Corporate Overview

December 2021

NASDAQ: AADI
www.aadibio.com

Aadi Bioscience Inc., Pacific Palisades, CA 90272, USA

Cautionary Note Regarding Forward-Looking Statements

Certain statements contained in this presentation regarding matters that are not historical facts, are forward-looking statements within the meaning of Section 21E of the Securities and Exchange Act of 1934, as amended, and the Private Securities Litigation Act of 1995, known as the PSLRA. These include statements regarding management's intention, plans, beliefs, expectations or forecasts for the future, and, therefore, you are cautioned not to place undue reliance on them. No forward-looking statement can be guaranteed, and actual results may differ materially from those projected. Aadi Bioscience, Inc. ("Aadi") undertakes no obligation to publicly update any forward-looking statement, whether as a result of new information, future events or otherwise, except to the extent required by law. We use words such as "anticipates," "believes," "plans," "expects," "projects," "intends," "may," "will," "should," "could," "estimates," "predicts," "potential," "continue," "guidance," and similar expressions to identify these forward-looking statements that are intended to be covered by the safe-harbor provisions of the PSLRA.

Such forward-looking statements are based on our expectations and involve risks and uncertainties; consequently, actual results may differ materially from those expressed or implied in the statements due to a number of factors, including, but not limited to, Aadi's plans to develop and commercialize its product candidates, including FYARRO™ (nab-sirolimus, ABI-009); Aadi's commercialization, marketing and manufacturing capabilities and strategy; the clinical utility, potential benefits and market acceptance of FYARRO and any other of Aadi's product candidates; risks related to the sufficiency Aadi's cash balance to fund operations; the timing of initiation of Aadi's planned clinical trials; the timing of the availability of data from Aadi's clinical trials; Aadi's plans to research, develop and commercialize its current and future product candidates; Aadi's ability to successfully enter into collaborations, and to fulfill its obligations under any such collaboration agreements; Aadi's ability to identify additional products or product candidates with significant commercial potential; developments and projections relating to Aadi's competitors and our its industry; the impact of government laws and regulations; Aadi's ability to protect its intellectual property position; the impact of the COVID-19 outbreak on Aadi's operations, the biotechnology industry and the economy generally and Aadi's estimates regarding future revenue, expenses, capital requirements and need for additional financing.

These risks are described in detail under the caption "Risk Factors" in Aadi's Quarterly Report on Form 10-Q for the quarter ended September 30, 2021, filed with the Securities and Exchange Commission (the "SEC") on November 10, 2021, and other documents filed from time to time with the SEC. Forward-looking statements included in this presentation are based on information available to Aadi as of the date of this presentation. Aadi undertakes any obligation to such forward-looking statements to reflect events or circumstances after the date of this presentation.

FYARRO Granted Full Approval Following Priority Review by the FDA

FDA Approval
Nov 22, 2021

First and only therapy approved to treat locally advanced unresectable or metastatic malignant PEComa

Albumin-bound mTOR inhibitor leveraging *nab*-technology to deliver a favorable clinical profile in an area of high unmet medical need

Efficacy defined by **ORR and DOR** in the prescribing information

Adverse event profile that is **predictable and manageable**

Aadi Bioscience is a Commercial-Stage, Precision Oncology Company Re-engineering mTOR Inhibition

NASDAQ: AADI

- Focused on the commercialization of FYARRO™ (sirolimus protein-bound particles for injectable suspension [albumin-bound], *nab*-sirolimus), an albumin-bound mTOR inhibitor developed using the validated nanoparticle albumin-based (*nab*) platform proven with ABRAXANE®
- FYARRO™ now FDA approved as the only product indicated for locally advanced unresectable or metastatic malignant PEComa; commercial preparations underway ahead of Q1 2022 launch
- New registrational study to be initiated pursuing a significant potential opportunity in tumor-agnostic *TSC1* or *TSC2* inactivating alterations in solid tumors – recent data (ASCO 2021) suggests encouraging activity of ABI-009
- Cash runway into 2024 to support commercialization and future clinical development of FYARRO™

Leadership: We are Building a World-Class Team with Deep Expertise in Oncology Commercialization and Development

Neil Desai, PhD
Founder, CEO and President

- Former Sr VP, Global R&D at **Abraxis** Bioscience
- Previously Vice President, Strategic Platforms, **Celgene**; prior positions of increasing seniority at **American BioScience, Inc.**
- Inventor of the *nab* technology (Abraxane and ABI-009)
- 25+ years in R&D

Brendan Delaney, MBA
Chief Operating Officer

- Previously CCO at **Constellation** Pharma (acquired by MorphSys)
- Former CCO at **Immunomedics** (acquired by Gilead); VP of US Hematology /Oncology at **Celgene**
- 25+ years commercial experience including prior roles at **Novartis Oncology** and **Genentech**

Scott Giacobello, CPA
Chief Financial Officer

- Previously CFO **GW Pharmaceuticals**
- Former CFO **Chase Pharmaceutical Corp.**; EVP&CFO **VIZI Health Solutions**; VP Finance-Global R&D **Allergan** as well as VP Corporate Finance and VP Finance – Audit and Compliance
- 13+ years pharmaceutical finance experience

Loretta Itri, MD
Chief Medical Officer

- Previously CMO at **Immunomedics**
- Former EVP Global Health Sciences & Regulatory Affairs, **The Medicines Company**; President, Pharmaceutical Development, CMO **Genta, Inc.**; SVP, WW Clinical Affairs at **Johnson & Johnson, Ortho Biotech**
- 40+ years drug development experience

Mitchall Clark, BPharm, MRPharmS
SVP Regulatory Affairs and Quality Assurance

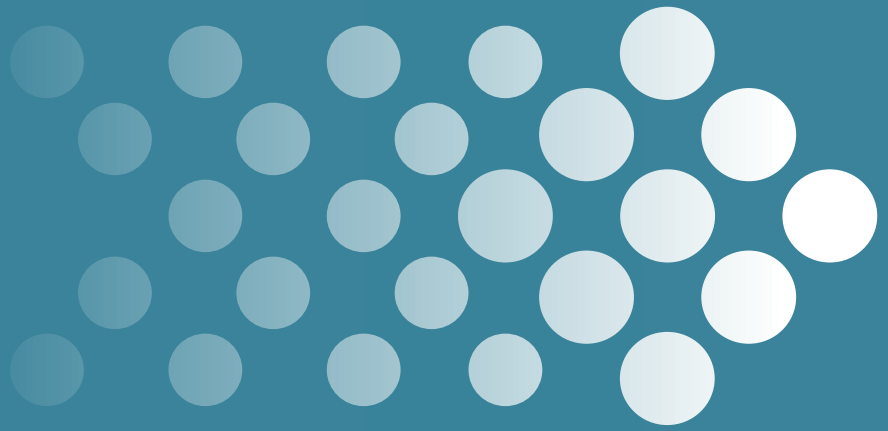
- Previously Chief Regulatory / Quality Officer, **Atara** Biotherapeutics
- Former SVP Regulatory Affairs at **Abraxis** Bioscience
- Worldwide regulatory experience with Abraxane; 25+ years in regulatory affairs

Lynn Samuel
VP Manufacturing and Supply Chain

- Previously SVP Global Supply Chain, **Nant BioScience**
- Former SVP Global Quality Assurance, **ImmunityBio**; VP Manuf. Operations, **NantPharma**;
- Sr. Dir., Nanotechnology Program, **Abraxis** BioScience; led Abraxane manufacturing in >37 countries
- 40+ years in pharmaceutical operations

Andrew Kwon, PhD, MBA
VP BD & Corporate Strategy

- Previously Principal, **Acsel** Health; Director, **Syneos** Health Consulting
- Former Research Scientist at **Memorial Sloan Kettering Cancer Center**
- 14+ years biotech industry experience focused on Business Development and Commercial Strategy in oncology



FYARRO™

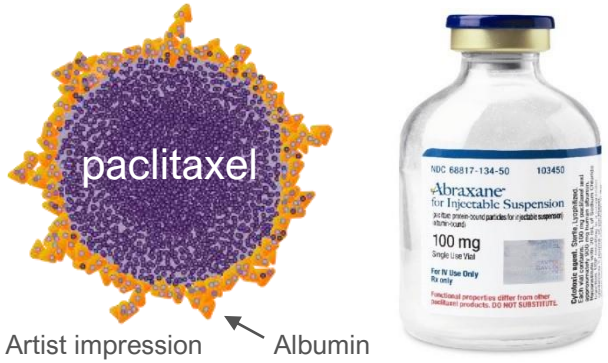
sirolimus protein-bound particles for injectable suspension (albumin bound)

Nanoparticle Albumin-Bound (*nab*) Technology

Nab Platform

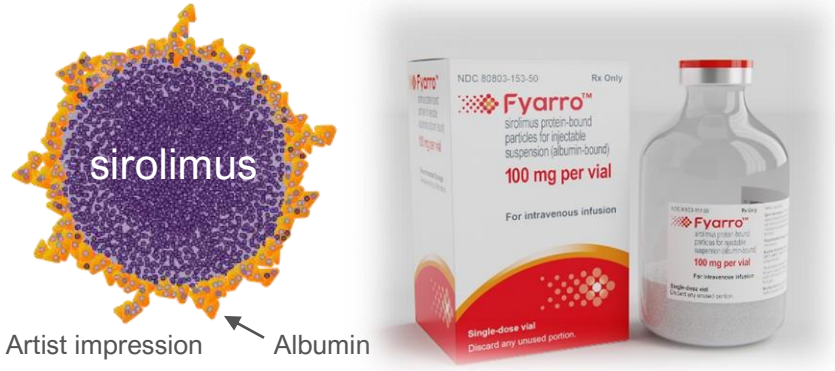
- Proprietary, complex, multi-step manufacturing process with trade secrets

Abraxane®
(*nab*-paclitaxel)



- Superior efficacy[1], safety[1], and PK/PD[2] vs. standard formulation paclitaxel
- Approved for breast cancer, NSCLC, and pancreatic cancer[1]
- Proven efficacy in difficult to treat cancers where paclitaxel is not approved including pancreatic cancer[1]
- Commercially successful with >$1B in annual sales[4]

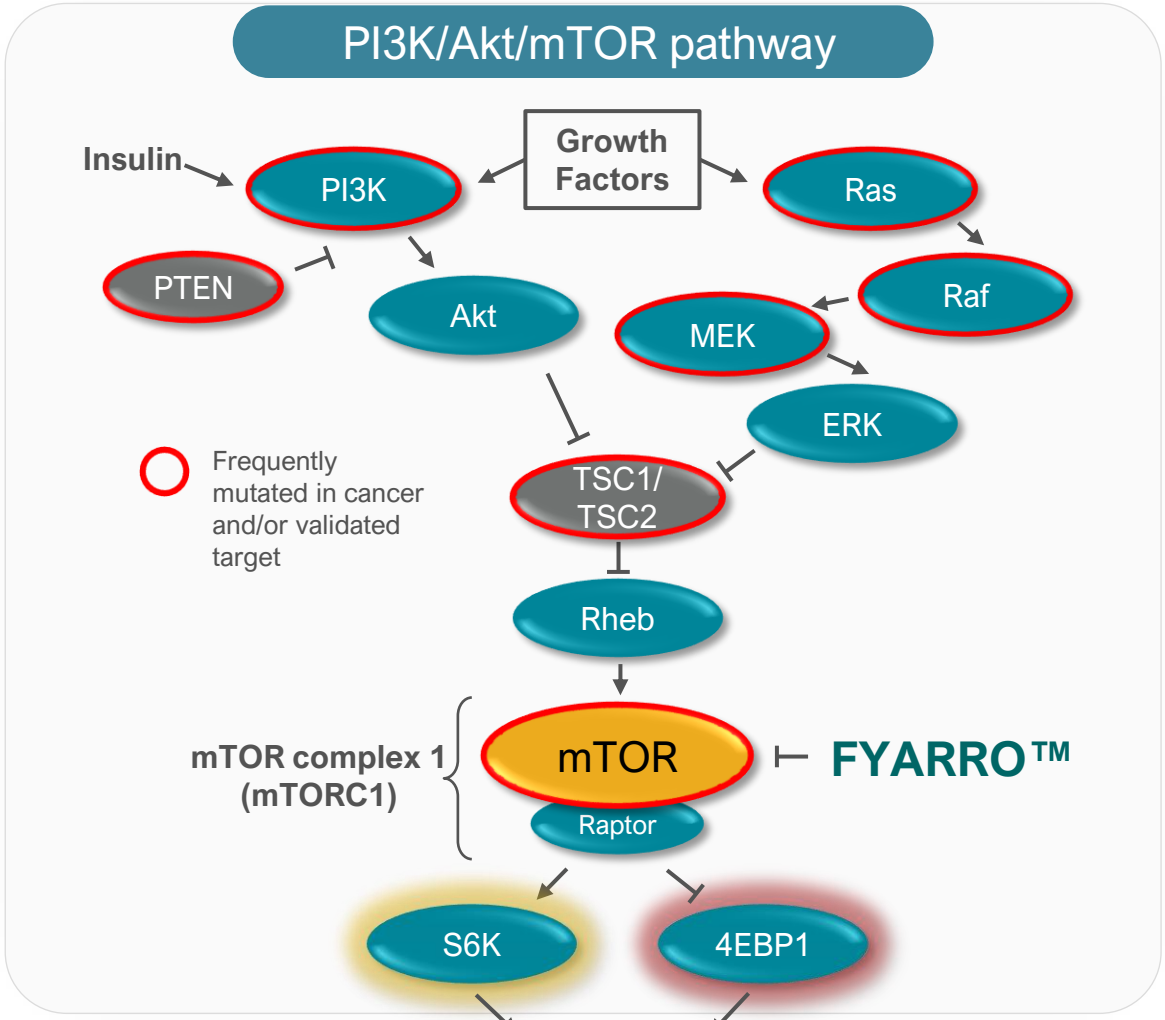
Fyarro™
sirolimus protein-bound particles for injectable suspension (albumin-bound)



- Higher intratumoral drug accumulation, increased target suppression, and stronger tumor growth inhibition in preclinical animal models[5]
- Approved for advanced malignant PEComa
- "*nab*" technology adapted for sirolimus
- Licensed from Celgene in 2014
- WW patent portfolio with issued patents providing coverage to 2036

Note: ABRAXANE® is a registered trademark of Bristol-Myers Squibb Company; Sources: 1) ABRAXANE prescribing information; 2) N Desai et al., Clin Cancer Res. 2006;12(4):1317-1324; 3) EM Hersh et al. Ann Oncol. 2015;26(11)2267-2675; 4) Evaluate Pharma. Accessed April 2021; 5) See following slides

FYARRO™ Targets mTOR, a Key Signaling Pathway in Cancer



FYARRO™ Inhibits Key Signaling Pathways

Limitations of Currently Approved mTOR Inhibitors:

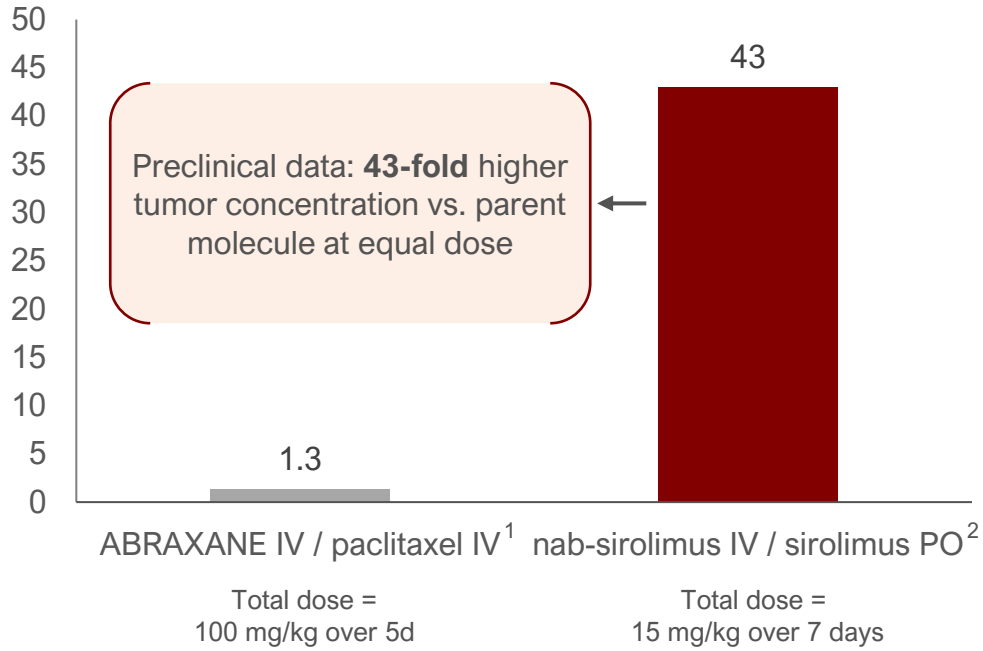
- **Single digit response rates** as monotherapy in oncology[1,2]
- **Poor PK and exposure** in tumor or target tissues resulting in **incomplete target suppression**[3]
- **Highly variable oral absorption** requiring therapeutic monitoring[1,4,5]
- **Narrow therapeutic index** presents challenges for significant dose adjustments[1,2,4,5]
- **Approval in select indications:** cancers[1,2], Tuberous Sclerosis Complex (TSC)[1], and transplant rejection[4,5]

Sources: 1) AFINITOR prescribing information; 2) TORISEL prescribing information; 3) Hou et al., AACR Molecular Targets 2021 (Abstr P138); 4) ZORTRESS prescribing information; 5) RAPAMUNE prescribing information

nab-sirolimus Achieves Larger PK/PD Difference vs Reference Drug

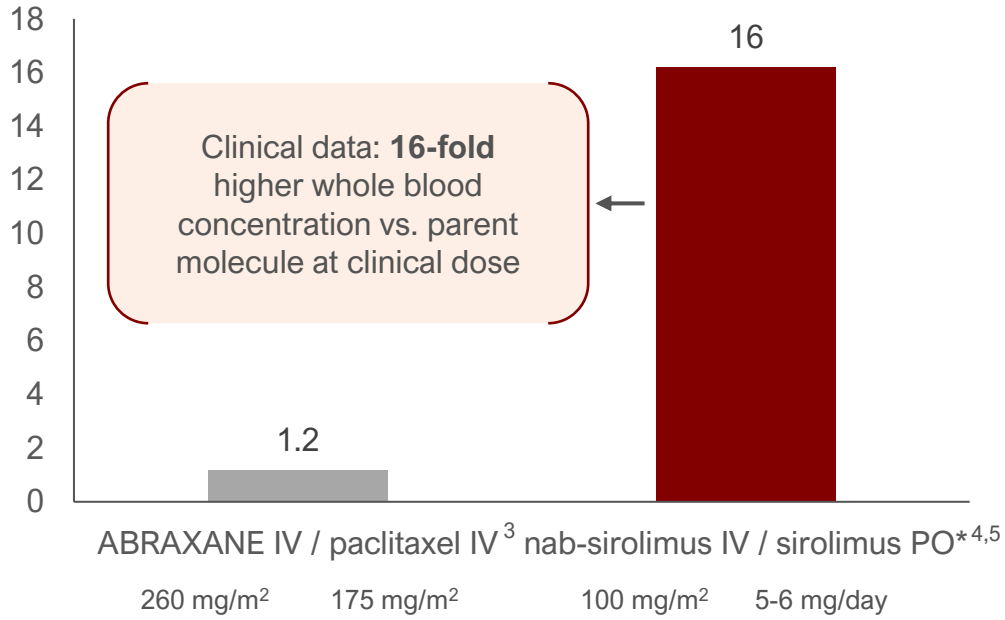
Preclinical Tumor Exposure (AUC)

Fold Change vs. Reference Drug at Equal Dose (Xenograft Model)



Clinical Whole Blood AUC

Fold Change vs. Reference Drug at Clinical Dose (Clinical Data)



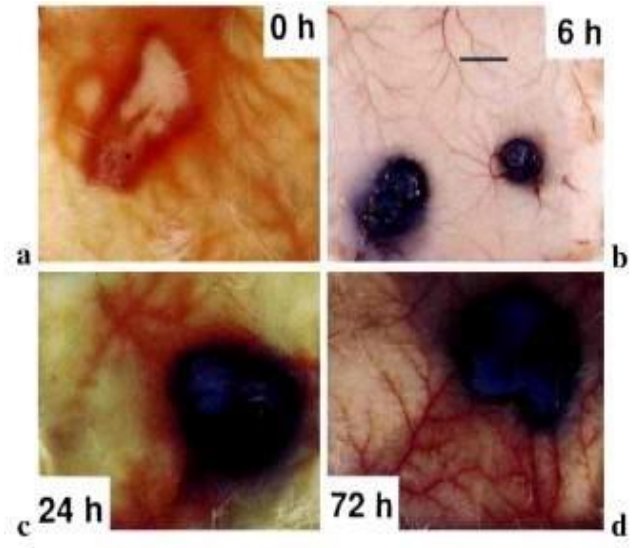
Note: FDA approved dosing & admin schedule used for ABRAXANE, paclitaxel, and sirolimus. PEComa registrational trial dose used for *nab*-sirolimus

* Indirect comparison of *nab*-sirolimus clinical data to published clinical data for sirolimus

Sources: 1) N Desai et al., Clin Cancer Res. 2006;12(4):1317-1324; 2) S Hou et al. AACR 2019; 3) A Sparreboom et al., Clin Cancer Res. 2006;11(11):4136-4143; 4) ABI-009: AM Gonzalez-Angulo et al., Clin Cancer Res 2013;19:5474-5484; 5) Sirolimus: Mean of the following two sources: (a) A Jimeno et al., J Clin Oncol. 2008;26(25):4172-4179. and (b) I Garrido-Laguna et al., Br J Cancer. 2010;103(5):649-655.

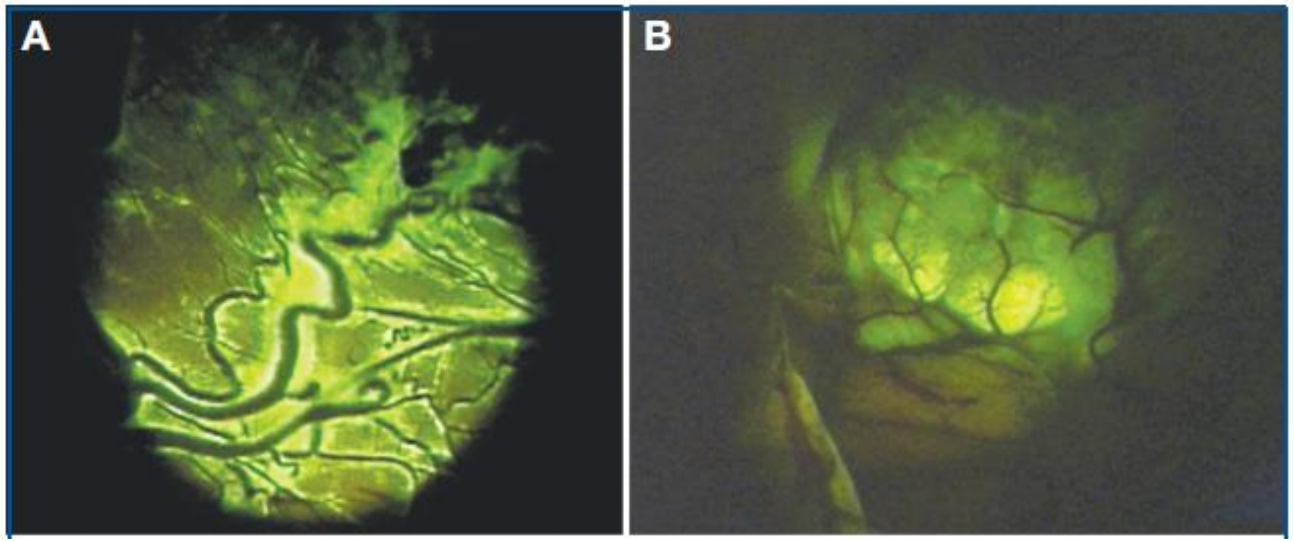
Role of Albumin in Tumor Targeting

Albumin accumulation in tumors established in multiple preclinical models[1]



Accumulation of the evans blue albumin complex in subcutaneously growing sarcoma 180 tumors over 72 h

Labeled albumin can be used intraoperatively to guide surgical resection of tumors in humans[2]



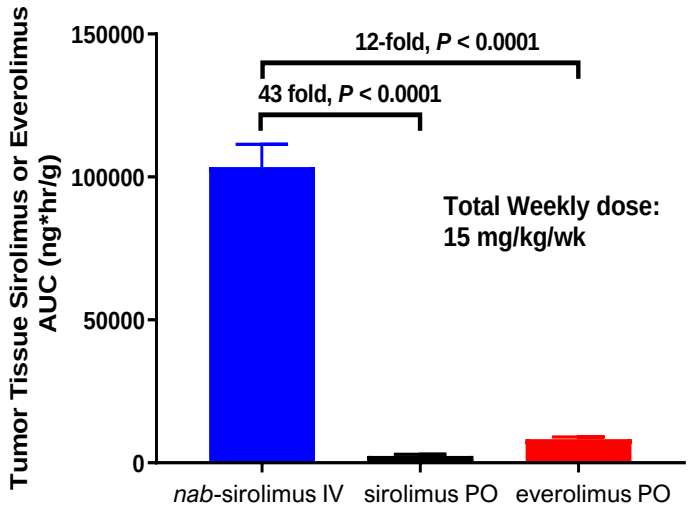
- 5-Amino Fluorescein labelled albumin administered IV (0.5-1 mg/kg) in 13 patients, 0.5-4 days before surgery
- Tumor fluorescence was bright in 11 patients (84%), resulting in complete resection in 9 patients (69%)

High accumulation of albumin in tumors potentially driven by: tumor vessel leakiness (EPR effect); increased caveolar transport; increased albumin catabolism

Note: EPR- Enhanced permeability and retention effect; Sources: 1) Y Shahzad et al., Curr Cancer Drug Targets. 2014;14(8):752-63; 2) P Kremer et al., Neurosurgery. 2009;64(3 Suppl):ons53-60; discussion ons60-1

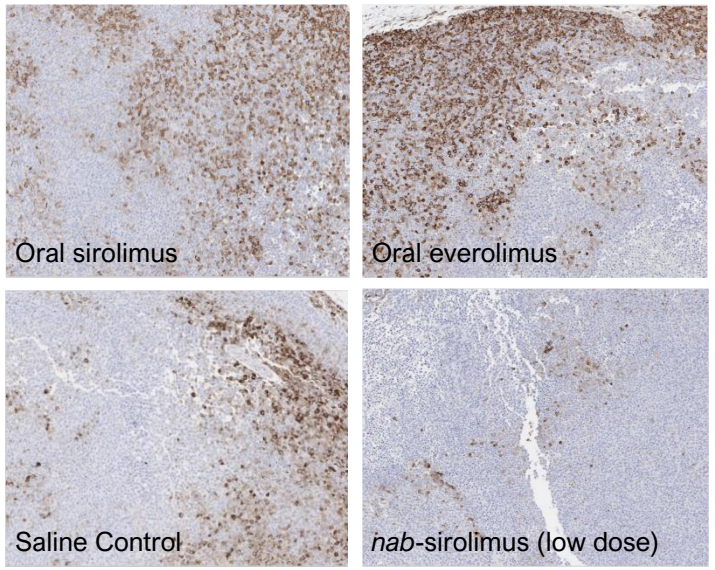
Higher *nab*-sirolimus Intratumoral Concentrations Drive Increased Target Suppression and Tumor Growth Inhibition in a Bladder Cancer Xenograft (UMUC3)

Significantly Higher Intratumoral Drug Accumulation



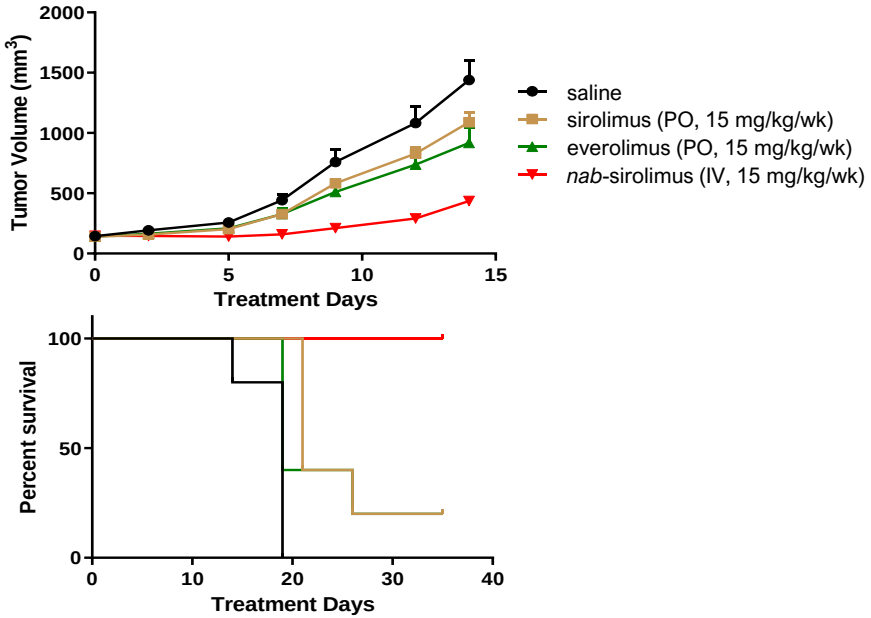
Tumor concentration of *nab*-sirolimus, oral sirolimus, and oral everolimus measured over 7 days at equal weekly dose (15 mg/kg/wk) in mice bearing tumor xenografts

Increased mTOR Target Suppression (pS6)



Tumor IHC pS6 suppression on D7 post dose at equal doses (15 mg/kg/wk).
pS6 is a downstream target of mTOR.
nab-sirolimus vs oral sirolimus: P = 0.0001 (ANOVA)
nab-sirolimus vs oral everolimus P = 0.0034 (ANOVA)

Stronger Inhibition of Tumor Growth and Longer Survival in Animals



UMUC3 (aggressive human bladder cancer) Xenograft (n=8/group):
Oral Rapamycin and Everolimus 15 mg/kg/wk (3 mg/kg, 5x/wk);
IV *nab*-sirolimus 15 mg/kg/wk (7.5 mg/kg, 2x/wk)
Tumor volume: *nab*-sirolimus vs oral sirolimus: P < 0.0001 (ANOVA)
nab-sirolimus vs oral everolimus P = 0.0023 (ANOVA)
Survival: *nab*-sirolimus vs oral sirolimus: P < 0.05 (Log-rank test)
nab-sirolimus vs oral everolimus P < 0.05 (Log-rank test)

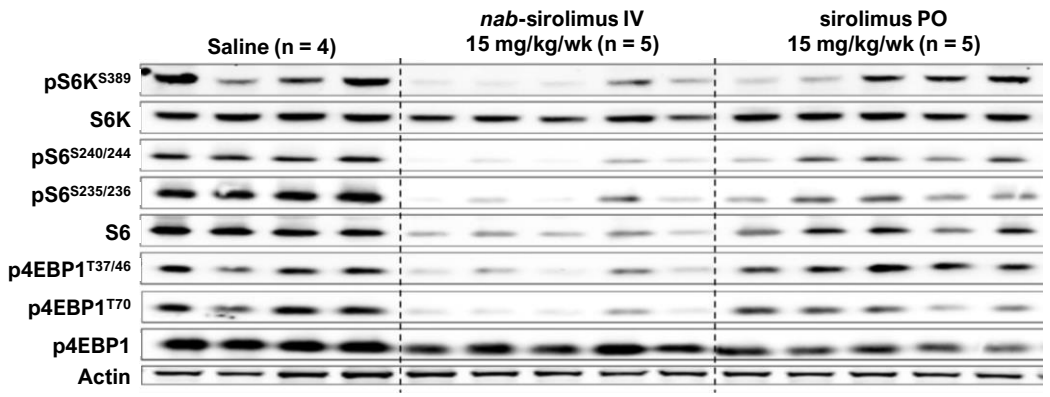
nab-sirolimus demonstrated enhanced anti-tumor activity vs. currently approved mTOR inhibitors in animal models at clinically relevant doses.

Source: S Hou et al., AACR. 2019

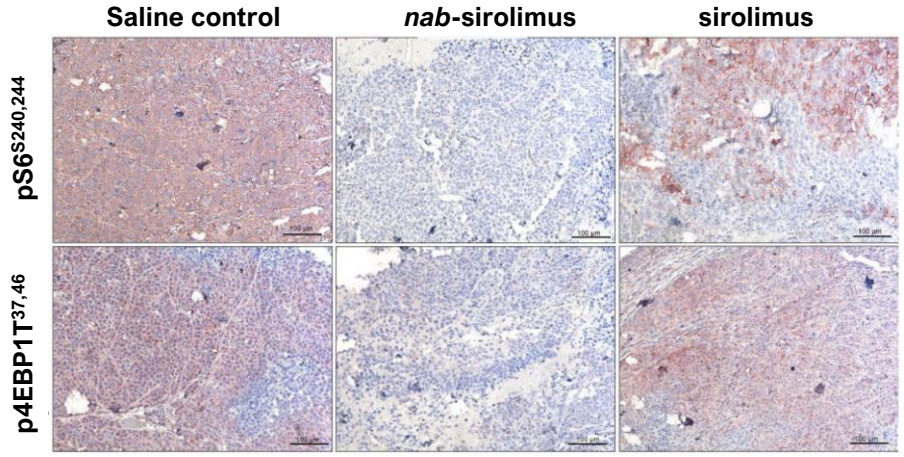
Improved mTOR Pathway Inhibition and Tumor Growth Inhibition in *TSC2*-null, Human Liver Cancer Xenograft Model (SNU398)

Increased Suppression of mTOR Targets S6K and 4EBP1

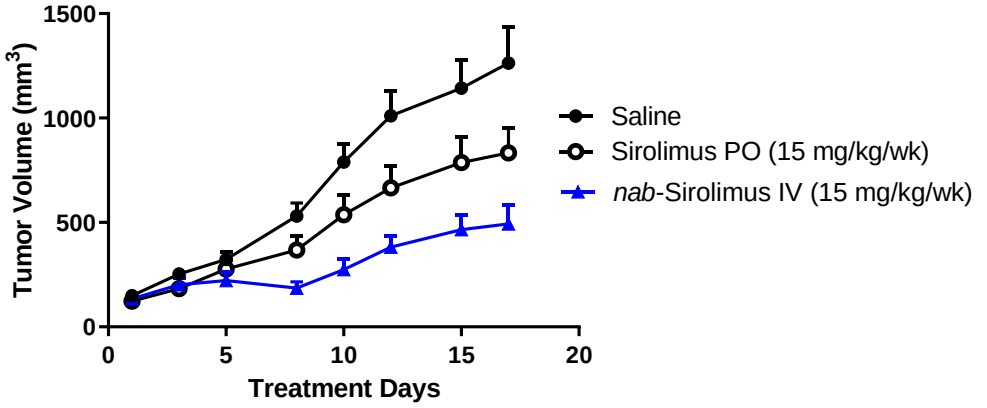
Western Blot for pS6K, pS6 and p4EBP1

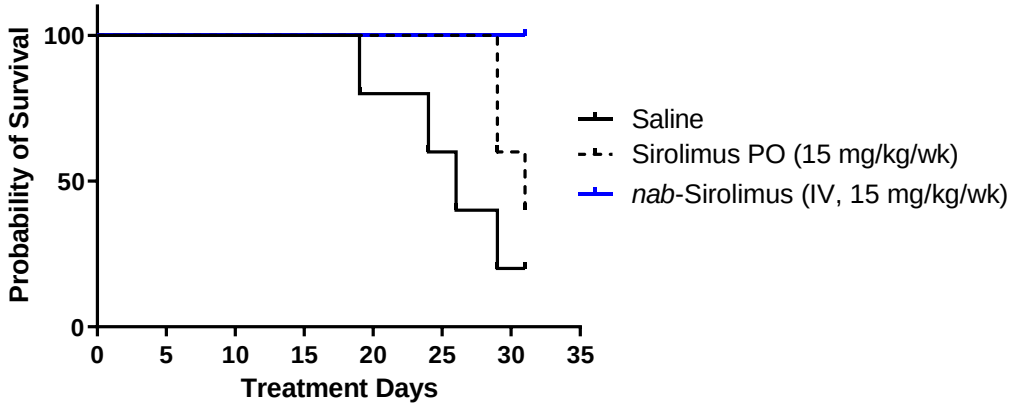


IHC for pS6 and p4EBP1



Stronger Inhibition of Tumor Growth and Longer Survival in Animals



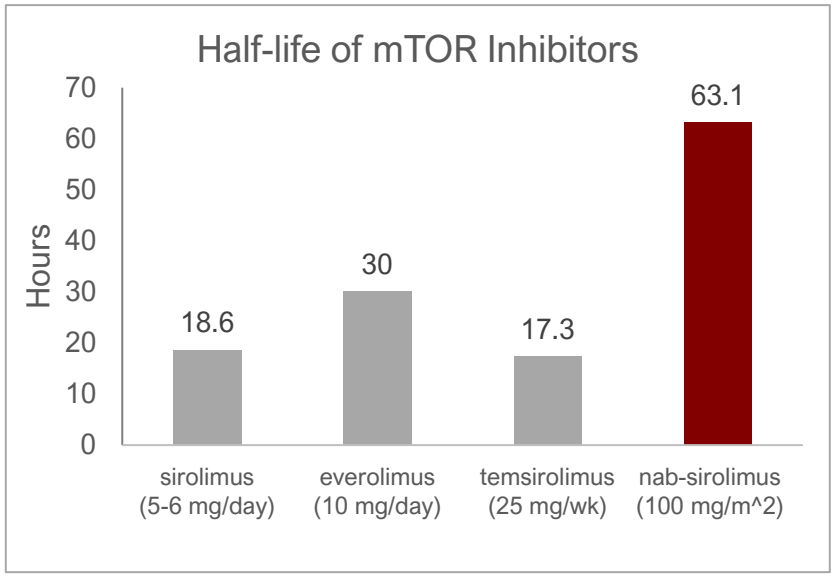


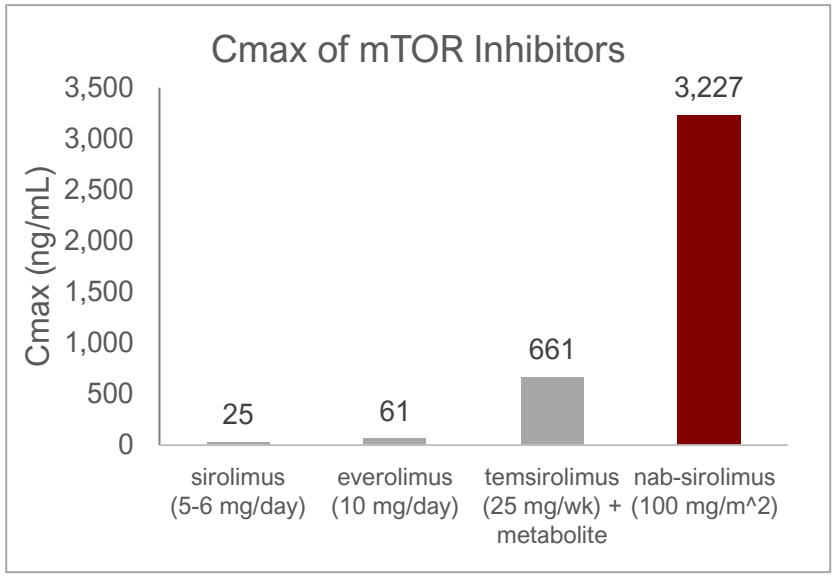
- Tumor volume: *nab*-sirolimus vs oral sirolimus: TGI 67.8% vs 36.2%, $P < 0.05$ (ANOVA)
- Survival: *nab*-sirolimus vs oral sirolimus: $P < 0.05$ (Log-rank test)

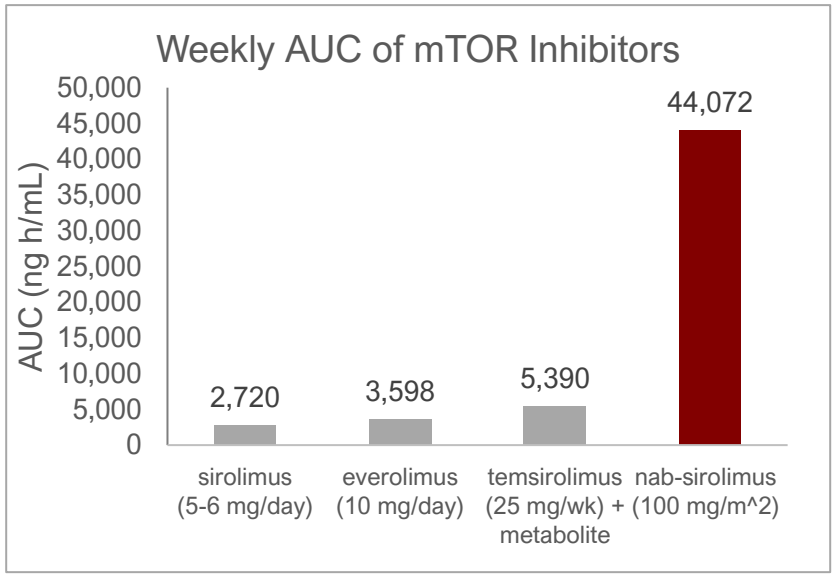
Sources: Hou et al., AACR Molecular Targets 2021 (Abstr P138)]

Differentiated Clinical PK Profile Compared to Other mTOR Inhibitors

PK Comparison at Clinical Doses







nab-sirolimus achieves higher AUC, Cmax and longer half-life in humans at its clinical dose when compared with published clinical data for other mTOR inhibitors.

Sources: 1) Mean of the following two sources: (a) A Jimeno et al., J Clin Oncol. 2008;26(25):4172-4179. and (b) I Garrido-Laguna et al., Br J Cancer. 2010;103(5):649-655; 2) R Danesi et al., Cancer Treatment Reviews. 2013;39:784–792; 3) ABI-009: AM Gonzalez-Angulo et al., Clin Cancer Res 2013;19:5474-5484.

FYARRO™ Advanced Oncology Development Pipeline

Investigational Uses

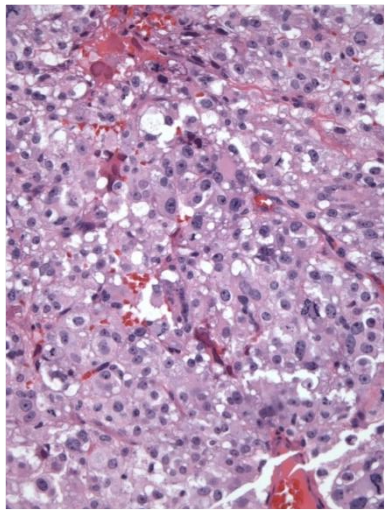
Populations	Phase 1b	Phase 2	Registrational	Approved	Current Status
Advanced Malignant PEComa	**Single Agent**				First FDA approved therapy for advanced malignant PEComa
Pan-Tumor *TSC1* Inactivating Alterations	**Single Agent**				Planned tumor-agnostic pivotal study with independent arms for *TSC1* or *TSC2* inactivating mutations; trial to be initiated by the end of 2021 or early 2022
Pan-Tumor *TSC2* Inactivating Alterations	**Single Agent**				
Expanded Access Program (Solid Tumors ± mTOR Pathway Alterations)	**Single Agent**				Provide access to ABI-009 prior to commercial availability to patients with PEComa or solid tumors with mTOR pathway mutations
Dose Finding Combination Studies (Multiple)					Continue ongoing combination partner trials and initiate new trials with adjacent pathway combinations that may be synergistic
Undisclosed indication: single agent expansion					Expand into a sub-population with strong mTOR mechanistic rationale

Ongoing To be initiated Planned/ initial stages

Locally Advanced Unresectable or Metastatic Malignant Perivascular Epithelioid Cell Tumor (PEComa)

PEComa Disease Overview and Current Standard of Care

Advanced, Malignant PEComa



- Ultra rare sarcoma
- Mesenchymal tumor (sarcoma) consisting of perivascular epithelioid cells
- Distinctive cells that show a focal association with blood-vessel walls[1]
- Usually express both melanocytic and smooth muscle markers[1]
- Biological evidence of mTOR pathway activation; cancer type with highest rate of *TSC1* & *TSC2* mutations[2-4]
- Can arise at any site but most commonly at visceral (especially gastrointestinal and uterine), retroperitoneal, and abdominopelvic sites and with female predominance
- Estimated survival of 12-16 months[5]
- Estimated 100-300 new patients per year in the US[6]

Current Standard of Care

- No approved treatments and no prior clinical trials conducted
- Retrospective data supports use of mTOR inhibition
- Often misdiagnosed and treated with other sarcoma treatments

Currently Used (Unapproved) Treatments

Chemotherapy[7] (e.g., doxorubicin and ifosfamide)	mTOR Inhibitor[7] (e.g., everolimus, sirolimus)
Standard sarcoma treatment; more frequently used in community setting despite minimal efficacy	More frequently used in academic setting and by high volume community treaters

Sources: 1) Ben-Ami et al., Expert Opinion on Orphan Drugs. 2018; 2) Akumalla S, et al. Oncology. 2020;98(12):905-912; 3) nab-Sirolimus AMPECT Clinical Trial mutation rates: TSC1=20%, TSC2=36%; 4) Mutation frequencies based on TCGA database "likely" and "definite" impact mutation rate and published literature rates by cancer type where available (sources available at request); 5) JS Bleeker, JF Quevedo, and AL Folpe, Sarcoma. 2012;541626; 6) No formal published epidemiology information; Aadi analysis based on multiple sources including Aadi internal data and external research conducted by Tessellon Group and Corsica Life Sciences 7) Primary Oncologist Market Research (N=10) conducted July and August 2019 by Corsica Life Sciences

AMPECT PEComa Registrational Trial Met its Endpoints

AMPECT PEComa Phase II Registrational Trial Design

Advanced Malignant PEComa Patients (mTOR naïve)	ABI-009 100 mg/m^2 IV D1,8 q 21d until progression or unacceptable toxicity	Primary Endpoint: **ORR** Secondary Endpoints: DOR, PFS at 6m, mPFS, mOS, Safety

Sample Size: **Target ORR of ~30% in 30 evaluable patients** to exclude the lower bound of the 95% CI of 14.7%

FDA Orphan, Fast-Track, and Breakthrough Therapy Designations granted

ASCO Journal of Clinical Oncology®

***nab*-Sirolimus for Patients With Malignant Perivascular Epithelioid Cell Tumors**

Andrew J. Wagner, MD, PhD[1]; Vinod Ravi, MD[2]; Richard F. Riedel, MD[3]; Kristen Ganjoo, MD[4]; Brian A. Van Tine, MD, PhD[5]; Rashmi Chugh, MD[6]; Lee Cranmer, MD, PhD[7]; Erlinda M. Gordon, MD[8]; Jason L. Hornick, MD, PhD[9]; Heng Du, MD[9]; Berta Grigorian, BS[10]; Anita N. Schmid, PhD[10]; Shihe Hou, PhD[10]; Katherine Harris, DrPH[10]; David J. Kwiatkowski, MD, PhD[9]; Neil P. Desai, PhD[10]; and Mark A. Dickson, MD[11]

†JCO: 1.5-year follow-up, data cut-off Nov 2020[1]

Efficacy Results in AMPECT[1]	Independent Radiology Review
Overall Response Rate (95% CI)	**39%** (22%, 58%)
Duration of Response (DOR)	**(N=12)**
Median (95% CI) in months	NR (6.5, NE)
Range in months	5.6-55.5+
DOR rate at 6 months	92%
DOR rate at 12 months	75%
DOR rate at 24 months	66%

Additional Findings from AMPECT Trial*[2]	Independent Radiology Review
Complete Response[1,2]	7% (2/31)
Partial Response[2]	32% (10/31)
Stable Disease[2]	52%
Progressive Disease[2]	10%
Disease Control Rate‡[2]	71%
Median Duration of Response[2]	**>36 months**
Median Progression Free Survival[2]	**10.6 months (5.5-NR)**
Median Overall Survival†[3]	**40.8 months (22.2-NR)**

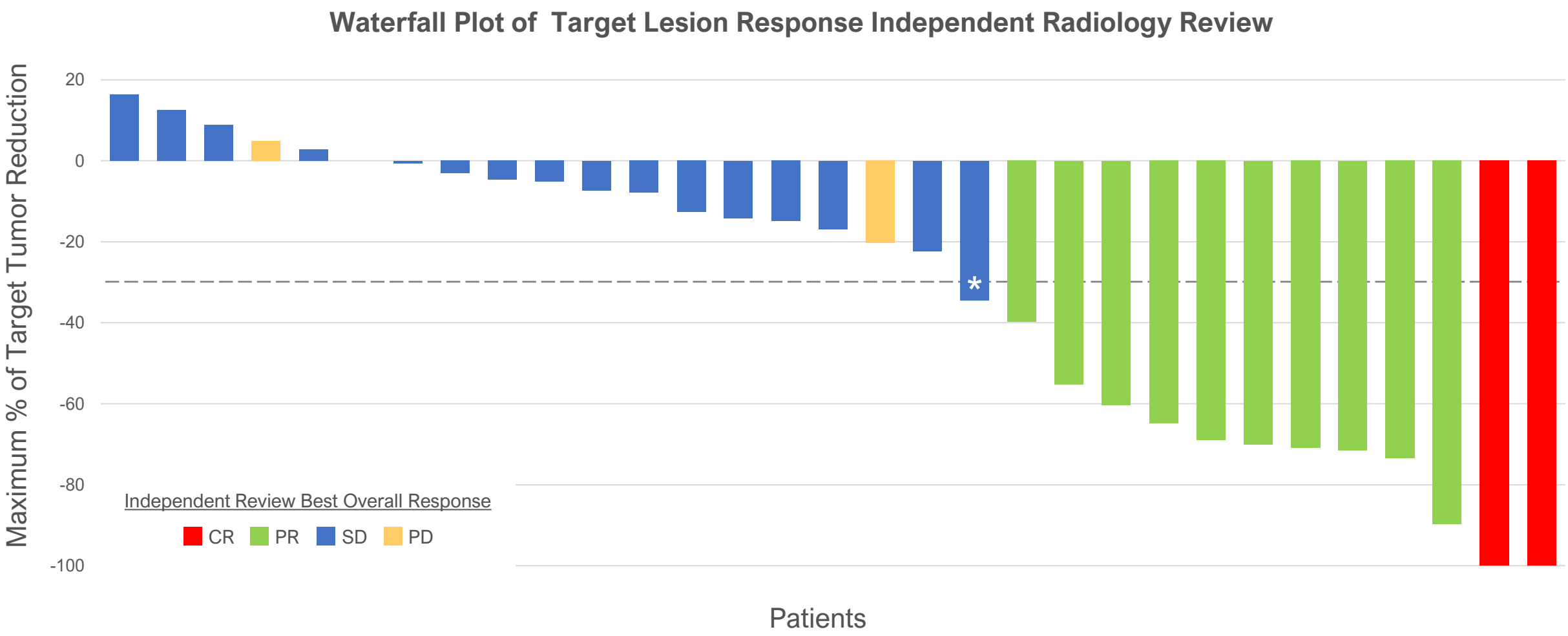
*At 2.5-year follow-up, data cut-off June 30, 2021[2]

- 2 patients converted from a PR to complete response (CR) during the follow-up period, after 11mo and 34mo of treatment each
- mDOR has not been reached, 50% of patients had a DOR of 36.1+ months

The AMPECT Trial met its primary endpoint, exceeding the 30% target ORR agreed upon by the FDA, resulting in approval of FYARRO™ as the first and only therapy specifically indicated for advanced malignant PEComa

Note: ‡Disease control rate defined as complete response + partial response + stable disease ≥12 weeks; Sources: 1) FYARRO™ Prescribing Information; 2) AJ Wagner, CTOS 2021; 3) AJ Wagner, JCO 2021

AMPECT Response Assessment



Note: * 1 patient had an unconfirmed partial response and thus best response is stable disease as per RECIST v1.1; Source: AJ Wagner, CTOS 2021

FYARRO™ Safety Summary

Select Adverse Reactions ≥10% in Patients with PEComa Who Received FYARRO in AMPECT*		
	FYARRO (n=34)	
Adverse Reaction	All Grades (%)	Grade 3 to 4* (%)
Stomatitis	79	18
Fatigue	68	2.9
Rash	68	0
Infections	59	0
Edema	50	2.9
Nausea	50	0
Diarrhea	47	2.9
Musculoskeletal pain	47	2.9
Weight decreased	47	0
Decreased appetite	44	0
Cough	35	0
Dysgeusia	32	0
Vomiting	32	2.9
Abdominal pain	29	6
Headache	29	0
Hypertension	29	2.9
Pneumonitis	18	0

Serious Adverse Reactions	n (%)
Patients with Any Serious Adverse Reaction	14 (41)
Serious Adverse Reaction in ≥5% of Patients	
Infection	4 (12)
Abdominal pain	2 (6)
Dehydration	2 (6)
Upper gastrointestinal hemorrhage	2 (6)

- No drug-related grade 4 Adverse Events and no drug-related deaths
- No unexpected treatment-related AEs
- Pneumonitis 6/34 (18%), G1/G2 only
- Discontinuation due to AE: 3/34 (9%) patients (pneumonitis, anemia, noninfective cystitis)
- Dose reductions occurred in 13/34 (38%) of patients; 11 patients had a dose reduction from 100 mg/m^2 to 75 mg/m^2 and 2 patients had a dose reduction to 56 mg/m^2

Note: *See FYARRO™ prescribing information for complete list of Adverse Reactions

PEComa Commercial Strategy and Launch Plan

PEComa Launch Strategic Imperatives

- Establish FYARRO™ as a standard of care for malignant PEComa
- Ensure HCPs have a positive first clinical experience with FYARRO™
- Entrench Aadi as a recognized leader in precision oncology

Target PEComa Patients at Major Centers

- Majority of PEComa patients treated at top cancer centers
- Focus on high-volume sarcoma centers including NCCN member institutions and NCI designated cancer centers

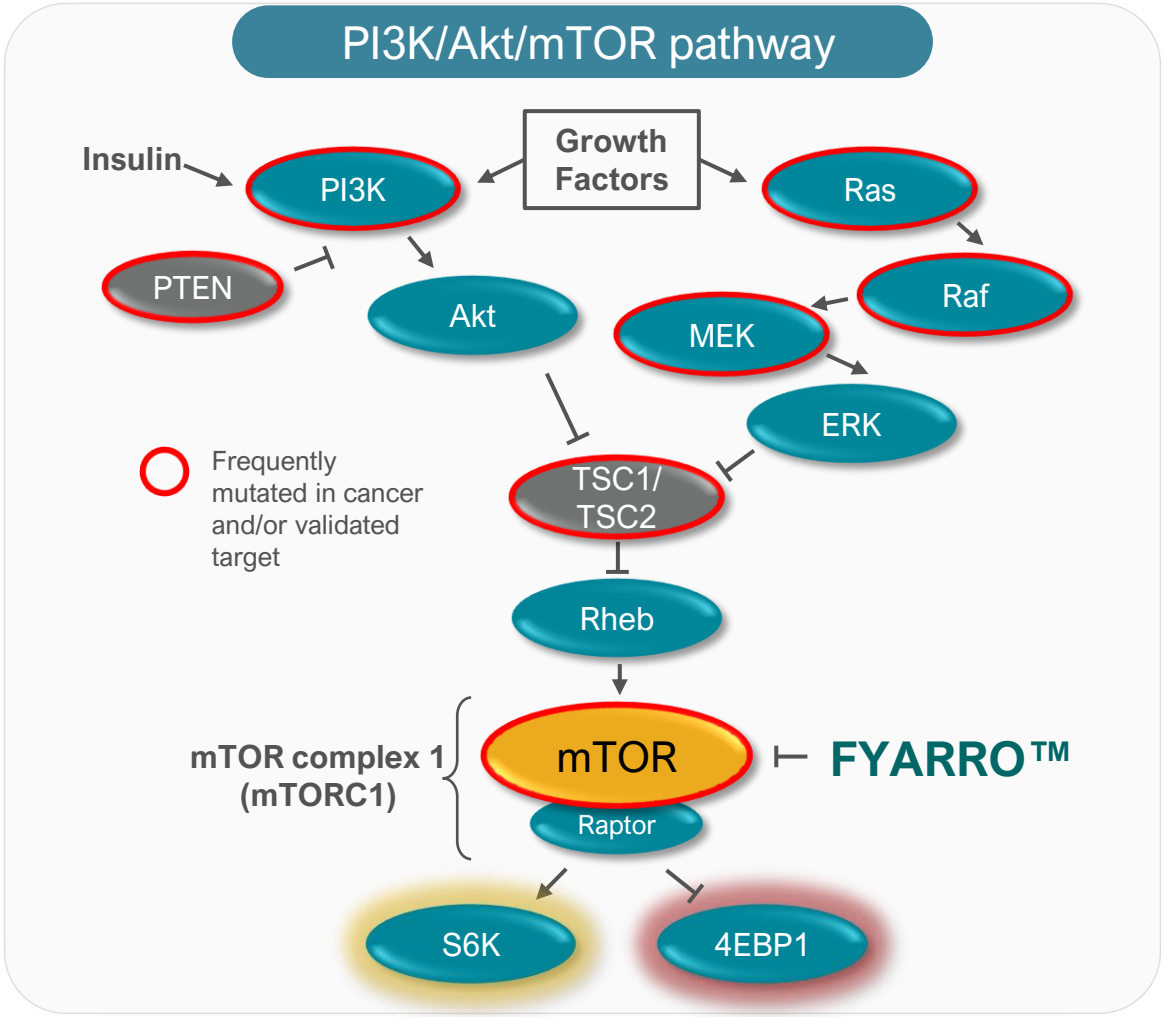
Build a Lean but Effective Commercial Infrastructure

- Commercial infrastructure build out ongoing including field team (e.g., sales reps, account managers, etc.)
- Recently appointed **Chief Operating Officer, Brendan Delaney**, was previously CCO of Immunomedics and led the launch of TRODELVY® prior to Gilead acquisition
- On track for Q1 2022 commercial launch

Note: NCCN- National Comprehensive Cancer Network; NCI- National Cancer Institute

Rationale for Clinical Development in Tumors with *TSC1* and *TSC2* Inactivating Alterations

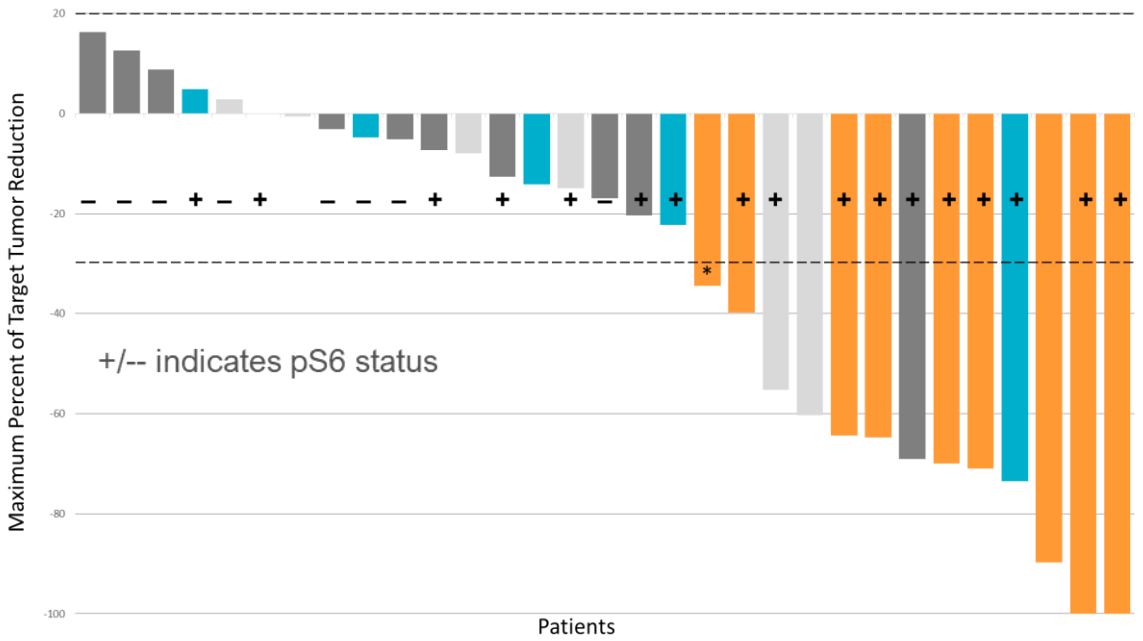
TSC1 and *TSC2* Mutations: Key Oncogenic Drivers in the mTOR Pathway

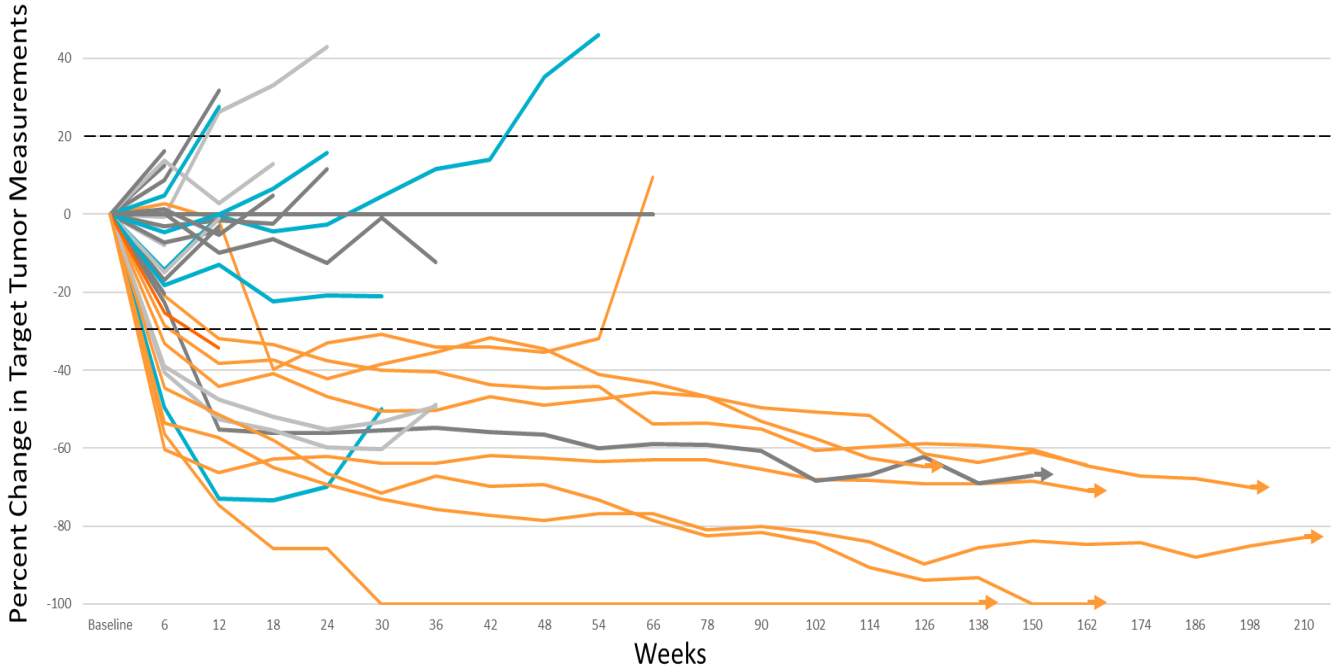


Inactivating mutations in *TSC1* and *TSC2* drive mTOR pathway activation and tumor growth

- TSC1 and TSC2 are upstream regulators of mTOR activity within the PI3K/Akt/mTOR pathway
- *TSC1* and *TSC2* mutations occur at a rate of approximately 1-2% each across cancers
- No approved therapies for *TSC1* and *TSC2* mutant patients but numerous case reports with durable responses to mTOR inhibition
- Standard CLIA-certified NGS panels already capture *TSC1* and *TSC2* mutations

Exploratory Analysis in AMPECT in *TSC1* or *TSC2* Inactivating Alterations Supports Further Investigation





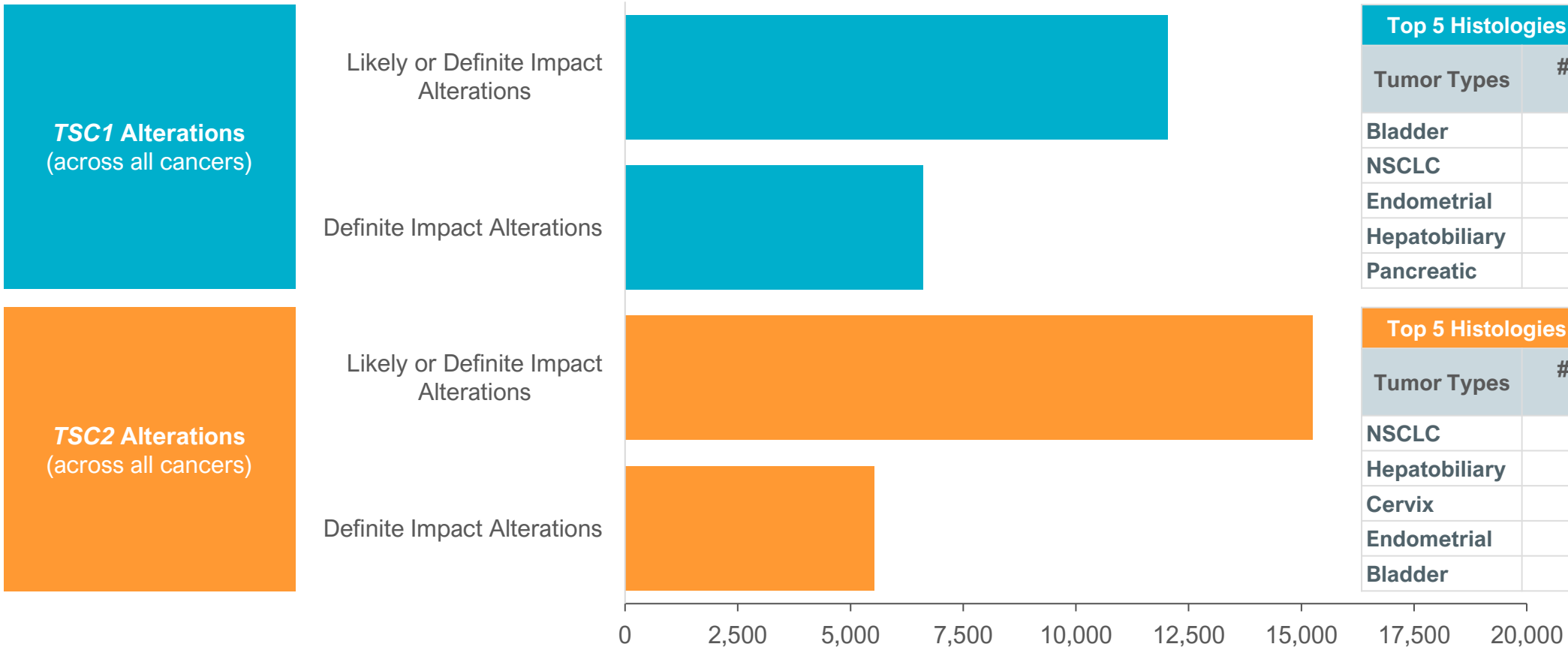
TSC2 mutation
TSC1 mutation
No *TSC1* or *TSC2* mutation
UNK mutational status

Note: *1 patient with *TSC2* mutation had an unconfirmed PR and thus best response is an SD as per RECIST 1.1; Source: AJ Wagner et al., JCO. 2021

TSC1 and *TSC2* Inactivating Alterations Represent Significant Opportunities

***Projected Annual Incidence of Cancers with TSC1* and *TSC2* Alterations**

Estimated US Patients Available for 1st Line Therapy in 2030



Top 5 Histologies by # of *TSC1* Patients	
Tumor Types	**# with Definite Impact Mutations (%)**
Bladder	1,772 (6.33%)
NSCLC	1,297 (0.77%)
Endometrial	835 (2.10%)
Hepatobiliary	445 (1.27%)
Pancreatic	344 (0.57%)

Top 5 Histologies by # of *TSC2* Patients	
Tumor Types	**# with Definite Impact Mutations (%)**
NSCLC	1,945 (1.16%)
Hepatobiliary	1,157 (3.31%)
Cervix	808 (0.71%)
Endometrial	487 (1.22%)
Bladder	477 (1.70%)

Definitions:
Likely Impact Alterations (harmful missense variants): missense mutations predicted to be deleterious by SIFT or possibly or probably damaging by PolyPhen
Definite Impact Alterations (truncating and deep deletions): out-of-frame frameshift insertions/deletions, nonsense mutations, splice-site mutations, and deep deletions (e.g., copy number "-2" in cBioPortal)

Source: analysis of TCGA, cBioPortal, and SEER databases conducted by Tessellon Group in June 2021

Early Experience in Non-PEComa Patients with *TSC1* or *TSC2* Inactivating Alterations from Expanded Access Program (presented at ASCO 2021[1])

- Study Design: **Multi-institutional Expanded Access for an Intermediate-size Population**
- N=8 patients with *TSC1* or *TSC2* inactivating alterations
  - 6 mTOR-naïve
  - 2 previously treated with an mTORi
- 100 mg/m^2 ABI-009 (*nab*-sirolimus) given D1, D8 of a 21-day cycle
- Response Analysis: RECIST v1.1

Patient Population

Disease	# Prior Rx	Prior mTORi	Lis of Prior Rx	Mut	Pt #
Endometrial Stromal Sarcoma	3	No	EXE, LET, FUL	*TSC2*	1
Ovarian Cancer	4	No	CIS+PAC, BEV, PAR, CAR, LDX+GEM	*TSC1*	2
Angiosarcoma	2	No	DOX+IFO+MES, PAC	*TSC1*	4*
Leiomyosarcoma	4	No	DOX+OLA, TRA, GEM+DOC, ERI+PEM	*TSC2*	8
Lymphangioleiomyoma¥	0	No	None	*TSC2*	6
Endometrial Cancer	3	Yes	ANA, LEU, SIR	*TSC2*	5§
High-grade Sarcoma	5	No	DOX+IFO, GEM+DOC, GEM, PAZ, PEM+DEN	*TSC2*	7
Ovarian Cancer	6	Yes	LDX, CAR, BEV, GEM, ENZ, MLN-0128	*TSC2*	3§

Drug Abbreviations: ANA = anastrozole; BEV = bevacizumab; CAR = carboplatin; CIS = cisplatin; DEN = denosumab; DOC = docetaxel; DOX = doxorubicin; ENZ = enzalutamide; ERI = eribulin; EXE = exemestane; FUL = fulvestrant; GEM = gemcitabine; IFO = ifosfamide; L = liposomal; LET = letrozole; LEU = leuprolide; MLN-0128 = sapanisertib (mTORi); OLA = olaratumab; PAC = paclitaxel; PAZ = pazopanib; PEM = pembrolizumab; SIR = sirolimus (mTORi); TRA = trabectedin

* After initial SD, Pt# 4 had treatment break due to infection/surgery/healing, totaling ~ 2.5 months. Subsequent imaging showed PR in target lesions along with new lesions. The patient resumed therapy with ongoing benefit

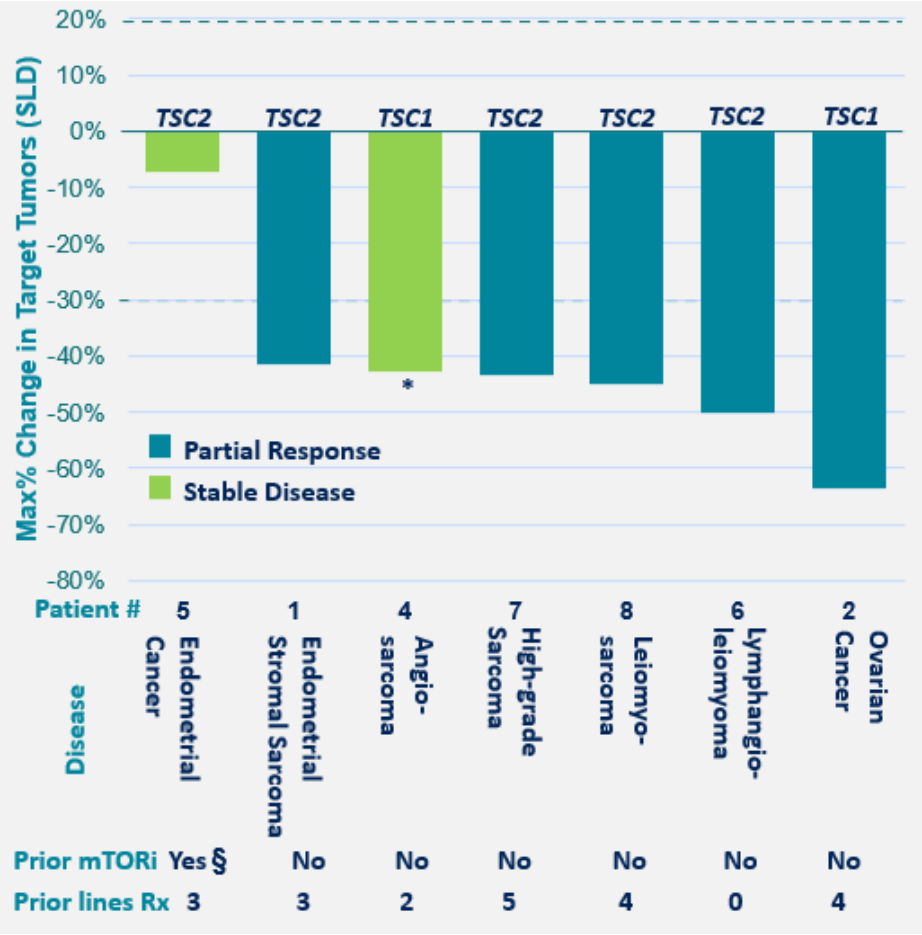
§ Progressed on mTORi prior to receiving ABI-009

¥ Pt #6 had a progressive retroperitoneal mass

Source: 1) MA Dickson. ASCO. 2021. Abstract # 3111

Early Experience in Non-PEComa Patients with *TSC1* or *TSC2* Inactivating Alterations from Expanded Access Program (presented at ASCO 2021[1])

Efficacy: 8 patients treated, 7 evaluable for response



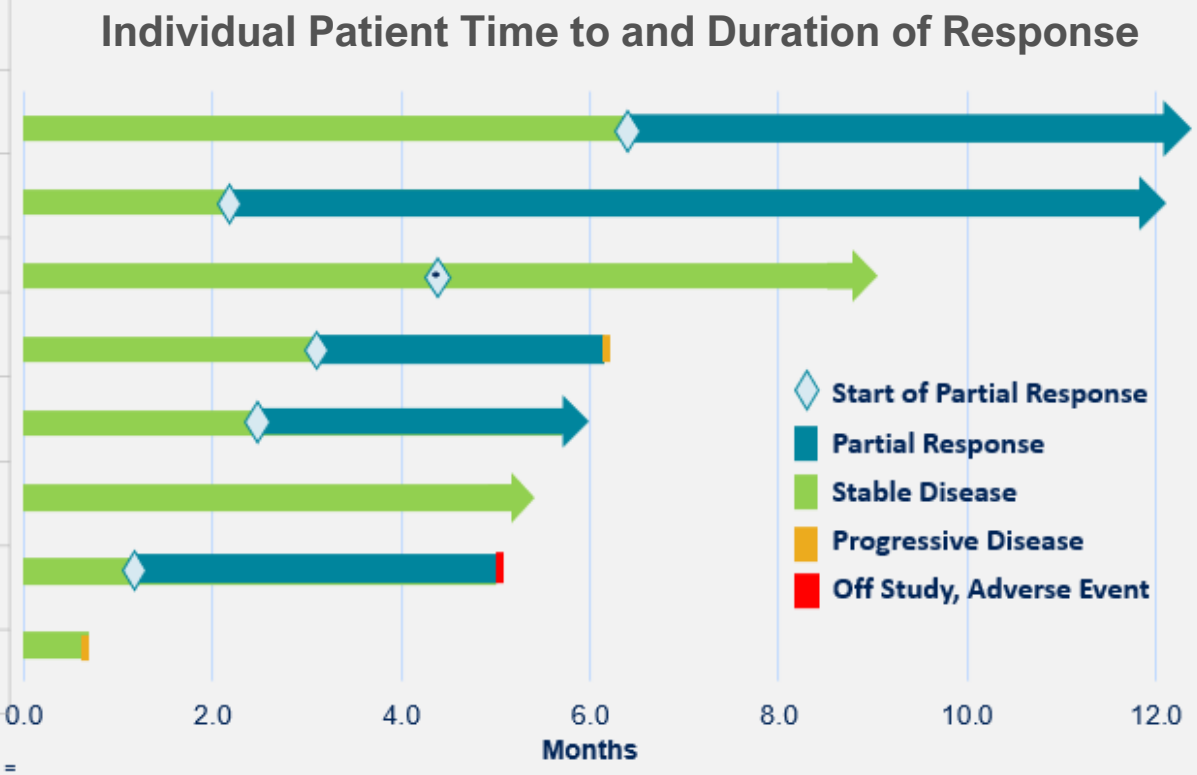
Safety: 8 patients evaluable for adverse events

- Treatment-emergent AEs (≥30%) included edema, infections, mucositis, and pain (71% each), nail changes and vomiting (57% each), and hypertension and nausea (43% each).
- Majority of events were G1/G2
- Treatment-related SAEs were reported in 2 patients and included hyperglycemia and infection (Pt#4) and acute kidney injury (Pt#7) possibly secondary to administration of contrast
- Dose reductions occurred in 3/8 patients (38%) from 100 mg/m^2 to 75 mg/m^2

Source: 1) MA Dickson. ASCO. 2021. Abstract # 3111

Early Experience in Non-PEComa Patients with *TSC1* or *TSC2* Inactivating Alterations from Expanded Access Program (presented at ASCO 2021[1])

Disease	# Prior Rx	Prior mTORi	List of Prior RX	Mut	Pt #
Endometrial Stromal Sarcoma	3	No	EXE, LET, FUL	*TSC2*	1
Ovarian Cancer	4	No	CIS+PAC, BEV, PAR, CAR, LDX+GEM	*TSC1*	2
Angiosarcoma	2	No	DOX+IFO+MES, PAC	*TSC1*	4 *
Leiomyosarcoma	4	No	DOX+OLA, TRA, GEM+DOC, ERI+PEM	*TSC2*	8
Lymphangioleio-Myoma ¥	0	No	none	*TSC2*	6
Endometrial Cancer	3	Yes	ANA, LEU, SIR	*TSC2*	5 §
High-grade Sarcoma	5	No	DOX+IFO, GEM+DOC, GEM, PAZ, PEM+DEN	*TSC2*	7
Ovarian Cancer	6	Yes	LDX, CAR, BEV, GEM, ENZ, MLN-0128	*TSC2*	3 §



Drug Abbreviations: ANA = anastrazole; BEV = bevacizumab; CAR = carboplatin; CIS = cisplatin; DEN = denosumab; DOC = docetaxel; DOX = doxorubicin; ENZ = enzalutamide; ERI = eribulin; EXE = exemestane; FUL = fulvestrant; GEM = gemcitabine; IFO = ifosfamide; I = liposomal; LET = letrozole; LEU = leuprolide; MLN-0128 = sapanisertib (mTORi); OLA = olaratumab; PAC = paclitaxel; PAZ = pazopanib; PEM = pembrolizumab; SIR = sirolimus (mTORi); TRA = trabectidine;

* After initial SD, Pt# 4 had treatment break due to infection/surgery/healing, totaling ~ 2.5 months. Subsequent imaging showed PR in target lesions along with new lesions. The patient resumed therapy with ongoing benefit

§ Progressed on mTORi prior to receiving ABI-009

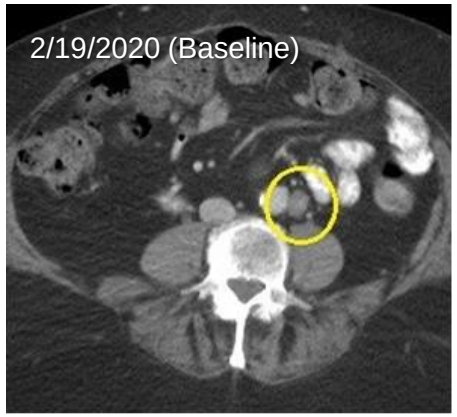
¥ Pt #6 had a progressive retroperitoneal mass

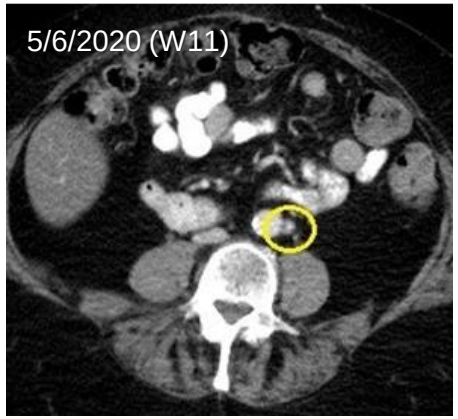
Note: everolimus study[2] in *TSC1*/*TSC2* inactivating alterations showed 7% (2/30) responses

1) MA Dickson. ASCO. 2021. Abstract # 3111; 2) Adib et al . Clinical Can Res 2021 Jul 15;27(14):3845-3853

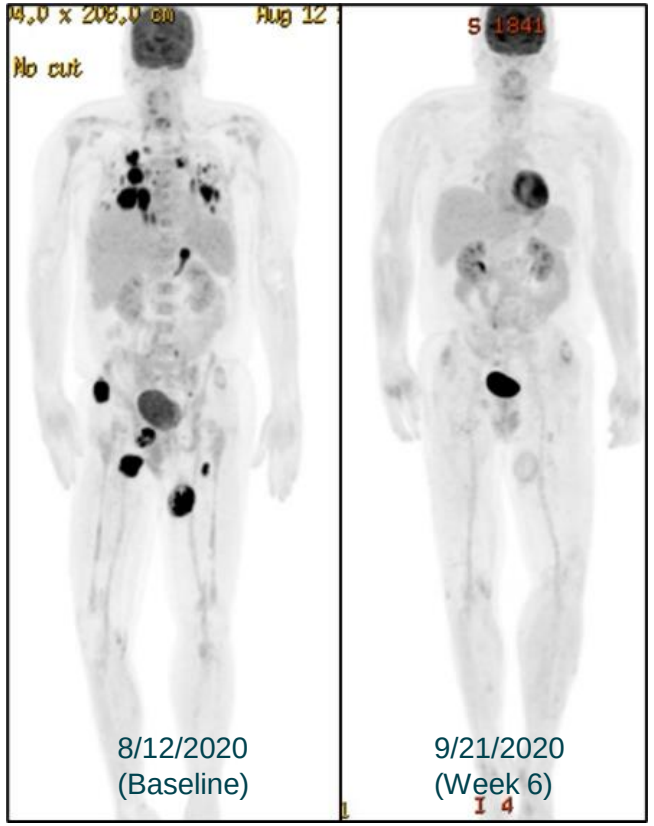
Early Experience in Non-PEComa Patients with *TSC1* or *TSC2* Inactivating Alterations from Expanded Access Program (presented at ASCO 2021[1])

Pt #2: Ovarian Cancer with *TSC1* mutation (4 prior lines Rx). Retroperitoneal and pelvic metastases

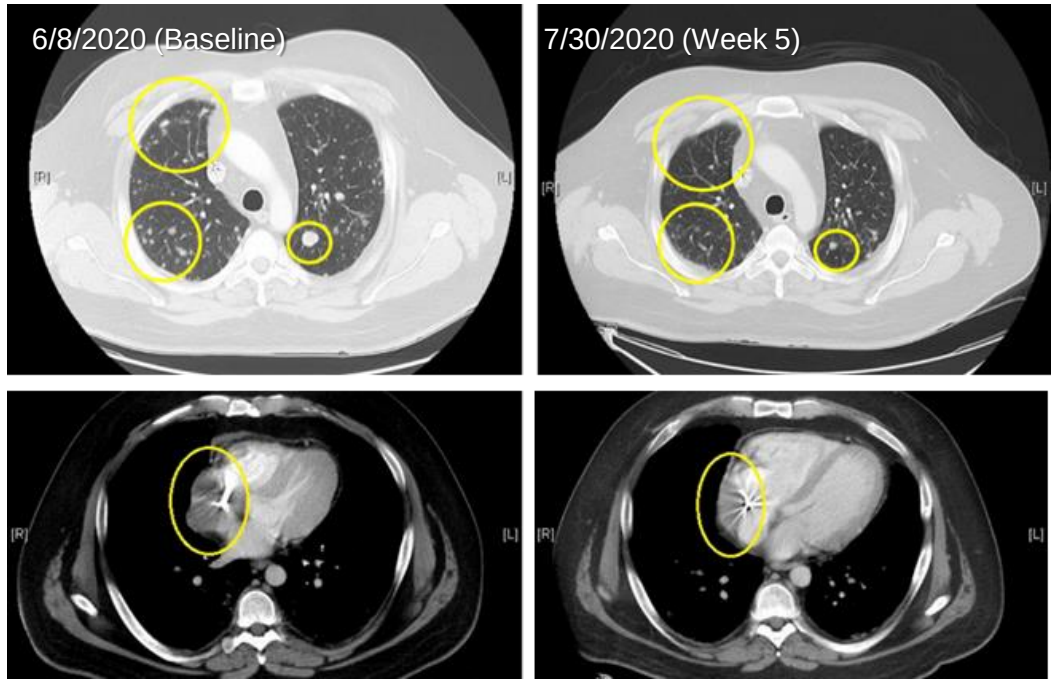




Pt #7: High-grade Sarcoma with *TSC2* mutation (5 prior lines Rx). Metastasis to lung, bone, and soft tissue; Li-Fraumeni syndrome



Pt #4: Angiosarcoma with *TSC1* mutation (2 prior lines Rx) involving right atrium, pericardium and with pulmonary metastasis



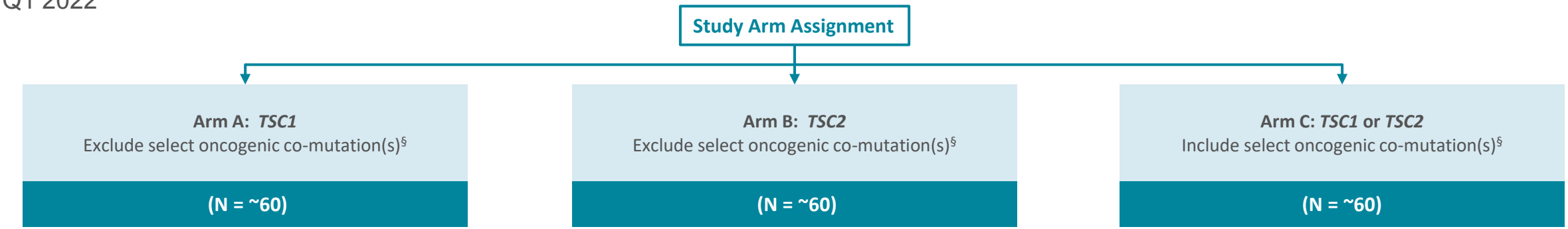
Source: 1) MA Dickson. ASCO. 2021. Abstract # 3111

nab-sirolimus Basket Study for *TSC1* or *TSC2* Inactivating Alterations Tumor-Agnostic Registrational Trial : PRECISION-1

- FDA Type B meeting conducted to discuss study design and strategy
- Independent arms for TSC1 and TSC2
- Primary endpoint : ORR
- Secondary Endpoints : DOR, DCR
- Patient accrual based on local NGS results
- Study initiation planned by end of 2021 or early Q1 2022

Key Eligibility Criteria

- Metastatic or locally advanced disease ineligible for surgery
- Naïve to mTOR inhibitor treatment
- Pathogenic *TSC1* or *TSC2* inactivating alterations identified through NGS
- Must have received standard therapy for the disease or in investigator opinion unlikely to benefit



Note: §List of co-mutations for inclusion/exclusion not currently disclosed

We Aim to Develop the Next Potential Tumor-Agnostic Target: Inactivating Alterations in *TSC1* and *TSC2*

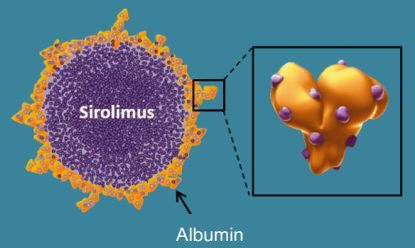
PAN TUMOR DRUG APPROVALS

2017	2018	2019	2020
Keytruda Anti-PD1	Vitrakvi TRK inhibitor	Rozlytrek TRK inhibitor	Keytruda Anti-PD1
Micro-satellite Instability-High (MSI-High) or mismatch repair deficient tumors	Adult solid tumors with an NTRK gene fusion	Adult or pediatric solid tumors with an NTRK gene fusion	Tumor mutational burden-high solid tumors

Single arm trials with ~50-70 patients and ORR as primary endpoint

Fyarro™
sirolimus protein-bound particles for injectable suspension (albumin-bound)

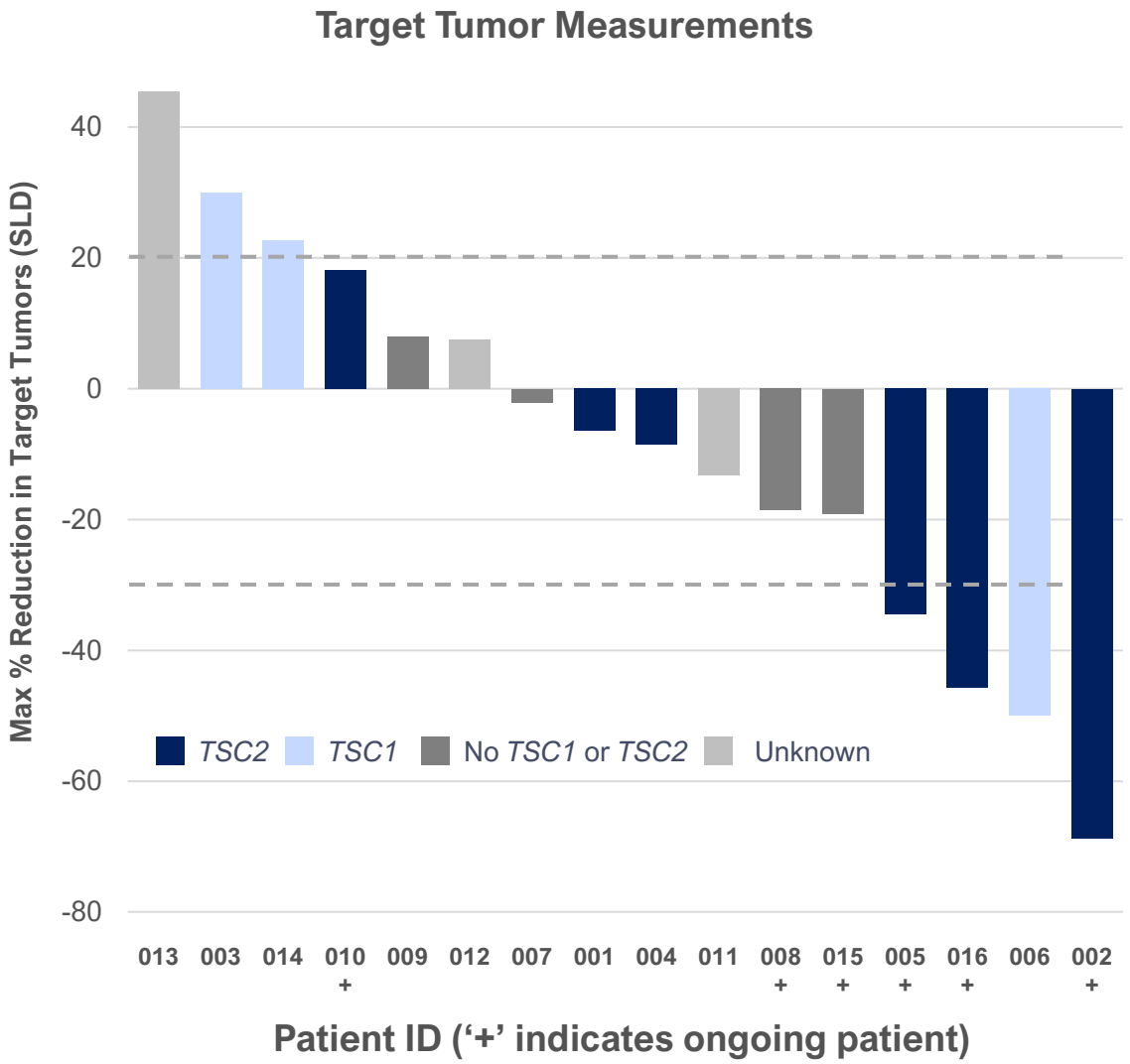
mTOR inhibitor



- *TSC1* & *TSC2* inactivating alterations in solid tumors

Activity of *nab*-sirolimus in Patients with Prior mTORi Experience

Efficacy of *nab*-sirolimus in Prior mTORi Treated Malignant PEComa Patients: Emerging Experience from an Expanded Access Program



Best Overall Responses	All Patients
	N = 16
Partial Response	**4/16 (25%)**
Stable Disease	**8/16 (50%)**
Stable Disease ≥12 weeks	6/16 (38%)
Progressive Disease	**4/16 (25%)**

- 10/16 (63%) patients had Disease Control (CR or PR or SD ≥3 months)
- 4 *nab*-sirolimus responders:
  - BOR on prior mTORi: 1/4 SD, 2/4 PD, 1/4 NE due to toxicity
  - 2/4 had 3 prior lines of Rx

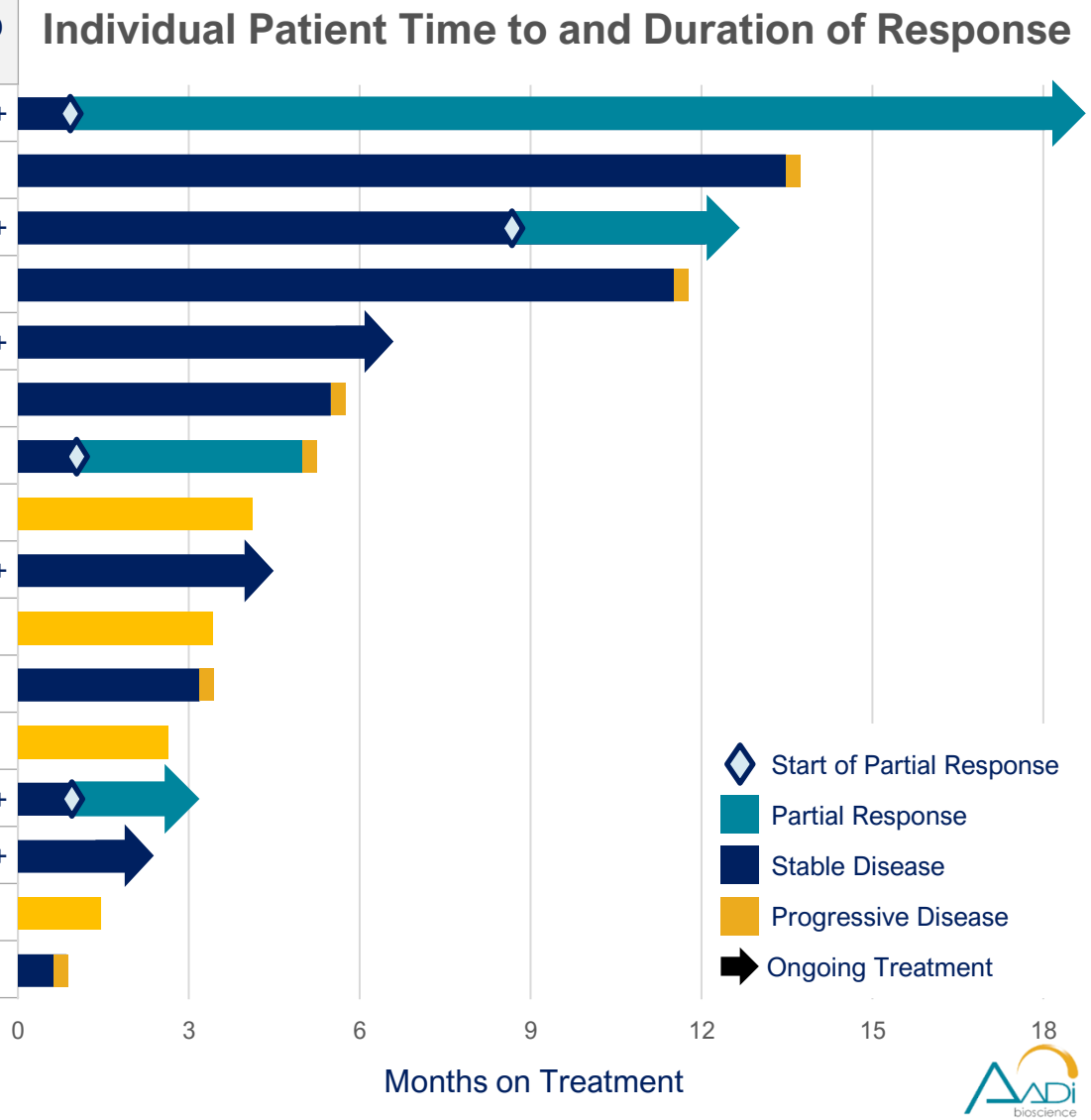
Best Overall Responses Patients with NGS* (N=13)	*TSC1*	*TSC2*	*Non TSC1/TSC2*
	n = 3	n = 6	n = 4
Partial Response	**1/3 (33%)**	**3/6 (50%)**	**0**
Stable Disease	**0**	**3/6 (50%)**	**3/4 (75%)**
Stable Disease ≥12 weeks	**0**	2/6 (33%)	3/4 (75%)
Progressive Disease	**2/3 (66%)**	**0**	**1/4 (25%)**

- 13 patients had available NGS reports
- Responders: 4/9 (44%) pts with *TSC1*/*TSC2* vs 0/4 with no *TSC1*/*TSC2* alterations

Note: NE tox- Pt came off for toxicity prior to any evaluation; NE- Not Evaluable; '+' indicates ongoing patient; * 3 patients had Unknown mutational status; NGS reports included MSK-IMPACT, MDA Molecular Diagnostic Lab, Foundation One, Oncopanel; Source: MA Dickinson, CTOS 2021

Efficacy of *nab*-sirolimus in Prior mTORi Treated Malignant PEComa Patients: Emerging Experience from an Expanded Access Program

# Prior Rx	List of Prior Rx and Best Overall Response (BOR) for Each Line	BOR on prior mTOR*	*TSC1 / TSC2*	Pt ID
3	sirolimus (SD), bempegaldesleukin + nivolumab (tox NE), pazopanib (PD)	SD	*TSC2*	002+
4	everolimus (PD), nivo + Ipi (PD), axitinib + pembro (PD), sunitinib (PD)	PD	*None*	007
3	sirolimus (PR), everolimus (tox NE), sirolimus (PD)	PD	*TSC2*	016+
3	everolimus (PD), letrozole + everolimus (PD), pazopanib (SD)	PD	*TSC2*	001
1	everolimus (PR)	PR	*None*	015+
4	sirolimus (tox NE), gemcitabine (NE), nivo + ipilimumab (SD), temsirolimus (SD)	SD	*TSC2*	004
1	everolimus (PD)	PD	*TSC1*	006
1	everolimus (PD)	PR	*TSC1*	014
1	sirolimus (PD)	PD	*None*	008+
1	everolimus (PR)	PR	UNK	013
1	everolimus (PD)	PD	UNK	011
1	everolimus (PD)	PD	*None*	009
1	sirolimus (tox NE)	NE tox	*TSC2*	005+
1	everolimus (PD)	PD	*TSC2*	010+
3	sirolimus (SD), ipi/nivo (PD), sapanisertib (PD), gemcitabine+docetaxel (PD), pazopanib (SD), pazopanib + sirolimus (SD)	SD	*TSC1*	003
1	everolimus (PR)	PR	UNK	012



Note: NE tox- Pt came off for toxicity prior to any evaluation; NE- Not Evaluable; '+' indicates ongoing patient; *BOR on last mTOR if patient had >1 prior mTOR ; All patients came off prior mTORi due to PD, except patient 005+ who had intolerable toxicities and patient 015+ who had a surgery, after which the patient had a recurrence; Source: MA Dickinson, CTOS 2021

Safety of *nab*-sirolimus in Prior mTORi Treated Patients (N=16)

Treatment-related Adverse Events (TR AE) by Frequency

- The most common (>20%) TR AEs:
  - anemia (50%)
  - rash and fatigue (31% each)
  - thrombocytopenia (25%)
- No Grade ≥4 TR AEs occurred

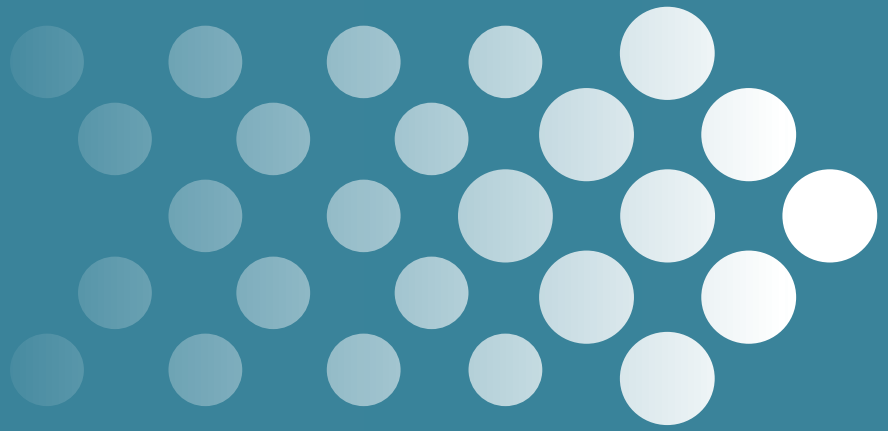
Treatment-related Serious Adverse Events (TR SAE) by Patient

- 5/16 (31) patients had at least 1 TR SAE: anemia, colitis, mucositis, rash, thrombocytopenia

Dose Reductions

- 4/16 (25%) patients had dose reductions including 2 patients with a PR, who maintained response
  - 3/4 were dose reduced to 75 mg/m^2
  - 1/4 was dose reduced to 30 mg/m^2

Source: Dickson et al., CTOS 2021



Summary and Milestones

Development Data Differentiating *nab*-sirolimus vs. Other mTOR Inhibitors

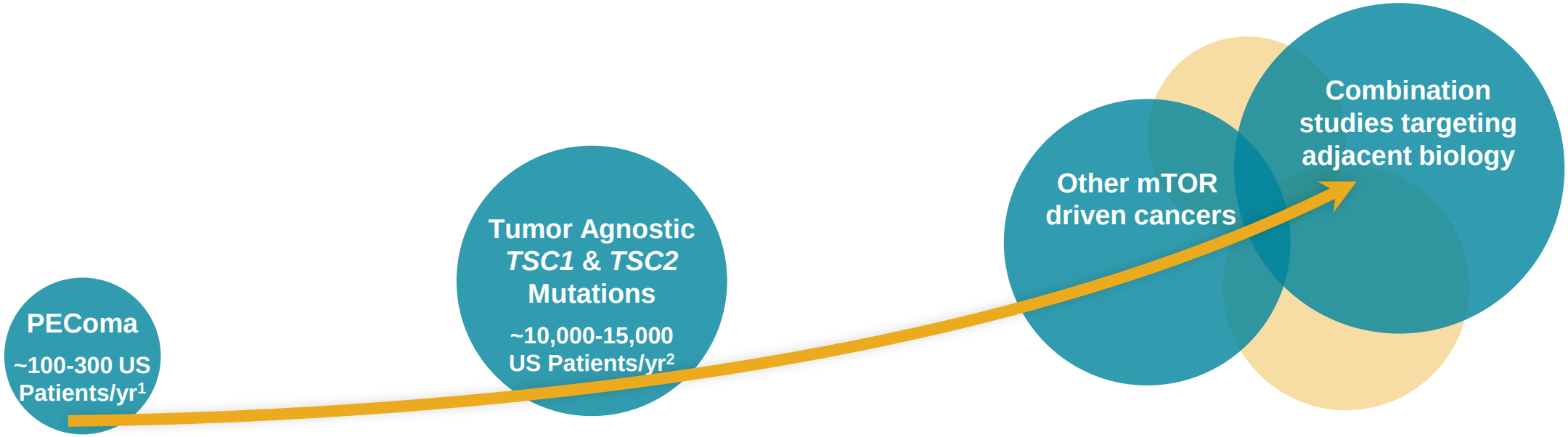
Key Differentiators of *nab*-sirolimus

- High tumor drug levels driven by albumin transport in preclinical animal models
- More efficient mTOR pathway suppression[1] of key downstream targets pS6 and p4EBP1
- Clearly differentiated and favorable PK/exposure profile[2]
- Encouraging long-term durability of response[3]
- Activity in *TSC1*/*TSC2* altered, non-PEComa, solid tumors: 5 PR of 7 evaluable[4*]
- Activity in patients after failure of mTOR inhibitors[5]
- Wide therapeutic index allows much greater flexibility particularly for new targeted combinations

Note: *Prior study evaluating everolimus in patients with advanced solid tumors harboring *TSC1, TSC2* or mTOR mutations noted 2 PR of 30 patients[6]; Sources: 1) S Hou, et al. AACR-NCI-EORTC. 2021. 2) AM Gonzalez-Angulo et al., Clin Cancer Res. 2013;19:5474-5484. 3) AJ Wagner et al., JCO. 2021. 4) MA Dickson. ASCO. 2021. Abstract # 3111 ; 5) Dickson CTOS 2021; 6) Adib et al . Clinical Can Res 2021 Jul 15;27(14):3845-3853

With the Approval of FYARRO, Aadi is on the Path to Becoming a Leading Precision Oncology Company

Pre-commercial | Commercial | Multi-Indication, Precision Oncology Company | Multi-Asset, Precision Oncology Company



Q1 2022:
Commercial-launch

Q4 2021 – Q1 2022:
Registrational trial initiation

H1 2024:
Final data anticipated

H2 2022:
Initiate additional trials in other cancer sub-populations with strong mTOR mechanistic rationale

H2 2022 – H1 2023:
Initiate additional trials of FYARRO in rational combinations with other targeted agents

Evaluate potential in-licensing or M&A opportunities focusing on assets with synergistic potential with mTOR inhibition

Note: 1) No formal published epidemiology information; Aadi analysis based on multiple sources including Aadi internal data and external research conducted by Tessellon Group and Corsica Life Sciences; 2) Analysis of TCGA, cBioPortal, and SEER databases conducted by Tessellon Group in June 2021

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