November 4, 2013

OUTPERFORM

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Reason for report:

INITIATION

LEERINK SWANN

HEALTHCARE EQUITY RESEARCH

MACROGENICS, INC.

Antibody Platform and a Growing Pipeline; Initiate at Outperform

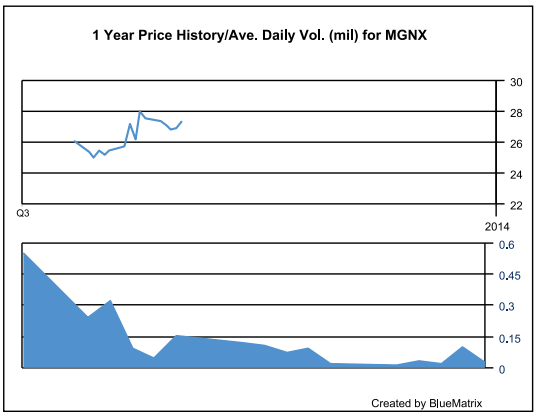
• **Bottom Line**: We initiate coverage of MGNX with an Outperform rating and a $34 price target. MGNX is a leader in the area of immune-modulation and is a fully integrated R&D driven biotech company. The company is focused on developing new antibody-based therapeutics for cancer and autoimmune diseases and is based on a suite of platform technologies that allow generation of therapeutic antibodies with superior properties. MGNX has applied its antibody discovery and engineering platform to generate a proprietary product pipeline and to enter into major strategic collaborations that provide the company with funding and leverage the additional expertise of partners.

• **Lead product candidate Margetuximab is an Fc-enhanced anti-Her2 antibody in Phase IIa trials** for treatment of metastatic breast cancer patients with only moderate Her2 expression and who are not eligible for Herceptin or Kadcyla therapy. We believe positive Phase IIa data in late 2014 would significantly derisk this program and preclinical and Phase I data support activity of the therapeutic antibody, which could expand the addressable market of Herceptin (Roche).

• **MGA271, is a first-in-class, Fc-enhanced monoclonal antibody that targets B7-H3, currently in Phase Ib trials for a wide range of solid tumors**. B7-H3 is a tumor-specific antigen and a novel member of the B7 family of immune regulators. We believe MGA271 addresses a promising new target in immuno-oncology that could be active in a wide array of solid tumor indications, based on preclinical data. Positive Phase I expansion phase data in 2014 will be a key catalyst for this program for which Servier has an option to license European rights. Based on our due-diligence, we believe MGA271 is attractive, since it has potentially two mechanisms by which it could exert its anti-cancer activity: 1) tumor cell-killing via ADCC, and 2) enhancement of anti-tumor immunity by blockade of T-cell inhibition.

• **Partnerships with GILD (OP), Boehringer Ingelheim, PFE (MP), and Servier validate MGNX's leading bi-specific antibody ("DART") platform, in our view.** We believe MGNX's technology has potential advantages over other bi-specific mAb technologies including BiTEs (AMGN [MP]), since it can generate highly stable molecules that are potentially more active. The company qualifies for an impressive $5Bn in total theoretically possible milestone payments plus royalties from its partners which cover up to 19 drugs. Although it is uncertain whether each of the 19 DARTs will be developed, we believe the MGNX platform has already demonstrated the ability to generate cashflows for the company. MGNX received $106MM in milestone payments over the last 3 years, and we believe there is a high likelihood that it will receive at least another $100MM through 2015 as preclinical programs advance. The first two partnered DARTs will enter the clinic in 2014.

Key Stats:	(OTC Un:MGNX)
S&P 600 Health Care Index:	1,208.38
Price:	$27.32
Price Target:	$34.00
Methodology:	DCF with 12% discount rate
52 Week High:	$29.30
52 Week Low:	$23.10
Shares Outstanding (mil):	24.8
Market Capitalization (mil):	$677.5
Book Value/Share:	$0.00
Cash Per Share:	$3.95
Dividend (ann):	$0.00
Dividend Yield:	0.0%



Dec Yr	1Q	2Q	3Q	4Q	FY Rev	1Q	2Q	3Q	4Q	FY EPS	
2012A	--	--	--	--	$63.8	--	--	--	--	$7.72	NM
2013E	$10.6A	$12.3A	$20.5	$10.5	$53.8	($2.80)A	($0.29)A	$2.79	($0.34)	($1.06)	NM
2014E	--	--	--	--	$44.0	--	--	--	--	($1.53)	NM
2015E	--	--	--	--	$48.0	--	--	--	--	($2.22)	NM

Source: Company Information and Leerink Swann LLC Research
Revenues in $MM; GAAP EPS

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The Healthcare Investment Bank™

MacroGenics, Inc. (NASDAQ: MGNX)
Initiation of Coverage
Outperform

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MGNX Investment Thesis

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- **MacroGenics (Nasdaq: MGNX) is a leader in the area of immune-modulation and is a fully integrated R&D driven biotechnology company based in Rockville, MD**. The company is focused on developing new antibody-based therapeutics for cancer and autoimmune diseases and is based on a suite of platform technologies that allow generation of therapeutic antibodies with superior properties. MGNX has applied its antibody discovery and engineering platform to generate a proprietary product pipeline and to enter into strategic collaborations that provide the company with funding and leverage the additional expertise of partners.
- **Lead product candidate Margetuximab is a Fc-enhanced anti-Her2 antibody that is in Phase IIa trials for treatment of metastatic breast cance**r patients with moderate Her2 over expression who are not eligible for Herceptin or Kadcyla therapy. We believe positive Phase IIa data in late 2014 would significantly derisk this program and preclinical and Phase I data support activity of the therapeutic antibody, which could significantly expand the addressable market of Herceptin (Roche). Based on our model we believe margetuximab addresses a market opportunity of $590MM in the US in metastatic breast and gastric cancer.
- **MGA271, is a first-in-class, Fc-enhanced monoclonal antibody that targets B7-H3, currently in Phase Ib trials for a wide range of solid tumors.** B7-H3 is a tumor-specific antigen and a novel member of the B7 family of immune regulators. We believe MGA271 addresses a promising new target in the area of immuno-oncology that could be active in a wide array of solid tumor indications, based on preclinical data. Positive Phase I expansion phase data in 2014 will be a key catalyst for this program for which partner Servier has an option to license European rights. Based on our due-diligence we believe MGA271 has potentially two mechanisms by which it could exert its anti-cancer activity: 1) tumor cell-killing via ADCC, and 2) enhancement of anti-tumor immunity by blockade of T-cell inhibition.
- **Partnerships with Gilead (OP), Boehringer Ingelheim, Pfizer (MP), and Servier validate MGNX's leading bi-specific antibody ("DART") platform, in our view**. We believe MGNX's technology has potential advantages over other bi-specific mAb technologies including BiTEs (AMGN), since it can generate highly stable DARTs that are more active. Existing partnerships validate the DART (Dual Affinity Re-Targeting) platform in our view and additional partnerships could be sources of upside. The company currently theoretically qualifies for an impressive $5Bn in total potential milestone payments from existing partners. MGNX received over $100MM in milestone payments over the last 3 years, and we believe there is a high likelihood that it will receive at least $100MM until 2015 as preclinical programs advance

Valuation and Risks

- Valuation:
  - We estimate a $34 fair value for MGNX shares in 12 months, based on a discounted cash flow (DCF) sum-of-parts analysis. We use a 12% discount rate for probability of success-weighted margetuximab (25%) and MGA271 (12%) sales. Based on our DCF analysis, we attribute $8/share to margetuximab, $9/share to MGA271, and $14/share to the preclinical pipeline and platform and the remainder to expected cash in one year.
- Risks:
  - Developmental pipeline agents face clinical and regulatory development risk, as well as commercial risks. MGNX also faces execution risk and financial risk. We estimate that MGNX current cash will be sufficient to fund operations through the end of 2015, and the company may have additional financing needs before turning cash flow positive

	Valuation ($MM)	Per share
Margetuximab (25% POS)	209	$ 8
MGA271 (12% POS)	212	$ 9
Platform and early pipeline	350	$ 14
Enterprise value ($MM)	770	$ 31
Cash (2014E)	66	$ 3
Total	**836**	**$ 34**
Common shares outstanding 2014E	24.8	

Source: Leerink Swann estimates

MGNX Pipeline and Upcoming Events

Program	Target	Platform	Partner	Indication	Current Status	Next milestone	Timing
Margetuximab	Her2	Fc	proprietary	Breast cancer (Her2 IHC2+)	Phase IIa	Phase IIa data	late 2014
						Initiate Phase IIb/III	2015
				3rd line Gastric cancer	Phase I	Initiate Phase III	2H14
						Phase III data	2018
				Other cancers (bladder)	Phase I	Initiate Phase II	2015
MGA271	B7-H3	Fc, CSLC	Servier (EU rights)	Solid tumors	Phase Ib	Phase Ia DE data	mid-2014
						Servier opt-in	1H14
						Phase Ib expansion data	2H14
						Initiation, Phase II	1Q15
MGD006	CD123 x CD3	DART	Servier (EU rights)	AML	Preclinical	Preclinical data at ASH	4Q13
						IND accepted	1Q14
						Servier opt-in	2014
						Initiate Phase I	1H14
MGD007	gpA33 x CD3	DART, CSLC	Servier (EU rights)	Colorectal cancer	Preclinical	IND accepted	mid-14
						Initiate Phase I	2H14
						Servier opt-in	2015
MGD010	CD32B x CD79B	DART	proprietary	Autoimmune (SLE, RA)	Preclinical	IND prep	2014
						IND accepted	2015
Teplizumab	CD3	Fc	proprietary	Type 1 Diabetes	Investigator-Sponsored Study	Partnership	n/a

Source: SEC Filings, Leerink Swann Estimates

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Fc-optimized monoclonal antibody targeting Her2

MARGETUXIMAB (MGAH22)

Margetuximab – Key Takeaways

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- **Margetuximab is MGNX's lead product candidates, currently in Phase IIa clinical trials**. It is an anti-Her2 monoclonal antibody that has been modified with MGNX's proprietary optimized Fc domain which can significantly increase antibody-dependent cellular cytotoxicity (ADCC). MGNX developed margetuximab with the goal to have an improved, more potent, anti-Her2 treatment than trastuzumab (Herceptin). We believe margetuximab may potentially be effective in a broader patient population than trastuzumab or in patients refractory to trastuzumab. Herceptin is currently approved to treat ~25% of breast and gastric cancer patients over expressing Her2. We believe margetuximab may potentially have utility in a breast cancer patients with only moderate Her2 over expression and in other cancers that express Her2, such as gastroesophageal or bladder cancer.
- **Margetuximab is superior to trastuzumab in preclinical studies.** Based on our checks with MEDACorp specialists, we believe that there is strong evidence that ADCC plays a key role in the activity of trastuzumab. MGNX preclinical data show that margetuximab has the same Her2 binding affinity as trastuzumab while having significantly increased ADCC activity. In addition to that, margetuximab also has strong cell-killing activity in preclinical models representing a genetic polymorphism that is common in 20% of breast cancer patients that do not respond to trastuzumab.
- **Positive clinical data from MGNX's Phase IIa trial in late 2014 is the key de-risking event for margetuximab, we believe.** MGNX is currently enrolling Phase IIa, which will assess margetuximab in metastatic breast cancer patients with only moderate Her2 expression which are currently not eligible for Herceptin-based therapy. In Phase I data presented at ASCO 2013, margetuximab was well tolerated when dosed up to 6mg/kg/week. The drug produced a clinical benefit in patients with Her2 positive metastatic breast cancer, including in patients relapsed from prior Her2-targeted (including Herceptin) therapy. In Phase I most responses were in "hard-to-treat" patients with low affinity Fc-gamma polymorphisms.
- **MGNX is also planning to initiate a pivotal Phase II/III trial in patients with Her2-positive gastroesophageal cancer in 2H14.** Based on feedback from MEDACorp KOLs, we believe the rationale to test margetuximab in an indication known to respond to Herceptin therapy first before pursuing other indications is strong and there is a strong medical need for new gastric cancer therapies in 2nd and 3rd line therapy. In Phase I, MGNX had activity in a patient with metastatic gastric cancer relapsed from prior lines of non-Her2-targeted regimen.

Margetuximab (MGAH22) – Product Profile

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- **Margetuximab is MGNX's lead product candidates, currently in Phase IIa clinical trials**. It is an anti-Her2 monoclonal antibody that has been modified with MGNX's proprietary Fc-domain which can increase antibody-dependent cellular cytotoxicity (ADCC). ADCC is a critical mechanism utilized by many therapeutic antibodies that recruits immune cells, such as macrophages, through their Fcγ receptors (FcγRs), which then kill the targeted cancer cells.
- **MGNX has optimized a specific Fc-region of margetuximab** and thereby improved the cell-killing properties of margetuximab, compared to trastuzumab (Herceptin). MGNX increased binding to activating FcγRs and decreased binding to the inhibitory receptor on immune effector cells.
- **Response to trastuzumab (Herceptin) in metastatic breast cancer (mBC) correlates with expression of the high binding variant (158V) of the activating Fc-gamma receptor IIIA (CD16A).** Margetuximab is a monoclonal antibody with specificity and affinity similar to trastuzumab, with an Fc-domain engineered for increased binding affinity to both alleles of human CD16A, including low binding variants expressed in 80% of patients.
- **Margetuximab has two mechanisms of action**:
  - Enhanced cell killing through ADCC
  - Inhibition of Her2 signaling
- **Current status and development plans**:
  - Positive dose-escalation data from Phase I has been presented at ASCO 2013
  - MGNX is currently conducting a Phase IIa trial in patients with non-amplified moderate level Her2 expressing metastatic breast cancer with data expected in late 2014
  - Depending on Phase IIa results, MGNX plans to also conduct additional exploratory Phase II trials in patients with other HER2 expressing malignancies (e.g., bladder cancer), where Herceptin is not FDA-approved
  - Initiation of a pivotal study in 3rd line Her2-positive metastatic gastric cancer is planned in 2H14

ADCC is a Key Mechanism of Trastuzumab

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- Although the exact mechanisms by which trastuzumab exerts its antitumor activity are unknown, several possibilities have been identified, including inhibition of extracellular domain cleavage, abrogation of intracellular signaling, and antibody-dependent cellular cytotoxicity (ADCC), among others.
- Based on feedback from MEDACorp specialists, we believe ADCC plays a key role in the activity of trastuzumab. Margetuximab in our view captures the validated activities of trastuzumab that relate to Her2 signaling. In addition to that, improved ADCC activity of margetuximab may be able to increase its activity beyond that of trastuzumab. We thus believe margetuximab could be a "biobetter" of trastuzumab and may have efficacy in patients where Herceptin has not been able to show a benefit.
- Trastuzumab contains an IgG1 Fc structure. Both preclinical and pilot clinical studies support a role for trastuzumab engaging Fc receptors on immune effector cells, and ADCC as a key mechanism of action, according to MEDACorp specialists.
- Data from xenograft models showed that trastuzumab exhibit markedly less antitumor activity in mice in which the Fc receptor, which is necessary for ADCC, has been deleted. In addition, tumor growth inhibition is reduced substantially when animals are treated with a modified Her2 antibody incapable of activating Fc receptors on effector cells.
- A study by Anrould et. al. (Br J Cancer 94:259-267, 2006) evaluated the role of different immune cells in the clinical response to trastuzumab. Pre- and postoperative breast tissue samples were obtained from patients (n=23) receiving neoadjuvant trastuzumab plus docetaxel. Trastuzumab-based treatment was associated with significantly increased infiltration of tumors by NK cells and increased lymphocyte activity versus controls. There was approximately four times as much NK cell–related immunohistochemical staining in tumors exposed to trastuzumab than in non-trastuzumab controls.
- A study by Musolino et al. (J Clin Oncol 26:1789-1796, 2008) of 54 patients with HER2-overexpressing breast cancer identified a specific high binding Fc receptor genotype (FcRIIIa-158 valine/valine) that was significantly associated with an improved objective response rate (ORR) and improved progression-free survival (PFS).

ADCC is Important for Trastuzumab Efficacy

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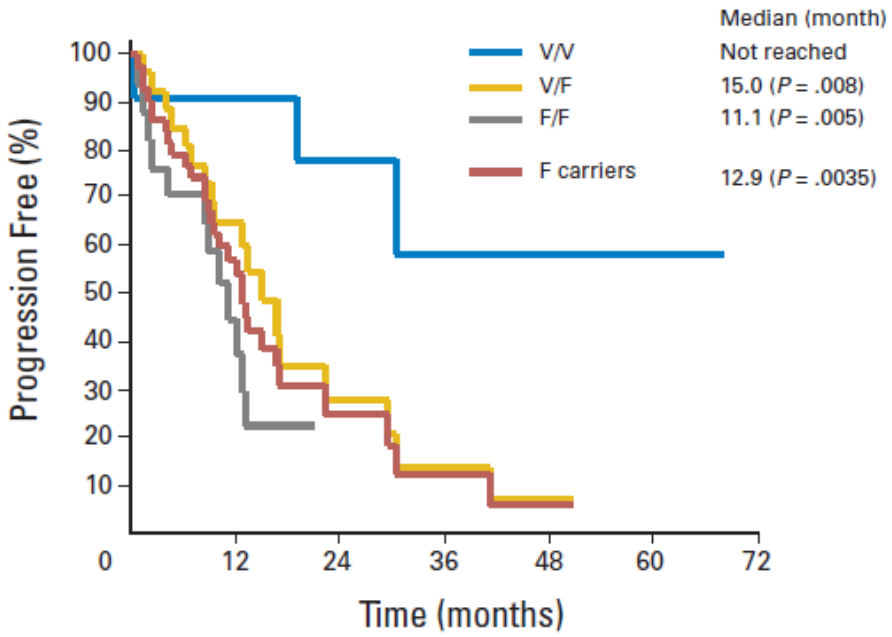
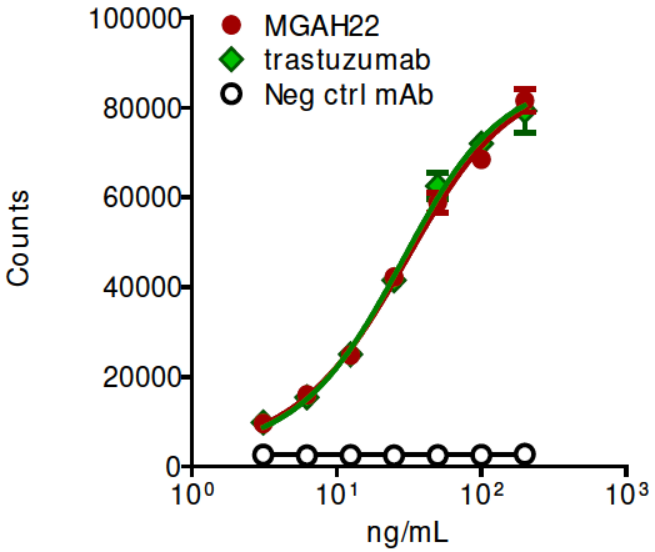
- Gene association analysis from *Musolino et al*. shows improved outcomes in metastatic breast cancer patients who have a higher binding form of the activating Fcγ receptor (RIIIa-158 V/V genotype), in response to treatment with chemotherapy plus trastuzumab
- The FcγRIIIa 158 V/V polymorphism (occurs in 20% of patients) has a naturally occurring high affinity to IgG1 indicating high ADCC activity
- Margetuximab's optimized Fc region binds with high affinity to all FcγRIIIa receptors
- MGNX introduced five amino acid substitutions in the IgG1 Fc domain
- Margetuximab recognizes the same Her2 epitope as Herceptin



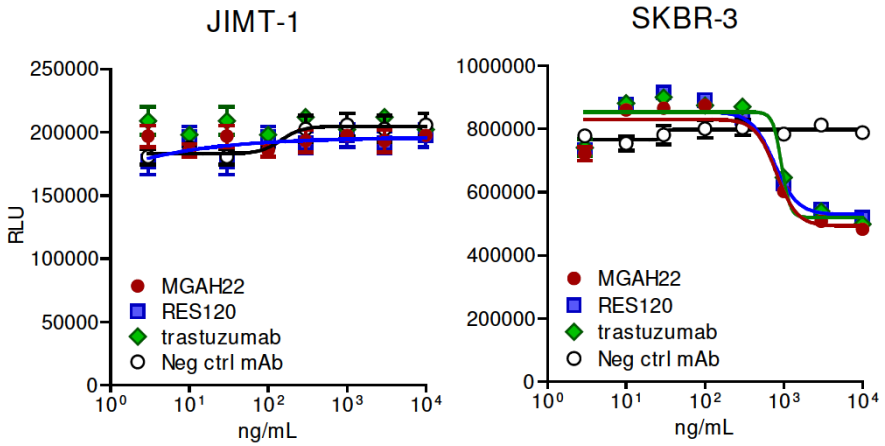
Source: Musolino et al., JCO 2008

Margetuximab In-Vitro Data Shows Same Her2 Binding and anti-proliferative activity as Trastuzumab (without ADCC)

HER2 binding activity of MGAH22 was compared to trastuzumab by antigen capture ELISA



Proliferation of breast cancer cells (JIMT-1 or SKBR-3) in the presence of MGAH22 or trastuzumab



Source: Nordstrom JL, et al., Breast Cancer Research 13:R123, 2011

Margetuximab is More Effective in Inducing ADCC

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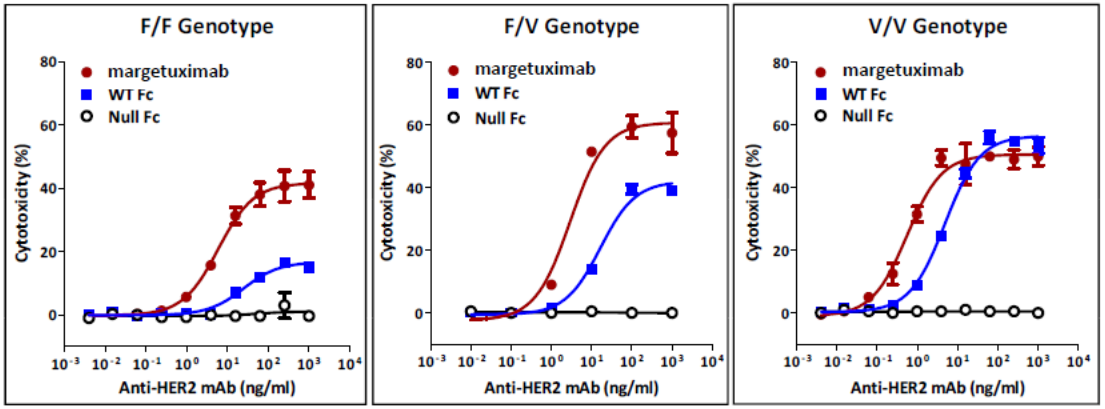
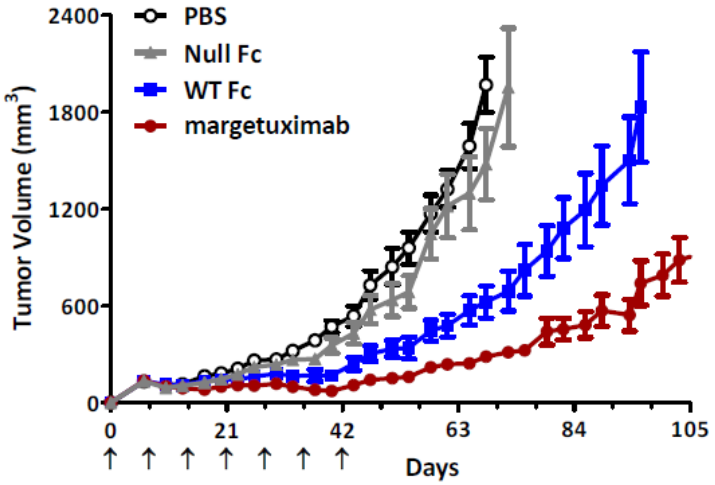
In vitro ADCC activity*



Tumor control depends on ADCC



- Xenograft: JIMT-1 human breast cancer cell line (HER2 2+, amplified, PI3K mutant)
- Mice: mCD16-/- hCD16A 158F transgenic
- Anti-HER2 mAbs: Administered IV at 2mg/kg

**WT Fc = trastuzumab*

Source: Nordstrom JL, et al., Breast Cancer Research 13:R123, 2011

Margetuximab – Phase I Clinical Trial

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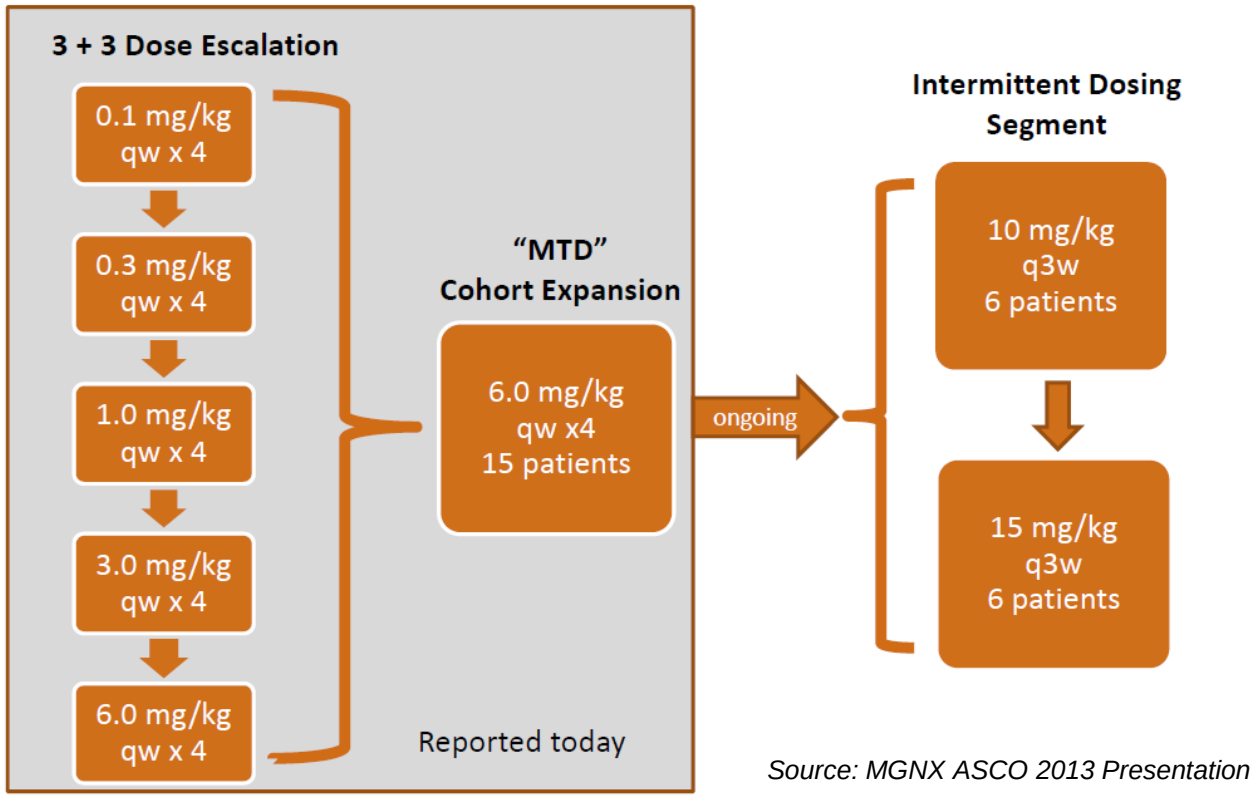
- The margetuximab Phase I clinical trial was an open-label, multi-dose, single-arm, dose-escalation study conducted to define the safety profile and pharmacokinetics, or PK, of margetuximab and to begin to explore the antitumor activity of margetuximab in patients with refractory HER2+ tumors.
- MGNX enrolled a total of 34 patients in the dose escalation (0.1 to 6.0 mg/kg) and expansion (6.0 mg/kg) phases of the trial. This patient population was heavily pre-treated with prior therapies, including 19 patients with other prior anti-HER2 therapies. In the absence of dose limiting toxicity, an additional cohort of patients was treated at the top dose.



Source: MGNX ASCO 2013 Presentation

Phase I Baseline Patient Characteristics

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- 34 patients
- Relapsed or refractory metastatic Her2 positive cancers (2+ or 3+ by IHC); any histology
- 19 patients had prior Her2 directed therapy

Number of patients enrolled	34
Male/Female	15/19
Age median (range)	63 (36 – 83)
Tumor Type	
Gastroesophageal	12 (35%)
Breast	10 (29%)
Colorectal	5 (15%)
Lung	2 (6%)
Ampulla of Vater	1 (3%)
Bladder	1 (3%)
Endometrium	1 (3%)
Esophageal – squamous	1 (3%)
Salivary gland	1 (3%)
HER2 Status (Central Laboratory)	
IHC	
ND	1 (3%)
<2+	6 (18%)
2+	12 (35%)
3+	15 (44%)
FISH	
ND	1 (3%)
Non-amplified	17 (50%)
Amplified	16 (47%)
Number of Prior ChemorRx Regimens	3 (1 – 7)
Prior Anti-HER2 Therapy	19 (56%)
trastuzumab	15 (44 %)
lapatinib	13 (38 %)
other	5 (15%)

Source: MGNX ASCO 2013 Presentation

Phase I Data Promising, In Our View

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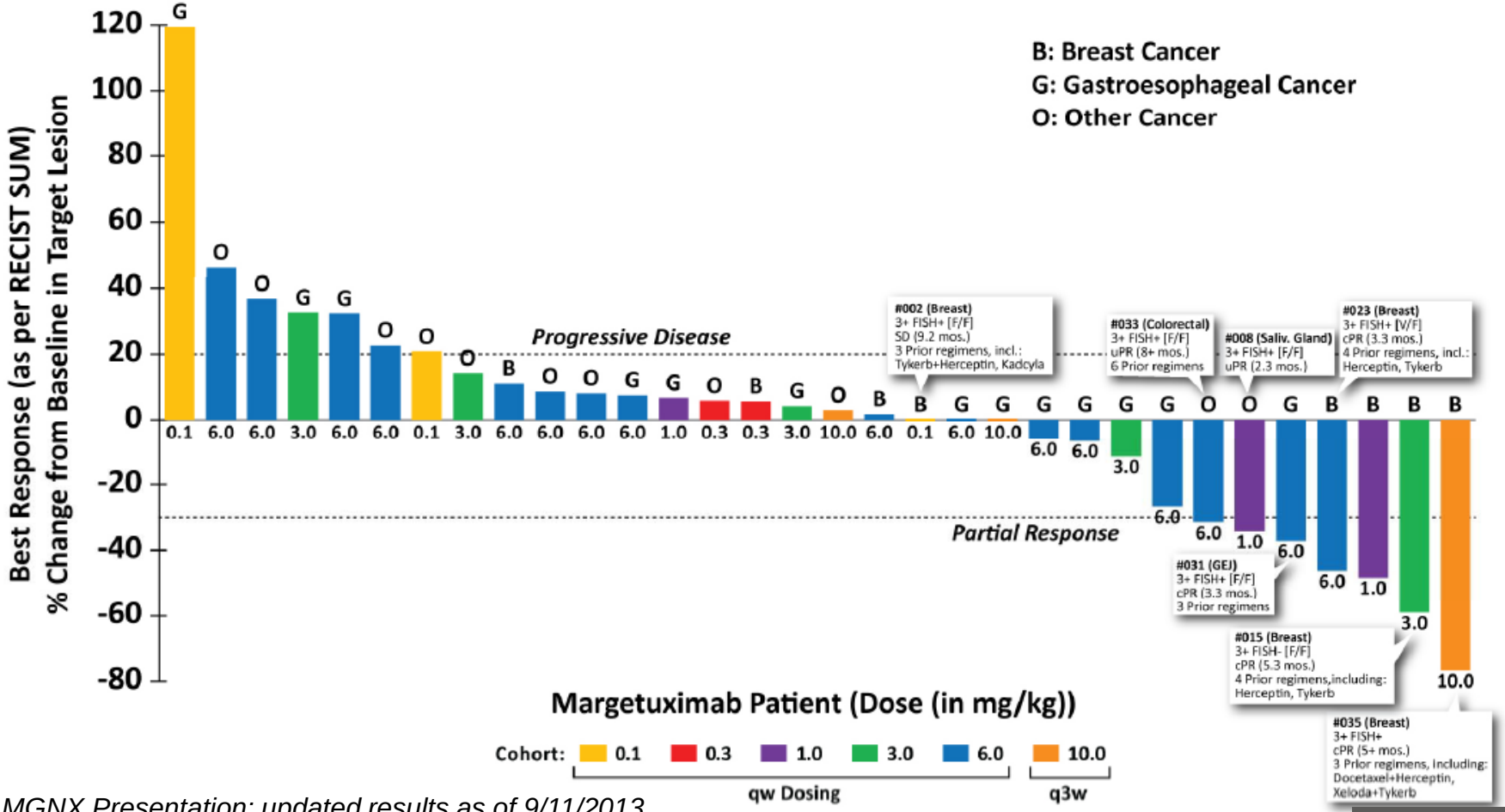
- During the dose escalation and expansion segments of the Phase I clinical trial, a dose of 6.0 mg/kg has been well-tolerated in patients with refractory HER2+ tumors who were treated weekly for four weeks.
- Approximately one-third of patients received additional cycles of margetuximab treatment.
- Using margetuximab as a single agent, tumor response was observed even in patients who had failed prior therapies including other anti-HER2 treatment.



Source: MGNX Presentation; updated results as of 9/11/2013

Phase I Data Promising, In Our View

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- **34 patients treated in total across all doses***
  - 12 gastric cancer patients: Response Rate: 1/12 = 8%
  - 10 breast cancer patients: Response Rate: 3/10 = 30%
  - 12 other cancers patients: Response Rate: 2/12 =17%
- **24 patients treated at doses >/= 1mg/kg***
  - 11 gastric cancer patients: Response Rate: 1/11 = 9%
  - 8 breast cancer patients: Response Rate: 3/8 = 38%
  - 10 other cancers patients: Response Rate: 2/10 = 20%

Patient (Tumor Type)	HER2 IHC/FISH	CD16A genotype	Marge-tuximab Dose Cohort	Regimen Number	Treatment	Treatments Duration (cycles)	Treatment Duration (approx. mos)	Best Overall Response
002 (Breast)	3+ Pos 4.9X	F/F	0.1 mg/kg	1	docetaxel + lapatinib + trastuzumab	6	4 (36 t)	SD
				2	mTOR inhibitor	?	6	SD
				3	T-DM1	17	12	SD
				4	margetuximab	9	9.2	SD
008 (salivary gland)	3+ Pos 6.7X	F/F	1.0 mg/kg	1	Tegafur	?	4	NA
				2	margetuximab	2	2.3	uPR
015 (Breast)	3+ Neg 1.2X	F/F	3.0 mg/kg	1	docetaxel + trastuzumab (neoadjuvant)	4	3	NA
				2	FEC + trastuzumab (adjuvant)	3 (17t)	2 (9t)	-
				3	paclitaxel + trastuzumab	16	11	PR
				4	capecitabine+lapatinib	8	6	NA
				5	margetuximab	5	5.3	cPR
023 (Breast)	3+ Pos 2.7X	V/F	6.0 mg/kg	1	Adriamycin-Docetaxel (neoadjuvant)	3	2	SD
				2	Docetaxel-Herceptin	21	14	PR
				3	Xeloda-Lapatinib	12	7	PR
				4	Pan-HER TKI	8	6	PR
				5	margetuximab	3	3.3	cPR
031 (GEJ*)	3+ Pos 2.3X	F/F	6.0 mg/kg	1	Taxol – carboplatin (neoadjuvant)	UNK	2	SD
				2	5-FU-leucovorin (adjuvant)	4	3	NA
				3	FOLFOX-tivantinib	4	2	PD
				4	margetuximab	3	3.3	cPR

Source: MGNX ASCO 2013 Presentation

**Updated results as of 9/11/2013*

Margetuximab Breast Cancer Phase IIa Data in Late 2014 is the Key Derisking Event, in Our View

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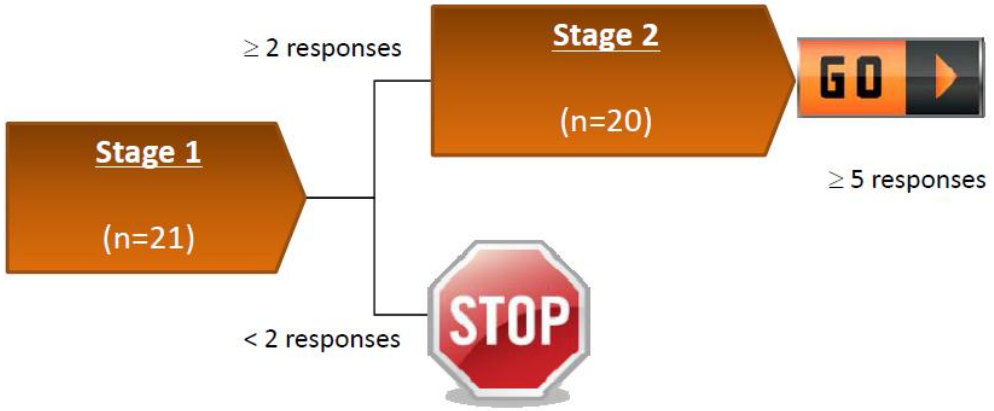
- **We believe positive data from MGNX's margetuximab Phase IIa trial is the key de-risking event for this program which if positive would significantly increase its probability of success of margetuximab, in our view**. Based on mechanistic, preclinical, and Phase I data, we believe there is a reasonable chance of success for positive Phase IIa data. We note, that patients in Phase I were not selected for moderate (IHC2+) Her2 expression, so the ultimate proof-of-concept for margetuximab will be the Phase IIa data, expected in late 2014, according to management. Although it is clear that patients with breast tumors overexpressing HER2 respond best to trastuzumab therapy, the benefits of treatment may extend to patients with lower level HER2 expression according to MEDACorp KOLs we spoke to. A retrospective analysis of samples from the NSABP B-31 trial showed a significant DFS benefit with trastuzumab in tumors that showed a normal HER2 gene copy number or were negative by FISH with an IHC score less than 3+ after HER2 retesting.
- **MGNX is currently enrolling the Phase IIa trial** to determine if margetuximab has activity in patients with metastatic breast cancer who are not candidates for trastuzumab therapy. For that purpose, MGNX are enrolling patients with metastatic breast cancer with moderate HER2 expression (2+ level by IHC) and lack evidence of HER2 gene amplification by FISH.
- **Margetuximab will be administered as a 6 mg/kg** intravenous (IV), weekly on Days 1, 8, and 15 of each 28-day cycle. If fewer than two partial or complete responses (PRs, CRs) are observed in the first 21 patients evaluable for response at the first tumor re-evaluation on day 22 of cycle 2, no additional patients will be enrolled and the trial will end. If two or more responses are observed MGNX will expand the clinical trial to include a total of 41 patients evaluable for response. MGNX considers five or more PRs or CRs in these 41 patients as adequate.



Patient Population:	Relapsed or refractory advanced breast cancer patients
HER2 Positivity:	IHC 2+, non-amplified

Source: MGNX ASCO 2013 Presentation

Pivotal Trial in Her2+ Gastric Cancer to Start in 2H14

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- **MacroGenics expects to initiate a Phase III trial in HER2+ gastroesophageal cancer in 2H14.** The trial is expected to be a randomized study to evaluate the addition of margetuximab to standard chemotherapy (irinotecan or paclitaxel) in the 3rd line treatment of patients with advanced gastroesophageal cancers which have progressed after standard frontline and 2nd line treatment of advanced disease. The primary analysis will compare the overall survival (OS) of patients randomized to chemotherapy plus placebo to the overall survival of patients randomized to chemotherapy plus margetuximab. Mgmt expects to enroll the trial within three years (2H17).
- **We believe that there is a high unmet medical need for efficacious second- and third line treatment regimens for HER2+ gastric and GEJ cancers.** No 2nd or 3rd line treatments are currently FDA-approved for gastric cancer. Paclitaxel or irinotecan are commonly used for that purpose following relapse from first line Herceptin and/or cisplatin plus capecitabine or 5-FU based regimen. Primary and secondary (acquired) resistance to trastuzumab has become a major problem according to MEDACorp specialists and new targeted agents to overcome this resistance are needed.
- **Based on feedback from MEDACorp KOLs, we believe the rationale is sound to test Margetuximab in an indication known to respond to Herceptin therapy first before pursuing other indications that over express Her2, such as bladder cancer.** MGNX is currently planning a pivotal trial in 3rd line metastatic GEJ patients which would likely position Margetuximab after a potential future standard of care involving Kadcyla in 2nd line following relapse from Herceptin-based 1st line therapy, depending on data from the Roche GASBY trial. Recall, Kadcyla has a different mechanism from Herceptin and margetuximab that is independent of ADCC.
- **We believe breast cancer data (e.g., EMILIA trial) clearly shows that targeting Her2 benefits in trastuzumab-resistant patients.** Thus MEDACorp KOLs believe that it is likely that targeting Her2 third-line with a differentiated targeting agent could be beneficial in certain patients, especially those with low-affinity binding Fc-receptor (CD16A) mutations. Herceptin works best in patients that are Her2 FISH-positive. Thus MEDACorp specialists reasoned that Herceptin resistance in those patients is likely harder to treat than e.g., patients that are IHC+, but FISH-negative given that the IHC test is more variable and in these patients Herceptin may not work as well if the target is expressed at lower levels.

Design for Margetuximab Gastric Cancer Trial

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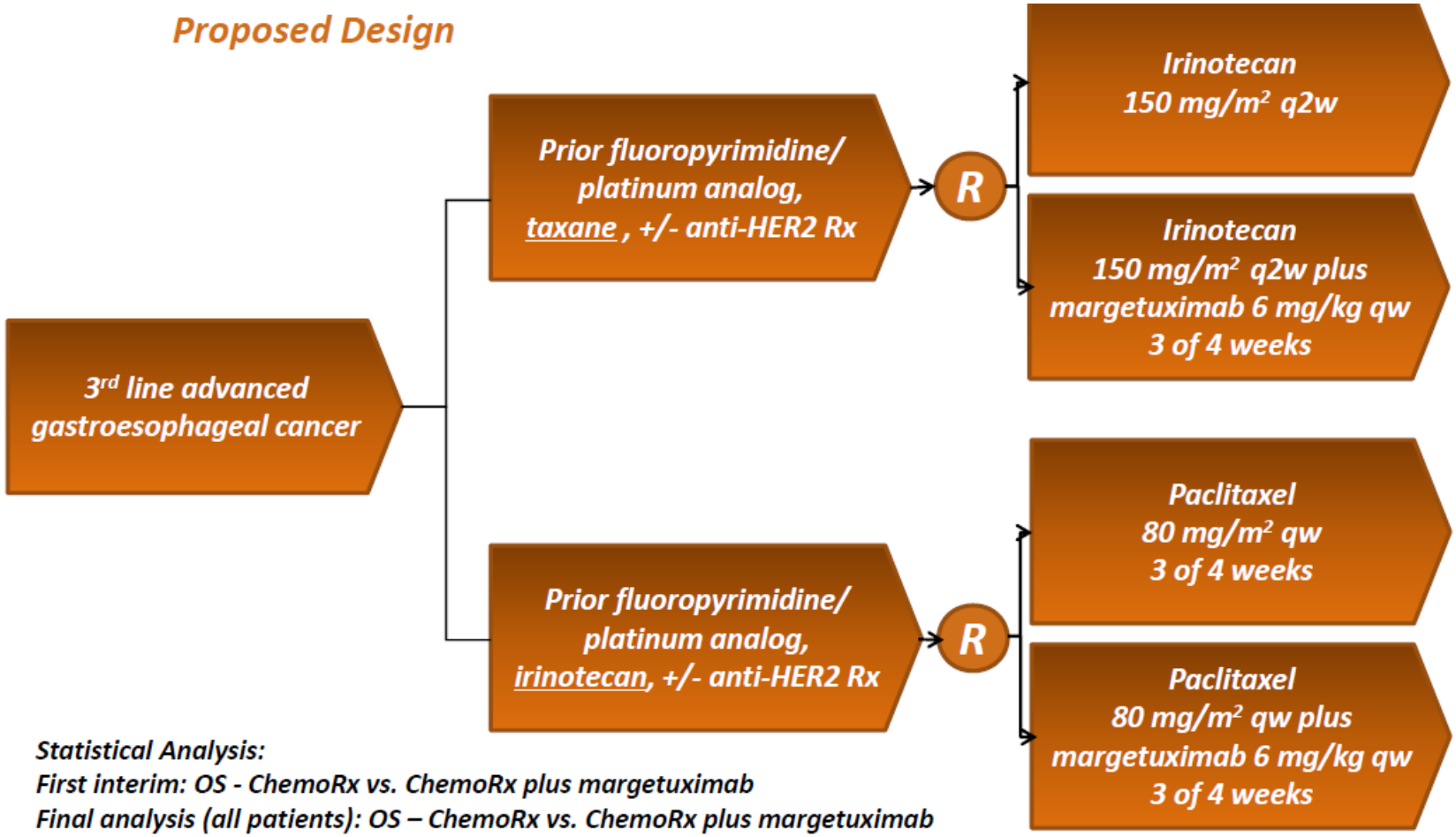

Statistical Analysis:
First interim: OS - ChemoRx vs. ChemoRx plus margetuximab
Final analysis (all patients): OS – ChemoRx vs. ChemoRx plus margetuximab

Source: MGNX ASCO 2013 presentation

Herceptin + Chemotherapy is Standard of Care for 1st Line Her2+ Gastric Cancer

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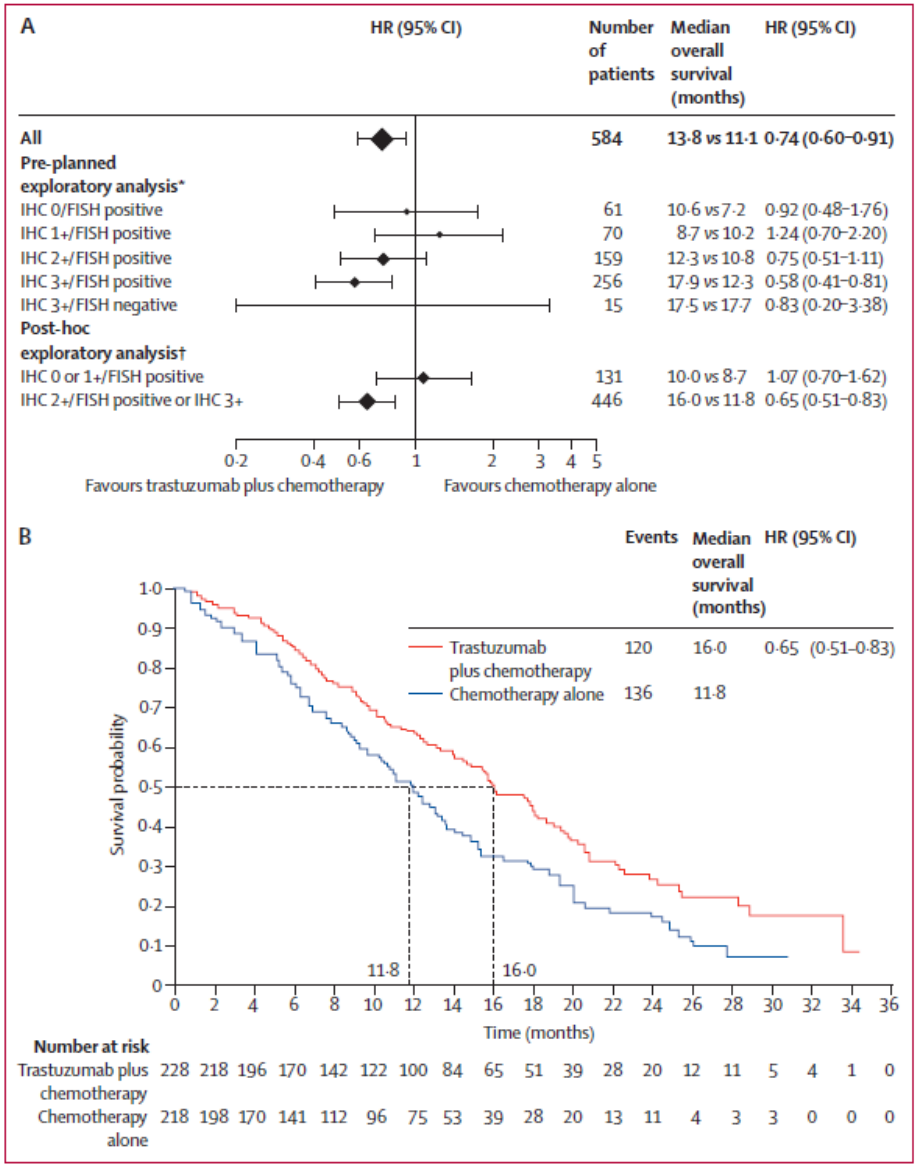
- **In October 2011, Herceptin was FDA-approved for first-line treatment of HER2-positive metastatic gastric cancer** based on positive data from Roche's ToGA trial. ToGA enrolled 594 patients with locally advanced or metastatic, gastric cancer who were randomized to receive Herceptin plus chemotherapy (cisplatin + capecitabine or 5-FU) or chemotherapy alone. Patients were eligible if their tumor samples were scored as 3+ on immunohistochemistry (IHC) or if they were FISH-positive (any IHC).
- **Herceptin plus chemotherapy improved overall survival by 37% compared to chemotherapy alone** (based on HR=0.74, 95 percent CI 0.60-0.91, p=0.0046, median OS 13.8 vs. 11.1 months).
- **Patients with tumors exhibiting high levels of HER2 (IHC 2+/FISH positive or IHC 3+, 16% of patients tested in the ToGA study) experienced a greater benefit from the addition of Herceptin**. For these patients, overall survival in the study was 16.0 months on average versus 11.8 months for patients receiving chemotherapy alone. All patients in ToGA had their tumors tested for HER2 status using two companion diagnostics. Based on HER2 screening results using both HER2 IHC3+ or a FISH-positive diagnostic test in ToGA, approximately 22% of patients with metastatic stomach cancer have HER2-positive tumors.



Source: The Lancet

Her2+ Gastric Cancer Pipeline is Limited

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Drug	MoA	Company	Patients	Status	Comments
Lapatinib (Tykerb)	EGFR, HER2 TKI	GSK	1st line w/ capecitabine + oxaliplatin	Phase III	Missed primary endpoint (OS HR=0.91; 95% CI: 0.73, 1.12; p=0.3492)
T-DM1 (Kadcyla)	Her2 ADC	Roche	2nd, 3rd, 4th line single agent vs. a taxane	Phase III	Exp BLA 2015
Pertuzumab (Perjeta)	Her2 mAb	Roche	1st, 2nd, 3rd line, w/ trastuzumab and chemo	Phase III	Exp BLA 2016/17
Margetuximab	Her2 mAb	MGNX	3rd line	Phase II/III	To be initiated in 2H14
M-111	Her2, Her3 bispecific	MACK	2nd line w/ paclitaxel, +/- trastuzumab	Phase II	Initiated 06/05/2013
Neratinib	EGFR, Her2, Her4 TKI	PBYI	pts w/ Her2 activating mutations	Phase II	Initiated October 2013

Source; Leerink Swann Research, Company filings, Clinicaltrials.gov

- **Lapatinib (Tykerb) did not achieve a significant improvement in overall survival in metastatic gastric cancer**. We believe neratinib (PBYI) prospects are better, but the development strategy in gastric cancer is not yet clear. Based on MEDACorp breast cancer KOL feedback, we believe neratinib has the potential to be the best-in-class TKI against HER2. Data from GSK's LOGiC trial presented at ASCO 2013 showed that lapatinib in combination with chemotherapy (oxaliplatin + capecitabine) in patients with previously untreated HER2-positive advanced gastric cancer did not meet the primary endpoint of improved overall survival (OS) compared to chemotherapy alone. The median OS for patients in the lapatinib plus chemotherapy group was 12.2 months compared to 10.5 months for patients randomized to placebo plus chemotherapy (HR=0.91; 95% CI: 0.73, 1.12; p=0.3492). Median PFS lapatinib + chemotherapy was 6.0 months vs. 5.4 months in the control group. Response rate was 53% for patients in the lapatinib plus chemo arm, and 39% for those in the control group. Another Phase III trial in the second line setting in combination with paclitaxel produced only a HR of 0.84 (CI: 0.64 to 1.11; p=0.2088). PFS was 5.4 vs. 4.4 months.
- **Roche is currently enrolling the GATSBY trial testing T-DM1 single agent vs. taxane in previously treated advanced HER2+ gastric cancer.** Given Kadcyla's strong efficacy in metastatic Her2 + breast cancer, we believe it is likely that it will also be efficacious in gastric cancer patients. This Phase II/III trial is comparing Kadcyla 3.6mg/kg q3w vs. Kadcyla 2.4mg/kg weekly vs. Docetaxel or paclitaxel in the first stage. At the end of the first stage, the dose and schedule of Kadcyla that will be used in the second stage of the study will be selected. The regimen selection analysis will be made after approximately 100 patients across all three study arms have been treated for at least 4 cycles (12 weeks). In Stage II of the study, additional patients will be recruited and randomized to either the selected regimen of trastuzumab emtansine or to the standard taxane therapy. Roche started dosing in 3Q12 and expects to file a BLA in 2015.
- **Pertuzumab (Perjeta) is currently in a Phase III trial in combination with Herceptin (trastuzumab),** fluoropyrimidine and cisplatin as first-line treatment in patients with HER2-positive metastatic gastroesophageal junction or gastric cancer. This trial started enrolling in 2Q13 and Roche currently expects to submit the BLA in 2016/17, assuming the trial is positive.

Margetuximab Revenue Patient Build, mGC

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US metastatic Gastric Cancer	2012E	2013E	2014E	2015E	2016E	2017E	2018E	2019E	2020E	2021E	2022E	2023E	2024E	2025E	2026E	2027E	2028E	2029E	2030E
Advanced stage incidence	21,400	21,767	22,141	22,521	22,907	23,300	23,700	24,106	24,520	24,941	25,369	25,804	26,247	26,697	27,155	27,621	28,095	28,577	29,067
% growth		*1.7%*	1.7%	1.7%	1.7%	1.7%	1.7%	1.7%	1.7%	1.7%	1.7%	1.7%	1.7%	1.7%	1.7%	1.7%	1.7%	1.7%	1.7%
HER2 tested	16,200	16,478	16,761	17,048	17,341	17,700	18,004	18,313	18,627	18,946	19,271	19,602	19,938	20,281	20,629	20,983	21,343	21,709	22,081
% of Stage IV	*76%*	*76%*	*76%*	*76%*	*76%*	*76%*	*76%*	*76%*	*76%*	*76%*	*76%*	*76%*	*76%*	*76%*	*76%*	*76%*	*76%*	*76%*	*76%*
HER2+	4,400	4,492	4,586	4,682	4,779	4,900	4,984	5,070	5,157	5,245	5,335	5,427	5,520	5,614	5,711	5,809	5,908	6,010	6,113
% of tested	*27%*	*27%*	*27%*	*27%*	*28%*	*28%*	*28%*	*28%*	*28%*	*28%*	*28%*	*28%*	*28%*	*28%*	*28%*	*28%*	*28%*	*28%*	*28%*
1st line mGC therapy	4,400	4,492	4,586	4,682	4,779	4,900	4,984	5,070	5,157	5,245	5,335	5,427	5,520	5,614	5,711	5,809	5,908	6,010	6,113
2nd line mGC therapy	3,520	3,594	3,669	3,745	3,823	3,920	3,987	4,056	4,125	4,196	4,268	4,341	4,416	4,492	4,569	4,647	4,727	4,808	4,890
% progressed from 1st line	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*
3rd line mGC therapy	2,816	2,875	2,935	2,996	3,059	3,136	3,190	3,245	3,300	3,357	3,414	3,473	3,533	3,593	3,655	3,718	3,781	3,846	3,912
% progressed from 2nd line	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*
Margetuximab patients								**811**	**1,650**	**2,685**	**2,732**	**2,778**	**2,826**	**2,875**	**2,924**	**2,974**	**3,025**	**3,077**	**3,130**
% of 3rd line patients								*25%*	*50%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*

Source: Leerink Swann Estimates

EU metastatic Gastric Cancer	2012E	2013E	2014E	2015E	2016E	2017E	2018E	2019E	2020E	2021E	2022E	2023E	2024E	2025E	2026E	2027E	2028E	2029E	2030E
Advanced stage incidence	37,900	38,019	38,139	38,259	38,379	38,500	38,621	38,743	38,865	38,987	39,109	39,233	39,356	39,480	39,604	39,729	39,854	39,979	40,105
% growth		*0.3%*	0.3%	0.3%	0.3%	0.3%	0.3%	0.3%	0.3%	0.3%	0.3%	0.3%	0.3%	0.3%	0.3%	0.3%	0.3%	0.3%	0.3%
HER2 tested	27,200	29,187	31,185	33,196	35,220	34,700	34,809	34,919	35,029	35,139	35,249	35,360	35,472	35,583	35,695	35,807	35,920	36,033	36,146
% of Stage IV	*72%*	*77%*	*82%*	*87%*	*92%*	*90%*	*90%*	*90%*	*90%*	*90%*	*90%*	*90%*	*90%*	*90%*	*90%*	*90%*	*90%*	*90%*	*90%*
HER2+	3,500	3,756	4,013	4,272	4,532	4,400	4,501	4,602	4,704	4,807	4,910	5,014	5,119	5,224	5,329	5,436	5,543	5,650	5,758
% of tested	*13%*	*13%*	*13%*	*13%*	*13%*	*13%*	*13%*	*13%*	*13%*	*14%*	*14%*	*14%*	*14%*	*15%*	*15%*	*15%*	*15%*	*16%*	*16%*
1st line mGC therapy	3,500	3,756	4,013	4,272	4,532	4,400	4,501	4,602	4,704	4,807	4,910	5,014	5,119	5,224	5,329	5,436	5,543	5,650	5,758
2nd line mGC therapy	2,800	3,004	3,210	3,417	3,626	3,520	3,601	3,682	3,764	3,846	3,928	4,011	4,095	4,179	4,263	4,348	4,434	4,520	4,607
% progressed from 1st line	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*
3rd line mGC therapy	2,240	2,404	2,568	2,734	2,900	2,816	2,881	2,945	3,011	3,076	3,143	3,209	3,276	3,343	3,411	3,479	3,547	3,616	3,685
% progressed from 2nd line	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*
Margetuximab patients								**295**	**1,505**	**2,461**	**2,514**	**2,567**	**2,621**	**2,675**	**2,729**	**2,783**	**2,838**	**2,893**	**2,948**
% of 3rd line patients								*10%*	*50%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*	*80%*

Source: Leerink Swann Estimates

Margetuximab US Patient Build, Her2 (IHC 2+, FISH) mBC

LEERINK SWANN

US metastatic Breast Cancer	2012E	2013E	2014E	2015E	2016E	2017E	2018E	2019E	2020E	2021E	2022E	2023E	2024E	2025E	2026E	2027E	2028E	2029E	2030E
Stage IV incidence (De Novo)	12,000	12,070	12,140	12,210	12,281	12,352	12,424	12,496	12,568	12,641	12,715	12,788	12,862	12,937	13,012	13,088	13,164	13,240	13,317
% growth		*0.6%*	*0.6%*	*0.6%*	*0.6%*	*0.6%*	*0.6%*	*0.6%*	*0.6%*	*0.6%*	*0.6%*	*0.6%*	*0.6%*	*0.6%*	*0.6%*	*0.6%*	*0.6%*	*0.6%*	*0.6%*
HER2 tested	11,300	11,366	11,431	11,498	11,564	11,632	11,699	11,767	11,835	11,904	11,973	12,042	12,112	12,182	12,253	12,324	12,396	12,468	12,540
% of Stage IV	*94%*	*94%*	*94%*	*94%*	*94%*	*94%*	*94%*	*94%*	*94%*	*94%*	*94%*	*94%*	*94%*	*94%*	*94%*	*94%*	*94%*	*94%*	*94%*
HER2 2+ non-amplified	859	864	869	874	879	884	889	894	899	905	910	915	921	926	931	937	942	948	953
% of tested	*7.6%*	*7.6%*	*7.6%*	*7.6%*	*7.6%*	*7.6%*	*7.6%*	*7.6%*	*7.6%*	*7.6%*	*7.6%*	*7.6%*	*7.6%*	*7.6%*	*7.6%*	*7.6%*	*7.6%*	*7.6%*	*7.6%*
Adjuvant relapse	2,126	2,138	2,151	2,163	2,176	2,188	2,201	2,214	2,227	2,227	2,227	2,227	2,227	2,227	2,227	2,227	2,227	2,227	2,227
% of adjuvant treated	*15%*	*15%*	*15%*	*15%*	*15%*	*15%*	*15%*	*15%*	*15%*	*15%*	*15%*	*15%*	*15%*	*15%*	*15%*	*15%*	*15%*	*15%*	*15%*
1st line mBC therapy	2,985	3,002	3,020	3,037	3,055	3,072	3,090	3,108	3,126	3,131	3,137	3,142	3,147	3,153	3,158	3,163	3,169	3,174	3,180
Margetuximab patients (1st line mBC)								**155**	**782**	**1,566**	**1,882**	**1,885**	**1,888**	**1,892**	**1,895**	**1,898**	**1,901**	**1,905**	**1,908**
% of 1st line patients								*5%*	*25%*	*50%*	*60%*	*60%*	*60%*	*60%*	*60%*	*60%*	*60%*	*60%*	*60%*
2nd line mBC therapy	2,298	2,312	2,325	2,339	2,352	2,366	2,380	2,393	2,407	2,411	2,415	2,419	2,423	2,428	2,432	2,436	2,440	2,444	2,448
% progressed from 1st line	*77%*	*77%*	*77%*	*77%*	*77%*	*77%*	*77%*	*77%*	*77%*	*77%*	*77%*	*77%*	*77%*	*77%*	*77%*	*77%*	*77%*	*77%*	*77%*
2nd line pts progressed from 1st line Margetuximab								120	602	1,206	1,449	1,452	1,454	1,457	1,459	1,462	1,464	1,467	1,469
Margetuximab patients (2nd line mBC)								**114**	**451**	**904**	**725**	**726**	**727**	**728**	**730**	**731**	**732**	**733**	**735**
% of 2nd line patients								*5%*	*25%*	*75%*	*75%*	*75%*	*75%*	*75%*	*75%*	*75%*	*75%*	*75%*	*75%*
3rd line mBC therapy	1,724	1,734	1,744	1,754	1,764	1,774	1,785	1,795	1,805	1,808	1,811	1,815	1,818	1,821	1,824	1,827	1,830	1,833	1,836
% progressed from 2nd line	*75%*	*75%*	*75%*	*75%*	*75%*	*75%*	*75%*	*75%*	*75%*	*75%*	*75%*	*75%*	*75%*	*75%*	*75%*	*75%*	*75%*	*75%*	*75%*
3rd line pts progressed from 1st or 2nd line Margetuximab								175	790	1,582	1,630	1,633	1,636	1,639	1,641	1,644	1,647	1,650	1,653
Margetuximab patients (3rd line mBC)								**162**	**254**	**203**	**163**	**163**	**164**	**164**	**164**	**164**	**165**	**165**	**165**
% of 3rd line patients								*10%*	*25%*	*90%*	*90%*	*90%*	*90%*	*90%*	*90%*	*90%*	*90%*	*90%*	*90%*

Source: Leerink Swann Estimates

Margetuximab EU Patient Build, Her2 (IHC 2+, FISH) mBC

LEERINK SWANN

EU metastatic Breast Cancer	2012E	2013E	2014E	2015E	2016E	2017E	2018E	2019E	2020E	2021E	2022E	2023E	2024E	2025E	2026E	2027E	2028E	2029E	2030E
Stage IV incidence (De Novo)	12,000	12,070	12,140	12,210	12,281	12,352	12,424	12,496	12,568	12,641	12,715	12,788	12,862	12,937	13,012	13,088	13,164	13,240	13,317
% growth		*0.6%*	*0.6%*	*0.6%*	*0.6%*	*0.6%*	*0.6%*	*0.6%*	*0.6%*	*0.6%*	*0.6%*	*0.6%*	*0.6%*	*0.6%*	*0.6%*	*0.6%*	*0.6%*	*0.6%*	*0.6%*
HER2 tested	11,500	11,567	11,634	11,701	11,769	11,837	11,906	11,975	12,045	12,115	12,185	12,255	12,327	12,398	12,470	12,542	12,615	12,688	12,762
% of Stage IV	*96%*	*96%*	*96%*	*96%*	*96%*	*96%*	*96%*	*96%*	*96%*	*96%*	*96%*	*96%*	*96%*	*96%*	*96%*	*96%*	*96%*	*96%*	*96%*
HER2 2+ non-amplified	874	879	884	889	894	900	905	910	915	921	926	931	937	942	948	953	959	964	970
% of tested	*7.6%*	*7.6%*	*7.6%*	*7.6%*	*7.6%*	*7.6%*	*7.6%*	*7.6%*	*7.6%*	*7.6%*	*7.6%*	*7.6%*	*7.6%*	*7.6%*	*7.6%*	*7.6%*	*7.6%*	*7.6%*	*7.6%*
Adjuvant relapse	3,285	3,304	3,323	3,343	3,362	3,381	3,401	3,421	3,441	3,441	3,441	3,441	3,441	3,441	3,441	3,441	3,441	3,441	3,441
% of adjuvant treated	*19%*	*19%*	*19%*	*19%*	*19%*	*19%*	*19%*	*19%*	*19%*	*19%*	*19%*	*19%*	*19%*	*19%*	*19%*	*19%*	*19%*	*19%*	*19%*
1st line mBC therapy	4,159	4,183	4,207	4,232	4,256	4,281	4,306	4,331	4,356	4,361	4,367	4,372	4,378	4,383	4,388	4,394	4,399	4,405	4,411
Margetuximab patients (1st line mBC)									**218**	**1,090**	**2,183**	**2,623**	**2,627**	**2,630**	**2,633**	**2,636**	**2,640**	**2,643**	**2,646**
% of 1st line patients									*5%*	*25%*	*50%*	*60%*	*60%*	*60%*	*60%*	*60%*	*60%*	*60%*	*60%*
2nd line mBC therapy	2,995	3,012	3,029	3,047	3,065	3,082	3,100	3,118	3,136	3,140	3,144	3,148	3,152	3,156	3,160	3,164	3,168	3,172	3,176
% progressed from 1st line	*72%*	*72%*	*72%*	*72%*	*72%*	*72%*	*72%*	*72%*	*72%*	*72%*	*72%*	*72%*	*72%*	*72%*	*72%*	*72%*	*72%*	*72%*	*72%*
2nd line pts progressed from 1st line Margetuximab					-	-	-	-	157	785	1,572	1,889	1,891	1,893	1,896	1,898	1,901	1,903	1,905
Margetuximab patients (2nd line mBC)									**149**	**589**	**1,179**	**944**	**946**	**947**	**948**	**949**	**950**	**951**	**953**
% of 2nd line patients									*5%*	*25%*	*75%*	*75%*	*75%*	*75%*	*75%*	*75%*	*75%*	*75%*	*75%*
3rd line mBC therapy	1,377	1,385	1,394	1,402	1,410	1,418	1,426	1,434	1,443	1,444	1,446	1,448	1,450	1,452	1,453	1,455	1,457	1,459	1,461
% progressed from 2nd line	*46%*	*46%*	*46%*	*46%*	*46%*	*46%*	*46%*	*46%*	*46%*	*46%*	*46%*	*46%*	*46%*	*46%*	*46%*	*46%*	*46%*	*46%*	*46%*
3rd line pts progressed from 1st or 2nd line Margetuximab									141	632	1,265	1,303	1,305	1,306	1,308	1,310	1,311	1,313	1,315
Margetuximab patients (3rd line mBC)									**130**	**203**	**163**	**130**	**130**	**131**	**131**	**131**	**131**	**131**	**131**
% of 3rd line patients									*10%*	*25%*	*90%*	*90%*	*90%*	*90%*	*90%*	*90%*	*90%*	*90%*	*90%*

Source: Leerink Swann Estimates

Margetuximab US Revenue Model

US Margetuximab sales	2019E	2020E	2021E	2022E	2023E	2024E	2025E	2026E	2027E	2028E	2029E	2030E
1st line mBC patients	155	782	1,566	1,882	1,885	1,888	1,892	1,895	1,898	1,901	1,905	1,908
Duration of therapy (months)	12	12	12	12	12	12	12	12	12	12	12	12
Monthly cost ($)	9,800	10,045	10,296	10,554	10,817	11,088	11,365	11,649	11,940	12,239	12,545	12,858
Cost/patient ($)	117,600	120,540	123,554	126,642	129,808	133,054	136,380	139,789	143,284	146,866	150,538	154,301
1st line mBC sales ($MM)	**18**	**94**	**193**	**238**	**245**	**251**	**258**	**265**	**272**	**279**	**287**	**294**
2nd line mBC patients	114	451	904	725	726	727	728	730	731	732	733	735
Duration of therapy (months)	9.0	9.0	9.0	9.0	9.0	9.0	9.0	9.0	9.0	9.0	9.0	9.0
Monthly cost ($)	9,800	10,045	10,296	10,554	10,817	11,088	11,365	11,649	11,940	12,239	12,545	12,858
Cost/patient ($)	88,200	90,405	92,665	94,982	97,356	99,790	102,285	104,842	107,463	110,150	112,903	115,726
2nd line mBC sales ($MM)	**10**	**41**	**84**	**69**	**71**	**73**	**74**	**76**	**79**	**81**	**83**	**85**
3rd line mBC patients	162	254	203	163	163	164	164	164	164	165	165	165
Duration of therapy (months)	4.0	4.0	4.0	4.0	4.0	4.0	4.0	4.0	4.0	4.0	4.0	4.0
Monthly cost ($)	9,800	10,045	10,296	10,554	10,817	11,088	11,365	11,649	11,940	12,239	12,545	12,858
Cost/patient ($)	39,200	40,180	41,185	42,214	43,269	44,351	45,460	46,596	47,761	48,955	50,179	51,434
3rd line mBC sales ($MM)	**6**	**10**	**8**	**7**	**7**	**7**	**7**	**8**	**8**	**8**	**8**	**9**
Total mBC sales ($MM)	**35**	**145**	**286**	**314**	**322**	**331**	**340**	**349**	**358**	**368**	**378**	**388**
3rd line mGC patients	811	1,650	2,685	2,732	2,778	2,826	2,875	2,924	2,974	3,025	3,077	3,130
Duration of therapy (months)	5.0	5.0	5.0	5.0	5.0	5.0	5.0	5.0	5.0	5.0	5.0	5.0
Monthly cost ($)	9,800	10,045	10,296	10,554	10,817	11,088	11,365	11,649	11,940	12,239	12,545	12,858
Cost/patient ($)	49,000	50,225	51,481	52,768	54,087	55,439	56,825	58,246	59,702	61,194	62,724	64,292
3rd line mGC sales ($MM)	**40**	**83**	**138**	**144**	**150**	**157**	**163**	**170**	**178**	**185**	**193**	**201**

Source: Leerink Swann Estimates

Fc-optimized monoclonal antibody targeting B7-H3

MGA271

MGA271 – Key Takeaways

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- **We believe B7-H3 is an attractive new target for anti-cancer therapy.** B7-H3 is a tumor-specific antigen and a novel member of the B7 family of immune regulators. The B7 family of cell surface molecules consists of structurally related protein ligands that bind to receptors on lymphocytes and regulate immune responses. B7-H3 is a novel member of the B7 family of immune regulatory molecules which also includes PD-L1 and others. B7-H3 is over-expressed in 70%-99% of solid tumors, including prostate, pancreatic, melanoma, renal cell, ovarian, colorectal, gastric, bladder, and non-small cell lung cancers.
- **MGNX selected MGA271 from a panel of hits based on strong selectivity for cancer tissue and cancer stem-like cells** and not binding to a broad set of normal tissues. The initially identified antibody was humanized and fused with MGNX's optimized human IgG1 Fc domain that has increased ADCC activity. We this believes MGA271 may have two mechanisms of action: 1) tumor and tumor stem cell-killing via ADCC, and 2) enhancement of anti-tumor immunity by blockade of T-cell inhibition.
- **Positive Phase I expansion phase data in 2014 will be a key catalyst for this program which could trigger Servier to exercise its option to license European rights.** Based on data release so far, we believe MGA271 is safe. The dose-escalation portion of a ongoing Phase I trial has been completed without exceeding a maximally tolerated dose (MTD), a key positive in our view. In Phase Ia, 26 refractory patients not responding to standard treatment anymore (9 dose cohorts, 15 different tumor types) were enrolled. Servier has indicated that it intends to evaluate MGA271 in up to 90 additional cancer patients in 4Q13, according to MGNX and based on feedback from management, we believe it is highly likely that Servier will exercise its license option in 1H14 which would trigger a $30MM milestone payment to MGNX .
- **Dose expansion data in 2014 should provide insight into the potential utility for MGA271.** During the dose expansion phase, which is currently ongoing, MGNX is recruiting 15 patients to each of three cohorts, 1)patients with melanoma, 2)patients with prostate cancer, and 3)patients with any B7-H3 positive tumor other than melanoma or prostate cancer. Based on MEDACorp Specialist feedback, we believe MGA271 could be particularly attractive in tumor types with low response rates for other checkpoint inhibitors (e.g., colorectal cancer, prostate cancer), as single agent or in combination with CTLA-4 or PD-1/PD-L1 inhibitors, or in patients with low PD-L1 expressing tumors.

MGA271 – Product Profile

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- MGA271 is a first-in-class, Fc-enhanced monoclonal antibody that recognizes human B7-H3, currently in Phase Ib trials
- B7-H3 is a tumor-specific antigen and a novel member of the B7 family of immune regulators (same family as PD-L1, CTLA-4)
- MGA271 has potentially two mechanisms of action:
  - Enhanced cell killing through ADCC by binding to the cancer specific antigen B7-H3
  - Enhancement of anti-tumor immunity by blockade of T-cell inhibition
- MGNX optioned MGA271 to Servier in Dec, 2011:
  - MGNX has retained commercialization rights in North America, Japan, Korea and India
  - Servier has the right to license EU + rest-of-world rights
  - Prior to exercise of the license, both parties will fund and conduct specified research and development activities
  - MGNX received a $20MM upfront payment and could receive a license fee of $30MM if Servier exercises its option upon completion of the Phase I expansion cohorts (expected in 1H14)
  - MGNX has the right to receive to an additional $390MM in clinical, regulatory and commercialization milestone payments plus tiered, double-digit royalties on future net sales
  - Both parties will share the clinical development costs following the option exercise

MGA271 is Selectively Targeting B7-H3, A Tumor-Specific Antigen

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- MGNX identified MGA271 in an immunological screen for antigens expressed on the surface of cancer cells.
- MGA271 was selected from a panel of hits based on strong selectivity for cancer tissue and not binding to a broad set of normal tissues. The initially identified antibody was humanized and fused with MGNX's optimized human IgG1 Fc domain that has increased affinity for both alleles of the human activating FcgR, CD16A, and decreased affinity for the inhibitory FcgR, CD32B.
- Because of the possible immune regulatory capacity of B7-H3, reactivity of MGA271 in lymphatic tissues was also examined. No reactivity of MGA271 with lymph node and spleen tissues was observed.
- B7-H3 is expressed across a broad range of cancer types and we believe an overwhelming number of clinically relevant studies have shown that B7-H3 exhibits complex and predominately inhibitory interactions with host T-cells in cancer patients, and it is thought to potentially play a role in promoting tumor invasion and/or metastasis.

Table 2. Summary of immunohistochemical staining of FFPE tumor specimens with anti–B7-H3 mAb BRCA69D to evaluate B7-H3 expression across a broad range of cancer types

		Positive staining (any grade)		Moderate to high staining (2+ or greater)	
Tissue	Type	Positive/total	%Positive	Positive/Total	%Positive
Melanoma	Primary	48/51	94	25/51	49
	Metastatic	18/19	95	7/19	37
	Total	66/70	94	32/70	46
Kidney cancer	Primary	77/78	99	75/78	96
Prostate cancer	Primary	88/99	89	51/99	52
Pancreatic cancer	Primary	69/78	88	45/78	58
Gastric cancer	Primary	100/115	87	100/115	87
Breast cancer	Primary	76/90	84	74/90	82
Ovarian cancer	Primary	39/52	75	19/52	37
	Metastatic	4/8	50	2/8	25
	Total	43/60	72	21/60	35
Small cell lung cancer	Primary	12/75	16	6/75	8

Source: Clin Cancer Res; 18(14) July 15, 2012

MGA271 May Have Two Mechanisms of Action

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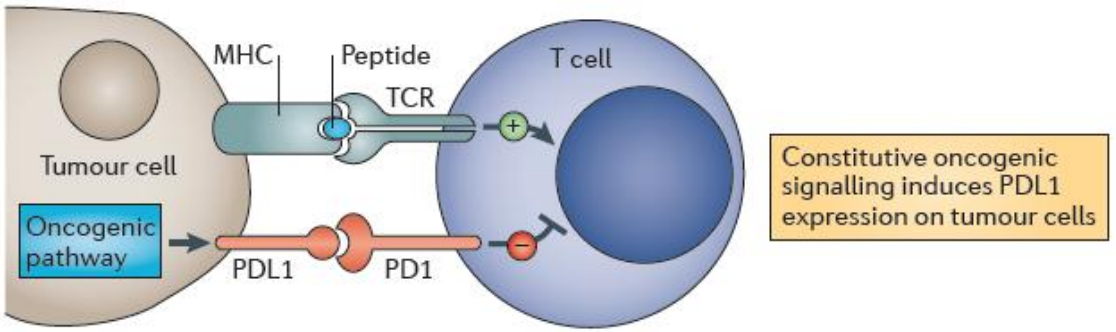
- Mechanism #1: Enhanced cell killing through ADCC by binding to the cancer specific antigen B7-H3:
  - B7-H3 is highly expressed on cancer cells, cancer stem-like cells and the surrounding tumor microenvironment
  - MGA271 has been designed to have enhanced binding to CD16A and reduced binding to CD32B. It thus mediate the killing of B7-H3-positive cancer cells through antibody-dependent cellular cytotoxicity (ADCC)
- Mechanism #2: Enhancement of anti-tumor immunity by blockade of T-cell inhibition:
  - The immune system can use antigens to distinguish tumor cells from their normal counterparts
  - Amplitude and quality of the immune-response are regulated by a balance between co-stimulatory and inhibitory signals (="immune checkpoints")
  - Under normal physiological conditions, immune checkpoints are critical for maintenance of self-tolerance (prevention of autoimmunity), but immune-checkpoint proteins can be deregulated by tumors as an important immune resistance mechanism
  - The aim of cancer immunotherapy is to treat malignant disease by inducing or enhancing cancer-specific immune responses. this can be achieved using antagonists of inhibitory signals, which results in the amplification of anti-tumor T cell responses (e.g. anti-PD1/PD-L1 antibodies)
  - Given that B7-H3 is an immune-inhibitory ligand, inhibiting B7-H3 with an antibody could have an immune-modulary effect

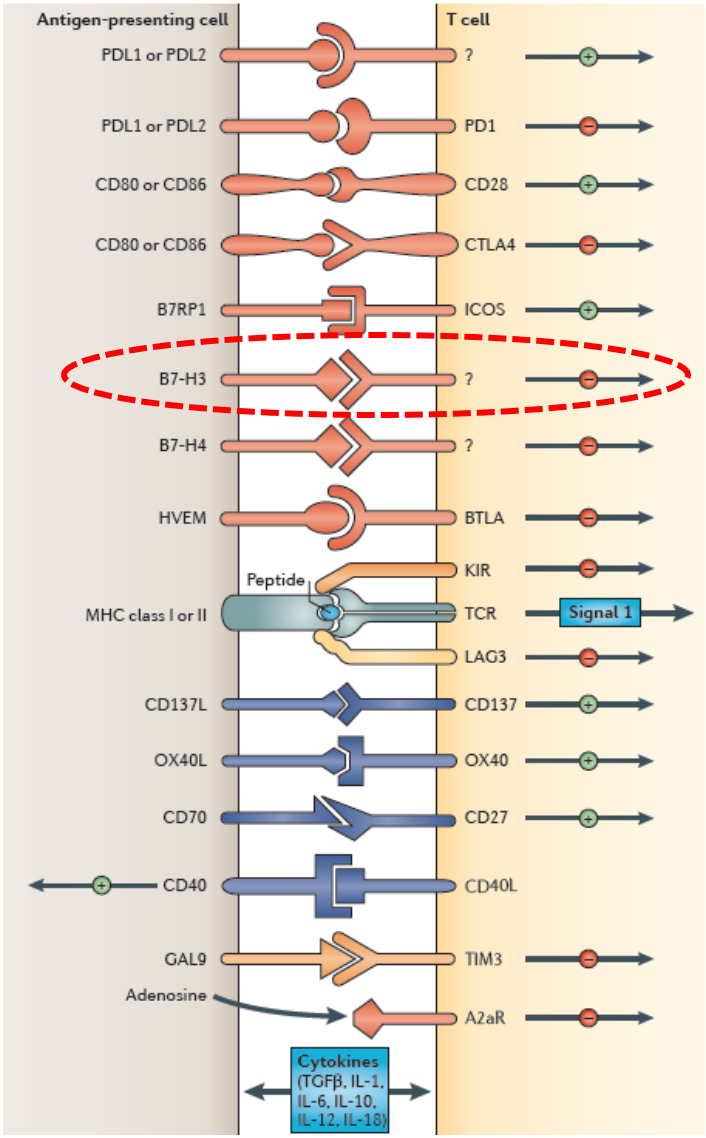


Source: Nat Rev Cancer. 2012 Mar 22

B7-H3 is A Recently Identified Member of the B7/CD28 Family of Co-stimulatory Molecules

LEERINK SWANN

- Basic immunological studies have demonstrated that various immune-checkpoint receptors are expressed coordinately under circumstances of tolerance to self antigens and chronic infections, as well as in inflammatory settings.
- In addition to defined lymphocyte inhibitory receptors, numerous B7 family inhibitory ligands — in particular B7-H3 and B7-H4 do not yet have defined receptors; we believe work in animal models support an immune inhibitory role for these ligands.
- In addition, B7-H3 and B7-H4 are upregulated on tumor cells or tumor-infiltrating cells. B7-H3 seems to be upregulated on endothelial cells of the tumor vasculature, and B7-H4 has been reported to be expressed on tumor-associated macrophages.
- Preclinical mouse models of cancer have shown that blockade of many of these individual immune checkpoint ligands or receptors can enhance antitumor immunity, and dual blockade of coordinately expressed receptors can produce additive or synergistic antitumor activities.
- Inhibitors for a number of these immune checkpoint targets are either entering the clinic or are under active development.



Source: Nat Rev Cancer. 2012 Mar 22

The Exact Molecular Function of B7-H3 is Still Being Investigated; Data Strongly Suggests an Immune-Inhibitory Function

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- **B7-H3 is one of the most recently identified members of the B7/CD28 family of co-stimulatory molecules** serving as an accessory modulator of T-cell response. The B7-H3 receptor(s) and its exact functions are still unknown, but data points to a likely inhibitory immune checkpoint function, in our view.
- **B7-H3 is selectively expressed on human cancers indicating a function of B7-H3 as a regulator of antitumor immunity**. With exception of a couple of studies that may have, in our view, some limitations, we believe expression of B7-H3 in human cancers appears to be generally associated with an immune-suppressive activity. In one retrospective renal cell carcinoma (RCC) study for example (Clin. Cancer Research, 2008) , 17% of tumor cells and 95% of tumor vasculature in 743 examined patients expressed B7-H3. B7-H3 expression in either tumor cells or tumor vasculature was found to significantly associated with an increased risk of death from clear cell RCC. Similar results were shown in another study that investigated B7-H3 expression in 823 patients with prostate cancer (PANS vol. 104, no. 49). Tumor B7-H3 expression was found in 93% of patients treated with radical prostatectomy. Strong B7- H3 expression in the resected specimens correlated with disease spread and poor outcome.
- **The molecular functions B7-H3 have not yet been resolved in detail, but several possible mechanisms may account for the ability of B7-H3-expressing cells to evade tumor immunity**. The receptor(s) for B7-H3 have not yet been identified. Similar to other molecules, B7-H3 might interact with both inhibitory and stimulatory receptors. Tumor-associated B7-H3 might exert distinct functions depending on different affinities for several existing receptors. Similar to CTLA-4, under specific circumstances, B7-H3 may have a much higher affinity for binding of its inhibitory receptor and is thought to directly compete with its stimulatory receptor to prevent over initiation of the co-stimulatory signal, leading to decreased T-cell activation. In addition, B7-H3 may also affect other immune cells than T cells.
- **As a tumor-associated antigen, B7-H3 may also play a non-immunological role in cancer progression which could explain its role in promoting resistance to chemotherapy.** One of several studies studies showed that decreased B7-H3 expression resulted in increased sensitivity of human breast cancer cell lines to paclitaxel as a result of enhanced drug-induced apoptosis.

MGA271 Mediates Potent ADCC in Vitro and Tumor Shrinkage in Animal Models

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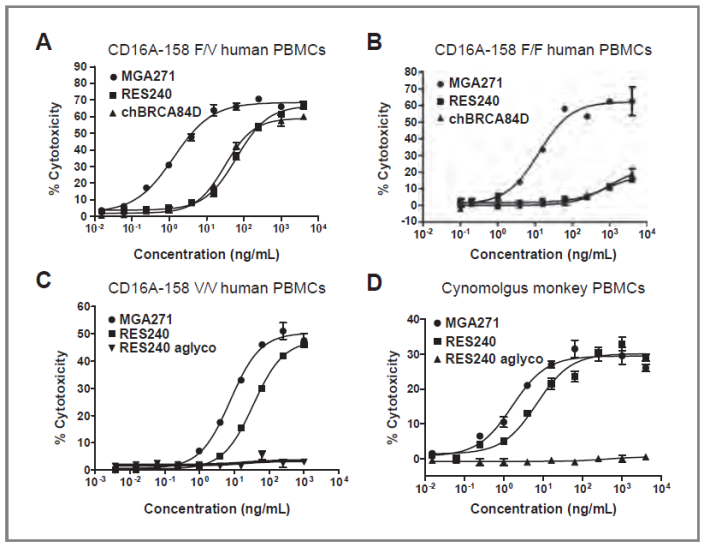
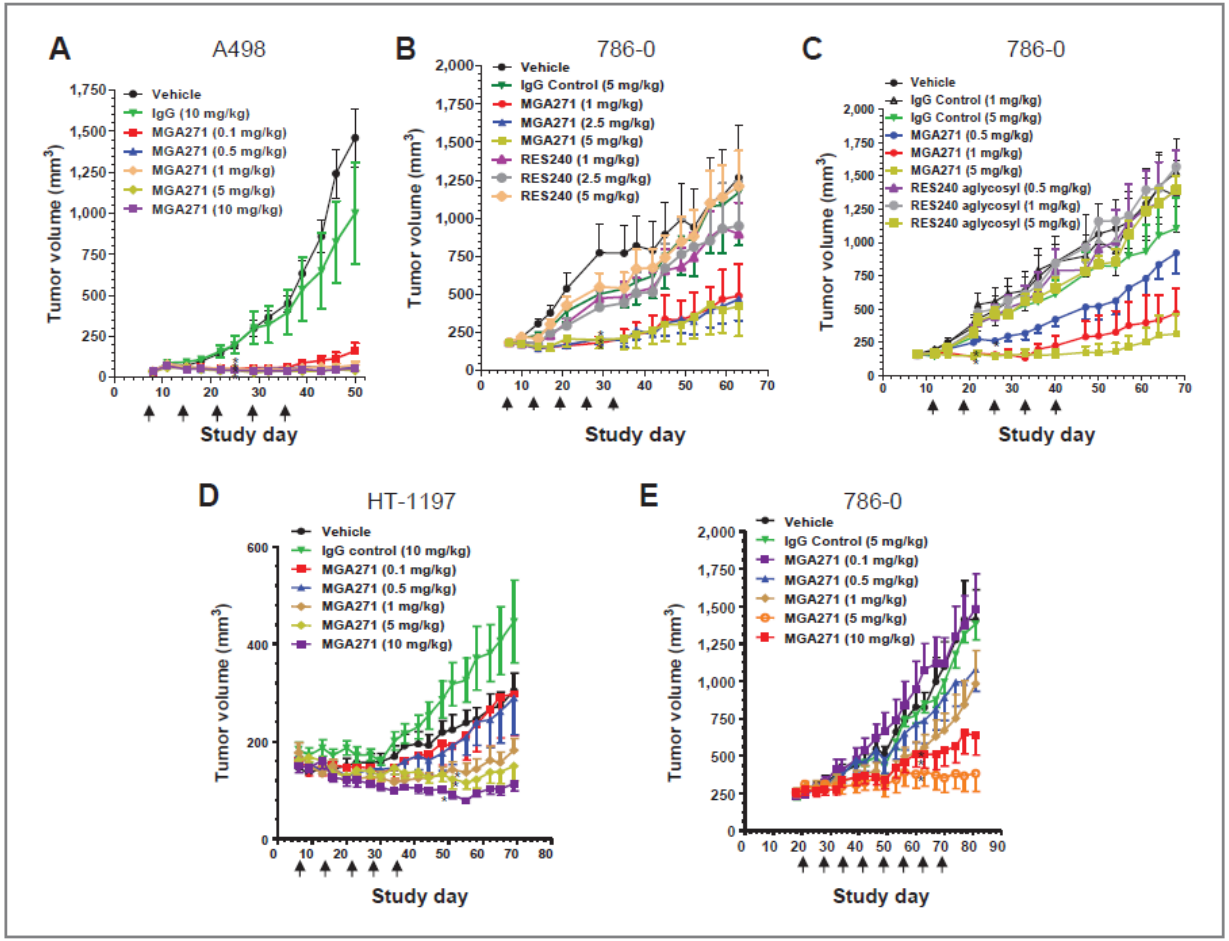
MGA271 mediates in vitro ADCC on A498 renal cell carcinoma cells with human PBMC effector cells representing all 3 CD16A-158 genotypes and with cynomolgus monkey PBMC effector cells



Source: Clin Cancer Res; 18(14) July 15, 2012

MGA271 exhibits potent in vivo antitumor activity toward tumor cell carcinoma xenografts



Source: Clin Cancer Res; 18(14) July 15, 2012

MGA271 Appears Very Safe, Based on Phase Ia Dose-Escalation Data

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- MGNX is currently conducting a Phase I open-label, multi-dose, single-arm, multi-center, clinical trial of MGA271. First data from this trial is expected to be presented in mid-2014.
- The trial includes patients with B7-H3-expressing tumors, such as prostate cancer, pancreatic cancer, melanoma and ovarian cancer, and tumors whose vasculature exhibits B7-H3 expression, such as glioblastoma, renal cell carcinoma and ovarian cancer.
- Dose-escalation portion of the trial has been completed without exceeding a maximally tolerated dose (MTD), a key positive in our view. In Phase Ia, 26 refractory patients not responding to standard treatment anymore (9 dose cohorts, 15 different tumor types) were enrolled.
- MGNX has not observed any dose limiting toxicity (DLT) in Phase Ia, and initiated dose-expansion at a dose of 15 mg/kg weekly in August '13. The Phase I trial began with a dose escalation segment in which patients were treated with increasing weekly doses of MGA271 from 0.01 mg/kg up to 15 mg/kg. Most frequent adverse effects (AE) were mild/moderate infusion reactions.
- 10 of the 26 Phase Ia patients had stable disease (SD). Tumor response assessments occurred at approximately six weeks after the first MGA271 dose for each patient. Patients with PR, CR or SD (RECIST or RANO criteria) were allowed to continue therapy at the same dose (10 patients). Subsequent cycles consist of MGA271 administration days 1, 8, and 15 of each 28-day cycle, with tumor evaluation every other cycle .
- During the expansion phase (triggered $10MM milestone payment) which is currently ongoing, MGNX is recruiting 15 patients to each of three cohorts:
  - Patients with melanoma
  - Patients with prostate cancer
  - Patients with any B7-H3 positive tumor other than melanoma or prostate cancer with the limitation of a maximum of five patients with any single histological type

Upcoming Phase Ia and Ib Data Presentations are Key Catalysts Which Could Indicate Activity in Specific Tumors

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- We expect investors will have more specific insight on the market opportunity and positioning for MGA271 based Phase Ib dose expansion data in 2H14 which is the key near-term catalyst for MGNX in our view
- We believe MGA271 could potentially be developed across multiple solid tumors, depending on the magnitude and range of activity seen in different solid tumor types in Phase Ib.
- Based on MEDACorp Specialist feedback, we believe MGA271 could be particularly attractive in tumor types with low response rates for other checkpoint inhibitors (e.g., colorectal cancer, prostate cancer), as single agent or in combination with CTLA-4 or PD-1/PD-L1 inhibitors, or in patients with low PD-L1 expressing tumors.
- MGNX expects a companion diagnostic to be ready for Phase III and is currently working with two vendors for the development of a test measuring B7-H3 expression.
- Servier has indicated that it intends to evaluate MGA271 in up to 90 additional cancer patients in 4Q13, according to MGNX and based on feedback from management, we believe it is highly likely that Servier will exercise its license option in 1H14, which would trigger a $30MM milestone payment to MGNX .
- Phase Ia dose escalation phase data will be presented in mid-2014
- Initial Phase Ib dose expansion phase data will be available in late 2014
- MGNX plans to initiate a Phase II clinical trial in early 2015 should Phase Ib dose-expansion data be positive.

Checkpoint Inhibitors in Development are Largely Focused on the PD1/PD-L1 System

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Ligand	Receptor	Product Candidates	Company	Status
CD80 (B7-1) or CD86 (B7-2)	CTLA4	Ipilimumab (Yervoy)	BMY	approved
		Tremelimumab	AZN	Phase II
CD80 (B7-1) or CD86 (B7-2)	CD28	n/a		
PD-L1 (B7-H1) or PD-L2 (B7-DC)	PD1	lambrolizumab	MRK	Phase III
		Nivolumab	BMY	Phase III
		Pidilizumab	Curetech	Phase II
		RG7446	Roche	Phase II
		AMP-224	Amplimmune/AZN	Phase I
		BMS-936559	BMY	Phase I
		MEDI-4736	AZN	Phase I
		MSB0010718C	Merck KGaA	Phase I
B7RP1 (B7-H2)	ICOS	AMG 557	AMGN	Phase I
B7-H3	Unknown	MGA271	MGNX	Phase I
		8H9 MAb	MSKCC	Phase I (status unclear)
B7-H4	Unknown	AMP-110	Amplimmune/AZN	Phase I (RA)
B7-H5 (VISTA)	Unknown	n/a		
B7-H6	NKp30	n/a		

Source: Clinicaltrials.gov

Nivolumab Phase Ib data monotherapy (Study 003):

- ❑Safety: Grade 3 or 4 drug related AEs in 14% of patients; 3 deaths from pulmonary toxicity (pneumonitis)
- ❑Efficacy (ORR), all doses (pivotal 3mg/kg dose)
  - ❑Non-small cell lung cancer (n=127): 17% (24%)
  - ❑Melanoma (n=107): 31% (41%)
  - ❑Renal-cell cancer (n=34): 29% (n/a)
  - ❑Castration-resistant prostate cancer (n=17) : n/a
  - ❑Colorectal cancer (n=19): n/a

Phase I data Yervoy/nivolumab combination (Study 004):

- ❑Melanoma ORR: 40% (all doses); 53% (1mg/kg nivolumab+3mg/kg Yervoy)
- ❑Seven potential registration studies are ongoing in NSCLC, melanoma, renal cell carcinoma

- ❑Phase III trials for nivolumab and lambrolizumab are focused on melanoma, NSCLC, and Renal Cell Cancer (RCC)

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PLATFORM AND PRECLINICAL PIPELINE

MGNX Is an R&D Driven Biotech Company Based on a Suite of Three Platform Technologies

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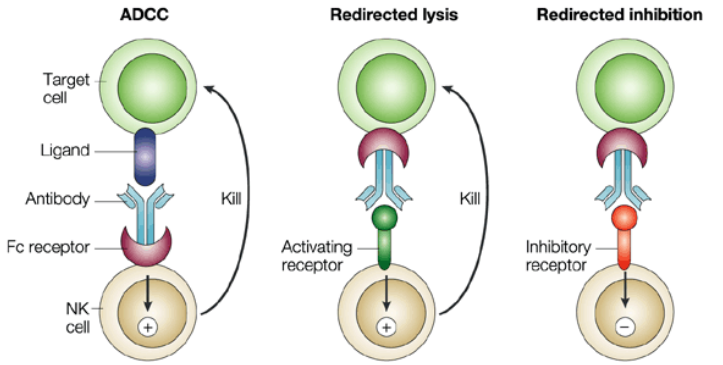
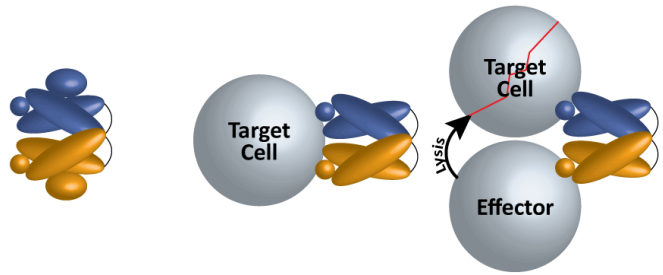
MGNX's suite of platforms allows generation of custom-designed antibodies or antibody-derived molecules that are optimized to treat specific diseases.

1. Fc-Optimization platform
   - Enhances the body's immune system to mediate the killing of cancer cells through antibody-dependent cellular cytotoxicity (ADCC)
   - Specific antibody modules with increased affinity for the human activating Fc-gamma receptor IIIA (CD16A) and decreased affinity for the inhibitory Fc-gamma RIIB (CD32B)
2. DART platform (Dual Affinity Re-Targeting)
   - Enables the targeting of multiple antigens or cells by using a single molecule with an antibody-like structure
   - Ability to recruit any T cell in a patient's body to destroy targeted cancer cells
   - Potential best-in class platform with improved stability, half-lives, and manufacturing efficiencies for DARTs
3. Cancer Stem-like Cell (CSLC) platform
   - Unique discovery tool to identify cancer targets shared both by tumor initiating cells (CSLCs) and the differentiated cancer cells derived from them



Source: Nature Reviews Immunology Nature Reviews | Immunology



Source: MacroGenics

MGNX's DART Platform Drives Partnerships

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- Platform generated $106MM in milestone payments from partnerships, 2010-1H13
- MGNX is eligible for $5.3Bn in potential total milestone payments ($1.0Bn clinical milestones) across all products
- We believe there may be upside to MGNX expectation of likely receipt of $60MM in DART-partnership related milestone payments 2H13-2015
- Four DART partnerships to date:
  - Servier: Identified all 3 DARTs to date – INDs for MGD006, MGCD007 in 2014; 3rd undisclosed
  - GILD: specified two of targets to date; executed 1 WW license, has 3 additional WW options
  - PFE: currently pursuing one of two possible DART programs; MGNX R&D ends in October 2013
  - Boehringer Ingelheim: Identified several targets; 2 more years to go in 5-year R&D deal

Boeringer Ingelheim	**26-Oct-10**
10 DARTs	WW; US co-promote option for some
Upfront payment	$15MM upfront + rec'd 2 annual maintenance payments; exp 3rd annual maintenance payment in 4Q13
Preclinical payments to MCGN	$60
Clinical/reg/sales milestones	up to $210MM per DART
Royalties on sales	mid single-digit
Research costs	shared
Clinical dev't costs	Boehringer, unless MGNX opt-in

Pfizer	**26-Oct-10**
2 DARTs	WW
Upfront payment	$5MM
Preclinical payments to MCGN	undisclosed
Clinical/reg/sales milestones	up to $210MM per DART
Royalties on sales	mid-single digit to low double digit
Research costs	MGNX will rec research funding
Clinical dev't costs	PFE only

Servier	**20-Sep-12**
3 DARTs	MGNX retains full rights to each program in North America, Japan, Korea and India
Upfront payment	20
Preclinical payments to MCGN	80
Clinical/reg/sales milestones	up to $1Bn
Royalties on sales	tired low double digit to mid-teens
Research costs	shared pre-license
Clinical dev't costs	shared post-lincense

Gilead	**7-Jan-13**
4 DARTs	WW license option for 3, MGNX copromote ROW for 1
Upfront payment/lincese fees	30
Preclinical payments to MCGN	85
Clinical/reg/sales milestones	up to $1Bn
Royalties on sales	tiered high-single to low double-digit
Research costs	Gilead
Clinical dev't costs	Gilead

Source: SEC Filings

MGNX Has Generated a Promising Preclinical DART Pipeline

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- **MGD006** is a humanized DART that recognizes CD123 and CD3 (T-cell engager).
  - Optioned to Servier; MGNX retained North America, East Asia rights
  - CD123 is expressed on leukemic stem cells (LSCs), but not on the corresponding normal hematopoietic stem cell population in normal human bone marrow
  - IND is expected in early 2014 (will trigger $5MM milestone from Servier); Opt-in expected in 2014 ($15MM milestone)
  - Initiation of Phase I clinical trial in patients with relapsed or refractory acute myeloid leukemia (AML) or in patients with untreated AML who are not candidates for standard induction chemotherapy is planned for 1H14
- **MGD007** is a humanized DART that recognizes gpA33 and CD3 (T-cell engager).
  - Optioned to Servier; MGNX retained North America, East Asia rights
  - Extended serum half-life through fusion to Fc domain
  - gpA33 is highly expressed in colorectal cancer cells and not in normal mucosa
  - IND is expected in mid-2014 ($5MM milestone from Servier); Initiation of Phase I in 2H14
  - Potential Servier opt-in in 2015 (triggers undisclosed license fee)
- **MGD010** is a DART targeting CD32B (inhibitory Fc receptor) and CD79B
  - Modulates the function of human B-cells without B-cell depletion by triggering an inhibitory “immune checkpoint”
  - Proprietary to MGNX; Potential development in autoimmune diseases (RA, Crohn’s, SLE, etc.)

DART Platform Is Source of MGNX's Partnerships

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- MGNX's DART platform allows creation of derivatives of antibodies with the ability to bind to multiple targets simultaneously instead of a single target found in traditional monoclonal antibodies.
- By enabling the simultaneous engagement of two different targets, DARTs broaden the potential utility of traditional antibody-based therapies and allow:
  - Redirected T-cell Activation and Killing (e.g., by targeting CD19 and CD3 [AMGN's bilantumumab]).
  - Modulation of receptor signaling (e.g., by targeting CD79B and CD32B [MGNX's MGCD010]).
  - Simultaneous targeting of multiple pathologic factors (e.g., by targeting Her2 and Her3 [MACK's MM-111]).
- Dual specificity antibodies can be constructed in several different formats, e.g., by generation of IgG-like designs, Fc fusions, Fab fusions, single-chain variable fragment (ScFv)-based molecules and IgG–non-IgG fusions.
- Although the concept of dual specificity antibodies has been around for decades, challenges in creating these molecules have slowed their advancement. Key challenges have been the instability of the resulting constructs and their short half-lives, as well as the inefficiencies in manufacturing these compounds.
- MGNX believes that its DART platform has overcome these challenges by incorporating proprietary covalent di-sulfide linkages and particular amino acid sequences that efficiently pair the chains of the DART molecule. This results in a structure with enhanced manufacturability, long-term structural stability, and the ability to tailor the half-lives of the DARTs to their clinical needs. The engineered antibody-like protein has a very compact and stable structure and enables the targeting of multiple different antigens within a single recombinant molecule.
- In addition, MGNX has the ability to tailor a DART molecule's valency (number of binding sites), the strength by which the binding sites attach to its targets, and its half-life in the blood circulation after delivery to a patient. When an Fc domain is incorporated in a DART, changes can be included that can modulate the DART's engagement with different immune cells (e.g., MGCD007).

MGNX's DARTs vs. AMGN's BiTEs

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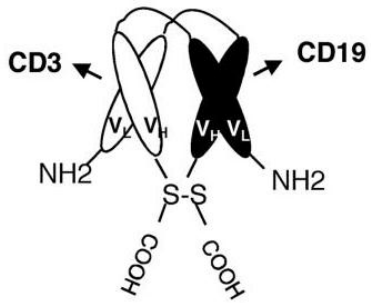
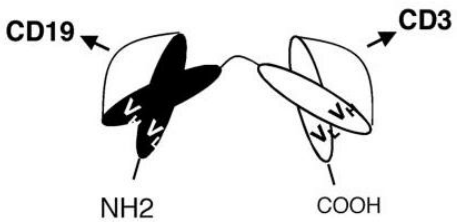
- In January, 2012 AMGN acquired Micromet for $1.16MM in cash. Micromet like MGNX was a pioneer in the area of dual specificity antibodies, in our view. The acquisition not only provided AMGN with Micromet's Bispecific T-cell Engager (BiTE) technology, but also included blinatumomab, a BiTE in Phase II clinical development for acute lymphoblastic leukemia (ALL).
- Blinatumomab is currently the most clinically advanced dual-specificity antibody, and results from early clinical studies of blinatumomab have demonstrated impressive response rates in both relapsing non-Hodgkin lymphoma and B-cell acute lymphoblastic leukemia patients, providing additional proof of concept of the T-cell engaging treatment concept.
- Despite this success, scFv-based bispecific strategies including BiTEs have limitations in our view, including constraints imparted by the linker sequences that connect the V regions, resulting in reduced or altered antigen recognition and potency. Furthermore, scFv-based constructs have a tendency to form aggregates due to "domain exchange" of the V regions with partners from other molecules.
- To address the functional and structural limitations of existing bispecific molecules, MGNX has developed its alternative bispecific antibody platform called dual affinity retargeting (DART). In DART proteins, each Fv is formed by the association of a VL partner on one chain with a VH partner on the second chain in a VLA-VHB VLB-VHA configuration. This configuration lacks the constraint of an intervening linker sequence and therefore is more analogous to the natural association in an IgG molecule. The covalent linkage between the 2 chains limits the freedom of the component Fv domains to undergo domain exchange, resulting in a high degree of stability that is independent of the strength of the VL-VH interface.

CD19 x CD3 DART





CD19 x CD3 BiTE (blinatumomab)

Source: BLOOD, 28 APRIL 2011 VOLUME 117, NUMBER 17

MGNX's DARTs Outperforms BiTEs In-Vitro

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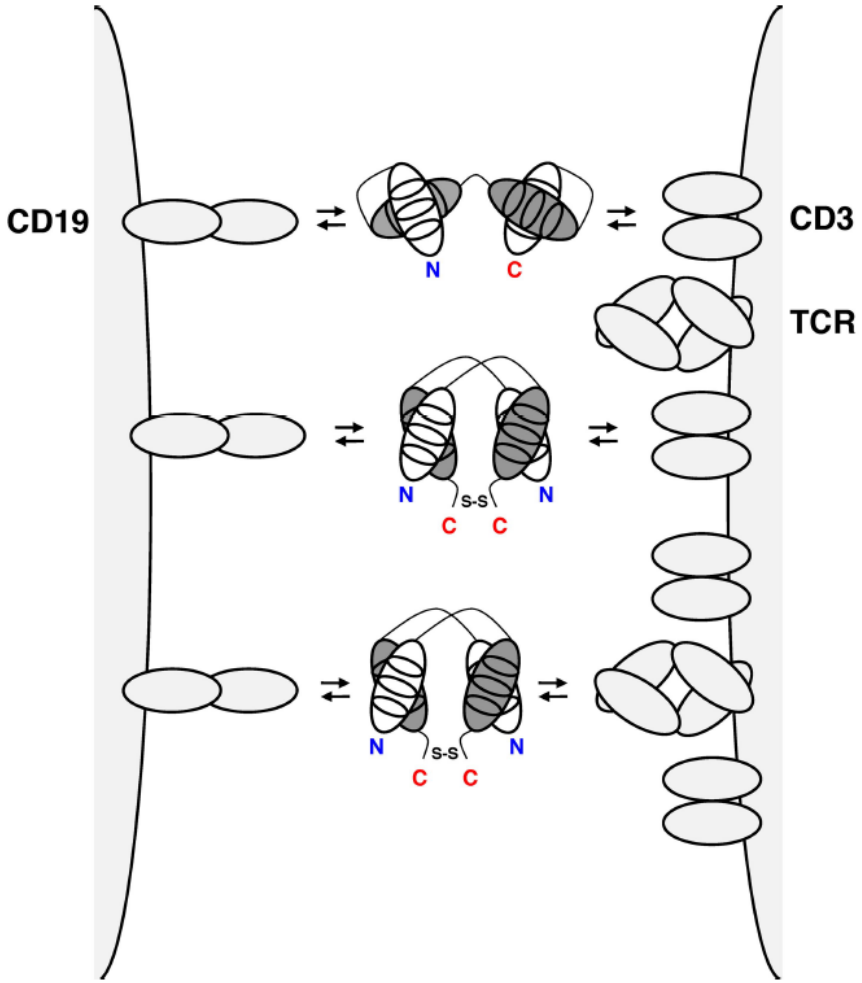
- The DART format separates cognate variable domains of heavy and light chains of the 2 antigen binding specificities on 2 separate polypeptide chains. Whereas the 2 polypeptide chains associate non-covalently in the diabody format, the DART format provides additional stabilization through a C-terminal disulfide bridge.
- A side-by-side comparison of the in-vitro performance of CD19xCD3 DART and BiTE molecules that were based on the same parental mouse anti–human CD3 and mouse anti–human CD19 monoclonal antibodies as blinatumomab showed that the bispecific antibody in the DART format consistently outperformed the BiTE format with respect to the maximal level of B-cell lysis, the concentration required for half-maximal B-cell lysis, and the induction of molecular markers of T-cell activation
- DARTs can be produced in high quantity and quality and reveal exceptional stability in both formulation buffer and human serum.



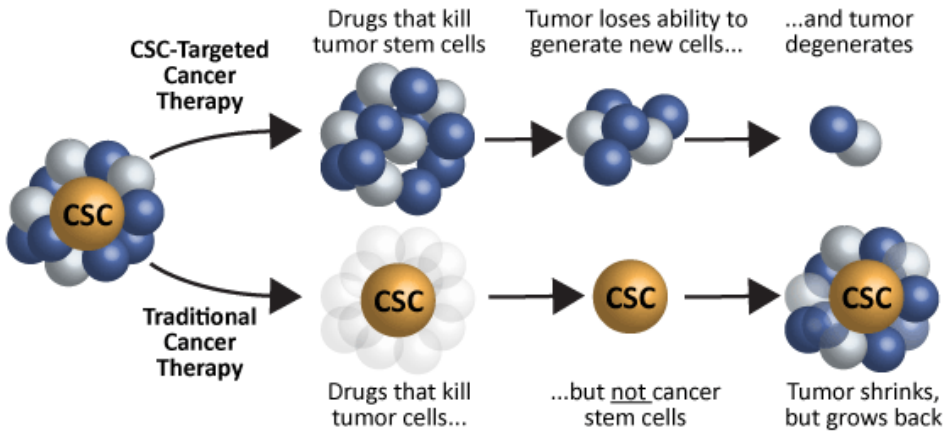
CD19xCD3 BiTE (top), CD19xCD3 DART (middle), and CD19xTCR DART (bottom) cross-link a normal or malignant B cell through CD19 (left) and a T cell through the TCR complex (right).

Source: BLOOD, 28 APRIL 2011 VOLUME 117, NUMBER 17

Cancer Stem-like Cell (CSLC) Platform: MGNX's Proprietary Approach to Discover Cancer Targets

LEERINK SWANN

- MGNX's CSLC platform provides new approaches to discover and identify cancer targets that are unresponsive to current cancer therapies.
- Cancer stem cells represent important potential targets in oncology drug development because they are theorized to be the basis for tumor re-growth and metastasis and are refractory to much standard chemotherapy.
- Therefore, the ability to specifically target and destroy CSLCs could potentially address an unmet medical need in many hard-to-treat cancers today.
- MGNX has developed an in-house expertise in growing stem cells from normal tissues using proprietary media and culture conditions, and have produced CSLCs from primary human tumor tissues (colon, lung, ovary).
- MGNX has created a library of over 1,900 novel monoclonal antibodies that target antigens on both CSLCs and bulk differentiated tumor cells, which are derived from the CSLCs.
- This antibody library has been screened for low binding to normal, non-malignant tissues and MGNX are using this antibody library as a source to develop new product candidates.
- MGA271 and MGD007 have been discovered using MGNX's CSLC platform



Source: MacroGenics

MGNX Applied its Fc-Optimization Capability to Margetuximab and MGA271

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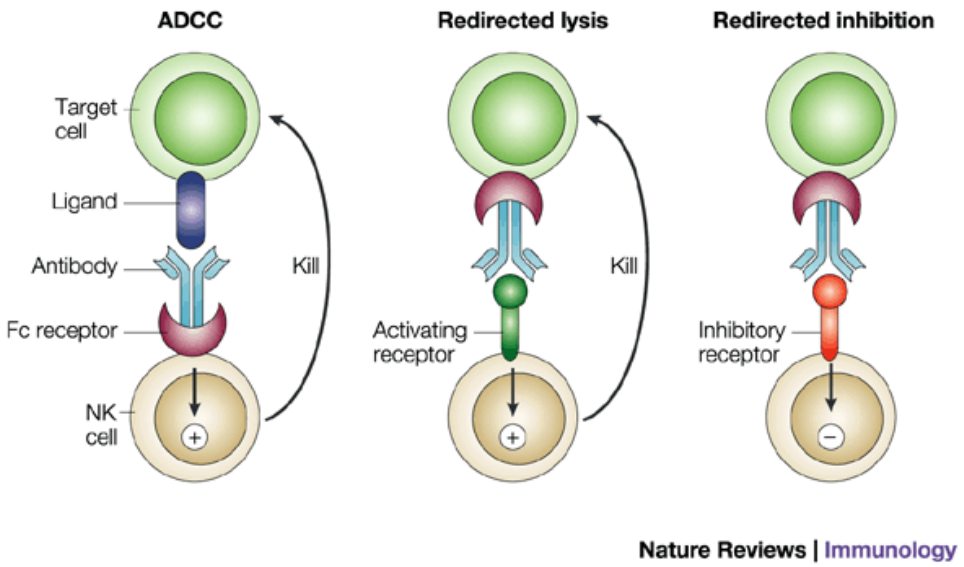
- We believe MGNX has been a pioneer in the area of antibody engineering and its detailed knowledge of antibody structure and activity has allowed it to generate therapeutic antibodies with improved properties.
- Modifying effector functions can be achieved by engineering the constant (Fc) regions of antibodies to either improve or reduce their binding to FcgRs and thus modulate interactions with immune cells.
- The binding of IgG to the activating (FcgRI, FcgRIIa, FcgRIIIa and FcgRIIIb) and inhibitory (FcgRIIb) FcgRs depends on residues located in the hinge region and the CH2 domain.
- MGNX has identified and patented certain five mutations in within the Fc region which are able to enhance the body's ability to mediate the killing of cancer cells through antibody-dependent cellular cytotoxicity (ADCC). Specifically, MGNX has generated a modular Fc domain with decreased affinity to the inhibitory FcgRIIb and increased affinity to FcgRIIIa and IIIb.
- MGNX pre-clinical data demonstrated that these Fc variants have substantially improved the antibody's therapeutic effects, and MGNX has incorporated its Fc variants in Margetuximab and MGA271.



Backgrounder – Antibody Structure

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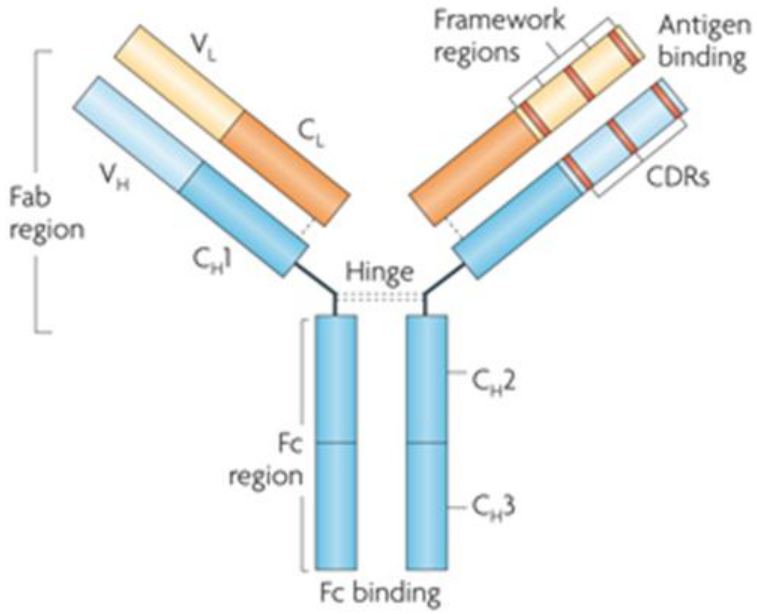
- Antibodies can be subdivided into two distinct functional units:
  - the fragment of antigen binding (Fab) and
  - the constant fragment (Fc)
- The Fab contains the variable region (Fv), which consists of three hypervariable complementarity-determining regions (CDRs) that form the antigen binding site of the antibody and confer antigen specificity.
- Antibodies are linked to immune effector functions by the Fc fragment, which is capable of initiating complement-dependent cytotoxicity (CDC) and antibody-dependent cellular cytotoxicity (ADCC).



Source: Nature Reviews Drug Discovery

Backgrounder – How Do Therapeutic Antibodies Work?

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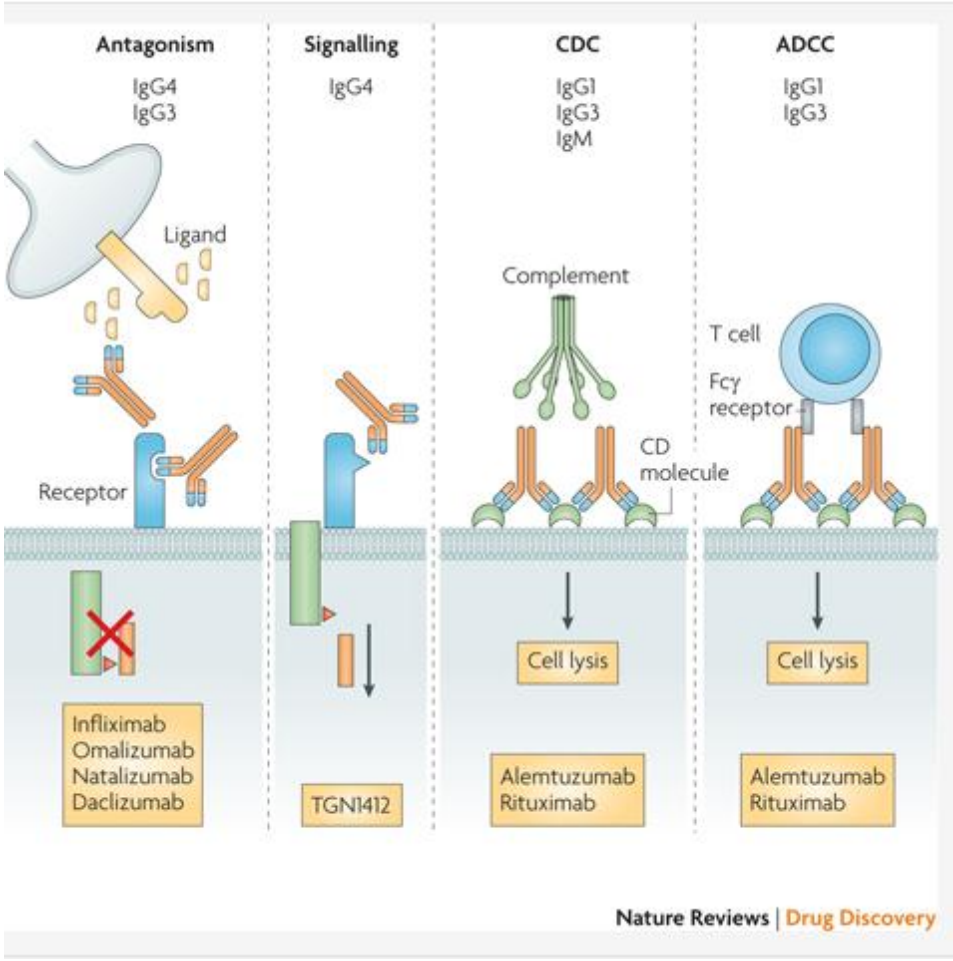
1) **Interference with cellular signaling**. Binding of the antibody to either the receptor or its ligand can prevent ligands from activating their cognate receptors, thus blocking cellular signaling. Binding of cell surface receptors by antibodies can also result in stimulation of signaling or in their internalization and down regulation to limit cell surface receptors that can be activated by the ligand.

2) **Complement-dependent cytotoxicity (CDC).** Binding of cell surface receptors can result in depletion of antigen-bearing cells through complement-mediated lysis and opsonization. Subclasses of IgG, especially IgG1 and IgG3, are potent activators of the classical complement pathway. The binding of IgG molecules to the cell surface leads to high-affinity binding of complement component 1q (C1q) to the Fc domain, followed by subsequent activation of downstream complement proteins resulting in tumor cell lysis.

- **Antibody-dependent cellular cytotoxicity (ADCC).** In ADCC, the Fc region of an antibody binds to Fc receptors (FcgRs) on the surface of immune effector cells such as natural killers and macrophages, leading to the phagocytosis or lysis of the targeted cells. Following tumor cell lysis, antigen-presenting cells can present tumor-derived peptides on MHC class II molecules and promote CD4+ T cell activation.



MGNX has Solid IP on its Platform and on Specific Antibodies

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- Fc-optimization platform:
  - Covers Margetuximab and MGA271. Three issued patents and four pending patent applications, if issued, will expire between 2024 and 2030.
  - Margetuximab: One issued US patent (expires in 2025) and one pending US patent application (expires in 2029)
  - MGA271: Two pending U.S. patent applications (expires in 2031)
- DART Platform:
  - Covers all DARTs. Seven pending DART patents (expires between 2026 and 2032)
  - MGD006. One US pending provisional patent application (expires in 2034)
  - MGD010. Four pending US patent applications (expires between 2022 and 2034)
- Cancer Stem-like Cell Platform.
  - One issued US patent that will expire in 2028

Source: SEC Filings

Teplizumab (Anti-CD3)

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- **Teplizumab failed in Phase III and MGNX is currently not developing the product**. Teplizumab is a humanized, anti-CD3 monoclonal antibody developed by MGNX for the treatment of Type 1 Diabetes, or T1D. Results of Protégé trial, a Phase III clinical study of teplizumab in T1D, were published in The Lancet in June, 2011. The primary clinical endpoint of this trial, a composite of glycated hemoglobin, or HbA1c, and insulin usage, was not met.
- **MGNX believes teplizumab could be outlicensed for potential future development**. Although the Protégé trial did not meet its primary clinical endpoint, an exploratory, post-hoc analysis suggested that teplizumab may preserve insulin production by beta cells in the pancreas, as measured by C-peptide, and increase the percentage of patients requiring very low doses of insulin compared to those on placebo.
- **Teplizumab is currently being evaluated in an Investigator-Sponsored Phase II clinical trial**, called At Risk, for the prevention or delay of onset of T1D in patients determined to be at very high risk for developing the disease. This clinical trial is being sponsored by the National Institute of Diabetes and Digestive and Kidney Diseases, or NIDDK.
- **In 2007, MGNX entered into a collaboration with LLY (OP) which was terminated in 2011.** MGNX is actively seeking a collaborator for further development of teplizumab.

Experienced Management Team

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- Scott Koenig (CEO since 2001)
  - Previously Senior Vice President of Research at MedImmune, Inc; participated in selection and maturation of the company's product pipeline for 11 years
- James Karrels (CFO since 2008)
  - Previously at JAZZ as Executive Director of Finance; involved in JAZZ IPO process in 2007
- Ezio Bonvini (SVP Research sine 2003)
  - 24 years of research, regulatory and clinical trial design experience spent at the National Cancer Institute, the National Institutes of Health, and more recently at the Food and Drug Administration (FDA) in the Center for Biologics Evaluation and Research (CBER)
- Kathryn Stein (SVP Product Development & Regulatory Affairs since 2002)
  - Previously Director, Division of Monoclonal Antibodies in the Office of Therapeutics Research and Review at CBER; 22 years FDA experience
- Jon Wigginton (SVP Clinical Development since 2013)
  - Previously Head of Immuno-Oncology, Early Clinical Research and Discovery at BMY; led early clinical development of the BMS Immuno-oncology portfolio including anti-PD-1 and anti-PD-L1

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MODEL

Near-Term Catalysts

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Near-term catalysts (chronologically):

- MGD006 preclinical data at ASH
- MGD006, 007 INDs in 1H14 ($10MM milestone payment)
- MGA271 Servier opt-in in 1H14 ($30MM milestone payment)
- MGA271 Phase I data, mid-2014
- MGD006 Servier opt-in in 2014 ($15MM milestone payment)
- Margetuximab Phase II data in Mbc, late 2014
- MGA271 dose-expansion data, specific tumor types
- MGD007 Servier opt-in in 2015 ($15MM milestone payment)
- Potential disclosure of additional partnered compounds (Gilead, Boehringer Ingelheim) and receipt of additional milestones (expect $35MM additional milestones until 2015)
- Potential additional DART partnerships

MGNX Model

MGNX P&L	2011A	2012A	1Q13A	2Q13A	3Q13E	4Q13E	2013	2014	2015
Collaborative research revenue	47.1	59.6	10.1	11.8	20.0	10.0	51.9	43.0	48.0
Grant revenue	10.2	4.2	0.5	0.5	0.5	0.5	1.9	1.0	-
Royalties	-	-	-	-	-	-	-	-	-
Product sales	-	-	-	-	-	-	-	-	-
Total Revenue	**57.2**	**63.8**	**10.6**	**12.3**	**20.5**	**10.5**	**53.8**	**44.0**	**48.0**
COGS	-	-	-	-	-	-	-	-	-
R&D	41.1	45.4	10.1	11.1	12.0	13.0	46.1	62.0	78.0
SG&A	10.9	10.2	3.8	1.5	4.0	6.0	15.3	20.0	25.0
Operating expenses	52.0	55.6	13.9	12.6	16.0	19.0	61.5	82.0	103.0
Operating income (expense)	5.2	8.2	(3.3)	(0.3)	4.5	(8.5)	(7.7)	(38.0)	(55.0)
Total Other income (expense)	1.5	0.2	(0.0)	(0.0)	-	-	(0.1)	-	-
EBT	6.7	8.4	(3.3)	(0.3)	4.5	(8.5)	(7.7)	(38.0)	(55.0)
Tax expense (income)	-	-	-	-	-	-	-	-	-
Net income	**6.7**	**8.4**	**(3.3)**	**(0.3)**	**4.5**	**(8.5)**	**(7.7)**	**(38.0)**	**(55.0)**
GAAP EPS	6.55	7.72	(2.80)	(0.29)	2.19	(0.34)	(1.06)	(1.53)	(2.22)
Common shares outstanding	1.0	1.1	1.2	1.2	2.0	24.8	7.3	24.8	24.8

BS & CFS	2011A	2012A	1Q13A	2Q13A	3Q13E	4Q13E	2013	2014	2015
Cash & equivalents	55.2	47.7	43.5	33.8	28.5	98.0	98.0	65.6	17.8
Debt	-	-	-	-	-	-	-	-	-

Change in Cash	18.3	(7.5)	(4.2)	(9.7)	(5.2)	64.3	45.1	(32.4)	(47.8)
Cash from operations	**6.8**	**(6.6)**	**(3.8)**	**(9.9)**	**(4.7)**	**(17.7)**	**(36.3)**	**(30.4)**	**(45.8)**
Net income (loss)	6.7	8.4	(3.3)	(0.3)	4.5	(8.5)	(7.7)	(38.0)	(55.0)
Share based comp	2.3	0.8	0.1	0.1	0.5	0.5	1.3	6.6	8.2
D&A	1.1	1.0	0.3	0.3	0.3	0.3	1.1	1.0	1.0
Other (Change in WC)	(3.5)	(16.7)	(0.9)	(10.0)	(10.0)	(10.0)	(30.9)	-	-
Cash from investing	**(0.5)**	**(0.9)**	**(0.4)**	**(0.5)**	**(0.5)**	**(0.5)**	**(1.9)**	**(2.0)**	**(2.0)**
CapEx	(0.5)	(0.9)	(0.4)	(0.5)	(0.5)	(0.5)	(1.9)	(2.0)	(2.0)
Acquisitions	-	-	-	-	-	-	-	-	-
Other	-	-	-	-	-	-	-	-	-
Cash from financing	**12.1**	**0.0**	**0.1**	**0.7**	**-**	**82.5**	**83.2**	**-**	**-**
Equity issue (buyback)	12.1	0.0	0.1	0.7	-	82.5	83.2	-	-
Debt issue (principal payment)	-	-	-	-	-	-	-	-	-
Other	-	-	-	-	-	-	-	-	-

Source: Leerink Swann Estimates and Company Filings

Margetuximab Model

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Breast Cancer	2012E	2013E	2014E	2015E	2016E	2017E	2018E	2019E	2020E	2021E	2022E	2023E	2024E	2025E	2026E	2027E	2028E	2029E	2030E
US mBC sales								35	145	286	314	322	331	340	349	358	368	378	388
EU mBC sales								-	38	168	336	372	382	392	403	413	424	435	447
EU mBC Royalty								-	6	25	50	56	57	59	60	62	64	65	67
RR								*15%*	*15%*	*15%*	*15%*	*15%*	*15%*	*15%*	*15%*	*15%*	*15%*	*15%*	*15%*
COGS								5	22	43	47	48	50	51	52	54	55	57	58
Milestone Payments	-	-	-	15	-	-	25	25	25	-	-	-	-	-	-	-	-	-	-
R&D		6	8	20	20	20	20	10	-	-	-	-	-	-	-	-	-	-	-
SG&A								60	62	64	66	68	70	72	74	76	78	81	83
Operating income		(6)	(8)	(5)	(20)	(20)	5	(16)	92	204	252	262	269	276	283	291	298	306	314
Tax											71	73	75	77	79	81	83	86	88
Tax rate											*28%*	*28%*	*28%*	*28%*	*28%*	*28%*	*28%*	*28%*	*28%*
Net Income		**(6)**	**(8)**	**(5)**	**(20)**	**(20)**	**5**	**(16)**	**92**	**204**	**181**	**189**	**194**	**199**	**204**	**209**	**215**	**220**	**226**
POS		*100%*	*100%*	*70%*	*70%*	*70%*	*70%*	*25%*	*25%*	*25%*	*25%*	*25%*	*25%*	*25%*	*25%*	*25%*	*25%*	*25%*	*25%*
P/W Net Income		**(6)**	**(8)**	**(4)**	**(14)**	**(14)**	**4**	**(4)**	**23**	**51**	**45**	**47**	**48**	**50**	**51**	**52**	**54**	**55**	**56**

Gastric Cancer	2012E	2013E	2014E	2015E	2016E	2017E	2018E	2019E	2020E	2021E	2022E	2023E	2024E	2025E	2026E	2027E	2028E	2029E	2030E
US mGC sales								40	83	138	144	150	157	163	170	178	185	193	201
EU mGC sales								12	64	108	113	118	123	129	135	141	148	154	161
EU mBC Royalty								2	10	16	17	18	19	19	20	21	22	23	24
RR								*15%*	*15%*	*15%*	*15%*	*15%*	*15%*	*15%*	*15%*	*15%*	*15%*	*15%*	*15%*
COGS								6	12	21	22	23	24	25	26	27	28	29	30
Milestone Payments	-	-	-	15	-	-	25	25	25	-	-	-	-	-	-	-	-	-	-
R&D		6	10	18	18	18	10	5	-	-	-	-	-	-	-	-	-	-	-
SG&A								30	31	32	33	34	35	36	37	38	39	40	42
Operating income		(6)	(10)	(3)	(18)	(18)	15	26	74	102	107	112	117	122	128	134	140	147	154
Tax											30	31	33	34	36	38	39	41	43
Tax rate											*28%*	*28%*	*28%*	*28%*	*28%*	*28%*	*28%*	*28%*	*28%*
Net Income		**(6)**	**(10)**	**(3)**	**(18)**	**(18)**	**15**	**26**	**74**	**102**	**77**	**80**	**84**	**88**	**92**	**97**	**101**	**106**	**111**
POS		*100%*	*100%*	*70%*	*70%*	*70%*	*70%*	*25%*	*25%*	*25%*	*25%*	*25%*	*25%*	*25%*	*25%*	*25%*	*25%*	*25%*	*25%*
P/W Net Income		**(6)**	**(10)**	**(2)**	**(13)**	**(13)**	**11**	**6**	**19**	**25**	**19**	**20**	**21**	**22**	**23**	**24**	**25**	**26**	**28**

Assumptions:

- 25% probability of success (POS) in Her2 2+ mBC and 3rd line mGC
- Launch in 2019 in both indications
- Priced at $9,800/cycle at launch
- ROW partnership in 2015 after positive Phase IIa data, 15% ex-US RR

Sources of upside:

- Increased POS driven by positive clinical data (e.g. Phase IIa in late 2014)
- Additional indications (e.g., bladder cancer, CRC)
- Pricing

Source: Leerink Swann estimates

MGA271 Model

Valuation MGA271	2013	2014	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024	2025	2026	2027	2028	2029	2030	2031	2032
US Sales									67	374	708	1,073	1,657	1,732	1,811	1,894	1,980	2,070	2,164	2,262
ROW Royalty										6	34	69	105	174	182	204	213	223	233	243
RR										*12.0%*	*12.0%*	*13.0%*	*13.0%*	*14.0%*	*14.0%*	*15.0%*	*15.0%*	*15.0%*	*15.0%*	*15.0%*
COGS									10	56	106	161	249	260	272	284	297	310	325	339
Milestone Payments	10	30	10		20				25	25										
R&D	8	10	20	20	40	40	40	40	40	42	44	46	49	51	54	56	59	62	65	68
SG&A									100	175	212	322	497	520	543	568	594	621	649	679
Operating income		20	(10)	(20)	(20)	(40)	(40)	(40)	(58)	132	379	613	967	1,076	1,124	1,189	1,243	1,299	1,358	1,419
Tax										37	106	172	271	301	315	333	348	364	380	397
Tax rate										*28%*	*28%*	*28%*	*28%*	*28%*	*28%*	*28%*	*28%*	*28%*	*28%*	*28%*
Net Income		**20**	**(10)**	**(20)**	**(20)**	**(40)**	**(40)**	**(40)**	**(58)**	**95**	**273**	**441**	**696**	**775**	**810**	**856**	**895**	**935**	**978**	**1,022**
POS		*80%*	*80%*	*80%*	*70%*	*70%*	*70%*	*50%*	*12%*	*12%*	*12%*	*12%*	*12%*	*12%*	*12%*	*12%*	*12%*	*12%*	*12%*	*12%*
P/W Net Income		**16**	**(8)**	**(16)**	**(14)**	**(28)**	**(28)**	**(20)**	**(7)**	**11**	**33**	**53**	**84**	**93**	**97**	**103**	**107**	**112**	**117**	**123**

Source: Leerink Swann estimates

Assumptions:

- 12% probability of success (POS) in prostate cancer (HRPC) and metastatic melanoma
- Servier Opt-in in 2014 at 80% POS
- Launch in 2021
- Priced at $100k/course (similar to Yervoy [$120k/course])
- 12-15% tired royalty rate on EU sales

Sources of upside:

- Increased POS driven by positive Phase Ib extension phase data in 2014
- Efficacy in additional indications
- Partnerships for additional geographies (e.g. east Asia)
- Pricing

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Disclosures Appendix

Analyst Certification

I, Michael Schmidt, Ph.D., certify that the views expressed in this report accurately reflect my views and that no part of my compensation was, is, or will be directly related to the specific recommendation or views contained in this report.

Distribution of Ratings/Investment Banking Services (IB) as of 09/30/13				
			IB Serv./Past 12 Mos.	
Rating	Count	Percent	Count	Percent
BUY [OP]	111	64.90	27	24.00
HOLD [MP]	60	35.10	0	0.00
SELL [UP]	0	0.00	0	0.00

Explanation of Ratings

Outperform (Buy): We expect this stock to outperform its benchmark over the next 12 months.

Market Perform (Hold/Neutral): We expect this stock to perform in line with its benchmark over the next 12 months.

Underperform (Sell): We expect this stock to underperform its benchmark over the next 12 months.The degree of outperformance or underperformance required to warrant an Outperform or an Underperform rating should be commensurate with the risk profile of the company.

For the purposes of these definitions the relevant benchmark will be the S&P 600® Health Care Index for issuers with a market capitalization of less than $2 billion and the S&P 500® Health Care Index for issuers with a market capitalization over $2 billion.

Important Disclosures

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