

Marina Biotech, Inc. (MRNA)

DOWNGRADE REPORT

December 8, 2011

Rating Target

New: Avoid/Sell New: \$0.10

Old: Neutral Old: \$0.30

Analysts

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- ◆ Downgrading to Avoid/Sell Due to Program Delays
- ◆ Recent Financings Continue to Weigh Heavily on Stock
- ◆ Expected Reverse-Split Could Add to Pressure

1.) **Development for FAP and Bladder Cancer Slower Than Anticipated:** On June 9, 2011 Marina announced the dosing completion of the first 3-patient cohort in its START-FAP (“Safety and Tolerability of An RNAi Therapeutic in Familial Adenomatous Polyposis”) Phase I trial for CEQ508. The second cohort, which will receive 10x the initial dose, was expected to commence dosing in Q3 2011. Marina now expects dosing for the second cohort to start in early-2012. Additionally, on November 29, 2011 Marina announced that their joint R&D team with Debiopharm had advanced a lead DiLA2 formulation and multiple UsiRNA candidates for their bladder cancer program. While this is a positive development for the program, Marina stated that the R&D team expects to select the lead candidate in early-2012, which is significantly later than anticipated. These delays in the development timelines have resulted in reductions to our financial model.

2.) **Financings Continue to Weigh on Stock:** Marina Biotech’s financing in May brought much needed cash to the company but at the cost of very heavy dilution. In addition to the issuance of the 22.3M shares in the base unit and the 22.3M shares available through the Series B unit warrants, there could be up to an additional 44.6M shares that could potentially be issued from Series A warrants after 1 year at \$0.39. As of June 30, 2011, 7,121,500 of the Series B Warrants had been exercised, and in July 2011, an additional 15,172,000 of the Series B Warrants were exercised prior to their July 12, 2011 expiration date. (Continued next pg.)

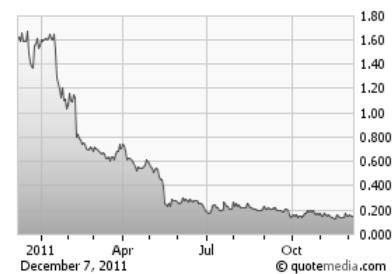


Symbol: MRNA
Exchange: Nasdaq

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CEO – J. Michael French
Interim CFO – Philip C. Ranker

Market Data		Share Data		Most Recent Quarter	
Price	\$0.14	Outstanding	91.4M	Revenue	\$0.3M
52-Week	\$0.11-\$1.92	Cash/Share	\$0.01	Net Income	(\$4.4M)
Market Cap	\$12.8M	Book/Share	\$0.17	EPS	(\$0.05)
Avg. Daily Vol.	4,529,470	Price/Book	0.8x	Cash	\$1.0M
% Short	11.2%	Debt/Share	\$0.00	Debt	\$0.0M
Financial Results and Projections					
FYE Dec. 31	2010	2011E	2012E	2013E	2014E
Revenue	\$2.5M	\$0.7M	\$0.4M	\$0.4M	\$2.0M
Net Income	(\$27.8M)	(\$18.6M)	(\$26.6M)	(\$29.0M)	(\$26.9M)
EPS	(\$1.58)	(\$0.27)	(\$0.14)	(\$0.13)	(\$0.10)



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2.) **Financings Continue to Weigh on Stock (Continued):** This resulted in 22,293,500 additional shares of dilution and 22,293,500 of additional Series A warrants exercisable at \$0.39 after one year. In addition, Marina announced on October 17, 2011 that they had entered into a purchase agreement with an institutional investor, whereby the investor committed to invest at Marina’s option, for up to 30 months, up to \$15 million of equity capital. Again, this brings much needed capital to Marina, but adds to the dilution from the previous financing.

3.) **Reverse-Split Nearly Certain:** On November 30, 2011 Marina announced that NASDAQ had granted the company until January 31, 2012 to establish a closing bid price of its common stock of \$1.00 or more per share for a minimum of 10 consecutive business days. Given the stock’s recent performance, future dilution concerns from both the warrant overhang and the equity purchase agreement and lack of catalysts until “early 2012”, we believe it is highly unlikely that Marina will regain the minimum bid requirement without executing a reverse-split, which could put additional pressure on the shares.

4.) **Downgrading to Avoid/Sell with \$0.10 Price Target:** While we continue to believe in Marina’s RNAi science and pipeline opportunities, delays in development timelines combined with additional dilution from recent financings and the risks of a reverse-split has negatively impacted our financial models. Therefore, we are downgrading our recommendation to Avoid/Sell (from Neutral) and reducing our target to \$0.10 (from \$0.30) based on a 35x multiple on projected 2015 earnings adjusted for the additional shares with a risk discount of 55%.

Company Description



Bothell, Washington-based Marina Biotech focused on development of pharmaceuticals based on small interfering RNA (siRNA), as well as siRNA delivery, designed to elicit specific therapeutic effects on a target-by-target basis. This includes pre-clinical manufacturing of siRNA and short hairpin RNA (shRNA) and delivery materials, and the ability to optimize compounds.

Specifically, Marina Biotech is currently employing two RNAi platforms to down-regulate the expression of specific proteins that cause disease. The first is TauRNAi, a combination of proprietary UsiRNA (siRNA modified with their Unlocked Nucleobase Analogs (UNA) chemistry) technology and their novel dialkylated amino acid-based liposome (DiLA²) delivery system. The second platform is their TransKingdom RNAi (tkRNAi), which is an expressed RNA in a bacterial delivery system. These approaches give Marina Biotech the flexibility to optimize oral, systemic and local delivery of RNAi-based therapies to target a wide range of human diseases based on the unique characteristics of the cells and organs involved in each disease.

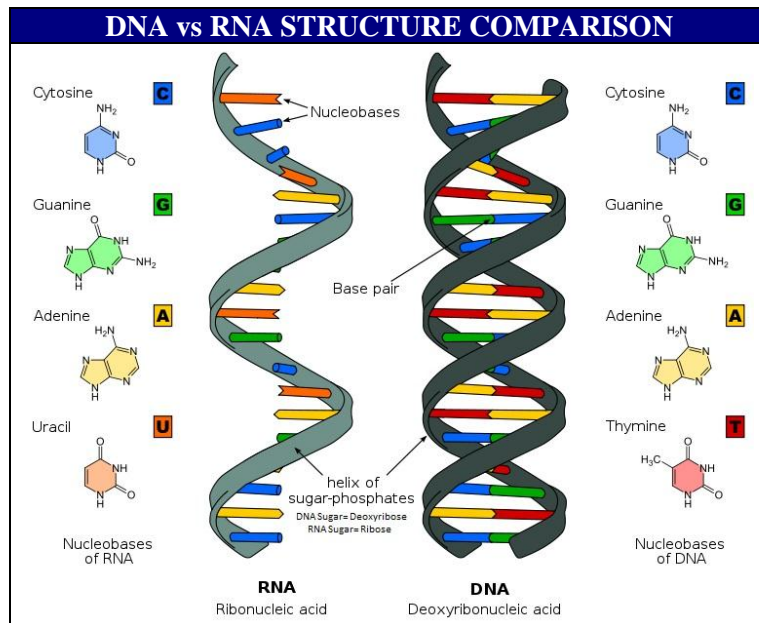
Calendar Quarter	MILESTONES & EVENTS (estimates subject to significant change)	
	CEQ508 (tkRNAi)	tauRNAi (Debiopharm)
Q1 2011	<ul style="list-style-type: none"> ✓ Toxicology for Phase II ✓ Initiate Phase Ib/IIa – FAP 	
Q4 2011		
Q2 2012		Final Candidate Selection
Q3 2012	Data Phase Ib/IIa – FAP	Initiate Phase I - Bladder
Q1 2013	Initiate Phase IIb/III - FAP	
Q1 2014		Data Phase I- Bladder
Q3 2014	Data Phase IIb/III - FAP	Initiate Phase II - Bladder
Q2 2015	FDA Approval – FAP	
Q3 2015	Launch - FAP	Data Phase II - Bladder

Source: Marina Biotech and LifeTech Capital Estimates

RNA in Gene Expression and Regulation

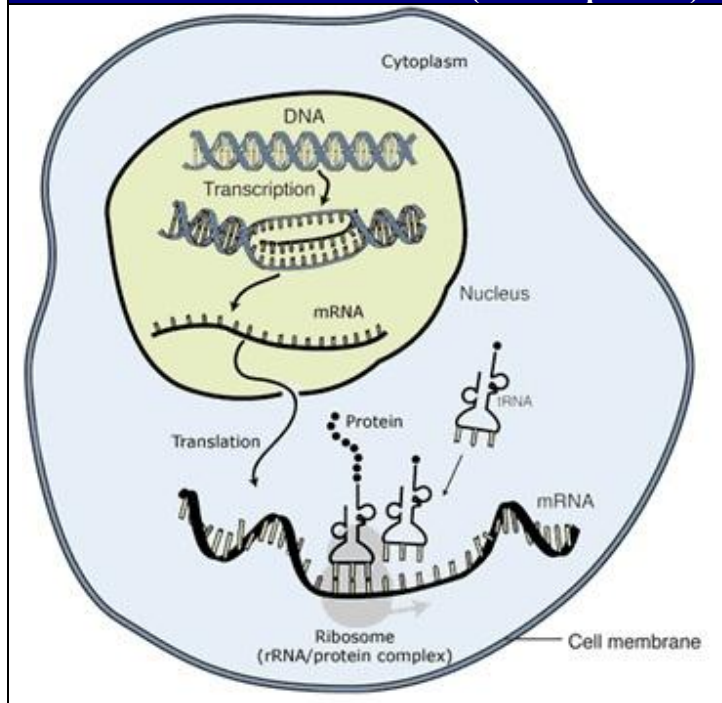
Ribonucleic Acid (RNA) is an essential macromolecule found in all known living things. Though structurally similar to the more commonly well known Deoxyribonucleic Acid (DNA), RNA differs in a few very important ways. As their names suggest, DNA and RNA are both nucleic acids, composed of repeating units of nucleotides. Each nucleotide consists of a sugar, a phosphate and a nucleic acid base. The sugar in DNA is deoxyribose while the sugar found in RNA is ribose, which contains an extra hydroxyl group. Another major difference is that RNA contains the nucleobase (nucleic acid base/nucleotide base/nitrogenous base) Uracil in place of Thymine found in DNA.

There are different types of RNA that can be signal stranded, double stranded or can have complex 3-dimensional confirmations where a single strand folds back on to itself in certain regions. These different types/confirmations have many very important biological functions inside of the cell.



Source: Virginia Hughes and LifeTech Capital

RNA's Role in Protein Production (Gene Expression)



Source: National Human Genome Research Institute

One of the primary functions of RNA is to facilitate the translation of DNA into protein. This process begins in the nucleus of the cell with a series of enzymatic reactions that transcribe DNA into heterogeneous nuclear RNA (hnRNA) by complementary base pairing. hnRNA is a direct copy of DNA and therefore it contains exons and introns, which are coding (regions of the RNA that are directions for protein production) and noncoding regions, respectively. hnRNA undergoes post-transcriptional processing that involves removal of the introns and the addition of a cap to the 5' end of the molecule, as well as the addition of multiple adenines (poly A tail) to the 3' end of the single stranded RNA molecule. The result is called messenger RNA (mRNA). mRNA is transported out of the nucleus into the cytoplasm of the cell, where cellular machinery made up of proteins as well as other forms of RNA (tRNA and rRNA) read and "translate" the code found in mRNA to produce a protein that the original gene in the DNA had coded for. In this way, mRNA functions as a carrier or "messenger" for information from the cells DNA to the protein synthesizing cellular machinery.

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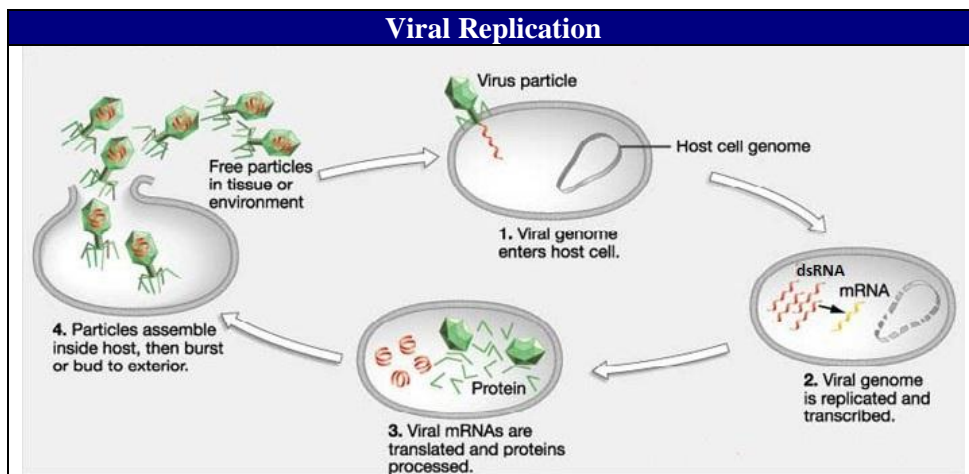
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RNAi - One of Nature's Best Kept Secrets

RNA Interference (RNAi) is a post transcriptional system found within living cells central to the regulation of gene expression that until recently had slipped below the radar of medicinal science. The first evidence of this process was discovered in by a plant virologist while attempting to introduce foreign RNA into a petunia in order to increase the intensity of the pigmentation in the flower. Instead of seeing the desired effect, inhibition of the intended protein production was observed and partially or fully white flowers were produced. After further investigation it was discovered that this phenomenon was highly conserved along diverse branches of the evolutionary tree from plants, to fungus, to round worms, to man. The official discovery of RNAi has been accredited to work done by Craig C. Mello and Andrew Fire in a paper submitted to Nature that detailed potent gene silencing effects after injecting double stranded RNA into *C. elegans* (round worm). As a result of the work, the term RNAi was born and Mello and Fire received the 2006 Nobel Prize in Medicine.

RNAi is a naturally occurring process that regulates gene expression through the inhibition of mRNA translation. This occurs both endogenously by micro RNA (miRNA) activation of RNAi, as well as through foreign triggers such as short interfering RNA (siRNA) derived from foreign dsRNA. miRNAs are endogenous non-coding RNA's that are important in activating RNAi, and hence certain gene expression, especially in early development. miRNA and dsRNA undergo different methods of processing in the cell, but both use the same cellular machinery/mechanism of action downstream to

invoke RNAi.

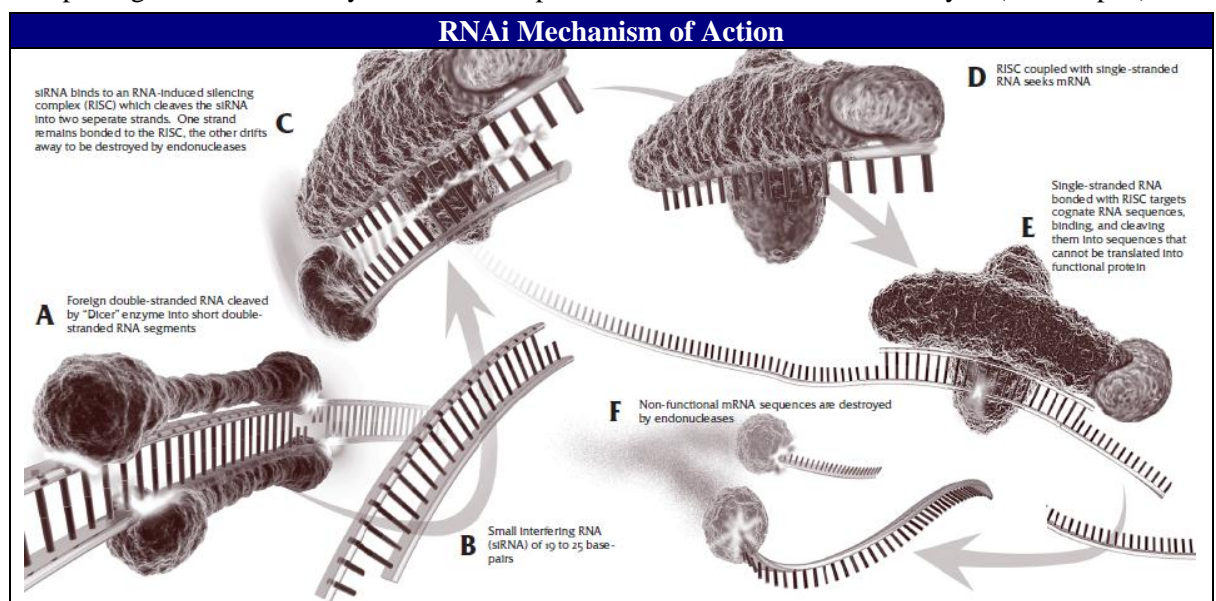


Source: King Saud University

One of the theories behind the development of RNAi is it may be an evolutionary mechanism by cells to protect against, among other things, certain types of viral infections. Certain viruses, such as double stranded RNA viruses (dsRNA viruses), contain RNA as their primary genetic material. In order to replicate these viruses first bind to a specific protein or receptor on the surface of a healthy cell. The virus then in a sense “hijacks” the internal machinery of the cell it is attached to. It injects its viral

RNA into the cell where it is associated into its transcriptional capabilities and viral proteins are produced. The viral RNA then copies itself and is packaged into the newly formed viral protein. These new viruses then lyse (break open) the infected cell and move on to infect new healthy cells.

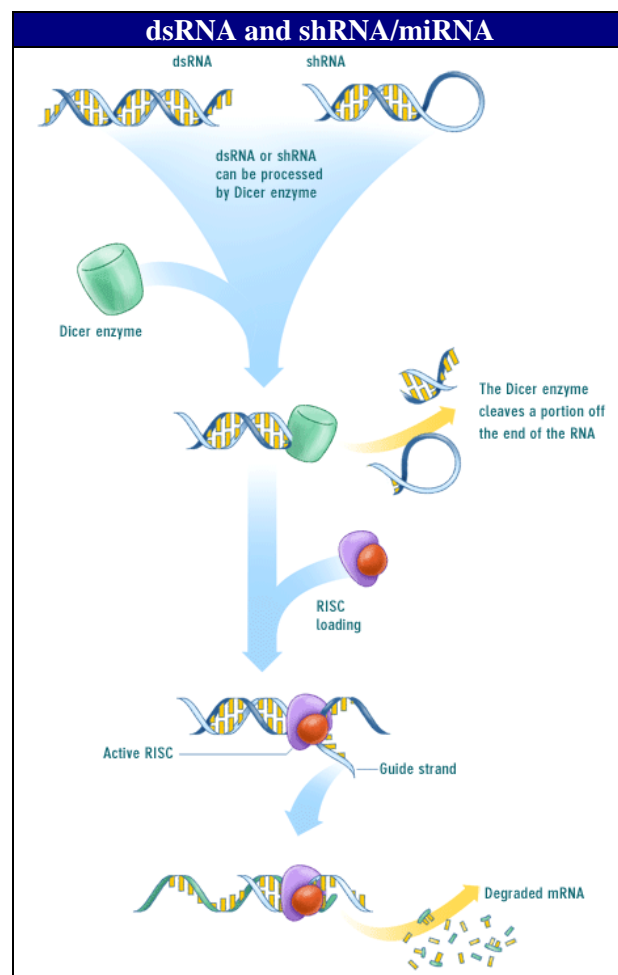
RNAi is thought to be a cellular response to these certain types of viral infections. Within the cytoplasm of the cell are proteins that have the task of finding the foreign genetic material and stopping the production of the encoded protein.



Source: University of Georgia

Foreign dsRNA is first cleaved by an enzyme called dicer into small double stranded fragments that are typically from 19 to 25 base pairs long. These segments are called short interfering RNA's (siRNA). The siRNA is recognized by RNA-induced silencing complex (RISC) in the cytoplasm, where it is incorporated into the molecule and the two strands are unwound. One strand, called the passenger strand, is ejected from the complex and degraded through normal cellular processes by endonucleases. The now "loaded" or active RISC complex carries the other strand, called the guide strand, of RNA to corresponding mRNA's in the cytoplasm that have the complementary code of the guide strand (i.e. the same code as the originating passenger strand). Once bound in a site specific manor to the mRNA the RISC complex cleaves the mRNA into sequences that cannot be translated into functional proteins. The non-functional mRNA sequences are then degraded in the cytoplasm by endonucleases. The still loaded RISC complex then moves on to find another matching mRNA and the process continues.

The miRNAs mentioned earlier use the same internal machinery to inhibit protein production, though they differ in structure, origination and method of processing before integration into the RNAi machinery. miRNAs are dsRNAs that originate from within the cell (endogenous). They are processed from their primary pre-miRNA structure into a "short hairpin" RNA (shRNA) structure, where a single stranded RNA folds back upon itself to form a hair pin like shape. The shRNA then acts similarly to the dsRNA in the example above where it is cleaved by dicer to produce siRNAs that go on to be incorporated into the RISC complex inducing gene specific silencing.



Source: Marina Biotech

When the researchers first mentioned in this section introduced extra copies of the gene for chalcone synthase, an enzyme key to the pigmentation of flowers, into a petunia in an attempt to intensify the color of the flower, they inadvertently stumbled across an ancient cellular mechanism that produces the exact opposite effect. RNAi not only inhibited the production of the "extra" copies of the pigmentation enzyme, but also inhibited the mRNA messengers from the endogenous genes found within the organism, hence white flowers. Once the mechanism of action was more clearly defined by the work of Mello and Fire, the medicinal opportunities of the discovery became much more apparent. Essentially, RNAi holds the potential to selectively turn off any gene in the genome.

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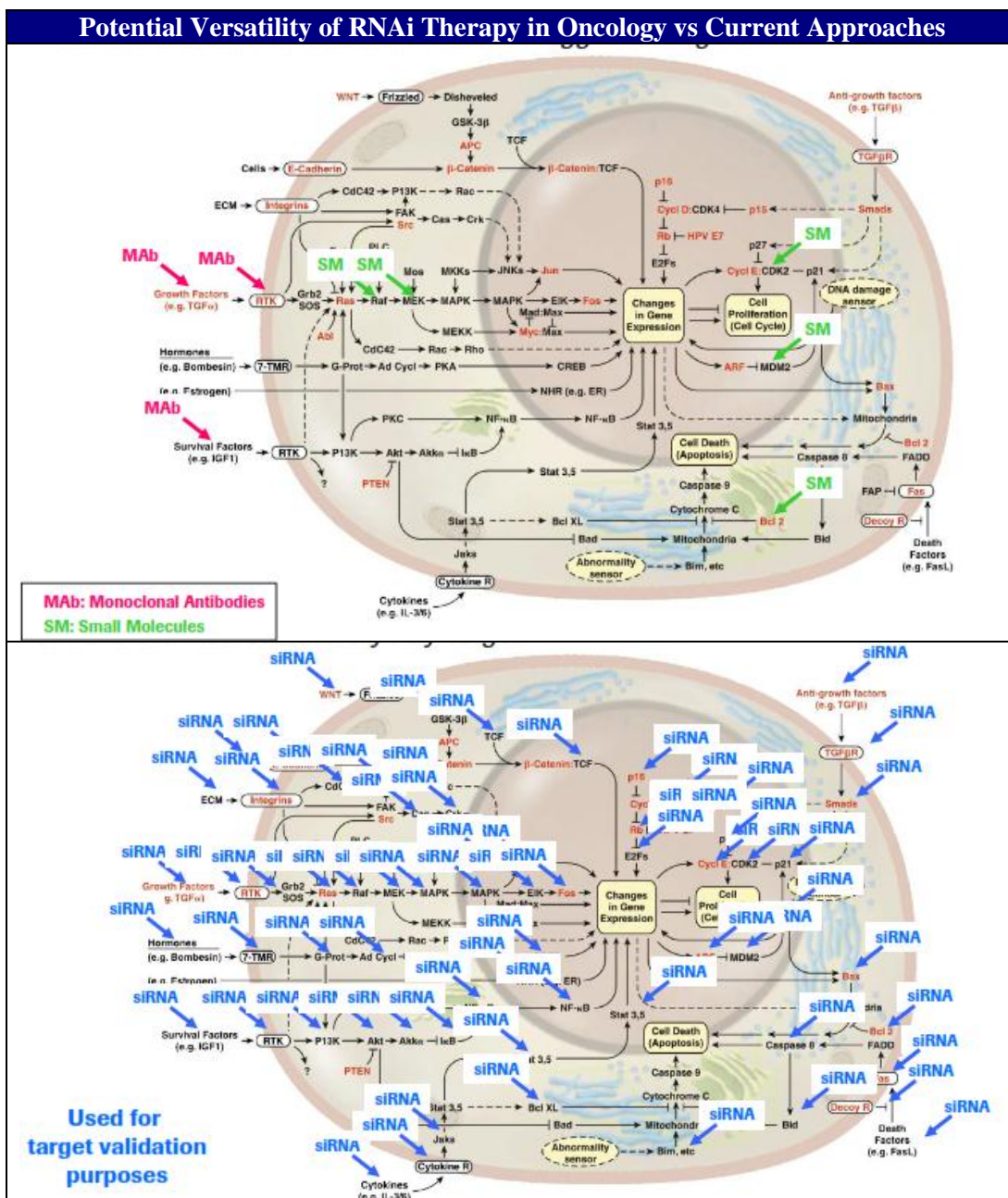
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RNAi's Potential Therapeutic Value

The therapeutic value of RNAi based treatments could potentially be enormous. Most disease, in simplest terms, is the result of malformed and/or misused proteins in the body. Most therapies to date are designed to target and bind to these errant proteins in order to inhibit their enzymatic activity and therefore end the cascade signal that is causing the disease/disorder. This can be accomplished through designing small molecule inhibitors that bind to the active site of enzymes such as kinases, or by designing much larger recombinant protein molecules, such as monoclonal antibodies that can target ligands and/or the receptors to which they bind. Though a valid and proven approach to combating disease, these techniques do have their limitations. The decoding of the human genome has led to an immense amount of information regarding our molecular biology and how errant genes contribute numerous different diseases found within the body. Somewhat ironically though, most of all the new potential targets for therapeutics that have been recently discovered are considered “un-druggable” by current conventional therapies. Most current therapies are targeted towards extracellular proteins, cell-surface receptors, and enzymes with well-defined catalytic sites for which the design molecules will have specificity and strong affinity. These types of targets make up what is known as the “druggable” genome. RNAi as a construct has the potential to silence any gene in the human genome, and therefore has the ability to target many (if not all) of the potential targets that have thus far been deemed un-druggable. This opens possibilities across many different diseases (if not all) where traditional techniques have been unsuccessful or are in need of improvement.

In addition to the broad potential applications of RNAi, it also has a number of other characteristics that are favorable to current drug development methods. Lead optimization under current methods can take years; in contrast RNAi therapy development can shorten the timeframe of this step significantly from years to months. The broad utility of the platform and uniform mechanism of action allows for the potential treatment of multiple targets with predictable pharmacodynamic and pharmacokinetic properties. Longer duration of action and a catalytic process of target inactivation could also prove to be advantageous as this field matures.



Potential Advantages of RNAi Therapies over Conventional Drugs

	RNAi Drugs	Conventional Drugs
Druggable genome	~23,000 (All)	~ 7,000 (~30%)
Time for lead generation	2-5 months	2-3 years
Treat multiple targets with similar PK/PD	Yes, simple	No, long optimization
Target inactivation	Catalytic	Non-catalytic
Duration of action	Up to 3 weeks	Usually hours, sometimes days

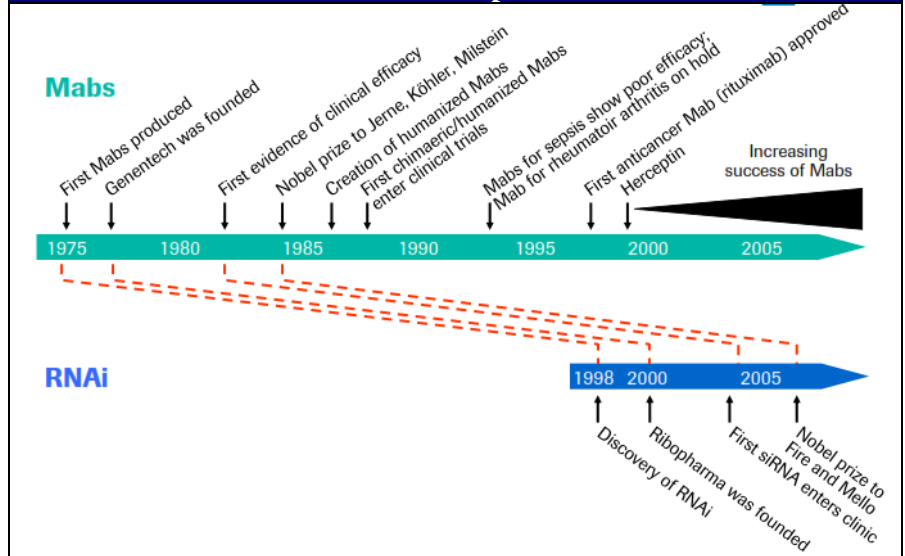
Source: RXI Pharmaceuticals

While RNAi is a fairly new concept in medicine, there have been previous successes in RNA-based therapeutics. ISIS Pharmaceuticals (Nasdaq ISIS: Not Rated) has had success in developing antisense oligonucleotide RNA therapeutics. Though similar in some ways to RNAi, antisense therapy is a different approach to gene expression knockdown. RNA antisense uses a single stranded RNA that is complementary to the mRNA to be targeted by the therapy. This causes a mechanical inhibition of the translation process that would produce the protein the mRNA coded for. RNA antisense does not utilize the mechanism of action found in RNAi (i.e. Dicer, RISC complex,

etc.). ISIS has one drug approved using this therapeutic technique called Vitravene® (Fomivirsen). Vitravene® was approved by the FDA in 1998 for the treatment of cytomegalovirus (CMV) retinitis in AIDS patients. Cytomegalovirus retinitis is an inflammation of the retina caused by CMV infection that can lead to blindness. Vitravene® is the antisense strand of mRNA essential to CMV replication inside the cell and hence blocks the production of viral proteins. Improvements in HIV therapy since the approval of Vitravene® has decreased the mortality rate for those infected and has concurrently decreased the incidence of many opportunistic infections, including CMV retinitis. As a result, Novartis no longer markets Vitravene, but its approval is strong validation of RNA based therapies potential.

While development of RNAi is still in the early stages and inherently carries risk, we believe that success in this field would create a paradigm shift in how we view and treat disease. Parallels to current RNAi development could be drawn to the early stages of therapeutic monoclonal antibody development completed decades earlier. There are now approximately two dozen FDA approved therapeutic monoclonal antibodies to treat various different human diseases. Sales of therapeutic monoclonal antibodies (and Fc-fusion proteins) exceeded \$45 billion dollars worldwide in 2009.

Monoclonal Antibodies Development Timeline vs RNAi



Source: Roche Kulmbach GmbH

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TransKingdom RNA Interference (tkRNAi) Platform (CEQ508)

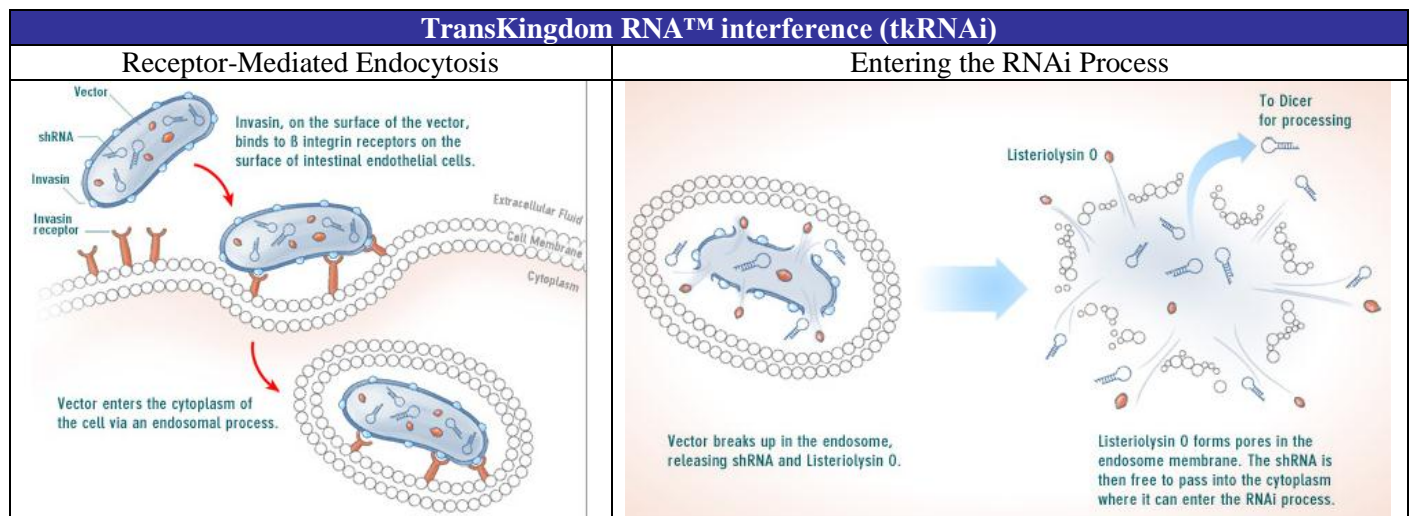
Acquired as a result of Marina Biotech's buyout of Cequent Pharmaceuticals in July 2010, their tkRNAi platform involves a genetically modified live, non-pathogenic, *E.coli* strain of bacteria with two proteins, invasin and listeriolysin, to deliver shRNA to cells in the intestinal tract. The engineered bacteria allows for oral delivery of the drug due to its unique properties:

Quantity: Each individual bacterium can deliver several copies of the shRNA upon each delivery event. This is due to the production of high levels of shRNA, specific to β -catenin mRNA.

Targeting: It has the ability to efficiently enter the epithelial cells lining the intestine. This is due to the protein invasin on the outer surface of the bacteria. Invasin interacts with β 1-integrin receptors on the outer membrane of the epithelial cells which facilitates intracellular uptake via an endosomal process.

Delivery: It then releases the shRNA into the cytoplasm of the epithelial cell. This is due to a protein engineered into the bacterium, listeriolysin O, which forms pores selectively rupturing the endosome and releasing the shRNA into the cytoplasm.

Action: The shRNA in the cytoplasm of the epithelial cell is processed by the RNAi machinery to induce degradation (silencing) of β -catenin mRNA. The suppression of β -catenin protein has been shown to arrest or slow the growth of the intestinal cells responsible for polyp formation.



Source: Marina Biotech

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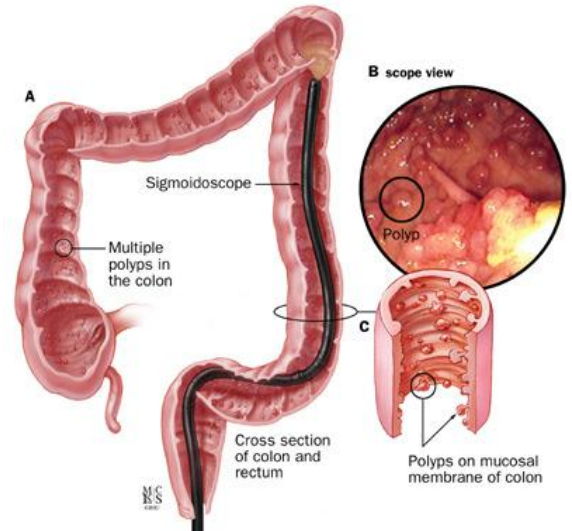
<http://www.ncbi.nlm.nih.gov/books/NBK6085/>

CEQ508 for Familial Adenomatous Polyposis (FAP)

Marina Biotech's first candidate reaching human trials is CEQ508 for Familial Adenomatous Polyposis (FAP). FAP is an inherited genetic disorder characterized by the development of polyps in the large intestine and rectum that can eventually lead to cancer of those regions if not treated surgically. One of the known causes of FAP is a genetic mutation in the Adenomatous Polyposis Coli (APC) gene. The APC gene controls the production of APC protein which, among other functions, is integral in the regulation of cellular levels of beta-catenin (β -catenin). Regulation of β -catenin prevents genes that stimulate cell division from being turned on too often and therefore prevents cell overgrowth. The APC gene is classified as a tumor suppressor gene as it keeps β -catenin level in check; if it is dysfunctional then the polyps that are characteristic of FAP arise.

Polyps can develop in patients' with classic type familial adenomatous polyposis as early as in their teenage years and without treatment the polyps will eventually become malignant (cancerous). For many patients, complete colectomy, or surgical removal of the entire large intestine (and sometimes rectum), remains as the primary treatment for FAP. This procedure is typically performed in the late teens or the early twenties. However, surgical intervention is not curative as the risk of polyps forming in the remaining portions of the intestinal tract and in the small intestine remains after colectomy. The average age at which an individual with classic familial adenomatous polyposis develops colon cancer is 39 years of age. There is a variant of FAP where there is a mutation at a different location on the APC gene than in the classic type. The variant is called attenuated familial adenomatous polyposis. Polyp growth is delayed in the attenuated form of the disease, but the end results are often the same as the average age of colorectal cancer onset for attenuated familial adenomatous polyposis is 55 years.

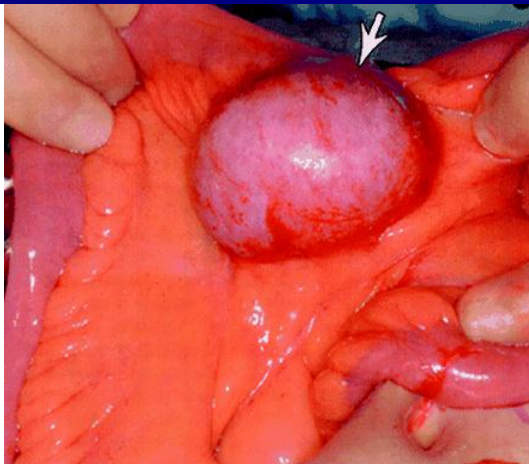
Familial Adenomatous Polyposis



Source: Johns Hopkins Medicine

Mutations in a separate gene from APC can cause a different form of FAP. Mutations in the MUTYH gene cause the autosomal recessive familial adenomatous polyposis, also called MYH-associated polyposis. The MUTYH gene encodes for the DNA repair enzyme MYH glycosylase. MYH glycosylase is responsible for fixing mistakes that arise during DNA replication with the nucleobase guanine. When the enzyme is not functional, mistakes in the DNA are not corrected and build up over time in the patient's genome. This increases the likelihood of cell overgrowth, leading to colon polyps and the possibility of colon cancer. MYH-associated polyposis differs from the classic and attenuated versions of FAP as the

Desmoid Tumor on Small Intestine



Source: American Journal of Roentgenology

polyps are caused by different genetic defects within the cell. MYH-associated polyposis has an autosomal recessive inheritance pattern, and polyp growth is much less numerous (often fewer than 100) and seen later in adulthood in comparison to the other forms of FAP.

In patients with classic familial adenomatous polyposis, the number of polyps increases with age starting in adolescence. Hundreds to thousands of polyps can develop in the colon and large intestine in both classic and attenuated FAP, benign and malignant tumors are sometimes found in other places in the body, including the duodenum (a section of the small intestine), stomach, bones, skin, and other tissues. By age 35, 95% of people with FAP will have developed polyps and the majority will experience serious health complications as a result. These complications include; bleeding with the potential to develop anemia, intestinal obstruction, abdominal pain, severe diarrhea and/or constipation. People with FAP are also at increased risk for developing desmoids tumors,

which usually occur in the tissue covering the intestines. Desmoid tumors are fibrous, locally aggressive, benign growths that can be damaging to nearby organs. They often recur after complete resection and are frequently an important factor in evaluating whether to perform a colectomy in a patient.

The reported incidence of FAP varies from 1:7,000 to 1:22,000 people. Marina Biotech itself estimates the incidence to be about 1:10,000 people.

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Clinical Trials

Marina Biotech's first RNAi drug candidate CEQ508 (from their tkRNAi platform) commenced human clinical trials for Familial Adenomatous Polyposis (FAP), an inherited genetic disorder triggering excessive polyp growth in the large intestine and rectum which eventually turns cancerous. CEQ508 is an oral suspension consisting of attenuated bacteria engineered to enter dysplastic tissue and release a payload of short-hairpin RNA (shRNA) to reduce the levels of β -catenin protein.

PHASE Ib/IIa HUMAN CLINICAL TRIAL PROTOCOL

Title	A Phase Ib/IIa Open-Label, Escalating-Doses Study, of the Safety and Tolerability of Single Daily Doses of CEQ508, an RNAi-Based Therapy for Familial Adenomatous Polyposis
# of Patients	Maximum 30 patients (male and female)
Trial Design	Open-Label, Dose-Escalating, Safety and Tolerability study
Ages	18-65 years old
Endpoints	<p><u>Primary:</u> Evaluate/establish general safety for orally administered CEQ508 in a daily dosing schedule and to determine the Maximum Tolerated Dose (MTD) (of 4 planned doses) and/or the Highest Safest Dose.</p> <p><u>Secondary:</u></p> <ul style="list-style-type: none"> Examine shedding of CEQ508 in the stool of patients during and after daily oral dosing with CEQ508 for 28 days, and the 28 day recovery period. Examine gene expression changes after oral dosing of CEQ508 in GI mucosa of FAP patients.
Arm 1:	Dose Escalation Arm: 12 patients, at least 4 patients from each sex
Arm 2:	Stable Dose Arm: 6 patients, who may be newly enrolled or re-enrolled from the 12 patients who were part of the Dose Escalation Phase.
Inclusion	<p>Clinical or genetic diagnosis of FAP</p> <p>Pre and post colectomy; no immediate need for colectomy</p> <p>Known endoscopic history of polyposis</p> <p>Eligible to undergo baseline and endpoint endoscopies</p> <p>Ability to be taken off other chronic FAP medication (Sulindac, Aspirin, etc.)</p> <p>Informed consent</p>
Exclusion	<p>Inability to return for scheduled treatment and assessments</p> <p>Chronic or intercurrent acute medical disorder</p> <p>Significant clinical laboratory and hematology observations</p> <p>Pregnancy, nursing (or anticipated pregnancy)</p> <p>Antibiotic use (current or anticipated antibiotic treatment during the study period; antibiotic use within the past 2 weeks)</p> <p>Active ulcerations or inflammation found at baseline endoscopy</p>
Center	Massachusetts General Hospital, Boston MA.
Contact	Daniel Chung, MD

Source: Marina Biotech, Inc.

On February 24, 2011 Marina Biotech announced they completed the CEQ508 9-month toxicology study in non-human primates which was required for the planned Phase II trial. 18 cynomolgus monkeys were administered daily oral doses of either CEQ508 or a control for 281 days. No CEQ508 adverse responses were identified in clinical observations, body weights and temperatures, serum chemistry, coagulation, hematology, urinalysis, cytokines and gross pathology. Additionally, monthly biopsies of colonic mucosa showed no evidence of local immune activation. Preliminary results

showed No Observed Adverse Effect Level (NOAEL) for long-term daily oral administration of CEQ508 as 1×10^{11} colony forming units (cfu)/day. This was the highest dose administered to these animals. The data was presented on April 4, 2011 in a poster titled "Long term safety observed with CEQ508: An oral RNAi drug targeting beta-catenin of GI polyps" at the annual American Association of Cancer Research (AACR) meeting.

UPDATE:

On June 9, 2011 Marina announced the dosing completion of the first 3 patient cohort. The first cohort received the starting dose of 1×10^8 colony forming units (cfu)/day for up to 28 days of continuous oral dosing. The next cohort will receive 10 times the initial dose (1×10^9 cfu/day) and is expected to start dosing in early 2012. The trial is still on track to complete the dose escalation phase by the end of 2011. The dose escalating phase of the START-FAP trial involves an oral regimen, administered daily for 28 days. The trial is being conducted at Massachusetts General Hospital in adult patients with FAP. Enrollment involves a thorough screening process, including an in-depth assessment and assurance that the patient meets the study inclusion criteria, and occurs between 7 and 30 days prior to drug dosing. In addition, the patient must undergo endoscopy procedures one to seven days prior to the initiation of drug dosing. Following the course of drug administration, a second round of endoscopy procedures will be performed. Biopsies taken at baseline and end-of-treatment time points will allow safety evaluations as well as analysis of biomarker changes.

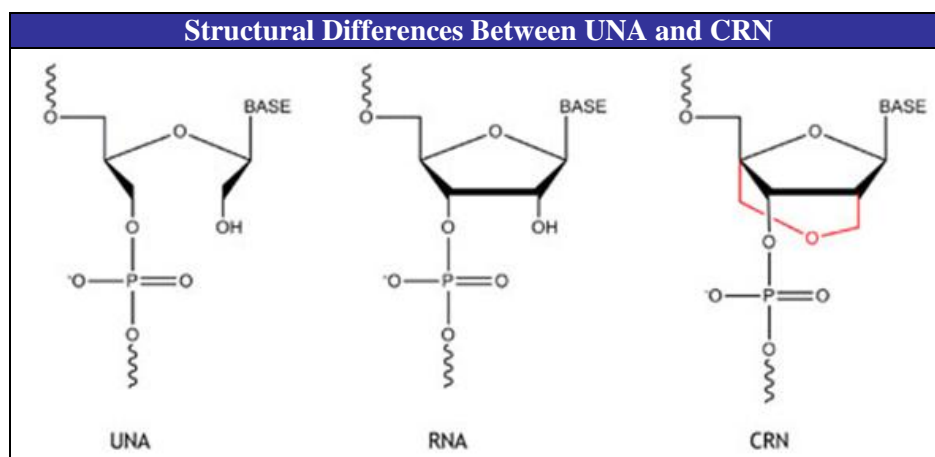
Tau RNA Interference (tauRNAi) Platform

The tauRNAi platform incorporates Marina Biotech's UsiRNA construct technologies with their DiLA² liposome delivery system. By merging these two technologies, Marina Biotech can customize both the UsiRNA construct and the DiLA² liposome characteristics to target specific tissues and diseases resulting from overexpression of specific proteins.

UsiRNA Constructs

UsiRNA constructs involve the strategic placement of Unlocked Nucleobase Analogs (UNA) and Conformationally Restricted Nucleotides (CRN) to provide stability to nucleases, mitigate a cytokine response, and reduce off-target effects, while maintaining activity against the intended target.

- Unlocked Nucleobase Analogs (UNA) do not have the bond between the two adjacent carbon atoms that form the ribose portion of RNA. UNAs are considered to be conformationally flexible.
- Conformationally Restricted Nucleotides (CRN) have the ribose portion locked into a rigid conformation by a small chemical linker.

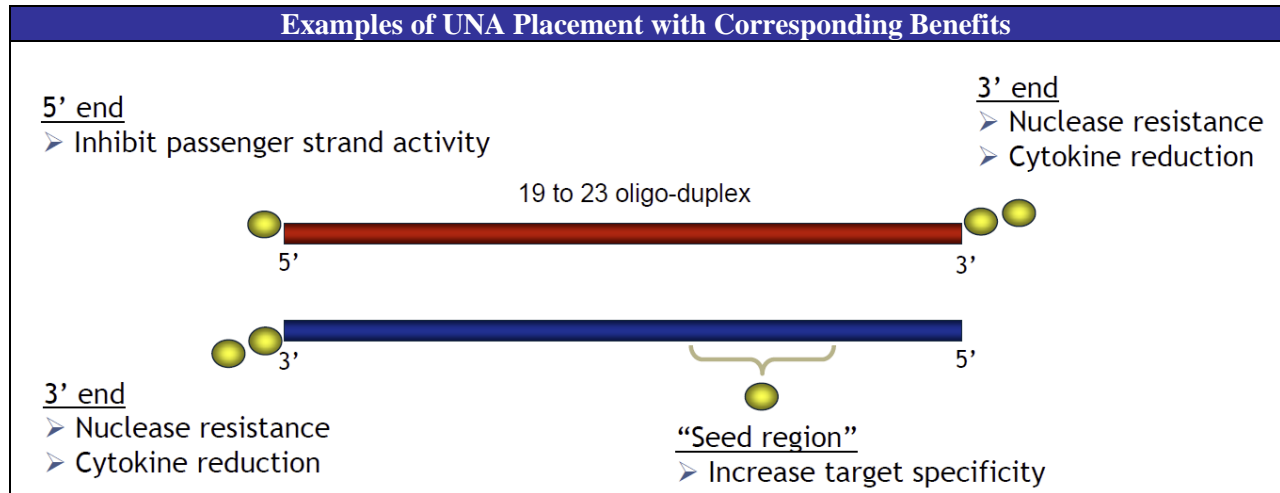


Source: Marina Biotech

When UNA and CRN monomers are included in a siRNA, Marina Biotech can customize the characteristics to yield greater specificity and activity to the UsiRNA construct due to the following:

- ✓ UNAs prevent passenger strand participation in RNAi, thus lowering the potential for off-target effects

- ✓ UNAs increase the specificity of the guide strand for its intended target mRNA by eliminating microRNA-like off-target effects
- ✓ UNA and CRN provide resistance to nuclease degradation
- ✓ UNA and CRN decrease the potential for a cytokine response
- ✓ CRNs impart the highly stable A-form of RNA to the duplex, resulting in increased thermal stability
- ✓ In addition, CRN substitution into single-stranded oligonucleotides affords unprecedented stability of single-stranded RNA thus enabling development of novel RNA-based therapeutics that function outside of the RNAi mechanism.



Source: Marina Biotech

DiLA² (Di-Alkylated Amino Acids) Delivery Technology

Update:

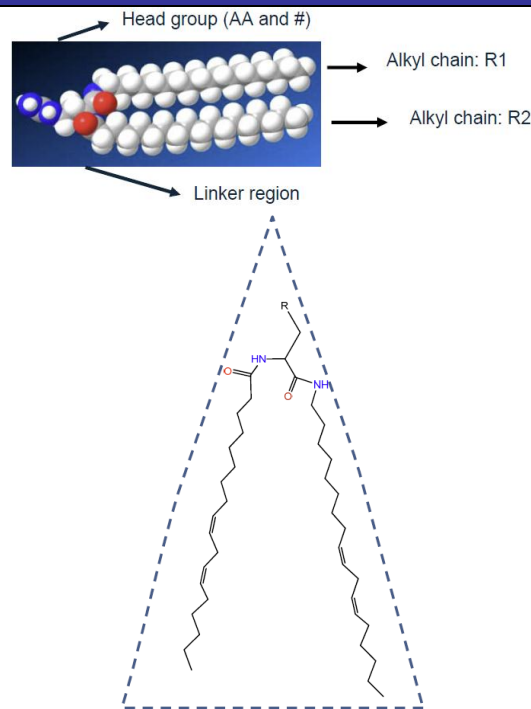
Marina Biotech announced the publication of data for their DiLA²-based delivery technology for systemic administration of siRNAs in the latest issue of *Molecular Therapy*. The paper titled “*An Amino Acid-based Amphoteric Liposomal Delivery System for Systemic Administration of siRNA*” describes the development of an amphoteric DiLA² delivery system which enabled efficient knockdown of multiple targets in liver. One of the DiLA²-based delivery formulations administered systemically, resulted in >80% knockdown of ApoB, TTR, Factor VII and PCSK9 messenger RNA (mRNA) with a single 2 mg/kg dose. The ED₅₀ (effective dose in 50% of population) values for knockdown of these mRNAs ranged from 0.1 to 0.25 mg/kg. The formulation was well tolerated with single and multiple dose regimens. **Importantly, frozen DiLA²-based formulations demonstrated physical and chemical stability for greater than one year, a necessary attribute for developing liposomal-based siRNA drug products.** The paper can be access at: <http://www.nature.com/mt/journal/vaop/ncurrent/abs/mt201156a.html> Marina Biotech’s DiLA² delivery system is currently being developed in their bladder cancer program which is partnered with Debiopharm (see *Debiopharm Partnership for Bladder Cancer*).

The DiLA² Technology allows custom liposome formulations from dialkylated amino acids for delivery of UsiRNA. Key aspects of the delivery system can be modified such as charge, linker and acyl chains so that the properties of the liposome can be optimized for delivery to a specific target tissue. It is also designed to permit inclusion of peptides to improve a variety of delivery characteristics including encapsulation of nanoparticles, cellular uptake, endosomal release and cell/tissue targeting. A DiLA²-containing liposome has several potential advantages over other liposomes:

- ✓ a structure that may enable safe and natural metabolism by the body
- ✓ the ability to adjust liposome size, shape, and circulation time, to influence bio-distribution
- ✓ the ability to attach molecules that can influence other delivery-related attributes such as targeting and cellular uptake

DiLA2 Liposomal Delivery - Design and Characteristics

- Composed of unique combinations of head groups, linkers and alkyl chains
- Self-assembly into liposomes with siRNA and other components
 - Size range from 60 - 130 nm; controlled by process
 - Typical range 80 to 120 nm
 - >80% siRNA encapsulation
 - Narrow polydispersity index (PDI)
 - One year stability has been demonstrated
- Cone Shaped
 - Small head-group
 - Unsaturated and/or asymmetric tails
- Hexagonal Phase Formation
 - pH-dependent interaction with anionic membrane lipids
 - Transitions from lamellar phase to inverted hexagonal phase
- Stability
 - Chemically stable amide linkages
 - Proteolytic elimination pathways
- Categories
 - Ionizable pKa - pKa between 5 to 7
 - Constitutive cationic - pKa greater than 7



Source: Marina Biotech

pH-Responsive Formulations Facilitate Uptake and Endosomal Escape

Ionizable Formulations

- Most appropriate for DiLA2 having pKa between 5 and 7
- Numerous head-group functionalities

Amphoteric Formulations

- Most appropriate for DiLA2 having pKa >7
- Formulated with anionic ionizable component

Source: Marina Biotech

Preclinical Programs

Marina currently has 2 programs in preclinical development for bladder cancer and hepatocellular carcinoma (HCC), both of which utilize their UsiRNA/DiLA2 (i.e. tauRNAi) proprietary platform. Due to their early stage of development and uncertain timelines we have decided to not include them in our financial modeling. Investor should note that these programs could provide significant potential upside given future successes in their development.

Bladder Cancer

UPDATE:

On November 29, 2011 Marina Biotech announced that the Marina/Debiopharm Research and Development Team had selected and confirmed the lead DiLA2 formulation for a RNAi-based therapy for the treatment of non-muscle invasive bladder cancer. The team had also advanced the lead UsiRNA candidates towards in vivo evaluation to identify a candidate drug product for continued development. The joint R&D Team expected to **select the lead candidate in early 2012.**

On February 3, 2011 Marina Biotech entered into an exclusive partnership agreement with Swiss-based Debiopharm (private) for the development and commercialization of Marina Biotech's pre-clinical RNAi program in non-muscle invasive bladder cancer. Debiopharm will pay Marina Biotech up to \$25M based on predefined research and development milestones as well as royalties on sales. All Marina Biotech research and development costs for the bladder cancer program will be funded by Debiopharm beginning in February 2011. Debiopharm will be responsible for the development and commercialization of any products arising from the partnership.

Swiss-based Debiopharm Group was founded in 1979 and is a global biopharmaceutical group with a focus on drug development and companion diagnostics and has brought five products to the market, including Eloxatin®/Elplat®, a DACH platin for the treatment of colorectal cancer and Decapeptyl®/Trelstar®/Pamorelin® for the treatment of prostate cancer. More information can be found at <http://www.debiopharm.com>

Principle preclinical testing in this indication has revolved around inhibition of the “inhibitor of apoptosis” (IAP) family target, survivin. Survivin is known to be up-regulated in many cancer models, and is considered “undruggable” by current small molecule and recombinant protein approaches. Marina has also tested RNAi inhibition of survivin in combination with other known molecular alterations in bladder cancer that play key roles in cancer cell progression including Fibroblast Growth Factor 3 (FGFR3), HRAS and Polo-Like Kinase 1 (PLK-1). In their most recent published preclinical test, survivin UsiRNA was paired with FGFR3, HRAS, or PLK1 UsiRNAs, and compared then to the survivin UsiRNA alone. All UsiRNAs were encapsulated in the Company's proprietary DiLA2-based formulation, and delivered directly to the bladder of a mouse bladder cancer model.

Results (as compared to survivin UsiRNA alone):		
Combination	Tumor Bioluminescence Reduction	Survivin mRNA Expression Reduction
survivin/PLK1	~30%	~30%
survivin/FGFR3	~30%	~30%
survivin/HRAS	~40%	~50%

Source: Marina Biotech

An earlier study by Marina had reported a dose-dependent decrease greater than 90% reduction in bioluminescence using a PLK1 UsiRNA in a mouse bladder cancer model, with at a dose of 1 mg/kg. Marina has also shown activity of PLK1 UsiRNA and survivin UsiRNA in non-human primate bladder cancer models, where there were no reported alterations in hematology parameters or apparent induction of cytokines.

Investors should note there are approximately 70,000 new cases of bladder cancer diagnosed each year in the U.S. with over 14,000 deaths annually.

Hepatocellular Carcinoma (HCC)

Marina has shown promising results in preclinical studies using systemic delivery of their tauRNAi construct to inhibit certain gene expression in liver cells. PLK1 UsiRNA and survivin UsiRNA have both shown activity in mouse liver cancer models as well as non-human primate models. The non-human primate study model's safety profile following a single administration reported no increase in ALT and AST levels at 24 or 48 hours post-dose nor elevation in other liver enzymes or kidney function markers. In addition, there were no alterations in hematology parameters and no apparent induction of cytokines.

Marina, using a mouse model and a UsiRNA targeting FVII mRNA in hepatocytes (liver cells), was able to demonstrate 50% inhibition of FVII protein activity from a single systemically delivered dose of 0.08 mg/kg, further demonstrating the potential of the therapeutic and its proprietary delivery.

Investors should note there are approximately 24,000 new cases of liver cancer diagnosed each year in the U.S. with nearly 19,000 deaths annually.

Financial Model Assumptions

The reported incidence of the genetic disorder Familial Adenomatous Polyposis (FAP) varies from 1:7,000 to 1:22,000 people, which yields between 21,000 and 66,000 patients in the U.S. and which we are estimating 30,000 patients in our model. We are also estimating a sales price of \$25,000 per year per patient which is in line for a first-in-class drug treating a genetic disorder. However, we only expect 20% of patients at any given time will be prescribed CEQ508 due to their current disease status and treatment stage as well as potential financial considerations. This results in a peak market potential of \$150M (30,000 x \$25,000 x 20%). We have not yet included ex-U.S. revenue in our model as the regulatory pathway has not yet been determined however we expect ex-U.S. pricing will be significantly lower due to economic considerations.

We have not yet included Marina Biotech's second drug candidate, combining UsiRNA with DiLA² delivery, in bladder cancer as the regulatory pathway has not been finalized. Investors should note there are approximately 70,000 new cases of bladder cancer diagnosed each year in the U.S. with over 14,000 deaths annually.

Although it is possible that Marina Biotech may raise sufficient operating funds for development through warrant conversions and potential partnerships, we believe that the company will be required to raise additional funds through our forecast horizon and have included future share issuances in our financial model.

Roche Cuts RNAi Development

On November 17, 2010, Roche announced plans to reduce their work force by 4,800 positions worldwide over the next two years with the largest reductions in sales and marketing and in manufacturing. However, Roche also decided to discontinue all RNAi research in Kulmbach, Germany, Nutley, New Jersey and Madison, Wisconsin. This was noteworthy as Roche had paid \$331M upfront to Alnylam in July 2007, acquired Mirus Bio for \$125M in July 2008 and up to \$18M upfront (over 2 years) to Tekmira in May 2009.

Although adversely affecting investor confidence in the space, we believe Roche was driven to eliminate costs associated with early-development projects and long time horizons. In other words, Roche exited their in-house biotech efforts in solving the technical issues of RNAi. Roche stated *"While there has been some progress in solving the scientific and technical hurdles with RNAi, the hurdles remain, in particular cell specific delivery. Also, the most promising indications where we could achieve successful delivery do not fit with our strategy."* Instead, Roche stated they are *"actively pursuing partnering options for our RNA sites and assets"* which indicates a return to the traditional biotech partnering model for high-risk, high-reward development projects.

Pfizer Expands Alliance with Santaris Pharma A/S

On January 4, 2011, Pfizer expanded their existing alliance with Denmark-based Santaris Pharma A/S for access to Santaris Pharma A/S Locked Nucleic Acid (LNA) drug platform to develop RNA-targeted drugs. Pfizer made an upfront payment of \$14 million with potential milestone payments of up to \$600M as well as royalties on sales of up to 10 new RNA targets that result in approved drugs. Investors should note that this builds on the original collaboration formed in January 2009 between Santaris Pharma A/S and Wyeth, which was subsequently acquired by Pfizer Inc.

Investors should note that in April 2010, Marina Biotech and Pfizer (NYSE:PFE) began working together where Marina is formulating Pfizer's oligonucleotides in DiLA² formulations for in vivo preclinical evaluation to be performed at Pfizer. Additionally, Marina will design and synthesize UsiRNAs directed against targets specified by Pfizer.

Tuschl I & II RNAi Patents

Although Marina Biotech is not involved, there is a significant ongoing patent issue within the RNAi industry regarding the structure and use of RNAi therapeutics concerning the Tuschl I & II patents¹. **Importantly, our discussions with Marina Biotech management indicate that their UNA (unlocked nucleic acid) construct technology (exclusively licensed from Ribotask ApS) falls outside the scope of the granted Tuschl patents.** Although Marina Biotech is not involved, investors should be aware of the potential for ongoing litigation regarding intellectual property within the RNAi development space regardless of its merit.

UPDATE:

On March 15, 2011 Alnylam Pharmaceuticals, Inc. (Nasdaq:ALNY), the Max Planck Society, the Whitehead Institute for Biomedical Research and the University of Massachusetts resolving their ongoing litigation regarding the Tuschl patents. The Massachusetts Institute of Technology, formerly a party to the litigation, also agreed to the terms of the settlement. The litigation was initiated in June 2009 and scheduled for trial in March 2011 in the United States District Court for the

District of Massachusetts in Boston, Massachusetts. **As part of the settlement agreement, Max Planck, Whitehead, UMass, and MIT have agreed that future prosecution of the Tuschl I and Tuschl II patent families in the United States should be coordinated and led by Max Planck in addition to their ongoing prosecution of the Tuschl II patent family outside the United States. UMass will lead future prosecution of the Tuschl I patent family outside the United States.** Further, Alnylam has granted UMass the right to sublicense the U.S. Tuschl II patent family to Merck (NYSE:MRK), subject to certain Alnylam third-party obligations and other limitations, in exchange for a share of certain future sublicense income.

Tuschl I Patent: The Tuschl I patent covers the introduction of double-stranded RNA (dsRNA) into an in vitro system that led to gene silencing as the result of cutting the dsRNA into smaller fragments that mediated the destruction of the target messenger RNA (mRNA). Dr. Thomas Tuschl and his collaborators published their findings in March 2000 in *Cell*.²

Tuschl II Patent: The Tuschl II patent determined that the optimal structure of exogenous interfering RNA in mammalian cells was a 21-23 nucleotide dsRNA with one or two nucleotides at each 3'-strand end unpaired (3'-overhangs). This was published in the January 2001 in *Genes & Development*³ where they coined the term "siRNA" for "short interfering RNA." The authors described 21-23 nucleotide synthetic dsRNAs with 3'-overhangs and demonstrated efficient gene silencing in a drosophila cell lysate and later in human cells four months later without further processing of the siRNA molecule.

Research References

¹ Leavitt, John et al., "RNAi Litigation Max-Planck and Alnylam v. Whitehead Institute and University of Massachusetts" Nerac Inc. <http://blog.nerac.com/rmailitigation/>

² Zamore, Tuschl et al., "RNAi: Double-Stranded RNA Directs the ATP-Dependent Cleavage of mRNA at 21 to 23 Nucleotide Intervals" *Cell* Mar 31 2000
http://web.wi.mit.edu/bartel/pub/publication_reprints/Zamore_Cell00.pdf

³ Elbashir et al. "RNA interference is mediated by 21- and 22-nucleotide RNAs" *Genes & Development* Jan 15 2001
<http://www.shutcm.edu.cn/shutcm/zyyjs/xsyd/wxsj/images/2006/12/19/3498.pdf>

Recent Financing Activity

On October 11, 2011, Marina entered into a Purchase Agreement with an institutional investor to purchase up to \$15 million of the common stock over a thirty month period. Marina also entered into a registration rights agreement with the investor pursuant to which the Marina agreed to file a registration statement related to the transaction with the SEC covering the shares that have been issued or may be issued to the investor under the Purchase Agreement. The SEC declared the registration statement effective on October 31, 2011. Marina has the right, at their sole discretion, over a 30-month period following the effective date of the registration statement, to sell up to \$15 million of common stock to the investor, depending on certain conditions as set forth in the Purchase Agreement.

There are no upper limits to the price the investor may pay to purchase shares of common stock, and the purchase price of the shares related to the future funding under the Purchase Agreement will be based on the prevailing market prices of the common stock immediately preceding the time of sales without any fixed discount. Marina controls the timing and amount of future sales, if any, of shares to the investor. The investor does not have the right or the obligation to purchase any shares of common stock on any business day that the price of the common stock is below the floor price of \$0.10. Marina will not issue more than 17,779,127 shares in connection with the Purchase Agreement, unless the average purchase price of all shares of common stock issued by us to the investor equals or exceeds \$0.225 per share.

Marina issued 1,452,785 shares of common stock to the investor as a commitment fee, and is required to issue up to 2,905,569 shares of common stock pro rata when and if the investor purchases the \$15 million of common stock over the 30-month period. Additionally, Marina issued 50,000 shares of common stock as an expense reimbursement to the investor. Marina can terminate the Purchase Agreement at any time without any cost incurred.

On May 17th, 2011, Marina announced the pricing of an underwritten public offering. 22,318,500 units were sold at a price of \$0.31 per unit, for gross proceeds of approximately \$6.9 million to Marina, before deducting underwriting

discounts, commissions and other estimated offering expenses. Each unit consists of one share of common stock and one Series A warrant to purchase one share of common stock. The Series A warrants have an exercise price of \$0.39 per share and are exercisable beginning one year and one day from the date of issuance (subject to stockholder approval of an increase in authorized common stock) and will expire on the fifth anniversary of the date they first become exercisable. In addition, the offering includes 22,318,500 Series B warrants, each to purchase one unit. The Series B warrants are exercisable immediately at an initial exercise price of \$0.31 per Unit, but are subject to adjustment. Beginning at the close of business on the 30th trading day following the date of issuance of the Series B warrants, and effective beginning on the third trading day immediately preceding such 30th trading day, the Series B warrants will be exercisable at a per Unit exercise price equal to the lower of (i) the then-effective exercise price per Unit and (ii) 80% of the closing bid price of the Common Stock on The NASDAQ Global Market on such 30th trading day.

The Series B warrants will expire at the close of business on the 35th trading day following the date of issuance. If fully exercised the Series B warrants will result in the issuance of an additional 22,318,500 shares of common stock and Series A warrants exercisable for an additional 22,318,500 shares of common stock. If fully exercised the 44,637,000 series A warrants are only exercisable pending shareholder approval of increasing authorized common shares. The Company agreed with the investors in the offering to hold a stockholders' meeting by August 15, 2011 to seek stockholder approval for an increase in the authorized shares of Common Stock. If the Company is unable to obtain the required stockholder approval on or before the first anniversary of the issuance of the Series A Warrants, the Company will be required to pay the holders of the Series A Warrants liquidated damages of \$2,500,000 in the aggregate. The offering is expected to close on or about May 20, 2011, subject to customary closing conditions.

In connection with the May underwritten offering, as of June 30, 2011, 7,121,500 of the Series B Warrants had been exercised, and in July 2011, an additional 15,172,000 of the Series B Warrants were exercised prior to their July 12, 2011 expiration date. As a result of the exercises of warrants Marina received additional cash proceeds of approximately \$2.2 million in June 2011, and \$0.9 million in July 2011, with the July cash proceeds being net of refunds as a result of the adjustment in the warrant exercise price on July 5, 2011.

In addition, during the July 14th shareholder meeting shareholders approved a proposal to change Marina's capital structure by increasing the number of authorized shares of common stock from 90,000,000 to 180,000,000. This removes the risk of the \$2.5M cash penalty payable to investors if the company had not been able to gain approval for the increase in approved shares.

On February 10, 2011, Marina Biotech entered into an underwriting agreement for the sale of 6,375,000 Units consisting of one share of its common stock and 0.1746 of a warrant to purchase one share of its common stock (1,113,075 shares of common stock) at a public offering price of \$0.80 per Unit. The Warrants will generally be exercisable for a period of seven years commencing on February 15, 2011, at an exercise price of \$0.80 per share and are also subject to adjustment if Marina engages in a "Fundamental Transaction". Marina received net proceeds of approximately \$4.7 million from the offering. Also in February 2011, the exercise price of warrants to purchase 686,260 shares which were issued in November 2010 was adjusted to \$1.06 per share and these warrants are no longer subject to further adjustment. Marina also issued warrants to purchase 1,113,075 shares of common stock at \$0.80 per share. The warrants are exercisable until February 15, 2018 and are not subject to repricings. As a result of the issuance of common stock and warrants at a price of \$0.80 per share, 687,500 warrants previously outstanding that were issued in June 2009 were repriced to \$0.80 per share. In addition, warrants assumed in the Cequent acquisition were adjusted from a price of \$1.75 per share to \$1.59 per share, and the number of shares purchasable under the warrants was adjusted from 10,556 shares to 11,627 shares.

As of September 30, 2011, there were 49,560,047 warrants outstanding with a weighted average exercise price of \$0.69. In addition, there were 2,046,596 stock options outstanding and exercisable with a weighted average exercise price of \$7.29. As of November 4, 2011, Marina Biotech had 91,369,157 shares outstanding.

Intellectual Property

Marina Biotech has intellectual property covering siRNAs, chemistry, delivery and targets that includes 67 issued or allowed patents, 43 U.S. patent applications, 135 foreign patent applications and 6 PCT applications.

SELECTED MARINA BIOTECH U.S. INTELLECTUAL PROPERTY FILINGS			
NUMBER	DESCRIPTION	FILED	ISSUED
7,939,505	Amino acid lipids and uses thereof	May 2, 2008	May 10, 2011
7,772,189	Phage Displayed Cell Binding Peptides	February 5, 2010	August 10, 2010
7,704,953	Phage Displayed Cell Binding Peptides	January 26, 2007	April 27, 2010
7,696,343	Method For Opening Tight Junctions	January 18, 2007	April 13, 2010
7,435,720	Compositions And Methods For Enhanced Mucosal Delivery Of Parathyroid Hormone	October 17, 2006	October 14, 2008
20100311655	Intranasal Carbetocin Formulations And Methods For The Treatment Of Autism	September 28, 2007	Pending
20100240731	Lipopeptides For Delivery Of Nucleic Acids	March 30, 2010	Pending
20100210506	Intranasal Administration Of Rapid Acting Insulin	October 20, 2006	Pending
20100112042	Processes And Compositions For Liposomal And Efficient Delivery Of Gene Silencing Therapeutics	October 16, 2009	Pending
20100184688	Compositions And Methods For Enhanced Mucosal Delivery Of Parathyroid Hormone	March 17, 2010	Pending
20100166811	Gras Composition For Intranasal Delivery Of Parathyroid Hormone	March 17, 2010	Pending
20100144843	Rnai Therapeutic For Respiratory Virus Infection	February 12, 2010	Pending
20100137225	Phage Displayed Cell Binding Peptides	February 5, 2010	Pending
20100112687	Nucleic Acid Compounds For Inhibiting Erbb Family Gene Expression And Uses Thereof	February 28, 2008	Pending
20100112042	Processes And Compositions For Liposomal And Efficient Delivery Of Gene Silencing Therapeutics	October 16, 2009	Pending
20100105134	Nucleic Acid Compounds For Inhibiting Gene Expression And Uses Thereof	September 1, 2009	Pending
20100056768	Hydroxymethyl Substituted Rna Oligonucleotides And Rna Complexes	May 21, 2008	Pending
20100055784	Nucleic Acid Compounds For Inhibiting Wnt Gene Expression And Uses Thereof	March 3, 2008	Pending
20100055783	Nucleic Acid Compounds For Inhibiting Ras Gene Expression And Uses Thereof	March 3, 2008	Pending
20100055782	Nucleic Acid Compounds For Inhibiting Myc Gene Expression And Uses Thereof	March 3, 2008	Pending
20100047909	Nucleic Acid Compounds For Inhibiting Vegf Family Gene Expression And Uses Thereof	February 28, 2008	Pending
20100041140	Nucleic Acid Compounds For Inhibiting Bcl2 Gene Expression And Uses Thereof	February 29, 2008	Pending
20100015708	Ribonucleic Acids With Non-Standard Bases And Uses Thereof	June 18, 2009	Pending
20100015706	Nucleic Acid Compounds For Inhibiting Hif1a Gene Expression And Uses Thereof	February 28, 2008	Pending
20090042823	Uses Of Broad Spectrum Rnai Therapeutics Against Influenza	August 11, 2008	Pending
20090042298	Compositions And Methods For Enhancing Delivery Of Nucleic Acids Into Cells And For Modifying Expression Of Target Genes In Cells	September 8, 2008	Pending
20090018097	Modification Of Double-Stranded Ribonucleic Acid Molecules	May 25, 2006	Pending
20100189691	E. Coli Mediated Gene Silencing Of Beta-Catenin	November 13, 2009	Pending

Source: U.S. Patent and Trademark Office

Partnerships

As a result of the emerging RNAi space and Marina Biotech's efforts to build multiple RNAi platforms and delivery technologies, the company has a significant number of licensing and development agreements covering a broad spectrum of activities as shown below:

Debiopharm: In February 2011, Marina entered into a Research and License Agreement with Switzerland-based Debiopharm S.A. which granted to Debiopharm an exclusive license to develop and commercialize Marina Biotech's pre-clinical program in bladder cancer, for all uses in humans and animals for the prevention and treatment of superficial (non-muscle invasive) bladder cancer. Debiopharm will pay up to \$25 million based on predefined research and development milestones, royalties from the sales of products resulting under the Agreement and sublicensing payments. The Agreement provides for certain licenses of Marina Biotech's UsiRNA and liposomal technologies. Debiopharm has

full responsibility for the development and commercialization of any products arising from the partnership, and will fund all research and development costs for the bladder cancer program.

Roche: In February 2009, Marina entered into an agreement with F. Hoffmann-La Roche Inc., a New Jersey corporation, and F. Hoffmann-La Roche Ltd., a Swiss corporation (collectively, Roche), pursuant to which Marina granted to Roche a worldwide, irrevocable, non-exclusive license to a portion of their technology platform, for the development of RNAi-based therapeutics, in consideration of the payment of a one-time, non-refundable licensing fee of \$5.0 million. No additional royalties are payable to Marina under the agreement. The agreement will expire on a country-by-country basis upon the expiration date of the last to expire of the licensed patents in such country. Either party may terminate the agreement for material breach by the other party (subject to a 30-day cure period), or upon certain events involving the bankruptcy or insolvency of the other party. Marina believes this agreement represents strong third-party validation of the siRNA construct aspect of their RNAi drug discovery platform.

Novartis: In March 2009, Marina entered into an agreement with Novartis Institutes for BioMedical Research, Inc. (Novartis), pursuant to which Marina granted to Novartis a worldwide, non-exclusive, irrevocable, perpetual, royalty-free, fully paid-up license, with the right to grant sublicenses, to the DiLA²-based siRNA delivery platform in consideration of a one-time, non-refundable fee of \$7.25 million, which was recognized as license fee revenue in the nine months ended September 30, 2009. Novartis may terminate this agreement immediately upon written notice to Marina. Additionally, Marina entered into a separate agreement with Novartis to provide them with an exclusive period in which to negotiate a potential research and development collaboration as well as possible broader licensing rights related to the RNAi drug delivery platform. This exclusive period expired in 2009. Approximately \$0.3 million was recognized as license fee revenue in 2009 under the separate agreement.

Novosom: In July 2010, Marina entered into an agreement pursuant to which they acquired the intellectual property of Novosom AG of Halle, Germany for Novosom's SMARTICLES[®] liposomal-based delivery system, which significantly broadens the number of approaches Marina may take for systemic and local delivery of their proprietary UsiRNA therapeutics. Marina issued an aggregate of 1,419,487 shares of their common stock to Novosom as consideration for the acquired assets. The shares had an aggregate value equal to approximately \$3.8 million, which was recorded as research and development expense. As additional consideration for the acquired assets, Marina will pay to Novosom an amount equal to 30% of the value of each upfront (or combined) payment actually received by Marina in respect of the license of liposomal-based delivery technology or related product or disposition of the liposomal-based delivery technology by Marina, up to a maximum of \$3.3 million, which amount will be paid in shares of common stock, or a combination of cash and shares of common stock, at Marina's discretion.

University of Michigan: In May 2008, Marina entered into an exclusive license agreement of Intellectual Property from the University of Michigan covering cationic peptides for enhanced delivery of nucleic acids. These peptides have unique characteristics that Marina believes play an important role in improving the efficacy of delivery of RNAi-based therapeutics. Marina is currently using these peptides to create siRNA nanoparticles to enhance mRNA knockdown. Together with the DiLA² technology, these delivery peptides may improve the therapeutic potential of the company's drug candidates. In connection with the agreement, Marina paid a license issue fee of \$120,000, which was paid in full in three equal installments. An additional fee of \$25,000 is payable annually and is creditable against royalty payments.

Subject to the meeting of certain milestones triggering the obligation to make any such payments, Marina may be obligated to make product development milestone payments of up to \$425,000 in the aggregate for each product developed under a licensed patent under this agreement. To date, Marina has not made, and is not under any current obligation to make, any such milestone payments. The royalty payment required to be made by Marina to the University of Michigan under this agreement is a percentage of net sales in the low single digits.

Marina sublicensed the IP under this agreement to Novartis on a nonexclusive basis in March 2009, at which time they paid an additional one-time fee of \$362,500 to the University of Michigan, which eliminated the obligation to pay the University of Michigan any future royalties or milestones with respect to the Novartis sublicense. This fee was included in research and development expense.

This agreement will terminate on the expiration date of the last to expire patent licensed under the agreement, which has an expiration date in 2019. Under the agreement, Marina agreed to use diligent and commercially reasonable efforts to

exploit the patent rights and bring licensed products to market. If Marina fails to meet certain research and development milestones, the University of Michigan may terminate the agreement subject to a thirty day cure period. In addition, the University of Michigan may terminate this license upon written notice if the first commercial sale of a product does not occur on or before May 2017. Marina may terminate the agreement at any time upon ninety days written notice.

University of Helsinki: In June 2008, Marina entered into a collaboration agreement with Dr. Pirjo Laakkonen and the Biomedicum Helsinki. The goal of the work involves Marina's patented phage display library, the Trp Cage library, for the identification of peptides to target particular tissues or organs for a given disease. In December 2009, Marina received a patent allowance in the US covering a targeting peptide for preferential delivery to lung tissues that was identified by using the Trp Cage Library. Marina believes the Trp Cage library will be a source of additional peptides for evaluation in delivery programs, and believes they will have a strong IP position for these peptides and their use. In 2010 Marina extended the term of the agreement and it will now terminate in June 2012. Either party may terminate the agreement for material breach by the other party, subject to a 30-day cure period.

Under this agreement, Marina may be obligated to make product development milestone payments of up to €275,000 in the aggregate for each product developed under this research agreement if certain milestones are met. To date, Marina has not made, and is not under any current obligation to make, any such milestone payments. In addition, upon the first commercial sale of a product, Marina is required to pay an advance of €250,000 against which future royalties will be credited. The percentage royalty payment required to be made by Marina to the University of Helsinki under the terms of this agreement is a percentage of gross revenues derived from work performed under the Helsinki Agreement in the low single digits.

Ribotask ApS.: In June 2009, Marina announced the revision of a October 2008 agreement in which they had acquired the intellectual property related to Unlocked Nucleobase Analogs (UNA) from Ribotask ApS, a privately held Danish company. The original agreement provided Marina with exclusive rights for the development and commercialization of therapeutics incorporating UNAs. The amended agreement eliminated Marina's obligation to pay all milestone and royalty payments and provided full financial and transactional control of the proprietary UNA technology.

In June 2010, Marina expanded the previous agreement with RiboTask to include exclusive rights to the development and commercialization of UNA-based diagnostics. In connection with this amendment, Marina agreed to pay Ribotask \$750,000 in three equal payments in each of October 2010, January 2011 and April 2011. There are no milestone or royalty payments due under the agreement.

Under the October 2008 agreement Marina made payments to Ribotask totaling \$500,000. Marina sublicensed the IP under this agreement to Roche on a nonexclusive basis in February 2009, at which time they paid an additional \$250,000 to Ribotask, which eliminated the obligation to pay Ribotask any future royalties or milestones with respect to the Roche sublicense. In connection with the June 2009 amendment, Marina issued 151,515 shares of common stock valued at approximately \$1.0 million to Ribotask ApS and agreed to pay \$1.0 million in four installments of \$250,000 each due at various intervals through July 2010.

In connection with their agreements, as amended, Marina granted Ribotask a royalty-bearing, world-wide exclusive license to use the assigned patents to develop and sell products intended solely for use as reagents or for testing. The royalty rates to be paid to Marina by Ribotask are in the low single digits and as of the end of September 2010, Marina has not recognized any revenue under this agreement, as amended.

University of British Columbia: In November 2009, Marina expanded and extended a previous agreement established in 2008 with University of British Columbia/Vancouver Prostate Centre (VPC) in the area of bladder cancer. The VPC is a National Centre for Excellence for translational research and this agreement provides Marina with access to cutting-edge bladder cancer models, evaluation techniques and interactions with world-renowned researchers and clinicians. Data derived from studies conducted under this agreement have demonstrated the potency of UsiRNAs and DiLA2 -based delivery for inhibition of target mRNA and reduction in tumor growth. The focus of the expanded agreement will be the evaluation of additional critical targets in bladder cancer and the therapeutic impact on tumor biology and growth. The research agreement requires that Marina make payments for work completed under an agreed work plan. Through September 30, 2010, Marina had recognized approximately \$0.2 million as research and development expense under this

agreement. The agreement may be terminated by either party with ninety days written notice. The current contract period terminated October 31, 2010 but may be further extended by mutual agreement between Marina and VPC.

Valeant Pharmaceuticals: In March 2010, Marina acquired intellectual property related to Conformationally Restricted Nucleotides (CRN) from Valeant Pharmaceuticals North America in consideration of payment of a non-refundable licensing fee of \$0.5 million due in equal portions in April and July 2010, which were included in research and development expense in the nine months ended September 30, 2010 and have been paid in full. Subject to meeting of certain milestones triggering the obligation to make any such payments, Marina may be obligated to make a product development milestone payment of \$5.0 million within 180 days of FDA approval of a New Drug Application for their first CRN related product and another product development milestone payment of \$2.0 million within 180 days of FDA approval of a New Drug Application covering their second CRN related product. To date, Marina has not made, and is not under any current obligation to make, any such milestone payments. Valeant is entitled to receive earn-outs in the low single digits based upon future commercial sales and earn-outs in the low double digits based upon future revenue from sublicensing. Under the agreement Marina is required to use commercially reasonable efforts to develop and commercialize at least one covered product. If Marina has not made earn-out payments of at least \$5.0 million dollars prior to the sixth anniversary of the date of the agreement, they are required to pay Valeant an annual amount equal to \$50,000 per assigned patent which shall be creditable against other payment obligations. The term of the financial obligations under the agreement shall end, on a country-by-country basis, when there no longer exists any Valid Claim in such country. Marina may terminate the agreement upon 30 days notice, or upon 10 days notice in the event of adverse results from clinical studies. Upon termination, Marina is obligated to make all payments accrued as of the effective date of such termination but shall have no future payment obligations.

ViThera Laboratories: ViThera Laboratories LLC has a sublicense to Marina Biotech's tkRNAi platform for certain non-human applications of tkRNAi including veterinary and agriculture fields.

Intranasal-Related

Cypress Bioscience, Inc.: In August 2010 Marina entered into an Asset Purchase Agreement with Cypress Bioscience, Inc. (Cypress) under which Cypress acquired their patent rights and technology related to carbetocin, a long-acting analog of oxytocin, a naturally produced hormone that may benefit individuals with autism. Under the agreement, Marina received an upfront payment of \$750,000 and could receive milestone payments up to \$27 million. Cypress will be responsible for all future development and IP related expenses. In addition, Cypress will pay Marina Biotech royalties, in the single digits, on commercial sales.

Amylin Pharmaceuticals, Inc.: In January 2009 Marina amended a 2006 License Agreement with Amylin Pharmaceuticals, Inc. for the development of intranasal exenatide. The License Agreement, as amended, provided for an accelerated \$1.0 million milestone payment to Marina in January 2009, a reduction in the aggregate amount of milestone payments that could be due to Marina from \$89 million to \$80 million, and a reduction in the royalty rate payable upon commercial sales of a product to the low single digits. Additionally, as a result of the amendment, Marina is no longer responsible for any further development of the nasal spray formulation of intranasal exenatide or its manufacture. Either party may terminate the agreement for breach of any material provision of the agreement upon sixty days notice of the breach and subject to a sixty day cure period. Amylin may also terminate the agreement upon ninety days written notice.

Par Pharmaceutical: In 2009 Marina entered into an Asset Purchase Agreement with Par Pharmaceutical pursuant to which, among other things, a 2004 License and Supply Agreement with Par, and a 2005 Supply Agreement with QOL Medical LLC, were terminated. Under the Asset Purchase Agreement, Par acquired certain assets pertaining to calcitonin, including Marina's ANDA for generic calcitonin-salmon nasal spray, inventories, tooling and equipment, and the related technology, trade secrets, know-how, proprietary information and other intellectual property rights, and assumed certain contracts, including Marina's manufacturing obligation to QOL Medical as well as their two building leases related to their operations in Hauppauge, New York. Marina received \$0.8 million in cash and is entitled to receive earn-out payments for five years based on commercial sales of calcitonin. Calcitonin received full FDA approval and was launched in June 2009. On December 22, 2010, Marina entered into an amendment of the Asset Purchase with Par Pharmaceutical, pursuant to which Marina will receive from Par a lump-sum cash payment in the amount of \$700,000 in lieu of profit sharing for five years on commercial sales of generic calcitonin-salmon nasal spray.

Other

QOL Medical LLC: In October 2005, Marina entered into a supply agreement with QOL under which, subject to certain limitations, Marina was obligated to manufacture and supply, and QOL was obligated to purchase from Marina, all of QOL's requirements for Nascobal® brand products for vitamin B12 (cyanocobalamin) deficiency in patients with pernicious anemia, Crohn's Disease, HIV/ AIDS and multiple sclerosis. Under the terms of the QOL Agreement Marina received a \$2.0 million upfront fee, which was being recognized ratably over the five-year life of the QOL Agreement. In connection with the Asset Purchase Agreement with Par Pharmaceutical which Marina entered into in March 2009, the QOL Agreement was terminated. Marina recognized approximately \$0.7 million in deferred revenue related to the Supply Agreement in the nine months ended September 30, 2009.

Collaborations

In addition to the partnerships mentioned above, Marina Biotech has also entered into multiple early collaborative efforts with major pharmaceutical companies. Marina currently has 6 collaborations with 5 companies to develop their therapeutic and drug delivery platforms. One of the six collaborations is with a RNA-based therapeutics company focused on microRNA directed oncology therapies. Two of the six collaborations have been disclosed:

AstraZeneca Investment China Company, Ltd.: This collaboration is focused on Marina Biotech's DiLA² delivery technology and UsiRNA constructs for systemic delivery in hepatocellular carcinoma (HCC)

Pfizer: This collaboration is focused on the evaluation of Marina Biotech's DiLA² delivery technology and UsiRNA constructs for RNAi therapeutics

Management

J. Michael French: Mr. French has served as Chief Executive Officer since June 23, 2008, as President since October 1, 2008, and as a member of the Board of Directors since September 11, 2008. Prior to joining, Mr. French served as President of Rosetta Genomics, Inc. from May 2007 to August 2007. Mr. French also served as Senior Vice President of Corporate Development for Sirna Therapeutics, Inc. from July 2005 to January 2007, when Sirna was acquired by Merck and Co., Inc., and he served in various executive positions, including Chief Business Officer, Senior Vice President of Business Development and Vice President of Strategic Alliances, of Entelos, Inc., a pre-IPO biotechnology company, from 2000 to 2005. Mr. French holds a B.S. in aerospace engineering from the U.S. Military Academy at West Point and a M.S. in physiology and biophysics from Georgetown University.

Philip C. Ranker: Currently Interim Chief Financial Officer, he most recently served as Chief Financial Officer of Suneva Medical, a start-up aesthetics company, as well as a member of the Board of Directors and Audit Committee Chair of ImaRx Therapeutics, which executed an Initial Public Offering during his tenure on the Board. Previously, Mr. Ranker served as Vice President of Finance at Amylin Pharmaceuticals, supporting the company through a significant restructuring which set Amylin on a path toward positive operating cashflow. Prior to Amylin, Mr. Ranker held the positions of Chief Financial Officer and VP of Finance at Nastech Pharmaceutical Company (predecessor to Marina Biotech) and Director of Finance for ICOS Corporation during the commercialization of Cialis. He also previously served in various positions in corporate accounting, managed care contracting and research and development, including Senior Finance Director, at Aventis Pharma and its predecessor companies during his nearly fifteen year tenure with the organization. Prior to Aventis, Mr. Ranker was employed by Peat Marwick (currently KPMG) as a Certified Public Accountant.

Richard T. Ho, M.D., Ph.D.: Dr. Ho serves as Executive Vice President, Research and Development. He previously served as Senior Medical Director at Entelos, Inc. from 2008 to 2011 where he oversaw academic and governmental collaborations including a Cooperative Research and Development Agreement with the FDA. From 2007 to 2008, he was a Principal at Rosa and Co. where he worked with pharmaceutical and biotechnology companies on physiological modeling efforts in several disease areas including metabolic disorders, respiratory disease, and bioterrorism agents. Dr. Ho began his industry career at Johnson & Johnson Pharmaceutical Research & Development starting as a fellow in Medical Informatics and advancing to the position of Director of Disease Modeling. Over a ten year period at J&J, he

built and led a team which championed systems biology and personalized medicine approaches to understanding and treating human disease. In this position, he coordinated model-based analysis and research with global clinical and preclinical teams developing both small and large molecule compounds. Before joining J&J, Dr. Ho completed a residency in Internal Medicine and a fellowship in Rheumatology at Yale School of Medicine. Dr. Ho received his M.D.-Ph.D. from the University at Buffalo School of Medicine with his thesis work at the Grace Cancer Drug Center of Roswell Park Cancer Institute and received his A.B. in physics from Harvard College.

Michael V. Templin, Ph.D., DABT: Dr. Templin serves as Senior Vice President and Chief Technology Officer. He joined Marina Biotech in December 2004. While at Marina Biotech (and its predecessors) he has served in a variety of management positions leading teams in the areas of discovery research and preclinical development. Before joining Marina Biotech he held research and development positions at Isis Pharmaceuticals, Amgen, and Zymogenetics. Dr. Templin's pharmaceutical development experience includes regulatory toxicology from bench research through IND and NDA filings for small- and large-molecule platforms (oligonucleotides, peptides, proteins, and antibodies). Dr. Templin received a Ph.D. in Pharmacology/Toxicology from Washington State University and completed a Postdoctoral Fellowship at the Chemical Industry Institute of Toxicology (RTP, NC). Dr. Templin has held certification as a Diplomate of the American Board of Toxicology since 1998.

Alan W. Dunton, M.D.: Dr. Dunton currently serves as consulting Chief Medical Officer and is the founder of Danerius, LLC. He has held significant senior positions in major pharmaceutical companies and has been directly responsible for, or has overseen the successful development/original/line extension approvals of Levaquin®, Regranex®, Aleve®, Procrit/EPREX®, Sporanox®, Reminyl® and Risperdal®. Most recently, he served as President and Chief Executive Officer of Panacos Pharmaceuticals, Inc. a company focused on treatment resistant HIV therapeutics. Previously, he was the President and CEO of Metaphore Pharmaceuticals, which focused on anti-inflammatory and analgesic products. Dr. Dunton was a senior executive in various capacities in the Pharmaceuticals Group of Johnson & Johnson including President and Managing Director of The Janssen Research Foundation. He also served as group vice president of global clinical research and development of Janssen as well as the R.W. Johnson Pharmaceutical Research Institute, also a Johnson & Johnson company. Dr. Dunton has also held positions in clinical research and development at Syntex Corporation, CIBA-GEIGY Corporation and Hoffmann La Roche Inc. Dr. Dunton holds an M.D. degree from New York University School of Medicine, where he completed his residency in internal medicine. He also was a Fellow in Clinical Pharmacology at the New York Hospital/Cornell University Medical Center.

June D. Ameen, RN, MBA: Ms. Ameen serves as Vice President of Corporate Development, she joined Marina Biotech in September 2009. Ms. Ameen has held progressively responsible positions in both public and private life sciences companies for the past 20 years. Prior to joining Marina Biotech, Ms. Ameen served first as Vice President, Alliance Management and then Vice President, Business Development and Alliances of Entelos, Inc., a privately held systems biology company based in Foster City, California. While there, she was responsible for expanding the business from early-adopter technology-based research partnerships to therapeutic focused co-development collaborations with major pharmaceutical and biotechnology companies. As Vice President, Business Development and Alliances, annual revenues increased from less than \$3 million to over \$21 million in two years. Prior to Entelos, Ms. Ameen was with PAREXEL International Corporation in several positions focused on client relations and alliance management. Ms. Ameen has an M.B.A. from Babson College and a B.S.N., magna cum laude, from Boston College.

Michael Houston, Ph.D.: Dr. Houston serves as Vice President of Chemistry and Formulations, he joined Marina Biotech in February 2004. While at Marina Biotech (and its predecessors), he led both the RNAi and peptide chemistry efforts including both research and development activities. In June of 2008, he assumed the role of head of Formulations and Chemistry at Marina Biotech. Prior to joining Marina Biotech, he held the position of Director of Chemistry at Cytovax Biotechnologies where he led the research and manufacturing efforts of the company's peptide-protein conjugate-based vaccine. Dr. Houston received his B.Sc. and Ph.D. in Chemistry from the University of Waterloo and completed a Postdoctoral Fellowship in Protein Engineering in the laboratory of Dr. Robert Hodges at the University of Alberta.

Alison D. Silva: Ms. Silva serves as Vice President of Drug Development, she was recently Vice President of Drug Development at Cequent Pharmaceuticals and joined Marina Biotech through its acquisition of Cequent Pharmaceuticals in July 2010. In August 2007, Ms. Silva joined Cequent where she was responsible for preclinical toxicology, manufacturing, regulatory affairs and clinical trial design and management. She successfully completed the

Investigational New Drug (IND) submission for Cequent's clinical candidate in Familial Adenomatous Polyposis in December 2009 - just nine months after the Food and Drug Administration (FDA) pre-IND meeting. Prior to joining Cequent, Ms. Silva was a Clinical Trials/Laboratory Manager at Pfizer where she was responsible for clinical-stage cardiovascular programs, dealing primarily with clinical trial management, investigator and site initiation, and regulatory and Institutional Review Board (IRB) submissions. In this position, she led a team of Clinical Research Associates, study monitors and study coordinators, to support Pfizer's therapeutic heads in clinical trial strategy, design, and execution. Prior to Pfizer, Ms. Silva was Laboratory Manager of an infertility department at Massachusetts General Hospital (MGH) and Co-Principal Investigator and Study Head at the Cardiac Catheterization Laboratory of University of Massachusetts – Worcester. Ms. Silva received her B.S. degree in Mathematics and Microbiology from Clark University and an M.S. degree in Cardiovascular Medicine from the University of Massachusetts Medical Center.

BOARD OF DIRECTORS

James M. Karis, Chairman: Mr. Karis has served on the Board of Directors since August 2009, and has been Chairman since July 2011. Mr. Karis has spent 30 years in the pharmaceutical, healthcare services and medical device industries and brings extensive corporate strategy, operations, M&A and financing experience to the company. Previously, Mr. Karis served as President, Chief Executive Officer and a Director of Entelos from January 2000 until May 2009. Prior to Entelos, he held senior positions in the contract research industry, serving as Chief Operating Officer and President of PAREXEL International Corporation, and earlier, as Chief Operating Officer of Pharmaco International. He was the Vice President of International Operations for Baxter International and a founder of KMR Group, a leading pharmaceutical R&D benchmarking consulting firm. Mr. Karis serves on the Board of BayBio, an advocacy group for Northern California's life science community. He has a B.S. in management and economics from Purdue University and a Masters in applied economics from The American University.

Peter Parker: Mr. Parker joined Marina Biotech's Board of Directors as part of Marina Biotech's acquisition of Cequent Pharmaceuticals in July 2010. He co-founded Cequent after two decades of very successful venture capital experience in healthcare investing. Prior to Cequent, Peter was a General Partner at Ampersand Ventures for over 15 years. While there, he was pivotal in raising 6 Ampersand funds, totaling over \$500MM in capital. He focused his investments in healthcare, serving as founding investor and a Director in several firms including: ACLARA Biosciences, AC Tech, Alexis, CoPharma, Cyclis Pharmaceuticals, Dynex, Huntington Laboratories, Magellan Biosciences, Nanodyne, NOVEX, Panacos Pharmaceuticals, Pentose Pharmaceuticals, Protein Ingredient Technologies, TekCel, and Tomah Products. After leaving Ampersand, Peter also started Boston Heart Lab, BioEssences, and StemSpan Solutions. Prior to Ampersand, Mr. Parker held various senior management positions at AMAX Inc. Mr. Parker holds a B.S. and an M.S. from Columbia University.

Gregory Sessler: Mr. Sessler has served on the Board of Directors since June 2008 and currently serves as Chair of the Audit Committee and as a member of the Compensation Committee and of the Nominating and Corporate Governance Committee of the Board of Directors. Mr. Sessler has served as the Chief Operating Officer since December 2008, and as the Executive Vice President and Chief Financial Officer since 2002, of Spiration, Inc., and is also currently a director and chairman of the audit committee of VLST, Corp. Prior to joining Spiration, Mr. Sessler served as Senior Vice President and CFO of Rosetta Inpharmatics, a leader in informational genomics, from March 2000 until its acquisition by Merck & Co., Inc. in July 2001 for \$540 million. Mr. Sessler is a member of the AICPA and FEI, and he previously served on the board of directors of Corixa Corporation. He also serves on the Executive Committee and is a past chairman of the board of directors of the Washington Biotechnology and Biomedical Association. Mr. Sessler holds a bachelors degree, magna cum laude, from Syracuse University and an M.B.A. from the Stanford Graduate School of Business.

J. Michael French: Chief Executive Officer of Marina Biotech (*see Management above*)

John Fletcher: Mr. Fletcher is a Founding Partner of Fletcher Spaght Ventures, a venture capital firm investing in emerging growth healthcare and high technology companies. He also serves as the CEO of Fletcher Spaght Inc., a management consulting firm he founded to provide strategy and financing assistance to new companies. Mr. Fletcher has over twenty-five years providing strategy and financing assistance to new companies. Prior to launching Fletcher Spaght Inc. in 1983, he served as a Senior Manager at The Boston Consulting Group, managing client relationships in healthcare and high technology companies. Mr. Fletcher attended the Wharton School of Business at the University of Pennsylvania and completed all coursework for a Ph.D. He holds an MA in International Finance from Central Michigan University, an

MBA from Southern Illinois University, and a BBA from George Washington University. He is a current or past board member for many companies, including Axcelis, GlycoFi and Spectranetics.

Michael Taylor, Ph.D.: Dr. Taylor joined Marina Biotech's Board of Directors as part of Marina Biotech's acquisition of Cequent Pharmaceuticals in July 2010. He has been President and CEO of Ensemble Therapeutics since July 2007 with more than 20 years experience in the pharmaceutical industry with extensive experience in drug discovery and development, licensing and business development, and managing R&D alliances with pharmaceutical and biotech partners. Prior to joining Ensemble, Dr. Taylor was Senior Vice President for Pfizer's Global R&D division responsible for global project and portfolio management. In other positions with Pfizer (and previously Warner-Lambert/Parke-Davis), he led early and late-stage development projects across multiple therapeutic areas, including Lipitor® and Neurontin®. He has authored or coauthored 65 manuscripts and published abstracts and holds 6 patents. Dr. Taylor earned a Ph.D. in Medicinal Chemistry from the State University of New York at Buffalo and was awarded an NIH postdoctoral fellowship in natural products synthesis and structure elucidation at the University of Pennsylvania.

SCIENTIFIC ADVISORY BOARD

Barry Polisky, Ph.D.: Dr. Polisky has served as Chief Scientific Officer since January 2, 2009. Previously, he served as a consultant to Merck from February 2008 to August 2008, and served as Research Vice President of Merck from January 2007 to January 2008. Dr. Polisky also served as Chief Scientific Officer and Senior Vice President of Sirna from March 2005 to January 2007, when Sirna was acquired by Merck, and served Sirna as Senior Vice President of Research from December 2003 to February 2005 and as Vice President of Research from June 2002 to December 2003. Prior to joining Sirna, Dr. Polisky served as Vice-President of Research at ThermoBiostar, Inc. from 1999 to 2002, where he developed a non-instrumented SNP diagnostic platform. Dr. Polisky, age 64, received his Ph.D. in molecular biology from the University of Colorado and conducted post-doctoral work in the Department of Biochemistry and Biophysics, University of California, San Francisco.

Beverly L. Davidson, Ph.D.: Dr. Davidson is currently Professor of Medicine, Neurology and Physiology & Biophysics in the Department of Internal Medicine at The University of Iowa. She holds the Roy J. Carver Biomedical Research Chair and is Director of The Davidson Laboratory, which is focused on inherited genetic diseases that cause central nervous system dysfunction, with a focus on:

- Recessive, childhood onset neurodegenerative disease, in particular the lysosomal storage diseases such as the mucopolysaccharidoses and Battens disease.
- Dominant genetic diseases for example the CAG repeat disorders, Huntington's disease and spinal cerebellar ataxia type I.
- Understanding how noncoding RNAs participate in neural development and neurodegenerative diseases processes.

Dr. Davidson is a member of the American Association for the Advancement of Science, American Federation for Clinical Research (Midwest Section), American Society for Neuroscience, American Society for Gene Therapy, and the American Society for Microbiology. Dr. Davidson serves on the Board of Directors for the American Society for Gene Therapy, and is Associate Director of the Center for Gene Therapy for Cystic Fibrosis and other Genetic Diseases, and is past Co-Director of the Iowa Biosciences Advantage Program. Dr. Davidson received her Ph.D. from the University of Michigan in 1987 and was a Fellow of the University of Michigan from 1990 to 1992.

Carl Novina, M.D., Ph.D.: Dr. Novina is an Assistant Professor in the Department of Pathology at Harvard Medical School, an Assistant Professor of Cancer Immunology/AIDS at Dana-Farber Cancer Institute and an Associate Member at the Broad Institute. His research focuses on investigating the mechanisms and applications of mammalian RNAi. To discover the biological roles of microRNAs and their interacting proteins, his group has developed cell-free, microRNA-dependent translational gene silencing reactions and cell-based reporter systems for translational repression and mRNA cleavage by microRNAs. His laboratory is engaged in collaborative projects to profile microRNA expression as well as microRNA and RNAi factor gene loci, in an effort to understand the roles of microRNAs in cancer, including hematopoietic and solid tumors. Dr. Novina received his M.D. from Columbia University, College of Physicians and Surgeons in 2000 and his Ph.D. from Tufts University, Sackler School of Graduate Biomedical Sciences in 1998. He did his graduate studies on transcriptional regulation of TATA-less promoters by TFII-I in Dr. Ananda Roy's laboratory. Dr.

Novina completed his postdoctoral training with Dr. Phillip Sharp, Nobel Laureate, at Massachusetts Institute of Technology investigating small RNA-directed gene silencing.

GLOSSARY

APC Gene: Adenomatous Polyposis Coli gene. A gene that codes for APC protein that is essential in the management of intracellular β -catenin levels. Mutations in this gene lead to elevated levels of β -catenin, causing polyps in the digestive tract often leading to colorectal cancer, in a condition called familial adenomatous polyposis (FAP).

CRN: Conformationally Restricted Nucleotides (CRN) are novel nucleoside analogs in which the ribose portion is locked into a rigid conformation (unlike UNA) by a small chemical linker.

Desmoid Tumor: Desmoid tumors are cytologically bland fibrous neoplasms originating from the musculoaponeurotic (Muscle and fibrous connective tissue) structures throughout the body. FAP can often cause these growths along the digestive tract with often lead to surgical procedures such as a colectomy.

Dicer: An enzyme which catalyzes the splicing of dsRNA into siRNAs.

dsRNA: Double-Stranded RNA. RNA macromolecule that consists of 2 strands covalently bound together in a similar fashion to DNA. dsRNA is known to activate RNAi.

FAP: Familial Adenomatous Polyposis. FAP is an inherited genetic condition in which numerous polyps to form in the epithelium of the digestive tract, primarily the large intestine. This condition is often caused by a mutation in the APC gene.

hnRNA: Heterogeneous Nuclear RNA. A single stranded RNA molecule that is the direct transcription of a defined region of DNA. It has to be processed by removing non-coding and adding a poly-A tail in order to become messenger RNA.

miRNA: Micro Interfering RNA. Small RNA molecules endogenous to an organism (encoded in their genomes) that are essential to control of gene expression through the activation of RNAi.

mRNA: Messenger RNA, A molecule of RNA encoding a chemical "blueprint" for a protein product. It is transcribed from a DNA template and carries coding information to the ribosomes for protein synthesis

RISC: RNA-Induced Silencing Complex. A multicomponent, ribonucleoprotein complex that cleaves specific mRNAs targeted for degradation by homologous dsRNAs during the process of RNAi. It includes siRNA that is generated from the specific dsRNA.

RNA: Ribonucleic Acid. RNA is a polymeric constituent of all living cells and many viruses, consisting of a long, usually single-stranded chain of alternating phosphate and ribose units with the bases adenine, guanine, cytosine, and uracil bonded to the ribose. RNA molecules are involved multiple cellular processes including protein synthesis and the transmission of genetic information.

RNAi: RNA Interference. RNAi is an intracellular mechanism that inhibits gene expression by causing the degradation of specific RNA molecules or hindering the transcription of specific genes, thereby stopping the production of target proteins within the cell.

shRNA: Short Hairpin RNA. A sequence of RNA that makes a tight hairpin turn that can be used to silence gene expression via RNA interference.

siRNA: Short interfering RNA. A short inhibitory double stranded RNA that activates RNAi through incorporation into the RISC complex.

tauRNAi: The tauRNAi platform incorporates Marina Biotech's UsiRNA construct technologies with their DiLA² liposome delivery system. By merging these two technologies, Marina Biotech can customize both the UsiRNA construct and the DiLA² liposome characteristics to target specific tissues and diseases resulting from overexpression of specific proteins.

tkRNAi: TransKingdom RNATM interference. Marina's drug platform using modified live, non-pathogenic, *E.coli* bacteria to orally deliver shRNA to cells of the intestinal tract.

UNA: Unlocked Nucleobase Analogs (UNA) are distinct entities, as the bond between two adjacent carbon atoms that form the ribose portion of RNA is absent and are considered to be conformationally flexible (unlike CRN)

UsiRNA: Duplex siRNAs that are modified with non-nucleotide acyclic monomers termed unlocked nucleobase analogs (UNA) and if appropriate, conformationally restricted nucleotides (CRN). UsiRNAs are designed to enter the RNAi pathway via Dicer enzyme or directly into RISC. UsiRNAs are fully recognized by the RNAi machinery and provide for RNAi activity.

Risks

Some of the operational and financial risks to Marina Biotech are:

- **Shareholder Dilution & Need to Raise Additional Funds:** Marina's latest offerings include the financing in May 2011 with the issuance of 22.3M shares in the base unit and 22.3M shares that were converted from the Series B unit warrants and up to an additional 44.6M shares that could potentially be issued from Series A warrants after 1 year at \$0.39. On July 14, 2011 Marina received shareholder approval to increase the number of authorized shares to 180M (from 90M) to fulfill this obligation. In addition Marina entered into a 30-month, \$15 million Purchase Agreement with a single institutional investor in October 2011 that could issue an additional 17,779,127 shares if fully exercised. We also believe the company will be required to raise additional funds through additional issuance of stock which would be dilutive to existing shareholders and could potentially affect the share price and have included our estimate of future share issuance in our financial model but there can be no guarantee that our estimates are accurate. (*see Recent Financing Activity for more details*)
- **Reverse-Split Likely:** With the overhang of the newly issued shares (and potentially future issued shares), we believe it unlikely that Marina will regain the minimum bid requirements putting the company at risk for delisting or a reverse-split which could put additional pressure on their shares.
- **FDA and Regulatory risks:** All of Marina Biotech's products are reliant on approvals by the U.S. FDA and other national regulatory bodies. There can be no guarantee of timely or definite FDA or other national regulatory body approvals for any of their products.
- **Partnerships:** Marina Biotech may be dependent on partners for future development, clinical trials and regulatory filings of its products and may be reliant on future partners to successfully market its products. Failure of Marina Biotech's existing or future partners to perform satisfactorily or in a timely fashion could adversely impact the company's financial position.
- **Long Time-Horizons:** Due to the relatively new nature of RNAi therapeutics, the development timelines may be significantly longer than typical drug development programs.
- **Patent Litigation:** Third-party claims of infringement of intellectual property could require Marina Biotech to spend time and money on defending their intellectual property rights up to and including adverse judgments against Marina Biotech. Although Marina Biotech is not currently involved, there is a significant ongoing patent dispute within the RNAi industry regarding the structure and use of RNAi therapeutics. (*see Tuschl I & II RNAi Patents*)

- **Sector Rotation:** Marina Biotech is a small biotechnology development company often kept in a portfolio with similar companies. In such cases, a significant event for one company may have a material impact on the valuation of all similar companies regardless of their unique qualities.

Marina Biotech																			
Consolidated Income Statement																			
FYE DEC 31st	1Q09	2Q09	3Q09	4Q09	2009	1Q10	2Q10	3Q10	4Q10	2010	1Q11	2Q11	3Q11	4Q11E	2011E	2012E	2013E	2014E	2015E
CEQ-508 FAP	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	35,000
License and Other Revenues	14,151	309	64	208	14,732	184	193	1,130	953	2,460	214	129	286	100	729	400	400	2,000	6,000
Total Revenues	14,151	309	64	208	14,732	184	193	1,130	953	2,460	214	129	286	100	729	400	400	2,000	41,000
Cost of Goods Sold	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	5,250
Gross Profit	14,151	309	64	208	14,732	184	193	1,130	953	2,460	214	129	286	100	729	400	400	2,000	35,750
Research & Development	4,116	4,340	3,319	3,107	14,882	3,599	3,837	7,500	3,169	18,105	3,350	2,772	2,955	3,398	12,475	13,723	14,409	14,409	15,129
Selling, General and Administrative	2,107	2,251	3,478	2,252	10,088	2,559	2,248	3,956	1,596	10,359	1,903	2,748	1,852	2,315	8,818	11,023	12,676	12,125	13,943
Restructuring [2]	133	178	118	26	455	26	260	460	2,780	3,526	228	58	1,104	175	1,565	0	0	0	0
Total Operating Expenses	6,356	6,769	6,915	5,385	25,425	6,184	6,345	11,916	7,545	31,990	5,481	5,578	5,911	5,888	22,858	24,745	27,085	26,534	29,073
Income from Operations	7,795	(6,460)	(6,851)	(5,177)	(10,693)	(6,000)	(6,152)	(10,786)	(6,592)	(29,530)	(5,267)	(5,449)	(5,625)	(5,788)	(22,129)	(24,345)	(26,685)	(24,534)	6,677
Interest Income	2	1	1	1	5	0	0	0	244	244	0	0	0	0	0	0	0	0	0
Interest and Other Expense	(143)	(168)	0	(227)	(538)	(780)	(544)	(321)	(1,162)	(2,807)	0	0	0	(200)	(200)	(800)	(800)	(400)	(200)
Change in Value for Adjustable Warrants	(1,027)	(905)	(160)	4,618	2,526	(2,710)	2,595	2,806	1,669	4,360	1,602	1,864	1,238	(1,000)	3,704	(1,500)	(1,500)	(2,000)	(2,500)
Gain on Settlement of Liabilities, net	654	0	0	0	654	0	0	29	(49)	(20)	0	0	0	0	0	0	0	0	0
Total Other Income/Expense	(514)	(1,072)	(159)	4,392	2,647	(3,490)	2,051	2,514	702	1,777	1,602	1,864	1,238	0	3,504	(2,300)	(2,300)	(2,400)	(2,700)
Income Before Tax	7,281	(7,532)	(7,010)	(785)	(8,046)	(9,490)	(4,101)	(8,272)	(5,890)	(27,753)	(3,665)	(3,585)	(4,387)	(5,788)	(18,625)	(26,645)	(28,985)	(26,934)	3,977
Provision for Income Taxes [1]	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Net Income (Loss)	7,281	(7,532)	(7,010)	(785)	(8,046)	(9,490)	(4,101)	(8,272)	(5,890)	(27,753)	(3,665)	(3,585)	(4,387)	(5,788)	(18,625)	(26,645)	(28,985)	(26,934)	3,977
EPS - Diluted	\$0.90	(\$0.84)	(\$0.69)	(\$0.08)	(\$0.86)	(\$0.80)	(\$0.34)	(\$0.39)	(\$0.23)	(\$1.58)	(\$0.12)	(\$0.08)	(\$0.05)	(\$0.05)	(\$0.27)	(\$0.14)	(\$0.13)	(\$0.10)	\$0.01
Shares Outstanding - Diluted	8,061	8,991	10,178	10,328	9,364	11,832	12,219	20,982	25,262	17,574	31,090	45,395	80,405	116,596	68,372	183,915	220,698	264,838	291,322

Balance Sheets

(in \$Millions)

	12/31/09	12/31/10	9/30/11
Assets:			
Cash and Cash Equivalents	\$748	\$1,066	\$1,044
Restricted Cash	998	1,017	1,157
Accounts Receivable	211	59	10
Inventories	0	0	0
Prepaid Expenses & Other	700	818	455
Total Current Assets	\$2,657	\$2,960	\$2,666
Property and Equipment, Net	4,569	3,695	2,690
Intangible Assets	0	22,734	22,734
Other Assets	3	54	45
TOTAL ASSETS	\$7,229	\$29,443	\$28,135
Liabilities:			
Accounts Payable	\$2,114	\$3,922	\$2,197
Liability for Refunds of Warrant Exercise Proceeds	0	0	0
Accrued Payroll and Employee Benefits	913	781	1,075
Other Accrued Liabilities	1,361	1,225	496
Notes Payable, Net of Discount	317	0	0
Deferred Revenue	0	34	514
Accrued Restructuring- ST	425	312	0
Total Current Liabilities	\$5,130	\$6,274	\$4,282
Accrued Restructuring- LT	\$281	\$148	\$0
Deferred Rent and Other Liabilities	1,461	1,384	1,281
Change in Value for Adjustable Units	0	1,483	10
Change in Value for Adjustable Warrants	7,243	1,783	5,485
Deferred Income Taxes	0	1,202	1,202
Stockholders' Equity	(6,886)	17,169	15,875
TOTAL LIAB. & EQ	\$7,229	\$29,443	\$28,135

NOTES

[1] As of December 31, 2010, Marina Biotech had federal net operating loss carryforwards of \$288.2M

[2] 4Q10 Restructuring Charge for Facility Discontinuation

DISCLOSURES



Ratings and Price Target Changes over Past 3 Years

Initiated January 28, 2011 – Strong Speculative Buy - Price Target \$2.50
 Downgrade May 18, 2011 – Neutral - Price Target \$0.30
 Downgrade December 8, 2011 – Sell - Price Target \$0.10

Analyst Certification: We, Stephen M. Dunn and William D. Dawson, the authors of this research report certify that a.) All of the views expressed in this report accurately reflect our personal views about any and all of the subject securities or issuers discussed b.) No part of our compensation is directly or indirectly related to the specific recommendations or views expressed in this research report and c.) We may be eligible to receive other compensation based upon various factors, including total revenues of the Firm and its affiliates as well as a portion of the proceeds from a broad pool of investment vehicles consisting of components of the compensation generated by investment banking activities, including but not limited to shares of stock and/or warrants, which may or may not include the securities referenced in this report.

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Does the Analyst or any member of the Analyst's household have a financial interest in any securities of the Company?	NO
Does the Analyst or any member of the Analyst's household or Firm serve as an officer, director or advisory board member of the Company?	NO
Has the Analyst or any member of the Analyst's household received compensation directly or indirectly from the Company in the previous 12 months?	NO
Does the Firm or affiliates beneficially own ≥1% of the Company's common stock?	NO
Has the Firm or affiliates received investment banking services compensation in previous 12 months?	NO
Has the Firm or affiliates received non-investment banking securities-related services compensation in previous 12 months?	NO
Does the Firm or affiliates expect to receive or intend to seek investment banking compensation in next 3 months?	YES
Has the Firm or affiliates received non-securities services compensation in previous 12 months?	NO
Does the Firm or affiliates make a market in the Company's securities?	NO

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Ratings Definitions: 1) **Strong Buy:** the stock is expected to appreciate and produce a total return of at least 40% over the next 12-18 months; 2) **Buy:** the stock is expected to appreciate and produce a total return of at least 20% over the next 12-18 months; 3) **Strong Speculative Buy:** the stock is expected to appreciate and produce a total return of at least 40% over the next 12-18 months but **the volatility and investment risk is substantially higher** than our "Strong Buy" recommendation; 4) **Speculative Buy:** the stock is expected to appreciate and produce a total return of at least 20% over the next 12-18 months but **the volatility and investment risk is substantially higher** than our "Buy" recommendation; 5) **Neutral:** the stock is fairly valued for the next 12-18 months; 6) **Avoid/Sell:** the stock is expected to decline at least 20% over the next 12-18 months and should be avoided or sold if held; 7) **Under Review:** the previous rating and/or price target is suspended due to a significant event which now requires additional analysis and the previous rating and/or price target cannot be relied upon; 8) **Not Rated:** the stock has too much business or financial uncertainty to form an investment conclusion or is currently in the process of being acquired and 9) **Restricted:** coverage cannot be initiated or has been temporarily suspended to comply with applicable regulations and/or firm policies in certain circumstances such as investment banking or an advisory capacity involving the company.

LifeTech Capital Research	Research Coverage	Investment Banking	FINRA RULE 2711	Research Coverage	Investment Banking
Ratings Distribution	% of Total	% of Total	Ratings Distribution	% of Total	% of Total
Strong Buy	17%	0%	Buy	75%	33%
Strong Speculative Buy	58%	43%	Hold/Neutral	8%	100%
Buy	0%	0%	Sell	17%	0%
Speculative Buy	0%	0%	Total	100%	33%
Neutral	8%	100%			
Avoid/Sell	17%	0%			
Under Review	0%	0%			
Not Rated	0%	0%			
Restricted	0%	0%			
Total	100%	33%			

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