AEGIS CAPITAL CORP

Biotechnology
Raghuram Selvaraju, Ph.D.
(646) 502-2464
rselvaraju@aegiscap.com

Yi Chen, Ph.D.
(646) 502-2463
ychen@aegiscap.com

Initiating Coverage

July 25, 2012

Galectin Therapeutics, Inc.

Rating: Buy

Galectin Therapeutics: A Diamond In The Rough

Key Metrics

GALT - NASDAQ	$2.48
Pricing Date	Jul 24 2012
Price Target	$7.00
52-Week Range	$7.08 - $1.61
Shares Outstanding (mm)	15.7
Market Capitalization ($mm)	$38.9
3-Mo Average Daily Volume	39,230
Institutional Ownership	0%
Debt/Total Capital	NM
ROE	NM
Book Value/Share	$0.40
Price/Book	6.2x
Dividend Yield	NM
LTM EBITDA Margin	NM

EPS ($) FY: December

	2011A	Prior 2012E	Curr. 2012E	Prior 2013E	Curr. 2013E
1Q-Mar	(0.04)	--	(0.17)A	--	(0.17)E
2Q-Jun	(0.06)	--	(0.16)E	--	(0.16)E
3Q-Sep	(0.03)	--	(0.18)E	--	(0.16)E
4Q-Dec	(0.31)	--	(0.19)E	--	(0.16)E
FY	(0.23)	--	(0.71)E	--	(0.65)E
P/E	NM		NM		NM

Company Description:

Galectin Therapeutics, Inc., an emerging biotechnology firm (http://www.galectintherapeutics.com/), is headquartered in Newton, MA. The firm focuses on developing therapies for cancer and fibrotic diseases.

Investment Highlights:

- **Initiating Coverage.** We are initiating coverage of Galectin Therapeutics with a Buy rating and an 18-month price target of $7.00 per share. In our view, the firm is an undiscovered gem that benefits from a focus on significant unmet medical needs, unaddressed markets, and chronic conditions. Furthermore, we believe that this company possesses a technology platform founded upon cutting-edge science and validated targets.
- **Galectin Science.** Many scientists have been working to improve their understanding and application of galectin science because of the vast nature of its application in oncology and immunology. In our opinion, Galectin Therapeutics is far ahead of the competition with respect to developing drugs that can target galectin proteins. Because the backbone of this technology is carbohydrate-based, the company's drugs have the advantages of antibody technology (i.e., a long half-life and targeting) and are very safe because of simple breakdown molecules.
- **Turnaround Story.** Over the past few years, Galectin's research team has substantially developed galectin-targeting drugs despite tough macroeconomic conditions and inadequate strategies set forth by previous top management. Galectin's current CEO, Dr. Peter Traber, formerly the Chief Medical Officer of GlaxoSmithKline, has a very strong background in research, medicine, and business. In our opinion, Dr. Traber has been able to quickly reevaluate the company's business, redirecting its focus to areas of strategic interest that, in our opinion, could yield significant share price appreciation from current levels.
- **Strong Data From Lead Drug.** From Phase 1 and 2 studies with the firm's lead drug candidate GM-CT-01, there is solid proof of safety and encouraging signs of efficacy in colorectal cancer. In addition, Dr. Traber has begun to focus the company on developing a drug for the treatment of liver fibrosis, currently designated GR-MD-02. Given the high unmet need in liver fibrosis, we believe peak global sales for a drug that can reverse fibrosis could be $1.7 billion in 2020. In our opinion, there is very strong preclinical proof that the company's drugs can reverse liver fibrosis. Management expects to initiate a Phase 1 trial in liver fibrosis patients in early 2013, with possible proof-of-concept data by early 2014.

The Disclosure section may be found on pages 36 - 37 of this report.

Investment Thesis

Galectin Therapeutics is a development-stage company engaged in drug development to create new therapies for cancer and fibrotic disease. Formerly known as Pro-Pharmaceuticals, Galectin has developed a technology platform in which drug candidates are based on a proprietary method of targeting galectin proteins, which are key mediators of biologic and pathologic function. The firm uses naturally occurring plant materials to create complex carbohydrates with specific molecular weights and pharmaceutical properties. Deploying these unique carbohydrate-based candidate compounds that bind and inhibit galectin proteins, Galectin Therapeutics is undertaking the pursuit of therapies for indications where galectins have a clear role in the pathogenesis of a given disease. Galectin focuses on serious, life-threatening, chronic conditions, wherein current treatment options are typically limited.

We are initiating coverage with a Buy rating and an 18-month price target of $7.00 per share, implying a total firm value of $250mm and assuming 34mm fully-diluted shares outstanding as of the end of 2013. An investment in Galectin Therapeutics shares may involve above-average risk and volatility, in our opinion, since the firm has not obtained regulatory approval for any drug candidates to date.

Investment Positives

Highly Validated Drug Development Target With Proven Mechanism. We believe that galectin blockade as a therapeutic strategy constitutes a substantially validated approach, since it has been shown that galectins are expressed at high levels by both fibrotic as well as tumorigenic tissue, that the expression of galectins and their interaction with galectin receptors mediates various pathological processes, and that selective inhibition of galectin signaling pathways is able to attenuate these cascades. The result of selective galectin blockade is the suppression – and, in some situations, the reversal – of fibrosis as well as cancer growth and metastasis.

Prior Clinical Proof-of-Concept Data Established. The company's most broadly tested clinical candidate, GM-CT-01 (formerly known as DAVANAT®), has been assessed in patients with various types of cancer, including end-stage, advanced cancer patients; individuals with advanced colorectal cancer; and patients with metastatic melanoma. Several of the studies conducted provided intriguing signs of efficacy. It was notable in these studies that, when the results of adverse events were pooled, there appeared to be a marked reduction in the severity of 5-fluorouracil (5-FU) related adverse events vs. historical controls. Accordingly, therefore, there may be a significant protective effect of galectin inhibition from the toxic side effects of chemotherapy in treated subjects.

Significant Unmet Medical Need With Favorable Competitive Landscape. In our view, the competitive landscape in the oncology setting remains favorable, even when considering the vast numbers of oncology-focused drug candidates and the frequency with which novel oncology drugs have emerged over the course of the past decade. Many currently-utilized anti-cancer agents are extremely toxic. In addition, cancers that may be treatable with GM-CT-01 (e.g., colorectal cancer, metastatic melanoma) remain recalcitrant to existing therapy. We note that the competitive landscape in non-alcoholic steatohepatitis (NASH), Galectin's other chosen development domain, is even more favorable; nothing is formally approved to treat this condition, and there are no other drug candidates currently in clinical development. Accordingly, therefore, we believe that if Galectin Therapeutics is successful in entering the clinical with GR-MD-02, substantial value should be ascribed to the product candidate's potential in NASH because there is a high likelihood that, if approved, the drug could be free of competition for several years at least. Since NASH is a debilitating chronic disorder, the drug could command premium pricing as well, contingent upon substantial efficacy being shown.

Investment Risks

Financial Outlook and History of Unprofitable Operations. Galectin Therapeutics has incurred operating losses since inception and, in our view, may not achieve sustainable profitability for several years. We estimate that the firm may raise additional funds within the next 12 – 18 months to support testing of its pipeline candidates in the U.S. Because of these factors, Galectin Therapeutics shares may constitute above-average risk and volatility, in our opinion.

FDA Unpredictability. Drug development is a multi-year process that requires human clinical trials prior to market entry. The agency may require substantial pivotal clinical trial data from Galectin Therapeutics prior to granting approval for its pipeline candidates, necessitating lengthy development times for the firm's lead drug candidates. Also, review times at the FDA may prove longer than originally expected. If clinical data and/or other supporting evidence are not accepted by the FDA, marketing authorization for Galectin's lead candidates could be delayed or might not occur at all, preventing the firm from realizing the commercial potential of its pipeline.

Potential Dependency on Partners to Provide Enhanced Market Penetration. Galectin Therapeutics currently lacks any direct sales and marketing organization or commercial infrastructure. The firm does not, at this point, have any plans to forward integrate and could elect to either be acquired or partner its lead candidates with an established pharmaceutical firm once clinical proof-of-concept is achieved. We think that a lack of partnering or acquisition interest could prevent the firm from commercializing its product candidates, should they achieve regulatory approval.

Competitive Landscape. Galectin is likely to compete with other companies within the drug development industry, many of which have more capital, more extensive research and development capabilities, and greater human resources. Some of these competitors with documented interest in both the fibrosis and oncology arenas include Amgen Inc. Biogen Idec, Bristol-Myers Squibb, Dendreon Corporation, GlaxoSmithKline and Pfizer.

Intellectual Property Risk. The company relies on patents and trade secrets to protect its products from competition. A court might not uphold Galectin's intellectual property rights, or it could find that Galectin infringed upon another party's property rights. In addition, generics firms could potentially launch generic versions of GM-CT-01, GR-MD-02, or other candidates prior to the expiration of patent protection on these products.

Reimbursement Risk. Following the institution of broad-based healthcare reform policy, reimbursement agencies have grown more wary of systematically reimbursing for drugs that are either unnecessary or provide marginal benefit at excessive cost. If Medicare spending growth continues to outpace GDP growth, and governmental ability to fund healthcare becomes impaired, changes could be made to reimbursement policy that would negatively affect Galectin Therapeutics, despite what we believe to be the compelling value proposition inherent in both GR-MD-02 and GM-CT-01.

Additional Risks. As of March 31st, 2012, Galectin Therapeutics had $15.3 million in cash and equivalents. Other sources of cash could include: licensing fees from partnerships, warrant and option exercises or issuance of more shares. If GM-CT-01 and GR-MD-02 fail in proof-of-concept studies, Galectin may not be able to raise cash at all.

Industry Risks. Emerging biotechnology and pharmaceuticals stocks are inherently volatile and increasingly subject to development and regulatory risk. Meeting or missing commercialization milestones may result in a significant change in the perception of the company and the stock price. We do not anticipate volatility subsiding in the near term.

For additional risk considerations, please refer to the company's SEC filings.

Valuation

Comparables Analysis: Given that Galectin Therapeutics is currently unprofitable and considering our belief that sustainable profitability is a few years away, we use a discounted cash flow-based approach to value the shares. Based on a comparables analysis, it appears the stock is worth roughly $7.00 per share, utilizing our estimate of a $210 million risk-adjusted net present value (rNPV) for the firm's products. This assumes that the shares trade in-line with the comp group's present average enterprise value of roughly $200 million and that the firm has 34 million shares outstanding (fully-diluted) and $40 million in cash at end-2013 (including proceeds from warrant exercises).

Table 1: Comparable Company Analysis
(Millions, Except Per-Share Data)

Development	Therapeutic Area	Company	Ticker	Rating	Closing price 7/24/2012	Shares (MM)	Market cap ($MM)	Cash ($MM)	Debt ($MM)	Enterprise value ($MM)
Phase 2	Infectious Diseases	Achillion Pharmaceuticals	ACHN	Not Rated	$6.11	71	432	65	1	368
Pre-registration	Inflammation / Metabolic Disorders / Diagnostics	Ampio Pharmaceuticals	AMPE	Not Rated	$3.13	31	98	8	0	89
Preclinical	Stem Cells	BioTime, Inc.	BTX	Not Rated	$4.46	50	225	16	0	208
Phase 1 / 2	CNS Disorders	Neuralstem, Inc.	CUR	Buy	$0.91	54	49	5	0	44
Phase 1 / 2	Orphan Disorders	Synageva BioPharma	GEVA	Not Rated	$49.10	21	1043	233	0	810
Phase 1 / 2	Biosimilars	Medgenics	MDGN	Not Rated	$12.05	10	117	11	0	106
Preclinical	CNS Disorders	InVivo Therapeutics	NVIV.OB	Buy	$2.30	64	147	20	0	128
Preclinical	Stem Cells	Organovo	ONVO.OB	Not Rated	$1.60	44	70	10	0	60
Phase 2	Monoclonal Antibodies	Sorrento Therapeutics	SRNE.OB	Not Rated	$0.21	261	55	2	0	52
Preclinical	Oncology	VeraStem	VSTM	Not Rated	$9.64	21	203	41	0	162
Phase 2 / 3	Hematology / Oncology	YM BioSciences	YMI	Not Rated	$1.93	157	304	133	0	171
		Average					249			200
								Discrepancy		
Current valuation	Fibrosis / Oncology	Galectin Therapeutics	GALT	Buy	$2.48	16	39	15	0	24
					Derived 18-month comparable value					
										Projected
Target valuation (18-month)	Fibrosis / Oncology	Galectin Therapeutics	GALT	Buy	$7.00	34	240	40	0	200

Source: First Call and Aegis Capital Corp. estimates

Free Cash Flow: We estimate that Galectin Therapeutics will be free cash flow negative for the foreseeable future. We define free cash flow as operating cash flow minus capital expenditures and dividend payments. We utilize a discounted cash flow analysis supporting a risk-adjusted Net Present Value (rNPV) framework to derive our 18-month $7.00 price target. This approach is described further in the next section of the report. Our detailed analysis is split into three components – our discounted cash flow model comprising the rNPV of GR-MD-02 and GM-CT-01, along with our assessments of the market sizes for GR-MD-02 and GM-CT-01 and the associated sales models for these drug candidates; the residual value of the company's drug development technology platforms; and the near-term financial outlook for the company. Our historical income statement and financial projections are presented at the back of this report.

Risk-Adjusted Net Present Value Analysis

The table below depicts our rNPV breakdown of the sum-of-the-parts discounted cash flow valuation approach used for Galectin Therapeutics. We ascribe a $90 million rNPV to GR-MD-02, the firm's principal value driver, along with an $80 million rNPV value to GM-CT-01, the company's most broadly-tested clinical candidate. We also provide a $40 million residual value for the company's technology platform, yielding a total enterprise value of $210 million – in-line with the comparables-based analysis above.

Table 2: Risk-Adjusted NPV-Based Sum-of-the-Parts Analysis

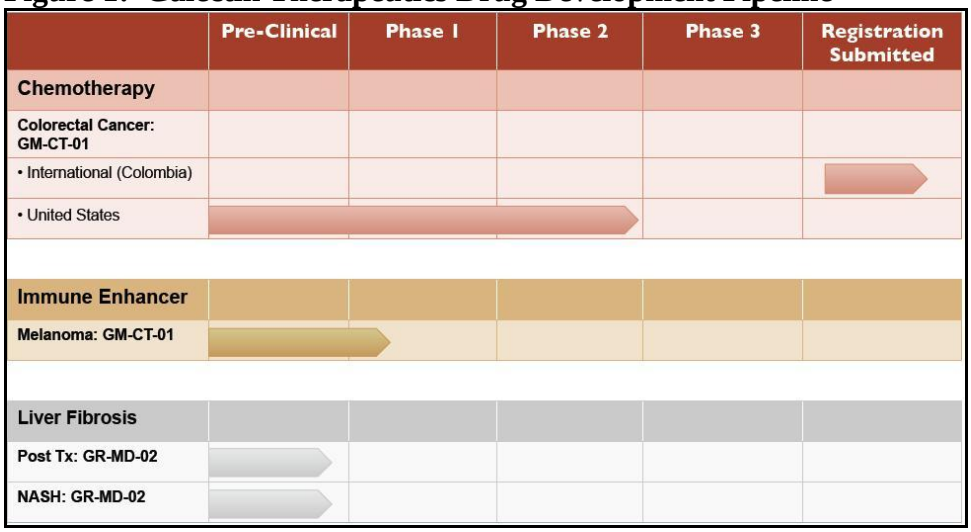
Galectin Therapeutics	Product	Launch Year	Patent Expiry	Peak Sales Estimate	Royalty Rate	Probability To Launch	NPV	Amount Per Share
Preclinical								
Liver Fibrosis	GR-MD-02	2017	2028	$5.3B	12-22%	25%	$90MM	$2.67
Cancer	GM-CT-01	2015	2028	$3.4B	10-18%	25%	$80MM	$2.37
Total							$170MM	$5.03
Technology Platform	Galectin Inhibition	NA		NA	NA	NA	$40MM	$1.18
Cash at end-2013							$40MM	$1.20
Firm Value							$250MM	$7.00

Source: Company reports; Aegis Capital Corp. estimates

Company Overview

Galectin Therapeutics, a small biopharmaceutical company based in Newton, MA, is a leader of galectin science, applying its expertise to drug development for fibrotic disease and oncology. The company recently changed its name from Pro-Pharmaceuticals to Galectin Therapeutics, which more accurately reflects its core expertise in galectin science. Although Galectin could pursue a broad range of diseases, considering the vast array of conditions in which galectin function has been implicated, management has instead chosen to concentrate on diseases with serious, life-threatening consequences, especially those where current treatment options are limited or non-existent. The firm is currently focused on oncology and liver fibrosis because these are life-threatening diseases for which there exists strong proof-of-concept data. In the future, the firm's technology could be applicable in both dermatology and neurology (stroke).

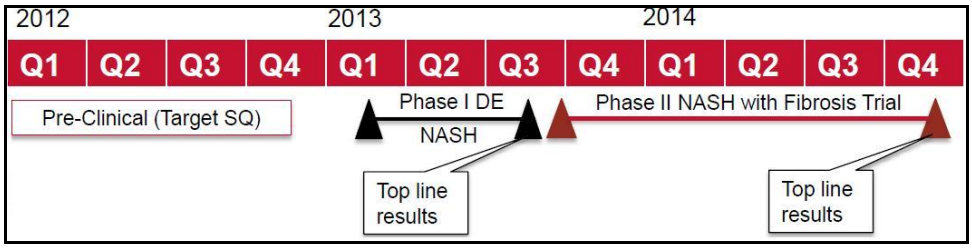
Figure 1: Galectin Therapeutics Drug Development Pipeline



Source: Company presentations

Even though the company is small, it developed a broad-based development program for one of its lead compounds in cancer. More recently, Galectin Therapeutics has characterized the function of the galectin pathway and developed a therapeutic approach to the treatment of liver fibrosis. Galectin is currently developing the liver fibrosis program on its own. This program targets a specific sub-indication of hepatic fibrosis known as non-alcoholic steatohepatitis (NASH), for which there are no current treatments and no clinical-stage compounds in development at this time. GR-MD-02 could potentially achieve a rapid path to market and enjoy exclusivity along with premium pricing if demonstrated to be effective in human proof-of-concept clinical studies. In our view, the company could eventually build a commercial infrastructure around this compound if it chose to pursue such a strategy, or out-license it under favorable terms. The timeline for this agent's clinical development is depicted below.

Figure 2: GR-MD-02 Clinical Development Timeline

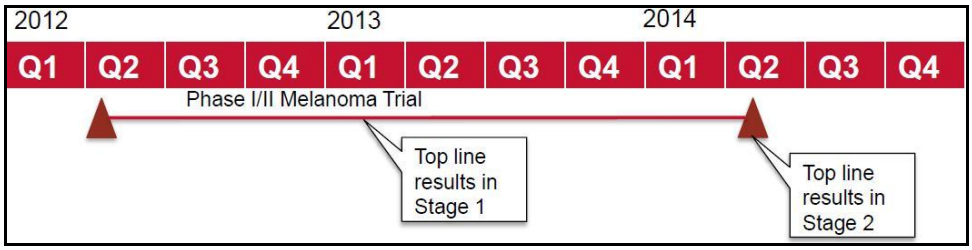


Source: Company presentations

The firm's second lead molecule, which was originally being developed under the trade name DAVANAT®, is now designated GM-CT-01. Like GR-MD-02, this agent makes use of the proprietary Galectin technology platform focusing on carbohydrate chemistry. Unlike GR-MD-02, however, which has thus far not been tested in humans, GM-CT-01 has been exposed to over 200 human subjects with a positive safety track record and clear indications of activity in the target indication, oncology.

While Galectin was originally developing DAVANAT® as an adjunctive agent for combination with chemotherapy, the firm has recently elected to explore the possibility of using the compound – under its new name – as a potential adjunct to immunotherapy. The key target indications are both in the solid tumor setting – colorectal cancer and malignant melanoma. In both indications, overall median survival is relatively short, with newly diagnosed metastatic colorectal cancer patients surviving for only 14 months (7.8 months in patients between 65 and 75 years of age) and metastatic malignant melanoma patients surviving for only 7.5 months.

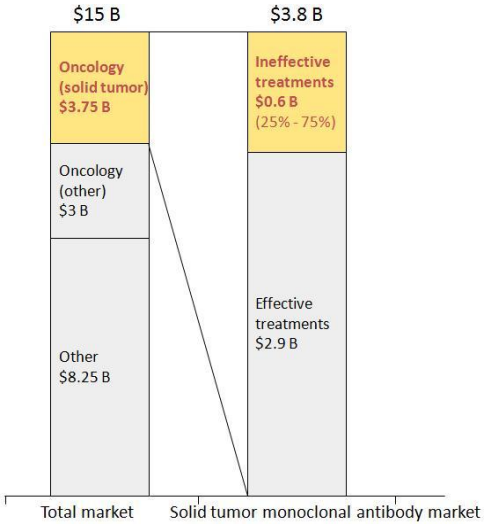
Figure 3: GM-CT-01 Clinical Development Timeline



Source: Company presentations

Galectin inhibition could play a key role in the burgeoning area of cancer immunotherapy. For example, two immunotherapy drugs were recently approved – Provenge (sipuleucel-T) from Dendreon; a dendritic cell tumor vaccine; and Yervoy (ipilimumab), developed by Medarex (which was acquired by Bristol-Myers Squibb) a monoclonal inhibitor of the CTLA-4 antigen, which activates cytotoxic T-cells. This market could grow to over $7 billion by 2015. The bar chart below depicts the size of the therapeutic market sub-segments in the oncology sector. Many monoclonal antibodies are often ineffective, and Galectin's approach could address this problem.

Figure 4: Therapy Sub-Segments In Oncology (U.S. Annual Market)



Source: Harvard University School of Medicine

Firm History

The initial predecessor of Galectin Therapeutics, DTR-Med Pharma Corp., or DTR, was incorporated in Nevada on January 26th, 2001. On April 25th, 2001, DTR entered into a stock exchange agreement with Pro-Pharmaceuticals, Inc., a Massachusetts corporation, whereby DTR acquired all of the outstanding shares of common stock of Pro-Pharmaceuticals, Inc. On May 10th, 2001, DTR changed its name to "Pro-Pharmaceuticals, Inc." and on June 7th, 2001, the Massachusetts corporation was merged into the Nevada corporation. On May 26th, 2011, Pro-Pharmaceuticals, Inc. changed its name to "Galectin Therapeutics Inc."

On March 22nd, 2012, Galectin Therapeutics entered into an underwriting agreement, relating to the offer and sale of 1,159,445 units, each of which consisted of two shares of the company's common stock and one warrant to purchase one share of the firm's common stock. The sole book-runner on the offering was Aegis Capital Corp. The public offering price for each unit was $9.00. Each warrant has an initial exercise price of $5.63 per share, is exercisable upon separation of the units, and expires on March 28th, 2017. In connection with the offering, Galectin Therapeutics conducted a one-for-six reverse stock split of its common stock. This was performed in order to enable the company to comply with minimum bid price requirements for listing on a regulated exchange. On March 23rd, 2012, the company's common stock ceased to trade on the OTC Bulletin Board and began trading on the NASDAQ Capital Market exchange under the symbol "GALT".

Pursuant to the underwriting agreement, the company granted the underwriters a 45-day option to purchase up to an additional 173,916 units to cover over-allotments, which was exercised in full on March 26th, 2012. On March 28, 2012, the company sold 1,333,361 units (2,666,722 shares of common stock and related warrants to purchase 1,333,361 shares of common stock) for gross proceeds of $12.0 million. Net proceeds of approximately $10.5 million accrued to the firm after the underwriting discount and offering costs. Galectin expects the net proceeds from this offering, in addition to the previous cash position, to be sufficient to fund operations through the end of 2013. On March 28th, 2012, the units and warrants that the firm sold in the offering began trading on that exchange under the symbols GALTU and GALTW, respectively.

PROCAPS S.A. Agreement

On October 18, 2011, Galectin Therapeutics entered into a Collaboration, Supply, Marketing and Distribution Agreement, which granted PROCAPS S.A., or PROCAPS, exclusive rights to market and sell GM-CT-01 to treat cancer in Colombia, South America. PROCAPS is a large, international, privately-held pharmaceutical firm based in Barranquilla, Colombia. Under the terms of the agreement, PROCAPS is responsible for obtaining regulatory and pricing approval in Colombia for GM-CT-01, along with vial filling, packaging, marketing, and distribution.

In October 2010, Galectin received a payment of $200,000 and shipped GM-CT-01 to PROCAPS to be used by PROCAPS to qualify its vial filling process and to replicate Galectin's previously-conducted stability study. At this stage, we do not have visibility as to when or if PROCAPS will be able to secure formal approval for GM-CT-01 to treat colorectal cancer from INVIMA, the Colombian regulatory agency. Accordingly, we do not currently forecast additional revenues accruing from Galectin Therapeutics' agreement with PROCAPS. Should PROCAPS succeed in entering the Colombian market with GM-CT-01, royalties on net sales of the agent in Colombia would be payable to Galectin. Accordingly, additional upside from this agreement could occur and is not reflected in our current estimates for the company.

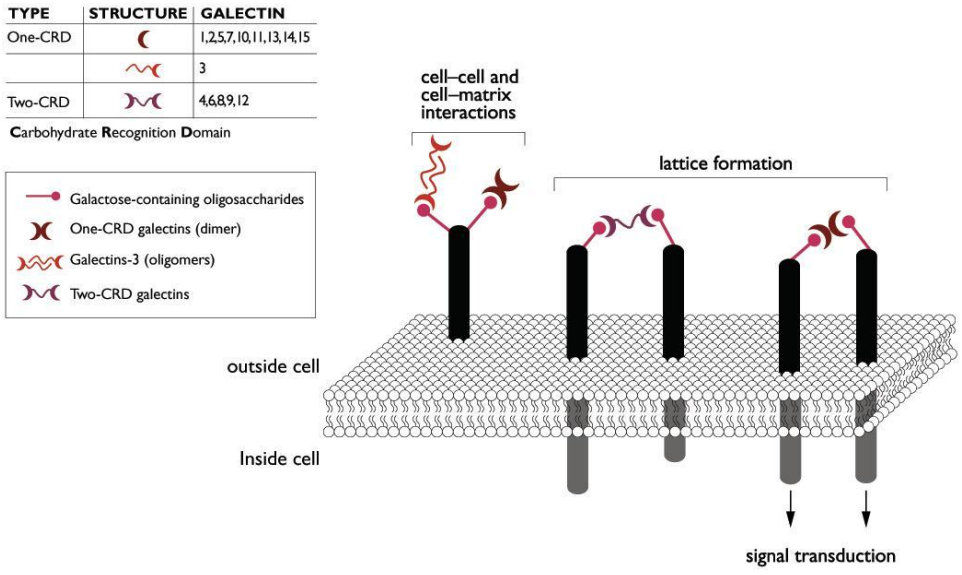
Galectin Technology Overview

We believe Galectin Therapeutics is one of the few companies in the biotechnology sector with expertise in carbohydrate technologies. The company is developing drugs that are based on a carbohydrate backbone. Carbohydrate chemistry is extremely complicated, and today there is a dearth of carbohydrate-based drugs globally, despite the wide range of conditions in which carbohydrate-based molecular pathways have been implicated. Very few chemists possess commanding knowledge of carbohydrates, even within the world's largest and most established pharmaceutical companies. While there are natural drugs with carbohydrates linked to them, and some antibiotics, there are currently very few synthetic carbohydrate-based drugs that have been approved for human therapeutic use. As a result, there are very few companies in this sector that focus on carbohydrate technologies, making Galectin Therapeutics one of a decidedly rare breed. In our view, this improves the competitive-landscape outlook for the firm.

Structure of Galectin Proteins

The company has established a technology platform to develop unique carbohydrate-based molecules that target several galectin proteins collectively. There are roughly 15 different galectin proteins, all of which have a common structure and some variable element. The common structure in every galectin contains a carbohydrate recognition domain that binds to ß-galactose-containing carbohydrates and glycoproteins. The figure below depicts the basic structural components of galectins, and how they are presented on cells. Interactions between galectin molecules on cell surfaces (cell-cell interactions) and between cells and the extracellular matrix (cell-matrix interactions) can initiate signal transduction cascades, stimulating cell proliferation, migration, and differentiation. It can also cause cells to produce various pro-fibrotic and pro-inflammatory factors.

Figure 5: Galectin Subunit Structure



Source: Galectin Therapeutics, Inc.

As shown above, the variable element in galectins is an oligomerization domain that enables binding of one protein to another. Each unit of drug can target several galectin proteins. Collective targeting is important in this case because of the synchronous action of several galectins. Therefore, we believe the company's technology has a competitive edge over single galectin-targeting small molecules, RNA-based drugs, and antibodies.

Role of Galectin Proteins

Galectin proteins, which are secreted from several cells in different tissues in the body, are a series of proteins that normally act as key mediators of disparate biologic and pathologic functions. Galectins can bind with very high affinity to galactose-containing glycoproteins on the surface of cells and in the extracellular matrix. In addition, their affinity for other galectins of similar type enables them to form lattice structures on cell surfaces, which can promote cell-to-cell and/or cell-to-matrix interactions. Galectins help the binding between cells, promote modulate cell signaling, and mediate cell-to-cell interactions. The presence of galectins is markedly increased in pathological reactions like fibrosis, cancer, and inflammation, and further promotes pathology. Under normal conditions, there are small amounts of galectins present in different cell tissues; however, in conditions involving immune regulation, inflammation, fibrogenesis, and tumor cell biology, a marked increase in secreted galectins is evident. Increased secretion could be an underlying cause of pathological conditions like fibrosis.

One of the core fundamental characteristics of the Galectin Therapeutics technology platform, in our view, is the focus on highly validated science. The main components of the data required to validate a suitable clinical target are as follows:

- Altered expression of the target protein or receptor of interest in pathological or disease conditions (in cases where a gain-of-function intervention is desired, the target of interest should be down-modulated in disease conditions, whereas in cases where a loss-of-function intervention is proposed, the expression of the target should be increased under disease conditions)
- Specificity of the target in question, such that intervention at the level of the target pathway is unlikely to have significant unforeseen or deleterious side effects; this can be proven through the use of transgenic animal models that delete the gene encoding the target (so-called knockout animals) or arrest its expression (if the target protein or receptor of interest is not necessary for normal development and function – e.g., knockout animals develop normally, have normal function and normal life span – then interference with it is considered less likely to cause side effects or have major safety issues stemming from systemic drug toxicity)
- Proof-of-concept for the therapeutic relevance of the target in animal models of disease through the existence of specific mutations that may cause aberrant function of the target in question, change its expression pattern, or ablate its expression altogether – such models must also mimic pathological and clinical hallmarks of the diseases of interest.

Table 3: Target Validation Criteria

Category of Evidence	1	2	3	4	5
Expression/ activity	Widely expressed/ broadly active	Expressed/active in at least the target organ	Expression/activity modulated in many patient tissues	Expression/activity modulated in effector tissues from patients	
Pharmacological experimental data on target modulation	*In vitro*, in cell lines causes relevant pathway modulation	*Ex vivo*, in native tissue causes relevant pathway modulation	In vivo activity mimics valid disease process/ therapeutic response	Target-based model of disease process responds to target modulation	
Non-human genetic models		Model organism has treatment-relevant phenotype	Rodent/primate has treatment-relevant phenotype	Human genetic mutations in an animal model mimics and mitigates disease	
Human genetics		Robust genetic association in one study	Robust genetic association replicated in many studies	Relationship between genotype and disease process established	
Clinical experience			Drug affects symptoms in patients but target not established	Efficacy and safety via target established in proof-of-concept (PoC) study	Efficacy and safety via target established in pivotal studies

Source: http://www.pdonlineresearch.org/

The table shown on the previous page describes the different criteria necessary for target validation. As one advances from left to right across the table, the level of validation is increased. While efficacy and safety have not thus far been established in clinical studies for galectin inhibitors, we note that every other level of validation has been attained. Accordingly, therefore, we consider the galectin inhibition hypothesis to be substantially validated. We are particularly convinced by three pieces of evidence – the phenotypic characteristics of the Galectin-3 knockout mice, which appear to be both developmentally and functionally normal, yet are resistant to fibrotic disease; the circumstances under which expression of Galectin-3 is up-regulated, which correlate with disease conditions; and the results obtained via administration of galectin-inhibiting-compounds to mice, which demonstrate reduction of fibrosis. We shall discuss each of these lines of evidence supporting the galectin hypothesis later in this section of the report.

Galectin Normal Function

The physiological role played by galectins remains poorly characterized. However, we believe that their function is of significant importance, partially because they are extraordinarily well-conserved from an evolutionary standpoint[1]. The presence of galectins in so many evolutionarily divergent species suggests that they participate in basic cellular functions. On the other hand, the evidence that there may be dozens of galectins within a single species suggests that they have evolved to participate in a variety of more specific functions. Indeed, there is abundant evidence that members of this family interact with glyco-conjugates on or around cells and influence adhesion, migration, chemotaxis, proliferation, apoptosis, and neurite elongation[2].

Even a single galectin can apparently affect cells in a variety of ways depending on the cell type and circumstances. For instance, galectin-1 can stimulate or inhibit cell proliferation and can either stimulate or inhibit cell adhesion to extracellular matrix constituents[3]. There is also evidence that galectins can simultaneously have distinct intracellular and extracellular functions. For instance, both galectin-1 and galectin-3 have been implicated in pre-mRNA splicing. There are more than 20 different types of galectins that have been characterized thus far. One reason why we are intrigued by the therapeutic potential of galectin function modulation is because it appears that – while widespread – these molecules are not all necessary for normal development and function.

Galectin-3, a beta-galactoside-binding animal lectin of 30 kDa, is one of the key galectin proteins. It comprises two domains: a carboxyl-terminal domain that contains the carbohydrate-binding region and an amino-terminal domain consisting primarily of tandem repeats of nine amino acids to cross-link both carbohydrate and non-carbohydrate ligands. Since Galectin-3 is one of the galectins being focused on by Galectin Therapeutics, we believe it is particularly important to highlight the fact that, while it has been implicated in a range of physiological processes, animal evidence appears to indicate that this member of the galectin family is not necessary for normal physiological development and function. The Galectin-3 knockout mouse develops normally, exhibits no aberrant behavioral characteristics, and possesses a normal life span[4]. Data has demonstrated that these mice exhibit accelerated lipid-induced atherogenesis, which could indicate a protective role for Galectin-3 in the trafficking of lipids. However, we believe that the phenotypic normality of the Galectin-3 knockout mice represents one crucial piece of evidence indicating that blocking the function of Galectin-3 in human subjects suffering from NASH could be beneficial without causing harmful side effects. Accordingly, therefore, the knockout mouse validates eligibility of Galectin-3 as a target.

[1] Cooper and Barondes. Glycobiology 9: 979-984 (1999)
[2] Puche *et al*., Developmental Biology 179: 274-287 (1996)
[3] Yamaoka *et al*., Journal of Immunology 154: 3479-3487 (1995)
[4] Iacobini *et al.*, Arteriosclerosis, Thrombosis and Vascular Biology 29: 831-836 (2009)

Galectins In Disease

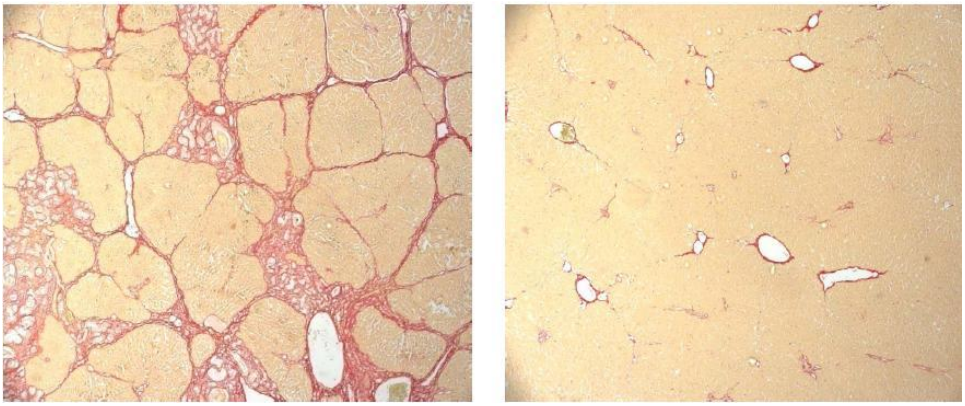
Galectin-3 has been shown to have a role in several biological processes, including cell proliferation, adhesion, and survival[5]. Research has shown that disruption of the Galectin-3 gene blocks myofibroblast activation and pro-collagen (I) expression *in vitro* and *in vivo*, followed by attenuation of liver fibrosis[6]. Preclinical data also strongly suggest that Galectin-3 is required for myofibroblast activation and matrix production, which eventually lead to liver fibrosis. Furthermore, it has been demonstrated that the selective ablation of Galectin-3 is protective in mouse models of liver fibrosis, including the diet-induced model of NASH[7]. Accordingly, in our view, there is strong evidence that Galectin-3 has a direct causal relationship with liver fibrosis.

Galectin Inhibitors

The company develops galectin inhibitors, which are carbohydrates with galactose residues that bind galectins. The backbones of galectin-targeting drugs are naturally-occurring carbohydrate polymers of varying molecular weights. Making modifications to the galactose residues on these polymers helps create compounds that bind to specific galectin proteins, which can then be used for the treatment of a particular disease. Multiple galectin proteins are bound by a single drug molecule. The company's drugs generally have a high molecular weight and, consequently, a long half-life and low toxicity (metabolize to CO_2 and H_2O), and are inexpensive to manufacture.

Galectin Therapeutics designs drug candidates that can bind and inhibit the functioning of secreted galectins. Scientists specializing in galectin science have hypothesized that the inhibition of such activities can alleviate and reverse the pathological conditions created in the presence of galectins. In the experiment shown below, which was performed by a researcher at Fudan University in Shanghai, China, rats suffering from extensive liver fibrosis induced by the sustained administration of a chemical toxin for eight weeks were treated with Galectin Therapeutics' lead drug candidate, GR-MD-02, for four weeks. The result was complete reversal of liver fibrosis, as seen is the pictures below. On the left, extensive fibrosis can be seen in a rat treated with the toxin. On the right, the fibrosis is entirely eliminated and there is normal tissue physiology. Although we cannot guarantee that the beneficial effects seen in animals will be replicated in humans, we believe that the evidence seen to date justifies classifying Galectin-3 as a validated target.

Figure 6: Galectin Blockade Inhibits Toxin-Induced Liver Fibrosis



Source: Ji-Yao Wang, Fudan University, Shanghai, China

[5] Henderson and Sethi. Immunology Reviews 230: 160-171 (2009)
[6] De Oliveira *et al*., Cytotherapy 14: 339-349 (2012)
[7] Iacobini *et al*., Journal of Hepatology 54: 975-983 (2011)

Overview of GM-CT-01

Potential exists for galectin inhibition to play an important role in cancer therapy, based on recently conducted scientific studies. Galectin proteins, particularly galectin-1 and galectin-3, have been shown to be highly expressed in the majority of cancers and have multiple roles in promoting cancer progression, including tumor cell invasion, metastasis, angiogenesis, and tumor evasion of the immune system.

Galectin Therapeutics believes that the unique applicability of the galectin-blocking technology to the enhancement of cancer immunotherapy effectiveness is directly linked to the role of galectins – such as Galectin-3 – in mediating cancer's ability to fool the immune system. Under normal circumstances, aberrantly growing cells such as those found in tumors ought to be recognized as abnormal by the body's immune system and thus eliminated; however, many cancers exhibit the ability to evade detection by the immune system and thus continue to grow and metastasize. If Galectin can conclusively show the synergistic effect of combining a galectin inhibitor with an immunotherapeutic agent for treatment of cancer, it could potentially revolutionize cancer treatment and significantly enhance the effectiveness of cancer immunotherapy agents.

GM-CT-01 has progressed in development for the therapy of colorectal cancer and is currently in a Phase 1/2 clinical trial as a combination therapy with a tumor vaccine in patients with advanced melanoma. The current developmental approach for GM-CT-01 is to enhance the activity of the immune system against the cancer.

We believe the potential exists for galectin inhibition to play a key role in the burgeoning area of cancer immunotherapy. For example, there have been two recent approvals of drugs for using the patient's immune system to fight cancer, Provenge (Dendreon; a dendritic cell tumor vaccine) and Yervoy (Bristol-Myers Squibb; a monoclonal inhibitor of CTLA-4, which activates cytotoxic T-cells). With many additional vaccines and immune stimulatory agents in development, industry analysts forecast that this market could grow to over $7 billion by 2015. Galectin Therapeutics aims to produce an effective galectin inhibitor that enhances the immune system's ability to fight cancer and, most important, that complements other approaches to this type of therapy.

The role of galectins in cancer immunotherapy can be understood through the "Galectin Effect", a recent discovery of how tumors avoid the body's own immune system. Galectin Therapeutics' current program to block the "Galectin Effect" is based on the research of Dr. Pierre van der Bruggen (of the Ludwig Institute of Cancer Research in Brussels, Belgium), demonstrating that galectin-3, which is produced by the vast majority of human cancers, binds to and blocks the actions of tumor-infiltrating T-lymphocytes, the major immune cell in the body's defense against cancers. Based on these results, we believe that the body's immune cells cannot attack and kill tumor cells in the presence of galectins. Using this approach, the mechanism of action for GM-CT-01 seeks to block galectins and, in turn, restore the ability of the T-lymphocytes to kill tumor cells.

The company recently initiated a Phase 1/2 clinical trial of GM-CT-01 in Belgium in combination with a tumor vaccine in patients with advanced melanoma, a deadly skin cancer. The Belgian Federal Agency of Medicine and Health Products (FAMHP) granted approval for this clinical trial, which is being conducted at three centers in Belgium and one in Luxembourg. The operational conduct of the trial is under the control of the Cancer Centre at the Cliniques Universitaires Saint-Luc and the Ludwig Institute for Cancer Research. The study has been initiated and patients are beginning to be enrolled. We expect the first stage of this trial (involving 12 evaluable patients) to be completed within a year of enrollment of the first patient and that it will provide data that could deliver an indication of efficacy. Depending on the results of Stage 1, the study could continue enrollment to complete Stage 2 (46 total patients), initiate a new Phase 2 trial

based on positive results or be halted because of lack of efficacy. Stage 1 of the trial is being funded by the Cancer Centre at the Cliniques Universitaires Saint-Luc and Stage 2 requires funding from Galectin Therapeutics, currently estimated at approximately $1 million. Positive results from this study could indicate that this approach of inhibiting the "Galectin Effect" would be an enabling technology for therapy in other tumor types. The Phase 1/2 clinical trial in Belgium is not being conducted under an FDA-approved IND, but there is an open IND on file with the FDA for GM-CT-01 that would permit U.S. clinical development to be conducted.

There are two additional pathways for the development of GM-CT-01 for use in the treatment of cancer. GM-CT-01 was found to be generally safe when studied in a Phase I clinical trial in end-stage cancer patients with multiple tumor types alone and in combination with 5-fluorouracil (5-FU), which is an FDA-approved chemotherapeutic agent used for treatment of various types of cancer. Three Phase 2 studies were conducted, but were only partially completed due to financing issues. DAVFU-003 was a Phase 2 multi-center open-label trial in end-stage, line 3/4 metastatic colorectal cancer patients, who were treated with a combination of GM-CT-01 and 5-FU. In the 20 enrolled patients, the median survival was 6.7 months and there was a notable reduction in the expected adverse events related to 5-FU therapy.

DAVFU-003 was terminated in 2007. Although only partially completed, when compared to historical controls, the data collected for DAVFU-003 suggested a favorable effect of the therapy, since the controls had an overall survival of 4.6 months. DAVFU-006 was a Phase 2, open-label clinical trial in line 1 patients with locally advanced and unresectable or metastatic colorectal cancer (who were unable to tolerate intensive chemotherapy), who were treated with a regimen of GM-CT-01, 5-FU, leucovorin, and Avastin®. Ten patients were enrolled in this study. DAVFU-006 was terminated in March 2010. Finally, DAVFU-007 was a Phase 2, multi-center, open-label clinical trial to evaluate the efficacy and safety of GM-CT-01 in combination with 5-FU when administered as first line chemotherapy in patients with advanced biliary cancer. Seventeen patients were enrolled in this study. This study was stopped in March 2010.

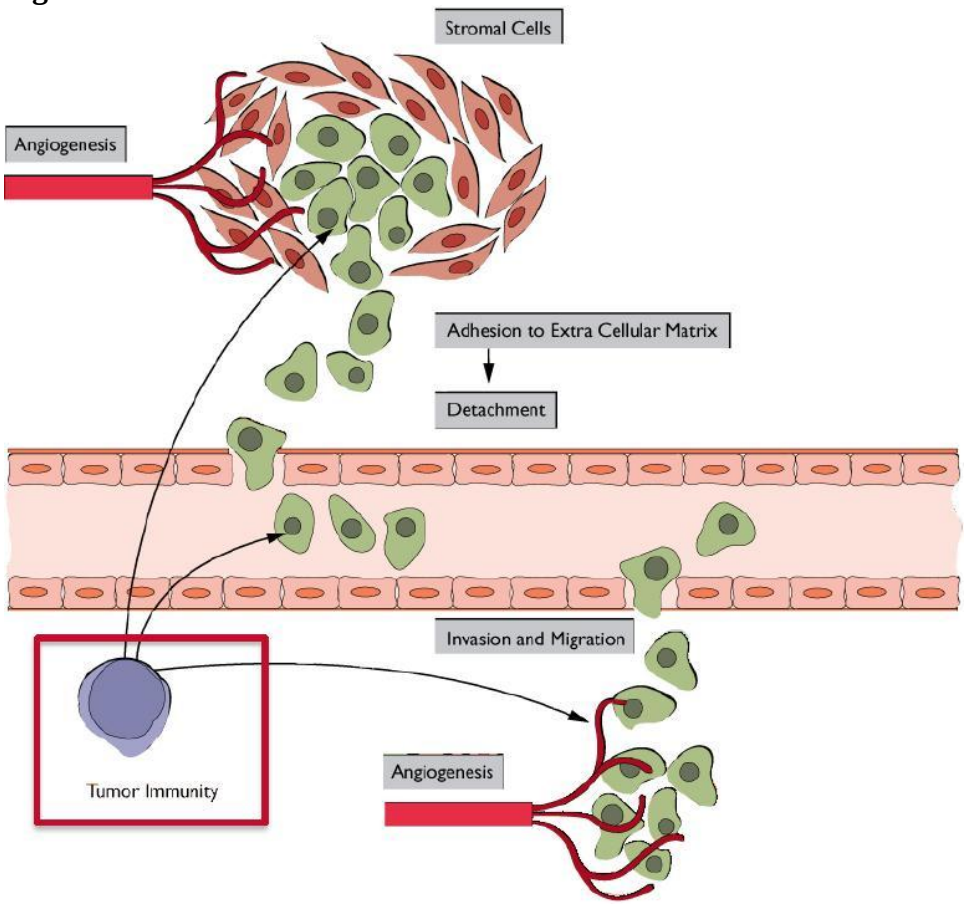
It was notable in these four studies that, when the results of adverse events were pooled, there appeared to be a marked reduction in the severity of 5-FU related adverse events when compared to historical controls. In order to examine 5-FU related side effects in patients receiving GM-CT-01 in all of the clinical trials, a post-hoc analysis was conducted of adverse events typically related to 5-FU including, diarrhea, nausea and vomiting, mucositis and neutropenia/leukopenia. Studies for comparison to Galectin Therapeutics' data were culled from the literature, providing a broad spectrum of 1,128 patients treated with 5-FU. Comparison of adverse events between the literature-derived patients and the 57 patients in the Galectin clinical trials (conducted while the company was still known as Pro-Pharmaceuticals) who received 5-FU with full dose GM-CT-01 demonstrates that patients in these trials had a markedly lower grade: 3/4 adverse events for all of the 5-FU related toxicities. These data suggest that GM-CT-01 may ameliorate toxicities related to 5-FU, which are important limiting events in cancer chemotherapy.

Based on these completed Phase 1 and partially completed Phase 2 clinical trials, Galectin Therapeutics is currently exploring two additional potential indications for the use of GM-CT-01 in combination with cancer chemotherapy:

- The firm is currently seeking potential strategic partners to assist in researching the use of GM-CT-01 in the amelioration of 5-FU related side effects.
- Galectin is seeking to explore the possibility of combining GM-CT-01 with various forms of cancer immunotherapy in order to assess how much the drug could enhance the efficacy of such therapeutic approaches.

The expression of galectins by tumors allows the tumors to influence the nature of the extracellular matrix in their immediate surroundings. This could enable the tumors to enhance their ability to invade surrounding tissues, thus increasing the efficiency of metastasis. In addition, angiogenesis can be increased, enabling tumors to enhance the blood supply that is crucial to fueling their continued growth.

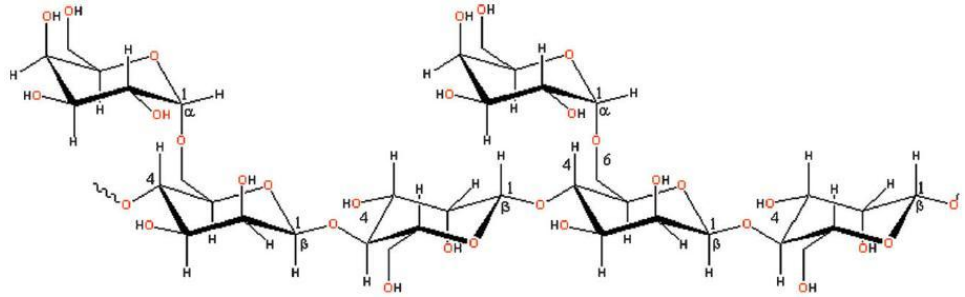
Figure 7: Galectin Function In Cancer



Source: Galectin Therapeutics, Inc.

Galectin's lead oncology-focused drug candidate, GM-CT-01, is a galactomannan with a backbone composed of (1 → 4) linked β-D-mannopyranosyl units, to which single α-D-galactopyranosyl residues are periodically attached via a (1 → 6)-linkage, with an average repeating unit of 17 β-D-mannopyranosyl units and 10 α-D-galactopyranosyl units and an average polymeric molecule containing about 12 such repeating units.

Figure 8: GM-CT-01 Repeat Unit Chemical Structure



Source: Galectin Therapeutics, Inc.

Cancer Immunotherapy Background

The underlying mechanism upon which cancer immunotherapy is based depends upon the body recognizing, or rather being taught, that cancer cells have unique features that distinguish themselves from normal cells in the body. Tumor-associated antigens (TAAs) are markers (typically proteins) present either only in cancer cells or in a much higher concentration in cancer cells than in normal cells. Thus, many therapies target TAAs as a mechanism to attack cancers. HER2/neu, for example, is a well-known TAA that has been found on breast, ovarian, and colon cancers. Therefore, a drug targeting HER2/neu may serve the purpose of treating these three indications and possibly more.

Several companies mentioned below are attempting to use immunotherapy agents to target TAAs. Unlike conventional cancer drugs, which directly attack cancers, cancer immunotherapies stimulate patients' immune systems to inhibit cancer growth. Activating the patients' immune systems to target TAAs is highly specific, typically durable, and usually much less toxic than conventional cancer therapies. Not only are the conventional chemotherapy drugs in use today highly cytotoxic (i.e., they are cellular poisons that kill both normal and abnormal cells indiscriminately), but they also weaken the patients' immune systems, muting their own immune responses and making the anti-cancer fight wholly dependent on the toxic drug, preventing the body from defending itself. Additionally, the subjects are more susceptible to secondary infections by viruses or bacteria during their weakened states.

It is important to note that immunotherapeutic approaches that cover a broad range of TAAs or specific TAAs that are found on a variety of different cancers have the potential to be used in many different cancer indications. The marketing potential for these types of drugs is phenomenal. From an investor's standpoint, the upside potential in an early-stage biotech developing a drug with such a range of TAAs could be very lucrative. The following section is a summary of several healthcare firms with immunotherapy drugs at varying stages of development. We believe early success in one indication would quickly legitimize these drugs for other indications expressing the same TAAs.

- Dendreon's Provenge (sipuleucel-T) was the first cancer immunotherapy to receive FDA approval in 2010 for the treatment of prostate cancer. Provenge targets prostatic acid phosphatase (PAP), a TAA commonly found on prostate cancer cells but not on other cells. Dendreon's second product currently in development is DN24-02, which targets HER2/neu, a TAA widely found on numerous cancers, including breast, ovarian, and colorectal malignancies. DN24-02 is currently in a Phase 2 trial for urothelial carcinoma and is still actively recruiting patients.
- ImmunoCellular Therapeutics' lead product, ICT-107, is currently recruiting for a Phase 2 trial to treat newly diagnosed glioblastoma multiforme (GBM), a common form of brain cancer. The firm has enrolled roughly 200 subjects at 25 clinical sites. ICT-107 targets six TAAs including TRP-2, MAGE-1, HER-2, IL-13 receptor a2, gp100 and AIM-2. The Phase 1 data showed median overall survival (OS) for the patient set of 38.4 months with median progression-free survival of 17 months. The three-year OS for the patient set was 55% with 6 patients having no recurrence of the tumor after three years. With the current prognosis for GBM at about 3 months median OS with no treatment and about 15 months for those receiving current standard of care, the implications are profound. A second product, ICT-140, targets EphA2, which is involved in ovarian, breast, colorectal, prostate, and lung cancers, as well as aggressive melanomas and mesothelin, which is found in high concentrations in mesothelioma, in addition to ovarian and pancreatic cancers. Their third cancer vaccine, ICT-121, targets CD133, which is a marker for cancer stem cells, and can be potentially used as a universal cancer therapy. The company plans to initiate Phase 1 trials for ICT-140 and ICT-121 this year. Several catalysts are

expected near-term, such as the completion of enrollment in the ICT-107 Phase 2 trial, with interim data analysis in late 2012 or early 2013.

- Galena Biopharmaceuticals recently initiated a Phase 3 trial for NeuVax in treating breast cancer patients. Like Dendreon's and ImmunoCellular Therapeutics' cancer vaccines, NeuVax also targets the HER2/neu antigen. The upside in ImmunoCellular's ICT-107 is due to its orphan drug status for GBM along with its potential for multiple indications likely coming. However, we believe Galena's NeuVax breast cancer indication has substantial upside, with the potential target market representing an indication in which almost 300,000 new cases are diagnosed every year. A 53-patient subset in the NeuVax Phase 2 trial that received a booster vaccination once every six months after the initial treatment protocol yielded a dramatic increase in efficacy as evident in a statistically significant disease-free survival rate of 95.9% vs. 79.7% in the control group ($p = 0.016$). The projected Phase 3 trial is based on the same type of booster schedule and the company is hopeful that these pivotal results will be comparable. The firm's second cancer vaccine, FBP, which is in Phase 1 development, targets folate binding protein (FBP). FBP is found on 90% of ovarian and endometrial cancers as well as 20-50% of lung, breast, colon, and renal cancers.
- Celldex Therapeutics' lead product, rindopepimut, which targets variant III of the epidermal growth factor receptor (EGFRvIII), is currently in a Phase 3 trial in patients with newly diagnosed gliblastoma multiforme (GBM) – one of the most aggressive and difficult-to-treat forms of brain cancer – and a Phase 2 trial in patients with recurrent GBM. The EGFRvIII target is expressed in about one third of GBM patients but also reported in lung, breast, ovarian, and prostate cancers. Rindopepimut was originally partnered with Pfizer earlier, but the rights were returned to Celldex in 2011 for unspecified reasons. Celldex's pipeline includes another product, CDX-1401, in a Phase 1/2 trial for several cancers. CDX-1401 is an antibody targeting dendritic cells linked to the TAA, NY-ESO-1. This TAA is widely expressed on several tumors: approximately a quarter of all lung, prostate, melanoma, bladder, esophageal, and ovarian cancers.
- Oncothyreon's Stimuvax, which is partnered with Merck KGaA and in pivotal Phase 3 trials for non-small cell lung cancer, targets the antigen mucin 1 (MUC1). MUC1 is found on more than 90% of breast carcinomas and is also associated with colon, ovarian, and pancreatic, as well as lung cancers. The Stimuvax trials were halted several years ago due to a case of encephalitis observed in a separate study, but recent interim analysis showed that the Phase 3 trials should continue to completion.

With the FDA approvals of Provenge and, more recently, Bristol-Myers Squibb's Yervoy (ipilimumab), cancer immunotherapy has been validated as a legitimate treatment for cancer. There is sufficient scientific rationale that cancer immunotherapies can be developed for many indications. We think the success of the Provenge and Yervoy drugs will likely only be the beginning given the technology is becoming more robust, more antigens are being discovered, the drug manufacturing process is being more fully perfected, and investors are now more willing to commit capital to the cancer immunotherapy domain because of recent successful product approvals in this domain.

The addressable cancer indications are numerous, not only because of the antigens presented in the different cancers, but also due to the ways the drugs can be administered. Future possibilities include more vaccines given as adjuvants to current standards of care of chemotherapy, hormone therapy, radiotherapy, and resection. Some of the more aggressive immunotherapy agents in development could also potentially be given as front-line treatments in which they would be the actual agents to directly attack the cancers initially. These clinical studies may also give rise to true cancer vaccines capable of teaching immunity systems in patients with risk factors for developing certain types of cancers to fight cancers at their earliest stages of development, thereby averting a cancer diagnosis in the first place.

GM-CT-01 Market Model

We have modeled sales of GM-CT-01 in colorectal cancer and malignant melanoma, indications for which we expect the drug to be used as an adjunct to immunotherapy. One of the main potential advantages of this agent, as we have noted previously, could be its ability to amplify the effectiveness of both passive as well as active immunotherapy.

The colorectal cancer (CRC) market was estimated to be worth about $6.5 billion in 2011 with a projected compound annual growth rate (CAGR) of 9.8% and a projected market value that exceeds $11.6 billion in 2016. The replacement of chemotherapies by target therapies in adjuvant treatments and neo-adjuvant therapies has boosted the growth of the CRC therapeutics market in recent years. Current statistics for product candidates in development for the treatment of colorectal cancer demonstrate a healthy level of activity in this area, in our view, with roughly 160 molecules in various stages of clinical development. There are more than 77 first-in-class molecules in different stages of clinical development. These molecules have a distinct advantage over the current market players and display robustness in the CRC pipeline. Chemotherapy with or without combinational targeted therapies may improve the symptoms and prolong the lives of cancer patients. Huge research activities in combinational targeted therapies are ongoing. GM-CT-01, with its prior clinical data showing improvement of side effects of chemotherapy drugs such as 5-FU, could potentially be capable of substantial clinical utility in this indication.

The incidence of metastatic melanoma has increased over the last three decades, and the death rate continues to climb faster than that of most other cancers. According to the American Cancer Society, there were approximately 75,000 new cases of melanoma in the U.S. in 2011, and roughly 10,000 melanoma-related deaths. Melanoma accounts for only about 3% of all skin cancers, but 80% of skin cancer deaths, and is extremely difficult to treat once it has spread beyond the skin to other parts of the body (metastasized). Very few treatment options currently exist for people with metastatic melanoma. We note that recent drug approvals in metastatic melanoma have been extremely rapid, with agents such as Yervoy (ipilimumab) and Zelboraf (vemurafenib) being fast-tracked. Indeed, Zelboraf was approved by the FDA within less than three months, and in Canada within a month of submission.

We have estimated relatively conservative pricing for GM-CT-01 in cancer, with $25,000 being the average starting price in the U.S. and $15,000 being the price in Europe. In our market model, we project launch of the drug in 2015 in the U.S. and by 2016 in Europe. The agent could initially be launched in treatment of metastatic melanoma, wherein development may be relatively rapid, involving a single pivotal trial since the disease constitutes such a significant unmet medical need.

Approximately one out of every four deaths in 2011 could be attributed to some form of cancer. Between synthetic vaccines, immune modulators, immunotherapy antibodies and cell based investigational agents, the cancer immunotherapy field has a forecasted market potential of roughly $7 billion by 2015. Additionally, recent approval by the FDA of two immunotherapeutic drugs has sparked new interest in these technologies; there are already an estimated 150 potential products being studied, many of which are currently undergoing Phase 2 and Phase 3 clinical trials.

Accordingly, we believe that Galectin Therapeutics could have a vast array of potential partners to collaborate with in developing GM-CT-01 as an adjunct to cancer immunotherapy. One of the principal advantages of this drug, as we have previously noted, is the fact that it has a benign safety profile and also appears to reduce the toxicity associated with chemotherapy. Therefore, we believe that it is likely to be used widely if the hypothesis that it enhances cancer immunotherapy can be proven conclusively.

Table 4: GM-CT-01 Estimated Sales – Oncology Indications Market Size Model

	2012	2013	2014	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024	2025	2026	2027	2028	2029	2030
US Population																			
Colorectal cancer	115,000	120,750	126,788	133,127	139,783	146,772	154,111	161,817	169,907	178,403	187,323	196,689	206,523	216,850	227,692	239,077	251,031	263,582	276,761
Malignant melanoma	76,000	79,800	83,790	87,980	92,378	96,997	101,847	106,940	112,287	117,901	123,796	129,986	136,485	143,309	150,475	157,999	165,898	174,193	182,903
GM-CT-01 Penetration (colorectal cancer)	0%	0%	0%	0%	1%	2.5%	4%	7%	9%	12%	15%	18%	15%	14%	11%	9%	5%	2%	1%
GM-CT-01 Penetration (melanoma)	0%	0%	0%	1%	3%	5%	7%	9%	11%	15%	21%	25%	30%	35%	31%	23%	18%	7%	3%
Patients on GM-CT-01				880	4,169	8,519	13,294	20,952	27,643	39,093	54,096	67,900	71,924	80,517	71,693	57,857	42,413	17,465	8,255
Cost per patient ($)				25,000	25,750	26,523	27,318	28,138	28,982	29,851	30,747	31,669	32,619	33,598	34,606	35,644	36,713	37,815	38,949
US GM-CT-01 sales ($ MM)				22	107	226	363	590	801	1,167	1,663	2,150	2,346	2,705	2,481	2,062	1,557	660	322
European Population																			
Colorectal cancer	95,000	97,375	99,809	104,301	109,516	114,992	120,741	126,778	133,117	139,773	146,762	154,100	161,805	169,895	178,390	187,309	196,675	206,508	216,834
Malignant melanoma	65,000	68,250	71,663	75,246	79,008	82,958	87,106	91,462	96,035	100,836	105,878	111,172	116,731	122,567	128,696	135,130	141,887	148,981	156,430
GM-CT-01 Penetration (colorectal cancer)	0%	0%	0%	0%	0%	1.2%	2.5%	5%	9%	11%	12%	15%	16%	12%	9%	6%	4%	2%	1%
GM-CT-01 Penetration (melanoma)	0%	0%	0%	0%	1%	2.5%	4%	7%	11%	14%	18%	21%	25%	21%	18%	16%	12%	9%	3%
Patients on GM-CT-01				0	790	2,074	3,484	6,402	10,564	14,117	19,058	23,346	29,183	25,739	23,165	21,621	17,026	13,408	4,693
Cost per patient ($)					15,000	15,450	15,914	15,450	15,914	16,391	16,883	16,391	16,883	17,389	16,883	17,389	17,911	17,389	17,911
European GM-CT-01 sales ($ MM)				0	12	32	55	99	168	231	322	383	493	448	391	376	305	233	84
Global GM-CT-01 sales ($ MM)				**22**	**119**	**258**	**419**	**688**	**969**	**1,398**	**1,985**	**2,533**	**2,839**	**3,153**	**2,872**	**2,438**	**1,862**	**894**	**406**
Galectin Therapeutics Net Income ($ MM)				**2**	**14**	**39**	**67**	**110**	**165**	**238**	**337**	**431**	**483**	**536**	**488**	**414**	**317**	**152**	**69**

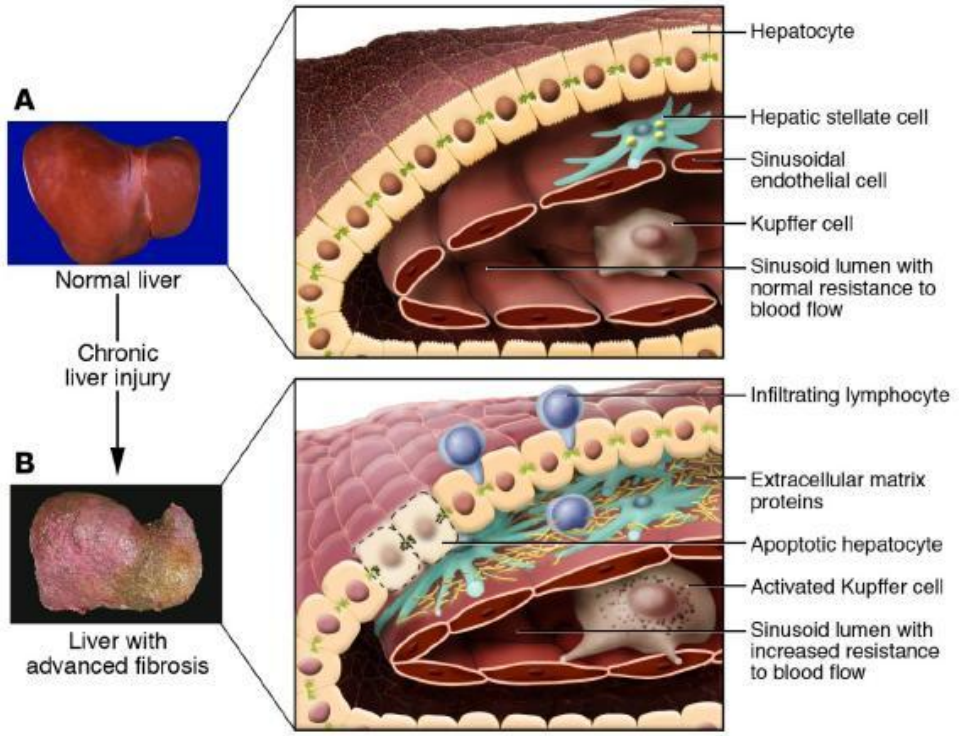
Source: Company Reports and Aegis Capital Corp. estimates

Liver Fibrosis Indication Overview

Liver fibrosis is the scarring process that represents the liver's response to injury. In the same way as skin and other organs heal wounds through deposition of collagen and other matrix constituents, the liver repairs injury through the deposition of new collagen. Over time, this process can result in liver cirrhosis, in which the architectural organization of the functional units of the liver becomes so disrupted that blood flow through the liver and liver function become disrupted.

Once cirrhosis has developed, serious complications of liver disease may occur, such as portal hypertension, liver failure, and liver cancer. Risk of liver cancer is increased once cirrhosis develops and cirrhosis should be considered a pre-malignant condition. Cirrhosis and liver cancer are now among the top ten causes of death worldwide and, in many developed countries, liver disease is now one of the top five causes of death in middle age. In the U.S. alone, liver cirrhosis is now ranked among the top ten causes of death, and roughly 30,000 individuals die from this condition each year. Advanced cirrhosis carries a mortality rate of 34% – 66%, depending on the nature of the condition.

Figure 9: Liver Fibrosis



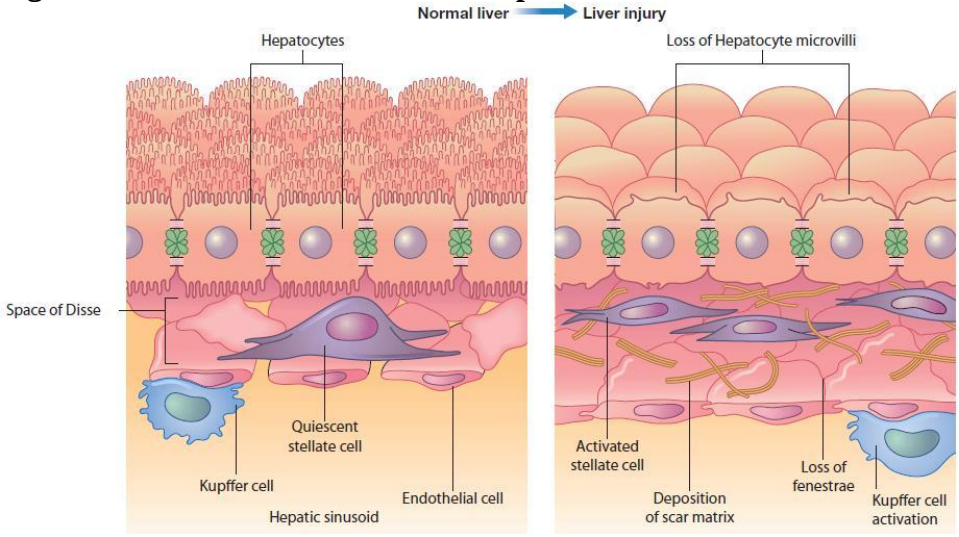
Source: Journal of Clinical Investigation 115: 209-218 (2005)

The main liver cells that produce collagen-based matrix are hepatic stellate cells (HSCs)[8]. This resident cell population exists in a resting phenotype as the body's major store of vitamin A. However, upon activation, they transform to adopt a myofibroblast phenotype that can secrete collagen. This fibrous tissue can then be remodeled through digestion of matrix by matrix metalloproteinases (MMPs). In turn, the digestion of matrix is checked through the inhibition of MMPs by tissue inhibitors of matrixmetaloproteinases (TIMPs), of which TIMP-1 is of major importance. Liver fibrosis, previously thought to be merely the accumulation of scar tissue, is now recognised to be a dynamic process that can progress or regress over periods as short as a few months.

[8] Sanchez-Valle *et al*., Current Medicinal Chemistry [e-publication ahead of print] (2012)

The figure below shows how normal liver cellular structure changes over time as chronic liver injury occurs. Cirrhosis of the liver is at its heart a response to chronic tissue injury, generally due to toxic insult (e.g. via excessive chronic consumption of alcohol).

Figure 10: Cellular Mechanism of Hepatic Fibrosis



Source: Scott Friedman, Mount Sinai School of Medicine, New York, NY

All chronic liver diseases (CLD) can lead to liver fibrosis[9]. The principal causes of CLD have been chronic viral hepatitis B (CHB) and alcoholic liver disease (ALD). While rates of alcoholism and ALD are falling in many countries, hazardous drinking amongst young people is resulting in alarming rates of ALD in several northern European countries. Over the last few decades, two other diseases have emerged to make a major contribution to the burden of CLD. Chronic hepatitis C (CHC) and non-alcoholic fatty liver disease (NAFLD), also known as non-alcoholic steatohepatitis (NASH), are recognised to have already had a major impact on CLD incidence. Hepatitis C virus (HCV) is transmitted in blood and blood products through unsafe injection practices and use of infected blood products. The world prevalence of CHC may well exceed 200 million people. In the developed world, with rapidly increasing rates of obesity, NAFLD is considered to represent a major cause of fibrosis. Although only 20% of patients with NAFLD develop significant fibrosis, due to the vast prevalence of the at-risk overweight population, we think NAFLD / NASH may give rise to an epidemic of liver fibrosis.

There is a critical need for better data about natural history, risk factors for fibrosis, and the rate of fibrosis progression in NASH, which are issues being addressed in several multi-center studies. Patients with only steatosis and no inflammation appear to have a benign course when followed for up to 19 years; however, it is unclear whether this type of lesion is completely distinct from steatohepatitis, or instead represents a precursor of NASH. Hepatitis C viral infection-related fibrosis progression rates were underestimated shortly after the virus was first identified, as many patients had a relatively early fibrosis stage. With continued infection, however, a sizeable fraction progressed to advanced disease. In a parallel situation, the obesity epidemic in the US and the developed world is only now being fully appreciated, and a threshold level of obesity may have only begun to confer a risk of liver disease that may become clinically significant in the next decade. It is widely accepted among hepatologists nowadays that a body mass index (BMI) of >28 kg/m^2 correlates with severity of fibrosis and risk of cirrhosis[10].

[9] Poynard *et al*., BMC Gastroenterology 10: 40-53 (2010)
[10] Rockey and Friedman, Pathophysiology of the Liver (2006)

Liver Fibrosis Competitive Landscape

In liver fibrosis, we have determined that competition for Galectin Therapeutics is limited. We are not particularly concerned about competition in this area, as: (1) we believe several drugs will likely be used in combination for the treatment of liver fibrosis; and (2) because the company's galectin inhibitors are mostly broken down into water and carbon dioxide (being largely constructed of carbohydrate chains), the safety and tolerability (and, therefore, the combinability) of Galectin Therapeutics' drugs, in our opinion, will likely be substantially superior to that achieved by drugs belonging to the firm's direct competitors. The following paragraphs identify the company's competitors and their current activities involving the treatment of liver fibrosis:

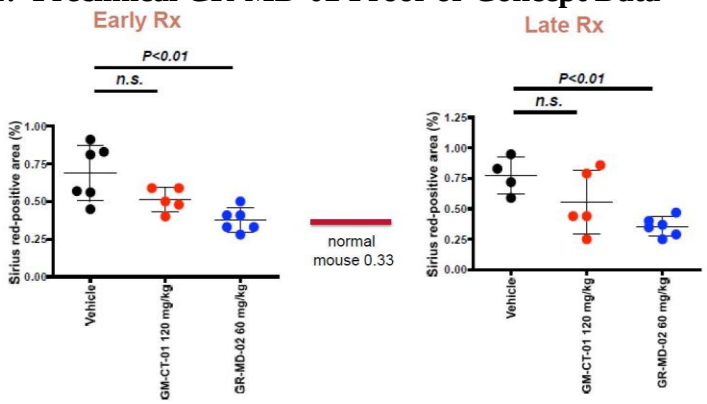
- FibroGen: Based in San Francisco, FibroGen is a private company. FibroGen is developing FT-3019, a monoclonal antibody to connective tissue growth factor (CTGF). CTGF has been shown to have a role in liver fibrosis. CTGF is known to be over-expressed in fibrosis; elevated levels of CTGF have been observed in patients with various chronic liver diseases, including viral and alcoholic hepatitis, NASH, biliary atresia, and idiopathic portal hypertension. The company started a Phase 2 trial in Asian patients with Hepatitis B and fibrosis in October 2010 and is expecting to complete the trial in December 2013. We know that CTGF knockout mice demonstrate severe vascular and developmental effects, showing a broad function for this factor. Therefore, complete CTGF inhibition could lead to severe unwanted side-effects. Of FibroGen's four areas of disease development, fibrotic diseases seem to be the least significant.
- ViroBay: A private biotechnology company based in Menlo Park, CA, which is developing an oral cathepsin B inhibitor called VBY-376. This is one of six drugs ViroBay has in clinical development. With this drug candidate, the company has shown, post-treatment, a reduction in collagen accumulation in toxin-induced fibrosis in rats. There is some evidence from knockout mice models that Cathepsin B has a role in liver cell apoptosis and injury. The company is currently developing this drug in Phase I trials, which are in the process of completion. However, since this is a small molecule drug candidate, we believe there is a higher probability of toxicity issues because of off-target effects, as well as metabolites.
- Angion Biomedica Group: A privately-held firm that is developing Hepatocyte Growth Factor (HGF) for the treatment of fibrosis; this is one of six targeted technologies the company is working on. Angion is expected to begin Phase 1 studies soon. Preclinical data from this drug candidate is not yet public, but we are assuming that positive animal model results prompted the company to progress to Phase 1 studies. We believe there could be a significant side-effect issue that might affect the progress of this drug candidate, as promoting the uncontrolled or excessive growth of hepatocytes can potentially lead to hepatocellular carcinoma (HCC), a major complication of cirrhosis in the first place.
- Intercept Pharmaceuticals: This privately-held firm has completed Phase 2 trials with an FXR agonist in primary biliary cirrhosis (PBC), which showed efficacy with the primary endpoint of reduction in alkaline phosphatase. Since there is little evidence of a correlation between alkaline phosphatase levels and fibrosis, we are skeptical of efficacy. A Phase 2 trial in patients with Type 2 Diabetes/NAFLD is ongoing.
- 7TM Pharma: Another privately-held firm that claims to be working on the treatment of liver fibrosis using, of all things, cannabinoid receptor modulation as a therapeutic strategy. In 2009, 7TM Pharma successfully conducted and completed a Phase 1 clinical trial with TM38837, which was discovered internally at 7TM Pharma and is being developed principally for treatment of obesity and related metabolic disorders. The Phase 1 clinical trial was a first-in-man single ascending dose study in healthy male subjects with additional cohorts in other populations. The aim of the study was to determine the safety and tolerability of a range of single ascending doses of

TM38837 and, additionally, to describe the pharmacokinetic properties of the compound. TM38837 is a first-in-class, second-generation CB1 receptor antagonist. It was designed to circumvent the CNS side effect profile displayed by the first generation CB1 receptor antagonists by homing in on peripherally located CB1 receptors in the body. This is in contrast to the first-generation CB1 receptor antagonists, which targeted CB1 receptors in the CNS. Although clinically effective, these candidates had unfavorable CNS side effects, including depression and anxiety.

Some other companies and private investigators are working on liver fibrosis drug candidates, but we believe those drugs are less promising than the ones we mention above. Furthermore, although several drugs could potentially be utilized for the treatment of liver fibrosis, approvability, in our opinion, is a much more important driver of valuation than a drug candidate's competitive profile. In any case, we believe that Galectin has certain key advantages over its competitors:

1) Apart from Cathepsin B knockouts, there is little evidence to suggest that any of the other candidates being developed are working on a pathway that is critical in the development of liver fibrosis. Compared to its competitors, we believe Galectin Therapeutics has the best scientific proof that it is working on the right target.
2) Since Galectin Therapeutics' drugs have metabolites that are easily excreted from the body, and because we know the company's drugs have a very safe profile from Phase II clinical trials, we believe Galectin Therapeutics holds a significant safety advantage over its peers. In our opinion, this advantage will likely become even more meaningful if and when the drug candidates are tested in less and less severe patients, and these drug candidates are dosed chronically.
3) Galectin Binding: Galectin Therapeutics' carbohydrate drug candidates avidly bind to multiple galectin molecules per single molecule of drug and bind both Galectin-1 and Galectin-3, the two most prominent galectins involved in pathological processes. These drug candidates selectively target extracellular galectins.
4) Because of their high molecular weight and low blood metabolism, these candidates circulate in the blood for a prolonged period of time. Below are preclinical data on GR-MD-02's therapeutic activity in a mouse model of liver fibrosis.

Figure 11: Preclinical GR-MD-02 Proof-of-Concept Data



Source: Galectin Therapeutics

We note that there is one other notable entrant in the galectin inhibition sector – the reinvented La Jolla Pharmaceutical Co., which in mid-April 2012 acquired the principal technology asset of the privately-held firm Solana Therapeutics, based in San Diego, CA. According to the companies, Solana's lead molecule – GCS-100 – is a first-in-class inhibitor of Galectin-3. La Jolla Pharmaceutical Co. currently holds the global development and commercialization rights to this agent. In connection with the acquisition, George Tidmarsh, the former CEO of Solana, was appointed as La Jolla's Chief Executive Officer. The terms of the transaction were not disclosed.

While we are skeptical of the claim that GCS-100 is a "first-in-class" molecule, we note that La Jolla Pharmaceutical Co.'s development plans for the agent do not encompass liver fibrosis *per se*, though the firm has acknowledged potential applicability of GCS-100 in this domain. Thus far, La Jolla Pharmaceutical Co. appears more focused on renal fibrosis – particularly end-stage renal disease (ESRD) – and the targeting of its new lead compound towards this arena. Therefore, we do not consider La Jolla Pharmaceutical to be a direct competitor to Galectin Therapeutics at this time; however, this could change if La Jolla Pharmaceutical elected near-term to start a clinical program in liver fibrosis. We would note that La Jolla Pharmaceutical Co. itself has an extremely checkered history, having previously failed very publicly with the anti-lupus medication Riquent – a setback that nearly drove the firm out of existence. In addition, we note that Tang Capital Management – an institutional investment firm headed by Kevin Tang, a former biotech sell-side analyst with Deutsche Bank Alex. Brown – has had an interest in galectin-targeting technologies for some time and is currently the most substantial investor in La Jolla Pharmaceutical Co., having driven the transaction with Solana Therapeutics.

In essence, the compound designated GCS-100 is a chemically modified citrus pectin of high molecular weight. It was assessed in a number of Phase 1 and Phase 2 clinical trials in cancers with little effect. Solana Therapeutics claims that this compound binds to galectin-3. In our conversations with Galectin Therapeutics management, it opined that its lead anti-fibrotic compound, GR-MD-02, is superior from a manufacturing standpoint as well as from a safety perspective. GCS-100 had fairly severe side effects in preclinical and human studies, including, in particular, leukocytoclastic angiitis, which required steroid therapy. This may be acceptable in cancer, but not in the treatment of fibrosis, which would necessitate chronic dosing. Galectin has not seen anything like these kinds of side effects with much higher doses of GR-MD-02 than were used by Solana Therapeutics in its development work with GCS-100.

We note that La Jolla Pharmaceutical Co. may be pursuing end-stage renal disease partially because of a recent, highly lucrative transaction inked between Abbott Laboratories and a privately-held firm, Reata Pharmaceuticals. In December 2011, these firms announced that they had entered into a worldwide collaboration agreement to jointly develop and commercialize Reata's portfolio of second-generation oral antioxidant inflammation modulators (AIMs). This agreement is in addition to the partnership between the two companies announced in September 2010 in which Reata granted to Abbott exclusive rights to develop and commercialize its lead AIM compound, bardoxolone methyl, outside the U.S., excluding certain Asian markets. The partnership deal inked in late 2011 is a global agreement and includes a large number of molecules in a broad range of therapeutic areas, including pulmonary, central nervous system disorders, and immunology. Abbott and Reata plan to equally share costs and profits for all new AIMs in all newly licensed indications except for rheumatoid arthritis and select other autoimmune diseases, in which Abbott assumes 70% of costs and profits and Reata assumes 30%. The deal also involves collaboration in early-stage R&D activities. Abbott made a one-time license payment of $400 million to Reata. The companies expect the first compound in this collaboration to enter into human clinical trials in 2012.

AIMs are potent activators of the transcription factor Nrf2. Activation of Nrf2 promotes the production of a wide range of antioxidant, detoxification, and anti-inflammatory genes. Activation of Nrf2 also inhibits NF-κB, a transcription factor that regulates many pro-inflammatory enzymes. Suppression of Nrf2 and activation of NF-κB have been associated with numerous chronic diseases, including multiple sclerosis, rheumatoid arthritis, chronic kidney disease, neurodegenerative disease, and COPD. Therefore, agents that activate Nrf2 and inhibit NF-κB may be beneficial in the treatment of these chronic diseases. Reata and Abbott are currently conducting the BEACON study, a multi-national Phase 3 clinical trial of bardoxolone methyl in patients with stage 4 chronic kidney disease and type 2 diabetes.

GR-MD-02 Market Model

We have modeled sales of Galectin Therapeutics' proprietary drug candidate GR-MD-02 in the U.S. and European markets, assuming approval and launch for treatment of non-alcoholic steatohepatitis (NASH). Although we believe that the drug could have important applicability in alcoholic cirrhosis of the liver as well as fibrosis of other organs, including end-stage renal disease (ESRD), we do not currently project sales of the agent in these indications as a clinical development pathway has yet to be proposed in these areas. Nevertheless, we note that these other indications represent potential upside.

Herewith, we present our market assumptions for GR-MD-02 going forward (Table 5, overleaf). We project the drug's launch in mid-2017 in the U.S. and mid-2018 ex-U.S. The drug could achieve significant market penetration for the treatment of NASH, in our view, since we believe that it is unlikely to have substantial competition at the time of launch. Our market model assumptions include the pricing of the drug at $85,000 per patient annually in the U.S. and $60,000 per patient annually in Europe. We factor in 3% price increases each year to account for inflation, and assume generic competition in the U.S. beyond 2030, as the year of patent expiry could easily be later than this time frame. In Europe, we assume a similar time window for commercialization.

Furthermore, we note that GR-MD-02 could continue to benefit from two significant demographic trends – the emergence of chronic hepatitis C infection as a major cause of chronic liver disease, and the rise of obesity in the developed world. As the number of individuals on direct-acting antivirals aimed at reducing hepatitis C viral load increases, the likelihood of long-term non-alcoholism-related liver injury rises as well. This is a particularly probable scenario because several direct-acting antiviral agents that are aimed at treating chronic hepatitis C infection were shown to cause liver injury in clinical trials. We note that, because of the favorable side effect profile expected for GR-MD-02, it would be very easy to envision the drug being dosed alongside direct-acting antiviral agents in the hepatitis C patient population.

We direct investors' attention to one important relative valuation consideration. While NASH is clearly an orphan indication, we think pricing in this indication is likely to be comparatively high because of the anticipated lack of competition and the fact that this is a chronic condition that – if untreated – would cause severe complications for the patient. Galectin is entering proof-of-concept clinical development with GR-MD-02, so it is still an early-stage initiative. However, we note that the firm's proposed timeline for development of the drug is likely to permit the release of top-line data from a Phase 1 dose-escalation trial in NASH sufferers – an initial value inflection point – in late 2013 and data from a Phase 2 study – the main value inflection point, in our view – in late 2014 or early 2015. We project the launch in the U.S. in 2017 and in Europe in 2018. One of the companies we have listed as a comparable to Galectin Therapeutics, Synageva BioPharma, currently has a single compound in Phase 1/2 development. This agent – designated SBC-102 – is a recombinant human lysosomal acid lipase (rhLAL) enzyme replacement therapy for the treatment of lysosomal acid lipase (LAL) deficiency, a lysosomal storage disease classified as an ultra-orphan disorder. LAL deficiency is estimated to affect 1 in 40,000 people, which would place the total addressable target population in the roughly 25,000-patient worldwide range. SBC-102 is slated to enter pivotal development in late-onset LAL deficiency patients in early 2013. The drug could be approved in 2015, only two years ahead of Galectin's drug in NASH. Synageva BioPharma, which recently raised $100 million in a follow-on offering, currently trades at a market cap of roughly $1 billion, while Galectin has a market cap of roughly $30 million. Accordingly, we consider Galectin's current valuation attractive as well as largely risk-mitigated, since Galectin is both targeting a much larger patient population with GR-MD-02 as well as developing another pipeline agent that already has shown favorable clinical data, GM-CT-01, in a completely different indication.

Table 5: GR-MD-02 Estimated Global Sales – Non-Alcoholic Steatohepatitis (NASH) Market Size Model

	2012	2013	2014	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024	2025	2026	2027	2028	2029	2030
US Population																			
Cirrhosis population	450,000	453,825	457,683	461,573	465,496	469,453	473,443	477,468	481,526	485,619	489,747	493,910	498,108	502,342	506,612	510,918	515,261	519,640	524,057
Non-alcoholic steatohepatitis sufferers	67,500	70,875	74,419	78,140	82,047	86,149	90,456	94,979	99,728	104,715	109,950	115,448	121,220	127,281	133,645	140,328	147,344	154,711	162,447
GR-MD-02 Penetration	0%	0%	0%	0%	0%	3%	7%	9%	11%	15%	18%	21%	23%	25%	23%	21%	18%	17%	16%
Patients on GR-MD-02				0	0	2,584	6,332	8,548	10,970	15,707	19,791	24,244	27,881	31,820	30,738	29,469	26,522	26,301	25,991
Cost per patient ($)						85,000	87,550	90,177	92,882	95,668	98,538	101,494	104,539	107,675	110,906	114,233	117,660	121,190	124,825
US GR-MD-02 sales ($ MM)				0	0	220	554	771	1,019	1,503	1,950	2,461	2,915	3,426	3,409	3,366	3,121	3,187	3,244
European Population																			
Cirrhosis population	600,000	615,000	630,375	658,742	691,679	726,263	762,576	800,705	840,740	882,777	926,916	973,262	1,021,925	1,073,021	1,126,672	1,183,006	1,242,156	1,304,264	1,369,477
Non-alcoholic steatohepatitis sufferers	84,000	86,100	88,253	92,224	96,835	101,677	106,761	112,099	117,704	123,589	129,768	136,257	143,069	150,223	157,734	165,621	173,902	182,597	191,727
GR-MD-02 Penetration	0%	0%	0%	0%	0%	0%	4%	7%	9%	12%	14%	16%	18%	17%	16%	15%	12%	10%	9%
Patients on GR-MD-02				0	0	0	4,270	7,847	10,593	14,831	18,168	21,801	25,753	25,538	25,237	24,843	20,868	18,260	17,255
Cost per patient ($)							60,000	61,800	63,654	65,564	67,531	69,556	71,643	73,792	76,006	78,286	80,635	83,054	85,546
European GR-MD-02 sales ($ MM)				0	0	0	256	485	674	972	1,227	1,516	1,845	1,885	1,918	1,945	1,683	1,517	1,476
Global GR-MD-02 sales ($ MM)				**0**	**0**	**220**	**811**	**1,256**	**1,693**	**2,475**	**3,177**	**3,977**	**4,760**	**5,311**	**5,327**	**5,311**	**4,803**	**4,704**	**4,721**
Galectin Therapeutics Net Income ($ MM)				**0**	**0**	**26**	**113**	**188**	**254**	**371**	**477**	**597**	**714**	**797**	**799**	**797**	**720**	**706**	**708**

Source: Company Reports and Aegis Capital Corp. estimates

Intellectual Property Portfolio

Galectin Therapeutics owns the rights to several issued patents and a wide array of pending patent applications. The table below lists the issued patent portfolio for the company. Since the firm – under its original name, Pro-Pharmaceuticals – was originally focusing on oncology applications for its platform, the issued patent estate principally covers various galectin inhibitors from composition-of-matter and method-of-use perspectives in the cancer domain. In particular, the patent estate covers one particular carbohydrate-based polysaccharide, galactomannan, which was shown to illustrate the principle of binding to galectins and inactivating them.

The claims in the issued patent estate cover both the structures of the various drug candidates, as well as their combination with various known chemotherapy agents. Furthermore, the scope of the patent claims allows Galectin Therapeutics to pursue the development of its carbohydrate-based drug platform in a wide range of cancer indications, irrespective of tissue or cell type. The firm recently received notice of patent issuance for U.S. Patent No. 8,236,780, which will protect its liver fibrosis drug candidate GR-MD-02 until October 2026 (not counting patent term extensions).

Table 6: Galectin Therapeutics Issued Intellectual Property

Patent Number	Title	Issue Date	Expiry Date	Country	Description
6,642,205	Methods and Compositions for Reducing Side Effects in Chemotherapy Treatments	11/4/2003	9/24/2021	United States	Reduction of chemotherapy toxicity via co-administration with a galectin inhibitor
6,645,946	Delivery of a Therapeutic Agent in a Formulation for Reduced Toxicity	11/11/2003	3/27/2021	United States	Reducing toxicity of chemotherapy via combined therapy with a galectin blocker (covers GM-CT-01)
6,914,055	Delivery of a Therapeutic Agent in a Formulation for Reduced Toxicity	7/5/2005	8/27/2023	United States	Reducing toxicity of chemotherapy via combined therapy with a galectin blocker (covers GM-CT-01)
6,982,255	Delivery of a Therapeutic Agent in a Formulation for Reduced Toxicity	1/3/2006	8/27/2023	United States	Reducing toxicity of chemotherapy via combined therapy with a galectin blocker (covers GM-CT-01)
7,012,068	Co-administration of a Polysaccharide with a Chemotherapeutic Agent for the Treatment of Cancer	3/14/2006	3/27/2022	United States	Reducing toxicity of chemotherapy via combined therapy with a galectin blocker (covers GM-CT-01)
7,893,252	Selectively Depolymerized Galacto-mannan Polysaccharide	2/22/2011	2/25/2028	United States	Compositions and methods covering combinations of polysaccharide admixtures with chemotherapy drugs
8,236,780	Galactose-Pronged Polysaccharides in a Formulation for Antifibrotic Therapies	7/23/2012	10/21/2026	United States	Proprietary multi-valent galectin inhibitors for treatment of fibrosis
272022B2	Co-administration of Polysaccharide with a Chemotherapeutic Agent for the Treatment of Cancer	NA	NA	Australia	Reducing toxicity of chemotherapy via combined therapy with a galectin blocker (covers GM-CT-01)
2002 731786	Co-administration of Polysaccharide with a Chemotherapeutic Agent for the Treatment of Cancer	NA	2022	European Union	Reducing toxicity of chemotherapy via combined therapy with a galectin blocker (covers GM-CT-01)
2002 4744782	Co-administration of Polysaccharide with a Chemotherapeutic Agent for the Treatment of Cancer	NA	2022	European Union	Reducing toxicity of chemotherapy via combined therapy with a galectin blocker (covers GM-CT-01)

Source: Company reports

The table overleaf lists the pending applications that Galectin Therapeutics has filed in the U.S. and (in certain cases) other countries. These applications cover many of the most recently-discovered features of the galectin-modulating technology platform. In particular, the most valuable patent applications in our view provide composition-of-matter and method-of-use protection to GR-MD-02 in both liver fibrosis as well as other diseases in which fibrotic processes are implicated. We believe that these patent applications have a high likelihood of being issued because, to our knowledge, no other firms aside from Galectin Therapeutics have hitherto pursued the development of carbohydrate-based multivalent galectin-blocking drugs, and only Galectin Therapeutics is pursuing the development of such agents in liver fibrosis. We note that Galectin Therapeutics' patent estate – which has been largely driven by the work of Anatole Klyosov, one of the foremost carbohydrate-based drug development experts in the world, and his collaborators – has also followed many of the most cutting-edge discoveries in galectin-inhibiting research, including the discovery that galectin blockade may be useful in the treatment of cardiovascular disorders such as ischemic heart disease. If the galectin-blocking approach does prove useful in cardiac indications, we believe this may provide upside for Galectin Therapeutics beyond liver fibrosis and cancer indications.

Table 7: Galectin Therapeutics Pending Intellectual Property

Title	Filing Date	Publication Date	Country	Description
Methods for reducing the incidence of chemotherapy-induced mucositis	8/7/2009	NA	United States	Application of galectin blockade to treatment of oral mucositis (sores in the mouth following chemotherapy)
Compounds, formulations and methods for reducing mucositis and/or other galectin-dependent pathologies	5/7/2010	NA	United States	Application of galectin blockade to treatment of oral mucositis (sores in the mouth following chemotherapy)
Co-Administration of a Polysaccharide with a Chemotherapeutic Agent for the Treatment of Cancer	NA	8/28/2008	United States	Continuation-in-part of previous patents on combination therapy with galectin inhibitors and chemotherapy
Compositions and Methods for the Enhancement of Chemotherapy with Microbial Cytotoxins	NA	11/20/2008	United States	Improving chemotherapy via combination therapy with microbial cytotoxins
Compositions and methods for targeting metastatic tumors using multivalent ligand-linked carbohydrate polymers	NA	8/28/2008	United States	Method-of-use for GM-CT-01 in oncology and composition-of-matter protection for related drug candidates
Galectin Binding and Blocking Polysaccharides	2/4/2011	NA	United States	Structure and function of galectin-binding carbohydrate compounds
Methods of Treatment of Ischemic Disease and/or Chronic Cardiovascular Disease with Galectin Binding Agents	NA	NA	United States	Usage of galectin inhibitors in treatment of cardiovascular fibrosis and associated diseases
Co-Administration of a Polysaccharide with a Chemotherapeutic Agent for the Treatment of Cancer	8/9/2003	NA	United States	Continuation-in-part of previous patents on combination therapy with galectin inhibitors and chemotherapy
Composition of novel carbohydrate drug for treatment of human diseases	NA	12/30/2011	United States	Novel undisclosed galectin inhibitor candidate agent in company pipeline
The optimal structural signature for α-galactomannan binding to galectin-1	NA	9/13/2011	United States	Structure-activity relationship claims for galectin inhibitor drug design
Methods of treatment for Non-Alcoholic Steatohepatitis (NASH)	NA	9/16/2011	United States	Method-of-use claims on GR-MD-02 and related candidates in liver fibrosis
Co-administration of a polysaccharide with a chemotherapeutic agent for the treatment of cancer	NA	NA	European Union	Continuation-in-part of previous patents on combination therapy with galectin inhibitors and chemotherapy
Co-administration of a polysaccharide with a chemotherapeutic agent for the treatment of cancer	NA	NA	Australia	Continuation-in-part of previous patents on combination therapy with galectin inhibitors and chemotherapy
Co-administration of a polysaccharide with a chemotherapeutic agent for the treatment of cancer	NA	NA	Israel	Continuation-in-part of previous patents on combination therapy with galectin inhibitors and chemotherapy
Co-administration of a polysaccharide with a chemotherapeutic agent for the treatment of cancer	NA	NA	Japan	Continuation-in-part of previous patents on combination therapy with galectin inhibitors and chemotherapy
Co-administration of a polysaccharide with a chemotherapeutic agent for the treatment of cancer	NA	NA	Brazil	Continuation-in-part of previous patents on combination therapy with galectin inhibitors and chemotherapy
Compositions and Methods for Treating Metastatic Tumors with Multivalent Ligand-Linked Carbohydrate Polymers	NA	NA	Japan	Composition-of-matter and method-of-use claims on galectin inhibitors for treatment of metastatic cancer
Methods for Reducing the Incidence of Chemotherapy-Induced Mucositis	NA	NA	PCT Application	Blocking galectins to treat chemo-induced oral mucositis (mouth sores)

Source: Company reports

Capital Structure and Financing History

The table below depicts Galectin Therapeutics' current capital structure. Following the raising of $10.5 million in net proceeds from a follow-on offering in the first quarter of 2012, the firm reported $15.3 million in cash as of end-March 2012. We believe that the current cash position is sufficient to fund operations through the end of 2013. Our 18-month price target assumes the impact of exercise of all currently-outstanding options and warrants.

Table 8: Galectin Therapeutics Capital Structure

	Number of Shares	Exercise Price	Expiration Date	Total Cash
Cash, cash equivalents and marketable securities				$15,314,000
Common Stock	15,721,003			
Preferred Stock	2,627,110			
Options	2,997,468	$6.84		$20,502,681
Warrants	7,424,241	$3.71		$27,543,934
Fully Diluted Shares	**28,769,822**			**$63,360,615**

Source: Company reports

In the table below, we list all of Galectin Therapeutics' previous financings, including those conducted while the firm was still being known as Pro-Pharmaceuticals. We note that the company has raised, since inception, roughly $57 million in net cash proceeds from various equity financings since 2001. In our view, while the total amount raised represents a significant sum, we note that the firm has successfully established proof-of-concept data for its galectin-inhibiting platform and has also shown that the platform has applicability in multiple areas. In our opinion, this firm remains capable of advancing the development of its two primary drug candidates in a cost-effective manner.

Table 9: Galectin Therapeutics Financing History

		Net Cash Proceeds	Offering Costs	Common Shares	Per Share Price	Notes
Public Company						
	Common stock and warrants in 2001	$ 2,221,000	$ 17,000	114,884	$ 19.48	
	Common stock and warrants in 2002	$ 602,000	$ 49,000	31,000	$ 21.00	
	Common stock, 2002 Private Placement	$ 4,081,000	$ 230,000	718,561	$ 6.00	
	Common stock, 2003 Private Placement	$ 4,671,000	$ 128,000	399,917	$ 12.00	
	Common stock and warrants, October 2003 Private Placement (PIPE)	$ 4,041,000	$ 559,000	219,096	$ 21.00	
	Common stock and warrants, April 2004 Private Placement (PIPE)	$ 3,983,000	$ 466,000	206,019	$ 21.60	
	Common stock and warrants, August 2004 Private Placement (PIPE)	$ 5,515,000	$ 485,000	333,334	$ 18.00	
	Convertible debenture conversions 2006 & 2007	$ 11,023,000	$ -	2,174,897	$ 5.07	
	Series A convertible preferred	$ 1,690,500	$ 52,000	290,417	$ 6.00	common equivalent shares
	Common stock, February 2008	$ 1,044,000	$ 369,000	1,250,000	$ 1.13	common equivalent shares
	Series B-1 convertible preferred	$ 1,463,000	$ 337,000	600,000	$ 3.00	common equivalent shares
	Series B-2 convertible preferred	$ 4,012,000	$ 188,000	1,400,000	$ 3.00	common equivalent shares
	Series C super dividend convertible preferred	$ 2,203,000	$ 47,000	375,000	$ 6.00	common equivalent shares
	Common stock and warrants March 2012	$ 10,403,000	$ 1,597,000	2,666,722	$ 4.50	
Total Amount		$ 56,952,500	$ 4,524,000	10,779,847		

Source: Company reports

Financial Review and Outlook

Revenue: We forecast no revenue for 2012 and 2013, respectively. Management does not provide guidance.

Gross Margins: As a development-stage company, there are historically no costs of goods sold. We project that the gross margins on both GR-MD-02 and GM-CT-01 could exceed 85%, which should enable healthy cash flow generation, even accounting for the fact that Galectin Therapeutics will likely need to find commercially-established marketing partners for both drug candidates in order to optimize their potential.

Operating Expenses: For 2012, we estimate operating expense levels that are generally similar to those seen in 2010 and 2011. We estimate R&D expense of $5 million in 2012, as the company advances candidates from its pipeline into the clinic.

Taxes: We assume a 35% corporate tax rate and net operating loss carry-forwards of roughly $5 million remaining after GM-CT-01 and GR-MD-02 reach the market. As of December 31st, 2011, Galectin Therapeutics had $7.6 million in net operating loss carry-forwards remaining, which are slated to expire between 2026 and 2031.

Share Count: The outstanding fully-diluted share count stands at 15.7 million shares. The fully-diluted shares account for the conversion of roughly 3 million shares in the form of options, with a total of 13 million shares, warrants and preferred stock units being potentially dilutive. Given the company's cash position, strategic goals, and capital structure, a share repurchase program is unlikely, in our view.

EPS: We forecast EPS of ($0.71) and ($0.65) for 2012 and 2013, respectively. We do not anticipate any revenue generation near-term.

Balance Sheet: The firm held $15.7 million in cash as of March 31st, 2012.

Cash Flow: We estimate that the firm will consume roughly $8.2 million in operating cash flows during 2012 and $10 million in 2013. Additional funding may be required in 2013 to support continued operational activities.

Guidance: The firm does not provide financial guidance.

Management Team

The firm's management team comprises individuals with substantial track records in the biotechnology and healthcare industries. Their backgrounds are described below.

Peter G. Traber, M.D.
President, Chief Executive Officer and Chief Medical Officer
Dr. Traber is President Emeritus of Baylor College of Medicine, where he was Chief Executive Officer from 2003 to 2008. From 2000 to 2003 he was Senior Vice President of Clinical Development and Medical Affairs and Chief Medical Officer of GlaxoSmithKline plc. Dr. Traber served as Chief Executive Officer of the University of Pennsylvania Health System and was Chair of the Department of Internal Medicine and Chief of Gastroenterology for the University of Pennsylvania School of Medicine. Dr. Traber has also managed a molecular biology research laboratory and published over 100 articles of original research, reviews, and book chapters. Dr. Traber received his M.D. from Wayne State School of Medicine and his B.S. in chemical engineering from the University of Michigan.

Anatole Klyosov, Ph.D., D. Sc.
Chief Scientist
Dr. Klyosov, a founder of the company, is a co-inventor of the firm's patented technology. From 1996 to 2000, he was Vice President of Research and Development for Kadant Composites, Inc., a subsidiary of Kadant, Inc. (KAI-NYSE), where he directed a laboratory specializing in biochemistry, microbiology and polymer engineering. From 1990 to 1998, Dr. Klyosov was Visiting Professor of Biochemistry, Center for Biochemical and Biophysical Sciences at Harvard Medical School. From 1981 to 1990, he was Head of the Carbohydrates Research Laboratory at the A.N. Bach Institute of Biochemistry, USSR Academy of Sciences. Dr. Klyosov is the recipient of distinguished awards including the USSR National Award in Science and Technology. He holds over 20 patents, has published over 250 peer-reviewed articles in scientific journals, written books on enzymes, carbohydrates, and biotechnology, and has edited two books highly pertinent to Galectin Therapeutics' platform technology, *Carbohydrates In Drug Design* and *Galectins*. Dr. Klyosov earned his Ph.D. and D.Sc. degrees in physical chemistry and an M.S. degree in enzyme kinetics from Moscow State University.

Eliezer Zomer, Ph.D.
Executive Vice President, Manufacturing & Product Development
Since 2003, Dr. Zomer has been developing the manufacturing processes necessary to produce galectin-blocking compounds at clinical scale. Dr. Zomer was previously the founder of Alicon Biological Control, where he worked from 2000 to 2002. Prior to that, he was Vice President of product development at SafeScience, Inc., from 1998 to 2000, and from 1987 to 1998 was Vice President of Research and Development at Charm Sciences, Inc. Dr. Zomer received a B.Sc. degree in industrial microbiology from the University of Tel Aviv, a Ph.D. in biochemistry from the University of Massachusetts in 1978, and did postdoctoral work at the National Institutes of Health (NIH).

Thomas A McGauley, CPA
Acting Financial Officer
Mr. McGauley joined Galectin Therapeutics in 2009, serving as Director of Finance and Accounting. He was previously Director of Financial Reporting at deCODE Genetics, a former publicly-traded life sciences firm, from 2005 to 2010. Mr. McGauley has over seven years of public accounting experience, most recently as a manager at PricewaterhouseCoopers where he focused on life science firms. He also served as a Captain and Company Commander in the U.S. Army and Massachusetts National Guard (active duty 1995 to 1998 and reserves 1998 to 2000). Mr. McGauley is a Certified Public Accountant and holds a B.S. in business administration from Stonehill College.

Maureen E. Foley
Chief Operating Officer, Corporate Secretary
Ms. Foley has 30 years of business and operations management experience including facility design, project management, information technology, human resources, press and public relations, as well as accounting and finance with startup companies. Between 1999 and 2000, she managed business operations for eHealthDirect, Inc., a developer of medical records processing software, and ArsDigita, Inc., a web development company. From 1996 to 1999, Ms. Foley was Manager of Operations with Thermo Fibergen, Inc., a subsidiary of Thermo Fisher Scientific, Inc. Ms. Foley graduated from The Wyndham School in Boston, MA, with a major in mechanical engineering.

Board of Directors

The firm's Board of Directors includes several senior-level individuals with substantial expertise in the biopharmaceutical industry, along with individuals possessing significant track records in the legal and corporate domains.

James C. Czirr
Executive Chairman
Mr. Czirr has served as Chairman of the Board of Galectin Therapeutics since February 2009, and as Executive Chairman since February 2010. He was one of the original founders of the firm, and also co-founded and is manager and general partner of 10X Fund, L.P., the holder of the firm's Series B Preferred Stock. He was instrumental in the early-stage development of Safe Science Inc., a developer of anti-cancer drugs. From 2005 to 2008, Mr. Czirr was CEO of Minerva Biotechnologies Corporation, a developer of nano particle bio chips to determine the cause of solid tumors. He has also been a consultant to Metalline Mining Company Inc. (NYSE Amex: MMG), now known as Silver Bull Resources, Inc., a mineral exploration company seeking to become a low-cost producer of zinc. Mr. Czirr received a B.B.A. from the University of Michigan.

Peter G. Traber, M.D.
See management bios in previous section.

Gilbert F. Amelio, Ph.D.
Dr. Amelio, who began his career at Bell Labs, has been Senior Partner of Sienna Ventures, a privately held venture capital firm, since 2001. He was appointed a director on the board of Galectin Therapeutics in February 2009. Dr. Amelio was Chairman and CEO of Jazz Technologies, Inc., a specialty wafer foundry, from 2005 until his retirement in 2008, when he was named Chairman Emeritus. From 1999 to 2005, Dr. Amelio was Chairman and CEO of Beneventure Capital, LLC, a venture capital firm. From 1997 to 2004, he was Principal of Aircraft Ventures, LLC, a consulting firm. Dr. Amelio was elected a Director of AT&T in 2001 and had previously served as an Advisory Director of AT&T from 1997 to 2001. In 1996 and 1997, Dr. Amelio was CEO of Apple, Inc., and from 1991 to 1996, he was CEO of National Semiconductor Corporation. He was also a director of Chiron, now a part of Novartis, from 1991 to 1996.

Rod D. Martin
Vice Chairman
Mr. Martin co-founded and is a manager and general partner of 10X Fund, L.P., the holder of Galectin Series B Preferred Stock. Mr. Martin was senior advisor to Peter Thiel, founder of PayPal, Inc., during its startup phase, initial public offering and later acquisition by eBay Inc. Subsequently, Mr. Martin served as a senior advisor at Clarium Capital, Thiel's global macro hedge fund with more than $7.8 billion under management. Mr. Martin is founder and Chairman of Advanced Search Laboratories, Inc., and a director of Proxomo Software. He previously served as Director of Policy Planning & Research for former Arkansas Governor, Mike Huckabee. He is an author and speaker

and leads several non-profit organizations. Mr. Martin holds a J.D. from Baylor Law School, a B.A. from the University of Arkansas, and was a Sturgis Fellow at Cambridge University in Great Britain.

Marc Rubin, M.D.
Dr. Marc Rubin has served as President and CEO of Titan Pharmaceuticals, Inc. (OTC: TTNP) since October 2007, and has been a Director since November 2007. He has served on the Galectin Therapeutics Board of Directors since October 2011. Dr. Rubin previously served as Head of Global Research and Development for Bayer Schering Pharma, as well as a member of the Executive Committee of Bayer Healthcare and the Board of Management of Bayer Schering Pharma. Prior to the merger of Bayer Pharmaceuticals and Schering AG in June 2006, Dr. Rubin was a member of the Executive Board of Schering AG, as well as Chairman of Schering Berlin Inc. and President of Berlex Pharmaceuticals, a division of Schering AG. From 1990 until August 2003, Dr. Rubin was employed by GlaxoSmithKline, where he held positions of responsibility in global clinical and commercial development overseeing programs in the United States, Europe, Asia, and Latin America. From 2001 through 2003, he was Senior Vice President of Global Clinical Pharmacology & Discovery Medicine. Dr. Rubin holds an M.D. from Cornell University Medical College, completed an internship and residency in internal medicine at the Johns Hopkins Hospital, and obtained fellowships in medical oncology and infectious diseases at the NIH. Dr. Rubin also served on the Board of Directors of Medarex, Inc. until its acquisition by Bristol-Meyers Squibb in 2009, and currently serves on the Board of Directors of Curis Inc. and The Rogosin Institute.

Kevin D. Freeman, CFA
Mr. Freeman possesses 20 years of financial expertise. A member of the Galectin Board of Directors since May 2011, he is currently President of Cross Consulting Services, LLC and Chief Investment Officer of Capitalist Publishing Co., Inc. Over the past 20 years, Mr. Freeman has served as portfolio manager, entrepreneur, management executive, and consultant. From 2000-2004, Mr. Freeman was chairman of Separate Account Solutions, Inc., a wealth management solutions company, which he also co-founded. From 1991-2000, Mr. Freeman worked for Franklin Templeton Investment Services, Inc., where he served as Managing Director, Associate Portfolio Manager, Portfolio Consultant, Director of Portfolio Consulting and co-developer of the Portfolio Operations Group. Mr. Freeman holds a B.S. in Business Administration from the University of Tulsa.

Arthur R. Greenberg
Mr. Greenberg, with nearly 40 years in the semiconductor equipment and materials industry, is the President and founder of Prism Technologies, Inc., a provider of professional sales and marketing services and business development consulting services. He has been a director on the Galectin Board since August 2009, and is a member of the board of UV Tech Systems, a maker of equipment used to fabricate semiconductor devices. Previously, he was the first President of SEMI, North America, a semiconductor equipment and materials industry trade association representing the interests, including public policy, of more than 2000 members doing business in North America. Mr. Greenberg received his B.S. in business administration from Henderson State University.

John Mauldin
Mr. Mauldin, who has been on the Board of Directors at Galectin Therapeutics since May 2011, is a renowned financial expert and *New York Times* best-selling author. Mr. Mauldin is currently the President of Millennium Wave Advisors. Prior to that position, he was CEO of the American Bureau of Economic Research. Mr. Mauldin is a regular contributor to publications including *The Financial Times* and *The Daily Reckoning*, as well as a frequent guest on CNBC, Yahoo Tech Ticker, and Bloomberg TV. He is the author of *Thoughts from the Frontline*, a free weekly e-newsletter, and several books, including: Bull's Eye Investing: Targeting Real Returns in a Smoke and Mirrors Market,

Just One Thing: Twelve of the World's Best Investors Reveal the One Strategy You Can't Overlook, and Endgame: The End of the Debt Supercycle and How it Changes Everything. He also edits *Outside the Box*, a free weekly e-letter. Mr. Mauldin holds a B.A. from Rice University and a Master of Divinity degree from Southwestern Baptist Theological Seminary.

Steven Prelack, CPA
A director since April 2003, Mr. Prelack became Senior Vice President of Operations and CFO of VetCor in July 2010, an owner and operator of veterinary hospitals, and from 2001 was Senior Vice President, CFO and Treasurer of VelQuest Corporation, a provider of automated compliance software solutions for the pharmaceutical industry. Mr. Prelack is a director of Codeco Corporation, a designer and manufacturer of custom resisters and switches. From 2007 through 2009, Mr. Prelack served as Director and Audit Committee Chair for BioVex, a biotechnology company focused on cancer, and he currently serves on its Strategic Advisory Board. Mr. Prelack, a Certified Public Accountant, received a B.B.A. degree from the University of Massachusetts at Amherst.

Paul Pressler
Mr. Pressler, a member of the Galectin Therapeutics Board of Directors since May 2011, has been a partner in the law firm of Woodfill & Pressler since 1995. He has served as a director of Revelation, Inc., and was also in private mediation practice. A retired justice of the Texas Court of Appeals, Judge Pressler was appointed Justice of the Texas Court of Appeals in 1978, serving until 1992. Judge Pressler also served as District Judge from 1970 to 1978. From 1958 to 1970, he worked with the law firm of Vinson & Elkins. Judge Pressler has been a director of Salem Communications Corp. since March 2002.

Jerald K. Rome
Mr. Rome, now retired, has been a private investor since 1996 and has served on the Galectin Therapeutics Board of Directors since March 2004. In 1993, Mr. Rome founded Amberline Pharmaceutical Care Corp., a marketer of non-prescription pharmaceuticals, and served as its President from 1993 to 1996. From 1980 to 1990, he served as Chairman, President and CEO of Moore Medical Corp., a national distributor of branded pharmaceuticals and manufacturer and distributor of generic pharmaceuticals. Mr. Rome received a B.S. degree in pharmaceutical sciences from the University of Connecticut.

Scientific and Clinical Advisory Boards

Galectin Therapeutics maintains an extensive network of experts to advise the firm on its strategic and clinical development initiatives. The firm's Scientific Advisory Board is composed of three individuals – Scott L. Friedman, M.D., Professor of Medicine and Chief of Liver Diseases at Mount Sinai School of Medicine; Gilbert S. Omenn, M.D., Ph.D., a member of the Amgen Inc. Board of Directors and Professor of Internal Medicine, Human Genetics and Public Health, and Director of the Center for Computational Medicine and Bioinformatics at the University of Michigan; and Irwin J. Goldstein, Ph.D., an emeritus Professor in the Department of Biological Chemistry and former Chair of the Department of Medicine at the University of Michigan. Dr. Goldstein is an internationally-recognized expert on carbohydrate-protein interactions. Dr. Friedman serves as a Principal Consultant to Galectin on clinical development issues. He was also the first clinical researcher to isolate the hepatic stellate cell, the key cell type responsible for scar production in the liver. The firm's Clinical Advisory Board includes physicians such as Dr. Raymond Chung, Vice Chief of Gastroenterology and Chief of Hepatology at Massachusetts General Hospital and the Harvard School of Medicine.

Public Companies Mentioned in this Report:

Abbott Laboratories (ABT/NYSE)

Achillion Pharmaceuticals (ACHN/NASDAQ)

Ampio Pharmaceuticals (AMPE/NASDAQ)

Amgen (AMGN/NASDAQ)

BioTime (BTX/AMEX)

Bristol-Myers Squibb (BMY/NYSE)

Dendreon Corporation (DNDN/NASDAQ)

ImmunoCellular Therapeutics (IMUC.OB/OTCBB)

InVivo Therapeutics (NVIV.OB/OTCBB)

La Jolla Pharmaceutical Co. (LJPC.OB/OTCBB)

Medgenics (MDGN/AMEX)

Merck & Co. (MRK/NYSE)

Micromet Inc. (MITI/NASDAQ)

Neuralstem (CUR/AMEX – Buy)

Oncothyreon (ONTY/NASDAQ)

Organovo (ONVO.OB/OTCBB)

Pfizer Inc. (PFE/NYSE)

Sanofi S.A. (SNY/NYSE)

Sorrento Therapeutics (SRNE.OB/OTCBB)

Synageva BioPharma (GEVA/NASDAQ)

VeraStem (VSTM/NASDAQ)

YM BioSciences (YMI/AMEX)

Table 10: Galectin Therapeutics (GALT) – Historical Income Statements, Financial Projections

FY end December 31
$ in thousands, except per share data

				2012E					2013E				
	2009A	2010A	2011A	1QA	2QE	3QE	4QE	2012E	1QE	2QE	3QE	4QE	2013E
Revenue													
Product revenue	-	-	-	-	-	-	-	-	-	-	-	-	-
Service revenue	-	-	-	-	-	-	-	-	-	-	-	-	-
Research and other	-	-	-	-	-	-	-	-	-	-	-	-	-
Total revenue	-	-	-	-	-	-	-	-	-	-	-	-	-
Expenses													
Cost of product and service revenue	-	-	-	-	-	-	-	-	-	-	-	-	-
Research & development	1,110	1,066	3,552	901	1,200	1,400	1,500	5,001	1,600	1,700	1,800	1,900	7,000
General and administrative	4,983	3,817	6,857	1,052	1,100	1,200	1,300	4,652	1,300	1,300	1,300	1,300	5,200
Total expenses	6,093	4,883	10,409	1,953	2,300	2,600	2,800	9,653	2,900	3,000	3,100	3,200	12,200
Gain (loss) from operations	(6,093)	(4,883)	(10,409)	(1,953)	(2,300)	(2,600)	(2,800)	(9,653)	(2,900)	(3,000)	(3,100)	(3,200)	(12,200)
Other income/expense													
Interest income/expense	3	6	18	3	5	4	2	14	8	6	4	3	21
Change in fair value of convertible debt instrument	-	-	-	-	-	-	-	-					
Change in fair value of warrant liabilities	(1,374)	(1,241)	(524)	-	-	-	-	-	-	-	-	-	-
Other income	2	489	-	-	-	-	-	-	-	-	-	-	-
Total investment income and other	(1,369)	(746)	(506)	3	5	4	2	14	8	6	4	3	21
Loss before provision for income taxes	(7,462)	(5,629)	(10,915)	(1,950)	(2,295)	(2,596)	(2,798)	(9,639)	(2,892)	(2,994)	(3,096)	(3,197)	(12,179)
Series A 12% Convertible Preferred Stock Dividend	(209)	(192)	(232)	(58)	(58)	(58)	(58)	(232)	(58)	(58)	(58)	(58)	(232)
Series B 12% Convertible Preferred Stock Dividend	(341)	(710)	(1,336)	(139)	(139)	(139)	(139)	(556)	(139)	(139)	(139)	(139)	(556)
Series B-1 Redeemable Convertible Preferred Stock Accretion	(1,407)	(2,178)	(230)	(57)	(57)	(57)	(57)	(228)	(57)	(57)	(57)	(57)	(228)
Net loss/income	(9,419)	(8,709)	(12,713)	(2,204)	(2,549)	(2,850)	(3,052)	(10,655)	(3,146)	(3,248)	(3,350)	(3,451)	(13,195)
Net loss per share (basic)	(0.20)	(0.15)	(0.23)	(0.17)	(0.16)	(0.18)	(0.19)	(0.71)	(0.17)	(0.16)	(0.16)	(0.16)	(0.65)
Net loss per share (diluted)	(0.20)	(0.15)	(0.23)	(0.17)	(0.16)	(0.18)	(0.19)	(0.71)	(0.17)	(0.16)	(0.16)	(0.16)	(0.65)
Weighted average number of shares outstanding (basic)	48,274	56,301	55,644	13,010	15,746	15,796	15,846	15,100	18,396	20,946	20,996	21,046	20,346
Weighted average number of shares outstanding (diluted)	48,274	56,301	55,644	13,010	15,746	15,796	15,846	15,100	18,396	20,946	20,996	21,046	20,346

Source: Company Reports and Aegis Capital Corp. estimates

Required Disclosures

Price Target

Our 18-month price target for GALT is $7.00 per share.

Valuation Methodology

We utilize a discounted cash flow analysis supporting a risk-adjusted Net Present Value framework to derive the price target. Intrinsic value for the company's product candidates is calculated based upon the size of the market, projected peak penetration rate, competitive landscape, probability of approval based on publicly available clinical data, length of patent term protection and other factors. Intrinsic values are then added to derive the price target.

Risk Factors

Issues that could prevent the achievement of our price objective include, but are not limited to, clinical, regulatory, competitive, reimbursement and financial risks. Drugs in clinical development may not advance due to inadequate safety, efficacy, or tolerability. Regulatory agencies may decline to approve regulatory submissions in a timely manner, or may not approve a drug candidate at all. The firm may require substantial funding to advance the clinical progress of its candidates, which could be dilutive to current shareholders. We expect competition for the company's drugs from several public and private companies developing pharmaceuticals. Sales of the firm's drugs could depend upon reimbursement from private, as well as public, reimbursement agencies.

For important disclosures go to www.aegiscap.com.

We, Raghuram Selvaraju and Yi Chen, the authors of this research report, certify that the views expressed in this report accurately reflect our personal views about the subject securities and issuers, and no part of our compensation was, is, or will be directly or indirectly tied to the specific recommendations or views contained in this research report.

Research analyst compensation is dependent, in part, upon investment banking revenues received by Aegis Capital Corp.

Aegis Capital Corp. intends to seek or expects to receive compensation for investment banking services from the subject company within the next three months.

Aegis Capital Corp. has performed investment banking services for Galectin Therapeutics, Inc. within the past 12 months.

Rating	Percent	Investment Banking Services/Past 12 Mos. Percent
BUY [BUY]	91.67	18.18
HOLD [HOLD]	8.33	0.00
SELL [SELL]	0.00	0.00

Meaning of Ratings

A) A Buy rating is assigned when we do not believe the stock price adequately reflects a company's prospects over 12-18 months.
B) A Hold rating is assigned when we believe the stock price adequately reflects a company's prospects over 12-18 months.
C) A Sell rating is assigned when we believe the stock price more than adequately reflects a company's prospects over 12-18 months.

Other Disclosures

The information contained herein is based upon sources believed to be reliable but is not guaranteed by us and is not considered to be all inclusive. It is not to be construed as an offer or the solicitation of an offer to sell or buy the securities mentioned herein. Aegis Capital Corp., its affiliates, shareholders, officers, staff, and/or members of their families, may have a position in the securities mentioned herein, and, before or after your receipt of this report, may make or recommend purchases and/or sales for their own accounts or for the accounts of other customers of the Firm from time to time in the open market or otherwise. Opinions expressed are our present opinions only and

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Aegis Capital Corp.
(212) 813-1010
810 Seventh Avenue, 18th Floor
New York, New York 10019