July 10, 2014

HEALTHCARE/BIOTECHNOLOGY

Stock Rating:

OUTPERFORM

12-18 mo. Price Target	$22.00
XNCR - NASDAQ	$10.33
3-5 Yr. EPS Gr. Rate	NA
52-Wk Range	$14.41-$5.75
Shares Outstanding	31.4M
Float	24.2M
Market Capitalization	$324.0M
Avg. Daily Trading Volume	79,788
Dividend/Div Yield	$0.00/0.00%
Book Value	$2.89
Fiscal Year Ends	Dec
2014E ROE	NA
LT Debt	$0.0M
Preferred	NA
Common Equity	$70M
Convertible Available	No

EPS	Q1	Q2	Q3	Q4	Year	Mult.
2013A	(63.78)	(3.88)	(57.87)	(0.37)	(3.85)	NM
2014E	(0.12)A	(0.15)	(0.16)	(0.18)	(0.61)	NM
2015E	(0.17)	(0.17)	(0.02)	(0.16)	(0.66)	NM

Revenue ($/mil)	Q1	Q2	Q3	Q4	Year	Mult.
2013A	1.3	3.9	3.2	1.7	10.2	25.4x
2014E	2.2A	2.2	2.2	2.2	8.7	29.8x
2015E	2.2	2.2	2.2	2.2	8.7	29.8x

Christopher Marai, Ph.D.
415 399-5721
Christopher.Marai@opco.com

Xencor, Inc.

Initiating Coverage with an Outperform Rating and $22 Price Target

SUMMARY

We are initiating coverage of Xencor, Inc, (XNCR) with an Outperform rating and a $22 price target. Xencor's proprietary Fc-engineered antibody technologies enable the creation of potentially best-in-class therapeutics and long-half-life bispecifics. We expect Xencor's first bispecific antibodies to substantially drive the value of the company, following a year-end 2014 update and as they enter the clinic in 2015. Lead asset XmAb7195, an anti-IgE mAb (Xolair biosuperior) for the treatment of severe asthma, is in our opinion significantly de-risked for safety and efficacy ahead of an anticipated year-end 2014 Phase I readout.

KEY POINTS

- The XmAb7195 program is significantly de-risked ahead of year-end data. We note that though just entering the clinic, '7195, XNCR's Xolair biosuperior, is likely to be safe and to have anti-IgE activity given that the component Fc-domain is currently in Phase II trials with '5871 and the target of the Fv-domain is validated by Xolair.
- Xencor's T-cell targeting bispecific antibodies may offer a paradigm-shifting approach to immunotherapy for cancer. Set to enter the clinic in 2015, XNCR's candidates address validated targets, CD38 for multiple myeloma and CD123 for AML, and appear potent and long-lived, and can be produced at high yields (for low COGS) vs. other technologies.
- XmAb5871 inhibits B-cell activation but does not deplete B-cells like Rituxan, making it a potential blockbuster therapy for auto-immune disorders. XmAb5871, a CD19-targeted immune inhibitory antibody, uniquely employs Xencor's technology to co-engage FcγRIIb, inhibiting BCR signaling. Data from an ongoing Phase Ib/IIa trial in patients with RA is expected by year-end 2014.
- Potential milestone payments of $1.3 billion plus additional royalties from collaborations validate the novelty, breadth and value of Xencor's Fc-engineering tool kit.
- Our $22 PT is based on a sum-the-parts approach based on multiples on sales and royalties from XNCR's proprietary and partnered products discounted annually.

Stock Price Performance

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 need.

Table of Contents

See our initiations of MacroGenics (Outperform) and Prothera (Outperform), also released today.

Investment Thesis

We are initiating coverage of Xencor, Inc. (XNCR) with an Outperform rating and a 12 to 18-month price target of $22. Xencor is a biopharmaceutical company focused on developing and commercializing engineered antibody therapies to treat severe diseases with unmet medical need. Xencor's engineered Fc domains enable high-yield production of IgG-like bispecific antibodies that may enable cost-effective immunotherapy for the treatment of cancer. This potentially best-in-class platform, which has two partnered products in Phase II studies, a wholly owned candidate in a Phase I trial, five further internal early-stage programs and a total of seven collaborations is, in our opinion, the core of Xencor's current value.

We believe that Xencor's bispecific candidates could represent a significant value driver for the company as they enter the clinic in 2015. Importantly, Xencor has also created Fc variants that enable heterodimeric Fv domains for the creation of novel bispecific antibodies. Clinical experience of current bispecific antibody-like molecules such as BiTEs and TandAbs has highlighted substantial potency, though we note duration of action (due to longer half-life) may be important in attracting and stimulating a meaningful T-cell response against tumors. Nonetheless, single agent activity of T-cell engaging bispecifics is promising, though we note additional opportunity for efficacy may exist should these bispecific molecules be combined with checkpoint inhibitors and other immuno-modulating agents. Finally, we highlight that T-cell engaging bispecifics may also offer a more convenient immuno-oncology approach than CAR-T therapies which currently require autologous or allogeneic transplant.

We believe that XmAb7195, though just entering the clinic, is likely to be safe and have anti-IgE activity given that the component Fc domain module is currently in Phase II trials with XmAb5871 and the target of the Fv domain has been validated by Xolair. Xencor's lead candidate, XmAb7195, an anti-IgE antibody therapy for the treatment of allergic asthma and IgE mediated disease, employs Fc-modification to enhance FcγRIIb binding up to 400x, enhancing liver clearance of IgE, and suppresses IgE production by B-cells. Xencor's newly engineered Fc dependent functionalities, in our opinion, extend functionality beyond that of current anti-IgE therapies that sell $1.65 billion (Xolair), make XmAb7195 a best-in-class anti-IgE therapy and highlight the potential broad utility of Xencor's technology. XmAb7195 is in a Phase I trial with data, including IgE levels, anticipated in Q4:14.

XmAb5871, Xencor's Phase II candidate for autoimmune disorders optioned to Amgen, represents a novel B-cell inhibiting strategy targeting 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 IIa study of '5871 is anticipated in late 2014.

XmAb5574/MOR208, partnered with MorphoSys, is Xencor's other CD19 therapy that utilizes an Fc domain engineered for FcγRIIIa and FcγRIIa receptors to increase ADCC, potently depleting CD19-expressing B-cells. This approach could be utilized in B-cell malignancies where CD20-directed therapies fail or cannot eradicate all vestiges of disease and potentially ahead of CD20 therapies if it demonstrates a greater efficacy.

We believe that partnerships with Amgen, Alexion, MorphoSys, Janssen, Merck, Boehringer Ingelheim and CSL are validating of Xencor's approach. These partnerships also provide nondilutive capital in up-front payments and up to $1.31 billion in milestone payments as well as additional royalties (single to double-digit percentages).

Valuation

We arrive at our $22 price target by a sum of the parts analysis. We ascribe $12/share by applying a typical oncology multiple of 6x on our estimated 2022 revenues of $1.2 billion for XmAb7195 in moderate to severe asthma, discounted 45% annually. We ascribe $3/share to each of Xencor's two partnered programs XmAb5871 and XmAb5574 based on a typical multiple of 15x royalties (estimated 10%) from estimated US and EU sales in 2022 of $900M and $600 million discounted 40% and 30% annually, respectively. We ascribe $3/share to XmAbCD123 as a surrogate for the bispecific candidate Xencor expects to advance into the clinic in 2015.

Exhibit 1: Valuation Table

Sales / Royalties	Stage	Valuation Year	Sales/ Royalties	Sales Multiple	Discount Rate	Program Value	Est. Shares (000's)	Per Share
XmAb7195 in Allergic Asthma	Phase I	2022	$1,258,031	6	45%	$386,277	31,486	$12.27
CD123xCD3 in AML	Preclinical	2022	$606,726	6	55%	$109,267	31,486	$3.47
XmAb5871 Royalties in RA	Phase II	2022	$85,514	15	40%	$86,916	31,486	$2.76
MOR208 Royalties in ALL	Phase II	2022	$59,331	15	30%	$109,101	31,486	$3.47
Total						***$691,561***	***31,486***	***$22***

Source: Oppenheimer & Co. Inc.

Key Risks to Our Price Target

Clinical Risk. We would expect a material decline in XNCR shares in the event of unsuccessful clinical data for any of Xencor's candidates.

Regulatory Risk. The regulatory process to attain approval of drugs is complex, requiring collection and production of extensive sets of data from expensive and time-consuming studies. Decisions on approval are at the discretion of the respective regulatory agencies, which can be unpredictable. Finally, following approval, regulatory agencies retain the power and ability to remove these drugs from the market if deemed to present sufficient danger.

Commercialization Risk. Xencor will be competing against numerous other agents both in oncology and auto-immune markets. Physicians may be more comfortable with more established products and fail to prescribe Xencor's novel therapies. Furthermore Xencor must build and maintain a successful sales force in order to successfully commercialize the company's wholly owned candidates.

Intellectual Property Risk. There is inherent uncertainty in both the interpretation of patent claims and the application of patent law, regardless of the apparent strength of Xencor's patent portfolio. Upon expiration of patents, Xencor may be unable to prevent third parties from creating biosimilar copies of Xencor's products. Furthermore, competitors may challenge the scope/validity of the patents, or simply find ways to circumvent them.

Manufacturing Risk. Xencor does not possess its own manufacturing capabilities to supply sufficient quantities of its drugs. Any disruption or contaminant problems could result in delays to clinical studies or future commercialization until such problems are resolved. Moreover, upon commercialization, any impact on the company's supply of drug product would adversely affect revenue.

Competitive Risk. In addition to the commercialization risks discussed above, we note that other biotechnology companies with greater resources may pursue development of a competing topical product, the potential approval/commercialization of which could negatively impact Xencor's market share and revenue.

Strategic Risk. If Xencor becomes overly confident in signing a partnership or becoming acquired and does not take adequate steps to prepare for self-commercialization, investors may react negatively if a strategic deal fails to materialize.

Financing Risk. If the company raises more money than we estimate or raises at a lower valuation than we estimate, the dilutive effect of the new shares could result in a material decline in the share price of the company's securities.

Insider Ownership Risk, A single insider owns approximately 25% of Xencor's voting stock, and his family members beneficially own an additional 10.4% of voting stock. The interests of these parties may not be aligned with the interests of public shareholders.

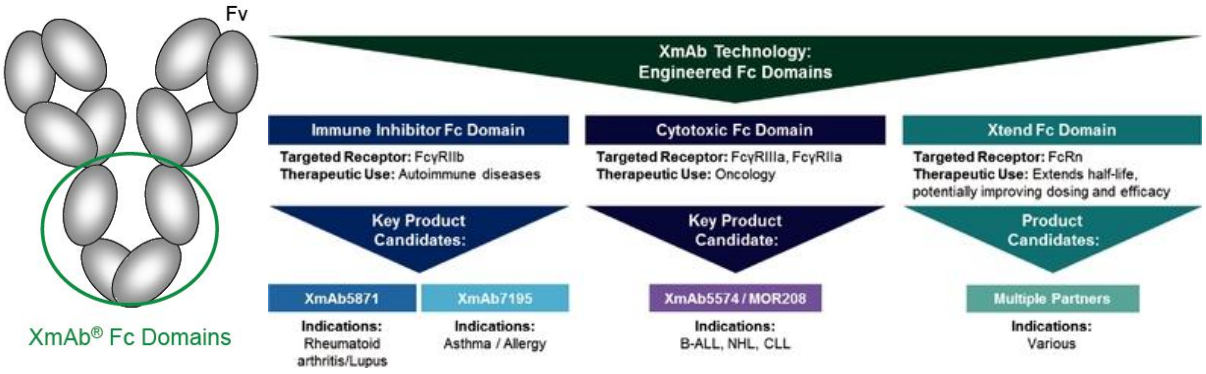
Upcoming Milestones

Mid-2014	Initiation of IND-enabling studies for a bispecific candidate (either CD38xCD3 or CD123xCD3)
Aug. 2014	Estimated first cohort completed in Phase I trial of XmAb7195 (anti-IgE) in healthy volunteers
H2:14	Top-line data from the Phase II trial of XmAb5574/MOR208 (CD19, enhanced ADCC Fc) in ALL
H2:14	Top-line data from the Phase IIa trial of XmAb5871 (CD19, immune inhibitor Fc) in rheumatoid arthritis
Dec 6-9, 2014	Potential full data from a Phase II trial of MOR208 (CD19, enhanced ADCC Fc) in ALL at ASH
Dec 6-9, 2014	Potential interim data from a Phase II trial of MOR208 (CD19, enhanced ADCC Fc) in NHL at ASH
YE:14	Top-line data from a Phase Ia trial of XmAb7195 (anti-IgE) in healthy subjects with IgE reduction data
Q1:15	Initiation of a Phase Ib trial of XmAb7195 (anti-IgE) in mild to moderate asthma with IgE reduction and clinical benefit data
H2:15	Complete recruitment in Phase II trial of MOR208 (CD19, enhanced ADCC Fc) in CLL
2015	Complete recruitment in Phase II trial of MOR208 (CD19, enhanced ADCC Fc) in NHL
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 (either CD38xCD3 or CD123xCD3)
Q1:16	Potential top-line data from the Phase Ib trial of XmAb7195 (anti-IgE) in mild to moderate asthma
2016	Initiation of a Phase IIb proof-of-concept trial of XmAb7195 (anti-IgE) in poorly controlled asthma
2017	Potential top-line data from the Phase IIb POC trial of XmAb7195 (anti-IgE) in patients with poorly controlled asthma
2017	Potential top-line data from the Phase IIb trial of XmAb5871 (CD19, immune inhibitor Fc) in patients with rheumatoid arthritis
2017	Potential exercise of Amgen's option to license XmAb5871 (CD19, immune inhibitor Fc)

Company Overview

Xencor develops next-generation antibodies and advanced protein therapeutics by rationally engineering Fc-domains to enhance or optimize functionality. Xencor has developed a set of Fc domains that augment naturally occurring antibody functions by enhancing ADCC via FcγRIIIa, inhibiting the immune system via FcγRIIb, extending the half-life via the FcRn receptor or enabling long-lived bispecific antibodies.

Exhibit 2: Xencor's Engineered Fc Domain Technology



Source: Xencor, Inc.

Xencor's pipeline consists of antibodies that are directed against well-validated targets (Fv domain) that exhibit improved function by incorporating the companies' Fc-technologies. Xencor is also developing bispecific antibodies that can engage and redirect T-cells. In addition to disclosed partnerships with MorphoSys and Amgen, Xencor has seven undisclosed partnered products, four in clinical development, with Boehringer Ingelheim, Janssen, Merck, CSL Limited and Alexion.

Exhibit 3: Xencor's PIpeline

Name	Fv Target	Fc domain / Ab Type	Status	Indication	Next Event	Partner
XmAb5871	CD19	Immune Inhibitor	Phase II initiated	RA	PIIa Data H2:14	Option to Amgen
XmAb7195	IgE	Immune Inhibitor	Phase I initiated	Allergic Asthma	Data YE:14	-
MOR208	CD19	ADCC	Phase II ongoing	ALL/NHL/CLL	B-ALL and interim NHL potentially at ASH 2014	Morphosys
XmAb13694	**CD38**	**CD3 Bispecific**	**Preclinical**	**Oncology**	**IND 2015**	**-**
CD3 x CD123	**CD123**	**CD3 Bispecific**	**Preclinical**	**Oncology**	**IND 2015**	**-**
Xtend-TNF	TNF	Xtend	Preclinical	Autoimmune	IND	-
Anti-X/CD23b	-	Immune Inhibitor	Preclinical	TBD	IND	-
Undisclosed	-	ADCC	Phase I	Oncology	-	BI
Undisclosed	-	ADCC	Phase I	Oncology	-	BI
Undisclosed	-	ADCC	Phase I	Oncology	-	CSL / Janssen
Undisclosed	-	Stability	Phase I	Autoimmune	-	Merck
Undisclosed	-	Xtend	Preclinical	Autoimmune	-	Janssen
Undisclosed	-	Xtend	Preclinical	Hematology	-	CSL
Undisclosed	-	Xtend	Preclinical	Undisclosed	-	Alexion

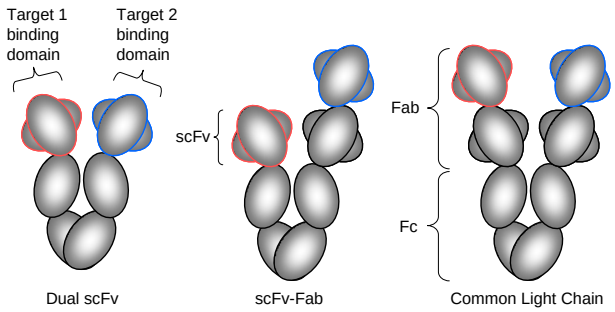
Source: Oppeheimer & Co. Inc.

T-cell Redirecting Bispecific Antibodies

Xencor is developing two bispecific antibodies in preclinical work. The first is a CD123xCD3 bispecific for acute myeloid leukemia (AML) and/or blastic plasmacytoid dendritic cell neoplasm (BPDCN), a rare cancer of dendritic cells, and the second is a CD38xCD3 bispecific for multiple myeloma (MM). The company intends to begin IND-enabling studies in 2014 and file an IND on at least one of these compounds in 2015.

We anticipate bispecific antibodies to substantially drive the value of the company as they enter the clinic. Xencor is expected to provide an update with preclinical data at the American Society of Hematology meeting (ASH 2014) (December 6-9, 2014, San Francisco).

Exhibit 4: Xencor's Bispecifics Include FC Regions Enhancing Half-life



Source: Xencor, Inc.

Xencor's Fc-Engineering Enables Better Bispecifics

Xencor's Fc-engineered antibody technologies enable the creation of potentially best-in-class, novel, long half-life bispecifics that we believe have been overlooked by the Street. Over the past 50 years, numerous bispecific antibody technologies have been developed; however, most constructs currently suffer from poor manufacturability (Removab, blinatumomab), short half-life (blinatumomab), and/or serious safety concerns. Incorporation of Xencor's Fc-domains allows for bispecifics with antibody-like half-lives (Exhibit 5) and high production yields that are potentially superior to previous generations of bi-specific technology. Importantly, experts we spoke with highlighted the need for long-lived bispecifics; emphasizing half-life over potency may be necessary to elicit maximal clinical effect.

Fc-knock-out technology, utilized in the bispecific program, allows for:

- Long duration of action (half-life) mediated by FcRn
- Removes undesirable Fc effector functions
- IgG-like industry-standard manufacturing and COGS
- Lower immunogenicity risk and humanized antibodies that cross react with nonhuman primates for preclinical safety
- Plug and play CD3 domains with existing antibodies

Exhibit 5: Xencor's Bispecifics Have IgG-like Half-life

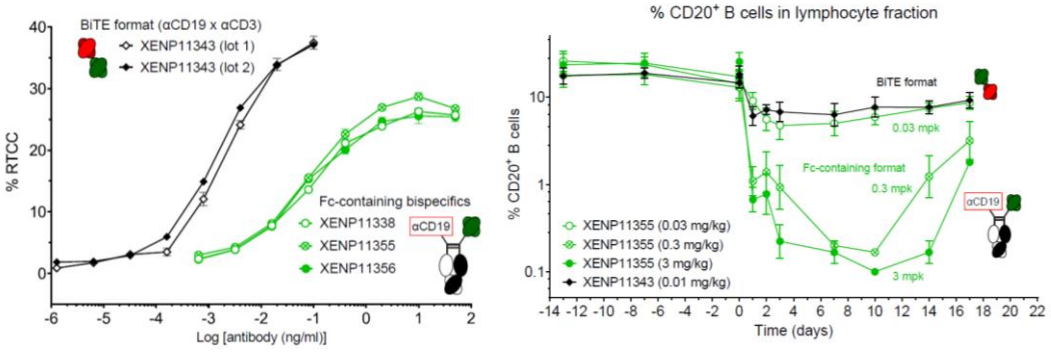
Name	Fc-Domain	Mouse	Primate	Human
Blinatumomab (CD19xCD3)	No	~1-3 hrs	~2 hr	1-1.5 hrs
XmAb (CD38xCD3)	**Yes**	**8 days**	**-**	**-**
MGD007 (GPA33xCD3)	Yes	-	6.8 days	-
MGD010 (FcγRIIbxCD79b)	Yes	-	5.9 days	-
MGD006 (CD123xCD3)	No	1-1.5 hrs in preclinical models		

Source: Oppenheimer & Co. Inc.

Xencor's CD19xCD3 Demonstrates Superior Properties vs. Earlier Constructs

Xencor has generated CD19xCD3 bispecific antibodies that have long half-lives (six days) and high potency in vivo. We note that although they are not as potent on a ng/ml basis, this may not impact efficacy at feasible doses and may allow for finer tuning of administration, potentially better facilitating combination therapies (note: the Fc-containing bispecific is roughly twice as heavy, shifting the curves slightly closer together on a molar basis) (Exhibit 6). Xencor found that CD19xCD3 bispecific antibodies induced long-lasting and potent B-cell depletion of greater duration and depth than a CD19xCD3 BiTE molecule produced by MicroMet/Amgen.

Exhibit 6: Xencor Offers a better Format for T cell-recruiting Bispecifics



Source: Xencor, Inc.

Engaging T-Cells to Retarget the Immune System

T-cells have been shown to have the ability to control tumor growth and survival in both early and late stages of the disease. Regrettably, tumor-specific T-cell responses have been difficult to mount and sustain in patients, limited by the numerous counteracting immune escape mechanisms of tumors.

Engineered antibodies that are bispecific for a surface target antigen on cancer cells (Fv domain), and for CD3 (Fv domain) on T-cells offer an alternative approach to engage T-cells for cancer therapy. Bispecific (CD3 targeted) antibodies are capable of connecting cytotoxic T-cells to a cancer cell, independent of T-cell receptor specificity, co-stimulation, or peptide antigen presentation. Through this mechanism, T-cells are retargeted to the desired tumor antigen, where it will exert its cytotoxic effect through the natural pathways of degranulation and perforin release. Bispecific antibodies can also induce serial lysis, a process in which a single antibody and a single T-cell kill multiple target cells. T-cells, localized to the tumor by bispecifics, may also activate other beneficial pathways of the immune system through controlled cytokine release.

Combinations with Immunostimulatory Therapeutics May Be Paradigm Shifting

We believe that T-cell engaging bispecific therapies, although likely to be highly efficacious monotherapy, may offer a paradigm-shifting approach and potentially significant efficacy in oncology when combined with immuno-modulatory compounds (PD1, PDL1, CTLA4, B7H3, CD27) that inhibit factors that aid a tumor's immunological escape.

Bispecific Technologies in Development

Bispecific antibodies have been developed in a variety of formats; the most advanced being blinatumomab utilizing Amgen/MicroMet's BiTE technology, currently in Phase III trials. Trion's original full-length antibody bispecific technology, a rat-mouse hybridoma, is no longer being clinically developed, and its only approved drug, Removab (ertumaxomab), never received US approval.

General Properties of Advanced Bispecific Technologies

- BiTEs (AMGN– Perform) have a limited practical utility due to extremely short half-life, a result of their relatively small size and the lack of an Fc domain, requiring continuous infusion.

- DARTs (MGNX – Outperform) consist of two dimerized chains bound by a stabilizing disulfide bonds (to enhance manufacturability). This format can be manufactured in g/L yields and may be more potent than BiTEs. DART formats incorporating an Fc domain have reached half-lives of up to seven days. Preclinical work on DART's also suggests that they may be more potent than Amgen's BiTE technology.

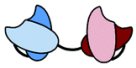
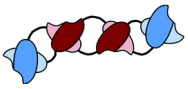
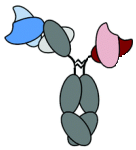
- Xencor's (XNCR – Outperform) XmAb bispecifics have an IgG-like long half-life (six to seven days) through incorporation of their KO Fc-domain and contain no peptide-linkers, facilitating straightforward manufacturing and potentially significantly lower cost of goods sold.

- Trion's Removab (ertumaxomab) mouse/rat hybrid is difficult to manufacture and immunogenic. The company discontinued a Phase II trial of an anti-HER2 bispecific citing strategic priorities.

- TandAbs are tetravalent bispecifics that have demonstrated relatively long half-life (one day) in clinical trials.

- ImmunoCore's ImmTac utilizes TCRs instead of Fab fragments and thus recognizes internal (endogenous) antigens, presented on the surface of tumor cells in conjunction with MHC class I molecules. This approach could potentially open entirely new targets to immunotherapy, previously unavailable to extracellular antibodies.

Exhibit 7: Comparison of Bispecific T-cell and NK-cell Redirecting Antibodies Approved and in Clinical Development

Tech Name	Company	Graphical Representation	Tech	Mechanism	Drug(s) (Targets)	Key References	Status
Hybridoma	Trion (Private)		Rat/mouse quadroma	T-cell redirected cytotoxicity	Removab, ertumaxomab (EpCAMxCD3, HER2xCD3)	Heiss (2010), Baumann (2011)	EU Approved
BiTE®	Amgen (AMGN)		Peptide-linked scFv fragments	T-cell redirected cytotoxicity	Blinatumomab, MEDI-565, AMG 212 (CD19xCD3, CEAxCD3, PSMAxCD3)	Topp (2012)	Phase III
ImmTAC	Immunocore (Private)		scFv fragment linked to soluble affinity enhanced TCR	T-cell redirected cytotoxicity with TCR	IMCgp100 (Gp100xCD3)	Liddy (2011), Bossi (2014), corporate info	Phase IIa
DART	MacroGenics		Recombinant dual-chain Fv fragments	T-cell redirected cytotoxicity	MGD006 (CD123xCD3)	Moore (2008)	Phase I
TandAb®	Affimed (Private)		Tetravalent peptide-linked scFv fragments	NK-cell redirected cytotoxicity	AFM13 (CD16axCD30)	Rajkovic (2012)	Phase I
XmAb®	**Xencor (XNCR)**		**One normal V_H/V_L pair, one scFv fused to Fc**	**T-cell redirected cytotoxicity**	**XmAb13694 (CD38xCD3)**	-	**Preclinical**

Source: Oppenheimer & Co. Inc.

Directing T-Cells: CAR-Ts versus Bispecifics

T-cells redirected to specific antigen targets are emerging as powerful therapies in oncology. T-cell/CD3-directed bispecific antibodies function by binding the CD3 receptor on T-cells, redirecting the T-cell to the desired antigen. However, another approach to targeting cancer via T-cells utilizes chimeric antigen receptors (CARs) transduced into T-cells. The chimeric antigen receptor consists of an scFv fragment, a trans-membrane domain and the cytoplasmic domain of the TCR which is introduced to the T-cell by viral transduction. This facilitates targeting of T-cells to a desired antigen, similar to the way a bispecific antibody retargets a patient's T-cells to a desired antigen.

We note, however, that bispecific antibodies are easier to manufacture, store and administer than cell-based products. Large, complex, cell-based CAR-T cells are also potentially more immunogenic than lower-surface area bispecific antibodies.

CAR-T therapies have also demonstrated toxicities in clinical trials, including infusion reactions, cytokine release syndrome (CRS), macrophage activation syndrome (MAS) and CNS toxicities. However, we note that bispecific antibodies are not without their own safety and immunogenicity risks. Blinatumomab, for instance, is so potent that doses are limited to µg/day and is still associated with febrile neutropenia.

Exhibit 8: CD19 Directed Bispecific and CAR-T Therapy Comparison

Drug	Blinatumomab	Memorial Sloan Kettering CAR-T	Memorial Sloan Kettering CAR-T	Bethesda CAR-T
Phase	Phase II	Phase I	Phase I	Phase I
Type	Bispecific	Autologous CAR-T	Autologous CAR-T	Autologous CAR-T
Indication	Ph-negative R/R ALL	CLL pts in CR or PR with residual disease	CLL pts relapsed on purine-analog therapy	B-lymphoma or CLL
N	189	7	8	8
Dose / Design	Open label, single-arm, continuous IV 4 wks on/2 wks off for 5 cycles (cycle 1 only: 9 µg/d days 1-7; then 28 µg/d)	3 dose cohorts of 3x106 - 3x107 CAR+ T cells/kg	Open-label, 3+3 design, subsequently modified	Open-label, single arm
Primary Endpoint	CR or CR with (CRh) response rate	Toxicity, MTD	Toxicity, MTD	Toxicity, MTD
Efficacy	At interim, 43% achieved CR/CRh, 34% of pts that had ≥2 prior therapies achieved CR/CRh	1 CR, 2 bone marrow CR, 3 PR, 1 PD	2 Sds, both in the 4 patient cohort that was treated with cyclophosphamide	1 CR, 5 SD, 1 SD, 1 not evaluable
Safety	Most frequent Grade ≥3 AEs were febrile neutropenia (26%), anemia (15%) and neutropenia (15%); 2% had G ≥3 cytokine release syndrome	No DLT was observed. Mild and self-limiting cytokine release syndrome (CRS) was observed in 3 pts	Patients had fever, chills within 24 hours, 1 death (pt in cylposphamide cohort) within 48 hours of infusion due to sepsis-like syndrome, potentially related to pre-infusion infection, cytokine elevations not seen in cyclophosphamide treated patient	"significant toxicities" including B-cell depletion (as expected), hypotension, fevers, fatigue, renal failure, and obtundation
Author	Topp (2014)	Park (2014)	Brentjens (2011)	Kochenderfer (2011)
NCT	NCT01466179	NCT01416974	NCT00924326	NCT00924326

Source: Oppenheimer & Co. Inc.

XmAb-CD123: Targeting T-Cells to CD123 for AML and Hematological Malignancies

Xencor is developing a CD123xCD3 bispecific, but has not yet presented preclinical data for this asset. CD123, or Interluekin-3 receptor, is a transmembrane heterodimer composed of an IL3 specific α subunit and a β_C subunit shared with the GM-CSF receptor and the IL-5 receptor. CD123 expression marks certain subsets of hematopoietic stem cells (HPCs) including myeloid (except erythroid) progenitors. CD123 is expressed at high levels on dendritic cells and on mature, but not immature, B-cells. The target is clinically validated in acute myeloid leukemia (AML) and other hematological malignancies. CD123 is overexpressed in hairy cell leukemia (HCL), AML, B-ALL, blastic plasmacytoid dendritic cell neoplasm (BPDCN), mastocytosis (Teodosio 2010, 2013) and other cancers (Exhibit 9).

Exhibit 9: CD123 Expression on Rare Cancers

Indication	Reference
Mastocytosis	Teodosio 2010, 2013
Hypereosinophilic syndrome	Brooks 2013
Hairy cell leukemia	Shao 2013
Chronic myelomonocytic leukemia	Orazi 2006
Basophilic Leukemia	Yokohama 2002
B Cell-Acute Lymphoblastic Leukemia	Lhermitte 2006
T Cell-Acute Lymphoblastic Leukemia	De Smet 2012

Source: Oppenheimer & Co. Inc.

We believe that strong clinical data for XmAb13694 (Xencor's CD38xCD3) will de-risk Xencor's CD123xCD3 clinical program given clinical validation of the target. Recall that CD123 is highly validated with other CD123 antibodies and bispecifics reporting promising clinical and preclinical results to date. Consequently, in our opinion, much of the remaining risk rests with Xencor's bispecific technology and KO Fc engineering approach. Furthermore, although competition exists among bispecifics targeted to CD123, data for Xencor's bispecific half-lives (anticipated to be much longer duration than others) could separate its molecule in both efficacy and market opportunity.

CD123 (IL-3R) Is a Clinically Validated Target for AML and Hematologic Cancers

We note that current therapies targeting IL-3R for leukemic stem cells in patients with AML have provided strong clinical validation this target. Recent data for Stemline Therapeutics' (STML, Not Covered) SL-401, a protein therapeutic consisting of the first 388 amino acids of diphtheria toxin conjugated to human IL3, displayed surprising results in BPDCN, AML, MSD among others (Exhibit 10).

We highlight that BPDCN, an extremely rare cancer categorized under AML by the World Health Organization, may represent a unique sub-set population for CD123xCD3 from a regulatory standpoint. Additionally, since the vast majority, 77- 90%, of cases present with cutaneous lesions, similar to a cutaneous lymphoma (Fachetti 2009), BPDCN may offer quick proof-of-concept and clinical indicators of improvement. The disease is also associated with a poor prognosis and a median survival of less than 18 months, making gold-standard OS studies potentially possible. However, we caution that recruitment for such a study could be slow. We estimate that roughly 150 patients (and up to 1,000) are diagnosed with BPDCN in the US each year, based on a 7.7/1,000,000 person-years incidence rate for cutaneous lymphomas (Bradford 2009) and a small (30-case) sample indicating that BPDCN makes up ~0.7% of cutaneous lymphomas (Petrella 2005).

Targeting CD123: Clinical Efficacy has Been Observed without Redirecting T-Cells

We note that there are at least seven CD123 directed therapies are in development; however, those currently in the clinic do not actively engage T-cells like XmAbCD123. The most advanced clinical candidate is Stemline'sSL-401, an immunotoxin consisting of a modified diphtheria toxin fused to the IL3 protein (Exhibit 10). SL-401 recently completed a Phase I trial demonstrating it is safe and well tolerated and highly active in BPDCN, a rare hematologic cancer (Frankel 2013). CSL has also been testing, Fc-enhanced and chimeric mAbs against CD123, though we anticipate these approaches to be less potent than a T-cell redirecting bispecific (Exhibit 10).

Exhibit 10: CD123 Directed Therapies in Clinical Development

Company	MacroGenics	CSL Limited	CSL Limited	Stemline (STML)
Name	MGD006	CSL362	CSL360	SL-401
Type	Bispecific	Fc-enhanced mAb	Chimeric mAb	mAb-diptheria toxin
Status	Phase I initiated June 2014	Phase I initiated Aug. 2012	Phase I complete 2012, not continued	Phase I/II
Indication	R/R AML	CD123+ AML	R/R or high-risk AML	Advanced hematologic malignancies: 59 R/R AML , 11 AML unfit for chemo, 7 MDS (refractory, high risk), 9 BPDCN
N	ND	Est. 36	40	81
Dose / Schedule	ND	IV every 14 days for 6 cycles, up to 12 mg/kg	IV infusions 1x/wk for 12 weeks, from 0.1-10mg/kg	IV daily for 5 days, doses from 4-22µg/kg/day, single cycle
MTD	ND	ND	MTD not reached	16.6 µg/kg/day, DLTs of hypoalbuminemia and edema
AEs	ND	ND	5/62 (8%) SAEs related or possibly related to treatment	Grade ≥ 3 adverse events included transient transaminase elevations (20%) and vascular leak (4%). Absolute neutrophil count and hemoglobin remained stable.
Response	ND	ND	1 CR, no other definitive clinical benefit	in 59 patients with R/R AML, 2 CRs and 25% of pts had tumor shrinkage: In 9 evaluable BPDCN patients, 5 CRs (56%) and 2 PRs (ORR: 78%), median CR duration >5 months
Other	ND	In Dec. 2013, Janssen signed a partnership agreement with CSL Limited, presumably on the basis of unpublished interim results	Immunogenicity detected in lower dose cohorts only	3/6 BPDCN patients developed ADA's, at day 15 (ADA's take at least 2 wks to develop, continue increasing for at least a further 2 wks)
NCT	NCT02152956	NCT01632852	NCT00401739	NCT02113982

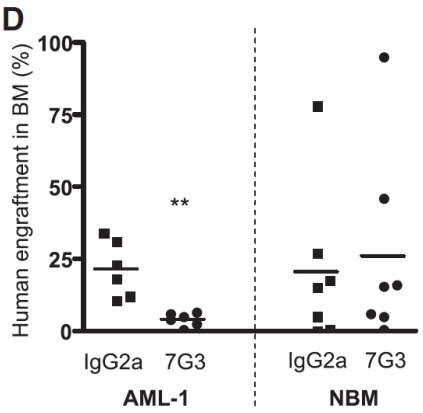
Source: Oppenheimer & Co. Inc.

CD123 Is Present on Cancer Stem-like Cells, and Inhibition Inhibits Engraftment

IL3 is a cytokine that engages CD123 (IL3R) to stimulate proliferation and maturation of a wide range of hematopoietic stem cells. CD123 (IL3R) is expressed on HPCs, and is hypothesized to be a marker of a highly regenerative set of malignant cells, termed cancer stem-like cells. CD123 overexpression marks a subset of malignant AML cells with greater regeneration and engraftment capability than $CD123^-$ cells, and is a marker of poor prognosis in AML.

Importantly, killing CD123-positive cells with an ADCC-inducing anti-CD123 antibody (7G3) inhibits the engraftment of AML cells, but not normal bone marrow (NBM) cells in SCID mice. This further indicates the specificity of CD123 for malignant cells. Incubation of NBM cells with 7G3 prior to engraftment reduced engraftment efficiency by an average of 70% (range 35%-140%), compared to an average of 18% (range 1% - 97%) in malignant AML cells (Jin 2009).

Exhibit 11: Anti-CD123 Antibody 7G3 Inhibits AML Engraftment but not NBM engraftment in SCID mice



Source: Jin et al. Cell Stem Cell. 2009 Jul 2;5(1):31-42

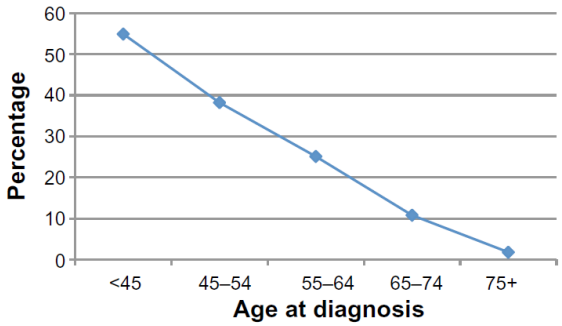
XmAbCD123 Potential Market in AML

XmAbCD123, if demonstrated to have significant activity in AML may find utility in in a broad range of oncology settings where CD123 is expressed. Although potentially broadly applicable, we choose to assign value to XmAbCD123 in reference to a potential market in AML, noting upside may exist, should efficacy in other disease be replicated.

AML is a heterogenous group of myeloid neoplasms predominantly affecting the elderly (median age at diagnosis: 67 years). Five-year overall survival (OS) from diagnosis is ~25% today, but this is predominantly due to the high survival rate in younger patients (Exhibit 12), in part due to the use of ASCT, which is not performed in elderly patients.

SEER-Medicare data indicate that in the period 1999-2002, just 36% of AML patients aged >65 years received any chemotherapy, and median survival in this group is just 1.7 months. Recent evidence suggests that elderly patients can benefit from low-dose chemotherapy. While this evidence may have increased the rate of chemotherapy use in elderly patients, there remains significant unmet medical need for a well-tolerated therapy in this fragile patient population. Approximately 14,500 patients are diagnosed with AML in the US each year, ~1/2 over the age of 65, or ~7,000 elderly patients.

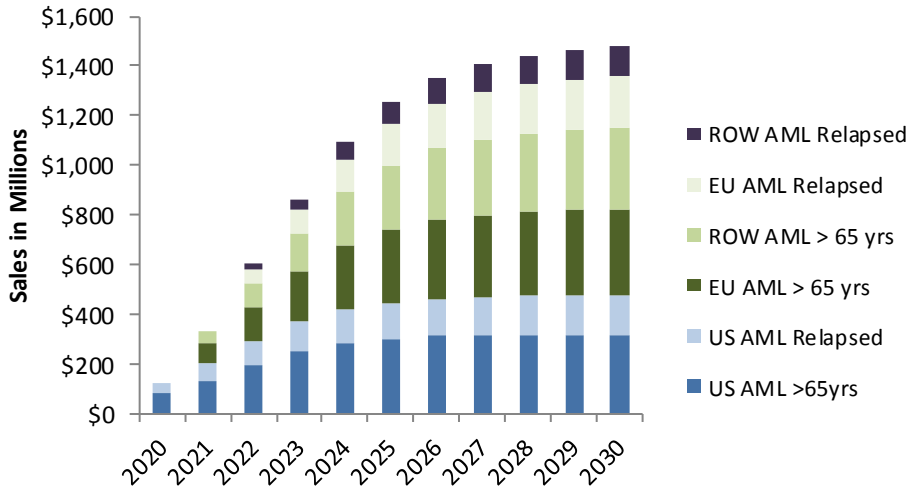
Exhibit 12: Five-Year Survival by Age of Diagnosis in AML



Source: Lim et al. Clin Interv Aging. 2014 May 6;9:753-762

We estimate that if XmAbCD123 received approval for AML patients unfit current treatments (high dose chemotherapy or ASCT), or relapsed on front-line therapy, it could generate peak sales in excess of $1.3B. We estimate that that XmAbCD123 may achieve 60% penetration in the estimated 7,000 patients diagnosed with AML each year that are unfit for intensive therapy. We further note that these patients if unfit for intensive therapies may also be unfit for CAR-T therapies. We estimate that XmAbCD123 may achieve 60% penetration in the 4,500 relapsing-remitting (R/R) patients available each year. We estimate XmAbCD123 could be priced at $10,000 per cycle, on par with recently approved therapies in hematological malignancies, and be used for an average of 10 cycles in unfit patients and 6 cycles in R/R patients. We estimate that XmAbCD123 could receive approval in 2020. We ascribe $3/share to XmAbCD123 by applying a 6x multiple to estimated 2022 sales, discounted 55% annually.

Exhibit 13: XmAbCD123 Sales Estimates in Unfit and Relapsed AML Patients



Source: Oppenheimer & Co. Inc.

XmAb13694—CD38 x CD3

XmAb13694 is a CD38xCD3 bispecific antibody utilizing XNCR's Fc knockout (KO) domain technology and has no immunologic effector functions to mitigate the risk of T-cell cross-linking and cytokine storm. XmAb13694 has a half-life of about eight days in murine models (Exhibit 5, p. 8), significantly longer than other bispecifics for the target, and is highly potent with half-max redirected T cell cytotoxicity of ~0.1 ng/ml. We anticipate that Xencor will bring XmAb13694 into the clinic in 2015.

XmAb13694 redirects T-cells through the CD3 receptor to CD38-expressing plasma cells. CD38 is a small Type II transmembrane glycoprotein widely represented on lymphoid and myeloid lineages but absent from most mature resting lymphocytes with the notable exception of terminally differentiated plasma cells. In many lymphoid tumors, including most cases of myeloma, it is present on the cell surface at levels which render it an attractive target for therapeutic antibody.

CD38 Is a Clinically Validated Target in Multiple Myeloma

CD38 represents a validated target for the treatment of multiple myeloma as several anti-CD38 antibodies are in late-stage clinical trials, including daratumumab in Phase III. Notably, daratumumab demonstrated statistically significant increases in progression free survival (PFS) in a Phase I/II trial in relapsed/refractory MM. CD38 is one of the few antigens expressed on the plasma cells deregulated in MM. Additionally, checks we conducted suggest that anti-CD38 compounds are among the most exciting approaches for novel combination therapy in multiple myeloma. We believe that strong results for XmAb13694 in the clinic could make the asset or Xencor an attractive acquisition candidate.

There are currently three CD38 targeting antibodies in clinical trials, daratumumab (Phase III, Genmab), SAR650984 (Phase I, ImmunoGen/Sanofi) and MOR03087 (Phase I, Morphosys/Celgene). Janssen initiated a Phase III trial of daratumumab in March 2013 on the basis of interim, unreleased, data from ongoing Phase II trials.

Exhibit 14: CD38-Directed Antibodies in Clinical Development

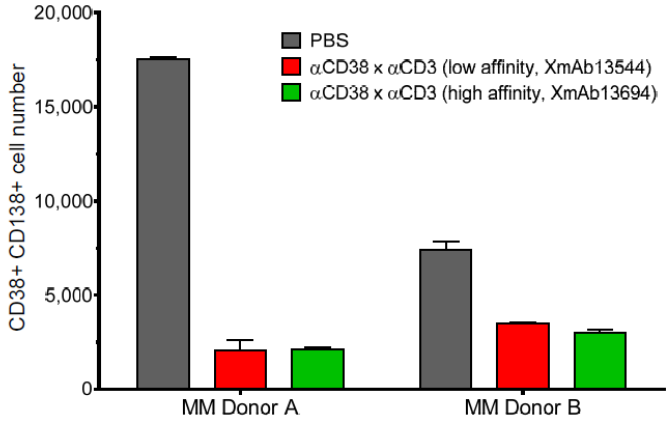
Drug	Daratumumab	SAR650984	MOR03087
N	32 (Dose escalation) + 16 (dose expansion)	24	82
Status	Phase I/II	Phase I	Phase I/II
Indication	MM R/R to 2+ lines	CD38+ MM or other	MM R/R to 2+ lines
Dose / Schedule	0.005 -> 24 mg/kg weekly iv infusion	up to 20 mg/kg	nd
Prior lines of therapy	5.5 (3-12)	nd	nd
Response	In 12 pts ≥ 4 mg/kg, 5 PRs (42%), 3 MRs (25%)	Among 22 evaluable pts, 2 CR (10%), 4 PR, 2 MR, 9 SD and 5 PD for ORR of 28% Among 11 pts treated ≥ 10 mg/kg 2 CR (20%), 2 PRs, 1 MR , 4SDs and 2 PDs for ORR of 40%	Ongoing
Duration of response	At doses from 4-24 mg/kg (n=12) Median PFS not reached at median follow up of 18.4 weeks (~ 4 months) vs. at 0.005-2 mg/kg (n=20) PFS is ~1 month (p=0.007)	Of 8 pts with objective response, 4 were on treatment for > 26 weeks, 1 > 52 weeks	Ongoing
TESAEs	Bronchospasm (2 pt), anemia (1 pt), thrombocytopenia (1 pt), ASAT > 5.2x ULN (1 pt), cytokine release syndrome (1 pt)	MTD not yet found at 20 mg/kg TEAE's fatigue, nausea, pyrexia, cough, anemia, headache	Ongoing
NCT	NCT00574288	NCT01084252	NCT01421186

Source: Oppenheimer & Co. Inc.

Ex Vivo and In Vivo Preclinical Models Suggest Superior Activity to Daratumomab

We are encouraged by signs of potential clinical efficacy in ex vivo models of XmAb13694. Xencor previously reported that XmAb13694 depleted CD38+ cells in *ex vivo* multiple myeloma patient plasma at 1µg/ml (Exhibit 15) and killed multiple myeloma cells *in vitro* with half-max redirected T-cell cytotoxicity (RTCC) of ~0.1 ng/ml. XmAb13694 was also found to induce serial lysis and was potent at low effector-to-target cell ratios.

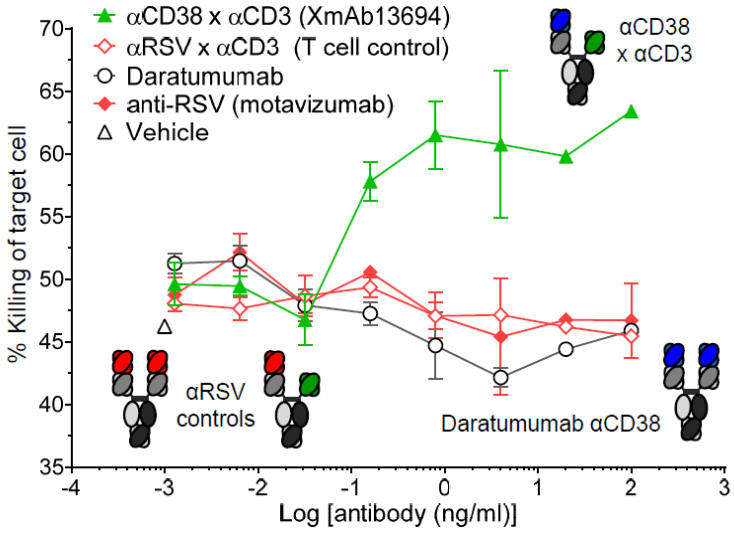
Exhibit 15: CD38xCD3 Bispecific Exhibits Strong Cytotoxicity *Ex Vivo*



Source: Xencor, Inc

Xencor's CD38xCD3 bispecific also demonstrated strong cytotoxicity against MM cell lines, significantly superior to daratumumab, a compound with promising single-agent activity in MM (Exhibit 16**Error! Reference source not found.**). Myeloma cells were incubated in the presence of PBMCs and antibodies for 24 hours.

Exhibit 16: CD38xCD3 Bispecific Exhibits Strong Cytotoxicity Against a Myeloma Cell Line



Source: Xencor, Inc.

Market—Multiple Myeloma

Multiple myeloma (MM) is a hematologic malignancy characterized by proliferation of plasma cells. Although median survival is high, approximately eight years, MM remains incurable due to high rates of relapse and resistance. Front-line therapy relies on several different chemotherapy regimens and ASCT (autologous stem cell transplant) in eligible patients. Although newer agents such as Velcade may eventually supplant ASCT, until prospective RCTs demonstrate improved survival without ASCT, it is likely to remain a mainstay of treatment.

We estimate there are roughly 24,000 patients diagnosed with multiple myeloma in the US each year. The National Institute for Health and Care Excellence (NICE) estimates that >90% of MM patients will receive third-line therapy. We estimate that XmAb13694 could be priced at $10,000 per cycle or ~$120,000 per year of therapy. We do not include XmAb13694 in our valuation at this time.

XmAb7195 for Severe Allergic Asthma and IgE Mediated Disease

Xencor's lead candidate, XmAb7195 is an IgE-directed antibody incorporating Xencor's proprietary Immune Inhibitory Fc domain. Xencor is developing XmAb7195 as a Xolair (omalizumab) bio-superior for the treatment of allergic asthma and other IgE mediated diseases. XmAb7195 entered Phase I trials in 2014, and data is expected by year-end 2014 (NCT02148744).

XNCR's Fc modifications incorporated into '7195 impart two novel mechanisms of action, resulting in superior clearance and reduction of IgE levels compared to Xolair and other high-affinity anti-IgE candidates in the clinic.

XmAb7195 improves upon approved anti-IgE therapy Xolair (omalizumab) by:

1) Clearing IgE from the bloodstream through liver sinusoidal cells;

2) Inhibiting further IgE production on B-cells by engaging the inhibitory FcγRIIIa receptor on B-cells; and

3) Binding IgE with 1.5x greater affinity than Xolair.

XmAb7195 Is Significantly De-risked by Xolair and XmAb5871

We believe that XmAb7195, though just entering the clinic, is likely to be safe and have anti-IgE activity given that the component Fc domain module is currently in Phase II trials with XmAb5871 and the target of the Fv domain has been validated by Xolair. We discuss XmAb5871's clinical safety experience in the sections below and note that Xolair (omalizumab), by targeting IgE, has demonstrated statistically significant reduction in clinically significant asthma exacerbations. Additionally, since animal models suggest safety of XmAb7195, we believe the sum of the anti-IgE and XmAb5871 Fc components is likely to also be safe in humans.

We expect Phase I data in Q4:14 to represent a significant de-risking event for this compound and to represent a major value inflection point for XNCR.

We Expect Safety and IgE Clearance Data from the Phase I Trial

Xencor initiated a Phase Ia, single ascending dose trial of XmAb7195 in May 2014 to investigate the safety and PK of XmAb7195 administered by IV infusion (NCT02148744). The trial will enroll eight cohorts of healthy and allergic (IgE > 300 IU/ml) patients and will record both free and total IgE levels. Following the Phase Ia portion, a Phase Ib multiple dose trial will be initiated in patients with mild to moderate asthma. We anticipate data, demonstrating safety and IgE clearance from the Phase 1a study by year-end 2014.

We anticipate results from the Phase I study of XmAb7195 in healthy volunteers, to be reported around year-end, will demonstrate that XmAb7195 results in a drop in IgE greater than or equal to Xolair's or below the detectable limit. Recall that treatment with Xolair resulted in a >90% drop in IgE, and treatment with QGE031 resulted in free IgE below the limit of quantification (est. 0.004 µg/ml) in all patients except the lowest dose in one SC cohort.

Exhibit 17: XmAb7195 Phase I Trial and Comparators

	XmAb7195 Phase Ia	Quilizumab Phase I	Xolair Pivotal Trials	QGE031 Phase I Trials
Condition	**Healthy volunteers (5 cohorts) and high-IgE allergic subjects (3 cohorts)**	Healthy volunteers	Moderate to severe allergic asthma uncontrolled by daily ICS and β-agonists	Atopic, but otherwise healthy patients
Design	**Single-ascending dose, 8 pts per cohort, randomized 6:2**	Single-ascending dose	RCT, 16-week stable steroid phase, 12-week steroid reduction phase	Single/multiple ascending dose
Doses	**Not disclosed**	0.003 – 5mg/kg IV, 3 mg/kg SC	Xolair dosing table	0.1-10 mg/kg (IV); 0.2-4 mg/kg (SC)
N	**56 (randomized 3:1)**	45	525 + 546, (randomized 1:1)	110 + 73 + 32 (215 total)
Safety	**Ongoing**	Well tolerated	Well tolerated	Well tolerated. 9 urticarial events in 8/215 (4%) patients
Primary endpoint	**Safety and tolerability**	Safety and tolerability	Incidence of asthma exacerbations	Safety and tolerability
Efficacy	**Ongoing**	Total IgE decreased 23% at 5 mg/kg IV, no reductions in SC or other lower dose IV cohort	Reduced free IgE by >90% vs. placebo. Reduced asthma exacerbations by 47% in steroid-stable phase and 53% in steroid reduction phase vs. placebo	Reduced free IgE below quantification levels (likely 0.004 µg/ml) at all doses except lowest SC dose (0.6 mg/kg) in Japanese subjects
Reference	**N/A**	Scheerens (2012)	Solèr (2001), Busse (2001), Hamliton (2005)	Bottoli (2014)

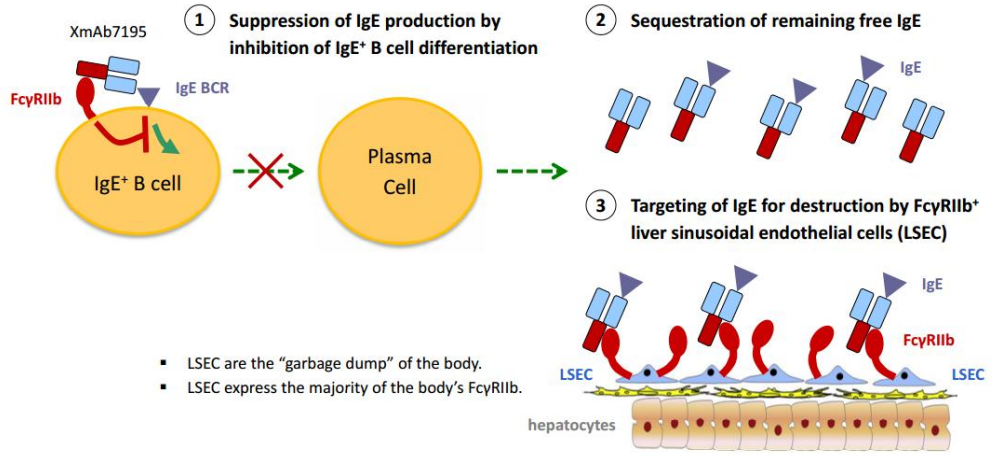
Source: Oppenheimer & Co. Inc.

Potential Phase II trials could enroll patients with poorly controlled asthma, including those ineligible for Xolair due to IgE/BMI restrictions (i.e., off the dosing table). Xencor has indicated that it may run a six-month Phase II trial. We anticipate further Phase II/III trials may include a 24-week reduction phase and endpoints of reduction in ICS dose and frequency of asthma exacerbations, similar to the pivotal Xolair trials in severe asthma. Xencor is also planning an IV-to-SC bridging study to enable SC dosing for the Phase II and III trials.

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. *In vivo* experiments highlight that XmAb7195's differentiated mechanisms contribute to greater IgE reductions, reducing IgE 10-fold better than Xolair. In preclinical studies in mice and chimpanzees, 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 applications in other indications beyond those in which Xolair has utility, because of '7195's differentiated mechanisms of action.

Exhibit 18: Novel Mechanisms of Action Imparted to XmAb7195 by Xencor's Fc-engineering

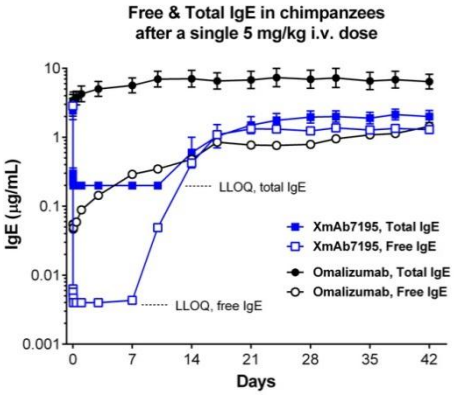


- XmAb7195 has two differentiating mechanisms of action due to its enhanced affinity for FcγRIIb: suppression of IgE+ B cells by IgE-FcγRIIb crosslinking plus rapid clearance of total IgE by liver sinusoidal endothelial cells (LSEC).
- We show that XmAb7195 rapidly clears total IgE in high-IgE chimpanzees in minutes and is >100-fold more effective than omalizumab at reducing free IgE.

Source: Xencor, Inc

In chimpanzees, XmAb7195 lowered both free and total IgE levels faster and for a greater duration than Xolair. A single 5 mg/kg dose of XmAb7195 reduced free IgE ~10x greater than Xolair (Exhibit 19). We also note that after XmAb7195 treatment, chimpanzees' free IgE levels stayed at trough levels for seven days, whereas the Xolair group began rebounding immediately. XmAb7195 was well tolerated with no adverse events in a 12-week, multiple-dose toxicology study in cynomolgus monkeys at doses up to 100 mg/kg.

Exhibit 19: XmAb7195 Reduced IgE more Potently and for Longer Duration than Xolair



Source: Moore et al. American Thoracic Society, C33. CYTOKINES AND ASTHMA MEDIATORS. May 1, 2014, A4261-A4261

Free IgE Is a Biomarker that Correlates with Clinical Indicators

IgE's Role in Asthma

Immunoglobulin E starts the allergic cascade by binding to the FcεR1 receptors on basophils and mast cells, triggering cytokine release, further IgE production and downstream inflammation pathways. IgE also plays a direct role in the long-term airway remodeling in asthma patients that leads to progressive airway constriction (Roth 2013).

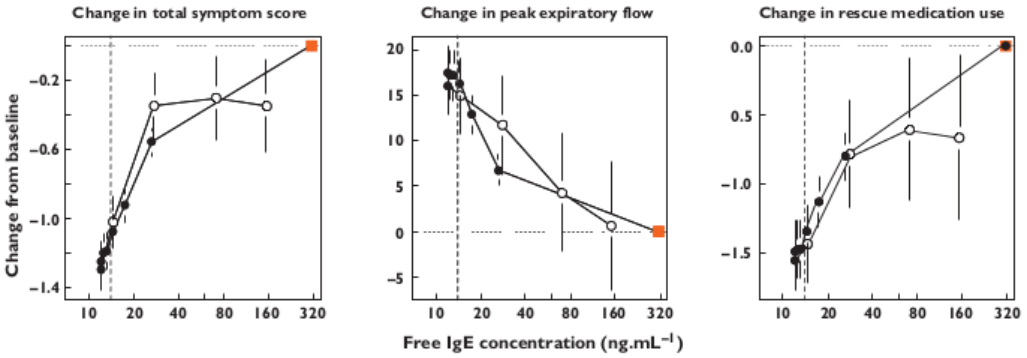
Free IgE levels represent an important biomarker of response, since they correlate strongly with several clinically important symptom scores including peak expiratory flow, rescue medication use and total symptom score. The correlation has been shown down to at least 10 ng/ml, below the ”ideal target” levels of IgE of 14 ng/ml and the commonly used target of 50 ng/ml.

IgE, Lower Is Better—How Low Can You Go?

IgE’s role in asthma and other allergic conditions suggests that lowering IgE beyond what Xolair is capable of could potentially produce greater clinical benefit (Exhibit 20). Recall that only 50% of patients receiving Xolair achieve 14 ng/ml free IgE levels, which may limit efficacy in allergic asthma patients and also other indications.

Additionally, XmAb7195’s ability to clear IgE from the plasma facilitates the use of a widely available total serum IgE test as a marker of potential clinical responses. This represents a noteworthy difference compared to Xolair, since Xolair increases total IgE, even though it decreases free IgE, necessitating the expensive free IgE test to monitor response. Since XmAb7195 clears IgE from the serum, 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 regimens and potentially faster routes to registration.

Exhibit 20: IgE Levels Correlate with Clinical Important Endpoints

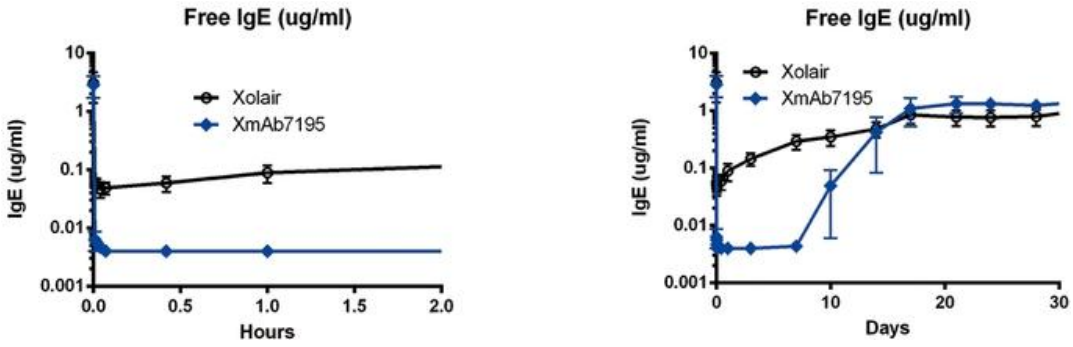


Source: Lowe et al. Br J Clin Pharmacol. 2009 Jul;68(1):61-76. Dotted line is 14 ng/ml

XmAb7195 Was Well Tolerated and Demonstrated Activity in Preclinical Studies

In preclinical studies in mice and chimpanzees, XmAb7195 was well tolerated and resulted in a larger drop and more sustained inhibition of IgE compared to Xolair (Exhibit 21). Xencor evaluated the activity of XmAb7195 compared to Xolair in a study of six 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 treatment resulted in free IgE below the 0.004 µg/ml detection limit for seven 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.)

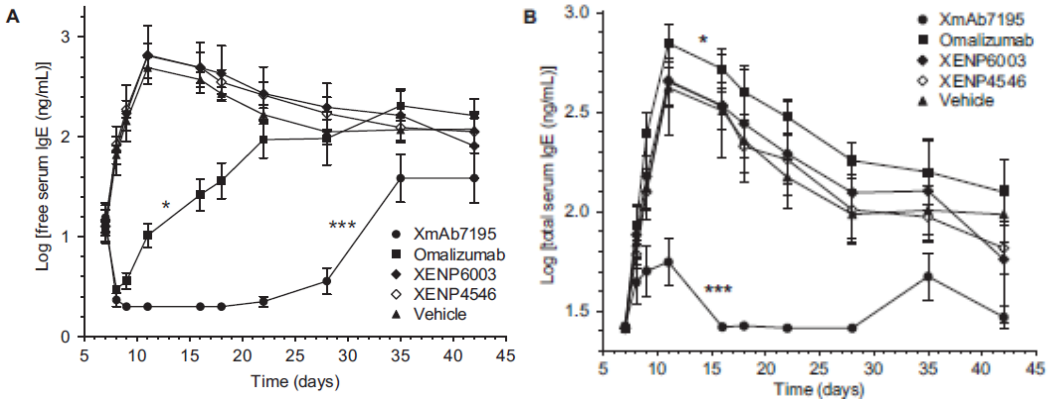
Exhibit 21: XmAb7195 at 5 mg/kg Reduces Free IgE Levels more than Xolair in a Chimpanzee Model



Source: Xencor, Inc.

XmAb7195 also reduced IgE levels, but generally not IgG or IgM levels, in huSCID mice, mice transgenic for the Fcγ receptors, and human fractionated plasma models, further validating the ability of this drug to lower IgE levels (Exhibit 22). 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 (this is well above our expectation of a 20 mg/kg highest human dose to be tested).

Exhibit 22: 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

We Anticipate that XmAb7195 Will Be Safe and Well Tolerated

We expect XmAb7195 to be safe and well tolerated based on similarity to Xolair, the natural history of genetically IgE deficient patients and preliminary but informative preclinical studies. Additionally, we do not believe that XmAb7195's novel engineered Fc-domain represents a significant threat to safety since it is the Fc domain employed by XmAb5871, which is in ongoing Phase Ib/IIa trials with no safety concerns noted to date (p. 36).

Recall that Xolair was extremely well tolerated in large (3,507 patients) clinical trials. Significant adverse events (SAEs) were rare and most significantly included anaphylaxis in 0.1% of patients. Importantly, there are no serious consequences of reducing IgE too much. A randomized, controlled clinical trial in Brazil in patients at risk of geohelminth infection found a small trend toward a higher rate of infection; however, we note that a small increase in the risk of a treatable and rare disease is unlikely to be of clinical significance.

Finally, there does not appear to be a safety concern with IgE levels of 0 as patients born with a complete genetic deficiency in IgE are known to be healthy and asymptomatic.

Significant Unmet Medical Need Exists in Asthma Despite Xolair

Xolair, despite being approved for the treatment of severe asthma and with annual sales in excess of $1.6 billion worldwide, is contraindicated in many patients with high body weight or very high IgE levels (Exhibit 23) due to the large dosing volumes of Xolair required. Recall that Xolair dosing is calculated based on a table designed so that each patient receives ~0.016 mg/kg/IgE; however, no more than 375mg can be given at one time in the US, and no more than 600mg in the EU. If the patient's weight or IgE is too high to receive the proper dose of Xolair, the drug should not be administered.

Restrictions on Xolair dosing mean that ~1/3 of severe allergic asthma patients are ineligible for Xolair in the US. Furthermore, even in this restricted population, only ~1/2 of eligible patients are able to achieve ideal (14 ng/ml) IgE levels (Lowe 2009). Since XmAb7195 has demonstrated an ability to produce greater reductions in IgE (at lower doses) than Xolair in preclinical models, '7195 may find utility in this patient population. This potentially indicates a market opportunity for XmAb7195 first in the Xolair ineligible patients, then in Xolair-eligible patients and finally beyond asthma in IgE mediated diseases, such as urticaria.

We believe that activity in the Xolair restricted patient population (i.e., off the Xolair dosing table) and the high non-responder rate leave significant unmet medical need and a significant opportunity for XmAb7195.

Exhibit 23: US and EU Xolair Dosing Tables

US

Baseline IgE (IU/ml)	Body Weight (kg) 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

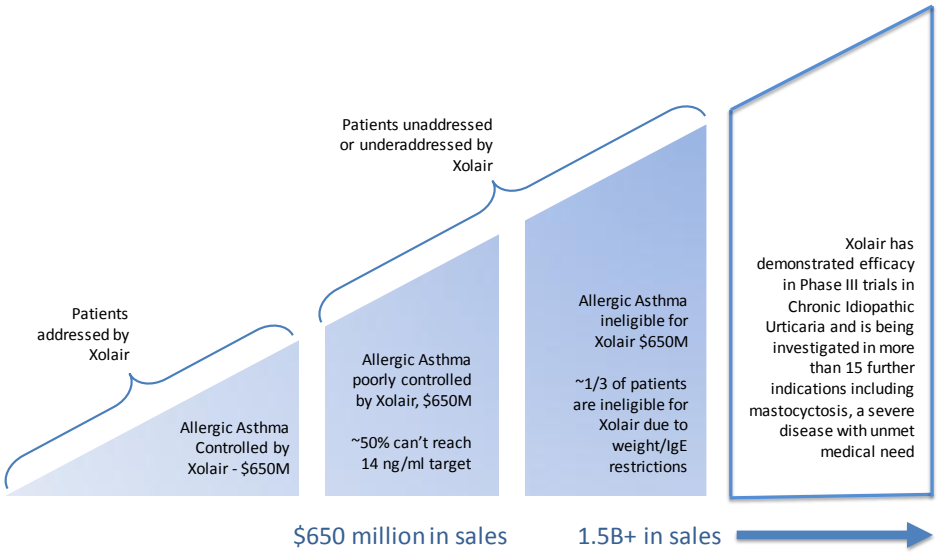
EU

Baseline IgE (IU/ml)	Body Weight (kg) 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

Source: Oppenheimer & Co. Inc.

Exhibit 24: Potential XmAb7195 Market Opportunity



Source: Oppenheimer & Co. Inc.

Indications Beyond Asthma Offer Attractive Markets and Routes to Registration

IgE is involved in inflammatory diseases beyond allergic asthma. Recall, Xolair recently received FDA approval in chronic idiopathic urticaria, and is being tested in several other urticaria types. We highlight that XmAb7195 may offer greater efficacy than Xolair in indications outside of asthma as well and that indications such as urticaria that could offer label expansion opportunities or a more rapid route to registration (Exhibit 25). Additionally, we highlight several case reports in systemic mastocytosis that have indicated improvement in signs and symptoms after treatment with Xolair. If XmAb7195 demonstrates efficacy in systemic mastocytosis, it could be a profound and potentially life-altering therapy that may support a rapid route to registration.

Exhibit 25: IgE Directed Therapy in Indications Beyond Asthma

Indication	Author	Type of Evidence
Allergic rhinitis	Humbert 2009	Retrospective analysis
Nasal polyposis	Penn 2007	Pilot study
Allergic bronchopulmonary aspergillosis	Lebecque 2009, Wong 2013	Case reports / case series
Atopic Dermatitis	Sheinkopf 2008	Pilot study
Urticaria	Maurer 2013	PIII RCT
Mastocytosis	Douglass 2013	Case reports
Ménière disease w/ mastocytosis	Siebenhaar 2007	Case reports
Food-triggered anaphylaxis	Leung 2003	RCT
Idiopathic anaphylaxis	Jones 2008	Case reports
Food allergy	Rafi 2010	RCT
Eosinophilic esophagitis	Rocha 2011	Case reports
Latex Allergy	Williams 2005	Pilot study

Source: Oppenheimer & Co. Inc.

Competition—IgE Directed Therapies

Two other IgE directed therapies are currently in clinical development for asthma (Exhibit 17, p. 23). Quilizumab (Roche), an antibody directed to the M1' (M1 prime) segment of membrane bound IgE designed to kill IgE producing B-cells, is currently in Phase II trials, and QGE031 (Novartis) is an anti-IgE antibody with higher affinity for IgE than Xolair. We note that a previous Phase II trial of an affinity-enhanced anti-IgE antibody (Genentech, PRO98498) was discontinued after completing a Phase II trial.

Quilizumab—Roche/Genentech

Quilizumab, RG7449/MEMP1972A, is a humanized antibody targeted to the M1' segment of membrane-bound IgE. This antibody utilizes fucosyl modifications to enhance ADCC (antibody-dependent cell-mediated cytotoxicity), resulting in specific killing of IgE-expressing B cells, preventing all IgE production. This antibody can also induce cross-linking of the BCR complex, which leads to apoptotic signaling and can cause cell death if the B-cell does not also receive co-stimulatory signals.

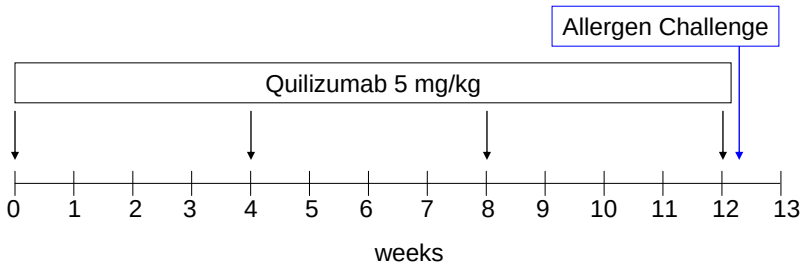
Exhibit 26: Quilizumab Trials

	Phase II	Phase I	Phase I	Phase II
Condition	Mild asthma	Allergic rhinitis	Healthy volunteers	Refractory Chronic Spontaneous Urticaria
N	28	36	45	30
Safety	Well tolerated	Well tolerated	Well tolerated	Ongoing
Primary endpoint	AUC of FEV1 in late allergic response	Safety and tolerability	Safety and tolerability	Change from baseline to week 20 in itch score, safety
Efficacy	AUC of FEV1 in early allergic response decreased by 26% (p=0.046). AUC of FEV1 in late allergic response decreased by 35% (p=0.21)	Total IgE decreased 24% in 5 mg/kg IV and 26% in 3 mg/kg SC	Total IgE decreased 23% at 5 mg/kg IV	Ongoing
NCT	NCT01196039	NCT01160861	N/A	NCT01987947
Reference	Gauvreau 2012	Scheerens 2012	Scheerens 2012	N/A

Source: Oppenheimer & Co. Inc.

A phase II trial, SOLARIO, enrolled 29 subjects to investigate the safety and efficacy of quilizumab in mild asthma following allergen-induced airway obstruction. Patients were randomized 1:1 to receive placebo or quilizumab 5 mg/kg Q4W for 12 weeks. The primary outcome measure was the area under the curve (AUC) of FEV1 decline in the late (3-7 hours) asthmatic response (LAR) after allergen challenge.

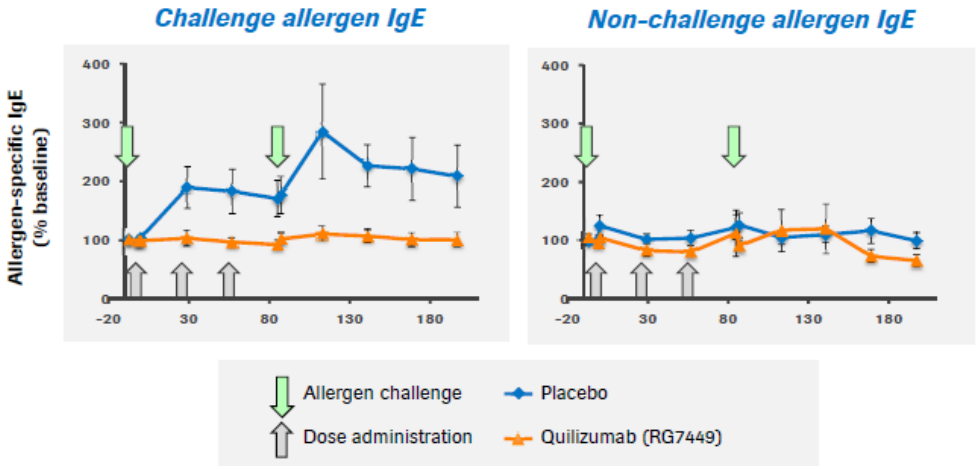
Exhibit 27: Quilizumab Trial Design



Source: Oppenheimer & Co. Inc.

Of the enrolled subjects, 28 completed the trial, 15 in the treatment group and 13 in the placebo group. One patient in the placebo group discontinued due to worsening symptoms. At week 12, AUC of the late allergic response was 36% lower in the quilizumab treated group vs. placebo (p=0.21). AUC of the early allergic response was 26% lower in treated vs. placebo groups (p=0.046). No effect was noted on airway hyper-responsiveness to methacholine. Quilizumab reduced total serum IgE by ~20% from baseline to eight weeks (p<0.01 vs. placebo). Allergen challenge at baseline and week 12 induced a ~2-fold increase in serum allergen-specific IgE, which was completely prevented by quilizumab (Exhibit 28).

Exhibit 28: Quilizumab Reduces IgE



Source: Roche, Inc.

Two Phase I trials investigated the safety and tolerability of quilizumab in healthy volunteers (n=31 quilizumab, n=14 placebo) and in adult patients with allergic rhinitis (n=24 quilizumab, n=12 placebo). Quilizumab was safe and well tolerated in both studies. Half-life was 20-21 days. In healthy volunteers, the highest dose tested, 5 mg/kg IV single dose, resulted in a 23% reduction in total IgE at day 85, a 3 mg/kg IV single dose resulted in a 28% reduction compared to no reduction in the 3 mg/kg SC cohort or placebo cohorts. In patients with allergic rhinitis, three monthly doses at 5 mg/kg IV resulted in total IgE reductions of 24% and 3 mg/kg SC resulted in mean reduction of 26%. No significant reductions were observed in the placebo cohort. Serum total IgE reduction was sustained for six months after dosing in both studies.

AUC of FEV1 decline is a validated endpoint, used as the primary endpoint in one of the two pivotal clinical trials that led to Dulera's approval in asthma and is used extensively in COPD.

QGE031 (ligelizumab)—Novartis

Novartis is developing QGE031, an anti-IgE antibody with higher binding affinity than Xolair. We note that a previous Phase II trial of an affinity-enhanced anti-IgE antibody (Genentech, PRO98498) was discontinued after completing a Phase II trial.

QGE031 has been tested in three Phase I trials in atopic (allergic) but otherwise healthy patients.

The largest trial 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. Two patients receiving QGE031 at 2 mg/kg withdrew due to symptoms considered unrelated to the drug. Four 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 (est. 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.

Another trial enrolled 73 patients to receive IV QGE301 (n=36), IV placebo (n=29), or SC Xolair (n=8). The discontinuation rate was 17.8% (unrelated to adverse events, AEs). Urticaria occurred in 4/73 subjects; 3 at the higher IV doses and 1 on placebo.

A third trial in Japanese patients (NCT01596712) enrolled 32 patients to receive SC QGE301 (n=28) or placebo (n=8) at single doses of 0.6, 2 and 4 mg/kg. A Phase II trial (NCT01716754) was initiated early in 2013 with an estimated 457 patients comparing the safety and efficacy of QGE031 against placebo and Xolair comparators in allergic asthma.

Competition—Asthma Therapies in Development

There are at least 19 therapies in development for asthma. Many of these therapies are cytokine inhibiting therapies, designed to halt the uncontrolled immune activation that leads to swelling. While anti-cytokine therapies, including anti-IL1β, anti-IL-12/23, and anti-IL-6R, have demonstrated efficacy and safety in Phase II trials in inflammatory and auto-immune diseases, the cytokine cascade is complex and still poorly understood. Consequently, we believe that many of the upcoming asthma therapies represent high-risk approaches compared to Xencor's XmAb7195.

Exhibit 29: Asthma Therapies in Development

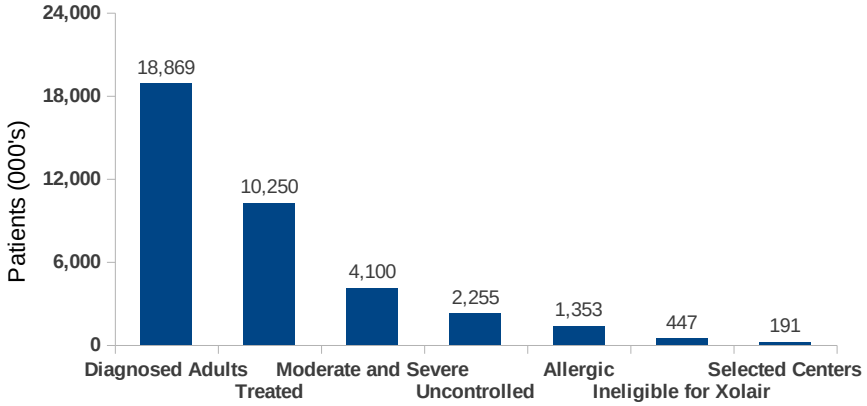
Company	Name	Type	Target	Phase	Notes / next event
Teva	Cinquil (reslizumab)	mAb	IL-5	Phase III	PIII Trial Results mid-14
MedImmune	Benralizumab	mAb	IL-5R	Phase III	PIII FPI planned Q3:14, BLA 2016
GSK	Mepolizumab	mAb	IL-5	Phase III	BLA filing planned for YE:14
Genentech	Lebrikizumab	mAb	IL-13	Phase III	PIII ongoing, BLA 2016
Amgen / Kyowa Hakko Kirin	Mogamulizumab	mAb	CCR4	Phase III	Partnership terminated by Amgen April '14
Amgen	Brodalumab	mAb	IL-17	Phase III	Asthma RCT negative, possible subset, postive PIII data in psoriasis, next event update on path forward in asthma
AB Science	Masitinib	SMI	TKI	Phase III	3rd rejection by CHMP for mastinib in oncology, PIII asthma FPI 2011
Sanofi/Regeneron	Dupilumab	mAb	IL-4Ra	Phase III	2 yr outcome, 560pt Phase III initiated in May 2013
Amgen	AMG-853	SMI	CRTh2	Phase II	Phase II negative, no ongoing trials
Novartis	**QGE031**	**mAb**	**IgE**	**Phase II**	**Phase II completed Jan. '14, awaiting results and update**
Roche	**Quilizumab**	**mAb**	**Bound IgE**	**Phase II**	**Phase II results positive, awaiting update on path forward, BLA not expected by Roche until later than 2016**
Novartis	QAW039	SMI	CRTh2	Phase II	336 pt, 12 wk, PII in non-atopic asthma pts initiated May '14
Novartis	QAX576	mAb	IL-13	Phase II	60 pt, 26wk, PII in asthma ongoing (clincialtrials.gov), FPI April '12
MedImmune	Tralokinumab	mAb	IL-13	Phase II	PIII initiation expected Q3:14
MediciNova	Ibudilast	SMI	PDE-4	Phase II	Used for asthma in japan since 1990, US PII trials in MS and substance dependence ongoing, no US asthma trial ongoing
JNJ	JNJ-40929837	SMI	Lta4h	Phase II	Phase II negative, no ongoing trials
Revalesio	RNS60	Ionic	-	Phase I	Phase I data: well tolerated, no FEV1 effect, no PII asthma ongoing
Aztrazeneca/MedImmune	**MEDI-4212**	**mAb**	**IgE**	**Terminated**	**High affinity anti-IgE mAb, failed two Phase II trials**
Genentech	**PRO98498**	**mAb**	**IgE**	**Terminated**	**High affinity anti-IgE mAb based on Xolair, discontinued due to hypersensitivity reactions in PII trial**

Source: Oppenheimer & Co. Inc.

Market—Moderate to Severe Allergic Asthma

We estimate that if approved in the Xolair-ineligible population, XmAb7195 could generate peak sales in excess of $1.4B. We estimate there are ~200,000 Xolair-ineligible patients in the US seen at leading centers that are likely to prescribe XmAb7195. We estimate XmAb7195 is worth $12/share to Xencor by applying a typical 6x multiple to our estimated 2022 US revenues of $1.2 billion for XmAb7195 in moderate to severe asthma, discounted 45% annually.

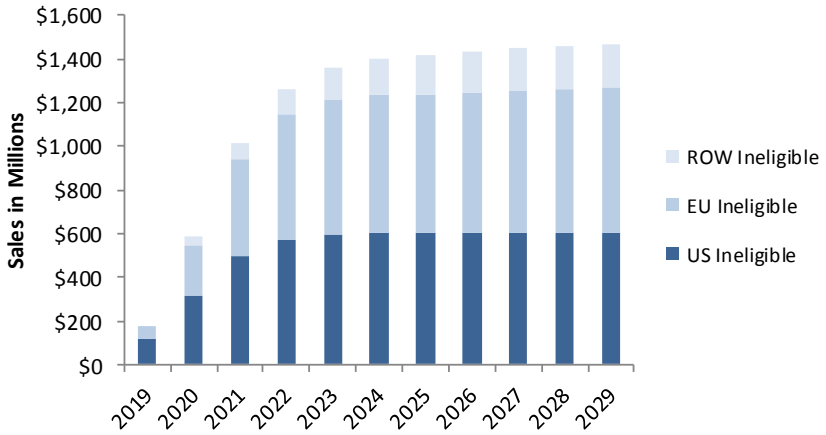
Exhibit 30: Estimated Patient Population Eligible for XmAb7195



Source: Oppenheimer & Co. Inc.

We estimate a market penetration of 10%, 8% and 3% in Xolair-ineligible patients in the US, EU and rest of world (ROW), respectively. We estimate 2022 sales of $1.2B worldwide.

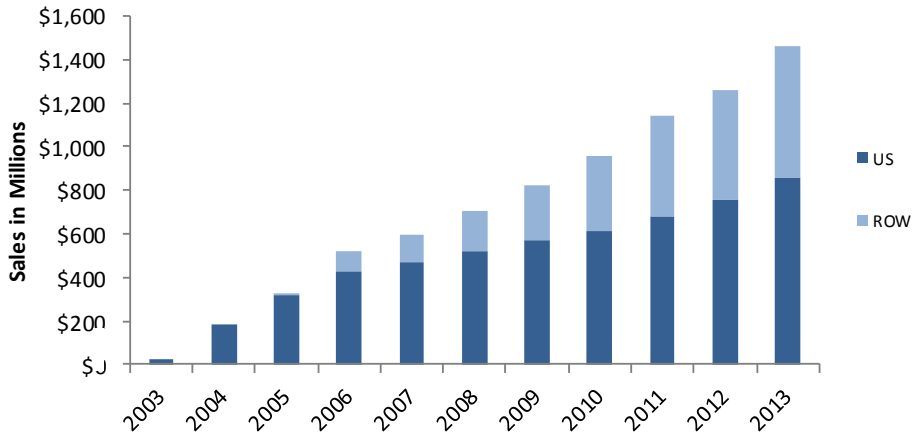
Exhibit 31: XmAb7195 Sales Projections



Source: Oppenheimer & Co. Inc.

Xolair sold $1.65B worldwide and $850M in the US in 2013, by our estimates representing ~27,000 patients per year. This represents an estimated market penetration of ~7%. Our patient estimate is based on an a 2013 average wholesale price (AWP) of $875.14 per 150mg vial and retrospective data indicating the average US Xolair patient receives ~36 150mg (or equivalent) vials per year (Campbell 2010).

Exhibit 32: Xolair Historical Sales



Source: Oppenheimer & Co. Inc.

We estimate that XmAb7195 is worth $12/share to Xencor based on a 6x multiple of our estimated 2022 revenues of $1.2 billion for XmAb7195 in moderate to severe asthma, discounted 45% annually.

XmAb5871 for Autoimmune Disease

XmAb5871 is a CD19 targeted immune inhibitory antibody that uniquely employs Xencor's technology to co-engage FcγRIIb to inhibit B-cell activation. Importantly, unlike XmAb5574/MOR208 and CD20 antibodies (such as rituximab), it does not kill B-cells, but instead cross-links the FcγRIIb receptor to the B-cell receptor (BCR), where FcγRIIb inhibits BCR signaling and B-cell activation.

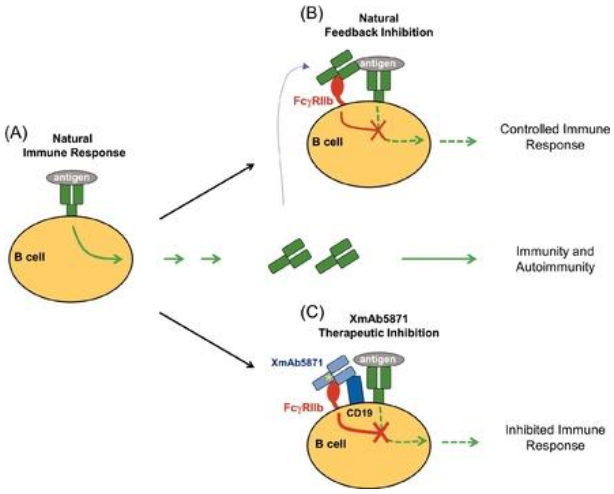
XmAb5871 is in a Phase IIa trial in patients with rheumatoid arthritis. Amgen's option to license is valid only after the completion of a predefined Phase IIb trial. Data from the ongoing Phase Ib/IIa trial in patients with RA is expected around year-end 2014.

FcγRIIb Inhibits BCR Signaling—Opportunities in Autoimmune Disorders and Oncology

FcγRIIb is an inhibitory receptor expressed on B-cells that normally binds the Fc domain of IgG antibodies with low affinity where it functions in a negative feedback loop to limit the normal antibody response. FcγRIIb only inhibits B-cell signaling when it is recruited to the BCR. Xencor's proprietary Fc modifications increase the affinity of their antibodies for this receptor.

Inhibiting B-cell activity represents a validated as a target in rheumatoid arthritis by Rituxan (rituximab), a B-cell depleting therapy. XmAb5871 inhibits B-cells, but does not kill them as Rituxan does. This may allow physicians to rapidly reverse B-cell inhibition in the event of an infection, improving the safety profile and broadening the potential patient population. Rituxan treatment, by contrast, depletes B-cells for months or years (Leandro 2006), inhibiting the immune response and causing an increased risk of severe infection and death in patients with auto-immune diseases (Díaz-Lagares 2011).

Exhibit 33: XmAb5871 Recruits FcγRIIb to the BCR and inhibits B-cell Activation



Source: Xencor, Inc

Phase Ib/IIa Trial Design

In January 2013, Xencor initiated a Phase Ib/IIa placebo-controlled, double-blind, multiple ascending dose Phase IIa 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 by IV administration. In October 2013, Xencor initiated the second part of the study that will enroll 30 patients with active disease randomized 2:1 XmAb5871 vs. placebo at the highest 10 mg/kg dose every 14 days for a total of six cycles. Inclusion criteria includes 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, hsCRP ≥ 10 mg/L, or morning stiffness ≥ 45 minutes. The primary outcome measures of the study include determination of safety, tolerability and immunogenicity of XmAb5871. Secondary endpoints will assess clinical outcomes measured as Disease Activity Score 28 using C-reactive protein (DAS28-CRP) after 13 weeks.

XmAb5871 has been found to be well-tolerated in trials reported to date. 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 late 2014. A potential IV to subcutaneous formulation bridging study for XmA5871 is also planned.

Exhibit 34: XmAb5871 Phase Ib/IIa Trial and RA SOC Comparator

	XmAb5871 Phase IIa	XmAb5871 Phase Ib
Indication	Patients with active rheumatoid arthritis on stable non-biologic disease modifying anti-rheumatic drug therapy (DMARD)	Patients with active rheumatoid arthritis on stable non-biologic disease modifying anti-rheumatic drug therapy (DMARD)
N	30	29
Design	Double-blind, placebo-controlled, randomized 2:1	Double-blind, placebo-controlled, randomized
Dose	10 mg/kg XmAb7195 in biweekly infusions for 6 cycles	0.3, 1, 3 and 10 mg/kg in biweekly infusions for 6 cycles
Primary endpoint / results	Primary endpoint is disease activity score 28 (DAS28) using C-reactive protein at 13 weeks	Safe and well tolerated, reduced B-cells in a dose-dependent manner
Eudra-CT #	2012-003057-29	2012-003057-29

Source: Oppenheimer & Co. Inc.

XmAb5871 Demonstrated Safety and Activity in First-in-Human Phase Ia Trials

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 (one 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 (Exhibit 35). Challenge based tests of XmAb5871 were conducted by immunizing healthy subjects with tetanus and KHL to elicit antibody responses. XmAb5871 effectively suppressed immune responses in 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.

Exhibit 35: XmAb5871 Transiently Reduced B-cells in a Phase I Trial

		Days								
Cohort	Dose (mg/kg)	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

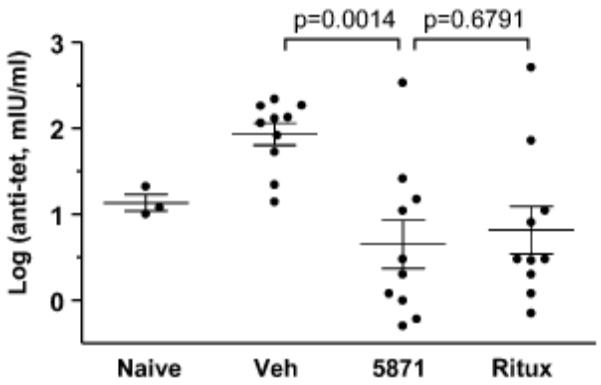
> 70%
30 - 70%
< 30%

Source: Xencor, Inc

Preclinical Data Highlight Reversibility and Efficacy vs. Rituximab

Preclinical data demonstrate that XmAb5871 inhibits immune response to tetanus challenge in SCID mice engrafted with SLE PBMC (Exhibit 36). 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 preclinical studies in monkeys. No adverse events were noted in doses up to 200 mg/kg.

Exhibit 36: XmAb5871 Inhibits Immune Response in SCID Mice Engrafted with SLE PBMC



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

XmAb5871 an Alternative to B-cell Depletion for Autoimmune Disease

We believe that XmAb5871, by inhibiting B-cell activation instead of depleting B-cells, may be similarly efficacious to current B-cell targeted therapies but better tolerated, with a potentially lower risk of serious infections. 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. Rituximab (Rituxan/MabThera), an increasingly adopted B-cell depletion therapy for autoimmune disease, improves symptoms but also depresses the immune system and increases infection risk. Additionally, TNF-alpha inhibitors (such as adalimumab, Humira) are also associated with infection risk and other serious side effects, and also frequently stop working in patients after as little as one year on therapy.

XmAb5871 Market Opportunity—Rheumatoid Arthritis and Lupus

Xencor and potential option/partner Amgen may initially develop XmAb5871 to establish proof of concept in systemic lupus erythematosus (SLE) or rheumatoid arthritis (RA), or other orphan indications, with high-unmet medical need that may offer more targeted and faster routes to registration. **Regardless of initial indication, however, we would anticipate, based on the mechanism of action, that XmAb5871 would have label expansion opportunities and be broadly used off-label for autoimmune disorders, paralleling the current use of Rituxan in these indications.**

The Competitive Landscape in B-cell Targeted Therapies

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, it will suffer from the same irreversibility problems as Rituxan.

Exhibit 37: B-cell Directed Therapies for Autoimmune Diseases

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

Significant unmet need and large markets for drugs that treat inflammatory disorders suggest to us that despite competition, there is likely a place for novel therapies like Xencor's '5871. However, recent small molecules that function on the BCR-signaling pathway may play a role in future treatments for inflammatory disease. We highlight that Imbruvica (ibrutinib) and next-generation BTK inhibitors may offer a similar approach to XmAb5871 by working to quiet B-cells to elicit disease response without B-cell depletion.

Market Opportunity

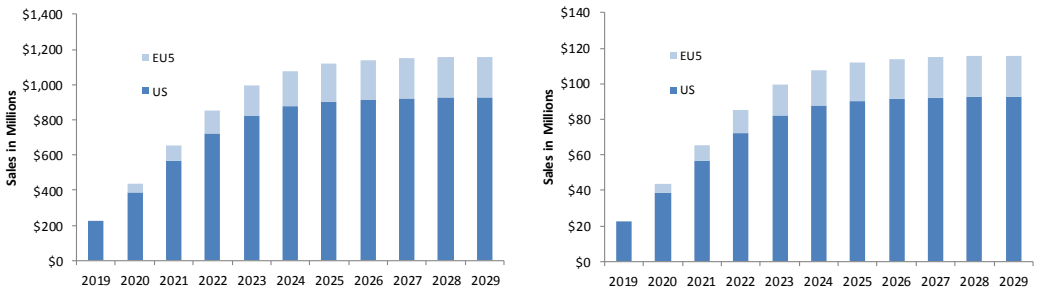
Rheumatoid arthritis (RA) is a chronic auto-immune disease that causes joint pain, difficulty walking and cardiovascular disease. RA affects nearly 1.3 million adults in the US. The current standard of care consists of disease-modifying anti-rheumatic drug (DMARD) therapy including steroids, TNF-α, and B-cell depleting rituximab, yet despite these options, not all patients are adequately controlled. .

Systemic lupus erythematosus (SLE) is a chronic auto-immune disease of unknown etiology and highly heterogeneous presentation. High dose steroids and immunotherapy have improved five-year survival to 90% from 50%, but despite this, patients are still afflicted by regularly occurring flare-ups. 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.

If approved in refractory rheumatoid arthritis, we estimate that XmAb5871 could achieve peak sales in excess of $1.1B. We estimate that there are approximately 370,000 RA patients refractory to biologic and DMARD therapy in the US and 85,000 in the top five EU countries. If priced similarly to Humira (adalimumab), a TNFα inhibitor approved for the treatment of RA, ($25,000/year), XmAb5871 could, with a conservative 10% penetration rate, generate $1.2 billion in sales in RA alone in the US and top five EU.

We estimate that XmAb5871 is worth $3/share to Xencor based on a 15x multiple of estimated 10% royalties from expected 2022 US and EU5 sales of $900M, discounted 40% annually. We believe upside to our sales expectations exists, should XmAb5871 show efficacy in additional indications including SLE.

Exhibit 38: XmAb5871 Sales Projections

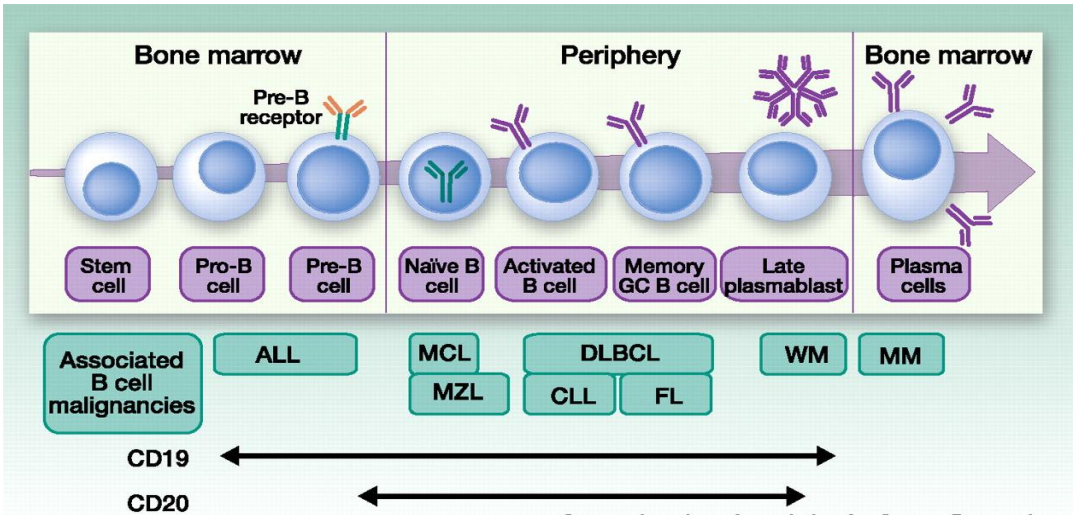


Source: Oppenheimer & Co. Inc.

MOR208 (XmAb5574)

XmAb5574/MOR208, licensed to MorphoSys, is a CD19 targeted antibody with Xencor's proprietary enhanced ADCC Fc domain. The antibody was engineered to deplete B-cells more effectively than other B-cell depleters such as rituximab (Rituxan/MabThera) but is directed against CD19, a target expressed more broadly on B-cells. CD19 is expressed only on B-cells like CD20, but is expressed both earlier and later in B-cell maturation than CD20 (Exhibit 39).

Exhibit 39: CD19 Is Expressed Both Earlier and Later than CD20



Source: Blanc et al. Clin Cancer Res. 2011 Oct 15;17(20):6448-58

XmAb5574/MOR208 is being tested in three ongoing Phase II trials in NHL, B-ALL and CLL/SLL. Full data from the B-ALL trial and interim data from the NHL trial may be presented at ASH 2014 (Dec. 6-9, San Francisco, CA).

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).

Phase II Trial in B-ALL

The open-label trial in B-ALL (NCT01685021) is enrolling 30 patients with B-ALL refractory to at least one prior therapy; however, 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 seven months; secondary outcome measures include duration of response as measured by bone marrow aspirates, safety, pharmacokinetics and anti-XmAb5574 antibodies.

Phase II Trial in NHL

The 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 Rituxan. The primary outcome measure is ORR at four years; secondary outcome measures include duration of response as measured by bone marrow aspirates, safety, pharmacokinetics and anti-XmAb5574 antibodies.

Investigator-sponsored Trial in CLL

Additionally, Dr. Jennifer 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, in patients with CLL patients, half treatment-naïve and half relapsed/refractory. 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 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 six months. Secondary outcomes of the study will include ORR, PFS, time to next treatment and overall survival (OS) at 12 months. The study will also examine cytogenetic factors, NK and T-cell activation and changes in protein expression.

Exhibit 40: Ongoing Phase II Trials of MOR208

Investigator	MorphoSys	MorphoSys	Dr. Woyach
Design	Open-label	Open-label	Open-label
Indication	FL, MCL, DLBCL, MALT/MZL or other indolent B-cell NHL refractory to Rituxan	R/R B-ALL	Richter negative intermediate or high risk CLL, SLL or B-PLL patients, half naïve and half relapsed/refractory
N	120	30	40
Dose / schedule	MOR208 12 mg/kg on Days 1, 2, 8, 15 and 22 of a 28-day cycle	MOR208 12 mg/kg on Days 1, 2, 8, 15 and 22 of a 28-day cycle	MOR208 12 mg/kg on Days 1, 2, 8, 15 and 22 of a 28-day cycle and lanolidomide PO for ≤ 12 cycles
Primary endpoint	ORR at 4 years	ORR at 7 months	ORR at 6 months
Secondary endpoints	Duration of response by bone marrow aspirates	Duration of response by bone marrow aspirates	ORR, PFS, 12-month OS
NCT	NCT01685008	NCT01685021	NCT02005289

Source: Oppenheimer & Co. Inc.

MOR208/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, 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 Rituxan). 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. No complete responses were observed, though this observation is not unexpected given the highly pre-treated patients, and progression on prior anti-CD20 therapies.

Exhibit 41: Adverse Events Occurring in the MOR208 Phase I Trial

Dose	0.3 m/kg	2 mg/kg	2 mg/kg	2 mg/kg	2 mg/kg	12 mg/kg	Total (%)
N	1	1	3	3	3	16	27
Dose Limiting Toxicities							
Grade 4 Neutropenia lasting ≥7 days with febrile neutropenia	-	-	-	-	-	1	1 (3.7)
Grade ≥ 3 Toxicities							
Neutropenia	-	1	-	-	-	2	3 (11.1)
Thrombocytopenia	-	-	-	-	-	2	2 (7.4)
Febrile Neutropenia	-	-	-	-	-	1	1 (3.7)
Tumor Lysis Syndrome	-	-	-	-	-	1	1 (3.7)
Increased AST	-	-	-	-	-	1	1 (3.7)

Source: Oppenheimer & Co.

Grade 1/2 infusion reactions occurred in 89% of patients. Initial evidence of efficacy was also noted, including 3 PRs (11%) using IWCLL 1996 guidelines and 13 PRs (42%) by IWCLL 2008 guidelines which include CT scans.

Exhibit 42: MOR208 Phase I Trial and Comparator

	MOR208/XmAb5574 Phase I	MEDI-551 Phase I/II	Gazyva (anti-CD20) Phase I
Indication	Relapsed / refractory CLL or SLL	Relapsed / refractory B-cell malignancies	Relapsed / refractory CD20+ NHL
Design	Accelerated titration for dose levels 1 and 2, followed by 3+3 with expansion at MTD.	3+3 dose escalation	3 + 3 design
N	27	91	21
Dose levels	0.3, 1, 3, 6, 9, and 12 mg/kg administered IV on days 1, 4, 8, 15, 22 in Cycle 1 and days 1, 8, 15, 22 in Cycle 2 on 28-day cycles	3+3 dose escalation at 0.5, 1, 2, 4, 8, or 12 mg/kg on days 1 and 8 of cycle 1, then on day 1 of subsequent cycles	50 mg to 2000 mg (flat) given IV on days 1, 8 and 22 and subsequently every 3 weeks for a total of 9 infusions
Adverse Events	Grade 4 febrile neutropenia at 12 mg/kg, TEAE's Grade 3-4 included neutropenia (3 pts, 11%), thrombocytopenia (2 pts, 7%), increased AST, febrile neutropenia, and tumor lysis syndrome (1 pt each). All on 12 mg/kg except 1 pt on 1 mg/kg who experienced Grade 3-4 neutropenia. 18 pts (75%) experienced Grade 1-2 infusion reactions	MTD not reached, TEAEs included Grade 4 neutropenia in 5 pts (5.5%), Grade 3 neutropenia in 6 (6.6%), Grade 3 back pain and infusion reaction in 2 (2.2%)	TEAE Grade 3 included neutropenia (2 pts, 9.5%), anemia and thrombocytopenia (1 pt each). No DLT's, dose-reductions or Grade 4 TEAE's
Responses	3 PRs (11%), all in CLL patients. Using non-CT response criteria, 13 pts (42%) had PR. Only 2 pts had PD at 8-weeks.	Of 83 evaluable pts, 9 CRs (11%), 12 PRs, 42 SDs for an ORR of 25%, median PFS ~9 months	5 CR/CRu (23%), 4 PR (19%), for ORR of 43%.
NCT	NCT01161511	NCT01957579	NCT00517530
Reference	Woyach 2012	Forero-Torres (2013)	Salles (2012)

Source: Oppenheimer & Co. Inc.

CD19 Targeted Therapy Competitive Landscape

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

Amgen is developing blinatumomab, a CD19/CD3 bi-specific T-cell engaging (BiTE) antibody, in Phase II trials in NHL and ALL that has received breakthrough designation from the FDA. A Phase II trial (Topp 2014) enrolled 189 patients with R/R B-ALL and administered blinatumomoab four weeks on, two weeks off to receive for up to five cycles 9 µg/day days 1-7, then 28 µg/d. Of enrolled patients, 43% achieved CR or CR with partial hematological recovery (CRh). AEs included pyrexia (59%), headache (35%) and febrile neutropenia (29%). Three patients (2%) had Grade 5 TEAEs, 2 sepsis and 1 candida infection.

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 Kettering-Sloan Cancer Center. All five patients achieved complete response as assessed by PCR. Therapy was well-tolerated although cytokine release syndrome required steroid therapy in some patients. Earlier CD19-targeted CAR-T therapies are also discussed in the introduction (Exhibit 8, p. 12).

Two CD19-targeted ADCs are also in the clinic, SAR3419 (Sanofi, licensed from IMGN, Perform) and SGN-CD19A (SGEN, Perform). A Phase II trial (STARLYTE) of SAR3419 found an ORR of 43.9% in 41 patients with CD19+ DLBCL R/R to at least one treatment (Trneny 2014). A Phase I trial of SGN-CD19A found an ORR of 40% in 20 patients in a dose escalation trial in patients with B-cell NHL R/R to at least one treatment (Forero-Torres 2014).

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.

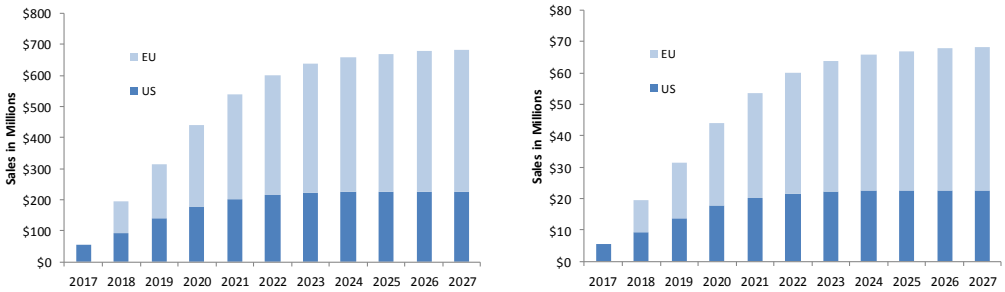
The market for B-cell related therapies is becoming, in our opinion, crowded with several new therapies including BTK-inhibitors (ibrutinib, IMBRUVICA), PI3K-inihbitors (idelalisib, CAL-101 – GILD, Outperform) and new B-cell depleters (Gazyva/obinutuzumab/GA-101 - ROG: Not covered) as well as potentially 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. 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 adopted in earlier lines of therapy as physician adoption mirrors Rituxan'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 and anticipate an annual course of therapy could cost $110,000, assuming a one year of therapy (average duration of current therapies for ALL is estimated to be 1.5-3 years).

We estimate that if approved in ALL, MOR208 could achieve peak sales in excess of $600M. We estimate 5,500 ALL patients are diagnosed in the US each year. 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 one year of therapy and $110,000 annual cost of therapy. Based upon a 15x multiple, 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.

Exhibit 43: MOR208 Sales Projections and Royalties to Xencor



Source: Oppenheimer & Co. Inc.

Xencor's Fc Domains—Building Blocks of Engineered Immunotherapy

Xencor leverages its patented three-dimensional structure screening technology to explore small mutations that alter the Fc domain to modulate or augment antibody performance. Xencor has discovered a set of Fc domains that augment naturally occurring antibody functions by enhancing ADCC, inhibiting the immune system or extending the half-life.

Improving Response to Antibody Therapy by Enhanced ADCC

Xencor has discovered two point mutations in the Fc domain of a human antibody, S239D and I332E, that enhance the affinity of the Fc region of the antibody for the activating receptors FcγRIIIa, on NK cells and T cells, and FcγRIIa on macrophages. These mutations can increase ADCC by 20-fold compared to unmodified antibodies.

There are two alleles of the FcγRIIIa receptor, 158V and 158F. The 158V allele binds to the Fc domain of antibodies with higher affinity than the 158F allele. Patients homozygous for the 158V allele (15-20% of population) have significantly improved responses to antibody therapy (Mellor 2013, Musolino 2008). Importantly, Xencor's mutations increase the 158F/XmAb binding affinity by ~20x to an affinity ~10x fold greater than the 158V/IgG affinity

Exhibit 44: Relevant ADCC Enhancing Technologies

	Xencor	MacroGenics	Roche	Kyowa Kirin Hakko	Glycotope
Mutations / Changes	S239D, I332E	L235V, F243L, R292P, Y300L, P396L	Afucosylated Ab	Afucosylated Ab	"Glyco-optimization"
Name	XmAb™	Fc optimization	GlycoMab™	POTELLIGENT®	GlycoExpress™
Technology	Amino acid mutations	Amino acid mutations	Overexpression of GnT-III and rMan-II enzymes	FUT8 KO CHO line	Expression of various glycosylation proteins
Compounds in Clinical Development (Target)	XmAb5574/MOR208 (CD19)	Margetuximab (HER2) MGA271 (B7-H3)	Gazyva (CD20) GA-201 (EGFR) RG-7116 (HER3)	Mogamulizumab (CCR4) Medi-551 (CD19) MDX-1342 (CD19) Benralizumab (Il-5R)	CetuGEX (EGFR) TrazGEX (HER2)
Most advanced phase	Phase II	Phase II	Approved	Approved in Japan, Phase II US	Phase IIb

Source: Oppenheimer & Co. Inc.

FcγRIIb—A Versatile Immune Inhibitory Antibody Receptor

Xencor has developed Fc domains that selectively bind the inhibitory FcγRIIb receptor on B-cells (BCR). This receptor functions naturally in a feedback inhibition loop to halt the production of antibodies. FcγRIIb is recruited to the BCR, where it inhibits BCR signaling and inactivates B-cells. Importantly, this happens only when FcγRIIb is in proximity to the BCR. This means that antibodies with this immune inhibitor domain will not non-specifically inhibit B-cells, but must be targeted to a BCR-associated protein. Xencor has exploited this technology in XmAb5871 by incorporating an immune inhibitor domain in the FC region of an antibody directed against CD19, a protein associated with the BCR (details, p. 36).

FcRn—Xtend Technology for Longer Half-Life

Xencor has developed antibodies with 3-fold half-life improvements by specifically increasing the affinity of the Fc region for the FcRn receptor in acidic environments. FcRn receptors, particularly on liver sinusoidal cells (LSECs), salvage antibodies that are pino- or endo-cytosed, recycling them from the acidic lysosome to the surface of the cell where they are released back into the bloodstream. Xencor's technology increases the efficiency of recycling, resulting in an increase in half-life and potentially higher serum concentrations of antibody. Xencor has been able to increase the half-life by up to 4x over conventional antibodies through this modification.

FC Knock-Out Domains for Bispecifics

Xencor has also developed knockout Fc domains, Fc-KO, that do not bind Fc receptors on immune cells, inhibiting the normal antibody-mediated immune response, while maintaining IgG-like half-lives. This domain is incorporated in Xencor's bispecific antibodies, and can be used to mitigate the risk of potential side effects such as cytokine storm.

Licenses, Collaborations and Intellectual Property

Xencor's technology and approach to antibody engineering have been validated through several significant partnerships that may 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 nine partnered products with seven companies, and four of these partnered products are currently in clinical trials. We believe that Xencor's strategic partnering of its product pipeline selectively diversifies clinical, regulatory and commercial risk while offering non-dilutive capital to further develop internal candidates.

Exhibit 45: Xencor Is Eligible for Up to $1.3B in Milestone Payments

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

Source: Xencor, Inc

Collaborations

Amgen—XmAb5871

In December 2010, Xencor entered into a collaboration and option agreement with Amgen, Inc. pursuant to XmAb5871. Under the terms of the agreement, Xencor granted to Amgen an exclusive license to research and commercialize XmAb5871 and certain related compounds. This license is exercisable only during the option period after Amgen 1) notifies Xencor that it is exercising the option and 2) pays Xencor $50M. The option period ends on the earliest of 1) 90 days after Xencor delivers a clinical trial report package from the Phase II POC clinical trial to Amgen; 2) termination of the agreement; 3) March 23, 2017 or March 23, 2012 if Amgen exercises an option to take over development for Xencor's failure to meet contractual obligations.

Xencor is eligible for milestone payments of up to $437M and tiered royalties in the high single-digit to high-teens range until the latest of last-to-expire patent expires or 10 years following first commercial sale on a country-by-country basis.

MorphoSys—MOR208

In June 2012, Xencor entered into a collaboration agreement with MorphoSys, amended in March 2012. Under the agreement, Xencor grants MorphoSys an exclusive, world-wide license to develop MOR208 and other anti-CD19 antibodies with Xencor's cytotoxic Fc domain.

Xencor is eligible for milestones payments of up to $299M and tiered royalties in the high single-digit to high-teens range until the latest of last-to-expire patent expires or 11 years following first commercial sale on a country-by-country basis.

Alexion—Xtend

In January 2013, Xencor entered into a license agreement under which Xencor granted Alexion an exclusive research license to six compounds incorporating Xtend technology. This research license is valid through the completion of the first multi-dose clinical trial. Xencor also granted Alexion an option to purchase commercial licenses on a target-by-target basis.

Xencor is eligible for up to $66.5M in milestone payments for each target as well as low single-digit royalties.

Boehringer Ingelheim—Anti-TNF Manufacturing

In February 2012, Xencor entered into a manufacturing agreement with BI pursuant to Xencor's anti-TNF compound. Under the agreement, BI will establish manufacturing processes and provide drug substance. Xencor is required to use commercially reasonable efforts to complete Phase I testing and find a partner for the product. BI will defer all payments until the signing of a partnering agreement for the compound or until February 10, 2017, if Xencor elects to develop the product without a partner. BI has a first right to negotiate to manufacture and supply drug product for further clinical testing and commercial sale.

Cook Pharmica—XmAb7195 Manufacturing

In October 2012, Xencor entered into an agreement under which Cook Pharmaica will supply cGMP XmAb7195 drug substance to Xencor. Xencor is responsible for Cook's costs plus a low double digit markup.

Catalen—XmAb5871 Manufacturing

In September 2005, Xencor entered into an agreement under which Catalent will develop a manufacturing process using its proprietary GPExgene technology and supply cGMP drug product. Xencor has an option to license any cell lines developed by Catalent for 10 years for a $0.3M up-front payment and a $30,000 annual fee.

In December 2011, Xencor bought a cell line from Catalent used for the manufacture of XmAb7195 for an up-front payment of $125,000, and royalty payments of less than 1% on net sales.

Exhibit 46: Xencor's Exlusive Collaborations and Licenses

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: Xencor, Inc

Other Collaborations and Licenses

Xencor also licenses a specific XmAb technology for a specific program outside of Xencor's core strategic focus. Xencor is eligible for royalties and milestone payments.

Exhibit 47: Xencor's Non-Exclusive Collaborations and Licenses

Licensee	Year	XmAb Technology	Indication	Latest Phase
BI	2007	Cytotoxic	Oncology	Phase I
Janssen	2009	Xtend	Autoimmune	Preclinical
CSL Limited	2009	Cytotoxic	Oncology	Phase I
CSL Limited	2013	Xtend	Hematological diseases	Preclinical
Merck	2013	Fc Optimization	Autoimmune diseases	Preclinical

Source: Xencor, Inc

Intellectual Property

Xencor owns composition of matter patents covering each of its product candidates as well as 20 issued and 44 pending US patents covering its technology platform. They also hold 53 issued and 62 pending foreign patents covering each XmAb Fc domain.

Exhibit 48: Xencor's Patents Cover Each Candidate and The Platform Technology

Drug / Platform	Patent Type	Expiration
XmAb5871	Fv Composition of Matter	2027
XmAb5871	Fc Composition of Matter	2027
XmAb5871	Method of Use	2028
XmAb7195	Fv Composition of Matter	2030
XmAb7195	Fc Composition of Matter	2027
XmAb5574	Fv Composition of Matter	2027
XmAb5574	Fc Composition of Matter	2025

Source: Xencor, Inc

Management

Bassil I. Dahiyat, Ph.D.— President and CEO

Dr. Bassil Dahiyat co-founded Xencor in 1997 with Dr. Stephen Mayo. Dr. Dahiyat is the inventor on 60 patents and co-author on 18 published scientific papers. He holds a Ph.D. in chemistry from Caltech, and BS and MSE degrees in biomedical engineering from Johns Hopkins University.

Edgardo Baracchini, Ph.D.— Chief Business Officer

Dr. Baracchini joined Xencor in 2010 as chief business officer. Previously he served as senior VP of Business Development at Metabasis Therapeutics until its merger with Ligand Pharmaceuticals. He served as VP of business development at Elitra Pharmaceuticals and as director of business development at Agouron Pharmaceuticals. Prior to Agouron, he was assistant director of business development at ISIS Pharmaceuticals. Dr. Baracchini holds a Ph.D. In molecular and cellular biology from the University of Texas at Dallas, an MBA from the University of California, Irvine, and a BS in microbiology from the University of Notre Dame.

John R. Desjarlais, Ph.D. - VP of Research

Dr. Desjarlais oversees development and research at Xencor. Previously he was an assistant professor of chemistry at Penn State University. He holds a Ph.D. in biophysics from Johns Hopkins University and a BS in Physics from the University of Massachusetts.

Paul Foster, M.D.—Chief Medical Officer

Dr. Foster joined Xencor as CMO in 2010. Previously he provided Medical/Clinical consulting services as SVP Development and was CMO of Development and Strategic Consulting Associates, LLC. He has held senior leadership positions at Biogen Idec, IDEC Pharmaceuticals, Abbott Laboratories, Alpha Therapeutics, Reata Pharmaceuticals, Cardium Therapeutics and Dade Behring. Dr. Foster received an MD from the Duke University School of Medicine and a BS in chemistry from the University of Michigan.

John J. Kuch – VP of Finance

Mr. Kuch oversees the financial reporting, budgeting and cash-flow management of Xencor, Inc. Previously he served as a director for PriceWaterhouseCoopers. Mr. Kuch holds a BS and MS in accounting from the University of Illinois and is a CPA.

Financial Model

Xencor, Inc
Ticker: XNCR (NASDAQ)
7/9/2014
Annual Financial Results and Projections
($ in thousands except per share data)

Income Statement	FY:12A	FY:13A	Q1	Q2	Q3	Q4	FY:14E	FY:15E	FY:16E	FY:17E	FY:18E	FY:19E
Revenues:												
XmAB7195 Sales	0	0	0	0	0	0	0	0	0	0	0	0
Licensing and Milestones	0	0	0	0	0	0	0	0	0	0	0	15,000
Collaboration revenue	9,524	10,172	2,184	2,184	2,184	2,184	8,736	8,736	8,736	8,736	8,736	8,736
Total Revenues	**$ 9,524**	**$ 10,172**	$ 2,184	$ 2,184	$ 2,184	$ 2,184	**$ 8,736**	**$ 8,736**	**$ 8,736**	**$ 8,736**	**$ 8,736**	**$ 23,736**
Cost and Expenses:												
Costs of goods sold	0	0	0	0	0	0	0	0	0	0	0	0
Research and Development	0	17,001	4,228	5,500	5,500	6,000	21,228	22,000	23,000	27,000	29,000	28,000
Sales, General and Administrative	9,524	3,691	1,723	1,700	1,900	2,100	7,423	9,200	10,000	12,000	12,000	12,000
Other	0	0	0	0	0	0	0	0	0	0	0	0
Total Costs and Expenses	**$ 9,524**	**$ 20,692**	$ 5,951	$ 7,200	$ 7,400	$ 8,100	**$ 28,651**	**$ 31,200**	**$ 33,000**	**$ 39,000**	**$ 41,000**	**$ 40,000**
Operating Income (loss)	**0**	**(10,520)**	(3,767)	(5,016)	(5,216)	(5,916)	**(19,915)**	**(22,464)**	**(24,264)**	**(30,264)**	**(32,264)**	**(16,264)**
Net Interest Income (Expense)	(2,450)	(1,206)	16	218	201	183	618	678	830	1,008	1,139	822
Other income / (Expense)	86	(48,532)	0	0	0	0	0	0	0	0	0	0
Income Before Income Taxes	(2,364)	(60,258)	(3,751)	(4,798)	(5,015)	(5,733)	(19,297)	(21,786)	(23,434)	(29,256)	(31,125)	(15,442)
Net Income	**$ (2,364)**	**$ (60,258)**	$ (3,751)	$ (4,798)	$ (5,015)	$ (5,733)	**$ (19,297)**	**$ (21,786)**	**$ (23,434)**	**$ (29,256)**	**$ (31,125)**	**$ (15,442)**
GAAP Net Income	**$ (2,364)**	**$ (60,258)**	$ (3,751)	$ (4,798)	$ (5,015)	$ (5,733)	**$ (19,297)**	**$ (21,786)**	**$ (23,434)**	**$ (29,256)**	**$ (31,125)**	**$ (15,442)**
GAAP Basic EPS with sFAS123	**(32.70)**	**(3.85)**	(0.12)	(0.15)	(0.16)	(0.18)	**(0.61)**	**(0.66)**	**(0.68)**	**(0.78)**	**(0.81)**	**(0.40)**
GAAP Diluted EPS with sFAS123	(32.70)	(3.85)	(0.12)	(0.15)	(0.16)	(0.18)	(0.61)	(0.66)	(0.68)	(0.78)	(0.81)	(0.40)
Weighted shares outstanding	72	15,646	31,361	31,386	31,411	31,436	31,398	32,998	34,598	37,323	38,298	38,398
Fully diluted shares outstanding	72	15,646	31,361	31,386	31,411	31,436	31,398	32,998	34,598	37,323	38,298	38,398
Cash Burn	(2,364)	(60,258)	(5,439)	(5,528)	(6,015)	(6,733)	(19,297)	(21,786)	(23,434)	(29,256)	(31,125)	(15,442)
Cash Balance	2,312	77,975	72,536	67,008	60,993	54,260	54,260	77,597	54,163	106,282	74,882	57,163

Balance Sheet	FY:12A	FY:13A	Q1	Q2	Q3	Q4	FY:14E	FY:15E	FY:16E	FY:17E	FY:18E	FY:19E
Total cash and cash equivalents	**2,312**	**77,975**	**72,536**	**67,008**	**60,993**	**54,260**	**54,260**	**77,597**	**54,163**	**106,282**	**74,882**	**57,163**
Accounts receivable	354	0	511	240	240	240	240	240	240	240	515	2,792
Prepaid Expenses and other current assets	173	119	159	159	159	159	159	159	159	159	159	159
Total current assets	2,839	78,094	73,206	67,408	61,393	54,660	54,660	77,996	54,562	106,682	75,556	60,114
Property, plant and equipment, net	283	307	340	340	340	340	340	340	340	340	340	340
Other	8,537	8,914	8,949	8,949	8,949	8,949	8,949	8,949	8,949	8,949	8,949	8,949
Total assets	$ 11,659	$ 87,315	$ 82,495	$ 76,697	$ 70,682	$ 63,949	$ 63,949	$ 87,285	$ 63,851	$ 115,971	$ 84,845	$ 69,403
Current liabilities:												
Accounts payable	1,315	4,026	2,104	2,104	2,104	2,104	2,104	2,104	2,104	2,104	2,104	2,104
Accured expenses	1,286	0	1,465	1,465	1,465	1,465	1,465	1,465	1,465	1,465	1,465	1,465
Current portion of deferred revenues	1,948	3,444	3,251	3,251	3,251	3,251	3,251	3,251	3,251	3,251	3,251	3,251
Current portion of lease obligations	7	0	0	0	0	0	0	0	0	0	0	0
Other	20,923	9	7	7	7	7	7	7	7	7	7	7
Total current liabilities	25,479	7,479	6,827	6,827	6,827	6,827	6,827	6,827	6,827	6,827	6,827	6,827
Other Non-current liabilities	5,682	6,302	5,603	4,603	3,603	2,603	2,603	295	295	295	295	295
Total liabilities	31,161	13,781	12,430	11,430	10,430	9,430	9,430	7,122	7,122	7,122	7,122	7,122
Stockholders' equity:	(19,502)	73,534	70,065	65,267	60,252	54,519	54,519	80,163	56,729	108,849	77,723	62,281
Total liabilities and stockholder's equity	$ 11,659	$ 87,315	$ 82,495	$ 76,697	$ 70,682	$ 63,949	$ 63,949	$ 87,285	$ 63,851	$ 115,971	$ 84,845	$ 69,403

Oppenheimer & Co.

Source: Company reports; Oppenheimer & Co. Inc. estimates.

Stock prices of other companies mentioned in this reports (as of 07/08/14)

Name	Ticker	Exchange	Price	Currency	Rating
AstraZeneca PLC	AZN	London	43.6	British Pounds	Not Covered
CSL Limited	CSL	ASX	68.3	Australian Dollar	Not Covered
GlaxoSmithKline plc	GSK	London	15.5	British Pounds	Not Covered
Isis Pharmaceuticals, Inc.	ISIS	NASDAQ	31.3	U.S. Dollar	Not Covered
Johnson & Johnson	JNJ	NYSE	105.7	U.S. Dollar	Not Covered
Merck & Co., Inc.	MRK	NYSE	58.2	U.S. Dollar	Not Covered
MorphoSys AG	MOR	Xetra	69.6	Euro	Not Covered
Novartis AG	NOVN	SIX Swiss	80.3	Swiss Franc	Not Covered
Roche Holding AG	RO	SIX Swiss	262.5	Swiss Franc	Not Covered
Sanofi	SAN	Euronext Paris	76.2	Euro	Not Covered
Stemline Therapeutics, Inc.	STML	NASDAQ	13.4	U.S. Dollar	Not Covered

Investment Thesis

Xencor is a biopharmaceutical company focused on developing and commercializing engineered antibody therapies to treat severe diseases with unmet medical need. Xencor's engineered Fc domains enable high-yield production of IgG-like bispecific antibodies that may enable cost-effective immunotherapy for the treatment of cancer. This potentially best-in-class platform, which has two partnered products in Phase II studies, a wholly owned candidate in a Phase I trial, five further internal early-stage programs and a total of seven collaborations is, in our opinion, the core of Xencor's current value. Merck, Boehringer Ingelheim and CSL are validating of Xencor's approach. These partnerships also provide non-dilutive capital in upfront payments and up to $1.31 billion in milestone payments as well as additional royalties (single to double-digit percentages).

Price Target Calculation

We arrive at our $22 price target by a sum of the parts analysis. We ascribe $12/share by applying a typical oncology multiple of 6x on our estimated 2022 revenues of $1.2 billion for XmAb7195 in moderate to severe asthma, discounted 45% annually. We ascribe $3/share to each of Xencor's two partnered programs XmAb5871 and XmAb5574 based on a typical multiple of 15x royalties (estimated 10%) from US & EU sales in 2022 of $900M and $600 million discounted 40% and 30% annually, respectively. We ascribe $3/share to XmAbCD123 as a surrogate for the bispecific candidate Xencor expects to advance into the clinic in 2015.

Key Risks to Price Target

These risks include: 1) failure to reach sales expectations for Xmab7195, XmAb5574/MOR208, or XmAb5871; 2) failure in the clinic of Xmab7195, XmAb5574/MOR208, or XmAb5871; 3) changes to or discontinuation of Xencor's partnerships for XmAb5574/MOR208 or XmAb5871; 4) intellectual property risk; 5) manufacturing risk; 6) competitive risk from biotech companies with more resources; 6) strategic risk; 7) the risk of a dilutive financing; and 8) insider ownership risk as approximately 35% of the shares are closely held.

Important Disclosures and Certifications

Analyst Certification - The author certifies that this research report accurately states his/her personal views about the subject securities, which are reflected in the ratings as well as in the substance of this report. The author certifies that no part of his/her compensation was, is, or will be directly or indirectly related to the specific recommendations or views contained in this research report.

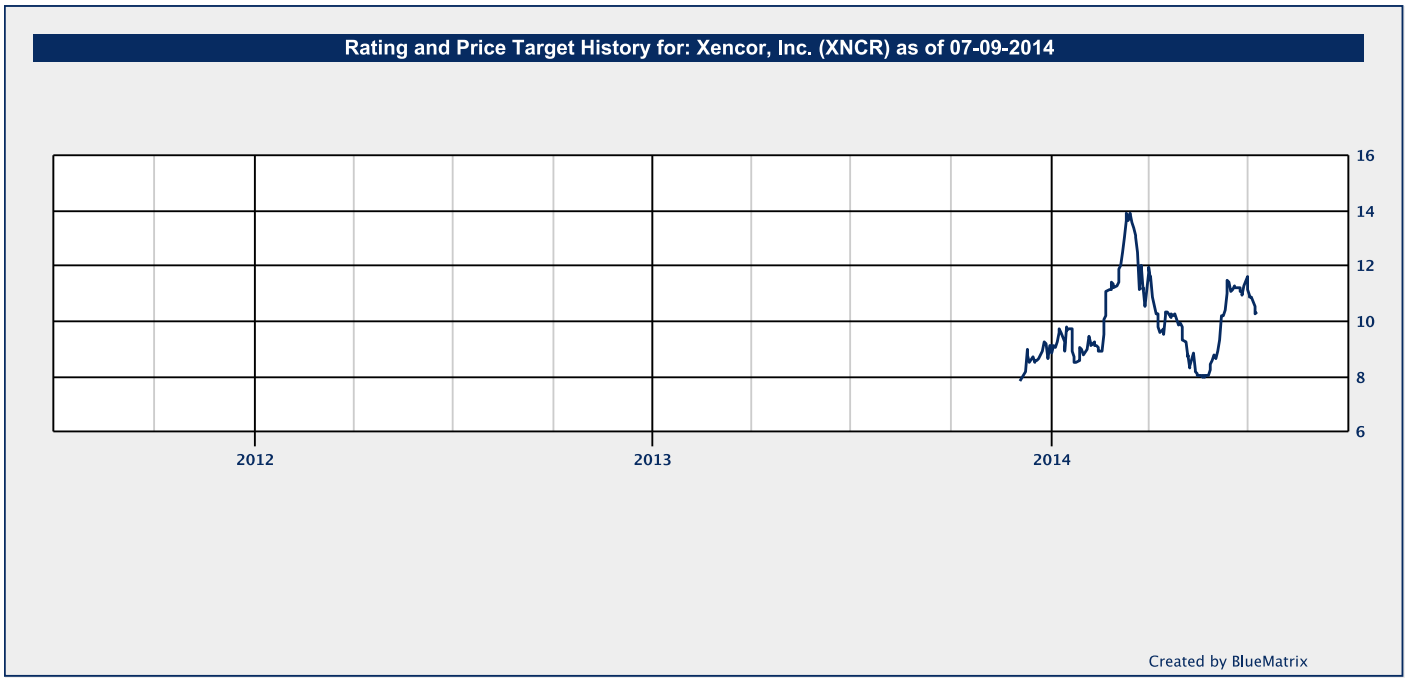
Potential Conflicts of Interest:

Equity research analysts employed by Oppenheimer & Co. Inc. are compensated from revenues generated by the firm including the Oppenheimer & Co. Inc. Investment Banking Department. Research analysts do not receive compensation based upon revenues from specific investment banking transactions. Oppenheimer & Co. Inc. generally prohibits any research analyst and any member of his or her household from executing trades in the securities of a company that such research analyst covers. Additionally, Oppenheimer & Co. Inc. generally prohibits any research analyst from serving as an officer, director or advisory board member of a company that such analyst covers. In addition to 1% ownership positions in covered companies that are required to be specifically disclosed in this report, Oppenheimer & Co. Inc. may have a long position of less than 1% or a short position or deal as principal in the securities discussed herein, related securities or in options, futures or other derivative instruments based thereon. Recipients of this report are advised that any or all of the foregoing arrangements, as well as more specific disclosures set forth below, may at times give rise to potential conflicts of interest.

Important Disclosure Footnotes for Companies Mentioned in this Report that Are Covered by Oppenheimer & Co. Inc:

Stock Prices as of July 10, 2014

Amgen Inc. (AMGN - NASDAQ, $119.57, PERFORM)
Gilead Sciences (GILD - NASDAQ, $88.68, OUTPERFORM)
ImmunoGen, Inc. (IMGN - NASDAQ, $11.23, PERFORM)
Prothena Corp (PRTA - NASDAQ, $21.09, OUTPERFORM)
Seattle Genetics (SGEN - OTC, $35.74, PERFORM)



All price targets displayed in the chart above are for a 12- to- 18-month period. Prior to March 30, 2004, Oppenheimer & Co. Inc. used 6-, 12-, 12- to 18-, and 12- to 24-month price targets and ranges. For more information about target price histories, please write to Oppenheimer & Co. Inc., 85 Broad Street, New York, NY 10004, Attention: Equity Research Department, Business Manager.

Oppenheimer & Co. Inc. Rating System as of January 14th, 2008:

Outperform(O) - Stock expected to outperform the S&P 500 within the next 12-18 months.

Perform (P) - Stock expected to perform in line with the S&P 500 within the next 12-18 months.

Underperform (U) - Stock expected to underperform the S&P 500 within the next 12-18 months.

Not Rated (NR) - Oppenheimer & Co. Inc. does not maintain coverage of the stock or is restricted from doing so due to a potential conflict of interest.

Oppenheimer & Co. Inc. Rating System prior to January 14th, 2008:

Buy - anticipates appreciation of 10% or more within the next 12 months, and/or a total return of 10% including dividend payments, and/or the ability of the shares to perform better than the leading stock market averages or stocks within its particular industry sector.

Neutral - anticipates that the shares will trade at or near their current price and generally in line with the leading market averages due to a perceived absence of strong dynamics that would cause volatility either to the upside or downside, and/or will perform less well than higher rated companies within its peer group. Our readers should be aware that when a rating change occurs to Neutral from Buy, aggressive trading accounts might decide to liquidate their positions to employ the funds elsewhere.

Sell - anticipates that the shares will depreciate 10% or more in price within the next 12 months, due to fundamental weakness perceived in the company or for valuation reasons, or are expected to perform significantly worse than equities within the peer group.

Distribution of Ratings/IB Services Firmwide

			IB Serv/Past 12 Mos.	
Rating	Count	Percent	Count	Percent
OUTPERFORM [O]	309	51.50	145	46.93
PERFORM [P]	282	47.00	100	35.46
UNDERPERFORM [U]	9	1.50	2	22.22

Although the investment recommendations within the three-tiered, relative stock rating system utilized by Oppenheimer & Co. Inc. do not correlate to buy, hold and sell recommendations, for the purposes of complying with FINRA rules, Oppenheimer & Co. Inc. has assigned buy ratings to securities rated Outperform, hold ratings to securities rated Perform, and sell ratings to securities rated Underperform.

Company Specific Disclosures

Oppenheimer & Co. Inc. makes a market in the securities of AMGN, GILD, IMGN, ISIS, MEDI, MGNX and SGEN.

Oppenheimer & Co. Inc. expects to receive or intends to seek compensation for investment banking services in the next 3 months from IMGN and SGEN.

Additional Information Available

Please log on to http://www.opco.com or write to Oppenheimer & Co. Inc., 85 Broad Street, New York, NY 10004, Attention: Equity Research Department, Business Manager.

Other Disclosures

This report is issued and approved for distribution by Oppenheimer & Co. Inc., a member of the New York Stock Exchange ("NYSE"), NASD and SIPC. This report is provided, for informational purposes only and does not constitute an offer or solicitation to buy or sell any securities discussed herein in any jurisdiction where such offer or solicitation would be prohibited.

The securities mentioned in this report may not be suitable for all types of investors. This report does not take into account the investment objectives, financial situation or specific needs of any particular client of Oppenheimer & Co. Inc. Recipients should consider this report as only a single factor in making an investment decision and should not rely solely on investment recommendations contained herein, if any, as a substitution for the exercise of independent judgment of the merits and risks of investments. Before making an investment decision with respect to any security recommended in this report, the recipient should consider whether such recommendation is appropriate given the recipient's particular investment needs, objectives and financial circumstances. Non-client recipients of this report who are not institutional investor clients of Oppenheimer & Co. Inc. should consult with an independent financial advisor prior to making any investment decision based on this report or for any necessary explanation of its contents. Oppenheimer & Co. Inc. will not treat non-client recipients as its clients solely by virtue of their receiving this report.

Past performance is not a guarantee of future results, and no representation or warranty, express or implied, is made regarding future performance of any security mentioned in this report. The price of the securities mentioned in this report and the income they produce may fluctuate and/or be adversely affected by exchange rates, and investors may realize losses on investments in such securities, including the loss of investment principal. Oppenheimer & Co. Inc. accepts no liability for any loss arising from the use of information contained in this report, except to the extent that liability may arise under specific statutes or regulations applicable to Oppenheimer & Co. Inc.

Information, opinions and statistical data contained in this report were obtained or derived from sources believed to be reliable, but Oppenheimer & Co. Inc. does not represent that any such information, opinion or statistical data is accurate or complete (with the exception of information contained in the Important Disclosures section of this report provided by Oppenheimer & Co. Inc. or individual research analysts), and they should not be relied upon as such. All estimates, opinions and recommendations expressed herein constitute judgments as of the date of this report and are subject to change without notice.

Nothing in this report constitutes legal, accounting or tax advice. Since the levels and bases of taxation can change, any reference in this report to the impact of taxation should not be construed as offering tax advice on the tax consequences of investments. As with any investment having potential tax implications, clients should consult with their own independent tax adviser.