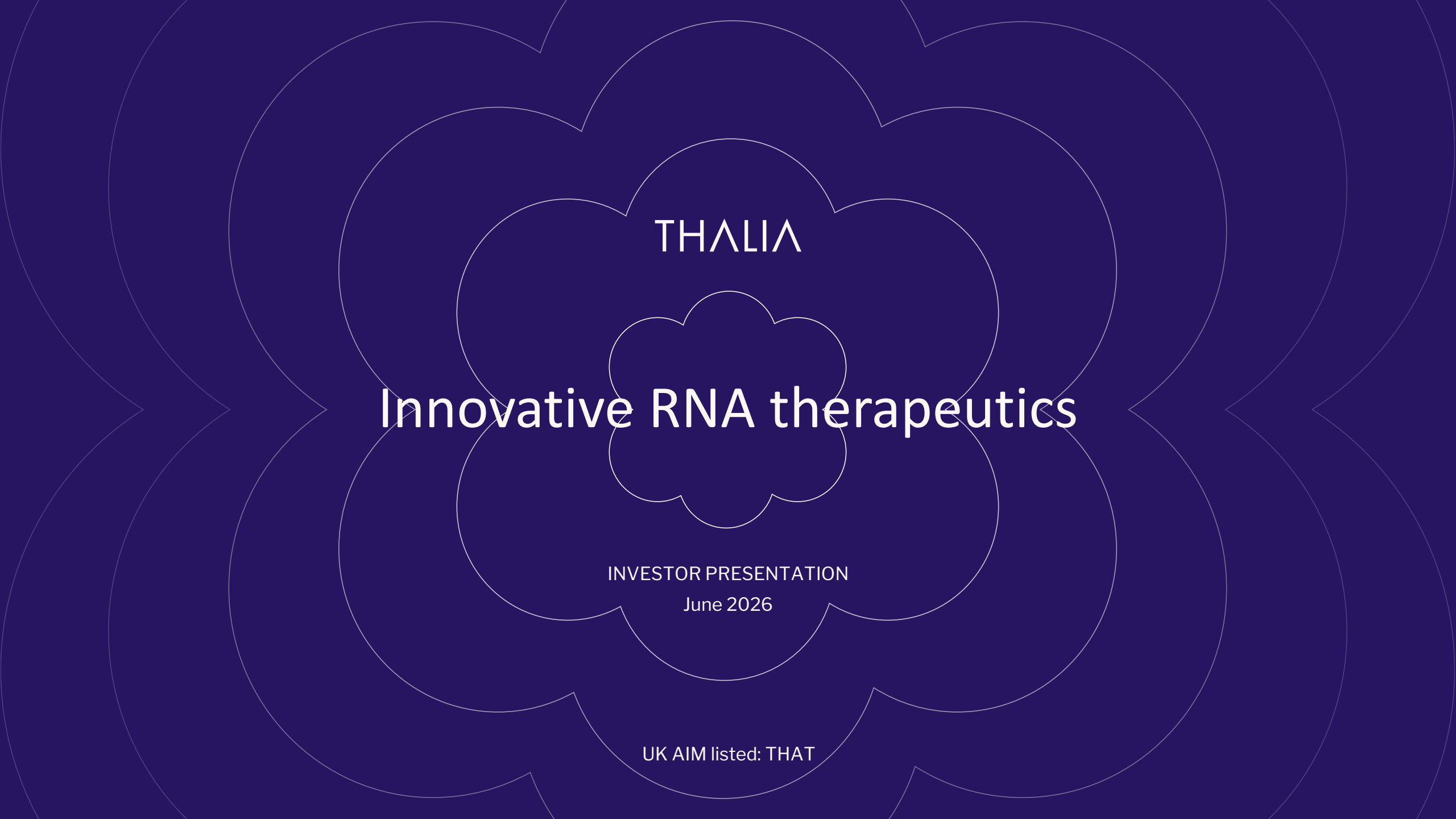




THALIA

Innovative RNA therapeutics

INVESTOR PRESENTATION

June 2026

UK AIM listed: THAT

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THALIA

Ancient Greek origin, meaning to flourish or bloom

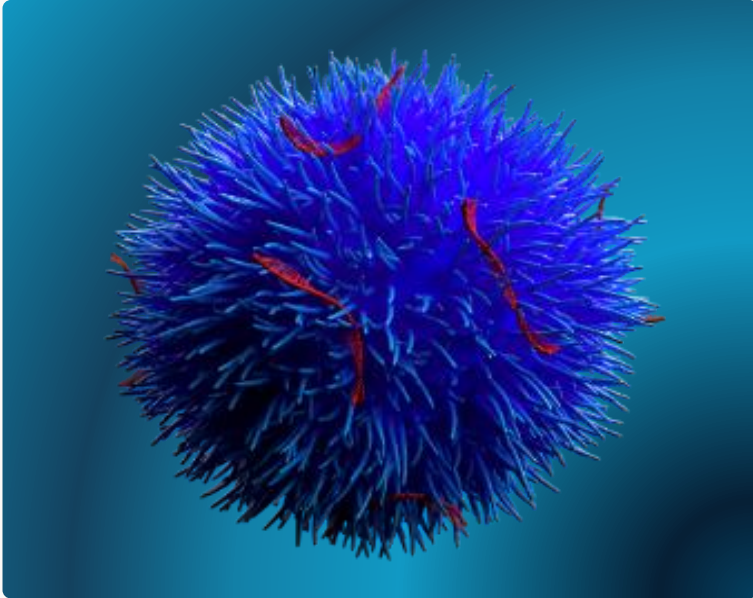
Building a new RNA therapeutics leader

A diversified portfolio of innovative RNA therapeutics
to treat cancer and cardiovascular disease

Led by experienced management
with proven track records in RNA therapeutics

LSE AIM listed: THAT

Acquisition transforms Thalia into clinical stage company and emerging RNA therapeutics pioneer



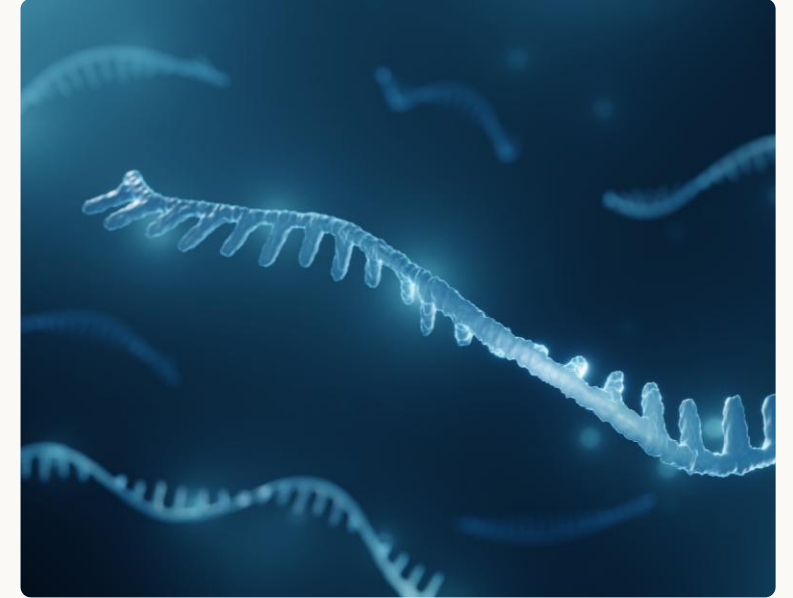
Targeted delivery - Nuvec®

- Versatile drug delivery system with many advantages over lipid or viral approaches
- Optimizes proprietary pipeline
- Out-licensing potential



siRNA

- siRNA silences specific genes that cause disease
- Thalia is developing a bispecific siRNA
- Addresses two key pathological targets of atherosclerotic cardiovascular disease (Lp(a) and PCSK9), thus reducing cardiovascular risk



miRNA

- miRNA regulates gene expression
- miRisten is an anti-miR-126 therapeutic
- Targets the root cause of acute myeloid leukemia (AML)

Transformational acquisition with significant value creation potential

Adds clinical stage asset in new RNA therapeutic modality opening significant market opportunity

SANMIRNA ACQUISITION

miRisten, a clinical stage RNA therapeutic for rare disease acute myeloid leukemia (AML)

- Differentiated Phase 1 asset (City of Hope) targeting the root cause of AML
- Clinical asset is a **key valuation driver** for public markets
- Acquisition and milestone structure minimizes dilution to existing shareholders



£2.75M OVERSUBSCRIBED FUNDRAISE

strengthens Thalia's balance sheet to continue executing strategy

- Funds pipeline development through miRisten Phase 1 readout in H1 2027
- Funds IND-enabling studies of Thalia's own assets
- Enables shareholders to participate in long term value-creation



KEY STEP IN TRANSITION

to RNA therapeutic leadership

- Transforms Thalia into clinical stage RNA company
- Adds second asset in pipeline of differentiated, validated therapeutics in a new class of RNA therapeutics



MYELOID NEOPLASIA

Pharmacological inhibition of miR-126 enhances venetoclax activity in acute myeloid leukemia

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¹Department of Hematologic Malignancies Translational Science, Gehl Family Center for Leukemia Research, Beckman Research Institute, City of Hope National Medical Center, Duarte, CA; ²Early Detection and Prevention Division, Translational Genomics Research Institute, Phoenix, AZ; ³Integrated Mass Spectrometry Shared Resource, City of Hope National Medical Center, Duarte, CA; ⁴Department of Nutritional Science, Dell Pediatric Research Institute, The University of Texas at Austin, Austin, TX; ⁵Irell and Manella Graduate School of Biological Sciences, Beckman Research Institute, City of Hope, Duarte, CA; ⁶Department of Systems Biology, Beckman Research Institute, City of Hope, Monrovia, CA; and ⁷Hematology Malignancies Research Institute, Gehl Family Center for Leukemia Research, Beckman Research Institute, City of Hope National Medical Center, Duarte, CA; ⁸Division of Hematology and Hematopoietic Cell Transplantation, City of Hope National Medical Center, Monrovia, CA.

KEY POINTS

- **miR-126 enhances BCL-2 signaling to sustain FAO/OXPHOS and mitofusion, promoting metabolic homeostasis and leukemogenic activity in AML LSCs.**
- **miRisten, an anti-miR-126 inhibitor, synergizes with the BCL-2 inhibitor VEN to eliminate LSCs and sensitize to VEN.**

KEY POINTS

- miR-126 enhances BCL-2 signaling to sustain FAO/OXPHOS and mitofusion, promoting metabolic homeostasis and leukemogenic activity in AML LSCs.
- miRisten, an anti-miR-126 inhibitor, synergizes with the BCL-2 inhibitor VEN to eliminate LSCs and sensitize to VEN.

Leukemic stem cells (LSCs) in acute myeloid leukemia (AML) depend on oxidative phosphorylation (OXPHOS) sustained by fatty acid oxidation (FAO) and mitochondrial fusion (mitofusion). In this study, we demonstrate that microRNA-126 (miR-126) maintains LSC function by promoting B-cell lymphoma 2 (BCL-2)-dependent FAO, OXPHOS, and mitofusion, whereas its inhibition disrupts mitochondrial metabolism, induces mitochondrial fission (mitofission), and triggers apoptosis. Mechanistically, miR-126 stabilizes BCL-2 through the SPRED1/extracellular signal-regulated kinase axis, which upregulates CPT1B (FAO) and NRF2 (antioxidant response) while regulating mitochondrial dynamics through DRP1 phosphorylation (inhibiting mitofission) and MFN1/2 phosphorylation (enhancing mitofusion). miRisten, a CpG-conjugated anti-miR-126 oligonucleotide used in clinical trials (NCT07025564), synergized with venetoclax (VEN) to suppress FAO/OXPHOS, promote mitofission, and impair LSC homeostasis. In vivo, miRisten potentiated the VEN/azacitidine regimen, an US Food and Drug Administration-approved therapy for older or unfit patients with AML, significantly prolonging survival in patient-derived xenograft models. VEN/miRisten combination also reduced LSC burden and restored VEN sensitivity, establishing miR-126 inhibition as a transformative therapeutic strategy for AML.

Introduction

Acute myeloid leukemia (AML) is a complex hematologic malignancy driven by leukemia-initiating cells, or leukemia stem cells (LSCs). This primitive subset of leukemic cells has self-renewal capacity and sustains disease growth indefinitely. Because of their high treatment resistance, LSCs are considered one of the root causes of therapy failure in AML.^{1,2} Targeting and eliminating LSCs is therefore essential for a cure. Unlike normal hematopoietic stem cells (HSCs), LSCs primarily rely on mitochondrial oxidative phosphorylation (OXPHOS) to maintain their activity.³⁻⁸ Mitochondrial dynamics, including fission and fusion processes, are crucial for maintaining survival and treatment resistance of LSCs. Despite elevated OXPHOS, LSCs remain quiescent with low reactive oxygen species (ROS) and high expression of antiapoptotic protein B-cell lymphoma 2 (BCL-2), an emergent therapeutic target in AML.⁹

Venetoclax (VEN), a BH3-inhibitor of BCL-2, is US Food and Drug Administration (FDA)-approved with hypomethylating agents (HMA) for AML treatment.¹⁶ Beyond its proapoptotic activity, VEN/HMA reduces OXPHOS by decreasing succinate dehydrogenase glutathionylation, thereby inhibiting electron transport chain complex II, which helps lower LSCs burden.^{11,12} However, VEN/HMA therapy is not curative, and most patients relapse without subsequent allogeneic stem cell transplantation.¹³ Reported mechanisms of VEN resistance include upregulation of other antiapoptotic proteins (eg, MCL-1,¹⁴⁻¹⁶ TP53 mutations,¹⁷⁻¹⁹ and increased fatty acid oxidation (FAO) and amino acid metabolism that sustain OXPHOS.²⁰⁻²⁴

MicroRNAs are short noncoding RNAs that regulate target mRNA and protein expression. Aberrant microRNA expression occurs in AML and contributes to LSC self-renewal, maintenance, and therapy resistance.^{25,26} Among them,

mechanistic explanation for infection risk with BCMA-targeted bispecific antibodies. These insights should inform individualized prophylaxis and guide the design of safer immunotherapy regimens in combination. As we embrace the promise of bispecific antibodies for all patients, balancing efficacy with immune competence will be key to optimizing patient outcomes.

Conflict-of-interest disclosure: C.F.d.L. reports participation on the advisory boards of Johnson & Johnson, Bristol Myers Squibb (BMS), Amgen, Pfizer, Sanofi, BeiGene, GSK, Roche, and Menarini; honoraria from Amgen, Johnson & Johnson, BMS, GSK, Sanofi, Pfizer, BeiGene, and Miltenyi; and grants from BMS, Johnson & Johnson, Amgen, and GSK. ■

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<https://doi.org/10.1182/blood.2025032329>

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MYELOID NEOPLASIA

Comment on [Zhang et al.](#), page 1083

Tuning mitochondrial fate to disarm leukemic stem cells

Xufeng Chen | The University of Texas MD Anderson Cancer Center

In this issue of *Blood*, Zhang et al¹ report that pharmacologic inhibition of microRNA-126 (miR-126) using the CpG-conjugated anti-miR-126 oligonucleotide, named miRisten, potentially perturbs the metabolic homeostasis and leukemogenic activity of leukemic stem cells (LSCs) in acute myeloid leukemia (AML). Furthermore, miRisten resensitizes AML cells to the US Food and Drug Administration (FDA)-approved B-cell lymphoma 2 (BCL-2) inhibitor venetoclax (VEN), offering new mechanistic insight and translational promise for overcoming VEN resistance.

AML remains difficult to cure largely because therapy-resistant LSCs survive initial treatment and drive relapse.² Notably, LSCs are known to depend on mitochondrial oxidative metabolism including oxidative phosphorylation (OXPHOS) and fatty acid oxidation (FAO), to meet their bioenergetic needs, rendering them more vulnerable to mitochondrial targeting regimens, including VEN.²⁴ In fact, LSCs like AMLs are more sensitive to VEN treatment compared with monocytic AMLs,²⁵ and VEN-resistant AML cells show unique mitochondrial metabolism/structure features, making mitochondrial metabolism/structure key therapeutic targets.²⁶

Chronic therapy has identified a quiescent, chemotherapy-resistant LSC subclone that is marked by low OXPHOS signatures and high miR-126, a key regulator of LSC homeostasis.^{1,10} This suggests that miR-126 helps maintain LSC quiescence and survival in AML, thereby contributing to therapy resistance.

In this study, Zhang et al's findings provide a compelling mechanistic explanation for how miR-126 controls LSC fate. They

that milk-126 is highly expressed in AML LSCs, which effectively blocks LSCs in a prosurvival, low-reactive oxygen species state. By intersecting milk-126 with known signaling and metabolic pathways, they uncovered a regulatory network that links milk-126 to BCL-2 and lipid metabolism. In AML cells, milk-126 and milk-126 suppresses sprouty-related EVH1 domain-containing protein 1 (SPRED1), thereby driving extracellular signal-regulated kinase (ERK)/cAMP response element-binding protein (CREB)-mediated phosphorylation and stabilization of BCL-2. The elevated BCL-2 appears to activate nuclear factor erythroid 2-related factor 2 (NRF2)-dependent ubiquitination of dynamin-related protein 1 (DRP1), a GTPase that drives mitochondrial fission) and to augment the expression of carnitine palmitoyltransferase 1B (CPT1B, the rate-limiting enzyme of FAO). Therefore, milk-126 upregulates FAO while maintaining fused mitochondrial networks, which confer resistance to apoptosis. Conversely, milk-126 inhibition upregulates SPRED1, which blunts ERK activity and lowers BCL-2. This shift increases DRP1-mediated mitochondrial fission and

A transformational step to drive further value creation

Rationale for transaction

- Adds **key value driver for public markets**: clinical stage asset in new RNA therapeutic category
- **Differentiated approach** addressing root cause of rare aggressive blood cancer
- **Significant market opportunity** with high unmet need, ~22,000 new US patients annually, 33% 5-year survival
- **Accelerates Thalia's strategy** to create an RNA therapeutic leader
- **Funding required to conclude study not commence** – study already underway funded by City of Hope (CoH)

Pre-clinical to clinical Phase 1 case studies

Company	Indication	Preclinical to Phase 1	On initial data
Avidity	DM1	From ~\$0.8B To \$1.16B ~45%	From \$570M To \$1.6-1.9M 50-100%
Dyne	DM1/DMD	From \$447- \$770M >70%	From \$760M To >\$1.1B >100%
Wave	CNS/rare	From ~\$350M To ~\$470M ~35%	To \$2B+ with data in Oct 2024 6x lift

DMD: Duchenne Muscular Dystrophy; DM1: myotonic dystrophy type 1

Acquisition of Sanmirna brings miRisten asset

Key transaction terms and timeline

Consideration to Vendors	▪ Sanmirna shareholders to receive up to £16.675M, including up to £13M deferred milestone payments ▪ £3.675M initial consideration comprised of 485,107,215 shares and £764,357 convertible loan note*
miRisten asset	▪ Exclusive global development and commercialization rights to miRisten technology ▪ Global patent portfolio (compound, composition, methods of use) to 2040-45 (US) ▪ Under the license to Sanmirna, CoH will be reimbursed up to \$1.2M clinical costs within 5 years
Balance sheet strengthened	▪ £2.75M oversubscribed fundraise enables execution of strategy to value-creating events ▪ Finances completion of phase 1 miRisten trial and IND-enabling studies of Thalia bispecific siRNA
Timing	▪ Transaction approved by Thalia Board and Sanmirna Board ▪ Transaction expected to close on 17 July 2026

*Vendors limited to no more than 29.9% of Thalia's issued share capital

Value accretive merger grows RNA therapeutic pipeline

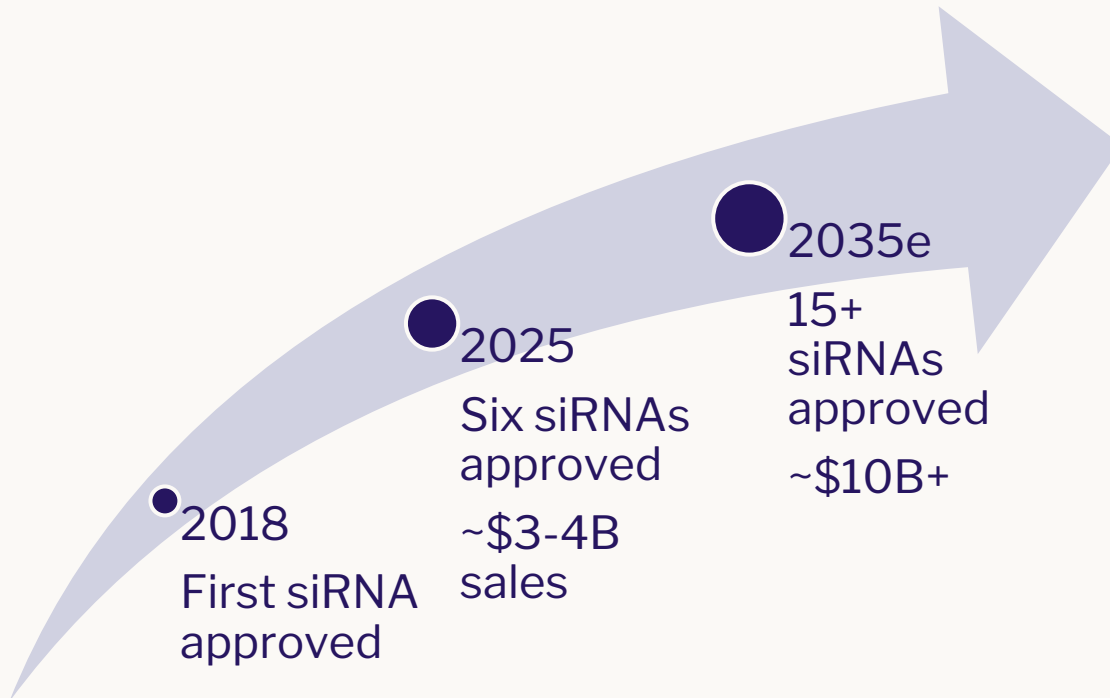
Pipeline candidate	Target	Indication	Discovery	Preclinical/ IND enabling	Phase I	Phase II-III	Milestones
miRisten (THAT-001)	Anti-miR-126	Acute myeloid leukemia (AML)					Phase 1 data H1 2027
THAT-002	LP(a)-PCSK9 – GalNAc	Atherosclerotic cardiovascular disease					Preclinical PoC H1 2027
THAT-003	LP(a)-PCSK9 – GalNAc-Nuvec	Atherosclerotic cardiovascular disease					Preclinical PoC H1 2027

Nuvec® proprietary nucleic-acid delivery technology facilitates

- Out-licensing opportunities
- Enhancement of pipeline assets

Thalia joins a blossoming RNA therapeutics landscape

RNA therapeutics an expanding class



amvuttra
(vutrisiran) injection
25 mg/0.5 mL

onpattro
(patisiran) lipid complex injection
10 mg/5 mL

GIVLAARI
(givosiran) injection for subcutaneous use
189 mg/mL

With significant future market opportunity

- Focus extending from rare liver disease to broader markets e.g., cardiovascular and cancer
- Majority of late-stage RNA-based clinical trials are in siRNA
- miRNA represents a new potential class of RNAi therapeutics reaching the market in ~2030s

siRNA temporarily silences specific genes that cause disease

miRNA regulates gene expression

Experienced management team with domain expertise



David Solomon
Chief Executive Officer

Over 30 years of international leadership in biotech including as former CEO of Nasdaq-listed RNA therapeutics company Silence Therapeutics, as well as CEO of Zealand Pharma, where he led the company through its IPO



Fiona McLaughlin
Head of R&D

Highly experienced oncology drug developer with 30 years of experience in research and translational drug development. Former CSO of Avacta Therapeutics, with senior research roles at Algeta, Antisoma, BTG



Mark Edbrooke
Head of Strategy

Expert in nucleic acid therapeutics and cellular delivery systems including 25 years at GSK, where he set up their therapeutic nucleic acid unit, and 3 years in oncology with AstraZeneca working with Ionis and Moderna



Simon Bennett
Head of Business Dev

Seasoned business development executive, with significant experience supporting business development, licensing and fund-raising. Successfully advised and supported over 80 commercial deals



Margaret Courtney
Head of Pharma Dev

Expert in transition from bench to clinical studies and commercialization. Specific expertise in drug delivery systems, including lipid and silica nanoparticles for delivery of small molecules and nucleic acids



Rachel Schiller
Director of Operations

Over 15 years of experience in drug discovery research including at Adaptimmune. Rachel also bring significant experience project managing complex and cross-functional projects



Board of Directors



Dr Chris Britten

Non-Executive Chair

Over 30 years of leadership experience in pharma & biotech including senior corporate and BD roles at Neuraxpharm, Sandoz and Sanofi Pasteur. Former SVP Global BD at Grünenthal, leading M&A licensing post-merger integration and partnering



Dr David Solomon

Chief Executive Officer

Over 30 years of international leadership in biotech including as former CEO of Nasdaq-listed RNA therapeutics company Silence Therapeutics, as well as CEO of Zealand Pharma, where he led the company through its IPO



Luke Cairns

Executive Director

Over 25 years' experience in capital markets advising growth companies on IPOs, fundraisings, acquisitions and corporate restructurings. Former Head of Corporate Finance and Managing Director at Northland Capital Partners, later acquired by SP Angel



Dr Alastair Smith

Non-Executive Director

Scientist and entrepreneur with extensive experience building and scaling biotechnology businesses, including as founder and long-serving CEO of AIM-listed Avacta Group. Led the company through its AIM IPO and subsequent M&A-led expansion



Edward Wardle

Non-Executive Director

Experienced business leader and investor with expertise in strategy, governance and BD. Representative of Northern Standard Limited, Thalia Therapeutics' largest shareholder. Board role with AIM-listed TheraCryf PLC; senior executive at AIM-listed Ironveld PLC



Dr Michael G Palfreyman

Non-Executive Director

Over 40 years of drug discovery and development experience across major pharmaceutical and biotechnology companies. Co-founder and senior advisor at AlNivo Therapeutics, with multiple successful biotech exits, including the sale of Scriptgen to Roche for US\$230 million





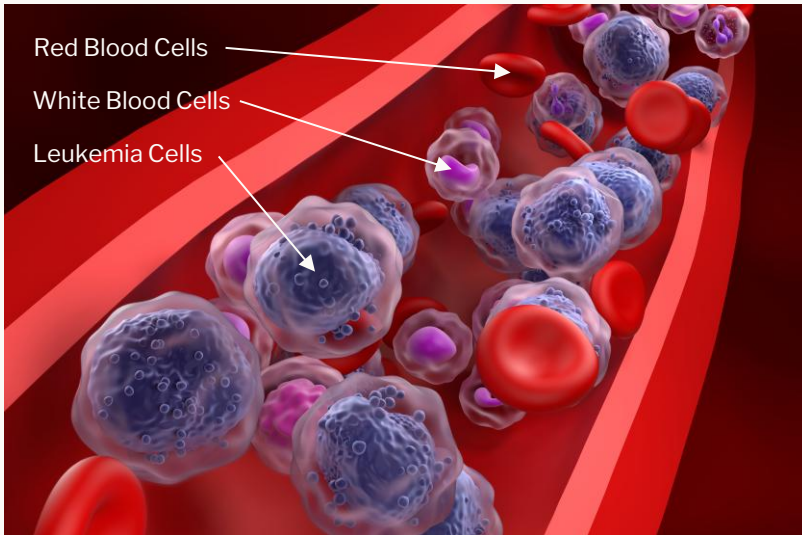
miRisten (THAT-001)

Novel approach to treat Acute Myeloid Leukemia (AML)

Targeting micro-RNA 126

Acute Myeloid Leukemia: a rare blood cancer with significant unmet need

Acute myeloid leukemia (AML) is a rare and aggressive cancer of the white blood cells characterized by a build up of leukemic blasts in the bone marrow and blood



~83,000 people with AML in the US¹

~22,000 new cases annually

~11,500 deaths annually

32.9% 5-year survival

High relapse rates to chemotherapy;
precision therapies transforming treatment potential

✓ Gene targeted and better tolerated

✓ Driving market size from \$3.9B to \$9.8B in 2035²

Yet no treatment targets leukemic stem cells, the root of AML

Patient can rapidly develop resistance to
AML treatments venetoclax and azacitidine

¹ SEER, 2023 <https://seer.cancer.gov/statfacts/html/amyl.html>

² Source: Fortune Business Insights, March 2026

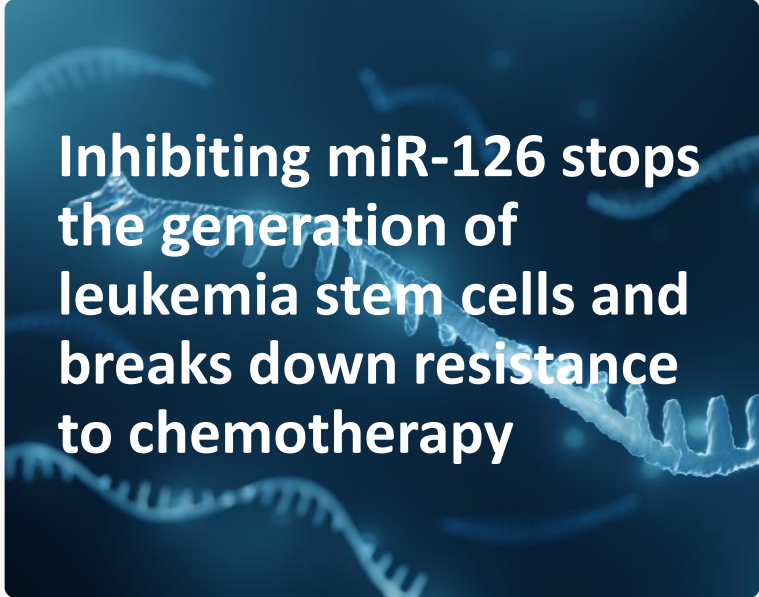
miR-126: a validated target for AML

microRNAs (miRs)

- Short non-coding single-stranded RNA molecules, that regulate mRNA stability and function
- Can regulate multiple genes
- Nobel Prize in 2024 in medicine awarded to Victor Ambros and Gary Ruvkun who discovered miRs

miR-126 in AML

- Over-expressed in AML and associated with poor prognosis
- Promotes leukemia stem cell survival and renewal
- Promotes chemotherapy resistance



Inhibiting miR-126 stops the generation of leukemia stem cells and breaks down resistance to chemotherapy

miRisten mounts triple attack on AML

Depletes miR-126 from leukemia stem cells:

1. Eliminates the source of leukemic blasts by stopping leukemia stem cell regeneration
2. Shifts leukemia stem cells out of quiescence (hibernation), making them vulnerable to anti-cancer therapy
3. Sensitizes blasts and leukemia stem cells to chemotherapy or tyrosine kinase inhibitors

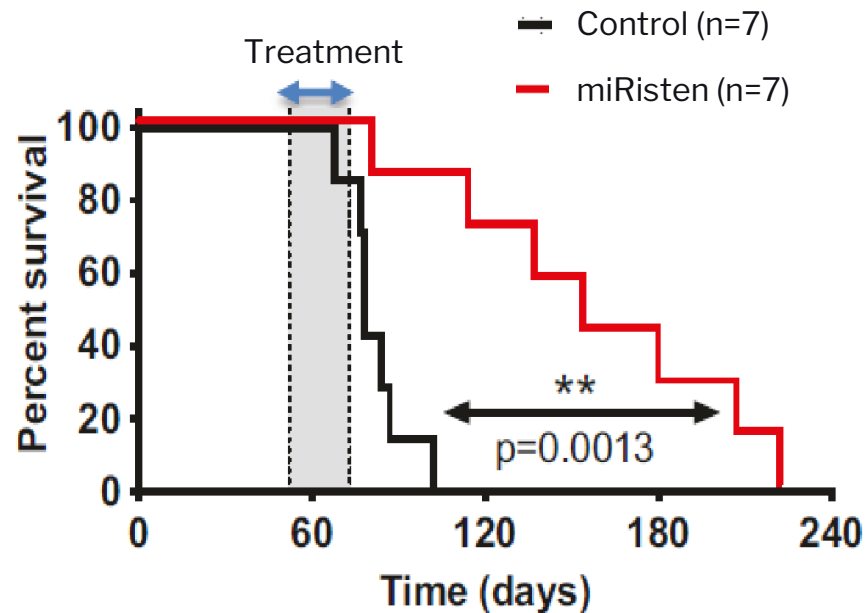
This innovative validated^{1,2} mechanism is expected to limit development of treatment resistance and relapses in AML patients

¹Zhang L. et al. Targeting mir-126 in inv(16) acute myeloid leukemia inhibits leukemia development and leukemia stem cell maintenance. *Nature Communications* 12, 6154 (2021). <https://doi.org/10.1038/s41467-021-26420-7>

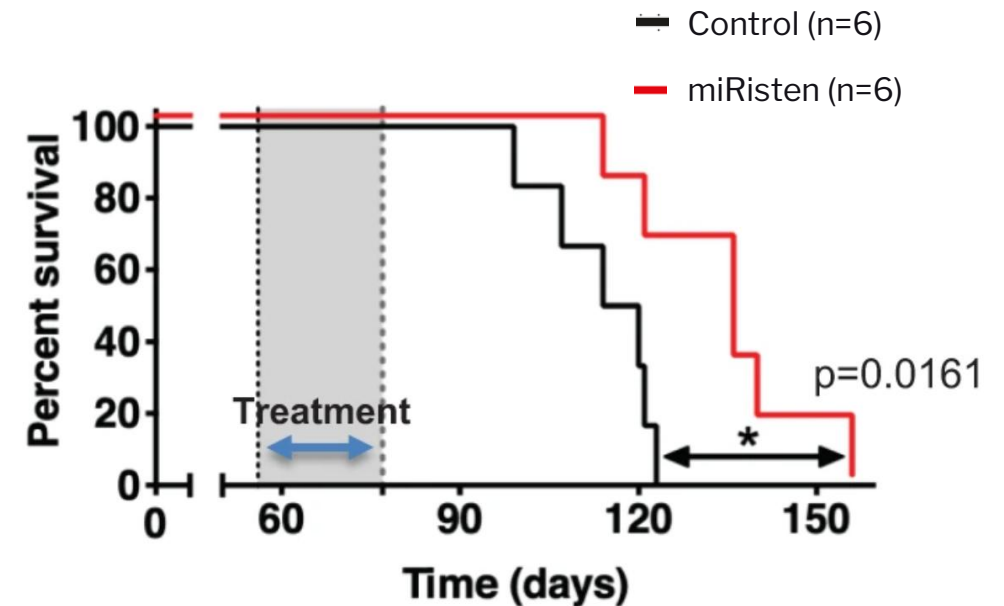
²Zhang L, Kang H, Valerio M, et al. Pharmacological inhibition of miR-126 enhances venetoclax activity in acute myeloid leukemia. *Blood*. 2026;147(10):1083–1097. <https://doi.org/10.1182/blood.2025029875>

Once daily dosing significantly prolongs survival in AML mouse models

Median survival significantly increased from 89 to 154 days in treated mice vs untreated control



Median survival significantly increased from 117 to 136 days in treated mice bearing human tumor vs untreated control



- No toxicity was seen in rodent and primate studies
- Sustained depletion of LSC reduces capacity to initiate disease

Source: Zhang, et al., Nature Communications(2021) <https://doi.org/10.1038/s41467-021-26420-7>. Assess impact of daily treatment of miRisten (20mg/kg) vs control for 3 weeks in mouse models

Phase 1 study in AML patients ongoing with data due H1 2027

Single arm open label dose-escalation study in up to 20 patients with relapsed or refractory AML

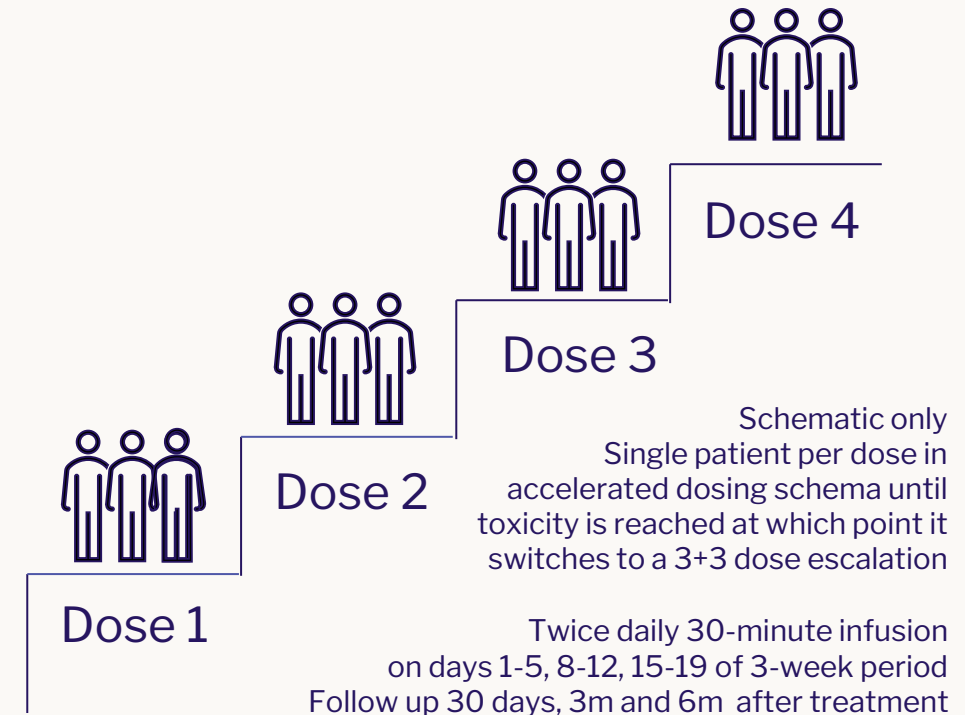
Primary objective

- Evaluate safety and tolerability of miRisten
- Determine optimal dose and recommended Phase 2 dose

Secondary objectives

- Evaluate anti-leukemic activity: complete remission, overall response and minimal residual disease negative rate and duration over the study period
- Estimate overall survival, progression free survival and duration of response at 6 months
- Establish pharmacokinetic profile

Data readout: H1 2027





THAT-002 and THAT-003

Bispecific gene silencing
for reducing cardiovascular risk

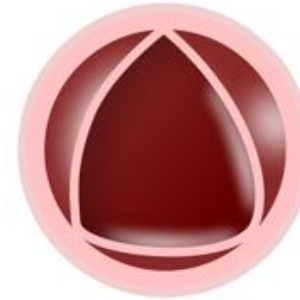
Targeting Lp(a) has the potential to significantly impact atherosclerotic cardiovascular disease

- **Silent genetic risk factor:** Lp(a): a genetically determined “sticky” LDL-like particle that significantly increases the risk of cardiovascular disease
- **Prevalent:** Up to 10% of the population worldwide have Lp(a) above 80mg/dL, which doubles their risk of myocardial infarction
- **Untreated:** Lp(a) levels are not significantly modifiable through diet or approved pharmacologic therapies

Advances in nucleic acid-based gene silencing treatments offer new potential to target Lp(a)

Cardiac function with low Lp(a)

Normal aortic valve

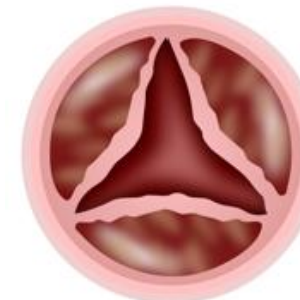


Arterial flow unrestricted

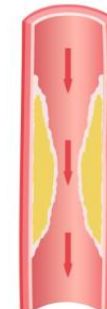


Cardiac function with high Lp(a)

Severe aortic stenosis



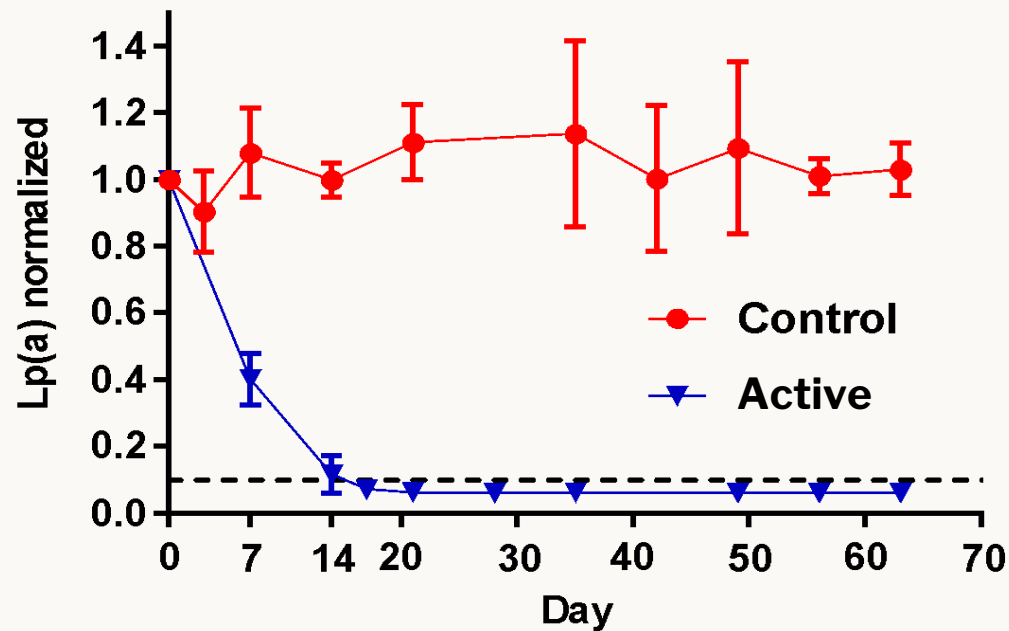
Atherosclerosis restricts flow



siRNA against Lp(a) validated by growing body of evidence

siRNA approach validated

Lp(a) siRNA reduces Lp(a) over 2+ months*



*Source: Amgen, AHA 2016

AMG 890 chart reproduced from Melquist et al "Targeting apolipoprotein(a) with a novel RNAi delivery platform as a prophylactic treatment to reduce risk of cardiovascular events in individuals with elevated lipoprotein(a)" AHA 2016 Scientific Sessions

Lp(a) target validation

- Surgical removal of Lp(a) reduces major adverse cardiovascular events 61-81%¹
- PCSK9 inhibition reduces Lp(a) and is shown to reduce cardiovascular risk ^{1,2}
- Novartis is evaluating the impact of lowering Lp(a) on cardiovascular events. Phase 3 data due early 2028³

1. Nugent et al. *Lipoprotein Apheresis: First FDA Indicated Treatment for Elevated Lipoprotein(a)*, Journal of Clinical Cardiology Vol. 1, Issue 1, DOI: <https://doi.org/10.33696/cardiology.1.002>
2. Bittner VA et al. ODYSSEY OUTCOMES Committees and Investigators. Effect of Alirocumab on Lipoprotein(a) and Cardiovascular Risk After Acute Coronary Syndrome. J Am Coll Cardiol. 2020 Jan 21;75(2):133-144. doi: 10.1016/j.jacc.2019.10.057. PMID: 31948641.
3. Novartis/Ionis' pelacarsen is an antisense oligonucleotide targeting hepatic LPA mRNA

Bispecific-Lp(a)-PCSK9: potential complementary strategy to reduce CV risk

Inhibiting PCSK9 improves the body's ability
to clear LDL (bad) cholesterol and Lp(a)

Two PCSK9 antibodies are approved as biweekly treatments:
generating sales of \$3.8B in total in 2025 (Repatha and Praluent)

PCSK9 siRNA Novartis' Leqvio (inclisiran) approved more recently, a
twice annual subcutaneous injection:

- Leqvio peak sales expectations \$2-4B
- Novartis is exploring combination treatment with Lp(a) lowering treatment (pelacarsen, once monthly subcutaneous injection)

Advantages of Thalia's
bispecific siRNA-Lp(a)-PCSK9



Addresses two independent
drivers of cardiovascular risk –
one genetic and one behavioral



Potential for additive or
synergistic Lp(a) lowering



Long-acting monotherapy:
6-month to annual administration
(superior compliance)



Fast time to market with
validated targets and regulatory
pathway

Long-acting bispecific siRNA to reduce cardiovascular risk

Simultaneously silences
Lp(a) and PCSK9 genes, two
clinically validated targets

Evaluation of different ratios
of siRNAs enabled by two
targeted delivery systems in
THAT-002 and THAT-003

Both medicines use GalNAc
to target liver cells where
the target genes are
expressed

THAT-002

LP(a)-PCSK9 – GalNAc

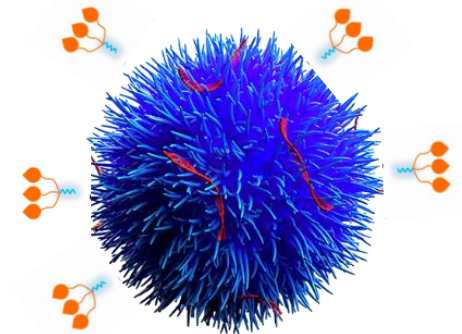
- Direct delivery of siRNA by GalNAc to liver hepatocytes
- Clinically validated with nine approved siRNA and/or antisense oligonucleotide products using GalNAc



THAT-003

LP(a)-PCSK9 – GalNAc- Nuvec®

- Versatile loading of siRNA in Nuvec® for optimal dosing
- Targeted delivery of Nuvec® to liver hepatocytes via GalNAc



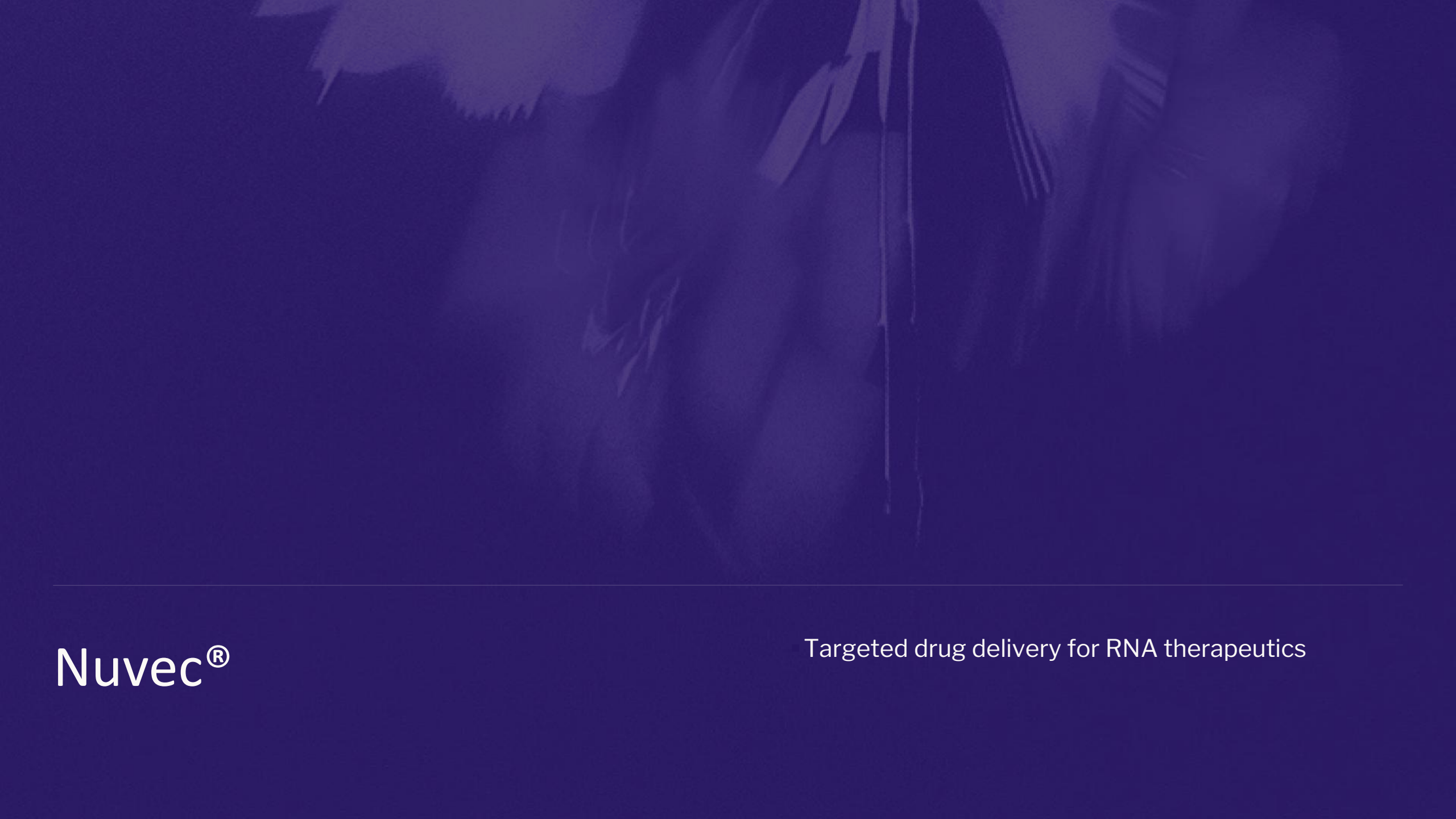
Development plan for THAT-002 and THAT-003

Current status

- IP filed in 2026
- Bispecific siRNA offers potential for fast regulatory path approval on a single pivotal trial following primate data
- Study also is expected to provide proof of concept for Nuvec® proprietary targeted delivery system for RNA therapeutics

Next steps

- Entering rodent and NHP toxicology studies simultaneously in mid 2026
- Preclinical proof of concept expected in H1 2027
- Working to submit IND in H2 2027

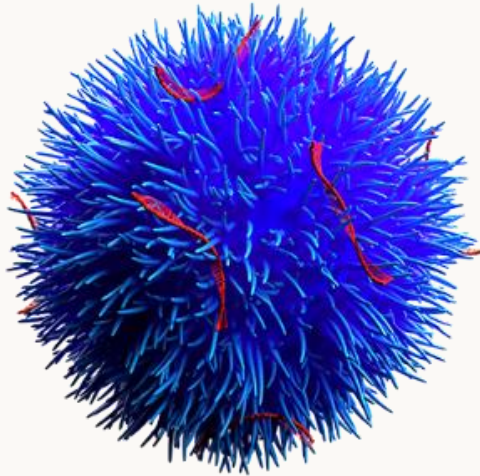


Nuvec®

Targeted drug delivery for RNA therapeutics

Nuvec[®]: Engineered to solve the challenges of RNA delivery

A silica-based nanoparticle that can hold therapeutic RNA within its spiky surface structure.



Licensing potential
with many advantages
over existing delivery systems



Structure supports **versatile loading of multiple payloads**



Modifiable nanoparticles enable **targeted delivery** to specific cell types or tissues



Nanoparticles significantly **enhance the stability** of RNA therapeutics

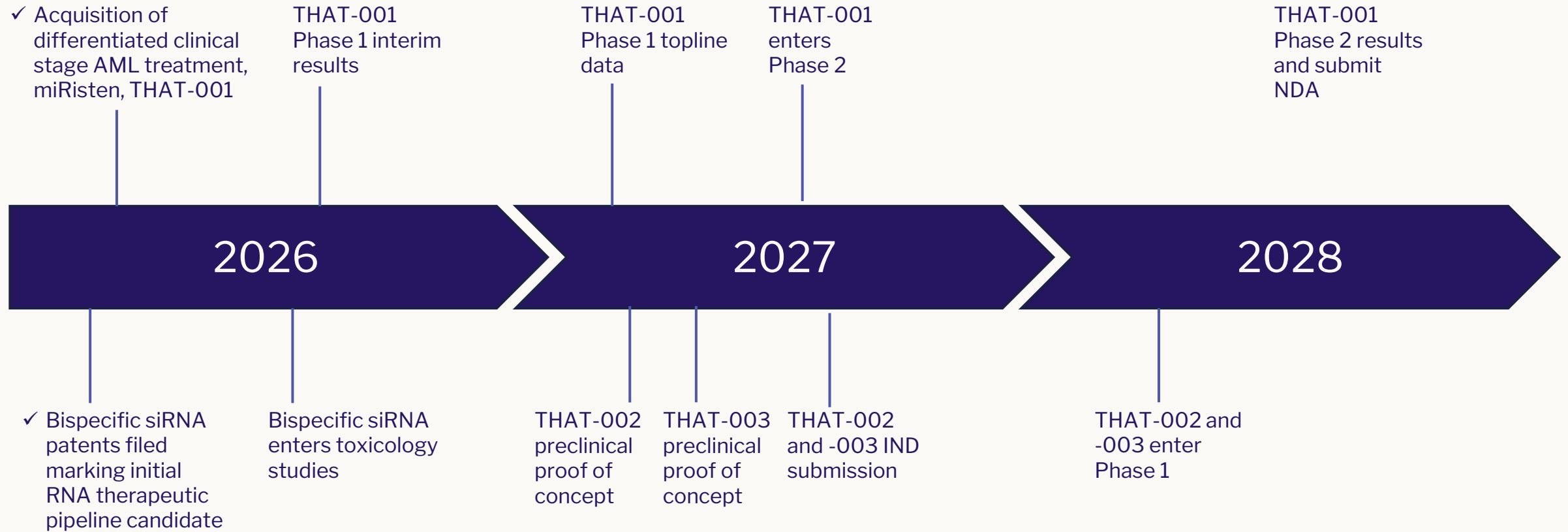


Non viral and non lipid based: avoids immune complications and enables repeat dosing



Next steps

Value-creating milestones



Investment case: Differentiated RNA therapeutics addressing significant unmet needs in cancer and cardiovascular disease

Growing pipeline of differentiated, validated RNA therapeutics

- **THAT-001:** First-in-class miRNA therapeutic targeting root cause of AML in Phase 1
- **THAT-002 & THAT-003:** Long-acting bispecific siRNA to reduce cardiovascular risk
- **Nuvec®:** Versatile RNA delivery system with many advantages over standard approaches

Upcoming milestones provide path to value creation

- Acquisition and milestone structure minimizes dilution to existing shareholders
- **Funded** through next key value inflection milestone, miRisten Phase 1 data in AML
- **Second** RNA therapeutic for cardiovascular disease working to **submit IND in 2027**

Underpinned by solid foundations

- **Experienced management** team, expertise in RNA therapeutics
- Supported by **long-term investors and vendor investment**
- Strict **capital discipline** in executing strategic transformation

THALIA

Thank you

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▪ Dr David Solomon