



Base-Editing and Gene-Silencing Approaches to Lipid Lowering: A One-Off Treatment and a Lifetime of Consent

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Introduction

For three decades, lowering low-density lipoprotein cholesterol (LDL-C) has meant a drug the patient keeps taking: a daily statin, a twice-monthly injectable antibody, or -- most recently -- a twice-yearly small interfering RNA (siRNA) injection. Two technologies now promise something categorically different. In vivo CRISPR base editing aims to permanently disable the PCSK9 gene in the liver with a single intravenous infusion, so that no further treatment, and no further adherence, is ever required. Long-acting gene silencing with siRNA -- already approved and in wide use as inclisiran -- achieves a similar reduction in circulating PCSK9 protein without touching the gene itself, and its effect fades if the patient stops returning for injections. Both approaches target the same protein and report broadly similar percentage reductions in LDL-C, which makes it tempting to treat them as interchangeable options separated only by dosing schedule.

This review argues that framing is incomplete. A one-time genomic edit and a reversible, re-dosed drug are not the same kind of medical decision, even when their headline efficacy numbers look alike, and the honest complication in this literature is not a safety statistic but a consent problem: an irreversible change made once, on the promise of a lifetime of benefit, cannot lean on the same evidence base -- or the same conversation with the patient -- as a drug the patient can stop tomorrow.

We read the two base-editing trials of VERVE-101 and VERVE-102 alongside the inclisiran phase 3 and long-term extension literature, and alongside a qualitative bioethics study of stakeholder attitudes toward somatic gene editing in inherited cardiomyopathy, to ask what a cardiologist recommending either approach -- and a patient consenting to either -- should actually be told.

Material and Methods

This is a narrative review drawing on the primary and press disclosures of VERVE-101 (Heart-1) and VERVE-102 (Heart-2), the phase 3 ORION-10 and ORION-11 inclisiran trials, the ORION-3 four-year open-label extension, the ongoing VICTORION-2 Prevent cardiovascular outcomes trial design paper, corporate and trade-press reporting on the VERVE-101 safety halt and the Lilly-Verve acquisition, and a qualitative interview study of stakeholder moral attitudes toward somatic cardiac gene editing, identified through the sources cited here and citation tracking between them, without a pre-registered protocol or systematic search string. All percentages, dose levels, patient counts, and follow-up durations reported here are taken directly from the cited sources; none has been invented, estimated, or extrapolated. Where a base-editing efficacy figure comes from a company press release rather than a peer-reviewed paper, that is stated explicitly in the text.

Results

VERVE-101: first-in-human proof of concept, and the safety signal that halted it

VERVE-101 (Heart-1) was an open-label phase 1b trial of a single intravenous infusion of a CRISPR base editor in ten patients with heterozygous familial hypercholesterolemia (HeFH) and established atherosclerotic cardiovascular disease, uncontrolled despite maximal oral therapy, with a mean baseline LDL-C of 193 mg/dL [1]. Across four dose cohorts (0.1, 0.3, 0.45, and 0.6 mg/kg), the two higher doses produced dose-dependent, durable reductions: at 0.45 mg/kg, PCSK9 fell by 59% and 84% in two patients with corresponding LDL-C reductions of 39% and 48%; at 0.6 mg/kg, PCSK9 fell 47% and LDL-C fell 55%, durable to 180 days [1]. Figure 1 sets this dose-response alongside VERVE-102's larger cohort.

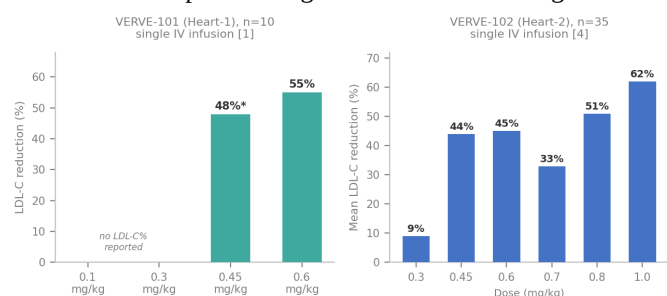


Figure 1: LDL-C reduction by dose in the two base-editing trials reviewed here [1,4].

Safety at the lower doses was unremarkable, but higher doses produced transient infusion reactions and transient liver enzyme elevations, and the trial recorded two serious events whose relationship to treatment required careful adjudication: a fatal cardiac arrest about five weeks after dosing in the 0.3 mg/kg cohort, judged unrelated to treatment, and a myocardial infarction the day after dosing in the 0.45 mg/kg cohort, considered potentially treatment-related given its timing [1]. The event that actually halted enrollment came later: one patient dosed at 0.45 mg/kg developed a grade 3 elevation in a liver transaminase and grade 3 thrombocytopenia, was hospitalized for two days of observation while remaining asymptomatic, and had both abnormalities resolve independently with no bleeding complications [2]. Verve halted enrollment, notified regulators, and consulted the trial's data monitoring committee before deciding not to continue VERVE-101 as designed [2].

VERVE-102: a reformulated program, a cleaner safety readout, and a still-short follow-up

Rather than continuing VERVE-101, Verve redesigned the base editor's delivery: VERVE-102 pairs the same PCSK9-targeting edit with a new ionizable-lipid, GalNAc-targeted lipid nanoparticle intended to improve hepatic delivery and reduce the exposure associated with the earlier formulation's safety signal [3]. An interim readout of the resulting Heart-2 trial in 14 patients (0.3, 0.45, and 0.6 mg/kg) reported time-averaged LDL-C reductions from day 28 of 21%, 41%, and 53% respectively, with a maximum individual reduction of 69%, no treatment-related serious adverse events, no dose-limiting toxicities, and no clinically significant change in liver enzymes or platelet counts [5]. The full 35-patient Heart-2 cohort, published in the New England Journal of Medicine and presented at the European Atherosclerosis Society Congress in May 2026, extended dosing to six cohorts up to 1.0 mg/kg and reported mean LDL-C reductions of 9%, 44%, 45%, 33%, 51%, and 62% by ascending dose, with PCSK9 reductions ranging from 51% to 88% [4,11]. No treatment-related serious adverse events or dose-limiting toxicities were reported in this larger cohort; adverse events were limited to low-grade infusion reactions and fatigue, and all participants completed their planned dose [4].

The detail that matters most for this review is how little of that cohort has been followed for long: at the 27 February 2026 data cutoff, only 15 of the 35 participants had reached one year of follow-up, with a median follow-up across the cohort of just nine months [4,5,11]. Figure 2 places that follow-up duration against inclisiran's much longer track record, and Figure 3 places the headline efficacy numbers from every trial reviewed here on a single comparative scale.

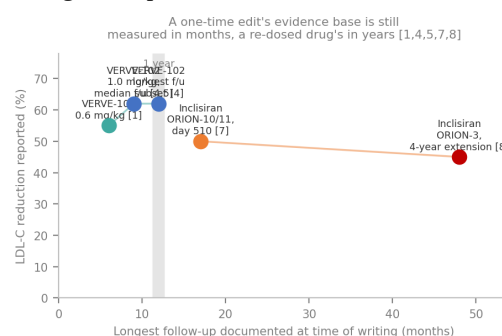


Figure 2: Longest follow-up documented for each agent at the time of writing, plotted against the LDL-C reduction reported at that point [1,4,5,7,8].

It is worth being explicit about what Figure 2 does and does not show. The months plotted are the longest follow-up publicly reported for each agent at the time of writing, not a guarantee that every patient on that agent has been followed that long, and the assays, dosing schedules, and patient populations behind each point differ enough that the figure

should be read as an illustration of the maturity gap between the two technologies rather than as a precise, apples-to-apples survival curve [1,4,5,7,8]. Even with that caveat, the gap itself is large enough to matter clinically: a factor of roughly five between VERVE-102's longest reported follow-up and ORION-3's four completed years, and a factor of eight between VERVE-102's own median follow-up and that same four-year mark [4,5,8].

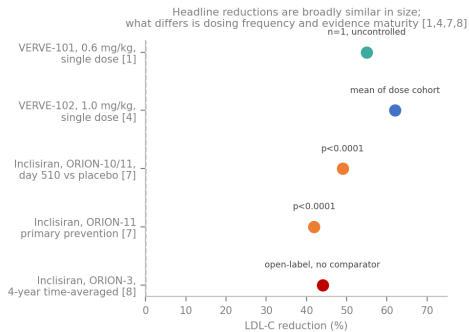


Figure 3: Headline LDL-C reductions across all trials reviewed, base editing and gene silencing together [1,4,7,8].

The dose-response curve within the full Heart-2 cohort is itself worth reading carefully rather than reducing to its single highest number: mean LDL-C reduction rose from 9% at 0.3 mg/kg to 44-45% at 0.45-0.6 mg/kg, but then fell to 33% at 0.7 mg/kg before rising again to 51% and 62% at 0.8 and 1.0 mg/kg [4]. A non-monotonic step of that size, in cohorts of only four to six patients per dose level, is exactly the kind of noise a phase 1b dose-escalation study is designed to characterize rather than to explain away, and it is a reminder that the 62% figure quoted throughout this review is a single dose cohort's mean, not a reduction every future patient should expect from a fixed 1.0 mg/kg dose [4].

Eli Lilly agreed to acquire Verve Therapeutics for \$1.3 billion in June 2025, before the full Heart-2 data were public, describing VERVE-102 as a potential first in vivo gene-editing therapy for broad cardiovascular populations and explicitly framing the goal as converting chronic disease management into a single intervention [6]. Figure 4 sets that acquisition inside the same timeline as the VERVE-101 halt and the VERVE-102 readout, because the commercial and clinical narratives are not separable here: a company changed hands, in large part, on the strength of a dataset with a nine-month median follow-up.

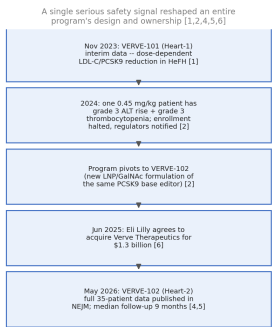


Figure 4: From VERVE-101's safety halt to VERVE-102's full data and the Lilly acquisition: one program's timeline [1,2,3,4,5,6].

Inclisiran: the reversible comparator, and its own unfinished business

Inclisiran achieves a comparable reduction in circulating PCSK9 by triggering RNA interference against the PCSK9 messenger RNA in hepatocytes, leaving the PCSK9 gene itself untouched; the effect is sustained only by re-dosing, twice yearly after an initial loading dose [7]. The pooled phase 3 ORION-10 and ORION-11 trials randomized 1,561 and 1,617 patients respectively with atherosclerotic cardiovascular disease or an equivalent risk, and reported an LDL-C reduction of 49-51% versus a 1-4% rise with placebo at day 510 ($p<0.0001$); a smaller primary-prevention subgroup within ORION-11 ($n=203$) showed a similar 41.9% reduction versus a 1.8% placebo rise [7]. Treatment-emergent adverse events were similar between arms (83% versus 82%), as were serious adverse events (22% versus 23%); the only clearly drug-attributable signal was a protocol-defined injection-site "skin event" in 4.7% of inclisiran-treated patients versus 0.5% on placebo, with no signal of liver, kidney, or muscle toxicity [7]. An exploratory, non-powered cardiovascular event rate of 7.8% versus 10.3% was reported but was not a formal trial endpoint [7].

The dosing schedule itself is part of what makes inclisiran a reversible therapy in a way neither VERVE-101 nor VERVE-102 can be: patients receive an initial subcutaneous dose, a second dose at three months, and then a maintenance dose every six months thereafter, so the 49-51% reduction at day 510 in ORION-10 and ORION-11 reflects three cumulative doses rather than a single administration [7]. Each subsequent dose is itself a renewed decision point: a patient who develops a concerning symptom, a new comorbidity, or simply changes their risk-tolerance can decline the next scheduled injection, something a base-edited patient has no equivalent option to do once the single infusion has been given [7]. That the treatment-emergent and serious adverse event rates were essentially identical between inclisiran and placebo (83% versus 82%, and 22% versus 23%, respectively) is itself a consequence of the drug's long track record and large denominator -- 1,561 and 1,617 randomized patients across the two trials, an order of magnitude larger than either base-editing cohort discussed here [1,4,7].

Inclisiran's own durability data are considerably longer than either base-editing trial's: the ORION-3 open-label extension followed roughly 500 patients from the earlier ORION-1 trial for four years of continued twice-yearly dosing and reported a sustained time-averaged LDL-C reduction of approximately 44-45%, achieved with 8 to 9 total injections and with no new safety signal beyond the same mild, transient injection-site reactions seen in the pivotal trials [8]. Table 1 sets the design and headline result of all four trials out together.

Trial	N	Modality	Comparator	Headline LDL-C result
VERVE-101 (Heart-1) [1]	10	Base editing, IV	None (open-label)	-55% at 0.6 mg/kg, durable to 180d
VERVE-102 (Heart-2) [4]	35	Base editing, IV	None (open-label)	-62% mean at 1.0 mg/kg
ORION-10/11 [7]	1561/1617	siRNA, SC, twice-yearly	Placebo	-49% to -51% at day 510 (p<0.0001)
ORION-3 (4-yr ext.) [8]	~500	siRNA, SC, twice-yearly	None (open-label)	~44-45% time-averaged over 4 years

Table 1: Design and headline efficacy of the four trials reviewed here [1,4,7,8].

Yet inclisiran's own hardest question remains open too: nearly six years after its pivotal efficacy trials, a dedicated cardiovascular outcomes trial, VICTORION-2 Prevent, is still recruiting and has not yet reported whether the drug reduces major adverse cardiovascular events, as distinct from LDL-C itself [9]. Gene silencing is the reversible, better-studied technology of the two reviewed here, but even it has not closed every evidentiary gap that matters to a patient deciding whether to start it.

What an honest consent conversation has to cover

A qualitative interview study of 18 stakeholders in the Netherlands -- patients, clinicians, researchers, and regulators -- examining somatic gene editing for inherited cardiomyopathy (a different cardiac indication, but the same consent problem as PCSK9 base editing) found broad conditional optimism, framed as "the right treatment for the right person at the right time", but also identified two consent-specific risks that a purely efficacy-focused review would miss [10]. First, severely affected patients' evident enthusiasm for an early-phase, irreversible intervention may not reflect fully free voluntariness when few or no alternatives exist, a tension the study's authors describe as optimism and limited alternatives together putting voluntariness "under pressure" [10]. Second, the same optimism creates a risk of therapeutic misconception or misestimation, in which patients overestimate direct clinical benefit and underestimate risk precisely because a one-time, curative-sounding intervention is being offered in place of a manageable chronic condition [10]. Figure 5 sets out why these two mechanisms -- permanent DNA change versus reversible RNA silencing -- are not interchangeable from a consent standpoint even when their percentage reductions in LDL-C look similar, and Figure 6 turns that contrast into the specific questions a consent conversation for each approach needs to answer.

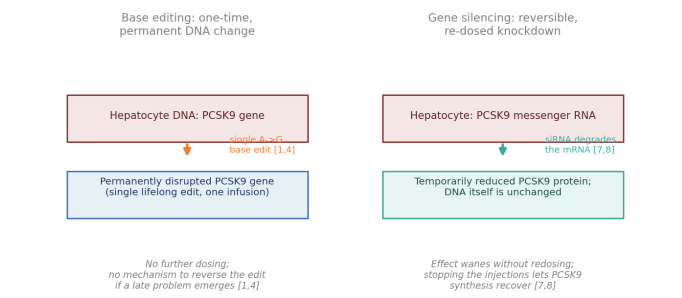


Figure 5: Mechanistic contrast: a permanent, one-time DNA edit versus a reversible, re-dosed RNA knockdown [1,4,7,8].

A one-time edit and a re-dosed drug raise different consent questions [1,4,5,7,8,9,10]

Consent question	Base editing (one-time)	Gene silencing (re-dosed)
Can treatment be stopped or reversed?	No -- edit is permanent [1,4]	Yes -- effect wanes off-drug [7,8]
Longest human follow-up available today	~1 year (subset) [4,5]	4 years (ORION-3) [8]
Hard CV outcome trial reported?	No -- not yet powered/run [1,4]	Not yet -- MACE trial ongoing (VICTORION-2 Prevent) [9]
Risk of therapeutic misconception	Flagged by stakeholders as a familiar drug consent framework applies [10]	

Figure 6: What an honest consent conversation must cover for a one-time base edit versus a re-dosed siRNA, drawn from the trial and stakeholder literature reviewed here [1,4,5,7,8,9,10].

Discussion

Read only for efficacy, base editing and gene silencing of PCSK9 look like two roads to the same destination: both push LDL-C down by roughly 40-60% relative to baseline, both target the same protein, and both are being pursued because daily-adherence therapies fail a meaningful fraction of patients over time -- a point made explicitly by commentators on the VERVE-102 data, who note that roughly half of patients discontinue conventional lipid-lowering therapy within a year [5]. That adherence problem is real, and it is the strongest honest argument for a one-time intervention. But the two approaches are not equivalent as decisions a patient is asked to make. Gene silencing is reversible in a meaningful clinical sense: a patient who develops a late problem, or who simply changes their mind, can stop returning for injections and the drug's effect wanes. A base-edited patient has no equivalent exit -- the edit made in a single infusion is, by design, permanent, and no mechanism exists to reverse it if a problem is discovered five, fifteen, or forty years later [1,4].

VERVE-101's own history illustrates why that asymmetry matters in practice, not just in principle: a single patient's grade 3 laboratory abnormalities, though they resolved without sequelae, were enough to halt an entire trial's enrollment and trigger a wholesale reformulation of the delivery vehicle [2]. That is exactly the correct response to a safety signal in a reversible-drug trial -- pause, investigate, redesign. But it also demonstrates that even a company running this program in real time did not, and could not, know in advance which formulation would prove safe; the patients dosed with

VERVE-101 carry whatever edit was made to their genome regardless of what was later learned. A drug trial that halts for safety leaves its patients able to simply stop the drug. A gene-editing trial that halts for safety leaves its patients with the edit already made.

The durability numbers in Figure 2 make the same point quantitatively. Fifteen of thirty-five VERVE-102 participants have reached one year of follow-up; the ORION-3 extension has four years of continuous data on roughly five hundred patients, gathered specifically because a re-dosed drug's long-term safety has to be demonstrated dose after dose, not proven once and assumed forever [4,5,8]. A patient's remaining lifetime after a base-editing procedure performed in their forties or fifties may span four or five decades; the entire published human evidence base for VERVE-102's durability currently spans about one fiftieth of that horizon. This is not a criticism of the trial design -- no phase 1b study could ethically or practically enroll patients for decades before reporting -- but it is a fact the consent process has to state plainly rather than let a "one-time, lifelong" framing imply an assurance the data do not yet support [5].

The stakeholder-interview findings on somatic cardiac gene editing generalize directly to this PCSK9 setting: the same optimism that makes an irreversible, curative-sounding intervention attractive to patients with few alternatives is the same optimism that risks eliding the distinction between a promising phase 1b signal and a guaranteed lifetime benefit [10]. Cardiologists discussing VERVE-102, or any future base-editing therapy that reaches broader practice, should treat that distinction as a required, not optional, part of consent -- alongside the more familiar disclosures about injection-site reactions and periodic monitoring that already accompany a drug like inclisiran. Notably, even inclisiran, the more mature and reversible of the two technologies, has not yet produced the one number that would settle the clinical argument for either approach: a completed, adequately powered cardiovascular outcomes trial. VICTORION-2 Prevent's continued recruitment is a reminder that LDL-C reduction, however large or durable, remains a validated surrogate rather than a demonstrated reduction in heart attacks and strokes for either drug class discussed here [9].

Study limitations

This is a narrative review, not a systematic review with a pre-registered protocol, and its efficacy numbers for the base-editing programs rely partly on company press releases and conference presentations rather than exclusively on peer-reviewed publication, because that is the current state of the public record for VERVE-101 and much of VERVE-102's interim data [1,3,5]; the full VERVE-102 Heart-2 cohort has since been published in a peer-reviewed journal and is treated as the primary source where the two diverge [4]. Both base-editing trials reviewed here are small, open-label, and uncontrolled phase 1b studies without a sham-controlled comparator, so the LDL-C reductions reported, while internally consistent and dose-dependent, have not been tested against placebo the way inclisiran's have [1,4]. The one-year and four-year follow-up figures quoted in Figure 2 describe different patient populations, indications for dosing, and assay methods across trials, so they should be read as an illustration of the maturity gap between the two technologies rather than as a head-to-head comparison of equivalent cohorts [1,4,5,7,8]. The stakeholder-interview study cited for the consent discussion examined somatic gene editing for inherited cardiomyopathy, not PCSK9 base editing specifically, and its 18 participants were all recruited in the Netherlands, so its findings on voluntariness and therapeutic misconception are drawn from a related but distinct clinical and cultural context and should be read as a transferable hypothesis about consent dynamics rather than as direct empirical data on VERVE-101 or VERVE-102 patients themselves [10].

A further limitation worth stating plainly: neither base-editing trial reviewed here was powered, or designed, to detect a difference in hard cardiovascular outcomes, and neither has followed patients anywhere near long enough to characterize late off-target effects, clonal evolution in edited hepatocytes, or other theoretical harms specific to a permanent genomic change, as opposed to a temporary pharmacologic one [1,4]. Nothing in the published data reviewed here suggests such harms have occurred; the point is narrower and more basic -- the follow-up windows involved (180 days for VERVE-101's longest reported readout, a median of nine months for VERVE-102) are simply too short to have detected a harm that took longer than that to emerge, even if one existed [1,4,5]. Readers should also note that the VERVE-101 safety events -- one unrelated fatal cardiac arrest, one potentially related myocardial infarction, and the grade 3 laboratory abnormalities that halted enrollment -- occurred in a trial of only ten patients, a sample far too small to estimate a true event rate for any of them with precision, which cuts in both directions: it should discourage over-interpreting a single alarming event, but it should equally discourage over-interpreting the subsequent clean safety readout from VERVE-102's larger but still modest 35-patient cohort as proof the underlying risk has been resolved [1,2,4].

Conclusion

Base editing and gene silencing of PCSK9 both lower LDL-C by a clinically meaningful margin, and both are genuine responses to a real adherence problem with existing therapy. But they differ in a way that percentage reductions cannot capture: one is a single, irreversible change to a patient's genome, evaluated so far over months, while the other is a reversible, re-dosed drug evaluated over years and still awaiting its own definitive outcomes data. Until base-editing therapies for lipid lowering accumulate follow-up measured in years rather than months, informed consent for them should say so explicitly, distinguishing a promising early safety and efficacy signal from a settled, lifelong guarantee -- a distinction the bioethics literature on somatic gene editing suggests patients and clinicians alike find genuinely hard to hold onto once a cure is on offer.

Clinical bottom line

For a cardiologist counseling a patient today, the numbers reviewed here support three practical statements rather than one simple recommendation. First, on efficacy alone, the two technologies are genuinely comparable: the highest reported reductions -- 55% for VERVE-101 at 0.6 mg/kg, 62% for VERVE-102 at 1.0 mg/kg, and 49-51% for inclisiran at day 510 -- sit within the same broad range, and none of them should be presented to a patient as obviously superior to the others on LDL-C lowering alone [1,4,7]. Second, on evidence maturity, the technologies are not comparable: inclisiran carries four years of continuous open-label follow-up in hundreds of patients and a completed, published, placebo-controlled phase 3 program, while VERVE-102's entire published human experience, at the time of writing, covers 35 patients with a median follow-up of nine months [4,7,8]. Third, neither drug class has yet answered the question patients usually care about most -- whether lowering LDL-C this way prevents a heart attack or stroke -- since VICTORION-2 Prevent remains open for inclisiran and no comparable outcomes trial of either VERVE compound has been reported [9].

None of this is a reason to withhold either therapy from patients who may benefit, particularly patients with genetically driven, difficult-to-treat hypercholesterolemia and poor adherence to daily oral therapy, the population both technologies were designed for [1,4,7]. It is a reason to keep the consent conversation honest about which parts of the story are settled and which parts are still being written. A patient told that a single infusion will permanently fix their cholesterol deserves to also be told, in the same conversation, that the therapy's only human safety experience beyond a year comes from fifteen people, and that the mechanism gives that patient no ability to opt out later the way stopping an injection would allow. That asymmetry, more than any single efficacy or safety

number in the tables and figures above, is what a one-time treatment asks a patient to accept on trust.

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