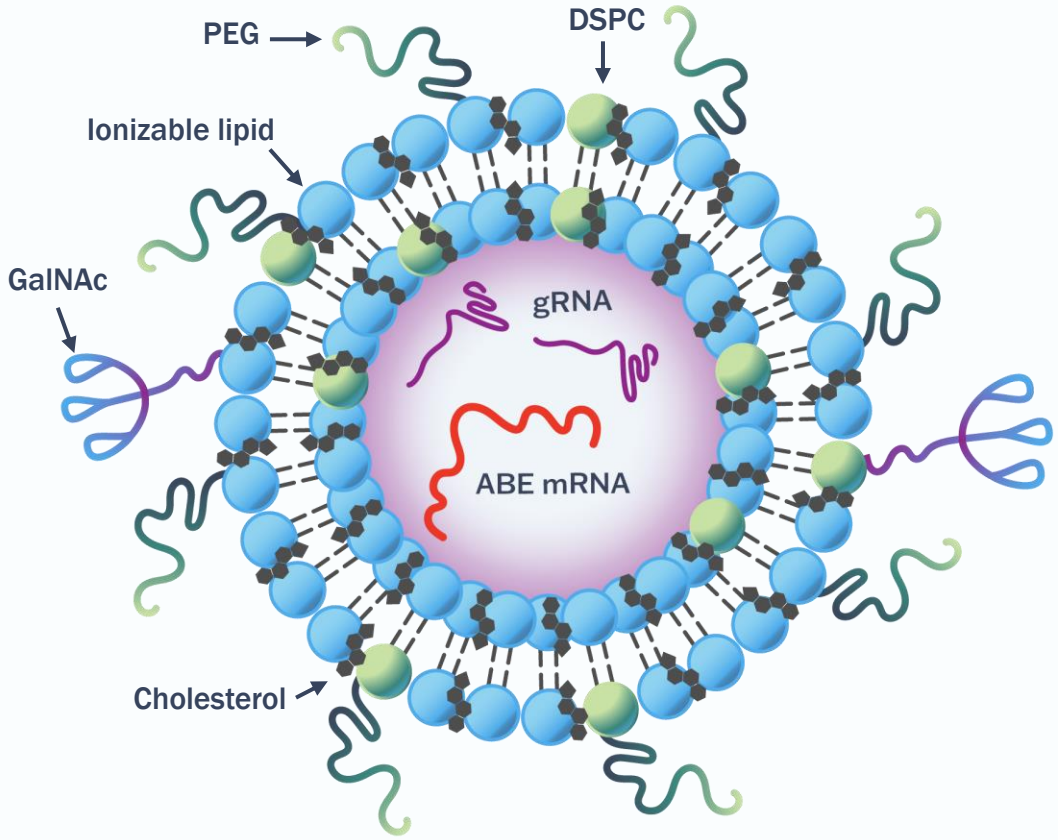
Cholesterol Triglyceride *Apolipoprotein B* *Apolipoprotein(a)*

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

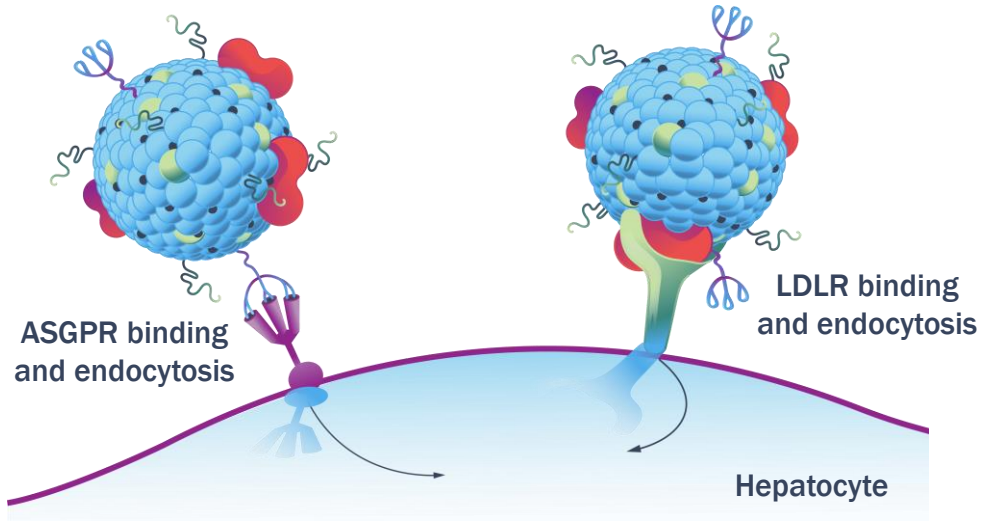
1. de Ferranti et al. Circulation 2016;15: 1067-72
2. Gu J et al. Am J Prev Cardiol 2022;10: 100336
3. Ray KK et al. European Journal of Preventive Cardiology 2021;28: 1279–1289

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 LNP



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



The translated adenine base editor (ABE) pairs with the gRNA to target and inactivate *PCSK9* with precise DNA edit

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

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

heart-2

POPULATION SUMMARY

- Males and females (age 18 to 70)
- HeFH and/or premature CAD
- Require additional LDL-C lowering despite maximally tolerated oral therapies
- Not currently taking a PCSK9 inhibitor

SINGLE ASCENDING DOSE

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

- **0.3** mg/kg Cohort (n ≥ 3)
- **0.45** mg/kg Cohort (n ≥ 3)
- **0.6** mg/kg Cohort (n ≥ 3)
- **Fourth Dose** Cohort (n ≥ 3)

TRIAL ENDPOINTS

- **PRIMARY:** Safety and tolerability
- Pharmacokinetics of VERVE-102
- Changes in blood PCSK9 and LDL-C levels

HeFH, heterozygous familial hypercholesterolemia; CAD, coronary artery disease; LDL-C, low-density lipoprotein cholesterol; PCSK9, proprotein convertase subtilisin/kexin type 9
Clinical trial registration: NCT06164730
Women of childbearing potential are excluded from the study.

verve THERAPEUTICS

Heart-2 is progressing as planned; now dosing 0.6 mg/kg cohort

Data cut-off as of October 29, 2024

Dosing has been completed in seven participants in the first two dose cohorts, 0.3 mg/kg and 0.45 mg/kg, in the Heart-2 clinical trial.

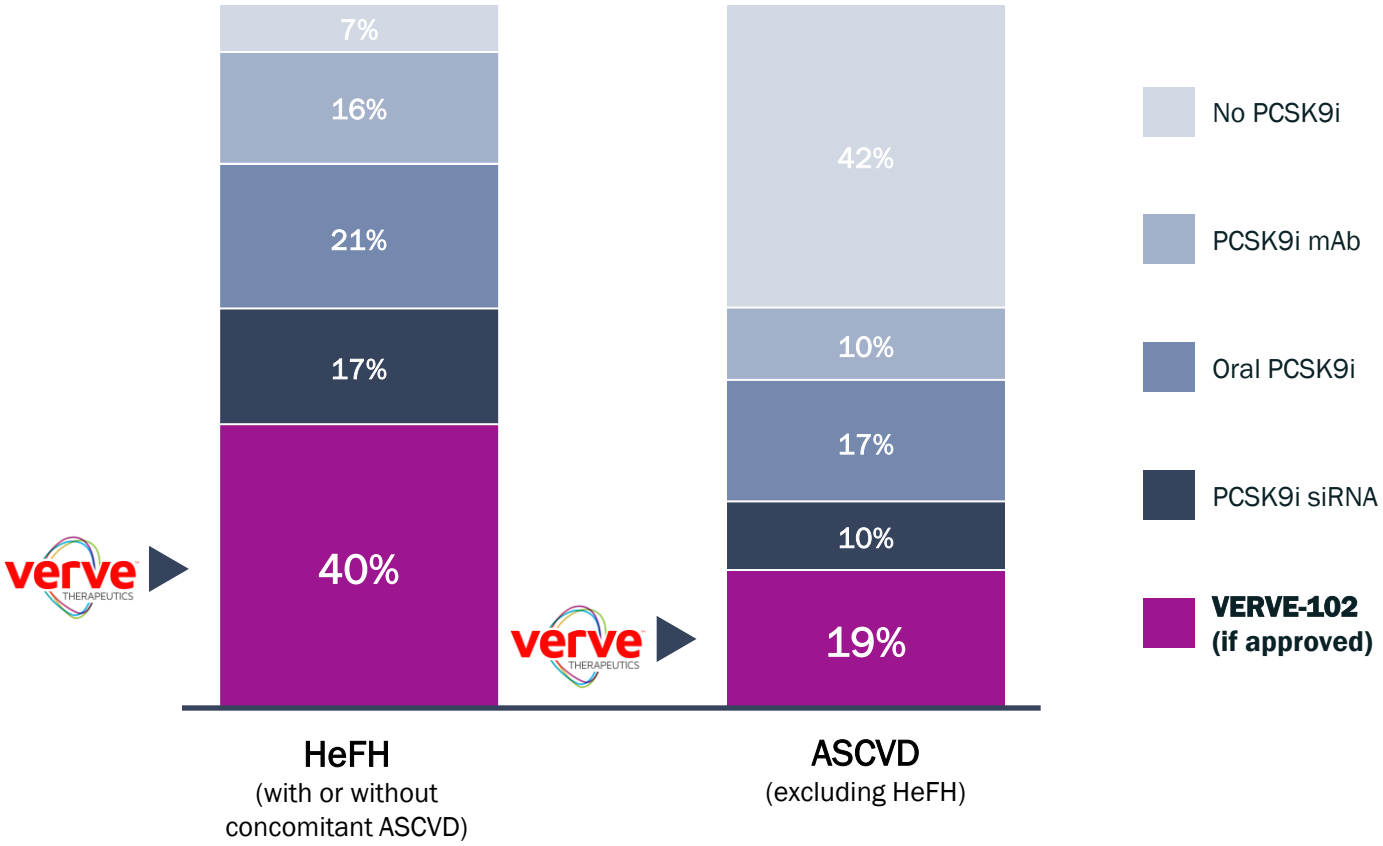
VERVE-102 has been well-tolerated. No serious adverse events and no clinically significant laboratory abnormalities have been observed.

Initial clinical data from 10–12 participants across 3 dose cohorts expected in 2Q 2025.

Will physicians be open to a one-time gene editing treatment as a solution?

Cardiologists prefer VERVE-102 for 40% of their HeFH patients

Cardiologist Treatment Selection in Future Cholesterol-lowering Landscape (N=100)



> Younger patients could benefit from [VERVE-102] for a longer period of time…and for people with very high LDL-C, there's impetus to get levels under control quickly.

HeFH, heterozygous familial hypercholesterolemia; ASCVD, atherosclerotic cardiovascular disease; LDL-C, low-density lipoprotein cholesterol; mAb, monoclonal antibody; PCSK9i, proprotein convertase subtilisin/kexin type 9 inhibitor; siRNA, small interfering RNA

Source: HCP Survey; HCP Interviews; ClearView Analytics

Will patients be open to a one-time gene editing treatment as a solution?

35% of surveyed patients show openness to a one-time gene editing treatment

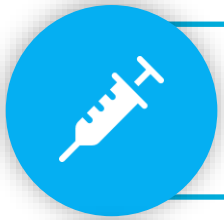
Assuming you will have lifelong therapy in the treatment of high cholesterol and/or cardiovascular disease, please select the therapeutic option that is most appealing to you (N=484)

Once daily, oral pill *(non-gene editing)*

46%

One-time gene/base editing treatment, IV injection performed by healthcare provider

35%



Twice annually, subcutaneous injection performed by healthcare provider *(non-gene editing, requiring doctor visit)*

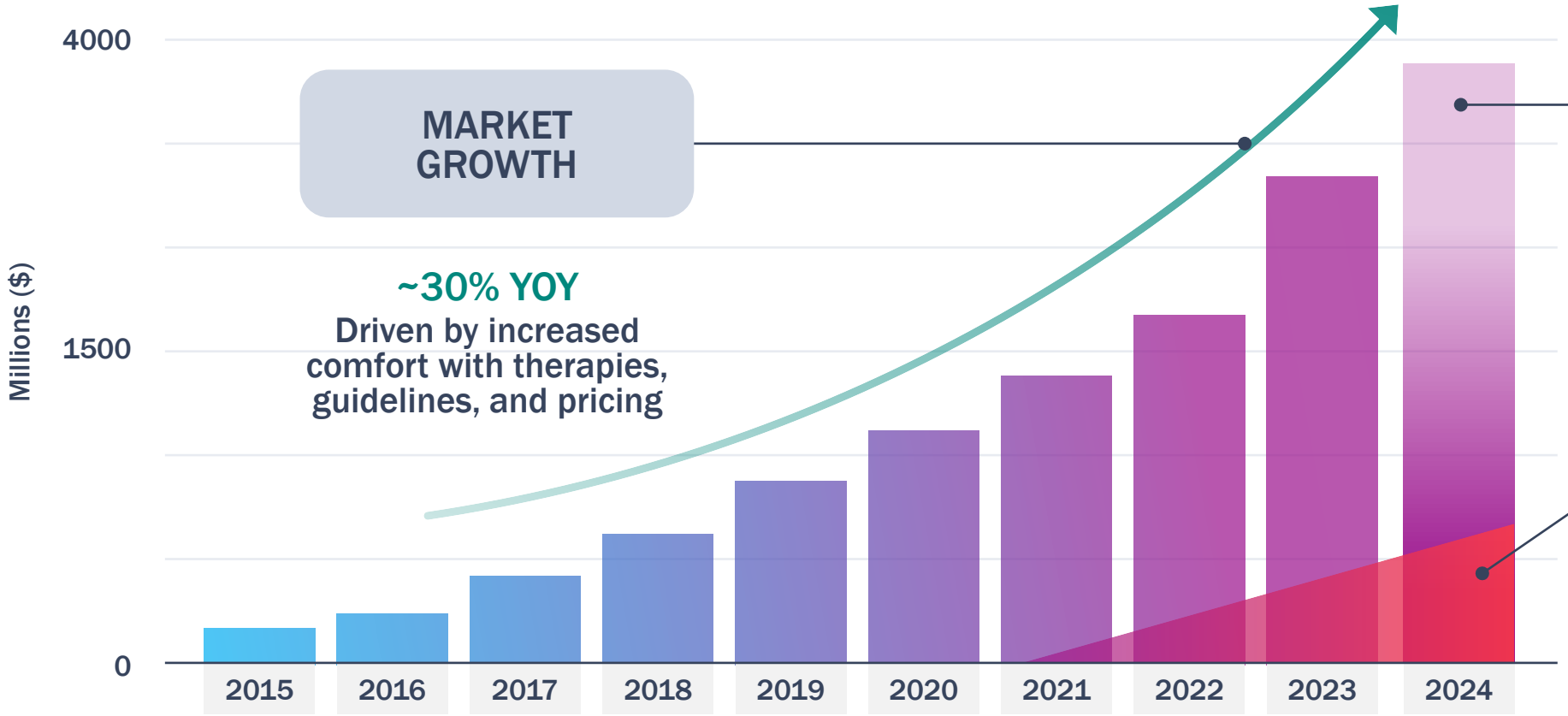
14%

Twice monthly, subcutaneous injection performed by self *(non-gene editing)*

4%

Source: Patient preferences survey conducted by polling firm Morning Consult; LifeSci Capital

PCSK9 inhibitor market growing rapidly



MARKET GROWTH

~30% YOY

Driven by increased comfort with therapies, guidelines, and pricing

2024 PROJECTIONS

$3.5–4.0B GLOBAL MARKET

>$700M Leqvio® (inclisiran)

INCREASING USE OF LEQVIO

Increased adoption rate illustrates patient desire for a long-acting treatment to lower LDL-C

DESPITE GROWTH, MARKET REMAINS LARGELY UNTAPPED

Likely 30–40% of HeFH patients and <5% of eligible ASCVD patients have received a PCSK9 inhibitor in the U.S.[1,2]

PCSK9, proprotein convertase subtilisin/kexin type 9; ASCVD, atherosclerotic cardiovascular disease; LDL-C, low-density lipoprotein cholesterol

1. Duell P et al. Atherosclerosis 2019;289: 85–93 and Family Heart Foundation 2023 CASCADE FH Registry; 2. Navar et al. American Heart Journal 2025; 279: 107-117.

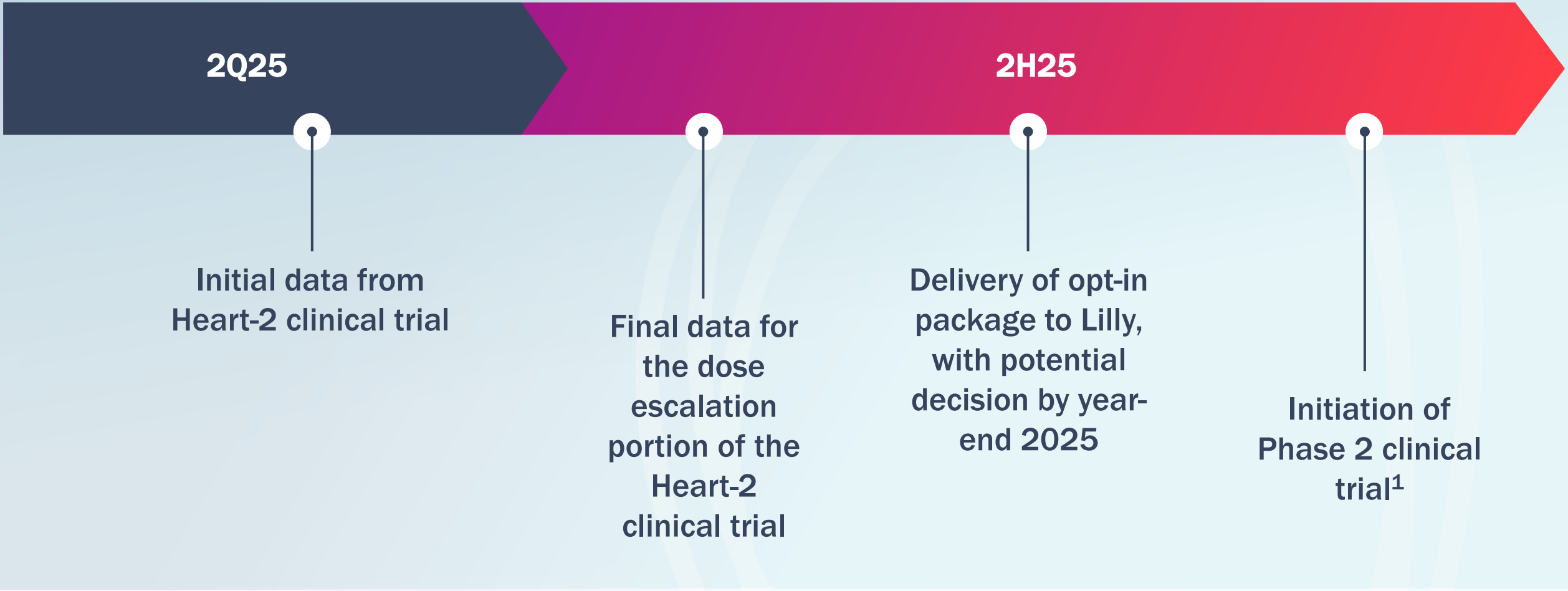
Overview of Eli Lilly collaboration for *PCSK9* program

	Development			Commercial	
	Current	**Opt-In Process**	**Post Opt-In**	**U.S.**	**Ex-U.S.**
verve THERAPEUTICS	Verve leads and funds development through opt-in decision	Opt-in package delivered to Lilly after last patient dosed in Phase 1 study	Verve leads worldwide development and pays for 67% of worldwide development costs	Verve books worldwide sales Verve pays for 50% of commercialization expenses and retains 50% of U.S. profits	Verve holds full rights to ex-U.S. commercialization
Lilly		Lilly has a specified number of months to opt-in once package has been delivered	Lilly pays for 33% of worldwide development costs	Lilly pays for 50% of commercialization expenses Lilly receives 50% of U.S. profits	Lilly is eligible to receive low- to mid-single-digit royalties as well as milestone payments

Assuming Lilly Opt-In

If Lilly elects not to opt-in, Lilly is eligible to receive low- to mid-single-digit royalties as well as milestone payments.

Multiple anticipated milestones for the PCSK9 program in 2025



PCSK9, proprotein convertase subtilisin/kexin type 9
1. Subject to regulatory clearance

ANGPTL3 program: VERVE-201

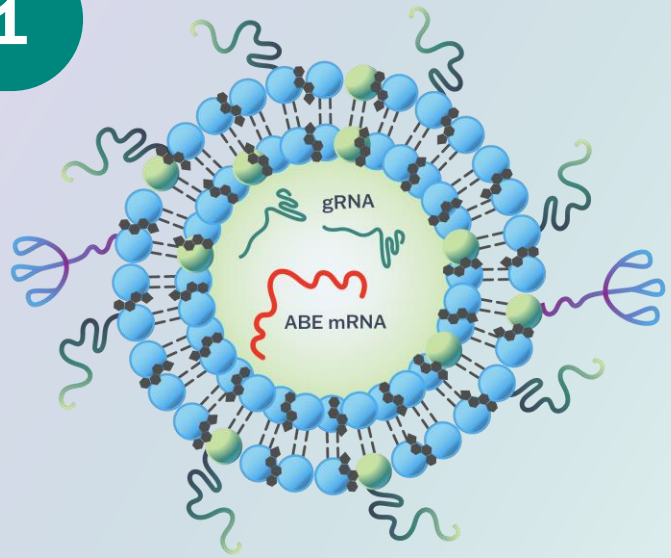
~4M addressable patients (3K HoFH + 4M refractory hypercholesterolemia) in the U.S./EU[1,2]

ANGPTL3, angiopoietin-like protein 3; LDL, low-density lipoprotein; TRL, triglyceride rich lipoprotein; Lp(a), lipoprotein(a)

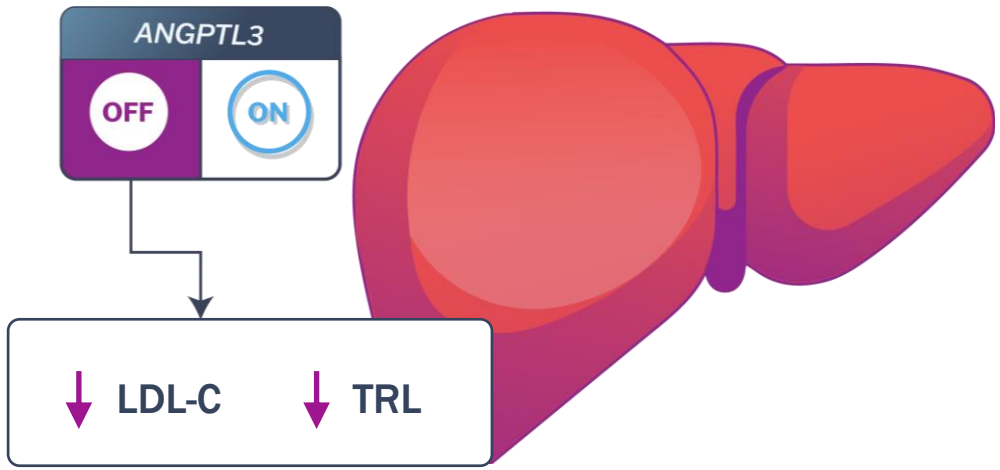
1. Cuchel M et al. Eur Heart J 2023;44: 2277-2291.
2. O'Donoghue ML et al. Circulation 2022; 146: 1109-1119.

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

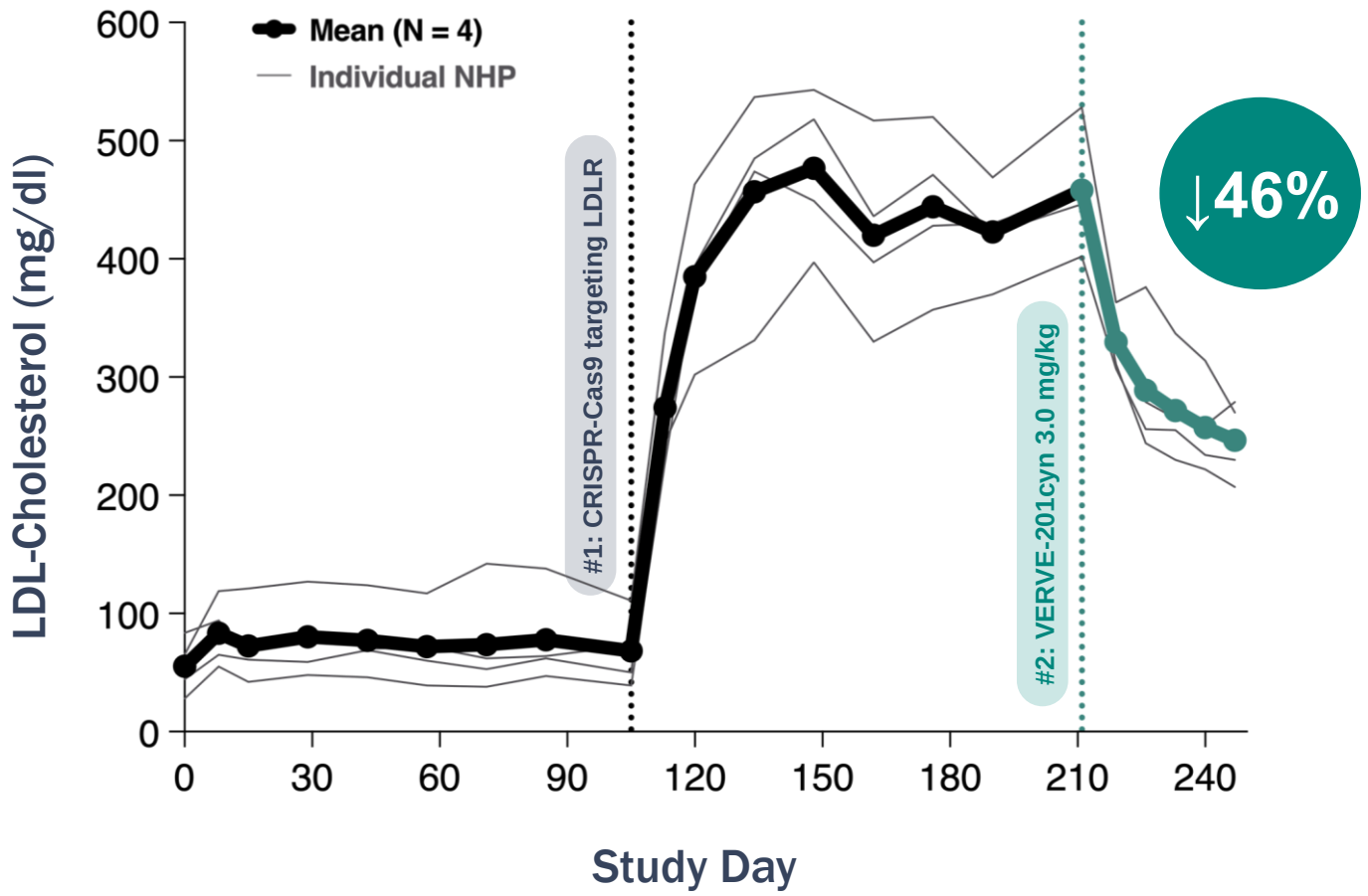
> 10 million

PREVALENCE (U.S. + EU)

ANGPTL3, angiopoietin-like protein 3; ASCVD, atherosclerotic cardiovascular disease; FH, familial hypercholesterolemia; HTG, hypertriglyceridemia; LDL-C: low-density lipoprotein cholesterol; TG, triglycerides

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.

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 SUMMARY

- 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:** Safety and tolerability
- Pharmacokinetics of VERVE-201
- 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.

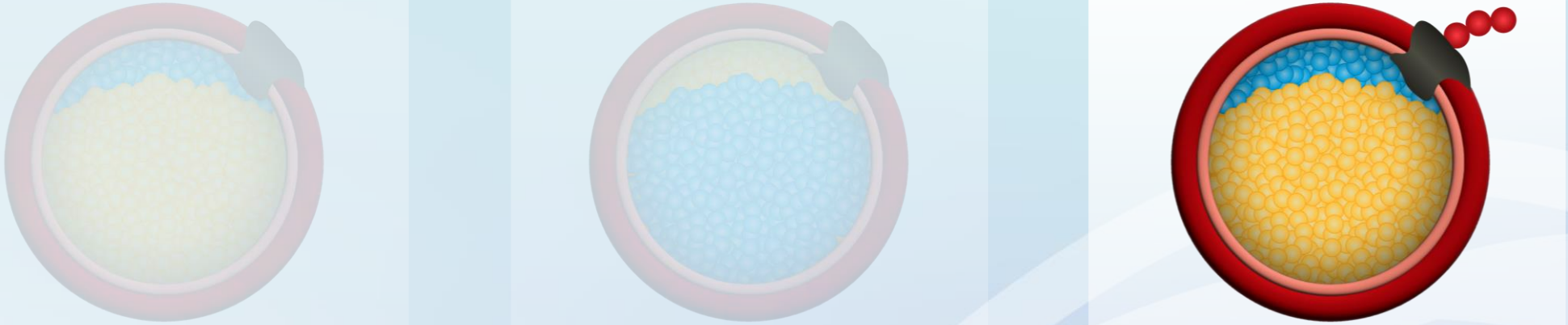
Lp(a) program: VERVE-301

> 13M addressable patients with high levels of Lp(a) in the U.S./EU[1]

LDL

TRL

Lp(a)



VERVE-102 (PCSK9)

VERVE-201 (ANGPTL3)

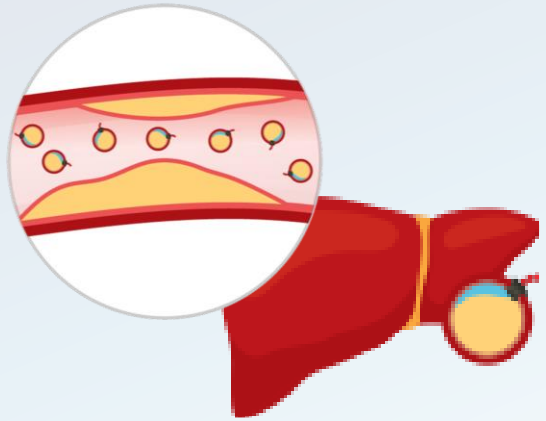
VERVE-301 (LPA)

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

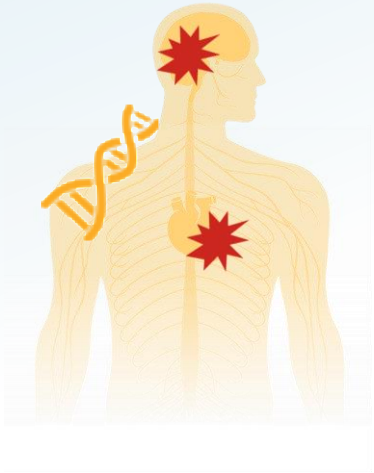
PCSK9, proprotein convertase subtilisin/kexin type 9; ANGPTL3, angiopoietin-like protein 3; LDL, low-density lipoprotein; TRL, triglyceride rich lipoprotein; Lp(a), lipoprotein(a)

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

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 expected 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

Overview of Eli Lilly collaboration for *LPA* program

	Verve	Lilly
CURRENT	Verve is responsible for all research activities and Phase 1 clinical development	Lilly will reimburse research expenses and Phase 1 clinical development expenses
	Completion of Phase 1	
VERVE NO OPT-IN	Verve is eligible to receive milestone and royalty payments (high single and low double digit royalties on global net sales)	Lilly controls development after Phase 1 and subsequent commercialization
VERVE YES OPT-IN	Verve allocated 40% of cost and margin share, in lieu of milestone and royalty payments and subject to payment of opt-in fee	Lilly controls development after Phase 1, subsequent commercialization, and retains 60% of the cost and margin share

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