ADVAXIS

IMMUNOTHERAPIES™

November 2015 Company Presentation

Forward Looking Statements

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This presentation contains forward-looking statements, including, but not limited to: statements regarding Advaxis's ability to develop the next generation of cancer immunotherapies; and the safety and efficacy of Advaxis's proprietary immunotherapy, axalimogene filolisbac. These forward-looking statements are subject to a number of risks, including the risk factors set forth from time to time in Advaxis's SEC filings, including but not limited to its report on Form 10-K for the fiscal year ended October 31, 2014, which is available at http://www.sec.gov.

Advaxis undertakes no obligation to publicly release the result of any revision to these forward-looking statements, which may be made to reflect the events or circumstances after the date hereof or to reflect the occurrence of unanticipated events, except as required by law. You are cautioned not to place undue reliance on any forward-looking statements.

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Our Company

ADVAXIS OVERVIEW

Advaxis Company Overview

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Background

- Core technology – live attenuated *Listeria monocytogenes* (*Lm*) bacterial vector stimulates the immune system to view tumor cells as bacterial infected cells and target them for elimination
- Alters tumor microenvironment by increasing tumor fighting cells and decreasing tumor protecting cells
- ~50 employees with lab, office, and vivarium located in Princeton, NJ

Financial Snapshot

- Raised ~$165M since October 2013
- Cash: ~$120M as of August 2015

Summary of Strengths

- Extremely versatile platform technology can be used to treat any type of cancer through targeting driver mutations and/or neoepitopes
- Existing collaborations with Merck & Co., Inc.; AstraZeneca/MedImmune, LLC; and Incyte Corp.
- Straightforward and scalable manufacturing process
- Highly proprietary technology (80+ patents) with low royalty obligation (2.5%)

Key Value Drivers

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Lm Technology™ Candidates in Development

- Axalimogene filolisbac – Comprehensive clinical development program in early and late stage HPV-associated cancers
- ADXS-HER2 – Clinical development program in multiple HER2 expressing solid tumors
  - AT-014 for canine osteosarcoma anticipated launch in 2016 (licensed to Aratana/NASDAQ:PETX)
- ADXS-PSA – Clinical development program in metastatic castration-resistant prostate cancer (mCRPC) as monotherapy and in combination with KEYTRUDA®
- Orphan Drug Designations for invasive cervical cancer, head and neck cancer, anal cancer, and osteosarcoma

Preclinical Pipeline

- ADXS-NEO – Neoepitope-based immunotherapy targeting mutations identified in an individual patient's tumor using massive parallel sequencing; IND anticipated mid-2016
- ADXS-TNBC – Triple negative breast cancer; IND anticipated mid-2016
- Other *Lm* Technology™ immunotherapy product candidates targeting tumor driver mutations, including *survivin, ISG15, PSCA, WT1, and others*

Combination with Other Cancer Therapies

- Synergistic response with checkpoint inhibitors (PD-1 and PD-L1) and costimulatory molecules (OX40 and GITR) in preclinical models
- Enhanced response in combination with radiation in prostate cancer models

Experienced Management Team

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Daniel O'Connor
Chief Executive Officer

Gregory Mayes
Chief Operating Officer

David Mauro, MD, Ph.D.
Chief Medical Officer

Robert Petit, Ph.D.
Chief Scientific Officer

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Sara Bonstein, MBA
Chief Financial Officer

Chris French, MBA
VP, Compliance

Mayo Pujols
VP, Manufacturing

Robert Ashworth
VP, Regulatory

Tom Hare
VP, Clinical Operations

Strong Collaborations with Academic Institutions and Foundations

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Our Immunotherapy

LM TECHNOLOGY™

Lm Technology™ Overview:
Harnessing Unique Life Cycle of *Lm* in APCs

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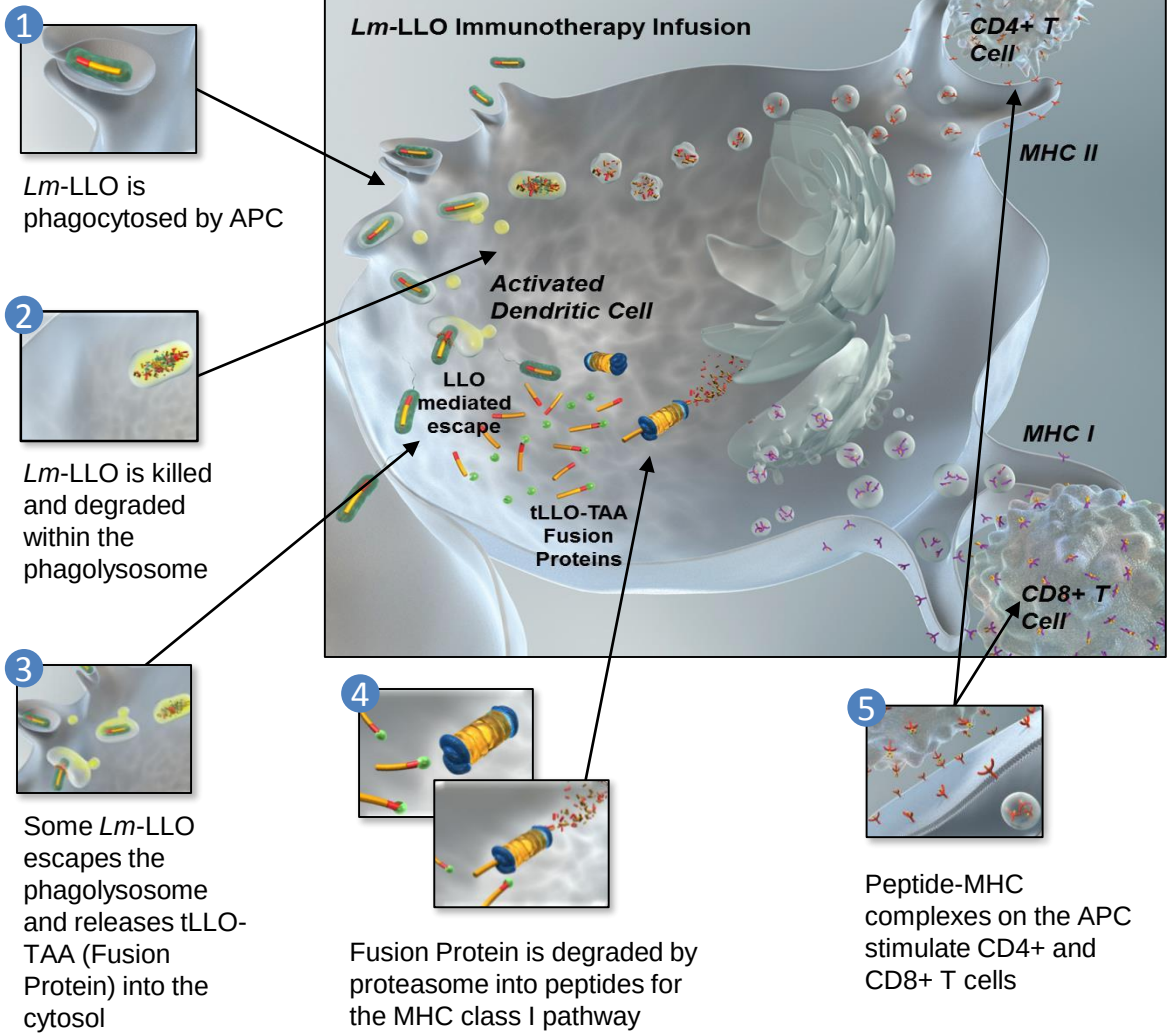

Summary

- *Lm*-LLO and Tumor Associated Antigen (TAA) presented and taken up by dendritic cells (antigen presenting cells or APCs)
- Dendritic cells activated and generate an immune response through both the MHC I and MHC II pathways
- Robust T-cell response generated towards antigen secreted by *Lm*-LLO and redirected to tumors expressing the same TAA
- "Perceived" acute infection stimulates a strong innate immune response through multiple pathways (e.g. STING)
- Over-rides checkpoint inhibitors and negative regulators of cellular immunity

MHC, major histocompatibility complex

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Our Immunotherapy

IMPACT ON THE TUMOR MICROENVIRONMENT

Lm Technology™ & Tumor Microenvironment: Suppressing Treg and MDSC Activity

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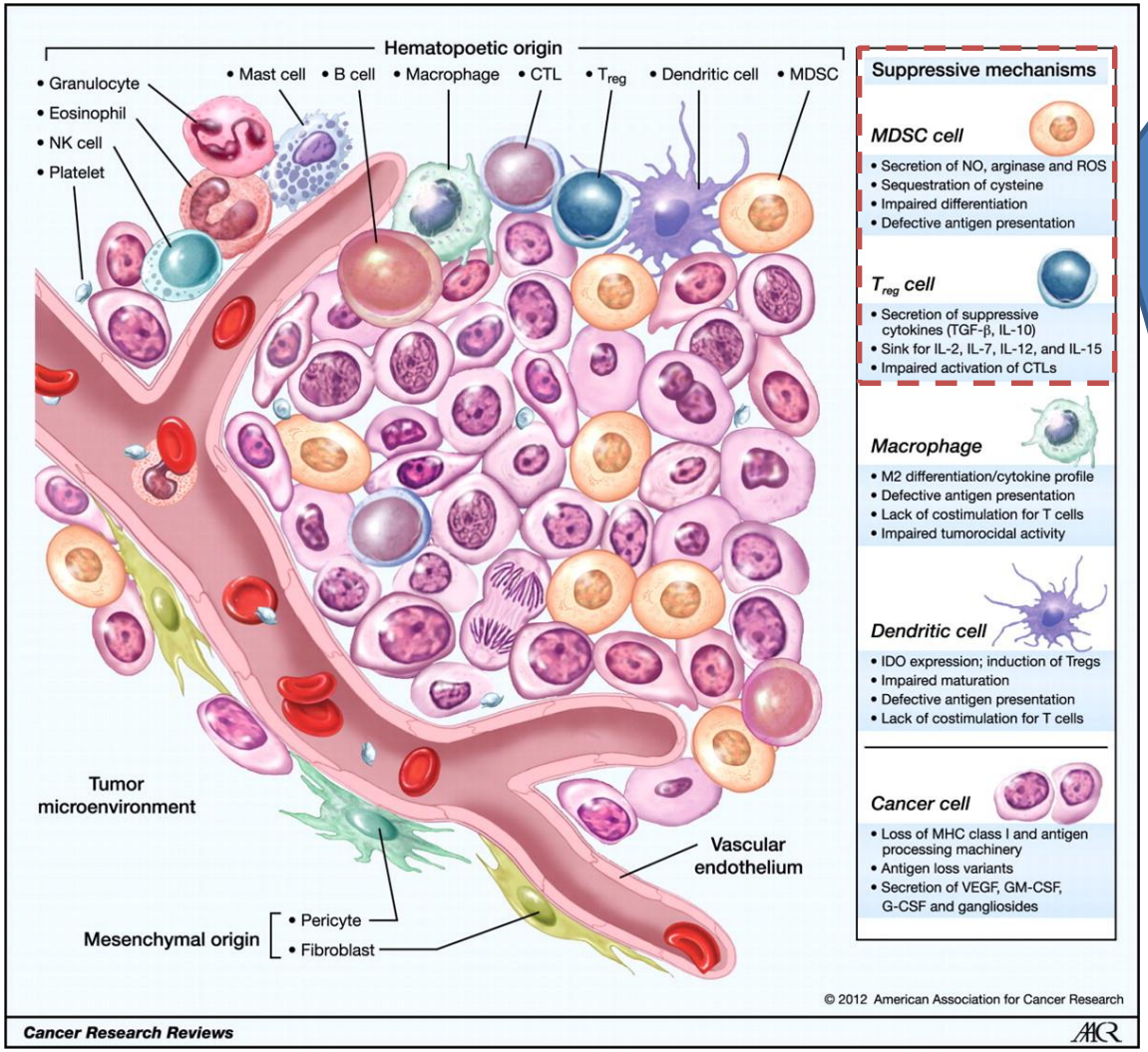

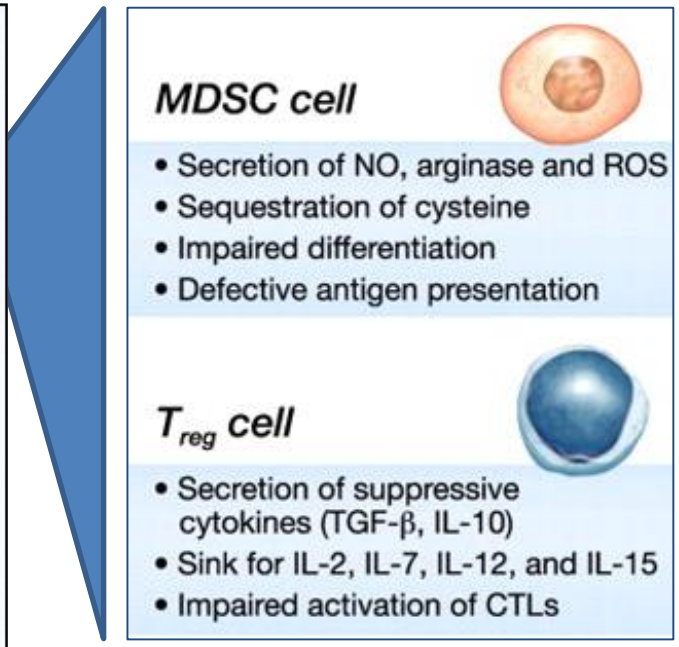

- **Tregs and MDSCs are the key cells that tumors manipulate to avoid detection by the immune system and evade destruction**
- ***Lm-LLO*, Advaxis's proprietary technology, decreases the relative % of Tregs and MDSCs in the TME**

Sid P. Kerkar, and Nicholas P. Restifo. Cancer Res 2012;72:3125-3130

Lm Technology™ & Tumor Microenvironment: Suppressing Treg and MDSC Activity (Preclinical)

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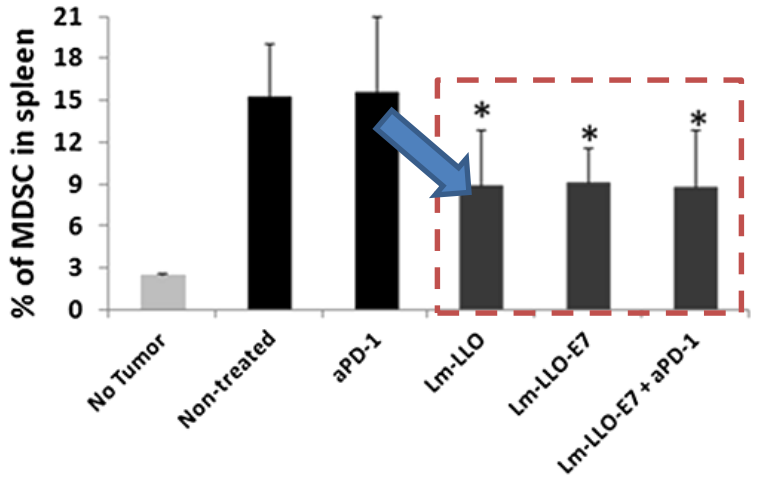
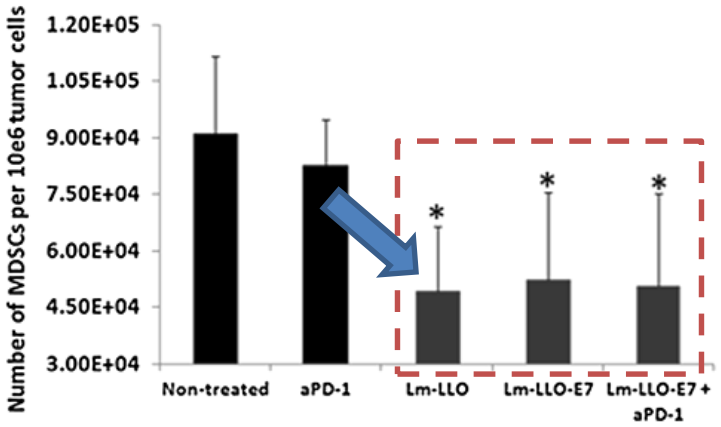
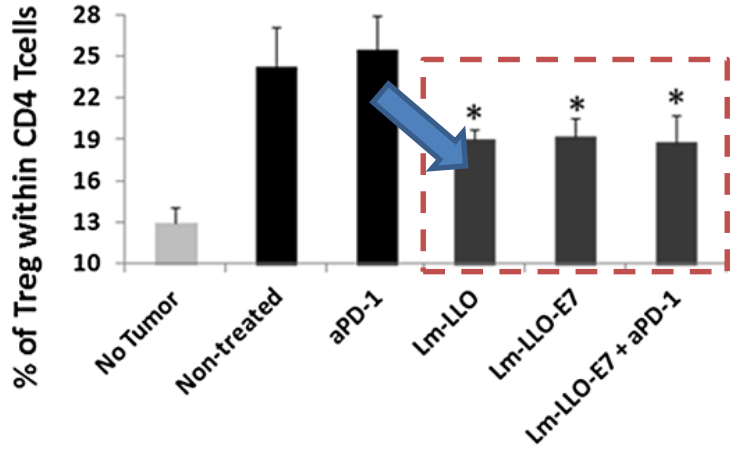
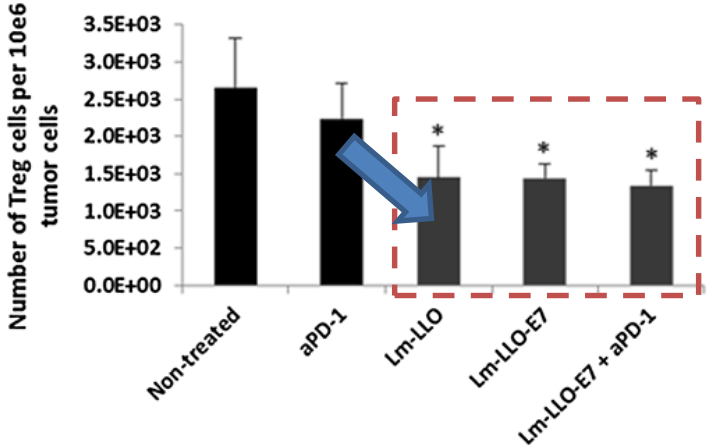
Lm-LLO is solely responsible for ↓MDSCs





Lm-LLO is solely responsible for ↓Tregs





Mkrtichyan M et al. Anti-PD-1 antibody significantly increases therapeutic efficacy of Listeria monocytogenes (Lm)-LLO immunotherapy. Journal for ImmunoTherapy of Cancer 2013; 1: 15.

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SYNERGY WITH CHECKPOINT INHIBITORS & COSTIMULATORY MOLECULES

Lm Technology™ & Checkpoint Inhibitors: Synergistic Tumor Killing in the Microenvironment

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CTLA-4[1] block results in:

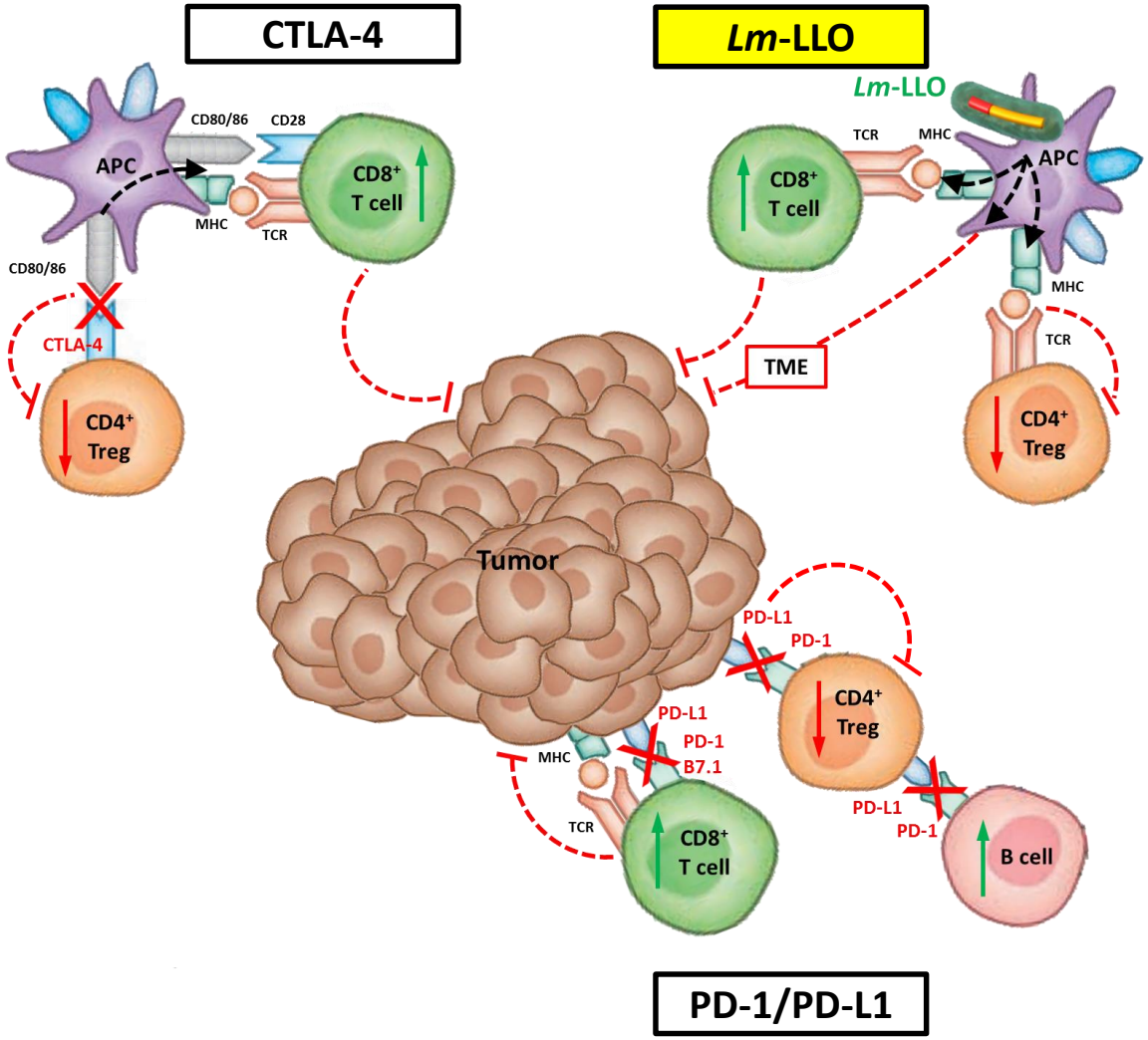
- Down-regulation of $CD4^+$ Tregs
- Upregulation of $CD8^+$ tumor infiltrating effector T cells

PD-1[1] block results in:

- Recognition of tumor cells by T cells
- Proliferation and function of $CD8^+$ tumor infiltrating effector T cells

PD-L1[1] block results in:

- Recognition of tumor cells by T cells
- Proliferation and function $CD8^+$ tumor infiltrating effector T cells
- No $CD4^+$ Treg activation
- Increase in B cell antibody production



APC, antigen presenting cell; TME, tumor microenvironment; 1. Okazaki et al. Nat Immunol. 2013 Dec;14(12):1212-8

14

Lm Technology™ & Checkpoint Inhibitors: Axalimogene Filolisbac & PD-1 Preclinical Data

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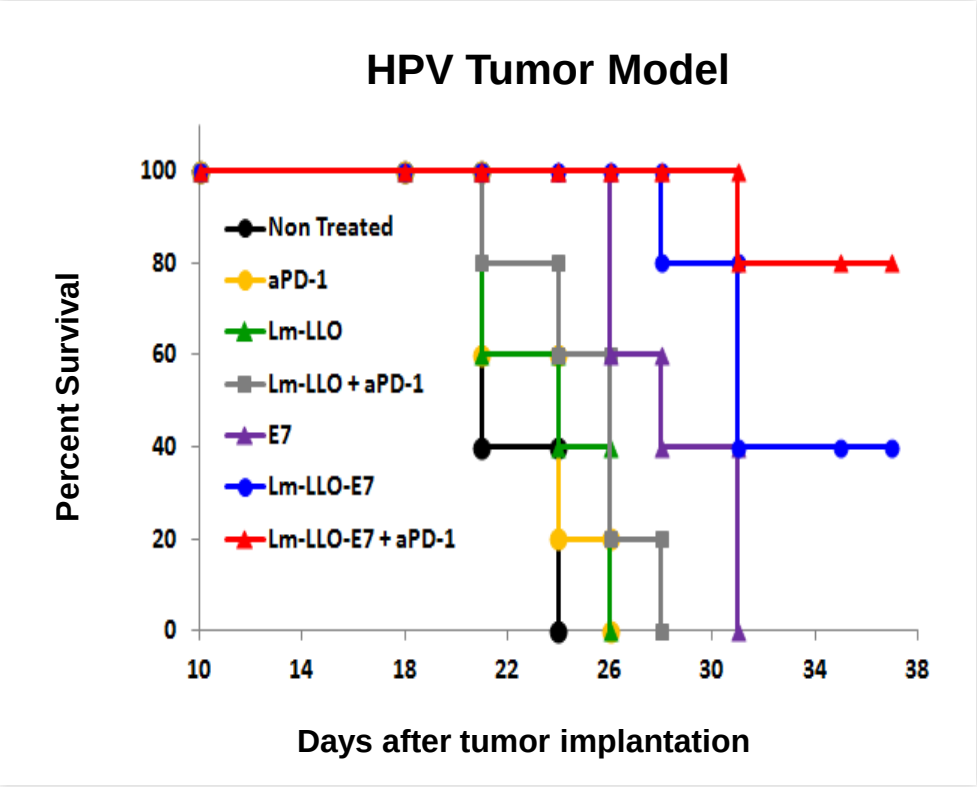

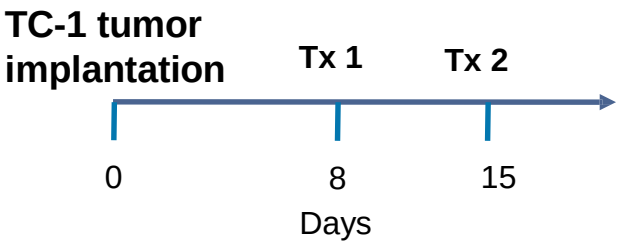

Treatments:	
Lm-LLO-E7:	5x10^6 cfu
CT-011 mAb:	50 µg

Low dose *Lm*-LLO immunotherapy can be combined with a checkpoint inhibitor

Mkrtichyan M et al. Anti-PD-1 antibody significantly increases therapeutic efficacy of Listeria monocytogenes (Lm)-LLO immunotherapy. Journal for ImmunoTherapy of Cancer 2013; 1: 15.

Lm Technology™ & Costimulatory Molecules: Improved Targeting and Priming of CTLs

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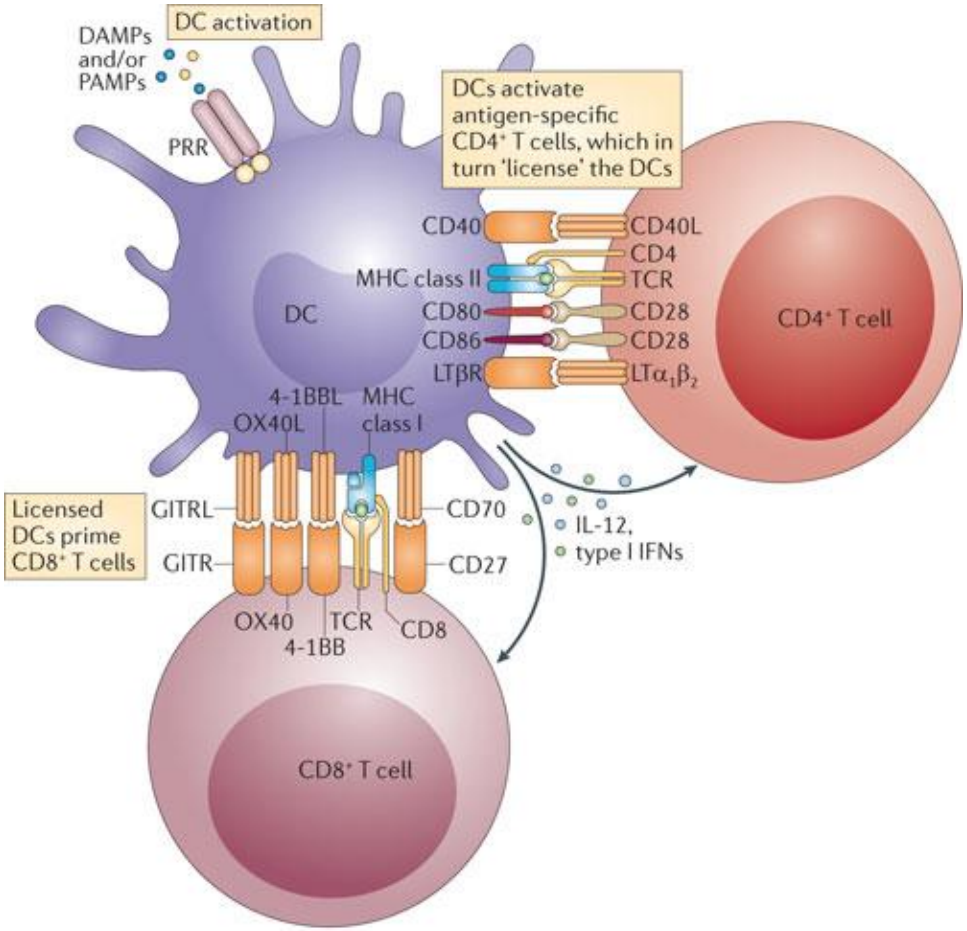
Role of Costimulatory Molecules in the Adaptive Immune Response

- DCs are activated by pathogen-associated molecular patterns (PAMPs)
- CD40 signaling results in the production of IL-12 and upregulation of OX40 ligand (OX40L) and GITR ligand (GITRL)
- Priming of CD8+ T cells by MHC class I peptides upregulates OX40 and GITR
- Stimulation of OX40 and GITR generates robust CD8+ T cell activation, proliferation, and effector function



Lm Technology™ & Co-Stimulatory Molecules: Axalimogene Filolisbac & GITR or OX40 Antibodies

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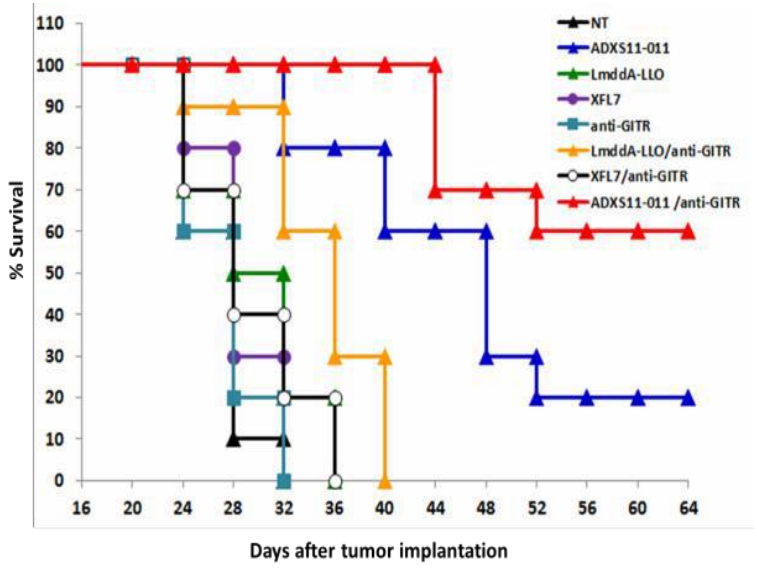
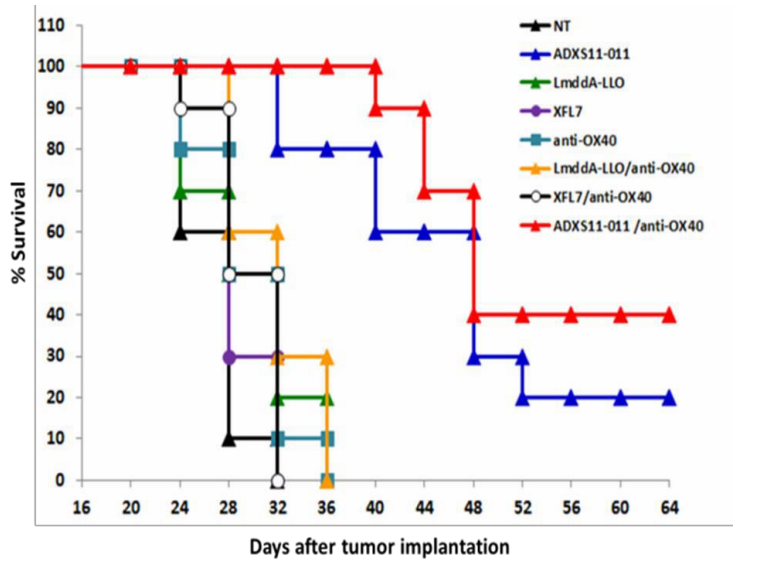
HPV Tumor Model

Axalimogene filolisbac + Anti-GITR



Axalimogene filolisbac + Anti-OX40



***Lm*-LLO immunotherapy can be combined with agonistic antibodies to immune co-stimulatory molecules**

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Our Clinical Trials

TIMELINES, DESIGN, AND RESULTS

Clinical Development
Three Active Programs with 4 Orphan Indications

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Product		Indication	Phase I	Phase 2	Phase 3	Partner
Axalimogene Filolisbac (ADXS-HPV)		**Cervical Cancer***				
	M	AIM2CERV – Adjuvant Randomized vs. Placebo			Phase 3	GOG
	M	Metastatic – Randomized vs Cisplatin/Axalimogene Filolisbac	Phase 1	Phase 2		
	M	Metastatic – GOG-0265		Phase 2		GOG
	M	Metastatic – Single Arm High Dose	Phase 1/2			
	C	Metastatic – Combo with durvalumab (MEDI4736)	Phase 1/2			MedImmune
	C	Stage I-IIa – Combo with epacadostat (INCB24360)		Phase 2		Incyte
		Head and Neck Cancer*				
	M	Neoadjuvant – Window of Opportunity		Phase 2		Mount Sinai
	C	Metastatic – Combo with durvalumab (MEDI4736)	Phase 1/2			MedImmune
		Anal Cancer*				
	M	Adjuvant Randomized vs Control		Phase 2/3		RTOG
	M	Adjuvant – Single Arm High Risk	Phase 1/2			BROWN
	M	Metastatic – Single Arm (FAWCETT)		Phase 2		
ADXS-PSA		**Prostate Cancer***				
	C	Metastatic – Combo with KEYTRUDA® (pembrolizumab)	Phase 1/2			MERCK
ADXS-HER2		**HER2-positive Solid Tumors (including Osteosarcoma*)**				
	M	Metastatic – Single Arm	Phase 1			
	M	Pediatric Osteosarcoma		Phase 2		CHILDREN'S ONCOLOGY GROUP

M Monotherapy C Combination In Progress (FDA accepted IND and/or ongoing trial Planned

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AXALIMOGENE FILOLISBAC: COMPANY SPONSORED PHASE 2 STUDY

Axalimogene Filolisbac Phase 2 Study
Study Schema—Recurrent Cervical Cancer

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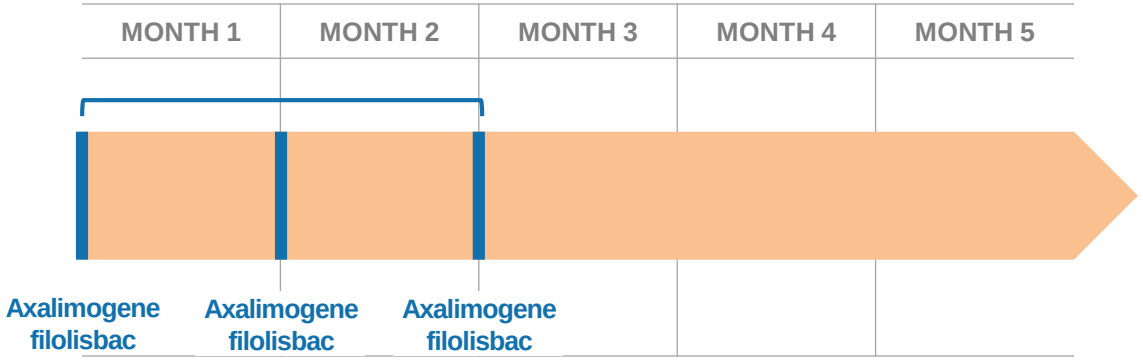
Primary Efficacy Endpoint: Overall Survival

ARM A

Axalimogene Filolisbac Monotherapy
$1x10^9$ cfu x 3 doses q 28 days (days 0, 28, 56) as an 80 ml infusion over 15 min

N = 56

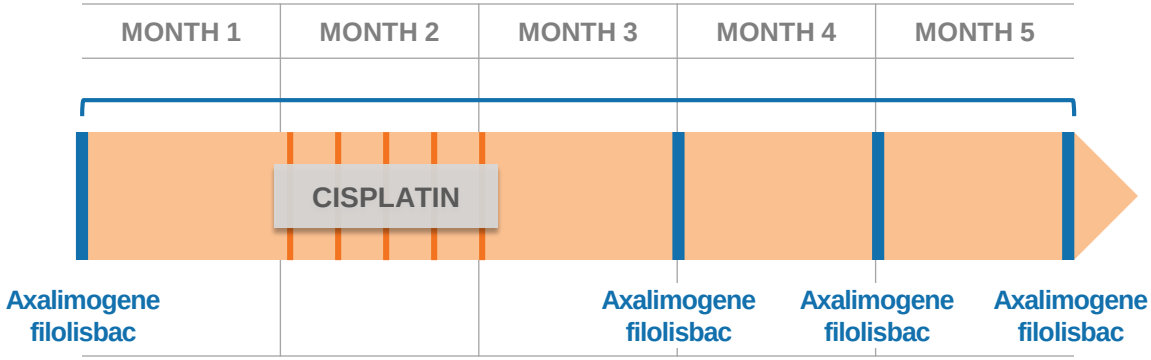


ARM B

Axalimogene Filolisbac + Cisplatin
$1x10^9$ cfu x 4 doses q 28 days (days 0, 88, 106, 134) as an 80 ml infusion over 15 min

Cisplatin 40 mg/m^2 weekly x 5 (days 30, 37, 44, 51, 58)

N = 54



Naproxen 500 mg BID (day -1, 0) and promethazine 25 mg BID (pre-dose 8 hours) administered as premedications Ampicillin 500 mg QID (days 3-9) administered post-infusion

Axalimogene Filolisbac Phase 2 Study
Safety in Recurrent Cervical Cancer

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109 patients received 264 doses of axalimogene filolisbac at 1x10^9 cfu	(N=109)
Grade 1-2 AEs (76 patients reported)	**41 (38%)**
Chills/Shivering	41 (38%)
Flu-Like Symptom	13 (12%)
Vomiting	6 (6%)
Nausea	5 (5%)
Fever	5 (5%)
Dizziness	2 (2%)
Cytokine Release Syndrome	1 (1%)
Headache	1 (1%)
Weight Decreased	1 (1%)
Blood Alkaline Phosphatase Increased	1 (1%)
Grade 3 AE (1 patient reported)	
Fever	1 (1%)

Axalimogene Filolisbac Phase 2 Study
Efficacy in Recurrent Cervical Cancer

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Survival Analyses at 12, 18 and >24 Months

Patients	Overall (N=109)	Axalimogene Filolisbac Alone (N=55)	Axalimogene Filolisbac + CISPLATIN (N=54)
12-Month Survival	32% (35 / 109)	29% (16 / 55)	35% (19 / 54)
18-Month Survival	22% (24 / 109)	22% (12 / 55)	22% (12 / 54)
≥ 24-Month Survival	18% (16 / 91*)	15% (7 / 46)	20% (9 / 45)

- There was no statistically significant difference in median overall survival between axalimogene filolisbac alone or in combination with cisplatin
- Tumor response was comparable between treatment groups at 12, 18, and 24 months
- The objective response rate in both arms was 10% and the disease control rate was 38%

* >24 month survival rate is based on 16 known to be alive out of 91 patients from the OS efficacy population with at least 24 months of documented follow-up data

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Our Clinical Trials

AXALIMOGENE FILOLISBAC: GOG-0265 STUDY

Axalimogene Filolisbac: GOG-0265 Study
Study Schema—Phase II Open Label, 2-Stage

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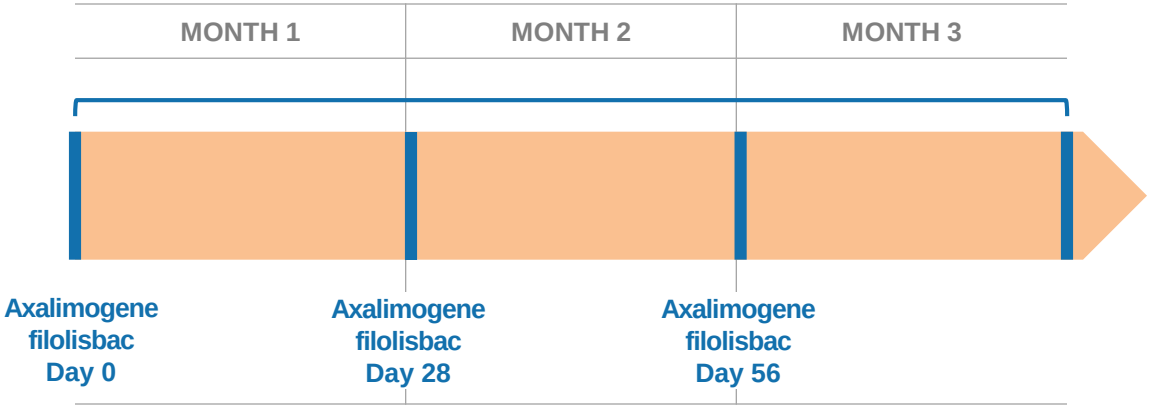
Primary Efficacy Endpoint: 12-month survival

Axalimogene Filolisbac Monotherapy in Recurrent Cervical Cancer

$1x10^9$ cfu x 3 doses q 28 days (month 1, 2, 3) as an 80 ml infusion over 15 min

N = ~67 (Stage 1 and 2)

- Persistent or recurrent metastatic cervical cancer (PRmCC)
- ≥ 1 prior chemotherapy regimen for PRmCC, excluding that received as a component of primary treatment
- GOG PS 0/1
- Measurable disease ≥ 1 target lesion (RECIST 1.1)



(Tewari KS, Monk BJ. *Curr Oncol Rep* 2005; 7(6):419-34.)

GOG, Gynecologic Oncology Group

Axalimogene Filolisbac: GOG-0265 Study
Stage 1 Final Data (September 2015)

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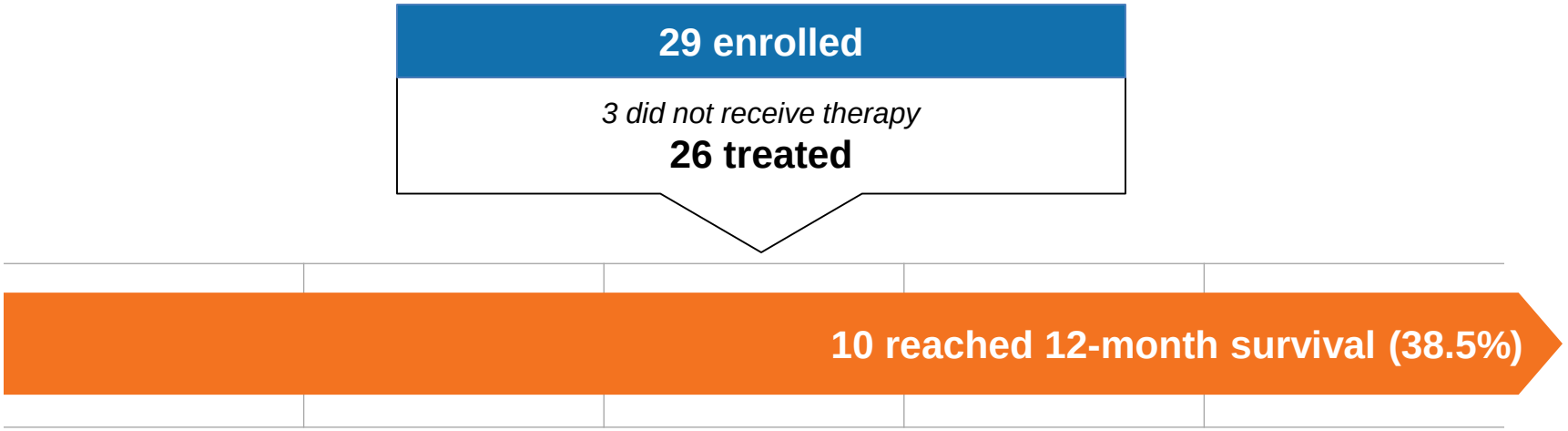

- **Met statistically pre-defined 24.5%12-month survival efficacy threshold* and has proceeded to Stage 2 additional enrollment of n=37**

Adverse Event Summary (N = 26 patients)	
Pts with ≥ 1 Adverse Event (AE)	26 (100%)
Pts with ≥ 1 treatment-related AE	24 (92%)
Grade 1 - 2 only	19 (73%)
Grade 3	4 (15%)
Grade 4	1 (4%)

* Threshold derived from pooled review of 12-month survival rates in completed GOG phase II trials in similar patient population

Axalimogene Filolisbac: GOG-0265 Study
Stage 1 Final Data—Overall Survival

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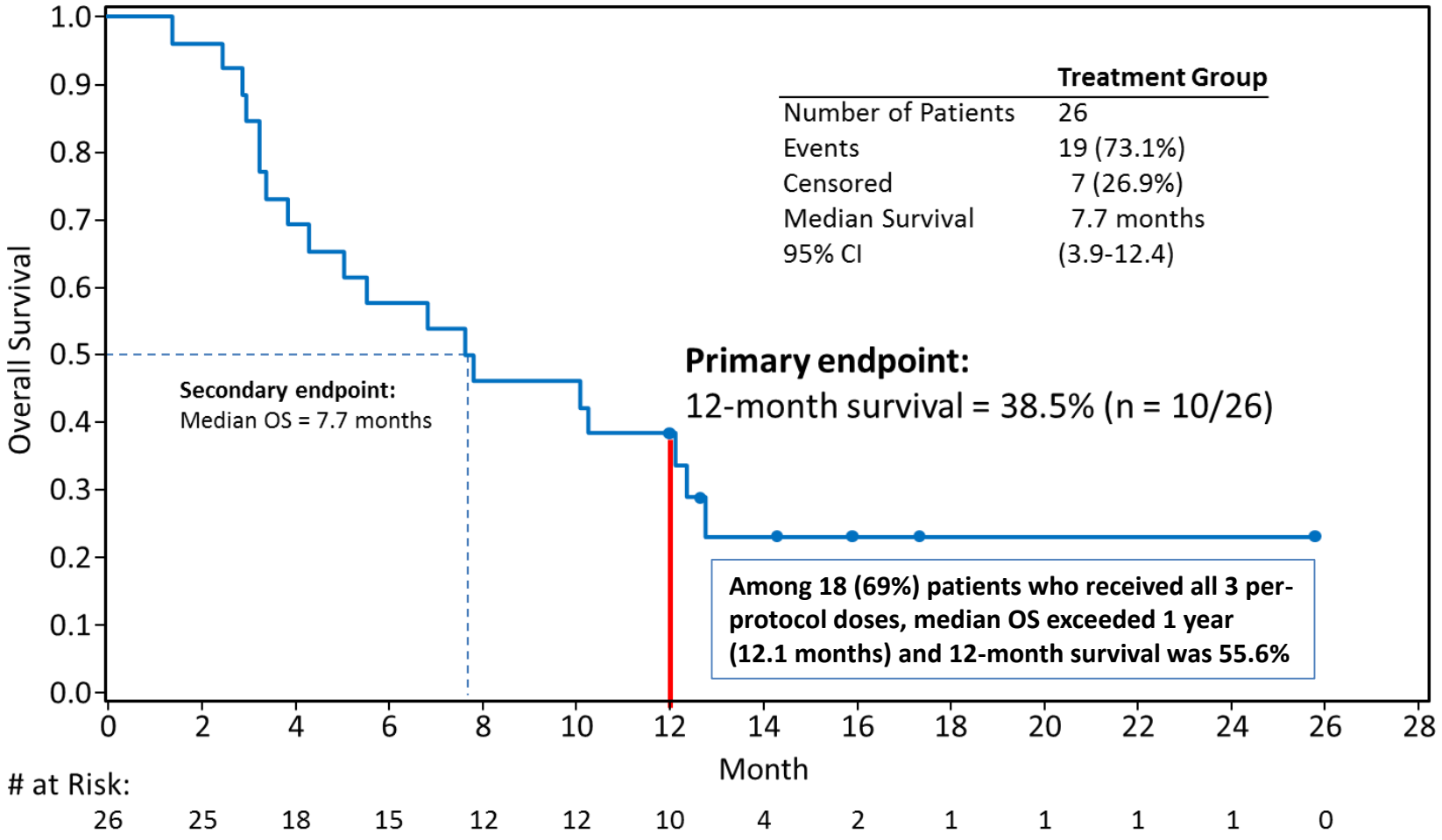

Herzog T et al. Presented at AGOS 2015

Axalimogene Filolisbac: GOG-0265 Study
In the Context of Historical 12-Month Survival Rates

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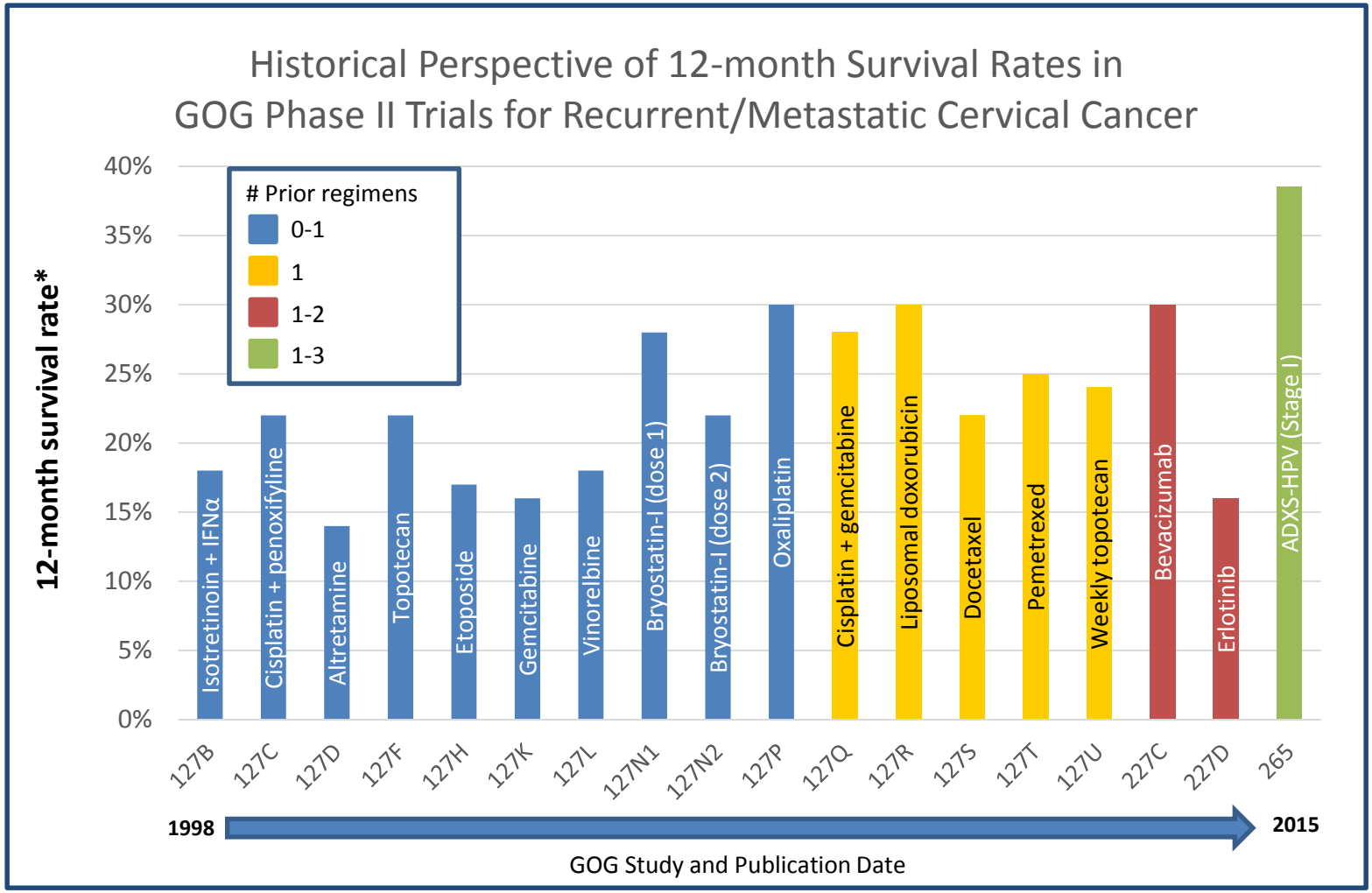

*derived from product limit estimate of probability of surviving >12 months

Clinical Significance of GOG-0265 Stage 1

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Historical Perspective

- Between 1998 and 2015, the GOG conducted serial single arm two-stage trials of various monotherapy and combination regimens in recurrent persistent metastatic cervical cancer
- Trial inclusion criteria has evolved from initially restricting to 0 or 1 prior regimens in the metastatic setting, to most recent inclusion of up to 3 or more prior therapies
- Prior to 2015, the 12-month survival rate in a GOG study has never exceeded 30%
  - When isolating only studies including ≥ 1 prior regimen, this value is ~15-20%, and informed the GOG 0265 Stage I → Stage 2 minimum threshold

Current Standard of Care

- First-line treatment of recurrent persistent metastatic cervical cancer is cisplatin-based chemotherapy, plus bevacizumab in those patients appropriate for anti-angiogenic therapy
  - Trials studying second-line and later therapy now need to address management of the chemotherapy/bevacizumab pretreated population

GOG-0265

- GOG 0265 study includes a more refractory patient population (1 – 3 prior regimens) than previous trials and includes post-progression chemotherapy / bevacizumab patients
- As of June 2015, 10 (38.5%) patients treated with ADXS-HPV have reached 12-months survival, exceeding historical survival rates in a refractory and heterogeneous patient population

Axalimogene Filolisbac: GOG-0265 Study
Next Steps

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- GOG-0265 opened to stage 2 enrollment of an additional 37 patients
  - Stage I efficacy threshold met (24.5% 12-month survival)
  - Accrual update: 44/67 (including stage 1)

- Safety and early efficacy findings support protocol amendment to allow continuous cycles of ADXS11-001 treatment until disease progression

- An international Advaxis-sponsored Phase 3 study of ADXS11-001 as adjuvant treatment of high-risk locally advanced cervical cancer (AIM2CERV) is under development in collaboration with the GOG Foundation

Axalimogene Filolisbac: Planned Phase 3 Study Schema—ADXS AIM2CERV Study

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Randomization 1:2 between Reference and Treatment Groups

Primary objective is progression free survival

- High risk
- FIGO stage I-II with positive pelvic nodes
- FIGO stage III-IV
- Any FIGO stage with para-aortic nodes

Cisplatin (at least 4 weeks exposure) and
Radiation (minimum 40 Gy external beam radiation therapy)

RANDOMIZE

Reference Group

Placebo IV Up to 1 year

Treatment Group

ADXS11-001
(1 x 10^9 cfu) Up to 1 year

Follow-up for overall survival

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Our Clinical Trials

AXALIMOGENE FILOLISBAC: ANAL CANCER

Axalimogene Filolisbac: Phase 1/2 Anal Cancer Study Schema—Open Label Combo at BrUOG

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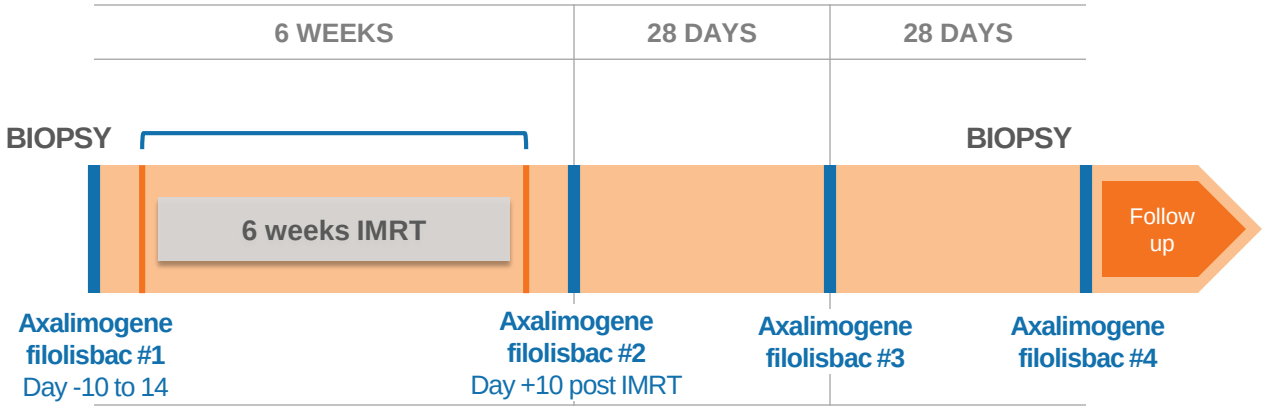
Primary Efficacy Endpoint: 6-month CR-rate

Axalimogene Filolisbac

$1x10^9$ cfu x 4 (1 prior to chemoRT and 3 post, q 28 days) as a 500 ml infusion over 30 min

- N = 25
- Primary stage II-III anal cancer
- High risk of recurrence
- HPV-positive



IMRT = 5-FU, Mitocyin, and Radiation

BrUOG, Brown University Oncology Group

Perez K et al. IANS 2015; Abstract 23

Axalimogene Filolisbac: Phase 1/2 Anal Cancer Preliminary Data

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Study open: April 2013

N = 10 / 25 patients enrolled

Efficacy Summary as of March 2015:

- 10 patients received study treatment
- All patients who have completed treatment achieved CR
- No patient has developed recurrence
  - *Historical 3-year recurrence rate in similar patient population = ~45%*
- Follow-up range: 0.5 months – 24 months

Safety Summary as of March 2015:

- Chills, occasional rigors, flu-like symptoms → resolved prior to leaving clinic (~2 hours)

Axalimogene Filolisbac: Planned Phase 2/3
NRG/RTOG Study for Locally Advanced Cancer

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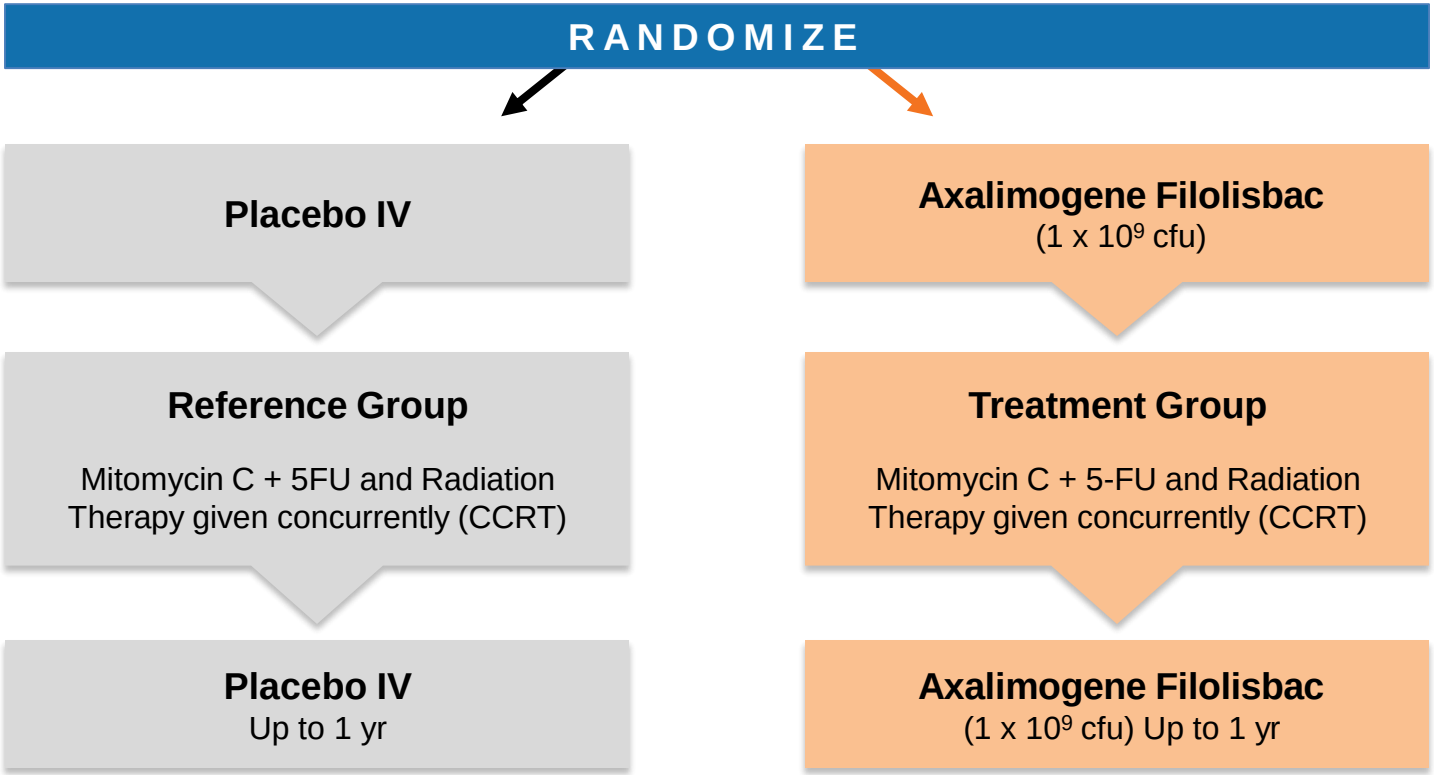
High Risk, Locally Advanced Anal Cancer



Study design is currently being proposed to NCI CTEP

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Our Clinical Trials

ADXS-HER2

ADXS-HER2
Phase 1 Study in Canine Osteosarcoma (UPENN)

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Study Goals:

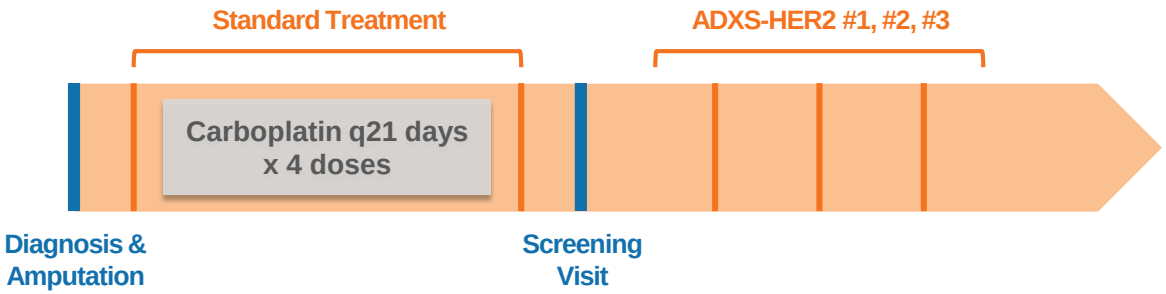
- Identify MTD
- Safety
- Tumor-specific immunity
- Prevention of metastases
- Prolongation of survival

ADXS-HER2

4 dose levels tested:

$2x10^8$ cfu
$5x10^8$ cfu
$1x10^9$ cfu
$3x10^9$ cfu

- N = 18 dogs
- Canine osteosarcoma (OSA)
- Post amputation and chemotherapy



UPENN, University of Pennsylvania

* Paolini M., BMC Genomics, 2009

ADXS-HER2
Phase 1 Study in Canine Osteosarcoma: Safety & Efficacy

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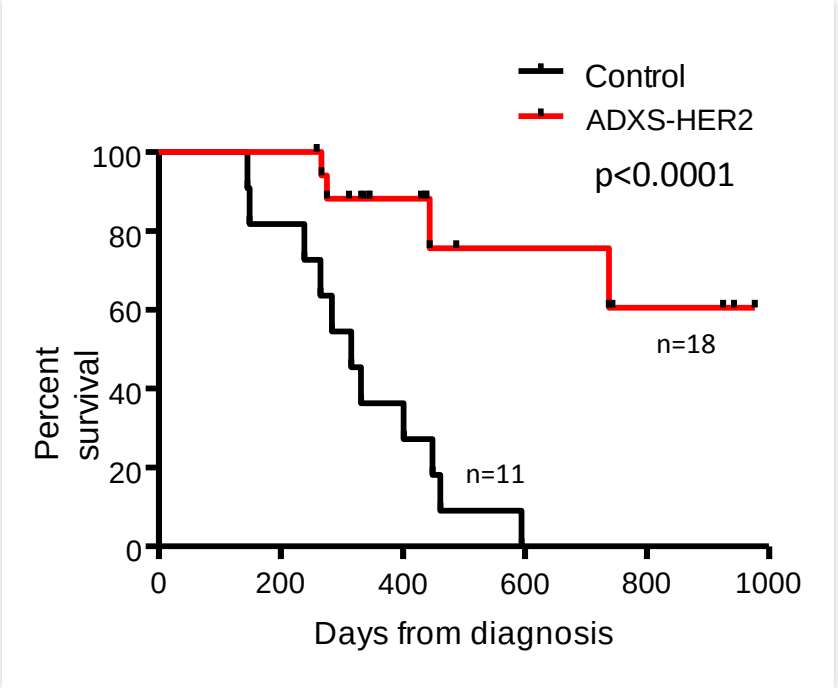
Pet Dogs with Treatment Related Adverse Events
(All toxicities reported are Grade 1)

ADXS-HER2 Dose	$2x10^8$	$5x10^8$	$1x10^9$	$3x10^9$	Total
Number of dogs recruited	N=3	N=3	N=9	N=3	N=18
General Disorders					
Pyrexia (>103)	2	1	5	2	10
Fatigue	1	1	7	2	11
GI Disorders					
Vomiting	2	1	8	1	12
Nausea	2	1	9	2	14
Cardiovascular					
Arrhythmias	0	1	1	1	3
Tachycardia	0	0	1	1	2
Hypotension	0	0	0	0	0
Hematological parameters					
Thrombocytopenia	0	0	5	0	5
Biochemical parameters (increase)					
γ-GT	0	2	0	0	2
Alkaline Phosphatase	1	1	4	1	7
ALT	1	1	1	0	3
AST	1	1	5	1	8
BUN	0	0	0	0	0
CREA	0	0	0	0	0
Cardiac Troponin I	0	0	1	0	1

ADXS-HER2 and Overall Survival
Median survival: Case-matched control: 316 days
ADXS: not yet reached



2 dogs censored from ADXS arm, deaths unrelated to OSA

Next Steps: Pending approval USDA for veterinary use; Currently under investigation in combination with RT in OSA

ADXS-HER2
Combo with Radiation in Untreated Canine Osteosarcoma

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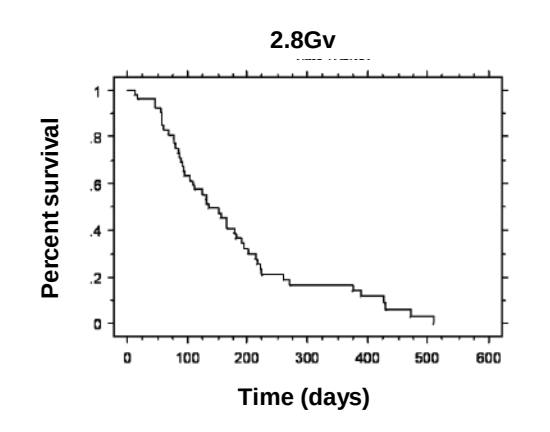
Radiation alone

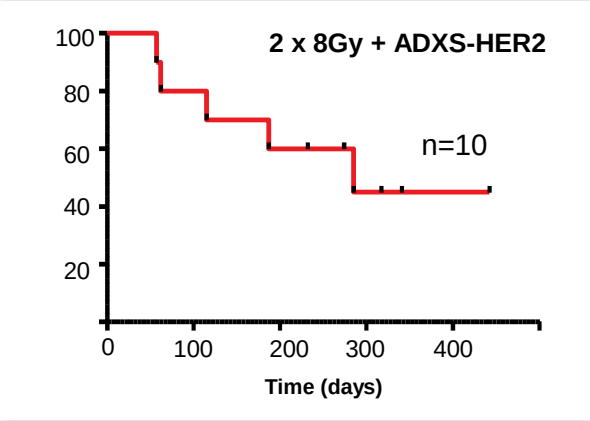


Historical Perspective:
Knapp-Hoch et al. J Am Anim Hosp Assoc. 2009 Jan-Feb;45(1):24-32.

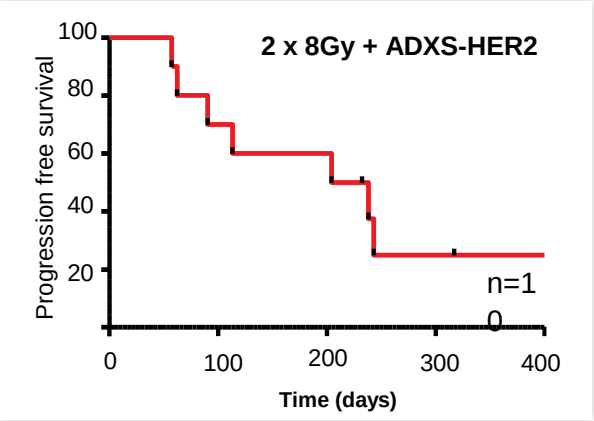
Median OS = ~120 days
(expected OS range for dogs that cannot undergo amputation and receive only palliative radiation and analgesics is 3 - 5 months)

ADXS-HER2 and Radiation: N = 10 pet dogs with untreated primary OSA

Radiation plus ADXS-HER2



Median OS = 285 days



Median TTP = 221 days

ADXS-HER2 Human Study
Phase Ib Dose-Escalation in HER2+ Solid Tumors

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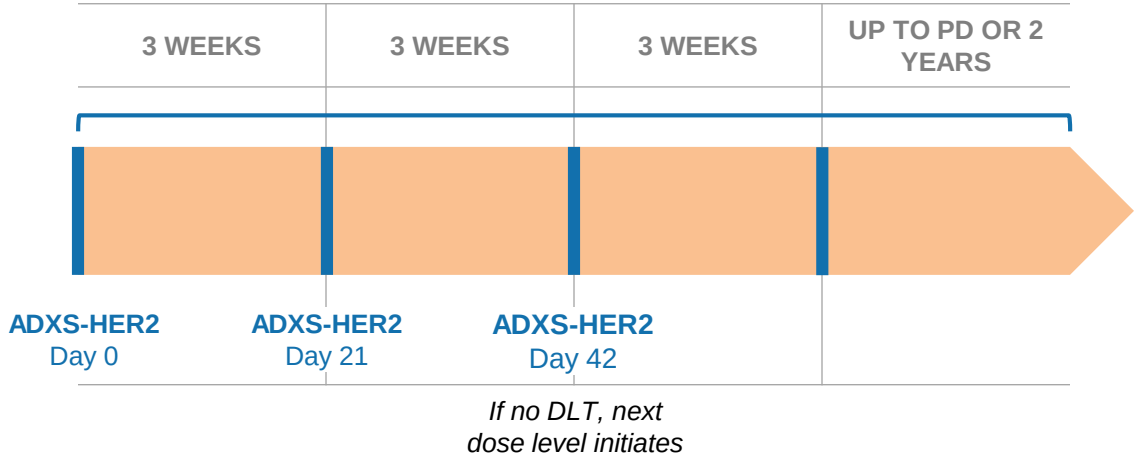
Primary Endpoint: Safety and RP2 Dose

ADXS-HER2 Monotherapy

Dose level 1: 1x10^{9} cfu q 3 wks
Dose level 2: 5x10^{9} cfu q 3 wks
Dose level 3: 1x10^{10} cfu q 3 wks

- N < 18 (Dose finding); N < 80 (Expansion phase) [Total N ~100]
- HER2-positive solid tumor (>1+ positivity in 1% of cells by IHC)
- Disease progressed or intolerant to standard therapy
- ECOG PS 0-1
- 3+3 Phase I Design



Next Steps

- Determine recommended Phase 2 dose
- Evaluate signals and set clinical development plan

PD, disease progression; RP2, Recommended phase 2

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Our Clinical Trials

ADXS-PSA

ADXS-PSA: Phase 1/2 Study
Monotherapy vs. Combo with Pembrolizumab

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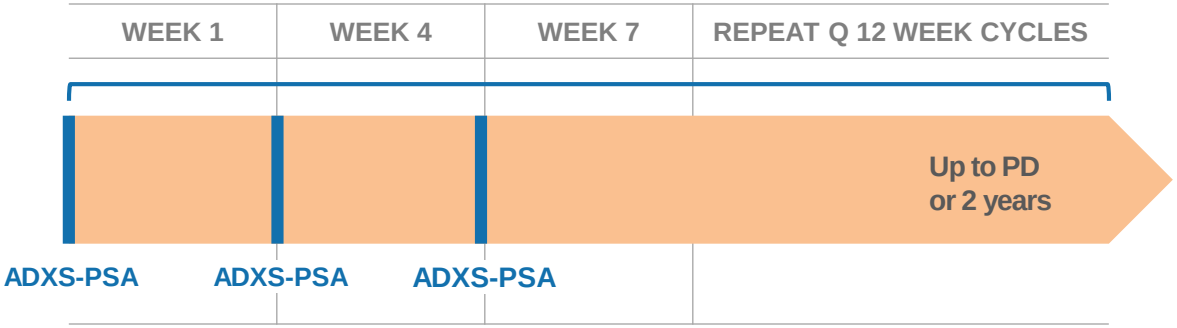
- N = 21 (Part A); N = 30 (Part B) [Total N = 51]
- Pretreated metastatic castration-resistant prostate cancer (CRPC)
- No more than 3 prior lines of systemic therapy (<1 chemotherapy)
- mTPI Design (Part A) □ RP2 Dose
- Part B ADXS-PSA Dose = Part A RP2 DL-1 + pembrolizumab

PART A

ADXS-PSA Monotherapy

Dose level 1: $1x10^9$ cfu d1 wk 1,4,7 q12 wks
Dose level 2: $5x10^9$ cfu d1 wk 1,4,7 q12 wks
Dose level 3: $1x10^{10}$ cfu d1 wk 1,4,7 q12 wks

N = 21

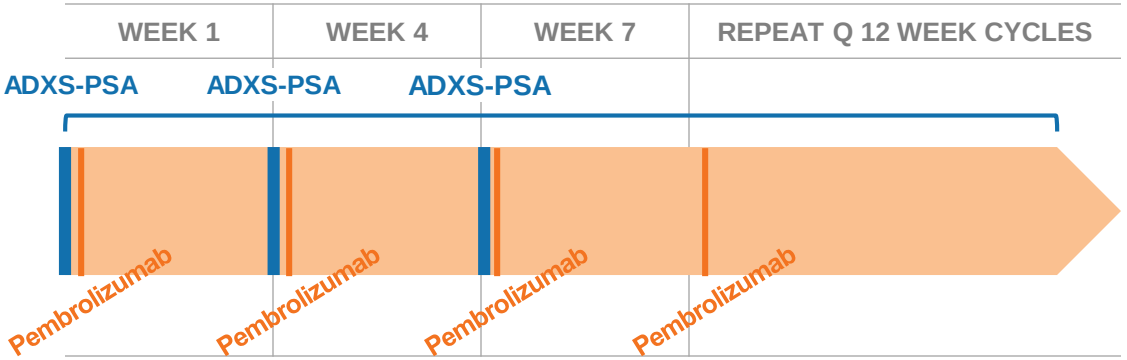


PART B

ADXS-PSA + Pembrolizumab

ADXS-PSA Part A Dose –DL1 d1 wk 1,4,7 q12 wks
Pembrolizumab 200 mg d1 q 3wks in 12 wk cycles

N = 30



ADXS-PSA: Phase 1/2 Study
Preclinical Data—Combination with PD-1

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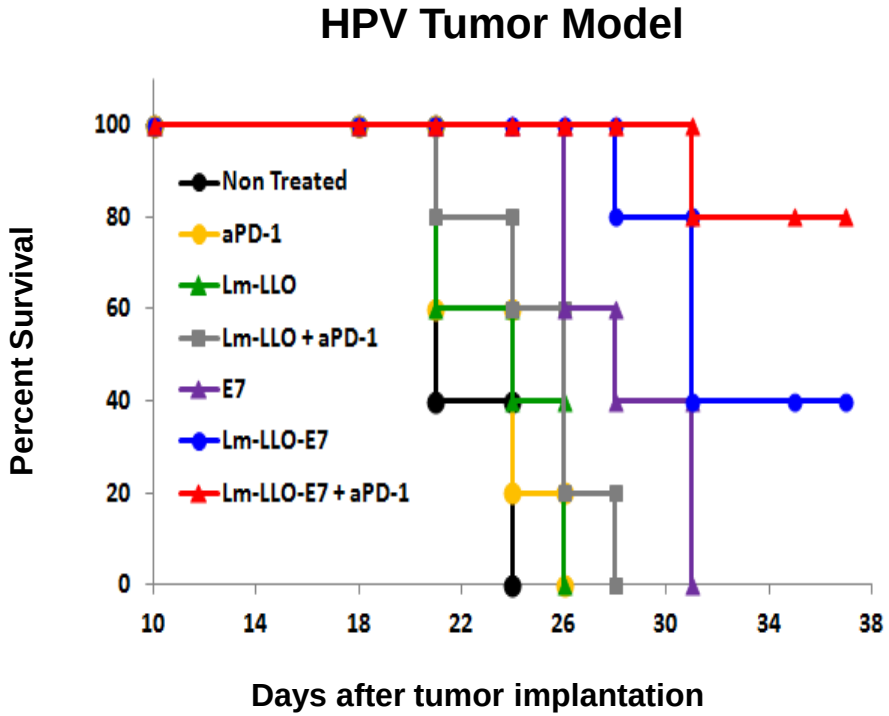

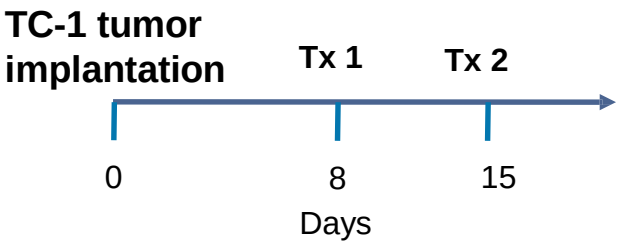

Treatments:	
Lm-LLO-E7:	5x10^6 cfu
CT-011 mAb:	50 µg

Data published in *Journal for ImmunoTherapy of Cancer* 2013, 1:15 doi:10.1186/2051-1426-1-15

Low dose *Lm*-LLO immunotherapy can be combined with a checkpoint inhibitor

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Our Preclinical Projects

ADXS-NEO

PERSONALIZED NEOEPITOPE-BASED IMMUNOTHERAPY

The Case for Neoepitopes

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Why does cancer develop "neoepitopes"?

- Tumors develop because of mutations in genes coding for key regulatory and functional proteins
- Expression of mutated proteins causes aberrant cellular functions that result in malignancy
- Malignant cells that can avoid normal mechanisms of immune surveillance are allowed to survive
- Normal peptides are weakly immunogenic – central tolerance deletes high avidity clones
- Mutated proteins in the cancer differ from those in normal cells, they can be targets for immunologic treatment
- High avidity T cell clones can be developed that recognize the mutated peptides – Not deleted by the thymus

The Case for Neoepitopes

ADVAXIS IMMUNOTHERAPIES™

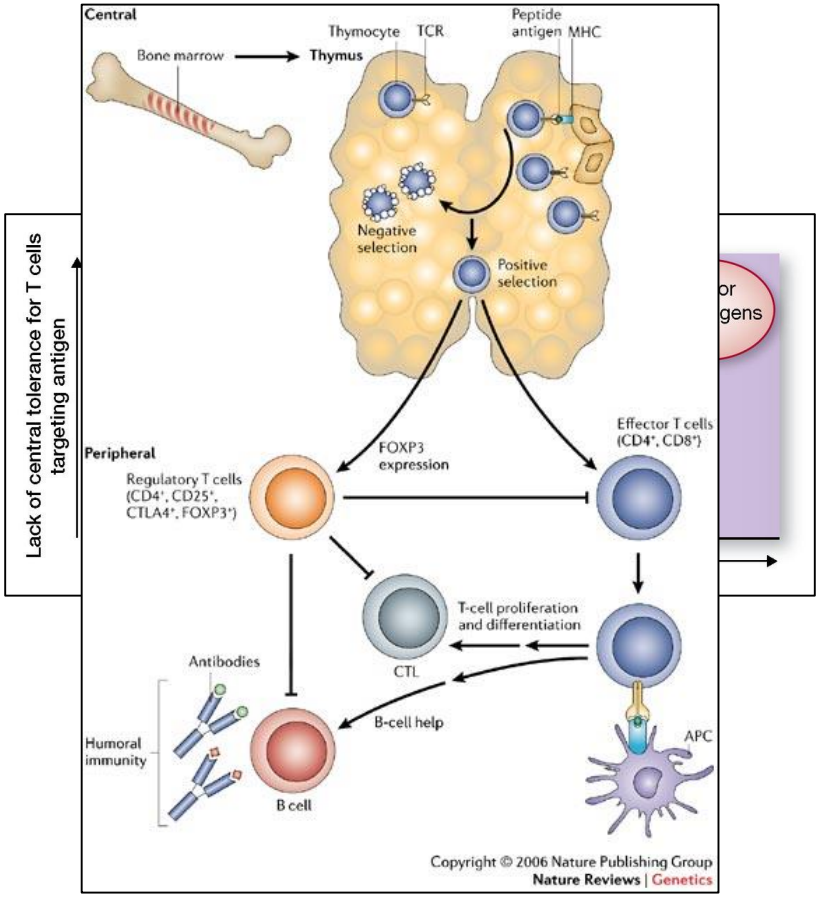
Why is this a good method to treat cancer?

- Immunotherapies work by "activating" the patient's immune system to target epitopes in cancer cells
  - High avidity cytotoxic T cells can be generated against neoepitopes
  - T cells targeting epitopes caused by mutations can be expanded therapeutically
  - Checkpoint inhibition appears to work by enabling pre-existing T cells responses against neoantigens to expand and become tumoricidal
  - Immunizing patients against their own neo-antigens with an attenuated live vector will generate or enhance T cell responses against neoepitopes
- Because the T cell responses are only against the mutated neoepitopes, and there is no systemic blockade of tolerance, there should be no off-target toxicity

Neoepitopes in Cancer Treatment

ADVAXIS IMMUNOTHERAPIES™

- Effective Immunotherapies work by "enabling" the patient's immune system to amplify T cells that target neoepitopes in cancer cells
  - High avidity cytotoxic T cells can be generated against neoepitopes
  - T cells targeting epitopes caused by mutations can be expanded therapeutically
  - T cells against non-synonymous tumor-associated antigens are not deleted by central tolerance in the thymus
  - Checkpoint inhibition appears to work by enabling pre-existing T cell responses against neoepitopes to expand and become tumoricidal
  - Immunizing patients against their own neo-antigens with an attenuated live vector will generate or enhance T cell responses against neoepitopes

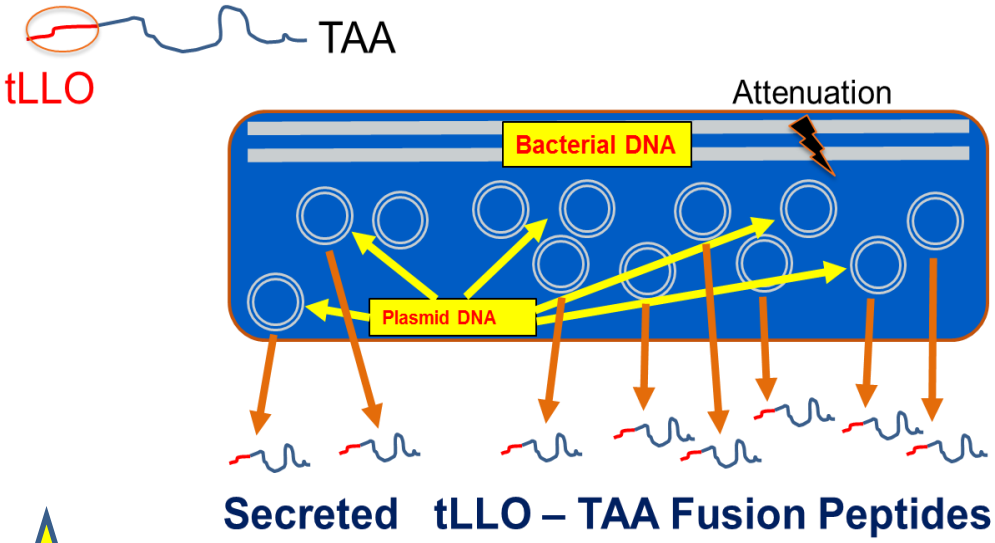


Because the T cell responses are only directed against mutated neoepitopes there should be no off-target toxicity

Gregersen PK et al. Genetics of autoimmune diseases—disorders of immune homeostasis. Nature Reviews Genetics 2006; 7: 917-928.
Nir Hacohen et al. Cancer Immunol Res 2013;1:11-15.

Targeting Neoepitopes with *Lm* Technology™
Advantages for Personalized Immunotherapy

ADVAXIS IMMUNOTHERAPIES™



Advaxis's *Lm* Technology™ may have advantages for targeting neoepitopes

1) Bandwidth—5 constructs can present >250 tumor neoepitopes to T-cells
2) CTLs infiltrating the tumor are more effective at killing tumor cells because of decreased Tregs and MDSCs
3) *Lm* is synergistic with checkpoint inhibitors

- tLLO—TAA fusion protein is a synthetic peptide presenting multiple neoepitopes secreted into the cytoplasm of the APC
- 80-100 plasmid copies per bacteria
- Payload for up to >50 neoepitopes per construct–up to 2k+ amino acids
- Multiple constructs can be administered for larger numbers of neoepitopes
- Adjuvants built in (TLRs, PAMP, STING, DAMP, NOD1, NOD2, CpG)
- Treatments can be given repeatedly without neutralizing antibodies
- Generate strong innate and adaptive T cell response, even to lower avidity epitopes
- Decreases Tregs and MDSCs in the tumor microenvironment

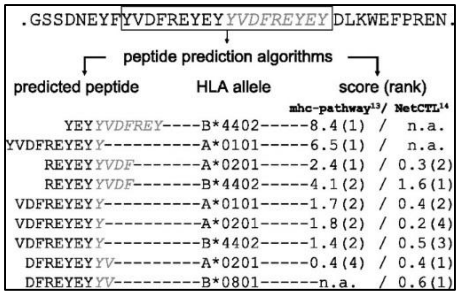
Project APOLLO Schema
How would it work?

ADVAXIS IMMUNOTHERAPIES™

Academic or Commercial Massively Parallel Sequencing

Sequencing to identify non-synonomyous mutations

Identify neoepitopes

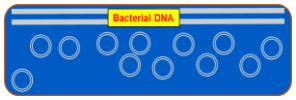


Advaxis Immunotherapies

Advaxis designs vector based on neoepitopes

DNA synthesis – molecular cloning into plasmids

Transfection into personalized vector, QA/QC – OK



Ship to patient's institution

Patient's Hospital or Treating Institution

Treat patient with personalized immunotherapy vector based on his/her neoepitopes

Multiple cycles of treatment and combination with RT, PD-1, co-stims possible

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Our Collaborations & Milestones

LM-LLO IMMUNOTHERAPY: A VERSATILE, INNOVATIVE PLATFORM

Advantages of *Lm* Technology™

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Efficacy Attributes

- High expression and secretion of tLLO/ fusion protein (tumor associated antigen [TAA])
- Large payload size for delivering tumor antigens—up to 2,000 amino acids per construct
- Efficacy as monotherapy (includes CR, PR and increased survival)
- No need for preconditioning agents to enhance therapeutic effect
- Potential for synergy with checkpoint inhibitors, including inhibitors of PD-1, PD-L1, IDO, and CTLA4, as well as co-stimulatory molecules, such as OX40 and GITR
- Impacts tumor microenvironment (TME) by disabling Tregs & MDSCs

Safety Attributes

- Strong attenuation of axalimogene filolisbac with established safety
- Dosed up to 5x10^9 in humans with potential to go higher
- Predominantly Grade 1 and 2 AEs in 260+ patients treated to date
- No cases of lymphopenia
- ~1% Grade 3 AEs

IP Attributes

- Exclusively in-licensed original IP from UPENN where *Lm* platform was invented
- Any other *Lm* technologies must avoid infringing on this IP
- 80+ issued and 80+ pending patents worldwide for platform, product candidates, methods, manufacture, process and formulation

Novel Combination Therapy Collaborations

ADVAXIS IMMUNOTHERAPIES™

Entered into an R&D Collaboration with

ADVAXIS IMMUNOTHERAPIES™

Phase 1/2 study evaluating the safety and efficacy of axalimogene filolisbac in combination with durvalumab (MEDI4736) (anti-PD-1)	Phase 1/2 study evaluating the safety and efficacy of ADXS-PSA in combination with KEYTRUDA® (pembrolizumab) (anti-PD-1)	Phase 2 study evaluating the safety and efficacy of axalimogene filolisbac as a monotherapy and in combination with INCB24360 (epacadostat) (IDO1)	Evaluation of *Lm* Technology™ immunotherapies plus antibodies targeting GITR, OX40, LAG-3 and TIM-3
July 2014	**August 2014**	**February 2015**	**May 2015**

KEYTRUDA is a registered trademark of Merck & Co., Inc.

Strategic, Value-Building Opportunities

ADVAXIS IMMUNOTHERAPIES™

Entered into a licensing agreement with

ADVAXIS IMMUNOTHERAPIES™

- ADXS-HER2 (animal health)
- Canine osteosarcoma + 3 additional products

- Axalimogene filolisbac
- HPV-associated cervical cancer

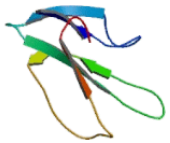
- Axalimogene filolisbac
- HPV-associated cancers

Platform Versatility

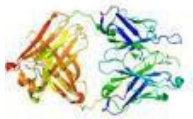
ADVAXIS IMMUNOTHERAPIES™

Single Antigen Constructs

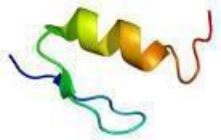
***Lm*-LLO +**



PSCA
Prostate Cancer Pre-Clinical

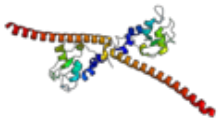


CA9
Renal and Others Pre-Clinical



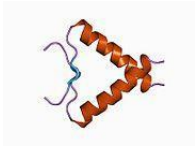
WT-1
Several (Pan) Pre-Clinical

CEA
Ovarian Pre-Clinical



Survivin
Lymphoma Pre-Vet

FAP
Breast CA Pre-Clinical



P53
Breast CA Pre-Clinical

SCCE-KLK7
Ovarian, others Pre-Clinical

ISG 15
Bladder Pre-Clinical

HMW-MAA
Lymphoma Pre-Clinical

IL13RA2
Solid Tumors Pre-Clinical

VEGF-r2
Solid Tumors Pre-Clinical

Endoglin (CD-105)
Breast CA Pre-Clinical

Advaxis has developed several product constructs leveraging the company's platform technology

Financial Summary

ADVAXIS IMMUNOTHERAPIES™

Cash Summary

- Cash as of April 30, 2015
  - ✓ $45.9M
- Cash as of July 31, 2015
  - ✓ $97.1M
- Cash receivables since Jul‘15
  - ✓ $25.0M (gross) – Registered Direct (August)
- Capital raised since October ’13
  - ✓ ~$165M
- No Debt

Equity Summary

- Basic Shares Outstanding (as of 9/9/15)
  - ✓ 33.4M
- Warrants and Options
  - ✓ 3.3M and 1.9M (as of 7/31/2015)
- Pro-forma Fully Diluted
  - ✓ 38.6M

Leadership Accountability

ADVAXIS IMMUNOTHERAPIES™

	Out of Pocket Funds (1)		Company Incentive Awards (1)	
	Gross $	net shares	vested	unvested
Daniel J. O'Connor	$662,903	153,316	115,760	83,333
David J. Mauro	$45,550	6,196	32,884	151,333
Gregory T. Mayes	$180,737	27,036	36,845	75,000
Robert G. Petit	$128,648	28,427	47,132	56,652
Sara M. Bonstein	$96,710	26,406	34,530	33,333

(1) Above figures are as of September 1, 2015

Represents RSU awards & share purchases only; Does not include option and/or warrants.

Management voluntarily purchases restricted stock directly from the Company every two weeks at market price

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305 College Road East, Princeton, NJ
www.advaxis.com
ir@Advaxis.com