RACURA ONCOLOGY

ASX Announcement

Racura RC220 MYC-silencing data to be presented at the 2026 Frontiers in Cancer Science Conference

- **Poster presentation accepted for the Frontiers in Cancer Science 2026 (FCS) Conference to be held in Singapore, 11-13 November 2026**
- **Presentation reveals preclinical data showing that (E,E)-bisantrene is the active isomer stabilising the G-quadruplex (G4) DNA region found in the MYC gene promoter leading to MYC suppression**
- **Results support the MYC-driven mechanism of action of (E,E)-bisantrene and use of RC220 in the EMILI, HARNESS and CPACS clinical programs**

8 September 2026

Racura Oncology Limited ("Racura" or the "Company") is pleased to announce that it has been selected to present at the 2026 Frontiers in Cancer Science (FCS) Conference, held in partnership with the American Association for Cancer Research (AACR), in Singapore from 11-13 November 2026. The FCS presentation describes results from preclinical studies undertaken by Racura scientists that have established (E,E)-bisantrene is the active bisantrene isomer stabilising the G-quadruplex (G4) region found in the promoter region of the MYC gene, leading to reduced MYC gene expression and MYC activity.

The accepted abstract entitled *"Light-Induced Isomerization Attenuates (E,E)-Bisantrene G-Quadruplex Binding and c-MYC Silencing*" is attached to this announcement and will be later published in the AACR peer-reviewed journal, *Cancer Research*.

Racura Oncology CEO and Managing Director, Dr Daniel Tillett commented: *"These results reinforce our focus on (E,E)-bisantrene and underpin the clinical development strategy for RC220. Confirming (E,E)-bisantrene as the active isomer driving MYC G4 stabilisation and MYC silencing provides important mechanistic support for our anthracycline cardioprotection, EGFR-mutant non-small cell lung cancer and acute myeloid leukaemia clinical programs. We are pleased this work has been accepted for FCS 2026, a respected international cancer science conference held in partnership with AACR."*

Importance of MYC & G4-DNA in cancer

The MYC protein controls the expression of many cancer-related genes, including those involved in cell growth, differentiation, survival, metabolic reprogramming, anticancer therapy resistance, and immune

surveillance.[1] MYC is dysregulated in up to 70% of all cancers, but is widely regarded as undruggable by the pharmaceutical industry.[2] The MYC gene promoter contains a G4 DNA structural region known to suppress MYC expression when stabilised by specific small molecule binders.[3] Data revealing (E,E)-bisantrene can reduce MYC gene expression via G4 DNA binding and stabilisation was presented at the 2026 AACR Annual Meeting (ASX Announcement: 22 April 2026).

The data to be presented at the FCS conference identifies (E,E)-bisantrene as the active isomer, with the light-induced (E,Z)-isomer showing significantly less G4 stabilisation, MYC silencing, and anticancer activity than the (E,E)-isomer.

IP significance

The results presented at the FCS conference support the use of pure (E,E)-bisantrene in the RC220 formulation and strengthen the Company's patent filings covering protection of (E,E)-bisantrene from light-induced photoisomerisation, manufacture and formulation of pure (E,E)-bisantrene, and associated composition of matter claims on (E,E)-bisantrene, (E,Z)-bisantrene and various isomer mixtures.

References

1. González-Larreategui, Í., Valdés-Bango Martín, M., Casacuberta-Serra, S. and Soucek, L. (2026) 'MYC at the tumor–immune interface: mechanisms of immune escape and immunotherapy resistance', *Frontiers in Immunology*, 17, 1738440.
2. Llombart, V. and Mansour, M.R. (2022) 'Therapeutic targeting of "undruggable" MYC', *eBioMedicine*, 75, 103756.
3. Siddiqui-Jain, A., Grand, C.L., Bearss, D.J. and Hurley, L.H. (2002) 'Direct evidence for a G-quadruplex in a promoter region and its targeting with a small molecule to repress c-MYC transcription', *Proceedings of the National Academy of Sciences of the United States of America*, 99(18), pp. 11593–11598.

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About Racura Oncology

Racura Oncology (ASX: RAC) is a Phase 3 clinical-stage biopharmaceutical company with a mission to silence cancer.

Racura's lead asset, (E,E)-bisantrene, is a clinically derisked small-molecule anticancer agent that primarily acts by stabilising G4-DNA and RNA structures, driving potent silencing of c-MYC, a key cancer gene dysregulated in up to 70% of all cancers. (E,E)-bisantrene has shown therapeutic activity in cancer patients and has a well-characterised safety profile. Racura's discoveries have supported composition-of-matter IP filings that, if granted, could provide up to 20 years of patent protection for (E,E)-bisantrene.

Racura is advancing RC220, its proprietary formulation of (E,E)-bisantrene, to address significant unmet need across multiple MYC-driven oncology indications. The Company's clinical programs include the EMILI Phase 3 trial in acute myeloid leukaemia, the HARNESS Phase 1a/b trial in combination with osimertinib for EGFR-mutated non-small cell lung cancer, and the CPACS Phase 1a/b trial in combination with doxorubicin, where Racura aims to deliver both anthracycline cardioprotection and enhanced anticancer activity.

Racura has collaborated with Astex, Emory University, Purdue University, MD Anderson Cancer Center, Sheba Medical Center, University of North Carolina School of Medicine, University of Wollongong, and the University of Newcastle. Racura is actively exploring partnerships, licence agreements, and potential commercial merger and acquisition opportunities to accelerate global patient access to RC220. Learn more at www.racuraoncology.com.

For questions about this announcement or previous Racura Oncology announcements, please visit our Interactive Announcements page.

Racura encourages our investors to go paperless by registering their email details with the Company's share registry, Automic Registry Services, at www.automicgroup.com.au.

Release authorised by

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Light-Induced Isomerization Attenuates (E,E)-Bisantrene G-Quadruplex Binding and *c-MYC* Silencing

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Background: (E,E)-bisantrene consists of an anthracene core with identical (E)-hydrazone substituents at the 9- and 10-positions. It can stabilize G-quadruplex (G4) DNA structures leading to silencing of *c-MYC* gene expression. In solution, visible light converts (E,E)-bisantrene into (E,E)/(E,Z)/(Z,Z) mixtures, raising the possibility that photoisomerization compromises G4-directed activity and anticancer activity. We compared pure (E,E)-bisantrene with mixtures containing increasing (E,Z)- and (Z,Z)-bisantrene for effects on *c-MYC* promoter G4 stabilization, *c-MYC* expression, and cancer-cell viability.

Methods: (E,E)-bisantrene solutions were exposed to blue LED light to generate (E,E)/(E,Z)/(Z,Z) mixtures, which were quantified by HPLC. *c-MYC* promoter G4 binding and stabilization were measured by circular dichroism spectroscopy. Molecular dynamics simulations estimated isomer-specific G4 binding energies, and *c-MYC* gene/protein expression and viability were assessed across cancer cell lines.

Results: Blue LED light exposure progressively increased (E,Z)- and (Z,Z)-bisantrene and depleted (E,E)-bisantrene. Depletion of (E,E)-bisantrene reduced *c-MYC* promoter G4 stabilization, *c-MYC* gene/protein suppression, and cancer-cell growth inhibition. Molecular dynamics simulations supported more favorable G4 binding by (E,E)- than (E,Z)-bisantrene.

Conclusion: (E,E)-bisantrene is the active isomer. Photoisomerization to (E,Z)- and (Z,Z)-bisantrene weakens *c-MYC* G4 binding/stabilization, reduces *c-MYC* silencing, and diminishes anticancer activity, supporting light protection and clinical use of pure (E,E)-bisantrene. These findings indicate that formulation, storage, and handling conditions that avoid light exposure to preserve the (E,E)-isomer are important for maintaining therapeutic potency in clinical settings.