

Antibodies by Design:

XmAb[®] Antibody Therapeutics

Analyst Day Welcome

June 28, 2016

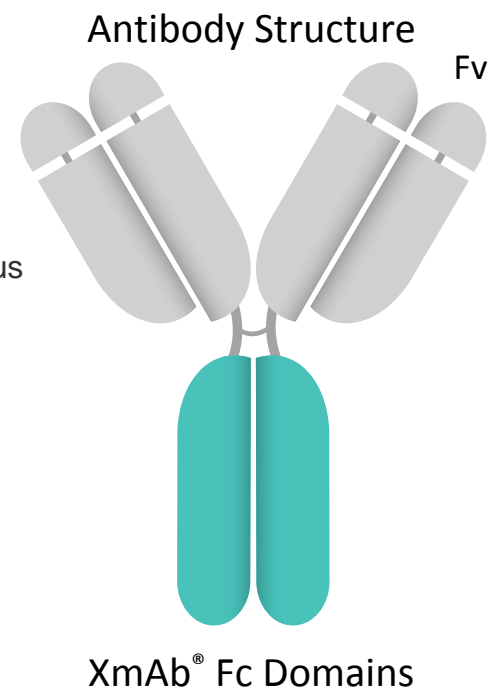


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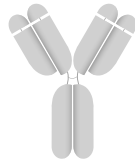
Xencor: Engineering Antibody Immune Functions To Make Better Drugs

- XmAb® Fc domains: small changes, big functional impacts
 - Augments native immune functions, preserves half-life, stability and production
 - Over 100 issued and over 150 pending patents worldwide
- Lead internal programs in clinical development
 - **XmAb5871** in Phase 2 in IgG4-Related Disease and Systemic Lupus Erythematosus
 - **XmAb7195** completing Phase 1a, in development for allergic disease
- Expansive bispecific oncology pipeline advancing
 - Planning to initiate clinical trials of **XmAb14045** and **XmAb13676** in 2016
 - Additional bispecific programs planned to start clinical trials in 2017
 - Amgen advancing six XmAb programs in oncology and inflammation
 - \$45M upfront, \$1.7B potential milestones
- Nine XmAb clinical programs ongoing internally or with partners

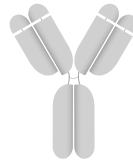


XmAb[®] Fc Domains Augment Natural Antibody Functions

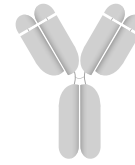
Natural Fc Function



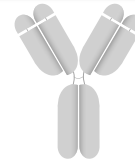
Immune regulation
Antigen clearance



Cytotoxicity
(immune cell)



Circulating
half-life



Stable homodimer
structure

Fc Receptor

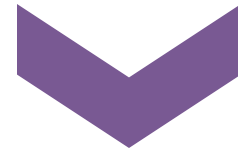
FcγRIIb

FcγRIIa, FcγRIIIa

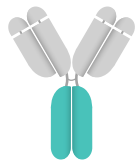
FcRn

N/A

Fc Domain Redesigns

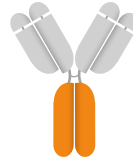


XmAb Enhanced Function



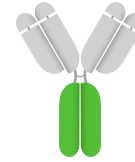
Immune Inhibitor Domain

Immune inhibition
Rapid clearance



Cytotoxic Domain

Enhanced cytotoxicity
(immune cell)



Xtend Domain

Prolonged
half-life



Bispecific Domain

Stable heterodimer
structure

Additional Fc domains: stability, complement activation

99.5% identical to natural antibody
Plug-and-play substitution into any antibody

Agenda

Welcome

Bassil Dahiyat, Ph.D., CEO

XmAb®5871 and XmAb®7195 Development Programs

Paul Foster, M.D., Chief Medical Officer

XmAb® Anti-CD3 Bispecific Platform and Oncology Pipeline Overview

*John Desjarlais, Ph.D., Chief Scientific Officer
Paul Foster, M.D., Chief Medical Officer*

XmAb® Checkpoint Inhibitor Bispecific Platform and Oncology Pipeline Overview

*John Desjarlais, Ph.D., Chief Scientific Officer
Paul Foster, M.D., Chief Medical Officer*

Hypoxia drives tumor immune suppression and immunotherapy resistance

*Michael Curran, Ph.D.
Assistant Professor, Department of Immunology, The University of Texas MD Anderson Cancer Center
Member, Graduate Faculty, The University of Texas Graduate School of Biomedical Science*

Clinical Trial Landscape in NHL: Focus on Immunotherapy

*Paul Hamlin, M.D.
Chief, Basking Ridge Medical Oncology Service, Memorial Sloan Kettering Cancer Center*

Closing

Bassil Dahiyat, Ph.D., CEO

Agenda Highlights

- Novartis collaboration
 - Ex-US commercial rights to XmAb14045 and XmAb13676 licensed, 50/50 development cost share worldwide
 - Access to bispecific platform for 4 Novartis programs; Fc platform access
 - \$150M upfront, \$2.41B milestones, royalties
- XmAb5871
 - Review of Phase 2 trials design in IgG4-Related Disease and Systemic Lupus Erythematosus
 - Review of mechanism and Phase 1 and 2a clinical data
 - Plan for XmAb5871 subcutaneous clinical trial in 3Q2016
- XmAb7195
 - Review of Phase 1a clinical data
 - Plan for XmAb7195 subcutaneous clinical trial in 4Q2016
- XmAb oncology bispecifics new programs
 - XmAb18087: SSTR2 x CD3 for neuroendocrine tumors, IND expected 2017
 - XmAb20717: PD-1 x CTLA-4 dual checkpoint inhibitor, IND expected 2017

Novartis Collaboration for XmAb14045 and XmAb13676 Boosts Development Resources and Retains US Commercial Rights



















Links Novartis' leadership in development and commercialization of oncology drugs with Xencor's XmAb Bispecific programs

- Novartis receives ex – US commercial rights to XmAb14045 and XmAb13676
 - Low double-digit royalties on ex-US sales
- Xencor retains all U.S. commercial rights to XmAb14045 and XmAb13676
- Worldwide 50/50 development collaboration and cost share
- Research collaboration for XmAb Bispecific Technology in 4 Novartis programs
 - Novartis starting antibodies plugged into XmAb bispecific constructs, Xencor provides molecular engineering; mid single-digit royalties
 - Xencor has opt-in right to one Novartis program for US profit and cost share, co-detail
- Non-exclusive access to Xencor Fc Technologies for 10 programs

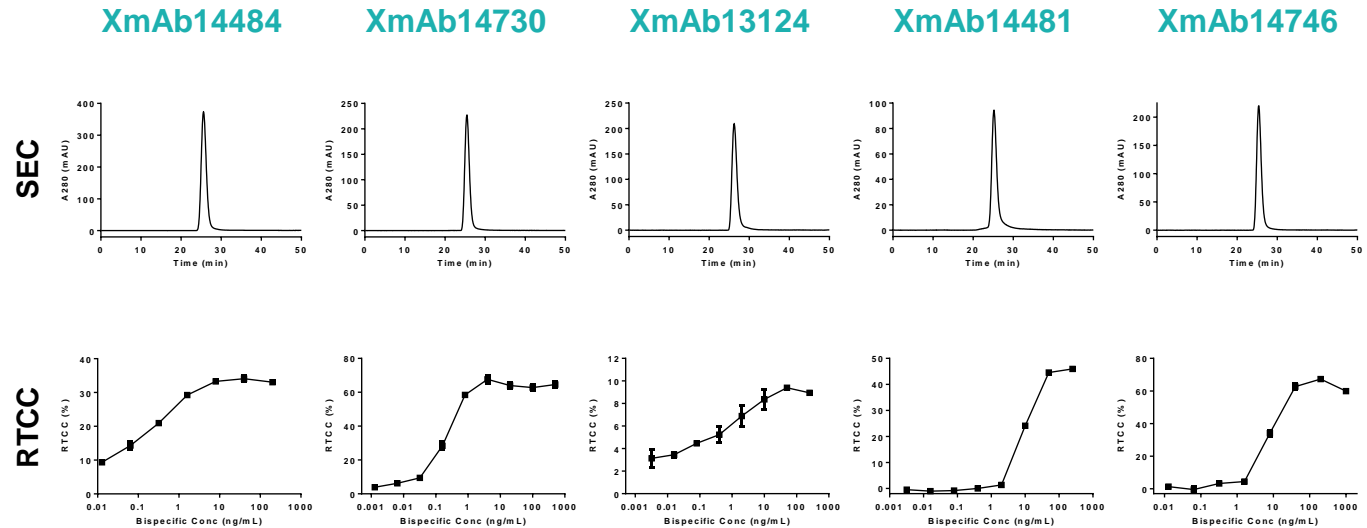
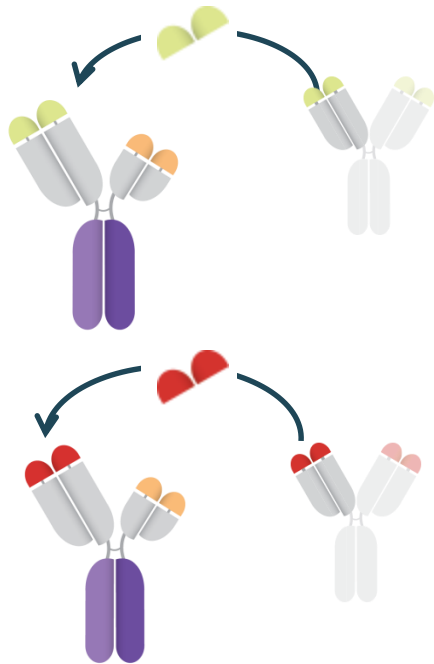
\$150M upfront, \$2.4B potential milestones, royalties

Development Pipeline Focused on Immune Inhibitor and Bispecific Fc Domains

| Program (Target) | Fc Domain | Primary Indication | Discovery Lead | Preclinical | Phase 1 | Phase 2 | Commercial Rights |
|-------------------------------------|---------------------|----------------------------|--|-------------|---------|---------|---|
| XmAb5871 (CD19) | Immune Inhibitor | IgG4-RD SLE |  | | | |  |
| XmAb7195 (IgE) | Immune Inhibitor | Asthma/ allergy |  | | | |  |
| XmAb5574/MOR208 (CD19) | Cytotoxic | CLL/NHL/ ALL |  | | | |  |
| XmAb14045 (CD123 x CD3) | Bispecific | AML |  | | | |  * |
| XmAb13676 (CD20 x CD3) | Bispecific | B-cell malignancy |  | | | |  * |
| XmAb13551 (CD38 x CD3) | Bispecific | Myeloma |  | | | |  |
| XmAb18087 (SSTR2 x CD3) | Bispecific | Neuroendo- crine tumors |  | | | |  |
| XmAb20717 (PD-1 x CTLA-4) | Bispecific Xtend | Oncology |  | | | |  |

* Novartis licensed ex-US commercial rights, worldwide co-development

Plug-and-play Fc Domain Enables Rapid Pipeline Generation and Prototyping

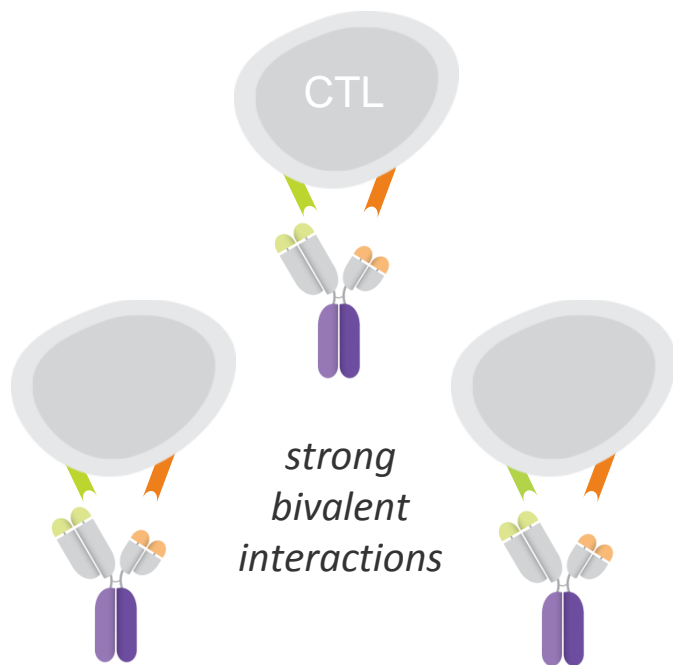


- Portfolio of CD3 bispecific molecules generated and ready for development
- New immuno-oncology programs rapidly prototype different target combinations

Xencor Checkpoint Bispecifics: Selective Tumor T-cell Targeting through Bispecific Avidity

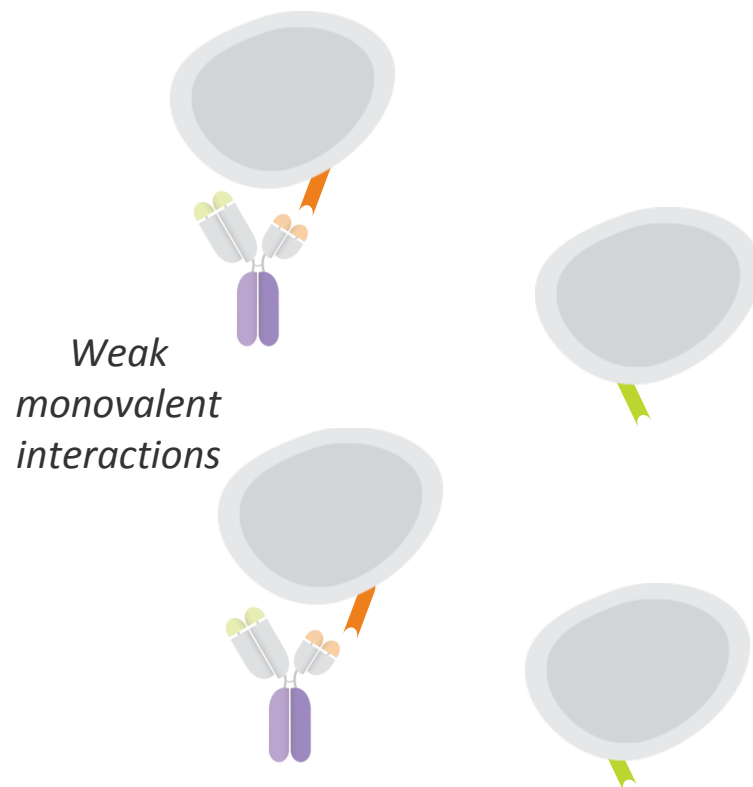
Tumor Environment:

- Tumor infiltrating lymphocytes (TILs) coexpress multiple checkpoints (Matsuzaki 2010, Fourcade 2012, Gros 2014)
- Bivalent binding increases avidity



















Enhance anti-tumor activity

Periphery



Avoid peripheral toxicity

Xencor's Growing Bispecific Oncology Pipeline to Enter Clinical Trials in 2016

| Program (Target) | Fc Domain | Primary Indication | Discovery Lead | Preclinical | Phase 1 | Commercial Rights |
|---|----------------------|--------------------|--|-------------|-----------|---|
| XmAb14045 (CD123 x CD3) | Bispecific | AML |  | | (2016) |  xencor* |
| XmAb13676 (CD20 x CD3) | Bispecific | B-cell cancer |  | | (2016/17) |  xencor* |
| XmAb13551 (CD38 x CD3) | Bispecific | Myeloma |  | | |  AMGEN |
| XmAb18087 (SSTR2 x CD3) | Bispecific | Oncology |  | | |  xencor |
| XmAb20717 (PD1 x CTLA4) | Bispecific/ Xtend | Oncology |  | | |  xencor |
| Undisclosed CI x CI | Bispecific | Oncology |  | | |  xencor |
| Undisclosed (x CD3) | Bispecific | Oncology |  | | |  xencor |
| Undisclosed Immune Modulation | Bispecific | Oncology |  | | |  xencor |

* Novartis licensed ex-US commercial rights, worldwide co-development

- CD3 bispecifics to target T cells to tumors, entering clinic in 2016
- SSTR2 x CD3 IND mid-2017, followed by PD1 x CTLA4
- Scalable platform process for GMP manufacturing developed

XmAb[®]5871

Development Program

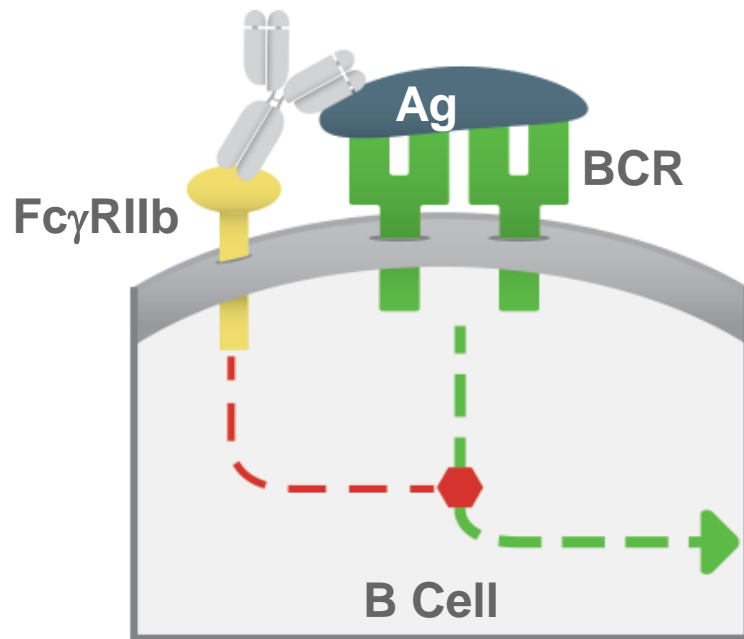
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XmAb5871 Enhances Natural Regulatory Role of FcγRIIb

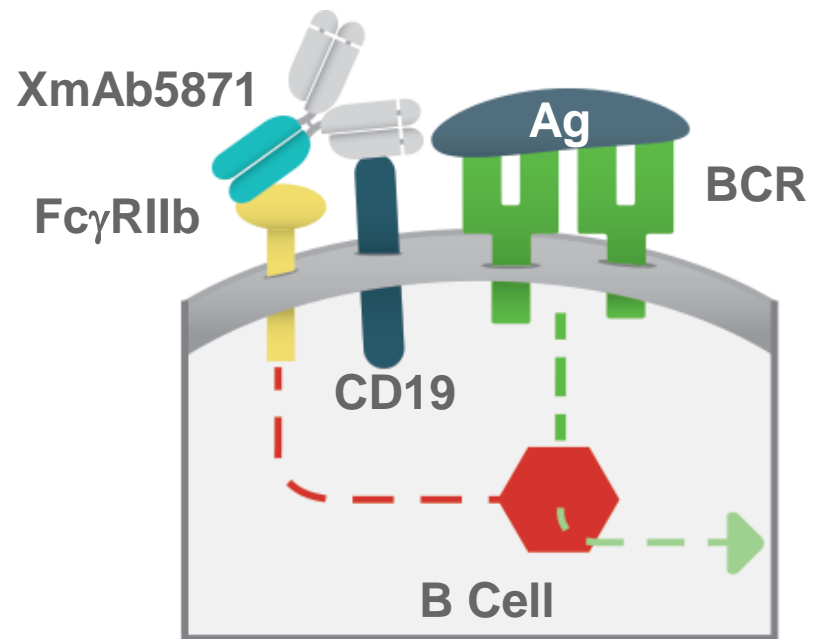
Natural:

Ag + αAg Immune Complex



XmAb5871:

anti-CD19 with Immune Inhibitor Fc domain



- FcγRIIb inhibitory activity requires bridging to specific co-targets
- Inhibits many activation pathways in both healthy and diseased B cells
- Potent suppression of B-cell responses without destroying B cells

XmAb5871 Clinical Development - Completed Studies

- Phase 1a FIH SAD study in HV completed in 4Q 2012
- Phase 1b/2a study in patients with RA on stable non-biologic disease modifying anti-rheumatic drug (DMARD) therapy in 3Q 2014

Design

- Multi-center, randomized, placebo controlled, double-blinded
- Phase 2a portion of trial (27 patients, 2:1 randomized)
- Six bi-weekly IV infusions, 10 mg/kg

Primary objective of safety and tolerability characterization:

- Generally well tolerated
- Two treatment related SAEs and two placebo treated subjects with SAEs

Secondary and exploratory efficacy objectives:

Phase 2a Disease Response Assessments at Week 13

| | DAS28 CRP* | ACR70 | ACR50 |
|-----------------|------------|-------|-------|
| XmAb5871 | 33% | 20% | 40% |
| Placebo | 0% | 0% | 13% |

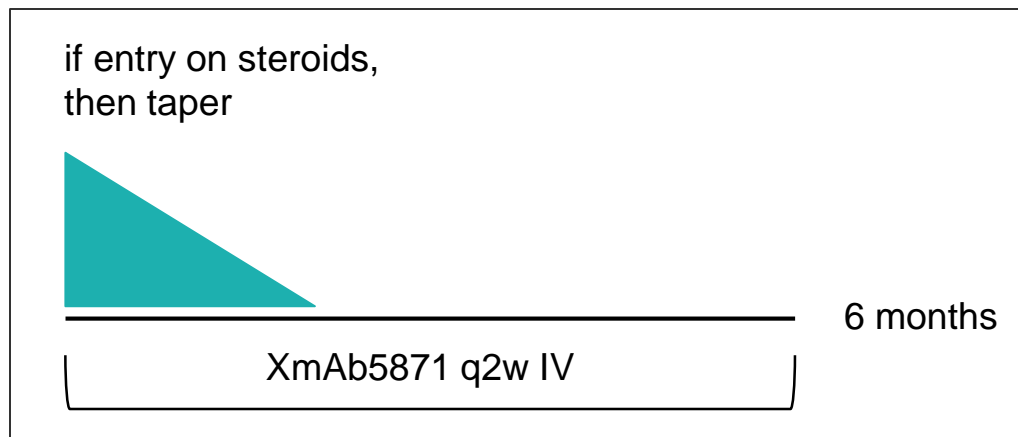
Phase 2a portion of trial: 23
evaluable patients: 15 XmAb5871
treated, 8 placebo treated
Protocol specified disease
response evaluation at Week 13

* Remission or low disease
activity

XmAb5871 Clinical Development - Ongoing Studies

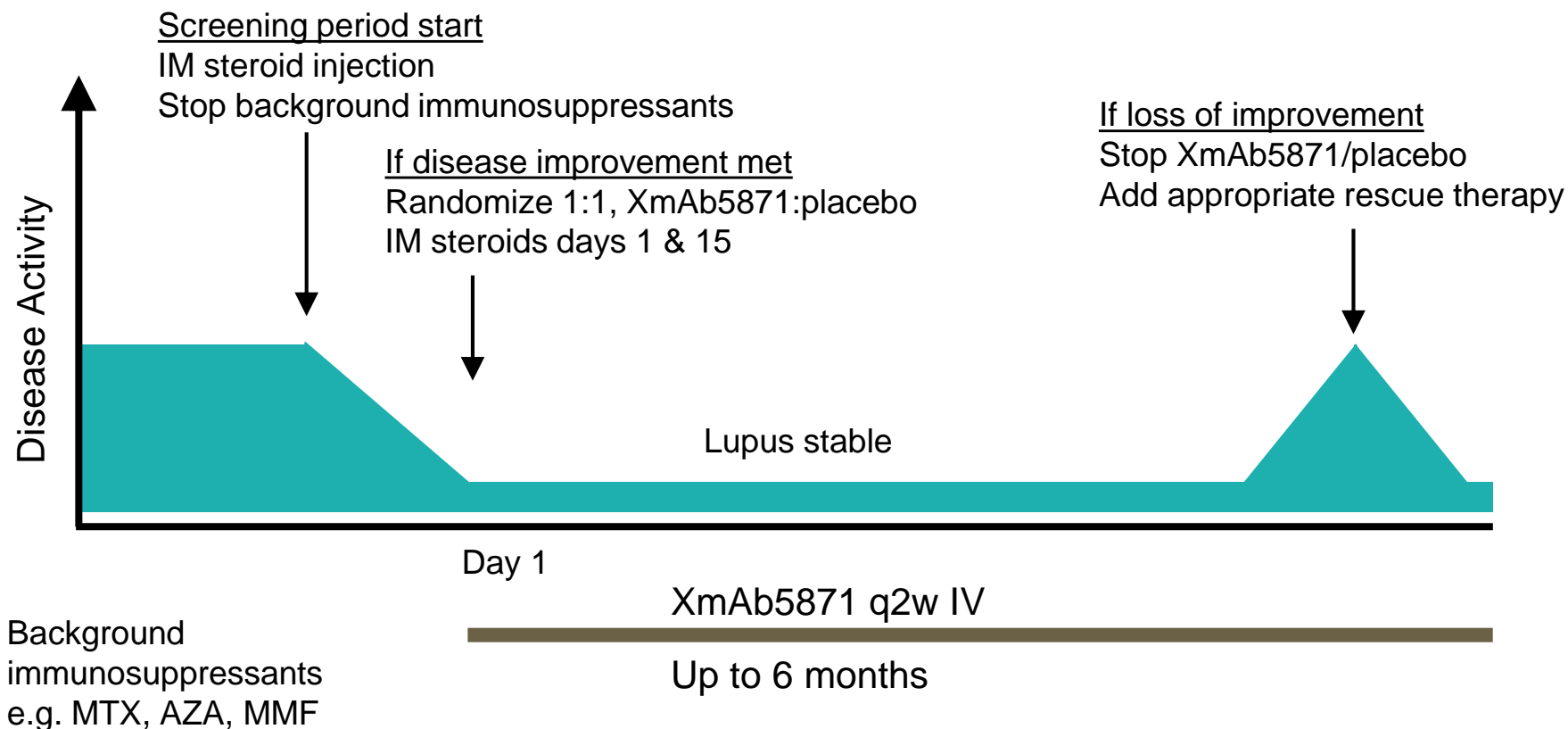
1. Phase 2 open-label, single-arm, single-site (MGH) pilot study in patients with active IgG4-RD (IgG4-RD RI ≥ 3)
 - An immune-mediated condition responsible for fibro-inflammatory lesions that can lead to irreversible damage to virtually any organ
 - No approved therapies; glucocorticosteroids are standard of care
2. Phase 2 randomized double-blind, placebo-controlled study in patients with moderate to severe, non-organ threatening, SLE

IgG4-RD Pilot Trial Design To Characterize Activity Of XmAb5871 at Reducing IgG4-RD RI



- **Dose schedule:** IV infusion every 2 weeks for 12 doses (6 months)
- **Number:** Up to 15 patients
- **Primary endpoint:** the proportion of patients at 6 months with an improvement of disease activity score as defined by a decrease of IgG4-RD Responder Index of ≥ 2 points from Day 1 pre-dose disease activity score
- Based on design of an open label study of rituximab in IgG4-RD (Carruthers Ann Rheum Dis, 2015)
- Study is enrolling with 1st patient dosed Mar2016. First possible preliminary data report at a medical meeting in 4Q2016

XmAb5871 SLE Phase 2 Study Design



Randomized, double blinded, placebo controlled

N = ~90 patients, ~20 US sites

Primary objective: ability of XmAb5871 to maintain SLE disease activity improvement

First patient enrolled Mar2016

XmAb5871 Clinical Development – Planned Studies

XmAb5871-25: Subcutaneous Bioavailability Study

- **Design:** Phase 1, single-site, open-label, parallel-group, multiple-dose comparison of relative bioavailability and PK of XmAb5871 administered IV versus SC in healthy volunteers
- **Dose schedule:** Q 14 days X 3 doses
- **Number:** 50 subjects
- **Primary endpoint:** Relative bioavailability and PK of SC administration of XmAb5871
- **Expected start:** 3Q2016

XmAb[®]7195

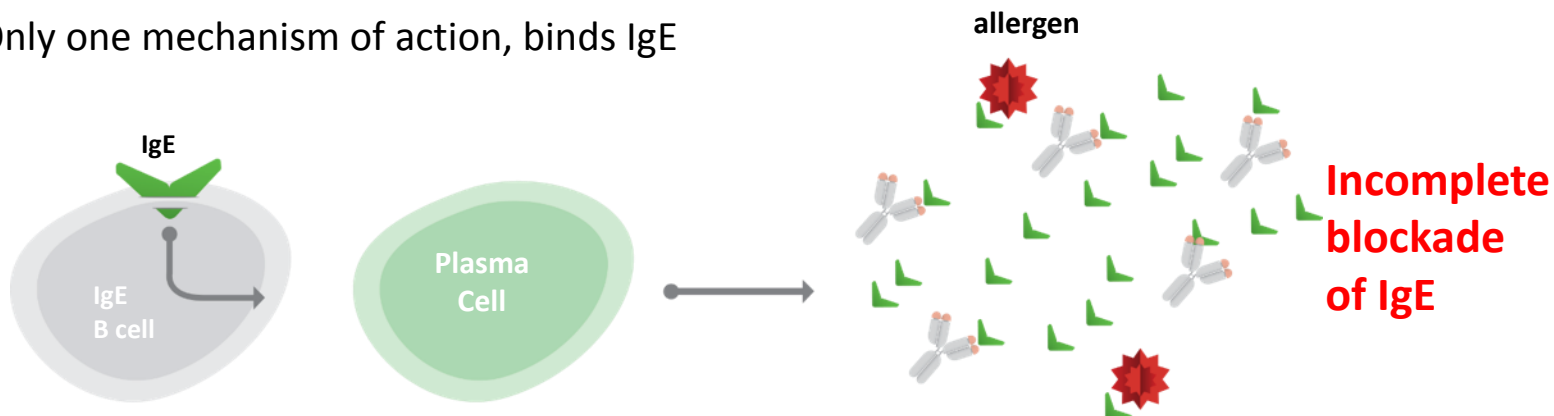
Development Program

June 28, 2016



XmAb7195 Multiple Mechanisms of Action

Xolair: Only one mechanism of action, binds IgE



XmAb7195

1

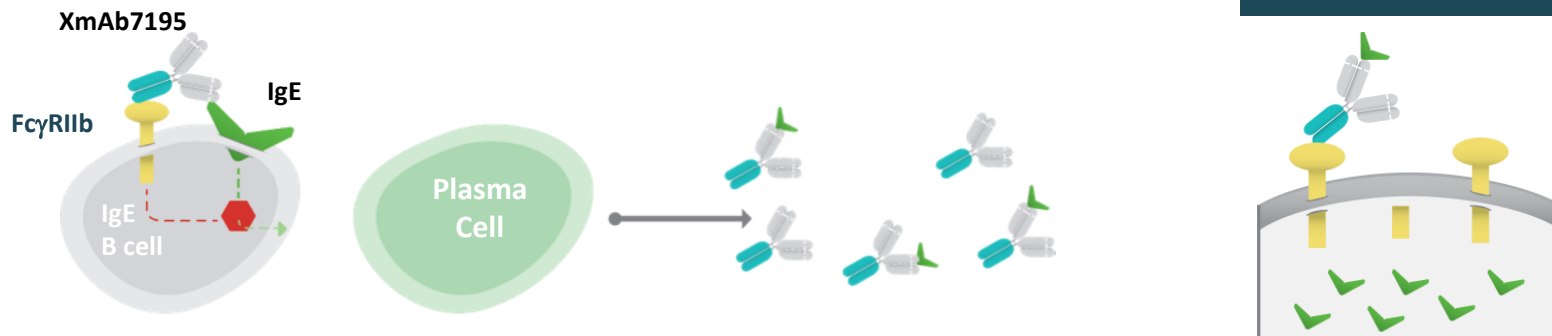
Reduces IgE Production

2

Binds IgE

3

Sends IgE to liver sinusoidal endothelial cells for destruction



XmAb7195 Clinical Development - Completed Studies

XmAb7195-01: Phase 1 FIH randomized, double-blind, placebo-controlled, ascending dose study of IV administered XmAb7195 in healthy subjects and subjects with a history of atopic disease with elevated serum IgE (300 – 3000 IU/mL inclusive) completed 3Q 2015

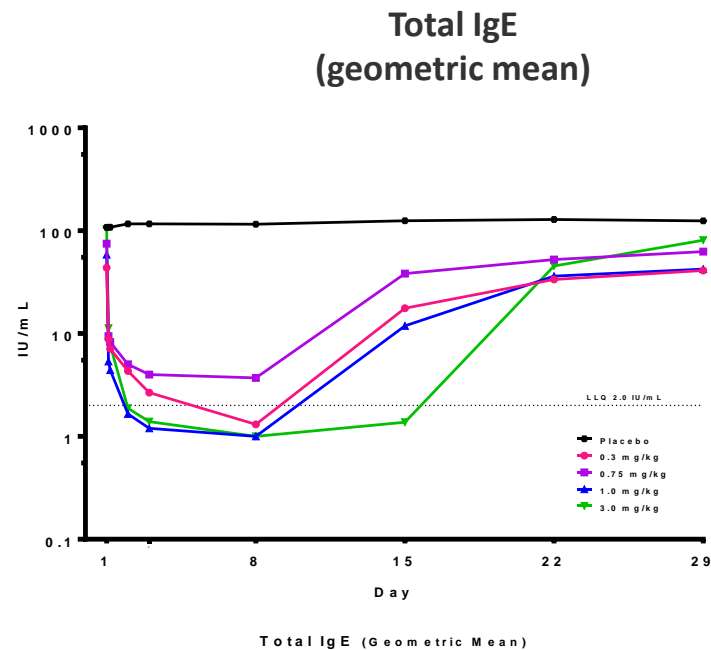
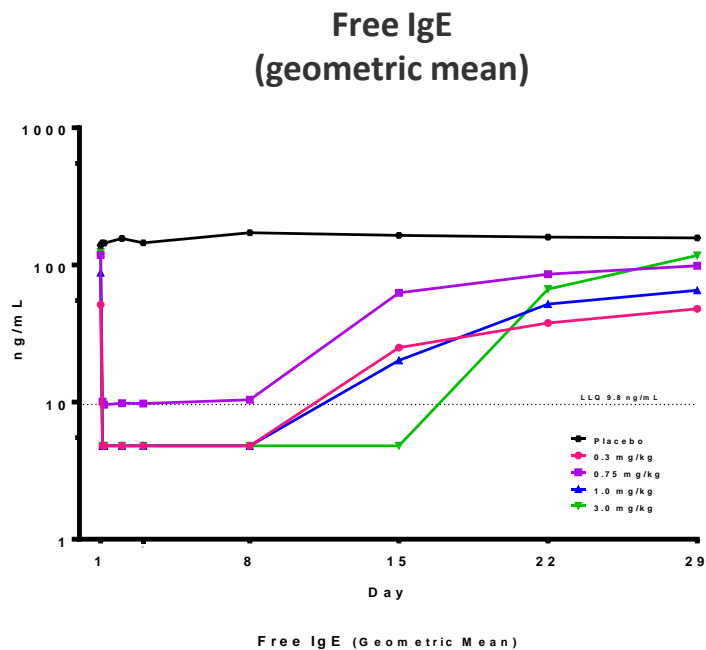
- **Design:** Single IV administration of XmAb7195 or placebo (0.3-3.0 mg/kg in HV; 0.6-2.0 mg/kg in atopic subjects with high IgE). Part 3: Two-dose sequential IV administration of XmAb7195 or placebo on Day 1 and Day 8 (0.3/0.3, 0.3/1.0 mg/kg)
- **Primary endpoint:** To determine the safety and tolerability profile following single-dose IV administration of XmAb7195 and after a priming IV dose followed by an escalating second IV dose of XmAb7195

XmAb7195-01 Results

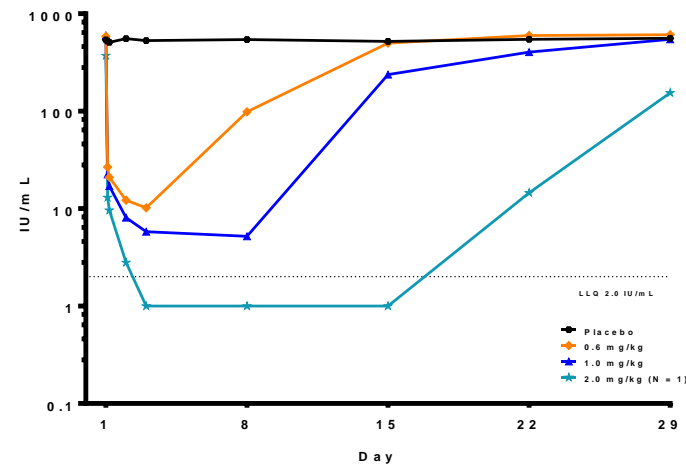
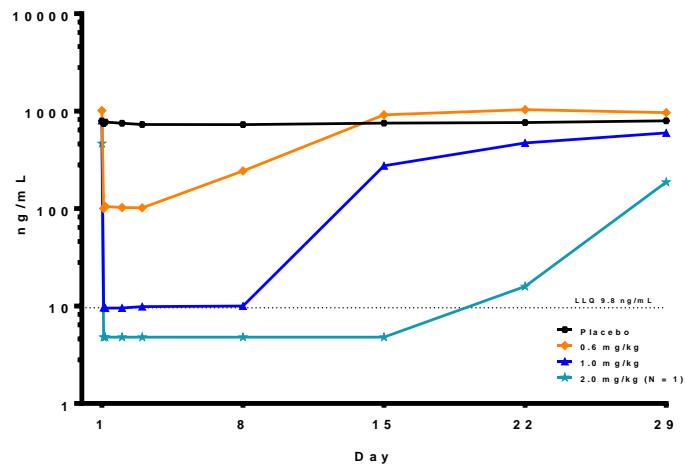
- XmAb7195 was generally well tolerated when administered as a single IV infusion with transient, asymptomatic thrombocytopenia occurring at doses ≥ 2.0 mg/kg
- One SAE
 - Bronchospasm was reported in an atopic subject with a history of perennial and seasonal allergies experienced severe bronchospasm 25 minutes after the start of the XmAb7195 infusion. The event responded quickly to discontinuation of the infusion and medical intervention
- XmAb7195 induced rapid and extensive depletion of serum free IgE, serum total IgE, basophil surface IgE and basophil Fc ϵ RI expression levels at all doses tested
- Across all dose levels tested, 93% of healthy adults and 75% of atopic subjects with predose total IgE of >300 IU/ml had reduction of free IgE levels to BLQ (<9.59 ng/ml) following a single dose of XmAb7195

XmAb7195 reduces free and total IgE in humans

Healthy Subjects



High IgE Subjects
(300 – 3000 IU/mL);



XmAb7195 Clinical Development – Planned Studies

XmAb7195-02: Subcutaneous Bioavailability/MAD Study

- **Design:** Phase 1, open-label, parallel-group, multiple-dose (4 doses) comparison of relative bioavailability and PK of XmAb7195 and a randomized (3:1) double-blinded, placebo-controlled, multiple ascending dose PK and safety study in healthy volunteers and individuals with atopic disease
- **Dose schedule:** Part A: XmAb7195 administration weekly for a total of 4 doses.
- **Number:** Approximately 62 subjects. 30 subjects in Part A; 32 subjects in Part B
- **Primary endpoint:** Relative bioavailability and PK of SC administration of XmAb7195
- **Anticipated start:** 3Q-4Q2016

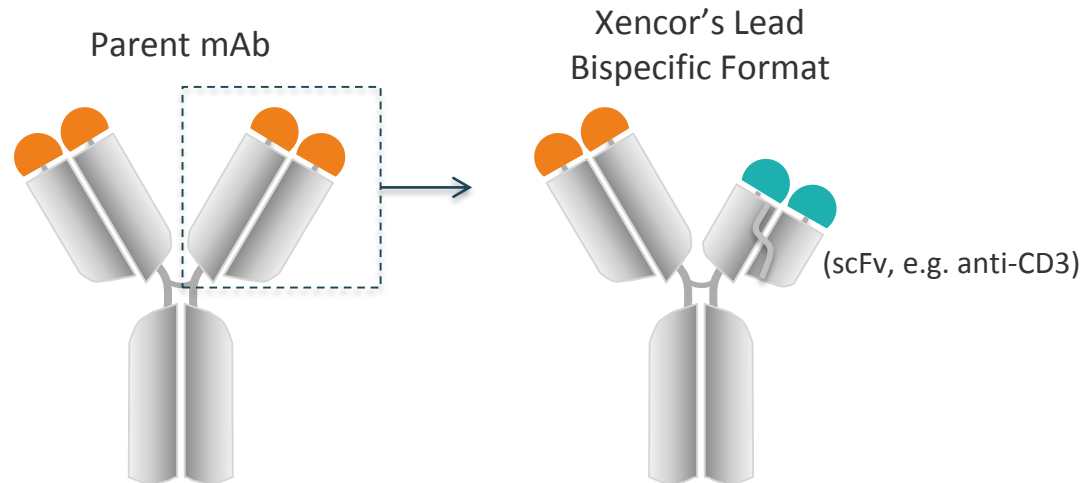
XmAb[®] Anti-CD3 Bispecific Platform and Oncology Pipeline Overview

June 28, 2016

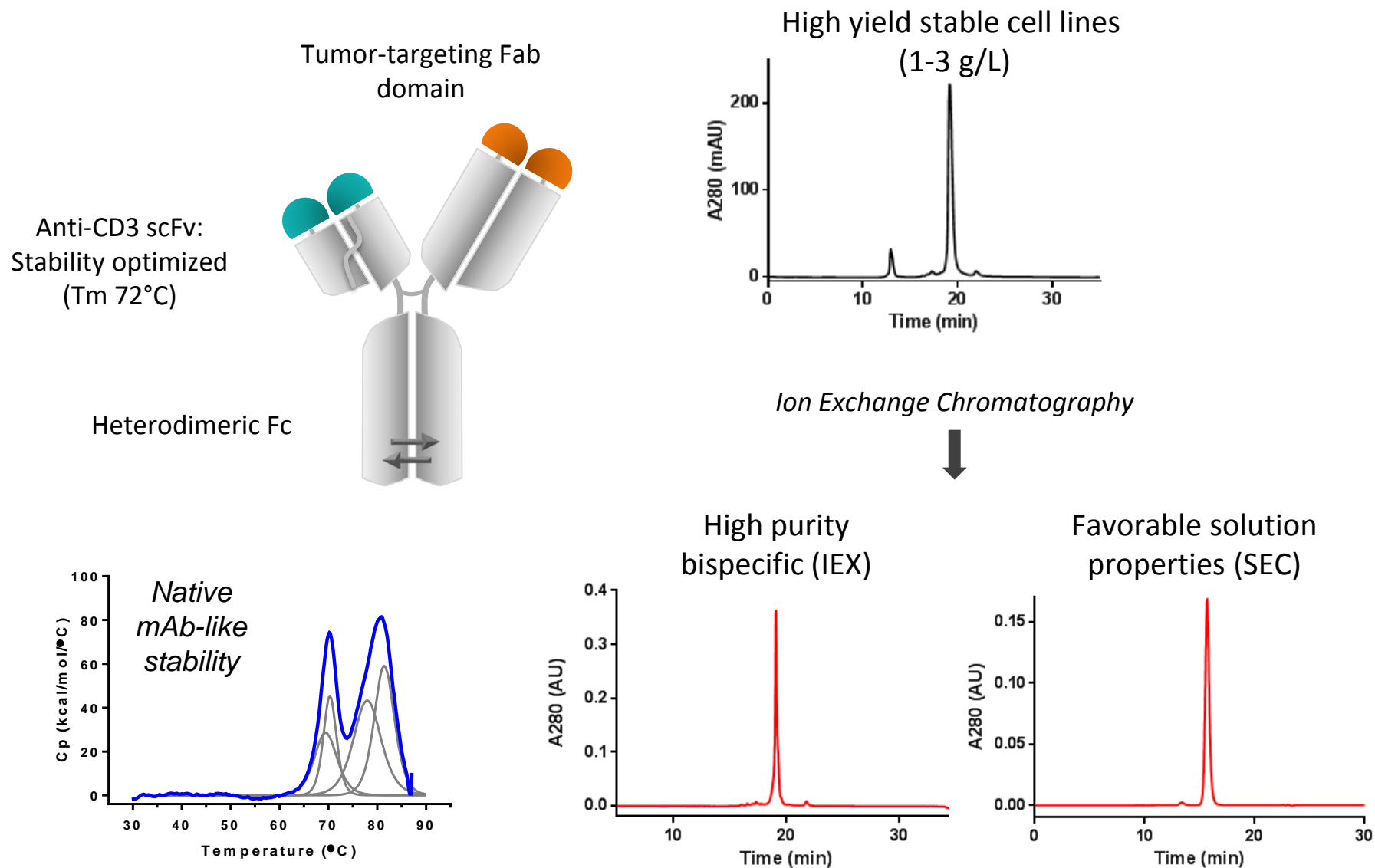


Bispecific antibodies in immuno-oncology

- Tremendous potential across wide range of diseases
- Historically plagued by difficult production
- Xencor bispecifics platform
 - Xencor's expertise in Fc engineering applied to enable facile production and long half-life
 - Plug-and-play format → rapid lead prototyping & lead generation
 - Potency tuning to maximize therapeutic index

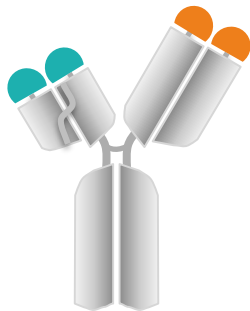


Plug-and-play scFv-Fab format is stable and well-behaved, and easily purified

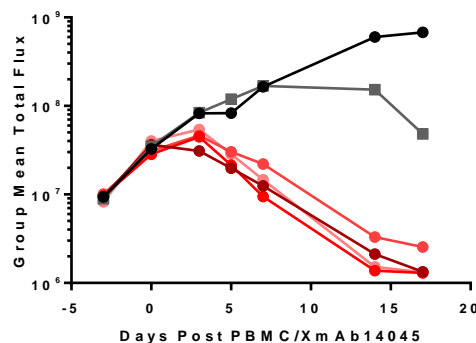


Xencor Lead Bispecific Programs: T-Cell Engagement with Tuned Potency and mAb-like PK

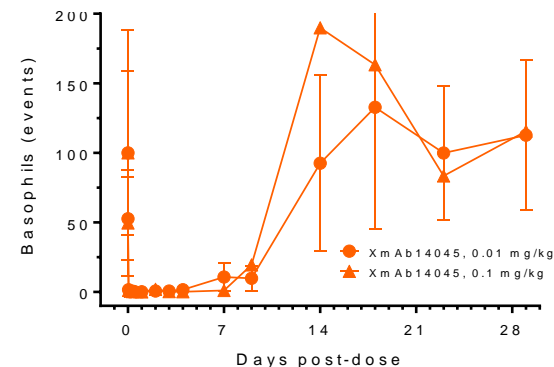
CD3 x CD123
for AML



Strong anti-tumor activity in mice

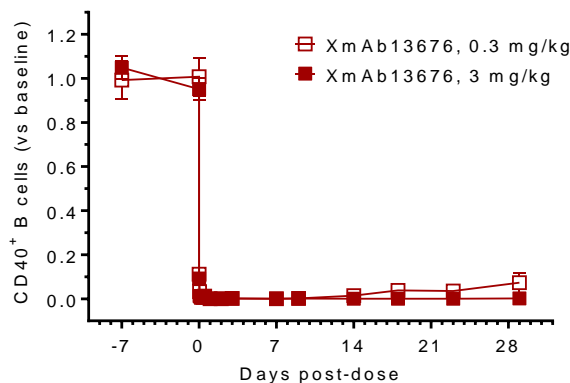


Basophil depletion in monkeys

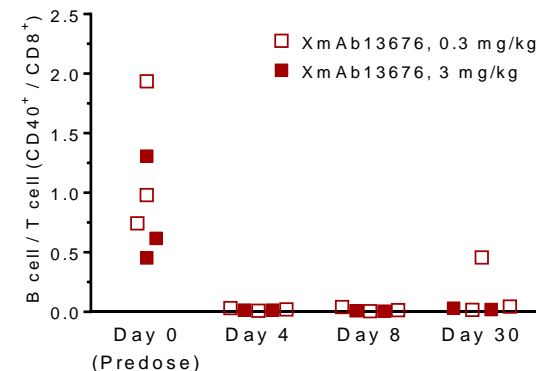


B cell depletion in monkeys

Peripheral B cells



Lymph Node B cells



- Both programs display antibody-like PK in vivo

Xencor's CD3 bispecifics are easily manufactured at scale

- XmAb14045 (CD123 x CD3):
 - Process and product validated GMP at clinical scale
 - 200L scale (100 L working volume)
 - Standard 3-step purification process
 - 6 month Drug Product stability (1 mg/ml, 5°C):
 - 100% heterodimer by CEX
 - 100% main peak by SEC
- XmAb13676 (CD20 x CD3)
 - Upstream & downstream process identical to XmAb14045
 - 200L scale (100 L working volume)
 - 1 month Drug Product stability (5 mg/ml, 5°C)
 - 100% heterodimer by CEX
 - 100% main peak by SEC

Plug-and-play platform enables rapid prototyping and lead generation

XmAb14484

XmAb14730

XmAb13124

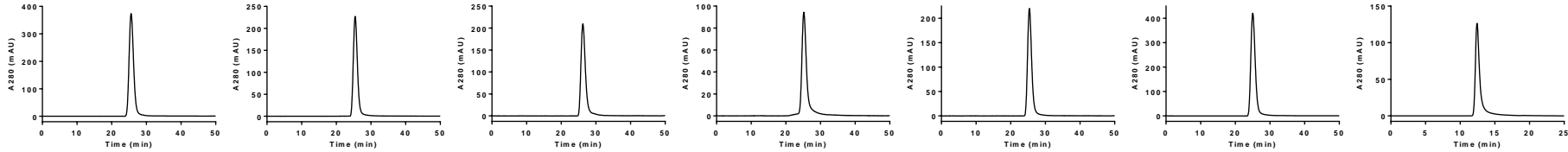
XmAb14481

XmAb14746

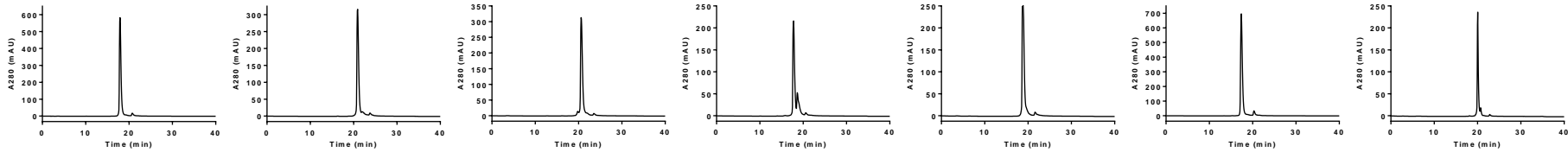
XmAb14455

XmAb18941

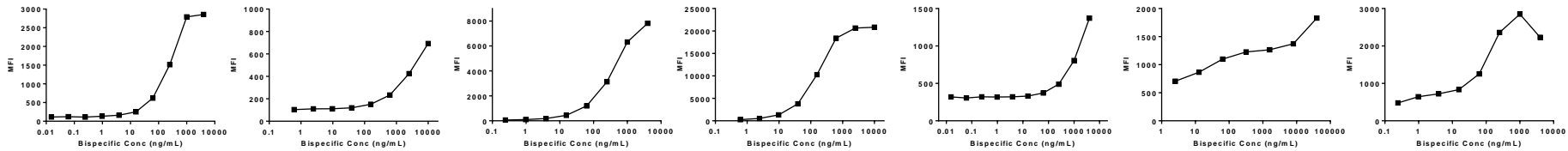
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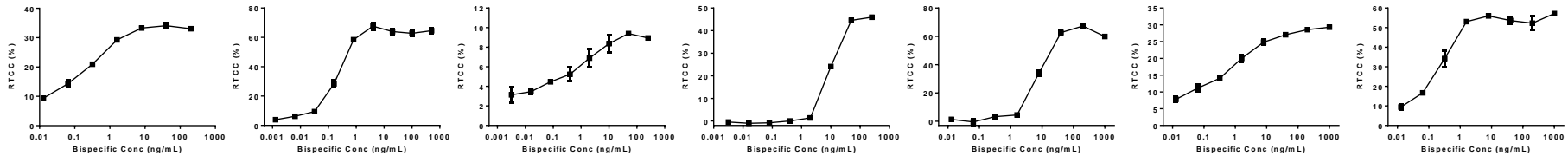
CEIX



Cell Binding



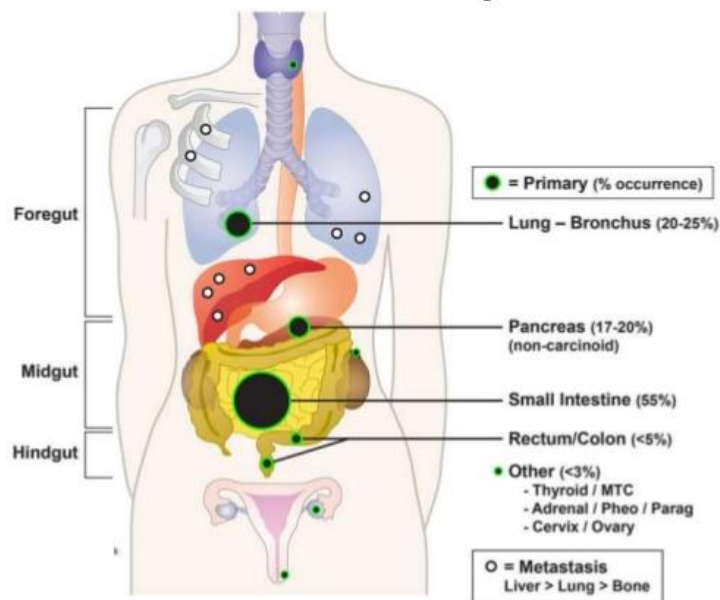
RTCC



Numerous other CD3 and non-CD3 bispecifics produced

SSTR2 x CD3 bispecific antibody for the treatment of neuroendocrine tumors

Neuroendocrine System



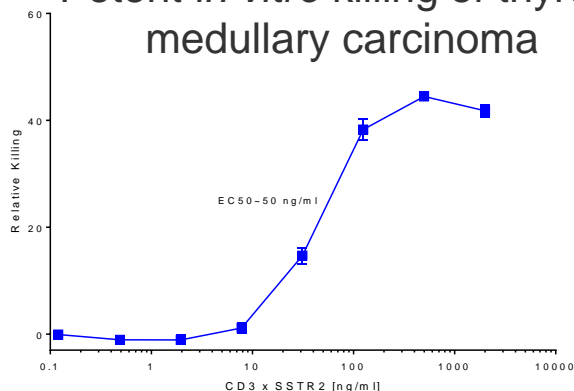
Disease Overview

- Neuroendocrine tumors (NETs) are relatively rare, slow-growing tumors arising from the neuroendocrine system.
- Neuroendocrine system is formed of cells that produce and release hormones to control a wide range of biological functions
- NETs arise from neuroendocrine cells in a wide range of organs (pancreas, digestive system, respiratory system, thyroid, and pituitary gland)
- Functional tumors often present with symptoms specific to the hormone produced by the tumor
- Unresectable NETs are largely thought to be incurable with 3 – 19 years' survival post-diagnosis
- Current US incidence ~ 18000

SCLC provides expansion opportunity: “SSTR2 is highly expressed in many SCLC tumors ...” J Clin Oncol 34, 2016 (suppl; abstr e20090)

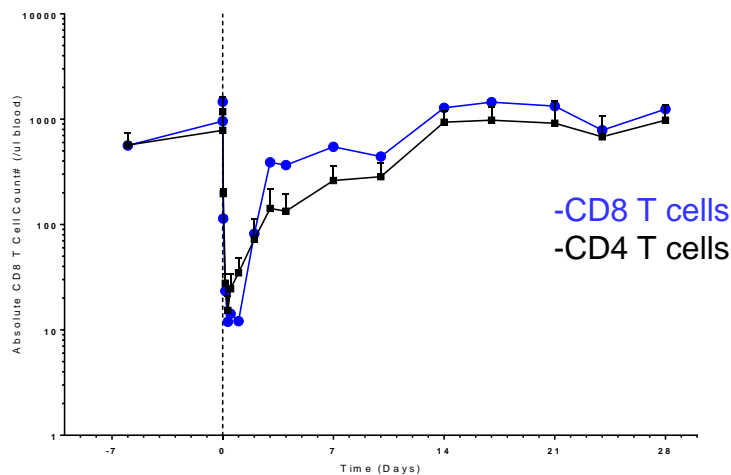
XmAb18087 (SSTR2 x CD3) bispecific kills NET cells in vitro and activates T cells in monkeys

Potent *in vitro* killing of thyroid medullary carcinoma

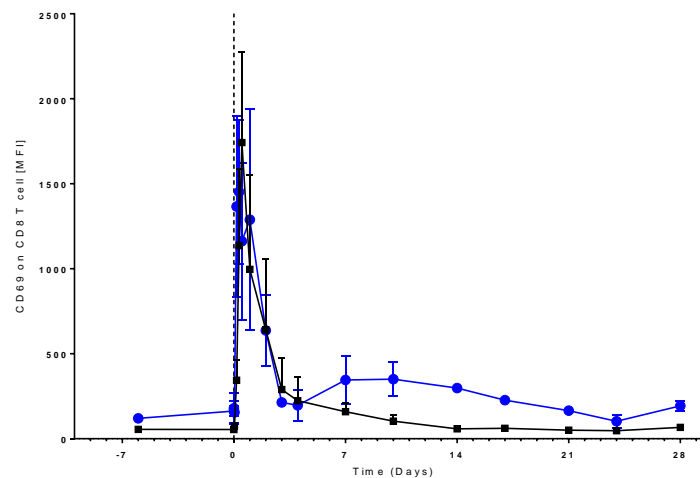


Non-human primate study

T cell redistribution



T cell activation



Target IND filing mid-2017

XmAb[®]14045

Clinical Development Program

June 28, 2016



XmAb14045 Clinical Development – Planned Studies

XmAb14045-01

- **Design:** Phase 1, open-label, multiple ascending dose study in patients with CD123 expressing hematologic malignancies
- **Sites:** Up to 8 clinical investigation sites
- **Dose schedule:** XmAb14045 will be administered IV at ascending dose levels. Each cycle will consist of 1 dose per week for 4 weeks (28 days cycle); each patient will receive 2 cycles of therapy. After these 2 cycles, patients will be allowed to continue on therapy as long as there is evidence of clinical benefit
- **Number:** Up to 60 patients
- **Primary endpoint:** Identification of the maximum tolerated dose for first infusion and subsequent infusions and characterization of safety and tolerability
- **Anticipated start:** 3Q-4Q2016

XmAb[®]13676

Clinical Development Program

June 28, 2016



XmAb13676 Clinical Development – Planned Studies

XmAb13676-01

- **Design:** Phase 1, open-label, multiple ascending dose study in patients with relapsed or refractory NHL and CLL
- **Sites:** Up to 12 clinical investigation sites
- **Dose schedule:** XmAb13676 will be administered IV over 2 hours at ascending dose levels. Each cycle will consist of 1 dose per week for 4 weeks (28 day cycle); each patient will receive 2 cycles of therapy. After these 2 cycles, patients will be allowed to continue on therapy as long as there is evidence of clinical benefit
- **Number:** Approximately 65 patients
- **Primary endpoint:** Safety and tolerability, maximum tolerated dose determination
- **Anticipated start:** 4Q2016-1Q2017

XmAb[®] Checkpoint Inhibitor Bispecific Platform and Oncology Pipeline Overview

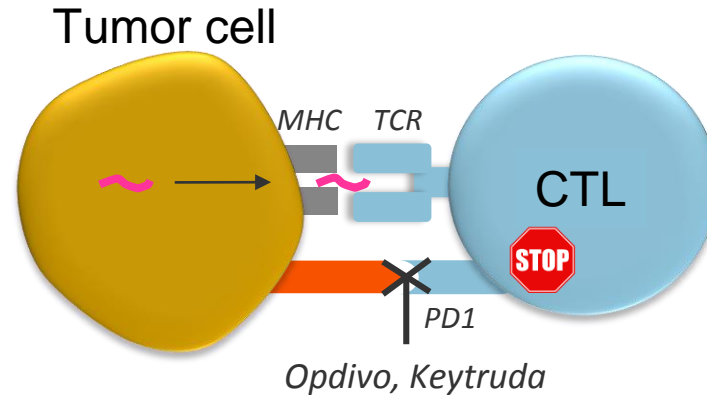
June 28, 2016



Immuno-Oncology

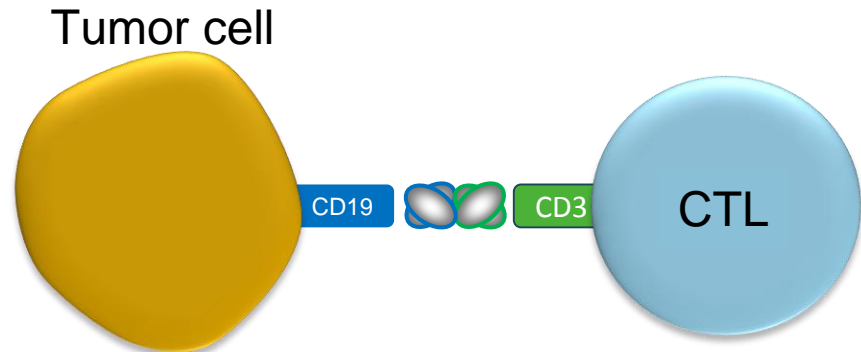
Natural T cell response

- Targets peptide:MHC complex
- Activated by checkpoint inhibitors



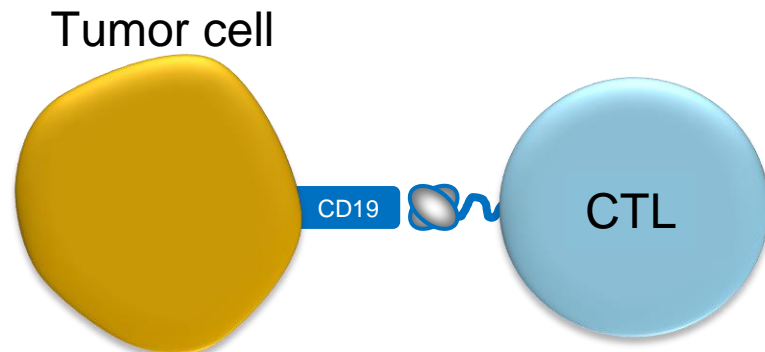
CD3 bispecifics

- Redirect endogenous T cells
- Target quasi-selective tumor surface markers (CD19, CD38, CD20, etc.)

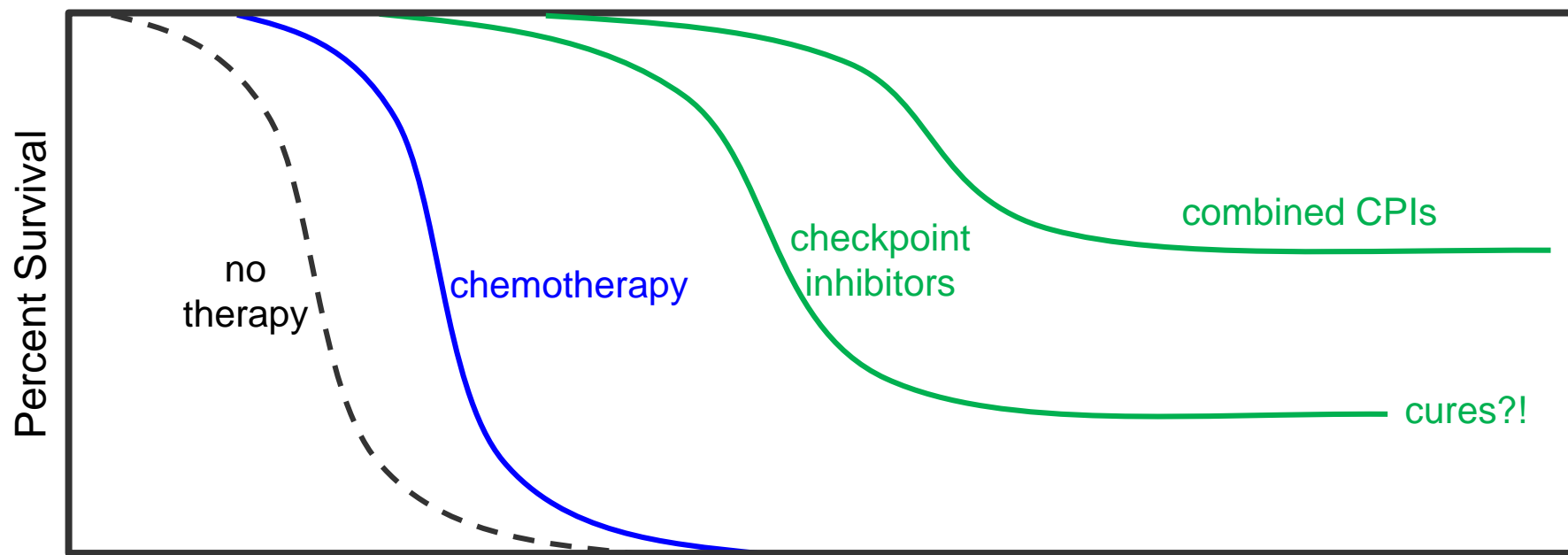


CAR-T

- Engineered T cells
- Target quasi-selective tumor surface markers



Checkpoint inhibitors are changing the cancer treatment landscape dramatically



BUT, combinations synergistic in efficacy and toxicity (and cost)

Response rates:

Monotherapies : ~10-30%

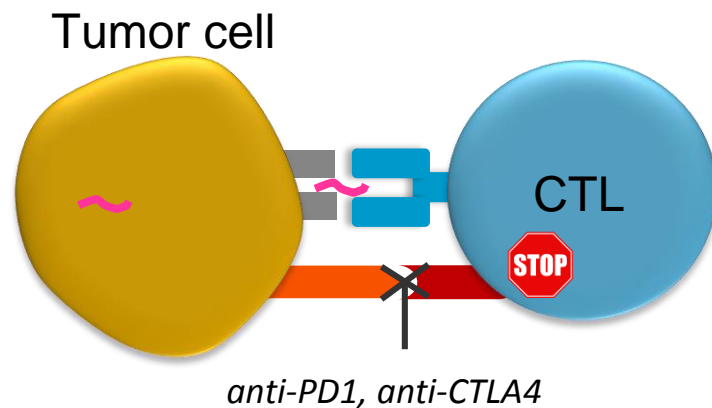
Combinations: ~40-60%

Grade 3/4 Adverse Events

Monotherapies: ~10-20%

Combinations: ~30-60%

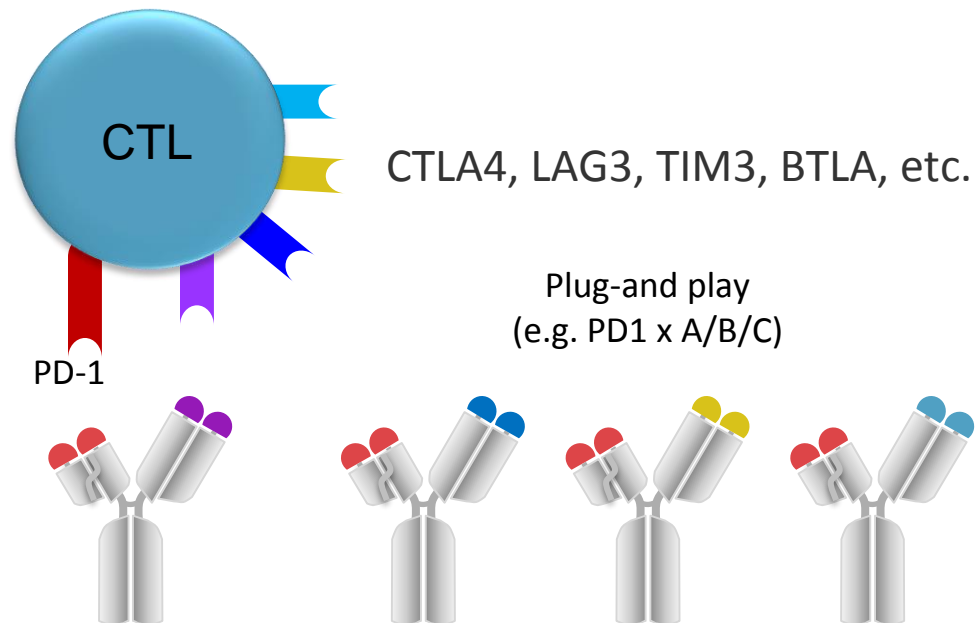
Bispecific checkpoint inhibitors can improve on combinations



Tumor T cells typically co-express multiple checkpoints
(Matsuzaki 2010, Fourcade 2012, Gros 2014)

Why Bispecifics?

- Reduced costs
- Improved selectivity & safety



Checkpoint bispecific hypothesis: target tumor-reactive TILs preferentially

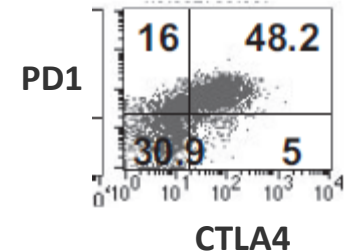
Tumor Environment:

- TILs coexpress multiple checkpoints:

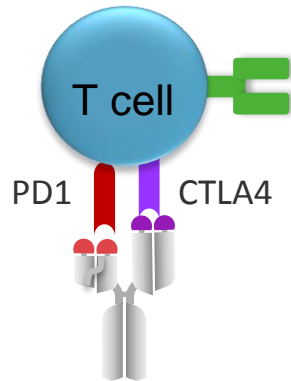
Matsuzaki 2010, Fourcade 2012, Gros 2014, Ahmadzadeh 2009

- Bivalent binding increases avidity

Melanoma TILs



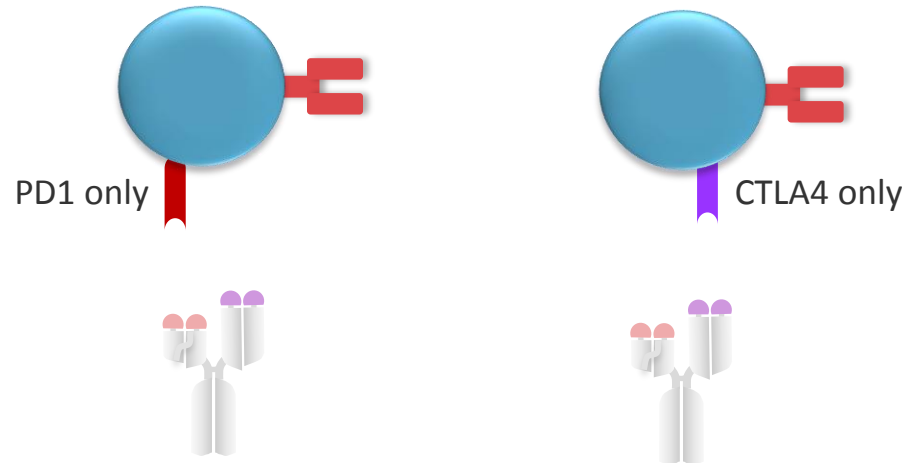
Tumor-reactive



- Strong interactions
- TIL activation

Enhance anti-tumor activity

Non-tumor reactive

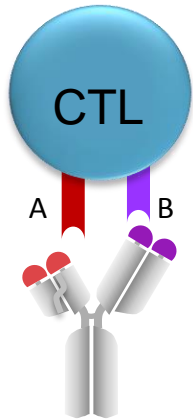


- Weak interactions
- No activation

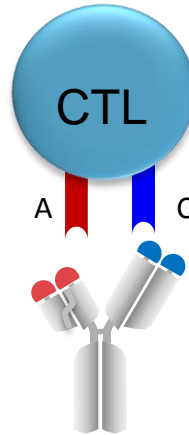
Avoid peripheral toxicity

Exploring numerous hypotheses in checkpoint bispecifics

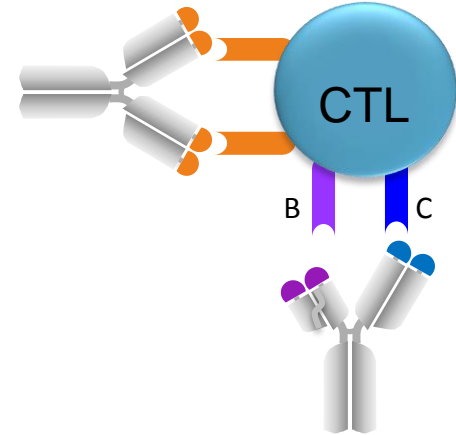
Lead 1: PD1 x CTLA4



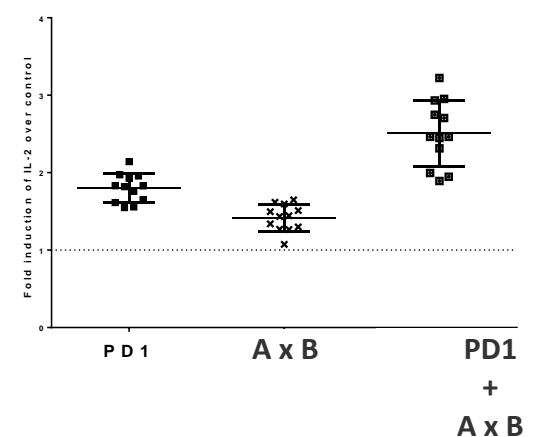
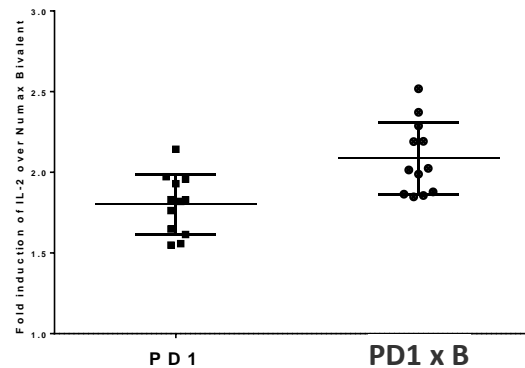
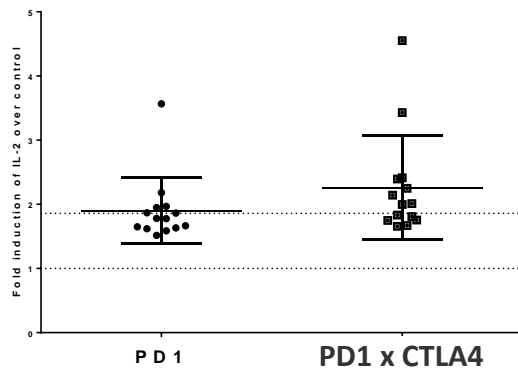
Lead 2: PD1 x B



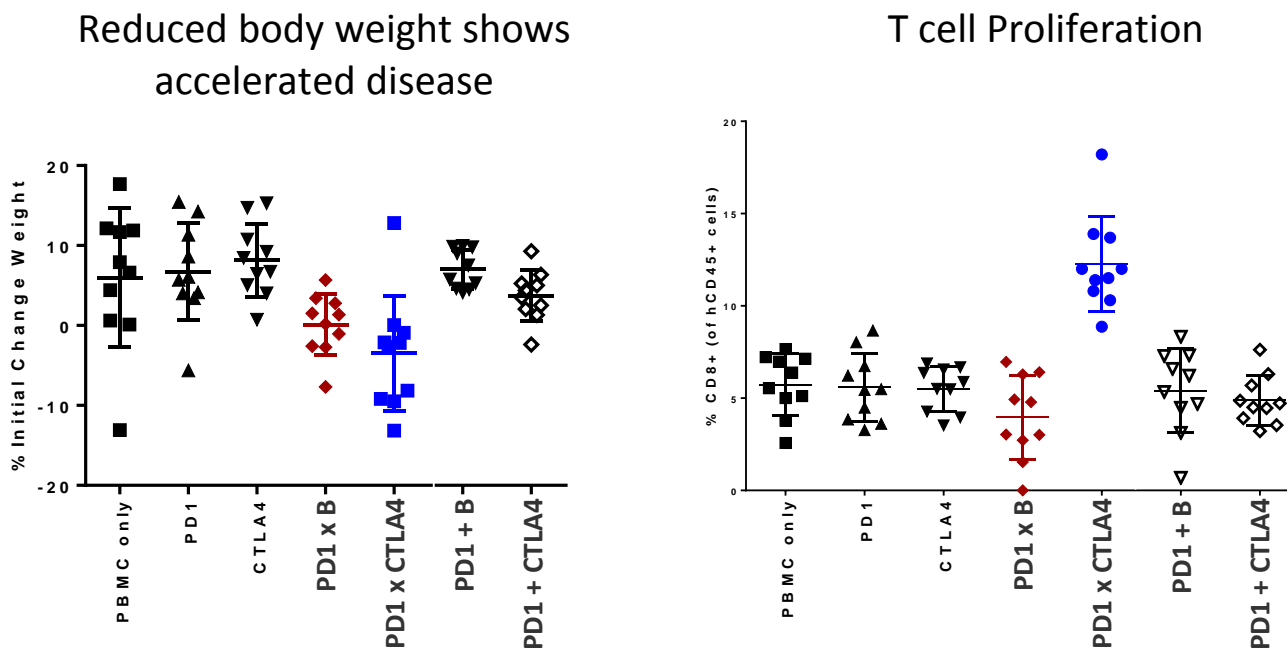
Lead 3: A x B
(combine with anti-PD1)



In vitro SEB stimulation assays (multiple donors), IL2 production



In vivo model for checkpoint blockade: acceleration of GVHD with prototype bispecifics

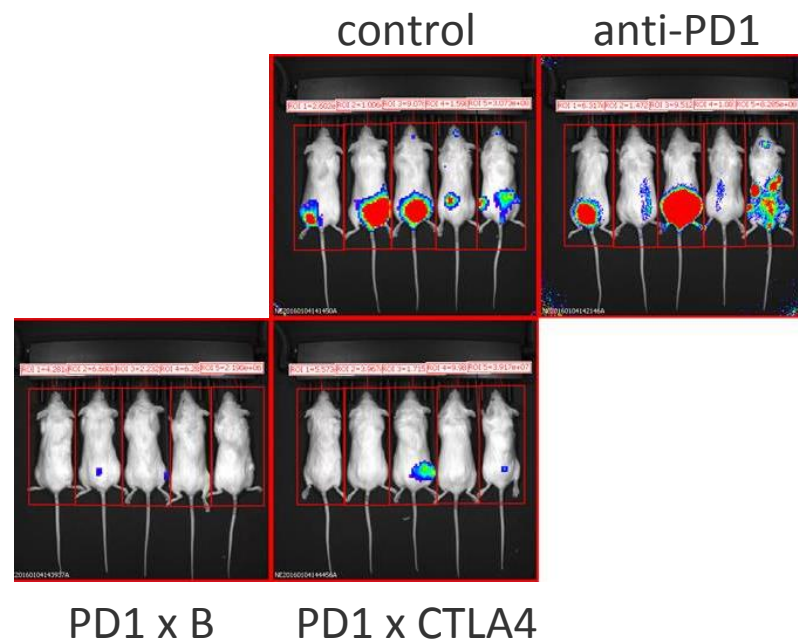
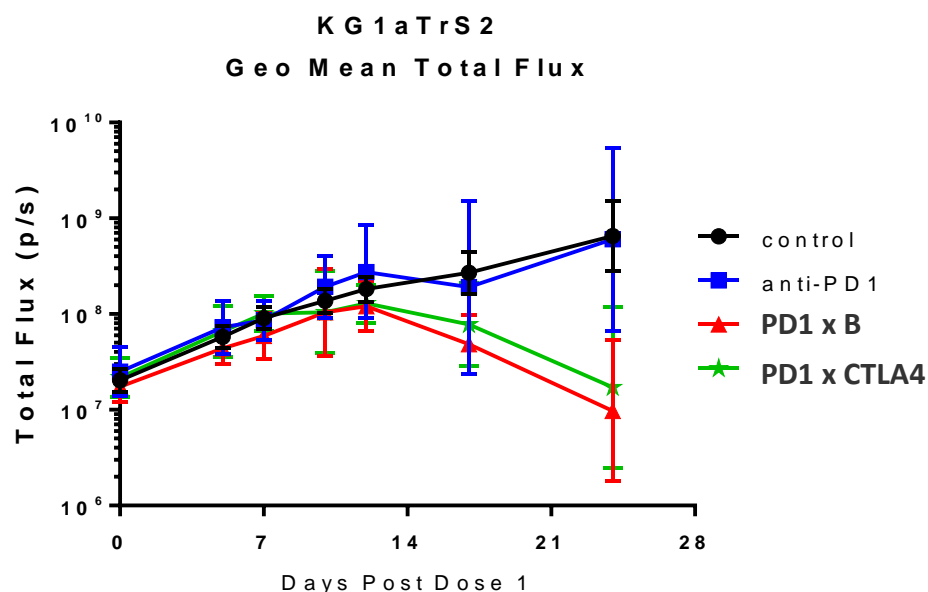


Summary:

- **Bispecifics accelerate disease relative to combinations**
- New biology emerges with bispecific checkpoint blockade
- Bispecifics have complex biological differences compared to each other
- Bispecifics different from combinations!

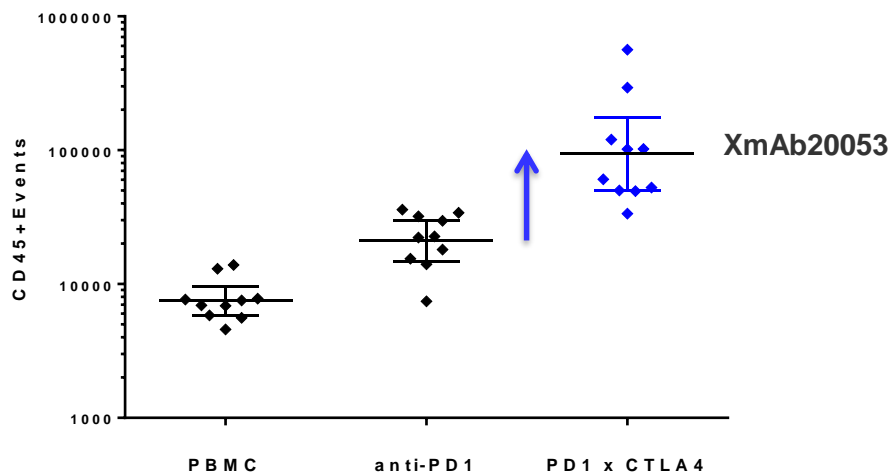
Checkpoint bispecific prototypes have superior anti-tumor activity in a pilot xenograft study

huPBMC model with KG1a established tumors

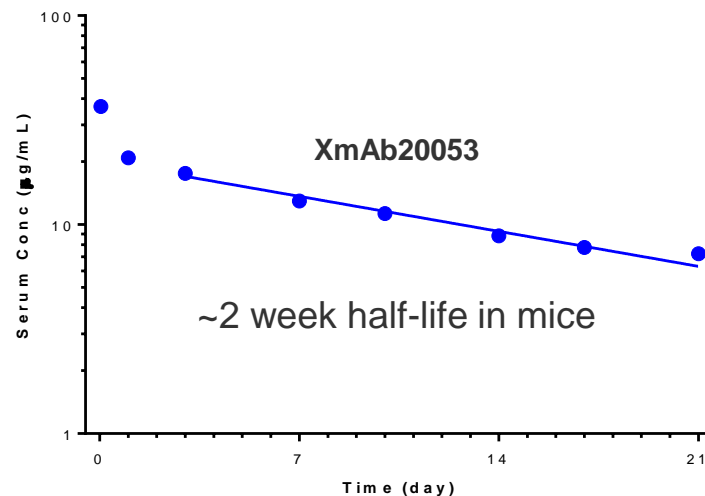


Selected PD1 x CTLA4 candidate bispecific has superior activity to nivolumab in vivo, and long half-life

Mouse model (huPBMC-NSG):
superior T cell activation



Antibody-like PK in Mice

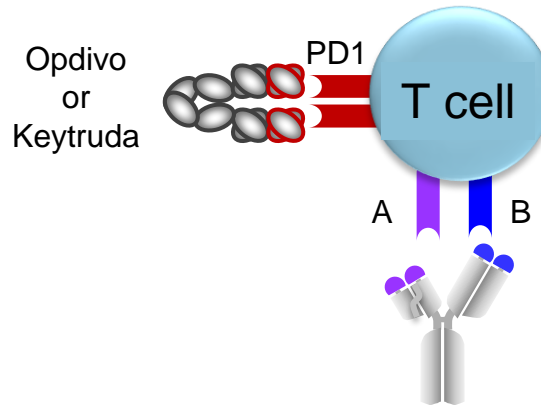


Two candidates:

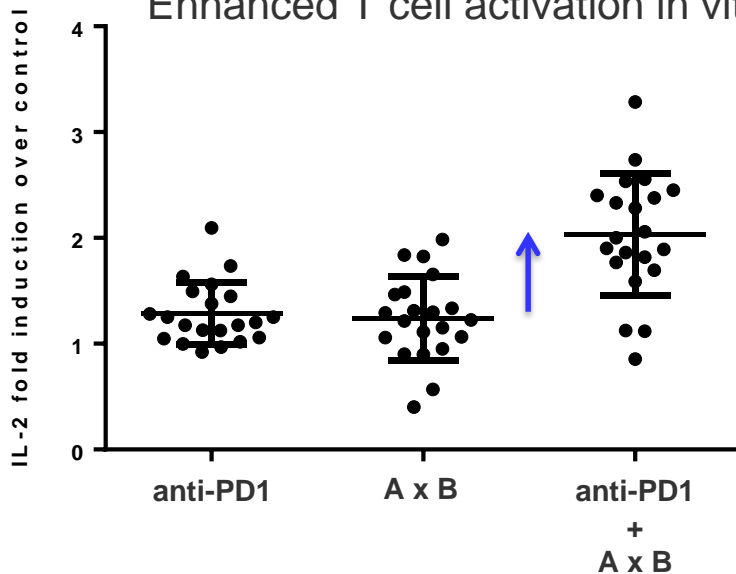
- XmAb20053
- XmAb20717 (Xtend version)
- Stable cell lines in progress
- Projected IND filing late 2017

A x B bispecific combines with anti-PD1 for additional T cell activation (lead optimization in progress)

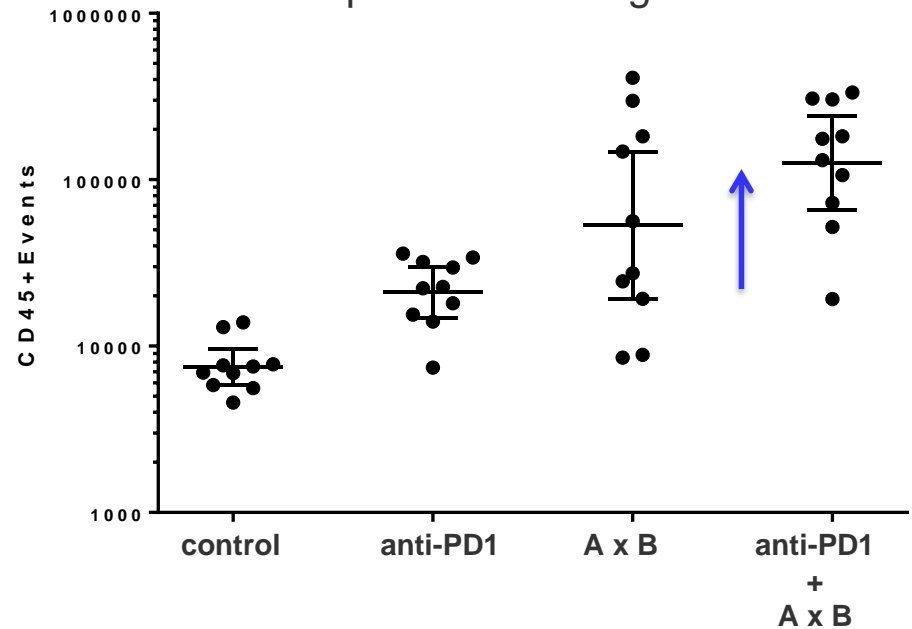
Triple checkpoint blockade



Enhanced T cell activation in vitro



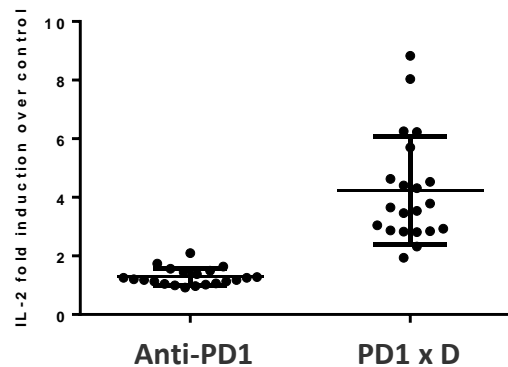
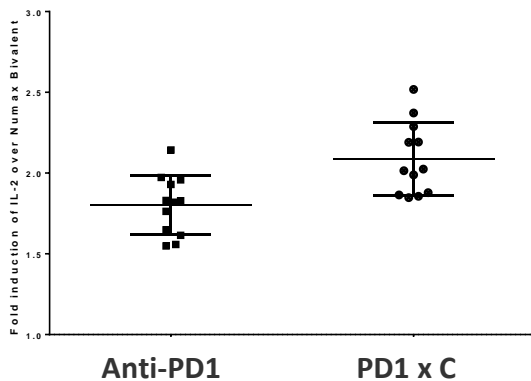
Mouse model (huPBMC-NSG): superior T cell engraftment



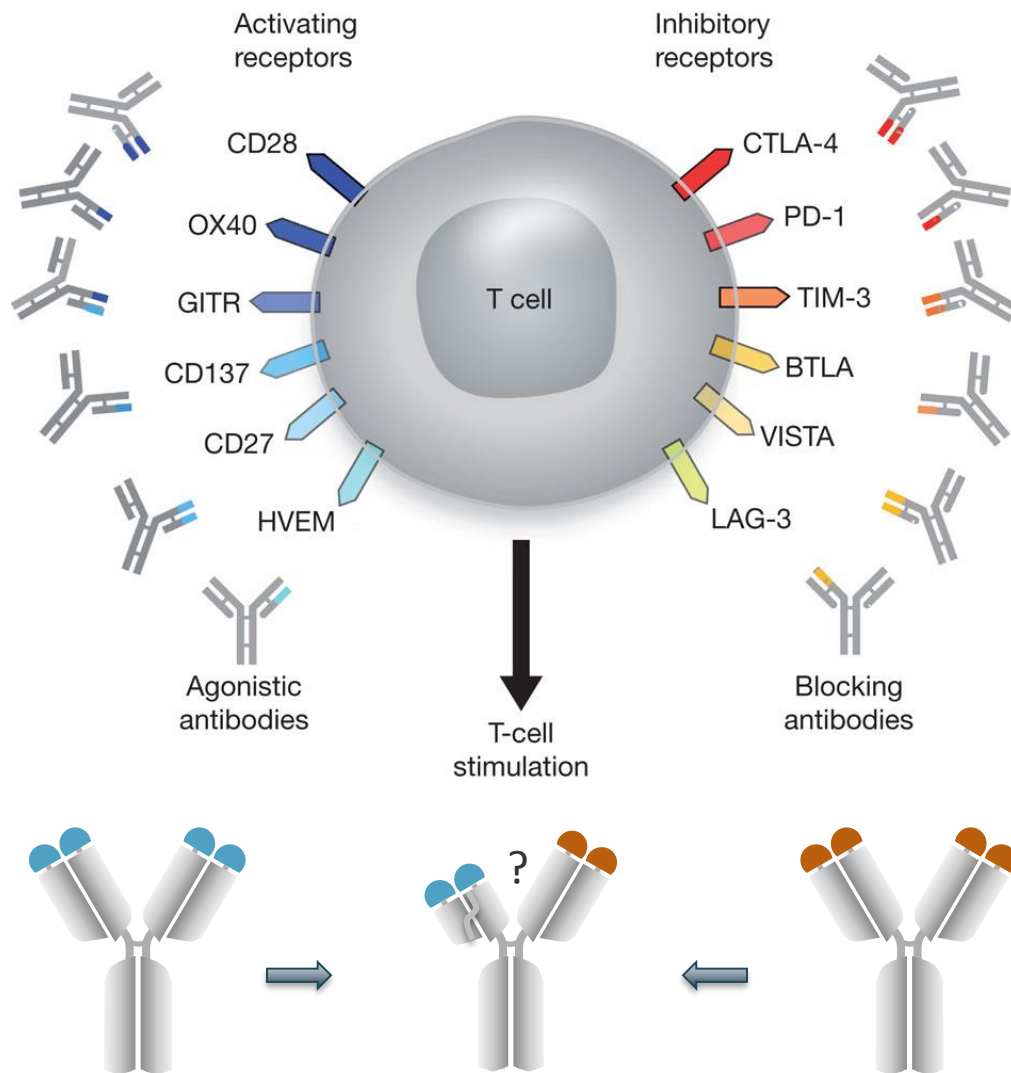
Additional bispecific checkpoint combinations in lead optimization or screening



















In vitro SEB stimulation assays (multiple donors), IL2 production



Can checkpoint blockade and costimulation be productively combined in a bispecific antibody?



Xencor's Growing Bispecific Oncology Pipeline to Enter Clinical Trials in 2016

| Program (Target) | Fc Domain | Primary Indication | Discovery Lead | Preclinical | Phase 1 | Commercial Rights |
|---|----------------------|--------------------|--|-------------|-----------|---|
| XmAb14045 (CD123 x CD3) | Bispecific | AML |  | | (2016) |  xencor* |
| XmAb13676 (CD20 x CD3) | Bispecific | B-cell cancer |  | | (2016/17) |  xencor* |
| XmAb13551 (CD38 x CD3) | Bispecific | Myeloma |  | | |  AMGEN |
| XmAb18087 (SSTR2 x CD3) | Bispecific | Oncology |  | | |  xencor |
| XmAb20717 (PD1 x CTLA4) | Bispecific/ Xtend | Oncology |  | | |  xencor |
| Undisclosed CI x CI | Bispecific | Oncology |  | | |  xencor |
| Undisclosed (x CD3) | Bispecific | Oncology |  | | |  xencor |
| Undisclosed Immune Modulation | Bispecific | Oncology |  | | |  xencor |

* Novartis licensed ex-US commercial rights, worldwide co-development

- CD3 bispecifics to target T cells to tumors, entering clinic in 2016
- SSTR2 x CD3 IND mid-2017, followed by PD1 x CTLA4
- Scalable platform process for GMP manufacturing developed

Antibodies by Design:

XmAb® Antibody Therapeutics

Analyst Day Closing

















June 28, 2016



Agenda Highlights

- Novartis collaboration
 - Ex-US commercial rights to XmAb14045 and XmAb13676 licensed, 50/50 development cost share worldwide
 - Access to bispecific platform for 4 Novartis programs; Fc platform access
 - \$150M upfront, \$2.41B milestones, royalties
- XmAb5871
 - Review of Phase 2 trials design in IgG4-Related Disease and Systemic Lupus Erythematosus
 - Review of mechanism and Phase 1 and 2a clinical data
 - Plan for XmAb5871 subcutaneous clinical trial in 3Q2016
- XmAb7195
 - Review of Phase 1a clinical data
 - Plan for XmAb7195 subcutaneous clinical trial in 4Q2016
- XmAb oncology bispecifics new programs
 - XmAb18087: SSTR2 x CD3 for neuroendocrine tumors, IND expected 2017
 - XmAb20717: PD-1 x CTLA-4 dual checkpoint inhibitor, IND expected 2017

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| Undisclosed CI x CI | Bispecific | Oncology |  | | |  xencor |
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Milestones and Goals 2015-2016

| 2015 | |
|------|---|
| ✓ | XmAb7195 Phase 1a healthy volunteer IgE reduction data |
| ✓ | XmAb5871 Phase 1b/2a data in RA |
| ✓ | XmAb14045, anti-CD123 x CD3 GMP production |
| ✓ | XmAb13676, anti-CD20 x CD3 GMP production |
| | Additional partner milestones |
| ✓ | <ul style="list-style-type: none"> • <i>Amgen research collaboration</i> |
| ✓ | <ul style="list-style-type: none"> • <i>Janssen/CSL Phase 2</i> |

| 2016 | |
|------|--|
| ✓ | XmAb5871 clinical trial start IgG4-Related Disease |
| ✓ | XmAb5871 clinical trial start in SLE |
| ✓ | XmAb7195 Phase 1a complete data, |
| | XmAb7195 Phase 1 subcutaneous trial start |
| | XmAb14045 Phase 1 start in AML |
| | XmAb13676 Phase 1 start in B-cell cancer |

Cash at March 31, 2016 \$178.7 million; \$150M upfront payment from Novartis