

Reason for report:

INITIATION

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HEALTHCARE EQUITY RESEARCH

## XENCOR, INC.

### Unique Antibody Platform Drives Pipeline Targeting Large Markets; Initiate OP

• **Bottom Line:** We are initiating coverage of XNCR with an Outperform rating and an \$11 price target in 1 year. XNCR has used its proprietary "XmAb" platform to develop an antibody pipeline focused on asthma, autoimmune diseases and cancer and to generate incremental near-term revenue and long-term upside in form of technology licenses and partnerships. We believe XNCR's key value drivers are its 2 proprietary lead product candidates XmAb7195 and XmAb5871, addressing large market opportunities in allergic asthma and autoimmune diseases respectively. Both programs have clinical catalysts approaching in 2H14 that could serve as early clinical validation for those agents, thus potentially driving XNCR shares higher. In addition, we believe advances by XNCR's partners and licensees could provide additional incremental cash flows while new licenses could further validate XNCR's Fc-platform as a value driver.

• **Phase I IgE data in 2H14 could provide early validation for XmAb7195 as potential "biosuperior" of Xolair (Roche) for allergic asthma.** We believe XmAb7195 addresses a validated target and preclinical data has been promising thus far. Similar to Xolair, XmAb7195 is an anti-IgE monoclonal antibody (mAb), but in contrast to Xolair, it uses XNCR's proprietary immune-inhibitor Fc domain, which makes it a more potent inhibitor of IgE. We believe XmAb7195 could potentially be more efficacious in patients eligible for Xolair and potentially efficacious in 20% of patients who are currently ineligible for Xolair therapy due to body weight/IgE level limitations. We believe XmAb7195 could potentially address a patient population that is 20% larger than that of Xolair, which generated \$1.3Bn in global sales in 2012.

• **XmAb5871 is a unique B-Cell inhibitor with first Phase IIa rheumatoid arthritis (RA) data in 2H14. XmAb5871 simultaneously targets the B-cell proteins CD19 and FcγRIIb, which could have potential advantages over rituximab (anti-CD20) which has only limited utility in treating autoimmune diseases.** XmAb5871 Phase Ia data shows immunosuppression with only transient B-cell reduction and Phase IIa disease activity data in rheumatoid arthritis (RA) in 2H14 should provide further proof-of-concept for XmAb5871. We believe XmAb5871 could potentially be developed for a wide range of autoimmune diseases, including RA, lupus, or Sjögren syndrome among others. Positive Phase IIa data in 2H14, in our view, also increases the likelihood of partner AMGN (MP) to license the product following a larger controlled Phase IIb trial.

• **MOR208 is an antibody targeting CD19, which contains XNCR's high ADCC Fc domain, which has been outlicensed to MorphoSys (MOR), which is currently conducting two Phase II trials in ALL and NHL.** An investigator-sponsored study in CLL is ongoing and data from all three studies potentially in 2015 could validate MOR208's activity.

Dec Yr	1Q	2Q	3Q	4Q	FY Rev	1Q	2Q	3Q	4Q	FY EPS	
2012A	--	--	--	--	\$9.5	--	--	--	--	(\$118.86)	NM
2013E	--	--	\$8.4A	\$1.0	\$9.4	--	--	(\$4.10)A	(\$0.20)	(\$5.54)	NM
2014E	--	--	--	--	\$5.0	--	--	--	--	(\$0.78)	NM

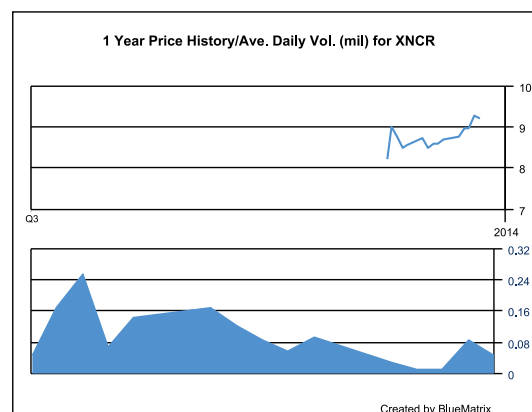
Source: Company Information and Leerink Swann LLC Research  
Revenues in \$MMs; Diluted GAAP EPS; Q313 represents Q1-Q3:13, pre-IPO

#### Key Stats:

(NASDAQ:XNCR)

<b>S&amp;P 600 Health Care Index:</b>	<b>1,285.86</b>
<b>Price:</b>	<b>\$9.20</b>
Price Target:	\$11.00
Methodology:	Sum of parts DCF w/ 12% discount rate
52 Week High:	\$10.90
52 Week Low:	\$5.75
Shares Outstanding (mil):	31.3
Market Capitalization (mil):	\$288.0
Cash Per Share:	\$31.95
Dividend (ann):	\$0.00
Dividend Yield:	0.0%

General: Shares outstanding accounts for IPO, which closed 12.06.13



Please refer to Pages 37 - 39 for Analyst Certification and important disclosures. Price charts and disclosures specific to covered companies and statements of valuation and risk are available at <https://leerink2.bluematrix.com/bluematrix/Disclosure2> or by contacting Leerink Swann LLC Publishing Department, One Federal Street, 37th Floor, Boston, MA 02110.



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*The Healthcare Investment Bank™*

# **Xencor, Inc. (NASDAQ: XNCR)**

## **Initiation of Coverage with Outperform**

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# Investment Thesis

- **Bottom Line: We are initiating coverage of XNCR with an Outperform rating and an \$11 price target in 1 year.** XNCR has used its proprietary “XmAb” platform to develop an antibody pipeline focused on asthma, autoimmune diseases and cancer and also to generate incremental near term revenue and long term upside in form of technology licenses and partnerships. We believe XNCR’s key value drivers are its two proprietary lead product candidates XmAb7195 and XmAb5871, both addressing large market opportunities in allergic asthma and autoimmune diseases respectively. Both programs have clinical catalysts approaching in 2H14 which could serve as early clinical validation for those agents, thus driving XNCR shares higher. In addition to that, we believe advances by XNCR’s partners and licensees could provide additional incremental cash flows while new licenses could further validate XNCR’s Fc-platform as a value driver.
- **Phase I IgE data in 2H14 could provide early validation for XmAb7195 as potential “biosuperior” of Xolair (Roche) for allergic asthma.** We believe XmAb7195 addresses a validated target and preclinical data has been promising thus far. Similar to Xolair, XmAb7195 is an anti-IgE mAb, but in contrast to Xolair it uses XNCR’s proprietary immune-inhibitor Fc domain which makes it a more potent inhibitor of IgE. We believe XmAb7195 could potentially be more efficacious in patients eligible for Xolair and potentially efficacious in 20% of patients who are currently ineligible for Xolair therapy due to body weight/IgE level limitations. We believe XmAb7195 could potentially address a patient population that is 20% larger than that of Xolair, which generated \$1.3Bn in global sales in 2012.
- **XmAb5871 is a unique B-Cell inhibitor with first Phase IIa rheumatoid arthritis (RA) data in 2H14.** XmAb5871 simultaneously targets the B-cell proteins CD19 and FcγRIIb which could have potential advantages over rituximab (anti-CD20) which has only limited utility in treating autoimmune diseases. XmAb5871 Phase Ia data shows immunosuppression with only transient B-cell reduction and Phase IIa disease activity data in rheumatoid arthritis (RA) in 2H14 should provide further proof-of-concept for XmAb5871. We believe XmAb5871 could potentially be developed for a wide range of autoimmune diseases, including RA, lupus, or Sjögren syndrome among others. Positive Phase IIa data in 2H14, in our view, also increases the likelihood of partner AMGN to license the product following a larger controlled Phase IIb trial.
- **MOR208 is an antibody targeting CD19 which contains XNCR’s high antibody-dependent cell cytotoxicity (ADCC) Fc domain** which has been outlicensed to MorphoSys (MOR) which is currently conducting two Phase II trials in acute lymphoblastic leukemia (ALL) and Non Hodgkin’s Lymphoma (NHL). An investigator-sponsored study in chronic lymphocytic leukemia (CLL) is ongoing and data from all three studies potentially in 2015 could validate MOR208’s activity.



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# Valuation and Risks

## Valuation:

- We estimate an \$11 per share value in 12 months for XNCR based on a discounted sales multiple analysis. We apply a 12% discount rate to for probability of success-weighted 2025 XmAb7195 (15%), XmAb5871 (20%), and MOR208 (30%) derived revenues. Our probability of success rates are higher than industry average of 9% for Phase I stage therapeutics, given our higher conviction of the respective programs' success rates. We apply a 6X multiple to XmAb7195 sales, reflecting current trailing Midcap (\$1-\$10Bn) biotech industry average and a 10X multiple to royalty streams for XmAb 5871 and MOR208. We also value XNCR's XmAb platform at \$100MM, applying a 15% probability of success to potential future royalties (5%) for XNCR's licensed portfolio. Based on our sum-of-parts analysis, we attribute \$2/share to XmAb7195, \$3/share to XmAb 5871, and \$2/share to MOR208. We also attribute \$3/share to the XmAb platform and \$2/share to expected cash in 12 months.

## Risks:

- Early stage developmental pipeline agents face high clinical and regulatory development risk, as well as commercial and competitive risks. As small cap biotech company, XNCR also faces execution risk and financial risk. We estimate that XNCR current cash will be sufficient to fund operations to 2017 assuming continued development of XmAb8195 and XmAb 5871, and the company may have additional financing needs before turning cash flow positive.

### Sum-of-the-parts DCF Forecast

	Valuation (\$MM)	Per Share
XmAb7195 (15% p/w)	71	\$ 2.26
XmAb5871 (20% p/w)	80	\$ 2.55
MOR208 (30% p/w)	51	\$ 1.64
XmAb Platform	100	\$ 3.19
Total EV	302	\$ 9.64
Cash (2014E)	54	\$ 1.73
<b>Price Target</b>	<b>356</b>	<b>\$ 11.38</b>
Diluted shares outstanding (2014E)	31	

	XmAb 7195	XmAb 5871	MOR208	Total
2025 sales (\$MM)	238	696	596	1,530
2025 royalty to XNCR (\$MM)	36	139	60	234
2025 total revenue to XNCR	273	139	60	472
Multiple	6	10	10	
Discount rate	12%	12%	12%	12%
Present value	471	400	171	1,043
Probability of success	15%	20%	30%	
<b>P/W NPV</b>	<b>71</b>	<b>80</b>	<b>51</b>	<b>202</b>

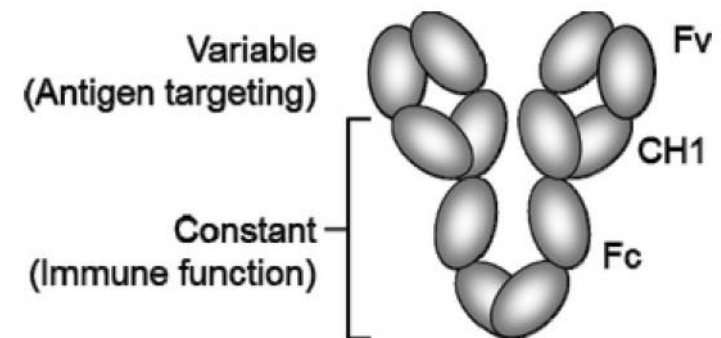


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# Fc Domain Engineering is Central to XNCR's "XmAb" Platform Technology

- ❑ Antibodies are composed of 2 structurally independent parts, the variable Fv domain, and the constant Fc and CHA domains.
  - ❑ The Fv domain is responsible for recognition of highly specific antigens and varies between antibodies.
  - ❑ The Fc domain interacts with receptors on immune cells and other cells to regulate an immune response.
- ❑ XNCR has identified certain amino-acid changes within Fc domains which can enhance antibody performance. Based on its discoveries, XNCR has developed or licensed IP around three categories of Fc domains which can be used to substitute "natural" Fc domains of therapeutic antibodies in a "plug-and-play" fashion.
  1. **Cytotoxic Fc Domain** — increased ADCC, targeting the receptors FcγRIIIa and FcγRIIa expressed on natural killer (NK) cells and macrophages
  2. **Immune Inhibitor Fc Domain** — selective immune inhibition and rapid target clearance, targeting the receptor FcγRIIb expressed on B-cells
  3. **Xtend Fc Domain** — extended antibody half-life, increases binding to the receptor FcRn on endothelial cells
- ❑ XNCR's Fc domains only involves 2 amino acid changes so that the Fc domains maintain over 99.5% identity to natural Fc domains.



Source: Company presentation





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# XmAb Domains are a Source of Technology Licenses which Could Generate Future Cash Flows

- ❑ XNCR's XmAb platform has been source of technology licenses (i) and product development licenses (ii) which generated \$65MM in cash flows over the last 5 years.
- ❑ Existing licenses and partnerships have the theoretical potential to receive \$1.3Bn in total milestones combined (\$240M clinical, \$541M regulatory, and \$526.5M sales milestones).
- ❑ **Technology Licenses:** aggregate potential milestones \$172.5M (\$65M clinical), \$36M regulatory, \$71.5M commercial); low single-digit royalties; minimal work commitment by XNCR:

Licensee	Year	Xencor Technology	Indication	Milestones	Royalties	Current Development Stage
BI	2007	Cytotoxic	Oncology	Yes	Yes	Phase 1 trials (two candidates)
Janssen R&D, LLC	2009	Xtend	Autoimmune disease	Yes	Yes	preclinical
CSL Limited	2009	Cytotoxic	Oncology	Yes	Yes	Phase 1
CSL Limited	2013	Xtend	Hematological diseases	Yes	Yes	Preclinical
Merck	2013	Fc optimization	Autoimmune disease	Yes	Yes	Preclinical

Source: Company Filings

- ❑ Technology License with Alexion (OP) (Jan 2013):
  - ❑ Alexion received a 5-year research license for Xtend around 6 targets and the option to obtain a worldwide license for each target to develop and commercialize products (\$4M option fee per target)
  - ❑ XNCR to receive: \$3M upfront, annual research maintenance fee of \$0.5M, up to \$66.5M milestones per target, low single-digit royalty on WW sales



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## XNCR Also Has Higher Value Product Specific Development Partnerships in Place

- ❑ **XmAb5871**: Research, develop and commercialization option agreement with AMGN (Dec 2010)
  - ❑ License option is exercisable until completion of a Phase IIb proof-of-concept trial or March 2017
  - ❑ XNCR is responsible for R&D and manufacturing activities for XmAb5871 until completion of data review following the Phase IIb proof-of-concept trial. If AMGN exercises the WW license option, it will be solely responsible for continued development, commercialization, and manufacturing costs
  - ❑ XNCR has the right to receive a \$50M exercise fee and up to \$439M milestones (\$64M clinical, \$150M regulatory, and \$225M commercial) plus tiered royalties in the high single-digit to high-teen percent range
  - ❑ XNCR already received \$11M upfront and \$2M for initiation of a Phase Ib/IIa trial in January, 2013
- ❑ **XmAb5574/MOR208**: License agreement with MorphoSys (MOR) (June, 2010)
  - ❑ MorphoSys has an exclusive worldwide license to XmAb5574/MOR208 for all indications
  - ❑ XNCR has been responsible for completing a Phase I trial of XmAb5574/MOR208 in CLL, which was completed in January 2013
  - ❑ MorphoSys is now responsible for all other development and commercialization activities.
  - ❑ XNCR received \$13M upfront and received \$3M development milestones in 2013 for initiation of Phase II trials
  - ❑ XNCR has the right to receive an additional \$299M in milestone payments (\$62M clinical, \$187M regulatory, and \$50MM commercial) plus high single-digit to low-teen percentage tiered royalties (until patent expiration or 11 years)



# Three Internally Developed Pipeline Assets are XNCR's Key Value Drivers

## 1. XmAb7195 is being developed for the treatment of severe asthma and allergic diseases.

- ❑ XmAb7195 is an anti-IgE mAb which uses XNCR's immune-inhibitor Fc domain. It has three specific mechanisms of action which give it potential advantages over Xolair (Xolair), Roche's approved IgE mAb: increased IgE binding, inhibition of IgE production and rapid clearance of IgE from circulation.
- ❑ XNCR plans to initiate Phase I clinical trials in 1H14, following promising preclinical data. Biomarker data is expected in 2H14.

## 2. XmAb5871 is being developed for the treatment of autoimmune diseases, including rheumatoid arthritis (RA) and lupus (SLE).

- ❑ XmAb5871 is an anti-CD19 mAb targeting B-cells and uses XNCR's immune-inhibitor Fc domain. Preclinical data indicates XmAb5871 exhibits potent B-cell inhibition and in contrast to Rituximab (anti-CD20) without long-term depletion.
- ❑ XNCR is currently conducting a Phase Ib/IIa clinical trial for XmAb5871 in RA patients with active disease on stable non-biologic disease-modifying antirheumatic drugs (DMARDs). Data is expected in 2H14.

## 3. MOR208 is being developed for the treatment of hematologic malignancies.

- ❑ MOR208 is an anti-CD19 mAb targeting B-cells and uses XNCR's high ADCC Fc domain. CD19 is expressed more broadly and earlier in B-cell development than CD20, the target of Rituxan (Roche), therefore potentially allowing for an even broader use of MOR208 in cancer therapy as compared to Rituxan.
- ❑ In a Phase Ib/IIa trial conducted by XNCR, MOR208 has shown encouraging signs of preliminary anti-tumor activity and an acceptable safety and tolerability profile in patients with chronic lymphocytic leukemia (CLL). In 2013, MorphoSys initiated two Phase II clinical trials of MOR208 in B-cell acute lymphoblastic leukemia (B-ALL) and non-Hodgkin's lymphoma (NHL). A third Phase II Study in CLL will evaluate MOR208 in combination with lenalidomide (Revlimid). First Phase II data could be available in 2015.





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# Three Key Value Drivers For XNCR

Agent	Target	Platform	Indication	Status	Event	Timing
XmAb5871	CD19	Immune-inhibitory FcγRIIb domain	Autoimmune	Phase Ib/IIa	File IND	1H14
					Phase Ib/IIa RA data	2H14
					Initiate Phase IIb	1H15
					Phase IIb data	1H17
					AMGN option exercise	1H17
XmAb7195	IgE	Immune-inhibitory FcγRIIb domain	Asthma	preclinical	Initiate Phase III, RA/SLE	2H17
					Initiate Phase Ia	1H14
					Phase Ia data	4Q14
					Initiate Phase Ib	1Q15
					Phase Ib data	1H16
MOR208/XmAb5574	CD19	ADCC enhancing FcγRIIIa domain	Hematological Cancers	Phase II	Initiate Phase II POC	1H16
					Phase II POC data	1H18
					Initiate Phase II CLL trial (Revlimid combo)	1Q14
					Phase II data NHL	4Q15
					Phase II data ALL	4Q15

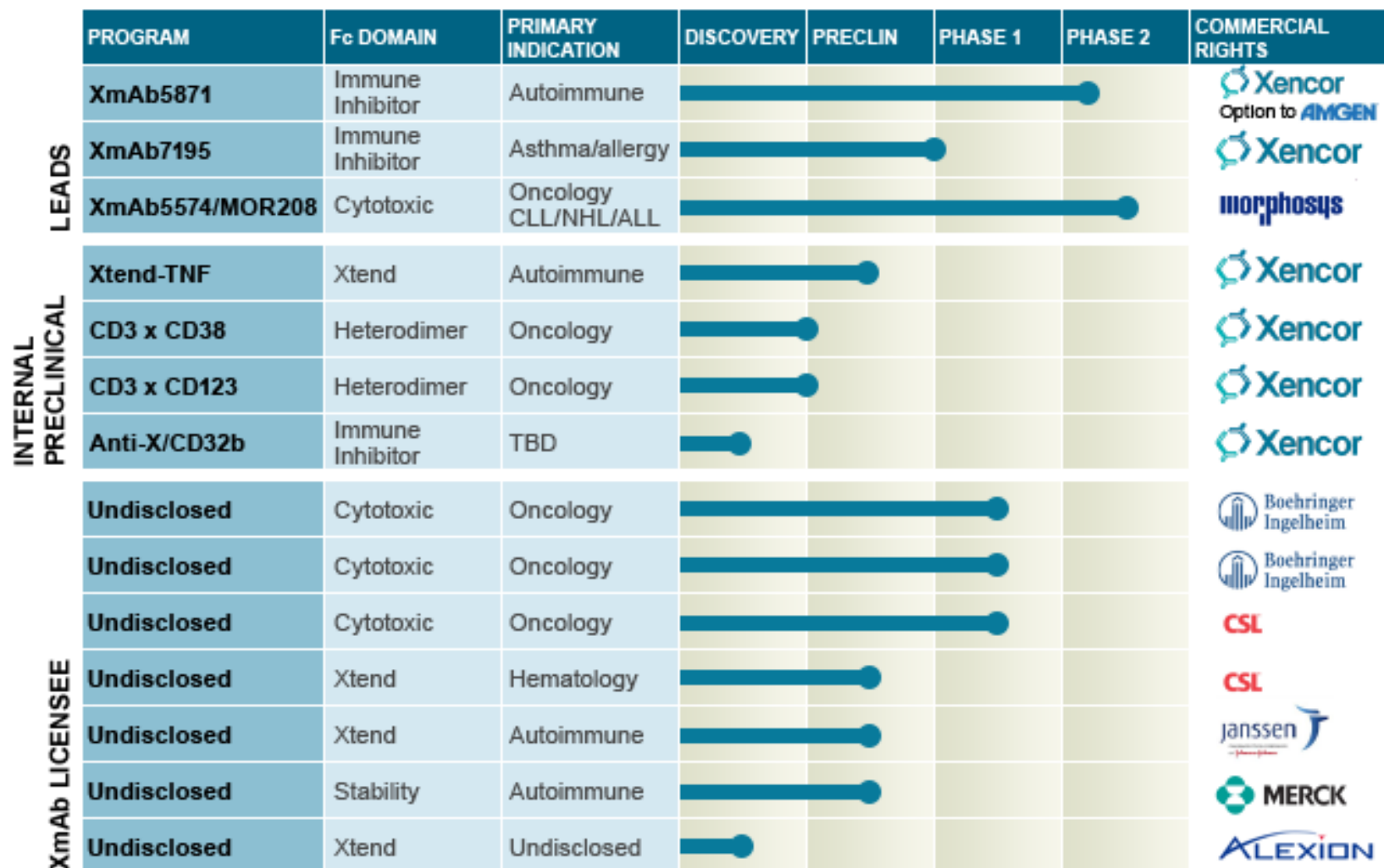
Source: Company Filings and Leerink Swann Estimates



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# Pipeline Overview



Source: Xencor



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# **XMAB 7195**



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# XmAb7195 is Positioned as Potential Xolair “Biobetter”

- ❑ **XmAb7195 is a monoclonal antibody targeting anti-IgE which contains XNCR’s Immune inhibitor Fc domain.** It is potentially differentiated from Xolair (Xolair) in three ways:
  1. Three times higher affinity to the same IgE epitope as Xolair
  2. Potential inhibition of IgE production via FcγRIIb co-stimulation on B-cells (400-fold higher affinity for FcγRIIb than Xolair)
  3. More rapid clearance of XmAb7195-bound IgE from circulation due to FcγRIIb co-stimulation on liver endothelial cells
- ❑ **Xolair is a recombinant humanized IgG1 monoclonal anti-IgE antibody that binds to the IgE molecule at the same epitope on the Fc region that binds to FcεRI.** This design means that Xolair blocks interaction of IgE from attachment to surfaces of effector cells. An 89% to 99% reduction in free serum IgE occurs soon after the administration of Xolair and low levels persist throughout treatment. Xolair treatment also reduces the density of IgE-loaded high affinity Fc receptors (FcεRI) on IgE effector cells. A reduction in the expression of FcεRI on basophils and mast cells decreases the binding of circulating IgE, thus preventing the release of inflammatory mediators upon allergen challenge. IgE naturally also binds to low-affinity Fcε receptors (FcεRIIs, CD23) on B-cells, where it alters differentiation and regulation of further IgE synthesis.
- ❑ **Total serum IgE level is required to be measured in all patients who are being considered for treatment with Xolair,** because the dose of Xolair is determined on the basis of the IgE baseline level and body weight. The recommended dose is 0.016 mg per kilogram of body weight per international unit of IgE every four weeks, administered subcutaneously at either two-week or four-week intervals. This dose is based on the estimated amount of the drug that is required to reduce circulating free IgE levels to less than 10 IU per milliliter.
- ❑ **XNCR believes that due to its properties, XmAb could potentially overcome two of Xolair’s key limitations:**
  - ❑ Due to its higher potency, 7195 it may be able to treat an additional 20% of asthma patients that have high body weight and high IgE levels which are currently ineligible for Xolair therapy.
  - ❑ 7195 may have better efficacy in Xolair-eligible patients or in Xolair “non-responders”

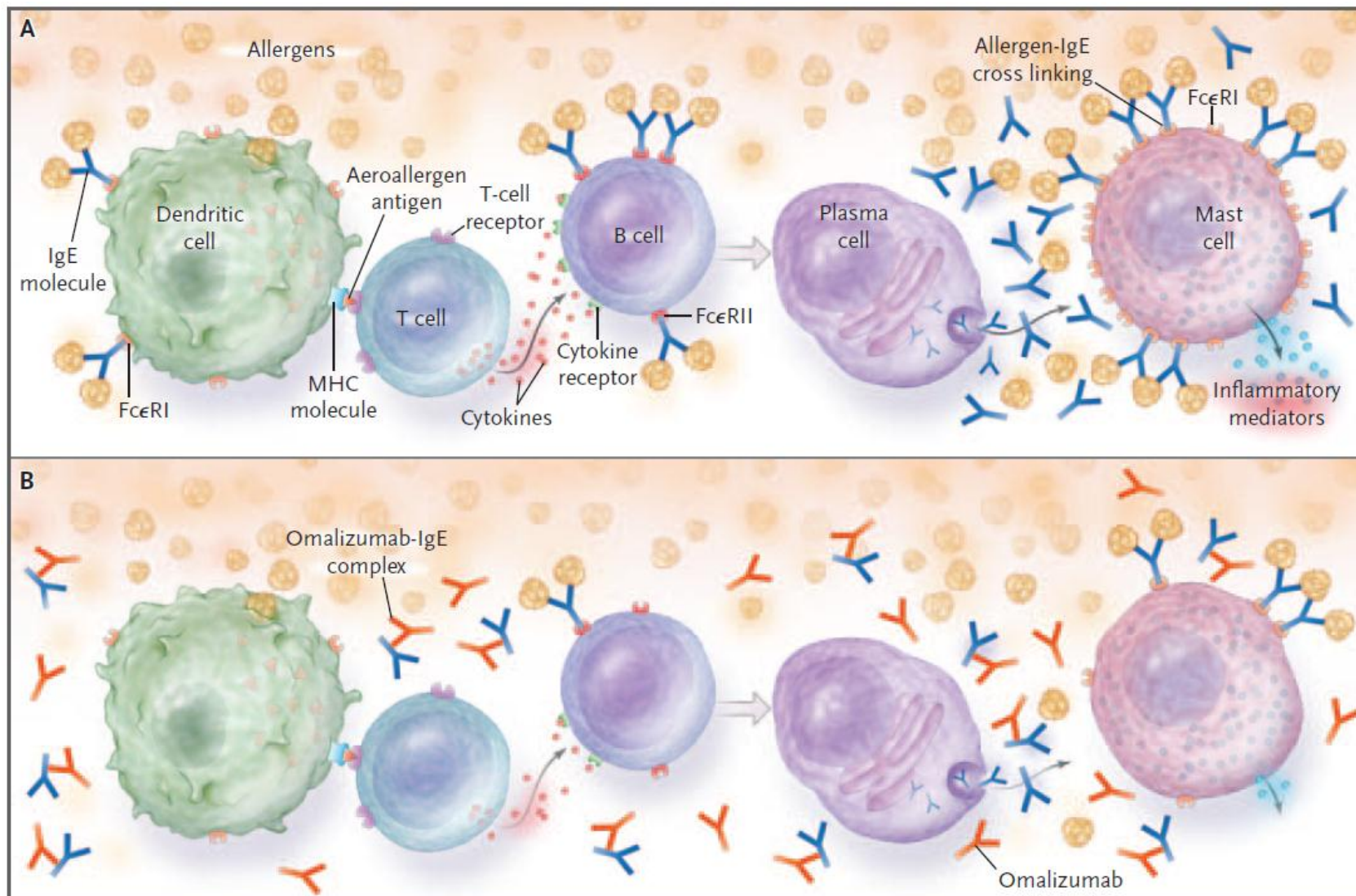




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# How Xolair Works



Source: New England Journal of Medicine



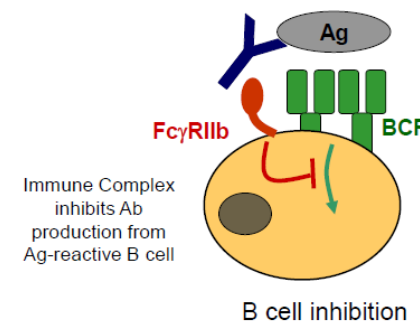
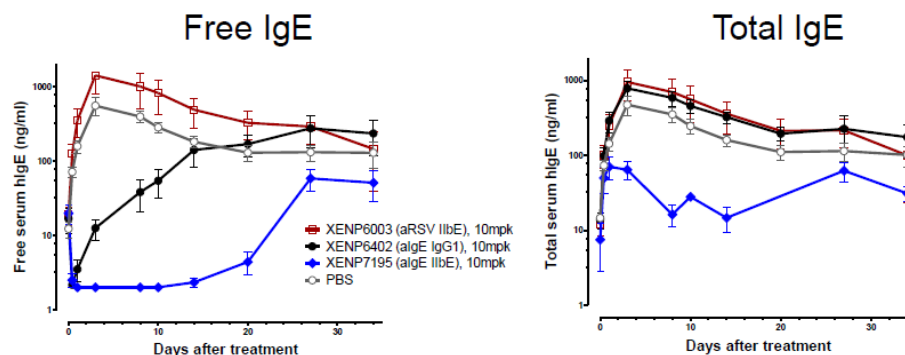


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# Mechanism of XmAb 7195 May be Differentiated Since It Binds to FcγRIIB, In Addition to IgE

- ❑ In addition to strong binding affinity to IgE which is mediated by its Fv domain, XmAb 7195 also binds to the inhibitory Fc receptor FcγRIIB.
- ❑ **FcγRIIB is the only FcγR that has an inhibitory function.** The receptors for the Fc region of IgG (FcγRs) are expressed by many immune cells and are important in regulating the immune and inflammatory response to immune complexes. Most FcγRs are activating receptors and include the high-affinity receptor FcγRI and a family of low affinity receptors, including FcγRIIA, FcγRIIC, FcγRIIIA and FcγRIIIB in humans.
- ❑ **The main function of FcγRIIB is to inhibit activating signals, which is achieved through co-ligation of FcγRIIB with either activating FcγRs or with the B-cell receptor (BCR) by immune complexes.** FcγRIIB regulates B-cell activation by increasing the BCR activation threshold and suppressing B-cell mediated antigen presentation to T-cells. In addition, cross-linking FcγRIIB in the absence of BCR ligation can induce apoptosis of mature B cells. Recent studies have shown that cross-linking FcγRIIB on plasma cells can also result in apoptosis. In addition to its effect on B-cells, FcγRIIB inhibits FcγR-dependent internalization and presentation of antigen, as well as subsequent T cell priming.
- ❑ **Lower and more prolonged free and total IgE reduction in mouse models** (see below) can be interpreted as a result of a higher elimination rate of XmAb7196-IgE immune complexes or less production of new IgE or a combination thereof. According to XNCR, 7195 and Xolair bind the Fc domain of membrane anchored IgE on B-cells. 7195, but not Xolair, then engages the B cell FcγRIIb and inhibits the cell from differentiating and secreting soluble IgE.



Source: Compartmental

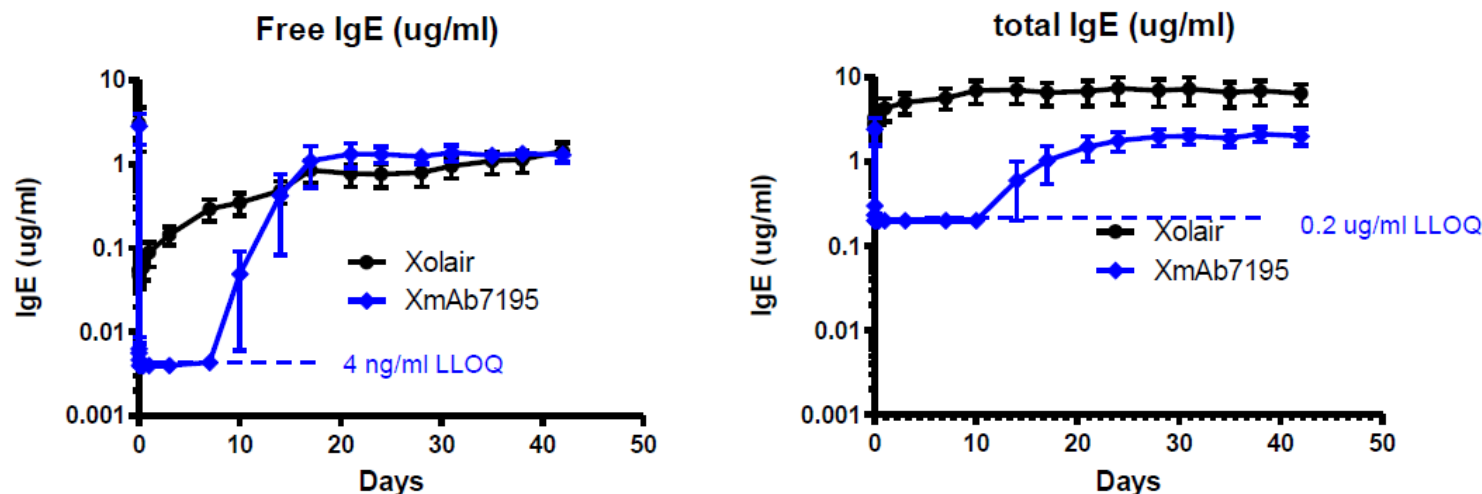


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# XmAb7195 Preclinical Data Suggest Greater Reduction in Free and Total IgE than Xolair

- ❑ **XmAb7195 is able to reduce free IgE by one order of magnitude lower levels than Xolair in chimpanzees.** Chimpanzees normally have IgE levels that, in humans, would be considered ineligible for Xolair. XmAb7195 was able to reduce free IgE levels below 0.004  $\mu\text{g/ml}$  (limit of detection). Xolair was able to reduce free IgE levels to 0.050  $\mu\text{g/ml}$  (3 chimpanzees per arm). The reduction caused by Xolair was also more transient than the reduction caused by XmAb7195.
- ❑ **Rapid reduction in total IgE in chimpanzees consistent with suggested mechanisms of action.** These proposed MoAs include both the engineered Fc domain reducing IgE production, and the rapid clearance of IgE bound to XmAb7195. Total IgE measurements reflect both, free IgE and IgE-anti-IgE immune complexes.
- ❑ **XmAb7195 is well tolerated in multiple dose toxicology studies in cynomolgus monkeys up to 100 mg/kg.** This study lasted 12 weeks with no adverse effects observed. XmAb7195 was also well tolerated at 5 mg/kg at both single and multiple doses in chimpanzees.





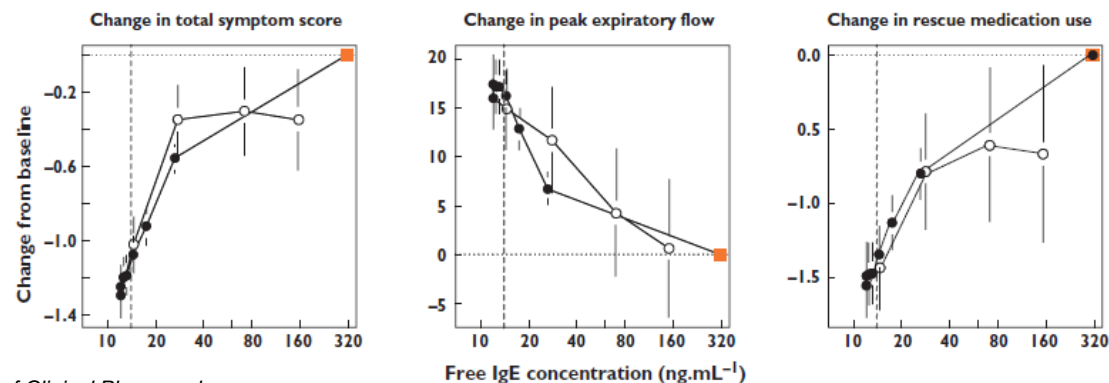
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# XmAb7195 Phase I IgE Data in Late 2014 Should Provide Early Proof-of-Concept in Humans

- ❑ **XNCR plans to file an IND for XmAb7195 for asthma with the FDA and to initiate a Phase Ia clinical trial in 1H14.** The Phase Ia single ascending dose clinical trial in healthy volunteers will include parallel cohorts in allergen-sensitive subjects with high IgE levels. This clinical trial will be designed to study safety and pharmacokinetics in humans and validate XmAb7195's ability to suppress both free and total IgE levels and to report preliminary data at the end of 2014.
- ❑ **We believe Phase Ia data in late 2014 could potentially provide early validation for XNCR's approach.** The causal role of IgE and asthma is well established and according to a study by *Lowe et al.* (British Journal of Clinical Pharmacology, 2009), IgE reduction is correlated with asthma symptom scores and peak expiratory flow, a measure of airway obstruction in the lungs.
- ❑ **If Phase Ia clinical data is positive, XNCR anticipate starting a Phase Ib multiple ascending dose clinical trial of XmAb7195 in healthy adult volunteers and in patients with mild-to-moderate asthma in early 2015** to study safety, pharmacokinetics, and IgE reduction. XNCR has received correspondence from the FDA following a pre-IND meeting request that concurred with XNCR's plans, pending review of a full IND submission.
- ❑ **Following the Phase Ia and Ib clinical trials, XNCR anticipates initiating a Phase II Point-of Care (POC) clinical trial of XmAb7195 in 1H16** for intermediate-term treatment of patients with poorly-controlled asthma, including patients with high IgE levels and/or high body mass. Dosing for this clinical trial will be based on data from the Phase I studies.

## Correlation between free IgE and Asthma Symptoms based on Xolair Phase III Data (4 trials pooled)



Source: Lowe et al. 2009 British Journal of Clinical Pharmacology



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# XmAb7195 Could Potentially Be Positioned as “Biobetter” to Xolair

- We believe due to its Method of Action (MoA), 7195 could be potentially efficacious in an estimated 20% of patients currently not eligible for Xolair due to high baseline serum IgE or body weight since it would require a monthly dose of Xolair of more than 750mg.
- We also believe 7195 could have potential superior efficacy in currently Xolair-eligible patients at equal doses in achieving target serum free IgE levels. Based on clinical response in Xolair Phase I and II trials, 25 ng/mL was set as the target serum free IgE level which ensured that 95% of patients in Phase III achieved free IgE levels of 50 ng/mL which was related to improved clinical outcomes.
- The FDA approved Xolair in 2003, to treat adults and adolescents (12 years of age and above) with moderate to severe persistent asthma who have a positive skin test or in vitro reactivity to a perennial aeroallergen and whose symptoms are inadequately controlled with inhaled corticosteroids. Initial approval was based on data from two nearly identically designed Phase III trials showing a reduction in asthma exacerbations.

## Xolair Dosing Table

Administration Every 2 Weeks

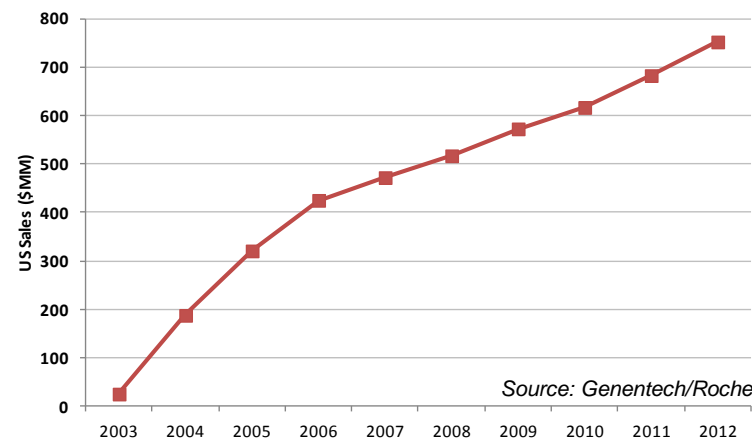
Xolair Doses (milligrams) Administered by Subcutaneous Injection  
Every 2 Weeks for Adults and Adolescents 12 Years of Age and Older

Pre-treatment Serum IgE (IU/mL)	Body Weight (kg)			
	30–60	> 60–70	> 70–90	> 90–150
≥ 30–100	SEE TABLE 1			
> 100–200				
> 200–300		225	225	300
> 300–400	225	225	300	DO NOT DOSE
> 400–500	300	300	375	
> 500–600	300	375		
> 600–700	375			

Source: Xolair Label

## Xolair US Sales

### Xolair US Net Sales (\$MM)





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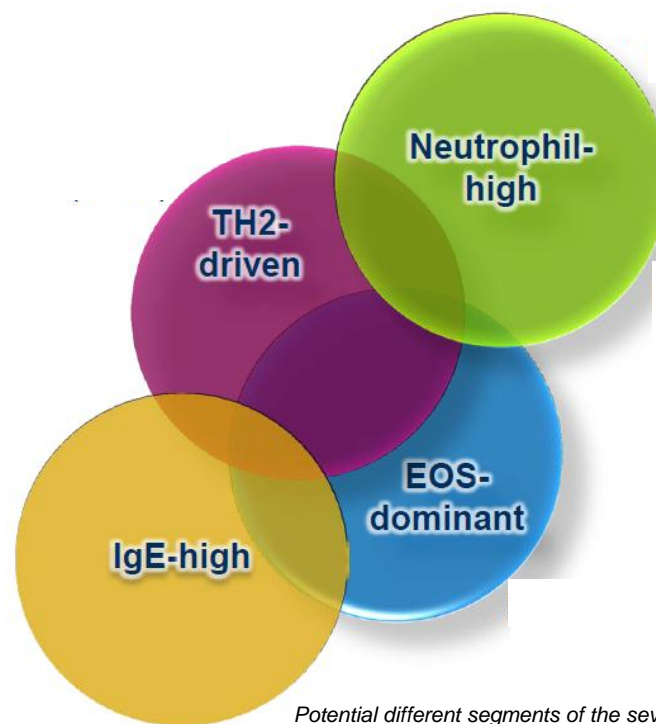
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# Several Antibodies are in Clinical Development for Asthma ...

- ... But we believe these target different segments of the severe asthma spectrum. We believe as a potentially superior anti-IgE therapeutic XmAb7195 could coexist with new biologics which could enter the market over the next few years.
- Additional anti-IgE mAbs in development include agents by Roche and PFE (MP). Interestingly, Roche's quilizumab was designed to target IgE-producing B-cells targeting the M1 prime segment of membrane IgE. Whereas Roche's Phase I showed, in our view, promising blockage of allergen-induced increases in serum IgE levels, we believe 7195's combined sequester of free IgE and potential reduction in IgE production is a more intriguing MoA (Method of Action).

Agent	Target	Company	Status
Benralizumab	IL-5/IL-5R	AZN	Phase III
Bosatria	IL-5/IL-5R	GSK	Phase III
Cinquil	IL-5/IL-5R	TEVA	Phase III
Lebrikizumab	IL-13	Roche	Phase III
Tralokinumab	IL-13	AZN	Phase IIb
Brodalumab	IL-17R	AMGN	Phase II
Dupilumab	IL-4R	REGN/SNY	Phase II
KB003	GM-CSF	KBIO	Phase II
QAX-576	IL-13R	NVS	Phase II
QBX258	IL-13	NVS	Phase II
Quilizumab	IgE	Roche	Phase II
Secukinumab	IL-17	NVS	Phase II
QGE031	IgE	NVS	Phase I/II
PF-06444752	IgE	PFE	Phase I
RPC4046	IL-13	RCPT	Phase I

Source: BioMedTracker, ClinicalTrials.gov



Potential different segments of the severe asthma spectrum  
Source: AZN presentation





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# XmAb7195 Model

US Asthma Model (in 1,000s)		2021E	2022E	2023E	2024E	2025E	2026E	2027E	2028E	2029E	2030E
Diagnosed asthmatics	1%	20,290	20,493	20,698	20,905	21,114	21,325	21,538	21,754	21,971	22,191
Treated asthmatics	54%	10,957	11,066	11,177	11,289	11,402	11,516	11,631	11,747	11,864	11,983
Moderate and severe	40%	4,383	4,426	4,471	4,515	4,561	4,606	4,652	4,699	4,746	4,793
Uncontrolled	55%	2,410	2,435	2,459	2,483	2,508	2,533	2,559	2,584	2,610	2,636
Allergic (IgE mediated)	60%	1,446	1,461	1,475	1,490	1,505	1,520	1,535	1,551	1,566	1,582
Within Xolair Dosing Table	67%	969	979	988	998	1,008	1,018	1,029	1,039	1,049	1,060
Seen by AIs, PUDs, and select PCPs	43%	417	421	425	429	434	438	442	447	451	456
Not eligible for Xolair	20%	104	105	106	107	108	109	111	112	113	114
Patients eligible for XmAb7195		521	526	531	537	542	547	553	558	564	570
XmAb7195 Penetration		0.5%	1.0%	2.0%	3.0%	4.0%	5.0%	5.5%	6.0%	6.5%	7.0%
Patients treated with XmAb7195		3	5	11	16	22	27	30	34	37	40
Annual WAC/patient (\$)	4%	15,000	15,600	16,224	16,873	17,548	18,250	18,980	19,739	20,529	21,350
XmAb7195 US Sales (\$MM)		39	82	172	272	380	500	577	661	753	851
XmAb7195 Ex-US RR (\$MM)	15%	6	12	26	41	57	75	87	99	113	128
<b>7195 WW Revenue (\$MM)</b>		<b>45</b>	<b>94</b>	<b>198</b>	<b>312</b>	<b>437</b>	<b>574</b>	<b>664</b>	<b>761</b>	<b>865</b>	<b>979</b>

## Assume launch in 2021

2025 Revenue (\$MM)	437
Multiple	6
Discount rate	12%
Present value	754
Probability of success	10%
P/W NPV	75

Source: Leerink Swann Estimates



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# XMAB 5871

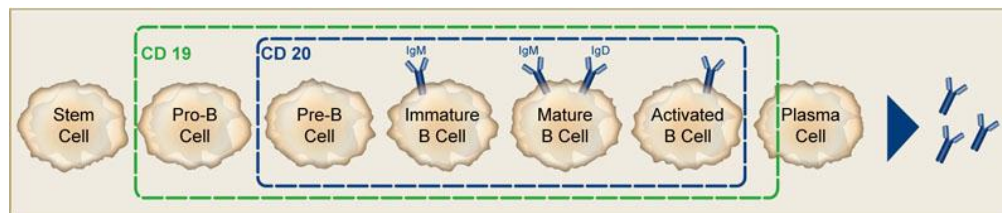


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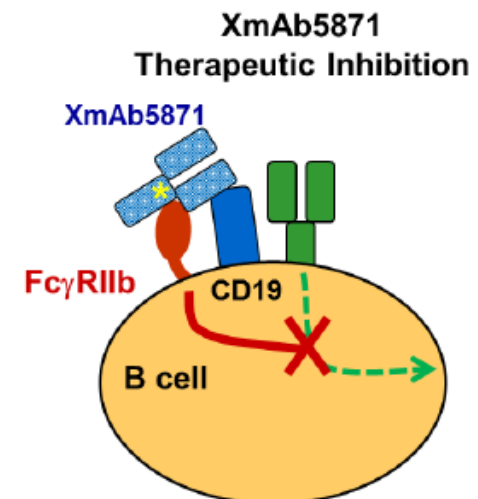
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# XmAb5871 is a Broad B-Cell Inhibitor in Development for Autoimmune Diseases

- ❑ **XmAb5871 is a monoclonal antibody targeting CD19 which contains XNCR's Immune inhibitor Fc domain.** XNCR develops XmAb5871 for the treatment of autoimmune diseases, including rheumatoid arthritis (RA) and lupus (SLE).
- ❑ **XmAb5871's, mechanism of action (MoA) is highly differentiated from other agents, including AMGN's rituximab (Rituxan, anti-CD20).** 5871's Fv domain utilizes CD19 targeting to bind B-cells and its Fc domain to crosslink the inhibitory FcγRIIb. By simultaneously targeting the B-cell proteins, CD19 and FcγRIIb, XmAb5871 has an ability to engage the natural inhibitory pathway provided by FcγRIIb, preventing further activation of B-cells by autoantigens and potentially also suppressing the ability of B cells to further provoke downstream autoimmune responses from T cells.
- ❑ **CD19 and FcγRIIb are expressed broadly throughout B-cell development,** so XmAb5871 may confer broad but temporary suppression of B-cell activation and downstream events such as antibody production.
- ❑ **Rituximab also targets B-cells via cell-surface antigen CD20, but depletes B-cell populations long term via ADCC.** As a result, rituximab is widely used across several hematological B-cell cancers, but has only limited utility in autoimmune diseases such as RA or SLE. Rituximab is FDA-approved for treatment of RA, but based on our MEDACorp checks only used as drug of last resort after failure of TNF-targeted agents. Based on a prior MEDACorp [survey](#), we believe rituximab is used off-label in ~5% of patients with SLE.



Source: MorpoSys



Source: Company slides



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## XmAb5871 Phase Ia Data Show Solid Immunosuppression and Only Transient B-Cell Reduction

- ❑ **Phase Ia data in 48 healthy volunteers showed drug is well tolerated and has good immunosuppressive activity based on biomarkers observed during the trial.** Subjects received a single intravenous (IV) infusion of XmAb5871 or placebo in one of seven dose cohorts ranging from 0.03 mg/kg to 10.0 mg/kg.
  - ❑ **B-cells were reduced by up to ~50% from baseline at all doses.** The extent of the B-cell reduction did not increase as dose level increased, and B-cell counts recovered to pre-treatment values with the clearance of XmAb5871
  - ❑ **XmAb5871 suppressed CD86, a B-cell activation marker at all doses;** recovery of B-cell activation was concurrent with the clearance of XmAb5871 from the subjects' serum.
  - ❑ **XmAb5871 reduced responses to tetanus toxoid vaccination,** a model for B-cell inhibition
  - ❑ **XmAb5871 was able to reduce anti-tetanus antibody responses** at doses 0.1, 0.2, 0.6, 2, 5, and 10mg/kg. Placebo treated subjects showed an increase in anti-tetanus antibody levels of over 12-fold vs. a 4-fold increase for XmAb 5871 treated subjects.
- ❑ **The most frequently reported adverse events associated with XmAb5871 were gastrointestinal symptoms** including nausea, vomiting, abdominal pain, abdominal discomfort, epigastric discomfort (upper stomach pain) and diarrhea. All but one were mild to moderate, with one subject experiencing severe nausea. 44% of subjects had anti-drug antibodies (immunogenicity), with only half of these subjects having an immunogenicity signal greater than two-fold above baseline. These antibody responses did not appear to impact drug activity or disposition.
- ❑ **XNCR plan to file an IND for XmAb5871 with the FDA in 2014.** To date, all clinical development for XmAb5871 has been conducted in Western and Central Europe. XNCR have examined the ability of XmAb5871 to inhibit B-cells in preclinical studies, including in vitro and in vivo studies. XNCR observed no depletion of human B-cells in culture, inhibition of human B-cells stimulated by a variety of agents, and suppression of antibody responses in humanized mouse models. XNCR also observed suppression of disease in mouse models of arthritis and multiple sclerosis without B-cell depletion and 5871 was well tolerated at high doses in monkeys. In addition, FcγRIIb overexpression on B-cells in mice suppresses autoimmune disease, which suggests that activating FcγRIIb via B cell-specific antibody could also be efficacious. XNCR completed 12-week and 24-week, multiple dose, preclinical monkey toxicology studies and found no AEs in doses up to 200 mg/kg.



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# XmAb5871 Phase Ia Data Indicates B-cell Inhibition

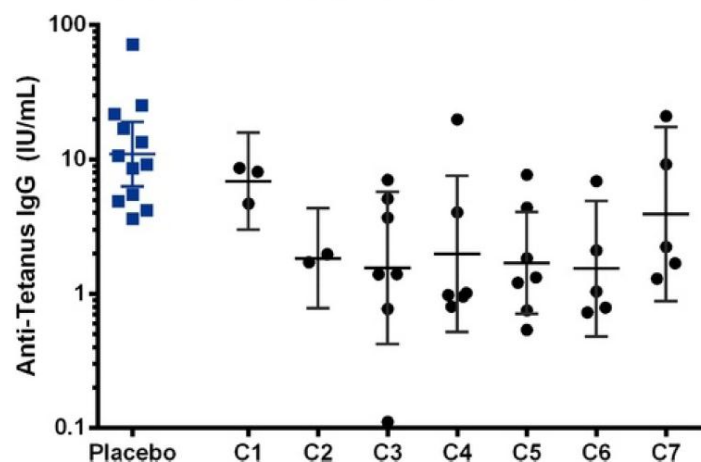
B cells were transiently reduced, but not depleted  
(Group mean B cell counts over time)

Cohort	Dose (mg/kg)	Days								
		1	2	4	8	15	22	29	43	71
Placebo	0	100	112	101	92	99	84	87	94	108
C1	0.03	100	59	52	73	107	77	92	99	97
C2	0.1	100	48	44	39	83	77	73	84	76
C3	0.2	100	59	59	44	62	68	86	89	92
C4	0.6	100	56	48	44	53	76	85	92	137
C5	2	100	60	59	41	49	54	72	105	99
C6	5	100	79	68	47	53	49	57	71	92
C7	10	100	71	62	49	50	52	44	69	87

> 70%  
30 - 70%  
< 30%

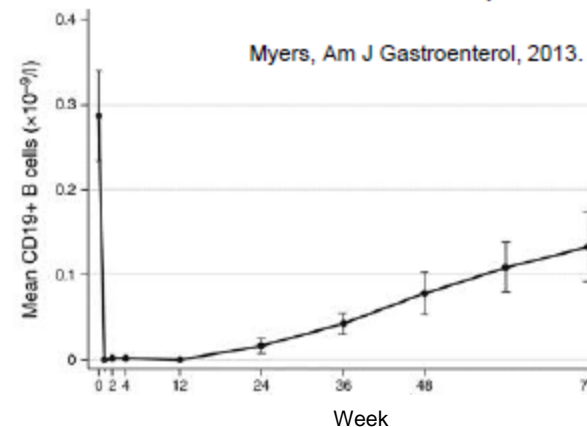
Source: Company presentation

anti-tetanus, 3 weeks after immunization



Source: Company filings

Rituxan Has Months of Depletion



Myers, Am J Gastroenterol, 2013.

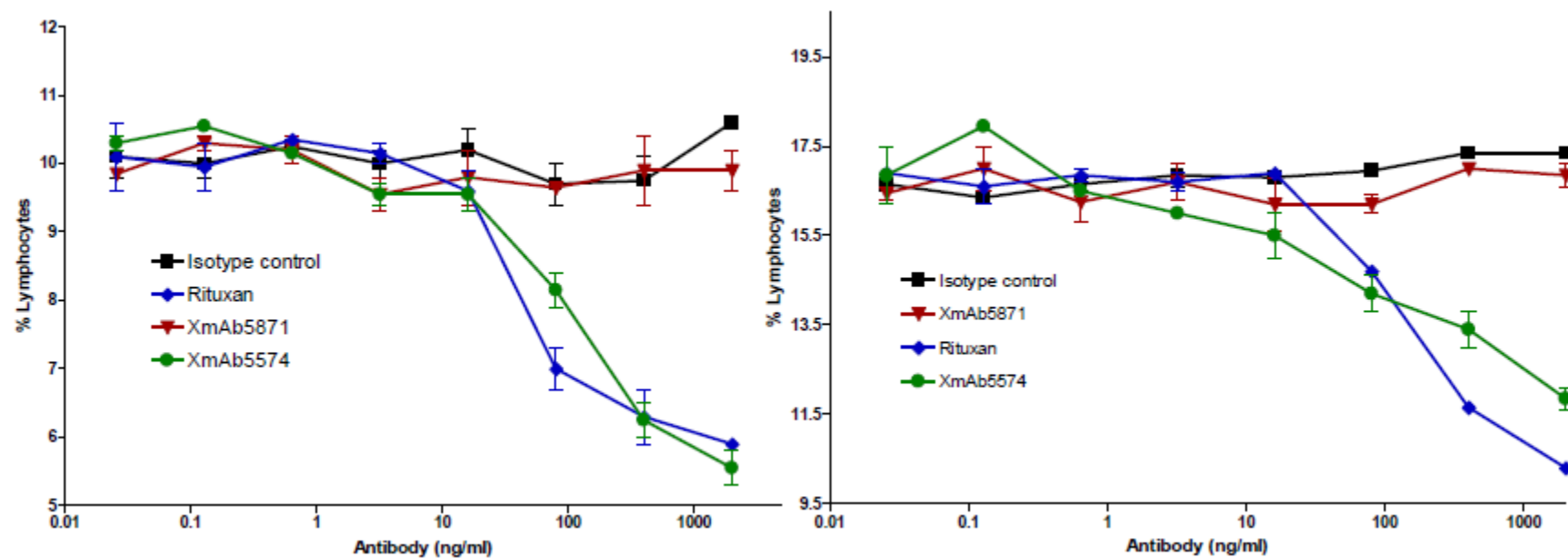




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# XmAb5871 In-Vitro Data Indicates B-cell Inhibition without Depletion



Source: Company presentation



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## Phase IIa Disease Activity Data in RA in 2H14 Should Provide Proof-of-concept

- ❑ **A Phase Ib/IIa clinical trial of XmAb5871 was initiated in January 2013.** This clinical trial is a multi-center, randomized, placebo-controlled, double-blinded, ascending multiple dose study of the safety, tolerability, pharmacokinetics and pharmacodynamics of XmAb5871 in RA patients with active disease on stable non-biologic c therapy.
- ❑ **In Phase Ib, 29 rheumatoid arthritis patients with active disease on stable non-biologic disease-modifying anti-rheumatic drug (DMARD) therapy have been enrolled** into four consecutive dose cohorts (0.3 to 10.0 mg/kg) randomized approximately 6:2 (XmAb5871 to placebo), other than for the lowest dose, where it was 3:1. Each patient will be administered XmAb5871 or placebo every 14 days for a total of six doses. XNCR has enrolled the fourth and highest dose cohort.
- ❑ **Through September 30, 2013, XmAb5871 has been reported to be well-tolerated.** One patient has experienced a serious adverse event (infusion-related reaction with hypotension), and this patient has discontinued the study prematurely. Other adverse events that have been reported by investigators as related to treatment and have occurred in more than one patient include: nausea, vomiting, fever increased temperature, headache and bronchitis. Preliminary immunogenicity testing data for the first 2 cohorts have been negative.
- ❑ **In Phase IIa, 30 additional rheumatoid arthritis patients with active disease on stable non-biologic DMARD therapy will be enrolled** in an expansion cohort, randomized 2:1 at the highest dose studied in Phase Ia (10mg/ml). Each patient will be administered XmAb5871 or placebo every 14 days for a total of six doses. The trial will assess response as measured by changes in Disease Activity Score 28 using C-reactive protein (DAS28 CRP) at Week 13.
- ❑ **AMGN may exercise its license option after Phase IIb.** Further planned clinical trials include an intravenous to subcutaneous bridging study. XNCR also plans to initiate a Phase IIb POC clinical trial in 1H15 and expects to enroll 150-200 moderate-to-severe rheumatoid arthritis patients on stable DMARD therapy. This clinical trial will be designed to assess efficacy at 24 weeks. Data from this trial, if positive, will support pivotal Phase II plans in RA and potentially trigger the AMGN license.
- ❑ **Additional auto-immune indications are a further source of upside.** XNCR may explore the utility of XmAb5871 in other diseases, including multiple sclerosis, myasthenia gravis, Sjogren's syndrome and a variety of orphan diseases.



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# XmAb 5871 Model

- Given its potentially broad utility in treating B-cell mediated autoimmune diseases, we believe XmAb5871 may potentially address a large market opportunity, if developed and marketed successfully. Based on its MoA, we believe future development in RA and lupus (SLE) are most likely, although XNCR/AMGN may develop the product in a range of additional rarer autoimmune diseases where autoantibodies are believed to play a role.

Year	2021E	2022E	2023E	2024E	2025E	2026E	2027E	2028E	2029E	2030E	2031E	2032E
US+EU RA pts diagnosed (1,000 pts)	4,223	4,265	4,308	4,351	4,395	4,439	4,483	4,528	4,573	4,619	4,665	4,712
Mod/severe RA	3,032	3,062	3,093	3,124	3,155	3,187	3,219	3,251	3,283	3,316	3,349	3,383
Treated	2,599	2,625	2,651	2,678	2,704	2,731	2,759	2,786	2,814	2,842	2,871	2,899
Treated w/ Biologics	964	973	983	993	1,003	1,013	1,023	1,033	1,044	1,054	1,065	1,075
1st line DMRAD pts	514	519	524	529	535	540	545	551	556	562	567	573
2nd line after DMRAD pts	450	455	459	464	468	473	478	482	487	492	497	502
5871 penetration	1.0%	1.5%	2.0%	3.0%	3.4%	4.4%	5.4%	5.9%	6.4%	6.9%	7.4%	7.9%
5871 pts (1,000s) (RA)	5	7	9	14	16	21	26	29	31	34	37	40
SLE Prevalence (1,000 pts) (US+EU)	704	711	718	725	732	740	747	755	762	770	777	785
Mod/severe SLE pts treated	433	437	442	446	451	455	460	464	469	474	478	483
5871 penetration	1.0%	1.5%	2.0%	3.0%	3.4%	4.4%	5.4%	5.9%	6.4%	6.9%	7.4%	7.9%
5871 pts (1,000s) (SLE)	4	7	9	13	15	20	25	28	30	33	36	38
Cost of therapy/pt (\$)	20,000	20,500	21,013	21,538	22,076	22,628	23,194	23,774	24,368	24,977	25,602	26,242
Sales (\$MM) (SLE + RA)	177	274	379	588	696	931	1,181	1,335	1,499	1,672	1,856	2,051
Royalty to XNCR	35	55	76	118	139	186	236	267	300	334	371	410

## Assume launch in 2021

2025 WW Sales (\$MM)	696
2025 WW Royalty (\$MM)	139
Multiple	10
Discount rate	12%
Present Value (\$MM)	400
Probability of success	20%
P/W NPV	80

Source: Leerink Swann Estimates



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# MOR 208

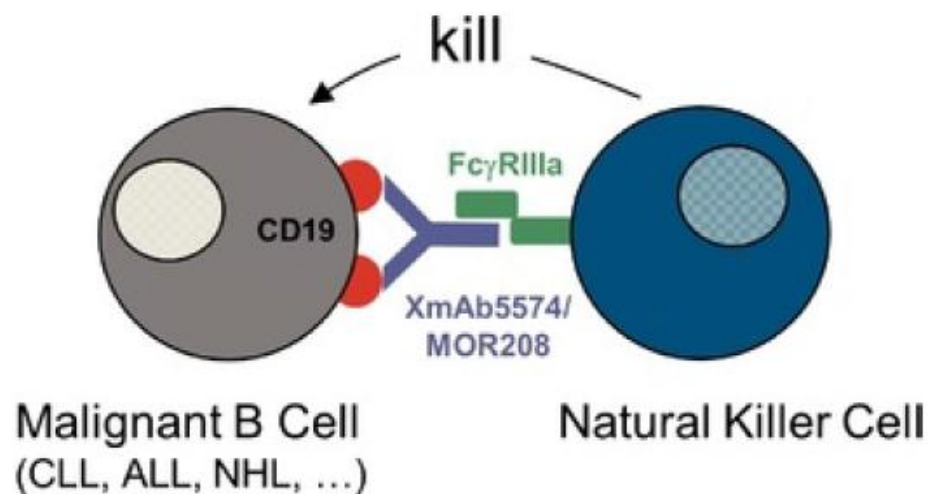


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## MOR208/XmAb5574 is being Developed Across a Broad Spectrum of Hematological Malignancies

- ❑ **MOR208 is a monoclonal antibody targeting CD19 which contains XNCR's high ADCC Fc domain.** In contrast to XmAb5871, which uses the immune inhibitor Fc domain, XmAb5574 uses the cytotoxic Fc domain with the intent of depleting B-cells in B-cell cancers. The Fc domain has enhanced binding to Fc receptors FcγRIIIa and FcγRIIa, which enhances recruitment of natural killer cells.
- ❑ **XmAb5574 is in development by MorphoSys (MOR) for hematological cancers.** CD19, the target of MOR208's Fv domain, is a B-cell surface protein that is highly expressed on the tumor cells in NHL and many leukemias, including ALL and CLL.
- ❑ **CD19 as a target may allow XmAb5574 to target a broader spectrum of lymphoid malignancies than rituximab.** CD19 is expressed earlier in B cell development and persists longer through B-cell maturation as compared to CD20, the target of rituximab. XmAb5774/MOR208 may be able to target a broader spectrum of lymphoid malignancies, such as ALL and CLL, where Rituxan (which targets CD20) may have limited efficacy.



Source: Company filings



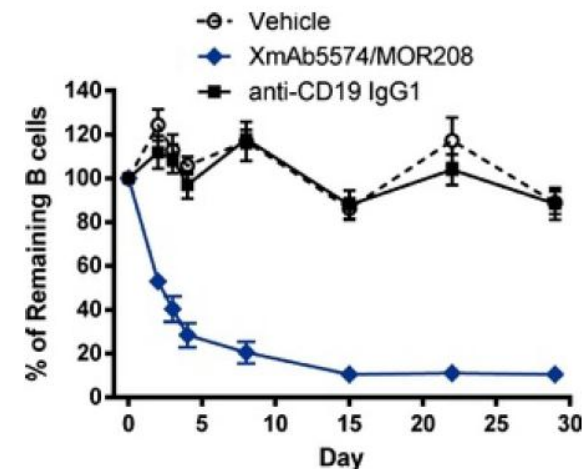


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# MOR208/XmAb5574 Phase I Showed Encouraging Signs of Preliminary Activity

- ❑ **XNCR completed Phase I/IIa trials in relapsed/refractory CLL in January, 2013:**
  - ❑ **Overall response rate by IWCLL (international workshop on CLL) 2008 criteria was 29.6%** (eight partial responses in 27 evaluable patients). Using IWCLL 1996, response criteria resulted in a response rate of 66.7% (18 partial responses)
  - ❑ **Dose levels from 0.3 to 12 mg/kg were tested.** The trial protocol was amended to include a period of extended dosing for a total of eight patients at the 12 mg/kg dose to study the effect of longer duration of exposure on safety and response rate.
  - ❑ **Clinically-significant, treatment-related adverse events classified as Grade 3 or higher occurred in 5 out of 27 patients.** One patient treated at the 1 mg/kg dose level experienced neutropenia. Four patients at the 12 mg/kg dose level experienced one or more of neutropenia, febrile neutropenia, thrombocytopenia, elevated aspartate aminotransferase or tumor lysis syndrome. Only one dose-limiting toxicity, neutropenia, was observed and this was in one of the 16 patients treated at the 12 mg/kg dose level. No Maximum Tolerated Dose (MTD) was reached in this trial.
- ❑ **Preclinical data:**
  - ❑ Figure on the right compares the ability of XmAb5574/MOR208 to an unmodified IgG1 control antibody (anti-CD19 IgG1) to deplete monkey B cells at a 3mg/kg dose. XmAb5574/MOR208 achieved a significant reduction in peripheral B cell counts, while the unmodified antibody can't be distinguished from the vehicle control.



Source: Company filings



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## Additional MOR208 Catalysts Likely in 2015

- ❑ **MorphoSys has initiated two single-agent Phase II clinical trials of MOR208 in B-cell acute lymphoblastic leukemia (B-ALL) and non-Hodgkin's lymphoma.** The B-ALL trials is an open-label, multicentre study to characterize the safety and preliminary efficacy of the human MOR00208 in adult subjects with relapsed/refractory B-cell acute lymphoblastic leukemia (B-ALL). MOR started enrolling 30 patients in April 2013. ORR (overall response rate) is the primary endpoint. The second study will assess the ORR in 120 patients Relapsed or Refractory Non-Hodgkin's Lymphoma (NHL) patients.
- ❑ **An additional investigator-sponsored trial was initiated in CLL which underscores the interest in MOR208.** The Phase II study in CLL will evaluate MOR208 in combination with lenalidomide (Revlimid). The investigator-sponsored trials is being conducted at the Ohio State University (OSU) led by investigator Dr. Jennifer Woyach. The trial is expected to enroll up to 20 treatment naïve and 20 relapsed/refractory CLL patients. The open-label, multi-dose, double-arm trial is entitled "A Phase II Study of MOR00208 in Combination with Lenalidomide for Patients with Relapsed or Refractory Chronic Lymphocytic Leukemia (CLL) / Small Lymphocytic Lymphoma (SLL), Prolymphocytic Leukemia (PLL) or Patients with Untreated CLL/SLL/PLL". MorphoSys will provide MOR208 drug supply for the study. MOR208 has been shown to act synergistically with the immunomodulator lenalidomide in non-clinical models according to MOR. In contrast to other potential combination partners, lenalidomide leaves the FcR-stimulated NK cell function, specifically ADCC, intact, preserving the anti-lymphoma efficacy of both agents.



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# CD19 Landscape Is Competitive, But It's Early Days

- **A number of investigational anti-CD19 agents are currently in development and MOR208 has a good shot at goal, in our view.** Most of the agents in development are based on new antibody enhancing technologies with the aim to better exploit CD19 as a target. Initial attempts to target CD19 with “naked” mAbs have been unsuccessful.
- **Three ADCC-enhanced CD19 antibodies from AZN (MP), MorphoSys (NR), and Medarex (acquired by BMY [OP]) have shown promising activity in Phase I/II.** AMGN's blinatumomab (CD19 x CD3 bi-specific antibody) and NVS/UPenn's CTL019 (CD19-targeted Chimeric Antigen Receptor-modified T-cells [CART]) have also shown strong activity in Phase I/II, which validates the mechanism of selectively killing CD19-expressing cells, in our view, but toxicity will likely restrict these agents to ALL, according to KOLs. SGEN's SGN-CD19A and IMGN/SNY's SAR3419 are CD19 antibody-drug conjugates (ADCs) starting in Phase I and Phase II respectively. Occular toxicities and a mediocre activity appear to be limiting for ADCs.

Product	Company	Status
MOR208	Morphosys	Phase II trials ongoing in NHL, CLL, B-ALL
AMG103	AMGN	Phase II trials ongoing in B-ALL, DLBCL
MEDI-551	AZN	Phase II trials ongoing in CLL, DLBCL
SAR3419	SNY/IMGN	Phase II trials ongoing in B-ALL, DLBCL
CTL019	NVS/UPenn	Phase II ongoing in CLL, NHL
SGEN-CD19A	SGEN	Phase I ongoing in NHL
MDX-1342	BMY	Unclear; Phase I in CLL completed

Source: Leerink Swann Research, Clinicaltrials.gov



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## Model Based on Rituxan as Proxy

- Given its potentially broad utility in treating B-cell mediated cancers, we believe MOR208 may potentially address a large proportion for the future CD19 targeting market, if developed and marketed successfully.
- For valuation purposes, we took the approach to use world-wide Rituxan sales as a proxy for a potential future CD19 market and assumed MOR208 could capture a 40% share of this market.

Year	1997	1998	1999	2000	2001	2002	2003	2004	2005	2006	2007	2008
Rituxan WW sales (\$MM)	6	163	279	444	819	1,163	1,489	1,711	1,989	2,252	2,515	2,852

Year	2019E	2020E	2021E	2022E	2023E	2024E	2025E	2026E	2027E	2028E	2029E	2030E
WW anti-CD19 Sales (\$MM)	6	163	279	444	819	1,163	1,489	1,711	1,989	2,252	2,515	252
MOR 208 Mkt share	40%	40%	40%	40%	40%	40%	40%	40%	40%	40%	40%	40%
WW MOR Sales (\$MM)	2	65	112	178	327	465	596	684	796	901	1,006	101
Royalty rate	10%	10%	10%	10%	10%	10%	10%	10%	10%	10%	10%	10%
MOR208 royalty to XNCR	0	7	11	18	33	47	60	68	80	90	101	10

<b>Assume launch in 2019</b>	
2025 WW Sales (MM)	596
2025 WW Royalty (\$MM)	60
Multiple	10
Discount rate	12%
Present Value	171
Probability of success	30%
P/W NPV	51

Source: Leerink Swann Estimates



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# IP Summary

- ❑ XmAb technology platform is protected by 21 US issued patents and 44 US patent applications, in addition to foreign counterparts.
- ❑ The three lead product candidates are covered by issued US composition-of-matter patents relating to both the XmAb Fc domains and the individual product candidates.
  - ❑ XmAb5871
    - ❑ Fv composition patent expiry 2027
    - ❑ Fc composition patent expiry 2027
    - ❑ Methods-of-use expiry 2028
  - ❑ XmAb7195
    - ❑ Fv composition patent expiry 2031
    - ❑ Fc composition patent expiry 2027
  - ❑ MOR208
    - ❑ Fv composition patent expiry 2027
    - ❑ Fc composition patent expiry 2025
- ❑ In addition new biologics have 12 years data exclusivity in the US and 10 years in Europe.





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## XNCR Management Team with Relevant Experience

- ❑ **CEO: Bassil I. Dahiyat, Ph.D.** – co-founded Xencor in 1997
- ❑ **CBO: Edgardo Baracchini, Jr., Ph.D.** - Joined Xencor in January 2010
  - ❑ Previously SVP, Business Development at Metabasis Therapeutics (until its merger with Ligand Pharmaceuticals in 2009).
  - ❑ Prior to joining Metabasis, he was VP of business development at Elitra Pharmaceuticals, and director of business development at Agouron Pharmaceuticals, until its acquisition by Warner-Lambert in 1999.
- ❑ **CMO: Paul Foster, M.D.** - Joined Xencor in August 2012
  - ❑ Prior to joining Xencor, he provided consulting services as SVP Development and CMO of Development and Strategic Consulting Associates, LLC
  - ❑ CMO of Cardium Therapeutics from June 2008 to May 2009.
- ❑ **VP Research: John R. Desjarlais, Ph.D.** - Joined Xencor in July 2001
  - ❑ Prior to joining Xencor, he was an Assistant Professor of Chemistry at Penn State University
- ❑ **VP Finance: John J. Kuch** – Joined Xencor in October 2000
  - ❑ Worked over 15 years in public accounting, most recently as a Director at Price Waterhouse
- ❑ **Chairman of the Board and Director: Bruce L.A. Carter** – Joined Xencor in September 2009
  - ❑ Former CEO of Zymogenetics from April 1998 to January 2009



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# Summary of Collaboration and Licensing Revenue by Partner

## Revenues in \$MM

Partner	2011	2012	1Q-3Q13
Amgen	2.0	1.8	1.7
MorphoSys	2.2	2.0	3.0
CSL	1.3	1.8	2.0
Janssen	1.0	1.4	-
BI	-	1.2	-
Merck	-	-	1.0
Other	0.3	1.3	0.7
<b>Total</b>	<b>6.8</b>	<b>9.5</b>	<b>8.4</b>

Source: SEC Filings

XNCR P&L (in \$ MM)	2011	2012	1-3Q13Q	4Q13E	2013E	2014E	2015E	2016E	2017E	2018E	2019E	2020E
Collaboration and licensing revenue	6.8	9.5	8.4	1.0	9.4	5.0	10.0	10.0	20.0	25.0	25.0	25.0
Product sales	-	-	-	-	-	-	-	-	-	-	-	-
Royalty revenue	-	-	-	-	-	-	-	-	-	-	0.2	6.5
<b>Total revenue</b>	<b>6.8</b>	<b>9.5</b>	<b>8.4</b>	<b>1.0</b>	<b>9.4</b>	<b>5.0</b>	<b>10.0</b>	<b>10.0</b>	<b>20.0</b>	<b>25.0</b>	<b>25.2</b>	<b>31.5</b>
COGS	-	-	-	-	-	-	-	-	-	-	-	-
R&D	12.7	12.7	12.9	5.1	18.0	24.0	30.0	36.0	39.6	43.6	47.9	52.7
SG&A	3.6	3.1	2.4	2.0	4.4	5.5	6.5	7.2	7.9	8.7	9.5	10.5
Operating expenses	16.3	15.8	15.2	7.1	22.4	29.5	36.5	43.2	47.5	52.2	57.4	63.2
Operating income	(9.5)	(6.2)	(6.8)	(6.1)	(13.0)	(24.5)	(26.5)	(33.2)	(27.5)	(27.2)	(32.2)	(31.7)
Total other income (expense)	(1.8)	(2.4)	(49.7)	-	(49.7)	-	-	-	-	-	-	-
EBT	(11.2)	(8.6)	(56.6)	(6.1)	(62.7)	(24.5)	(26.5)	(33.2)	(27.5)	(27.2)	(32.2)	(31.7)
Income tax expense	-	-	-	-	-	-	-	-	-	-	-	-
Net income (loss)	(11.2)	(8.6)	(56.6)	(6.1)	(62.7)	(24.5)	(26.5)	(33.2)	(27.5)	(27.2)	(32.2)	(31.7)
Diluted EPS	(154.95)	(118.86)	(4.10)	(0.20)	(5.54)	(0.78)	(0.85)	(1.06)	(0.66)	(0.66)	(0.78)	(0.68)
Diluted shares outstanding	0.1	0.1	13.8	31.3	11.3	31.3	31.3	31.3	41.3	41.3	41.3	46.3

Source: Leerink Swann Estimates and Company Filings

BS & CFS	2011	2012	1-3Q13Q	4Q13E	2013E	2014E	2015E	2016E	2017E	2018E	2019E	2020E
Cash and STI	14.5	2.3	9.6	75.9	75.9	54.3	31.5	2.6	79.9	57.9	31.5	106.1
Debt	18.5	20.9	-	-	-	-	-	-	-	-	-	-

Change in Cash	(2.4)	(12.2)	7.3	66.3	73.6	(21.6)	(22.9)	(28.8)	77.3	(22.0)	(26.5)	74.6
<b>Cash from operations</b>	<b>(1.1)</b>	<b>(11.1)</b>	<b>(1.3)</b>	<b>(6.1)</b>	<b>(7.4)</b>	<b>(21.6)</b>	<b>(22.9)</b>	<b>(28.8)</b>	<b>(22.7)</b>	<b>(22.0)</b>	<b>(26.5)</b>	<b>(25.4)</b>
Net income (loss)	(11.2)	(8.6)	(56.6)	(6.1)	(62.7)	(24.5)	(26.5)	(33.2)	(27.5)	(27.2)	(32.2)	(31.7)
Share based comp	(0.1)	0.0	0.1	-	0.1	3.0	3.7	4.3	4.7	5.2	5.7	6.3
D&A	0.6	0.5	0.4	-	0.4	-	-	-	-	-	-	-
Other (Change in WC)	9.6	(3.0)	54.8	-	54.8	-	-	-	-	-	-	-
<b>Cash from investing</b>	<b>(1.3)</b>	<b>(1.2)</b>	<b>(1.3)</b>	<b>-</b>	<b>(1.3)</b>	<b>-</b>	<b>-</b>	<b>-</b>	<b>-</b>	<b>-</b>	<b>-</b>	<b>-</b>
CapEx	(0.1)	(0.0)	(0.1)	-	(0.1)	-	-	-	-	-	-	-
Acquisitions	(1.4)	(1.2)	(1.1)	-	(1.1)	-	-	-	-	-	-	-
Other	0.1	0.1	0.0	-	0.0	-	-	-	-	-	-	-
<b>Cash from financing</b>	<b>(0.0)</b>	<b>(0.0)</b>	<b>9.9</b>	<b>72.4</b>	<b>82.3</b>	<b>-</b>	<b>-</b>	<b>-</b>	<b>100.0</b>	<b>-</b>	<b>-</b>	<b>100.0</b>
Equity issue (buyback)	-	-	10.0	72.4	82.4	-	-	-	100.0	-	-	100.0
Debt issue (principal payment)	-	-	-	-	-	-	-	-	-	-	-	-
Other	(0.0)	(0.0)	(0.1)	-	(0.1)	-	-	-	-	-	-	-

Source: Leerink Swann Estimates and Company Filings



## Disclosures Appendix

### Analyst Certification

I, Michael Schmidt, Ph.D., certify that the views expressed in this report accurately reflect my views and that no part of my compensation was, is, or will be directly related to the specific recommendation or views contained in this report.



Distribution of Ratings/Investment Banking Services (IB) as of 09/30/13				
Rating	Count	Percent	IB Serv./Past 12 Mos.	
			Count	Percent
BUY [OP]	111	64.90	27	24.00
HOLD [MP]	60	35.10	0	0.00
SELL [UP]	0	0.00	0	0.00

## Explanation of Ratings

**Outperform (Buy):** We expect this stock to outperform its benchmark over the next 12 months.

**Market Perform (Hold/Neutral):** We expect this stock to perform in line with its benchmark over the next 12 months.

**Underperform (Sell):** We expect this stock to underperform its benchmark over the next 12 months. The degree of outperformance or underperformance required to warrant an Outperform or an Underperform rating should be commensurate with the risk profile of the company.

For the purposes of these definitions the relevant benchmark will be the S&P 600® Health Care Index for issuers with a market capitalization of less than \$2 billion and the S&P 500® Health Care Index for issuers with a market capitalization over \$2 billion.





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