

November 2021

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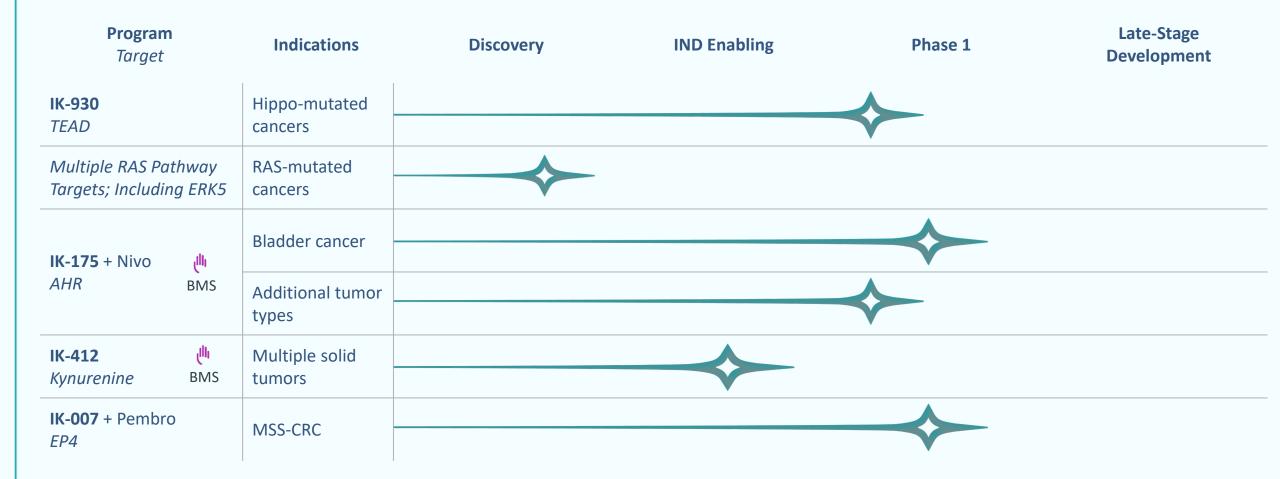


Developing Biology-Driven Medicines and Expanding the Impact of Targeted Oncology for Patients

Ikena Mission	By the Numbers 3 programs in clinical development or clinic-ready	Current Focus Targeted Oncology	
Patient-driven drug development targeting oncogenic drivers and pathways of therapeutic resistance			
	2 programs partnered with oncology partner of choice, BMS	Hippo Pathway	RAS Pathway
	Multiple targeted oncology programs in discovery across 2 key pathways		
Using known and novel biomarkers and approaches for	60 diverse, experienced team members		
targeted therapy development and patient identification	\$245M in cash; Runway through 2023	Immune-signaling in the tumor- microenvironment	



Pipeline of Biomarker-Defined Therapies Designed to Improve Patient Outcomes





Targeting TEAD & the Hippo Pathway

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IK-930

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Patients with Hippo-Driven Cancers Could Benefit from IK-930 Monotherapy

Hippo landscape is developing; the identification of additional related indications continues

NF2 **GENETIC ALTERATIONS MST1/2** LATS1/2 YAP1/TAZ YAP1/TAZ **TEAD**

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

• Malignant Mesothelioma: ~40% have 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

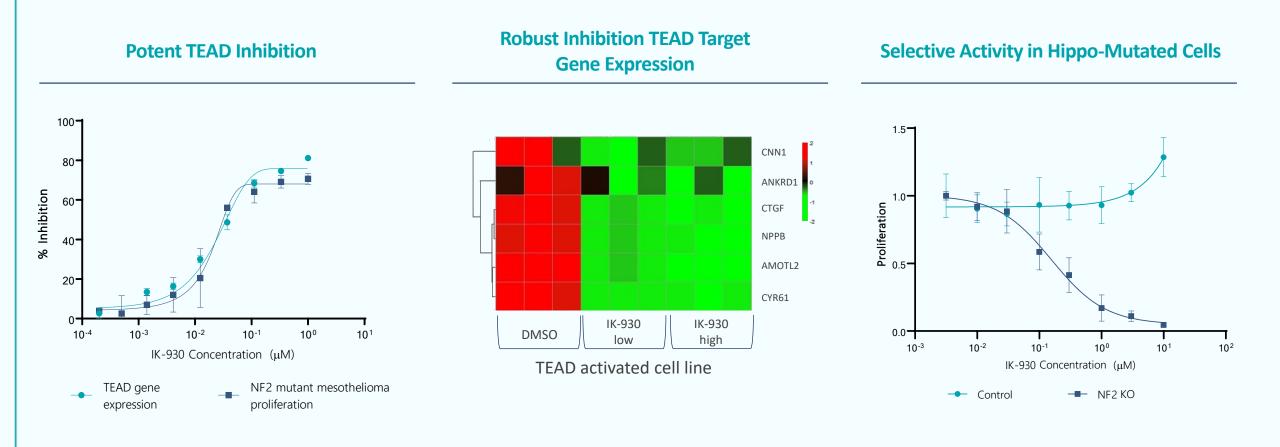


• Soft Tissue Sarcomas: ~90% of epithelioid hemangioendothelioma, or EHE, have TAZ-CAMTA1 fusions; 10% of EHE have YAP1-TFE3 fusions



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

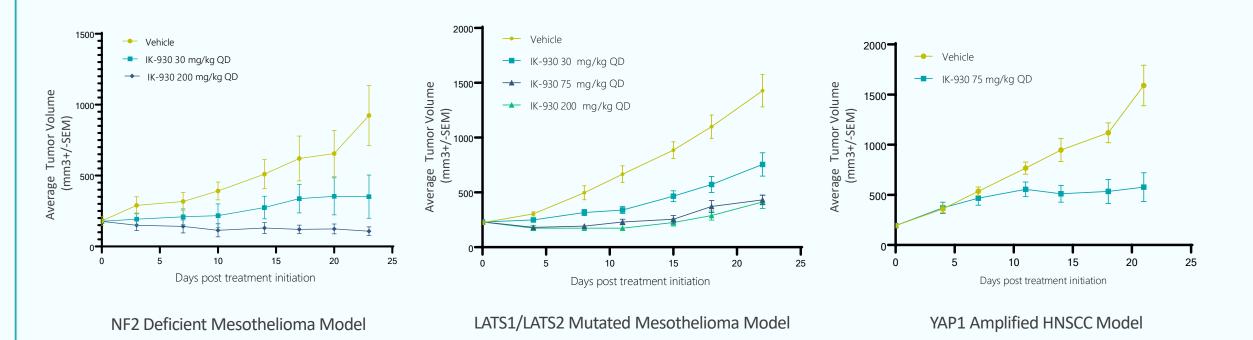
IK-930 was well tolerated preclinically while showing significant impact on TEAD dependent gene expression





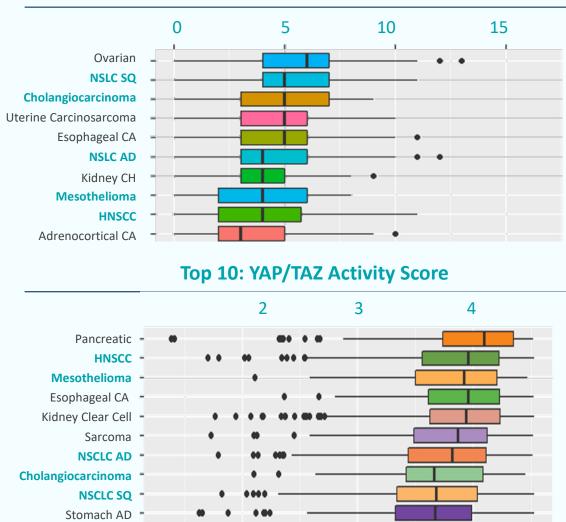
IK-930 Monotherapy Has Potential Across Genetic Mutations in the Hippo Pathway





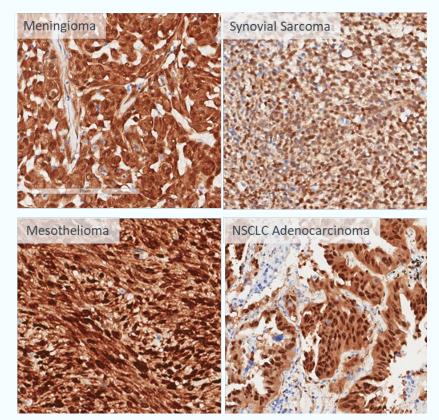


Robust Translational Data to Drive Indication Selection



Top 10: Hippo Alterations

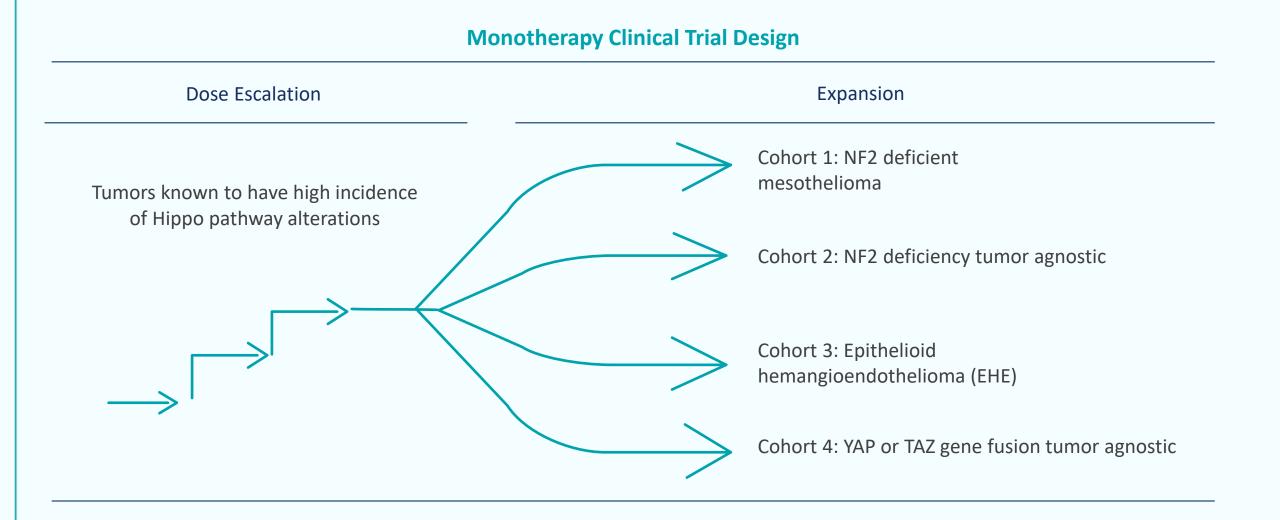
YAP/TAZ Nuclear Localization



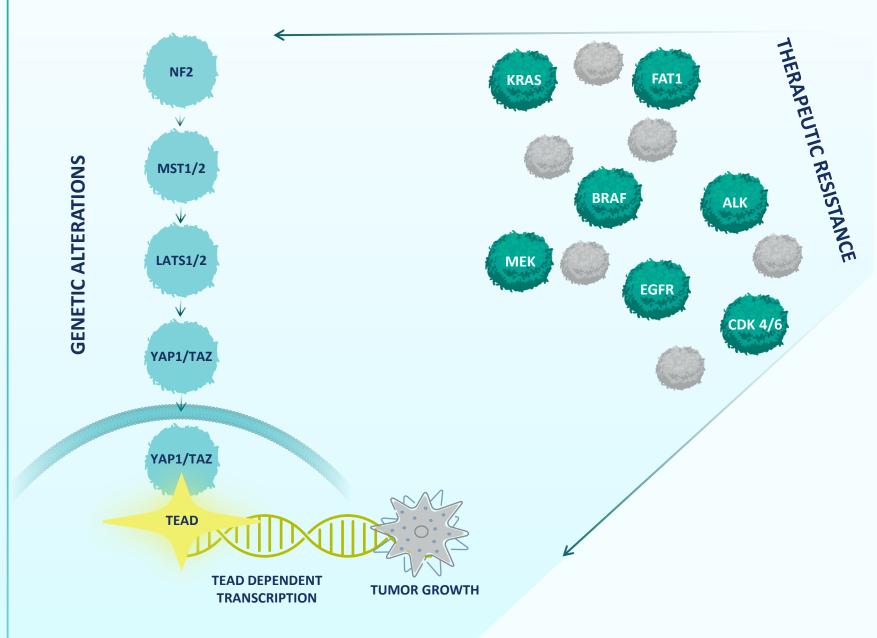
High YAP1 nuclear protein expression indicative of pathway activation in select indications



IK-930 First-in-Human Trial Monotherapy Targeting Hippo-Driven Cancers



Hippo Pathway Engagement in Therapeutic Resistance and Tumor Escape



- Combining IK-930 with other targeted therapies has the potential to combat therapeutic resistance
- Resistance to multiple targeted therapies and tumor recurrence can be linked to YAP/TEAD activation
- Overcoming resistance mechanisms and escape could not only deepen and prolong responses but could address de novo resistance, allowing more patients to respond to target therapies overall

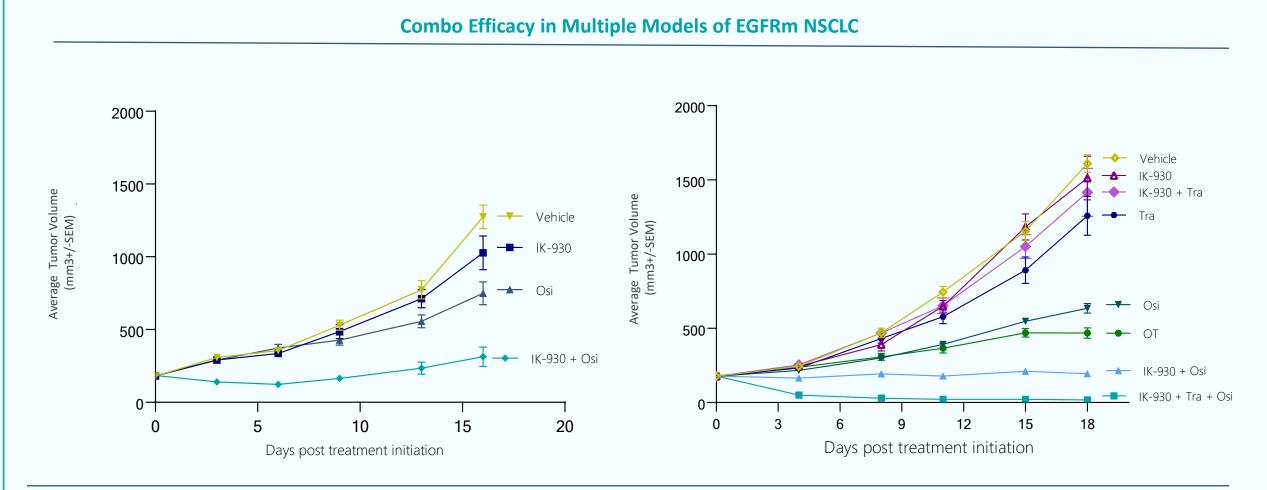
Targeted-Therapy Treated EGFRm NSCLC Shows Potential for IK-930 Combo Benefit

IK-930 Combo with MEKi & EGFRi in EGFRm NSCLC **EGFR Inhibitor Promotes YAP1 Nuclear Localization Model Shows Significant Increase in Targeted Apoptosis** DMSO 2500-OT+9301µM 2000-OT+930 0.3 µM Normalized Apoptosis OT+930 0.1 µM 1500-Osimertinib 1000-Osi + Tra Osi 50 nM 500-Tra 30 nM 930 1 µM --- Vehicle 0-12 24 36 0 48 Tra: Trametinib Hours Osi: Osimertinib OT: Osi + Tra Combo **IF:** α-**YAP1**

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IK-930 Combo with EGFRi & MEKi Shows Preclinical Efficacy in EGFRm Cancer Models

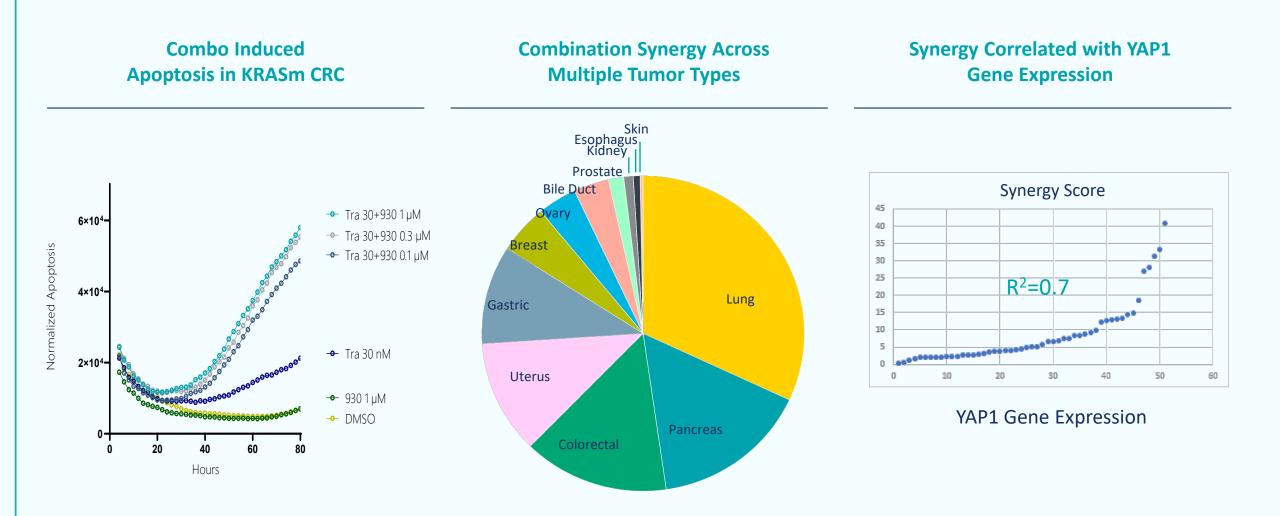
Combo could have potential for first-line approach in EGFRm cancers



Triplet combo demonstrated complete responses in mice



IK-930 Combos Could Potentially Expand the KRASm Cancer Populations that Could Benefit from Targeted Therapies

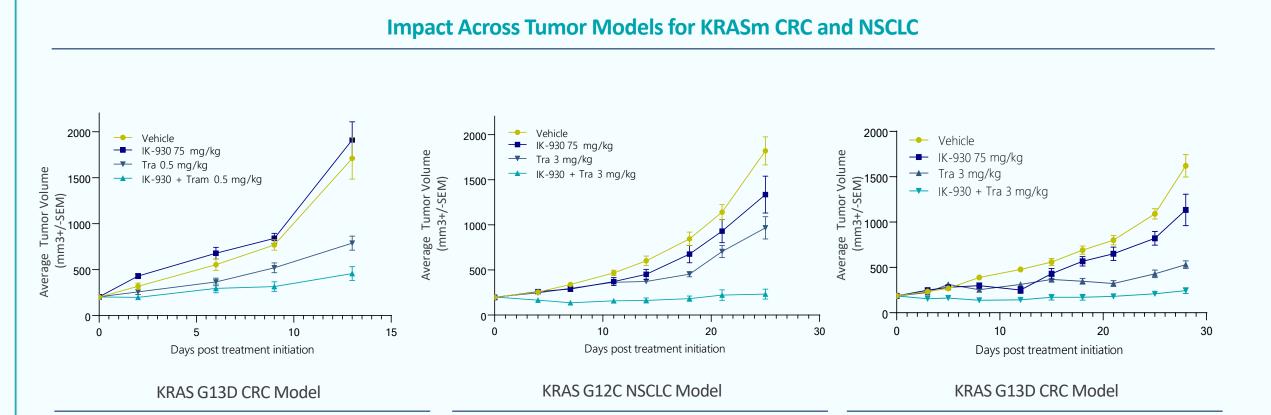


Cell lines showing synergy across multiple KRAS mutations, including G12C, G12V, G13D and others



RAS Mutated Cancer Show Potential for IK-930 Combo Benefit Across Tumor Types

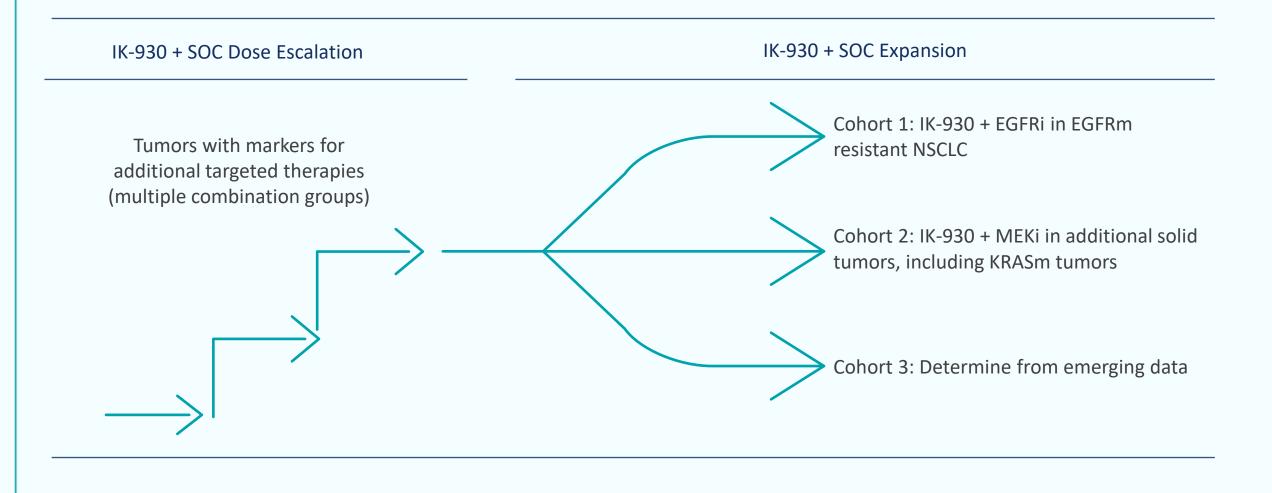
Potential for IK-930 combo benefit across tumor types



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IK-930 Combinations with Other Targeted Therapies in First-in-Human Trial

Plans to explore multiple combinations to address therapeutic resistance





Targeting AHR to Counter Immunosuppressive TME

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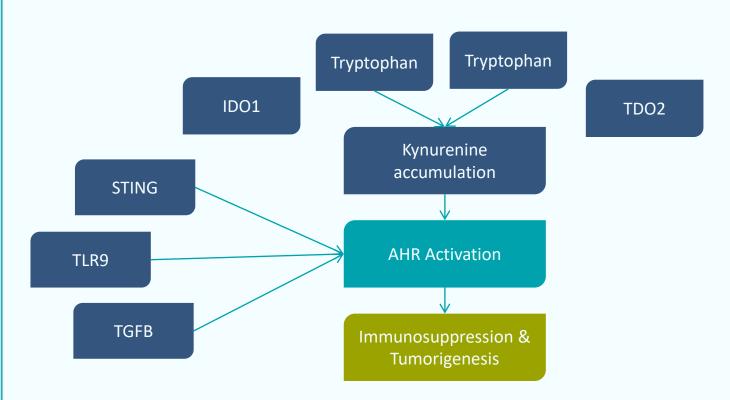
IK-175



AHR's Role in Immune Signaling

Selecting indications that can potentially benefit with from AHR inhibition with IK-175

Aryl Hydrocarbon Receptor (AHR) Signaling



- Activated AHR prevents immune recognition of a range of cancers by modulating both innate and adaptive immunity
- AHR activity has been linked to activity in multiple cancer types, including:
 - Bladder cancers
 - Head & neck cancer
 - Melanoma
 - Ovarian
 - Acute myeloid leukemia
 - Malignant gliomas
 - Resistant prostate cancer



Identifying Bladder Cancer as Potential Patient Population to Benefit from IK-175

Novel assays to determine indications and prospectively select patients with nuclear-AHR

Novel Assays to Optimize Indication Selection

AHR

AHR in Bladder Cancer & the Unrealized Unmet Need

- Poor prognosis of patients with bladder cancer is associated with a high AHR transcript profiling score
- Patient with metastatic diseases have a five-year survival rate of just 5%

IK-175 Preclinical Data Supports Clinical Trial Approach

IK-175 shows anti-tumor activity as a monotherapy and enhances impact in combination with anti-PD-1 in a murine model

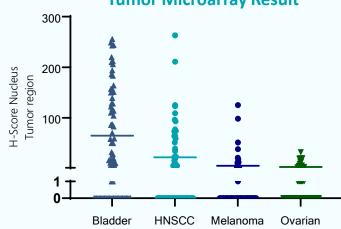
Proprietary transcriptional signature

AHR

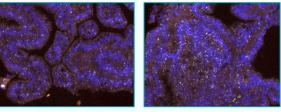


ture Tumor Microarray Result

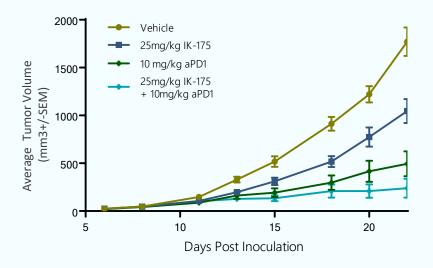
Gene



AHR transcripts in bladder cancer sample (white)

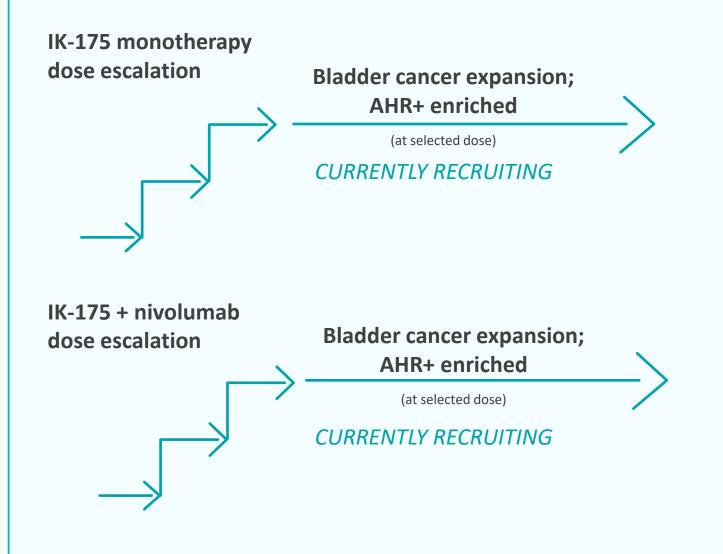


Pre-selecting patients with nuclear-AHR in ongoing clinical trial





IK-175 Ph1 Study Ongoing & Utilizing Novel Assay to Prospectively Select Patients



- Recently expanded bladder cancer monotherapy cohort and completed doseescalation in the combination cohort
- No dose limiting toxicities, or DLTs, to date
- Maximum tolerated dose not observed to date
- AHR+ patient selection utilizing novel, Ikenadeveloped IHC assay



Integrated Targeted Oncology Strategy

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Pipeline of Biomarker-Defined Therapies Designed to Improve Patient Outcomes

Efficiently investing capital to advance programs with high-impact value-building potential

