July 10, 2014

HEALTHCARE/BIOTECHNOLOGY

Stock Rating:

OUTPERFORM

12-18 mo. Price Target	$48.00
MGNX - NASDAQ	$19.72

3-5 Yr. EPS Gr. Rate	NA
52-Wk Range	$41.00-$17.96
Shares Outstanding	26.3M
Float	18.3M
Market Capitalization	$544.7M
Avg. Daily Trading Volume	366,062
Dividend/Div Yield	$0.00/0.00%
Book Value	$5.83
Fiscal Year Ends	Dec
2014E ROE	NA
LT Debt	$0.0M
Preferred	NA
Common Equity	$153M
Convertible Available	No

EPS	Q1	Q2	Q3	Q4	Year	Mult.
2013A	(2.93)	(0.24)	0.01	(0.14)	(0.04)	NM
2014E	(0.12)A	(0.56)	(0.63)	(0.64)	(1.97)	NM
2015E	(0.69)	(0.70)	(0.72)	(0.73)	(2.83)	NM

Revenue ($/mil)	Q1	Q2	Q3	Q4	Year	Mult.
2013A	10.6	12.3	20.2	14.9	58.0	7.0x
2014E	14.7A	5.3	5.3	5.3	30.7	13.2x
2015E	4.3	4.3	4.0	4.0	16.6	24.5x

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MacroGenics, Inc.

Initiating Coverage with an Outperform Rating and $48 Price Target

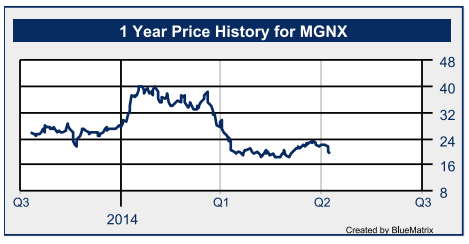
SUMMARY

We are initiating coverage of MacroGenics, Inc. (MGNX) with an Outperform rating and $48 price target. MacroGenics is an advanced protein therapeutic platform technology company. The company combines its understanding of the immune system and advanced protein engineering techniques to produce antibodies and antibody-like proteins that bind better and/or to multiple antigens through their DART (Dual Affinity Re-Targeting) bispecifics, to elicit a stronger immune response to kill cancer cells (Fc optimization) and avoid unfavorable immune reactions.

KEY POINTS

- MGA271 is an Fc-optimized antibody that both targets and blocks the B7-H3 T-cell inhibitory antigen on tumor and tumor vasculature. B7-H3 may have an immuno-modulatory role similar to other checkpoint proteins and ligands (PD1,PDL1,CTLA4,CD-27). Data from the PI expansion study in patients with advanced solid tumors is expected in 2015.
- Ex vivo results for MGD006, MGNX's bispecific DART, suggest significant activity in recruiting, expanding and activating T-cells to target CD123-expressing AML cells. Since CD123 is a clinically validated target in AML and hematological malignancies, these results support the potential for MGD006's efficacy and also provide proof-of-concept for the DART platform.
- Data from an open-label Phase I study of MGD006 in the advanced AML setting is expected around year-end 2014. We anticipate that no news is good news with respect to the safety of MDG006 and DART technology. MGD007, another bispecifc DART targeting GPA33, a novel target, is likely to be de-risked by strong MGD006 results.
- Top-line results from the Phase IIa study of margetuximab in the metastatic breast cancer setting are expected year-end 2014 We believe that margetuximab, an Fc-optimized HER2 antibody, may address low HER2 expressing indications of unmet medical need, creating a floor to MGNX's valuation.
- Our $48 price target is derived from the sum of multiples of sales and royalties from MacroGenics' proprietary and partnered products discounted annually.

Stock Price Performance



Company Description

MacroGenics is a clinical-stage biopharmaceutical company focused on discovering and developing engineered monoclonal antibody-based therapeutics for the treatment of cancer and autoimmune diseases.

Table of Contents

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

Investment Thesis

We are initiating coverage of MacroGenics Inc. (MGNX) with an Outperform rating and $48 price target. MacroGenics is an advanced protein therapeutic platform technology company. The company combines its understanding of the immune system and advanced protein engineering techniques to produce antibodies and antibody-like proteins that bind better and/or to multiple antigens through its DART (Dual Affinity Re-Targeting) bispecifics, elicit a stronger immune response to kill cancer cells (Fc optimization) and avoid unfavorable immune reactions. The company's Cancer Stem Cell platform (CSC) provides a source of novel cancer antigens against which to discover Fc-optimized antibodies or DARTs.

MGA271, targeting B7-H3, may have an immune modulatory role similar to other checkpoint inhibitors that are transforming cancer immunotherapy. We expect that MGA271 will synergize with other immunologically active therapies and/or following the appropriate conditioning regime to deplete T-regulatory cells. Data from the Phase I expansion study in patients with advanced solid tumors is expected to be a significant catalyst for MacroGenics in 2015.

Safety data expected year-end 2014 is likely to significantly de-risk MGD006, in our opinion, as the therapeutic engages a clinically validated target. Compelling preclinical data for MGNX's first DART (MGD006—CD123xCD3) demonstrates that the drug recruits, expands and activates human T-cells that elicit dose-dependent killing of IL-3R positive tumor cells. Although preclinical, ex vivo evidence that MGD006 causes a cellular immunologically mediated anti-cancer effect against primary human AML samples is encouraging. Initial data from an open-label Phase I study of MGD006 in the advanced AML settings is expected around year-end 2014.

We believe that margetuximab, though not driving investors' interest in MGNX, provides a reasonable floor to the company's valuation, should the more attractive (and novel but risky) assets fail to live up to expectations. We anticipate that margetuximab's superior immunological profile may significantly expand the utility of HER2 targeted therapy beyond breast cancer patients, into the bladder and gastroesophageal settings. Additionally, future combination studies of margetuximab and advancing immunoregulatory proteins (MGA271 for instance) may significantly challenge Roche's HER2 franchise in the market place. A Phase III study of margetuximab in the 3rd line gastric cancer setting is planned for the second half of 2014, and data from an ongoing Phase II study in the metastatic breast cancer setting is expected in 2014.

Valuation

We arrive at our $48 price target by a sum of the parts analysis. We ascribe $19/share to margetuximab in HER2 low expressing MBC and HER2+ R/R gastric cancer based on a typical oncology multiple of 6x on estimated 2021 sales of $600M discounted 30% (MBC) and 35% (gastric) annually. We ascribe $17/share to MGA-271 in renal and melanoma markets based on a 6x multiple of estimated sales and a typical royalty multiple of 15x of estimated royalties discounted 40% annually. We ascribe $6/share to MGD006 in AML patients unfit for intensive therapy and r/r AML based on a 6x multiple of estimated sales and an 18x multiple of estimated royalties, discounted 45% annually. We ascribe $5 to MGD007 in mCRC based on a 6x/15x multiple of sales/royalties discounted 50% annually. We ascribe $1 to MGD010 based on a 6x/15x multiple of sales/royalties discounted 45% annually.

Exhibit 1: Valuation Table

Sales / Royalties	Stage	Valuation Year	Sales/ Royalties	Sales Multiple	Discount Rate	Program Value	Est. Shares (000's)	Per Share
MGA271 in Renal (B7H3)	Phase Ib	2023	$720,851	6	40%	$209,336	30,895	$6.78
MGA271 in Melanoma (B7H3)	Phase Ib	2023	$635,291	6	40%	$184,489	30,895	$5.97
MGD006 in AML (CD3xCD123)	Phase Ia	2022	$368,230	6	45%	$113,065	30,895	$3.66
MGD007 in mCRC (CD3xGPA33)	Pre Clinical	2022	$450,000	6	50%	$105,350	30,895	$3.41
MGA271 Royalties in Renal	Phase Ib	2023	$91,510	15	40%	$66,437	30,895	$2.15
MGA271 Royalties in Melanoma	Phase Ib	2023	$78,778	15	40%	$57,193	30,895	$1.85
MGD006 Royalties in AML	Phase Ia	2022	$86,428	15	45%	$66,344	30,895	$2.15
MGD007 Royalties in mCRC	Pre Clinical	2022	$83,831	15	50%	$49,065	30,895	$1.59
MGD010 Royalties in RA	Pre Clinical	2023	$76,385	15	45%	$40,438	30,895	$1.31
Early Stage Products Subtotal						*$891,717*		*$29*
Margetuximab in MBC (HER2)	Phase IIa	2021	$522,144	6	30%	$499,273	30,895	$16.16
Margetuximab in Gastric (HER2)	Phase I	2021	$107,324	6	35%	$78,797	30,895	$2.55
Margetuximab Subtotal						*$578,070*		*$19*
Total						***$1,469,786***	***30,895***	***$48***

Source: Oppenheimer & Co. Inc.

Key Risks to Our Price Target

Clinical Risk. We would expect a material decline in MGNX shares in the event of unsuccessful clinical data for any of MacroGenics' 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. MacroGenics will be competing against numerous other agents both in oncology and auto-immune markets. Physicians may be more comfortable with previous products and fail to prescribe MacroGenics' novel therapies. Furthermore MacroGenics 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 MacroGenics' patent portfolio. Upon expiration of patents, MacroGenics may be unable to prevent third parties from creating biosimilar copies of MacroGenics' products. Furthermore, competitors may challenge the scope/validity of the patents, or simply find ways to circumvent them.

Manufacturing Risk. MacroGenics 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 the market share and revenue.

Strategic Risk. If MacroGenics 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 results in a material decline in the share price of the company's securities

Insider Ownership Risk, MacroGenics' directors, executive officers and principal stockholders, together with their affiliates and related persons beneficially own a substantial portion of MacroGenics outstanding stock (78% as of December 2013). The interests of these parties may not be aligned with the interests of public shareholder.

Upcoming Milestones

H2:14	Initiate a Phase III trial (MAGENTA) of margetuximab (anti-HER2) in HER2+ gastric cancer
H2:14	Initiate a Phase I trial of MGD007 (GPA33xCD3) in advanced cancer patients
YE:14	Complete enrollment in the first stage of the simon two stage Phase IIa trial of margetuximab in MBC, announce decision to enlarge/cancel
2014	Initiate additional dose expansion cohorts with MGA271 (anti-B7-H3) in undisclosed tumor types
YE:14	Complete first three dose expansion cohorts in a Phase I trial of MGA271 (anti-B7-H3)
2015	Potential expansion cohort data from the Phase I Trial of MGA271 at a scientific meeting
2015	File two additional DART INDs
2015	Potential initiation of Phase I/II trials of MGD010 in auto-immune diseases
2015	Potential initiation of Phase II trials of MGA271 in combination with other agents
2015/2016	Top-line data from Phase I trials of MGD006 (CD123xCD3) and MGD007 (GPA33xCD3) in advanced cancer patients
2017	Complete enrollment in a Phase III trial of margetuximab in gastric cancer, MAGENTA
2017/2018	Top-line data from a Phase III trial of margetuximab in gastric cancer, MAGENTA

Company Overview

MacroGenics is an immuno-oncology focused company developing advanced antibody technologies and bispecific T-cell redirecting antibodies based on the company's DART platform. MacroGenics also develops therapies aimed at novel targets identified through their proprietary cancer stem cell technology. Cancer stem-like cells are super-regenerative cells that disproportionately impact tumor growth and survival. Targeting these cells could deplete a tumor's regenerative capability and inhibit recurrence and metastasis.

Exhibit 2: MacroGenics' Pipeline

Drug	Target	Type	Indication	Status / Next Event	Partner (Region)
Margetuximab	HER2	Fc-enhanced mAb	Gastroesophageal	PIII planned H2:14, data 2016/2017	-
			MBC	Phase II underway, data H2:14	-
			Other Solid Tumors	Phase I complete	-
MGA-271	B7-H3	Fc-enhanced mAb	Solid Tumors	Phase I data YE:14	Servier* (ex NA)
MGD006	CD123 (IL3-R)xCD3	T-cell redirecting DART	AML / BPCDN	Phase I data H2:15/2016	Servier* (ex NA)
MGD007	GPA33xCD3	T-cell redirecting DART	Colorectal cancer	Phase I initiation planned H2:14	Servier* (ex NA)
MGD010	CD32(FcγRIIb)xCD79b	DART	SLE / RA	Partnered with Takeda Q1:14	Takeda (WW)

*Servier has rights to compounds they license in all territories except the US, Canada, Mexico, Japan, South Korea and India

Source: MacroGenics, Inc.

MGA271—B7-H3 Immunotherapy

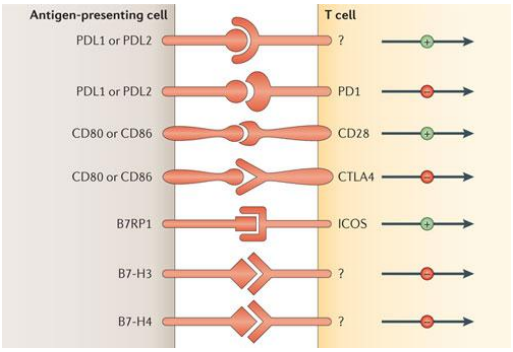
MacroGenics' MGA271 is an Fc-optimized Mab that both targets and blocks the B7-H3 T cell inhibitory antigen on tumor and tumor vasculature currently in a Phase I monotherapy trial in patients with advanced solid tumors. MGA271 is an Fc-enhanced antibody that exhibits enhanced ADCC capability. B7-H3 is a recently identified member of the immuno-modulatory B7 family of ligands (Exhibit 4). MGA271 is being developed in partnership with French pharmaceutical company Servier, and MacroGenics retains commercial rights in North America, Japan, Korea and India. Potential efficacy data from an on-going Phase I study is anticipated H2:15.

B7-H3 May Be Immuno-Modulatory and Demonstrate Efficacy Across Multiple Tumor Types

B7-H3s may have an immune-modulatory role similar to other checkpoint proteins and ligands that are transforming cancer immunotherapy (Exhibit 4). Since many tumors upregulate B7-H3, it is likely that its role is advantageous to tumor immune escape (Exhibit 6). MGA271's potential differentiation from other immune-modulator arises from the restricted expression pattern of B7-H3 to tumor and tumor vasculature and the antibodies dual B7-H3 signal blockade and ADCC activity.

We expect that MGA271 will synergize with other immunologically active therapies and/or following the appropriate conditioning regime to deplete T-regulatory cells. B7 ligands bind to receptors on lymphocytes and regulate immune response. This family includes the PD-1 ligand PD-L1 and the CTLA-4 ligands, B7-1 and B7-2.

Exhibit 4: Checkpoint Proteins and Ligands in Cancer Immunotherapy



Source: Pardoll et al. Nat Rev Cancer. 2012 Mar 22;12(4):252-64

Immune regulatory compounds, especially checkpoint inhibitors, have the promise to be effective across many indications, in contrast to current targeted therapies. A CTLA-4 inhibitor, Yervoy (ipilimumab), was the first checkpoint inhibitor to be approved, demonstrating a long-term survival benefit in melanoma. There are three PD-1/PD-L1 inhibitor antibodies in late-stage development from Bristol-Meyers Squibb, Merck and Roche (Exhibit 5). These antibodies have demonstrated excellent safety and strong efficacy data in melanoma and NSCLC. Merck's pembrolizumab has a PDUFA of October 28, 2014; BMS anticipates completing a rolling BLA for nivolumab by year-end 2014; and Roche has just initiated pivotal trials of MPDL3280A in metastatic bladder cancer.

Exhibit 5: Select Immuno-modulatory Therapeutics

Drug	Other Names	Developer	Target	Intended Effect	Compound Type	Status	Indication(s)
Nivolumab	MDX-1106, BMS-936558	BMS	PD-1	Checkpoint inhibitor	Human, IgG4	BLA complete by YE:14	Melanoma, NSCLC, RCC
Pembrolizumab	MK-3475, lambrolizumab	Merck	PD-1	Checkpoint inhibitor	Humanized, IgG4, Fc-KO	PDUFA Oct. 28, 2014	Melanoma
Pidilizumab	CT-011	CureTech	PD-1	Checkpoint inhibitor	Humanized, IgG1	Phase II	Melanoma, pancreatic, lymphoma, CRC, RCC
AMP224		GSK/Amplimune	PD-1	Checkpoint inhibitor		Phase I	Melanoma, ovarian
AUNP-12	NP-12	Aurigene/Pierre Fabre	PD-1	Checkpoint inhibitor	29aa peptide engineered from PD-L1/L2 binding domain	Preclinical	-
MEDI-4736		MedImmune	PD-L1	Checkpoint inhibitor	Human IgG1	Phase III	NSCLC
MPDl3280A		Roche	PD-L1	Checkpoint inhibitor	Human, IgG1, Fc-KO	Phase III	Melanoma, NSCLC, RCC, Colon, Gastric, HNSCC, Lymphoma, Bladder
BMS-936559		BMS	PD-L1	Checkpoint inhibitor	Human IgG4	Phase I	HIV
Yervoy	ipilimumab, MDX-010, MDX-101	BMS	CTLA4	Checkpoint inhibitor	Human, IgG1	Marketed	Melanoma (approved)
tremelimumab	ticilimumab, CP-675,206	MedImmune/Pfizer	CTLA4	Checkpoint inhibitor	Human, IgG2	Phase II	Melanoma, mesothelioma, NSCLC
AMG557		Amgen	B7RP-1	Immune Activator	Human	Phase I	SLE
MGA271		MacroGenics	B7-H3	Checkpoint inhibitor	Human, Fc-enhanced	Phase I	Solid Tumors
IMP321		GSK/Immutep	MHCII	Checkpoint inhibitor	Soluble LAG3	Phase I	Melanoma., MBC, mRCC
CDX-1127		CellDex	CD27	Immune Activator	Human IgG1	Phase I	Advanced Tumors
Indoximod		NewLink Genetics	IDO	Immune Activator	Human IgG	Phase II	MBC, mCRPC, melanoma

Source: Oppenheimer & Co. Inc.

B7-H3 Is Over-Expressed in a Broad Set of Solid

Tumors

MacroGenics identified B7-H3 as a target that is over-expressed on cancer cells, specifically on cancer stem-like cells, with the use of the company's proprietary library of cancer stem-like cells. B7-H3 is an inducible ligand present on activated T, B and DC cells as well as monocytes. Immunohistochemical studies have identified B7-H3 on an array of solid tumors including kidney, glioblastoma, thyroid, gastric, breast, pancreas, prostate, melanoma, and ovarian cancers (Exhibit 6) and colorectal cancer.

Exhibit 6: Relevant Clinical Studies of B7-H3 Expression and Prognosis

Reference	Malignancy	N	% B7-H3 Positive by IHC	% B7-H3 ≥2+ by IHC	Positive / negative correlation	Synopsis
Crispen 2008	RCC	743	99%	96%	-	Either tumor cell or diffuse tumor vasculature B7-H3 expression was significantly associated with an increased risk of death from ccRCC
-	Glioblastoma	-	98%	95%	-	-
-	Thyroid cancer	-	97%	94%	-	-
Wu 2006	Gastric	102	87%	87%	+	B7-H3 expression was associated with better postoperative survival
-	Breast Cancer	-	73%	70%	-	-
Loos 2009	Pancreatic cancer	68	88%	58%	+	High tumor B7-H3 expression associated with better postoperative prognosis
Yamato 2009	Pancreatic cancer	59			-	Strong tumor B7-H3 expression was significantly associated with lymph node metastasis and advanced pathological stage
Parker 2010	Prostate	148	89%	46%	-	Increased risk of biochemical recurrence for patients with moderate and marked B7-H3 staining
Zang 2007	Prostate	823			-	Patients with strong tumor B7-H3 expression were sat significantly increased risk of recurrence and cancer-specific death
Roth 2007	Prostate	338			-	B7-H3 intensity correlated with tumor volume, extra-prostatic extension, Gleason score, seminal vesicle involvement, surgical margins; Marked B7-H3 intensity associated with cancer progression
-	Melanoma	-	94%	46%	-	-
Zang 2010	Ovarian carcinoma	103	72%	35%	-	Significantly shorter OS and higher incidence of recurrence for patients with B7-H3+ tumor vasculature
Sun 2010	Colorectal	102	-	-	-	Higher tumor B7-H3 is correlated with more advanced tumor grade
Greorio 2008	Neuroblastoma	53	-	-	-	High tumor B7-H3 expression was associated with a worse event-free survival
Sun 2006	NSCLC	70	-	-	-	High tumor B7-H3 was significantly more common in cases with lymph node metastasis

Source: Adapted from Loos et al. Clin Dev Immunol. 2010;2010:683875 and MacroGenics, Inc.

Phase I Trial—MGA271, Safe and Well Tolerated, Appears Well Suited for Combination Immunotherapy

MGA271's initial preliminary Phase I results are, in our opinion, promising given the highly refractory patient population, safety and potential for combination therapy with the product. Initial results from a single-dose, dose-escalation Phase I trial of MGA271 in 26 patients with B7-H3 over-expressing carcinoma, melanoma, or glioblastoma were positive with 10/26 (38%) achieving objective responses (all stable disease) and received further doses of MGA271. The dose-limiting toxicity (DLT) was not reached, and the most frequent adverse events (AEs) were mild to moderate infusion reactions.

In August 2013, MGNX began dosing patients in an expansion phase of the trial evaluating MGA271 at a weekly dose (IV) of 15 mg/kg in three cohorts. Of these patients, 45 will be enrolled in three cohorts, two distinct 15-patient cohorts of undisclosed tumor types and a third comprised of all B7-H3 overexpressing tumors. MacroGenics anticipates completion of the dose expansion phase by year-end 2014 and plans to initiate further monotherapy trials in 2H14 as well as combination trials in 2H15.

Checkpoint Inhibitors Highlight MGA271's Immune-modulatory Potential

We anticipate that comparisons of immunomodulatory agents (with obvious caveats) across trials are likely, and that these will help elucidate B7-H3's role in immune modulation and potential solid tumor activity. We anticipate that significant de-risking and value will be attributed to MGA271, should results equal or rival those of other immunomodulatory therapies and combinations in the clinic. (Exhibit 7).

Nivolumab, an anti-PD-1 antibody being developed by BMS, has been tested in renal cancer in two trials, CheckMate-016 and Check-Mate-010. CheckMate-016 is a multi-arm Phase Ib trial that enrolled 44 RCC patients either previously treated (n=34) or treatment naïve (n=10) to receive nivolumab and Yervoy. Patients were randomized 1:1 to either nivolumab 3 mg/kg + Yervoy 1 mg/kg or nivolumab 1 mg/kg + Yervoy 3 mg/kg, Q3W for 4 doses, both followed by nivolumab 3 mg/kg Q2W until progression or toxicity. Responses in both arms of the trial were similar.

CheckMate-010 is a Phase II trial enrolling 168 patients with advanced or metastatic renal cell cancer (RCC) with a previous anti-angiogenic therapy, VEGF therapy and ≤3 prior systemic therapies. Patients were randomized 1:1:1 to nivolumab 0.3, 2 or 10 mg/kg received IV Q3W.

IMP321 is a recombinant soluble LAG-3Ig fusion protein being developed by Immutep in partnership with GlaxoSmithKline. A Phase I dose escalation trial (Brignone 2009) in 21 advanced RCC patients found no objective responses. Seven of eight evaluable patients treated at doses > 6 mg achieved stable disease at 3 months compared to 3/11 at < 6 mg.

Exhibit 7: Checkpoint Inhibitors in Renal Cancer

Sponsor	BMS					Roche	GSK/Immutep	CureTech
Trial	Phase I/II - CheckMate-016		Phase I/II - CheckMate-010			Phase I	Phase I	Phase I
Regime	Combination Therapy		Single Agent			Single Agent	Single Agent	Single Agent
Population	Treatment naïve + previously treated RCC patients		RCC with prior angiogenic, VEGF, and <=3 systemic therapies			Metastatic RCC	Advanced RCC	Stage IV RCC
Drug(s)	Nivolumab(N) + Yervoy (Y) Q3w for 4 doses, followed by N		Nivolumab Q3W			MPDL3280A Q3W	IMP321 biweekly for 6 cycles	Pidilizuamab + Dendritic cell vaccine
Class	PD-1 + CTLA4		PD-1			PD-L1	soluble Lag3	PD-1
Arm	N 3 mg/kg + Y 1 mg/kg	N 1 mg/kg + Y 3 mg/kg	0.3 mg/kg	2 mg/kg	10 mg/kg	3-20 mg/kg	0.05-30 mg	Ongoing
N	21	23	60	22	20	47	21	Est. 44
ORR	43%	48%	20%	20%	20%	13%	0%	Ongoing
SD	**24%**	**35%**	**-**	**-**	**-**	**60%**	**52.6%**	**-**
Median DOR (wks)	21+	NR	11.7	17.3	18.2	-	N/A	-
Median PFS (wks)	37	38	78.9	110.5	107.0	-	NR	-
Other	24wk PFS: 65%	24wk PFS:64%	1yr OS: 63%	1yr OS: 72%	1yr OS: 70%	24wk PFS: 50%	NR	-
AEs	NR	NR	TE-SAEs: 3.4%	TE-SAEs: 11%	TE-SAEs: 7.4%	G3/4 TEAEs in 13%	no clinically significant TEAEs	-
NCT	NCT01472081		NCT01354431			NCT01375842	NCT00351949	NCT01441765

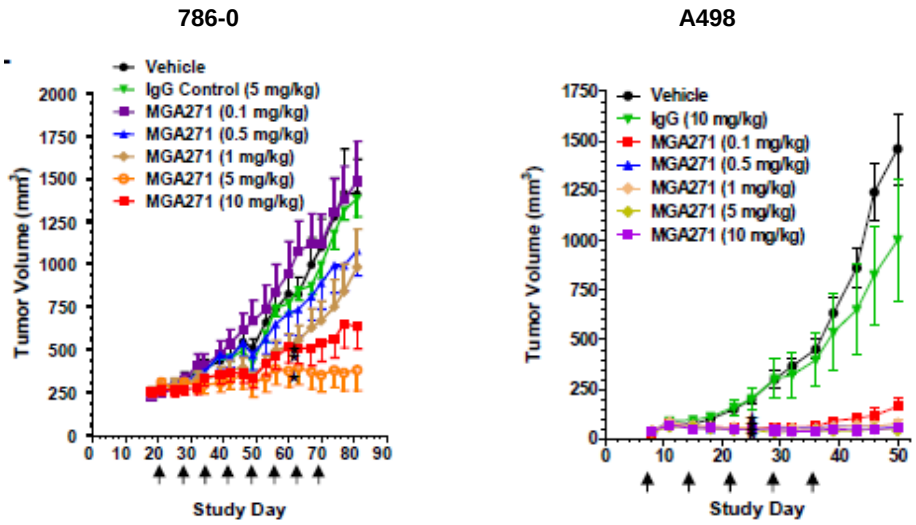
- = not reported or not applicable

Source: Oppenheimer & Co. Inc.

Preclinical Results Suggest Activity as a Monotherapy

MGA271 is effective *in vivo* against 786-0 (renal cancer) and A498 (renal cancer) xenograft models in transgenic mice. The compound exhibited a potent cytostatic effect in these models (Exhibit 8). MGA271 was well tolerated up to 150mg/kg the highest dose tested, in cynomolgus monkey trials, and had an 8-12-day terminal half-life sufficient to support weekly dosing.

Exhibit 8: MGA271 Exhibits a Potent Cytostatic Effect in Renal Cancer Xenograft Models



Source: Loo et al. Clin Cancer Res. 2012 Jul 15;18(14):3834-45

Enhanced ADCC

MGA271 also exhibits enhanced antibody-dependent cell-mediated cytotoxicity (ADCC) compared to non-Fc enhanced B7-H3 directed antibodies *in vitro* through incorporation of MacroGenics' Fc-engineering technology. ADCC enhancement is particularly notable in PBMCs homozygous for the lower-affinity FcγRIIIa receptor allele 158F, compared to PBMCs heterozygous or homozygous for the higher-affinity allele 158V. The 158F allele is correlated with poorer response to antibody therapy.

MGA271 Market Opportunity

MGA271, if demonstrated to have significant immuno-modulatory activity, may find utility in in a broad range of oncology settings. We highlight recent positive data for Bristol-Myers Squib's nivolumab in renal cell and others both as a single agent and in combination with ipilimumab. Since mechanistically we anticipate broad applicability of MGA271 across solid tumor types but lack cancer specific data, we have chosen to value MGA271 on an opportunity in renal cancer and melanoma. We believe these are reasonable surrogates for potentially applicable solid tumor markets with significant unmet medical need.

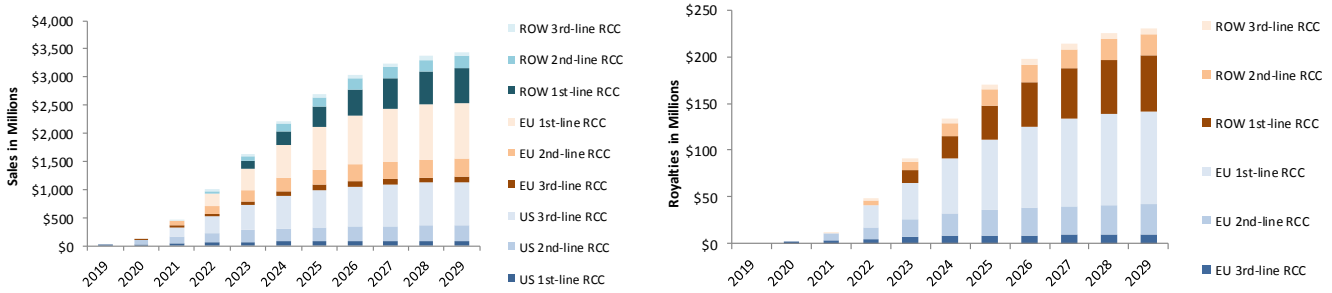
Renal and Melanoma Cancer—Example Indications and Markets for MGA271

Some 65,000 patients are diagnosed with renal cancer in the US each year, 16% of which are metastatic. Locally advanced RCC (17% of diagnoses) is treated by resection with recurrence rates of ~20%. Localized renal cancer (64% of diagnoses) is generally resectable, with recurrence rates of 2-4%.

Roughly 70-85% of RCCs are clear cell, 7-15% are papillary, 5-10% are chromophobe. Chemotherapy is ineffective against RCC, and treatment relies on TKIs, mTOR inhibitors, angiogenesis inhibitors and interferon treatments. Interferon-α treatment has long been the mainstay of care due to the ease of use (home administration), despite low response rates of ~15% and no durable remission. Cytokine therapy with high dose IL-2 is the only treatment that has demonstrated long-term durable remissions, albeit in only 5% of patients. TKIs and mTOR inhibitors are used as first- and second-line therapy with the sequence and drug dependent on prognosis. Roughly 16% of patients will receive third-line therapy, while another 27% will receive second-line therapy only.

We estimate that if MGA-271 receives approval in mRCC and melanoma, peak sales could exceed $5 billion at peak. We estimate that MGA271 could receive approval in third-line clear-cell RCC in 2019, with second and first-line approvals following in 2020 and 2021. We estimate that it could achieve 40%, 50% and 60% penetration in third-line, second-line, and front-line respectively and be priced at $10,500 per 28-day cycle, in line with Sutent (sunitinib, indicated for RCC, GIST and pNET), and patients may receive an average of 10 cycles.

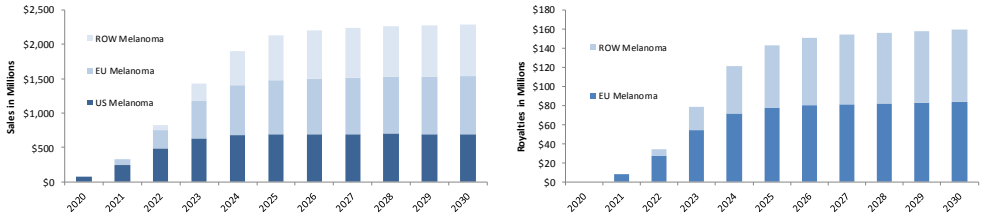
Exhibit 9: MGA271 Sales Estimates in mRCC



Source: Oppenheimer & Co. Inc.

We see potential upside to our sales expectations in B7-H3+ cancers beyond renal cancer. Melanoma, for instance, is a particularly attractive market where a checkpoint inhibitor (Yervoy) has already demonstrated efficacy. We estimate there are 76,000 US melanoma patients diagnosed each year, 82,000 in the EU and 74,000 in rest-of-world (ROW) markets. We estimate 50% are available for treatment with an immunotherapy and that MGA271 may achieve 25% penetration in the available patient population due to competition from Yervoy. We estimate patients may receive an average of 7 cycles of therapy. We estimate that MGA-271 is worth $17/share to MacroGenics based on a 6x/15x multiple of sales/royalties discounted 40% annually.

Exhibit 10: MGA271 Sales Estimates in Melanoma



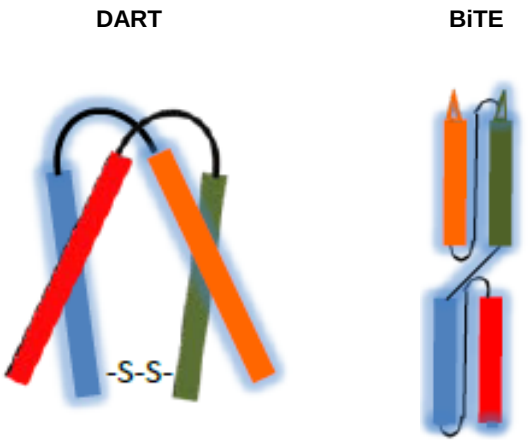
Source: Oppenheimer & Co. Inc.

DARTs—Bispecific Targeting of T-Cells and the Immune System

MacroGenics has developed DARTs, a bispecific antibody technology that can be produced in high yields and incorporate Fc-domains to extend half-life. Numerous bispecific antibody technologies have been developed; however, most constructs currently suffer from poor manufacturability (Removab (catumaxomab), blinatumomab) and/or short half-life (blinatumomab). MacroGenics can produce bispecific antibodies in high yields and has demonstrated ng/ml potency in preclinical models.

MacroGenics is developing three DART molecules, one currently in the clinic, MGD006 (CD123xCD3) in a Phase I trial, and two others, MGD007 (GPA33xCD3), MGD010 (CD32BxCD79B) in preclinical work. MGD007 is expected to enter the clinic in 2H14. Servier has rights in certain territories outside of North America to MGD006 and MGD007. Japan-based Takeda holds a worldwide license to MGD010.

Exhibit 11: DART Bispecific Formats (DART vs BiTE)

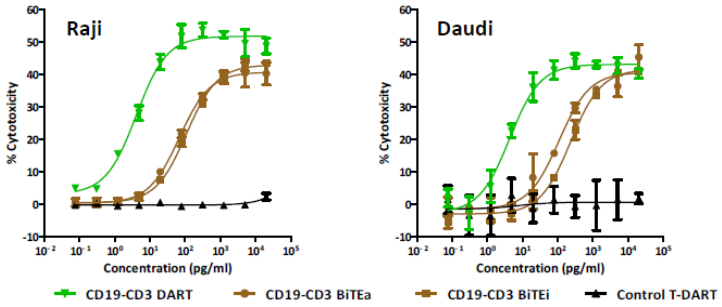


Source: MacroGenics, Inc.

DARTs Enable Better Bispecifics

DARTs, or dual affinity retargeting antibodies, consist of two chains each consisting of a V_H and a V_L domain linked with a peptide spacer. The two chains are dimerized so that each binding domain contains a V_H from one chain and a V_L from the other. A disulfide bond forms between the two V_H domains, stabilizing the molecule. These structural changes may aid the potency DARTs relative to BiTE technologies (Exhibit 12). Additionally, DARTs can be produced by standard antibody techniques in yields up to g/L, in contrast to earlier generation bispecific T-cell engagers, or BiTEs, such as blinatumomab with yields as low as 4 mg/L in early work (Löffler 2000). Finally, MGNX's knock-out Fc-domains incorporated into DARTs are capable of facilitating IgG-like half-lives (7 days vs. 1 day).

Exhibit 12: Darts Are More Potent than BiTEs *in vitro*



Source: MacroGenics, 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, and are 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 in oncology when combined with immuno-modulatory compounds (PD1, PDL1, CTLA4, B7H3, CD27) that inhibit factors that aid a tumor's immunological escape could potentially offer significant efficacy.

Bispecific Technologies in Development

Bispecific antibodies have been developed in a variety of formats. The most advanced is 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 the company's only approved drug, Removab (ertumaxomab), never received US approval.

General Properties of Advanced Bispecific Technologies:

- BiTEs (AMGN–Perform) may have a limited field of practicality 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 7 days. Preclinical work on DARTs also suggests that they may be more potent than Amgen's BiTE technology (Exhibit 12).

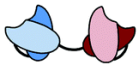
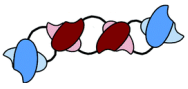
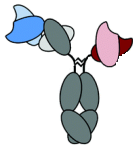
- Xencor's (XNCR–Outperform) XmAb bispecifics have an IgG-like long half-life (6-7 days) through incorporation of their KO Fc-domain and contain no peptide-linkers facilitating straightforward manufacturing and potentially significantly lower cost of goods sold.

- 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 (1 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 13: 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 the low-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 among other side effects.

Exhibit 14: 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 patients	"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.

MGD006, A DART for AML—Targeting T-Cells to CD123 (IL3R)

MGD006 is a CD123 x CD3 bispecific T-cell redirecting DART. MGD006 is also MacroGenics' first DART molecule to enter the clinic. 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 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 15).

In June 2014, MacroGenics initiated a Phase I trial of MGD006 in patients with relapsed or refractory AML. We anticipate initial data from the study around year-end 2015.

Exhibit 15: CD123 Expression on Rare Cancers

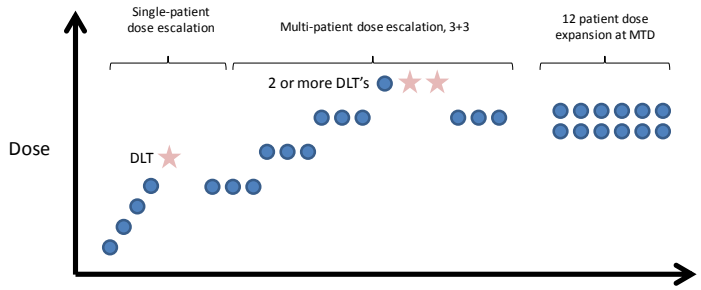
Indication	Reference
Mastocytosis	Teodosio 2010, 2013
Hypereosinophilic syndrome	Brooks 2013
Hairy cell leukemia	Shao 2013
Chronic myelomonocytic leukemia	Orazi 2006
Basophilic Leukemia	Yokohama 2002
B Cell-Acute Lymphoblastic Leukemia	Lhermitte 2006
T Cell-Acute Lymphoblastic Leukemia	De Smet 2012

Source: Oppenheimer & Co. Inc.

The First DART Enters the Clinic: MGD006 Phase I Trial

The Phase I, dose-escalation trial will enroll up to 58 patients with R/R AML to assess the safety and tolerability of MGD006. The trial will be conducted in three stages including a single-patient dose escalation, a multi-patient dose escalation segment in a standard 3+3 design and a 12-patient MTD expansion cohort segment once MTD (maximum tolerated dose) is reached (Exhibit 16).

Exhibit 16: MGD006 Phase I Trial Design



Source: Oppenheimer & Co. Inc.

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

We note that current therapies targeting IL-3R for leukemic stem cells in patients with AML have provided strong clinical validation for 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, and MSD, among others (Exhibit 17).

We highlight that BPDCN (blastic plasmacytoid dendritic cell neoplasm), an extremely rare cancer categorized as a subtype of AML and may represent a unique target sub-set population for MGD006 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 (POC) and clinical indicators of improvement. The disease is also associated with a poor prognosis and a median survival of less than 18 months possibly facilitating efficient overall survival studies, though recruitment for such a study could be slow. We estimate that roughly 150 (and as many as 1,000) patients 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 in development; however, those currently in the clinic do not actively engage T-cells like MGD006. The most advanced clinical candidate is Stemline's SL-401, an immunotoxin consisting of a modified diphtheria toxin fused to the IL3 protein (Exhibit 17). SL-401 recently completed a Phase I trial demonstrating that it is safe and well tolerated and highly active in BPDCN (Frankel 2013). CSL has also tested Fc-enhanced and chimeric monoclonal antibodies against CD123, though we anticipate these approaches will be less potent than MGD006, a T-cell redirecting DART.

Exhibit 17: Other CD123 Directed Therapies in Clinical Development

Company	CSL Limited	CSL Limited	Stemline (STML)
Name	CSL362	CSL360	SL-401
Type	Fc-enhanced mAb	Chimeric mAb	mAb-diptheria toxin
Status	Phase I initiated Aug. 2012	Phase I complete 2012, not continued	Phase I/II
Indication	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	Est. 36	40	81
Dose / Schedule	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	MTD not reached	16.6 µg/kg/day, DLTs of hypoalbuminemia and edema
AEs	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	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	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	NCT01632852	NCT00401739	NCT02113982

Source: Oppenheimer & Co. Inc.

CD123-Directed CAR-T Analogs Look Promising Preclinically

Several different CAR-T therapies directed against CD123 have recently demonstrated promising results in preclinical models, further supporting the notion that redirecting T-cells to CD123 expressing cells may be a viable therapeutic option (Mardiros 2013, Gill 2013, Tettamanti 2013).

Gill et al. found near complete eradication of bone marrow cells, while the Mardiros et al. group found limited (<50%) killing of healthy progenitor cells incubated with CAR-T cells.

A CD123xCD3 tetravalent bispecific consisting of two CD123 Fv regions fused to an Fc region, with a pair of CD3 Fv regions fused to the C-terminus (tail of the Y) end of the Fc domain, demonstrated redirected T-cell killing at picomolar concentrations *in vitro*. Yields are still low, however, in the 2-5 mg/L range, potentially limiting the commercial viability of the approach (Kuo 2012).

Researchers in Germany also demonstrated that a CD123xCD16 bispecific scFv molecule achieved potent ADCC capacity against AML cells *in vitro*. CD16 is another name for FcγRIII, an activating Fc receptor present on NK-cells that is primarily response for antibody-dependent cell-mediated cytotoxicity (ADCC).

Exhibit 18: CD123-Directed Therapies in Preclinical and Clinical Development

Type	CAR-T	CD123xCD3	CD123xCD16	DIP-IL3 fusion	CD123 mAb	CD123xCD3	CD123xCD3
Company	Academic	Academic	Academic	Stemline	CSL	Xencor	MacroGenics
Drugs	-	-	-	SL-401	CSL362, CSL360	CD123xCD3	MGD006
Notes	Effective in AML xenograft models, Gill found complete bone marrow eradication	Effective against AML cell lines, low yields	Effective against AML cell lines, low yields	Effective in a Phase I trial in BPDCN	Chimeric Ab ineffective, human, Fc-enhanced trial underway	IgG-like T-cell redirecting bispecific	DART T-cell redirecting bispecific
Reference	Mardiros 2013, Gill 2013, Tettamanti 2013	Kuo 2012	Stein 2009	Frankel 2008	Roberts 2010	Unpublished data	Hussaini 2013

Source: Oppenheimer & Co. Inc.

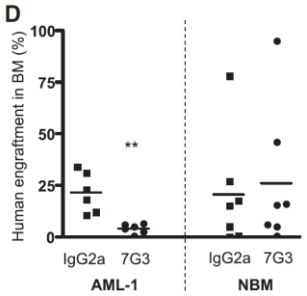
CD123 Is Present on Cancer Stem-like Cells, and Inhibition Inhibits Engraftment in Preclinical AML Models

MGD006, by targeting CD123, may facilitate T-cell destruction of cancer stem-like cells in the most difficult to treat AML patients.

IL3 is a cytokine that stimulates proliferation and maturation in 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 negative 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).

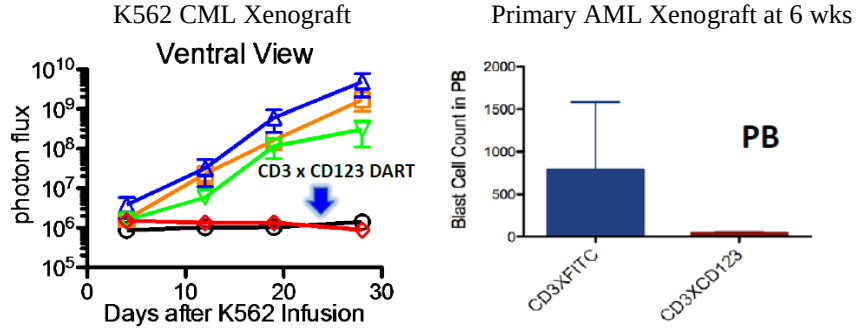
Exhibit 19: Anti-CD123 Antibody 7G3 Inhibits AML Engraftment but not NBM engraftment in SCID mice



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

MGD006 suppressed leukemia growth both in primary (patient-derived) AML and cultured CML xenograft murine models. In primary AML xenograft models, a CD3xCD123 DART reduced blood plasma, spleen and bone marrow blast counts.

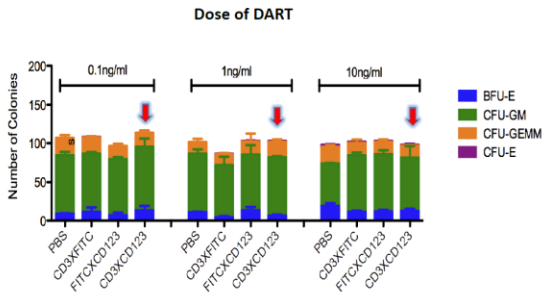
Exhibit 20: MDG006 Suppresses Leukemia Growth in a K562 Xenograft Murine Model



Source: MacroGenics, Inc.

Importantly, MGD006 does not kill non-malignant progenitor cells such as CFU-GEMM cells in umbilical cord blood (Exhibit 21).

Exhibit 21: MGD006 is Not Cytotoxic against Umbilical Cord Blood Cells



Source: MacroGenics, Inc.

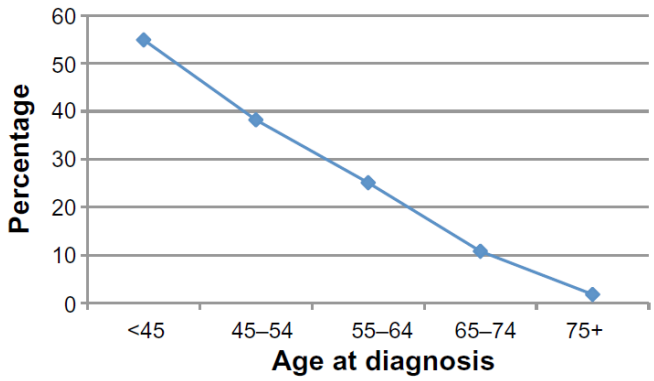
MGD006—Potential Market in AML

MGD006, if demonstrated to have significant activity in AML, may find utility in in a broad range of oncology settings where CD123 is expressed. However, although potentially broadly applicable we choose to assign value to MGD006 in reference to a potential market in AML, noting that 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 22), in part due to the use of ASCT (autologous stem cell transplant), which is not performed in elderly patients.

SEER-Medicare data indicate than 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.

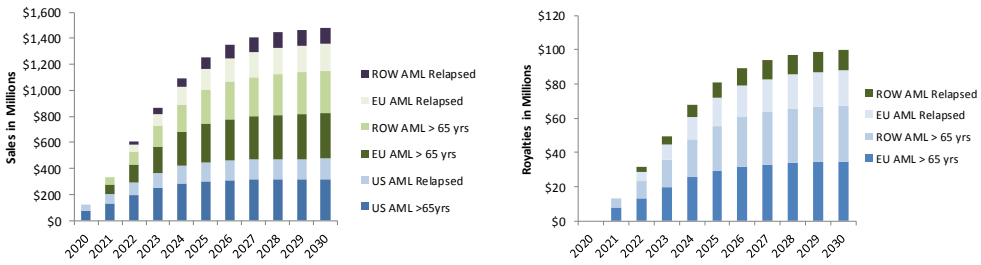
Exhibit 22: 5 Year Survival by Age of Diagnosis in AML



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

We estimate that if MGD006 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 $1.3 billion. We estimate that MGD006 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 MGD006 may achieve 60% penetration in the 4,500 relapsing-remitting (R/R) patients available each year. We estimate MGD006 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 MGD006 could receive approval in 2020. We estimate that MGD006 is worth $6/share to MacroGenics based on a 6x multiple of estimated sales and an 18x multiple of estimated royalties, discounted 45% annually.

Exhibit 23: MGD006 Sales Estimates in Unfit and Relapsed AML Patients



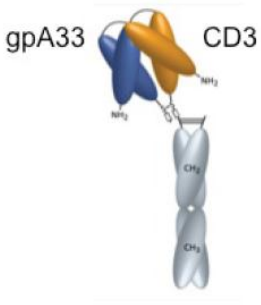
Source: Oppenheimer & Co. Inc.

MGD007—GPA33 x CD3

MDG007 is a GPA33xCD3 T-cell redirecting bispecific DART that contains an FcRn-binding Fc domain to increase half-life through FcRn scavenging. The Fc-domain in MGD007 is modified to reduce binding to immune effector Fc domains, mitigating the risk of cytokine storm or other undesirable immune responses. MGD007 targets glycoprotein-A33 (GPA33), a cell surface glycoprotein expressed on the surface of epithelial cells in the small intestine and colon.

GPA33 is expressed in >95% of colon cancers and >50% of gastric cancers, but has not been detected outside of the intestine. Importantly, this protein is not rapidly internalized in the majority of colon cancer cell lines, allowing a GPA33 directed antibody to accumulate on the cell surface and be available to cells of the immune system. GPA33-directed antibodies can stay localized to the tumor cells for weeks after administration and infiltrate into the center of solid tumors.

Exhibit 24: Structure of MDG007



Source: MacroGenics, Inc.

Naked Anti-GPA33 Antibodies—Safe But Ineffective in the Clinic

Both radio-labeled and naked anti-GPA33 antibodies have been tested in at least five therapeutic clinical trials (Exhibit 25). These trials have demonstrated that GPA33 antibodies are safe, but ineffective, with no reported objective responses to therapy. Several forms of radio-labeled iodine are utilized: 123I, 124I and 125I are gamma-emitting radioisotopes generally used for imaging (PET) though they do have antineoplastic capacity. The beta-emitter 131I is is used therapeutically (e.g., Bexxar/tositumomab).

DART Technology May Offer Differentiated Clinical Activity

We note that since MGD007 not only targets the GPA33 antigen on colorectal cancer cells, but also recruits, expands and activates T-cells via interaction with CD3, an approach that may offer clinical efficacy not seen with other GPA33 directed approaches.

Exhibit 25: Clinical Trials of GPA33 Directed Therapies

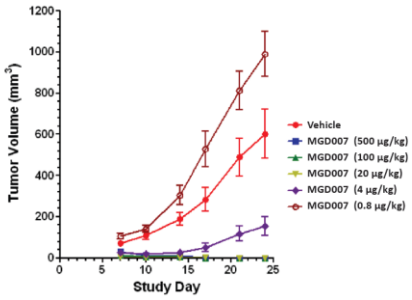
Name	Type	N / Indication	Responses	AEs	Author
KRN330	Humanized mAb	29 / CRC	12/38 (32%) SD	MTD: 3 mg/kg weekly with GI-related DLTs	Infante 2013
-	^{131}I/^{125}I-murine mAb	22 / Colon	12/21 (57%) SD	MTD not reached, transient grade 3 thrombocytopenia in 1 pt	Welt 1996
-	^{131}I/^{125}- humanized mAb	12 / CRC	Not reported	MTD not reached, no TEAEs	Scott 2005
-	^{124}I-humanized mAb	15 / CRC	Imaging study	Imaging study	O'Donoghue 2011
-	^{131}I- humanized mAb	15 / CRC	4/15 (27%) SD	MTD 40 mCi/m^2 DLT of G4 neutropenia / G3 liver function test abnormalities. Cutaneous toxicity in 7 pts	Chong 2005
-	^{131}I- humanized mAb	13 / Gastric carcinoma	No responses reported	MTD not reached, Grade 1 fever only AE	Sakamoto 2006

Source: Oppenheimer & Co. Inc.

Preclinical Results Indicate Promising Efficacy

In preclinical models, MGD007 effectively induced T-cell redirected cytotoxicity *in vitro*. The compound also inhibited tumor growth in colorectal cancer xenograft murine models (Exhibit 26). Female SCID mice were implanted with Colo205 + human T-cells and treated with MGD007 on days 0-3. MGD007 had a half-life of ~7 days.

Exhibit 26: MDG007 Inhibits Tumor Growth in Colorectal Xenograft Models

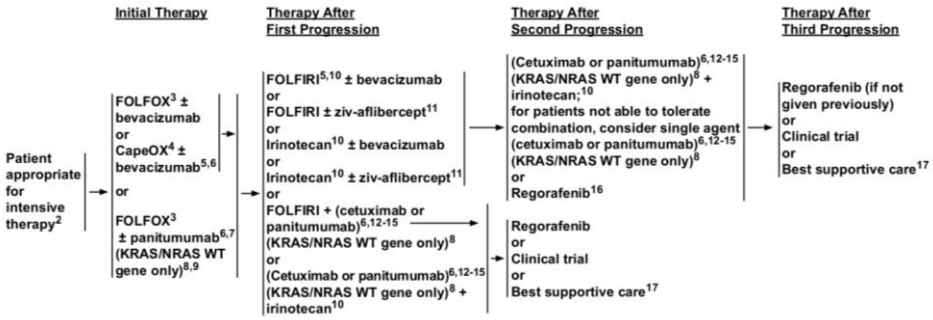


Source: MacroGenics, Inc.

MGD007 Potential Market in the GPA33-Expressing Colorectal Cancer Market

Metastatic colon and rectal cancer are treated with essentially identical drug regimens. Although 75% of colorectal cancer patients are diagnosed with non-metastatic disease, 50% of patients will relapse following front-line resection. The vast majority, ~95%, of colorectal cancers express GPA33. Chemotherapy with 5-FU-based regimens (FOLFOX) is the long-time staple of front-line therapy. Second-line therapy is FOLFIRI, which replaces the oxaliplatin in FOLFOX with irinotecan. In K-RAS wildtype (Wt) patients (~60% of diagnoses), EGFR-targeting Erbitux/Vectibix are effective. Avastin has also demonstrated improved PFS and OS in mCRC. Third/fourth-line therapy is Stivarga (regorafenib) a broad kinase inhibitor, though median OS improved just 20% to 6.4 months from 5 months in the pivotal clinical trial and Grade 3-4 AEs increased to 54% from 14% in the BSC arm.

Exhibit 27: NCCN Guidelines for Rectal Cancer and Colon Cancer



Source: Oppenheimer & Co. Inc.

Stivarga recorded sales of €59M in Q4 (annualized €236M, or ~$400M) after being approved in third-line mCRC in September 2012, and in GIST in February 2013 in the US and in mCRC in the EU in August 2013. Stivarga delays mCRC progression but does not induce clinical response (ORR: 1% in mCRC).

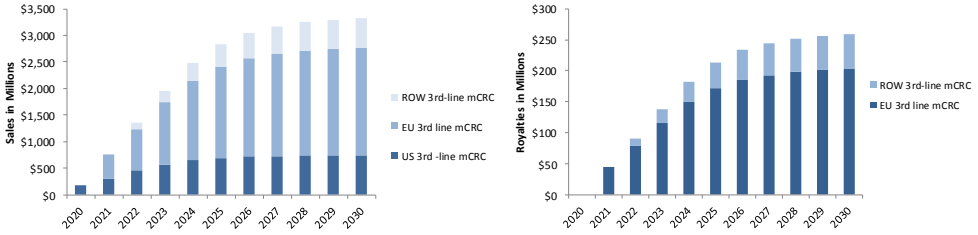
Exhibit 28: Current Colorectal Cancer Drugs

mCRC	First-line	Second-line	Third-line
Drug / Regimen	FOLFOX	FOLFIRI	Stivarga
ORR	40%	31%	1% (DCR 41%)
PFS months	18.7	7 (TTP)	1.9
OS months	61	14	6.4

Source: Oppenheimer & Co. Inc.

If MGD007 receives approval in all lines of metastatic colorectal cancer, we estimate that sales could reach $3B at peak. We estimate there are ~30,000 third-line mCRC patients in the US each year. We estimate that with a conservative 50% penetration into this market, an average of five cycles of therapy MGD007 could achieve peak sales of $750M in the US alone. We anticipate that MGD007 could be approved in third-line mCRC over Stivarga in 2020 and be priced at $10,000 per cycle, in line with Stivarga. We estimate that MGD007 is worth $5/share to MacroGenics based on a 6x multiple of sales and a 15x multiple of royalties discounted 50% annually.

Exhibit 29: Market Estimates for MGD007 in 3rd-line GPA33 Expressing mCRC



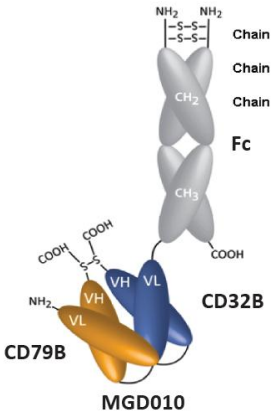
Source: Oppenheimer & Co. Inc.

MGD010—Autoimmune Program

MGD010 is a preclinical DART targeting FcγRIIb (CD32b) and CD79b, a BCR-associated protein. Unlike MGD006 and MGD007, MGD010 does not induce T-cell redirected cytotoxicity, but instead cross-links the FcγRIIb receptor to the BCR, where FcγRIIb inhibits BCR signaling and B-cell activation.

In May, 2014 MacroGenics entered a strategic alliance option-agreement with Takeda for the world-wide rights to MDG010. Under the agreement, Takeda received an option to obtain an exclusive worldwide license for MDG010 following the completion of a pre-defined Phase Ia study. MacroGenics has co-promote rights in the US and may share late-stage costs in exchange for a North American profit share. MacroGenics is eligible for an option license fee and early development milestones totaling $33M and later-stage clinical, regulatory and commercialization milestones worth a further $486.5M. MacroGenics is also eligible for double-digit royalties on global net sales.

Exhibit 30: MGD010 Inhibits Immunoglobulin Production in Transgenic Mice

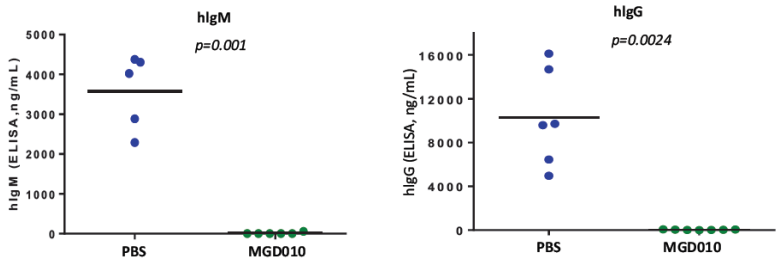


Source: MacroGenics, Inc.

MGD010 inhibits B-cells, but does not kill them as B-cell depleting therapy rituximab (Rituxan/Mabthera) 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. Rituximab, 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).

MGD010 has a half-life of ~6 days, and appears well tolerated as no ADAs (anti-drug antibodies) were found in non-human primate safety studies over 162 days. MGD010 inhibits IgG and IgM production in NOD-SCID IL2Rγ^{null}/Jax (NSG) mice that lack T, B or NK cells, subsequently injected with human PBMCs from healthy donors (Exhibit 31).

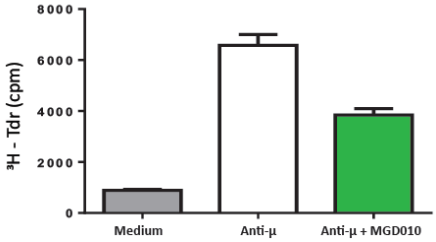
Exhibit 31: MGD010 Inhibits Immunoglobulin Production in Transgenic Mice



Source: MacroGenics, Inc.

MGD010 also inhibit B-cell proliferation in *ex vivo* samples from SLE patients. Purified B cells were incubated with 100nM MGD010 for 30 minutes and stimulated with anti-μ (IgM) antibody for 48 hours.

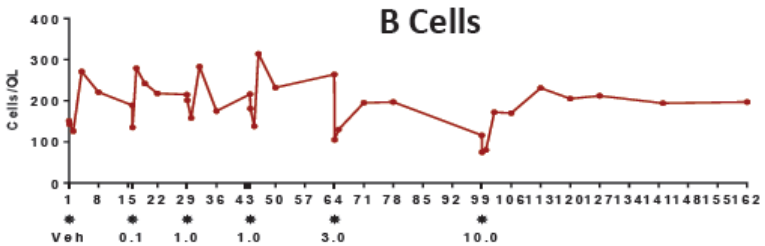
Exhibit 32: MGD010 Inhibits B-cell Proliferation in *ex vivo* SLE Patient Plasma



Source: MacroGenics, Inc.

Importantly MGD010 inhibits, but does not deplete, B-cells in cross-reactive non-human primates.

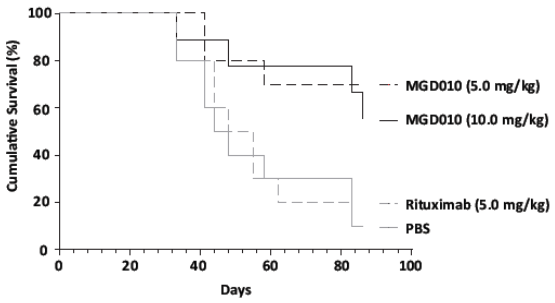
Exhibit 33: MGD010 Does Not Deplete B-Cells in Nonhuman Primates



Source: MacroGenics, Inc.

MGD010 inhibited the development of graft-versus host disease in a humanized mouse model. Adult NSG mice were injected with human PBMCs isolated from a healthy human donor on day 0. Mice were treated with PBS or MGD010 every four days for a total of nine doses. Survival times of MGD010-treated groups were significantly longer than PBS-treated mice by log-rank test ($p<0.05$, 10mg/kg vs. PBS)

Exhibit 34: MGD010 Improves Survival in a Humanized Mouse Model of GVD



Source: MacroGenics, Inc

MGD010 an Alternative to B-cell Depletion for Autoimmune Disease

Current therapies for autoimmune disease leave much to be desired. If found to be safe and effective, MGD010 may find broad utility in autoimmune disease and B-cell associated disorders. Rituxan, an increasingly adopted B-cell depletion therapy for autoimmune disease, improves arthritis symptoms but depresses the immune system and increases infection risk. We believe that MGD010, by inducing inhibition of B-cell activation instead of B-cell depletion, may be similarly efficacious to current B-cell targeted therapies but better tolerated, with a lesser potential infection risk. Additionally, TNF alpha inhibitors are associated with infection risk and other serious side effects, and also frequently stop working in patients after as little as one year on therapy. Ultimately, we believe MGD010 may offer a compromise between B-cell depletion and potentially limited efficacy of TNF-alpha inhibitors, potentially meeting a significant unmet medical need in autoimmune disease.

Takeda may initially develop MGD010 to establish proof of concept in systemic lupus erythematosus (SLE) or rheumatoid arthritis; however, several other orphan indications with high-unmet medical need may offer more targeted and faster routes to registration. We would anticipate, however that eventually the therapy may find off-label use broadly for autoimmune disorders, paralleling the current use of Rituxan and TNF-alpha inhibitors in these indications.

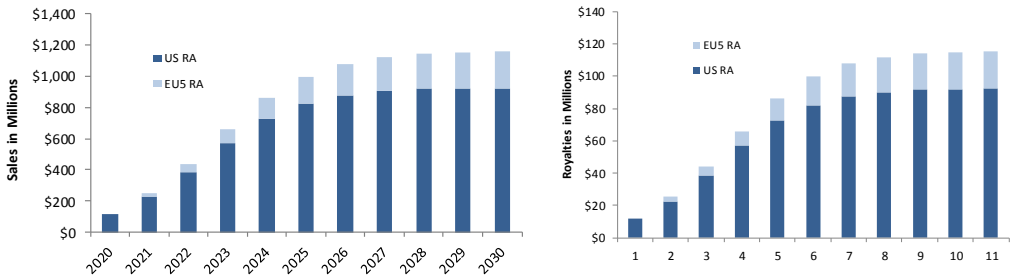
Market—Rheumatoid Arthritis

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

Systemic lupus erythematosus 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 flare-ups still occur regularly. Constant flare-ups eventually lead to renal failure, neurological damage and death. Lupus is estimated to affect 60,000 patients in the US and constitutes a significant unmet medical need.

If MGD010 received approval in RA, we estimate that it could reach peak US and EU5 sales of $1.1 billion. We estimate that there are approximately 370,000 rheumatoid arthritis patients refractory to biologic and DMARD therapy in the US and 85,000 in the top five EU countries. If priced similarly to Humira (TNF-alpha inhibitor) ($25,000/yr), MGD010 could generate $900M in sales in RA alone in the US and $200M in the EU5 with just 10% penetration in this market. We believe upside to our sales expectations exists, should MGD010 show efficacy in additional indications including SLE. We estimate that MGD010 is worth $1/share based on a 6x multiple of sales and a 15x multiple of royalties, discounted 45% annually.

Exhibit 35: MGD010 Sales and Royalty Estimates



Source: Oppenheimer & Co. Inc.

Margetuximab

Margetuximab is a Herceptin (trastuzumab) biobetter that may find utility in lower HER2 expressing cancers where Herceptin is not effective. A biobetter is a related or improved version of an existing biologic drug or new drug candidate. Margetuximab is currently being evaluated in a Phase IIa trial in patients with HER2 low-expressing breast cancer (IHC 2+, FISH-). Additionally, a Phase III in HER2+ (FISH+ or IHC 3+) in patients with gastroesophageal cancer (MAGENTA) planned for H2:14.

HER2 Expression Sets Margetuximab Apart

IHC (immunohistochemistry) and FISH (fluorescent in situ hybridization) measure HER2 antigen and HER2 DNA levels, respectively. IHC is scored on a 0, 1+, 2+, 3+ scale, and FISH is scored as negative or positive based on the ratio of HER2 DNA in the cell, though specific criteria are still under debate. Recall, Perjeta and Kadcyla are currently approved only in patients with FISH+ or IHC 3+, and Herceptin is approved in patients that are FISH+ or IHC 2+/3+, though it is less effective in 2+ patients.

Phase I Results Indicate Promising Activity

Margetuximab's Phase I trial enrolled patients with relapsed or refractory HER2+ cancer to receive IV margetuximab from 0.1-6mg/kg weekly followed by an expansion phase at 6 mg/kg weekly. Margetuximab was well tolerated and demonstrated promising clinical activity. MacroGenics is also investigating Q3W dosing.

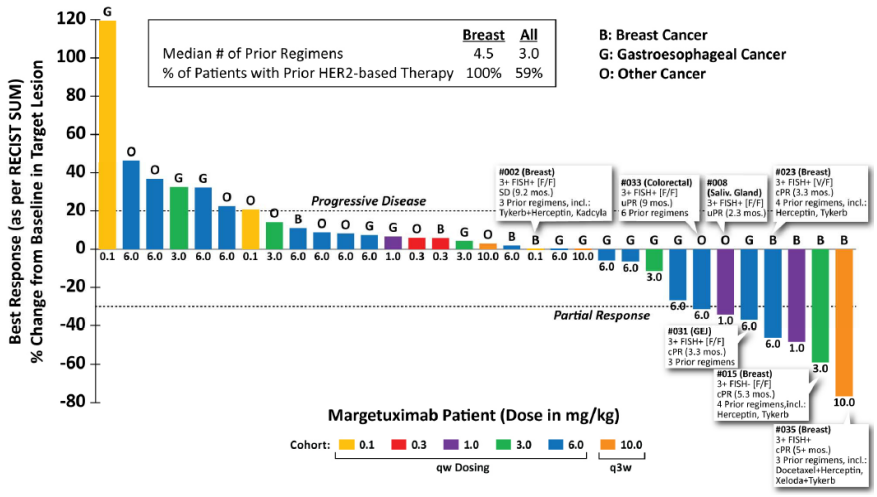
Exhibit 36: Margetuximab Phase I and Comparator

	Margetuximab	Kadcyla Phase I
Design	3+3	3+3
Dose	0.1-6mg/kg weekly, followed by 6 mg/kg QW	0.3 - 4.8 mg/kg Q3W
Indication	R/R HER2+ pts (IHC 2/3+)	R/R HER2+ MBC
N	34	24
Responses	7/32 (22%) had PR, 18 (56%) had SD	6 PRs (25%), of 15 pts treated at MTD (3.6 mg/kg) 33% ORR, 73% CBR
ORR	22%	25%
AEs	AEs ≥ Grade 3 were limited to a single infusion reaction, 2 episodes of brief lymphopenia confounded by steroids, and transient worsening anemia	TEAEs: two G4 thrombocytopenia, one G3 thrombocytopenia, 1 G3 (SAE) pulmonary hypertension
NCT	NCT01195935	NCT00932373

Source: MacroGenics, Inc. Krop et al. J Clin Oncol. 2010 Jun 1;28(16):2698-704

Responses to margetuximab were primarily in breast cancer, with one response in a gastroesophageal cancer patient and one response in a colorectal cancer patient.

Exhibit 37: Responses to Margetuximab



Source: MacroGenics, Inc.

Ongoing and Upcoming Trials

Margetuximab is currently being tested in a Phase II trial (NCT01828021) in R/R MBC patients that are HER2 low-expressers (IHC 2+ / FISH -) with data anticipated in 2H14. MacroGenics also plans to initiate a Phase III trial (MAGENTA) in gastroesophageal cancer in 2H14.

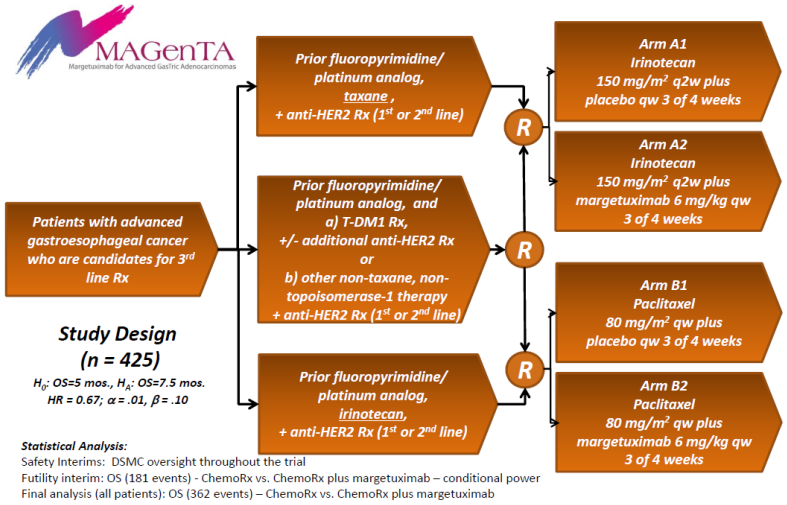
Exhibit 38: Ongoing and Upcoming Trials with Margetuximab

	Margetuximab Phase Iia	MAGENTA
Indication	R/R MBC HER2 (ICH 2+, FISH -)	HER2+ Gastroesophageal
N	41	425
Design	Single arm, Simon two-stage design with initial 21-pt cohort. If 2 responses are seen on day 22 of cycle 2, expansion to 41 pts. Further development warranted if 5 responses in 41 patients	Three cohorts based on prior therapy, randomized to 4 arms, Irinotean + margetuximab/placebo and paclitaxel + margetuximab/placebo (see Exhibit 39)
Dose / Schedule	6 mg/kg IV weekly	6 mg/kg IV weekly
Primary Endpoint	Response Rate (CR + PR)	OS
Secondary Endpoint	Duration of response, PFS, OS, Safety	N/A
Initiated	Q1:13	Planned H2:14
Data read-out	H2:14	H2:16 estimated
NCT	NCT01828021	N/A

Source: Oppenheimer & Co. Inc.

The planned Phase III Trial of margetuximab in gastric cancer (MAGENTA) is 90% powered to detect a difference between chemoRx and margetuximab assuming a hazard ratio (HR) of 0.67. An interim futility analysis at 181 events is planned.

Exhibit 39: Phase III Trial Design for Margetuximab in Gastric Cancer (MAGENTA)



Source: MacroGenics, Inc.

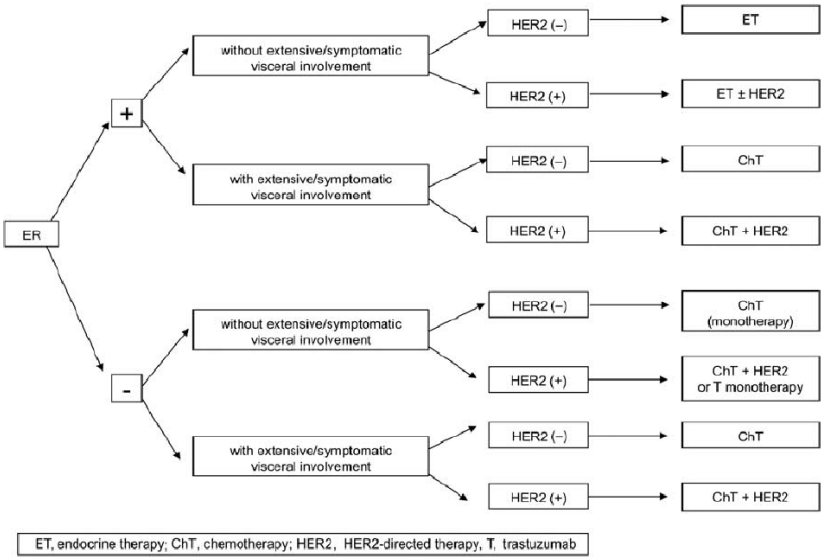
HER2-Directed Therapy Market in Metastatic Breast Cancer

HER2-directed therapies make up a nearly $8B market. Because upcoming biosimilars likely will degrade Herceptin pricing, Roche has a strong incentive to shift therapy to replacements, especially higher priced ones (Kadcyla $95,000/year, Perjeta $71,000/year) even at the expense of Herceptin ($54,000/year). The HER2 market consists of Herceptin (Roche, $6.9B), Perjeta (Roche, $375M), Tykerb (GSK, $325M), Kadcyla (Roche, $265M) and an upcoming pan-HER inhibitor, neratinib (Puma Biotechnology), currently in Phase III trials.

Breast Cancer and its Treatment

- Breast cancer is diagnosed in 232,000 new patients annually in the US
- Staging is based on degree of metastasis, estrogen receptor (ER) status, and HER2 status
- 6% of cases present with stage IV (metastatic) disease
- Roughly 20% of early stage (stage I-III) patients will relapse within 10 years
- 20% of breast cancer patients are HER2+ by immunohistochemistry (IHC) scored 2+ or 3+ or fluorescence in situ hybridization (FISH) testing
- HER2+ disease is treated with Herceptin (trastuzumab) and/or Tykerb (lapatinib)
- Metastatic breast cancer is considered incurable and the goal of therapy is palliative, despite numerous therapies on the market
- 5-year survival of stage IV breast cancer is 24.3%

Exhibit 40: MBC Treatment Guidelines by Estrogen Receptor and HER2+ Status



Source: Oppenheimer & Co. Inc.

Front line Therapy

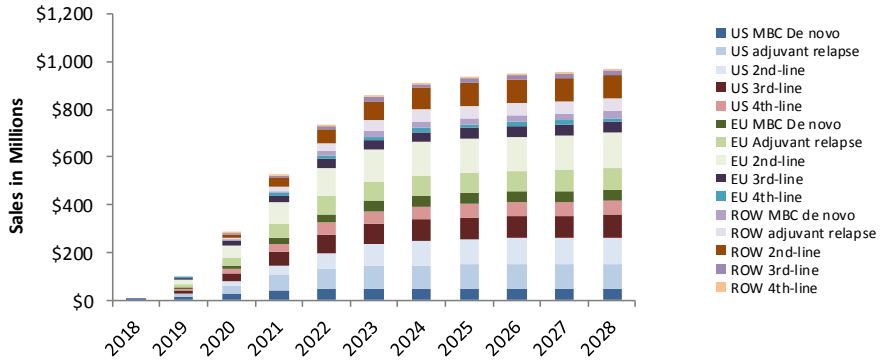
- ER+ patients receive hormone therapy, consisting of tamoxifen and/or other estrogen receptor inhibitors or aromatase inhibitors (Arimidex)
- HER2+ patients are treated with Herceptin or Tykerb in addition to chemotherapy
- No consensus has emerged on the preferred chemotherapy regimen, paclitaxel and doxorubicin in combination are widely used. Docetaxel, gemcitabine, capecitabine, and vinorelbine have also demonstrated some efficacy in metastatic breast cancer.

Second-line and Beyond

- Median time to relapse in patients who achieve a complete response (CR) or CR with bone metastasis on front-line therapy is 18.7 months.
- MBC patients go through several successive rounds of chemotherapy with different regimens as they relapse or progress on each treatment
- Response rates are roughly 20% and responses are short-lived
- Roche estimates that 39% of front-line HER2+ MBC patients will eventually receive fourth-line therapy, despite evidence that chemotherapy beyond third-line has no additional benefit

We estimate that if it receives approval in HER2 low expressing metastatic breast cancer, sales of margetuximab could reach $800M. We estimate that roughly 2,700 HER2+ stage IV breast cancer patients are diagnosed with Stage IV breast cancer in the US each year. We estimate that 20% of early stage breast cancer patients relapse, representing 8,500 patients, and that a total of 12,500 second-line through fourth-line treatments are given each year in the US. We estimate that margetuximab may be utilized in 60% of each of second-, third- and fourth-line treatments and 40% of front-line HER2+ BC patients if approved. We estimate that patients may receive 10 cycles of therapy in second-line, third-line and fourth-line therapy and 10 cycles in front-line. We estimate that margetuximab could be priced at $10,000 per cycle, in line with Kadcyla, and be approved in 2018.

Exhibit 41: Margetuximab Sales Estimates in HER2 Low Expressing MBC



Source: Oppenheimer & Co. Inc.

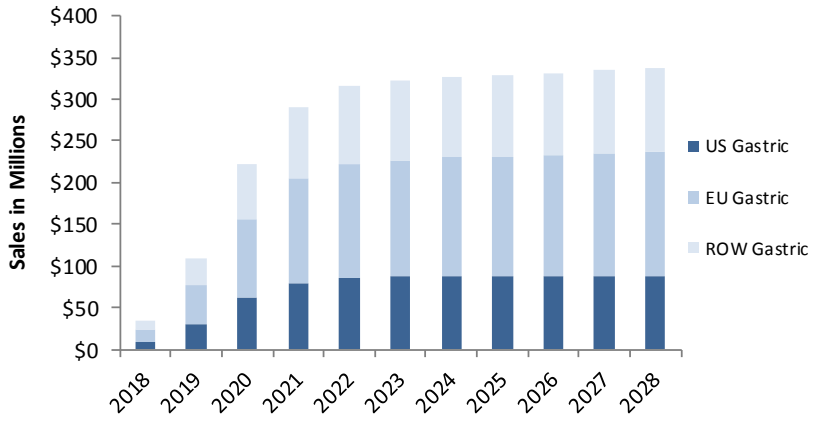
Exhibit 42: Margetuximab Sales Breakout in HER2 Low Expressing MBC

US Market Opportunity	De Novo MBC	Adjuvant Relapse	2nd-line	3rd-line	4th-line	*Total*
Number of Patients	14,000	27,000	31,500	23,500	16,250	
HER2 Low Expressing Patients	980	1,890	2,205	1,645	1,138	
Penetration	50%	50%	50%	65%	65%	
Treated Patients	490	945	1,103	1,069	739	
Price per Cycle (000's)	$10	$10	$10	$10	$10	
Average Number of Cycles	10	10	9	8	7	
Annual Market Opportunity (M)	$49	$95	$99	$86	$52	*$380*
EU Market Opportunity						
Number of Patients	16,500	32,000	35,000	16,000	7,500	
HER2 Low Expressing Patients	1,155	2,240	2,450	1,120	525	
Penetration	45%	45%	60%	60%	60%	
Treated Patients	520	1,008	1,470	672	315	
Price per Cycle (000's)	$10	$10	$10	$10	$10	
Average Number of Cycles	8	8	7	6	5	
Annual Market Opportunity (M)	$42	$81	$103	$40	$16	*$281*
ROW Market Opportunity						
Number of Patients	13,200	25,600	28,000	12,800	6,000	
HER2 Low Expressing Patients	924	1,792	1,960	896	420	
Penetration	45%	45%	50%	50%	50%	
Treated Patients	416	806	980	448	210	
Price per Cycle (000's)	$10	$10	$10	$10	$10	
Average Number of Cycles	6	6	5	4	3	
Annual Market Opportunity (M)	$25	$48	$49	$18	$6	*$147*
Grand Total						***$808***

Source: Oppenheimer & Co. Inc.

We estimate that should margetuximab receive approval in Herceptin-refractory HER2+ gastric cancer, worldwide sales could reach $325M at peak. We conservatively estimate margetuximab may be utilized in 25% of the HER2+ gastric cancer patients to take into account progressions and failures on Herceptin. We estimate 3,000 advanced or metastatic HER2+ gastric cancer patients are diagnosed in the US each year. We estimate patients will receive an average of eight cycles of therapy in the US. We anticipate approval could occur in 2018 and pricing could be $10,000 per cycle.

Exhibit 43: Margetuximab Sales Estimates in Gastric Cancer



Market Opportunity	US Gastric	EU Gastric	ROW Gastric	*Total*
Number of Patients	22,000	38,000	30,400	
HER2 Expressing Patients	4,400	7,600	6,080	
Penetration	25%	25%	25%	
Treated Patients	1,100	1,900	1,520	
Price per Cycle	$10	$10	$10	
Average Cycles	8	7	6	
Annual Market Opportunity (M)	$88	$133	$91	***$312***

Source: Oppenheimer & Co. Inc.

We estimate that margetuximab is worth $19/share to MacroGenics based on a 6x multiple of estimated 2021 sales of $600M in Gastric Cancer and Metastatic Breast Cancer discounted 30% (MBC) and 35% (gastric) annually.

MacroGenics' Fc-Optimization Technologies

MacroGenics utilized a yeast library screen to identify five point mutations that enhance Fc-domain binding to Fcγ receptors on NK cells. Antibodies incorporating these modified Fc domains exhibit up to 20x increased ADCC compared to unmodified antibodies.

Enhanced ADCC Technologies

MacroGenics has discovered five point mutations 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, MacroGenics' mutations increase the 158F/Ab binding affinity by 6.5X to an affinity 4x greater than the 158V/Ab 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.

MGNX's FC Knock-Out Domains

MacroGenics has also developed Fc domains that bind FcRn, but not immune-effector Fc receptors. The company has incorporated these domains into its MGD007 and MGD010 DART compounds to increase half-life via FcRn interactions without inducing cytokine storm or other unwanted immune activation.

Licenses, Collaborations and Intellectual Property

MGNX has a strong patent portfolio consisting of technology patents covering DARTs, Fc-optimizations and the company's proprietary cancer stem-like cell target discovery platform. MacroGenics' technology and DART platform have been validated through several significant partnerships that may provide non-dilutive capital and as much as $5.4 billion in total milestone payments. MacroGenics has partnered several of its products with Servier, keeping key US rights and receiving non-dilutive financing. We believe that MacroGenics' strategic partnering of its DART platform diversifies clinical, regulatory and commercial risk.

Exhibit 45: MacroGenics Is Eligible for >$5B in Milestone Payments

	Development	Regulatory	Sales	Total
Servier - MGA271	**$47**	**$140**	**$208**	**$395**
Servier - MGD006, MGD007 + 1 further DART	$294	$900	$1,890	$3,084
Gilead - Three DARTs	$68	-	-	$803
Boehringer - Ten DARTs	$41	$89	$83	$213
Pfizer - Two DARTs	$34	-	-	$424
Takeda - MGD010	$33			$520
Total				***$5,438***

Source: Oppenheimer & Co. Inc.

Collaborations

Servier—MGA271

In a 2011 agreement, Servier and MacroGenics entered into a right-to-develop collaboration under which Servier is granted an option to develop and commercialize MGA271 in all countries outside of the US, Canada, Mexico, Japan, South Korea and India. MacroGenics is eligible for up to $30M in license grant fees, royalties in the low double-digit to mid-teens range and $395 in milestone payments split into $47M clinical, $140M regulatory, and $208M sales based. MacroGenics received a $20M option grant fee for this license.

Servier—MGD006, MGD007 and One Further DART

In a 2012 agreement, Servier and MacroGenics entered into a right-to-develop collaboration under which Servier is granted an option to develop and commercialize MGD006, MGD007 and a third DART molecule in all countries outside of the US, Canada, Mexico, Japan, South Korea and India. MacroGenics and Servier will share Phase II and Phase III development costs. MacroGenics is eligible for up to $65M in license grant fees, high single-digit to mid-teens royalties and $1,028M in milestones split into $98M clinical, $300M regulatory and $630M sales based. MacroGenics received a $20M option grant fee for this license.

Gilead—Three DARTs

Under a 2013 agreement, Gilead has a worldwide license to one DART molecule and options to two further DART-based compounds. MacroGenics is eligible for a $7.5M license grant fee for each option Gilead exercises, $20-$25M in preclinical milestones, and $240-$250M in clinical, regulatory and sales-based milestones in each of the programs. MacroGenics is also eligible to receive royalties in the range of high single digit to 12%. MacroGenics received a $7.5M license grant fee for the first target pair.

Boehringer—Ten DARTs

Under a 2010 agreement, Boehringer has an exclusive, worldwide license to discover, develop and commercialize up to 10 DART-based molecules. MacroGenics is eligible for milestones payments of $213 split into $41M preclinical and clinical development, $89M regulatory and $83M in sales-based milestones for each product. MacroGenics is also eligible for mid-single digit royalties on net sales. MacroGenics received a $15M upfront payment as part of this collaboration.

Pfizer—Two DARTs

Under a 2010 agreement, Pfizer has a non-exclusive, worldwide license to discover, develop and commercialize up to two DART-based molecules. MacroGenics is eligible for milestone payments of $212M, split into $17M preclinical and clinical development and $195M commercialization and sales. MacroGenics is also eligible for mid-single digit to low-teen royalties on net sales. MacroGenics received a $5M upfront payment as part of this license.

Green Cross—Margetuximab

Under a 2010 agreement, Green Cross has an exclusive license to run specified Phase I and Phase II trials as well as commercialize margetuximab in South Korea. MacroGenics is eligible for $4.5M in clinical and commercial milestone payments and royalties on net sales. MacroGenics received a $1M upfront payment and a $2M equity investment as part of this collaboration.

Eli Lilly—Teplizumab

Under a 2007 agreement, Eli Lilly acquired exclusive rights to the teplizumab molecule. In 2011, MacroGenics required rights to the molecule following Eli Lilly's decision to terminate the agreement. Eli Lilly continues to reimburse MacroGenics for monitoring patients in one active clinical trial.

Takeda—MGD010

Under a 2014 agreement, Takeda acquired world-wide rights to MDG010 following a Phase Ia trial. MacroGenics has co-promote rights in the US and may share late-stage costs in exchange for a North American profit share. MacroGenics is eligible for an option license fee and early development milestones totaling $33M and later-stage clinical, regulatory and commercialization milestones worth a further $486.5M. MacroGenics is also eligible for double-digit royalties on global net sales.

Intellectual Property

Exhibit 46: MacroGenics' Patent Portfolio

Drug / Platform	Patent Type	Expiration
Fc Optimization	Three Composition of Matter for Fc domain	2024
Margetuximab	Composition of Matter	2025
Margetuximab	Composition of Matter	2029*
MGA271	Composition of Matter	2031*
MGA271	Composition of Matter	2031*
DART	Seven composition of matter patents pending	2026-2031*
MGD006	Composition of Matter	2034*
MGD006	Composition of Matter	2034*
MGD010	Four pending patents	2022-2034*
CSCP	One US patent	2028

*patent(s) pending

Source: MacroGenics, Inc.

Management

Scott Koenig, M.D., Ph.D.—President and CEO and Director

Dr. Koenig is a co-founder of MacroGenics and has served as president, CEO and director since 2001. Previously he served as senior vice president of research at MedImmune, Inc. Prior to MedImmune he worked in the Laboratory of Immunoregulation at NIAID investigating the immune response to retroviruses including AIDS. Dr. Koenig currently serves as the chairman of the board of directors of Applied Genetic Technologies Corporation and of the Children's Research Institute of Children's National Medical Center as well as a board member of the biotechnology Industry Organization (BIO) and Children's National Medical Center. Dr. Koenig received an AB and PhD from Cornell University and an MD from the University of Texas Health Science Center in Houston.

James Karrels—Vice President, Chief Financial Officer and Secretary

Mr. Karrels joined MacroGenics as vice president and chief financial officer in May 2008. Previously he was at Jazz Pharmaceuticals, Inc. , most recently as Executive director of finance. Prior to Jazz, he spent 11 years with the Investment Banking Group at Merrill Lynch, most recently as director in the Global Healthcare Group. Mr. Karrels received an MBA from Stanford University and a BBA from the University of Notre Dame.

Ezio Bonvini, M.D.—Senior Vice President, Research

Dr. Bonvini joined MacroGenics as senior vice president, research in June 2003. Previously, he was with the FDA in the Center for Biologics Evaluation and Research (CBER), most recently as acting deputy director, Division of Monoclonal Antibodies and Chief, Laboratory of Immunobiology. Previously he was a visiting fellow at the National Cancer Institute at the NIH. Dr. Bonvini received a diploma in science from the Scientific Lyceum in Genoa, Italy and an MD from the University of Genoa, School of Medicine.

Kathryn Stein, Ph.D.—Senior Vice President, Product Development and Regulatory Affairs

Dr. Stein joined MacroGenics as vice president, Product Development and Regulatory Affairs in May 2002 and was promoted to senior vice president in 2006. Previously, she was at the FDA, most recently as director, Division of Monoclonal Antibodies in the Office of Therapeutics Research and Review at CBER. Dr. Stein received a Ph.D. in microbiology and immunology from the Albert Einstein College of Medicine of Yeshiva University and a BA in chemistry from Bard College.

Jon Wigginton, M.D.—Senior Vice President, Clinical Development

Dr. Wigginton joined MacroGenics as senior vice president, Clinical Research in August 2013. Previously, he was at Bristol-Myers, most recently serving as therapeutic area head, Immuno-Oncology, Early Clinical Research and executive director, Discovery Medicine-Clinical Oncology. Prior to Bristol-Myers, he was at Merck where he served as director of clinical oncology. Dr. Wigginton received his MD and BS in biology from the University of Michigan.

Stanford Stewart, M.D.—Vice President, Clinical Oncology Research

Dr. Stewart joined MacroGenics as vice president, Clinical Oncology Research in July 2008. Previously he served as vice president, Clinical Research at Raven Biotechnologies, Inc., which was acquired by MacroGenics in July 2008. Prior to Raven, he was with Corixa Corp. most recently as vice president, Clinical Research. Previously, he was with ALZA corporation and Genentech, where he was clinical scientist on the Herceptin project. He has served as a member of the faculty of the School of Medicine at Vanderbilt University for more than 12 years. Dr. Steward received an MD from Baylor College of Medicine and a BA from Rice University.

Eric Risser—Vice President, Business Development

Mr. Risser joined MacroGenics as vice president, Business Development in May 2009. Previously he was at Johnson & Johnson most recently as senior director, Business Development in the pharmaceutical group. Prior to J&J, he started a consulting practice that provided counsel to emerging life science companies in the US and EU. Previously Mr. Risser held venture capital positions at BA Venture Partners and investment banking positions at Lehman Brothers. Mr. Risser received an MBA from Stanford University and a BA from Yale University

Lynn Cilinski—Vice President, Controller and Treasurer

Ms. Cilinski joined MacroGenics as vice president, controller and treasurer in October 2003. Previously she spent a year as a consultant to various companies providing services to the government. Prior to that, she was with Covanta Energy Inc. (formerly Ogden Corp.) most recently as corporate controller for four subsidiary companies that provided services to the government. Ms. Cilinski received a BS from Strayer University.

Financial Model

MacroGenics, Inc
Ticker: MGNX (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:												
Margetuximab Sales	0	0	0	0	0	0	0	0	0	0	3,489	102,253
Collaboration Revenue	59,646	56,753	14,401	5,000	5,000	5,000	29,401	16,000	16,000	16,000	16,000	16,000
Grants	4,180	1,282	318	318	318	318	1,272	636	0	0	0	0
Total Revenues	**$ 63,826**	**$ 58,035**	$ 14,719	$ 5,318	$ 5,318	$ 5,318	**$ 30,672**	**$ 16,636**	**$ 16,000**	**$ 16,000**	**$ 19,489**	**$ 118,253**
Cost and Expenses:												
Costs of goods sold	0	0	0	0	0	0	0	0	0	0	0	0
Research and Development	45,433	46,582	14,569	18,000	20,000	20,200	72,769	82,840	86,204	89,704	93,347	97,137
Sales, General and Administrative	10,188	11,087	3,259	3,291	3,324	3,357	13,231	13,768	14,327	37,530	62,130	64,653
Other	0	0	0	0	0	0	0	0	0	0	0	0
Total Costs and Expenses	**$ 55,621**	**$ 57,669**	$ 17,827	$ 21,291	$ 23,324	$ 23,557	**$ 86,000**	**$ 96,609**	**$ 100,531**	**$ 127,235**	**$ 155,477**	**$ 161,790**
Operating Income (loss)	**8,205**	**366**	**(3,109)**	**(15,973)**	**(18,006)**	**(18,239)**	**(55,328)**	**(79,973)**	**(84,531)**	**(111,235)**	**(136,336)**	**(52,366)**
Net Interest Income (Expense)	0	0	0	596	549	497	1,642	1,428	1,769	1,159	1,997	643
Other income / (Expense)	156	(627)	0	0	0	0	2	2	2	2	2	2
Income Before Income Taxes	8,362	(261)	(3,108)	(15,377)	(17,457)	(17,742)	(53,684)	(78,543)	(82,761)	(110,074)	(134,338)	(51,722)
Net Income	**$ 8,362**	**$ (261)**	$ (3,108)	$ (15,377)	$ (17,457)	$ (17,742)	**$ (53,684)**	**$ (78,543)**	**$ (82,761)**	**$ (110,074)**	**$ (134,338)**	**$ (51,722)**
GAAP Net Income	**$ 8,362**	**$ (261)**	$ (3,108)	$ (15,377)	$ (17,457)	$ (17,742)	**$ (53,684)**	**$ (78,543)**	**$ (82,761)**	**$ (110,074)**	**$ (134,338)**	**$ (51,722)**
GAAP Basic EPS with sFAS123	**-**	**(0.04)**	(0.12)	(0.56)	(0.63)	(0.64)	**(1.97)**	**(2.83)**	**(2.68)**	**(3.47)**	**(3.95)**	**(1.52)**
GAAP Diluted EPS with sFAS123	-	(0.04)	(0.12)	(0.56)	(0.63)	(0.64)	(1.97)	(2.83)	(2.68)	(3.47)	(3.95)	(1.52)
Weighted shares outstanding	1,083	6,848	26,262	27,620	27,645	27,670	27,299	27,732	30,832	31,682	34,032	34,132
Fully diluted shares outstanding	1,083	6,848	26,262	27,620	27,645	27,670	27,299	27,732	30,832	31,682	34,032	34,132
Cash Burn	0	0	82,240	(15,704)	(17,457)	(17,632)	(53,684)	(78,543)	(82,761)	(110,074)	(134,338)	(51,722)
Cash Balance	0	116,481	198,722	183,018	165,561	147,929	147,929	69,421	131,740	216,966	80,896	25,271

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	47,743	116,481	198,722	183,018	165,561	147,929	147,929	69,421	131,740	216,966	80,896	25,271
Accounts receivable	2,046	2,004	1,746	585	585	475	475	440	440	440	2,172	6,076
Prepaid expenses	138	972	1,937	1,937	1,937	1,937	1,937	1,937	1,937	1,937	1,937	1,937
Other current assets	0	0	0	0	0	0	0	0	0	0	0	0
Total current assets	49,927	119,457	202,404	185,540	168,083	150,341	150,341	71,798	134,117	219,343	85,005	33,283
Restricted Cash	405	405	405	405	405	405	405	405	405	405	405	405
Property and equipment, net	3,268	5,035	5,084	5,084	5,084	5,084	5,084	5,084	5,084	5,084	5,084	5,084
Other	147	885	2,280	2,280	2,280	2,280	2,280	2,280	2,280	2,280	2,280	2,280
Total assets	$ 53,747	$ 125,782	$ 210,173	$ 193,308	$ 175,851	$ 158,109	$ 158,109	$ 79,566	$ 141,886	$ 227,111	$ 92,773	$ 41,052
Current liabilities:												
Accounts payable and accrued liabilities	3,739	3,169	5,133	5,133	5,133	5,133	5,133	5,133	5,133	5,133	5,133	5,133
Accrued Expenses	1,237	3,584	2,735	2,735	2,735	2,735	2,735	2,735	2,735	2,735	2,735	2,735
Current portion of Lease Exit Liability	0	1,439	1,488	0	0	0	0	0	0	0	0	0
Current portion of deferred revenues	24,123	20,267	19,666	19,666	19,666	19,666	19,666	19,666	19,666	19,666	19,666	19,666
Other	0	363	363	363	363	363	363	363	363	363	363	363
Total current liabilities	29,099	28,822	29,385	27,897	27,897	27,897	27,897	27,897	27,897	27,897	27,897	27,897
Long Term Debt less Current Portion	0	0	0	0	0	0	0	0	0	0	0	0
Lease Exit Liability, less current	0	8,006	7,610	7,610	7,610	7,610	7,610	7,610	7,610	7,610	7,610	7,610
Deferred Rent	0	2,904	2,799	2,799	2,799	2,799	2,799	2,799	2,799	2,799	2,799	2,799
Deferred Revenue, less current	0	7,136	17,161	17,161	17,161	17,161	17,161	17,161	17,161	17,161	17,161	17,161
Other Non-current liabilities	19,956	0	0	0	0	0	0	0	0	0	0	0
Total liabilities	49,056	46,868	56,954	55,466	55,466	55,466	55,466	55,466	55,466	55,466	55,466	55,466
Stockholders' equity:	(8,237)	78,914	153,219	137,843	120,386	102,643	102,643	24,101	86,420	171,645	37,308	(14,414)
Total liabilities and stockholder's equity	$ 53,747	$ 125,782	$ 210,173	$ 193,308	$ 175,851	$ 158,109	$ 158,109	$ 79,566	$ 141,886	$ 227,111	$ 92,773	$ 41,052

Oppenheimer & Co.

Stock prices of other companies mentioned in this reports (as of 07/09/14)

Name	Ticker	Exchange	Price	Currency	Rating
AstraZeneca PLC	AZN	London	43.6	British Pounds	Not Covered
Bristol-Myers Squibb Company	BMY	NYSE	47.9	U.S. Dollar	Not Covered
CSL Limited	CSL	ASX	68.3	Australian Dollar	Not Covered
Eli Lilly and Company	LLY	NYSE	62.5	U.S. Dollar	Not Covered
GlaxoSmithKline plc	GSK	London	15.5	British Pounds	Not Covered
Merck & Co., Inc.	MRK	NYSE	58.2	U.S. Dollar	Not Covered
Newlink Genetics Corporation	NLNK	NASDAQ	24.5	U.S. Dollar	Not Covered
Pfizer Inc.	PFE	NYSE	30.2	U.S. Dollar	Not Covered
Roche Holding AG	RO	SIX Swiss	262.5	Swiss Franc	Not Covered
Stemline Therapeutics, Inc.	STML	NASDAQ	13.4	U.S. Dollar	Not Covered
Takeda Pharmaceutical Co. Ltd.	4502	Tokyo	4,684.0	Japanese Yen	Not Covered

Investment Thesis

MacroGenics is an advanced protein therapeutic platform technology company. The company combines its understanding of the immune system and advanced protein engineering techniques to produce antibodies and antibody-like proteins that bind better and/or to multiple antigens through their DART (Dual Affinity Re-Targeting) bispecifics, elicit a stronger immune response to kill cancer cells (Fc optimization) and avoid unfavorable immune reactions. The company's Cancer Stem Cell platform (CSC) provides a source of novel cancer antigens against which to discover Fc-optimized antibodies or DARTs. MGA271, targeting B7-H3, may have an immune modulatory role similar to other checkpoint inhibitors that are transforming cancer immunotherapy. Safety data expected year-end 2014 is likely to de-risk MGD006 MGNX's first clinical stage DART bispecific, in our opinion, as the therapeutic engages a clinically validated target. We believe that margetuximab, though not driving investors interest in MGNX's, provides investor a reasonable floor to the companies valuation should the more attractive (and novel but risky) assets fail to live up to expectations.

Price Target Calculation

We arrive at our $48 price target by a sum of the parts analysis. We ascribe $19/share to margetuximab in HER2 low expressing MBC and HER2+ R/R gastric cancer based on a typical oncology multiple of 6x on estimated 2021 sales of $600M discounted 30% (MBC) and 35% (gastric) annually. We ascribe $17/share to MGA-271 in renal and melanoma markets based on a 6x multiple of estimated sales and a typical royalty multiple of 15x of estimated royalties discounted 40% annually. We ascribe $6/share to MGD006 in AML patients unfit for intensive therapy and r/r AML based on a 6x multiple of estimated sales and an 18x multiple of estimated royalties, discounted 45% annually. We ascribe $5/share to MGD007 in mCRC based on a 6x/15x multiple of sales/royalties discounted 50% annually. We ascribe $1/share to MGD010 based on a 6x/15x multiple of sales/royalties discounted 45% annually.

Key Risks to Price Target

These risks include: 1) failure to reach sales expectations for margetuximab, MGA-271, MGD006, MGD007 or MGD010; 2) failure in the clinic of margetuximab, MGA-271, MGD006, MGD007 or MGD010; and 3) changes to or discontinuation of MacroGenics' partnerships for MGA-271, MGD006, MGD007 or MGD010; 4) intellectual property risk; 5) strategic risk; 6) competive risk; 7) financing risk and 8) high insider stake potentially leading to conflicts with interests of public shareholders.

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.

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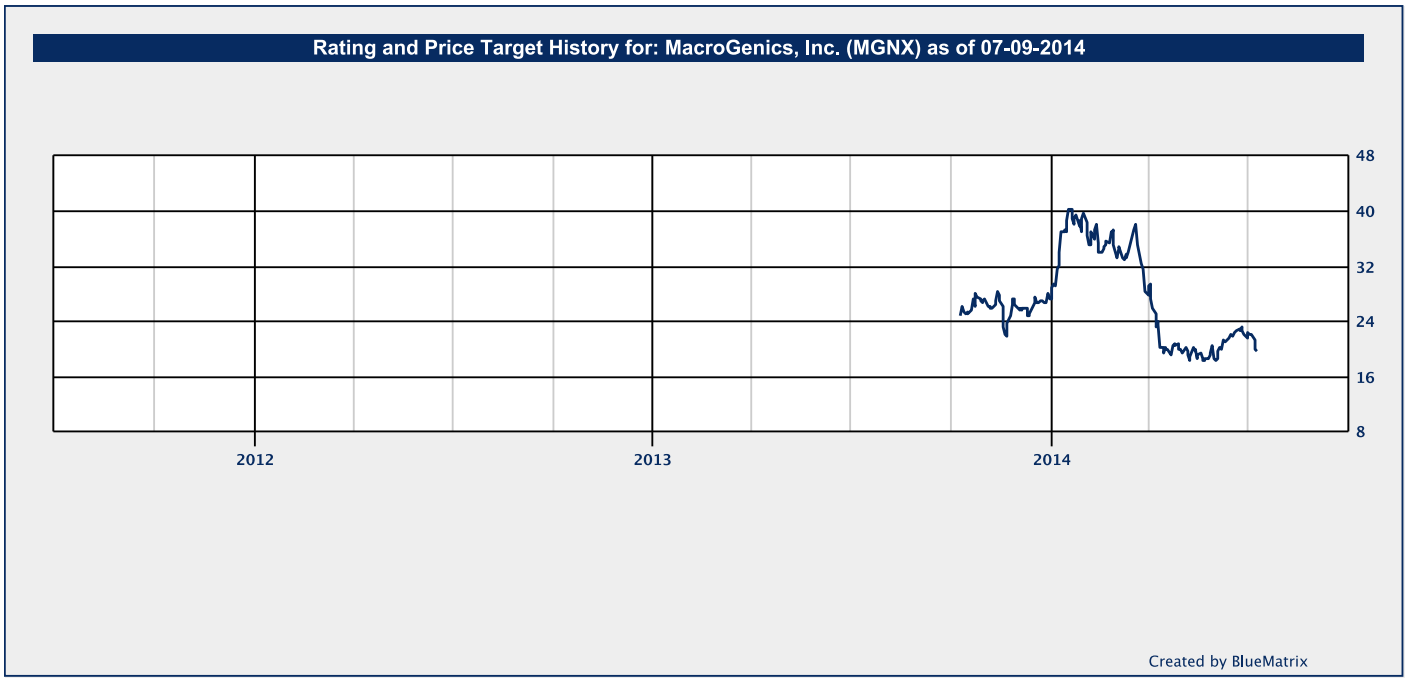
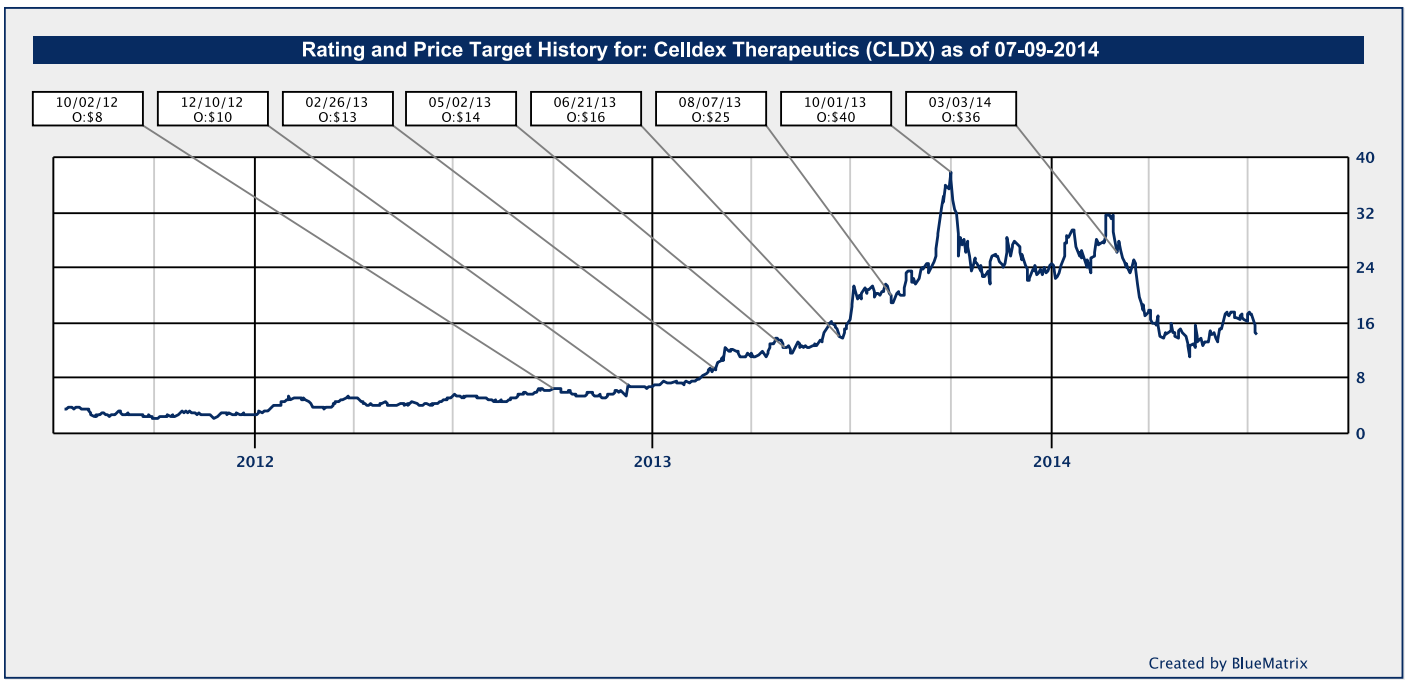
Important Disclosure Footnotes for Companies Mentioned in this Report that Are Covered by Oppenheimer & Co. Inc:

Stock Prices as of July 10, 2014

Celldex Therapeutics (CLDX - NASDAQ, $14.31, OUTPERFORM)
Gilead Sciences (GILD - NASDAQ, $88.68, OUTPERFORM)
Prothena Corp (PRTA - NASDAQ, $21.09, OUTPERFORM)


Rating and Price Target History for: MacroGenics, Inc. (MGNX) as of 07-09-2014
48
40
32
24
16
8
2012
2013
2014
Created by BlueMatrix


Rating and Price Target History for: Celldex Therapeutics (CLDX) as of 07-09-2014
10/02/12 O:$8
12/10/12 O:$10
02/26/13 O:$13
05/02/13 O:$14
06/21/13 O:$16
08/07/13 O:$25
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03/03/14 O:$36
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16
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2014
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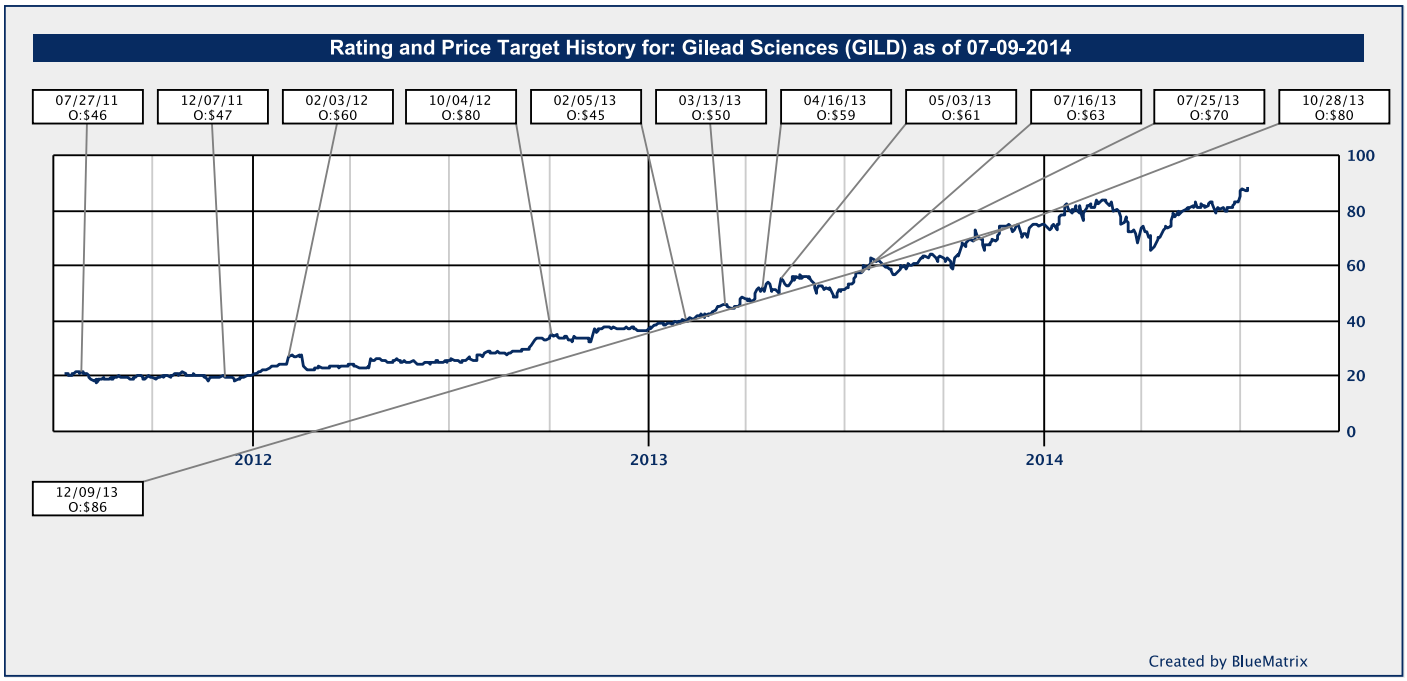

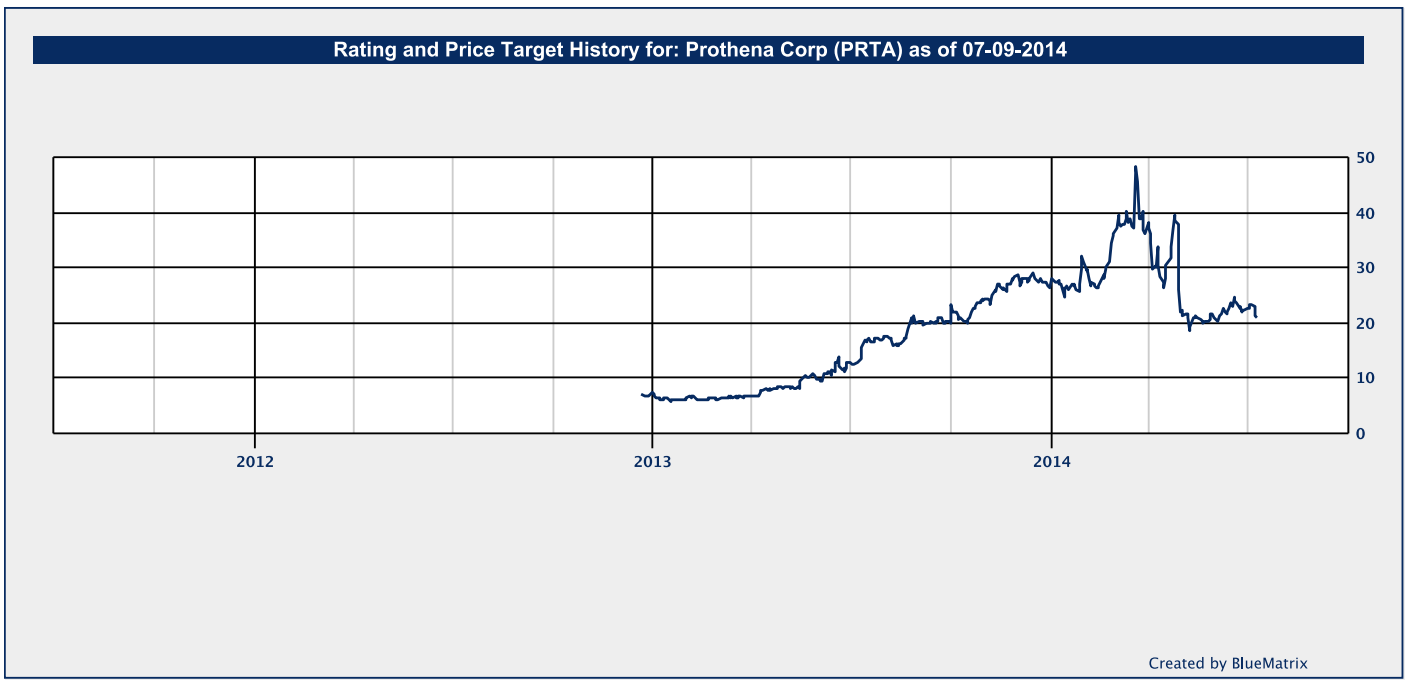

All price targets displayed in the chart above are for a 12- to- 18-month period. Prior to March 30, 2004, Oppenheimer & Co. Inc. used 6-, 12-, 12- to 18-, and 12- to 24-month price targets and ranges. For more information about target price histories, please write to Oppenheimer & Co. Inc., 85 Broad 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.

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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

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