Animal Model Workshop

The Path of Progress: Lessons from RCC Animal Models

Payal Kapur
James Brugarolas

Kidney Cancer Program
UT Southwestern Medical Center

@Jbrugarolas
@PayalKapur5
@KCPUTSW
brugarolaslab.com
kcp.utsw.edu

September 11-13, 2025

Session 2

Driving advances with kidney cancer mouse models

BIOLOGY:

Vignettes 1-7: Tumor suppressor gene cooperativity

- VHL sufficiency for ccRCC development
- BAP1 and PBRM1 drive tumor grade
- BAP1 and PBRM1 as determinants of metastatic latency and tropism
- BAP1 and PBRM1 shape the TME
- Transformation of PBRM1-deficient tumors (TSC1 & SETD2)

Vignette 8: Cell of origin

TRANSLATION:

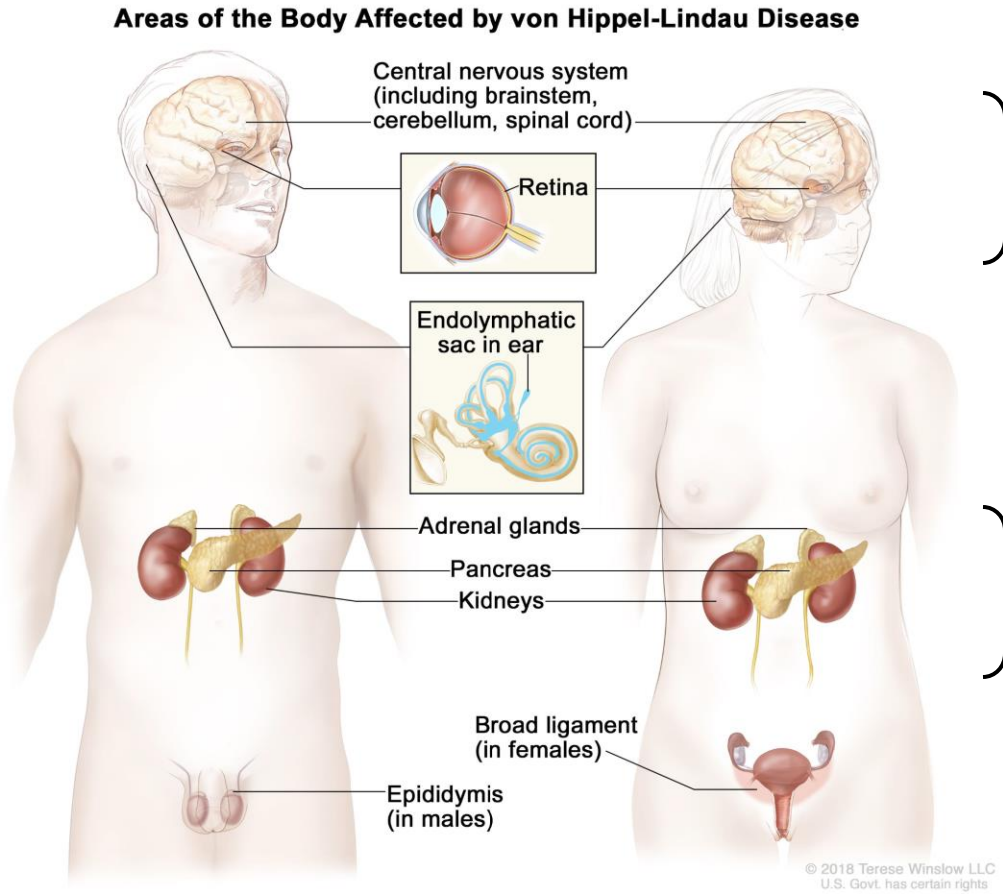
Vignette 9: Targeting HIF-2

- Predictive biomarkers (HIF-2α PET tracer, paraneoplastic syndromes)
- Resistance mechanisms
- Second generation tumor targeted siRNA therapeutic

Vignette 10: Rapalogs

- Resistance mechanisms

Germline mutations in *VHL* predispose to ccRCC and other tumors



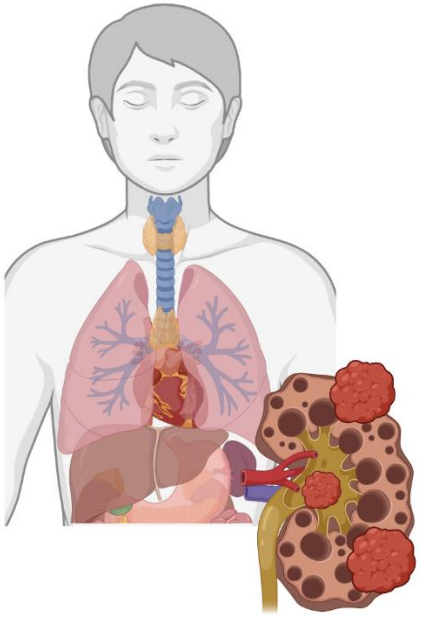
Hemangioblastomas

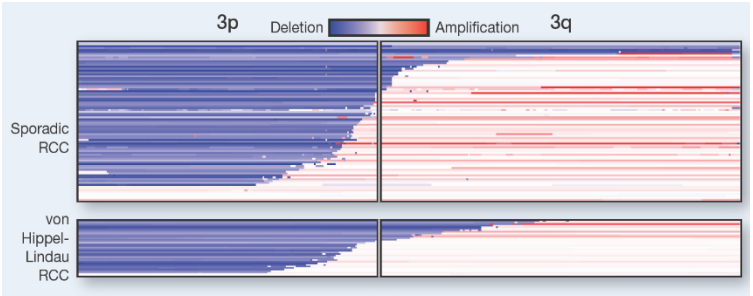
Pancreatic neuroendocrine tumors
Renal cell carcinoma (ccRCC)

https://nci-media.cancer.gov/pdq/media/images/793775.jpg

Vignette 1. Unlike most 2-hit TSG, *VHL* is insufficient for tumorigenesis

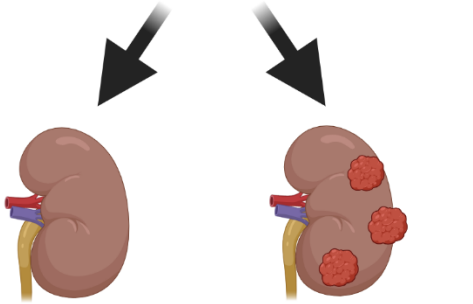
***VHL+/-* Human**





***Vhl+/-* mouse**

(Haase et al, *PNAS* 2001)



***Vhl-/-* kidney**

***Vhl-/-; Bap1+/-* kidney**

Vhl

Chr 6

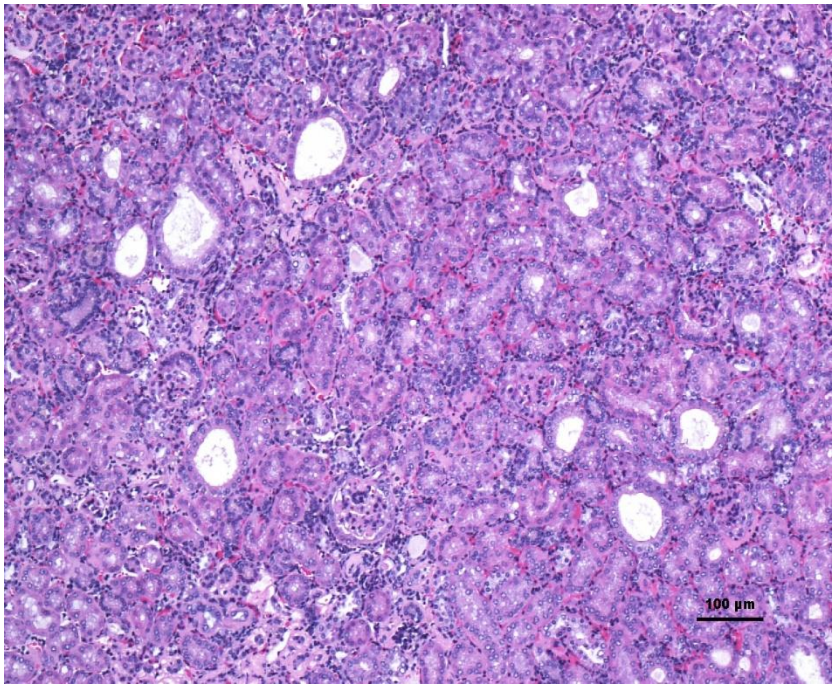
Bap1
Pbrm1

Chr 14

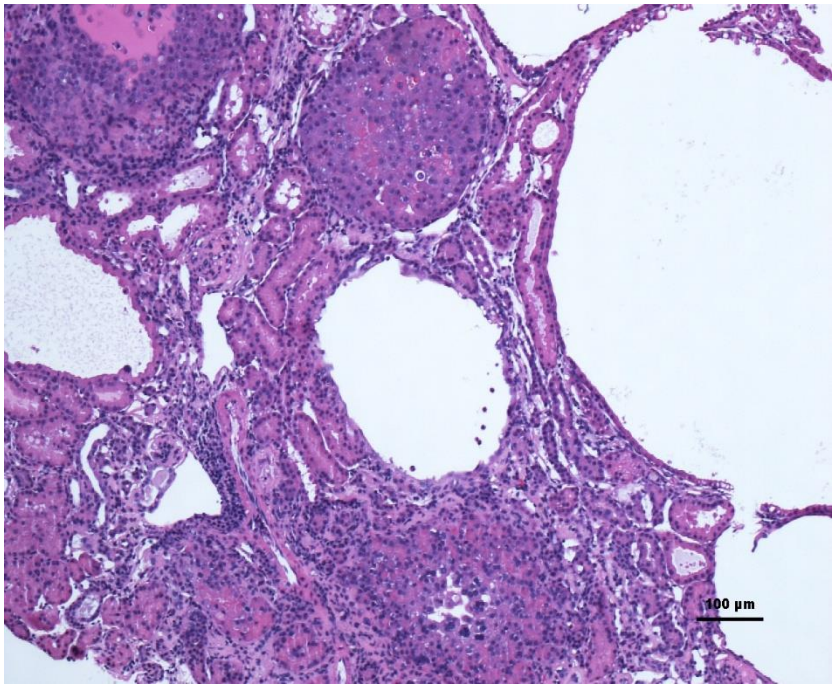
Pena-Llopis et al, *Nat Genet* (2012)
Wang et al, *PNAS* (2014)

Co-inactivation of *Vhl* & *Bap1* in murine nephron progenitor cells causes ccRCC

Six2-Cre;Vhl$^{F/F}$



Six2-Cre;Vhl$^{F/F}$;Bap1$^{F/+}$

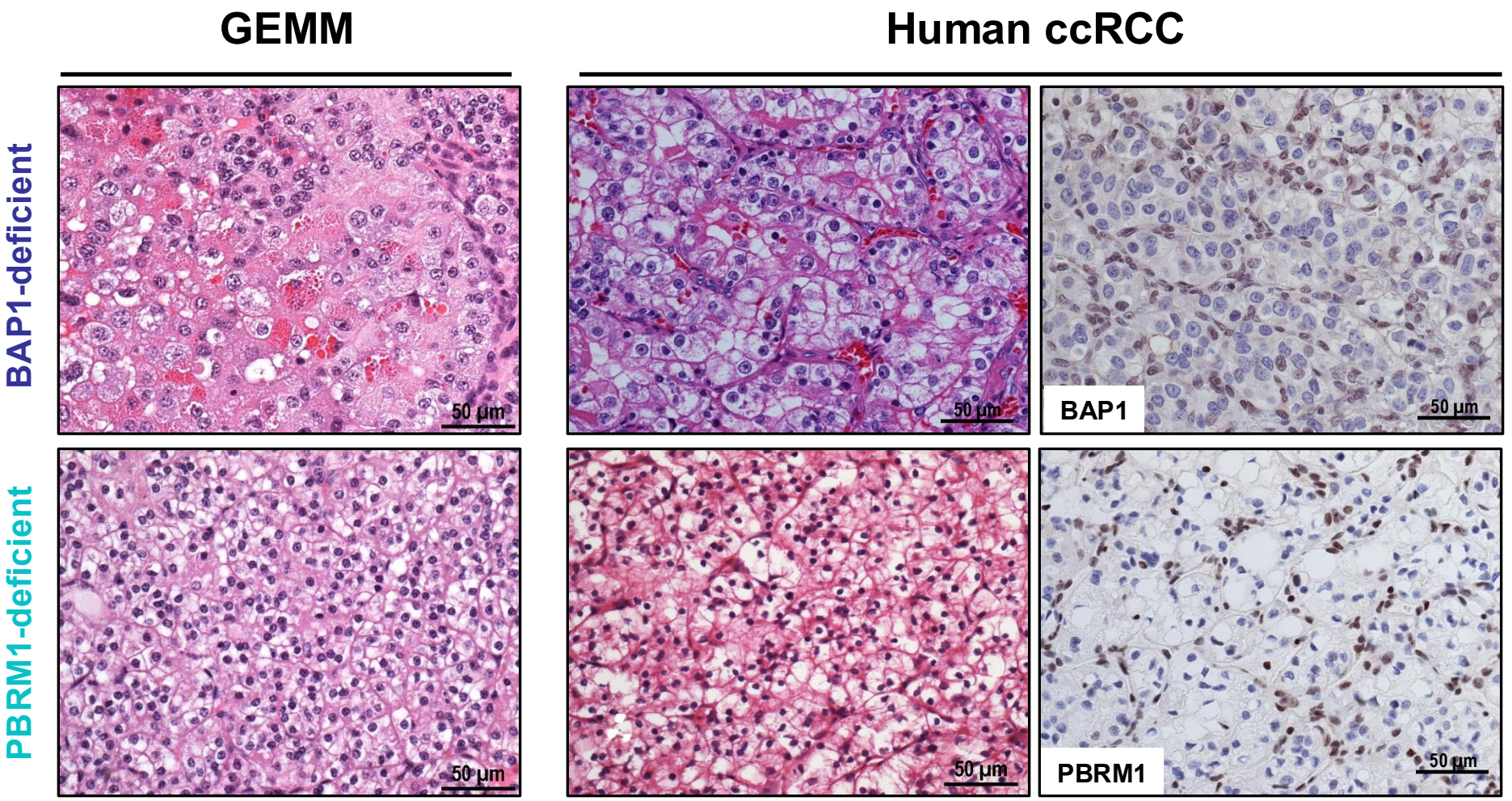


Wang et al, *PNAS* (2014)

Vignette 2. BAP1 and PBRM1 are not simply indicators but drivers of tumor grade and aggressiveness

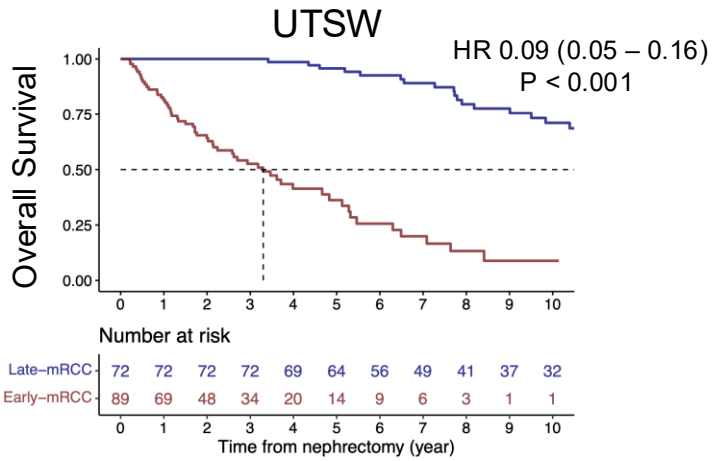
Pena-Llopis et al, *Nat Genet* (2012)
Kapur et al, *Lancet Oncology* (2013)
Pena-Llopis et al, *Cancer Res* (2013)
Wang et al, *PNAS* (2014)
Joseph et al, *J Urol* (2016)
Gu et al, *Cancer Discovery* (2017)
Wang et al, *Cancer Discovery* (2018)
Singla et al, *JCI Insight* (2020)

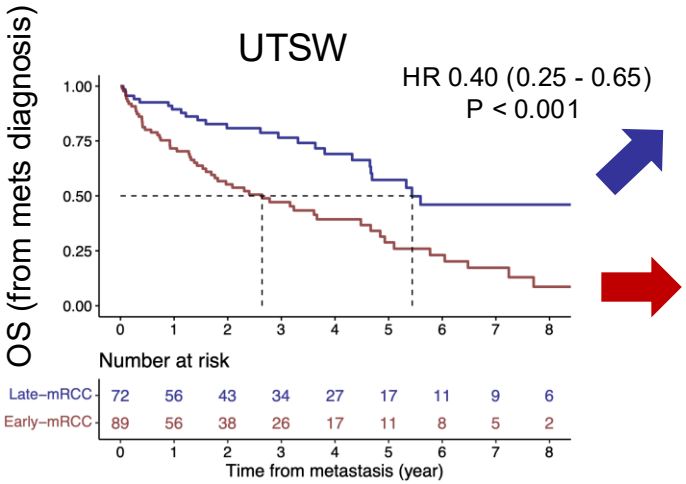
GEM tumors resemble human subtypes



Gu et al, *Cancer Discovery* (2017)

Vignette 3. BAP1 and PBRM1 differentially account for early (≤1 yr) and late (>3 yrs) metastasizing tumors

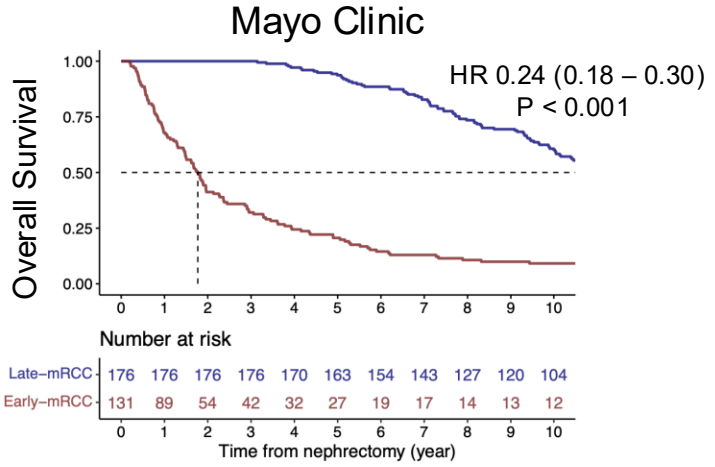


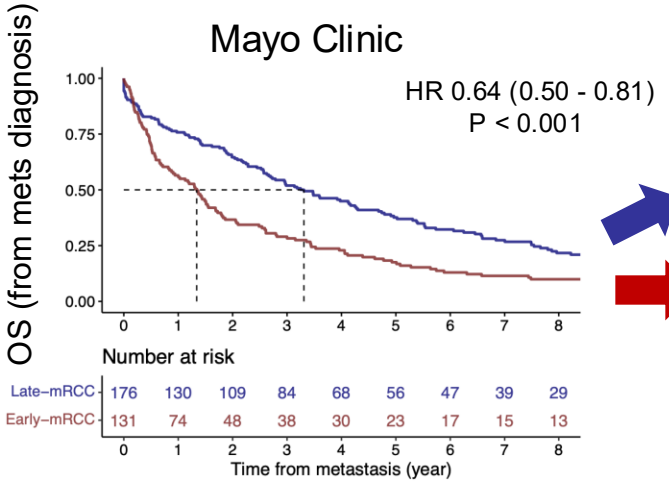


58% PBRM1-
8% BAP1-

47% PBRM1-
33% BAP1-

p=0.05





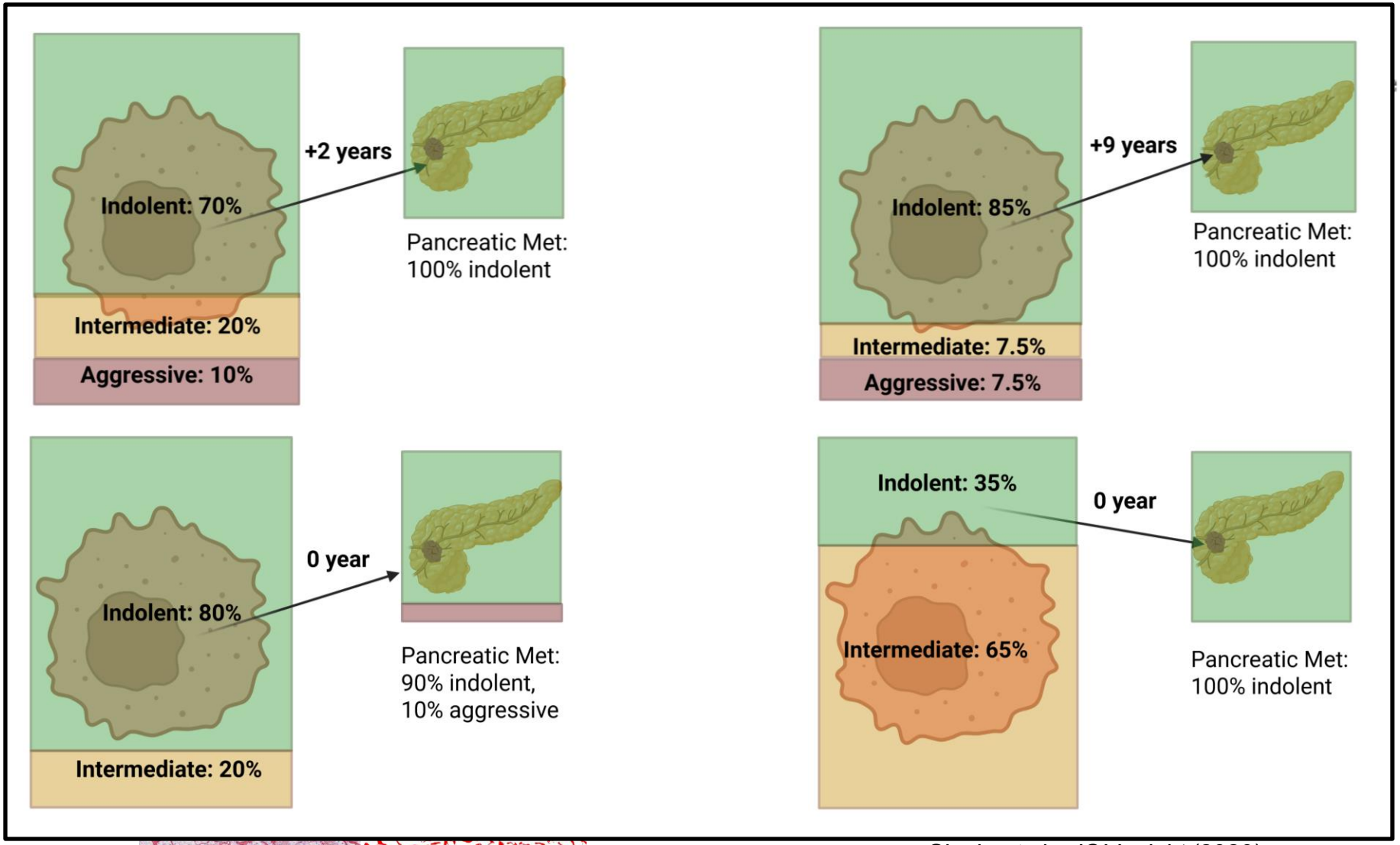
59% PBRM1-
13% BAP1-

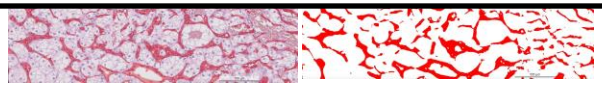
48% PBRM1-
28% BAP1-

p=0.007

Kapur et al., *JCI Insight* (2024)

Vignette 4. PBRM1 loss likely contributes to improved survival of tumors with pancreatic tropism and response to anti-angiogenic drugs





H&E Overlay DL Angioscore

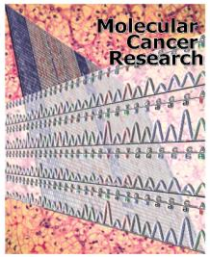
Singla et al., *JCI Insight* (2020)
Kapur et al., *Kidney Cancer Journal* (2020)
Duarte et al., *EClinicalMedicine* (2023)
Xu and Kapur et al., submitted

Vignette 5. mTORC1 activation drives progression of Vhl/Pbrm1 tumors

Gu et al., *Cancer Discovery* 2017

Vignette 5. mTORC1 activation drives progression of Vhl/Pbrm1 tumors

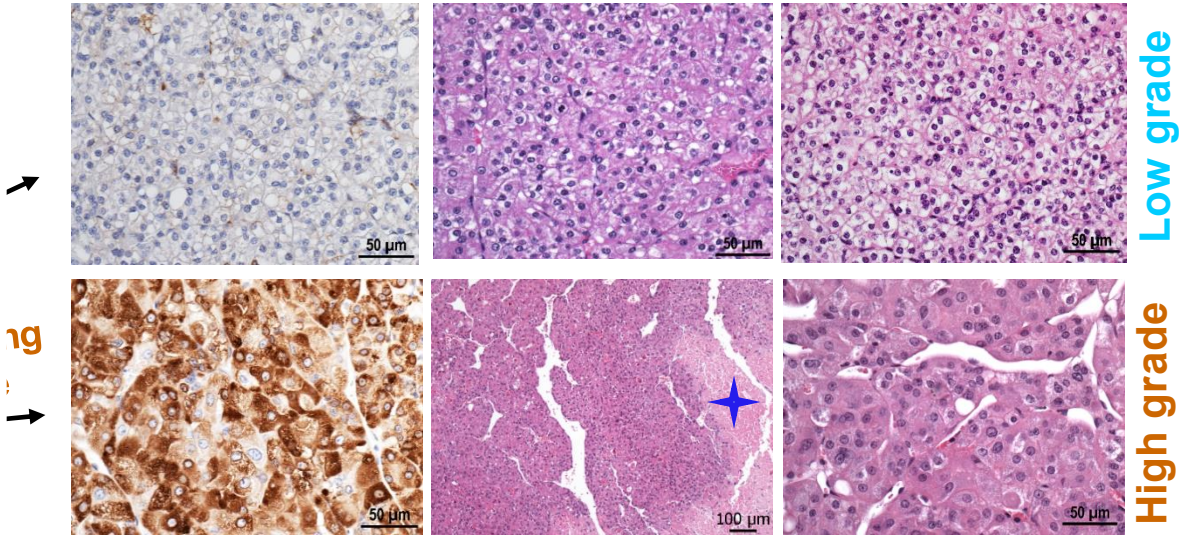
Kucejova et al., *Mol Can Res* 2011



ID	Gene	cDNA	Protein
78	*REDD1*	c.550delC	Fs
9	*TSC1*	c.2459dupA	Fs
3246	*TSC1*	c.1546C > T	p.Q516X
4562	*TSC1*	IVS211-2A > T[a]	Sp
5533	*TSC1*	c.1342C > T[b]	p.P448S
4363	*PTEN*	c.494G > T	p.G165V

TSC1 mutations in ccRCC 4/77 (5%)

phospho-S6



Gu et al., *Cancer Discovery* 2017

Vignette 6. Another route to increasing PBRM1- tumor aggressiveness: loss of SETD2

Review

Cancer Research

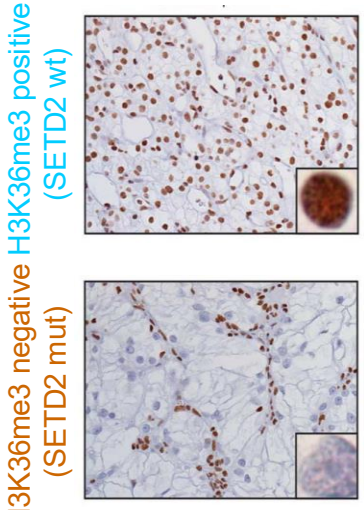
Cooperation and Antagonism among Cancer Genes: The Renal Cancer Paradigm

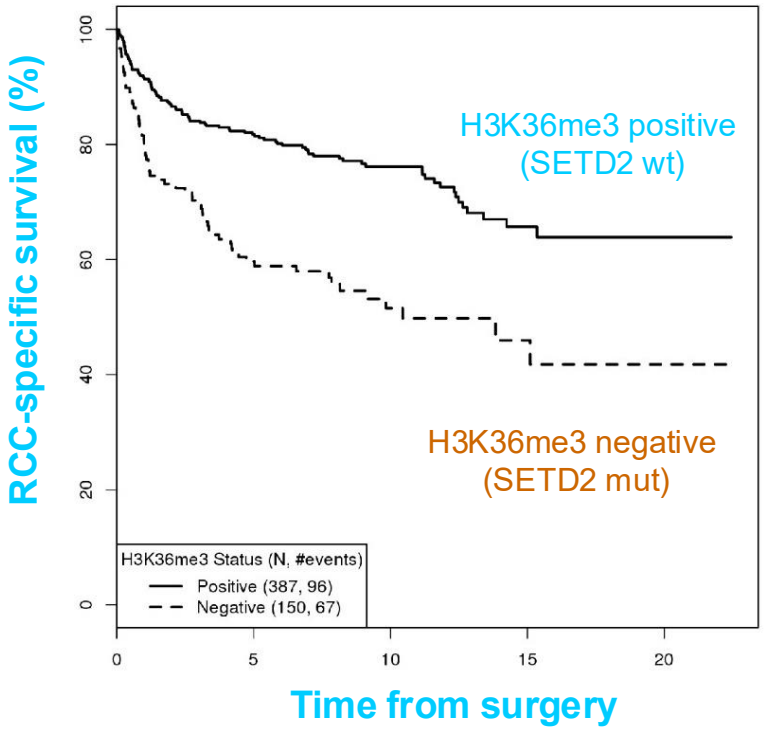
Samuel Peña-Llopis[1,2,3], Alana Christie[3], Xian-Jin Xie[3], and James Brugarolas[1,2,3]

Study	*n*	*PBRM1*	*SETD2*	*SETD2/ PBRM1*	Expected double mutants	*P*	OR (95% CI)
Sanger Institute (16, 18)	348	111	7	8	5 (2–8)	0.16	2.3 (0.8–6.5)
Guo et al. (17)	98	18	1	3	1 (0–2)	**0.03**	12.7 (1.2 –129)
Hakimi et al. (29)	185	48	8	6	4 (2–7)	0.24	1.9 (0.6–5.8)
TCGA	293	90	17	16	12 (7–14)	0.13	1.8 (0.9–3.7)
Total	**924**	**267**	**33**	**33**	**21 (17–25)**	**0.003**	**2.1 (1.3–3.5)**

Loss of histone H3 lysine 36 trimethylation is associated with an increased risk of renal cell carcinoma-specific death

Thai H Ho[1,2,14], Payal Kapur[3,4,14], Richard W Joseph[5], Daniel J Serie[6], Jeanette E Eckel-Passow[7], Pan Tong[8], Jing Wang[8], Erik P Castle[9], Melissa L Stanton[10], John C Cheville[11], Eric Jonasch[12], James Brugarolas[4,13] and Alexander S Parker[6]

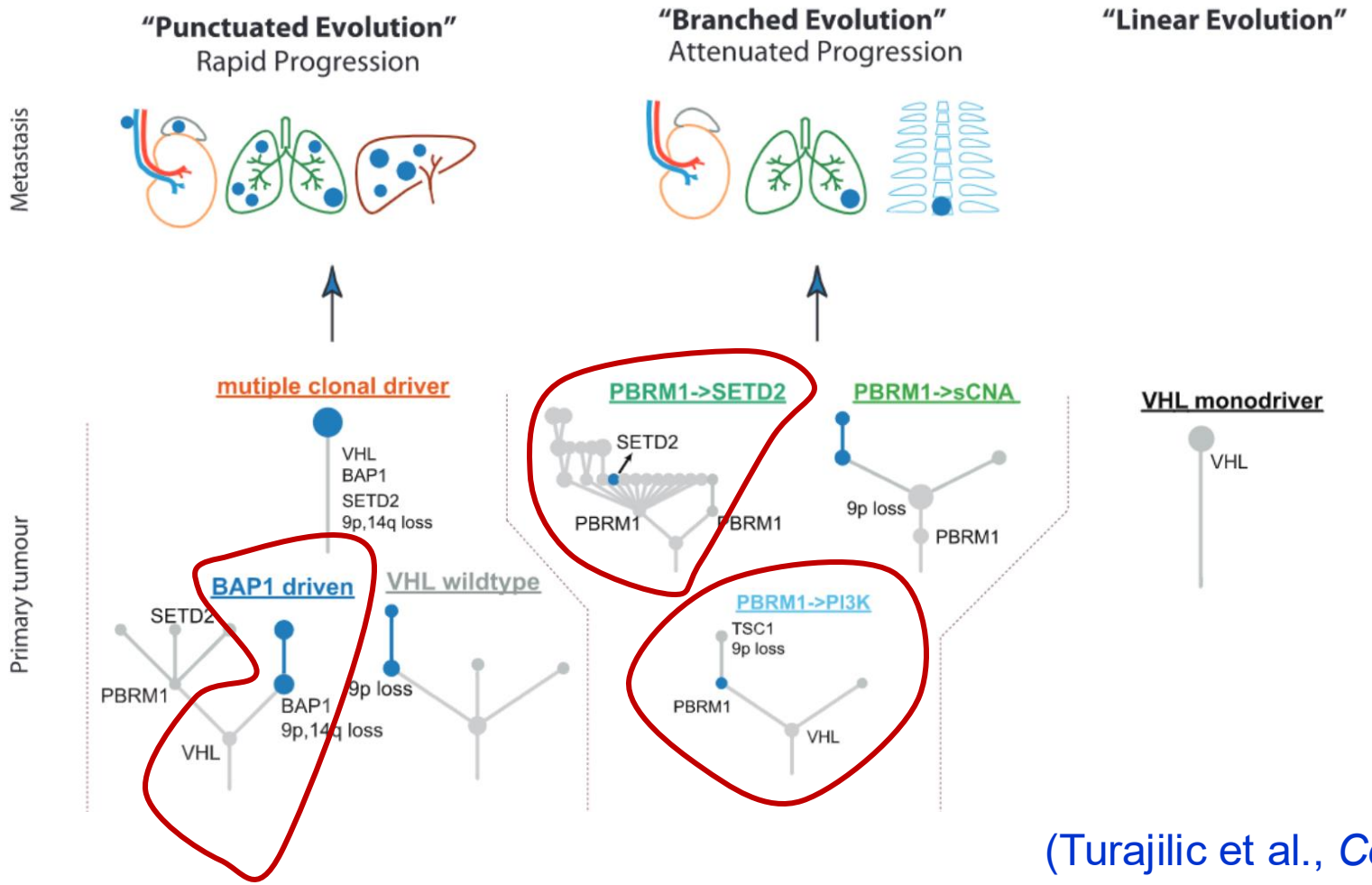




Pena-Llopis et al., *Can Res* 2013
Ho et al., *Mod Path* 2016

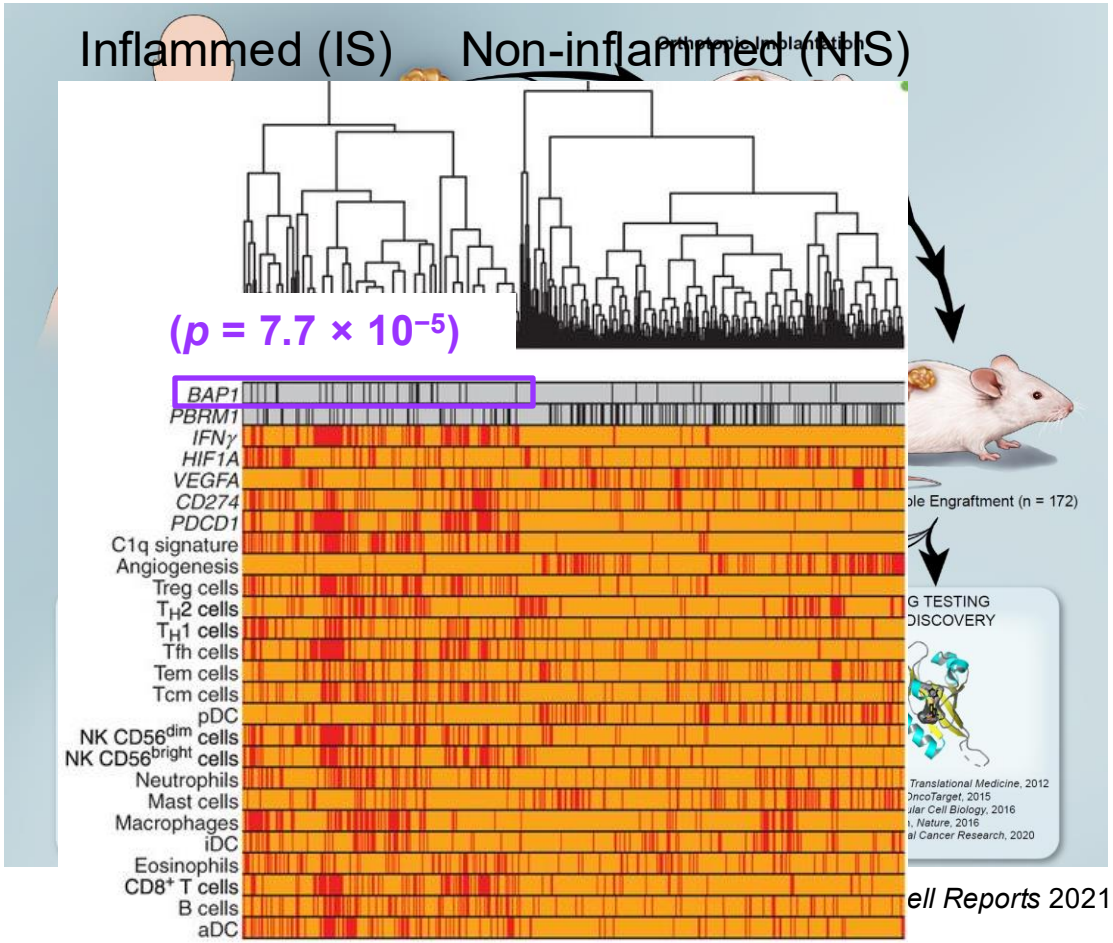
Tracking Cancer Evolution Reveals Constrained Routes to Metastases: TRACERx Renal

Samra Turajlic,[1,2,20] Hang Xu,[1,20] Kevin Litchfield,[1,20] Andrew Rowan,[1,20] Tim Chambers,[1,20] Jose I. Lopez,[3,20] David Nicol,[4,20] Tim O'Brien,[5,20] James Larkin,[2,20] Stuart Horswell,[6] Mark Stares,[1,2] Lewis Au,[2] Mariam Jamal-Hanjani,[7] Ben Challacombe,[5] Ashish Chandra,[8] Steve Hazell,[9] Claudia Eichler-Jonsson,[1] Aspasia Soultati,[10] Simon Chowdhury,[10] Sarah Rudman,[10] Joanna Lynch,[2] Archana Fernando,[5] Gordon Stamp,[11] Emma Nye,[11] Faiz Jabbar,[1] Lavinia Spain,[2] Sharanpreet Lall,[10] Rosa Guarch,[12] Mary Falzon,[13] Ian Proctor,[13] Lisa Pickering,[2] Martin Gore,[2] Thomas B.K. Watkins,[1] Sophia Ward,[1,7] Aengus Stewart,[3] Renzo DiNatale,[14] Maria F. Becerra,[14] Ed Reznik,[15] James J. Hsieh,[16] Todd A. Richmond,[17] George F. Mayhew,[17] Samantha M. Hill,[18] Catherine D. McNally,[18] Carol Jones,[18] Heidi Rosenbaum,[17] Stacey Stanislaw,[18] Daniel L. Burgess,[17] Nelson R. Alexander,[18] Charles Swanton,[1,7,19,21,*] PEACE, and the TRACERx Renal Consortium

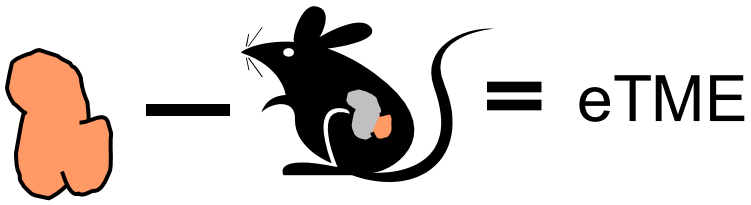


(Turajilic et al., *Cell* 2018)

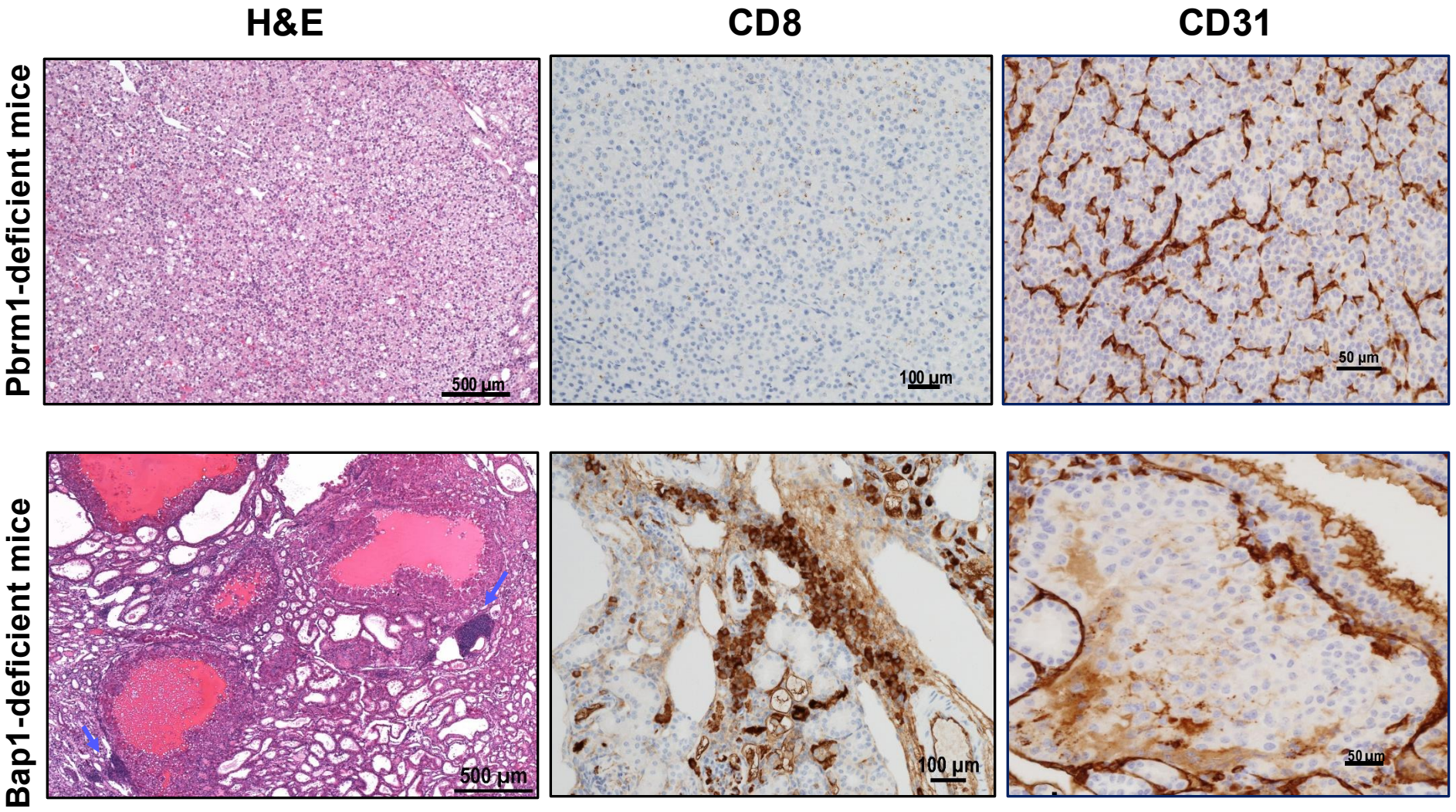
Vignette 7. BAP1 loss induces inflamation with systemic manifestations (thrombocytosis & neutrophilia), which likely accounts for their prognostic value



ell Reports 2021



As shown in GEM, differences in TME are associated with genotype in humans

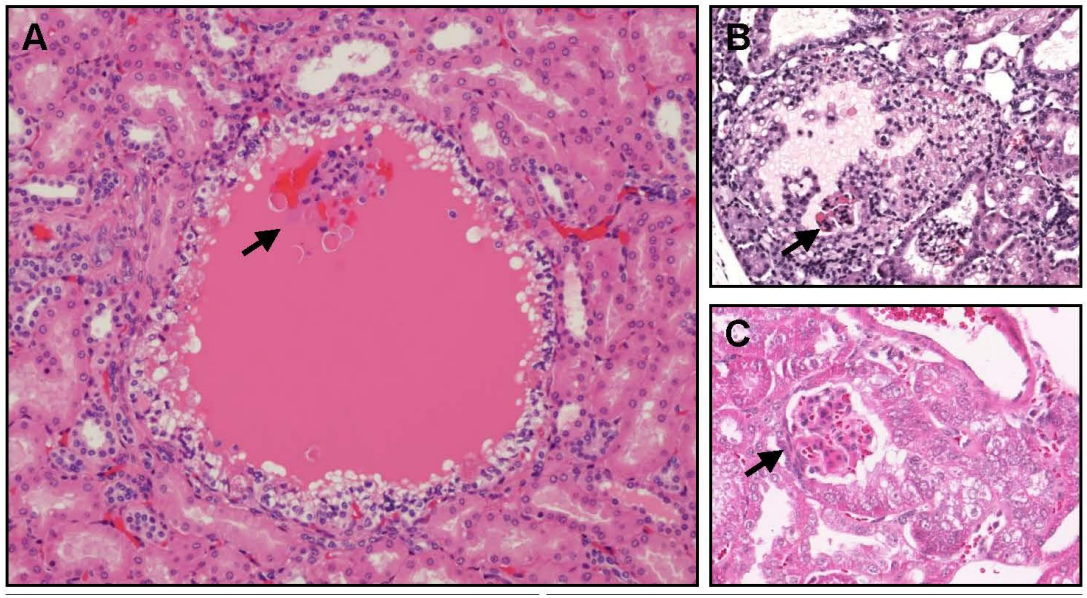


UT Southwestern Medical Center
Kidney Cancer Program

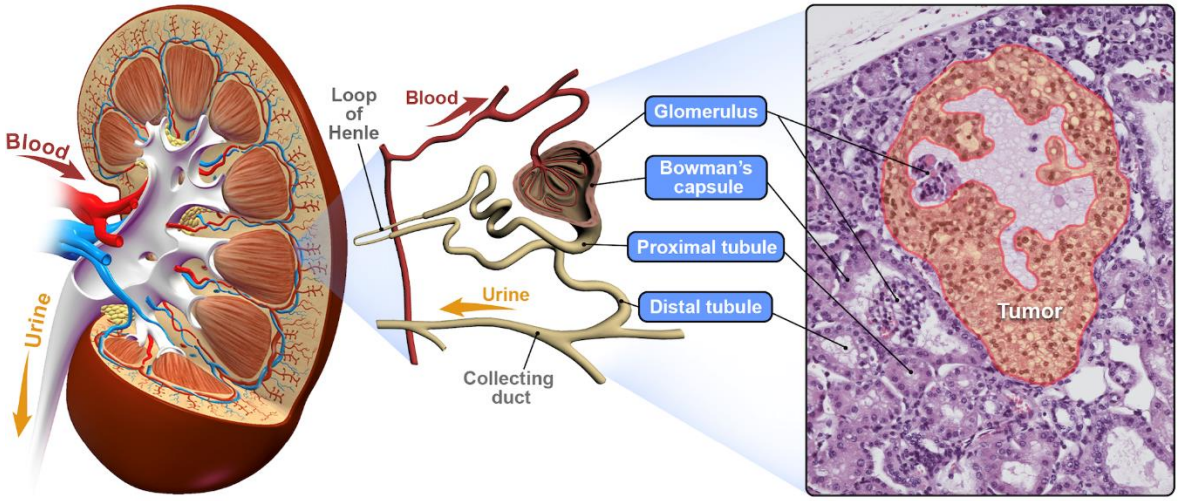
Gu et al., *Cancer Discovery* 2017

Vignette 8. While ccRCC is thought to arise from PCTs, PCT S1/S2 cells give rise to tRCC, but not ccRCC

Pathological studies support PEC as source of ccRCC



A, Human preneoplastic cyst constituted of atypical clear cells lining Bowman's capsule
B, *Pax8-Cre;Vhl$^{F/F}$;Pbrm1$^{F/F}$* model (Gu YF et al., *Cancer Discov.* 2017)
C, *Ksp1.3-CreERT2; Vhl$^{F/F}$;Trp53$^{F/F}$;Rb1$^{F/F}$* model

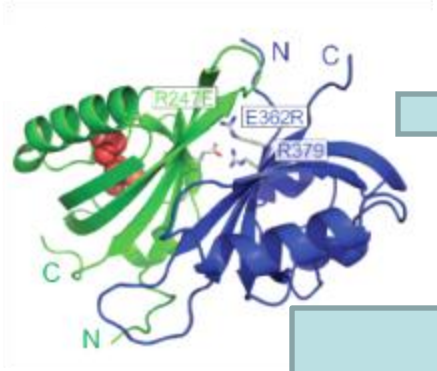


Gu et al., *Cancer Discov* 2017
(Harlander et al., *Nat Med*. 2017)
Kapur et al., Unpublished

Vignette 9. HIF2 targeting and biomarker identification in mouse models

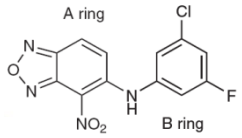
- Development of a first-in-class HIF2 inhibitor
- The quest for biomarkers (HIF2α, paraneoplastic syndromes)
- Identification of resistance mutations
- Development of a second-generation tumor targeted siRNA drug

Development of a HIF2 inhibitor at UTSW

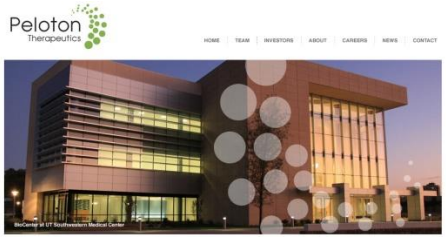


Scheuermann et
PNAS 2009

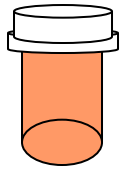
UTSW high-throughput library screen



Scheuermann et al.
Nat Chem Biol 2013

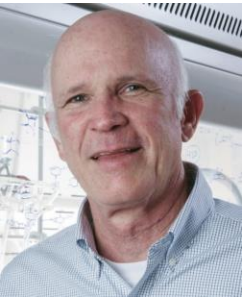


UT Southwestern BioCenter



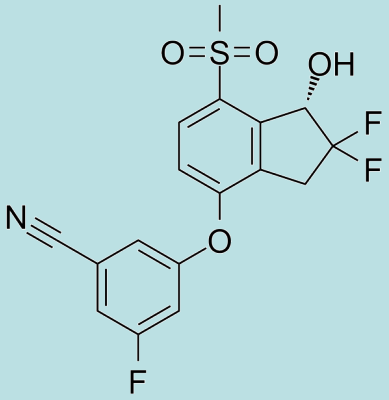
HIF2-I

HIF2α

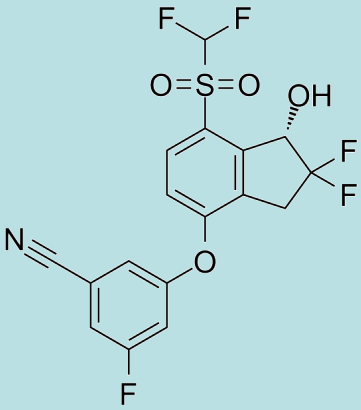


Tian et al.
Genes & Dev 1997

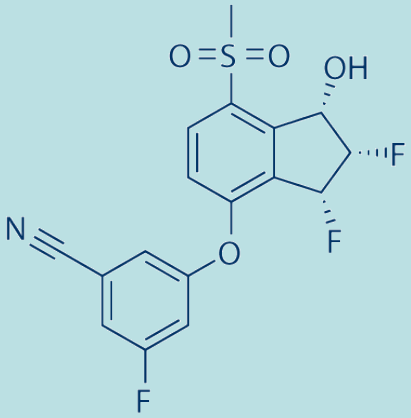
Peloton Therapeutics (PT) HIF2 inhibitors



PT2385

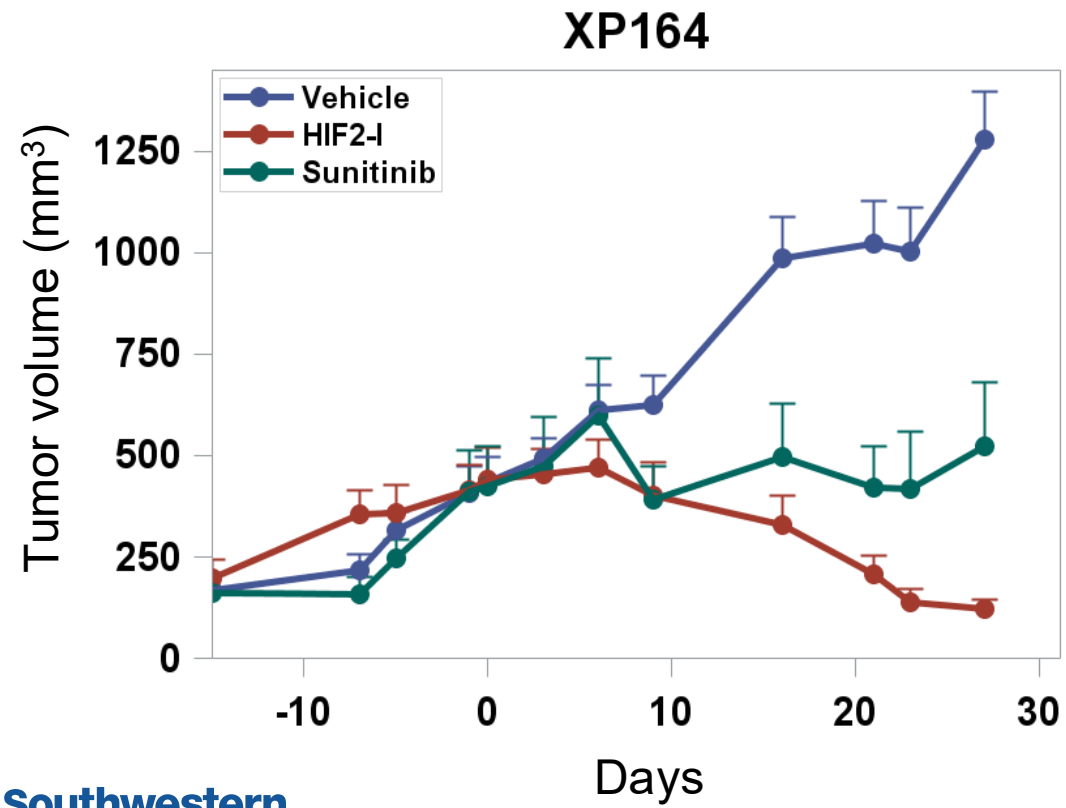


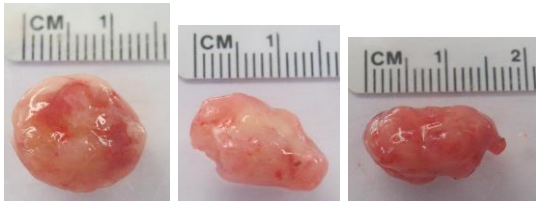
PT2399



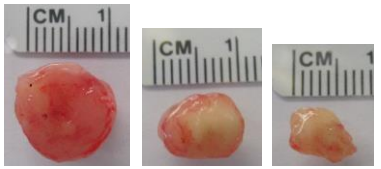
PT2977
Belzutifan

PT2399 is active against human ccRCC transplants in mice

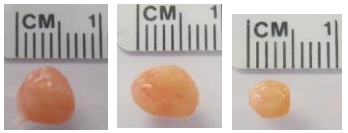




Vehicle



Sunitinib



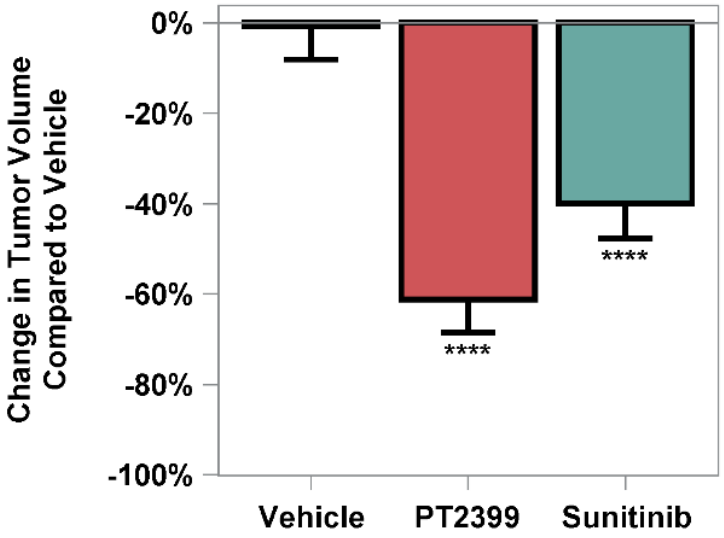
PT2399

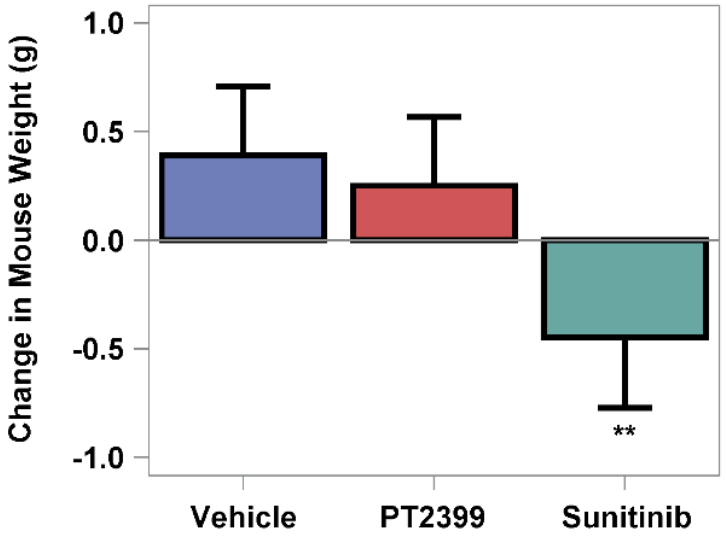
UT Southwestern Medical Center

Kidney Cancer Program

Chen et al., *Nature* 2016

PT2399 has greater activity than sunitinib and is better tolerated





267 mice from 22 independently derived TG lines (18 ccRCC)

Chen et al., *Nature* 2016

VOLUME 36 · NUMBER 9 · MARCH 20, 2018

JOURNAL OF CLINICAL ONCOLOGY

ORIGINAL REPORT

Phase I Dose-Escalation Trial of PT2385, a First-in-Class Hypoxia-Inducible Factor-2α Antagonist in Patients With Previously Treated Advanced Clear Cell Renal Cell Carcinoma

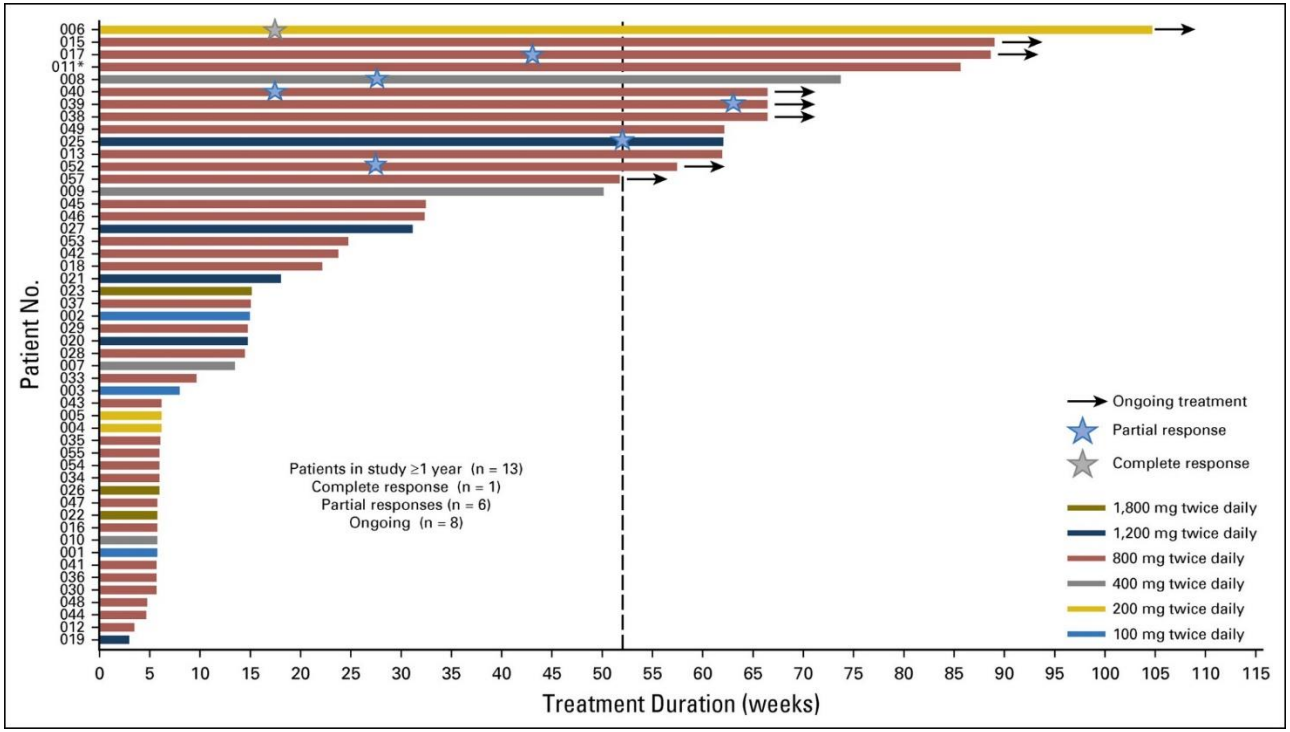
Kevin D. Courtney, Jeffrey R. Infante, Elaine T. Lam, Robert A. Figlin, Brian I. Rini, James Brugarolas, Naseem J. Zojwalla, Ann M. Lowe, Keshi Wang, Eli M. Wallace, John A. Josey, and Toni K. Choueiri

Author affiliations and support information (if applicable) appear at the end of this article.

Published at jco.org on December 19, 2017.

Clinical trial information: NCT02293980.

Corresponding author: Kevin D. Courtney, MD, PhD, Kidney Cancer Program, Simmons Comprehensive Cancer Center and Department of Medicine, Division of Hematology/Oncology, University of Texas Southwestern Medical Center, 5323 Harry Hines Blvd, Dallas, TX 75390-8852; e-mail: Kevin.Courtney@utsouthwestern.edu.



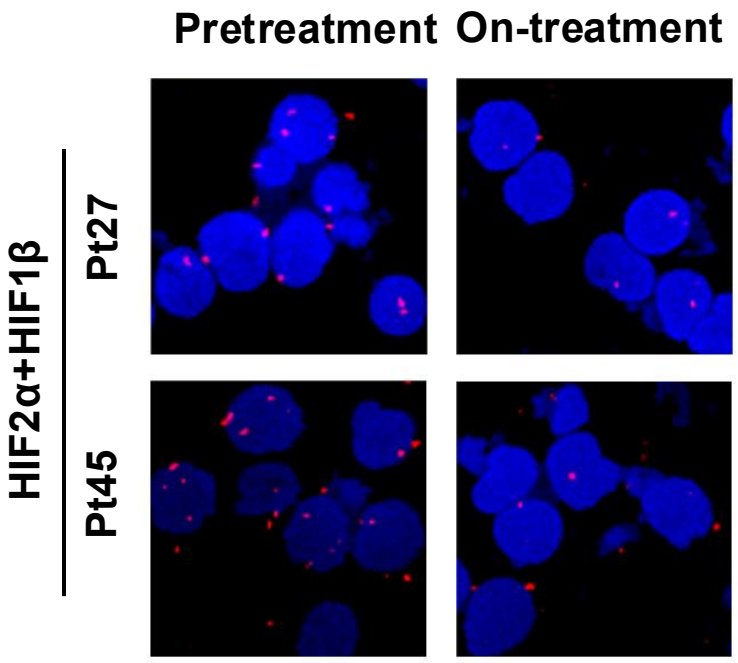
UT Southwestern Medical Center

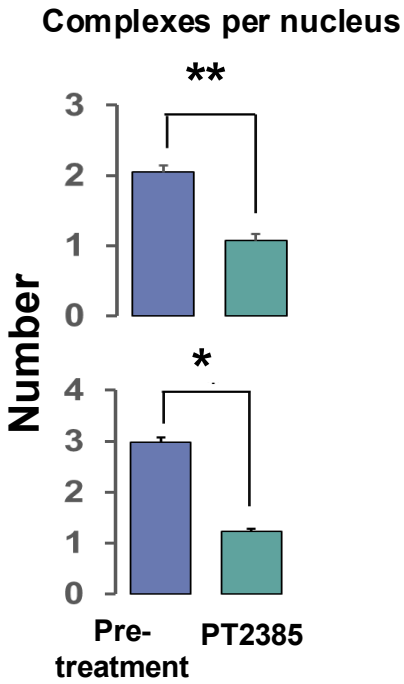
Kidney Cancer Program

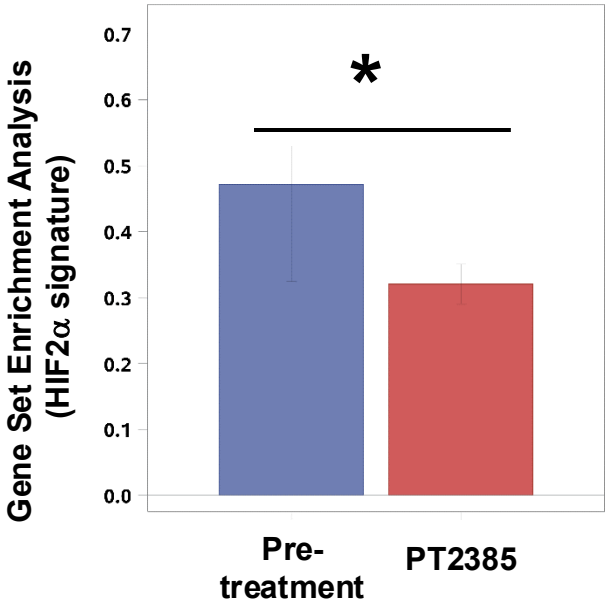
Courtney et al., *JCO* 2018

HIF-2 Complex Dissociation, Target Inhibition, and Acquired Resistance with PT2385, a First-in-Class HIF-2 Inhibitor, in Patients with Clear Cell Renal Cell Carcinoma

Kevin D. Courtney[1,2], Yuanqing Ma[1,2], Alberto Diaz de Leon[2,3], Alana Christie[2], Zhiqun Xie[2,4], Layton Woolford[1,2], Nirmish Singla[2,5], Allison Joyce[1,2], Haley Hill[1,2], Ananth J. Madhuranthakam[2,3], Qing Yuan[2,3], Yin Xi[3,4], Yue Zhang[3], Jenny Chang[1,2], Oluwatomilade Fatunde[1,2], Yull Arriaga[1,2], Arthur E. Frankel[1,2], Sanjeeva Kalva[3], Song Zhang[2,6], Tiffani McKenzie[2,7], Oscar Reig Torras[2], Robert A. Figlin[8], Brian I. Rini[9], Renée M. McKay[1,2], Payal Kapur[2,7], Tao Wang[2,4], Ivan Pedrosa[2,3], and James Brugarolas[1,2]



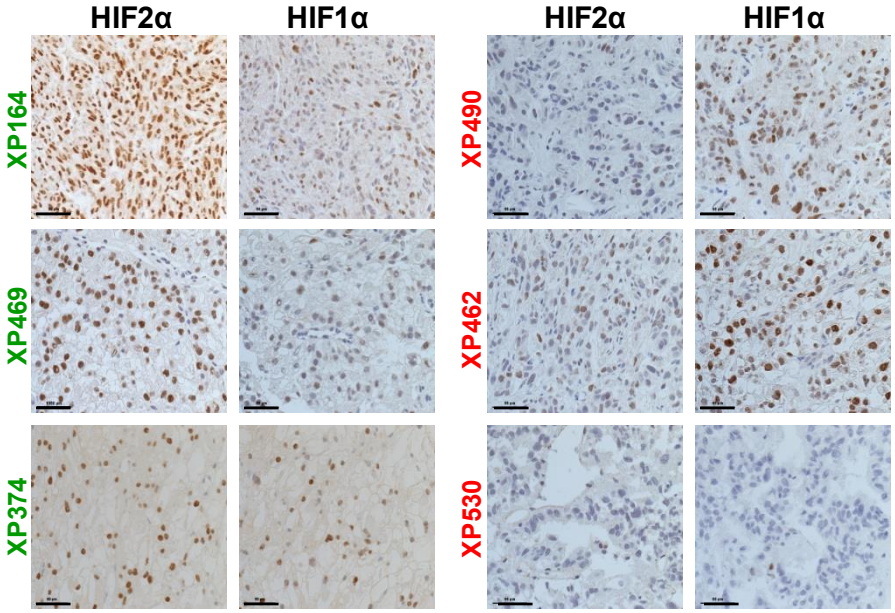


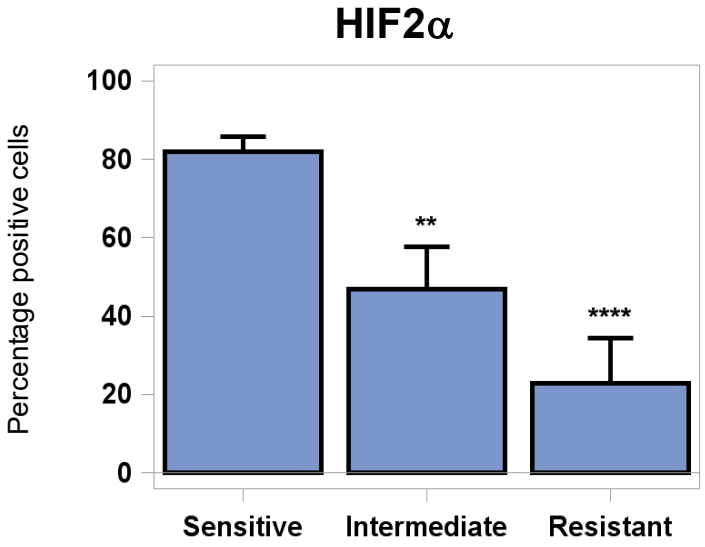


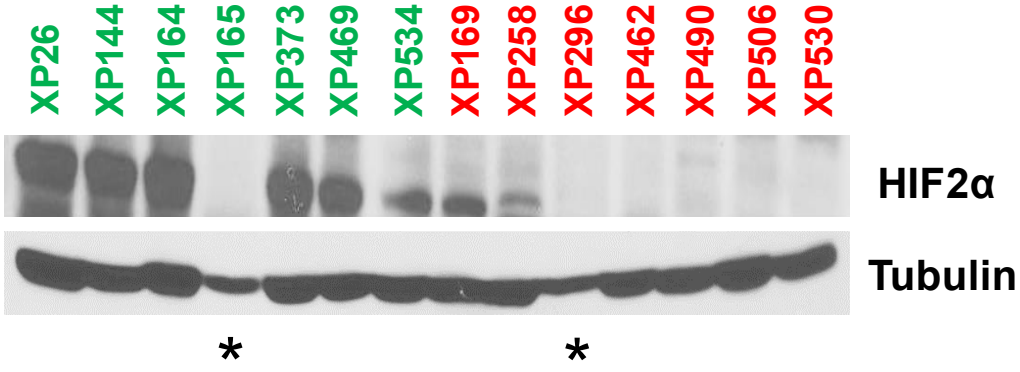
UT Southwestern Medical Center
Kidney Cancer Program

Courtney et al., *Clin Can Res* 2020

How are resistant and sensitive tumors different? HIF2α levels



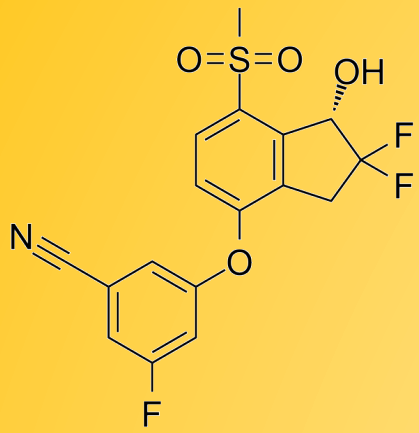




UT Southwestern Medical Center
Kidney Cancer Program

Chen et al., *Nature* 2016

Testing for HIF2α levels in patients: Turning PT2385 into a PET tracer

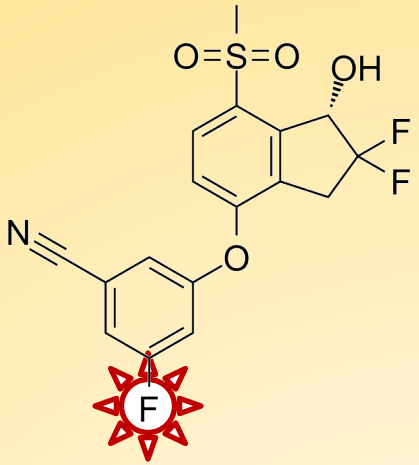


PT2385

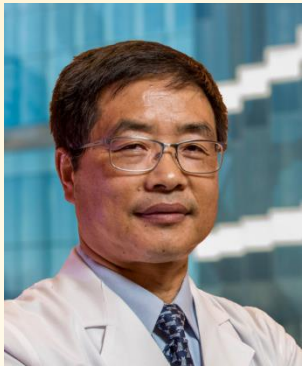
+

GE PETtrace 880 cyclotron
(11 US FDA INDs, 2 ANDAs, 1 RDRC for clinical trials
>30 investigational/novel radiotracers)

=



[^{18}F]PT2385



[^{18}F]PT2385 PET identifies HIF2α expressing ccRCC tumorgrafts in mice

FDA U.S. FOOD & DRUG ADMINISTRATION

IND 156933

STUDY MAY PROCEED

NIH National Library of Medicine
National Center for Biotechnology Information

ClinicalTrials.gov BETA

Resc

Home › Search Results › Study Record

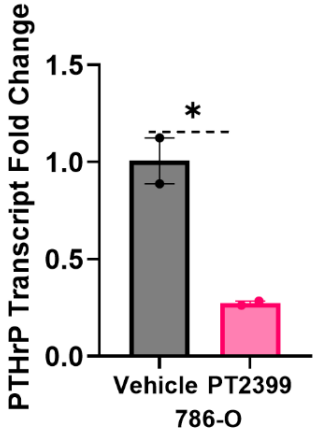
ClinicalTrials.gov

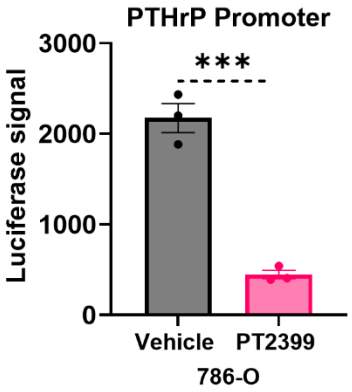
RECRUITING

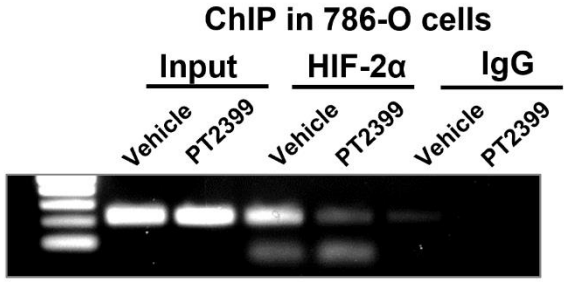
[18F]PT2385 PET/CT in Patients With Renal Cell Carcinoma

Principal Investigator: James Brugarolas, M.D., Ph.D.

Hypercalcemia of malignancy (HCM): A biomarker of HIF-2 dependency?

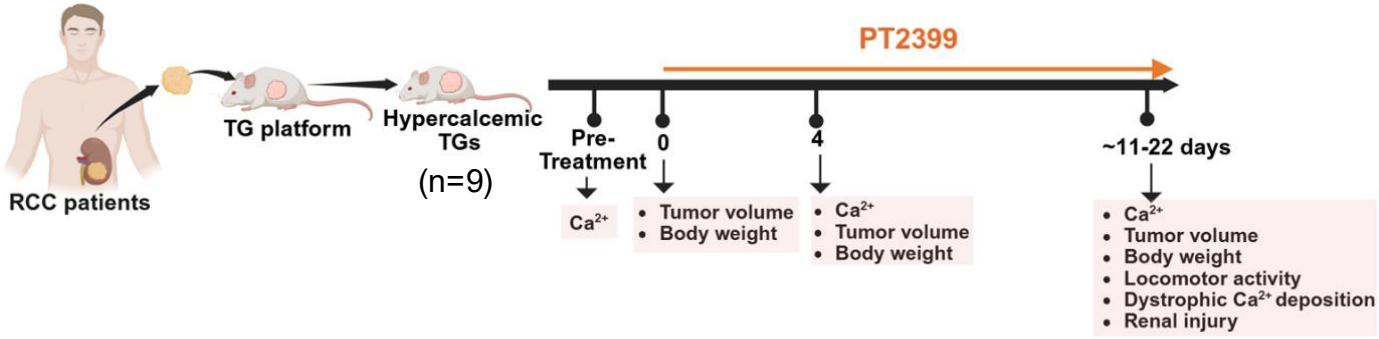


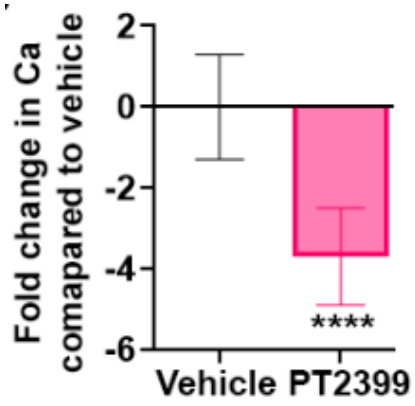


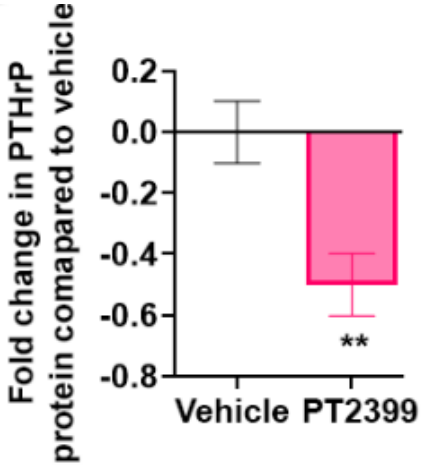


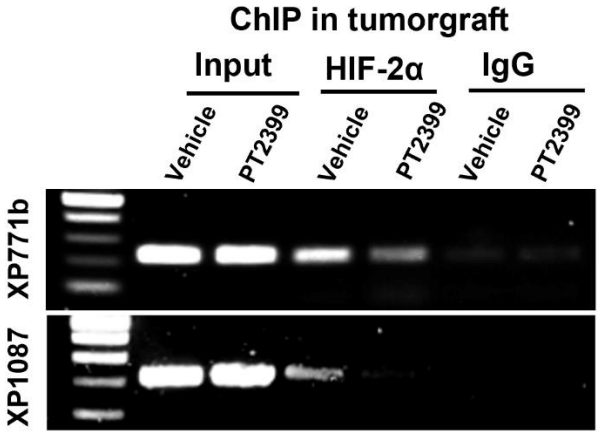
PT2399 downregulates *PTHrP* (*PTHLH*) expression and HIF-2 binding to promoter sequences

PT2399 (belzutifan analogue) suppresses PTHrP and hypercalcemia





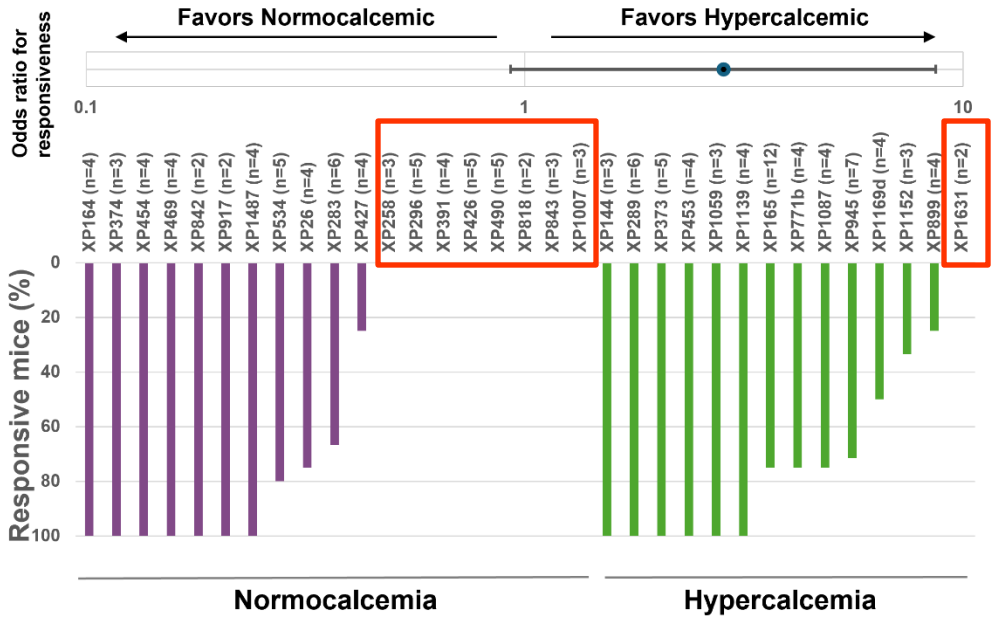




UT Southwestern Medical Center
Kidney Cancer Program

Unpublished, please do not post

HCM as a biomarker of HIF-2 dependency

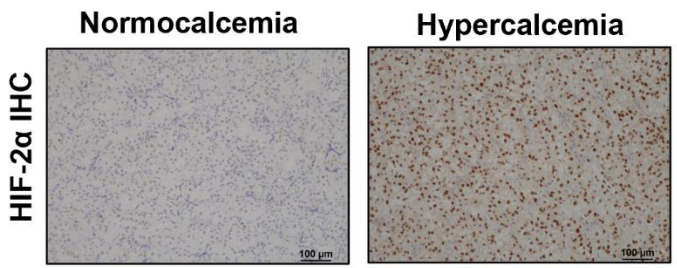


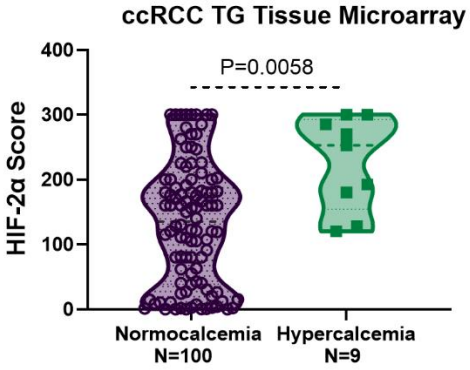
UT Southwestern Medical Center
Kidney Cancer Program

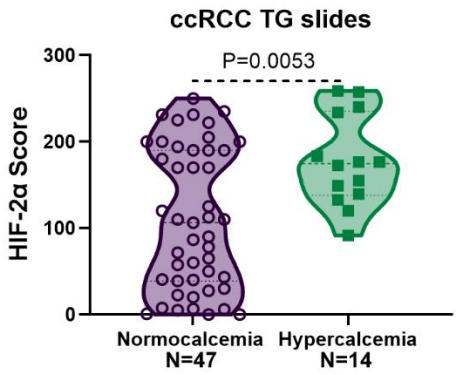
Unpublished, please do not post

HCM tumors express high levels of HIF-2α & are frequently sarcomatoid

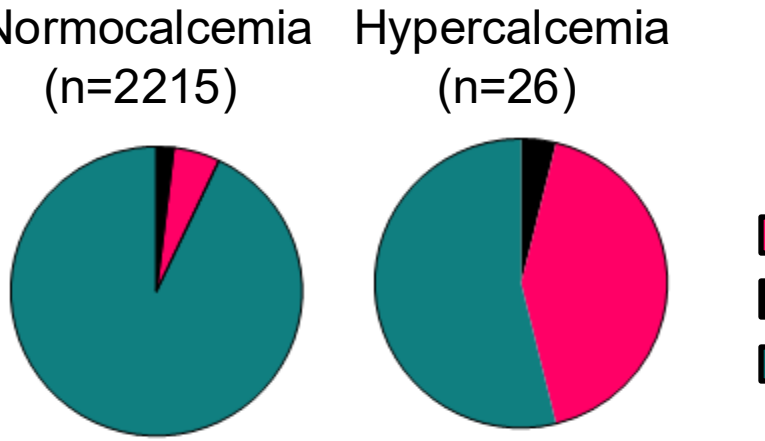
Mice





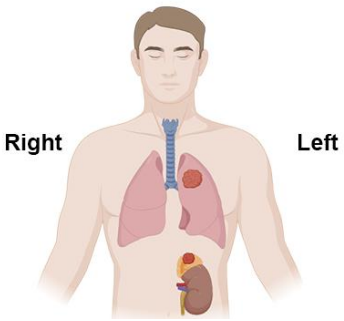


Humans

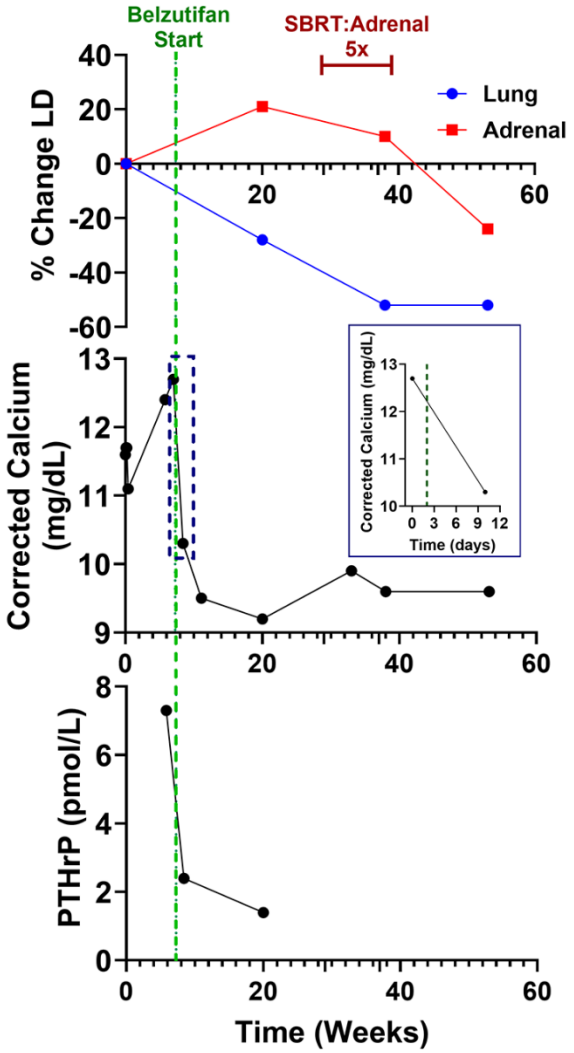


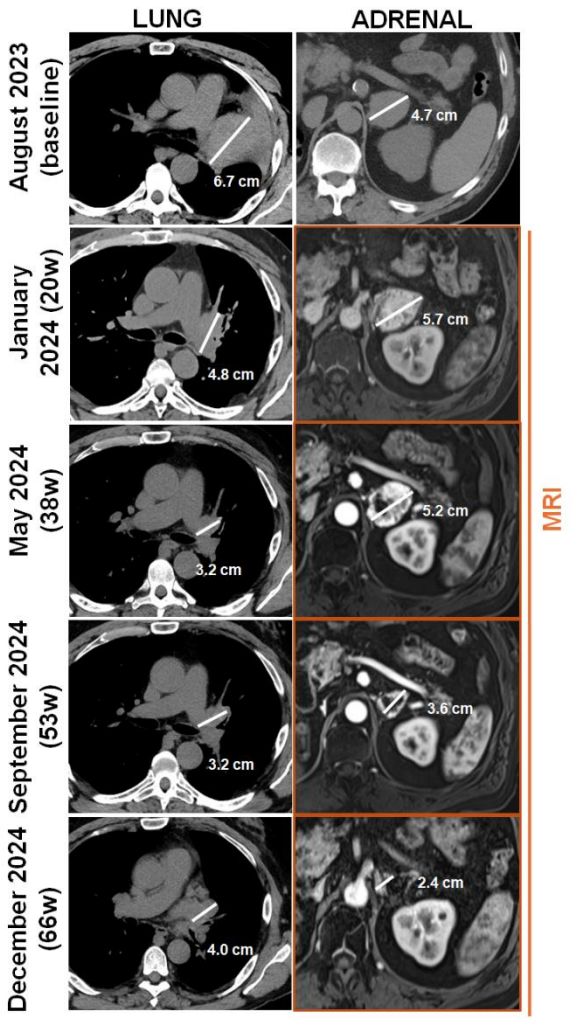
Unpublished, please do not post

Prospective Patient Case



ccRCC (sarc)

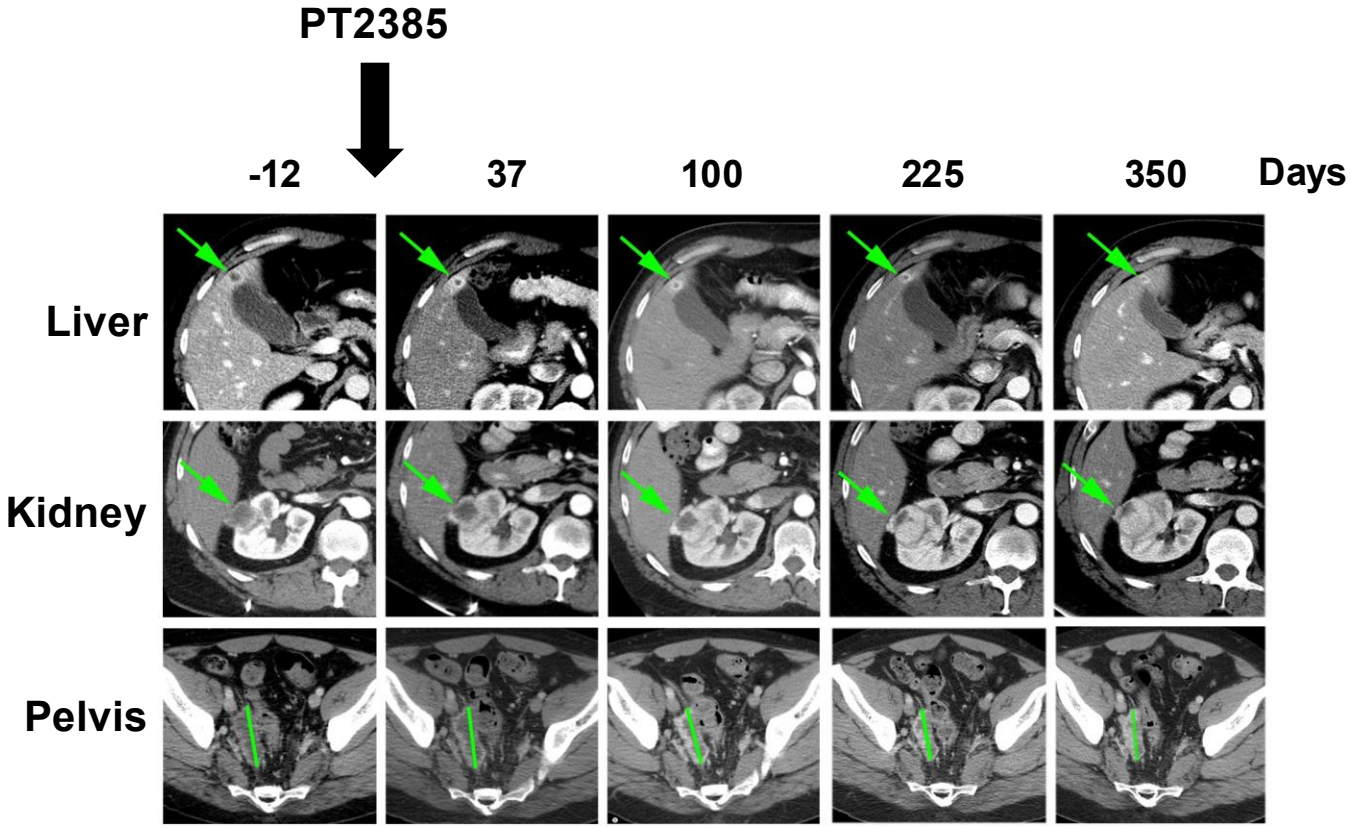




UT Southwestern Medical Center
Kidney Cancer Program

Unpublished, please do not post

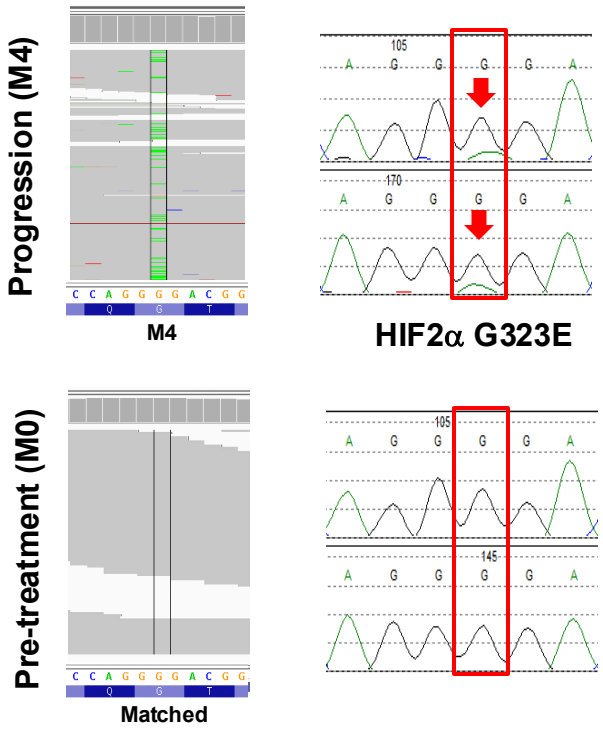
Resistance development to HIF2 antagonists



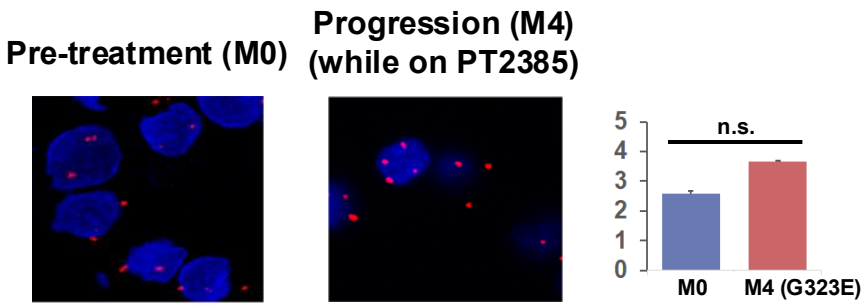
Chen et al., *Nature* 2016

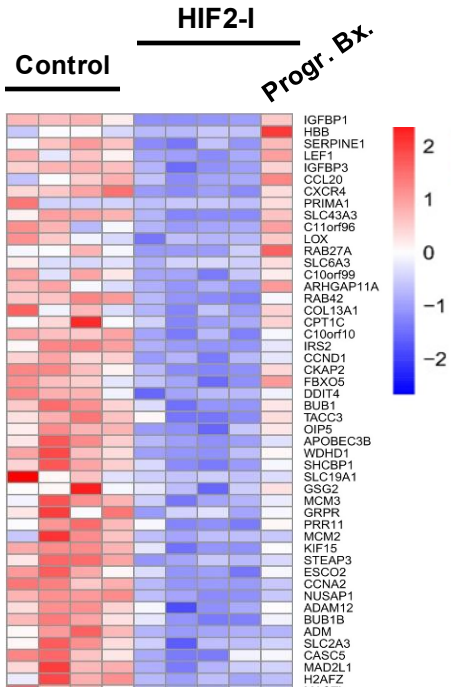
Acquired HIF2α mutation in drug binding pocket (G323E)

Patient 11



HIF2α+HIF1β

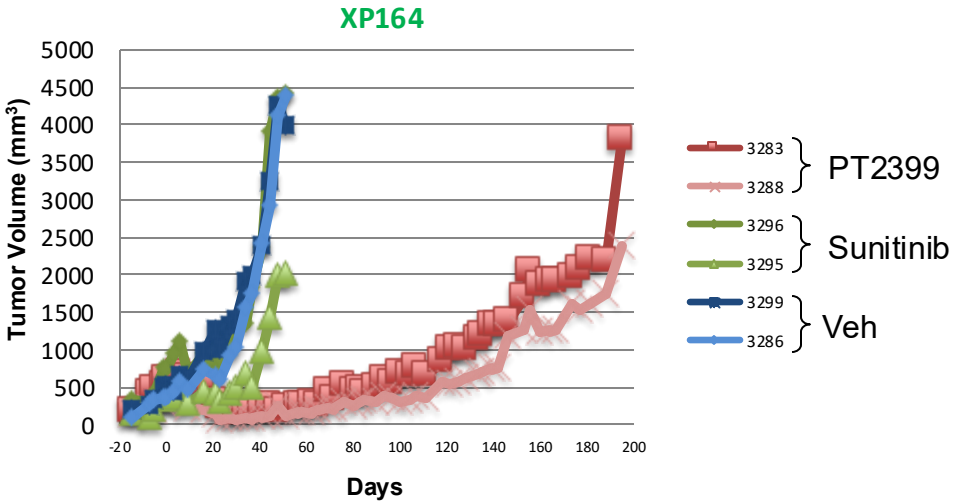


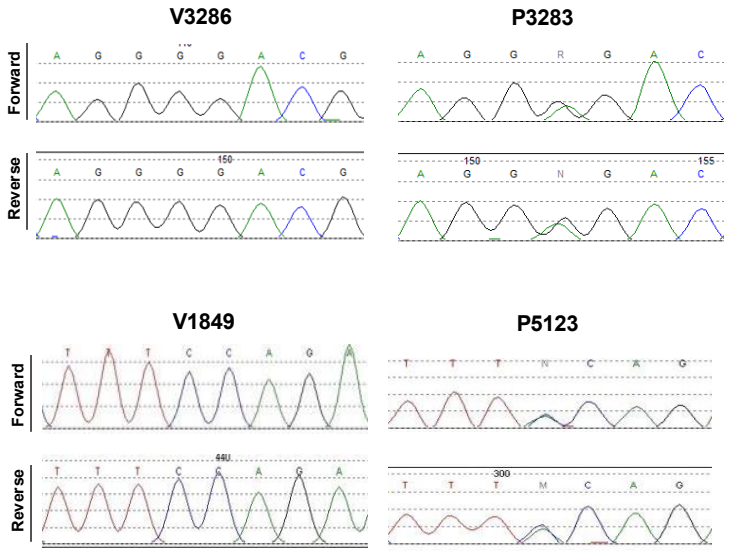


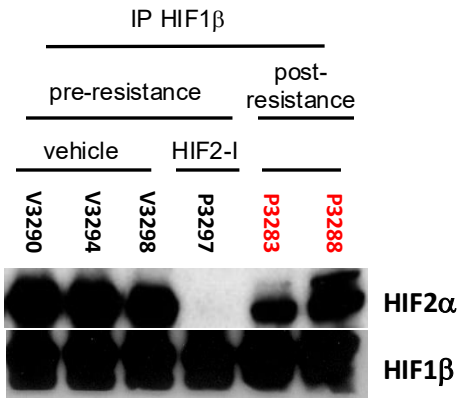
UT Southwestern Medical Center
Kidney Cancer Program

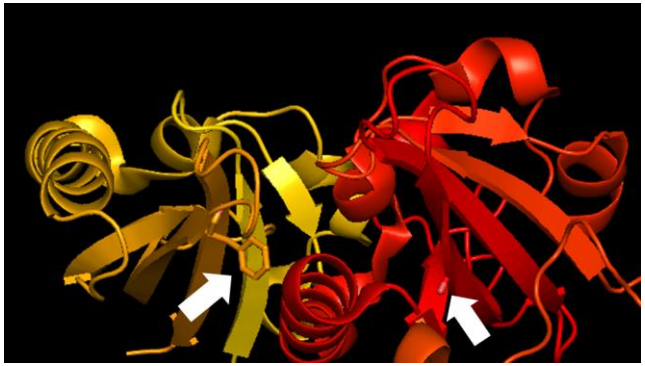
Courtney et al., *Clin Cancer Res* 2019

Resistance mutations were previously identified in TGs









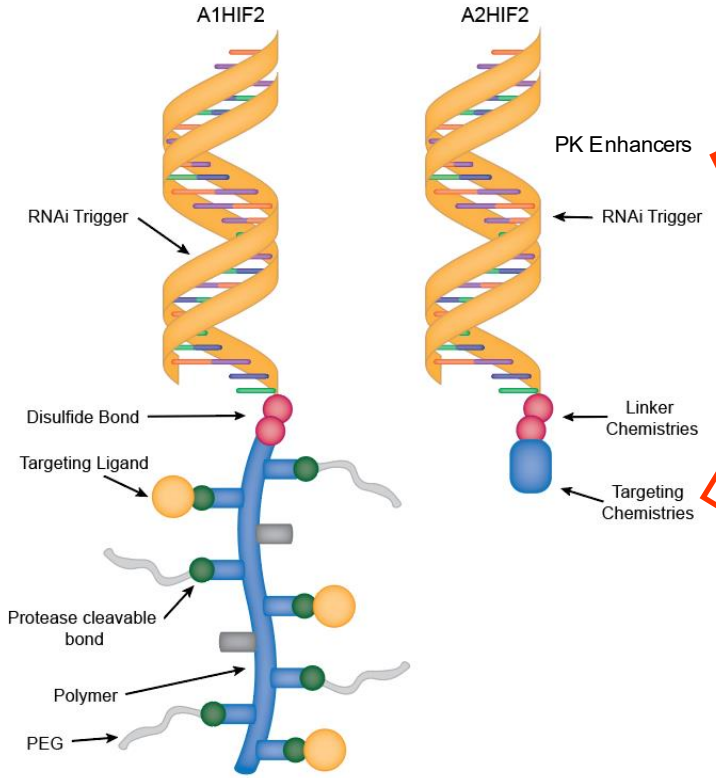
HIF1β HIF2α

UT Southwestern Medical Center
Kidney Cancer Program

Chen et al., *Nature* 2016

Towards 2nd generation HIF2 inhibitors with activity against resistant mutant HIF2

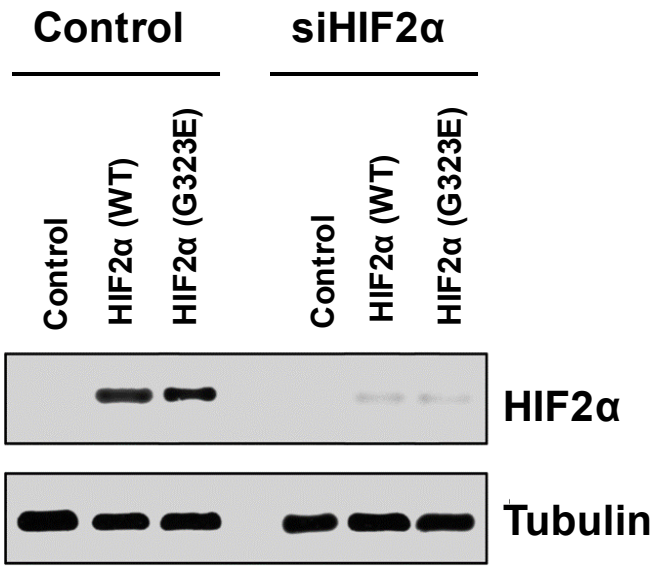
siHIF2α



Arrowhead Pharmaceuticals

HIF2α knockdown

αvβ3 interaction (RCC specificity)

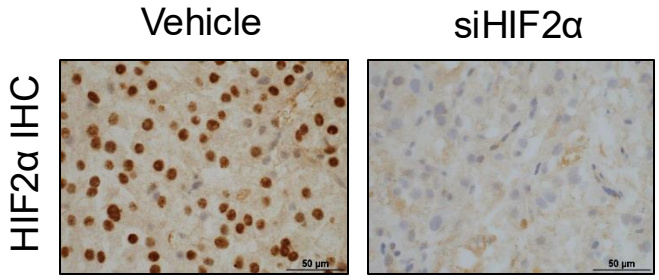


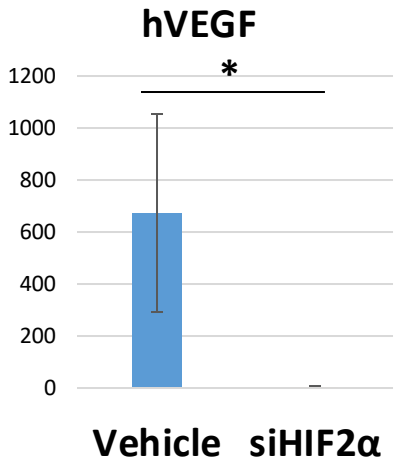
UT Southwestern Medical Center
Kidney Cancer Program

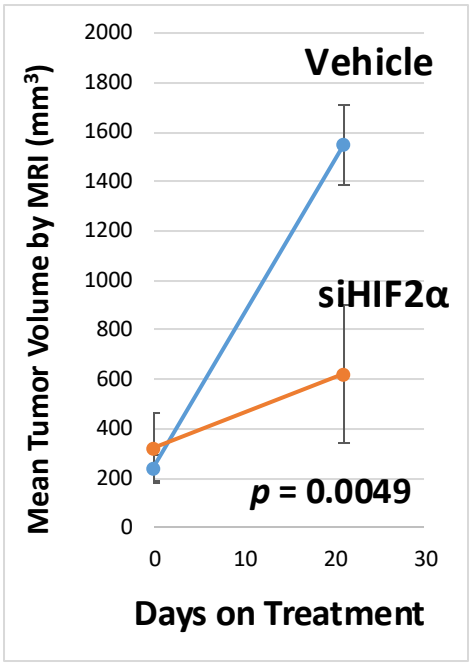
Ma et al., *Clin Cancer Res* 2022

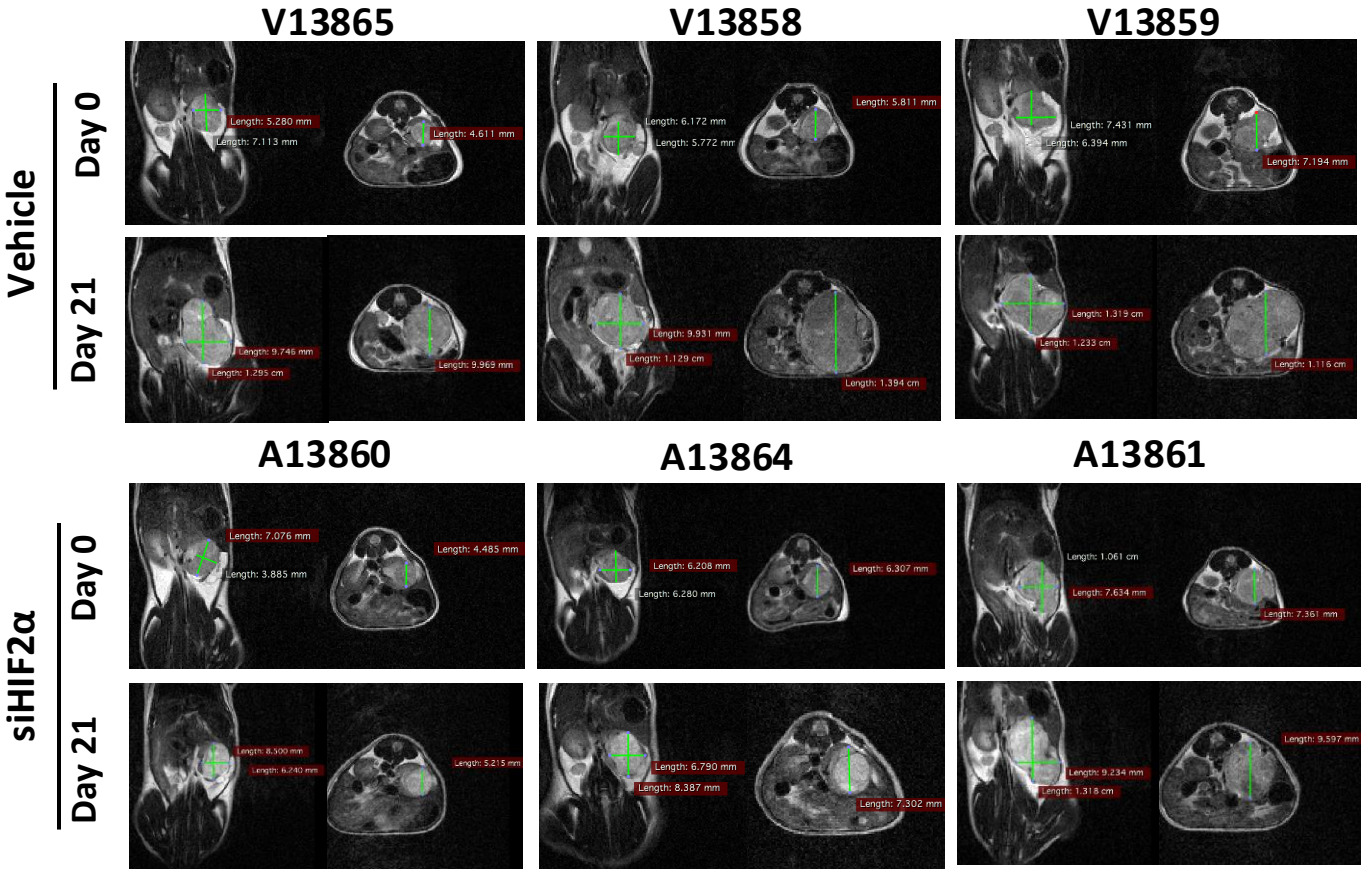
XP374

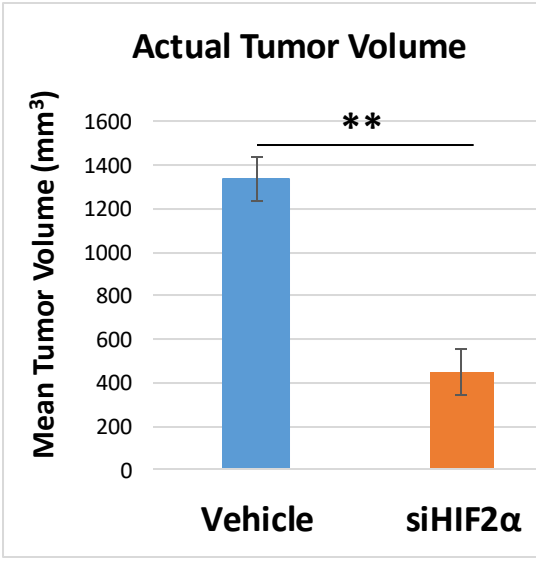
47 yo WM
L Nx (T4NxM0)
ccRCC











Ma et al., *Clin Cancer Res* 2022

Phase 1 trial offers proof-of-principle for HIF2α targeting by siRNA

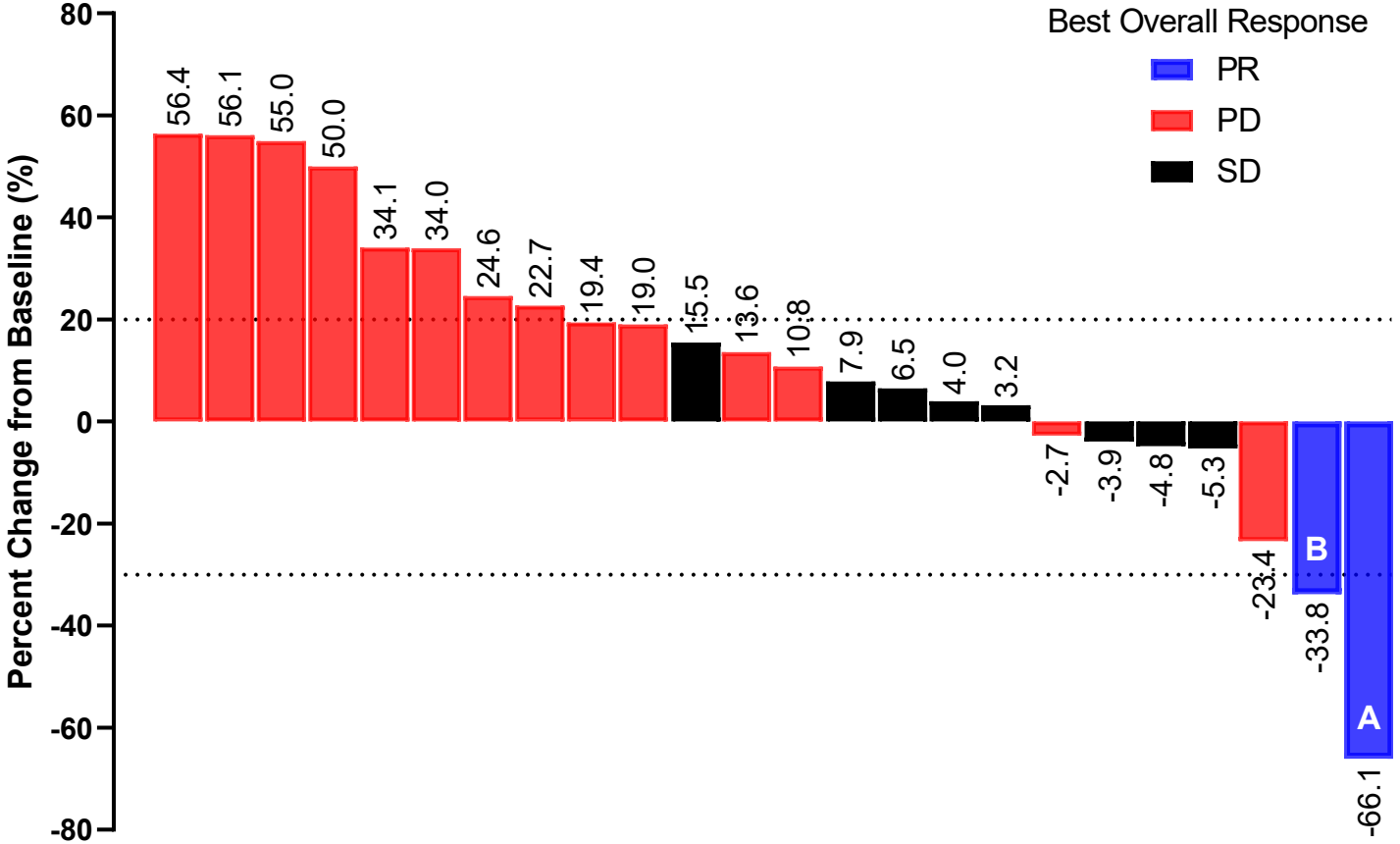
CLINICAL CANCER RESEARCH | CLINICAL TRIALS: TARGETED THERAPY

A First-in-Human Phase 1 Study of a Tumor-Directed RNA-Interference Drug against HIF2α in Patients with Advanced Clear Cell Renal Cell Carcinoma

James Brugarolas[1], Gregory Obara[2], Kathryn E. Beckermann[3], Brian Rini[3], Elaine T. Lam[4], James Hamilton[5], Thomas Schluep[5], Min Yi[5], So Wong[5], Zhongping Lily Mao[5], Erick Gamelin[6], and Nizar M. Tannir[7]

Brugarolas et al., *Clin Cancer Res* 2024

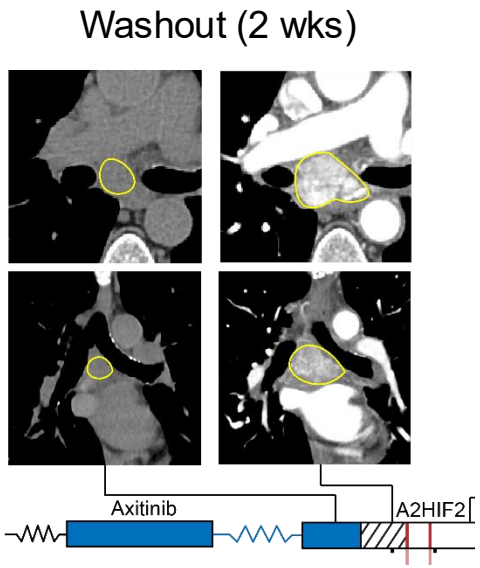
Waterfall Plot of Best Percent Change for Target Lesions

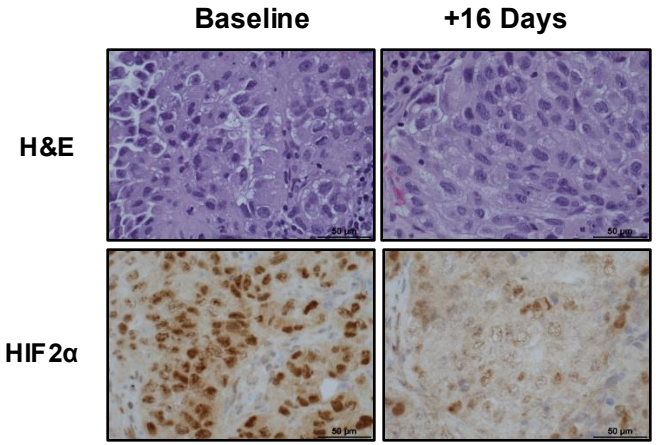


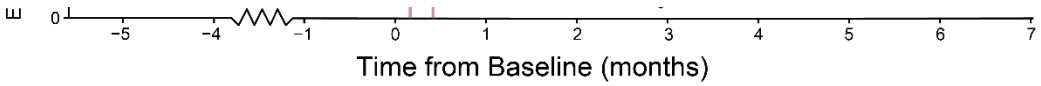
UT Southwestern
Medical Center
Kidney Cancer Program

Brugarolas et al., *Clin Cancer Res* 2024

Case Study: Patient A





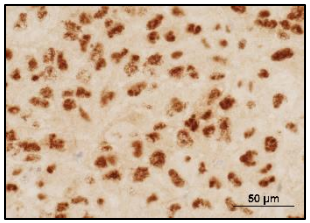


UT Southwestern Medical Center
Kidney Cancer Program

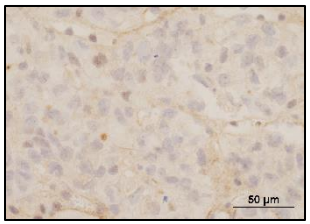
Ma et al., *Clin Cancer Res* 2022

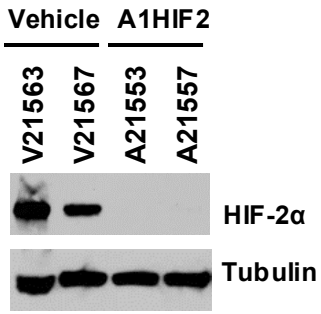
TG generated from patient A trial biopsy shows response to siRNA drug

Vehicle



A1HIF2





Ma et al., *Clin Cancer Res* 2022

Vignette 10. Probing resistance mechanisms to rapalogs

- Rapalogs (temsirolimus, a sirolimus/rapamycin prodrug, and everolimus), were first approved for mRCC by the FDA in 2007 (Hudes et al., *NEJM* 2007 and Motzer et al., *Lancet* 2008).
- Today, most often used in combination with lenvatinib (everolimus) in the refractory setting (and for nccRCC).
- Resistance uniformly arises, but how it develops has been a mystery since they were approved more than a decade ago.
- While rapalogs specifically bind mTORC1 and there are known mutations in mTOR that prevent rapalog binding, these mutations have not been identified in ccRCC (Hamieh et al., *PLOS Genet* 2018).

PLOS GENETICS

RESEARCH ARTICLE

Mechanisms of acquired resistance to rapalogs in metastatic renal cell carcinoma

Lana Hamieh[1,2], Toni K. Choueiri[1,2,3]*, Barbara Ogórek[1,2], Damir Khabibullin[1,2], Daniel Rosebrock[4], Dimitri Livitz[4], Andre Fay[1,5], Jean-Christophe Pignon[2,6], David F. McDermott[2,7], Neeraj Agarwal[8], Wenhua Gao[1,2,3], Sabina Signoretti[3,6]*, David J. Kwiatkowski[1,2,3]*

1 Department of Medicine, Brigham and Women's Hospital, Boston, MA, United States of America, **2** Department of Medicine, Harvard Medical School, Boston, MA, United States of America, **3** Department of Medical Oncology, Dana-Farber Cancer Institute, Boston, MA, United States of America, **4** Cancer Program, Broad Institute of Harvard and MIT, Cambridge, MA, United States of America, **5** Department of Medical Oncology, PUCRS School of Medicine, Porto Alegre, Brazil, **6** Department of Pathology, Brigham and Women's Hospital, Boston, MA, United States of America, **7** Department of Medicine, Beth Israel Deaconess Medical Center, Boston, MA, United States of America, **8** Division of Medical Oncology, University of Utah Huntsman Cancer Institute, Salt Lake City, UT, United States of America

* toni_choueiri@dfci.harvard.edu (TKC); ssignoretti@partners.org (SS); dk@rics.bwh.harvard.edu (DJK)

"We were somewhat surprised that we did not identify any secondary mutations in MTOR (…) in any of these six patients, nor were MTOR mutations associated with resistance development in even a single case."

"We conclude that mechanisms of resistance to rapalog therapy in RCC are not easily explained by mutations in most cases, and likely depend on more subtle transcriptional and/or epigenetic changes."

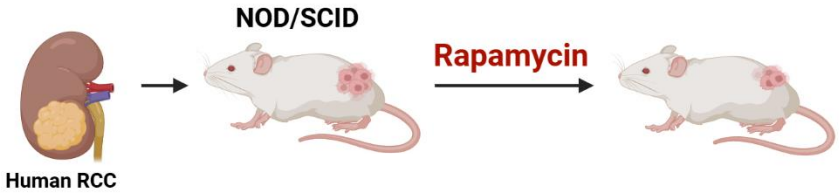
Unexpected resistance mechanisms to rapalogs

PNAS RESEARCH ARTICLE | MEDICAL SCIENCES OPEN ACCESS

Unconventional mechanism of action and resistance to rapalogs in renal cancer

Juan Yang[a,b], Ramesh Butti[a,b], Shannon Cohn[c], Vanina Toffessi-Tcheuyap[a,b], Arijit Mal[a,b], Mylinh Nguyen[d], Christina Stevens[a,b], Alana Christie[a], Akhilesh Mishra[a,b], Yuanqing Ma[a,b], Jiwoong Kim[e], Robert Abraham[f], Payal Kapur[a,g], Robert E. Hammer[d], and James Brugarolas[a,b,1]

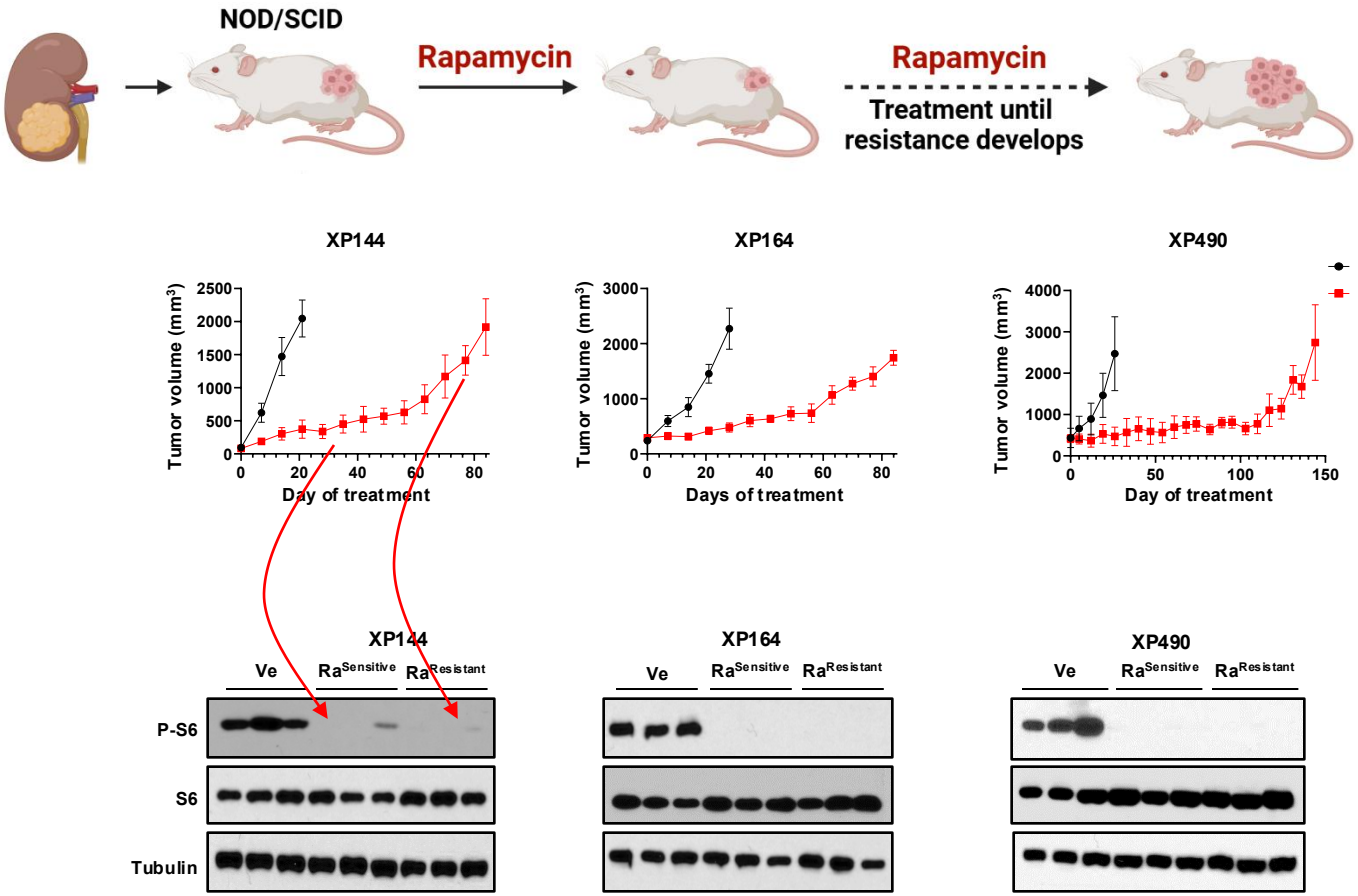
Edited by Joan Brugge, Harvard Medical School, Boston, MA; received June 29, 2023; accepted April 25, 2024



Rapamycin (sirolimus) accounts for 70% of circulating drug after temsirolimus and is an everolimus analog

Image generated with BioRender

As for PT drugs, TGs develop resistance to rapamycin with prolonged treatment

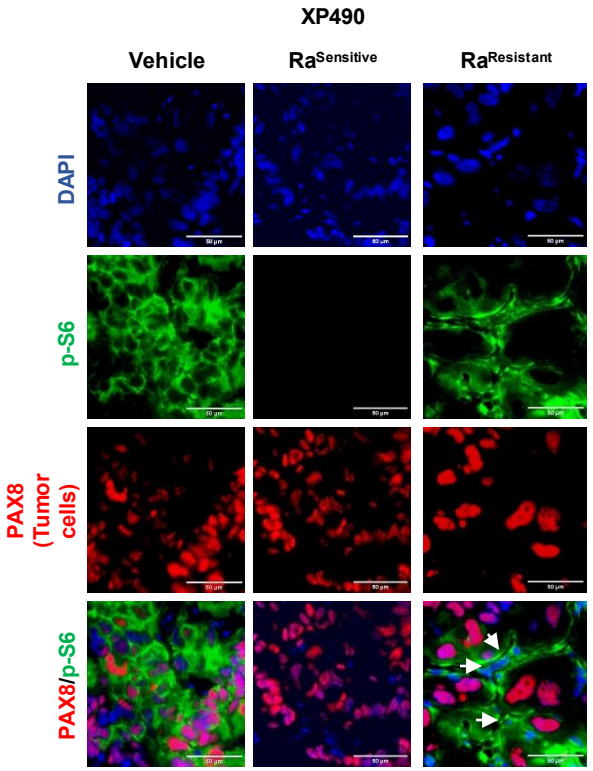


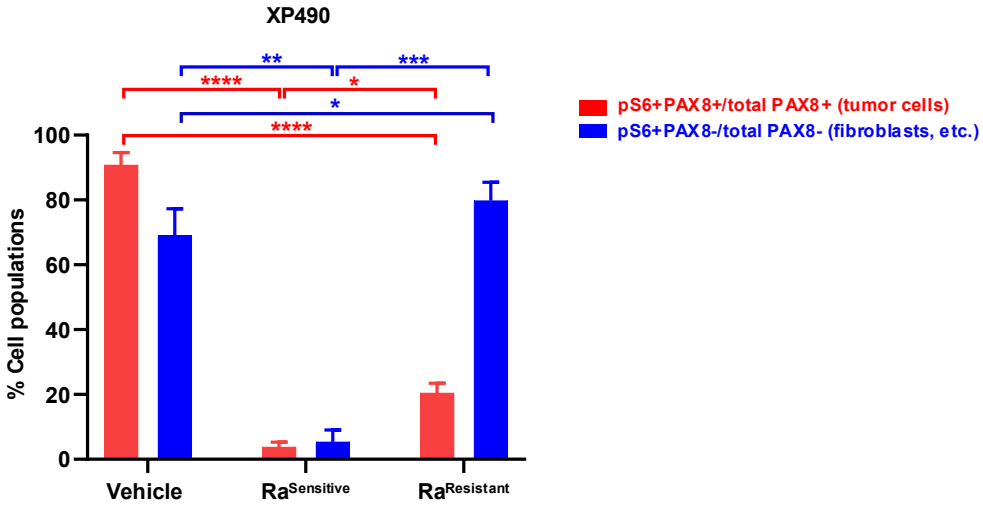
UTSouthwestern Medical Center

Kidney Cancer Program

Yang et al., *PNAS* 2024

Resistance development correlates with mTORC1 reactivation in stromal (but not tumor) cells suggesting that the stroma plays key role

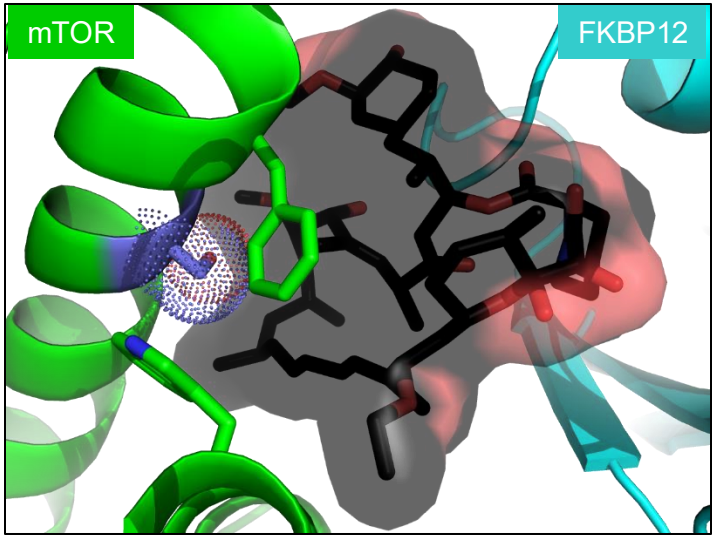




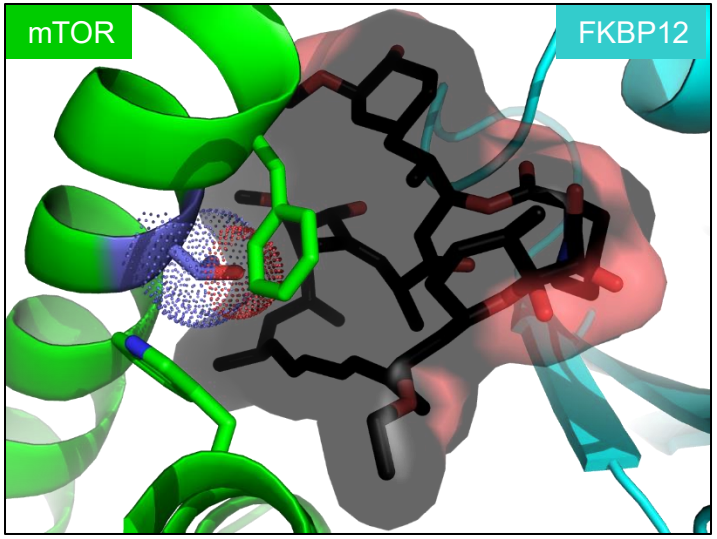
Yang et al., *PNAS* 2024

Engineering a mutation in NOD/SCID recipient mice to block rapamycin binding

Ser2035 VDW Radius



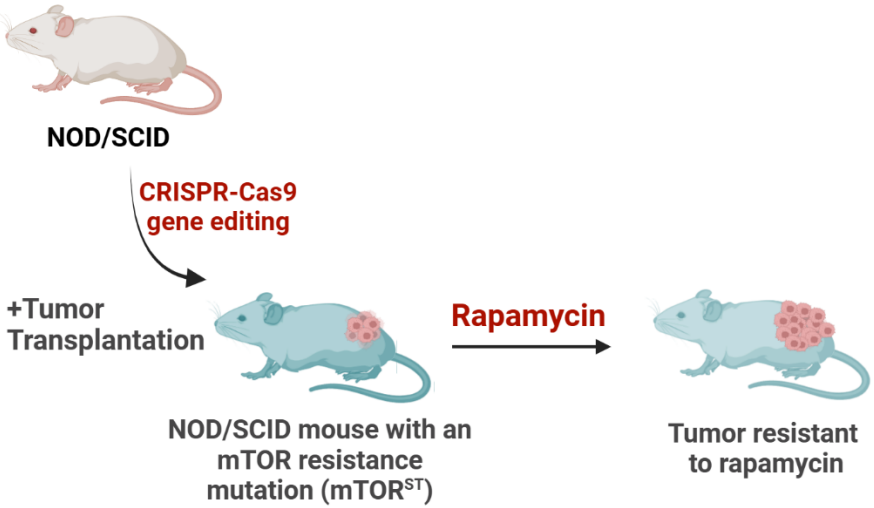
Thr2035 VDW Radius

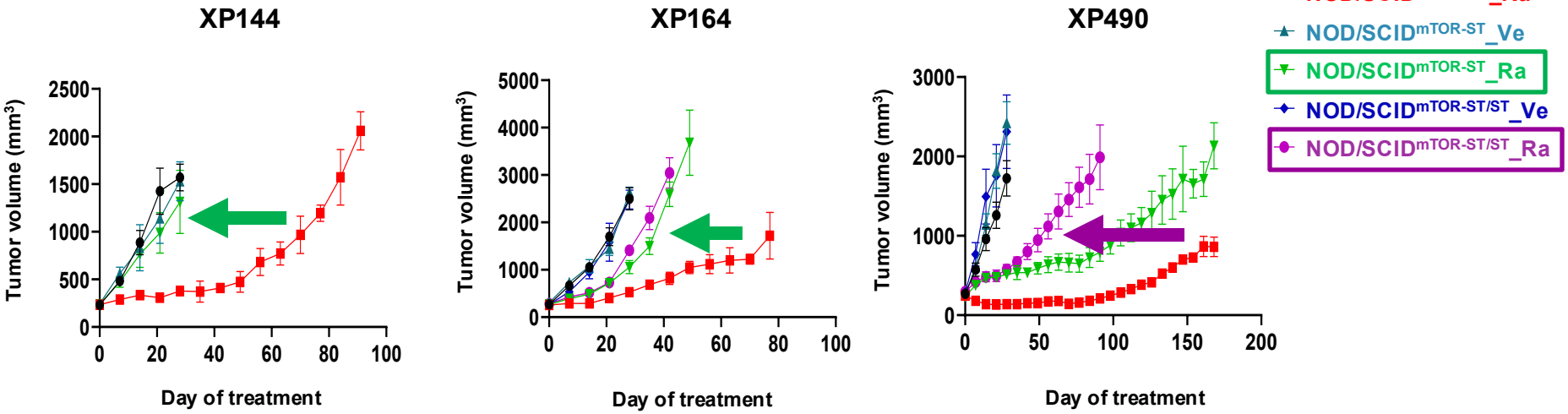


mTOR S2035T substitution ($mTOR^{ST}$) induces a steric clash and alters polarity precluding rapamycin binding

Courtesy of Lisa Kinch
From 2fap (PubMed: 10089303)

Engineering an mTOR resistance mutation in the NOD/SCID mouse genome is sufficient to induce resistance in transplanted tumors





Yang et al., *PNAS* 2024

Brugarolas Lab

Tolulope Ayo
Ramesh Butti
Alana Christie
Nazmul Hasan
Sophronia Hipsh
Arijit Mal
Jeffrey Miyata
Gopinath Prakasam
Vanina Toffessi Tcheuyap
Bingqing Xie

Olivia Brandenburg
Wenfang Chen
Shannon Cohn
Roy Elias
Ming Gao
Yifeng Gu
Haley Hill
Allison Joyce
Blanka Kucejova
Eric Ma
Akhilesh Mishra
Tiffani McKenzie
Renee McKay
Oreoluwa Onabolu
Andrea Pavia-Jimenez
Samuel Peña-Llopis
Oscar Reig Torras
Nirmish Singla
Sharanya Sivanand
Charlotte Small
Christina Stevens
Tram Anh Tran
Silvia Vega-Rubin de Celis
Shanshan Wang
Nick Wolff
Layton Woolford
Toshinari Yamasaki
Juan Yang
Anum Yousuf
Haitao Xu

Kidney Cancer Program

Debra Harvey
Jenny Beth Humann
Kathy Worland

Urology
Jeff Cadeddu
Jeff Gahan
Kris Gaston
Brett Johnson
Yair Lotan
Vitaly Margulis
Xiaosong Meng
Ganesh Raj
Solomon Woldu

Oncology
Waddah Arafat
Suzanne Cole
Kevin Courtney
Hans Hammers
Charles Jiang
Janie Qin
Tian Zhang
Jue Wang

Radiation Oncology
Aurelie Garant
Raquibul Hannan
Robert Timmerman

Pathology
Payal Kapur
Dinesh Rakheja

Radiology
Jovan Begovic
Ivan Pedrosa
Orhan Oz
Xiankai Sun

Bioinformatics

Lindsay Cowell
Andrew Jamieson
Satwik Rajaram
Phillip Reeder
Tao Wang
Hua Zhong

Clinical Coordinators
Vida Rhodes
Amy Rowell

UTSW Collaborators

Laura Banaszynski
Rick Bruick
Tom Carroll
Maralice Conacci Sorrell
Diego Castrillon
Ralph DeBerardinis
Kevin Gardner
Bob Hammer
Lisa Kinch
Weibo Luo
Sri Malladi
Denise Marciano
Steve McKnight
Josh Mendell
Bruce Posner
Ben Sabari
Andrew Wang
Noelle Williams
Chen Yao
Qing Zhang

Mayo Clinic

Richard W. Joseph
John C. Cheville
Jeanette Eckel-Passow
Thai Ho
Brad Leibovich
Alexander Parker
Daniel J. Serie

NCI

Laura Schmidt
Marston Linehan

Other Collaborators

Brian Rini

PATIENTS & THEIR FAMILIES

Peloton

John Josey
Eli Wallace

ARO-HIF2 Trial

Gregory Obara
Katy Beckermann
Brian Rini
Elaine Lam
James C Hamilton
Thomas Schluep
Min Yi
So Wong
Zhongping Lily Mao
Erick Gamelin
Nizar Tannir

Genentech

Anwesha Dey
Steffen Durinck
Zora Modrusan
Sekar Seshagiri

Funding

Sherry Wigley Crow Endowment
Virginia Murchison Linthicum Endowment
NIH, National Cancer Institute
Cancer Prevention and Research Institute of Texas
Department of Defense Kidney Cancer Program
V Foundation
Joey's Wings Foundation

Genentech/Roche
Peloton Therapeutics
Arrowhead Pharmaceuticals

Slides generated with BioRender