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 Q4 2026

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Why Coiled?

1 Unlocking a Validated Cancer Dependency with No Approved Targeted Therapy

AO-252 is the only clinical-stage programme targeting TACC3, a biologically validated cancer dependency implicated across multiple aggressive solid tumours with independent DepMap analysis supporting TACC3 as a selective tumour vulnerability.

2 Human Proof-of-Concept Already Emerging

An 80% Clinical Benefit Rate has been observed before dose optimisation. Newer lipid-based formulation now active, with initial safety and PK data anticipated Q4 2026.

3 Designed to Win Where Others Cannot

AO-252 combines oral administration, blood-brain barrier penetration, a multi-modal mechanism of action, and biomarker-driven patient selection to support both monotherapy and combination therapy in a single small molecule.

4 Catalyst-Rich Through 2027

Initial safety and PK data anticipated Q4 2026, followed by preliminary efficacy data, biomarker validation, combination studies and regulatory interactions in 2027.

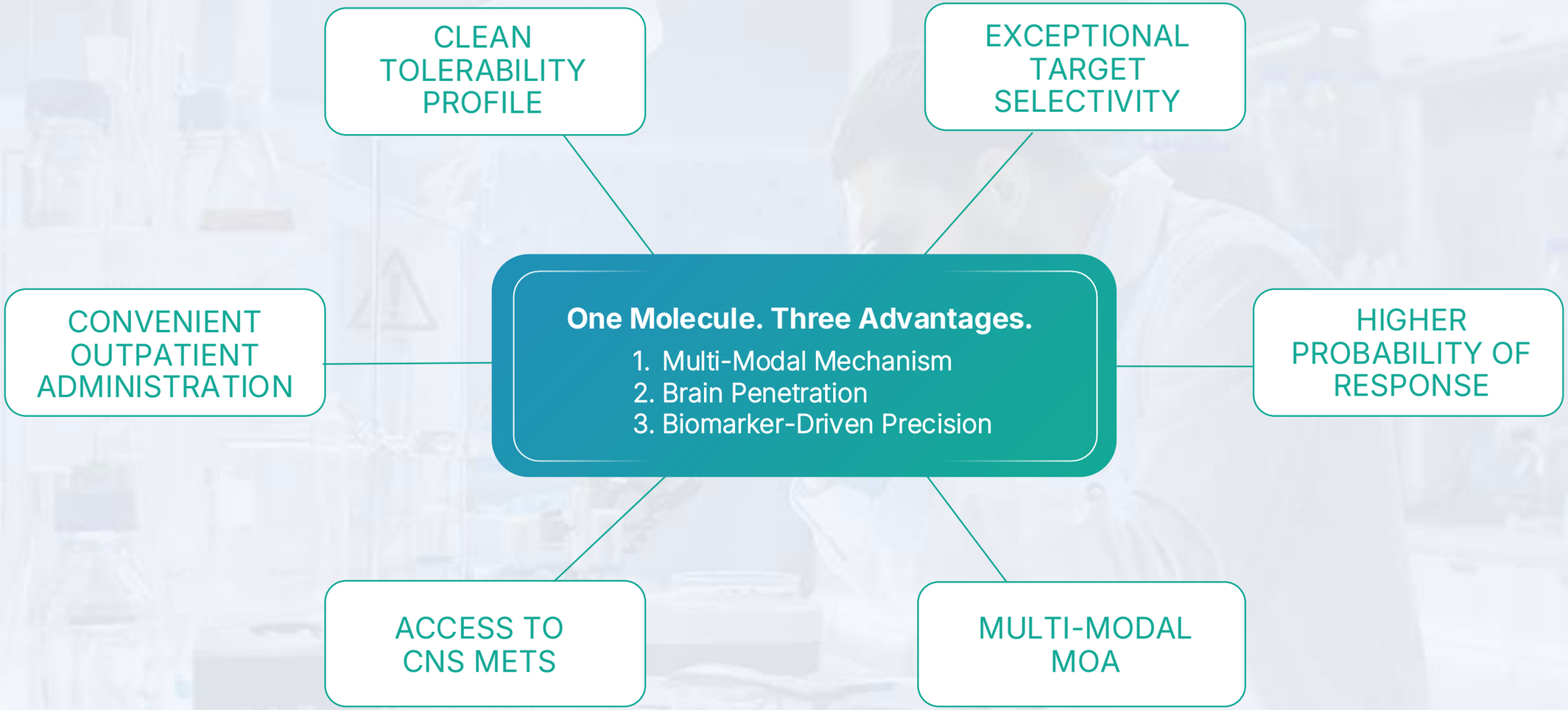
5 Multiple Paths to Shareholder Value

Clinical development is focused on commercially attractive indications and partner-relevant data generation, preserving multiple pathways to value creation, including independent development, licensing and strategic partnerships.

6 Experienced Oncology Leadership

Management previously repeatedly advanced oncology assets from discovery through clinical development, public markets, and strategic transactions.

AO-252 Clinical Differentiation



TACC3: A Validated Cancer Dependency With No Approved Targeted Therapy

Validated Biology

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.

Cancer Dependency

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.

Now Druggable

Advances in structure-guided drug design have transformed TACC3 from an historically undruggable target into a clinically actionable opportunity.

AO-252: One Molecule. Multi-Modal MOA.

1

Multi-Modal Mechanism of Action

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

2

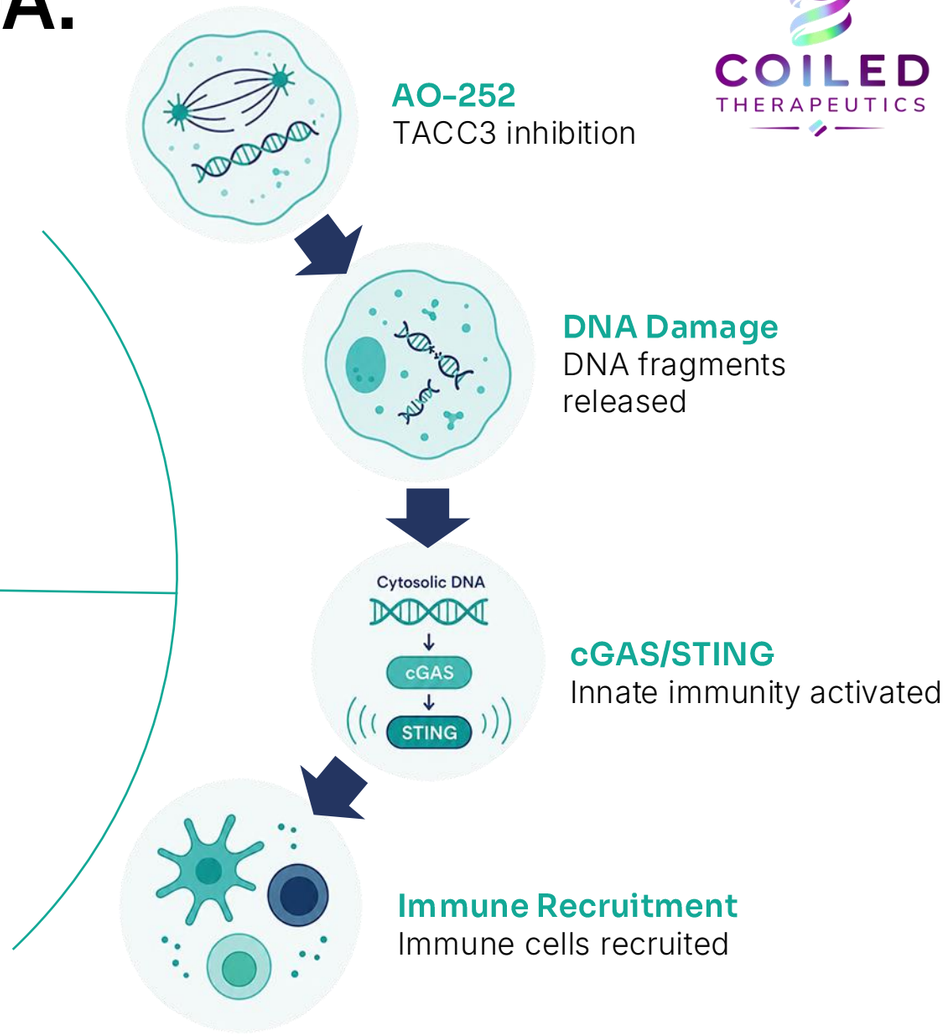
Innate Immune Activation (cGAS/STING)

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



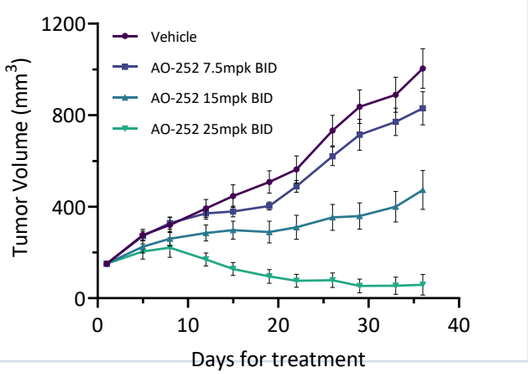
AO-252 combines targeted cytotoxicity with innate immune activation in a single oral small molecule.

AO-252: Robust Pre-Clinical Validation

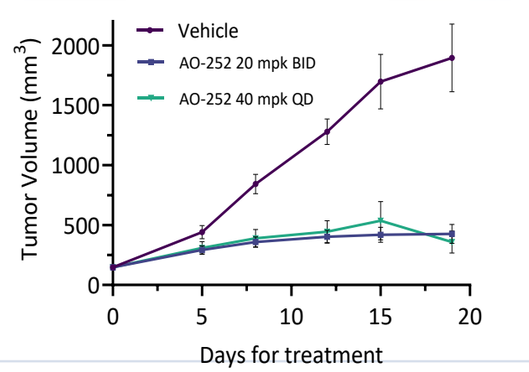
Demonstrated **tumour regression as monotherapy** across multiple difficult-to-treat solid tumour models, including ovarian, triple-negative breast cancer (TNBC), endometrial, gastric, and prostate cancers. Notably, the compound showed robust efficacy in in vivo bra in metastases models, addressing a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

Tumour growth inhibition (TGI) 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.

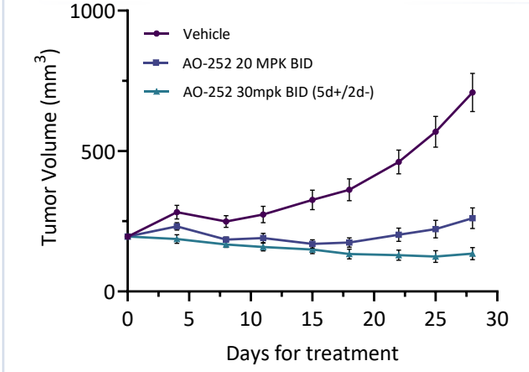
MDA-MB-231 (TNBC)



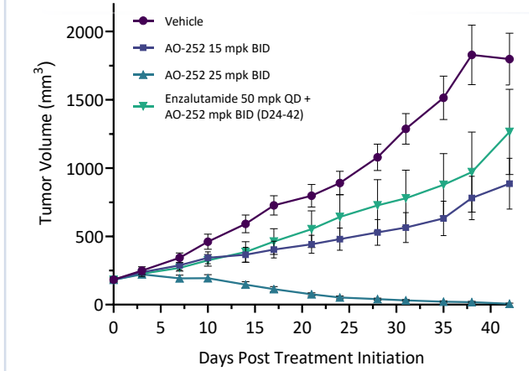
HEC-59 (Endometrium)



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

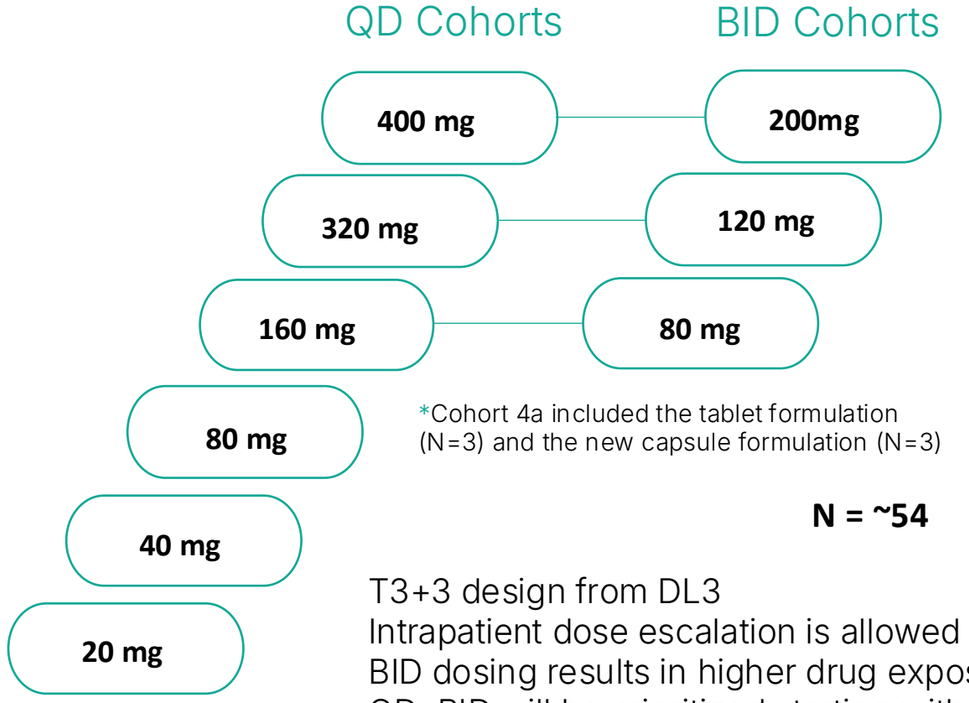
- Only 4 of 468 kinases inhibited >65% at 1µM concentration
- Demonstrates exceptional target selectivity

Strong Therapeutic Index

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

AO-252: Phase I Design and Summary

Part A: Monotherapy Dose Escalation
Accelerated titration design



*Cohort 4a included the tablet formulation (N=3) and the new capsule formulation (N=3)

N = ~54

T3+3 design from DL3
Intrapatient dose escalation is allowed
BID dosing results in higher drug exposure than QD; BID will be prioritized starting with cohort 5

Part B: Dose Expansion
2 Doses

Advanced Solid Tumors

With or without Brain Metastases

N = ~32 (max 16/dose)

Tumor specific cohort

TBD

Endpoints

Primary Endpoints:
Safety and tolerability
Determine the MTD/RP2D

Secondary & Exploratory Endpoints:
Pharmacokinetics
Pharmacodynamics
Response rate (RECIST 1.1)
Duration of Response
Progression free survival
Overall survival

AO-252 Phase 1 Clinical Trial: Robust Early Signals

Clinical benefit while dose optimisation remains ongoing.

PHASE 1 (NCT06136884)

80%
Clinical Benefit Rate
Cohort 4b — 2x daily dosing; 20% ORR

NO SAEs
No Serious Adverse Events
Room to optimise as maximum tolerated dose not reached

5
Median Prior Therapies
Heavily pre-treated population

≥6 mo
Duration of Response
vs. 2–3 mo. salvage benchmark

ENCOURAGING CLINICAL SIGNALS

Bid Dosing increased CBR from 40% to 80%

Durability & Tumour Reductions observed in ovarian and endometrial cancer, before targeted exposure levels have been achieved

Maximum Tolerated Dose not yet reached, supporting continued optimisation

Newer Lipid-based Softgel Formulation Active, designed to enhance bioavailability and reduce absorption variability, building on 80% CBR signal

120mg BID And 160mg QD Cohorts Enrolled, all patients progressing per protocol; on track to dose 30 patients by YE 2026

AO-252: Clinical Development Strategy

Initially focused on indications where differentiated biology, early clinical activity and significant unmet need create the clearest path to value creation.

Ovarian Cancer

Lead Expansion Cohort
Early Efficacy Observed

Why?

Advanced disease following PARP inhibitor resistance with few effective treatment options remaining

Current Treatment

- PARP inhibitors & ADCs

Why AO-252?

- Distinct DNA damage mechanism
- Brain-penetrant
- Early clinical activity observed
- Large unmet need

Prostate Cancer

Second Expansion Cohort
Biomarker-driven development

Why?

Treatment resistance develops after androgen receptor therapies despite multiple approved options

Current Treatment

- AR inhibitors, Taxanes & Radioligands

Why AO-252?

- AR-independent mechanism
- Biomarker strategy
- Combination potential
- Large validated commercial market

Platform Expansion Opportunities

Following clinical validation, potential to expand across:

- Brain metastases
- Additional solid tumours
- IO combinations
- ADC combinations

PURPOSE-BUILT CLINICAL DESIGN

To generate relevant clinical evidence for partners supporting Phase 2 planning, biomarker validation, future indication expansion.

Targeting enrollment of up to 30 by end of 2026

AO-252 Commercial Opportunity

Development within commercially validated oncology markets where precision medicines have already created multi-billion-dollar value.

350K+

Estimated Addressable Patients

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

>$20B

Proven Oncology Markets

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

>$3B

Recent Strategic Transaction Benchmark

Johnson & Johnson's acquisition of Halda highlights continued pharmaceutical demand for differentiated early-stage precision oncology assets.

Commercial Validation

DNA Repair PARP inhibitors established multi-billion-dollar markets by targeting DNA repair biology.

Precision Medicine CDK4/6 inhibitors demonstrated the value of biomarker-informed treatment selection.

Resistant Disease AR inhibitors created large commercial opportunities by treating patients with therapy-resistant disease.

AO-252 Value Creation Roadmap

2026–2027 CLINICAL MILESTONES

Significant progress achieved

- New formulation introduced
- Two initial dose-escalation cohorts enrolled
- Accelerated patient enrolment, on track to dose up to 30 patients with new formulation

H2 2026
Clinical Dataset Build-Out

- Initial safety and pharmacokinetic data from newer lipid-based formulation cohorts anticipated before YE 2026
- Dose escalation / expansion data
- Combination trial initiation

H1 2027

- Key Value Inflection
- Preliminary efficacy dataset
- ~50-patient safety database
- Biomarker validation
- Indication specific cohort updates
- Partnering data package

H2 2027

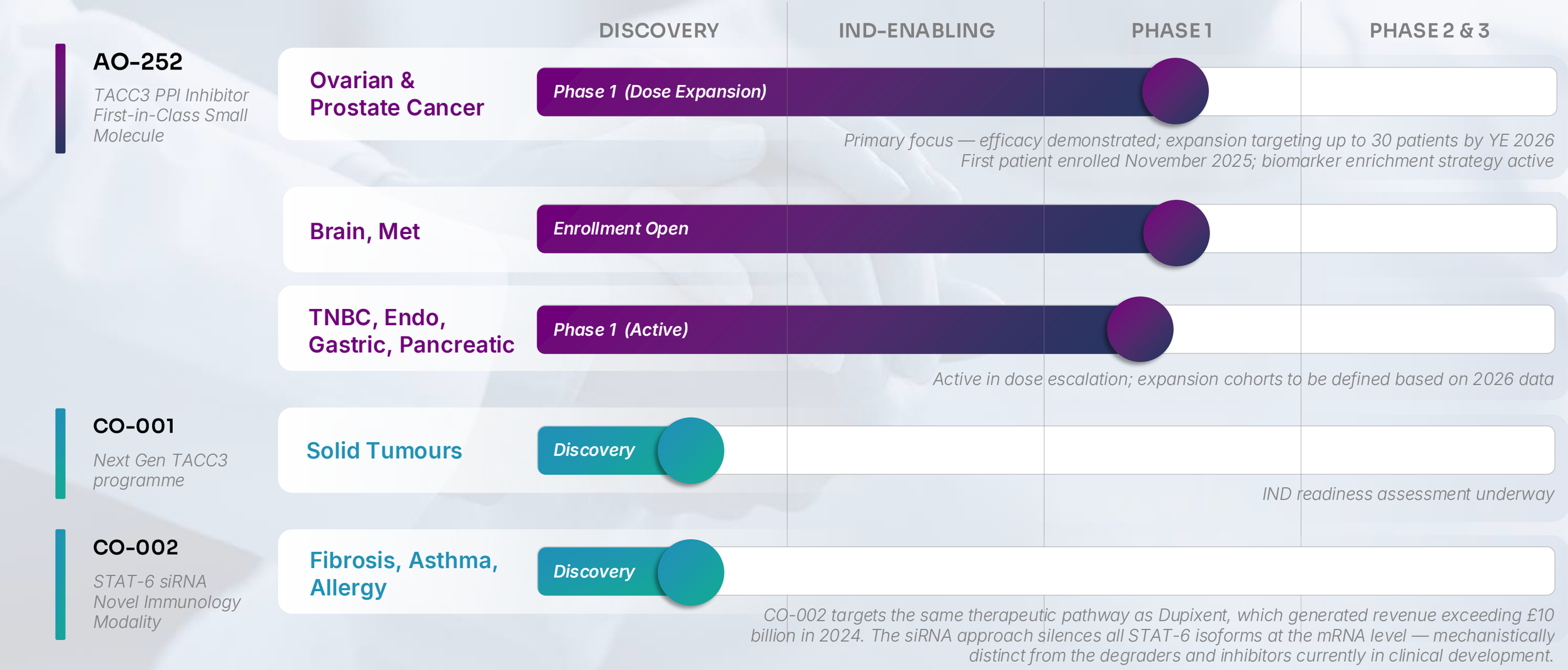
- Strategic Execution
- Big Pharma partnering discussions
- Registrational pathway alignment
- RP2D confirmation
- Potential Phase 2 strategy

Recent Oncology Transactions

Comparable transactions vary by modality and maturity but demonstrate sustained pharmaceutical demand for differentiated early-stage oncology assets.

DATE	ACQUIRER	TARGET	UPFRONT PAYMENT	MILESTONES / CVRS	TOTAL DEAL VALUE	PATIENT NUMBER	PHASE	INDICATIONS
Jul-26	Novartis	Myricx Bio	$1.1B	Up to $400M	~$1.5B	18	Pre-clinical / ADC payload	Multiple solid tumours
Apr-26	Eli Lilly	Kelonia	$3.25B	$3.75B	$7.0B	4	(Platform play)	Leukemia
Mar-26	Merck	Terns Pharma	-	-	$6.7B	85	Phase 1/2	Chronic Leukemia
Nov-25	J&J	Halda	$3.05B	-	$3.05B	31	Phase 1/2	Prostate and ER Breast
Jan-25	**Eli Lilly**	**Scorpion**	**$1.5B**	**$1.0B**	**$2.5B**	**40-50**	**Phase 1**	**ER+ Breast**
May-24	Genmab	Profound Bio	$2.0B	-	$2.0B	80	Phase 1/2	Ovarian & Endometrium
Jan-24	J&J	Ambrx	$2.0B	-	$2.0B	51	Phase 1	Prostate

Pipeline: Multi-Modal, Multi-Indication



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

NON-EXECUTIVE DIRECTORS

Jean Duvall (Independent NED)

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

Craig Tooman (Independent NED)

- President, Chief Executive Officer, Board Member and 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

Five Reasons

1 First-in-Class Clinical Asset

- Human Proof-of-Concept Emerging
- Only Clinical-Stage TACC3 Programme
- Clinical Activity Before Optimisation

2 Commercially Relevant Development Strategy

- Ovarian
- Biomarkers
- Prostate

3 Near-Term Value Inflection Points

- 2026–2027 Catalysts

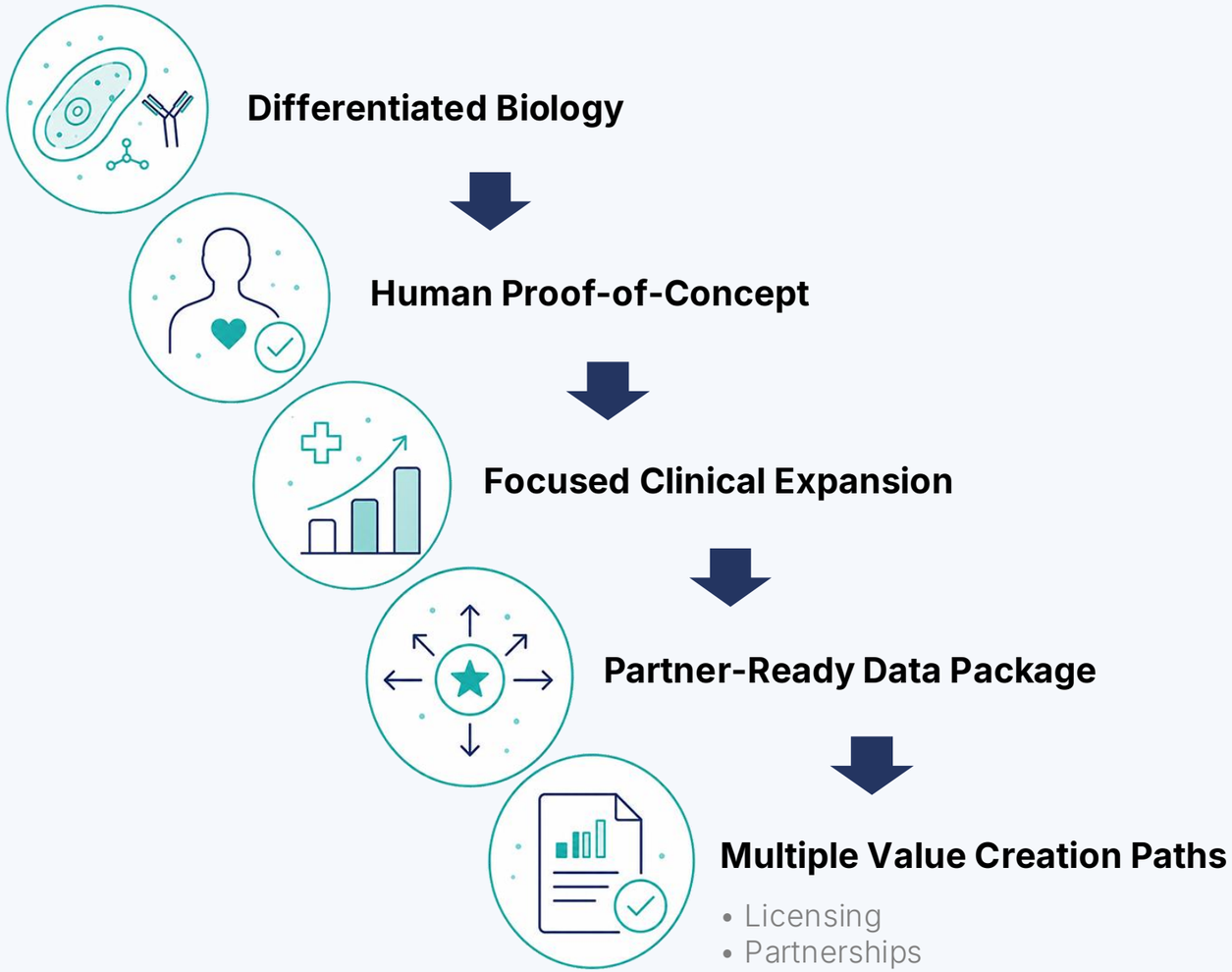
4 Multiple Paths to Value Creation

- Independent development
- Regional deals
- Licensing
- Acquisition

5 Experienced Team With Prior Execution

- Biomea
- Silence
- Ipsen
- Harvard

Coiled Development Strategy



Thank You

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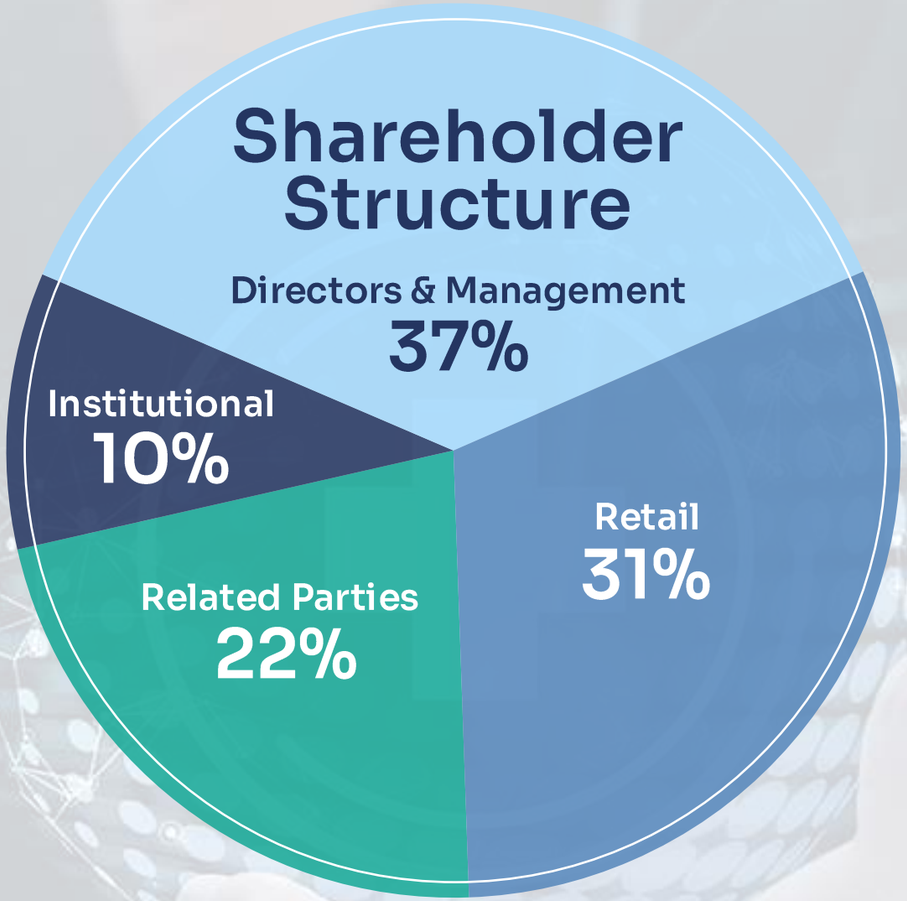
Appendix

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

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	**426,791,322**

Fully funded into 2027, securing operations through major H2 2026 clinical data readouts.



Company Strategy & History

2025

Coiled Therapeutics, Inc. spun out of A2A Pharmaceuticals to advance AO-252, a first-in-class TACC3 inhibitor already in Phase 1 clinical development.

2026

March – 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.

Prior Execution

The founders previously created Biomea Fusion through the spin-out of A2A's MLL-Menin programme, culminating in a Nasdaq IPO and a market capitalisation exceeding US$1 billion.

Coiled applies the same disciplined development model to a more advanced clinical asset.

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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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.

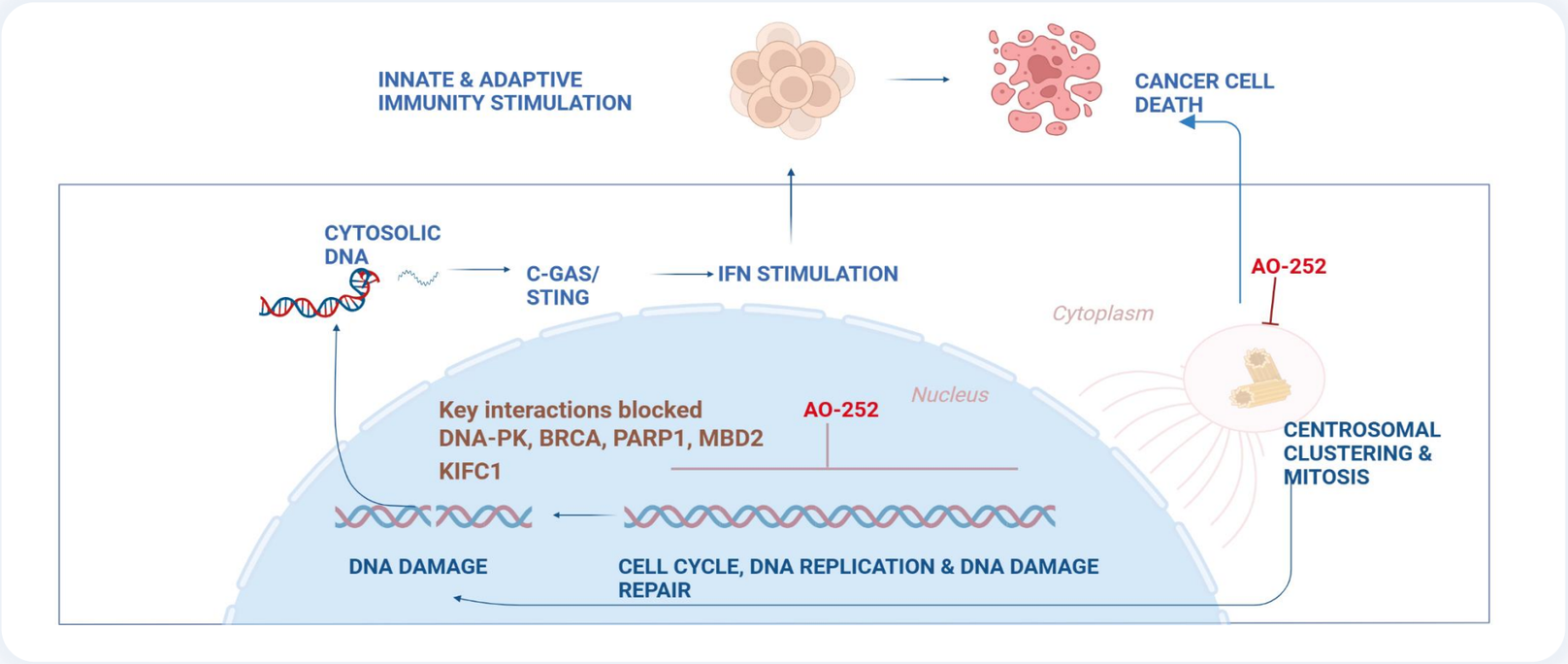
WHY DOES IT MATTER NOW?

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.



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-002 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.