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): November 28, 2022

IKENA ONCOLOGY, INC.
(Exact name of registrant as specified in its charter)

Delaware	**001-40287**	**81-1697316**
(State or other jurisdiction of incorporation)	**(Commission File Number)**	**(I.R.S. Employer Identification No.)**

Ikena Oncology, Inc.
645 Summer Street, Suite 101
Boston, Massachusetts 02210
(Address of principal executive offices, including zip code)

(857) 273-8343
(Registrant's telephone number, including area code)

Not Applicable
(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	Trade Symbol(s)	Name of each exchange on which registered
Common Stock, $0.001 par value per share	**IKNA**	**The Nasdaq Global Market**

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 7.01 Regulation FD Disclosure.

On November 28, 2022, Ikena Oncology, Inc. (the "Company") issued a press release announcing a next-generation mitogen-activated protein kinase ("MEK")-RAF complex inhibitor, IK-595, as the Company's first development candidate in the RAS pathway, as well as providing a corporate update. The Company also updated its corporate presentation. A copy of the press release and corporate presentation are attached as Exhibits 99.1 and 99.2, respectively, to this Current Report on Form 8-K. The corporate presentation will also be available in the investor relations section of the Company's website at https://www.ikenaoncology.com/.

The information in this Current Report on Form 8-K (including Exhibits 99.1 and 99.2) 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 the liabilities of that section, nor shall it be deemed incorporated by reference in any filing under the Securities Act of 1933, as amended, or the Exchange Act, except as expressly set forth by specific reference in such filing.

Item 8.01 Other Items.

On November 28, 2022, the Company announced a next-generation MEK-RAF complex inhibitor, IK-595, has been nominated as the company's first development candidate in the RAS pathway.

The RAS pathway is implicated in at least half a million new cancer diagnosis each year in the United States alone. Ikena aims to target the pathway on multiple levels, including preventing known resistance mechanisms to achieve deep and sustained responses. Ikena's new development candidate, IK-595, traps MEK and RAF in an inactive complex, more completely inhibiting RAS signals than existing inhibitors. IK-595's ability to complex CRAF, in particular, prevents a well-recognized signaling bypass mechanism that cancer cells employ to drive therapeutic resistance to other drugs in this class. In addition, trapping CRAF in an inactive complex prevents the kinase independent anti-apoptotic function in RAS and RAF mutant cancers, a mechanism that cannot be addressed with first generation MEK inhibitors or pan-RAF inhibitors. IK-595 is being developed as an oral therapy, with a half-life enabling a pharmacokinetic profile potentially superior to other drugs, with the goal of developing an optimal therapeutic window for patients. The company plans to submit an investigational new drug application (IND) for IK-595 to the US Food & Drug Administration (FDA) in the second half of 2023.

Development Highlights, Corporate Updates, and Upcoming Milestones

- Advancing new development candidate, IK-595, through IND-enabling studies, targeting MEK-RAF through novel mechanisms aiming to address existing gaps in the MEK inhibitor space
  - Preclinical differentiation data planned for presentation in the first half of 2023
  - IND targeted in the second half of 2023
- Progressing novel, paralog-selective transcriptional enhanced associate domain (TEAD) inhibitor, IK-930, in the clinic and further defining differentiation profile from panTEAD inhibition
  - Monotherapy program progressing as planned, advanced through multiple dose escalation cohorts
  - Preclinical data on differentiation and advantages of paralog selectivity planned for presentation in first half of 2023
  - Initiation of osimertinib combination cohort clinical program expected in first half of 2023
  - Initial clinical data from IK-930 monotherapy program expected in second half of 2023
- Following a portfolio review, discontinuing the internal clinical development of the EP4 antagonist IK-007 and exploring strategic alternatives for this program
  - Clinical data from IK-007 in microsatellite stable colorectal cancer (MSS-CRC) will be presented in a poster at 2022 European Society for Medical Oncology Immuno-Oncology Congress
- Portfolio reprioritization and streamlining of discovery and clinical activities contribute to extension of cash runway into 2025
  - Runway does not include any potential licensing revenue from the AHR antagonist IK-175 program, currently in development in collaboration with Bristol Myers Squibb and eligible for opt-in through early 2024

Item 9.01. Financial Statements and Exhibits.

(d) Exhibits

99.1	Ikena Oncology, Inc. Press Release
99.2	Ikena Oncology, Inc. Corporate Presentation
104	Cover Page Interactive Data File

SIGNATURE

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

Ikena Oncology, Inc.

Date: November 28, 2022

By: /s/ Mark Manfredi
Mark Manfredi, Ph.D.
President and Chief Executive Officer

Exhibit 99.1

Ikena Oncology Announces New Program in Next-Generation MEK-RAF Inhibition and Provides Corporate Update

Novel best-in-class MEK-RAF complex inhibitor IK-595 targets RAS pathway; IND in 2023

Novel paralog-selective TEAD inhibitor IK-930 advancing as planned in dose escalation; clinical data in 2023

Company prioritizes advancement of targeted oncology portfolio; cash runway extended into 2025

BOSTON, November 28, 2022 – Ikena Oncology, Inc. (Nasdaq: IKNA, "Ikena"), a targeted oncology company forging new territory in patient-directed cancer treatment, today announced a next-generation mitogen-activated protein kinase (MEK)-RAF complex inhibitor, IK-595, has been nominated as the company's first development candidate in the RAS pathway.

The RAS pathway is implicated in at least half a million new cancer diagnosis each year in the United States alone. Ikena aims to target the pathway on multiple levels, including preventing known resistance mechanisms to achieve deep and sustained responses. Ikena's new development candidate, IK-595, traps MEK and RAF in an inactive complex, more completely inhibiting RAS signals than existing inhibitors. IK-595's ability to complex CRAF, in particular, prevents a well-recognized signaling bypass mechanism that cancer cells employ to drive therapeutic resistance to other drugs in this class. In addition, trapping CRAF in an inactive complex prevents the kinase independent anti-apoptotic function in RAS and RAF mutant cancers, a mechanism that cannot be addressed with first generation MEK inhibitors or pan-RAF inhibitors. IK-595 is being developed as an oral therapy, with a half-life enabling a pharmacokinetic profile potentially superior to other drugs, with the goal of developing an optimal therapeutic window for patients. The company plans to submit an investigational new drug application (IND) for IK-595 to the US Food & Drug Administration (FDA) in the second half of 2023.

"I am thrilled to announce this addition to our pipeline, one that adds to our holistic approach to the Hippo & RAS oncosignaling network. IK-595 inhibits multiple nodes of MEK-RAF signaling, including avoiding known mechanisms of resistance," said Mark Manfredi, PhD, Ikena's Chief Executive Officer. "The industry's excitement about MEK inhibition stems from its great potential as a target, but the challenges of CRAF bypass and achieving optimal target inhibition have kept first generation treatments from meeting the full potential of the target. We are hopeful that our MEK-RAF trapping approach with an optimized therapeutic window can make IK-595 a game-changing candidate for multiple indications in the RAS space."

Development Highlights, Corporate Updates, and Upcoming Milestones

- Advancing new development candidate, IK-595, through IND-enabling studies, targeting MEK-RAF through novel mechanisms aiming to address existing gaps in the MEK inhibitor space
    - Preclinical differentiation data planned for presentation in the first half of 2023
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    - Monotherapy program progressing as planned, advanced through multiple dose escalation cohorts

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- Following a portfolio review, discontinuing the internal clinical development of the EP4 antagonist IK-007 and exploring strategic alternatives for this program
  - Clinical data from IK-007 in microsatellite stable colorectal cancer (MSS-CRC) will be presented in a poster at 2022 European Society for Medical Oncology Immuno-Oncology Congress
- Portfolio reprioritization and streamlining of discovery and clinical activities contribute to extension of cash runway into 2025
  - Runway does not include any potential licensing revenue from the aryl hydrocarbon receptor (AHR) antagonist IK-175 program, currently in development in collaboration with Bristol Myers Squibb and eligible for opt-in through early 2024

“Ikena has a strong track record of internal drug discovery and development that has built our robust clinical and early-stage pipeline,” said Jotin Marango, MD, PhD, Chief Financial Officer and Head of Corporate Development of Ikena. “Our goal is to continue our leadership in targeted oncology by discovering and advancing best-in-class candidates like IK-930 and IK-595, therapies designed to address the needs of specific patient populations while at the same time building value for our shareholders.”

About Ikena Oncology

Ikena Oncology™ is focused on developing differentiated therapies for patients in need that target nodes of cancer growth, spread, and therapeutic resistance in the Hippo and RAS onco-signaling network. The Company’s lead targeted oncology program, IK-930, is a paralog-selective TEAD inhibitor addressing the Hippo signaling pathway, a known tumor suppressor pathway that also drives resistance to multiple targeted therapies. The Company’s additional research spans other targets in the Hippo pathway as well as the RAS signaling pathway, including developing IK-595, a novel MEK-RAF inhibitor. Additionally, IK-175, an AHR antagonist, is being developed in collaboration with Bristol Myers Squibb. Ikena aims to utilize their depth of institutional knowledge and breadth of tools to efficiently develop the right drug using the right modality for the right patient. To learn more, visit www.ikenaoncology.com or follow us on Twitter and LinkedIn.

Forward-Looking Statements

This press release contains forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995, as amended, including, without limitation, statements regarding: the timing and advancement of our targeted oncology programs, including the timing of updates; our expectations regarding the therapeutic benefit of our targeted oncology programs; our ability to efficiently discover and develop product candidates; our ability to obtain and maintain regulatory approval of our product candidates; the implementation of our business model, and strategic plans for our business and product candidates. The words "may," "will," "could," "would," "should," "expect," "plan," "anticipate," "intend," "believe," "estimate," "predict," "project," "potential," "continue," "target" and similar expressions are intended to identify forward-looking statements, although not all forward-looking statements contain these identifying words. Any forward-looking statements in this press release are based on management's current expectations and beliefs and are subject to a number of risks, uncertainties and important factors that may cause actual events or results to differ materially from those expressed or implied by any forward-looking statements contained in this press release, including, without limitation, those risks and uncertainties related to the timing and advancement of our targeted oncology programs; our expectations regarding the therapeutic benefit of our targeted oncology programs; expectations regarding our new executive officer; our ability to efficiently discover and develop product candidates; the implementation of our business model, and strategic plans for our business and product candidates, and other factors discussed in the "Risk Factors" section of Ikena's Form 10-Q for the quarter ended September 30, 2022, which is on file with the SEC, as updated by any subsequent SEC filings. We caution you not to place undue reliance on any forward-looking statements, which speak only as of the date they are made. We disclaim any obligation to publicly update or revise any such statements to reflect any change in expectations or in events, conditions or circumstances on which any such statements may be based, or that may affect the likelihood that actual results will differ from those set forth in the forward-looking statements. Any forward-looking statements contained in this press release represent our views only as of the date hereof and should not be relied upon as representing its views as of any subsequent date. We explicitly disclaim any obligation to update any forward-looking statements.

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Exhibit 99.2

ikena™ ONCOLOGY

Corporate Presentation

Fourth Quarter 2022

Developing Biology-Driven Medicines and Expanding the Impact of Targeted Oncology

ikena ONCOLOGY

We develop differentiated therapies for patients in need that target nodes of cancer growth, spread, and therapeutic resistance in the Hippo and RAS onco-signaling network.

Hippo Pathway

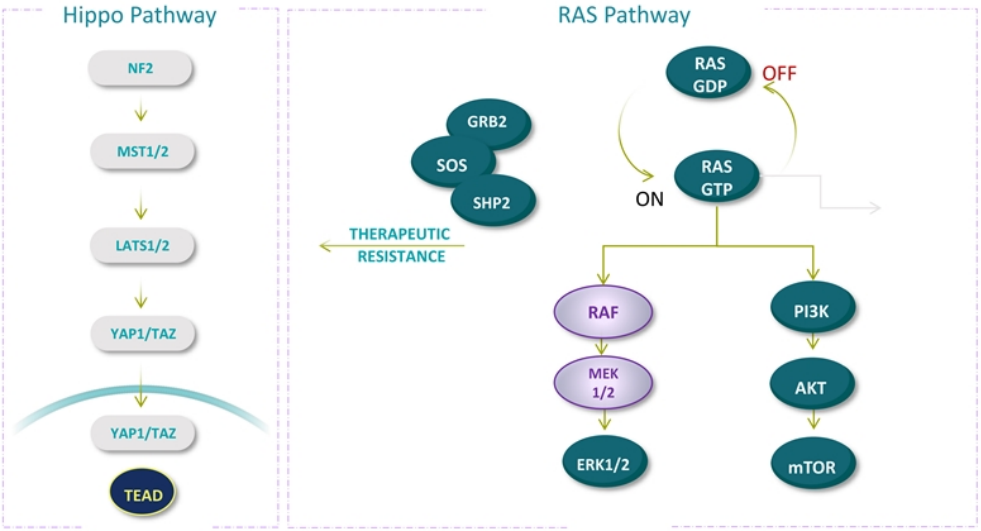
RAS Pathway

- Multiple ongoing clinical trials with **expected data readouts in the next 12 months**
- **Leaders in Hippo pathway** with clinical stage paralog-selective TEAD inhibitor **IK-930**
  - Initial mono-therapy in mesothelioma and EHE in 2023
  - Combination with osimertinib in NSCLS to start in 2023
  - Next generation Hippo candidate in lead optimization
- **Novel MEK/RAF inhibitor IK-595** in IND-enabling studies
  - IND in 2H 2023 with broad potential across RAF and RAS mutant cancers
- BMS partnered program **IK-175** with **clinical activity in bladder cancer**
  - Potential for **$50M in opt-in fees by early 2024**, $450M in milestones plus global royalties
- **>$170M** in cash; Runway into **2025**

Ikena Wholly Owned Pipeline Focused on Targeted Oncology in Hippo-Ras Oncosignaling Network

Connectivity Across RAS & Hippo Onco-signaling Network

Nodes in the RAS network are intricately connected to each other and other orthogonal pathways, including Hippo



Hippo genetically-altered cancers and Hippo activated resistance

RASm cancers – the most common pathway with genetic alteration in cancers – potential benefit from monotherapies and combination therapies

Ikena has deep institutional knowledge and broad capabilities that lay the foundation for discovery programs across the network

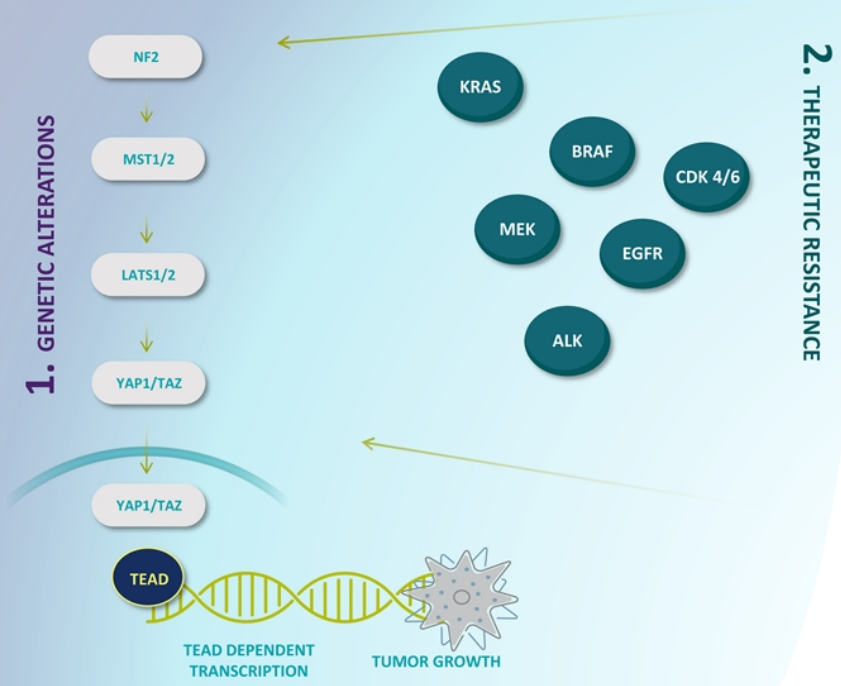
- Deep knowledge and characterization of the interconnected nature of oncogenic nodes
- Proven history of drugging difficult targets
- Leaders in drugging the Hippo pathway
- Advanced capabilities across biomolecular characterization, structural biology, chemistry, and translational medicine

Targeting TEAD & the Hippo Pathway

IK-930

ikena ONCOLOGY

Hippo Pathway Alterations and Activity Trigger TEAD Transcription-Dependent Tumor Growth



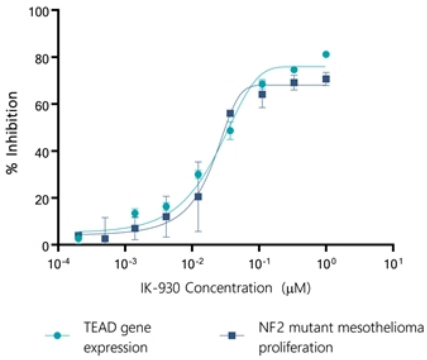
1. **GENETIC ALTERATIONS**: Treat patients with genetic alterations in the Hippo pathway with **IK-930 MONOTHERAPY**. The Hippo pathway is genetically altered in approximately 10% of all human cancers, including 40% of malignant mesothelioma patients and 100% of EHE patients

2. **THERAPEUTIC RESISTANCE**: **COMBINE IK-930** with other targeted therapies. Resistance to multiple targeted therapies and tumor recurrence can be linked to YAP/TEAD activation

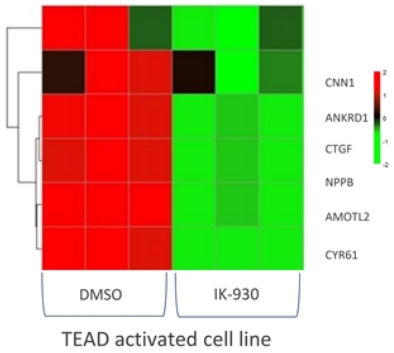
IK-930 is an Oral, Selective, Potent TEAD Inhibitor

IK-930 was well tolerated preclinically while showing significant impact on TEAD dependent gene expression

Potent TEAD Inhibition

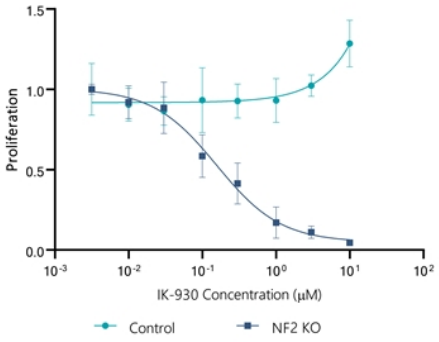


Robust Inhibition TEAD Target Gene Expression



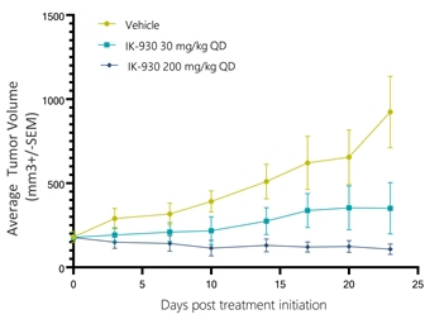
TEAD activated cell line

Selective Activity in Hippo-Mutated Cells

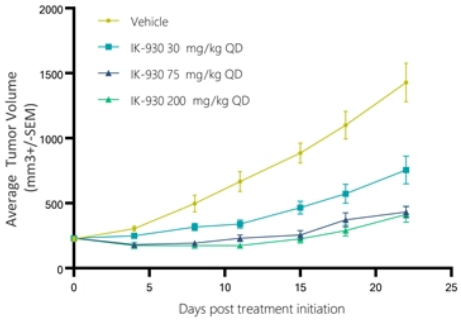


IK-930 Monotherapy Has Potential Across Genetic Mutations in the Hippo Pathway

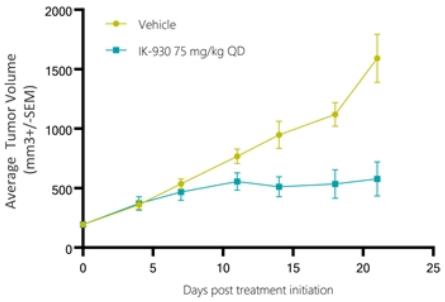
Impact Across Tumor Models for Hippo Pathways Genetic Alterations



NF2 Deficient Mesothelioma Model



LATS1/LATS2 Mutated Mesothelioma Model



YAP1 Amplified HNSCC Model

IK-930 Monotherapy Strategy and Clinical Development Plan; Initial Data Expected in 2H 2023

Growing Monotherapy Opportunity

~125,000 newly diagnosed cancer patients per year in the US with known Hippo pathway mutations and alteration

- **Malignant Mesothelioma:** ~**40%** NF2 loss of function mutations
- **NSCLC: 6%** YAP1 and **29%** TAZ amplification
- **Meningioma:** High frequency of NF2 deficiency; Most common CNS tumor, accounting for **~one-third** of primary CNS tumors
- **Head & Neck Cancers:** Growing body of knowledge on frequent YAP/TAZ amplification and FAT1 (upstream) deficiency
- **Soft Tissue Sarcomas: ~90%** of epithelioid hemangioendothelioma, or **EHE**, have TAZ-CAMTA1 fusions; **10%** of EHE have YAP1-TFE3 fusions

Ongoing Phase 1 Trial
Monotherapy Clinical Development Plan

Dose Escalation → **Dose Expansion**

Currently recruiting; advanced through multiple doses

All comers

Tumors known to have high incidence of Hippo pathway alterations

Cohort 1: NF2 deficient mesothelioma

Cohort 2: NF2 deficient solid tumors; agnostic approach

Cohort 3: Epithelioid hemangioendothelioma (EHE)

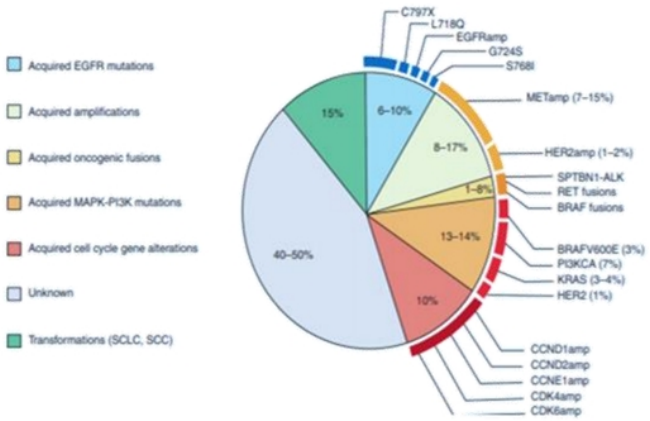
Cohort 4: YAP/TAZ gene fusion solid tumors; agnostic approach

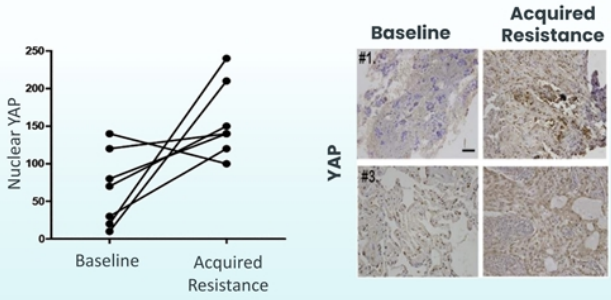
IK-930 Opportunity to Address Emerging Early-Use Osimertinib Resistance

40%+ of patients with Osimertinib resistance have unidentified mechanisms and represent a significant unmet need

Resistance Mechanisms to Osimertinib in EGFRm NSCLC

Leonetti, et al., Br J Cancer, 2019





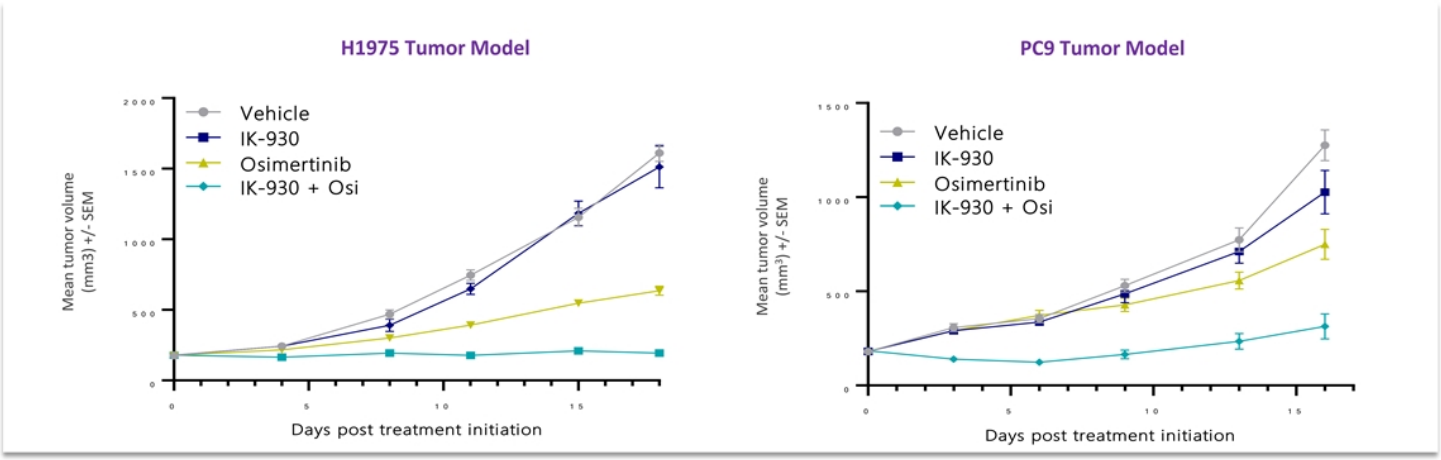
Lee, et al., BBRC, 2016

Opportunity for IK-930 combinations to address acquired Osimertinib resistance

Opportunity to identify subset of patients in whom addition of IK930 combo can delay/prevent the emergence of resistance

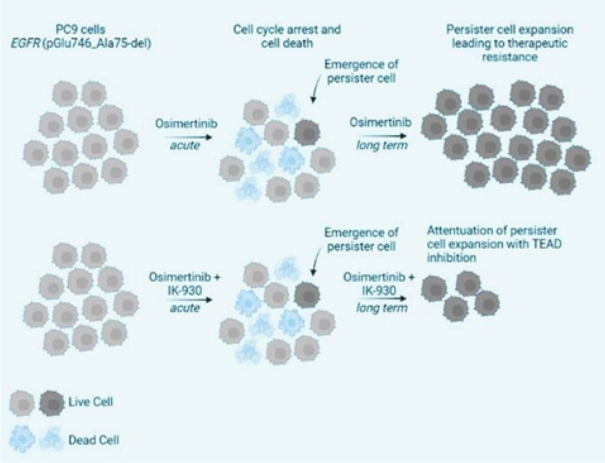
IK-930 Combination with EGFRi shows Improved Anti-tumor Activity

Multiple EGFRm Lung Cancer Models Show Benefit of IK-930-Osi Combination

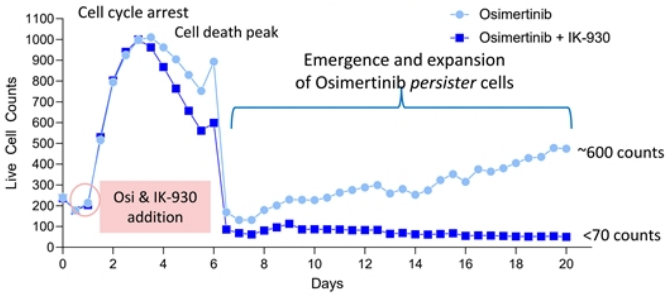


IK-930 Has Pre-Clinical Impact on Refractory *Persister* Cells

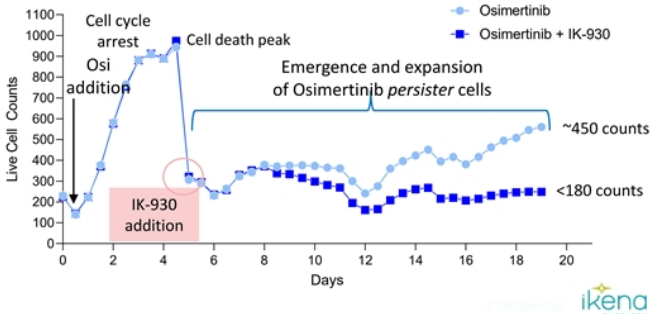
*Potential for IK-930 to **prevent** resistance to EGFR inhibitors and even **reverse** the effect when given after resistance has already emerged*



IK-930 + Osi Combined Prevents Emergence of *Persisters*



IK-930 Addition after *Persister* Emergence Attenuates Expansion



IK-930's Potential to Combat Therapeutic Resistance to Other Targeted Therapies

Combination strategy represents an independent mechanisms and potential opportunity for IK-930

Combination Clinical Development Plan
First Cohort to Initiate in 2023

Dose Escalation → **Dose Expansion**

All comers

Biomarker defined tumors treated with targeted therapies (multiple combination groups)

Cohort 1: IK-930 + osimertinib in EGFRm resistant NSCLC

Cohort 2: IK-930 + MEKi in solid tumors, including KRASm tumors

Additional Cohorts: Determine from emerging data

Addressing a Leading Limitation of Targeted Therapy - Primary and Secondary Therapeutic Resistance

Resistance to multiple targeted therapies and tumor recurrence can be linked to **YAP/TEAD activation**

Overcoming resistance mechanisms and escape could **deepen and prolong responses and address *de novo* resistance**, allowing more patients to respond to target therapies overall

"…underlying mechanisms through which malignant tumor cells acquire or develop resistance to anti-cancer treatment. The Hippo signaling pathway appears to play an important role in this process."
Zeng et al. Cancers 2021

"The biggest hurdle to targeted cancer therapy is the inevitable emergence of drug resistance."
Lim, et al. Journal of Hematology & Oncology 2019

"Despite [targeted oncology's] immense progress, advanced cancer is ultimately lethal for most patients due to treatment resistance."
Aldea, et al. Cancer Discovery 2021

Ikena Leads the Field in Targeting the Hippo Pathway

- **IK-930**: First-in-class, paralog-selective TEAD inhibitor
  - Ongoing phase 1 clinical trial currently in dose escalation
    - Monotherapy cohorts in NF2 mutant mesothelioma and EHE (100% YAP/TAZ)
    - Multiple planned combination cohorts combating therapeutic resistance
      - Data shows potential to prevent and reverse resistance to EGFR inhibitors
  - **Additional data on advantages of paralog-selectivity and combination approach in 1H 2023**
  - **Initial clinical data expected in 2H 2023**
- **Next-gen Hippo program** in lead optimization

MEK-RAF Complex Inhibitor

IK-595

The RAS Pathway is Highly Implicated in Cancer

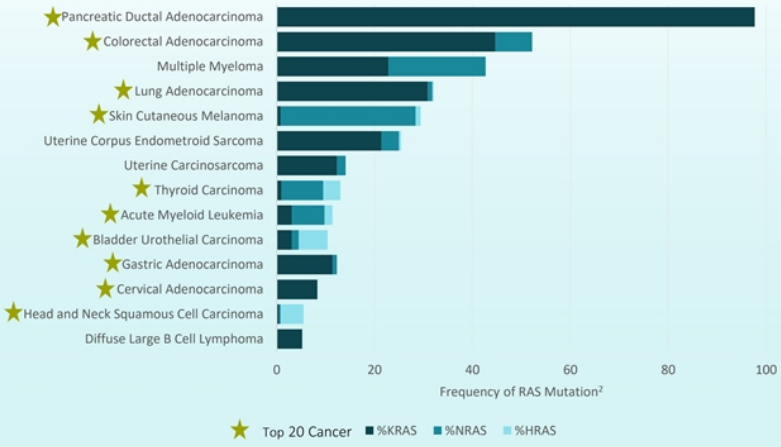
Targeting within the pathway could be impactful for a massive and diverse population

The **RAS pathway** is potentially implicated in **over half a million new cancer diagnoses each year** in the US alone[1]

New approaches in targeting the pathway need to consider key learnings

- Approved inhibitors can paradoxically activate MEK/ERK signaling
- CRAF is implicated as a key signaling bypass mechanism for targeted therapies, and has kinase independent activity that drives RAS mutant cancers

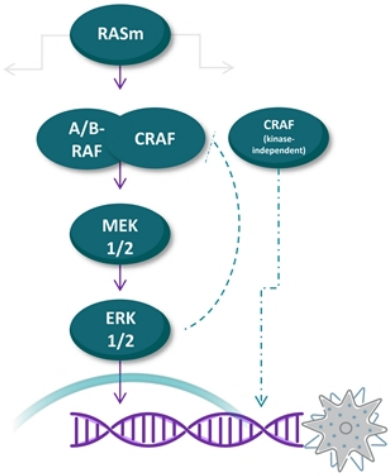
10 of the 20 most common cancers worldwide are associated with RAS pathway mutations



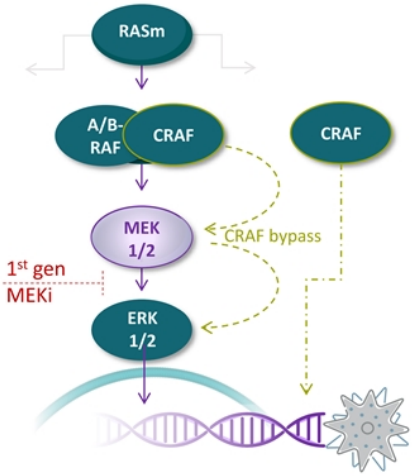
[1]ACS and https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3457779/
[2]Cox. Nature Reviews Drug Discovery (2014); World Cancer Research Fund International

First Generation MEK Inhibitors: Insufficient Targeting Leads to Limited Activity

MEK's role in driving ERK-mediated tumor growth



First gen MEK inhibitors missed the potential of CRAF to bypass MEK and trigger ERK



Approved MEK inhibitors like trametinib and binimetinib block MEK kinase activity

Feedback in the pathway however triggers CRAF activation

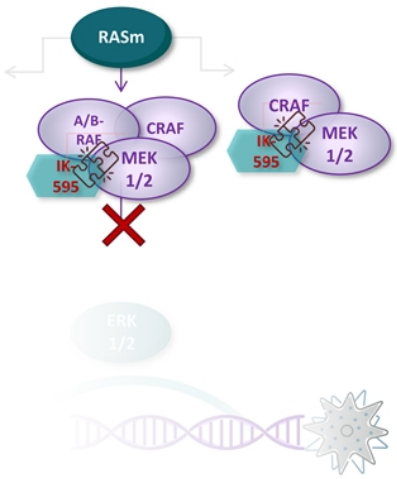
Cancer's survival mechanism utilizes CRAF to reactivate the pathway and bypass inhibition

Additionally, approved inhibitors miss blocking kinase-independent CRAF function that can trigger tumor growth

Leads to incomplete pathway inhibition

IK-595: A Best-in-Class Dual MEK-RAF Complex Inhibitor

IK-595 traps MEK & RAF in an inactive complex to prevent CRAF bypass and kinase-independent CRAF function



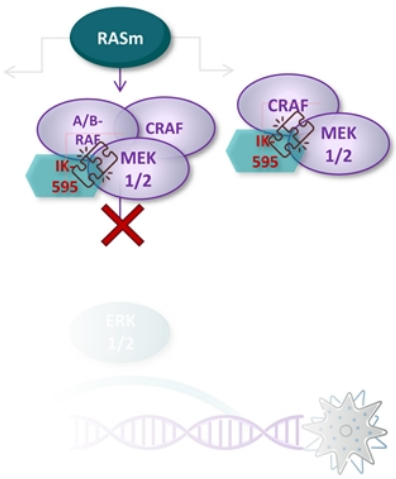
Key IK-595 Advantages

IK-595 is designed to and has shown preclinical evidence of superior profile than first generation and in-development MEK inhibitors

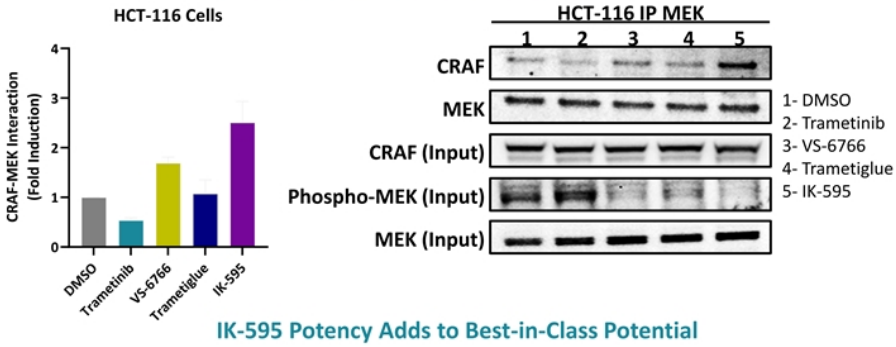
- ✓ Inhibit MEK mediated ERK1/2 phosphorylation
- ✓ Prevent MEK phosphorylation by RAF
- ✓ Alleviate therapeutic resistance through CRAF mediated bypass and pathway reactivation
- ✓ Block CRAF kinase independent activities
- ✓ Optimized PK profile to target IC90 plasma concentrations widening the therapeutic window

Key Advantages of IK-595 Including Robust Stabilization of MEK-CRAF Complex

IK-595 traps RAF and MEK in a stable, inactive complex providing advantages in blocking both bypass in the pathway and kinase-independent CRAF function



IK-595 Stabilizes CRAF-MEK Complex

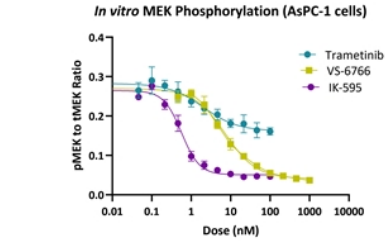


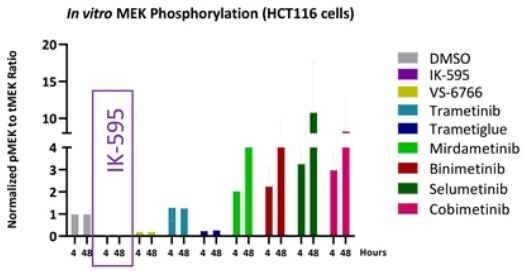
IK-595 Potency Adds to Best-in-Class Potential

Assay	Cellular pERK IC_{50}	Biochem uMEK IC_{50}	Cellular pMEK 4h / 48h IC_{50}	Proliferation AsPC-1 CTG IC_{50}
IK-595	0.1 nM	3 nM	0.6/1 nM	1.3 nM

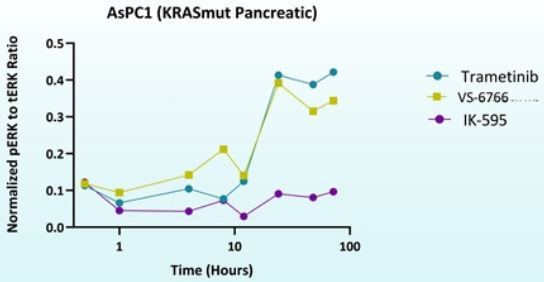
IK-595 Leads to Significantly More Durable Pathway Suppression than Other MEK Inhibitors

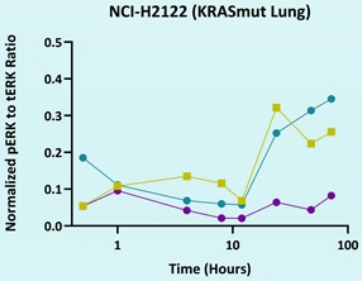
IK-595 Potently Inhibits MEK Phosphorylation In Vitro

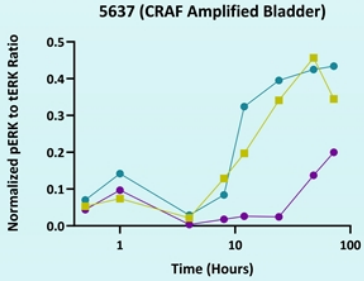




IK-595 Demonstrates Robust and Prolonged pERK Inhibition in Multiple Cell Lines



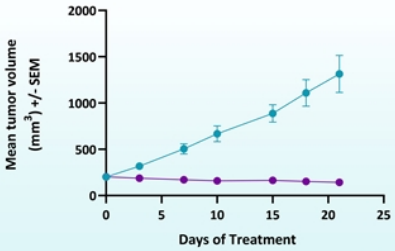




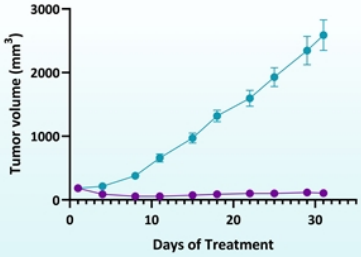
Robust Preclinical Efficacy in RAS and RAF Cancers with Great Sensitivity in CRAF Dependent Models

Antitumor Activity Across Models at Tolerated IK-595 Doses

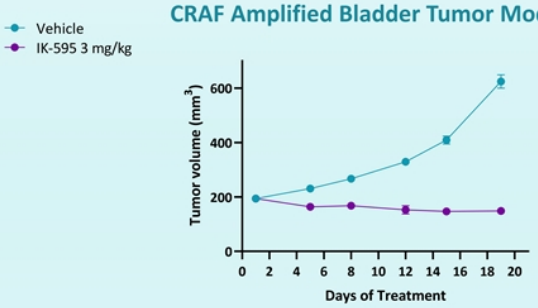
KRAS G12D Pancreatic Model



KRAS G12C Lung Tumor Model

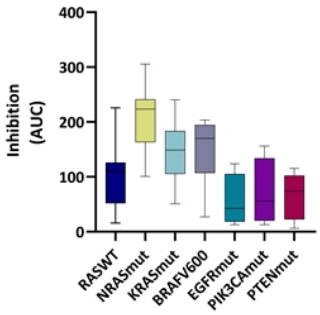


CRAF Amplified Bladder Tumor Model



Efficacy achieved with both continuous and intermittent dosing of IK-595

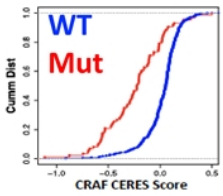
IK-595 Sensitivity



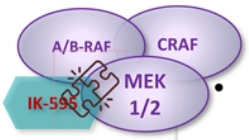
IK-595 has greatest sensitivity in NRAS and KRAS mutant cell lines which are dependent on CRAF

NRAS and KRAS – CRAF CERES Score

Jones, 4th RAS-Targeted Drug Development Summit 2022



IK-595: Best-in-Class Next Generation MEK-RAF Complex Inhibitor



- Novel, best-in-class inhibitor that traps MEK and RAF in an inactive complex for more complete inhibition of the pathway
- Durable, potent inhibition of the pathway demonstrated through multiple data sets
- Mechanisms prevents CRAF bypass and kinase-independent CRAF function
- Preclinical efficacy in multiple disease models
- Difficult to treat CRAF-dependent tumors show high sensitivity to IK-595 in cell lines
- **IND planned for 2H 2023**

Targeting AHR to Counter Immunosuppressive TME

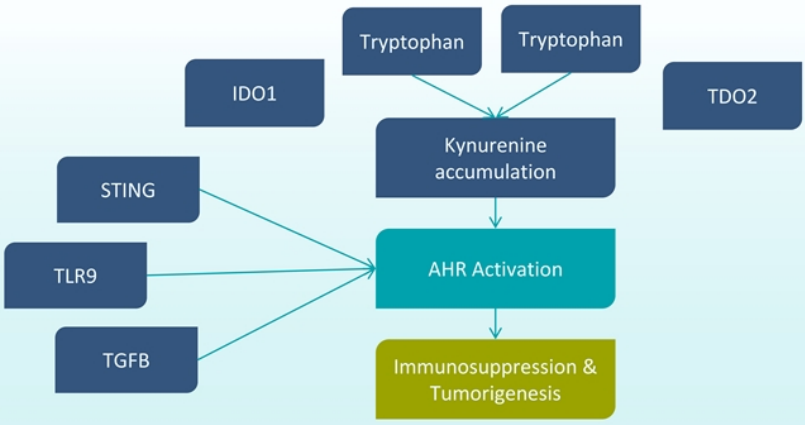
IK-175

Bristol Myers Squibb™

ikena ONCOLOGY

AHR's Role in Immune Signaling & Identifying Bladder Cancer as Key Population

Activated AHR can prevent immune recognition of cancer through both the innate and adaptive immune systems



AHR modulates activity in both the innate and adaptive immune systems

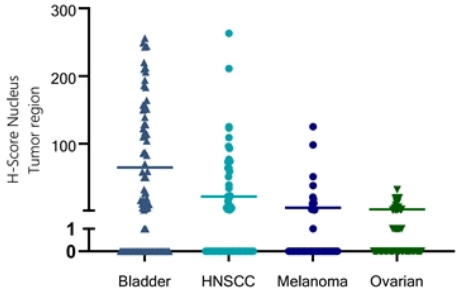
Novel Assays to Optimize Indication Selection

Proprietary transcriptional signature

Gene amplification

Proprietary IHC

Tumor Microarray Result



IK-175 Ph1 Study Ongoing in Urothelial Carcinoma Patients

Patients have exhausted SOC and progressed on CPIs

Clinical data presented at SITC 2022 including dose escalation (all-comers), and both mono and combo stage 1 expansion cohorts in urothelial carcinoma

- 43 total patients; 40 evaluable for anti-tumor activity
- 20 dose escalation
- 20 dose expansion (10 mono, 10 combo)

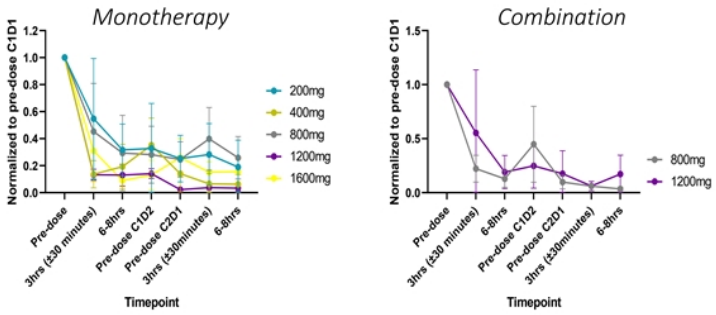
Pharmacodynamics seen at all doses

No DLTs observed

IK-175 was well tolerated with a predictable and manageable safety profile

Encouraging anti-tumor activity and duration of response seen in IK-175 nivolumab combination expansion cohort

Pharmacodynamics at All Doses



Last-line, Heavily Pre-treated Patients

Demographics of Evaluable Urothelial Carcinoma Patients in Initial Clinical Analysis

	Monotherapy (n=10)	Combination (n=10)
Prior lines of anti-cancer therapy		
1-3	2	4
4-10	8	6
ADC experienced	9	6

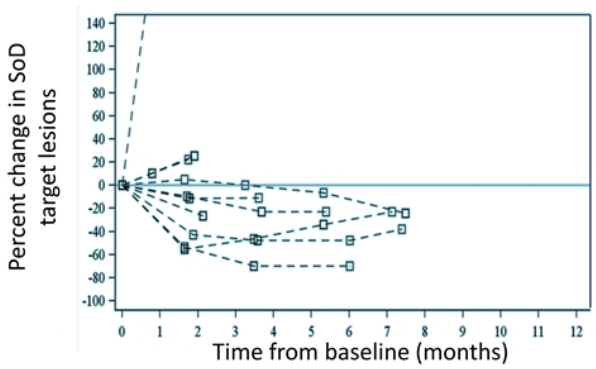
Initial Clinical Data in Urothelial Carcinoma Demonstrated Encouraging Anti-Tumor Activity

Clear evidence of monotherapy activity contributing to combination responses
Heavily pretreated patients exhausted all options -- failed checkpoints and have had up to 10 prior lines of therapy
Mono partial response ongoing over 15 months; Combo partial responses ongoing over 5 months

Initial Clinical Data from Stage 1 of Expansion Cohorts

	Monotherapy (n=10)	Combination (n=10)
Best overall response		
Confirmed partial response	1 (10%)	2 (20%)
Stable Disease	1 (10%)	2 (20%)
Progressive disease	6 (60%)	6 (60%)
ORR, n(%)	1 (10%)	2 (20%)
DCR, n(%)	2 (20%)	4 (40%)

Stage 1 of Combination Cohort in Urothelial Carcinoma Showed 40% DCR with Encouraging Anti-Tumor Activity



Combo result represent meaningful potential for patient population with significant and ongoing DoR

Currently recruiting in stage 2 of both mono and combo cohorts

Ikena Wholly Owned Pipeline Focused on Targeted Oncology in Hippo-Ras Oncosignaling Network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