Thalia Therapeutics plc

Thalia has conditionally raised £2.75m (gross) new funding through an oversubscribed equity placing and subscription (the 'Placing' or the 'Fundraise') priced at 0.60p/share (representing a 3.5% premium to Tuesday's mid-market close). In conjunction and through an equity-based and deferred compensation transaction (the 'Transaction'), the Group has also acquired 100% of Sanmirna Therapeutics Inc. ('Sanmirna'), a clinical-stage biotechnology company focused on microRNA-targeted therapies for oncological indications, for an initial consideration ('the Initial Consideration') of £3.75m (less debt of £75,000). Satisfied through the issuance of new, locked-in ordinary shares also priced at 0.60p/share along with issuance of £0.76m value in Convertible Loan Notes ('the CLNs'), Sanmirna brings a new therapeutic modality to Thalia, distinct from the dual-target small interfering RNA ('siRNA') it already has in preclinical development. Directors and Vendors subscribed for £1.29m of the Fundraise. A total of 375.83m Fundraise shares are being issued utilising the Group's existing authorities (for which trading should commence 30 June 2026), while 567.61m shares (including those for the Initial Consideration) remain subject to shareholder approval at a General Meeting to be held on 17 July 2026. Together with estimated present cash-in-hand of c.£0.25m, the net funds are expected to provide an operational runway to at least mid-2027.

Use of funds – Phase 1 data readout expected H1 2027

In a related party share-based subscription, Thalia's CEO, Dr David H. Solomon and Sanmirna founders, NLC Health, have further aligned with Thalia stakeholders by investing an aggregate £275,000 cash in the Placing. The family trust associated with Ed Wardle is also subscribing £875,000 along with £130,000 investment from other Executive/Non-Executive Directors, including the Chairman. Altogether, Directors subscribed for £1.12m (representing c.40%) of the total Fundraise.

Use of Funds

Proceeds Allocated	Amount
Funding miRisten to Phase 1 completion, with release of top line data expected in H1 2027	£1.00m
Funding Thalia's bispecific cardiovascular assets to IND status with FDA	£0.75m
Transaction costs, fund raise expenses, working capital and further ad hoc R&D budgeted through H1 2027	£1.00m
Total	**£2.75m**

Source: Thalia, RNS of 24 June 2026

Full year results for period ended 31 December 2025

Thalia released results for its full year to end-December 2025 after the London market close on Tuesday, 23 June 2026. These produced little in the way of surprises, particularly given that N4 Pharma plc's progress had already been outlined in a business/operational update issued on 17 December 2025, prior to its recent Board changes, shift in strategic direction and being renamed 'Thalia Therapeutics plc' in February 2026. The financial performance and operational highlights for the 12 months are summarised on page 14.

Stock Data

Share Price	0.63p
Market Capitalisation	£7.61m*
Shares in issue:	1,208.10m*
52 week high/low	1.00p/0.35p

**Post First Admission numbers*

Company Profile

Sector:	Pharmaceuticals
Ticker:	THAT
Exchange:	AIM

Activities

Thalia Therapeutics plc ('Thalia', 'THAT' or 'the Group') is a biotechnology company developing innovative RNA-based therapeutics and delivery technologies in oncology and cardiovascular disease. Its focus is on unlocking the full therapeutic potential of RNA by overcoming the fundamental delivery challenges, enabling targeted, scalable and clinically meaningful medicines for patients with significant unmet medical needs.

www.thaliatx.com/

1-year share price performance

5-year share price performance

Source: LSE

Past performance and forecasts are not a reliable indicator of future results.

Turner Pope contact details

Tel: 0203 657 0050
Email: info@turnerpope.com
Web: www.turnerpope.com

Andrew Thacker/Guy McDougall
Corporate Broking & Sales

Barry Gibb
Research Analyst

Turner Pope acts as joint broker to Thalia Therapeutics plc.

Attention is drawn to the disclaimers and risk warnings at the end of this document.

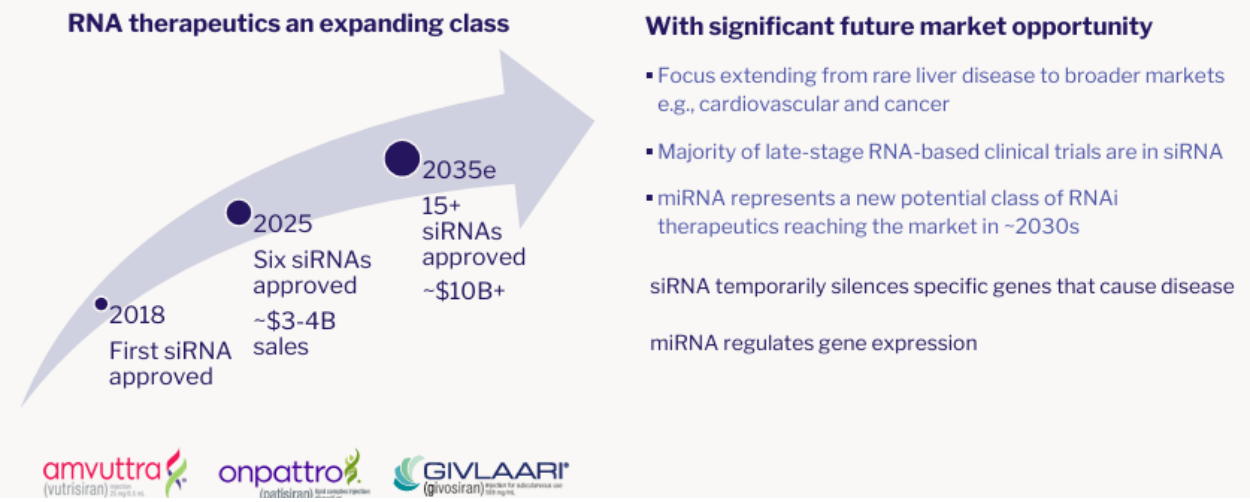
Aiming to build-out a portfolio of innovative RNA therapeutics

Further to today's Placing and in line with its CEO's stated ambition to build-out a diversified portfolio of innovative RNA therapeutics for treatment of cancer and cardiovascular diseases, Thalia has today also announced its conditional acquisition of 100% of Sanmirna. Sanmirna is developing a first-in-class microRNA-126 ('miR-126') inhibitor called miRisten, which is a differentiated Phase 1 asset targeting the root cause of Acute Myeloid Leukaemia ('AML'), a condition for which conventional therapies generally fail. The acquisition is based on a £3.75m share and CLN-based transaction, plus up to £13m in deferred consideration. The net proceeds of Thalia's completed Placing will be directed to fund miRisten (which is to be known by development code: 'THAT-001') through to completion of its Phase 1 clinical trial in H1 2027; they will also progress the Group's 'home grown' bispecific cardiovascular assets ('THAT-002' and 'THAT-003') to Investigational New Drug ('IND') status with the FDA, while also (along with existing cash) helping to satisfy anticipated corporate costs/ad hoc R&D/working capital needs. Recognising that well-resourced sector majors are presently keenly seeking to increase their involvement across the rapidly emerging category of RNA therapeutics, the Board expects to reward shareholders in due course through successful product development, leading to partnerships/joint venture agreements that may result in collection of remunerative upfront payments and/or milestone-based fees/licensing agreements.

RNA medicine now entering its second phase

The COVID-19 pandemic transformed RNA technologies into one of the most visible scientific breakthroughs of the decade. It accelerated public awareness and validated the platform on a global scale. mRNA vaccines have demonstrated unprecedented speed of development, large-scale manufacturing capability and global deployment, pushing genetic messenger therapeutics into the public spotlight.

RNA Therapeutics Landscape



Source: Thalia, Investor Presentation June 2026

Rather than representing a single technology, RNA therapeutics are built around the broader biological principle that RNA sits at the centre of gene expression and protein production. That diversity is increasingly allowing developers to approach RNA as a flexible, portable engineering platform capable of encoding multiple biological functions. It can be encoded with antigens, cytokines, antibodies or other therapeutic proteins and potentially combine multiple mechanisms simultaneously. Moreover, unlike traditional biologics or viral vector systems, RNA technologies can potentially be adapted more rapidly across different disease areas and targets.

Thalia sees major opportunities emerging in oncology, cardiovascular disease and gene modulation, while recognising that delivery remains one of the technology's defining areas for innovation. The Group's proprietary silicon nanoparticle delivery platform, Nuvec®, has been designed to allow developers to load multiple RNA therapeutics into a single nanoparticle and potentially adjust the ratio between payloads. It intends to explore this in cardiovascular disease using dual-acting siRNA combinations against both Lipoprotein(a) Lp(a) and PCSK9.

Sanmirna – Terms of acquisition and deferred milestone considerations

The 100% acquisition of Sanmirna is based on an initial valuation of £3.75m (less £75,000 debt) coming in the form of, (i) Initial Consideration Thalia Therapeutics plc shares priced at 0.6p/share that (along with any Fundraising Shares held by the Vendors) are locked-in for 12 months from Admission followed by an orderly market agreement for a further 12 months; (ii) 2-year term unsecured, zero interest CLNs to the value of £0.764m that are convertible at the Fundraise price and locked-in on the same terms as the Initial Consideration shares upon either party's request subject to certain qualifying conditions the primary one being that, following conversion, the Vendors shall not, in aggregate, hold more than 29.9% of the issued share capital of the Group; and (iii) Up to £13m in deferred consideration ('the Deferred Consideration'). It is understood that immediately prior to completion of the Transaction, Sanmirna's direct equity holders were: NLC Health (and affiliates) ('NLC'): 49.45%, City of Hope National Medical Center ('CoH'): 31.50%, and Dr David Solomon (Thalia's CEO): 19.05%. Upon successful completion of the total corporate action, the vendors of Sanmirna are together expected to hold c.29.9% of Thalia's ordinary shares.

Sanmirna Therapeutics Inc. – Terms of Acquisition*

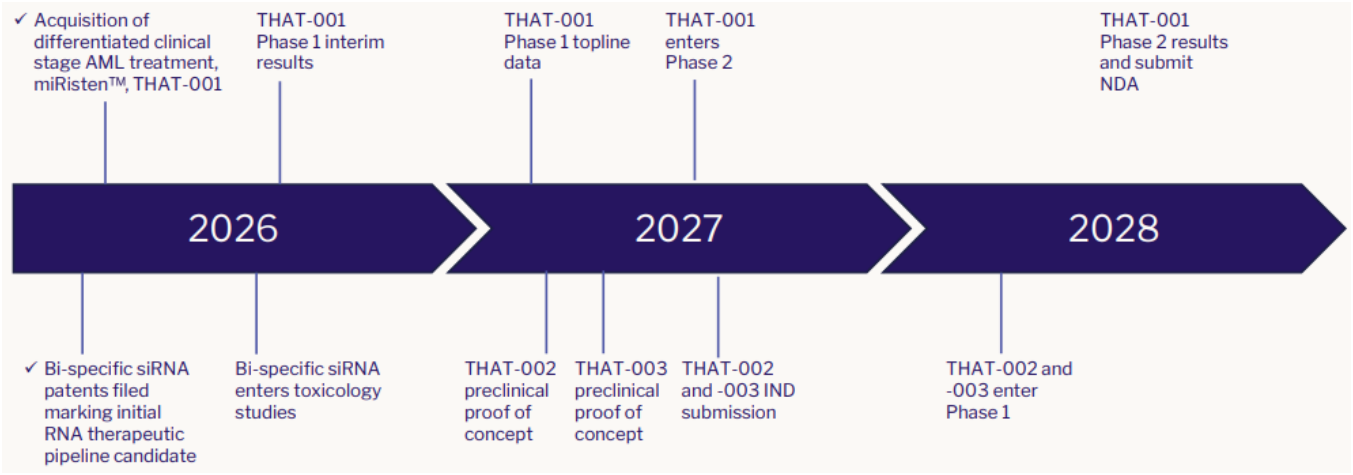
• **£3.75M (less debt of £75,000) in form of an Initial Consideration 485.1m shares plus £0.764m in CLNs****
• **With up to £13M in deferred consideration based on:**
• c.£4.1m payable in shares on successful completion of Phase 1a/b clinical trial (trial number NCT07025564), as evidenced by submission to the FDA of a Clinical Study Report (CSR), priced at 20-day volume weighted average price ('VWAP') preceding the last patient's final dose ('Milestone 1')
• c.£3.9m in cash on dosing of 3rd patient in Phase 3 or label-enabling study ('Milestone 2')***
• £2M in cash on filing for product approval in the US
• £1M in cash for each of:
· US product approval
· EU product approval
· Annual net sales exceeding US$250m

**Conditional on, amongst others, passing shareholder resolutions at AGM and CLNs only being convertible to the extent that the Vendor's shareholding would not exceed 29.9% **Less debt of £75,000, otherwise wholly satisfied in equity and CLNs ***Payment shall be reduced by the same amount as any Milestone 1 Payment adjustment is increased. GBP=1.32US$ Source: Thalia, RNS of 24 June 2026*

Additional development milestone payments of varying amounts may be received by CoH in the future, dependent on Thalia achieving specific patient dosing points as it progresses through Phase 2 and Phase 3 clinical trials, followed by low single-digit royalties on subsequent sales which will also change in the event of the Group paying a third party to make/use/sell the licensed product. Payments will also become due upon formal receipt of FDA and European Medicines Agency ('EMA') marketing approvals.

CoH will also be reimbursed the US$1.2m that it committed for miRisten's Phase 1 clinical trial up to September 2026 within 5 years, with earlier payment contingent on the sale or licensing or on dosing a patient in a Phase 3 trial.

Thalia Therapeutics – Prospective Value-creating Operational Milestones



Source: Thalia, Investor Presentation June 2026

Originally developed by City of Hope, miRisten is to become Thalia's lead asset

miRisten was originally developed by CoH, a world-renowned, independent biomedical research, treatment, and educational centre based in California. Its development was initially focused on targeted combination therapy alongside tyrosine kinase inhibitors ('TKIs') to treat rare and aggressive blood cancers like chronic myeloid leukaemia ('CML') and AML, both of which are blood cancers of the bone marrow affecting myeloid white blood cells. CoH's researchers began moving the development drug through expanded preclinical testing in 2015, following which it received grant funding (2016) and released its first major publication (March 2018) before transitioning to clinical trials (study identifier: NCT07025564) in October 2025.

Its first-in-human Phase 1 clinical trial (a CpG-conjugated anti-miR-126 oligonucleotide for relapsed/refractory AML) began officially enrolling patients in February 2026. Upon developing a candidate(s) to this point, CoH routinely chooses to leverage its commercialisation engine with a view to scaling the opportunity. Being a research hospital, as opposed to a customer-facing pharmaceutical business, it does not independently advance its candidates through the various early and late clinical phases, but instead seeks to attract drug developers/ experienced teams with the technical capability and ability to secure the funding necessary to progress them through to regulatory approval. Typically, this involves out-licensing to pharma/biotech companies or spinning off into dedicated startups.

Early in 2025, NLC Health Ventures ('NLC') recognised miRisten's exceptional clinical promise, along with opportunities to target colorectal cancer and Non-Small Cell Lung Cancer ('NSCLC') through the same biological research. Headquartered in Amsterdam, NLC is a prominent European early-stage health tech venture capital firm with unique approach spread across four specialised investment funds. Founded in 2015, it focuses on identifying promising, uncommercialised scientific discoveries from universities, academic hospitals and corporations, transforming them into viable medical startups. The firm manages a highly diversified portfolio of over 100 active healthcare opportunities spanning biotechnology/therapeutics, medtech, digital health/AI, and 'green health', operating on the basis that a broad approach lowers investor risk across the historically volatile healthcare incubation cycle. Based on an early-stage tech transfer agreement, CoH passed control of the future development of miRisten (and its related opportunities) to NLC, permitting them to be placed inside a standalone biotech venture. NLC then sought a visionary CEO capable of designing a roadmap to leverage miRisten; in early in 2026, it founded Sanmirna Therapeutics Inc. and appointed Dr David Solomon as its CEO.

NLC typically starts with c. 70% of the equity in its newly built ventures. The remaining shares may be allocated to the original intellectual property owner (in this case, CoH) and its recruited CEO/core management team. This ownership percentage gradually dilutes over time as the target company advances through financing rounds. It is understood that immediately prior to Thalia's proposed acquisition, Sanmirna's direct equity holders were: NLC (and affiliates) 50%, CoH 30%, and Dr. David Solomon (Thalia's CEO) 20%. See the Appendix for full details regarding: miRisten's licensed patents by country.

Sanmirna – A value-accretive, transformational acquisition

The 100% acquisition of Sanmirna introduces a clinical stage asset to Thalia's new RNA therapeutic modality pipeline. It also introduces a third pillar of expertise to the Group. These together will comprise miRNA (i.e., short, non-coding single-stranded RNA molecules that helps regulate gene expression, stability and function), siRNA (i.e., a short, double-stranded small interfering RNA capable of silencing specific genes that cause disease) and, Nuvec® (the Group's proprietary delivery vehicle for such nucleic acid therapies to target specific cells in the body). They have been assembled with a view to progressing the Board's strategy of establishing itself as a new force in the area of nucleic acids.

Harnessing the miRNA pathway opens a novel frontier in oligonucleotide therapeutics. The approach enables precise regulation of gene expression, an area in which the pharmaceutical and biotech industry widely expects to achieve first commercialisation during the early 2030s. miRisten is considered a suitable treatment for AML, given that it specifically targets miR-126, an overexpressed microRNA responsible for protecting leukaemia stem cells

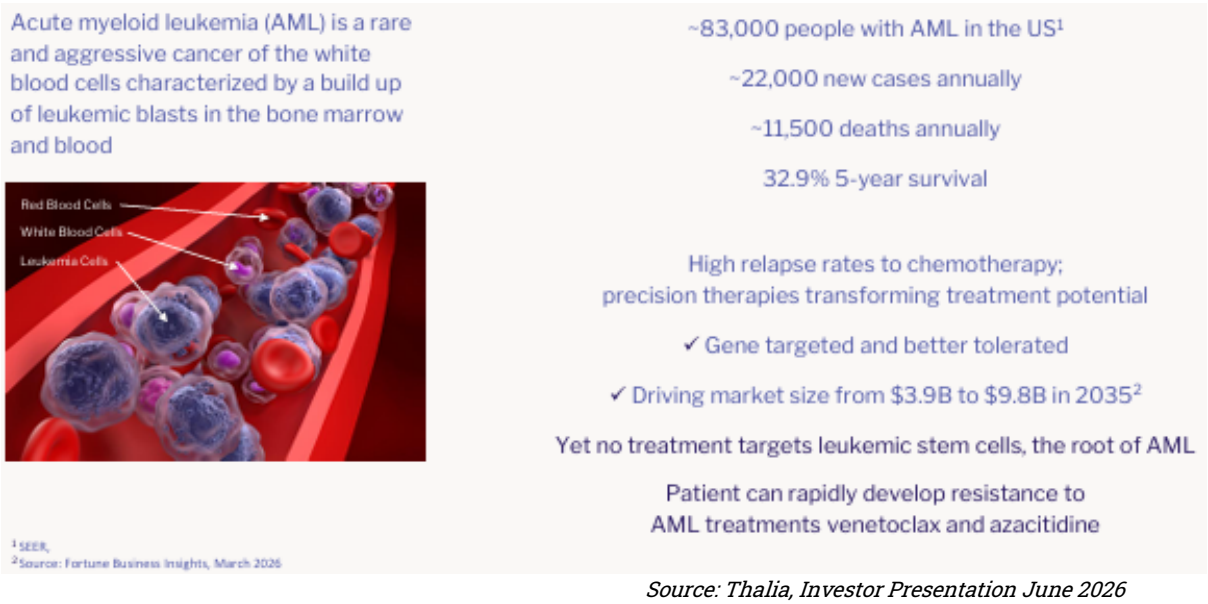
('LSCs') and maintaining their dormancy. By inhibiting miR-126, miRisten disrupts LSC homeostasis and drives them out of dormancy. In turn, this makes them vulnerable to traditional cancer therapies (such as combined venetoclax ('VEN') and azacitidine ('AZA') treatment), to which patients otherwise rapidly develop resistance. miRisten secured FDA regulatory clearance for its Phase 1 clinical trial, designed to evaluate the safety, side effects, and optimal dosing of the drug for patients with relapsed or refractory AML, and began recruiting participants on 24 October 2025. At that time it had an estimated study completion date of April 2027.

Sanmirna Acquisition Includes Clinical Stage Asset for Treatment of Acute Myeloid Leukaemia

Source: Thalia, Investor Presentation, June 2026

AML has a significant unmet medical need, affecting approximately 22,000 new US patients annually who face a 5-year survival rate of just 33%. With patients suffering high relapse rates to chemotherapy, there are c.11,500 deaths annually. There is no current treatment specifically targeting leukemic stem cells, which are the condition's root cause. Precision therapies (such as midostaurin and quizartinib) alternatively work by identifying and blocking specific genetic mutations (such as FLT3, IDH1, or IDH2) that force abnormal blood cells to continuously multiply. Instead of blindly killing all fast-growing cells like traditional chemotherapy, they cut off the cancer's survival signals or force the immature leukaemia cells to finally mature into normal, healthy blood cells.

Acute Myeloid Leukaemia: A Rare Blood Cancer with Significant Unmet Need



Source: Thalia, Investor Presentation June 2026

There are a number of drug development companies alongside Thalia working on precision and better tolerated therapies to address a market that has been projected by Fortune Business Insights to grow from US$3.9bn in 2025 to US$9.8bn by 2035. With the biology of miR-126 extending beyond just leukaemia, Sanmirna has further preclinical assets, including SAN-002 (developed to treat Colorectal Cancer by restoring downregulated miR-126 activity to suppress tumour growth and angiogenesis), and SAN-003 (that targets Non-Small Cell Lung Cancer to reduce cancer cell proliferation and re-sensitise tumours to existing treatment), although these are not presently included in Thalia's initial priority pipeline.

miRisten seeking to eradicate leukaemia stem cells and overcome chemotherapy resistance

Upon completion of Thalia's acquisition of 100% of Sanmirna, miRisten™ will be renamed 'THAT-001', becoming the Group's lead therapeutic asset alongside two first-in-class bispecific siRNA candidates, 'THAT-002' and 'THAT-003'. The latter are presently in Discovery phase with indications for reduction of atherosclerotic cardiovascular risk.

Thalia's Post Transaction Therapeutic Pipeline

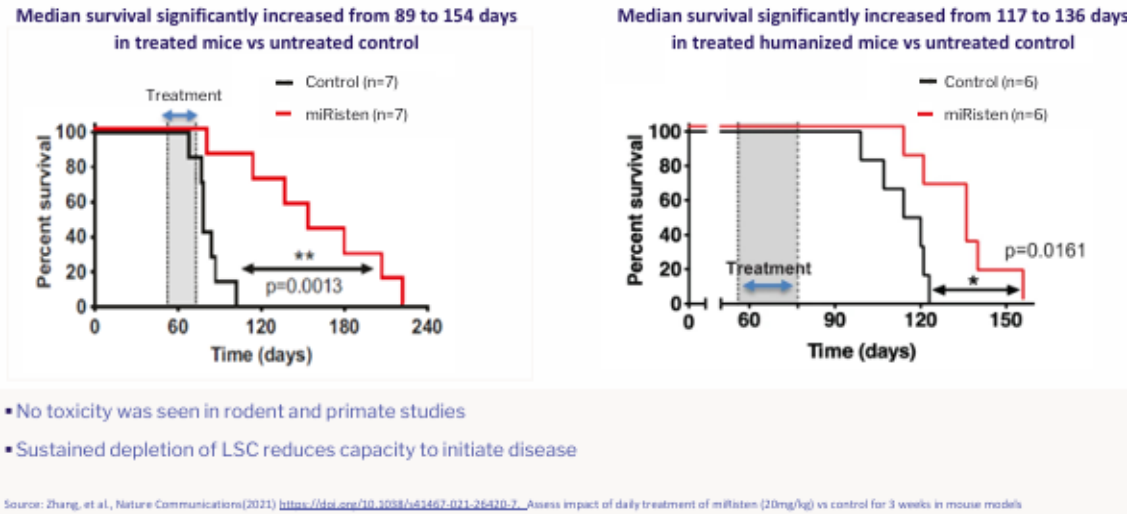
Pipeline candidate	Target	Indication	Discovery	Preclinical/ IND enabling	Phase I	Phase II-III	Milestones
THAT-001	Anti-miR-126	Acute myeloid leukemia (AML)					Phase 1 data H1 2027
THAT-002	LP(a)-PCSK9 – GalNAc	Atherosclerotic cardiovascular disease					Preclinical PoC H1 2027
THAT-003	LP(a)-PCSK9 –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

Nuvec® proprietary nucleic-acid delivery technology facilitates
- Out-licensing opportunities
- Enhancement of pipeline assets

Source: Thalia, Investor Presentation, June 2026

miRisten's therapeutic strategy is aimed primarily at eradicating leukaemia Stem Cells ('LSCs') and overcoming chemotherapy resistance. Its first-in-human Phase 1 trial comprises a single-arm, open-label, dose-escalation study in up to 20 patients. Its primary objectives are to evaluate the drug's safety and tolerability via a twice-daily, 30-minute intravenous infusion in patients with relapsed or refractory AML; these data are expected to determine the optimal recommended Phase 2 dose. Secondary objectives are to assess anti-leukemic activity by evaluating rates of complete remission, complete remission with incomplete hematologic recovery, partial remission, overall response rate, minimal residual disease negativity, and duration of response over a 6-month period.

Once Daily Dosing Significantly Prolongs Survival in AML Mouse Models



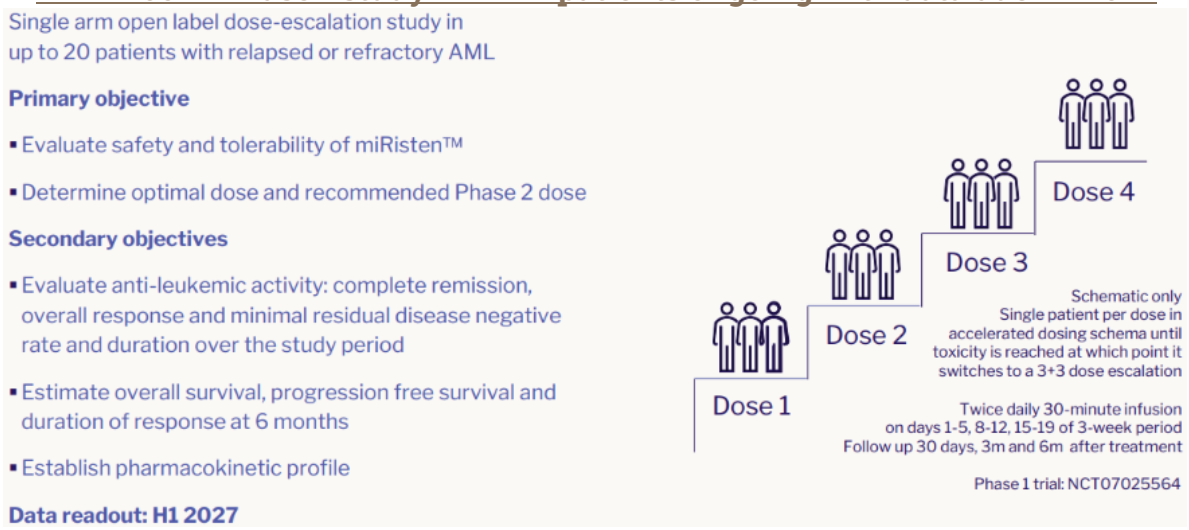
Source: Thalia, Investor Presentation June 2026

miR-126 plays a vital role in maintaining the function, metabolism, and survival of persistent LSCs. It is heavily

overexpressed in specific subtypes of AML, such as those with inv(16) chromosomal mutations. By knocking down this target, miRisten disrupts mitochondrial metabolism (specifically fatty acid oxidation and oxidative phosphorylation), forcing the cells into apoptosis while reversing resistance to chemotherapy. In inv(16) AML and human patient-derived xenograft ('PDX') mouse models, systemic administration of miRisten (20 mg/kg/dose) significantly reduced leukemic burden in the bone marrow and lowered the frequency of malignant CD34+ cells. *Ex vivo* and *in vivo* studies confirmed that miRisten specifically induces cell cycle entry, reduces colony-forming capacity and enhances programmed cell death in human MDS and AML stem cells while sparing normal, healthy hematopoietic cells. Similarly treated animal models also showed significantly prolonged median survival, increasing from 89 to 154 days in treated mice versus untreated controls (n=7,6), as illustrated overleaf. Its innovative validated mechanism is expected to limit development of treatment resistance and relapses in AML patients. It mounts a triple attack on AML summarised as follows:

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

THAT-001 - Phase 1 study in AML patients ongoing with data due H1 2027



Source: Thalia, Investor Presentation June 2026

Potential for miRisten to transition into a combination strategy

miRisten's Phase 1 clinical trial is designed strictly as a single-agent monotherapy study. Undertaken by CoH, it will serve as a foundational step to evaluate the safety, pharmacokinetics and optimal dosing for the chosen indication. Shortly after the trial's announcement, however, CoH also made preclinical translational data (derived from parallel laboratory and animal models) available to demonstrate that a combination of miRisten with the standard, FDA-approved regimen of VEN and AZA provides a highly synergistic strategy for treatment of AML (Chen, 2026; Zhang, 2026).

VEN is a selective BCL-2 inhibitor, while AZA is a hypomethylating agent that acts as its standard therapeutic partner (Stafylidis, 2026). The addition of miRisten suppresses mitochondrial energy production (specifically fatty acid oxidation and oxidative phosphorylation), to a far greater extent than either drug can alone, making LSCs hyper-susceptible to VEN/AZA-mediated killing (Chen, 2026; Zhang, 2026). Given that miRisten is inherently designed to dismantle resistance mechanisms, as opposed to acting as a standalone debulking agent, its true value may possibly be unlocked when paired with such targeted drugs. The drug's transition to a combination strategy remains, of course, legally gated by Phase 1 safety data.

Once a safe combination dose is established in Phase 1, the venture can officially transition into Phase 2 testing. This is the stage at which different therapeutic hypotheses can be evaluated in specific patient populations. Clinical objectives might, for example, be to assess whether adding miRisten to the frontline VEN/AZA regimen for older or

unfit patients increases the depth of complete remission and/or achieves higher rates of minimal residual disease negativity. Similarly, for VEN-resistant or relapsed patients, trials could test if miRisten can reverse acquired resistance after initial therapy has failed.

Although such a development strategy for miRisten is purely conceptual at this time, it highlights an important opportunity for value creation. A successful outcome from such studies could, for example, generate significant interest from VEN's joint developers (AbbVie and Genentech), who are likely to be seeking routes to extend their market exclusivity beyond the drug's core patent expiry in May 2032.

Thalia proposing a bispecific-Lp(a)-PCSK9 as complementary strategy to reduce CV risk

Thalia is developing a bispecific (i.e. two targets, being Lp(a) and PCSK9, through one therapeutic) siRNA for atherosclerotic cardiovascular disease ('ASCVD') in order to reduce cardiovascular ('CV') risk. It aims to simultaneously block the PCSK9 protein and bind to Lipoprotein(a) ('Lp(a)') particles, through a dual-action designed to drastically lower bad cholesterol ('LDL-C') and high Lp(a) levels at the same time. Its prospective commercialisation could offer a powerful, single-injection approach to prevent cardiovascular events.

Nucleic acids, including plasmid DNA ('deoxyribonucleic acid'), mRNA ('messenger RNA'), siRNA ('small interfering RNA') and miRNA (micro-RNA) represent one of the four major classes of biological macromolecules upon which all known life is based. They demonstrate unprecedented capability to be engineered to penetrate cell membranes and modulate genetic pathways. Their therapeutic application is consequently projected to be one of the biotechnology sector's primary growth drivers over the coming decade. According to data from Towards Healthcare, the global RNA therapeutics and vaccines market size is projected to reach approximately US$205.34 billion by 2035, expanding at a CAGR of 87%. The report highlights that while there were six approved siRNA drugs globally in 2025, the pipeline is on track to yield over 15 approved treatments by 2035, each generating more than US$10 billion in dedicated product revenue.

The majority of existing late-stage RNA-based clinical trials utilise siRNA platforms. These therapies temporarily silence specific disease-causing genes, with maintenance dosing schedules ranging from every few weeks to an ultra-convenient 6 to 12 months. There are currently eight FDA-approved siRNA drugs: patisiran, givosiran, lumasiran, inclisiran, vutrisiran, nedosiran, fitusiran, and plozasiran. Collectively, these therapies accounted for approximately US$5.7 billion in global sales in 2025. Those spearheading research in the siRNA therapeutics space include Arrowhead Pharmaceuticals, which develops drugs via its TRiM platform, alongside Alnylam Pharmaceuticals, the pioneer with multiple FDA-approved siRNA medicines, and others in the area of RNA interference, such as Silence Therapeutics and Ionis Pharmaceuticals. All essentially target liver cells by utilising N-acetylgalactosamine ('GalNAc'), which is a modified carbohydrate that uniquely binds to a specific receptor in hepatocytes, permitting medicinal targeting.

Comparison of Existing Lipoprotein-lowering Therapies with Thalia's THAT-002/003

Feature	Repatha / Praluent *(evolocumab / alirocumab)*	Leqvio *(inclisiran)*	THAT-002/003 Bispecific Lp(a)-PCSK9
Drug Class	Monoclonal antibody	Small interfering RNA (siRNA)	Small interfering RNA (siRNA)
Primary Target	PCSK9 protein (circulating)	PCSK9 synthesis (intracellular)	Both PCSK9 and Lp(a) proteins
LDL-C Lowering	High (**55%–60%**)	High (**~50%–54%**)	High (Similar mechanism)
Lp(a) Lowering	Modest (**20%–30%**)	**Negligible or zero**	Profound (**70%–90%+** targeted arm)
Dosing Frequency	Every 2 weeks, or once monthly	Every **6 months** *(after starter doses)*	Every 6 months to 1 year (after starter doses)
Administration	Self-injection	Healthcare professional clinic	Self-injection or oral
Primary Limitation	High injection frequency	Ineffective for high Lp(a)	Early-stage development

Source: Commercial information, TPI

Once inside the liver cell, the therapeutic ambition is to reduce the target's gene expression and so modify or cure disease. The opportunity identified by Thalia is not only to use dual acting or bispecific siRNAs to target ASCVD, but also to examine the potential of its proprietary delivery system, Nuvec®, to leapfrog beyond the liver toward multiple other targets. Its modifiable nanoparticle structure enables targeted delivery to specific cell types or tissues along with versatile loading of different therapeutics.

There are currently two PCSK9 antibodies approved as biweekly treatments, Repatha and Praluent, which generated total sales of c.US$3 billion in 2024. Both are exceptionally potent for lowering LDL-C (by 55% to 60%), but only yield a modest 20% to 30% secondary reduction in Lp(a). Novartis' siRNA therapeutic, inclisiran (Leqvio), was also approved recently as a twice annual subcutaneous injection for ASCVD patients who need further LDL-C reduction despite taking maximally tolerates statins. Its peak sales expectation is in the range of US$2 to US$4 billion. Novartis is exploring combination treatment with Lp(a) lowering treatment pelacarsen, which uses an antisense mechanism to reduce production of apolipoprotein(a) in the liver based on once monthly subcutaneous injection. Eli Lilly spent US$1 billon this time last year buying Verve Therapeutics, following which it published Phase 1b Heart-2 study results for VERVE-102, a gene-editing medicine, on 25 May 2026 that produced dose-dependent lowering of PCSK9 and LDL-C of up to 88% and 62% respectively sustained for up to 18 months. Thalia's THAT-002 and THAT 003 are proposed as long-lasting monotherapies with 6-months to 1 year administration.

Lipid-Lowering Interventions – Approved and Investigational Agents*

Drug Name	Mechanism of Action	Dosing Frequency
HMG-CoA Reductase Inhibitors (Statins)		
Statins (class) *Lovastatin, pravastatin, simvastatin, fluvastatin, atorvastatin, rosuvastatin, pitavastatin*	Competitive inhibition of HMG-CoA reductase, the rate-limiting enzyme in hepatic cholesterol biosynthesis → reduced intracellular cholesterol → upregulation of hepatic LDL receptors → increased LDL-C clearance from plasma	Once daily
Cholesterol Absorption Inhibitor		
Ezetimibe (Zetia)	Inhibits NPC1L1 transporter at the intestinal brush border → reduced intestinal cholesterol absorption → compensatory upregulation of hepatic LDL receptors	Once daily
Bile Acid Sequestrants		
Cholestyramine (Questran)	Anion-exchange resins bind bile acids in the intestinal lumen → prevent enterohepatic recirculation → liver depletes cholesterol to synthesize new bile acids → compensatory upregulation of hepatic LDL receptors	2–4× daily
Colestipol (Colestid)		2–4× daily (granules); 2× daily (tablets)
Colesevelam (Welchol)		2× daily (tablets); once daily (powder)
ATP Citrate Lyase (ACL) Inhibitor		
Bempedoic acid (Nexletol)	Inhibits ATP citrate lyase, upstream of HMG-CoA reductase in hepatic cholesterol biosynthesis → reduced hepatic cholesterol synthesis → upregulation of LDL receptors. Requires hepatic ACSVL1 for activation, thus sparing skeletal muscle	Once daily
Microsomal Triglyceride Transfer Protein (MTP) Inhibitor		
Lomitapide (Juxtapid) *HoFH only (orphan indication)*	Directly inhibits MTP in the ER of hepatocytes and enterocytes → impairs assembly and secretion of VLDL and chylomicrons → reduced circulating ApoB-containing lipoproteins including LDL-C	Once daily

**As of June 2026* *Source: Peter Attia 'The beginning of the end of atherosclerosis?'*

Tabulation continued overleaf:

ApoB-100 Antisense Oligonucleotide		
Mipomersen (Kynamro) *HoFH only (orphan indication)*	Antisense oligonucleotide complementary to ApoB-100 mRNA → RNase H–mediated degradation of ApoB-100 mRNA in hepatocytes → reduced synthesis and secretion of VLDL and LDL	Once weekly (SC)
PCSK9 Inhibitors		
Evolocumab (Repatha)	Fully human (evolocumab) or humanized (alirocumab) IgG monoclonal antibody → binds circulating PCSK9 protein → prevents PCSK9-mediated lysosomal degradation of hepatic LDL receptors → increased LDL receptor recycling to hepatocyte surface → enhanced LDL-C clearance	140 mg every 2 weeks or 420 mg monthly (SC)
Alirocumab (Praluent)		75–150 mg every 2 weeks or 300 mg monthly (SC)
Lerodalcibep (Lerochol) *FDA approved December 2025*	Engineered protein → high-affinity subnanomolar binding to PCSK9 → prevents PCSK9 from targeting LDL receptors for degradation	300 mg once monthly (SC)
Inclisiran (Leqvio)	GalNAc-conjugated siRNA → RISC-mediated cleavage of PCSK9 mRNA → silences hepatic PCSK9 production at the transcript level → sustained reduction in circulating PCSK9 → preserved LDL receptor recycling	284 mg at day 1, month 3, then every 6 months (SC)
Enlicitide decanoate *(Investigational)* *Phase 3 complete (CORALreef program); FDA CNPV granted Dec 2025; NDA under review*	Macrocyclic peptide prodrug (decanoate ester) → cleaved to active enlicitide → directly binds PCSK9 protein → blocks PCSK9–LDL receptor interaction; same downstream mechanism as mAbs but orally bioavailable	Once daily (oral, fasting)
In Vivo Gene Editing		
VERVE-102 *(Investigational)* *Phase 1b (HEART-2)*	LNP delivers mRNA encoding an adenine base editor (ABE) + sgRNA targeting the PCSK9 gene in hepatocytes → A·T-to-G·C transition at a critical codon → permanent loss-of-function edit → lifelong reduction in hepatic PCSK9 production → sustained LDL receptor upregulation; does not require double-strand DNA breaks	Single IV infusion (intended as one-time)

Note: Enlicitide decanoate and VERVE-102 have not received FDA or EMA approval as of June 2026.

**As of June 2026* *Source: Peter Attia 'The beginning of the end of atherosclerosis?'*

Lp(a) and PCSK9 are both are highly important biomarkers for cardiovascular (CV) disease risk

Lp(a) is a lipoprotein particle composed of fat and protein that carries cholesterol through the bloodstream. A person's Lp(a) level is genetically determined by the traits inherited from their biological parents, specifically via the LPA gene. Approximately 20% of the global population has elevated Lp(a) levels (i.e., >50mg/dL) and individuals with levels >80mg/dL face a drastically increased risk of myocardial infarction. While mechanical removal via lipoprotein apheresis has historically been used to manage this risk, modern therapeutic strategies are shifting toward targeted treatments. These include PCSK9 inhibitors, which offer modest Lp(a) reductions, and novel siRNA approaches currently in clinical trials that aim to radically silence Lp(a) production. PCSK9 is a protein primarily produced in the liver to regulate blood cholesterol; it binds to low-density LDL receptors on the surface of liver cells, leading to their destruction; inhibiting this protein preserves receptors, thereby accelerating the removal of 'bad' cholesterol from the body. While PCSK9 expression is also largely genetic, it can be influenced to a limited extent by lifestyle factors like diet and exercise. Consequently, assessing both Lp(a) and traditional lipid panels remains vital for comprehensive cardiovascular disease risk stratification.

Thalia's proposed bispecific Lp(a)-PCSK9 medicines ('THAT-002' and 'THAT-003') have been designed to provide

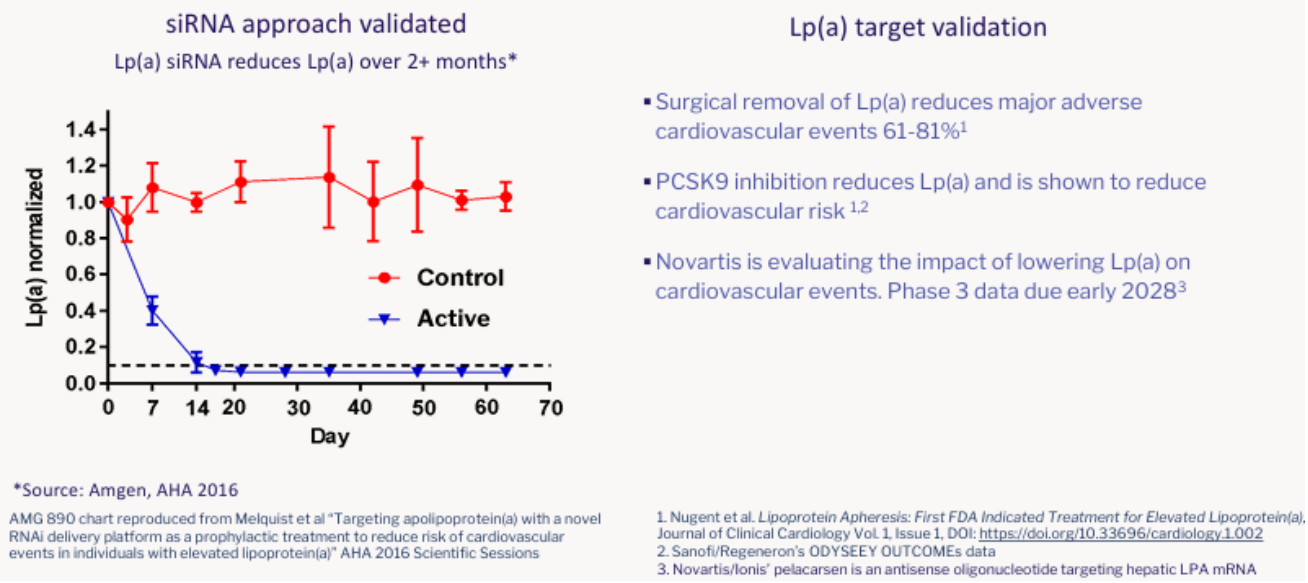
broad cardiovascular protection with the relative flexibility of long-duration repeat dosing, while also investigating opportunity for much wider cellular targeting through utilisation of the Group's proprietary delivery system, Nuvec®. The siRNA molecules within these two candidate drugs act like a 'molecular eraser' to temporarily block genetic messages after the DNA has already been read, without altering the subject's genome. These candidate drugs utilise the target cells' RNA-induced silencing complex ('RISC') to destroy the target mRNA before it can be translated into disease-driving proteins. Because this mechanism only targets transient mRNA, the effect is temporary and the body eventually resumes production of new messages from the unaltered gene. Consequently, siRNA medicines (such as inclisiran) require repeat injections approximately every six months to maintain low cholesterol levels. As synthetic oligonucleotide therapeutics, they are regulated as small molecule drugs and are reviewed under the NDA pathway rather than as biological products.

The therapeutic strategy proposed by Thalia is similar in its transient RNA-targeting approach to that utilised in Novartis's Phase 3 study of pelacarsen, an antisense oligonucleotide (ASO), rather than an siRNA, to control Lp(a) levels. However, it differs fundamentally from adenine base-editing medicines, such as Eli Lilly's early-stage investigational drug VERVE-102, which use molecular machinery to permanently rewrite DNA and thereby achieve a one-time, durable reduction in low-density lipoprotein cholesterol (LDL-C). VERVE-102, for which Phase 1b Heart-2 study results were published on May 25, 2026, is designed to permanently inactivate the PCSK9 gene in the liver to lower blood LDL cholesterol levels. This paradigm shift is generating growing interest in genetically targeted approaches to cardiovascular disease and PCSK9-targeted therapies, a trend clearly reflected in recent clinical research published in The New England Journal of Medicine.

Growing body of validated evidence for use of siRNA therapeutic payloads to target both Lp(a) and PCSK9

Thalia is targeting two distinct, destructive and genetically driven cardiovascular risk factors. Firstly, circulating in the bloodstream, Lp(a) significantly increases the risk of cardiovascular disease, myocardial infarction, and stroke. The LPA gene (located on chromosome 6q) provides instructions for building a protein called apolipoprotein(a), which is produced almost exclusively in the liver. This protein combines with LDL cholesterol to form a sticky, lipid-protein Lp(a) particle. The lowering of Lp(a) is considered something of a 'holy grail' for preventive cardiology, primarily because plasma levels of 70% to 90% are seen to be genetically determined, meaning that statins and traditional lifestyle interventions (such as dietary changes) have virtually no effect on it.

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



Source: Thalia, Investor Presentation June 2026

As such, validated evidence for using siRNA therapeutics to target Lp(a) represents an important paradigm shift, moving the focus from merely clearing circulating lipids to silencing the LPA gene directly in the liver to achieve unprecedented, sustained reductions. They utilise RNA interference (RNAi) to selectively degrade the mRNA

encoding apolipoprotein(a), the critical, rate-limiting structural component of the Lp(a) particle. Most such investigational siRNA agents are conjugated with GalNAc, a chemical tag that acts like a homing missile by binding specifically to asialoglycoprotein receptors on hepatocytes. Once inside the liver cell, the siRNA is loaded into the RISC, which continuously cleaves the LPA mRNA and thereby blocks protein translation. PCSK9 by contrast is a hepatic protein that binds to LDL receptors on liver cells and targets them for degradation. By silencing this protein, the liver can express more surface receptors to clear circulating 'bad' LDL cholesterol from the bloodstream. Unifying the profound lipid clearance of PCSK9 inhibition with the specialised anti-inflammatory and anti-thrombotic benefits of Lp(a) reduction, allows therapies to simultaneously target two distinct, genetically driven pathways of cardiovascular disease.

Recent Phase I and Phase II randomised controlled trials plus subsequent meta-analyses further support this assessment, demonstrating a consistent, robust profile for these siRNA candidates. Olpasiran (Amgen), for example, was evaluated in the Phase II OCEAN(a)-DOSE trial in patients with established ASCVD. Subcutaneous doses of 75 mg or higher, administered every 12 to 24 weeks, achieved a 70% to greater than 95% reduction in Lp(a) levels. Similarly, zerlasiran (Silence Therapeutics), evaluated in the ALPACAR-360 Phase II trial, demonstrated time-averaged placebo-adjusted reductions of 81% to 85% across 36 weeks of treatment at doses of 300 mg or 450 mg.

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

Source: Thalia, Investor Presentation June 2026

Thalia's two therapeutic candidates THAT-002 (Lp(a)-PCSK9–GalNAc) and THAT-003 (Lp(a)-PCSK9–GalNAc-Nuvec®) target these genetically validated biomarkers. Adopting a bispecific Lp(a)-PCSK9 strategy offers potential to significantly reduce residual cardiovascular risk by hitting both targets with a single therapeutic agent.

Long-acting Bispecific siRNA to Reduce Cardiovascular Risk

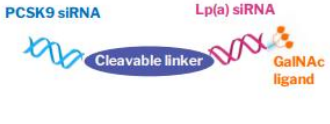
Simultaneously silences Lp(a) and PCSK9 genes, two clinically validated targets

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

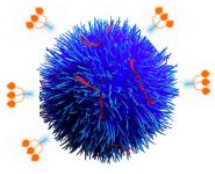
THAT-002
LP(a)-PCSK9 – GalNAc

- 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



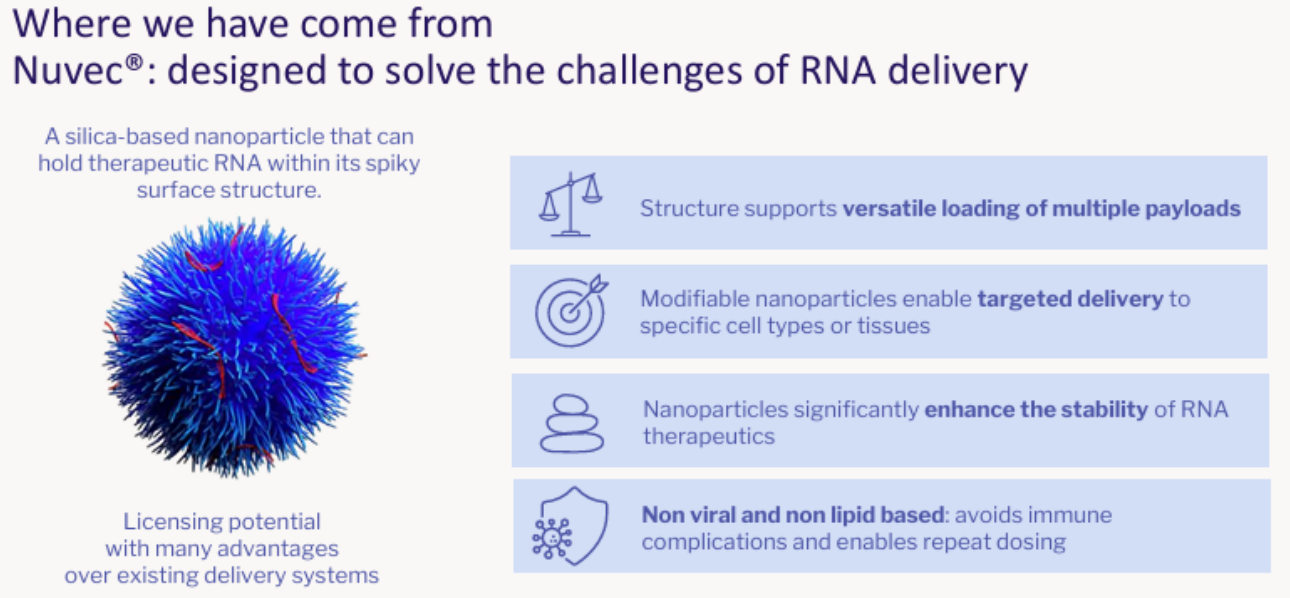
Source: Thalia, Investor Presentation June 2026

The approach simultaneously addresses hyperlipidemia alongside the pro-inflammatory and pro-thrombotic properties of Lp(a). Conjugated with GalNAc to drive deep, durable hepatic gene silencing, these candidates offer the potential for a convenient dosing schedule, potentially just one injection every 6 to 12 months, thereby fostering superior patient compliance.

THAT-003 differs from THAT-002 in that it has been formulated to utilise the Group's proprietary Nuvec® targeted delivery system. This silica nanoparticle platform was developed by N4 Pharma plc (prior to its being renamed, Thalia) and is engineered to deliver genetic material (such as mRNA or siRNA) directly into target cells. The platform overcomes a significant challenge in modern medicine by protecting payloads from degradation and preventing adverse immune responses. Nuvec® features a unique, irregular, 'spiky' surface topography that safely traps and shields oligonucleotides during transit. The nanoparticles can be surface modified to precisely target specific cell populations while sparing healthy tissue. Furthermore, they are amenable to oral administration via enteric-coated capsules and naturally attract antigen-presenting cells, making them highly effective for immunomodulatory applications like vaccines and cancer immunotherapies. These upcoming preclinical evaluations are also expected to establish definitive PoC for the Nuvec® platform in siRNA delivery.

Currently in the discovery phase, both will initially target ASCVD, which is the leading cause of morbidity and mortality globally. It is characterised by the narrowing and hardening of arteries due to the buildup of fatty plaques (atheromas), encompassing serious conditions such as coronary heart disease, stroke, and peripheral artery disease.

Patent application GB2603718.4 was filed for both THAT-002 and THAT-003 earlier in 2026. Thalia plans to initiate rodent and non-human primate ('NHP') toxicology studies simultaneously in mid-2026, aiming to achieve preclinical PoC in H1 2027. This timeline positions the Group to submit an IND application in H2 2027.



Source: Thalia, Investor Presentation June 2026

Thalia believes that its bispecific siRNA platform offers potential for an expedited regulatory pathway, potentially utilising a single pivotal clinical trial supported by robust non-human primate ('NHP') data. This outlook is based on the evolving stance of regulatory bodies, such as the FDA, which increasingly evaluate new siRNA candidates through the lens of established platform technologies rather than as entirely novel chemical entities. Because the chemical backbone modifications (such as phosphorothioate linkages, 2'-O-methyl (2'-OMe), and 2'-fluoro (2'-F) alterations) and the delivery vehicles remain consistent across an asset class, their toxicological profiles are highly predictable. Consequently, regulatory evaluation focuses primarily on sequence-specific safety and off-target risks, rather than re-evaluating a delivery platform already validated through prior approvals. Given that an NHP model (such as the cynomolgus monkey) shares 100% sequence homology with the human target mRNA, the pharmacodynamic knockdown observed in primates translates to clinical human trials with near-total fidelity. Subject to these *in vivo* studies producing satisfactory results for safety, tolerability and efficacy in 2026, the Board

considers it will potentially be on a fast track for first clinical trials with the size, duration, and overlap of between phases possibly being substantially streamlined (through inclusion of a combined Phase 1/2 study, for example, or even a single pivotal Phase 3) based on ability to meet unmet medical need with high severity.

The only other company currently pursuing a similar bivalent, dual-acting Lp(a)-PCSK9 siRNA approach targeting ASCVD is Rona Therapeutics Inc. Its Phase 1a, first-in-human single ascending dose study (Trial ID: NCT07347678) is registered to primarily evaluate the drug's safety, tolerability, pharmacokinetics and pharmacodynamics in healthy volunteers along with initial lipid-lowering efficacy. Dosing commenced in Q1 2026. Beyond this specific pairing, other prominent players in the RNA therapeutics landscape are also advancing multi-target mechanisms. Companies for instance are actively exploring dual-targeting siRNAs designed to simultaneously hit PCSK9 alongside other crucial cardiometabolic targets, such as angiotensinogen ('AGT') and ApoC3.

Thalia Therapeutics full year results to 31 December 2025 – Financial & operational highlights

Operational highlights for the 12 months to 31 December 2025

- Positive in vivo efficacy data for lead programme (N4 101) in an established inflammatory bowel disease model, demonstrating reduced inflammation and supporting the potential of Nuvec® for oral nucleic acid delivery.
- Validation of Nuvec® as a delivery vehicle for RNA therapeutics, with data from SRI International demonstrating targeted delivery of RNA into cancer cells, reinforcing applicability in oncology.
- Advancement towards clinical readiness, supported by ongoing collaboration with the Centre for Continuous Manufacturing and Advanced Crystallisation ('CMAC') at the University of Strathclyde, focused on scale-up and preclinical development.
- Strengthened operational and leadership capabilities, with the establishment of a Senior Leadership Team to support progression towards development and commercialisation.
- £1.75 million fundraise completed in April 2025, providing capital to advance development activities and support strategic objectives.
- Strategic review undertaken during the year, resulting in a refined focus on developing high-value RNA therapeutics, leveraging Nuvec® as a key delivery technology.

Post period-end operational highlights

- Transition to RNA therapeutics company, with post-period rebrand in February 2026 to Thalia Therapeutics plc, reflecting a shift from platform-only to therapeutics-led business model.
- Appointment of Dr David Horn Solomon as Chief Executive Officer in February 2026, bringing significant experience in RNA therapeutics and biotechnology value creation.
- Development of an initial RNA therapeutics pipeline, including a dual-targeting siRNA programme focused on PCSK9 and lipoprotein (a) for cardiovascular disease.
- Nuvec® clinical development progresses with CMAC at the University of Strathclyde, advancing process and formulation studies towards clinical-grade siRNA-loaded Nuvec®; LNP comparative data expected in Q2 2026, and in vivo targeting evaluation anticipated in Q3 2026.
- Advanced stage discussions to acquire a microRNA therapeutic clinical stage asset.

Financial summary for the 12 months to 31 December 2025

- Operating loss for the year was in line with budget at £1,165,221 (31 December 2024: £1,221,101 loss). Expenditure was broadly in line with the budget and saw an increase in general and administrative costs, reflecting the addition of the Senior Leadership Team and initial consultancy fees for David Solomon.
- Loss per share for the year 0.17p (31 December 2024: 0.31p).
- Cash at the year-end was £1,079,307 (31 December 2024: £625,972), supported by the £1.75 million gross fundraise completed in April 2025.

Source: Thalia, RNS of 23 June 2026

Appendix

City of Hope license agreement: Licensed patents

- (1) PCT/US2016/057143 and its national stage patents and patent applications including the table
- (2) US patent: 11,801,266; and
- (3) PCT/US2025/043392.

Patent App Number	Patent Number	Country
3,001,723		Canada
201680073019.6		China
16856317.9	3364984	EPO Countries
21203981.2		EPO Countries
16856317.9	3364984	France
16856317.9	3364984	Germany
2018-519358	7038045	Japan
16856317.9	3364984	United Kingdom
15/768,405	10,801,026	United States
17/010,259	11,591,596	United States
18/166,204	12,227,745	United States
19/016,464		United States

Source: Thalia, Investor Presentation June 2026

This document has been communicated for the attention of Professional and Institutional clients only. Retail clients (as defined by the rules of the FCA) must not rely on this document.

Past performance is not a guide to future performance and forecasts are not a reliable indicator of future results. The value of securities, particularly those of smaller companies, can fall as well as rise and may be subject to large and sudden swings. In addition, the level of marketability of smaller company securities may result in significant trading spreads and sometimes may lead to difficulties in opening and/or closing positions.

AIM is a market designed primarily for emerging or smaller companies and the rules of this market are less demanding than those of the Official List of the UK Listing Authority; consequently, AIM investments may not be suitable for some investors. Liquidity may be lower and hence some investments may be harder to realise.

Specific disclaimers

TPI acts as Joint Broker to Thalia Therapeutics plc. ('Thalia Therapeutics') which is listed on the AIM Market of the London Stock Exchange ('AIM').).

TPI as a business, its officers, private clients, or institutional clients may hold, subscribe for or buy or sell Thalia Therapeutics securities. Opinions and estimates in this document are entirely those of TPI as part of its internal research activity. TPI has no authority whatsoever to make any representation or warranty on behalf of Thalia Therapeutics.

General disclaimers

This document, which presents the views of TPI's research analyst, cannot be regarded as "investment research" in accordance with the FCA definition. The contents are based upon sources of information believed to be reliable but no warranty or representation, express or implied, is given as to their accuracy or completeness. Any opinion reflects TPI's judgement at the date of publication and neither TPI nor any of its directors or employees accepts any responsibility in respect of the information or recommendations contained herein which, moreover, are subject to change without notice. Any forecast or valuation given in this document is the theoretical result of a study of a range of possible outcomes and is not a forecast of a likely outcome or share price. TPI does not undertake to provide updates to any opinions or views expressed in this document. TPI accepts no liability whatsoever (in negligence or otherwise) for any loss howsoever arising from any use of this document or its contents or otherwise arising in connection with this document (except in respect of wilful default and to the extent that any such liability cannot be excluded by applicable law).

The information in this document is published solely for information purposes and is not to be construed as a solicitation or an offer to buy or sell any securities or related financial instruments. The material contained in the document is general information intended for recipients who understand the risks associated with equity investment in smaller companies. It does not constitute a personal recommendation as defined by the FCA or take into account the particular investment objectives, financial situation or needs of individual investors nor provide any indication as to whether an investment, a course of action or the associated risks are suitable for the recipient.

This document is approved and issued by TPI for publication only to UK persons who are authorised persons under the Financial Services and Markets Act 2000 and to professional clients, as defined by Directive 2004/39/EC as set out in the rules of the Financial Conduct Authority. This document may not be published, distributed or transmitted to persons in Japan, Canada or Australia. This document may not be copied or reproduced or re-distributed to any other person or organisation, in whole or in part, without TPI's prior written consent.

U.S. Persons Research Disclaimer

To the extent that any TPI research is furnished to U.S. recipients, it is furnished in reliance on Rule 15a-6 ("Rule 15a-6") under the U.S. Securities Exchange Act of 1934, as amended. The information contained in this research is intended solely for certain "major U.S. institutional investors" (as such term is defined in Rule 15a-6, an "MII") and may not be used or relied upon by any other person for any purpose. Each U.S. recipient of this research represents and agrees, by virtue of its acceptance thereof, that it is a MII and that it understands the risks involved in executing transactions in such securities. Any U.S. recipient of this research that wishes to discuss or receive additional information regarding any security mentioned herein, or engage in any transaction to purchase or sell or solicit or offer the purchase or sale of such securities, should contact a registered representative of Beech Hill Securities, Inc., a U.S. broker-dealer registered with the Securities and Exchange Commission and a Member of FINRA, located at 880 Third Avenue, 16th Floor, New York, NY 10022. Any transaction by a U.S. person (other than a registered U.S. broker-dealer or bank acting in a broker-dealer capacity) must be effected with or through Beech Hill Securities, Inc., which may be contacted via telephone at +1 (212) 350-7200.

This research was prepared by the analyst named on the cover of this research, who is a non-U.S. research analyst of TPI and, as such, may not be subject to all requirements applicable to U.S.-based analysts. All of the views expressed in this research accurately reflect the research analyst's personal view about all of the subject securities or research subjects and no part of such analyst's compensation was, is, or will be related to the specific recommendation or view contained in this research.

To the extent this research relates to non-U.S. securities, note that investing in non-U.S. securities may entail particular risks. Such securities may not be registered under the U.S. Securities Act of 1933, as amended, and the issuer of such securities may not be subject to U.S. reporting and/or other requirements. Financial statements included in research with respect to such securities, if any, may have been prepared in accordance with non-U.S. accounting standards that may not be comparable to the financial statements of U.S. companies. Available information regarding the issuers of such securities may be limited, and such issuers may not be subject to the same auditing and reporting standards as U.S. issuers. Fluctuations in the values of national currencies, as well as the potential for governmental restrictions on currency movements, can significantly erode principal and investment returns. Market rules, conventions and practices may differ from U.S. markets, adding to transaction costs or causing delays in the purchase or sale of such securities. Securities of some non- U.S. companies may not be as liquid as securities of comparable U.S. companies.

The information contained herein may include forward-looking statements within the meaning of U.S. federal securities laws that are subject to risks and uncertainties. Factors that could cause a company's actual results and financial condition to differ from expectations include, without limitation: political uncertainty, changes in general economic conditions that adversely affect the level of demand for the company's products or services, changes in foreign exchange markets, changes in international and domestic financial markets and in the competitive environment, and other factors relating to the foregoing. All forward-looking statements contained in this research are qualified in their entirety by this cautionary statement.

No TPI party accepts any liability whatsoever for any direct or consequential loss of any kind arising out of the use or reliance on the information given. Research does not take into account the specific investment objectives and financial situation of any recipient, nor do they provide individually tailored investment advice or offer tax, regulatory, accounting or legal advice. Prior to entering into any proposed transaction, recipients should determine, in consultation with their own investment, legal, tax, regulatory and accounting advisors, the economic risks and merits, as well as the legal, tax, regulatory and accounting characteristics and consequences, of the transaction. Investors seeking to buy or sell any financial instruments discussed or recommended in research, should seek independent financial advice relating thereto. The products discussed in research are not FDIC insured, may lose value and are not guaranteed by any TPI party.