UPDATE REPORT

Biotechnology Industry ● April 5, 2011

RXi Pharmaceuticals Corp. (NasdaqCM: RXII)

- **RXi becomes a clinical-stage company via its Apthera acquisition and gains a path into oncology with the immunotherapy NeuVax.**
- **NeuVax has superb safety and efficacy profiles – 36 month follow-up data due soon.**
- **RXi selects its molecular target for an anti-scarring drug – one with great potential.**
- **We reiterate our BUY recommendation and set a 12-month target price of $4.00 per share.**

RXi Pharmaceuticals (NasdaqCM: RXII) is a leader in a field of regulatory biochemistry involving small-inhibitory RNA that controls gene expression at the level of protein synthesis. The latest development, the proposed acquisition of privately owned Apthera, reflects a desire to add a late clinical stage therapy to the R&D pipeline. The newcomer's lead candidate, dubbed NeuVax, is an immunotherapy that will enter a Phase 3 clinical trial in 2012 to test its efficacy against node-positive breast cancer.

NeuVax is a vaccine comprised of the E75 peptide, which is an epitope of the cancer marker HER2/neu, and the adjuvant granulocyte macrophage colony stimulating factor, or GM-CSF. The vaccine has a very favorable side-effect profile, and it provided significant protection against disease recurrence in patients with node-positive breast cancer that express HER2 at low to moderate levels. These patients are not candidates for **Roche's** blockbuster drug Herceptin®. NeuVax is designed for a patient population in need of an improved therapy.

RXi has identified its lead drug candidate, RXI-109, for preventing scarring. The molecular target is connective tissue growth factor (CTGF), which plays a central role in wound healing and pathological fibrotic conditions, including hypertrophic scars, liver and pulmonary fibrosis, ocular scarring, and restenosis. RXi plans to file an IND later this year and initiate a Phase 1 clinical study in 2012, testing it initially as a prophylactic agent for hypertrophic scarring associated with surgery.

Griffin Securities will host an interview with RXI's new CEO, Dr. Mark Ahn, and with Dr. George Peoples, an expert in cancer vaccines at the Walter Reed Army Medical Center. A recording will be available on RXi's website as of April 11th.

Share Price (4/1/11)	$1.47
52-Week Price Low / High	$1.10 - $5.23
Mkt. Capitalization (issued)	$27.0 million
Shares Outstanding (issued)	18.4 million
12-month Target Price	$4.00
Website	www.rxipharma.com

Source: BigCharts.com

The acquisition of Apthera and development of RXI-109 justify a higher valuation for RXi than its current share price – new data and clinical progress to drive the Company's valuation. We are maintaining our BUY recommendation and setting a 12-month target price of $4.00 per share.

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NEW MANAGEMENT WITH A FOCUS ON DEVELOPMENT

RXi has a new Chief Executive Officer, and the recently announced acquisition of Apthera adds another, a Chief Operating Officer to the management team.

MANAGEMENT

Mark Ahn, Ph.D. – Chief Executive Officer and Director

- Joined RXi's board of directors in 2010 and assumed his current position in 2011.
- Has more than 20 years of experience in the biopharmaceutical industry, as a consultant, founder and Chief Executive Officer of Hana Biosciences, and executive with Genentech, Amgen, and Bristol-Myers Squibb.
- Serves on the board of directors of Access Pharmaceuticals and Mesyntheses and on the scientific advisory board of Zygem.

Mark W.Schwartz, Ph.D. – Executive Vice President and Chief Operating Officer

- Joined Apthera as the Chief Executive Officer in January 2010 with more than 25 years of leadership experience in building companies in the pharmaceutical and life sciences industries
- Served as the CEO of Bayhill Therapeutics and Calyx Therapeutics, Chief Commercial Officer of Trega Biosciences, and various managerial roles with Argonaut Technologies, Synteni/Incyte Genomics and Tripos

Anastasia Khvorova, Ph.D. – Chief Scientific Officer

- Joined RXi in 2008 with extensive experience in RNAi and microRNA obtained during her tenure with Dharmacon (ThermoFisher Scientific) and in drug development with Abbott and Alcon
- Has more than 30 patents and 40 publications

Ramani Varanasi, MS, MBA, Vice President of Business Development

- Joined RXi in 2009 with more than 13 years of experience in business development and partnering
- Served managerial positions in business development with Millennium Pharmaceuticals, Momenta Pharmaceuticals, Archemix Corporation, and Merck Serono

Pamela Pavco, Ph.D., VP of Pharmaceutical Development

- Brought Sirna's lead RNAi candidate to Phase I in under 12 months and managed three additional RNAi drug candidates through IND
- Managed Sirna's Allergan and Huntington's Disease collaborations

Dmitry Samarsky, Ph.D., VP of Technology Development

- Joined RXi in 2007 with more than 10 years of experience in the development and licensing of RNAi technology
- Served in various managerial positions with Dharmacon, Invitrogen, and Sequitur

SCIENTIFIC ADVISORY BOARD

Craig Mello, Ph.D., Founder and SAB Chairman

- 2006 Nobel Prize in Medicine for RNAi
- Co-discovered RNAi and invented RNAi therapeutics
- Howard Hughes Medical Institute Investigator at University of Massachusetts Medical School

Victor R. Ambros, Ph.D.

- Discovered the first micro-RNAs, which are the endogenous targeting compounds of the RNAi pathway
- Awarded the Gairdner International Award, also known as the "Canadian Nobel"
- Awarded the Benjamin Franklin Medal in Life Sciences, the Genetics Society of America Medal, and Brandeis University's Lewis S. Rosenstiel Award
- Holds the position of Professor of Molecular Medicine at the University of Massachusetts Medical School

Michael Czech, Ph.D.

- Professor and Chair, Program in Molecular Medicine, University of Massachusetts Medical School
- Recipient of American Diabetes Association's Eli Lilly Award for Diabetes and the Banting Award for scientific achievement

Greg Hannon, Ph.D., Founder and SAB member

- Howard Hughes Medical Institute Investigator at Cold Spring Harbor Laboratory
- Discovered mechanism of RNAi in human cells and developed the widely used short hairpin RNA

Nassim Usman, Ph.D.

- Served as Chief Scientific Officer and Chief Operating Officer at Sirna
- Negotiated alliances between Sirna and Eli Lilly, Allergan, and GlaxoSmithKline
- Has 130 patents and patent applications related to basic RNAi synthesis chemistry

Todd Wolf, Ph.D., Founder and SAB member

- Served as CEO of RXi Pharmaceuticals at its inception and Sequitur
- Co-invented Stealth RNAi and other RNA technologies, and has 40 patents and publications

Near-Term Milestones

H1, '11	Manufacture RXI-109 for IND-enabling studies and Phase 1 clinical trial
Q2, '11	Present NeuVax 36-month follow-up data at the annual meeting of ASCO
H2, '11	Conduct IND-enabling toxicology studies for RXI-109
H2, '11	File IND for RXI-109 for dermal scarring related to surgery
2011	Make presentations on self-delivering RNAi technology at various scientific meetings
2011	Manufacture NeuVax in preparation for Phase 3 clinical trial
H1, '12	Initiate Phase 1 clinical trial of RXI-109
H1, '12	Initiate Phase 3 clinical trial of NeuVax
H2, '12	Evaluate RXI-109 Phase 1 data

APTHERA – A SOUND STRATEGIC INVESTMENT

On March 31st, RXi announced that it has offered to acquire Apthera, a privately owned company with an immunotherapy for preventing the recurrence of breast cancer, for 4.8 million shares of stock or $7.1 million based on the recent stock price, plus earn-out payments. The deal, which already has the backing of investors with more than 60% of the shares outstanding, is expected to close by the end of April.

The acquisition will diversify RXi's R&D pipeline into oncology with a ready-to-use cancer vaccine that is scheduled to enter a Phase 3 clinical trial in the first half of 2012. In addition, Apthera will give the Company an executive with experience in building biotechnology companies and facilitating drug development through clinical trials. This is a plus, since RXi's first drug, RXI-109, is being readied for a Phase 1 study that will commence early next year. The newcomer will have little impact on the Company's balance sheet, aside from adding intangible assets and boosting stockholders' equity.

NEUVAX – AN IMMUNOTHERAPY FOR MULTIPLE CANCERS

NeuVax is a peptide-based vaccine that stimulates a cancer patient's immune system to recognize cells expressing the receptor for human epidermal growth factor receptor 2, or HER2/neu. This cell membrane bound receptor-tyrosine kinase plays a role in signal transduction that supports cell growth and differentiation. It is amplified by as much as 100 fold in a wide variety of cancers, including Wilm's tumor, and bladder, pancreatic, breast, prostate, salivary gland, ovarian, endometrial, and non-small-cell lung cancer.[1,2,3] But HER2/neu is probably best understood, because of research into breast cancer, where it is found in more than 75% of all cases and 25%-30% of the invasive malignancies.[4,5] This receptor-enzyme complex is the target of **Roche/Genentech's** highly successful, antibody-based drug Herceptin (trastuzumab). Yet, that drug is effective in only 50% of patients and it is considered most appropriate for cancers that greatly overexpress HER2/neu.[3,6] Thus, there is a need for an improved therapy for cancers expressing HER2/neu, particularly at low to moderate levels.

NEUVAX'S IMPRESSIVE CLINICAL DATA

NeuVax is designed to fill a gap in the treatment regimen for HER2/neu cancers that is not addressed by Herceptin. The immunotherapy, which is being developed initially for breast cancer, is composed of a nine amino acid segment or epitope of an extracellular domain of HER2/neu, dubbed E75, and granulocyte macrophage colony-stimulating factor, or GM-CSF.

A large Phase 1/2 clinical trial tested a dosing regimen involving intradermal administration of the vaccine monthly for six months before booster inoculations were given once every six months. The Phase 1 portion of the trial was a dose escalation study that was also used to assess safety, while the Phase 2 portion was designed to optimize dosing and patient selection and to generate efficacy data. All Phase 1 patients transitioned into the Phase 2 study. Inclusion criteria for entry into the study included HLA-A2 or A3 haplotypes, any level of HER2/neu (judged via immunohistochemistry analysis as IHC 1+, 2+, or 3+) and node positive or high-risk node negative breast cancer. All patients had to complete standard surgery, chemotherapy, and/or radiation within six months of entry into the trial, and they had to be clinically free of disease and immunocompetent. Overall, 187 patients were enrolled.

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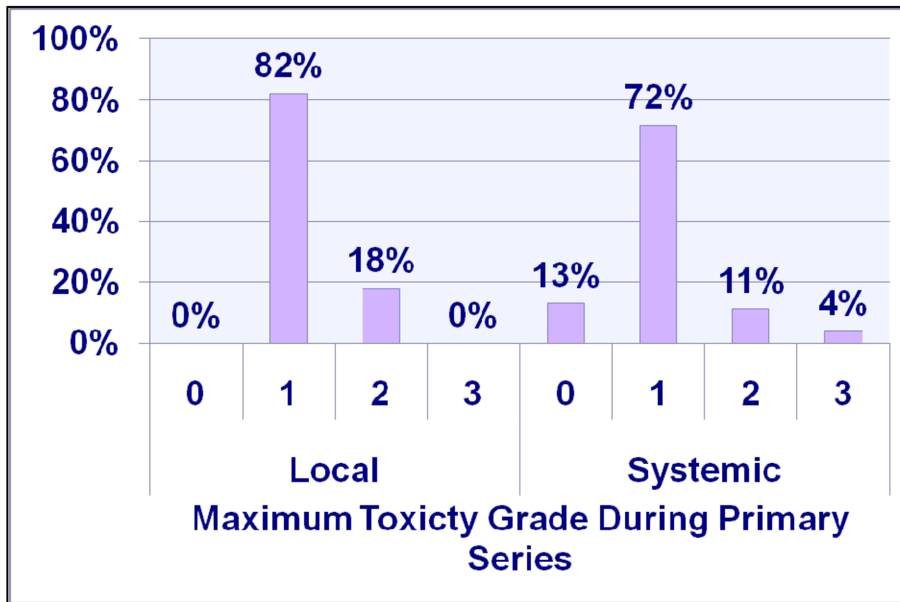
[1] Scholl, S, et al. Targeting HER2 in other tumor types. Ann Oncol (2000); 12 (suppl 1): S81.
[2] Menard, S, et al. HER2 overexpression in various tumor types, focusing on its relationship to the development of invasive breast cancer. Ann Oncol (2000); 12 (suppl 1): S15.
[3] Lohrisch, C and Piccart, M. HER2/neu as a predictive factor in breast cancer. Clin Breast Cancer (2001); 2(2): 129.
[4] Slamon, DJ, et al. Human breast cancer: correlation of relapse and survival with amplification of the HER2/neu oncogene. Science (1987); 235(4785): 177.
[5] Benavides, LC, et al. The impact of HER2/neu expression level on response to E75 vaccine: from U.S. Military Cancer Institute clinical trials group study I-01 and I-02. Clin Cancer Res (2009); 15(8): 2895.
[6] de Alava, E, et al. Neuregulin expression modulates clinical response to trastuzumab in patients with metastatic breast cancer. J Clin Oncol (2007); 25(19): 2656.

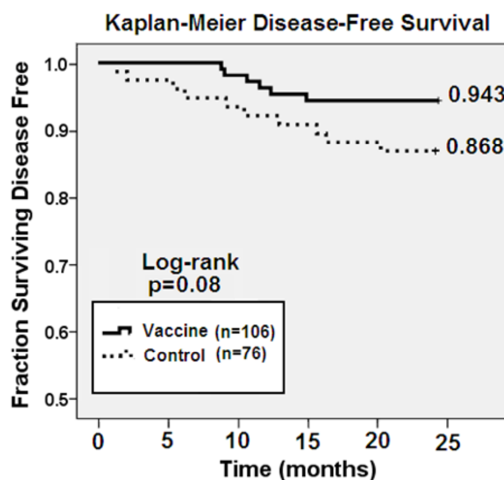
NeuVax has an excellent safety profile. As shown in Figure 1, adverse events reported during the clinical trial were low grade – no serious toxicities were reported.[7] This is consistent with the safety profiles of other peptide-based immunotherapies.

Figure 1. NeuVax's Excellent Toxicity Profile[6]



NeuVax is highly effective in preventing cancer recurrence, as measured by the number of evaluable patients who were disease free up to 24 months after treatment. This is illustrated in Figure 2, a Kaplan-Meier plot. Moreover, an examination of the immune response to E75 found evidence of "epitope spreading," meaning that the patients' immune systems were recognizing more portions of HER2/neu than just the nine amino acids in E75.[8] This provided evidence of a robust response to the vaccine.

Figure 2. NeuVax Prevents Disease Recurrence – All Patients[6]



[7] Clifton, GG. Clinical Efficacy of the E75 Peptide Vaccine: Cumulative Findings of Phase I/II Trials. Presented at the Breast Cancer Symposium, October 1-3, 2010.
[8] Mittendorf, EA, et al. Vaccination with a HER2/neu peptide induces intra-and inter-antigenic epitope spreading in patients with early stage breast cancer. Surgery (2006); 139: 407.

The only significant epidemiological difference between the vaccine-treated and control patient populations was that a larger proportion in the former group received Herceptin than in the latter (11.3% versus 2.7%). Other characteristics, including the proportion with node positive disease, tumor size, proportion with histological grade 3 disease, and use of therapies (hormonal, chemotherapy, and radiation therapy), were very similar between the groups.

These similarities made it possible to determine whether certain characteristics of the tumors could be used to identify a subset of patients who might benefit more than others from NeuVax. Node positive patients whose tumors were IHC 1+ or IHC 2+ for HER2/neu showed greater benefit from the immunotherapy. As shown in Figure 3, patients who received the optimal dose of E75 peptide vaccine, booster injections, and had node positive tumors and low- to moderate-expression of HER2/neu benefited the most. Thus, NeuVax significantly extended the period of disease-free survival of patients for whom Herceptin is inappropriate.

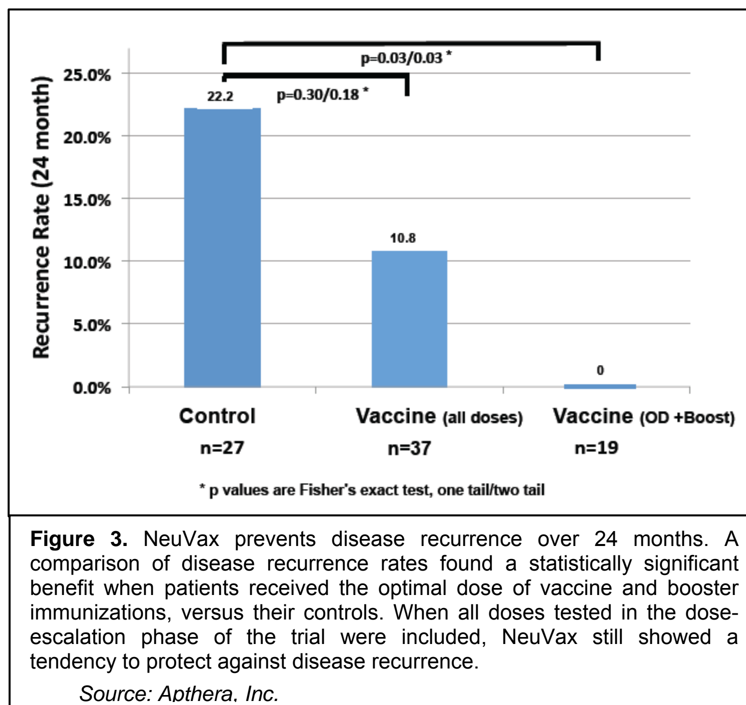


Figure 3. NeuVax prevents disease recurrence over 24 months. A comparison of disease recurrence rates found a statistically significant benefit when patients received the optimal dose of vaccine and booster immunizations, versus their controls. When all doses tested in the dose-escalation phase of the trial were included, NeuVax still showed a tendency to protect against disease recurrence.

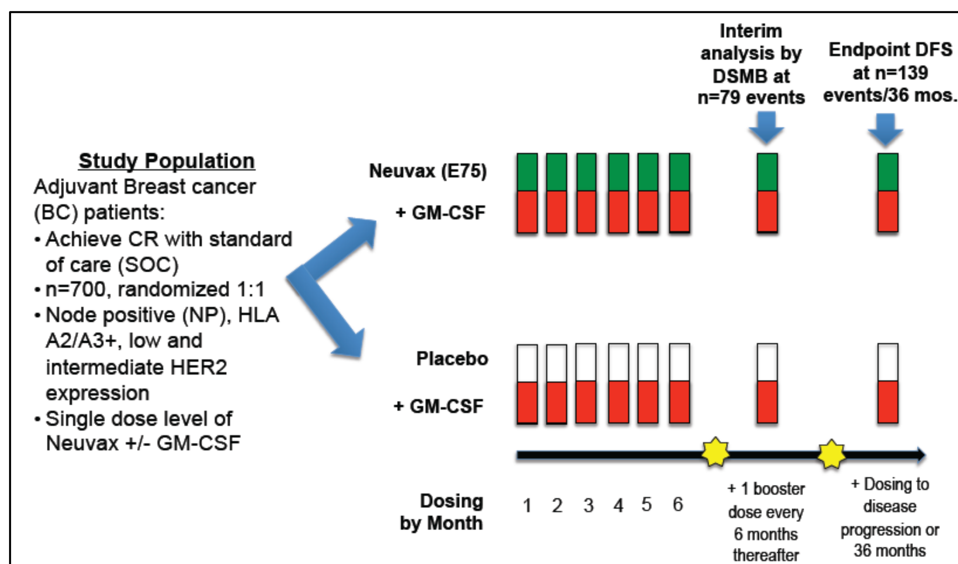
Source: Apthera, Inc.

The patients who participated in the Phase 1/2 study continue to receive booster immunizations every six months, and their health will be monitored for up to 60 months post-treatment. Results will be tabulated periodically. The next report, providing disease-free progression data at 36 months post-treatment, will be presented in conjunction with the annual meeting of the American Society of Clinical Oncology (ASCO) meeting that will convene on June 3-7.

The Phase 3 Clinical Trial

Apthera negotiated a Special Protocol Assessment with the FDA to establish the criteria that would be necessary to gain regulatory approval from a Phase 3 study. The outcome was that a 30% decrease in disease recurrence would be sufficient to merit approval. The trial will enroll 700 patients with HER2/neu IHC 1+ or 2+, node positive tumors in patients with HLA-A2 or HLA-A3 haplotypes (genetic traits associated with people from North America, Europe, and northern Asia). At this juncture, 14 medical centers have agreed to participate, though more will likely be added before enrollment commences, which is scheduled for the first half of 2012. An interim analysis will be conducted when 70 events (disease recurrences) have been reported, though the trial will continue until 139 events have occurred or patients have been treated for 36 months, whichever comes first. The study design is illustrated in Figure 4.

Figure 4. The Phase 3 Study Design



Source: Apthera, Inc.

Secondary objectives of the study will be to monitor for 3-year overall survival, 5- and 10-year disease-free and overall survival, time to recurrence (local, distant, and bone metastases), and overall safety profile and adverse events.

Financial Considerations

Phase 3 Trial Cost: NeuVax is not very expensive to manufacture, partly because the E75 peptide is a small, nine-amino acid peptide. Apthera engaged a contract manufacturer to prepare sufficient vaccine for the Phase 3 trial, and it should be ready for the start the trial in the first half of next year. The study will probably cost close to $30 million, with about $10 million expected to be incurred up to the 70-event interim analysis.

Marketing: RXi has no interest in outlicensing the vaccine at this juncture, but we believe a deal may be considered once the interim data are available. At that point, 50% of the total events will have been documented, and if the protection afforded by NeuVax is even close to that reported from the Phase 1/2 trial, we believe its commercial success would be assured. That probably would set the stage for a valuable licensing agreement, which could relieve the Company of at least some of the trial's remaining cost (roughly $20 million). Note that the oncology market is sufficiently small that RXi could sell NeuVax in the United States and outlicense it in foreign countries. Our financial analysis, though, is based on an assumption that the Company sells the vaccine domestically without consideration for overseas markets.

Patient Population: The patients who will be treated in the Phase 3 clinical trial will define the vaccine's initial commercial use. That population is a fraction of the invasive breast cancer market, but it is not insignificant in size. Approximately 192,000 cases of invasive breast cancer are diagnosed in the United States annually. Of these, 35% (or 67,000) are node positive, and within that group, about 57% (or 38,000) express HER2 at low to moderate levels. Of these patients, 65%-75% (or 25,000 – 28,500) have haplotypes HLA-A2 or HLA-A3. Our financial analysis is based on 25,000 cases per year being eligible for treatment. However, it is not unreasonable to think that many more patients will be treated, notably all haplotypes and perhaps even some node-negative individuals. This does not take into consideration the millions of patients with HER2/neu expressing tumors of other organs who fit the general criteria, except for the location within the body. As a result, we believe our financial analysis is somewhat conservative, but rightly so, at least until we know more about NeuVax's effectiveness in preventing disease progression.

Pricing NeuVax: Another important consideration in our financial analysis is the estimated cost of NeuVax. Since it is unknown how long patients will require the vaccine to be considered disease free and how much the vaccine will extend their lives, this estimate is tentative. An additional consideration is the nature of the therapy – it is an off-the-shelf vaccine, but one that will extend the lives of potentially large numbers of patients. On the one hand, NeuVax is similar to such drugs as **Celgene's** Revlimid® (lenalidomide) and some HIV therapies that command prices in the range of $26,000 - $100,000 per quality adjusted life year.[9,10] On the other, it is not that different than such common vaccines as **Merck's** Gardasil® (HPV) and Zostavax® (zoster), and **sanofi-aventis'** Menactra® (meningococcal) that cost under $200 per dose.[11] We settled on a price of $4,000 per NeuVax dose, which yielded a cost of $28,000 in the first year, a level well within the acceptable range of many healthcare payers. It also prices maintenance booster immunizations at $8,000 per year. Given the concerns over healthcare expenses and cost-cutting strategies, it seems prudent to assume NeuVax would be priced to maximize its acceptance and avoid unwanted scrutiny during budget cuts.

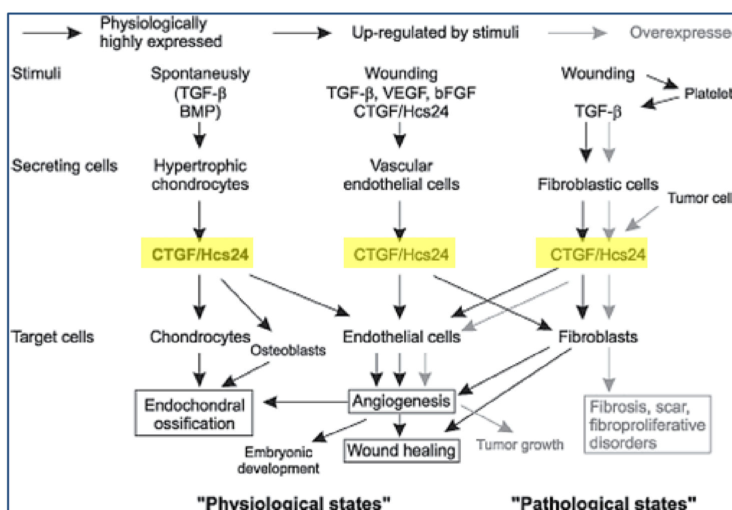
A Therapy for Fibrotic Diseases Advances

The Company's RNAi R&D efforts are beginning to pay off with a self-delivering rxRNA (sd-rxRNA) that targets a biological pathway with a wide range of potential prophylactic/therapeutic applications. Just as important, RXi has made a smart decision with its initial clinical development strategy, in our opinion.

RXi's Molecular Target

The Company has chosen a molecular target with a central role in fibrosis, Connective Tissue Growth Factor (CTGF), a signaling protein produced by a wide variety of cells including endothelial cells, smooth muscle cells, and fibroblasts. During development, this cytokine helps to regulate the formation of connective tissues, bone, and blood vessels. Later, expression of this 349-amino acid protein is increased by such stimuli as shear stress, hypoxia, and biomechanical deformation, and as such, it plays a key role in the process of normal wound healing. (See Figure 5.) However, it also participates in pathological scar formation when cell proliferation, cell adhesion, and extracellular matrix formation go awry.

Figure 5. CTGF's Central Role in Wound Healing and Scar Formation



Source: Takigawa, M.[12]

[9] Messori, A, et al. The role of bortezomib, thalidomide, and lenalidomide in the management of multiple myeloma. Pharmacoeconomics (2011); 29(4): 269.

[10] Broder, MS, et al. Cost effectiveness of atazanavir-ritonavir versus lopinavir-ritonavir in treatment-naive human immunodeficiency virus-infected patients in the United States. J Med Econ (2011); 14(2):167.

[11] CDC Vaccine Price List as of March 31, 2011, accessed on April 3, 2011 at http://www.cdc.gov/vaccines/programs/vfc/cdc-vac-price-list.htm

[12] Excerpted by Google Images from: Takigawa, M. CTGF/Hcs24 as a multifunctional growth factor for fibroblasts, chondrocytes and vascular endothelial cells. Drug News Perspect (2003); 16(1): 11.

WOUND HEALING VERSUS FIBROPROLIFERATIVE DISORDERS

Wound healing is a multi-step process that requires a delicate balance and timing of the expression of different cytokines. Normal dermal repair is built upon an extracellular matrix that is invaded by inflammatory cells, newly forming blood vessels, and fibroblasts. Myofibroblasts, which are derived from fibroblasts, figure importantly in the process by laying down the extracellular matrix, using contractile fibers to close the wound, and expressing various cytokines that attract other cells into the area. It is when this process malfunctions, leading to persistence of myofibroblasts in the wound area, that pathological scar formation occurs. Scar formation is part of the healing process for wounds that are at least one-third the depth of the local skin.[13] In contrast, aberrant scar tissue may form even in the absence of a wound.

As shown in Figure 5, numerous regulatory signals, as well as their relative abundances, determine whether a response to an insult is physiological or pathological. Regardless, a key commonality is CTGF's involvement. Indeed, the widely recognized inflammatory cytokine transforming growth factor-β (TGF-β) requires the presence of this protein to initiate fibrosis.[14] It also interacts directly with other biochemical signals by virtue of its unique structure. CTGF consists of four modular segments, each with specific binding capabilities that enable it to interact with membrane-associated proteins, extracellular matrix components, and such cytokines as insulin-like growth factor, integrins, and bone morphogenic protein. Moreover, the molecule's N-terminal portion mediates myofibroblast differentiation and collagen synthesis, while the C-terminal domain regulates fibroblast proliferation. Hence, it is not surprising that overexpression of CTGF is associated with certain pathological conditions, including diabetic retinopathy, keloids, muscular dystrophy, atherosclerosis, and systemic sclerosis.[15,16,17,18,19] But even patients with certain fibrotic conditions, the regulatory balance associated with normalcy is not completely lost – CTGF mRNA expression is elevated only in affected areas of the skin of scleroderma patients.[20]

POST-SURGICAL SCARS

Scars that form at incision sites comprise an important aspect of medicine that is not fully appreciated by the general public, perhaps because they are not life-threatening. They do, however, take a toll on the psychological well-being and quality of life of the patient. An estimated 44 million surgeries are performed annually in the United States and a similar number in Europe that could benefit from scar reduction therapy.[21]

Attempts to treat scars are not considered ideal, as they are often invasive (e.g., surgical excision and cryotherapy), which may lead to a worse outcome, or have highly variable results (e.g., intralesional corticosteroid injections and laser therapy). The favored approach to post-surgical scarring is prevention, but the options available today leave ample room for improvement. Pressure therapy and silicone gel sheeting attempt to provide an environment that favors healing, possibly through local fluid retention. This

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[13] Dunkin, CS, et al. Scarring occurs at a critical depth of skin injury: precise measurement in a graduated dermal scratch in human volunteers. Plast Reconstr Surg (2007); 119(6): 1722.

[14] Wang, Q, et al. Cooperative interaction of CTGF and TGF-β in animal models of fibrotic disease. Fibrogenesis Tissue Repair (2011); 4(1): 4.

[15] Tikellis, C, et al. Connective tissue growth factor is up-regulated in diabetic retina: amelioration by angiotensin-converting enzyme inhibition. Endocrinol (2004); 145(2): 860.

[16] Haginoya, SG, et al. Connective tissue growth factor is overexpressed in muscles of human muscular dystrophy. J Neurol Sci (2008); 267(1-2): 48.

[17] Sonnylal, S, et al. Selective expression of connective tissue growth factor in fibroblasts in vivo promotes systemic tissue fibrosis. Arthritis Rheum (2010); 62(5): 1523.

[18] Manetti, M, et al. Severe fibrosis and increased expression of fibrogenic cytokines in the gastric wall of systemic sclerosis patients. Arthritis Rheum (2007); 56(10): 3442.

[19] Cicha, I, et al, Connective tissue growth factor is overexpressed in complicated atherosclerotic plaques and induces mononuclear clear cell chemotaxis in vitro. Arterioscler Thromb Vasc Biol (2005); 25(5): 1008.

[20] Quan, T, et al. Connective tissue growth factor: expression in human skin *in vivo* and inhibition by ultraviolet irradiation. J Invest Dermatol (2002); 118: 402.

[21] Bush, J, et al. Therapies with emerging evidence of efficacy: avotermin for the improvement of scarring. Dermatol Res Prac (2010);

may sound simplistic, but data indicates that rapid epithelialization (in fewer than 10 to 14 days) is essential to avoid excessive scar formation.[22]

Fortunately, the multiple steps involved in wound healing create an opportunity to intervene pharmacologically before scars form without interfering with the healing process. The stages of wound healing are diagramed in Figure 6.[23]

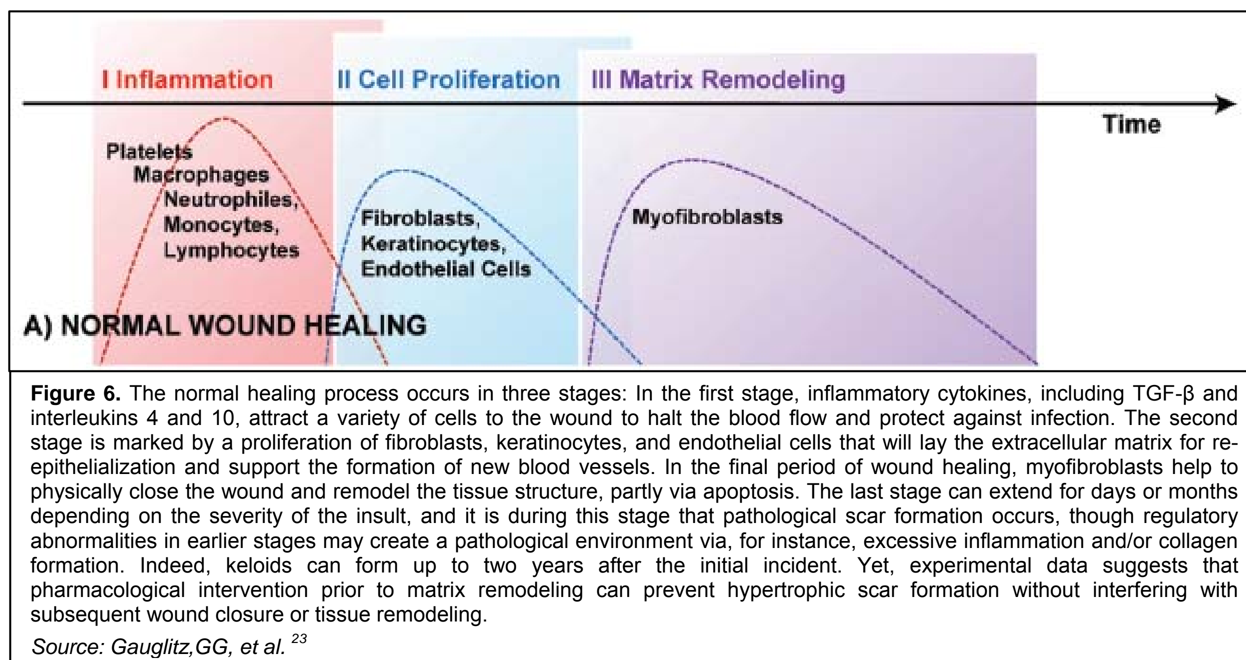

Figure 6. The normal healing process occurs in three stages: In the first stage, inflammatory cytokines, including TGF-β and interleukins 4 and 10, attract a variety of cells to the wound to halt the blood flow and protect against infection. The second stage is marked by a proliferation of fibroblasts, keratinocytes, and endothelial cells that will lay the extracellular matrix for re-epithelialization and support the formation of new blood vessels. In the final period of wound healing, myofibroblasts help to physically close the wound and remodel the tissue structure, partly via apoptosis. The last stage can extend for days or months depending on the severity of the insult, and it is during this stage that pathological scar formation occurs, though regulatory abnormalities in earlier stages may create a pathological environment via, for instance, excessive inflammation and/or collagen formation. Indeed, keloids can form up to two years after the initial incident. Yet, experimental data suggests that pharmacological intervention prior to matrix remodeling can prevent hypertrophic scar formation without interfering with subsequent wound closure or tissue remodeling.

Source: Gauglitz,GG, et al. [23]

An Anti-Scarring Medicine for Dermatological Applications

RXi's first drug candidate, RXI-109, has been created to prevent or minimize dermatological scars. Interest in CTGF has risen in the past few years, partly because of its central role in fibrotic diseases and because TGF-β is a poor target due to its involvement in a broad range of signaling pathways. As a result, a safe and effective treatment has the potential to address multiple fibrotic conditions of the skin and other tissues/organs. RXi's decision to pursue dermatological conditions was a smart move, in our opinion, since local drug administration increases the likelihood of clinical success – it reduces the risk of systemic side effects and increases the potential for the drug to reach the appropriate cells.

Targeting CTGF: RXi's decision to target CTGF is well founded on scientific research. Studies have used antisense probes, siRNA, and neutralizing antibodies to investigate the role of this cytokine in normal and pathological conditions in a variety of tissues.[24,25,26,27] Exposure of porcine fibroblasts to CTGF siRNA inhibits the expression of proteins associated with the extracellular matrix and selectively

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[22] Mustoe, TA, et al. International clinical recommendations on scar management. Plast Reconstr Surg (2002); 110(2): 560.
[23] Gauglitz, GG, et al. Hypertrophic scarring and keloids, pathomechanisms and current and emerging treatment strategies. Mol Med (2011); 17(1-2): 113.
[24] Guha, M, et al. Specific down-regulation of connective tissue growth factor attenuates progression of nephropathy in mouse models of type 1 and type 2 diabetes. FASEB J (2007); 21: 3355.
[25] Ponticos, M, et al. Pivotal role of connective tissue growth factor in lung fibrosis: MAPK-dependent transcriptional activation of type I collage. Arthritis Rheum (2009); 60(7): 2142.
[26] Kryger, SM, et al. Antisense inhibition of connective tissue growth factor (CTGF/CCN2) mRNA limits hypertrophic scarring without affecting wound healing in vivo. Wound Repair Regen (2008); 16(5): 661.
[27] Sherwood, MB. A sequential, multiple-treatment, targeted approach to reduce wound healing and failure of glaucoma filtration surgery in a rabbit model. Trans Am Ophthalmol Soc (2006); 104: 478.

down-regulates TGF-β mediated increases in extracellular matrix components, type I and III collagen.[28] Moreover, antisense inhibition of CTGF mRNA was found to have no effect on early wound healing in a preclinical model, although it significantly limits subsequent hypertrophic scarring.[29] This effect is associated with fewer myofibroblasts and decreased expression of types I and III collagen, as well as the tissue-inhibitor of metalloproteinase-1. A study involving human fibroblasts corroborated the effect of CTGF inhibition on collagen production.[30] More important, the research also found differential responses of normal and sclerodermic fibroblasts to a CTGF siRNA, as evidenced by an induction of matrix metalloproteinase-1 and a reduction in its endogenous inhibitor (tissue-inhibitor of metalloproteinase-1) in abnormal fibroblasts, but not normal cells. This is important since matrix metalloproteinase-1 is an enzyme that digests collagen in the extracellular matrix as a normal part of tissue remodeling. An increase in its expression, together with a drop in the level of endogenous inhibitor, probably creates an environment that favors a dampening, if not a reversal, of the pathological process. Indeed, normal aging is typified by low CTGF expression, which in turn causes the collagen content of the skin to decline.[31]

Administration of RXI-109 intradermally shows a good dose-response relationship in a preclinical model, with doses of 100µg and 300µg of the drug significantly reducing CTGF mRNA levels in the skin 48 hours after injection. When three doses were administered over a 10-day period, expression was suppressed significantly for 15 days. The results, which are depicted in Figure 7, set the stage for the Phase 1 trial.[32]

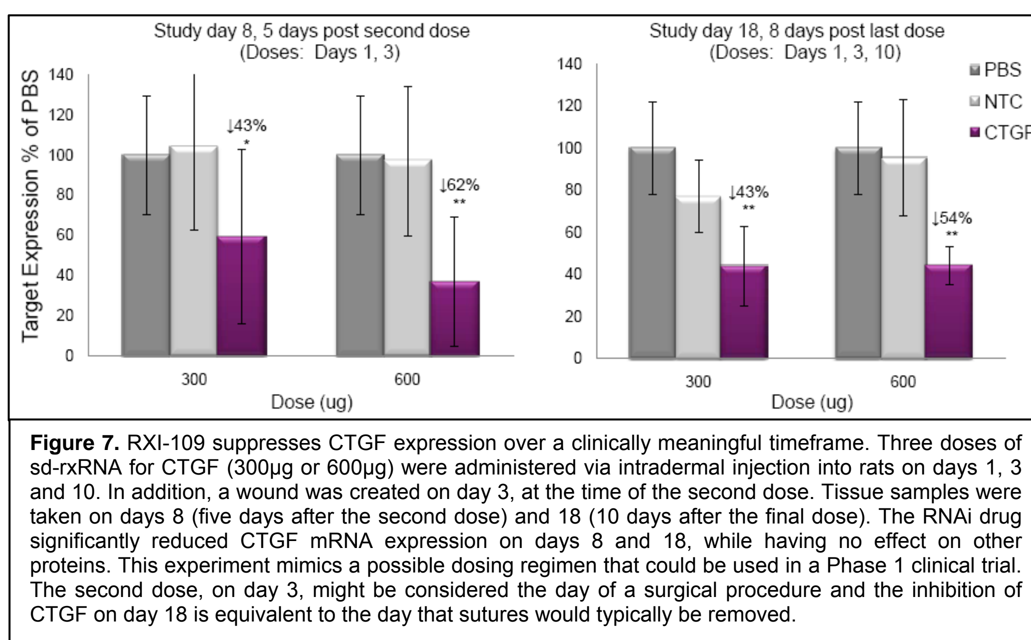

Figure 7. RXI-109 suppresses CTGF expression over a clinically meaningful timeframe. Three doses of sd-rxRNA for CTGF (300µg or 600µg) were administered via intradermal injection into rats on days 1, 3 and 10. In addition, a wound was created on day 3, at the time of the second dose. Tissue samples were taken on days 8 (five days after the second dose) and 18 (10 days after the final dose). The RNAi drug significantly reduced CTGF mRNA expression on days 8 and 18, while having no effect on other proteins. This experiment mimics a possible dosing regimen that could be used in a Phase 1 clinical trial. The second dose, on day 3, might be considered the day of a surgical procedure and the inhibition of CTGF on day 18 is equivalent to the day that sutures would typically be removed.

Phase 1 Clinical Trial: At this time, we are only able to provide a sketch of the Phase 1 study's design, since it will be the subject of a meeting between RXi and the FDA within the next few weeks. However, we do know that the Company will test RXI-109 for preventing/minimizing scars associated with surgery. This plan makes sense, since it will enable the Company to conduct the clinical trial in a well controlled manner, rather than under emergency conditions. The study will enroll approximately 25 patients

[28] Wang, JF, et al. Connective tissue growth factor siRNA modulates mRNA levels for a subset of molecules in normal and TGF-beta 1-stimulated porcine skin fibroblasts. Wound Repair Regen (2004); 12(2): 205.
[29] Sisco, M, et al. Antisense inhibition of connective tissue growth factor (CTGF/CCN2) mRNA limits hypertrophic scarring without affecting wound healing in vivo. Wound Repair Regen (2008); 16(5): 661.
[30] Ishibuchi, H, et al. Induction of matrix metalloproteinase-1 by small interfering RNA targeting connective tissue growth factor in dermal fibroblasts from patients with systemic sclerosis. Exp Dermatol (2010); 19: e111.
[31] Quan, TH, et al. Reduced expression of connective tissue growth factor (CTGF/CCN2) medicates collagen loss in chronologically aged human skin. J Invest Dermatol (2010); 130: 415.
[32] Beerman, ND. RXi Pharmaceuticals presentation at the BIO CEO Conference on February 14, 2011.

scheduled for an elective surgery, abdominoplasty or tummy tuck, which involves the removal of excess dermal tissue. The patients will initially undergo small incisions that will be used to assess the impact of locally administered RXI-109 on wound healing. (The subsequent tummy tuck will remove any evidence of the actual test.) Standard, objective methods[33] will be used to evaluate scar formation, and the results should provide an insight into the drug's safety and possibly, its effectiveness. In addition, tissue samples taken during the abdominoplasty will be analyzed morphologically to identify any impact of the drug on tissue structure at the site of the wound and surrounding area. Thus, the study should provide a rather complete picture of the effects of RXI-109.

BEYOND DERMATOLOGICAL APPLICATIONS

Diagnostics: Research has identified a single nucleotide polymorphism (substitution of guanine for cytosine) in the CTGF gene promoter region that is associated with increased CTGF expression and fibrotic conditions.[34] This genetic mutation is present in only a small fraction of the patients with a fibrotic disease, but one characteristic of fibrotic pathologies is that CTGF serves as a good biomarker. For instance, urinary levels of the cytokine are well correlated with kidney function in patients with diabetic nephropathy.[35] (Renal fibrosis is associated with high CTGF levels caused by advanced glycation end-products and reactive oxygen species.) Moreover, plasma levels are predictive for the development of end-stage renal disease and mortality in diabetic patients.[36] This should be important in optimizing a patient's therapy, because the cytokine may contribute to disease progression by attracting inflammatory cells into the kidney and supporting fibrogenesis.[37] Given the growing interest in combining diagnostic and therapeutic technologies to ensure that drugs are used appropriately, the ability to monitor CTGF levels should facilitate adoption of RXI-109 commercially.

Therapeutic Applications: One of the most attractive characteristics of RXi's CTGF drug is its ability to treat a wide range of conditions, well beyond scarring associated with surgeries. Indeed, this indication may not be the first that the Company decides to pursue commercially, but it does afford certain opportunities for the Phase 1 trial (i.e., simplified dosing regimen and ability to easily obtain tissue for morphological analyses). Figure 8 (on the next page) provides a list of the potential markets and information about the relative sizes of patient populations.

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[33] Vercelli, S, et al. How to assess postsurgical scars: a review of outcome measures. Disabil Rehabil (2009); 31(25): 2055.

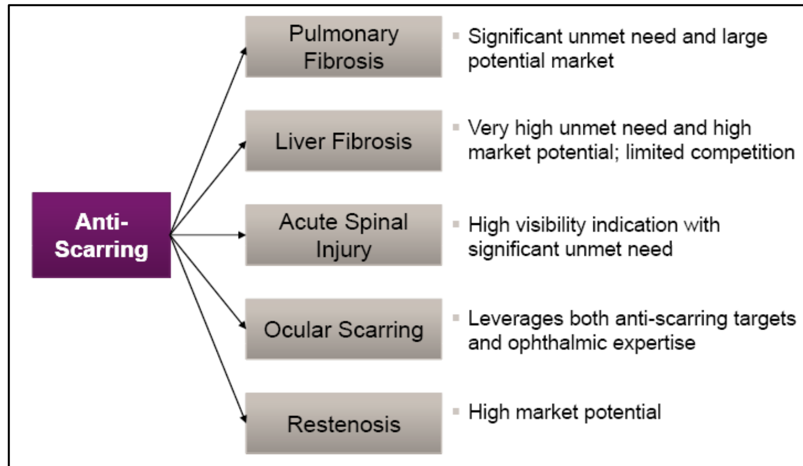
[34] Fonseca, C, et al. A polymorphism in the CTGF promoter region associated with systemic sclerosis. New Engl J Med (2007); 357: 1210.

[35] Nguyen, TQ, et al. Urinary connective tissue growth factor excretion correlates with clinical markers of renal disease in a large population of type 1 diabetic patients with diabetic nephropathy. Diabetes Care (2006); 29(1): 83

[36] Nguyen, TQ, et al. Plasma connective tissue growth factor is an independent predictor of end-stage renal disease and mortality in type 1 diabetic nephropathy. Diabetes Care (2008); 31(6): 1177.

[37] Sanchez-Lopez, E, et al. CTGF promotes inflammatory cell infiltration of the renal interstitium by activating NF-κB. J Am Soc Nephrol (2009); 20: 1513.

Figure 8. Large Commercial Opportunities for RXI-109 Abound



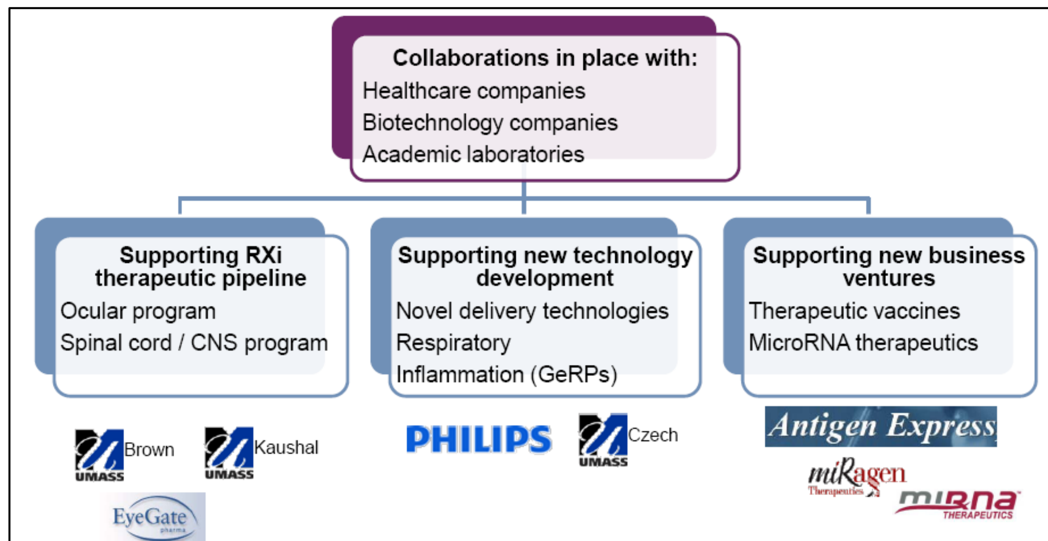
Source: Beerman, ND [32]

Within these categories are some with multiple indications, such as ocular scarring where the addressable market includes retinal disorders (i.e., age-related macular degeneration, diabetic retinopathy, and diabetic macular edema) and glaucoma-related surgery. Similarly, liver fibrosis occurs with such distinct diseases as non-alcoholic fatty liver disease, cirrhosis, and hepatitis.

COLLABORATIONS

Over the past few years, RXi has invested in its basic technology, which has yielded a self-delivering RNAi molecule that is chemically modified to protect it from being rapidly degraded *in vivo*. In addition, the R&D program has created a unique particle (GeRP) for delivery of RNAi molecules via Peyer's patches in the small intestine to the immune system. These investments have attracted the interest of collaborators, as shown in Figure 9:

Figure 9. Collaboration Agreements Advancing the Therapeutic Pipeline & Platform



Source: Beerman, ND[32]

More deals are expected in the months ahead, since the Company's technologies are applicable to numerous indications. Nonetheless, collaborations are not reflected in our financial analysis.

INVESTMENT CONCERNS AND RISKS

For a complete description of risks and uncertainties related to RXi Pharmaceuticals' business, see the "Risk Factors" section in RXi's SEC filings, which can be accessed directly from the SEC Edgar filings at www.sec.gov. Potential risks include:

- **Stock risk and market risk:** There is a limited trading market for the Company's common stock. There can be no assurance that an active and liquid trading market will develop or, if developed, that it will be sustained, which could limit one's ability to buy or sell the Company's common stock at a desired price. Investors should also consider technical risks common to many small-cap or micro-cap stock investments, such as small float, risk of dilution, dependence upon key personnel, and the strength of competitors that may be larger and better capitalized.

- **Competitive risk:** The pharmaceutical and biotechnology markets are rapidly evolving, and research and development are expected to continue at an accelerated pace. Other companies are also actively engaged in the development of therapies to directly or indirectly treat those disorders being pursued by RXi. These companies may have substantially greater research and development capabilities, as well as significantly greater marketing, financial, and human resources than RXi.

- **Products still in development phases:** RXi's products are still at the discovery and clinical testing stages. Such products may appear to be promising, but may not reach commercialization for various reasons, including failure to achieve regulatory approvals, safety concerns, and/or the inability to be manufactured at a reasonable cost. And even if its products are commercialized, there can be no assurance that they will be accepted, which may prevent the Company from becoming profitable.

- **Funding requirements:** It is difficult to predict the Company's future capital requirements. The Company may need additional financing to continue funding the research and development of its products and to expand its business. There is no guarantee that it can secure the desired future capital or, if sufficient capital is secured, that current shareholders will not suffer significant dilution.

- **Regulatory risk:** There is no guarantee that RXi's products will be approved by the U.S. Food and Drug Administration (FDA) or international regulatory bodies for marketing in the U.S. or abroad.

- **Patent risk:** The field of RNAi pharmaceuticals is at an early stage of development, and although RXi Pharmaceuticals has licensed and/or filed for numerous patents to secure its right to commercialize its technology, not all of these patents have been challenged, and therefore some may not protect the Company's rights adequately in a competitive marketplace.

Financial Forecasts & Valuation Analyses

We used two valuation methods to prepare a 12-month target price for RXi. A Discounted Cash Flow Analysis, which is based on the assumptions that begin on this page, yielded a valuation of $4.62 per share. A Comparative Analysis, which is discussed on page 20, used the current values of companies similar to RXi to estimate what its valuation will be in the first half of 2012 when its two clinical trials are enrolling patients. That analysis resulted in a valuation of $3.30 per share. Accordingly, we have set a 12-month target share price near the mid-range of these valuations, at $4.00.

Sources of Revenue

Our model includes three revenue streams for RXi over the next 15 years. Two revenue streams pertain to the use of NeuVax for preventing breast cancer recurrence: (i) the first year in which the patient receives seven doses to generate an immune response and (ii) subsequent years in which two doses are administered annually to maintain an effective immune surveillance. The basic assumptions related to these revenue streams are summarized in the following boxes:

NeuVax - Treatment			
Year penetration starts	2017	Incidence	25000
Starting penetration rate	25%	Percent addressable	90%
Years between penetration start and peak	5	Market growth rate	1%
Peak penetration	50%	Price per patient	$28,000
Duration of peak penetration in years	10	Treatment price growth	0%
Retention rate in decline years	90%	Royalty rate	0%
Stage of development	Phase 2	Probability of commercialization	35%

NeuVax - Maintenance			
Year penetration starts	2018	Incidence	25000
Starting penetration rate	30%	Percent addressable	50%
Years between penetration start and peak	8	Market growth rate	1%
Peak penetration	60%	Price per patient	$8,000
Duration of peak penetration in years	10	Treatment price growth	0%
Retention rate in decline years	90%	Royalty rate	0%
Stage of development	Phase 2	Probability of commercialization	35%

The third revenue stream derives from RXI-109 and is based on the indication that will be tested in the Phase 1 clinical trial, surgery-related scarring.

RXI-109			
Year penetration starts	2018	Incidence	44,000,000
Starting penetration rate	1%	Percent addressable	25%
Years between penetration start and peak	10	Market growth rate	1%
Peak penetration	10%	Price per patient	$1,000
Duration of peak penetration in years	5	Treatment price growth	0%
Retention rate in decline years	90%	Royalty rate	0%
Stage of development	Preclinical	Probability of commercialization	10%

Assumptions related to Revenue Sources:

- NeuVax is launched in 2017 to treat women who were clinically cured of node-positive breast cancer, characterized by low- to moderate-HER2 expression, and whose haploytype is either HLA-A2 or HLA-A3. Of this 25,000 patient population, approximately 90% are considered eligible for vaccination. Due

to the vaccine's success in preventing disease recurrence in clinical trials, its penetration rate rises from 30% the first year to 60% in its fifth year on the market. The price of the first year's treatment is $28,000, a figure that we have not adjusted for inflation after launch. In addition, we've set the probability of commercialization at 35%, which is in keeping with historical success rates for therapies that have completed a Phase 2 clinical trial.

- The second revenue source from NeuVax is related to its use in maintaining immune surveillance. This involves two doses of the vaccine per year at a price of $4,000 per dose. We've built this part of our model by assuming that patients receive booster vaccinations for only one year beyond the initial year of therapy, and that of those, only 50% are eligible for the booster vaccine. This may prove to be a conservative stance, since patients who are doing well probably would continue indefinitely. On the other hand, it provides ample cushion in the event that disease progression is seen beyond the 24 months of protection witnessed in the Phase 2 study. Other assumptions related to booster dosing is that only 30% opt to continue to receive treatment beyond the first year initially, though acceptance rises to 60% within five years.

- Our modeling of RXI-109 reflects an assumption that the drug is commercialized to prevent surgery-related scarring, the indication for which it will be tested in the Phase 1 trial. This patient population in the United States is estimated to number 44 million. However, we've assumed that only 25% of surgeries may generate scars in areas sensitive to patients' self-esteem. And despite our belief that the drug will be highly effective, we've assumed that its penetration of the market rises from 1% to 10% over a 10-year span, restrained by competition from other products. Furthermore, we've set a price of $1,000 for each surgical procedure and did not allow for price increases. The current probability of commercialization is 10%, which is in line with other drugs that are at a preclinical stage of development.

INCOME STATEMENT (All data are in thousands of dollars, except per-share figures.)

Fiscal year ends December 31st.

	2011	2012	2013	2014	2015
Total revenue	$ -	$ -	$ -	$ -	$ -
COGS	-	-	-	-	-
Gross profit	$ -	$ -	$ -	$ -	$ -
Operating expenses					
R&D	$ 8,050	$ 10,000	$ 12,000	$ 15,000	$ 18,000
Selling & marketing					
General & administrative	8,600	8,500	8,500	8,750	8,750
Total expense	16,650	18,500	20,500	23,750	26,750
Operating profit	$ (16,650)	$ (18,500)	$ (20,500)	$ (23,750)	$ (26,750)
Total non-operating	10	50	50	50	50
Pretax profit	$ (16,640)	$ (18,450)	$ (20,450)	$ (23,700)	$ (26,700)
Income tax					
Net income	$ (16,640)	$ (18,450)	$ (20,450)	$ (23,700)	$ (26,700)
Earnings (loss) per share	$ (0.52)	$ (0.51)	$ (0.51)	$ (0.53)	$ (0.53)
Diluted shares outstanding	32,000	36000	40000	45000	50000

Assumptions related to the Income Statement:

- We've assumed that RXi spends $16.6 million this year, including stock-based compensation of $5 million. The amounts devoted to R&D and general expenses are similar. Thereafter, we expect to see

R&D costs rise as the clinical trial of NeuVax enrolls more patients and RXI-109 advances into larger studies, while general corporate costs are restrained. The estimated cost of the NeuVax clinical trial is $10 million through the first interim analysis (at 70 events) and approximately $20 million to complete the trial (through 139 events).

- Non-operating costs over the next five years a nominal.
- The number of shares outstanding rises in 2011 due partly to the acquisition of Apthera. Other factors contributing to a growing number of shares outstanding are the exercise of stock warrants and options and external financing.
- Our long-range projections (beyond 2015) allow for R&D costs of 16% of sales; selling/marketing expenses at 20%, and general/administrative costs of 9%. In addition, we've applied an effective tax rate of 38%, starting in 2018 although net operating loss carryforwards will limit actual cash outlays for at least two years.

BALANCE SHEET # (ALL DATA ARE IN THOUSANDS OF DOLLARS.)

Fiscal year ends December 31st. The September 2010 balance sheet does not reflect the $8.1 million financing that RXi completed in 2011.

ASSETS	**9/30/2010**	**12/31/2009**
Current Assets		
Cash & equivalents	8,832	5,684
Accounts Receivable	-	-
Other	334	120
Total Current Assets	$ 9,166	$ 5,804
Property & equipment	$ 423	$ 432
Intangible assets	-	-
Other	16	16
Total Assets	$ 9,605	$ 6,252
LIABILITIES		
Current Liabilities		
Accounts payable	$ 836	$ 625
Capital lease obligations	52	52
Accrued liabilities	1,176	1,077
Warrant-related liabilities	4,210	3,721
Total Current Liabilities	$ 6,274	$ 5,475
Capital lease obligations	$ 43	$ 36
Other	-	-
Total Long-Term Liabilities	$ 43	$ 36
Shareholders Equity		
Common Stock, par value	$ 3	$ 2
Additional Paid-In Capital	61,046	44,489
Accumulated Deficit	(53,912)	(43,750)
Treasury Stock	(3,849)	-
Total Shareholders Equity	$ 3,288	$ 741
Total liabilities & equity	$ 9,605	$ 6,252

DISCOUNTED CASH FLOW VALUATION (ALL DATA ARE IN THOUSANDS OF DOLLARS, EXCEPT PER-SHARE FIGURES.)

	2011	2012	2013	2014	2015
Revenue	$ -	$ -	$ -	$ -	$ -
Operating income	-16650	-18500	-20500	-23750	-26750
Net income	-16640	-18450	-20450	-23700	-26700
Depreciation/amortization	150	150	150	150	150
Stock-based compensation	5000	5000	5000	5000	5000
Tax loss carryforwards	0	0	0	0	0
Capital expenditures	-75	-75	-75	-100	-200
Asset purchases					
Other					
Total cash flow adjustments	5,075	5,075	5,075	5,050	4,950
Free cash flow	$ (11,565)	$ (13,375)	$ (15,375)	$ (18,650)	$ (21,750)
Risk-adjusted free cash flow	$ (11,565)	$ (13,375)	$ (15,375)	$ (18,650)	$ (21,750)

		PV of Terminal Value at a Perpetual growth rate of rFCF			Enterprise Value		
Discount Rate	Discounted Cash Flows (2009 - 2024)	2.0%	3.0%	4.0%	2.0%	3.0%	4.0%
7.5%	$163,818.32	$ 486,202	$ 600,073	$ 779,013	$650,020	$763,891	$942,831
10.0%	$114,487.33	$ 236,770	$ 273,247	$ 321,883	$351,257	$387,734	$436,370
12.5%	$78,706.35	$ 128,774	$ 143,725	$ 162,193	$207,481	$222,431	$240,900
15.0%	$52,580.87	$ 74,799	$ 81,827	$ 90,132	$127,380	$134,408	$142,713
17.5%	$33,400.25	$ 45,437	$ 49,046	$ 53,191	$78,837	$82,447	$86,591

		Total Equity Value			Value per Diluted Share		
Discount Rate	Net Debt	2.0%	3.0%	4.0%	2.0%	3.0%	4.0%
7.5%	$ (8,737)	$658,757	$763,891	$951,568	$ 13.18	$ 15.28	$ 19.03
10.0%	(8,737)	$359,994	$396,471	$445,107	$ 7.20	$ 7.93	$ 8.90
12.5%	(8,737)	$216,218	$231,168	$249,637	$ 4.32	$ 4.62	$ 4.99
15.0%	(8,737)	$136,117	$143,145	$151,450	$ 2.72	$ 2.86	$ 3.03
17.5%	(8,737)	$87,574	$91,184	$95,328	$ 1.75	$ 1.82	$ 1.91

	Terminal Value as % Enterprise Value			Implied EBITDA Multiple		
Discount Rate	2.0%	3.0%	4.0%	2.0%	3.0%	4.0%
7.5%	74.8%	78.6%	82.6%	11.60	14.32	18.59
10.0%	67.4%	70.5%	73.8%	7.98	9.21	10.85
12.5%	62.1%	64.6%	67.3%	6.08	6.78	7.66
15.0%	58.7%	60.9%	63.2%	4.91	5.37	5.92
17.5%	57.6%	59.5%	61.4%	4.12	4.44	4.82

Assumptions related to the Discounted Cash Flow Analysis:

- The DCF model projects cash flow through 2026, discounted back at multiple annual rates (7.5%, 10.0%, 12.5%, 15.0%, and 17.5%) to demonstrate the potential variability related to this assumption. It also includes three perpetual growth rates (2%, 3%, and 4%) to show the impact on the present value of the company's terminal value. The rates used in calculating the per-share value for Unilife Corporation are a 12.5% annual discount rate and a perpetual growth rate of 3%. The number of fully-diluted shares estimated to be outstanding in 2015, 60 million, is used in the per-share calculation.
- The cash flows are risk adjusted, based on the proportional gross profit contribution by each production line on an annual basis and the probability of that line starting up as projected. For any years in which we are projecting negative cash flow, the probability is conservatively set at 100%.

COMPARATIVE VALUATION ANALYSIS

We used a separate, comparative valuation analysis to assess what RXi's share price would be in early 2012, when RXI-109 and NeuVax are expected to be in Phase 1 and 3 clinical trials. We selected companies with drugs derived from a variety of technologies, including RNAi, antisense, DNA or peptide immunotherapy, and a well-tolerated chemotherapy. Moreover, the drugs are being developed as therapies for cancer and/or dermatological applications.

The companies differed greatly in their market capitalizations, ranging from $18.6 million to $910.1 million. To utilize their valuations for estimating RXi's market capitalization in the first half of 2012, we divided each company's market capitalization by the number of candidates adjusted for each drug's clinical stage of development. The adjustment was effected by giving each drug in a Phase 1 trial a value of 1, Phase 2 drugs a value of 2, and Phase 3 drugs a value of 3. These figures generally reflect the relative probabilities of drugs in these stages of development being approved.[38]

Company	Ticker	Number of ClinicalCandidates	Market Cap	# of Candidates Phase Adjusted	Value per Candidate Phase Adjusted
Alnylam	ALNY	6	$406.10	7	$58.01
Immunocellular Ther.	IMUC.OB	1	$48.90	2	$24.45
Isis	ISIS	16	$910.10	31	$29.36
Provectus	PVCT.OB	3	$91.40	5	$18.28
Scancell Holdings	LON: SCLP	1	$18.60	1	$18.60
				Average Value Per Phase Adjusted Candidate:	$29.74
				Estimated Market Cap of RXII shares:	**$118.96**
				Estimated Share Price:	**$3.30**

The results of this analysis found that each drug candidate contributed $29.74 million on a phase-adjusted basis to a company's market capitalization. This did not take into consideration differences in the market sizes that the drugs may ultimately reach or characteristics of the companies, such as their ability to raise capital. But the technologies used to develop these compounds and the general indications that they will target are similar to those of RXi. Based on the average, phase-adjusted contribution to a corporation's market capitalization, we calculated that RXi would merit a valuation of approximately $119 million in early 2012, or a price of $3.30 per share, based on an estimated 36 million shares outstanding at that time.

[38] DiMassi, JA. Risks in new drug development: Approval success rates for investigational drugs. Clin Pharmacol Ther (2001); 69: 297.

DISCLOSURES

ANALYST(s) CERTIFICATION: The analyst(s) responsible for covering the securities in this report certify that the views expressed in this research report accurately reflect their personal views about RXi Pharmaceuticals Corporation (the "Company") and its securities. The analyst(s) responsible for covering the securities in this report certify that no part of their compensation was, is, or will be directly or indirectly related to the specific recommendation or view contained in this research report.

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2-YEAR PRICE CHART

Source: BigCharts.com

9/03/08 – Initiating Coverage: share price: $6.89; rating: BUY; 12-month price target: $23.00; **7/30/2009** – Update Report: share price: $4.59, rating: BUY, 12-month price target: $22.00; **5/13/2010** – Update Report: share price: $4.42; rating: BUY, 12-month price target: $22.00; **7/19/2010** – Update Report: share price: $2.17, rating: BUY, 12-month price target: $8.50; **12/21/2010** – Update Report; share price: $2.75; rating: BUY, 12-month price target: $8.50; **4/5/2011** – Update Report; share price: $1.47, rating: BUY, 12-month price target: $4.00.

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