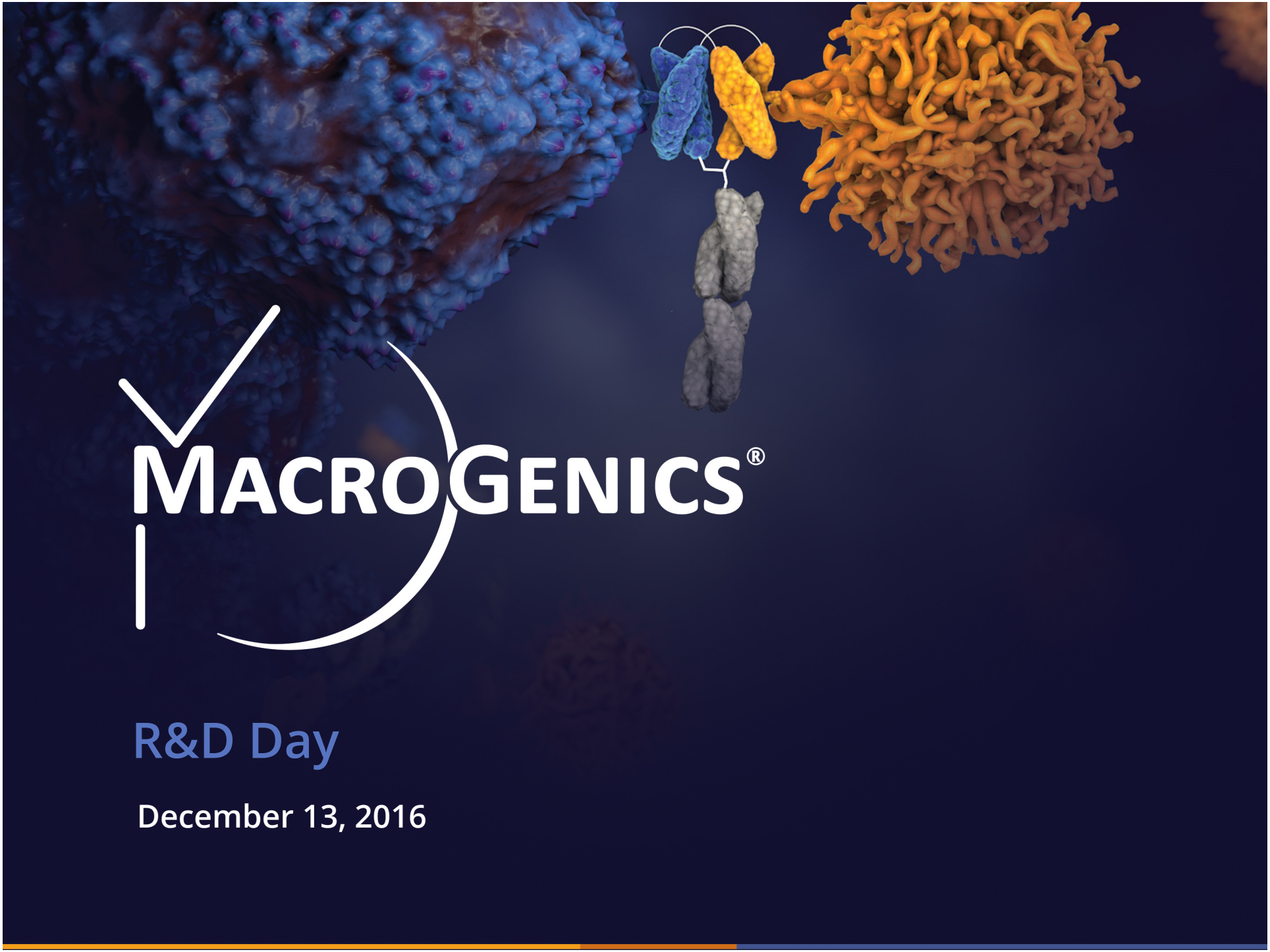

MacroGenics®

R&D Day

December 13, 2016

Legal Notices

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Any statements in these materials about future expectations, plans and prospects for MacroGenics (“Company), including statements about the Company's strategy, future operations, clinical development of the Company's therapeutic candidates, milestone or opt-in payments from the Company's collaborators, the Company's anticipated milestones and future expectations and plans and prospects for the Company and other statements containing the words "subject to", "believe", "anticipate", "plan", "expect", "intend", "estimate", "project", "may", "will", "should", "would", "could", "can", the negatives thereof, variations thereon and similar expressions, or by discussions of strategy constitute forward-looking statements within the meaning of Section 27A of the Securities Act of 1933 and Section 21E of the Securities Exchange Act of 1934. Actual results may differ materially from those indicated by such forward-looking statements as a result of various important factors, including: the uncertainties inherent in the initiation and enrollment of future clinical trials, expectations of expanding ongoing clinical trials, availability and timing of data from ongoing clinical trials, expectations for regulatory approvals, other matters that could affect the availability or commercial potential of the Company's product candidates and other risks described in the Company's filings with the Securities and Exchange Commission. In addition, the forward-looking statements included in this press release represent the Company's views only as of the date hereof. The Company anticipates that subsequent events and developments will cause the Company's views to change. However, while the Company may elect to update these forward-looking statements at some point in the future, the Company specifically disclaims any obligation to do so, except as may be required by law. These forward-looking statements should not be relied upon as representing the Company's views as of any date subsequent to the date hereof.

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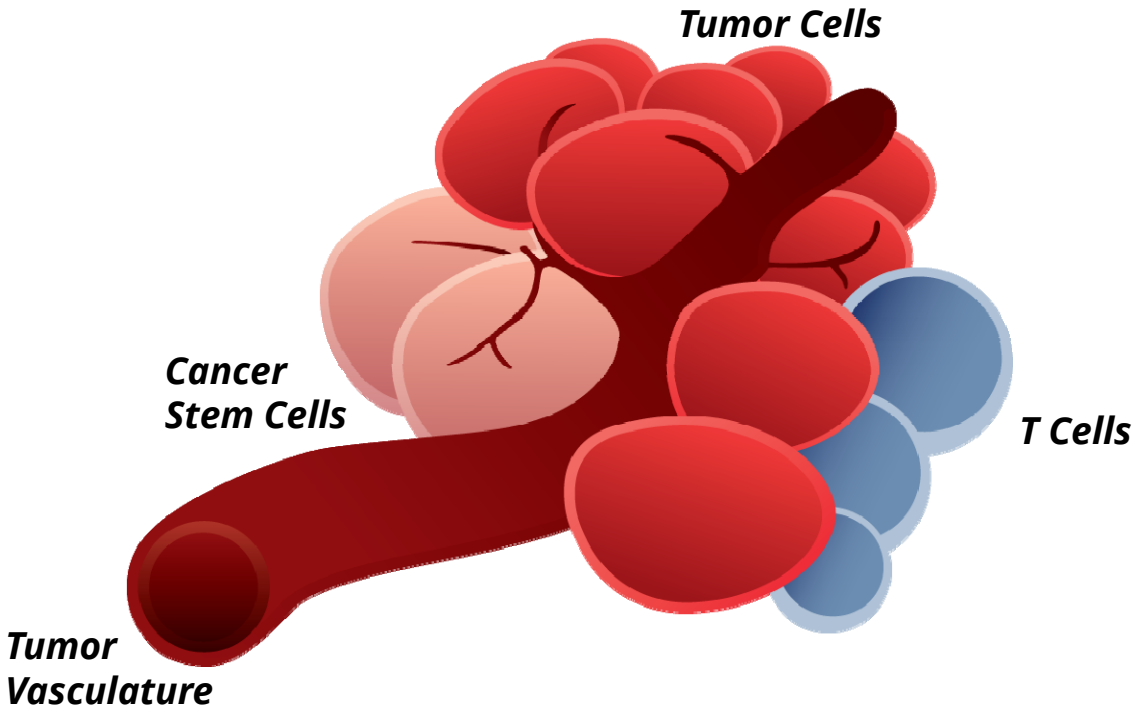
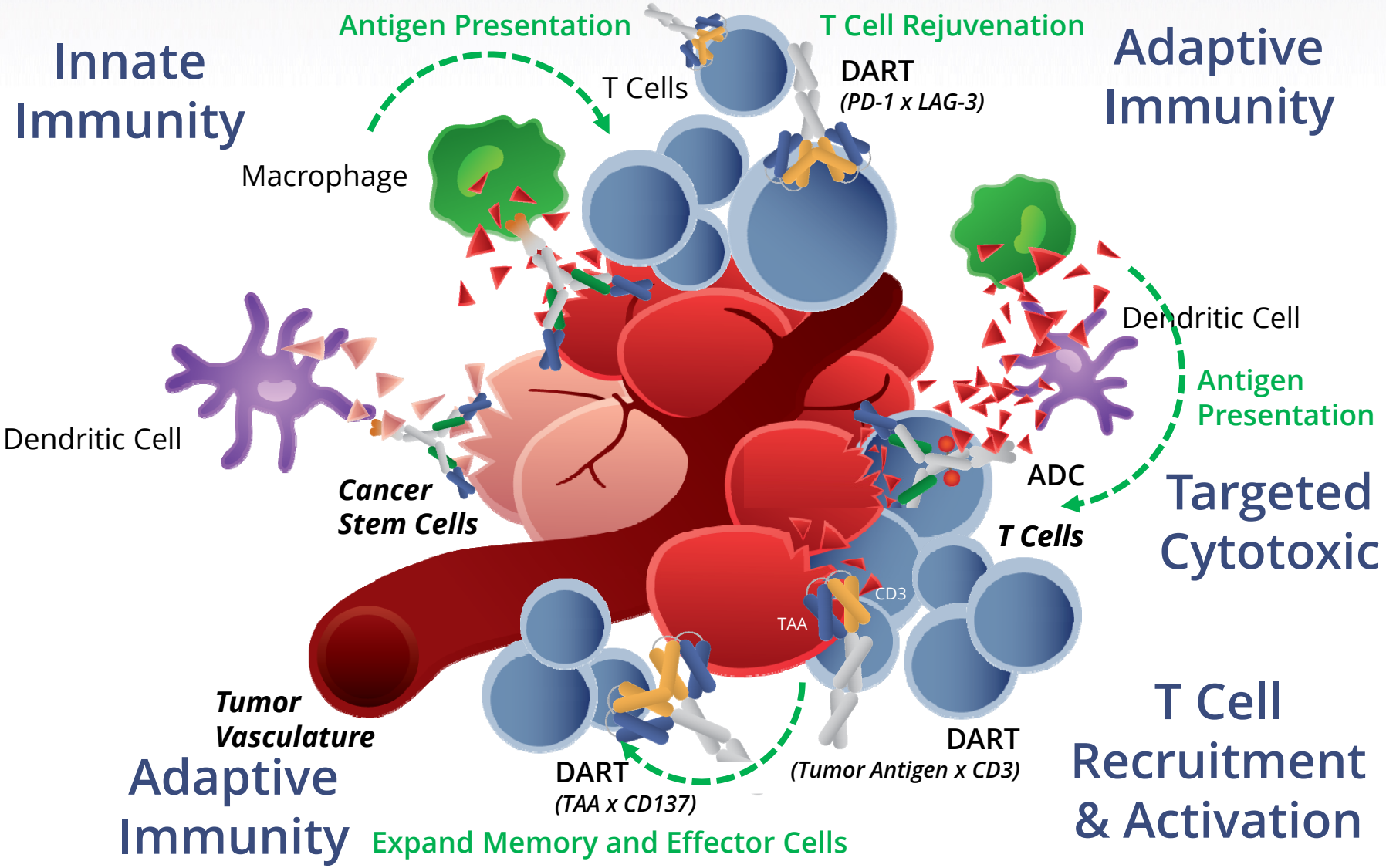
Committed to Breakthrough Biologics

Focus 	• Unmet medical needs in cancer and autoimmune disease • Integrated, highly productive R&D organization
Products 	• Nine clinical candidates, including seven in immuno-oncology • ≥1 New IND projected annually (6 INDs in last 3 years)
Platforms 	• DART® and TRIDENT™– versatile multi-specific mAb technologies • Fc Optimization – more potent therapeutic mAbs • Cancer Stem Cells – novel target ID and drug screening
Funding 	• Well capitalized to advance pipeline ($314M cash @ 9/30/16) • Alliances with BI, Janssen, Pfizer, Servier and Takeda
Employees 	• 314 Employees as of 11/30/16 (Rockville, MD and SSF, CA) • Leadership team with extensive track-record

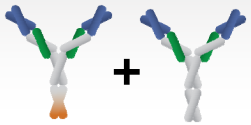
Integrated Immuno-Oncology Portfolio



Integrated Immuno-Oncology Portfolio

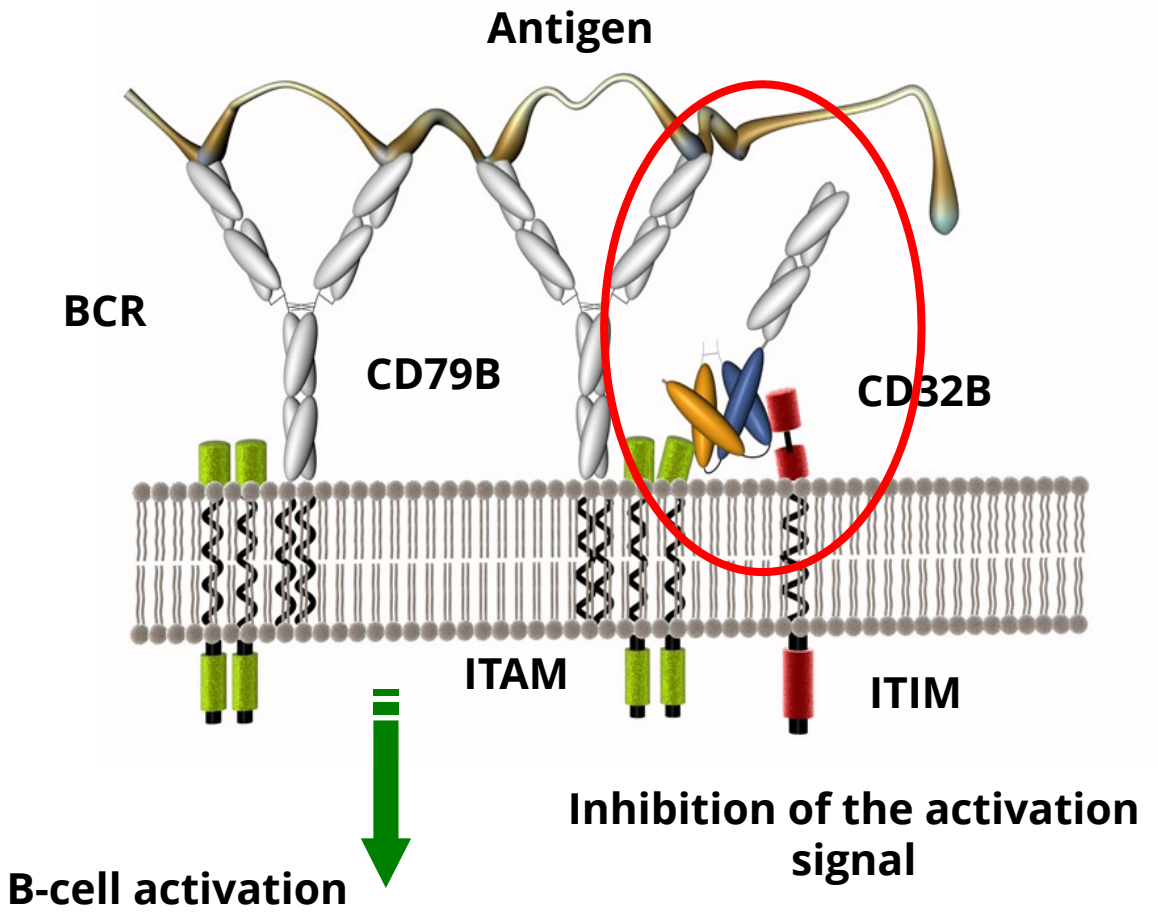


Building Comprehensive Immunotherapeutic Franchises

Platform	HER2 *Validated*	B7-H3 *Novel*	PD-1 *Emerging Backbone*
mAb/ Fc-Optimized 	margetuximab	enoblituzumab	MGA012 (anti-PD-1)
DART/TRIDENT 	CD137 x HER2	MGD009	• PD-1 x LAG-3 • PD-1 x CTLA-4 • PD-1 + MGD006 • PD-1 + MGD007 • PD-1 + MGD009
Checkpoint Combination 	margetuximab + anti-PD-1	enoblituzumab + anti-PD-1 enoblituzumab + anti-CTLA-4	enoblituzumab + anti-PD-1
ADC 	—	MGC018 (B7-H3 ADC)	anti-PD-1 + MGC018 (B7-H3 ADC)

Leveraging Checkpoint Biology for Autoimmunity

MGD010 (CD32B x CD79B DART) establishes clinical proof-of-principle



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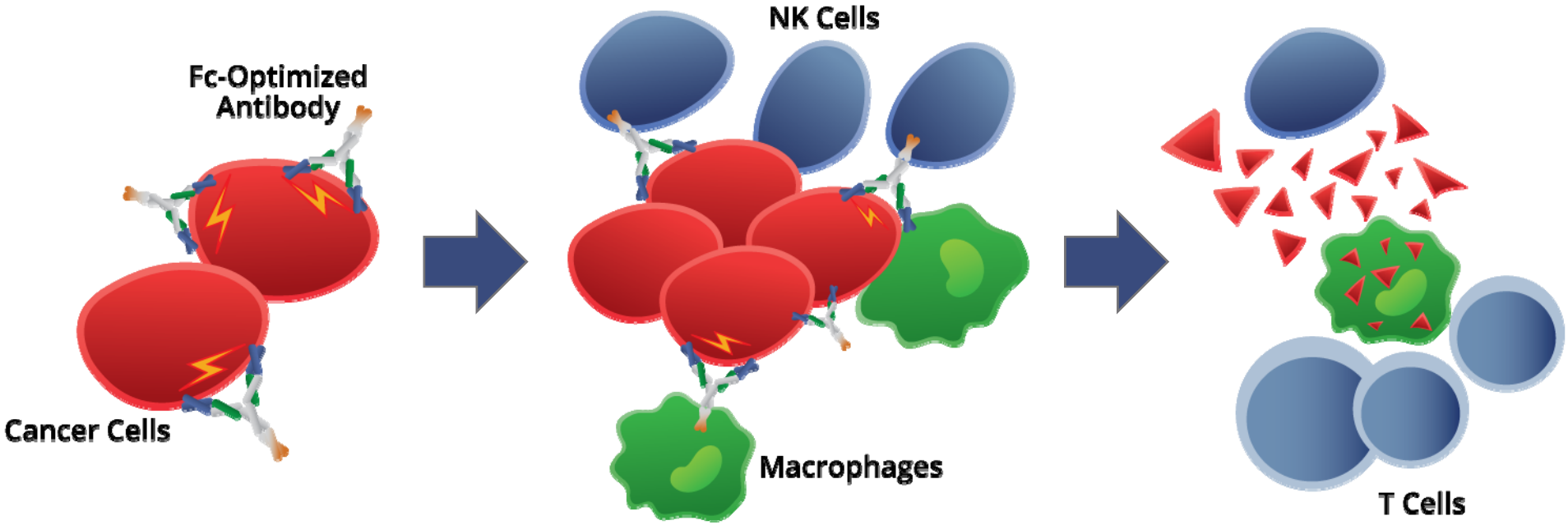
Margetuximab: Potential Best-in-Class Anti-HER2 mAb

Leveraging immune modulation through Fc optimization

Candidate	• Fc-optimized anti-HER2 mAb 
Function/ MoA	• Inhibits HER2 signaling (consistent with trastuzumab) • Fc Optimization potentially enhances ADCC – Increases binding to activating FcγR (CD16A) – Decreases binding to inhibitory FcγR (CD32B)
Lead Indications	• Ph. 3 SOPHIA study (HER2+ metastatic breast cancer) • Ph. 1b/2 combo study with pembrolizumab (HER2+ gastric cancer)
Partner	• MacroGenics has global rights (ex-South Korea)

Enhanced Antitumor Activity through Effector Cell Engagement

Margetuximab: same signaling properties as trastuzumab plus enhanced ADCC



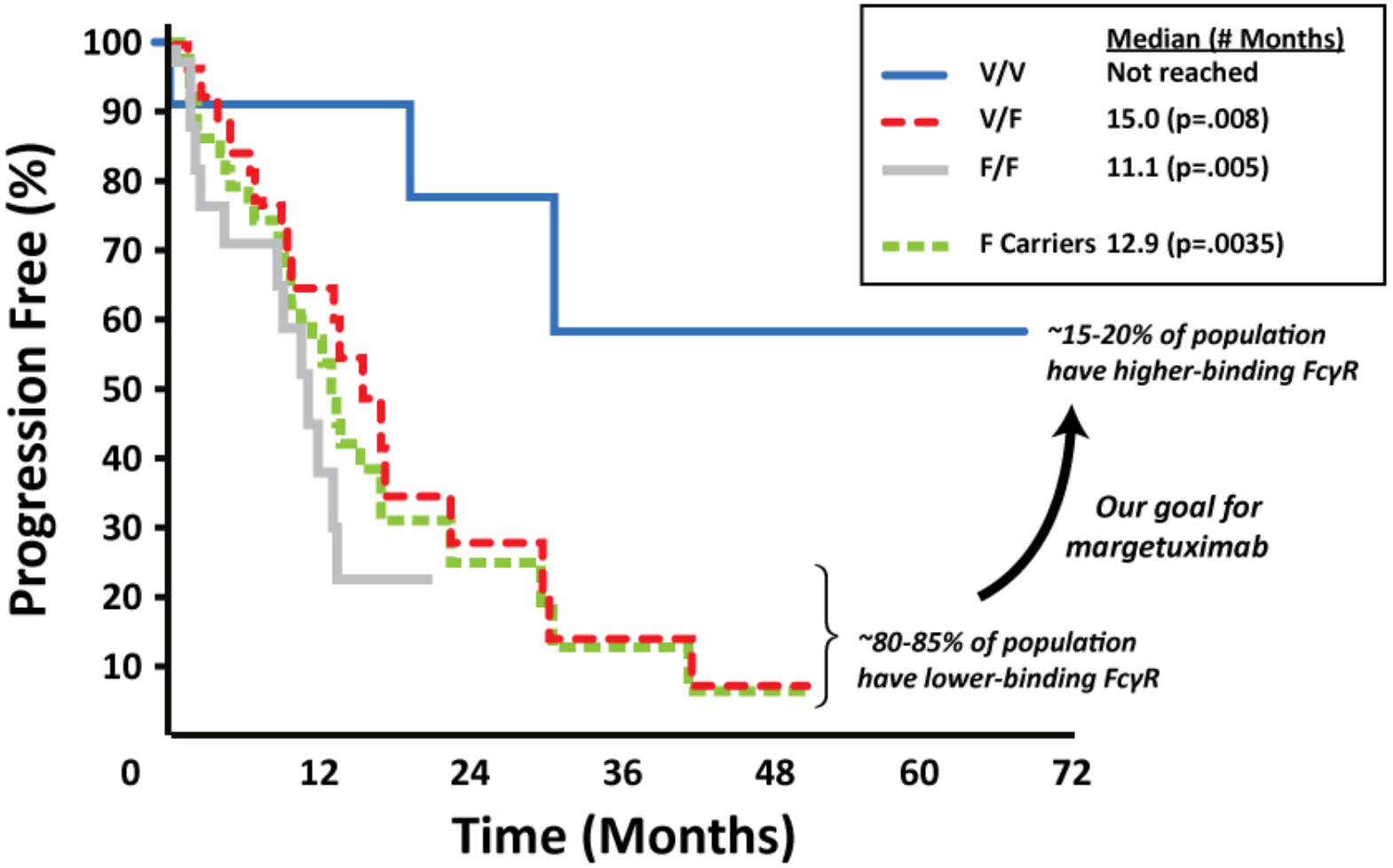
Anti-proliferative Activity (Same as trastuzumab)

Superior Engagement of Effector Cells and ADCC (Enhanced CD16A Binding, Reduced CD32B Binding)

Tumor Antigen Capture & Presentation to T Cells

Role of FcγR Polymorphism in Response to Trastuzumab

*Improved outcomes in patients homozygous for higher-affinity CD16A V-allele**

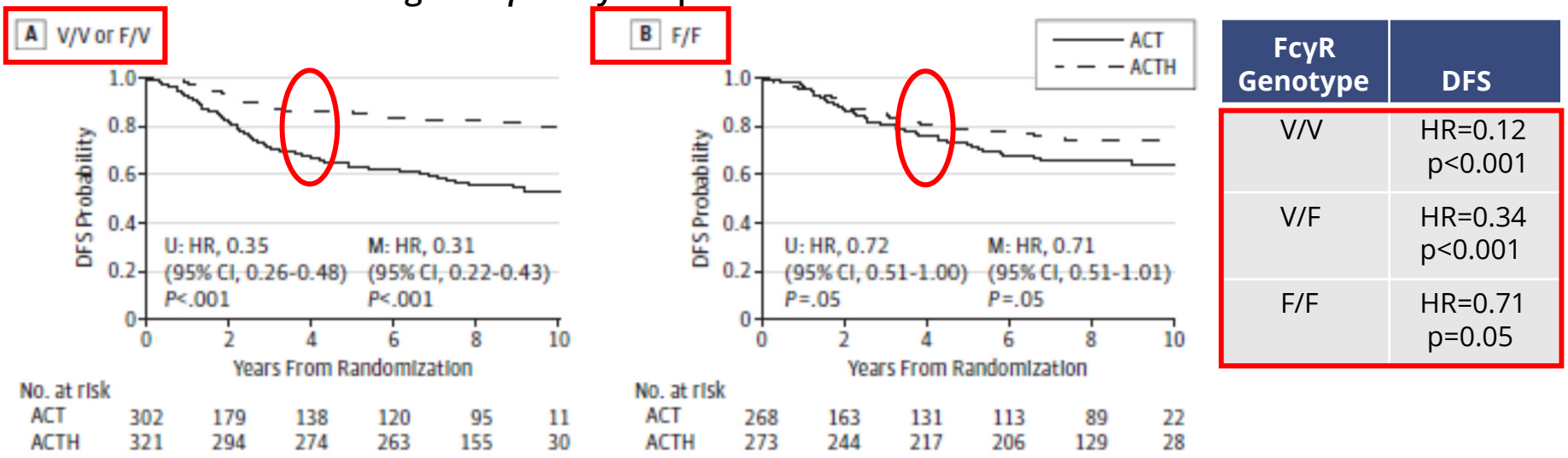


* Musolino, *et al.*, JCO 2008. Kaplan-Meier estimates of PFS to trastuzumab-based therapy by IgG fragment C receptor (FcγR) polymorphisms. PFS curves plotted by FcγRIIIa 158 valine(V)/phenylalanine(F) genotype. F carriers represent patients with either 158 V/F or 158 F/F genotype.

FcγR Status Can Influence Benefit from Trastuzumab Therapy

Lower affinity F-allele associated with reduced disease-free survival (DFS)

Treatment Effect According to FcγR Polymorphism



U: Univariable; M: Multivariable

FcγR Genotype	DFS
V/V	HR=0.12 p<0.001
V/F	HR=0.34 p<0.001
F/F	HR=0.71 p=0.05

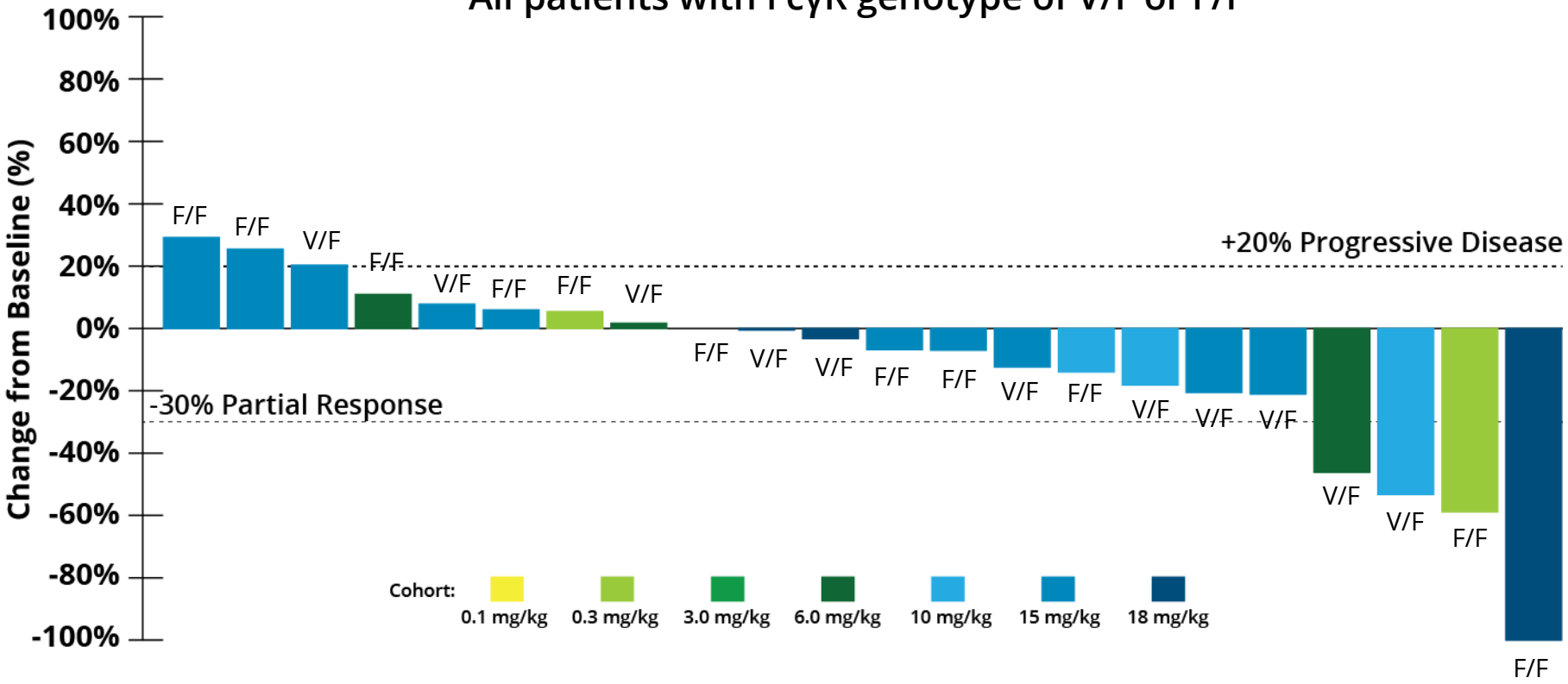
- NSABP B-31 study (n = 2043) compared standard chemo (AC-T) *vs.* chemo + trastuzumab (AC-TH) in adjuvant setting for 3+ HER2+ breast cancer[1]
  - Adding trastuzumab improved outcome [HR = 0.46, 95% CI 0.37-0.57, p < 0.001][2]
- Pre-treatment FcγR polymorphism analysis performed on subset of 1,251 samples[2]

[1] Romond EH et al. N Engl J Med 2005; 353:1673-1684
[2] Gavin PG et al. *JAMA Oncol*. Published online November 3, 2016

Margetuximab's Promising Activity Profile

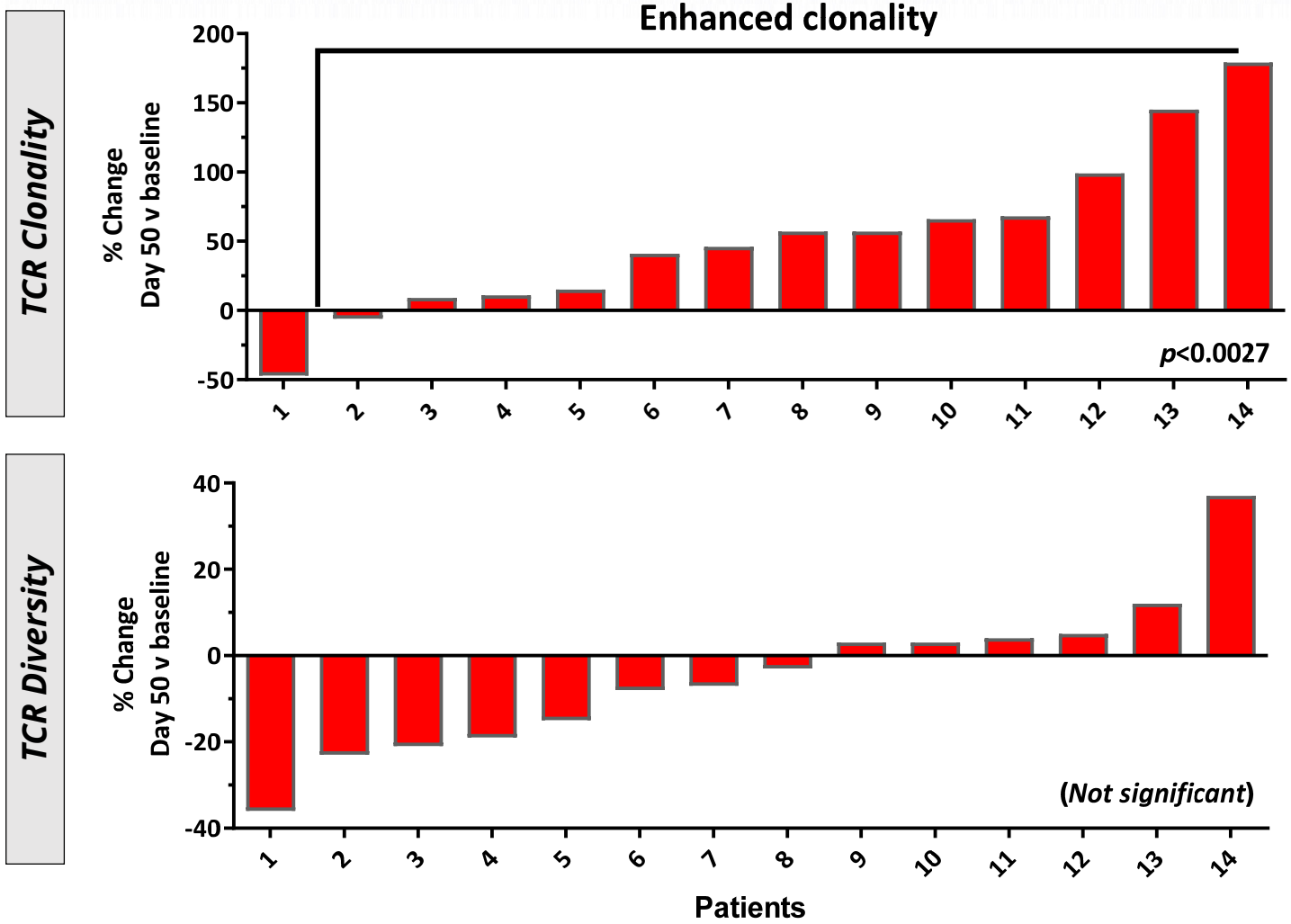
Phase 1 results for metastatic breast cancer patients

Median # prior lines of therapy = 4
All received anti-HER2 agent in earlier line(s) of therapy
All patients with FcγR genotype of V/F or F/F



Margetuximab Treatment Enhances T-cell Clonality

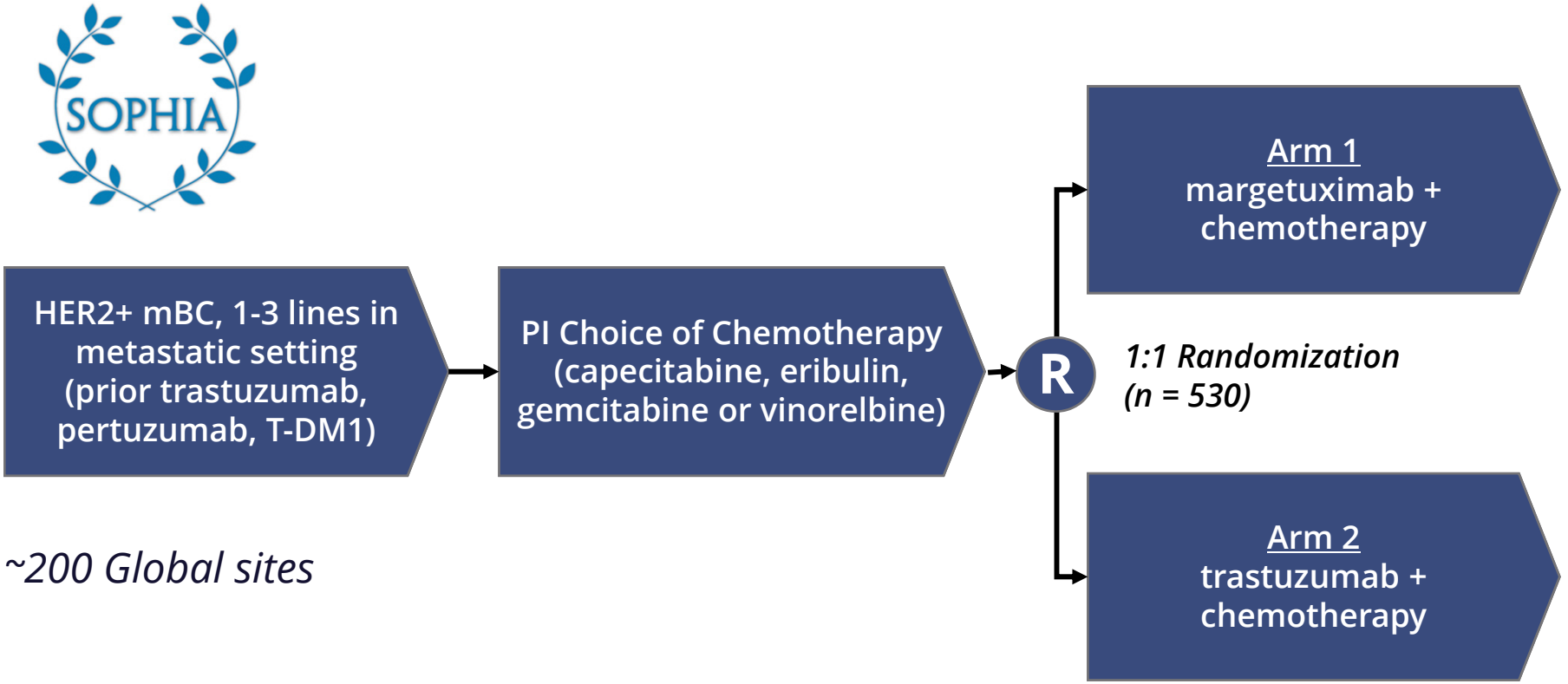
TCR spectrotype analysis indicates an effect on adaptive immunity



Analysis performed by Adaptive Biotechnologies on PBMC from Ph. 1 pts

SOPHIA Study to Establish Superiority to Trastuzumab

Study anticipated to complete enrollment in 2018



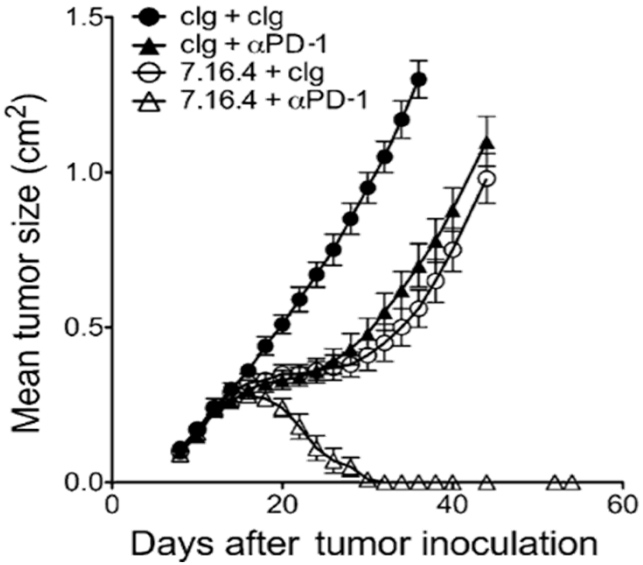
Sequential primary endpoints: Progression-Free Survival & Overall Survival:

PFS (N=257, HR=0.67, α=0.05, power=90%)

OS (N=358, HR=0.75, α=0.05, power=80%)

Why Combine HER2 and Anti-PD-1 for Gastric Cancer?

- HER2 is validated target in gastric cancer; trastuzumab is component of 1L SoC
- No HER2 SoC after 1L; existing therapy (chemo, ramucirumab) is sub-optimal
- Negative GATSBY trial with T-DM1 in 2L gastric cancer may highlight importance of immune-mediated mechanisms in clinical activity of anti-HER2 agents
- Checkpoint inhibitors are clinically active in patients with gastric cancer (ORR, OS)
- Clear non-clinical rationale for combining anti-HER2 directed therapy and I-O agents
- Synergistic antitumor activity using combination of anti-HER2 and anti-PD-1 antibodies in HER2+ murine mammary adenocarcinoma model *(Stagg, et al. PNAS, 2011)*



Phase 1b/2 Study in Adv./Metastatic Gastric & GEJ Cancers

Potential for chemotherapy-free regimen

MERCK

Dose Escalation √
(n=3-6 per margetuximab dose)

margetuximab 10 – 15 mg/kg q3w
+
pembrolizumab 200 mg q3w

Dose Expansion

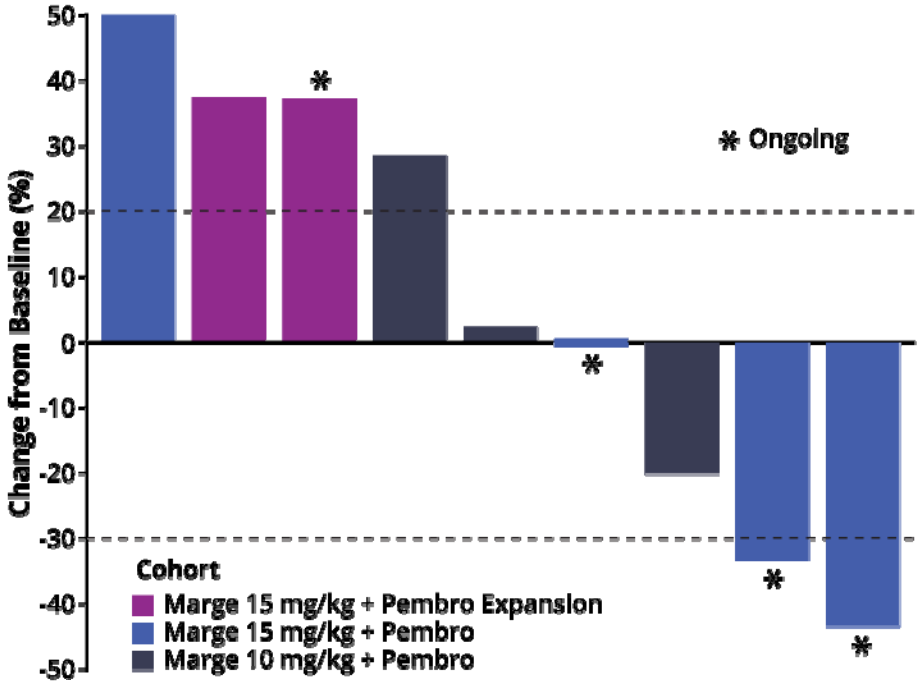
margetuximab MTA/MTD
+
pembrolizumab

- Treatment
  - Pembrolizumab: Day 1 of every cycle
  - Exploring two dose levels for margetuximab: Day 1 of every cycle
- Inclusion/exclusion criteria
  - IHC HER2 2+ / 3+ or FISH-amplified with prior trastuzumab progression
  - Received ≥ 1 prior line of chemotherapy treatment
  - No prior immunotherapy
- Endpoints
  - Primary: safety, tolerability and efficacy (as evaluated by ORR) of combo
  - Secondary: PFS, PFS-6, OS-6 / OS, Immunogenicity

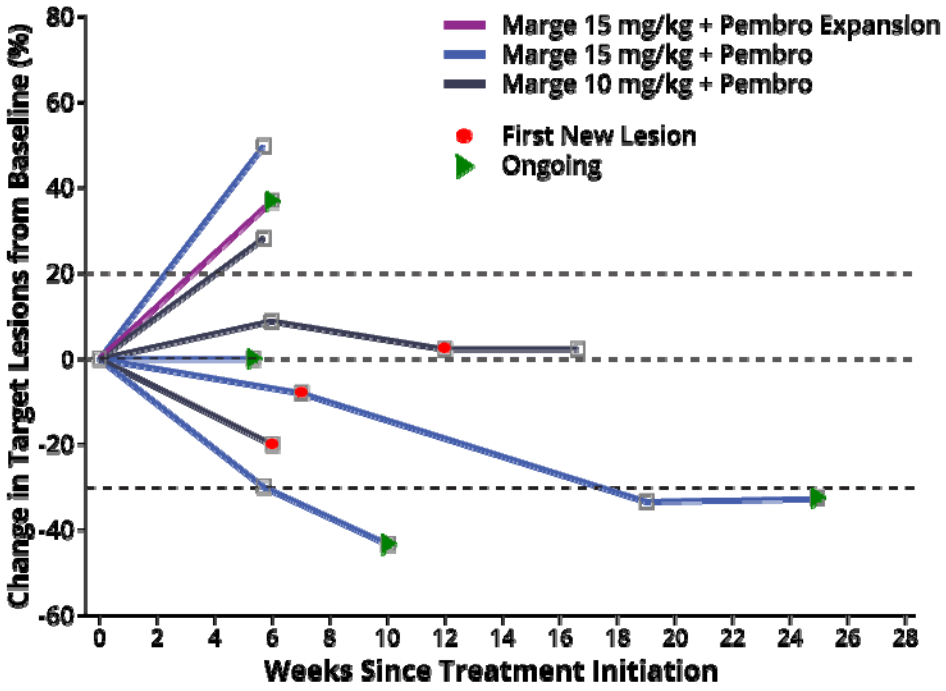
Margetuximab + Pembrolizumab Initial Clinical Activity

Phase 1b/2 study in advanced/metastatic gastric & GEJ cancers

Best Percent Change from Baseline of Target Lesions in Response-evaluable Population



Percent Change in Target Lesions Over Time in Response-evaluable Population

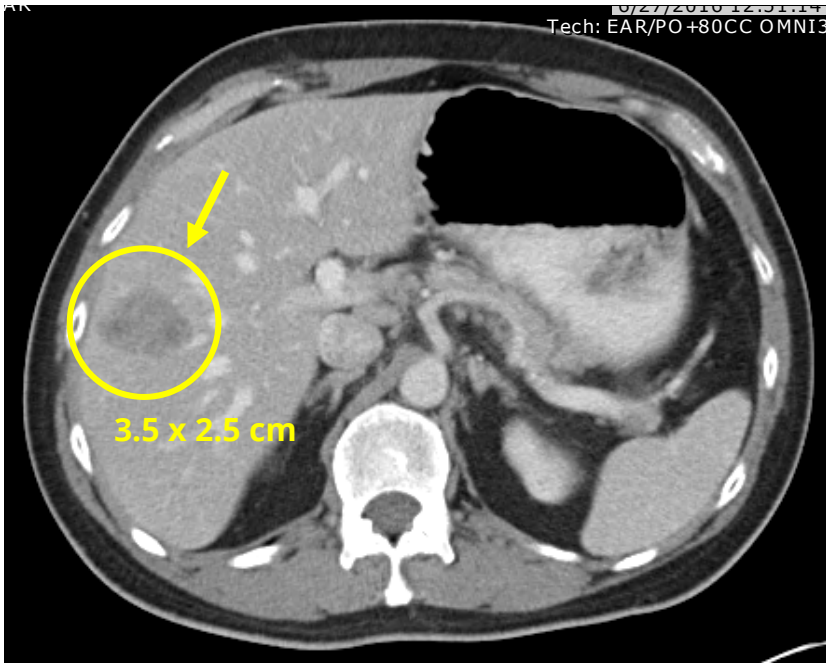


Note: December 1, 2016 data cutoff

Confirmed PR in Gastric Cancer: 50% Reduction

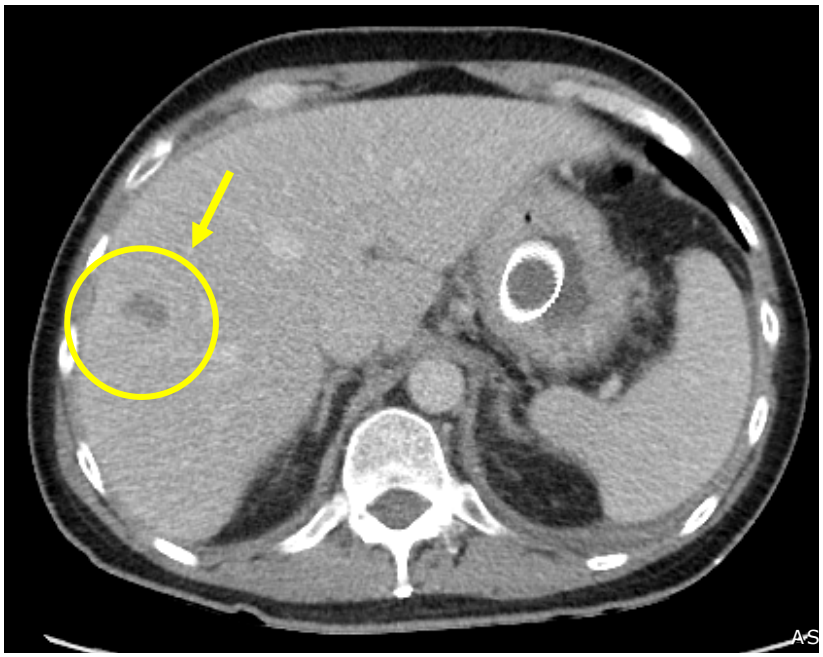
M+P combo case study #1: 50 y/o male w/metastatic HER2+ gastric cancer

Baseline



- Patient had metastatic GEJ and gastric adeno-carcinoma, with gastric cardia and liver lesions
- Progressed after prolonged 1L treatment with FOLFOX/trastuzumab followed by maintenance capecitabine/trastuzumab

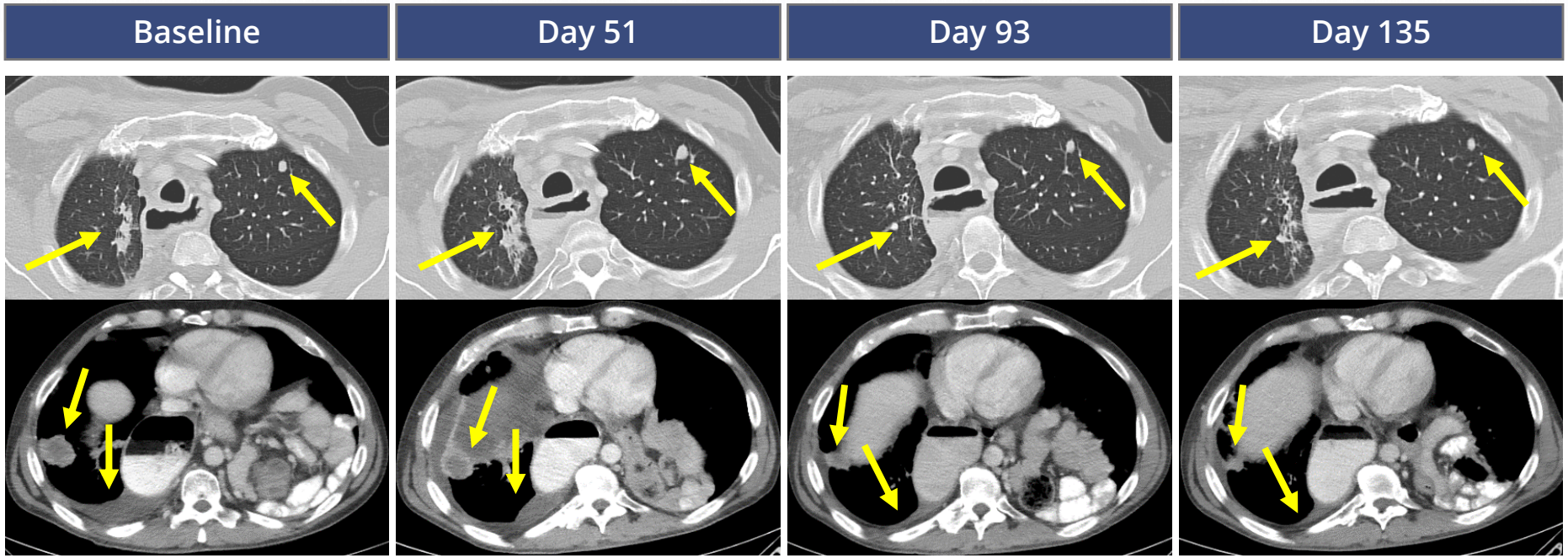
Day 72



- Confirmed PR with margetuximab + pembrolizumab combination (**50% reduction**)
- Later in course, experienced clinical deterioration, not felt to be candidate for treatment beyond progression, withdrawn from study

Confirmed irPR in Gastric Cancer: 30% Reduction

M+P combo case study #2: 50 y/o male w/metastatic HER2+ gastric cancer



- Patient had metastatic HER2+ GEJ/gastric adenocarcinoma with lesions in liver, lungs and supraclavicular LNs
- Progressed following 3 prior lines of therapy:
  - Carboplatin, taxol and radiotherapy
  - Cisplatin, capecitabine and trastuzumab → maintenance trastuzumab
  - 5-FU and trastuzumab
- Confirmed irPR (**30% reduction**)
- Currently on Cycle 9 (well tolerated)

HER2 Franchise Summary

- Fc optimization designed to enable antitumor activity across FcγR genotypes
  - Enhanced antitumor activity compared to trastuzumab in ~80% of patients who are low affinity carriers (either V/F or F/F)
  - Enhanced ability to induce cell killing and secondary engagement of T-cell immunity
- If SOPHIA is successful, positioned to move into earlier lines of therapy
- Combo with anti-PD-1 could establish "chemo-free" treatment paradigm for advanced/metastatic gastric & GEJ cancers
- Follow-on programs, including CD137 x HER2 DART, to deliver tumor-specific immune modulation
- Upcoming milestones:
  - Complete gastric study enrollment in 2017
  - Complete SOPHIA enrollment in 2018

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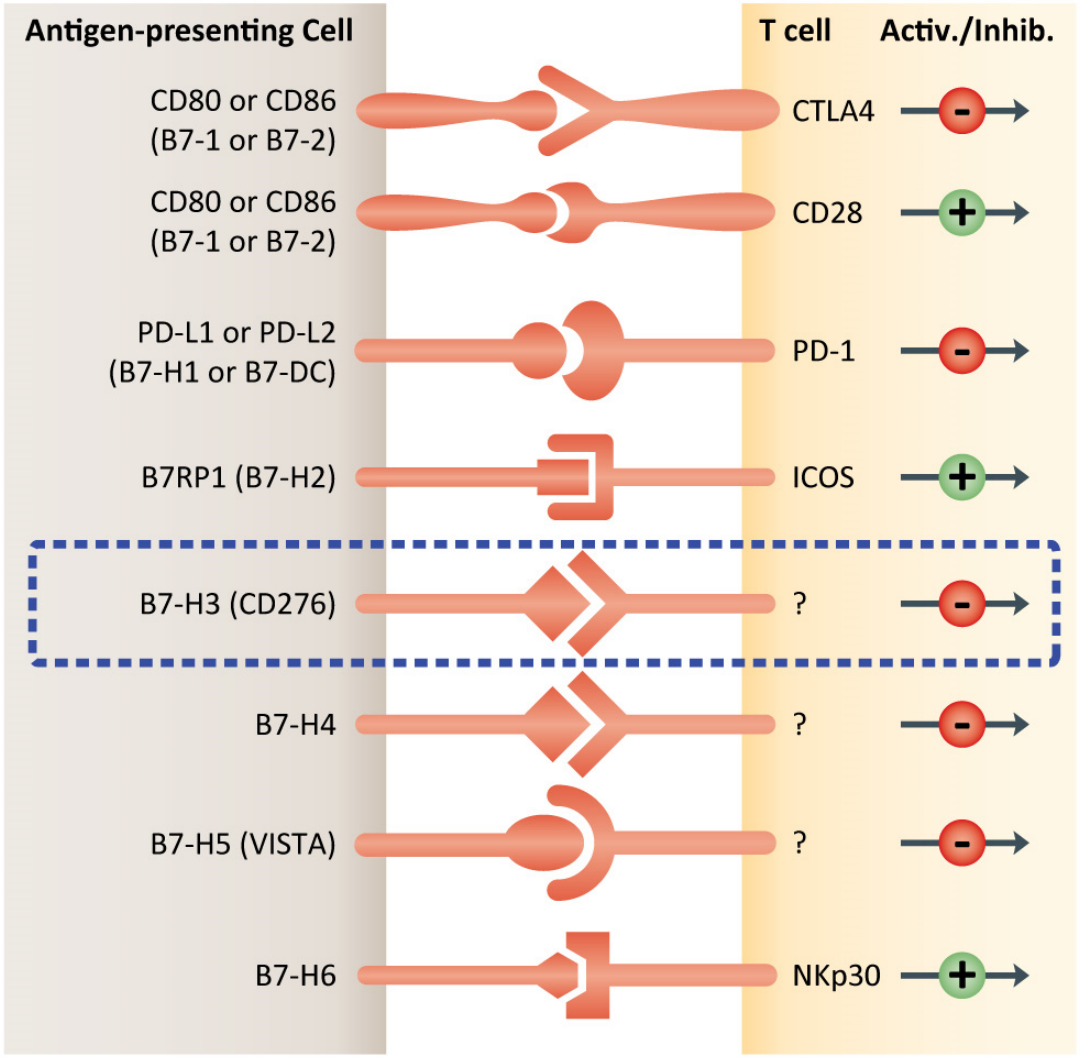
B7-H3: Member of B7 Family of Immune Regulators



Adapted from Pardoll, et al., Nature, April 2012.

Immunosuppressive Role

- Crystal structure resolved: T-cell inhibitory domain mapped *(Vigdorovich 2013)*
- Expression on lung cancer cells and macrophages suppresses T-cell mediated antitumor immune response *(Chen 2013)*
- B7-H3 negatively regulates acute GVHD *(Veenstra 2015)*

Tumor Metabolism & Metastatic Role

- Enhances metastatic potential of melanoma cells *(Tekle 2012)*
- Promotes epithelial-mesenchymal transition and expression of CSC markers in colorectal cancer (*Jiang 2016*)
- Reprograms glucose metabolism in breast cancer (*Lim 2016*)

(see handout w/ updated bibliography of B7-H3 scientific references)

Confirmed High Penetrance in Broad Set of Solid Tumors

Minimal/no expression on normal tissues

Fixed Tumor MicroArray	IHC Summary of Samples Screened			
	B7-H3 Positive		2+ or Above	
Potential Indications:				
Head and Neck	19/19	100%	19/19	100%
Kidney Cancer	77/78	99%	75/78	96%
Glioblastoma	65/66	98%	63/66	95%
Thyroid Cancer	34/35	97%	33/35	94%
Mesothelioma	41/44	93%	39/44	89%
Melanoma	132/146	90%	94/146	64%
Prostate Cancer	88/99	89%	51/99	52%
Pancreas Cancer	69/78	88%	45/78	58%
Bladder	134/156	86%	123/156	79%
Lung Cancer	324/379	85%	300/379	79%
Breast Cancer	189/249	76%	156/249	63%
Ovarian Cancer	59/79	75%	36/79	46%

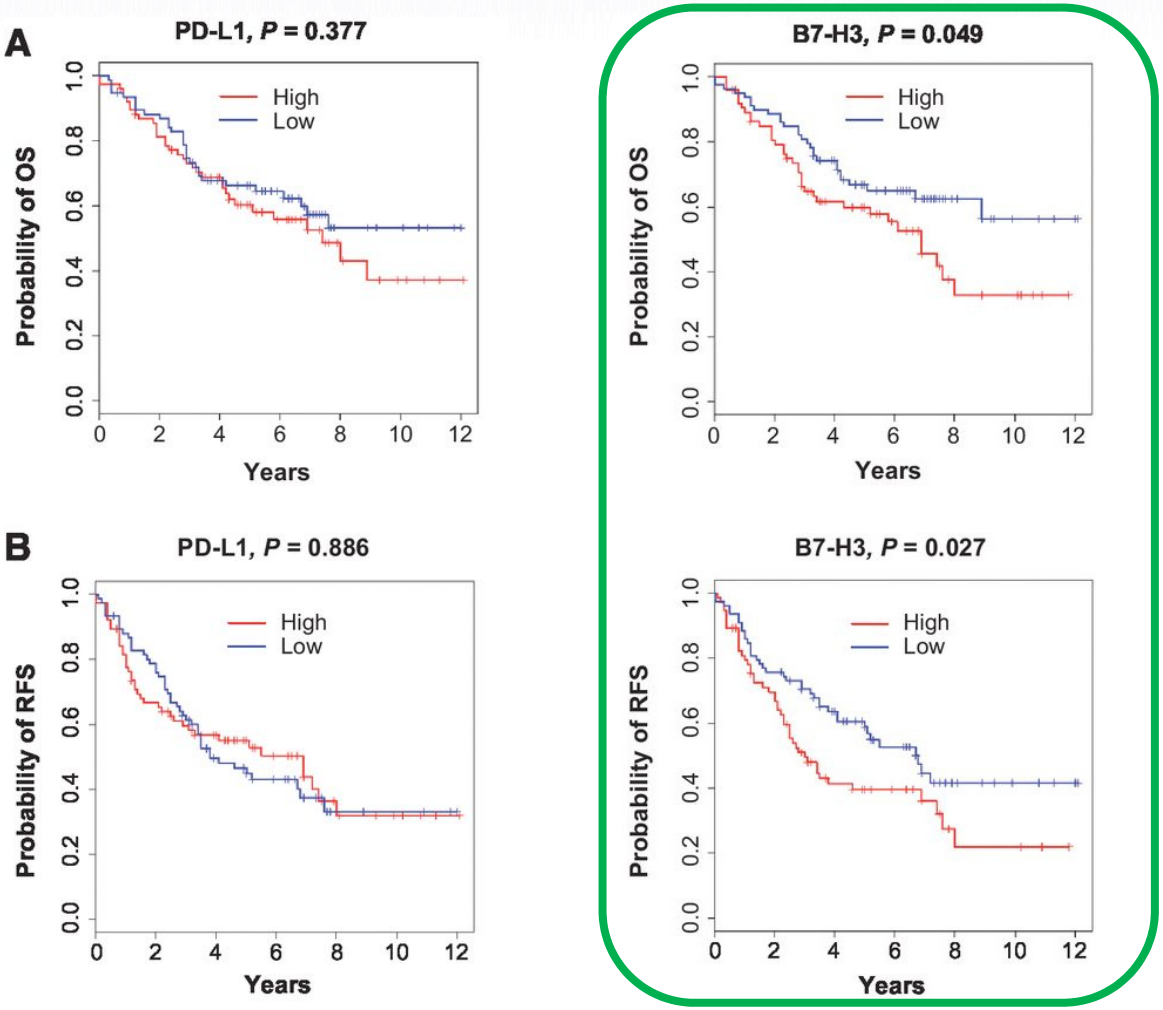
Target expression on both tumor cells and tumor vasculature

B7-H3 Identified as Prognostic Lung Cancer Biomarker

B7-H3 expression correlates with poor OS and RFS in NSCLC

Analyses of >450 lung adenocarcinoma patients revealed enhanced expression of checkpoint molecules (PD-1, PD-L1, PD-L2, B7-H3, CTLA-4, LAG-3, BTLA and TIM-3) in "mesenchymal" lung adenocarcinoma

Among checkpoints, only B7-H3 demonstrates correlation with overall survival and relapse-free survival across lung adenocarcinoma

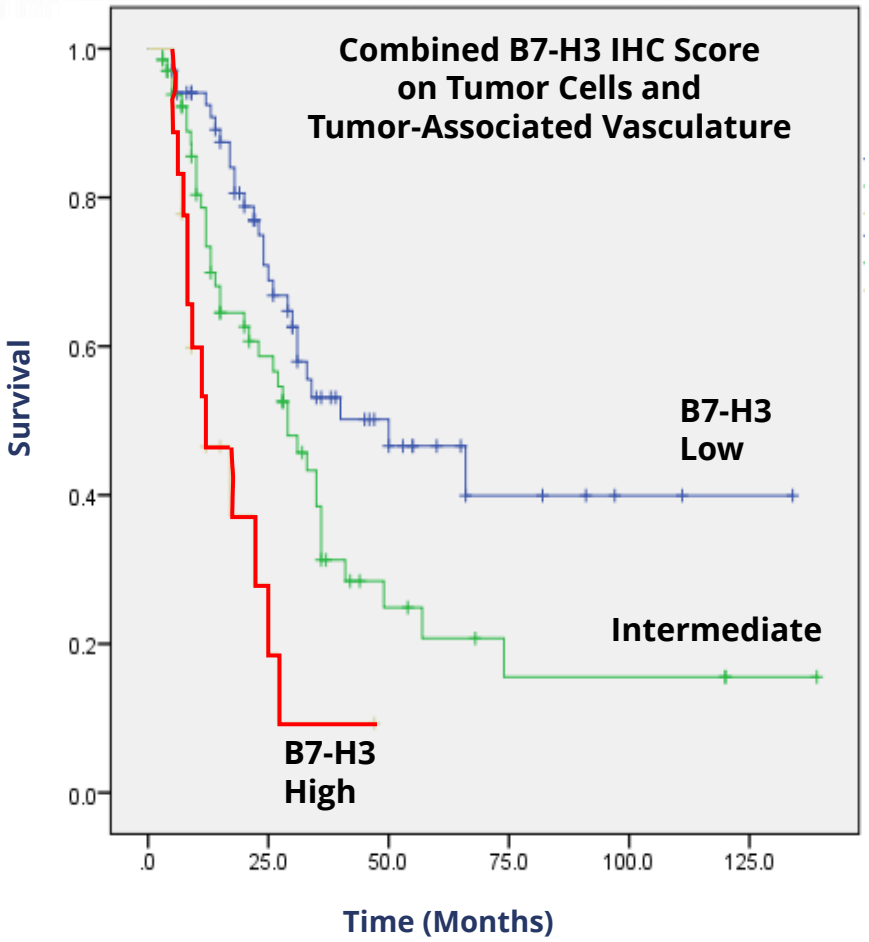


"Epithelial-mesenchymal transition is associated with a distinct tumor microenvironment including elevation of inflammatory signals and multiple checkpoints on lung adenocarcinoma"; Lou, Y, et al.; Clin Cancer Res. 2016 Jul 15;22(14):3630-42.

B7-H3 Expression Level Correlates w/ Patient Outcome

IHC analyses of 154 pancreatic ductal adenocarcinoma (PDAC) patients

- **High statistical inverse correlation between B7-H3 expression and patient survival**
- Consistent with prior studies, lower CD8 T-cell infiltration also correlates with poorer patient outcome



B7-H3 membrane expression determined using diagnostic test supporting enoblituzumab and MGD009 clinical studies; Ongoing collaboration with Mass General Hospital (Ferrone)

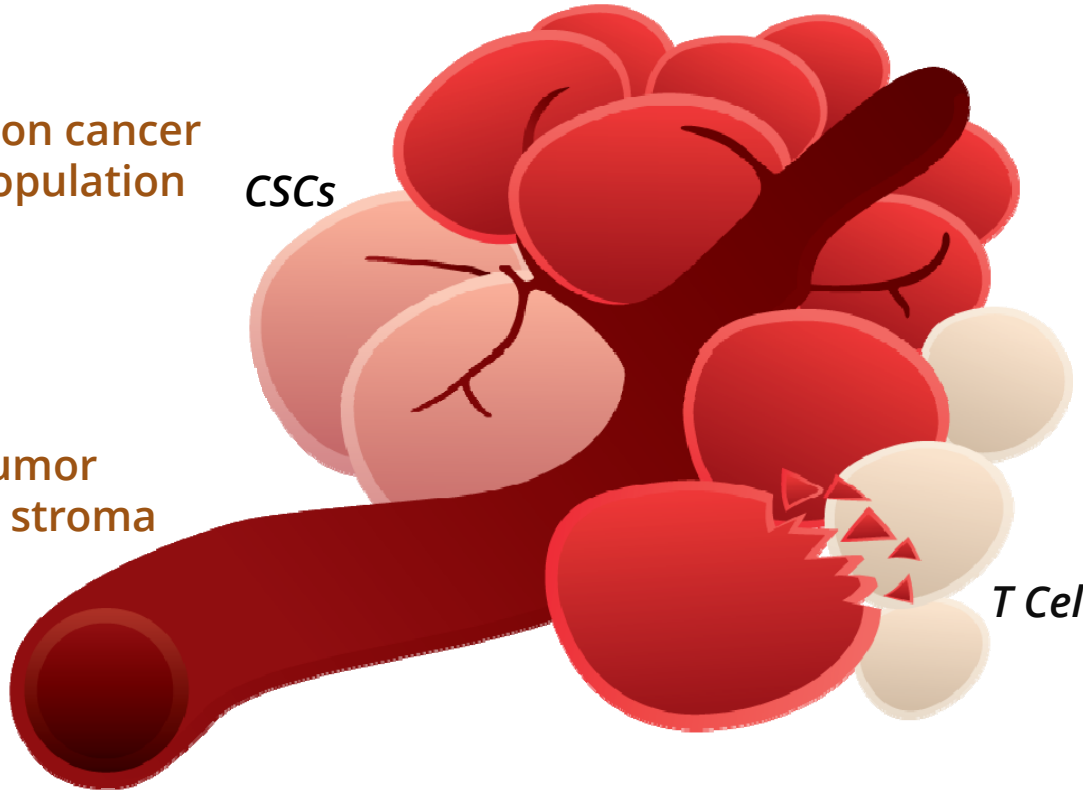
Rationale for Targeting B7-H3 in Cancer

Direct expression by primary and metastatic tumors

Tumor Cells

Expression on cancer stem cell population

CSCs



Expression on tumor vasculature and stroma

T Cells

Role in mediating migration, invasion, resistance and tumor metabolism

Role in T cell immune modulation

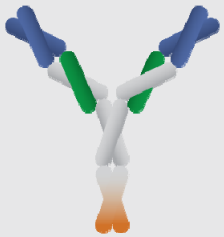
Tumor Vasculature

Comprehensive B7-H3 Franchise

MacroGenics retains global rights

	Enoblituzumab (MGA271)	MGD009	MGC018
Candidate	• Fc-optimized mAb 	• B7-H3 x CD3 DART (Fc-bearing) 	• B7-H3 Antibody-Drug Conjugate 
Potential MoA	• Direct tumor killing • Adaptive T-cell immune response enhancement	• Recruitment and expansion of T cells • Potent redirection of T cells to kill tumor cells	• Direct tumor killing • Leverage Synthon's drug-linker
Current Development Status	• Monotherapy Ph. 1 trial (7 solid tumor types) • Combo studies	• Phase 1 dose escalation	• 2018 IND planned

Enoblituzumab: "First-in-class," Fc-Optimized Anti-B7-H3 mAb

Candidate	• Humanized, Fc-optimized anti-B7-H3 mAb 
Opportunity	• Very large commercial opportunity for targeting B7-H3, given vast expression across many different solid tumor types
Targeted Indications	• Exploring seven solid tumor indications
Development	• Monotherapy Ph. 1 study • Combination studies ongoing with ipilimumab, pembrolizumab
Partner	• MacroGenics retains global rights

Enoblituzumab Monotherapy

Phase 1 Monotherapy Study: Seven Solid Tumor Types

Tumor-specific expansion cohorts

Initial Expansion Cohorts

Enrollment Complete

- **Prostate (n=20)**
- **Bladder (high B7-H3) (n=8)**
- **Melanoma (post-CPI failure) (n=31)**
- **Head & Neck (n=19)**
- **Triple-negative Breast (n=16)**
- **Renal Cell (n=16)**
- **NSCLC (high B7-H3) (n=8)**

New Expansion Cohorts

Enrollment Started October 2016

- **Prostate (n=16)**
- **Bladder (n=16)**

Note: Enoblituzumab Phase 1 monotherapy study; October 17, 2016 data cutoff

Favorable Safety Profile in Phase 1 Study

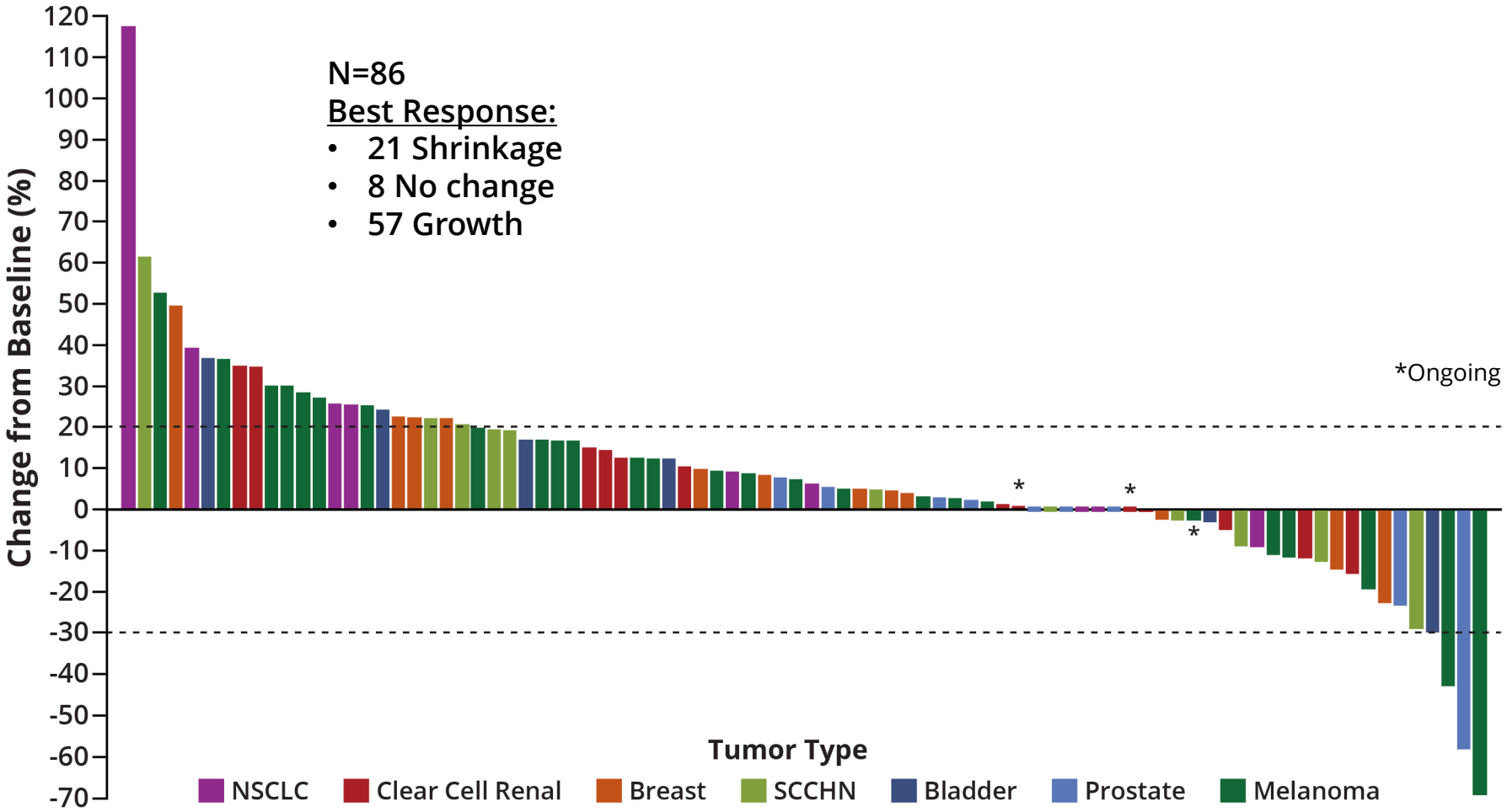
No severe immune-related AEs (15 mg/kg)

Drug-Related Adverse Event (≥ 10% of Patients)	No. (%) of Patients	
	All Grades (N=130)	Grades 3-4 (N=130)
Any adverse event	125 (80)	11 (9)
Infusion-related reaction	57 (37)	4 (3)
Fatigue	51 (33)	2 (2)
Nausea	31 (20)	0
Vomiting	20 (13)	0
Chills	23 (15)	0

Note: Enoblituzumab Phase 1 monotherapy study; October 17, 2016 data cutoff

Tumor Reduction in Heavily Pretreated Patients

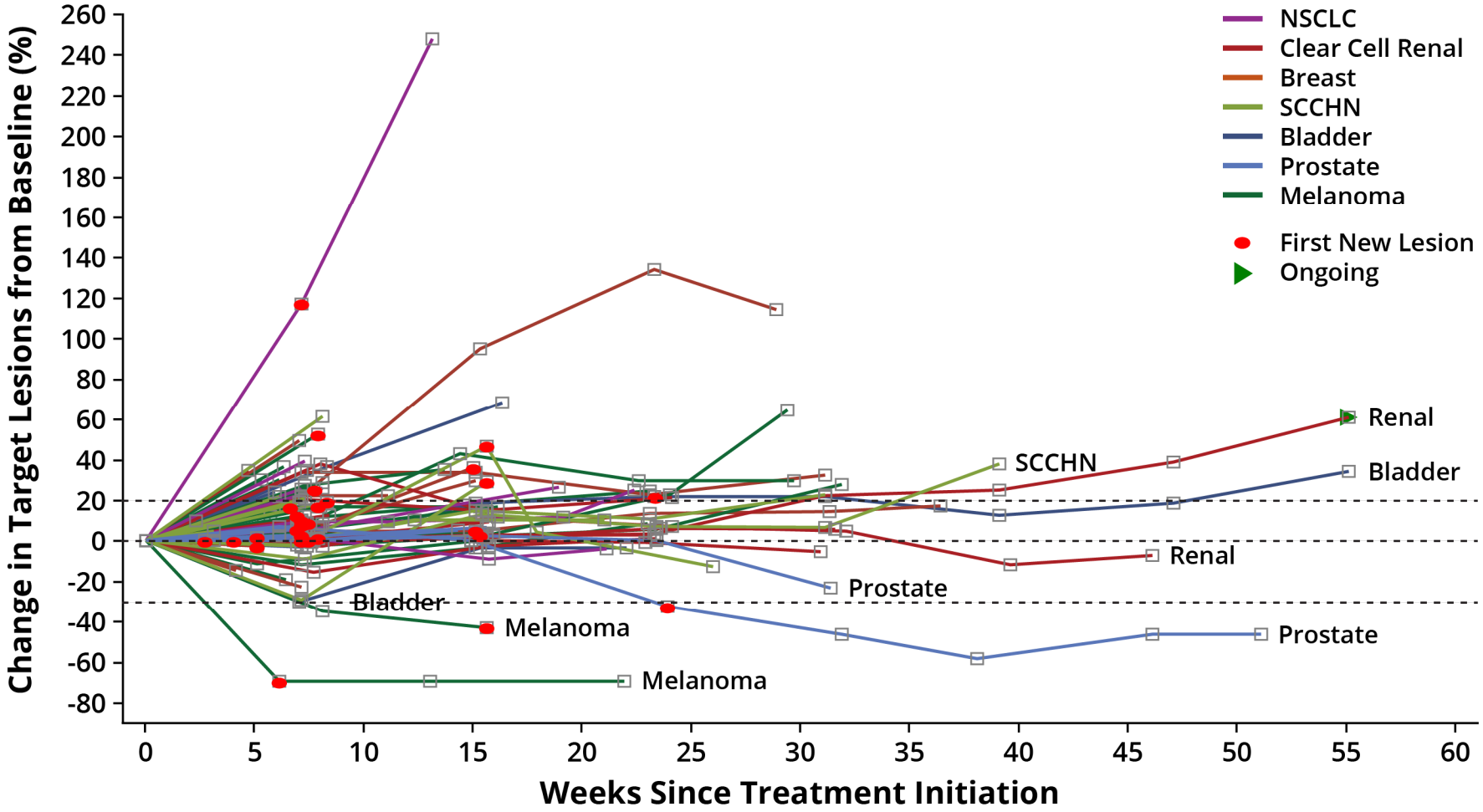
Best % change in response-evaluable tumor-specific cohorts (15 mg/kg)



Note: Enoblituzumab Phase 1 monotherapy study; October 17, 2016 data cutoff

Multiple Patients with Durable Tumor Reductions

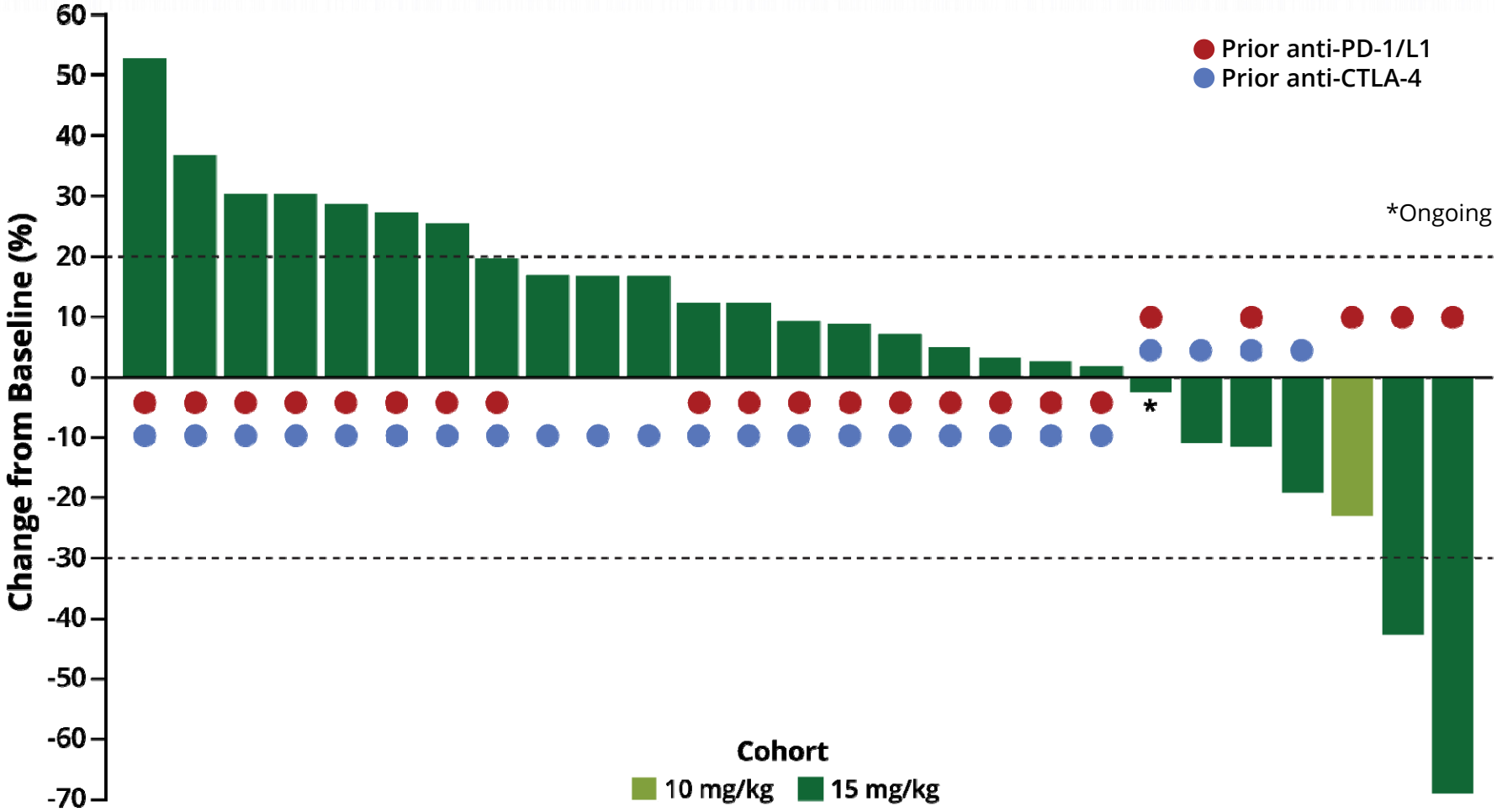
Best % change in response-evaluable tumor-specific cohorts (15 mg/kg)



Note: Enoblituzumab Phase 1 monotherapy study; October 17, 2016 data cutoff

Activity in Post-Checkpoint Melanoma Patients

Best % change in response-evaluable melanoma patients

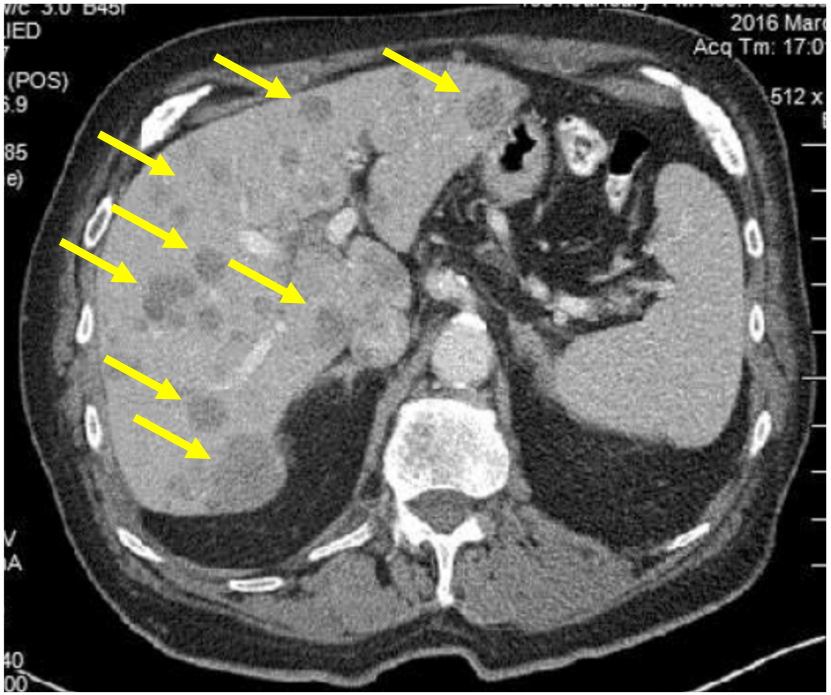


Note: Enoblituzumab Phase 1 monotherapy study; October 17, 2016 data cutoff

Rapid Reduction in Multiple Tumor Lesions

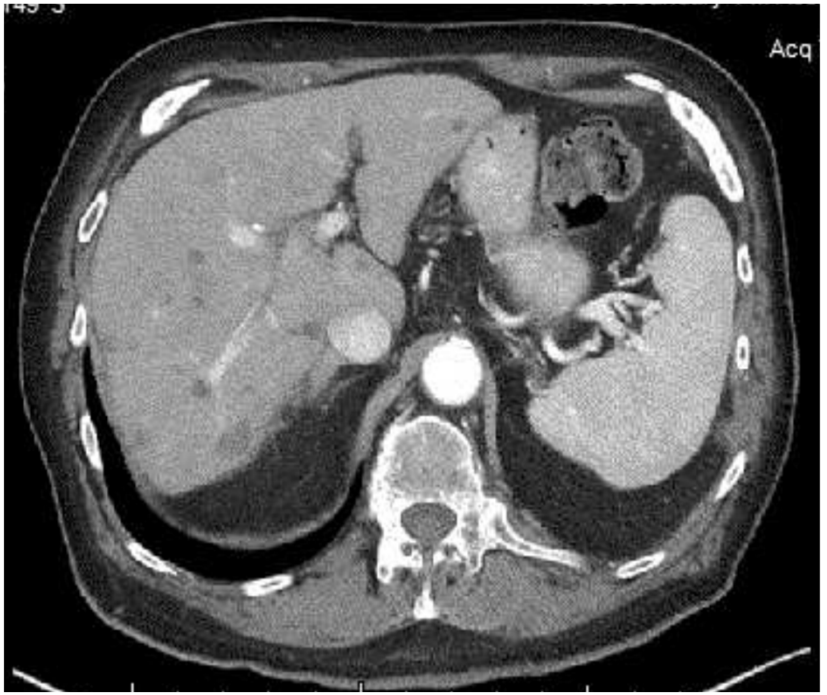
Enoblituzumab case study #1: 87 y/o man w/ metastatic melanoma

Baseline



- Melanoma patient had liver and bone metastases
- Had progressed on nivolumab
  - Treated Oct. 2015 to Jan. 2016
  - Only prior systemic cancer therapy

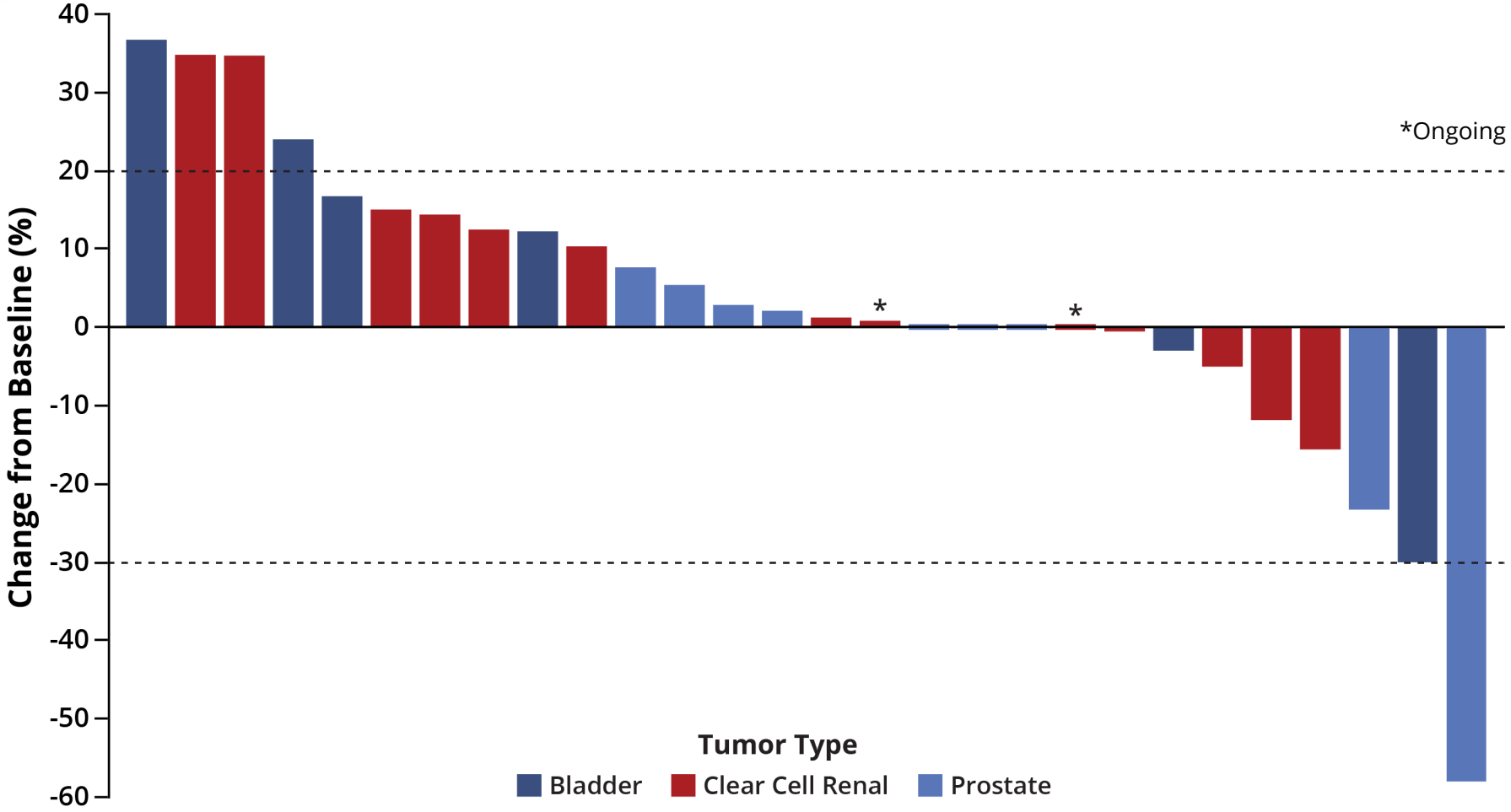
Day 100



- Enrolled in enoblituzumab monotherapy (Mar. 2016)
- **Rapid reduction in multiple tumor lesions**
  - Cycle 1: **35% shrinkage** (w/smaller bone lesions)
  - Cycle 2: **42% shrinkage**
- MRI showed small brain mets (no baseline MRI)
  - Gamma knife brain radiation

Activity in Urological Malignancies (Monotherapy)

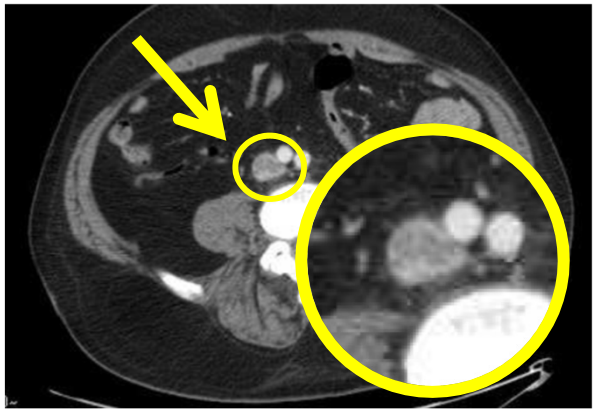
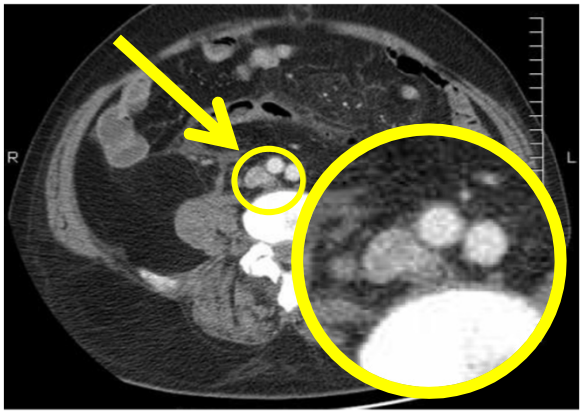
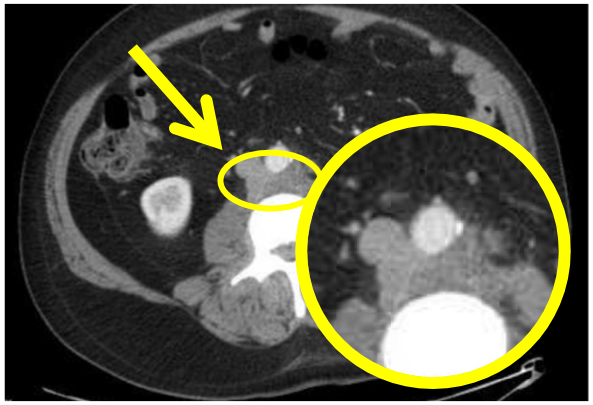
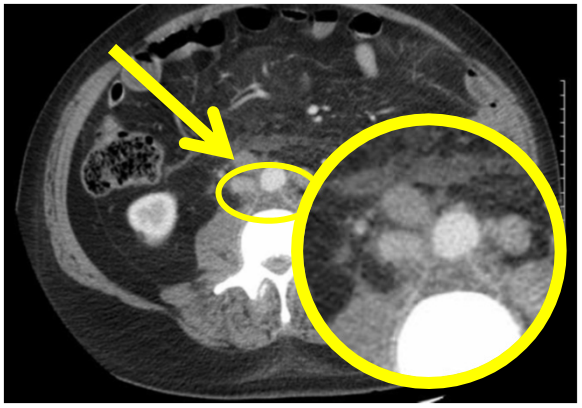
Best % change in tumor burden in response-evaluable patients (15 mg/kg)



Note: Enoblituzumab Phase 1 monotherapy study; October 17, 2016 data cutoff

Selected Prostate Patients with Antitumor Activity

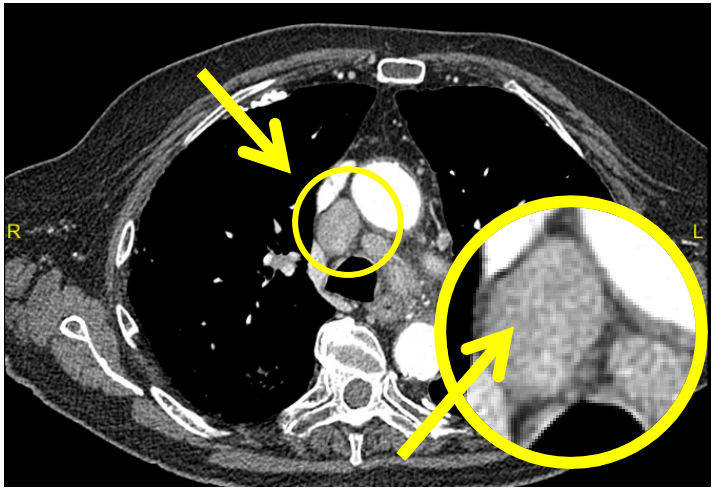
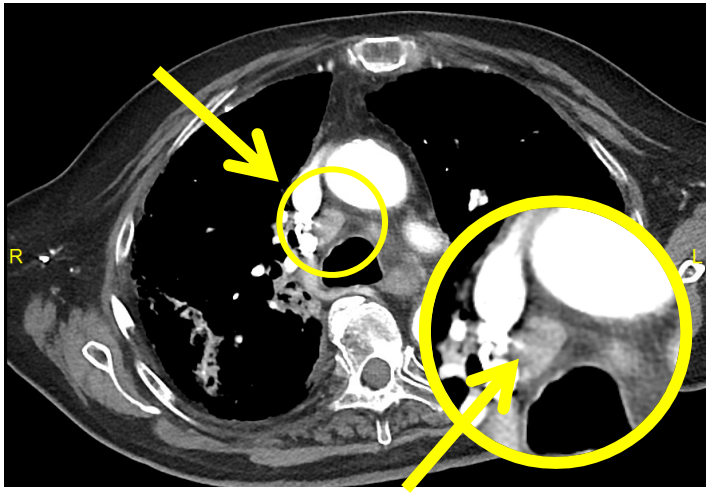
Enoblituzumab case study #1: 65 y/o man with prostate cancer

	Baseline	Day 220
Aorto-Caval Lymph Node		
Retro-Aortic Mass		
	• Mets: retroperitoneal LN, bone • Previous therapy: sipuleucel-T, abiraterone, enzalutamide, radium 223, taxotere	• Tumor: **23% reduction** RECIST • PSA: **46% decrease** • Remained on therapy for 13 mos.

Selected Prostate Patients with Antitumor Activity

Enoblituzumab case study #2: 87 y/o man with prostate cancer

Right Paratracheal Lymph Node

Baseline	Day 290
	
• Mets: mediastinal and retroperito-neal LN, bone, porta-caval LN • Previous therapy: radiation	• Tumor: **58% reduction** RECIST • PSA: **51% decrease**

Key Enoblituzumab Monotherapy Takeaways

- Well tolerated at doses of up to 15 mg/kg q week
  - Safety profile supports combinability with various other molecules, including checkpoint inhibitors
- Evidence of modulation of T-cell function, including induction of cytokine/chemokine production and enhanced clonality of T-cell repertoire
- Most promising monotherapy activity observed in prostate, bladder and post-checkpoint melanoma
  - Bladder and prostate cancer enrollment ongoing
  - Melanoma being further evaluated as monotherapy as well as in combo with anti-PD-1 or anti-CTLA-4

Enoblituzumab Combination Therapy

Rationale for Enoblituzumab Combination Studies

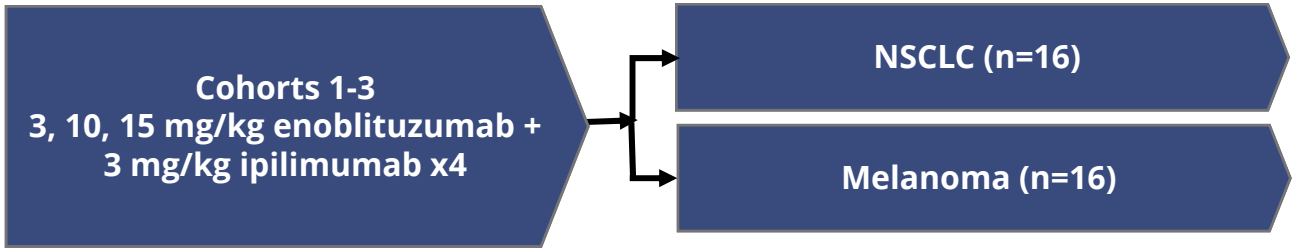
- Coordinate engagement of innate and adaptive immunity by combining agents that modulate T cell function and potentiate ADCC
- Combinations of two molecules targeting B7 family members can synergize clinically (e.g., anti-CTLA-4/PD-1)
- Anticipate easier combinability with either anti-CTLA-4 or anti-PD-1
  - Limited B7-H3 expression on normal cells appears to limit disruption of self tolerance and trigger of immune-related AEs (irAEs) by enoblituzumab
  - Improved risk-benefit compared to anti-CTLA-4/anti-PD-1 combination

Enoblituzumab + Anti-CTLA-4 Combination Study

Anticipate initiation of dose expansion in 2017

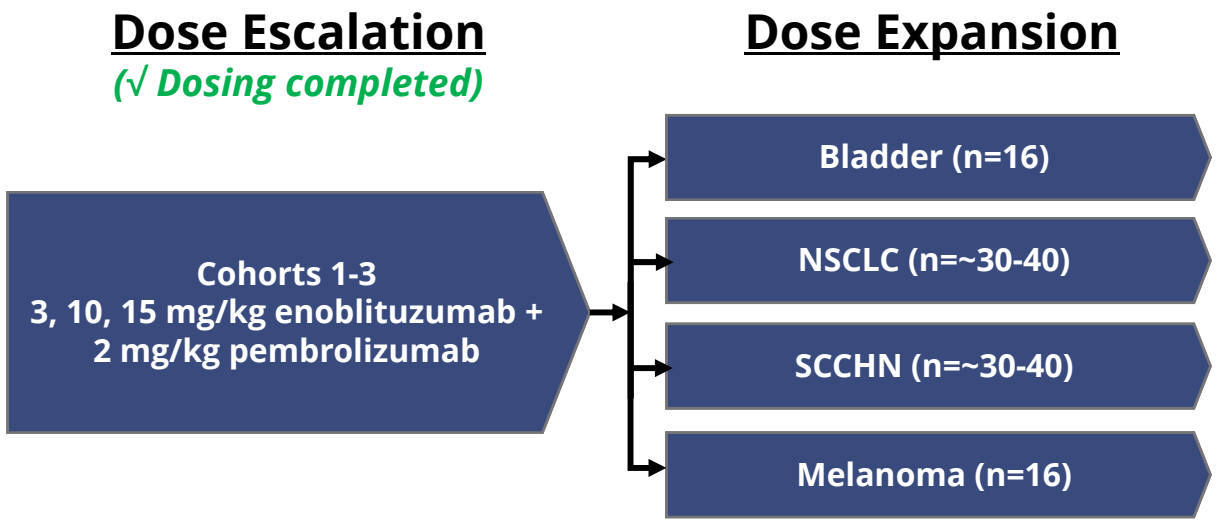
Dose Escalation
(currently completing Cohort 3)

Dose Expansion



Enoblituzumab + Anti-PD-1 Combination Study

Dose expansion ongoing

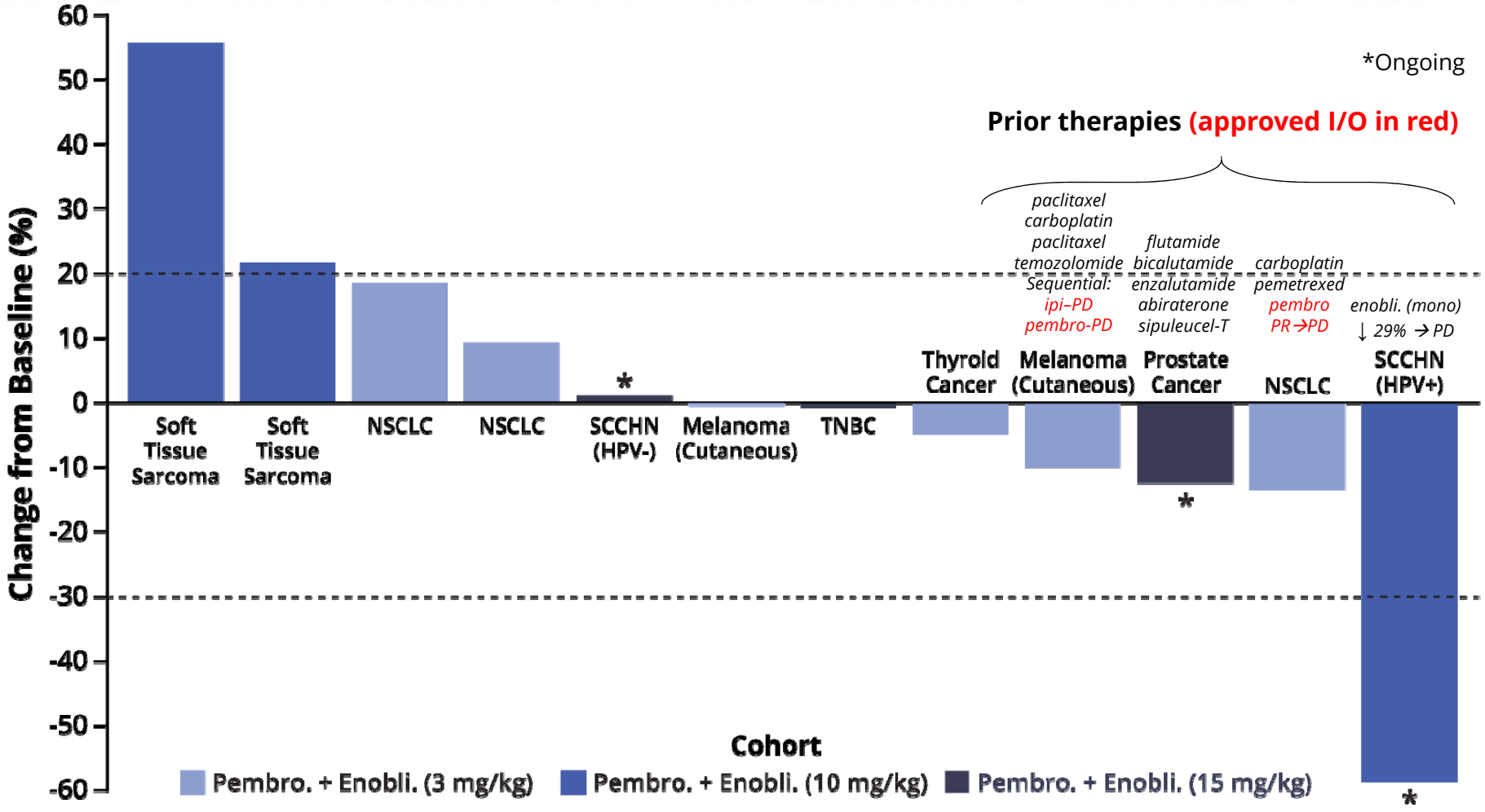


Observations to date

- Manageable AEs
- Initial signs of antitumor activity
- DLT period: first 6 weeks of dosing

Initial Signs of Activity in Heavily Pretreated Patients

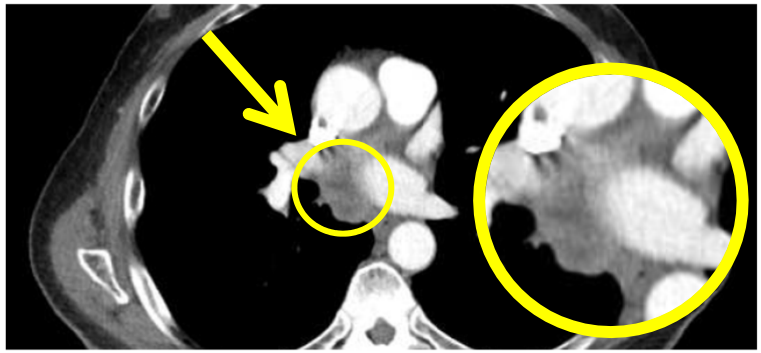
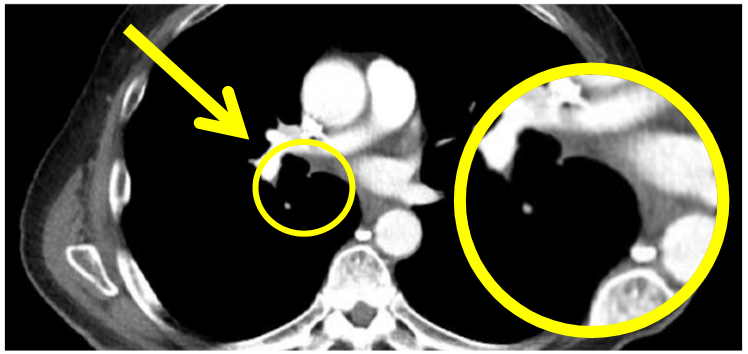
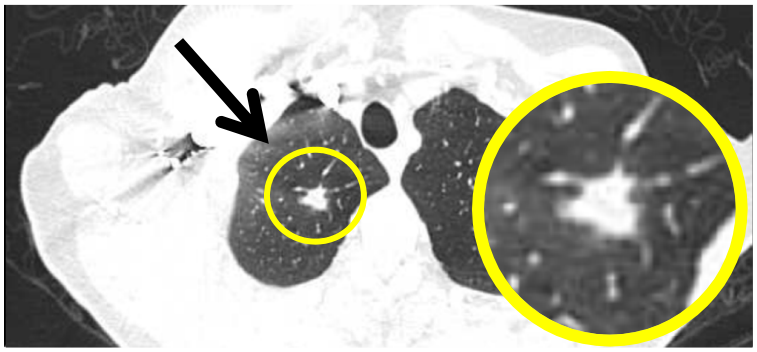
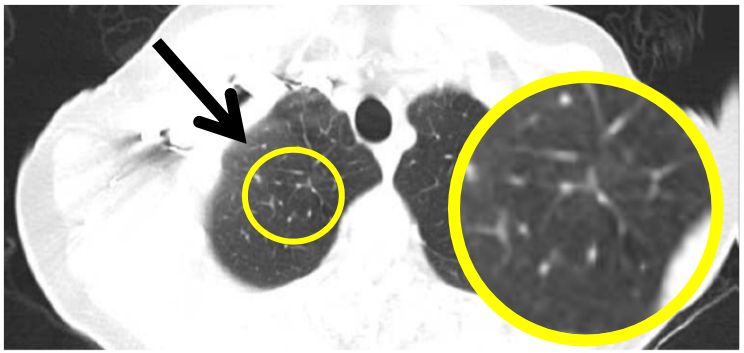
Best % change in tumor burden in response-evaluable patients



Note: Enoblituzumab + anti-PD-1 (pembrolizumab) combination study; October 17, 2016 data cutoff

Enoblituzumab + Anti-PD-1: Confirmed PR in SCCHN

Case study: 70 y/o man w/ unresectable locally adv./metastatic HPV+ SCCHN

	Baseline	Cycle 2
Mediastinal Lymph Node		
RUL Lung Mass		

- Disease in submandibular lymph nodes, bone, lung & mediastinum
- Only prior systemic cancer therapy was enoblituzumab (mono)
  - Rapid tumor reduction of 29.2% on Cycle 1 w/ subsequent regrowth / PD

- 10 mg/kg enoblituzumab qW + 2 mg/kg / pembrolizumab q3W (dose esc. Cohort #2)
- **Confirmed PR after 2 cycles**
- Rapid **tumor reduction of 45%** on Cycle 1
- Subsequent **further reduction to 58%**

Enoblituzumab: Advancing Combination Therapy

- Combine administration of enoblituzumab with checkpoint inhibitors to achieve additive or synergistic antitumor activity
- Identify enoblituzumab combination with clinical activity in tumors where anti-CTLA-4 or anti-PD-1 have limited activity
- Define enoblituzumab-based combination regimens that are superior to anti-CTLA-4 or anti-PD-1 alone in tumor types where these agents are active
- Identify enoblituzumab-based regimens that benchmark favorably against existing combos (i.e., anti-CTLA-4/anti-PD-1) — safety, efficacy or both

MGD009: Expanding B7-H3 Franchise

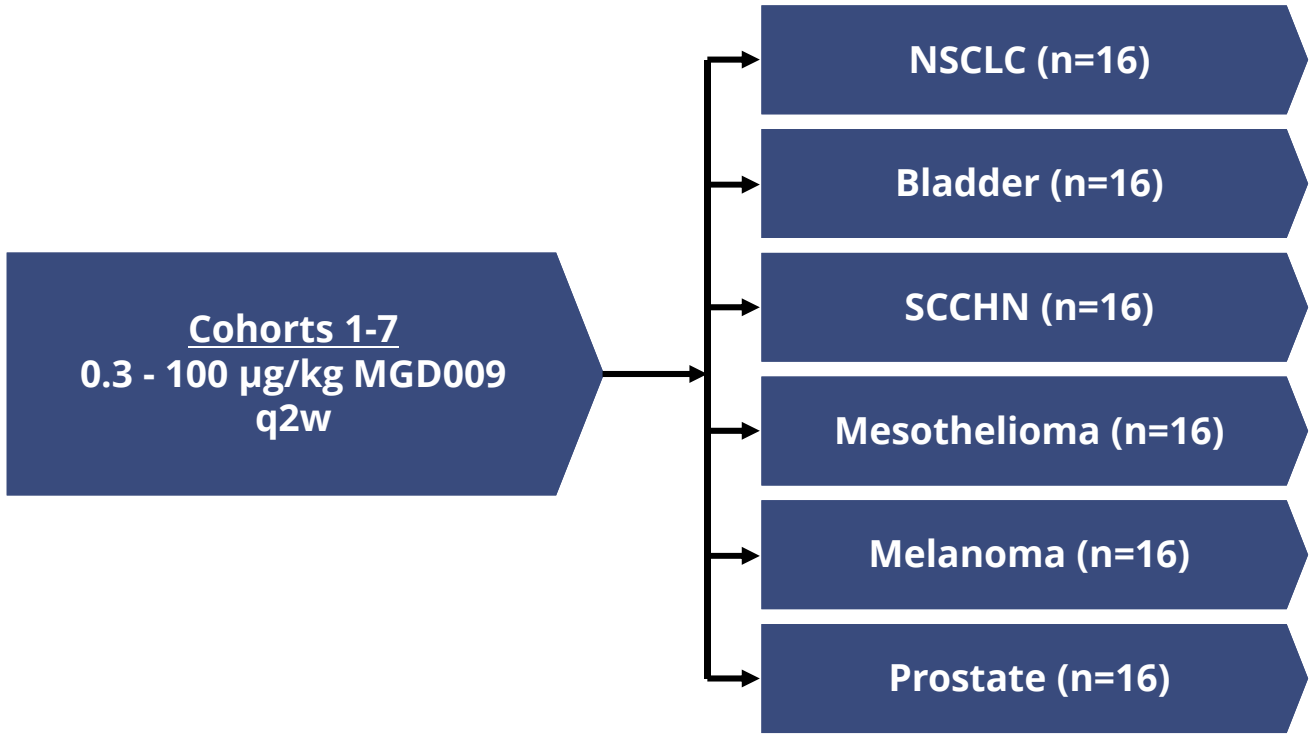
Candidate	• Humanized, Fc-bearing B7-H3 x CD3 DART 
Opportunity	• Large opportunity (vast expression across different tumors types) • B7-H3 expression correlated with disease severity and outcome
Function/MoA	• Redirected T-cell killing – Recruitment and activation of T cells, irrespective of TCR specificity and MHC restriction – Potential expansion of tumor-specific T cells • MoA complementary to enoblituzumab: – Fc-mediated killing & priming for optimized T-cell response
Indications	• NSCLC, melanoma, head & neck, mesothelioma, bladder, others
Development	• Phase 1 study ongoing (dose escalation)
Partner	• MacroGenics retains global rights

MGD009 Phase 1 Trial Dose Escalation Ongoing

Dose Escalation: 3 + 3 + 3 Design

(Open to selected B7-H3+ tumor types)*

Dose Expansion

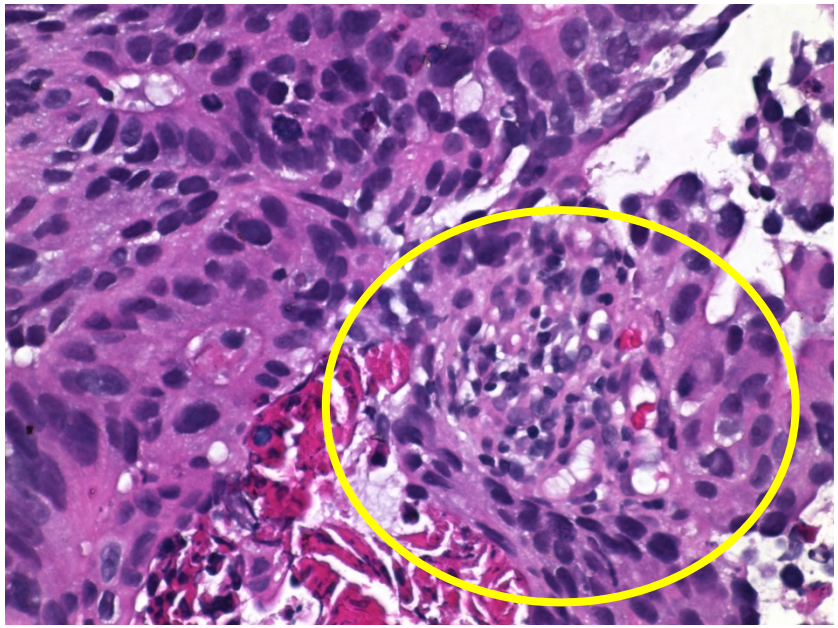


** B7-H3 positivity defined as > 10% tumor, ≥ 2+ and/or > 25% of vasculature positive*

Lymphocytes Associated w/ Areas of B7-H3 Expression

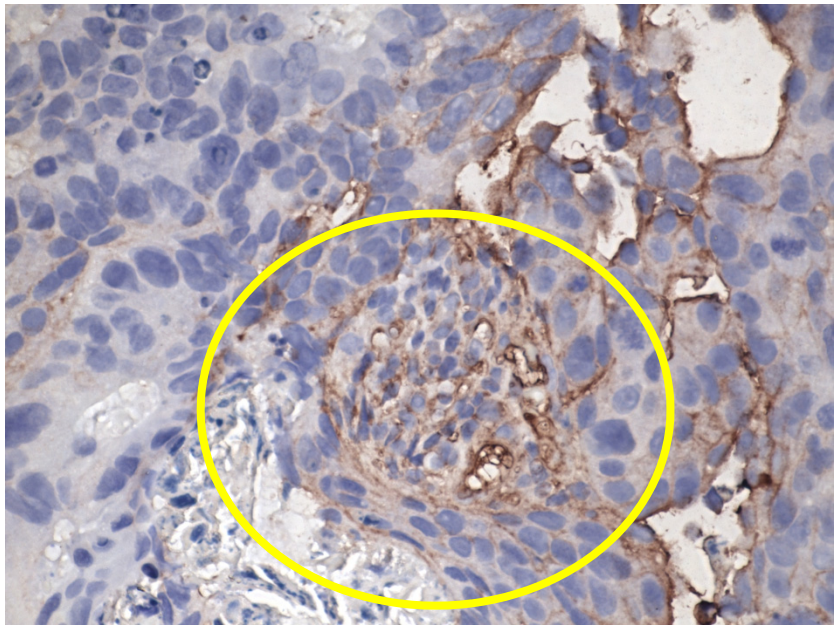
Tumor biopsy in MGD009 patient (dosed 1 µg/kg)

H&E Staining



- Glandular architecture
- Lymphocytes within tumor region
- Non-viable tumor cells associated with inflammatory cell infiltration

Anti-B7-H3 Staining



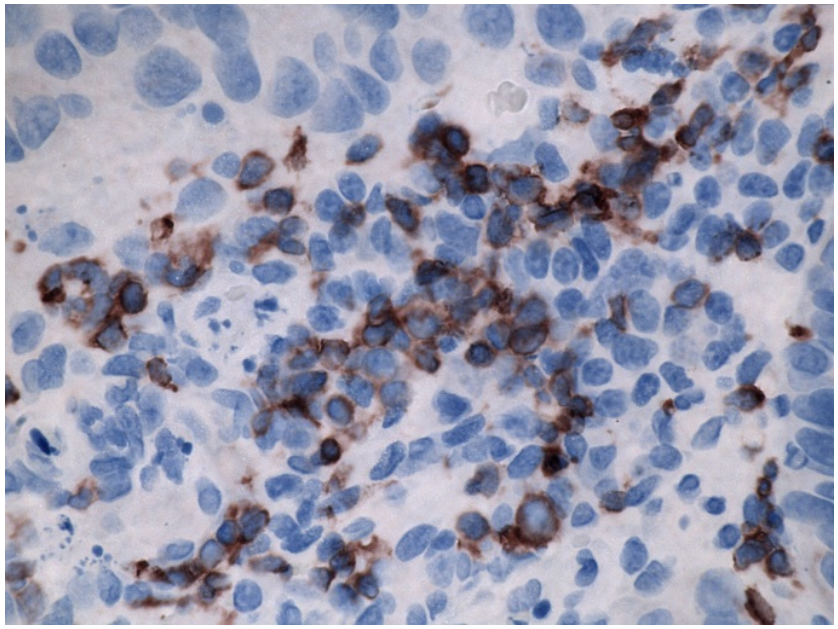
R&D Systems Goat anti-hu B7-H3, 1 µg/mL

- B7-H3 positive tumor cells surrounded by lymphocytes

T-cell Recruitment and Proliferation at Tumor Site

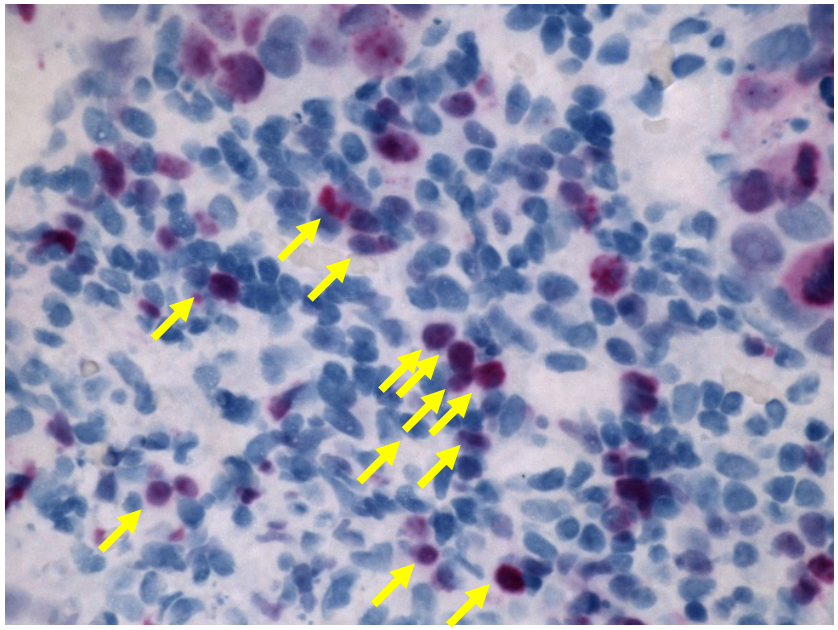
Tumor biopsy in MGD009 patient (dosed 1 µg/kg)

CD3 Staining



- Lymphocyte infiltration (brown) adjacent to tumor cells

Ki67 Staining (Proliferation Marker)



- Subset of infiltrating lymphocytes (some indicated with arrows) are proliferating (red)

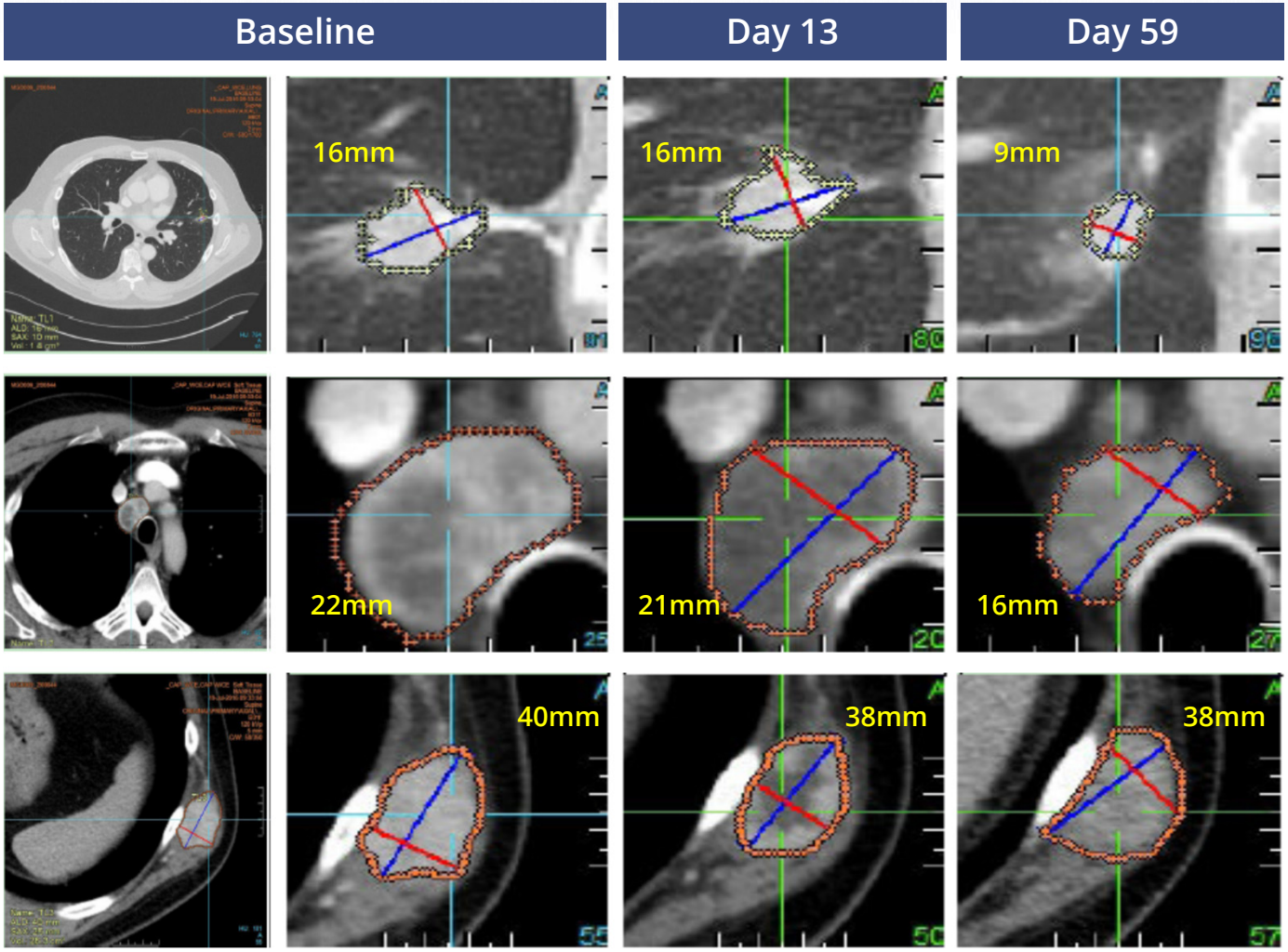
MGD009 Dose Escalation: Antitumor Activity in RCC

Case study #1: 60 y/o man with metastatic RCC

- Patient had metastases in brain, mediastinal LN, lungs, liver and subcutaneous chest wall
- Progression on multiple previous systemic therapies:
  - IL-2, sorafenib, temsirolimus, nivolumab and sunitinib
- After 1st dose of MGD009 at 10 µg/kg
  - Transient infusion reaction considered DLT (fever/chills/rash, transient increase in liver and renal function tests)
- Rapid antitumor activity observed 2 weeks after 1st dose
  - Subcutaneous chest lesion palpably softer vs. baseline (noticed within first week)
  - Decreased vascularity of multiple lesions and lymph nodes with central necrosis
  - 4% Tumor reduction on CT scan
- Treatment continued x 1 dose: **tumor reduction 19%**
  - Patient required brain radiation for new brain disease: treatment discontinued

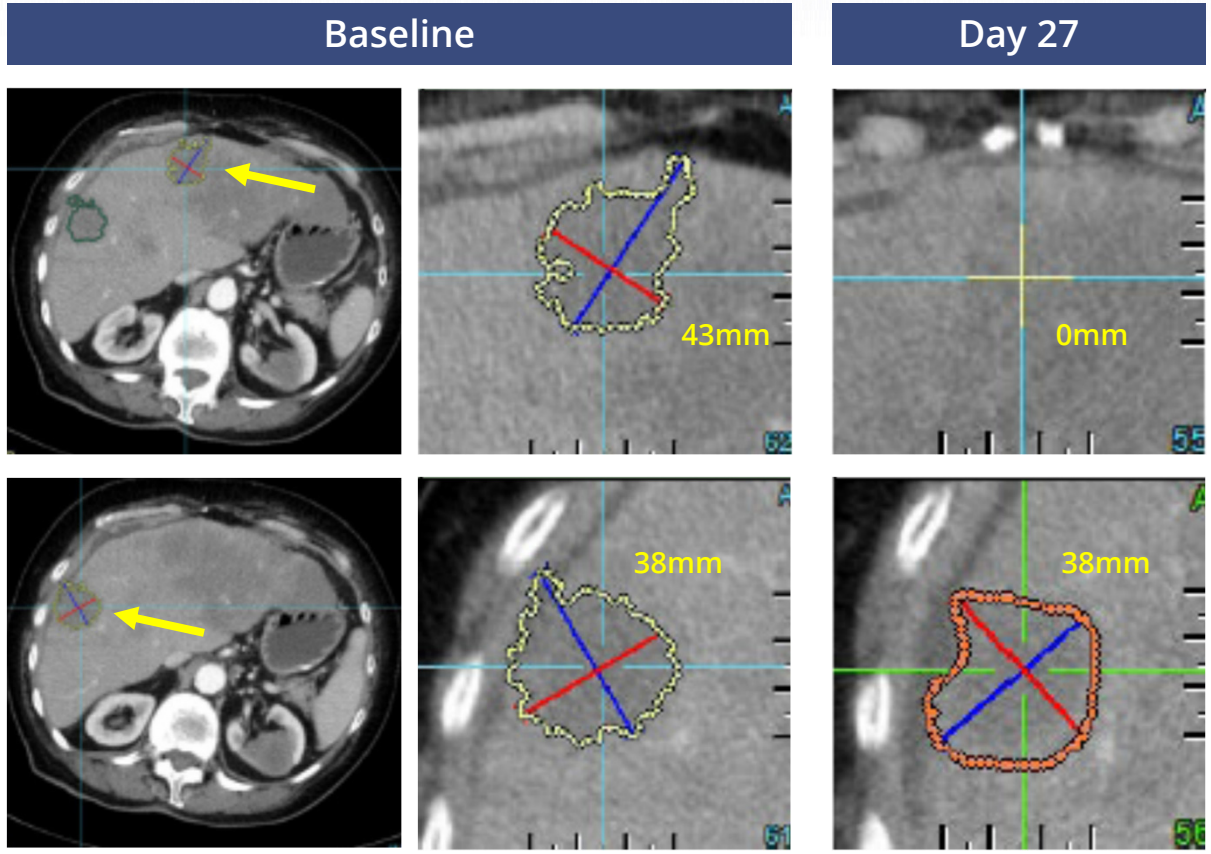
MGD009 Dose Escalation: Antitumor Activity in RCC

Case study #1: 60 y/o man with metastatic RCC



MGD009 Dose Escalation: Antitumor Activity in TNBC

Case study #2: 78 y/o woman with metastatic TNBC to lung & liver (dosed 10 µg/kg)

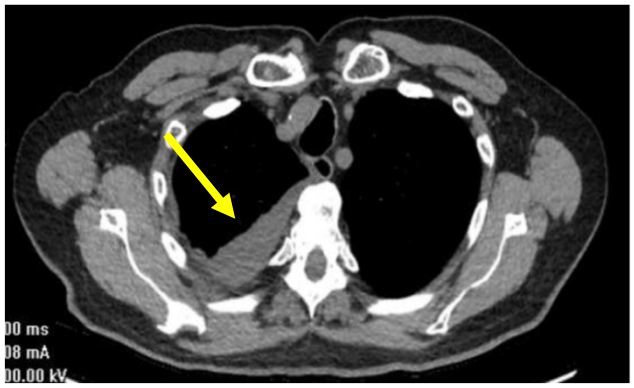
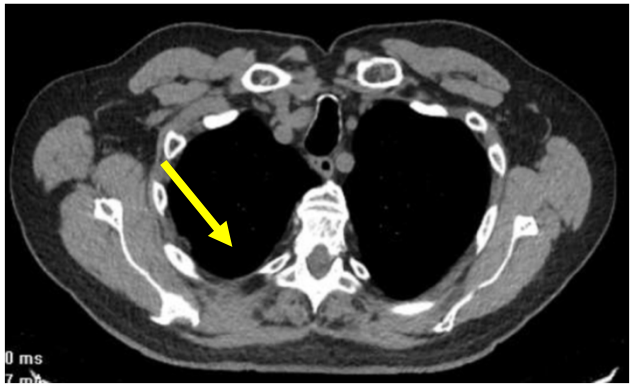
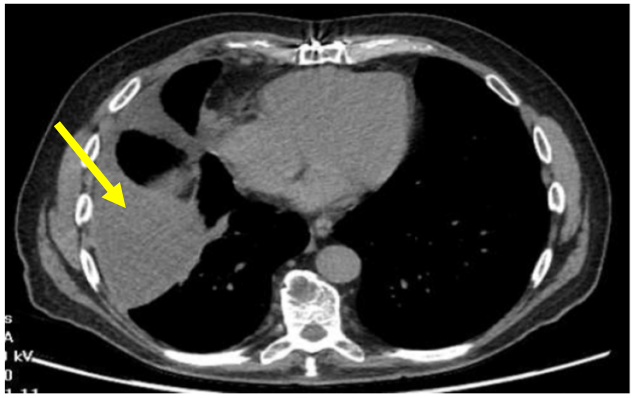
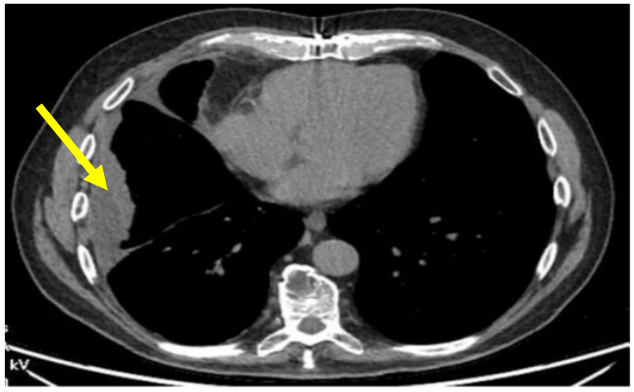


- Previously on enoblituzumab + pembro. study
- Stable disease, but discontinued after 1st Cycle
- Secondary to previous medical conditions/ low-grade infusion reactions

- Received two doses MGD009
- Discontinued following infusion reaction, transient transaminase elevation
- Rapid **20% tumor reduction** observed

MGD009 Dose Escalation: Antitumor Activity in Mesothelioma

Case study #3: 82 y/o man with pleural mesothelioma (dosed 10 µg/kg)

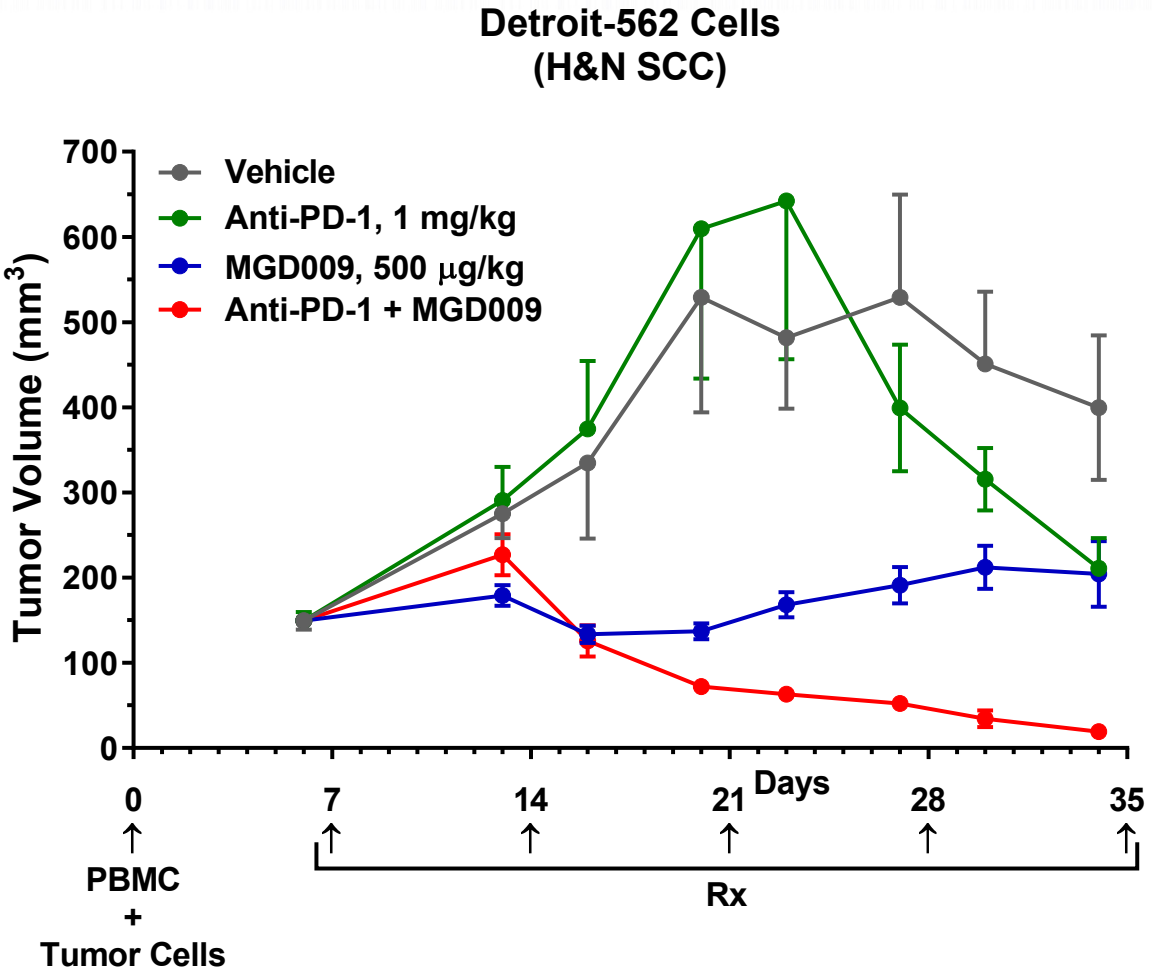
	Baseline	Day 45
Upper Chest		
Mid Chest		

- Pleural tumors with large loculated effusion
- Progression on cisplatin /pemetrexed

- Rapid antitumor activity after 1st Cycle
  - Resolution of loculated effusion
  - Tumor stable after 1st dose
  - Symptomatic improvement
- Continues on 2nd Cycle of treatment

Anti-PD-1 Enhances MGD009-mediated T-cell Killing in Vivo

Opportunity for combinatorial clinical strategy in solid tumors

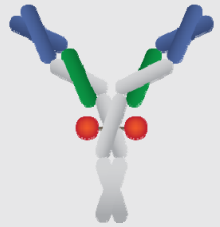


NSG MHC-I$^{-/-}$ mice, 5x10^6 tumor cells ID, PBMC, 10^6 cells IP

MGD009: Advancing DART Molecule in Solid Tumors

- Dose escalation ongoing in Cohort #4 (10 µg/kg)
  - Manageable AEs
  - Initial signs of antitumor activity observed
- MoA complementary to enoblituzumab
  - Enoblituzumab promotes Fc-mediated killing by NK and macrophages, primes for optimized T cell response
  - MGD009 recruits and activates T cells (irrespective of TCR specificity) with potential expansion of tumor-specific T cells
- Plans for future combination studies with anti-PD-1

MGC018: B7-H3 Antibody-Drug Conjugate

Candidate	• Humanized B7-H3 antibody drug conjugate • Alternate epitope, non-overlapping with enoblituzumab and MGD009 • Drug-linker licensed from Synthon Biopharmaceuticals 
Opportunity	• Complementary mechanism for targeting B7-H3 – Targeted payload delivery – Potential for de-bulking and combination strategies • Large opportunity given broad B7-H3 expression across tumor types
Function/MoA	• Duocarmycin-based payload with cleavable peptide linker – Highly potent DNA-damaging agent – Targets non-dividing cancer stem cells as well as dividing tumor cells – Not subject to multi-drug resistance (MDR)
Status	• IND targeted for 2018 • Favorable potency in B7-H3 xenografts • Acceptable non-human primate toxicology profile
Partner	• MacroGenics retains global rights

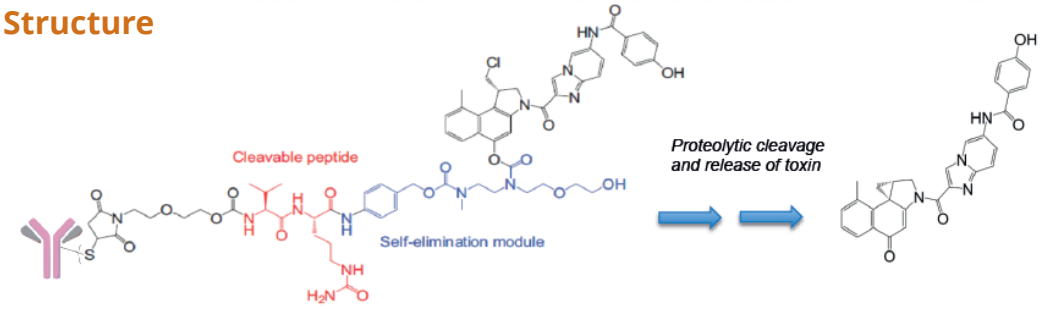
Duocarmycin-based Linker Drug Payload

vc-seco-DUocarmycin-hydroxyBenzamide Azaindole (DUBA)

- Fully synthetic, cell-cycle independent, DNA alkylating agent
- Retain activity in MDR lines
- Cleavable peptide linker
  - Facilitates bystander effect
- Clinical-stage
  - Synthon's SYD985 in Ph. 1
    - Anti-HER2-DUBA

Synthon

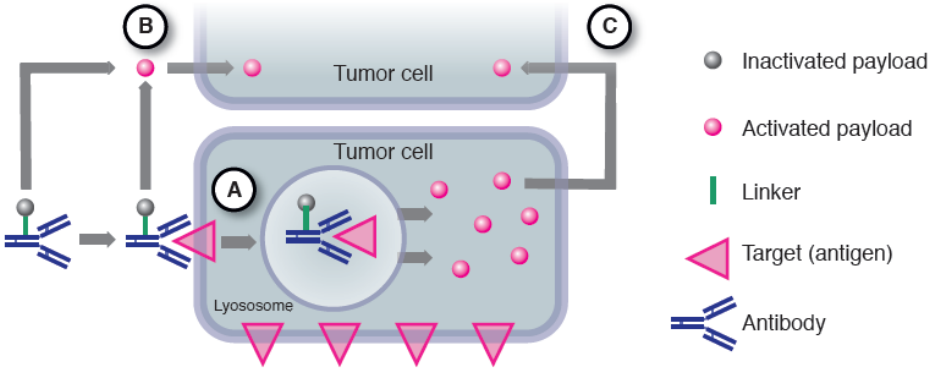
Structure



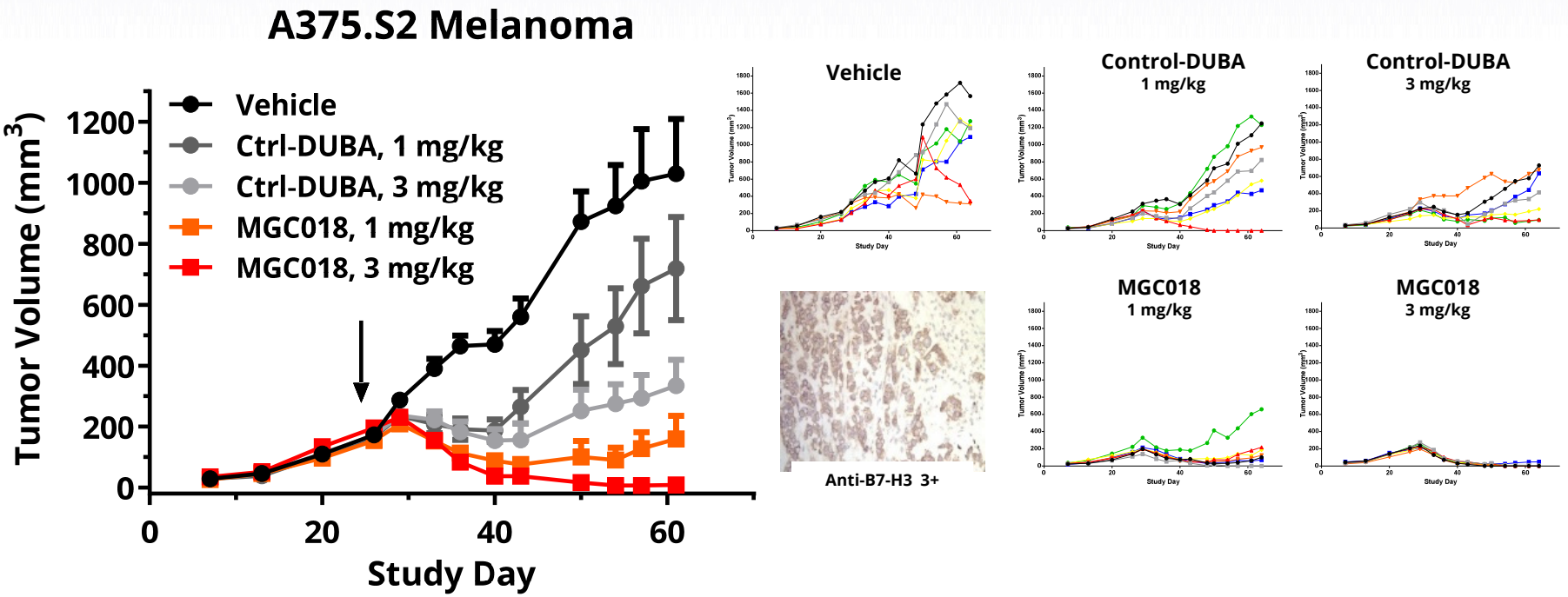
Active Toxin (DUBA)

Mode of Action

- Uptake of ADC by internalization and intracellular release of payload (A)
- Proteolytic cleavage of payload in tumor microenvironment (B)
- Diffusion of active payload to neighboring tumor cells (C)



MGC018: Potent Activity in Xenograft Models



Treatment	Dose qW (mg/kg)	Tumor Control Ratio (%)	Complete Response
Control mAb-DUBA	1	70	1/7
Control mAb-DUBA	3	33	0/7
MGC018	1	16	1/7
MGC018	3	1	5/7

B7-H3 Franchise Summary

- Broad target expression on most solid tumors
- Ideal target for multiple mechanisms of action
  - Fc-mediated killing and antigen presentation (enoblituzumab)
  - T-cell re-targeting (MGD009)
  - ADC (MGC018)
  - Combination therapy
- Encouraging data from ongoing enoblituzumab and MGD009 trials
  - Acceptable safety profile to date
  - Initial antitumor activity
  - Rationale for combination with anti-PD-1
- Upcoming milestones:
  - *Enoblituzumab - 2017*
    - Complete enrollment in new expansion cohorts for bladder and prostate cancer
    - Define future development plans based on monotherapy and combination study results
  - *MGD009:* enroll patients in expansion cohorts in 2017
  - *MGC018 (ADC)*: submit IND in 2018

Today's Agenda

Welcome
Scott Koenig, M.D., Ph.D. – President & CEO, MacroGenics

Margetuximab: Potential Best-in-Class Anti-HER2 mAb
Jon Wigginton, M.D. – Senior VP, Clinical Development & CMO, MacroGenics

Comprehensive B7-H3 Franchise: Fc-Optimized mAb, DART and ADC
Paul Moore, Ph.D. – VP, Immunology & Cell Biology, MacroGenics
Jim Vasselli, M.D. – VP, Clinical Development, MacroGenics

DART and TRIDENT: Leading Multi-specific Antibody Platforms
Syd Johnson, Ph.D. – VP, Antibody Engineering, MacroGenics

Clinical DART Programs Update
Jon Wigginton, M.D. – Senior VP, Clinical Development & CMO, MacroGenics

Q&A / Break

"Combination Treatments with Checkpoint Blockade"
F. Stephen Hodi, Jr., M.D. – Director, Center for Immuno-Oncology, Dana-Farber Cancer Institute

Immuno-Oncology: Targeting Immune Regulators
Jon Wigginton, M.D. – Senior VP, Clinical Development & CMO, MacroGenics
Ezio Bonvini, M.D. – Senior VP, Research & CSO, MacroGenics

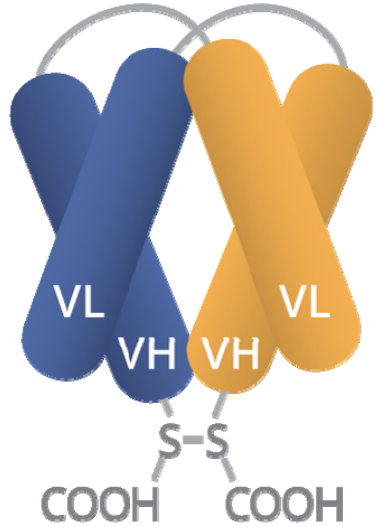
Wrap-up / Q&A
Scott Koenig, M.D., Ph.D. – President & CEO, MacroGenics

DART Platform

DART: The Most Advanced Bispecific Platform

- Robust, flexible bispecific platform
  - Multiple applications across different disease areas
  - Predictable manufacturability
  - Long-term structural stability
  - Ability to tailor half-life and valency
- Six DART molecules in clinical testing(1)
- Multiple pre-clinical programs advancing
- Validating DART collaborators

Basic DART
(No Fc Domain)

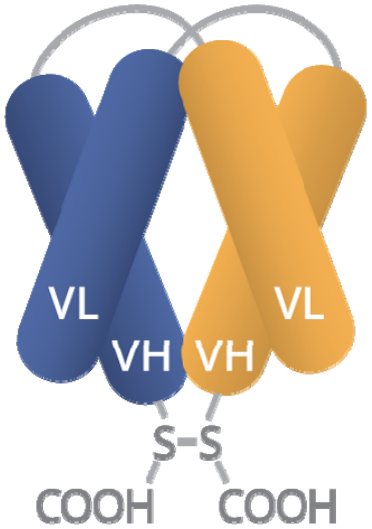


Boehringer Ingelheim
SERVIER
Takeda
NIH
National Institute of Allergy and Infectious Diseases
Pfizer
janssen

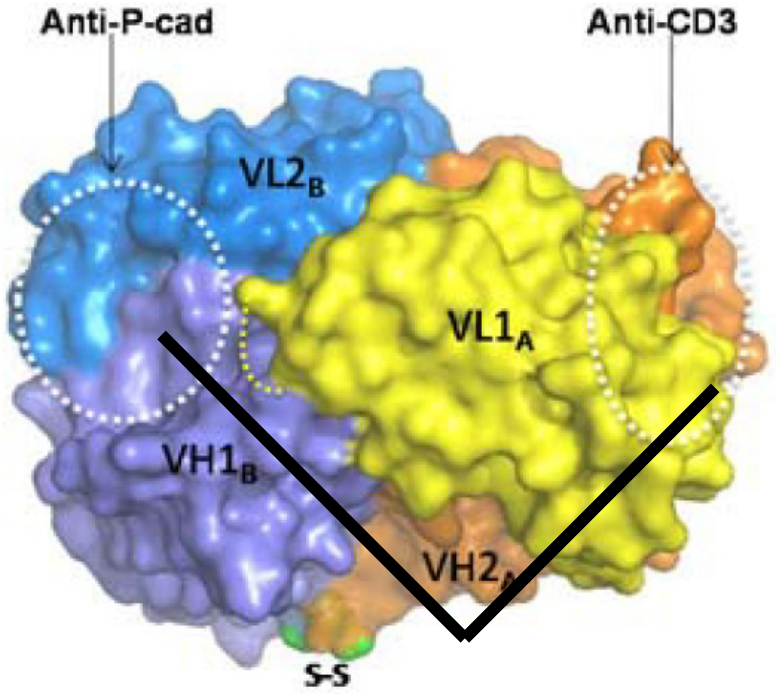
(1) Two clinical DART molecules are being developed by collaboration partners (MGD011/duvortuxizumab by Janssen and PF-06671008 by Pfizer).

Basic DART Structure: Asymmetry of Binding Domain

Basic DART (No Fc Domain)

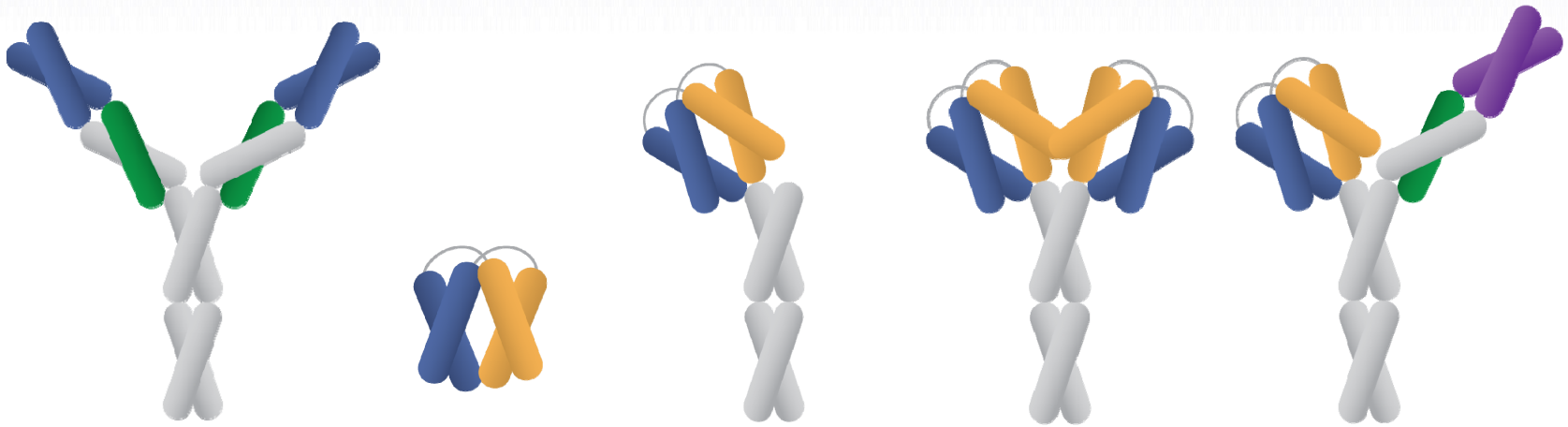


DART Crystal Structure (Pfizer[a])



(a) *Crystallography of Pfizer's P-Cadherin x CD3 DART molecule. The two antigen binding sites (shown by red dot circles) are separated from each other by approximately 30 Å and are facing away from each other at an angle of approximately 90°. Source: Root, et al.,* Antibodies *2016, 5, 6; March 4, 2016.*

Formats Tailored for Different Applications



Construct	mAb	DART	DART	DART	TRIDENT
Specificity	Monospecific	Bispecific	Bispecific	Bispecific	Tri-specific
Valency	Bivalent	Bivalent	Bivalent	Tetravalent	Trivalent
Half-life	Days to weeks	Hours	Days to weeks	Days to weeks	Days to weeks
Candidates	margetuximab, enoblituzumab, MGA012	MGD006	MGD007, MGD009, MGD010, MGD011 (duvortuxizumab), MGD014, PF-06671008	MGD013	Multiple programs in pre-clinical development

DART: Leading Bispecific Platform

Technology Originator (Platform)	Construct	# of Clinical Programs	Characteristics of Clinical Candidate(s)			
			Potential for IgG-like Half-life	Cis-binding Modality	Redirected T-cell Killing Modality	Monovalent Binding to CD3
MacroGenics (DART)	**Diabody**	**6**	✓	✓	✓	✓
Roche (CrossMAb)	Ig-Like	6	✓	✓	✓	✓
Genmab (DuoBody®)	Ig-Like	2	✓	✓	✗	–
Merus (Biclonics®)	Ig-Like	2	✓	✓	✓	✓
Trion (Triomab®)	Ig-Like	1	✓	✗	✓	✓
Oncomed (BiMab™)	Ig-Like	1	✓	✓	✗	–
Regeneron	Ig-Like	1	✓	✗	✓	✓
AbbVie (DVD-Ig™)	Dual-Ig	2	✓	✓	✗	–
Sanofi	Dual-Ig	1	✓	✓	✗	–
Amgen (BiTE®)	scFv-Based	6	✗	✗	✓	✓
Affimed (TandAb®)	scFv-Based	2	✗	✗	✓	✗
Eli Lilly	IgG + scFv	2	✓	✓	✗	–
Xencor	IgG + scFv	1	✓	✗	✓	✓
Aptevo (ADAPTIR®)	scFv-Based	1	✓	✗	✓	✗
Merrimack	IgG + scFv	1	✓	✓	✗	–
Immunocore (ImmTAC)	TCR + scFv	1	✗	✗	✓	✓
Ablynx (Nanobodies®)	Alt. Scaffold	1	✓	✓	✗	–

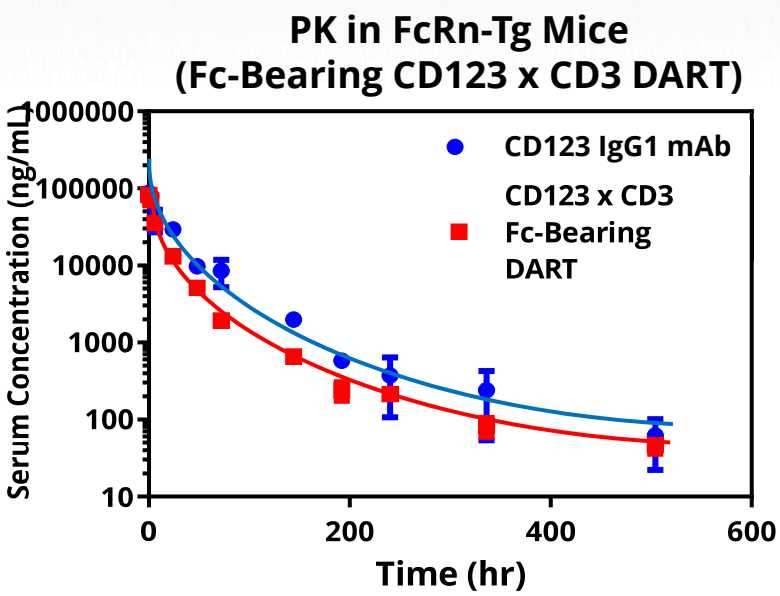
** Based on available public information as of December 5, 2016. Excludes in-licensed and inactive programs.*

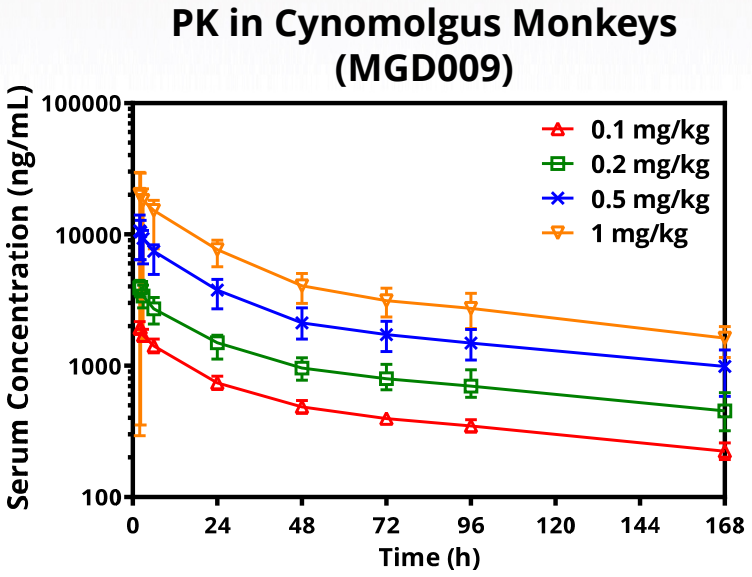
Key Attributes of Different Bispecific Platforms

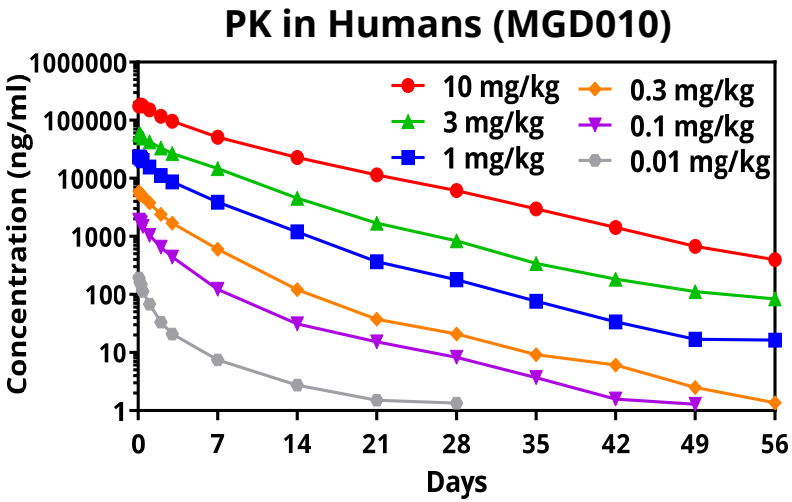
	DART	Ig-Like	scFv-based
Ease of Manufacturing	Standard mAb platform	More complex CMC	Unpredictable
Stability Against Aggregation	Stable	Stable	Unstable (domain exchange)
Valency Flexibility	Customizable (2x or 4x)	Fixed 2x	Feasible, but complex
Half-life Extension	Customizable (short to IgG-like)	IgG-like	Feasible, but complex
High Potency			
Tri-specific Variant	TRIDENT (fully customizable)	Unavailable	Feasible, but complex
"Plug-and-play" w/ Virtually Any Fv Pair	Highly predictable	Less predictable (for some)	Unpredictable

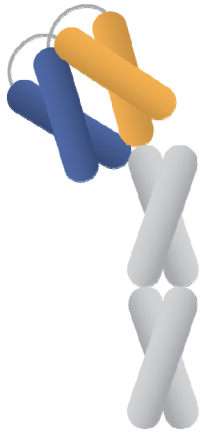
More Favorable Less Favorable

Fc Domain Extends DART Half-Life









Established Platform for DART Manufacturing

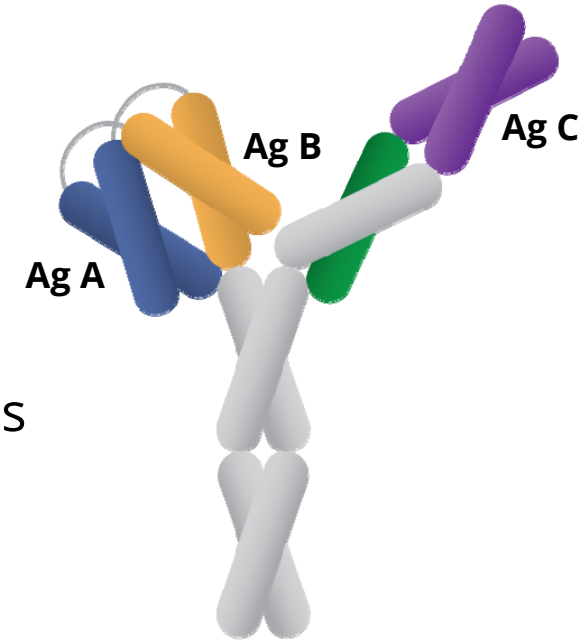
16 GMP lots across 7 distinct molecules at MacroGenics

- 500 Liter single-use bioreactors
  - Commercially available media and feeds
  - Scalable performance from Development (2L, 10L, 50L) to GMP (500L)
- Standard downstream unit operations
- Current titer range = 1.3 – 3.7 g/L
- Drug Product typically stored as liquid at 2-8 °C
  - Earliest lots > three years old
- Successful tech transfer to multiple pharma partners

TRIDENT Platform: Extending Multispecific Capability

Customizable trispecific platform

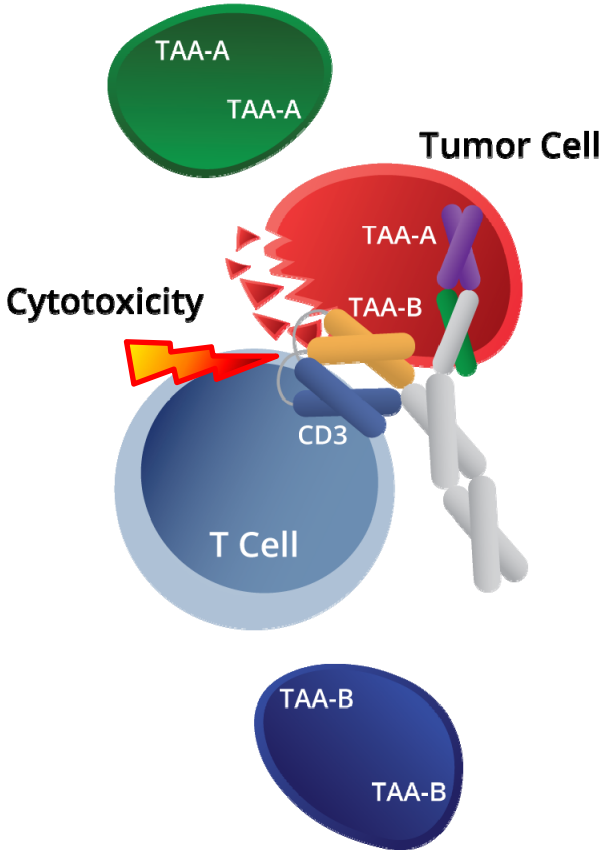
- Recognition of two or three separate antigens
- Ability to independently control valency of individual targets
- Enhanced potency and/or selectivity via bi-epitopic target recognition plus effector arm
- Selective recruitment of subsets of effector cells



TRIDENT™
MW: ~ 155 kDa

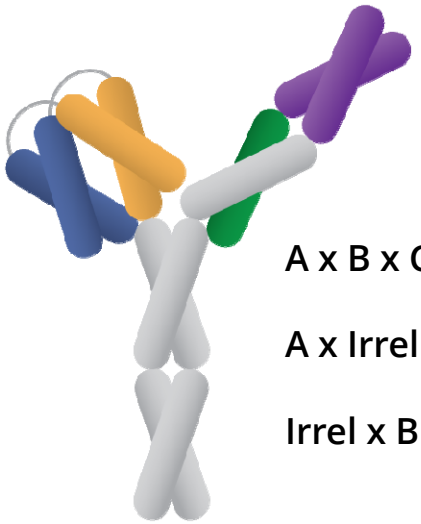
Dual-Antigen Recognition for Increased Tumor Selectivity

Dual Cancer Antigen Targeting



Concept: Targeting of two cancer antigens co-expressed on tumor cells (e.g., Targets A and B) but displaying mutually exclusive normal tissue expression provides opportunity to increase therapeutic window

Validation Approach: TRIDENT molecules incorporating single or dual cancer antigen specificity and an anti-CD3 targeting arm were evaluated for relative efficacy in redirected T-cell killing



A x B x CD3

A x Irrel x CD3

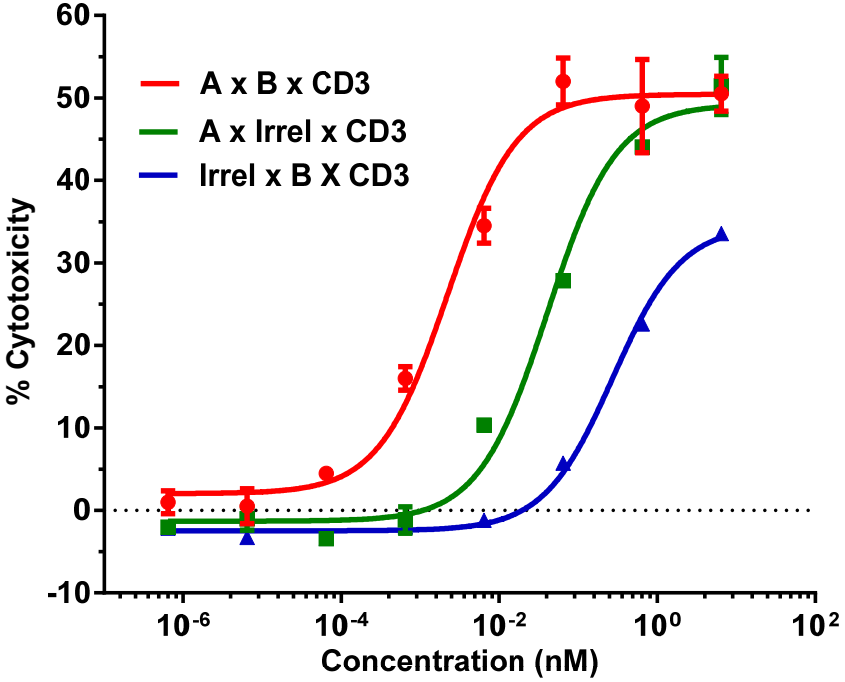
Irrel x B x CD3

TRIDENT molecules generated for biological characterization

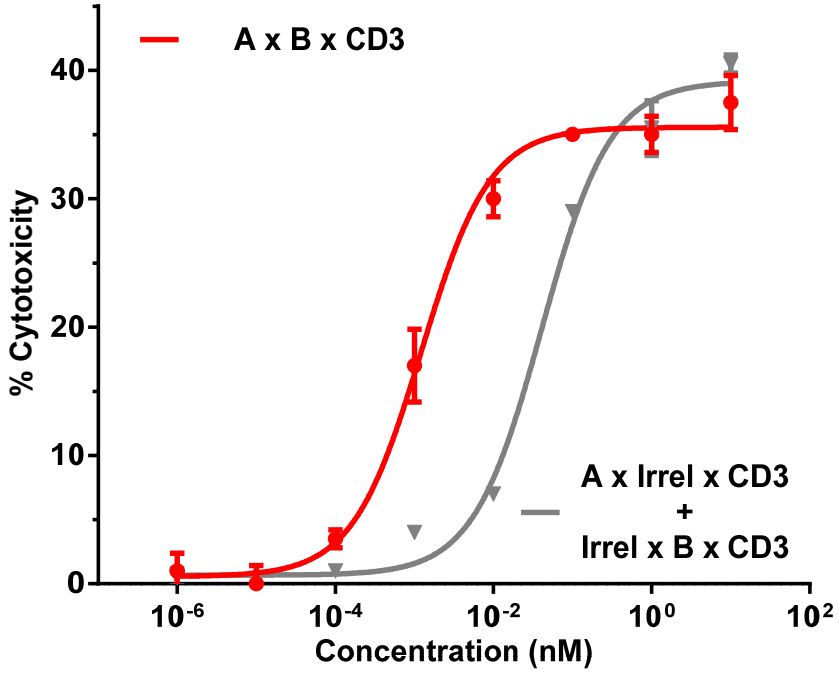
TAA = Tumor-associated antigen

TRIDENT-Enhanced CTL Activity via Dual Cancer Ag Recognition

Lysis of Target Cells Expressing A & B Antigens



>10x increase in potency with dual-antigen targeting TRIDENT



Two single-specificity TRIDENT molecules cannot reconstitute potency of A x B x CD3 TRIDENT

Effectors = T-cells; E:T = 10:1 or 1:1; Cytotoxicty based on LDH Release Assay

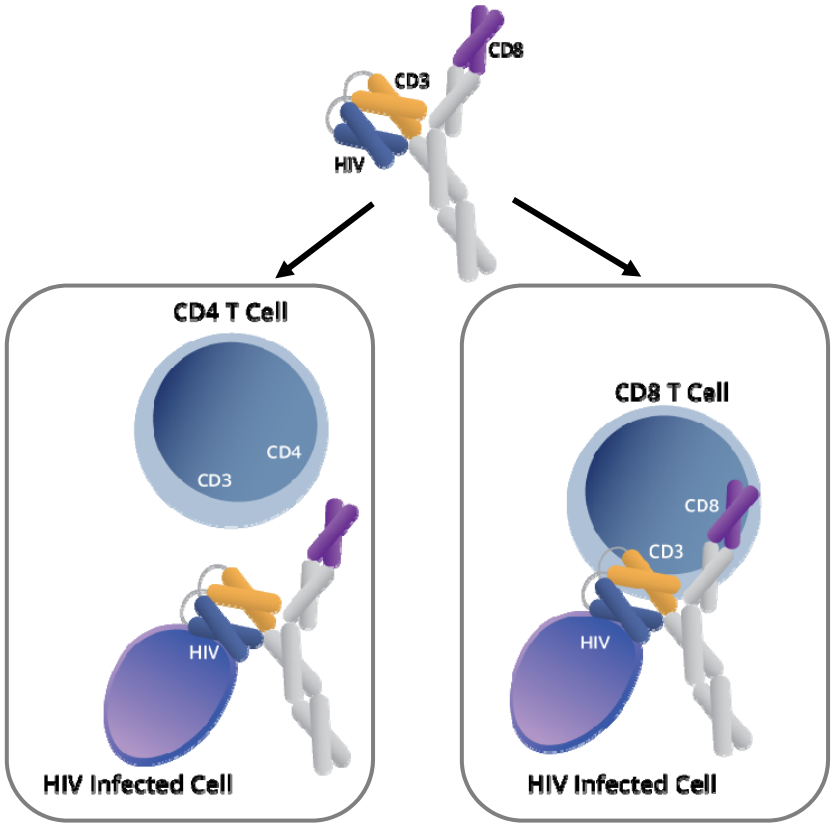
Preferential Engagement of CD8 Killer T Cells

Hypothesis:

- Preferential recruitment and activation of CD8 T cells to enhance potency of redirected killing and reduce cytokine release

Approach:

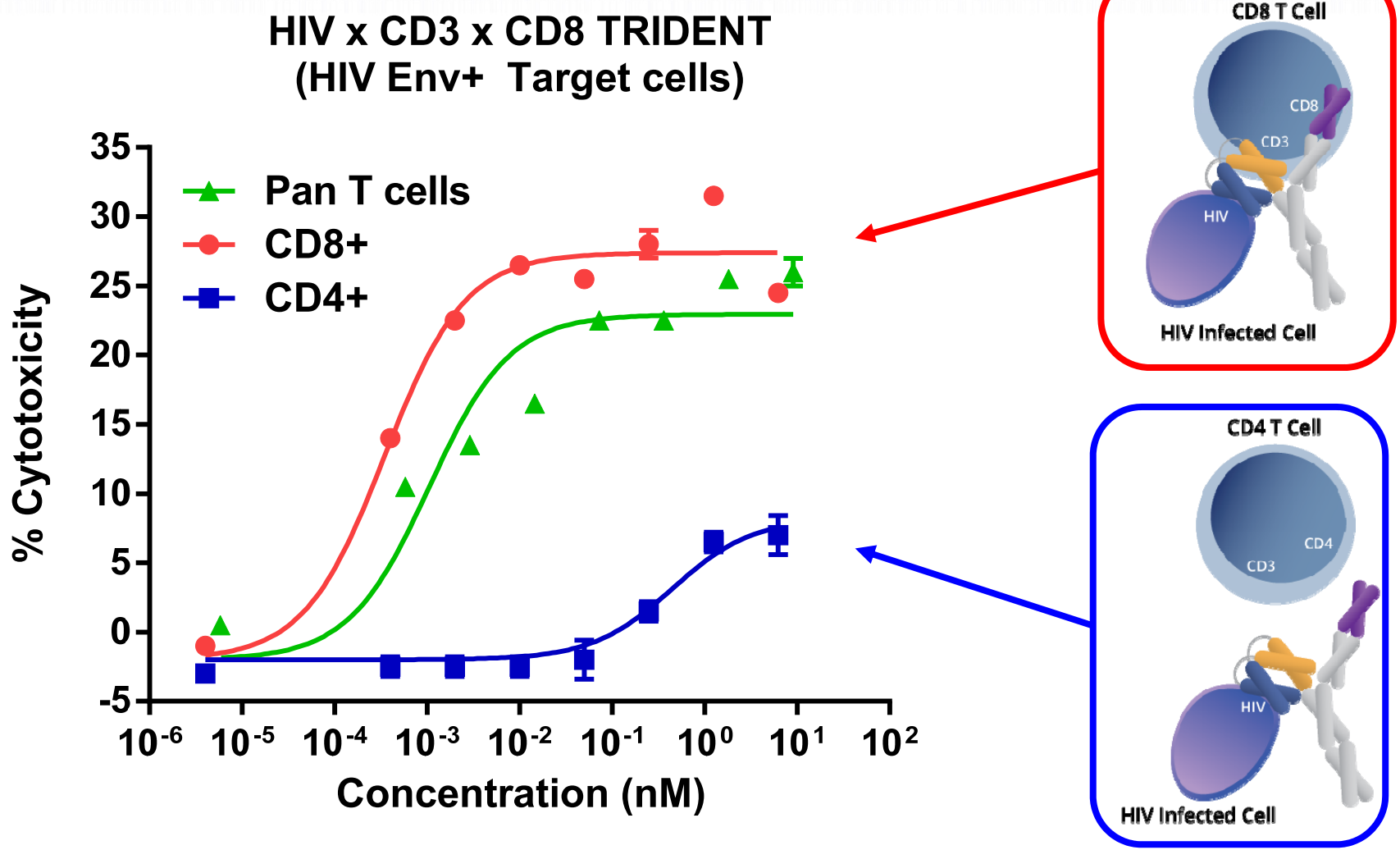
- Combine anti-CD8 with anti-CD3 and HIV-targeting arm in same molecule
- Evaluate TRIDENT vs. DART activity in killing assay



Minimal T-cell Activation

Optimal T-cell Activation & Cell Killing

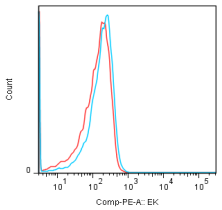
HIV x CD3 x CD8 TRIDENT: Enhanced CD8-mediated Killing



TAA x CD3 x CD8 TRIDENT Binds CD8 Killer T Cells In Vivo

Flow cytometry analysis of TRIDENT-treated cynomolgus monkeys

CD4 T-Cells



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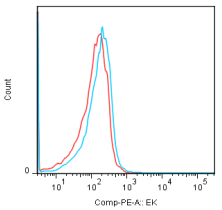
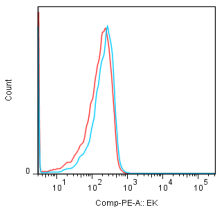


24H

CD8 T-Cells



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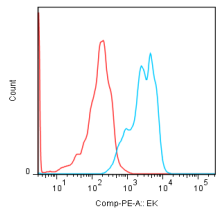
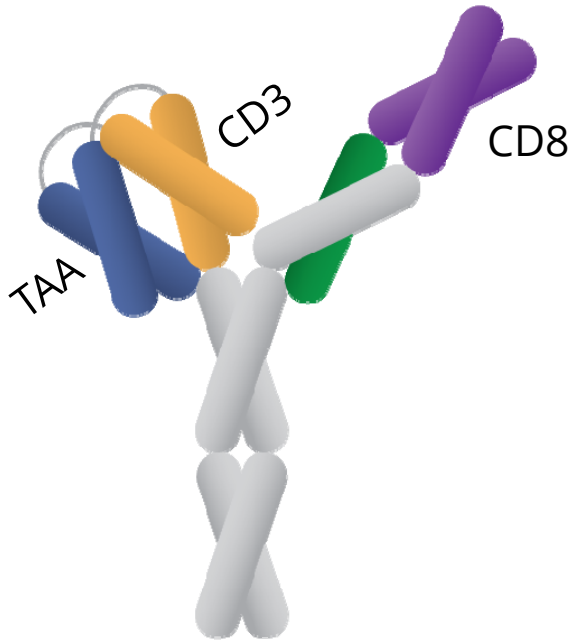


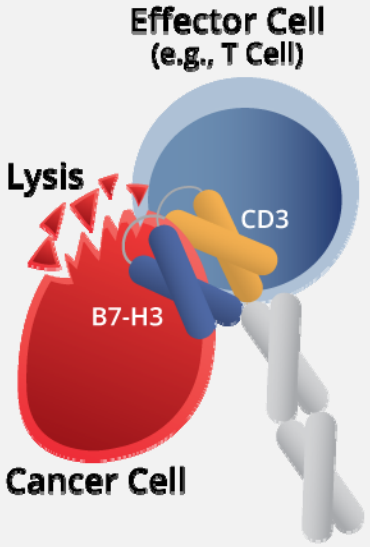
24H



DART & TRIDENT: Designed for Broad Range of Modalities

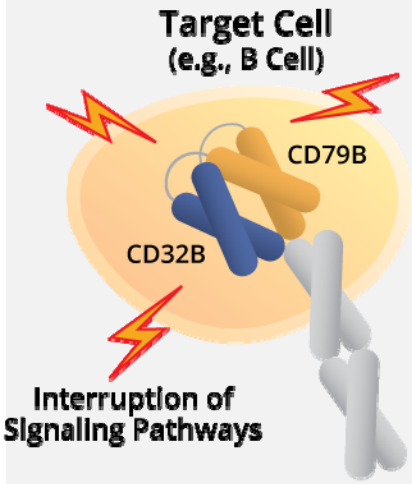
Significant advantage over other multi-specific technologies

Redirected T-cell Activation and Killing



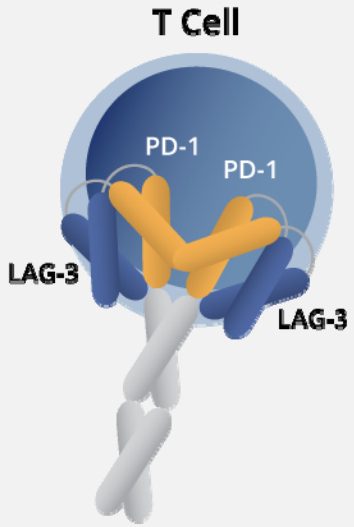
Product Candidates:
MGD006 (CD123 x CD3)
MGD007 (gpA33 x CD3)
MGD011 (CD19 x CD3)
MGD009 (B7-H3 x CD3)
MGD014 (HIV x CD3)

Modulation of Receptor Signaling



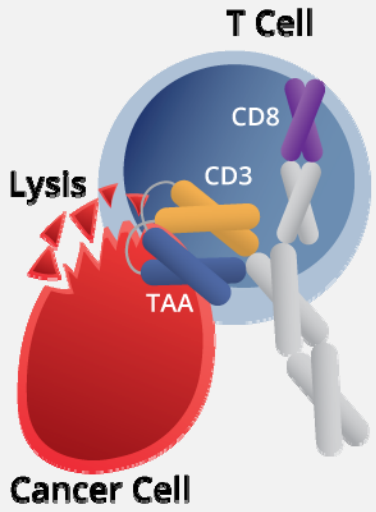
Product Candidate:
MGD010 (CD32B x CD79B)

Simultaneous Targeting of Multiple Checkpoint Inhibitors



Product Candidate:
MGD013 (PD-1 x LAG-3)

Redirected T-cell Activation and Killing (TRIDENT)



Product Candidate:
TBD
(TAA x CD3 x CD8)

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Six DART Molecules in Phase 1 Development

Product Candidate	MGD006	MGD007	MGD011	MGD009	PF-06671008	MGD010
Targets	CD123 x CD3 *(S80880)*	gpA33 x CD3	CD19 x CD3 *(duvortuxizumab)*	B7-H3 x CD3	P-cadh. x CD3	CD32B x CD79B
MoA	Redirected T-Cell Killing					Signal Modulation
Current Dosing	Continuous IV	q3W	q2W	q2W	Undisclosed	Expected weekly or longer
Indications	AML, MDS	Colorectal cancer	B-cell heme malignancies	Solid tumors	Solid tumors	Autoimmune disorders
Our Commercial Rights	North America, Japan, Korea, India	North America, Japan, Korea, India	U.S. Co-promote	Worldwide	Royalties/ Milestones	Worldwide
Partner	Servier	Servier *(Option)*	Janssen	—	Pfizer	—
Data Pres.	ASH 2013, *Sci Transl Med* 2015	AACR 2014	ASH 2014	Keystone Symposia 2016	AACR 2015	AAI 2014, EULAR 2016

CD3-Directed DART Molecules – Learnings to Date

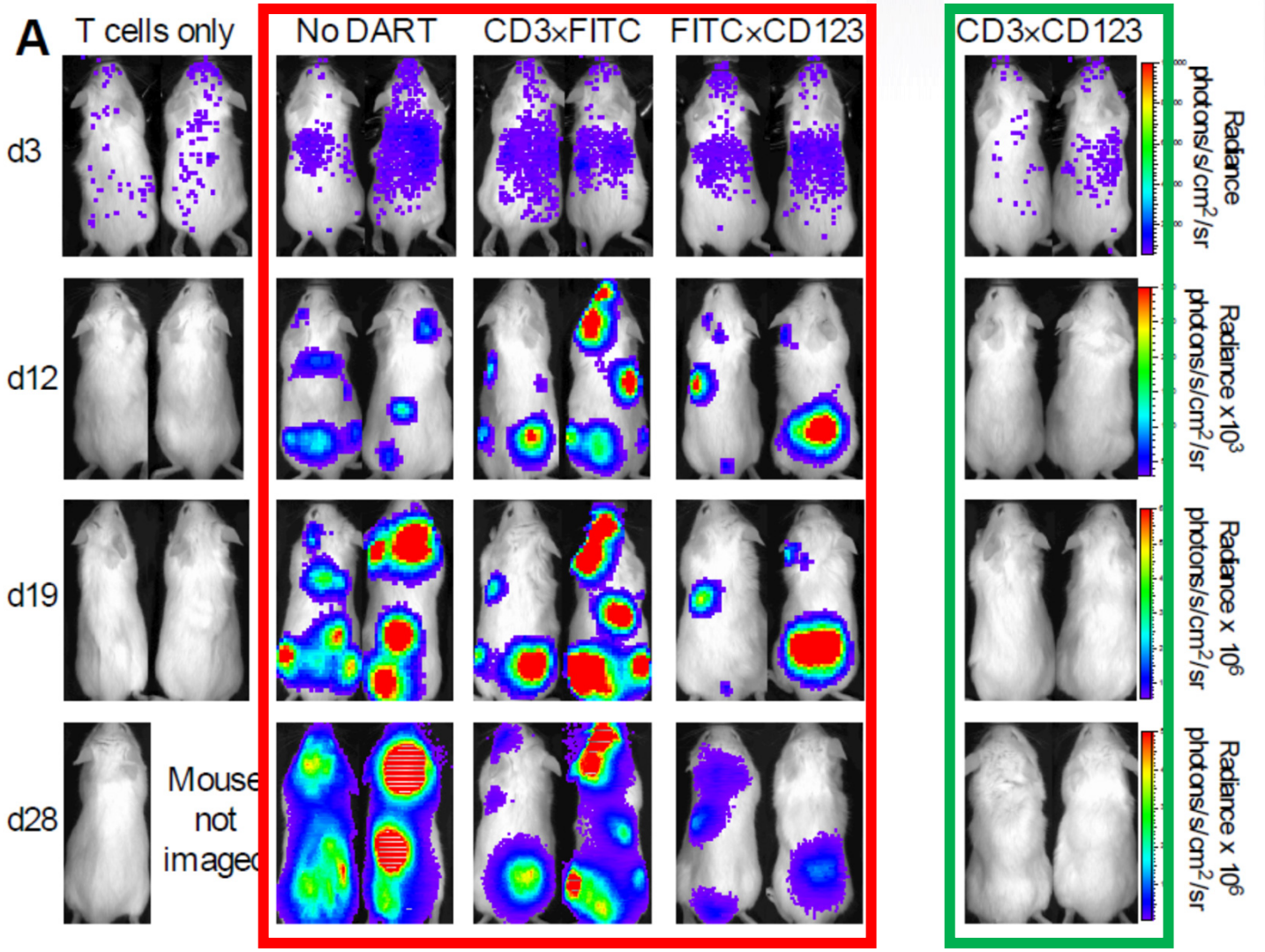
- On-target engagement
- Significant potency and low-dosing requirement (vs. mAb)
- Manageable safety/tolerability (i.e., Cytokine Release Syndrome)
- Preliminary evidence of biological and clinical activity
- Rationale for combination with anti-PD-1

MGD006 Clinical Update

MGD006: CD123 x CD3 DART

Candidate	• Humanized CD123 x CD3 DART 
Function/MoA	• Redirected T-cell killing against targeted leukemia cells – Elimination of leukemic stem cells – Sparing of normal hematopoietic stem cells – Capable of engaging any T-cell without HLA-restriction • Potent in vivo preclinical activity in preclinical models • Extremely low clinical dosing (ng/kg)
Indications	• Lead: acute myeloid leukemia (AML) and myelodysplastic syndrome (MDS) • Other hematologic neoplasms including B-cell ALL
Development	• Phase 1 study ongoing in US and EU (dose escalation)
Partner	• MacroGenics retains full rights in No. Amer., Japan, Korea & India • Servier has rights for all other territories

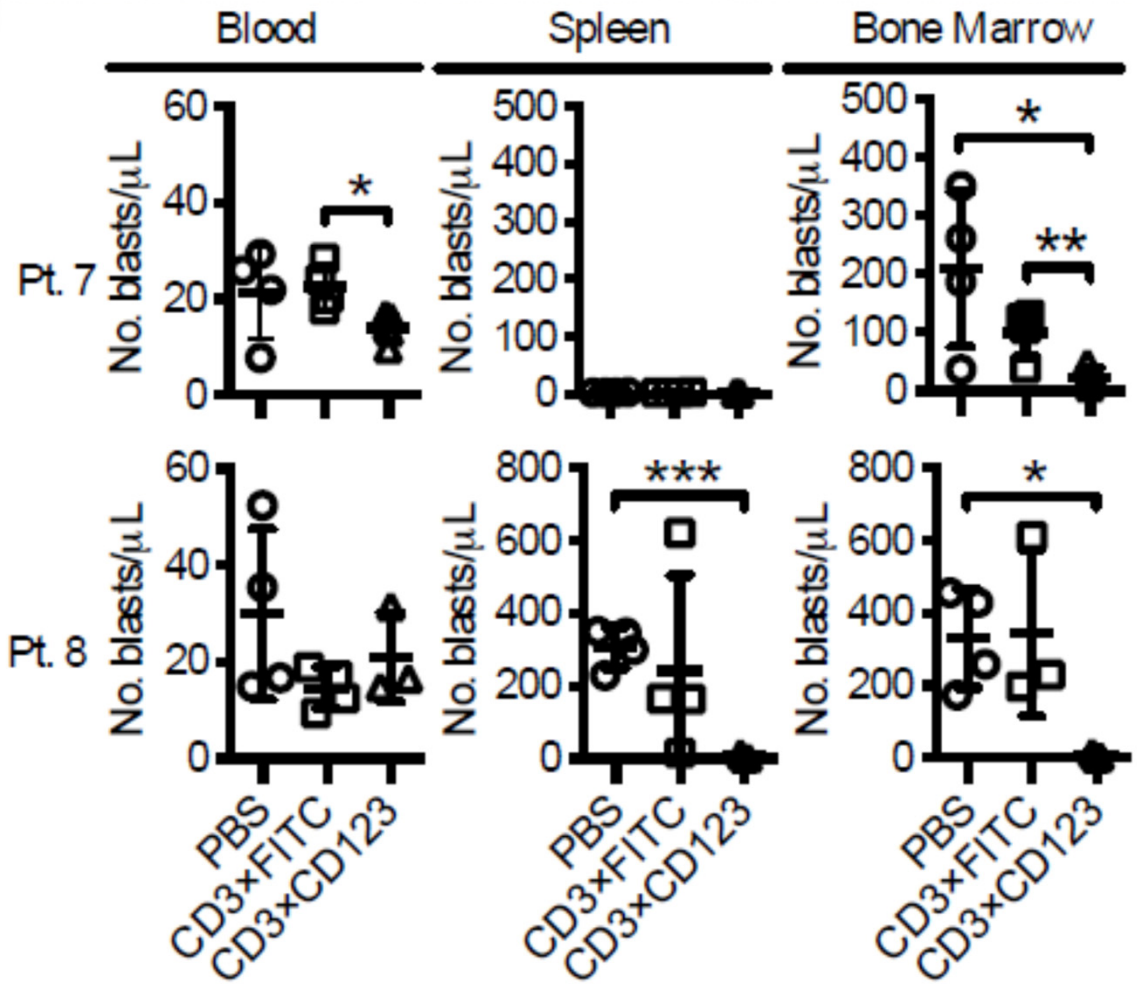
MGD006 Suppresses CD123+ Leukemia Xenografts



Irradiated NSG mice (n=5/group) injected with K562GFP-CD123 cells and treated with DARTs. Bioluminescence imaging on days 3, 12, 19, and 28 showed significant inhibition ($p<0.0001$) of tumor growth in CD3×CD123 DART; *Al-Hussaini, et al. Blood 2016,127:122-31*

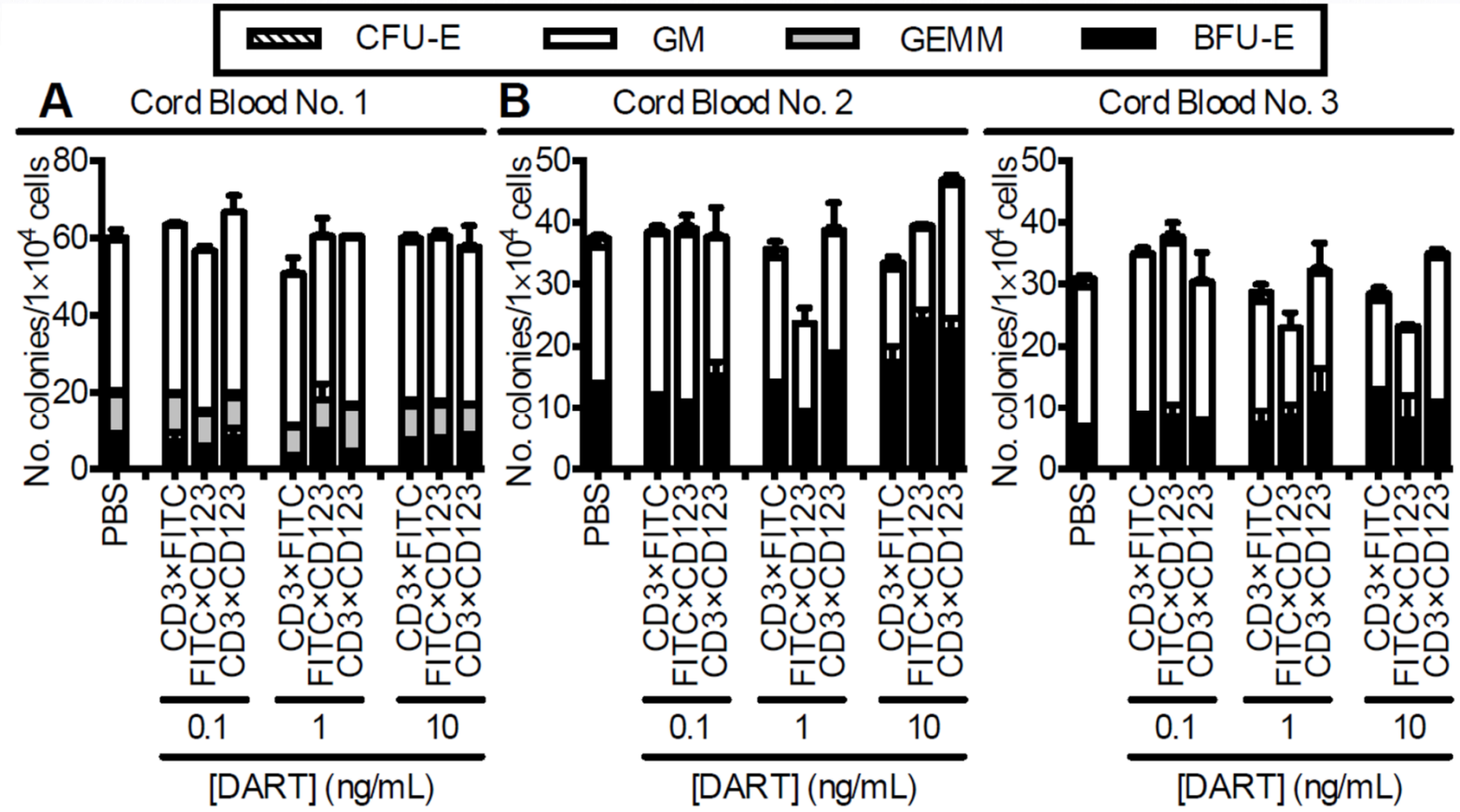
MGD006 Suppresses Leukemia Patient Xenografts

NSG mice reconstituted with human AML cells



Al-Hussaini et al. Blood 2016,127:122-31

MGD006 Shows Minimal/No Impact on Bone Marrow Precursors



- Cord blood cells from 3 healthy donors, incubated with agents for (A) 4 hours or (B) 18 hours
- Plated in methylcellulose-based medium; colonies were scored on day 7; bars = SD of duplicate plates

Al-Hussaini et al. Blood 2016,127:122-31

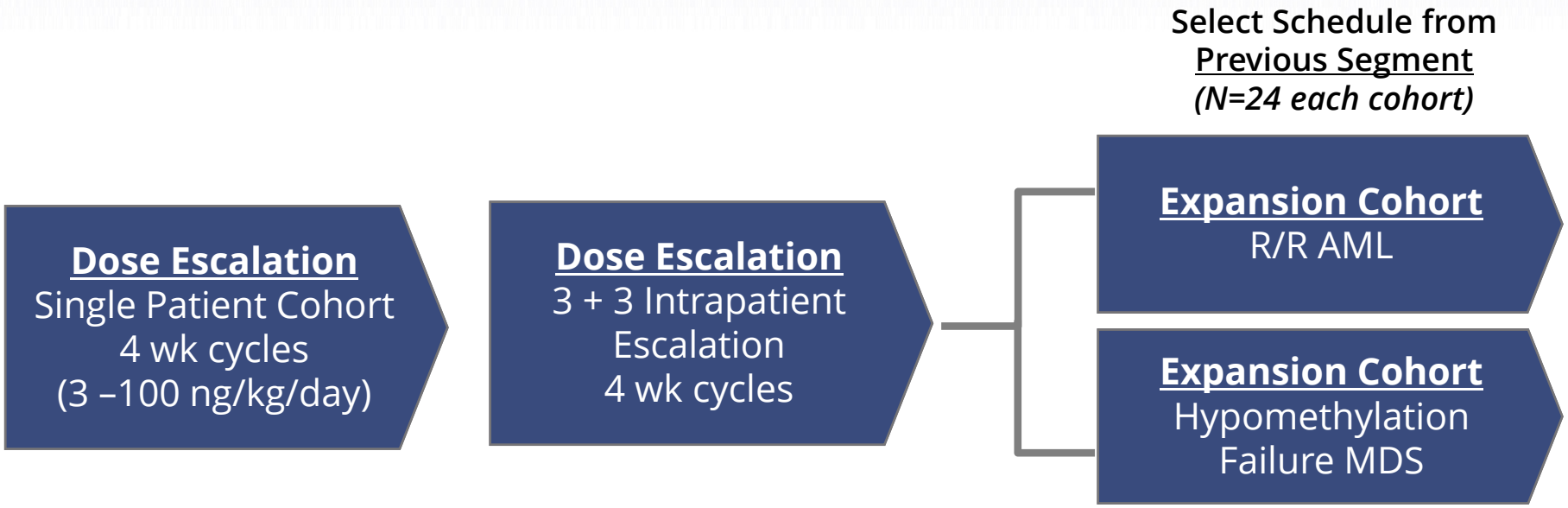
AML Treatment Overview

- Acute myeloid leukemia (AML): heterogeneous group of diseases with neoplastic infiltration of blasts in blood, bone marrow, viscera
  - 20,000 new cases in US (2016)
  - Incidence increases with age; median age at onset is 67 years
  - Overall 5-year survival rate is 26.6%
  - Untreated AML patients succumb within weeks
- Current therapy is suboptimal:
  - Standard therapy comprises “7+3” induction chemotherapy (cytarabine and daunorubicin) followed by consolidation chemotherapy +/- transplantation
  - Limited by toxicity and high rates of relapse
  - Salvage therapies compromised by short response duration & high relapse rate
- Large unmet need given toxicity and high rates of relapse with standard therapy

Key Goals for MGD006 FIH Study

- Define safety and preliminary clinical activity in patients with AML and MDS
- Optimize approach to delivery and supportive care
- Define consistent supportive care regimen to manage CRS and minimize corticosteroid use where possible to limit systemic immune suppression
- Define PK, PD and PK/PD relationships
- Position MGD006 for testing in other CD123+ therapeutic opportunities including ALL patients post-blinatumomab or post-CD19 CART cell therapy
- Set stage for follow-on mechanism-based combination studies with internal and/or external assets

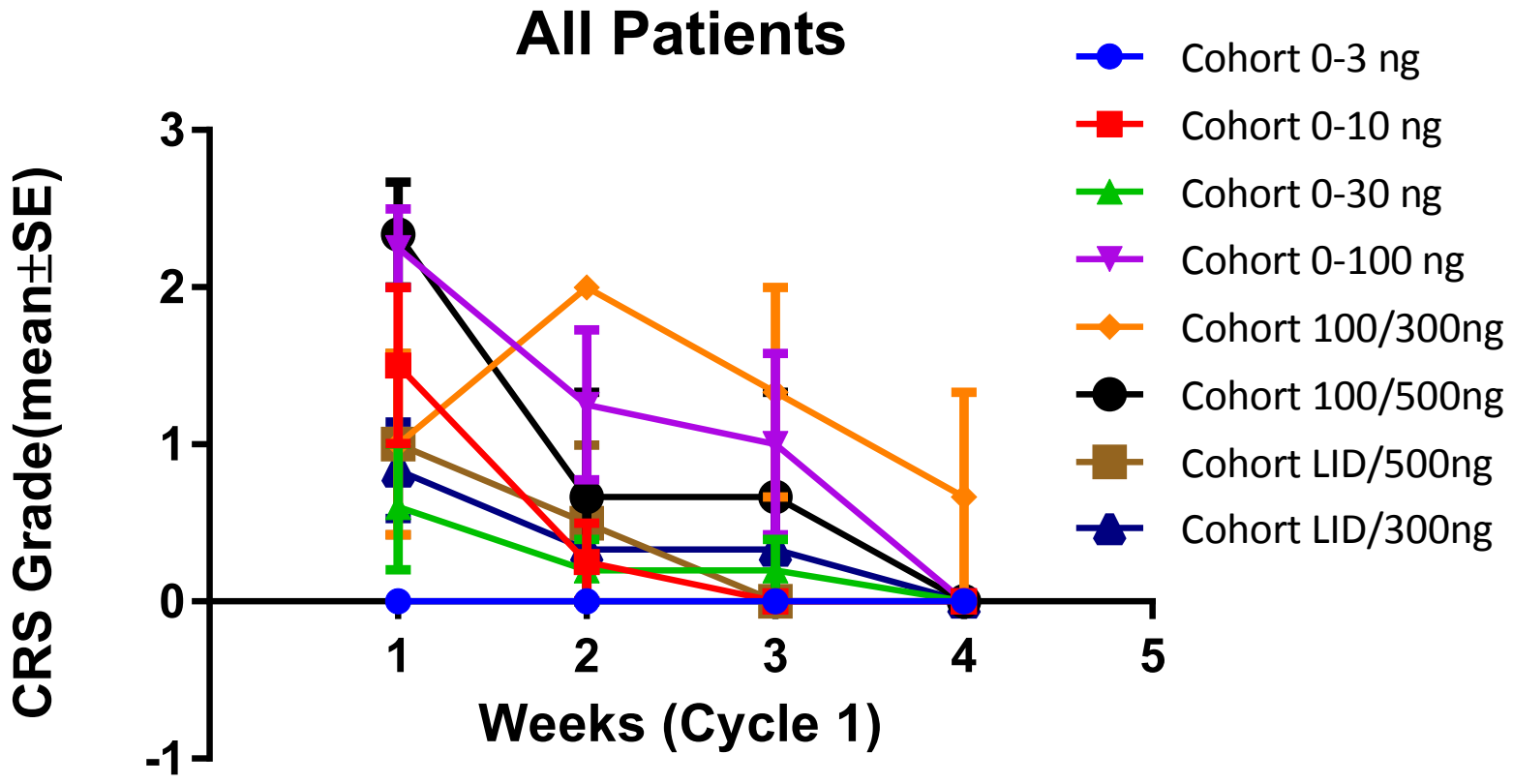
MGD006 (CD123 x CD3): Phase 1 Study in Dose Escalation



Patient Population: Patients with relapsed/refractory AML and hypomethylation failure Int-2/High Risk MDS

Dosing Regimen: Continuous intravenous infusion

Overview of CRS Across Dosing Cohorts to Date



- IRR/CRS is primary toxicity of significance
- Manifestations include fever, chills, hypotension, tachycardia

** LID, lead-in dose*

MGD006: Evolution of CRS Management

Original Protocol: No Prophylaxis/ Early Intervene

Anti-Cytokine Gr 4 CRS Only

Prophylactic Premeds (incl. steroid)

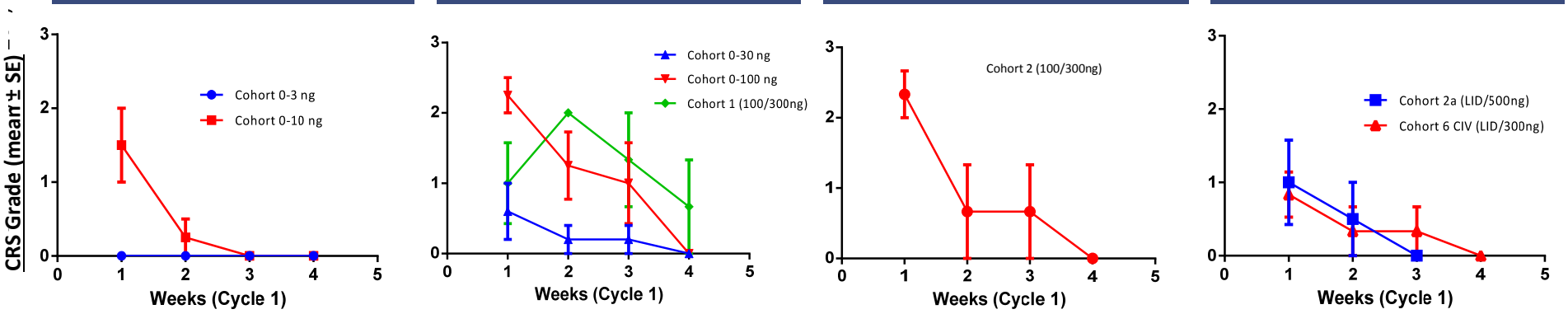
Early Anti-Cytokine (for Gr 2 CRS)

Two-Step Dose (LID) Escalation & New CRS Grading (Lee, et al. criteria)

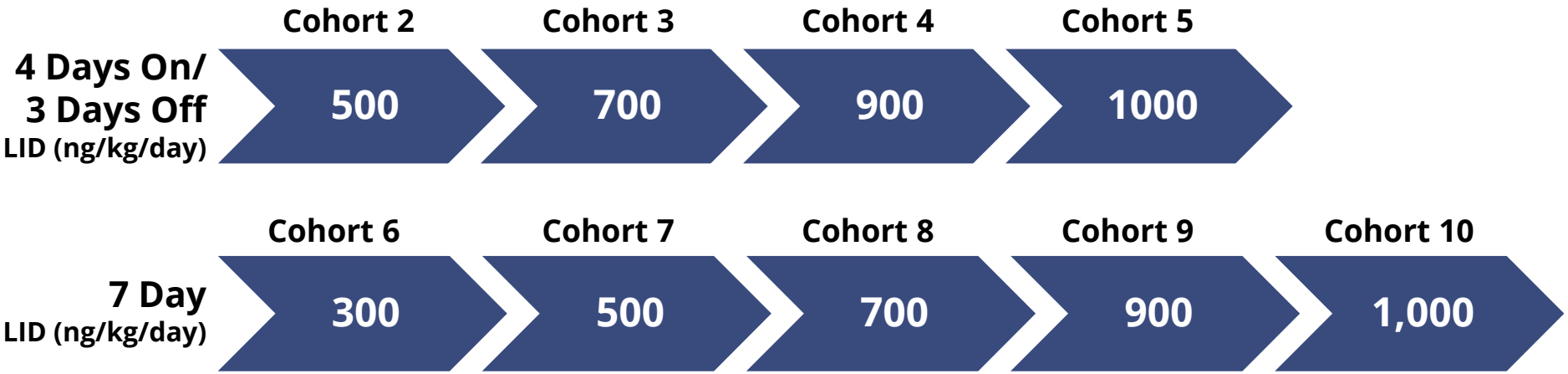
Single Patient Dose Escalation

Multi-Patient Dose Escalation

No Prophylaxis	Prophylactic Dexamethasone	Prophylactic Dexamethasone +Anti-Cytokine	Prophylactic Dexamethasone +Anti-Cyto +2-Step Escal.

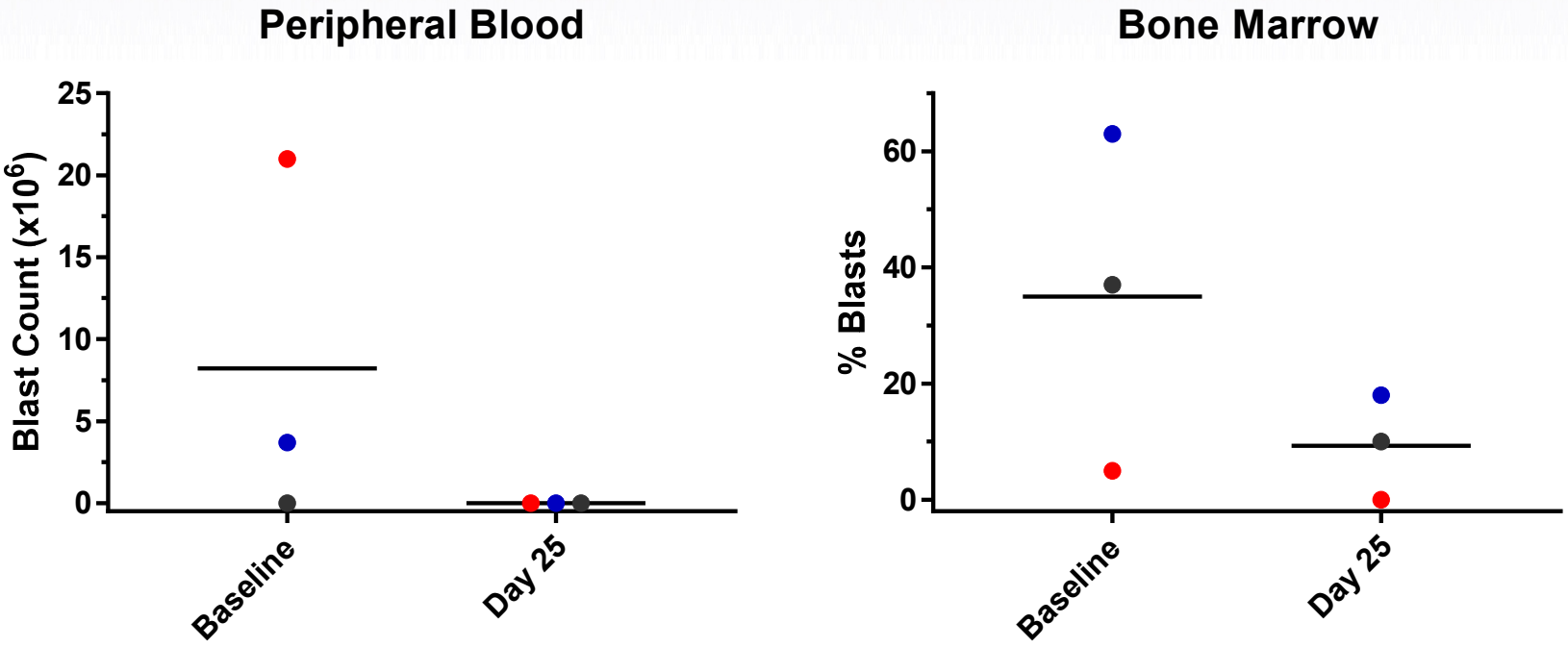


Current Two-Step Approach to Dose Escalation



- Lead-in-Dose (LID): 30 ng/kg/day x 3 days, 100 ng/kg/day x 4 days week 1, then:
  - Cohorts 2-5: Single Step-Up, 4-on 3-off schedule, Cycle 1+
  - Cohorts 6-10: Single Step-up for 21 days continuous infusion (Cycle 1) followed by 4-on 3-off schedule for Cycle 2 and beyond

MGD006: Decreased Blast Counts in 3 Patients



Patient ID	Diagnosis	Cohort	Relevant Hx
1	AML M0	30ng/kg/d	Refractory >2 induction Tx
2	AML M2	100ng/kg/d	PR duration <6 months Refractory to 2 salvage attempts
3	AML *FLT3* mutated	100/500ng/kg/d	CR duration <6 months

MGD006: What Have We Learned?

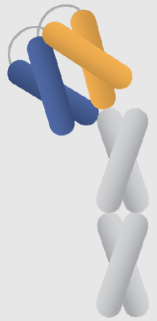
- Defined safety profile at doses tested to date in 30 patients
- Refined supportive care to enable substantial limitation of CRS
- Positioned asset to enable outpatient administration after Cycle 1
- Characterized predictable PK properties
- Established biological and preliminary clinical activity in AML patients
- Mechanism-based rationale for combining MGD006 with anti-PD-1

MGD006: Next Steps

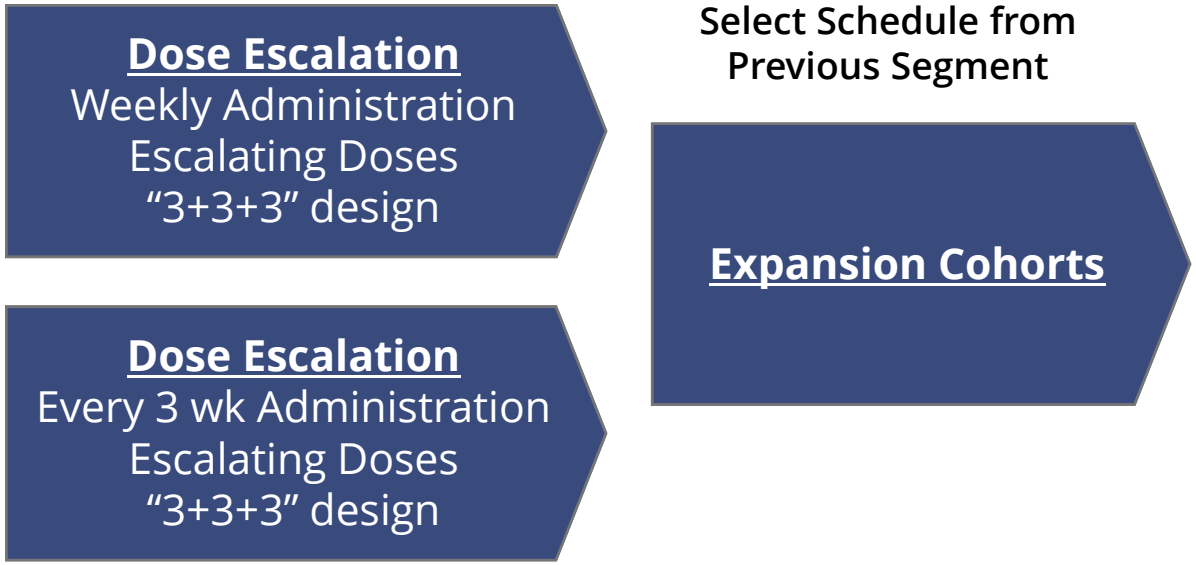
- Continue aggressive dose escalation given substantial progress in CRS control
  - Early anti-cytokine therapy and two-step intra-patient dose escalation
- Execution of cohort expansions in AML and MDS→ positioned for acceleration guided by strength of efficacy signal
- Assess opportunity for clinical testing of combination with anti-PD-1
- Characterize potential role of MGD006 in treating/preventing antigen-loss relapse in ALL patients post CD19-CAR-T cells or blinatumomab
- Longer half-life version of MGD006 now established

MGD007 Clinical Update

MGD007: gpA33 x CD3 DART Molecule

Candidate	• Humanized, Fc-bearing gpA33 x CD3 DART 
Rationale	• gpA33 is homogenously expressed on >95% colorectal cancer - Expressed on cancer stem cells and differentiated cell populations - Normal tissue expression primarily restricted to intestinal epithelium
Function/MoA	• Redirected T-cell killing against cancer cells
Indications	• Lead: Colorectal cancer • Other: Pancreatic and gastric cancers
Development	• Phase 1 study ongoing
Partner	• MacroGenics retains full rights in No. Amer., Japan, Korea & India • Servier has option for all other territories

MGD007 (gpA33 x CD3): Phase 1 Study in Dose Escalation



Objectives: Establish safety, determine MTD, characterize PK, evaluate alternate schedules, and describe early evidence of anti-tumors activity

Patient Population: Patients with relapsed / refractory metastatic colorectal carcinoma

Dosing Regimen: Intravenous infusion, qW or q3W

Evaluations: RECIST and irRECIST

MGD007 Related Adverse Events

Related Adverse Events* †	N = 30		Related Adverse Events* †	N = 30	
	All	≥ Gr 3		All	≥ Gr 3
Diarrhea	27 (90.0)	7 (23.3)	Dizziness	4 (13.3)	-
Nausea	23 (76.7)	6 (20.0)	Lymphopenia	4 (13.3)	3 (10.0)
Vomiting	22 (73.3)	6 (20.0)	Lipase Increased	4 (13.3)	1 (3.3)
Pyrexia	12 (40.0)	-	Dyspepsia	3 (10.0)	-
Abdominal Pain	11 (36.7)	2 (6.7)	Hyperglycemia	3 (10.0)	-
Fatigue	11 (36.7)	3 (10.0)	Hyponatremia	3 (10.0)	1 (3.3)
Chills	9 (30.0)	-	Hypophosphotemia	3 (10.0)	3 (10.0)
Dehydration	7 (23.3)	1 (3.3)	Cytokine Release Syndrome	3 (10.0)	1 (3.3)
Tachycardia	7 (23.3)	1 (3.3)	Influenza like illness	3 (10.0)	-
Decreased Appetite	6 (20.0)	-	Headache	3 (10.0)	-
Hypocalcemia	4 (13.3)	1 (3.3)	Myalgia	3 (10.0)	-
Lymphocyte count decreased	4 (13.3)	2 (6.7)	Leukopenia	3 (10.0)	2 (6.7)

** MGD007 related adverse event ≥ 10% of patients as assessed by NCI CTCAE v4.03*
† MedDRA Preferred Term

MGD007: Evolution of Supportive Care

Base protocol w/ aggressive intervention,
but without premedication

↓

Supportive care with scheduled premeds: acetaminophen,
antihistamines, scheduled steroid 24-48 hours

↓

Prophylactic oral non-absorbable steroid (budesonide);
Local immunosuppression in GI tract;
Limit potential for systemic immune suppression

↓

Early anti-cytokine for selected Grade 2 GI toxicity;
→Hypothesize local effect on cytokines in GI tract
→Limit "off-tumor," "on-target" toxicity

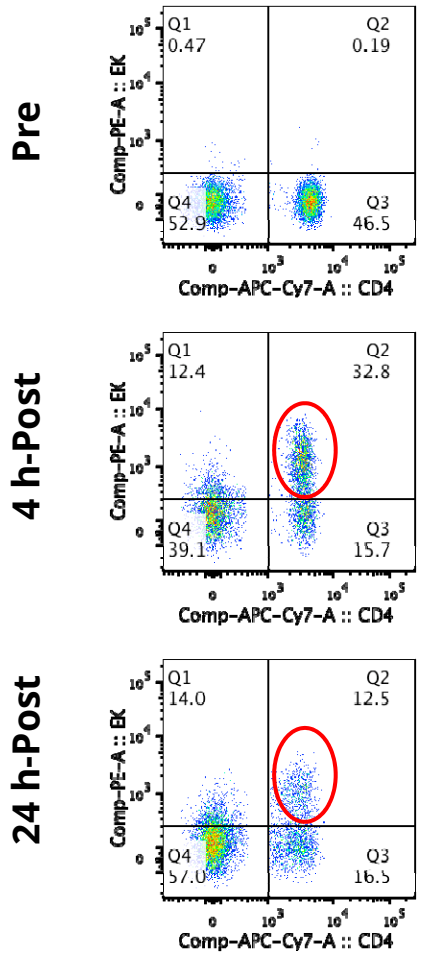
↓

Focus on q3W dosing regimen, escalate 1→3 μg/kg

MGD007 Binds Circulating T Cells

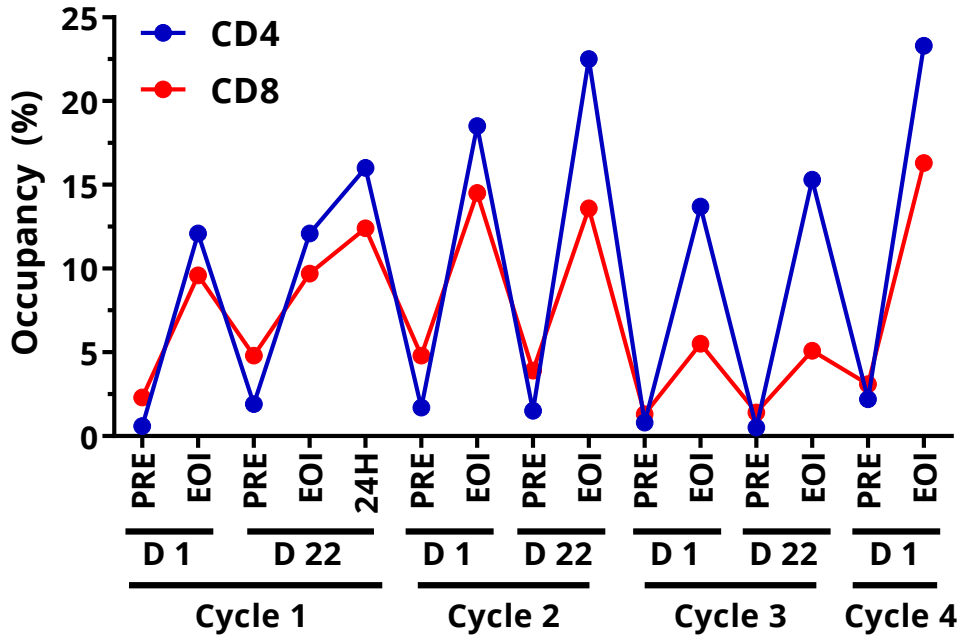
Flow cytometry demonstrates consistent T-cell loading across multiple cycles

MGD007 Binding to CD4 Cells



MGD007, 0.6 µg/kg patient

T-cell Occupancy

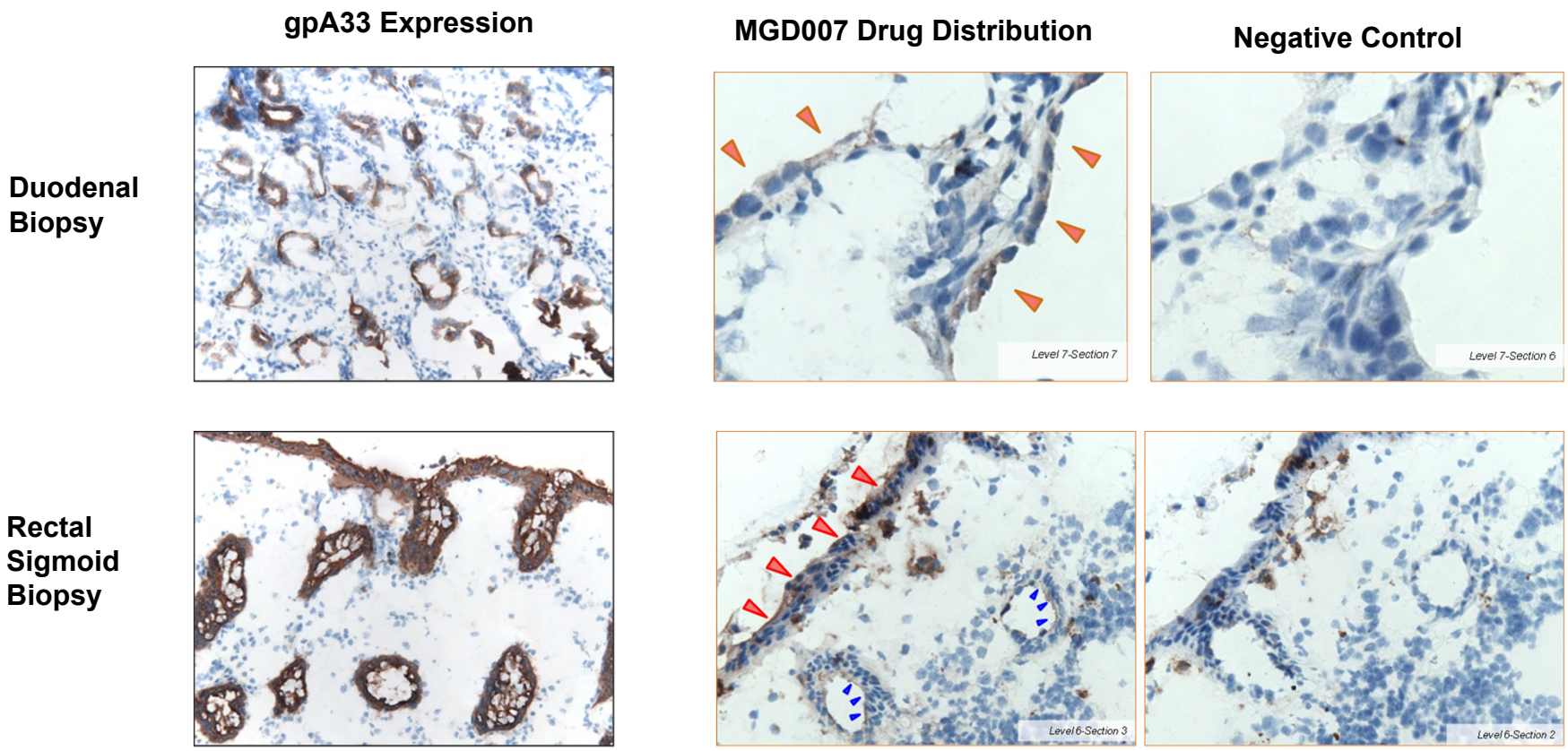


Pre = Pre-infusion; EOI = End of infusion (2h); 24h = 24h from EOI

MGD007, 1 µg/kg, representative patient
1 Cycle = 6 weeks

MGD007 Drug Distribution: gpA33 Target Cell Binding

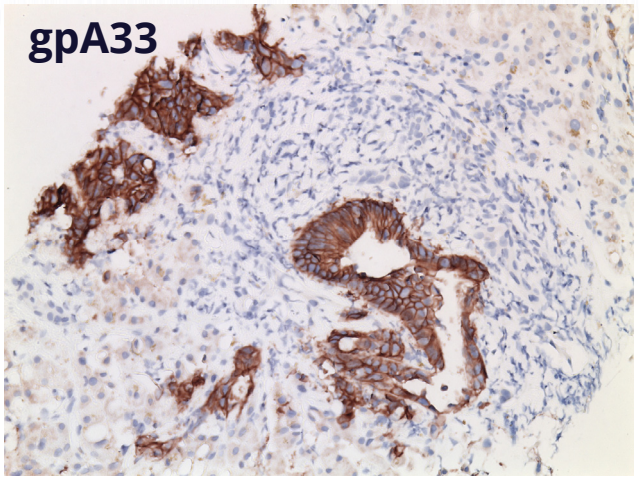
Evidence of MGD007 binding to upper & lower intestinal epithelium (3 µg/kg)

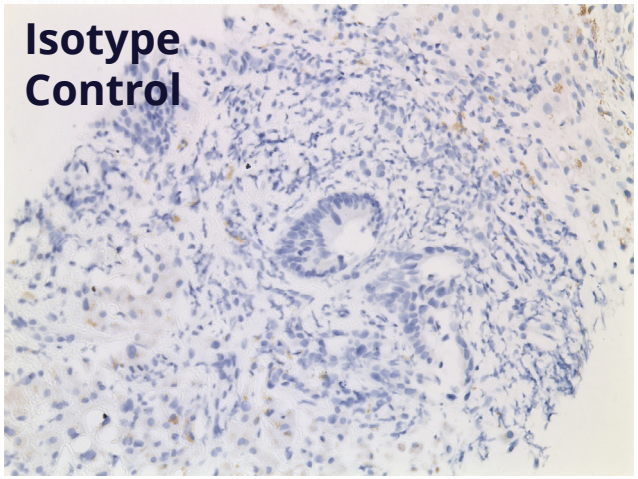


gpA33, CD3 and PD-1 Expression in CRC Patient Tumor Biopsy

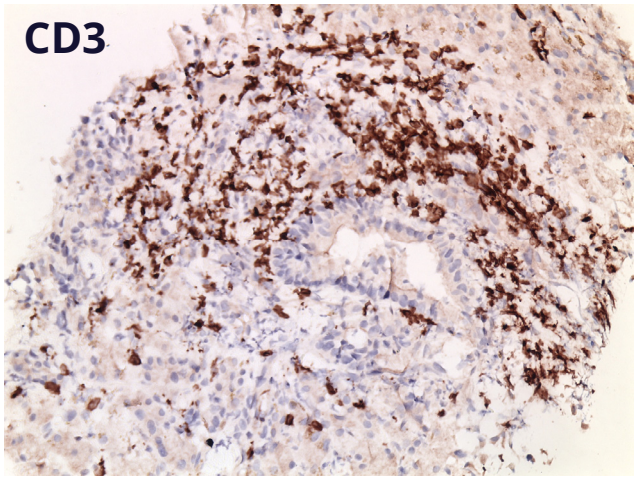
Liver biopsy obtained 24 hours post MGD007 dosing (1 µg/kg)

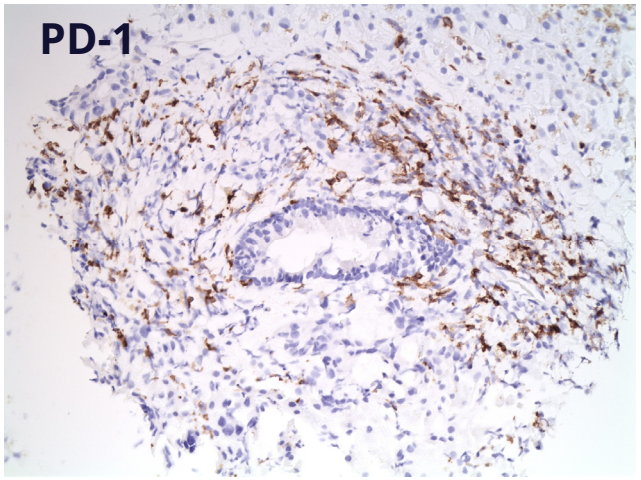
gpA33 expression on colorectal cancer liver metastasis





Sheet of CD3+ T cells surrounding gpA33+ve tumor cells in colorectal cancer liver metastasis (subset expressing PD-1)



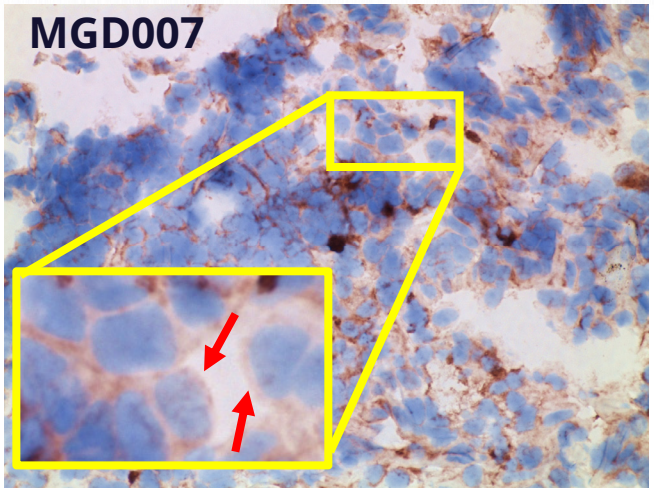


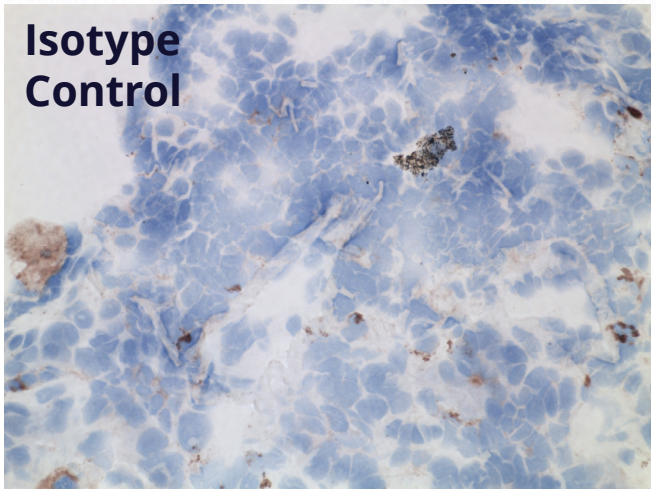
FFPE analyses; objective: 20x

Drug On Target in Tumor Cells (via Biopsy)

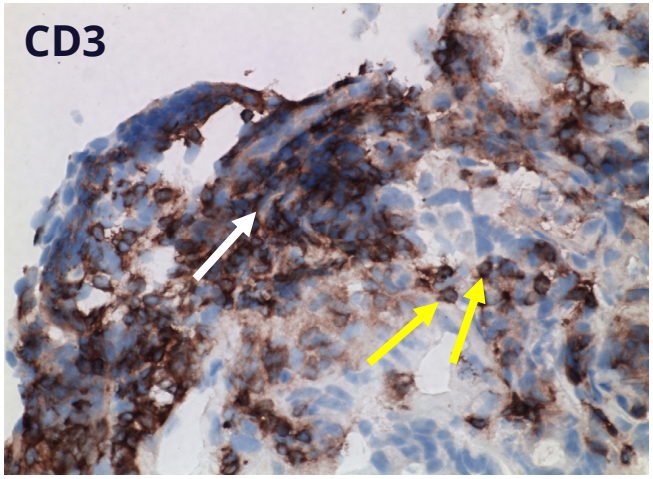
Lung biopsy obtained 48 hours post MGD007 dosing (1 µg/kg)

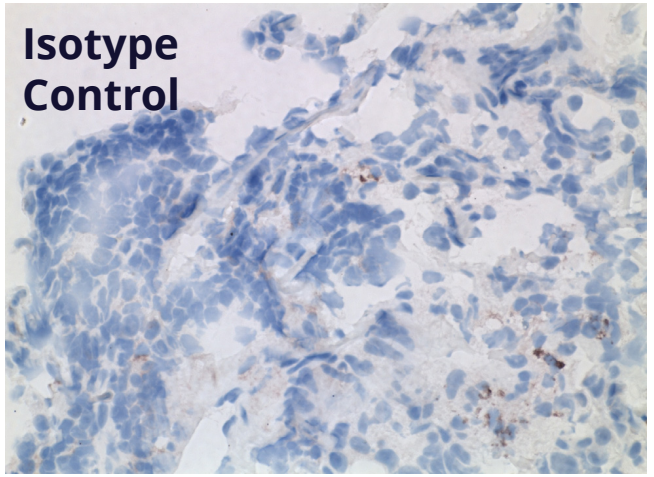
MGD007 detected on membrane of tumor cells (red arrows)





Tumor infiltrating T-cells (yellow arrows) and tumor-adjacent lymphoid nodule (white arrow)

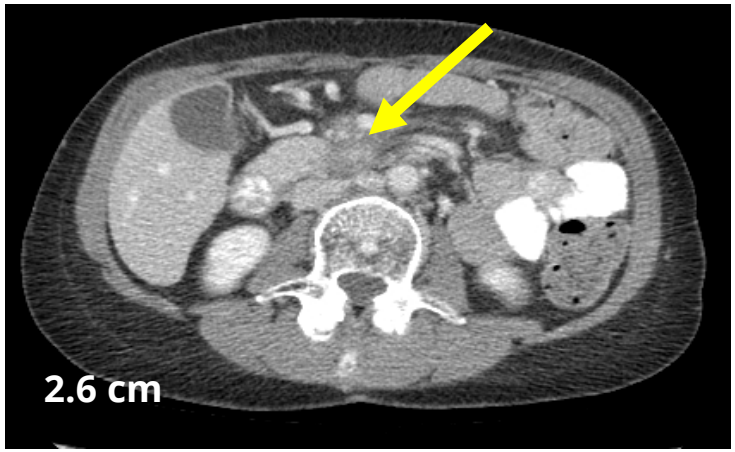
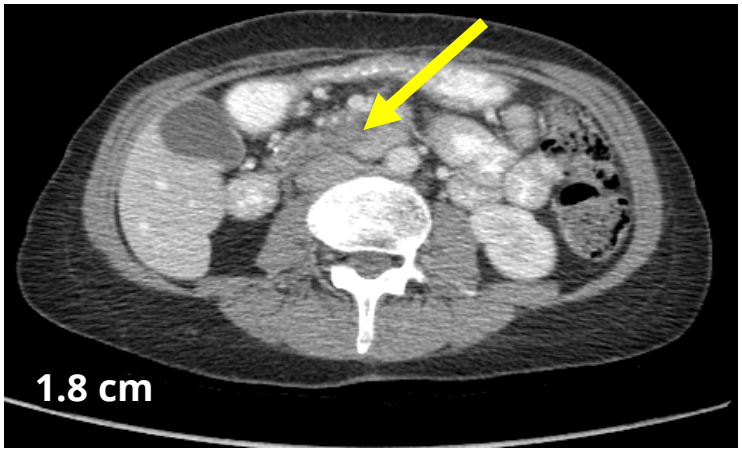
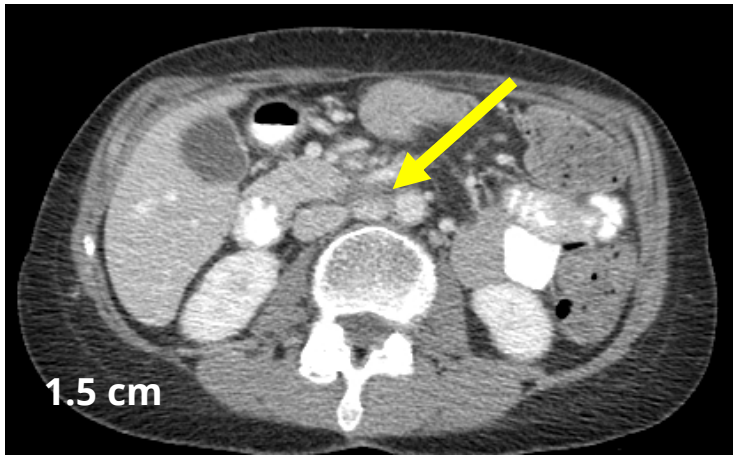
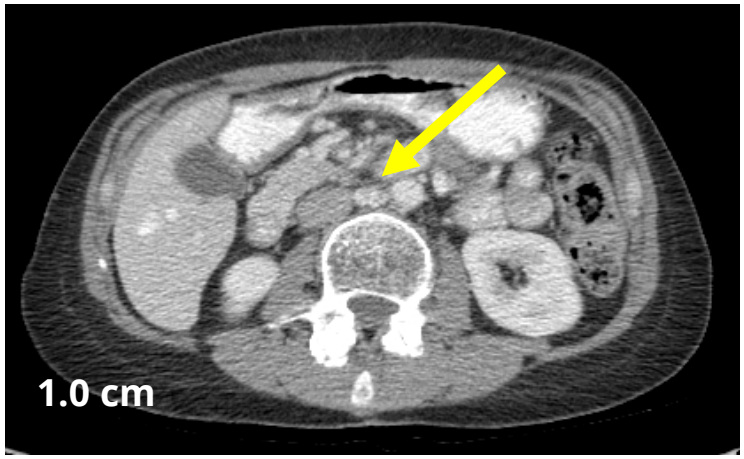




Fresh frozen analyses; objective 40x

MGD007 Dose Escalation: Anti-tumor Activity in CRC

Case Study: 50 y/o female w/ MSI-low CRC w/ intra-abdominal metastasis

	Baseline	End of Cycle 1
Central Mesenteric Mass		
Aortocaval Lymph Node		

Prior treatment history: FOLFOX / irinotecan + bevacizumab / FOLFOX + aflibercept / regorafenib
Treatment & assessment: Received two doses of MGD007, **~32% reduction** in target lesions

MGD007: What Have We Learned to Date?

- Translational findings
  - Dose-dependent binding to CD4+ and CD8+ T-cells in peripheral blood
  - IHC demonstrates DART binding to tumor cells and gpA33^{+} intestinal epithelium
  - Evidence of T cell infiltration within tumor
  - Detectable levels of IL-6, IFN-γ and TNF-α in serum of patients treated with MGD007
- GI/constitutional symptoms reversible and consistent with target distribution
  - Diarrhea appears to be secretory in nature (distinct from checkpoint inhibitor colitis)
  - Current regimen can be delivered and managed in outpatient setting
  - Supportive care consisting of PO/IV corticosteroids, PO non-absorb steroid, anti-cytokine
- No hepatic, pulmonary, cardiac, renal or neurological toxicities related to MGD007

MGD010 Clinical Update

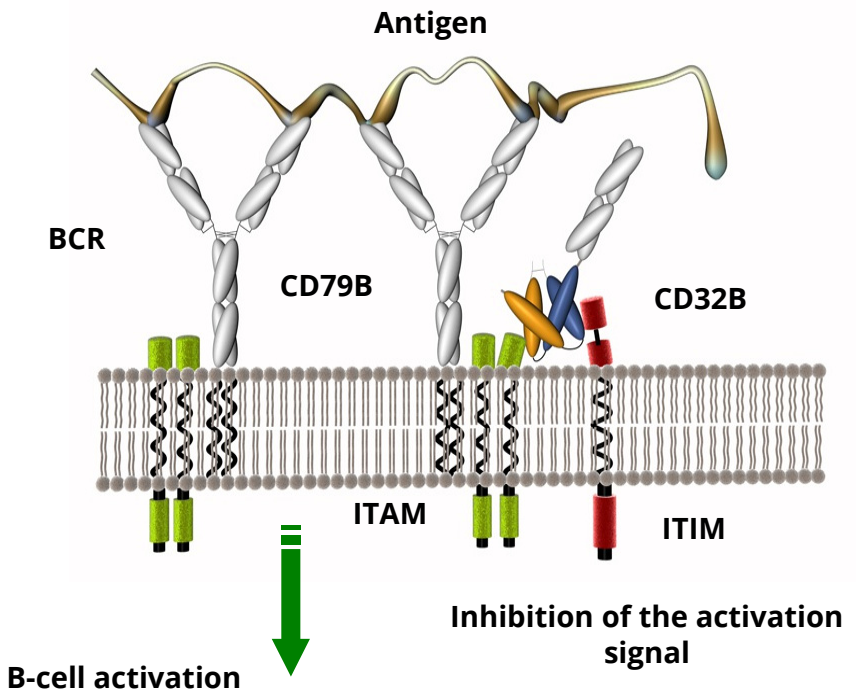
MGD010: CD32B x CD79B DART Product Candidate

Novel mechanism for treatment of autoimmune disorders

Candidate	• Humanized CD32B x CD79B DART with extended PK 
Target/MoA	• Co-ligation of CD32B and CD79B on B lymphocytes – Triggering of negative CD32B-coupled inhibitory loop • Decrease B-cell activation without broad depletion • Rapid onset of action
Development	• Enrollment completed in Phase 1 study (healthy volunteers)
Partner	• MacroGenics retains global rights

Novel Therapeutic Approach to Autoimmune Disorders

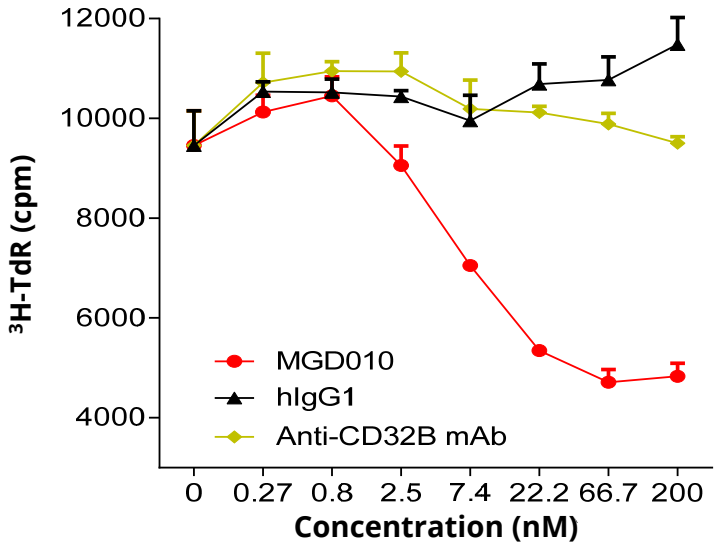
- B cells play an important role in immune tolerance and autoimmunity
  - CD32B (FcyRIIb): checkpoint molecule on B cells
  - CD79B: signal transducing component of B-cell receptor
- MGD010 inhibits B-cell activation
  - Co-ligation of CD32B with CD79B leverages natural physiologic B-cell inhibitory loop, delivering negative signal that limits B-cell activation
- MGD010 potential mechanistic benefits
  - Non-depleting intervention (*vs.* rituximab)
  - Rapid onset of activity (*vs.* belimumab)



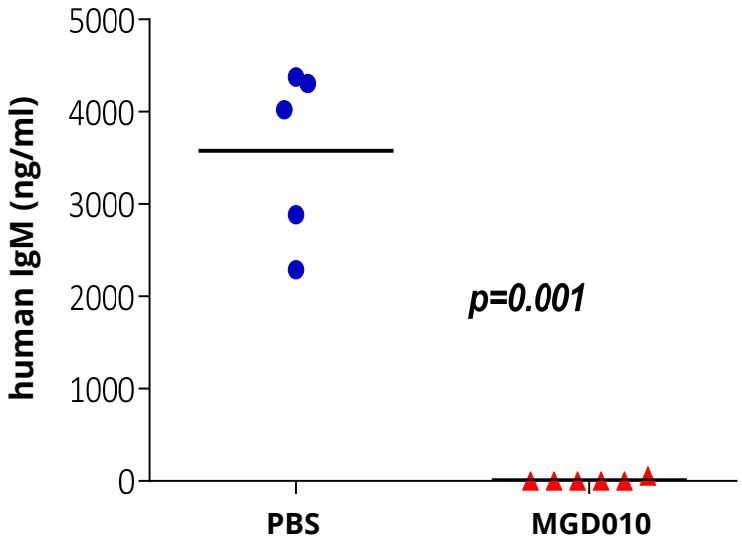
MGD010-mediated Inhibition of B-cell Function

Inhibition of B-cell proliferation in vitro and immunoglobulin secretion in vivo

Inhibition of BCR-induced B-cell Proliferation



Blockade of Ig Secretion in Humanized Mouse Model



- Co-ligation of both targets is essential for functional activity of MGD010
  - Monospecific engagement of CD32B does not result in B-cell inhibition
  - Preclinical models confirm inhibitory properties of MGD010 with decrease in B-cell proliferation and suppression of immunoglobulin secretion

Phase 1 Study: Interim Results Presented at EULAR 2016

Single Ascending Dose in Healthy Subjects

Escalation Cohort (n=49)

Completed ✓

6 Escalating Doses
0.01 – 10 mg/kg

Expansion Cohort with Hep A Vaccination (n=24)

Fully Enrolled

10 mg/kg

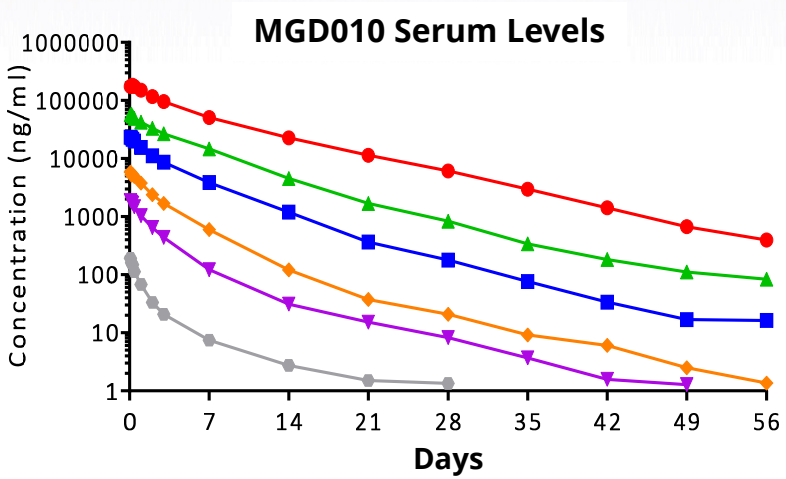
3 mg/kg

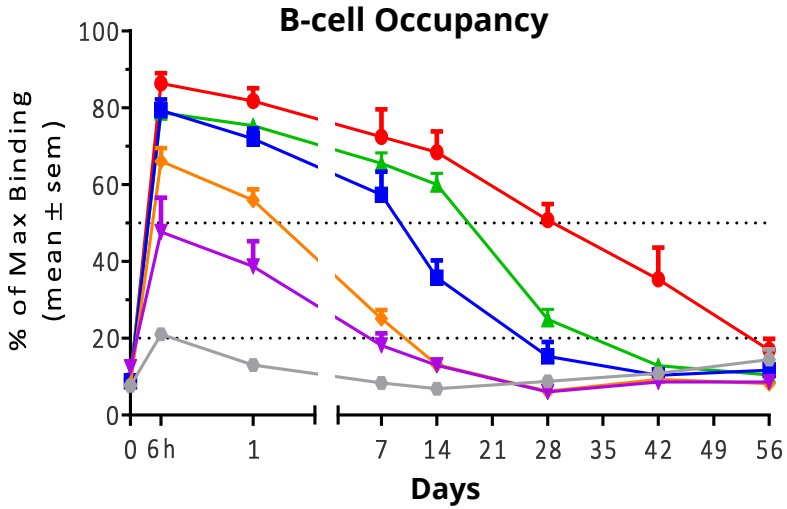
Placebo

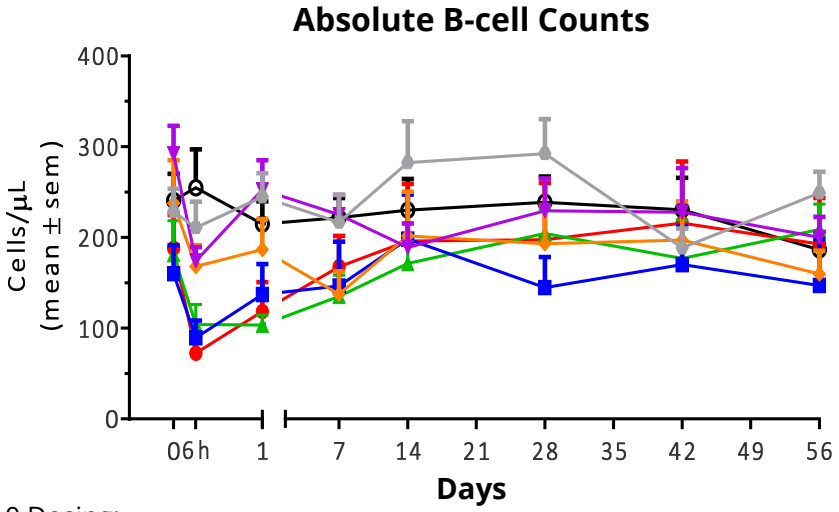
- **Primary Objective**
  - Assess safety and tolerability of single dose
- **Secondary Objectives**
  - Evaluate pharmacokinetics and pharmacodynamics effects
  - Evaluate potential anti-drug antibodies
- **Exploratory Objectives**
  - Evaluate binding and activation status on peripheral B cells and B-cell subsets
  - Assess immune phenotype, including modulation of B-cell subsets
  - Assess response of peripheral B cells to ex-vivo BCR stimulation

Pharmacokinetics and Circulating B-cell Occupancy

- MGD010 serum concentrations increase linearly with dose
  - Half-life: ~8 days at ≥1mg/kg
- Maximum B-cell occupancy at doses ≥1 mg/kg
  - Sustained receptor occupancy beyond one week at doses ≥ 1 mg/kg
- No B-cell depletion or cytokine release (data not shown) at any dose levels



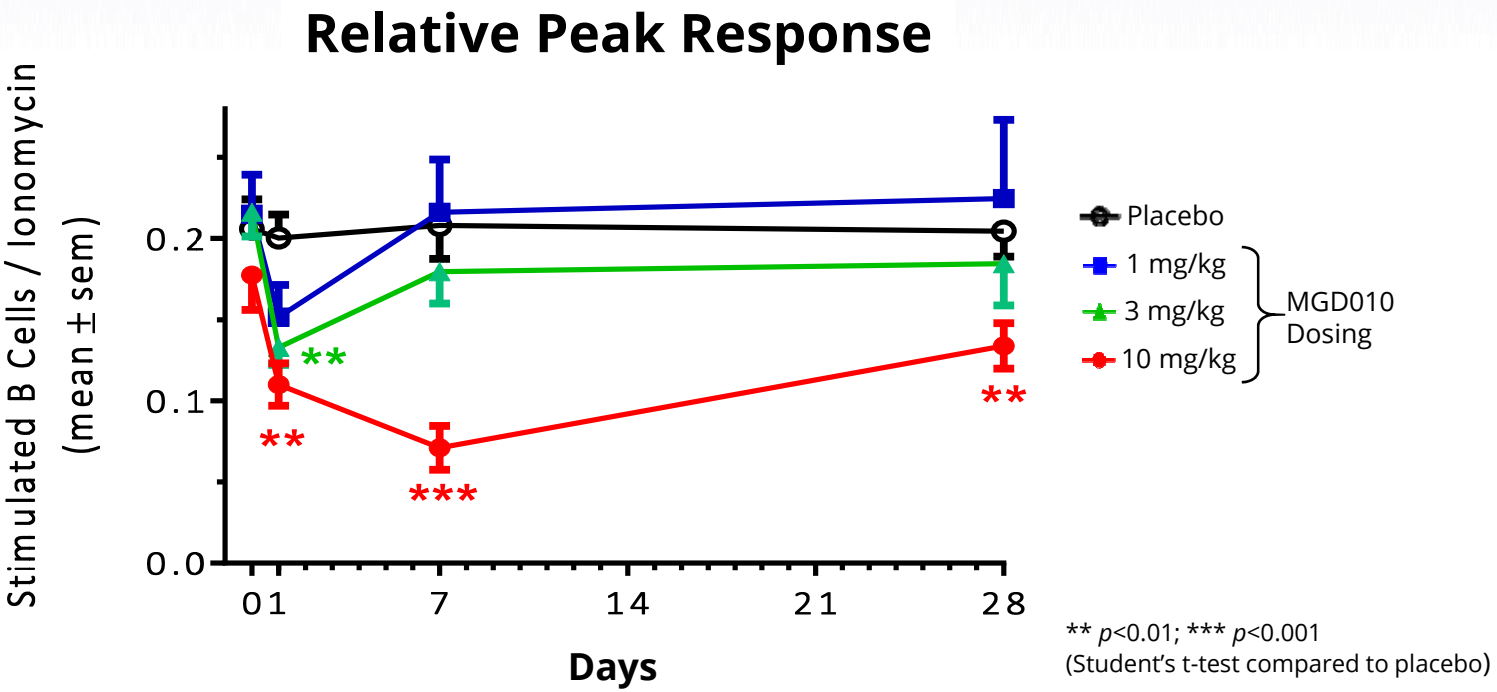




MGD010 Dosing:

Placebo | 0.01mg/kg | 0.1mg/kg | 0.3mg/kg | 1 mg/kg | 3 mg/kg | 10 mg/kg

Treatment with MGD010 Inhibited B-Cell Activation



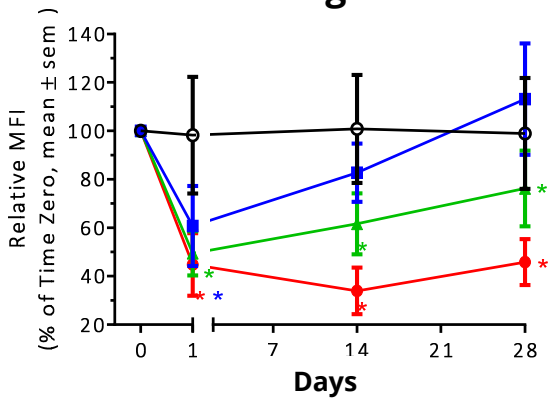
- PBMCs from enrolled subjects collected longitudinally after MGD010 treatment
  - Ca^{2+} flux (hallmark of B-cell activation) induced *ex vivo* by B-cell receptor ligation using anti-IgM
    - Data normalized to maximum Ca^{2+} permeability (maximum induction) via ionomycin
- Dose dependent B-cell inhibition demonstrated with increasing doses of MGD010
  - Inhibition sustained for several weeks at highest dose levels

MGD010 Modulated Cell Surface BCR and Serum Ig Levels

MGD010 Down-Regulation of Cell Surface Ig Expression

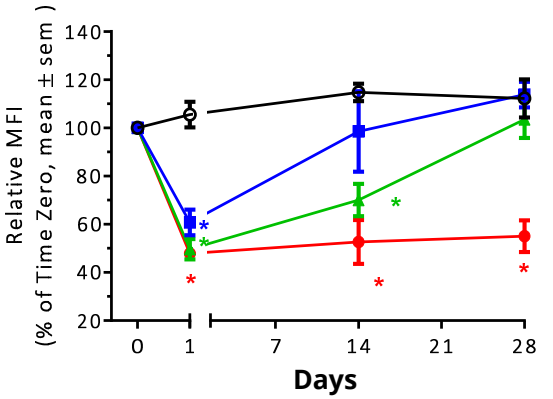
CD27 $^{-}$ Naïve B Cells

mIgD



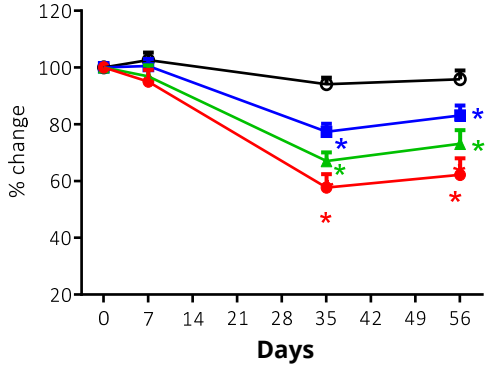
CD27 $^{+}$ Memory B Cells

mIgG

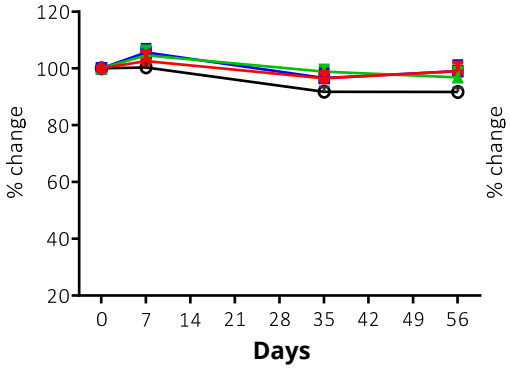


MGD010 Modulation of Circulating Serum Ig Levels

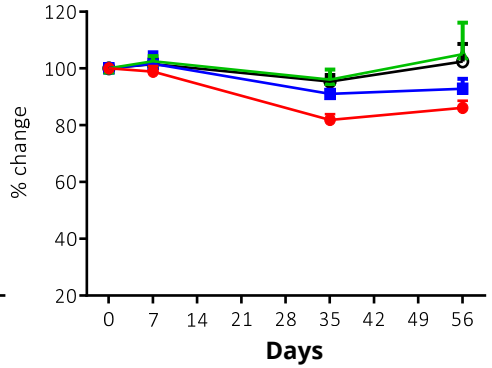
Serum IgM



Serum IgG



Serum IgA



- Dose-dependent down modulation of cell surface BCR expression on both naïve and memory B cells

* p<0.05 (Paired t-test compared to baseline)

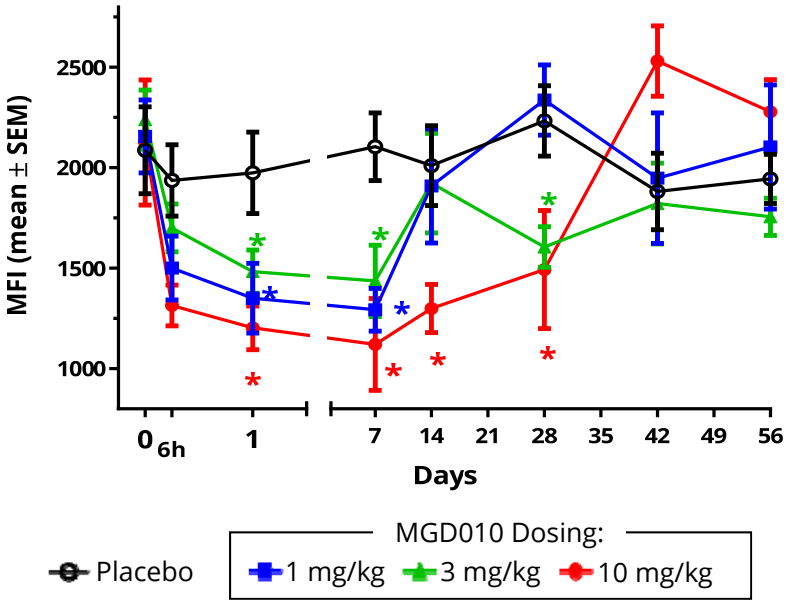
Placebo | MGD010 Dosing: 1 mg/kg, 3 mg/kg, 10 mg/kg

MGD010 Reduces CD40 Expression and Response

Potential impact on B-cell : T-cell interactions

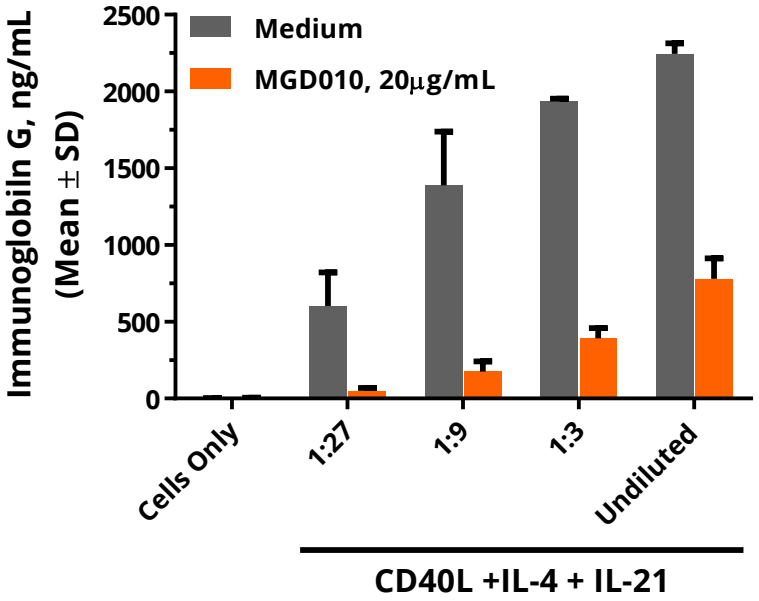
- CD40 is co-stimulatory molecule involved in T:B cell cross-talk
- CD40/CD40L interaction differentiates B cells & activates antigen presenting cells

Decreased CD40 Expression in Subjects Treated with MGD010



* $p<0.05$ (Paired t-test compared to baseline)

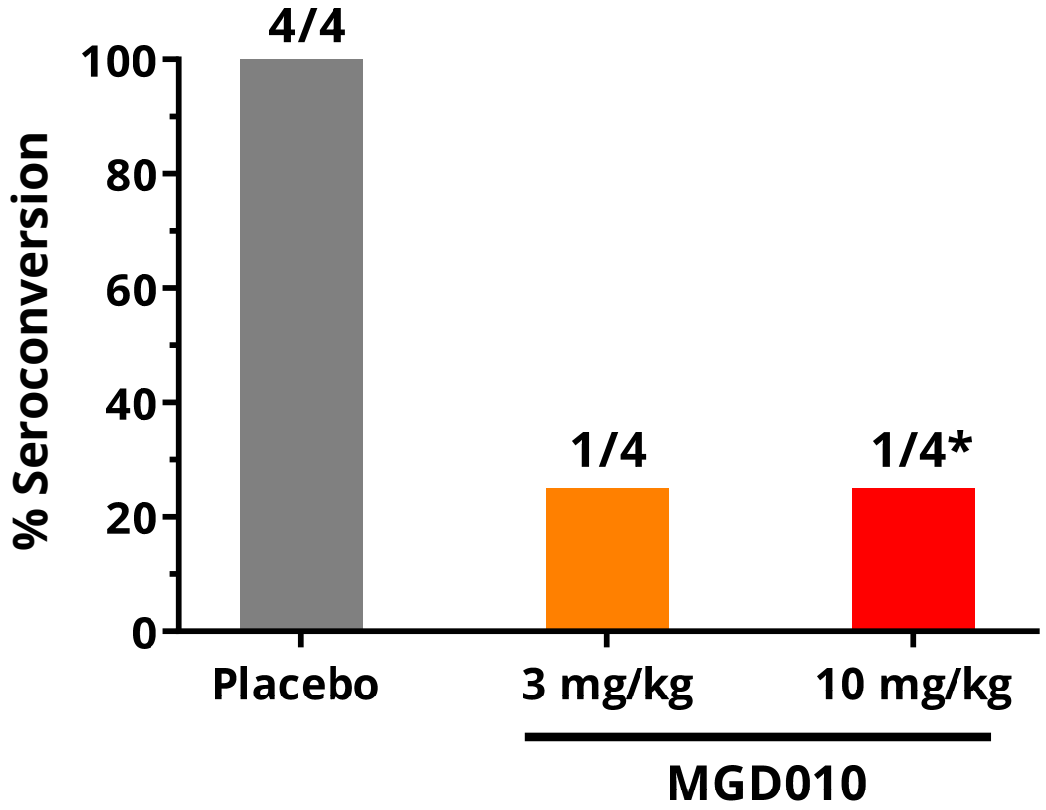
MGD010 Decreases CD40L-dependent IgG Production in vitro



- Purified human B cells exposed to CD40L (500ng/mL), IL-4 (100ng/mL, a Th2 cytokine) and IL-21 (20ng/mL, a Tfh cytokine) to induce differentiation towards Ig-producing cells
- IgG levels determined on day 5

MGD010 Inhibits Ag-specific Response in Healthy Volunteers

Response to hepatitis A vaccine (a model antigen) in Phase 1 expansion cohort



- Interim seroconversion data (day 56 post vaccination)
- Quantitative results pending

** One additional patient did not complete day 56 follow-up, but was negative until day 29*

Exploring Broad Range of Potential Indications to Pursue

Larger, More Established Indications*

- Systemic Lupus Erythematosus (SLE)
- Multiple Sclerosis (MS)
- Rheumatoid Arthritis (RA)

Niche, Less Established treatment options

- Sjogren's Syndrome (SS)
- Primary Vasculitis (e.g. Polymyalgia rheumatica/Giant cell arteritis/Behçets)
- Graft vs. Host Disease (GVHD)
- Myasthenia Gravis
- Pemphigus
- Neuromyelitis Optica
- Anti-NMDA receptor encephalitis
- Guillain–Barré syndrome
- Chronic inflammatory demyelinating polyneuropathy (CIDP)
- Grave's opthalmopathy
- IgG4 RD
- Idiopathic thrombocytopenic purpura (ITP)

** Indications with currently approved and marketed agents*

MGD010: Inhibits Multiple Aspects of B-Cell Function

- Well tolerated up to 10 mg/kg in healthy subjects in single dose study
- Inhibits antigen-specific response in healthy volunteers
- Sustained biological effects for as long as two months
- Activity consistent with preclinical models
  - No B-cell activation or cytokine release
  - No depletion of peripheral B cells
  - BCR saturated at ≥ 1mg/kg with sustained receptor occupancy
- Down-modulates B-cell function at multiple levels
  - Reduction in BCR-induced Ca^{2+} mobilization
  - Decrease surface Ig expression and serum IgM levels
  - Down-modulation and inhibition of CD40 function
- Broad opportunity across multiple autoimmune disorders
- Upcoming milestone: report updated Ph. 1 study data (Hep A cohort) in 2017

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Scott Koenig, M.D., Ph.D. – President & CEO, MacroGenics

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Jon Wigginton, M.D. – Senior VP, Clinical Development & CMO, MacroGenics

Comprehensive B7-H3 Franchise: Fc-Optimized mAb, DART and ADC
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DART and TRIDENT: Leading Multi-specific Antibody Platforms
Syd Johnson, Ph.D. – VP, Antibody Engineering, MacroGenics

Clinical DART Programs Update
Jon Wigginton, M.D. – Senior VP, Clinical Development & CMO, MacroGenics

Q&A / Break

"Combination Treatments with Checkpoint Blockade"
F. Stephen Hodi, Jr., M.D. – Director, Center for Immuno-Oncology, Dana-Farber Cancer Institute

Immuno-Oncology: Targeting Immune Regulators
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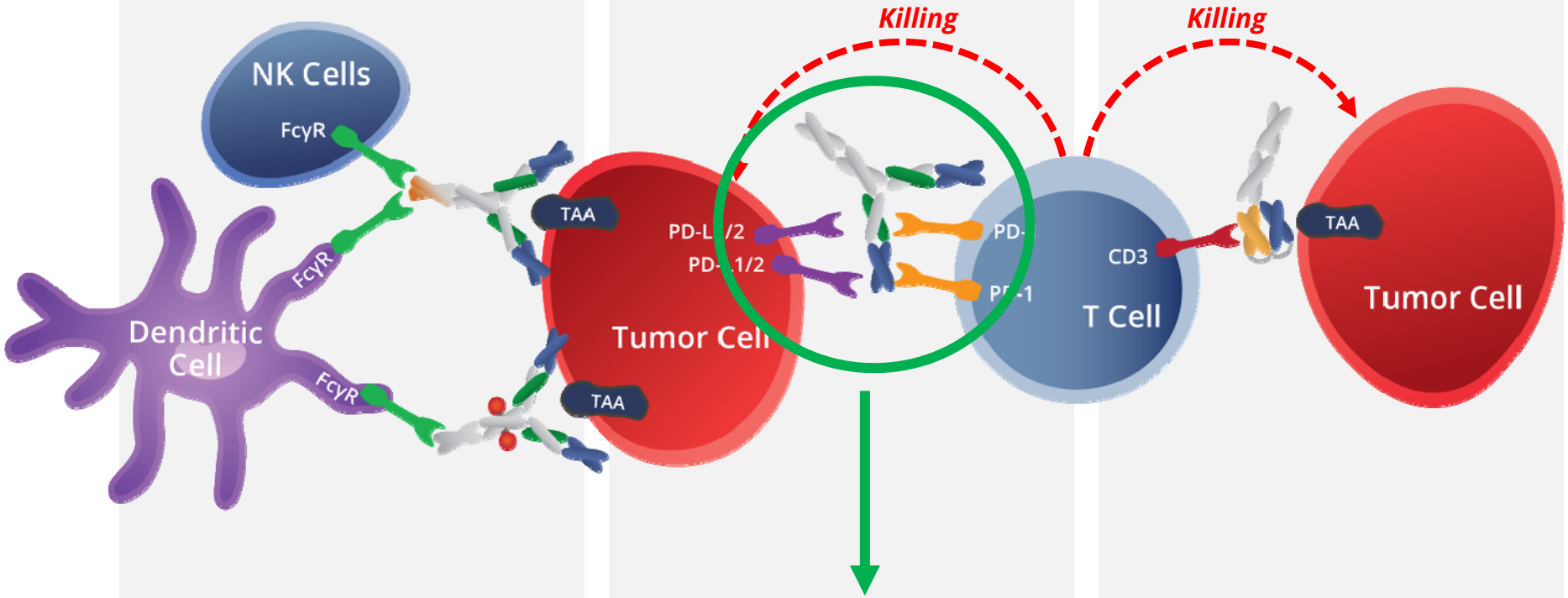
Integrating PD-1 Blockade into MacroGenics' Portfolio

Tumor Destruction and Antigen Presentation: Tumor-targeted Interventions

Restore T-Cell Function: Checkpoint Inhibition

Enhance T-cell Function: T-Cell Recruitment



margetuximab
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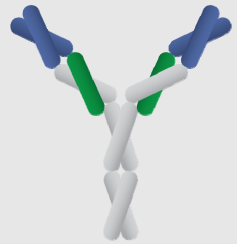
MGA012 (anti-PD-1)

MGD006
MGD007
MGD009

TAA: tumor-associated antigen

MGA012: Anti-PD-1 Antibody

Introducing MGA012: Anti-PD-1 mAb

Candidate	• Humanized proprietary anti-PD-1 mAb – Hinge stabilized humanized IgG4 – Benchmarks favorably against leading mAbs 
Rationale	• Anti-PD-1 as mainstay of cancer immunotherapy • Basis for combination immunotherapy with proprietary assets • Potential commercial/reimbursement advantages
Function/MoA	• Blockade of PD-1 interaction with PD-L1 & PD-L2, which down-regulates T-cell activation • Disrupts negative signaling pathway in T cells directed against tumors (checkpoint inhibition)
Indications	• Multiple solid tumors
Development	• Phase 1 trial dosing ongoing
Partner	• MacroGenics retains global rights

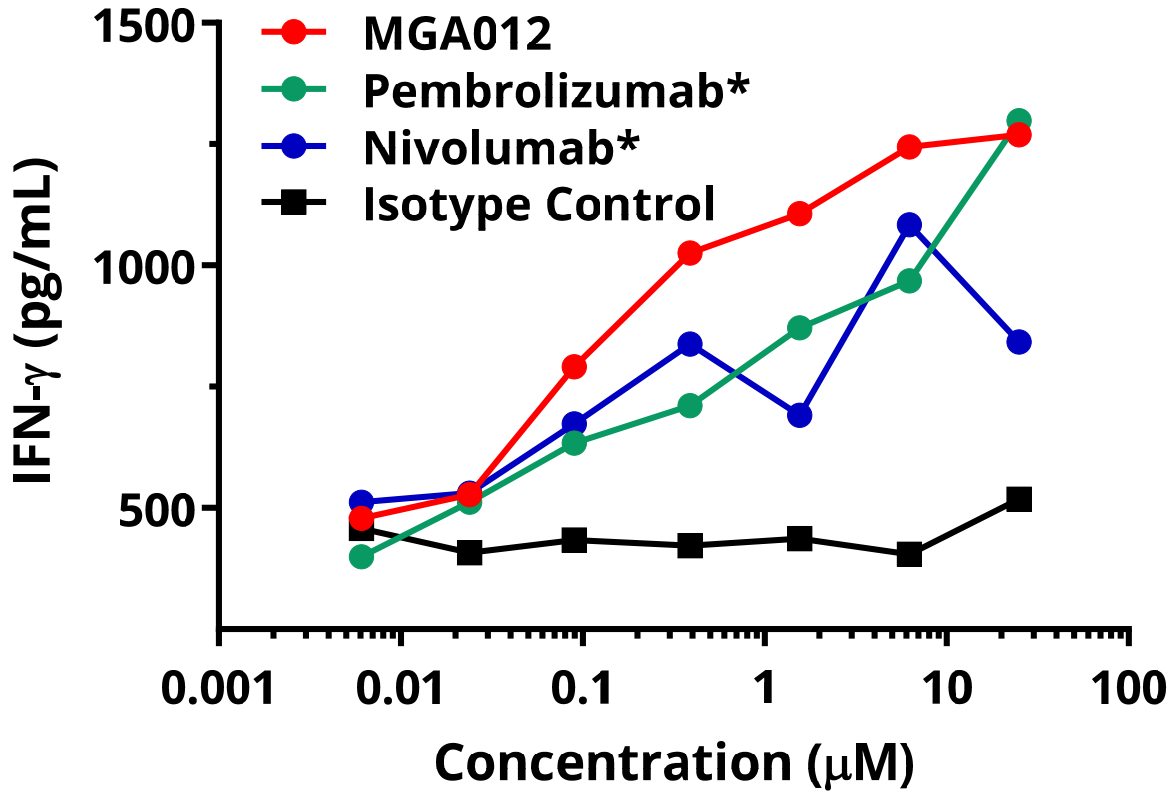
MGA012: Favorable Technical Profile

MGA012	MGA012 Compared to:	
	Nivo*	Pembro*
Affinity for human PD-1	>4x greater	>6x greater
Off-rate for human PD-1	~2x slower	~6x slower
Cell binding (MFI)	>	Equivalent
PD-L1/PD-L2 binding blockade	>	>
T-cell activation (IFNγ)	Equivalent	Equivalent
PK in cynomolgus monkeys	>	Equivalent

MGA012	Results
Tissue cross-reactivity	No unanticipated findings
Toxicology in cynomolgus monkeys: IV at 10, 40 or 150 mg/kg; QW x 4	Well tolerated at all doses No unanticipated findings NOAEL = 150 mg/kg
Predicted half-life in humans	~18 days

**Replicas of nivolumab and pembrolizumab produced at MacroGenics*

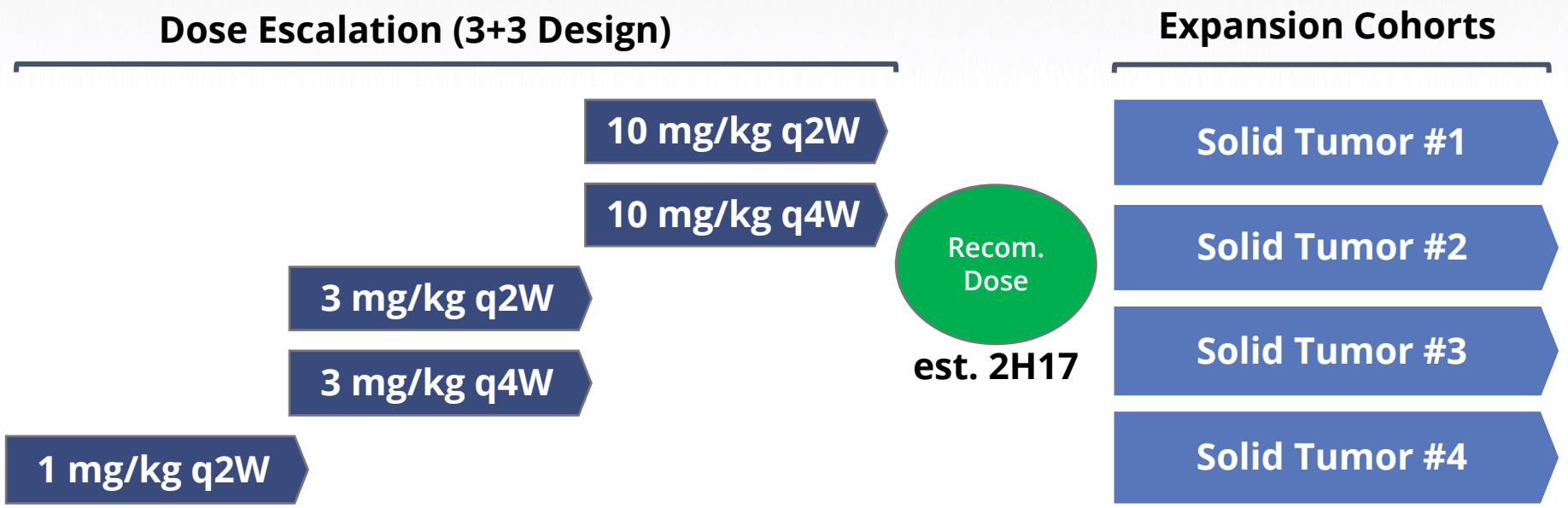
MGA012 Enhances Activation of SEB-stimulated Human T Cells



Human PBMCs were pre-stimulated with 0.5 ng/ml SEB for 48h and re-stimulated for 48h in presence or absence of indicated mAbs. IFNγ in supernatant was measured by ELISA.

**Replicas of pembrolizumab and nivolumab produced at MacroGenics*

MGA012 (Anti-PD-1): Phase 1 Study Design



Objectives: Establish safety profile and initial clinical activity
Confirm that MGA012 compares favorably to benchmark PD-1 data

Patient Population: Any relapsed / refractory advanced or metastatic solid tumor

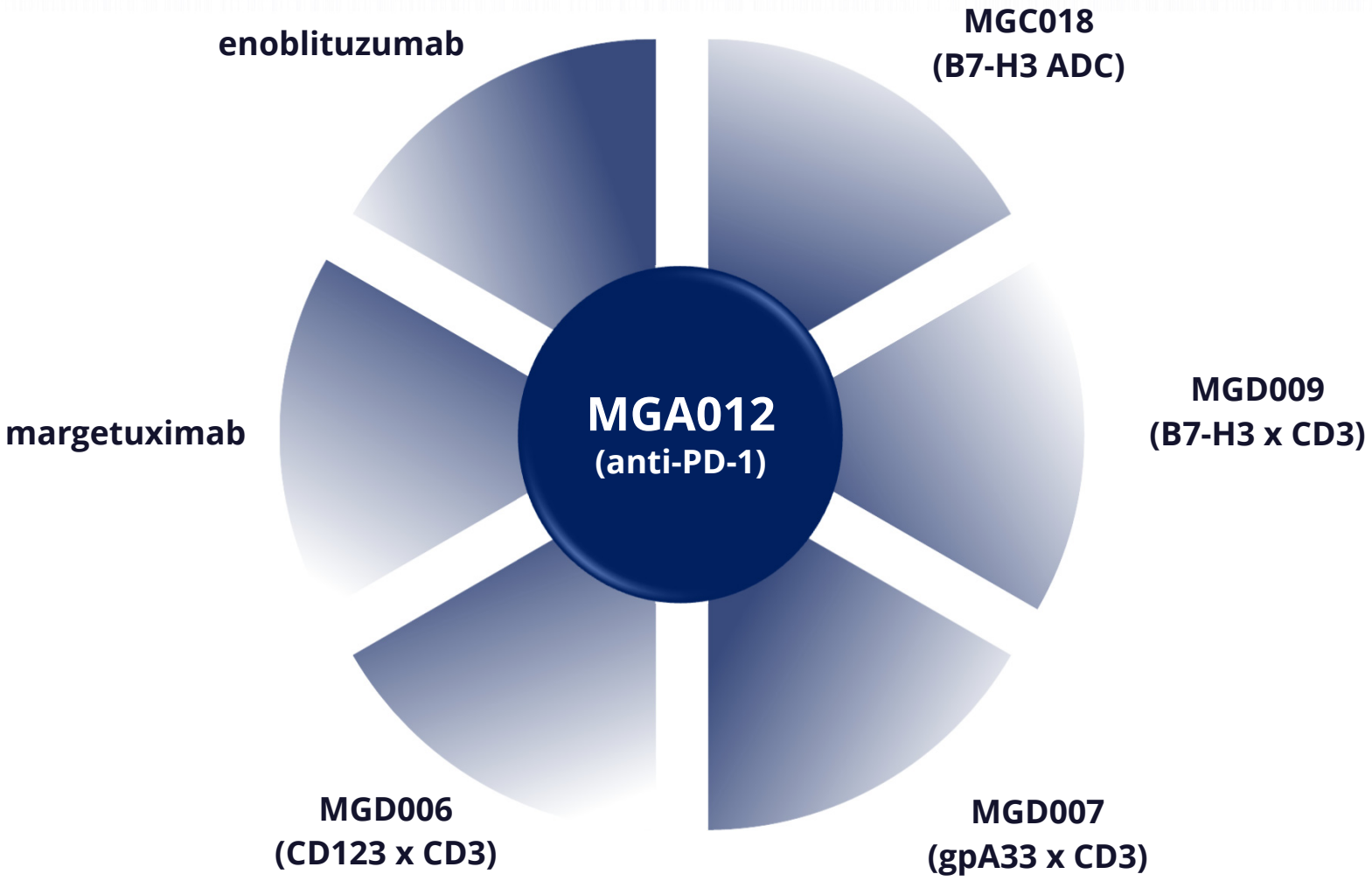
Dosing Regimen: 1-hr Intravenous infusion, q2W or q4W

Evaluations: RECIST and irRECIST

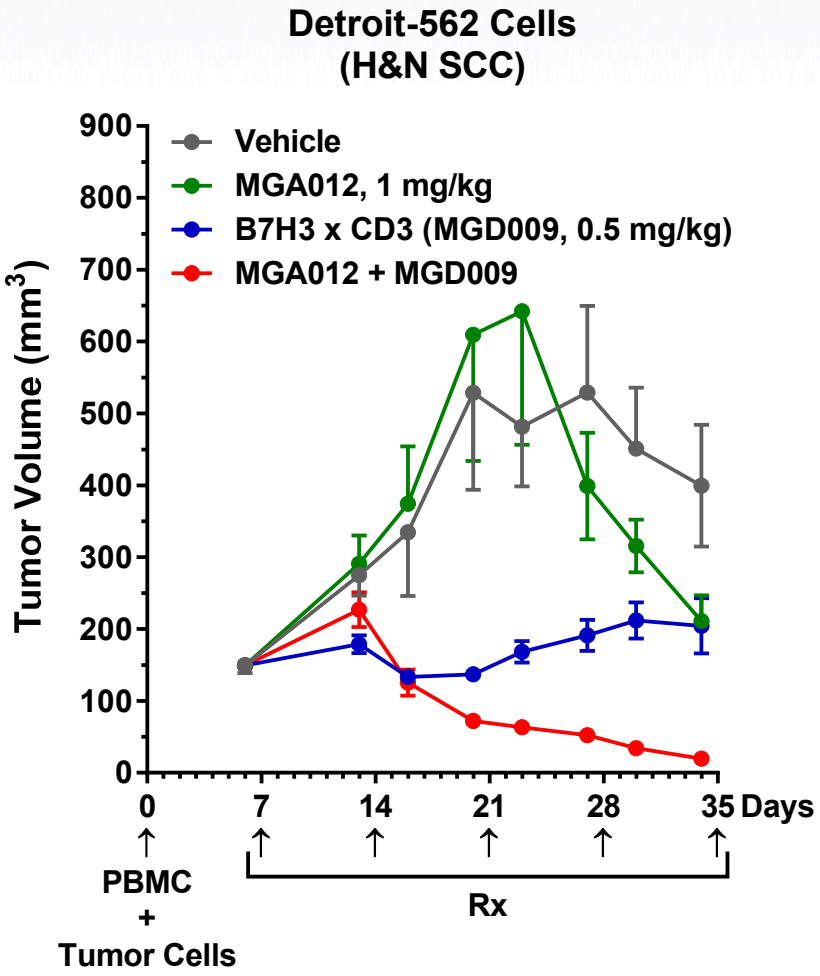
Site Deployment: ~30 Sites across United States, Europe and Australia

Enabling Multiple Combo Opportunities with MGA012

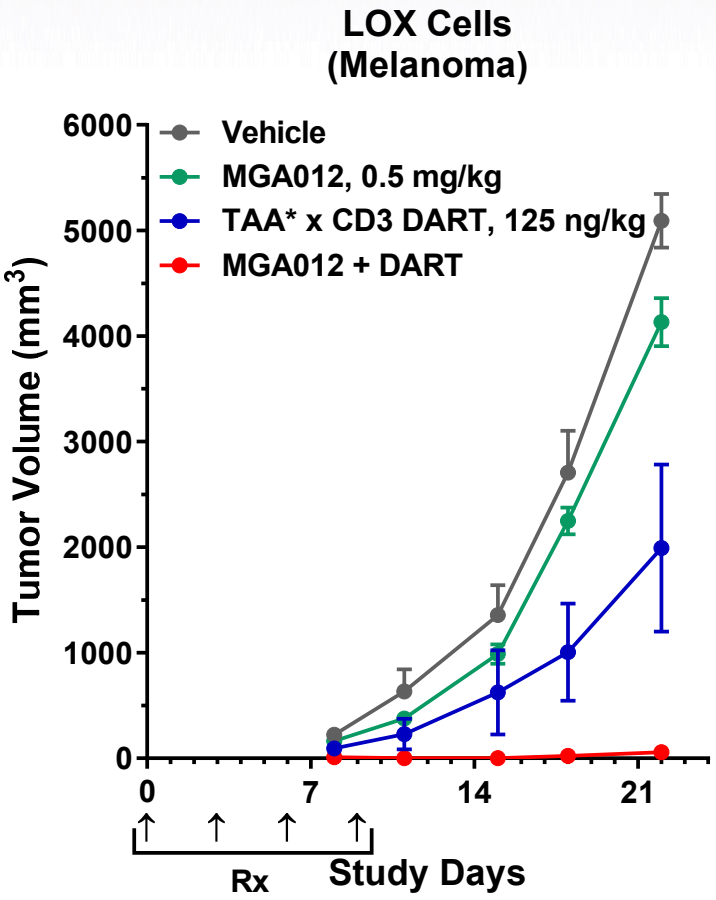
Primary goal: early combination with internal pipeline



MGA012 Enhances DART-mediated T-cell Killing in vivo



NSG MHC-I$^{-/-}$ mice
Detroit-562, 5x10^6 cells, ID
PBMC, 10^6 cells, IP



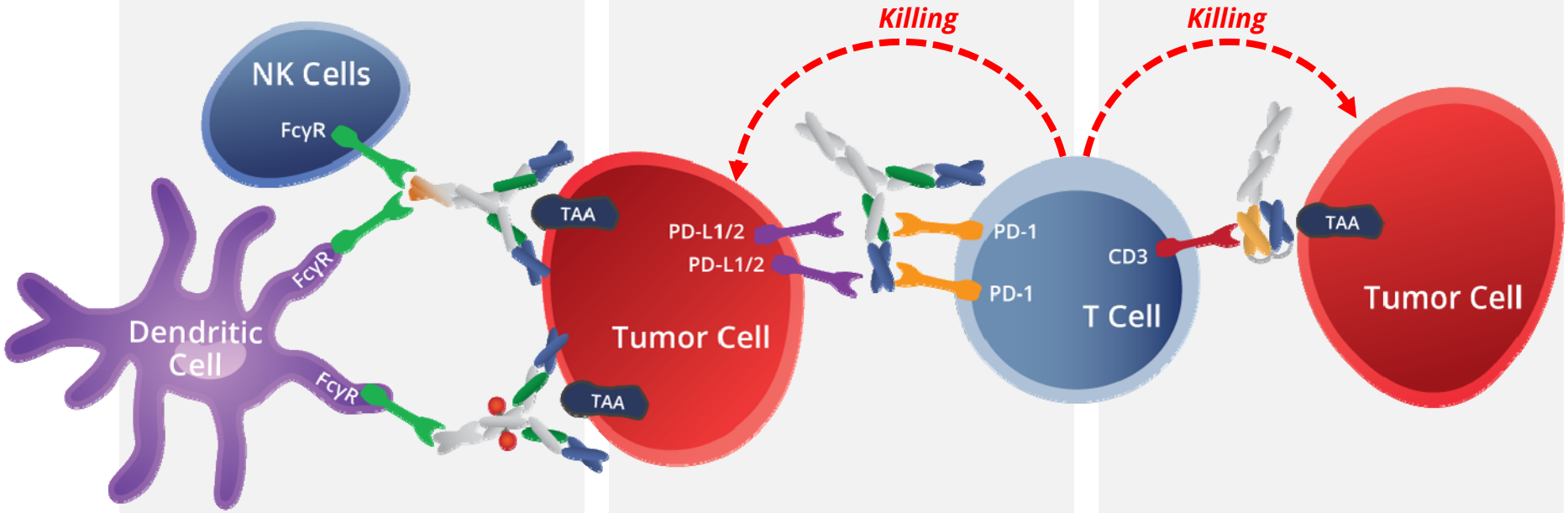
NSG mice co-mix model
LOX cells, 2.5x10^6 cells &
activated human T cells, 1x10^6 cells, SC
* TAA: tumor-associated antigen

Integrating PD-1 Blockade in MacroGenics' Portfolio

Tumor Destruction and Antigen Presentation: Tumor-targeted Interventions

Restore T-Cell Function: Checkpoint Inhibition

Enhance T-cell Function: T-Cell Recruitment



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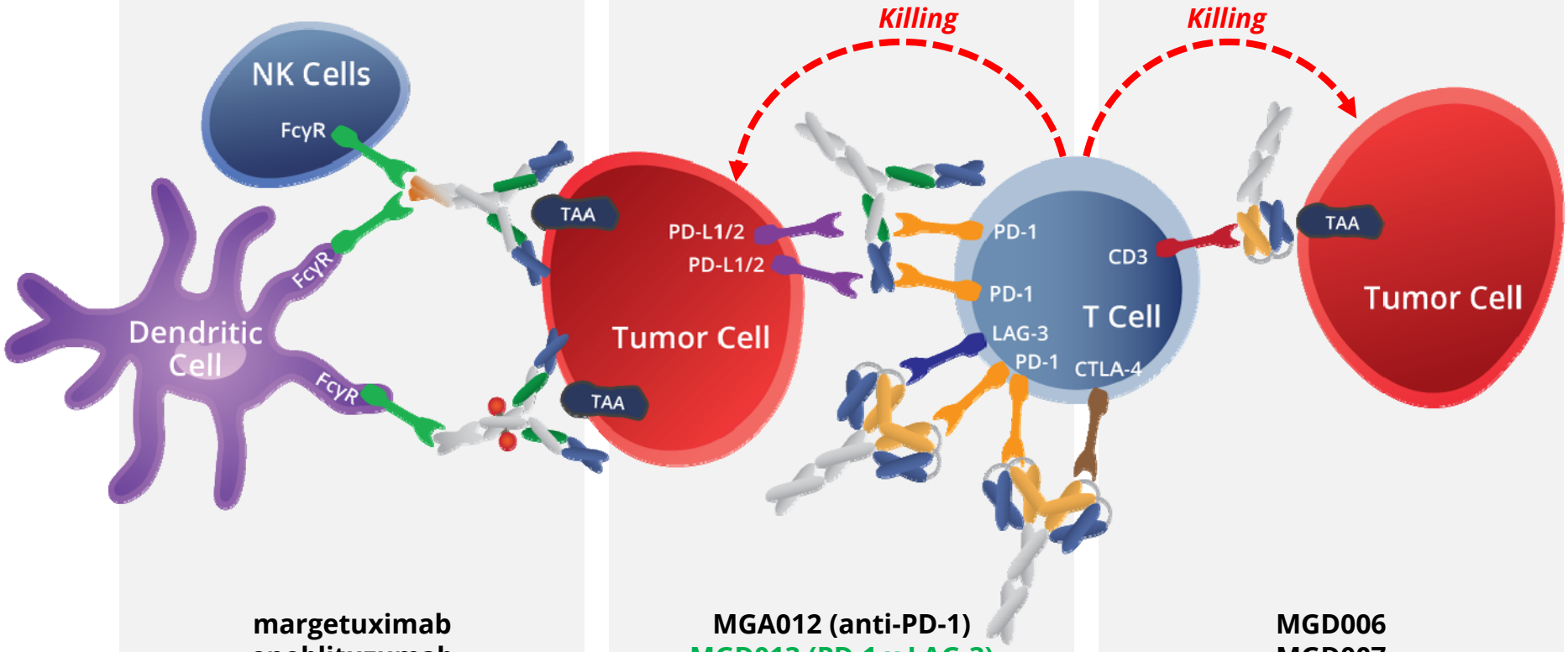

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MGD013 (PD-1 x LAG-3)
PD-1 x CTLA-4

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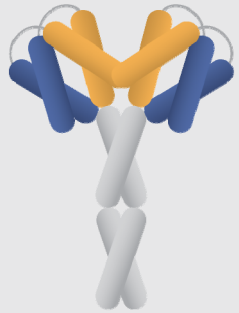
MGD007
MGD009

TAA: tumor-associated antigen

Combinatorial Checkpoint Inhibition

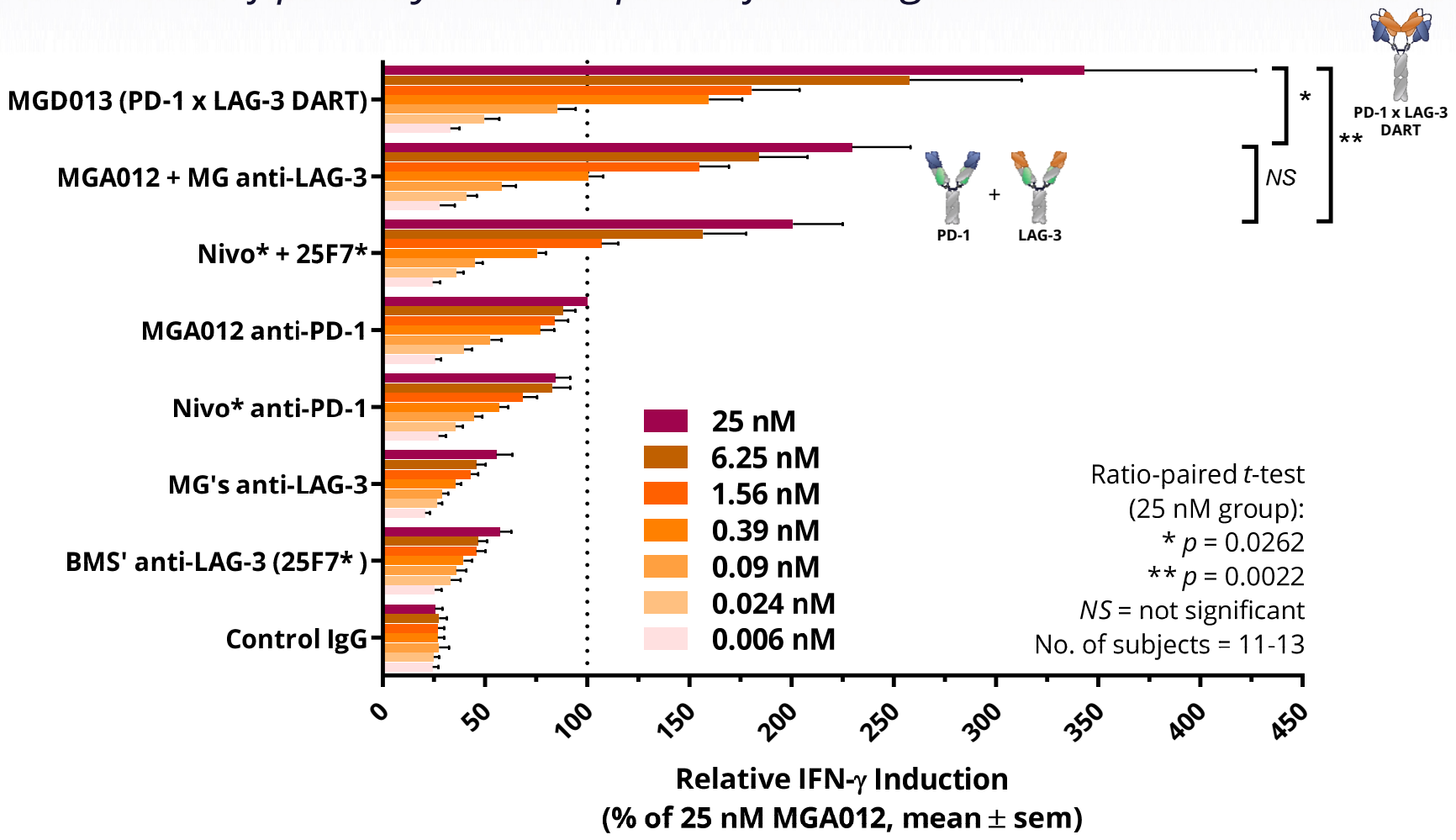
Strategy for enhancing antitumor adaptive responses

MGD013 Poised for Clinical Development in 2017

Candidate	• Humanized, proprietary PD-1 x LAG-3 DART – Hinge-stabilized human IgG4 – Benchmarks favorably against leading mAbs 
Rationale	• Coordinate blockade of two checkpoint co-expressed on T cells
Function/MoA	• "Rejuvenation" of "exhausted" T cells
Indications	• Multiple solid tumors and hematological malignancies
Development	• IND targeted for 1H2017
Partner	• MacroGenics retains global rights

MGD013 Enhances TCR-driven Activation In Vitro

Enhancement of primary T-cell response following SEB stimulation



IFNγ release by 25 nM MGA012 = 3276 ± 744 pg/mL

MGD013: Synergistic Checkpoint Inhibition

Pre-clinical development:

- Superiority compared to two stand alone mAbs
- Favorable toxicology profile in cynomolgus monkeys
- Projected human dosing at ≥ 2 week intervals
- IND-enabling activities & GMP manufacturing successfully completed

Clinical opportunities:

- Salvage patients who have progressed on prior checkpoint inhibitor
- Superiority opportunity against PD-1 mAb or PD-1 mAb + LAG-3 mAb combo

Advantages:

- Potential clinical superiority
- Simpler clinical development path than mAb combination
- Enhanced patient convenience
- Potential commercial advantages

PD-1 x CTLA-4 DART & TRIDENT Program

Tailoring valency to precise pharmacology

PD-1 x CTLA-4 Dual Checkpoint Targeting Rationale

- PD-1 and CTLA-4: clinically validated co-inhibitory molecules
- Complementary mechanisms of action:
  - Anti-PD-1: release of T-cell inhibition at tumor sites
  - Anti-CTLA-4: polyclonal activation/expansion
- Coordinated CTLA-4 and PD-1 blockade has achieved synergistic antitumor activity in clinic

Challenge - Maintain potency of combinatorial blockade via:

- **Full PD-1 blockade**
- **Tunable levels of CTLA-4 blockade**

PD-1 x CTLA-4 Dual Checkpoint Targeting Strategy

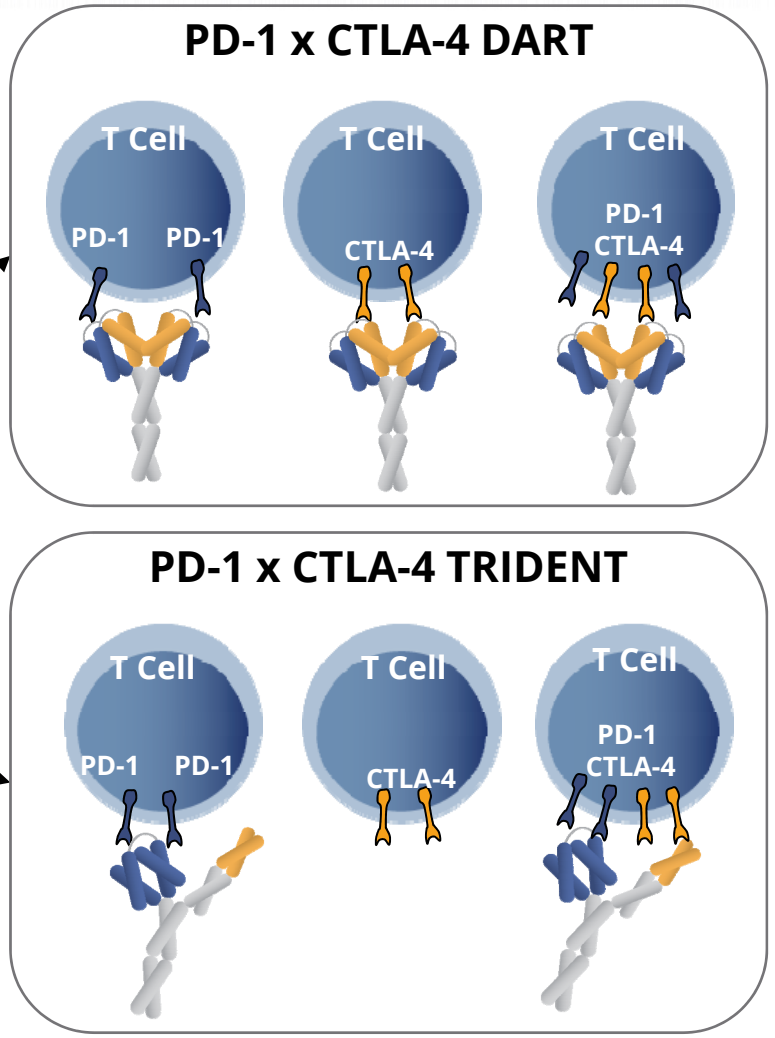
Tailoring valency to control stand-alone CTLA-4 blockade

PD-1 arm: Bivalent

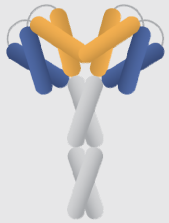
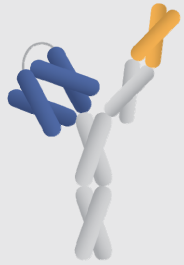
- Maintain full PD-1 blockade, irrespective of CTLA-4 co-expression

CTLA-4 arm: Variable valency

- **DART**: bivalent anti-CTLA-4
  - Full CTLA-4 blockade, irrespective of PD-1
- **TRIDENT**: Monovalent anti-CTLA-4
  - CTLA-4 blockade biased toward PD-1 co-expression

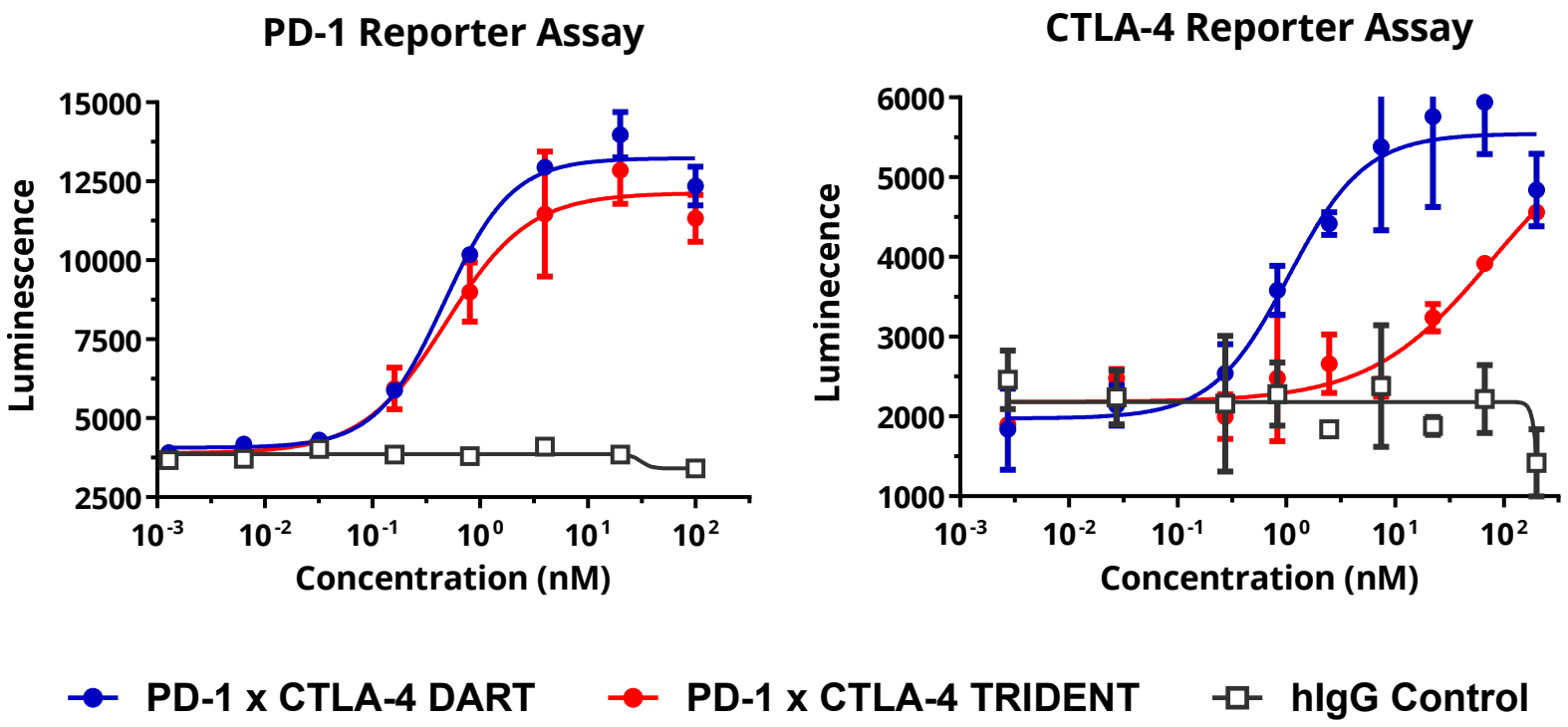


PD-1 x CTLA-4 DART / TRIDENT Program

Candidates	• Humanized anti-PD-1 x anti-CTLA-4	
Constructs	• Fc-DART (IgG4) PD-1 x CTLA-4 x CTLA-4 x PD-1 	• TRIDENT (IgG4) PD-1 x PD-1 x CTLA-4 
Preferred Profile	• Independent blockade of both PD-1 and CTLA-4	• Full PD-1 blockade independent of CTLA-4 expression • CTLA-4 blockade biased toward co-expression with PD-1
Indications	• Multiple solid tumors and hematological malignancies	
Development	• Candidate selection ongoing	
Partner	• MacroGenics retains global rights	

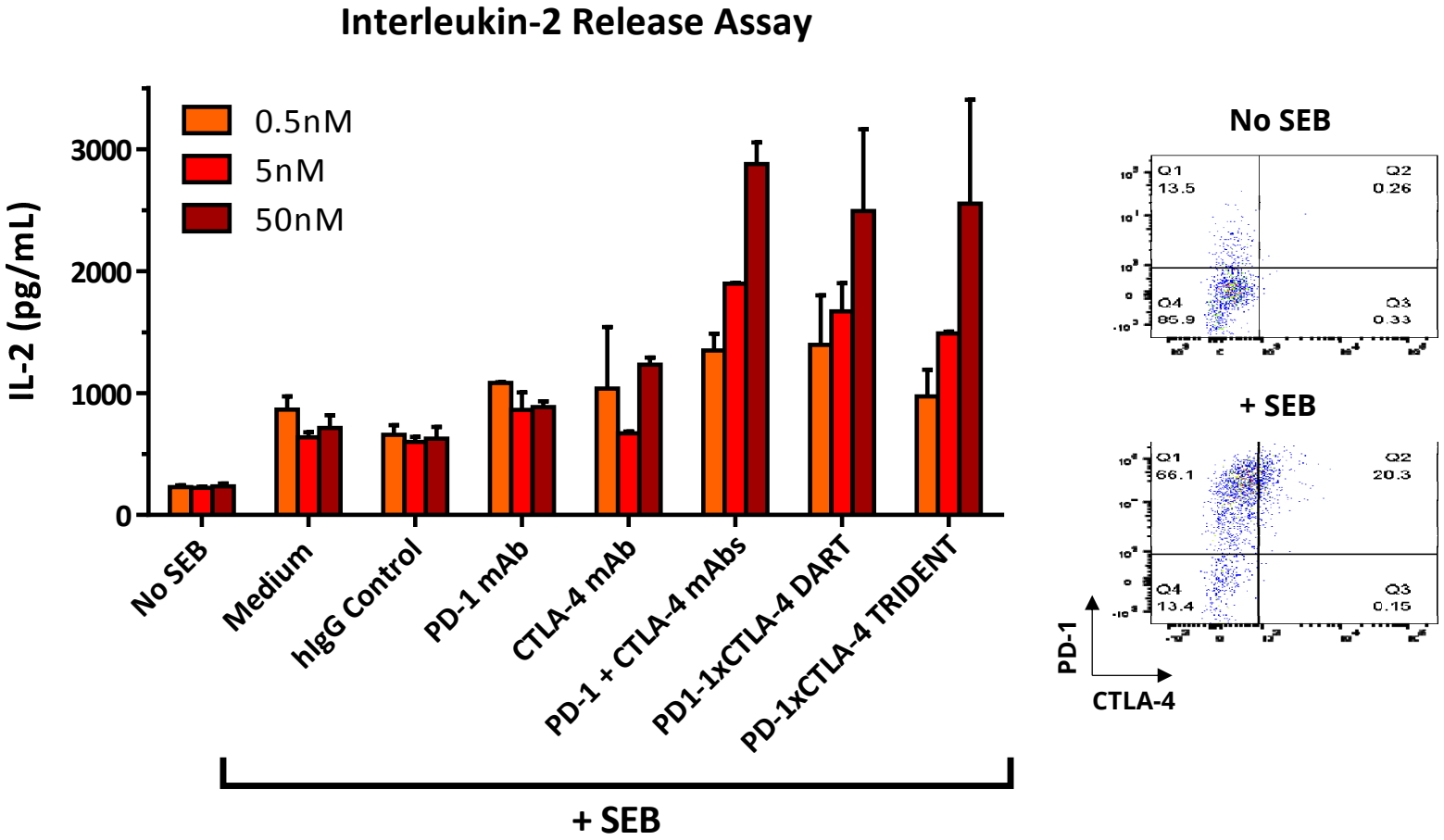
PD-1 x CTLA-4-mediated Enhancement of TCR Signal Transduction

Differential CTLA-4 blockade by DART & TRIDENT molecules



PD-1 x CTLA-4 DART & TRIDENT Enhance T-cell Responses

PD-1 x CTLA-4 DART & TRIDENT recapitulate individual mAbs' activity



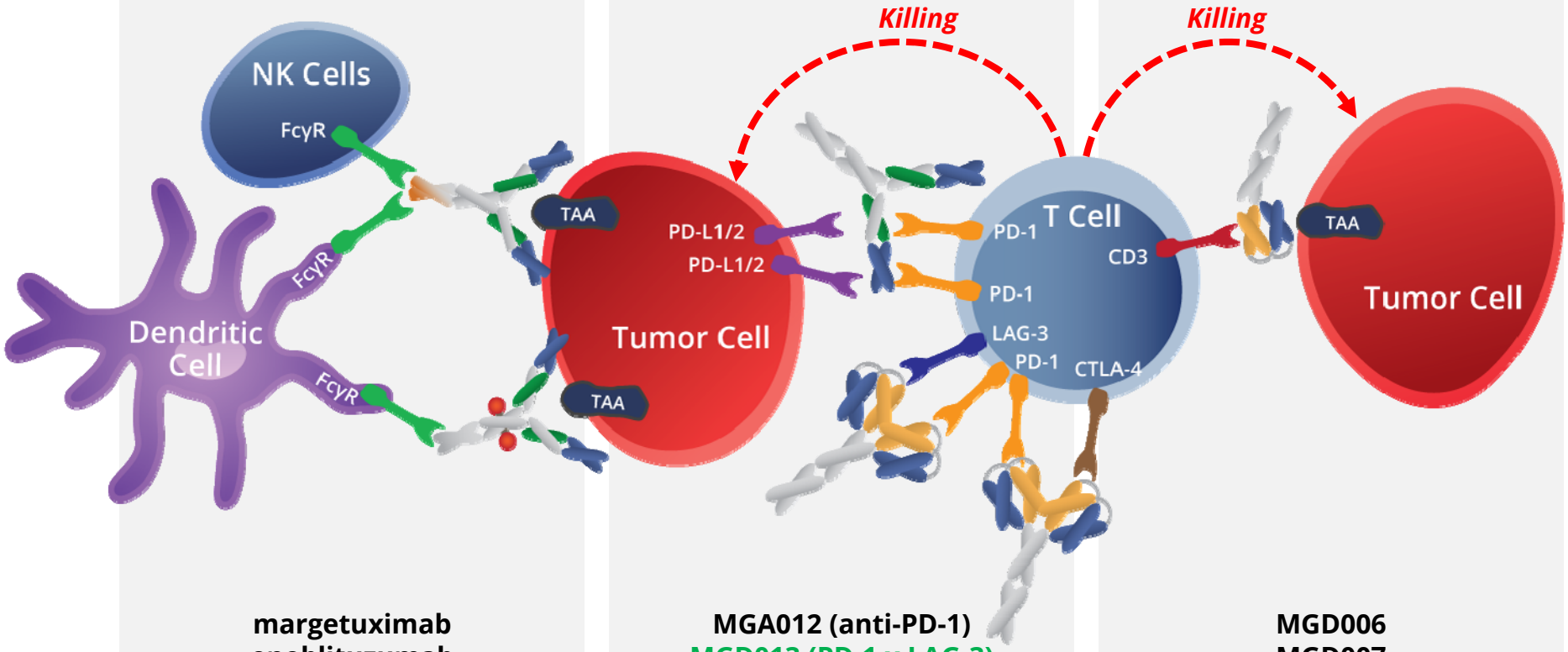
PBMCs were stimulated with 0.5 ng/ml SEB for 48 hours, harvested, washed and re-plated in 96-well plates with fresh SEB and the indicated molecules for an additional 48 hours

Targeting Independent Pathways for Combinatorial Activity

Tumor Destruction and Antigen Presentation: Tumor-targeted Interventions

Restore T-Cell Function: Checkpoint Inhibition

Enhance T-cell Function: T-Cell Recruitment



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PD-1 x CTLA-4

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MGD007
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TAA: tumor-associated antigen

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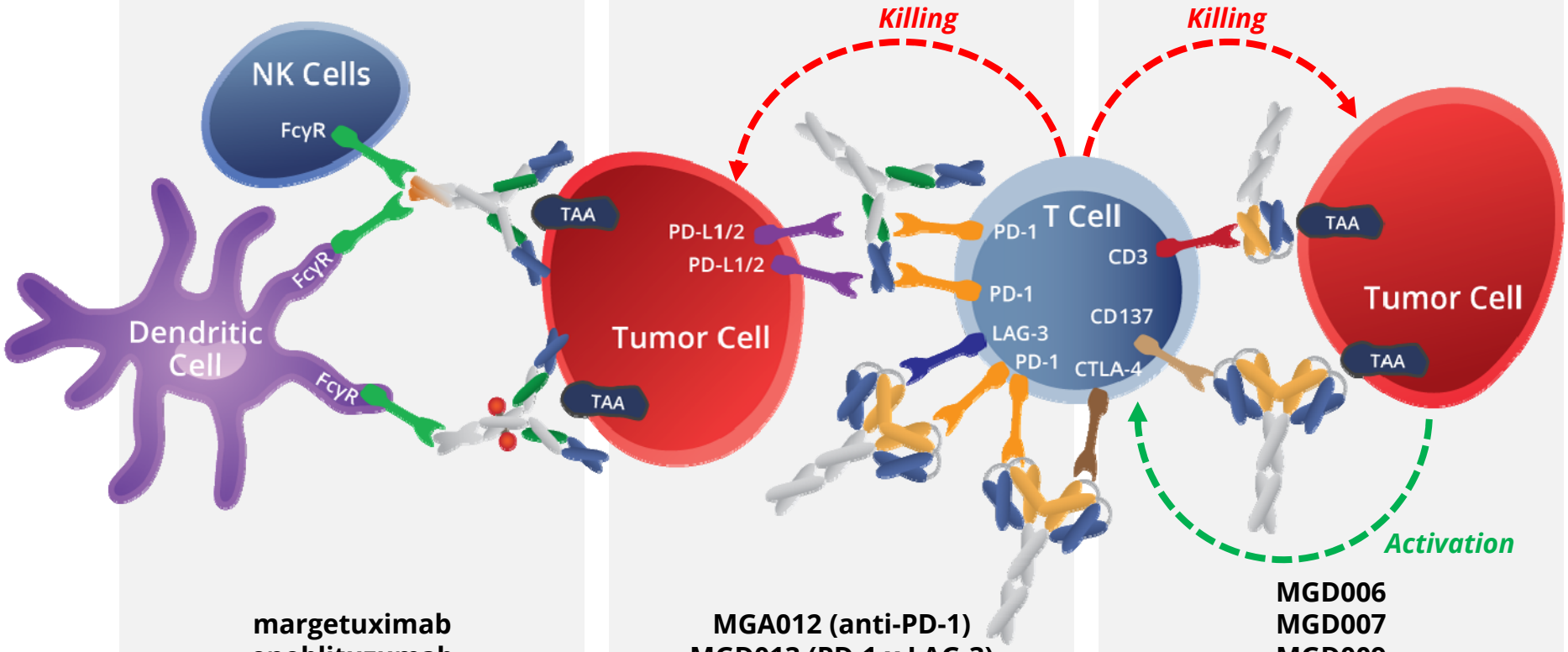

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HER2 x CD137

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Tumor-cell Anchored T-cell Co-stimulation

Limiting generalized activation by targeting tumor micro-environment

CD137 (4-1BB): Potent Inducible Co-stimulatory Molecule

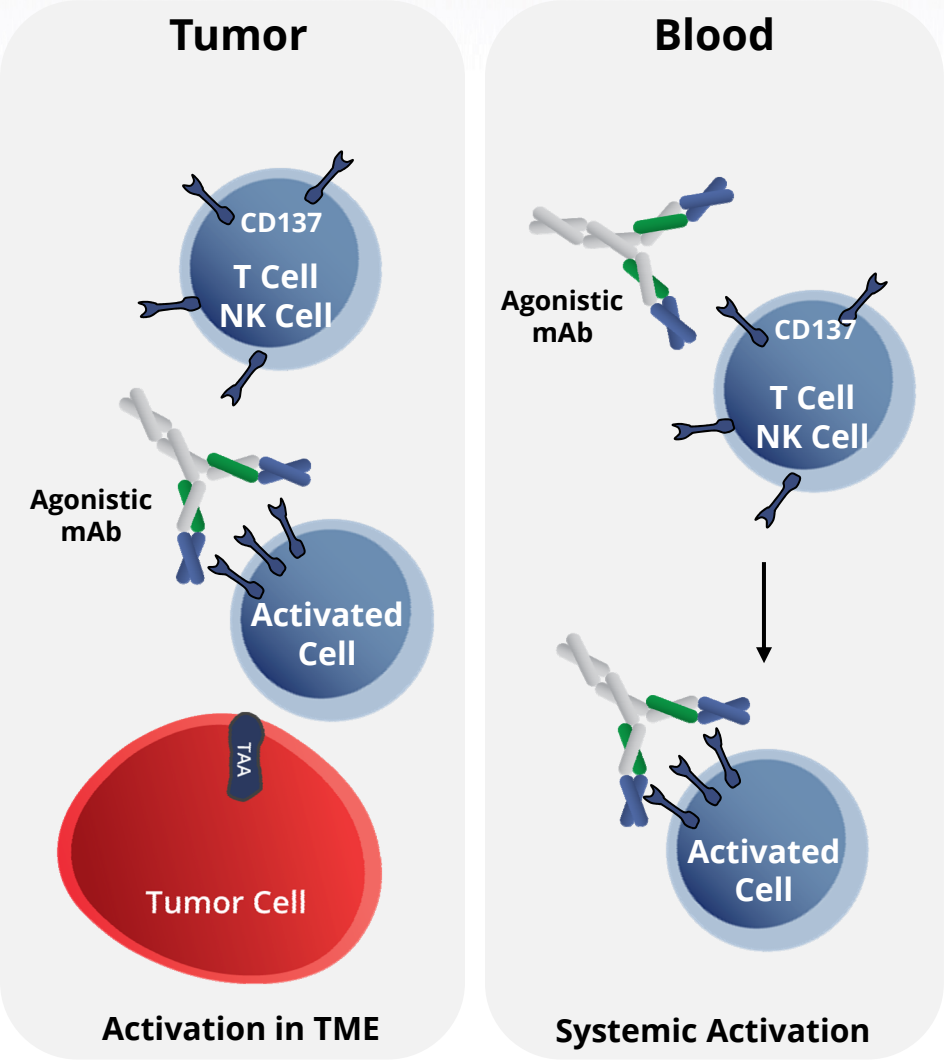
- Expressed upon activation of CD8, CD4, NK & dendritic cells
  - Potent co-stimulatory signal engineered in certain CAR-T cells
- Expressed by immune cells and vascular endothelium in tumor
  - **Induced expression on TILs** following MHC-peptide/TCR engagement
  - **Induced expression on NK cells** by mAb-opsonized tumor cells
- CD137L expressed by fraction of tumors, but insufficient to fully activate

CD137 activation results in:

- ↑ Endothelial adhesion molecules and chemokines
  - ↑ CD8 homing
- ↑ Immune-cell proliferation and anti-tumor cytolytic activity
- Countering of immune cell exhaustion and apoptosis
  - Synergy with adoptive T-cell therapy, anti-PD-1 or anti-CTLA-4 in mouse models
- ↑ ADCC by NK cells:
  - Synergy with rituximab, trastuzumab or cetuximab in mouse models

Agonistic anti-CD137 mAbs in the Clinic

- Urelumab (BMS, huIgG4)
- PF-05082566 (Pfizer, huIgG2)
- Single agent activity against melanoma and lymphoma in Ph. 1/2
- Urelumab associated with dose-dependent hepatitis (some fatal):
  - Passive accumulation in liver
  - Inflammation via liver-resident CD137+ cells (unidentified), enhanced by FcyRs



TAA: tumor-associated antigen

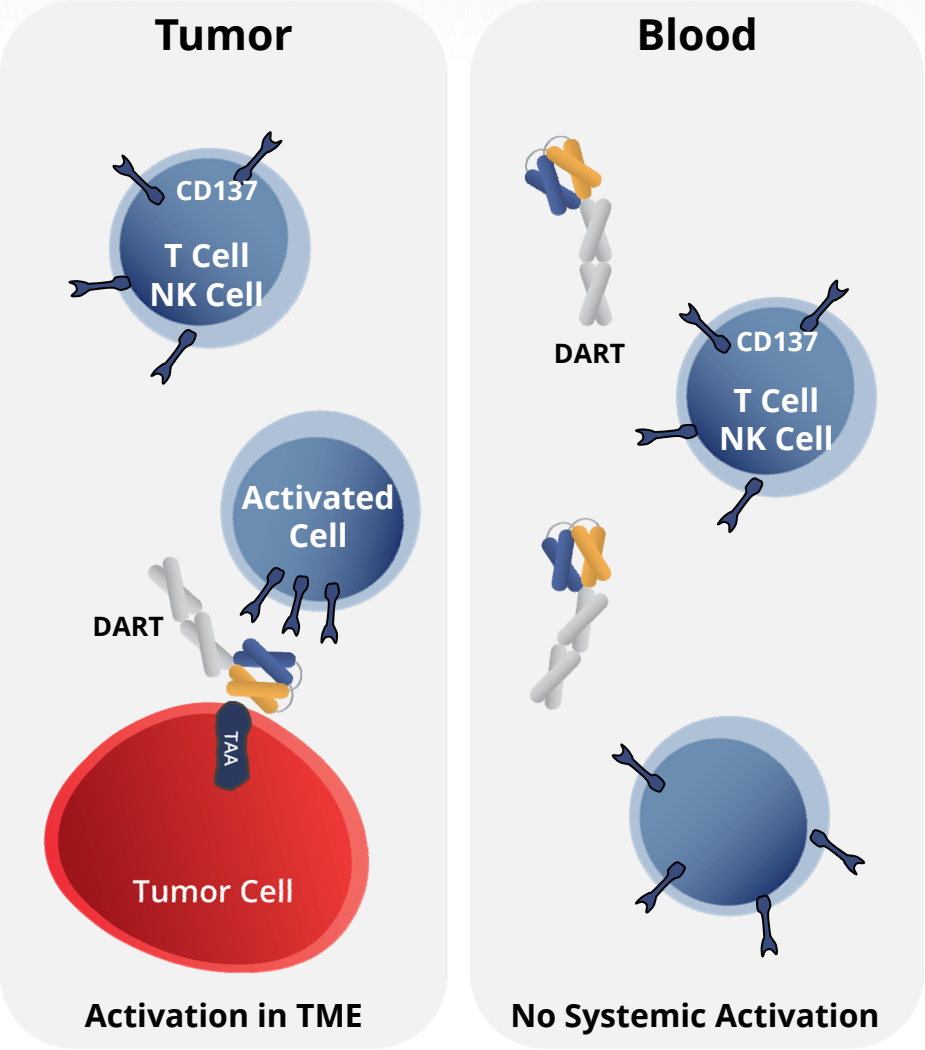
CD137-based DART: Building Tumor-anchored Activator

Challenge:

- Direct CD137 activation within tumor microenvironment
- Limit systemic effects of CD137 therapy

Solution:

- TAA x CD137 DART molecules

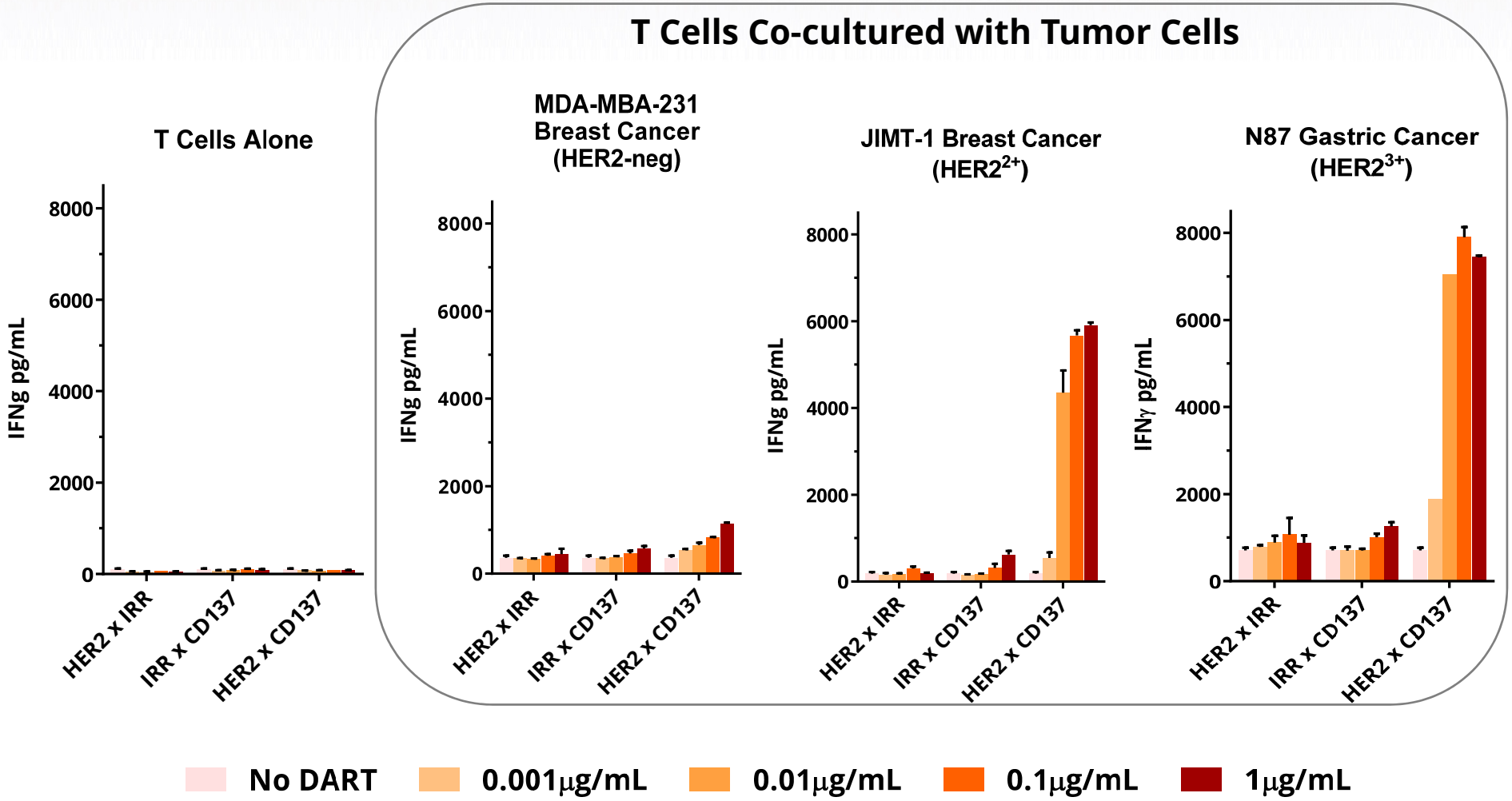


TAA: tumor-associated antigen

HER2 x CD137 DART Program

Candidates	• Anti-CD137 x humanized anti-HER2 – CD137: **agonistic upon secondary clustering** – HER2: **epitope independent of margetuximab**, trastuzumab & pertuzumab
Constructs	• Fc-bearing DART – *Null* Fc domain to eliminate FcR-mediated clustering
Preferred Profile	• Tumor antigen-dependent CD137-mediated agonistic activity – No systemic T-cell or NK-cell activation – Increased therapeutic window
Indications	• $HER2^{+}$ amplified/non-amplified solid tumors • Combination therapy for solid tumors – Margetuximab, trastuzumab, pertuzumab – Checkpoint inhibitors
Development	• Candidate selection ongoing
Partner	• MacroGenics retains global rights

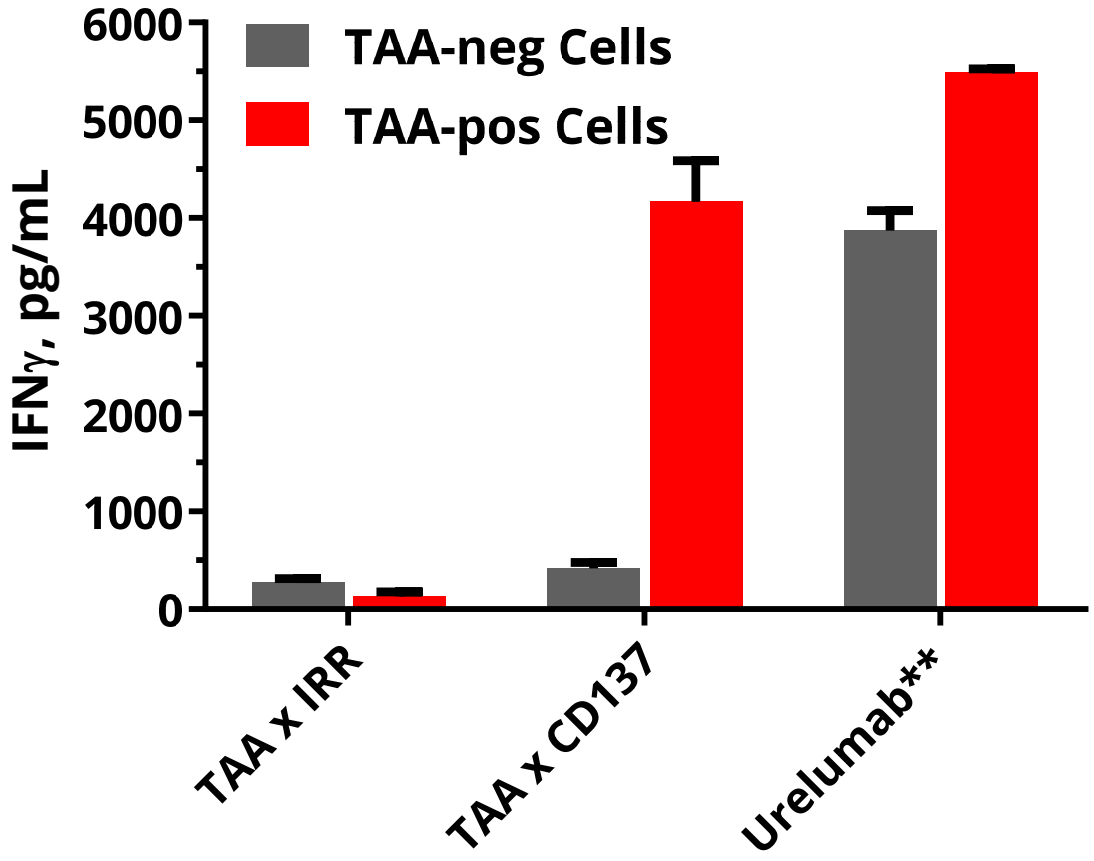
HER2 x CD137 DART: HER2-dependent T-cell Activation



T cells stimulated with aCD3 Beads (Beads/T = 1:1) ± HER2 x aCD-137 DARTs ± Tumor cells

DART Format: Plug-and-play for Additional Cancer Targets

TAA x CD137 DART: Co-stimulation in presence of TAA-positive cells*



T cells stimulated with CD3 beads (beads : T cells = 1:1) ±0.1ug/ml DARTs or mAb
* TAA: tumor-associated antigen (undisclosed)
** Replica of urelumab generated at MacroGenics

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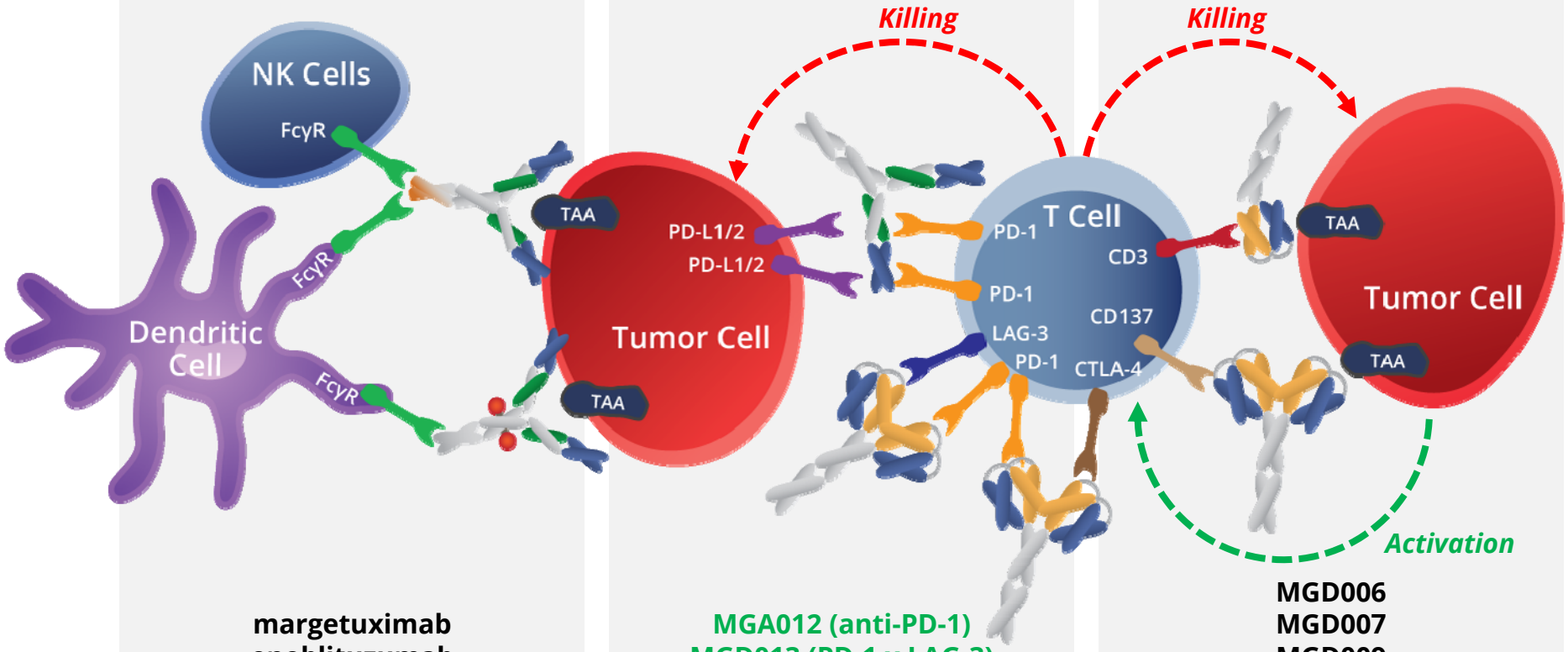

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Wrap-up / Q&A
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Balanced Portfolio Across Novel and Validated Targets

- Three core franchises
  - HER2 (validated target)
  - B7-H3 (novel)
  - PD-1 (emerging backbone)
- Utilization of core technology platforms
  - Target discovery
  - mAb / Fc engineering
  - DART / TRIDENT multispecifics
- Fully-integrated drug development operations
  - Rapid throughput with >1 IND annually (6 INDs in last 3 years)
- Leveraging partnerships to secure non-dilutive capital, broaden portfolio and access external expertise

Breakthrough Biologics, Life-Changing Medicines®

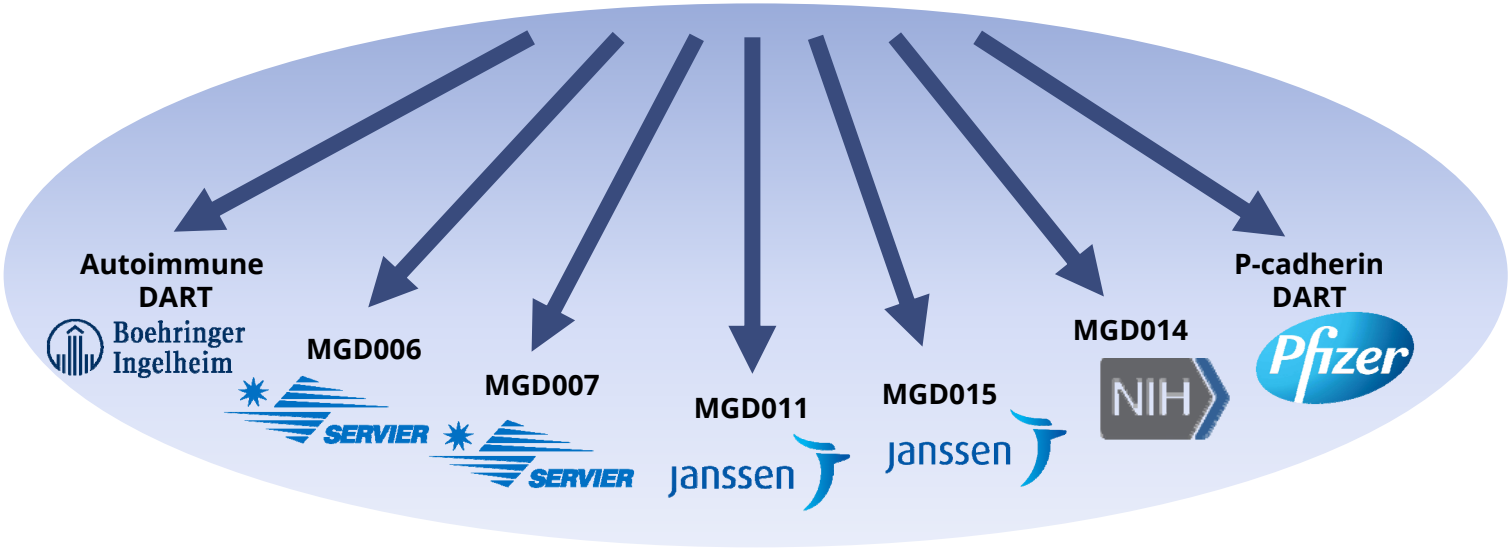
Extending Our Portfolio Through Collaborations

Wholly Owned

- **HER2 Franchise**
- **B7-H3 Franchise**
- **PD-1 Franchise**

Partnerships Enable Additional Shots on Goal

Partner Portfolio



Key Upcoming Milestones

2017

- **Margetuximab**
  - Complete enrollment in Phase 1b/2 gastric trial
- **Enoblituzumab**
  - Complete enrollment in new expansion cohorts for bladder and prostate cancer
  - Define future development plans based on monotherapy and combination study results
- **Oncology DART Portfolio** (MGD006, MGD007, MGD009)
  - Establish dose and schedule, and define future development strategy based on results
  - Initiate expansion cohorts
- **MGD010**
  - Report updated Phase 1 study data (Hepatitis A cohort)
- **MGA012**
  - Define recommended dose and schedule
  - Initiate first combination study
- **Preclinical Pipeline**
  - File IND for MGD013 (PD-1 x LAG-3 DART)
  - File IND for MGD014 (HIV DART)

2018

- **Margetuximab**
  - Complete enrollment of SOPHIA study
- **Preclinical Pipeline**
  - File IND for MGC018 (anti-B7-H3 ADC)

Our Pipeline of Product Candidates

Nine clinical-stage programs with ≥ 1 new INDs annually

Program (Target)	Indication	Pre-IND	Phase 1	Phase 2	Phase 3	Partner	Our Commercial Rights
ONCOLOGY							
margetuximab (HER2)	Breast (HER2+) "SOPHIA"					Green Cross	Worldwide, excl. S. Korea
	Gastric (+pembrolizumab)						
enoblituzumab (B7-H3)	Solid Tumors (mono.)					—	Worldwide
	Solid Tumors (+ipi.)						
	Solid Tumors (+pembro.)						
MGD006 (CD123 x CD3)	AML/MDS					Servier	North America, Japan, South Korea, India
MGD007 (gpA33 x CD3)	Colorectal						
MGD009 (B7-H3 x CD3)	Solid Tumors					—	Worldwide
MGD011 (CD19 x CD3)	B-cell Malignancies					Janssen	U.S. Co-promote*
▶ **MGA012** (PD-1)	Solid Tumors					—	Worldwide
MGD013 (PD-1 x LAG-3)	Solid Tumors					—	Worldwide
▶ **MGC018** (B7-H3 ADC)	Solid Tumors					—	Worldwide
▶ (PD-1 x CTLA-4)	Solid Tumors					—	Worldwide
▶ (CD137 x HER2)	HER2+ Solid Tumors					—	Worldwide
AUTOIMMUNE & INFECTIOUS DISEASES							
teplizumab (CD3)	Type 1 Diabetes Prev.					NIDDK/NIH	Worldwide
MGD010 (CD32B x CD79B)	Autoimmune Disorders					—	Worldwide
MGD014 (HIV x CD3)	HIV					NIAID/NIH	Worldwide

** MacroGenics has option to fund late-stage development in exchange for U.S. and Canada profit share.*

"MGD" = DART | "MGA" = Antibody | "MGC" = ADC