MacroGenics (MGNX)

Initiating Coverage with an OUTPERFORM Rating and $70 Price Target-- Well-Positioned for the Immunotherapy Renaissance

- **We believe MGNX's immunologically potent protein product candidates and discovery platform are ideally positioned to participate in the current immunotherapy renaissance – as such, we see the inherent value of the company as well above where the stock currently trades.** Novel immunoregulatory therapeutics (PD1, PDL1, CTLA4, IDO, B7-H3, CD-27, CD-47) are elevating the likelihood for unprecedented efficacy for next-generation antibody and antibody-like therapies that appropriately target and recruit the immune system to treat cancer.
- **Initial clinical data for MGNX's Fc-optimized HER2 antibody margetuximab suggests a superior immunologically active profile, which we believe is likely to significantly expand the population of breast cancer patients suitable for HER-2 therapy, expand the use of HER2 therapy in the bladder and gastroesophageal settings and potentially compete with existing HER2 breast cancer therapeutics.** A Phase III study of margetuximab in the third-line gastric cancer setting is expected to begin in 2014, and data from an ongoing Phase II study in the metastatic breast cancer setting is expected in H2:14.
- **MGA271, an Fc-optimized Mab that both targets and blocks the B7-H3 T cell inhibitory antigen on tumor and tumor vasculature, is at present in a Phase I expansion study in patients with advanced solid tumors.** We expect the best results for MGA271 to be achieved in combination studies with other immunologically active therapies and/or following the appropriate conditioning of the patients to deplete T regulatory cells.
- **Ex-vivo results for MGNX's leading bispecific protein therapeutic DART, MGD006, suggest significant activity in recruiting, expanding and activating T-cells to target IL-3R-positive AML cells.** In addition to previewing potential efficacy for this drug in 2014 clinical studies, these results provide proof-of-concept for MGNX and its up to 19 partnered programs.
- **Initiating coverage of MacroGenics with an OUTPERFORM rating and $70 price target.** Our $70 price target is derived from the sum of multiples of sales and royalties from the company's proprietary and partnered products, each discounted back to YE:14 (Figure 20).

FYE Dec	2012A	2013E			2014E		
REV	ACTUAL	CURR.	PREV.	CONS.	CURR.	PREV.	CONS.
Q1 Mar	--	$11.4A		N/AA	$4.0E		N/AE
Q2 Jun	--	11.4A		N/AA	4.0E		N/AE
Q3 Sep	--	20.2E		20.2E	4.0E		N/AE
Q4 Dec	--	5.0E		12.7E	4.0E		N/AE
Year*	**$63.8A**	**$48.1E**		**$53.0E**	**$16.0E**		**$65.3E**
Change	--	-25%			-67%		
	2012A	2013E			2014E		
EPS	ACTUAL	CURR.	PREV.	CONS.	CURR.	PREV.	CONS.
Q1 Mar	--	($0.09)A		N/AA	($0.57)E		N/AE
Q2 Jun	--	(0.09)A		N/AA	(0.57)E		N/AE
Q3 Sep	--	0.29E		0.01E	(0.56)E		N/AE
Q4 Dec	--	(0.47)E		(0.38)E	(0.56)E		N/AE
Year*	**$0.39A**	**($0.53)E**		**($0.73)E**	**($2.25)E**		**($0.65)E**
P/E	--	--			--		
Change	--	-237%			-324%		

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

December 3, 2013

Price
$25.81

Rating
OUTPERFORM

12-Month Price Target
$70

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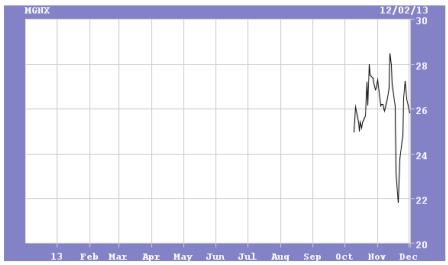
Company Information	
Shares Outst (M)	19.0
Market Cap (M)	$491.0
52-Wk Range	$21.50 - $30.25
Book Value/sh	$-7.07
Cash/sh	$1.76
Enterprise Value (M)	$457.5
LT Debt/Cap %	-0.0
Cash Burn (M)	$23.0

Company Description

MacroGenics is developing advanced protein therapeutics for the treatment of cancer, autoimmune, and infectious diseases using their Dual Affinity Re-Targeting (DART), Fc Optimization, and Cancer Stem Cell platforms.



Source: Thomson Reuters

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 (DART – Dual Affinity Re-Targeting), 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.

We are in the midst of an immunotherapy renaissance. Novel immunostimulatory drugs (PD1, PDL1, CTLA4, IDO, B7-H3, CD-27, CD-47) are elevating the likelihood for unprecedented efficacy for next-generation antibody and antibody-like therapies that appropriately target and recruit the immune system to treat cancer. MacroGenics has, in a capital efficient manner, established the infrastructure and knowledge base required for the discovery, design, manufacture and pre- and clinical evaluation of advanced protein therapeutics. This infrastructure has been built and receives ongoing support from partners who have paid the company $106 million cumulatively in the past 3 years and from whom a further $100 million is expected by YE:15. We view the established ongoing support of the MGNX product engine as a key competitive advantage, and we expect the rate of transactions to accelerate as the central role of immune cell targeting and recruitment to effective immunotherapy becomes more broadly appreciated. We do not include these transactions in our model.

We expect margetuximab to increase the breadth of effective HER2 breast cancer therapy to the 18% of the HER2+ patient population who are 2+ by IHC and non-amplified (7% of the total breast cancer population). If as successful as Herceptin, margetuximab could generate over $1 billion per year in sales in this group. We do not project margetuximab revenues in the competitive 3+/2+ amplified HER2 setting where Roche's (Not Covered) Herceptin, Perjeta and Kadcyla are focused; however, we note that if future combination studies of margetuximab and the advancing immunoregulatory proteins demonstrate superior pathologic-CR, RR, PFS and OS comparable to 3+/2+ HER2 amplified settings, combinations containing margetuximab may compete with the product's in Roche's HER2 franchise.

We believe the best responses to MGA271, MGNX's B7-H3 Fc-optimized antibody, will be achieved with future combination studies with the advancing immunoregulatory therapeutics noted above, and/or properly sequenced with other agents designed to delete/reset inappropriate immune function. The widespread expression and importance of B7-H3 in regulating T-cell function may ultimately see the drug candidate useful in a variety of tumor types and importantly, those adjuvant settings currently without proven antibody therapeutics (colorectal, lung).

Ex-vivo results for MGNX's leading DART, MGD006, suggest significant activity in recruiting, expanding and activating T-cells to target IL-3R positive AML cells. In addition to previewing potential efficacy for this drug in 2014 clinical studies, these results provide provocative proof-of-concept for MGNX and its up to 19 partnered DART programs, about half of which we believe utilize CD3 to stimulate T-cells. Importantly, MGD007 which targets the gpa33 antigen on colorectal cancer, also recruits, expands and activates T-cells via CD3 interaction giving us greater confidence in positive clinical data from this drug.

We believe MGNX's margetuximab (HER2), MGA271 (B7-H3) and MGD007 (gpa33•CD3) drug candidates may play roles in expanding the use of biologic therapy in the adjuvant setting. The majority of Herceptin sales, estimated to be nearly $6.4 billion in 2013, are in the adjuvant setting, despite being indicated for only 25% of patients (about 52,000 patients in the US each year). Margetuximab could find utility in the 7% of breast cancer patients who have 2+/non-amplified disease and if as successful as Herceptin, potentially generate a commensurate level of sales in the adjuvant setting. About 75% of the ~143,000 colorectal cancer patients (about 107,000 in the US) are diagnosed with local or regionally restricted disease. Many of these patients are elderly and relatively intolerant of current standard adjuvant chemotherapy regimens. We believe there is an obvious need for a targeted, well-tolerated therapeutic in this setting to reduce recurrence. Either or both MGA271 and MGD007 could find utility here.

Valuation

Our price target of $70 is derived from 6x multiples of product sales and 15x multiples of royalties for each of MGNX's products (Figure 20). Using valuation years ranging from 2021 to 2024, 6x multiples and 35% annual discount rates back to YE:14 for nonadjuvant breast, gastric, bladder, and esophageal cancer and 30% annual discount rate back to YE:14 for adjuvant breast cancer, we value margetuximab at $1.14B ($34.03 per share). Using a 6x multiple of MGNX's MGA271 2024 sales and 15x multiple of 12% royalties from Servier with a 35% annual discount rate back to YE:14 for MGA271, we value MGA271 at $465M ($13.84). Using a 6x multiple of 2022 MGD006 sales and a 15x multiple of 12% royalties from Servier with 30% and 35% annual discount rates respectively, we value MGD006 at $361M ($10.74). Using a 6x multiple of 2025 MGD007 sales and 15x multiple of 12% royalties from Servier with an annual discount rate of 35% back to YE:14, we value MGD007 at $377M ($11.21 per share).

We do not yet include $10/share in value we derive from 15x multiple of our estimate of royalties from each of the undisclosed partnered DART programs, each discounted 35% annually back to YE:14. For the undisclosed DART programs, we assume that each will have global peak sales of approximately $500 million, with royalty rates of 5%, 10%, and 10% for Boehringer Ingelheim, Pfizer, and Gilead, respectively. We anticipate each partner will launch their first undisclosed DART 7 years out and that each will launch an equal number of DARTS per year such that within 5 years of the first launch, all DARTS are on the market. We believe this valuation methodology to be conservative as we are using what we believe to be the minimally acceptable peak sales per product for it to be pursued by the partner, conservative launch timing, relatively high discount rates and a fully-diluted share count that takes into account 2 future equity financings. We expect the value of these drug candidates to accrue to the stock as these programs advance into the clinic and receive increased visibility from Wall Street.

Risks

Risks to the achievement of our price target include clinical failures of margetuximab, MGA271, MGD006, MGD007, and product advancement and development success rates for MGNX's licensees below that for which we have modelled.

Key Points

- Single agent activity for margetuximab in patients that have failed a HER2 targeted therapeutic is early evidence that MGNX's discovery and design (Fc-optimization) product engine produces a differentiated therapeutic.
- We expect margetuximab's superior immunological profile to significantly expand the population of breast cancer patients suitable for HER2 therapy, expand HER2 therapy in the bladder and gastroesophageal settings and potentially compete with existing HER2 breast cancer therapeutics, alone or combined with other immunoregulatory therapeutics.
- A Phase III study of margetuximab in the 3rd line gastric cancer setting is expected to begin in H2:14, and data from an ongoing Phase II study in the metastatic breast cancer setting is expected in 2014.
- We expect the best results for MGA271 (B7-H3) will be forthcoming from studies in combination with other immunoregulatory products, and/or when sequenced or combined with agents that delete/reset inappropriate immune function.
- Compelling pre-clinical data for MGNX's first DART (MGD006 – ILRR•CD3), that demonstrates the drug recruits, expands and activates human T-cells that elicit dose-dependent killing of IL-3R positive cells is to be presented at ASH (Dec. 7-10, New Orleans). Although pre-clinical, ex-vivo evidence that MGD006 causes a cellular immunologically mediated anti-cancer effect against primary human AML samples is encouraging. These findings are early validation of MGNX's DART platform, and we believe precede significant clinical activity from MGD006 studies that get under way in 2014.
- Based upon the recruitment, expansion and T-cell mediated killing demonstrated ex vivo for MGD006, investors should have increased confidence in the potential for MacroGenics' MGD007 which targets the colorectal and other GI tumor antigen gpA33 and similarly directs T-cells against the tumor via CD3 binding. An IND submission is expected in 2014 and initiation of Phase I in H2:14.
- We believe investors should expect a significant level of productivity and continued success from the MGNX product engine. With more than 1,900 antibodies for 70 antigens currently in the company's discovery library, we expect at least one IND per year.
- We believe MacroGenics' product engine runs in a capital-efficient manner, with the expertise required for the discovery, design, manufacture and evaluation of advanced protein therapeutics built and supported from partners who have paid the company $106 million cumulatively in the past 3 years.
- MGNX has several pre-clinical DART assets that are being developed in collaboration with Servier, Gilead, Pfizer, and Boehringer Ingelheim in various cancer and autoimmune settings. $100 million in additional revenue from existing deals is expected by YE:15.

MacroGenics Overview

Macrogenics is headquartered in Rockville, Maryland and has operations at the Raven facility in South San Francisco, California. The company has built internal discovery, pre-clinical and clinical development and biologic manufacturing capabilities and is focused on advanced protein therapeutics for the treatment of cancer and autoimmune diseases. MacroGenics currently has two oncology products in clinical development (margetuximab and MGA271) and several candidates in pre-clinical studies. The company's partnerships with Servier, Gilead, Boehringer Ingelheim and Pfizer for MGA271 should be a continuing source of substantial ongoing financial support.

Upcoming Milestones

Dec. 9, 2013	Oral presentation of pre-clinical data of MGD006 (IL-3R•CD3) titled "Targeting CD123 in Leukemic Stem Cells Using Dual Affinity Re-Targeting Molecules (DARTs)" at ASH (New Orleans, LA)
H1:14	IND filing for margetuximab in the advanced gastroesophageal cancer setting (our estimate)
H1:14	Initiate a Phase 1 clinical trial of MGD006 (IL-3R•CD3) target in the AML setting
H2:14	Initiate a Phase 3 study of margetuximab with or without paclitaxel or irinotecan in the third line advanced gastroesophageal cancer setting (primary endpoint is overall survival)
H2:14	Initiate a Phase 1 clinical trial of MGD007 (gpa33•CD3) (second DART candidate) in the colorectal cancer setting
2014	Complete enrollment of the Phase 2a clinical trial of margetuximab (HER2) in the metastatic breast cancer setting
2014	Complete enrollment (n=45) of the dose-expansion portion of the Phase 1 clinical trial of MGA271 (B7-H3) target as a single-agent in patients with advanced solid tumors
2014	Top-line results from the Phase IIa study of margetuximab (HER2) in the metastatic breast cancer setting
2015	Initiate a Phase 2 study of MGA271 (B7-H3) in the solid tumor setting to be identified from ongoing Phase I/II studies
2017	Potential top-line data from the Phase III study of margetuximab in the second line advanced gastroesophageal cancer setting (our estimate)

Figure 1: Pipeline Overview

Program	Target	Antibody Technology	Partner	MacroGenics Commercial Rights	Indication (Development Phase)
Margetuximab	Her2	Fc Opt	Green Cross	Worldwide except Korea	Gastroesophageal Cancer (initiate PhIII in H2:14) Breast Cancer (Phase II) Solid Tumors (Phase I)
MGA271	B7-H3	Fc Opt CSC	Servier	North America, Japan, Korea, India	Solid Tumors (Phase I)
MGD006	IL-3R•CD3	DART	Servier	North America, Japan, Korea, India	Acute Myeloid Leukemia (pre-clinical)
MGD007	gpA33•CD3	DART CSC	Servier	North America, Japan, Korea, India	Gastrointestinal Cancers (pre-clinical)
Multiple DARTs	various	DART CSC	-	Worldwide	Various (pre-clinical)
Up to Four DARTs	undisclosed	DART	Gilead	Outside of North America, Europe, Australia, and New Zealand for one of the four programs	Cancer and other life-threatening indications (Research)
DART	undisclosed	DART	Pfizer	None	Various (Research)
teplizumab	CD3	Fc Opt	-	Worldwide	T1 Diabetes Prevention (Phase II, failed in Phase III)
MGD010	CD32B•CD79B	DART	-	Worldwide	Lupus, Rheumatoid Arthritis (Pre-clinical)
Multiple DARTS	undisclosed	DART	Boehringer Ingelheim	Co-promote certain Boehringer DARTs in the US	Various Autoimmune and Other Indications (Research)

Source: MacroGenics, Wedbush Securities, Inc.

MacroGenics's Drug Discovery Approach and Platforms

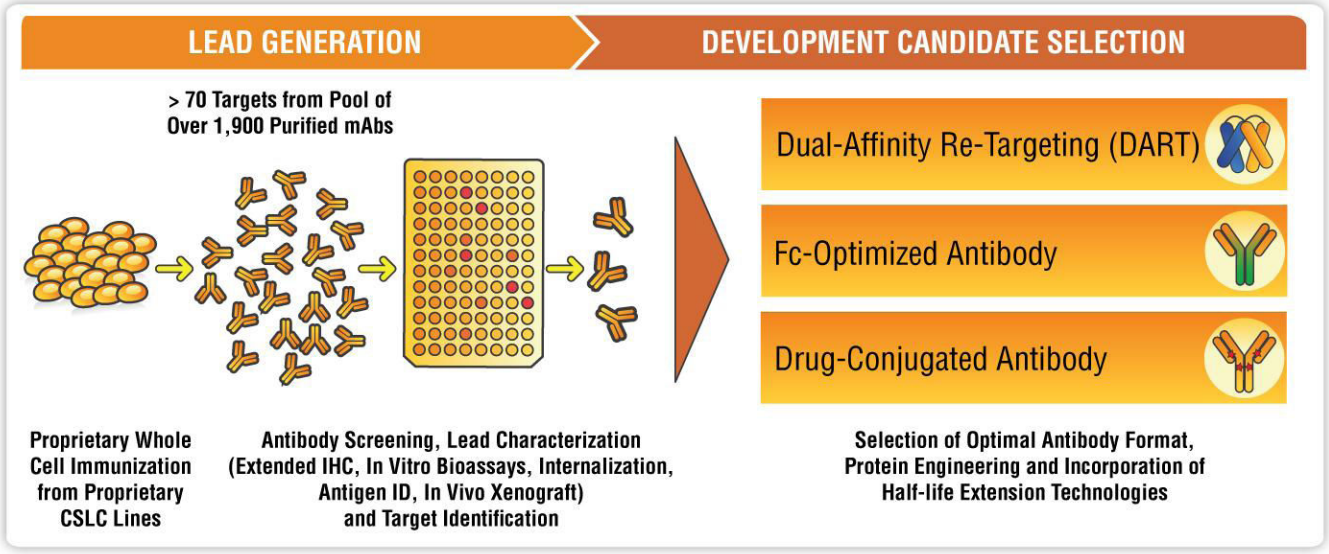
MacroGenics takes an integrated approach in developing novel advanced protein therapeutics (Figures 1 and 2). The company identifies targets from validated or existing targets or its own target identification platform (CSC) and designs and manufactures potent advanced protein therapeutic candidates using the capabilities for dual affinity re-targeting (DART) and Fc-optimization.

Improved immunologic function of standard antibodies is achieved through Fc modification (optimization). The Fc region of an antibody is located at the base of the Y-shaped antibody protein (Figure 3) and is responsible for modulating the immune response through binding of specific receptors of immune cells. Through Fc-optimization, MacroGenics is developing therapeutics that enhance the immune response towards cancer cells (antibody dependent cellular cytotoxicity (ADCC)) (Figure 4). Margetuximab (HER2) and MGA271 (B7-H3) were developed using MGNX's Fc-optimization platform.

DART provides a process for developing a biologic that targets and binds to multiple antigens or cells (Figure 5). MGD006 (IL-3R/CD3) and MGD007 (gpa33•CD3) target specific cancer antigens and are designed to recruit, expand and activate T cells via the interaction with CD3. There are 19 potential DARTs that could emerge from the partnerships with Gilead, Pfizer, Boehringer Ingelheim, and Servier.

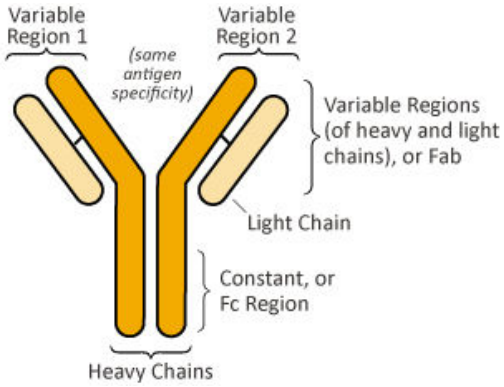
MacroGenics' CSC platform generates cancer stem-like cells from primary human tumor tissues. Cancer stem cells typically comprise of 2% of a tumor and are believed to be the drug resistant component that returns following therapy. Tumor cell derived stem-like cells provide MacroGenics with a significant number of targets (Figure 6).

Figure 2: MacroGenics's Integrated Approach to Therapeutics Development



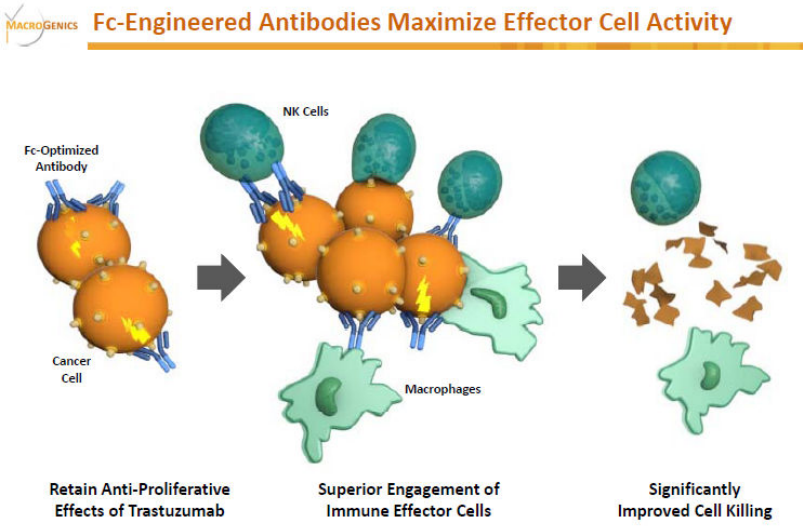
Source: MacroGenics

Figure 3: Monoclonal Antibody Structure



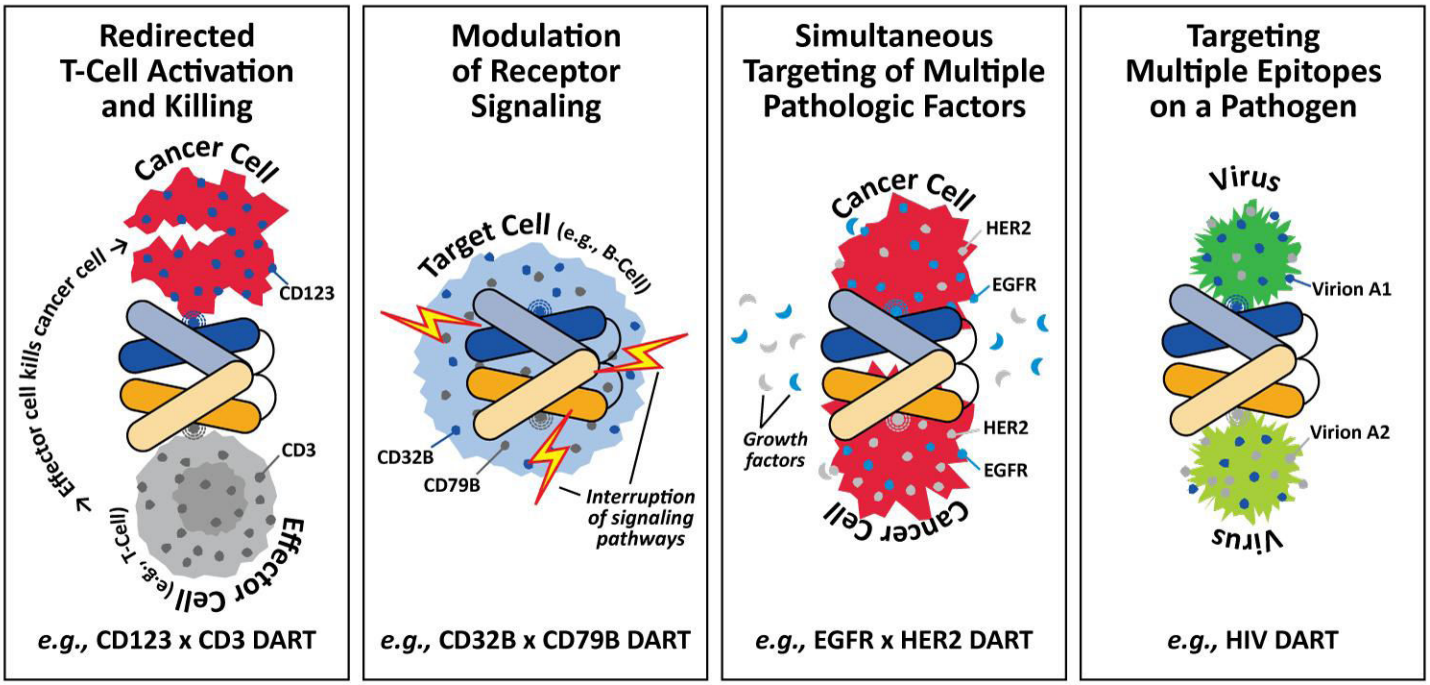
Source: MacroGenics

Figure 4: Fc Optimization



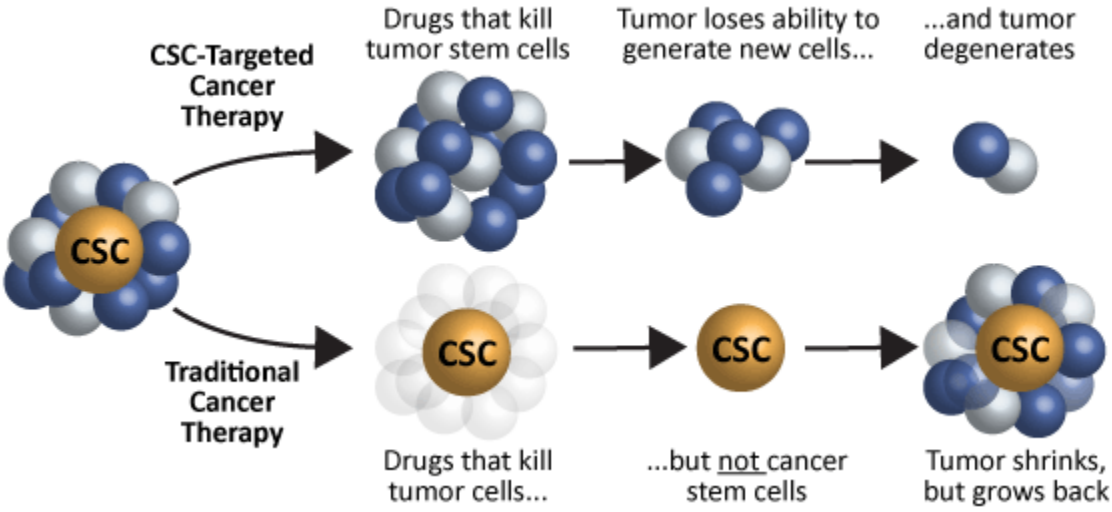
Source: MacroGenics

Figure 5: DART Platform



Source: MacroGenics

Figure 6: CSC Platform

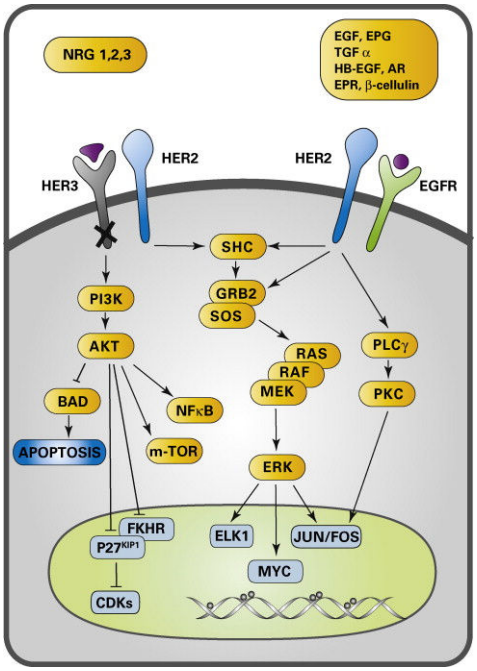


Source: MacroGenics

Margetuximab

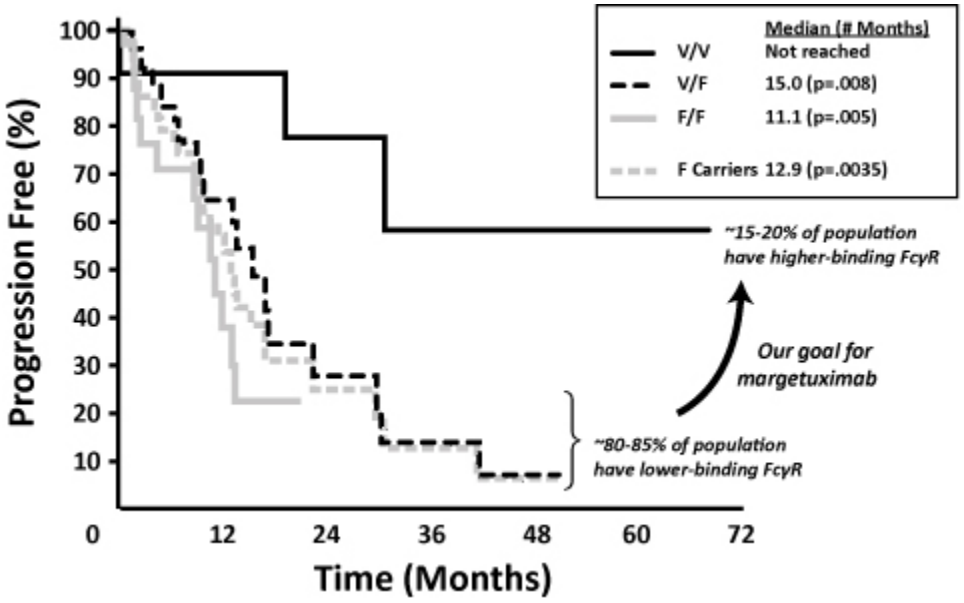
Margetuximab is a next-generation and Fc-optimized epidermal growth factor receptor 2 (HER2) targeting monoclonal antibody (mAb). HER2 is a transmembrane tyrosine kinase receptor that upon ligand binding leads to active homodimers or heterodimers. Dimerization leads to receptor phosphorylation and a cascade of signals (Figure 7) that affect proliferation, apoptosis, and migration. Roche's Herceptin is the most widely used HER2 targeted therapy and is on pace to sell $6.4 billion globally in 2013. A key aspect of the mechanism of anti-HER2 monoclonal antibody is the mediation of ADCC, the binding of the antibody and recruitment and activation of immune cells such as macrophages through FcγRs. Through MacroGenics' Fc optimization, margetuximab was developed to have increased binding to the immune effector cell's activating receptors and reduced binding of the inhibitory receptor, thus improving the cytotoxic activity of margetuximab. The potential for differentiated efficacy, relative to Herceptin, as a result of an optimized Fc is suggested by the data that shows progression-free survival is greatly increased in the metastatic breast cancer patient population with higher binding FcγR, CD16A, than those with lower binding FcγR when treated with trastuzumab plus chemotherapy (Figure 8). The pre-clinical studies of margetuximab suggest that it has activity in current HER2 therapy-resistant tumors.

Figure 7: HER2 Pathway



Source: Zhongren Zhou and David G. Hick (2013). HER2 Amplification or Overexpression in Upper GI Tract and Breast Cancer with Clinical Diagnosis and Treatment, Oncogene and Cancer - From Bench to Clinic, Dr. Yahwardiah Siregar (Ed.), ISBN: 978-953-51-0858-0

Figure 8: Progression-Free Survival Higher for Patients with a Higher-Binding Fc



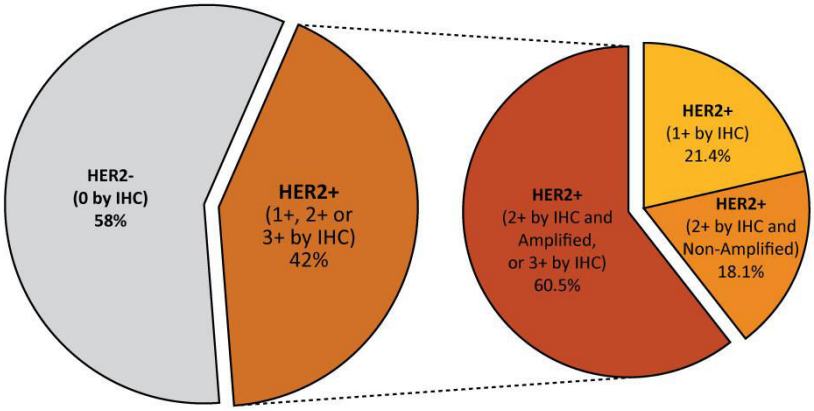
Source: MacroGenics

Breast Cancer

Breast cancer is the second most frequently diagnosed cancer in women, with a global incidence estimated at nearly 1.4 million and approximately 458,000 deaths. In the US, there will be an estimated 232,000 new cases and 39,620 deaths in 2013. Most (61%) patients are diagnosed with localized disease in the US, with about one-third having disease that has metastasized to regional lymph nodes and the remainder with distant metastatic or un-staged disease. Effective breast cancer screening programs have resulted in a high number of patients identified with early disease, and a large population undergoing adjuvant systemic therapy following surgical resection.

HER2 is overexpressed in the tumors of about 25% of women with breast cancer. Over expression predicts more aggressive disease, greater likelihood of recurrence, a poorer prognosis and decreased survival. The level of HER2 protein on tumors can be detected by laboratory testing and is scored 0, 1+, 2+ or 3+, where 3+ indicates the highest level of HER2. Fluorescence in situ hybridization (FISH) testing is a method used to determine the number of HER2 gene copies that are in a tumor cell. Currently, anti-HER2 therapies are only approved for treating approximately 25% of all breast cancer patients whose tumors over express HER2 at the 3+ level, or if 2+, when accompanied by HER2 gene amplification. This population represents 25% of breast cancer patients leaving approximately 7.5% of breast cancer patients with 2+ HER2/non-amplified for which an effective HER2 targeted therapy may have utility (Figure 9).

Figure 9: Breast Cancer Market Opportunity



Source: MacroGenics

Surgery, either lumpectomy or mastectomy, potentially with neo-adjuvant systemic therapy is the first-line treatment for localized breast cancer. Following surgery, radiation therapy reduces the risk of local recurrence and depending upon the tumor's characteristics (hormone receptor expression / HER2 status) and degree of lymph node involvement, patients may also receive systemic post-surgical adjuvant therapy to improve disease-free survival and reduce the likelihood for recurrence. Adjuvant regimens include chemotherapy, hormone therapy, anti-HER2 drugs (e.g., Herceptin), or a combination of these treatments. Several drugs directed at HER2 have been approved in the early and advanced stage breast cancer settings. These include trastuzumab (Herceptin), pertuzumab (Perjeta), lapatinib (Tykerb), and ado-trastuzumab emtansine (Kadcyla, or T-DM1) (Figure 10).

Figure 10: HER2 Targeting Drugs

Product (Company/Trial) Mechanism	Setting	Median Time to Progression (mTTP)/ Median Overall Survival (mOS)/ Partial Response (PR)/ Progression Free Survival (PFS)/ Disease Free Survival (DFS), Hazard Ratio (HR)
Margetuximab (MacroGenics, Phase 1) Fc optimized anti-HER2 antibody	refractory HER2+ (1/2/3+ levels) breast cancer, dose escalation (0.1-6.0 mg/kg qw) and expansion (6.0 mg/kg qw) n=10	PR to date- 4 (40%), n=10, see Figure 13
Herceptin/trastuzumab (Roche, Phase 3) Anti-HER2 antibody	Previously untreated metastatic HER2+ (2/3+ levels) Chemotherapy only (PREVIOUS ANTHRACYCLINE THERAPY: paclitaxel 175 mg/m2 over 3 hrs every 21 days for at least six cycles; ALL OTHER PATIENTS: (AC) doxorubicin 60 mg/m2 or epirubicin 75 mg/m2 plus 600 mg/m2 cyclophosphamide every 21 days for 6 cycles) vs. Chemotherapy + Herceptin (4 mg/kg loading dose followed by 2 mg/kg qw) n=469	mTTP: Herceptin+chemotherapy 7.2 mos (95% CI 7-8), n=235 vs chemotherapy 4.5 mos (95% CI 4-5), n=234; p<0.0001 mOS: Herceptin+chemotherapy 25.1 mos (95% CI 22,30) n=235 vs. chemotherapy 20.3 mos (95% CI 17-24), n=234; p=0.05
Herceptin/trastuzumab (Roche, Study 3) Anti-HER2 antibody	HER2 (3+ level) or gene amplification breast cancer Post-surgery patients were randomized to receive four cycles of chemotherapy followed by one year of Herceptin or no treatment (n=3386)	DFS: Chemotherapy+Herceptin 127 events, n=1693 vs chemotherapy 219 events, n=1693; HR: 0.54, 95% CI 0.44-0.67, p=<0.0001
Perjeta/pertuzumab (Roche, Phase 3) Anti-HER2 antibody	HER2+ (3+ level) or gene amplification metastatic breast cancer (n=808) Placebo or Perjeta (840 mg initial dose followed by 420 mg q3w) + trastuzumab (8 mg/kg initial dose followed by 6 mg/kg q3w) + docetaxel (75 mg/m2 initial dose q3w for at least 6 cycles, could be escalated to 100 mg/m2)	PFS: Perjeta arm 191 (47.5%) events with 18.5 mos median, n=402 vs. placebo arm 242 (59.6%) events with 12.4 mos median, n=406; HR: 0.62, 95% CI 0.51-0.75, p<0.0001 OS: Perjeta arm 113 (28.1%) events with a median that was not reached vs 154 (37.9%) events with 37.6 mos median; HR: 0.66, 95% CI 0.52-0.84, p=0.0008*
Kadcyla/ado-trastuzumab emtansine (Roche, Phase 3) Anti-HER2 antibody drug conjugate (with DM1)	HER2 + (3+ level) or gene amplification, unresectable locally advanced or metastatic breast cancer patients with prior taxane and trastuzumab-based therapy (n=991) Kadcyla (3.6 mg/kg on day 1 of a 21-day cycle) Capecitabine (1000 mg/m2 twice daily on Days 1-14 of a 21-day cycle) and lapatinib (1250 mg/day, 21 day cycle)	PFS: Kadcyla 265 (53.5%) events with 9.6 mos median, n=495 vs. lapatinib+capecitabine 304 (61.3%) events with 6.4 mos median, n=496; HR: 0.650, 95% CI 0.549-0.771, p<0.0001 OS: Kadcyla 149 (30.1%) events with 30.9 mos median vs. lapatinib+capecitabine 182 (36.7%) events with 25.1 mos median; HR: 0.682, 95% CI 0.548-0.849, p=0.0006
Tykerb/lapatinib (GlaxoSmithKline, Phase 3) HER2 and EGFR intracellular tyrosine kinase domains inhibitor	HER2+ (2/3+ level) locally advanced or metastatic breast cancer patients with prior treatments including anthracyclines, taxanes and trastuzumab (n=399) Tykerb (1,250 mg/day) + capecitabine (2,000 mg/m2/day on Days 1-14, 21 day cycle Capecitabine (2,500 mg/m2/day on Days 1-14, 21 day cycle	TTP: Tykerb+capecitabine 121 events with 23.9 weeks median, n=198 vs. capecitabine only 126 events with 18.3 weeks median, n=201; HR: 0.72, 95% CI 0.56-0.92, p=0.00762

Source: Company data, Wedbush Securities, Inc.

Gastroesophageal Cancer

Gastroesophageal cancer is composed of gastric and proximal esophagogastric junction (EGJ) cancers. Although gastric and esophageal cancers are not as common in the US, these are the third most commonly diagnosed with the second highest mortality globally, with 1.4 million cases annually leading to approximately 740,000 deaths. HER2 gene amplification and protein overexpression is reported in varying frequencies with EGJ gene amplification in 24-32% of patients, protein overexpression in 2-45% of patients, and gastric cancer patients having gene amplification in ~9-18% of tumors, and protein overexpression in 8-53% of patients (Hechtman, 2012).

Recurrence after resection occurs frequently, highlighting the need for effective systemic therapy. Thirty to seventy percent of the patients who undergo 1st line systemic therapy progress and go on to receive a 2nd line systemic therapy regimen.

The Phase III randomized ToGA trial (Trastuzumab for Gastric Cancer) established trastuzumab (Herceptin) plus chemotherapy as a new therapy for treatment naïve patients with HER2 positive gastric or gastroesophageal junction cancer. This 594-patient study showed a median overall survival of 13.5 months for trastuzumab in combination with chemotherapy (cisplatin in along with 5-fluorouracil or capecitabine) compared to 11.0 months for chemotherapy alone (hazard ratio 0.73, 95% CI 0.60-0.91, p=0.0038). An updated analysis presented one year after found a median survival rate of 13.1 months for Herceptin and chemotherapy combination versus 11.7 months for chemotherapy only (hazard ratio 0.80, 95% CI 0.67-0.97).

Margetuximab Phase I Study Results

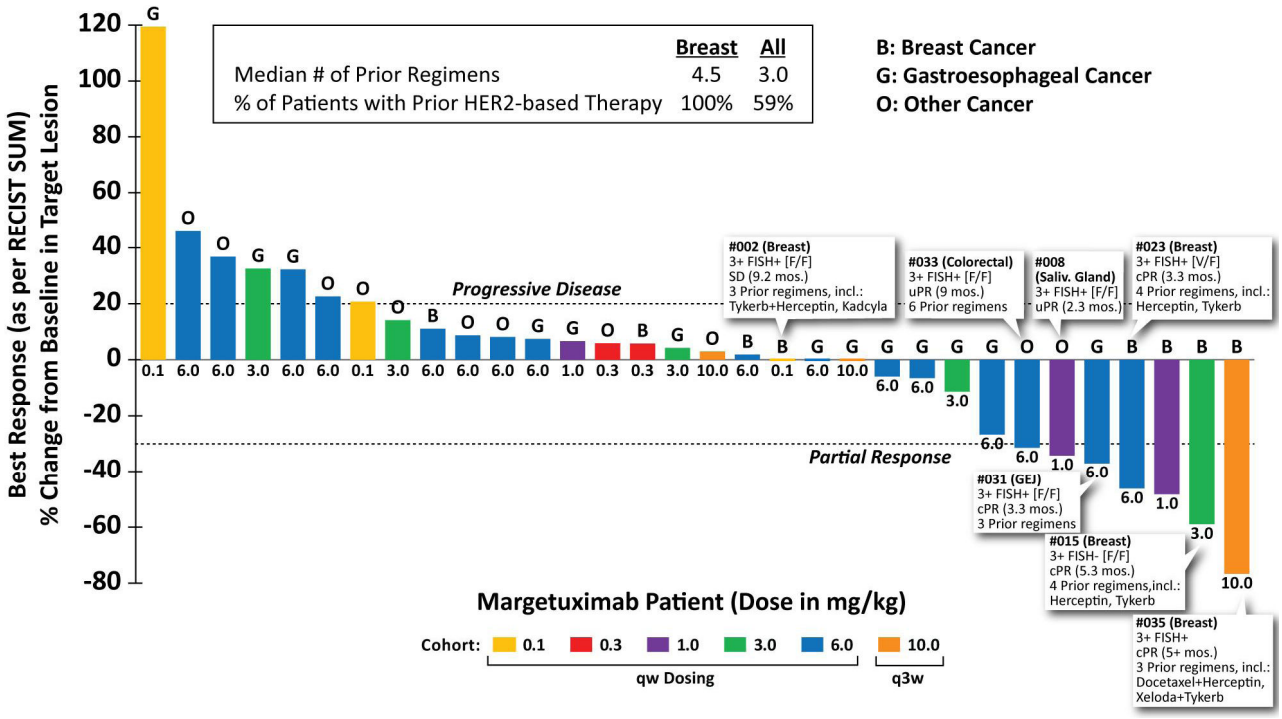
An open-label, multi-dose, single-arm, dose-escalation Phase I study in 34 advanced metastatic patients was conducted to test margetuximab's antitumor activity in refractory HER2+ tumors. Patients received one of five escalating doses between 0.1 to 6.0 mg/kg qw. An expansion cohort of 15 patients at 6.0 mg/kg weekly was also enrolled. Additionally, 6 patients have been enrolled in an alternative dosing regimen at 10 mg/kg q3w and 6 patients at 15 mg/kg q3w (expected to complete in 2014).

The study enrolled 10 breast, 13 gastroesophageal, 5 colon, 2 lung, 1 salivary, 1 ampulla of Vater, 1 endometrium, and 1 bladder cancer patients. Margetuximab was well-tolerated up to the highest tested dose of 6.0 mg/kg qw, which is above the preclinically predicted minimally effective dose of 0.1 mg/kg. The most common adverse events (AE) were Grade 1-2 constitutional symptoms and infusion-related reactions. AEs greater than or equal to Grade 3 were from a single infusion reaction. The study observed antitumor activities in the dose range used. Two of the breast cancer patients who had previously failed trastuzumab and lapatinib realized partial responses lasting 3.5 and 5.5 months. Four out of thirteen patients (30%) of the gastroesophageal cancer patients had stable disease ranging from 1.5 to 5.3 months with a median of 3.6 months with all of the patients, but one having previously failed anti-HER2 treatment.

These margetuximab clinical data are promising in comparison to a recent study on the use of docetaxel in the relapsed gastric cancer setting. In this study, docetaxel in the 3rd line setting for patients with relapsed gastric cancer who have undergone modified oxaliplatin-fluorouracil (m-FOLFOX)-4 and modified irinotecan-fluorouracil (m-FOLFIRI) treatments (n=33) reported time to progression to be 2.1 months (95% CI, 1.63-2.58) and median OS to be 4.7 months (95% CI, 3.20-6.20) with 15% of the patients experiencing PR, 27% experiencing SD, and 58% with PD (Lee, JH et al. Korean J Intern Med (2013) 28:314-321). Additionally, less severe adverse events were reported in the margetuximab study than the docetaxel study (including neutropenia (58%), anemia (15%) and thrombocytopenia (6%)). Combining the efficacy and safety profile to date supports the evaluation of margetuximab in the 3rd line metastatic gastric and gastroesophageal cancer settings.

Single agent tumor responses to date include: unconfirmed partial response for mucoepidermoid carcinoma of the salivary gland (1 patient, 1.0 mg/kg), unconfirmed partial response for colorectal cancer (1 patient, 6.0 mg/kg), confirmed partial responses for breast cancer (1 patient at 3.0 mg/kg and 1 patient at 6.0 mg/kg), confirmed partial response for gastroesophageal junction tumor (1 patient, 6.0 mg/kg), and time-to-progression exceeding five months (4 patients). Additionally, a breast cancer patient experienced a partial response with a time to progression surpassing 5 months while receiving 10.0 mg/kg every three weeks. Figure 11 shows the percent change of tumor size for patients treated with various doses of margetuximab. MacroGenics is moving forward with a 6.0 mg/kg dose in their Phase II studies in the refractory breast cancer setting. Infusion-associated adverse events are the most common adverse events noted in the dose escalation portion (~ 27%).

Figure 11: Phase I Responders as of October 2013



Source: MacroGenics

Margetuximab Ongoing and Anticipated Clinical Trials

A single arm, open-label, Phase IIa study in relapsed or refractory advanced breast cancer patients with HER2 overexpression at the 2+ level as determined by immunohistochemistry without HER2 gene amplification is currently enrolling. Patients in this study will receive 6 mg/kg on Days 1, 8, and 15 of 28-day cycles. The primary outcome will be tumor response by RECIST.

A randomized Phase III study of the addition of margetuximab to irinotecan or paclitaxel as 3rd line therapy in the advanced gastroesophageal cancer setting is anticipated to begin in the second half of 2014. The primary endpoint will be OS. The company also anticipates conducting exploratory studies in other HER2 expressing tumors such as metastatic bladder cancer.

MGA271 – Fc-optimized Monoclonal Antibody for B7-H3 Expressing Tumors

MGA271 is an Fc-optimized monoclonal antibody that targets the B7-H3 antigen. B7-H3 is a member of the B7 superfamily of molecules that regulate T-cell activity and therefore simultaneously blocking B7-H3 while recruiting and activating immune cells may alone, or in combination with other immunoregulatory therapeutics, initiate an antitumor effect. While there are multiple companies advancing products/product candidates that target other B7 members (Figure 12), MGA271's potential differentiation 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.

Immunohistochemical studies have identified B7-H3 on an array of solid tumors including kidney, glioblastoma, thyroid, gastric, breast, pancreas, prostate, melanoma, and ovarian cancers (Figure 13) and colorectal cancer (50% of patients). MGA271 is being developed in partnership with Servier, and MacroGenics retains commercial rights in North America, Japan, Korea and India.

Figure 12: Selected Immunoregulatory Products/Product Candidates

Antigen Presenting Cell or Immune Checkpoint Protein	T Cell	Function	Product	Development Phase (Sponsor)
B7-1 (CD80) or B7-2 (CD86)	CTLA4	Inhibitory	ipilimumab (Yervoy) Anti-CTLA4 tremelimumab	Marketed (Bristol-Myers) PhII (AstraZeneca) PhI/II (MedImmune)
B7-H1 (B7-H1) or B7-DC (PD-L2)	PD1 PDL1	Inhibitory	lambrolizumab/MK-3475 (PD1) nivolumab/BMS-936558 (PD1) BMS-936559 (PD-L1) AMP-224 (PD1) MPDL3280A (PDL1) pidilizumab/CT-011 (PD1) MEDI4736 (PD1) NP-12 (PD-1)	PhII/III (Merck) PhIII (Bristol-Myers) PhI (Bristol-Myers) PhI (GSK/Amplimmune) PhII (Roche/Genentech) PhII (CureTech Ltd) PhI (MedImmune/Astrazeneca) Pre-clinical (Aurigene)
B7-H2 (B7RP1)	ICOS	Activating	AMG557	PhI (Amgen/AstraZeneca)
B7-H3	Unknown	Inhibitory	MGA271	PhI (MacroGenics)
Lymphocyte activation gene 3 (LAG3)	LAG3	Inhibitory	IMP321	PhI/II (Immutep)
CD-27	CD-70	Activating	CDX-1127	Phase I/II (Celldex)
Indoleamine (2,3)-dioxygenase (IDO)	_	Blocks kynurenine inhibition of T-Cells	indoximod INCB024360	PhII (NewLink Genetics Corp) PhII (Incyte)

Source: MacroGenics, Wedbush Securities, Inc.

Figure 13: B7-H3 Overexpression in Solid Tumors

Fixed Tumor MicroArray	IHC Summary			
	B7-H3 Positive		2+ or Above	
Kidney Cancer*	77 / 78	99%	75 / 78	96%
Glioblastoma	65/66	98%	63/66	95%
Thyroid Cancer	34/35	97%	33/35	94%
Gastric Cancer	100/115	87%	100 / 115	87%
Breast Cancer **	119/164	73%	115/164	70%
Pancreas Cancer	69 / 78	88%	45 / 78	58%
Prostate Cancer	88 / 99	89%	51 / 99	52%
Melanoma	66 / 70	94%	32 / 70	46%
Ovarian Cancer*	43 / 60	72%	21 / 60	35%

* Also target expression in tumor vasculature, ** triple negative: 8/17 positive, 2+ or above (47%)

Source: MacroGenics

MGA271 Phase I Study

Patients with advanced B7-H3 (2/3+ based on IHC) expressing tumors (e.g., prostate cancer, pancreatic cancer, melanoma, ovarian cancer, glioblastoma, or renal cell carcinoma) received increasing doses ranging from 0.01 mg/kg to 15 mg/kg of MGA271 in an open-label, multi-dose, single-arm, multi-center, dose-escalation Phase I trial. With no dose limiting toxicity observed, a 15 mg/kg qw an expansion phase was initiated in Q3:13. There are three cohorts in the expansion phase. These cohorts (melanoma, prostate, and other tumors with a maximum of 5 patients per tumor type) of 15 subjects each are being recruited. Servier plans to initiate a separate study of up to 90 patients in other tumor types in Q4:13.

As of September 2013, MacroGenics has enrolled 26 patients through the dose escalation portion with 15 types of tumors. At the first tumor assessment, 10 patients received additional cycles of MGA271 and all have had stable disease and the most common adverse events were mild to moderate infusion reactions. This clinical trial is expected to be completed in 2014.

MacroGenics is also working on developing a B7-H3 companion clinical test with two third party vendors to identify patients potentially suitable for MGA271 therapy by their B7-H3 expression levels.

MGD006 – DART-Based Candidate Targeting IL3R for AML

Acute myeloid leukemia (AML) is caused by the proliferation of abnormal white blood cells leading to inadequate bone marrow function, and unless cured, death due to anemia, bleeding or serious infection. There are about 15,000 and 18,000 new cases each year in the US and Europe, respectively according to the American Cancer Society and the European Journal of Cancer.

MGD006 was developed from MacroGenics's DART platform and targets CD123, the interleukin-3 receptor (IL3R) and recruits, expands and activates T cells via CD3 binding. The IL3R alpha chain is expressed on leukemia cells and leukemic stem cells (LSCs), and is not expressed on normal hematopoietic stem cells. This preferential expression on diseased cells reduces the likelihood for hematologic toxicity in this already hematologically impaired patient population and as LSCs have been associated with the relapse of acute myeloid leukemia (AML), MGD006 therapy may increase the frequency and duration of complete remission. MGD006 is one of the DART product candidates covered by the option agreement between with Servier.

Validation of IL-3R for targeting leukemic stem cells for treatment of AML

Stemline Therapeutics Inc.'s (STML, OUTPERFORM, David Nierengarten) early demonstration of activity for SL-401 in AML provides clinical validation of this target. SL-401 is a protein therapeutic consisting of the first 388 amino acids of diphtheria toxin conjugated to human IL3. Results from a single one-week course of SL-401 in several malignancies are shown below (Figure 14).

Figure 14: Clinical Responses from a Single One-Week Course of SL-401 in Phase I/II Trial

	BPDCN (n=6)	AML *Relapsed, refractory* (n=59)	AML *≥3rd line* (n=35*)	AML *Not chemo candidate* (n=11)	MDS *Refractory, High risk* (n=7)
Tumor shrinkages / disease stabilization	83%	46%	43%	55%	43%
Tumor shrinkages	83% 4 CRs	25% 2 CRs	23% 1 CR	27%	29%

AML=Acute myeloid leukemia; MDS=Myelodysplastic syndrome; BPDCN=Blastic plasmacytoid dendritic cell neoplasm
CR = Complete response
*Subpopulation of relapsed, refractory

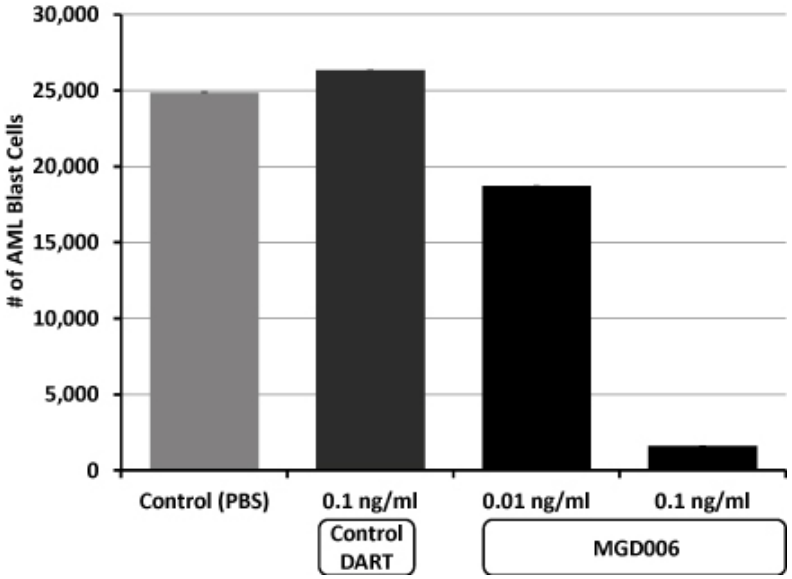
Source: Stemline Therapeutics, Inc.

MGD006 Pre-clinical Data and Anticipated Clinical Trial

Preliminary evidence of the activity of MGD006 has been obtained from *ex-vivo* studies in which the drug caused dose-dependent killing of human leukemia cells derived from AML patients (Figure 15). Data to be presented at the upcoming ASH meeting is, we believe, highly suggestive that MGD006 will be active in the clinic. The most compelling finding from this *ex vivo* study using human AML samples was the observation that MGD006 elicited a dramatic expansion of T cells (2-15 fold) from the limited number that would have been present in the sample, and a concentration dependent reduction in blasts. While a pre-clinical observation, it is important to note that the anticancer effects demonstrated in this study are immunologically mediated and this represents an important proof-of-concept result for MacroGenics' DART platform, product candidates and the DART programs being pursued by partners.

Phase I is anticipated in H1:14.

Figure 15: Dose-Dependent Killing of Primary AML PBMC



Source: Company data

MGD007: Dart-Based Product Candidate for Colorectal Cancer

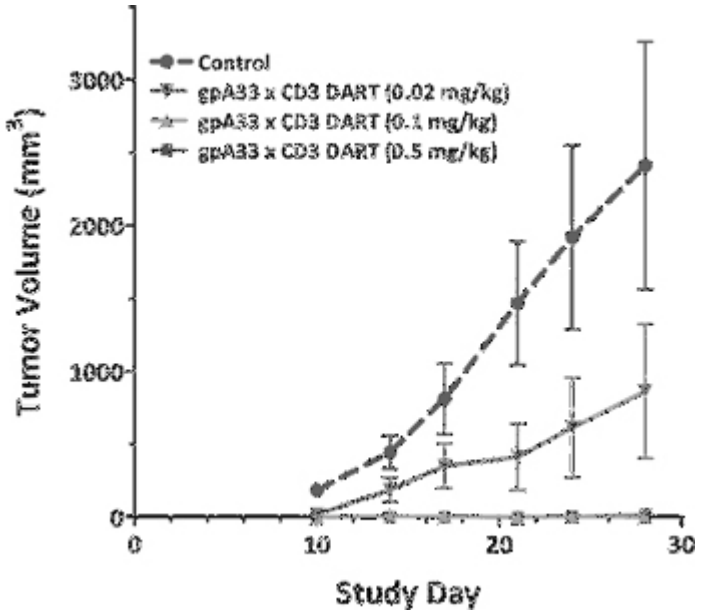
According to SEER, colorectal cancer is responsible for 142,820 new cases and 50,830 deaths in the U.S. The average 5-year survival rate is 64.9% and the median age of patients diagnosed is 69 years.

MGD007 was developed with MGNX's DART platform and targets glycoprotein gpA33 and CD3 for the potential treatment of colorectal cancer and potentially other gastrointestinal tumors (pancreatic, gastroesophageal). The Fc-domain of MGD007 has been engineered to extend serum half-life. The cell surface antigen gpA33 is present on more than 95% of human colon cancers and approximately half of gastroesphogeal and pancreatic cancers. MGD007 is one of the DART product candidates in the option agreement between MacroGenics and Servier.

MGD007 Pre-clinical Data

MacroGenics has completed proof of concept *in vitro* and *in vivo* studies of MGD007 that demonstrate T cell-mediated removal of gpA33 expressing cancer cells. Immunodeficient mice implanted with human colorectal cancer cells and activated human T cells were administered various doses of MGD007 (0.02 mg/kg to 0.5 mg/kg) or an MGD007 control (lack Fc-domain). A dose-dependent stabilization of tumor size was seen (Figure 16). MacroGenics plans to design and initiate the IND enabling GLP toxicology study by YE:13. An IND submission is expected in 2014 and initiation of Phase I in H2:14.

Figure 16: *In Vivo* Activity of MGD007 in Mice



*immunodeficient mice were implanted with human colorectal cancer and activated human T cells

Source: MacroGenics

MGD010: DART-Based Ab for Autoimmune Diseases

MGD010 is an antibody therapeutic developed from the DART platform that targets CD32B and CD79B. MGD010 was engineered to regulate the B cell immune response through preferential blockade of the activated B cells that promote an autoimmune response. CD32B is an inhibitory Fc receptor present on B cells and myeloid dendritic cells, and normally functions as a negative regulator to prevent attack on self and autoimmunity. Engagement of CD32B prevents the expansion of B cell clones that share the same specificity as the engaged IgG. CD79B is the beta chain of CD79, a transmembrane protein that complexes with the B-cell receptor. CD79B is expressed almost exclusively on B cells and B-cell neoplasms and contains an intracellular immunoreceptor tyrosine-based activation motif that can initiate a signal transduction in the B cell.

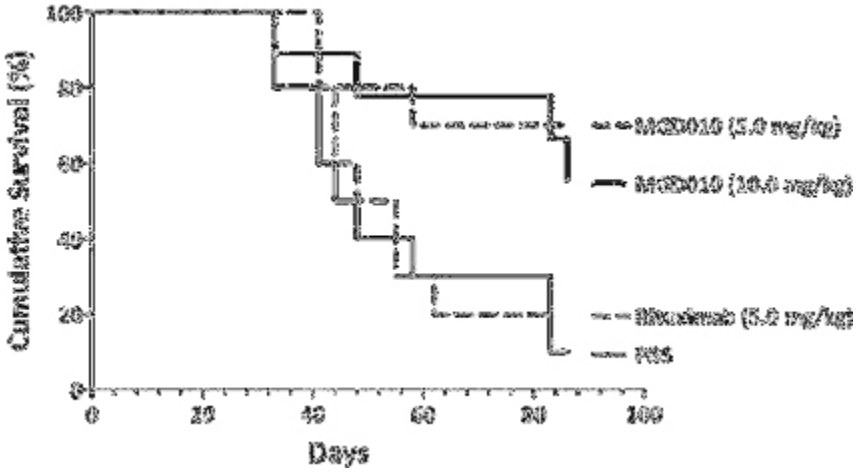
By blocking activated B cells, MGD010 may have utility in the B-cell mediated autoimmune diseases rheumatoid arthritis (RA), Crohn's, systemic lupus erythematosis (SLE), or multiple sclerosis (MS). Current treatments include corticosteroids, anti-inflammatory agents such as tumor necrosis factor inhibitors, anticytokine therapies, inhibition of intracellular pathways, costimulation inhibition, T cell inhibition, B cell depletion, and stem cell transplantation. Current B cell therapies have limitations due to either depletion of B cells that can lead to infections or limited efficacy and delayed onset. In *ex vivo* studies using cells from SLE patients, MGD010 was shown to block B cell activation without B cell depletion.

There is an estimated 127,000 new RA, 22,382 new SLE, and 4,650 new MS cases in the U.S. each year.

MGD010 Preclinical Data

SLE patient B cells and humanized mouse models treated with MGD010 showed blockage of B cell activation in the absence of B cell depletion. A mouse model of graft versus host disease showed prolonged survival when treated with MGD010 (5 or 10mg/kg) compared to mice on PBS or rituximab (5 mg/kg) (Figure 17). Studies of MGD010 performed in non-human primates demonstrated a favorable safety profile, and no B-cell depletion suggesting a potentially safer approach to reducing B-cell mediated autoimmunity without increasing the risk of infection.

Figure 17: Efficacy of MGD010 in a chronic graft versus host disease (transplantation) mouse model



Immunodeficient mice were administered human PBMC's by injection and treated intravenously with MGD010 (5.0 or 10.0 mg/kg) or PBS control every four days (9 doses) or with rituximab at 5.0 mg/kg (1 dose).

Source: MacroGenics

Teplizumab: Fc-Modified Antibody for Type I Diabetes

Teplizumab is an anti-CD3 antibody developed for Type I Diabetes (T1D). The product is designed to bind to CD3 receptors on the T cell and subsequently alter the function of the T cells that mediate the removal of the insulin-producing pancreatic beta cells.

According to the 2011 National Diabetes Fact Sheet, there are 25.8 million people with diabetes and 1.9 million new cases of diabetes in individuals 20 years or older. Using the American Diabetes Association's 5% estimate, there are 1.29 million people with Type I diabetes and 95,000 new cases per year.

Teplizumab is currently in a Phase 2 study for the prevention or delay of T1D onset for patients with high risk. This study is being sponsored and conducted by the National Institute of Diabetes and Digestive and Kidney Diseases.

Between 2007 and 2011, this asset was being developed in collaboration with Eli Lilly. The partnership was terminated due to failure of meeting the primary endpoints of a Phase 3 clinical study. MacroGenics has reacquired full commercial rights for teplizumab. The primary endpoints for this study were a composite glycated hemoglobin (HbA1c) level of <6.5%, which is an indicator of average plasma glucose concentration over long periods of time, and insulin usage of <0.5 units per day. Insulin only placebo arm and the 14-day teplizumab regimen experimental arm had comparable readouts. A post-hoc analysis suggests that a full dose regimen of teplizumab may preserve beta cell insulin production.

Figure 18: Agreements/Partnerships

Partner/Product	Terms
Servier MGA271 and DART-based molecules	(2011) Ex-NA and Asian rights to MGA271. MGNX has received a $20 million up-front payment (option fee) and a $10 million milestone payment. MGNX could receive up to $415 million in pre- and post-commercialization payments if Servier exercises the option and successfully commercializes MGA271. (2012) This second agreement granted Servier options on three separate DART-based molecules (MGD006, MGD007, and a third), in all countries other than the United States, Canada, Mexico, Japan, South Korea and India. Servier made an initial $20 million option fee payment and MGNX could receive up to $1 billion in pre- and commercial milestones (cumulative across all three products). Under both the 2011 and 2012 option agreements, Servier pays low double digit to mid-teen royalties on product sales in its territories.
Gilead DART-based molecules	(2013) Exclusive worldwide (except for one which MGNX retains Asian rights) license to up to four DART-based molecules. MGNX received an initial $7.5 million license grant fee for the first DART-based molecule and could receive up to an additional $22.5 million on the other three molecules. MGNX could also receive up to an additional $85 million in pre-clinical milestones across the four DART programs and approximately $1 billion in additional clinical, regulatory and sales milestone payments if Gilead exercises all four of the options and achieves all of the requisite milestones under each option and license. Gilead also funds internal and external research costs. MGNX could receive tiered high-single to low double digit (max 12%) royalties on the net sales.
Boehringer Ingelheim DART-based molecules	(2010) This research and licensing agreement covers the discovery, development and commercialization of up to ten DART-based molecules across multiple therapeutic areas. For the worldwide exclusive royalty-bearing license BI paid an upfront payment of $15 million. MGNX has received three annual maintenance payments, the most recent of $4 million received in Q4:13. MGNX also just received a $5 million milestone for the recent DART candidate nomination on November 11, 2013. MGNX could earn undisclosed development, regulatory and sales milestones and royalty payments for each of the DART programs. BI supports both internal and external research costs under the agreement.
Pfizer DART-based molecules	(2010) Initiated in October 2010 this three year deal calls for the discovery, development and commercialization of up to two DART-based molecules. Pfizer received a non-exclusive worldwide, royalty-bearing license and MGNX received upfront and milestone payments and funding for their internal and external research costs. MGNX could receive additional milestones and royalty payments for each drug. Presently, one DART program remains ongoing it is anticipated that the research component of the deal will conclude in January 2014.
Green Cross margetuximab	(2010) This deal grants Green Cross an exclusive license to conduct clinical trials and commercialize margetuximab in South Korea. MacroGenics received a $1 million upfront fee and may receive clinical, development and commercial milestone payments of up to $4.5 million. There can also receive royalties of low-single digits to low twenties on net sales. Green Cross is responsible for commercialization of margetuximab in South Korea and MacroGenics will supply margetuximab for the clinical South Korean clinical development.

Source: Company data, Wedbush Securities, Inc.

MANAGEMENT

Dr. Scott Koenig – Co-founder, President and CEO. Mr. Koenig joined Macrogenics in September 2001. Prior to MacroGenics, Dr. Koenig served as Senior Vice President of Research at MedImmune Inc. From 1984 to 1990, he worked in the Laboratory of Immunoregulation at the National Institute of Allergy and Infectious Diseases at the National Institutes of Health. Dr. Koenig received his A.B. and Ph.D. from Cornell University and his M.D. from the University of Texas Health Science Center in Houston.

James (Jim) Karrels – CFO. Mr. Karrels joined as Chief Financial Officer in May 2008. Prior to MGNX, he was at Jazz Pharmaceuticals, Inc., most recently serving as Executive Director of Finance. Prior to Jazz Pharmaceuticals, Mr. Karrels spent 11 years in the Investment Banking Group at Merrill Lynch, most recently serving as a Director in the Global Healthcare Group. Mr. Karrels holds an M.B.A. from Stanford University and a B.B.A. from the University of Notre Dame.

Dr. Bonvini - Senior Vice President, Research. Dr. Bonvini joined in June 2003. From 1985 to 2003, Dr. Bonvini was with the FDA in the Center for Biologics Evaluation and Research ultimately serving as Acting Deputy Director, Division of Monoclonal Antibodies and Chief, Laboratory of Immunobiology. From 1982 to 1984, Dr. Bonvini was a Visiting Fellow at the National Cancer Institute at the National Institutes of Health. Dr. Bonvini received a Diploma in Science from the Scientific Lyceum in Genoa, Italy, and his M.D. and Specialty Certification in Clinical Hematology from the University of Genoa, School of Medicine.

Dr. Stein - Vice President, Product Development and Regulatory Affairs. Dr. Stein joined in May 2002 and has served as a Senior Vice President since 2006. From 1980 to 2002, Dr. Stein was at the FDA, including serving as Director, Division of Monoclonal Antibodies in the Office of Therapeutics Research and Review at CBER from 1992 to 2002. Dr. Stein received her Ph.D. in Microbiology and Immunology from the Albert Einstein College of Medicine of Yeshiva University and her B.A. in Chemistry from Bard College.

Dr. Wigginton - Senior Vice President, Clinical Research. Dr. Wigginton joined in August 2013. Dr. Wigginton was previously the Therapeutic Area Head, Immuno-Oncology, Early Clinical Research and Executive Director, Discovery Medicine-Clinical Oncology at Bristol-Myers from October 2008 to August 2013. Prior to Bristol-Myers, Dr. Wigginton was the Director of Clinical Oncology at Merck Research Laboratories from May 2006 to October 2008. Dr. Wigginton received his M.D. and B.S. in Biology from the University of Michigan.

Dr. Stewart - Vice President, Clinical Oncology Research. Dr. Stewart joined in July 2008. From 2005 to 2008, Dr. Stewart served as Vice President, Clinical Research at Raven Biotechnologies, Inc., which MGNX acquired in July 2008. From 2001 to 2005, Dr. Stewart was with Corixa Corporation, most recently as Vice President, Clinical Research. Dr. Stewart was with ALZA Corporation in 2001 and from 1998 to 2001, he was with Genentech, where he was Clinical Scientist on the Herceptin project. Dr. Stewart trained in Medical Oncology at Stanford University, and served as a member of the faculty of the School of Medicine at Vanderbilt University for more than twelve years. Dr. Stewart received his M.D. from Baylor College of Medicine and his B.A. degree from Rice University.

Figure 19: MGNX Clinical Trials

Product (Indication)	Clinical Trial Status and Results	Next Event
Margetuximab **(metastatic breast cancer and gastroesophageal cancer)**	**Phase IIa in refractory breast HER2+ (2+ by IHC and no gene amplification) setting** is currently enrolling **Phase I in the refractory HER2+ tumors settings** Setup: 34 patients received one of five escalating doses between 0.1 to 6.0 mg/kg qw. An expansion cohort of 15 patients at 6.0 mg/kg weekly was also tested. Additionally, 6 patients have been enrolled for alternative dosing at 10 mg/kg q3w and 6 patients at 15 mg/kg q3w Responses to date: unconfirmed partial response for mucoepidermoid carcinoma of the salivary gland (1 patient, 1.0 mg/kg), unconfirmed partial response for colorectal cancer (1 patient, 6.0 mg/kg), confirmed partial responses for breast cancer (1 patient at 3.0 mg/kg and 1 patient at 6.0 mg/kg), confirmed partial response for gastroesophageal junction tumor (1 patient, 6.0 mg/kg), and time-to-progression exceeding five months (4 patients). Additionally, in testing intermittent administration of margetuximab, a breast cancer patient experienced partial response with a time to progression surpassing 5 months while receiving 10.0 mg/kg every three weeks.	**2014:** Complete Phase I **2014:** Complete enrollment of Phase 2a study in the metastatic **H2:14:** Commence Phase III clinical trial to evaluate the addition of margetuximab to standard cytotoxic chemotherapy (irinotecan or paclitaxel) in the third line treatment of patients with advanced gastroesophageal cancers
MGA271 (solid tumors)	**Phase I in B7-H3 (2/3+ based on IHC) expressing tumors** Setup: Patients with advanced B7-H3 (2/3+ based on IHC) expressing tumors received increasing doses ranging from 0.01 mg/kg to 15 mg/kg of MGA271 in an open-label, multi-dose, single-arm, multi-center, dose-escalation Phase I trial. With no dose limiting toxicity observed, a 15 mg/kg qw expansion phase was initiated in Q3:13. There are four cohorts in the expansion phase. Three cohorts (melanoma, prostate, and other tumors with a maximum of 5 patients per tumor type) of 15 subjects each are being recruited. An immunohistochemistry based companion diagnostic is planned to be incorporated in PhIII trials.	**Q4:13:** Servier enrolls up to 90 additional patients representing additional types of cancers **2014:** Complete Phase I **H1:15 or earlier:** Initiate Phase II

Source: Company data, Wedbush Securities, Inc.

Figure 20: Valuation Table

Product	Indication	US Launch Date	Valuation Year/ Multiple/Discount Rate	Sales ($K)	Royalties ($K)	Valuation ($K)	Value/Share ($)	Total Value/Share ($)
margetuximab	Breast cancer	6/6/2018	2022 / 6x / 30%	770,512	N/A	566,740	16.85	34.03
	Breast Cancer, Adjuvant	1/1/2022	2024 / 6x / 35%	398,512	N/A	118,920	3.54	
	Gastric Cancer	6/6/2017	2021 / 6x / 30%	363,399	N/A	347,481	10.33	
	Bladder Cancer	6/6/2019	2021 / 6x / 30%	58,962	N/A	56,379	1.68	
	Esophageal Cancer	6/6/2019	2022 / 6x / 30%	74,988	N/A	55,157	1.64	
MGA271	Melanoma	6/6/2020	2024 / 6x / 35%	721,363	N/A	215,262	6.40	13.84
	Melanoma (Servier)	6/6/2021	2024 / 15x / 35%	667,167*	80,060	59,727	1.78	
	Renal Cell Carcinoma	6/6/2020	2024 / 6x / 35%	512,696	N/A	152,994	4.55	
	Renal Cell Carcinoma (Servier)	6/6/2021	2024 / 15x / 35%	417,474*	50,097	37,374	1.11	
MGD006	Acute Myeloid Leukemia	1/1/2019	2022 / 6x / 30%	284,591	N/A	209,327	6.22	10.74
	Acute Myeloid Leukemia (Servier)	1/1/2020	2024 / 15x / 35%	314,676*	37,761	28.171	0.84	
	Other IL-3R Rare Cancers	1/1/2019	2022 / 6x / 30%	168,023	N/A	123,587	3.67	
MGD007	Colorectal Cancer	1/1/2021	2025 / 6x / 35%	494,115	N/A	109,221	3.25	11.21
	Colorectal Cancer, Adjuvant	1/1/2024	2025 / 6x / 35%	523,313	N/A	115,675	3.44	
	Colorectal Cancer (Servier)	2/1/2022	2024 / 15x / 35%	1,020,135*	122,416	91,326	2.72	
	Colorectal Cancer, Adjuvant (Servier)	2/1/2025	2026 / 15x / 35%	1,236,961*	148,435	60,761	1.81	
DARTs	*Various*	*2017 - 2025*	*2024 / 15x / 35%*	*6,970,115**	*468,599*	*349,586*	*10.39*	*10.39*
								$70/share

*Partner's Sales; Outside of Servier's partnership on MGA271, MGD006, and MGD007, DART partnerships ($10.39 per share) are not included in the $70/share valuation.

Source: Company data, Wedbush Securities, Inc.

12/3/2013
Ticker: (MGNX:Nasdaq)
Macrogenics, Inc.

WEDBUSH
PAC GROW LIFE SCIENCES
Gregory R. Wade, Ph.D.
415-274-6863

Macrogenics, Inc (MGNX)
in thousands except per share data

	2012A	Q1A	Q2A	Q3A	Q4E	2013E	2014E	2015E	2016E	2017E	2018E	2019E	2020E
Revenues:													
Product Sales	$0	$0	$0	$0	$0	$0	$0	$0	$0	$268	$16,541	$256,575	$889,203
Royalties	$0	0	0	0	0	0	0	0	0	96	15,584	63,371	132,347
Milestones and collaboration revenues	$63,826	11,448	11,448	20,232	5,000	48,128	16,000	20,000	20,000	0	0	0	0
Total Revenues	**63,826**	**11,448**	**11,448**	**20,232**	**0**	**48,128**	**16,000**	**20,000**	**20,000**	**364**	**32,124**	**319,946**	**1,021,551**
Cost and Expenses:													
Cost of Sales	0	0	0	0	0	0	0	0	0	54	3,308	51,315	177,841
R&D	45,433	10,573	10,573	11,088	13,500	45,734	54,000	56,755	61,433	66,497	71,978	79,977	177,841
SG&A	10,188	2,668	2,668	1,987	2,700	10,023	11,924	13,611	16,222	20,639	24,158	74,565	207,417
Total Operating Expenses	**55,621**	**13,241**	**13,241**	**13,074**	**16,200**	**55,756**	**65,924**	**70,365**	**77,655**	**87,190**	**99,444**	**205,857**	**563,098**
Operating Income (Loss)	8,205	(1,793)	(1,793)	7,158	(16,200)	(7,628)	(49,924)	(50,365)	(57,655)	(86,826)	(67,320)	114,089	458,452
Net Interest Income (Expense)/Other Income	157	37	37	(554)	(550)	(1,030)	(6,064)	(9,164)	(11,730)	(10,300)	(5,481)	(3,555)	(15,799)
Income Before Income Taxes	8,362	(1,756)	(1,756)	6,604	(16,750)	(8,658)	(55,989)	(59,530)	(69,384)	(97,126)	(72,802)	110,534	442,653
Provision for Income Taxes	0	0	0	350	0	350	0	0	0	0	0	5,858	72,398
Net Income (Loss)	8,362	(1,756)	(1,756)	6,254	(16,750)	(9,008)	(55,989)	(59,530)	(69,384)	(97,126)	(72,802)	104,676	370,255
GAAP EPS	**0.39**	**(0.09)**	**(0.09)**	**0.29**	**(0.47)**	**(0.53)**	**(2.25)**	**(2.19)**	**(2.48)**	**(3.25)**	**(2.43)**	**3.17**	**11.17**
Total Shares Outstanding	17,825	19,022	19,022	19,022	24,772	16,946	24,834	27,172	28,009	29,909	30,009	30,109	30,209
Cash Burn	44,154					(9,008)	(55,989)	(59,530)	(69,384)	(97,126)	(72,802)	104,676	370,255
Cash Balance	47,743	37,930	33,781	33,569	113,456	113,456	97,965	204,425	135,431	116,239	42,406	127,623	446,120

Source: Wedbush PacGrow Life Sciences

Covered Public Companies Mentioned in this Report (priced intraday 12/03/13):

COMPANY	TICKER	RATING	PRICE	PRICE TARGET
Stemline Therapeutics	STML	OUTPERFORM	$21.09	$49
Celldex Therapeutics	CLDX	OUTPERFORM	$27.05	$40

Analyst Biography

Gregory Wade, Ph.D.

Greg is a Managing Director and joined Wedbush in March 2009 from Pacific Growth Equities where he was a Senior Research Analyst covering emerging Pharmaceutical and Biotechnology companies. He started at Pacific Growth in February 2000 as a Research Associate and became an Analyst in 2004. Prior to Pacific Growth Equities, Greg was a Director in the business development group at ISIS Pharmaceuticals and prior to that was with Procyon BioPharma in London, Canada. While completing his Ph.D. in Physiology at the University of Western Ontario Greg worked as an Associate at the venture capital company Helix Investments Canada where he focused on early stage investments in life science companies.
Greg's team includes Drs. David Nierengarten (Analyst) and Chris Marai (Analyst) and together they cover 40+ companies focused on antibiotics, rare diseases, prostate cancer, hematology/oncology, gastrointestinal disorders, vaccines, biodefense and drug/device combinations.
Greg's Edge: Greg's edge comes from the breadth and duration of his tenure on the sell-side. Coverage of nearly 60 different companies over 14 years provides him with a measured perspective and industry and key opinion leader contacts help to inform his view.

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

Disclosure information regarding historical ratings and price targets is available at http://www.wedbush.com/ResearchDisclosure/DisclosureQ313.pdf

Investment Rating System:
Outperform: Expect the total return of the stock to outperform relative to the median total return of the analyst's (or the analyst's team) coverage universe over the next 6-12 months.
Neutral: Expect the total return of the stock to perform in-line with the median total return of the analyst's (or the analyst's team) coverage universe over the next 6-12 months.
Underperform: Expect the total return of the stock to underperform relative to the median total return of the analyst's (or the analyst's team) coverage universe over the next 6-12 months.

The Investment Ratings are based on the expected performance of a stock (based on anticipated total return to price target) relative to the other stocks in the analyst's coverage universe (or the analyst's team coverage).*

Rating Distribution (as of September 30, 2013)	**Investment Banking Relationships (as of September 30, 2013)**
Outperform:55% Neutral: 41% Underperform: 4%	Outperform:14% Neutral: 2% Underperform: 0%

The Distribution of Ratings is required by FINRA rules; however, WS' stock ratings of Outperform, Neutral, and Underperform most closely conform to Buy, Hold, and Sell, respectively. Please note, however, the definitions are not the same as WS' stock ratings are on a relative basis.

The analysts responsible for preparing research reports do not receive compensation based on specific investment banking activity. The analysts receive compensation that is based upon various factors including WS' total revenues, a portion of which are generated by WS' investment banking activities.

Wedbush Equity Research Disclosures as of December 3, 2013

Company	Disclosure
MacroGenics	1,3,4,5,7
Celldex Therapeutics	1,3,4,5
Stemline Therapeutics	1

Research Disclosure Legend
1. WS makes a market in the securities of the subject company.
2. WS managed a public offering of securities within the last 12 months.
3. WS co-managed a public offering of securities within the last 12 months.
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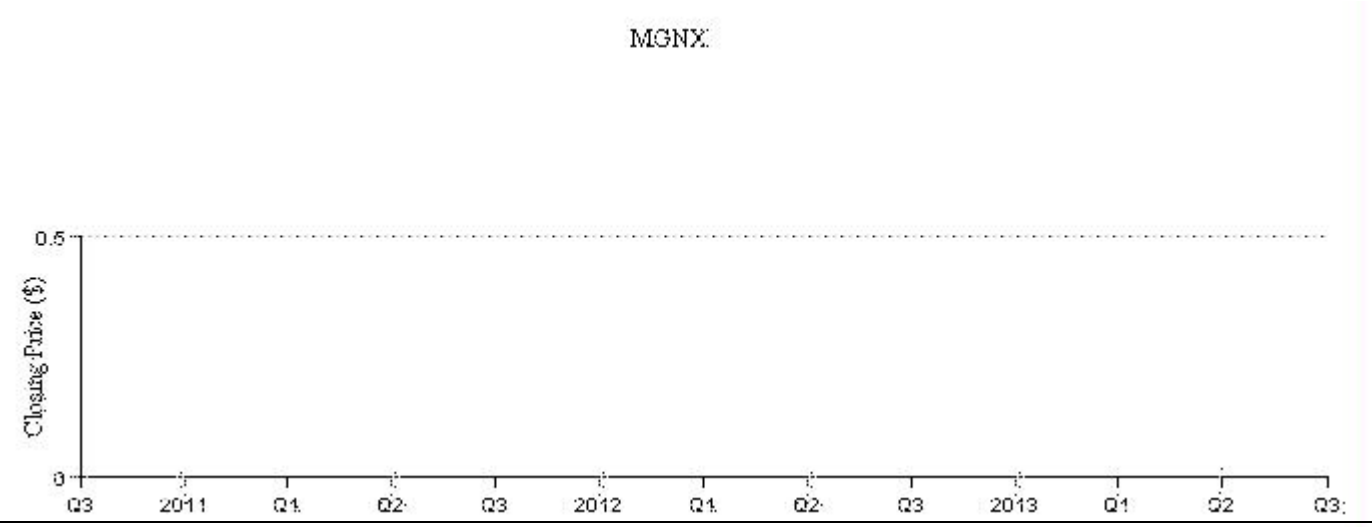

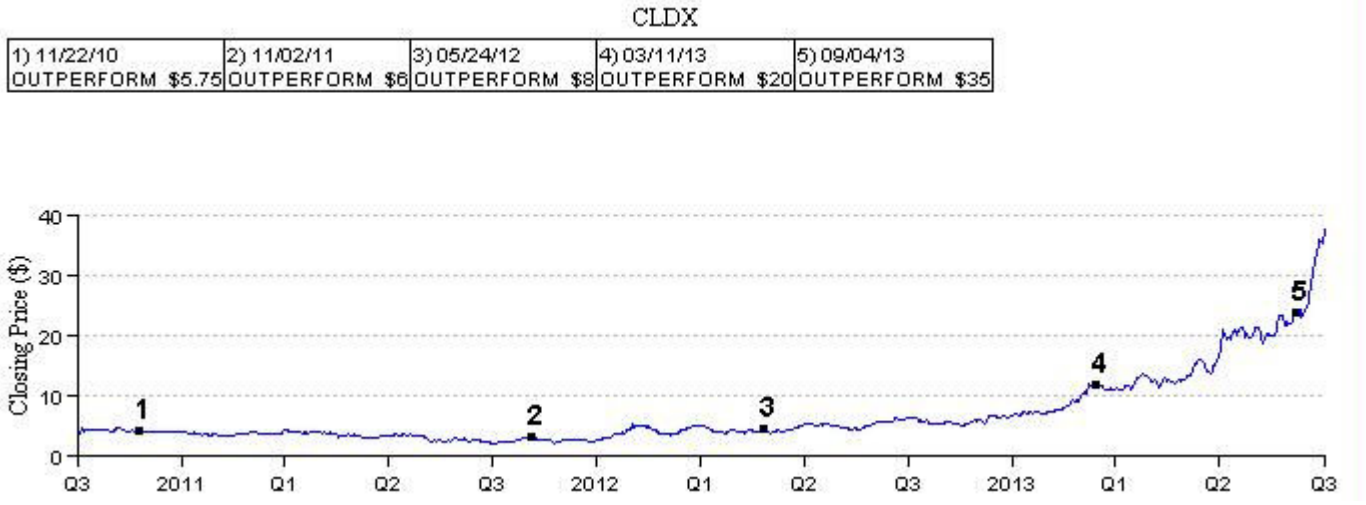

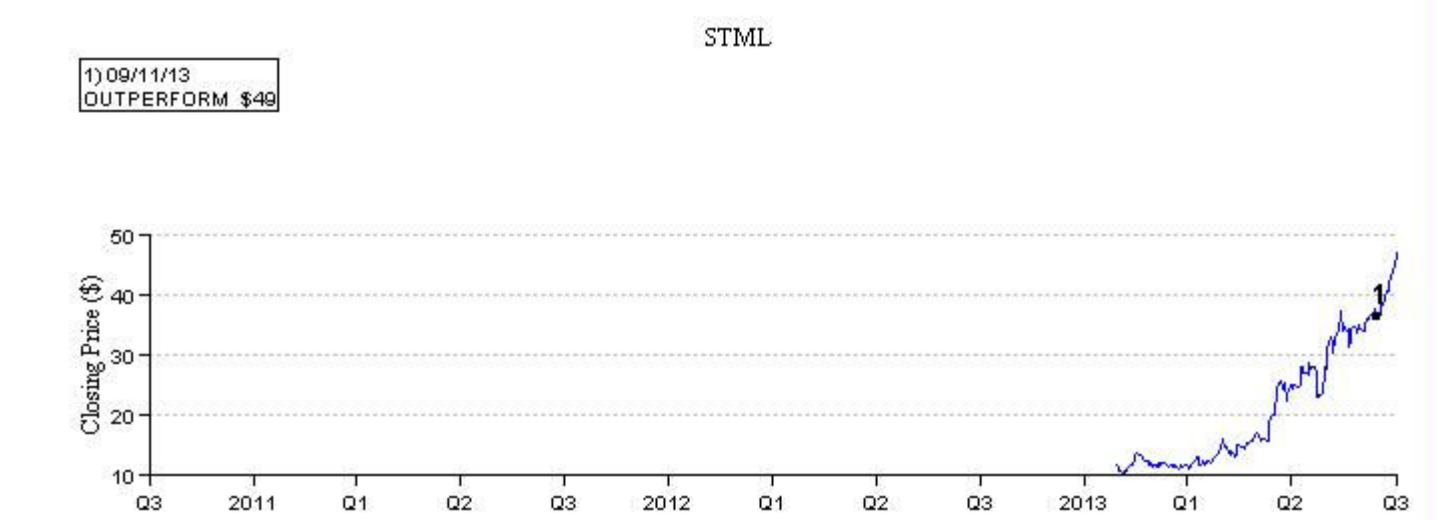

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