COILED THERAPEUTICS

Unravelling Cancer's Blueprint

Precision oncology advancing first-in-class therapeutics for patients with high-risk cancers.

AIM: **COIL** | OTCQB: **COTXF**

INVESTOR PRESENTATION Q2 2026

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Why Coiled?

COILED THERAPEUTICS

1

Clinical Activity Before Targeted Exposure Levels Achieved

Clinical activity has emerged before targeted exposure levels and before maximum tolerated dose has been established. Cohort 4b demonstrated an 80% Clinical Benefit Rate versus 40% with once-daily dosing.

2

Only Clinical-Stage TACC3 Inhibitor Globally

AO-252 is the only clinical-stage TACC3 inhibitor globally, targeting a biologically validated oncology pathway with no approved competitors. Independent DepMap analysis confirms TACC3 as a selective tumour dependency.

3

Differentiated Pharmacology & Brain Penetration

AO-252 combines oral administration, blood-brain barrier penetration (most therapies cannot), direct cytotoxicity, and cGAS/STING immune activation in a single small molecule, supporting monotherapy and combination strategies.

4

Proven Development Model & Shareholder Alignment

Leadership previously advanced the MLL-Menin programme into Biomea Fusion, which completed a Nasdaq IPO and later exceeded a $1 billion market capitalisation. Management invested £1 million alongside institutional investors at AIM admission.

5

Active Strategic Engagement

Multiple pharmaceutical organisations are engaged with the Company, including three under Confidentiality Agreement. These discussions have informed the design of the 2026 clinical programme and partnering data package.

6

Catalyst-Rich 12-Month Outlook

Upcoming milestones include food effect PK data, next-generation formulation rollout, expansion cohorts, IO combination studies and development of a partner-relevant H2 2026 data package.

AO-252 First –in-Class Oncology Asset with Emerging Clinical Activity and Multiple Near-Term Catalysts

Clinical activity, differentiated biology and a catalyst-rich development strategy support multiple pathways to value creation.

Validated Target

Only clinical-stage TACC3 inhibitor with no direct competitors currently in the clinic

Emerging Clinical Activity

80% clinical benefit rate observed while dose optimisation remains ongoing

Expansion Cohorts Underway

Ovarian and prostate cancer cohorts designed to evaluate efficacy, biomarkers and commercial relevance

Active Pharmaceutical Engagement

Multiple pharma organisations engaged, including three under CDA, and all informing programme development

Phase 1 Trial: Evidence of Clinical Activity Before Maximum Exposure

Clinical benefit, durable responses and tumour reductions have emerged while dose optimisation remains ongoing.

PHASE 1 (NCT06136884)

80%

Clinical Benefit Rate

Cohort 4b — twice-daily dosing

≥6 mo

Duration of Response

vs. 2–3 mo salvage benchmark

5

Median Prior Therapies

Heavily pre-treated population

NO SAEs

No Serious Adverse Events

Maximum tolerated dose not reached. Clinical activity emerging before target exposure

SUPPORTING CLINICAL OBSERVATIONS

- BID dosing increased CBR from 40% to 80%
- Tumour reductions observed in ovarian and endometrial cancer
- Maximum tolerated dose not yet reached
- Clinical activity observed before targeted exposure levels achieved
- Clinical activity, durability and tumour reductions have emerged before targeted exposure levels have been achieved, supporting continued optimisation potential

2026 Clinical Expansion Strategy: Ovarian & Prostate Cancer

Expansion strategy guided by emerging clinical activity, biomarker rationale and strategic relevance.
Targeting ~40 Patients Across Ovarian and Prostate Expansion Cohorts by Q3 2026

Ovarian Cancer

Efficacy demonstrated. Expansion programme underway.

Why it matters

- Significant unmet need following PARP resistance
- Limited treatment options in advanced disease
- Potential CNS opportunity

Why AO-252 fits

- Distinct DNA damage repair mechanism
- Brain-penetrant small molecule
- Early clinical activity observed

Prostate Cancer

First patient enrolled. Biomarker enrichment hypothesis strongest.

Why it matters

- Resistance emerges following AR-targeted therapies
- Strong biomarker enrichment opportunity
- Significant unmet need in advanced disease

Why AO-252 fits

- Independent of androgen receptor signalling
- Biomarker-driven approach targeting TP53/TACC3 biology
- Combination potential with IO and ADC therapies

Ovarian and prostate cancer offer the clearest path to demonstrating clinical relevance, biomarker utility and partnering potential.

Why Pharma Cares

PARTNER-RELEVANT CLINICAL DESIGN

2026 expansion cohorts designed to generate clinically meaningful and commercially relevant data.

STRATEGIC ONCOLOGY CATEGORIES

Focused on tumour types with established partnering and acquisition activity.

ACTIVE ENGAGEMENT

Multiple pharmaceutical organisations engaged, including three under Confidentiality Agreement

Why AO-252 Matters Commercially

350K+

Estimated Addressable Patients

Biomarker-defined populations across ovarian, prostate, TNBC, endometrial, gastric, and lung cancers in the US and Europe.

>$20B

Established Commercial Oncology Markets

PARP, CDK4/6 and AR inhibitors demonstrate the commercial value of precision oncology and biomarker-driven treatment selection

>$3B

Recent Strategic Transaction Benchmark

Johnson & Johnson's acquisition of Halda Therapeutics (November 2025) highlights continued pharmaceutical demand for differentiated Phase 1 oncology assets in advanced prostate cancer.

COMMERCIAL VALIDATION

- **DNA Damage Repair**
  PARP inhibitors established the commercial importance of targeting DNA repair pathways.
- **Precision Oncology**
  CDK4/6 inhibitors demonstrated how differentiated targeted therapies can evolve into multi-billion-dollar franchises.
- **Resistant Disease**
  AR inhibitors validated large commercial opportunities in treatment-resistant patient populations.

Value Creation Roadmap

2026–2027 CLINICAL MILESTONES

Late Q2 2026

- Dose escalation completion
- Food effect PK data
- Recommended expansion strategy

H2 2026
Clinical Dataset Build-Out

- New formulation rollout
- Enrolment acceleration
- Expansion cohorts initiated
- Target 20–30 patients treated

H1 2027

- Key Value Inflection
- Preliminary efficacy dataset
- ~50-patient safety database
- Biomarker validation
- Prostate cancer cohort update
- Partnering data package

H2 2027

- Strategic Execution
- Big Pharma partnering discussions
- Registrational pathway alignment
- RP2D confirmation
- Potential Phase 2 strategy

PARTNERING STRATEGY

Capital-Efficient Development
Advance AO-252 through key clinical value inflection points while preserving capital and strategic flexibility.

Partner-Relevant Data Package
Building the clinical dataset — efficacy, safety, biomarker — to support formal pharma BD discussions

Strategic Partnerships
Pursue licensing, co-development and strategic investment opportunities to accelerate development and share risk.

Flexible Commercialisation
Preserve optionality through independent development, regional licensing or broader strategic transactions.

Transaction Benchmark: J&J's acquisition of Halda Therapeutics (November 2025, $3.05B) confirms continued pharma demand for differentiated Phase 1 oncology assets in advanced prostate cancer.

Recent Oncology Transactions

Recent oncology transactions demonstrate continued strategic demand for differentiated Phase 1 assets in commercially significant indications.

DATE	ACQUIRER	TARGET	UPFRONT PAYMENT	MILESTONES / CVRS	TOTAL POTENTIAL VALUE	PATIENT NUMBER	PHASE	INDICATIONS
Apr-26	Eli Lilly	Kelonia	$3.25 Billion	$3.75 Billion	$7.0 Billion	4	(Platform play)	Leukemia
May-26	Merck	Terns Pharma	$6.7Billion	-	$5.8 Billion	85	Ph. 1/2	Chronic Leukemia
Nov-25	**J&J**	**Halda**	**$3.05 Billion**	**-**	**$3.05 Billion**	**31**	**Ph. 1/2**	**Prostate and ER Breast**
Jan-25	Eli Lilly	Scorpion	$1.5 Billion	$1.0 Billion	$2.5 Billion	40-50	Ph. 1	ER+ Breast
Jan-24	J&J	Ambrx	$2.0 Billion	-	$2.0 Billion	51	Ph. 1	Prostate
May-24	Genmab	Profound Bio	$2.0 Billion	-	$2.0 Billion	80	Ph. 1/2	Ovarian & Endometrium

Recent oncology transactions demonstrate that differentiated Phase 1 assets in large commercial indications continue to command multi-billion-dollar strategic valuations.

AO-252 Investment Case

AO-252 combines early clinical activity, multiple near-term catalysts and strategic optionality within a first-in-class oncology programme.

1

Clinical Validation Already Established

80% Clinical Benefit Rate in Cohort 4b, tumour reductions observed, no serious adverse events and MTD not yet reached.

2

Five Catalysts Before YE 2026

Expansion cohorts, next-generation formulation, combination strategy initiation, partner-relevant data package and enrollment milestones.

3

Partner-Relevant Development Strategy

Clinical expansion priorities have been designed to generate data relevant to future strategic discussions.

4

Valuation Anchored to Transactions, Not Projections

Recent oncology transactions have demonstrated multi-billion-dollar valuations for differentiated Phase 1 assets in ovarian and prostate cancer. AO-252 is advancing within those same categories while maintaining a market capitalisation substantially below comparable transaction benchmarks.

TACC3: An Untapped Oncology Target

TACC3 is overexpressed across multiple aggressive cancers and appears essential for tumour survival, while healthy adult tissue shows limited dependence.

Pan-Cancer Relevance. TACC3 is overexpressed across numerous commercially significant cancer types including ovarian, prostate, triple-negative breast, endometrial, gastric, bladder, sarcoma, lung, and brain metastases.

Cancer Requires It. Adult Tissue Does Not. Preclinical research suggests many cancer cells depend on TACC3 for survival, while healthy adult tissue demonstrates significantly lower dependence.

Centrosome Amplification Driver. TACC3 drives centrosome amplification, genomic instability and tumour progression—hallmarks of aggressive cancers. Once considered undruggable due to its protein structure, TACC3 is now targetable through Coiled's AO-252.

Independent Validation. Broad Institute DepMap analysis across thousands of cancer cell lines confirms TACC3 as a selective survival dependency in multiple tumour types. Similar approaches helped validate targets including BRCA, KRAS and MET before successful therapies emerged.

Limited Competitive Landscape. Despite growing scientific interest in TACC3, AO-252 is currently the only clinical-stage programme directly targeting the pathway.

AO-252: How it Works & Why It Matters

Combines direct cancer-cell killing and immune activation in a single oral therapy.

1

Targeted DNA Damage Induction

AO-252 disrupts TACC3, causing DNA damage and replication stress in cancer cells

2

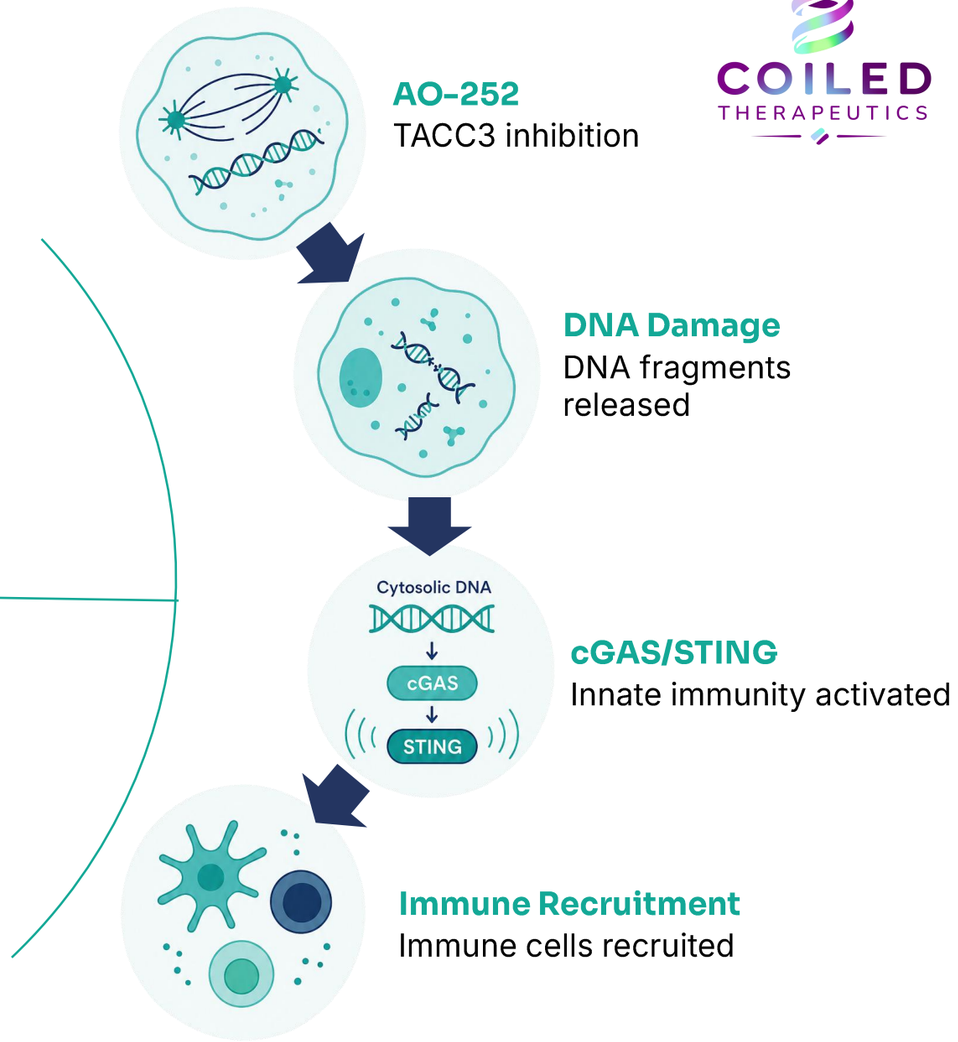
Innate Immune Activation (cGAS/STING)

Damaged DNA activates cGAS/STING, triggering an anti-tumour immune response

3

Direct Cancer Killing + Immune Activation

AO-252 attacks tumours through direct cancer-cell killing and immune activation



AO-252 combines two anti-tumour mechanisms in a single oral therapy, potentially broadening activity across multiple cancer types and treatment settings.

AO-252 converts DNA damage into immune activation

AO-252: Multiple Sources of Clinical Differentiaton

CLEAN TOLERABILITY PROFILE
No serious adverse events observed to date. MTD not yet reached.

EXCEPTIONAL TARGET SELECTIVITY
Designed to maximise tumour targeting while minimising off-target effects.

ORAL ADMINISTRATION
Oral tablet with combination therapy potential.

One Molecule. Three Advantages.
1. **Tumour disruption**
2. **Immune activation**
3. **Brain penetration**

BIOMARKER STRATEGY
Enables targeted patient selection and clinical development efficiency.

BRAIN PENETRATION
Demonstrated CNS activity in brain metastasis models.

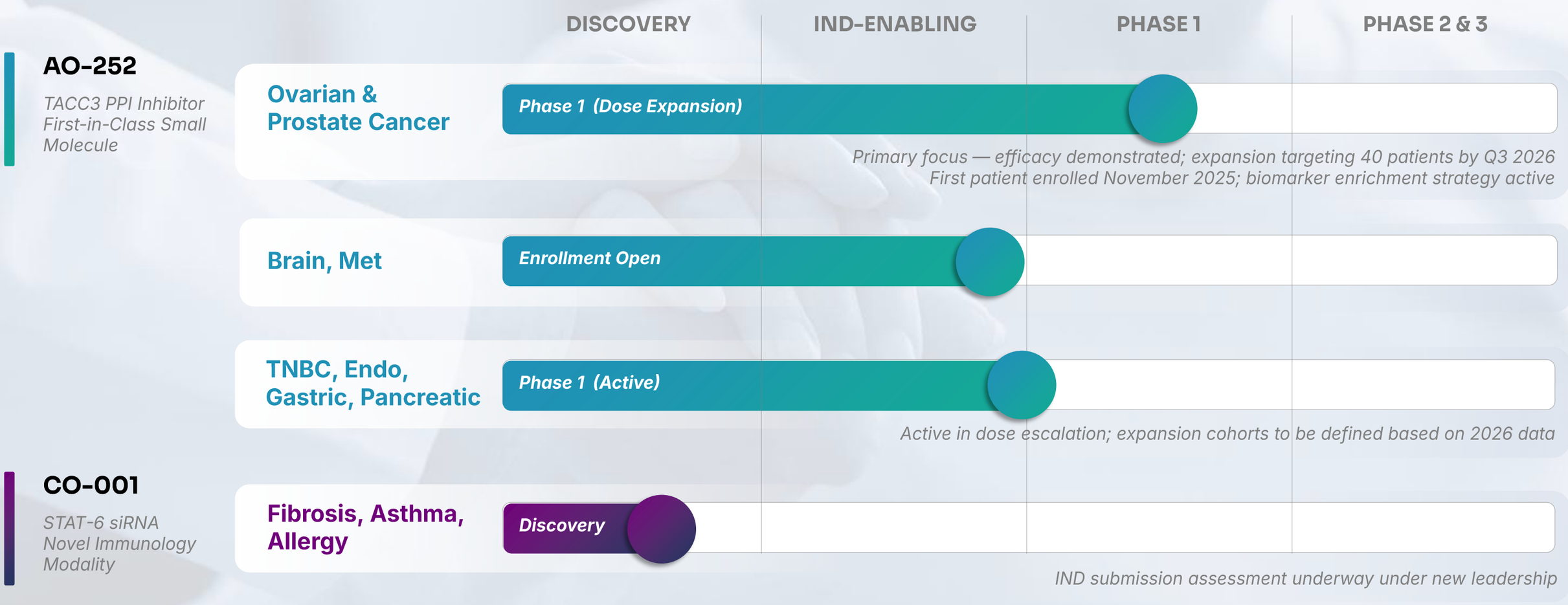
DUAL MECHANISM OF ACTION
Direct tumour disruption plus, innate immune activation in a single small molecule.

Potential Clinical Opportunities for AO-252

CLINICAL PROBLEM	EXISTING THERAPIES	CURRENT TREATMENT GAPS	AO-252 ADVANTAGE
Advanced Ovarian Cancer After PARP Resistance Most patients with advanced ovarian cancer eventually develop resistance to existing therapies, with limited treatment options remaining.	PARP inhibitors, Antibody drug conjugates	PARP resistance develops and brain metastases remain largely untreated.	Distinct mechanism beyond PARP inhibitors
Castration-Resistant Prostate Cancer Resistance to next-generation AR therapies continues to narrow treatment options in advanced prostate cancer.	Any indication after 1L of an anti-AR therapy, taxanes, radioligands	Resistance emerges following AR-targeted therapies and treatment options narrow significantly.	AR-independent mechanism with biomarker strategy
Solid Tumours with Brain Metastases Brain metastases remain difficult to treat because most targeted therapies fail to achieve meaningful CNS penetration.	ADCs, kinase inhibitors, biologics	Most targeted therapies fail to achieve meaningful CNS penetration.	Oral and brain-penetrant
Potential Combination Therapy Applications	Tubulin agents disruption potential	Toxicity and limited tolerability	Differentiated profile with combination potential

This analysis reflects the current hypothesis underlying the AO-252 programme. Clinical outcomes in dose expansion cohorts will determine the degree to which these limitations are overcome.

Pipeline



CO-001 (STAT-6 siRNA IND submission assessment underway under new leadership): Targets the same therapeutic pathway as Dupixent, which generated revenue exceeding £10 billion in 2024. The siRNA approach silences all STAT-6 isoforms at the mRNA level — mechanistically distinct from the degraders and inhibitors currently in clinical development.

Management & Board of Directors

Dr Sotirios Stergiopoulos

EXECUTIVE CHAIRMAN

- Physician executive with extensive pharmaceutical experience, particularly in Oncology
- Former CMO of multi-billion dollar Euronext-listed Ipsen
- Former Attending Physician and trainee at Albert Einstein College of Medicine, Harvard Medical School, and National Institutes of Health
- Masters in Biotechnology Enterprise and Entrepreneurship (MBEE) from Johns Hopkins University; Medical Degree from Poznan University of Medical Sciences (Poland)

Sridhar Vempati

CHIEF EXECUTIVE OFFICER

- ~20 years in drug discovery, oncology research & business strategy; proven track record advancing novel therapeutics from concept to clinical development
- Co-founded A2A Pharmaceuticals in 2016, previous business development roles at Ironwood Pharmaceuticals and Rafael Pharmaceuticals, and Equity Research analyst at Jefferies LLC
- Postdoctoral fellowship in leukaemia research at Dana Farber Cancer Institute (Harvard University), PhD in molecular biology from Ludwig-Maximilians-University, Germany, MBA from Boston University

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- CEO & Director at Repronovo SA
- Former director, Exec VP & Group General Counsel at Ferring International Center

Craig Tooman (Independent NED)

- President, Chief Executive Officer and Board Member adnd former CFO, Silence Therapeutics
- Former CuraVac, Vyome Therapeutics, Aratana Therapeutics

Stephen West

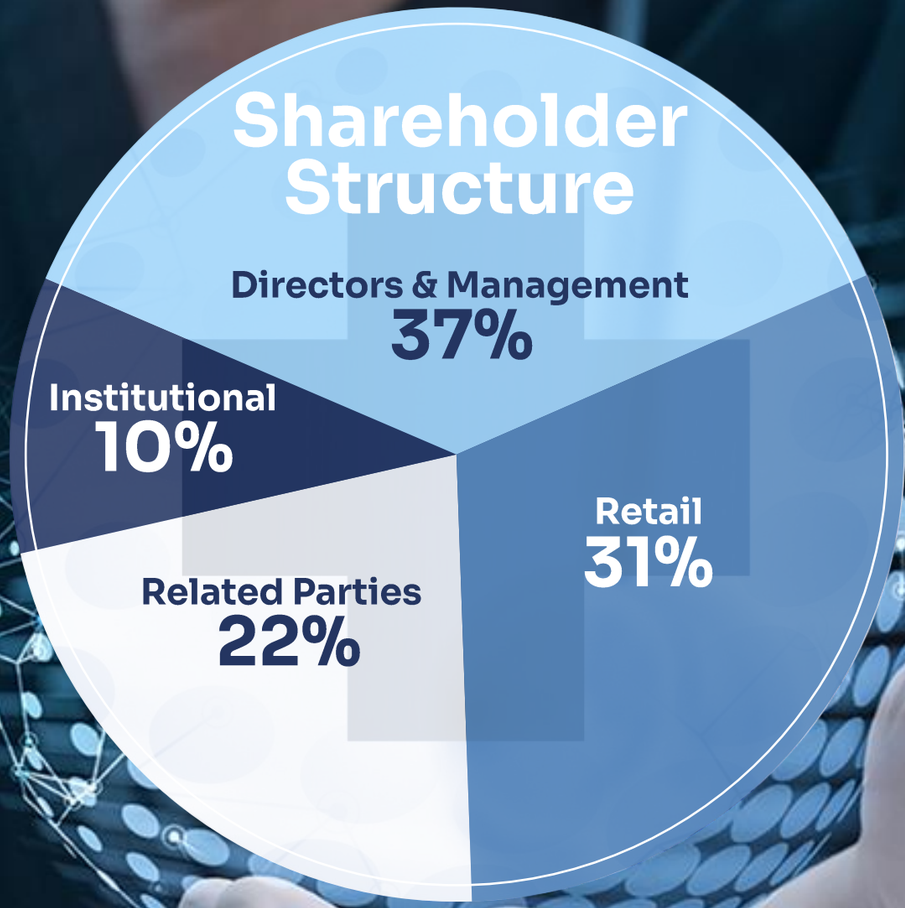
- Fellow Chartered Accountant, 30+ years' international finance, corporate and public company experience
- Co-founder of EnergyPathways plc (AIM:EPP), Roquefort Therapeutics plc, TollCyto Therapeutics Ltd & ParisBio Ltd

Dr Andrew Dean

- Head of Oncology, St John of God Subiaco Hospital, Western Australia; consults for GenesisCare
- Board member, Valo Therapeutics; research expertise in solid tumour molecular profiling and novel ovarian cancer therapies

Capital Structure & Financials

AIM Admission	**27 March 2026**
Placing Price	**10 pence per share**
Gross Fundraise	**£8.5 million (~$11.3M USD)**
Market Cap	**~£40 million (May 2026)** *Share price ~10p*
Total Shares Outstanding	**425,856,539**



Capital Structure & Financials

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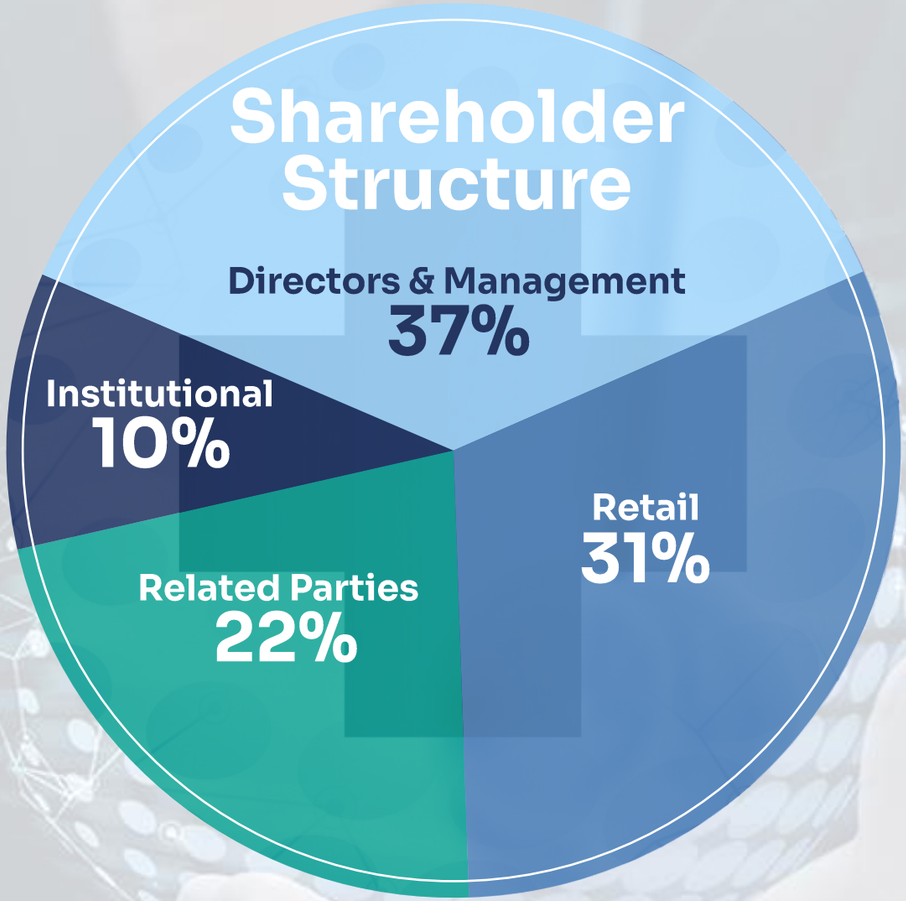

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COILED THERAPEUTICS

Appendix

TACC3: A Validated Cancer Dependency

WHAT IS TACC3?

TACC3 is a protein essential for cell division. Many cancers become highly dependent on TACC3 to sustain growth and survive genetic stress.

Why is it important?

Why it matters

- Frequently overexpressed in aggressive cancers
- Linked to tumour progression and poorer outcomes
- Supports chromosome stability and cancer cell division
- Identified as a survival dependency across multiple tumour types

Why is it considered validated?

TACC3 is considered a validated target because cancer cells depend on it for survival, and disrupting its function has been shown to impair tumour growth.

Why it matters

- Decades of research confirm TACC3's critical biological role
- Cancer studies consistently show elevated TACC3 in tumours
- Genetic and pharmacological inhibition reduces tumour growth in preclinical models
- Clinical datasets associate high TACC3 expression with worse patient outcomes

Novel Drug Target. Proven Cancer Biology.

TACC3 has been extensively validated as a cancer dependency but has historically remained difficult to target therapeutically, creating a differentiated development opportunity.

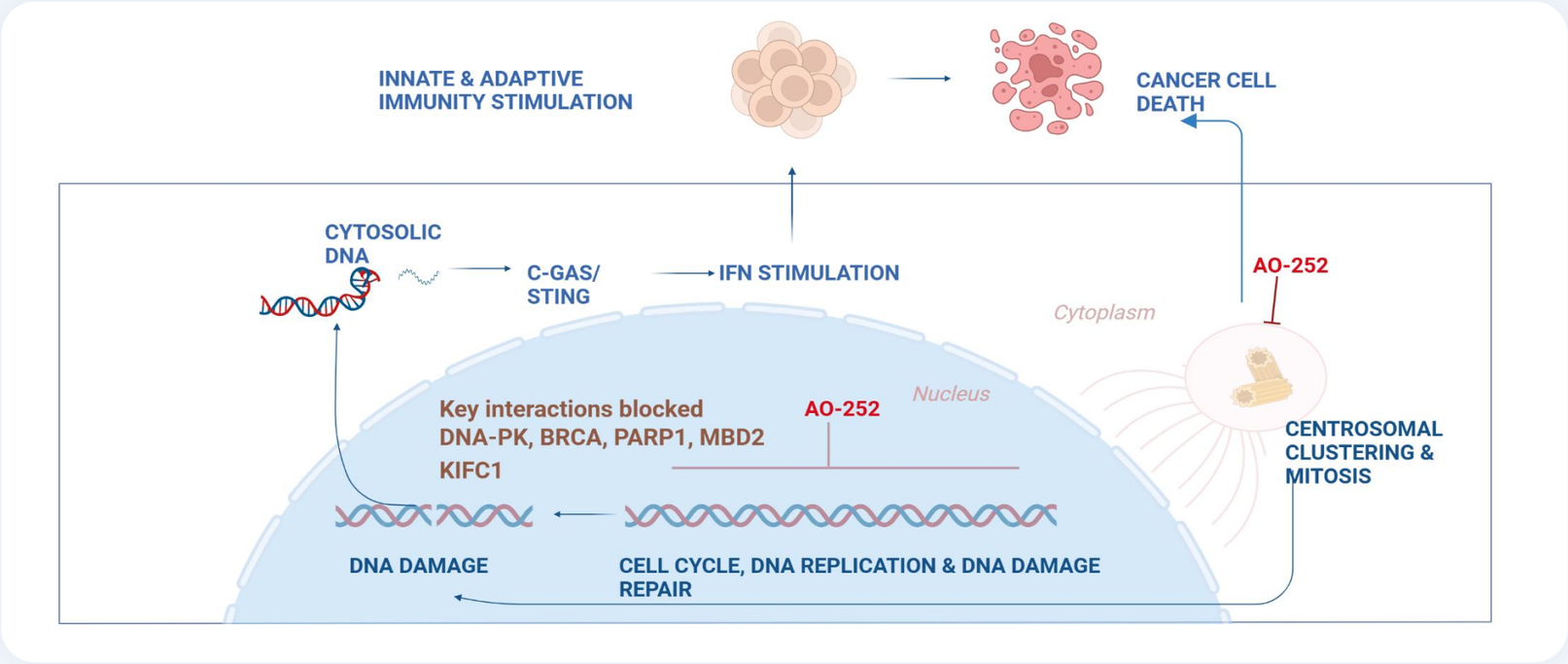
WHY DOES IT MATTER NOW?

Historically considered difficult to target, advances in drug design are now enabling companies such as Coiled to pursue this well-validated cancer vulnerability.

AO-252 Mechanism of Action Visual

MULTI-TARGETED PPI DISRUPTION

By disrupting TACC3's protein-protein interactions, AO-252 induces mitotic & replication stress through impairment of the DNA damage repair process and activation of immunity, leading to cancer cell death, particularly in TP53-mutant cells, while showing minimal toxicity in healthy cells. AO-252 operates through a sophisticated mechanism that inhibits multiple critical protein-protein interactions at the TACC3 C-terminal domain. This disruption cascades across key proteins including DNA-PK, PARP1, BRCA, KU70, KIFC1, MBD2, and HDAC2 - collectively involved in mitosis, DNA damage repair, replication, and transcription.



Biomarker Strategy

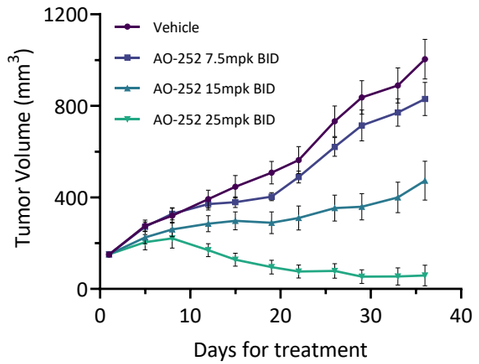
Patient selection based on TP53 mutation status, high centrosomal amplification, and elevated TACC3 expression ensures precision targeting of responsive populations.

Robust Pre-Clinical Validation

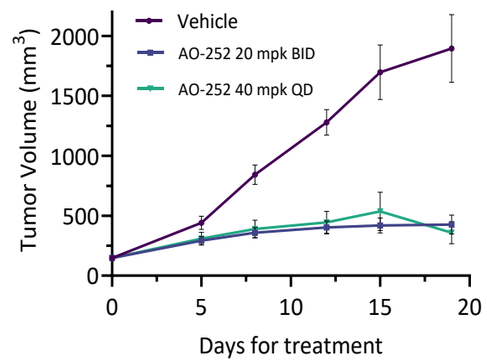
AO-252 demonstrated **tumour regression as monotherapy** across multiple difficult-to-treat solid tumour models, including ovarian, triple-negative breast cancer (TNBC), endometrial, gastric, and prostate cancers. Notably, the compound showed robust efficacy in in vivo brain metastases models, addressing a critical unmet medical need.

Tumour regression rates of 88-120% were observed, with partial to complete responses consistently achieved across all tested indications. Additional strong activity was confirmed in NSCLC, SCLC, bladder, and esophageal cancer models.

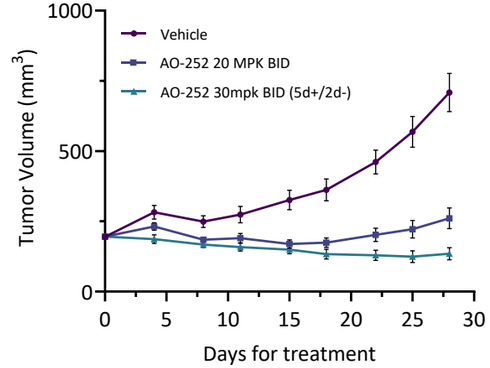
MDA-MB-231 (TNBC)



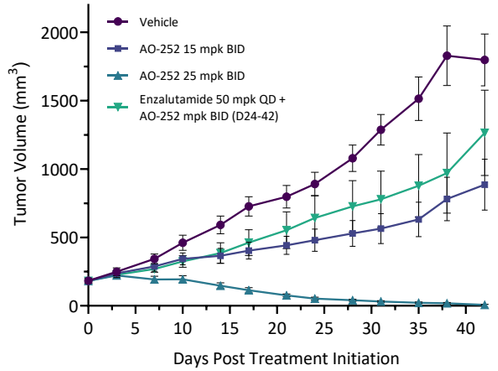
HEC-59 (Endometrium)



SKOV3 (Ovarian)



VCAP (Prostate)



Clean Safety Profile

- No toxicities observed in 28-day GLP studies (rats & dogs)
- Negative genetic toxicology across AMES and clastogenicity assays

Selective Kinase Inhibition

- Only 4 of 468 kinases inhibited >65% at 1µM concentration
- Demonstrates exceptional target selectivity

Strong Therapeutic Index

- Therapeutic window of ≥3-5X maintained across all indications tested
- Supports favourable risk-benefit profile

STAT-6 Programme — A Second Pipeline Opportunity

STAT-6 is the master transcription factor behind IL-4/IL-13 signalling — the same pathway Dupixent targets upstream to generate over £10 billion in 2024 revenue. CO-001 uses siRNA to silence STAT-6 at the mRNA level, a mechanistically distinct and potentially more complete approach than the degraders and inhibitors currently in clinical development.

Current Landscape

Multiple STAT-6 degraders have entered the clinic with early efficacy signals, confirming target validity. CO-001 is differentiated: siRNA silencing operates upstream of protein production, avoiding the limitations of degradation-dependent or binding-dependent strategies.

Broader Silencing

mRNA-level silencing prevents all STAT-6 isoforms from forming including splice variants that degraders and inhibitors may leave active. This completeness of suppression is the core mechanistic advantage.

Reduced Risk

Partial target engagement (characteristic of degraders and inhibitors) can trigger compensatory isoform upregulation. Silencing at the mRNA level removes this escape mechanism, reducing the risk of resistance or attenuated response over time.

Ind Assessment Underway

The new leadership team is conducting a formal assessment of the CO-001 programme for IND submission readiness and Phase 1 trial design. A go/no-go decision is expected within six to twelve months.

Company History

2025

Coiled Therapeutics plc was spun out of A2A Pharmaceuticals in 2025 to advance AO-252, a first-in-class TACC3 inhibitor already in Phase 1 human trials.

2026

March – Completed a reverse takeover of AIM-listed Roquefort Therapeutics, raised £8.5 million at 10 pence per share from institutional and retail investors, and began trading on AIM under the ticker COIL on 27 March 2026.

May – The Company commenced cross-trading on the OTCQB Venture Market (COTXF), extending access to US-based investors.

This is not the first time this team has executed this model. In 2018, A2A Pharmaceuticals spun out its MLL-Menin inhibitor programme into Biomea Fusion. Biomea completed a Nasdaq IPO in 2021, raising $153 million at a $464 million listing market capitalisation — peaking above $1 billion.

The same founders. The same model.
A clinical asset at a more advanced stage than any previous Coiled predecessor.

Company Facts

Exchange	**AIM (London) + OTCQB (US)**
Tickers	**COIL (AIM) | COTXF (OTCQB)**
AIM Admission	**27 March 2026**
OTCQB Listing	**19 May 2026**
Shares Outstanding	**425,856,539**
Placing Price	**10 pence per share**
Gross Proceeds	**£8.5 million (~$11.3M USD)**
Market Cap	**~£40 million (May 2026)**
ISIN	**GB00BSHRN331**

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