EVOLUTION CAPITAL

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Old Drug, New Tricks: Bisantrene Reimagined

Racura Oncology Ltd.

Racura Oncology (ASX: RAC) is developing RC220, a proprietary intravenous formulation of (E,E)-bisantrene, across three concurrent clinical programmes in oncology. The company's thesis rests on a 2025 mechanistic discovery: that bisantrene acts primarily by stabilising G-quadruplex DNA and RNA structures, suppressing the master oncogene MYC; a target implicated in over 70% (est.) of human cancers and historically considered undruggable. This reframing transforms a shelved 1980s chemotherapeutic into a potentially differentiated precision oncology asset with multi-indication reach. The pipeline spans three distinct unmet needs.

Non-Small Cell Lung Cancer (NSCLC). HARNESS-1 (Phase 1a/b) combines RC220 with osimertinib in EGFR-mutant non-small cell lung cancer, targeting the near-universal acquired resistance that develops after approximately 18 months on third-generation EGFR tyrosine kinase inhibitors (TKIs). This is a setting where no approved targeted therapy exists post-progression.

Cardioprotection Program (CPACS). Racura's Phase 1a/b combines RC220 with doxorubicin in advanced solid tumours, testing the dual hypothesis that bisantrene enhances anticancer activity while protecting the heart from anthracycline-induced cardiotoxicity – a decades-old problem with only one approved pharmacological solution.

Relapsed or Refractory Acute Myeloid Leukaemia (R/R AML). A Phase 3 in R/R AML leverages bisantrene's historical 46% monotherapy response rate and two positive Phase 2 investigator-sponsored trials (40% response rates in heavily pretreated patients), though this programme is partner-dependent.

Our risk-adjusted NPV analysis values Racura at A$4.81/sh, representing 84% upside to the current A$2.61 share price. The valuation is anchored by the cardioprotection programme (58% of enterprise value), which addresses an estimated 750,000 anthracycline-treated patients annually in developed markets with a differentiated dual-benefit profile, and the NSCLC programme (45% of EV), which targets a resistance pathway (the MYC-driven transcriptional bypass) that no approved or late-stage competitor addresses. With A$19.4 million in pro-forma cash (runway through CY2027), composition-of-matter patents filed to extend exclusivity to approximately 2045, and first clinical data readouts from both CPACS and HARNESS-1 expected in H2 2026, we see a catalyst-rich 12-month window that the current valuation does not reflect.

Recommendation	**SPEC BUY**
Price Target	**$4.81**
Share Price	**$2.61**
TSR	**+84%**

Company Profile	
Market Cap	$493M
Enterprise Value	$472M
SOI (diluted)	206.9M
Free Float	71.7%
ADV (3-month)	$549.5k
52-Week Range	$1.115 - $4.90

Price Performance

%	1M	3M	12M
Absolute	-14.1%	8.7%	91%
ASX/S&P200	-5.3%	-5.7%	1.9%

Company Overview

Racura Oncology (ASX: RAC) is a clinical-stage Australian oncology company developing RC220, a proprietary IV formulation of (E,E)-bisantrene, across three concurrent programmes. Near-term catalysts include initial CPACS dose-escalation data and HARNESS-1 (RC220 + osimertinib in EGFRm NSCLC) safety and PK readouts, both expected in H2 2026.

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Figure 1: Key upcoming catalysts. Source: Company guidance, ASX announcements. Timing based on company disclosures and analyst estimates.

Catalyst	Est. Timing	Significance
HARNESS-1 first patient dosed	Q2 CY2026	Proof of operational execution
CPACS dose escalation cohort 2 initiation	Q2 CY2026	Dose-response data begins
CPACS interim safety/PK data	H2 CY2026	First human PK and cardiac biomarker signals
HARNESS-1 initial safety/PK data	H2 CY2026	First human data in NSCLC combination
AML Phase 3 partnering / regulatory discussions	CY2026-2027	Pathway to pivotal AML trial

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Investment Thesis

Three persistent oncology unmet needs share a common thread. In EGFR-mutant lung cancer, virtually every patient on osimertinib (a US$7B-annual drug) develops resistance within ~18 months, with no approved targeted therapy after progression; MYC amplification and overexpression are increasingly implicated, yet MYC has resisted direct pharmacological intervention for decades. In anthracycline chemotherapy, dose-limiting cardiotoxicity forces oncologists to stop effective treatment before maximum benefit; the only approved cardioprotectant (dexrazoxane) is restricted to late-stage cumulative dosing and used in a minority of eligible patients. In R/R AML, patients failing two or more lines face survival measured in weeks with few salvage options beyond palliative care.

Racura's thesis is that a single mechanism - G4-quadruplex stabilisation and the resulting MYC suppression - can address all three. The 2025 discovery that bisantrene acts via G4 binding rather than conventional DNA damage explains decades of anomalous clinical observations. Lower cardiotoxicity versus doxorubicin (4% vs 23% serious cardiac events, Phase 3 head-to-head) was puzzling under the prior anthracycline classification but is mechanistically coherent under a fundamentally different mode of action. Broad-spectrum activity across diverse cancer types, kinase-inhibitor synergy, and immune-stimulatory properties are all consistent with multi-target G4-mediated pharmacology. The RC220 formulation resolves the solubility limitations that prevented bisantrene from commercial viability despite clinical activity in 40+ historical trials and a 1988 French approval for AML.

The investment case rests on three pillars. First, asymmetric risk-reward: Racura trades at A$2.61 against our A$4.81 rNPV-based fair value, anchored by cardioprotection (58% of EV) and NSCLC (45%) – both addressing large, well-defined populations with clear commercial pathways. The rNPV is corroborated by three independent cross-checks - US peer trading comps (A$4.00–5.50 central), implied PoS scenarios (A$4.80 base, A$6.02 catalyst-adjusted), and AML precedent-transaction sensitivity (A$5.00–5.50) - converging on a triangulated band of A$3.40–6.30/sh; cross-method convergence reinforces conviction that the central estimate is not a model artefact. Second, a catalyst-dense 12 months: CPACS dose escalation data (with the first human cardiac biomarker signals) and HARNESS-1 initial safety/PK are both expected in H2 2026 - two independent readouts on the G4/MYC mechanism in humans. Positive signals from either would materially de-risk the platform thesis and compress the discount applied to a Phase 1 asset. Third, partial downside cover: an 1,800+ patient bisantrene clinical database and an AML programme with reproducible 40% response rates that provides near-term partnering optionality independent of the earlier-stage programmes.

Figure 2: Pipeline summary. Source: Company announcements, clinicaltrials.gov.

Programme	Indication	Phase	Combination	Status
RAC-010 / CPACS	Advanced Solid Tumours	Phase 1a/b	+ Doxorubicin	3 patients dosed; 1st cohort complete
HARNESS-1	EGFRm NSCLC	Phase 1a/b	+ Osimertinib	Governance approved; site initiation Mar 2026
AML Phase 3	R/R AML	Phase 3-ready	Mono / Combo	Bridging strategy; partner-dependent

Financial Summary

VALUATION DETAILS	
Share Price (A$)	$2.61
Market Cap (A$m)	$493
EV (A$m)	$472
Fair Value/Sh (A$)	$4.81

STATEMENTS (A$m)	FY25	FY26E	FY27E	FY28E	FY29E
Income Statement					
Revenue	0.0	0.0	0.0	8.6	8.6
EBITDA	-11.8	-13.3	-15.4	-9.7	-13.3
EBIT	-11.8	-13.4	-15.5	-9.8	-13.4
Net Income	-11.3	-13.2	-15.1	-9.0	-12.8
Balance Sheet					
Cash & Equivalents	3.4	8.9	19.4	14.2	69.5
Inventory	0.0	0.0	0.0	0.0	0.0
Receivables	0.0	0.0	0.0	0.7	0.7
Other Assets	3.8	3.8	3.8	3.8	3.8
Total Assets	7.2	12.8	23.2	18.7	74.1
Total Debt	0.0	0.0	0.0	0.0	0.0
Other Liabilities	1.5	1.7	1.9	2.3	2.7
Total Liabilities	1.5	1.7	1.9	2.3	2.7
Shareholders' Equity	5.8	11.1	21.2	16.4	71.3
Cash Flow Statement					
Net Income	-11.3	-13.2	-15.1	-9.0	-12.8
Add: D&A	0.1	0.1	0.1	0.1	0.1
Less: Change in NWC	0.0	0.2	0.3	-0.4	0.5
Cash Flow from Ops.	-10.2	-11.7	-13.6	-7.8	-10.8
Cash Flow from Inv.	-0.1	-0.1	-0.1	-0.1	-0.1
Equity Raised (net)	0.0	17.3	24.1	2.6	66.3
Less: Dividends Paid	0.0	0.0	0.0	0.0	0.0
Cash Flow from Fin.	0.0	17.3	24.1	2.6	66.3
UFCF	-10.3	-11.8	-13.6	-7.9	-10.9

PER SHARE DATA	FY25	FY26E	FY27E	FY28E	FY29E
Diluted SOI (m)	206.9	220.7	229.8	231.0	258.2
Normalised EPS (A$)	-0.05	-0.06	-0.07	-0.04	-0.05
DPS (A$)	0.00	0.00	0.00	0.00	0.00
Payout	0%	0%	0%	0%	0%
Franking	0%	0%	0%	0%	0%

RATIOS	FY25	FY26E	FY27E	FY28E	FY29E
Liquidity					
Current Ratio	3.2	6.1	10.7	7.0	26.0
Quick Ratio	2.3	5.4	10.0	6.5	25.6
Solvency					
Debt to Equity	0.00	0.00	0.00	0.00	0.00
Equity to Assets	0.80	0.87	0.92	0.88	0.96
Profitability					
ROA (Return on Assets)	-155.7%	-103.6%	-65.1%	-48.2%	-17.3%
ROE (Return on Equity)	-195.7%	-119.5%	-71.1%	-55.0%	-18.0%
EBITDA Margin	0.0%	0.0%	0.0%	-113.5%	-155.5%
NPAT Margin	0.0%	0.0%	0.0%	-105.0%	-149.8%
Growth					
Revenue	0.0%	0.0%	0.0%	0%	0.0%
EBITDA	0.0%	13.1%	16%	-37%	37%
Underlying NPAT	0.0%	17.4%	14%	-40%	43%
EPS	0.0%	13.6%	8.4%	-42%	34%
Valuation					
P/E	-51.4	-45.3	-41.8	-71.7	-53.4
EV/Revenue	0	0	0.0	73.8	76.2
EV/EBITDA	-42.7	-45.8	-40.5	-65.0	-49.0
Dividend Yield	0.0%	0.0%	0.0%	0.0%	0.0%

1. The Unmet Need

EGFR-Mutant Non-Small Cell Lung Cancer: The Osimertinib Resistance Problem

Lung cancer kills ~1.8M people annually from ~2.2M new diagnoses globally; NSCLC accounts for ~85%. Activating EGFR mutations (predominantly exon 19 deletions and L858R substitutions) occur in ~15% of Western and 40–50% of East Asian cases, yielding 350,000–500,000 EGFRm NSCLC diagnoses globally each year. ~70% present with metastatic disease at diagnosis and five-year survival remains poor.

First-line standard of care is osimertinib (Tagrisso®, AstraZeneca), a third-generation irreversible EGFR TKI targeting both sensitising and T790M resistance mutations. FLAURA established 1L positioning: median PFS 18.9 vs 10.2 months versus first-generation TKIs, median OS 38.6 vs 31.8 months. The osimertinib + platinum-doublet combination (FLAURA2) extends median PFS to ~25.5 months but adds substantial toxicity. Osimertinib generates >US$7B annually for AstraZeneca, one of the highest-grossing oncology drugs globally.

The fundamental limitation of osimertinib is acquired resistance. Virtually all patients progress, with median time to resistance ~18–20 months on monotherapy. Resistance mechanisms are heterogeneous: on-target EGFR mutations (most commonly C797S, which disrupts osimertinib's covalent binding site), MET and HER2 amplification, histological transformation to small-cell lung cancer, and bypass signalling (PI3K/AKT/mTOR, RAS-MAPK). MYC amplification/overexpression are increasingly recognised as resistance mechanisms and markers of aggressive biology post-progression.

After osimertinib progression, no approved targeted therapy exists. Guidelines recommend platinum-based chemotherapy (median PFS ~5–6 months). Amivantamab-lazertinib (J&J) has shown ~36% response and median PFS ~6 months in the post-osi setting (CHRYSALIS-2, MARIPOSA-2), but with substantial toxicity (infusion reactions, VTE, skin/nail effects). No current approach targets resistance prevention - extending first-line osimertinib's benefit window before resistance emerges. This is HARNESS-1's clinical gap.

The rationale for combining G4/MYC inhibition with osimertinib is mechanistic: MYC amplification/upregulation drives both intrinsic and acquired osimertinib resistance, and MYC suppression via G4 stabilisation may remove a key bypass pathway. The RAMOSE trial of osimertinib + ramucirumab (median PFS 15.6 → 24.8 months) provides clinical precedent for combination-driven resistance delay in EGFRm NSCLC. Whether G4/MYC suppression achieves comparable or superior effect is HARNESS-1's central question.

Anthracycline Cardiotoxicity: A Decades-Old Problem with No Pharmacological Solution

Anthracyclines (principally doxorubicin and epirubicin) remain among the most widely prescribed chemotherapy agents globally - foundational in first-line regimens for breast cancer (AC-T, dose-dense AC), sarcoma, lymphoma, and paediatric cancers. Doxorubicin alone is administered to 1–2 million patients annually worldwide.

Doxorubicin's utility is constrained by cumulative dose-dependent cardiotoxicity: clinical heart failure incidence rises sharply above 400–550 mg/m² cumulative (~5% at 400, 26% at 550, 48% at 700 mg/m²). Subclinical damage - detectable by troponin elevation, echocardiography, or cardiac MRI - begins at lower doses and can manifest as heart failure years after treatment. Oncologists therefore cap cumulative doxorubicin at 450–500 mg/m², frequently stopping effective therapy before maximum anticancer benefit. Cardiotoxicity is irreversible in most cases.

The only approved cardioprotective agent is dexrazoxane (Zinecard®/Cardioxane®), an iron chelator reducing ROS generation. Randomised trials show reduced heart failure and troponin elevation, but use is restricted by regulatory limits (cumulative doses >300 mg/m²), concerns about potential interference with anticancer efficacy, and an early (subsequently disputed) safety signal regarding secondary malignancies. In practice it is used in a minority of doxorubicin-treated patients; no other pharmacological cardioprotective agent has been approved.

An agent that protects the heart while preserving or enhancing anticancer activity would expand doxorubicin's therapeutic window across multiple solid tumour and haematological indications. Bisantrene's lower historical cardiotoxicity versus doxorubicin (4% vs 23% serious cardiac events, Phase 3 head-to-head) suggests a differentiated position; CPACS tests whether co-administration actively protects cardiac function.

Relapsed or Refractory Acute Myeloid Leukaemia

AML is the most common adult acute leukaemia: ~20,000 US diagnoses annually and 150,000–170,000 globally, predominantly in older adults (median diagnosis age ~68). Most patients are not candidates for intensive induction chemotherapy. Even among those reaching complete remission with frontline therapy (typically '7+3' cytarabine + anthracycline, or venetoclax + azacitidine for unfit patients), relapse rates are high: ~40–50% under 60 and 70–80% over 60.

The R/R AML landscape has evolved with targeted approvals: venetoclax (BCL-2), gilteritinib (FLT3), ivosidenib/enasidenib (IDH1/IDH2), and gemtuzumab ozogamicin (CD33 ADC). Despite these, outcomes remain poor: median OS after first relapse ~6 months and five-year survival <10%. Patients failing two or more lines have median survival of weeks to a few months. A critical need persists for additional salvage options, particularly for patients who have failed or are ineligible for targeted therapies.

Bisantrene was approved in France for R/R AML in 1988 based on a 46% monotherapy response rate averaged across ten studies (146 patients). It was never commercially launched due to formulation challenges - poor aqueous solubility requiring central venous access and causing phlebitis. RC220 overcomes these limitations, enabling peripheral IV administration. Racura's Phase 3 AML strategy leverages the historical database via a bridging study aligned with FDA's Project Optimus framework.

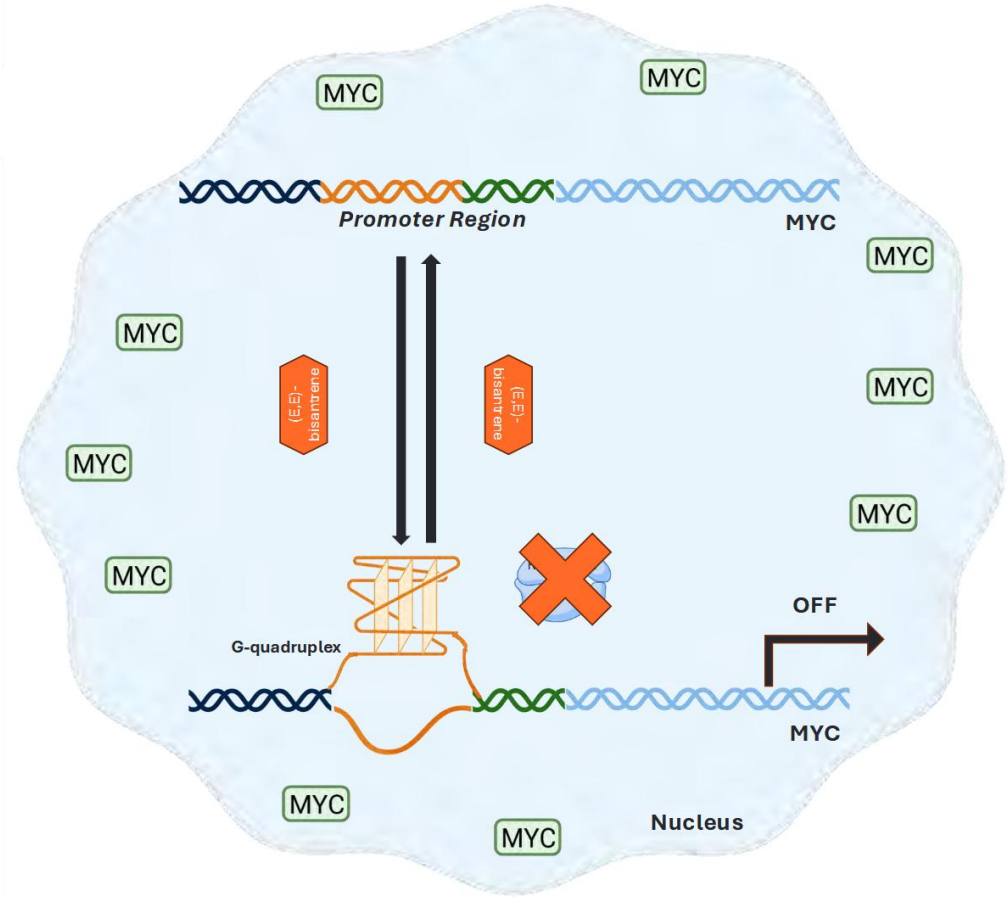
2. The Solution

RC220: Mechanism of Action

RC220 is the purified (E,E) photoisomer of bisantrene, a small molecule originally developed by Lederle Laboratories in the 1970s and studied in 40+ clinical trials through the 1980s–early 1990s. Bisantrene was historically assumed to act like doxorubicin (direct DNA damage and ROS generation) - an incorrect classification that contributed to suboptimal clinical development.

In October 2025, Racura disclosed that RC220 acts primarily by binding specialised DNA/RNA structures called G-quadruplexes (G4s) - three-dimensional knots forming in guanine-rich genome regions, particularly the promoter 'control regions' of cancer-driving genes (MYC, KRAS, BCL-2) and at the telomeric caps of chromosomes. G4s act as molecular switches: locking them in place silences the regulated genes; destabilising them allows activation.

Figure 3: Mechanism of action – (E,E)-bisantrene silences MYC by stabilising the promoter G-quadruplex. Two bisantrene molecules bind the G4 structure in the MYC promoter region (consistent with the 2:1 stoichiometry confirmed by NMR titration; see Section 3, Figure 9), locking it in the folded state. The folded G4 blocks transcription machinery from reading the gene, switching MYC production OFF. Source: Racura Oncology R&D Symposium, 24 March 2026.



The discovery came from comparing RC220's kill pattern across 102 cancer cell lines against 195 known drugs: RC220 matched established G4-binders, not doxorubicin or other conventional chemotherapies. Lab studies confirmed RC220 locks the MYC-promoter G4, rapidly suppressing MYC across multiple cancer types. Beyond MYC, RC220 hits several cancer-relevant targets simultaneously: telomerase inhibition (limiting cancer-cell replicative immortality), indirect topoisomerase II inhibition, and increased m6A RNA modification (a cancer-suppressive epigenetic mark). **Multi-target activity makes resistance harder to develop than against single-target drugs.**

MYC suppression is the thesis centrepiece. MYC is overactive in 70%+ of human cancers and drives the core processes by which cancer cells grow, divide, and resist treatment. Decades of pharmaceutical effort have failed to drug MYC directly - the protein has no well-defined binding pocket, making it among oncology's most notorious 'undruggable' targets. Indirect approaches (BET inhibitors targeting MYC gene activity epigenetically; the Omomyc mini-protein blocking MYC at the protein level) have not yet delivered approvals, constrained by toxicity or limited efficacy.

RC220's approach is different: rather than targeting the MYC protein, it locks the upstream G4 switch in the gene's control region, suppressing MYC before it is produced. Two prior G4-targeting drugs - quarfloxin (discontinued due to binding issues) and CX-5461 (in early clinical trials for DNA-repair-deficient cancers) - have shown the concept works in humans, but no G4-targeting drug has been approved.

RC220: Proprietary Formulation

RC220 is Racura's proprietary (E,E)-bisantrene formulation, designed to overcome the solubility and delivery limitations that prevented the original drug from reaching commercial viability. The historical formulation required central venous access due to

phlebitis risk - a key driver of abandonment despite demonstrated clinical efficacy. RC220 enables both peripheral and central IV administration, dosed on Day 1 of a 21-day cycle in current protocols.

A critical discovery underpinning RC220 and Racura's IP strategy: bisantrene consists of three photoisomers - (E,E), (E,Z), (Z,Z) - that rapidly interconvert under visible light. The (E,E) isomer is the biologically active form, with the others exhibiting different and potentially inferior anticancer activities. This enabled Racura to file composition-of-matter patents over the purified (E,E) isomer - significantly stronger IP than the method-of-use patents available for the racemic mixture.

IP

In 2025, Racura filed three global patent applications covering composition, manufacture, formulation, and use of (E,E)-bisantrene. Composition-of-matter patents are the gold standard of pharmaceutical IP because they cover the drug substance itself regardless of indication or formulation. If granted, exclusivity runs to ~2045 - a 20-year runway from filing, materially stronger than the company's prior position (method-of-use patents plus orphan-drug regulatory exclusivity). The company also holds a worldwide licence from City of Hope covering bisantrene's role as an FTO inhibitor, with academic collaborations including Astex Pharmaceuticals, MD Anderson, Sheba Medical Centre, the University of Wollongong, and the University of Newcastle.

Differentiation from Prior Failed Approaches

RC220 is mechanistically distinct from prior G4-targeting candidates. Quarfloxin (CX-3543, Cylene) targeted ribosomal DNA G4 structures and was discontinued in Phase II due to excessive albumin binding. CX-5461/pidnarulex (CCTG/Senhwa) is a DNA-damage-inducing G4 stabiliser active in DNA-repair-deficient cancers but with a different binding mode and selectivity profile. APTO-253 (Aptose) was a MYC G4 stabiliser in Phase I for AML, discontinued due to formulation instability. RC220's differentiation: multi-target pharmacology (G4/MYC, telomerase, topoisomerase II, FTO/m6A), an extensive human safety database, and a now-resolved formulation.

Differentiation from Competitors

In cardioprotection, the only competitor is dexrazoxane (limitations described in Section 1). The more complex competitive picture is EGFRm NSCLC, where several approaches are approved or in late-stage development for 1L or post-osimertinib resistance. The table compares the key programmes against RC220 on mechanism, efficacy, toxicity, and status.

Figure 4: Competitive landscape in EGFRm NSCLC. Sources: Published trial data (MARIPOSA, HERTHENA-Lung02, FLAURA2), company announcements.

Agent	Amivantamab + Lazertinib (J&J)	Patritumab deruxtecan / HER3-DXd (Daiichi-Sankyo / Merck)	Osimertinib + Platinum/Pemetrexed (AstraZeneca)	RC220 / RC220 (Racura)
Mechanism	EGFR-MET bispecific Ab + 3rd-gen EGFR TKI	HER3-targeted antibody-drug conjugate (topo I payload)	3rd-gen EGFR TKI + cytotoxic chemotherapy	G4-DNA/RNA stabiliser; MYC suppression, telomerase/topo II inhibition, FTO/m6A modulation
Key Efficacy Data	1L: mPFS 23.7 mo vs 16.6 mo osi (MARIPOSA); mOS: NR vs 36.7 mo (HR 0.75). Post-osi: mPFS 6.3 mo vs 4.2 mo chemo (MARIPOSA-2)	Post-osi: mPFS 5.8 mo vs 5.4 mo chemo (HERTHENA-Lung02); mOS 16.0 mo vs 15.9 mo (HR 0.98, NS)	1L: mPFS 25.5 mo vs 16.7 mo osi mono (FLAURA2); mOS 47.5 mo vs 37.6 mo (mono)	Preclinical synergy with osi (Emory); HARNESS-1 Phase 1a/b enrolling Q2 2026. No human efficacy data yet
Key Toxicities	Gr≥3 AEs 80% (vs 52% osi); rash 17%, paronychia 12%, PE 9%; requires prophylactic anticoagulation	Gr≥3 TRAEs 58%; thrombocytopenia 30%; ILD 5.2%; 4 TRAE-linked deaths	Substantially increased haem toxicity (neutropenia, anaemia) and GI toxicity vs osi mono	Historical: low cardiotoxicity (4% vs 23% doxorubicin); no DLTs at 40 mg/m² in CPACS (n=3)
Status & Limitations	FDA-approved 1L (Aug 2024). Substantial toxicity burden; does not prevent resistance, manages it	BLA withdrawn (May 2025) after OS failed. Modest PFS gain, no OS benefit, safety concerns	FDA-approved 1L (2024). Extends PFS via cytotoxic addition; does not address targeted resistance mechanisms	Phase 1. Targets MYC-driven resistance - a different pathway to all competitors above

Note: mPFS = median progression-free survival; mOS = median overall survival; osi = osimertinib; PE = pulmonary embolism; ILD = interstitial lung disease; DLT = dose-limiting toxicity; NS = not statistically significant.

Three observations emerge. First, each approved/advanced competitor addresses a different layer of the resistance problem. Amivantamab-lazertinib blocks EGFR and MET signalling (MET amplification, EGFR-dependent resistance) but not MYC-driven transcriptional bypass. Osimertinib + chemotherapy adds non-specific cytotoxic killing to delay progression - at substantial toxicity cost. Patritumab deruxtecan delivered a HER3 cytotoxic payload, but its Phase 3 OS failure (16.0 vs 15.9 months for chemotherapy) drove FDA withdrawal in May 2025, removing it as a near-term competitive threat.

Second, none of the current approaches targets the MYC pathway, despite MYC amplification/overexpression being increasingly implicated in both intrinsic and acquired EGFR-TKI resistance. If RC220's G4-mediated MYC suppression proves clinically active, it would address an orthogonal resistance pathway to amivantamab-lazertinib (EGFR/MET) or chemotherapy (cytotoxic) - creating potential for sequential or combinatorial use rather than direct competition. A patient could receive amivantamab-lazertinib first-line and add or switch to RC220 when MYC-driven resistance emerges; or RC220 could combine with osimertinib from the outset (HARNESS-1's design) to delay resistance.

Third, the toxicity profiles are non-overlapping. Amivantamab-lazertinib carries substantial skin, infusion, and VTE burden (grade 3+ AEs in 80% of MARIPOSA patients). Osimertinib + chemotherapy adds haematological toxicity. RC220's historical low cardiotoxicity and manageable haem profile suggest it could be layered onto existing regimens without compounding dominant toxicities. Speculative pending HARNESS-1 combination safety data, but the absence of overlapping toxicity signals is a favourable starting point.

3. Clinical Evidence

Historical Clinical Data: Bisantrene in Breast Cancer and AML

Bisantrene was studied in 40+ clinical trials during the 1980s–early 1990s. The most significant historical dataset is a large Phase 3 in advanced breast cancer versus doxorubicin (Cowan et al. 1991), which showed equivalent OS but markedly different cardiac safety - 4% serious cardiac events on bisantrene versus 23% on doxorubicin - alongside substantially lower alopecia. Seven breast cancer trials collectively confirmed single-agent activity.

In AML, bisantrene was studied across ten monotherapy trials (146 R/R patients), with an average 46% complete response rate - the basis for French approval in 1988 (never commercially launched due to formulation limitations). This historical database is unusual: a substantial body of human efficacy and safety data pre-dating the current programme.

Sheba Medical Centre Phase 2 Trials in R/R AML

Racura sponsored two investigator-led Phase 2 trials at the Chaim Sheba Medical Centre in Israel under Professor Arnon Nagler.

Phase 2 monotherapy trial (RC110, completed 2020): Ten heavily pretreated patients with R/R AML (median three prior lines of therapy) received bisantrene as a single agent. Four patients (40%) responded, meeting the trial's efficacy threshold. All four responding patients had extramedullary disease. No significant cardiotoxicity was observed.

Phase 1b/2 combination trial (Bis/Clo/Flu, results published 2025): This open-label trial (NCT04989335) evaluated bisantrene in combination with clofarabine (30 mg/m²) and fludarabine (10 mg/m²) in patients with very advanced R/R AML. Twenty-one patients were enrolled (median age 47 years, 55% female). Sixteen had relapsed post-allogeneic stem cell transplantation, and six had extramedullary disease. The median number of prior therapy lines was four (range 3–9), and the median bone marrow blast count at study entry was 50%. All patients were refractory to prior regimens.

Figure 5: Bis/Clo/Flu Phase 1b/2 trial design schematic. RP2D = recommended Phase 2 dose; CR = complete response. Source: BJH publication (Danylesko et al., 2025); ASH 2023 poster (Blood 2023;142(S1):4292).

Phase 1b: Dose Escalation	Phase 2: Simon Two-Stage	Post-Treatment
3+3 design. Cohort 1: Bis/Clo/Flu x 4 days. Cohort 2: Bis/Clo/Flu x 5 days. Established RP2D at 4-day schedule.	Stage 1: 9 patients treated. ≥1 CR observed → proceed to expansion. Stage 2: expanded to 15 evaluable patients. Efficacy threshold: ≥3 CRs required.	Responders assessed for bridge to allogeneic stem cell transplant (allo-SCT). Long-term follow-up for survival, relapse, and cardiac endpoints.

In the Phase 2 efficacy stage, six of 15 evaluable patients (40%) demonstrated a clinical response, comprising five complete responses and one partial response. Three of the six responders had active extramedullary disease. The complete response rate met the prespecified Simon two-stage efficacy threshold of at least three complete responses. Five of the six responders were bridged to a potentially curative stem cell transplant within one to three months of treatment. One patient remains alive and disease-free more than two years after receiving Bis/Clo/Flu therapy. The maximum tolerated duration of Bis/Clo/Flu was four days due to rapidly reversible hepatic transaminitis. The combination was otherwise well tolerated without clinically relevant cardiotoxicity or tumour lysis syndrome. The results were published in the British Journal of Haematology in August 2025.

The table below compares the key parameters and outcomes of the two Sheba trials:

Figure 6: Comparison of Sheba Medical Centre bisantrene trials. Source: Canaani et al., Eur J Haematol 2021; Danylesko et al., Br J Haematol 2025.

Parameter	Monotherapy (RC110, 2020)	Bis/Clo/Flu Combo (2025)
Design	Phase 2, single-arm, open-label	Phase 1b/2, open-label, Simon two-stage
Patients Enrolled	10	21 (15 evaluable in Ph2)
Median Age (range)	Not reported (adults)	47 years (19-69)
Median Prior Lines (range)	3	4 (3-9)
Post-Transplant Relapse	7/10 (70%)	16/21 (76%)
Median Blast % at Entry	Not reported	50%
Regimen	Bisantrene 250 mg/m² x 7 days	Bis 250 + Clo 30 + Flu 10 mg/m² x 4–5 days
Overall Response Rate	4/10 (40%)	6/15 (40%)
Complete Responses	3	5
Partial Responses	1	1
Bridge to Transplant	Not reported	5/6 responders (83%)
Cardiotoxicity	None observed	None observed
Key Non-Cardiac Toxicity	Thrombocytopenia, mucositis	Transaminitis (reversible), infections

Individual outcomes for the six Bis/Clo/Flu responders are below. At this sample size, depth and durability of response - and the ability to bridge to transplant - matter as much as aggregate response rates.

Figure 7: Individual patient outcomes for Bis/Clo/Flu responders (Phase 2 expansion, n=6 responders of 15 evaluable). Source: Danylesko et al., Br J Haematol 2025; Racura Oncology ASX announcements.

Pt	Response	EMD at Entry	Bridge to SCT	Outcome	Status at Publication
1	CR	Yes	Yes, within 1-3 mths	GVHD, died Day 45 post-SCT	Deceased
2	CR	No	Yes, within 1-3 mths	Relapsed 4 mo post-SCT	Deceased
3	CR	Yes	Yes, within 1-3 mths	Infection, died Day 610	Deceased
4	CR	No	Yes, within 1-3 mths	Disease-free >2 years	Alive, disease-free
5	CR	Yes	Yes, within 1-3 mths	Not reported in detail	Not reported
6	PR	No	No	Not reported in detail	Not reported

Note: CR = complete response; PR = partial response; EMD = extramedullary disease; SCT = stem cell transplant; GVHD = graft-versus-host disease.

The Sheba response rates are benchmarked against established R/R AML salvage regimens below. Cross-trial comparisons are indicative only - populations, eligibility criteria, and endpoints differ across trials.

Figure 8: Contextual comparison of bisantrene response rates versus established R/R AML salvage regimens. Source: Published trial data; company announcements.

Regimen	ORR	CR Rate	Median Prior Lines	Notes
Bis/Clo/Flu (Sheba)	40% (6/15)	33% (5/15)	4 (range 3-9)	76% post-allo-SCT relapse; 0% cardiotoxicity
Bisantrene mono (Sheba)	40% (4/10)	30% (3/10)	3	All responders had EMD; 0% cardiotoxicity
FLAG-Ida	~40-55%	~30-45%	1-2	Standard R/R AML salvage; substantial haem toxicity
Clofarabine mono	~30%	~15-20%	1-2	Approved for paediatric R/R ALL; cardiac risk up to 27%
Gilteritinib (FLT3+)	21% CR	21%	1-2	FLT3-mutant only (~30% of AML); mOS 9.3 mo
Venetoclax + HMA	~55-65% (1L unfit)	~35-45%	0 (frontline)	Now standard 1L for unfit pts; lower ORR in R/R

Note: cross-trial comparisons are indicative only due to differing patient populations and study designs. ORR = overall response rate; CR = complete response; EMD = extramedullary disease; HMA = hypomethylating agent.

The Sheba data are notable against this landscape. The 40% Bis/Clo/Flu response was achieved in a substantially more advanced population than most comparator trials - median four prior lines (versus one to two for FLAG-Ida and clofarabine), 76% post-transplant relapse, 50% median bone marrow blast count at entry - patients typically receiving palliative care. Comparable salvage regimens in less heavily pretreated populations achieve 30–55%, so bisantrene's activity in this advanced population is clinically meaningful.

Cardiac safety is central to the CPACS thesis; comparator AE rates are below.

Figure 9: Cardiac safety comparison across bisantrene datasets and relevant comparators. Source: Phase 3 breast cancer trial (historical); Danylesko et al., Br J Haematol 2025; Canaani et al., Eur J Haematol 2021; published literature.

Agent / Regimen	Cardiac AE Rate	Context
Bisantrene monotherapy (Ph3 breast)	4% serious cardiac events	Large Phase 3 trial vs doxorubicin; cumulative doses >5,440 mg/m² tolerated
Bis/Clo/Flu combination (Sheba)	0% cardiotoxicity	21 patients, R/R AML; despite clofarabine's known cardiac risk
Bisantrene monotherapy (Sheba)	0% cardiotoxicity	10 patients, R/R AML
Doxorubicin	23% serious cardiac events	Phase 3 comparator; dose-limited at 450–500 mg/m² cumulative
Clofarabine-containing regimens	Up to 27% LV systolic dysfunction	Published literature; significant cardiac monitoring required
Dexrazoxane (cardioprotectant)	Reduces dox cardiac AEs ~50%	Only approved cardioprotectant; limited to cumulative dox >300 mg/m²

Note: LV = left ventricular; AE = adverse event; dox = doxorubicin.

The absence of cardiotoxicity in 31 patients across both Sheba trials – including when combined with clofarabine, an agent with known cardiac risk of up to 27% LV dysfunction – is striking. While the sample size is too small for definitive conclusions, the consistency of the cardiac safety signal across monotherapy, combination therapy, and the historical Phase 3 dataset reinforces the mechanistic thesis that bisantrene is fundamentally less cardiotoxic than standard anthracyclines. Whether this extends to active cardioprotection when co-administered with doxorubicin is the question the CPACS trial is designed to answer.

RAC-010 / CPACS: Phase 1a/b in Solid Tumours (RC220 + Doxorubicin)

The Cardioprotection and Anticancer Synergy (CPACS) trial is a Phase 1a/b, open-label, multi-centre study evaluating RC220 as a single agent and in combination with doxorubicin in patients with advanced solid tumours where doxorubicin use is indicated.

Trial Design
Stage 1 enrols up to 33 patients across ascending dose cohorts to determine safety, tolerability, PK, and MTD of the combination. Stage 2 adds ~20 patients at the recommended dose for further safety and preliminary efficacy. Biomarkers include cardiac troponin (cardioprotection), m6A RNA levels, and other G4/MYC-pathway markers. Sites: Southside Cancer Care Centre (Australia), Queen Mary Hospital (Hong Kong), and South Korean sites (MFDS IND obtained, activation in progress).

Endpoint Strategy: Why VO_2peak, Not Just LVEF
The endpoint strategy draws on research by Assoc. Prof. Erin Howden (UNSW), a specialist in exercise physiology and cancer-related cardiovascular dysfunction, who presented the rationale at Racura's March 2026 R&D Symposium. Howden's framework reframes anthracycline cardiotoxicity as a multi-system problem that conventional cardiac monitoring fails to capture..

The standard measure of anthracycline cardiotoxicity is left ventricular ejection fraction (LVEF) by echocardiography - a blunt instrument that captures only resting cardiac pumping efficiency. Anthracycline damage is not confined to the cardiac pump. Howden's research (prospective cohort of 206 cancer patients enrolled pre-treatment) shows anthracycline chemotherapy accelerates cardiometabolic ageing across the entire oxygen-utilisation cascade - pulmonary gas exchange, cardiac output, peripheral oxygen extraction - but LVEF measures only the pump component.

In breast cancer survivors >1 year post-anthracycline treatment, Howden's group showed VO_2peak (peak oxygen consumption at maximal exercise) remains significantly impaired versus age-matched controls - only partially explained by reduced cardiac function, with peripheral and vascular deconditioning contributing independently. Correcting a single cascade parameter (e.g. cardiac output alone) yields limited functional recovery.

VO_2peak is a strong independent predictor of long-term outcomes: each 1-MET increase in cardiorespiratory fitness reduces all-cause mortality by ~26%, cardiovascular mortality by 14%, and cancer-specific mortality by 25% (Groarke et al., EHJ - Quality of Care and Clinical Outcomes, 2020).

Consequently, CPACS Phase 1b uses VO_2peak as primary functional efficacy endpoint alongside cardiac-specific structural and biomarker measures (GLS by echocardiography, cardiac MRI, troponin). Because VO_2peak integrates damage across the cardiopulmonary system rather than only the pump, it is a more sensitive marker of anthracycline-induced functional decline than LVEF - detecting a cardioprotective signal with fewer patients and shorter follow-up than LVEF, which often shows measurable decline only once damage is advanced and largely irreversible.

Progress to Date
First patient dosed (RC220 single agent) at Southside Cancer Care Centre, Australia, in May 2025; first combination dose (RC220 + doxorubicin) in June 2025; third patient at Queen Mary Hospital, Hong Kong, in March 2026, completing the first 40 mg/m² cohort. No DLTs, phlebitis, or other treatment-related AEs reported - significant given phlebitis was the historical formulation's primary limitation.

On 15 May 2026, the trial's independent Safety Review Committee (SRC) reviewed all safety and PK data from the three Cohort 1 patients and cleared escalation to Cohort 2

at 80 mg/m² RC220 – a dose-doubling. The SRC identified no treatment-related safety concerns; all three patients remain alive despite advanced metastatic disease at enrolment. Screening for Cohort 2 patients is underway across Australia, Hong Kong, and South Korea. Cohort 2 also activates a protocol update introducing a doxorubicin lead-in monotherapy cycle, enabling assessment of RC220's cardioprotective potential via a blood-based molecular biomarker.

The clean first-dose safety profile is encouraging but very preliminary; the therapeutic dose may be substantially higher after dose escalation. The cardioprotection hypothesis will be tested primarily through cardiac biomarker endpoints (troponin, echo) in patients receiving doxorubicin. Racura has disclosed development of a proprietary cardioprotection blood test to enable earlier readouts. The open-label design will generate data progressively through 2026.

HARNESS-1: Phase 1a/b in EGFRm NSCLC

HARNESS-1 is a Phase 1a/b, open-label trial evaluating RC220 in combination with osimertinib in patients with EGFR-mutant NSCLC who are currently receiving osimertinib and show evidence of emerging resistance via circulating tumour DNA (ctDNA) monitoring.

Trial Design
Phase 1a uses ctDNA screening to identify EGFRm NSCLC patients on osimertinib showing molecular evidence of resistance (rising ctDNA, emerging resistance mutations). Eligible patients receive IV RC220 on Day 1 of a 21-day cycle alongside continued osimertinib. A Bayesian dose-escalation starts with three single-patient cohorts before expanding; total Phase 1a enrolment is 12–40 patients. Treatment continues until progression, unacceptable toxicity, or withdrawal. Once MTD is established, Phase 1b is a double-blind randomised expansion for efficacy.

Trial Rationale: Significant Synergy Between RC220 & Osimertinib
Preclinical data from the Emory collaboration (Prof. Shi-Yong Sun; see Preclinical Data Package) demonstrated synergy between RC220 and osimertinib in osimertinib-resistant EGFR-mutant cell and mouse models. The mechanistic rationale: MYC upregulation drives osimertinib resistance, and G4-mediated MYC suppression may delay or prevent emergence of resistant clones. ctDNA as a screening and enrolment tool is innovative but unvalidated in this specific context.

Status
Human Research Ethics Committee (HREC) approval has been received. Governance approval from Monash Health was granted in March 2026. Site initiation training was scheduled for 23 March 2026, with first patient recruitment expected shortly thereafter. Four additional sites are planned, making HARNESS-1 a multi-centre study.

HARNESS-1 is the portfolio's highest-risk, highest-reward programme. The hypothesis - that MYC suppression via a G4 stabiliser can delay acquired osimertinib resistance - has biological plausibility but no clinical precedent at this mechanism. ctDNA as a dynamic resistance biomarker could provide early, interpretable signals but introduces selection complexity. The 12–40-patient Phase 1a limits statistical power for efficacy, but the open-label rolling design generates progressive safety and PK data.

Regulatory Precedent: Accelerated Approval from Phase 1 Data in NSCLC
The FDA has recently granted accelerated approval to NSCLC therapies on Phase 1 and Phase 1/2 datasets with comparable or smaller patient numbers than HARNESS-1's.

Zongertinib (Hernexeos®, Boehringer Ingelheim) is the headline precedent: approved August 2025 for HER2-mutant NSCLC on 71 patients from the Phase 1a/b Beamion LUNG-1 trial (ORR 71%, mPFS 12 months). The path from Phase 1 data to US approval took two years (Fast Track Dec-23, BTD Aug-24, Priority Review Feb-25, approval Aug-25). The confirmatory Phase 3 (Beamion LUNG-2) ran in parallel but was not required for initial

approval. Two further NSCLC approvals reinforce the pattern: sevabertinib (Hyrnuo®, Nov-25) on a 70-patient Phase 1/2 dataset (71% ORR); sunvozertinib (Zegfrovy®, Jul-25) on a 315-patient Phase 1/2 programme (46% ORR) (Lee A, Drugs 2026;86(3):397-401).

This is highly relevant for HARNESS-1. If RC220 + osimertinib generates a meaningful Phase 1a/b efficacy signal, an accelerated pathway becomes plausible. The threshold is high: recent NSCLC accelerated approvals required ORR >45% in molecularly defined populations. Our base case models a conventional FY2033E approval and does not incorporate this optionality.

Preclinical Data Package

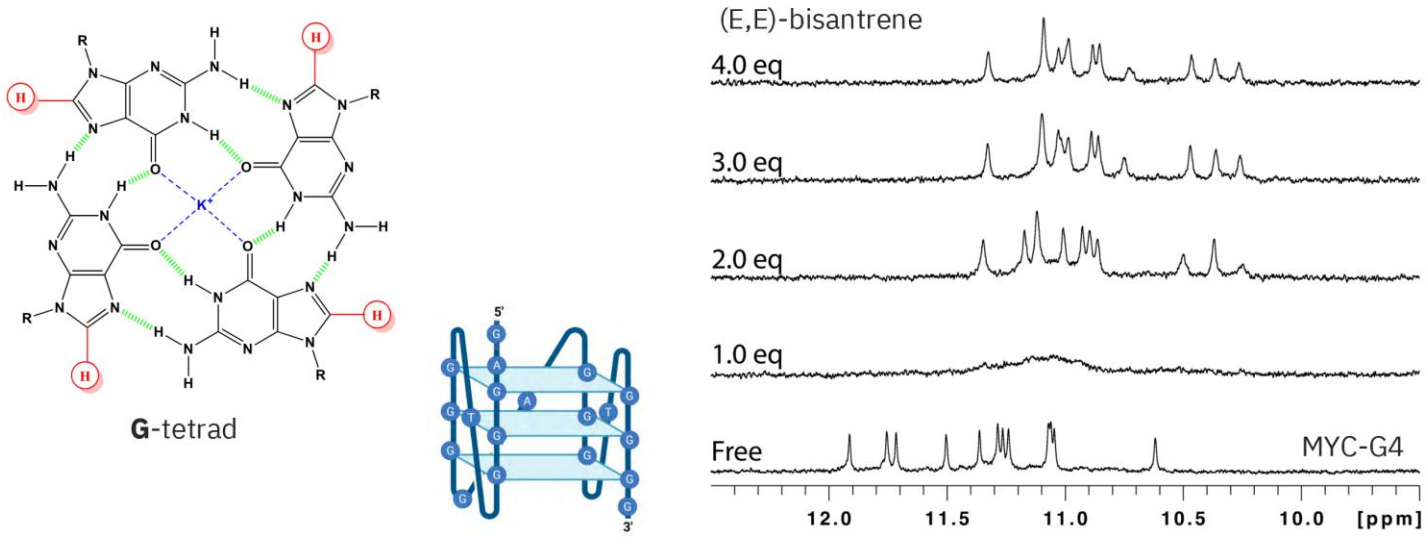
Racura's preclinical programme, conducted internally and through collaborations with multiple academic institutions, generated the mechanistic and pharmacological data that underpin all three clinical programmes. The key datasets are described below, organised by the clinical hypothesis they support.

G4 Mechanism Confirmation and MYC Suppression

The foundational finding is that RC220 acts via G4-DNA/RNA binding rather than as a conventional anthracycline. A cluster analysis compared RC220's cytotoxicity profile across 102 cancer cell lines against 195 known anticancer drugs (similar mechanisms produce similar cell-killing patterns). RC220 clustered with established G4-binding agents - most notably actinomycin D - and was clearly separated from the anthracycline cluster (epirubicin, doxorubicin, daunorubicin), providing the initial evidence that bisantrene's mechanism had been misclassified for decades.

Second, a biophysical package presented at AACR 2026 (Sahni et al., Abstract #5751) directly characterises how RC220 engages the c-MYC promoter G4. 1H-NMR titration indicated that (E,E)-bisantrene binds to c-MYC G4 with a 2:1 (E,E)-bisantrene: c-MYC G4 stoichiometry.

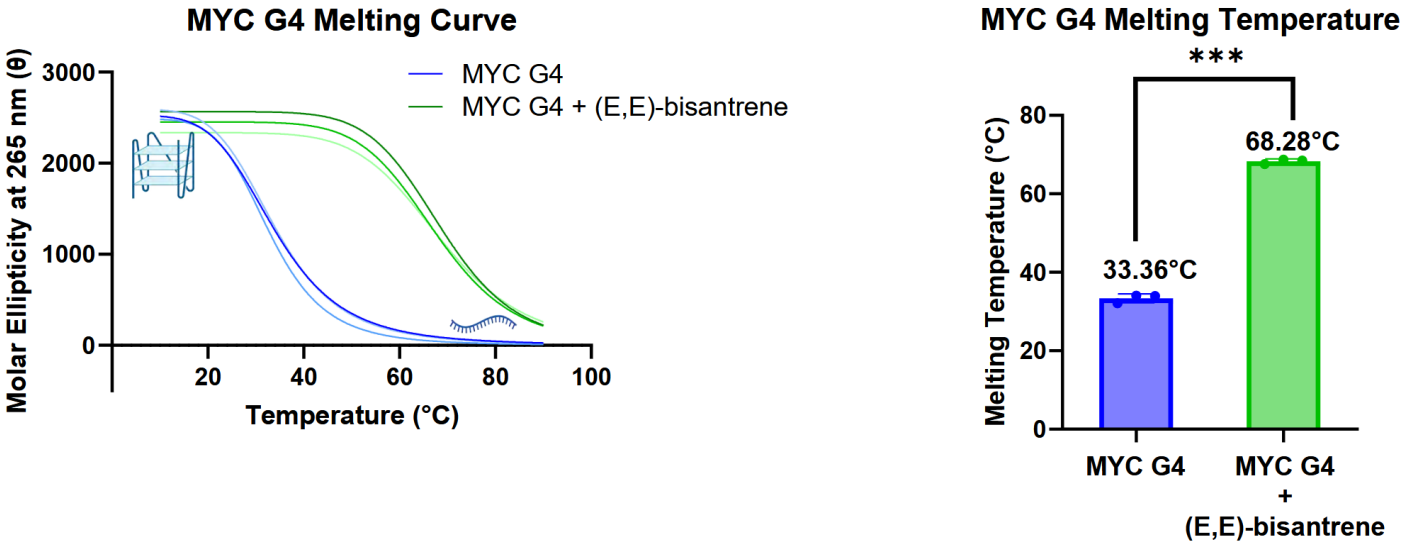
Figure 10: 1H-NMR titration confirms RC220 binds the MYC G-quadruplex at a defined 2:1 stoichiometry (two drug molecules per G4 structure). Source: Sahni et al., AACR 2026 (Abstract #5751); Racura Oncology R&D Symposium, 24 March 2026.



NMR titration works by adding a drug to a target molecule in stepwise increments and observing changes in the target's hydrogen atom signals. New peaks appearing in the 10–12 ppm region – where G-tetrad hydrogen bonds resonate – indicate that the G4 structure is being perturbed by drug binding. The signal pattern stabilises completely at 2.0 equivalents and does not change with further drug addition, which is the fingerprint of a defined 2:1 stoichiometry: exactly two RC220 molecules per G4, one stacking on each face of the structure. This is direct atomic-level evidence of the binding mode, not an inference from a functional assay.

Melting-temperature data also provides strong biophysical evidence for G4 stabilisation.

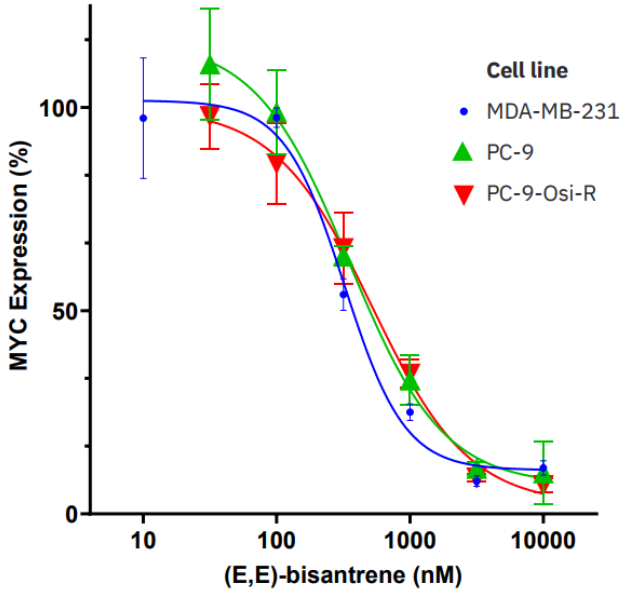
Figure 11: RC220 stabilises the MYC G-quadruplex, raising its melting temperature from 33.4°C to 68.3°C. Source: Racura Oncology R&D Symposium, 24 March 2026.



The melting temperature is the point at which a G4 structure unfolds. A drug that raises it is physically locking the G4 in the closed position - the higher the shift, the stronger the grip. A 35°C increase is a large stabilisation effect, indicating RC220 binds the MYC G4 with sufficient affinity to keep it folded at physiological temperature. A folded MYC G4 cannot be read by the cellular machinery that would otherwise transcribe the MYC gene, so stabilisation directly translates to MYC silencing.

Third, RC220 potently and dose-dependently suppresses MYC gene expression in MDA-MB-231 triple-negative breast cancer cells, with an EC50 of 322.5 ± 42.6 nM (Figure 12). The dose-response has been confirmed in additional cancer cell types. The nanomolar potency is pharmacologically relevant - it sits well within the concentration range achievable via IV dosing.

Figure 12: RC220 dose-dependent suppression of MYC expression in MDA-MB-231 breast cancer cells. Left: Graphical presentation; right: Table summary. Data derived from Racura Oncology ASX announcement (2 October 2025) and MOA webinar (October 2025).



RC220 Concentration (nM)	MYC Expression (% of control)	Interpretation
~100	~80-90%	Minimal suppression at low dose
~300 (EC50)	~50%	Half-maximal suppression; EC50 = 322.5 ± 42.6 nM
~1,000	~10–15%	Near-complete MYC suppression
~3,000–10,000	~5–10%	Maximal suppression plateau

Note: Table values are approximate; MDA-MB-231 cell line (blue line) is Breast cancer; PC-9 cell line (green line) is NSCLC; PC-9-Osi-R cell line (red line) is NSCLC.

Beyond MYC, G4-stabilisation produces multiple downstream anticancer effects: telomerase inhibition (via telomeric G4 stabilisation), indirect topoisomerase II inhibition, DNA hypomethylation (via DNMT1 inhibition), increased m6A RNA levels (mimicking FTO inhibition), and reduced expression of multiple G4-containing oncogene promoters (MYC, KRAS, EGFR, c-KIT, BCL2, VEGF, HIF1A, MYB, PDGFA). This breadth explains bisantrene's anomalous historical properties - immune-stimulatory

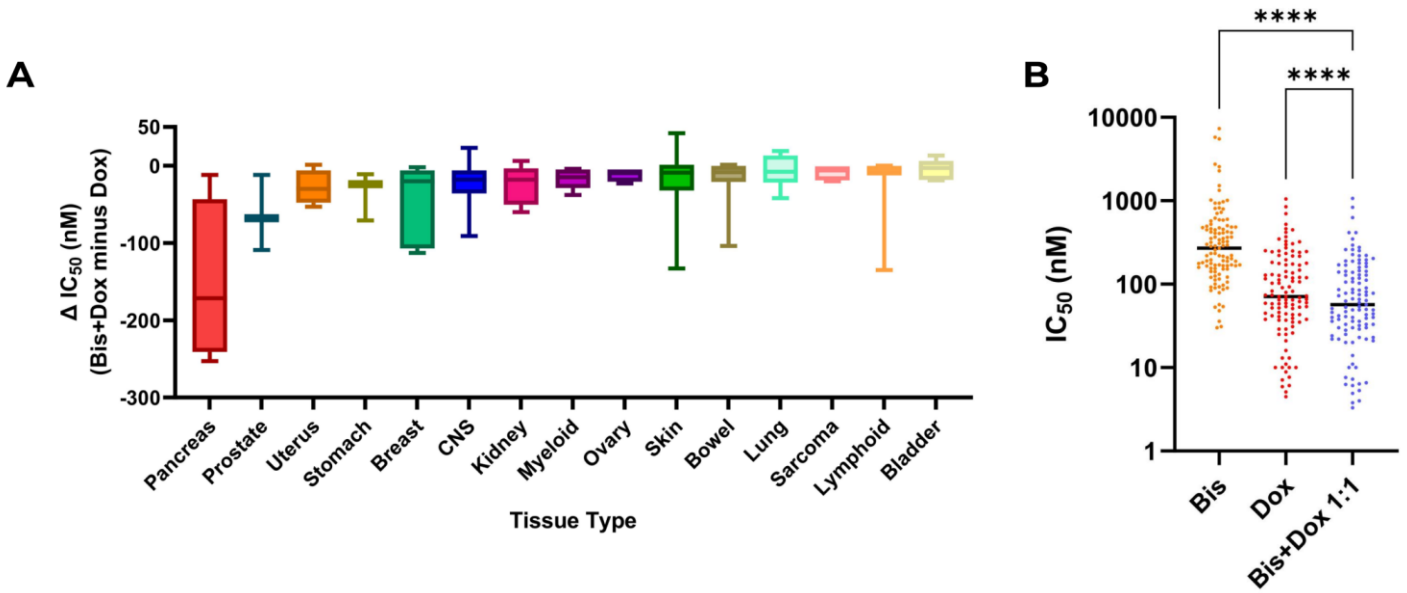
effects, kinase-inhibitor synergy, broad-spectrum activity - all coherent under the G4 framework.

Anticancer Combination with Doxorubicin

ESMO 2025 data evaluated RC220 + doxorubicin at a 1:1 molar ratio across 111 cancer cell lines from 22 tissue types (Figure 13). For every tissue type, combination IC50 was lower than doxorubicin alone - the combination was significantly more potent across the full panel ($p < 0.0001$).

Synergy analysis showed a predominantly additive interaction: bisantrene's contribution is independent of and additional to doxorubicin's, not contingent on a synergistic effect that might not translate clinically.

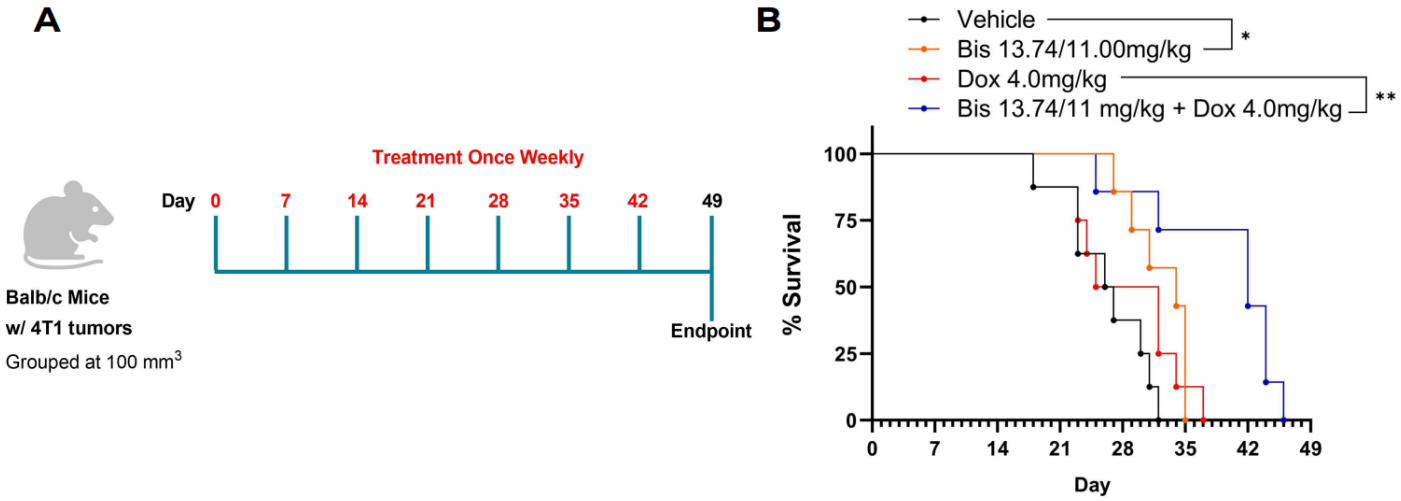
Figure 13: Adding bisantrene (Bis) to doxorubicin (Dox) enhances in vitro anticancer activity across 111 cancer cell lines from 22 tissue types. (A) Change in IC50 (Bis+Dox minus Dox alone) by tissue type; (B) IC50 values for Bis alone, Dox alone, and the 1:1 molar combination across the full panel, showing significantly lower IC50 for the combination ($p < 0.0001$). Source: ESMO 2025, poster 1010eP (Tillett et al.).



Panel A: change in IC50 (Bis+Dox minus Dox alone) by tissue type - every tissue type sits below zero, meaning the combination is more potent than Dox alone in every cancer type tested. Pancreas and prostate show the largest improvements (IC50 drops of 100–200+ nM); other tissue types show gains of 10–50 nM. **Panel B:** IC50 distributions across all 111 cell lines for Bis alone (median ~200 nM), Dox alone (median ~60–80 nM), and the combination - which shifts the entire distribution downward and achieves a significantly lower median IC50 than Dox alone ($p < 0.0001$).

In vivo confirmation came from a 4T1 syngeneic breast cancer mouse model (Figure 14). The combination arm showed enhanced tumour growth inhibition and improved survival versus either agent alone. The syngeneic model preserves immune competence - an advantage over xenografts - allowing any G4-mediated immune-stimulatory effects to contribute to the observed activity.

Figure 14: Mice model combining Bis with Dox (1:1 mol) enhances in vivo anticancer efficacy. BALB/c mice with 4T1 breast tumours were treated weekly with vehicle, Bis, Dox, or Bis+Dox (4:1 molar ratio). The combination significantly improved survival versus either agent alone (*p < 0.01). Source: ESMO 2025, poster 1010eP (Tillett et al.).



Panel A: experimental design - BALB/c mice with 4T1 tumours (grouped at 100 mm³) received weekly IV vehicle, bisantrene (13.74/11 mg/kg), doxorubicin (4.0 mg/kg), or the combination from Day 0–42, with survival assessed to Day 49. **Panel B:** Kaplan-Meier survival curves. Vehicle mice were all deceased by ~Day 28; both monotherapy arms reached 0% survival by Day 35–42. The combination arm showed a statistically significant survival advantage over both single agents, with animals alive at the Day 49 endpoint.

Cardioprotection

The ESMO 2025 poster presented cardioprotection data across three in vitro models, two in vivo models, and the underlying mechanism (Figure 15).

Bisantrene reduces doxorubicin-induced double-strand DNA breaks (DSBs) in cardiomyocytes (measured by γ H2AX staining) - the same mechanism through which dexrazoxane, the only FDA-approved cardioprotective agent, operates. Bisantrene achieved DSB reduction at lower concentrations than dexrazoxane, suggesting comparable or superior cardioprotective potency. In differentiated RA-H9c2 cardiomyocytes, bisantrene pre-treatment (2 hours) followed by doxorubicin exposure (4 hours) produced statistically significant reductions in γ H2AX versus doxorubicin alone ($p < 0.0001$), with bisantrene's effect evident at concentrations where dexrazoxane was inactive.

Figure 15: Summary of preclinical cardioprotection evidence. Source: ESMO 2025 poster 1010eP (Tillett et al.).

Model System	Endpoint	Result
Primary human cardiomyocytes (PHCs)	Viability after Dox + Bis co-treatment	Bis protected PHCs from Dox-induced cytotoxicity in a concentration-dependent manner
Neonatal rat ventricular myocytes (NRVMs)	LDH release (cytotoxicity marker)	Bis reduced Dox-induced LDH release
iPSC-derived cardiomyocytes (iPSC-CMs)	Beat rate (CiPA assay)	Bis mitigated Dox-induced increases in beat rate
Mouse model (Dox cardiotoxicity)	Left ventricular ejection fraction (LVEF) by echo	Bis+Dox significantly improved LVEF vs Dox alone
Dog model (Dox cardiotoxicity)	LVEF by echocardiography	Bis+Dox significantly improved LVEF vs Dox alone
RA-H9c2 cells (mechanism)	γH2AX (DSB marker) by flow cytometry	Bis reduced Dox-induced DSBs at lower concentrations than dexrazoxane

Note: PHC = primary human cardiomyocyte; NRVM = neonatal rat ventricular myocyte; iPSC-CM = induced pluripotent stem cell-derived cardiomyocyte; CiPA = Comprehensive In Vitro Proarrhythmia Assay; LVEF = left ventricular ejection fraction; DSB = double-strand DNA break.

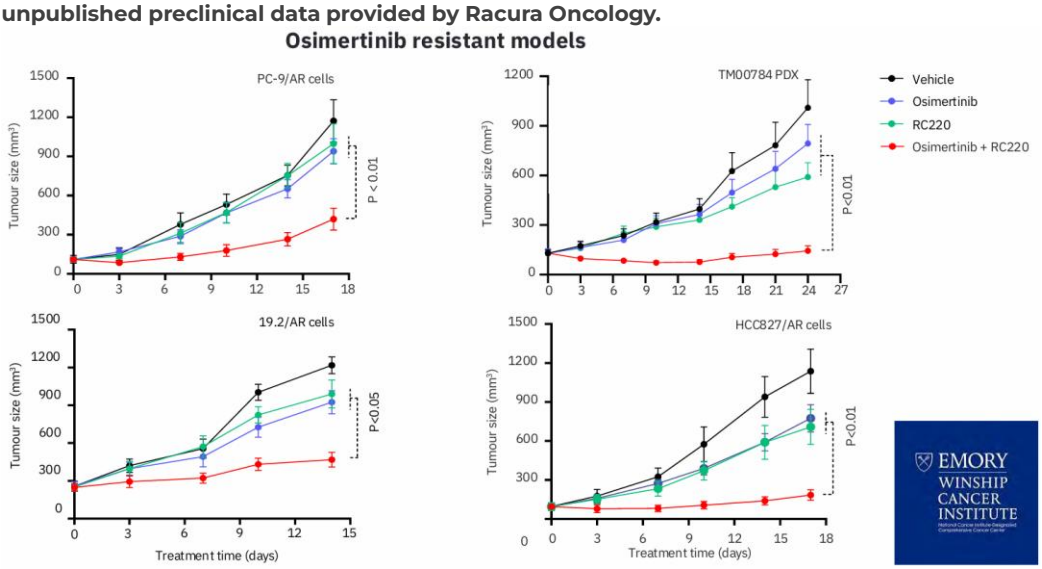
Bisantrene protects the heart via the same molecular pathway as dexrazoxane (Top2 β -mediated DSB reduction), but unlike dexrazoxane, simultaneously provides additive anticancer activity via a non-overlapping mechanism (G4/MYC suppression). This dual-

activity profile is the core CPACS thesis and has no precedent in approved oncology drugs.

Osimertinib Synergy (Emory University Collaboration)

Preclinical data from a collaboration with Emory University (Prof. Shi-Yong Sun, lung cancer resistance) demonstrated significant synergy between RC220 and osimertinib in EGFR-mutant cell line and mouse models engineered to recapitulate the resistance mechanisms observed in patients progressing on third-generation EGFR TKIs. The combination produced greater tumour growth inhibition than either agent alone in resistant models (Figure 16), supporting the hypothesis that MYC suppression via G4 stabilisation can overcome or delay resistance.

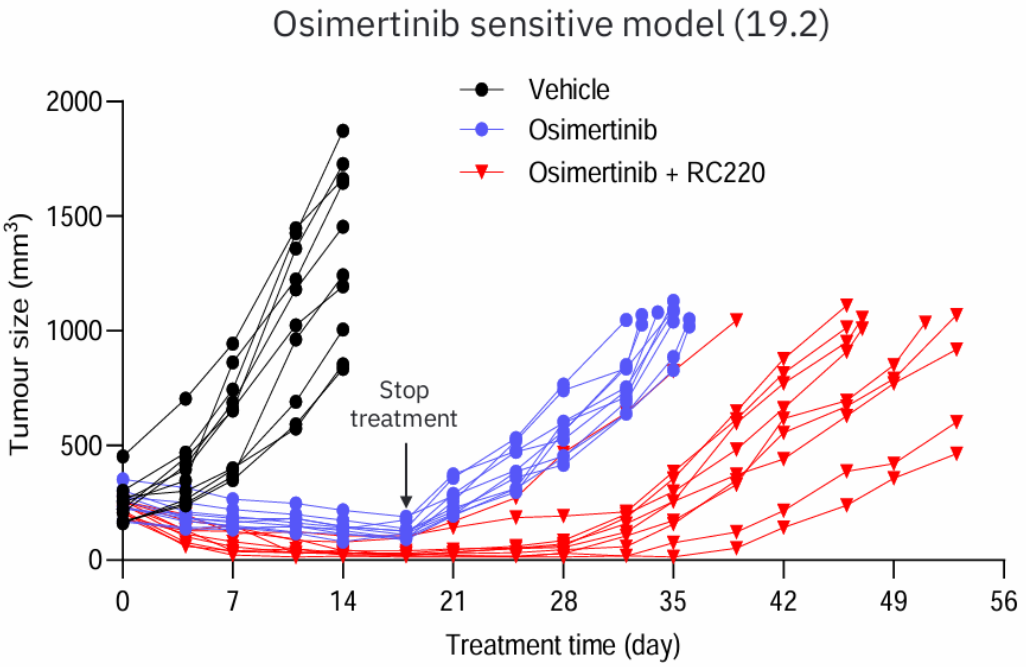
Figure 16: Tumour growth curves across four osimertinib-resistant EGFR-mutant models. Source: Emory University collaboration (Prof. Shi-Yong Sun, Winship Cancer Institute), unpublished preclinical data provided by Racura Oncology.



Four osimertinib-resistant EGFR-mutant models: PC-9/AR (upper left), TM00784 PDX (upper right), 19.2/AR (lower left), HCC827/AR (lower right). In all four, neither osimertinib nor RC220 as single agents meaningfully inhibited tumour growth (consistent with engineered resistance). The combination produced statistically significant inhibition ($p < 0.05$ in 19.2/AR; $p < 0.01$ in PC-9/AR, TM00784, HCC827/AR). Consistency across three cell-line-derived models and one PDX strengthens confidence that the synergy is not an artefact of any single resistance mechanism.

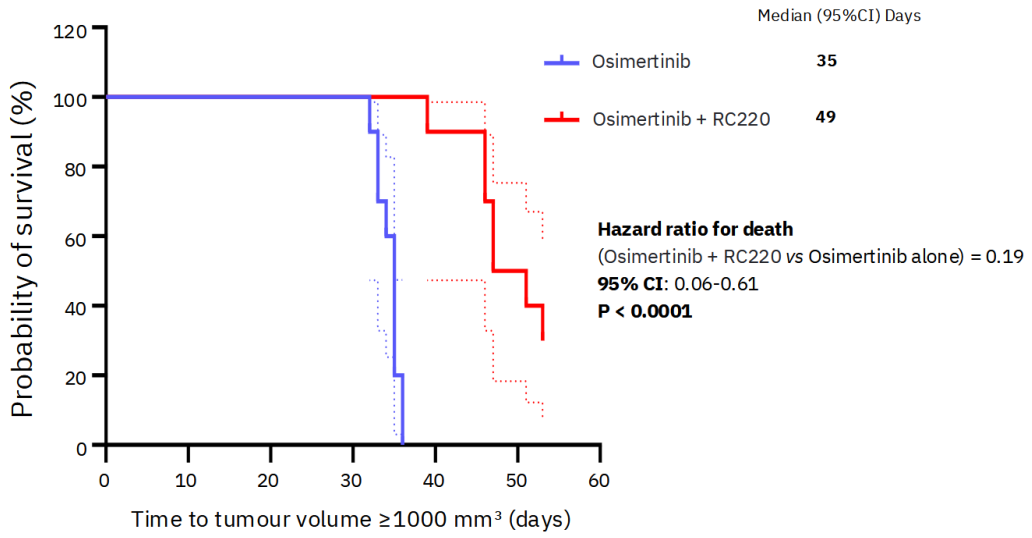
The Emory collaboration has also shown RC220 extends osimertinib durability in osimertinib-sensitive disease. In a sensitive xenograft model, adding RC220 did not deepen on-treatment response (both arms reached near-complete regression) but delayed post-withdrawal regrowth by ~2 weeks versus osimertinib monotherapy (Figure 14). This is mechanistically consistent: if MYC suppression delays outgrowth of resistant subclones, the effect should appear both as enhanced activity against established resistant tumours and as delayed resistance in initially sensitive ones. The two datasets are complementary support for HARNESS-1's rationale.

Figure 17: Tumour growth curves in an osimertinib-sensitive EGFR-mutant xenograft model. Source: Emory University collaboration (Prof. Shi-Yong Sun), unpublished preclinical data provided by Racura Oncology.



Treatment was administered for ~18 days then withdrawn (arrow). Vehicle tumours grew to ~1,500–2,000 mm³ by day 14. Both osimertinib arms induced deep regression during dosing, but the monotherapy arm regrew rapidly post-cessation (~1,000 mm³ by day 35). The osimertinib + RC220 combination delayed regrowth by ~2 weeks (comparable tumours not seen until day 49+).

Figure 18: Kaplan-Meier survival in the osimertinib-sensitive 19.2 EGFRm NSCLC xenograft. Source: Emory University collaboration (Prof. Shi-Yong Sun), unpublished preclinical data provided by Racura Oncology.



Kaplan-Meier survival analysis in the osimertinib-sensitive EGFRm NSCLC 19.2 xenograft model. Endpoint is time to tumour volume ≥1,000 mm³ following treatment withdrawal at Day 18. The osimertinib + RC220 combination arm achieved a median of 49 days versus 35 days for osimertinib monotherapy – a 40% extension in time to endpoint (HR for death 0.19; 95% CI 0.06–0.61; P<0.0001).

The Emory collaboration is ongoing and is expected to generate additional data characterising the specific resistance mechanisms (C797S, MET amplification, MYC amplification) against which the combination is most active.

4. Market Opportunity

EGFRm NSCLC: Osimertinib Combination and Resistance Market

The EGFRm NSCLC treatment market is large and growing. Osimertinib alone generates ~US$7B annually (AstraZeneca FY2025), with expanding adjuvant (ADAURA) and neoadjuvant indications driving further growth. Addressable population for an osimertinib resistance-delaying combination: 350,000–500,000 EGFRm NSCLC diagnoses globally per year; ~70% advanced disease at diagnosis (~245,000–350,000); virtually all advanced patients receive osimertinib or another third-generation TKI 1L, and virtually all eventually develop resistance.

A combination partner that meaningfully extends osimertinib PFS (e.g. ~19 to ~25+ months, comparable to RAMOSE) represents a substantial opportunity. NSCLC combination pricing precedents are US$100,000–200,000 per year. Even conservative 10–15% penetration of US+EU5 1L EGFRm patients implies peak revenue of US$500M–1.5B annually - highly speculative at Phase 1. Realistic near-term value lies in partnering: a resistance-delay dataset in EGFRm NSCLC would be highly attractive to large oncology companies (AstraZeneca, J&J, Roche, Daiichi-Sankyo) either as a bolt-on to existing EGFR franchises or as a standalone licence.

Pharmacoeconomics & Reimbursement

For reimbursement, RC220 would be co-administered with osimertinib (oral) or standard IV chemotherapy, positioning for US buy-and-bill under Medicare Part B. In HTA markets, the cost-effectiveness case rests on incremental PFS and associated QALY gains relative to drug cost. At US willingness-to-pay thresholds of US$100,000–150,000 per QALY, a combination extending median PFS by ≥6 months (~0.3–0.5 incremental QALYs) supports US$50,000–100,000 annual pricing while remaining cost-effective. In the UK (NICE) and Australia (PBAC), where thresholds are lower (£20,000–30,000 and A$45,000–75,000 respectively), pricing would adjust accordingly, but the PFS gains supporting the clinical case also support the pharmacoeconomic case at lower price points.

Cardioprotection in Anthracycline-Treated Solid Tumours

1–2 million patients annually receive anthracycline-containing regimens globally. The largest single indication is breast cancer (700,000–900,000 patients on AC/EC regimens annually across the US, EU, and China), followed by lymphoma, sarcoma, and paediatric cancers.

If RC220 demonstrates both cardioprotection and additive/synergistic anticancer activity with doxorubicin, it occupies a unique position: a combination partner that makes chemotherapy both safer and more effective. Pricing would likely follow supportive oncology precedents (e.g. G-CSF for neutropenia prevention, multibillion-dollar globally). At US$5,000–15,000 per course and 20–30% penetration of anthracycline-treated patients, the global revenue opportunity is US$1–5B - illustrative and contingent on unresolved clinical, regulatory, and competitive variables.

Reimbursement Pathway & Pricing Precedents

RC220 would be administered IV in hospital outpatient or infusion-centre settings alongside doxorubicin, positioning for US Medicare Part B buy-and-bill reimbursement. Dexrazoxane is reimbursed at ~US$95–120 per 250 mg vial (Medicare HCPCS J1190); typical courses (500–1,000 mg per administration across multiple cycles) cost US$2,000–6,000 per patient. The global dexrazoxane market is US$250–500M annually despite limited use (restricted to patients exceeding 300 mg/m^2 cumulative doxorubicin and used in a minority of eligible patients). An RC220 cardioprotection label approved from the first anthracycline cycle would address a substantially larger population. Broader

supportive oncology context: pegfilgrastim (G-CSF for neutropenia prevention) is priced at US$3,000–13,000 per injection; the biosimilar market alone was ~US$4.5B globally in 2025. RC220's value proposition is analogous but potentially broader - G-CSF prevents transient haematological toxicity, while cardioprotection addresses an irreversible long-term consequence.

Pharmacoeconomic Case: Downstream Cardiac Care Cost Offset
Heart failure is among the most expensive chronic conditions in developed healthcare systems. US average HF expenditure is ~US$29,000 per patient annually (5× the non-HF baseline; 65–80% hospitalisation-driven), with median single-admission cost US$13,400 plus US$6,300 post-discharge; total US HF spend is projected above US$70B by 2030. Anthracycline-treated cancer survivors face 5× the HF risk of non-anthracycline cohorts; up to 65% of paediatric doxorubicin patients show echocardiographic abnormalities. Illustrative cost-effectiveness: if RC220 cuts clinically significant cardiotoxicity from 5–10% (observed at 400–500 mg/m² cumulative doxorubicin) to 1–2% (bisantrene's 4% Phase 3 rate and 0% across 31 Sheba patients), NNT to prevent one HF case is 15–30. At US$5,000–15,000 per course, that costs US$75,000–450,000 versus an avoided US$150,000–300,000 lifetime HF management cost (5–10 years of US$29,000 annual expenditure, discounted) - cost-neutral to cost-saving on cardiac offset alone, before anticancer benefit. In QALY terms (0.1–0.2 utility loss/year × ~50% five-year HF mortality), preventing one HF case preserves 0.5–2.0 discounted QALYs, placing ICERs well within accepted thresholds. Cardioprotection's pharmacoeconomic profile differs structurally from most oncology drugs - benefits accrue over years through prevention of a chronic condition, not months of PFS/OS extension - and is increasingly relevant as payers and HTA bodies focus on cancer-survivorship cardiovascular burden.

Relapsed or Refractory AML

The R/R AML market is smaller but growing, with orphan drug designation providing regulatory incentives (accelerated pathways, market exclusivity). ~20,000 US AML diagnoses annually (20,800 in 2024, SEER/NCI); ~40–50% enter the R/R setting. Addressable population for a bisantrene-based salvage regimen is the R/R subset who have failed or are ineligible for targeted therapies - ~3,000–5,000 patients annually in the US alone. R/R AML pricing precedents are high: gilteritinib ~US$200,000/year; venetoclax-based regimens ~US$100,000–150,000/year.

Racura's AML Phase 3 strategy is a bridging study linking historical bisantrene data with RC220, aligned with FDA's Project Optimus dose-optimisation framework - potentially a more streamlined registration path than a fully de novo Phase 3. The programme is partner-dependent; Racura will seek a licensing or partnering arrangement to fund the pivotal trial. The historical 46% monotherapy response rate and 40% Sheba combination response rate provide a compelling partnering dataset.

R/R AML drugs are also reimbursed through Medicare Part B. At the pricing precedents above (US$100,000–200,000/year), 3,000–5,000 US patients annually implies a US$300M–1B opportunity in that market alone, with additional upside from EU and RoW. The pharmacoeconomic justification is primarily clinical: at this disease stage patients face survival measured in weeks, and any therapy achieving meaningful response and bridging to curative transplant generates substantial QALY gains versus best supportive care.

Platform Optionality

The G4/MYC mechanism is not indication-specific. MYC deregulation drives 70%+ of human cancers, and G4 structures sit in the promoters of multiple oncogenes beyond MYC. Clinical proof of concept in any one indication opens applicability to additional cancers (TNBC, pancreatic, melanoma, other MYC-driven malignancies). The FTO/m6A RNA epigenetic dimension adds further potential, as m6A dysregulation is increasingly recognised as a driver of cancer stem cell maintenance and treatment resistance.

For partnering and licensing, platform breadth is a significant asset. Precedent transactions for G4- or MYC-targeting clinical-stage programmes are limited, but oncology platform deals for differentiated mechanisms with multi-indication potential have commanded US$50–200M upfront and total deal values exceeding US$1B (e.g. Omomyc/Peptomyc, Relay Therapeutics' SHP2 programme). Racura's leverage in any partnering negotiation depends on the quality and scale of clinical data over the next 12–18 months.

5. Valuation

Bottom-Up Key Assumptions

Probability-of-Success (POS) of Clinical Programs

Each indication is valued using a risk-adjusted NPV (rNPV) framework: projected cash flows are multiplied by a probability-of-success (PoS) factor reflecting the likelihood of advancing from current stage to approval. Industry oncology base rates (BIO/Informa, 2011–2020): ~5–8% from Phase 1 entry, ~15–20% from Phase 2, ~35–45% from Phase 3. Programme-specific PoS deviates from benchmarks based on mechanism novelty, biomarker availability, regulatory pathway, and the strength of preclinical and early clinical data. The PoS assumptions applied are summarised below.

Figure 19: Probability-of-success assumptions by indication. Source: Analyst estimates; industry benchmarks from BIO/Informa Pharma Intelligence (2011-2020).

Programme	Current Stage	PoS Applied	Industry Benchmark (Stage → Approval)
EGFRm NSCLC (HARNESS-1)	Phase 1a/b (pre-enrolment)	8%	~5-8% (Phase 1 oncology)
Cardioprotection (CPACS)	Phase 1a/b (Cohort 1 complete)	10%	~5-8% (Phase 1 oncology)
R/R AML	Phase 3-ready (bridging strategy)	20%	~15-20% (Phase 2 oncology)

EGFRm NSCLC - 8% PoS, at the upper end of the Phase 1 oncology benchmark, reflecting three factors that modestly favour this programme. First, the drug substance has an extensive human safety database from 40+ historical bisantrene trials, substantially de-risking Phase 1 safety. Second, the combination partner (osimertinib) is well-characterised with a known toxicity profile, reducing the probability of unexpected combination toxicities that frequently derail early-phase oncology programmes. Third, Emory preclinical synergy data in osimertinib-resistant models provide a mechanistic rationale testable from the earliest cohorts via pharmacodynamic biomarkers (ctDNA, MYC expression) - interpretable go/no-go signals with relatively small patient numbers.

Cardioprotection (CPACS) – 10% PoS: three patients have been dosed at 40 mg/m² with no dose-limiting toxicities, and while preliminary, is consistent with expectations. The cardioprotection hypothesis is supported by a multi-model preclinical dataset (primary human cardiomyocytes, neonatal rat ventricular myocytes, iPSC-derived cardiomyocytes, mouse model, and dog model) with a defined molecular mechanism (Top2β-mediated double-strand break reduction) that is the same pathway by which dexrazoxane, the only approved cardioprotective agent, operates. The historical Phase 3 breast cancer trial provides a direct human comparator: 4% serious cardiac events for bisantrene versus 23% for doxorubicin at cumulative doses exceeding 5,440 mg/m², and 0% cardiotoxicity across 31 patients in the Sheba AML trials.

R/R AML - 20% PoS, based on three factors. First, bisantrene has demonstrated reproducible R/R AML activity across independent studies: 46% average monotherapy response across ten historical trials (146 patients), 40% in the Sheba monotherapy trial, and 40% (33% CR) in the Sheba Bis/Clo/Flu combination in heavily pretreated patients

(median 4 prior lines). Second, bisantrene was approved in France in 1988 for R/R AML - a historical regulatory precedent. Third, the bridging Phase 3 strategy offers a potentially streamlined registration pathway versus a fully de novo Phase 3. Caveats: Racura will seek a licensing partner to fund the pivotal trial (timing and terms not assured); FDA acceptance of the bridging design has not been confirmed through formal regulatory interaction; the Sheba trials' small sample sizes (10 and 21 patients) and open-label, single-arm designs limit the statistical confidence on the 40% response rate as a Phase 3 predictor.

Indication 1: EGFRm NSCLC

The NSCLC model assumes RC220 commercialises as an osimertinib combination partner in advanced EGFRm NSCLC, with 100% economics retained (out-licensing terms captured implicitly through the PoS adjustment). Key inputs are below.

Figure 20: EGFRm NSCLC revenue model assumptions. Source: Analyst estimates; epidemiological data from Globocan 2022, published trial data.

Input	Assumption	Basis
Global EGFRm NSCLC diagnoses p.a.	400,000	Mid-point of published range (350,000–500,000); reflects ~15% EGFR mutation prevalence in Western populations, ~40–50% in East Asian populations, applied to ~2.2 million annual NSCLC diagnoses.
% presenting with advanced disease	70%	Consistent with published literature; majority of EGFRm patients present with stage III/IV disease.
Addressable patient population (base year)	280,000	400,000 × 70%
Epidemiology growth rate	2% p.a.	Reflects rising NSCLC incidence in Asia-Pacific, partially offset by screening-driven stage migration in developed markets.
Geographic reach factor	40%	Limits the commercially addressable population to the US, EU5, Japan, and Australia - markets where RC220 could realistically obtain reimbursement within the model horizon. Excludes China and other emerging markets where pricing and access dynamics differ materially.
Price per patient p.a. (US$)	US$75,000	Positioned below amivantamab-lazertinib (~US$200,000+ annually) and below osimertinib (~US$170,000 annually in the US), reflecting RC220's likely positioning as an add-on combination therapy rather than a backbone agent. Pricing precedents for IV oncology combination partners in NSCLC (e.g. ramucirumab at ~US$60,000–80,000 annually; bevacizumab biosimilars at ~US$40,000–60,000) support this range.
Economics share	100%	Full economics; adjusted implicitly through PoS for partnering optionality.
Approval year	FY2033E	Assumes HARNESS-1 Phase 1a/b completes enrolment by CY2028, pivotal Phase 2/3 initiates CY2029, and approval follows CY2032–2033 - aggressive for a programme yet to dose its first patient; treat as a best-case scenario. Recent FDA NSCLC precedent (zongertinib, sevabertinib, sunvozertinib - all approved 2025 from Phase 1/1b datasets of 70–315 patients) suggests an earlier approval is plausible if Phase 1a/b data are sufficiently compelling - upside optionality not captured in base case.
AUD/USD exchange rate	0.65	Applied to convert US-dollar revenues to Australian dollars throughout the model.
Penetration Ramp	FY33: 0.5% FY34: 1.5% FY35: 3.5% FY36: 6.5% FY37: 10% FY38+: 15%	Begins conservatively at 0.5% in the approval year, reflecting the time required for guideline incorporation, payer negotiations, and physician adoption of a novel G4/MYC mechanism in a setting where established combinations already exist. Peak penetration of 15% is reached in FY2038E and held flat thereafter. This ceiling assumes RC220 captures a meaningful but minority share of the first-line EGFRm combination market, constrained by competition from amivantamab-lazertinib (already approved), osimertinib-chemotherapy combinations, and emerging agents.

At FY2038E peak penetration, the model yields ~21,700 treated patients generating ~A$201M risk-adjusted revenue (8% PoS). Unadjusted gross revenue at peak is ~A$2.5B, underscoring the NSCLC valuation's PoS sensitivity: each 1ppt PoS increase adds ~A$25M in risk-adjusted peak revenue.

Indication 2: Cardioprotection (CPACS)

RC220 is modelled as a co-administered agent with doxorubicin (and potentially other anthracyclines) in advanced solid tumours, assuming CPACS supports a dual cardioprotection-and-anticancer-enhancement label. Revenue is on a direct-sales basis (100% economics). The commercial model resembles a supportive oncology agent (analogous to pegfilgrastim) rather than a primary therapeutic - reflected in pricing and penetration assumptions below.

Figure 21: Cardioprotection revenue model assumptions. Source: Analyst estimates; epidemiological data from published literature.

Input	Assumption	Basis
Global anthracycline-treated patients p.a.	1,500,000	Published estimates of 1-2 million patients receiving anthracycline-containing regimens annually worldwide; mid-point adopted. Includes breast cancer (AC-T, EC), lymphoma, sarcoma, and paediatric cancers.
Addressable subset (developed markets)	50%	Restricts the commercially addressable population to the US, EU5, Japan, Australia, and other markets with established oncology reimbursement infrastructure. Excludes markets where IV supportive oncology agents face pricing or access barriers.
Addressable patients (base year)	750,000	1,500,000 × 50%
Epidemiology growth rate	1% p.a.	Lower than the NSCLC assumption, reflecting the mature and relatively stable use of anthracycline regimens in developed markets. Modest growth is driven by expanding cancer incidence in ageing populations, partially offset by the gradual substitution of anthracyclines with non-cardiotoxic regimens in selected settings.
Price per treatment course (US$)	US$8,000	Positioned as a supportive oncology agent rather than a primary therapeutic. Dexrazoxane treatment courses cost approximately US$2,000–6,000 depending on cumulative anthracycline exposure; RC220's pricing reflects a premium for the dual cardioprotection-plus-anticancer value proposition but remains well below the cost of managing clinical heart failure (estimated lifetime cost of US$150,000–300,000 per case). Pegfilgrastim, the closest commercial analogue as a chemotherapy co-administered supportive agent, is priced at approximately US$3,000–13,000 per injection.
Economics share	100%	Full economics; adjusted implicitly through PoS.
Approval year	FY2032E	One year ahead of NSCLC, reflecting the more advanced clinical status of CPACS (three patients dosed versus zero for HARNESS-1) and the potentially more straightforward regulatory pathway. Assumes Phase 1a/b completion by CY2028, a pivotal Phase 2/3 trial in CY2029-2031, and approval in CY2031–2032. The supportive care component of the label (cardioprotection) may be approvable on the basis of biomarker endpoints (troponin, echocardiographic parameters) under accelerated pathways, though confirmatory outcomes data would likely be required post-approval.
Penetration Ramp	FY32: 0.5% FY33: 2.0% FY34: 4.0% FY35: 7.0% FY36: 10.0% FY37: 15.0% FY38+: 25.0%	Higher terminal penetration than NSCLC (25% vs 15%) reflects different competitive dynamics: dexrazoxane is the only approved cardioprotectant, its use is restricted to patients exceeding 300 mg/m² cumulative doxorubicin, and it carries unresolved concerns about anticancer interference. An RC220 label permitting use from the first cycle would address a substantially larger population with a differentiated value proposition. The 25% ceiling assumes a significant majority of anthracycline-treated patients continue to be managed without pharmacological cardioprotection. The trajectory steepens in later years (10% FY2036E → 25% FY2038E) as accumulating real-world cardiac outcomes data drive accelerating adoption - particularly among paediatric oncologists and breast cancer specialists where long-term survivorship amplifies the cardioprotection value.

At FY2040E peak penetration, the model yields ~217,700 treated patients generating ~A$268M risk-adjusted revenue (10% PoS). Unadjusted gross revenue at peak is ~A$2.7B.

Indication 3: R/R AML

The AML model assumes a licensing transaction: a partner funds and conducts the Phase 3 trial, manufactures and commercialises the product, and pays Racura a royalty. Racura captures a fraction of total product revenue but avoids the US$50–150M capital burden of a haematology Phase 3.

Figure 22: R/R AML revenue model assumptions. Source: Analyst estimates; epidemiological data from SEER, published literature.

Input	Assumption	Basis
US AML diagnoses p.a.	11,000	Consistent with SEER estimates (~20,000 total US AML diagnoses annually; ~55% are not candidates for intensive therapy or achieve durable remission and do not enter the R/R pathway).
% entering R/R setting	45%	Reflects published relapse rates: ~40–50% of patients under 60 and ~70–80% of patients over 60. The 45% blended rate weights toward the younger, intensively treated population most likely to receive salvage therapy.
US R/R patients eligible (base year)	4,950	11,000 × 45%
EU5 patients (added from Year 3+)	2,500	Reflects the expected lag in European regulatory approval and reimbursement relative to the US. The EU5 R/R AML population is broadly comparable in size to the US; the 2,500 figure assumes ~50% of eligible patients are commercially accessible within the model horizon.
Epidemiology growth rate	1% p.a.	AML incidence is rising modestly with ageing populations but is partially offset by improving frontline outcomes (venetoclax-based regimens) reducing the proportion entering the R/R setting.
Price per treatment course (US$)	US$50,000	Below gilteritinib (~US$200,000/year) and venetoclax-based regimens (~US$100,000-150,000/year), reflecting bisantrene's positioning as a salvage agent in heavily pretreated patients with short expected treatment durations. The price assumes a single induction course; patients bridged to transplant would not require ongoing therapy.
Royalty rate on net sales	17.5%	Consistent with precedent royalty rates for licensed oncology assets where the licensor contributes the molecule, formulation, and clinical dataset but the licensee funds pivotal development and commercialisation. Comparable transactions in haematology have yielded royalty rates in the 10–20% range depending on stage and data maturity; 17.5% reflects the strength of the historical efficacy database and the Phase 3-ready status.
Approval year	FY2031E	The earliest approval date in the portfolio, reflecting the bridging Phase 3 strategy that leverages the historical bisantrene database and the two Sheba Phase 2 trials. Assumes a partnering transaction closes in FY2028E, the bridging Phase 3 study initiates in FY2029E, and an accelerated regulatory review (supported by orphan drug designation and the historical database) yields approval in FY2031E. This timeline is contingent on FDA acceptance of the bridging study design, which has not been confirmed.
Penetration Ramp	FY32: 3.0% FY33: 8.0% FY34: 15.0% FY35: 20.0% FY36+: 25.0%	Patients at this stage have few options and poor outcomes (median survival in weeks to months after failing two or more lines), driving rapid adoption of new salvage agents with meaningful response rates. Gilteritinib reached ~20% penetration of the FLT3-mutant R/R AML population within two years of launch; bisantrene's broader eligibility (no molecular restriction) supports a comparable trajectory across the unselected R/R population. Peak 25% is reached by FY2035E and held flat - a ceiling reflecting competition from established salvage regimens (FLAG-Ida, clofarabine-based combinations), emerging targeted agents (menin inhibitors, particularly ziftomenib), and the practical constraint that some R/R AML patients are too frail for any active therapy. 25% assumes RC220 becomes a standard salvage option for patients failing or ineligible for targeted therapies.

At peak (FY2035E+), the model yields ~2,000–2,100 treated patients annually (US+EU5), or ~A$154–162M gross revenue at US$50,000 per course. Racura's 17.5% royalty share is ~A$27–28M before risk adjustment; after the 20% PoS, risk-adjusted royalty income plateaus at ~A$5.4–5.7M p.a.

Operating Cost Assumptions

R&D follows an inverted-U: A$7M in FY2025A rising to a A$25M peak in FY2031E (concurrent CPACS dose escalation and expansion, HARNESS-1 Phase 1a/b and potential Phase 2/3 initiation, regulatory and manufacturing work). The sharp decline to A$10M in FY2032E reflects pivotal trials being partner-funded (NSCLC) or partner-conducted (AML), with Racura's R&D reducing to formulation, biomarker, and lifecycle management. From FY2033E onward R&D stabilises at A$5–8M. The AML Phase 3 is excluded from Racura's cost base. G&A grows from A$3M to A$8M, reflecting incremental headcount in regulatory, medical affairs, BD, and IR as the company transitions from single-programme preclinical to multi-programme clinical-stage. Total opex peaks at A$33.7M in FY2031E and declines to a A$15–17M steady state from FY2033E.

Figure 23: Operating cost assumptions (A$'000). Source: Analyst estimates; company disclosures (FY2025A base year).

OPERATING COST ASSUMPTIONS (A$'000)	FY26E	FY27E	FY28E	FY29E	FY30E	F31E	FY32E
R&D / Clinical Trial Expenditure	8,000	10,000	12,000	15,000	20,000	25,000	10,000
G&A / Corporate Overheads	3,500	3,500	4,000	4,500	5,000	5,500	6,000
IP / Patent Costs	600	700	800	900	1,000	1,000	1,000
Depreciation & Amortisation	50	50	50	75	75	100	100
Capital Expenditure (A$'000)	100	100	100	100	100	100	100
Share-Based Compensation (non-cash)	1,200	1,200	1,500	1,500	1,500	2,000	2,000
Total Operating Expenses	**13,450**	**15,550**	**18,450**	**22,075**	**27,675**	**33,700**	**19,200**

Discount Rate

All cash flows are discounted at a weighted average cost of capital (WACC) of 13%.

Figure 24: WACC build-up. Source: Analyst estimates.

Component	Assumption
Risk-free rate (Rf)	5.0%
Equity market risk premium (EMRP)	5.0%
Beta	1.2
Equity risk premium (Rf + β × EMRP)	11.0%
Size premium	1.0%
Liquidity premium	1.0%
Target leverage (D/E)	0%
WACC	**13%**

Funding & Capital Structure

Racura held A$19.4M in pro-forma cash at the valuation date, no debt, and guided runway through CY2027. The model assumes two equity raises plus progressive exercise of in-the-money (ITM) options over FY2026–2029E to fund clinical development through to self-sustaining cash flows. All ITM options (exercise price below A$2.61) are assumed exercised in the final year before expiry.

Figure 25: Option conversion assumptions by financial year. Source: Company Appendix 3Y filings; analyst estimates. Only ITM options shown; OTM options (RACAM, RACAL, RACAN, RACAP, RACAV, RACAY, RACAAF) are assumed to lapse.

Financial Year	Options Exercised	Shares Issued	Capital Raised (A$'000)	Key Tranches
FY2026E	13,857,672	13,857,672	17,322	RACAAA ($1.25, exp. May 2026) (shareholders – loyalty options)
FY2027E	132,000	132,000	325	RACAO ($2.46, exp. Jun 2027)
FY2028E	1,251,738	1,251,738	2,641	RACAAB ($2.11, exp. Jun 2028)
FY2029E	5,713,958	5,713,958	9,270	RACAW ($1.32), RACAT ($2.23), RACAX ($1.45), RACAZ ($1.39), RACAD ($1.67), RACAAE ($1.67), RACAAC ($2.05)
Total	**20,955,368**	**20,955,368**	**29,558**	

The largest single conversion event occurs in FY2026E, when the 13.86 million RACAAA options (exercise price A$1.25) expire in May 2026. These options are deeply in-the-money and their exercise contributes A$17.3 million in cash.

Capital raises are modelled at the current share price of A$2.61, with 5% issue costs deducted to reflect placement fees and transaction expenses. The two modelled raises are sized to fund the peak R&D expenditure period (FY2027-2031E).

Figure 26: Capital raise assumptions. Source: Analyst estimates.

Financial Year	Gross Amount (A$'000)	Net Proceeds (A$'000)	New Shares Issued	Purpose
FY2027E	25,000	23,750	8,928,571	Fund CPACS dose escalation and expansion; HARNESS-1 Phase 1a/b enrolment; ongoing IP prosecution
FY2029E	60,000	57,000	21,428,571	Fund peak concurrent clinical activity (CPACS expansion, HARNESS-1 pivotal preparation); bridge to partnering income from AML deal (modelled from FY2028E)

At ~A$579M market cap (206.9M shares × A$2.61), the modelled A$85M aggregate raise represents ~15% dilution of post-raise shares. Manageable relative to the clinical programme scale, but a material sensitivity: raises priced meaningfully below A$2.61 would increase both dilution and per-share valuation impact proportionally.

AML Priority Review Voucher (PRV)

The FDA's Priority Review Voucher (PRV) program awards a transferable voucher on approval of a qualifying drug for a rare paediatric disease, tropical disease, or medical countermeasure; vouchers can be sold to a third party and applied to a future NDA/BLA to compress review timelines from ten months to six. The model assumes RC220 secures a PRV via the Rare Paediatric Disease pathway, predicated on Racura pursuing a paediatric AML indication alongside the adult R/R AML programme (paediatric AML qualifies as a rare paediatric disease in the US, with ~500 cases annually). Modelled inputs: A$200M gross voucher sale value (~US$140M at 0.70 AUD/USD, broadly consistent with recent transaction prices of US$100–200M); 20% PoS, conservatively held flat with the AML programme rather than uplifted for the paediatric pathway; voucher receipt in FY2031E, aligned with the AML approval year; and discounting at 13% WACC over 5.5 years from valuation date. The resulting risk-adjusted NPV is A$20.4M (A$0.10/sh, ~2% of EV) - a modest but non-trivial contribution that is also conservative across multiple dimensions (PoS not uplifted; no inflation on voucher price; no consideration of Racura retaining and applying the voucher itself).

SOTP Model Outputs

Sum-of-the-Parts Overview

The valuation is an rNPV on a sum-of-the-parts basis: each indication's cash flows are probability-weighted, discounted at 13% WACC, and summed with a 10× terminal multiple on the final-year risk-adjusted cash flow. Corporate costs are deducted as a separate NPV (preserving each indication's standalone contribution); net cash is added to reach equity value.

Figure 27: SOTP valuation summary and value attribution by indication. Source: Analyst estimates.

Component	rNPV (A$'000)	A$/share	% of EV
EGFRm NSCLC	435,021	$2.10	44.6%
Cardioprotection	570,278	$2.76	58.5%
R/R AML (Royalties)	52,490	$0.25	5.4%
AML Partnering Income	28,525	$0.14	2.9%
AML PRV	20,423	$0.10	2.1%
Corporate Costs	(131,121)	($0.63)	-13.4%
Net Cash	19,375	$0.09	
Total Equity Value	**994,991**	**$4.81**	**100.0%**

Figure 28: Enterprise value to fair value per share bridge. Source: Analyst estimates.

	A$'000	A$/share
Enterprise Value (risk-adjusted)	975,616	
+ Net cash (valuation date)	19,375	0.09
− Debt	-	-
Equity Value	**994,991**	
÷ Shares on issue (fully diluted)	206,858,913	
Fair Value per Share		**A$4.81**
Current Share Price		A$2.61
Implied Upside		+84%

The valuation is dominated by two programmes. Cardioprotection contributes 58% of EV (A$570M rNPV, A$2.76/sh), driven by the 750,000-patient addressable population, 25% peak penetration, and 10% PoS (modestly above the Phase 1 benchmark). NSCLC contributes 45% (A$435M rNPV, A$2.10/sh) - a smaller treated pool but substantially higher per-patient pricing. The two indications together exceed 100% of EV before the corporate cost deduction.

The AML programme adds A$101M combined (A$52M royalties + A$29M partnering income + A$20M PRV, A$0.49/sh). The modest headline understates strategic importance: the US$30M upfront + US$150M milestones partnering stream provides non-dilutive cash from FY2028E that reduces equity funding during peak development. The 17.5% royalty on a small population caps long-term revenue but the stream is capital-light and carries the portfolio's highest PoS (20%).

Equity value of A$995M over 206.9M fully diluted shares yields A$4.81/sh - 84% upside to A$2.61. The valuation is highly sensitive to PoS, WACC, and peak penetration, explored below.

Sensitivity Analysis

WACC vs PoS MULTIPLIER SENSITIVITY					
WACC \ PoS Mult.	**0.50x**	**0.75x**	**1.00x**	**1.25x**	**1.50x**
9.00%	$3.61	$5.77	$7.93	$10.09	$12.25
11.00%	$2.77	$4.47	$6.16	$7.86	$9.55
13.00%	$2.13	$3.47	**$4.81**	$6.15	$7.49
15.00%	$1.65	$2.71	$3.77	$4.83	$5.90
17.00%	$1.28	$2.12	$2.97	$3.82	$4.67

CARDIOPROTECTION PEAK PEN. vs PRICE SENSITIVITY					
CARDIO PEN. \ PRICE	**US$4,000**	**US$6,000**	**US$8,000**	**US$10,000**	**US$12,000**
15%	$2.88	$3.29	$3.71	$4.12	$4.53
20%	$3.16	$3.71	$4.26	$4.81	$5.36
25%	$3.43	$4.12	**$4.81**	$5.50	$6.19
30%	$3.71	$4.53	$5.36	$6.19	$7.02
35%	$3.98	$4.95	$5.91	$6.88	$7.84

EGFRm NSCLC PEAK PEN. vs PRICE SENSITIVITY					
NSCLC PEN. \ PRICE	**US$25,000**	**US$50,000**	**US$75,000**	**US$100,000**	**US$125,000**
5%	$2.94	$3.17	$3.41	$3.64	$3.88
10%	$3.17	$3.64	$4.11	$4.58	$5.04
15%	$3.41	$4.11	**$4.81**	$5.51	$6.21
20%	$3.64	$4.58	$5.51	$6.45	$7.38
25%	$3.88	$5.04	$6.21	$7.38	$8.55

R/R AML PEAK PEN. vs PRICE SENSITIVITY					
AML PEN. \ PRICE	**US$20,000**	**US$35,000**	**US$50,000**	**US$65,000**	**US$80,000**
30%	$4.65	$4.68	$4.71	$4.74	$4.77
40%	$4.68	$4.72	$4.76	$4.80	$4.84
50%	$4.71	$4.76	**$4.81**	$4.86	$4.91
60%	$4.74	$4.80	$4.86	$4.92	$4.98
70%	$4.77	$4.84	$4.91	$4.98	$5.05

WACC vs PoS Multiplier. The PoS multiplier scales all three indication PoS proportionally (e.g. 0.75× takes NSCLC 8%→6%, cardio 10%→7.5%, AML 20%→15%). Fair value ranges A$1.28 (17% WACC, 0.50× PoS) to A$12.25 (9% WACC, 1.50× PoS). At base 13% WACC, fair value exceeds the current share price for all multipliers above ~0.63×. Sensitivity to PoS revisions is approximately linear in this construction - each 0.25× step in the multiplier moves fair value by ~A$1.34 at the base 13% WACC - meaning upside and downside PoS scenarios scale symmetrically around the central A$4.81.

Cardioprotection Peak Penetration vs Price. The largest EV contributor and the single most important valuation driver. Fair value ranges A$2.88 (15% penetration, US$4,000) to A$7.84 (35%, US$12,000), remaining above the current share price in all but the most punitive combinations. At base 25% penetration, halving the price to US$4,000 still yields A$3.43 (23% upside); raising to US$12,000 yields A$6.19. The large addressable population means volume sustains a material revenue base even at conservative pricing.

EGFRm NSCLC Peak Penetration vs Price. Fair value ranges A$2.94 (5% penetration, US$25,000) to A$8.55 (25%, US$125,000) - greater absolute dispersion than cardioprotection given higher per-patient pricing. Penetration and pricing contribute roughly equally: at base US$75,000 pricing, 5%→25% penetration adds ~A$2.80; at base 15% penetration, US$25,000→US$125,000 pricing adds ~A$2.80. NSCLC valuation is

balanced between market-share execution in a competitive landscape and the per-patient price RC220 can command as a combination partner.

R/R AML Peak Penetration vs Price. The total fair-value spread across all AML scenarios is just A$0.40 (A$4.65–A$5.05), confirming the indication's financial relevance under royalty economics is marginal in long-term revenue terms. AML's primary model contribution is near-term partnering income and the PRV monetisation, providing a non-dilutive cash-flow bridge during the peak development period.

US-Listed Peer Trading Comparables

A larger sample of analytically engaged investors prices these names, so the trading data reflects a more complete view of clinical-stage risk than the comparatively shallow ASX biotech investor base. The comparable set spans three buckets: (i) direct programme comparators for EGFRm NSCLC and R/R AML, (ii) multi-asset precision oncology platforms similar in shape to RAC's three-programme structure, and (iii) stage-matched Phase 1 multi-asset oncology peers.

Figure 29: US-Listed Peer Trading Comparables: EGFRm NSCLC, R/R AML, and Multi-Asset Precision Oncology Platforms. Source: Bloomberg, company filings.

Company	Stage	Mcap (US$M)	EV (A$M)	Relevance
RAC (current)	Ph 1a/b ×3	350	337	Reference
Black Diamond (BDTX)	Ph 2	175	47	Direct EGFRm NSCLC comp; Ph 2 ORR 60% (1L); EV near zero
Cullinan (CGEM)	NDA filed (zipalertinib)	895	545	Multi-asset oncology + AML; closest pipeline shape to RAC
Kura Oncology (KURA)	Commercial (KOMZIFTI launch Q1 2026)	870	290	Direct AML comp; commercial menin franchise valued near zero
Erasca (ERAS)	Ph 1/1b	3,150	2,920	Multi-asset RAS/MAPK precision oncology; recent 4× rerating on ERAS-0015
Aktis Oncology (AKTS)	Ph 1b	1,030	815	Recent Ph 1 multi-asset oncology IPO (Jan 2026); platform benchmark
IDEAYA (IDYA)	Ph 2/3 (darovasertib NDA pending)	2,480	1,370	Premier multi-asset precision oncology platform; bull-case anchor
Relay Therapeutics (RLAY)	Ph 3 (zovegalisib BTD)	2,530	1,970	Multi-asset precision oncology platform

The bifurcation between single-asset and multi-asset peers is clear. RAC's current EV sits above most direct single-asset Ph1–2 comparators, so a bull case cannot rest on re-rating to single-asset peer levels. It rests instead on RAC re-rating into the multi-asset precision oncology platform tier (IDEAYA, Relay, Cogent) - companies with established platform credentials via multi-asset pipelines, validated mechanism, and at least one programme through Ph2/3 inflection. The thesis: positive H2 2026 readouts compress the discount between the stage-matched comp set and the platform comp set.

Cullinan Therapeutics is a notable comparator. Its pipeline - zipalertinib (EGFR ex20ins NSCLC, NDA filed Q1 2026), CLN-049 (FLT3xCD3 bispecific in AML), CLN-978 (CD19xCD3 in autoimmune) - combines a near-term commercial NSCLC asset with haematology and adjacent indications, structurally similar to RAC's NSCLC + AML + multi-indication construct. At US$518M EV ex-cash (A$797M), Cullinan trades at ~1.5× RAC's current EV despite having an NDA on file and a Feb-2027 PDUFA date. That premium reflects a single discrete piece of clinical de-risking; the gap between RAC's and Cullinan's EV provides a tangible reference for the value of positive H2 2026 HARNESS-1 or CPACS data.

Implied PoS Analysis

Back-solving from the current share price yields market-implied PoS factors of 5.1% (HARNESS-1), 6.4% (CPACS), and 12.7% (R/R AML) - every programme priced below the industry base rate for its clinical stage. The convex nature of the rNPV (demonstrated in the sensitivity analyses) is visualised below.

Figure 30: Figure 30: Implied PoS Scenario Analysis: Bear to Bull Fair Value Outcomes. Source: Evolution Capital Estimates.

Scenario	PoS Mult.	NSCLC PoS	Cardio PoS	AML PoS	Fair Value (A$/sh)	Description
Bear (market-implied)	0.65×	5.1%	6.4%	12.7%	A$2.61	Current share price level
Pessimistic	0.50×	4.0%	5.0%	10.0%	A$2.09	Significant data setback (from existing sensitivity table)
Stage-adjusted base rate	0.75×	6.0%	7.5%	15.0%	A$3.40	Industry-average PoS, no programme-specific adjustment
Report base case	**1.00×**	**8.0%**	**10.0%**	**20.0%**	**A$4.81**	**Modest programme-specific uplift; central PT**
Catalyst-adjusted	1.25×	10.0%	12.5%	25.0%	A$6.02	Positive H2 2026 CPACS / HARNESS-1 data
Bull	1.50×	12.0%	15.0%	30.0%	A$7.34	Mature Ph 2 data + AML partnering signed

The implied PoS analysis is an analytically powerful cross-check on the rNPV: the current share price requires PoS below industry base rates to hold, and even modest positive H2 2026 readouts compress the discount toward the analytical anchor.

Precedent Licensing & M&A

External BD benchmarks address two questions: what the AML partnering deal might command, and the strategic value of a multi-asset oncology platform in big pharma M&A.

Figure 31: Precedent Licensing & M&A Transactions in Oncology: Benchmarks for AML Partnering and Multi-Asset Platform *Value.* *Source: Company announcements, Evolution Capital estimates.*

Deal	Date	Asset / Stage at Deal	Upfront (US$M)	Total (US$M)	Relevance to RAC
Kura / Kyowa Kirin (License)	Nov-24	Ziftomenib (menin inhibitor), R/R AML; Phase 1/2 with BTD	330	1,491	Direct AML partnering comp; 11× RAC upfront
Syndax / Incyte (License)	Sep-21	Axatilimab (anti-CSF-1R), cGVHD; Phase 2	117 + 35 eq	602	Heme-adjacent floor; 50/50 US profit, double-digit ex-US royalty
BMS / Mirati (Acquisition)	Oct-23	Krazati (KRAS G12C, NSCLC) + multi-asset Ph 1/2 platform	4,800	5,800	Multi-asset precision oncology M&A benchmark (EV ~US$3.7B)
AstraZeneca / Fusion (Acquisition)	Mar-24	FPI-2265 (PSMA Ac-225 radiopharm), mCRPC; Phase 2	2,000	2,400	Phase 2 multi-asset oncology M&A; EV ~US$1.77B at 97% premium
BMS / RayzeBio (Acquisition)	Dec-23	RYZ101 (alpha-radiopharm), GEP-NETs; Phase 1/2	4,100	4,100	Demonstrates big-pharma willingness to acquire novel Ph 1/2 oncology platforms ahead of pivotal data
Janux / BMS (License)	Jan-26	T-cell engager, solid tumour; preclinical single target	50	850	Most recent benchmark; preclinical platform-pricing floor
RAC AML model (current)	-	AML programme partnering (Phase 3-ready bridging)	30	180	Reference: report's modelled AML partnering assumption

The Kura Oncology / Kyowa Kirin transaction (November 2024) is the most relevant precedent in the table. A Phase 1/2 menin inhibitor in R/R AML with Breakthrough Therapy Designation commanded US$330M upfront and up to US$1.49B in total deal value, with a 50/50 US profit share and double-digit ex-US royalty.

The current rNPV model assumes US$30M upfront and US$150M milestones (US$180M total) for RAC's AML programme. The Syndax/Incyte transaction (September 2021) provides a more conservative anchor: a Phase 2 oncology/heme licensing deal with the same profit-share structure commanded US$117M upfront and US$602M total, even without BTD. Our modelled US$30M upfront sits below this floor.

The table below shows the impact of upward revision to modelled deal economics; Kura represents the upper bound.

Figure 32: AML Partnering Deal Sensitivity: Impact on Fair Value vs. Precedent Transactions. Source: Evolution Capital Estimates.

Scenario	Upfront (US$M)	Total Deal (US$M)	AML Partnering rNPV (A$'000)	Fair Value (A$/sh)	Δ vs. base
Current model	**30**	**180**	**28,525**	**A$4.81**	**-**
2× current	60	360	57,050	A$4.95	+A$0.14
Syndax / Incyte floor	120	500	79,242	A$5.06	+A$0.25
Kura w/ 50% haircut	165	750	118,835	A$5.25	+A$0.44
Kura ex opt-in	330	1,161	184,043	A$5.56	+A$0.75
Kura full deal	330	1,491	236,244	A$5.81	+A$1.00

Even with substantial haircuts to Kura terms, AML partnering adds $0.44–$1.00/sh. A deal at half the Kura level lifts fair value to A$5.25; at the Kura ex-opt-in level (US$1.16B total) to A$5.56. The sensitivity is conservative on two dimensions: it holds AML PoS at 20% (a successful partnering deal should justify a modest PoS uplift), and it does not adjust the timing of partnering income for the more advanced deal value. Either adjustment extends the upper bracket modestly. The takeaway: the current AML partnering income line is materially conservative against the precedent set, and signing a deal at any plausible point within the Kura/Syndax range adds meaningful per-share value without requiring revision of any other model input.

Triangulated Valuation Range

The four cross-checks bracket the A$4.81 rNPV fair value within an evidence-based range:

Figure 33: Triangulated Valuation Summary. Source: Evolution Capital Estimates.

Method	Range (A$/sh)	Central (A$/sh)	Notes
rNPV (bottom-up SOTP)	**-**	**A$4.81**	**Base methodology; PoS-weighted DCF across three programmes**
US peer trading comps	A$1.50 - A$12.00	A$4.00 - A$5.50	Wide range reflects bifurcation: single-asset Ph 1/2 framing implies lower end; multi-asset platform framing implies upper end
Implied PoS scenarios (13% WACC)	A$2.09 - A$7.34	A$4.81	Catalyst-adjusted (1.25×) scenario implies A$6.02; bull (1.50×) implies A$7.34
Precedent transactions (AML)	A$4.81 - A$5.81	A$5.06 - A$5.25	Sensitivity on AML partnering income alone; M&A optionality additive
Triangulated band	**A$3.40 - A$6.30**	**A$4.81**	**Cross-method convergence; rNPV remains analytical anchor**

The four cross-checks converge on a triangulated band of A$3.40–6.30/sh, with the rNPV-based A$4.81 as the analytical central estimate. The lower bound is supported by the implied PoS analysis slightly above the market-implied 0.635× multiplier and by single-asset framing of the peer trading comp set. The upper bound is supported by the catalyst-adjusted PoS scenario, the AML partnering sensitivity at the Kura-haircut level, and the lower end of the multi-asset platform peer trading range.

Three asymmetries reinforce the risk-reward. First, H2 2026 provides two independent catalysts (CPACS dose escalation, HARNESS-1 safety/PK) to compress the bear-case discount; PoS convexity means modest positive movement compounds. Second, an AML partnering deal sits above every modelled precedent - upside not contingent on company-led execution. Third, multi-asset platform M&A precedents establish a strategic-value floor accessible once MYC/G4 is human-validated, providing an exit pathway absent from the rNPV but consistent with comparable platforms over the past three years.

Figure 34: Sum-of-the-parts rNPV build (A$'000). Shows gross profit, risk-adjusted cash flows (after PoS application), and present values for each indication, with terminal values (10× exit multiple) and summation to risk-adjusted enterprise value. The Risk-Adjusted Enterprise Value total also includes the AML PRV rNPV of A$20,423k (shown separately in Figure 27), which represents a single discrete cash flow not shown year-by-year here. Source: Analyst estimates.

		FY26E	FY27E	FY28E	FY29E	FY30E	FY31E	FY32E	FY33E	FY34E	FY35E	FY36E	FY37E	FY38E	F039E	FY40E
PART 1: EGFRm NSCLC rNPV																
Gross Profit (A$'000)									-	59,755	182,849	435,181	824,357	1,293,607	1,979,218	2,018,803
Risk-Adjusted Gross Profit									-	4,780	14,628	34,814	65,949	103,489	158,337	161,504
PV of Risk-Adjusted Gross Profit									-	1,911	5,176	10,902	18,276	25,380	34,364	31,019
Sum of PV (FY2026E–FY2040E)	155,028															
+ Terminal Value (10x exit multiple)	279,993															
NSCLC rNPV	**435,021**															
PART 2: CARDIOPROTECTION rNPV																
Gross Profit (A$'000)								-	39,056	157,788	318,731	563,357	812,844	1,231,459	2,072,956	2,093,685
Risk-Adjusted Gross Profit									3,906	15,779	31,873	56,336	81,284	123,146	207,296	209,369
PV of Risk-Adjusted Gross Profit								-	1,765	6,309	11,279	17,642	22,526	30,201	44,989	40,212
Sum of PV (FY2026E–FY2040E)	210,864															
+ Terminal Value (10x exit multiple)	359,414															
Cardio rNPV	**570,278**															
PART 3: R/R AML rNPV																
Royalty Income (A$'000)							-	2,956	7,961	22,107	35,724	52,619	75,922	76,681	77,448	78,222
Risk-Adjusted Royalty Income							-	591	1,592	4,421	7,145	10,524	15,184	15,336	15,490	15,644
PV of Risk-Adjusted Royalty Income							-	302	719	1,768	2,528	3,296	4,208	3,761	3,362	3,005
Sum of PV (FY2026E–FY2040E)	25,634															
+ Terminal Value (10x exit multiple)	26,856															
AML rNPV	**52,490**															
PART 4: AML PARTNERING INCOME rNPV																
Upfront + Milestones (A$'000)				42,857	42,857	42,857	42,857	42,857	42,857							
Risk-Adjusted Partnering Income				8,571	8,571	8,571	8,571	8,571	8,571							
PV of Risk-Adj. Partnering Income				6,315	5,588	4,945	4,376	3,873	3,427							
Partnering Income rNPV	**28,525**															
PART 5: AML PRV rNPV																
PRV Gross Sales Value (A$'000)	200,000															
Risk Adjusted PRV Value (A$'000)	40,000															
PV of Risk-Adjusted PRV (A$'000)	20,423															
PART 5: CORPORATE COSTS NPV																
Corporate Cost Cash Flow		-10,806	-12,365	-14,512	-16,498	-20,928	-26,775	-32,304	-14,821	-13,319	-12,781	-8,941	-7,351	-5,390	-122	-13,061
PV of Corporate Cost Cash Flow		-	-11,632	-12,082	-12,155	-13,644	-15,448	-16,494	-6,697	-5,326	-4,523	-2,800	-2,037	-1,322	-27	-2,508
Sum of PV (FY2026E–FY2040E)	-108,915															
+ Terminal Value (10x exit multiple)	-22,206															
Corporate Costs NPV	**-131,121**															
Risk-Adjusted Enterprise Value	**975,616**															

Appendix

Key Risks

Clinical Development & Asset-Concentration Risk

Racura has three concurrent clinical programmes at Phase 1 or Phase 3-ready stage; historical Phase 1 → approval probability in oncology is 5–10%. Binary trial outcomes - particularly first efficacy signals from CPACS and HARNESS-1 in H2 2026 - will drive significant share-price volatility. The HARNESS-1 hypothesis (MYC suppression delays osimertinib resistance) is mechanistically novel and untested in humans. All three programmes rely on the same molecule (bisantrene/RC220) and formulation, so a fundamental safety signal, formulation instability, or mechanistic invalidation in any one programme could undermine the entire portfolio. The photoisomer discovery - while enabling stronger IP - introduces manufacturing complexity and potential regulatory scrutiny around isomeric purity and stability.

Preclinical-to-Clinical Translation Risk

The G4/MYC mechanism and the preclinical synergy data (osimertinib and doxorubicin) were generated in cell lines and animal models, and oncology has a long history of preclinical promise failing to translate clinically. The cardioprotection hypothesis - that RC220 actively protects the heart when co-administered with doxorubicin - is a stronger claim than bisantrene's historical lower cardiotoxicity profile and may not survive clinical testing.

Funding and Dilution Risk

With A$20.94M cash at December 2025 and guided runway to CY2027, Racura can fund current Phase 1 programmes but may need additional capital for the AML Phase 3, expanded CPACS/HARNESS-1 cohorts, or regulatory activities. At current market capitalisation (~A$493M), a A$20–40M raise would imply 5–10% dilution. The company has relied on placements to sophisticated investors and option conversions; sustainability depends on continued share-price support driven by clinical catalysts.

Intellectual Property Risk

The composition-of-matter patent applications covering (E,E)-bisantrene were filed in 2025 and are not yet granted. Prosecution could narrow the claims; competitors could challenge on obviousness or prior art (bisantrene has been known since the 1970s). If the patents are not granted or are successfully challenged, the IP position reverts to method-of-use claims and regulatory exclusivity - substantially weaker protection.

Competitive Risk

The oncology landscape is intensely dynamic. In EGFRm NSCLC, amivantamab-lazertinib (MARIPOSA) and osimertinib-chemotherapy combinations (FLAURA2) are establishing new standards of care that may reduce HARNESS-1's addressable population. In AML, ziftomenib (Kura, menin inhibitor) and other targeted agents are advancing rapidly. In cardioprotection, novel approaches including liposomal doxorubicin may address the unmet need without a separate co-administered agent.

Partnering, Commercialisation & Key Personnel Risk

The AML Phase 3 programme is explicitly partner-dependent, and Racura is exploring licensing and M&A across its portfolio. Partnership terms and timing are uncertain, and negotiating leverage is directly linked to clinical data quality; failure to secure an AML partner would leave that indication undeveloped. The company has no commercial infrastructure and would require a partner for marketing and distribution. Execution depends heavily on a small leadership team: CEO Dr Daniel Tillett and Executive Chairman Dr Pete Smith anchor scientific and strategic direction (Tillett's binding option conversion aligns his interests but reinforces the dependency). Recent additions of Dr Simon Fisher (VP Medical) and Professor Laurence Hurley (SAB) strengthen the team but do not eliminate key-person risk at this scale.

Regulatory and Jurisdictional Risk
CPACS operates across Australia, Hong Kong, and South Korea; the AML Phase 3 strategy targets the FDA pathway. Multi-jurisdictional development introduces regulatory complexity. The AML bridging Phase 3 strategy - linking historical bisantrene data to RC220 - has not been tested with regulators and may require additional studies if the FDA rejects the bridging design. AUD/USD exposure is material given that most of the company's value will be realised in US-dollar markets.

Board & Management

Dr Peter Smith – Chairman
30+ years in pharma and biotech with an oncology therapeutics focus, spanning concept through Phase 3. Previously CEO of Myrio Therapeutics (private) and ASX-listed Alchemia and AMRAD. Before relocating to Australia, co-founder and CFO of London-based cancer immunotherapy company Onyvax. Earlier career as a top-rated pharmaceuticals analyst at UBS and HSBC across LSE/NASDAQ IPOs, fundraisings, and M&A. BA and PhD (cell signalling), University of Cambridge. Also currently a director of MycRx Inc. and Amala Therapeutics.

Dr Daniel Tillett – MD & CEO
Managing Director and CEO of Racura Oncology. 25+ years of biotech management experience across strategy, IP, sales and marketing, project management, licensing and fundraising. Also founder and CEO of Nucleics, a private Australian biotech whose SaaS genomics tools are used by 250+ companies and research institutions across 30+ countries. Previously Senior Lecturer at La Trobe University's School of Pharmacy, with research spanning pharmacy, phage therapy, virology, microbiology, bioinformatics, and cancer. BSc (Hons 1) and PhD in Molecular Genetics and Biochemistry, UNSW. 40+ peer-reviewed publications and granted patents.

Dr Serge Scrofani – Independent NED
28+ years in healthcare across research, strategy, corporate and business development. VP Strategy & Corporate Development at CSL for 13 years, including roles in CSL's global COVID-19 response and US$11.7B Vifor Pharma acquisition; previously led BD for CSL Behring in the US. Currently Principal at Poplar Advisory (healthcare strategy), board member of the Burnet Institute and The Centre for Eye Research, and founding Director of private equity firm FinCap Pty Ltd. PhD in Structural Biology (La Trobe), postdoctoral research at the University of Melbourne and a Fulbright fellowship at The Scripps Research Institute. MBA, Melbourne Business School.

Dr Megan Baldwin – Independent NED
25+ years in angiogenesis and therapeutics development for cancer and ophthalmic disease. Founder, CIO and (2014–2023) CEO/MD of Opthea Ltd, leading the company from research venture to late-stage clinical enterprise - advancing sozinibercept (OPT-302) from preclinical to global Phase 3 trials in wet AMD, with significant capital raised including a dual Nasdaq/ASX listing. Earlier career at Genentech (now Roche): postdoctoral fellow in Dr Napoleone Ferrara's angiogenesis lab and subsequently led corporate competitive intelligence in oncology. PhD in Medicine (University of Melbourne / Ludwig Institute for Cancer Research). Member of AICD, board member of AusBiotech, and experienced director of private and public companies.

Dr Jose Iglesias – Chief Medical Officer
Pharmaceutical executive and medical oncologist with 30+ years across pharma, biotech, and translational research. Senior leadership roles at Eli Lilly, AMGEN, Bionomics, Abraxis BioScience, Celgene Corporation and Biothera, with deep oncology clinical development and medical affairs expertise across North America, Asia-Pacific, and Europe. As VP Clinical Development at Celgene, oversaw Phase 3 development of Abraxane in pancreatic, lung, and metastatic breast cancer - central to Abraxane's global approvals and its establishment as standard of care in advanced pancreatic cancer. Experienced liaising with global regulators (US, Europe, Australia, China, Korea, Japan), reimbursement agencies, oncology cooperative groups, and translational research

groups. 70+ peer-reviewed publications (12,000+ citations). Active member of ASCO, AACR, and ESMO.

Evolution Capital Ratings System

Recommendation Structure	• **Buy:** The stock is expected to generate a total return of >10% over a 12-month horizon. For stocks classified as 'Speculative', a total return of >30% is expected. • **Hold:** The stock is expected to generate a total return between -10% and +10% over a 12-month horizon. • **Sell:** The stock is expected to generate a total return of <-10% over a 12-month horizon.
Risk Qualifier	• **Speculative ('Spec'):** This qualifier is applied to stocks that bear significantly above-average risk. These can be pre-cash flow companies with nil or prospective operations, companies with only forecast cash flows, and/or those with a stressed balance sheet. Investments in these stocks may carry a high level of capital risk and the potential for material loss.
Other Ratings:	• **Under Review (UR):** The rating and price target have been temporarily suppressed due to market events or other short-term reasons to allow the analyst to more fully consider their view. • **Suspended (S):** Coverage of the stock has been suspended due to market events or other reasons that make coverage impracticable. The previous rating and price target should no longer be relied upon. • **Not Covered (NC):** Evolution Capital does not cover this company and provides no investment view.

Expected total return represents the upside or downside differential between the current share price and the price target, plus the expected next 12-month dividend yield for the company. Price targets are based on a 12-month time frame.

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