Xencor (XNCR)

Initiating with an OUTPERFORM Rating and $18 Price Target: Rationally Fc-Engineering a Novel Portfolio of Antibodies Targeting Unmet Medical Needs

- **Xencor is a clinical-stage protein engineering company focused on developing novel Fc-optimized antibody therapies.** The company has discovered Fc-domain modifications that augment antibody function in several ways including novel inhibitory activity, enhanced clearance, increased cytotoxicity, extended half-life and Fc-variants that facilitate the creation of novel bispecifics.
- **We expect Xencor's first bispecific antibodies to substantially drive the value of the company as they enter the clinic in 2015.** Xencor's Fc-engineered antibody technologies enable the creation of potentially best-in-class, novel, long-half-life bispecifics. Incorporation of Xencor's Fc-domains allows for bispecifics with antibody-like half-lives (6-7 days) and high production yields that are potentially superior to previous generations of bispecific technology.
- **XmAb7195, Xencor's lead compound, is a Xolair (omalizumab) bio-superior for the treatment of allergic asthma and other IgE mediated disease.** XNCR's Fc-modifications impart two novel mechanisms of action to XmAb7195 resulting in superior clearance and reduction of IgE levels compared to Xolair and high-affinity anti-IgE candidates in the clinic. XmAb7195 is likely to be a best-in-class drug for IgE related diseases, including those beyond severe asthma, and may replace and expand the $1.3B annual Xolair market in poorly controlled or ineligible patients. First-in-human results, including IgE reduction data, are anticipated before YE:14.
- **XmAb5871, a CD19 targeted immune inhibitory antibody, uniquely employs Xencor's technology to co-engage FcγRIIb inhibiting BCR signaling.** XmAb5871 inhibits B-cell activation, but does not deplete B-cells like rituximab. Data from the ongoing Phase Ib/IIa trial in patients with RA is expected in YE:14.
- **We believe that the novelty, breadth and value of XNCR's Fc-engineering toolkit are broadly underappreciated by the market.** The company also has 6 internal pre-clinical candidates that exploit their Fc-modification toolkit. Xencor's platform has been validated through 7 ongoing collaborations that may result in a total of $1.31 billion in non-dilutive milestones and additional royalties.
- **Initiating coverage with an OUTPERFORM rating and an $18 price target.** Our $18 price target is derived from the sum of multiples on sales and royalties from XNCR's proprietary and partnered products each discounted back to YE:14 (Pg 2).

FYE Dec	2013E	2014E			2015E		
REV	ACTUAL	CURR.	PREV.	CONS.	CURR.	PREV.	CONS.
Q1 Mar	-A	$2.5E		--	2.5E		--
Q2 Jun	-A	2.5E		--	2.5E		--
Q3 Sep	8.4A	2.5E		--	2.5E		--
Q4 Dec	2.8E	$2.5E		--	2.5E		--
Year*	**$11.2E**	**$10.0E**		**--**	**$10.0E**		**--**
Change	--	--			--		
	2013E	2014E			2015E		
EPS	ACTUAL	CURR.	PREV.	CONS.	CURR.	PREV.	CONS.
Q1 Mar	-A	($0.09)E		--	($0.16)E		--
Q2 Jun	-A	($0.11)E		--	($0.16)E		--
Q3 Sep	(782.22)A	($0.12)E		--	($0.15)E		--
Q4 Dec	(0.13)E	($0.14)E		--	($0.15)E		--
Year*	**($2.75)E**	**($0.14)E**		**--**	**($0.62)E**		**--**
P/E	--	--			--		
Change	--	--			--		

Consensus estimates are from Thomson First Call.
** Numbers may not add up due to rounding.*

December 31, 2013

Price
$8.51

Rating
OUTPERFORM

12-Month Price Target
$18

Christopher N. Marai, Ph.D.
(415) 274-6861
christopher.marai@wedbush.com

Gregory R. Wade, Ph.D.
(415) 274-6863
greg.wade@wedbush.com

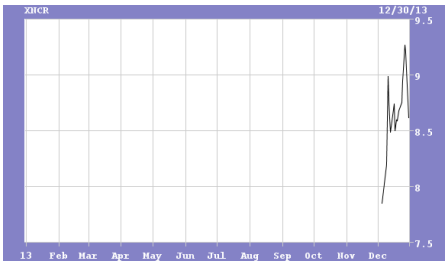
David M. Nierengarten, Ph.D.
(415) 274-6862
david.nierengarten@wedbush.com

Company Information

Shares Outst (M)	31.4
Market Cap (M)	$271.9
52-Wk Range	$5.75 - $10.90
Book Value/sh	$7.87
Cash/sh	$2.53
Enterprise Value (M)	$192.6
LT Debt/Cap %	0.0
Cash Burn (2014) (M)	$14.4
Current Cash (M)	$79.2

Company Description

Xencor is a clinical-stage biopharmaceutical company focused on discovering and developing engineered monoclonal antibodies to treat severe and life-threatening diseases with unmet medical needs.



Source: Thomson Reuters

Investment Thesis

Xencor is a biopharmaceutical protein engineering company focused on developing and commercializing engineered monoclonal antibodies to treat severe and life-threatening diseases with unmet medical needs. The company has developed a substantial antibody toolkit based upon their proprietary Fc-engineering platform. Xencor has systematically and rationally engineered novel Fc-domains that augment the ability of antibodies in several ways including: immune inhibitor Fc domains that target inhibitory and clearance functionality (FcγRIIb target); cytotoxic Fc domains, that increase cytotoxicity/ADCC (targeting FcγRIIIa and FcγRIIa receptors), Xtend Fc domains that extend half-life (targeting FcRn) and allow for the creation of novel bispecifics. We believe this best-in-class platform, which has produced two partnered products in Phase II studies, 6 internal early stage programs and a total of 7 collaborations is the core of Xencor's value.

Importantly Xencor has also created Fc variants that enable heterodimeric Fv domains for the creation of novel bispecific antibodies. We believe that Xencor's bispecific candidates could represent a significant value driver for the company as they enter the clinic potentially in 2015.

Xencor's lead candidate is XmAb7195, an anti-IgE antibody therapy for the treatment of allergic asthma employing Fc-modification to enhance FcγRIIb binding up to 400x. These modifications lead to enhanced liver clearance of IgE and suppressed production through co-engagement with B-cell bound IgE. These new functionalities, in our opinion, make XmAb7195 a best-in-class anti-IgE therapy and highlight potential broad utility of Xencor's technology. The Fc-domain in XmAb7195 is also utilized in their Phase II compound XmAb5871, supporting the safety and tolerability of these Fc-modifications. XmAb7195 is in IND-enabling studies and a Phase I trial is planned for early 2014. Importantly Phase I data, expected around YE:14, will include IgE levels, a marker of activity and potential clinical benefit.

We believe that partnerships with Amgen, Alexion, MorphoSys, Janssen, Merck, Boehringer Ingelheim and CSL have validated Xencor's approach with $65M in revenues to date, potentially $1.31 billion in additional milestones and single to double digit royalties. XmAb5871, optioned to Amgen, is Xencor's Phase II candidate for autoimmune disorders that represents a novel targeted strategy for selectively inhibiting B-cells without depletion. XmAb5871 targets both CD19 and FcγRIIb to uniquely inhibit B-cell activity without depletion, a potentially broadly applicable and valuable approach in autoimmune disorders and oncology. Top-line data from a Phase 2a study is anticipated late 2014. XmAb5574/MOR208, partnered with MorphoSys is Xencor's other CD19 therapy that employs Fc-engineered higher affinity for FcγRIIIa and FcγRIIa receptors to potently deplete CD19 expressing B-cells offering utility in B-cell malignancies where CD20 therapies are not effective.

Valuation Methodology

We arrive at our $18 price target by a sum-of-the-parts analysis. We ascribe $12/share to applying a 6x multiple of our estimated peak revenues of $1.2 billion (2022) for XmAb7195 in severe asthma and IgE mediated diseases, discounted 45% annually. We ascribe $6/share to Xencor's two partnered programs XmAb5871 and XmAb5574 based on royalties (mean 10%) from sales in 2022 of $1 billion and $600 million discounted 45% and 35% annually, respectively. Similarly we also arrive at our $6/share value to Xencor's potential milestones based upon the NPV discounted 45% over an estimated 10 year realization period. We do not value Xencor's early stage pipeline novel bispecifics at this time.

Sum-of-the-Parts Valuation Components

Product	Indication	Est. Addressable Market	Penetration	Annual Cost	Sales	Multiple	Royalty rate	Year	Discount rate	Value per share
XmAb7195	Severe asthma	350,000*	15%	$22K	$1.2B	6x	NA	2022	45%	$12
XmAb5574	ALL	16,000	38%	$100K	$600M	15x	~10%	2022	35%	$3
XmAb5871	RA	460,000	10%	$25K	$1.2B	15x	~10%	2022	45%	$3
Total										$18

**US market only*

Risks

Risks to the attainment of our price target include 1) failure to reach sales expectations for XmAb7195, XmAb5574, XmAb5871 2) failure in the clinic of either XmAb7195, XmAb5574, XmAb5871, 3) changes or discontinuation of Xencor's partnerships for XmAb5574, XmAb5871 or other partnered programs.

Key Points

- Xencor has developed a differentiated, systematic, Fc-engineering platform that has yielded antibodies capable of both positively and negatively regulating the immune system, extending half-lives, or creating novel bispecifics with just 2 substitutions
- Xencor's Fc-engineered antibodies feature as much as 40x enhanced binding to FcγRIIIa and 10x binding to FcγRIIa for enhanced cytotoxicity, 400x binding to FcγRIIb for immune inhibition and rapid target clearance and 20x by binding to FcRn for longer half-life
- The company has engineered Fc variants that enable heterodimeric Fv domains for the creation of novel bispecific antibodies with antibody-like half-life (6-7 days), significant flexibility in targeting domains and improved production yields
- We expect Xencor's first bispecific antibodies to substantially drive the value of the company as they enter the clinic in 2015
- Xencor's unique inhibitory enhanced binding FcγRIIb domain and co-engagement with Fv-targets facilitates differentiated activity of their two lead candidates from similarly targeted antibodies in the clinic
- XmAb7195, an IgE targeted mAb, offers differentiated mechanisms of action allowing greater than 10-fold IgE reductions compared to Xolair and allows Xencor to measure IgE level reduction as a biomarker of response in early trials
- We expect XmAb7195 to be safe and well tolerated based on preliminary but informative pre-clinical studies, similarity to Xolair and natural history of genetically IgE deficient patients. First-in-humans data for XmAb7195 including IgE levels is expected in H2:14
- XmAb5871 for autoimmune disorders inhibits B-cell activation via a novel mechanism of co-engagement of FcγRIIb with the BCR
- Importantly, XmAb5871 only represents one example of Xencor's differentiated FcγRIIb Fc-technology, as several other co-targets beyond CD19 could be contemplated. Data from the Phase Ib/IIa trial of XmAb5871 is expected in the second half of 2014.
- Xencor's Fc modification technologies have been validated by several focused partnerships that maintain the companies ownership of their platform worth in excess of $1.3 billion should all milestones be exercised/achieved

Milestones

H1:14	Initiation of a Phase Ia trial of XmAb7195 (IgE, immune inhibitor Fc) in healthy subjects
YE:14	Top-line data from the Phase IIa trial of XmAb5871 (CD19, immune inhibitor Fc) in rheumatoid arthritis
YE:14	Top-line data from the Phase Ia trial of XmAb7195 in healthy subjects including IgE reduction
2015	Initiation of a Phase IIb proof-of-concept trial of XmAb5871 (CD19, immune inhibitor Fc) in rheumatoid arthritis
2015	IND filing and first-in-human clinical trials for Xencor's first clinical bispecific candidates
Q1:16	Potential top-line data from the Phase Ib trial of XmAb7195 in mild to moderate asthma
2016	Potential top-line data from the Phase II trial of XmAb5574/MOR208 (CD19, enhanced ADCC Fc) in ALL
2016	Initiation of a Phase IIb proof-of-concept trial of XmAb7195 in poorly controlled asthma
2017	Potential top-line data from the Phase IIb POC trial of XmAb7195 in patients with poorly controlled asthma
2017	Potential top-line data from the Phase IIb trial of XmAb5871 in patients with rheumatoid arthritis
2017	Potential exercise of Amgen's option to license XmAb5871

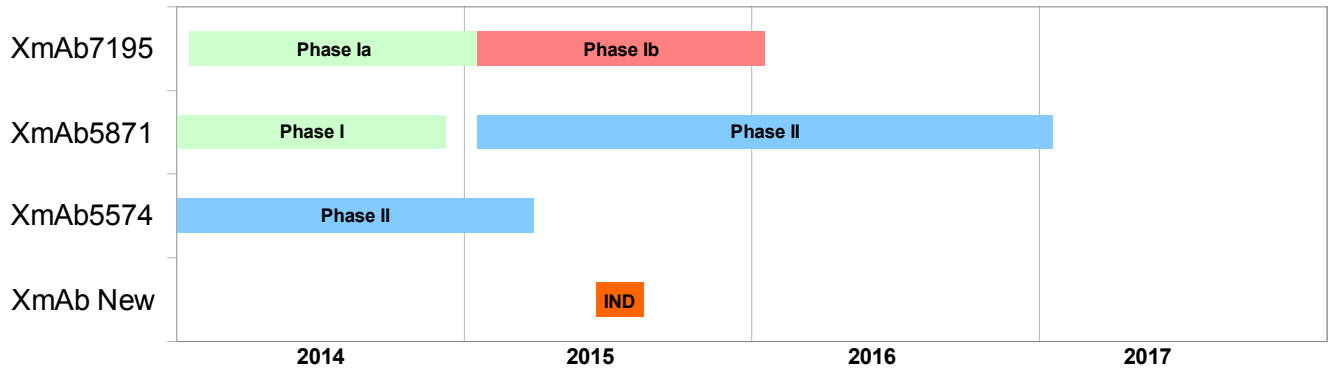
Xencor' Pipeline

Figure 1: Xencor's Development Pipeline

Name	Fv Target	Fc Target	Fc Modification	Indication	Status	Next Event	Partner
XmAb7195	IgE	FcγRIIb	Immune Inhibitor	Allergic asthma	Phase I	PIa initiation in H1:14	-
XmAb5871	CD19	FcγRIIb	Immune Inhibitor	Auto-immune	Phase II	PIIa data in RA in H1:14	Amgen
XmAb5574/MOR208	CD19	FcγRIIIa, FcγRIIa	ADCC	ALL/NHL	Phase II	PII data in NHL/B-ALL 2016*	MorphoSys
Xtend-TNF	TNF	FcRn	X-tend	Auto-immune	Pre-clinical	IND filing in 2015*	-
CD3 x CD38	CD38	-	Bi-specific	Oncology	Pre-clinical	IND filing in 2015*	-
CD3 x CD123	CD123	-	Bi-specific	Oncology	Pre-clinical	IND filing in 2015*	-
Xtend-CTLA4	CTLA4	FcRn	Xtend	Auto-immune	Pre-clinical	IND filing in 2015*	-
Anti-X/CD32b	-	FcγRIIb	Immune Inhibitor	TBD	Discovery	IND filing*	-
Undisclosed	-	FcγRIIIa, FcγRIIa	ADCC	Oncology	Phase I	-	BI
Undisclosed	-	FcγRIIIa, FcγRIIa	ADCC	Oncology	Phase I	-	BI
Undisclosed	-	FcγRIIIa, FcγRIIa	ADCC	Oncology	Phase I	-	CSL
Undisclosed	-	FcRn	Xtend	Hematology	Pre-clinical	-	CSL
Undisclosed	-	FcRn	Xtend	Autoimmune	Pre-clinical	-	Janssen
Undisclosed	-	-	Stability	Autoimmune	Pre-clinical	-	Merck
Undisclosed	-	FcRn	Xtend	Undisclosed	Pre-clinical	-	Undisclosed

*Source: Company data, Wedbush Securities, Inc. *Wedbush estimates*

Figure 2: Xencor's Near-term Product Development Pipeline



Source: Company data, Wedbush Securities, Inc.

Xencor Company Overview

Xencor is a clinical-stage biopharmaceutical company focused on discovering and developing engineered monoclonal antibodies to treat severe and life-threatening diseases with unmet medical needs. Xencor leverages their proprietary engineered Fc domain platform, "XmAb technology", to create next-generation antibody product candidates designed to treat autoimmune and allergic diseases, cancer, and other conditions. Their technology has been applied to a growing pipeline of antibody-based drug candidates to increase immune inhibition, improve cytotoxicity, extend half-life or offer bispecific functionality. Xencor is headquartered in Monrovia, California.

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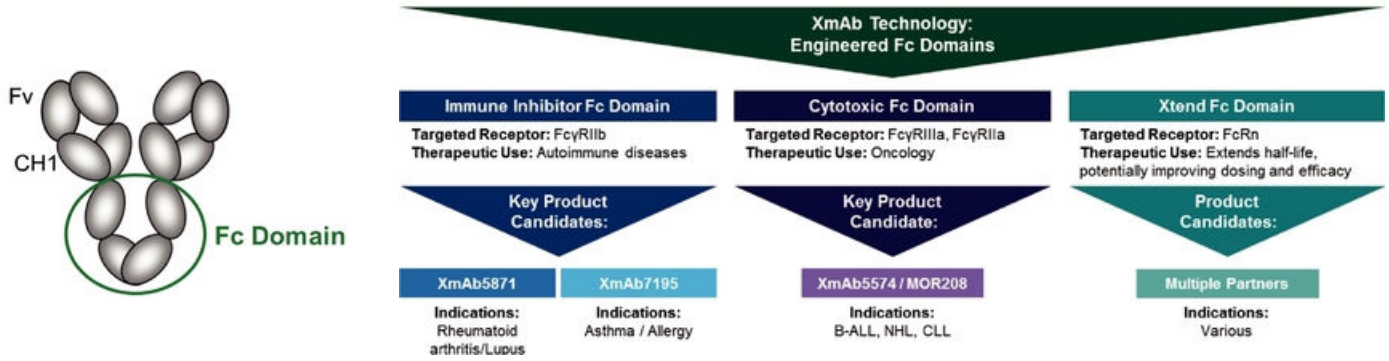
Xencor's Fc-Engineered Antibody Technology

Xencor develops novel advanced protein therapeutics through a rational, systematic approach based on engineering the Fc domain of antibodies. The company leverages their patented three-dimensional structure screening technology to rationally explore small modifications in amino acids (typically 2-amino acids, maintaining 99.5% structure identity) that alter the Fc domain to significantly augment antibody performance. The Fc region of an antibody is located at the base of the Y-shaped antibody protein and is responsible for modulating the immune response through binding to specific receptors on immune cells (Figures 3, 4). Modification of Fc-mediated functions such as antibody-dependent cellular cytotoxicity (ADCC), complement-dependent cytotoxicity (CDC), and neonatal Fc receptor (FcRn) mediated storage/recycling, may, based upon our current clinical experience with several therapeutic antibodies, improve clinical outcomes of these biologic therapies. Xencor has discovered a set of Fc domains that augment naturally occurring antibody functions based on Fc-binding, these include immune inhibitor Fc domains that enhance inhibitory and rapid clearance functionality (FcγRIIb target); cytotoxic Fc domains, that increase cytotoxicity/ADCC (targeting FcγRIIIa and FcγRIIa receptors) and Xtend Fc domains that extend half-life by targeting receptor FcRn on endothelial cells. Xencor has also created Fc variants that form heterodimeric Fc domains that enable the creation of novel bispecific antibodies with different Fv domains.

Figure 3: Xencor's Engineered Fc Domain Technology



Source: Company data, Wedbush Securities, Inc.

Xencor's development pipeline includes several antibodies directed against well-validated targets (Fv domain) that also incorporate their engineered Fc domains to create superior alternatives to currently marketed antibodies as well as new antibodies with novel functionality. Importantly, Xencor is also developing antibodies with two different Fv domains that can be bispecific for two different antigens. For example, CD3 x CD38 antibodies bind T-cells (CD3) and tumor cells (CD38) recruiting cytotoxic T-cells to the tumor or other cell targeted for destruction. In addition to disclosed partnerships, Xencor has 7 undisclosed partnered products, 3 in clinical development, with Alexion, Boehringer Ingelheim, Janssen, Merck and CSL.

Xencor's Fc Domains – Building Blocks of Engineered Immunotherapy

Antibody engineering efforts have for the past decade focused primarily on Fv domain modifications to modulate binding affinity to increase or decrease the potency as desired. The Fv region of an antibody is located at the top of the Y-shaped antibody protein and is responsible for binding to a structurally defined target called an epitope (Figure 5). However, discoveries have led to the appreciation that engineering the constant region (Fc) which interacts with the immune system can modulate the immune response and generate novel classes of antibody therapeutics.

Xencor has developed a differentiated, rational Fc-engineering platform that is capable of both positively and negatively regulating the immune system as well as having other effects such as increasing half-life or creating novel long-half life bispecifics. Prior to Xencor's approach, Fc-modification efforts have primarily focused on enhancing the antibody dependent cellular cytotoxicity (ADCC) abilities of antibodies. Notable ADCC enhanced antibodies based on Fc modifications include Roche's obinutuzumab (Gazyva/GA-101), targeting CD20 and modified for higher binding to FcγRIII through enriched bisected non-fucosylated glycosylation variants, Macrogenics (MGNX: Outperform) margituximab, a Her2 targeted mAb, and modified for higher binding to Fcγ receptors, and Xencor/MorphoSys' MOR208/XmAb5574 targeting CD19, with modified binding to FcγRIIa and FcγRIIIa.

Xencor modifies antibody-immune interactions through Fc-variants that alter their interaction with the seven canonical IgG binding Fc receptors, classified into four categories, FcγRI, -II, -III and FcRn. They consist of four activating receptors (FcγR1, FcγRIIa, FcγRIIc, and FcγRIIIa), one inhibitory receptor (FcγRIIb), a receptor with unclear function (FcγRIIIb), and one receptor involved in recycling and transport (FcRn). Consequently, the company has Fc-variants that enhance ADCC, improve complement-dependent cytotoxicity, inhibit immune function, eliminate cytotoxic binding, increase half-life and facilitate heterodimer Fv region attachment for the creation of bispecifics. These novel immune modulatory functions open up new arenas for antibody development, exemplified in part by Xencor's lead candidates XmAb7195, XmAb5871, MOR208/XmAb5574.

Figure 4: Fc Receptors Targeted by Xencor

XmAb Fc Domain	Receptor	Affinity increase (decrease) over IgG	Function	Cell Types	Disease Area
Immune inhibitor	FcγRIIb	400x	Cell inhibition	B cell, other immune cells	Autoimmune
			Rapid target clearance	Liver sinusoidal endothelial cells	Various
Cytotoxicity / ADCC	FcγRIIa	10x	Phagocytosis	Macrophages	Oncology
	FcγRIIIa	40x	Cytotoxicity	NK cells	
Xtend	FcRn	20x	Antibody recycling	Endothelial cells	Various
Fc Knockout	FcγRIIa	Estimated* (10x)	Phagocytosis	Macrophages	Various/Oncology
	FcγRIIIa	Estimated* (3x) to (10x)	Cytotoxicity		
Bispecific	N/A	N/A	Hetero-dimerization	T-cells (CD3)	Oncology

Source: Company data, Wedbush Securities, Inc.

FcγRIIIa and FcγRIIa – Improving Response to Antibody Therapy by ADCC

Xencor has developed a next-generation cytotoxic Fc domain that increases FcγRIIIa binding by 40-fold over traditional antibody Fc domains. FcγRIIIa is an excitatory receptor on natural killer (NK) cells and macrophages primarily responsible for inducing ADCC. Additionally, their modification of FcγRIIIa has resulted in greater enhancement of binding of "F" allelic variants correlated with poor response to antibody therapy compared to "V" allelic variants of the receptor. Xencor's products may be differentiated from currently approved therapies by inducing responses in patients insensitive to non Fc-engineered antibodies. The cytotoxic Fc domain also improves FcγRIIa receptor binding roughly 10-fold, which recruits and activates myeloid cells to induce phagocytosis. Importantly, increasing cytotoxicity via two independent mechanisms reduces the potential for immune escape, strategies by which tumor cells down-regulate specific parts of the immune system to acquire resistance to immune mediated killing.

FcγRIIb – A Versatile Immune Inhibitory Antibody Receptor

FcγRIIb binds IgG with low-affinity on the surface of B-cells and functions in a negative feedback loop in the natural immune response to inhibit antibody production once concentrations have reached therapeutic levels. Xencor has exploited this function by specifically increasing the binding affinity of their Fc domain for the FcγRIIb protein 400-fold, generating antibodies that inhibit immune activity, inducing an effect opposite of a normal activating antibody. Increasing FcγRIIb binding affinity or cross linking FcγRIIb is not enough, a drug targeting this receptor must recruit it to the BCR complex in order to have an inhibitory effect. FcγRIIb also clears antibody-antigen complexes from the bloodstream via uptake and recycling in liver sinusoidal endothelial cells (SLEC's) further increasing clearance rates.

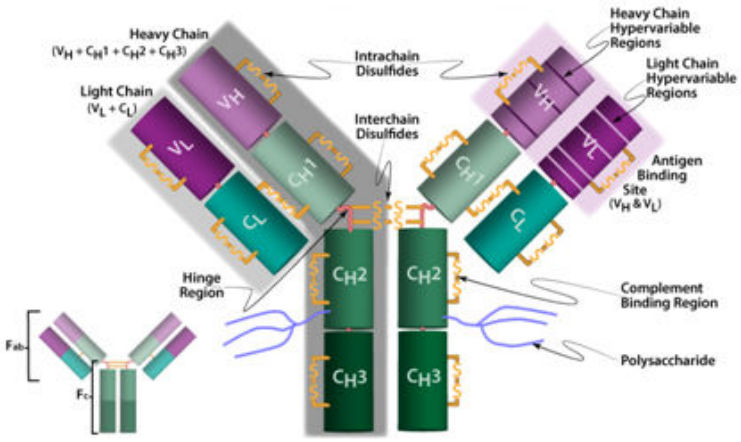
Xencor demonstrated the effect of the FcγRIIb Fc domain modification by producing XmAb7851 targeted to CD19 on B-cells as well as with their XmAb7195 candidate targeted to IgE on B-cells. XmAb7851 recruits FcγRIIb to the BCR via interaction with the BCR complex co-receptor CD19 to shut down BCR signaling. This antibody is an effective, non-specific B-cell inhibitor currently in Phase II trials that is anticipated to find use in lupus, rheumatoid arthritis and broadly any auto-immune disorders where B-cell depleters such as rituximab might be effective.

The potential of this platform is far greater than XmAb5871's non-specific immune inhibition strategy. It is being investigated in asthma with XmAb7195 as an IgE-specific B-cell inhibitor where it could be used broadly to inhibit B-cell response only to a specific antigen. For example, in food allergies an antibody targeted to the allergen with this Fc domain could down-regulate immune response before or after a suspected exposure. Additionally, an antibody with this Fc domain could be used to rapidly clear immune complexes or other proteins of interest from the bloodstream via liver uptake.

FcRn – Xtend Technology for Longer Half-Life

Xencor has been able to achieve dramatic half-life extensions through Fc-engineering. The company has demonstrated half-life extensions approaching three-fold in some cases, by specifically increasing FcRn affinity in acidic environments, but not in basic environments, allowing efficient antibody release into the blood. The neonatal Fc receptor (FcRn) is expressed at very high levels in early development, but despite the name, continues to be expressed on endothelial cells, neutrophils, dendritic cells and macrophages in adulthood. FcRn functions in a salvage role by binding IgG-antibodies in the acidic environment of the early lysosome and is recycled to the surface where it releases antibodies back into the basic environment of the blood.

Figure 5: Antibody Structure and Segments



Source: Wedbush Securities, Inc.

Fc-Enabled Bispecifics

Xencor has developed a novel technique for generating high yields of bispecific antibodies that facilitate significant flexibility in targeting domains. Xencor's bispecific technology allows for the production of humanized bispecific antibodies incorporating scFv fragments and Fc modifications, without linkers or other novel antibody formats. Structurally, the company's bispecific technology can produce novel antibodies where one or both heavy/light chain pairs is missing the C_H1 and C_L domains (Figures 5, 6), generating an Fv fragment directly linked to the Fc domain.

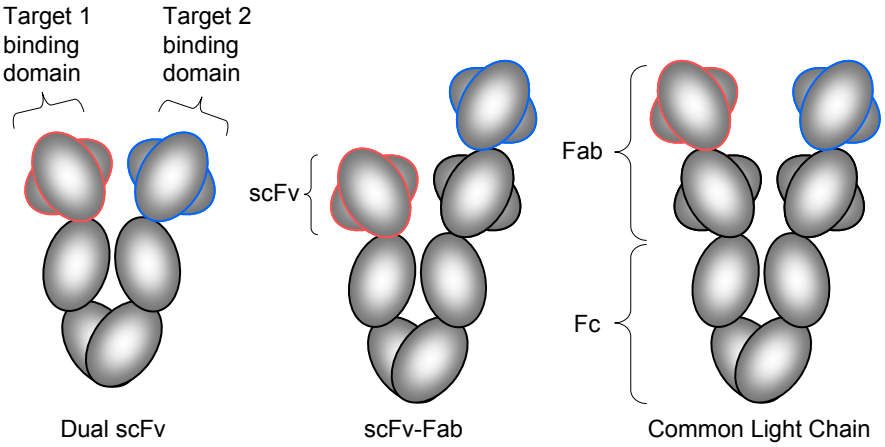
Bispecific antibodies and fragments function by activating the CD3 receptor on T-cells inducing T-cell activation as though it had been presented with its specific antigen. These molecules re-target the T-cell from the antigen its CD3 receptor is specific for to the antigen bound by the other arm of the bispecific antibody. (Figure 6) In pre-clinical studies in monkeys, Xencor's molecules resulted in bispecific T-cell engagement via two distinct Fv domains while retaining Fc domain mediated half-life extension and potentially allowing for potential further cytotoxic, Xtend or other Fc domain optimization. Xencor's technology also allows for Fc-knock-out variants that keep the FcRn-mediated half-life of the drug without inducing FcγR-mediated cross-linking and potential off-target T cell activation.

Xencor's technology, by incorporating Fc domains allows for a natural antibody-like half-life (6-7 days) superior to previous generations of bispecific technology. Although promising, early generations of bispecific technology such as the Micromet (Amgen) BiTE have been limited by the requirement for continuous infusion due to their extremely short half-life (3 hours). In addition to half-life extension Xencor's technology may potentially reduce immunogenicity, and cause killing via ADCC as well as T-cell engagement. Xencor's technology also allows for Fc-knock-out variants that maintain structural Fc-domains without inducing FcγR-mediated cross-linking and potential off-target T cell activation.

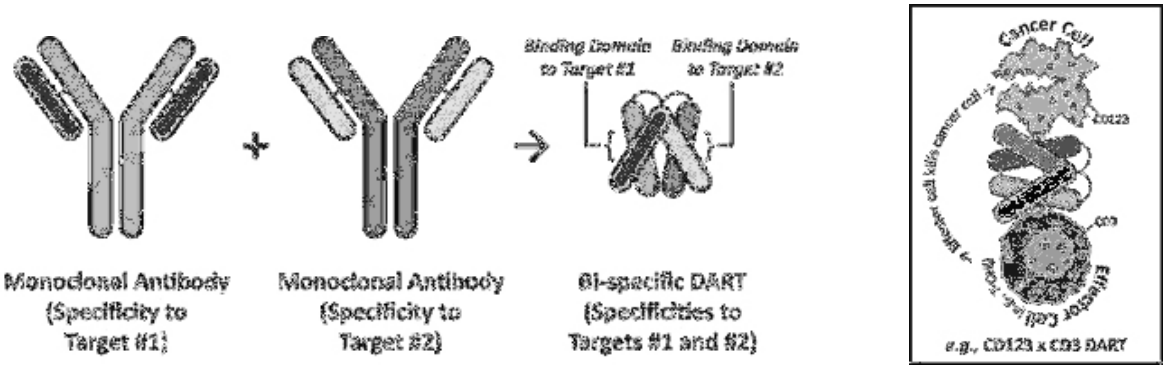
Xencor's bispecific technology facilitates manufacturing at high yields and ease of isolation compared to other approaches. Due to manufacturing difficulties, current bispecific antibodies are either non-humanized or incorporate novel peptide-linked antibody structures. Trion Pharmaceuticals has developed several bispecific rat/mouse chimeric antibodies including the EU-approved catumaxomab (EpCAM/CD3), ertumaxomab (HER2/CD3), Bi20 (CD20/CD3), and two unnamed GD2/CD3 and GD3/CD3 antibodies in early development. Amgen has developed blinatumomab, an anti-CD19 Fv fragment linked to an anti-CD3 Fv fragment by a peptide linker that demonstrated efficacy at extremely low levels in Phase II studies, though short half-life on the order of 3 hours requires constant infusion. MedImmune's novel MEDI-565 consists of two single chain antibodies linked by a flexible peptide linker and is currently in Phase I studies. Xencor's bispecific antibodies do not contain potentially immunogenic multi-peptide linkers, exhibit stability and half-life similar to well studied antibodies and have the potential to induce killing both by CD3-mediated T cell activation and FcγR-mediated ADCC.

Figure 6: Xencor's, DART, BiTE Bispecific Antibodies

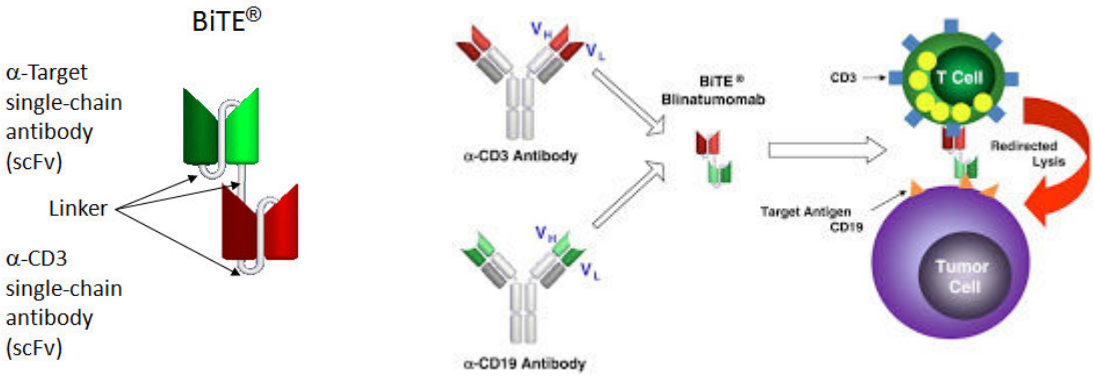
XNCR's Bispecifics Include FC Regions Enhancing Half-life



Macrogenics' DART Antibodies



Amgen's BiTE: Blinatumomab Structure and Function



Source: Adapted from Wickramasinghe, Discov Med. 2013 Oct;16(88):149-52.

Xencor's Novel Bispecific Candidates

Xencor is developing CD3 bispecific antibodies in pre-clinical studies for potential clinical application in liquid tumors. Xencor has disclosed two bispecific candidates targeting CD3/CD38 and CD3/CD123. Both CD38 and CD123 are promising potential bispecific targets and despite competition, provide important validation of Xencor's novel bispecific targeting technology. We believe that these compounds represent early-stage proof-of-concept of Xencor's potentially best-in-class bispecific technologies.

CD38, also known as cyclic ADP ribose hydrolase, is expressed on the surface of many immune cells including committed progenitor cells, B lymphocytes in germinal centers, plasma cells and at very low levels by mature resting cells and lymphocytes. It is expressed at high levels in many myelomas and is used as a prognostic marker in CLL where CD38 expression is correlated with a more aggressive cancer. Three traditional (non-bispecific) anti-CD38 antibodies are currently in development: GenMab (GEN-OMX: not Covered)/J&J (JNJ: not covered) are developing daratumumab in a Phase II trial in multiple myeloma, Sanofi (SNY: not covered) is developing SAR650984 in Phase I trials and MorphoSys (MOR-XETRA: not Covered)/Celgene (CELG: not covered) are jointly developing MOR202 in Phase I trials.

CD123, also known as the interleukin-3 α receptor (IL3α), is a cytokine receptor that is broadly up-regulated in B-cell malignancies, especially hairy cell leukemia. CSL Behring (CSL-AX: not covered) is developing a neutralizing antibody directed against CD123 in Phase I trials that has demonstrated promise in xenograft models of acute myelogenous leukemia (Data presented at ASH 2012). Additionally, a recombinant protein specifically targeting CD123+ cells consisting of diphtheria toxin bound to IL3α demonstrated excellent cytotoxicity against acute myeloid leukemia progenitor cells and spares normal progenitors, but a Phase I trial found it was poorly tolerated.

XmAb7195 – Severe Allergic Asthma and IgE Mediated Disease

Xencor's lead candidate, XmAb7195, is an anti-IgE antibody incorporating Xencor's proprietary immune inhibitor Fc domain and an Fv domain that targets the same IgE epitope as Xolair (omalizumab).

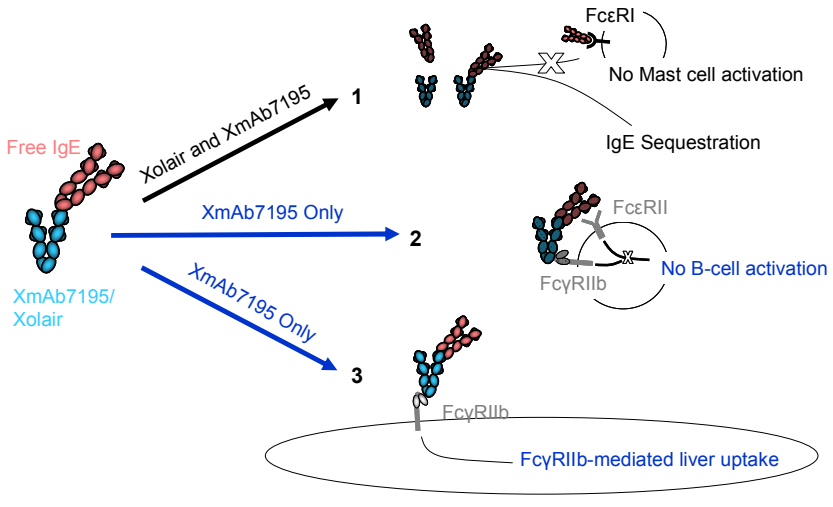
Xencor is planning to file an IND and initiate a Phase Ia single ascending dose trial of XmAb7195 in H1:14. The first-in-human study will assess safety and PK of XmAb7195 administered intravenously (doses to be determined following pre-IND meeting with the FDA) in parallel cohorts including healthy and allergic subjects. The study will assess efficacy in suppression of free and total IgE levels. Following successful safety assessment, a phase Ib multiple ascending dose trial in patients with mild to moderate asthma will be enrolled, potentially in early 2015. Since XmAb7195 shares the same Fc domain as XmAb5871, currently in Phase II studies, we anticipate it will be well tolerated in first-in-human studies. We anticipate data from the Phase 1a study by YE:14.

Xencor also intends to run an intravenous to subcutaneous bridging study in humans to prepare for subcutaneous administration in future clinical trials. The potential Phase II trial of XmAb7195 could occur in patients with poorly controlled asthma, including those ineligible for Xolair due to IgE/BMI restrictions (i.e.: those "off the dosing table"). Trials will likely run for 28-weeks with a 24-week extension to study long-term safety and benefit. Endpoints may include reduction in inhaled corticosteroid (ICS) dose and frequency of asthma exacerbations, similar to the Xolair trials in severe asthma.

XmAb7195 Reduces IgE Faster, More Potently and for Longer than Xolair

XmAb7195 was designed to work better than the current severe asthma standard of care, Xolair (omalizumab) an anti-IgE antibody. XmAb7195 differs from Xolair by two key mechanisms: 1) inhibiting IgE production by B-cells via binding the inhibitory FcγRIIb receptor and 2) removing bound IgE from the bloodstream via uptake facilitated by FcγRIIb binding to liver sinusoidal endothelial cells (Xolair merely sequesters it). XmAb7195's differentiated mechanisms reduces IgE 10-fold greater than Xolair *in vivo*. In pre-clinical studies in mice and chimpanzee's, XmAb7195 resulted in a larger drop and more sustained inhibition of IgE compared to Xolair and was well tolerated. We note that in addition to potential efficacy in severe asthma XmAb7195 may have potential application in other indications beyond those in which Xolair has utility, because of its differentiated mechanisms of action.

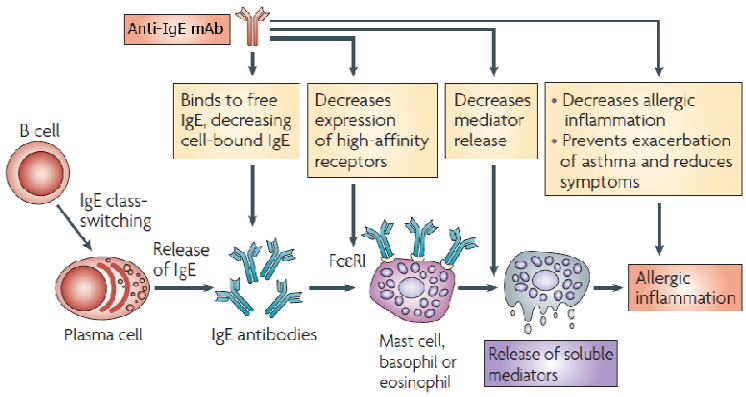
Figure 7: XmAb7195 – Novel Mechanisms of Action Imparted by Xencor's Fc-Engineered Technology



Source: Company data, Wedbush Securities, Inc.

IgE plays a critical role in allergic hypersensitivity reactions (type I hypersensitivity), functionally however, IgE's role is less clear though it is thought to provide immunity to parasitic infection. IgE is produced by B-cells and starts the allergic cascade by binding to FcεRI on the surface of basophils and mast cells, triggering cytokine release, further B-cell IgE production and ultimately inflammation. Reducing free IgE has been shown to result in profound reductions in elevated white blood cell counts, T-cell and B-cell numbers and FcεR1 receptors on dendritic cells in asthma patients. Additionally, IgE has recently been shown to play a direct role in long-term airway remodeling via interactions with mast cells, dendritic cells, macrophages and B-cells. This raises the possibility that effective anti-IgE therapies will cause long-term benefit independent of their inflammation-mediating effects.

Figure 8: Anti-IgE Antibodies Function by Mediating Inflammatory Response Pathways

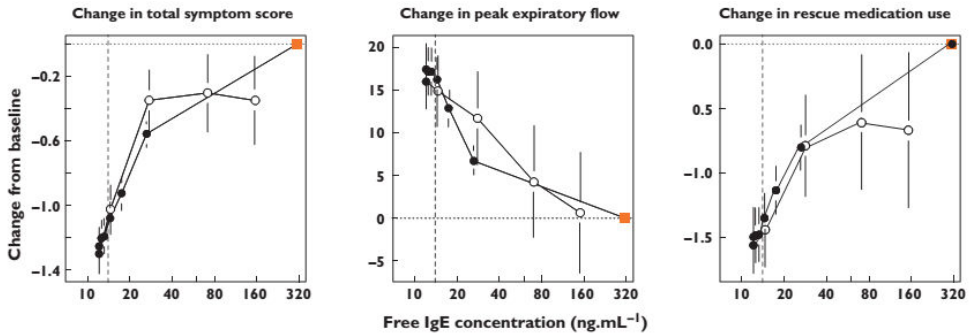


Source: Holgate and Polosa Nat Rev Immunol. 2008 Mar;8(3):218-30

Free IgE is a Biomarker that Correlates with Symptom Scores and Other Clinical Indicators

Importantly, XmAb7195's ability to clear IgE facilitates the use of a commercially available total serum IgE test as a marker of potential clinical response. This is noteworthy since free IgE is the critical mediator of immune response, but is too expensive to regularly test in the clinic. Free IgE levels may represent an important biomarker of response to XmAb7195 for asthma since they have correlated strongly with several important clinical outcomes including rescue inhaler use, peak expiratory flow and total symptom score (Lowe et al. 2005). Since XmAb7195 clears IgE from the serum (instead of simply binding it like Xolair), Xencor may be afforded the unique advantage of measuring IgE levels in the clinic to assess drug activity, dosing and safety, which could lead to increased physician comfort, optimized dosing regimes and potentially faster routes to registration.

Figure 9: Free IgE Correlates with Several Clinically Important Indicators

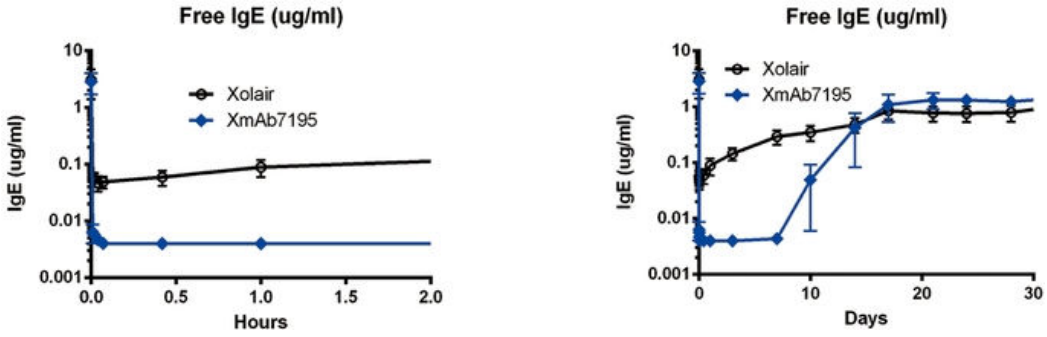


Source: Lowe et al. 2012 - Note that at concentrations below 14 ng/ml (dotted line) asthma symptoms were maximally reduced.

XmAb7195 was Well Tolerated and Demonstrated Activity in Pre-Clinical Studies

In pre-clinical studies in mice and chimpanzees XmAb7195 was well tolerated and resulted in a larger drop and more sustained inhibition of IgE compared to Xolair (omalizumab). Xencor studied the activity of XmAb7195 compared to Xolair in a study of 6 chimpanzees in which the animals received a single dose of either 5mg XmAb7195 or 5mg Xolair. XmAb7195 reduced free IgE to <0.004 µg/ml compared to a 0.05 µg/ml reduction in Xolair treated chimps. Importantly it was observed that XmAb7195 resulted in free IgE below the 0.004 µg/ml detection limit for 7 days. (Note that chimpanzees have much higher resting IgE levels than humans, roughly 2-4 µg/ml in this study compared to 0.096 µg/ml in humans).

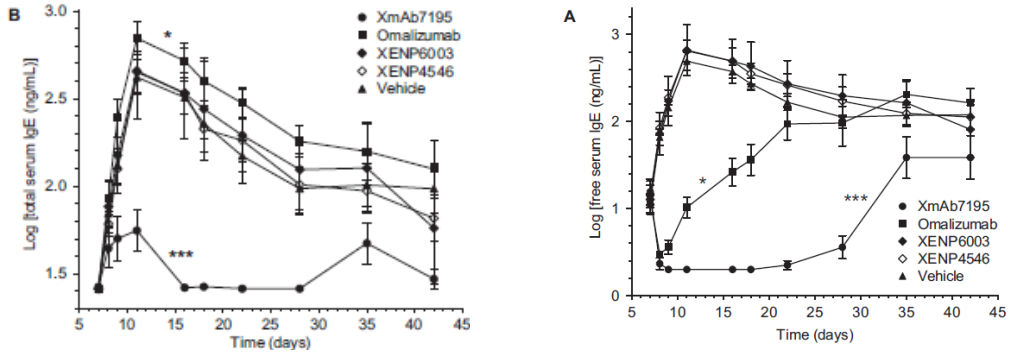
Figure 10: XmAb7195 Reduces Free IgE Levels more than Xolair in a Chimpanzee Model



Source: Company data, Wedbush Securities, Inc.

XmAb7195 also reduced IgE levels, but generally not IgG or IgM levels, in huSCID mice, mice transgenic for the Fcγ receptor and human fractionated plasma models further validating the ability of this drug to lower IgE levels. XmAb7195 was also well-tolerated with no adverse events in 12-week, multiple dose toxicology studies in cynomolgus monkeys at doses of up to 100 mg/kg.

Figure 11: XmAb7195 Reduces Free (A) and Total (B) IgE more than Xolair and Clears IgE Faster in huSCID mice



Source: Chu et al. J Allergy Clin Immunol. 2012 Apr;129(4):1102-15

Safety

We expect XmAb7195 to be safe and well tolerated based on preliminary but informative pre-clinical studies, on similarity to Xolair and natural history of genetically IgE deficient patients. Additionally, we note that XmAb7195 contains the same engineered Fc domain as Xencor's XmAb5871 that has been safely tested in humans.

Xolair (omalizumab) was extremely well tolerated in large clinical trials. Serious adverse events were rare and most significantly included anaphylaxis occurring in 3 of 3507 (0.1%) of patients. In post-marketing reports, Xolair causes anaphylaxis in 0.2% of patients, almost all in the first 3 doses of drug. This led to a black box warning for anaphylaxis on the Xolair product label. Since IgE is associated with the body's ability to fight parasitic infection, increased risk of these infections is broadly associated with IgE lowering therapies. This has not posed a substantial barrier to Xolair's adoption or clinical path in developed countries where parasitic infections are rare. We anticipate that results from clinical studies will determine if XmAb7195 will also carry a black box warning.

XmAb7195 is expected to lower IgE levels more substantially than Xolair, which may result in additional potential toxicities. Importantly, however, we note that the experience with IgE lowering therapies and genetic models suggests that there appears to be no serious adverse effects associated with even very low levels of IgE. An investigational antibody QGE031 (Novartis) that binds IgE with higher affinity than Xolair was well tolerated in Phase I trials. Furthermore, it has been observed that patients born with a genetic deficiency in IgE are known to be healthy and asymptomatic. Consequently we believe that XmAb7195 will be well tolerated in human studies.

XmAb7195 for Severe Asthma - Xolair Does Not Control IgE Sufficiently Limiting Efficacy

Asthma is a chronic inflammatory airway disease involving episodic breathlessness and wheezing with airway hyper-responsiveness to environmental stimuli. The disease manifests in allergic, or IgE-mediated, and non-allergic forms and affects more than 300 million people worldwide. Inhaled corticosteroids and short-acting β_2-adrenergic agonists control asthma for the majority of patients, however 5-10% of asthma patients fail to respond to steroid therapy and, if eligible (Figure 12), may be placed on Xolair. In severe asthma patients, control of the disease is critical to prevent long-term airway remodeling caused by chronic inflammation that leads to narrowing of the airway, progressive disease, and ultimately risk of mortality.

Xolair (omalizumab) is a monoclonal antibody that binds free IgE approved for moderate and severe allergic asthma patients in the US and severe patients in the EU. Xolair must be given in such large doses to be effective that roughly 1/3 patients with too high free IgE or too great a body mass are ineligible (Figure 12). Even in the eligible patient population, many patients cannot get to target free IgE levels or control disease. In this restricted market, Xolair sold ~$750M in the US and $1.3B worldwide in 2012.

Figure 12: EU (right) and US (left) Xolair Dosing Tables

Baseline IgE — Body Weight (kg)

(IU/ml)	20-25	<30	<40	<50	<60	<70	<80	<90	<125	<150	Dosing
30-100	75	75	75	150	150	150	150	150	300	300	Q4wk
<200	150	150	150	300	300	300	300	300	450	600	
<300	150	150	225	300	300	450	350	350	600	375	
<400	225	225	300	450	450	450	600	600	450	525	Q2wk
<500	225	300	450	450	600	600	375	375	525	600	
<600	300	300	450	600	600	375	450	450	600		
<700	300	225	450	600	375	450	450	525			
<800	225	225	300	375	450	450	525	600			
<900	225	225	300	375	450	525	600				
<1000	225	300	375	450	525	600					
<11000	225	300	375	450	600						
<12000	300	300	450	525	600						
<13000	300	375	450	525							
<15000	300	375	525	600							

Do Not Dose

Baseline IgE — Body Weight (kg)

(IU/ml)	30-60	<70	<80	<90	<150	Dosing
30-100	150	150	150	150	300	Q4wk
<200	300	300	300	300	225	Q2wk
<300	300	225	225	225	300	
<400	225	225	300	300		
<500	300	300	375	375		
<600	300	375				
<700	375					

Do Not Dose

Source: Company data, Wedbush Securities, Inc.

XmAb7195 – Indications Beyond Asthma

Xencor has indicated that they may look to further develop XmAb7195 in other indications beyond severe asthma. We note that a highly effective anti-IgE therapy may have broad utility in IgE mediated diseases. Xolair (omalizumab) has, for example, been tested in several studies beyond labeled indications (Figure 13). We highlight that XmAb7195 may offer efficacy to a greater extent than Xolair in indications other than severe asthma that could potentially offer label expansion opportunities or a more rapid route to registration, possibly including in conditions where Xolair has failed to offer efficacy. We believe that chronic idiopathic urticaria (CIU) a chronic and debilitating form of hives with limited treatment options, mastocytosis a mast-cell a rare disorder with a debilitating symptomology caused by mast-cell degranulation, and atopic dermatitis may offer either faster routes to registration or additional label opportunities. In CIU, the FDA accepted Genentech/Roche's supplement BLA for Xolair in CIU in October 2013 and a decision is expected in H2:14. We highlight several case reports in systemic mastocytosis that have indicated improvement in symptoms and laboratory findings after treatment with Xolair. We note that efficacy in systemic mastocytosis could be profound and life-altering potentially supporting a rapid route to registration for XmAb7195. Additionally, case reports in atopic dermatitis are promising, but a 20 patient placebo-controlled trial found only a small, non-significant, improvement in patch test scores, indicating some reduction in allergic hypersensitivity.

Figure 13: Xolair has been Tested in Many Indications

Condition	Phase	N
Dermatitis	IV	NR
Bullous Pemphigoid	IV	2
Cystic Fibrosis	IV	14
Urticaria (CIU)	III	~1000
Acute Interstitial Nephritis	II	20
Allergis Rhinitis	II	>55
Chronic Rhinosinusitis	II	21
Mastocytosis	II	40
Gastroenteritis	II	30
Food Allergies	II	>50
Esophagitis	II	30
Anaphylaxis	II	30
Systemic Lupus Erthematosus	I	30
Hyper IgE Syndrome	I	1
Chronic Sinusitis	IV	Withdrawn
Chronic Obstructive Pulmonary Disease	II	Withdrawn

Source: Company data, Wedbush Securities, Inc.

Asthma and the Anti-IgE Competitive Landscape

Several anti-IgE based therapies are currently in development across several indications, additionally Xolair is being tested in more than 13 clinical indications beyond severe asthma.

Novartis (NVS: Not Covered) is developing QGE031, a bio-superior Xolair that binds IgE with higher affinity. We note however, that QGE031 only binds IgE with higher affinity vs. Xolair and is not Fc-engineered and thus unlikely to confer benefits of higher IgE clearance and result in lower production of IgE like XmAb7195. First data for QGE031 was presented in September 2013 at European Respiratory Society (ERS) Annual Congress 2013 in Barcelona, Spain.

QGE031 was tested in a Phase Ib trial that enrolled 110 patients randomized 70:28:12 to QGE031, placebo or Xolair respectively. Subjects received 2-4 doses of 0.2-4mg/kg QGE031 subcutaneously. 2 patients receiving QGE031 at 2 mg/kg withdrew due to symptoms considered unrelated to the drug. 4 urticaria events were recorded, 1 in the 0.6 mg/kg and 3 in placebo, in 3 patients. QGE031 reduced free IgE below quantification levels (likely 0.004 µg/ml) at all doses, significantly better than Xolair. QGE031 was well tolerated with no serious AEs and reduced free circulating IgE, FcεRI, surface IgE, and SPT responses significantly greater and for a longer duration than Xolair. A Phase II trial was initiated early this year with an estimated 457 patients comparing the safety and efficacy of QGE031 against placebo and Xolair comparators

Genentech/Roche ran a phase II trial of PRO98498 in 2007, a monoclonal antibody with tighter IgE binding than Xolair, but development has been discontinued. Roche is also currently running a phase II trial of quilizumab, a monoclonal antibody targeted to membrane-bound IgE, aiming to deplete IgE B-cells rather than neutralizing free IgE.

An AstraZeneca/MedImmune partnership was developing MEDI-4212, an anti-IgE antibody, but development has been terminated.

Figure 14: Selected Therapies in Development for Asthma/IgE Related Disorders

Company	Name	Type	Target	Phase
Teva	reslizumab	mAb	IL-5	Phase III
MedImmune	benralizumab	mAb	IL-5R	Phase III
GSK	mepolizumab	mAb	IL-5	Phase III
Genentech	lebrikizumab	mAb	IL-13	Phase III
Amgen	mogamulizumab	mAb	CCR4	Phase III
Amgen	brodalumab	mAb	IL-17	Phase III
Amgen	AMG-853	SMI	CRTh2	Phase III
AB Science	masitinib	SMI	TKI	Phase III
Novartis	**QGE031**	**mAb**	**IgE**	**Phase II**
Roche	**quilizumab***	**mAb**	**Bound IgE**	**Phase II**
Sanofi/Regeneron	dupilumab	mAb	IL-4Ra	Phase II
Revalesio	RNS60	Ionic	Non-specific	Phase II
Novartis	QAW039	SMI	CRTh2	Phase II
Novartis	QAX576	mAb	IL-13	Phase II
MedImmune	tralokinumab	mAb	IL-13	Phase II
MediciNova	ibudilast	SMI	PDE-4	Phase II
JNJ	JNJ-40929837	SMI	Lta4h	Phase II
Genentech	MEMP1972A	mAb	M1 prime	Phase II
Aztrazeneca/MedImmune	**MEDI-4212**	**mAb**	**IgE**	**Terminated**
Genentech	**PRO98498**	**mAb**	**IgE**	**Terminated**

*data in H2:14 in Chronic idiopathic urticaria (CIU)

Source: Company data, Wedbush Securities, Inc.

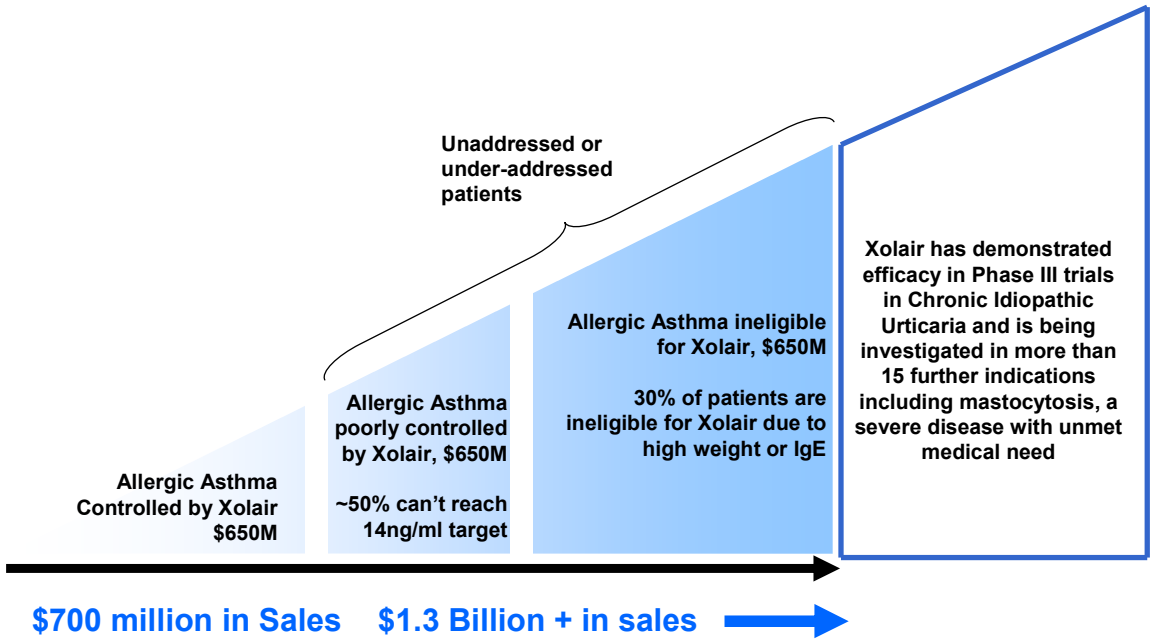
Market and Competition

There are roughly 18 million diagnosed asthma sufferers in the US, 1 million of whom have moderate and severe allergic asthma uncontrolled by steroid and β2-adrenergic agonist therapy. By our estimates, roughly 33,000 patients are treated with Xolair in the US every year of an eligible/accessible patient population of 322,000, implying a 10% market penetration. We estimate 1/3 of patients are not eligible for Xolair and that as many as 50% are experiencing inadequate control of their asthma symptoms on Xolair. Xolair sales in 2012 were $1.3 billion globally.

We estimate XmAb7195, which is likely to be more broadly efficacious than Xolair and find utility where Xolair does not, with 15% market penetration could achieve worldwide sales of $1.2 billion in the severe asthma setting. This represents $12 per share based upon a 6x multiple on sales of 2022 sales discounted annually 45%.

We expect that XmAb7195's potential superior IgE control will lead to wide spread off-label use in other IgE mediated diseases. Upside to our estimates exists in urticaria, where Xolair has demonstrated efficacy in phase III trials, rhinosinusitis, EGID, and peanut allergy response among other indications in which Xolair is currently being clinically investigated. We view use beyond the asthma setting as upside to our price target.

Figure 15: XmAb7195 Market Opportunity



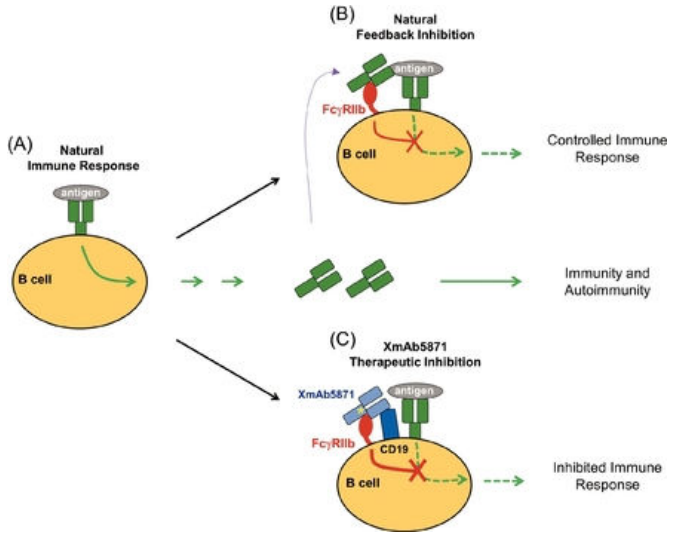
Source: Company data, Wedbush Securities, Inc.

XmAb5871 – Autoimmune Diseases

Xencor is developing XmAb5871, an anti-CD19 monoclonal antibody incorporating Xencor's proprietary immune inhibitor Fc domain (FcγRIIb) for the treatment of auto-immune disease. XmAb5871 was designed to inhibit B-cell activation via *co-engagement* with B-cell receptor (BCR), but does not kill or deplete B-cells like rituximab. XmAb5871 binds the CD19 receptor on B-cells (Fv-domain) and recruits FcγRIIb receptor (Fc-domain) blocking the BCR signaling pathway, preventing activation and potentially blocking disease pathology. FcγRIIb binds normal antibodies (IgG) with low affinity, serving as a check on unregulated immune activation by inhibiting antibody production (i.e.: negative feedback). FcγRIIb is a mechanistically validated therapeutic target for B-cell mediated inflammation, autoimmunity and allergy. Additionally, pre-clinically FcγRIIb-knock-out mice have been shown to spontaneously develop lupus-like autoimmune disease, implicating a dependence upon this receptor in B-cell disorders. FcγRIIb receptor activation prevents B-cell mediated T-cell activation, cytokine release and B-cell antibody production and is also expressed on dendritic cells where XmAb5871 may inhibit dendritic cell mediated T-cell activation and response. Importantly, XmAb5871 only represents one example of Xencor's differentiated FcγRIIb Fc-technology, as several other co-targets beyond CD19 could be contemplated.

Under a 2010 agreement, Amgen has an option to purchase a license to XmAb5871 after completion and review of a Phase IIb trial. Xencor is eligible for tiered high-single-digit to high-teen royalties on net sales and $62M in clinical milestones, $150M in filing and approval milestones and $225M in payments for meeting certain sales goals. We anticipate that Amgen is likely to exercise their option on XmAb5871.

Figure 16: Inhibiting B-cells by FcγRIIb Activation.



Source: Company data, Wedbush Securities, Inc.

Phase Ib/IIa Trial Design

In January 2013, Xencor initiated a Phase Ib placebo-controlled, double-blind, multiple ascending dose trial to assess the safety, tolerability and PK/PD of XmAb5871 in patients with active rheumatoid arthritis on stable non-biologic DMARD therapy. In the first part of the study Xencor dosed 29 RA patients at 0.3, 1, 3, and 10 mg/kg biweekly for six weeks. Inclusion criteria include patients with active RA at screening defined as ≥ 4 swollen joints and ≥ 4 tender/painful joints (out of 28 joints examined) and at least 1 of erythrocyte sedimentation rate (ESR) ≥ 28 mm/hr, C-reactive protein (hsCRP) ≥ 10 mg/L, or morning stiffness ≥ 45 minutes.

In October 2013, Xencor initiated the second "Phase IIa" part of the study that will enroll 30 patients with active disease on non-biologic DMARD therapy randomized 2:1 XmAb5871 to placebo at the highest 10 mg/kg dose every 14 days for a total of six doses. In this part of the study, active disease is defined as ≥ 5 swollen joints and ≥ 5 tender/painful joints (out of 28 joints examined), positive Rheumatoid Factor (RF) or anti-citrullinated protein antibodies (ACPA), and hsCRP ≥ 10 mg/L. The primary outcome measures of the study include determination of safety and tolerability, including measures of immunogenicity of multiple biweekly doses XmAb5871 administered by IV infusion. Secondary endpoints will assess clinical outcome measured as Disease Activity Score 28 using C-reactive Protein (DAS28-CRP) after 13 weeks. As of September 2013, XmAb5871 was found to be well-tolerated. A serious adverse event, infusion-related reaction with hypotension, was noted in one patient resulting in discontinuation. No other patients discontinued therapy and other adverse events reported in more than one patient (that may be related to therapy) include nausea, vomiting, fever-increased temperature, headache and bronchitis. Preliminary immunogenicity testing data for the first 2 cohorts through the treatment phase of the study have been negative. Data from the Phase Ib/IIa trial is expected in the second half of 2014.

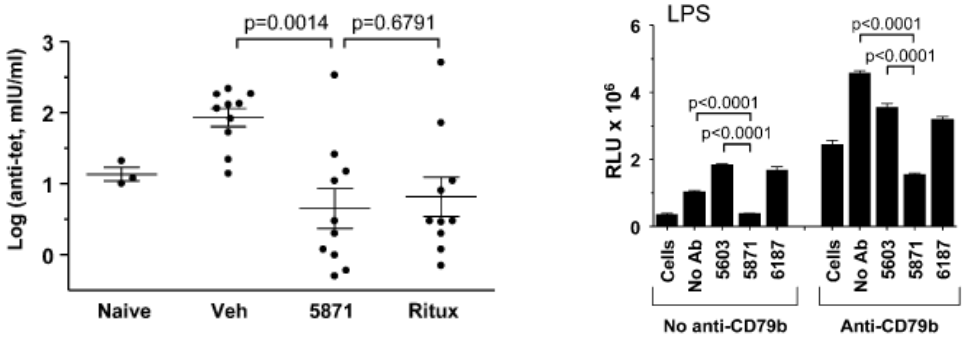
First-in-Human Phase Ia Trials Demonstrated Activity

In December 2012, Xencor reported results from their Phase 1a clinical trial of XmAb5871. The randomized, blinded, placebo-controlled study assessed a single ascending dose of XmAb5871 in 48 healthy male volunteers. Subjects were randomized 3:1 to receive IV infusions of XmAb5871 or placebo in one of seven dose cohorts (0.03-10.0 mg/kg). The study assessed the PK and immunogenicity of single-dose XmAb5871 and also measured several biomarkers of efficacy. XmAb5871 was well tolerated and no dose-limiting toxicities or serious adverse events were observed. The most common adverse events observed included (mild-moderate) GI symptoms including nausea (1-severe), vomiting, abdominal pain, epigastric discomfort and diarrhea. Anti-drug antibodies, that did not impact drug activity, were observed in 44% patients, 22% of which were greater than 2-fold above baseline. Biomarkers of efficacy showed target saturation and B-cell suppression at low-bi-weekly doses (0.03 mg/kg). XmAb5871 reduced B-cells by 50% from baseline at all doses, and B-cell counts recovered proportionally to drug serum clearance. Challenge-based tests of XmAb5871 were conducted by immunizing healthy subjects with tetanus and KHL to elicit antibody responses. XmAb5871 effectively suppressed immune responses at all but the lowest dose level, with placebo, treated patients exhibiting a 12-fold increase in anti-tetanus antibody levels vs. a 4-fold increase in XmAb5871 treated subjects.

Pre-clinical Data Highlight Reversibility and Efficacy vs. Rituximab

Pre-clinical data demonstrate XmAb5871 inhibits immune response to tetanus challenge in SCID mice engrafted with SLE PBMC (Figure 17). XmAb5871 was shown, both *in vitro and in vivo,* to suppress activity of B-cells donated from patients with lupus and rheumatoid arthritis. Importantly, we note that XmAb5871 is able to inhibit the immune response as effectively as rituximab without killing B-cells. Additionally, Xencor demonstrated that XmAb5871 was well tolerated in 12- and 24-week multiple dose pre-clinical studies in monkeys. No adverse events were noted in doses up to 200 mg/kg.

Figure 17: XmAb5871 Inhibits Immune Response in SCID Mice Engrafted with SLE PBMC and in Cultured Cells (left)



Source: Horton et al. J Immunol. 2011 Apr 1;186(7):4223-33

XmAb5871 an Alternative to B-cell Depletion for Autoimmune Disease

Current therapies for autoimmune disease leave much to be desired; if found to be safe and effective XmAb5871 may find broad utility in autoimmune disease and B-cell-associated disorders. Increasingly adopted B-cell depletion therapy for autoimmune disease, rituximab, improves arthritis symptoms but depresses the immune system and increases infection risk. We believe that XmAb5871, by inducing inhibition of B-cell activation instead of B-cell depletion may be similarly efficacious to current B-cell targeted therapies but better tolerated, with lesser potential infection risk. Additionally, TNF-alpha inhibitors are associated with infection risk and other serious side effects, and also frequently stop working in patients after as little as 1 year on therapy. Ultimately, we believe XmAb5871 may offer a compromise between B-cell depletion and limited efficacy of TNF-alpha inhibitors, potentially meeting a significant unmet medical need in autoimmune disease.

Xencor and potential option/partner Amgen may initially develop XmAb5871 to establish proof of concept in systemic lupus erythematosus (SLE) or rheumatoid arthritis, however several other orphan indications, with high-unmet medical need may offer more targeted and faster routes to registration. We would anticipate however that eventually the therapy may find off-label use broadly for autoimmune disorders paralleling the current use of rituximab in these indications.

B-cell Targeted Therapies Competitive Landscape

Numerous therapies are in development for treatment of B-cell related autoimmune diseases. Medi-551 (MedImmune), a CD19 antibody with sugar modifications to enhance ADCC is under development in relapsing remitting multiple sclerosis, but because it results in B-cell depletion will suffer from the same irreversibility problems as rituximab.

Figure 18: Selected B-cell Depleting Therapies Approved and in Trials

Company	Therapeutic	Target	Format	Indication	Clinical stage
Roche	rituximab (Rituxan)	CD20	Chimeric IgG1	RA	Approved
GSK	belimumab (Benlysta)	BAFF	Human IgG1	SLE	Approved
Eli Lilly	tabalumab	BAFF	Human IgG4	SLE	Phase 3
Anthera	blisibimod	BAFF	Peptibody	SLE	Phase 3
GSK	ofatumumab (Arzerra)	CD20	Humanized IgG1	RA	Phase 3
Roche	ocrelizumab	CD20	Humanized IgG1	SLE	Phase 3
UCB/Immunomedics	epratuzumab	CD22	Humanized IgG1	SLE	Phase 3
Merck	atacicept	BAFF/APRIL	Receptor Fc fusion	SLE	Phase 2/3
Immunomedics	veltuzumab	CD20	Humanized IgG1	RA	Phase 2
Amgen	AMG-557	B7RP1	Human IgG1	SLE	Phase 1
MedImmune	**MEDI-551**	**CD19**	**Humanized IgG1 afucosylated**	**RRMS**	**Phase 1**
UCB	CDP7657	CD40L	Pegylated Fab	SLE	Phase 1
MedImmune	MEDI-570	ICOS	Humanized IgG1 afucosylated	SLE	Phase 1
Novo Nordisk	NN8828	IL-21	Human IgG1	RA	Phase 1

Source: Blüml et al. Arthritis Research & Therapy 2013, 15(Suppl 1):S4

Market Opportunity

Rheumatoid arthritis is a chronic auto-immune disease that causes joint pain, difficulty walking and cardiovascular disease. RA affects nearly 1.3 million adults in the US. Current treatment calls for disease-modifying anti-rheumatic drug (DMARD) therapy including steroids, TNF-α inhibitors, and B-cell depleting rituximab, but not all patients are adequately controlled.

Humira (AbbVie), a TNF-α inhibitor for the treatment of auto-immune diseases sold $9.5 billion last year in rheumatoid arthritis, psoriatic arthritis, juvenile idiopathic arthritis, ankylosing spondylitis, Crohn's disease, ulcerative colitis and moderate to severe chronic psoriasis.

Systemic lupus erythematosus (SLE) is a chronic auto-immune disease of unknown etiology and highly heterogeneous presentation. High dose steroids and immunotherapy have improved 5 year survival to 90% from 50%, but flare-ups still occur regularly. Constant flare-ups eventually lead to renal failure, neurological damage and death. Lupus is estimated to affect 60,000 patients in the US[1] and constitutes a significant unmet medical need.

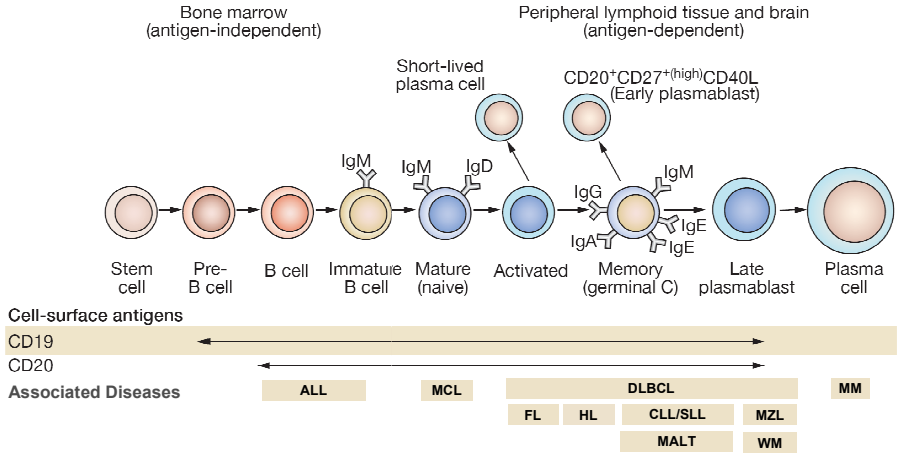
We estimate that there are approximately 370,000 rheumatoid arthritis patients refractory to biologic and DMARD therapy in the US and 85,000 in the top 5 EU countries. If priced similar to Humira (TNF-α inhibitor) ($25,000/yr), XmAb5871 could generate $1.2 billion in sales in RA alone in the US and to 5 EU markets with just 10% penetration of these markets. We believe upside to our sales expectations exist should XmAb5871 show efficacy in additional indications including SLC. We estimate that with a 15x multiple of royalties from potential partner Amgen (10%) on sales of $1.2 billion could occur in 2022 and discounted 45% annually represent ~$3/share to Xencor's valuation.

[1] http://www.ncbi.nlm.nih.gov/pubmed/22472925

XmAb5574 / MOR208 – CD19 Targeted mAb for B-cell Malignancies

Xencor has developed XmAb5574, an anti-CD19 monoclonal antibody incorporating Xencor's proprietary enhanced ADCC Fc domain, targeting FcγRIIIa (~40-fold higher affinity) and FcγRIIa (~10-fold higher affinity), for the treatment of leukemias and lymphomas. Xencor partnered XmAb5574, under a 2010 agreement with MorphoSys and is eligible for high-single-digit to low-teen royalties on net sales, $62 million in clinical milestones, $187 million in filing and approval milestones and $50 million of payments for meeting certain sales goals. CD19 is an early marker of B-cell lineage commonly used as a diagnostic tool in lymphomas and leukemia's because it is a rarely lost in neoplastic transformation. XmAb5574 is designed to bind CD19 to induce ADCC killing of B-cells earlier in their maturation cycle and more effectively than rituximab (Rituxan) (Figures 18, 19).

Figure 19: CD19 is an Early Marker of B-cell Lineage



Source: Adapted from Raufi et al. Cancer Manag Res. 2013 Aug 27;5:225-33

Phase II Trials

Xencor's partner MorphoSys is currently running two Phase II trials, one in non-Hodgkin's lymphoma (NHL) and one in B-cell acute lymphoblastic leukemia (B-ALL). The open-label trial in B-ALL (NCT01685021) is enrolling 30 patients with B-ALL refractory to at least one prior therapy and patients with Philadelphia chromosome can only be enrolled if they are refractory to prior tyrosine kinase inhibitor (TKI) therapy. The primary outcome measure is overall response rate (ORR) at 7-months; secondary outcome measures include duration of response as measured by bone marrow aspirates, safety, pharmacokinetics and anti-XmAb5774 antibodies. A second open-label trial in NHL (NCT01685008) is currently enrolling 120 patients with FL, MCL, DLBCL, MALT/MZL or other indolent B-cell NHL refractory to CD-20 directed therapy rituximab. The primary outcome measure is overall response rate (ORR) at 4-years; secondary outcome measures include duration of response as measured by bone marrow aspirates, safety, pharmacokinetics and anti-XmAb5774 antibodies.

Additionally, Dr. Woyach at The Ohio State University Comprehensive Cancer Center, is planning to enroll an open-label, investigator-sponsored Phase II trial (NCT02005289) of MOR208 in combination with lenalidomide. This trial plans to enroll 40 Richter negative intermediate or high risk CLL, SLL or B-PLL patients, half naïve and half relapsed/refractory to receive MOR208 IV on days 1, 2, 8, 15 and 22 of a 28-day cycle and lenalidomide PO daily after day 8 for up to 12 cycles. The primary endpoint of the study will include proportion of patients achieving a response (CR, CRi, nPR, or PR) as defined by IWCLL 2008 criteria at 6 months. Secondary outcomes of the study are expected to include ORR, PFS, time to next treatment and OS at 12-months. The study will also examine cytogenetic factors, NK and T-cell activation and changes in protein expression.

Figure 20: Response Rate in the Phase I/IIa Trial of XmAb5574 in CLL

Responses by Physical Exam/Dose	0.3	1	3	6	9	12	Total (%)
Complete Response	-	-	-	-	-	-	0
Partial Response	-	-	2	1	3	12	18 (66.7)
Stable Disease	1	1	1	1	0	4	8 (29.6)
Progressive Disease	-	-	-	-	-	-	0
Unknown	-	-	-	-	-	-	1 (3.7)
Responses by CT Scan							
Complete Response	-	-	-	-	-	-	0
Partial Response	-	-	-	1	2	1	4 (14.8)
Stable Disease	1	1	2	1	1	14	20 (74)
Progressive Disease	-	-	1	1	-	-	2 (7.4)
Unknown	-	-	-	-	-	-	1 (3.7)

Source: Company data, Wedbush Securities, Inc.

XmAb5574 Was Well Tolerated in Phase I/IIa Trials

Xencor conducted an open-label, multi-dose, single-arm Phase I trial of XmAb5574 in 27 heavily pre-treated patients with CLL (median 4 treatments, range 1-13). Patients received 0.3, 1, 3, 6, 9 or 12 mg/kg (1, 1, 3, 3, 3 and 16 patients respectively) on day 1 and 4 of week 1 and day 1 of weeks 2-8 of the study. All patients had received a prior CD20 antibody treatment (96% were rituximab). Primary endpoints were to determine maximum tolerated dose (MTD), describe toxicity and characterize pharmacokinetics. XmAb5574's half-life was determined to be 14-days and the drug was safe and well tolerated. One dose limiting toxicity event occurred in the 12 mg/kg arm of neutropenia lasting ≥7 days with febrile neutropenia. Grade ≥ 3 neutropenia occurred in 3 patients, thrombocytopenia in 2. Grade < 3 infusion reactions occurred in the majority of patients. (Figure 21) Initial evidence of efficacy was also noted, including 18 (67%) partial response' (PR) IWCLL 1996 guidelines (physical exam) and 4 PRs (15%) by IWCLL 2008 guidelines (CT scan). No complete responses were observed, though this observation is not unexpected given the highly pre-treated patients, and progression on prior anti-CD20 therapies.

Figure 21: Adverse Events Occurring in the Phase I/IIa Trial of XmAb5574 in CLL

Preferred Name	0.3	1	3	6	9	12	*Total (%)*	*Preferred Name*	0.3	1	3	6	9	12	*Total (%)*
Dose Limiting Toxicities								*All Grade Toxicities In ≥ 10% of Patients*							
Neutropenia lasting ≥7 days	-	-	-	-	-	1	*1 (3.7)*	*Infusion Reaction*	1	1	3	3	2	14	*24 (88.9)*
with febrile neutropenia								*Increased AST*	1	-	2	1	-	1	*5 (18.5)*
Grade ≥ 3 Toxicities								*Increased ALT*	-	-	2	1	-	2	*5 (18.5)*
Neutropenia	-	1	-	-	-	2	*3 (11.1)*	*Neutropenia*	1	1	-	-	-	3	*5 (18.5)*
Thrombocytopenia	-	-	-	-	-	2	*2 (7.4)*	*Thrombocytopenia*	-	-	-	-	1	4	*5 (18.5)*
Febrile Neutropenia	-	-	-	-	-	1	*1 (3.7)*	*Fever*	1	-	1	-	1	1	*4 (14.8)*
Tumor Lysis Syndrome	-	-	-	-	-	1	*1 (3.7)*	*Chills*	1	-	1	1	-	-	*3 (11.1)*
Increased AST	-	-	-	-	-	1	*1 (3.7)*	*Diarrhea*	-	-	-	1	-	2	*3 (11.1)*
								Hypocalcemia	1	-	1	-	-	1	*3 (11.1)*

Source: Company data, Wedbush Securities, Inc.

CD19 Targeted Therapy Competitive Landscape

CD19 has been known as an attractive target for B-cell related malignancies for many years; however, early monoclonal antibody treatments were ineffective, though safe. Recently, several engineered anti-CD19 therapies have demonstrated promise in the clinic.

Amgen is developing blinatumomab, a CD19/CD3 bispecific T-cell engaging (BiTE) antibody in Phase II trials in NHL and ALL. The Phase II (Topp et. al, 2011) enrolled 21 patients with B-lineage ALL and administered blinatumomab 15 $\mu g/m^2$/24 hours continuously for week 1-4 of a 6-week cycle. HSCT was allowed at any time after cycle 1 of the trial if a donor was available. 16/20 patients achieved a minimal residual disease (MRD) response within 4 cycles. After 33 months, 12/20 patients were still in CR, resulting in a relapse-free survival of 61%. 4 of 6 patients who did not receive HSCT or any other therapy after blinatumomab were still in remission at median follow-up of 30 months. Blinatumomab was well-tolerated; the most common serious AE was reversible lymphopenia. Several trials in B-cell ALL and one in DLBCL are ongoing.

MedImmune has developed an anti-CD19 antibody (MEDI-551) that exhibits enhanced ADCC properties by engineering the glycosylation profile. A Phase I/IIa trial found 4 of 20 (20%) evaluable CLL patients achieved partial response and 13 (65%) had stable disease.

A CD19-specific dual-signaling chimeric antigen receptor (CAR-T therapy) was recently tested in B-cell ALL at the Memorial Sloan Kettering Cancer Center. All 5 patients achieved complete response as assessed by PCR. Therapy was well-tolerated although cytokine release syndrome required steroid therapy in some patients.

Market Opportunity

XmAb5574/MOR208 may find use in almost all B-cell related malignancies where rituximab is used, including CLL, ALL, B-cell NHL, MCL, Follicular lymphomas, MALT lymphomas and DLBLC. The market for B-cell related therapies is becoming, in our opinion, crowded with several new therapies including BTK-inhibitors (ibrutinib, IMBRUVICA - PCYC: Outperform), PI3K-inihbitors (idelalisib, CAL-101 – GILD: not-covered) and new B-cell depleters (obinutuzumab/GA-101 – ROG: not covered) as well as potentially promising CAR-T therapies. However, we note that XmAb5574/MOR208 is differentiated in potential applicability as a single-agent to acute lymphoblastic leukemia (ALL), a fast-growing cancer of a type of less-mature white blood cells that do not express CD20 sufficiently for current B-cell depleters to be efficacious (Figure 19). Approximately 6,000 patients are diagnosed with ALL each year in the US, and it is commonly treated with chemotherapy in the front-line setting (remission induction). We believe that similar to the use of B-cell depleters such as rituximab in the R-CHOP regime common to the treatment of CD20 expressing B-cell malignancies (NHL, CLL etc.), XmAb5574/MOR208 could be used in front-line therapy in CD19 expressing malignancies together with current chemotherapy. Furthermore, we believe XmAb5574/MOR208 will be rapidly being adopted in earlier lines of therapy as physician adoption mirrors rituximab's use in other hematologic malignancies.

We estimate the market opportunity for XmAb5574/MOR208 in the front-line ALL setting alone could be worth $600M in the US alone and anticipate an annual course of therapy could cost $110,000, assuming 1-year of therapy (average duration of current therapies for ALL is estimated to be 1.5-3 years). Additionally, approximately 10,000 new cases of ALL are diagnosed annually in the EU. We estimate that in the ALL setting alone, XmAb5574/MOR208 could achieve peak penetration into 75% of an addressable market of 50% (to take into account competition from current and emerging therapies) representing an estimated $600 million annual opportunity at an average 1 year of therapy and $110,000 annual cost of therapy. We estimate that with a 15x multiple of royalties from partner MorphoSys (10%) on peak sales of $600 million could occur in 2022 and discounted 35% annually represent an estimated $3/share to Xencor’s valuation.

Partnerships and Licensing Agreements

Xencor's technology and approach to antibody engineering has been validated through several significant partnerships that provide the company with non-dilutive capital and as much $1.31 billion in total milestone payments ($240M clinical, $541M regulatory, $526.5M sales goal related). Xencor is currently developing a pipeline of 9 partnered products with 7 companies, 4 of these partnered products are currently in clinical trials. We believe that Xencor's strategic partnering of their product pipeline selectively diversifies clinical, regulatory and commercial risk while offering non-dilutive capital to further develop internal candidates.

Figure 22: Xencor's Partnerships Milestone/Disclosed Targets

Partner	Development Milestones	Regulatory Milestones	Sales Milestones	Total
MorphoSys	$62	$187	$50	*$299*
Amgen	$62	$150	$225	*$437*
Alexion	$51	$168	$180	*$399*
Boehringer Ingelheim	$9	$6	$12	*$27*
CSL 2009	$38	$20	$31	*$89*
CSL 2013	$8	$4	$25	*$37*
Janssen	$6	-	$4	*$10*
Merck	$4	$6	-	*$10*
Total	**$240**	**$541**	**$527**	***$1,308***

Partner	Year	Licensed Technology / Antibody	Indication	Milestones	Royalties	Stage
Product Development:						
Amgen	2010	XmAb5871	Autoimmune disease	Yes	Yes	Phase 1
MorphoSys	2010	XmAb5574/MOR208	Oncology	Yes	Yes	Phase 2
Technology License:						
Alexion	2013	Xtend technology	Various	Yes	Yes	Preclinical

Source: Company data, Wedbush Securities, Inc.

Amgen – XmAb5871

Under a 2010 agreement, Amgen holds an option to license the commercial rights to XmAb5871 and certain related products worldwide. If the option is exercised by Amgen (following Phase II POC data) Xencor will be eligible for royalties in the high single-digit to high-teen's on net sales worldwide subject to minimal annual royalty payments and milestones of up to $437M split into $62M in clinical milestones, $150M in regulatory milestones and $225M in milestones related to sales goals. Amgen's royalty obligations continue on a country-by-country basis until the later of the last valid patent claim expires or 10 years following first commercial sales.

Under the option agreement, Xencor will lead research, manufacturing and clinical development up to completion of the phase II POC trial. Amgen's option is exercisable following completion of the Phase II POC clinical trial and expiration of the 90-day review period following delivery of the trial results. The agreement terminates on March 23, 2017, or March 23, 2021 if Amgen exercises an option to take over clinical development due to Xencor's failure to perform. If Amgen exercises their option to license, further development, commercialization, manufacture, distribution, market, promotion and other costs will be the sole responsibility of Amgen. We anticipate positive data from the Phase II POC trial and that Amgen will exercise their option to license the product based upon results from previous trials of XmAb5871.

MorphoSys – XmAb5574

Under a 2010 agreement with MorphoSys, Xencor is eligible for high single-digit to low-teen royalties on net sales worldwide of XmAb5574 and other CD19 antibodies with Xencor's cytotoxic Fc technology. Xencor is also eligible for $229M in milestones split into $62M in clinical milestones, $187M in regulatory milestones and $50M in payments related to sales goals. MorphoSys' royalty obligation continues on a country-by-country basis until the later of the last valid patent claim expires or 11 years after the first commercial sales.

Alexion - Xtend

Under a 2013 agreement, Alexion was granted a five-year research license for Xencor's Xtend technology in six different target programs as well as an option to purchase commercial rights for those compounds. If Alexion exercises their option, Xencor will be eligible for low single digit royalties on worldwide net sales and payments of up to $70.5M including a $4M upfront option fee. Alexion's royalty obligations continue on a country-by-country basis until the expiration of the last-to-expire valid claim.

Merck – Fc domains

Under a 2013 agreement with Merck Sharp Dohme Corp (Merck), Xencor provided Merck with a non-exclusive commercial license to certain of Xencor's Fc domains and contingent options to purchase additional non-exclusive commercial licenses. Xencor is eligible for undisclosed milestones and royalties as well as a $0.5M annual maintenance fee.

CSL Limited

Under a 2013 agreement, CSL limited was granted a non-exclusive commercial license to apply Xencor's Xtend technology to one of CSL's compounds. Xencor received an up-front payment of $0.5M and is eligible for clinical development milestones on the licensed product.

In 2013, Xencor and CSL amended a 2009 agreement covering CSL362, eliminating a contingent milestone and reducing the royalty rate in exchange for an up-front payment of $2.5M. Under the 2009 agreement, CSL was granted a non-exclusive research license and an option to purchase up to 5 commercial licenses. In 2011, CSL elected to exercise one of its options for a commercial license for CSL362. Xencor is eligible for annual license payments of $0.3M a $1M option exercise fee per license, milestones and royalties on net sales.

Boehringer Ingelheim – Manufacturing anti-TNF

Under a 2012 agreement, Boehringer Ingelheim International BmGH (BI) will establish manufacturing processes and produce drug product for Xencor's Anti-TNF antibody development program and receives first right to negotiate to manufacture and supply commercial product. Under the terms of the agreement, BI will extend Xencor credit for services performed and costs incurred at an annual interest rate in the low double-digits. BI will extend the deferment until 1) the effective date of a license agreement for the compound 2) completion of the clinical summary report for a Phase I trial or 3) February 10, 2017. Xencor is absolved of all payments if it is not able to continue development for technical or scientific reasons or decides not to proceed with further development within an agreed upon period after the Phase I trial.

Cook Pharmica – Manufacturing XmAb7195

Under a 2012 agreement, Cook Pharmica will produce and supply drug substance for Xencor's XmAb7195 program. Under the terms of the five year agreement, Xencor will pay for services performed and drug product as well as costs incurred by Cook Pharmica during development on a cost plus basis.

Catalent – Manufacturing XmAb5871

In 2011, Xencor bought a GPEx-derived cell line from Catalent used to produce XmAb5871 drug product under a previous agreement. Under the amended agreement, Xencor owes less than 1% royalties on net sales of XmAb5871 and milestones totaling $2.0 million.

Intellectual Property

Xencor's technology platform is covered by 20 issues and 44 pending US patents as well as 53 issued and 62 pending foreign patents including patents covering each XmAb Fc domain. Xencor's main clinical candidates are covered by US patent terms as follows:

XmAb5871

- Fv composition patent expiry 2027
- Fc composition patent expiry 2027
- Method of use expiry 2028

XmAb7195

- Fv composition patent expiry 2030
- Fc composition patent expiry 2027

XmAb5574

- Fv composition patent expiry 2027
- Fc composition patent expiry 2025

Management

Bassil Dahiyat, PhD – President & CEO

Dr. Dahiyat co-founded Xencor in 1997 to develop and commercialize his and Dr. Stephen Mayo's work at Caltech on proteins and Protein Design Automation technology. Under his leadership Xencor has developed a pipeline of proprietary antibody and antibody drug conjugate therapies in clinical and pre-clinical development. Dr. Dahiyat leads Xencor's scientific programs and is the inventor on 60 patents and patent applications as well as a co-author on 18 published scientific papers. He received a PhD in chemistry from CalTech and BS and MSE degrees in biomedical engineering from Johns Hopkins University.

Edgardo Bracchini, PhD – Chief Business Officer

Dr. Bracchini joined Xencor as Chief Business Officer in January 2010. Previously, he served as Senior Vice President of Business Development at Metabasis Therapeutics until its merger in 2009. Dr. Baracchini was the vice president of business development at Eltra Pharmaceuticals and director of business development at Agouron Pharmaceuticals. Prior to that, he was Assistant Director of Business Development at Isis Pharmaceuticals. Dr Bracchini received a PhD in molecular and cell biology from the University of Texas at Dallas and conducted his postdoctoral research at UCSD and The Scripps Research Institute. He received an MBA from the University of California, Irvine and a BS in microbiology from the University of Notre Dame.

John Desjarlais, PhD – Vice President, Research

Dr. Desjarlais joined Xencor more than 5 years ago and oversees all aspects of discovery and research. Previously he was an Assistant Professor of Chemistry at Penn State University, where he worked on protein engineering methods. Dr. Desjarlais receives a PhD in Biophysics form Johns Hopkins University and a BS in Physics from the University of Massachusetts.

Paul Foster, MD – Chief Medical Officer

Dr. Foster joined Xencor as Chief Medical Officer in January 2010. Previously he served as Chief Medical Officer of Development and Strategic Consulting Associates, LLC where he provided medical and clinical consulting services. Prior to that he served as Chief Medical Officer at Cardium Therapeutics and senior leadership positions at Biogen Idec, IDEC Pharmaceuticals, Abbott laboratories, Alpha Therapeutics, Reata Pharmaceuticals and Dade Behring. Dr. Foster received his MD from the Duke University School of Medicine and a BS in Chemistry from the University of Michigan.

John Kuch – Vice President, Finance

Mr Kuch has primary responsibility for financial reporting, budgeting and investment management at Xencor. He has over 15 years of experience in public accounting, most recently as a Director at Price Waterhouse. Mr. Kuch is a CPA and received his BS and MS in Accounting from the University of Illinois.

Financial Model

WEDBUSH
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Christopher N. Marai Ph.D.

12/30/2013

Xencor Inc.

Annual Financial Results & Projections
($ in thousands except per share data)
Ticker: XNCR (Nasdaq)

	FY:12A	Q4:13	FY:13E	Q1:14	Q1:14	Q3:14	Q4:14	FY:14E	FY:15E	FY:16E	FY:17E	FY:18E	FY:19E	FY:20E
Revenue:														
XmAB7195 Sales	0	0	0	0	0	0	0	0	0	0	0	0	0	85,738
Royalty Revenues	0	0	0	0	0	0	0	0	0	0	0	0	15,000	20,000
Collaboration revenue*	9,524	2,800	11,228	2,500	2,500	2,500	2,500	10,000	10,000	10,000	10,000	10,000	10,000	10,000
Total Revenues	**$9,524**	$2,800	**$11,228**	$2,500	$2,500	$2,500	$2,500	**$10,000**	**$10,000**	**$10,000**	**$10,000**	**$10,000**	**$25,000**	**$120,738**
Cost and Expenses:														
Costs of goods sold	0	0	0	0	0	0	0	0	0	0	0	0	0	12,861
Research and Development	12,668	4,000	16,857	4,000	4,500	4,500	5,000	18,000	22,000	23,000	27,000	29,000	28,000	30,000
Sales, General and Administrative	3,086	1,600	3,981	1,600	1,700	1,900	2,100	7,300	9,200	10,000	12,000	12,000	12,000	14,000
Other	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Total Costs and Expenses	**$15,754**	$5,600	**$20,838**	$5,600	$6,200	$6,400	$7,100	**$25,300**	**$31,200**	**$33,000**	**$39,000**	**$41,000**	**$40,000**	**$56,861**
Operating Income (loss)	(6,230)	(2,800)	(9,610)	(3,100)	(3,700)	(3,900)	(4,600)	(15,300)	(21,200)	(23,000)	(29,000)	(31,000)	(15,000)	0
Net Interest Income (Expense)	(2,450)	0	(1,205)	238	222	209	195	863	749	919	1,113	1,260	961	838
Other income / (Expense)	86	0	(48,541)	0	0	0	0	0	0	0	0	0	0	0
Income Before Income Taxes	(8,594)	(2,800)	(59,356)	(2,862)	(3,478)	(3,691)	(4,405)	(14,437)	(20,451)	(22,081)	(27,887)	(29,740)	(14,039)	838
Net Income	**($8,594)**	($2,800)	**($59,356)**	($2,862)	($3,478)	($3,691)	($4,405)	**($14,437)**	**($20,451)**	**($22,081)**	**($27,887)**	**($29,740)**	**($14,039)**	**$838**
GAAP Net Income	**($8,594)**	($2,800)	**($59,356)**	($2,862)	($3,478)	($3,691)	($4,405)	**($14,437)**	**($20,451)**	**($22,081)**	**($27,887)**	**($29,740)**	**($14,039)**	**$838**
GAAP Basic EPS with sFAS123	-	(0.13)	**(2.75)**	(0.09)	(0.11)	(0.12)	(0.14)	**(0.46)**	**(0.62)**	**(0.64)**	**(0.75)**	**(0.78)**	**(0.37)**	**0.02**
GAAP Diluted EPS with sFAS123	-	(0.13)	**(2.75)**	(0.09)	(0.11)	(0.12)	(0.14)	**(0.46)**	**(0.62)**	**(0.64)**	**(0.75)**	**(0.78)**	**(0.37)**	**0.02**
Weighted shares outstanding	72	21,591	21,591	31,357	31,382	31,407	31,432	31,357	32,995	34,595	37,320	38,295	38,395	38,495
Fully diluted shares outstanding	72	21,591	21,591	31,357	31,382	31,407	31,432	31,395	32,995	34,595	37,320	38,295	38,395	38,495
Cash Burn	(6,230)	(2,800)	(59,356)	(2,862)	(3,478)	(3,691)	(4,405)	(14,437)	(20,451)	(22,081)	(27,887)	(29,740)	(14,039)	-
Cash Balance	2,312	79,221	79,221	74,084	69,606	64,915	59,509	59,509	84,481	62,401	115,889	86,149	71,560	69,133

*Milestone payments not modeled

Source:Wedbush Securities and PacGrow Life Sciences

Covered Companies Mentioned Table

Company Name	Ticker	Price Target	Rating	Current Price
Macrogenics	MGNX	$70	OUTPERFORM	$28.14
Pharmacyclics	PCYC	$165	OUTPERFORM	$105.68

Analyst Biography

Chris Marai is an Analyst covering the Biotechnology/Biopharmaceuticals/BioDefense sector. Prior to Wedbush PacGrow Life Sciences, Dr. Marai was at Morgan Stanley where he specialized in quantitative modeling; he has also consulted for structure-based drug design companies and biotechnology startups.
Dr. Marai holds a B.S. in Chemistry from Trinity College, University of Toronto and a Ph.D. in Biochemistry and Structural Biology from Stony Brook University, New York.
Christopher's Edge: Dr. Marai has covered a wide range of therapeutic technologies and disease areas including novel antibody and antibody drug conjugates, reformulated and novel delivery of therapies for oncology, auto-inflammatory disease, rare diseases and CNS disorders.

Analyst Certification
I, Christopher N. Marai, Ph.D., Gregory R. Wade, Ph.D., David M. Nierengarten, Ph.D., certify that the views expressed in this report accurately reflect my personal opinion and that I have not and will not, directly or indirectly, receive compensation or other payments in connection with my specific recommendations or views contained in this report.

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Investment Rating System:
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Outperform:55% Neutral: 41% Underperform: 4%	Outperform:14% Neutral: 2% Underperform: 0%

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Wedbush Equity Research Disclosures as of December 31, 2013

Company	Disclosure
Xencor	1,3,5,7
MacroGenics	1,3,4,5,7
Pharmacyclics	1

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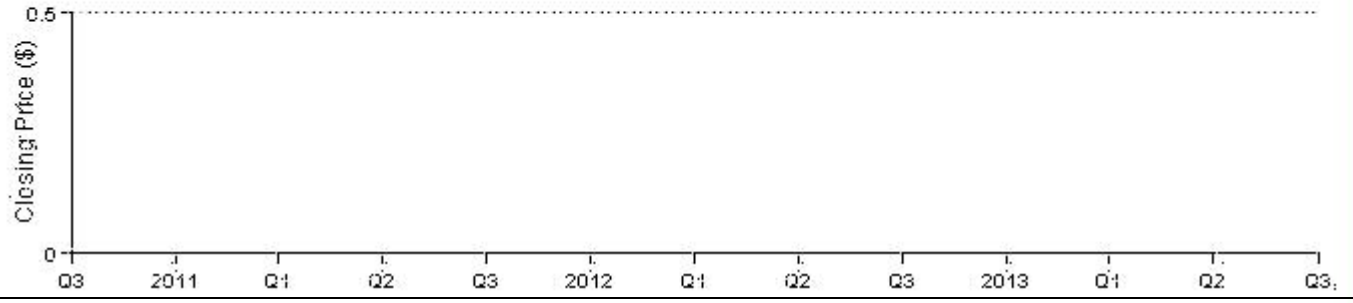
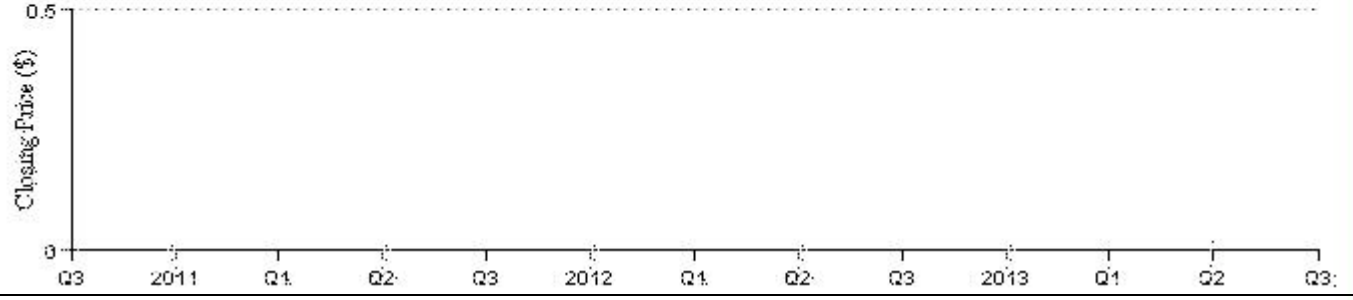
XNCR



MGNX



PCYC

1) 06/13/11 OUTPERFORM $17	2) 12/13/11 OUTPERFORM $25	3) 03/20/12 OUTPERFORM $40	4) 06/06/12 OUTPERFORM $55	5) 09/07/12 OUTPERFORM $93	6) 12/11/12 OUTPERFORM $110
7) 03/19/13 OUTPERFORM $165					

* WS changed its rating system from (Strong Buy/Buy/Hold/Sell) to (Outperform/ Neutral/Underperform) on July 14, 2009.
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CORPORATE HEADQUARTERS (213) 688-8000

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EQUITY RESEARCH DEPARTMENT

(213) 688-4529

DIRECTOR OF RESEARCH
Mark D. Benson (213) 688-4435

MANAGER, RESEARCH OPERATIONS
Ellen Kang (213) 688-4529

RETAIL AND CONSUMER

Consumer Products
Rommel T. Dionisio (212) 938-9934
Kurt M. Frederick, CFA CPA (415) 274-6822

Footwear, Apparel and Accessories
Corinna Freedman (212) 668-9876
Alicia Reese (212) 938-9927

Healthy Lifestyles
Kurt M. Frederick, CFA CPA (415) 274-6822

Restaurants
Nick Setyan (213) 688-4519
Colin Radke (213) 688-6624

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Joan L. Storms, CFA (213) 688-4537
John Garrett, CFA (213) 688-4523

Seth Basham, CFA (212) 938-9954

RETAIL/CONSUMER MARKET RESEARCH

Gabriella Santaniello (213) 688-4557

INDUSTRIAL GROWTH TECHNOLOGY

Clean Technology
Craig Irwin (212) 938-9926
Min Xu (212) 938-9925

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Al Kaschalk (213) 688-4539
Taryn Kuida (213) 688-4505

Water and Renewable Energy Solutions
David Rose, CFA (213) 688-4319
James Kim (213) 688-4380

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Shyam Patil, CFA (213) 688-8062

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Rohit Chopra (212) 668-9871
Sanjit Singh (212) 938-9922
Ryan Flanagan (212) 938-9942

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Aaron Turner (213) 688-4429

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Steve Koenig (415) 274-6801
Kevin Ikeda (213) 688-4423

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Michael Pachter (213) 688-4474
Nick McKay (213) 688-4343
Nick Citrin (213) 688-4495

Entertainment: Software
Michael Pachter (213) 688-4474
Nick McKay (213) 688-4343
Nick Citrin (213) 688-4495

Internet: Media and Gaming
Michael Pachter (213) 688-4474
Nick McKay (213) 688-4343
Nick Citrin (213) 688-4495

Internet: Social Media, Advertising & Technology
Shyam Patil, CFA (213) 688-8062

Media
James Dix, CFA (213) 688-4315

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Michael Pachter (213) 688-4474
Nick McKay (213) 688-4343
Nick Citrin (213) 688-4495

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Betsy Van Hees (415) 274-6869
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Gregory R. Wade, Ph.D. (415) 274-6863
David M. Nierengarten, Ph.D. (415) 274-6862
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CORPORATE HEADQUARTERS
1000 Wilshire Blvd., Los Angeles, CA 90017-2465
Tel: (213) 688-8000 www.wedbush.com