

UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
Washington, D.C. 20549

FORM 8-K

CURRENT REPORT
Pursuant to Section 13 OR 15(d) of The Securities Exchange Act of 1934

Date of Report (Date of earliest event reported): May 18, 2026

Kairos Pharma, Ltd.
(Exact name of registrant as specified in its charter)

Delaware
(State or other jurisdiction
of incorporation)

001-42275
(Commission
File Number)

46-2993314
(IRS Employer
Identification No.)

2355 Westwood Blvd., #139
Los Angeles CA 90064
(Address of principal executive offices) (Zip Code)

(310) 948-2356
Registrant's telephone number, including area code

N/A
(Former name or former address, if changed since last report)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions:

- ☐ Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)
- ☐ Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)
- ☐ Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))
- ☐ Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Securities registered pursuant to Section 12(b) of the Act:

Title of each class	Trading Symbol (s)	Name of each exchange on which registered
Common Stock, par value \$0.001, per share	KAPA	NYSE American

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (§230.405 of this chapter) or Rule 12b-2 of the Securities Exchange Act of 1934 (§240.12b-2 of this chapter).

Emerging growth company ☒

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act. ☐

Item 8.01. Other Events.

On May 18, 2026, Kairos Pharma, Ltd., a Delaware corporation (the “Company”), issued a new investor presentation, which will be published on the Company’s website, www.kairospharma.com, and presented today at the 2026 LD Micro Invitational XVI (the “LD Micro Invitational”), set to take place in Los Angeles, CA May 18-19, 2026. As part of its participation in the LD Micro Invitational, the Company’s Chief Executive Officer, John Yu, will present the investor presentation to provide an update on the Company’s clinical program and partnering activities.

A copy of the investor presentation is being furnished as Exhibit 99.1 to this Form 8-K. The information included in this Item 8.01, including Exhibit 99.1, is being furnished and shall not be deemed “filed” for purposes of Section 18 of the Securities Exchange Act of 1934, as amended (the “Exchange Act”), or otherwise subject to liabilities under that section, and shall not be deemed to be incorporated by reference into the filings of the Company under the Securities Act or the Exchange Act, regardless of any general incorporation language in such filings.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits.

Exhibit No.	Description of Document
99.1	Investor Presentation
104	Cover Page Interactive Data File - the cover page XBRL tags are embedded within the Inline XBRL Document

SIGNATURE

Pursuant to the requirements of the Securities and Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

Date: May 18, 2026

KAIROS PHARMA, LTD.

By: /s/ John S. Yu
John S. Yu
Chief Executive Officer



**KAIROS
PHARMA**

INVESTOR PRESENTATION

NYSE American: KAPA

kairospharma.com



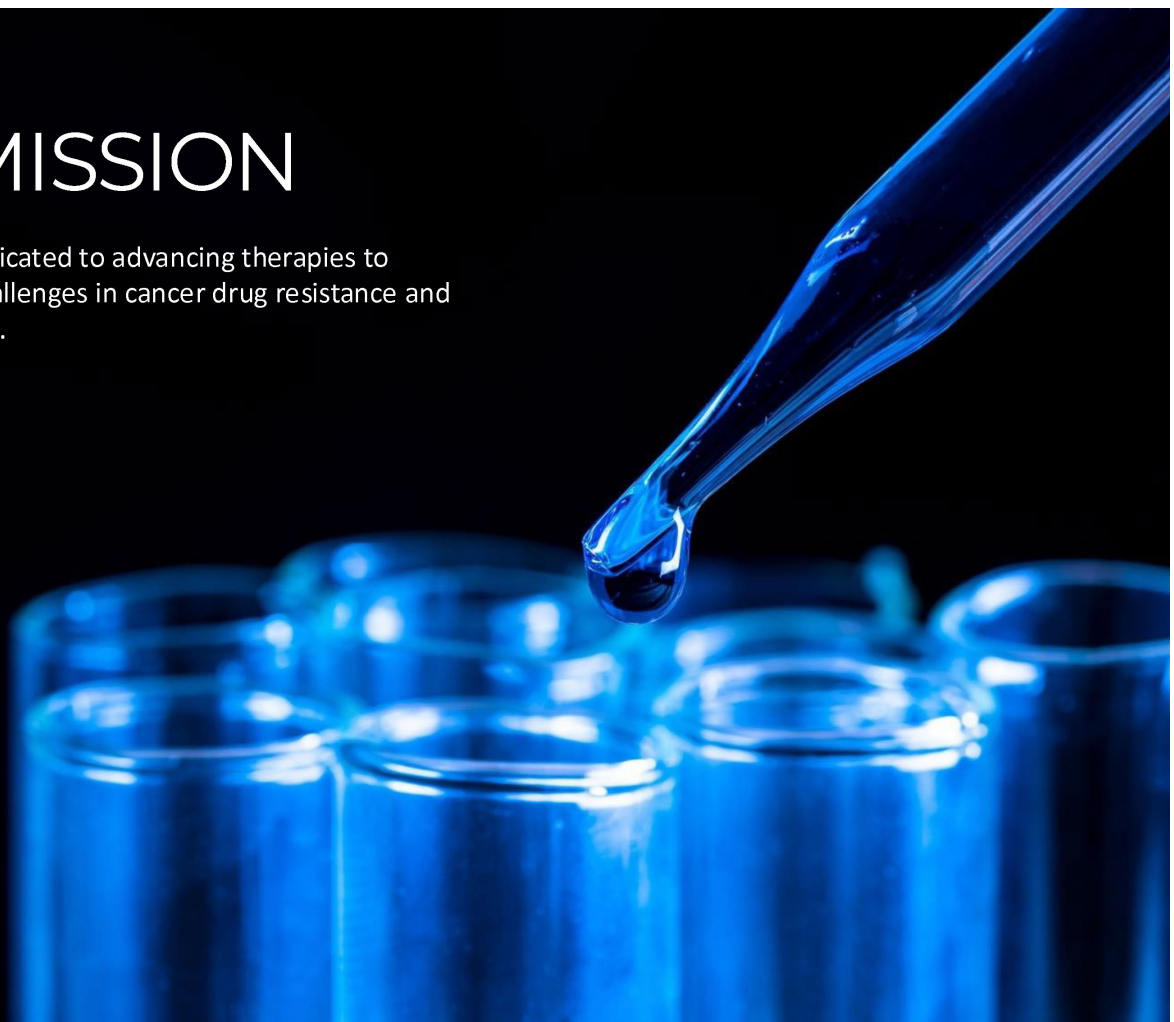


FORWARD LOOKING STATEMENTS

This presentation may contain “forward-looking statements” regarding the current expectations and projections about future events of Kairos Pharma or the “Company”). “Forward-looking statements” (statements as to matters other than historical facts) as defined in the Private Securities Litigation be identified by terminology such as: “will,” “potential,” “could,” “can,” “believe,” “intends,” “continue,” “plans,” “expects,” “projects,” “estimates,” “or similar language. These statements are based upon current beliefs and expectations and are subject to many risks and uncertainties which are predict. No forward-looking statements can be guaranteed, and actual results may differ materially from such statements. The information in this only as of the date of this presentation and Kairos Pharma takes no obligation to update any forward-looking statements contained in this pres information of future events and/or results. These forward-looking statements are subject to known and unknown risks, uncertainties, assumption could cause the Company’s actual results, performance or achievements to be materially different than any future results, performance or achi implied by the forward-looking statements. Actual results may differ materially from those indicated by these forward-looking statements as a res factors, including the Company’s ability to discover and develop its novel product candidates and successfully demonstrate the efficacy and safety o the pre-clinical and clinical results for its product candidates, which may not support further development of product candidates; actions of the C regarding continued product development and product commercialization; actions of regulatory authorities, which may affect the initiation, timing trials or the ability of the Company to obtain marketing authorization for its product candidates; the Company’s ability to obtain, maintain and property; the Company’s ability to enforce its patents against infringers and defend its patent portfolio against challenges from third parties; compe technology similar to the Company’s and others developing products for similar uses; the Company’s ability to manage operating expenses; the Cor additional funding to support its business activities and establish and maintain its existing and future collaborations and new business initiatives; the on collaborators and other third parties for development, manufacture, marketing, sales and distribution of products; the outcome of litig expenditures. Any forward-looking statements represent the Company’s views only as of today and should not be relied upon as representing its vie date. The Company explicitly disclaims any obligation to update any forward-looking statements.

OUR MISSION

Kairos Pharma is dedicated to advancing therapies to overcome critical challenges in cancer drug resistance and immune suppression.





The Problem:

Drug Resistance develops for even blockbuster drugs, reducing their effectiveness over time

The Solution:

CD105 is elevated with resistance. Kairos' lead candidate ENV 105 reverses resistance for hormone therapy and EGFR targeted therapy for prostate and lung cancer.

INNOVATIVE CANCER THERAPEUTICS

New technologies reverse drug resistance and immunotherapy in cancer.



KAIROS INVESTMENT HIGHLIGHTS

Three clinical programs already in progress

ENV 105

- *Phase 2 trial treating castrate resistant prostate cancer*
- *Phase 1 trial treating EGFR-driven Lung Cancer*

KROS 201

- *immunotherapy cleared IND for glioblastoma*

New clinical programs to be initiated

KROS 741 (cMET antagonist) initiating

- *Phase 1 trial treating biomarker driven lung and esophageal-gastric cancers*

INNOVATIVE CANCER THERAPEUTICS

New technologies reverse key mechanisms of drug resistance and immune suppression in cancer.



OrbiMed
Healthcare Fund Management

Experienced Team and Cedars Sinai to drive enrollment and grant funding
has created non-dilutive value grounded by science



PORTFOLIO OF INNOVATIVE DRUGS



Pre-clinical assets

KROS-301

Small molecule targeting cancer growth and immune evasion

KROS-102

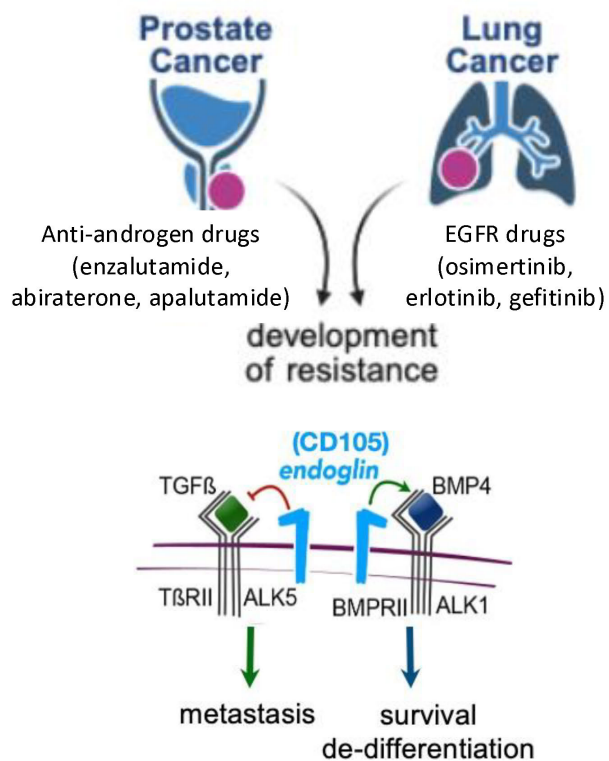
Small molecule decreasing T cell # and function

ENV-205

Antibody targeting secreted mtDNA reverse muscle wasting

KROS-401

Peptide that makes pro-tumor macrophages into anti-tumor macrophages

**THE PROBLEM: Resistance**

As cancers become resistant to standard of care, their cancer cells express CD105 on the surface, that mediates tumor cell survival and the resistance.

CD105 is elevated in resistant cancer cells that recur or are incompletely treated.

THE SOLUTION: Block CD105

ENV105 blocks CD105 to restore sensitivity to the entire class of hormone therapies for prostate cancer patients (eg. enzalutamide, abiraterone and apalutamide) and EGFR-targeted therapies for cancer patients (e.g. Osimertinib)

Broadly applicable to reverse resistance in cancer:

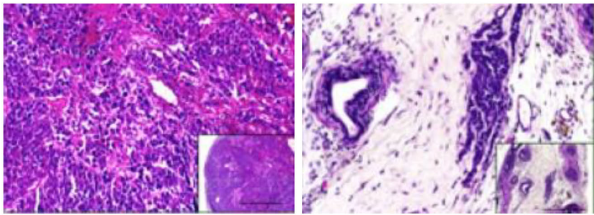
Although prostate and lung cancer are the first indications for ENV105, this drug has been shown to be effective in models of colon, breast, and head & neck cancers in the resistance developed after radiation and chemotherapy.

ENV105 restores sensitivity to hormone therapy

Targets CD105-mediated PROSTATE CANCER castrate resistance

ENV105 kills cancer cells resistant to AR Tx (H&E stain in PDX model)

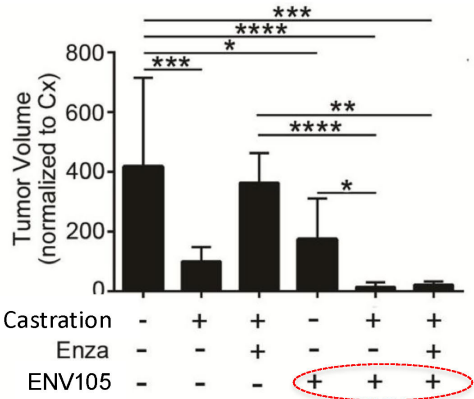
More differentiated smaller tumors



Enzalutamide

Enzalutamide+ ENV105

ENV105 + AR Tx reduces tumor volume more than AR Tx alone



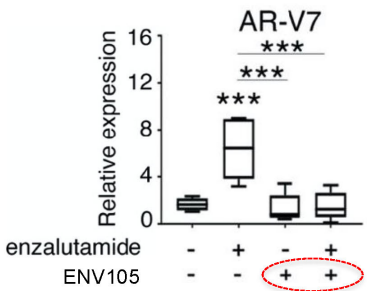
ENV105 inhibits the creation of the AR splice variant which has resistance to AR Tx

AR splice variant

AR-full length

AR-V7

ENV105
Car



Basis for clinical trials: NCT03418324 and NCT05534646

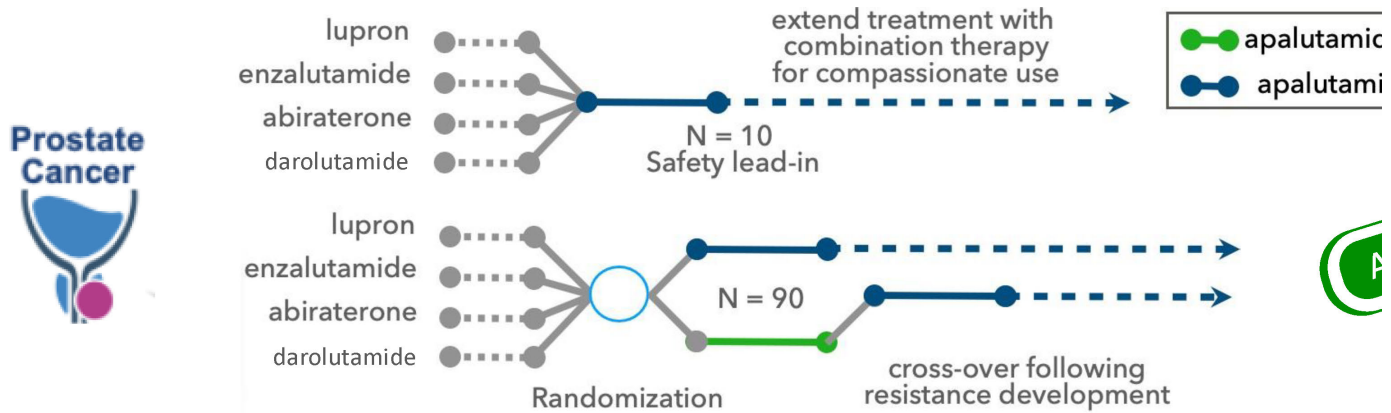
Mol Therapy (2023) 31:
Oncogene (2019) 38

ENV105

IN PROSTATE CANCER

PRESENT RANDOMIZED PHASE 2 TRIAL: NCT03418324

Apalutamide with or without ENV105



PRIMARY ENDPOINT: Progression free survival based on RECIST 1.1 and PCWG 3 criteria

SECONDARY ENDPOINT: Companion biomarker validation

Cedars
Sinai

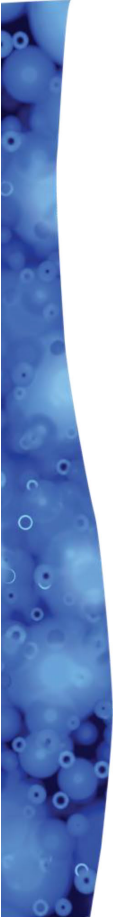


Edwin Posadas

HUNTSMAN
CANCER INSTITUTE
UNIVERSITY OF UTAH



Umang S



Phase 2 Patient Summary NCT05534646

Apalutamide and ENV105 for the Treatment of Metastatic Castration-Resistant Prostate Cancer

Patient ID	Age at enrollment	Pre-treatment PSA	Previous therapies	RECIST/PW @ week 1
001	68	7.59	Abiraterone	Stable dise
002	76	278.55	Enzalutamide, Lu-PSMA	Not assessable (unre
003	73	32.35	Enzalutamide	Stable dise
004	82	59.03	Enzalutamide, Abiraterone	Stable dise
005	60	4.49	Apalutamide	Stable dise
006	68	278.43	Abiraterone, Enzalutamide	Stable dise
007	79	5.63	Enzalutamide	Stable dise
008	71	3.37	Abiraterone	Stable dise
009	69	20.59	Abiraterone	clinical PD at 3
010	85	0.82	Enzalutamide, Darolutamide, Lu-PSMA	Stable dise

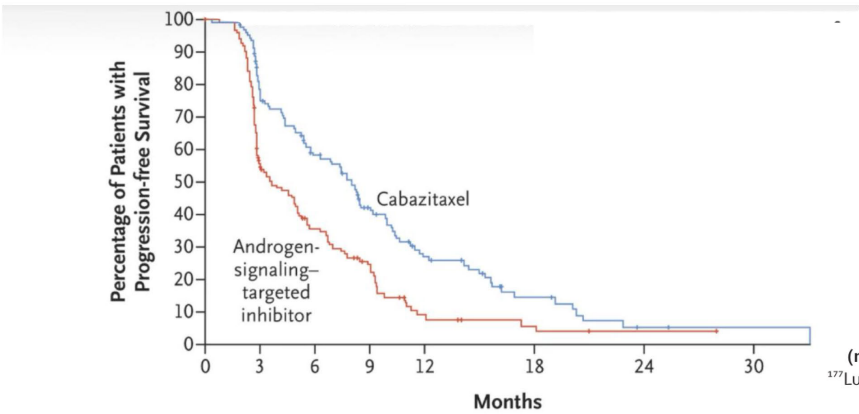
Radiographic PFS points of reference

CARD Trial

ARPI-switch median PFS = **3.7 months**

Cabazitaxel median PFS = 8 months

N = 255



No. at Risk

	129	91	64	41	23	9	2	1
Cabazitaxel	126	61	36	22	7	3	1	0
Androgen-signaling-targeted inhibitor								

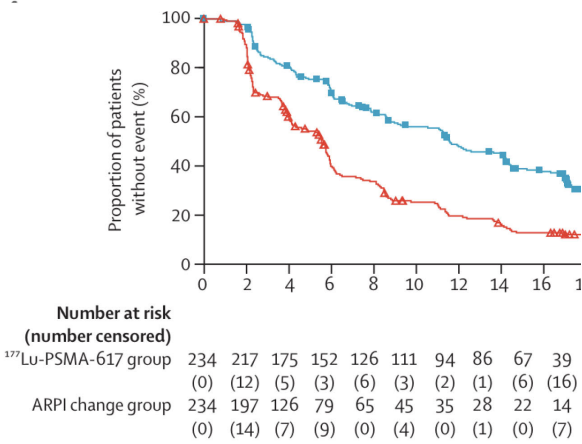
NEJM (2019) 318:2506

PSMAfore trial

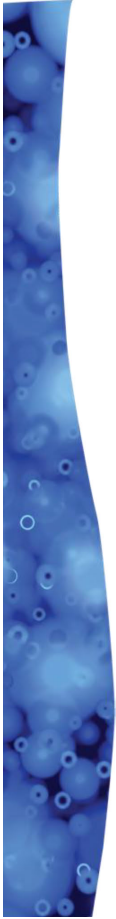
ARPI-switch median PFS = 5.6 months

¹⁷⁷Lu-PSMA median PFS = 11 months

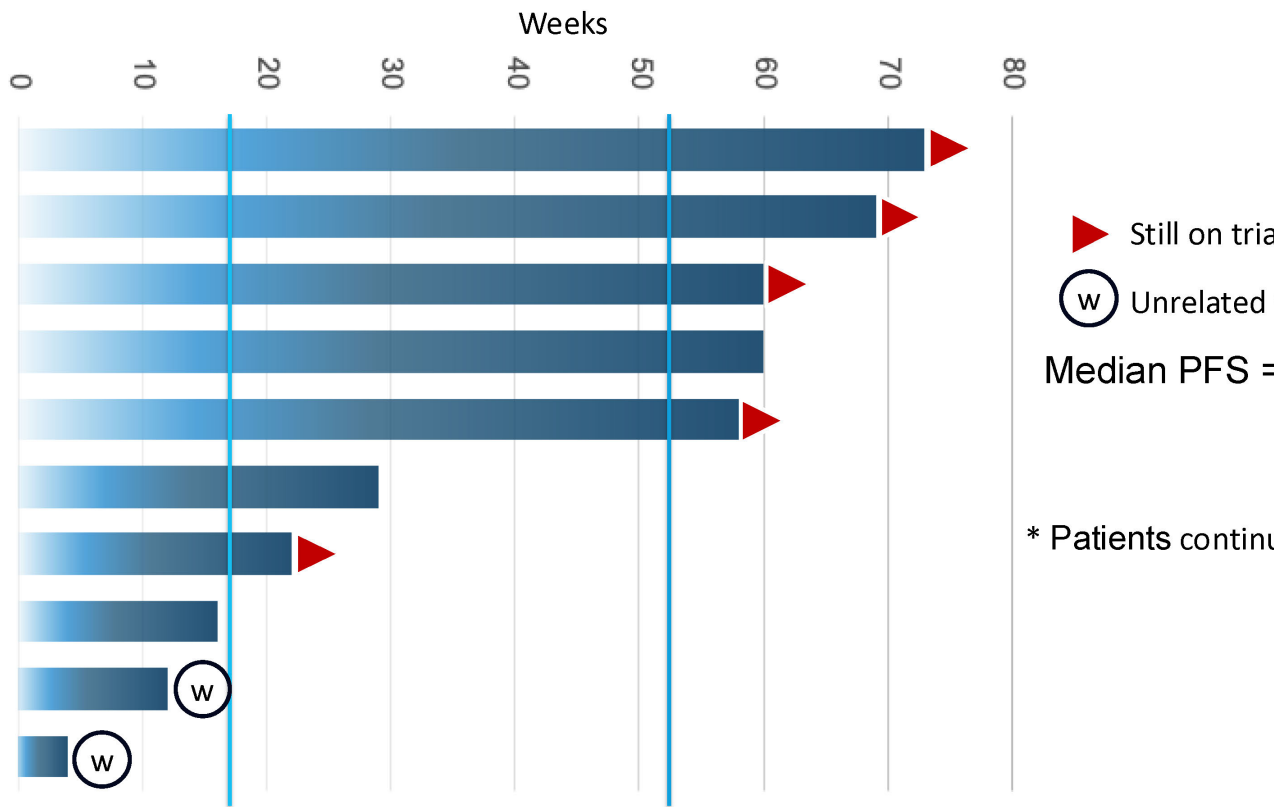
N = 468



Lancet (2024) 404:1227

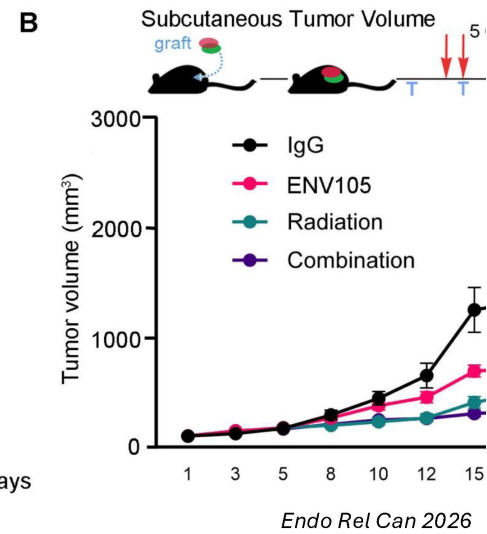


Progression Free Survival NCT05534646



Partnering ENV105 for synergy

ENV-105 and radiation combination shows increased efficacy in a metastatic mouse model



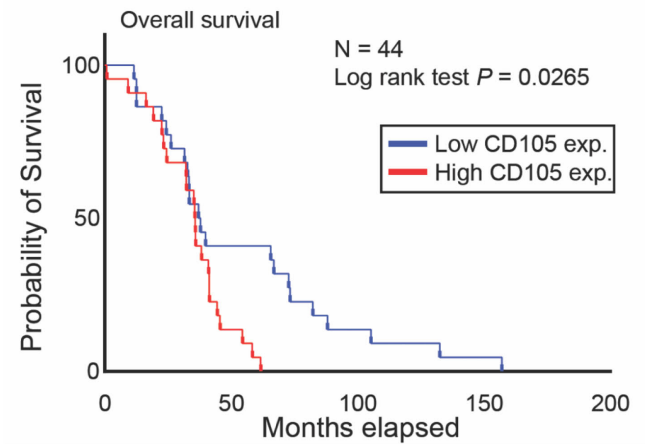
Preclinical collaboration

Combining ENV-105 and alpha-emitting isotope in animal models of bone metastatic prostate cancer

Supporting a Phase 2 Clinical Trial

ENV-105 and alpha-emitting isotope combination in metastatic prostate cancer pat

NON-SMALL CELL LUNG CANCER BECOMES RESISTANT TO OSIMERTINIB (TAGRISSO)



OSIMERTINIB RESISTANCE IS DEPENDENT ON CD105

- 45,000 EGFR driven NSCLC diagnosed last year
- Tagrisso is the standard of care for EGFR driven N. 80% of patients have only a partial response

Drug Res Updates (2025) 81:101237



ENV105 in EGFR-Driven Lung Cancer

AstraZeneca's EGFR inhibitor Tagrisso (\$7B in 2024 sales) offers only a partial response in 80% of patients

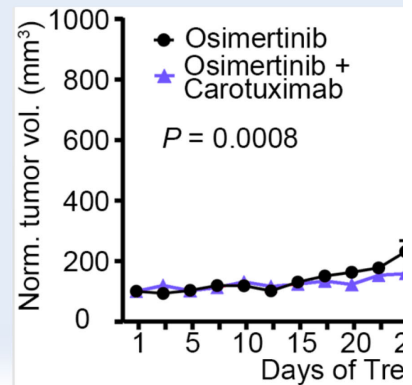
CD105 is elevated with Tagrisso treatment

Preclinical models show ENV105 can overcome resistance and shrink tumors

ENV105 could not only reverse resistance but make Tagrisso work better to enable complete responses

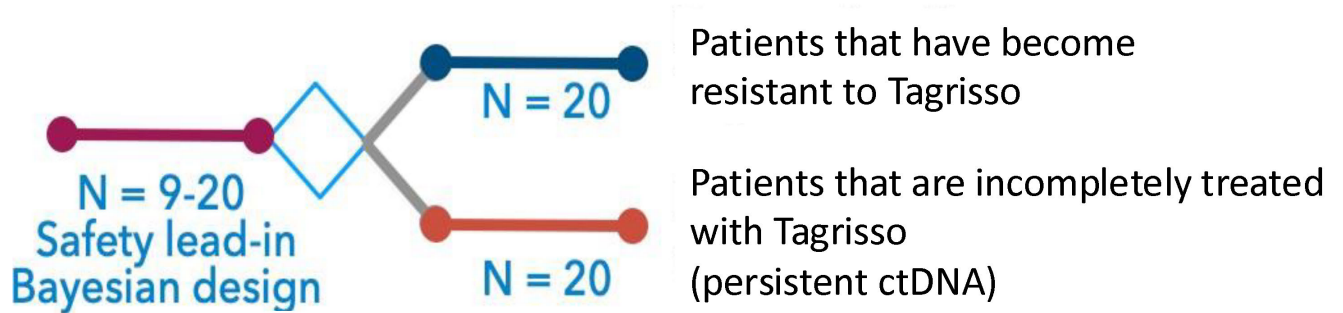
Accruing Phase I study of patients resistant to Tagrisso or partial responders with persistent circulating tumor cell DNA

Drug Res Updates (2025) 81:101237



ENV105 IN LUNG CANCER: NCT05401110

PHASE 1 TRIAL FOR EGFR-DRIVEN LUNG CANCER: Osimertinib (Tagrisso) + ENV105



PRIMARY ENDPOINT: Determine safety and effective dose of ENV105 in patients with EGFR lung cancer

SECONDARY ENDPOINT: Identify biomarkers for patients most responsive to ENV105



Clinical Trials

Cedars-Sinai
Medical Center

Mayo Clinic

Stanford

Vanderbilt

Rutgers

Cleveland Clinic

CRO consortium leveraging
the the efficiencies of each
institute to achieve 40 day
clinical trial activation
(June 1, 2026)

Centralized regulator
oversight and institut
investigator-initiated
are subsidized

Extend The Reach Of Innovative Therapies To
Patients Across Diverse Communities



Current Non-dilutive funding

\$3.2M (NCI)

ENV105 prostate cancer companion
biomarker validation



\$2.4M (Donor)

ENV105 lung cancer preclinical and
phase 1 trial



\$1.8M (DOD)

ENV105 lung cancer companion
biomarker identification



\$0.6M (DOD)

ENV205 cachexia preclinical
studies

\$8M in non-dilutive funding for the preclinical and clinical assets



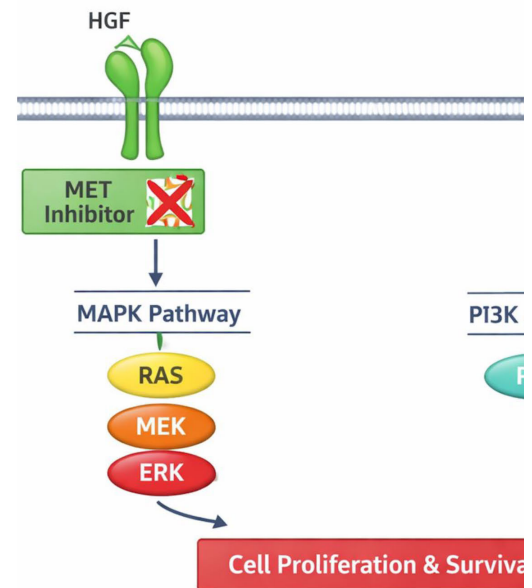
New Assets Addressing Resistance Mechanisms



- EGFR-family receptors and MET converge on PI3K–AKT, MAPK, and STAT pathways. Inhibition of one axis often leads to *compensatory* signaling through the other.
- MET amplification or HGF-driven activation is a common acquired resistance mechanism following EGFR blockade, particularly in NSCLC. MET-mediated HER3 transactivation can restore PI3K signaling despite EGFR inhibition.

KROS-741

Small molecule
cMET antagonist



*Stronger more defensible positioning both differentiation and pricing via biomarker defined patient
Better controlled resistance narrative - pre-empt vs react*



Best in class cMET inhibitor – KROS-741

	Capmatinib	Tepotinib	Cabozantinib	KROS-741
Plasma Free Fraction	4%	2%	0.2%	3%
Total plasma concentration needed to cover c-Met WT EC₅₀	40 ng/mL (3.2 nM)¹	525 ng/mL (19.2 nM)¹	26,600 ng/mL (53.3 nM)²	230 ng/mL (13.6 nM)¹
Clinical daily dose	400 mg X 2	450 mg	60 mg	100 mg X 2⁶
Clinical plasma C_{ave} at SS	~1800 ng/mL³	~1100 ng/mL⁴	~1000 ng/mL⁵	250 ng/mL⁶
Clinical plasma C_{ave} over c-Met WT EC₅₀	45x	2x	70-130x	1x
ORR	44%-67%	45%	10%	--
PFS	9.7 - 12.3 mo	8.5 mo	3.5 mo	--

(1) Dotmatics: GeoM value from Kyinno BaF3-TPR-MET CTG assay.

(2) Clin Pharmacokinet (2017) 56:477-491

(3) Cancer science (2020) 111:536-547.

(4) Clinical Cancer Research (2020) 26: 1237-1246. Result is from 500 mg daily dose.

(5) Clinical Cancer Research (2019) 25: 4924-4932.

(6) Projected for 100 mg BID human daily dose to reach TG180. DL1 or DL2 pending on GLP.

% inhibition at
100nM

≤75

76-90

≥90

Kinase	
MET	
MET_D1228A	
MET_D1228G	
MET_D1228H	
MET_D1228N	
MET_D1228V	
MET_D1228Y	
MET_F1200I	
MET_G1163R	
MET_H1094L	
MET_H1094Y	
MET_K1244R	
MET_L1195F	
MET_L1195V	
MET_M1250I	
MET_M1250T	
MET_P991S	
MET_T1173I	
MET_T992I	
MET_V1092I	
MET_Y1230A	
MET_Y1230C	
MET_Y1230D	
MET_Y1230H	
MET_Y1230S	
MET_Y1235D	
MET-KIF5B (Kex24Mex14)	
MET-TFG	



Best in class cMET inhibitor – KROS-741

A: Exon 14 Skipping

Impaired protein degradation - clinically validated in NSCLC with approved Type 1b inhibitors.

Primary oncogenic driver.

B: Amplification

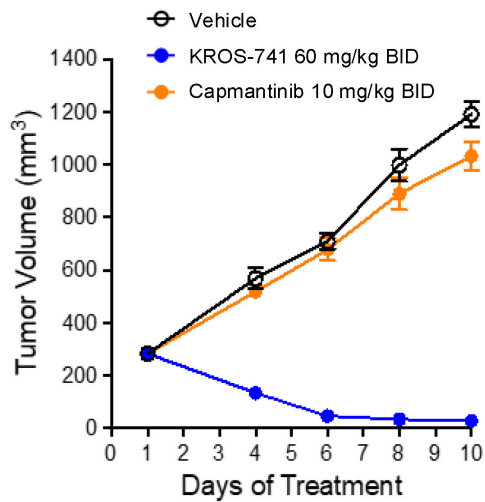
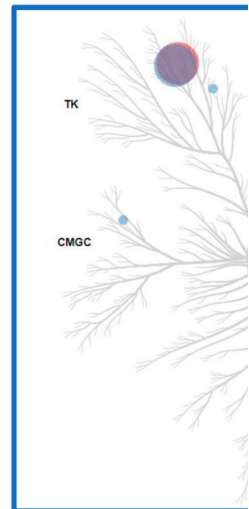
Ligand-independent dimerization. Focal amplifications are more likely to be drivers with ≥ 10 copies

Node Color

- Target
- Off-Target

Node Size

- 51-75
- 76-90
- 90-100



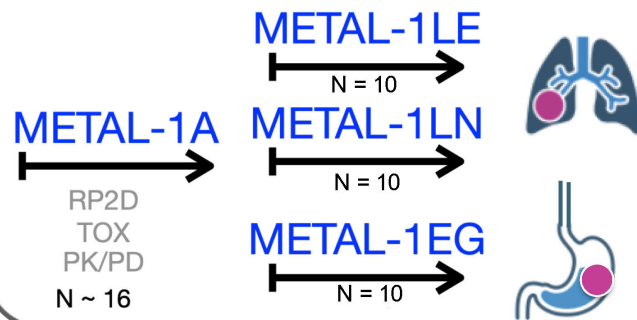
- 3-4% of NSCLC patients present with act MET Δ exon 14 alterations
- 35% of EGFR-driven lung cancer develop resistance mutations with MET inhibitors



cMET inhibitor – KROS-741 trial design

Phase 1 Objectives: Dose finding for optimal dose – RP2D and initial indication determination

KROS-741 Phase 1 trial: METAL-1



Primary endpoint:
Safety

Secondary endpoint:
Biomarker-driven efficacy

- Continuous suppression of ligand-driven
- Reduced PK fluctuation in cirrhotic patients
- Potential synergy with immunotherapy
- Broader coverage of tumor heterogeneity

Phase 2 trial: METAL-2 *potential*

Marker driven lung cancer



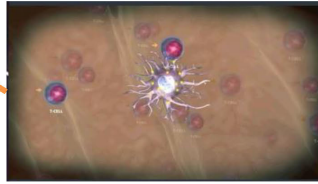
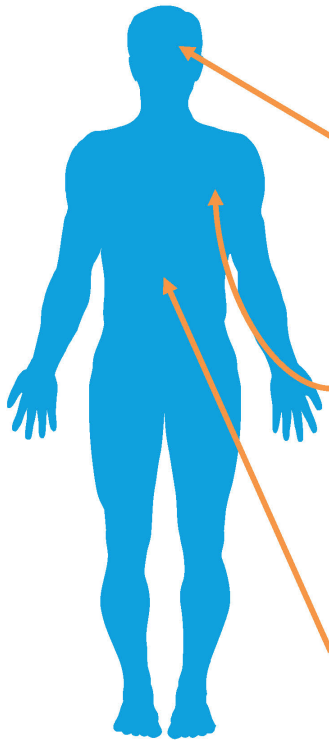
Marker driven esophageal/gastric

- KROS-741 single agent
- Orphan designation
- FastTrack/Breakthrough designation



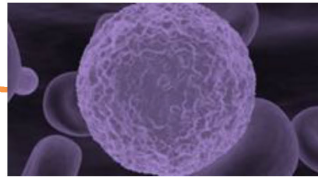
KROS DRUG CANDIDATES OVERCOMING IMMUNE SUPPRESSION

Immunotherapy is a \$118b market with only a 17% response rate.



THE PROBLEM:

Cancer is comprised of billions of growing cells. However, it seldom generates enough T cells to kill all the cancer cells.



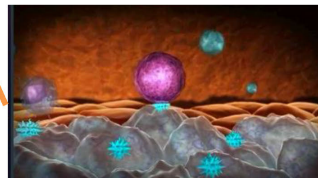
THE SOLUTION:

KROS drug candidates allow regulation of the immune response cells or by generating T cells outside of the immunosuppressed body.

KROS 101 Using our novel mechanism through GITR ligand, number of T cells and renders them more effective at killing

201 Generates T cells outside of the body and delivers to cancer

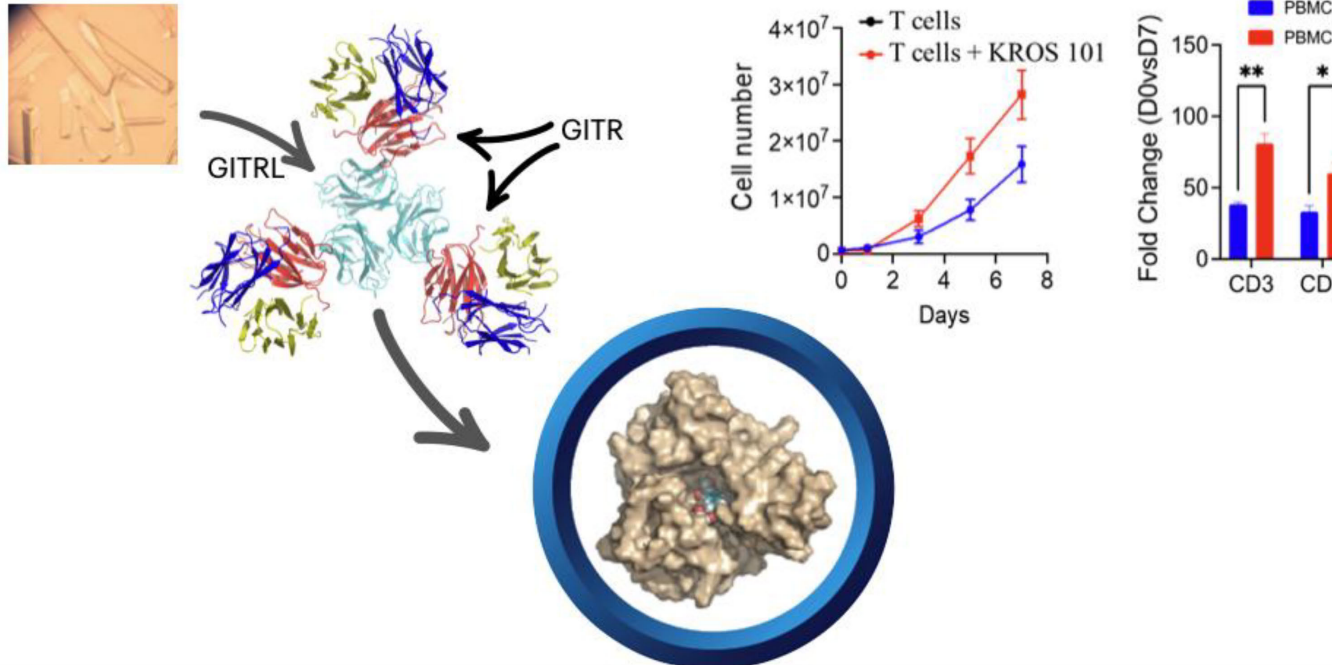
KROS 401 Converts pro-tumor macrophages into cancer-killing





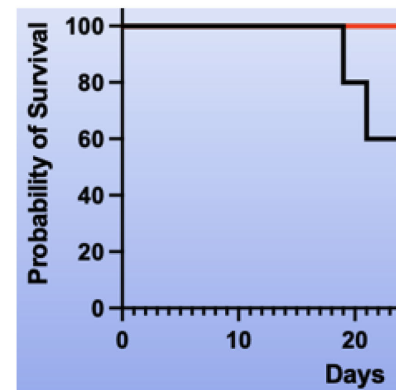
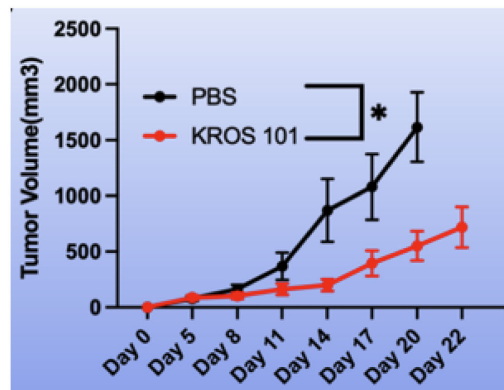
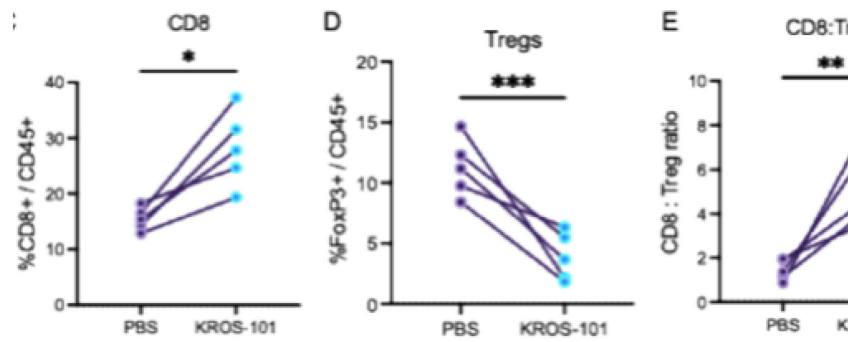
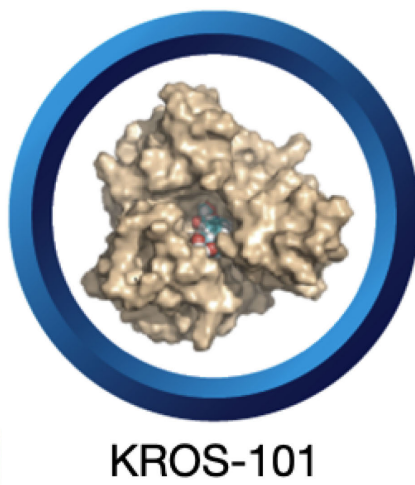
Targeting GITR to regulate T cell numbers

- **KROS101** targets the GITR ligand and enables T cells to increase in numbers and kill cancer more effectively
- By increasing T cell numbers and function **KROS101** may complement checkpoint inhibition



KROS 101 reduces tumor growth and improves survival in murine tumor models.

(A) Representative IVIS images of B16-F10-LUC2 tumor-bearing mice treated with PBS or KROS 101 at the indicated time points. (B) Mean tumor volume of B16-F10-LUC2 tumors measured by caliper. (C) Kaplan–Meier survival curves of MC38-bearing mice treated with PBS or KROS 101. (D) Mean tumor volume of MC38 tumors measured by caliper.

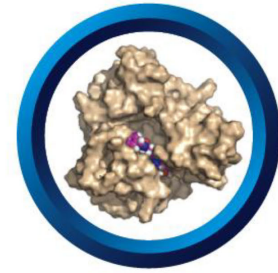




KROS102 GITR ANTAGONIST

Immunity by T effector/Treg

- **KROS102:** GITR antagonist that decreases T cell numbers and activity.
- KROS-102 has been shown to decrease T effector cells (killer T cells) and increase Treg cells (inhibitory T cells) which could reduce overactive immune response in autoimmune diseases.



Kairos identified structure of GITR ligand through X-ray crystallography
PNAS 2008

KROS-102 pictured bound to the GITR protein

SIDE EFFECTS WITH OTHER AUTOIMMUNE DRUGS

Corticosteroids and chemotherapy are the main inhibitors of an immune response, but they have many side effects such as hip necrosis, gastritis and infections.

NEW SELECTIVE MECHANISM FOR KROS102:

- KROS102 is a novel GITR inhibitor that we believe will impact the abnormal immune responses against the body without the serious side effects of current treatments.
- Treatment for autoimmune diseases such as Crohn's disease, multiple sclerosis, and rheumatoid arthritis as organ transplantation may be targets for such a molecule.



IP extends to 2040



Key IP generated from Murali, Bhowmick and Yu labs



Published patents executed internationally



Exclusive, worldwide rights to IP licensed from Cedars Sinai Medical Center

Pub #	KROS101 PCT/US2019/045478	KROS201 PCT/US2020/045570	KROS301 PCT/US2015/050906	KROS401 PCT/US2016/035318	EN PCT
Title	Compositions And Methods For Treating Cancer And Autoimmune Diseases	Method Of Generating Activated T Cells For Cancer Therapy	Compositions And Methods For Treating Fibrosis	Methods And Use Of Compounds That Bind To RELA Of NF-KB	Sens Ther Anta



LEADING TEAM



John S. Yu, MD
CEO & CHAIRMAN

Professor of Neurosurgery, Director Surgical Neuro-Oncology at Cedars-Sinai Medical Center

- BAS from Stanford, MD from Harvard Medical School and MIT
- Numerous immunotherapies and nanotechnologies from his NIH funded laboratory
- Developed 8 new investigational drugs with the FDA and has led numerous clinical trials
- Most recently CEO and Chairman of AcTcell, and Director of Enviro Therapeutics
- Immunology Fellowship at the Institute Pasteur, Paris, Neurosurgery Residency at Massachusetts General Hospital/Harvard Medical School



Doug Samuelson
CHIEF FINANCIAL OFFICER

- 25+ year finance and accounting professional
- Former CFO of .Wellness Center USA
- Former Director of Accounting of Second Sight Medical Products, Inc.
- Former Chief Financial Officer of AdvaVet, Inc. and Chief Financial Officer of Solis Tek, Inc.
- CPA in State of California



Ramchandran Murali, Ph.D
VP, RESEARCH AND DEVELOPMENT

Professor, Biomedical Sciences and Research in Immunology at Penn/Cedars-Sinai Medical Center.

- Leading expert in X-ray crystallography, biophysical, biochemical, and immunology, having made significant advances in molecular engineering and cell surface biology, developing pharmacological agents which include new paradigms in structural biology and peptidomimetics drug discovery
- Developed numerous technologies that reverse key mechanisms of immunosuppression in cancer. Previous technologies from Penn licensed by Cepion (bought by T



Neil Bhowmick, Ph.D
CHIEF SCIENTIFIC OFFICER & PRESIDENT, ENVIRO THERAP

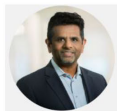
Professor of the Department of Medicine and Director of the Cancer Program at Cedars-Sinai Medical Center

- Fellowship at Vanderbilt University Medical Center Research Director, Organ Reference Laboratory (OURLab)
- Holds 6 patents for biomarker detection platforms and stromal targeted therapies (inclusive of ENV105 and ENV205)
- Consultant at Xencor Inc. (NASDAQ: XNCR) and Traccon Pharma (NASDAQ: TRACN)
- NCI/NIH funded for over 15 years and cited over 11,700 times





INDEPENDENT DIRECTORS



Rahul Singhvi, Sc.D.

Chemical engineering from MIT. MBA from the Wharton School of the University of Pennsylvania, where he graduated as a Palmer Scholar.

- Global leader in the Life Sciences industry and is cofounder of the biomanufacturing company, Resilience (National Resilience, Inc.).
- Former Operating Partner at Flagship Pioneering, where he founded and operated companies launched from Flagship's innovation foundry, Flagship labs.
- Former Chief Operating Officer of Takeda's Vaccine Business Unit where he led worldwide vaccine CMC manufacturing.
- Former President and CEO of Novavax, Inc. where he transformed the company from a specialty pharmaceutical business into a vaccine development company.
- Career began at Merck & Co in 1994, where he held several positions in R&D and manufacturing.
- Serves on the Board of Trustees of the Keck Graduate Institute, and on the Board of Directors for Codexis (Nasdaq:CDXS), and Garuda Therapeutics (private).



Hyun W. Bae, MD

BS in Biomechanics from Columbia, MD cum laude from Yale, former NIH Howard Hughes Research Fellow Bethesda, MD.

- Professor of Surgery in Orthopaedic Surgery at CSMC, Director of Education and clinical partner of the Orthopaedic Stem Cell and Tissue Engineering Laboratory
- 10+ years experience in drug development, PI for 3-4 FDA-approved randomized clinical trials with over 30 clinical studies, 60+ published scientific papers, 5 review articles and 30 patents
- Chief Medical Officer and Director of Prosidyan, Scientific Advisory Board Member of Mesoblast, Tissuegene, Spine Biopharma and Engage Surgical



Michael Keyoung, MD, Ph.D

MD and Ph.D. in Neuroscience and Neurology from Cornell at (MSKCC), UCSF Health, Biomedical Fellow at Rockefeller and I

- Senior ME, Head of N.A. for CBC Group, a healthcare-dedicated PE firm
- 20+ year career as a physician, executive and institutional investor advising Eli Lilly, Bausch & Lomb, and Samsung Electronics/Biologics on Asian expansion, global drug development and commercial/partnerships
- Former CEO of Genexine (KOSDAQ: 095700 w/ \$1 Billion MC), led commercial expansion in Europe and Asia, raised \$100M and created partnerships with Merck Pharma and Kalbe Pharma valued at over \$250M
- Former President of Catalyst Biosciences, (NASDAQ: CBIO) a clinical ophthalmology company partnered with Pfizer, MedImmune and Isu Abxis
- Previous Board Chair of AffaMed Therapeutics and Director of Graybug
- Board member of Hugel Inc. (kosdaq: 145020.KQ)





Kairos Pharma SUMMARY

- Clinical stage company with an antibody that impacts a central mechanism of **CANCER DRUG RESISTANCE**
- Extensive pipeline targets **IMMUNE SUPPRESSION**
- Enrolling in phase 1 and 2 trials with interim efficacy data in 2025, 2026
- Strategic relationship with Cedars-Sinai Medical Center (ranked #2 in the nation) provides access to medical expertise, drives clinical trial efficiency and accelerates therapeutic innovations
- Novel small molecule acquisition counter lung cancer EGFR therapy resistance

INNOVATIVE CANCER THERAPEUTICS

New technologies reverse key mechanisms of cancer drug resistance and immune suppression



Kairos Pharma CATALYSTS

- **PARTNERING ADVANCES**

Research collaboration with major pharmaceutical SECOND QUARTER 2026

- **PHASE 1 TRIAL FOR LUNG CANCER**

Safety data and interim efficacy SECOND QUARTER 2026

- **PHASE 2 TRIAL FOR PROSTATE CANCER**

Efficacy update THIRD QUARTER 2026

- **IND FOR KROS 741 CMET INHIBITOR**

Third quarter 2026

- **RESEARCH COLLABORATION FOR KROS 101 GITRL CHECKPOINT INHIBITOR**

Third quarter 2026



**KAIROS
PHARMA**

INVESTOR RELATIONS

CoreIR

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