COWEN AND COMPANY

Merrimack Pharmaceuticals

Initiating With Outperform (1)

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A Modern Era Oncology Company

Conclusion: We are initiating coverage of Merrimack Pharmaceuticals with an Outperform rating. The company is developing therapeutics and companion diagnostics for oncology utilizing a novel Network biology-based approach. Merrimack has a pipeline of eight biologic and nanoparticle-based drug candidates, five of which are in the clinic. We expect steady newsflow to drive outperformance relative to the market over the next 12 months.

- **MACK: Like A Knife With Multiple Tools.** A deeper understanding of signaling networks (as opposed to single gene defects) has enabled Merrimack to identify targets with broad application in oncology. The company's tool kit, comprised of technologies that enable the design and production of monoclonal, bi-specific, and polyclonal antibodies, and a variety of nanotherapeutics, allows for an optimal method of intervention. Diagnostic capabilities should facilitate smarter, more personalized clinical trial design.

- **MM-121 and MM-111 Curb The ErbB Pathway.** MM-121 is an antibody that blocks ErbB3's ability to co-stimulate signaling through the EGFR and HER2 receptors. In partnership with Sanofi, Merrimack has shown that MM-121 is active in a variety of tumor types (breast, ovarian, lung). Multiple Phase II trials are underway. MM-111 is a bi-specific antibody directed at blocking the HER2/ErbB3 interaction and overcoming resistance to Herceptin/Tykerb. This wholly owned candidate is in multiple Phase I trials.

- **MM-398 In Phase III For Pancreatic Cancer.** MM-398 is a liposomal formulation of irinotecan designed to enable differential uptake in tumors. A 270-patient Phase III trial in second line pancreatic cancer is recruiting patients and could provided data in late 2013. Promising data from a Phase I trial in second-line colorectal cancer support an ongoing Phase II trial.

MACK (05/07)	**$7.50**
Mkt cap	**$697.5MM**
Dil shares out	93.0MM
Avg daily vol	77.9K
52-wk range	$5.8-9.0
Dividend	Nil
Dividend yield	Nil
BV/sh	$1.11
Net cash/sh	$1.11
Debt/cap	NA
ROE (LTM)	NA
5-yr fwd EPS growth (Norm)	NA
S&P 500	**1369.6**

Revenue $MM

FY Dec	2011 Actual	2012E Prior	2012E Current	2013E Prior	2013E Current	2014E Current	2015E Current
Q1	0.0	—	10.0	—	—	—	—
Q2	0.0	—	10.0	—	—	—	—
Q3	0.0	—	10.0	—	—	—	—
Q4	0.0	—	10.0	—	—	—	—
Year	**34.2**	**—**	**40.0**	**—**	**35.0**	**25.0**	**72.0**
EV/S	—	—	15.0x	—	17.1x	24.0x	8.3x

EPS $

FY Dec	2011 Actual	2012E Prior	2012E Current	2013E Prior	2013E Current	2014E Current	2015E Current
Q1	—	—	(0.26)	—	—	—	—
Q2	—	—	(0.24)	—	—	—	—
Q3	—	—	(0.24)	—	—	—	—
Q4	—	—	(0.24)	—	—	—	—
Year	**1.05**	**—**	**(0.97)**	**—**	**(0.90)**	**(0.95)**	**(0.95)**
P/E	—	—	—	—	—	—	—

Investment Summary

Merrimack Pharmaceuticals has aspirations of becoming a fully-integrated, global biopharmaceutical company. Merrimack currently has broad capabilities in antibody design/engineering/development, biologics manufacturing, nanotherapeutics, and diagnostics. Merrimack's therapeutic pipeline of five clinical and three preclinical candidates is directed exclusively at oncology, and many of these candidates are derived from a superior understanding of network biology. With one exception (MM-121 partnered with Sanofi), Merrimack retains full ownership of this pipeline. Following a $100MM IPO completed in March, Merrimack is funded through 2013 assuming no new business development activity. We expect multiple value creating milestones to drive stock outperformance over this time period.

Merrimack Pharmaceuticals R&D Pipeline

Candidate	Indication	P-C	I	II	III	FILING	MKT	Comments
MM-121								**ErbB3 Ab; partnered with Sanofi**
	NSCLC			•				With Tarceva
	Ovarian Cancer			•				With paclitaxel
	ER+/PR+ breast cancer			•				With exemestane
	Neo-adjuvant breast cancer			•				With paclitaxel
	Advanced solid tumors		•					
	Advanced solid tumors		•					With Erbitux and irinotecan
MM-111								**Bispecific ErbB3 & HER2 Ab**
	Advanced HER2(+) cancers		•					
MM-151								**Mixture of 3 Abs To HER2**
	Refractory solid tumors		•					
MM-398								**Liposomal Irinotecan**
	Advanced Pancreatic Cancer				•			Second-line setting
	Colorectal cancer			•				Combined with 5-FU/LV, vs. FOLFIRI
	Colorectal cancer		•					In oxaliplatin-resistant patients
	Glioma		•					
MM-302								**Liposomal Doxorubicin + HER2 Ab**
	HER2(+) breast cancer		•					
MM-141	Cancer	•						IGF-1R + ErbB3 bispecific Ab
MM-131	Cancer	•						Undisclosed
MM-310	Cancer	•						Undisclosed
Total		**3**	**7**	**5**	**1**			

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Source: Cowen and Company

Merrimack Pharmaceuticals Upcoming Milestones

Milestone	Timing
Begin MM-111 Phase II trial in HER2(+) breast cancer	Mid:12
Full data from MM-111's Phase I multi-arm combination trial	H2:12
Begin first Phase I trial of MM-141	H2:12
Data from MM-121's Phase II ER+/PR+ breast cancer trial	H1:13
Data from MM-121's Phase II NSCLC trial (patient group with mutant EGFR and EGFR inhibitor resistance)	H1:13
Data from MM-121's Phase II NSCLC trial (patient group with wild-type EGFR and EGFR inhibitor naive)	H1:13
Data from MM-302's Phase I trial in HER2(+) breast cancer	H1:13
Begin first Phase I trial of MM-301	H1:13
Data from MM-398's Phase III pancreatic cancer trial	Mid:13
Data from MM-398's Phase II colorectal cancer trial	Mid:13
Data from MM-121's Phase II neoadjuvant HER2(-) breast cancer trial	H2:13
Data from MM-151's Phase I trial	H2:13
Data from MM-141's Phase I trial	H2:13
Begin first Phase I trial of MM-131	H2:13

Source: Cowen and Company

Networking To Better Targets

Merrimack was founded on the view that Network biology (sometimes referred to as "Systems biology") can provide unique biological insights that are important for drug discovery. The basic premise behind Network biology is that diseases tend to be caused by a complex network of protein signaling pathways, as opposed to a single gene defect. Historically much of drug development in areas like oncology has been heavily reliant on the view that a single gene mutation (e.g. c-kit, EGFR, BRAF, Alk, etc) can code for a single mutant protein, driving over activation of a single pathway. This approach has been successful in that it has enabled targeted therapies like Gleevec, Tarceva, Zelboraf, and Xalkori, drugs that represent significant advances in several specific indications. However, in many cases the underlying biology behind tumor growth is much more complex, and involves an interplay of multiple pathways. Merrimack's view is that in order to successfully treat a majority of cancers, one needs to map out all of the functional defects that underlie tumorigenesis (i.e. which pathways are over activated, which are responsible for creating resistance, what is the optimal site of inhibition). While definitive validation of Merrimack's strategy is dependent upon commercial success, early clinical trial data provide proof-of-concept that Network biology can successfully identify targets that are not otherwise obvious.

Once a target for intervention has been identified, Merrimack employs its tool kit of technologies (expertise in monoclonal antibodies, multi-specific antibodies, polyclonal antibodies, nanotherapeutics) to create an optimal therapeutic.

The importance of individualized therapy is a key corollary to Merrimack's approach in Network biology. Different patients will have tumors that are driven by different pathways. Hence Merrimack intends to offer diagnostics that enable patient stratification in clinical trials and a more personalized approach to therapy in the commercial setting.

Lead Candidates Block HER Signaling

The ErbB signaling pathway in breast cancer provides an excellent example for the utility of Network biology in drug discovery. There are four receptors in the ErbB

signaling pathway (EGFR/ErbB1, HER2/ErbB2, HER3/ErbB3, and HER4/ErbB4). The important role played by EGFR (the target for Tarceva, Erbitux, Vectibix) and HER2 (Herceptin, Tykerb) in cancer cell growth has been known for some time. However Network biology allowed Merrimack to identify ErbB3 (a.k.a. HER3) as another important target in this pathway. This led Merrimack to design an inhibitor of ErbB3 (MM-121) that shuts down ErbB signaling across all axes. Insights from Network biology also enabled the creation of an agent (MM-111) directed at overcoming resistance to anti-HER2 therapies like Herceptin.

MM-121 targets ErbB3. MM-121 is a first-in-class human monoclonal antibody directed at ErbB3 (HER3). The candidate was designed to inhibit heterodimerization between ErbB3 and both EGFR and HER2. Phase I data in combination with paclitaxel (in breast and ovarian cancer) and in combination with Tarceva (in lung cancer) suggest broad anti-cancer activity. MM-121 has been partnered to Sanofi in a potential $530MM deal ($60MM upfront) and Merrimack stands to collect tiered double digit royalties. Sanofi is putting considerable resources behind MM-121, fully funding four Phase II and four Phase I trials.

MM-111 targets ErbB2 and ErbB3. MM-111 is a bi-specific antibody directed at HER2 and ErbB3. Whereas Genentech/Roche's pertuzumab has provided proof of concept that disrupting HER2/ErbB3 heterodimerization can restore sensitivity to HER2 inhibitors such as Heceptin, Merrimack believes MM-111 provides more complete blockade of HER2/ErbB3 dimerization, making tumors that express HER2 at lower levels more sensitive to drugs such as Herceptin and Tykerb. MM-111 is wholly-owned and Phase I trials in combination with Herceptin have suggested significant activity in patients resistant to Herceptin. Several Phase I trials are ongoing, and Phase II studies are being planned.

MM-398 Looks To Improve Upon Irinotecan

MM-398, a liposomal formulation of irinotecan, is Merrimack's most advanced candidate. Although MM-398 did not originate from Merrimack's Network biology platform, the company optimized its formulation to enable preferential uptake via the leaky vasculature that is often associated with tumor cells. MM-398 is in a Phase III trial designed to compare it efficacy vs. 5-FU/leucovorin in pancreatic cancer patients who have failed gemicitabine. The trial is enrolling 270-patients with data on the primary endpoint (overall survival) possible in mid-2013. Support for the trial comes from a 40-patient trial median PFS of 9.6 weeks and OS of 22.4 weeks. Merrimack has also presented favorable data from a Phase I trial in second-line colorectal cancer showing 20%+ response rate and a Phase II study in this tumor type is ongoing. Peak sales of irinotecan (Camptosar, Pfizer) approximated $1B. Pending successful development, MM-398 could have similar potential.

Multiple Other Shots On Goal

Merrimack has two additional clinical pipeline candidates: MM-151, an oligoclonal EGFR antibody that seeks to improve upon Erbitux and Vectibix; and MM-302, an encapsulated formulation of doxorubicin that is directed at cells overexpressing HER2. Neither compound has produced proof-of-concept results, though Phase I data on MM-151 both are expected in 2013. Three additional candidates are heading toward the clinic, including MM-141 (a bi-specific antibody against IGF-1R and ErbB3), MM-131 (a multi-specific antibody directed at undisclosed targets) and MM-310 (an undisclosed antibody-targeted nanotherapeutic).

What Are Shares Worth?

Following the company's March IPO, Merrimack has roughly 93M basic and 113MM fully diluted shares outstanding and estimated cash on hand of approximately $100-125MM, equating to a (fully diluted) enterprise value of approximately $725MM.

Based upon our discussions with investors during the company's IPO roadshow, it is clear that most viewed the company as having many favorable characteristics (discovery platform, Ab generation capability, broad/renewable pipeline, oncology focus, strong management, etc) and considerable potential. However, valuation was a stumbling block for many potential investors. We don't claim to have the magic formula that proves MACK shares are undervalued. However, we believe that within Merrimack are several valuable assets that tend to be overlooked when viewing the company as a whole. The sum-of the parts valuation below is admittedly somewhat subjective. However, it illustrates the point that one need not assign massive contribution to any one asset in this unique enterprise in order to be favorably inclined on valuation. We recommend that investors purchase MACK shares as we believe growing investor understanding of and confidence in the company's pipeline will drive stock price appreciation.

Merrimack Pharmaceuticals Sum-Of-The-Parts Valuation

Asset	Value	Value/share
MM-121	$250MM	$2.21
MM-111	$250MM	$2.21
MM-398	$250MM	$2.21
Net cash	$125MM	$1.11
Preclinical pipeline	$100MM	$0.88
Network biology platform	$50MM	$0.44
Antibody engineering platform and biologics capabilities	$0MM	$0.00
Diagnostic efforts	$0MM	$0.00
Manufacturing capabilities	$0MM	$0.00
Total	**$1.02B**	**$9.07**

Source: Cowen and Company

Merrimack Pharmaceuticals Quarterly P&L Model

	H1:11A	H2:11A	2011A	Q1:12E	Q2:12E	Q3:12E	Q4:12E	2012E
MM-398 Revenue	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
MM-121 Royalties	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
U.S. MM-121 End-User Revenue								
Ex-U.S. MM-121 End-User Revenue								
MM-111 Revenue	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
MM-302 Revenue	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
MM-151 Revenue	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Research and Development Revenues	13.1	21.2	34.2	10.0	10.0	10.0	10.0	40.0
MM-121 Cost Reimbursement	10.1	15.0	25.1	8.0	8.0	8.0	8.0	32.0
MM-121 Upfront Amort.	2.5	2.5	5.0	1.3	1.3	1.3	1.3	5.0
MM-121 Milestones	0.4	2.2	2.6	0.6	0.6	0.6	0.6	2.3
Other	0.1	1.5	1.6	0.1	0.1	0.2	0.2	0.7
Total Revenue	**13.1**	**21.2**	**34.2**	**10.0**	**10.0**	**10.0**	**10.0**	**40.0**
COGS	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Gross margin								
R&D	49.2	51.4	100.6	27.0	28.5	29.0	29.5	114.0
R&D as a % of sales								
SG&A	7.9	6.5	14.5	3.5	3.5	3.6	3.6	14.2
SG&A as a % of sales								
Contingent Consideration	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Total Expenses	**57.1**	**58.0**	**115.1**	**30.5**	**32.0**	**32.6**	**33.1**	**128.2**
Operating Income/Loss	**(44.1)**	**(36.8)**	**(80.9)**	**(20.5)**	**(22.0)**	**(22.6)**	**(23.1)**	**(88.2)**
Interest Income	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0
Interest Expense	(0.0)	(0.0)	(0.0)	0.0	0.0	0.0	0.0	0.0
Other Income (Expense)	1.3	(0.2)	1.2	0.0	0.0	0.0	0.0	0.0
Attributable to non-controlling interest	(0.2)	(0.2)	(0.5)	(0.2)	(0.2)	(0.2)	(0.2)	(0.6)
Pre-tax Income/Loss	**(42.5)**	**(36.7)**	**(79.2)**	**(20.4)**	**(21.9)**	**(22.4)**	**(22.9)**	**(87.6)**
Tax rate (%)	*0%*	*0%*	*0%*	*0%*	*0%*	*0%*	*0%*	*0%*
Provision for (Benefit from) income taxes	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Net Income (Loss)	**(42.5)**	**(36.7)**	**(79.2)**	**(20.4)**	**(21.9)**	**(22.4)**	**(22.9)**	**(87.6)**
GAAP EPS, Basic	**($0.56)**	**($0.49)**	**($1.05)**	**($0.26)**	**($0.24)**	**($0.24)**	**($0.24)**	**($0.97)**
Basic Shares Outstanding	75.3	75.3	75.3	78.0	93.0	94.0	95.0	90.0
Diluted Shares Outstanding	75.3	75.3	75.3	78.0	113.0	114.0	115.0	105.0

Source: Cowen and Company

Merrimack Pharmaceuticals Annual P&L Model

	2011A	2012E	2013E	2014E	2015E	2016E
MM-398 Revenue	0.0	0.0	0.0	0.0	50.0	180.0
MM-121 Royalties	0.0	0.0	0.0	0.0	0.0	0.0
U.S. MM-121 End-User Revenue						
Ex-U.S. MM-121 End-User Revenue						
MM-111 Revenue	0.0	0.0	0.0	0.0	0.0	0.0
MM-302 Revenue	0.0	0.0	0.0	0.0	0.0	0.0
MM-151 Revenue	0.0	0.0	0.0	0.0	0.0	0.0
Research and Development Revenues	34.2	40.0	35.0	25.0	22.0	25.0
MM-121 Cost Reimbursement	25.1	32.0	25.0	12.0	5.0	3.0
MM-121 Upfront Amort.	5.0	5.0	5.0	5.0	5.0	5.0
MM-121 Milestones	2.6	2.3	4.5	7.5	11.5	16.5
Other	1.6	0.7	0.5	0.5	0.5	0.5
Total Revenue	**34.2**	**40.0**	**35.0**	**25.0**	**72.0**	**205.0**
Y/Y growth					*90%*	*90%*
COGS	0.0	0.0	0.0	0.0	5.0	18.0
Gross margin						*78%*
R&D	100.6	114.0	120.0	125.0	130.0	140.0
R&D as a % of sales						*39%*
SG&A	14.5	14.2	16.0	18.0	66.0	70.0
SG&A as a % of sales						*0%*
Contingent Consideration	0.0	0.0	0.0	0.0	0.0	0.0
Total Expenses	**115.1**	**128.2**	**136.0**	**143.0**	**201.0**	**228.0**
Operating Income/Loss	**(80.9)**	**(88.2)**	**(101.0)**	**(118.0)**	**(129.0)**	**(23.0)**
Interest Income	0.1	0.0	0.0	0.0	0.0	0.0
Interest Expense	(0.0)	0.0	0.0	0.0	0.0	0.0
Other Income (Expense)	1.2	0.0	0.0	0.0	0.0	0.0
Attributable to non-controlling interest	(0.5)	(0.6)	(0.5)	(0.5)	(0.5)	(0.5)
Pre-tax Income/Loss	**(79.2)**	**(87.6)**	**(100.5)**	**(117.5)**	**(128.5)**	**(22.5)**
Tax rate (%)	*0%*	*0%*	*0%*	*0%*	*0%*	*0%*
Provision for (Benefit from) income taxes	0.0	0.0	0.0	0.0	0.0	0.0
Net Income (Loss)	**(79.2)**	**(87.6)**	**(100.5)**	**(117.5)**	**(128.5)**	**(22.5)**
GAAP EPS, Basic	**($1.05)**	**($0.97)**	**($0.90)**	**($0.95)**	**($0.95)**	**($0.15)**
Basic Shares Outstanding	75.3	90.0	112.0	124.0	135.0	145.0
Diluted Shares Outstanding	75.3	105.0	132.0	144.0	155.0	165.0

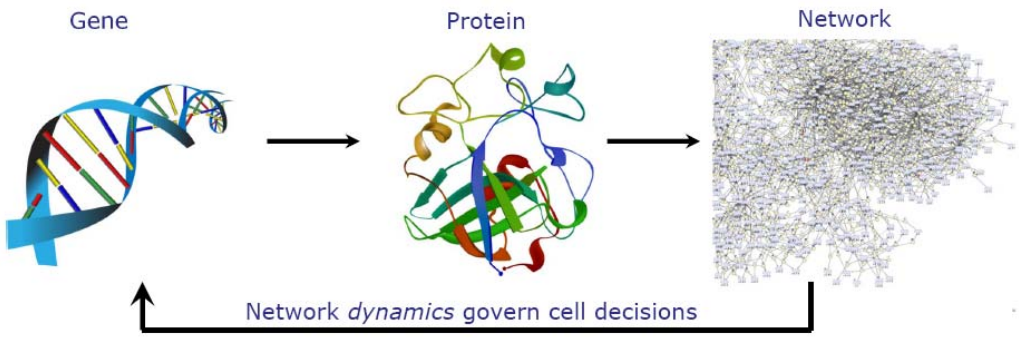
Source: Cowen and Company

Network Biology – Merrimack's Unique Approach

Merrimack is building a fully-integrated oncology company from the ground up. At the core of Merrimack's technology is its Network Biology approach to drug design. The company has been quite successful using this methodology, and now has five drug candidates in clinical trials. In addition, somewhat unusually for young biotech companies, Merrimack also has significant custom manufacturing capabilities in house, and is beginning to build a commercial organization in preparation for potential launch of its first commercial product, MM-398, now in Phase III.

Merrimack's unique approach to drug discovery and design is an interdisciplinary form of systems biology which the company terms Network Biology. Network Biology is Merrimack's solution to a sometimes over-reductionist approach to oncology drug development, where drug developers seek to identify and target single proteins that are critical to tumor growth. The Network Biology approach takes a more holistic view, recognizing that cellular behavior is governed by large, interdependent signaling pathways, such that the best intervention for treating a cancer may not be apparent without an understanding of the crucial nodes affecting the overall network. Merrimack is able to construct proprietary computer simulations of these cellular networks, which can be trained and refined with experimental data to simulate the global response of cells to drug perturbations over time. This approach has allowed Merrimack to derive a number of nonintuitive insights into methods of drug intervention in cancer, several of which have been translated into clinical candidates. Some of these include: (1) the key role of the ErbB3 receptor in mediating oncogenic signaling through both HER2 and EGFR, as well as chemotherapy resistance (used in developing MM-121 and MM-111); (2) the important implications of ligand binding affinity and signal amplification in EGFR-dependent cancers (used in developing MM-151); and (3) insights about transport dynamics of nanoparticles in the tumor microenvironment (used in developing MM-398 and MM-302). (For details on each of these insights, please see the relevant clinical candidate section below.) The depth of Merrimack's mechanistic understanding of each of its drug candidates has also allowed the company to develop companion diagnostics in each case to facilitate use.

Merrimack's Network Biology Approach



Network Biology incorporates the complexity of cellular signaling interactions

Source: Merrimack

Overview Of Clinical Development Candidates

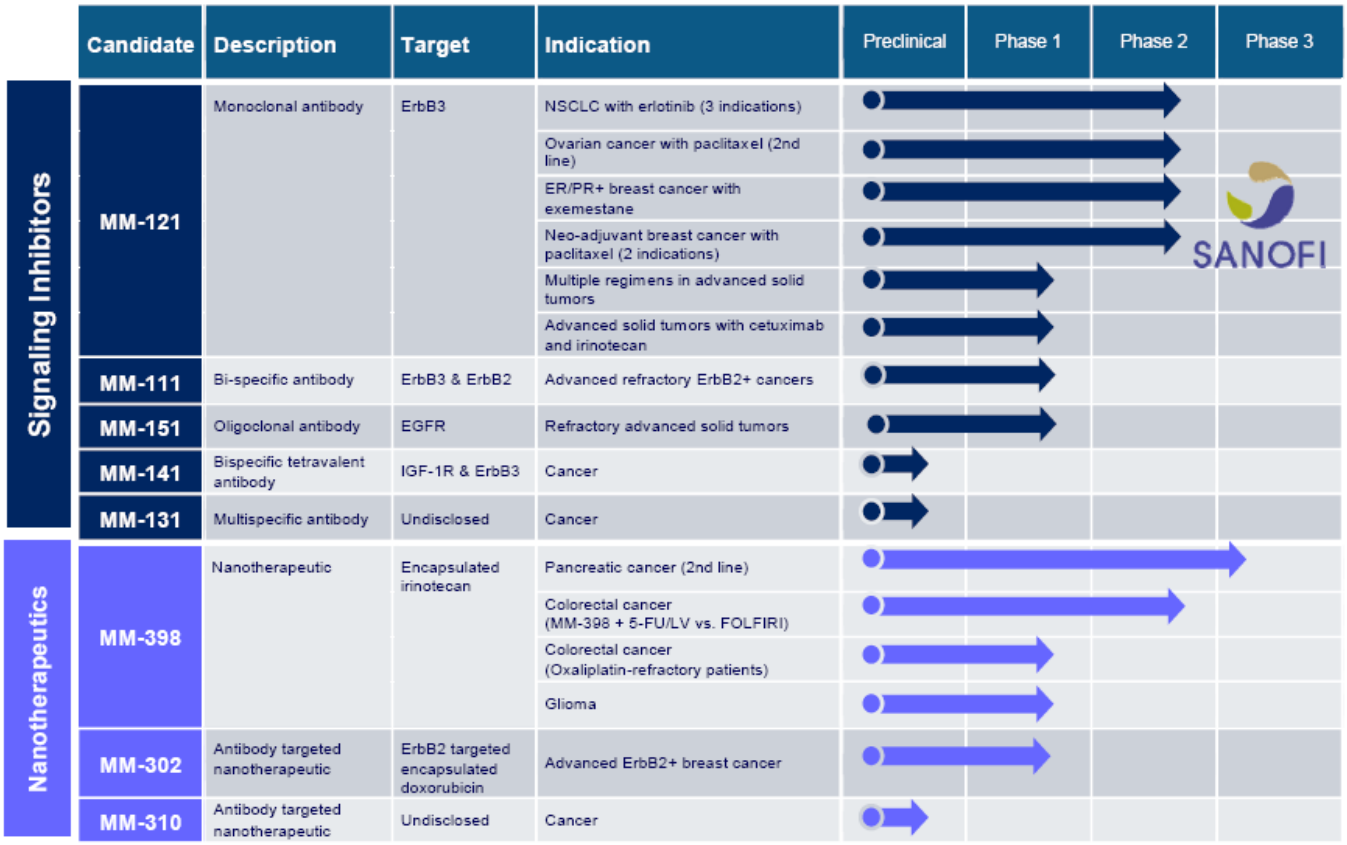
Merrimack currently has five clinical candidates, which include three antibodies and two nanoliposomal therapeutics.

The three antibodies are focused on disrupting ErbB-family growth factor signaling that drives cancer growth. MM-121 inhibits ErbB3, impairing its activity in the EGFR and HER2 signaling pathways, and is intended to enhance tumor sensitivity to current therapies. MM-111 is a bispecific antibody targeting both ErbB3 and HER2, intended to block heterodimerization and thus impair resistance to therapy in HER2(+) tumors. MM-151 is a mixture of three anti-EGFR antibodies that may offer superior efficacy to current EGFR-targeting agents.

The two nanoliposomal therapeutics are liposomal formulations of classic chemotherapies that Merrimack believes may offer superior efficacy and safety relative to the parent compounds. MM-398 is liposomal irinotecan, and is Merrimack's most clinically advanced candidate, currently in a Phase III trial for second-line pancreatic cancer. MM-302 is liposomal doxorubicin, and additionally is conjugated to an anti-HER2 antibody to enhance delivery into HER2(+) cancer cells.

Merrimack also has three disclosed candidates in preclinical development: two additional antibodies (MM-141 and MM-131), and one nanoliposomal therapeutic (MM-310).

Merrimack's Clinical Pipeline



Source: Merrimack

Overview Of Clinical Data

Merrimack's long and growing list of clinical trial results may be somewhat overwhelming to those new to the story. We therefore present here a summary tabulation of Merrimack's clinical data. These data will be described in further detail in the sections that follow.

Merrimack's Clinical Pipeline

Candidate	Phase	Cancer Setting	Background Therapy	N	ORR	DCR	PFS	OS
MM-121	I	late line breast & ovarian	paclitaxel	23	35%	74%		
MM-121	I	late line NSCLC	erlotinib	32	3%	47%		
MM-111	I	late line HER2+ cancer	paclitaxel, trastuzumab	10	40%	80%		
MM-111	I	late line HER2+ cancer	cisplatin, capecitabine, trastuzumab	9	33%	44%		
MM-111	I	late line HER2+ cancer	trastuzumab & lapatinib	13	8%	23%		
MM-398	II	pancreatic cancer	none	40	8%	48%	9.6 wk	22.4 wk
MM-398	I	oxaliplatin-refractory mCRC	none	18	22%	67%		
MM-398	II	gastric cancer	none	44	14%	61%	11.4 wk	33.6 wk
MM-398	I	advanced refractory tumors	none	10	20%	50%		

Source: Cowen and Company, Merrimack

Antibody Therapeutics

Merrimack's Network Biology models have highlighted the signaling pathways headed by the ErbB family of structurally related growth factor receptors as key drivers of tumor growth and drug resistance. Therefore, the company is developing several antibody therapeutics directed against members of the ErbB family, which include EGFR (HER1, ErbB1), HER2 (Neu, ErbB2), ErbB3 (HER3), and ErbB4 (HER4).

MM-121 Targets ErbB3

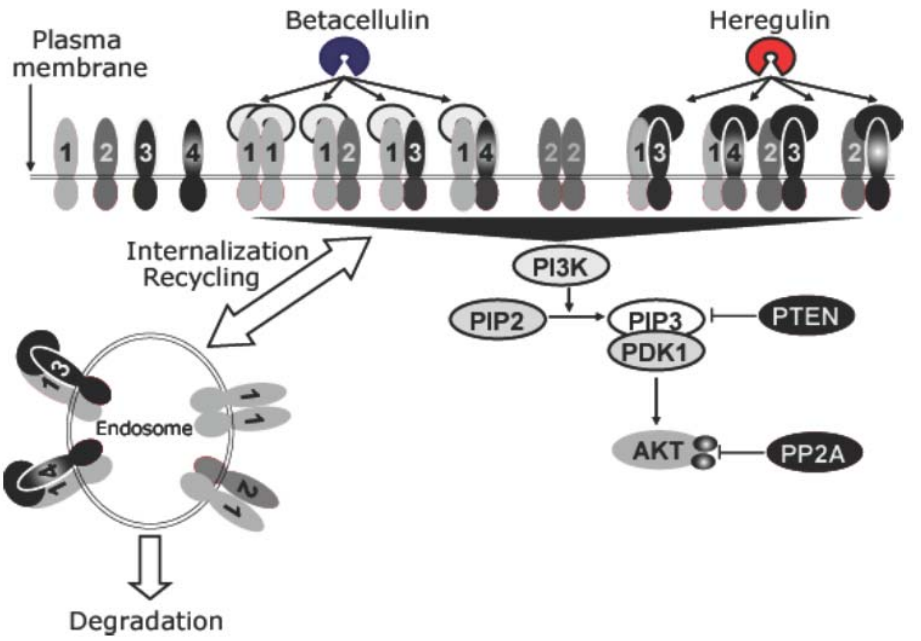
Merrimack's lead antibody candidate is MM-121 (a.k.a. SAR256212), which targets ErbB3. Merrimack's computational models and *in vitro* experiments permitted the identification of ErbB3 as a key lever in the signaling networks driving tumor growth (through both EGF and HER2-dependent pathways), as well as in chemotherapy resistance. MM-121 is being developed in a broad clinical program under a partnership with Sanofi, primarily focused on potential enhancement of the efficacy of existing drugs through synergistic tumor inhibition and/or thwarting resistance mechanisms.

The Role Of ErbB3 In Cancer

Merrimack's Network Biology approach led to identification of the key role of ErbB3 in cancers that are dependent on EGFR and HER2 signaling for growth. This discovery was somewhat non-intuitive and might not have readily emerged from traditional, reductive biological approaches. Traditional approaches commonly focus on a single oncogenic driver of cancer, exemplified by the oncogenic mutations frequently seen in EGFR tumors, or the HER2 amplifications, either of which drive elevated kinase activity and oncogenic addiction. ErbB3, in contrast, lacks kinase activity and is not overexpressed in cancer, so would not have been high on the target list for traditional drug development. Merrimack's approach, however, revealed that ErbB3 is a key player in combinatorial dimerization between the ErbB3 members (as receptor tyrosine kinases, the family members form homo-

or hetero-dimers during activation). Increasingly, published reports indicate that a common mechanism of resistance to a variety of anticancer drugs is ErbB3-dependent activation of HER2 signaling. Merrimack's models predict that interfering with ErbB3 function should not only impair oncogenic signaling from EGFR and HER2 receptors, but also disrupt cancer cells' ability to switch between the pathways as resistance mechanism when challenged with anti-cancer drugs.

Role Of ErbB3 In EGFR and HER2 Signaling Pathways

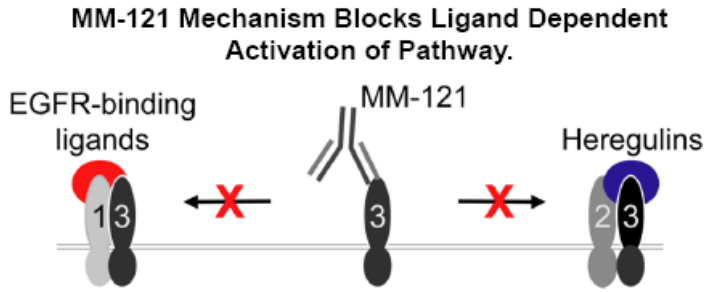


Source: Merrimack Pharmaceuticals

MM-121 Efficiently Inhibits ErbB3 *In Vitro*

MM-121 was the first human antibody targeting ErbB3 to enter the clinic (to our knowledge, Amgen's AMG 888 is the only other; Phase I data were reported at ASCO 2011). Experiments have shown that MM-121 is able to bind and inhibit ErbB3 with 1 nM efficiency. The only known ligand for ErbB3, heregulin, binds with low affinity to the receptor such that MM-121 is a powerful competitive inhibitor. In addition, MM-121 prevents ErbB3 heterodimerization with other family members, and also promotes internalization and degradation of ErbB3. Thus, MM-121 disrupts ErbB3's activity in both the HER2 and EGFR signaling pathways, and disrupts its activation either by direct heregulin binding, or indirectly by other ligands (through dimerization).

MM-121's Mechanism Of Action

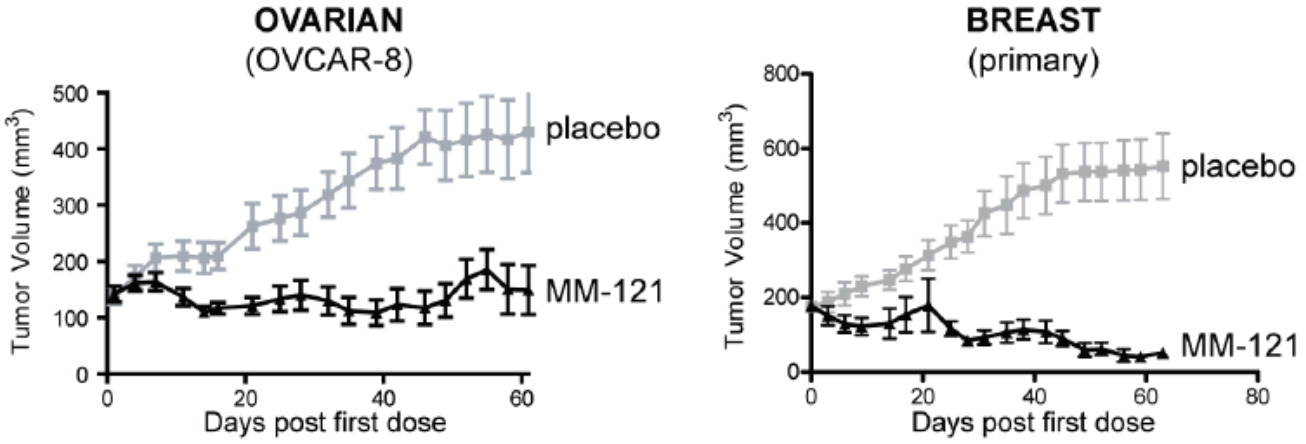


Source: Merrimack Pharmaceuticals

MM-121 Effective In Preclinical Cancer Models

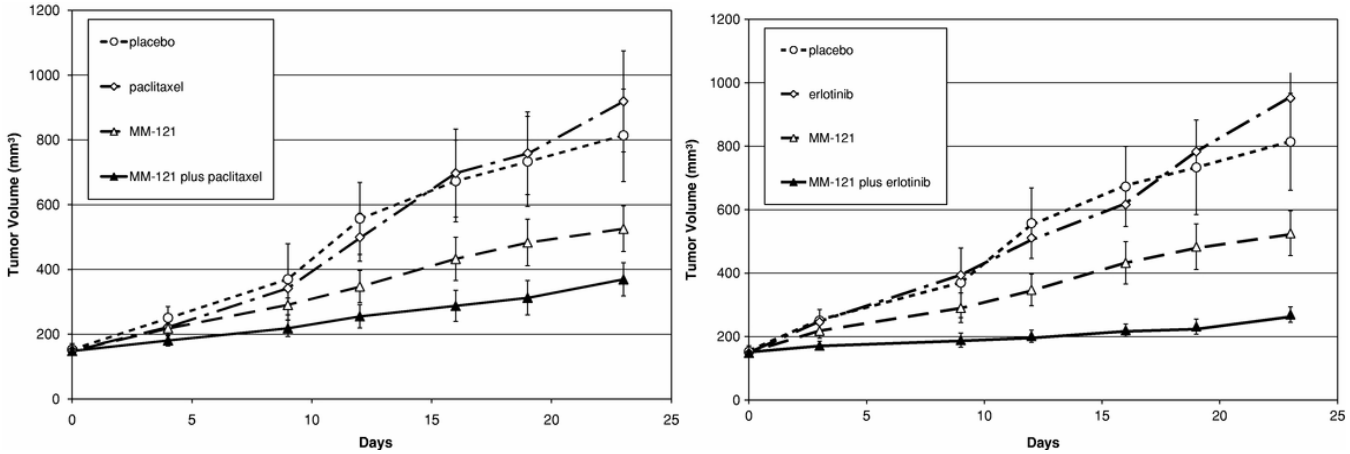
Merrimack has carried out extensive testing of MM-121 in preclinical murine tumor xenograft models. MM-121 has shown efficacy as a single agent in a variety of such models, such as lung, breast, ovarian, prostate and renal cancer, including in tumors that do not overexpress ErbB3. Moreover, Merrimack has found evidence that MM-121 may be capable of resensitizing resistant tumor lines to therapy when used in combination with other agents. For example, A549 human lung cancer cells are resistant to treatment with paclitaxel (Taxol) or erlotinib (Tarceva). In the xenograft model, paclitaxel or erlotinib treatment alone has no effect on the tumors. Treatment with MM-121 alone impaired tumor growth to some degree. However, combination treatment of the resistant tumors with either MM-121 plus paclitaxel, or M-121 plus erolitinib, profoundly suppressed tumor growth. That MM-121 can restore efficacy of several anti-cancer agents in different resistant lines supports Merrimack's hypothesis that ErbB3 mediates a common signaling module frequently used by cancer cells to drive treatment resistance in a variety of settings.

MM-121's Preclinical Single Agent Efficacy



Source: Merrimack Pharmaceuticals

MM-121's Preclinical Synergistic Efficacy In Resistant Tumors



Source: Merrimack Pharmaceuticals

Promising MM-121 Data In Three Phase I Trials

MM-121's initial single agent dose escalation trial was conducted primarily to identify a MTD and evaluate any objective response or PFS benefits. Initially, six cohorts of three to six patients with various advanced refractory solid tumors were enrolled (n = 25) and each cohort was given progressively higher weekly doses (3.2mg/kg – 20 mg/kg weekly, with the final cohort receiving 40 mg/kg in week 1 and 20 mg/kg weekly thereafter). The drug was very well tolerated and no MTD was identified. Of these 25 patients, 5 (20%) achieved SD as best response. Merrimack is now enrolling 20 to 30 additional patients in an expansion phase of this trial at what the company believes to be a therapeutic dose level, recruiting mainly HER2(-) breast cancer, ovarian cancer, and other tumor types where ErbB3 may be important. As of the last data update at AACR 2011, 13 additional patients had been enrolled, and four of these (29%) showed SD as best response. In all the 38 patients, the most common AEs were nausea, fatigue, and diarrhea. Grade 3 and 4 adverse events included fatigue (in four patients), nausea (in one patient), and vomiting (in one patient). In our view, the safety profile of MM-121 looks very benign, but as the evidence of single-agent efficacy is rather limited, most subsequent trials are focusing on combination approaches.

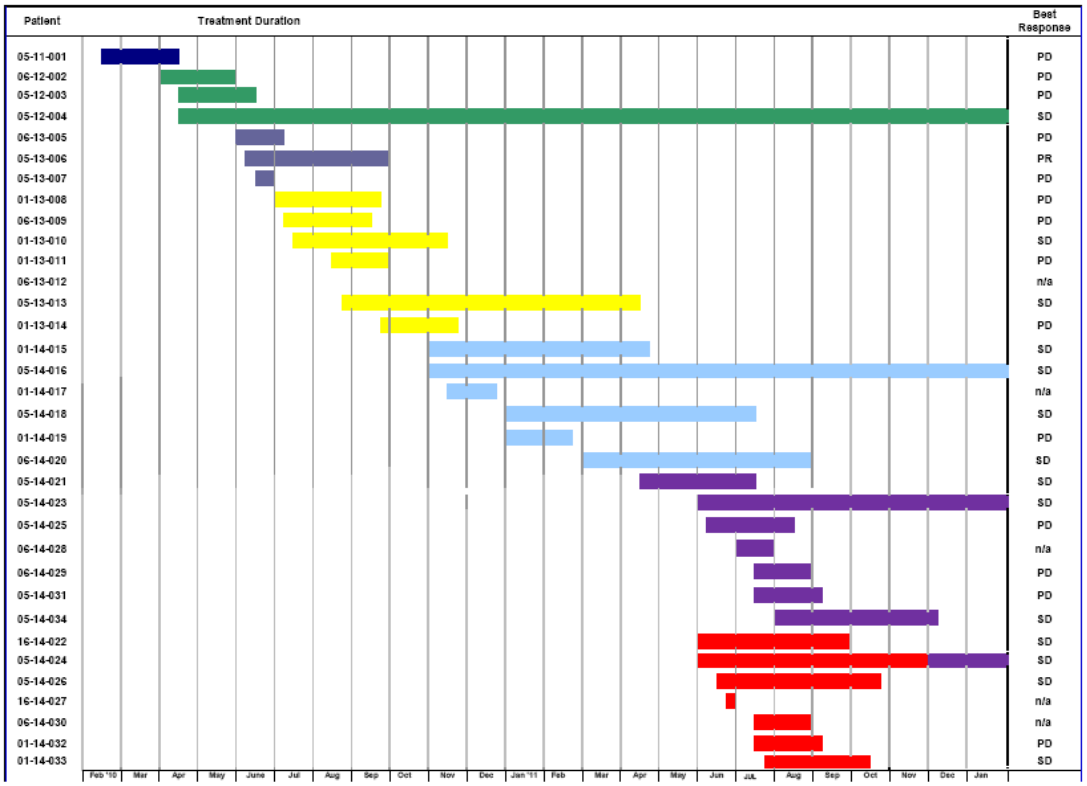
Promising data has also been reported from two additional Phase I combination trials.

The first Phase I combination trial tested M-121 plus erlotinib (Tarceva) in advanced NSCLC. Erlotinib targets oncogenic EGFR signaling, and literature evidence indicates that ErbB3 is frequently implicated in resistance to Tarceva, so the rationale for this combination appears sound. This ascending dose trial treated patients with daily eroltinib together with varying doses and schedules of MM-121 to evaluate safety and tolerability, as well as potential efficacy. Through July 2011, it was reported that 33 patients were enrolled (24 were erlotinib naïve and one had an EGFR mutation). Patients had had a median 2 prior lines of therapy (range 0-8). The combination was reasonably well tolerated, with the most common AEs being diarrhea (82%), fatigue (64%), and rash (64%). The most common Grade 3 AEs were fatigue (15%), and diarrhea (9%); there were no Grade 4 AEs. In a separate report as of January 2012, Merrimack indicated that the combination achieved a DCR rate of 47% and ORR of 3% (1 PR in 32 total patients). We calculate median PFS in these data at about 2.2

months. On the basis of these results, Merrimack and Sanofi have initiated a Phase II trial of the MM-121 + Tarceva combination in NSCLC (see next section, below).

A 700-patient Phase III study published in the 2005 *NEJM* compared Tarceva to placebo in advanced refractory NSCLC. These patients had received an average of 1.5 lines of prior chemotherapy (though response to Tarceva was not affected by number of prior lines of chemotherapy). For the Tarceva arm, the study reported ORR = 9%, DCR = 45%, PFS = 2.2 months, and OS = 6.7 months. We therefore believe it is difficult to say whether MM-121 is improving Tarceva's efficacy in this setting from the preliminary data available, and look forward to more robust data from the Phase II trial.

MM-121's Preliminary Phase I Efficacy Data (with Tarceva) In Advanced NSCLC



Source: Merrimack; Colors correspond to dose

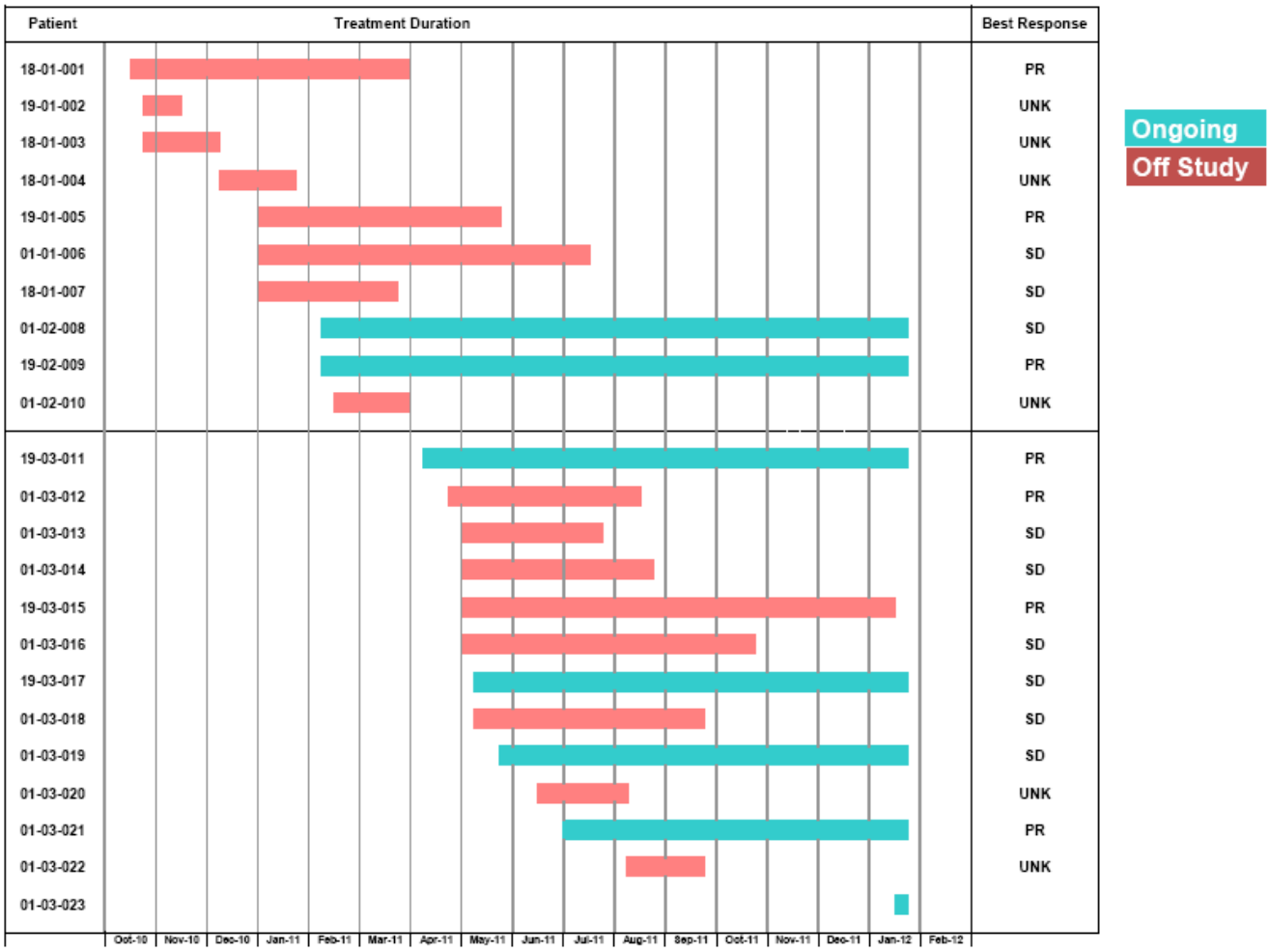
The second Phase I combination trial is testing MM-121 plus paclitaxel. This dose-escalation trial began in December 2010 and is enrolling patients with either (1) locally advanced/metastatic ovarian or other gynecological cancers or (2) locally advanced/metastatic, HER2(-) breast cancer. The dose escalation portion of the trial is complete, with an expansion cohort continuing to enroll. With 23 patients enrolled, Merrimack reported a DCR of 74% and ORR of 35% (all PRs), supporting continued development of MM-121 in combination with paclitaxel. Phase II trials with paclitaxel have begun in both breast and ovarian cancer (see next section, below).

The 700-patient, Phase III, E2100 study (published in the *NEJM* in 2007) compared paclitaxel alone to paclitaxel plus Avastin in metastatic breast cancer patients. 99% of these patients were HER2(-). The trial reported the following efficacy results for paclitaxel vs. paclitaxel plus Avastin, respectively: ORR = 21% vs. 37%, PFS = 11.8

months vs 5.9 months. Therefore, we believe the Phase I data, though preliminary, appear to suggest a substantial additional response benefit in HER2(-) breast cancer for MM-121 combined with paclitaxel over paclitaxel alone.

In ovarian/gynecologic cancer, data on expected response rate from single-agent paclitaxel appear more scant. A 50-patient Phase II JCO report from 2002 showed a 25% ORR in patients refractory to a prior paclitaxel plus platinum regimen. We believe more mature data are needed to assess MM-121's potential activity in this setting.

MM-121's Preliminary Phase I Efficacy Data (with Paclitaxel) In Ovarian And Breast Cancer



Source: Merrimack

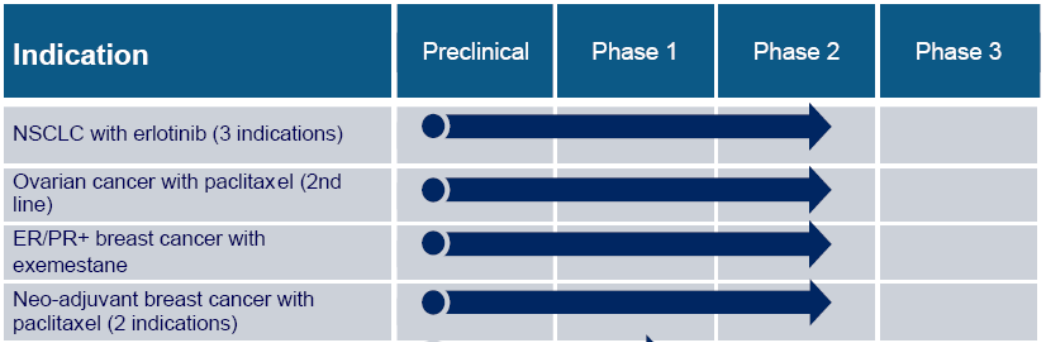
Merrimack has initiated two additional Phase I dose-finding combination trials, which remain in progress. The first trial began in November 2011 and is combining MM-121 with several standard anticancer therapies in advanced solid tumors, in order to determine safety and dosing in these combinations. The second trial began in December 2011 and is testing MM-121 plus cetuximab (Erbitux) and irinotecan in several types of EGFR-dependent cancers, including advanced colorectal cancer, squamous cell head and neck cancer, NSCLC, and triple-negative breast cancer. Literature evidence supports ErbB3-dependent resistance to EGFR inhibitor therapy, so the rationale makes sense in this trial.

Additionally, Sanofi and Merrimack are conducting a dose-finding Phase I combining MM-121 with SAR245408 (formerly XL147, a PI3K inhibitor Sanofi licensed from Exelexis Pharmaceuticals) in patients with locally advanced or metastatic solid tumors.

MM-121's Phase II Clinical Program Advancing On Multiple Fronts

MM-121's clinical development is currently advancing in four Phase II combination trials: with Tarceva in NSCLC, with exemestane in hormone-sensitive breast cancer, with paclitaxel in HER2(-) breast cancer, and with paclitaxel in platinum resistant or refractory ovarian cancer. In each case, tumor biopsies and serum biomarkers will be analyzed for markers that might predict likely responders and to develop Merrimack's companion diagnostic (Dx-121).

MM-121's Phase II Trials

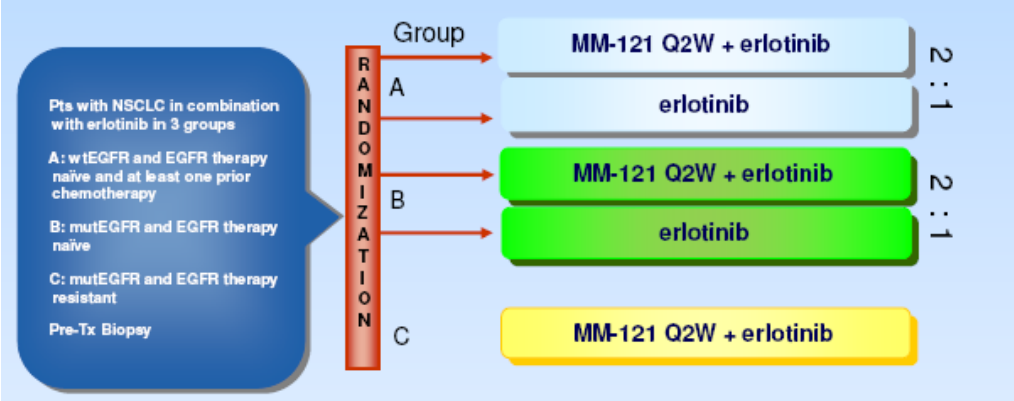


Source: AACR 2011 Poster

The Phase II trial with exemestane (Aromasin, Pfizer) for hormone-sensitive breast cancer began in July 2010 and is enrolling 130 women with metastatic breast cancer that is ER(+) and/or PR(+), but HER2(-), who have failed a estrogen deprivation therapy (other than exemestane). The hypothesis is that MM-121 may be able to disrupt (potentially ErbB3-dependent) tumor resistance to estrogen deprivation and improve efficacy vs. exemestane alone. Patients will be randomized 1:1 to receive either exemestane plus MM-121, or exemestane alone. The primary endpoint is PFS, and secondary endpoints include OS, ORR, and duration of response. Data are expected in H1:2013.

The Phase II NSCLC trial with Tarceva is a continuation of the Phase I portion described above and will increase total enrollment to 260 patients. The trial began in November 2011 and is enrolling three separate groups of patients: group A, wild-type EGFR and EGFR therapy naïve, with at least one prior chemotherapy regimen; group B, mutant EGFR and EGFR therapy naïve; and group C, mutant EGFR and EGFR therapy resistant. Groups A and B will be randomized to 2:1 to MM-121 q2w + Tarceva or Tarceva alone; group C will be uncontrolled. The primary endpoint is PFS and secondary endpoints include OS and ORR. This design should allow assessment of MM-121's potential both as an enhancer of erlotinib activity in naïve patients, as well as a means to resensitize resistant tumors. Data from groups A and C are expected in H1:2013, while data from group B are likely to come later.

MM-121's Phase II NSCLC Trial Design



Source: AACR 2011 Poster

The Phase II trial with paclitaxel (Taxol) in neoadjuvant HER2(-) breast cancer began in October 2011. The trial is enrolling 200 patients total in two separate groups: group A, treatment naïve ER(+) HER2(-); and group B, treatment naïve triple-negative. Each group of patients will be randomized 2:1 to receive paclitaxel plus MM-121 or paclitaxel alone for 12 weeks, then all patients will be treated with doxorubicin plus cyclophospamide (a standard regimen) until surgery. The goal of the study is to determine whether paclitaxel plus MM-121 is superior to paclitaxel alone on the primary endpoint of pathological complete response (pCR) at resection. Data are expected in H2:13.

The Phase II trial with paclitaxel in platinum resistant or refractory ovarian cancer is enrolling 210 patients and randomizing them to MM-121 plus paclitaxel or paclitaxel alone. The goal is to determine whether the combination is superior to paclitaxel alone on the primary endpoint of PFS. Secondary endpoints include OS and ORR. Data may be available in early 2015.

Companion Diagnostic For MM-121 Being Co-Developed

Using animal models, Merrimack has identified a suite of biomarkers that predict responsiveness to MM-121. In its Phase II trials, Merrimack is collecting biomarker data to test the effectiveness of its diagnostic responder prediction diagnostic, Dx-121. If successful, the hope is that Dx-121 could be used to inform patient inclusion in future clinical trials.

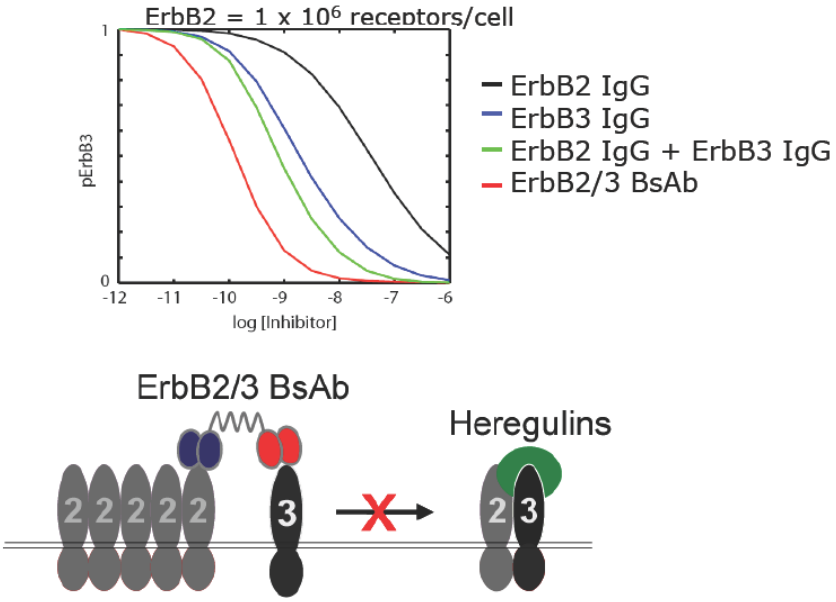
MM-121 Partnered With Sanofi

MM-121 is being developed under a partnership with Sanofi established in 2009. Sanofi has full global development and commercialization rights to MM-121 and Dx-121. Merrimack retains the right (though not obligation) to conduct mutually agreed-upon trials through Phase II proof of concept. Sanofi is responsible for all development and manufacturing costs. Merrimack received $60MM up front, and is eligible for a total of up to $410MM in development and regulatory milestones ($25MM received as of Q1:2012), plus an additional $60MM in sales milestones. Merrimack will received tiered royalties on net sales, beginning in the sub-teen range in the U.S. and high single-digit range ex-U.S., and also has a co-promote option in the U.S.

MM-111: Blocking Resistance In HER2(+) Cancer

MM-111 is a bispecific antibody targeting both HER2 and ErbB3, intended to treat HER2-positive cancer. The design grew from Merrimack's observation that, in HER2-amplified tumors treated with Herceptin (which blocks the binding of HER2 ligands, thus blocking direct HER2 activation), a common mechanism of escape is for increased heregulin expression to bind ErbB3, driving heterodimerization with HER2 and consequently, HER2 ligand-independent activation of the HER2 receptors. Because of the high affinity of heregulin for the ErbB3/HER2 complex, Merrimack's computational models predicted that single antibodies against either receptor, or a combination of two single antibodies, each targeting one of the receptors, would be relatively less effective at inhibiting heregulin's activation of HER2 signaling. The models predicted that heregulin binding and receptor heterodimerization could be more efficiently blocked by a bispecific antibody binding both HER2 and ErbB3 because it would increase local concentration of the antibody, particularly in the context of massive upregulation of HER2 on tumor cells.

Efficient Predicted Heterodimerization Blockade By ErbB2/B3 Bispecific Ab

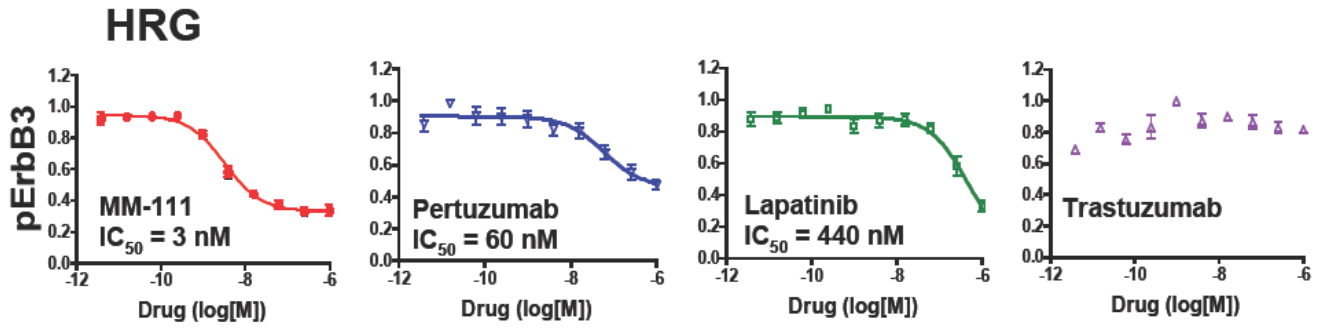


Source: Merrimack Pharmaceuticals

In vitro experiments with the highly HER2-overexpressing BT474 breast cancer line showed that addition of heregulin allows escape from Herceptin-mediated growth inhibition, presumably through heregulin-mediated ErbB3/HER2 heterodimerization. Merrimack's experiments show that even very high concentrations of Herceptin, well above the practical physiological limit of about 1 uM, are ineffective at blocking ErbB3 phosphorylation in the presence of heregulin. In contrast, MM-111 efficiently blocks ErbB3 phosphorylation in the presence of heregulin, with an IC50 of 3 nM. This inhibition of ErbB3 phosphorylation in the presence of heregulin is substantially better than than that of lapatinib (Tykerb, a HER2 kinase inhibitor; IC50 = 440 nM) and pertuzumab (a HER2 dimerization inhbitor; IC50 = 60 nM). Merrimack has also shown *in vitro* that BT474 growth inhibition by either Tykerb or Herceptin in the presence of heregulin can be improved by combination with MM-111. Merrimack hypothesizes that one of MM-

111's actions may be to suppress acquired ErbB3-dependent resistance to anti-HER2 therapy.

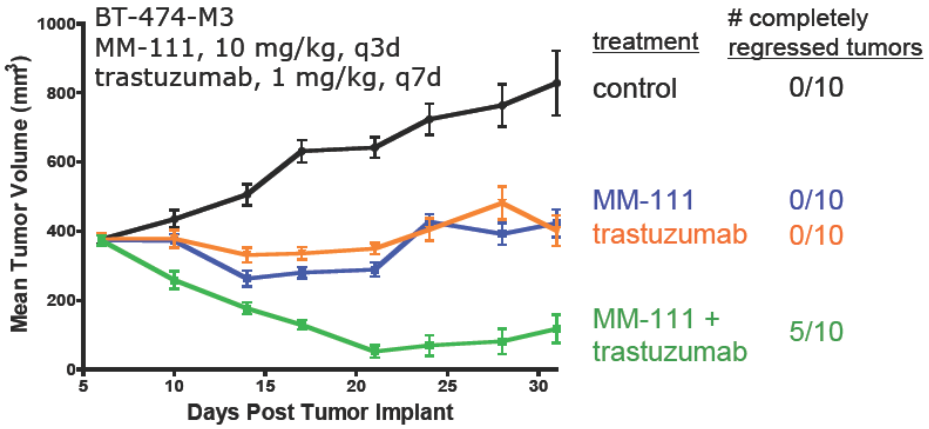
MM-111 Blocks Heregulin (HRG) Mediated Growth In A HER2(+) Breast Cancer Line



Source: Merrimack Pharmaceuticals

In vivo preclinical murine xenograft studies corroborate MM-111's efficacy against BT-474 breast cancer tumors and other HER2(+) tumors including lung and gastric cancer. In the BT-474 experiments, MM-111 alone produced growth inhibition akin to that produced by Herceptin alone. In combination, however, MM-111 potentiated Herceptin's efficacy, causing strikingly greater tumor inhibition and regression than either agent alone.

MM-111 Potentiates Herceptin's Efficacy In A Preclinical Xenograft Model



Source: Merrimack Pharmaceuticals

MM-111 Clinical Trials

Merrimack has advanced MM-111 into three Phase I clinical trials. Based on promising initial data, the company expects to commence randomized Phase II trials in HER2(+) breast cancer after the Phase I data mature.

The first Phase I trial was a standard dose-escalation, single agent trial that enrolled 20 patients with advanced, refractory HER2(+) solid tumors. The primary goal of the trial was to determine MTD, and secondary endpoints included ORR and PFS. The trial was completed in March 2012, though data remain under review.

The second Phase I trial is a dose-escalation study of the combination of MM-121 and Herceptin. The trial enrolled 15 patients with refractory, HER2(+) breast cancer. The primary goal is to assess safety of the combination, and the secondary endpoint is to determine recommended dose. The trial remains ongoing.

The final Phase I trial is a multi-arm combination (MAC) trial, enrolling patients with advanced HER2(+) tumors and testing MM-121 in combination with three different background regimens (in three separate groups): (1) paclitaxel and Herceptin; (2) cisplatin, capecitabine, and Herceptin; and (3) Tykerb and Herceptin. The primary goal of the study is to determine MTD and DLT of MM-121 in combination with each of the three background regimens, though efficacy measures are also being monitored, including ORR and DCR. The MAC trial is continuing to enroll patients toward its target 60 patients altogether. However, Merrimack has released interim efficacy data for the first patients enrolled in each group. In group (1), MM-121/paclitaxel/Herceptin, ORR was 40% and DCR 80%; in group (2), MM-121/cisplatin/capecitabine/Herceptin, ORR was 33% and DCR 44%; and in group (3), MM-121/Tykerb/Herceptin, ORR was 8% and DCR was 23%. (Interim responses for individual tumor types in each group can be seen in the table below). Merrimack plans to present full data from this trial in late 2012.

MM-111's Phase I Combination Interim Efficacy Data

Drug	Combination	Indication	N-value	ORR (CR+PR)	DCR
MM-111	• MAC-1: Paclitaxel, trastuzumab, MM-111	• Adv. HER2+ solid tumors	• 10	• 40%	• 80%
		• *Breast*	• *6*	• *33%*	• *83%*
		• *Bladder*	• *3*	• *33%*	• *66%*
		• *Esophageal*	• *1*	• *100%*	
	• MAC-2: Cisplatin, capecitabine, trastuzumab, MM-111	• Adv. HER2+ solid tumors	• 9	• 33%	• 44%
		• *Breast*	• *4*	• *25%*	• *25%*
		• *Bladder*	• *3*	• *33%*	• *66%*
		• *Colon*	• *1*	• *100%*	
		• *Esophageal*	• *1*	• *NA*	
	• MAC-3: Lapatinib, trastuzumab, MM-111	• Adv. HER2+ solid tumors	• 13	• 8%	• 23%
		• *Breast*	• *9*		• *22%*
		• *Ovarian*	• *2*	• *50%*	
		• *Esophageal*	• *2*	• *NA*	

Source: Merrimack Pharmaceuticals

For context, prior studies have shown objective response rates around 38% for first-line HER2(+) breast cancer treated with paclitaxel and trastuzumab. Regarding more heavily treated patients: in an extension study of Herceptin's pivotal trial (*JCO* 2004), 30 patients progressing on chemotherapy plus Herceptin achieved only a 7% ORR on subsequent treatment with paclitaxel plus Herceptin. Separately, a 66-patient Phase II trial of Herceptin plus pertuzumab (a dimerization inhibitor, like MM-111) in patents progressing on prior Herceptin reported a 24% ORR. Meanwhile, studies suggest a 10% ORR for refractory HER2(+) breast cancer treated with lapatinib and trastuzumab. Given the degree of pretreatment in the patients in MM-111's trial, we

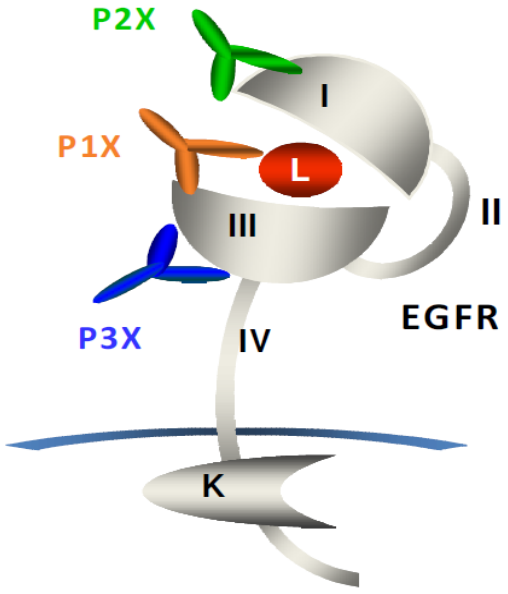
consider the demonstrated response rates encouraging, though a clear understanding of MM-111's efficacy benefit in particular combinations and tumor settings must await more mature data. Merrimack intends to begin a Phase II trial in HER2(+) breast cancer around mid:2012.

Merrimack is also developing a companion diagnostic, Dx-111, that is expected to help prospectively identify likely responders for MM-121.

MM-151: Mixing Up EGFR Signaling

MM-151 is a mixture of three antibodies, each targeting a non-overlapping EGFR epitope, that Merrimack believes will deliver more effective EGFR signaling inhibition than current agents. EGFR agonists include several high-affinity ligands (epidermal growth factor (EGF), betacellulin (BTC), heparin-binding epidermal growth factor (HB-EGF), and transforming growth factor alpha (TGFa)) and several low-affinity ligands (amphiregulin (AREG), epiregulin (EREG), and epigen (EPI)). The company believes that current EGFR inhibitors, Including Lilly's/Bristol's Erbitux and Amgen's Vectibix, fail to optimally inhibit EGFR because their single-epitope binding may not be sufficient to completely competitively block binding high affinity ligands to the receptor, especially in cases where tumors overexpress the ligands. Moreover, Merrimack believes that such upregulation of EGFR ligands may be a major mechanism of resistance to EGFR inhibitors. Merrimack's models indicate that, because of signal pathway amplification, even modest residual signaling can drive strong stimulation of the pathway's ultimate downstream targets. By binding to three different epitopes on EGFR, Merrimack's models indicate that MM-151 should provide superior inhibition of EGFR stimulation by both high- and low-affinity ligands. MM-151 also downregulates EGFR by promoting receptor internalization and degradation.

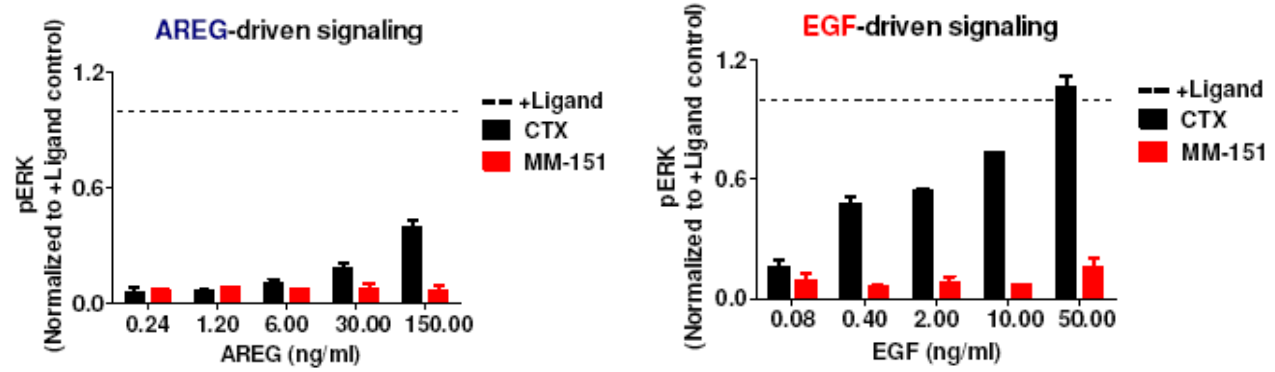
MM-151 – Mechanism of EGFR Binding



Source: Merrimack Pharmaceuticals

Preclinically, Merrimack's *in vitro* experiments have demonstrated that the three antibodies in MM-151 exhibit synergistic inhibition of EGFR signaling relative to any of the three alone. MM-151 also inhibits EGFR signaling more robustly than Erbitux or Vectibix *in vitro* – Erbitux only effectively blocks low-affinity ligand signaling, while MM-151 can block both high- and low-affinity ligand signaling. Furthermore, MM-151 exhibited activity in multiple xenograft models, and MM-151 has superior efficacy to Erbitux in xenografts of breast and lung cancer tumors with acquired Tarceva resistance. Preclinical AEs have been consistent with EGFR class effects (rash, diarrhea).

MM-151 (But Not Erbitux) Inhibits Both Low-Affinity (Left) and High-Affinity (Right) EGFR Signaling



Source: Merrimack Pharmaceuticals; CTX = cetuximab/Erbitux; AREG = amphiregulin (a low-affinity ligand)

Clinically, Merrimack hopes to (1) replace existing EGFR-targeted therapies in the treatment paradigm by showing superior efficacy for MM-151, and (2) expand EGFR inhibition into new tumor types. The company began a Phase I dose escalation trial of MM-151 in patients with advanced, refractory solid tumors in January 2012. Primary goals are to assess safety and determine the Phase II dose. Response rate (ORR) will also be recorded. The trial may be complete in late 2013.

Merrimack is also pursuing a companion diagnostic to prospectively identify potential responders, which is in preclinical development.

Nanoliposomal Chemotherapeutics

Merrimack is developing two nanoliposomal therapeutics in the clinic, MM-398 and MM-302. These are liposomal formulations of classic chemotherapies which may offer better safety and/or efficacy than the parent compound.

MM-398 Aims To Improve Irinotecan

MM-398 (formerly PEP02) is Merrimack's most advanced clinical candidate, in Phase III for gemcitabine-refractory metastatic pancreatic cancer. MM-398 was originally developed by Hermes Biosciences, which Merrimack acquired in 2009. Subsequently, in May 2011, Merrimack also purchased the European and Asian rights, which had been outlicensed to Taiwan's PharmaEngine, and now holds full global commercialization rights in all territories except Taiwan. Prior to that date, PharmaEngine had been leading the clinical development of MM-398.

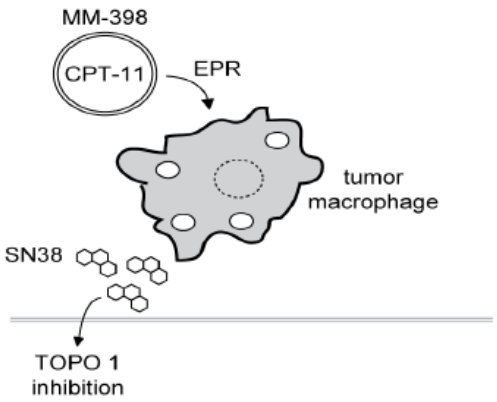
MM-398 is a "nanoparticle chemotherapeutic" comprised of the generic chemotherapy, irinotecan, encapsulated in a liposomal shell. Irinotecan (brand name

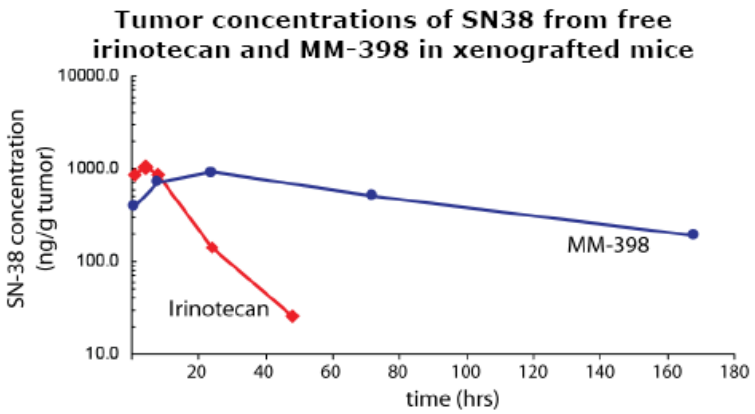
Camptosar, Pfizer) is used in many oncologic settings, including pancreatic, colorectal, ovarian, gastric, and lung cancer. Merrimack expects that MM-398 will improve on irinotecan's efficacy and safety profile because it will be concentrated and activated specifically in the vicinity of tumors, not in healthy tissues. There are two principle mechanisms by which this is expected to be achieved. The first of these is the so-called Enhanced Permeability and Retention (EPR) effect in tumors. The MM-398 nanoparticles have been designed to have a diameter of approximately 100 nm (larger than antibodies, at 10nm, or small-molecule chemotherapies, at 1 nm). This is important because the nanoparticles are thus small enough to leak out of the irregular, permeable vasculature formed in association with growing tumors, but the particles are large enough that their clearance is inefficient via the (typically compressed) draining lymph vessels. Thus, the nanoparticles will tend to accumulate in tumors, while smaller antibodies and chemotherapies are rapidly cleared back into circulation and do not accumulate efficiently. Merrimack's computational transport models allowed it to optimize nanoparticle size *in silico* to take advantage of these properties and optimize delivery.

A second mechanism of tumor selectivity is due to the prodrug nature of irinotecan. Normally, naked prodrug irinotecan (aka CPT-11) is metabolized to the active form (SN-38) in the liver and GI tract. The active form then travels systemically throughout the circulation, where it eventually reaches the tumor (though not being retained efficiently there, as just described), but also damages healthy tissue and causes side effects such as neutropenia and diarrhea. MM-398, in contrast, after exiting the circulation in the tumor vicinity is taken up by macrophages (enriched in the tumor environment) via phagocytosis and converted to active SN-38 locally to the targeted tumor cells. Merrimack believes this macrophage trafficking should also aid with delivery to hypoxic regions and stromal tissues.

The success of this approach has been borne out by *in vivo* preclinical results and initial human trials. In mouse model xenografts of human tumors, MM-398 has shown durable tumor responses that are superior to irinotecan. Merrimack indicates that approximately 10% of injected drug localizes to the tumor, as compared to 0.1% or less with naked irinotecan. Moreover, relative to irinotecan, in Phase I and II trials MM-398 PK results showed a more sustained release, longer circulation time, longer half life, lower Cmax, and negligible systemic exposure to free irinotecan.

MM-398 Produces Higher Concentrations Of Active Metabolite At Tumors Than Irinotecan Alone





Source: Merrimack

Early MM-398 Studies Show Signs Of Activity

Results from three Phase I studies on MM-398, conducted by PharmEngine in Taiwan, were reported at ASCO in 2008.

The first Phase I trial was conducted to assess the safety of a 3-week regimen of MM-398 monotherapy and identify the maximum tolerated dose. Eleven patients with advanced refractory solid tumors (including cervical, breast, pancreatic, NSCLC, neuroendocrine, thymoma) were recruited in an open label, single arm standard 3+3 dose escalation study. Dose was escalated through 60, 120, and 180 mg/m2 q3wk. At the highest dose, Grade 3 or 4 diarrhea and neutropenia was observed in two patients, so the MTD was determined to be 120 mg/m2 q3wk. PK studies indicated that irinotecan was released slowly from the nanoparticles over time, with dose a half-life of nearly 30 hours. Relative to published PK parameters after 125 mg/m2 of irinotecan, after a 120 mg/m2 dose of MM-398, SN-38 was present with a lower Cmax, longer half-life, and larger AUC. Of ten evaluable patients, there were 2 PRs (in cervical and pancreatic cancer) and 3 SD responses.

A second Phase I trial tested MM-398 in combination with 5-FU/leucovorin. The MTD was found to be 80 mg/m2 q3w, with DLTs at higher doses being Grade 3 diarrhea and Grade 4 hematological toxicity. Two PRs were seen (one each in gastric and breast cancer), and nine patients had SD.

A third Phase I trial sought to establish MTD of MM-398 monotherapy in oxaloplatin-resistant metastatic colorectal cancer. Irinotecan is already approved in colorectal cancer (in the FOLFIRI regimen) and frequently used second-line. MM-398 was dosed q2w in this trial, starting at 80 mg/m2 and escalating in 10 mg/m2 increments. Eighteen patients were enrolled, and the MTD was found to be 100 mg/m2 q2w, with Grade 3 diarrhea being the DLT. Of 17 patients evaluable for response, four (24%) showed PRs and eight (47%) maintained stable disease, a DCR of 71%. For perspective, a study of FOLFIRI in oxaloplatin failure mCRC published in *JCO* in 2004 showed an ORR of just 4% and at DCR of 34%.

MM-398 In Phase III For Refractory Pancreatic Cancer

MM-398 has produced promising Phase I and II data in gemcitabine-refractory pancreatic cancer, and is currently in a Phase III clinical trial with data expected in mid-2013. The goal of the study is to establish MM-398 as a second line therapy for metastatic pancreatic cancer, a disease which currently has a dismal prognosis and few successful therapeutic avenues. Merrimack is hopeful that MM-398 will mitigate systemic toxicity while delivering a more concentrated therapeutic payload to tumor cells. MM-398 has both U.S. and E.U. orphan drug designation in pancreatic cancer.

Pancreatic Cancer Overview

The pancreas is a common site of new cancers, and pancreatic cancer which is responsible for 6% of all cancer-related deaths, is a particularly dreadful disease. Difficult to diagnose in early stages because of the pancreas' anatomical location and the fact that it is asymptomatic until a relatively late stage, patients do not do well following diagnosis. At time of diagnosis, 52% of patients already have metastatic disease and 26% display regional spread. Consequently there is a low 1-year survival rate of 24% and a 5-year survival <5%.

The American Cancer Society (ACS) estimates there were approximately 43,140 new cases of pancreatic cancer responsible for 36,800 mortalities during 2010. Worldwide pancreatic cancer has remained a scourge as well with approximately 8-12 newly diagnosed cases/(100,000 patients-year). A disease of the middle aged, pancreatic cancer is infrequently seen in patients <45 years old but after 50 years, frequency increases and pancreatic cancer has a median age at diagnosis of 69 years.

Current treatment strategies for pancreatic cancer are minimal and include either surgical or chemotherapy. Locally occurring tumors are often treated surgically, which can be curative, but only 10-15% of patients actually have disease amenable to resection. Even following resection, quality of life is poor and cancer recurrence is high. Chemotherepeutic strategies are myriad, but have provided limited incremental increases in survival. Current first-line treatment is typically gemcitabine, sometimes in combination with Tarceva. A new option was recently unveiled when striking efficacy of an irinotecan-containing regimen (FOLFIRINOX - oxaliplatin, irinotecan, fluorouracil) was reported in 2011 from the ACCORD/PRODIGE trial. This trial demonstrated superior overall survival with FOLFIRINOX vs. gemcitabine (11 months vs. 6 months), though tolerability and side effects were poor. There is no consensus on treatment in second-line pancreatic cancer, though various chemotherapy regimens are used. Consultants indicate that gemcitabine-refractory metastatic pancreatic cancer patients can expect a median survival of two to three months.

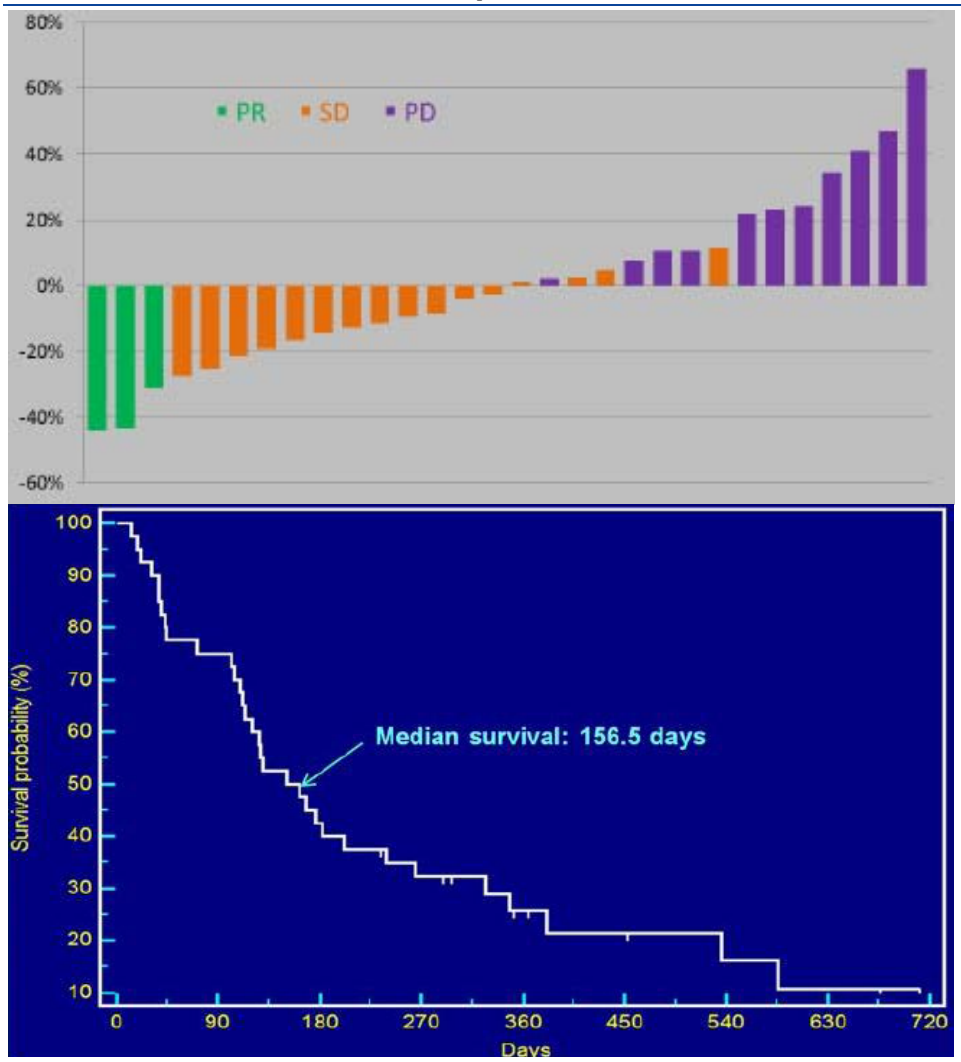
Signs Of MM-398 Activity In Early Pancreatic Studies

MM-398 has shown activity in a human pancreatic cancer orthotopic mouse xenograft model that were superior to an equivalent dose of irinotecan. In addition, seven chemotherapy-resistant advanced pancreatic cancer patients were treated with MM-398 as part of Phase I studies. Of those, one (treated with MM-398 monotherapy) had a PR, and four (treated with MM-398 plus 5-FU and leucovorin) had SD.

Promising Results In An MM-398 Phase II Pancreatic Trial…

MM-398 showed promising signs of efficacy in a single-arm Phase II trial, conducted by PharmaEngine at two sites in Taiwan and one at UC San Francisco. Forty patients with metastatic pancreatic cancer who had failed prior treatment with gemcitabine were enrolled. Most were treated with 120 mg/m^2 MM-398 every three weeks, but dose escalation and reduction were allowed. The primary endpoint was survival at three months, with the assumption being that 3-month survival would be 40% in these patients if untreated, and the bar for success in this trial being defined as 65% 3-month survival. The trial succeeded, with 75% of patients surviving 3 months or longer. Moreover, patients showed an ORR of 7.5% (comprised of three PRs), PFS was 9.6 weeks, and OS was 22.4 weeks. Importantly, 25% of patients survived for longer than one year. For perspective, gemcitabine was approved in first-line pancreatic cancer with a one-year survival of 18% (though it should be noted that this was in 1995). Thirty-two of the 40 patients (80%) had elevated CA19-9 at baseline, and of these, 34% showed a greater that 50% decline.

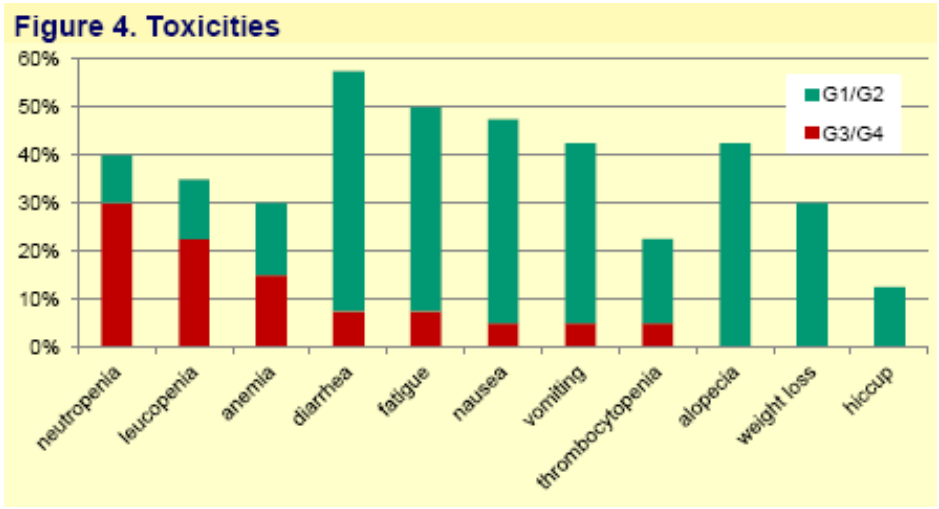
MM-398's Phase II Pancreatic Efficacy Data



Source: ASCO-GI 2011 Poster

The most common Grade 3 and 4 events included neutropenia, leukopenia, and anemia, with diarrhea, fatigue, nausea, and vomiting being the most common non-hemtologic Grade 3/4 events (see table below). Tolerability seems acceptable, in our opinion, given the seriousness of the proposed indication. Furthermore, physicians had the option to dose-reduce, and only 25% of patients had to have their dose reduced from the initial 120 mg/m^2 q3w, and nearly 30% of patients received MM-398 for six months or more.

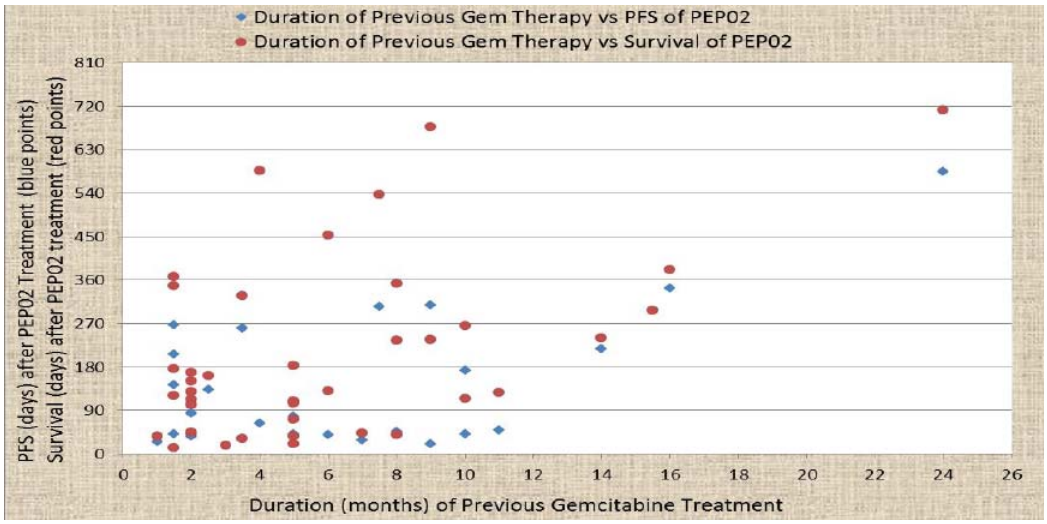
MM-398's Phase II Pancreatic Safety Data



Source: ASCO-GI 2011 Poster

An encouraging observation is that there seemed to be no clear correlation between survival on MM-398 and duration of treatment with prior gemcitabine (see figure below), consistent with the notion that longer survival may not be explained simply by patients' having had less aggressive baseline disease.

MM-398's Phase II Pancreatic Safety Data



Source: ASCO-GI 2011 Poster

...Support Initiation Of Pancreatic Phase III

In early 2012, Merrimack dosed the first patient in an open-label, international Phase III trial of 120 mg/m^2 q3w MM-398 monotherapy vs. a 5-FU plus leucovorin control regimen in gemcitabine-refractory second-line metastatic pancreatic cancer patients. The trial will enroll approximately 270 patients and has a primary endpoint of overall survival, with secondary endpoints of PFS, ORR, and time to treatment failure. The trial is 85% powered to detect a 6-week benefit in median OS, assuming a median OS of 3 months on 5-FU/leucovorin, which Merrimack believes is on the high end of what historical data predicts. Data are expected in mid:2013.

MM-398 Has Robust Activity, Decent Chance Of Success In Phase III

While it is always difficult to guess the outcome of Phase III trials based on uncontrolled Phase II data, we have compiled a tabulation of recent gemcitabine-refractory pancreatic cancer trials to put MM-398's Phase II data into context. The table shows that MM-398's 47.5% response rate is clearly superior to the ORR in any of the trials we identified, which have shown a median 11.5% response (range 0% - 29%), with a variety of regimens. MM-398's Phase II median OS of about 5.2 months is respectable, in about the same range as a variety of recent (generic) strategies, which have shown a median 5.9 months median OS (range 3.5 – 9.2 months). More importantly, MM-398's 5.2 month median OS is substantially better than any trial we have seen in this setting with the comparator agent in Merrimack's Phase III, 5-FU/lecovorin, which Merrimack expects will be about 3 months. Moreover, a clinical consultant (who has had hands-on experience with MM-398) believes the Phase II data are compelling enough to justify investment in a Phase III trial. Therefore we believe MM-398 has a decent chance of success in Phase III.

Recent Gemcitabine-Refractory Pancreatic Cancer Trials vs. MM-398

Study	Regimen	Number of Patients	Response Rate	Median PFS (months)	Overall Survival (months)
Pelzer et al (2009)	Oxaliplatin, 5-FU, leucovorin	37	6.0%	2.8	5.1
Androulakis et al (2005)	Oxaliplatin	18	5.0%	4	5.8
Demols et al (2006)	GEMOX	33	21.0%	4.2	6
Mazzer et al (2009)	Oxaliplatin, pemetrexed	16	19.0%	3.2	n/a
Reni et al (2006)	Oxaliplatin, raltitrexed	41	24.0%	n/a	5.2
Cantore et al (2004)	IROX	30	10.0%	4.1	5.9
Morizane et al (2007)	S-1	40	15.0%	2	4.5
Boeck et al (2009)	Capecitabine	39	0.0%	2.3	7.6
Blaya et al (2007)	Capecitabine, docetazel	24	12.5%	n/a	n/a
Togawa et al (2007)	Cisplatin, S-1	17	29.0%	n/a	9
Millela et al (2004)	5-FU, Celecoxib	17	12.0%	1.9	3.5
Chawla et al (2010)	Rexin-G	9	11.0%	7.65	9.2
Saif et al (2010)	Docetaxel	17	6.0%	2	4
Sudo et al (2011)	S-1	21	9.5%	4.1	6.3
	Median		**11.5%**	**3.2**	**5.9**
	Mean		**12.9%**	**3.5**	**5.9**
	MM-398	**40**	**47.5%**	**2.4**	**5.2**

Source: Cowen and Company

Second Line Pancreatic Cancer A Substantial Market Opportunity

Consultants say that there is no therapy for second-line pancreatic cancer that has been shown to improve survival. Hence, this indication represents a significant unmet need. Despite the lack of a proven treatment, our consultants estimate that 50% of chemotherapy patients in the U.S. go on to receive second- and sometimes third-line chemotherapy. Our consultants use the generic 5-FU, or Celgene's Abraxane off-label, in their gemcitabine failures (and gemcitabine in their FOLFIRINOX failures). Our model takes into account the fact that 20% of pancreatic cancer patients have resectable disease, and assumes that, of the remaining 80%, 60% will receive gemcitabine first line (the more robust patients instead starting on FOLFIRINOX). We assume that half of first-line gemcitabine patients will go on to second-line chemotherapy and be eligible for MM-398. Assuming modest growth in the incidence of pancreatic cancer due to an aging population, we estimate MM-398's addressable market will be just over 11,000 U.S. patients in 2015. Assuming an initial price of $80,000 per patient, we model penetration of nearly half the eligible population by 2019, generating $500MM in revenue.

MM-398 Revenue Model In Gemcitabine-Failure Pancreatic Cancer

	2015	2016	2017	2018	2019
Newly diagnosed cases (000s)	47.5	48.4	49.3	50.2	51.1
% eligible for chemotherapy	*80%*	*80%*	*80%*	*80%*	*80%*
Newly diagnosed metastatic or regionally advanced (000)	38.0	38.7	39.4	40.2	40.9
% receiving gemcitabine first-line	*60%*	*60%*	*60%*	*60%*	*60%*
Number of first line patients receiving gemcitabine (000)	22.8	23.2	23.7	24.1	24.5
% patients receiving second line therapy	*50%*	*50%*	*50%*	*50%*	*50%*
Number of second-line, gemcitabine failure patients (000)	11.4	11.6	11.8	12.0	12.3
% MM-398 penetration in the second line	*5%*	*19%*	*34%*	*39%*	*44%*
# Newly diagnosed patients treated (000s)	0.6	2.2	4.0	4.7	5.3
Average price per patient	$80,000	$83,200	$86,528	$89,989	$93,589
MM-398 Revenue ($MM)	**$50.0**	**$180.0**	**$350.0**	**$425.0**	**$500.0**

Source: Cowen and Company

MM-398 Also In Phase II For Second-Line mCRC

Following the encouraging Phase I monotherapy trial data in mCRC (described above), MM-398 is being evaluated in a controlled Phase II second-line mCRC trial, which began in mid-2011. The trial is being conducted by GERCOR, a French cooperative group, at six sites in France. This trial is enrolling 88 patients who have received prior oxaliplatin-based therapy, but not prior irinotecan. The patients are being treated with FOLFIRI in the control arm, or 5-FU + leucovorin + MM-398 in the experimental arm (the "FUPEP" regimen, which simply replaces the irinotecan component of FOLFIRI with MM-398). The primary endpoint is ORR and secondary endpoints include safety, PFS, OS, QoL, and correlation of UGTA1 polymorphism (an irinotecan metabolism gene) to toxicity of MM-398 or irinotecan. This trial includes an interim efficacy analysis, though details are undisclosed. Data are expected in mid:2013.

MM-398 Was Also Active In A Phase II Gastric Cancer Trial

MM-398 was additionally tested in a Phase II gastric cancer trial, with results reported at ASCO GI in 2011. However, Merrimack is not pursuing development in gastric cancer at this time.

The gastric cancer trial was done in patients with locally advanced or metastatic gastric or gastroesophageal junction adenocarcinoma who had failed prior chemotherapy. Patients also could not have had prior exposure to irinotecan. The trial was a randomized, three-arm study (MM-398 vs. docetaxel vs. irinotecan) enrolling 132 patients and conducted in Europe and Asia. The primary endpoint was objective tumor response. Though the parallel group design was not powered for comparisons across arms, ORR was numerically greater on MM-398 vs. irinotecan (14% vs. 7%), though similar vs. docetaxel (16%). However, DCR, PFS, and OS did not appear strikingly different between the arms.

PK results for SN-38 in this trial showed a higher AUC with MM-398 vs. irinotecan (3.3x higher, $p < 0.0001$), as well as a longer half-life (3.9x, $p = 0.0042$) and a lower Cmax (0.5x, $p = 0.0007$).

MM-398's Phase II Gastric Cancer Efficacy Data

Response	MM-398 (n=44)	Irinotecan (n=44)	Docetaxel (n=44)
ORR (CR + PR)	13.6% (6/44)	6.8% (3/44)	15.9% (7/44)
DCR (CR+PR+SD)	61.4% (27/44)	61.4% (27/44)	54.5% (24/44)
Median PFS (days)	81	79.5	82
Median OS (days)	218	235	219

Source: Merrimack Pharmaceuticals

The safety/tolerability profile of MM-398 was seemingly improved in some respects relative to irinotecan in this trial, though worsened in others. Hematologic toxicities, including neutropenia, anemia, and thrombocytopenia, were numerically lower with MM-398 vs irinotecan, but GI AEs such as diarrhea were numerically greater with MM-398. Safety of both looked better than docetaxel, with neurotoxicity and hand-foot syndrome in particular being worse with docetaxel.

MM-398's Phase II Gastric Cancer Efficacy Data

Grade 3/4 Hematological Toxicity

AE	MM-398 n = 44	Irinotecan n = 44	Docetaxel n = 44
Neutropenia (%)	5 (11.4)	7 (15.9)	7 (15.9)
Febrile Neutropenia (%)	3 (6.8)	5 (11.3)	2 (2.6)
Anemia (%)	2 (4.5)	2 (4.5)	3 (6.8)
Thrombocytopenia (%)	1 (2.3)	1 (2.3)	0 (0.0)

Grade 3/4 Non-hematological Toxicity

AE	MM-398 (n = 44)	Irinotecan (n = 44)	Docetaxel (n = 44)
Diarrhea (%)*	12 (27.3)	8 (18.2)	1 (2.3)
Nausea (%)	5 (11.4)	2 (4.6)	0 (0.0)
Vomiting (%)	2 (4.6)	6 (13.6)	3 (6.8)
Anorexia (%)	3 (6.8)	3 (6.8)	0 (0.0)
Fatigue (%)	2 (4.6)	1 (2.3)	1 (2.3)

* No statistical difference between MM-398 and irinotecan

Other Grade 1/2 Non-hematological Toxicity

AE	MM-398 (n = 44)	Irinotecan (n = 44)	Docetaxel (n = 44)
Stomatitis (%)	4 (9.1)	5 (11.4)	9 (20.5)
Peripheral sensory neuropathy (%)	0 (0.0)	0 (0.0)	7 (15.9)
Nail change/hand-foot syndrome (%)	0 (0.0)	3 (6.8)	8 (18.2)
Alopecia (%)	17 (38.6)	20 (45.5)	23 (52.3)

Source: ASCO GI 2011 Poster

MM-398 Trial In Glioma Also In Progress

An investigator-sponsored dose-finding Phase I trial in recurrent high-grade glioma is ongoing at UCSF. About 36 patients are being stratified based on UGTA1 genotype.

MM-398's Companion Diagnostic In Development

Merrimack is also developing a companion diagnostic, MM-Dx-929, that may be helpful in excluding patients who are unlikely to respond to MM-398. The diagnostic consists of a nanoliposomal formulation of copper, which can be directly visualized through a PET scan to determine whether a tumor accumulates active agent. MM-Dx-929 is in preclinical testing.

Global MM-398 Rights Licensed From PharmaEngine

Merrimack initially gained MM-398 through its 2009 acquisition of Hermes Biosciences, which initially developed the drug candidate. Hermes had previously outlicensed European and Asian rights to Taiwan's PharmaEngine, which had been leading clinical development of the drug under the name PEP02. In May 2011, Merrimack reacquired MM-398's European and Asian rights, and now owns global commercialization rights, except in Taiwan. In exchange, Merrimack paid PharmaEngine a $10MM upfront fee. Merrimack paid an additional $5MM milestone in Q1:12 as a result of dosing the first patient in MM-398's Phase III trial. Merrimack may also be required to pay up to $75MM in additional development and regulatory milestones and $130MM in sales milestones, as well as tiered high single digit to low teens royalties on net sales in Europe and parts of Asia. Merrimack is responsible for all development costs of MM-398 except those specifically required for licensure in Taiwan.

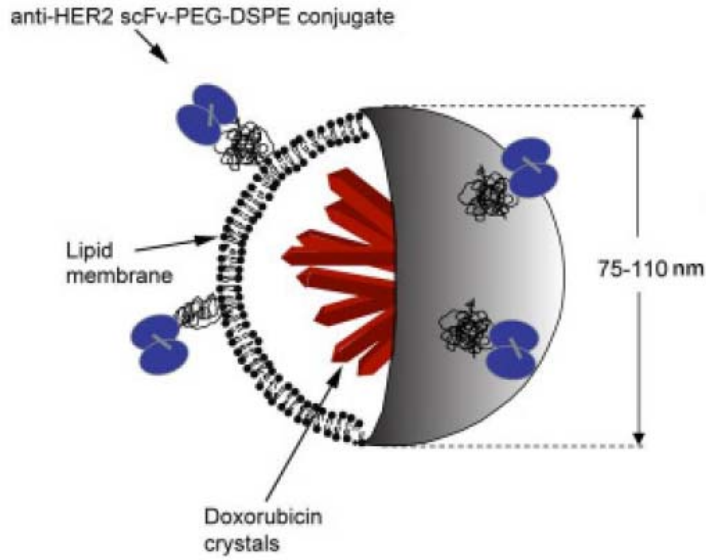
MM-398's Composition Patent Allowed

MM-398 received a notice of allowance from the USPTO for its composition patent in February 2012. Once issued, Merrimack expects the patent to last through at least 2027, inclusive of patent term extension.

MM-302 Is Liposomal, HER2-Targeted Doxorubicin

Merrimack's second candidate in the nanoliposomal therapeutic category is MM-302, which is a liposomal formulation of doxorubicin linked to a HER2 targeting antibody. The goal is to improve on the safety and efficacy profile of anthracyclines, thus gaining the opportunity to replace anthracyclines in current regimens and expand into indications where this class is believed to be effective but not used due to safety issues.

MM-302's Structure

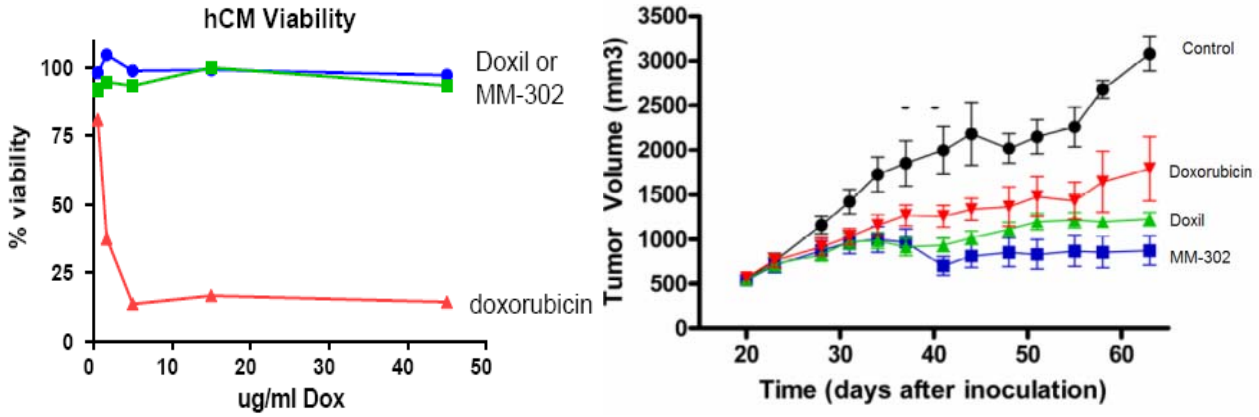


Source: Merrimack Pharmaceuticals

MM-302's liposomal formulation and HER2-targeting antibody could support improvements on doxorubicin's safety and efficacy profile. One of doxorubicin's most serious drawbacks is cardiotoxicity related to cumulative lifetime anthracycline dose, for which it has a black box. An existing liposomal formulation of doxorubicin, LLY's Doxil, has been shown in clinical trials to reduce cardiotoxicity, perhaps by limiting penetration into cardiomyocytes (though Doxil still has the black box). Merrimack expects to at least retain Doxil's cardiac safety advantage vs. doxorubicin as a consequence of MM-302's liposomal formulation. As with MM-398, MM-302's liposomal nature is also expected to lead to accumulation specifically in the tumor region via the EPR effect, thus increasing tumor exposure and reducing systemic exposure relative to doxorubicin; this effect is also observed with Doxil, though Merrimack believes its formulation further optimizes the phenomenon. However, Doxil has not been convincingly shown to be more effective than doxorubicin. Merrimack's experiments suggest this could be because Doxil's liposomal formulation, in addition to limiting penetration of heart cells, also limits uptake into tumor cells. Merrimack believes the addition of a HER2-docking antibody will promote uptake of MM-302 and improve efficacy without sacrificing safety. Moreover, the HER2 binding element does not impair HER2 signaling, suggesting the potential for side effects from this source is low (an important consideration, since HER2 plays a role in cardiac repair).

Merrimack's *in vitro* experiments indicate that MM-302's design reduces killing of cadiomyocytes vs. doxorubicin, and improves delivery to human HER2+ tumor cells vs. Doxil. In preclinical *in vivo* experiments, MM-302 showed low cardiac accumulation vs. doxorubicin, similar to Doxil, and MM-302 also showed superior activity to Doxil against tumor xenografts. Merrimack's initial data and modeling suggest that HER2 expression is too low on human heart cells to facilitate significant uptake.

MM-302's Effect On Cardiomyocyte Viability (Left) And HER2 3+ Mouse Xenografts (Right)



Source: Merrimack Pharmaceuticals, AACR 2011 Poster, Cowen and Company

In July 2011, Merrimack began a Phase I, dose escalation single-agent trial of MM-302 in advanced HER2+ breast cancer patients (an indication in which doxorubicin is routinely used currently). The trial will enroll 18 to 36 patients to find the MTD, at which point an expansion cohort will be enrolled. ORR is a secondary endpoint. Data may be available in H1:13. A companion diagnostic is also in preclinical development.

MM-302 Has Already-Issued IP

MM-302 has several issued U.S. and E.U. composition patents, expiring between 2014 and 2019. The company is pursuing an application for dosage and administration patent protection that may extend protection to 2031. All IP related to MM-302 has been licensed from the Regents of the University of California.

Preclinical Product Candidates

Merrimack's most advanced preclinical candidates are MM-141, MM-131, and MM-310. MM-141 is a bispecific antibody against IGF-1R and ErbB3, designed to inhibit insulin-like growth factor 1 signaling. An IND is planned in 2012. MM-131 is an undisclosed multispecific antibody. MM-141 and MM-131 are the first candidates in Merrimack's pipeline to target two growth signaling pathways simultaneously, which the company believes may improve response in tumors that co-utilize two pathways as growth drivers. MM-310 is an undisclosed targeted nanotherapeutic, with an IND planned in 2013.

Addendum

STOCKS MENTIONED IN IMPORTANT DISCLOSURES

Ticker	Company Name
MACK	Merrimack Pharmaceuticals

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Neutral (2)	Stock expected to perform in line with the S&P 500
Underperform (3)	Stock expected to underperform the S&P 500

(a) Assumptions: Time horizon is 12 months; S&P 500 is flat over forecast period.

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