

Xencor, Inc. (XNCR)

SMALL & MID CAP RESEARCH

Next-Generation Antibody Therapeutics

- We are initiating coverage of Xencor with an Outperform rating and a \$14 target price. The most exciting asset, in our opinion, is its proprietary Fc engineered anti-IgE antibody for asthma (XmAb7195). We also view XNCR's technology as the most diversified Fc engineering platform for next-generation antibodies and bispecifics. We expect initial proof-of-concept Phase I data for XmAb7195 in Q4:14, while progress from partnered programs could also generate value.
- **Leader in Fc Engineering:** Through Fc engineering, XNCR can modify and improve the activity of therapeutic antibodies, including better tumor killing, longer half-life, and immune down-regulation. This broad platform has generated clinical programs in lymphoma, asthma, and arthritis/lupus and supports multiple technology licenses and product deals.
- **XmAb7195 for Asthma:** Roche's Xolair is the only anti-IgE antibody for asthma, with global sales of \$1.3B. XmAb7195 is designed to be a better Xolair, with the ability to block IgE, shut off its production, and promote its clearance. We expect proof-of-concept data for this program in Q4:14.
- **XmAb5871 for RA and Lupus:** This anti-CD19 antibody program is optioned to AMGN, which mitigates late-stage risk and provides funding for near-term development. CD19 is a well validated target, but the space is crowded.
- **Valuation:** Our \$14 target price is based on a probability-adjusted DCF, assigning a 40% probability of success to XmAb7195 and a 25% probability to XmAb5871. We use a 12% discount rate through the products' lifecycles.

Rating	OUTPERFORM* [V]
Price (02 Jan 14, US\$)	8.88
Target price (US\$)	14.00 ¹
52-week price range	9.28 - 7.55
Market cap. (US\$ m)	278.23
Enterprise value (US\$ m)	212.56

*Stock ratings are relative to the coverage universe in each analyst's or each team's respective sector.

¹Target price is for 12 months.

[V] = Stock considered volatile (see Disclosure Appendix).

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Financial and valuation metrics

Year	12/12A	12/13E	12/14E	12/15E
EPS (CS adj.) (US\$)	-38.31	-1.95	-0.65	-0.64
Prev. EPS (US\$)	—	—	—	—
P/E (x)	-0.2	-4.6	-13.7	-13.8
P/E rel. (%)	-1.3	-27.5	-90.7	-101.1
Revenue (US\$ m)	9.5	10.1	4.6	7.0
EBITDA (US\$ m)	-5.7	-10.8	-20.8	-22.7
OCFPS (US\$)	-49.27	-0.27	-0.66	-0.48
P/OCF (x)	—	-32.6	-13.5	-18.4
EV/EBITDA (current)	-48.9	-25.7	-13.4	-12.3
Net debt (US\$ m)	5	-66	-45	-120
ROIC (%)	42.02	-174.30	-312.30	-327.78
Number of shares (m)	31.33	IC (current, US\$ m)		-14.83
BV/share (Next Qtr., US\$)	2.3	EV/IC (x)		32.9
Net debt (Next Qtr., US\$ m)	-65.7	Dividend (current, US\$)		—
Net debt/tot cap (Next Qtr., %)	-91.0	Dividend yield (%)		—

Source: Company data, Credit Suisse estimates

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Portfolio Manager Summary

XNCR was founded on its technology to optimize proteins, and its primary focus is to optimize the Fc region of antibodies. Modifications to the Fc can (1) enhance tumor killing or ADCC; (2) increase the half-life of antibodies, potentially reducing the dosing frequency; (3) facilitate the elimination of the target protein from the blood; and (4) specifically turn off immune reactions (a benefit in a variety of autoimmune and inflammatory diseases).

Its broad technology platform has generated a large number of clinical candidates, product collaborations, and technology licenses.

- **Proprietary Drugs:** The most exciting program, in our opinion, is the anti-IgE antibody for asthma (XmAb7195). This potentially better version of Roche's blockbuster Xolair will likely have biologic proof-of-concept data from Phase I in Q4:14. This program is owned 100% by XNCR.
- **Partnered Drugs:** MorphoSys is developing MOR208, an Fc enhanced anti-CD19 antibody for lymphoma and leukemia that is designed to have increased anti-tumor potency. It is in Phase II trials for B-cell non-Hodgkin lymphoma (NHL) and acute lymphoblastic leukemia (ALL). This deal includes milestones and high-single-digit to low-double-digit royalties.
- **Optioned Drugs:** Amgen has paid XNCR an upfront fee and ongoing milestones to retain a development option for XmAb5871 for rheumatoid arthritis (RA) and lupus. XNCR is conducting a Phase I/II trial, and AMGN will have the option to license the program after Phase IIb studies. This deal includes a significant option payment of \$50M and high-single-digit to midteens tiered royalties.
- **Technology Licenses:** Several companies, including ALXN, JNJ, MRK, CSL and Boehringer Ingelheim, have taken licenses to use XNCR's technology. Most are for the purpose of extending the half-life of antibodies. These deals typically include low-single-digit royalties.

The Antibody Investment Thesis:

We recommend investors have broad exposure to the antibody subsector of the biotechnology space to take advantage of three continuing trends: (1) antibodies have a higher clinical success rate, (2) pharmaceutical companies and some large biotechs still have a need for more biologics, and (3) next-generation technologies offer significant long-term growth.

Exhibit 1: XNCR Pipeline

Drug	Target	Technology	Indication	Stage	Partner
XmAb5574/MOR208	CD19	High ADCC	CLL, NHL, ALL	Phase II	Morphosys
XmAb5871	CD19	Immune inhibitory	Autoimmune	Phase I/II	AMGN has option
XmAb7195	IgE	Immune inhibitory	Asthma/Allergy	Phase I	Proprietary
BI 836826	CD37	High ADCC	CLL, NHL	Phase I	Boehringer Ingelheim
BI 836858	CD33	High ADCC	AML	Phase I	Boehringer Ingelheim
CSL362	IL3Ra	High ADCC	AML	Phase I	CSL
Xtend-TNF	TNF	Long half-life	Autoimmune	Preclinical	Proprietary
CD3 X CD38	CD38	Bispecific	Oncology	Preclinical	Proprietary
CD3 X CD123	CD123	Bispecific	Oncology	Preclinical	Proprietary
Xtend-CTLA4	CTLA4	Long half-life	Autoimmune	Preclinical	Proprietary
Anti-X/ CD32b	ND	Immune inhibitory	TBD	Discovery Lead	Proprietary
ND	ND	Long half-life	Hematology	Preclinical	CSL
ND	ND	Long half-life	Autoimmune	Preclinical	Janssen
ND	ND	Stability	Autoimmune	Preclinical	Merck
ND	ND	Long half-life	Undisclosed	Discovery Lead	Alexion

Source: Company data, Credit Suisse Research.

Exhibit 2: XNCR News Flow

Timing	Expected News Flow	Program
Early 2014	Announce proprietary program to move forward	TBD
Early 2014	Phase I initiation	XmAb7195
H2:14	Phase IIa activity data	XmAb5871
Q4:14	Phase Ia data in healthy volunteers (includes high IgE cohort)	XmAb7195
Q1:15	IND for proprietary program	TBD
Q1:15	Start Phase IIb (150-250 pts)	XmAb5871
Q1:15	Phase Ib start	XmAb7195
late 15/ early 16	Start Phase II in poorly controlled asthmatics	XmAb7195
late 16/ early 17	Phase IIb data/ AMGN option	XmAb5871

Source: Company data, Credit Suisse Research.

Investment Positives

- **Leader in Fc Engineering:** XNCR owns three technologies for improving antibody function. XNCR has significant IP protection around amino acid modification of the antibody Fc region, which requires many companies to seek licenses.
- **Immune Down-Regulation Is a Unique Technology:** XNCR is pursuing two opportunities to validate this technology with XmAb7195 (blocking IgE-producing cells) and XmAb5871 (blocking CD19-positive B-cells). Both of these antibodies can actively down-regulate the immune system, with limited cytotoxicity. Current antibody technologies block the antigen but do not send an inhibitory signal.
- **XmAb7195 Is Likely a Better Xolair:** IgE is well validated target (target for Xolair), which significantly derisks the opportunity from a clinical, regulatory, and commercial perspective. XmAb7195 is significantly more potent than Xolair because it inhibits/ down-regulates IgE by three distinct mechanisms. Phase I data are likely to provide strong proof of concept. (IgE levels are a proven surrogate for activity/ clinical benefit).
- **XNCR Has Partnered Programs with Highest Clinical/Competitive Risks:** We believe that XNCR has taken a smart approach in partnering the more expensive and higher-risk programs. In particular, the anti-CD19 programs in lymphoma/leukemia (MOR208) and autoimmune disease (XmAb5871) are very highly competitive markets. NHL/CLL are currently dominated by Rituxan (and soon Gazyva) and a number of new oral drugs. RA is currently dominated by a number of effective biologics and potentially new oral agents in the future.
- **Technology Licensees Provide Additional Support to Fund Proprietary Development:** The technology licenses have simply provided access to patented mutations. There is no significant opportunity cost to XNCR or significant transfer of technology.
- **Broader Platform Includes Bispecific Antibodies:** XNCR is expected to announce the pursuit of new clinical development of one to two bispecific programs in 2014. These antibodies are expected to be less toxic and easier to administer than BiTE technologies, due to their enhanced PK profile.

Investment Risks

- **Clinical Risk Is Higher for CD19 Program in Cancer Because of High Bar for Success:** The MOR208 program will be competing with anti-CD20 antibodies (Rituxan and Gazyva) and with new oral agents for lymphoma/leukemia (e.g., Imbruvica), which

have already shown good safety and efficacy data. In addition, other agents in development targeting CD19 could be more potent because of antibody-drug conjugate technology.

- **Clinical Risk Is Higher for CD19 Program in Autoimmune Diseases Because of High Bar for Success:** The autoimmune field for RA is fairly crowded with agents that have shown good efficacy and safety, including anti-TNF agents, anti-CD20 agents, and new orals for RA.
- **IgE Program Is Very Early Stage:** Toxicity or immunogenicity could emerge in Phase I, which could reduce the benefit/risk ratio for this program. Given the large market in asthma, safety is key for any new product.
- **Upside Is Limited by AMGN Option:** The terms of a licensing deal are already agreed, so XNCR cannot benefit if the licensing environment for the drug improves due to competitive forces or better-than-expected clinical data.
- **Enhanced ADCC Antibodies May Not Be as Potent as Antibody-Drug Conjugates:** Several of the targets in XNCR's pipeline include targets also being pursued by IMGN and SGEM using potent antibody-drug conjugate technology. SGEM has an anti-CD19 program and an anti-CD33 program. IMGN has an anti-CD19 program with Sanofi.
- **Financing Risk:** XNCR will need to raise additional capital. We project another capital raise by YE:15.

Valuation

Our valuation is supported by a DCF using probability-weighted sales estimates for XmAb7195 (40%; \$9/share) and XmAb5871 (25%; \$3/share) modeled through the expected life cycle for the products. We assign \$1/share to the MOR208 program partnered with MorphoSys (35% probability of success) and \$1/share to the other licenses (10% probability of success), which could emerge as important value drivers in the future. We use a 35% tax rate and a 12% discount rate and arrive at a target price of \$14.

The biggest levers in our valuations are the following:

- (1) **Probability of Success:** We assign a 40% probability of success to XmAb7195 and a 25% probability to XmAb5871. We assign a lower probability to XmAb5871 due to the greater competitive risk.
- (2) **Pricing of XmAb7195:** We model a net price of \$1,250/month. We believe this is a conservative estimate, given the current Xolair price of \$777 for the 150mg vial (dosed 150-300mg one to two times per month). If XmAb7195 demonstrates superior efficacy and is dosed with a simpler dosing regimen as expected, we believe XmAb7195 could be priced at a meaningful premium to Xolair.
- (3) **Timing of U.S. and EU Approvals:** We assume a U.S. launch for XmAb7195 and XmAb5871 in 2022. We expect that XmAb7195 will require clinical trials that will take significant time. However, if the efficacy is compelling in earlier studies and/or XNCR elects to pursue any underserved populations, XNCR may be able to file for approval ahead of our expectations.
- (4) **Introduction of New Pipeline Program(s):** XNCR is expected to announce a new bispecific pipeline candidate in 2014 that is produced via an optimized manufacturing process. We do not include this in our valuation. This program could add long-term value to the stock if it proves success in validated preclinical models/early-stage clinical programs.

- (5) **We Assume AMGN Exercises Its Option on XmAb5871:** AMGN has an option to the XmAb5871 program following the Phase IIb data release (study expected to start in early 2015). Any change to this deal (i.e., AMGN not exercising the option) could impact our valuation of the program by adding expenses and reducing near-term cash inflows.

Exhibit 3: Probability-Adjusted Valuation

Program	NPV	POS	per share
XmAb7195	\$288	40%	\$8.8
XmAb5871	\$98	25%	\$3.0
MOR208	\$42	35%	\$1.3
Other licenses	\$22	10%	\$0.7

Total	\$450	\$14
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Source: Company data, Credit Suisse estimates.

Exhibit 4: XNCR Earnings Model

	2012A	2013E	2014E	2015E	2016E	2017E	2018E	2019E	2020E	2021E	2022E
Revenues											
US sales of XmAb7195											26.9
Ex-US royalties on XmAb7195											1.0
Royalties on XmAb5871											50.0
Partnering, grants, milestones	9.5	10.1	4.6	7.0	11.0	26.1	15.0	20.0	20.0	50.0	50.0
Total Revenues	9.5	10.1	4.6	7.0	11.0	26.1	15.0	20.0	20.0	50.0	77.9
Expenses											
Cost of goods											3.2
Research and development	12.7	17.3	18.4	21.7	28.5	31.4	39.3	40.6	42.0	47.0	46.7
Sales, general, administrative	3.1	4.1	7.1	7.9	8.7	9.9	14.3	14.6	19.0	28.0	30.8
Total Operating Expenses	15.8	21.4	25.5	29.6	37.2	41.3	53.6	55.2	61.0	75.0	80.7
Operating income (loss)	(6.2)	(11.3)	(20.8)	(22.7)	(26.3)	(15.2)	(38.6)	(35.2)	(41.0)	(25.0)	(2.8)
Total Other Income (Expense)	(2.4)	(49.7)									
Pre Tax Income	(8.6)	(61.0)	(20.8)	(22.7)	(26.3)	(15.2)	(38.6)	(35.2)	(41.0)	(25.0)	(2.8)
Income tax											
Net Income	(8.6)	(61.0)	(20.8)	(22.7)	(26.3)	(15.2)	(38.6)	(35.2)	(41.0)	(25.0)	(2.8)
EPS - diluted (proforma)	(\$38.31)	(\$1.95)	(\$0.65)	(\$0.64)	(\$0.61)	(\$0.33)	(\$0.77)	(\$0.58)	(\$0.64)	(\$0.37)	(\$0.04)
Shares outstanding - basic (proforma)	0.22	31.33	32.12	35.18	43.02	46.56	50.35	60.87	63.91	67.11	70.46
Shares outstanding - diluted (proforma)	0.22	32.61	33.47	36.63	44.59	48.26	52.19	62.80	65.94	69.24	72.70

Source: Company data, Credit Suisse estimates

Key Modeling Assumptions

XmAb7195: Asthma Market

We model first XmAb7195 sales in 2022 and peak share in 2027. Our model projects \$496M (probability adjusted) in U.S. sales at a peak penetration of approximately 18%; ex-U.S. sales in that year are estimated at \$443M (probability adjusted). Sales continue to grow through price and market growth through 2031. Our model starts with a projection of the total severe refractory, perennial asthmatic population and an estimated penetration rate for XmAb7195. Several of these assumptions have built-in conservative estimates.

- **Pricing:** We assume that XmAb7195 is priced at \$1,250 per month (increases 2% per year thereafter). Currently, average pricing is almost \$2,000 per month for Xolair, so we believe that our pricing assumptions are conservative.

- **Market Size:** We used a starting patient population of 375,000 severe refractory, perennial asthmatics in the U.S. in 2013 (growing at 2%). We assume XmAb7195 reaches a peak penetration of 18% in 2027 (\$517M U.S. sales). This assumes ~90K patients on therapy in 2027, which we believe is reasonable/conservative, given there are currently 60K patients on Xolair. We assume sales growth through 2031 when the first patent expires and -50% terminal growth after that. We assume that Xencor markets the drug in the U.S. by itself and that XNCR finds an ex-U.S. partner. We assume ROW milestones and a 15% royalty rate.
- **Risk-Adjusted Market Model:** We assume a 40% probability of success. To account for this POS, we adjusted expected costs (SG&A, COGS, R&D) by 40% at the launch. Prior to the launch in 2022, development costs are probability-adjusted, dropping each year. While this probability of success is relatively high for a Phase I asset with no/minimal clinical data, we believe that the program has lower risk due to the target being well validated.

XmAb5871: Autoimmune Market

We model first XmAb5871 sales in 2022 and peak share in 2027. Our model projects \$2.0B in U.S. sales at a peak penetration of approximately 25% for TNF-refractory RA and 20% for lupus; ex-U.S. sales in that year are estimated at \$1.7B. Sales continue to grow through price increases and market growth through 2030. Our model starts with a projection of the total TNF-refractory RA population and the lupus population with a definitive diagnosis. Several of these assumptions have built-in conservative estimates.

- **Pricing:** We assume initial net pricing of \$2,400 per month in 2022. The pricing is in-line with other biologic therapies for RA and lupus.
- **Royalties:** Our model assumes tiered net royalties (high single digits to midteens) and milestones from AMGN. We assume minimal upfront development costs, as AMGN is covering most of the program's early costs through development and regulatory milestones.
- **Market Size:** We assume XmAb5871 gets approval in both RA and lupus. We assume a current TNF-refractory RA population of 60,000 in the United States, growing at 2% annually. We assume 160,000 patients with a definitive lupus diagnosis in the United States. We assume a maximum penetration of 25% in the TNF-refractory RA patients and 20% in lupus patients with a definitive diagnosis.
- **Risk-Adjusted Market Model:** We assume a 25% probability of success, which is applied to the NPV of the unadjusted royalty stream. If AMGN decides to not exercise the option after Phase IIb, there is minimal financial downside for XNCR, since AMGN will be covering most of the costs up until the option exercise.

Valuing the Partnerships

We estimated the value of the partnered programs by calculating the NPV of probability-adjusted expected milestones (development, regulatory, and sales) and royalty streams for each program. We assumed a 12% discount rate.

MorphoSys (MOR 208)

- **Milestones:** We assumed probability-adjusted development and regulatory milestones for multiple oncology indications (DLBCL, fNHL, and ALL); we have not included milestones for CLL. We do not assume any milestones or royalties for non-oncology indications. We have modeled the probability of success for milestones decreasing each year, from 80% in 2015 down to 35% in 2018 (expected Phase III readouts).

- For dosing in Phase III, we assumed approximately \$27.5M in milestones across three indications: DLBCL, fNHL, and ALL. We assume a Phase III start for ALL in 2015, with an 80% probability, and a Phase III start for DLBCL and fNHL in 2016, with a 75% probability.
 - Milestones for BLA filing mirror the Phase III milestone structure. We assume BLA filing in 2018 for ALL and 2019 for fNHL and DLBCL, with a 35% probability.
 - Milestones for BLA approval are assumed to be double the Phase III/ filing milestone payments. We assume 35% POS for approval in DLBCL, fNHL, and ALL in 2021.
- **Royalty:** We assume a 35% probability of approval in 2021 and peak sales of \$600M in 2029 (end of the royalty stream). We assumed a high-single-digit to low-teen royalty.

We calculate a total value for the MorphoSys program of \$42M (~\$1 per share), based on this method.

Other Partnered Programs

- **Milestones:** For the remaining programs (Alexion, Boehringer Ingelheim, CSL, and Janssen), we probability adjusted the total expected milestones. We assumed a 30% POS for development milestones to be received in ~4 years (2018), 20% POS for regulatory milestones to be received in ~7 years (2021), 8% POS for sales milestones to be received in ~12 years (2026).
- **Royalty:** For the royalty stream, we assume a 2% royalty rate, \$500M peak U.S. sales (\$1B WW), with sales beginning in 2023 and royalties ending in 2032. We assumed a 10% POS for five programs. (We assumed that partners with multiple programs, such as Alexion, will pick only one to move forward.)

We calculate a total value for the remaining partnered programs of \$22M (~\$1 per share).

Exhibit 5: Milestone Summary for Partnered Programs

Agreement	Develop	Regulatory	Sales	Royalties	Total
Alexion (X6)	8.5	28	30		66.5
BI (X2)	9	6	12		27
CSL 2009	6	4	5		15
CSL 2013	8	4	6.5		18.5
Janssen	6	0	4		10
Total	37.5	42	57.5		
Number of periods	4	7	12		
Probability of success	30%	20%	8%		
NPV	\$8	\$4	\$1	\$13	\$27
				per share	\$0.82

Source: Company data, Credit Suisse estimates.

The Antibody Investment Thesis

We recommend investors have broad exposure to the antibody subsector of the biotechnology space to take advantage of three continuing trends: (1) a higher clinical success rate, (2) industry consolidation by pharma and big biotech needing more antibodies and (3) next-generation technologies.

Own the Group: Investors have and we believe will continue to benefit from broadly owning the antibody sector. We favor companies with large pipelines, substantial funding from partners, and strong proprietary technologies. Among the emerging technologies, we like antibody-drug conjugates, Fc enhanced antibodies, and bispecifics.

Antibody Companies Have a Good Track Record for Investors: Upside as standalone companies – ALXN, REGN, SGEN, etc. – and premium take-outs – DNA, IMCL, FACT, MEDX, HGSI, etc. – have been the historic norm among antibody companies. Our top two pure plays are REGN and SGEN, but we continue to recommend that investors own a basket of these stocks including the emerging smaller-cap companies (See Exhibit 6).

- **Higher Success Rates:** The success rate at each stage of development is higher with antibodies than with traditional pharmaceuticals. Higher specificity, lack of active metabolites, predictable drug properties, and a growing use of companion diagnostics have and will likely continue to make antibody drug development lower risk.
- **History of Acquisitions:** Pharma and large-cap biotech have a history of acquiring antibody companies. Companies with technology platforms and retained rights are likely the most attractive targets. XNCR has a strong IP portfolio in Fc engineering and several wholly owned assets, including XmAb7195 and several preclinical bispecific programs.
- **Growing Focus on Next-Generation Drugs:** Ultra-potent antibodies, including higher affinity antibodies or technologies to increase antibody efficacy, are proliferating. Increased affinity is leading to less frequent dosing in some cases (a competitive advantage for injectables), lower cost of goods, and new market opportunities. Antibody-drug conjugates and Fc engineered antibodies have the potential to increase efficacy for cancer treatments and reduce systemic toxicity.

Exhibit 6: Universe of Antibody Companies

(\$ in MM, except per share price)						
Company	Ticker	Price 01/02/14	Stage of Development	Market cap.	CS Rating	TP
US Large cap						
Amgen	AMGN	\$115.80	Marketed	\$87,324	NEUTRAL	\$125
Biogen Idec	BIIB	\$280.33	Marketed	\$66,218	OUTPERFORM	\$375
Regeneron	REGN	\$274.59	Marketed	\$27,299	OUTPERFORM	\$340
Alexion	ALXN	\$133.44	Marketed	\$26,164	NEUTRAL	\$119
US SMID cap						
Seattle Genetics	SGEN	\$40.18	Marketed	\$4,921	OUTPERFORM	\$46
Celldex	CLDX	\$24.61	Phase III	\$2,168		
Immunogen	IMGN	\$14.98	Marketed	\$1,276	NEUTRAL	\$14
Dyax Corp.	DYAX	\$7.60	Marketed	\$920		
Oncomed	OMED	\$29.32	Phase I/II	\$818		
Emergent BioSolutions	EBS	\$24.02	Marketed	\$871		
MacroGenics	MGNX	\$28.83	Phase I	\$721		
XOMA Corporation	XOMA	\$6.92	Phase III	\$644	OUTPERFORM	\$8
Merrimack	MACK	\$5.59	Phase II	\$571		
Immunomedics	IMMU	\$4.91	Phase II	\$408		
Xencor	XNCR	\$8.88	Phase II	\$261	OUTPERFORM	\$14
Fiveprime	FPRX	\$15.96	Phase Ib	\$256		
KaloBios	KBIO	\$4.39	Phase III	\$139		
Median				\$721		
Ex-US listed						
Genmab A/S (Denmark)	GEN-DK	\$39.83	Phase III	\$11,138		
MorphoSys AG (Germany)	MOR-DE	\$78.06	Phase II	\$1,493		
Ablynx N.V. (Belgium)	ABLX-BE	\$9.74	Phase II	\$348		
BioInvent (Sweden)	BINV-SE	\$0.55	Phase II	\$302		
Median				\$920		

Source: Company data, Credit Suisse estimates.

Fc Engineering

What Is It and Why Is It Important?

Antibodies are complex proteins that are generally described by two functional domains: the variable region that binds the target (there are two of these regions on the antibody) and the Fc domain that provides the antibody with many of its desirable drug-like properties.

The Fc domain interacts with a variety of receptors in the body to mediate different reactions. Mutations in the Fc domain can enhance or inhibit these interactions, and XNCR has the most comprehensive portfolio of patented and described mutations.

XNCR's technology involves one or a small number of changes in the amino acid sequence of the Fc region. Once described, these modified Fc domains can be used in any number of antibodies (regardless of target binding) to provide enhanced functionality. These Fc domains have been licensed to partners for specific uses, at no cost to Xencor, providing added proof of concept and non-dilutive funding.

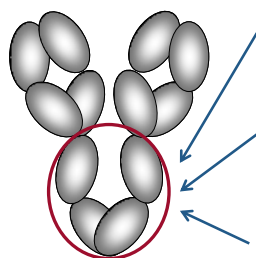
The three main functions that Xencor's Fc domains can address are:

- Enhanced antibody-dependent cellular cytotoxicity (ADCC),
- Prolonged half-life (Xtend technology), and
- Immune down-regulation / target clearance.

Xencor is also using its Fc engineering technology to facilitate the development of a proprietary class of bispecific antibodies.

Exhibit 7: Functional Modifications to the Fc Domain

This end binds the target



XmAb®
Fc domains with
patented mutations
and enhanced
function

- 1) **Immune modulation** – Can shut down specific immune functions. Applications in RA, lupus, asthma, etc.
- 2) **Enhanced antibody dependent cell-mediated cytotoxicity (ADCC)** – Proven method for improving cancer therapeutics. Recent approval of Roche's Gazyva (GA-101) provides POC.
- 3) **Increased half-life (Xtend)** – Longer half-life = less frequent injections. Many partnering opportunities for extending antibody franchises or extending into new areas. (Examples: deals with CSL, Janssen, Alexion)

Source: Company data, Credit Suisse Research.

Exhibit 8: Technology-Generated Pipeline

Technology	Fc Receptor	Drug	Stage	Partner
High ADCC	Fc γ RIIa	XmAb5574/MOR208	Phase II	Morphosys
		BI 836826	Phase I	Boehringer Ingelheim
		BI 836858	Phase I	Boehringer Ingelheim
		CSL362	Phase I	CSL
Immune inhibitory	Fc γ RIIb	XmAb5871	Phase I/II	AMGN has option
		XmAb7195	Phase I ready	Proprietary
		Anti-X/ CD32b	Discovery Lead	Proprietary
Long half-life	FcRn	Xtend-TNF	Preclinical	Proprietary
		Xtend-CTLA4	Preclinical	Proprietary
		ND	Preclinical	CSL
		ND	Preclinical	Janssen
		ND	Discovery Lead	Alexion
Stability		ND	Preclinical	Merck
Bispecific		CD3 X CD38	Preclinical	Proprietary
Bispecific		CD3 X CD123	Preclinical	Proprietary

ND not disclosed. Source: Company data, Credit Suisse Research.

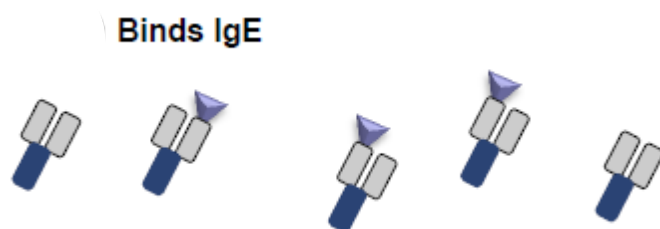
Immune Down-Regulation (and Target Clearance)

The ability to directly down-regulate the immune system is unique to XNCR's technology and is the source of both its proprietary XmAb7195 program (anti-IgE) and its XmAb5871 program (anti-CD19) optioned to AMGN.

The technology works in two steps:

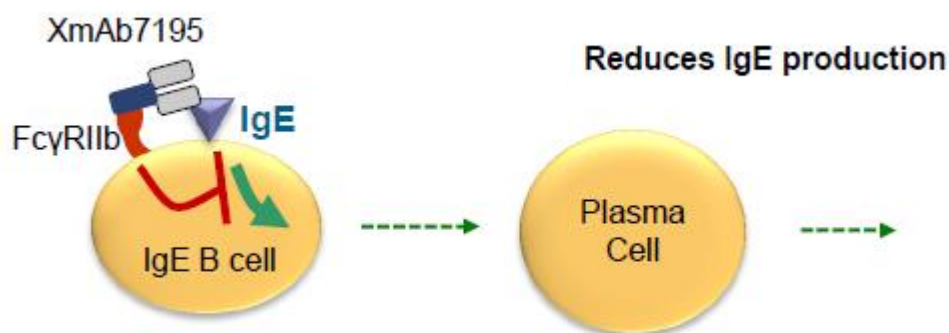
- 1) The antibody binds to its target. For XmAb5871, the target is CD19, which is expressed on nearly all B-cells. For XmAb7195, the target is IgE, which is both soluble and expressed on the surface of the cells that produce it.

Exhibit 9: Target Binding



Source: Company data, Credit Suisse Research.

- 2) Once the antibody is bound to the target (IgE), the modified Fc domain of the antibody binds tightly to the Fc γ RIIb receptor on B cells (also known as CD32b). This interaction (binding to both the target and the Fc receptor at the same time) sends an inhibitory signal to the immune cell and turns it off without killing the cells. In the case of XmAb7185, this process sends the signal not to differentiate into IgE-producing cells.

Exhibit 10: Turning Off IgE Production

Source: Company data, Credit Suisse Research.

Immune Down-Regulation

The mechanism of XmAb7195 contrasts with Xolair, which can block soluble IgE, but does not prevent production of IgE. For this reason, Xolair's activity is limited. XmAb7195 both sequesters soluble IgE and turns off the cells that produce it, providing a double hit to this disease-causing pathway.

This immune down-regulation has potential safety advantages, given that it is:

- Target specific,
- Non-depleting/ cytotoxic, and
- Reversible.

For example, XmAb5871 may have advantages over Rituxan because it does not kill/deplete the B-cells. Once the drug is removed, B-cell activity recovers. This may reduce the risk of infections and long-term immune suppression.

Exhibit 11: Advantages of Immune Down-Regulating Antibodies

Drug	Target	Indication(s)	Stage	Potential advantage
XmAb5871	CD19	Arthritis, lupus	Phase I/II	Non-depleting inhibition of B-cells
XmAb7195	IgE	Asthma	Phase I ready	blocks IgE production and increases IgE clearance
Anti-X/ CD32b	ND	ND	Discovery Lead	ND

ND not disclosed. Source: Company data, Credit Suisse Research.

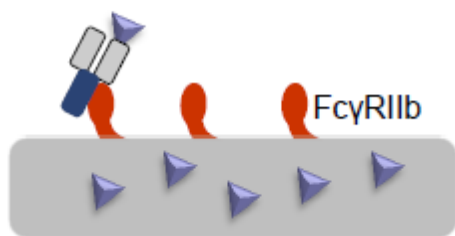
Target Clearance

The Fc γ RIIb receptor is also located on liver sinusoidal endothelial cells. When the antibody binds its soluble target (for example soluble IgE), the enhanced Fc binding to Fc γ RIIb facilitates the removal of the target from the bloodstream.

This function is particularly useful for drugs that target soluble factors (such as IgE). Binding to the target may inhibit its activity, but if the drug does not remove the target from the blood, then it will only be active when sufficient levels of the antibody are present. By eliminating the target, the antibody can have a longer-lasting effect. This is even more pronounced when the immune down-regulating activity is also employed to inhibit the production of the target.

Exhibit 12: IgE Clearance Mechanism

**Sends IgE to liver sinusoidal
endothelial cells for destruction**



Source: Company data, Credit Suisse Research.

Enhanced ADCC

The most widely explored and clinically validated function of the Fc is the ability to direct the cell-killing machinery of the immune system to eliminate the target cell in a process called ADCC. Antibodies with enhanced ADCC hold the promise of being superior anti-cancer antibodies compared to unmodified antibodies. Competitors are taking one of two approaches to modifying the Fc region in order to enhance effector function.

- **Mutations Can Be Made to the Protein Sequence:** Xencor and MacroGenics both use this method. One advantage of this approach is that no changes are needed to current manufacturing practices, and the implementation of the technology is relatively straight forward. Protein modification also has the advantage that multiple Fc functions can be altered simultaneously.
- **Changes in Glycosylation Can Enhance or Disrupt Effector Function:** Antibodies lacking a specific sugar called fucose have enhanced ADCC. Companies such as Glycart (acquired by Roche) and GlycoFi (acquired by Merck) engineered yeast to manufacture antibodies lacking fucose. BioWa (division of Kyowa Hakko) uses a modified mammalian cell line that naturally enriches in afucosylated forms. The advantage of the BioWa system is that it employs a more standard manufacturing system compared to the yeast products. A third approach in development at SGEN employs a proprietary inhibitor of the enzyme required for adding fucose to antibodies. The inhibitor is added to the production reaction for an antibody, generating afucosylated antibodies.

The role of Fc gamma receptor binding in enhanced ADCC function has been genetically and clinically validated for the treatment of non-Hodgkin lymphoma.

- **Genetic Validation:** Individuals naturally have one of three forms of the Fc gamma receptor. Individuals with the form that binds Fc with the highest affinity also have the best clinical response to the antibody Rituxan (anti-CD20), whose anti-cancer activity is believed to include ADCC.
- **Clinical Validation:** Roche (through its acquisition of Glycart) developed an enhanced anti-CD20 antibody called Gazyva. The antibody was designed to have enhanced ADCC through glycoengineering. In a Phase III trial, Gazyva demonstrated a statistically significant and clinically meaningful improvement in PFS (of almost one year) over Rituxan in patients with newly diagnosed CLL.

Given the number of approaches to enhance ADCC, the industry pipeline is quite large, and we show large sample of these in Exhibit 13.

Exhibit 13: Industry Pipeline of High ADCC Fc Enhanced Antibodies (Partial List)

Antibody	Company	Target	Stage	Technology source	Technology	Indication
Arzerra (ofatumumab)	GlaxoSmithKline	CD20	Marketed	Genmab	Increased CDC	CLL
Gazyva (obinutuzumab)	Roche	CD20	Marketed	Glycart	High ADCC	CLL
Mogamulizumab (KW-0761)	Kyowa Hakko Kirin	CCR4	Phase III	BioWa	High ADCC	CTCL
Ocaratuzumab (AME-133v)	Metrick Biotech	CD20	Phase I	AME	High ADCC	NHL
MEDI-551	MedImmune	CD19	Phase II	BioWa	High ADCC	CLL, DLBCL, MS
MGH22	MacroGenics	Her2	Phase II	MacroGenics	High ADCC	Her2 positive tumors
MOR208/XmAb5574	MorphoSys	CD19	Phase II	Xencor	High ADCC	NHL, ALL
KHK4563	Kyowa Hakko Kirin		Phase II	BioWa	Potelligent	Asthma
XmAb5871	Xencor	CD19/CD32	Phase I/II	Xencor	immune silencing	Autoimmune
BI 836826	Boehringer Ingelheim	ND	Phase I	Xencor	High ADCC	NHL, CLL
BI 836858	Boehringer Ingelheim	ND	Phase I	Xencor	High ADCC	AML
CSL 362	CSL	CD123	Phase I	Xencor	High ADCC	AML
MGA271	MacroGenics	B7-H3	Phase I	MacroGenics	High ADCC	Solid tumors

Source: Company data, Credit Suisse Research.

How Will High ADCC Antibodies Fit into the Broader World of Enhanced Antibodies?

Several other technologies to increase the potency of anti-cancer antibodies were developed in parallel to high ADCC approaches. These include antibody-drug conjugates (e.g., SGEN and IMGN) and bispecific antibodies (e.g., AMGN).

The available data for these drug classes allow us to draw three broad conclusions (which are still somewhat speculative):

1. High ADCC antibodies are more potent than standard antibodies
2. Antibody-drug conjugates and bispecifics are likely to be more potent than high ADCC antibodies against the same target
3. High ADCC antibodies are likely to be safer and more easily combinable with other drugs compared to antibody-drug conjugates and bispecifics.

The implication of these conclusions is that high ADCC antibodies may be best when combined with other agents, potentially in front-line settings (for example Gazyva in CLL).

It will be interesting to see how the high ADCC antibodies are ultimately developed when there are drug conjugates and bispecifics against the same target, for example CD19 and CD123 (IL3R).

Extended Half-Life

One reason antibodies have such long half-lives in circulation (days or weeks for antibodies compared to minutes or hours for most small molecules) is that the Fc domain interacts with the FcRn receptor to recirculate antibodies in the blood. Antibodies engineered to have enhanced binding to FcRn receptors have extended half-lives compared to antibodies with native Fc domains.

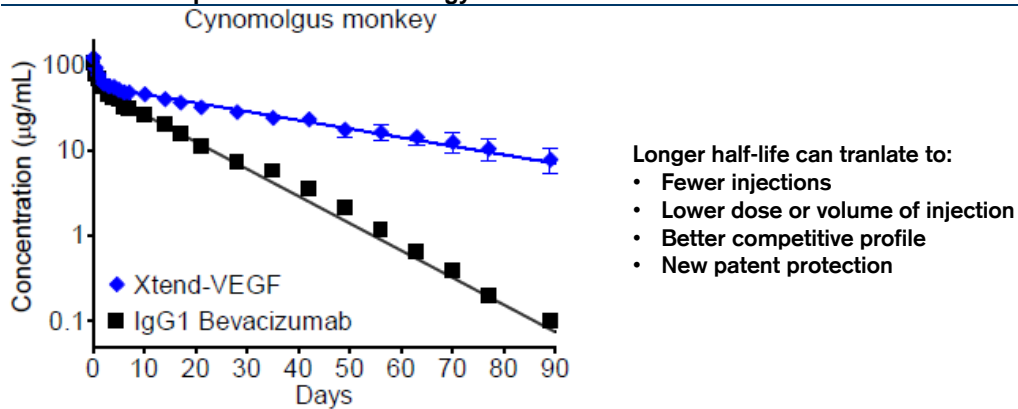
This technology, which XNCR calls Xtend, is ideal for partnering. Many companies want to improve the half-life of their marketed or development stage antibodies to (1) reduce the frequency of injection, (2) reduce the volume of dose per injections, or (3) extend the patent life of important franchises.

To date, Xencor has signed three deals with its Xtend technology. In general, these deals include relatively small upfront payments and low-single-digit royalties. Targets are typically not disclosed. The cost to XNCR is low because there is almost no technology transfer required (just access to the mutations), and the opportunity cost is also low for

certain programs, in which XNCR has no freedom to operate because of the currently marketed products and/or intellectual property.

Partners for this technology include Alexion, Janssen, and CSL. If the Alexion program turns out to be an improved version of the blockbuster Soliris, or Janssen uses this technology to improve Remicade, investors may begin to value these programs as future royalty streams to Xencor.

Exhibit 14: Example of Xtend Technology



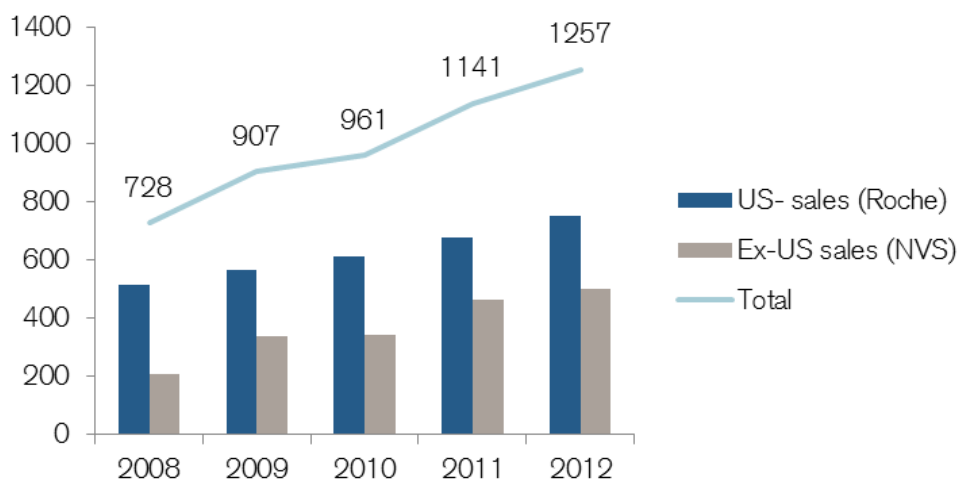
Source: Company data, Credit Suisse Research.

XmAb7195—A Better Xolair

The Good and Bad of Xolair

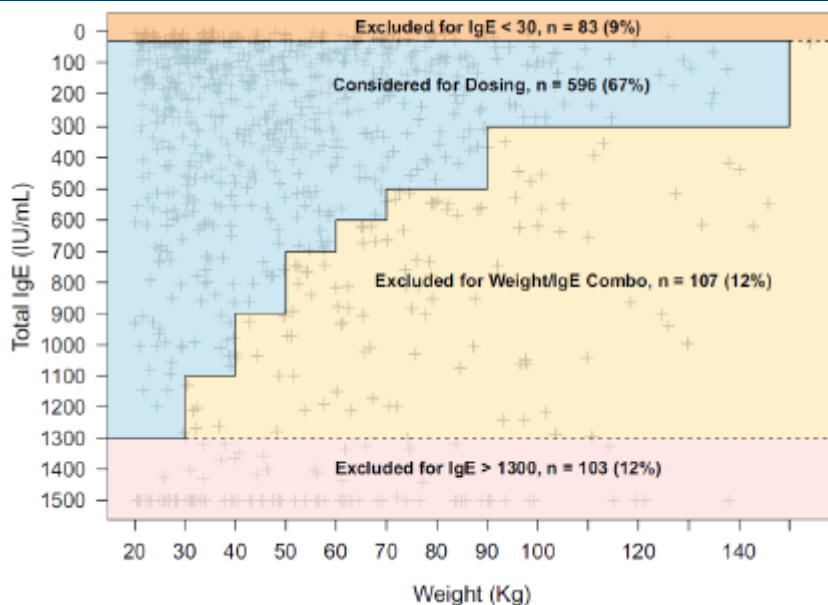
- **Clinically Validated:** One culprit in asthma is the over production of an antibody called IgE. Increased IgE levels are associated with worse symptoms, and inhibition of IgE by the antibody Xolair reduces symptoms in moderate to severe asthma patients. For these reasons, IgE is a clinically validated target, with reduced clinical and regulatory risk.
- **Commercially Validated:** Xolair is marketed by Roche and Novartis, and total worldwide sales were approximately \$1.25B in 2012 and growing at a three-year annualized rate of ~12%. (See Exhibit 15.) The ongoing adoption of Xolair makes IgE a commercially validated target.

Exhibit 15: Xolair Sales

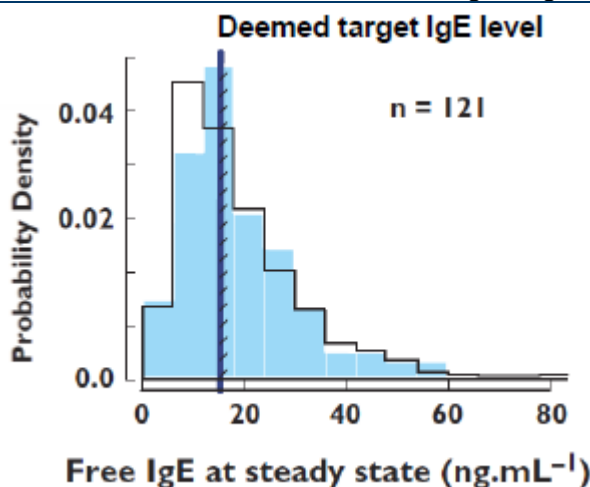


Source: Company data, Credit Suisse Research.

- **Imperfect Drug:** Despite the current blockbuster status, Xolair is generally viewed as an imperfect drug. For example, there is a complex dosing table for Xolair that is based on the weight of the patient and their baseline IgE levels. (See Exhibit 16). Large patients or patients with very high IgE levels cannot be treated with Xolair because the drug has a limited capacity to neutralize its target. Approximately 20% of asthma patients with high IgE or high body mass are ineligible for Xolair because they fall outside the dosing chart. This shortcoming may also reduce the activity of Xolair for patients who are on the margin within the dosing chart. Of the patients treated with Xolair, around 50% do not achieve target IgE reduction (See Exhibit 17).

Exhibit 16: Xolair Dosing Chart

Source: Company data, Credit Suisse Research.

Exhibit 17: IgE Levels After Treatment—50% Do Not Meet IgE Target Levels

Source: Company data, Credit Suisse Research.

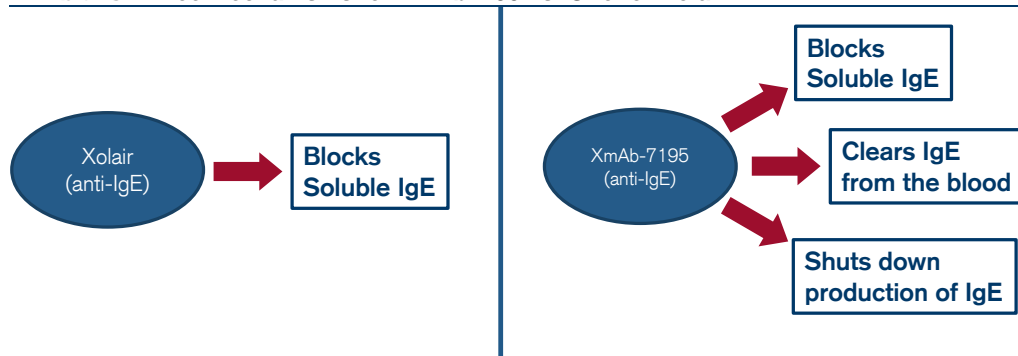
A Better Xolair—XmAb7195 Blocks IgE by Three Mechanisms

XmAb7195 is designed to have the same IgE binding region as Xolair, but its Fc domain has been modified for enhanced activity. While Xolair simply binds soluble IgE, XmAb7195 also shuts down the production of IgE and facilitates the clearance of IgE from the blood (See Exhibit 18). This triple mechanism of action is expected to have a greater impact on IgE levels and therefore asthma symptoms. It is also likely to overcome the dosing problems of Xolair.

The attributes of XmAb7195 have been demonstrated in monkeys who naturally produce high levels of IgE.

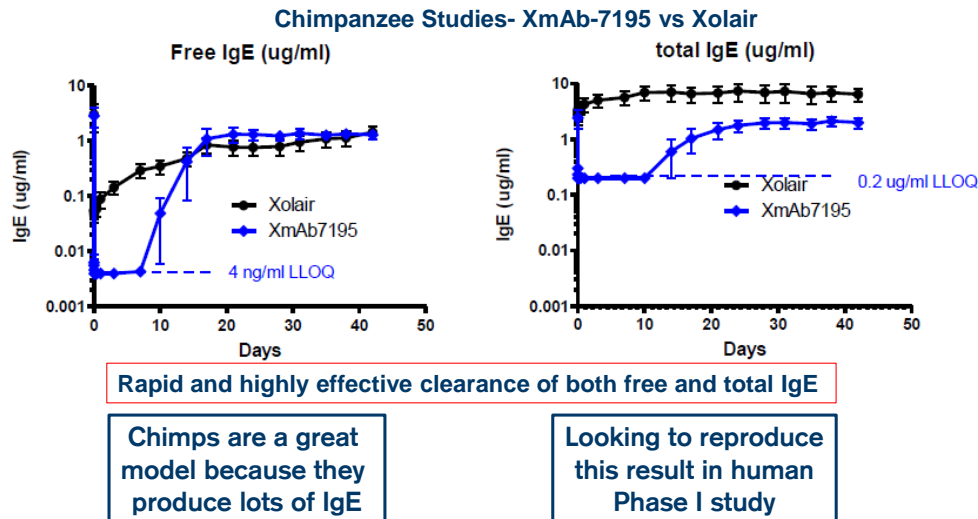
- **Xolair Rapidly Reduces Free IgE** (Exhibit 19 left panel): Xolair can effectively bind free IgE. The reduction in free IgE is associated with reduced asthma symptoms. XmAb7195 also binds free IgE and effects even greater reduction in free IgE compared to Xolair.
- **Xolair Has No Impact on Total IgE** (Exhibit 19 – right panel): Total IgE includes both antibody-bound and free IgE. Because Xolair does not induce the clearance of IgE, the bound IgE circulates and the free IgE levels return at the rate of antibody clearance. XmAb7195 lowers total IgE because the bound IgE is cleared from the blood. Total IgE levels recover but at a slower rate because of the immune down-regulation.

Exhibit 18: Three Mechanisms for XmAb7195 vs. One for Xolair



Source: Company data, Credit Suisse Research.

Exhibit 19: More Complete Blockade of IgE with XmAb7195



Source: Company data, Credit Suisse Research.

Timeline—Potential Biologic Proof of Concept by Q4:14

Phase I Start in H1:14: XNCR has completed preclinical toxicity and is completing production of GMP lots of XmAb7195. XNCR anticipates filing an IND in late Q1:14 and initiating Phase I in in early Q2:14.

The single ascending dose Phase I trial will include healthy volunteers and be conducted at a single clinical site. The study will also include an expansion cohort of healthy individuals with elevated IgE levels.

In addition to establishing safety and PK in Phase I, XNCR hopes to replicate the positive data observed in monkeys, which showed both a reduction in free IgE and total IgE. Thus, Phase I is expected to serve as a significant biologic proof of concept. This data could be available by late 2014.

Following completion of the Phase I trial, Xencor anticipates initiating the multi-ascending dose study in Q1:15 in patients with mild-to-moderate asthma.

Phase II Start in Late 2015 or Early 2016: A full Phase II program in the target higher-risk moderate-to-severe asthmatics with elevated IgE is anticipated in late 2015 or early 2016, with data by year-end 2017. The Phase II study may likely include a subcutaneous formulation, but the Phase I work is being done with an IV formulation.

Market for a Better Xolair

We assume there are ~380,000 severe refractory, perennial asthmatics in the United States. It is estimated that approximately 50-100K are ineligible for Xolair due to weight restrictions and/or high IgE. About half of the patients treated with Xolair do not meet IgE targets. Currently, around 60K patients in the U.S. are treated with Xolair. We believe XmAb7195 could expand the market through better efficacy and by treating patients currently ineligible for Xolair and patients who respond poorly to Xolair.

Exhibit 20: Industry Pipeline of Biologics for Asthma

Name(s)	Company	Stage	Target	Route
Xolair (Omalizumab)	Roche	Approved	IgE	Subcutaneous
Bosatria (Mepolizumab, SB-240563)	GlaxoSmithKline	III	IL-5	Intravenous
Lebrikizumab	Roche	III	IL-13	Subcutaneous
Cinquil (Reslizumab, SCH 55700)	Teva	III	IL-5	Intravenous, Subcutaneous
Benralizumab (MEDI-563)	AstraZeneca	III	IL-5R	Subcutaneous
Secukinumab (AIN457)	Novartis	II	IL-17R	Intravenous, Subcutaneous
Brodalumab (AMGN 827)	Amgen	II	IL-17R	Intravenous, Subcutaneous
Dupilumab (REGN668 / SAR231893)	Regeneron / Sanofi	II	IL-4R (IL-4/IL-13)	Subcutaneous
Tralokinumab (CAT-354)	AstraZeneca	II	IL-13	Subcutaneous
KB003	KaloBios	II	GM-CSF	Intravenous
QBX-576	Novartis	II	IL-13	Intravenous
QGE031	Novartis	II	IgE	N/A
Quilizumab	Roche	II	IgE	Intravenous
MEDI-4212	AstraZeneca	I	IgE	Intravenous, Subcutaneous
AMG 157	Amgen	I	Thymic stromal lymphopoietin (TSLP)	Intravenous, Subcutaneous
CNTO-3157	Janssen	I	NA	Intravenous
RPC4046	Receptos	I	IL-13	Intravenous, Subcutaneous

Source: Company data, Credit Suisse Research.

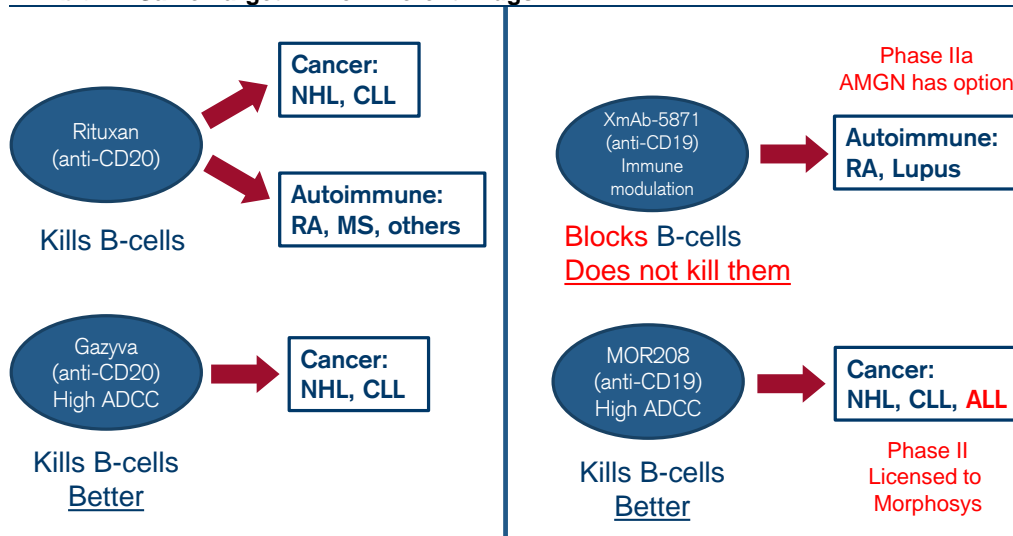
CD19 Highlights Technology and Business Model

Xencor has a large "tool box" of modified Fc domains that can be used to enhance certain functions of an antibody. These include greater ADCC, longer half-life, immune down-regulation, and others.

In some cases, these different Fc domains can be used against the same target to generate distinct drug candidates that are optimized for different therapeutic settings. This is the case with antibodies targeting CD19 and the two candidates XmAb5871 (immune down-regulation) and MOR208 (high ADCC).

The result for XNCR was two different partnerships – an out-licensing deal to MorphoSys for its anti-lymphoma/leukemia drug candidate and an option deal with AMGN for its autoimmune drug candidate

Exhibit 21: Same Target—Two Different Drugs



Source: Company data, Credit Suisse Research.

MOR 208 for Lymphoma / Leukemia

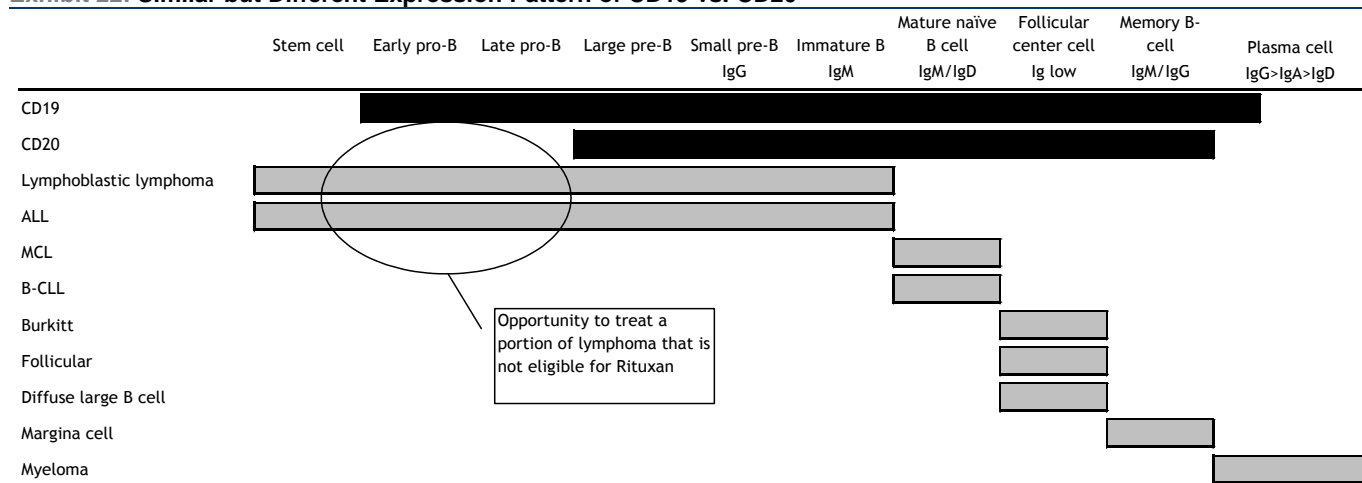
CD19 Is a "Hot" Target for Lymphoma and Leukemia

CD19 is a prime target for the development of drugs to treat lymphoma and leukemia. Its expression is limited to cells of the B-cell lineage, similar to CD20, which is the target for the blockbuster drug Rituxan and the recently launched Gazyva.

The two primary differences between CD19 and CD20 are:

- CD19 is expressed earlier in B-cell development, making it a valuable target for acute lymphoblastic leukemia (ALL); and
- CD19 is internalized (unlike CD20), making it better for the development of antibody-drug conjugates.

As a result of the similarities and differences to CD20 and the clinical and commercial success of Rituxan, multiple companies are pursuing enhanced antibody approaches to target CD19 primarily for diffuse large B-cell lymphoma (DLBCL) and ALL.

Exhibit 22: Similar but Different Expression Pattern of CD19 vs. CD20

Source: Company data, Credit Suisse Research

Exhibit 23: Multiple Approaches to Targeting CD19

Company	Drug	Format of the Antibody	Stage
Amgen/Micromet	MT-103	Bispecific	Phase II/III
Sanofi Aventis/Immunogen	SAR3419	Antibody drug conjugate	Phase II
Bristol/Medarex	MEDI-551	High ADCC (glyco engineered)	Phase II
Morphosys/Xencor	XmAb5574	High ADCC (Fc engineered)	Phase IIa
Novartis	CTL019	CAR(T) cell therapy	Phase I/II
Seattle Genetics	SGN-CD19a	Antibody drug conjugate	Phase I
Affimed Therapeutics AG	AFM11	Bispecific	Preclinical

Source: Company data, Credit Suisse Research

MorphoSys Is Committed to Advancing MOR208

MorphoSys is a fully integrated antibody development company, with a deep pipeline of partnered and proprietary antibodies. Their primary technology is focused on engineering human antibodies.

MorphoSys licensed MOR208 from XNCR, and this is now MorphoSys's lead proprietary drug candidate (several programs from big pharma licensing partners are more advanced). MorphoSys says it plans to take MOR208 through development on its own as its lead drug candidate.

Two Phase II Trials Are Enrolling, with Data Likely in 2014 and 2015; CLL Phase II Expected to Start Enrolling Soon

There are three Phase II trials for MOR208 in ALL (30 patients), NHL (120 patients), and CLL (40 patients). According to MorphoSys, the ALL and NHL trials are recruiting well and should be complete in H2:14 (ALL) and Q2:15 (NHL). The CLL trial was only recently announced.

Preliminary data from the open-label Phase II trials in ALL and/or NHL may be available for the ASCO 2014 in June 2014, and MorphoSys may release additional data from individual subgroups of the NHL trial (there are four subgroups) prior to the completion of the trial.

CLL Prior Data Show Decent Single-Agent Response; Revlimid Combo Is Path Forward

MorphoSys announced that a Phase II trial in chronic lymphocytic leukemia (CLL) in combination with Revlimid has been initiated and is expected to begin enrolling patients very soon.

MorphoSys released the first Phase I/II data for MOR208 in CLL at ASH 2012 in December 2012. At that time, the single-agent response rate was 15% (4/27). With additional follow up and longer duration of treatment, the response rate was later reported at 30% (8/27, all partial responses). The full results from the Phase I/II experience will likely be published in a journal in 2014.

Based on the Phase I/II results, MOR208 is clearly active. However, the lack of complete responses (CRs) suggests that MOR208 may be best if combined with other agents (typical for antibodies).

MorphoSys has decided to move forward in CLL with a Phase II combo trial with Revlimid in up to 20 treatment naïve and 20 relapsed/refractory CLL patients. The trial is designed as an open-label, dose-ranging trial.

Revlimid is known to enhance T-cell and NK-cell activity, which is the rationale behind Rituxan/Revlimid combos. By a similar mechanism, the combination of MOR208 and Revlimid appear to act synergistically in preclinical models of CLL.

The strategy moving forward in CLL is uncertain (specific patient populations, etc.), given the number of new agents to treat the disease (e.g., BCR agents). It is possible that MOR208 may find a place in combo therapy (a better Rituxan) or in patients who have failed other agents.

Exhibit 24: MOR208 Current Phase II Trials

	NHL	ALL	CLL
Stage	Phase IIa	Phase IIa	Phase II
# of pts	120 patients	30 patients	40 patients
Indication	Relapsed/refractory NHL	Relapsed/refractory B-ALL	Relapsed/refractory and treatment naïve CLL
Eligibility requirements	Failed at least 1 prior Rituxan regimen	Failed at least 1 prior regimen	Failed at least 1 prior regimen or ineligible for chemo based therapy
	Four cohorts - FL - MCL - DLBCL - Other indolent		
Status	Enrolling Expect to complete in 2015 Potential interim data at ASCO 2014	Enrolling Complete in H2:14	Enrolling Study was started in Dec 2013
Start	May-13	Apr-13	Dec-13
Completion	Nov-16	Jul-14	Oct-17
NCT#	NCT01685008	NCT01685021	NCT02005289

Source: Company data, Credit Suisse Research

Lymphoma Is a Challenging Space with Evolving Competitive Dynamics

In addition to the large number of CD19-targeted drugs in development (see Exhibit 23), there are an even larger number of drugs in development or recently approved that target the same B-cell malignancies. These include, but are not limited to, the following:

- **Fc Enhanced Antibodies:** As mentioned previously, Gazyva is a recently approved high ADCC antibody targeting CD20.
- **Antibody Drug Conjugates Against Other Targets:** Roche is developing two drug conjugates targeting CD22 and CD79b. Both are in Phase II for NHL.
- **New Oral B-Cell Receptor Signaling Inhibitors:** Drugs targeting both BTK and PI3K are now approved or in late-stage development for the treatment of B-cell lymphomas. These include drugs from PCYC/JNJ, GILD, and INFI.
- **New BCL-2 Inhibitors:** ABBV and Roche are developing an oral agent that directly kills tumor cells, with very strong activity in B-cell lymphoma. This drug can be used alone and potentially in combination with Rituxan or Gazyva to produce very high response rates.

Given the rapidly evolving space including very high and durable response rates with newer agents, we believe that new CD19 targeted drugs may find their first and best utility in ALL, where the unmet medical need is very high and most of the other targeted therapies (including Rituxan) are either not tested or not effective.

CD19 targeted drugs may also be used initially in relapsed/refractory patients of other B-cell malignancies (e.g., DLBCL), and may move into earlier stage treatment if novel drug combinations are tested and prove superior. This is the most risky part of the development of these agents.

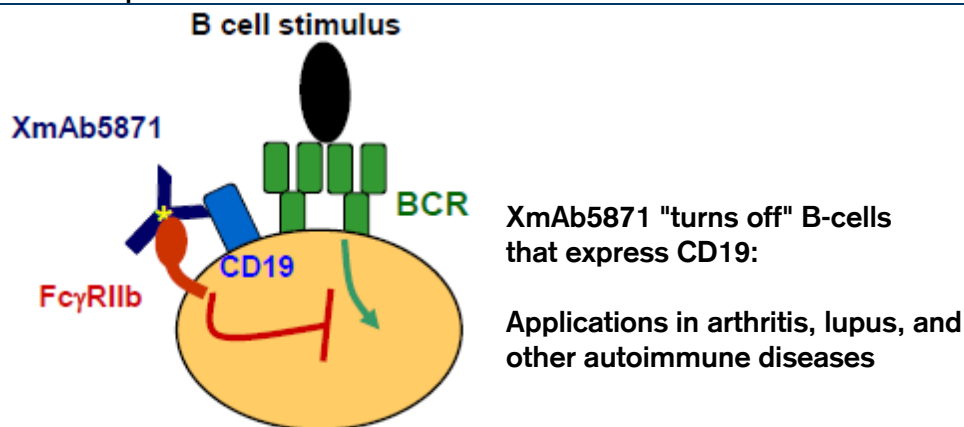
XmAb5871 for RA and Lupus

A Proven Pathway with Some Need for Improvement

Targeting B-cells is a validated pathway for the treatment of rheumatoid arthritis, multiple sclerosis, and a variety of other autoimmune diseases including lupus. Clinical trials with Roche/BiIB's Rituxan (anti-CD20) provide the best evidence for this approach. Rituxan works primarily by killing B-cells (thus its activity in B-cell NHL), which shuts off the production of auto-reactive antibodies in autoimmune disease. The drawback of this approach is that Rituxan wipes out B-cells for six months or more, which can lead to prolonged immunosuppression. Ideally, a drug would shut down the B-cells without killing them, so if a patient develops an infection, the drug's activity can be reversed and normal immune function restored.

XNCR's Solution: CD19-Targeted Immune Down-Regulation

XmAb5871 targets CD19, which is expressed on nearly all B-cells (like Rituxan's CD20). XNCR has modified the Fc region of XmAb5871 for enhanced binding to FcγRIIb, which causes the inhibition of B-cell function rather than targeting the B-cell for destruction through ADCC. The result is a B-cell specific immune modulator that is more easily reversed than Rituxan.

Exhibit 25: Specific Inhibition of B Cells

Source: Company data, Credit Suisse Research

Partnership Lowers Risk and Provides Funding to Prove the Technology

The field of RA is extremely crowded, with very effective biologics primarily targeting TNF alpha (Humira, Remicade, Enbrel, etc.). At the same time, RA is an ideal proving ground for immune-targeted therapies because the trials are relatively easy to conduct and the endpoints are clear and quantitative. Given AMGN is largely funding early development, we believe this is an ideal setting to prove the technology.

AMGN also views RA as the appropriate space for proof of concept, although we believe AMGN is primarily interested in the unmet need in lupus. Phase II data in RA are easier to generate and are the gating factor for Amgen to exercise its option.

Ongoing Clinical Program—Expected Timeline

XNCR is enrolling the Phase IIa portion of the Phase I/II clinical trial. This study will enroll up to 30 patients with active RA, randomized two-to-one to XmAb5871 or placebo. Patients are dosed every other week for 12 weeks (six injections) and then followed for an additional 12 weeks. Data from this study are expected by year-end 2014.

The Phase IIb trial is expected to kick off in Q1:15. Additional funding of \$12M is tied to the progress of the Phase IIb study. Xencor estimates the trial will include 150-250 patients, take approximately 12 months to enroll, and read out in late 2016 or early 2017.

The results of this trial will be the basis for AMGN to exercise its option for the program (likely in Q1:17)

Bispecifics—Fc Enabled Production

Xencor's Fc engineering technology has been applied to the development of bispecific antibodies. These are drugs that are designed to bind two separate targets using two variable regions.

Bispecifics can be developed for two different purposes

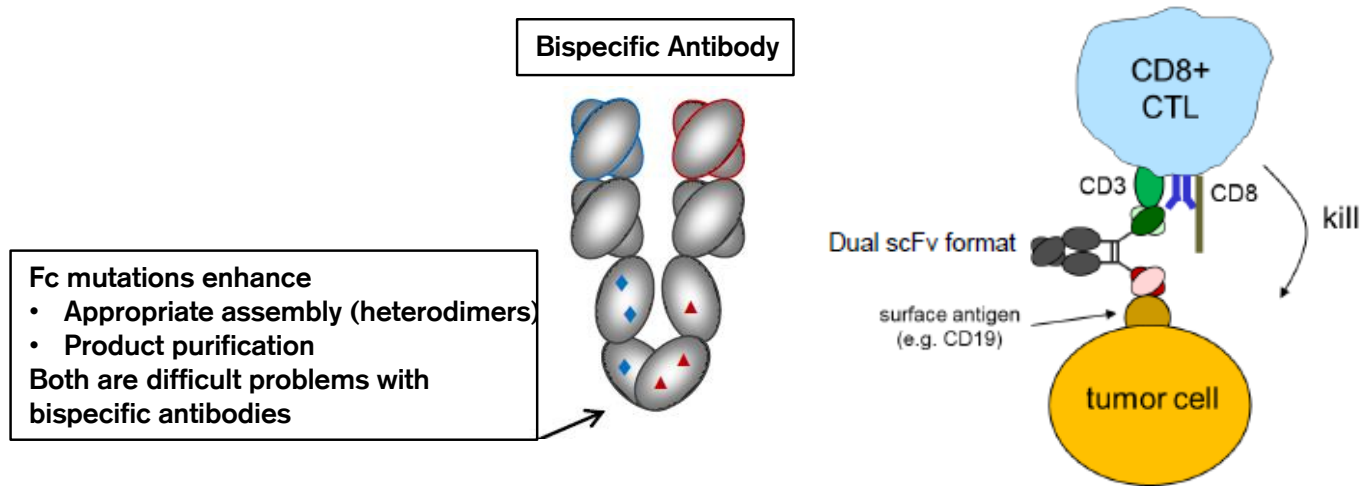
- **Enhanced Immune Cell Killing:** Bispecifics can be used to bring potent killer T-cells in close contact with targeted cancer cells. This is accomplished by binding a cell surface receptor on tumor cells (e.g., CD19 or others) and an activating receptor on T-cells (e.g., CD3).
- **Two-in-One Drug:** Bispecifics can potentially block two different soluble targets (e.g., VEGF and FGF) with the aim of doing the job of two drugs in a single molecule. To date, there are not good clinical examples of this.

Fc Engineering a Better Bispecific

Recall a standard antibody has two identical variable regions and an Fc. One of the difficulties in developing antibodies with two different variable regions is that you can generate three species of molecules during the manufacturing process (AA, AB, and BB).

Xencor has used its Fc engineering technology to create bispecifics that preferentially form the appropriate heterodimers (i.e., AB form). These same mutations also facilitate the purification and characterization of the desired antibody using standard antibody purification methods.

Exhibit 26: Xencor's Proprietary Bispecific Format



Source: Company data, Credit Suisse Research.

Expect Xencor to Announce Its Lead Bispecific in 2014

Xencor has two preclinical bispecific candidates and expects to advance one of them into IND-enabling preclinical studies in 2014, with an IND filing likely in H1:15.

The two lead candidates include a bispecific targeting CD38 and CD3 potentially for multiple myeloma and one targeting CD123 (IL3R) and CD3 potentially for acute myeloid leukemia. Both candidate targets have initial proof of concept from competitor programs, and there are currently no advanced-stage bispecifics against either target.

- **CD38 for Multiple Myeloma (MM):** There are currently no approved antibodies for the treatment of MM. Recent clinical data demonstrate that CD38 targeted antibodies may have significant activity in this setting. Currently, there are three CD38-targeted antibodies in clinical development: Daratumumab from Genmab/JNJ (Phase II), MOR03087 from MorphoSys (Phase I/II), and SAR650984 from Sanofi/Immunogen (Phase I).
- **CD123 (IL3R) for AML:** Initial proof of concept for AML-targeted drugs comes from SL-401 from STML. This drug is a protein fusion toxin, linking IL3 (the natural binding partner of CD123) and a potent bacterial toxin (diphtheria toxin). This drug has shown activity in rare CD123-expressing tumors. One of Xencor's partners, CSL, is advancing a high ADCC antibody against CD123 (CSL362). MacroGenics is also developing MGD006, targeting CD123-positive leukemia cells with its DART technology.

Of the two, we believe that CD38 is the best validated with the biggest market opportunity. However, it is unclear whether it is a safe target for more potent technologies because of expression in cells other than myeloma cells.

Multiple Platforms to Develop Bispecifics—Not a Winner-Take-All Opportunity

The development of bispecific antibodies has been challenging, and multiple solutions have emerged. At this point, there is no one winning technology, and we expect each will have its positives and negatives. As with most antibody technologies, target selection and development strategy still remain the key factors for determining success, and Xencor is very much in the race.

- **Amgen (Micromet):** The BiTE technology was developed at Micromet which was acquired by Amgen in 2012. The lead BiTE, blinatumomab (CD19/CD3), is set to begin Phase III studies for ALL and has generated significant proof of concept for the approach of targeting tumors and T-cells (CD3) to get potent tumor cell killing. The primary drawbacks for the BiTE technology are the short half-life and difficulty in manufacturing.
- **Macrogenics:** The Dual-Affinity-Re-Targeting (DART) technology consists of dual-specificity therapeutic proteins, with each region linked together through a covalent linkage. The platform can be adapted for ligand targeting, signal modulation, and redirected effector cell killing. Macrogenics has DART partnerships with Gilead, Pfizer, and Boehringer Ingelheim.
- **Regeneron:** Full-length bispecifics using universal light chain technology. The lead program is a CD20/CD3, which is in preclinical development.
- **Affimed:** The TandAbs platform is a tetravalent bispecific antibody format that has two binding sites for each antigen. Affimed's lead program AFM13 is a CD30 X CD16A in Phase I development for HL. They also have a preclinical program, AFM11 (CD19 X CD3) in NHL.

XNCR Management

The team at XNCR includes founding members with significant experience in protein/antibody engineering.

- **Bassil I. Dahiyat, Ph.D.**, has served as XNCR's president and chief executive officer since February 2005 to present, and from 1997 to 2003, and has been a member of its board of directors since August 1997. Dr. Dahiyat co-founded Xencor in 1997. Dr. Dahiyat holds a Ph.D. in chemistry from the California Institute of Technology and B.S. and M.S.E. degrees in biomedical engineering from Johns Hopkins University.
- **Edgardo Baracchini, Jr., Ph.D.**, has been XNCR's chief business officer since 2010. He has held previous positions as VP of business development at Metabasis Therapeutics, Inc. until its merger with Ligand Pharmaceuticals Inc., Elitra Pharmaceuticals Inc., and Agouron Pharmaceuticals, Inc. until its acquisition by Warner-Lambert Co. Dr. Baracchini holds a Ph.D. in molecular and cell biology from the University of Texas at Dallas and conducted his postdoctoral research at the University of California, San Diego, and The Scripps Research Institute. He has an M.B.A. from the University of California, Irvine, and a B.S. in microbiology from the University of Notre Dame.
- **Paul Foster, M.D.** became chief medical officer of XNCR in August 2012, after serving in a similar role as an outside consultant from January 2010 until August 2012. Previously, he was chief medical officer for Cardium Therapeutics. Prior to that, he provided medical/clinical consulting services as SVP development and chief medical officer of development at 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 his M.D. from Duke University School of Medicine and received a B.S. in chemistry from the University of Michigan.
- **John R. Desjarlais, Ph.D.** has been vice president, research since October 2006, and joined the XNCR in July 2001, as director of protein engineering. Prior to XNCR, Dr. Desjarlais was an assistant professor of chemistry at Penn State University from 1997 to 2001. Dr. Desjarlais received a B.S. in physics from the University of Massachusetts and a Ph.D. in biophysics from Johns Hopkins University.
- **John J. Kuch** has been XNCR's vice president, finance since October 2010, and joined the company in October 2000, as senior director of finance. Prior to XNCR, Mr. Kuch was director at Price Waterhouse. Mr. Kuch is a certified public accountant and received his B.S. and M.S. in accounting from the University of Illinois.

Financial Statements

Exhibit 27: Income Statement

	2012A	2013E	2014E	2015E	2016E	2017E	2018E	2019E	2020E	2021E	2022E
Revenues											
US sales of XmAb7195											26.9
Ex-US royalties on XmAb7195											1.0
Royalties on XmAb5871											50.0
Partnering, grants, milestones	9.5	10.1	4.6	7.0	11.0	26.1	15.0	20.0	20.0	50.0	50.0
Total Revenues	9.5	10.1	4.6	7.0	11.0	26.1	15.0	20.0	20.0	50.0	77.9
Expenses											
Cost of goods											3.2
Research and development	12.7	17.3	18.4	21.7	28.5	31.4	39.3	40.6	42.0	47.0	46.7
Sales, general, administrative	3.1	4.1	7.1	7.9	8.7	9.9	14.3	14.6	19.0	28.0	30.8
Total Operating Expenses	15.8	21.4	25.5	29.6	37.2	41.3	53.6	55.2	61.0	75.0	80.7
Operating income (loss)	(6.2)	(11.3)	(20.8)	(22.7)	(26.3)	(15.2)	(38.6)	(35.2)	(41.0)	(25.0)	(2.8)
Total Other Income (Expense)	(2.4)	(49.7)									
Pre Tax Income	(8.6)	(61.0)	(20.8)	(22.7)	(26.3)	(15.2)	(38.6)	(35.2)	(41.0)	(25.0)	(2.8)
Income tax											
Net Income	(8.6)	(61.0)	(20.8)	(22.7)	(26.3)	(15.2)	(38.6)	(35.2)	(41.0)	(25.0)	(2.8)
EPS - diluted (proforma)	(\$38.31)	(\$1.95)	(\$0.65)	(\$0.64)	(\$0.61)	(\$0.33)	(\$0.77)	(\$0.58)	(\$0.64)	(\$0.37)	(\$0.04)
Shares outstanding - basic (proforma)	0.22	31.33	32.12	35.18	43.02	46.56	50.35	60.87	63.91	67.11	70.46
Shares outstanding - diluted (proforma)	0.22	32.61	33.47	36.63	44.59	48.26	52.19	62.80	65.94	69.24	72.70

Source: Company data, Credit Suisse estimates

Exhibit 28: Condensed Balance Sheet

Balance sheet	2012A	2013E	2014E	2015E	2016E	2017E	2018E	2019E	2020E	2021E
Total Current Assets	2.8	77.1	56.2	137.2	103.6	87.9	49.9	163.5	122.5	97.5
Property and equipment		0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3
Total other assets		9.1	9.1	9.1	9.1	9.1	9.1	9.1	9.1	9.1
TOTAL ASSETS	11.7	86.5	65.6	146.6	113.0	97.3	59.4	172.9	131.9	106.9
LIABILITIES & EQUITY										
Total Current Liabilities	25.5	8.6	8.6	8.6	8.6	8.6	8.6	8.6	8.6	8.6
Total Liabilities	31.2	14.4	14.1	19.7	11.9	11.4	11.5	11.5	11.5	11.5
Total stockholders' deficit	-166.3	72.5	52.0	127.3	101.6	86.4	47.8	161.4	120.4	95.4
TOTAL LIABILITIES & MEZZANINE EQUITY & STOCK	11.7	86.9	66.1	147.0	113.4	97.8	59.3	172.9	131.9	106.9

Source: Company data, Credit Suisse estimates

Exhibit 29: Condensed Cash Flow Statement

Cash flow	2012A	2013E	2014E	2015E	2016E	2017E	2018E	2019E	2020E	2021E
Net Income	(8.6)	(61.0)	(20.8)	(22.7)	(26.3)	(15.2)	(38.6)	(35.2)	(41.0)	(25.0)
Net Cash Provided by Operating Activities	(11.1)	(8.5)	(21.1)	(16.9)	(33.9)	(15.5)	(40.1)	(35.3)	(42.5)	(28.1)
Net Cash provided by investing activities	(1.2)	(1.3)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Net Cash Provided by Financing Activities	(0.0)	82.3	0.0	97.7	0.0	0.0	0.0	148.8	0.0	0.0
Net Cash Increase (Decrease)	(12.2)	72.5	(21.1)	80.7	(33.9)	(15.5)	(40.1)	113.5	(42.5)	(28.1)
Beginning Cash	14.5	2.3	74.8	53.7	134.4	100.6	85.1	45.0	158.5	115.9
Ending Cash	2.3	74.8	53.7	134.4	100.6	85.1	45.0	158.5	115.9	87.8

Source: Company data, Credit Suisse estimates

Companies Mentioned (Price as of 02-Jan-2014)

Alexion Pharmaceuticals Inc. (ALXN.OQ, \$133.44)
Amgen Inc. (AMGN.OQ, \$115.795)
AstraZeneca (AZN.L, 3558.0p)
Biogen Idec (BIIB.OQ, \$280.33)
CSL Ltd (CSL.AX, A\$68.82)
Celldex Theracs (CLDX.OQ, \$24.61)
Dyax (DYAX.OQ, \$7.6)
Five Prime (FPRX.OQ, \$15.96)
GENMAB (GEN.CO, Dkr217.5)
GlaxoSmithKline plc (GSK.L, 1599.5p)
ImmunoGen, Inc. (IMGN.OQ, \$14.98)
Johnson & Johnson (JNJ.N, \$91.03)
KaloBios (KBIO.OQ, \$4.39)
Kyowa Hakko Kirin (4151.T, ¥1,159)
MacroGenics (MGNX.OQ, \$28.83)
Merck & Co., Inc. (MRK.N, \$49.49)
Merrimack (MACK.OQ, \$49.49)
Novartis (NOVN.VX, SFr71.2)
OncoMed (OMED.OQ, \$29.32)
Receptos (RCPT.OQ, \$30.23)
Regeneron Pharmaceutical (REGN.OQ, \$274.59)
Roche (ROG.VX, SFr249.2)
Sanofi (SASY.PA, €75.22)
Seattle Genetics (SGEN.OQ, \$40.18)
XOMA Corporation (XOMA.OQ, \$6.92)
Xencor, Inc (XNCR.OQ, \$8.88, OUTPERFORM[V], TP \$14.0)

Disclosure Appendix

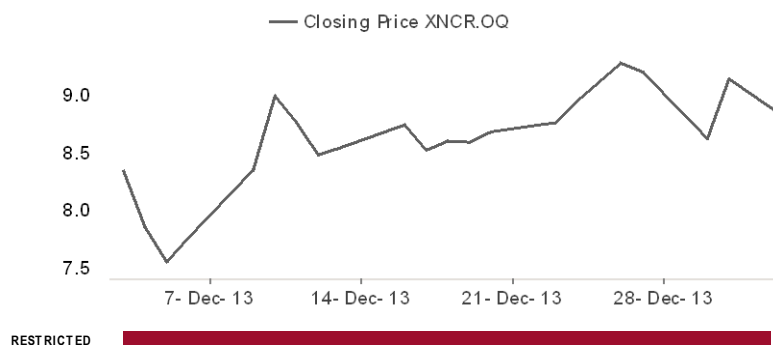
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3-Year Price and Rating History for Xencor, Inc (XNCR.OQ)

XNCR.OQ	Closing Price	Target Price	
Date	(US\$)	(US\$)	Rating
03-Dec-13	8.34		R

* Asterisk signifies initiation or assumption of coverage.



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Price Target: (12 months) for Xencor, Inc (XNCR.OQ)

Method: Our \$14 target for XNCR is derived using a probability-adjusted DCF, including \$9 for XmAb7195 (40% POS), \$3 for XmAb5871 (25% POS), and \$1 each for MOR208 and the technology licensees. We use a 12% discount rate and model through the products' entire lifecycle.

Risk: Risks to our \$14 TP include: 1) unexpected negative result for proprietary or partnered clinical programs, 2) financing risk from expected future equity raises, 3) competition in the CD19 and asthma programs, and 4) significant delay in one or more clinical programs that pushes potential approval timeline(s) out.

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