May 8, 2012

HEALTHCARE/BIOTECHNOLOGY

Stock Rating:

OUTPERFORM

12-18 mo. Price Target	$12.00
MACK - NASDAQ	$7.50
3-5 Yr. EPS Gr. Rate	NA
52-Wk Range	$9.00-$5.81
Shares Outstanding	92.3M
Float	81.3M
Market Capitalization	$693.0M
Avg. Daily Trading Volume	NA
Dividend/Div Yield	NA/NM
Book Value	NA
Fiscal Year Ends	Dec
2012E ROE	NA
LT Debt	NA
Preferred	NA
Common Equity	NA
Convertible Available	No

52-week range as of 3/29/12.

EPS Diluted	Q1	Q2	Q3	Q4	Year	Mult.
2010E	--	--	--	--	(4.60)	NM
2011E	(1.21)	(2.60)	(1.65)	(1.53)	(6.98)	NM
2012E	(0.21)	(0.22)	(0.24)	(0.25)	(0.93)	NM
2013E	--	--	--	--	(0.94)	NM

Merrimack Pharmaceuticals

Initiating at Outperform; Excellent Opportunities in Broad Cancer Pipeline

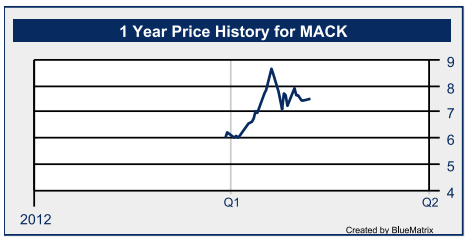
SUMMARY

We are initiating coverage of MACK at Outperform, with a $12 PT. With a novel network biology platform, MACK has developed a broad pipeline of antibody-based and nanotherapeutic cancer therapies. We view MM-121, an ErbB3 antibody, as the most promising. We believe ErbB3 inhibition has applicability in several cancers, and MACK, with partner SNY, is evaluating MM-121 in a comprehensive ph.II program. MM-398, a liposomal irinotecan, has shown strong ph.II pancreatic cancer results, and we see a good probability of ph.III success, mid-'13. Additionally, MACK's earlier antibody-based candidates, MM-111/MM-302/MM-151, address blockbuster markets. We believe MACK is an attractive long-term value based on the company's pipeline/platform, and we would position in the stock ahead of key '121/'398 data in '13.

KEY POINTS

- **'121 could address several cancers.** We believe there is increasing evidence of ErbB3's role in cell growth/signaling, and we believe '121 has promise in lung/breast/ovarian cancers. SNY/MACK currently has '121 in 4 large ph.II trials, increasing '121's probability of success. Based on '121's MoA/ph.I results, we are optimistic regarding ph.II NSCLC/breast cancer results in '13.
- **Looking toward '398 ph.III results in pancreatic cancer.** In a ph.II refractory pancreatic cancer study, the drug demonstrated 5-6mos survival vs. the ~2mos seen historically. These are strong results, given the poor prognosis/lack of treatment options in this setting. Consequently, we see a good probability of positive ph.III refractory pancreatic cancer results in mid-13.
- **Significant opportunities in the earlier pipeline.** We believe '111 (bispecific HER2/ErbB3 antibody) and '302 (liposomal doxorubicin attached to HER2 antibodies) have compelling MoAs and could improve on current therapies. Although '111/'302 are in ph.I, we believe these compounds would take share in the multi-billion dollar HER2+ breast cancer market if successful longer term.
- **Sum-of-the-parts NPV supports $12 PT.** Our valuation is based on '121/'398/'111/'302 sales and technology value. To reflect clinical risk, we employ a 15% discount rate and 25-40% prob. of success for each program. '151, a powerful EGFR inhibitor, and preclinical candidates represent upside to our valuation.

Stock Price Performance



Company Description

Merrimack is a biopharmaceutical company engaged in the discovery and development of novel cancer therapeutics.

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Investment Overview

We are initiating coverage on Merrimack Pharmaceuticals with an Outperform rating and a $12 price target. Merrimack is engaged in discovery and development of cancer therapeutics, based on insights from the company's novel network biology platform. The company currently has an impressive clinical pipeline consisting of five antibody-based and nanotherapeutic therapies. We believe Merrimack's most promising candidate is MM-121, an antibody that targets a somewhat untested, but highly promising protein in the EGFR/HER2 signaling axis, ErbB3. We believe MM-121 has potential in several major tumor types, and consistent with this, Merrimack with partner Sanofi Aventis is developing the drug in a comprehensive phase II program in breast, lung, and ovarian cancer. MM-398 is a nanoencapsulated irinotecan in phase III development for refractory pancreatic cancer. We believe the drug has significant promise in this indication, based on strong phase II results. Merrimack's earlier stage clinical pipeline includes MM-111, MM-302, and MM-151, which we believe have longer term blockbuster potential. Based on our expectations for favorable results from key clinical trials for MM-121 and MM-398 in 2013, along with maturation of Merrimack's earlier stage candidates, we believe the stock is a compelling long-term investment.

MM-121 has promise in multiple major tumor types. MM-121's target, ErbB3, has been implicated in tumorgenesis and may be critical for cancer cells to escape EGFR and HER2 inhibition. ErbB3 is an emerging target, as there has been, until recently, much less focus on ErbB3 relative to EGFR/HER2. However, we note a number of major oncology companies are now focusing on ErbB3, and we believe MM-121 is one of the most advanced ErbB3 inhibitors in development. Based on the potentially pivotal role of ErbB3 in modulating EGFR/HER2 signaling, we believe MM-121 may be effective in a wide range of cancers, particularly in combination with EGFR and HER2 inhibitors. MM-121 may also have potential in difficult to treat subpopulations, given ErbB3's potential role in tumor escape from other targeted therapies. Additionally, Merrimack's MM-121 development and commercialization partnership with Sanofi both highlight the drug's strong scientific rationale and ensure aggressive development of the drug. Importantly, MM-121 has demonstrated encouraging phase I activity in breast, lung, and ovarian cancer, and the drug is currently in large, controlled phase II trials in combination with exemestane/paclitaxel, erlotinib, and paclitaxel in these tumor types respectively. Merrimack expects the first phase II readouts for NSCLC in 1H13 and breast cancer in 2H13, and we see a good probability of success, based on early phase I results and MM-151's novel mechanism of action.

MM-398 has significant promise in pancreatic/colorectal cancer. Using proprietary nanoencapsulation delivery technology, Merrimack created MM-398, a liposomal irinotecan, which preclinical modeling suggests has improved therapeutic properties vs. unmodified irinotecan. Importantly, we believe MM-398 has potential in pancreatic cancer, a disease with poor prognosis and few therapeutic options. In a phase II refractory pancreatic cancer trial, MM-398 showed an impressive 5-6 month survival compared to the approximate 2 month survival seen historically in this setting. Although there is risk in translating phase II to phase III results in pancreatic cancer, we see a good probability of a positive phase III outcome in mid-2013, based on the substantial magnitude of benefit seen in the phase II results. Additionally, we believe MM-398 has potential in colorectal cancer, given irinotecan's proven efficacy in this indication and early phase I response rates. Phase II results in second-line colorectal cancer are expected in mid-2013.

MM-111 and MM-302, both in combination and individually, could play an important role in HER2+ breast cancer. MM-111 is a bispecific antibody which inhibits HER2 and ErbB3, disrupting the HER2/ ErbB3/heregulin complex. Notably, we see a strong rationale for this mechanism, given the recent phase III success with Roche's pertuzumab, which prevents HER2/ErbB3 dimerization. We believe MM-111 has the greatest potential as an add-on therapy to HER2 inhibitors, as MM-111 may be able to enhance anti-tumor efficacy and delay development of resistance. Merrimack is exploring MM-111 with several regimens with different HER2 inhibitors and chemotherapies in a phase I study. Although earlier combination results are somewhat difficult to interpret, the combination of Herceptin, paclitaxel and MM-111 appears to have compelling activity. We look forward to additional phase I results later this year. Merrimack is also developing MM-302, a nanotherapeutic encapsulation of doxorubicin conjugated to anti-HER2 antibodies. Although data is limited for MM-302, HER2 is clearly a well-validated target, and antibody

drug conjugates have made significant clinical progress, including Roche/Immunogen's T-DM1 and Takeda/Seattle Genetic's Adcetris. Notably, Merrimack plans to develop MM-111 with MM-302, which we believe has the potential to be a powerful treatment combination.

Potential long-term upside from Merrimack's earlier pipeline. Notably, we believe MM-151, which just entered phase I testing, has a compelling mechanistic rationale. The compound is a polyclonal therapeutic consisting of a mixture of three monoclonal antibodies to distinct epitopes of EGFR. EGFR is a clearly validated cancer target, and commercially available EGFR inhibitors have achieved great clinical and commercial success. MM-151 blocks high affinity and low affinity EGFR ligands, and based on this, the compound may more completely inhibit EGFR signaling, offering improved efficacy and a higher barrier to resistance compared to current EGFR inhibitors. Merrimack expects phase I data in 2H13, and we will monitor these results closely as we believe MM-151 has substantial longer term promise. Merrimack also has several preclinical antibody-based therapies including multi-specific antibodies, MM-141 (IGFR and ErbB3) and MM-131 (undisclosed targets), and an antibody targeted nanotherapeutic (undisclosed target). We note these compounds are very early in development. However, MM-151 and the preclinical candidates are not reflected in our valuation, and future clinical success with these candidates would lead to upside to our estimates.

Merrimack has an innovative network biology-based drug discovery platform. Traditional drug discovery typically focuses on developing drugs that target an individual protein that appears dysregulated in diseased vs. healthy tissue. Although this approach is common, it results in a high failure rate as cancer progression is rarely attributable to a single protein. In contrast, using network biology, Merrimack seeks to understand total biologic systems by investigating the relationships between several system components. This analysis is achieved through complex tools including molecular databases, high-throughput data collection, and computational modeling. Based on these proprietary analyses, Merrimack can build computational models of biological systems to guide drug discovery and development. We believe MM-121 is a prime example of a drug discovered using network biology. Many cancers escape EGFR and HER2 inhibition, even though these proteins play a critical role in disease progression. Despite a previously poor understanding of the biological role of ErbB3, Merrimack's models identified this protein as a potentially pivotal regulator in EGFR/HER2 signaling axis and, therefore, a compelling drug target.

MM-121, MM-398, and earlier pipeline candidates have significant market potential. We believe MM-121 has substantial potential in several indications, particularly breast and lung cancer. With Sanofi's backing, we see a good probability of MM-121's success in at least one of these major indications. Importantly, Sanofi bears all the development/commercial execution risk for MM-121, and Merrimack receives royalty payments and retains an option to co-promote in the US. We conservatively model MM-121 royalties to Merrimack of approximately $100M, $50M, and $35M only in the refractory settings for ER/PR+ breast, non-small cell lung, and ovarian cancer, respectively. However, we believe the drug has potential in earlier line breast, lung, and ovarian cancer and has promise beyond these indications. MM-398 is Merrimack's nearest term opportunity. Given strong phase II data in pancreatic cancer, we see a higher probability of success for MM-398 vs. other programs. We believe the drug can achieve sales of $290M in refractory pancreatic cancer, and longer term, sales of $885M in front-line pancreatic cancer and $600M in refractory colorectal cancer. We view MM-111 and MM-302 as high-risk/reward programs. We believe each of these drugs have blockbuster potential in HER2+ breast cancer, but given the early stage of these compounds, we have assigned modest probabilities of success. Notably, we currently do not model sales for MM-151 and Merrimack's preclinical candidates.

Merrimack's leadership team is experienced in oncology drug development. Merrimack's CEO, Robert Mulroy, has led the company since 1999. Mr. Mulroy has a breadth of management consulting experience in the biotechnology industry and has advised multiple biotechnology start-ups. Mr. Mulroy is joined by strong science leadership, including Merrimack's Chief Scientific Officer, Dr. Ulrik Nielsen, a co-founder of the company. Prior to Merrimack, Dr. Nielsen was engaged in research at MIT related to network biology. In addition, Merrimack's clinical programs are led by industry veterans such as Dr. Clet Niyikiza, who has held leadership roles in product development at GSK

and Lilly. Notably, Merrimack also has scientists who were previously involved in the development of pertuzumab at Roche and Tykerb at GSK.

Valuation

Our 12-18 month price target of $12 is a based on a sum-of-the-parts valuation. Our valuation is based on a 12-year discounted cash flow valuation for four of Merrimack's clinical programs: MM-121, MM-398, MM-111, MM-302 (Exhibit 1). As Merrimack's clinical candidates are fairly early in development, we utilized both a high 15% discount rate and 25-40% probability of success to reflect clinical risk for each program. Additionally, in the out years we utilized an industry average 45% EBIT margin and a conservative 30% blended US/EU tax rate. Our valuation also includes $350M technology value for the company's network biology drug development platform. However, the company may deserve higher technology value closer to $500M, based on acquisition comps of discovery-based companies.

Exhibit 1: Sum-of-the-Parts Valuation

Program	Unadjusted Peak Revs	Probability of Success	EBIT margin	Peak Cash Flow	Discount Rate	Terminal Growth	NPV per Share	Cancer Indications Included
MM-398	$1,773	*40%*	*46%*	$228	*15%*	*-3.5%*	$3.61	pancreatic (1st/2nd-line), colorectal (2nd-line)
MM-121	$180	*40%*	*83%*	$51	*15%*	*1.0%*	$2.00	NSCLC (2nd/3rd-line), ER/PR+ breast (2nd/3rd-line), ovarian (2nd/3rd-line)
MM-111	$1,144	*35%*	*46%*	$129	*15%*	*1.0%*	$1.24	HER2+ breast (alll-lines)
MM-302	$1,528	*25%*	*46%*	$176	*15%*	*1.0%*	$1.23	HER2+ breast (alll-lines)
Network Biology Platform				$350			$3.21	
Cash at YE				*$61*			$0.56	
						Price Target	**$12**	

Note: Sales and cashflow in millions USD. Source: Oppenhiemer and Co.

Pipeline Overview

Exhibit 2: Merrimack Pipeline

Program	Indication	Stage	Commercial rights
MM-121 *Anti-ErbB3 mAb*	Hormone-sensitive breast (+exemestane)	Phase II	Sanofi worldwide; Merrimack holds option to co-promote in the United States
	NSCLC (+erlotinib)	Phase II	
	Neoadjuvant HER2- breast (+paclitaxel)	Phase II	
	Platinum res/ref ovarian (+paclitaxel)	Phase II	
	HER2- breast, ovarian, gynecological (+paclitaxel)	Phase I	
	Solid tumors (+cetuximab+irinotecan)	Phase I	
	Solid tumors (+multiple anti-cancer therapies)	Phase I	
	Solid tumors (monotherapy)	Phase I	
MM-398 *Liposomal irinotecan*	Pancreatic	Phase III	Merrimack worldwide, except Taiwan
	Colorectal (+5-FU+leucov)	Phase II	
	Colorectal	Phase I	
	Gastric	Phase II	
	Glioma	Phase I	
MM-111 Anti-ErbB3/HER2 *bispecific antibody*	HER2+ cancers (+other anti-cancer therapies)	Phase II (planned)	Merrimack worldwide
	HER2+ cancers (monotherapy)	Phase I	
	HER2+ breast (+Herceptin)	Phase I	
	Multi-arm combination therapy safety trial	Phase I	
MM-302 HER2-targeted liposomal doxorubicin	HER2+ breast (monotherapy)	Phase I	Merrimack worldwide
MM-151 *anti-EGFR oligoclonal Ab*	Monotherapy	Phase I	Merrimack worldwide
MM-141 *Anti-IGF-1R/ErbB3 bispecific Ab*	Solid Tumors	Preclinical	Merrimack worldwide

Source: Company reports, Oppenheimer & Co.

MM-121 is a fully human monoclonal antibody that targets ErbB3. Research suggests that this cell surface receptor is critical for resistance to targeted chemotherapies in a number of cancer settings. MM-121 is designed to inhibit cancer growth directly, restore sensitivity to drugs to which a tumor has become resistant and delay the development of resistance by a tumor to other agents. MM-121 is partnered with Sanofi worldwide, and Merrimack holds a co-promote option in the US. MM-121 is currently in several phase II clinical trials in combination with both chemotherapies and targeted agents across several solid tumor types.

MM-398 is a liposomal formulation of irinotecan. MM-398 completed two phase II trials in pancreatic and gastric cancer. Merrimack is currently in a phase III trial for the treatment of patients with metastatic pancreatic cancer who have previously failed gemcitabine. MM-398 has orphan drug designation for the treatment of pancreatic cancer in the US and EU. MM-398 currently has potential uses in a number of other indications, including colorectal cancer, lung cancer and glioma. There are multiple ongoing phase I and phase II clinical trials of MM-398.

MM-111 is a bispecific antibody that binds to HER2 (ErbB2) and ErbB3. A bispecific antibody can bind simultaneously to two different proteins. Recent research suggests an HER2/ErbB3-containing complex can be a powerful driver of tumor growth and survival when overexpressed on cancer cells and activated. MM-111 was designed to block the HER2/ErbB3 complex from forming, thereby starving the cancer cells from what is believed to be an important driver of proliferation. MM-111 is currently in phase I clinical trials in monotherapy and combination therapy settings.

MM-302 is a liposomal formulation of doxorubicin, an approved drug for the treatment of breast cancer, with attached antibodies that target HER2 (ErbB2). MM-302 was designed to bind to HER2 overexpressing cancer cells and unload doxorubicin at the tumor site. While free doxorubicin is associated with cardiotoxicity, thereby limiting total amount given to a patient, liposomal has improved cardiac safety over the free drug. Targeting the liposomal doxorubicin particles to HER2 could enhance the efficacy of the drug by locally increasing drug levels while still preserving the improved safety profile. MM-302 is currently in phase I clinical trial in patients with advanced HER2 positive breast cancer.

MM-151 is a combination of three fully human monoclonal antibodies (oligoclonal) designed to bind to non-overlapping parts of the epidermal growth factor receptor, or EGFR (ErbB1). EGFR has been successfully targeted with small molecules and monoclonal antibodies for the treatment of lung, colon, pancreatic and head & neck cancers, to name a few. MM-151 is currently in a phase I clinical trial in patients with solid tumors.

MM-141 is a bispecific antibody that co-targets ErbB3 and IGF-1R. MM-141 blocks IGF-1, IGF-2, and HRG binding to IGF1R and ErbB3, and causes downregulation of these receptors, their phosphorylation and downstream activation of the PI3K/Akt pathway. Importantly, MM-141 does not bind the insulin receptor, thus reducing the risk of metabolic side effects.

Pipeline Milestones

Exhibit 3: Expected Clinical Results

Program	Event	Timing
MM-398	Phase I data in CRC	1H12
	Complete enrollment in Phase III trial in pancreatic cancer	1H13
	Phase III results (pancreatic cancer)	mid-13
	Complete enrollment in Phase II in CRC	1H13
	Phase II results in CRC	mid-13
MM-121	Phase I data in combo with Tarceva in NSCLC	1H12
	Phase I data in combo w/ pac	2H12
	Complete enrollment for Phase II trial in ER/PR+ mBC	2H12
	Complete enrollment in Groups A&C Phase II trial in NSCLC	2H12
	Phase II results from Groups A&C in NSCLC	mid-13
	Complete enrollment in neoadj. ER+/HER2- Phase II	1H13
	Phase II data in neoadj. ER+/HER2- mBC	2H13
	Complete enrollment in Group B Phase II trial in NSCLC	2H12
	Complete enrollment Phase II trial in ovarian cancer	2H13
	Complete enrollment in TNBC Phase II trial	2H13
MM-111	Initiate evaluation in I-SPY 2 Phase II study	1H12
	Initiate Phase II in HER2+ mBC	1H12
	Multi-arm Phase I data	2H12
MM-151	Initiate Phase I trial in solid tumors	1H12
	Phase I data	2H13
	Phase II initiation	2H13
MM-141	Initiate Phase I	2H12
	Phase I data	2H13
MM-302	Phase I data	1H13
	Phase II initiation	1H13
MM-310	Phase I initiation	1H13
MM131	Phase I initiation	2H13

Source: Company reports, Oppenheimer & Co.

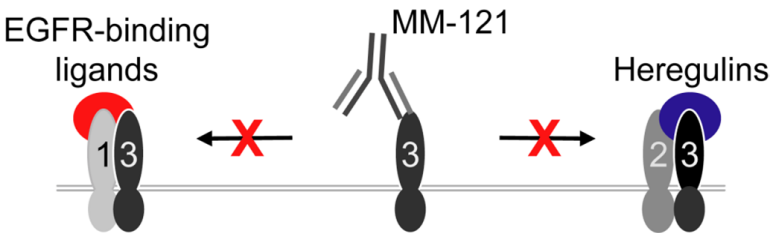
MM-121

MM-121 (SAR 256212) is a fully human monoclonal antibody against the ErbB3 cell surface receptor that is in several phase I and phase II clinical trials in combination with chemotherapies and other targeted therapies. The clinical trials include a companion diagnostic (DX-121) designed to determine whether a tumor is dependent on ErbB3 signaling and amenable to treatment with MM-121. MM-121 is part of a worldwide development and commercialization collaboration between Merrimack and Sanofi.

Design of MM-121

ErbB3 was discovered as a potential anticancer target through Merrimack's network biology platform for its role in ligand-induced activation of the ErbB pathway, which can lead to tumor growth and survival in cells resistant to inhibition with approved inhibitors of EGFR (Tarceva/erlotinib, Erbitux/cetuximab, Vectibix/panitumumab) and HER2 (Herceptin/trastuzumab and Tykerb/lapatinib). While EGFR and HER2 are known to get activated via ligand-dependent homodimerization, ErbB3 forms heterodimers with EGFR and HER2 when bound by heregulin, thereby providing an alternative way of activating these two growth factor receptors. Therefore, ErbB3 has been suspected to drive EGFR and HER2 activation in tumor cells resistant to EGFR and HER2 inhibitors. Since ErbB3 does not have a kinase domain whose activity could be blocked with small molecules, the scientists at Merrimack developed a human antibody that blocks the binding of ErbB3's activating ligand heregulin, thereby inhibiting ErbB3-dependent growth and survival signaling via heterodimerization with EGFR and HER2. Consequently, MM-121 was designed to block heterodimerization of ErbB3 with the other ErbB receptors. When bound by MM-121, ErbB3 gets internalized into the tumor cell, which renders it unavailable for signaling that would otherwise drive cancer growth and survival.

Exhibit 4: MM-121 blocks heterodimerization between ErbB3 and other ErbB family members



Source: Merrimack Inc.

ErbB3 has been shown to be overexpressed in a high portion of patients with lung, prostate, breast, colorectal, ovarian and pancreatic cancers. Preclinical data shows that MM-121 has synergistic/additive activity with kinase inhibitors and traditional chemotherapy. Furthermore, tissue samples from cancer patients resistant to kinase inhibitors have been shown to overexpress ErbB3. Finally, overexpression of heregulins, the natural ligands of the ErbB3 heterodimers, has been associated with Herceptin resistance in breast cancer models. ErbB3 overexpression is also believed to be associated with the development of resistance to EGFR kinase inhibitors, in particular Tarceva (approved for the treatment of lung and pancreatic cancers), Erbitux (approved for the treatment of colorectal and head & neck cancers), and Herceptin/Tykerb (both approved for the treatment of HER2+ breast cancer). Consequently, MM-121 may be able to prevent emergence or overcome resistance to the EGFR and HER2 inhibitors in these tumor types.

ErbB3 could also be a key target in HER2 negative cancers. Inhibition of HER2 has been shown to be a valid target in HER2+ cancers. However, incomplete inhibition of HER2 could still leave sufficiently high HER2 levels available for heterodimerization with ErbB3, which would form the more potent activator of downstream pathways and, consequently, lead to tumor progression despite HER2 inhibition by HER2 targeting therapies. In tumors that do not overexpress HER2, HER2 protein levels could still be sufficient for heterodimerization with the less abundant ErbB3. These tumors would potentially have the more potent HER2/ErbB3 heterodimer as the main/sole driver of

tumor growth despite normal levels of each protein. Furthermore, since tumors that don't overexpress HER2 have not responded to anti-HER2 therapies, we believe that HER2 homodimers are not significant drivers of tumor growth in HER2- cancers, and that the more potent ErbB3/HER2 heterodimers instead, whose formation is not blocked by these HER2 targeted therapies, may be responsible for tumor growth. As a result, Merrimack and Sanofi are evaluating MM-121 in HER2- cancers.

Clear rationale for ErbB3 Inhibition in EGFR-driven cancers. We believe ErbB3 is a naturally complementary target to EGFR in cancer settings where EGFR is mutationally activated. Even in cases where EGFR is not mutationally activated, we believe ErbB3 inhibition could be effective. Since EGFR is usually expressed at higher levels than ErbB3, incomplete inhibition of EGFR could leave EGFR proteins available for EGFR/ErbB3 heterodimer formation. Similar to HER2+ cancers, ErbB3 could provide an escape mechanism for tumor growth in EGFR driven tumors in the presence of EGFR targeting therapies or in tumors that are resistant to EGFR inhibition. Therefore, inhibiting the formation of EGFR/ErbB3 heterodimers with MM-121 could starve EGFR-dependent tumors of an essential growth signal. For these reasons, Merrimack and Sanofi are developing MM-121, in EGFR-dependent cancers.

MM-121 Clinical Development Program

Encouraging phase I experience with MM-121. Sanofi and Merrimack have initiated four monotherapy or combination studies (Exhibit 5). Early response rates from the combination trials in HER2+ cancers and non-small cell cancer were encouraging (Exhibit 6). Response rates may be better in later trials, as the phase I trials also evaluated lower doses of MM-121. MM-121 has shown generally favorable safety/tolerability as a monotherapy and in combination regimes. In the monotherapy trial, MM-121 was given with a higher loading dose and subsequent weekly doses. Grade 3 and grade 4 AEs observed in the dose escalation and expansion phases of this trial were fairly low and included 10.5% (4/38) fatigue, 2.6%(1/38) nausea, and 2.6%(1/38) vomiting.

Exhibit 5: Phase I trials for MM-121

Trial Identifier	N	Type of Cancer	Combo with	Start
NCT01447225	48	Solid Tumors	Gemcitabine, or Carboplatin, or Pemetrexed, or Cabazitaxel	10/2011
NCT01451632	45	EGFR-dependent	Irinotecan+Cetuximab	10/2011
NCT01436565	56	Solid Tumors	Monotherapy	11/2011
NCT01209195	24	Ovarian; Fallopian Tube; Peritoneal; HER2- breast	Paclitaxel (Taxol)	11/2010

Source: Clinicaltrials.gov and Oppenheimer & Co.

Exhibit 6: Response rates in Phase I studies with MM-121

Combination with:	Indication	N	ORR	DCR
Paclitaxel	HER2- breast, ovarian, other gynecological cancers	23	**35%**	**74%**
Erlotinib	NSCLC	32	3%	47%
Monotherapy	Solid tumors	30	0%	29%

Source: Company reports, Oppenheimer & Co.

Comprehensive phase II program under way in several indications. Favorable phase I results led to the initiation of four large controlled phase II studies evaluating MM-121 in combination with other therapies in breast, non-small lung, and ovarian cancer. Merrimack expects phase II NSCLC in 1H13 and phase II ER+/HER2- breast cancer in 2H13.

Exhibit 7: Controlled Phase II trials for MM-121

Trial Identifier	N	Type of Cancer	Combo with	Start
NCT00994123	260	NSCLC	Erlotinib	2/2010
NCT01151046	130	HER2- Breast	Exemestane	6/2010
NCT01421472	200	ER+/HER2- Breast	Paclitaxel	8/2011
NCT01447706	210	Epithelial Ovarian; Fallopian Tube; Peritoneal	Paclitaxel	10/2011

Source: Clinicaltrials.gov and Oppenheimer & Co.

Phase I/II trial for MM-121 in combination with erlotinib in NSCLC. The Phase I dose-escalation portion of the trial determined the optimal dose and dosing schedule for the combination. Among the 32 enrolled patients in the phase I portion of the trial, there was one partial response (PR) and 14 patients with stable disease (SD). The most common toxicities observed of any grade were diarrhea (82%), rash (64%) and fatigue (64%). The Phase II placebo-controlled portion of the trial is looking at the effect of MM-121 on progression-free survival (PFS) in 229 patients in three subgroups:

- **Group A**: patients without EGFR activating mutations, whose cancer has recurred or progressed following at least one chemotherapy-containing regimen and who have not received prior EGFR targeted therapy;
- **Group B**: patients with EGFR activating mutations and who have not received prior EGFR targeted therapy;
- **Group C**: patients whose tumors had previously responded to EGFR targeted therapy and subsequently acquired resistance.

An update of the Phase II portion of the trial will be presented on 6/2/12 at ASCO (Abstract #7556, Poster Board: #46F).

Phase II trial for MM-121 in combination with exemestane for hormone-sensitive breast cancer. MM-121 is also being tested in a randomized, double-blind phase II trial in combination with exemestane (Aromasin) to exemestane alone in 130 postmenopausal women with metastatic hormone-sensitive HER2- breast cancer who have previously failed treatment with an aromatase inhibitor or other anti-estrogen therapies. The primary efficacy endpoint of this trial is PFS, and secondary endpoints are overall survival (OS), objective response rate (ORR), duration of response and disease control rate (DCR).

Phase II trial of neoadjuvant MM-121 in combination with paclitaxel for HER2- breast cancer. A randomized, open-label phase II clinical trial of neoadjuvant MM-121 combined with paclitaxel in approximately HER2- breast cancer patients is under way. The primary efficacy endpoint of this trial is pathologic complete response (pCR) rate at time of surgery. Approximately 200 patients will be enrolled among two populations:

- **Group A**: ER+/HER2- and have not undergone prior treatment or surgery; and
- **Group B**: ER-/PR-/HER2- (triple negative) and have not undergone prior treatment or surgery.

Each population of patients is being randomized at 2:1 ratio to receive either MM-121 in combination with paclitaxel or paclitaxel alone.

Phase II trial of MM-121 in combination with paclitaxel for platinum resistant or refractory advanced ovarian cancer. MM-121 is also being studied in a randomized, open label Phase II clinical trial in combination with paclitaxel in approximately 210 patients with advanced ovarian cancer who are resistant or refractory to treatment with platinum-based chemotherapies. The primary efficacy endpoint of this trial is PFS, and the secondary endpoints include OS, ORR and DOR.

DX-121 is a companion diagnostic for MM-121 that should determine patients who will best benefit from the drug. Merrimack and Sanofi are also developing a companion diagnostic that is designed to assess the levels of five biomarkers to identify tumors that are responsive to MM-121 in animal models. This test measures the levels of five proteins involved in the ErbB pathway and predicts the activated state of ErbB3 and, therefore, the potential responsiveness of the tumor to MM-121 based on those levels. Therefore, biopsies from patients participating in the clinical trials for MM-121 will be collected in order to measure tumor biomarker levels.

Market Opportunity

We conservatively model peak MM-121 sales of $1.6B and royalties of $180M across several indications. Based on ongoing clinical trials, we model MM-121 sales only in breast, ovarian, and lung cancer, and we model sales only in the refractory settings. However, this is conservative as MM-121 has potential in earlier line settings and in additional cancer indications. We estimate peak sales of approximately $430M, $895M and $305M for NSCLC, HER- ER/PR+ breast cancer, and ovarian cancer, respectively. This translates to peak royalties of approximately $50M, $100M, and $35M for NSCLC,

breast, and ovarian cancer, based on a low-teens US royalty rate and high-single digit ex-US royalty rate. These estimates are based on penetration assumptions of 25-35% in refractory settings and pricing on par with other cancer drugs including Herceptin. Importantly, in our valuation, MM-121 cash flow has high margins, as Sanofi covers all development and commercial costs. We also probability adjust all MM-121 revenues by 40% in our valuation. See model section for revenue models.

Competitive Landscape for ErbB3 Inhibitors

Several compounds in development targeting ErbB3-mediated heterodimerization. While Roche's pertuzumab does not target ErbB3 directly, it does block HER2/ErbB3 heterodimerization, so, while it does not have the same market potential that successful ErbB3 targeting agents would have, we are including it in the competitive landscape as it relates to its potential future use in HER2+ tumors. Of all ErbB3 compounds in development, MM-121 is the farthest along in the clinic with the broadest phase II clinical program.

Exhibit 8: Compounds in development blocking ErbB3 Heterodimerization

Drug Name	Target	Manufacturer	Partners	Cancer Type	Stage
Petruzumab	HER2	Roche		Breast	Phase III
AZD8931	ErbB1/2/3	AstraZeneca		Breast, Solid	Phase II
MM-121 / SAR 256212	ErbB3	Merrimack	Sanofi, Dyax	Breast, Kidney, Lung Cancer, Ovarian, Prostate, Solid, HER2-negative Breast	Phase II
AMG 888	ErbB3	Amgen	Daiichi Sankyo	NSCLC	Phase II
MM-111	ErbB2/3	Merrimack		HER2-positive Breast	Phase I/II
AV-203	ErbB3	Aveo	Biogen Idec	Breast, Pancreatic, Prostate, Solid	Pre-Clinical
KB9520	ErbB3	Karo Bio		Cancer	Pre-Clinical
EZN-3920	ErbB3	Enzon	Santaris Pharma	Breast	Pre-Clinical

Source: Oppenheimer & Co.

MM-121 should have broader market potential than pertuzumab, outside of HER2+ breast cancer. However, since pertuzumab blocks only the interaction between HER2 and ErbB3 by binding to HER2, it cannot block the interactions between ErbB3 and EGFR (ErbB1) and ErbB4, which drive the growth in other types of solid tumors, such as NSCLC, head & neck, ovarian cancer, and colorectal cancer. In addition, pertuzumab cannot eliminate any contribution to tumor growth by ErbB1/ErbB3 and ErbB4/ErbB3 heterodimers. Therefore, we believe that while pertuzumab has validated the role of ErbB3 in driving tumor growth via heterodimerization with HER2, MM-121 has a much broader potential in other tumor types outside HER2+ breast cancer. Furthermore, pertuzumab could select for breast cancer cells that overexpress HER2 that cannot be blocked by pertuzumab/Herceptin, or that express variants that cannot be blocked by pertuzumab. MM-121, since it will be developed with inhibitors of the different ErbB receptors, would need to select for tumors that can escape the blockage of ErbB3 and the other ErbB receptors, which creates a much higher hurdle for developing resistance. Therefore, we believe that while pertuzumab serves as a useful validator of blocking ErbB3-containing heterodimers as a target in solid tumors, it will not have the broad therapeutic potential of MM-121 in other solid tumor types.

Collaboration with Sanofi

In October 2009, Sanofi licensed exclusive worldwide right to develop, manufacture and commercialize MM-121. Merrimack retained potential co-promotion rights in the United States. Under the terms of the agreement, Sanofi agreed to pay Merrimack an upfront cash payment of $60M for the research, development, manufacturing and commercialization rights. Merrimack is eligible for development and regulatory milestone payments up to $410M (of which Merrimack has already received $20M) on MM-121, royalties on the worldwide product sales and will receive additional performance milestones of up to $60M on worldwide sales. Merrimack will participate in the development of MM-121 until the completion of phase II trials. Merrimack is eligible for tiered, escalating royalties beginning in the sub-teen double digits based on net sales of

MM-121 in the US and beginning in the high-single digits based on net sales of MM-121 outside the US. In the case of co-promotion, Merrimack will be responsible for sales force costs and other sales and marketing costs for MM-121 in the United States, and will be eligible for tiered, escalating royalties beginning in the high teens. If a diagnostic is used with MM-121 in the treatment of solid tumors, Merrimack is entitled to an increase in the royalty rate. Sanofi is responsible for all development and manufacturing costs for MM-121 after phase II. Merrimack is currently manufacturing MM-121 for the current clinical trials, while Sanofi is responsible for commercial manufacturing of MM-121 at the time of phase III clinical trials. Finally, Merrimack gets reimbursed for development and costs related to the development of MM-121.

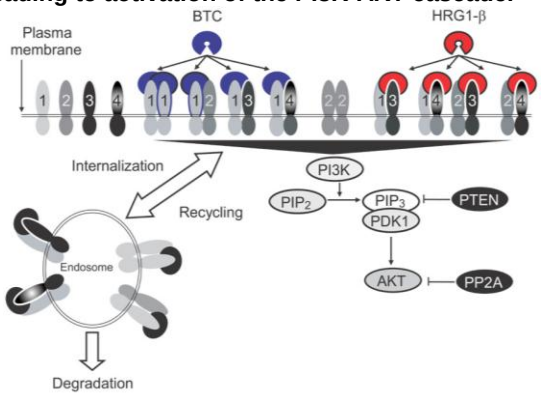
Background on ErbB3 as a Target for Cancer Therapies

ErbB receptor family background. The ErbB family consists of four closely related transmembrane tyrosine kinase receptors: epidermal growth factor receptor (EGFR; also known as HER1), ErbB2 (HER2), ErbB3 (HER3) and ErbB4 (HER4). Each receptor has an extracellular domain where the ligand binds, a transmembrane segment and an intracellular protein tyrosine kinase. Receptor dimerization (pairing) is required for ErbB function/signaling activity. Dimerization can occur between two different ErbB receptors (heterodimerization) or between two molecules of the same receptor (homodimerization). ErbB receptors normally exist as inactive monomers that cannot dimerize and signal, and ligand binding is required for ErbB receptor dimerization. Although all four ErbB receptors have the same essential domains, the functional activity is different. EGFR and ErbB4 have active tyrosine kinase domains and known ligands. ErbB3 can bind to several ligands but lacks tyrosine kinase activity and cannot form homodimers, and only forms heterodimers. ErbB2 possesses an active tyrosine kinase domain but no direct ligand. Different homodimer/heterodimer pairings allow for diverse signaling pathways to be activated and have different strength in activating these pathways. Homodimers weakly perpetuate signals compared with heterodimers. The ErbB2–ErbB3 heterodimer is considered the most potent ErbB pair with respect to strength of interaction, ligand-induced tyrosine phosphorylation and downstream signaling. ErbB3 might be a necessary partner for the oncogenic activity of ErbB2 in tumors overexpressing ErbB2. To add to the complexity of the system, ErbB receptor trafficking events are ligand and dimer specific; different ligand-receptor dimer complexes internalize, and recycle or degrade, at different rates.

Limitations of the current approach of drugs targeting ErbB family receptors. Drugs that inhibit oncogenic tyrosine kinases, specifically those of the ErbB family, such as imatinib, erlotinib, and trastuzumab, have become effective cancer therapies, further underlining the importance of these enzymes as valuable drug targets. Abnormal hyperactivation of tyrosine kinases occurs via overexpression of receptors (via gene amplification), constitutive activation (via activating mutations) and overexpression of ligands, and can lead to oncogene dependence. The current therapies were designed to block the activity of individual tyrosine kinases that were known to be overexpressed/ overactive in a subset of cancer patients who had the overexpressed/overactive form of the protein. This was the clear and obvious choice of initially targeting tyrosine kinases, and the low-hanging fruit for target selection for cancer drug development. However, the initial approach would not have addressed situations where the overexpressed/overactive protein is not the most potent driver of tumor growth, where the uninhibited heterodimerization partner can provide an escape mechanism, and, finally, and it would fail to identify potentially valid targets if they are not overexpressed/overactive.

Merrimack developed mathematical models to elucidate the individual components in ErbB signaling. The models were designed to model the interactions between ErbB1, ErbB2, ErbB3, and ErbB4 and the mitogen-activated protein kinase (MAPK) and phosphatidylinositol 3-kinase (PI3K) signaling cascades stimulated by epidermal growth factor family (EGF) members such as betacellulin (BTC) and heregulin 1–β (HRG1-β) (Exhibit 9). Previous research has shown that stimulating the ErbB signaling network with EGF and HRG1-β leads to activation of two downstream kinases, ERK (extracellular signal-regulated kinase) and AKT, and that ErbB2 overexpression transforms transient EGF-induced signaling into sustained signaling. Merrimack's scientists have shown that BTC and HRG1-β are potent inducers of AKT signaling in most tested cell lines obtained from solid tumors, and that these ligands were widely present in tumors obtained from patients.

Exhibit 9: The ErbB receptors ErbB1-4, BTC binding to ErbB1 and HRG1-β binding to the ErbB3 receptor, receptor dimerization, dimer internalization, and recycling, and interactions leading to activation of the PI3K-AKT cascade.



Source: Merrimack Inc.

Merrimack's scientists looked at the activation of the ligand-ErbB receptor–PI3K axis in response to HRG1-β and BTC, and have used the results to generate a mathematical model that incorporated the individual contributions of each of the ErbB receptors on activation of downstream signaling. Based on the simulations, the ErbB3 receptor was predicted to have a dominant role in downstream activation, suggesting that targeting this key node of the ErbB signaling network may result in therapeutic benefit to cancer patients. To verify this prediction and explore the therapeutic potential of inhibiting ErbB3, Merrimack developed MM-121, a fully human mAb that binds specifically to ErbB3, blocks HRG1-β binding to ErbB3, and inhibits HRG1-β– and BTC-induced AKT signaling.

ErbB3 is a key node in the ErbB receptor signaling network. Merrimack's mathematical models identified ErbB3 as the most sensitive node in the networks with either ligand. AKT activation was less sensitive to ErbB2 with HRG1-β stimulation, because the total number of ErbB2-ErbB3 heterodimers was constrained by the less abundant receptor, ErbB3. For AKT phosphorylation after BTC stimulation, sensitivity analysis identified ErbB3 and ErbB1 as equally sensitive nodes, indicating that BTC induces most of the AKT phosphorylation through ErbB1-ErbB3 heterodimers even though the abundance of ErbB1 is five times that of ErbB3, so that there are far more ErbB1 homodimers than ErbB1-ErbB3 heterodimers.

ErbB3 inhibition could be a valid target in HER2+, HER2- and EGFR-driven cancers. Incomplete inhibition of HER2 in HER2+ tumors could still leave HER2 molecules to form the more potent ErbB3/HER2 dimers, thereby providing an escape mechanism to HER2-targeted therapies. In cancers where HER2 is not overexpressed, HER2 molecules can still form the highly potent ErbB3 heterodimers, which would be the main/sole driver of tumor growth despite normal levels of each protein. Furthermore, since tumors that do not overexpress HER2 have not responded to anti-HER2 therapies, we believe that HER2 homodimers are not significant drivers of tumor growth in HER2- cancers, and that the more potent ErbB3/HER2 heterodimers instead, whose formation is not blocked by current HER2-targeted therapies, may play an important role in tumor growth. Finally, since EGFR is still expressed at higher levels than ErbB3, incomplete inhibition of EGFR in tumors overexpressing EGFR or that harbor activating mutations of EGFR could still allow for enough EGFR protein for the formation of EGFR/ErbB3 heterodimers. Similar to HER2+ cancers, ErbB3 could provide an escape mechanism for tumor growth in EGFR-driven tumors in the presence of EGFR-targeting therapies or in tumors that are resistant to EGFR inhibition. Therefore, inhibiting the formation of EGFR/ErbB3 heterodimers could starve EGFR-dependent tumors of an essential growth signal in tumors where EGFR is incompletely inhibited or the tumor is resistant to EGFR inhibition. For these reasons, Merrimack and Sanofi are developing MM-121, the anti-ErbB3 antibody in cancers where ErbB3-containing heretorimers could promote tumor growth, such as HER2- and EGFR-dependent cancers. Merrimack is also developing MM-111, a bispecific anti- HER2/ErbB3 antibody, in HER2+ cancers.

Background on Breast Cancer

Breast cancer basics. Although mortality from breast cancer is decreasing, worldwide, it remains the most common form of cancer in women. On diagnosis, several tests are carried out in order to determine the stage and biological characteristics of the tumor:

- **Lymph node involvement**. Node-negative breast cancer indicates that the tumor has not spread to the lymph nodes and is therefore easier to cure and less likely to recur. If the tumor has spread to axillary lymph nodes, the cancer has become node-positive and is more difficult to treat, with a higher chance of recurrence.
- **Histologic grade**. The grade of a breast cancer refers to how closely tumor cells resemble normal cells, giving an indication as to how quickly the cancer may develop. Grade 1 indicates a low-grade cancer, where the cells closely resemble normal cells, show slow growth and are less likely to spread. Grade 3 indicates a high-grade cancer, with cells of an abnormal appearance that are likely to grow and spread rapidly.
- **Hormone Receptor status**. Two female fertility hormones, estrogen and progesterone, are known drivers of some types of breast cancer. Estrogen receptor (ER) and progesterone receptor (PR) status are important prognostic factors since they indicate whether the tumor is stimulated to grow by these hormones. ER+ and PR+ tumors are able to respond to anti-hormonal therapy. Hormone receptor-negative tumors do not respond to hormonal therapy, and can be treated only by chemotherapy, making this form of breast cancer more difficult to treat successfully.
- **HER2 status**. HER2 is a growth factor cell-surface receptor, which aids control of normal cell growth and division. Overexpression of HER2 causes breast cells to divide uncontrollably and form a tumor. Overexpression of the HER2 is found in about 25% of breast cancer cases and is linked to more aggressive tumors capable of spreading faster, with an elevated rate of recurrence following treatment.

Treatment paradigm. Breast cancer patients with lymph node involvement/metastatic disease and/or histologic grade greater than 1 are almost always treated with one of many pharmacologic therapies, in addition to radiation therapies and surgery. Hormone-receptor positive breast cancer patients receive aromatase inhibitors, with or without additional chemotherapy. HER2+ breast cancer patients receive Herceptin or Tykerb with or without traditional chemotherapy. Finally, triple-negative breast cancer patients (TNBC) who are negative for HER2 and the hormone receptors receive traditional chemo-based regimens.

Where MM-121 fits in. We believe that MM-121 and MM-111 have potential in HER2+ breast cancer, in both hormone receptor positive and negative cancers. Furthermore, since MM-121 and MM-111 can block ErbB3 from forming heterodimers with EGFR and ErbB4, we believe that both antibodies could provide therapeutic benefit in HER2- breast cancer. Importantly, pertuzumab's potential in breast cancer will be limited to HER2 overexpressing breast cancer patients, since it blocks HER2/ErbB3 heterodimerization by binding to HER2, leaving ErbB3 free to heterodimerize with other ErbB family members.

Background on Non-Small Cell Lung Cancer

Lung cancer basics. Mortality from lung cancer has been on a downward trend with improving therapies and decreasing smoking rates. Nevertheless, lung cancer remains the most common form of cancer worldwide. Similar to breast cancer, staging, histology and molecular profiling of the primary tumors are performed on initial diagnosis with NSCLC.

- **Squamous vs. non-squamous NSCLC**. Broadly speaking, approximately 75% of NSCLC patients have non-squamous histology, while the remaining 25% present with squamous histology. Different chemo regimens are recommended as first-line therapy in patients with the two different histologies.
- **EGFR mutation status**. Patients harboring mutations in the EGFR gene are known not to respond well to EGFR-targeted therapies, while those with wild-type EGFR status have significant therapeutic benefit from this drug class.
- **ALK overexpression**. While only a small minority of NSCLC patients overexpress ALK (approximately 4% of all NSCLC cases), recently approved

ALK-inhibitor crizotinib (Xalkori) has shown impressive efficacy in this patient population.

Treatment paradigm. With the advent of molecular profiling and the clinical development of targeted therapies, the treatment of NSCLC has become an example of the emerging personalized therapy. Non-squamous patients are started with Alimta or a taxane-based platinum-containing regimen, while patients with squamous histology are not suitable for Alimta, and are initiated on traditional chemo doublets. Based on molecular biomarkers, Tarceva is recommended for the treatment of NSCLC patients with EGFR activating mutations, based on prior observations that the efficacy of EGFR inhibitors is much stronger and confined to patient subgroup. Importantly, Iressa (gefitinib), also an EGFR inhibitor, is approved for this subset of patients in Europe, while Tarceva is available for use in these patients in Europe and the United States. Finally, NSCLC patients who overexpress ALK are treated with Xalkori.

MM-121 should augment existing NSCLC therapies. We believe that MM-121 will show the highest degree of efficacy in patients positive for activating EGFR mutations, in combination with Tarceva. Current phase II trials for MM-121 are designed to test the benefit of adding MM-121 to Tarceva in patients with mutated EGFR across all histologies and ALK status.

MM-398

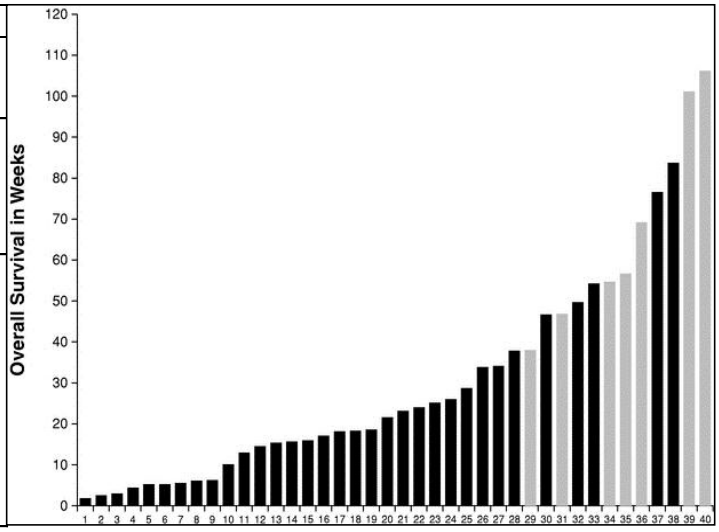
MM-398 is a nanotherapeutic encapsulation of irinotecan, a common generic chemotherapy. Merrimack is currently developing the drug in a phase III trial in 2nd-line pancreatic cancer and a phase II trial in 2nd-line colorectal cancer. Merrimack currently holds worldwide rights to develop and market MM-398. However, under an agreement with Taiwan-based PharmaEngine, Merrimack owes the company potentially up to $205M in milestones and tiered single to low double-digit royalties on drug sales in Europe and certain Asian territories. Merrimack holds patent applications on composition and methods of making and use of MM-398, providing protection until 2025-2027 in the US and 2025 in Europe.

Improving upon irinotecan with nanoencapsulation. MM-398 consists of irinotecan encapsulated within a liposome. When introduced into circulation, these liposomes steadily release irinotecan over time. Free irinotecan is then metabolized in the liver and converted into the metabolite, SN-38, which is 1,000-fold more active and largely responsible for irinotecan's efficacy. Theoretically, this encapsulation improves the therapeutic effect, as it prevents inactivation or elimination of irinotecan while in circulation and retains a larger amount of drug to be converted to active metabolite. Encapsulation may also potentially reduce toxicity by limiting systemic exposure to irinotecan. Additionally, Merrimack postulates that MM-398 is able to selectively enter tumor vs. normal tissue, given the size and characteristics of the drug. Macrophages in tumor tissue may then be able to break down the liposome and convert irinotecan to more active SN-38.

Compelling phase II results in refractory pancreatic cancer. PharmaEngine has conducted a phase II single-arm trial evaluating MM-398 at 120mg/m^2 every three weeks in pancreatic cancer patients who previously failed gemcitabine. The study enrolled 40 patients from three sites, two in Taiwan and one at the University of California, San Francisco. MM-398 demonstrated a 75% three-month survival rate, meeting the predefined study success criteria of ≥65%. The study results (Exhibit 10) are very promising in the context of historical survival rates. Importantly, the drug showed impressive median overall survival of 22.4 weeks or 5-6 months, which is greater than the approximately 2 month survival seen in gemcitabine-failures and is in line with survival in first-line pancreatic cancer. MM-398 also demonstrated six- and 12-month survival rates of 40% and 20%, respectively, similar to the 46% and 18% survival rates, respectively, seen with gemcitabine in healthier front-line patients. These results spurred the initiation of the ongoing phase III NAPOLI-1 trial in refractory pancreatic cancer.

Exhibit 10: Phase II results in gemcitabine-refractory pancreatic cancer with waterfall plot

	n=40
Objective response rate	7.5%
Partial response	7.5%
Stable disease	40%
3-month survival rate	75%
6-month survival rate	40%
12-month survival rate	20%
Progression free survival, weeks	9.6
Overall survival, weeks	22.4
Grade 3/4 events	
Neutropenia	30.0%
Leucopenia	22.5%
Anemia	15.0%
Diarrhea	7.5%
Fatigue	7.5%
Nausea	5.0%
Vomiting	5.0%
Thrombocytopenia	5.0%



Note: Waterfall plot shows survival as of 5/31/11, with grey bars representing patients who are still alive. Source: Company reports.

Early phase I results show promise in colorectal cancer. PharmaEngine conducted a phase I dose escalation study in 18 patients who failed first-line treatment with oxaliplatin-based chemotherapy. Patients received MM-398 every two weeks, with six patients evaluated in each of the three dose groups: 80, 90, and 100mg/m^2. MM-398 100mg/m^2 was determined to be the maximally tolerated dose, with grade 3 diarrhea being the dose limiting toxicity for one patient in each dose group. Neutropenia was the main cause of dose escalation delay. This side effect profile is not surprising as both diarrhea and neutropenia are the key side effects of irinotecan. The phase I study showed some early anti-tumor activity as well. Of the 17 evaluable patients, 4 had a partial response (23.5% ORR) and 8 had stable disease (70.6% DCR). These response rates appear favorable to what has been seen with FOLFIRI in a prior second-line trial (4% ORR, 34% DCR). Based on these results, the phase II trial will evaluate MM-398 at 80mg/m^2 every two weeks with fluorouracil and leucovorin in second-line colorectal cancer.

Modest phase II results in gastric cancer. PharmaEngine conducted a phase II trial evaluating MM-398 in metastatic gastric or gastroesophageal injunction adenocarcinoma patients, who failed one prior therapy. A total of 132 patients from Europe and Asia were randomized to 120mg/m^2 MM-398, 300mg/m^2 irinotecan, or 75mg/m^2 docetaxel every three weeks. The MM-398 arm met the study success criteria of five or more patients achieving an objective response. However, MM-398 did not appear to have substantially differentiated response rates, PFS, and OS compared to irinotecan and docetaxel (Exhibit 11). Based on these results, gastric cancer is unlikely an optimal indication for MM-398.

Exhibit 11: Phase II results in second-line gastric cancer

	MM-398	Irinotecan	Docetaxel
n=132	44	44	44
Objective response rate	6 (13.6%)	3 (6.8%)	7 (15.9%)
Disease control rate	27 (61.4%)	27 (61.4%)	23 (54.6%)
Progression free survival, days	81	79.5	82
Overall survival, days	218	235	219

Source: Company reports.

Companion diagnostics under preclinical evaluation. Merrimack's modeling shows tumor deposition of nanotherapeutics like MM-398, and also MM-302, can be highly variable and is a limiting factor for efficacy. DX-929, a nanoencapsulated copper, may allow Merrimack to evaluate nanotherapeutic deposition in patients through imaging. This allows potential identification of patients who may be more responsive to Merrimack's nanotherapeutics.

Key Clinical Readouts in 2013

Phase III results in pancreatic cancer should be available in mid-2013. Merrimack is conducting an open-label phase III trial evaluating MM-398 against a regimen of fluorouracil and leucovorin in metastatic pancreatic cancer patients who have failed gemcitabine. The study will enroll 250 patients from 90 sites around the world. Overall survival is the primary endpoint, with progression-free survival and objective response rate as secondary endpoints. Merrimack expects enrollment completion and results in mid-2013. On balance, we view the pancreatic program as high risk-reward. However, we are optimistic for a positive outcome, based on strong phase II results.

The bar for benefit in pancreatic cancer is low. We believe MM-398 simply needs to show a statistically significant survival benefit to gain market acceptance. We do not believe the magnitude of survival benefit has to be substantial, as there are no effective treatments for refractory patients, who can die within weeks. Even in the 1st-line setting, the bar for survival is low. Gemcitabine, the standard of care, provides a median survival of only 5.7 months in the front-line setting. Further, regulatory standards for OS are also low in pancreatic cancer. Tarceva gained approval as a 1^{s}-line add-on therapy, by extending gemcitabine's overall survival by only two weeks (6.4 vs. 6.0 months). Gemcitabine was approved for the 1^{s}-line setting with only a 1.5-month benefit over fluorouracil (5.7 vs. 4.2 months). Notably, the regulatory requirements for MM-398 should be even lower in the refractory setting.

However, pancreatic cancer remains a challenging indication. The failure rate for clinical development in this indication is high and largely attributed to the disease's rapid progression and lack of therapeutic targets. Consequently, there have been few significant advancements in pancreatic cancer in the last 15 years. MM-398 could potentially be the next new therapy for this indication, based on the strong phase II results in the refractory setting. However, we do see some risks with the program, as phase II results can be misleading in pancreatic cancer. These trials are typically single-arm in design and have small patient numbers. Additionally, patient variability is an issue in phase II trials, given the short survival duration in this disease. As a result, it is not uncommon for investigational pancreatic cancer drugs to show promising phase II results, but then subsequently fail in larger controlled phase III trials.

Prior clinical experience with irinotecan in pancreatic cancer is mixed, but may not apply to MM-398. We believe there is no conclusive evidence that irinotecan has efficacy in pancreatic cancer. Despite promising response rate results in earlier studies, two large investigator- sponsored phase III studies (Rocha Lima et al. JCO 2004, Strathopouolos et al. BJC 2006) showed irinotecan did not improve survival when added to gemcitabine in 1st-line pancreatic cancer. However, there was a recently positive phase III trial (Conroy et al. NEJM 2011), showing a regimen of oxaliplatin, irinotecan, fluorouracil, and leucovorin (FOLFIRINOX) resulted in a significant survival benefit vs. gemcitabine (11.1 vs. 6.8 months) in 1st-line patients. Although the historical data for irinotecan in pancreatic cancer is mixed, we do not believe this has significant bearing on MM-398. We believe MM-398 is better positioned for success vs. irinotecan, based on Merrimack's delivery technology. Further, preclinical modeling and positive phase II results provide support for the benefits of the nanoencapsulation technology. Additionally, it is difficult to draw parallels between the studies mentioned and NAPOLI-1. Notably, NAPOLI-1 is evaluating MM-398 as a monotherapy in the refractory setting.

We also expect phase II results in colorectal cancer in mid-2013. Independent investigators are conducting an open-label phase II trial evaluating leucovorin, fluorouracil, and MM-398 (FUPEP) vs. leucovorin, fluorouracil and irinotecan (FOLFIRI) in metastatic colorectal cancer patients failing 1st-line oxaliplatin chemotherapy. They will enroll 88 patients from France. The study will evaluate the primary endpoint of response rates and secondary endpoints of overall survival and progression-free survival. Merrimack expects results in mid-2013. Overall, we believe colorectal cancer represents an additional notable opportunity for MM-398.

The colorectal cancer program is also promising. Irinotecan has been proven to be effective in colorectal cancer. The compound is approved with leucovorin and fluorouracil (FOLFIRI) for colorectal cancer, and irinotecan-based regimens dominate the 2nd-line setting. Further, we believe, with the benefits of nanoencapsulation, FUPEP may be able to show an efficacy or tolerability benefit over FOLFIRI. Additionally, given the similarity

between the two regimens, we believe the phase II results will be a good measure of the benefits of the nanoencapsulation technology and provide a read-through for the entire MM-398 program.

Market Opportunity

Pancreatic cancer could represent a $1.2B opportunity for MM-398. We believe refractory pancreatic cancer represents the nearest term opportunity for Merrimack, and we estimate peak sales of $290M in this setting. Our estimate is based on approximately 30% penetration in gemcitabine-failures, although this penetration may be higher given the lack of approved therapies in this setting. We assume average treatment duration of 5 months with an approximate $33,000 US cost per patient. If the NAPOLI-1 trial is successful, Merrimack will conduct a front-line study. We note the front-line setting is substantially larger than the refractory setting, as there are over twice the number of patients, who also receive treatment for longer durations. We estimate peak front-line sales of $885M, based on 25% penetration, 7.5 month treatment duration and approximately $50,000 US cost per patient. For both front-line and refractory pancreatic cancer, there is clear upside to our pricing assumptions given the high unmet need in this disease. Importantly, we adjust our refractory and front-line pancreatic cancer sales estimates to reflect a 40% probability of success. See model section for revenue models.

Refractory colorectal cancer represents a $600M opportunity for MM-398. Our estimate is based on MM-398 use only in 2nd- and 3rd-line colorectal cancer, as these settings are Merrimack's current focus. We model peak penetration of 25% and 20% in addressable 2nd-line and 3rd-line patients, respectively. These estimates may be low, particularly given the very broad use of irinotecan-based regimens in refractory patients. However, we believe the high cost we assume for MM-398 in pancreatic cancer may somewhat limit MM-398 adoption over generic irinotecan. Importantly, we adjust our sales estimate to reflect a 40% probability of success. Our colorectal cancer estimates do not include 1st-line sales. However, we believe future studies evaluating FUPEP with Avastin in 1st-line colorectal cancer could unlock an even greater market opportunity longer term. See model section for revenue models.

Pancreatic Cancer Background

Pancreatic cancer represents a significant unmet need. This disease has a US incidence of 43,920 in 2012. Merrimack is focused on adenocarcinoma of the pancreas, which represents 95% of pancreatic cancer cases. Compared to other cancers, this type of pancreatic cancer has a dramatically worse prognosis and is the fourth leading cause of cancer death. Patients have a mean survival of 6 months with current treatments. Further, patients are typically diagnosed in the late stages of disease, given pancreatic cancer's rapid progression and the lack of symptoms in early disease.

Physicians' armamentarium for pancreatic cancer is limited. Gemcitabine (Gemzar) was approved in 1996 as a first-line monotherapy. Although the drug provided only 5.6-month survival in clinical testing, gemcitabine has become the standard of care in front-line pancreatic cancer. Erlotinib (Tarceva) is the only other agent to subsequently achieve approval for pancreatic cancer in 2005. The drug was approved as a gemcitabine add on therapy in the 1s-line setting. The meaningfulness of erlotinib's efficacy is controversial within the physician community, as the drug extends gemcitabine's survival benefit by only approximately 2 weeks. There is no standard 2nd-line treatment for pancreatic cancer. Physicians typically enroll refractory patients into clinical trials or employ an array of standard cytotoxic agents including capecitabine, fluorouracil, oxalplatin, cisplatin, pemtrexate, or targeted therapies cetuximab and bevacizumab. However, the efficacy of these regimens in refractory pancreatic cancer is not well supported.

Colorectal Cancer Background

Colorectal is one of the larger cancer markets. This disease is the third most common cancer type, with an estimate US incidence of 103,170 in 2012. Although diagnoses and treatment earlier in the disease can lead to notably improved chances for survival, the average five-year survival rate of colorectal cancer is approximately 65%. Although there are several risk factors for colorectal cancer, 90% of the disease occurs in people over the age of 50. As a result, there has been a significant focus on screening in this population to improve treatment outcomes.

There are several multi-chemotherapy regimens for colorectal cancer. Common regimens include oxaliplatin, leucovorin, and fluorouracil, abbreviated as FOLFOX and irinotecan, fluorouracil, and leucovorin abbreviated as IFL. Note another common regimen, FOLFIRI, is very similar to IFL and has the same components. However, for FOLFIRI, fluorouracil is administered via continuous infusion instead of as a bolus, which reduces IFL's increased 60-day mortality caused by a syndrome of diarrhea, neutropenia and sepsis. Among multi-chemotherapy regimens, FOLFOX is by the far the most frequently employed in the 1^{s}-line setting, as some studies have shown FOLFOX is superior to IFL.

A few targeted therapies have been approved for colorectal cancer, as these agents have shown improved efficacy, notably when added to chemotherapy regimens. Notably, bevacizumab (Avastin), a vascular endothelial growth factor inhibitor has been approved as a first-line treatment when added to FOLFOX or IFL. Notably, following Avastin's approval in this indication, FOLFOX+Avastin has taken dominant share in the 1^{s}-line setting. Cetuximab (Erbitux) and panitumumab (Vectibix), endothelial growth factor receptor antagonists are approved as second-line agents for colorectal cancer.

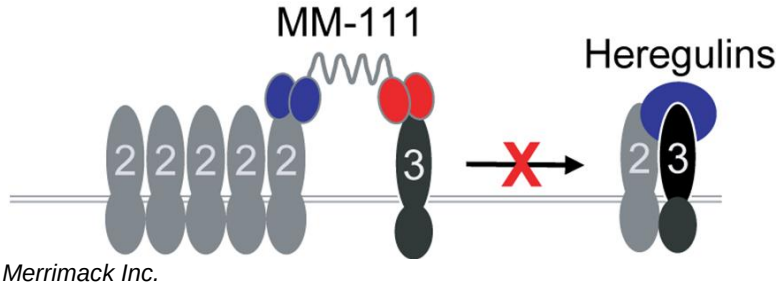
MM-111

Compound Overview

MM-111 is a bispecific antibody designed to target cancer cells that overexpress the HER2 and ErbB3 receptors. MM-111 is currently being evaluated in three Phase I clinical trials. Merrimack is also developing a companion diagnostic biomarker assay to identify patients who are most likely to respond to MM-111.

MM-111 has a novel mechanism of action. MM-111 inhibits growth and survival signaling in HER2+ cells through the inhibition of ErbB3. The bispecific antibody binds simultaneously to HER2 and ErbB3, preventing formation of the HER2/ErbB3/heregulin complex, which promotes tumor growth in HER2+ cells. As a result, MM-111 could be an effective therapy in HER2+ cancer, particular in combination with HER2 inhibitors. In fact, Merrimack believes MM-111 may be effective in patients whose tumors express HER2 at lower levels than those needed for current HER2 inhibitors. Additionally, by cross-linking ErbB3 to HER2, MM-111 may also render ErbB3 unavailable for heterodimerization with EGFR and ErbB4. Prevention of EGFR/ErbB3 and ErbB4/ErbB3 heterodimerization may further limit tumor growth. Therefore, MM-111 could have even greater activity than compounds that do not directly inhibit HER3, such as pertuzumab, which primarily prevents HER2/ErbB3 dimerization.

Exhibit 12: MM-111 blocks heterodimerization between ErbB3 milestones for MM-121



Source: Merrimack Inc.

Preclinical development supports MM-111's scientific rationale. The anti-tumor activity of MM-111 has been studied extensively in in vitro and in in vivo xenograft studies. MM-111 showed activity in several HER2+ xenograft models, including breast, lung and gastric cancer. Furthermore, MM-121 had higher anti-proliferative activity in tumors expressing higher HER2 levels. The drug also showed synergistic antitumor activity in combination with HER2-targeted therapies, as well as anti-hormone therapy.

Clinical profile still emerging for MM-111. Merrimack completed a phase I monotherapy study for MM-111 in refractory HER2+ cancers, and is conducting two phase I combination studies. One of the combination trials has multiple arms, evaluating MM-121 in different combination regimens with HER2 inhibitors and chemotherapies in HER2+

cancers. Interim data for 32 patients showed a 25% ORR and 47% DCR across arms. Of the combinations evaluated with initial results, MM-111+Herceptin+paclitaxel appears most promising (Exhibit 11). Although these early data are encouraging, it is difficult to parse out MM-111's contribution to efficacy in a combination regimen. We look to more mature data, which may yield improving response rates.

Exhibit 13: Response rates in phase I multi-arm study with MM-121

Combination with:	Indication	N	ORR	DCR
MM-111+Paclitaxel+Herceptin	HER2+ cancer	10	**40%**	**80%**
MM-111+ Cisplatin+capecitabine+ trastuzumab	HER2+ cancer	9	33%	44%
MM-111+ Herceptin+lapatinib	HER2+ cancer	13	8%	23%

Source: Company reports, Oppenheimer & Co.

Companion diagnostic under development. Merrimack is also developing a companion diagnostic for MM-111 (DX-111) to identify patients likely to respond to the drug based on expression levels of several biomarkers, including HER2, ErbB3 and heregulin.

Dual ErbB3-HER2 has clear rationale in HER2+ cancers. Incomplete inhibition of HER2-targeting drugs may leave sufficiently high HER2 levels available for heterodimerization with ErbB3, which would lead to persistent activation of growth signaling and, therefore, tumor progression. Merrimack plans to develop MM-111 in HER2+ cancer settings. We believe the recent clinical success of Roche's pertuzumab, an antibody that blocks the heterodimerization of ErbB3/HER2, validates targeting ErbB3/HER2 in breast cancer. Results from the phase III CLEOPATRA trial showed that pertuzumab in combination with Herceptin and chemo improved PFS to 18.5 months vs. a median of 12.4 months in the control group.

Clinical Program Maturing in 2012

Phase I data should inform phase II trial plans. Of the three phase I trials ongoing (Exhibit 14), we expect final data for the monotherapy study and additional updates from the two combination studies in 2012. We believe particularly the larger multi-arm phase I combination study (n=60) will inform upon a phase II dose. Merrimack plans to initiate a phase II study of MM-111+Herceptin+paclitaxel vs. Herceptin+ paclitaxel in HER2+ breast cancer potentially 1H12 and also a phase II study of MM-111+lapatinib+letrozole vs. lapatinib+letrozole in HER2+ HR+ breast cancer. As a result, we will be focusing on these particular combinations in the maturing phase I multi-arm combination study.

Exhibit 14: Phase I trials for MM-111

Trial Identifier	Size (n)	Type of Cancer	Combo with	Start Date
NCT01304784	60	HER2+ Tumors	Cisplatin, Capecitabine, Herceptin; Lapatinib, Herceptin; Paclitaxel, Herceptin; Lapatinib, letrozole	January 2011
NCT01097460	21	HER2+/Heregulin+ Breast	Herceptin	March 2010
NCT00911898	20	HER2+/Heregulin+ Tumors	Monotherapy	June 2009

Source: Clinicaltrials.gov and Oppenheimer & Co.

Market Opportunity

MM-111 could achieve $1.1B in peak sales in HER2+ breast cancer. We model MM-11 pervading all lines of HER2+ breast cancer as a combination therapy. We model minority penetration of approximately 15%, 18%, 30% and 10% in the 1st-line, 2nd-line, 3rd-line and adjuvant settings, respectively. MM-111 treatment durations may be longer than we model, as competitors with similar mechanisms of action such as pertuzumab have shown a lengthy 18.5-month PFS when combined with Herceptin. We note that our pricing assumptions are in line with Herceptin and other cancer therapies.

MM-302

Compound Overview

MM-302 is liposomal doxorubicin conjugated to HER2-targeting antibodies. The liposomal encapsulation is designed to limit systemic exposure to doxorubicin, which is associated with serious cardiovascular side effects.

Marketed liposomal doxorubicin formulations isolate doxorubicin's cardiotoxicity. The two approved liposomal doxorubicin formulations, Doxil (approved in the US and EU) and Myocet (approved in the EU), have successfully shown that encapsulation successfully reduces cardiotoxicity of the parent compound. However, Doxil has polyethylene glycol (PEG) coating, which results in hand-foot syndrome. Myocet does not have PEG coating and is not associated with this side effect. Given MM-302's lack of PEG coating, we believe the drug could improve doxorubicin's safety profile and avoid the hand-foot syndrome seen with Doxil.

The potential of a HER2-targeted liposomal doxorubicin seems fairly clear in breast cancer. We believe MM-302 could be combined with cardiotoxic therapies, unlike Herceptin. In fact, Herceptin has an FDA black box warning that it cannot be used in concurrent combination with doxorubicin, only in sequential combination. Concurrent combination of Herceptin and doxorubicin was associated with superior tumor response, but the combination resulted in unacceptable cardiotoxicity, including congestive heart failure (CHF). We believe that MM-302, like Doxil, should have lower cardiotoxicity than doxorubicin, while maintaining doxorubicin-like activity due to targeted delivery to HER2 overexpressing tumors.

Preclinical data support MM-302's potential safety benefits. Preclinical results show that at least 87% of all administered doxorubicin in MM-302 remained encapsulated while in the plasma, which we believe limits distribution to the heart and other non-target tissue. Furthermore, MM-302 did not cause microscopic signs of cardiac damage in either rats or monkeys.

Next Clinical Data Readout

Merrimack expects first phase I results in 1H13. Merrimack is evaluating MM-302 in an open-label, dose escalation phase I clinical trial of 18 to 36 refractory HER2+ cancer patients. The trial will determine a maximally tolerated dose for MM-302. Although the study will provide primarily a read on safety, we look for early signs of efficacy as well. An expansion cohort is planned following the dose escalation portion of the trial. Following completion of this study, we would expect Merrimack to focus on HER2+ breast cancer and particular settings where anthracyclines use is limited due to safety concerns.

Market Opportunity

MM-302 could achieve $1.5B in peak sales in HER2+ breast cancer. If successful, MM-302 could take a significant share of the multibillion-dollar Herceptin market. We model penetration of approximately 25%, 20%, 10% and 15% in the 1st-line, 2nd-line, 3rd-line and adjuvant settings, respectively. MM-302's clinical profile is still unfolding, so we model MM-302 treatment durations only modestly higher than that expected with Herceptin, the currently available HER2 inhibitor. Although MM-302 addresses a substantial market opportunity, the compound is early in development. As a result, our MM-302 sales estimates reflect a conservative 25% probability of success.

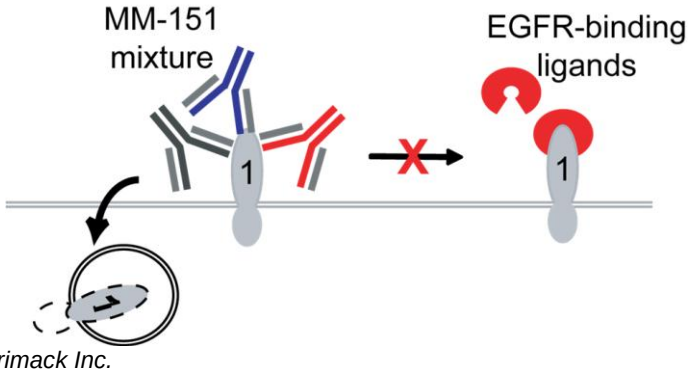
MM-151

Compound Overview

MM-151 is an oligoclonal antibody mixture targeting epidermal growth factor receptor (EGFR or ErbB-1). Merrimack advanced the compound into a phase I study in solid tumors in January 2012. MM-151 was discovered under an antibody discovery collaboration with Adimab. Merrimack owns worldwide rights to MM-151. However, the

company may potentially owe Adimab up to $13.5M in clinical and regulatory milestones per therapeutic area and also mid-single digit royalties on net sales for MM-151.

Exhibit 15: MM-151 blocks EGFR signaling



Source: Merrimack Inc.

MM-151 theoretically could be more effective than current EGFR inhibitors. EGFR is a proven target, given the clinical and commercial success of monoclonal antibodies, erlotinib (Tarceva) and cetuximab (Erbitux), in various oncology indications. MM-151 consists of an oligoclonal antibody mixture of three fully human monoclonal antibodies targeting distinct non-overlapping epitopes of EGFR. As a result, MM-151 inhibits signaling from a broader range of EGFR ligands compared to current EGFR inhibitors, which may translate to improved clinical benefit. Additionally, Merrimack's preclinical modeling suggests this broader coverage may more effectively block signal amplification in the EGFR pathway. Last, MM-151's binding to multiple EGFR epitopes may also create a higher barrier to resistances common to current EGFR inhibitors.

Preclinical results show encouraging activity relative to other compounds. In in vitro experiments, MM-151 showed near complete inhibition of EGFR and also demonstrated an inhibitory effect on signaling and proliferation greater than that of cetuximab. MM-151 also reduced tumor cell growth in xenograft models for lung, triple negative breast, and prostate cancer. Notably, the drug exhibited better activity vs. cetuximab in these models, and has shown activity in lung cancer models with erlotinib resistance. Animal toxicity studies have shown dermatologic and gastrointestinal side effects, which are common with current EGFR inhibitors.

Next Clinical Data Readout

Phase I results will provide initial read on safety and efficacy in 2H13. Merrimack is conducting a phase I dose escalation trial in approximately 60 refractory patients with solid tumors. There will be a focus on enrolling patients with colorectal, non-small cell lung, and triple negative breast cancer. The trial is focused on tolerability and will determine a dose for phase II testing. The study will also provide response rate results, providing an early picture of efficacy. Following phase I results, Merrimack expects to subsequently initiate a phase II data in 2H13.

Market Opportunity

MM-151 represents potential upside to our estimates longer term. It is too early to determine the ideal clinical path for MM-151. If the drug produces positive phase I and II results, we believe Merrimack will ultimately aim to replace current EGFR inhibitors, likely in key indications such as non-small lung cancer and colorectal cancer. However, Merrimack may also initially pursue areas where EGFR inhibitors are ineffective, such as erlotinib resistant patients, allowing for accelerated development. In any case, we currently do not include MM-151 sales estimates in our model and note meaningful clinical success with this compound would present upside to our valuation.

Preclinical Product Candidates

Merrimack has disclosed three preclinical candidates, MM-141, MM-310 and MM-131.

- **MM-141** is a bispecific antibody designed inhibiting insulin growth factor 1 receptor (IGF-1R) and ErbB3. Merrimack plans to file an IND for MM-141 in 2012.
- **MM-310** is targeted liposomal chemotherapy. Merrimack plans to file an IND for MM-310 in 2013.
- **MM-131** is a multi-specific antibody, but the targets have not been disclosed.

Drug Development Beyond Oncology

Merrimack is expanding network biology-based drug discovery through subsidiaries and third parties. Network biology has broad applicability outside of oncology in other areas of high unmet medical need. Merrimack established in 2010 a majority-owned private subsidiary, Silver Creek Pharmaceuticals, to explore network biology-based drug discovery in cardiovascular disease. Silver Creek is still an early-stage company and does not appear yet to have any notable clinical candidates. Merrimack notes they may consider forming additional businesses to apply network biology in places other than oncology. Merrimack has in the past attempted to develop drugs outside of oncology such as with MM-093 for rheumatoid arthritis. However, trials were unsuccessful for this compound and the company licensed MM-093 to GTC Biotherapeutics.

Discovery Platform

Network Biology

Network biology is Merrimack's core platform technology. Network biology takes a holistic approach to understanding a biologic system by analyzing the various relationships among its components. We believe this has clear potential to deliver novel insights for drug discovery. Merrimack's network biology approach involves complex analysis, and we believe the company's platform has advanced in the last decade with the advent of new tools such as 1) high-quality gene, protein, and metabolite databases, 2) high-throughput data collection methods, and 3) powerful computational approaches.

Network biology has the potential to address shortcomings of current drug discovery. In traditional drug discovery, companies typically identify and validate a single molecule, or target, that appears to be abnormally regulated in diseased vs. healthy tissue. This reductionist approach currently dominates drug research. However, the clinical failure rate of therapies developed in this manner is high, as this approach does not take into account the complexity of biologic systems. We believe there is a need for a network biology approach in diseases with poorly understood pathogenesis and a high level of heterogeneity, like cancer.

Merrimack's platform is based on experiments called "critical network identification." First, Merrimack identifies the signaling pathways critical in a given cancer and then uses protein arrays representing the components of these pathways to measure the impact of positive or negative molecular inputs, including existing cancer drugs. These experiments identify signaling networks, as opposed to individual signaling pathways, activated in response to these stimuli. Once critical signaling networks are identified, the company creates biochemical and mathematical models of each network. If the models are validated by further preclinical research, they are then used for drug discovery. Interestingly, based on critical network identification, Merrimack has found several cancers may rely on a limited number of signaling networks for proliferation, despite the complex etiology of a majority of cancers.

MM-151 is a compelling example of a drug developed from network biology. Merrimack noted current EGFR inhibitors had limited efficacy in patients who overexpress EGFR. Further, when these drugs were effective, they often caused resistance. Merrimack's model of the EGFR network revealed current EGFR inhibitors 1) did not account for EGFR signal amplification downstream of EGFR and 2) did not effectively block signaling by high affinity EGFR ligands. These factors may account for the limited efficacy of current EGFR inhibitors and routes for resistance. As a result, Merrimack developed MM-151, a potent inhibitor of EGFR signaling driven by both low and high affinity ligands.

Management Background

Exhibit 16: Select Members of Merrimack Leadership Team

Personnel	Position	Description
Robert J. Mulroy	President and Chief Executive Officer	Mr. Mulroy has served as Merrimack's President and Chief Executive Officer and a member of Merrimack's board of directors since May 1999. Prior to Merrimack, Mr. Mulroy worked as a management consultant in the pharmaceutical and healthcare industries. Mr. Mulroy has also worked as a consultant in the field of international development and has served as an advisor to multiple start-up companies in the biotechnology industry. Mr. Mulroy holds a master's degree in public and private management from Yale University and a B.A. from Stanford University.
Ulrik B. Nielsen, Ph.D.	SVP & Chief Scientific Officer	Dr. Nielsen has served as Merrimack's SVP and Chief Scientific Officer since March 2009. Dr. Nielsen has also served as President and Chief Executive Officer of Merrimack subsidiary Silver Creek Pharmaceuticals, Inc., since July 2010. Dr. Nielsen was one of Merrimack's co-founders and has been leading Merrimack's research and drug discovery since March 2002. Prior to joining Merrimack, Dr. Nielsen was a post-doctoral fellow at MIT, where he researched the interface among biology, engineering and computational biology. Dr. Nielsen holds a Ph.D. in molecular biology and an M.S. in biochemistry from the University of Copenhagen.
Clet Niyikiza, Ph.D.	EVP of Development	Dr. Niyikiza has served as Merrimack's EVP of Development since February 2010 and SVP of Product Development from July 2009 to February 2010. Previously, Dr. Niyikiza served as VP and Medicine Development Leader at GlaxoSmithKline, overseeing product development and global anti-cancer medicine development strategy. Prior to that, Dr. Niyikiza held multiple high level positions at Eli Lilly, where he ultimately led the oncology translational and applied genomics research division. Dr. Niyikiza holds a Ph.D. in mathematical sciences and an M.A. in mathematics from Indiana University.
William A. Sullivan	Chief Financial Officer & Treasurer	Mr. Sullivan has served as Merrimack's Chief Financial Officer since May 2011, Treasurer since February 2010, and Controller from November 2007 to February 2010. Previously, Mr. Sullivan served as Corporate Controller of Vette Corp., a thermal management solutions company. Mr. Sullivan began his career at Arthur Andersen LLP, where he obtained his certified public accountant license. Mr. Sullivan holds an M.B.A. and an M.S. in accounting from Northeastern University's Graduate School of Professional Accounting and a B.A. in economics from Williams College.
Peter K. Sorger, Ph.D.	Chairman of Scientific Advisory Board	Dr. Sorger is a Professor of Systems Biology at Harvard Medical School and holds a joint appointment in MIT's Dept. of Biological Engineering and Center for Cancer Research. A former Marshall and Markey scholar, he holds an A.B, from Harvard College and Ph.D. from the MRC Laboratory of Molecular Biology, and Trinity College, Cambridge, UK. Dr. Sorger's lab consists of 26 graduate students, postdoctoral fellows and staff scientists involved in both computational and experimental biology. Dr. Sorger has published over 70 scientific papers and holds several patents. Co-founder and first Director of the MIT Computational and Systems Biology Program (CSBi), Dr. Sorger is also a member of the Broad Institute, the MIT Center for Cancer Research and the Board of Directors of Applied Precision, LLC.

Source: Company Reports.

Other companies mentioned that are not covered by Oppenheimer & Co. (prices as of 5/4/12):

AstraZeneca (AZN-NYSE, $43.90, Not Rated)
Aveo Pharmaceuticals (AVEO, $11.31, Not Rated)
Daiichi Sankyo (4568-TYO, 1355 Yen, Not Rated)
Dyax (Dyax-NASD, $1.49, Not Rated)
Eli Lilly (LLY-NYSE, $41.28, Not Rated)
Enzon (ENZN-NASD, $5.86, Not Rated)
Roche (RHHBY-PINK, $44.95, Not Rated)
Sanofi (SNY-NYSE, $37.80, Not Rated)
Takeda (4502-TYO, 3495 Yen, Not Rated)

Financial Models

MM-121 Revenue Model

Breast Cancer											
MM-121	**2014**	**2015**	**2016**	**2017**	**2018**	**2019**	**2020**	**2021**	**2022**	**2023**	**2024**
US BC Incidence	234,580	234,861	235,143	235,425	235,707	235,990	236,273	236,556	236,840	237,124	237,409
US, ER/PR+ Her2-	118,932	119,075	119,217	119,360	119,504	119,647	119,790	119,934	120,078	120,222	120,366
Chemo (ER/PR positive)	44,081	44,133	44,186	44,239	44,292	44,346	44,399	44,452	44,505	44,559	44,612
2nd-line ER/PR positive chemo	13,224	13,240	13,256	13,272	13,288	13,304	13,320	13,336	13,352	13,368	13,384
Penetration	0%	0%	0%	5%	10%	13%	15%	23%	29%	33%	35%
# of patients	-	-	-	664	1,329	1,663	1,998	3,067	3,872	4,411	4,684
Avg # of courses/patient	8	8	8	8	8	8	8	8	8	8	8
Avg price/patient	$51,680.3	$52,713.9	$53,768.1	$54,843.5	$55,940.4	$57,059.2	$58,200.4	$59,364.4	$60,551.7	$61,762.7	$62,998.0
Revenues (2nd-line chemo ER/PR+	**$0.0**	**$0.0**	**$0.0**	**$36.4**	**$74.3**	**$94.9**	**$116.3**	**$182.1**	**$234.5**	**$272.5**	**$295.1**
3rd-line ER/PR positive chemo	11,020	11,033	11,047	11,060	11,073	11,086	11,100	11,113	11,126	11,140	11,153
Penetration	0%	0%	0%	5%	10%	13%	15%	20%	25%	25%	25%
# of patients	-	-	-	553	1,107	1,386	1,665	2,223	2,782	2,785	2,788
Avg # of courses/patient	7	7	7	7	7	7	7	7	7	7	7
Avg price/patient	$45,220.2	$46,124.6	$47,047.1	$47,988.1	$48,947.8	$49,926.8	$50,925.3	$51,943.8	$52,982.7	$54,042.4	$55,123.2
Revenues (3rd-line chemo ER/PR+)	**$0.0**	**$0.0**	**$0.0**	**$26.5**	**$54.2**	**$69.2**	**$84.8**	**$115.5**	**$147.4**	**$150.5**	**$153.7**
US Revenues for MM-121	**$0.0**	**$0.0**	**$0.0**	**$62.9**	**$128.5**	**$164.1**	**$201.1**	**$297.5**	**$381.8**	**$423.0**	**$448.8**
US Royalty to MACK	**$0.0**	**$0.0**	**$0.0**	**$7.9**	**$16.1**	**$20.5**	**$25.1**	**$40.17**	**$51.5**	**$57.1**	**$60.6**
Europe, BC Incidence	338,542	338,948	339,354	339,761	340,169	340,577	340,985	341,394	341,804	342,214	342,624
Europe, ER/PR+ Her2-	171,641	171,847	172,053	172,259	172,466	172,672	172,880	173,087	173,294	173,502	173,710
Chemo (ER/PR positive)	63,616	63,693	63,769	63,845	63,922	63,999	64,075	64,152	64,229	64,306	64,383
2nd-line ER/PR positive chemo	19,085	19,108	19,131	19,154	19,177	19,200	19,223	19,246	19,269	19,292	19,315
Penetration	0%	0%	0%	5%	10%	13%	15%	22%	29%	32%	35%
# of patients	-	-	-	664	1,329	1,663	1,998	2,934	3,872	4,278	4,684
Avg # of courses/patient	10	10	10	10	10	10	10	10	10	10	10
Avg price/patient	$59,680.7	$59,680.7	$59,680.7	$59,680.7	$59,680.7	$59,680.7	$59,680.7	$59,680.7	$59,680.7	$59,680.7	$59,680.7
Revenues (2nd-line chemo ER/PR+	**$0.0**	**$0.0**	**$0.0**	**$39.6**	**$79.3**	**$99.2**	**$119.2**	**$175.1**	**$231.1**	**$255.3**	**$279.6**
3rd-line ER/PR positive chemo	15,904	15,923	15,942	15,961	15,981	16,000	16,019	16,038	16,057	16,077	16,096
Penetration	0%	0%	0%	5%	10%	13%	15%	20%	25%	25%	25%
# of patients	-	-	-	553	1,107	1,386	1,665	2,223	2,782	2,785	2,788
Avg # of courses/patient	10	10	10	10	10	10	10	10	10	10	10
Avg price/patient	$59,680.7	$59,680.7	$59,680.7	$59,680.7	$59,680.7	$59,680.7	$59,680.7	$59,680.7	$59,680.7	$59,680.7	$59,680.7
Revenues (3rd-line chemo ER/PR+)	**$0.0**	**$0.0**	**$0.0**	**$33.0**	**$66.1**	**$82.7**	**$99.4**	**$132.6**	**$166.0**	**$166.2**	**$166.4**
EU Revenues for MM-121	**$0.0**	**$0.0**	**$0.0**	**$72.6**	**$145.4**	**$182.0**	**$218.6**	**$307.7**	**$397.1**	**$421.5**	**$446.0**
EU Royalty to MACK	**$0.0**	**$0.0**	**$0.0**	**$5.4**	**$10.9**	**$13.6**	**$16.4**	**$26.16**	**$33.8**	**$35.8**	**$37.9**
WW Revenues for MM-121	**$0**	**$0**	**$0**	**$136**	**$274**	**$346**	**$420**	**$605**	**$779**	**$844**	**$895**
WW Royalty to MACK	**$0**	**$0**	**$0**	**$13**	**$27**	**$34**	**$42**	**$66**	**$85**	**$93**	**$98**

NSCLC											
MM-121	**2014**	**2015**	**2016**	**2017**	**2018**	**2019**	**2020**	**2021**	**2022**	**2023**	**2024**
US											
Lung cancer incidence	223,126	223,796	224,467	225,140	225,816	226,493	227,173	227,854	228,538	229,223	229,911
% growth	0.3%	0.3%	0.3%	0.3%	0.3%	0.3%	0.3%	0.3%	0.3%	0.3%	0.3%
NSCLC incidence	189,657	190,226	190,797	191,369	191,943	192,519	193,097	193,676	194,257	194,840	195,424
% non-small cell	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*
NSCLC, adv/metastatic	142,243	142,670	143,098	143,527	143,958	144,389	144,823	145,257	145,693	146,130	146,568
%adv/metstatic	*75%*	*75%*	*75%*	*75%*	*75%*	*75%*	*75%*	*75%*	*75%*	*75%*	*75%*
Patients failing chemotherapy	123,277	123,647	124,018	124,390	124,763	125,137	125,513	125,889	126,267	126,646	127,026
%failure on ≥ 1 chemotherapy	*65%*	*65%*	*65%*	*65%*	*65%*	*65%*	*65%*	*65%*	*65%*	*65%*	*65%*
Patients on Tarceva (2nd/3rd-line)	24,655	24,729	24,804	24,878	24,953	25,027	25,103	25,178	25,253	25,329	25,405
%treated	*20%*	*20%*	*20%*	*20%*	*20%*	*20%*	*20%*	*20%*	*20%*	*20%*	*20%*
Patients on MM-121+Tarceva	0	0	0	498	1,248	1,752	3,765	4,658	5,303	5,826	6,097
% penetration	*0.0%*	*0.0%*	*0.0%*	*2.0%*	*5.0%*	*7.0%*	*15.0%*	*18.5%*	*21.0%*	*23.0%*	*24.0%*
Duration of therapy	5.0	5.0	5.0	5.0	5.0	5.0	5.0	5.0	5.0	5.0	5.0
Monthly price	$32,300	$32,946	$33,605	$34,277	$34,963	$35,662	$36,375	$37,103	$37,845	$38,602	$39,374
YoY price increase	2%	2%	2%	2%	2%	2%	2%	2%	2%	2%	2%
U.S. sales ($M)	**$0**	**$0**	**$0**	**$17**	**$44**	**$62**	**$137**	**$173**	**$201**	**$225**	**$240**
Royalty to Merrimack	**$0**	**$0**	**$0**	**$2**	**$5**	**$8**	**$17**	**$23**	**$27**	**$30**	**$32**
Europe											
Lung cancer incidence	234,189	234,892	235,597	236,303	237,012	237,723	238,436	239,152	239,869	240,589	241,311
% growth	0.3%	0.3%	0.3%	0.3%	0.3%	0.3%	0.3%	0.3%	0.3%	0.3%	0.3%
NSCLC incidence	199,061	199,658	200,257	200,858	201,460	202,065	202,671	203,279	203,889	204,501	205,114
% non-small cell	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*	*85%*
NSCLC, adv/metastatic	149,296	149,744	150,193	150,643	151,095	151,549	152,003	152,459	152,917	153,375	153,836
%adv/metstatic	*75%*	*75%*	*75%*	*75%*	*75%*	*75%*	*75%*	*75%*	*75%*	*75%*	*75%*
Patients failing chemotherapy	129,390	129,778	130,167	130,558	130,949	131,342	131,736	132,131	132,528	132,925	133,324
%failure on ≥ 1 chemotherapy	*65%*	*65%*	*65%*	*65%*	*65%*	*65%*	*65%*	*65%*	*65%*	*65%*	*65%*
Patients on Tarceva (2nd/3rd-line)	25,878	25,956	26,033	26,112	26,190	26,268	26,347	26,426	26,506	26,585	26,665
%treated	*20%*	*20%*	*20%*	*20%*	*20%*	*20%*	*20%*	*20%*	*20%*	*20%*	*20%*
Patients on MM-121+Tarceva	0	0	0	131	1,048	1,576	3,162	4,228	5,036	5,849	6,400
% penetration	*0.0%*	*0.0%*	*0.0%*	*0.5%*	*4.0%*	*6.0%*	*12.0%*	*16.0%*	*19.0%*	*22.0%*	*24.0%*
Duration of therapy	5.0	5.0	5.0	5.0	5.0	5.0	5.0	5.0	5.0	5.0	5.0
Monthly price	$29,840	$29,840	$29,840	$29,840	$29,840	$29,840	$29,840	$29,840	$29,840	$29,840	$29,840
YoY price increase	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%
E.U. sales ($M)	**$0**	**$0**	**$0**	**$4**	**$31**	**$47**	**$94**	**$126**	**$150**	**$175**	**$191**
Royalty to Merrimack	**$0**	**$0**	**$0**	**$0**	**$2**	**$4**	**$7**	**$11**	**$13**	**$15**	**$16**
WW sales ($M)	**$0**	**$0**	**$0**	**$21**	**$75**	**$110**	**$231**	**$299**	**$351**	**$399**	**$431**
Royalty to Merrimack	**$0**	**$0**	**$0**	**$2**	**$8**	**$11**	**$24**	**$34**	**$40**	**$45**	**$49**

Ovarian Cancer											
MM-121	**2014**	**2015**	**2016**	**2017**	**2018**	**2019**	**2020**	**2021**	**2022**	**2023**	**2024**
US											
Ovarian cancer incidence	22,189	22,255	22,322	22,389	22,456	22,523	22,591	22,659	22,727	22,795	22,863
% growth	0.30%	0.30%	0.30%	0.30%	0.30%	0.30%	0.30%	0.30%	0.30%	0.30%	0.30%
Advanced ovarian cancer	15,088	15,133	15,179	15,224	15,270	15,316	15,362	15,408	15,454	15,500	15,547
%metstatic	*68%*	*68%*	*68%*	*68%*	*68%*	*68%*	*68%*	*68%*	*68%*	*68%*	*68%*
Second-line patients	4,526	4,540	4,554	4,567	4,581	4,595	4,609	4,622	4,636	4,650	4,664
%failure rate	*30%*	*30%*	*30%*	*30%*	*30%*	*30%*	*30%*	*30%*	*30%*	*30%*	*30%*
Relapse patients	5,281	5,297	5,313	5,329	5,345	5,361	5,377	5,393	5,409	5,425	5,441
%relapse rate	35%	35%	35%	35%	35%	35%	35%	35%	35%	35%	35%
Patients on MM-121+paclitaxel	0	0	0	297	893	1,394	1,897	2,303	2,511	2,519	2,526
%penetration	0%	0%	0%	3%	9%	14%	19%	23%	25%	25%	25%
Avg # of courses/patient	7	7	7	7	7	7	7	7	7	7	7
Avg price/patient	$45,220	$46,125	$47,047	$47,988	$48,948	$49,927	$50,925	$51,944	$52,983	$54,042	$55,123
YoY price increase	2%	2%	2%	2%	2%	2%	2%	2%	2%	2%	2%
U.S. sales ($M)	**$0**	**$0**	**$0**	**$14**	**$44**	**$70**	**$97**	**$120**	**$133**	**$136**	**$139**
Royalty to Merrimack	**$0**	**$0**	**$0**	**$2**	**$5**	**$9**	**$12**	**$16**	**$18**	**$18**	**$19**
Europe											
Ovarian cancer incidence	36,243	36,352	36,461	36,570	36,680	36,790	36,901	37,011	37,122	37,234	37,345
% growth	0.30%	0.30%	0.30%	0.30%	0.30%	0.30%	0.30%	0.30%	0.30%	0.30%	0.30%
Advanced ovarian cancer	24,645	24,719	24,794	24,868	24,943	25,017	25,092	25,168	25,243	25,319	25,395
%metstatic	*68%*	*68%*	*68%*	*68%*	*68%*	*68%*	*68%*	*68%*	*68%*	*68%*	*68%*
Second-line patients	7,394	7,416	7,438	7,460	7,483	7,505	7,528	7,550	7,573	7,596	7,618
%failure rate	*30%*	*30%*	*30%*	*30%*	*30%*	*30%*	*30%*	*30%*	*30%*	*30%*	*30%*
Relapse patients	8,626	8,652	8,678	8,704	8,730	8,756	8,782	8,809	8,835	8,862	8,888
%relapse rate	35%	35%	35%	35%	35%	35%	35%	35%	35%	35%	35%
Patients on MM-121+paclitaxel	0	0	0	0	324	813	1,631	2,781	3,446	3,785	3,962
%penetration	0%	0%	0%	0%	2%	5%	10%	17%	21%	23%	24%
Avg # of courses/patient	7	7	7	7	7	7	7	7	7	7	7
Avg price/patient	$41,777	$41,777	$41,777	$41,777	$41,777	$41,777	$41,777	$41,777	$41,777	$41,777	$41,777
YoY price increase	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%
E.U. sales ($M)	**$0**	**$0**	**$0**	**$0**	**$14**	**$34**	**$68**	**$116**	**$144**	**$158**	**$166**
Royalty to Merrimack	**$0**	**$0**	**$0**	**$0**	**$1**	**$3**	**$5**	**$10**	**$12**	**$13**	**$14**
WW sales ($M)	**$0**	**$0**	**$0**	**$14**	**$57**	**$104**	**$165**	**$236**	**$277**	**$294**	**$305**
Royalty to Merrimack	**$0**	**$0**	**$0**	**$2**	**$6**	**$11**	**$17**	**$26**	**$30**	**$32**	**$33**

Source: Oppenheimer.

MM-389 Revenue Model

Pancreatic Cancer											
MM-398	**2014**	**2015**	**2016**	**2017**	**2018**	**2019**	**2020**	**2021**	**2022**	**2023**	**2024**
United States											
Pancreatic Cancer Incidence	45,161	45,680	46,206	46,738	47,276	47,820	48,370	48,927	49,490	50,060	50,636
First-line pancreatic cancer	27,096	27,408	27,724	28,043	28,365	28,692	29,022	29,356	29,694	30,036	30,381
Adjuvant chemo	4,516	4,568	4,621	4,674	4,728	4,782	4,837	4,893	4,949	5,006	5,064
Penetration in adjuvant	0%	0%	0%	1%	2%	3%	4%	5%	5%	5%	5%
Patients on adjuvant chemo	-	-	-	47	95	143	193	245	247	250	253
Metastatic (2/3 of 1st-line)	27,096	27,408	27,724	28,043	28,365	28,692	29,022	29,356	29,694	30,036	30,381
Penetration in metastatic 1st-line	0%	0%	0%	1%	4%	12%	18%	22%	24%	25%	25%
Patients (metastatic 1st-line)	-	-	-	280	1,135	3,443	5,224	6,458	7,127	7,509	7,595
Total 1st-line patients	-	-	-	327	1,229	3,586	5,417	6,703	7,374	7,759	7,849
Avg # of courses in 1st-line	7.5	7.5	7.5	7.5	7.5	7.5	7.5	7.5	7.5	7.5	7.5
Avg price / patient	$53,295	$54,361	$55,448	$56,557	$57,689	$58,842	$60,019	$61,220	$62,444	$63,693	$64,967
YoY price increase	2%	2%	2%	2%	2%	2%	2%	2%	2%	2%	2%
U.S. 1st-line revenues	**$0.0**	**$0.0**	**$0.0**	**$18.5**	**$70.9**	**$211.0**	**$325.2**	**$410.4**	**$460.5**	**$494.2**	**$509.9**
Total 2nd-line pancreatic	18,064	18,272	18,482	18,695	18,910	19,128	19,348	19,571	19,796	20,024	20,254
Pts that did not get MM398 in 1st-line	18,064	18,272	18,482	18,368	17,681	15,541	13,931	12,868	12,422	12,265	12,406
Penetration in 2nd-line	5%	9%	15%	20%	25%	28%	30%	31%	31%	31%	31%
Patients (2nd-line)	903	1,644	2,772	3,674	4,420	4,352	4,179	3,989	3,851	3,802	3,846
Avg # of courses in 2nd-line	5.0	5.0	5.0	5.0	5.0	5.0	5.0	5.0	5.0	5.0	5.0
Avg price / patient	$35,530	$36,241	$36,966	$37,705	$38,459	$39,228	$40,013	$40,813	$41,629	$42,462	$43,311
U.S. 2nd-line revenues	**$32.1**	**$59.6**	**$102.5**	**$138.5**	**$170.0**	**$170.7**	**$167.2**	**$162.8**	**$160.3**	**$161.4**	**$166.6**
U.S. sales ($M)	**$32.1**	**$59.6**	**$102.5**	**$157.0**	**$240.9**	**$381.7**	**$492.4**	**$573.2**	**$620.8**	**$655.6**	**$676.5**
Europe (EU5 only)											
Pancreatic Cancer Incidence	47,963	48,515	49,073	49,638	50,209	50,787	51,372	51,963	52,561	53,166	53,778
First-line pancreatic cancer	28,778	29,109	29,444	29,783	30,126	30,472	30,823	31,178	31,537	31,900	32,267
Adjuvant chemo	4,365	4,415	4,466	4,517	4,569	4,622	4,675	4,729	4,783	4,838	4,894
Penetration in adjuvant	0%	0%	0%	0%	1%	2%	3%	4%	5%	5%	5%
Patients on adjuvant chemo	-	-	-	-	46	92	140	189	239	242	245
Metastatic (2/3 of 1st-line)	28,778	29,109	29,444	29,783	30,126	30,472	30,823	31,178	31,537	31,900	32,267
Penetration in metastatic 1st-line	0%	0%	0%	0%	1%	4%	12%	18%	22%	24%	25%
Patients (metastatic 1st-line)	-	-	-	-	301	1,219	3,699	5,612	6,938	7,656	8,067
Total 1st-line patients	-	-	-	-	347	1,311	3,839	5,801	7,177	7,898	8,311
Avg # of courses in 1st-line	7.5	7.5	7.5	7.5	7.5	7.5	7.5	7.5	7.5	7.5	7.5
Avg price / patient	$49,237	$49,237	$49,237	$49,237	$49,237	$49,237	$49,237	$49,237	$49,237	$49,237	$49,237
YoY price increase	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%
E.U. 1st-line revenues	**$0.0**	**$0.0**	**$0.0**	**$0.0**	**$17.1**	**$64.6**	**$189.0**	**$285.6**	**$353.4**	**$388.9**	**$409.2**
Total 2nd-line pancreatic	19,185	19,406	19,629	19,855	20,084	20,315	20,549	20,785	21,024	21,266	21,511
Pts that did not get MM398 in 1st-line	19,185	19,406	19,629	19,855	19,737	19,004	16,710	14,984	13,847	13,369	13,200
Penetration in 2nd-line	0%	2%	7%	14%	18%	22%	26%	29%	30%	31%	31%
Patients (2nd-line)	-	388	1,374	2,780	3,553	4,181	4,345	4,345	4,154	4,144	4,092
Avg # of courses in 2nd-line	5.0	5.0	5.0	5.0	5.0	5.0	5.0	5.0	5.0	5.0	5.0
Avg price / patient	$32,824	$32,824	$32,824	$32,824	$32,824	$32,824	$32,824	$32,824	$32,824	$32,824	$32,824
E.U. 2nd-line revenues	**$0.0**	**$12.7**	**$45.1**	**$91.2**	**$116.6**	**$137.2**	**$142.6**	**$142.6**	**$136.4**	**$136.0**	**$134.3**
E.U. sales ($M)	**$0.0**	**$12.7**	**$45.1**	**$91.2**	**$133.7**	**$201.8**	**$331.6**	**$428.3**	**$489.7**	**$524.9**	**$543.5**
E.U. royalties owed to third party	**$0.0**	**$11.7**	**$41.5**	**$83.9**	**$123.0**	**$185.7**	**$305.1**	**$394.0**	**$450.6**	**$482.9**	**$500.1**
WW total revenue	**$32.1**	**$71.3**	**$144.0**	**$241.0**	**$363.9**	**$567.4**	**$797.5**	**$967.2**	**$1,071.3**	**$1,138.6**	**$1,176.5**

Colorectal Cancer											
MM-398	**2014**	**2015**	**2016**	**2017**	**2018**	**2019**	**2020**	**2021**	**2022**	**2023**	**2024**
United States											
Colorectal Cancer Incidence	137,016	135,646	134,289	132,946	131,617	130,301	128,998	127,708	126,431	125,166	123,915
Treated CRC	116,464	115,299	114,146	113,004	111,874	110,756	109,648	108,552	107,466	106,391	105,328
Chemotherapy	34,254	33,911	33,572	33,237	32,904	32,575	32,249	31,927	31,608	31,292	30,979
Total 2nd-line CRC	10,276	10,173	10,072	9,971	9,871	9,773	9,675	9,578	9,482	9,387	9,294
Penetration	0%	0%	0%	2%	3%	9%	17%	21%	25%	25%	25%
# of patients	-	-	-	274	543	1,612	3,015	3,688	4,346	4,303	4,260
Avg # of courses/patient	6	6	6	6	6	6	6	6	6	6	6
Avg price/patient	$42,636	$43,489	$44,359	$45,246	$46,151	$47,074	$48,015	$48,976	$49,955	$50,954	$51,973
Revenues (2nd-line CRC)	**$0.0**	**$0.0**	**$0.0**	**$12.4**	**$25.1**	**$75.9**	**$144.8**	**$180.6**	**$217.1**	**$219.2**	**$221.4**
Total 3rd-line CRC	5,138	5,087	5,036	4,985	4,936	4,886	4,837	4,789	4,741	4,694	4,647
Penetration	0%	0%	0%	3%	10%	15%	19%	20%	20%	20%	20%
# of patients	-	-	-	548	1,810	2,687	3,370	3,512	3,477	3,442	3,408
Avg # of courses/patient	4	4	4	4	4	4	4	4	4	4	4
Avg price/patient	$28,424	$28,993	$29,572	$30,164	$30,767	$31,383	$32,010	$32,650	$33,303	$33,969	$34,649
Revenues (3rd-line CRC)	**$0.0**	**$0.0**	**$0.0**	**$16.5**	**$55.7**	**$84.3**	**$107.9**	**$114.7**	**$115.8**	**$116.9**	**$118.1**
U.S. Total CRC Revenues	**0.0**	**0.0**	**0.0**	**28.9**	**80.7**	**160.2**	**252.7**	**295.3**	**332.9**	**336.2**	**339.5**
YoY price increase	2%	2%	2%	2%	2%	2%	2%	2%	2%	2%	2%
Europe											
Colorectal Cancer Incidence	224,258	222,015	219,795	217,597	215,421	213,267	211,134	209,023	206,933	204,863	202,815
Treated CRC	190,619	188,713	186,826	184,958	183,108	181,277	179,464	177,670	175,893	174,134	172,393
Chemotherapy	104,841	103,792	102,754	101,727	100,709	99,702	98,705	97,718	96,741	95,774	94,816
Total 2nd-line CRC	10,276	10,173	10,072	9,971	9,871	9,773	9,675	9,578	9,482	9,387	9,294
Penetration	0%	0%	0%	1%	2%	7%	12%	19%	23%	25%	25%
# of patients	-	-	-	183	362	1,254	2,128	3,336	3,998	4,303	4,260
Avg # of courses/patient	6	6	6	6	6	6	6	6	6	6	6
Avg price/patient	$39,389	$39,389	$39,389	$39,389	$39,389	$39,389	$39,389	$39,389	$39,389	$39,389	$39,389
Revenues (2nd-line CRC)	**$0.0**	**$0.0**	**$0.0**	**$7.2**	**$14.3**	**$49.4**	**$83.8**	**$131.4**	**$157.5**	**$169.5**	**$167.8**
Total 3rd-line CRC	15,726	15,569	15,413	15,259	15,106	14,955	14,806	14,658	14,511	14,366	14,222
Penetration	0%	0%	0%	3%	9%	14%	19%	20%	20%	20%	20%
# of patients	-	-	-	548	1,629	2,508	3,370	3,512	3,477	3,442	3,408
Avg # of courses/patient	4	4	4	4	4	4	4	4	4	4	4
Avg price/patient	$26,260	$26,260	$26,260	$26,260	$26,260	$26,260	$26,260	$26,260	$26,260	$26,260	$26,260
Revenues (3rd-line CRC)	**$0.0**	**$0.0**	**$0.0**	**$14.4**	**$42.8**	**$65.9**	**$88.5**	**$92.2**	**$91.3**	**$90.4**	**$89.5**
E.U. Total CRC Revenues	**0.0**	**0.0**	**0.0**	**21.6**	**57.0**	**115.3**	**172.3**	**223.6**	**248.8**	**259.9**	**257.3**
YoY price increase	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%
WW CRC Revenues	**0.0**	**0.0**	**0.0**	**50.5**	**137.8**	**275.5**	**425.0**	**518.9**	**581.7**	**596.0**	**596.7**

Source: Oppenheimer.

MM-111 Revenue Model

Breast Cancer											
MM-111	**2014**	**2015**	**2016**	**2017**	**2018**	**2019**	**2020**	**2021**	**2022**	**2023**	**2024**
US, HER2+	51,608	51,669	51,731	51,794	51,856	51,918	51,980	52,042	52,105	52,167	52,230
Chemotherapy HER2+	19,128	19,151	19,174	19,197	19,220	19,243	19,266	19,289	19,312	19,335	19,358
Total 1st-line HER2+	8,607	8,618	8,628	8,638	8,649	8,659	8,670	8,680	8,690	8,701	8,711
Penetration	0%	0%	0%	0%	0%	1%	2%	6%	8%	11%	15%
# of patients	-	-	-	-	-	87	173	521	695	957	1,307
Avg # of courses/patient	10	10	10	10	10	10	10	10	10	10	10
Avg price/patient	$64,600.3	$65,892.3	$67,210.2	$68,554.4	$69,925.5	$71,324.0	$72,750.5	$74,205.5	$75,689.6	$77,203.4	$78,747.4
Revenues (1st-line HER2+)	**$0.0**	**$0.0**	**$0.0**	**$0.0**	**$0.0**	**$6.2**	**$12.6**	**$38.6**	**$52.6**	**$73.9**	**$102.9**
Total 2nd-line HER2+	5,738	5,745	5,752	5,759	5,766	5,773	5,780	5,787	5,794	5,801	5,807
Penetration	0%	0%	0%	0%	1%	3%	7%	11%	13%	15%	18%
# of patients	-	-	-	-	58	173	405	637	753	870	1,016
Avg # of courses/patient	8	8	8	8	8	8	8	8	8	8	8
Avg price/patient	$51,680.3	$52,713.9	$53,768.1	$54,843.5	$55,940.4	$57,059.2	$58,200.4	$59,364.4	$60,551.7	$61,762.7	$62,998.0
Revenues (2nd-line HER2+)	**$0.0**	**$0.0**	**$0.0**	**$0.0**	**$3.2**	**$9.9**	**$23.5**	**$37.8**	**$45.6**	**$53.7**	**$64.0**
Total 3rd-line HER2+	4,782	4,788	4,793	4,799	4,805	4,811	4,816	4,822	4,828	4,834	4,840
Penetration	0%	0%	0%	0%	3%	8%	12%	17%	21%	25%	30%
# of patients	-	-	-	-	144	385	578	820	1,014	1,208	1,452
Avg # of courses/patient	6	6	6	6	6	6	6	6	6	6	6
Avg price/patient	$38,760.2	$39,535.4	$40,326.1	$41,132.6	$41,955.3	$42,794.4	$43,650.3	$44,523.3	$45,413.7	$46,322.0	$47,248.5
Revenues (3rd-line HER2+)	**$0.0**	**$0.0**	**$0.0**	**$0.0**	**$6.0**	**$16.5**	**$25.2**	**$36.5**	**$46.0**	**$56.0**	**$68.6**
Total Revenues (HER2+ chemo)	$0	$0	$0	$0	$9	$33	$61	$113	$144	$184	$236
Adjuvant Chemotherapy HER2+	32,480	32,519	32,558	32,597	32,636	32,675	32,714	32,754	32,793	32,832	32,872
Penetration	0%	0%	0%	0%	0%	0%	1%	2%	3%	7%	10%
# of patients	-	-	-	-	-	-	327	655	984	2,298	3,287
Avg # of courses/patient	12	12	12	12	12	12	12	12	12	12	12
Avg price/patient	$77,520.4	$79,070.8	$80,652.2	$82,265.3	$83,910.6	$85,588.8	$87,300.6	$89,046.6	$90,827.5	$92,644.0	$94,496.9
Revenues (Adjuvant HER2+)	**$0.0**	**$0.0**	**$0.0**	**$0.0**	**$0.0**	**$0.0**	**$28.6**	**$58.3**	**$89.4**	**$212.9**	**$310.6**
US revenues for MM-111	**$0.0**	**$0.0**	**$0.0**	**$0.0**	**$9.3**	**$32.5**	**$89.9**	**$171.3**	**$233.6**	**$396.5**	**$546.1**
Europe, HER2+	74,479	74,569	74,658	74,748	74,837	74,927	75,017	75,107	75,197	75,287	75,377
Chemotherapy HER2+	27,605	27,638	27,671	27,704	27,737	27,771	27,804	27,837	27,871	27,904	27,938
Total 1st-line HER2+	12,422	12,437	12,452	12,467	12,482	12,497	12,512	12,527	12,542	12,557	12,572
Penetration	0%	0%	0%	0%	0%	1%	3%	6%	9%	12%	15%
# of patients	-	-	-	-	-	125	375	752	1,129	1,507	1,886
Avg # of courses/patient	10	10	10	10	10	10	10	10	10	10	10
Avg price/patient	$59,680.7	$59,680.7	$59,680.7	$59,680.7	$59,680.7	$59,680.7	$59,680.7	$59,680.7	$59,680.7	$59,680.7	$59,680.7
Revenues (1st-line HER2+)	**$0.0**	**$0.0**	**$0.0**	**$0.0**	**$0.0**	**$7.5**	**$22.4**	**$44.9**	**$67.4**	**$89.9**	**$112.5**
Total 2nd-line HER2+	8,281	8,291	8,301	8,311	8,321	8,331	8,341	8,351	8,361	8,371	8,381
Penetration	0%	0%	0%	0%	1%	3%	7%	11%	13%	16%	18%
# of patients	-	-	-	-	42	250	584	919	1,087	1,339	1,467
Avg # of courses/patient	8	8	8	8	8	8	8	8	8	8	8
Avg price/patient	$47,744.6	$47,744.6	$47,744.6	$47,744.6	$47,744.6	$47,744.6	$47,744.6	$47,744.6	$47,744.6	$47,744.6	$47,744.6
Revenues (2nd-line HER2+)	**$0.0**	**$0.0**	**$0.0**	**$0.0**	**$2.0**	**$11.9**	**$27.9**	**$43.9**	**$51.9**	**$63.9**	**$70.0**
Total 3rd-line HER2+	6,901	6,909	6,918	6,926	6,934	6,943	6,951	6,959	6,968	6,976	6,984
Penetration	0%	0%	0%	0%	2%	7%	12%	17%	21%	25%	30%
# of patients	-	-	-	-	139	486	834	1,183	1,463	1,744	2,095
Avg # of courses/patient	6	6	6	6	6	6	6	6	6	6	6
Avg price/patient	$35,808.4	$35,808.4	$35,808.4	$35,808.4	$35,808.4	$35,808.4	$35,808.4	$35,808.4	$35,808.4	$35,808.4	$35,808.4
Revenues (3rd-line HER2+)	**$0.0**	**$0.0**	**$0.0**	**$0.0**	**$5.0**	**$17.4**	**$29.9**	**$42.4**	**$52.4**	**$62.5**	**$75.0**
Total Revenues (HER2+ chemo)	-	-	-	-	7	37	80	131	172	216	258
Adjuvant Chemotherapy HER2+	46,874	46,931	46,987	47,043	47,100	47,156	47,213	47,269	47,326	47,383	47,440
Penetration	0%	0%	0%	0%	0%	0%	2%	4%	6%	8%	10%
# of patients	-	-	-	-	-	-	944	1,891	2,840	3,791	4,744
Avg # of courses/patient	12	12	12	12	12	12	12	12	12	12	12
Avg price/patient	$71,616.9	$71,616.9	$71,616.9	$71,616.9	$71,616.9	$71,616.9	$71,616.9	$71,616.9	$71,616.9	$71,616.9	$71,616.9
Revenues (Adjuvant HER2+)	**$0.0**	**$0.0**	**$0.0**	**$0.0**	**$0.0**	**$0.0**	**$67.6**	**$135.4**	**$203.4**	**$271.5**	**$339.7**
EU revenues for MM-111	**$0.0**	**$0.0**	**$0.0**	**$0.0**	**$7.0**	**$36.8**	**$147.8**	**$266.5**	**$375.0**	**$487.8**	**$597.4**
WW revenues for MM-111	**$0**	**$0**	**$0**	**$0**	**$16**	**$69**	**$238**	**$438**	**$609**	**$884**	**$1,144**

Source: Oppenheimer.

MM-302 Revenue Model

Breast Cancer											
MM-302	**2014**	**2015**	**2016**	**2017**	**2018**	**2019**	**2020**	**2021**	**2022**	**2023**	**2024**
US, HER2+	51,608	51,669	51,731	51,794	51,856	51,918	51,980	52,042	52,105	52,167	52,230
Chemotherapy HER2+	19,128	19,151	19,174	19,197	19,220	19,243	19,266	19,289	19,312	19,335	19,358
Total 1st-line HER2+	8,607	8,618	8,628	8,638	8,649	8,659	8,670	8,680	8,690	8,701	8,711
Penetration	0%	0%	0%	0%	0%	2%	5%	9%	12%	18%	25%
# of patients	-	-	-	-	-	173	433	781	1,043	1,566	2,178
Avg # of courses/patient	10	10	10	10	10	10	10	10	10	10	10
Avg price/patient	$64,600.3	$65,892.3	$67,210.2	$68,554.4	$69,925.5	$71,324.0	$72,750.5	$74,205.5	$75,689.6	$77,203.4	$78,747.4
Revenues (1st-line HER2+)	**$0.0**	**$0.0**	**$0.0**	**$0.0**	**$0.0**	**$12.4**	**$31.5**	**$58.0**	**$78.9**	**$120.9**	**$171.5**
Total 2nd-line HER2+	5,738	5,745	5,752	5,759	5,766	5,773	5,780	5,787	5,794	5,801	5,807
Penetration	0%	0%	0%	0%	3%	6%	10%	14%	18%	20%	20%
# of patients	-	-	-	-	173	346	578	810	1,043	1,160	1,161
Avg # of courses/patient	8	8	8	8	8	8	8	8	8	8	8
Avg price/patient	$51,680.3	$52,713.9	$53,768.1	$54,843.5	$55,940.4	$57,059.2	$58,200.4	$59,364.4	$60,551.7	$61,762.7	$62,998.0
Revenues (2nd-line HER2+)	**$0.0**	**$0.0**	**$0.0**	**$0.0**	**$9.7**	**$19.8**	**$33.6**	**$48.1**	**$63.1**	**$71.7**	**$73.2**
Total 3rd-line HER2+	4,782	4,788	4,793	4,799	4,805	4,811	4,816	4,822	4,828	4,834	4,840
Penetration	0%	0%	0%	0%	1%	3%	5%	7%	9%	10%	10%
# of patients	-	-	-	-	48	144	241	338	435	483	484
Avg # of courses/patient	6	6	6	6	6	6	6	6	6	6	6
Avg price/patient	$38,760.2	$39,535.4	$40,326.1	$41,132.6	$41,955.3	$42,794.4	$43,650.3	$44,523.3	$45,413.7	$46,322.0	$47,248.5
Revenues (3rd-line HER2+)	**$0.0**	**$0.0**	**$0.0**	**$0.0**	**$2.0**	**$6.2**	**$10.5**	**$15.0**	**$19.7**	**$22.4**	**$22.9**
Total Revenues (HER2+ chemo)	-	-	-	-	12	38	76	121	162	215	268
Adjuvant Chemotherapy HER2+	32,480	32,519	32,558	32,597	32,636	32,675	32,714	32,754	32,793	32,832	32,872
Penetration	0%	0%	0%	0%	0%	0%	3%	3%	8%	12%	15%
# of patients	-	-	-	-	-	-	981	983	2,623	3,940	4,931
Avg # of courses/patient	12	12	12	12	12	12	12	12	12	12	12
Avg price/patient	$77,520.4	$79,070.8	$80,652.2	$82,265.3	$83,910.6	$85,588.8	$87,300.6	$89,046.6	$90,827.5	$92,644.0	$94,496.9
Revenues (Adjuvant HER2+)	**$0.0**	**$0.0**	**$0.0**	**$0.0**	**$0.0**	**$0.0**	**$85.7**	**$87.5**	**$238.3**	**$365.0**	**$465.9**
US revenues for MM-302	**$0.0**	**$0.0**	**$0.0**	**$0.0**	**$11.7**	**$38.3**	**$161.4**	**$208.6**	**$400.1**	**$580.0**	**$733.5**
Europe, HER2+	74,479	74,569	74,658	74,748	74,837	74,927	75,017	75,107	75,197	75,287	75,377
Chemotherapy HER2+	27,605	27,638	27,671	27,704	27,737	27,771	27,804	27,837	27,871	27,904	27,938
Total 1st-line HER2+	12,422	12,437	12,452	12,467	12,482	12,497	12,512	12,527	12,542	12,557	12,572
Penetration	0%	0%	0%	0%	0%	2%	5%	9%	12%	19%	24%
# of patients	-	-	-	-	-	250	626	1,127	1,505	2,386	3,017
Avg # of courses/patient	10	10	10	10	10	10	10	10	10	10	10
Avg price/patient	$59,680.7	$59,680.7	$59,680.7	$59,680.7	$59,680.7	$59,680.7	$59,680.7	$59,680.7	$59,680.7	$59,680.7	$59,680.7
Revenues (1st-line HER2+)	**$0.0**	**$0.0**	**$0.0**	**$0.0**	**$0.0**	**$14.9**	**$37.3**	**$67.3**	**$89.8**	**$142.4**	**$180.1**
Total 2nd-line HER2+	8,281	8,291	8,301	8,311	8,321	8,331	8,341	8,351	8,361	8,371	8,381
Penetration	0%	0%	0%	0%	2%	6%	10%	14%	18%	20%	20%
# of patients	-	-	-	-	166	500	834	1,169	1,505	1,674	1,676
Avg # of courses/patient	8	8	8	8	8	8	8	8	8	8	8
Avg price/patient	$47,744.6	$47,744.6	$47,744.6	$47,744.6	$47,744.6	$47,744.6	$47,744.6	$47,744.6	$47,744.6	$47,744.6	$47,744.6
Revenues (2nd-line HER2+)	**$0.0**	**$0.0**	**$0.0**	**$0.0**	**$7.9**	**$23.9**	**$39.8**	**$55.8**	**$71.9**	**$79.9**	**$80.0**
Total 3rd-line HER2+	6,901	6,909	6,918	6,926	6,934	6,943	6,951	6,959	6,968	6,976	6,984
Penetration	0%	0%	0%	0%	1%	3%	5%	7%	9%	10%	10%
# of patients	-	-	-	-	69	208	348	487	627	698	698
Avg # of courses/patient	6	6	6	6	6	6	6	6	6	6	6
Avg price/patient	$35,808.4	$35,808.4	$35,808.4	$35,808.4	$35,808.4	$35,808.4	$35,808.4	$35,808.4	$35,808.4	$35,808.4	$35,808.4
Revenues (3rd-line HER2+)	**$0.0**	**$0.0**	**$0.0**	**$0.0**	**$2.5**	**$7.5**	**$12.4**	**$17.4**	**$22.5**	**$25.0**	**$25.0**
Total Revenues (HER2+ chemo)	**$0.0**	**$0.0**	**$0.0**	**$0.0**	**$10.4**	**$46.2**	**$89.6**	**$140.6**	**$184.1**	**$247.3**	**$285.1**
Adjuvant Chemotherapy HER2+	46,874	46,931	46,987	47,043	47,100	47,156	47,213	47,269	47,326	47,383	47,440
Penetration	0%	0%	0%	0%	0%	0%	4%	9%	13%	14%	15%
# of patients	-	-	-	-	-	-	1,889	4,254	6,152	6,634	7,116
Avg # of courses/patient	12	12	12	12	12	12	12	12	12	12	12
Avg price/patient	$71,616.9	$71,616.9	$71,616.9	$71,616.9	$71,616.9	$71,616.9	$71,616.9	$71,616.9	$71,616.9	$71,616.9	$71,616.9
Revenues (Adjuvant HER2+)	**$0.0**	**$0.0**	**$0.0**	**$0.0**	**$0.0**	**$0.0**	**$135.2**	**$304.7**	**$440.6**	**$475.1**	**$509.6**
EU revenues for MM-302	**$0.0**	**$0.0**	**$0.0**	**$0.0**	**$10.4**	**$46.2**	**$224.9**	**$445.2**	**$624.7**	**$722.4**	**$794.7**
WW revenues for MM-302	**$0.0**	**$0.0**	**$0.0**	**$0.0**	**$22.1**	**$84.5**	**$386.2**	**$653.8**	**$1,024.8**	**$1,302.3**	**$1,528.2**

Source: Oppenheimer.

Merrimack Pharmaceuticals Income Statement 2009A-2017E

Amounts in thousands, except per-share figures

				2012									
	2009A	**2010A**	**2011A**	**1QE**	**2QE**	**3QE**	**4QE**	**2012E**	**2013E**	**2014E**	**2015E**	**2016E**	**2017E**
Revenues:													
MM-398 pancreatic cancer sales	-	-	-	-	-	-	-	-	-	32,091	71,318	143,976	240,960
MM-121 breast cancer royalties	-	-	-	-	-	-	-	-	-	-	-	-	13,312
Collaborative revenue (Sanofi)	2,148	20,305	34,215	12,000	12,200	12,400	12,600	49,200	50,700	55,200	57,200	59,763	68,575
Total operating revenue	**2,148**	**20,305**	**34,215**	**12,000**	**12,200**	**12,400**	**12,600**	**49,200**	**50,700**	**87,291**	**128,518**	**203,739**	**322,846**
Operating expenses													
Cost of goods	-	-	-	-	-	-	-	-	-	3,690	8,202	16,557	27,710
Research & development	37,658	58,278	100,630	28,084	29,045	30,258	31,374	118,760	124,791	130,447	136,134	139,604	143,108
Selling, general & administrative	12,178	11,381	14,454	3,750	3,950	4,250	4,650	16,600	24,070	44,530	57,888	69,466	76,413
Contingent consideration/other	-	(178)	-	-	-	-	-	-	-	-	-	-	-
Total operating expenses	**49,836**	**69,481**	**115,084**	**31,834**	**32,995**	**34,508**	**36,024**	**135,360**	**148,861**	**178,667**	**202,224**	**225,627**	**247,230**
Income (loss) from operations	**(47,688)**	**(49,176)**	**(80,869)**	**(19,834)**	**(20,795)**	**(22,108)**	**(23,424)**	**(86,160)**	**(98,161)**	**(91,375)**	**(73,706)**	**(21,888)**	**75,616**
Interest income	81	74	56	13	23	38	36	111	157	545	545	657	2,762
Interest expense	(4,909)	(3,726)	(13)	(4)	(4)	(4)	(4)	(16)	(21)	(31)	(47)	(75)	(120)
Other, net	41	2,669	1,150	(125)	(130)	(135)	(140)	(530)	(424)	(339)	(271)	(217)	(174)
Pretax income (loss)	**(52,475)**	**(50,159)**	**(79,676)**	**(19,950)**	**(20,905)**	**(22,209)**	**(23,532)**	**(86,595)**	**(98,449)**	**(91,200)**	**(73,479)**	**(21,523)**	**78,084**
Benefit from income taxes	3,402	-	-	-	-	-	-	-	-	-	-	-	(7,808)
Net income (loss)	**(49,073)**	**(50,159)**	**(79,676)**	**(19,950)**	**(20,905)**	**(22,209)**	**(23,532)**	**(86,595)**	**(98,449)**	**(91,200)**	**(73,479)**	**(21,523)**	**70,276**
Less net loss attributable to NCI	-	(55)	(453)	(110)	(116)	(122)	(128)	(475)	(508)	(534)	(555)	(572)	(589)
Net income attributed to Merrimack	**(49,073)**	**(50,104)**	**(79,223)**	**(19,839)**	**(20,790)**	**(22,087)**	**(23,404)**	**(86,120)**	**(97,941)**	**(90,666)**	**(72,924)**	**(20,951)**	**70,865**
Net loss per share	**($6.64)**	**($4.60)**	**($6.98)**	**($0.21)**	**($0.22)**	**($0.24)**	**($0.25)**	**($0.93)**	**($0.94)**	**($0.80)**	**($0.64)**	**($0.18)**	**$0.54**
Basic common shares outstanding	7,387	10,901	11,343	92,396	92,646	92,896	93,146	92,771	103,771	112,871	113,971	115,071	116,171
Diluted common shares outstanding	7,387	10,901	11,343	108,251	108,501	108,751	109,001	108,626	119,626	128,726	129,826	130,926	132,026
Margins													
Gross Margin	NM	NM	NM	NM	NM	NM	NM	NM	NM	11.5%	11.5%	11.5%	11.5%
R&D as percent of revenue	NM	NM	NM	NM	NM	NM	NM	NM	NM	149.4%	105.9%	68.5%	44.3%
G&A as percent of revenue	NM	NM	NM	NM	NM	NM	NM	NM	NM	51.0%	45.0%	34.1%	23.7%
Operating margin	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	23.4%
Pretax margin	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	24.2%
Profit margin	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	22.0%
Tax rate	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	10.0%

Source: Oppenheimer & Co.

Merrimack -- Balance Sheet

in thousands except per-share amounts

	2010A	2011A	2012E	2013E	2014E	2015E	2016E	2017E
Assets								
Cash and cash equivalents	30,713	50,454	71,129	83,908	92,520	31,240	21,621	104,339
Accounts receivable	3,745	7,426	7,723	8,032	8,353	8,687	9,035	9,396
Deferred financing cost	-	1,946	-	-	-	-	-	-
Prepaid expenses and other current assets	1,830	5,763	5,936	6,114	6,359	6,613	6,877	7,152
Current assets	36,288	65,589	84,788	98,054	107,232	46,540	37,533	120,887
Restricted cash	381	381	438	504	554	582	599	617
Property and equipment, net	7,458	6,206	7,758	9,309	10,705	11,241	11,803	12,393
Other assets	30	23	23	23	23	23	23	23
Intangible assets, net	2,805	2,485	2,485	2,485	2,485	2,485	2,485	2,485
In-process research and development	7,010	7,010	7,010	7,010	7,010	7,010	7,010	7,010
Goodwill	3,605	3,605	3,605	3,605	3,605	3,605	3,605	3,605
Total assets	**57,577**	**85,299**	**106,106**	**120,990**	**131,614**	**71,485**	**63,058**	**147,021**
Liabilities								
Accounts payable	1,440	4,656	4,796	4,940	5,088	5,240	5,398	5,560
Accrued expenses	7,256	12,855	13,241	13,638	14,047	14,468	14,902	15,350
Capital lease obligations	443	48	-	-	-	-	-	-
Deferred revenue	6,462	7,712	7,943	8,182	8,427	8,680	8,940	9,209
Deferred lease benefit	454	125	129	133	137	141	145	149
Deferred tax incentives	270	755	778	801	825	850	875	902
Total current liabilities	16,325	26,151	26,886	27,693	28,523	29,379	30,261	31,168
Capital lease obligations	48	-	-	-	-	-	-	-
Deferred revenues	67,320	78,033	80,374	82,785	85,269	87,827	90,462	93,175
Deferred lease benefits	102	23	24	24	25	26	27	27
Deferred tax incentives	810	1,267	1,305	1,344	1,384	1,426	1,469	1,513
Convertible preferred stock warrants	652	1,516	1,516	1,516	1,516	1,516	1,516	1,516
Total liabilities	**85,257**	**106,990**	**110,105**	**113,362**	**116,718**	**120,174**	**123,734**	**127,400**
Convertible preferred stock	191,257	268,225	-	-	-	-	-	-
Non-controlling interest	1,027	574	574	574	574	574	574	574
Common stick and APIC	51,652	60,349	432,862	542,937	641,406	651,300	660,836	670,857
Accumulated deficit	(271,616)	(350,839)	(437,434)	(535,883)	(627,084)	(700,563)	(722,086)	(651,810)
Shareholders' Equtiy	**(27,680)**	**(21,691)**	**(3,998)**	**7,627**	**14,896**	**(48,689)**	**(60,676)**	**19,620**

Source: Oppenheimer & Co.

Merrimack -- Cash Flow Statement

in thousands except per-share amounts

	2010A	2011A	2012E	2013E	2014E	2015E	2016E	2017E
Net loss	(50,159)	(79,676)	(86,595)	(98,449)	(91,200)	(73,479)	(21,523)	70,276
Noncash interest expense	3,673	-						
Depreciation and amortization	4,379	5,326	5,414	5,210	5,360	5,056	5,189	5,192
Stock-based compensation	4,551	6,952	6,768	6,699	7,147	7,078	6,769	7,417
Other items	(881)	134	-	-	-	-	-	-
Changes in working capital	12,068	14,447	1,441	1,332	1,172	1,080	997	923
Cashflow from operations	**(26,369)**	**(52,817)**	**(72,971)**	**(85,209)**	**(77,521)**	**(60,266)**	**(8,568)**	**83,807**
Capital expenditures	(4,999)	(3,754)	(1,552)	(1,552)	(1,396)	(535)	(562)	(590)
Acquisition/divestiture	-	-	-	-	-	-	-	-
Other	99	7	-	-	-	-	-	-
Cashflow from investing activities	**(4,900)**	**(3,747)**	**(1,552)**	**(1,552)**	**(1,396)**	**(535)**	**(562)**	**(590)**
Proceeds from Series G	-	76,949	-	-	-	-	-	-
Net issuance/repurchase of stock	294	1,745	95,650	100,000	88,000	-	-	-
Net issuance/repayment of debt	-	-	-	-	-	-	-	-
Capital lease obligations	(864)	(443)	(452)	(461)	(470)	(480)	(489)	(499)
Other	4,165	(1,946)	-	-	-	-	-	-
Cashflow from financing activities	**3,595**	**76,305**	**95,198**	**99,539**	**87,530**	**(480)**	**(489)**	**(499)**

Source: Oppenheimer & Co.

Investment Thesis

We are initiating coverage of MACK at Outperform, with a $12 PT. With a novel network biology platform, MACK has developed a broad pipeline of antibody-based and nanotherapeutic cancer therapies. We view MM-121, an ErbB3 antibody, as the most promising. We believe ErbB3 inhibition has applicability in several cancers, and MACK, with partner SNY, is evaluating MM-121 in a comprehensive ph.II program. MM-398, a liposomal irinotecan, has shown strong ph.II pancreatic cancer results, and we see a good probability of ph.III success, mid-'13. Additionally, MACK's earlier antibody-based compounds, MM-111/MM-302/MM-151, address blockbuster markets. We believe MACK is an attractive long-term value based on the company's pipeline/platform, and we would position in the stock ahead of key MM-121/MM-398 data in '13.

Price Target Calculation

Our 12-18 month price target of $12 is based on a sum-of-the parts NPV, which reflects value for MM-121, MM-398, MM-111, MM-302, MACK's technology platform and cash on hand. To determine the value of each clinical program, we performed a discounted cash flow valuation. For each program, our cash flows reflect a 15% discount rate and a 25-40% probability of success for clinical risk and are based on an out-year EBIT margin of ~45%.

Key Risks to Price Target

Key risks to our price target include, but are not limited to, clinical, regulatory, and commercial failure of MM-121, MM-398, MM-111 and MM-302. An additional risk includes an inability to raise future capital to support operations.

Important Disclosures and Certifications

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

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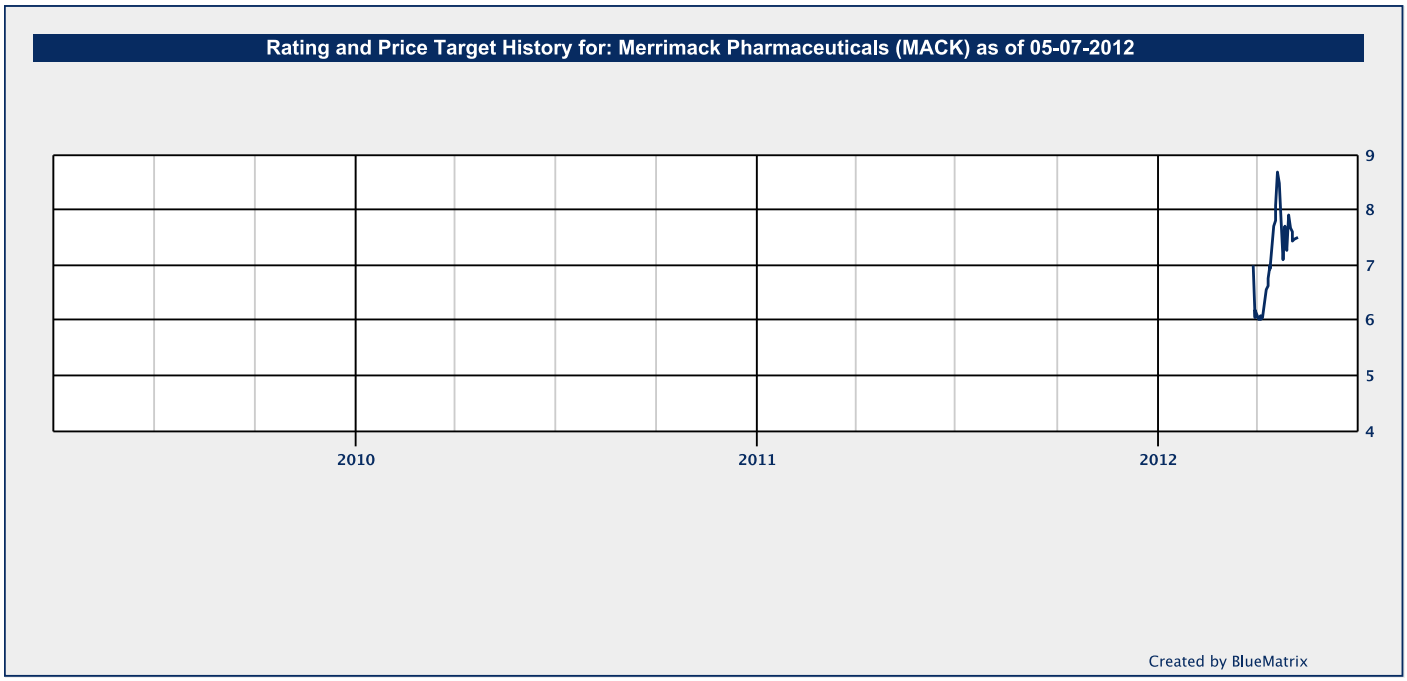
Stock Prices as of May 8, 2012

Amgen Inc. (AMGN - NASDAQ, 69.61, PERFORM)
Biogen Idec Inc. (BIIB - NASDAQ, 132.51, OUTPERFORM)
ImmunoGen, Inc. (IMGN - NASDAQ, 13.04, OUTPERFORM)
Seattle Genetics (SGEN - OTC, 19.20, OUTPERFORM)


Rating and Price Target History for: Merrimack Pharmaceuticals (MACK) as of 05-07-2012
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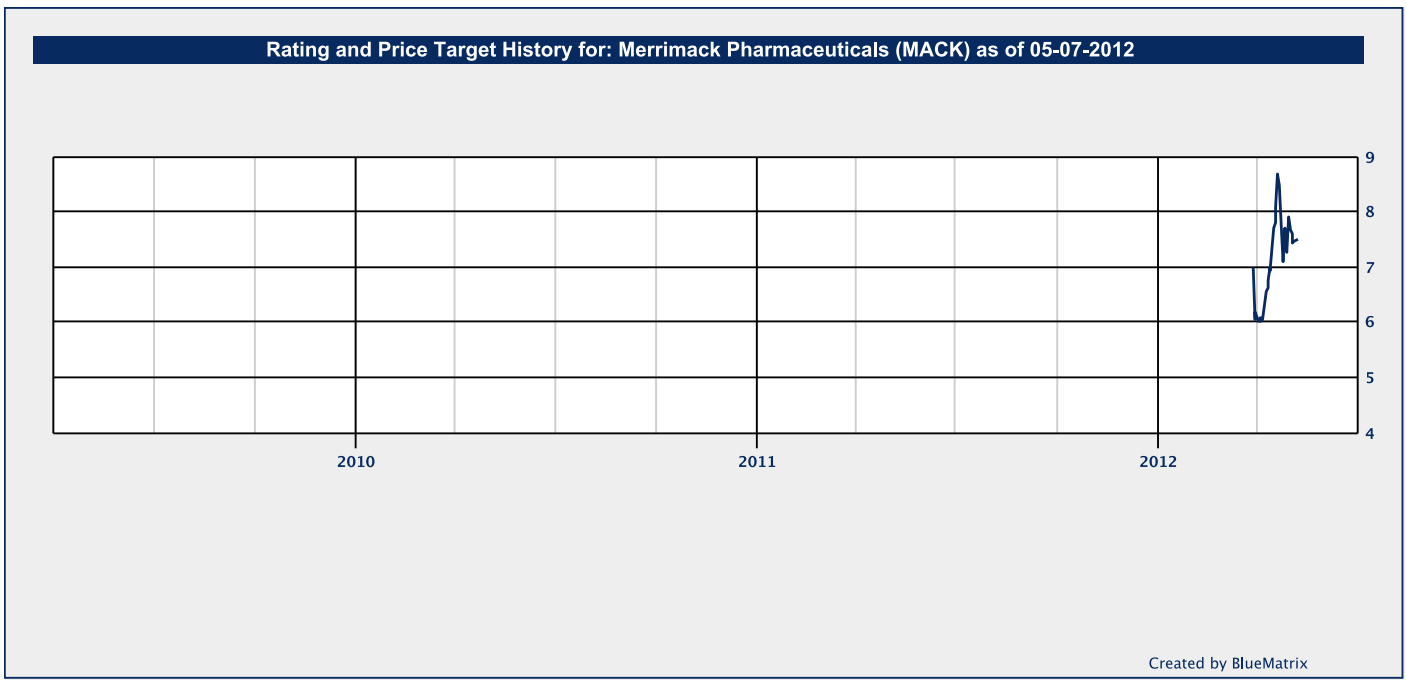
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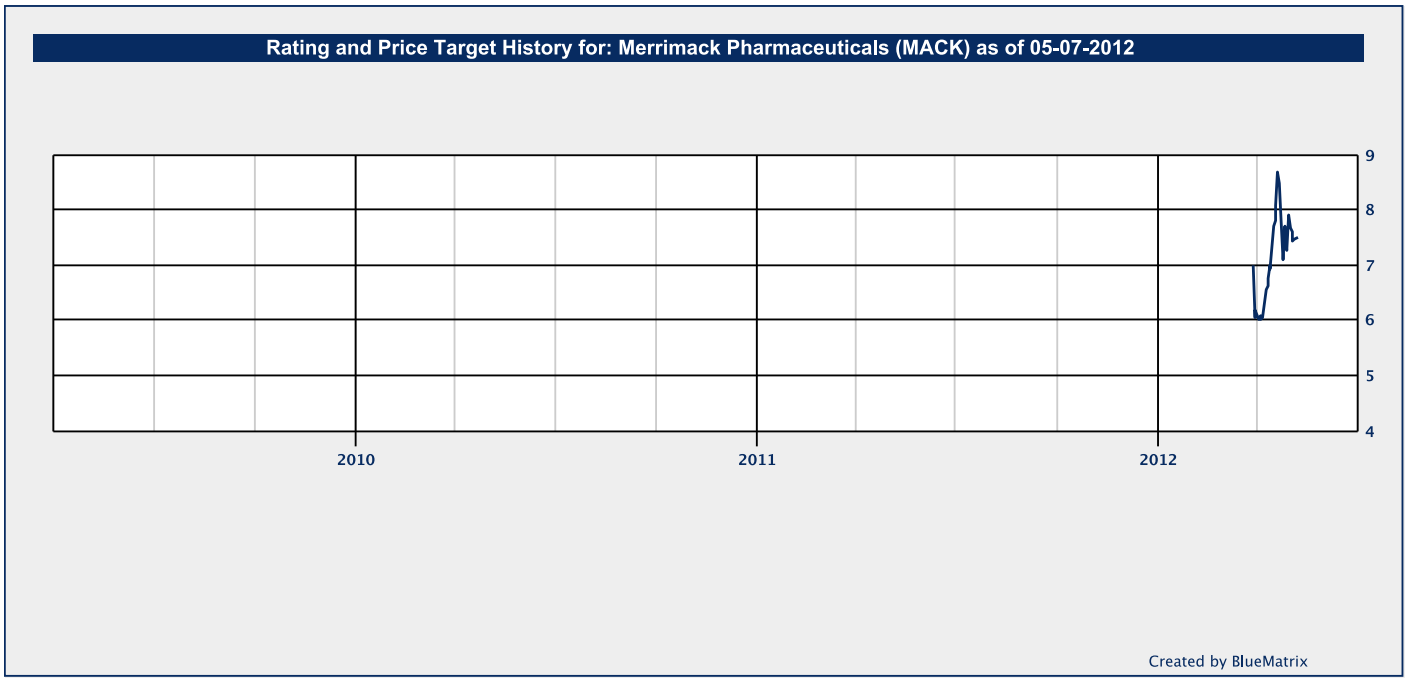
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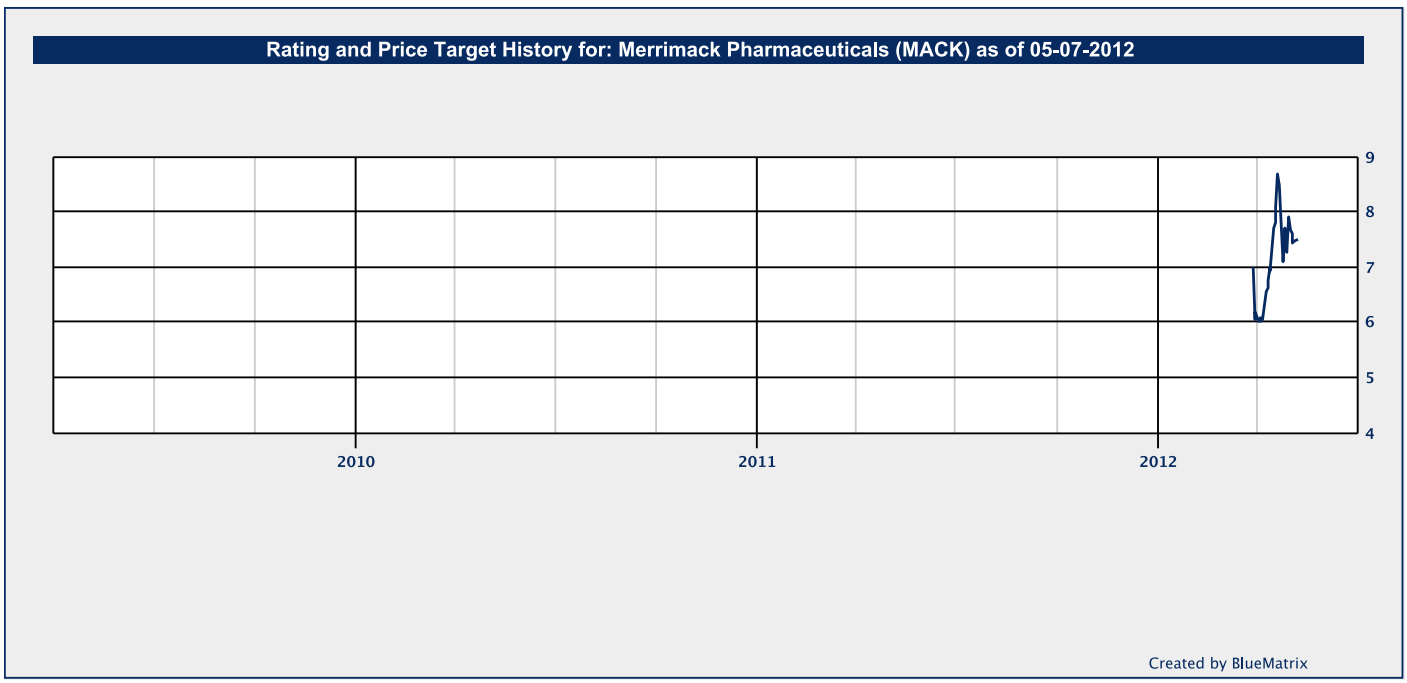
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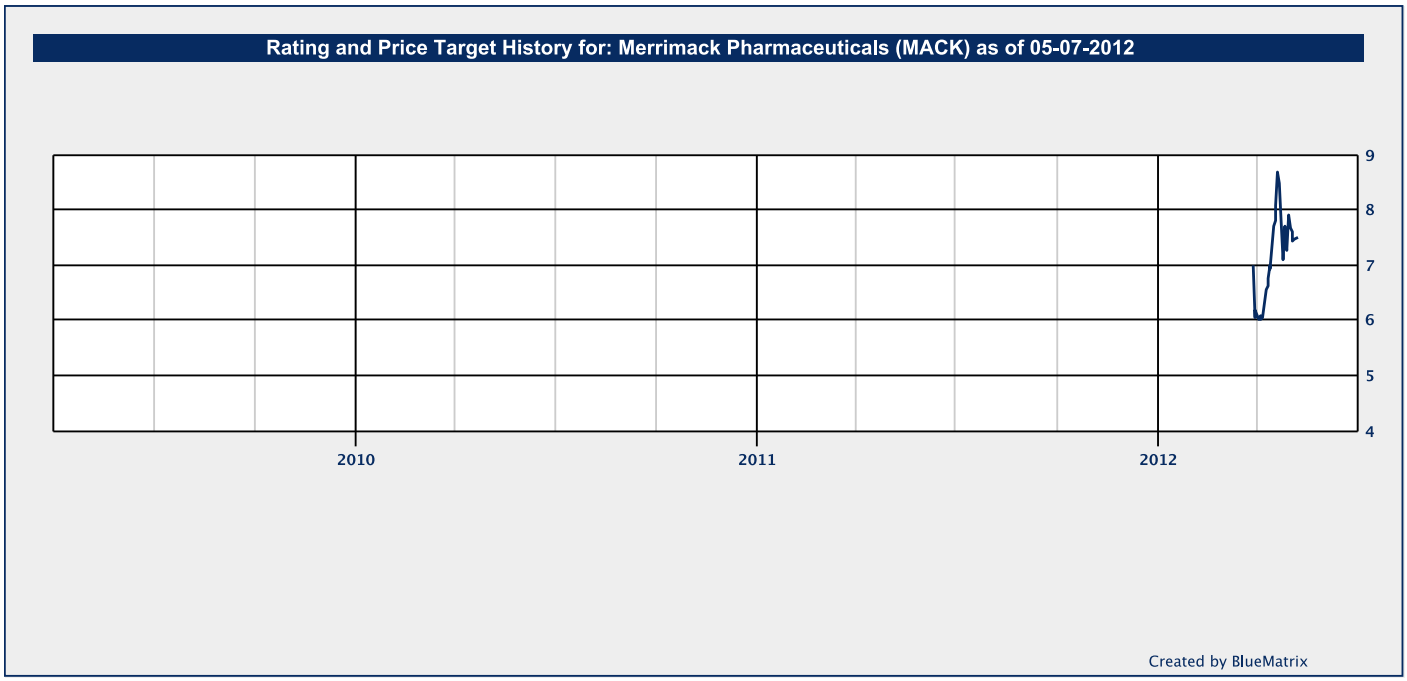

All price targets displayed in the chart above are for a 12- to- 18-month period. Prior to March 30, 2004, Oppenheimer & Co. Inc. used 6-, 12-, 12- to 18-, and 12- to 24-month price targets and ranges. For more information about target price histories, please write to Oppenheimer & Co. Inc., 85 Broad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

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Distribution of Ratings/IB Services Firmwide

			IB Serv/Past 12 Mos.	
Rating	Count	Percent	Count	Percent
OUTPERFORM [O]	323	55.59	144	44.58
PERFORM [P]	250	43.03	86	34.40
UNDERPERFORM [U]	8	1.38	3	37.50

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