Antibodies by Design:

XmAb® Antibody Therapeutics

October 2018

Forward-Looking Statements

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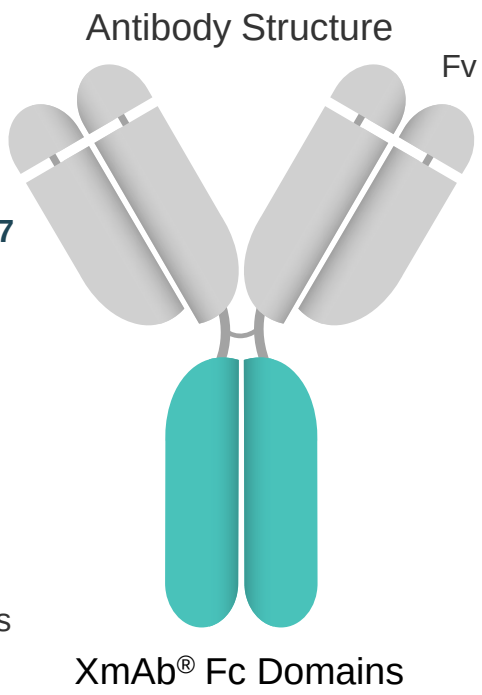
Xencor: Engineering Antibody Immune Functions to Make Better Drugs

- XmAb® Fc domains: small changes, big functional impacts
  - Augments native immune functions, preserves half-life, stability and production
  - Over 500 issued patents and pending patents worldwide

- Expansive bispecific oncology pipeline advancing
  - Phase 1 trials ongoing for **XmAb14045, XmAb13676, XmAb18087** and **XmAb20717**
  - Additional bispecific program INDs planned in 2018 and 2019
  - Novartis co-development and ex-US license for **XmAb14045** and **XmAb13676**
    - $150M upfront, $2.4B potential milestones
  - Amgen advancing six preclinical XmAb programs in oncology and inflammation
    - $45M upfront, $1.7B potential milestones

- Internal autoimmune programs in clinical development
  - **XmAb5871** in Phase 2 in IgG4-Related Disease and Systemic Lupus Erythematosus
  - **XmAb7195** in Phase 1 development for allergic disease

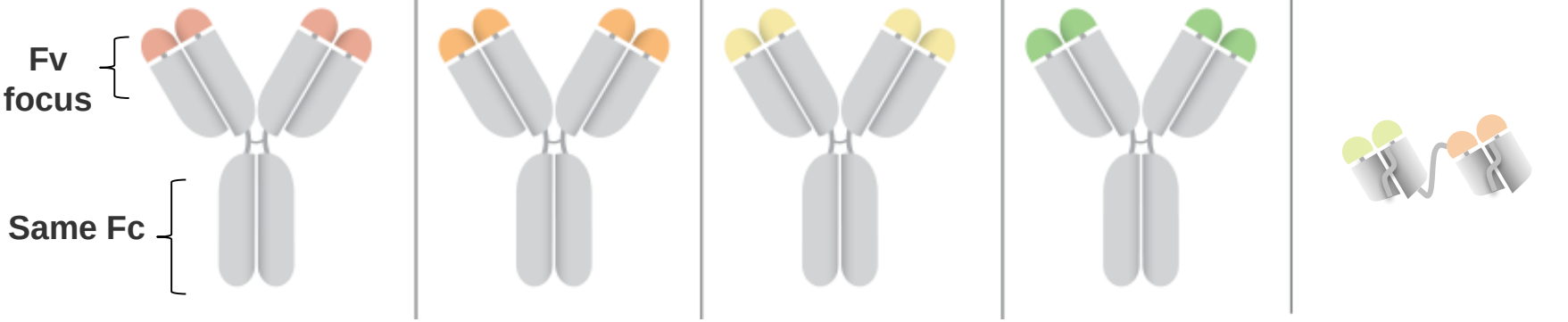


- 12 XmAb clinical programs ongoing internally or with partners, one BLA filed, one in Phase 3

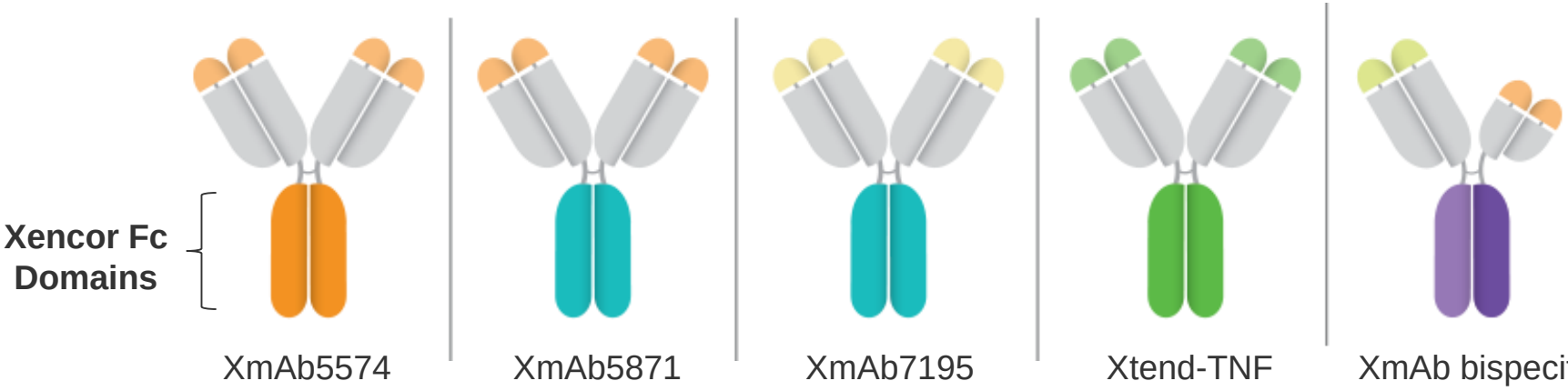
NOVARTIS morphosys ALEXION AMGEN CSL Boehringer Ingelheim

XmAb® Fc Domains Shift Focus of Antibody Drug Discovery by Creating New Axes for Differentiation

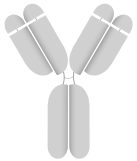
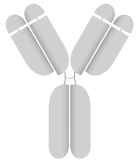
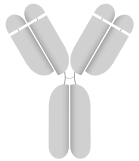
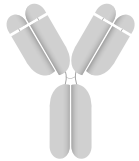
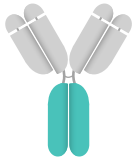
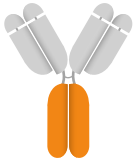
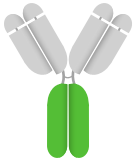
Standard Antibodies



XmAb Antibodies



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

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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					xencor
XmAb7195 (IgE)	Immune Inhibitor	Asthma/ allergy					xencor
XmAb14045 (CD123 x CD3)	Bispecific	AML					xencor NOVARTIS*
XmAb13676 (CD20 x CD3)	Bispecific	B-cell malignancy					xencor NOVARTIS*
XmAb18087 (SSTR2 x CD3)	Bispecific	GEP-NET GIST					xencor
XmAb20717 (PD-1 x CTLA-4)	Bispecific Xtend	Oncology					xencor
XmAb22841 (CTLA-4 x LAG-3)	Bispecific Xtend	Oncology					xencor
XmAb23104 (PD-1 x ICOS)	Bispecific Xtend	Oncology					xencor
XmAb24306 (IL-15/IL-15Rα)	Bispecific	Oncology					xencor

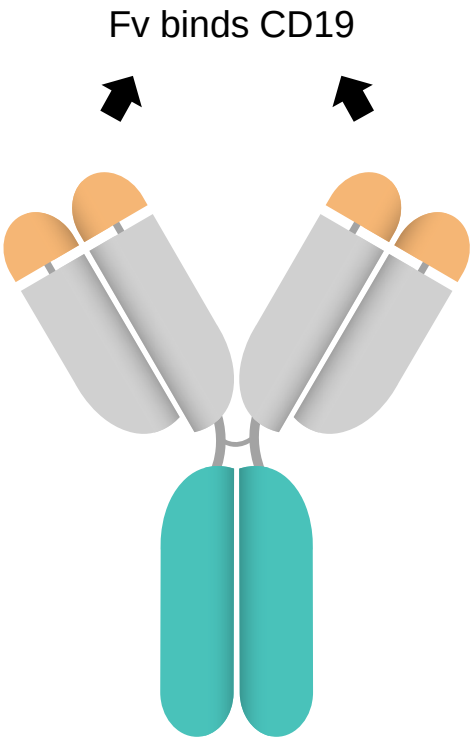
* Novartis licensed ex-US commercial rights, worldwide co-development

XmAb® Fc Domains Have Created Numerous Differentiated Antibodies for Technology Partners

Program	Fc Domain	Primary Indication	Preclinical	Phase 1	Phase 2	Phase 3	Commercial Rights
Ravulizumab (ALXN1210)	Xtend	PNH/aHUS				BLA Filed	ALEXION
XmAb5574/MOR208	Cytotoxic	NHL/CLL					morphosys
Talacotuzumab	Cytotoxic	Leukemia					CSL
BI 836858	Cytotoxic	Oncology					Boehringer Ingelheim
VRC01LS	Xtend	HIV					NIH
AMG424	Bispecific	Myeloma					AMGEN
XmAb bispecific x5	Bispecific	Oncology Inflammation					AMGEN
XmAb bispecific x4	Bispecific	Oncology					NOVARTIS

Technology licensing expands pipeline with very little opportunity cost

XmAb®5871 Inhibits Multiple Pathways of B Cells without Killing B Cells



Immune Inhibitor Fc Domain FcγRIIb binding up by ~400x.

- Phase 2 trials in IgG4-Related Disease and SLE
  - Final data on IgG4-RD presented at the American College of Rheumatology (ACR) Meeting on November 7, 2017
  - Data from SLE expected 4Q2018
- B-cell inhibition: proven for autoimmune disease
  - B-cell depletion (e.g. Rituxan) – RA, MS, others off-label
  - B-cell growth inhibition (Benlysta) – Lupus
- Current limitations of B-cell targeting antibodies
  - Tradeoff of potency against long-term B-cell ablation
  - No simple subcutaneous delivery

XmAb5871 Target Product Profile

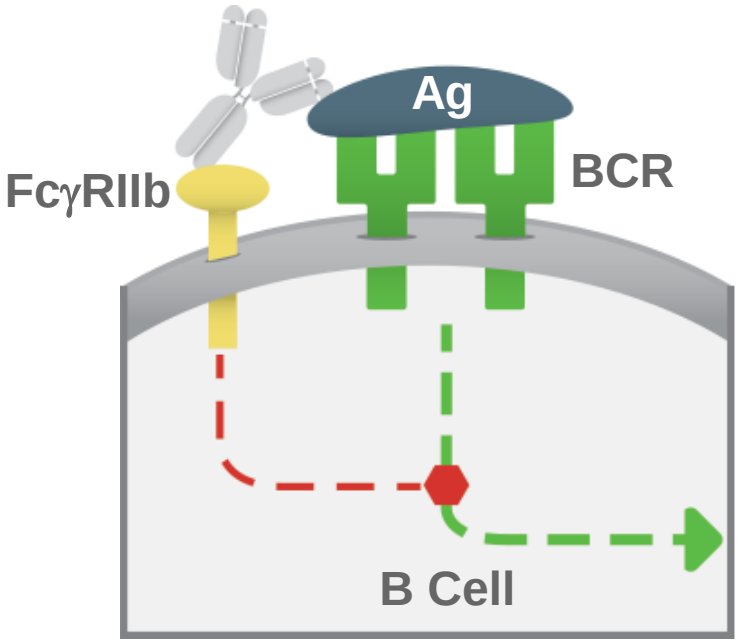
Monoclonal antibody that inhibits B-cell function to treat autoimmune diseases

No long-term immune suppression

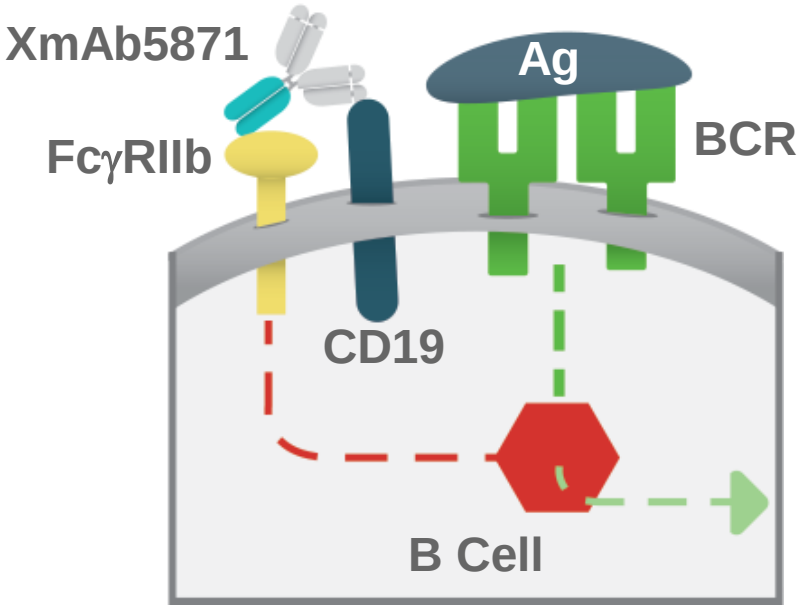
Subcutaneous injection, every other week

XmAb®5871 Enhances Natural Regulatory Role of FcγRIIb

Natural:
Ag + αAg Immune Complex



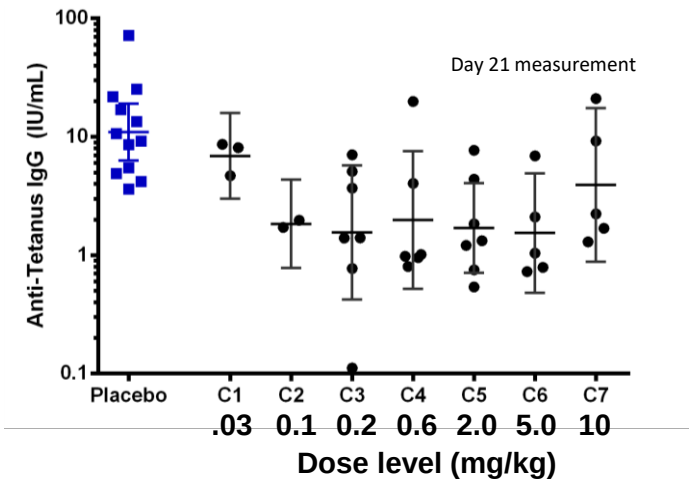
XmAb5871:
anti-CD19 with Immune Inhibitor Fc domain



- 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

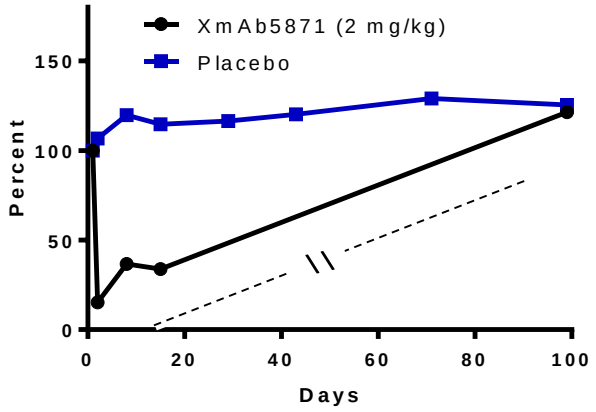
XmAb®5871 Phase 1a Data Shows Potent and Reversible B-cell Inhibition

Inhibition of Antigen Challenge

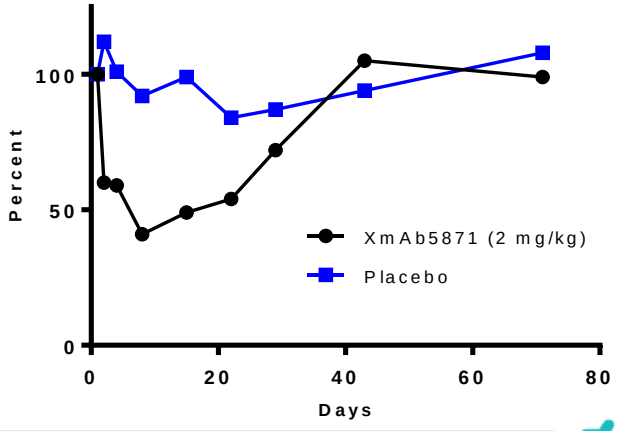


- **Single Ascending Dose in healthy male volunteers**
- **Potent, yet reversible, B-cell inhibition data**
  - Observed effective suppression of B-cell responses to antigen without destroying B cell
  - Target best-in-class efficacy with better tolerability
- **Well tolerated**
  - Partial reduction in B cell counts, rapid recovery upon drug clearance
  - No Dose Limiting Toxicity or Serious Adverse Events (SAE)

Reversible Biomarker Suppression Stimulated CD86 Expression



Reversible decline in peripheral B-cell counts



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XmAb®5871 Phase 1b/2a Trial Shows Clear Signs of Disease Modifying Activity in Rheumatoid Arthritis

Phase 1b/2a trial in patients with active rheumatoid arthritis on stable non-biologic disease modifying anti-rheumatic drug (DMARD) therapy

- 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:

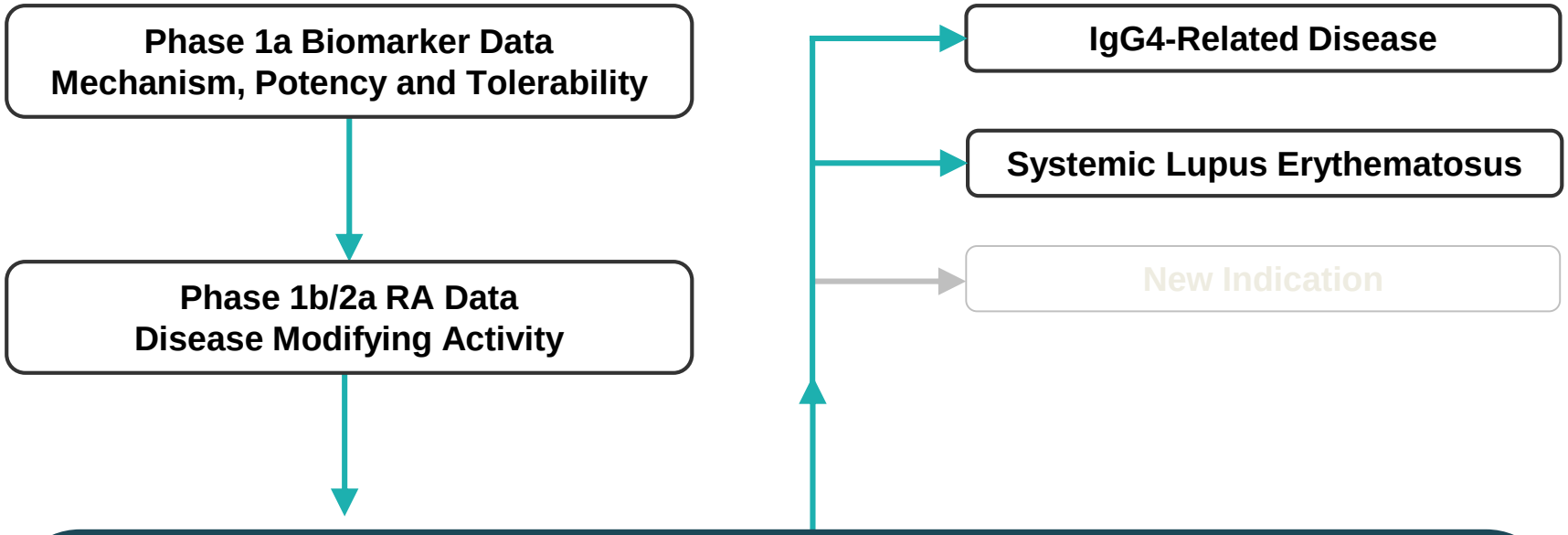
- Generally well tolerated
- Two treatment related SAEs and two placebo treated patients with SAEs

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

XmAb®5871 B-cell Inhibition Profile Presents Opportunity Across Numerous Indications



Key criteria considered: Competition, unmet need, cost/speed of development

Large Indications

- Ankylosing spondylitis (AS)
- Scleroderma
- Moderate-severe systemic lupus erythematosus (SLE)
- Sjogren's syndrome
- Lupus nephritis
- Relapsing-remitting multiple sclerosis (RRMS)
- Rheumatoid arthritis
- Crohn's disease
- IBD

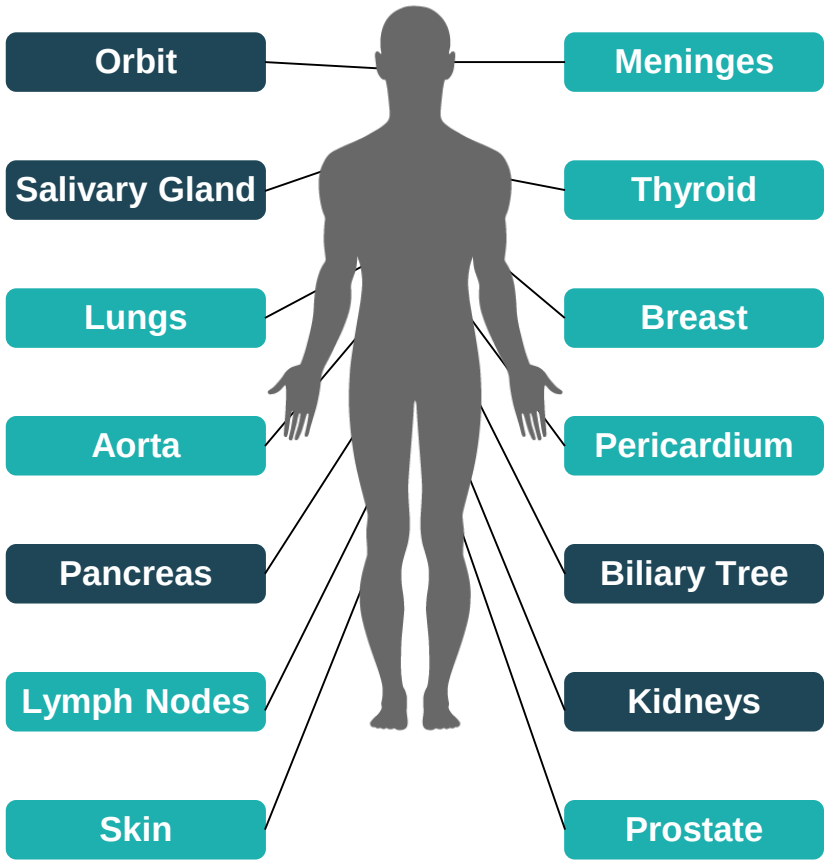
Small Indications

- Autoimmune hemolytic anemia
- Acquired Factor VIII inhibition
- Pemphigus
- Discoid lupus erythematosus (DLE)
- Waldenstrom's macroglobulinemia (WM)
- Desensitization for renal transplantation
- IgG4-Related Disease
- Idiopathic thrombocytopenic purpura (ITP)

XmAb®5871 Phase 2 Development Focused on IgG4-Related Disease

- **Multiorgan disease**
  - Pancreas, biliary ducts, salivary glands, lymph nodes common
  - More than one organ involved in majority
- **Common histopathology**
  - Dense lymphoplasmacytic infiltrate
    - IgG4+ plasma cells
    - T cells
  - Storiform fibrosis
  - Obliterative phlebitis
- **Established, accepted diagnostic histopathology**
  - Reduces issues of heterogeneous disease seen in other autoimmune indications
- **Prevalence**
  - ~ 40,000 patients in US

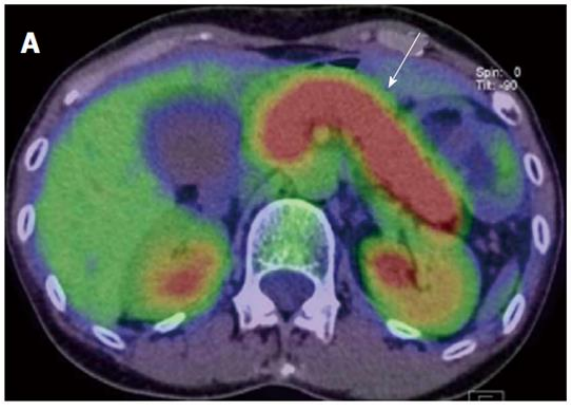
Select Organs Affected by IgG4-RD



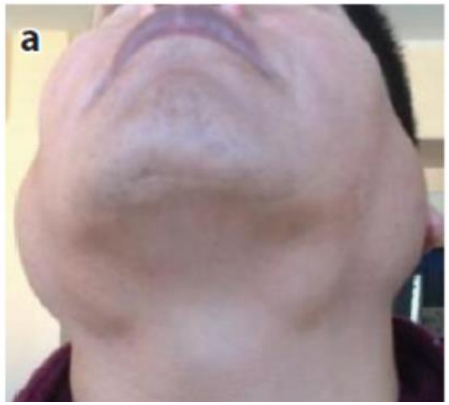
Deshpande V Mod Path 2012
Koshroshahi A Arth Rheum 2010
Yamamoto M Nat Rev Rheum 2014
Carruthers Ann Rheum Dis. 2015
Xencor physician survey

IgG4-RD Fibro-inflammatory Activity Causes Progressive Organ Damage, with No Approved Therapies

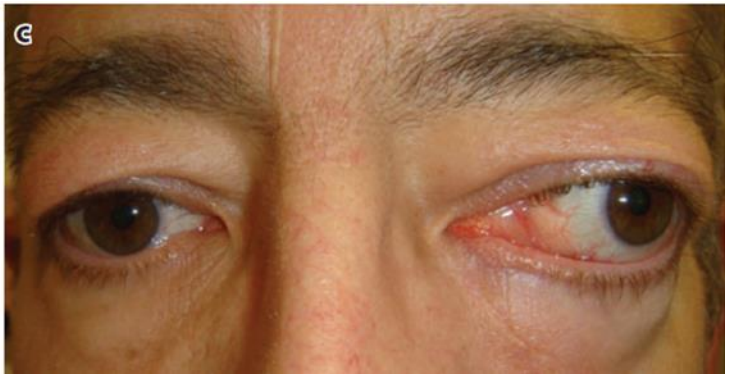
- **Patients present with tissue infiltration, damage and pseudo-tumors**
  - Pancreas: Pancreatitis, pancreatic insufficiency, diabetes, pain
  - Eye: vision loss, proptosis
  - Biliary tree: cirrhosis, cholangitis, pain
  - Submandibular glands: difficulty swallowing, pain
- **Awareness growing**
  - Rheumatologist and GI
  - Still underdiagnosed
- **No approved therapies**
  - Steroids are standard of care
- **IgG4-RD Responder Index (RI)**
  - Assessed in multi-specialty international validation study
  - RI found to be a valid measure of disease activity, regardless of the manifestation or specialist (Wallace Arthritis Care Res 2018)



PET/CT pancreatic swelling (arrow)



Submandibular swelling



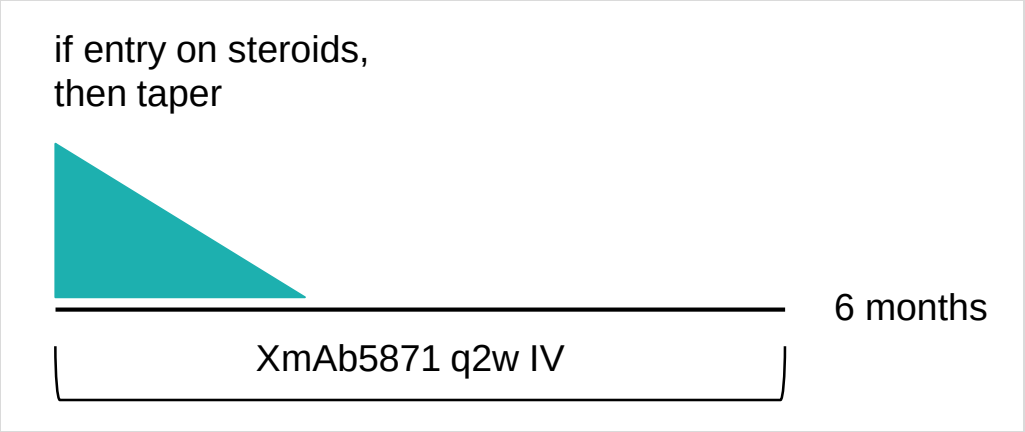
Proptosis

Mahajan Annu Rev Pathol 2014

Murakami World J Clinical Oncol 2011

Xencor physician survey

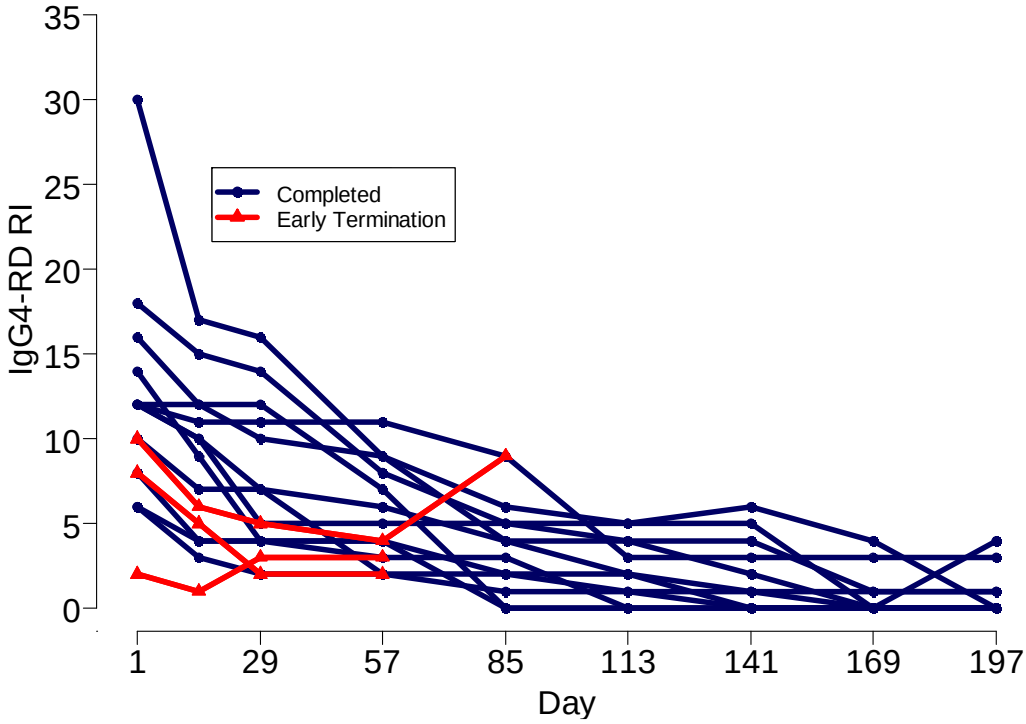
IgG4-RD Pilot Trial Design To Characterize Activity Of XmAb®5871 at Reducing IgG4-RD RI



- **Primary Objective:**
  - To evaluate the effect of XmAb5871 on the IgG4-RD Responder Index (RI) in patients with active IgG4-RD (proportion of patients with improvement in IgG4-RI at 6 months)
- **Final data presentation at the ACR Meeting on November 7, 2017**
- **Based on design of open label pilot study of Rituxan (Carruthers Ann Rheum Dis, 2015)**

Final Data from IgG4-RD Phase 2 Trial Shows Promising Activity

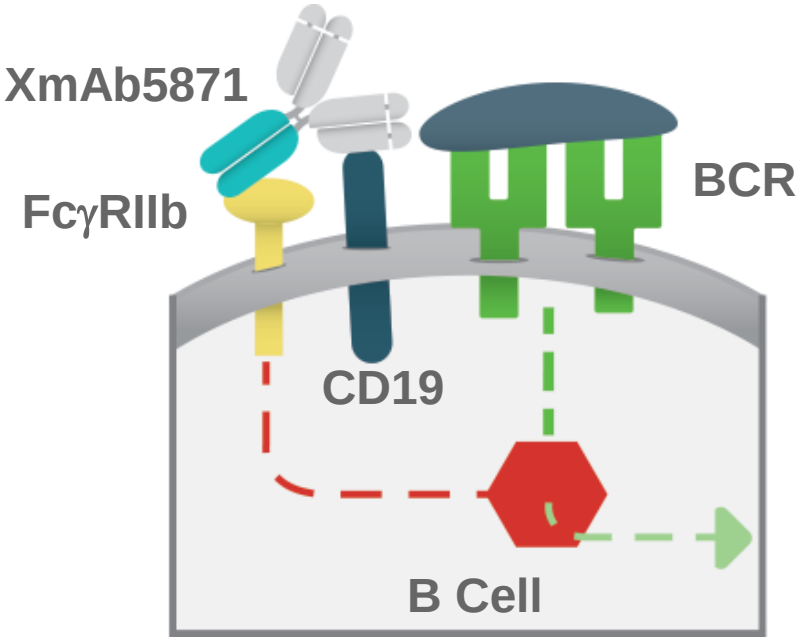
- XmAb®5871 in active IgG4-RD was well tolerated; the most frequent AEs were GI infusion-related symptoms.
- 12 patients (80%) completed the study. All 12 achieved the primary endpoint of a decrease of IgG4-RD RI of ≥ 2 at Day 169.
- Remission (IgG4-RD RI of 0 and no corticosteroids after month 2) was attained in 8 patients at Day 169; 4 others achieved an RI ≤ 4.
- Corticosteroids were tapered and discontinued in all five patients that were on corticosteroids at first XmAb5871 dose.
- 14 of 15 patients achieved a decrease of ≥ 5 in the IgG4-RD RI. Initial response to therapy occurred quickly, most within two weeks of first dose.
- Plasmablasts decreased by about 70-80%, while B cells decreased by 40-55%, both within 2 weeks.
- XmAb5871 was well tolerated and showed promising activity in IgG4-RD.



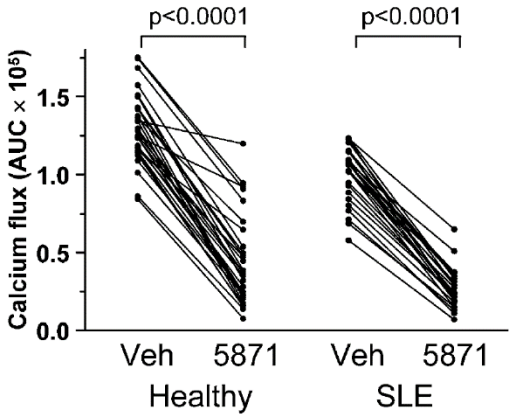
ACR Meeting November 7, 2017

XmAb®5871 Inhibits SLE Patient B Cells *Ex Vivo*

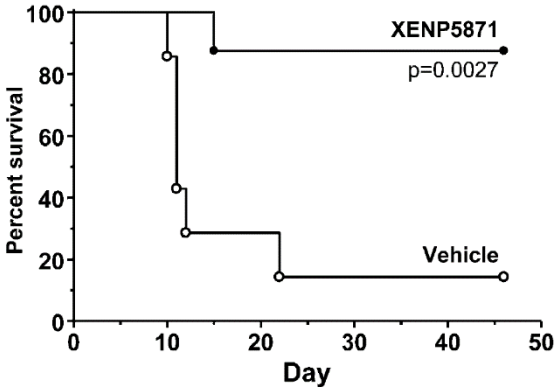
- ***Ex vivo* study of patient blood samples**
  - N=28 SLE, N=30 healthy donors
  - Blockade of IL-4, BAFF, LPS activation signals
  - Protection of SLE peripheral blood mononuclear cell engrafted mice from immune toxicity



XmAb5871 inhibits SLE B-cell activation

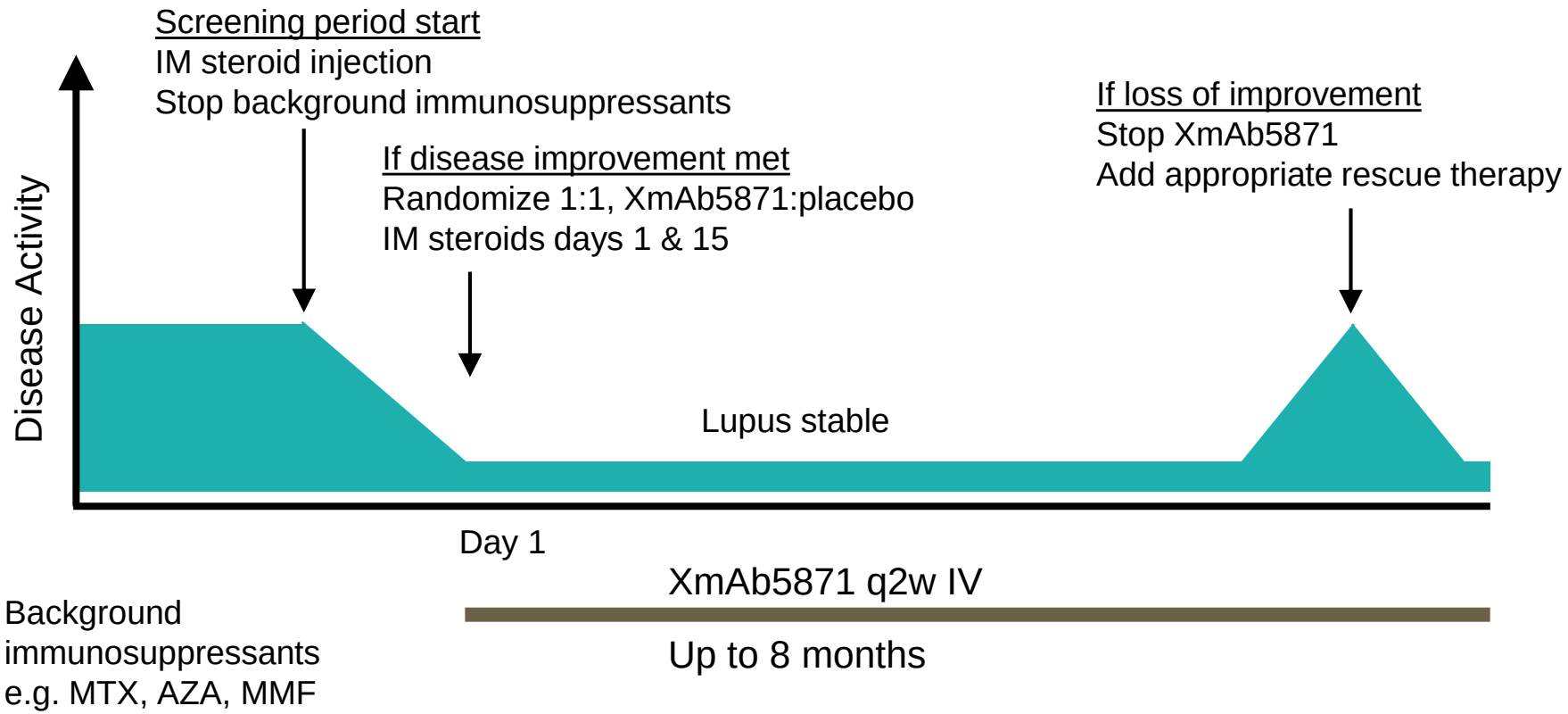


XmAb5871 protects mice from engrafted SLE PBMCs



Horton, J Immunol, 2011.

XmAb®5871 SLE Phase 2 Study Design
Data Expected 4Q2018



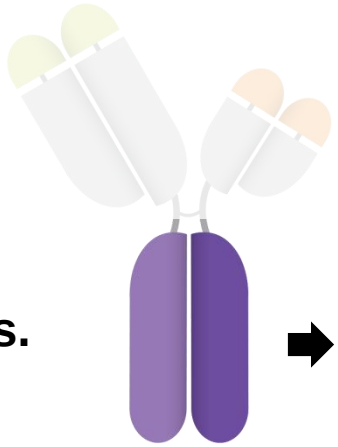
Primary objective: ability of XmAb5871 to maintain SLE disease activity improvement

Randomized, double blinded, placebo controlled

N = 104 patients, ~20 US sites, fully enrolled

XmAb Bispecific Fc Domains Retain Beneficial Natural Antibody Properties

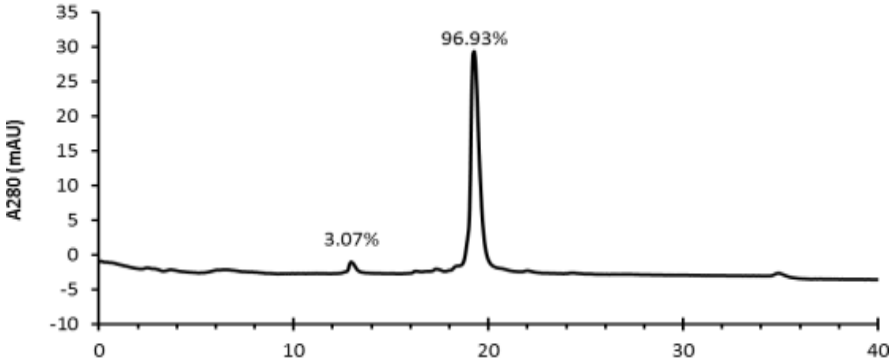
vs.



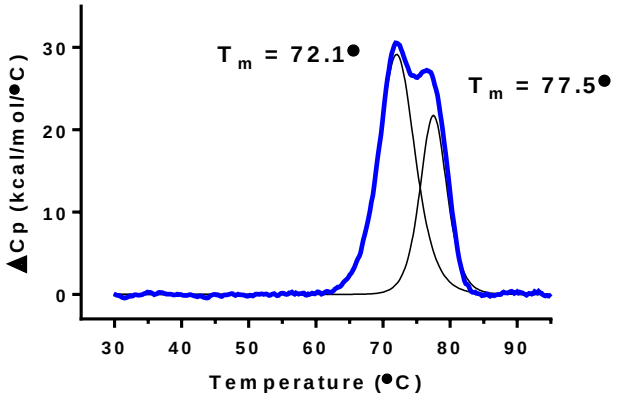
- **Enhanced separation**
- **Heterodimer favored**
- **High stability**

- **Antibody-like half-life *in vivo***
- **Readily produced and purified**
- **Stable scaffold for numerous formats**

Stable cell line expression of XmAb®14045



Heterodimer Fc domain thermal stability



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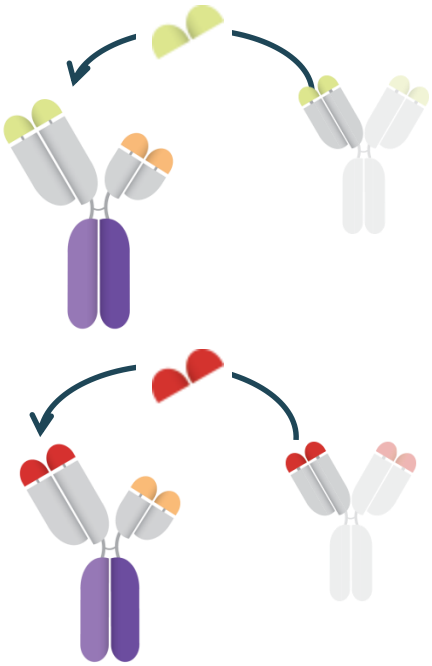
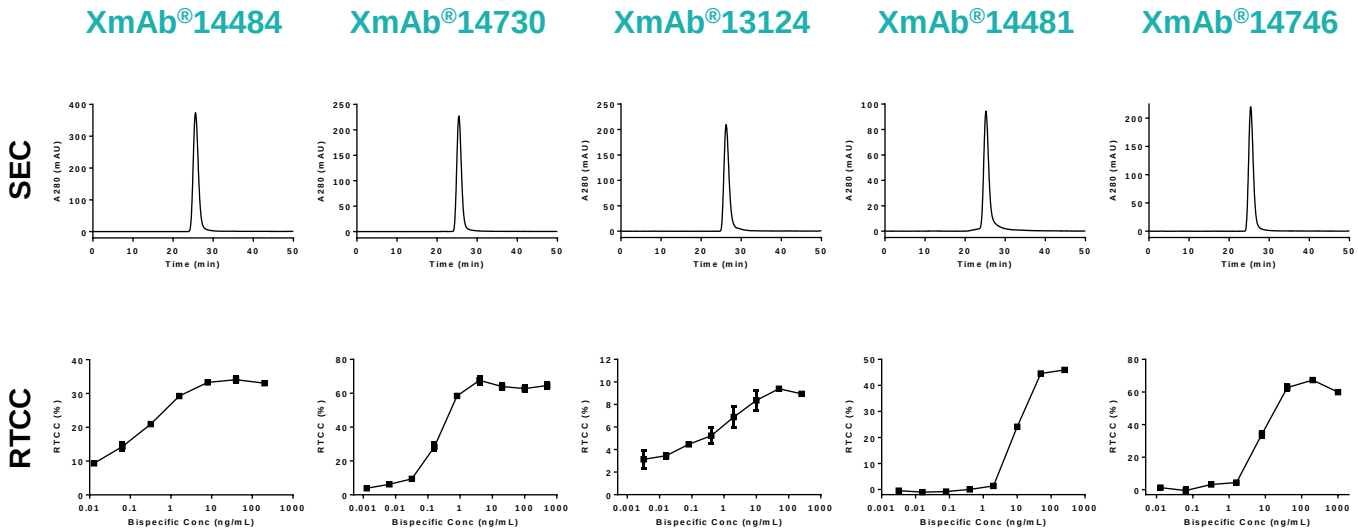
Plug-and-play Fc Domain Enables Rapid Pipeline Generation and Prototyping



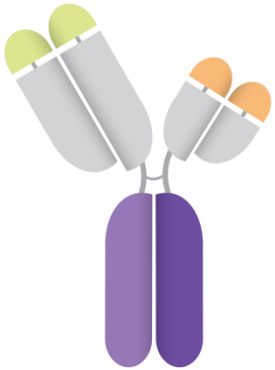


- Portfolio of CD3 bispecific molecules generated for development
  - Target T cells against tumors

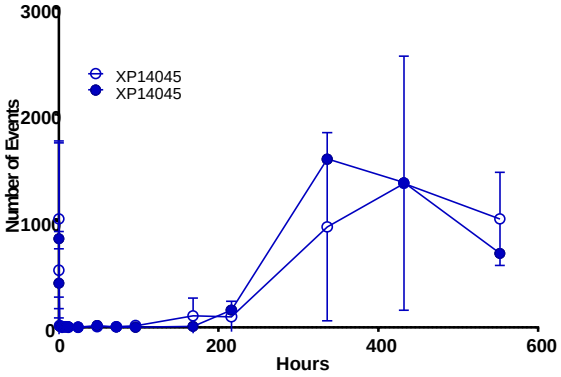
- New immuno-oncology programs rapidly prototype different target combinations

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

XmAb®14045 (CD123 x CD3)

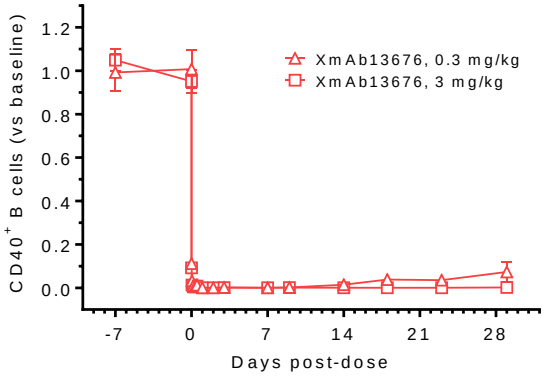


Cynomolgus monkey, single IV bolus
Profound, sustained basophil depletion



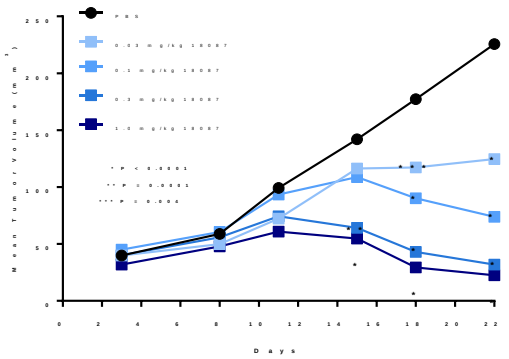
XmAb®13676 (CD20 x CD3)

Cynomolgus monkey, single IV bolus
Profound, sustained B-cell depletion



XmAb®18087 (SSTR2 x CD3)

huPBMC-SCID mouse xenograft
Potent, dose-dependent tumor reduction



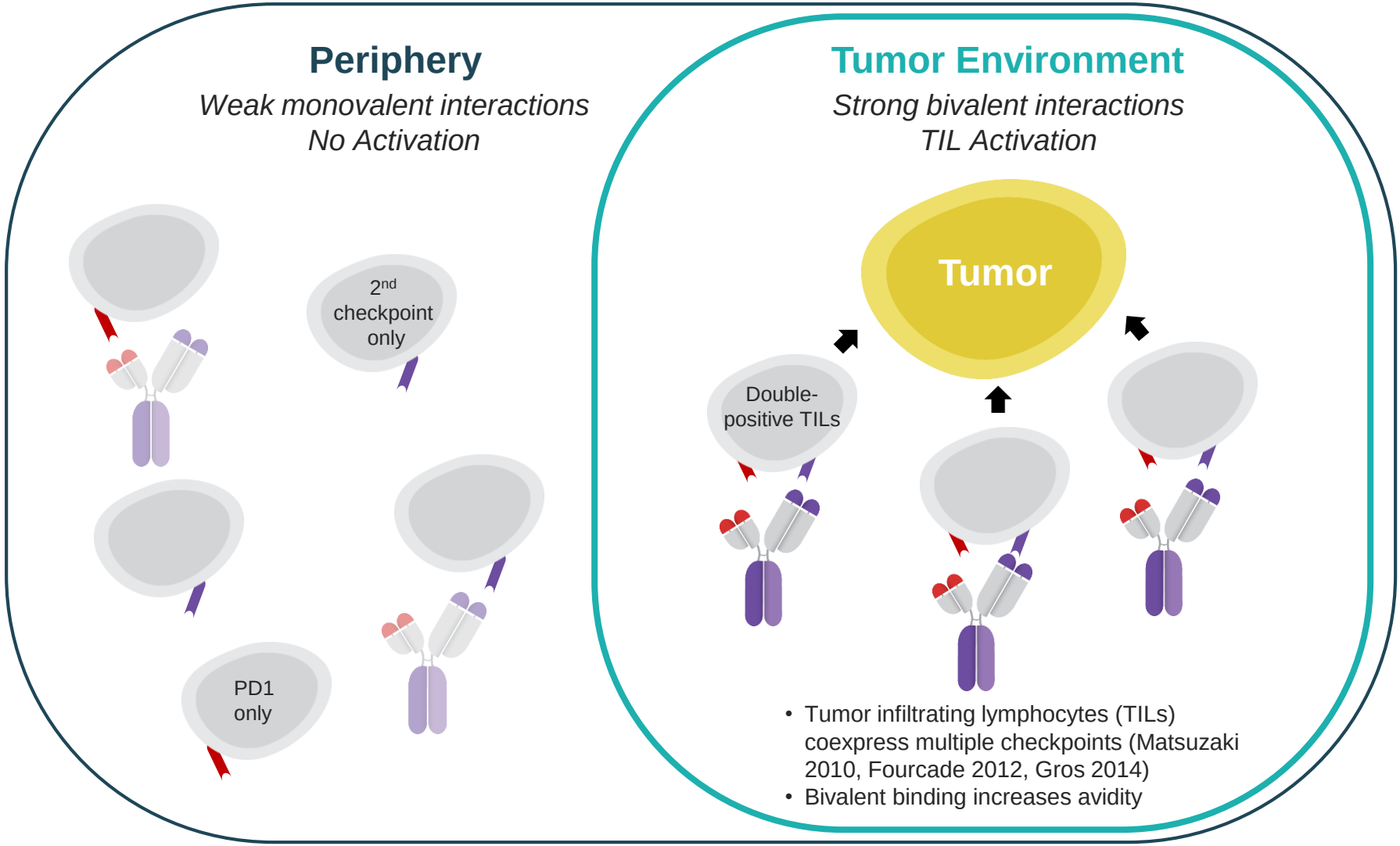
Novartis Collaboration for XmAb®14045 and XmAb®13676 Boosts Development Resources and Retains U.S. Commercial Rights

Links Novartis' leadership in oncology with Xencor's XmAb bispecific programs

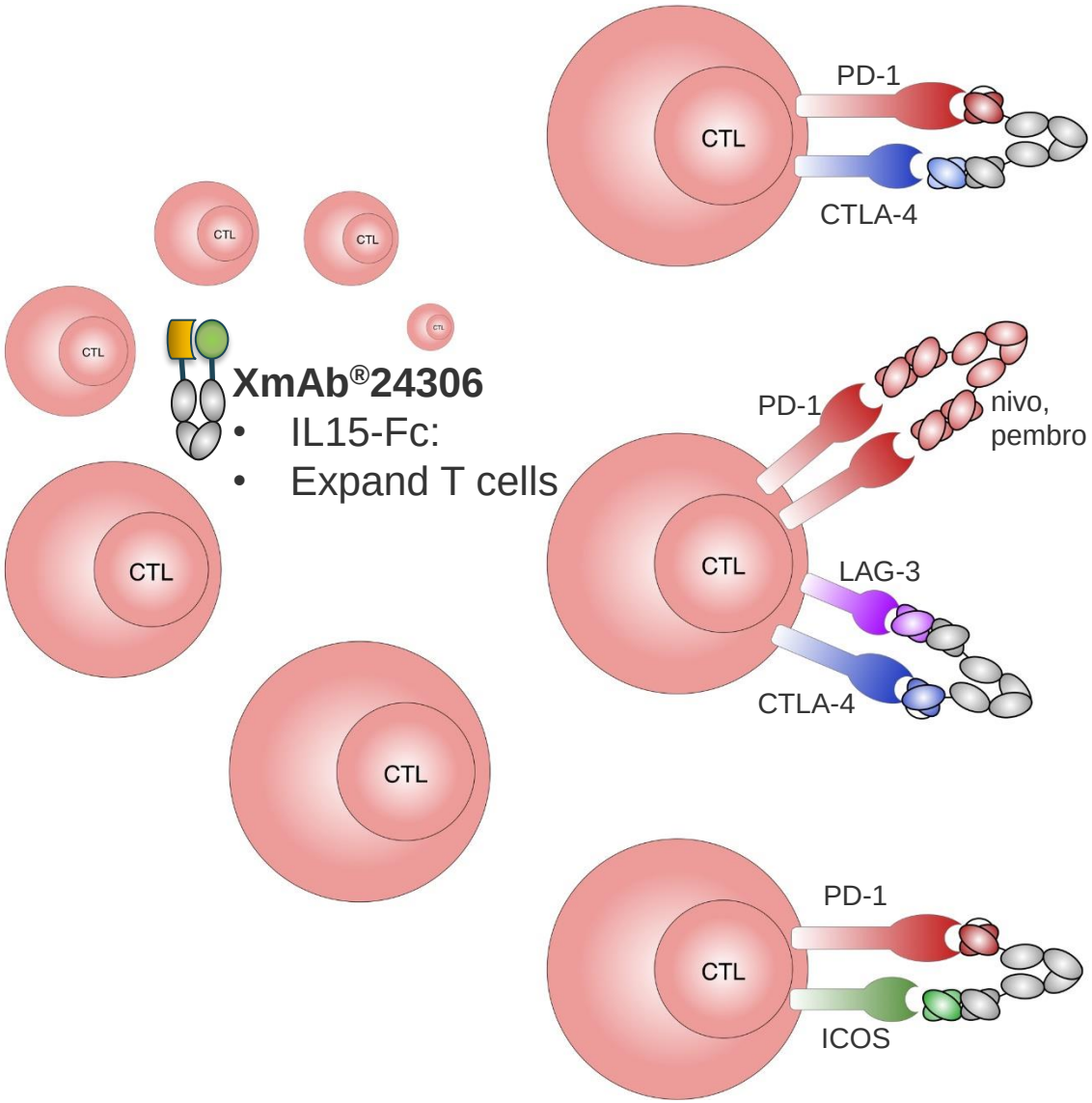
- Novartis receives ex-U.S. commercial rights to XmAb14045 and XmAb13676
  - $325M in milestones per program, including $90M in development milestones
  - Low double-digit royalties on ex-U.S. sales
- Xencor retains all U.S. commercial rights to XmAb14045 and XmAb13676
- Worldwide 50/50 development collaboration 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 U.S. profit and cost share, co-detail
- Non-exclusive access to Xencor Fc Technologies for 10 programs

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

Xencor Checkpoint Bispecifics are Designed to Promote Tumor-Selective T cell Targeting



Distinct and Novel Mechanisms-of-Action Define Xencor's Growing Immuno-Oncology Pipeline



XmAb®24306
- IL15-Fc:
- Expand T cells

XmAb®20717
- PD-1 x CTLA-4 bispecific
- Two most validated checkpoint receptors

XmAb®22841
- CTLA-4 x LAG-3 bispecific
- Combinable with anti-PD1
- Triple checkpoint blockade

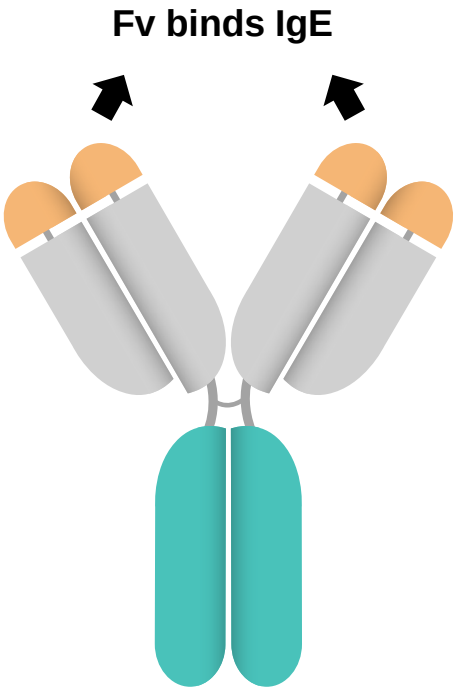
XmAb®23104
- PD-1 x ICOS bispecific
- Novel checkpoint x costim pairing

Xencor's Bispecific Oncology Pipeline Expanding

Program (Target)	Fc Domain	Primary Indication	Discovery Lead	Preclinical	Phase 1	Commercial Rights
XmAb14045 (CD123 x CD3)	Bispecific	AML				xencor / NOVARTIS*
XmAb13676 (CD20 x CD3)	Bispecific	B-cell cancer				xencor / NOVARTIS*
XmAb18087 (SSTR2 x CD3)	Bispecific	GEP-NET GIST				xencor
XmAb20717 (PD1 x CTLA4)	Bispecific/ Xtend	Oncology				xencor
AMG424 (CD38 x CD3)	Bispecific	Myeloma				AMGEN
XmAb22841 (CTLA-4 x LAG-3)	Bispecific/ Xtend	Oncology				xencor
XmAb23104 (PD-1 x ICOS)	Bispecific/ Xtend	Oncology				xencor
XmAb24306 (IL-15/IL-15Rα)	Bispecific/ Xtend	Oncology				xencor

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

XmAb®7195 Introduces a Novel Approach to Reducing IgE in Allergic Disease

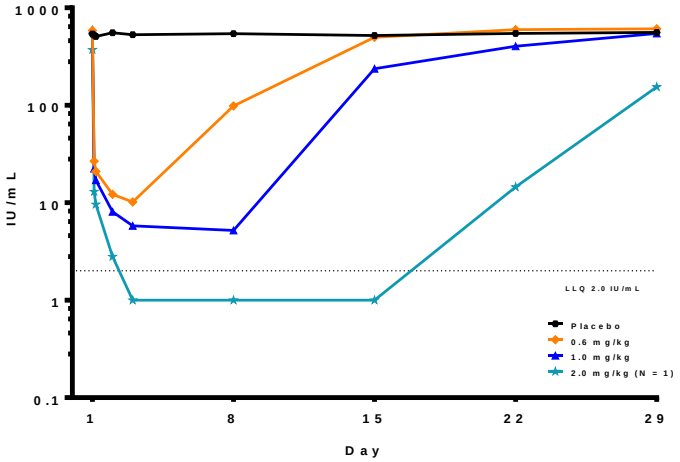


Immune Inhibitor Fc Domain FcγRIIb binding up by ~400x.

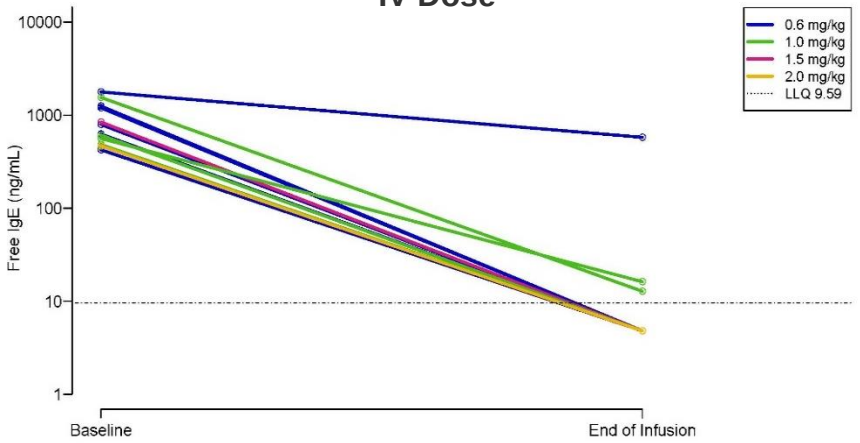
- Completed Phase 1a (intravenous dose) in 2016; Phase 1 subcutaneous formulation completed in 2017
- Data showed potent suppression of IgE
- Potential to address potency limitations of Xolair
- Three distinct mechanisms for reducing IgE including rapid clearance via liver sinusoidal endothelial cells

XmAb®7195 Rapidly Reduces Free and Total IgE in Humans Subcutaneous Dosing Well Tolerated

Total IgE
High IgE Patients
IV Dose



Free IgE
High IgE Patients
IV Dose



Completed Phase 1a study
American Thoracic Society 2016 International Conference

- Phase 1b subcutaneous Multiple Dose Study
  - Well tolerated, no apparent effect on platelet count at ≤ 1.0 mg/kg; mild platelet reduction at 2.0 mg/kg (4/15 patients)
  - Highly potent IgE reduction: 75% patients ≥ 0.3 mg/kg no detectable free IgE for duration of treatment, total IgE similarly effective

- Phase 1a intravenous Single Ascending Dose
  - Generally well tolerated in safety population of 54 patients
    - Dose limiting toxicity of asymptomatic, transient thrombocytopenia at 3.0 mg/kg
    - Dose-dependent, non-clinically significant platelet reductions for most patients dosed ≥0.75 mg/kg
    - Moderate urticaria observed during infusion in ten patients; non-diffuse with mild signs/symptoms and easily treated with oral antihistamine

Milestones and Goals 2017-2018

2017	
✓	XmAb5871 IgG4-RD complete data
✓	XmAb5871 Phase 1 subcutaneous trial data
✓	XmAb13676 Phase 1 start in NHL/CLL
✓	XmAb18087 IND filing
✓	XmAb7195 Phase 1 subcutaneous trial data
✓	Additional partner milestones • Phase 3 start Alexion Xtend • Phase 3 start CSL/Janssen • Phase 3 start MorphoSys

2018	
	XmAb5871 Phase 2 SLE data
	XmAb5871 Phase 3 start in IgG4-RD
✓	XmAb18087 Phase 1 start
	XmAb14045 Phase 1 data
✓	XmAb20717 Phase 1 start
✓	Additional partner milestones • BLA/MAA filed Alexion Xtend

Cash 6/30/2018 $555 million

Runway into 2023

XmAb14045 data announcements pending alignment on timing with Novartis