ATARA BIO

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Founder, President, & CEO

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Proprietary Materials

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Broad Clinical Pipeline

ATARA BIO

Total cash >$100M upon completion of IPO in Oct 2014*

Sep 2012 License

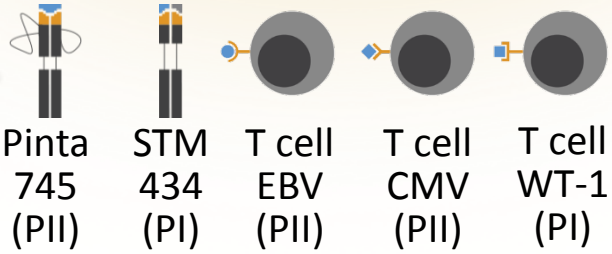
AMGEN®

- TGF-B family protein modulator
- Seven Molecules
- Scientific Support/Personnel

Sep 2014 Option

Memorial Sloan Kettering Cancer Center

- Clinical stage off the shelf T cell immunotherapies
- Platform technology to develop additional product candidates



* $51.7 million (as of 9/30/2014); Includes 55.8 million of net proceeds from IPO

Highlights

Licensed Clinical Programs

- PINTA 745: Anti-myostatin peptibody for protein-energy wasting (PEW) in dialysis patients in Phase 2
- STM 434: Ligand trap that binds to Activin for ovarian cancer and other solid tumors in Phase 1

Serious Unmet Needs In Large Markets

- PEW: Increased morbidity / mortality with an estimated ~250K patients in the US[1] and 800K worldwide; no approved therapeutics
- Ovarian Cancer: One of the deadliest cancers with blended 5-year survival rate of 44%;[2] ~22K new cases in 2013 in the US[3]

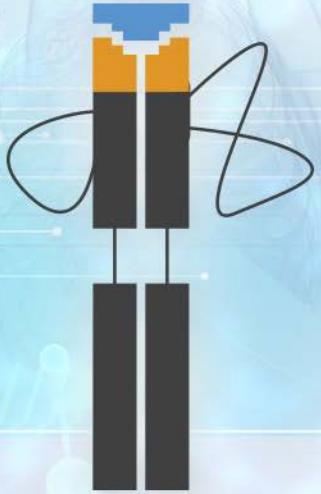
T cell Immunotherapy Collaboration with MSK

- Exclusive option and research agreement granting us the right to acquire worldwide license rights to T cell therapies targeting EBV, CMV and WT1
- Two programs in Phase 2, one in Phase 1 in US-based trials

Multiple Upcoming Milestones

- PINTA 745: Phase 2 data in 2H:15
- STM 434: Phase 1 data in 1H:16

[1] Calculated utilizing 2011 data from USRDS extrapolated to December 31, 2013 as well as data from the recent study completed with DaVita Clinical Research
[2] National Cancer Institute SEER Database
[3] National Cancer Institute estimate



PINTA 745

Protein Energy Wasting in
End Stage Renal Disease (ESRD)

PINTA 745: First in Class Molecule for PEW

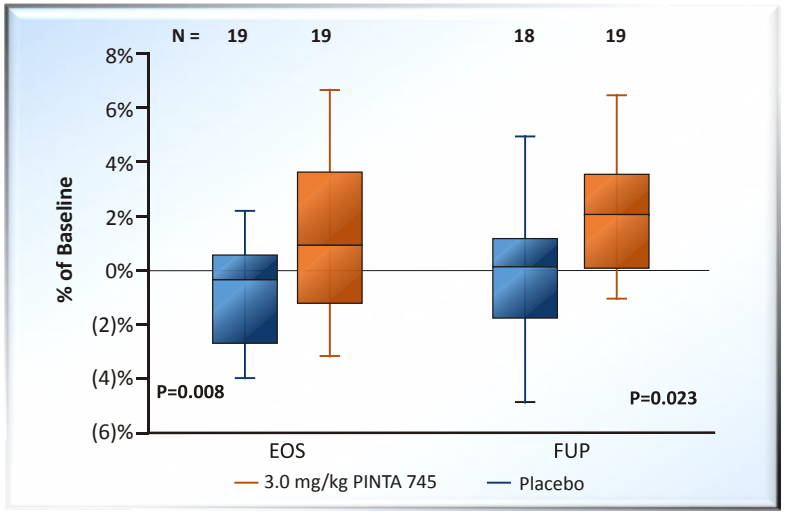
PINTA 745

MOA	➢ Biologic therapy targeting myostatin, blocking its role in inhibiting muscle production and maintenance
Indication	➢ PEW is a state of muscle wasting, inflammation and malnutrition associated with decreased physical function, increased morbidity and mortality in patients with ESRD
Proposition	➢ Multiple publications in peer reviewed journals showing the role of PINTA 745 and its surrogate in improving lean body mass, physical function, and inflammation
Clinical Data	➢ POC study in prostate cancer patients, with statistically significant increases in lean body mass and lower extremity muscle size
Clinical Status	➢ Ongoing, randomized, double-blinded, placebo controlled Phase 2 trial to enroll 48 ESRD patients with PEW ➢ Phase 2 data expected 2H:15

Clinical Proof of Concept in Patients with Prostate Cancer Receiving ADT

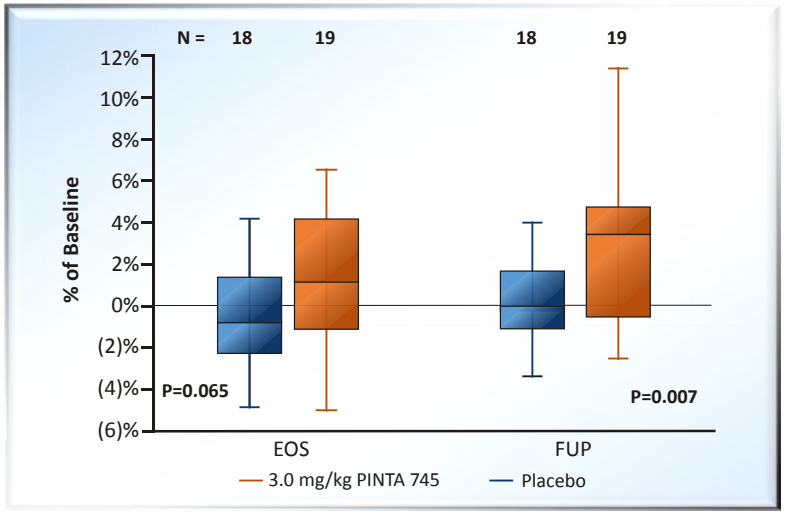
PINTA 745

Lean Body Mass Increase



- ~2% greater in lean body mass at EOS
- Difference in lean body mass compared to placebo continued to increase in 4 weeks after treatment (FUP)

Lower Extremity Muscle Size Increase



- At EOS, muscle size increased by ~1.2% from baseline
- At FUP, the change from baseline increased to 2.7%

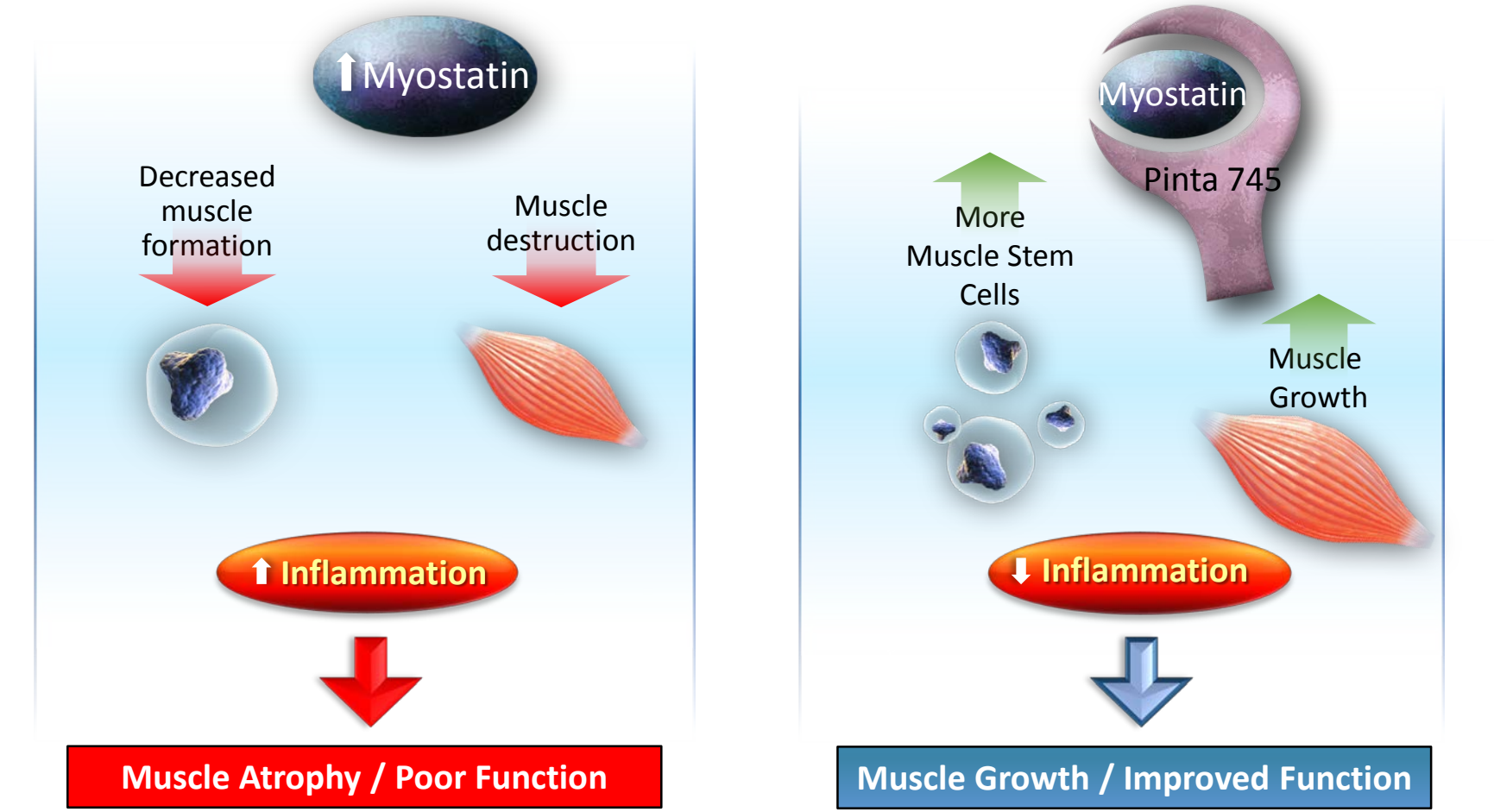
Note: The bottom and top of the boxes represent the first and third quartiles, and the horizontal band inside the box indicates the median value. The ends of the whiskers represent the minimum and maximum data in the range of observations
Note: EOS: end-of-study (at day 29); FUP: Follow-up Period (one month after day 29)

Padhi et al., 2014. Pharmacological inhibition of myostatin and changes in lean body mass and lower extremity muscle size in patients receiving androgen deprivation therapy for prostate cancer. J Clin Endocrinol Metab 99(10):E1967

Scientific Rationale for Myostatin Inhibition in PEW

PINTA 745

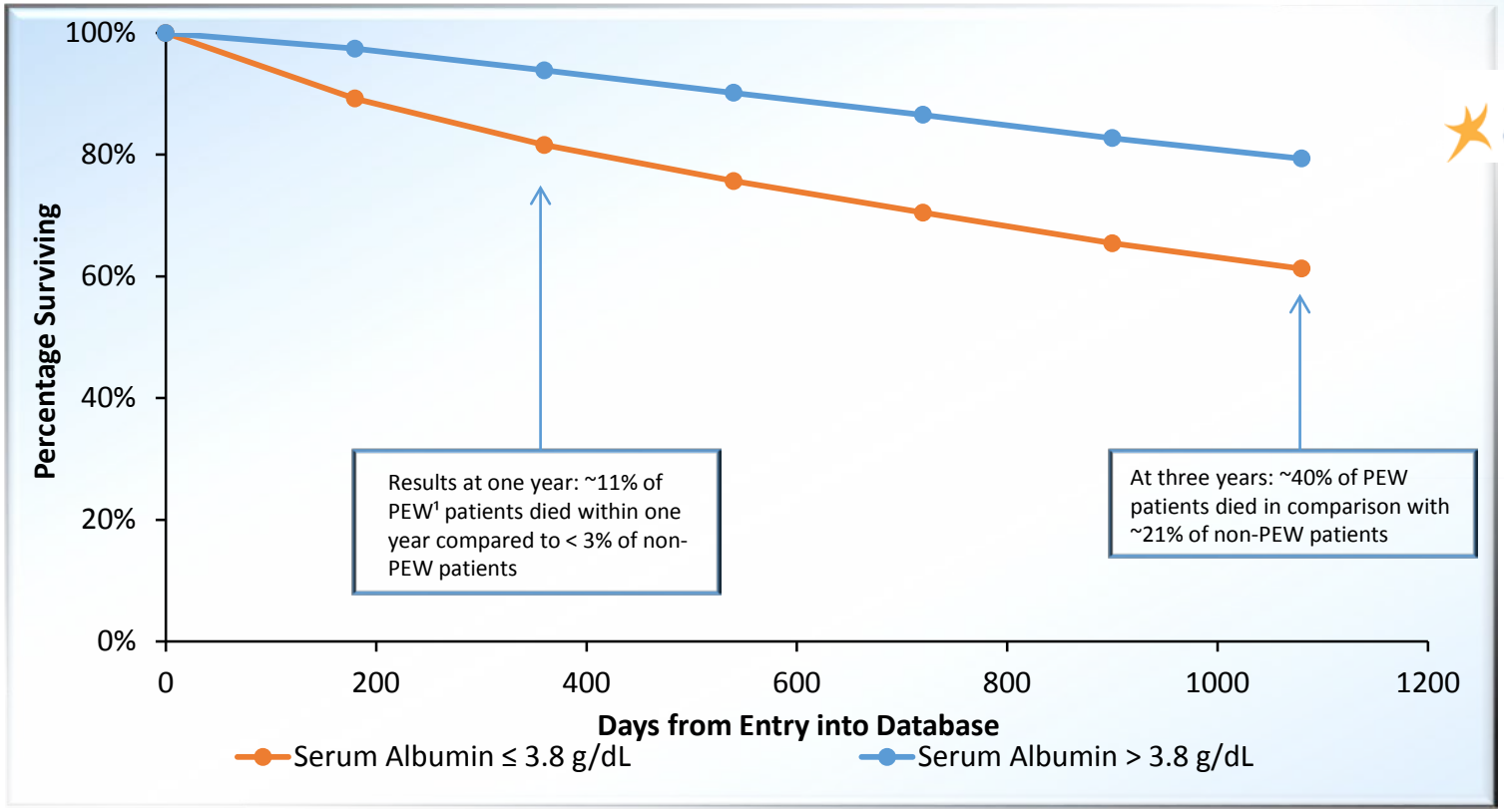
Inhibiting Myostatin Promotes Skeletal Muscle Growth in CKD



PINTA 745: Protein Energy Wasting in ESRD

PINTA 745

DaVita Clinical Research



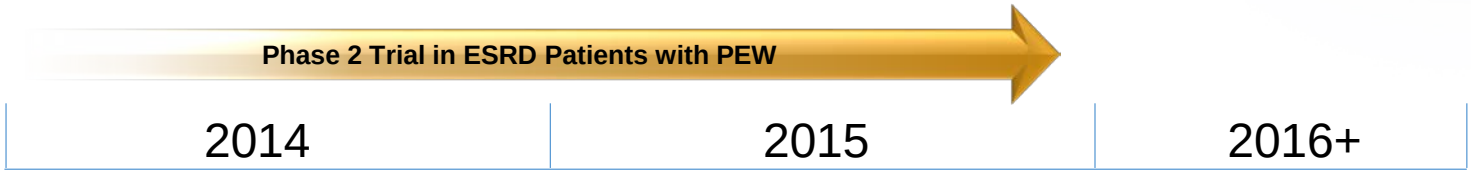
- Large unmet need; **~250,000[1] US; ~800,000 Worldwide**
- ~ 54% of dialysis patients suffer from PEW[2]; Currently treated with supplements
- Associated with increased morbidity and mortality
- Scientific basis for potential efficacy of myostatin inhibition

[1] Calculated utilizing 2011 data from USRDS extrapolated to December 31, 2013 as well as data from the recent study completed with DaVita Clinical Research
[2] Based on a recent study we completed with DaVita Clinical Research, a division of DaVita Healthcare Partners Inc.

PINTA 745: Ongoing Phase 2 Trial in ESRD Patients with PEW

PINTA 745

Phase 2 Data Expected 2H 15



- Randomized (3:1), double-blind, placebo-controlled trial in 48 patients
- Primary endpoint is change in muscle mass; secondary endpoints include physical function, monitoring of inflammatory markers, use of supportive care drugs, and QOL assessments
- Clinical Update
  - No treatment-related serious adverse events, grade ≥ 3 adverse events
  - Initial regimen safe and well tolerated
  - Study enrollment ongoing



STM 434

Ovarian Cancer and Other Solid Tumors

STM 434: Executive Summary



MOA	➢ Ligand Trap that binds activin A: implicated in the proliferation of ovarian cancer (OC) and other solid tumor cells
Indication	➢ Opportunity in difficult-to-treat clear cell and granulosa cell OC and in serous OC in combination with standard regimens ➢ Expansion opportunities in other solid tumors
Proposition	➢ Our preclinical data showed anti-tumor effects in treating OC ➢ Strong scientific basis for utility in other solid tumors
Pre-Clinical Data	➢ STM 434/s inhibited Activin A and reduced tumor size as both a single agent and in combination with chemotherapy ➢ STM 434/s demonstrated efficacy in granulosa and clear cell models of OC
Clinical Status	➢ Ongoing Phase 1 study : dose escalation, dose expansion and combination with chemotherapy; initial Phase 1 data expected 1H:16

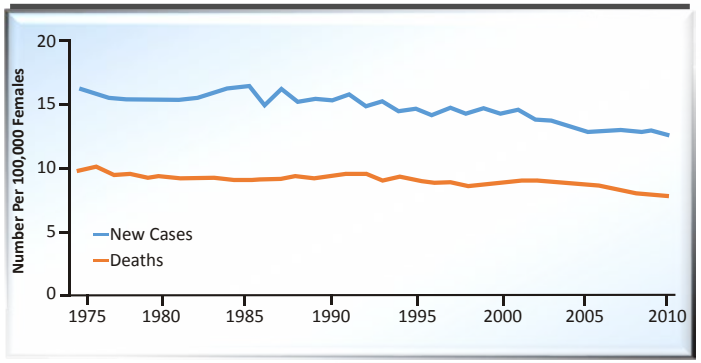
[1] National Cancer Institute SEER Database

Recurrent Ovarian Cancer Represents a Significant Unmet Need

STM 434

- ~22K new ovarian cancer cases and ~14K ovarian cancer deaths in the US in 2013[1]
- Current treatment options for ovarian cancer include surgery and cytotoxic chemotherapies; outcomes have changed little in 40 years

Ovarian Cancer[2] Outcomes Have Not Improved



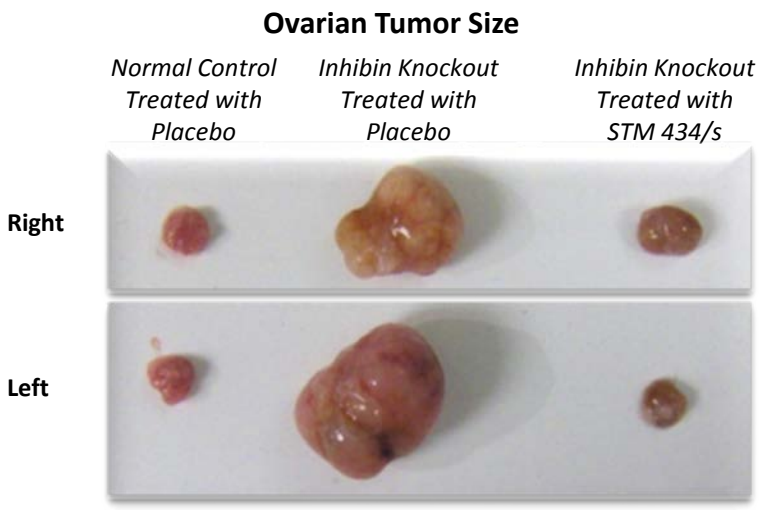
- **Ovarian cancers are divided in 3 main subtypes**
  - Serous adenocarcinoma: ~63% in the US
  - Clear cell cancers: ~11% in Western countries and a higher percentage in Asian countries (e.g. ~23% in Japan)
  - Granulosa cell tumors: ~2 to 5% in the US

[1] National Cancer Institute estimates
[2] Number of new cases and deaths in the US per 100,000 people (all races), age-adjusted

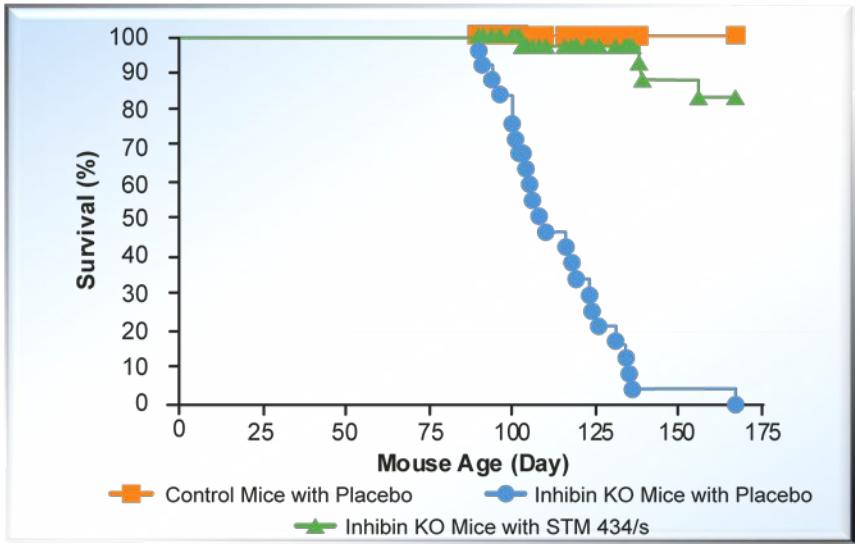
In Preclinical Models of Granulosa Cell Tumors, STM 434/s Treatment Reduces Tumor Burden and Enhances Survival

STM 434

Suppressed Tumor Growth



Improvement in Survival

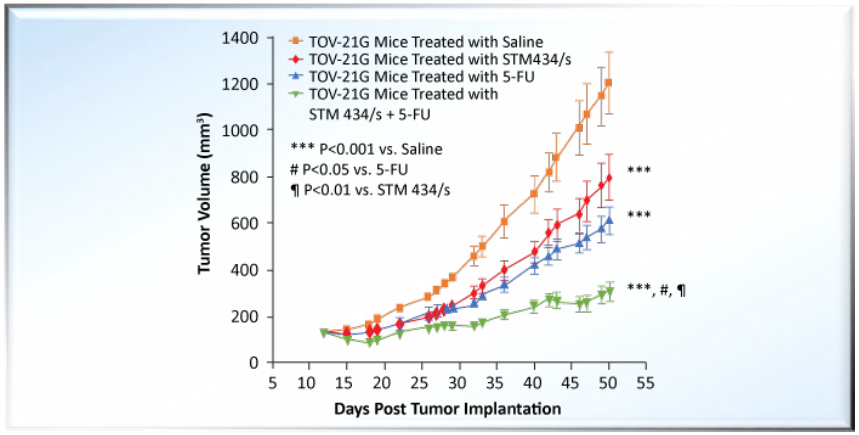


- FOXL2 C134W gene linkage
  - In a normal cell, FOXL2 protein turns on follistatin when an activin signal is received, and follistatin, a natural inhibitor of activin, then shuts off the activin signal
  - In granulosa cell tumors, mutant FOXL2 C134W is not able to turn on follistatin, leaving activin levels unchecked
  - This mutation was present in 97% (86 of 89) of granulosa cell tumors as reported in *The New England Journal of Medicine* (NEJM 2009 360:26)

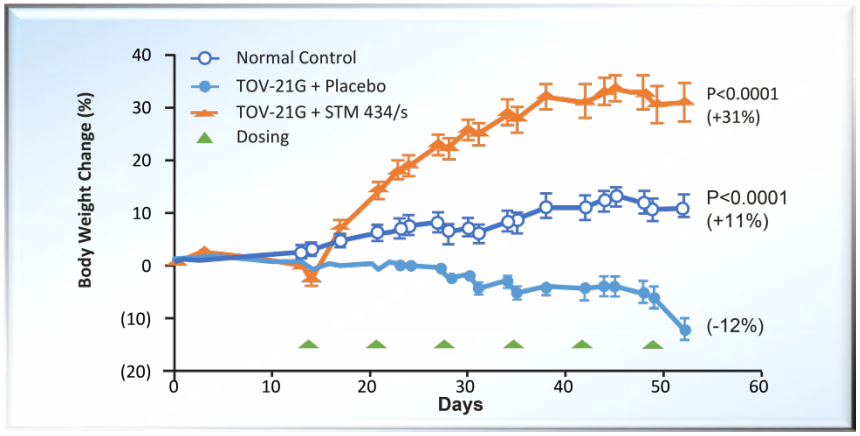
Zhou X, et al. *Cell*. 2010; 142(4):531-43.

In Preclinical Models of Clear Cell Ovarian Tumors, STM 434/s Demonstrates Anti-Tumor Effects

Significant Reduction in Tumor Volume[1]



Enhanced Body Weight[1]



- ARID1A gene linkage
  - Mutations in the ARID1A gene drive upregulation in the signaling cascade triggered by the ActR2B receptor
  - Mutations were present in 55 of 119 (46%) and 17 of 31 (55%) ovarian clear cell tumors as reported in *The New England Journal of Medicine* in 2010
  - We believe that increased levels of activin, like ARID1A mutations, may contribute to tumor proliferation in clear cell ovarian cancer

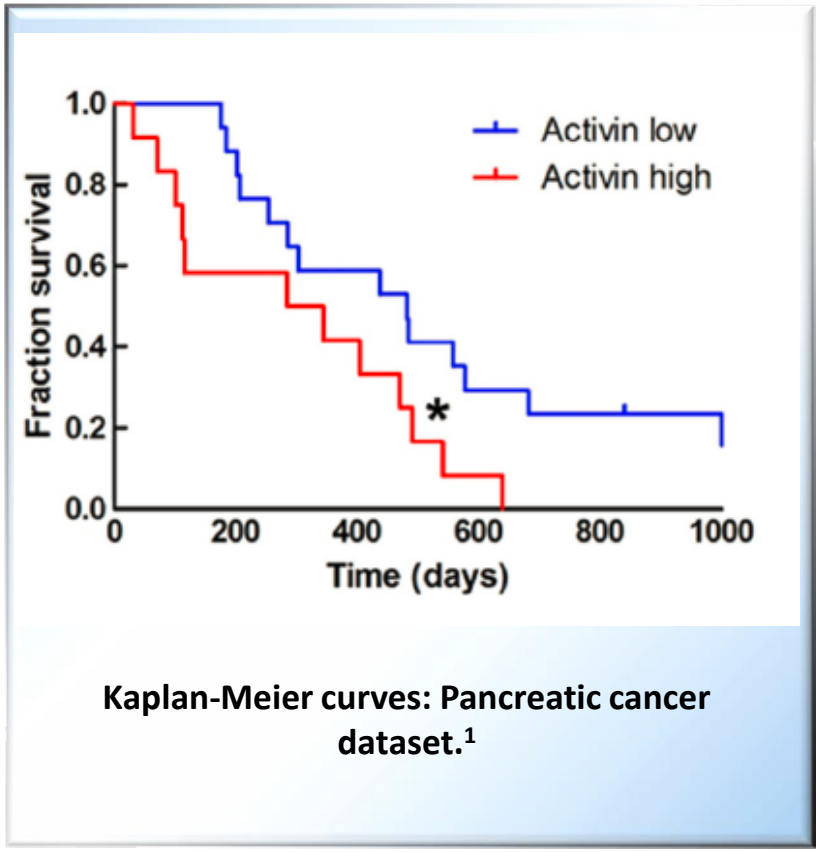
[1] Lu J, Haqq C, & Han HQ. ASCO Annual Meeting (2013)

STM-434: Scientific Rationale - Other Tumor Types

STM 434

Other tumors associated with overexpression of Activin A

- Pancreatic
- Head and neck
- NSCLC
- Gastric
- Colon
- Esophageal



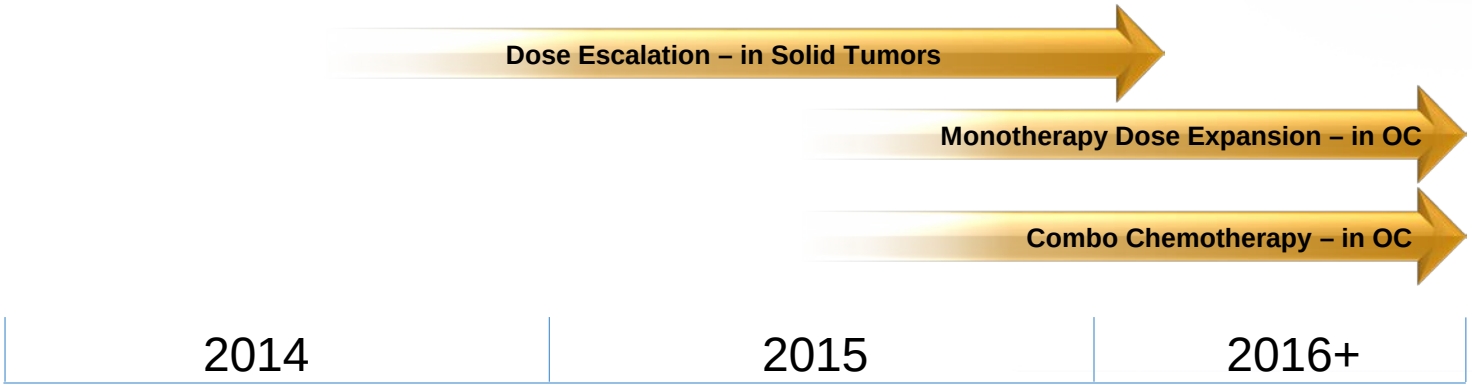
Kaplan-Meier curves: Pancreatic cancer dataset.[1]

1. Togashi et al., 2015 Cancer Letters 356:819 Epub

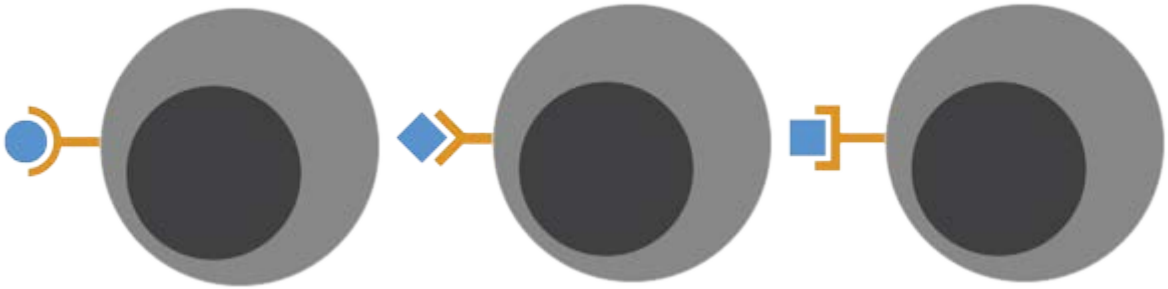
STM 434: First-in-Human (Phase 1) Study

STM 434

First Patient Dosed 10/14; Initial Data Readout Expected 1H 16



- Three part open-label Phase 1 study in up to 66 patients with a once-in-every-four weeks dosing schedule
- Objectives:
  - Test if STM 434 monotherapy is safe and well tolerated
  - Obtain preliminary efficacy data in ovarian cancer and other solid tumors
  - Assess safety and preliminary efficacy of STM 434 with liposomal doxorubicin chemotherapy or the current standard of care
  - Explore biomarkers predictive of response to treatment
  - Define the recommended Phase 2 dose



MSK – Collaboration

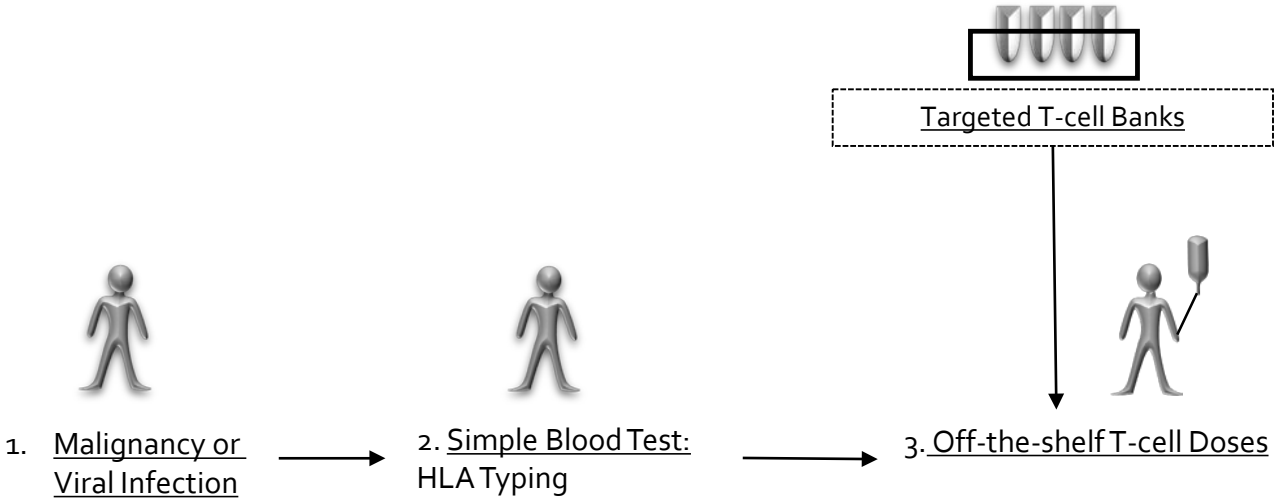
Allogeneic Targeted T cell Therapy
for Cancer and Infectious Diseases

MSK – Collaboration: Executive Summary

Opportunity	➢ Off-the-shelf third-party donor-derived T cells platform technology
Indications	EBV Targeted T cells – EBV-associated lymphoma and other cancers CMV Targeted T cells – CMV Infection or Persistent CMV Viremia WT1 Targeted T cells – Hematologic and solid tumors
Proposition	➢ Demonstrated efficacy in patients refractory to current treatment ➢ Response rate of ~68% in patients with EBV associated lymphoma (*Blood* 2012) ➢ Response rate of ~60% in patients with refractory CMV viremia or disease (ASH 2014)
Clinical Status	➢ MSK sponsored clinical trials ongoing for all three programs ➢ EBV and CMV programs currently in Phase 2 ➢ WT1 program currently in Phase 1
Agreement	➢ Exclusive option agreement with right to acquire exclusive, worldwide rights to all three clinical-stage programs ➢ Research collaboration to develop additional cellular therapies

Unique Off-the-shelf Immuno-oncology Platform

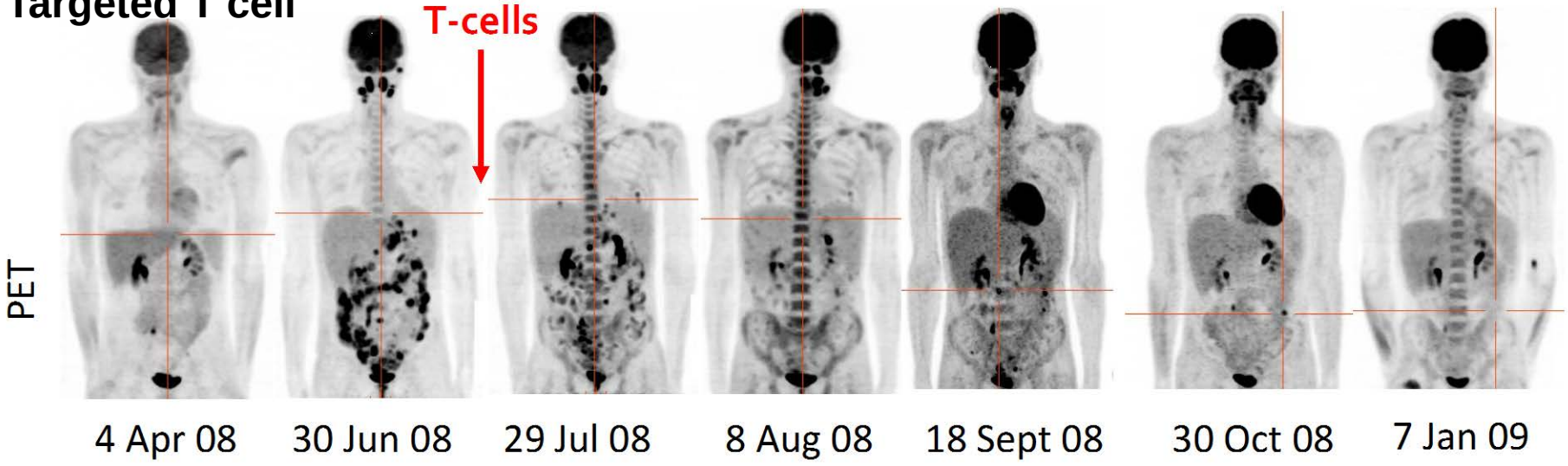
- Manufactured from donor derived activated T cells
  - T cells characterized and stored for future use in appropriate partially HLA[2] matched patient
- Initial applications in adoptive T cell therapy for EBV, CMV, WT1
- Broad potential utility
  - Clinical expansion with existing targeted T cells
  - Leveraging the existing technology to target other antigens
  - Development of additional cellular therapies and /or CAR-T against collaboration targets



Clinical Proof of Concept in EBV – Associated Lymphoma Using MSK T cells[1]

- 19 patients with EBV-associated lymphoma were treated with the EBV targeted T cell therapy
- The complete response rate was 68%, indicating that in 13 of 19 patients no visible evidence of tumor following treatment was observed
- Ten of these 19 patients had previously failed rituximab and had subsequently progressed. Of these ten patients, seven (70%) achieved a complete response

Time Course of a Complete Response Following Administration of EBV Targeted T cell



[1] Doubrovina, E et al., *Blood* (2012)
[2] HLA = Human Leukocyte Antigen

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