RACURA ONCOLOGY

INVESTOR WEBINAR · 19 NOVEMBER 2026

EMILI

Evaluation of a MYC-silencing Intervention in Acute Myeloid LeukemIa

RC220 in relapsed or refractory AML · Seamless Phase 2b/3 clinical trial

Dr Daniel Tillett · Chief Executive Officer & Managing Director

Dr Anupa Kudva · EMILI Lead

TRIAL SNAPSHOT

MILESTONE ACHEIVED
Sheba Medical Center ethics approval (18 November 2026)

PRINCIPAL INVESTIGATOR
Professor Arnon Nagler (Sheba, Israel)

TRIAL DESIGN
Seamless Phase 2b (up to 68 patients) → Phase 3

UPCOMING MILESTONES
First patient expected Q1 2027
First human MYC target engagement data expected after 12-18 patients

IMPORTANT NOTICE

Important notice and disclaimer

01 · DISCLOSURE BASIS

Summary information

Prepared by Racura Oncology Limited (ASX: RAC; “Racura” or the “Company”). Contains summary information only and should be read together with the 18 November 2026 ASX announcement and Racura’s other continuous disclosure filings.

02 · ESTIMATES & RISKS

Forward-looking statements

Includes statements on ethics and regulatory approvals, site governance, site activation, enrolment timing and Phase 3 progression. Such statements involve known and unknown risks and uncertainties; actual outcomes may differ materially. All timelines are estimates and subject to change.

03 · CLINICAL STAGE

Investigational product

RC220 is an investigational formulation of (E,E)-bisantrene and has not been approved by any regulatory authority in any jurisdiction. Preclinical findings and historical RC110 results do not predict the outcome of the EMILI trial.

04 · GENERAL INFORMATION

No financial product advice

Provided for general information only. Does not constitute investment, legal, tax, medical or financial product advice, nor an offer or invitation to subscribe for or purchase securities in Racura Oncology Limited.

18 NOVEMBER MILESTONE

Sheba ethics approval moves EMILI toward first patient

LEAD CLINICAL SITE **Sheba Medical Center, Israel** · **PRINCIPAL INVESTIGATOR** Prof. Arnon Nagler (AML & transplant clinician-scientist)

01

NOV 2026

Ethics approved

Sheba's Institutional Helsinki Committee approved EMILI (minor consent-form updates only; design unchanged). Israeli Ministry of Health confirmed no separate approval required. Institutional governance review is underway and well advanced.

02

Q1 2027 (EST.)

First patient Q1 2027

Following institutional governance approval and protocol training, Sheba site activation is expected by end 2026, with first patient enrolment expected in Q1 2027 (estimate).

03

PLANNED 2027

Expanding globally

Additional sites in Europe, the United States and Australia are planned to open in 2027, subject to required regulatory approvals, ethics clearances and site feasibility.

AML, acute myeloid leukemia.

THE DISEASE

Relapsed or refractory AML remains an area of acute unmet need

What is acute myeloid leukemia (AML)? An aggressive, fast-growing cancer of the blood and bone marrow. Immature white blood cells rapidly accumulate and crowd out healthy blood cells, causing life-threatening infections, bleeding and severe anemia.

33.4%

Five-year relative survival

Across all AML patients from diagnosis; long-term survival falls sharply once patients relapse or become refractory.

6.2–8.7 mth

Median overall survival after salvage

Reported median overall survival with standard intensive salvage chemotherapy in relapsed or refractory AML.

1–2 years

Typical relapse window & toxicity

Most relapses occur within 1–2 years of remission; salvage chemotherapy and transplant carry lasting cardiac, GVHD, infection and fertility risks.

Sources: SEER Cancer Stat Facts: AML (5-year relative survival 33.4%, 2016–2022); Megías-Vericat et al., Ann Hematol 2018 (median OS 6.2–8.7 months); Lee et al., Blood Adv 2020; Granroth et al., Curr Hematol Malig Rep 2022.

AML, acute myeloid leukemia; GVHD, graft-versus-host disease; OS, overall survival; SEER, Surveillance, Epidemiology, and End Results Program.

THE PATIENT REALITY

The clinical goal in relapsed AML

For patients with relapsed AML, the main curative option is an allogeneic stem cell transplant[1].

But first, their leukemia must be brought under control.



WITHOUT REMISSION (ACTIVE DISEASE)

Patients with uncontrolled leukemia blasts generally cannot proceed safely to a donor stem cell transplant.

WITH REMISSION + TRANSPLANT FITNESS

Achieving complete remission (CR or CRi) with manageable toxicity opens the path to potentially curative allo-HSCT.

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allo-HSCT, allogeneic hematopoietic stem cell transplant; AML, acute myeloid leukemia; CR, complete remission; CRi, complete remission with incomplete blood count recovery.

1. Döhner H et al., Blood 2022;140(12):1345–77 (ELN 2022 AML recommendations).

THE CRITICAL HURDLE

The critical hurdle: from relapse to transplant eligibility

01 STARTING POINT

Relapsed or refractory AML

Disease has returned after initial treatment or never responded. Patients may become eligible for transplant if disease control is restored.

→

02 THE BRIDGE

Remission hurdle (CR/CRi)

Leukemia blasts must be cleared to achieve complete remission (CR) or CR with incomplete blood count recovery (CRi).

→

03 CURATIVE GOAL

Allo-HSCT (donor transplant)

Allogeneic hematopoietic stem cell transplant: the main established curative treatment for eligible AML patients[1].

EMILI'S CLINICAL QUESTION

Can a single 7-day course of RC220 bring patients with R/R AML into remission and eligible for transplant?

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allo-HSCT, allogeneic hematopoietic stem cell transplant; AML, acute myeloid leukemia; CR, complete remission; CRi, complete remission with incomplete blood count recovery; R/R, relapsed/refractory.

1. Döhner H et al., Blood 2022;140(12):1345–77.

CLINICAL POSITIONING

How RC220 aims to address key gaps in R/R AML

SALVAGE CHEMOTHERAPY

Intensive cytotoxic salvage

REACH
Broadly used across R/R AML patients fit for intensive re-induction.

OUTCOMES
Median overall survival of 6.2–8.7 months reported in published series (Megías-Vericat et al., 2018).

KEY CONSTRAINTS
Substantial myelosuppression, severe infections and organ toxicity limit transplant fitness.

TARGETED AGENTS

Mutation-specific inhibitors

REACH
Restricted to patients carrying specific driver mutations (e.g. FLT3, IDH1/2).

OUTCOMES
Clinically active in selected subsets, though secondary resistance commonly emerges.

KEY CONSTRAINTS
Many patients with R/R AML lack actionable mutations and need broader options.

WHAT EMILI IS TESTING

RC220: G4 MYC gene strategy

BIOLOGICAL RATIONALE
MYC protein is overexpressed in ~90% of newly diagnosed AML patients (Ohanian et al., 2019).

HYPOTHESIS UNDER TEST
Promoter G4 MYC silencing may act across genotypes, independent of single driver mutations.

TRIAL SCOPE
EMILI evaluates remission rates, allo-HSCT eligibility, safety and MYC target engagement.

Note: EMILI has not yet dosed patients; comparative efficacy and safety of RC220 versus standard of care will be evaluated in the Phase 3 stage (comparator finalized after End-of-Phase 2 consultation).

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allo-HSCT, allogeneic hematopoietic stem cell transplant; AML, acute myeloid leukemia; FLT3, FMS-like tyrosine kinase 3; G4, G-quadruplex; IDH1/2, isocitrate dehydrogenase 1 and 2; R/R, relapsed/refractory.

THE BIOLOGICAL TARGET

MYC acts as a master switch for cancer cell growth

HEALTHY CELL · ON ONLY WHEN NEEDED

MYC is tightly controlled

In normal cells, MYC transcription switches on briefly to support regulated cell renewal and tissue repair, then shuts right back off.

CANCER CELL · STUCK ON

Persistent, unchecked proliferation

Extra gene copies, chromosomal rearrangements or overactive upstream signals lock the MYC gene permanently on—making it one of the most commonly dysregulated cancer genes[1].

LANDMARK PROOF · NATURE 2008

Preclinical proof of concept: switching off MYC reverses established tumors

In a landmark mouse study, systemic inhibition of MYC function caused established lung tumors to regress rapidly, while effects on normal tissues were mild and reversed once MYC function was restored.

Source: Soucek L, et al. Modeling Myc inhibition as a cancer therapy. *Nature*. 2008;455(7213):679–83.

1. Dang CV, Cell 2012;149(1):22–35.

THE 40-YEAR “UNDRUGGABLE” STATUS

Why MYC remained out of reach for conventional drugs

40 years

of research, and still no approved drug that directly targets the MYC protein.

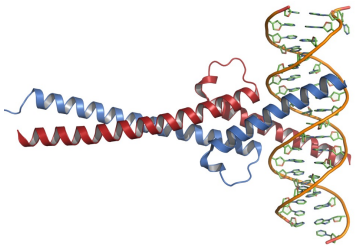
CLINICAL BENCHMARK: PEPTIDE APPROACH

One of the first direct MYC peptide inhibitors to reach the clinic, OMO-103, completed Phase 1 in advanced solid tumors—showing safety but limited clinical activity (Garralda et al., Nat Med 2024).

THE STRUCTURAL REALITY

The MYC–MAX heterodimer grips DNA across a flat, pocketless coiled-coil interface.

Targeting the upstream MYC DNA promoter bypasses this unstructured protein complex.



MYC (red) · MAX (blue) + DNA · PDB: 1NKP (CC BY-SA 3.0)

01 No small-molecule binding pocket

Unlike enzymes such as kinases, the MYC protein has no deep, well-defined pocket for a conventional small molecule to fit into like a key in a lock.

02 Intrinsically disordered protein

Inside the cell nucleus, free MYC lacks a fixed 3D fold—presenting a shifting surface that resists structure-based small-molecule design.

03 Large, flat protein–protein interface

MYC drives transcription by dimerizing with its partner protein MAX across a broad, flat interface that is extremely hard to block directly.

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MAX, MYC-associated factor X; PDB, Protein Data Bank.

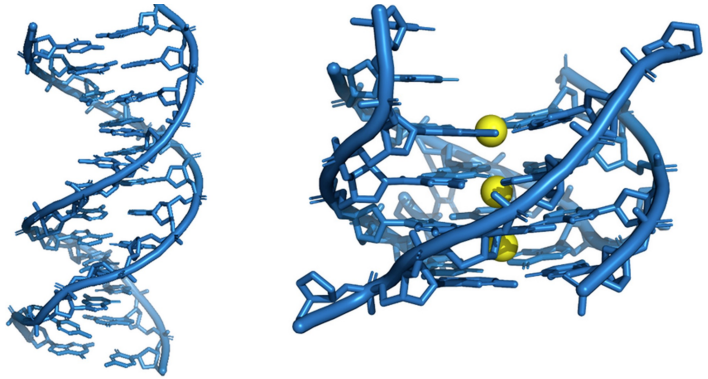
RACURA'S APPROACH

(E,E)-bisantrene turns down MYC at the gene, not the protein

THE USUAL APPROACH
Block the disordered MYC protein after it has already been produced.

RACURA'S MECHANISM
Bind G-quadruplex (G4) DNA in the MYC gene promoter to reduce transcription.

DNA FOLDING SWITCH IN THE MYC PROMOTER



Double-stranded B-DNA helix

Folded G-quadruplex (G4) with K^+ ions

HOW (E,E)-BISANTRENE SILENCES MYC PRECLINICALLY

01 Binds & stabilises promoter G4
Preclinical studies show (E,E)-bisantrene binds and stabilises the folded G4 structure in the MYC promoter.

02 Reduces MYC mRNA transcription
Turns down MYC gene transcription in a dose- and time-dependent manner rather than chasing existing protein.

03 Lowers MYC protein & proliferation
Reduces MYC protein levels and suppresses MYC-driven cell-cycle pathways in preclinical models.

Preclinical foundation: G4 promoter binding and MYC suppression in breast and lung models (ASX Announcements: 2 October 2025, 22 April 2026 & 12 November 2026); AML models (Murray et al., bioRxiv preprint, 16 September 2026). G4 biology ref: Varshney et al., *Nat Rev Mol Cell Biol* 2020. **Confirming this mechanism in patients is a central objective of EMILI.**

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G4, G-quadruplex; K^+, potassium ion; mRNA, messenger RNA.

WHY AML

AML is the right place to test MYC silencing in patients

~90%

MYC drives AML biology

MYC protein is overexpressed in ~90% of newly diagnosed AML patients, sustaining leukemia stem-cell self-renewal; higher levels associated with poorer survival (Ohanian et al., 2019).

nM

Potent preclinical AML activity

University of Newcastle & Racura studies showed nanomolar potency in most AML cell lines tested, dose-dependent MYC silencing, and prolonged survival in AML models (Murray et al., bioRxiv preprint, not peer reviewed, 16 Sep 2026; ASX: 6 Mar 2024).

40%

Prior clinical signal in AML

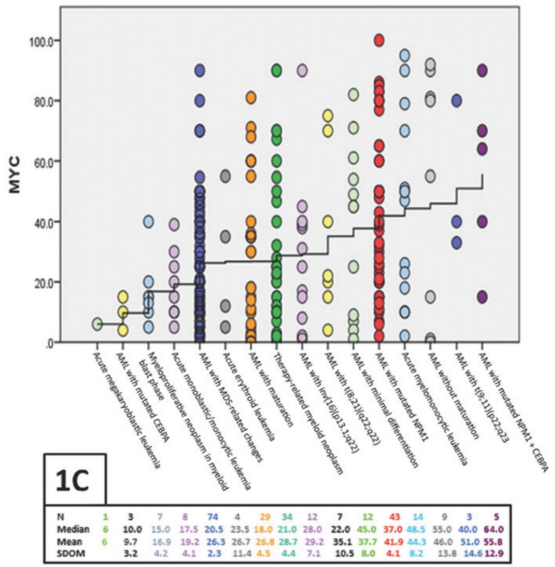
An earlier bisantrene formulation achieved a 40% objective response rate in AML (French approval 1988), and a 40% response rate was also observed in two Sheba studies of RC110.

Blood

Direct, serial biomarker sampling

Unlike solid tumors requiring repeat biopsies, AML cells circulate in blood—enabling serial measurement of MYC protein and MYC-regulated genes across a single 7-day cycle.

MYC expression across WHO AML subtypes



MYC IHC across 265 newly diagnosed AML patients (~90% overexpressed). Source: Ohanian M et al., *Leuk Lymphoma*. 2019;60(1):37–48 (Fig. 1C).

AML, acute myeloid leukemia; IHC, immunohistochemistry; nM, nanomolar; WHO, World Health Organization.

CLINICAL FOUNDATION & FORMULATION

Prior AML activity; RC220 is designed for peripheral IV

1988

French AML approval

Earlier bisantrene formulation approved in France for AML with an objective response rate of 40% (ASX Announcement: 13 May 2024).

Historical benchmark demonstrating prior clinical activity of the active bisantrene moiety in AML.

4 / 10 (40%)

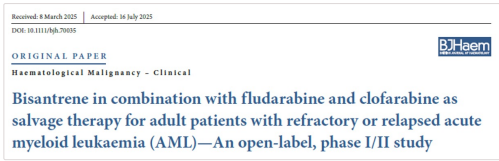
Sheba Phase 2 monotherapy (RC110)


Received: 13 September 2020 | Revised: 1 November 2020 | Accepted: 2 November 2020
DOI: 10.1111/ejh.13544
ORIGINAL ARTICLE
European Journal of Haematology WILEY
A phase II study of bisantrene in patients with relapsed/refractory acute myeloid leukemia

4 / 10 R/R AML patients responded, including 1 complete remission (Canaani et al., Eur J Haematol 2021). Single-center, non-randomized.

6 / 15 (40%)

Sheba Phase 1b/2 combination


Received: 8 March 2025 | Accepted: 16 July 2025
DOI: 10.1111/bjh.70035
ORIGINAL PAPER
Haematological Malignancy - Clinical
BJHaem
Bisantrene in combination with fludarabine and clofarabine as salvage therapy for adult patients with refractory or relapsed acute myeloid leukaemia (AML)—An open-label, phase I/II study

6 / 15 evaluable patients responded with fludarabine + clofarabine + RC110, including 5 CRs (Danylesko et al., Br J Haematol 2025).



Why RC220 instead of historical RC110? Designed for peripheral IV without a central line

Earlier bisantrene formulations precipitated at neutral blood pH (~7.4), causing vein inflammation unless delivered via a central venous line. In Good Laboratory Practice (GLP) non-clinical toxicology studies (ASX Announcement: 26 June 2024), **RC220 was delivered via peripheral veins with no adverse infusion-site or vein reactions** and showed similar systemic effects to central-line historical bisantrene.

AML, acute myeloid leukemia; CR, complete remission; GLP, Good Laboratory Practice; IV, intravenous; R/R, relapsed/refractory.

TRIAL DESIGN

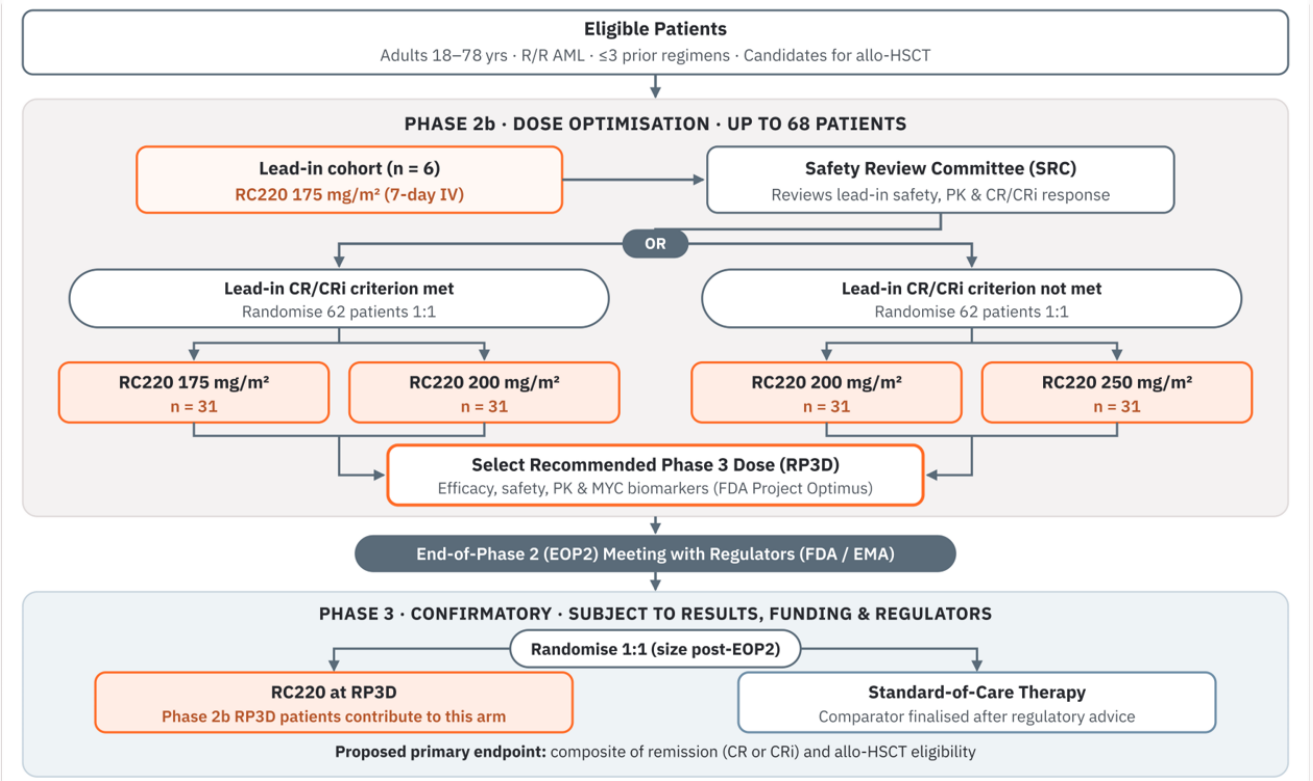
EMILI: seamless Phase 2b/3 designed for FDA Project Optimus

ELIGIBLE PATIENTS
Adults 18–78 years with R/R AML, ≤3 prior regimens, allo-HSCT candidates.

TREATMENT REGIMEN
Single 7-day cycle of RC220: 2-hour peripheral IV once daily, Days 1–7.

PHASE 2b
Up to 68 patients: 6-patient lead-in (175 mg/m²), then 62 randomised 1:1.

PHASE 3 (CONDITIONAL)
RC220 at RP3D vs standard of care (1:1); comparator & size set at End-of-Phase 2 (EOP2). Conditional on Phase 2b data and regulators.



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allo-HSCT, allogeneic hematopoietic stem cell transplant; AML, acute myeloid leukemia; CR, complete remission; CRi, complete remission with incomplete blood count recovery; EMA, European Medicines Agency; EOP2, End-of-Phase 2; FDA, US Food and Drug Administration; IV, intravenous; PK, pharmacokinetics; R/R, relapsed/refractory; RP3D, Recommended Phase 3 Dose; SRC, Safety Review Committee.

PHASE 2b MECHANISM

Adaptive design built to select the optimal dose efficiently

STEP 1 · LEAD-IN

6 patients at 175 mg/m²

Safety Review Committee (SRC) assesses initial safety, pharmacokinetics (PK) and clinical response data before opening randomization.

→

STEP 2 · ADAPTIVE GATE

Randomize 62 patients (1:1)

Depending on the lead-in CR/CRi rate, 62 patients randomize 1:1 to 175 vs 200 mg/m² or step up to 200 vs 250 mg/m².

→

STEP 3 · DOSE SELECTION

Select RP3D for Phase 3

Recommended Phase 3 Dose (RP3D) is chosen on integrated remission (CR/CRi), allo-HSCT eligibility, safety, PK and MYC biomarker data.

Patients ~11 · 16 · 21

Interim assessments per dose arm

Scheduled interim analyses occur after ~11, 16 and 21 patients per arm under Safety Review Committee oversight to monitor safety and futility criteria and pause an arm early if required.

FDA Project Optimus alignment



Under FDA Project Optimus, oncology developers compare doses head-to-head to select the dose offering the best balance of efficacy and tolerability—not simply the highest tolerated dose—strengthening Phase 3 readiness.

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allo-HSCT, allogeneic hematopoietic stem cell transplant; CR, complete remission; CRi, complete remission with incomplete blood count recovery; FDA, US Food and Drug Administration; PK, pharmacokinetics; RP3D, Recommended Phase 3 Dose; SRC, Safety Review Committee.

READOUTS & TRANSLATIONAL VALUE

What Phase 2b will evaluate and report to investors

CLINICAL ENDPOINTS

Clinical & dose-selection readouts

Remission rates by dose arm

Rates of complete remission (CR) and complete remission with incomplete blood count recovery (CRi) after a single 7-day cycle.

Transplant eligibility rate

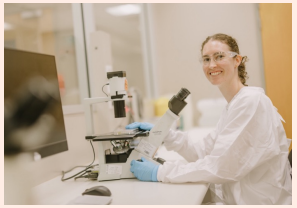
Proportion of treated patients who achieve disease control and are clinically eligible to proceed to allo-HSCT.

Safety, tolerability & PK

Full adverse-event profile and human pharmacokinetics (PK) across active dose levels.

Phase 3 confirmatory endpoint

Proposed composite endpoint combining remission (CR or CRi) and allo-HSCT eligibility, finalized after End-of-Phase 2 (EOP2) consultation.



TRANSLATIONAL SCIENCE

MYC biomarkers & pipeline relevance

Direct target engagement in blood

Exploratory analyses measure changes in MYC protein levels and MYC-regulated gene expression in circulating patient leukemia cells.

Biomarker readout window

Pharmacodynamic (PD) effects are expected to be measurable from the first patient; robust biomarker conclusions are expected once 12–18 patients are evaluated.

Relevant to HARNESS-1 & CPACS

Clinical confirmation that RC220 silences MYC in humans is relevant to Racura's Phase 1 HARNESS-1 (EGFR-mutated lung cancer) and Phase 1 CPACS programs.

RACURA ONCOLOGY

allo-HSCT, allogeneic hematopoietic stem cell transplant; CR, complete remission; CRi, complete remission with incomplete blood count recovery; EGFR, epidermal growth factor receptor; EOP2, End-of-Phase 2; PD, pharmacodynamic; PK, pharmacokinetics.

EXECUTION ROADMAP

Milestones from Sheba ethics approval to Phase 2b readout

NOV 2026

Ethics approved

Sheba Helsinki Committee approval received; minor consent-form updates only.

UNDERWAY

Site governance

Sheba contractual, financial & operational governance review is underway and well advanced.

BY END 2026

Trial initiation

Governance completion, protocol training and Sheba site opening expected by end 2026.

Q1 2027

First patient

First patient expected to be enrolled and treated at Sheba (6-patient lead-in at 175 mg/m²).

UPCOMING

CRO, registry & global sites

Timing TBA: CRO, ClinicalTrials.gov ID, FDA clearance & EMA (CTIS) approval. Planned 2027: US, European & Australian sites, subject to approvals.

18–24
MONTHS (EST.)

Phase 2b estimated duration

Duration depends on site activation pace, patient recruitment and the outcomes of scheduled interim assessments (~11, 16 and 21 patients per arm).

12–18
PATIENTS

MYC biomarker readout window

Initial MYC measurements are expected from patient 1; robust conclusions expected after 12–18 patients. All timelines are estimates and subject to change.

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CRO, contract research organization; CTIS, Clinical Trials Information System; EMA, European Medicines Agency; FDA, US Food and Drug Administration.

INVESTMENT CASE

Why EMILI matters for Racura investors

01 DUAL CLINICAL + BIOMARKER OBJECTIVE

Two core questions in one trial

Tests whether a single 7-day course of RC220 can bridge relapsed/refractory AML patients to transplant, while measuring direct MYC target engagement in patient leukemia cells.

02 CAPITAL & TIME EFFICIENCY

Seamless Phase 2b/3 efficiency

One protocol transitions Phase 2b into confirmatory Phase 3 without launching a new study, and Phase 2b RP3D patients contribute to the Phase 3 RC220 arm. Progression depends on Phase 2b results and regulatory consultation.

03 REGULATORY EXCLUSIVITY

US Orphan Drug Designation

RC220 holds US FDA Orphan Drug Designation for AML (ASX Announcement: 11 June 2024), providing eligibility for seven years of US market exclusivity on approval.

04 PORTFOLIO VALIDATION

Potential pipeline read-through

Demonstrating human MYC silencing and clinical activity in AML would strengthen the biological rationale across Racura's broader pipeline - relevant to Phase 1 HARNESS-1 and Phase 1 CPACS.

AML, acute myeloid leukemia; FDA, US Food and Drug Administration; RP3D, Recommended Phase 3 Dose.

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Questions & Discussion



Ethics approved at Sheba · First patient expected Q1 2027

WEBINAR SPEAKERS & LEAD SITE

Dr Daniel Tillett — Chief Executive Officer & Managing Director

Dr Anupa Kudva — EMILI Lead

Principal Investigator: Prof. Arnon Nagler (Sheba Medical Center)

CLINICAL & INVESTOR ENQUIRIES

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Authorised for release by Daniel Tillett, CEO