ASX Announcement

Racura R&D Symposium Presentation

24 March 2026

Racura Oncology Limited ("Racura") is pleased to share the presentation for the inaugural Racura Oncology Research & Development Symposium, being held at Museum of Sydney from 2.30pm today.

CEO and Managing Director, Dr Daniel Tillett commented, *"We welcome investors to our inaugural investor symposium today. The Racura team has worked hard to bring together a program that we believe provides a solid understanding of our science and pipeline. I would like to thank all those involved in the symposium preparation and delivery, particularly our external experts, for their contribution to this event."*

The attached presentation includes QR codes throughout which enable access to video and animation content unable to be included in this release. A video recording will be made available shortly after the event.

-ENDS-

About Racura Oncology

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

Racura's lead asset, (E,E)-bisantrene, is a small molecule anticancer agent that primarily functions via G4-DNA & RNA binding, leading to potent silencing of the important cancer growth regulator MYC. (E,E)-bisantrene has demonstrated therapeutic activity in cancer patients with a well characterised safety profile. Recent discoveries made by Racura have enabled composition of matter IP filings that provide for 20 years of patent protection over (E,E)-bisantrene.

Racura is advancing a proprietary formulation of (E,E)-bisantrene (RC220) to address the high unmet needs of patients across multiple oncology indications, with a Phase 3 clinical program in acute myeloid leukaemia (AML), a Phase 1a/b program in mutant epidermal growth factor receptor non-small cell lung cancer (EGFRm NSCLC), and a Phase 1a/b program in combination with the anthracycline doxorubicin, where we aim to deliver both cardioprotection and enhanced anticancer activity for solid tumour patients.

Racura Oncology has collaborated with Astex, Emory University, Purdue University, MD Anderson, Sheba City of Health, UNC School of Medicine, University of Wollongong, and University of Newcastle. Racura is actively exploring partnerships, licence agreements, or a commercial merger and acquisition to accelerate access to RC220 for patients with cancer across the world. Learn more at www.racuraoncology.com.

If you have any questions on this announcement, or any past Racura Oncology announcements please visit our Interactive Announcements page.

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

Release authorised by

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RACURA ONCOLOGY

Racura Oncology Symposium

Sydney, 24 March 2026

RACURA ONCOLOGY

Welcome

Dr Pete Smith, Executive Chair

Important notice and disclaimer

The material in this presentation has been prepared by Racura Oncology Limited (ACN 149 318 749) (Company).

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FORWARD-LOOKING STATEMENTS

This presentation may contain forward-looking statements that are subject to risk factors associated with an oncology company. Forward looking statements can be identified by the use of forward-looking terminology, including, without limitation, the terms “believes”, “estimates”, “anticipates”, “expects”, “predicts”, “intends”, “plans”, “goals”, “targets”, “aims”, “outlook”, “guidance”, “forecasts”, “may”, “will”, “would”, “could” or “should” or, in each case, their negative or other variations or comparable terminology. These forward-looking statements include all matters that are not historical facts. By their nature, forward-looking statements involve known and unknown risks, uncertainties and other factors because they relate to events and depend on circumstances that may or may not occur in the future and may be beyond the Company’s ability to control or predict which may cause the actual results or performance of the Company to be materially different from the results or performance expressed or implied by such forward-looking statements. Forward looking statements are based on assumptions and are not guarantees or predictions of future performance. No representation is made that any of these statements or projections will come to pass or that any forecast result will be achieved, nor as to their accuracy, completeness or correctness. Similarly, no representation is given that the assumptions upon which forward looking statements may be based are reasonable.

Agenda

Time	Session	Presenters
2.30pm	Welcome	Dr Pete Smith
2.35pm	Opening remarks	Dr Daniel Tillett
2.40pm	Science behind Racura Oncology	Prof Mike Kelso & Prof Laurence Hurley
3.25pm	CPACS clinical program	A/Prof Erin Howden & Dr Marinella Messina
3.50pm	HARNESS-1 clinical program	Prof Nick Pavlakis & Dr Rodney Cusack
4.15pm	Phase 3 AML clinical program	Dr Anupa Kudva
4.35pm	Protecting innovation & delivering milestones	Dr Pete Smith
4.45pm	General Q&A	All
5.00pm	Networking drinks & light canapes	All

RACURA ONCOLOGY

Opening remarks

Dr Daniel Tillett, CEO & MD

What makes Racura Oncology unique?

Addressing major clinical markets with a clinical proven drug and the team to deliver

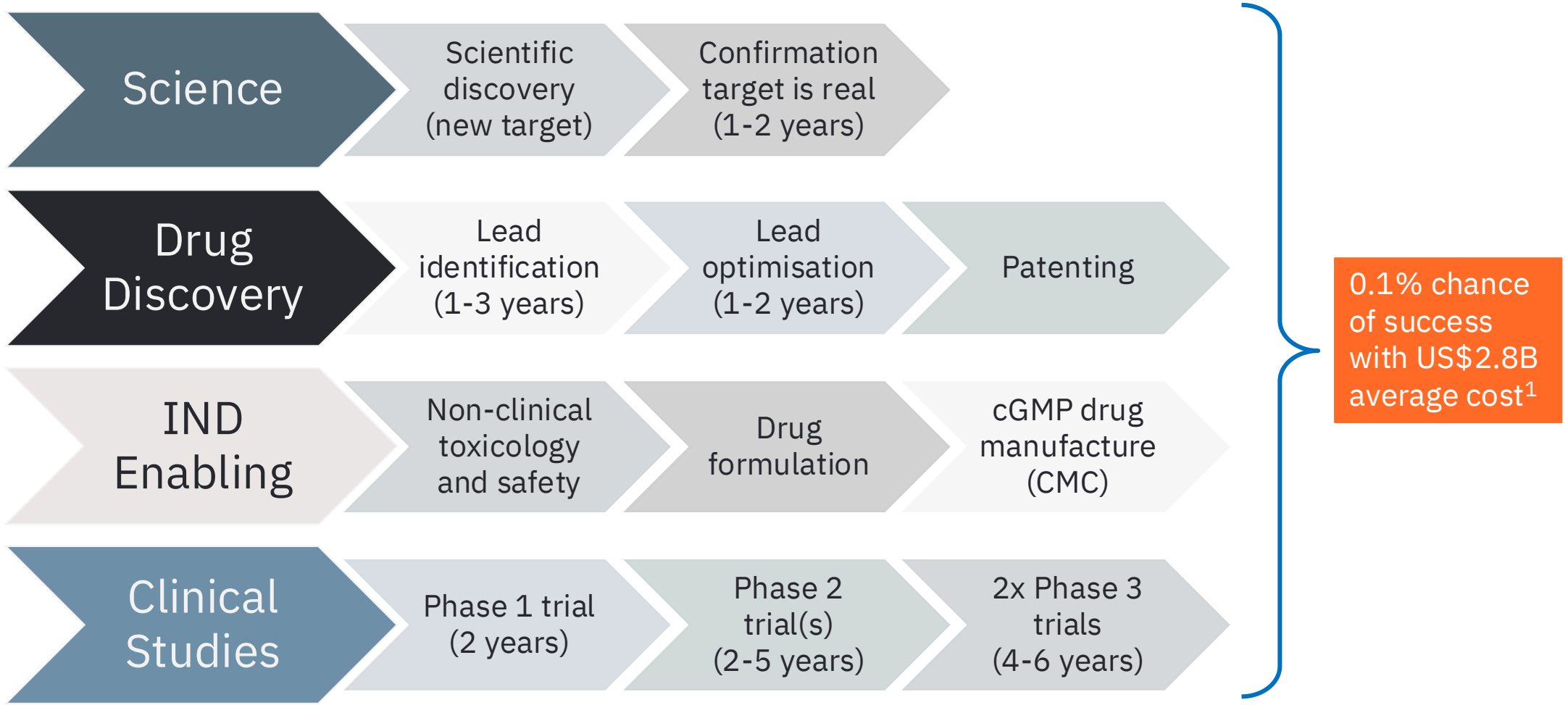
- **Clinically derisked** – our drug works!
  - Previously approved for AML in France (1988)
- Clinical problems that needed to be solved are those that are tractable
- We are targeting multiple clinical indications with >US$10 billion total addressable markets
- Many feasible pathways to a commercial return
- Most importantly, we have the team with the skills and experience to deliver on the promise

RACURA ONCOLOGY

AML, acute myeloid leukaemia

Standard oncology drug development process

Slow, expensive, and high risk

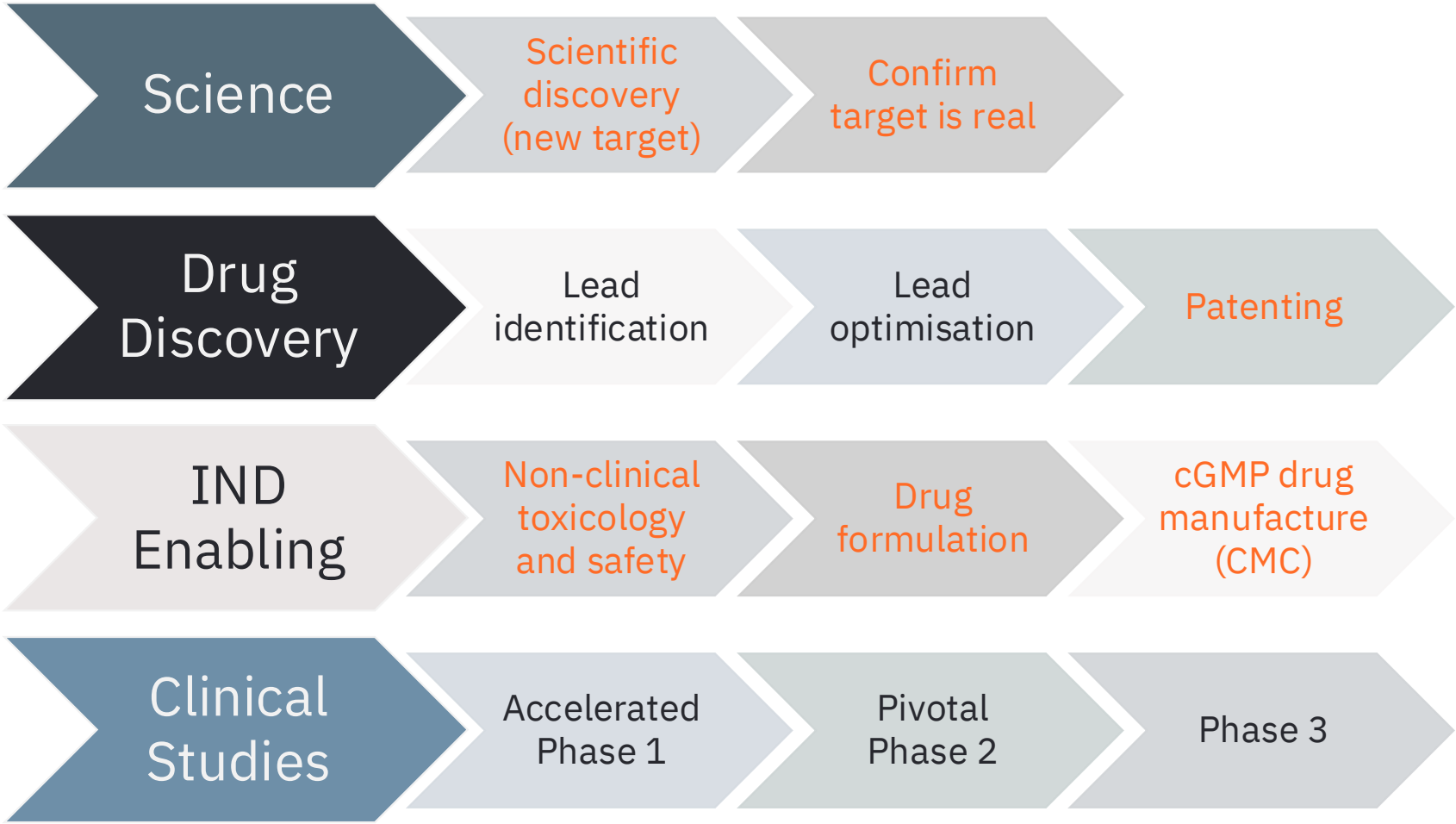


RACURA ONCOLOGY

1. Wouters OJ, McKee M, Luyten J (2020) JAMA, 323:844–853. | IND, Investigational New Drug; CMC, chemistry, manufacturing and controls

Racura Oncology drug development challenge

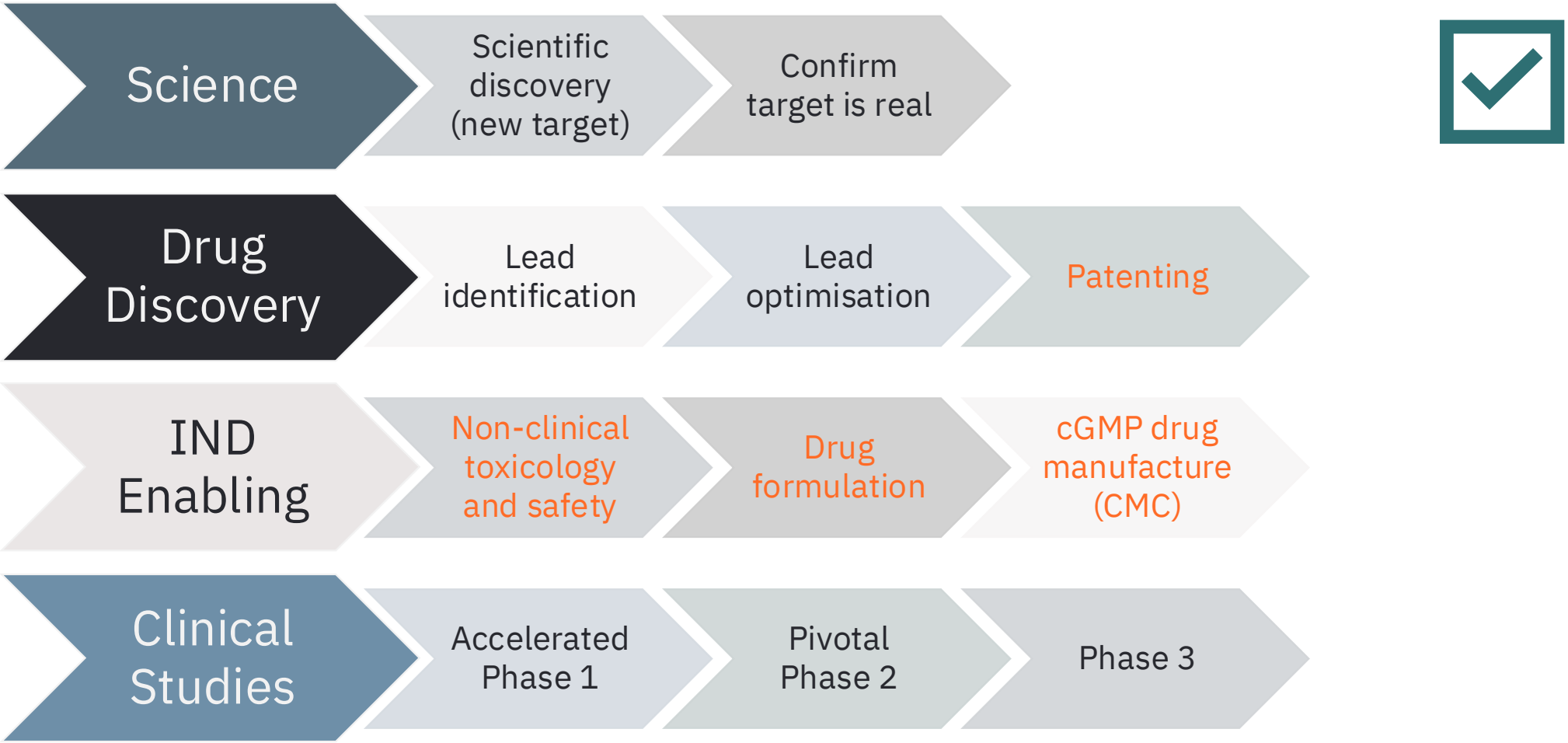
Systematically solving tractable scientific and clinical problems in cost effective manner



IND, Investigational New Drug, CMC; chemistry, manufacturing and controls

Racura Oncology drug development success

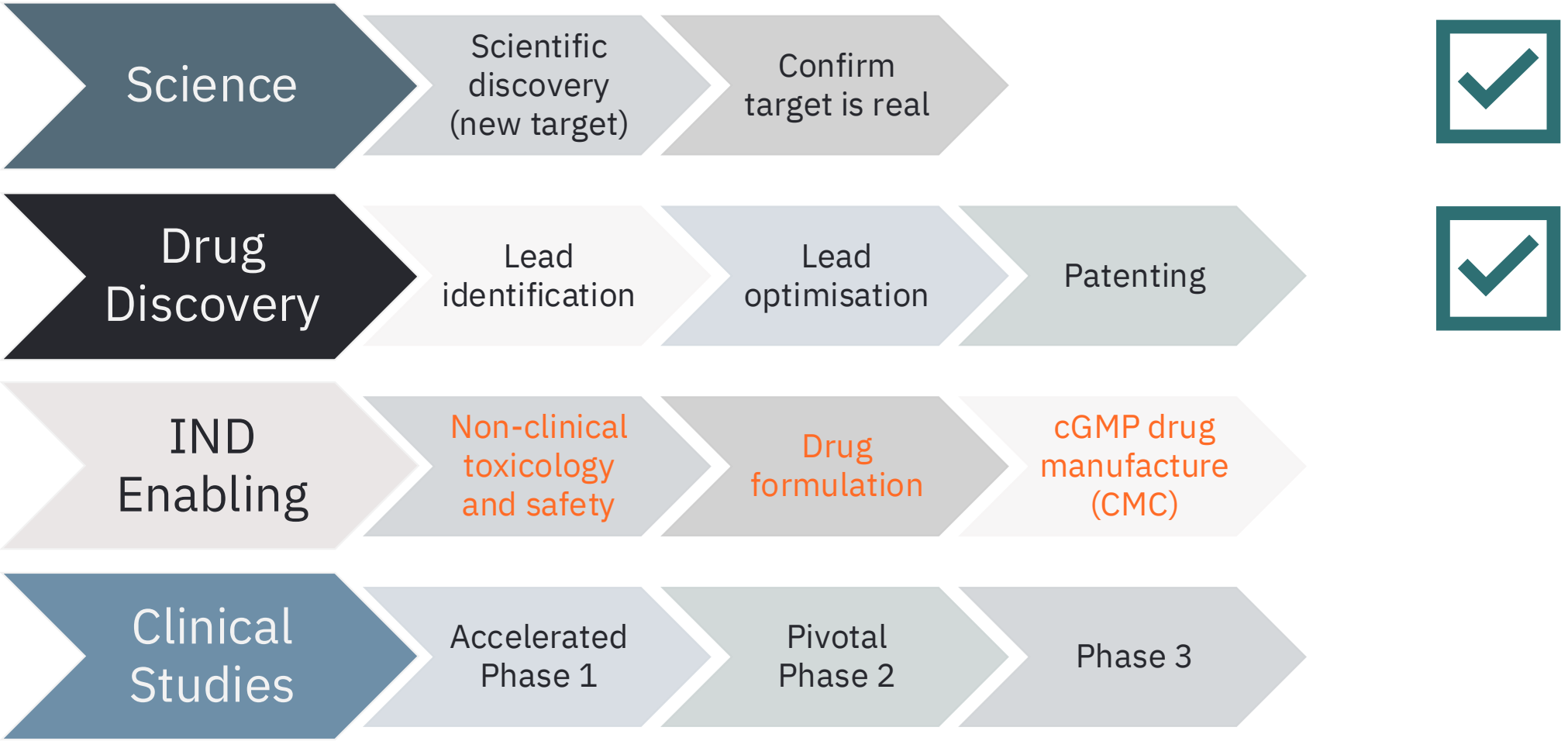
Systematically solving tractable scientific and clinical problems in cost effective manner



IND, Investigational New Drug, CMC; chemistry, manufacturing and controls

Racura Oncology drug development success

Systematically solving tractable scientific and clinical problems in cost effective manner

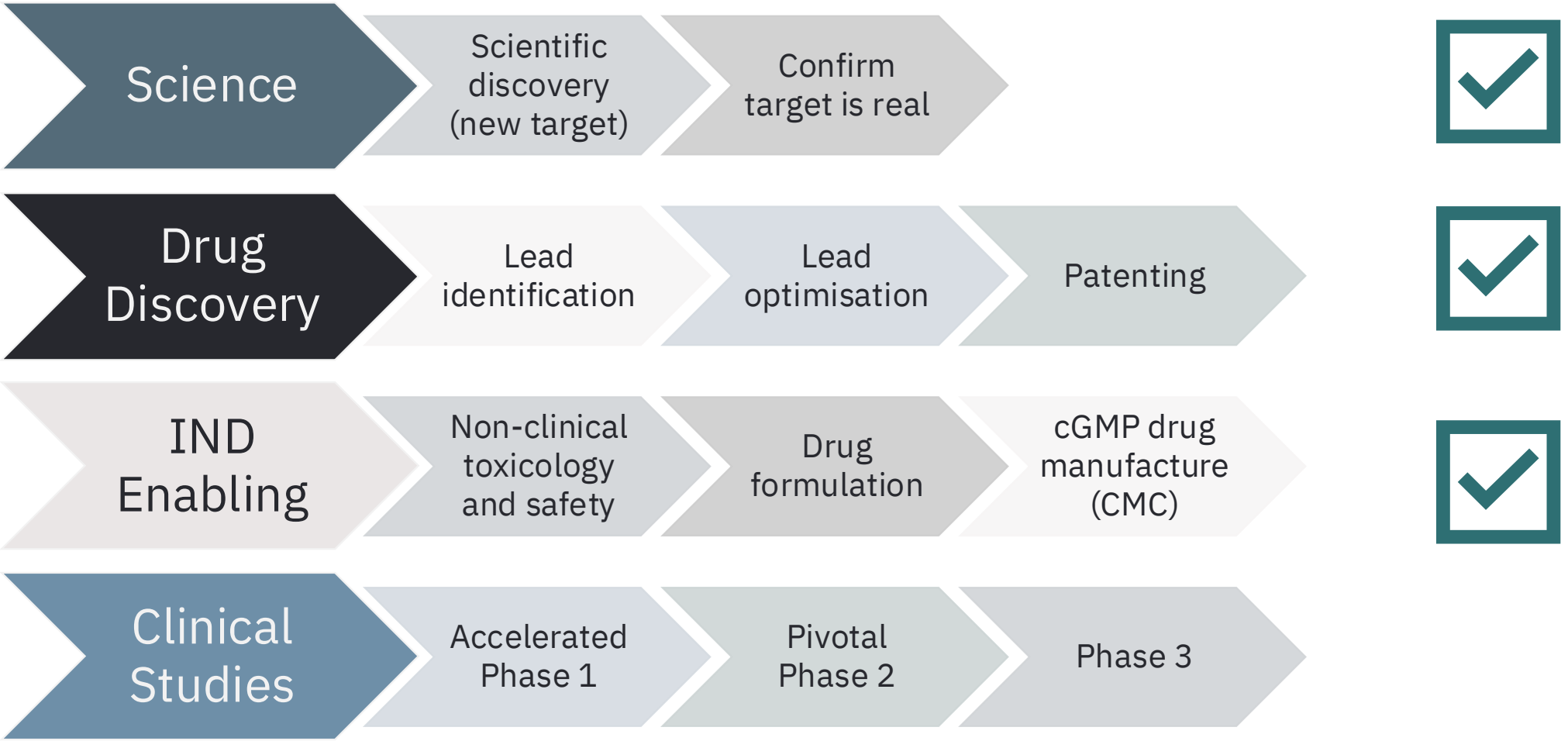


RACURA ONCOLOGY

IND, Investigational New Drug, CMC; chemistry, manufacturing and controls

Racura Oncology drug development success

Systematically solving tractable scientific and clinical problems in cost effective manner



RACURA ONCOLOGY

IND, Investigational New Drug, CMC; chemistry, manufacturing and controls

What have we accomplished at Racura Oncology?

Science

Discovered (E,E)-bisantrene and how it works

Identified where the drug is best used

- Which cancer types
- Which drug combinations

Patents

Filed new composition of matter patent based on photoisomerisation

Created a robust patent thicket covering drug composition, use, manufacturing & formulation

Up to 20 years of the strongest IP protection

IND enabling

Completed non-clinical data package

cGMP manufacturing

New drug formulation developed (RC220)

- Enabling practical intravenous administration

Clinical trials

Designed clinical program to optimise commercial value

CPACS (Phase 1)

- Cardioprotection & anticancer synergy (CPACS) with doxorubicin

HARNESS-1 (Phase 1)

- EGFRm lung cancer with osimertinib

AML (Phase 3)

- Approval pathway for RC220 & MYC targeting

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IP, intellectual property; cGMP, current good manufacturing practice; EGFRm, epidermal growth factor receptor mutant

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Science behind Racura Oncology

Prof Mike Kelso, Vice President of Research, Racura Oncology

Emeritus Prof Laurence Hurley, R. Ken Coit College of Pharmacy, University of Arizona

Agenda: Science behind Racura Oncology

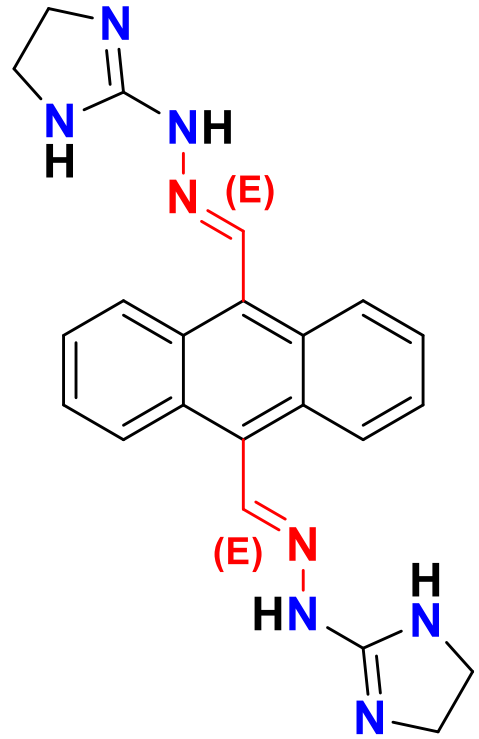
Topic	Presenter
Introduction: What are G-quadruplexes?	Prof Mike Kelso
G-quadruplex as a drug target	Emeritus Prof Laurence Hurley
Importance of MYC as a cancer driver	Prof Mike Kelso
(E,E)-bisantrene mechanism of action: MYC regulation by G-quadruplex	Prof Mike Kelso
Scientific evidence: (E,E)-bisantrene silences MYC via G-quadruplex binding	Prof Mike Kelso
Preclinical support for EGFRm NSCLC, AML & CPACS clinical programs	Prof Mike Kelso

(E,E)-bisantrene: G-quadruplex stabiliser & silencer of MYC transcription

Modern science has uncovered the mechanism of action (MOA) of (E,E)-bisantrene

(E,E)-bisantrene binds to and stabilises G-quadruplex DNA, leading to silencing of the MYC oncogene

Racura ASX Announcement: 2 Oct 2025



(E,E)-bisantrene

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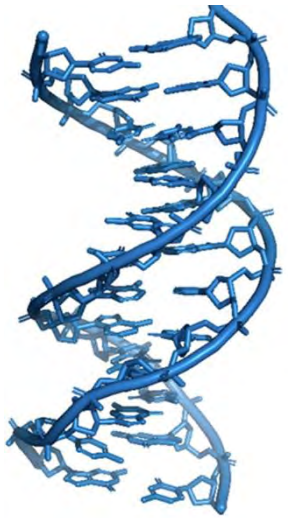
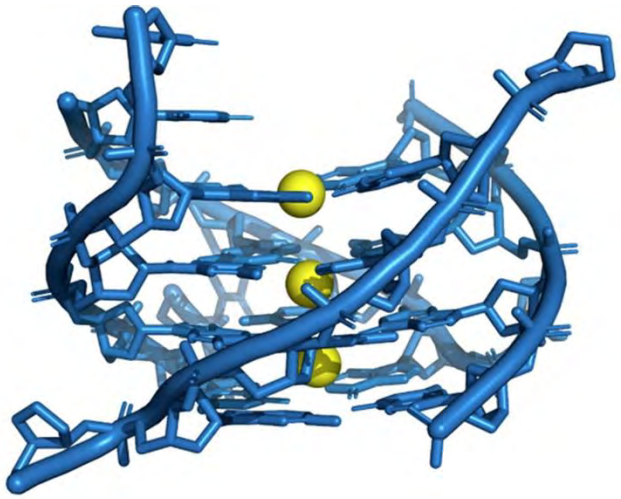
What are G-quadruplexes?

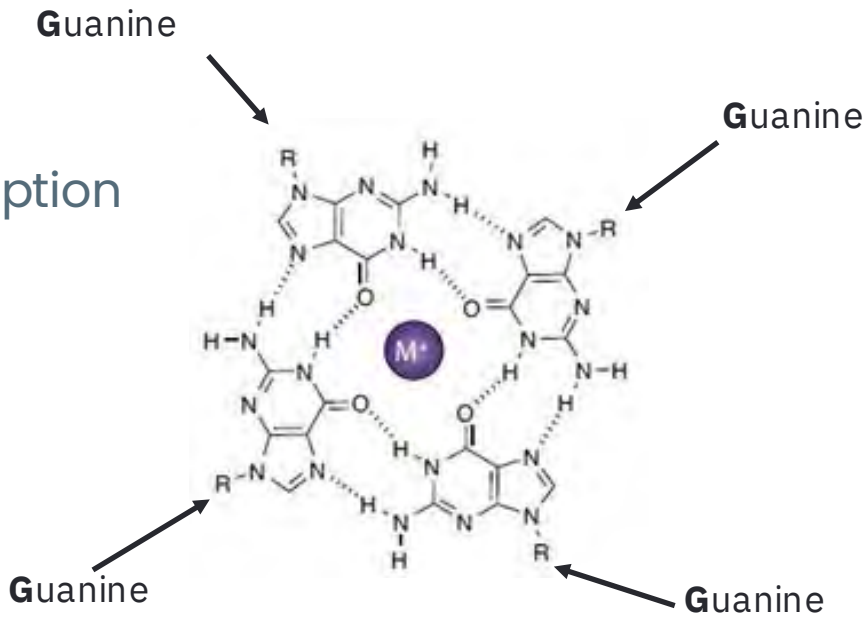
Key regulatory structures in DNA (& RNA) recognised by transcription & translation factors to regulate cell growth & division[1]



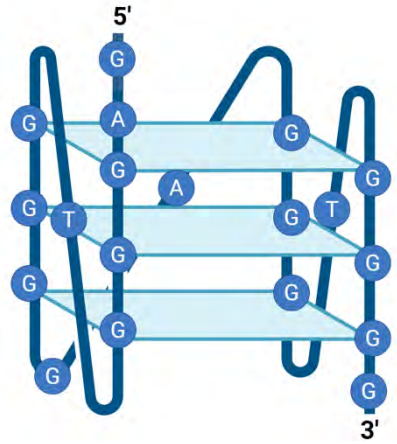
Double stranded DNA (double helix)



G-Quadruplex DNA (**G4**)



G tetrad



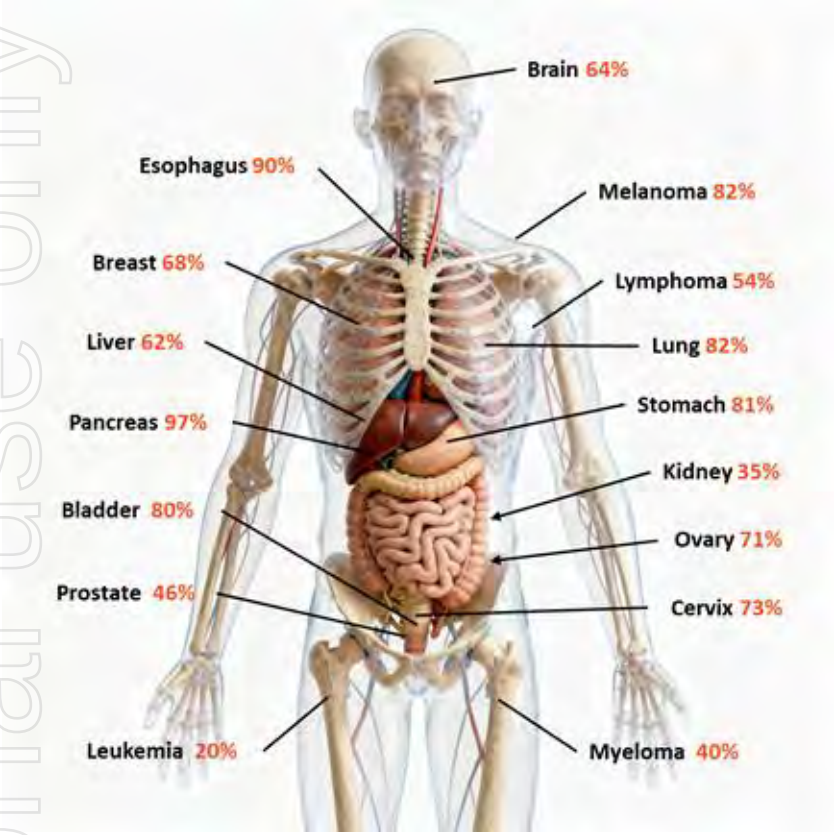
5’-GA**GGG**T**GGG**GA**GGG**T**GGG**G-3’

1. Varshney D et al (2020) Nat Rev Mol Cell, 21(8):459-474

Please scan QR code to view the video

MYC & cancer

Clinically effective MYC-targeting drugs are needed



Percentage of major cancers with elevated MYC activity[1]

MYC has long been considered the ‘holy grail’ of oncology drug targets

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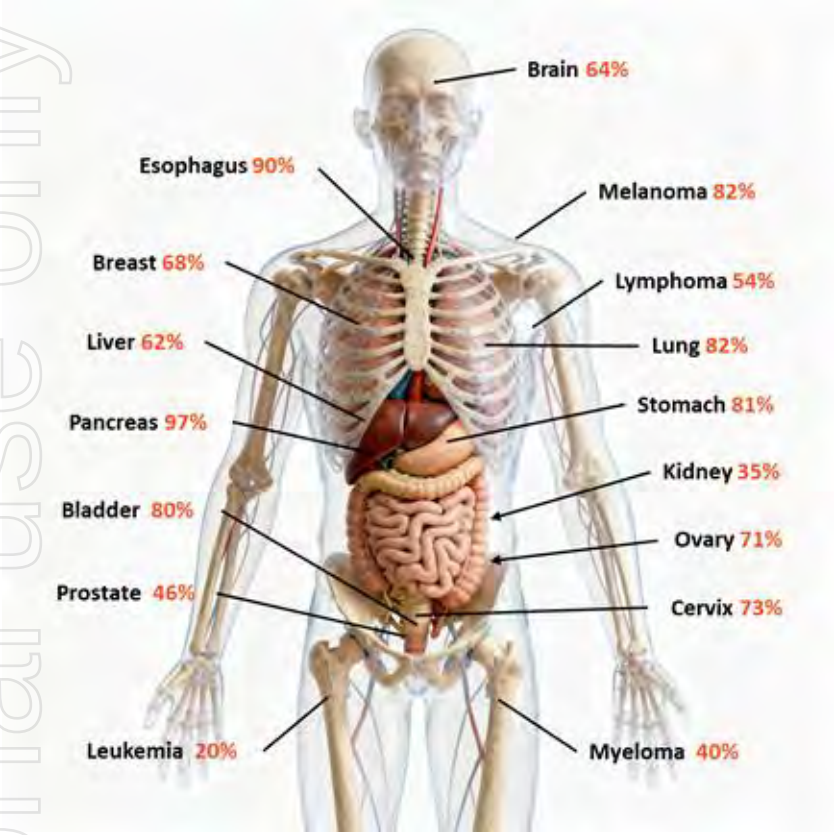
1. Data from TCGA and Schaub FX et al (2018) Cell Syst, 6(3):282–300

MYC & cancer

Clinically effective MYC-targeting drugs are needed



Percentage of major cancers with elevated MYC activity[1]

MYC is a difficult drug target

Effective MYC-targeting drugs have proven elusive

Is targeting MYC via G-quadruplex the solution?

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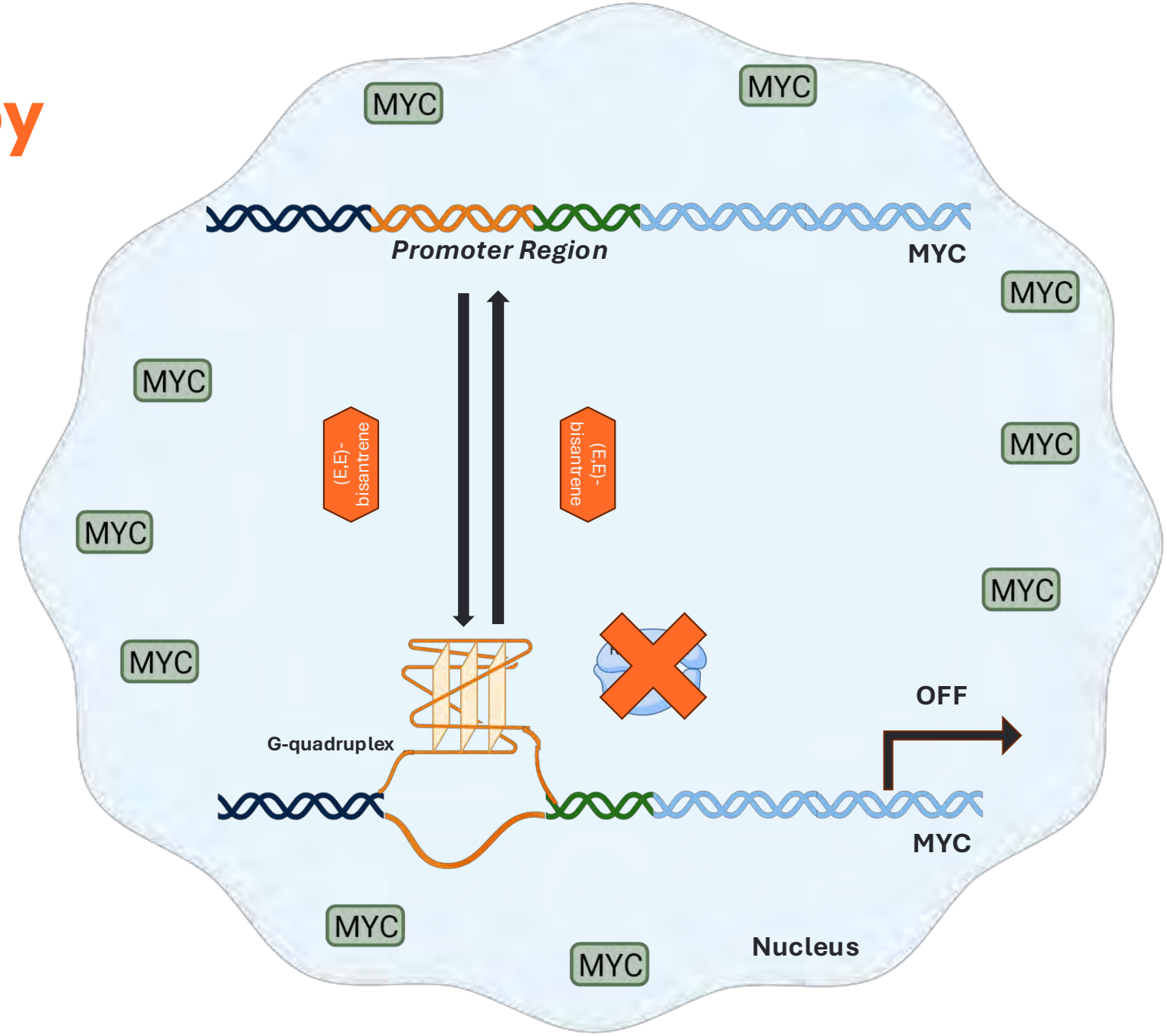
1. Data from TCGA and Schaub FX et al (2018) Cell Syst, 6(3):282–300

(E,E)-bisantrene mechanism of action video

Please scan QR code to view the video

MYC regulation by promotor region G-quadruplex

(E,E)-bisantrene silences MYC production and its ability to drive cancer progression



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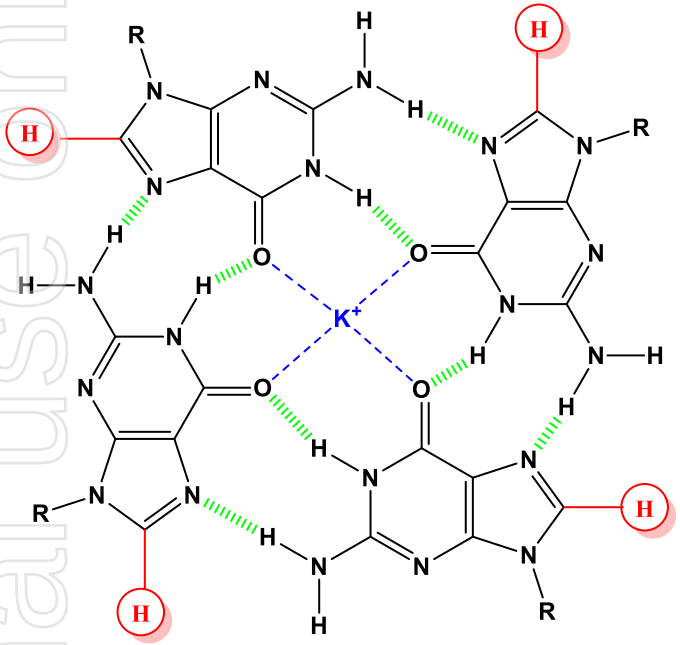
Scientific evidence

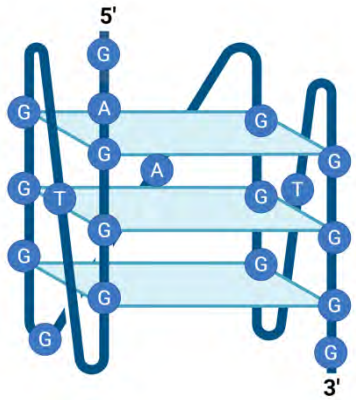
(E,E)-bisantrene silences MYC via G-quadruplex binding

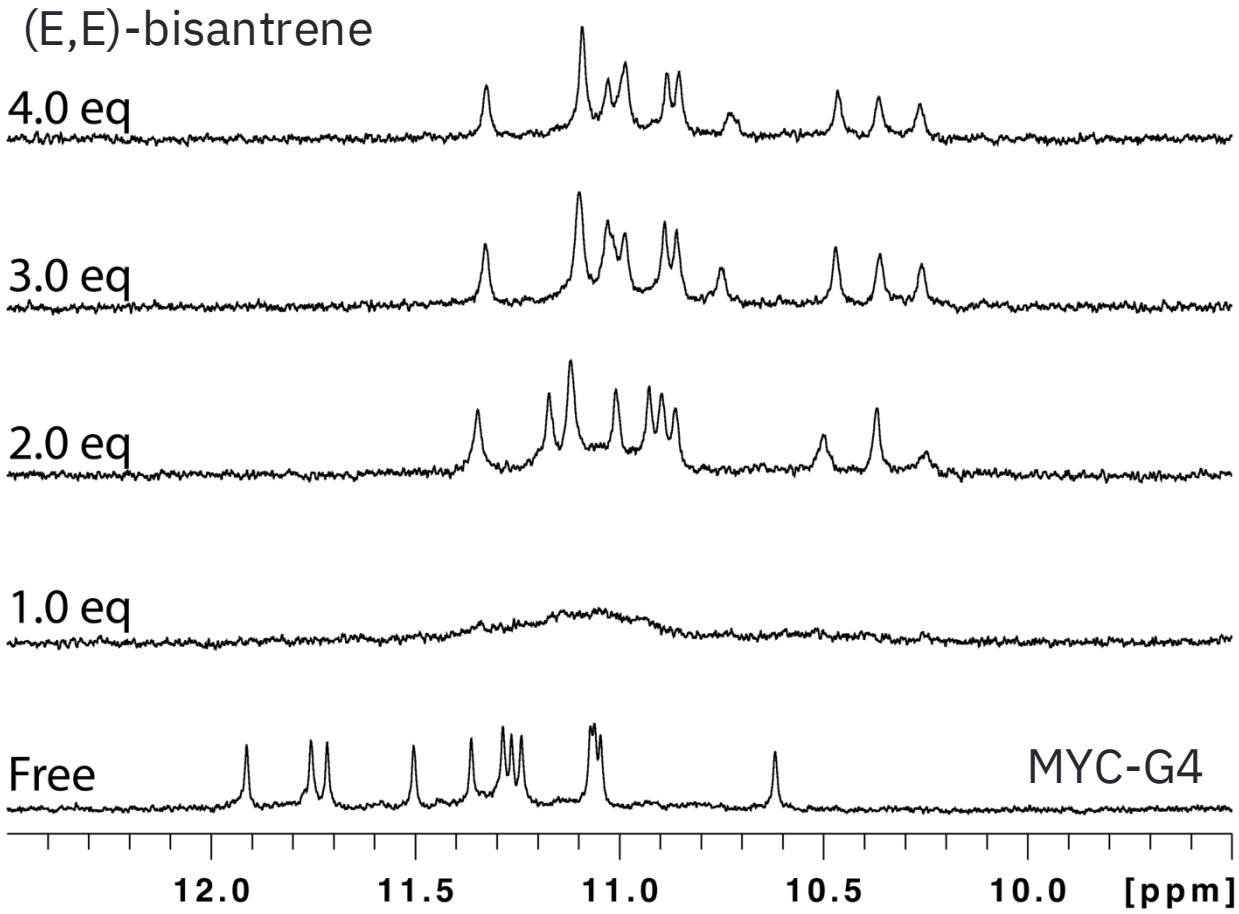
^{1}H nuclear magnetic resonance (NMR) analysis of MYC G-quadruplex binding by (E,E)-bisantrene

Two (E,E)-bisantrene molecules bind to each MYC G4 structure



G-tetrad





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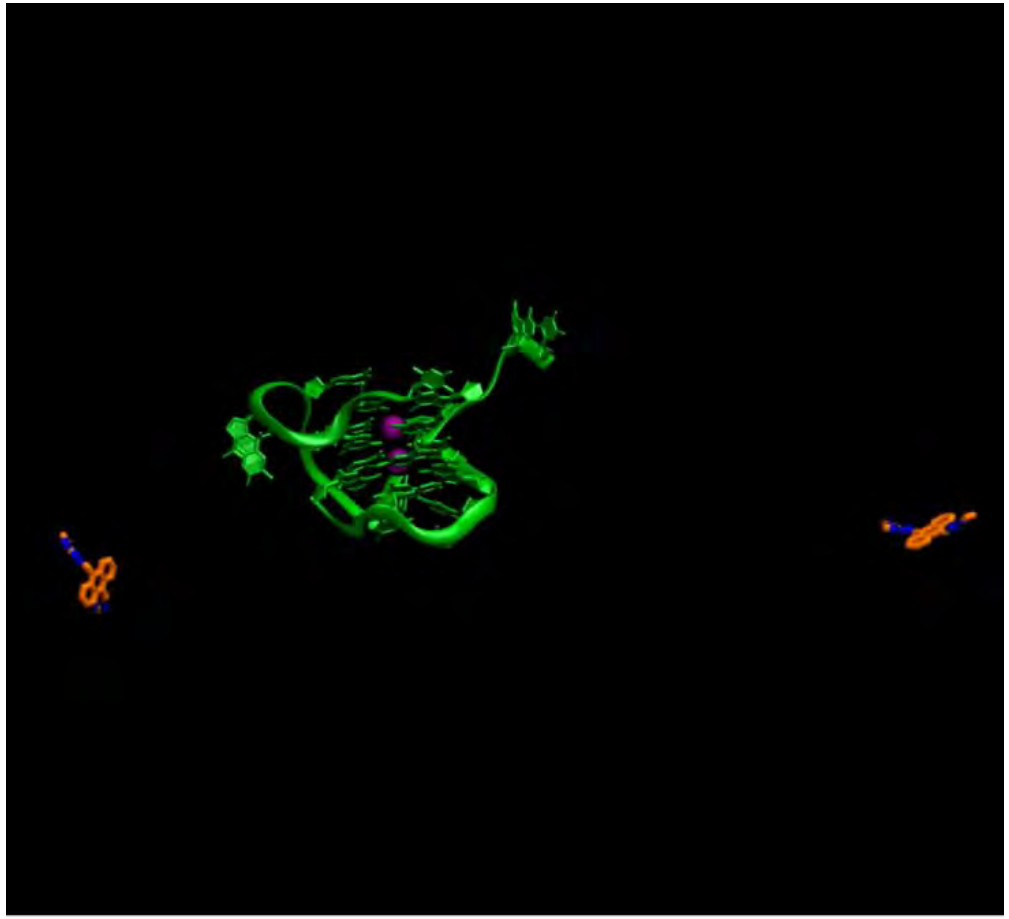
Data on file

Molecular dynamics simulation of (E,E)-bisantrene binding to MYC G-quadruplex

Simulation showing (E,E)-bisantrene seeks out and binds to G-quadruplex structures



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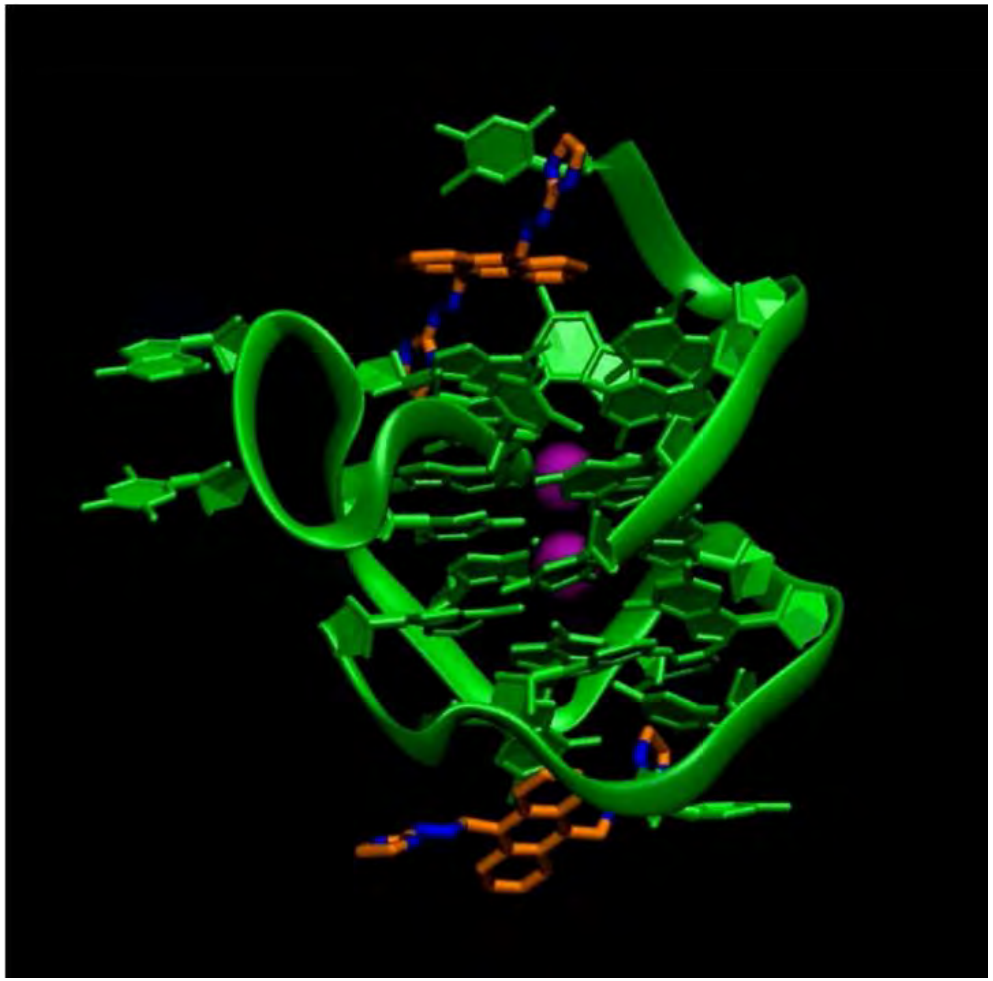
RACURA ONCOLOGY

Data on file

Molecular dynamics simulation of (E,E)-bisantrene binding to MYC G-quadruplex

Snapshot shows two (E,E)-bisantrene molecules binding to the top and bottom G-tetrads of the G-quadruplex



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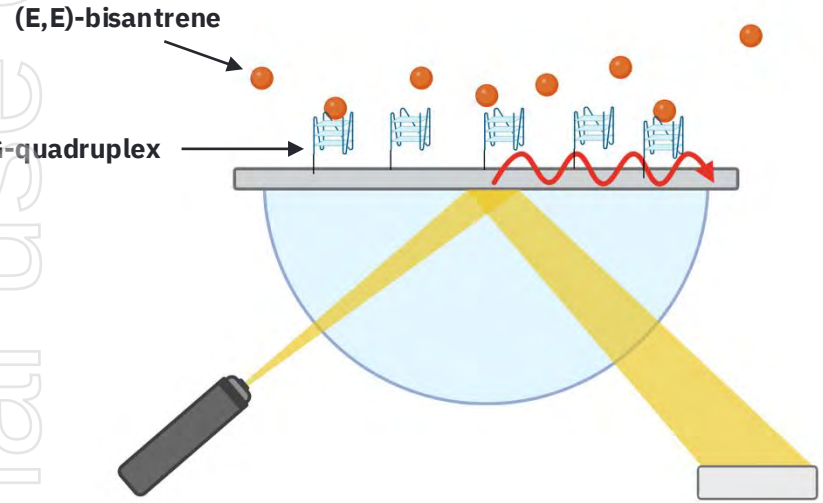
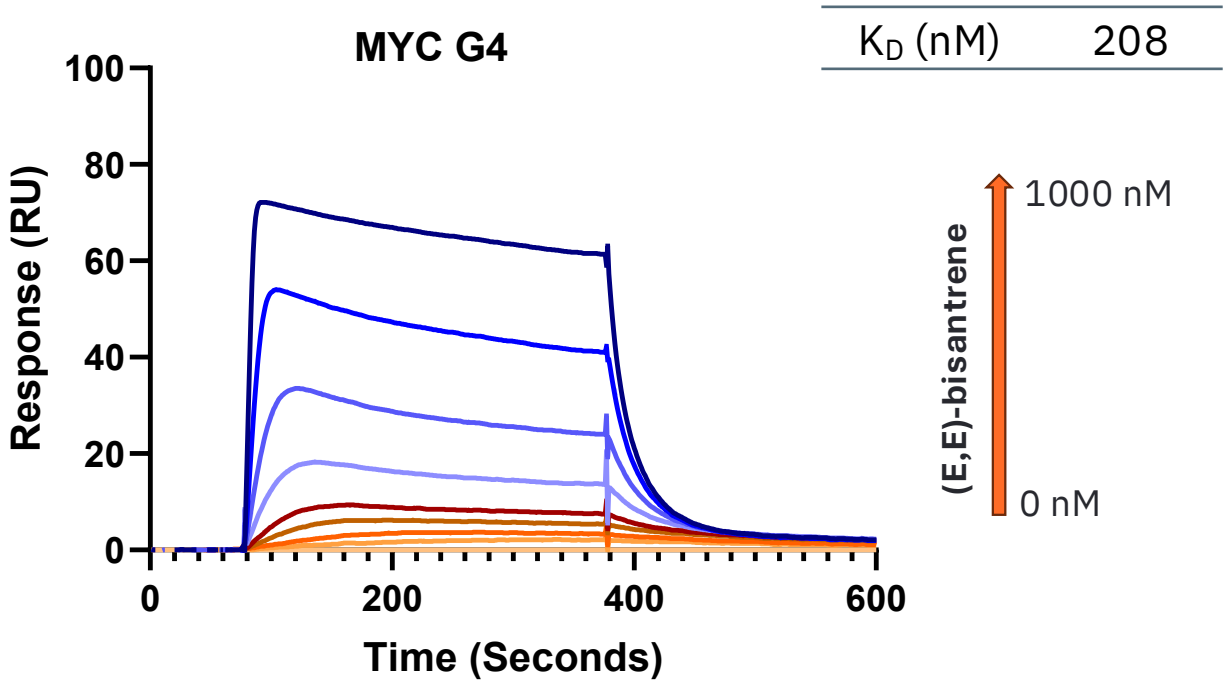
Data on file

Binding affinity of (E,E)-bisantrene to MYC G4

(E,E)-bisantrene binds the MYC G-quadruplex with high affinity at clinically relevant concentrations

Surface Plasmon Resonance spectroscopy





K_D (nM)	208

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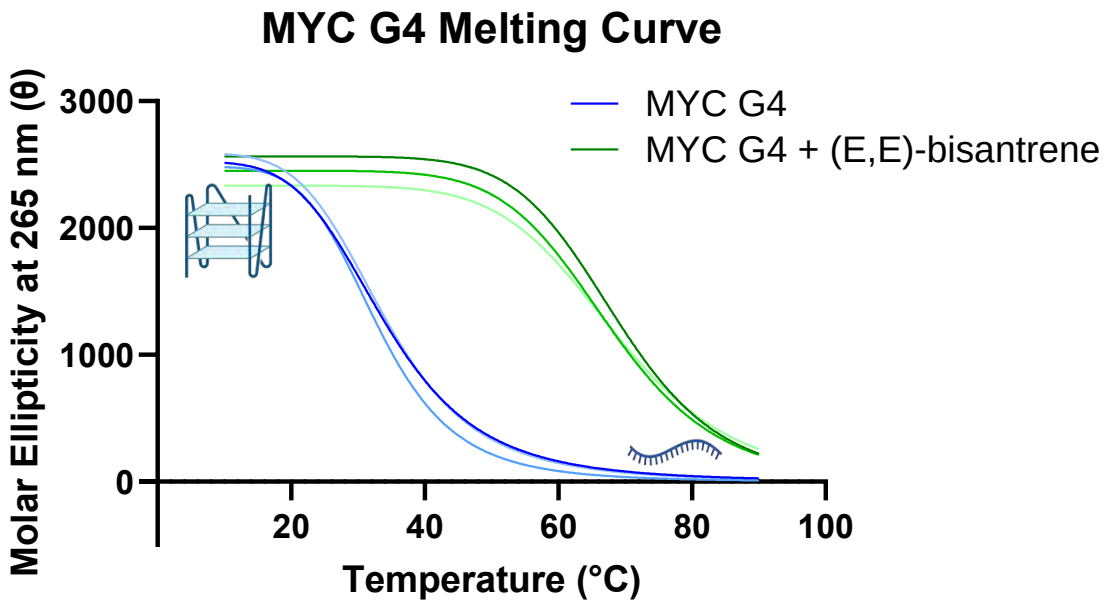
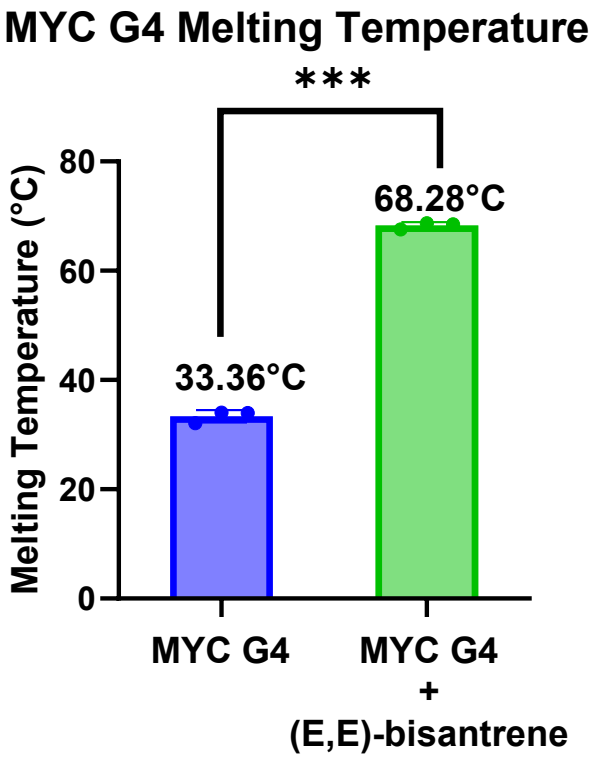
Data on file | G4, G-quadruplex

Stabilisation of MYC G4 by binding to (E,E)-bisantrene

Increase in MYC G-quadruplex melting temperature in the presence of (E,E)-bisantrene

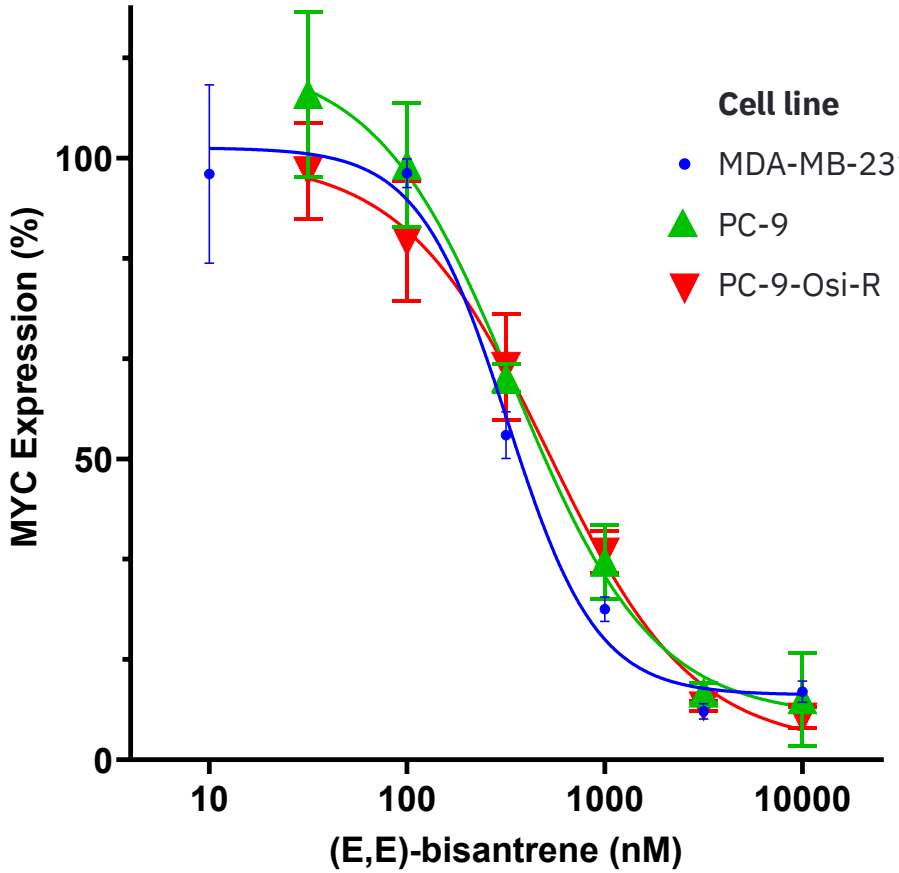
Circular dichroism spectroscopy



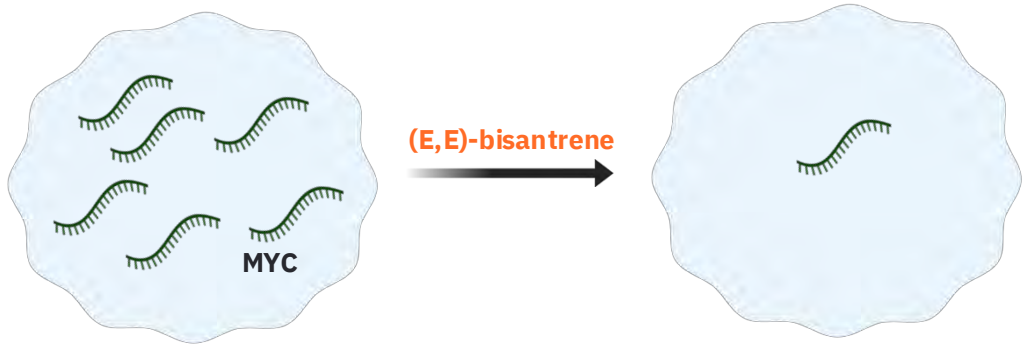


MYC gene expression analysis

(E,E)-bisantrene dose dependently silences MYC expression in human breast & lung cancer cells



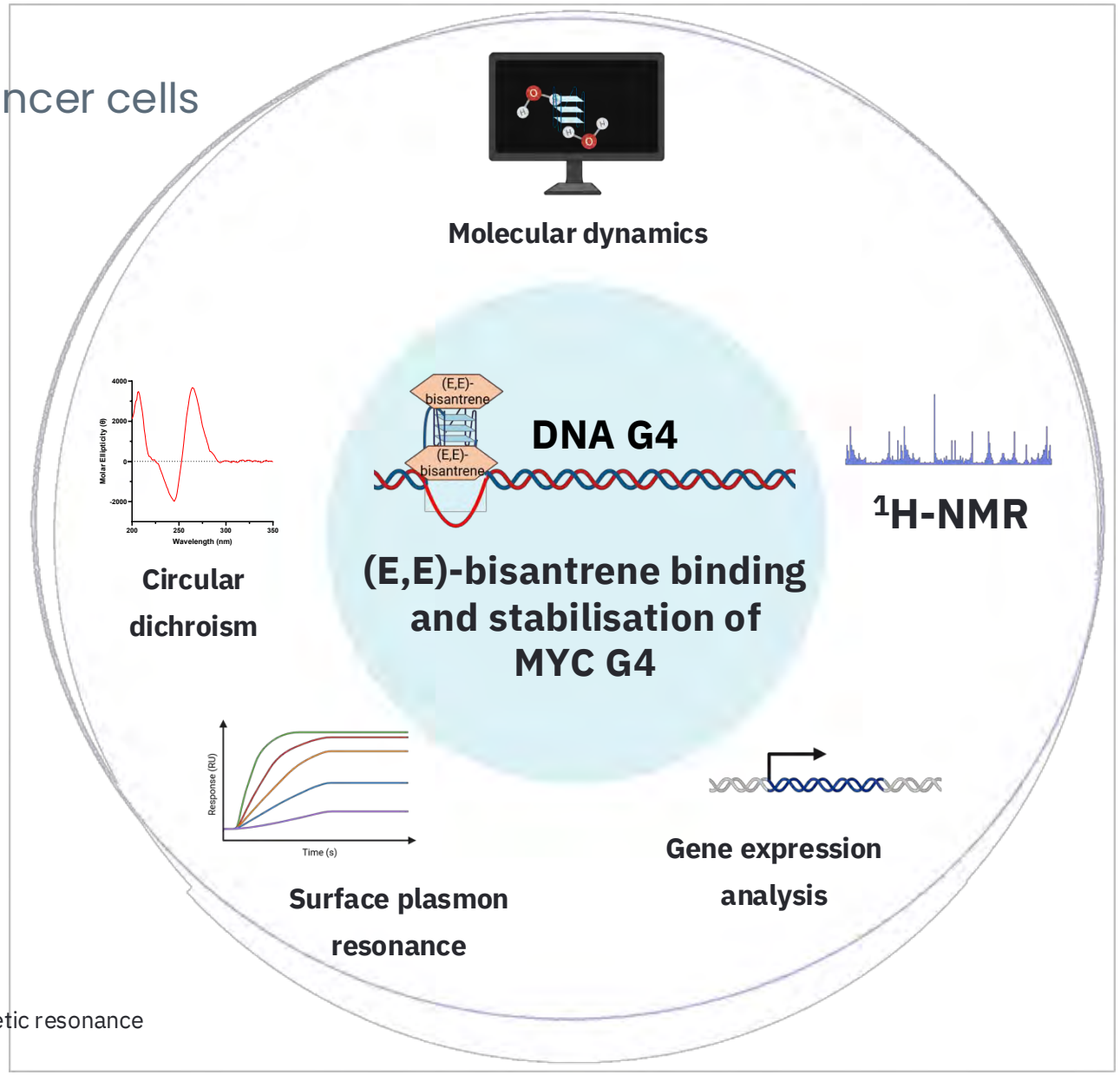
Cell line	Cancer type	EC_{50} (nM)
MDA-MB-231	Breast cancer	322 ± 43
PC-9	NSCLC	350 ± 63
PC-9-Osi-R	NSCLC	518 ± 85



Data on file | NSCLC, non-small cell lung cancer; EC_{50}, effective concentration at which half maximal effect is observed.

Evidence summary

(E,E)-bisantrene binds to and stabilises MYC G-quadruplex, silencing MYC in cancer cells



G4, G-quadruplex; ^{1}H-NMR, proton nuclear magnetic resonance

Importance of MYC in resistance to targeted therapies

MYC drives both cancer progression and resistance to targeted therapies

CANCER RESEARCH | TRANSLATIONAL SCIENCE

Targeting c-Myc to Overcome Acquired Resistance of EGFR Mutant NSCLC Cells to the Third-Generation EGFR Tyrosine Kinase Inhibitor, Osimertinib

Lei Zhu[1], Zhen Chen[1], Hongjing Zang[1,2], Songqing Fan[2], Jiajia Gu[1], Guojing Zhang[1], Kevin D.-Y. Sun[1], Qiming Wang[3], Yong He[4], Taofeek K. Owonikoko[1], Suresh S. Ramalingam[1], and Shi-Yong Sun[1]

SCIENCE ADVANCES | RESEARCH ARTICLE

CANCER

Enhanced *TP53* reactivation disrupts *MYC* transcriptional program and overcomes venetoclax resistance in acute myeloid leukemias

Yuki Nishida[1], Jo Ishizawa[1]†, Edward Ayoub[1]†, Rafael Heinz Montoya[1], Lauren B. Ostermann[1], Muharrem Muftuoglu[1], Vivian R Ruvolo[1], Tallie Patsilevas[1], Darah A. Scruggs[1], Shayaun Khazaei[1], Po Yee Mak[1], Wenjing Tao[1], Bing Z. Carter[1], Steffen Boettcher[2,3], Benjamin L. Ebert[3], Naval G. Daver[4], Marina Konopleva[4,5], Takahiko Seki[6], Kensuke Kojima[1,7], Michael Andreeff[1]*

CDDpress

www.nature.com/cddis

ARTICLE OPEN

MYC-targeted WDR4 promotes proliferation, metastasis, and sorafenib resistance by inducing CCNB1 translation in hepatocellular carcinoma

Peng Xia[1,2,3], Hao Zhang[1,2,3], Keguan Xu[1,2], Xiang Jiang[1], Meng Gao[1,2], Ganggang Wang[1,2], Yingyi Liu[1,2], Ye Yao[1,2], Xi Chen[1], Weijie Ma[1,2], Zhonglin Zhang[1,2]✉ and Yufeng Yuan[1,2]✉

JEM

ARTICLE

MYC is a clinically significant driver of mTOR inhibitor resistance in breast cancer

Targeting the PI3K–AKT–mTOR pathway is a promising therapeutic strategy for breast cancer treatment. However, low response rates and development of resistance to PI3K–AKT–mTOR inhibitors remain major clinical challenges. Here, we show that MYC activation drives resistance to mTOR inhibitors (mTORi) in breast cancer. Multiomic profiling of mouse invasive lobular carcinoma (ILC) tumors revealed recurrent *Myc* amplifications in tumors that acquired resistance to the mTORi AZD8055. MYC activation was associated with biological processes linked to mTORi response and counteracted mTORi-induced translation inhibition by promoting translation of ribosomal proteins. In vitro and in vivo induction of MYC conferred mTORi resistance in mouse and human breast cancer models. Conversely, AZD8055-resistant ILC cells depended on MYC, as demonstrated by the synergistic effects of mTORi and MYCi combination treatment. Notably, MYC status was significantly associated with poor response to everolimus therapy in metastatic breast cancer patients. Thus, MYC is a clinically relevant driver of mTORi resistance that may stratify breast cancer patients for mTOR-targeted therapies.

Park *et al. Molecular Cancer* (2024) 23:136
https://doi.org/10.1186/s12943-024-02031-w

Molecular Cancer

RESEARCH Open Access

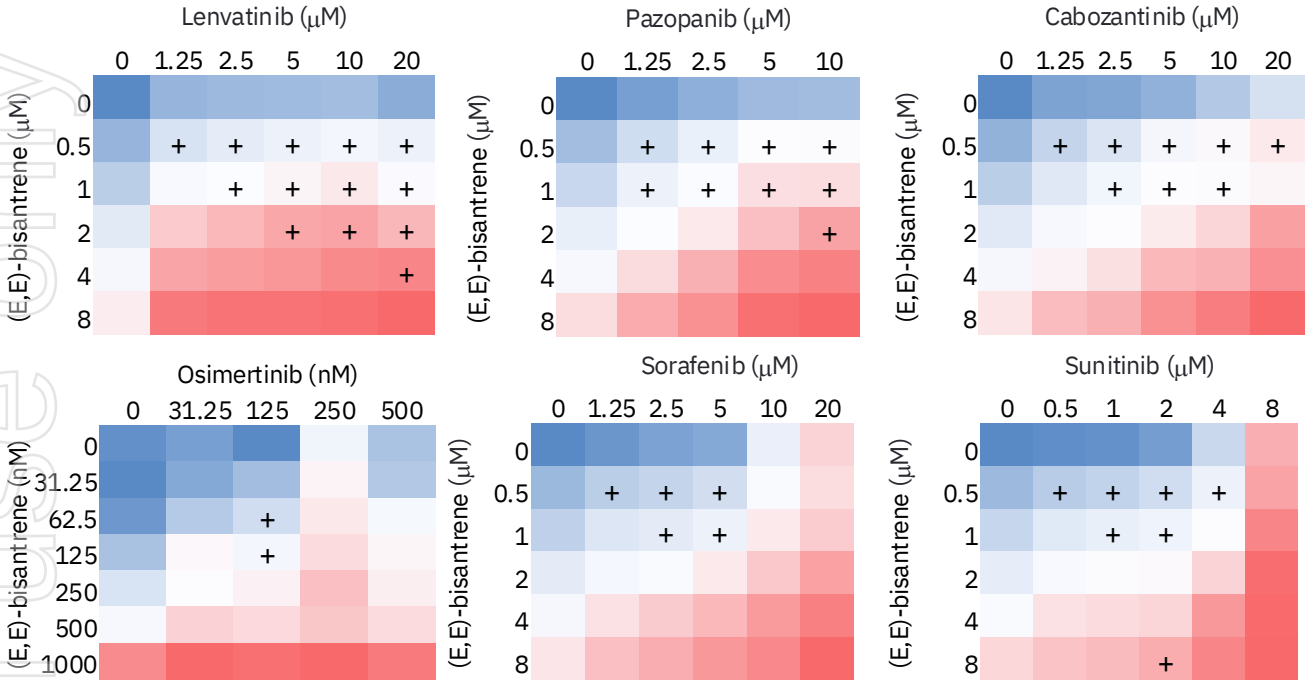
Polyamine and EIF5A hypusination downstream of c-Myc confers targeted therapy resistance in BRAF mutant melanoma

Byung-Sun Park[1,2], Heeju Jeon[1,2], Yeonseo Kim[3], Haejin Kwon[4], Ga-Eun Choi[1], Sung-Gil Chi[2], Hyun-Mee Park[4], Hyunbeom Lee[3] and Tackhoon Kim[1,2,5*]

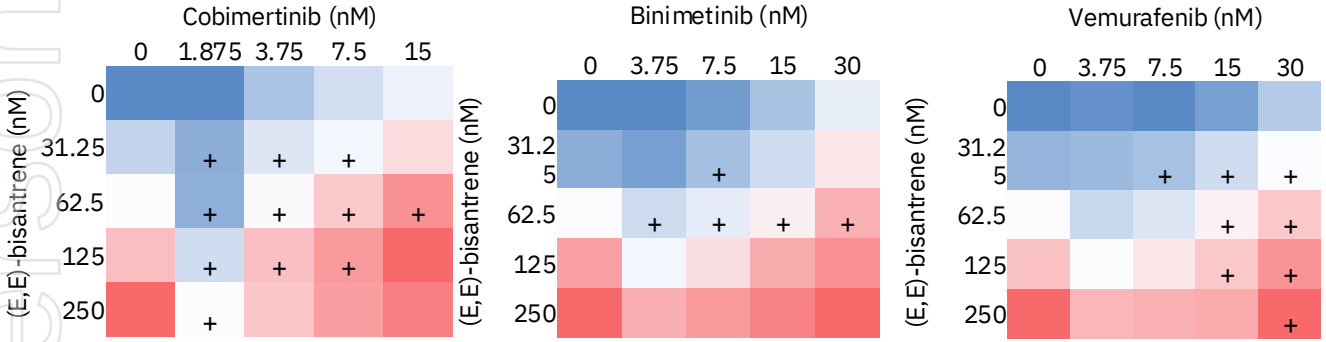
RACURA ONCOLOGY

(E,E)-bisantrene: broad synergy with targeted agents

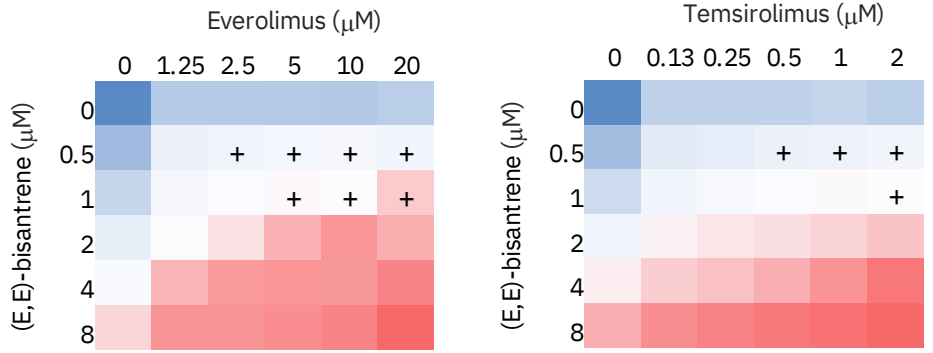
Tyrosine kinase inhibitors (TKIs) – ccRCC/NSCLC



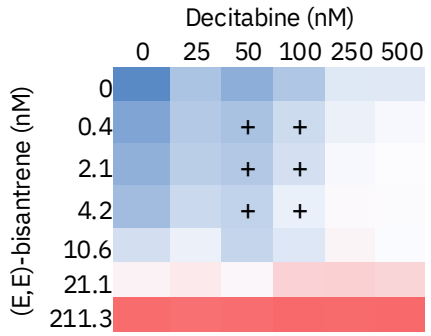
BRAF & MEK inhibitors (melanoma)



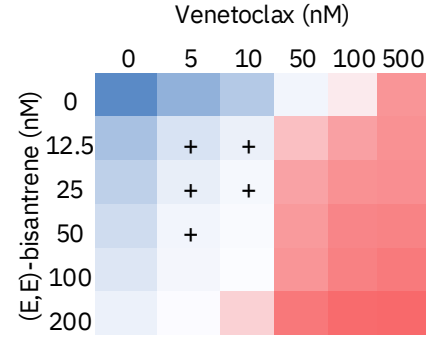
mTOR inhibitors (ccRCC)



Hypomethylating agent (AML)



BCL2 inhibitor (AML)



Data on file | ccRCC, clear cell renal cell carcinoma; NSCLC, non-small cell lung cancer; AML, acute myeloid leukaemia

Which unmet needs in oncology are MYC-driven and have the best clinical & commercial opportunity?

Acquired resistance to osimertinib in EGFRm non-small cell lung cancer

"...suggests targeting MYC as a potential strategy to overcome osimertinib acquired resistance."

Targeting c-Myc to Overcome Acquired Resistance of EGFR Mutant NSCLC Cells to the Third-Generation EGFR Tyrosine Kinase Inhibitor, Osimertinib

Lei Zhu[1], Zhen Chen[1], Hongjing Zang[1,2], Songqing Fan[2], Jiajia Gu[1], Guojing Zhang[1], Kevin D.-Y. Sun[1], Qiming Wang[3], Yong He[4], Taofeek K. Owonikoko[1], Suresh S. Ramalingam[1], and Shi-Yong Sun[1]

ABSTRACT

Osimertinib (AZD9291 or TAGRISSO) is a promising and approved third-generation EGFR tyrosine kinase inhibitor (TKI) for treating patients with advanced non–small cell lung cancer (NSCLC) harboring EGFR-activating mutations or the resistant T790M mutation. However, the inevitable emergence of acquired resistance limits its long-term efficacy. A fuller understanding of the mechanism of action of osimertinib and its linkage to acquired resistance will enable the development of more efficacious therapeutic strategies. Consequently, we have identified a novel connection between osimertinib or other EGFR-TKIs and c-Myc. Osimertinib rapidly and sustainably decreased c-Myc levels primarily via enhancing protein degradation in EGFR-mutant (EGFRm) NSCLC cell lines and xenograft tumors. c-Myc levels were substantially elevated in different EGFRm NSCLC cell lines with acquired resistance to osimertinib in comparison with their corresponding parental cell lines and could not be reduced any further by osimertinib. Consistently, c-Myc levels were elevated in the majority of EGFRm NSCLC tissues relapsed from EGFR-TKI treatment compared with their corresponding untreated baseline c-Myc levels. Suppression of c-Myc through knockdown or pharmacologic targeting with BET inhibitors restored the response of resistant cell lines to osimertinib. These findings indicate that c-Myc modulation mediates the therapeutic efficacy of osimertinib and the development of osimertinib acquired resistance. Furthermore, they establish c-Myc as a potential therapeutic target and warrant clinical testing of BET inhibition as a potential strategy to overcome acquired resistance to osimertinib or other EGFR inhibitors.

Significance: This study demonstrates a critical role of c-Myc modulation in mediating therapeutic efficacy of osimertinib including osimertinib acquired resistance and suggests targeting c-Myc as a potential strategy to overcome osimertinib acquired resistance.

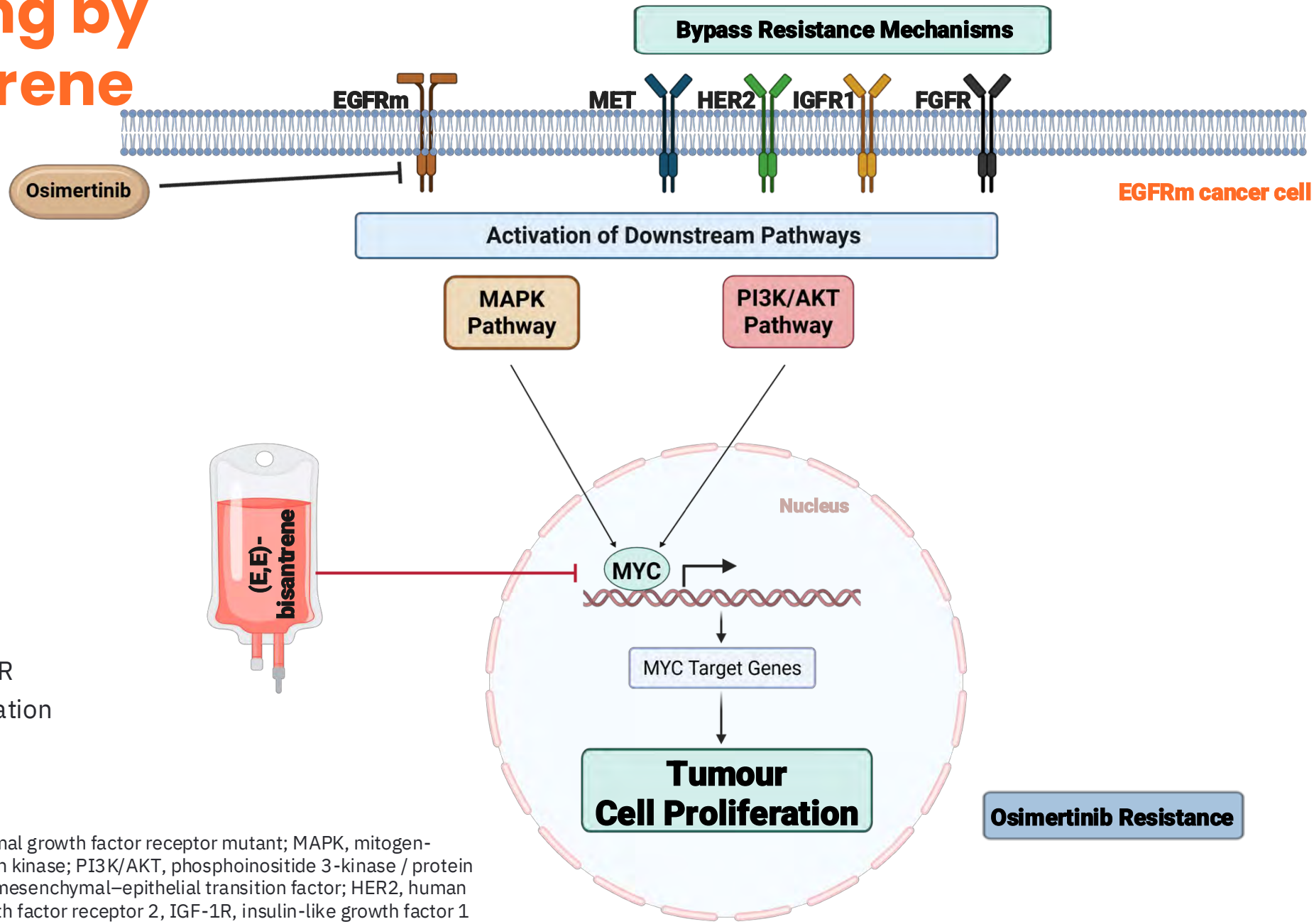
RACURA ONCOLOGY

Zhu L et al (2021) Cancer Res, 81(18):4822-4834 | EGFR, epidermal growth factor receptor mutant

RACURA ONCOLOGY

Preclinical support for EGFRm NSCLC program

MYC silencing by (E,E)-bisantrene



Please scan QR code for animation

EGFRm, epidermal growth factor receptor mutant; MAPK, mitogen-activated protein kinase; PI3K/AKT, phosphoinositide 3-kinase / protein kinase B; MET, mesenchymal–epithelial transition factor; HER2, human epidermal growth factor receptor 2, IGF-1R, insulin-like growth factor 1 receptor; FGFR, fibroblast growth factor receptor

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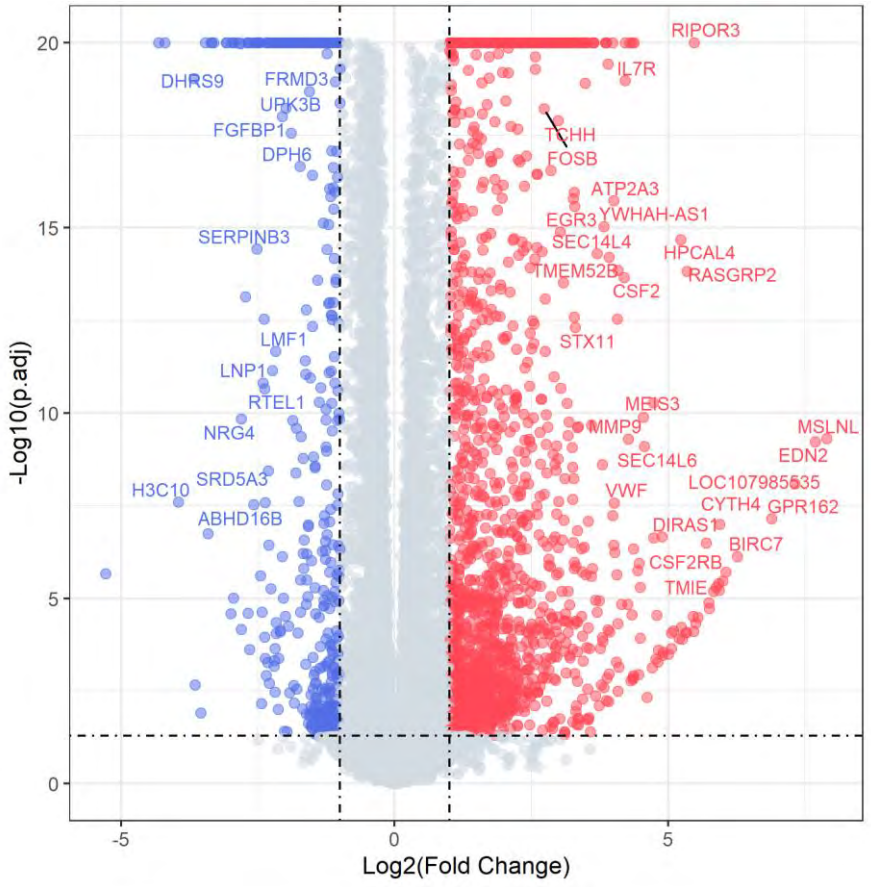
EGFRm NSCLC RNA-seq analysis

(E,E)-bisantrene upregulates (red) and downregulates (blue) many genes in lung cancer cell lines

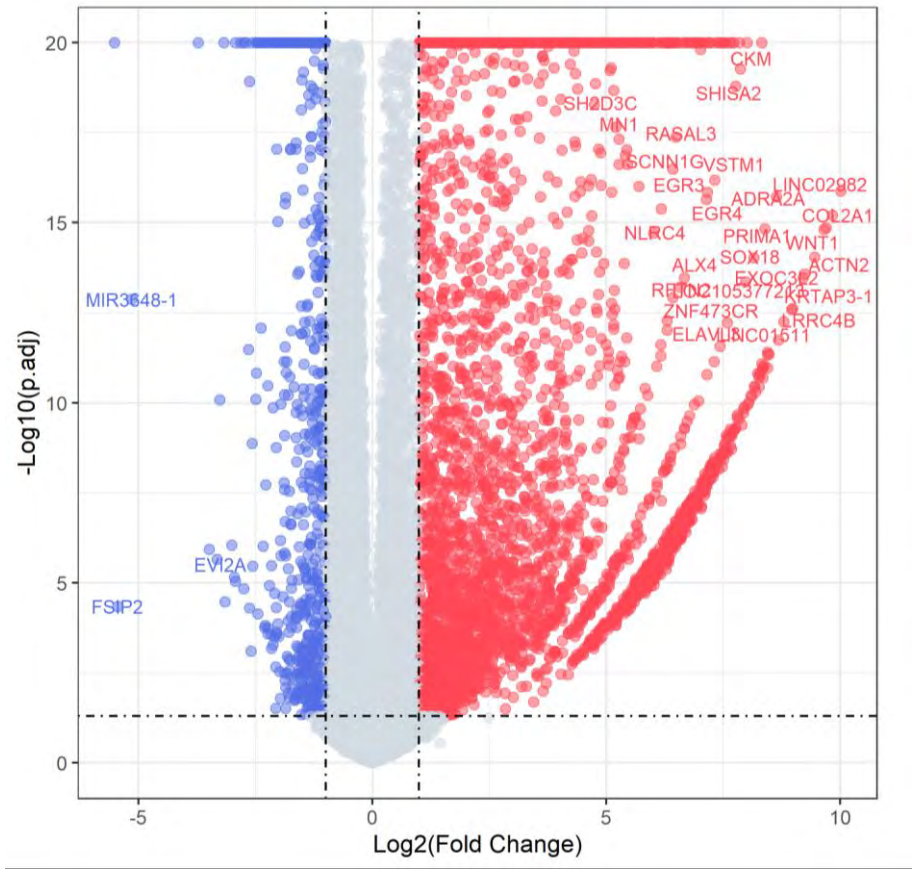
EGFRm NSCLC cell line: PC-9

(E,E)-bisantrene vs control



EGFRm NSCLC cell line: NCI-H820

(E,E)-bisantrene vs control



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Data on file | EGFRm, epidermal growth factor receptor mutant; NSCLC, non-small cell lung cancer

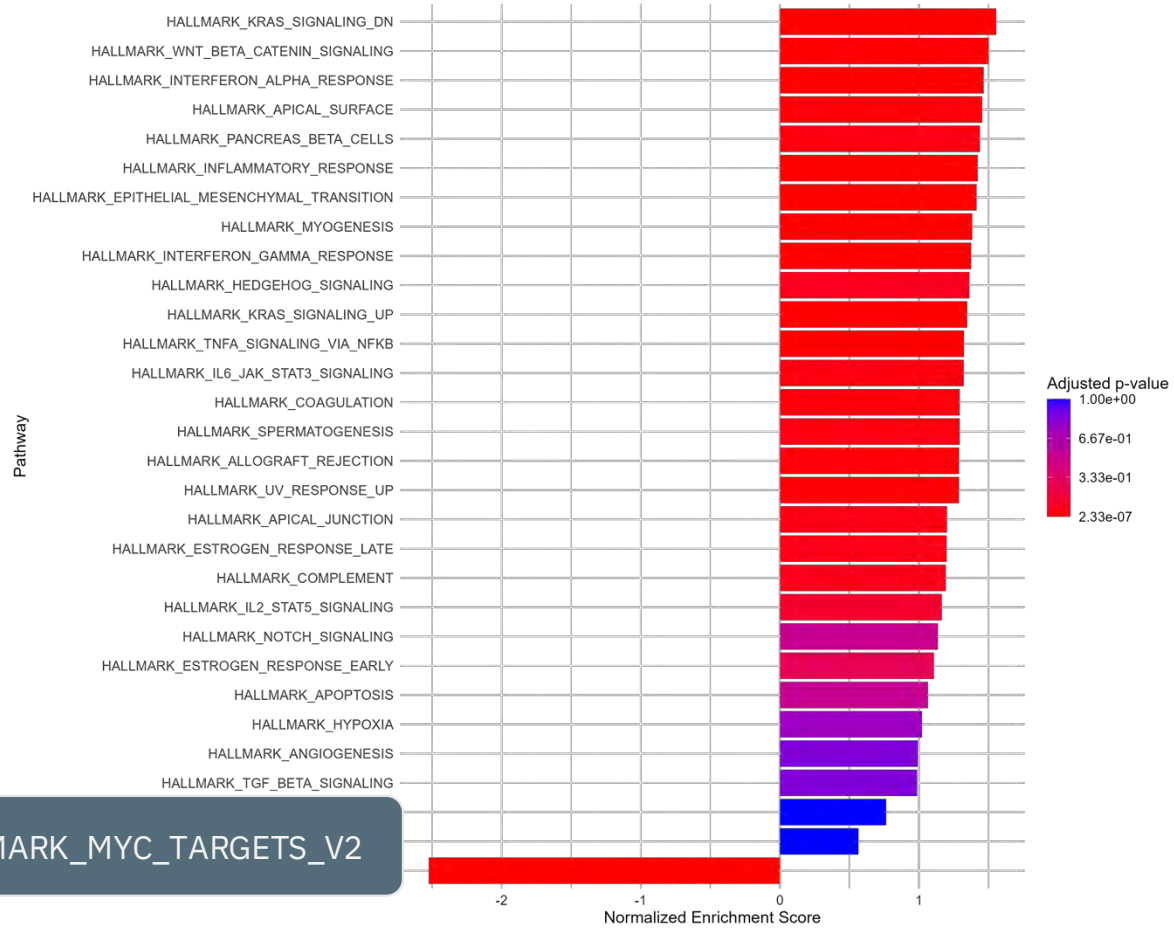
EGFRm NSCLC RNA-seq: pathway analysis

Pathways most strongly downregulated by (E,E)-bisantrene relate to MYC

EGFRm NSCLC cell line: PC-9

HALLMARK_MYC_TARGETS_V2
HALLMARK_MYC_TARGETS_V1

EGFRm NSCLC cell line: NCI-H820



HALLMARK_MYC_TARGETS_V2

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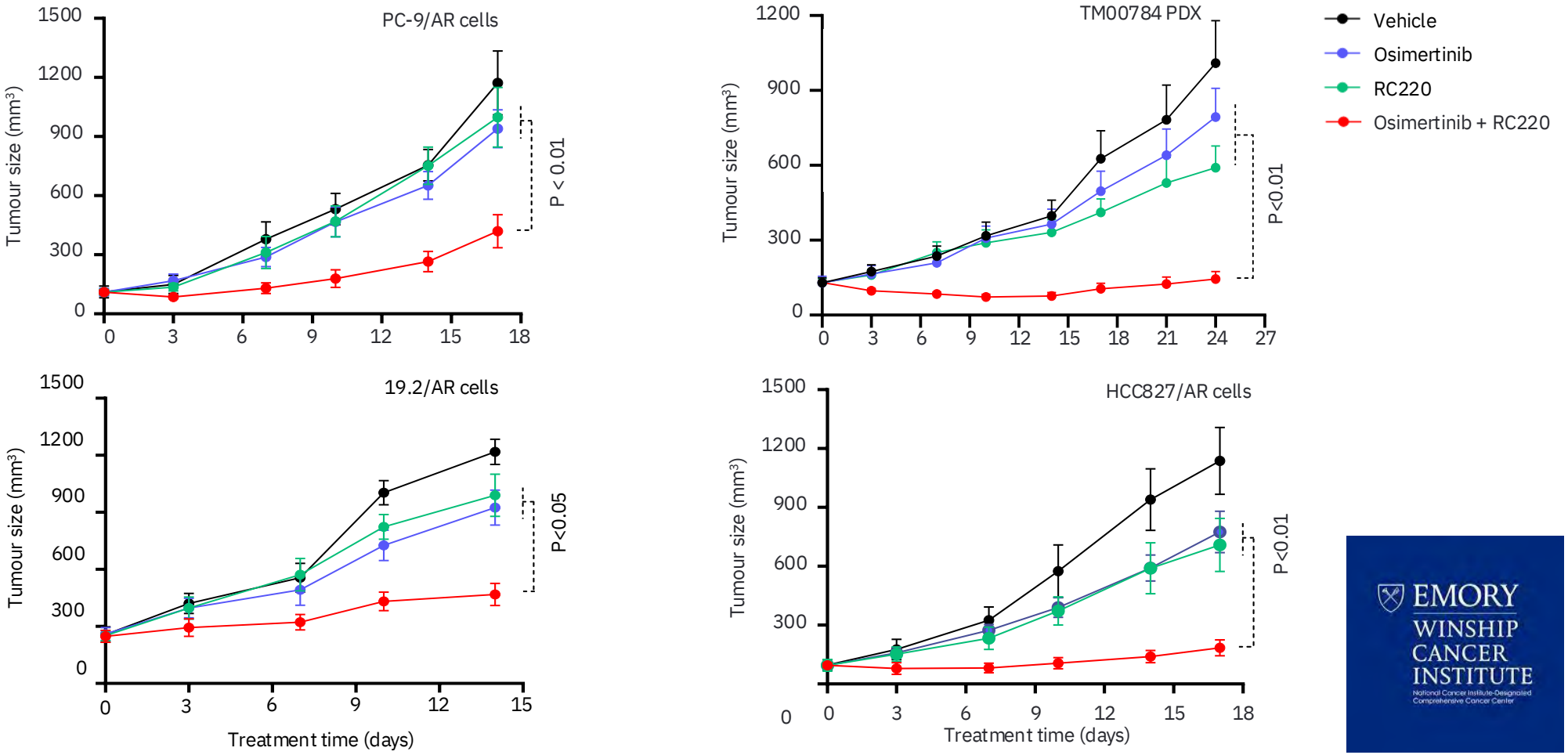
Data on file | EGFRm, epidermal growth factor receptor mutant; NSCLC, non-small cell lung cancer

RC220 highly active in combination with osimertinib

RC220 restores sensitivity to osimertinib in osimertinib-resistant EGFRm NSCLC models

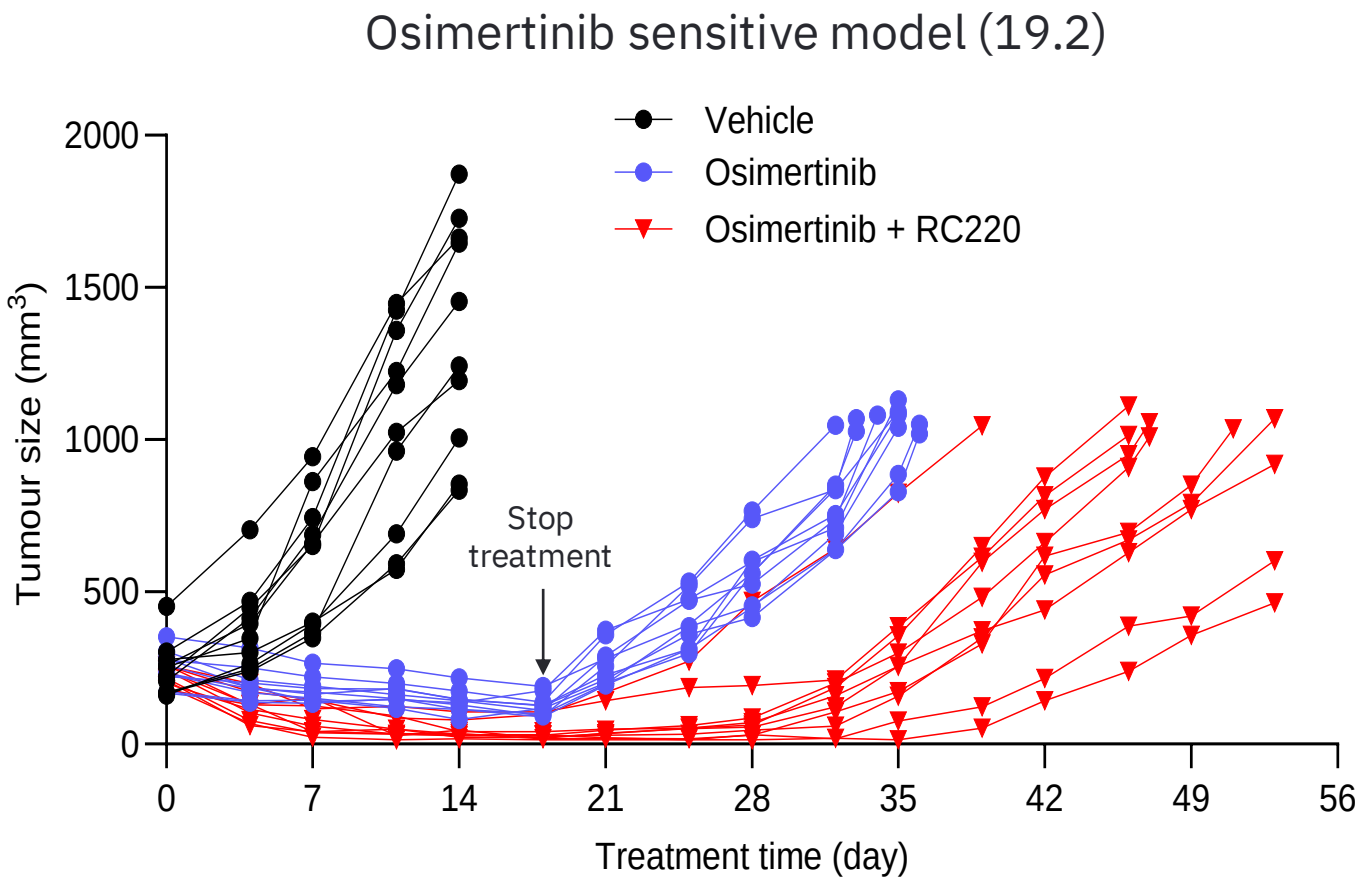
Osimertinib resistant models



Data on file | EGFRm, epidermal growth factor receptor mutant; NSCLC, non-small cell lung cancer; PDX, patient-derived xenograft

RC220 highly active in combination with osimertinib

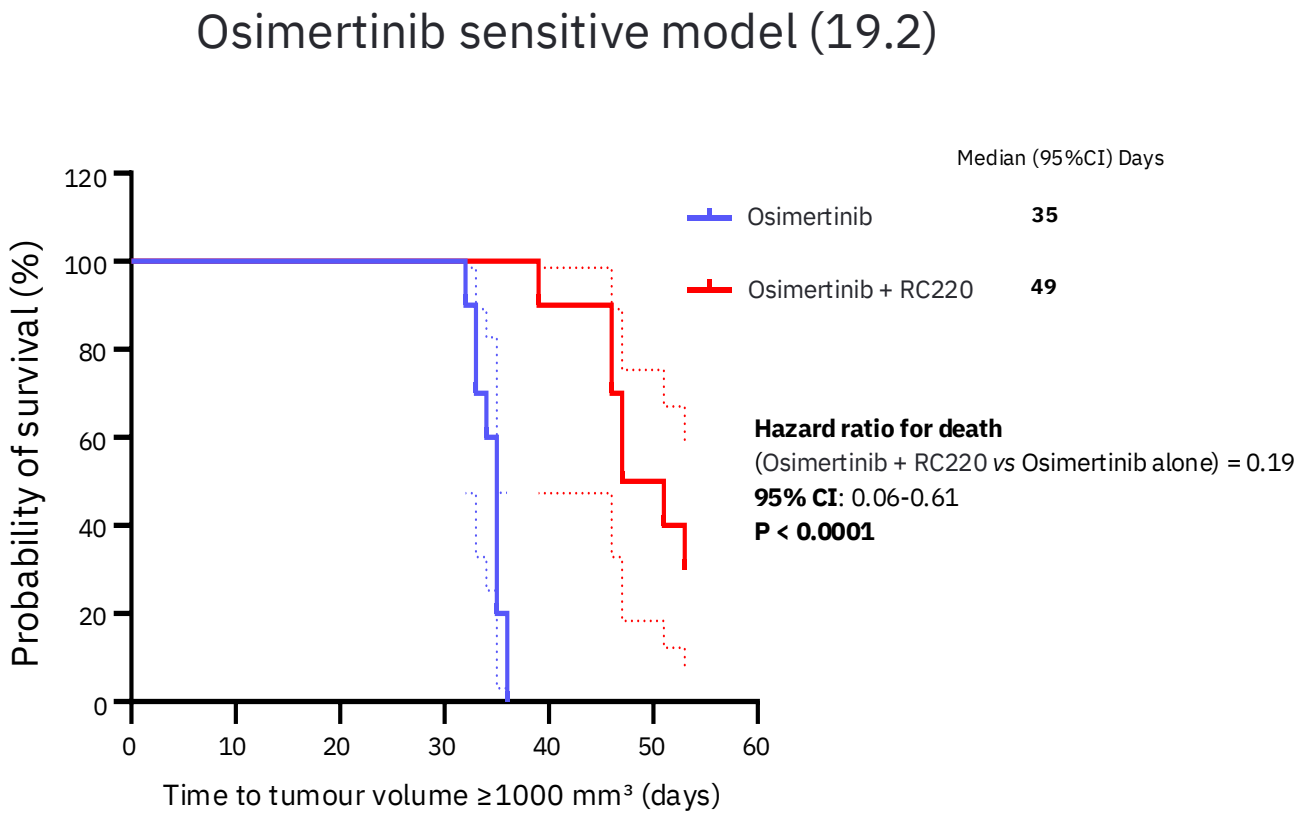
RC220-osimertinib combination delayed relapse in EGFRm NSCLC mouse models



Data on file | EGFRm, epidermal growth factor receptor mutant; NSCLC, non-small cell lung cancer

RC220 highly active in combination with osimertinib

RC220-osimertinib combination increased survival in EGFRm NSCLC mouse models



RACURA ONCOLOGY

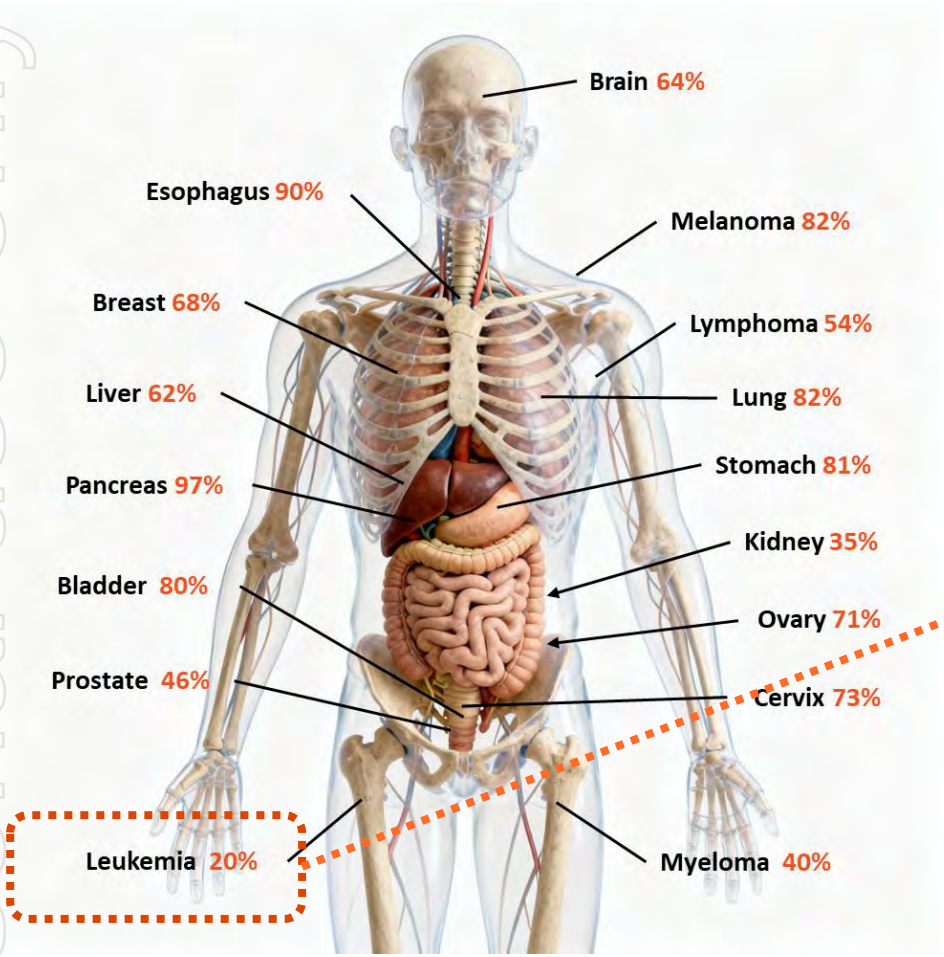
Data on file | EGFRm, epidermal growth factor receptor mutant; NSCLC, non-small cell lung cancer

RACURA ONCOLOGY

Preclinical support for AML program

MYC dependence in AML

AML is one of the most MYC driven of all cancers

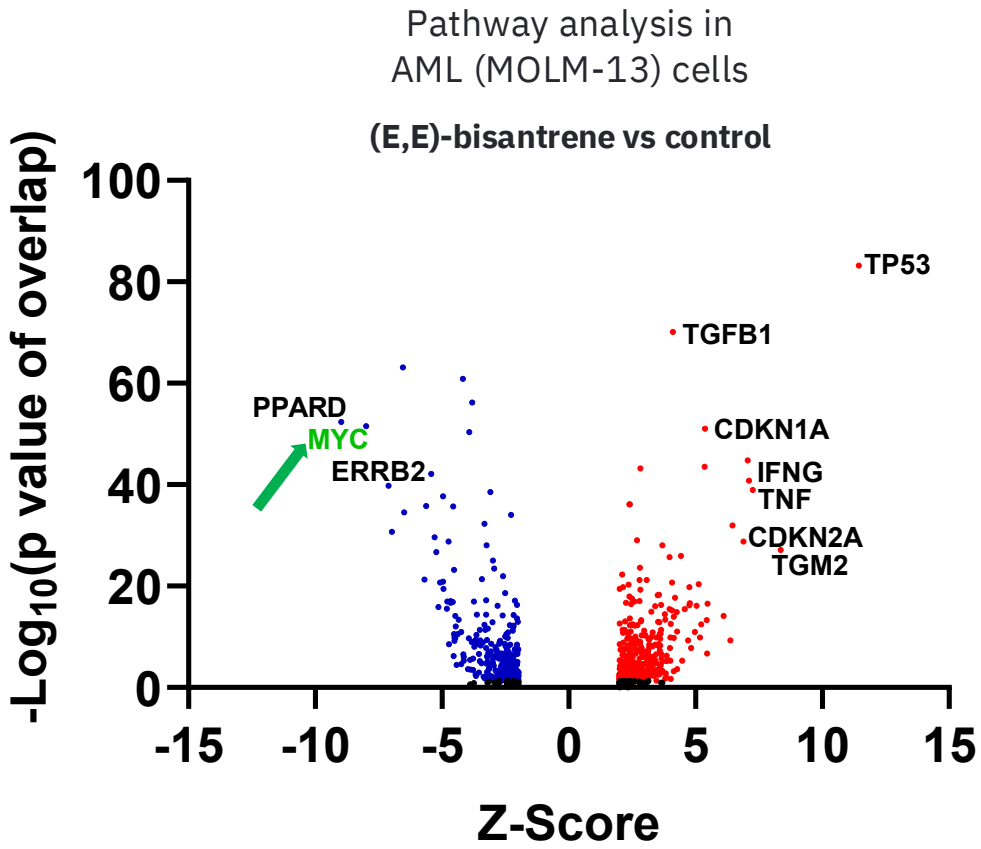


- **Acute myeloid leukaemia ~90%**
- Burkitts lymphoma: >90%
- Plasmablastic lymphoma: ~50%
- B-cell acute lymphoblastic leukaemia
  - General/adult: ~5%
  - Paediatric/relapse: 30-65%
- T-cell acute lymphoblastic leukaemia: >50%
- Chronic lymphoblastic leukaemia: <10%
- Multiple myeloma: 67%

Data from TCGA and Schaub FX et al (2018) Cell Syst, 6(3):282–300.

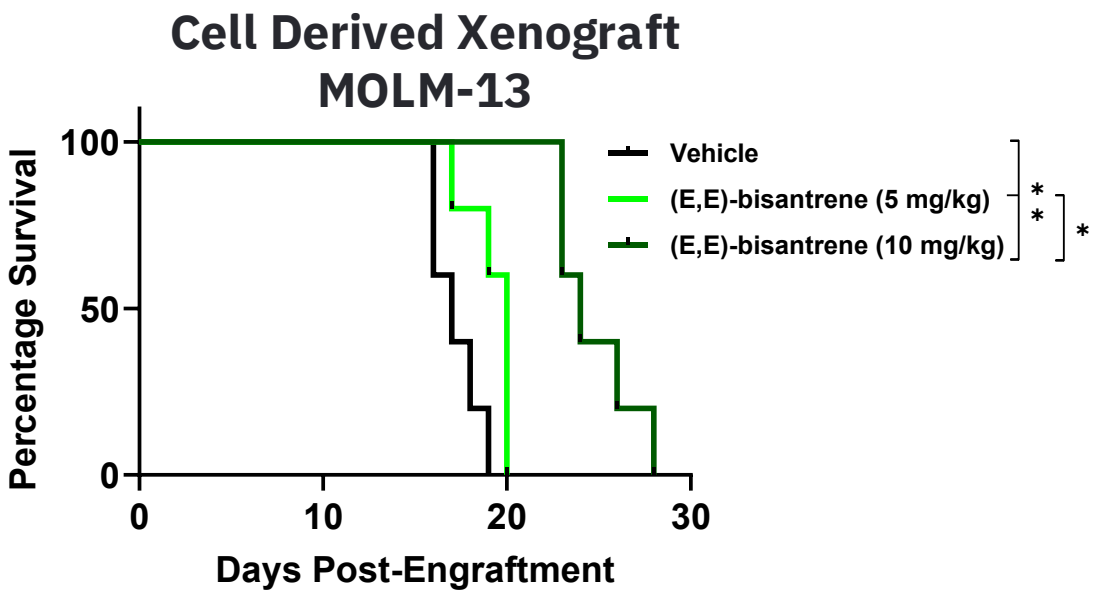
AML RNA-sequencing

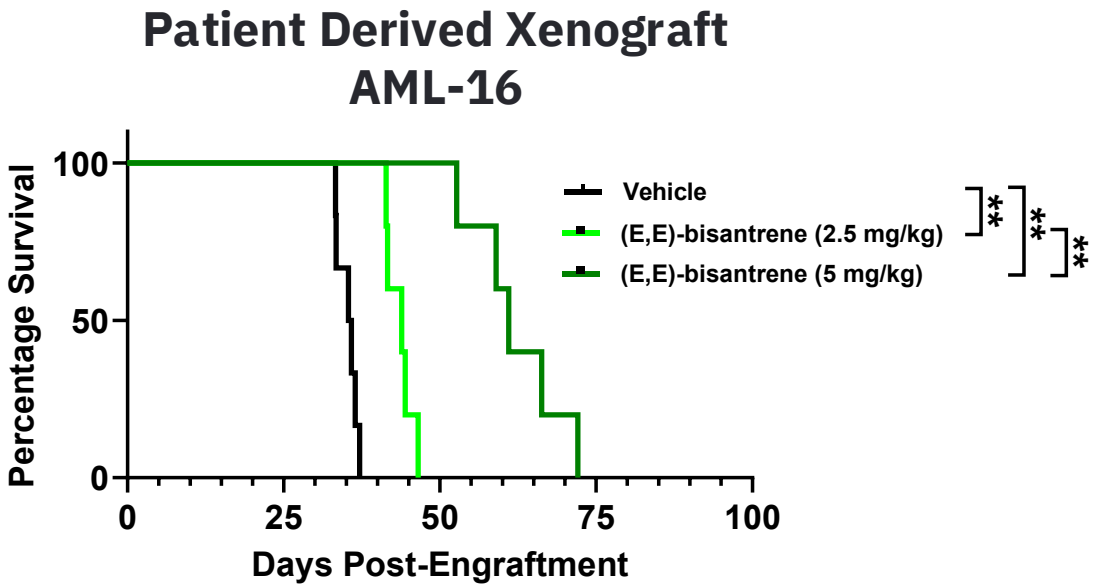
(E,E)-bisantrene potently suppressed MYC-mediated downstream pathways in AML cells



(E,E)-bisantrene is highly effective in AML

Significant increase in survival observed after (E,E)-bisantrene treatment in mouse AML models





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Data on file | * $p < 0.05$; ** $p < 0.01$ | AML, acute myeloid leukaemia

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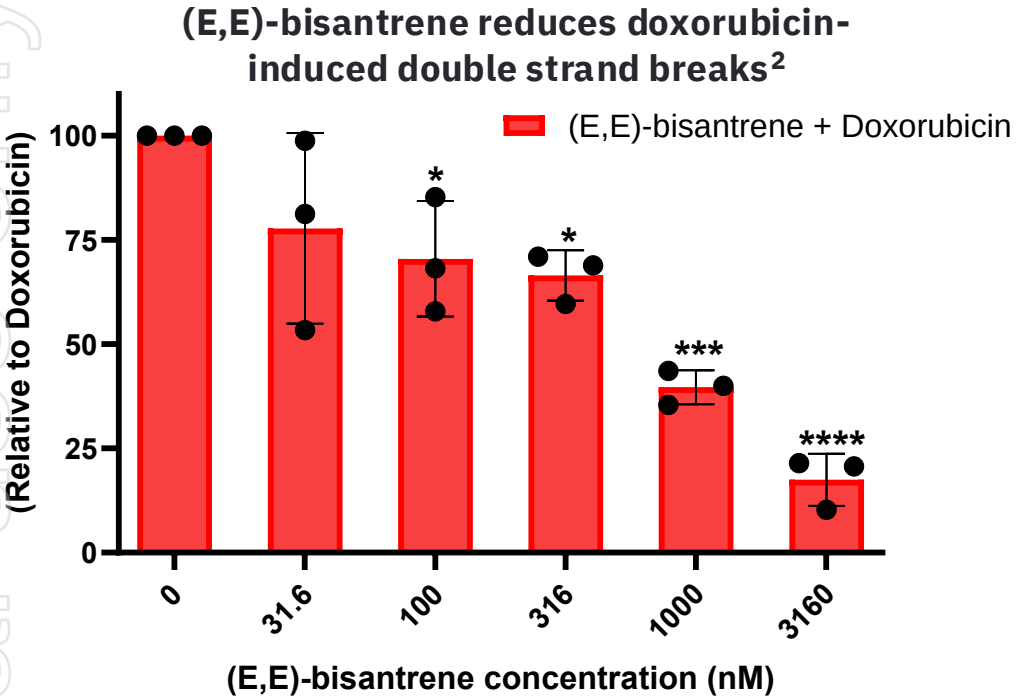
Preclinical support for CPACS program

How does MYC relate to (E,E)-bisantrene cardioprotection?

Molecular Cell paper (2022) suggests role for MYC in cardioprotection[1]



CellPress
OPEN ACCESS

Molecular Cell

Article

MYC assembles and stimulates topoisomerases 1 and 2 in a "topoisome"

Subhendu K. Das,[1,8] Vladislav Kuzin,[2,8] Donald P. Cameron,[2] Suzanne Sanford,[1] Rajiv Kumar Jha,[1] Zuqin Nie,[1] Marta Trullols Rosello,[2] Ronald Holewinski,[3] Thorkell Andresson,[3] Jan Wisniewski,[4] Toyoaki Natsume,[5,6] David H. Price,[7] Brian A. Lewis,[1] Fedor Kouzine,[1] David Levens,[1,8,*] and Laura Baranello[2,8,9,*]

[1]Laboratory of Pathology, National Cancer Institute, Bethesda, MD 20814, USA
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[7]Department of Biochemistry, University of Iowa, Iowa City, IA 52242, USA
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*Correspondence: levensd@mail.nih.gov (D.L.), laura.baranello@ki.se (L.B.)
https://doi.org/10.1016/j.molcel.2021.11.016

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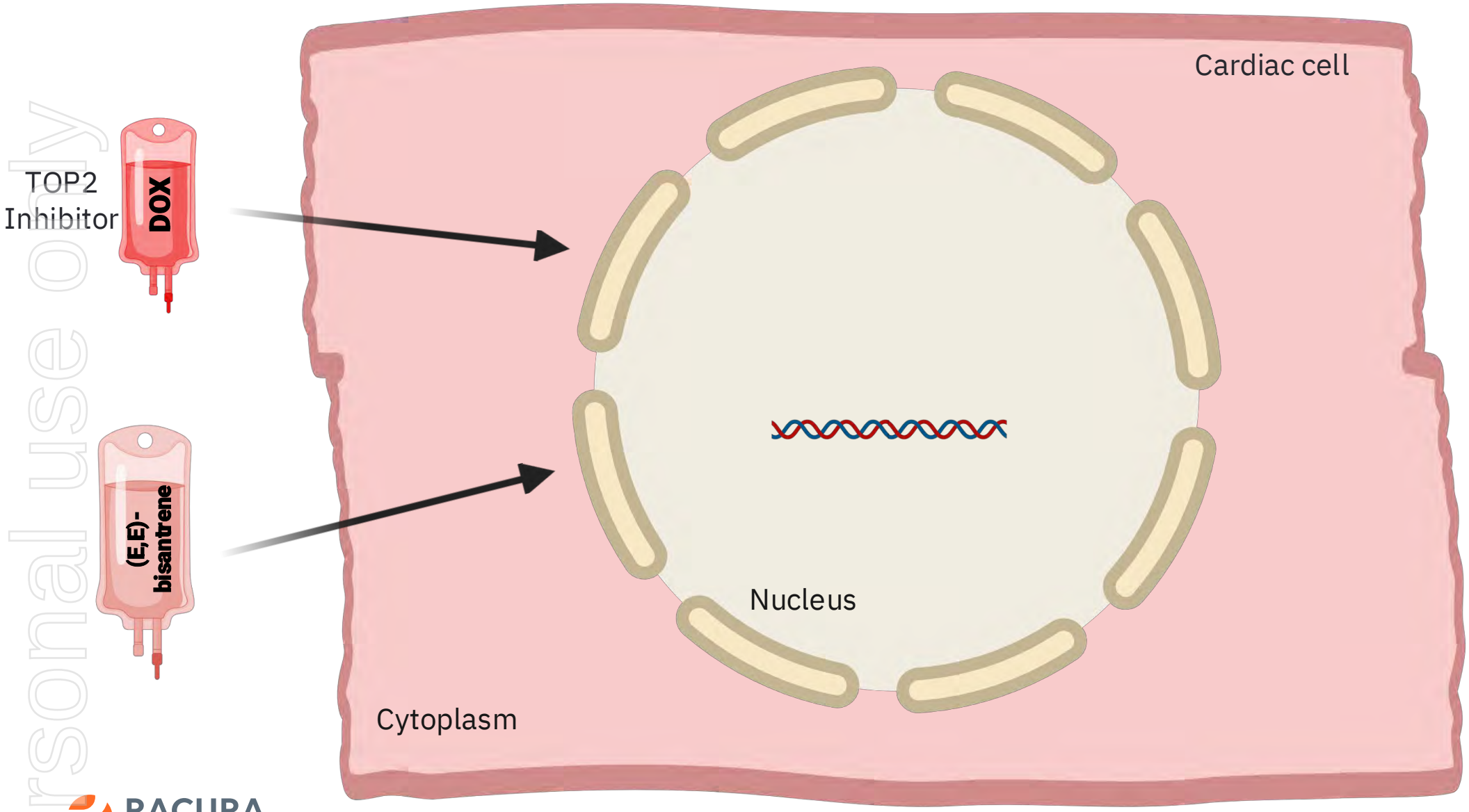
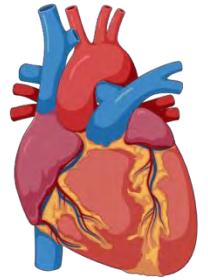
1. Das SK et al (2022) Mol Cell, 82(1):140–158
2. Tillett et al (2025) Ann Oncol, 36:S658

MYC silencing by (E,E)-bisantrene reduces DSBs





Cardioprotection

Please scan QR code for animation

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TOP1, topoisomerase I; TOP2,topoisomerase II; MAX, Myc-associated factor X; DBS, double-strand breaks

Science summary

Through rigorous science, Racura Oncology has discovered how (E,E)-bisantrene silences MYC

MYC G-quadruplex	(E,E)-bisantrene binds and stabilises G-quadruplex in MYC gene promoter region
MYC expression	(E,E)-bisantrene silences MYC expression in cancer cells
MYC activity	(E,E)-bisantrene inhibits MYC target genes

CPACS | HARNESS-1 | PHASE 3 AML

RACURA ONCOLOGY

CPACS, cardioprotection and anticancer synergy; AML, acute myeloid leukaemia

Preclinical team

Prof Michael Kelso
Vice President of Research

University of Wollongong Australia
Scripps Research – Science Changing Life

Sumit Sahni, PhD
Senior Scientist

KOLLING Institute of Medical Research
UIC
The University of Sydney

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University of Reading
murdoch children's research institute

David Bourke, PhD
Senior Manager, CMC

Pfizer
Monash University
Hospira

Peter Cuthbertson, PhD
Scientist

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Emily Ryan
Associate Scientist

University of Wollongong Australia

Chelsea Penney
Research Associate

University of Wollongong Australia

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Questions

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Clinical programs

Overview of Racura Oncology's clinical trials

Clinical pipeline

Three phase 1-3 clinical programs in AML, lung cancer and solid tumours

Cancer indication	Total addressable patients[1]	Phase 1	Phase 2	Phase 3	Comment
Phase 3 AML Acute myeloid leukaemia[2]	US/EU: 50,000	Phase 3			40% response rate in two Phase 2 trials[3] Bisantrene approved in 1988 in France
HARNESS-1 Lung Cancer EGFR Non-small lung cancer (NSCLC) in combination with osimertinib (TKI)[2]	China: 445,000 US/EU: 95,000	Phase 1			Osimertinib 2025 sales exceed US$7.2 billion[4]
CPACS Cardioprotection & anticancer synergy in combination with doxorubicin in solid tumours[5]	Global: 4 million	Phase 1			Patients have been safely dosed with RC220 and RC220+Dox[5]

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1. Estimated number of patient per year for each indication. US – USA & Canada; EU – Europe 2. ASX Announcement (17 Nov 2025) 3. ASX Announcement (30 Jul 2024) 4. AstraZeneca Annual Report 2025; Jannsen Annual Report 2025; Hansoh Annual Report 2025; Shanghai Allist Annual report 2025
5. ASX Announcement (20 Oct 2025) | AML, acute myeloid leukaemia; TKI, tyrosine kinase inhibitors; Dox, doxorubicin

Clinical team

Rodney Cusack, PhD
Principal Scientist

The University Of Queensland AUSTRALIA · CYTO MATRIX · QBiotics Group

Dr Simon Fisher MBBS
Vice President of Medical

NOVARTIS · Medical Developments International · Johnson&Johnson

Corli Merry
Senior Clinical & Quality Mgr

parexel · NOVARTIS · Roche

Dr Anupa Kudva, MD
Medical Director

Johnson&Johnson · ANTENGENE · Memorial Sloan Kettering Cancer Center

Dr Jose Iglesias MBBS FRACP
Chief Medical Officer

Lilly · Celgene · Abraxis BioScience · AMGEN

Marinella Messina, PhD
Vice President of Clinical

NOXOPHARM · KOLLING Institute of Medical Research · Datapharm Australia CLINICAL TRIAL SPECIALISTS

Michelle Huh
Clinical Trials Manager

NPS MEDICINEWISE · Ahpra & National Boards · NSW Government Health Western Sydney Local Health District

Kirsten Curnow, PhD
Regulatory Affairs

THE UNIVERSITY OF SYDNEY · illumina

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CPACS clinical program

A/Prof Erin Howden, Exercise Physiology, University of New South Wales

Dr Marinella Messina, Vice President of Clinical, Racura Oncology

Agenda: CPACS

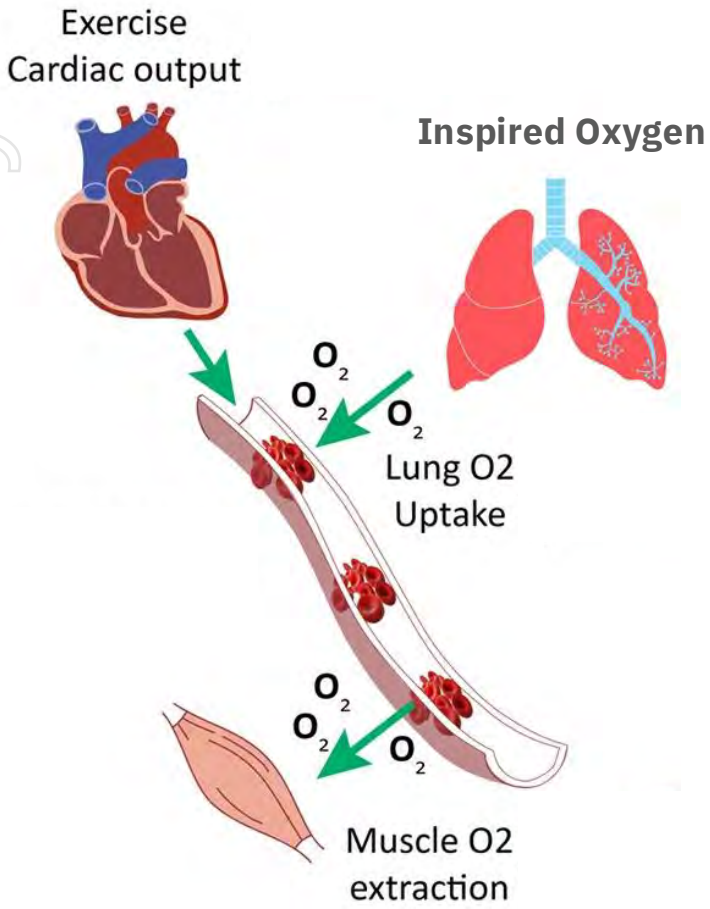
Topic	Presenter
Cardiotoxicity, patient journey & unmet need	A/Prof Erin Howden
CPACS trial	Dr Marinella Messina

RACURA ONCOLOGY

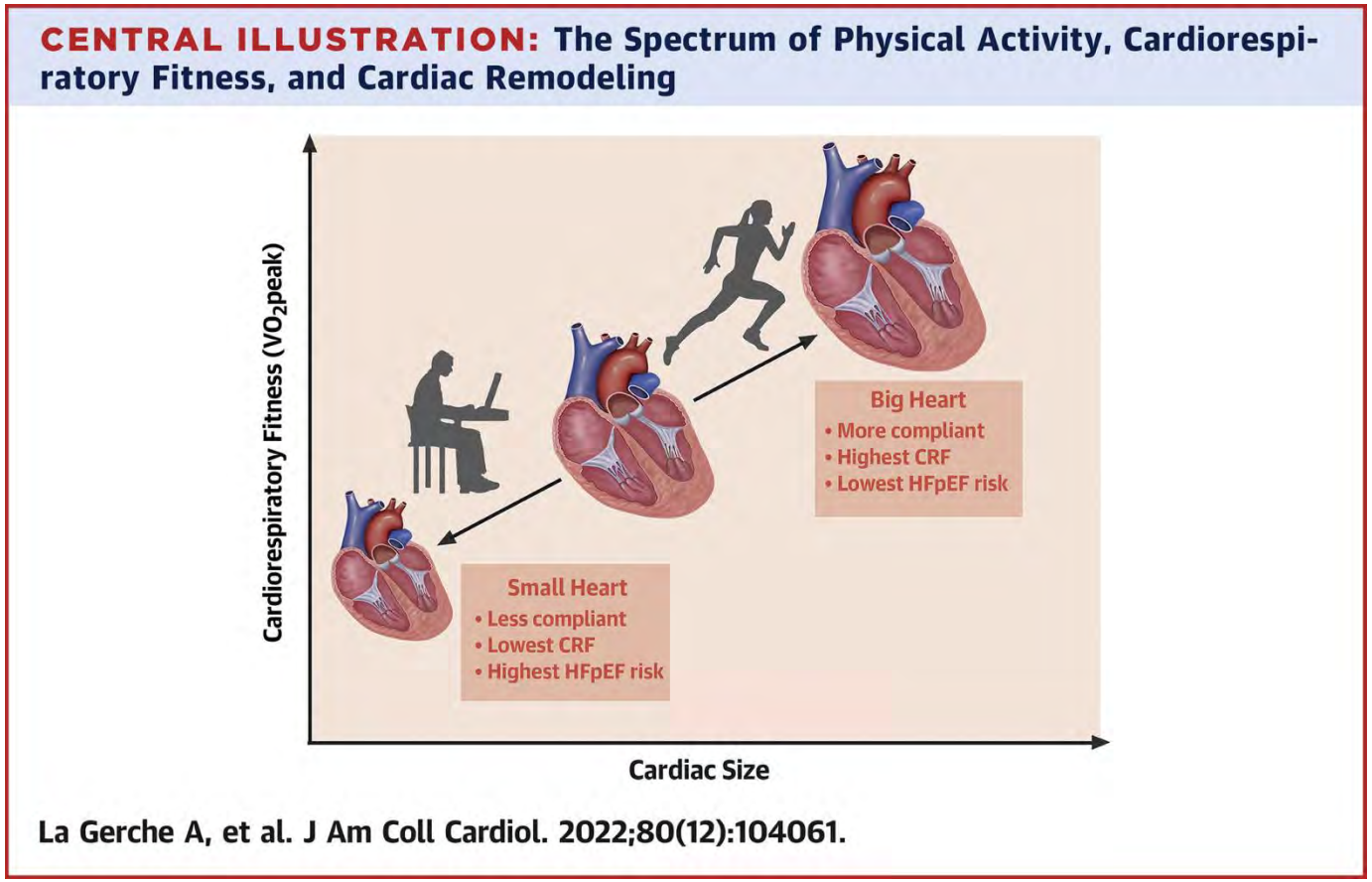
Cardiotoxicity, patient journey & unmet need

Associate Professor Erin Howden

My research program: Exercise as a framework to understand mechanisms and inform treatment of people at risk of heart failure



Oxygen Utilization Cascade



Adaptive Capability of Cardiovascular System

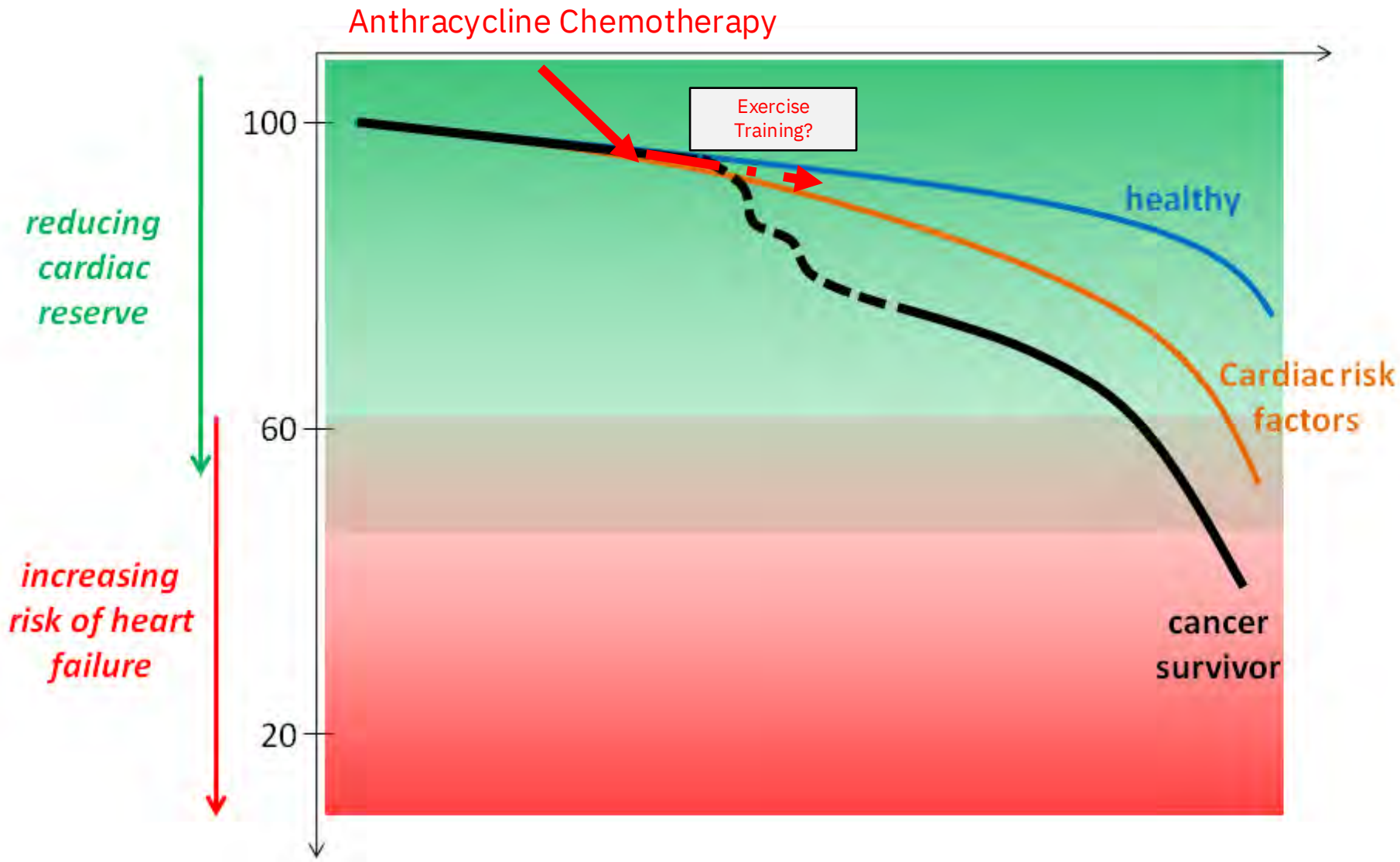
Howden et al., Circulation 2021; La Gerche, Howden et al., JACC 2022

UNSW SYDNEY

What's this got to do with people being treated for cancers?

UNSW SYDNEY

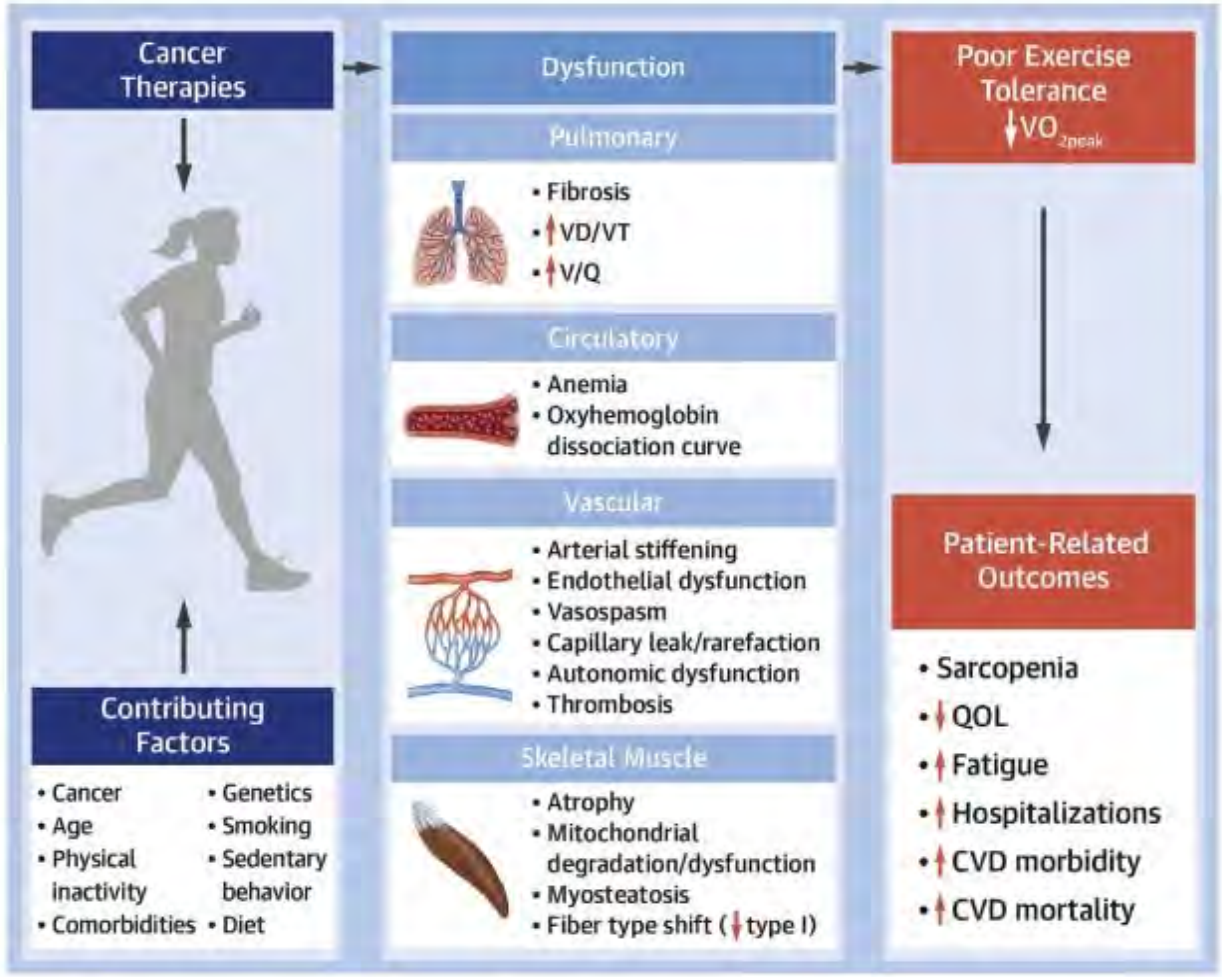
“CANCER” accelerates cardiometabolic aging



Criscitello et al. Breast Can Res 2012; Howden et al. Eur J Prev Cardiol 2018

UNSW SYDNEY

CENTRAL ILLUSTRATION: Multifactorial Contributors and Cardiorespiratory System Impact in Cancer Survivors That Drive Exercise Tolerance



Dillon HT, et al. JACC CardioOncol. 2024;6(4):496-513.

HFpEF, heart failure with preserved ejection fraction

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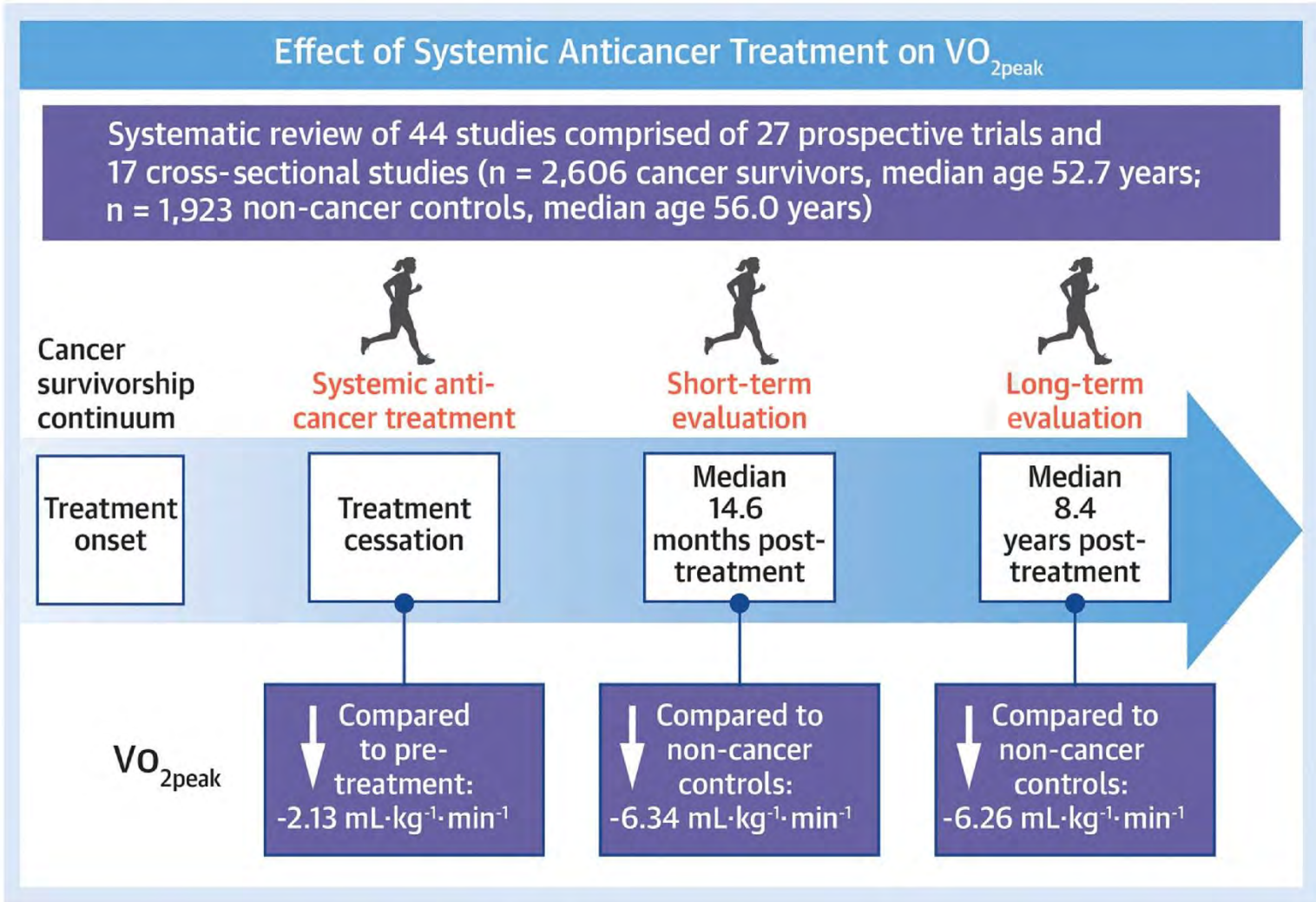
CENTRAL ILLUSTRATION: Impact of Systemic Anticancer Treatment on Vo_{2peak} During and After Therapy



Johansen SH, et al. JACC CardioOncol. 2025;7(2):96-106.

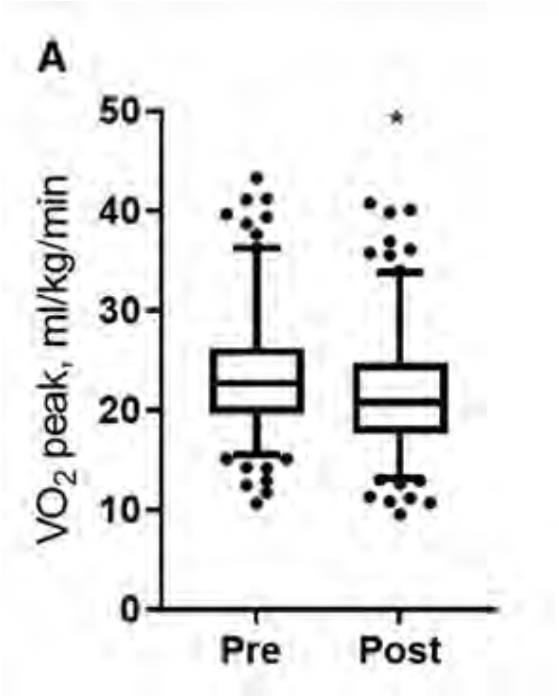
UNSW SYDNEY

ESC European Society of Cardiology
European Heart Journal - Cardiovascular Imaging (2021) **22**, 451–458
doi:10.1093/ehjci/jeaa421

Traditional markers of cardiac toxicity fail to detect marked reductions in cardiorespiratory fitness among cancer patients undergoing anti-cancer treatment

Erin J. Howden[1], Steve Foulkes[1,2], Hayley T. Dillon[1,2], Ashley Bigaran[1,3], Leah Wright[1], Kristel Janssens[1], Prue Comie[4,5], Benedict Costello[1,6], and André La Gerche[1,6]*

- 206 patients prospectively enrolled before commencing treatment
  - 53±13y, 35% male
- Mixed cancer diagnosis – majority breast or haematological cancers
  - 53% Anthracycline; 19% BTK; 19% Allogeneic Stem Cell Transplant; 10% ADT



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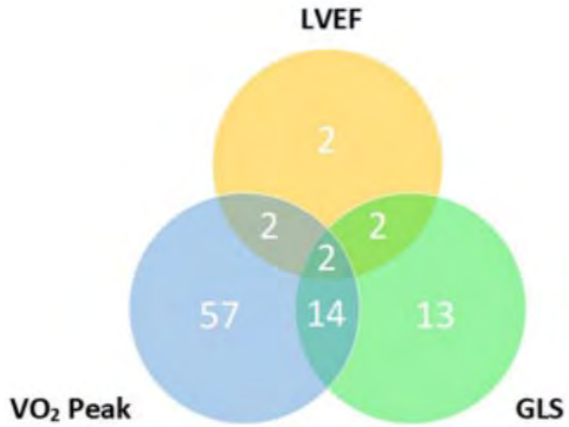
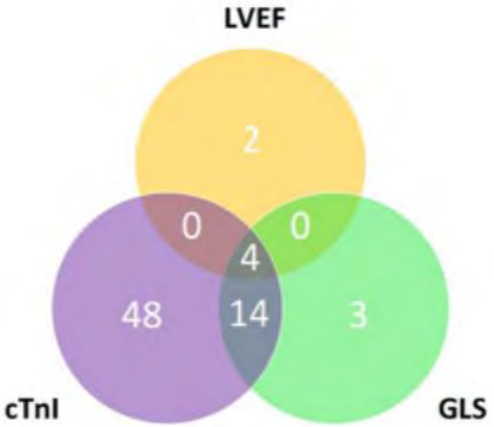
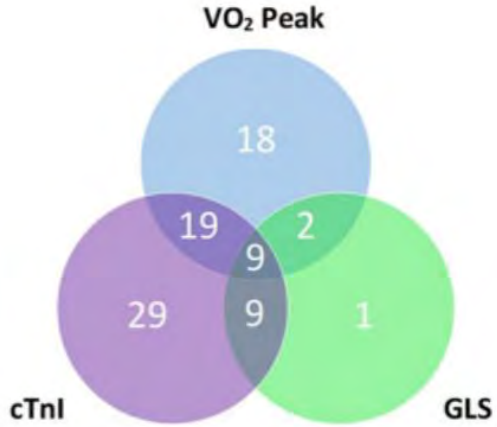
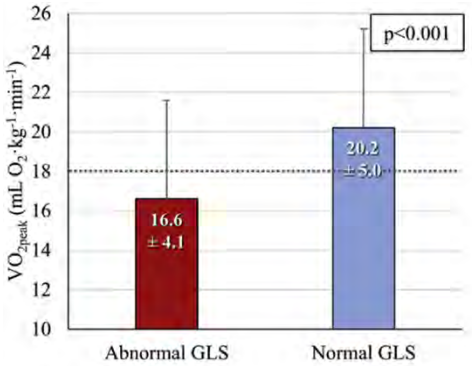
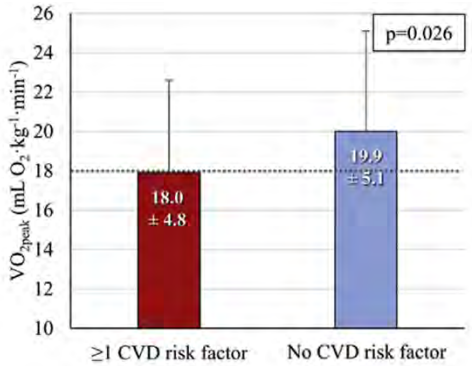
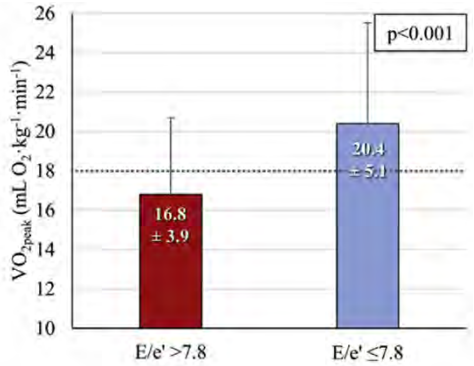


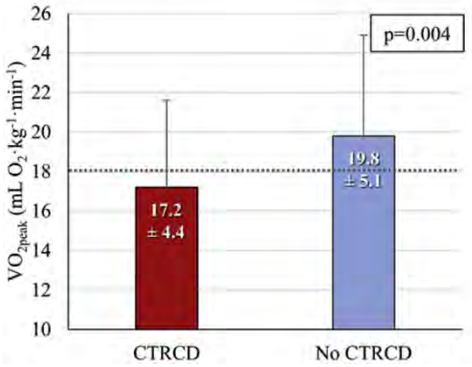


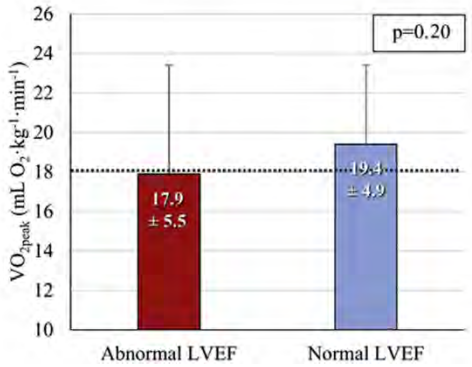


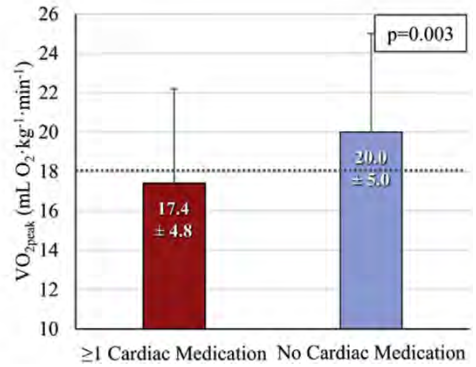








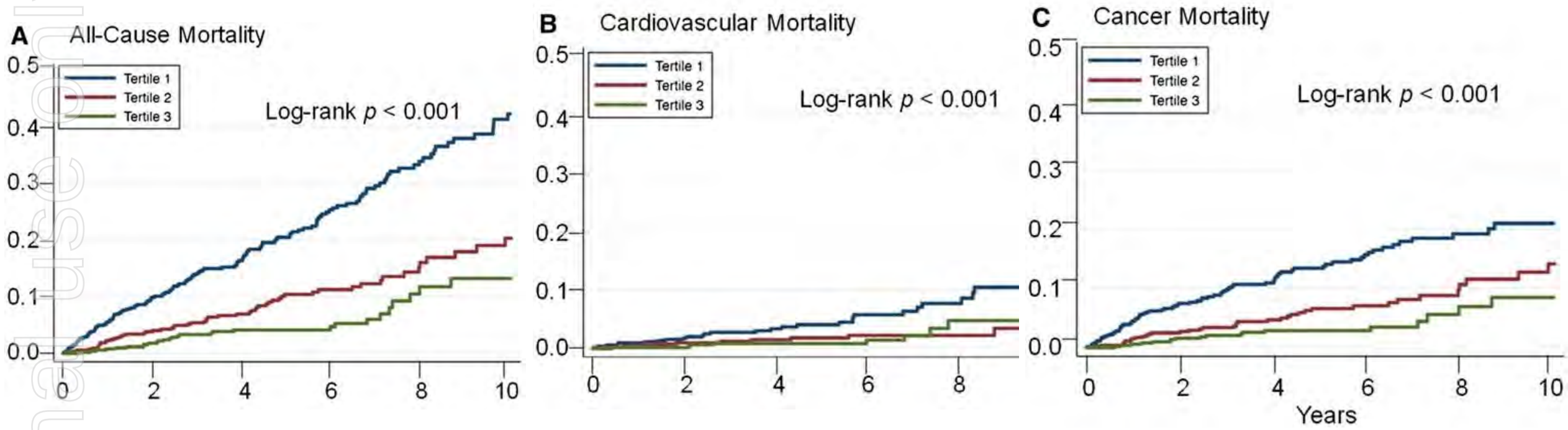




Howden et al., EHJ CVI 2021; Bonsignore et al., JACC CO 2021

UNSW SYDNEY

Post-diagnosis cardiorespiratory fitness is associated with cause-specific mortality

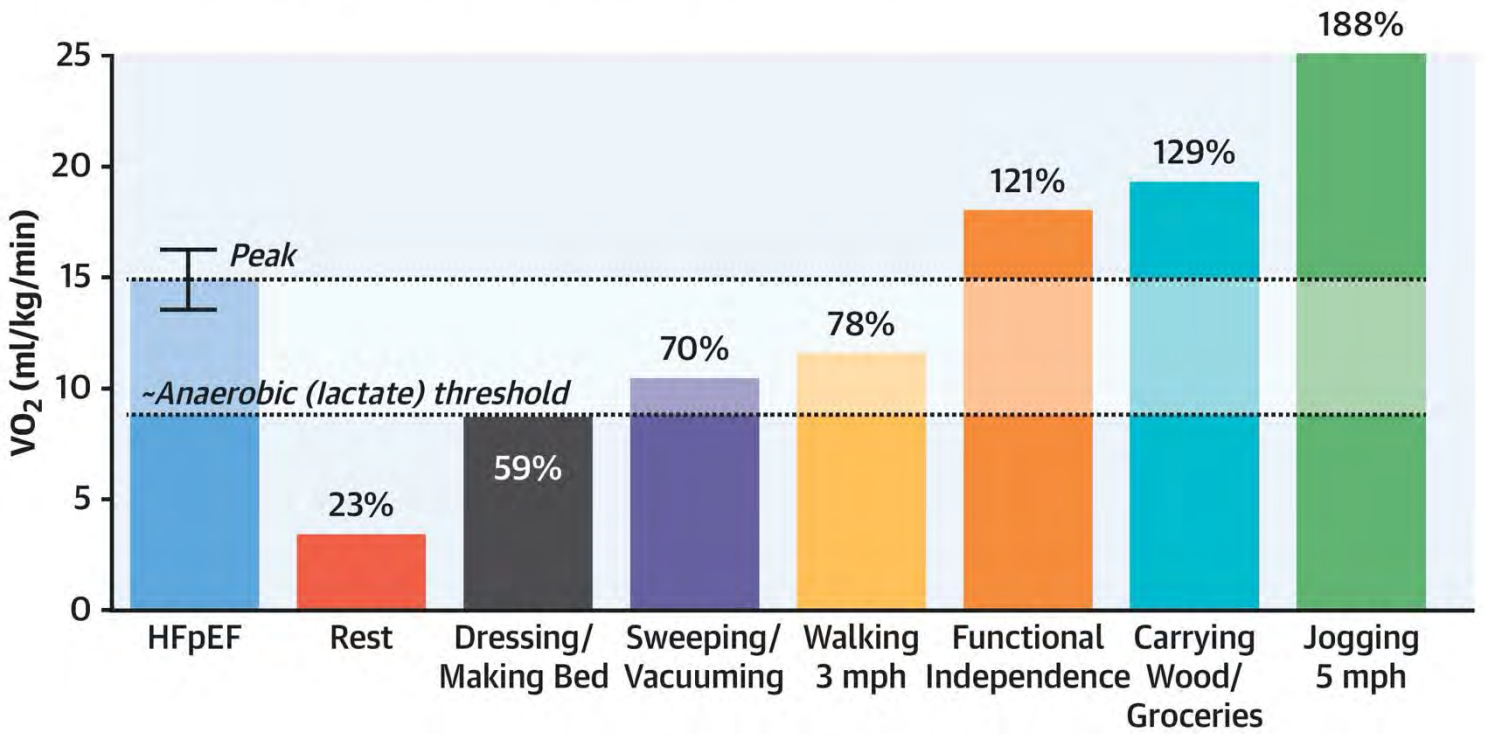


For each 1 MET Increase in CRF all-cause, CV, and cancer mortality decreased by 26%, 14%, and 25%

Groarke et al., EHJ - Quality of Care and Clinical Outcomes 2020

CRF, cardiorespiratory fitness; CV, cardiovascular; MET, peak metabolic equivalents

CENTRAL ILLUSTRATION: Peak VO_2 Required for Activities of Daily Living Relative to Average Peak VO_2 Observed in HFpEF

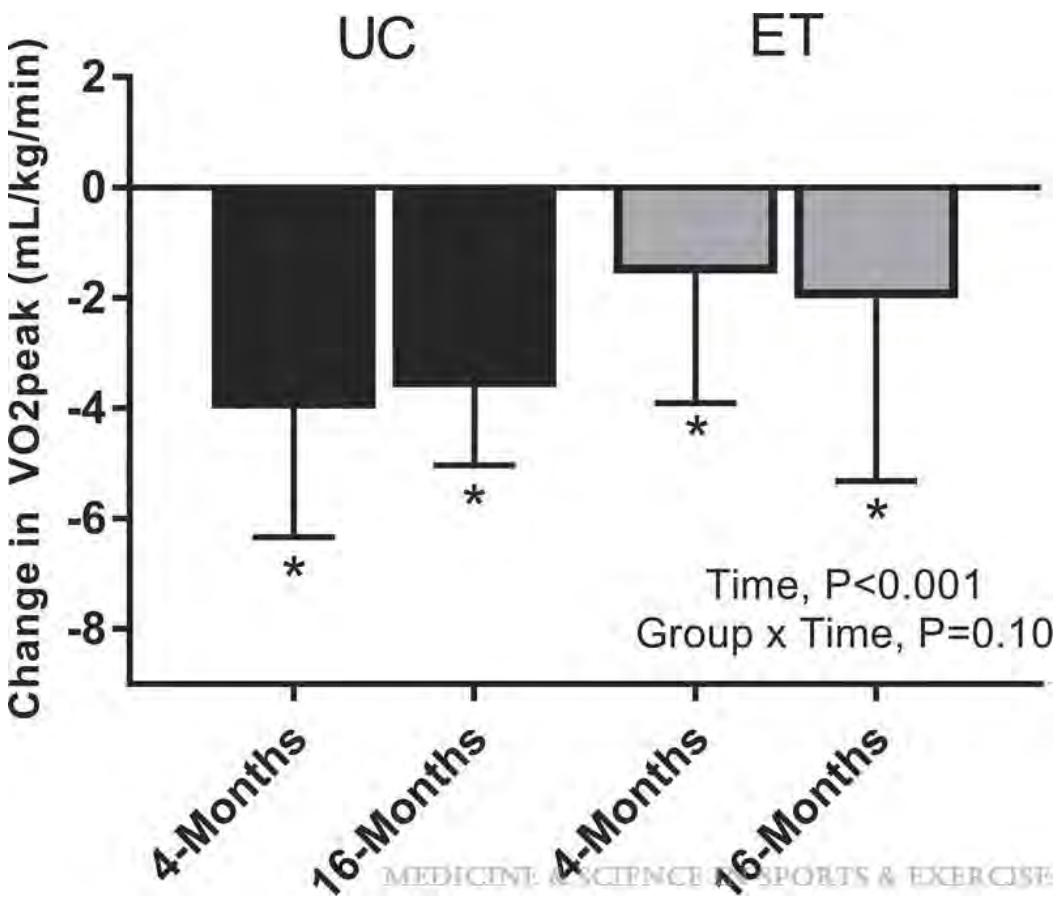


Nayor, M. et al. J Am Coll Cardiol HF. 2020;8(8):605-17.

HFpEF, heart failure with preserved ejection fraction

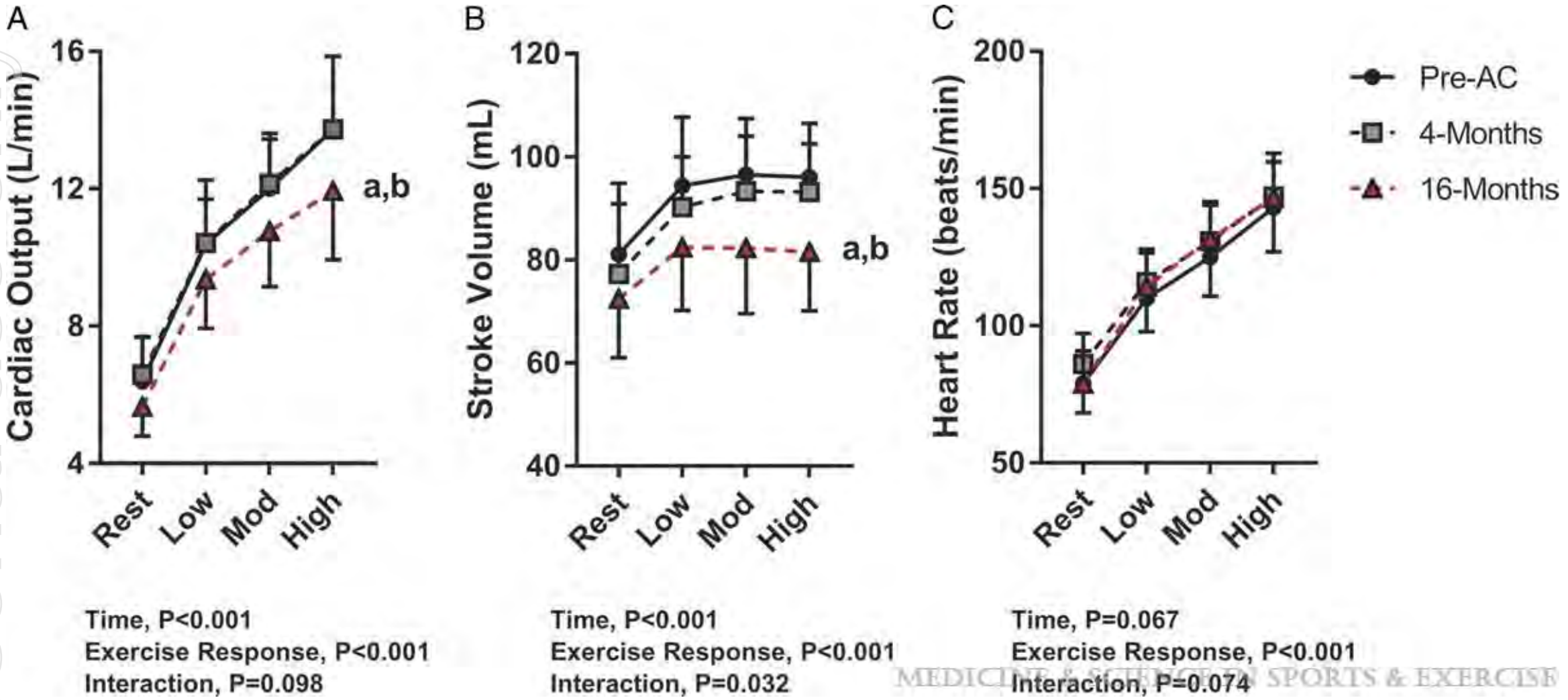
UNSW SYDNEY

In breast cancer survivors, VO_2 peak remains impaired >1 year post completion of anthracycline treatment



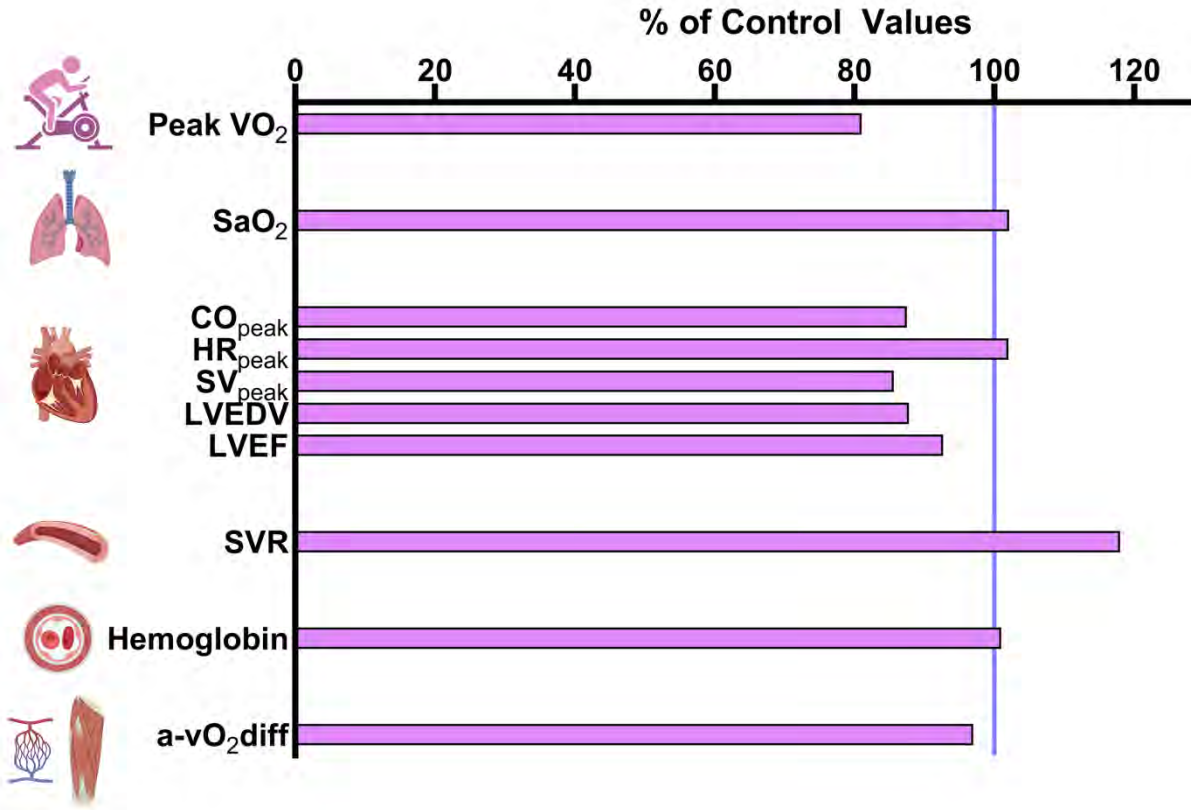
Foulkes, Howden et al., MSSE 2019

Reduced VO_2 peak is associated with impaired exercise cardiac function in breast cancer survivors



Foulkes, Howden et al., MSSE 2019

Reduced VO_2 peak in breast cancer survivors is not completely explained by impaired cardiac function



Foulkes et al., JACC CO 2024 | VO_2, volume of oxygen; SaO_2, arterial oxygen saturation; HR, heart rate; SV, stroke volume; LVEDV, left ventricular end diastolic volume; LVEF, left ventricular ejection fraction; SVR, systemic vascular resistance; a-vO_2 diff, arteriovenous oxygen difference.

Correcting one O_2 parameter defect

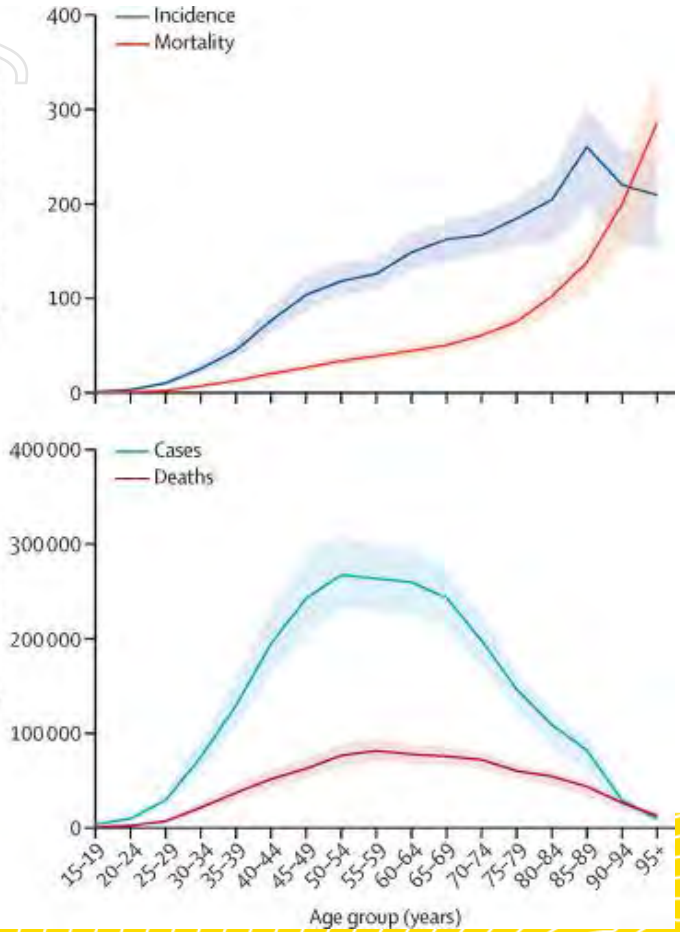
Performance increase = Limited

Changing multiple parameters – not only the engine matters

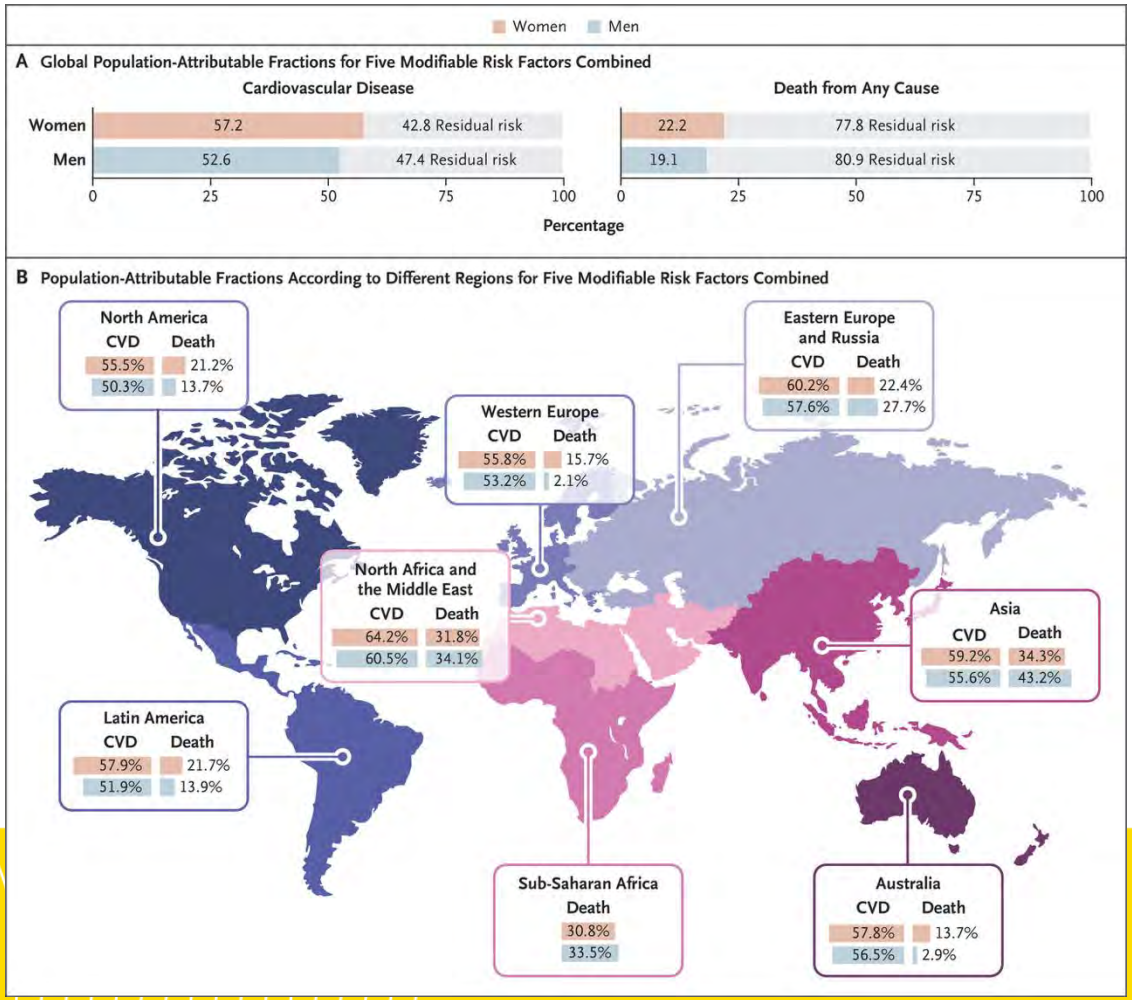
More profound increase in performance

We need new solutions

~50% increase in # cases per year



Heart disease is the most common cause of death



GBD 2023 Breast Cancer Collaborators Lancet Oncology, Global cardiovascular Risk Consortium NEJM 2023

UNSW SYDNEY

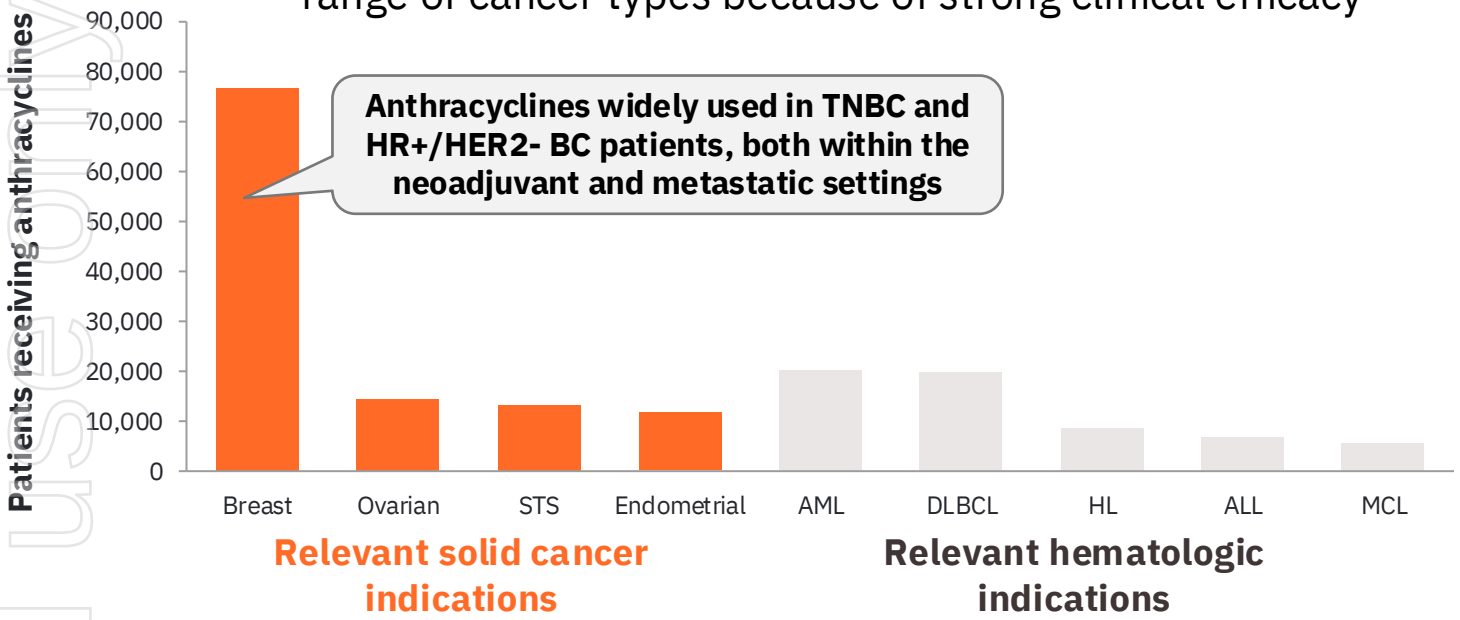
RACURA ONCOLOGY

CPACS trial

Dr Marinella Messina, Vice President of Clinical, Racura Oncology

Anthracycline use & cardiotoxicity

US data[1] – Anthracyclines remain broadly used across a range of cancer types because of strong clinical efficacy



20 million doses of anthracycline globally p.a.

1. SEER Cancer Stats; Cancer.org; Triangle Insights Analysis, Primary Research, 2022; Consultant Analysis
2. Camilli M et al (2024); Lee AR et al (2023); Narezkina A and Nasim K (2019)

ALL, acute lymphoblastic leukaemia; AML, acute myeloid leukaemia; BC, breast cancer; DLBCL, diffuse large B-cell lymphoma; HER2-, human epidermal growth factor receptor 2 negative; HL, Hodgkin's lymphoma; HR+, hormone receptor positive; LVEF, left ventricular ejection fraction; MCL, mantle cell lymphoma; p.a., per annum; STS, soft tissue sarcoma; TNBC, triple negative breast cancer.

RACURA ONCOLOGY

Anthracycline use & cardiotoxicity

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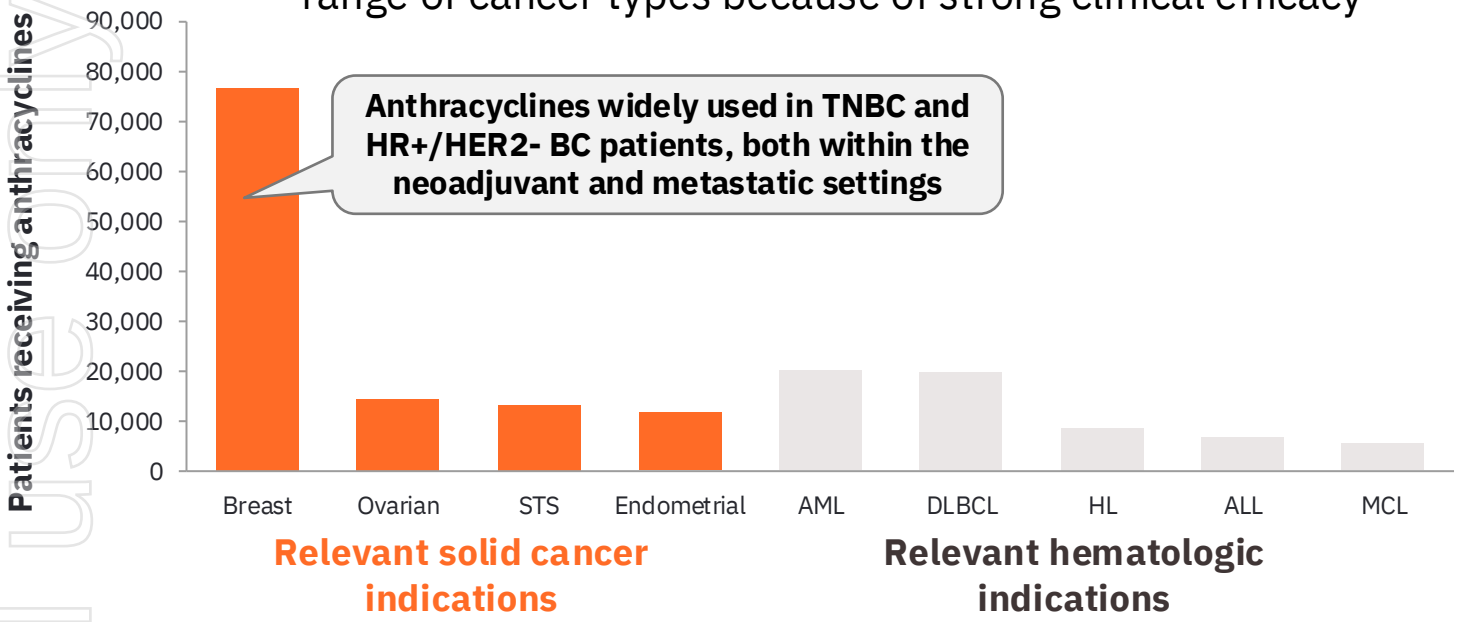

Anthracyclines associated with significant and permanent cardiotoxicity

Remains significant concern for oncologists

- Incidence of cardiotoxicity post-treatment by LVEF 9-17% (standard measure)
- 80-85% of US patients reach the lifetime limit by late-stage disease

Chemotherapy damages the heart in stages:
early microscopic injury → structural decline → loss of functional capacity

1. SEER Cancer Stats,; Cancer.org; Triangle Insights Analysis, Primary Research, 2022; Consultant Analysis
2. Camilli M et al (2024); Lee AR et al (2023); Narezkina A and Nasim K (2019)

ALL, acute lymphoblastic leukaemia; AML, acute myeloid leukaemia; BC, breast cancer; DLBCL, diffuse large B-cell lymphoma; HER2-, human epidermal growth factor receptor 2 negative; HL, Hodgkin's lymphoma; HR+, hormone receptor positive; LVEF, left ventricular ejection fraction; MCL, mantle cell lymphoma; p.a., per annum; STS, soft tissue sarcoma; TNBC, triple negative breast cancer.

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Clinical strategy in anthracycline-treated solid tumours

Integrated trial for early signals of both cardioprotection and anticancer activity

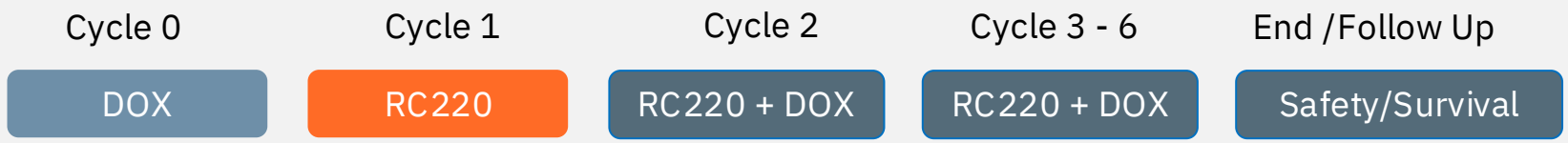
RACURA ONCOLOGY

MTD, maximum tolerated dose

CPACS trial design: Phase 1a

Safe RC220 + doxorubicin maximum tolerated combined dose

Patients: Advanced/metastatic patients with potential benefit from doxorubicin treatment
Design: RC220 dose escalation + 60 mg/m^2 doxorubicin (DOX)
Trial size: 15-33 patients
Trial status: **Ongoing** – Cohort 1 (combo with 40 mg/m^2 RC220) complete



Goals

- Evaluate safety and tolerability of a range of RC220 doses in combination
- Find MTCD
- Generate pharmacokinetic data
- Collect preliminary signals of
  - Cardioprotective effect (molecular biomarker)
  - Anti-tumour efficacy (imaging)

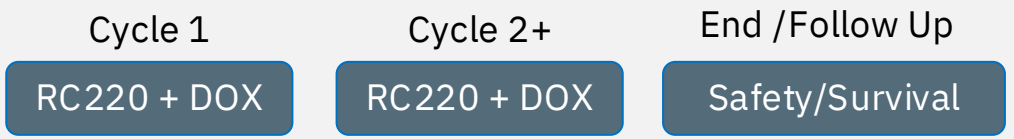
Interim analysis → Phase 1b dose expansion

DOX, doxorubicin; MTCD, maximum tolerated combined dose.

CPACS trial design: Phase 1b

Signals of anticancer & cardioprotection efficacy

Patients: Anthracycline naïve solid tumour patients
Design: RC220 MTCD + 60 mg/m^2 doxorubicin (DOX)
Trial size: 20 patients
Trial status: Pending interim analysis in phase 1a



Goals

Anticancer effects

- Confirm safety
- Determine recommended phase 2 dose
- Evaluating hair loss
- Evaluating Quality of Life

Cardioprotective effects

- Physiologic functional: **VO_2peak**
- Biological: PDx molecular biomarker of CP effect
- Heart specific (structure, function & damage)
  - Sub-clinical: GLS & cMRIs
  - Traditional LVEF & CTRCD

Clinically relevant dual effect → Advance to front line development

RACURA ONCOLOGY

MTCD, maximum tolerated combined dose; DOX, doxorubicin; PDx, pharmacodynamic; CP, cardioprotection; GLS, global longitudinal strain; cMRI, cardiac magnetic resonance imaging; LVEF, left ventricular ejection fraction; CTRCD, cancer therapy-related cardiac dysfunction

RACURA
ONCOLOGY

Questions

RACURA ONCOLOGY

HARNESS-1 clinical program

Prof Nick Pavlakis, Senior Staff Medical Oncologist, Royal North Shore Hospital

Dr Rodney Cusack, Principal Scientist, Racura Oncology

Agenda: HARNESS-1

Topic	Presenter
EGFR mutant NSCLC and HARNESS-1 trial	Prof Nick Pavlakis
Competitive landscape: Potential of RC220 use in EGFRm NSCLC	Dr Rodney Cusack
Commercialisation: Regulatory pathways and opportunities	Dr Rodney Cusack

RACURA ONCOLOGY

EGFR mutant NSCLC and HARNESS-1 trial

Prof Nick Pavlakis, Senior Staff Medical Oncologist, Royal North Shore Hospital

RACURA SYMPOSIUM
March 24, 2026

EGFR mutant NSCLC and HARNESS-1 trial

Prof Nick Pavlakis

Senior Staff Medical Oncologist, RNSH

Board Member (former Chair) – The Thoracic Oncology Group of Australasia (TOGA)

Financial disclosures

- **Advisory Boards**
  - Boehringer Ingelheim, MSD, Merck, BMS, Astra Zeneca, Takeda, Pfizer, Roche, Amgen, Beigene, Gilead, Zymeworks, BioNTech, Daiichi, DUO Oncology, Amplia, Johnson and Johnson, RACURA (formerly RACE) oncology (Advisor, HARNESS-1 investigator)
- **Speaking Honoraria**
  - Merck, Pfizer, Roche, Takeda, Illumina, Bayer, MSD, Gene Solutions, Limbic
- **Research Funding – to institution**
  - Bayer, Pfizer, Roche

Lung Cancer Statistics

Lung cancer is the most common cancer (12.4%)

Globally 2.5M new cases pa[1]
US >200K new cases pa[2]
China >1M new cases pa[3]

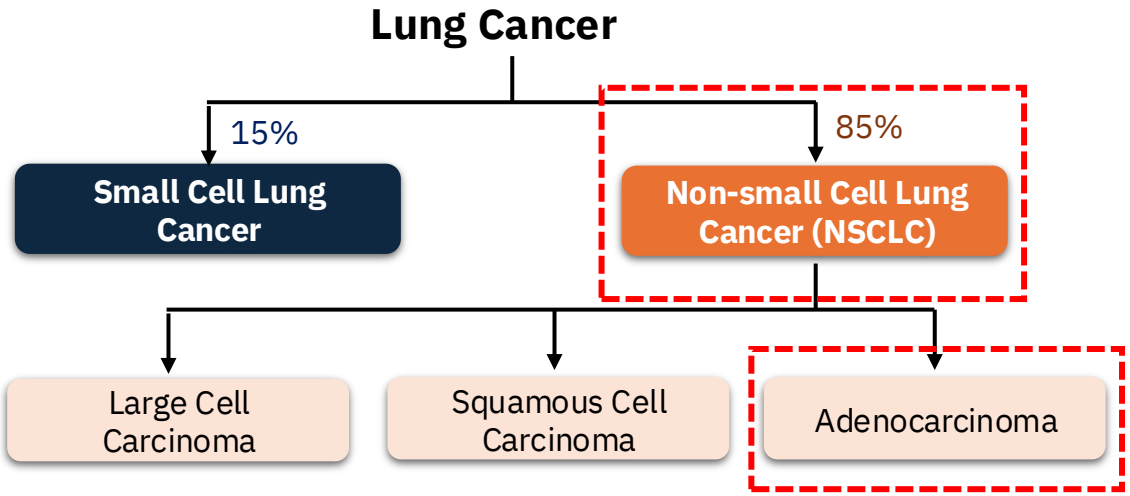
Lung cancer is the leading cause (18.7%) of cancer death – 1.8M deaths annually[1]

Smoking is the primary risk factor.
~25% of cases are attributed to causes other than smoking, such as environmental exposures
(e.g. air pollution, exposure to asbestos or radon).[1]

The number of new cancer cases is estimated to almost double by 2050,[1] increasing new lung cancer cases globally to ~4M per year (~3.4 M NSCLC).

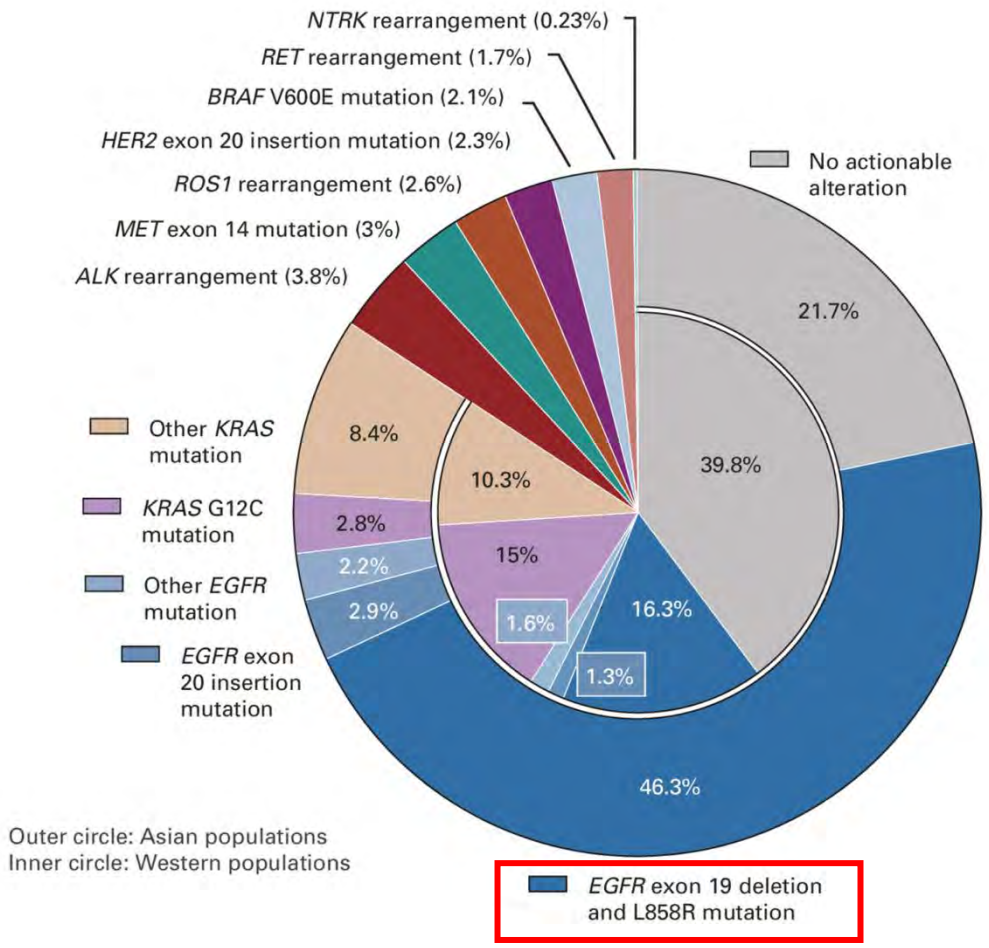
Lung Cancer sub-types

~85% of lung cancers are Non-small cell lung cancer (NSCLC)



1. 2022 Global cancer statistics Global cancer statistics 2022: GLOBOCAN estimates of incidence and mortality worldwide for 36 cancers in 185 countries
2. U.S. Cancer Statistics Working Group. U.S. Cancer Statistics Data Visualizations Tool. U.S. Department of Health and Human Services, Centers for Disease Control and Prevention and National Cancer Institute. https://www.cdc.gov/cancer/dataviz. Accessed June 16, 2025.
3. Han B, Zheng R, Zeng H, et al. Cancer incidence and mortality in China, 2022. J of the Nat Cancer Center. 2024;4(1):47-53

NSCLC is not one disease: molecularly heterogeneous disease



Diminishing smoking rates have changed histologic rates

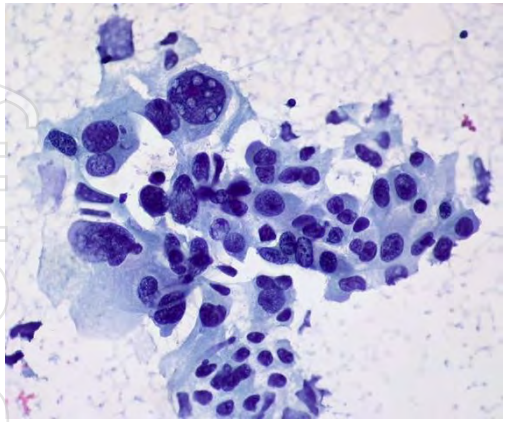
Small Cell Lung Cancer 15%
Immunotherapy
Non-small cell lung cancer (NSCLC) ~ 85%
Non –Squamous ~75%

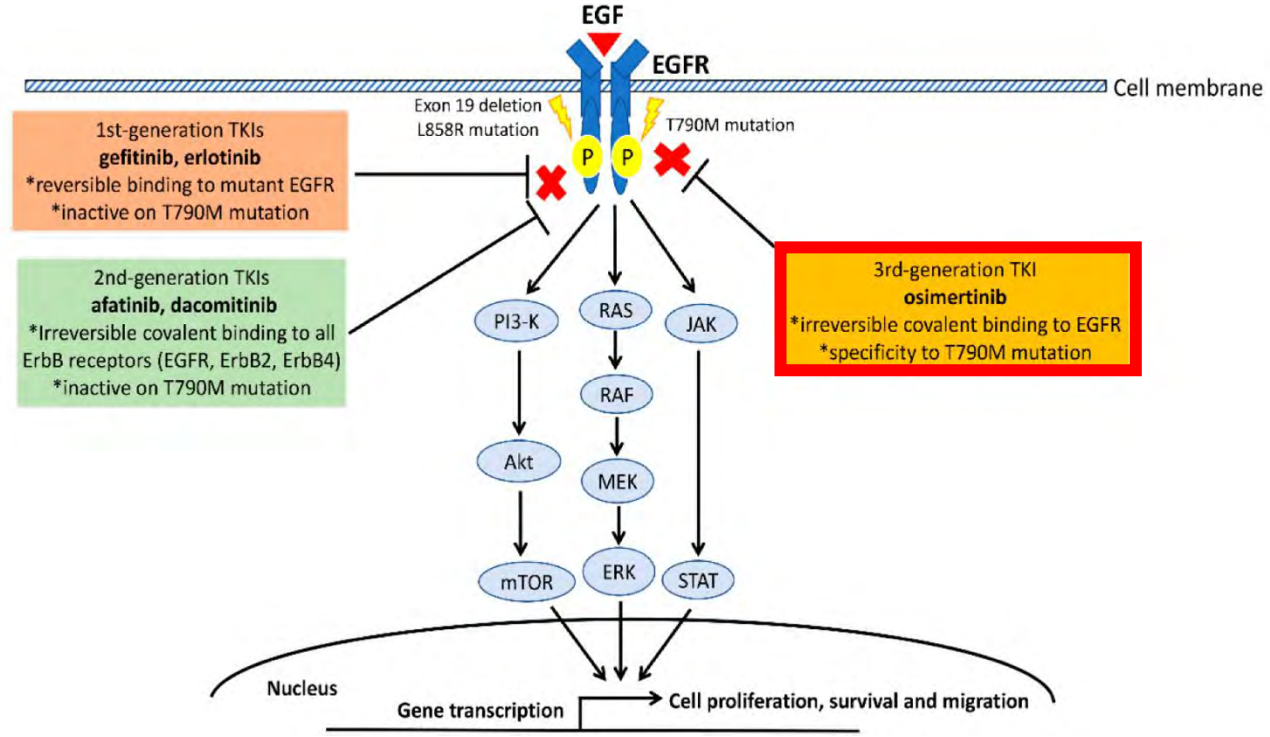
Targetable oncogenic driver molecular alterations in NSCLC (adenocarcinoma)
Treat with targeted therapy

Remainder: immunotherapy/chemotherapy

A Tan, D Tan. JCO
2022: DOI https://doi.org/10.1200/JCO.21.01626

EGFR mutant Non-Small Cell Lung Cancer (NSCLC)

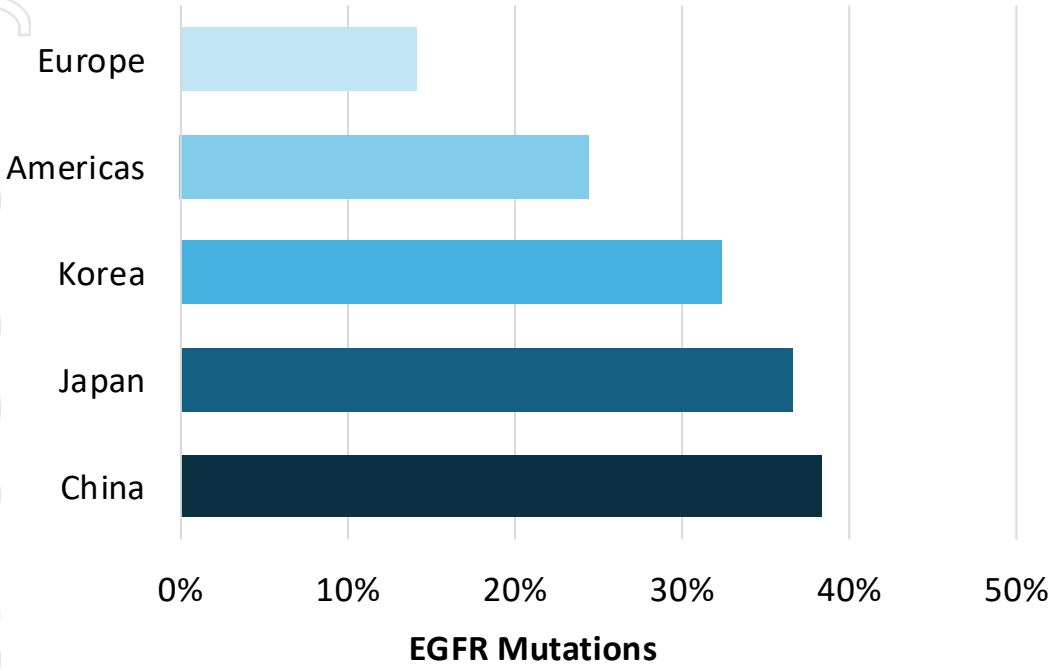




- **EGFR-mutated (EGFRm) NSCLC** is a subtype of lung cancer characterised by specific gene mutations in the epidermal growth factor receptor (EGFR) on the cancer cell surface that drives uncontrolled cell growth[1]
- The most common and treatable mutations are deletions in **exon 19** (E19del) (63%) and a point mutation in **exon 21** (L858R) (37%)[2,3]
- International guidelines recommend upfront targeted therapy with oral receptor tyrosine kinase inhibitors[4,5]
  - **Osimertinib** is standard in Australia

1. Bethune G et al. Epidermal growth factor receptor (EGFR) in lung cancer: an overview and update. J Thorac Dis 2010; 2(1): 48-51
2. Qi, Y.-T., Hou, Y. & Qi, L.-C. Efficacy of Next-Generation EGFR-TKIs in Patients With Non-Small Cell Lung Cancer: A Meta-Analysis of Randomized Controlled Trials. Technol. Cancer Res. Treat. 19 (2020).
3. Ramalingham SS et al. Overall Survival with Osimertinib in Untreated, *EGFR*-Mutated Advanced NSCLC. N Engl J Med 2020;382:41-50
4. NCCN Clinical Practice Guidelines in Oncology (NCCN Guideline®). Version 3- 2026, Dec 24, 2025
5. ESMO Clinical Practice Guideline: Oncogene –Addicted metastatic NSCLC. *Ann Oncol.* 2023;34(4):339-357

EGFR Mutations in NSCLC[1]



EGFR mutations more prevalent in:

- **Asian** patients
- **Females** (44%) > males (24%)
- **Non-smokers** (49%) > current or former smokers (22%)
- **Adenocarcinoma**

Typical clinical Presentation

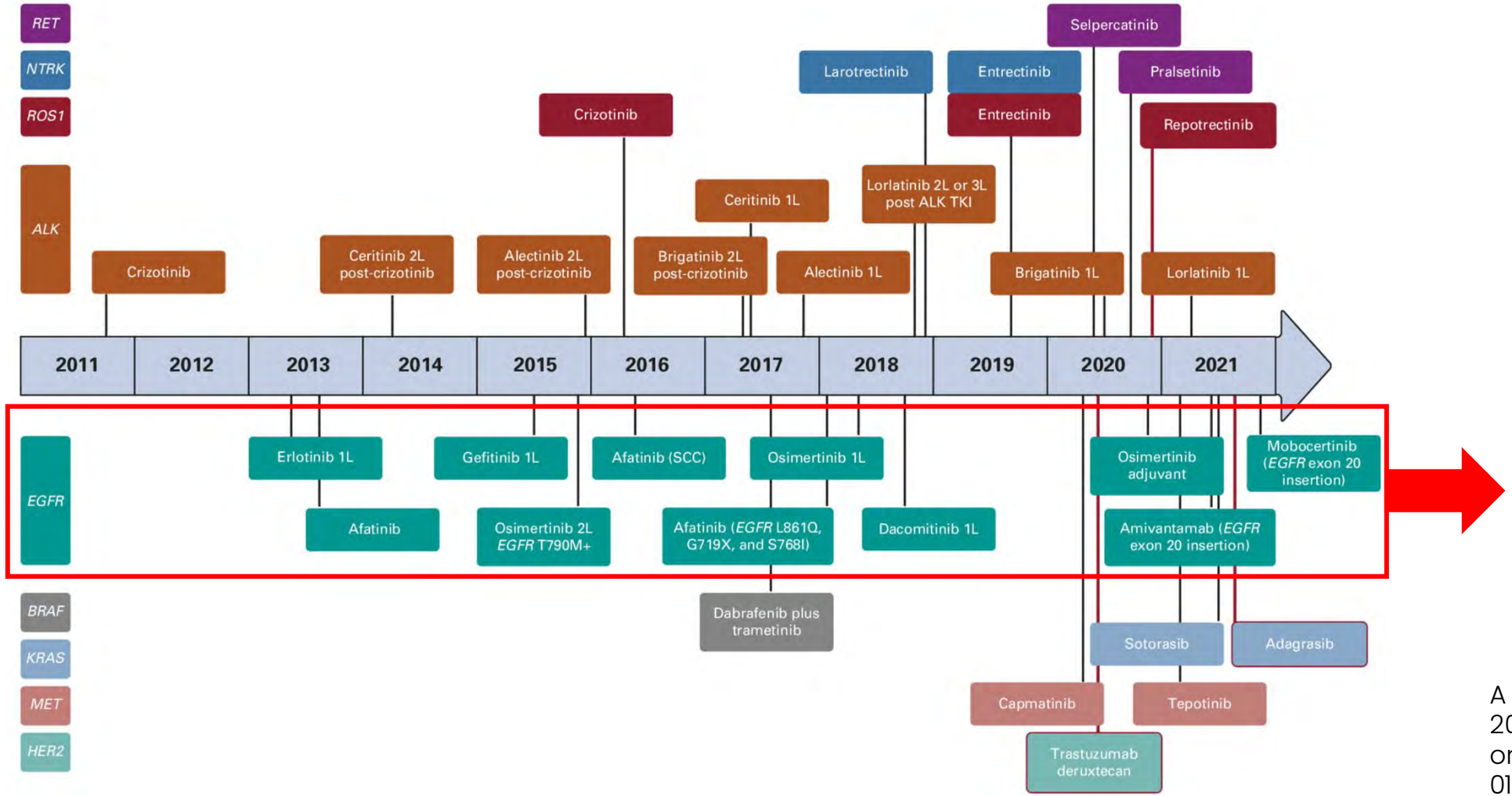
- **Symptomatic** – NB: Screening limited to smoking populations (except in some Asian countries (China/Taiwan)
- More commonly advanced/metastatic
  - Lung, lymph nodes, liver, bone, brain (~21% at diagnosis)[2]

High prevalence of EGFR mutations in Asia, is driving development of many new EGFR TKIs in this region

1. Zhang YL et al (2016) Oncotarget, 7(48):78985-78993
2. Ramalingham SS et al (2020) N Engl J Med, 382:41-50

Targetable oncogenic driver molecular alterations in NSCLC (adenocarcinoma)

Timeline of US FDA-approved targeted therapies for oncogene-driven NSCLC



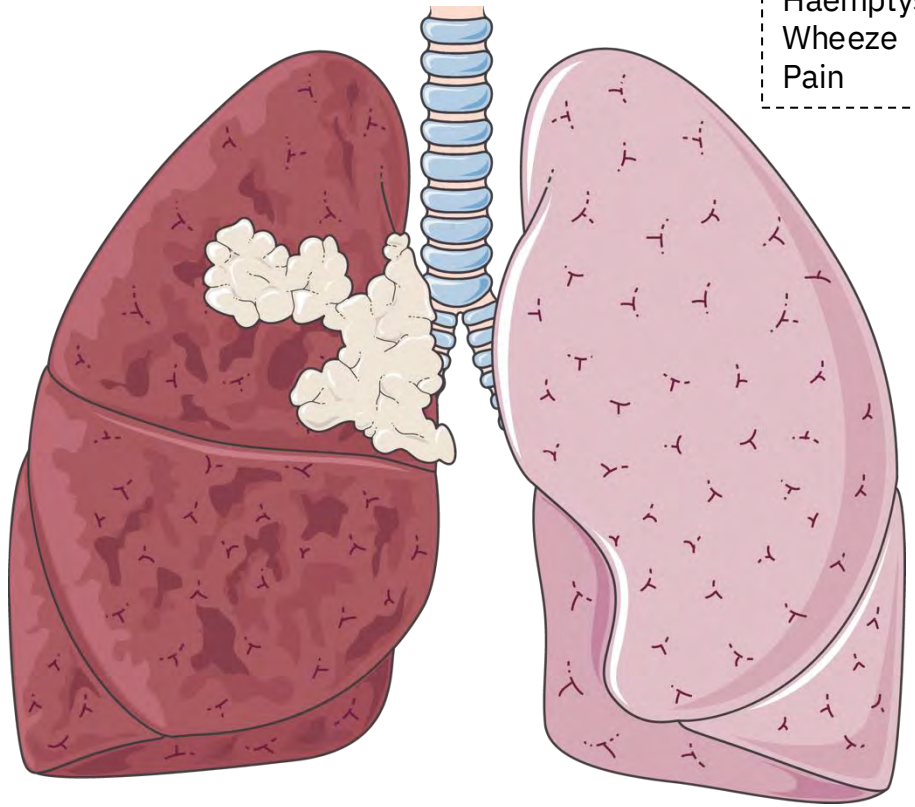
A Tan, D Tan. JCO January 5, 2022: DOI https://doi.org/10.1200/JCO.21.01626

EGFRm NSCLC Treatment with TKIs

- EGFRm NSCLC generally respond poorly to immunotherapy[1]
- Current mainstay of treatment are 3rd generation EGFRm Tyrosine Kinase Inhibitors (TKIs) – superior to chemotherapy[2]
- EGFRm TKIs are highly effective with **>80%** of patients with targetable mutations initially **responding to treatment[2]**

- **Response** = tumour shrinkage which in turns leads to resolution of symptoms

Local Symptoms
Cough
Breathlessness
Haemptysis (blood)
Wheeze
Pain



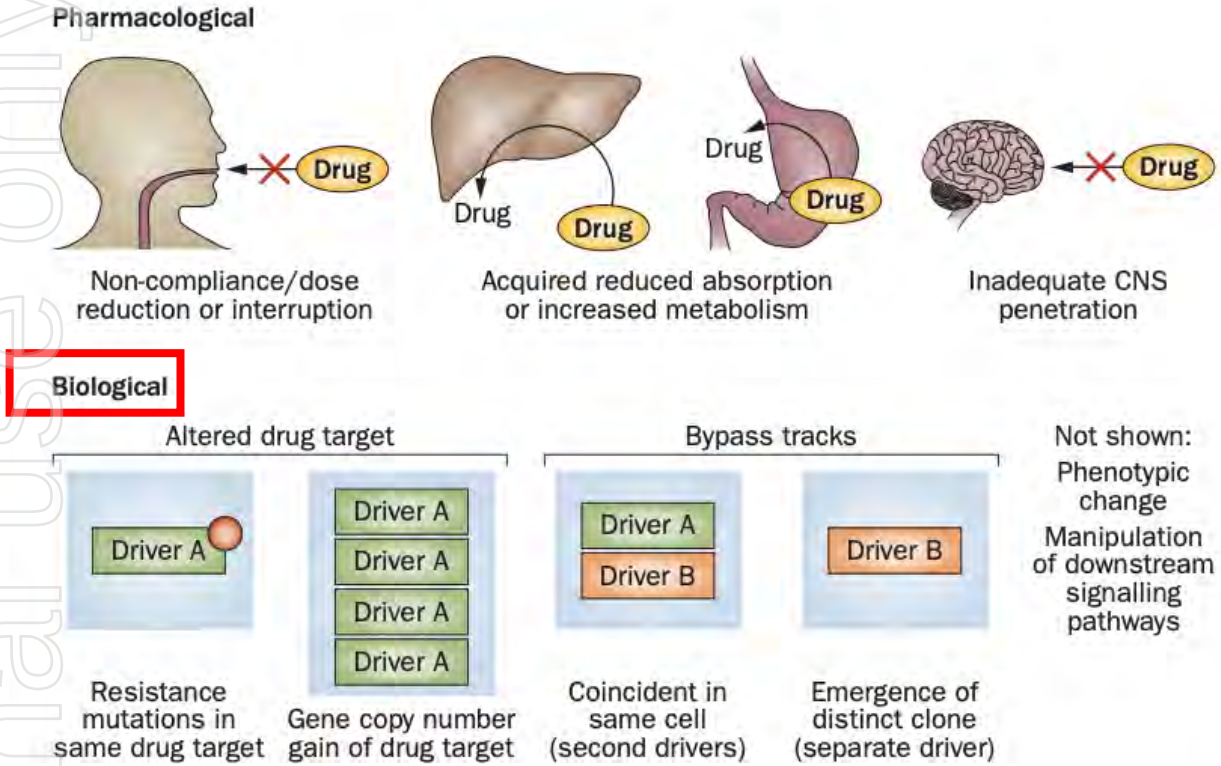
1. Zhang, W. et al. What is the optimal first-line regimen for advanced non-small cell lung cancer patients with epidermal growth factor receptor mutation: a systematic review and network meta-analysis. BMC Pulm. Med. 24, 620 (2024).
2. Veccia, A. et al. Osimertinib in the Treatment of Epidermal Growth Factor Receptor-Mutant Early and Locally Advanced Stages of Non-Small-Cell Lung Cancer: Current Evidence and Future Perspectives. Cancers 17, 668 (2025).

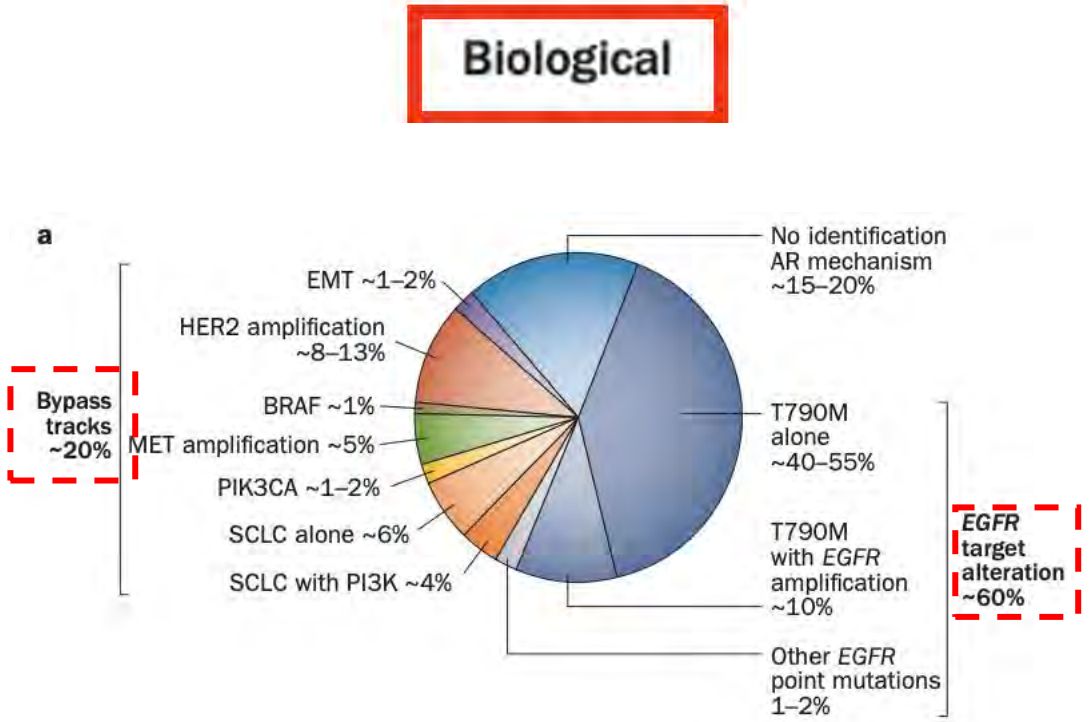
The Problem - Universal TKI Resistance

- Despite 14 approved EGRFm TKI and 8 in Phase 3 trials, virtually all patients develop resistance to EGFRm TKI therapy – the median progression free survival (PFS) is 18 months[1]
- No clinical difference between any of the 3rd generation EGFRm TKIs in regards time to resistance
- 4th generation EGFRm TKIs have been disappointing in the clinic
  - Why? Heterogeneous disease at progression. No one dominant resistance pathway.

1. Cho, B. C. et al. Amivantamab plus Lazertinib in Previously Untreated EGFR-Mutated Advanced NSCLC. N. Engl. J. Med. 391, 1486–1498 (2024).

Resistance in oncogene driven NSCLC – mainly biological



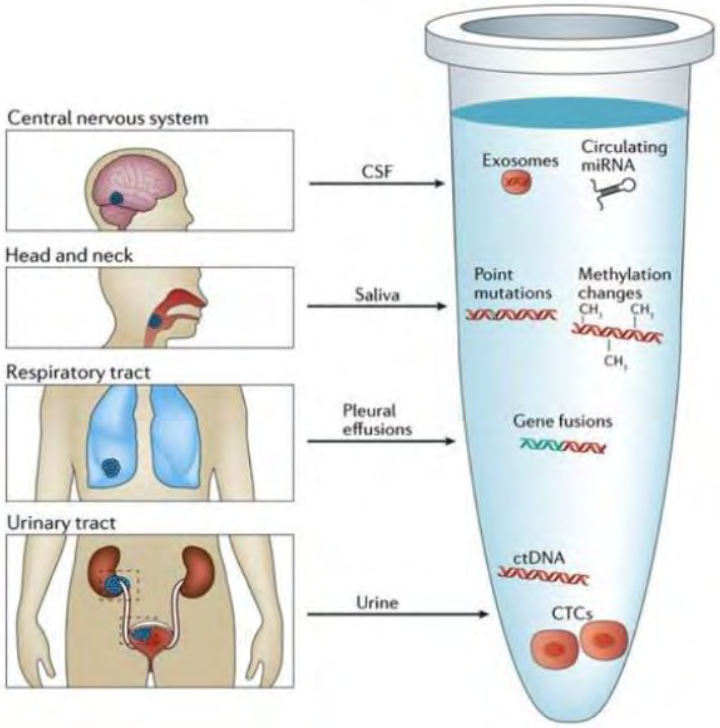


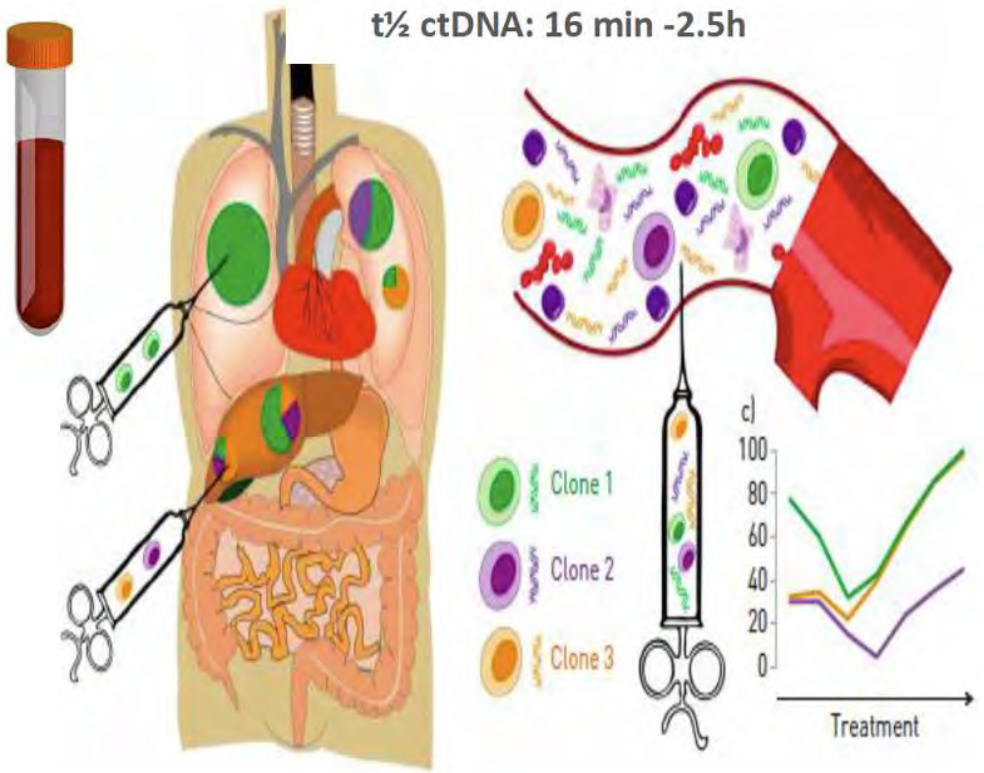
Camidge DR et al. Nat Rev Clin Oncol. 2014 (11): 473-481

NGS-Based Liquid Biopsy (LB) Instead of Tissue Biopsy

LB consist of isolating tumor-derived entities in any fluid
The circulating tumor DNA (ctDNA) is the most explored

Peripheral blood remains the most studied LB method as assesses spatial and temporal tumour heterogeneity

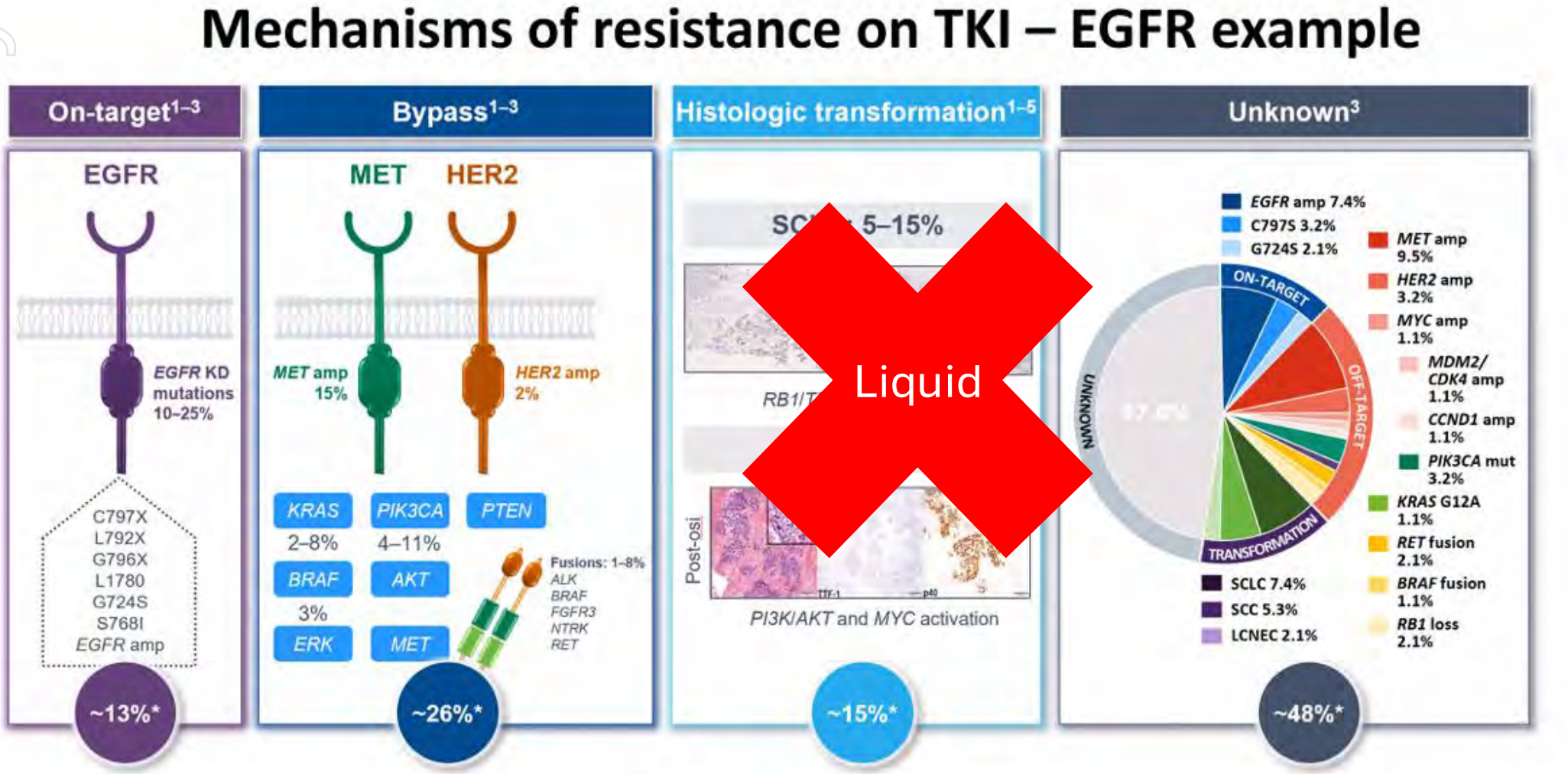




Identifying resistance mechanisms

Pellini – Thor Surg Clin 2020 * Saini – Diagnostics 2018 * Serrano –Cancer Discov 2021 * Siravegna – Nature Rev Clin Oncol 2017 * Alix-Panab

Defining drug resistance in oncogene + NSCLC: a liquid biopsy first approach is preferable – fast, informative



1. Passaro - *Nature Cancer*. 2021;2:377–91; 2. Leonetti - *Br J Cancer*. 2019;121:725–37; 3. Choudhury - *J Thorac Oncol*. 2023;18:463–75; 4. Leonetti - *Front Oncol*. 2021;11:642190; 5. Schoenfeld - *Clin Cancer Res*. 2020;26:2654–63.

TOGA
Thoracic Oncology Group Australasia

Proof of concept:

Using liquid biopsy to identify emerging treatment resistance BEFORE scan progression, THEN add new therapy To DELAY clinical resistance

Breast Cancer Example. Blood ESR1 and camizestrant: SERENA-6 study

2025 ASCO ANNUAL MEETING

SEREN

Camizestrant + CDK4/6 inhibitor for the treatment of emergent *ESR1* mutations during first-line endocrine-based therapy and ahead of disease progression in patients with HR+/HER2– advanced breast cancer: Phase 3, double-blind ctDNA-guided SERENA-6 trial

Nicholas Turner*
Royal Marsden Hospital, London, UK
Additional authors:
Erica Mayer, Yeon Hee Park, Wolfgang Janni, Cynthia Ma, Massimo Cristofanilli, Giampaolo Bianchini, Kevin Kalinsky, Hiroji Iwata, Stephen Chia, Peter A. Fasching, Adam Brufsky, Zbigniew Nowecki, Javier Pascual, Lionel Moreau, Shin-Cheh Chen, Sasha McClain, Steven Fox, Cynthia Huang Bartlett, François-Clément Bidard*
*Contributed equally

- Female/male patients with ER+/HER2– ABC*
- All patients that have received AI + CDK4/6i (palbociclib, ribociclib, or abemaciclib) as initial endocrine-based therapy for ABC for at least 6 months
- *ESR1*m detected in ctDNA with no evidence of disease progression

R 1:1
N=315

- Camizestrant (75 mg qd) + continuing CDK4/6i + placebo for AI
- Continuing AI (anastrozole/letrozole) + CDK4/6i + placebo for camizestrant

Stratification factors
- Visceral vs non-visceral
- *ESR1*m detection at first test vs at a subsequent test
- Time from initiation of AI + CDK4/6i to randomization: <18 vs ≥18 months
- Palbociclib vs ribociclib vs abemaciclib

Primary endpoint

PFS by investigator assessment (RECIST v1.

Secondary endpoints
- PFS2**
- OS**
- Safety
- Patient-reported outcomes

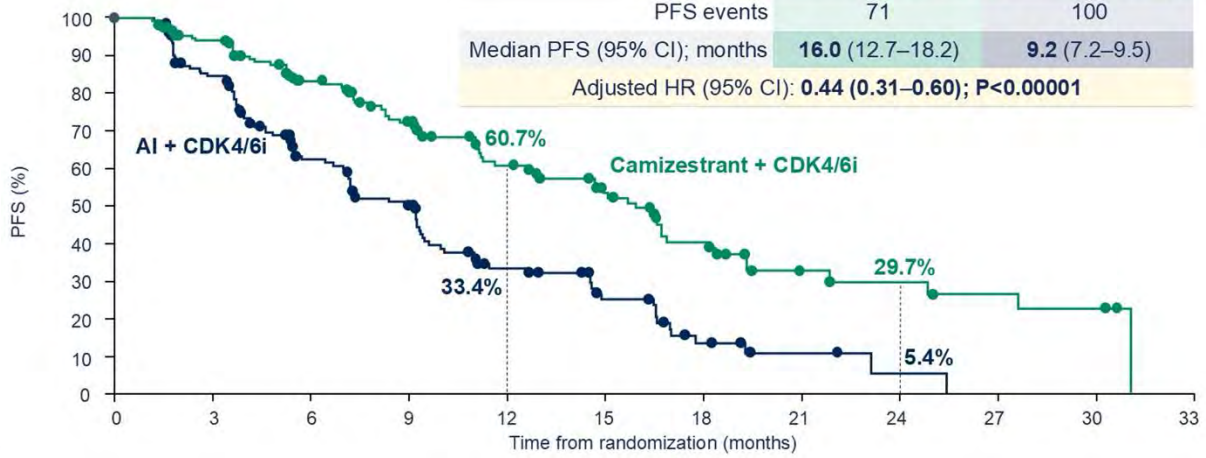
ORIGINAL ARTICLE

First-Line Camizestrant for Emerging *ESR1*-Mutated Advanced Breast Cancer

F.-C. Bidard,[1] E.L. Mayer,[2] Y.H. Park,[3] W. Janni,[4] C. Ma,[5] M. Cristofanilli,[6] G. Bianchini,[7] K. Kalinsky,[8] H. Iwata,[9] S. Chia,[10] P.A. Fasching,[11] A. Brufsky,[12] Z. Nowecki,[13] J. Pascual,[14] L. Moreau,[15] S.-C. Chen,[16] N. Karadurmus,[17] E.N. Gal-Yam,[18] K.H. Jung,[19] S. Pernas,[20] S. McClain,[21] W. He,[22] T. Klinowska,[23] C. Huang-Bartlett,[21] and N.C. Turner,[24] for the SERENA-6 Study Group*

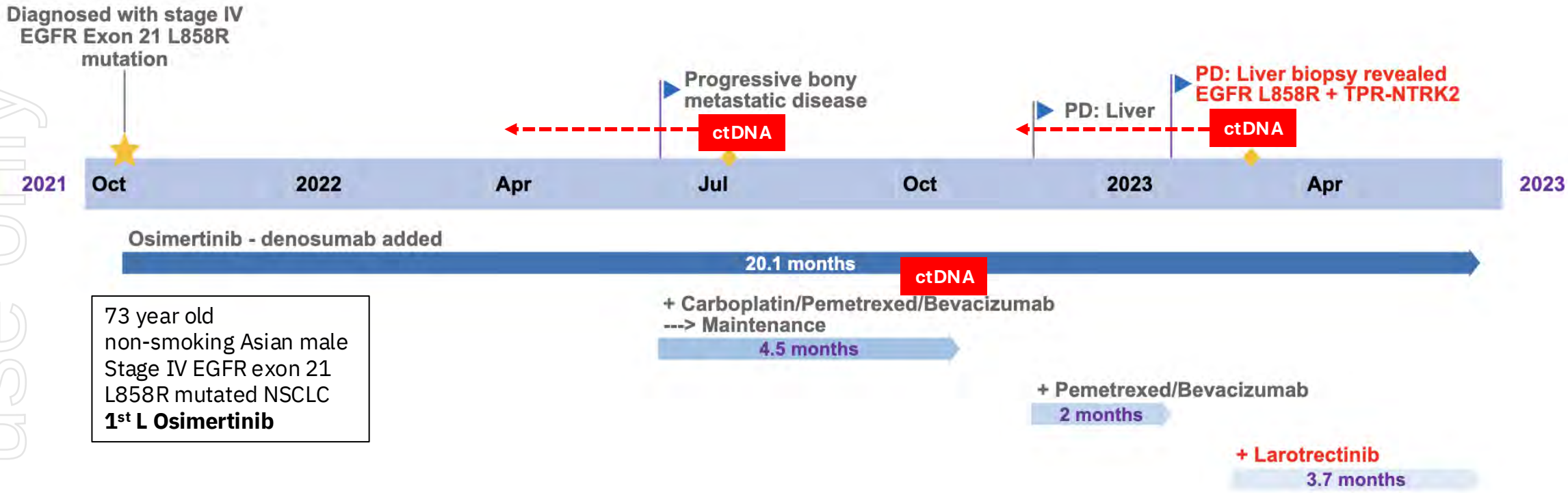
PFS on CT prolonged by 7 months

	Camizestrant + CDK4/6i (N=157)	AI + CDK4/6i (N=158)
PFS events	71	100
Median PFS (95% CI); months	**16.0** (12.7–18.2)	**9.2** (7.2–9.5)
Adjusted HR (95% CI): **0.44 (0.31–0.60); P<0.00001**		



1. Bidard FC, et al. *Lancet Oncol* 2022;23:1367–77; 2. Fribbens C, et al. *Ann Oncol* 2018;29:145–53; 3. Oliveira M, et al. *Lancet Oncol* 2024;25:1424–39.

Case Example – EGFR Exon 21 MT



- ctDNA was informative at progression (co-mutations), cleared with response after chemo.
- Acquired NTRK2 fusion on tissue biopsy
- Earlier use of ctDNA may have identified emerging resistance before clinical progression

1st L, first line; ctDNA, circulating tumour DNA; PD, progressive disease.

Current Approaches to delay EGFRm TKI Resistance

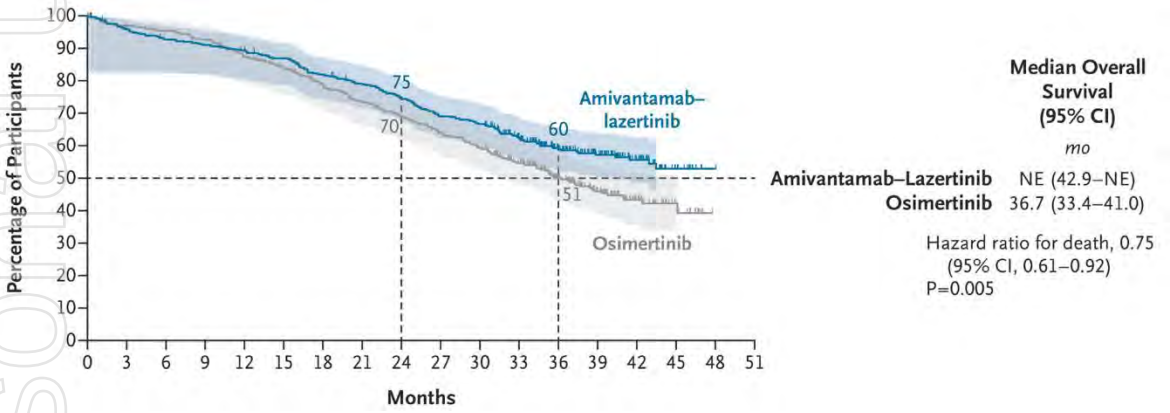
Combine EGRFm TKIs with other anticancer treatments in ALL patients (**empirical approach – one size fits all**)

- Using
  - New targeted agents (e.g. amivantinab/lazertinib in MARIPOSA trial)
  - Chemotherapy (FLAURA2 trial)

Benefit observed in PFS and overall survival, but at cost of toxicity (MARIPOSA >> chemotherapy)

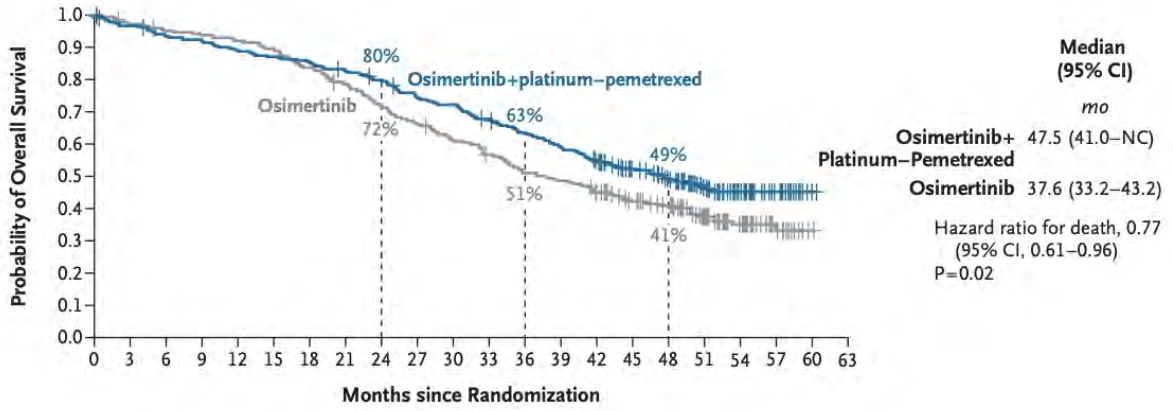
Optimal approach – personalised. Escalate/add therapy based on ctDNA predictor.

MARIPOSA trial – Overall survival



N Engl J Med 2025;393:1681-1693

FLAURA2 trial- Overall survival

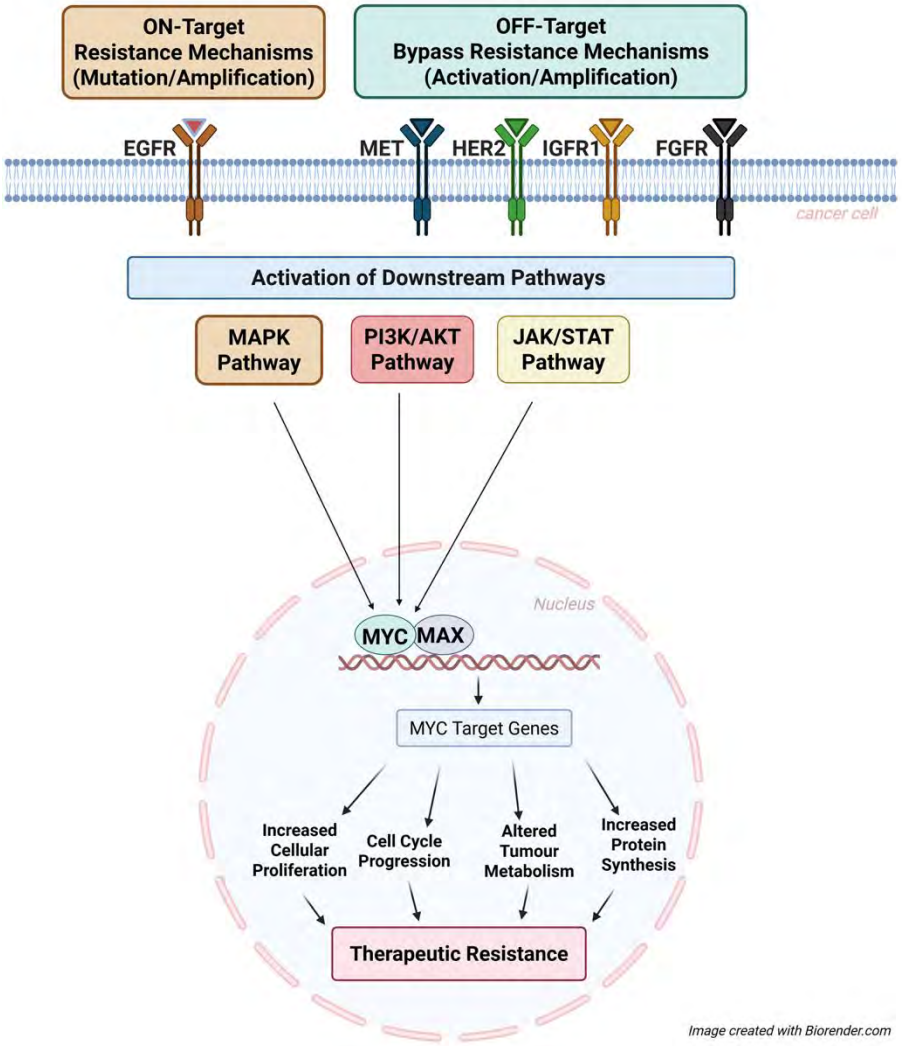


N Engl J Med 2026;394:27-38.

What is Needed to Delay TKI Resistance?

Any successful treatment MUST

- **Be able to prevent/delay bypass signalling** - block signalling downstream of all signalling pathways (e.g. MYC/MAX)
- **Be able to target the different resistance subclones together** – need to target multiple resistance pathways, not just a single pathway
- **Be able to be used indefinitely** – no lifetime or dose cycle limits for patients (e.g. doxorubicin or platinum drugs)
- **Moderate additional toxicity** – patient quality of life is paramount
- **Be cost-effective** – otherwise who pays for the practice changing Phase 3 trials?



Evolutionary approach to treating EGFRm NSCLC

- Upfront TKI. Osimertinib in Australia
- Use ctDNA to identify risk of clinical progression
- Add or change therapy before clinical progression develops
  - Enabling delay in symptomatic deterioration
  - Prolonging disease control and ultimately survival

- National ctDNA study: ASPiRATION-2L study (reactive)
- HARNESS-1 study (proactive)

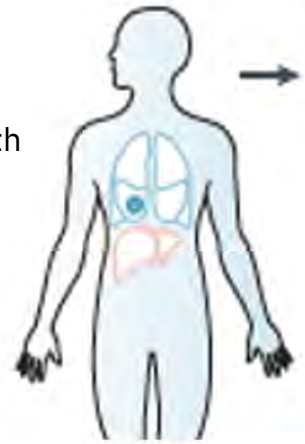
ASPiRATION-2 Liquid: Design

B Solomon (PI, PMCC), N Pavlakis – study co-chairs

TOGA
Thoracic Oncology Group Australasia

Progression on targeted therapy

Target 500 patients Nationally with oncogene driven NSCLC



Baseline ctDNA +/- tissue

ctDNA on treatment

ctDNA on treatment

Disease progression

Disease progression

Disease progression

ctDNA informs treatment at each point of disease progression

Response monitoring

Prognostication

And so forth

Aims

Feasibility

Clinical

Economic

No new actionable alteration (estimate 30%)

Liquid biopsy uninformative (estimate 30%)

New actionable alteration identified (estimate 40%)

Genomically informed therapy

Treatment Allocation

Standard of Care (SoC) Empiric therapy Chemotherapy and/or immunotherapy

TOGA Umbrella Trial: N = 150 Continue existing targeted therapy + add chemo or ADC

Drug A + Chemo

Drug B + Chemo

Drug C + Chemo

PBS: Targeted therapy

Access Program Targeted therapy

TOGA Trials Targeted therapy

$14.7 million MRFF Grant 2025

HARNESS-1. HAlt Resistance in NSCLC with EGFR Sensitising mutationS

A Racura Oncology sponsored Phase 1 trial of RC220 + osimertinib in EGFRm NSCLC patients who are tumour positive by ctDNA with the aim of delaying TKI resistance

- **Endpoints:** Safety, maximum tolerated combined dose, changes in ctDNA positivity, progression free survival and overall survival
- **Sites:** Five in Australia (Sydney, Melbourne, Brisbane)
- **Patients:** ~150 ctDNA screening, Phase 1a (12-40), Phase 1b (40)
- **Time:** Dose Escalation (9-12 months), Expansion (~9 months)
- **First patient:** Q1 2026 (assuming all approvals granted as expected)

HARNESS-1 study has approval at Monash Health, with more Australian sites to follow in the coming months

EGFRm, epidermal growth factor receptor mutant; NSCLC, non-small cell lung cancer; ctDNA, circulating tumour DNA; TKI, tyrosine kinase inhibitor

HARNESS-1. Phase 1 Trial Overview

Proof of concept trial demonstrating that RC220 can delay resistance to TKIs

ctDNA Screening

Screen EGFRm NSCLC patients on osimertinib (Osi) maintenance for tumour presence by ctDNA (~100 patients)

Interventional

Dose Escalation

Open label dose escalation & safety study (12-40 patients)

RC220 dose escalation + standard Osi to identify the maximum tolerated combined dose (MTCD)

Dose Expansion

Blinded & randomised two dose levels to identify optimal RC220 + standard Osi dose (~40 patients)

Lower Dose RC220 + Osi

Higher Dose RC220 + Osi

Optimal RC220 + Osi Dose Progression Free Survival (PFS)

Osi, osimertinib; ctDNA, circulating tumour DNA

HARNESS-1 Key Eligibility Criteria

- **Inclusion**
  - **Diagnosed with EGFRm NSCLC and stable on osimertinib for >6 months**
  - ECOG 0 or 1
  - 18 to 80 years old
  - **Positive or increasing levels of EGFRm ctDNA**
  - Adequate haematological, liver, kidney and cardiac function
- **Exclusion**
  - Pregnant or lactating women
  - Recent anti-cancer treatments except osimertinib
  - Persisting Grade 2 adverse events except alopecia and neuropathy
  - Severe or uncontrolled cardiac disease
  - Recent surgery

ECOG, Eastern Cooperative Oncology Group

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Competitive landscape

Potential of RC220 use in EGFRm NSCLC

Market potential for EGFR-mutated NSCLC

There is a large (>$10b) existing market for TKIs targeting EGFRm NSCLC

Target

Epidermal growth factor receptor (EGFR)

Incidence NSCLC[2]

China 890,000
US/EU 560,000

EGFR mutations[2]
China 445,000
US/EU 95,000

Major players – 3rd generation TKIs

Brand	Generic	Manufacturer	2025 sales (US$m)[1]
Tagrisso®	Osimertinib	AstraZeneca	7,200
Amelie®	Aumolertinib	Hansoh	660
Ivesa®	Furmonertinib	Shanghai Allist	>700
Lascluse®	Lazertinib	Janssen	730

Opportunity

Delaying the emergence of resistance would be of great value to patients and marketers of EGFR inhibitors - RC220 could be a significant addition to existing standards of care

Market dynamics

- First and second-generation EGFR TKIs superseded by third-generation EGFR TKIs
- Tagrisso® dominates (patent expiry - 2032)[2]
- AZ's major Pharma rival is Janssen with Lascluse® in combination with Rybrevant® (cMET/EGFR antibody)[2]
- Numerous third generation TKIs in development, especially in China
- When patients progress on TKIs they have few options and limited life expectancy[2]

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1. AstraZeneca Annual Report 2025; Jannsen Annual Report 2025; Hansoh Annual Report 2025; Shanghai Allist Annual report 2025 2. Frost & Sulivan, Innovative Small Molecule Drug Market Report. (2024) | EGFRm, epidermal growth factor receptor mutant; NSCLC, non-small cell lung cancer; EGFR, epidermal growth factor receptor; MET, mesenchymal–epithelial transition factor; TKIs, tyrosine kinase inhibitors; US, United States; EU, Europe

EGFRm NSCLC TKI competitive landscape

Drug name	Presenter	Target(s)	Mutation subtypes	Gen	Company	Clinical stage	Approvals	2024 sales (US$m)
Osimertinib	**Tagrisso®**	**EGFR, HER2, HER4**	**Ex19del, L858R, T790M**	**3rd**	**Astrazeneca**	**Approved**	**US, EU, CN**	**6580**
Dacomitinib	Vizimpro®	EGFR, HER2, HER4	Ex19del, L858R	2nd	Pfizer	Approved	US, CN	~200
Afatinib	Gilotrif®	EGFR, HER2, HER4	Sensitive mutations	2nd	Boehringer Ingelheim	Approved	US, CN	690 (2022)
Erlotinib	Tarceva®	EGFR	Ex19del, L858R	1st	Astellas/ Roche	Approved	US, CN	~1200
Gefitinib	Iressa®	EGFR	Ex19del, L858R	1st	Astrazeneca	Approved	US, CN	~250
Icotinib	Conmana®	EGFR	Ex19del, L858R	1st	Betta Pharma	Approved	CN	~100
Befotertinib	Surmana®	EGFR	Ex19del, L858R, T790M	3rd	Betta Pharma	Approved	CN	<30
Rezivertinib	Ruibida®	EGFR	T790M	3rd	Beta Pharma	Approved	CN	Approved in 2024
Furmonertinib	Ivesa®	EGFR	Ex19del, L858R, T790M	3rd	Allist Pharmaceutical	Approved	CN	~500
Almonertinib	Ameile®	EGFR	Ex19del, L858R, T790M	3rd	Hansoh Pharma	Approved	CN	~500
Rilertinib	Sanrisso®	EGFR	T790M	3rd	Sanhome Pharmaceutical	Approved	CN	Approved in 2024
Lazertinib	**Lazcluze®**	**EGFR, HER2, HER4**	**Ex19del, L858R, T790M**	**3rd**	**Yuhan Corporation / Jansen**	**Approved**	**US, EU, CN**	**141 (Q1 2025)**
Zorifertinib	Zorifer®	EGFR, HER2, HER4	Ex19del, L858R	3rd	Alpha Biopharma/ Astrazeneca	Approved	CN	Approved in 2024

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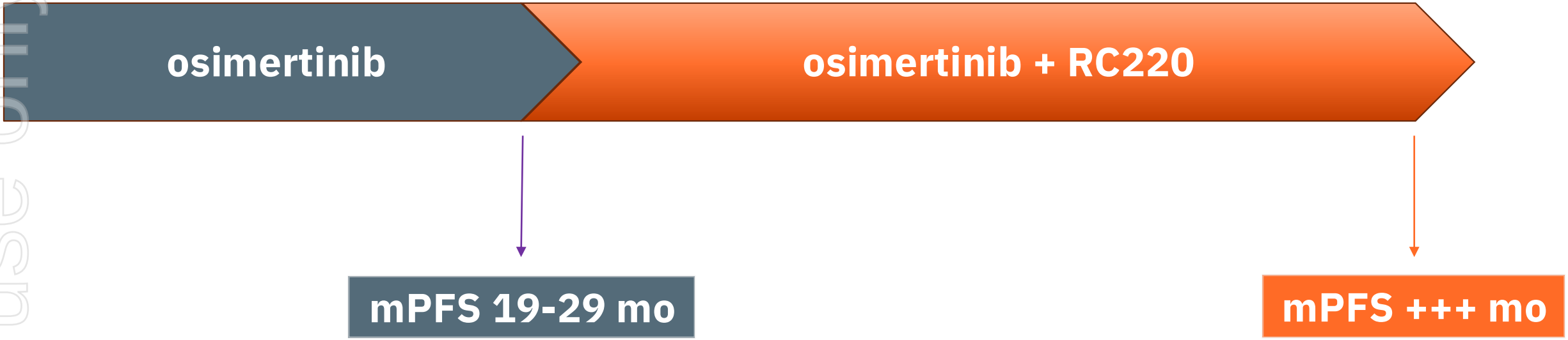
EGFRm, epidermal growth factor receptor mutant; HER2, human epidermal growth factor receptor 2; HER4, human epidermal growth factor receptor 4; US, United States; EU, European Union; CN, China

Our goal

Delay or prevent resistance to osimertinib



Use endpoints like mPFS as shorter-term regulatory-approvable endpoints

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mPFS, median progression free survival; mo, months

Strategic positioning

Broad market opportunity with multiple positioning approaches

Market leaders

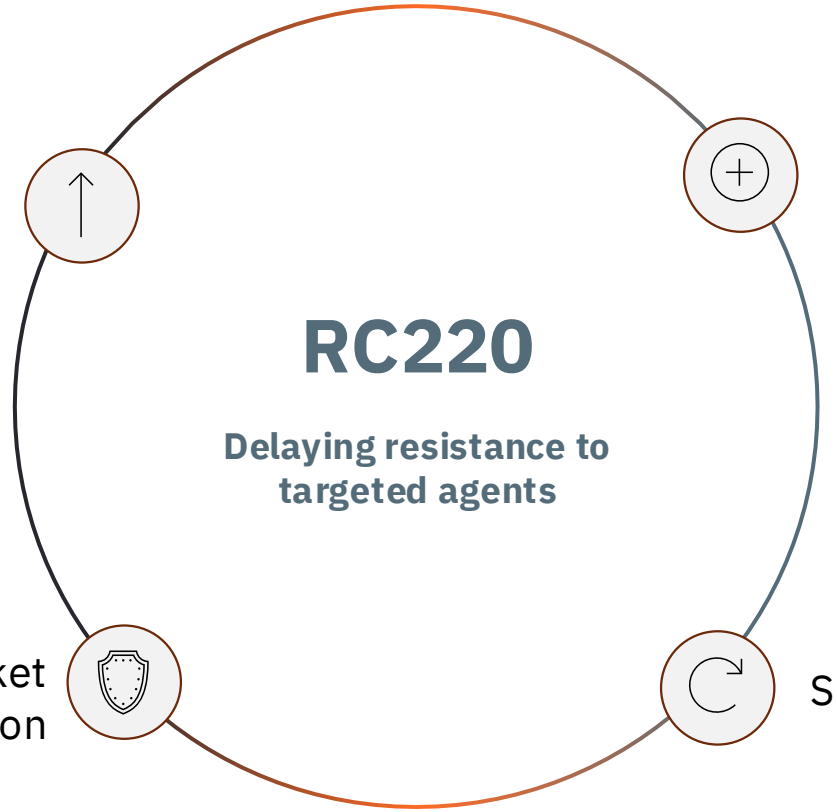
Increase use of existing targeted agents

Protect existing market from competition

New entrants

Provide competitive edge to new targeted agents

Supplant market leaders



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Commercialisation

HARNESS-1 regulatory pathways and approval opportunities for RC220

RC220 regulatory plan in EGFRm NSCLC

Potential pathway to approval of RC220 as a resistance EGFRm TKI delaying treatment

- Proceed with HARNESS-1 until end of dose escalation (MTD determination)
- Perform interim analysis
- Present data to US FDA at “End of Phase 1” meeting
  - Confirm study data are acceptable to FDA
  - Discuss Phase 1b/2 study design
  - Discuss Fast-Track and/or Breakthrough Designation

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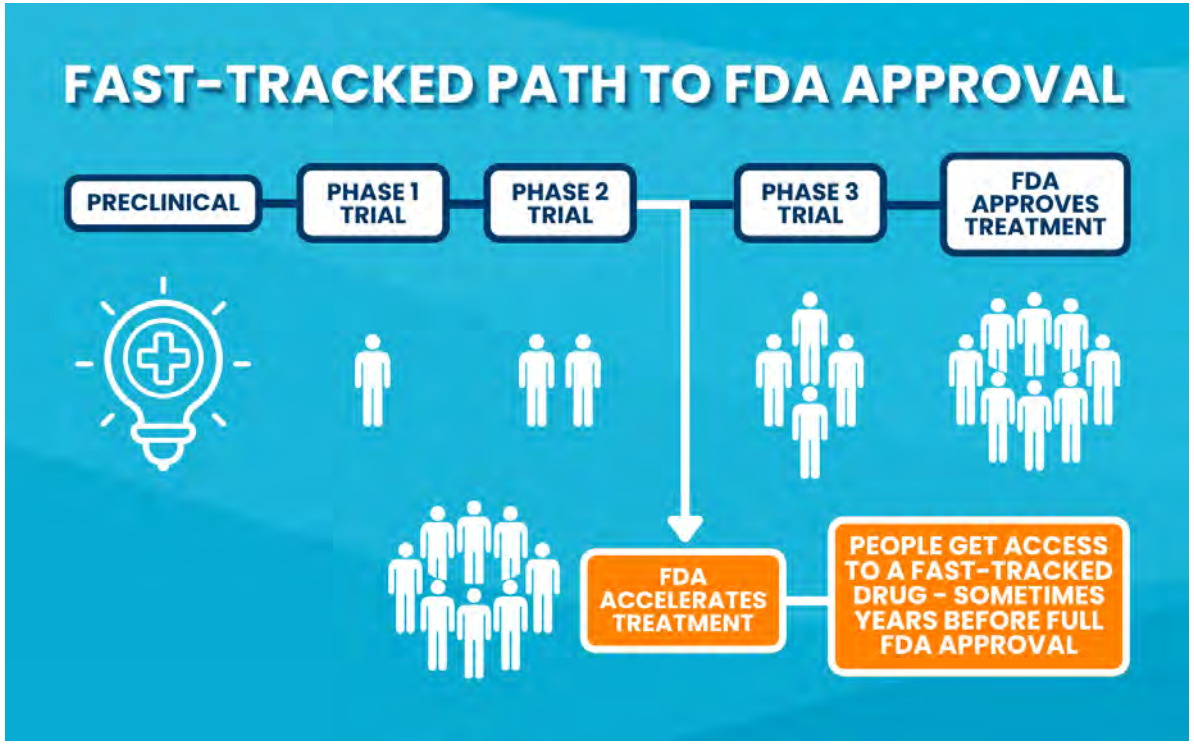
EGFRm, epidermal growth factor receptor mutant; TKIs, tyrosine kinase inhibitor; MTD, maximum tolerated dose; FDA, Food & Drug Administration

US FDA options to accelerate approval

If the data supports it, there is the possibility of early approval of RC220

- Priority Review
- Breakthrough Designation
- Real-Time Oncology Review
  - Allows Sponsors to submit clinical data in stages for rolling review
  - ~26 weeks target review time

Zongertinib approval example



FDA, Food & Drug Administration

Zongertinib (HERNEXEOS®, Boehringer Ingelheim)

Case study of FDA accelerated approval from 71 patient Phase 1 dataset

Fast Track Status (US) – Dec 23

Breakthrough Therapy Status (US, China)- Aug 24

Priority Review (US) – Feb 25

Approved US & China – Aug 25

2021 | 2022 | 2023 | 2024 | 2025 | 2026

Beamion LUNG-1 (Phase Ia/b)

71 pts

ORR 71%
mPFS 12 mo

Beamion LUNG-2 (Phase III)

PANTUMOR-1 (Phase II)

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Lee A (2026) Drugs, 86(3):397-401 | FDA, Food & Drug Administration; US, United States;
ORR, overall response rate; mPFS, median progression free survival; mo, months

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Questions

RACURA ONCOLOGY

Phase 3 AML clinical program

Dr Anupa Kudva, Medical Director

Agenda: AML

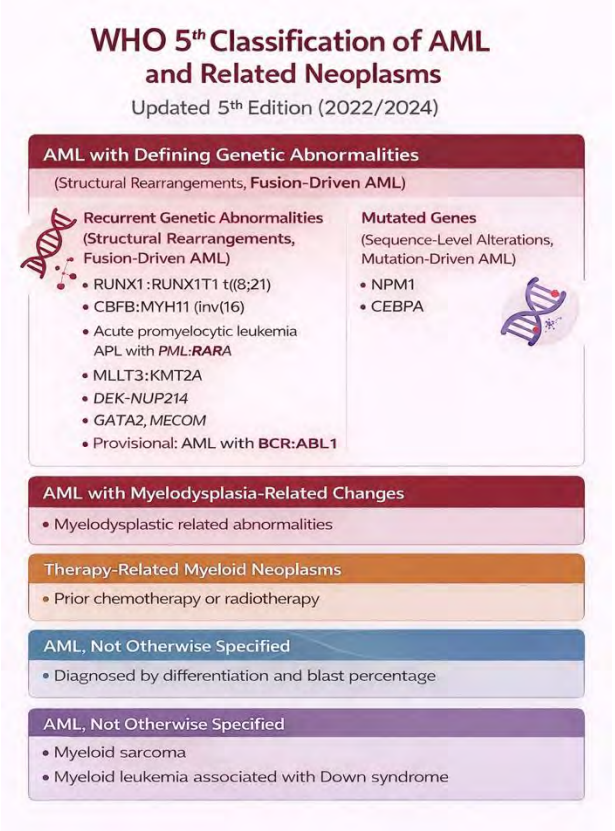
Topic	Presenter
AML landscape: Overview and current treatment options	Dr Anupa Kudva
History of bisantrene in AML	
Clinical benefit of second-line AML therapies	
Biology and clinical relevance of MYC in AML	
Rationale for use of RC220 in AML	
Clinical development: Study roadmap and design	
Commercialisation: Market positioning and expansion opportunities	

Acute myeloid leukaemia

Molecular heterogeneity creates both challenges and opportunities

Rare but aggressive blood cancer

- Rapid proliferation of immature myeloid cells in the bone marrow
- Globally ~145,000 new cases annually
  - Approximately 22,000 new AML cases are diagnosed each year in the US
- Incidence increases sharply with age (median age 68 years)
- Heterogeneous disease



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AML, acute myeloid leukaemia; US, United States

Frontline treatment landscape in AML

Intensive regimens, high toxicity, and frequent relapse

	Fit	Unfit
% of AML patients	40%	60%
Age range	<70–75 years	≥75 years
ECOG Performance status (ability to carry out daily activities)	0–2	≥3
Comorbidities / frailty	Limited	Significant
Upfront therapy	Intensive induction (e.g., 7+3 cytarabine + anthracycline backbone) ± Targeted agents Consolidation Allogeneic stem cell transplant	Lower-intensity regimens (e.g. hypomethylating agent + venetoclax) ± Targeted agents Transplant not feasible
CR rates	~60–80%	~40–70%
Relapse after remission	Approximately 30–60%	Approximately 65-70%
Median time to relapse	~10–18 months (without transplant)	~6-10 months

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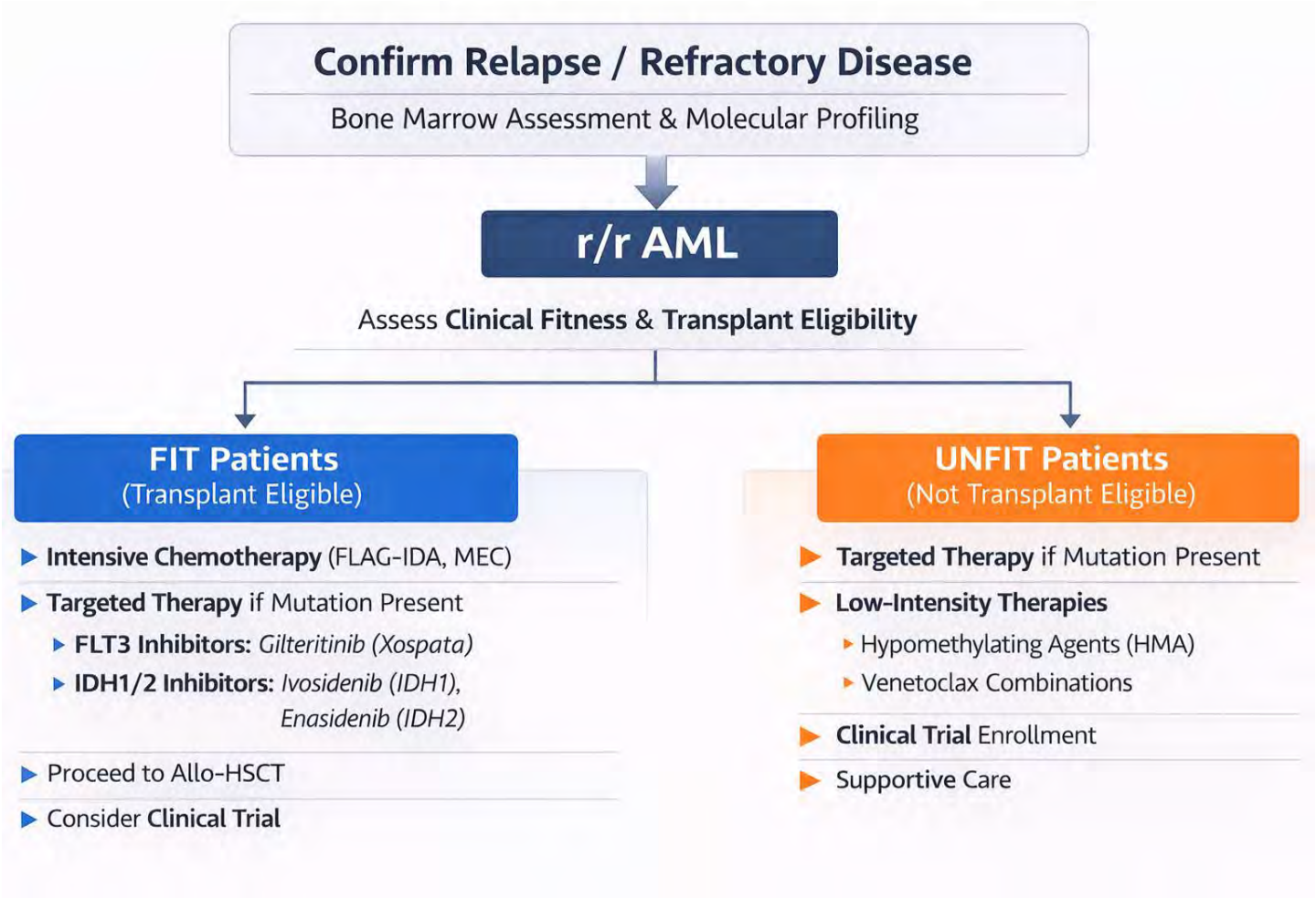
AML, acute myeloid leukaemia; CR, complete remission; ECOG, Eastern Cooperative Oncology Group

Treatment options in r/r AML

Outcomes are poor and therapeutic options are limited



Legend – Abbreviations

FLAG-IDA – Fludarabine, Cytarabine, G-CSF, Idarubicin
MEC – Mitoxantrone, Etoposide, Cytarabine
Allo-HSCT – Allogeneic Hematopoietic Stem Cell Transplant
HMA – Hypomethylating Agent

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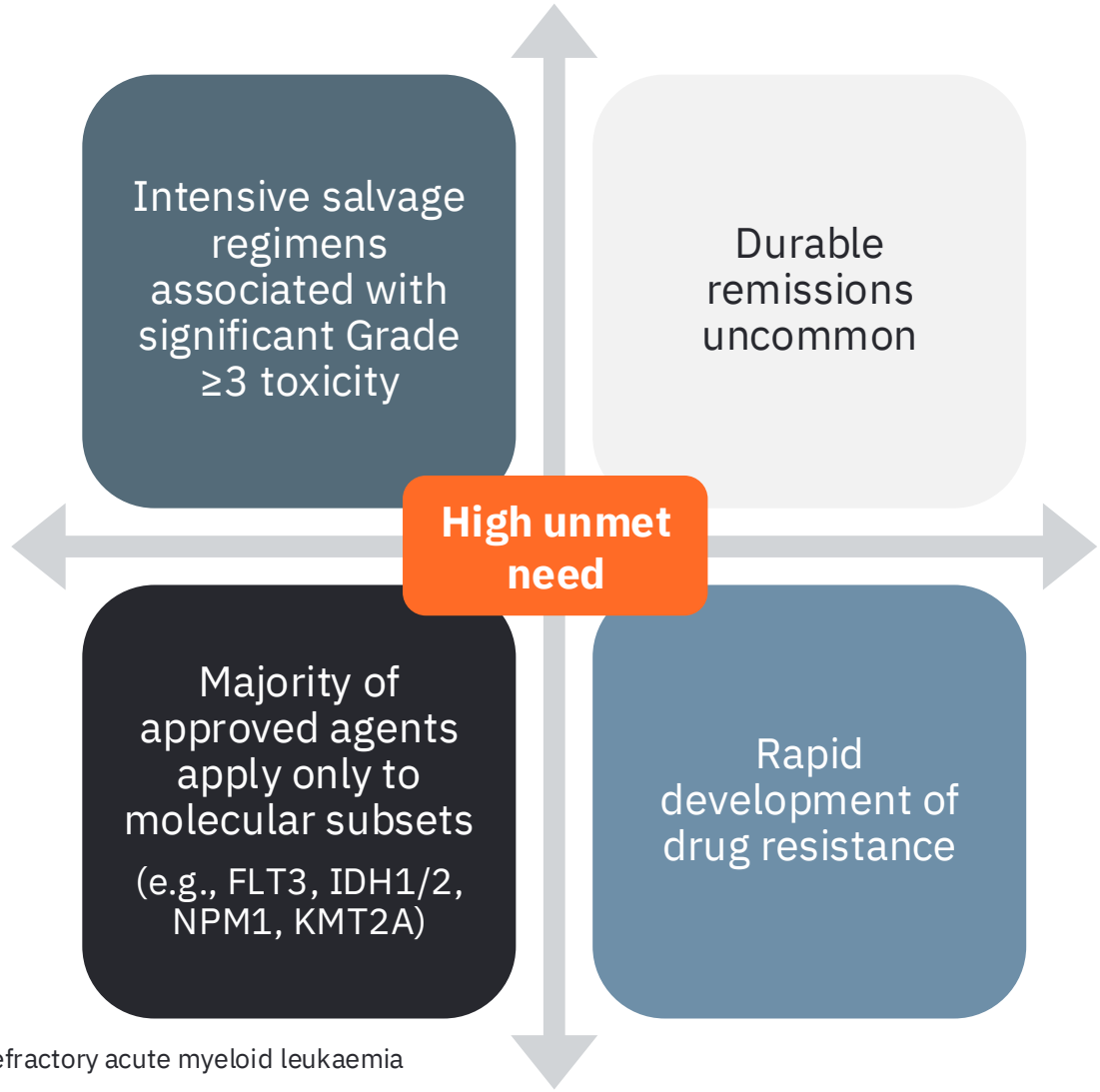
Limitations to current r/r AML treatment paradigm

Why current salvage therapies fall short



r/r AML, elapsed and/or refractory acute myeloid leukaemia

Paediatric AML

Rare but underserved patient population

Accounts for ~15–20% of childhood leukaemias

Different disease from adult AML

- Higher upfront intensity
- Fewer targetable mutations

Approximately one-third of children relapse

- Survival after relapse remains below 40%

Significant long-term toxicity burden

- Cardiac, endocrine, fertility complications
- ~10–20-year reduction in life expectancy after transplantation

Critical need for more effective and less toxic therapies in paediatric AML

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AML, acute myeloid leukaemia

History of bisantrene in AML

Existing clinical data supports continued development in AML

Bisantrene approved in France for AML in 1988

- Demonstrated anti-leukaemic activity – 40% CR
  - Minimal cardiotoxicity

(E,E)-bisantrene clinical AML studies

- Two Phase 2 trials in adults
  - 40% ORR
    - ➢ Monotherapy[1] AND in combination with fludarabine and clofarabine[2]
- Paediatric leukaemia study: combination with cytarabine[3]
  - 46% ORR
  - 5/13 AML patients achieved CR
    - ➢ Two patients reported as long-term survivors in 2018[4]

RACURA ONCOLOGY

1. Canaani J et al (2021) Eur J Haematol, 106(2):260-266
2. Danylesko I et al (2025) Br J Haematol,207(4):1425-1434
3. Leblanc T et al (1994) Med Pediatr Oncol, 22(2):119-124
4. Leverger & Bertrand (2018) Leukemia & Hematologic Oncology Conf

AML, acute myeloid leukaemia; CR, complete remission; ORR, overall response rate

History of bisantrene in AML

Existing clinical data supports continued development in AML

Bisantrene approved in France for AML in 1988

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  - o 4
  - o 5/13 AML patients achieved
    - ➢ Two patients reported as long-term survivors in 2018[4]

FDA: Orphan Drug & Rare Paediatric Disease Designation (AML)

Paediatric Priority Review Voucher eligible

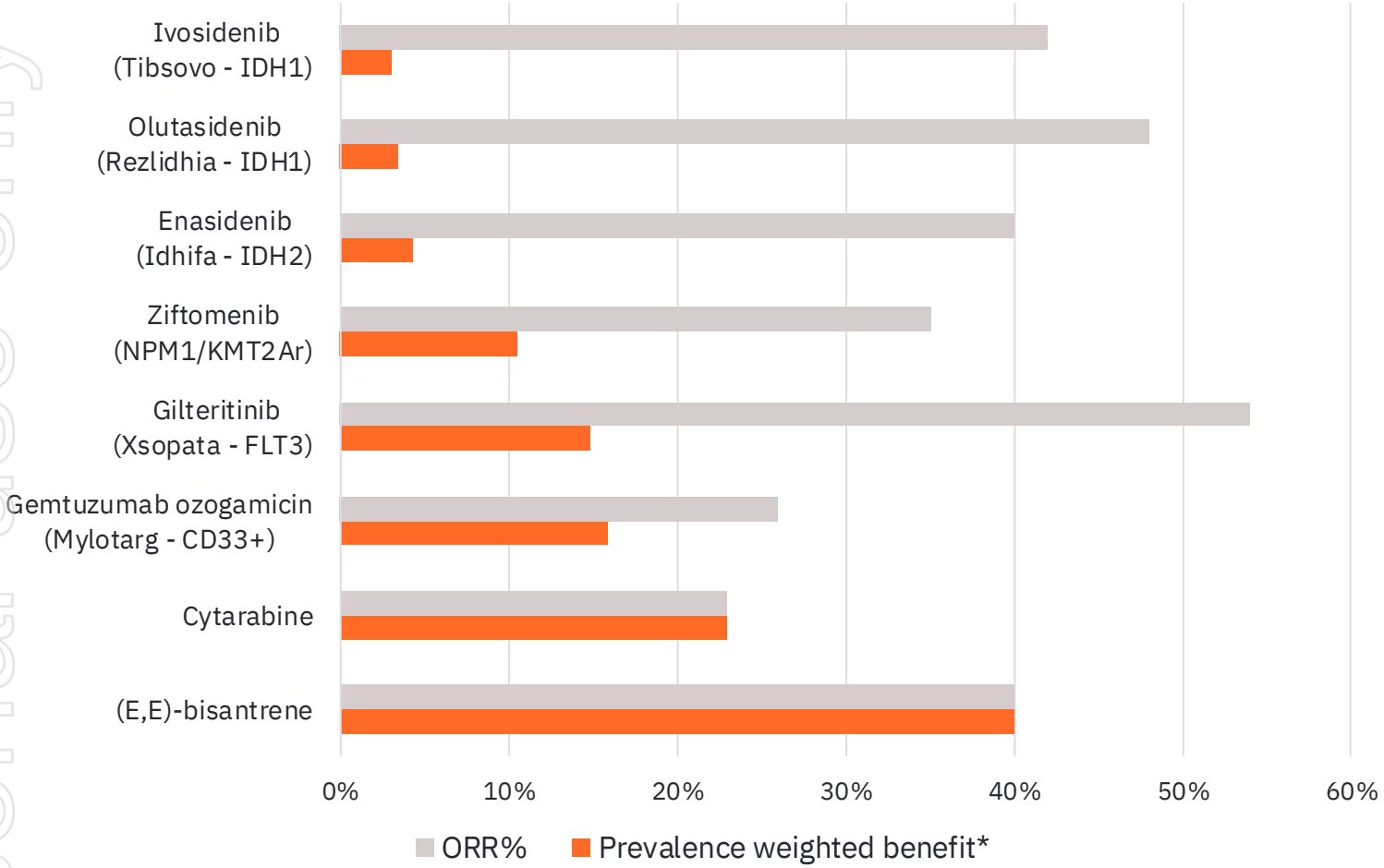
RACURA ONCOLOGY

1. Canaani J et al (2021) Eur J Haematol, 106(2):260-266
2. Danylesko I et al (2025) Br J Haematol,207(4):1425-1434
3. Leblanc T et al (1994) Med Pediatr Oncol, 22(2):119-124
4. Leverger & Bertrand (2018) Leukemia & Hematologic Oncology Conf

AML, acute myeloid leukaemia; CR, complete remission; ORR, overall response rate; FDA, Food & Drug Administration

Clinical benefit of second-line AML therapies

Mutation-specific therapies have limited population-level impact



(E,E)-bisantrene is mutation agnostic

- Broader potential impact across AML
- Less toxicity than standard chemotherapy

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* Prevalence weighted benefit for ORR accounts for patient eligibility based on the prevalence of targeted gene mutations/rearrangements (eg, IDH1, IDH2, FLT3, NPM1, KMT2Ar) or overexpressed proteins (eg, CD33+) in r/r AML. | AML, acute myeloid leukaemia; ORR, overall response rate

MYC expression in AML

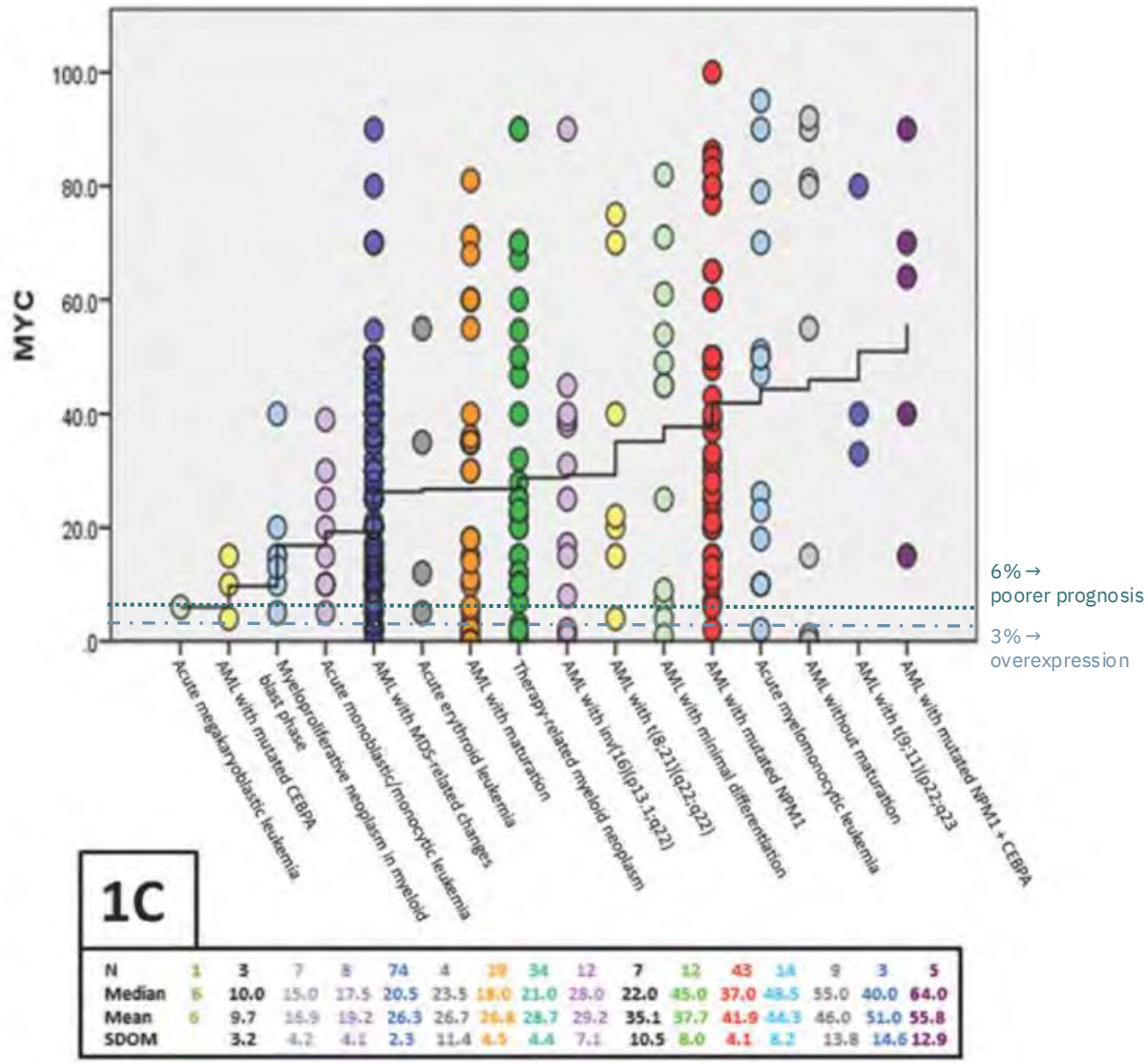
Key oncogenic driver in AML[1]

MYC is frequently upregulated in AML

- MYC overexpression observed in ~90% of AML cases
- Higher expression of MYC is associated with lower overall survival

Implications as a target

- MYC central transcriptional driver in AML
- Broad overexpression of MYC suggests wide applicability across diverse patient subsets



MYC-immunopositivity by IHC by WHO categories: MYC values of 265 patients are plotted against WHO classification-categories.

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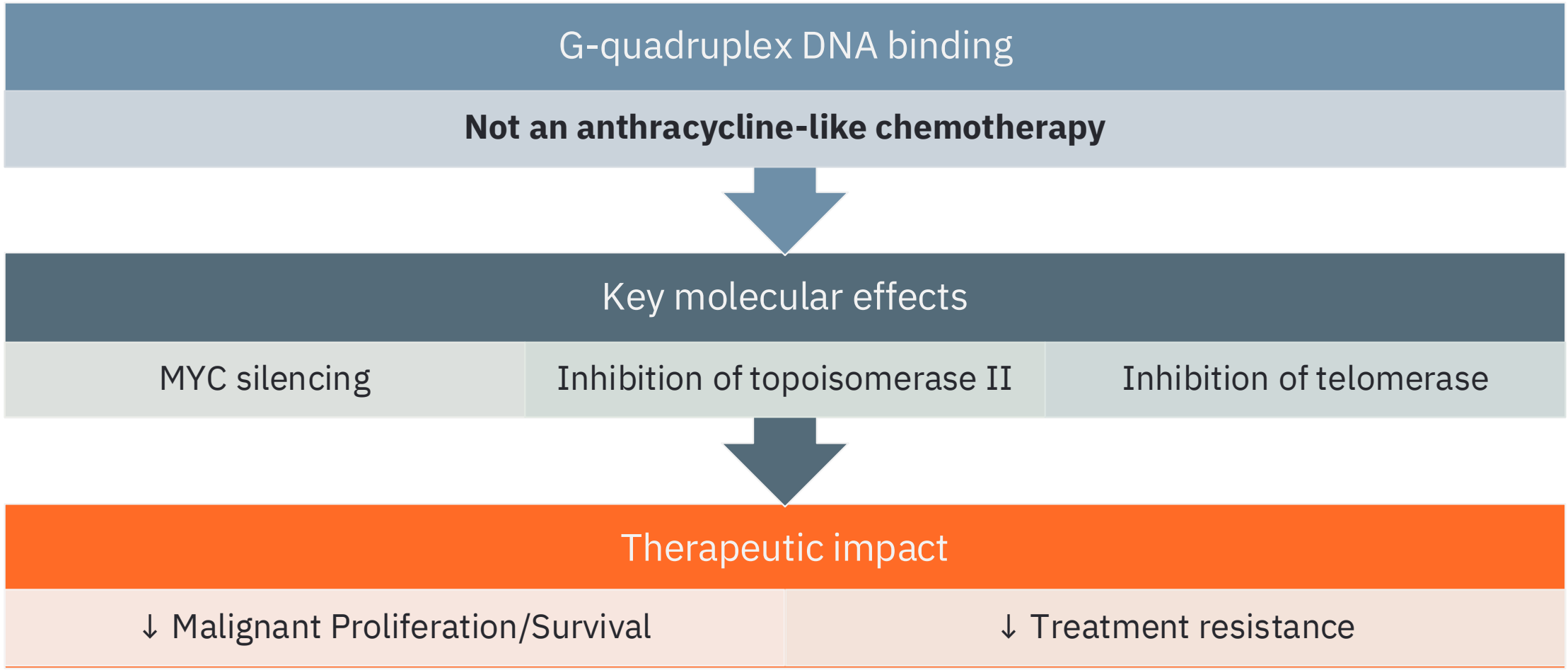
1. Ohanian M et al (2019) Leuk Lymphoma, 60(1):37–48

AML, acute myeloid leukaemia; IHC, immunohistochemistry; WHO, World Health Organisation

Mechanism of action of (E,E)-bisantrene in AML

Novel mechanism with broad anti-leukaemic activity



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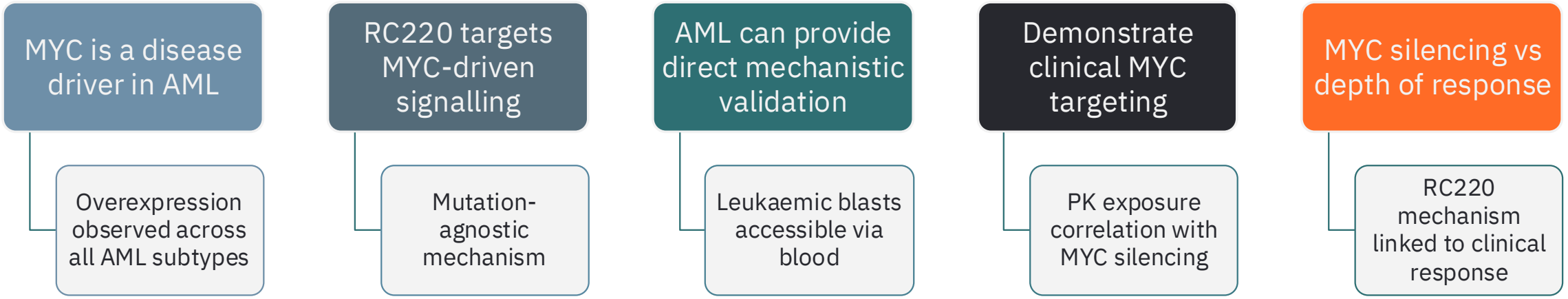
AML, acute myeloid leukaemia

Clinical validation of MYC silencing

Linking biological activity to clinical response



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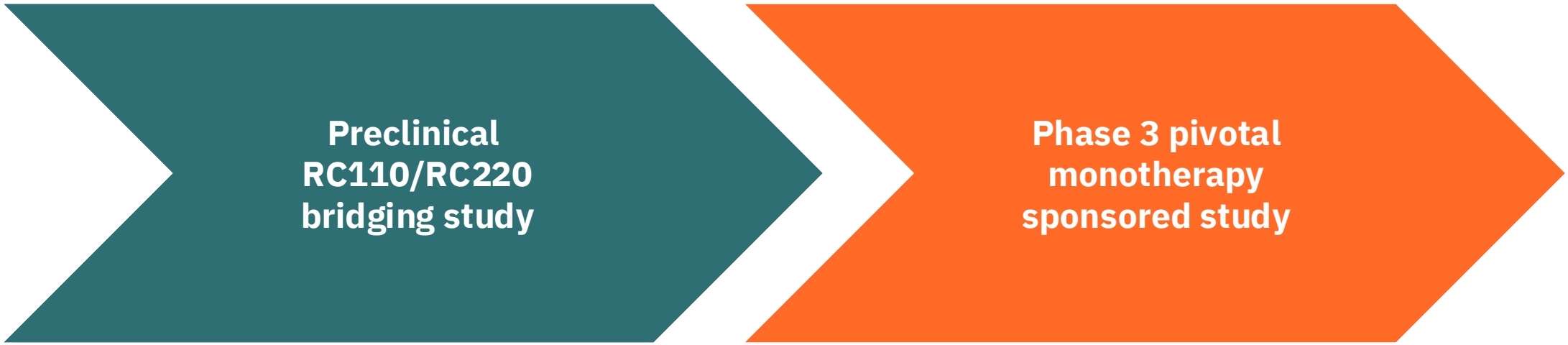
AML, acute myeloid leukaemia; PK, pharmacokinetics

RC220 clinical development strategy in r/r AML

Rapid, low-cost pathway to regulatory approval of RC220



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r/r AML, relapsed and refractory acute myeloid leukemia

Preclinical RC110/RC220 bridging pathway

Building on RC110 to streamline RC220 development

Study design overview

- Preclinical animal PK study
- Head-to-head comparison of RC110 vs RC220
- Primary objective: Demonstrate pharmacokinetic equivalence

Rationale

- Expedite development timeline
- Avoids need for human bridging study
- Align with FDA-accepted pathway

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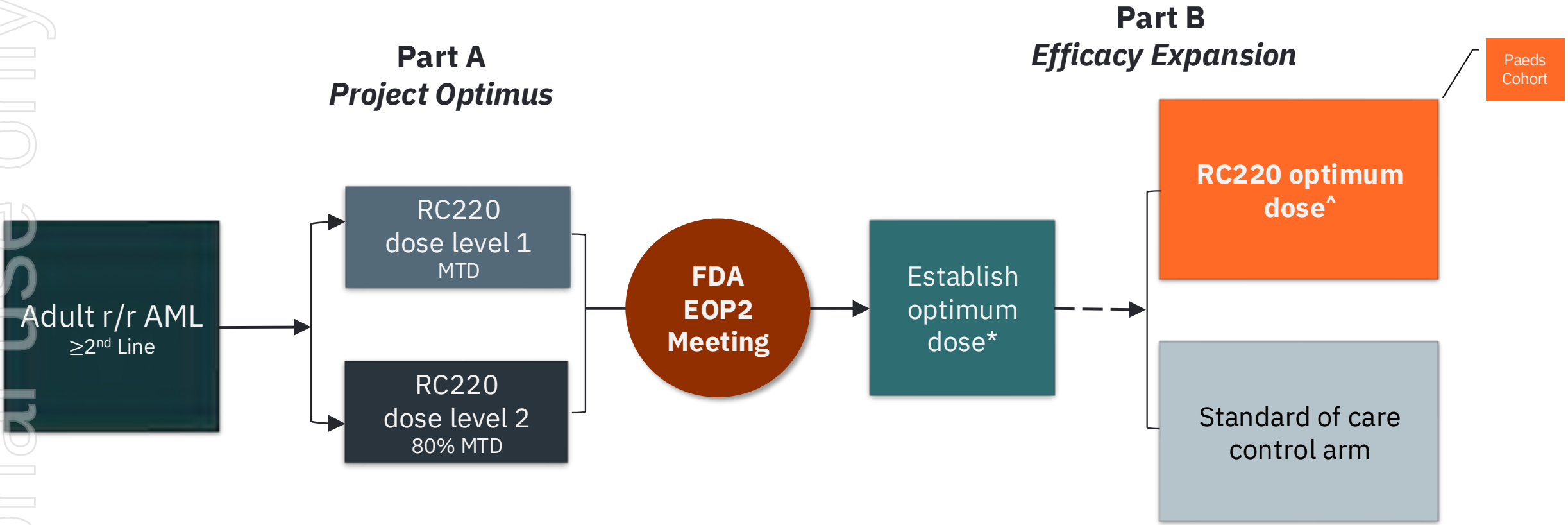
PK, pharmacokinetics; FDA, Food & Drug Administration

Phase 3 adaptive seamless trial design

RC220 monotherapy trial in r/r AML



EOP2, end of phase 2; MTD, maximum tolerated dose; r/r AML, relapsed and refractory acute myeloid leukemia; FDA, Food & Drug Administration
* Criteria: Efficacy, PK equivalence, safety, tolerability.
^ Dichotomous Endpoint, One-Sample Study: 25% base & 40% ORR; p=0.05; power=0.9

Commercial strategy in AML

Rapid, low-cost pathway to regulatory approval of RC220

- A defined orphan market with a clear regulatory pathway
- Efficient path to NDA
- Potential qualification for a Paediatric PRV upon approval ~US$200m[1]
- Establish clinical proof of MYC targeting
- Platform for expansion
  - Frontline AML
  - Paediatric leukaemia
  - Additional MYC-driven cancers

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Source: 1. Cyprium Therapeutics company announcement, 23 February 2026 | PRV, Priority Review Voucher

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Questions

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Protecting innovation & delivering milestones

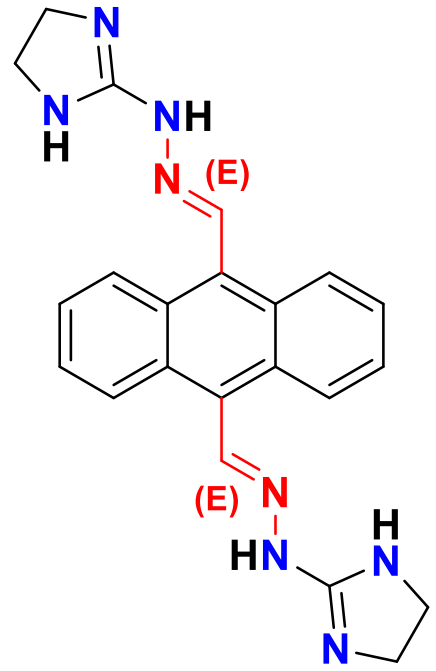
Dr Pete Smith, Executive Chair

IP Overview

Foundation for 20 years of the strongest composition of matter IP protection

Bisantrene photoisomers discovered by Racura Oncology

- Bisantrene found to consist of three photoisomers with different biological and anticancer activities, which rapidly interconvert upon exposure to visible light
- Racura has created a range of manufacturing and processes to deliver pure active (E,E)-bisantrene isomer to patients
- Three patent applications filed
- Granting of composition of matter claims would protect (E,E)-bisantrene for 20+ years
- Composition of matter patents would cover the chemical structure of the active drug in any formulation or dosage form



(E,E)-bisantrene

Composition of matter patents

If granted, these patents will add significantly to value any future licensing or partnering transactions

- Composition of matter patent claims protect the chemical structure of the active drug
- Strongest intellectual property (IP) possible in the pharmaceutical industry
- Composition of matter IP drives significant value uplift for any drug. Why?
  - No generic competition until end of composition of matter patent
  - Can’t be worked around by changing the formulation
  - Possibility of additional patent extension
- Patent life critical
  - Longer the better - increases net present value of drug

What is required for a patent?

Counsel advice is that composition of matter claims meet all the requirements for patent allowance

Novelty	Non-obviousness	Utility
Bisantrene photoisomerism not previously reported	Bisantrene photoisomerism not previously reported	Only (E,E)-isomer active
✓	✓	✓

Racura Oncology has filed three isomer patents

- Isomeric forms of bisantrene
- Pharmaceutical compositions suitable for intravenous administration
- Process for manufacturing and using high purity (E,E)-bisantrene

Conclusion

Composition of matter claims add significantly to (E,E)-bisantrene's value

- Bisantrene can exist in three isomeric forms: (E,E), (E,Z) and (Z,Z)
  - Photoisomerisation discovered by Racura Oncology
- Only (E,E)-bisantrene shown to have potent anticancer activity
- Three patent applications filed on isomers, including one with composition of matter claims
- Patent counsel advice is these patents meet the three requirements for allowance
  - Non-obviousness, novelty, and utility
- Expedited review process for rapid assessment
- Composition of matter claims are the strongest IP protection and if granted will add significantly to value any future licensing or partnering transactions

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Biotech valuation case studies

Dr Pete Smith, Executive Chair

Valuing biotechnology companies

Biotech is a specialised investment category with huge upside and risk

Valuing biotech is inherently challenging

- Often risk-adjusting a single asset which often has a binary outcome (success/fail)
- The market potential is often unknowable with many uncertainties in forecasts
  - Competitors, changing standard of care, politics, clinical results, etc.

Sophisticated investors turn to comparable companies (comps) to make decisions on value

MYC can be compared to another target, KRAS

- KRAS is a g-protein involved in the signalling of several important ligands e.g. EGFR, PDGF, VEGF etc.
- KRAS was, until recently, considered 'undruggable'
- Mutations in RAS family (primarily KRAS) occur in approximately 19% of all malignancies[1]
  - Most commonly occurring in pancreatic, lung and colorectal cancers

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1. Prior I et al (2020) Cancer Res, 80(14):2969–2974 | KRAS, Kirsten rat sarcoma viral oncogene homolog; EGFR, epidermal growth factor receptor; PDGF, platelet-derived growth factor; VEGF, vascular endothelial growth factor; RAS, Rat Sarcoma

Targeting the undruggable – Revolution Medicines

Extreme value placed on clinical success by sophisticated investors

Revolution Medicines (NASDAQ: RVMD)

- Price spike due to takeover rumours at JP Morgan healthcare conference
- MCap ~US$20B
- Cash US$1.9B

Developing several KRAS targeted drugs

- Daraxonrasib, pan-RAS, Phase 3
- Eloronrasib, G12C, Phase 1
- Zoldonrasib, G12D, Phase 1/2

The ‘undruggable’ has been drugged

- >70 investigational drugs listed on www.clinicaltrials.gov

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Mcap, market cap; KRAS, Kirsten rat sarcoma viral oncogene homolog; RAS, RAt Sarcoma virus

Targeting the undruggable – Erasca Inc

Extreme value placed on clinical success by sophisticated investors

Erasca Inc. (NASDAQ: ERAS)

- Benefited from RVMD price momentum and interest in RAS as a target
- Market Cap: US$4.8B
- US$300M in cash end Sept 25
- Raised additional US$260M in Jan 2026

Pipeline drugs

- ERAS-0015: pan-RAS, Phase 1
- ERAS-4001: pan-KRAS, Phase 1
- ERAS-0012: EGFR D2/D3 mAb, Phase 1

RACURA ONCOLOGY

RAS, RAt Sarcoma virus; KRAS, Kirsten rat sarcoma viral oncogene homolog; EGFR, epidermal growth factor receptor; mAB, monoclonal antibody

Targeting the undruggable – Mirati Therapeutics

Extreme value placed on clinical success by sophisticated investors

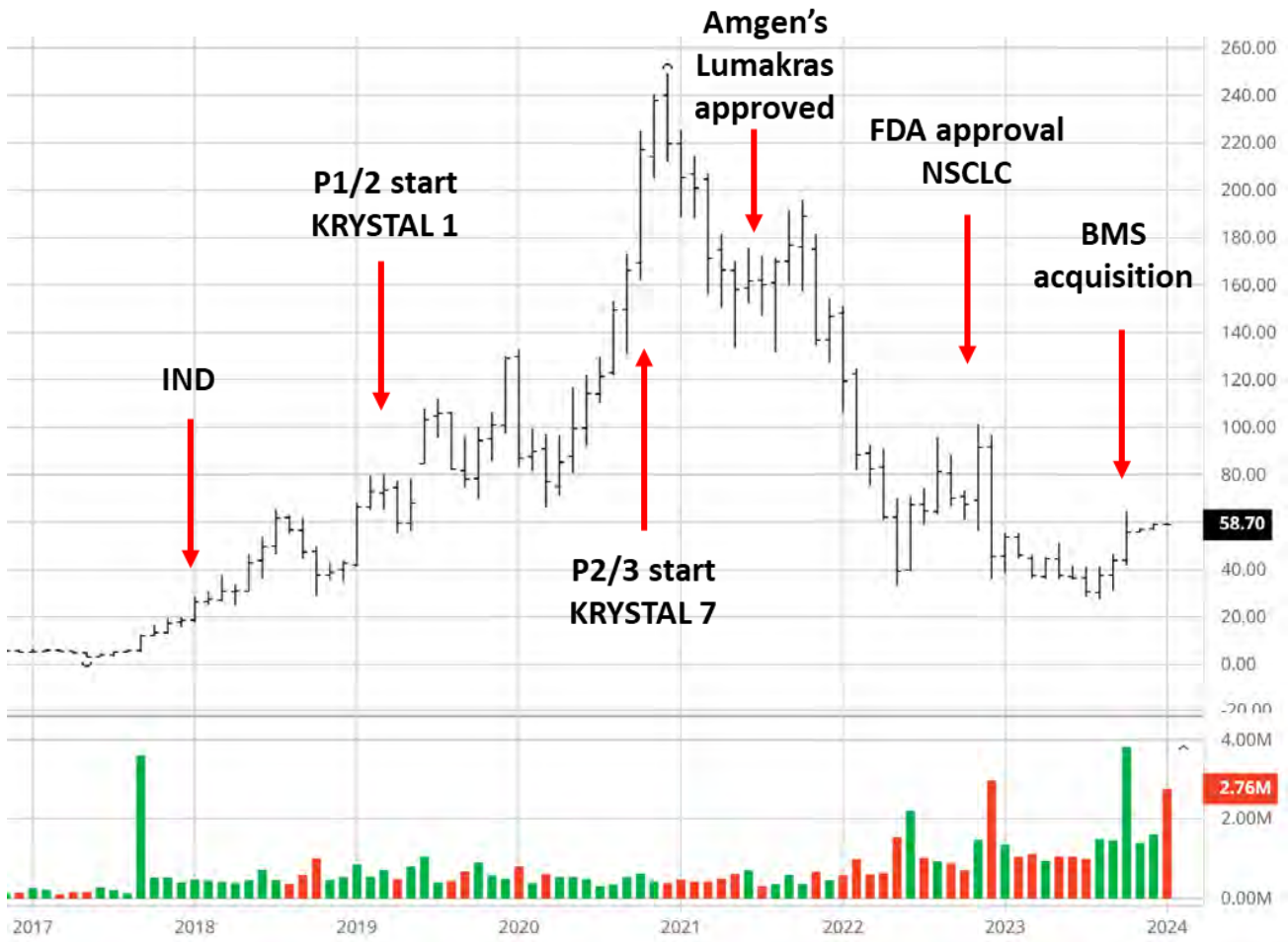
Mirati developed Krazati® (adagrasib, MRTX-849) targeting 'undruggable' KRAS G12C mutation

- G12C accounts for 15% of all KRAS mutations
- Most common KRAS muatation in lung cancer

As perceived market leader, Mirati's market cap peaked at US$12B

- Amgen's Lumakras® (sotorasib) approved first, in May 2021

Acquired by Bristol Myers Squibb for US$4.8B + US$1B CVR in October 2023



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RAS, RAt Sarcoma virus; KRAS, Kirsten rat sarcoma viral oncogene homolog; EGFRm, epidermal growth factor receptor; mAB, monoclonal antibody

Recent drug approvals in NSCLC

Early-stage approvals from small patient numbers underscores clinical need in NSCLC

Developer	Bayer	Boehringer Ingelheim	Dizal 迪哲
Drug (generic) name	Hyrnuo® (sevabertinib)	Hernexeos® (zongertinib)	Zegfrovy® (sunvozertinib)
MoA	HER2 TKI	HER2 TKI	EGFR TKI
FDA approval	19/11/2025	11/08/2025	02/07/2025
Data for approval	Phase 1/2	Phase 1	Phase 1/2
# patients in trial	70 patients	71 patients*	315 patients
Response rate	ORR 71%	ORR 75%	ORR 46%
Duration of response	DOR 9.2m	58% of pts >6m	DOR 11.1m

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* Data shown from one of two cohorts in study | NSCLC, non-small cell lung cancer; MoA, mechanism of action; FDA, Food & Drug Administration; HER2, human epidermal growth factor receptor 2; TKI, tyrosine kinase inhibitor; ORR, overall response rate; DOR, duration of response

MYC inhibitors in the clinic

RC220 competitors are early stage and have limited clinical activity

Drug	Developer	Mechanism	Clinical status & activity
Direct (protein/gene)			
RC220	Racura Oncology	G-quadruplex stabiliser	Phase 3 AML, Phase 1 NSCLC, Phase 1 solid tumours; 40% CR/PR in AML
OMO-103	Peptomyc S.L.	MYC-MAX inhibitor (Omomyc miniprotein)	Phase 2 (osteosarcoma); 8/22 SD, no CR or PR
IDP-121	IDP-Pharma	Stapled peptide direct inhibitor of MYC	Phase 1/2 in MM; No clinical efficacy data released
PC-002	Cothera Bio	De-ubiquitinase inhibitor	Phase 2 lymphoma; 1/13 CR, no PR
LMP744	Gibson Oncology	G-quadruplex stabiliser	Phase 1/2 GBM; 2/40 PR, no CR
CX-5461	Senhwa Biosciences	G-quadruplex stabiliser	Phase 1b BRCA1/2 or HR deficient solid tumours; 4/40 PR, 11/40 SD
WBC100	Hangzhou Weben	MYC-specific PROTAC degrader	Phase 1 in MYC+ solid tumours; No clinical efficacy release
Other pathways			
WP1066	Moleculin Biotech	p-STAT3 inhibitor (impacts MYC transcription)	Phase 2 GBM with radiotherapy
GP2250	Persevere Tx	Inhibits MYC and NFκB	Phase 1 pancreatic
PMR-116	Pimera Tx/ ANU	Inhibits pathway downstream of MYC	Phase 1 in MYC+ solid tumours

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Source: www.clinicaltrials.gov, company websites and announcements | PROTAC, Proteolysis Targeting Chimeras | p-STAT3, phosphorylated Signal Transducer and Activator of Transcription 3; NFκB, Nuclear Factor kappa-light-chain-enhancer of activated B cells; NSCLC, non small cell lung cancer; SD, stable disease; CR, complete remission; PR, partial response; MM, multiple myeloma; GBM, glioblastoma multiforme

Corporate snapshot

Racura Oncology is an ASX-listed, late-stage clinical biopharmaceutical company

Key metrics ($A)

ASX code	**RAC**
Share price	**$2.83[1]**
Market capitalisation	**$514.5m[1]**
Cash at bank	**$20.9m[2]**
Debt	**Nil**
Enterprise value	**$493.6m[1]**
Shares on issue	**181,806,949[1]**
Options on issue	**25,051,964[1]**

1. *As at 20 March 2026*
2. *As at 31 December 2025*

Racura Oncology 12-month trading history[1]

Current funding

- On 22 November 2023, Racura Oncology issued a 1 for 20 bonus and piggyback option series to existing shareholders
  - The conversion of bonus options ($0.75) raised $5m and the 19.9m piggyback options ($1.25) could raise a total of $25m before expiry 29 May 2026
- **Racura Oncology is fully funded to undertake the CPACS and HARNESS-1 trials through 2027 from current funds**

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General Q&A

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Glossary of terms

^{1}H-NMR, proton nuclear magnetic resonance
1st L, first line
(E,E)-bis, (E,E)-bisantrene
a-vO_2 diff, arteriovenous oxygen difference
ALL, acute lymphoblastic leukaemia
AML, acute myeloid leukaemia
BC, breast cancer
ccRCC, clear cell renal cell carcinoma
cGMP, current good manufacturing practice
CMC, chemistry, manufacturing, and controls
cMRI, cardiac magnetic resonance imaging
CN, China
CP, cardioprotection
CPACS, cardioprotection and anticancer synergy
CR, complete remission
CRF, cardiorespiratory fitness
ctDNA, circulating tumour DNA
CTRCD, cancer therapy-related cardiac dysfunction
CV, cardiovascular
DBS, double-strand breaks
DLBCL, diffuse large B-cell lymphoma
DOR, duration of response
DOX, doxorubicin
EC50, effective concentration maximal effect
ECOG, Eastern Cooperative Oncology Group
EGFR, epidermal growth factor receptor
EGFRm, epidermal growth factor receptor mutant
EOP2, end of phase 2
EU, Europe
EU, European Union
FDA, Food & Drug Administration
FGFR, fibroblast growth factor receptor
G4, G-quadruplex
GBM, glioblastoma multiforme
GI, gastrointestinal
GLS, global longitudinal strain
HER2-, human epidermal growth factor receptor 2 negative
HER2, human epidermal growth factor receptor 2
HER4, human epidermal growth factor receptor 4
HL, Hodgkin's lymphoma
HR, heart rate
HR+, hormone receptor positive
IDC, invasive ductal carcinoma
IGF-1R, insulin-like growth factor 1 receptor
IHC, immunohistochemistry
IND, Investigational New Drug
IP, intellectual property

Glossary of terms

KRAS, Kirsten rat sarcoma viral oncogene homolog
LVEDV, left ventricular end diastolic volume
LVEF, left ventricular ejection fraction
mAB, monoclonal antibody
MAPK, mitogen-activated protein kinase
MAX, Myc-associated factor X
Mcap, market cap
MCL, mantle cell lymphoma
MET, mesenchymal–epithelial transition factor
MM, multiple myeloma
mo, months
MOA, mechanism of action
mPFS, median progression free survival
MTCD, maximum tolerated combined dose
MTD, maximum tolerated dose
NDA, New Drug Application
NFκB, nuclear factor kappa-light-chain-enhancer of activated B cells
NSCLC, non-small cell lung cancer
ORR, objective response rate
Osi, osimertinib
p.a., per annum
p-STAT3, phosphorylated signal transducer and activator of transcription 3
PD, progressive disease
PDGF, platelet-derived growth factor
PD, pharmacodynamic
PDX, patient-derived xenograft
PI3K/AKT, phosphoinositide 3-kinase / protein kinase B
PK, pharmacokinetics
r/r, relapsed and refractory
RAS, Rat Sarcoma
SaO_2, arterial oxygen saturation
SCLC, small cell lung cancer
SD, stable disease
STS, soft tissue sarcoma
SV, stroke volume
SVR, systemic vascular resistance
TKI, tyrosine kinase inhibitor
TNBC, triple negative breast cancer
TOP1, topoisomerase I
TOP2, topoisomerase II
US, United States
VEGF, vascular endothelial growth factor
VO_2, volume of oxygen
WHO, World Health Organisation