MAXIM GROUP

Equity Research
Initiation

Biotechnology

KAPA – NYSE American	March 27, 2025
Closing Price 3/26/25	**$0.96**
Rating:	Buy
12-Month Target Price:	$4.00
52-Week Range:	$0.85 - $4.00
Market Cap (M):	$13.2
Shares O/S (M):	13.7
Float:	35.2%
Avg. Daily Volume (000):	2,350.3
Debt (M):	$0.1
Dividend:	$0.00
Dividend Yield:	0.0%
Risk Profile:	Speculative
Fiscal Year End:	December

Total Expenses ('000)

	2024E	2025E	2026E
1Q	222A	564	1,580
2Q	232A	589	1,648
3Q	383A	638	1,786
4Q	583	662	1,854
CY	966	2,452	6,868

On 9/16/24, KAPA completed an initial public offering with the issuance of 1.55M shares of common stock at an offering price of $4.00 per share, raising gross proceeds of $6.2M.

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Kairos Pharma, Ltd.

Buy

Targeting CD105 Could Extend SOC Prostate and Lung Cancer Treatments – Initiating Coverage with a Buy Rating and $4 PT

Summary

- Kairos is a clinical-stage oncology company developing lead asset, ENV-105, a novel CD105 (Endoglin) antibody designed to reverse cancer drug resistance by re-sensitizing patients to standard-of-care (SOC) therapies.
- Lead program is in metastatic castration-resistant prostate cancer (mCRPC) patients with resistance to SOC anti-androgen therapies. Interim P2 data is expected in 1H25. Partially de-risked by prior P2 proof-of-concept data.
- Additional opportunity for ENV-105 in EGFR-mutated non-small cell lung cancer (NSCLC) patients resistant to SOC drug Tagrisso. Phase 1 is ongoing, with initial data expected by YE25.
- Re-sensitizing patients to SOC drugs in oncology presents a significant opportunity for Kairos to benefit from the success of blockbuster drugs like Xtandi ($2B in 2024) and Tagrisso ($6B in 2024). Kairos has a $6.7M pro forma cash balance, and with a quarterly burn rate of ~$0.6M, the company should be funded into 2026. The upcoming P2 interim data, if positive, should represent a key de-risking event to unlock value and position the company to raise additional capital.

Details

ENV-105 — targeting cancer drug resistance

- ENV-105 is a novel neutralizing antibody targeting CD105 (Endolgin) designed to reverse cancer drug resistance.
- CD105 (Endolgin), a transmembrane protein, expressed on endothelial cells, plays a critical role in tumor angiogenesis.
- Target identified by Kairos as key driver of cancer resistance, upregulated in response to anti-androgen drugs (mCRPC) and anti-EGFR drugs (EGFR-mutant NSCLC).
- Blocking CD105 has potential to re-sensitize tumors to SOC therapies, making resistant tumors responsive again.

Ongoing Phase 2 in mCRPC — interim data 1H25

- First-line therapy in mCRPC includes anti-androgen drugs like Xtandi, although patients eventually become resistant.
- Prior P2 trial of ENV-105 + Xtandi (n=9) in resistant patients was positive, showing a 62% clinical benefit rate (CBR) vs. 0% expected.
- Ongoing larger P2 (N=90), ENV-105 + Erleada (SOC) vs. Erleada alone in resistant patients.
- Interim data is expected in 1H25.

Additional opportunity in EGFR-mutated NSCLC

- EGFR mutations occur in ~20% of NSCLC patients, 45K patients in the US.
- Tagrisso has emerged as first-line therapy (generated $6.5B in 2024).
- All EGFR-mutant NSCLC patients eventually develop resistance to anti-EGFR drugs.
- Preclinical data demonstrate re-sensitization to Tagrisso in resistant tumor models.
- Ongoing P1 of ENV-105 + Tagrisso, initial data is due by YE25.

Valuation. We model commercialization of ENV-105 in mCRPC in 2030 and EFGR-mutated NSCLC in 2031 in the US. An 80% revenue risk adjustment is factored in based on stage of development and clinical trial risk. A 30% discount rate is then applied to our free cash flow, discounted EPS, and sum-of-the-parts models, which are equally weighted to derive a 12-month price target of $4.00.

CORPORATE PROFILE

Kairos Pharma, Ltd.
2355 Westwood Blvd
Los Angeles, CA 90064
https://kairospharma.com/

Ownership summary:

Institutional Ownership:	2.9%
Insider Ownership:	64.8%*
Shares short:	1.4%

*John S. Yu, CEO and Chairman, owns >50%

Balance Sheet Summary:
(as of 9/30/24)

Cash:	$3.2M*
Debt:	$0.1M
Shareholders' equity:	$3.3M
Total assets:	$5.0M

*Subsequent to quarter-end, in January the company raised $3.5M in gross proceeds in an equity financing.

Analysts Covering the Co.: 2
# of Buys: 2
# of Holds: 0
# of Sells: 0
(*Excluding Maxim Group LLC*)

Investor Relations:
CORE IR
Louie Toma
investors@kairospharma.com

Company Background. Kairos Pharma is a clinical-stage biopharmaceutical company dedicated to developing innovative therapies designed to overcome drug resistance and immune suppression in cancer patients. The company's lead candidate, ENV-105, is an antibody that targets CD105, a protein identified as a key driver of resistance to various cancer treatments. By inhibiting CD105 on cancer cell surfaces, ENV-105 aims to reverse drug resistance and restore the effectiveness of standard therapies across multiple cancer types. Currently, ENV-105 is in a P2 trial for prostate cancer and a P1 trial for lung cancer. Kairos also has a diverse pipeline, which includes six preclinical programs. KAPA completed its initial public offering (IPO) on 9/16/24, issuing 1.5M shares of common stock at an offering price of $4 per share, raising gross proceeds of $6.2M. The IPO lockup expiration date was 3/17/25.

Senior Management:
John S. Yu, M.D., Chief Executive Officer & Chairman – Dr. Yu is the CEO and Chairman of Karios, as well as a professor and Clinical Chief of Neurosurgery and Director of the Brain Tumor Center at Cedars-Sinai Medical Center. Previously, he served as an officer at ImmunoCellular Therapeutics. Dr. Yu holds a BAS from Stanford University and an M.D. from Harvard Medical School and MIT. He completed an immunology fellowship at the Institut Pasteur in Paris and a neurosurgery residency at Massachusetts General Hospital/Harvard.

Doug Samuelson, Chief Financial Officer – Mr. Samuelson is a finance and accounting professional with over 25 years of experience. He is a certified accountant and currently serves as the CFO of Wellness Center, USA Inc. He has held multiple CFO positions, including at Second Sight Medical Products, Inc., AdvaVet, Inc., and Solis Tek, Inc. He holds an M.S. in computer science from California State University and a B.S. in accounting from the University of Utah.

Neil Bhowmick, Ph.D., Chief Scientific Officer – Dr. Bhowmick is a professor in the Department of Medicine and Director of the Cancer Biology Program at Cedars-Sinai Medical Center. He completed a fellowship at Vanderbilt University Medical Center and served as Research Director of the Oppenheimer Urologic Reference Laboratory. Dr. Bhowmick holds six patents for biomarker detection platforms and stromal-targeted therapeutics, including ENV-105 and EN-V205. Funded by the NCI/NIH for over 15 years, his work has been cited over 11,700 times.

Additional information about the company's management and Board of Directors can be found on the company website and in SEC filings.

INVESTMENT SUMMARY

Overview. Kairos Pharma is a clinical-stage biopharmaceutical company developing novel therapies targeting cancer drug resistance and immune suppression. The company's lead asset is ENV-105, an antibody that targets CD105, a protein identified as a key driver of resistance to various cancer treatments. By blocking CD105, ENV-105 has the potential to reverse drug resistance and resensitize tumors to standard-of-care (SOC) therapies across multiple cancer types. The company's lead program is in metastatic castration-resistant prostate cancer (mCRPC), with the goal of reversing resistance to front-line treatment anti-androgen drugs. A P2 is ongoing, evaluating ENV-105 + anti-androgen inhibitor Erleada (apalutamide) vs. Erleada alone. Interim data is expected in 1H25. A second program for ENV-105 in epidermal growth factor receptor (EGFR)-mutated non-small cell lung cancer (NSCLC) is ongoing in Phase 1, targeting resistance to front-line treatment with tyrosine kinase inhibitors (TKIs) such as Tagrisso (Osimertinib). Initial data is expected by YE25. While these programs represent the primary drivers for Kairos, in our view, the company also has a diverse pipeline of six preclinical programs, which could present additional opportunities for upside in the long term.

ENV-105 is a novel antibody targeting cancer drug resistance. ENV-105 (carotuximab) is a novel neutralizing antibody targeting CD105 (Endoglin). It is designed to overcome drug resistance to SOC therapies in 2L mCRPC and 2L EGFR-mutated NSCLC. Endoglin (CD105) is a transmembrane protein that, when elevated, drives bone morphogenetic protein (BMP) signaling, and plays a prominent role in tumor angiogenesis and supporting tumor survival, which led to its initial evaluation as a target. However, Kairos identified CD105 as a key driver of cancer drug resistance. It found that CD105 is upregulated in prostate and lung cancer in response to anti-androgen drugs and anti-EGFR drugs. This property is being leveraged by the ongoing development programs for ENV-105 that seek to re-sensitize tumors to SOC therapies by blocking CD105, making the resistant tumors responsive again. The company has demonstrated initial proof of concept in both a prior P2 trial in anti-androgen-resistant prostate cancer patients and preclinical work in Tagrisso-resistant cell lines in lung cancer, as well as a strong safety profile with >600 patients dosed, partially de-risking the lead clinical programs. Beyond these initial targets, preclinical models have shown efficacy in reversing resistance to radiation and chemotherapy in colon, breast, and head and neck cancers, providing opportunities for future expansion.

P2 mCRPC program ongoing, interim data 1H25. Metastatic castration-resistant prostate cancer (mCRPC) impacts 20% of prostate cancer patients, or 70K patients, in the US. First-line therapy in mCRPC includes anti-androgen drugs, namely Pfizer's Xtandi, as well as Janssen's abiraterone (Zytiga), and apalutamide (Erleada). However, almost all patients become resistant to anti-androgens eventually. ENV-105 would be positioned as a 2L combo with SOC in mCRPC after patients fail 1L anti-androgens. Xtandi alone generated ~$2B in revenue in 2024, highlighting a potentially significant market opportunity for ENV-105. The drug has demonstrated initial proof of concept in this setting with a prior P2 evaluating Xtandi or Zytiga + ENV-105 in n=9 heavily pre-treated patients with resistance to Xtandi or Zytiga. The combo demonstrated a clinical benefit rate (CBR) of 62% at 2 months, a highly encouraging result, in our view, given that the expected CBR after developing resistance should be 0%. Safety was also positive in this study with no grade 3–4 toxicities, comparing favorably to others in the space. The ongoing open-label P2 trial is evaluating (N=90) ENV-105 + Erleada vs. Erleada alone in patients who have failed anti-androgen therapy. Note that the trial is partially funded by a $3.2M NIH grant. The primary endpoint is progression-free survival (PFS). We would consider a 4-month PFS as a benchmark for success, doubling the current PFS in this patient population. Interim data is expected in 1H25. If positive, this would validate the prior P2 data in a much larger dataset and, in our view, represent a key catalyst for KAPA shares.

The P1 EGFR-mutated NSCLC program is ongoing, with initial data expected by YE25. Epidermal growth factor receptor (EGFR) mutations occur in ~20% of non-small cell lung cancer (NSCLC) patients, or 45K patients in the US. First-line therapy in these patients includes AstraZeneca's "third-generation" tyrosine kinase inhibitor (TKI) Osimertinib (Tagrisso), as well as Roche's Erlotinib (Tarceva) and AstraZeneca's Gefitinib (Iressa). Tagrisso alone generated $6.5B in revenue in 2024, which, in our view, points to a significant market opportunity for a drug like ENV-105 that could potentially re-sensitize patients to the drug. Similar to prostate cancer, patients treated with TKIs like Tagrisso eventually become resistant to therapy. ENV-105 would be positioned as a 2L combination with Tagrisso to reverse treatment resistance and restore sensitivity to treatment. Preclinical work demonstrated that Tagrisso-resistant EGFR-mutant NSCLC cells exhibit elevated CD105 levels, which ENV-105 effectively reduced. As expected, Tagrisso alone led to tumor growth, while the combo of ENV-105 significantly reduced tumor size and restored Tagrisso sensitivity. Overall, these findings, although preclinical, support the potential for ENV-105 to play a similar role in reversing Tagrisso resistance in NSCLC as in the more advanced mCRPC program with anti-androgen resistance. While the preclinical data are encouraging, Karios now needs to demonstrate this in the clinic. A P1 (N=60) is ongoing, evaluating ENV-105 + Tagrisso in resistant EGFR-mutated NSCLC patients. Initial data is expected to read out by YE25.

Bottom line. Kairos Pharma is advancing ENV-105, a CD105-targeting drug with the potential to resensitize cancer to leading cancer treatments, such as anti-androgens and EGFRi TKIs. Its lead program is in the highly active mCRPC space, where the bispecific T-cell engagers (TCEs) have demonstrated the potential to keep patients from progressing on anti-androgens. Essentially, these therapies could leapfrog Pluvicto radiotherapy in prostate cancer, and they have resulted in soaring valuations for some, as well as high-value M&A and capital inflows. Examples in this space include Janux Therapeutics (JANX – NR) and Vir Biotechnology (VIR - NR). Karios' ENV-105 interim P2 data is approaching in 1H25, and if it demonstrates a re-sensitization to anti-androgens and keeping patients from progressing for longer, it could be a significant catalyst for shares. Positive data would validate the approach of targeting CD105 and, in part, de-risk the potential for leveraging this approach in lung cancer, where Kairos's P1 in EGFR-mutated NSCLC is ongoing. An initial data update is expected by YE25. Kairos has very low opex, in part due to sources of non-dilutive capital, particularly from the NIH and DoD. As such, with a $6.7M pro forma cash balance, the company expects to have runway into 2026 and through the next key events for ENV-105. We would also watch for external events in these spaces, as positive updates could potentially provide tailwinds for KAPA shares.

Finances.

- On 1/14/25, Kairos Pharma entered into a private placement raising $3.5M in gross proceeds. The offering included 2.5M common units ($1.40 each), each consisting of one common share and a warrant for 1.5 shares. Pre-funded units ($1.399 each) contained one pre-funded warrant for a common share and 1.5 share purchase warrants. Combined with the $3.2M in cash as of 3Q24-end, we believe the company has sufficient runway into 2H26.
- On 11/14/24, Kairos Pharma reported 3Q24 results with a net loss of ($1.0M) and ended the period with $3.2M in cash on the balance sheet.
- On 9/16/24, Kairos Pharma completed its initial public offering, raising $6.2M. The company sold 1,550,000 shares at $4 per share, and the stock began trading on the NYSE under the symbol KAPA.

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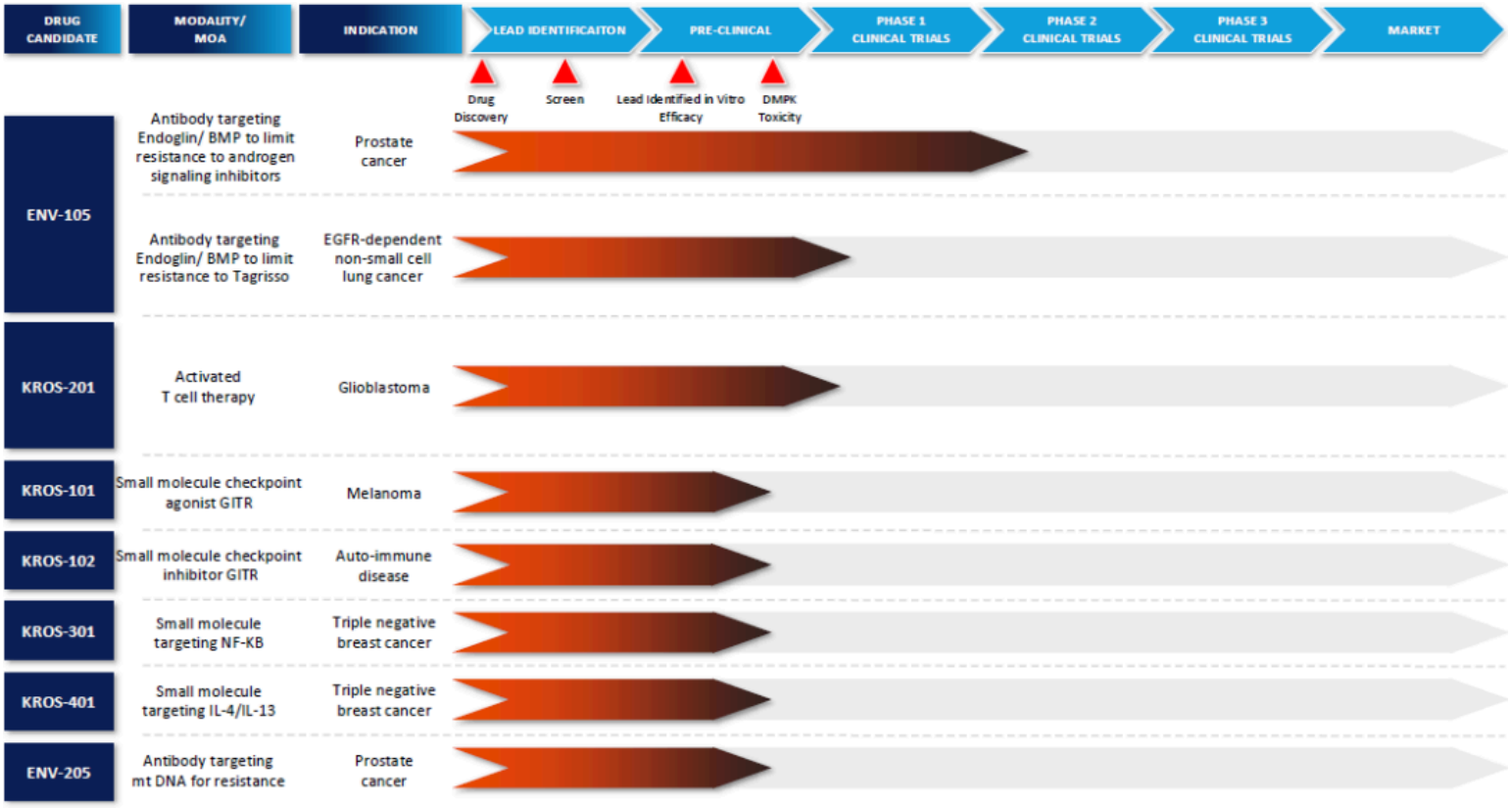
Upcoming catalysts.

Product	Indication	Event	Timeline	Impact
ENV-105	mCRPC	P2 inteirm data	1H25	+++
ENV-105	EGFR-mutant NSCLC	P1 initial data	YE25	++

Stock Significance Scale: + of moderate importance; ++ higher level; +++ very important

Source: Company reports and Maxim Forecasts

Pipeline.

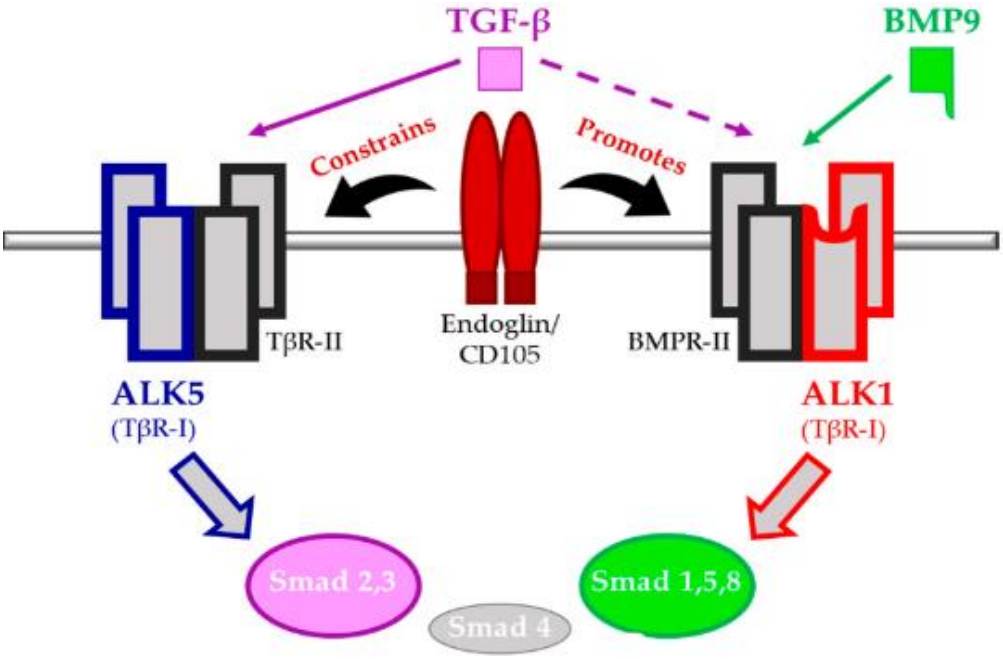


Source: Kairos corporate presentation

Endoglin (CD105)

Endoglin (CD105) is a transmembrane glycoprotein and a key marker of proliferating endothelial cells, making it a valuable target for cancer diagnosis and therapy. It plays a crucial role in angiogenesis by modulating the TGF-β signaling pathway, which influences endothelial cell proliferation, migration, and survival. Identified over two decades ago, Endoglin serves as an accessory receptor for TGF-β, a cytokine that regulates various cellular functions. Endoglin is upregulated in proliferating endothelial cells, particularly under hypoxic conditions. Human Endoglin is a 633 amino acid, 180 kDa glycoprotein that contains an extracellular domain with an RGD tripeptide, essential for its binding functions.[1] There are two isoforms of Endoglin: L-Endoglin, predominantly expressed in endothelial cells, and S-Endoglin, which has a shorter cytoplasmic tail. Both isoforms are constitutively phosphorylated, likely due to the activation of TGF-βR2. CD105 is overexpressed on tumor-associated blood vessels in various cancers, including breast, prostate, lung, and colorectal cancers, and its expression is linked to tumor progression, increased microvessel density, and poor prognosis.[2] Its expression is upregulated by hypoxia and pro-angiogenic factors like TGF-β1 and downregulated by TNF-α. Because CD105 is selectively expressed on actively growing tumor vasculature, it has been validated as a target for antiangiogenic therapies. Inhibiting CD105 can enhance the TGF-β1-mediated suppression of endothelial cell functions, disrupting angiogenesis and inhibiting tumor growth. Angiogenesis, the formation of new blood vessels, is crucial for both normal tissue function and the progression of cancer and other diseases.

Role of CD105 in endothelial cell signaling. Endoglin (CD105) drives angiogenic signaling by modulating TGF-β pathways. In active endothelial cells, it phosphorylates ALK1, activating Smad-1/5, which then complexes with Smad-4 to promote pro-angiogenic gene expression. ALK5 regulates TGF-β signaling in quiescent endothelial cells, maintaining vascular homeostasis.[3]



Source: Ellis et al.

Preclinical studies using monoclonal antibodies (mAbs) against CD105 have demonstrated significant reductions in tumor vasculature, growth, and metastasis, both alone and in combination with chemotherapeutic agents. Several other studies have demonstrated the critical role of Endoglin (CD105) in tumor malignancy and resistance to therapy, further establishing it as a key target in cancer treatment. In renal cell carcinoma (RCC) xenografts, a subpopulation of CD105-positive cells exhibits self-renewal properties, contributing to in vivo tumorigenicity and

[1] Ellis et al. Clin Cancer Res. 2008. 80(2):303-317
[2] Fonsatti et al. J Cell Physiol. 2011.10(6):182-184
[3] Nicotra et al. 2015. Oncogene. 29;22(42)

chemoresistance to gemcitabine.[4] Similarly, in ovarian cancer, high CD105 expression correlates with drug resistance, poor differentiation, advanced disease stage, and increased recurrence rates. Additionally, CD105 is upregulated in prostate cancer cells following radiation exposure, where it contributes to resistance. The latter point is particularly important for Kairos, as its most advanced program is in prostate cancer, with the objective of re-sensitizing tumors to cancer drugs due to CD105, which drives resistance by blocking CD105.

CD105 expression across solid tumors.

Tumor type	Lymph node metastasis	Distant metastasis	Higher tumor grade	Decreased survival	Comments	References
Gastric cancer		+		+	Ascites	Ding et al. (38) Nikiteas et al. (39)
Esophageal cancer	+			+		Saad et al. (40)
Colorectal cancer	+	+	+	+		Duff et al. (8) Fonsatti and Maio (4) Saad et al. (41) Akagi et al. (42)
Breast cancer		+	+	+	Response to neoadjuvant chemotherapy	Duff et al. (8) Fonsatti and Maio (4) Gomez-Esquer et al. (43) Li et al. (44) Beresford et al. (45)
Hepatocellular carcinoma		+		+		Yang et al. (46)
Non–small cell lung cancer				+		Fonsatti and Maio (4)
Prostate cancer	+	+	+	+	Preoperative PSA	Duff et al. (8) El-Gohary et al. (47) Fonsatti and Maio (4)
Head and neck cancers	+	+		+	VEGF levels	Chien et al. (48) Chuang et al. (49) Marioni et al. (50)
Primary brain malignancies			+	+	VEGF levels	Behrem et al. (51) Yao et al. (52)
Endometrial carcinoma				+	VEGF levels	Erdem et al. (53) Fonsatti and Maio (4)
Ovarian cancer				+	Suboptimal cytoreduction	Taskiran et al. (54)

Abbreviations: PSA, prostate-specific antigen; VEGF, vascular endothelial growth factor.

Source: Lim et al.

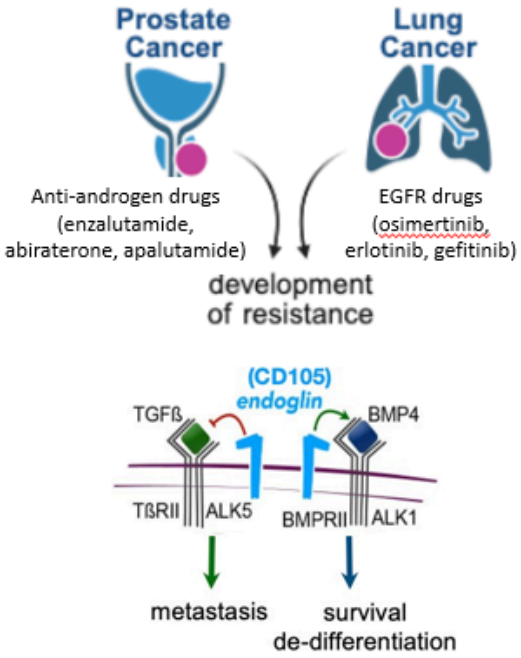
ENV-105 — CD105 targeting antibody for cancer drug resistance

ENV-105. ENV-105 (carotuximab) is a CD105 (Endoglin) targeting antibody designed to help overcome resistance in 2L metastatic castration-resistant prostate cancer (mCRPC) patients and EGFR-mutated NSCLC by restoring tumor sensitivity to standard-of-care (SOC) therapies. CD105 is upregulated in response to androgen-targeted drugs, EGFR inhibitors, chemo, and radiation, driving treatment resistance. ENV-105 synergizes with SOC therapies, disrupting resistance mechanisms but has no standalone activity. A P2 prostate cancer trial is ongoing, evaluating ENV-105 + apalutamide to address resistance seen with Xtandi, Zytiga, and Erleada, while a P1 lung cancer trial is evaluating ENV-105 + Osimertinib to reverse EGFR antagonist resistance. Data from 600+ patients, including prior NCI trials, confirm that CD105 is low in resting endothelial cells but upregulated in the tumor microenvironment (TME), correlating with angiogenesis, metastasis, and poor prognosis. ENV-105 blocks CD105, impairing tumor vascularization and breaking resistance. Prior P2 data showed a 62% response rate in prostate cancer patients with limited options. Preclinical and clinical models further demonstrate its ability to restore sensitivity to androgen inhibitors, EGFR antagonists, and radiation. CD105 expression extends beyond cancer cells to cancer-associated fibroblasts (CAFs), making ENV-105 a dual-action therapy that targets both tumor cells and their supportive stroma.

Mechanism of action (MOA). ENV-105 is a neutralizing antibody targeting CD105 (endoglin), a receptor upregulated in prostate and lung cancers as resistance emerges to androgen-targeted and EGFR inhibitor therapies. CD105 elevation drives bone morphogenetic protein (BMP) signaling and suppresses TGF-beta, supporting tumor survival. Blocking CD105 with ENV-105 has the potential to restore treatment sensitivity, making resistant tumors responsive again.

[4] Matsuno et al. 2018. Cancer Res. 88(2):167

Overcoming cancer drug resistance with ENV-105. Kairos identified CD105 as a key driver of cancer drug resistance, which is upregulated in response to treatment. ENV-105 blocks CD105, restoring sensitivity to anti-androgen therapies in prostate cancer and anti-EGFR therapies in lung cancer. Beyond these lead indications, preclinical models show efficacy in reversing resistance to radiation and chemotherapy in colon, breast, and head and neck cancers.



THE PROBLEM:

Cancers become resistant to the drugs that are used to treat them.

Kairos discovered a central resistance mechanism. As patients are treated with cancer drugs, their cancer cells start to make **CD105** on the surface which makes them resistant to the drug.

THE SOLUTION:

ENV105 blocks CD105 and reverses this resistance mechanism of prostate and lung cancer drugs (entire class of anti-androgen therapies for prostate cancer like enzalutamide, abiraterone and apalutamide or anti-EGFR therapies like osimertinib [Tagrisso from Astra Zeneca] as well as Tarceva/ Iressa).

REVERSING RESISTANCE IN CANCER:

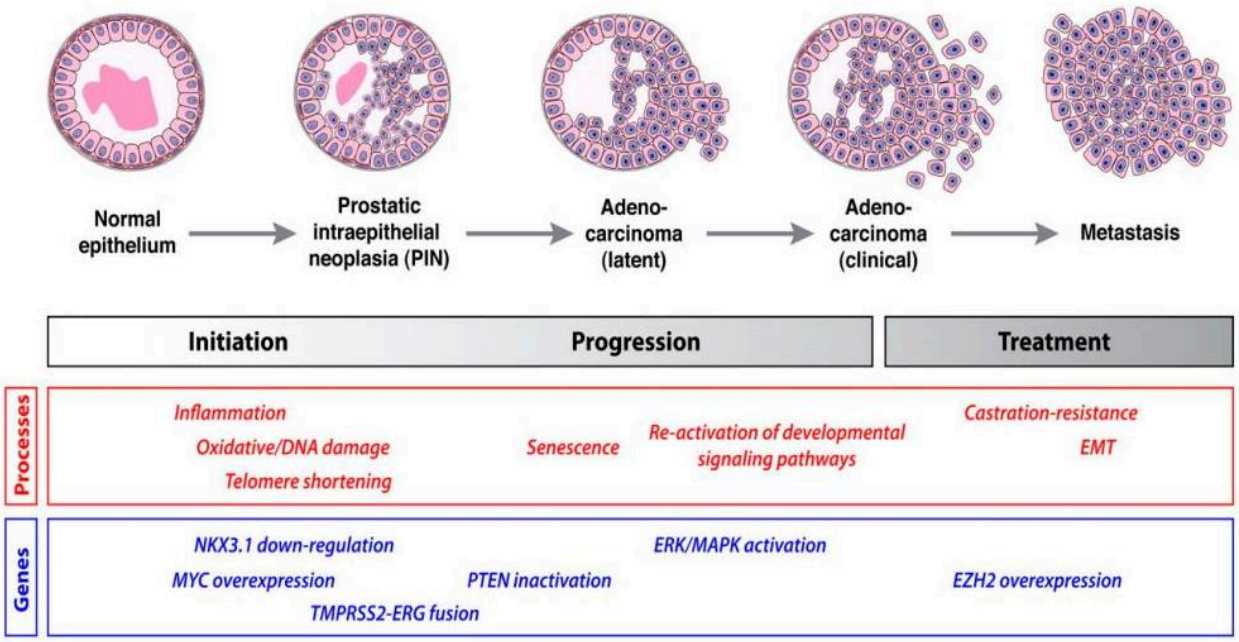
Although prostate and lung cancer are the first indications for ENV 105, this drug has been shown to be effective in models of colon, breast cancers, and head & neck cancers in the resistance developed against radiation and chemotherapy.

Source: Karios corporate presentation

Prostate cancer (PC). Prostate cancer is the most commonly diagnosed cancer in men worldwide and the fifth leading cause of cancer-related deaths in men. In 2023, 236,659 new cases were reported in the US. While most prostate cancers grow slowly and are low-grade, posing a low risk, advanced stages can lead to symptoms such as fatigue from anemia, bone pain, paralysis from spinal metastases, and renal failure from ureteral obstruction. Diagnosis typically relies on prostate-specific antigen (PSA) testing and transrectal ultrasound-guided (TRUS) biopsies, although PSA screening remains controversial.[5] New biomarkers like PCA3, along with advanced diagnostic tests such as the prostate health index, 4K score, and PCA3, have reduced unnecessary biopsies. Prostate cancer, mostly adenocarcinomas, begins in the prostate's glandular cells and often starts in the peripheral zone, growing slowly and often remaining asymptomatic for years. In advanced stages, it can spread to bones, lymph nodes, and other organs. Risk factors include age, family history, race, and genetic mutations like those in BRCA1/2. Diagnosis often involves elevated PSA levels, digital rectal exams (DRE), and biopsies. The androgen receptor (AR) signaling pathway is central to the growth of prostate cancer, leading to the use of androgen deprivation therapy (ADT) to block testosterone's effects. However, many cancers eventually become castration-resistant, continuing to grow despite low testosterone. Genetic alterations such as mutations in TP53, PTEN, and MYC, as well as overexpression of prostate-specific membrane antigen (PSMA), are linked to more aggressive forms of the disease. Metastatic castration-resistant prostate cancer (mCRPC) is a late-stage form of prostate cancer that progresses despite androgen deprivation therapy (ADT), which reduces testosterone levels.[6] While ADT blocks testosterone, prostate cancer cells in mCRPC adapt through mutations or upregulation of androgen receptors, allowing growth and spread despite low testosterone.

Development stages of prostate cancer. Prostate cancer initiation is driven by mutations in damaged DNA, leading to uncontrollable cell division and inflammation in the prostate gland. Telomere shortening, often caused by oxidative stress, plays a key role in the onset of the disease. Genetic factors like MYC, PTEN, NKX3.1, and TMPRSS2-ERG gene fusions are implicated in cancer initiation, with TMPRSS2-ERG fusions activating the ERG oncogenic pathway.[7] As cancer progresses and metastasizes, pathways related to cell division are reactivated, promoting uncontrolled proliferation. EZH2, which is overexpressed in metastatic prostate cancer, is a novel target due to its role in apoptosis and cell growth.

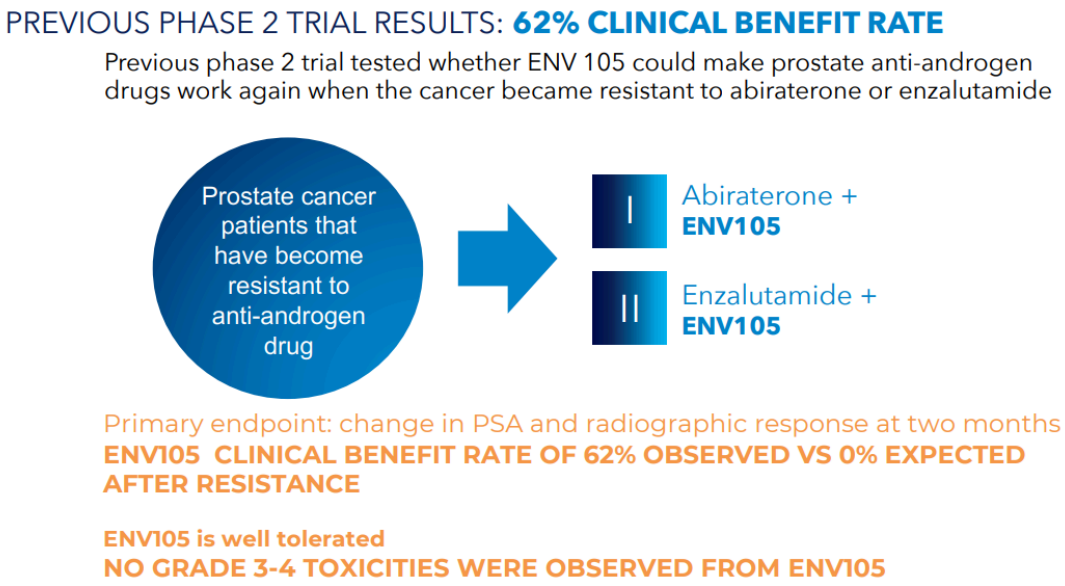
[5] Sekhoacha et al. 2022. Molecules. 21(15):670-800
[6] Heidenreich et al. 2016. Eur Urol. 65(2):467-479
[7] Rebello et al. 2021. Nature. 27(17):5730



Source: Shen et al.

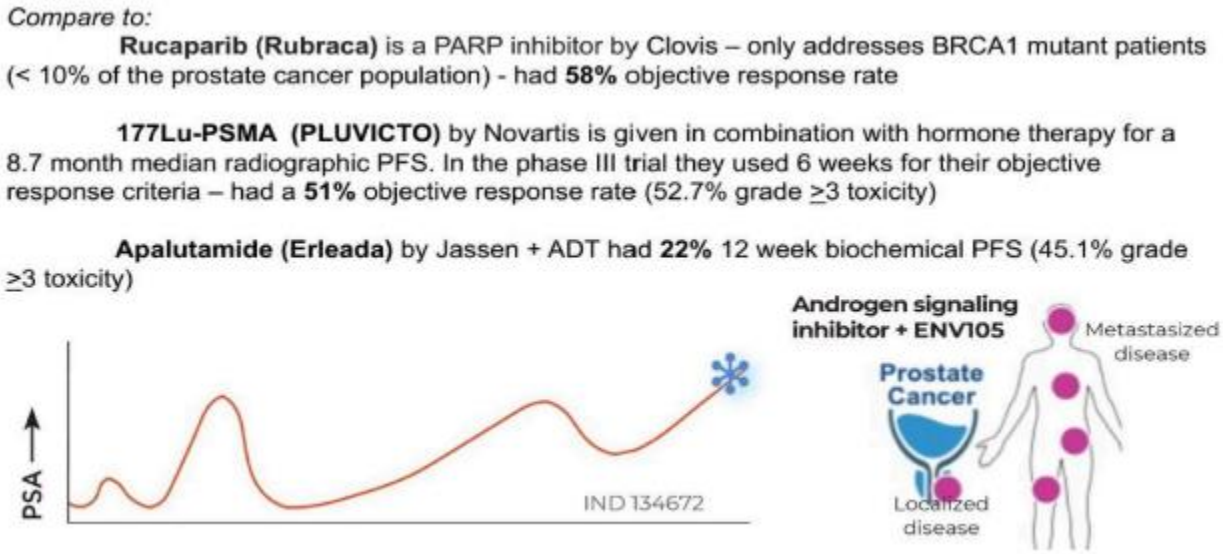
Current treatments. The current 1L therapy for early stage disease typically includes active surveillance, surgery (radical prostatectomy), or radiation therapy (external beam or brachytherapy), often combined with hormone therapy depending on the cancer's aggressiveness. Androgen deprivation therapy (ADT), a key treatment for advanced prostate cancer, is commonly used as the 1L approach for metastatic disease, blocking testosterone's effects to slow cancer growth. If cancer progresses after initial hormone therapy, 2L therapies may include newer generation hormone therapies like abiraterone acetate (Zytiga) or enzalutamide (Xtandi), which target the androgen receptor pathway, as well as chemotherapy with drugs like docetaxel. For metastatic castration-resistant prostate cancer (CRPC), therapies often involve targeted treatments such as Zytiga or Xtandi, depending on the individual case. In more advanced stages, radiopharmaceuticals such as radium-223 (Xofigo) and radioligand therapies like Pluvicto (lutetium Lu 177 vipivotide tetraxetan), which target prostate-specific membrane antigen (PSMA), provide additional treatment options for patients with mCRPC. These therapies are typically considered 3L+ treatments, especially for those with high PSMA expression.

Prior P2 data. A small Phase 2 trial at Cedars-Sinai Medical Center was initiated in 2018 to evaluate heavily pre-treated prostate cancer patients who had developed resistance to enzalutamide (Xtandi, Pfizer) or abiraterone (Zytiga, Janssen), both standard-of-care androgen-targeted therapies. The study's primary objective was to assess the clinical benefit rate (CBR) at two months, defined as disease stabilization or regression (complete or partial response). Among 11 enrolled patients (9 evaluable), a CBR of 62% was observed. The trial was initially designed to enroll 40 patients, but enrollment was halted early due to drug supply limitations from the manufacturer. The supply has since been expanded and secured by Kairos, allowing for further development in its ongoing P2.



Source: Karios corporate presentation

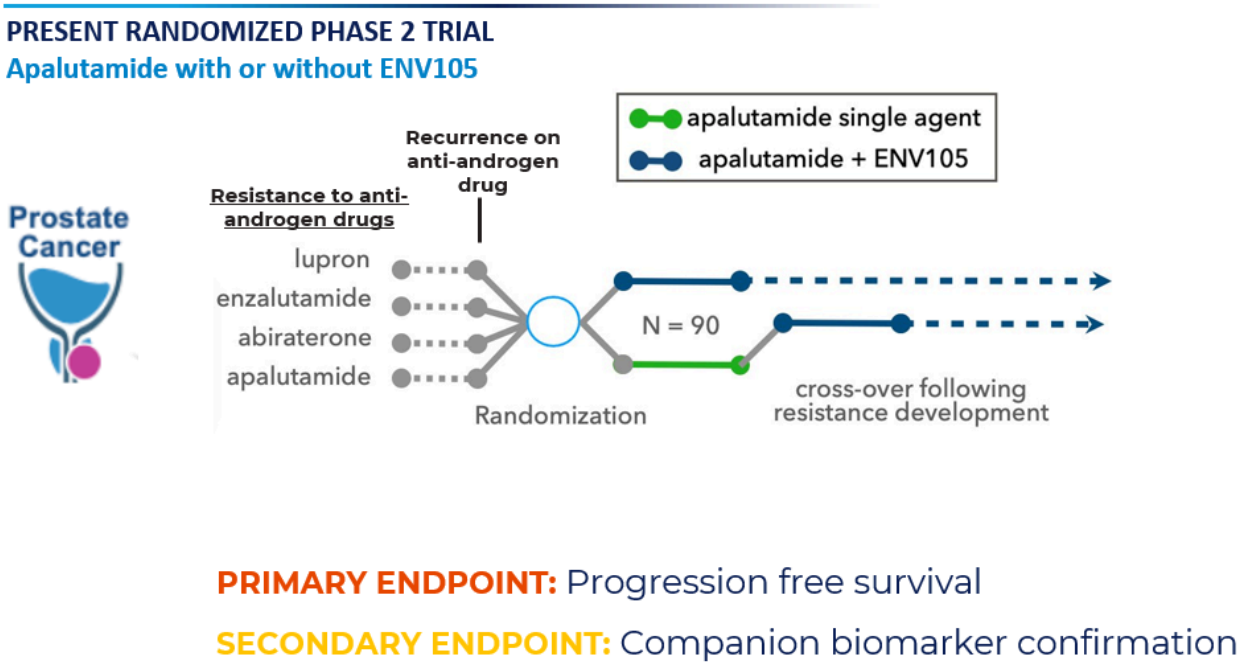
ENV-105 could be a potential first-in-class therapy. In addition to the 62% response rate observed in Phase 2, ENV-105 was also well tolerated compared to current AR inhibitors. No grade 3–4 toxicities were observed with the combination, which is superior to the drugs shown below.



Source: Karios corporate presentation

Ongoing P2 trial: ENV-105 + apalutamide (Erleada). This ongoing open-label P2 multicenter trial is being conducted at Cedars-Sinai, University of Utah, City of Hope, and Huntsman Cancer Institute. The trial aims to enroll N=90 patients, evaluating the combination of androgen signaling inhibitor (ARSI) Erleada (apalutamide) alone or in combination with ENV-105 for the treatment of castrate-resistant prostate cancer (CRPC) in patients who have failed ARSI therapy. Patients on single-agent Erleda will be allowed crossover to combination therapy upon recurrence, which is expected in ~2 months of treatment. The trial's primary endpoint is progression-free survival (PFS) as measured by

prostate-specific antigen (PSA) and radiographic response. The secondary endpoint is a companion biomarker evaluation. Note, the trial is partially funded by a $3.2M NIH grant The first patient was dosed on 1/30/24.



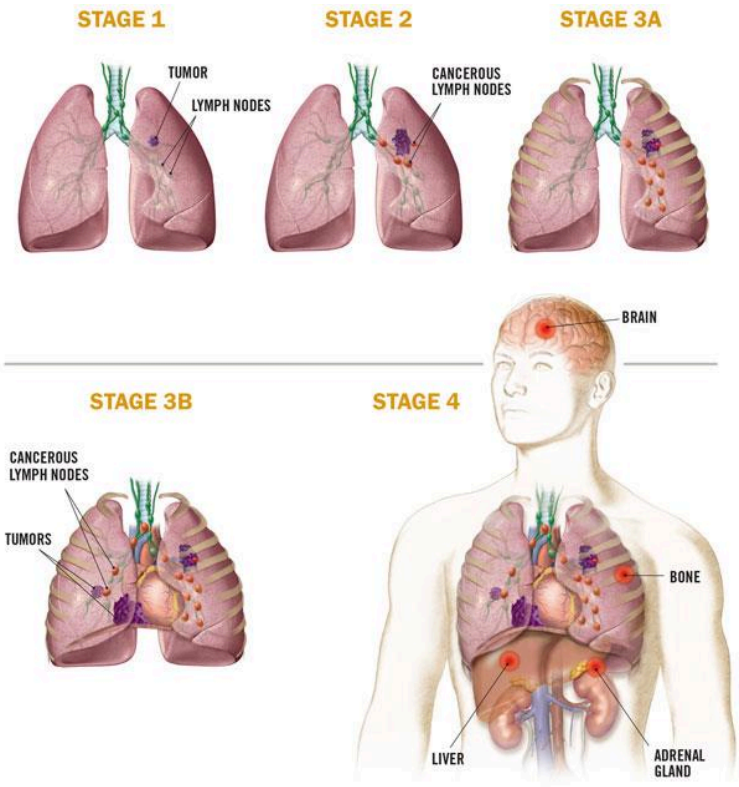
Source: Karios corporate presentation

Next steps. The company expects to report interim safety and efficacy data from the P2 study in 1H25.

Non-small cell lung cancer (NSCLC). NSCLC can be further divided into subtypes: adenocarcinoma (~40%), squamous cell carcinoma (25%–30%), and large cell (undifferentiated) carcinoma (10%–15%). While cancers found at an earlier stage generally have better prognosis, there are many other factors that can impact outcome, including the subtype of NSCLC, the person's age and overall health, and how susceptible the tumor is to treatment. The etiology of NSCLC can be further categorized into avoidable and unavoidable risk factors.[8] The most well-known avoidable risk factor for NSCLC is inhaled tobacco use. Other causes of lung cancer include alcohol use, environmental exposure to secondhand smoke, asbestos, radon, arsenic, chromium, nickel, as well as exposure to ionizing radiation, and polycyclic aromatic hydrocarbons. Symptoms typically begin to develop after the early stages of NSCLC and may include persistent coughing, shortness of breath, weight loss, or chest pain. When lung cancer metastasizes to distant organs, it may cause bone pain, headache, or dizziness. Despite recent advances in drug treatments, the 5-year survival rate for stage IV NSCLC patients is less than 1%. Survival was similar in recurrent disease regardless of stage at diagnosis, with median overall survival (OS) of 6.6 months for stage I, 6.7 months for stage II, and 6.9 months for those with initial stage III disease. Patients with de novo or recurrent stage IV disease had median OS of 4.9 months.[9]

Stages of non-small cell lung cancer (NSCLC).

[8] Febbraro et al. 2022. Curr Oncol. 29(3):1828-1839
[9] Joubert et al. 2020. Curr Oncol. 27(1):52-60



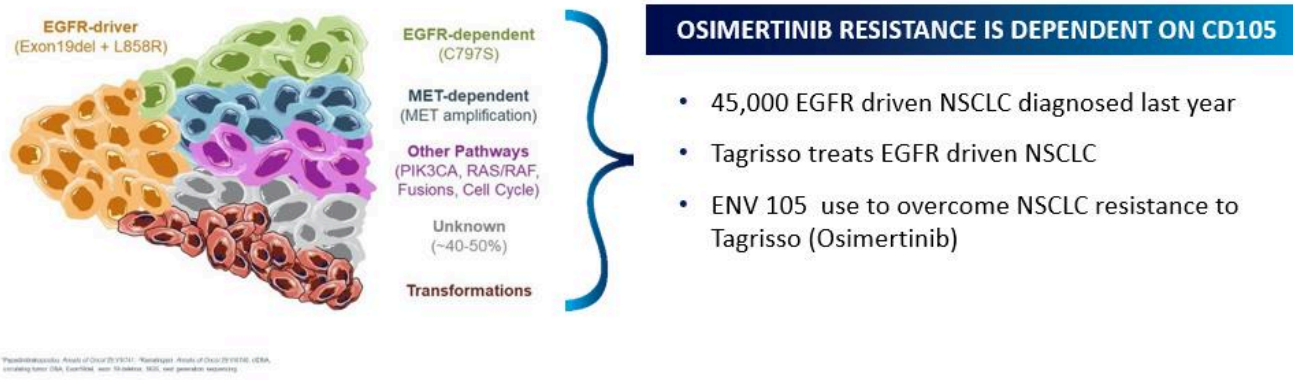
Source: Duma et al.

Current treatments. The standard treatment for the majority of advanced NSCLC cases is systemic therapy, where the medication is delivered intravenously or orally via the bloodstream to reach cancer cells throughout the body. Patients experiencing a recurrence of, or with advanced NSCLC, have few treatment options and are typically treated with chemotherapy. The most commonly used regimens include either cisplatin or carboplatin combined with one of several other drugs approved for the treatment of NSCLC, such as pemetrexed, paclitaxel, docetaxel, gemcitabine, irinotecan, or vinorelbine. Bevacizumab or ramucirumab, in combination with paclitaxel and carboplatin, is FDA-approved for 1L treatment of advanced, non-squamous NSCLC. Despite advances in treatment and management of recurrent advanced NSCLC, NSCLC represents a high unmet medical need. Novel therapies are needed to improve therapeutic outcomes, particularly for patients who do not have driver mutations for targeted therapies and/or immunotherapy, or those who have developed resistance to previous treatments. Systemic therapies include chemotherapy, targeted therapy, and checkpoint immunotherapy (e.g., anti-PD-1s and anti-PDL1s).

EGFR-mutated NSCLC. Epidermal growth factor receptor (EGFR)-mutated lung cancer occurs in 15–20% of patients with adenocarcinoma and is most commonly associated with nonsmokers and those of Asian ethnicity. EGFR-mutated NSCLC is primarily treated with EGFR tyrosine kinase inhibitors (TKIs) such as Erlotinib, Gefitinib, Afatinib, and Osimertinib.[10] Osimertinib (Tagrisso) is the standard 1L treatment for advanced or metastatic NSCLC patients with EGFR mutations. However, like other EGFR-TKIs, Osimertinib will inevitably develop acquired resistance, limiting its efficacy for EGFR-mutated NSCLC patients.[11] Therefore, there is a significant unmet need in this category, which presents a significant opportunity for Kairos and ENV-105 to potentially re-sensitize patients to Osimertinib in a 2L combination.

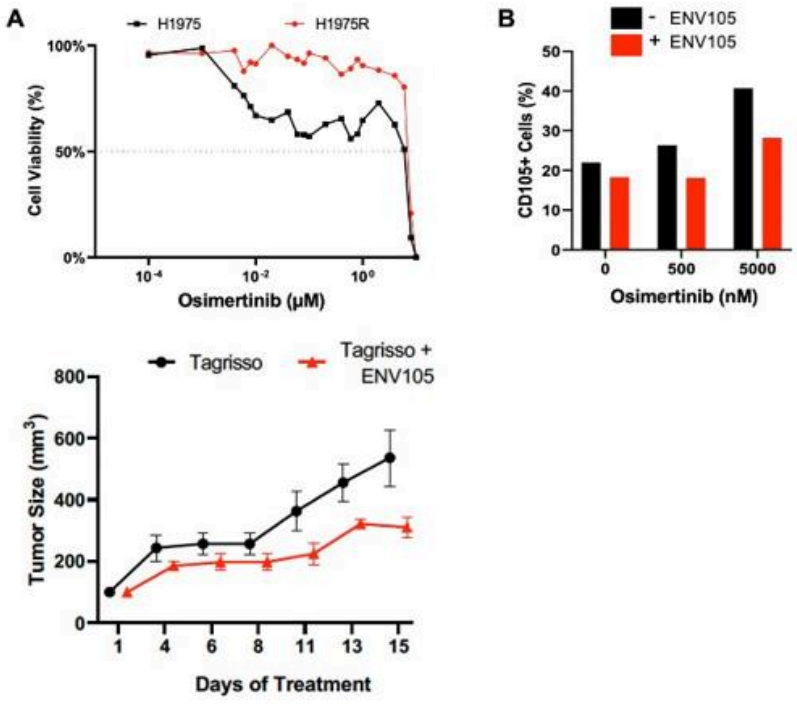
[10] Leonetti et al. 2019. Br J Cancer. 121(9):725-737
[11] Nilsson et al. 2021. J Thorac Oncol. 16(2):205-215

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



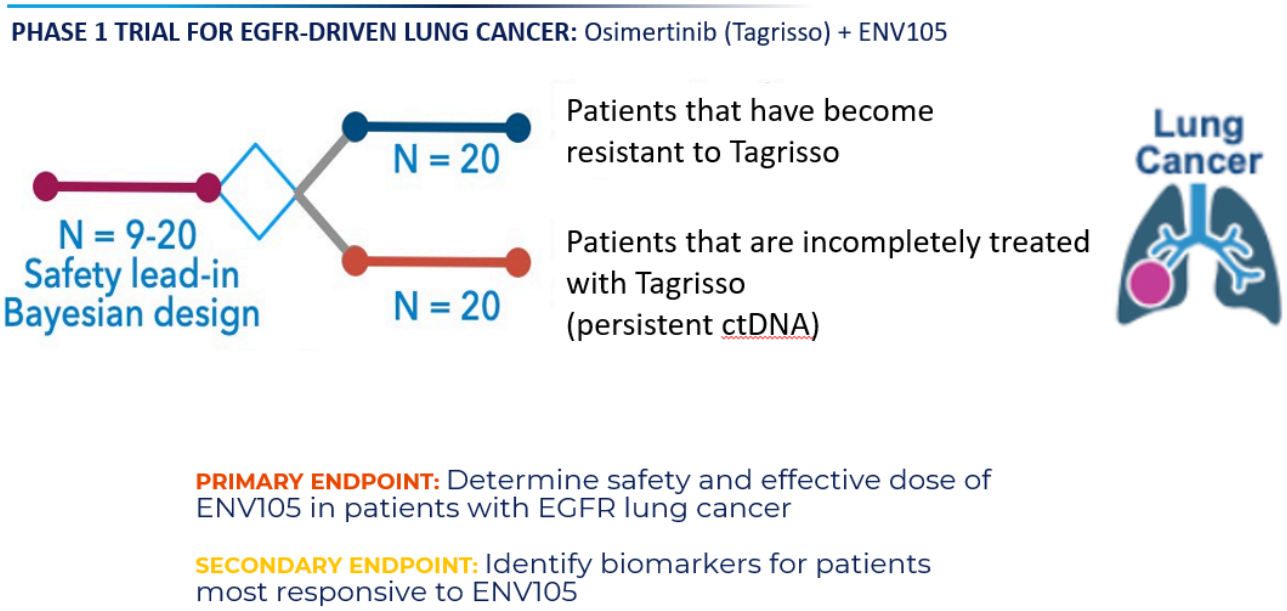
Source: Karios corporate presentation

Osimertinib-resistant lines appear sensitive to ENV-105 + Osimertinib combo. As shown below, EGFR-mutant NSCLC cells (H1975) made resistant to Osimertinib (H1975R) exhibited elevated CD105 expression, as confirmed by FACS analysis. Co-administration of ENV-105 effectively downregulated CD105 levels. In xenograft models of Osimertinib-resistant NSCLC, Osimertinib treatment alone led to tumor expansion, whereas the combination with ENV-105 significantly reduced tumor growth ($p < 0.0001$), demonstrating the potential of ENV-105 to restore Osimertinib sensitivity.



Source: Karios corporate presentation

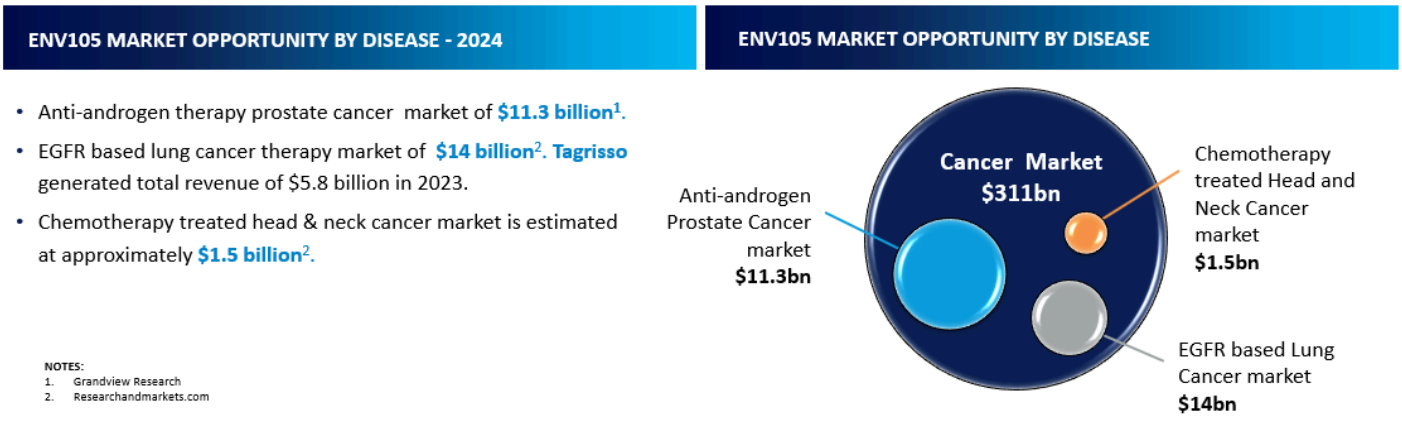
Ongoing P1 study: ENV-105 + Osimertinib (Tagrisso). This P1 study (initiated Sep. 2023) is evaluating the combination of ENV-105 and Osimertinib (Tagrisso) in EGFR-mutated non-small cell lung cancer (NSCLC) patients who have developed resistance or remain incompletely treated with detectable tumor ctDNA. The study, currently enrolling at Cedars-Sinai Medical Center, follows a Bayesian safety lead-in design with an initial cohort of 9–20 patients before expanding to two randomized arms of 20 patients each. One arm includes patients with progressive disease post-Tagrisso or other EGFR inhibitors, while the second arm consists of patients with persistent ctDNA at 6–12 weeks following frontline Osimertinib. The trial aims to determine the safety and recommended Phase 2 dose, with secondary endpoints assessing response and identifying predictive biomarkers. Patients exhibiting partial or complete resistance to Osimertinib will continue therapy while receiving ENV-105 to evaluate its potential in overcoming resistance. Biomarker analysis, including blood-based circulating tumor DNA, will guide patient selection and response assessment.



Source: Karios corporate presentation

Next steps. Kairos expects to report initial data from the P1 study by YE25.

ENV-105 market opportunity.



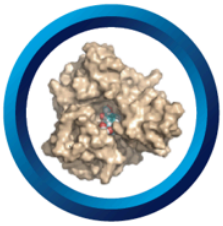
Source: Karios corporate presentation

Pipeline

KROS 101 is an orally available small molecule glucocorticoid-induced tumor necrosis factor receptor (GITR) ligand antagonist designed to deplete suppressive regulatory T cells (Tregs) and activate effector T cells, enhancing the antitumor immune response in solid cancers. By stabilizing the GITR ligand, KROS 101 signals the GITR receptor to promote effector T-cell functions and inhibit Treg suppression, making it a promising complement to current checkpoint inhibitors. Unlike antibody-based GITR treatments, KROS 101 fits into the GITR ligand to amplify T-cell proliferation through a more effective, dose-dependent mechanism. It also offers the potential for fewer side effects due to its modifiable half-life and reversibility. Currently in pre-IND studies, KROS 101 was developed through advanced structural biology techniques and is planned for a P1 trial.

KROS 101: GITR (glucocorticoid-induced tumor necrosis factor receptor) ligand is a powerful checkpoint that increases the T cell response against cancer.

Kairos Pharma developed a small molecule that increases T cell numbers and anti-tumor killing activity by acting as a GITR agonist.



Kairos identified structure of GITR ligand through X ray crystallography.

KROS-101 pictured here in the center of the GITR protein

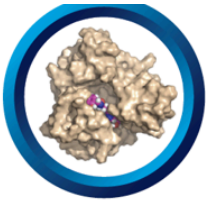
OTHER DRUGS TARGETING GITR RECEPTOR

COMPANY	DRUG	ACTIVITY
Kairos Pharma	**KROS101**	• Targets the GITR ligand (the signal for the receptor) enabling expansive growth of T cells • Small molecule has shorter half-life allows for more fine tuning of dose to limit side effects • Having both agonist (KROS101) and antagonist (KROS102) molecules allows reversal of side effects
AstraZeneca	**MEDI1873**	• Hexameric GITR ligand fusion protein does not enable significant T cell response
Merck	**MK4166**	• Agonist antibody has significant toxicity including autoimmune gastritis

Source: Karios corporate presentation

KROS 102 is a GITR antagonist designed to enhance Treg cell functions while suppressing T effector cell numbers and activity, targeting autoimmune diseases. It has been shown to increase Treg cells and decrease T effector cells in a dose-dependent manner. This novel GITR inhibitor is thought to address abnormal immune responses that attack the body's own tissues. Given GITR's central role in regulating Tregs, it is considered an ideal target for treating autoimmunity. KROS 102's potential therapeutic applications include autoimmune diseases such as Crohn's disease, multiple sclerosis, and rheumatoid arthritis. The compound is currently in preclinical studies.

- **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.

KROS-102 pictured here in the center of the GITR protein

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.

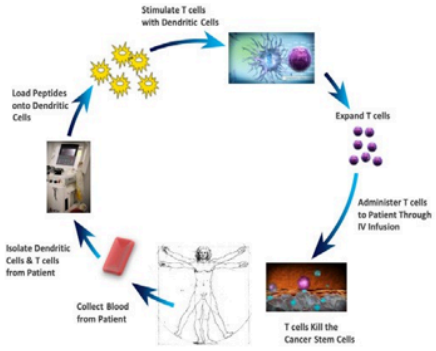
ADVANTAGE OF KROS102:

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

Source: Karios corporate presentation

KROS 201 is a proprietary technology that produces activated T cells from a patient's white blood cells. These killer T cells are generated by stimulating the cells with cytokines or T-cell activating signals and priming dendritic cells loaded with glioblastoma cancer stem cell-specific antigens. The activated T cells are then infused intravenously into patients with recurrent glioblastoma. KROS 201 aims to provide a "plug-and-play" T-cell therapy that can be tailored to a patient's specific tumor, enhancing cancer treatment by stimulating long-term populations of cytotoxic and helper T cells targeting the tumor. Kairos has completed IND-enabling pharmacology and toxicology studies and submitted an IND application.

KROS 201: Aims to generate activated T cells (ATC), ATC are killer T cells that are made from a patient's white blood cells in a cell culture by activating with cytokines or T cell activating signals and by priming dendritic cells loaded with glioblastoma cancer stem cell specific antigens



COMPETITIVE OFFERINGS

COMPETITOR	DRUG	ACTIVITY
Kairos Pharma	KROS-201	• **Generates potent activated T cells** that has been shown to improve cancer stem cell targeting • T cells kill the root of glioblastoma tumors by targeting several antigens
Ziopharm	Ad-RTS-hIL with veledimex and other gene therapies	• **Uses intracranial adenoviral delivery of viruses** which can cause cytokine storm and other untoward effects
Imvax	IGV-001 and other vaccines	• **Uses dendritic cells and antigen targets** that may not translate to a potent T cell response
Century Therapeutics	ET001 CAR and other CART cells	• T cells that target CD133 **may have off target untoward effects** on normal brain and blood stem cells. Antigen loss variants by targeting only one antigen.
Bristol Myers Squibb	Nivolumab and other checkpoint inhibitors	• **Failed due to absence of T cells in the tumor.** Activated T cells must be generated first such as through KROS-201

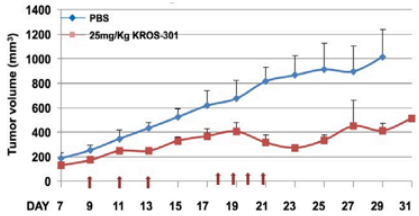
Source: Karios corporate presentation

KROS 301 is a tumor-targeting small molecule and checkpoint inhibitor that operates through two distinct mechanisms of action by blocking the intranuclear localization of RelA, a critical component of the NF-κB pathway. The NF-κB pathway plays a vital role in cancer growth, survival, and drug resistance, making it a key target for therapeutic intervention. KROS 301 specifically targets tumor cells in RelA/p65 biomarker-positive solid tumors, allowing for a more precise treatment approach. By identifying patients who express this biomarker, KROS 301 enables the selection of individuals most likely to respond to the therapy, optimizing the drug's effectiveness and minimizing unnecessary treatments.

KROS 301: A small molecule inhibitor aims to have two distinct mechanisms of action resulting from blocking intranuclear localization of RelA, a key component of the NF-KB pathway. NF-KB is a key component for cancer growth and immune resistance.

The use of RelA/p65 biomarker allows the treatment of only positive solid tumors which predicts responsiveness.

Efficacy of in a triple negative breast cancer animal model in inhibiting tumor growth



COMPETITIVE OFFERINGS

COMPETITOR	DRUG	ACTIVITY
Kairos Pharma	KROS-301	• Inhibits NF-KB activation without any effects on other signaling pathways. • Selectively targeting NF-KB signaling minimizes systemic toxicity. • NF-KB inhibition is transient and highly reversible to avoid long-term immunosuppression.
Takeda	Bortezomib (Velcade®)	• Bortezomib is a reversible 26S proteasome inhibitor that has been recently approved by FDA for the treatment of multiple myeloma. Although Bortezomib affects other signaling pathways, its efficacy may in part be due to inhibition of NF-KB activity. • The proteasome which is responsible for IκB degradation has many other important functions. Thus, inhibition of proteasome activity could potentially cause severe side effects.

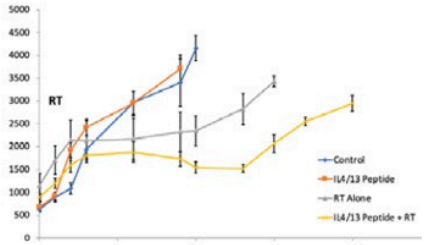
Source: Karios corporate presentation

KROS 401 is a tumor microenvironment immune modulator and a cyclic peptide inhibitor that targets IL-4 and IL-13, reversing the suppression caused by tumor-associated macrophages. By reducing the M2 macrophage population, KROS 401 helps limit fibrosis in the pancreas through its anti-inflammatory effects. Beyond pancreatic fibrosis, potential indications for KROS 401 include pulmonary fibrosis and other inflammatory conditions. Specifically, KROS 401 works by blocking IL-4 and IL-13 cytokine immune receptors, making it particularly effective for treating triple-negative breast cancer. Additionally, in preclinical animal models, KROS 401 has been shown to enhance the anti-tumor immune response when used in combination with radiation therapy, offering a promising approach to improving cancer treatment outcomes.

KROS 401: Cyclic peptide inhibitor of IL4/IL13 cytokine immune receptors for triple negative breast cancer. It aims to increase anti-tumor response in conjunction with radiation therapy

Recently, it became clear that macrophages in tumors are altered by the Th2 cytokines IL-4 and IL-13, inducing pro-tumor macrophages

AIM TO ENHANCE THE ANTI-TUMOR RESPONSE TO RADIATION THERAPY IN BREAST CANCER

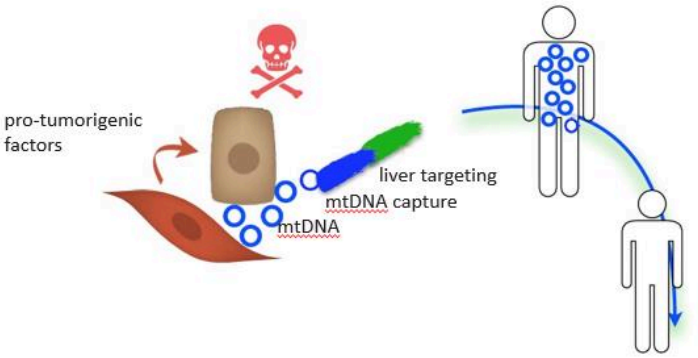


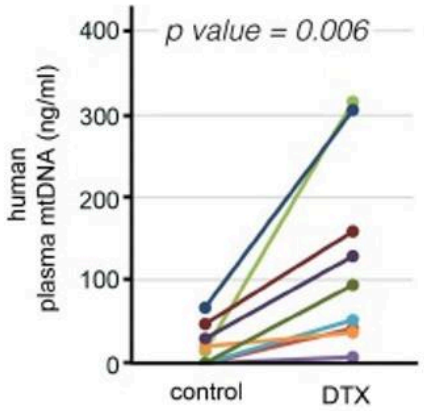
COMPETITIVE OFFERINGS

COMPETITOR	DRUG	ACTIVITY
Kairos Pharma	KROS-401	• Antibodies have not been studied in cancer or pancreatitis. • Our peptide binds to IL13R alpha1 and IL4R alpha1 (type I) receptor complex; blocks both IL-4 and IL-13 mediated signaling. • The implication is that targeting IL-4Ralpha is predominantly for indications such as asthma or eczema, while the type I is for macrophages/tumor growth (esp IL13R).
LEO Pharma	Tralokinumab	• Tralokinumab blocks IL-13 cytokine for asthma or eczema.
Regeneron Pharmaceuticals	Dupilumab	• Dupilumab binds to IL-4R alpha (both type I and 2) which then prevents IL-13 (forming a heterodimer) signaling for eczema.

Source: Karios corporate presentation

ENV 205 is a novel, first-of-its-kind biologic that targets prostate cancers that have become otherwise resistant to chemotherapy. ENV 205 targets the excretion of mitochondrial DNA, which is elevated in circulation when patients undergo chemotherapy. Higher blood levels of mitochondrial DNA are not only associated with chemotherapy resistance. However, they are more widely recognized as a mediator of cardiac toxicity and other systemic inflammatory events contributing to the negative side effects of chemotherapy use. As such, depleting mitochondrial DNA with the administration of ENV 205 restores chemotherapy sensitivity with reduced toxic side effects.





MECHANISM OF ACTION:
mtDNA depletion to limit inflammation-induced pro-tumorigenic activity

PRIMARY INDICATION: Prostate Cancer
COMBINATION THERAPY: Chemotherapy
PARTNER: Xencor Co.

OTHER INDICATIONS INCLUDE:

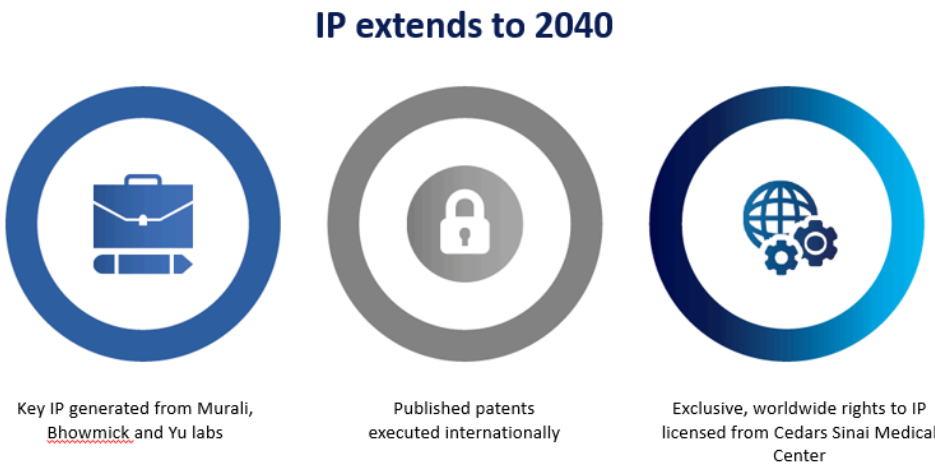
- Sensitize to docetaxel
- Radiation therapy lupus
- HIV

Source: Karios corporate presentation

Corporate history. Kairos Pharma, initially incorporated as NanoGB13, Inc. in California on June 17, 2013, and changed its name to Kairos Pharma on July 15, 2016. On May 10, 2023, the company converted to a Delaware corporation by filing certificates of conversion with both California and Delaware, and conducted a 1-for-2.5 reverse stock split, reducing its outstanding shares to 10,334,357. In 2019, Kairos merged with AcTcell Biopharma, issuing 5,045,000 shares for AcTcell's stock, and AcTcell's sole asset was a license agreement with Cedars-Sinai. Later in 2019, Kairos acquired Enviro Therapeutics in a share exchange, issuing 6,000,000 shares of Kairos stock, which gave Enviro's shareholders approximately 20% voting power in Kairos. This included the issuance of shares to Dr. Yu, Dr. Bhowmick, and Tracon Pharmaceuticals. Prior to the reverse stock split, Kairos had approximately 19,825,957 shares outstanding on a fully diluted basis.

License agreement with Tracon. On May 21, 2021, Enviro Therapeutics entered into a license agreement with Tracon Pharmaceuticals, granting Enviro access to inactive IND filings for TRC105 in the US, ownership of TRC105 drug product vials manufactured to GMP standards, and assignment of Tracon's CD105 patent rights. Enviro paid Tracon an upfront fee of $100,000 and is obligated to make additional payments based on equity financings. The agreement also includes a 3% royalty on net sales and non-royalty payments for sublicensing fees. In exchange for its Enviro shares, Tracon received 280,000 shares of Kairos stock, equal to 1.41% of Kairos on a fully diluted basis. The agreement grants exclusive licensing rights to multiple US and foreign patents. Tracon, no longer an operating company following the failure of its injectable PD-L1 inhibitor, had previously struggled with low response rates in clinical trials for its subcutaneous PD-L1 inhibitor, Envafolimab.

Intellectual property.



Pub #	KROS101 PCT/US2019/045478	KROS201 PCT/US2020/045570	KROS301 PCT/US2015/050906	KROS401 PCT/US2016/035318	ENV105 PCT/US2017/037558
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	Sensitization Of Tumors To Therapies Through Endoglin Antagonism

Source: Karios corporate presentation

Risks

The risk factors presented below could negatively impact the overall performance and prospects of Kairos Pharma Ltd. and its stock price:

- **Clinical Risk:** While antibody-based therapies have seen success in oncology, CD105 is a novel target that has not been commercially validated. Clinical evidence supporting ENV-105 in combination with anti-androgen therapy remains early and requires further validation. Success in clinical trials is not guaranteed, and setbacks or failures could significantly impact Kairos' ability to advance its pipeline and bring products to market.

- **Financial Risk:** Kairos has no approved products or revenue and has incurred losses since inception. The company will need to raise additional capital to fund its operations and pipeline development, and failure to secure sufficient funding could negatively impact its share price or force it to cease operations. There is also no assurance that Kairos will be able to raise capital on favorable terms.

- **Regulatory Risk:** The FDA, EMA, or other regulatory agencies may assess the benefit-risk profile of Kairos' product candidates differently, potentially leading to delays, additional trial requirements, or failure to obtain approval. The regulatory process is inherently uncertain, and setbacks could increase development costs and prolong time to market, adversely affecting the company's ability to commercialize its product candidates.

- **Commercial Risk:** Clinical and regulatory success does not guarantee commercial success. Kairos has no experience commercializing products and may face challenges with market adoption, pricing, and reimbursement. Payer restrictions and competitive dynamics could limit market penetration, impacting the company's ability to generate revenue.

- **Competitive Risk**: Kairos' ENV-105 faces competition from existing and emerging prostate cancer therapies. Pluvicto is approved for PSMA-positive metastatic CRPC, and experimental PSMA-targeting T-cell engagers such as JANX007 and VIR-5500 could compete in the same setting as ENV-105. If ENV-105 cannot demonstrate competitive differentiation from competing products, the company's commercial opportunities could be significantly impaired.

MODELING ASSUMPTIONS

1. We model commercialization of ENV-105 in 2L metastatic castration-resistant prostate cancer (mCRPC) in 2030 in the US.
2. We assume a prostate cancer incidence of 330K per year in the US and that 20% of patients have metastatic castration-resistant prostate cancer (mCRPC). This represents a potential total addressable market of ~70K patients**.**
3. We assume an initial pricing of $180K, which is on par with the average wholesale price (WAC) of Xtandi (enzalutamide). We factor in a 2% y/y price increase.
4. We factor in a revenue risk adjustment of 80% based on stage of development, clinical trial risk, regulatory risk, and commercial risk.

ENV-105, 2L metastatic castration-resistant prostate cancer (mCRPC) market model.

ENV-105, 2L mCRPC	2023E	2024E	2025E	2026E	2027E	2028E	2029E	2030E	2031E	2032E	2033E	2034E	2035E
Prostate cancer (PC) incidence	336,000	342,720	349,574	356,566	363,697	370,971	378,391	385,958	393,678	401,551	409,582	417,774	426,129
mCRPC patients (20%)	67,200	68,544	69,915	71,313	72,739	74,194	75,678	77,192	78,736	80,310	81,916	83,555	85,226
Market Penetration								2.00%	5.00%	8.00%	10.00%	12.00%	15.00%
Total Patients Treated								1,544	3,937	6,425	8,192	10,027	12,784
Cost of Treatment								180,000	183,600	187,272	191,017	194,838	198,735
Increase in Cost								2%	2%	2%	2%	2%	2%
Total revenue ('000)								$ 277,890	$ 722,792	$ 1,203,188	$ 1,564,747	$ 1,953,555	$ 2,540,598
Risk adjustment								80%	80%	80%	80%	80%	80%
Total Revenue ('000)								**$ 55,578**	**$ 144,558**	**$ 240,638**	**$ 312,949**	**$ 390,711**	**$ 508,120**

Source: Maxim Estimates

1. We model commercialization of ENV-105 in 2L EGFR-mutated non-small cell lung cancer (NSCLC) in 2031 in the US.
2. We assume a non-small lung cancer incidence of 200K per year in the US and that 20% of patients have EGFR-mutated disease. This represents a potential total addressable market of ~45K patients**.**
3. We assume an initial pricing of $100K, which is on par with the average wholesale price (WAC) of Tagrisso (Osimertinib). We factor in a 2% y/y price increase.
4. We factor in a revenue risk adjustment of 80% based on stage of development, clinical trial risk, regulatory risk, and commercial risk.

ENV-105 + Osimertinib, 2L EGFR-mutated non-small cell lung cancer (NSCLC) market model.

ENV-105 + osimertinib, 2L EGFR mutated NSCLC	2023E	2024E	2025E	2026E	2027E	2028E	2029E	2030E	2031E	2032E	2033E	2034E	2035E
Non-small cell lung cancer (NSCLC) incidence	195,500	199,410	203,398	207,466	211,615	215,848	220,165	224,568	229,059	233,641	238,313	243,080	247,941
2L EGFR-mutated patients (20%)	39,100	39,882	40,680	41,493	42,323	43,170	44,033	44,914	45,812	46,728	47,663	48,616	49,588
Market Penetration									5.00%	10.00%	12.00%	15.00%	18.00%
Total Patients Treated									2,291	4,673	5,720	7,292	8,926
Cost of Treatment									100,000	102,000	104,040	106,121	108,243
Increase in Cost									2%	2%	2%	2%	2%
Total revenue ('000)									$ 229,059	$ 476,627	$ 595,059	$ 773,874	$ 966,167
Risk adjustment									80%	80%	80%	80%	80%
Total Revenue ('000)									**$ 45,812**	**$ 95,325**	**$ 119,012**	**$ 154,775**	**$ 193,233**

Source: Maxim Estimates

VALUATION

We model commercialization of ENV-105 in metastatic castration-resistant prostate cancer (mCRPC) in 2030 and EGFR-mutated non-small cell lung cancer (NSCLC) in 2031 in the US. An 80% revenue risk adjustment is factored in primarily based on stage of development and clinical trial risk. A 30% discount rate is then applied to the free cash flow, discounted EPS, and sum-of-the-parts models, which are equally weighted to derive a 12-month price target of $4.

Free Cash Flow Model.

Average	$4

DCF Valuation Using FCF (mln):

	2024	2025	2026	2027	2028	2029	2030	2031	2032	2033	2034	2035
units ('000)	**2024E**	**2025E**	**2026E**	**2027E**	**2028E**	**2029E**	**2030E**	**2031E**	**2032E**	**2033E**	**2034E**	**2035E**
EBIT	(1,630)	(2,452)	(6,868)	(11,498)	(14,796)	(16,654)	14,574	94,511	180,881	237,445	304,473	396,853
Tax Rate	0%	0%	0%	0%	0%	0%	0%	0%	2%	5%	8%	10%
EBIT (1-t)	(1,630)	(2,452)	(6,868)	(11,498)	(14,796)	(16,654)	14,574	94,511	177,263	225,573	280,115	357,167
CapEx	-	-	-	-	-	-	-	-	-	-	-	-
Depreciation	120	-	-	-	-	-	-	-	-	-	-	-
Change in NWC												
FCF	(1,510)	(2,452)	(6,868)	(11,498)	(14,796)	(16,654)	14,574	94,511	177,263	225,573	280,115	357,167
PV of FCF	(2,038)	(2,452)	(5,087)	(6,309)	(6,014)	(5,014)	3,250	15,613	21,691	20,446	18,808	17,764
Discount Rate	35%											
Long Term Growth Rate	1%											
Terminal Cash Flow 2035E	1,523,483											
Terminal Value YE2035E	75,770											
NPV (2025 - 2035)	147,593											
NPV-Debt												
Shares out ('000)	31,956	2035E										
NPV Per Share	$5											

Source: Maxim estimates

Discounted-EPS Model.

Current Year	2025
Year of EPS	2035
Earnings Multiple	5
Discount Factor	30%
Selected Year EPS	11.18
NPV	$4

Source: Maxim estimates

	Discount Rate and Earnings Multiple Varies, Year is Constant						
	4.05	**5%**	**10%**	**15%**	**20%**	**25%**	**30%**
Earnings Multiple	**5**	34.31	21.55	13.81	9.03	6.00	4.05
	8	54.89	34.47	22.10	14.44	9.60	6.49
	10	68.62	43.09	27.63	18.05	12.00	8.11
	12	82.34	51.71	33.15	21.66	14.40	9.73
	15	102.93	64.64	41.44	27.08	18.00	12.16
	20	137.23	86.18	55.26	36.10	24.00	16.22
	25	171.54	107.73	69.07	45.13	30.00	20.27
	30	205.85	129.28	82.88	54.15	36.00	24.32

Sum-of-the-Parts Model.

	LT Gr	Discount Rate	Yrs to Mkt	% Success	Peak Sales (MM's)	Term Value
ENV-105, 2L mCRPC	1%	30%	6	50%	$508	$1,752
NPV						$2.89
ENV-105 + osimertinib, 2L EGFR mutated NSCLC	1%	30%	7	40%	$193	$666
NPV						$1
Net Margin						51%
MM Shrs OS (2035E)						32
Total						$4

Source: Maxim estimates

Karios Pharma, Ltd..: Income Statement ($000)																		
YE December 31	**3Q24A**	**4Q24E**	**2024E**	**1Q25E**	**2Q25E**	**3Q25E**	**4Q25E**	**2025E**	**2026E**	**2027E**	**2028E**	**2029E**	**2030E**	**2031E**	**2032E**	**2033E**	**2034E**	**2035E**
Revenue:																		
ENV-105, 2L mCRPC	-	-	-	-	-	-	-	-	-	-	-	-	55,578	144,558	240,638	312,949	390,711	508,120
ENV-105 + osimertinib, 2L EGFR mutated NSCLC	-	-	-	-	-	-	-	-	-	-	-	-	-	45,812	95,325	119,012	154,775	193,233
Net revenue	-	-	-	-	-	-	-	-	-	-	-	-	55,578	190,370	335,963	431,961	545,486	701,353
Collaborative revenue:																		
Revenues																		
Other Income			-					-	-	-	-	-	-	-	-	-	-	-
			-					-	-	-	-	-	-	-	-	-	-	-
Total Collaborative Revenue	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Total Revenue	-	-	-	-	-	-	-	-	-	-	-	-	55,578	190,370	335,963	431,961	545,486	701,353
Gross Margins:																		
Cost of Goods Sold	-	-	-	-	-	-	-	-	-	-	-	-	22,231	76,148	134,385	172,784	218,194	280,541
%Gross Margin													70%	70%	70%	70%	70%	70%
Gross Profit	-	-	-	-	-	-	-	-	-	-	-	-	33,347	114,222	201,578	259,177	327,291	420,812
Operating Expenses:																		
Research and Development	14	29	43	309	323	350	363	1,345	4,709	8,476	11,019	12,121	13,333	13,999	14,699	15,434	16,206	17,016
%R&D																		
Selling, General and Administrative	369	554	923	255	266	288	299	1,107	2,159	3,022	3,778	4,533	5,440	5,712	5,997	6,297	6,612	6,943
%SG&A																		
Total Expenses	383	583	966	564	589	638	662	2,452	6,868	11,498	14,796	16,654	41,004	95,859	155,082	194,516	241,013	304,500
Operating Income (Loss)	(383)	(583)	(966)	(564)	(589)	(638)	(662)	(2,452)	(6,868)	(11,498)	(14,796)	(16,654)	14,574	94,511	180,881	237,445	304,473	396,853
			-					-	-	-	-	-	-	-	-	-	-	-
Interest expense	(12)		(12)					-	-	-	-	-	-	-	-	-	-	-
Financing costs	(537)		(537)					-	-	-	-	-	-	-	-	-	-	-
Debt discount amortization	(115)		(115)					-	-	-	-	-	-	-	-	-	-	-
			-					-	-	-	-	-	-	-	-	-	-	-
			-					-	-	-	-	-	-	-	-	-	-	-
Total Other Income	**(664)**	-	**(664)**	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Pretax Income	(1,047)	(583)	(1,630)	(564)	(589)	(638)	(662)	(2,452)	(6,868)	(11,498)	(14,796)	(16,654)	14,574	94,511	180,881	237,445	304,473	396,853
Taxes on income	-	-	-	-	-	-	-	-	-	-	-	-	-	-	3,618	11,872	24,358	39,685
Tax Rate															2%	5%	8%	10%
GAAP Net Income (Loss)	(1,047)	(583)	(1,630)	(564)	(589)	(638)	(662)	(2,452)	(6,868)	(11,498)	(14,796)	(16,654)	14,574	94,511	177,263	225,573	280,115	357,167
Foreign currency translation loss																		
Total comprehensive loss	(1,047)	(583)	(1,630)	(564)	(589)	(638)	(662)	(2,452)	(6,868)	(11,498)	(14,796)	(16,654)	14,574	94,511	177,263	225,573	280,115	357,167
GAAP-EPS	(0.10)	(0.05)	(0.30)	(0.05)	(0.05)	(0.05)	(0.05)	(0.20)	(0.48)	(0.68)	(0.73)	(0.66)	0.51	3.01	5.61	7.12	8.80	11.18
GAAP-EPS (Dil)	(0.10)	(0.05)	(0.30)	(0.05)	(0.05)	(0.05)	(0.05)	(0.20)	(0.48)	(0.68)	(0.73)	(0.66)	0.51	3.01	5.61	7.12	8.80	11.18
Wgtd Avg Shrs (Bas) - '000s	10,910	10,921	5,458	10,932	12,443	12,455	12,468	12,074	14,376	16,937	20,133	25,221	28,704	31,449	31,575	31,701	31,828	31,956
Wgtd Avg Shrs (Dil) - '000s	10,910	10,921	5,458	10,932	12,443	12,455	12,468	12,074	14,376	16,937	20,133	25,221	28,704	31,449	31,575	31,701	31,828	31,956

Source: Company reports and Maxim

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Maxim Group LLC Ratings Distribution			As of: 03/26/25
		% of Coverage Universe with Rating	% of Rating for which Firm Provided Banking Services in the Last 12 months
Buy	Fundamental metrics and/or identifiable catalysts exist such that we expect the stock to outperform its relevant index over the next 12 months.	**84%**	**52%**
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	**See valuation section for company specific relevant indices*		

I, **Jason McCarthy, Ph.D.**, attest that the views expressed in this research report accurately reflect my personal views about the subject security and issuer. Furthermore, no part of my compensation was, is, or will be directly or indirectly related to the specific recommendation or views expressed in this research report.

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Maxim Group makes a market in Kairos Pharma, Ltd.

Maxim Group received compensation for investment banking services from Kairos Pharma, Ltd. in the past 12 months.

Maxim Group expects to receive or intends to seek compensation for investment banking services from Kairos Pharma, Ltd. in the next 3 months.

KAPA: For Kairos Pharma, Ltd., we use the BTK (Biotechnology Index) as the relevant index.

Valuation Methods

KAPA: We model commercialization of ENV-105 in metastatic castration-resistant prostate cancer (mCRPC) and EFGR-mutated non-small cell lung cancer (NSCLC). We apply a revenue risk adjustment based primarily on the stage of development and clinical trial

risk. A discount rate is then applied to the free cash flow, discounted EPS, and sum-of-the-parts models, which are equally weighted to derive a 12-month price target.

Price Target and Investment Risks
KAPA: Aside from general market and other economic risks, risks particular to our price target and rating for Kairos Pharma, Ltd. include: (1) the regulatory and clinical risk associated with product development; (2) the rate and degree of progress of product development; (3) the rate of regulatory approval and timelines to potential commercialization of products; (4) the level of success achieved in clinical trials; (5) the requirements for marketing authorization from regulatory bodies in the United States and other countries; (6) the liquidity and market volatility of the company's equity securities; (7) regulatory and manufacturing requirements and uncertainties; (8) product and technology developments by competitors, potentially with more resources and commercial infrastructure; (9) inability, of product(s), if approved, to gain adequate market share and maintain adequate revenue growth; (10) the ability of the company to maintain its exchange listing; (11) the ability to access capital to fund operations, if the company cannot secure sufficient capital, the company could cease operations; (12) Kairos is a controlled company, with insiders controlling over 50% of the voting rights; (13) recent changes to NYSE Section 802.01C limit listed issuers' ability to use multiple reverse stock splits to remedy listing requirements, thereby putting the stock at a higher risk of being delisted in the future.

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