Corporate Presentation

June-July 2022

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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; risks related to the sufficiency Aadi's cash balance to fund operations; the timing of Aadi's 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 March 31, 2022, filed with the Securities and Exchange Commission (the "SEC") on May 12, 2022, 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. Except as required by law, Aadi undertakes no obligation to revise or update any forward-looking statement, whether as a result of new information, future events or otherwise.

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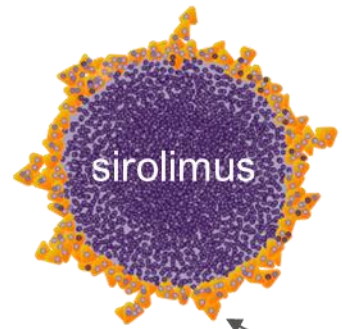
Aadi Bioscience is a Commercial-Stage, Precision Oncology Company Re-engineering mTOR Inhibition

NASDAQ: AADI

- Commercializing FYARRO™ (sirolimus albumin-bound nanoparticles, *nab*-sirolimus) for treatment of Advanced Malignant PEComa (FDA approval Nov 2021)
- Technology based on nanoparticle albumin-based (*nab*) platform proven with ABRAXANE®
- Focused on cancers that are highly mTOR dependent
- PRECISION 1 registrational study in tumor-agnostic *TSC1* or *TSC2* inactivating alterations in solid tumors now actively enrolling
- Cash runway into 2024 to support commercialization and ongoing and future clinical development of FYARRO



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

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Leadership: We have built 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

Stephen Rodin, JD
SVP / General Counsel

- Previously EVP / General Counsel at **The Medicines Company** (acquired by Novartis)
- Led or played a substantial role in **$750M+ acquisitions** and **$650M+ of divestitures** during 13-year tenure with The Medicines Company
- 15 years pharmaceutical legal experience

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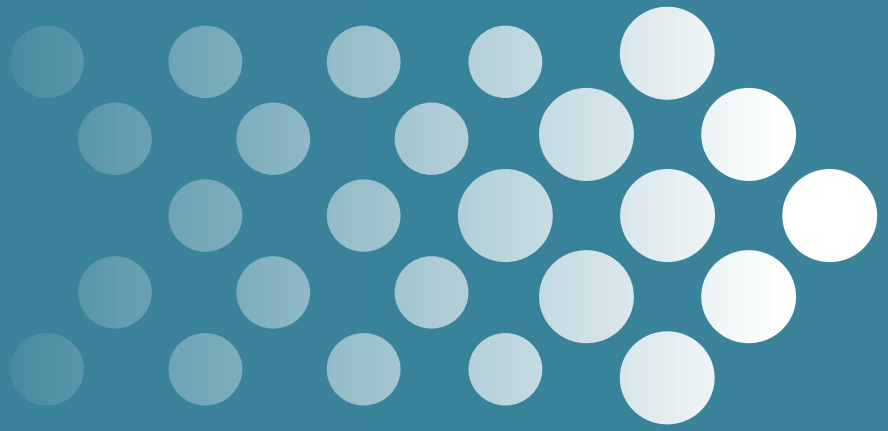


FYARRO™

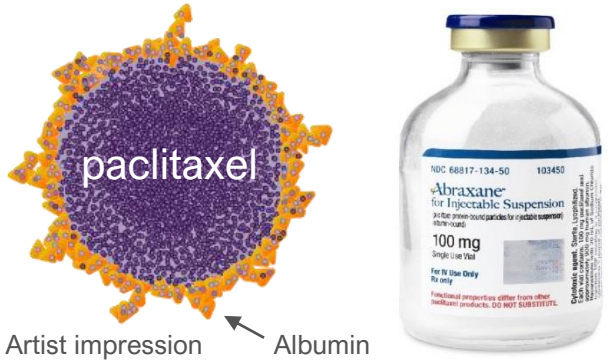
sirolimus protein-bound particles for injectable suspension (albumin bound)

Nanoparticle Albumin-Bound (*nab*) Technology

Nab Platform

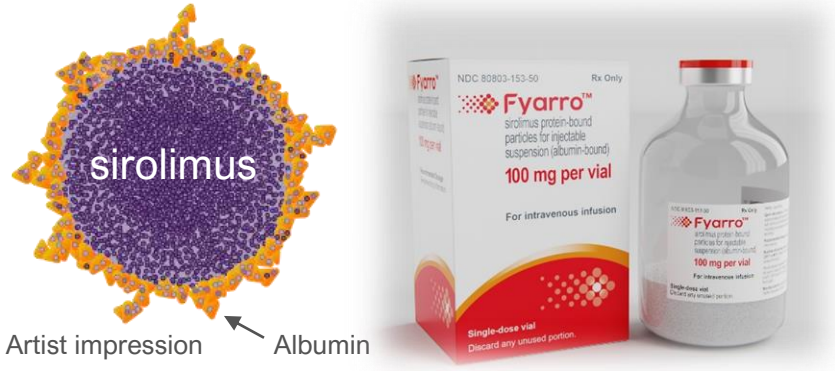
- Proprietary, complex, multi-step manufacturing process with trade secrets

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

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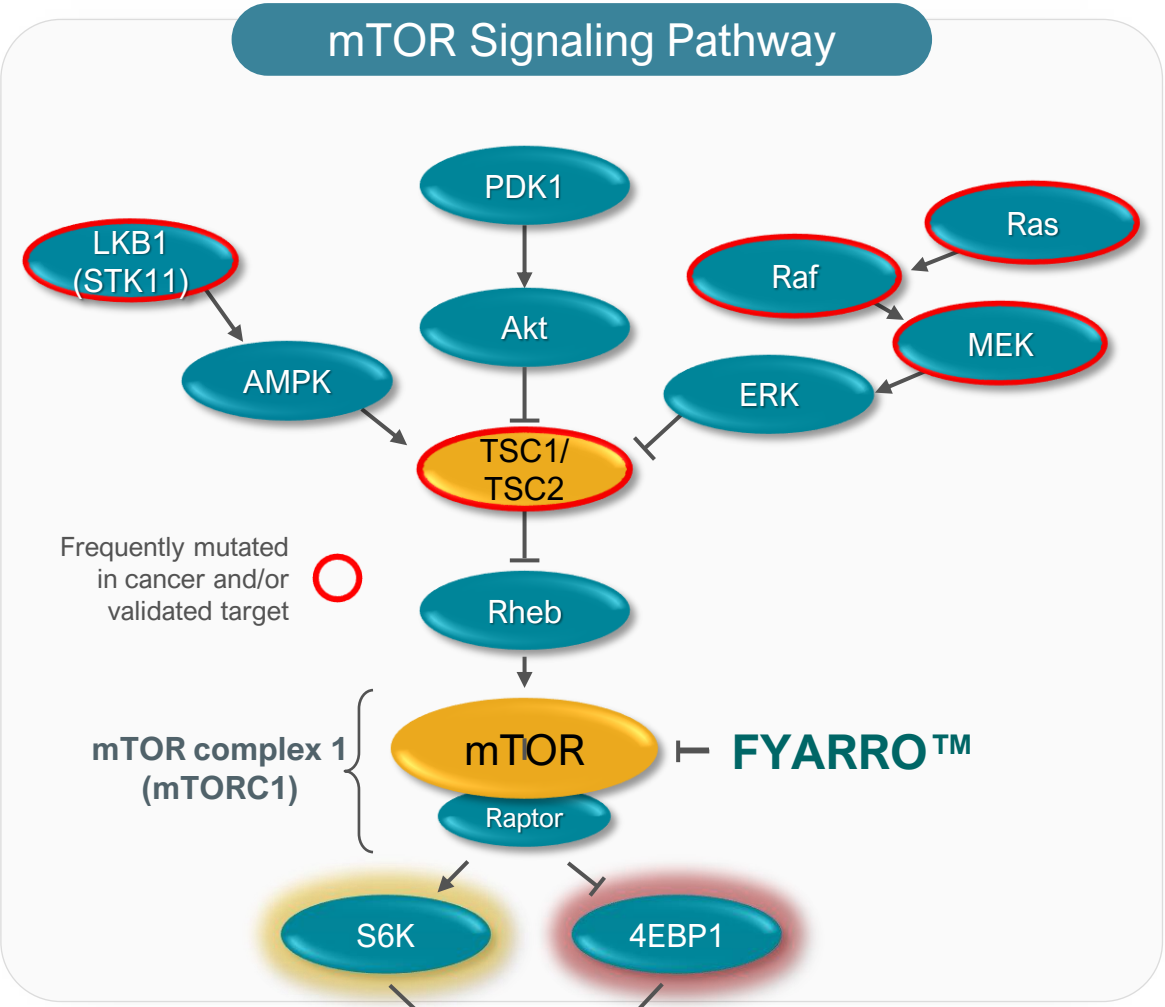
FYARRO™ Targets mTOR, a Key Signaling Pathway in Cancer



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]

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

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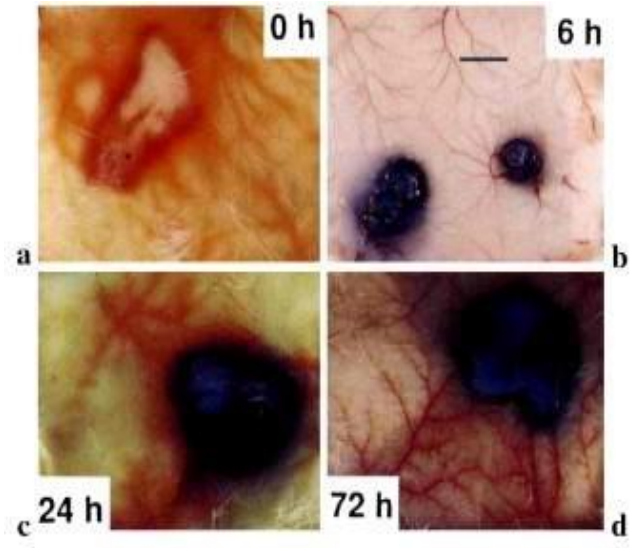
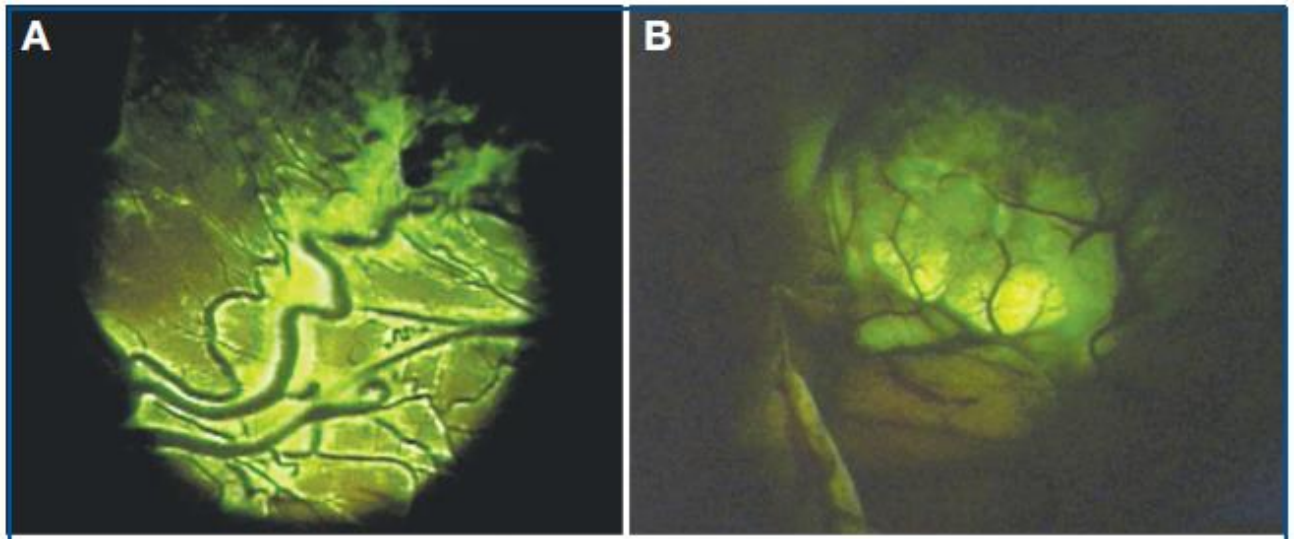
Role of Albumin in Tumor Targeting

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]



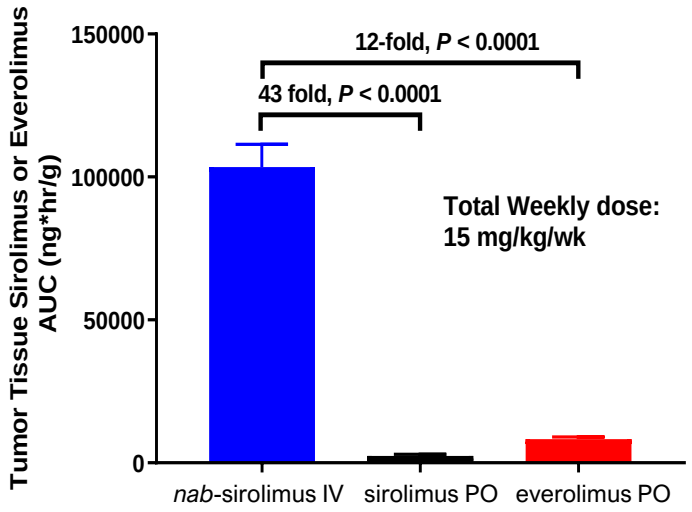
- 5-Amino Fluorescein labelled albumin administered IV (0.5-1 mg/kg) in 13 patients, 0.5-4 days before surgery
- Tumor fluorescence was bright in 11 patients (84%), resulting in complete resection in 9 patients (69%)

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

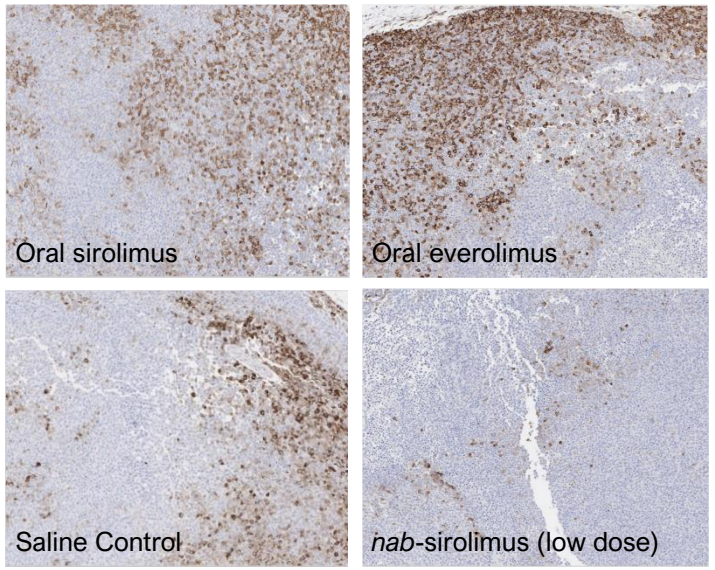
Higher *nab*-sirolimus Intratumoral Concentrations Drive Increased Target Suppression and Tumor Growth Inhibition in a Bladder Cancer Xenograft (UMUC3)

Significantly Higher Intratumoral Drug Accumulation



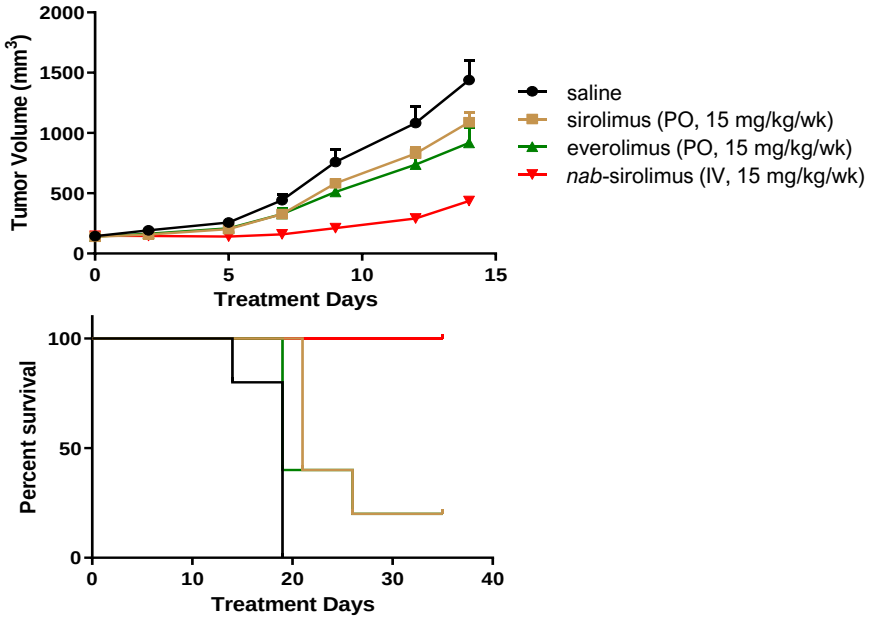
Tumor concentration of *nab*-sirolimus, oral sirolimus, and oral everolimus measured over 7 days at equal weekly dose (15 mg/kg/wk) in mice bearing tumor xenografts

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

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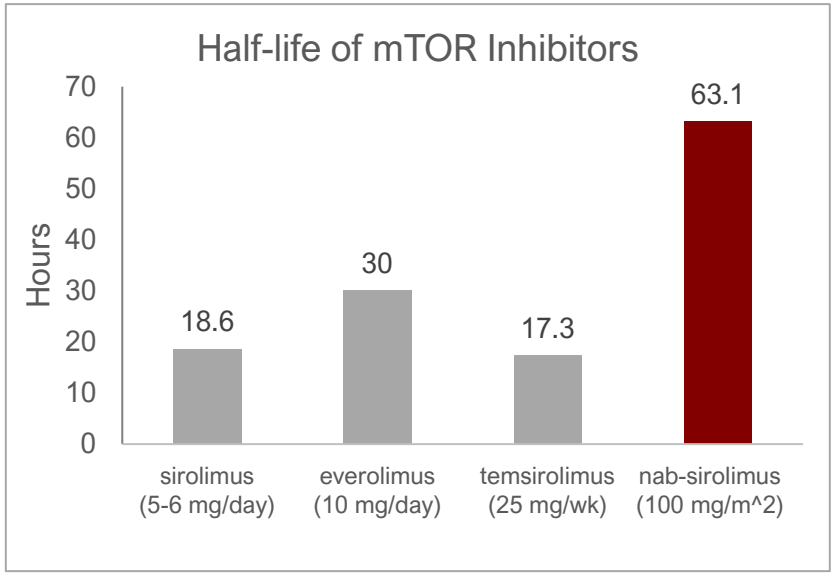
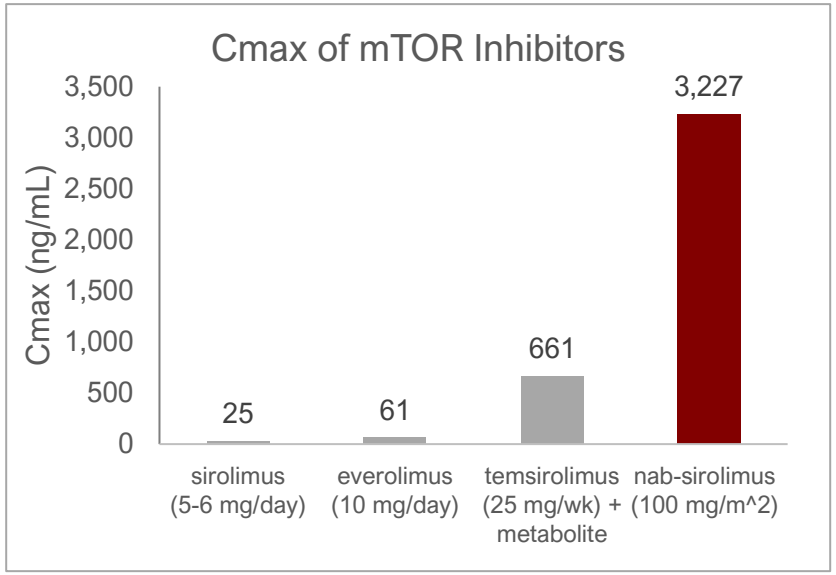
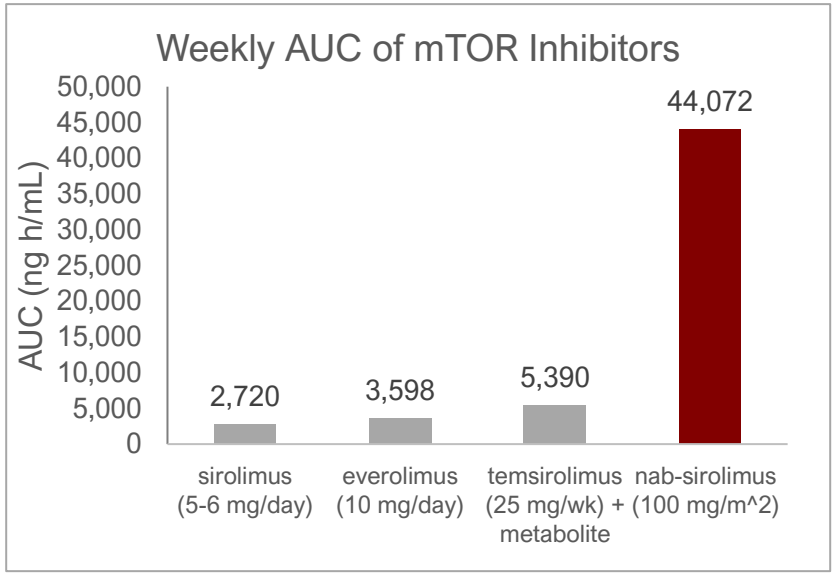
Differentiated Clinical PK Profile Compared to Other mTOR Inhibitors

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

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FYARRO™ Advanced Oncology Development Pipeline

Investigational Uses

Populations	Phase 1b	Phase 2	Registrational	Approved	Current Status
Advanced Malignant PEComa, AMPECT Clinical Trial	**Single Agent**				First FDA approved therapy for advanced malignant PEComa
Pan-Tumor *TSC1 / TSC2* Inactivating Alterations	***TSC1* Arm, Single Agent** ***TSC2* Arm, Single Agent**				Tumor-agnostic pivotal study with independent arms for *TSC1* or *TSC2* inactivating alterations; open for enrollment
Dose Finding Studies					Evaluate new single agent and combination strategies (e.g., mTOR pathway adjacent combinations) in addition to ongoing studies

Ongoing To be initiated / in planning

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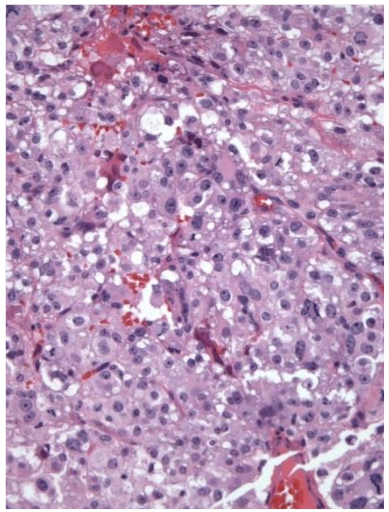
Locally Advanced Unresectable or Metastatic Malignant Perivascular Epithelioid Cell Tumor (PEComa)

PEComa Disease Overview and Standard of Care

Advanced, Malignant PEComa



- Ultra rare sarcoma
- Estimated 100-300 new patients per year in the US[6]
- Biological evidence of mTOR pathway activation; cancer type with highest rate of *TSC1* & *TSC2* mutations[2-4]
- Estimated survival of 12-16 months[5]
- Can arise at any site but most commonly at visceral (especially gastrointestinal and uterine), retroperitoneal, and abdominopelvic sites and with female predominance
- 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]

Previously 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

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

FYARRO now approved and **included in NCCN Guidelines** as the **only 'Preferred' treatment for malignant PEComa**

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%

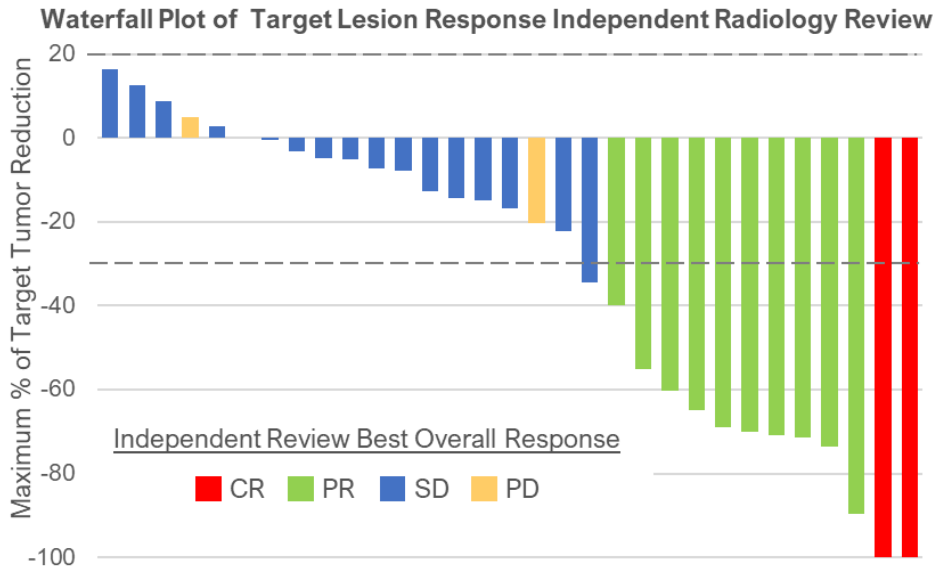
Efficacy Results in AMPECT[1,2]	Independent Radiology Review
Overall Response Rate (95% CI)	**39%** (22%, 58%)
Complete Response[1,2]	7% (2/31)
Partial Response[2]	32% (10/31)
Stable Disease[2]	52%
Progressive Disease[2]	10%
Disease Control Rate‡[2]	71%
Median Duration of Response[2]	**>36 months**
Median 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

Manageable Safety Profile (N=34)

- Most treatment-related adverse events (TRAEs) grade 1 or 2. No grade 4 or 5 TRAEs
- Most common nonhematologic TRAEs: mucositis (79%), fatigue (59%), and rash (56%)
- Most common hematologic TRAEs: anemia (47%) and thrombocytopenia (32%)
- Noninfectious pneumonitis occurred in 18% of patients and was grade 1 or 2
- Dose reductions occurred in 13/34 (38%) of patients
- Two patients discontinued due to a TRAE (grade 2 anemia and grade 1 cystitis)



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

PEComa Commercial Launch (Feb 22, 2022)

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

Q1 2022, $2.3M net sales for partial quarter (6 weeks): EAP and AMPECT patients transitioned to commercial drug; Pent up demand (Nov approval to Feb launch); Specialty Distributors carrying higher inventory in early launch period

NCCN GUIDELINES

Preferred: NCCN listed as the only 'Preferred' treatment for malignant PEComa

AADi assist

Accessible: Launched Aadi Assist, a comprehensive patient support program, to ensure access to FYARRO; National and Regional payers continue to adopt coverage policies

Engaged: Experienced commercial team is in place with Launch execution focused on establishing FYARRO as SOC in malignant PEComa

Field Team Engagement

10+ Customer facing team members in the field

~55 Sarcoma centers being targeted for early launch

100+ Sarcoma KOLs engaged including leading PEComa KOLs

Strong overall perceptions and high FYARRO awareness

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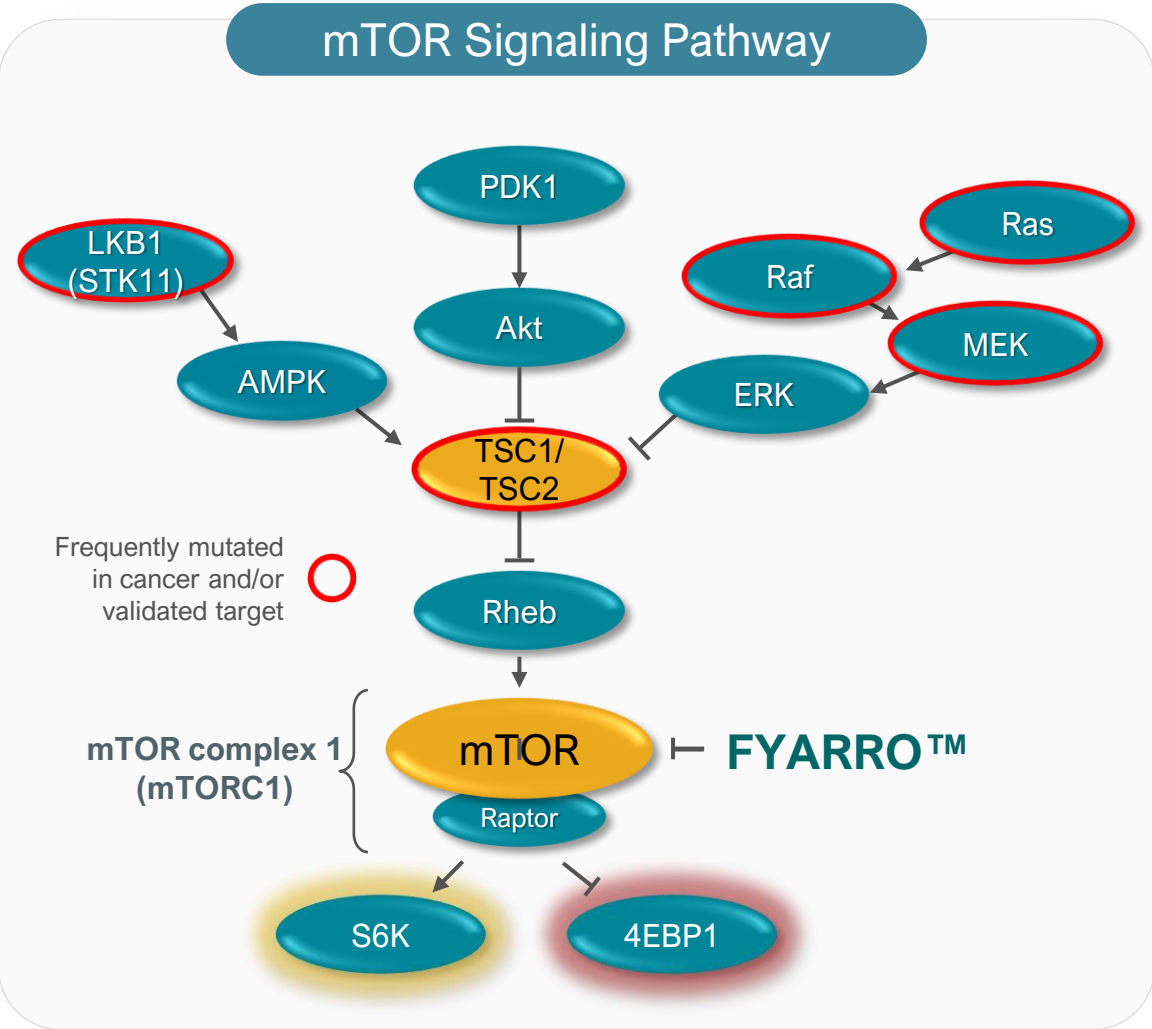
AADi bioscience

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

TSC1 and *TSC2* Alterations: 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

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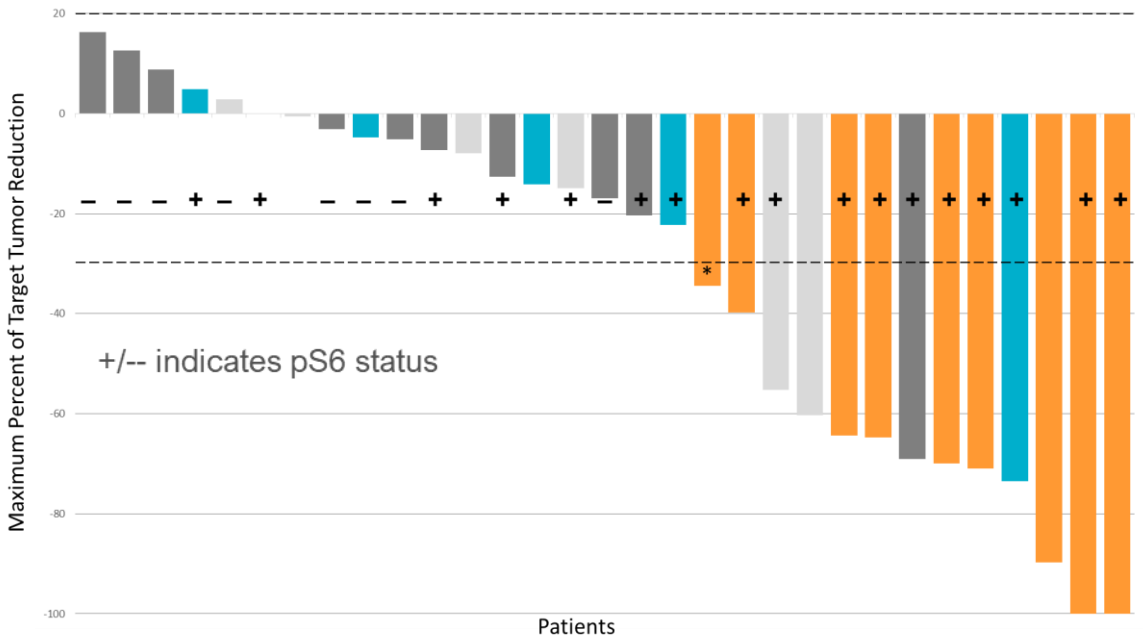
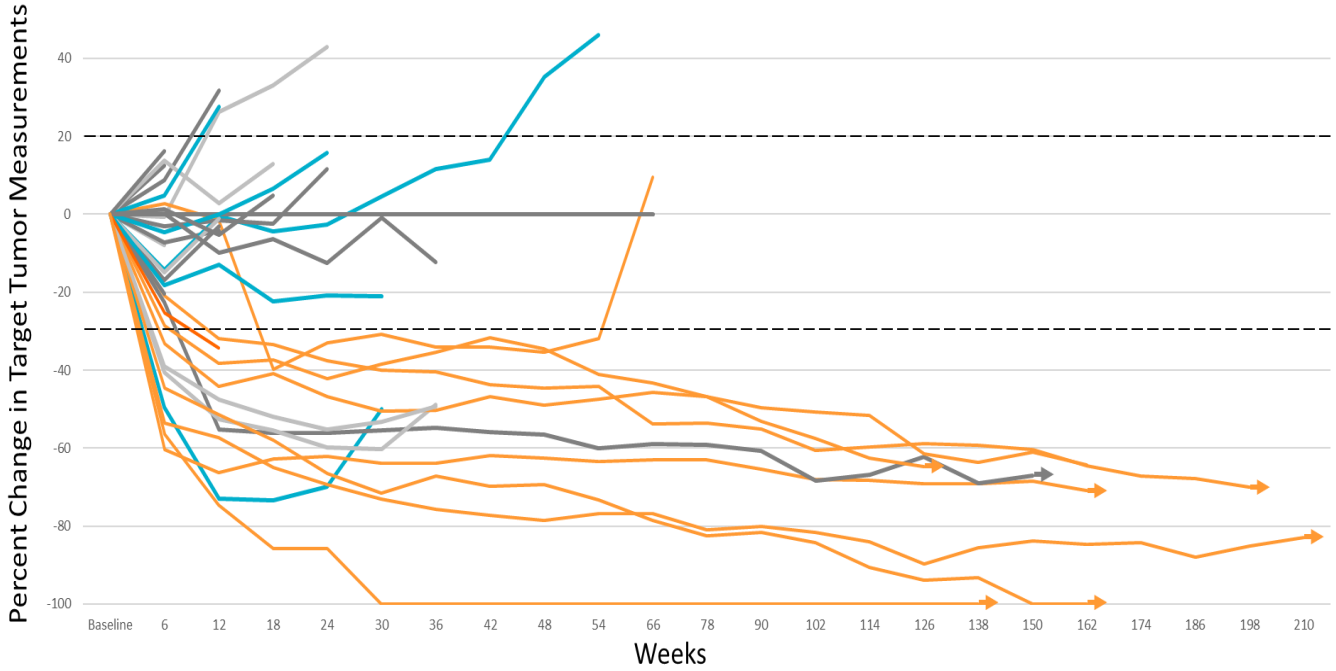
Data from AMPECT in *TSC1* or *TSC2* Inactivating Alterations Supports Further Investigation Across Different Tumor Types





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*

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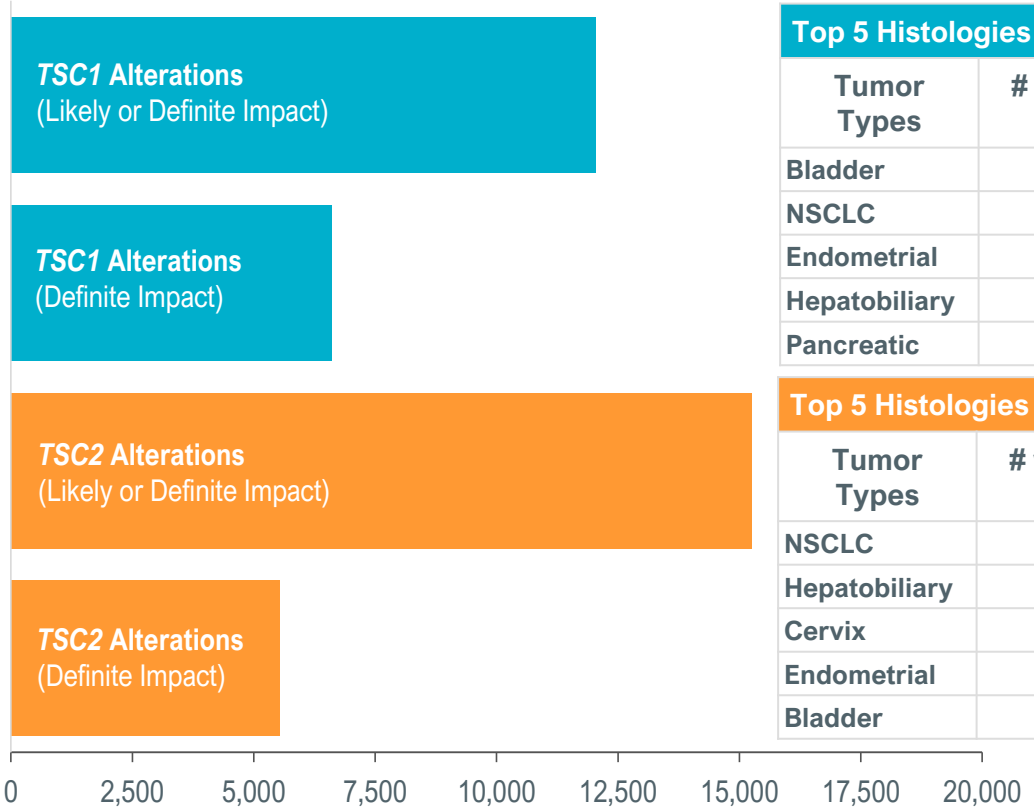
TSC1 and *TSC2* Inactivating Alterations Across All Cancers Represent Significant Opportunities

Projected Annual Incidence of Cancers with TSC1 and *TSC2* Alterations[1]

Estimated US Patients Available for 1st Line Therapy in 2030



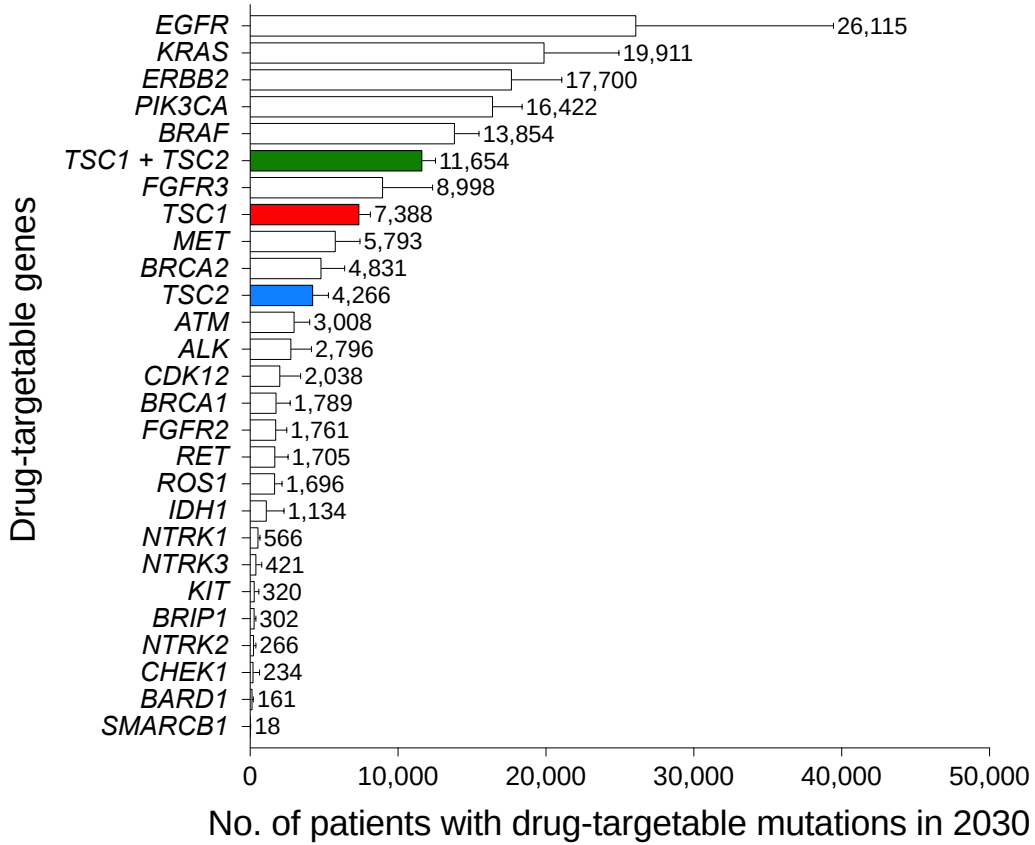
Top 5 Histologies by # of *TSC1* Patients

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%)

Incidence of TSC1 and *TSC2 Alterations vs. 26 Other Actionable Genes*[2]



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)

1) analysis of TCGA, cBioPortal, and SEER databases conducted by Tessellon Group in June 2021 2) G. Gulati, et al. AACR Annual Meeting 2022. Poster #5799

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Expanding Beyond PEComa
Early Experience Other Tumor Types with *TSC1* or *TSC2* Inactivating Alterations

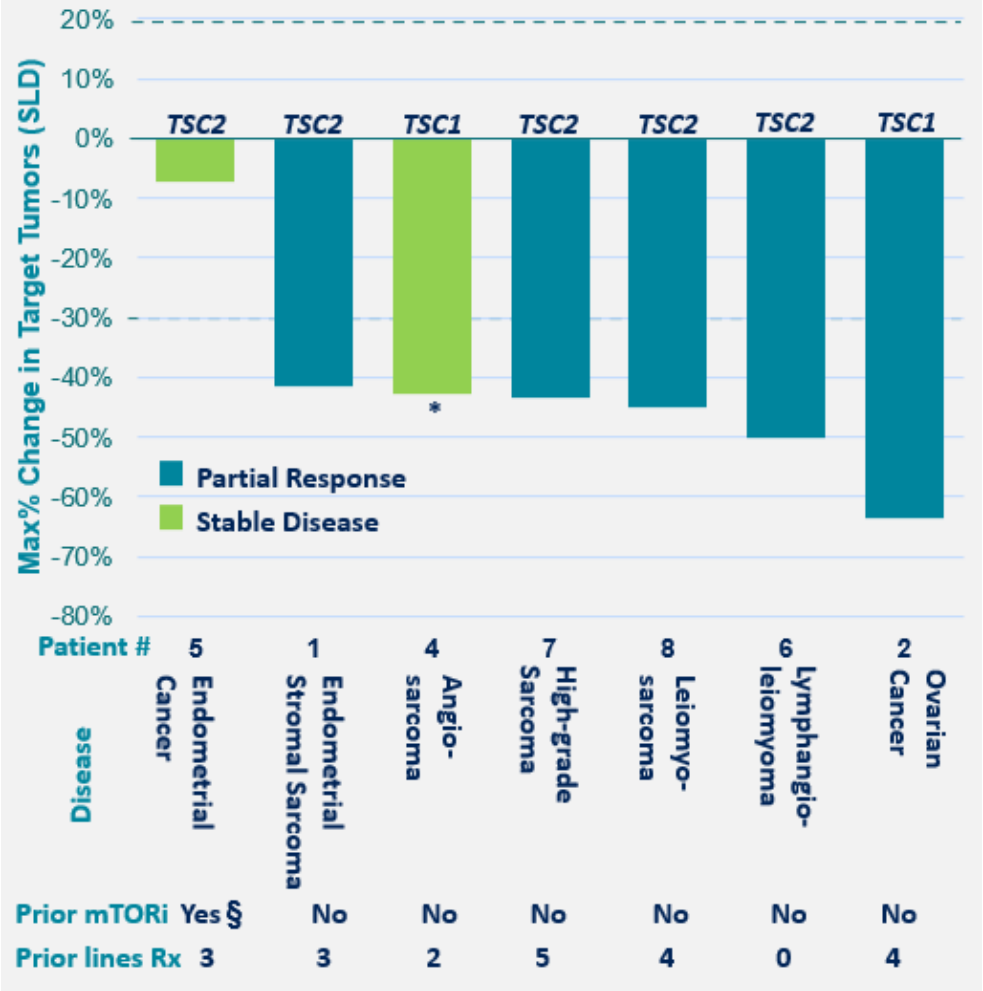
Expanded Access Program

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
- Tumor types: Ovarian cancer, endometrial cancer, angiosarcoma, leiomyosarcoma, lymphangio-leiomyoma, high grade sarcoma, endometrial sarcoma
- Lines of prior therapy: median 3.5 (range 0-6)

Efficacy

8 patients treated, 7 evaluable for response



Safety

- 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

ABI-009 is an investigational new drug and has not been approved for commercial distribution in the United States.

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

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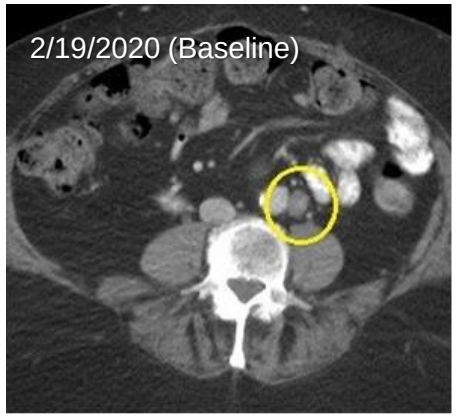
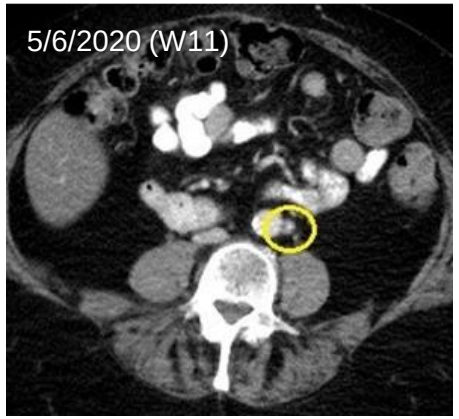
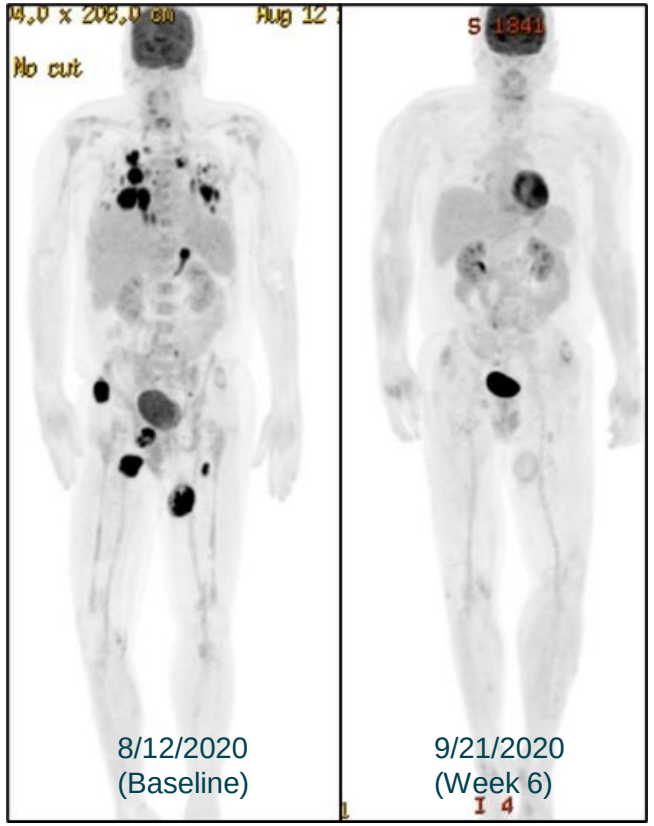
Early Experience in Non-PEComa Patients with *TSC1* or *TSC2* Inactivating Alterations from Expanded Access Program (presented at ASCO 2021[1])

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

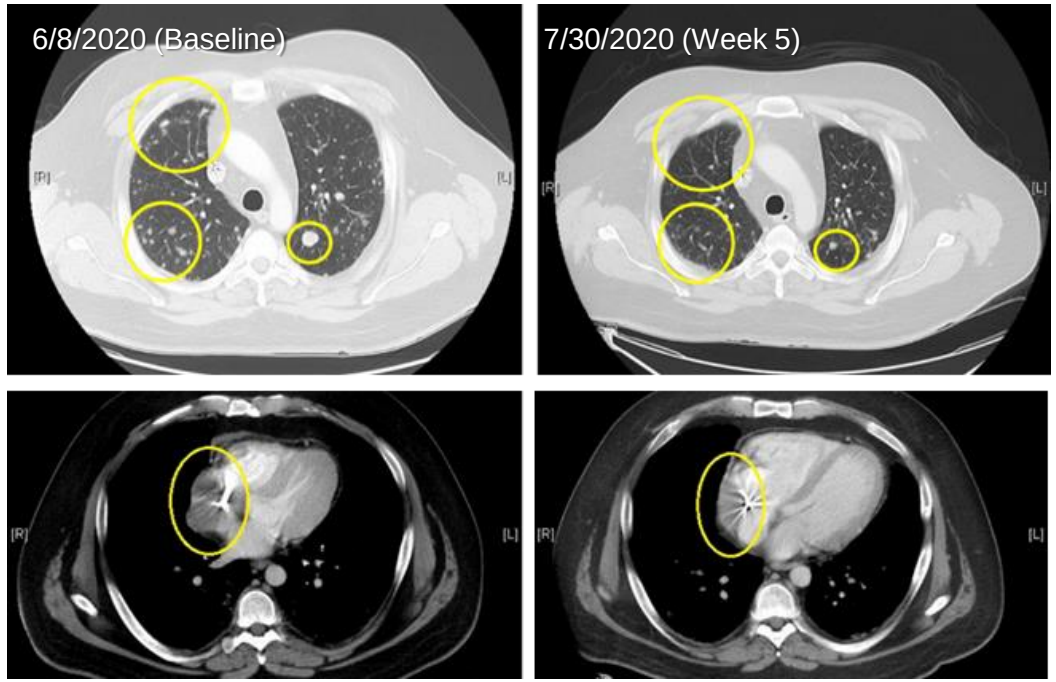




Pt #7: High-grade Sarcoma with *TSC2* mutation (5 prior lines Rx). Metastasis to lung, bone, and soft tissue; Li-Fraumeni syndrome



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

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

- Independently evaluable arms for *TSC1* and *TSC2*
- Primary endpoint : ORR
- Secondary Endpoints : DOR, DCR
- Patient accrual based on local NGS results
- First patient dosed (March 2022)
- Initial clinical data expected 1H 2023

Strategies to expedite enrollment:

- Partnered with NGS providers
- Partnered with US Oncology

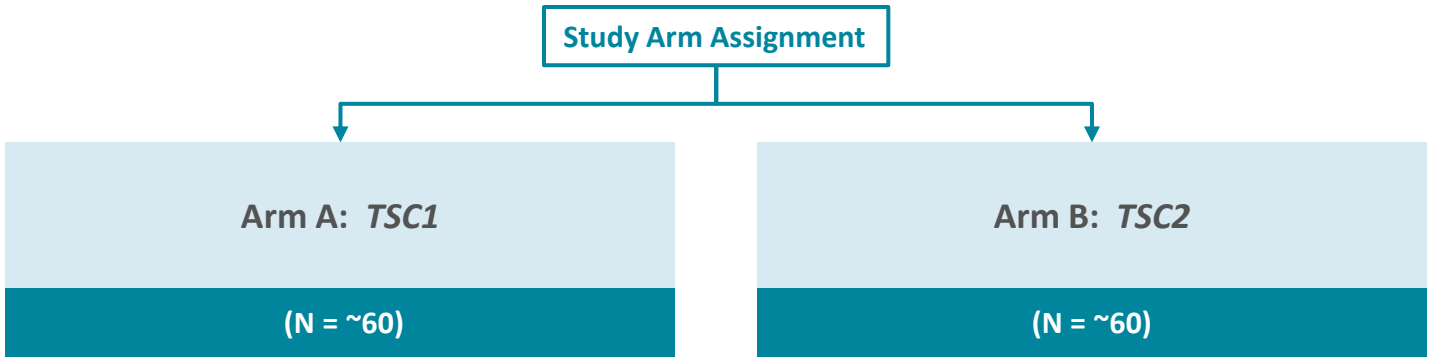
FOUNDATION MEDICINE

TEMPUS

The US Oncology Network

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



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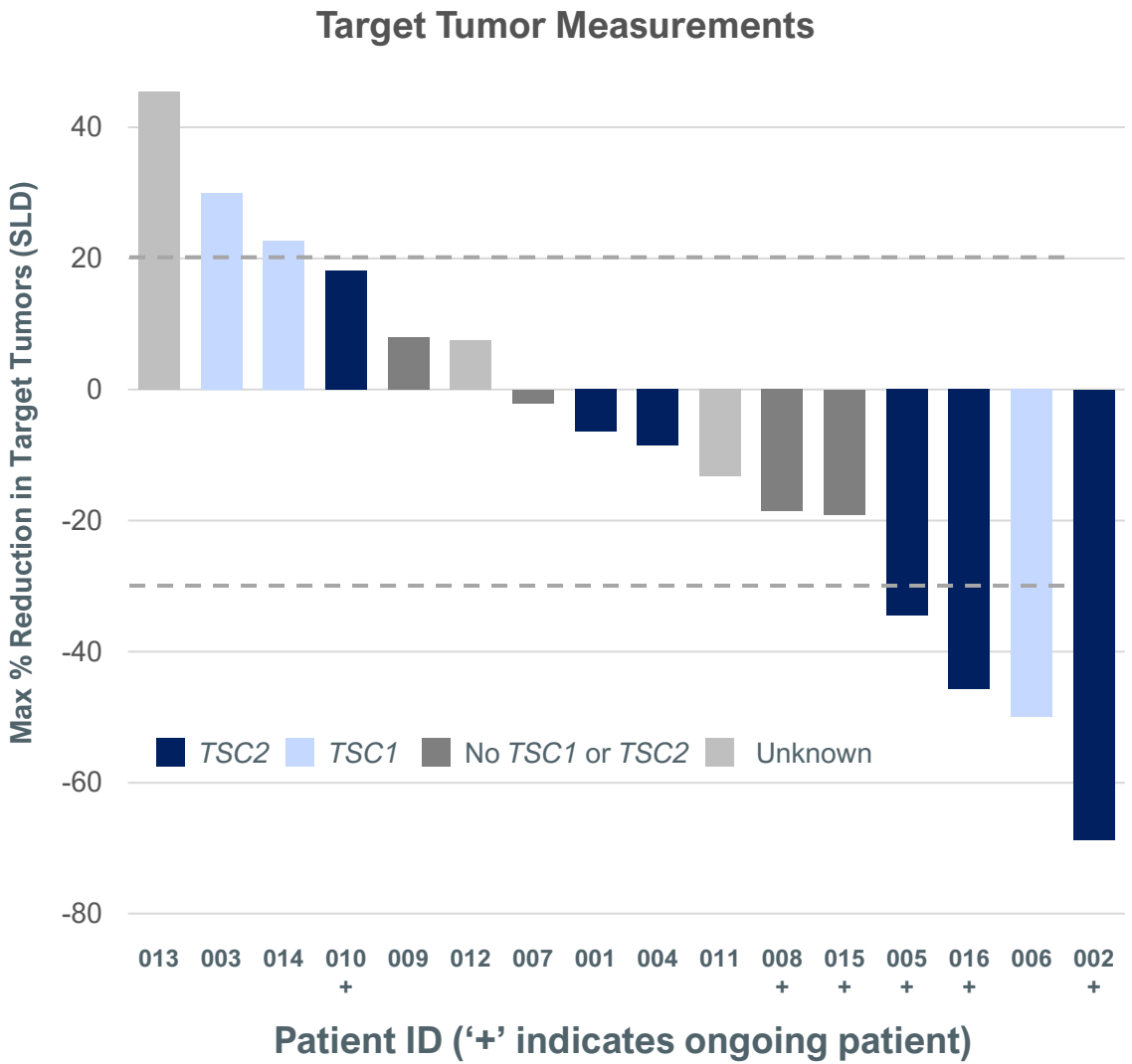
Efficacy of *nab*-sirolimus in Prior mTORi Treated Malignant PEComa Patients: Emerging Experience from an Expanded Access Program



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*

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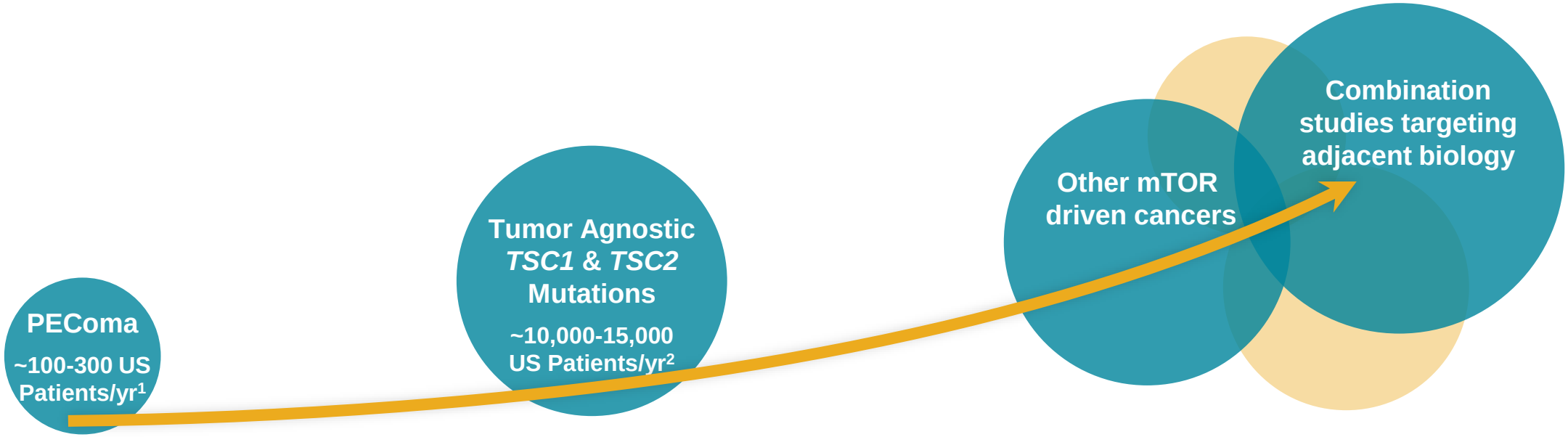
With the Approval of FYARRO, Aadi is on the Path to Becoming a Leading Precision Oncology Company

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



Q1 2022:
Commercial-launch

Q1 2022:
Registrational trial initiation

H1 2023:
Initial clinical data expected

H1 2024:
Final data anticipated

H2 2022 – H1 2023:
Evaluate new single agent and combination strategies (e.g., mTOR pathway adjacent combinations) in addition to ongoing studies

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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Aadi Bioscience Inc., Pacific Palisades, CA 90272, USA
NASDAQ: AADI | www.aadibio.com
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Contact Information

Contacts

Investors:

Marcy Graham
mgraham@aadibio.com

Media:

Darren Opland, PhD
darren@lifescicomms.com

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