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INITIATION REPORT

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September 6, 2011

Galectin Therapeutics, Inc. (OTC BB / GALT)

Buy The Leader in Advancing Galectin-Targeted Therapies

Clinical programs target rapid development pathways

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INVESTMENT HIGHLIGHTS

Initiating Coverage with a Buy Rating and Price Target of $4 Galectin Therapeutics Inc. is focused on the development of carbohydrate-based therapeutics that target galectins, which are proteins associated with various key biological processes that result in tumor growth, fibrosis, and immunity deactivation. With GALT shares trading at a discount to its peers, we rate the shares a Buy and set a 12-18 month price target of $4 (comparable group mean EV, divided by GALT's EV, and multiplied by GALT shares outstanding).

Galectin Inhibition Has Significant Potential as a Novel Therapeutic Approach Research shows inhibition of galectins has potential to enhance the activity of existing treatments for cancer, and has activity against fibrotic and inflammatory diseases. Galectin inhibition also improves the immune response to disease and could be synergistic with cancer vaccines and other immunotherapies. We also believe a galectin inhibitor program in liver fibrosis targets a large market opportunity, as no drug has yet emerged as an effective antifibrotic agent in humans.

Potential for Near-Term Revenues with Davanat Registration in Colombia We believe the licensing of exclusive marketing and distribution rights to commercialize Davanat in Colombia, and other Latin American countries, to privately-held pharmaceutical company, Procaps S.A., has near-term potential to realize revenues, would provide Galectin Therapeutics with additional clinical experience with GM-CT-01, and has potential to provide cash inflow. Colombia's government and healthcare officials place significant importance on improving the quality of life of its people suffering from life-threatening diseases and believe treatment with Davanat is a desirable option. We believe the near-term completion of pre-commercialization activities by Procaps, anticipated in 3Q11 and 4Q11, as well as official approval in 2Q12, would validate the safety of GM-CT-01 for human use, and could be significant drivers of the stock.

GALT – A Promising New Approach to Safer Therapies We believe that the scientific rationale for targeting galectin activity is growing, thus, we believe the company's galectin inhibitor program targets therapeutic indications where there is potential for rapid clinical development pathways, Orphan Disease status, Fast Track designation, Priority Review, and Accelerated Approval. With the shares at low prices, we recommend considering the compelling proposition and buy GALT shares.

Current Price	**$0.85**
Price Target	**$4.00**

MARKET DATA	09/02/11	
Stock Symbol	GALT	
Market	OTC BB	
52 Wk Low - High	$0.48	- $1.57
Market Cap. (MM)	$62.8	
Shares Out (MM)	73.9	
3-Month Av. Daily Vol (000s)	120.4	
Insider Ownership	19.6%	
Institutional Ownership	0.1%	

BALANCE SHEET METRICS (06/30/11)	
Cash (MM)	$8
Debt (MM)	$0
Debt/Capital	NM
Book Value / Share	NM
Price / Book	NM

EARNINGS DATA				
FY - 12/31	2009A	2010A	2011E	
1Q - 03/31	($0.05)	($0.06)	($0.04)	A
2Q - 06/30	($0.06)	($0.05)	($0.06)	A
3Q - 09/30	($0.03)	($0.03)	($0.03)	E
4Q - 12/31	($0.05)	($0.02)	($0.03)	E
EPS (fully diluted)	($0.20)	($0.15)	($0.15)	E
Revenue (MM)	$0.0	$0.0	$0.0	E

VALUATION METRICS			
Price/Earnings	NM	NM	NM
Price/Revenue	NM	NM	NM

Source: BigCharts.com, FactSet.

INVESTMENT SUMMARY

Galectin Therapeutics Inc. (Nasdaq: GALT), formerly Pro-Pharmaceuticals Inc., is a biotechnology company focused on the development of polysaccharide polymer-based therapeutics that target galectins, which are proteins that are associated with various key biological processes that result in, among other things, tumor cell proliferation and growth, drug resistance, and fibrosis. When administered in combination with anti-cancer agents, Galectin Therapeutics' lead investigational therapeutic from the GM series, GM-CT-01 (also known by the trade name, *Davanat*), increases the efficacy of existing cancer chemotherapies, biologics and vaccines, and reduces their associated serious adverse events. As GM-CT-01 has proven safety in animals and humans, the GM-CT and GR-MD series of candidates in the fibrosis program are more advanced than a typical preclinical development program. GM-CT-01's ability to enhance tumor killing by the immune system will be evaluated in a Phase I/II study scheduled to begin in 2H11. Davanat also has near-term potential for approval as an agent that decreases chemotherapy toxicity for patients with cancer in Colombia, South America, which could open the door to expanded approvals in the next 6-9 months in other countries in South America and Central America, resulting in revenues in 2012. With GTI shares trading at a 59% discount to comparable companies, we believe the stock represents an attractive investment proposition. At current prices, we therefore rate the shares a Buy and set a 12-month price target of $4.

KEY POINTS

- **Growing Body of Scientific Research Support Targeting Galectins as a Novel Therapeutic Approach** Galectin Therapeutics' drug candidates target galectins, which are proteins that are key mediators of biologic processes, such as cell adhesion, recognition, proliferation, differentiation, angiogenesis and apoptosis. A growing body of research shows inhibition of galectins has potential to enhance the activity of existing treatments for cancer, including cancer immunotherapies, and has activity against galectin-associated fibrosis and inflammatory disease. Galectin Therapeutics is in various stages of development with its galectin inhibitor compounds. The drug candidates are based on the company's carbohydrate technology, which combines naturally occurring carbohydrate polymers with galactose residues to create unique, complex carbohydrate-based compounds with pharmaceutical properties.
- **Galectin Inhibition Has Significant Potential as a Therapy for Liver Fibrosis** As inhibition of galectins has been shown to reduce inflammatory and gene expression in liver cells in culture, we believe the company's galectin inhibitors have potential as a novel therapy for liver fibrosis. With early mid-stage GM-CT-01 clinical studies demonstrating safety in humans, Galectin Therapeutics intends to advance its galectin inhibitors further into human testing, such as in post-organ transplant patients with recurrent hepatitis C virus infection (HCV), where Orphan Disease designation is possible. Additionally, the company may in the future explore the potential of its galectin inhibitors in peri-transplant indications, such as in patients with established cirrhosis of various etiologies but are not eligible for liver transplantation, but also chronic HCV fibrosis, chronic hepatitis B virus infection liver fibrosis, alcoholic fibrosis, and renal fibrosis, which we believe altogether are large market opportunities. Galectin Therapeutics intends to conduct a pre-IND meeting with the FDA in 4Q11, and pending a successful meeting, submit an IND in 1Q12, and after the waiting period, initiate a Phase I clinical trial with GM-CT-01, and one of its other galectin inhibitor drug candidates in mid-2012.
- **Potential Synergy with GM-CT-01 and Cancer Immunotherapy** In collaboration with Galectin Therapeutics, a team led by Dr. Pierre van der Bruggen at the Ludwig Institute for Cancer Research in Brussels, Belgium, showed that inhibiting the action of galectin-3 improves the efficacy of T lymphocytes in killing tumor cells. Earlier work showed that tumor-secreted galectin-3 binds to receptors on $CD8^+$ cytotoxic T cells and $CD4^+$ lymphocytes, rendering the T cells incapable of mounting an effective response to tumor antigens. Dr. van der Bruggen et al. showed that addition of Galectin Therapeutics' inhibitor, GM-CT-01, restores the ability of $CD8^+$ T cells to kill tumor cells. In collaboration with Galectin Therapeutics, the Ludwig Institute has scheduled the initiation of a Phase I/II study to evaluate the effects of treatment with GM-CT-01 in combination with a tumor-specific peptide vaccine in patients with advanced metastatic melanoma in 2H11. The results could provide insight into alternative dosing schedules that would result in synergies with autologous cell-based vaccines, tumor-antigen-based vaccines, and other immunotherapies such *Yervoy* (ipilimumab) for melanoma, as well as chemotherapy.

- **Early Results from Clinical Trials with GM-CT-01 as Adjuvant Chemotherapy are Promising** In 2004 the company initiated a multi-center, open-label Phase II clinical trial to evaluate GM-CT-01 as third- or fourth-line therapy in patients with metastatic colorectal cancer (CRC). The study was designed to measure response rate and stable disease, as well as the safety of GM-CT-01 plus 5-FU. Preliminary results on 20 patients from this trial presented in 2006 showed that the treatment combination extended median progression-free survival to 8.4 weeks, and 43% of evaluable patients showed significant tumor shrinkage. Updated data from 14 patients presented in March 2008 showed that the treatment combination extended median survival by 6.7 months with significantly reduced side effects, as compared to 4.6 months from historical data for best standard of care (BSC). Two patients survived more than two years with one patient alive at almost four years. Importantly, patients had a 41% reduction in the incidence of 5-FU related side effects, which altogether we believe supports the clinical benefit with GM-CT-01 and potential for quality of life improvement. Pending raising additional funds and positive stage one results from the GM-CT-01 plus cancer vaccine trial by the Ludwig Institute in 2012, the company has future plans to initiate a Phase III trial to evaluate whether treating patients with advanced CRC with GM-CT-01 plus 5-FU is superior to best standard of care and beneficial in patients unable to tolerate intensive chemotherapy.
- **Davanat (GM-CT-01) Registration in Colombia Has Potential to Result in Near-Term Revenues** We believe the licensing of exclusive marketing and distribution rights to commercialize Davanat in Colombia, South America and other Latin American countries, to privately-held pharmaceutical company, Procaps S.A., has near-term potential to realize revenues. Pursuing more rapid routes of registration and commercialization in other countries are strategies that would provide Galectin Therapeutics additional clinical experience with GM-CT-01, and has potential to provide cash inflow that could be used to support development programs in the U.S. Procaps intends to use Davanat's ability to minimize the side effects of chemotherapy drugs to assist the large number of patients in Colombia and other Latin American countries who cannot afford many current cancer treatments or supporting care. Procaps believes the use of Davanat in cancer patients and its high potential for approval in Colombia is supported by the FDA's approval of galactomannans like Davanat for use in the U.S. and elsewhere for a variety of food and non-food purposes, including new formulations of existing drugs. Colombia's government and healthcare officials place significant importance on improving the quality of life of its people suffering from life-threatening diseases such as cancer, thus, minimizing the toxicities associated with chemotherapy through co-administration of Davanat is a desirable option. We believe the near-term completion of pre-commercialization activities by Procaps, anticipated in 3Q11 and 4Q11, as well as official approval in 2Q12, would validate the safety of GM-CT-01 for human use, and be significant drivers of the stock.
- **GALT – Advancing a Promising New Approach to Treat Cancer and Fibrosis** We believe that the scientific evidence and rationale for targeting galectin activity is growing, thus, we believe Galectin Therapeutics' galectin inhibitors show potential to emerge as a promising new approach to treating cancer and fibrosis. We believe a galectin inhibitor development program in liver fibrosis targets a large market opportunity as liver fibrosis represents a large unmet, medical need, where there is potential for rapid clinical development pathways, Orphan Disease status, Fast Track designation, Priority Review, and an Accelerated Approval process. Our valuation analysis derived an implied value of $4 to $7 for GALT shares, thus, with the shares at attractively low valuations, we therefore recommend investors consider the compelling investment proposition in Galectin Therapeutics and buy GALT shares.

PRODUCT CANDIDATES

Galectin Therapeutics' drug candidates target galectins, which are proteins that are key mediators of biologic processes, such as cell adhesion, recognition, proliferation, differentiation, angiogenesis and apoptosis. A growing body of research shows inhibition of galectins has potential to enhance the activity of existing treatments for cancer, including cancer immunotherapies, and has activity against galectin-associated fibrosis and inflammatory disease. Galectin Therapeutics' development programs, therefore, naturally focus on advancing compounds that inhibit galectin activity in fibrosis and cancer, and enhance the immune response to cancer vaccines.

FIGURE 1: GALECTIN THERAPEUTICS, INC. – PRODUCT PORTFOLIO

	LEAD OPTIMIZATION	DISCOVERY	PRE-CLINICAL	PHASE I	PHASE II	PHASE III	BLA / MAA	IN-MARKET	COMMERCIAL RIGHTS
Cancer									
GM-CT-01 *Colorectal Cancer (International)*						Colombia, S.A.			PROCAPS S.A. (Colombia)
GM-CT-01 *Colorectal Cancer (US)*									Galectin Therapeutics
Vaccine									
GM-CT-01 *Various Tumor Types*									Galectin Therapeutics
Fibrosis									
GM-CT-01 *Liver Fibrosis*									Galectin Therapeutics
GM-CT-02 *Not disclosed*									Galectin Therapeutics
GR-MD-01 *Not disclosed*									Galectin Therapeutics
GR-MD-02 *Not disclosed*									Galectin Therapeutics

Source: Company reports

As shown in Figure 1 above, Galectin Therapeutics is in various stages of development with its GM (galactomannan) and GR (rhamnogalacturonan) series of galectin inhibitor compounds. The drug candidates are based on the company's carbohydrate technology, which combines naturally occurring carbohydrate polymers with galactose residues to create unique, complex carbohydrate-based compounds with pharmaceutical properties.

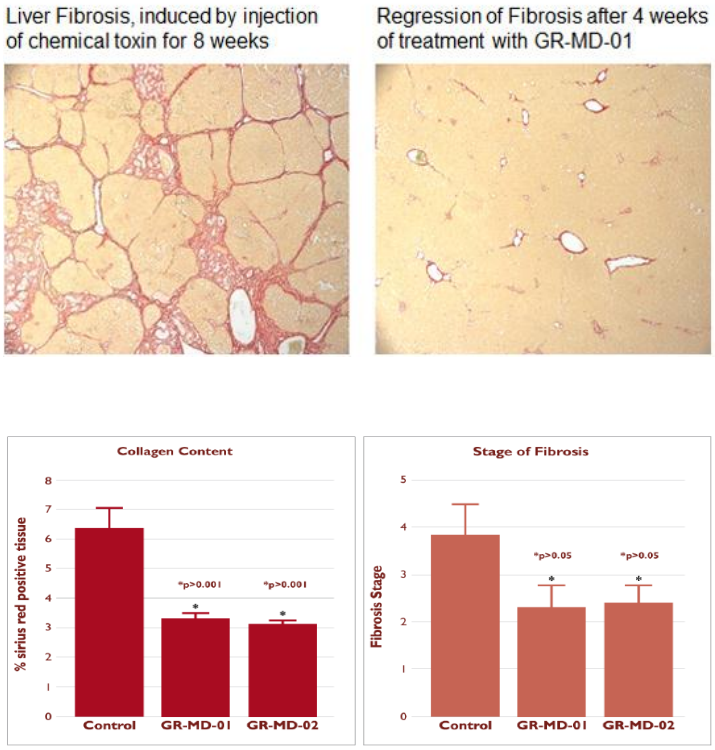
Fibrosis

Fibrosis is the formation of excess connective tissue in response to tissue damage, inflammation or repair. Uncontrolled collagen expression is a pathological process that occurs during the fibrotic process, leading to scar tissue formation and organ dysfunction when the excess connective tissue comes together and radically alters the architecture of the underlying organ. Exposure to toxic chemicals, viral infection or physical injury, are some of the causes of liver, renal and other types of fibrosis.

Cirrhosis of the liver is the result of chronic liver disease and is a major problem in the U.S. with the only therapy currently, liver transplantation. Cirrhosis is characterized by replacement of liver tissue by fibrosis, scar tissue and regenerative nodules, leading to loss of liver function. Galectin-3 is markedly increased in fibrotic liver. Studies in animals have shown that galectin-3 is central to the pathogenesis of liver fibrosis. The activation of stellate cells, for example, the fibrogenic cells of the liver, is thought to be directly dependent on the actions of galectin-3. Furthermore, fibrosis does not occur in galectin-3 gene knockout mice, and therefore, appears necessary for the development of liver fibrosis.

Liver Fibrosis Inhibition of galectins has been shown to reduce inflammatory and gene expression in hepatic stellate cells in culture. In collaboration with Dr. Scott Friedman, M.D., the Director of the Fibrosis Research Center and the Alcoholic Liver Disease Research Center at the Mount Sinai School of Medicine, the effect of three galectin-inhibiting drug candidates on galectin function and liver disease were evaluated in an experimentally-induced in vivo rat model of fibrosis. Results showed that the extensive liver fibrosis induced by administration of a chemical toxin was resolved, and collagen content and fibrosis grade reduced, after four weeks of treatment with the inhibitor, GR-MD-01 (see Figure 2 below).

FIGURE 2: GALECTIN THERAPEUTICS, INC. – EFFECTS OF GR-MD-01 ON LIVER FIBROSIS AND COLLAGEN CONTENT



Source: Company reports

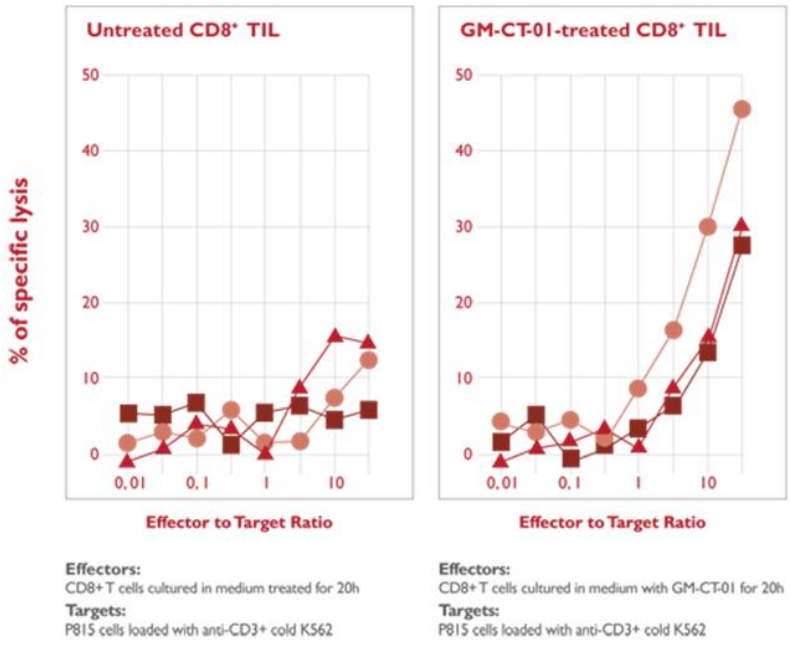
We believe the company's galectin inhibitors have potential to be the first therapy for liver fibrosis. With galectin validated as a target in preclinical studies and mid-stage clinical studies completed with lead candidate, GM-CT-01, Galectin Therapeutics intends to advance its galectin inhibitors further into human testing, such as near-term in post-organ transplant patients with recurrent hepatitis C virus infection (HCV), where Orphan Disease designation is possible. Additionally, the company may in the future explore the potential of its galectin inhibitors in peri-transplant indications, such as in patients with established cirrhosis of various etiologies but are not eligible for liver transplantation, but also chronic HCV fibrosis, chronic hepatitis B virus infection liver fibrosis, alcoholic fibrosis, and renal fibrosis. Supported by safety data with GM-CT-01 in humans, Galectin Therapeutics intends to conduct a pre-IND meeting with the FDA in 4Q11, and pending a successful meeting, submit an IND in 1Q12, and after the waiting period, initiate a Phase I clinical trial with GM-CT-01, and one of its other galectin inhibitor drug candidates in mid-2012.

Cancer

Galectins are widely distributed and expressed in various immune cells, and play important roles in immune response and tumor progression (see more below). In particular, it has been shown that blocking galectins enhances tumor killing by the immune system, thus, Galectin Therapeutics' inhibitors provide a potential new therapeutic approach to treating cancer in combination with anti-cancer drugs and immunotherapies.

Immunotherapy with GM-CT-01 In collaboration with Galectin Therapeutics, a team led by Dr. Pierre van der Bruggen at the Ludwig Institute for Cancer Research in Brussels, Belgium, showed that inhibiting the action of galectin-3 improves the efficacy of T lymphocytes in killing tumor cells. Earlier work showed that tumor-secreted galectin-3 binds to receptors on $CD8^+$ cytotoxic T cells and $CD4^+$ lymphocytes, rendering the T cells incapable of mounting an effective response to tumor antigens. Dr. van der Bruggen et al. showed that addition of Galectin Therapeutics' inhibitor, GM-CT-01, restores the ability of $CD8^+$ T cells to kill tumor cells (see Figure 3 below).

FIGURE 3: GALECTIN THERAPEUTICS, INC. – EFFECTS OF GM-CT-01 ON T CELL FUNCTION



Source: Company reports

In collaboration with Galectin Therapeutics, the Ludwig Institute has scheduled the initiation of a Phase I/II study to evaluate the effects of treatment with GM-CT-01 in combination with an undisclosed tumor-specific peptide vaccine in patients with advanced metastatic melanoma in 2H11. The primary endpoint of the study will be partial or complete response and GM-CT-01 will be administered in between vaccinations. The results will be compared to historical data from patients vaccinated with the peptide vaccine in previous studies, and could provide insight into alternative dosing schedules that would result in synergies with cancer vaccines, such as autologous cell-based vaccines and tumor-antigen-based vaccines, and other immunotherapies such *Yervoy* (ipilimumab).

Clinical Trials with GM-CT-01 (Davanat) as Adjuvant Chemotherapy Preclinical studies conducted in a mouse tumor model showed that, when co-administered with GM-CT-01, more 5-flurouracil (5-FU) accumulates in the tumor versus tumors in mice treated with 5-FU alone. This pointed to potential clinical benefit and safety with the combination. In 2002 Pro-Pharmaceuticals Inc. (as Galectin Therapeutics was known at the time) was granted an IND to evaluate the use of GM-CT-01 (previously referred to as *Davanat*) in combination with 5-FU, as a treatment for late-stage cancer patients with solid tumors.

In 2003 the company initiated a Phase I study to evaluate escalating doses of GM-CT-01 (30-280 mg/m^2) alone and in combination with 5-FU in end-stage patients with advanced solid tumors who failed chemotherapy, radiation therapy, and/or surgical intervention. The study was completed in 2005, and based on objective tumor assessment using RECIST and completion of the second cycle of treatment, the results showed 14 of 26 evaluable patients had stable disease. (According to RECIST, stable disease is treatment response that results in changes that do not meet the criteria for a

partial response, which is a 30% decrease in the sum of the longest diameter of target lesions, or progressive disease, which is a 20% increase in the sum of the longest diameter of target lesions.) Of the patients that received the highest dose of GM-CT-01, 7 of 10 patients had stable disease. The maximum tolerated dose was not reached, despite 5-FU remaining in the bloodstream up to eight times longer when GM-CT-01 is added, supporting the safety and efficacy of the treatment combination and the ability of GM-CT-01 to enhance 5-FU activity.

In 2004 the company initiated a multi-center, open-label Phase II clinical trial to evaluate GM-CT-01 as third- or fourth-line therapy in patients with metastatic colorectal cancer (CRC). The study was designed to measure response rate and stable disease, as well as the safety of GM-CT-01 plus 5-FU. Preliminary results on 20 patients from this trial presented in 2006 showed that the treatment combination extended median progression-free survival to 8.4 weeks, and 43% of evaluable patients showed significant tumor shrinkage. Updated data from 14 patients presented in March 2008 showed that the treatment combination extended median survival by 6.7 months with significantly reduced side effects, as compared to 4.6 months from historical data for best standard of care (BSC). As determined by a patient's physician, in addition to 5-FU BSC collectively includes *Xeloda* (capecitabine), irinotecan, oxaliplatin, *Avastin* (bevacizumab), and *Erbitux* (cetuximab). Two patients survived more than two years with one patient alive at almost four years. Importantly, patients had a 41% reduction in the incidence of 5-FU related side effects, which altogether we believe supports the clinical benefit with GM-CT-01 and potential for quality of life improvement. Select safety data compiled from several studies with GM-CT-01 plus 5-FU are shown in Figure 4 below.

FIGURE 4: GALECTIN THERAPEUTICS, INC. – COMPARISON OF CHEMOTHERAPY-RELATED SIDE EFFECTS

Event in percent of patients (%)	5-FU/LV Studies	5-FU+GM-CT01
	N=1128	N=57
Adverse Events	Grade 3-4 (%)	Grade 3-4(%)
Diarrhea	12-40	0
Nausea/Vomiting	4-9	<2
Mucositis	17-22	<2
Neutropenia/ Leukopenia	7-67	<2

Source: Company reports

In 2006 the company initiated a Phase II trial to evaluate whether treating locally advanced, unresectable or metastatic CRC with GM-CT-01, when added to first-line therapy (5-FU, leucovorin plus Avastin), is beneficial in patients unable to tolerate intensive chemotherapy. The study was designed to evaluate the treatment combination in two monthly cycles until disease progression or toxicity. The study was stopped in 2Q08 due to financial constraints, however, none of the patients that had been treated experienced hematological or gastrointestinal serious adverse events, inpatient hospitalization, persistent or significant disability, or death. Some of the patients completed more than one year of treatment. Due to financial constraints in 2Q08 the company also stopped a study that was designed to evaluate GM-CT-01 plus 5-FU as first-line treatment of patients with biliary cancer.

GM-CT-01 Section 505(b)(2) Registration in the U.S. Galectin Therapeutics explored obtaining accelerated approval in the U.S. by submitting a New Drug Application (NDA) through the Section 505(b)(2) pathway, which under the U.S. Food, Drug & Cosmetic Act, permits the FDA to approve an NDA in part on the basis of published literature, or on a previous finding of safety or effectiveness of a drug. Section 505(b)(2) makes it easier and potentially faster for drug developers to obtain approval of new formulations of drugs based, in part, on proprietary data from the developer of the original drug. A 505(b)(2) application can apply to new chemical entities or to changes to previously approved drugs. Examples of 505(b)(2) applications include changes to dosage forms and routes of administration. In February 2007 Galectin

Therapeutics initiated discussions with the FDA and requested a meeting to discuss data and plans for submitting an NDA, under Section 505(b)(2), for GM-CT-01 (Davanat). In brief, the key events occurred as follows:

- **February 12, 2007** – Requested meeting with the FDA to discuss NDA for co-administration of Davanat with 5-FU to treat cancer patients.
- **February 23, 2007** – Announced meeting with the FDA scheduled for April 11, 2007.
- **April 11, 2007** – Announced receipt of letter from the FDA recommending company provide chemistry, manufacturing and controls (CMC) information necessary to support Davanat NDA submission.
- **May 1, 2008** – Receipt of Drug Master File (DMF) for Davanat from Sigma-Aldrich Fine Chemicals (SAFC).
- **May 20, 2008** – Submission of DMF for Davanat to FDA.
- **September 15, 2008** – Supporting clinical, preclinical and manufacturing data submitted to FDA for Davanat NDA to treat advanced colorectal cancer.
- **October 6, 2008** – Pre-NDA meeting with the FDA scheduled for December 22, 2008.
- **January 29, 2009** – FDA requires company conduct a Phase III trial to demonstrate superiority of Davanat plus 5-FU versus best standard of care used in late-stage CRC.

Galectin Therapeutics estimated a registration trial for Davanat would need to enroll up to 300 patients to demonstrate superiority, however, the company did not have sufficient funds to conduct a large trial. Discussions with the FDA continued regarding the design of a Phase III trial, and in December 2010, Galectin Therapeutics received positive feedback from the FDA on the preliminary design of a pivotal trial with Davanat as adjuvant chemotherapy. As the outcome of the planned GM-CT-01 immunotherapy trial has potential to improve the design and outcome of a pivotal trial, the company deferred the initiation of Phase III pending this data.

Davanat Registration in Colombia While Galectin Therapeutics continued discussions with the FDA to refine the design of a pivotal trial with GM-CT-01 in the U.S., in March 2009 the company licensed exclusive marketing and distribution rights to commercialize GM-CT-01 in Colombia, where it retains its Davanat name, and other Latin American countries, to Procaps S.A., a privately-held pharmaceutical company located in Barranquilla, Colombia. Pursuing more rapid routes of registration and commercialization in other countries are strategies that would provide Galectin Therapeutics additional clinical experience with GM-CT-01, and has potential to provide near-term revenue that could be used to support development programs in the U.S.

Procaps intends to use Davanat's potential to minimize the side effects of chemotherapy drugs to assist a large number of patients in Colombia and other Latin American countries who cannot afford many current cancer treatments or supporting care. As we described above, although not approved for use as a drug or adjuvant, Davanat has been shown to be safe when co-administered with 5-FU in patients with cancer. Procaps believes the use of Davanat in cancer patients and its high potential for approval in Colombia is supported by the FDA's approval of galactomannans for use in the U.S. and elsewhere for a variety of food and non-food purposes, including new formulations of existing drugs. In April 2010 Galectin Therapeutics management met in Colombia with top officials of the government, including Colombia's President, Alvaro Uribe, the country's Vice Minister of Health, Carlos Cuervo Valencia, and Colombia's leading oncology thought leader, Dr. Carlos Rada, who is the Director of Colombia's National Cancer Institute. Colombia's government and healthcare officials place significant importance on improving the quality of life of its people suffering from life-threatening diseases such as cancer, thus, minimizing the toxicities associated with chemotherapy through co-administration of Davanat is a desirable option. The visit was crucial for continuing work on the approval process, and in October 2010 Galectin Therapeutics made its first shipment of Davanat to Procaps for drug stability studies and qualification of its vial filling process. We believe the near-term completion of these pre-commercialization activities, anticipated in 3Q11 and 4Q11, respectively, as well as official approval in 2Q12, would validate the safety of GM-CT-01 for human use, and be significant drivers of the stock.

MANUFACTURING

Galactomannans are used for a wide variety of purposes, such as food stabilizers to increase the viscosity of the water phase in emulsions and in artificial tears, to improve texture and reduce ice cream meltdown, as well as other uses. Galactomannan is a readily sourced carbohydrate that is isolated from seeds of *guar*, a legume that has been cultivated in India for centuries, but today is grown in the U.S. and elsewhere for a variety of food and non-food purposes. In May 2008 Galectin Therapeutics submitted a Drug Master File (DMF) for Davanat to the FDA as a step toward filing a Davanat NDA. A DMF contains confidential detailed information in support of the NDA about facilities, processes or articles used in the chemistry, manufacturing, controls, processing, packaging, and storing or stability of drugs. The DMF demonstrates the company's ability to produce commercial quantities of pharmaceutical-grade Davanat under current Good Manufacturing Process (cGMP) standards. A DMF can be cross-referenced by potential partners to use in combination with other therapies to expedite clinical studies and submission of NDAs. In anticipation of Davanat's commercialization in Colombia, South America, the company in May 2011 signed a multi-year agreement with SAFC, the custom manufacturing business unit of Sigma-Aldrich Corp. (SIAL, Not Rated) for the manufacture of bulk drug substance or active pharmaceutical ingredient for Davanat.

INTELLECTUAL PROPERTY

Galectin Therapeutics retains strong intellectual property positions for composition of matter and fields of use for its carbohydrate technology in chemotherapy with earliest expiration dates beginning in 2020. The patents are registered in the U.S. and include methods and compositions for reducing side effects of chemotherapeutics, delivery of a therapeutic in a formulation to reduce toxicity, co-administration of a polysaccharide with a chemotherapeutic for the treatment of cancer, and others. The company recently extended patent coverage to Australia and Japan, but has other U.S. and other internal patent applications pending.

KEY COLLABORATIVE RELATIONSHIPS

Procaps S.A.

In March 2010 Procaps S.A. was granted exclusive rights to market and sell Davanat to treat cancer in Colombia, South America. Under the terms of the agreement, Procaps, based in Barranquilla, Colombia, is responsible for obtaining regulatory and pricing approval in Colombia, as well as vial filling, packaging, marketing and distribution of Davanat in the region. Galectin Therapeutics retains all intellectual property rights and will be the owner of the regulatory approval of Davanat in the region. Procaps has first negotiation rights to other countries in South and Central American and the Caribbean. Upon its regulatory approval, Galectin Therapeutics will receive a transfer payment for each dose of Davanat shipped to Procaps, and a royalty above a minimum annual sales threshold. In October 2010 Galectin Therapeutics received a $200,000 payment and shipped Davanat to Procaps for testing purposes.

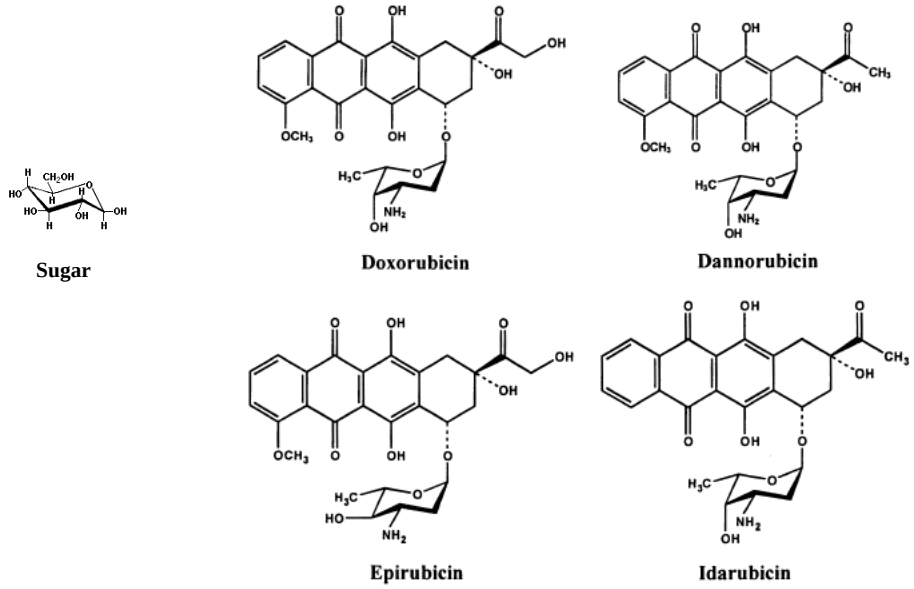
Numoda Corp.

In September 2010 Numoda Corporation was engaged to oversee the Phase III clinical trial of Davanat plus 5-FU in cancer. Although terms of the agreement have not been disclosed, Numoda is expected to complete a complete project plan and budget. After approval of the plan, Numoda will be responsible for helping manage the Phase III trial, including integration, oversight and logistical functions associated with interfacing with clinical research organizations, contract laboratories, and statistical analysis of clinical data.

CARBOHYDRATES AND CHEMICAL STRUCTURE OF DRUGS

Many drugs, such as anthracyclines, macrolides, nucleic acid analogs, and many other classes, contain carbohydrates such as sugars, as part of their chemical structure. Typically, removal of the sugar from the chemical structure of these drugs eliminates their activity. The anthracyclines, doxorubicin, daunorubicin, epirubicin, and idarubicin, for example, are compounds that contain an amino-sugar residue, and are commonly used to treat a broad range of cancers (see Figure 5 below). Doxorubicin's mechanism of action involves interacting with DNA and inhibiting its replication, thereby preventing cell division. When the carbohydrate portion of doxorubicin is removed, the rest of the compound is ineffective in interacting with DNA and interfering with replication.

FIGURE 5: GALECTIN THERAPEUTICS, INC. – CARBOHYDRATES IN THE STRUCTURE OF ANTHRACYCLINES



Source: Company reports

By virtue of its mechanism of action, doxorubicin shows severe toxicities, including cumulative cardiotoxicity, which has resulted in numerous efforts to modify the sugar moiety of doxorubicin in order to reduce its side effects and/or increase its efficacy. One strategy created a derivative with a longer sugar moiety by substituting the amino sugar in doxorubicin with D-galactose, which was chosen because of the important role that galectins play in tumor development. The result was lower cytotoxicity and higher efficacy, without cumulative cardiotoxicity, as shown in a mouse model of leukemia. One galectin-directed doxorubicin derivative evaluated, Davanat (doxo-galactomannan), was comprised of doxorubicin conjugated to polymeric galactomannan, and resulted in 20 to 90 times lower cytotoxicity than doxorubicin. Thus, by modifying the carbohydrates bound to them, the safety and efficacy of drugs can be enhanced, potentially preserving their activity at lower doses. Carbohydrates are a relatively untapped source of new drugs which we believe offer new opportunities to develop novel therapeutics.

BRIEF OVERVIEW OF GALECTINS

Galectins are a subfamily of the sugar-binding lectins, which are proteins that bind β-galactoside, a galactose-containing complex carbohydrate (saccharide). Fifteen galectins have been identified and isolated, but only 12 found in humans. They can be found in all cells and are located most often on the cell surface, with a type of galectin typically expressed at high concentration in a few but different cell types. The diversity of their occurrence is reflected by the multiple biological roles they exhibit in controlling cell adhesion, apoptosis, cell-cell and cell-matrix interactions, proliferation, pre-mRNA splicing, immunity, and inflammation.

Types of Galectins

Galectins found in humans have been classified into three groups according to their structure: prototypical, chimeric, and tandem repeat. However, on the basis of gene sequence homology, two general subgroups are distinguished: the galectin-1 subgroup, which includes galectin-1 and galectin-2, and the galectin-3 subgroup, which includes all others. The table below lists galectins whose activity has been evaluated.

TABLE 1: GALECTIN THERAPEUTICS, INC. – PUTATIVE FUNCTIONS AND ACTIVITY OF GALECTINS

Galectin-1
- involved in Treg cell function and enhances Treg formation
- conflicting results on effects on T-cell viability
- mediates adhesion of thymocytes to thymic epithelium
- induces apoptosis in $CD4^+CD8^+$ double positive thymocytes
- induces shift in Th1 response to Th2 (decreases IFNγ; increases IL-5)
- reduces TNFα, IL-1β, IL-12, IL-2 and IFNγ
- increases IL-10 production in both naive and activated T cells
- inhibits mast cell degranulation
- reduces pathology-associated graft-versus-host disease, Con A-induced hepatitis, experimental allergic encephalomyelitis, myasthenia gravis and rheumatoid arthritis
- reduces acute inflammatory responses
- expression in endothelial cells up-regulated by activation
- induces apoptosis-independent phosphatidylserine (PS) exposure (Ca^{++}-dependent) in neutrophils
- inhibits chemotaxis of neutrophils
- inhibits extravasation of neutrophils
- activates NADPH-dependent respiratory burst in neutrophils
- induces maturation of dendritic cells

Galectin-2
- induces T-cell apoptosis under some conditions
- decreases IFNγ and TNFα while increasing IL-10 and IL-5
- involved in the pathogenesis of atheroma formation
- induces apoptosis-independent PS exposure (Ca^{++}-dependent) of neutrophils

Galectin-4
- induces IL-6 production in T cells
- induces apoptosis-independent PS exposure (Ca^{++}-independent) of neutrophils

Galectin-7
- intracellular expression induces apoptosis of tumor cells
- extracellularly can inhibit growth of cells

Galectin-8
- activates Rac-1 in T cells
- activates NADPH-dependent respiratory burst of neutrophils
- modulates integrin-mediated neutrophil adhesion of neutrophils

Galectin-10
- highly expressed in eosinophils (Charcot-Leyden crystal protein)
- involved in Treg function

Galectin-3
- blocks apoptosis of T cells when overexpressed intracellularly
- endogenously involved in T-cell viability
- extracellularly induces apoptosis of T cells
- promotes adhesion of thymocytes to thymic epithelium
- enhances Th2 immune responses
- enhances adhesion of naive T cells to DCs
- binds TCR, reducing TCR mediated T cell activation
- inhibits IL-5 production in eosinophils
- induces mast cell degranulation independent of antigen-mediated IgE stimulation
- exacerbates Th2 immune responses (asthma)
- expressed on surface of macrophages (also called Mac-2 antigen)
- enhances phagocytosis of macrophages
- enhances respiratory burst of macrophages
- enhances LPS-induced IL-1β secretion of macrophages
- inhibits apoptosis (intracellularly)
- blocks IL-4-induced survival of activated B cells
- favors plasma cell differentiation
- exhibits an anti-apoptotic role in B-cell lymphomas
- expression induced in dendritic cells by *T. cruzi* infection
- enhances pro-inflammatory cytokine release in endothelial cells
- expression up-regulated in tumor endothelial cells
- induces chemotaxis of neutrophils
- enhances extravasation of neutrophils
- activates NADPH-dependent respiratory burst of neutrophils
- induces activation of neutrophils
- induces release of IL-8 of neutrophils
- mediates interaction of neutrophils with laminin and fibronectin (both directly and indirectly)
- enhances leukocyte adhesion to endothelium

Galectin-9
- induces apoptosis in thymocytes and T cells
- induces selective loss of $CD4^+$ Th1 cells
- induces selective loss of $CD8^+$ T cells
- induces eosinophil chemotaxis, activation, superoxide generation
- induces moderate degranulation of eosinophil
- expression in endothelial cells induced by virus infection
- induces maturation of dendritic cells

Galectin-12
- intracellular expression induces apoptosis of tumor cells
- can cause cell cycle arrest and growth suppression

Source: Company reports

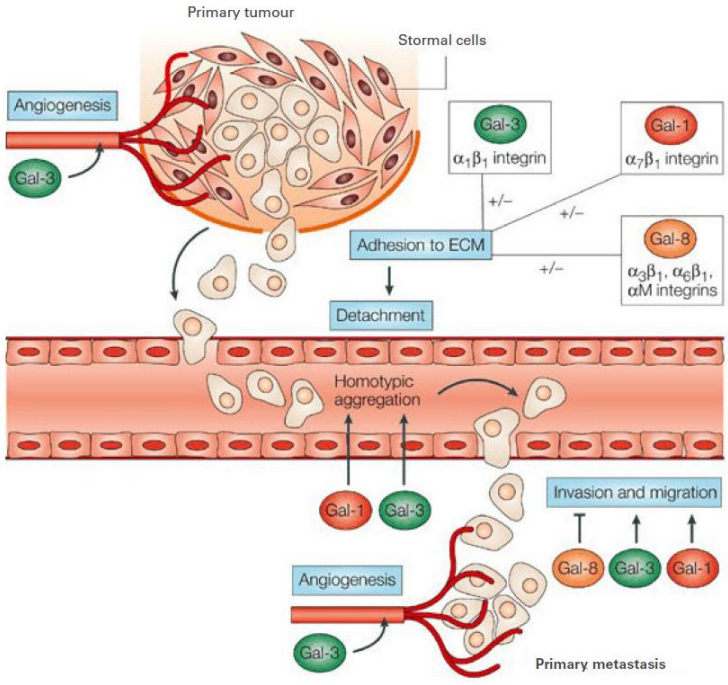
Galectin Function

Although the precise role of the interaction is not well understood, all galectins contain at least one carbohydrate recognition domain (CRD), or carbohydrate-binding site, with an underlying mode of action most often, but not always, related to interaction with the carbohydrate moiety of glycoproteins. A common function of galectins, for example, is to cross-link structures containing N-acetyl-lactosamine located at the cell surface and within the extracellular matrix. Therefore, based on studies on the type of galectin and type of cell in which it is expressed, it has been proposed that galectins have the following general functions:

- Bind to cell surface and matrix glycoproteins (galactose residues)
- Modulate cell signaling
- Promote cell-cell interactions
- Promote cell-matrix interactions

These indicate galectins potentially have diverse effects on many cellular functions including adhesion, migration, polarity, chemotaxis, proliferation, apoptosis, and differentiation. Galectins may therefore play a key role in many pathological states, including autoimmune diseases, allergic reactions, inflammation, tumor growth and metastasis, atherosclerosis, and diabetic complications. (For the purpose of brevity we focus only on the proposed role of galectins in cancer, fibrosis, and immunity in this report.)

FIGURE 6: GALECTIN THERAPEUTICS, INC. – PUTATIVE ROLES OF GALECTINS IN CANCER



Source: Company reports

Therapeutic Applications

Cancer Many types of tumors, such as melanomas, astrocytomas, and bladder and ovarian tumors, overexpress various galectins, and their heightened expression usually correlates with clinical aggressiveness of the tumor and the progression to a metastatic phenotype. Three galectins that have shown importance in cancer progression and metastasis are galectin-1, -3, and -8 (see Figure 6 above). As revealed by knockout studies, the immunosuppressive and apoptotic effects of galectins can contribute to tumor survival. Decreased galectin-1 expression, for example, results in

increased survival of IFN-γ-producing Th1 cells and heightened T-cell-mediated tumor rejection, and is associated with tumor regression. Recent studies using galectin-1 knockout cells have shown that expression of galectin-1 in tumor cell endothelium is essential for tumor angiogenesis. Similarly, studies suggest that overexpression of galectin-3 correlates well with neoplastic transformation and tumor progression toward metastasis, and may possibly be a histological tumor biomarker. Other studies suggest that blocking galectin-3 function may limit tumor metastasis, and thus, making this the subject of much study as a potential target for drug development. Therefore, as galectins are likely to play important roles in tumor progression and metastasis through indirect effects on the regulation of tumor immune response and direct effects in tumor angiogenesis, the advancement of a galectin inhibitor could have broad applications in treating cancer.

Fibrosis Several diseases, such as chronic hepatitis, alcoholic liver disease, non-alcoholic fatty liver, lead to liver fibrosis which results in serious medical consequences. Macrophages are a good example of immune cells that are intrinsically involved in tissue scarring and remodeling, and cells of the innate and adaptive immune system are known to play an important role in tissue fibrosis. Understanding the complex interplay between immune cells and tissue fibroblasts and elucidation of the mechanisms that drive the switch from fibroblasts to myofibroblasts could provide a strategy for development of targeted antifibrotic therapies.

Studies of the pathogenesis of tissue fibrosis identified a number of cells as central to fibrogenesis and important in the evolution of tissue scarring. The scarring of organs, for example, results from the activation of fibroblasts into matrix-secreting myofibroblasts. Galectin-3 expression is up-regulated in established human fibrotic liver disease, and could be a novel target for developing antifibrotic therapy, and therefore, has been the subject of much drug development. As was shown in a knockout mouse model, for example, galectin-3 is essential in mice for the development of liver fibrosis, and is produced in large amounts by fibrotic livers in humans. Importantly, galectin inhibitors reversed fibrosis in an experimental chemical toxin-induced rat model of fibrosis, and blocked production of fibrogenic markers in cultured key human cells responsible for liver fibrosis. Human studies now indicate that fibrosis and even cirrhosis could be reversible, especially if the underlying disease is eradicated. As liver transplantation is the only treatment option for patients with advanced fibrosis, the advancement of a galectin inhibitor could target a large market opportunity.

Immune Response and Inflammation Accumulating evidence indicates that galectins play critical regulatory roles in immune cell response and homeostasis. Galectins, for example, are expressed by activated T and B cells, regulatory T cells, dendritic cells, mast cells, eosinophils, monocytes/macrophages, and neutrophils, and promote pro- or anti-inflammatory responses, depending on the inflammatory stimulus, microenvironment, and target cells.

Galectin-1 function is generally associated with attenuating inflammatory responses. It can induce some anti-inflammatory cytokines, such as interleukin-5 (IL-5), IL-10, and transforming growth factor-β (TGF-β). Galectin-1 also can inhibit production of pro-inflammatory cytokines, such as IL-2, tumor necrosis factor-α (TNF-α), and interferon-γ (IFN-γ). Studies point to differential glycosylation of T-helper cells, and their differential responses to galectins, as determinants of the overall immune response. Interestingly, subcutaneous injections of recombinant human galectin-1 can reduce the severity of disease in several animal models of autoimmune disease, including experimental autoimmune encephalomyelitis, experimental autoimmune myasthenia gravis, and collagen-induced arthritis.

In contrast, galectin-3 appears to have a pro-inflammatory role. It has been associated with activation of T cells, perhaps by interacting with specific N-glycans on the T-cell receptor (TCR). Expression of these N-glycans is partly regulated by branching, which normally restricts TCR clustering, which hinders the development of a T-cell response. Galectin-3 can also inhibit IL-5 production in several immune cells, including human eosinophils. On the other hand, galectin-3 can activate mast cells, neutrophils, and monocytes, mediator release, and production of reactive oxygen species. Galectin-3 knockout mice exhibit reduced mast cell function, reduced accumulation of asthma-associated leukocytes in airway inflammation, and reduced peritoneal inflammatory responses. Endogenous galectin-3 has also been shown to play a role in phagocytosis by macrophages, mediator release, and cytokine production by mast cells. Overall, study results suggest a complex set of functions for galectin-3, however, one that presents opportunities for the development of novel immunotherapies.

LIVER FIBROSIS

Hepatic, or liver, fibrosis refers to the accumulation of interstitial or 'scar' extracellular matrix after either acute or chronic liver injury. Cirrhosis, the end-stage of progressive fibrosis, is characterized by the formation of rings of scar tissue that surround nodules of hepatocytes. The increasing burden of liver disease in the U.S. in part reflects a rising prevalence of cirrhosis among patients with chronic hepatitis C virus infection (HCV). The worldwide burden is equally compelling, with chronic liver disease, which affects hundreds of millions of individuals, and is associated with an accelerated risk of liver cancer (hepatocellular carcinoma). Also contributing to this burden is the explosive growth in obesity worldwide, which has led to a dramatic increase in the prevalence of non-alcoholic steatohepatitis (NASH), which also confers a risk of HCC once cirrhosis develops. HCC is the fastest rising incidence of any cancer in the U.S. and Western Europe. Despite significant recent progress, no drug has yet emerged as an effective antifibrotic agent in humans.

Efforts at understanding the underlying mechanism of liver fibrosis have focused on the stellate cell in the liver, as these cells can undergo activation into a state of proliferation and change into fibrogenic myofibroblast-like cells during liver injury. Some of the mechanisms regulating fibrosis that have been studied include:

- Necrosis resulting from a perturbation of normal liver homeostasis caused by injury
- Apoptosis generates hepatocyte fragments which have been found to be fibrogenic
- Hepatic stellate cells and sinusoidal endothelial cells as inflammatory effectors of angiogenesis
- Lymphocytes, as tissue infiltration is a major feature of many forms of chronic disease
- Growth factors, as they regulate stellate cell activation and local fibrogenic activity
- Transcription factors and signaling pathways (too many to detail here)
- Hepatitis C virus, which induces stellate cell proliferation and release of inflammatory signals such as TGF-β
- NASH, possibly due to downregulation of adiponectin in obese patients

Conditions in which cirrhosis develops over months include pediatric liver disease (e.g. biliary atresia), drug-induced liver disease, and viral hepatitis associated with immunosuppression after liver transplantation. Typically, fibrosis requires years or decades to become clinically apparent. The demonstration that liver fibrosis, and perhaps even cirrhosis, may regress, such as in patients with HBV and HCV, has accelerated enthusiasm for developing antifibrotic therapies. As a result, there is tremendous activity in evaluating investigational agents as treatments for liver fibrosis, with potential for significant progress in the near future.

Current Treatment Approaches

The most effective and direct current treatment for liver fibrosis is liver transplantation, and only a portion of a liver is needed for successful transplantation. According to the Scientific Registry of Transplant Recipients, which compiles an annual report that lists information related to transplant registration, there were 11,262 new patients registered for liver transplants in 2009. However, individuals registered for liver transplant face the issues of matching donor tissue type, blood type, and others, in order for the transplant to be effective, as well as evaluations for physical and emotional health. As a result, there were only 6,101 liver transplants from cadaver donors and 219 liver transplants from living donors performed in 2009, with 16,365 people on the waitlist at the end of 2009.

According to the United Network for Organ Sharing (UNOS), estimated charges for liver transplantation include:

- Estimated first-year charges of $314,000.
- Estimated annual follow-up cost of $21,900.
- Estimated cost of drugs to sustain good condition of transplanted liver of $30,000-$33,000.

The costs of an organ transplant involve transplant evaluation and testing, transplant surgery and follow-up care, and medication. Even before a transplant procedure is done, medical expenses for treatment by a physician, hospital, laboratories and medical specialists are incurred, thus, the costs associated with a liver transplant add up quickly and few are able to pay all of the costs using a single source.

Therapeutic Approach The liver offers unique opportunities for targeting because orally administrated agents are metabolized first in liver and need not reach extra-hepatic tissues, potentially allowing for lower, safer dosages, therefore, antifibrotic therapies might require only intermittent rather than continuous administration. Drugs that are most likely to reach clinical trial first are those that have already been approved for other indications, have a clear rational basis for use in liver fibrosis, and have amassed extensive safety data. However, once proof-of-principle for any antifibrotic is achieved, it is likely that interest in the development of similar agents will rapidly increase. As a result, the area of antifibrotics is an attractive and large opportunity for the developer of the first safe and effective therapeutic in what is a rapidly advancing field.

Multiple targets have been identified and many antagonists are being tested in cell culture and animal models. The 'optimal' antifibrotic would eradicate the underlying cause of liver disease wherever possible, for example, eradicate hepatitis B or C virus infection, or inhibit virus replication that leads to reversal of fibrosis, even in patients with histological cirrhosis. Early studies show fibrosis reverts in patients with hemochromatosis during iron depletion, after corticosteroid therapy in autoimmune hepatitis, and in patients with secondary biliary cirrhosis after decompression of bile duct obstruction. Other approaches include reducing hepatic injury and inflammation, attenuating stellate cell activation by manipulating intracellular signaling and transcriptional pathways, antagonizing cytokines and growth factors that drive stellate cell activation, provoking apoptosis of activated stellate cells, or accelerating degradation of extracellular matrix. Several agents have overlapping activities and many are nearing clinical trials. Finally, it is important to realize that for an antifibrotic drug to be successful, it need not eradicate hepatic fibrosis entirely, as the liver has an enormous functional reserve and thus most patients with fibrosis have normal liver function. Instead, any drug that sufficiently attenuates fibrosis progression and prevents the development of cirrhosis, and/or hepatocellular carcinoma, would be viewed as a success.

Many liver diseases, such as HCV, have an inflammatory component. Oxidative stress is also thought to play an important role in injury, stellate cell activation, and the stimulation of extracellular matrix production. Lastly, fibrogenesis requires collagen secretion, therefore, the approaches to treating liver fibrosis are varied and have included:

- Remove injurious agent (eradication of hepatitis virus with antiviral drugs)
- Anti-inflammatory agents (corticosteroids)
- Antioxidants (polyenylphosphatidylcholine in alcoholic hepatitis)
- Cytoprotective agents (ursodeoxycholic acid)
- Inhibit stellate cell activation (interferon-γ)
- Inhibit fibrogenesis (colchicine)

Given the current major effort in understanding the biology of liver fibrosis, it is not surprising that numerous pathways have been targeted as having therapeutic potential, and many compounds have been studied in experimental models and shown to have antifibrotic properties, however, there is still lack of a proof-of-principle clinical trial that establishes the value of attacking fibrosis directly.

The choice of patients and endpoints for clinical trials of antifibrotic drugs also presents a conundrum. On the one hand, antifibrotics are more urgently needed in patients with cirrhosis and are more likely, if successful, to yield improved survival and reduced morbidity. On the other hand, cirrhosis is probably less reversible than earlier stages of fibrosis and would likely require longer treatment. As a result, cirrhotic patients would likely be less able to tolerate any hepatotoxicity specific to an investigational agent. They are also at higher risk for hepatocellular cancer, therefore, antifibrotic therapies would need to be safe without long-term toxicity or increased risk of hepatocellular cancer. Investigational agents could potentially advance more rapidly if early-stage trials are aimed initially at 'soft' endpoints, such as noninvasive biomarkers or demonstrating the capacity for matrix resorption. Such a design would obviate the need to rely on liver biopsy and insistence that complete reversal of fibrosis or improved morbidity and mortality be documented, which obligate the conduct of a lengthy trial, increasing its cost and limiting the enthusiasm of sponsors. Findings from such studies should support the rationale for longer, larger trials with 'hard' clinical endpoints, such as survival and decreased complications.

Market Opportunity for Galectin Inhibitors in Liver Fibrosis

In part because of the large number of such patients and their well characterized natural history, current efforts in treating fibrosis focus mainly on patients with advanced fibrosis or cirrhosis who have failed antiviral therapies for hepatitis. According to the Centers for Disease Control and Prevention (CDC), there are 3.9 MM people in the U.S. with HCV. Recent studies showed that 11% of these people having advanced cirrhosis. Finally, a study using biomarkers estimated a prevalence of 2.8% of the general population 40 years and older have advanced fibrosis. We do not include revenues for Galectin Therapeutics' investigational agents in our models, however, we believe a galectin inhibitor development program in liver fibrosis targets a large market opportunity as:

- Liver fibrosis represents a large unmet medical need
- Galectin-3 shows promise as a target in models of liver fibrosis
- Galectin inhibitors reverse liver fibrosis in animals and show efficacy in human cell culture models of fibrosis
- Galectin inhibitors are non-toxic with little potential for drug interaction
- There is potential for rapid clinical development pathways
- There is potential for Orphan Disease status, Fast Track designation, Priority review, and Accelerated Approval process

Galectin Therapeutics near-term intends to advance its galectin inhibitors further into testing in humans, initially in post-organ transplant patients with recurrent HCV, where Orphan Disease designation is possible. Additionally, the company may in the future explore the potential of its galectin inhibitors in peri-transplant indications, such as in patients with established cirrhosis of various etiologies but are not eligible for liver transplantation, but also chronic HCV fibrosis, chronic hepatitis B virus infection liver fibrosis, alcoholic fibrosis, and renal fibrosis. We anticipate the submission of an IND to evaluate GM-CT-01 for liver fibrosis in 4Q11, followed by initiation of Phase II evaluation in mid-2012.

EXECUTIVE LEADERSHIP

James C. Czirr, Executive Chairman Mr. Czirr was appointed a director and became Chairman of the Board of Directors in February 2009, and Executive Chairman of the Board in February 2010. He is a co-founder of 10X Fund, L.P. and is a managing member of 10X Capital Management LLC, the general partner of 10X Fund, L.P. Mr. Czirr co-founded Pro-Pharmaceuticals in July 2000. He was instrumental in the early stage development of Safe Science Inc., a developer of anti-cancer drugs, served from 2005 to 2008 as Chief Executive Officer of Minerva Biotechnologies Corporation, a developer of nanoparticle bio chips, and was a consultant to Metalline Mining Company Inc., a mineral exploration company focused on low-cost production of zinc. Mr. Czirr received a B.B.A. degree from the University of Michigan.

Peter G. Traber, President, Chief Executive Officer, and Chief Medical Officer Dr. Traber was appointed President and CEO in March 2011, and was appointed Chief Medical Officer (CMO) in June 2010. He is also President Emeritus of Baylor College of Medicine, where he was Chief Executive Officer from 2003 to 2008. From 2000 to 2003, Dr. Traber was Senior Vice President of Clinical Development and Medical Affairs and CMO of GlaxoSmithKline plc (GSK, Not Rated). He served as CEO 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 its patented technology. From 1996 to 2000, he was Vice President of Research and Development for Kadant Composites, Inc., where he directed a laboratory specializing in biochemistry, microbiology and polymer engineering. From 1990 to 1998, Dr. Klyosov was a Visiting Professor of Biochemistry in the Center for Biochemical and Biophysical Sciences at Harvard Medical School. From 1981 to 1990 he was Professor and Head of the Carbohydrates Research Laboratory at the A.N. Bach Institute of Biochemistry, U.S.S.R. Academy of Sciences. Dr. Klyosov was elected to the World Academy of Art and Science and is the recipient of distinguished awards including the U.S.S.R. National Award in Science and Technology. He holds over 20 patents, has published more than 250 peer-reviewed articles in scientific journals, written books on enzymes, carbohydrates, and biotechnology, and edited two books. 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.

Anthony D. Squeglia, Chief Financial Officer and Vice President of Investor Relations Mr. Squeglia was appointed Chief Financial Officer in October 2007, and VP of Investor Relations in 2003. He was a Partner from 2001 to 2003 at JFS Advisors, a management consulting firm that delivers strategic services to entrepreneurial businesses, including raising capital, business planning, positioning, branding, marketing and sales channel development. From 1996 to 2001, Mr. Squeglia was Director of Investor Relations and Corporate Communications for Quentra/Coyote Networks, and previously held management positions with Summa Four, Unisys, AT&T, Timeplex, Colonial Penn and ITT. Mr. Squeglia received an M.B.A. from Pepperdine University and a B.B.A. from The Wharton School, University of Pennsylvania.

Eliezer Zomer, Ph.D., Executive Vice President of Manufacturing and Product Development Dr. Zomer was appointed EVP of Manufacturing and Product Development in 2003. He was the founder of Alicon Biological Control, where he served from 2000 to 2002. Dr. Zomer was VP of Product Development at SafeScience, Inc., from 1998 to 2000, and from 1987 to 1998, was VP of Research and Development at Charm Sciences, Inc. He received a B.Sc. degree in Industrial Microbiology from the University of Tel Aviv, a Ph.D. in Biochemistry from the University of Massachusetts, and completed his post-doctoral study at the National Institutes of Health.

Maureen E. Foley, Chief Operating Officer and Corporate Secretary Ms. Foley has 30 years of business and operations management experience at startup companies, including facility design, construction and fit out, project management, IT, HR, press and public relations, accounting and finance. Between 1999 and 2000 she managed business operations for eHealthDirect, Inc. and ArsDigita, Inc. From 1996 to 1999, Ms. Foley was Manager of Operations at Thermo Fibergen, Inc., a subsidiary of Thermo Fisher Scientific, Inc. (TMO, Not Rated). Ms. Foley is a graduate of The Wyndham School, Boston, Massachusetts, with a major in Mechanical Engineering.

VALUATION

Biotech companies with mid- to late-stage clinical development products currently trade at a mean enterprise value (EV) of $127 MM (see Figure 7), which is more than two times the current $61 MM EV of Galectin Therapeutics. By using the comparable company group mean EV, dividing by GALT's EV, and multiplying by the number of GALT shares outstanding, we derive a value of $2.25 per share for the galectin inhibitor program.

FIGURE 7: GALECTIN THERAPEUTICS, INC. – COMPARABLE MID-STAGE DEVELOPMENT COMPANIES

Company	Ticker	Price 9/2/11	SOS[1]	Mkt. Cap. ($ MM)	Cash[2] Total	Cash[2] /Share	Debt[3] Total	Debt[3] /Share	Enterprise Value Total	Enterprise Value /Share
ARQULE, INC.	ARQL	$4.11	54	**221**	84	$1.57	2	$0.00	**138**	2.58
ARRAY BIOPHARMA, INC.	ARRY	$2.16	57	**123**	64	$1.12	92	$1.61	**151**	2.65
CELL THERAPEUTICS, INC.	CTIC	$1.19	193	**230**	39	$0.20	12	$0.06	**203**	1.05
CURIS, INC.	CRIS	$2.88	77	**220**	33	$0.43	0	$0.00	**188**	2.45
GTX, INC.	GTXI	$3.35	63	**210**	91	$1.45	0	$0.00	**119**	1.90
INFINITY PHARMACEUTICALS, INC.	INFI	$6.89	27	**183**	82	$3.07	0	$0.00	**102**	3.82
KERYX PHARMACEUTICALS, INC.	KERX	$3.85	70	**268**	53	$0.75	0	$0.00	**216**	3.10
SANGAMO BIOSCIENCES, INC.	SGMO	$5.19	52	**272**	91	$1.74	0	$0.00	**181**	3.45
SYNTA PHARMACEUTICALS CORP.	SNTA	$3.84	49	**190**	63	$1.27	17	$0.34	**144**	2.91
TALON THERAPEUTICS, INC.	TLON	$0.96	22	**21**	11	$0.48	24	$1.09	**34**	1.56
Median				**215**	63	$1.20	1	$0.00	**147**	$2.61
Mean				**194**	61	$1.21	15	$0.31	**148**	$2.55
GALECTIN THERAPEUTICS, INC.	**GALT**	**$0.93**	**74**	**69**	**8**	**$0.11**	**0**	**$0.00**	**61**	**$0.82**

All numbers are in $ millions except per share data. EV = enterprise value

[1]Shares outstanding (SOS) as of most recent reported quarter adjusted for effects of recent financing activities.

[2, 3]As of most recent reported 10-Q or 8-K filing.

GALT Percent of Mean -65% **GALT Percent of Mean** -59%

Comparable EV Multiple (Mean/GALT) 2.4

Target Price (comparable EV divided by GALT shares out) $2.25

Source: Company reports, FactSet and DJSI research.

We believe Galectin Therapeutics, however, has a near-term revenue opportunity, i.e., Davanat in Colombia, South America. We conducted a sensitivity analysis of 2015 discounted projected earnings and price-to-projected sales and derived an implied value of $4 to $7 for GALT shares (see Figure 8 below). With GALT shares trading at a discount to its peers and unrecognized value for Davanat, we therefore rate the shares a Buy and set a 12-18 month price target of $4.

FIGURE 8: GALECTIN THERAPEUTICS, INC. – SENSITIVITY ANALYSIS OF 2015 DISCOUNTED EARNINGS AND SALES

Sensitivity to Discounted Earnings	
2015 EPS	$0.53
P/E Multiple	35
Discount Rate	35%
Discount Years (YE1	3.25
Value per Share	$7.04

Sensitivity to Price-to-Sales Multiple	
2015 Sales ($MM)	$232
Sales Multiple	4
Discount Rate	35%
Discount Years (YE1	3.25
Shares Out (YE12)	85
Value per Share	$4.12

	Discount Rate				
	25%	30%	35%	40%	45%
P/E Multiple					
20	$5	$5	$4	$4	$3
25	$6	$6	$5	$4	$4
30	$8	$7	$6	$5	$5
35	$9	$8	**$7**	$6	$6
40	$10	$9	$8	$7	$6
45	$12	$10	$9	$8	$7
85	$22	$19	$17	$15	$14

	Discount Rate				
	25%	30%	35%	40%	45%
Price/Sales Multiple					
2	$3	$2	$2	$2	$2
3	$4	$3	$3	$3	$2
4	$5	$5	**$4**	$4	$3
5	$7	$6	$5	$5	$4
6	$8	$7	$6	$5	$5
7	$9	$8	$7	$6	$6
8	$11	$9	$8	$7	$7

Source: Company reports, FactSet and DJSI research.

FINANCIAL OUTLOOK

We believe Galectin Therapeutics has valuable carbohydrate technology and promising drug candidates, however, due to their mid-stage nature, we do not anticipate potential revenue contributions from them. We tentatively include potentially revenues in 2015 from Davanat in Colombia that could meaningfully offset operating expenses. With the potential for Davanat approval in Colombia, we include moderate revenues the late 2011-2012 timeframe in our models.

We estimate R&D expenses will primarily be driven by development costs for advancing GM-CT-01 and other GM series compounds in fibrosis, which we anticipate could enter Phase II testing in mid-2012, resulting in accelerating operating expenses from 2012 onward. We have modeled financing activities in the years, 2011–2015, to coincide with the demonstration of value-creating clinical milestone results and favorable market conditions.

We project Galectin Therapeutics will record a loss of $11.0 MM or ($0.15) per share in 2011. The company has not provided guidance regarding its operating expenses, therefore, our estimates use the best available information only. With $8 MM in cash as of June 30, 2011, we project the company will need to raise cash by year-end 2011. Based on our revenue and expense projections, we estimate Galectin Therapeutics will not become profitable for some time, but may potentially realize the company's first quarter of profitability in 2014, driven by accelerating sales of Davanat in Latin America. We view the potential for licensing and milestone payments from a partnership for Davanat development in the U.S., as upside to our projections.

FIGURE 9: GALECTIN THERAPEUTICS, INC. – KEY MILESTONES

Date	Milestone
3Q11	Exercise by Procaps SA of option to submit Davanat for approval in Colombia as a therapy to improve chemotherapy toxicity
4Q11	Initiation of a Phase I/II study by the Ludwig Institute to evaluate GM-CT-01 immunotherapy in patients with advanced melanoma
4Q11	Conduct pre-IND meeting with the FDA to discuss design of a Phase I study to evaluate GM-CT-01 in patients with liver fibrosis
4Q11	Submission by Procaps SA of permission to commercialize Davanat in Colombia with chemotherapy in cancer patients
4Q11	Submission of an IND to evaluate GM-CT-01 in patients with liver fibrosis
2Q12	Initiation of a Phase II study to evaluate GM-CT-01 in patients with liver fibrosis
2Q12	Formal regulatory permission to commercialize Davanat in Colombia with chemotherapy in cancer patients
3Q12	Initiation of a Phase I study to evaluate a GR series compound in patients with liver fibrosis
2012	Potential confirmation by FDA of Phase III trial design to evaluate GM-CT-01 plus 5-FU vs. best standard of care
2013	Announce topline results from Phase II study to evaluate GM-CT-01 in patients with liver fibrosis
2013	Announce topline results from Phase I evaluation of GM-CT-01 in patients with advanced melanoma

Source: Company reports

INVESTMENT RISKS

The key risks are:

- **Development Risk** Although GM-CT-01 (Davanat) previously has demonstrated promising results in combination with 5-FU, there is no guarantee that it will be successful in future and ongoing clinical trials. Further, with the initiation of a GM-CT-1 pivotal trial awaiting results of the Phase I/II immunotherapy study by the Ludwig Institute, there is moderate risk in delay with further advancement of GM-CT-01. Additionally, as there is lack of a proof-of-principle clinical trial that establishes the value of attacking fibrosis directly, including the wide acceptance of biomarkers for fibrosis, advancement of GM-CT-01 would enter still unchartered drug development territory.
- **Regulatory Risk** As the FDA has required the company conduct a Phase III trial comparing GM-CT-1 plus 5-FU versus best standard of care used in cancer, the demonstration of superiority by GM-CT-1 plus 5-FU does not guarantee the agency will approve the use of the combination based on the results.
- **Commercialization Risk** Given the company is relying on Procaps S.A. to move ahead with commercialization of Davanat in Colombia, which has its own regulatory approval process that is different from the FDA, there is risk associated Davanat commercialization in countries outside the U.S. As the company currently does not have the financial resources to advance GM-CT-1 into a registration trial in the U.S., we believe collaborating with an experienced partner would help mitigate commercialization risk.
- **Financial Risk** Based on our analysis of the historical costs of clinical trials, we anticipate the cost to conduct the planned mid-stage clinical trials with GM-CT-1 ranges from $15MM to $25 MM. As by our estimates, the company currently has cash reserves adequate to finance operating burn into 2012, we believe the company needs to conduct successful financing activities every year through 2015 in order to fund operations and achieve value-creating clinical milestones.
- **Market Risk** Galectin Therapeutics shares are offered by the Nadaq but listed on the over-the-counter (OTC) bulletin board (BB). OTCBB stocks are not considered especially large or stable and considered very risky. Because these stocks tend to trade infrequently, their share price is also more volatile. GALT, however, is required to file current financial statements with the SEC, and as such, we believe the company can meet our, and investors', requirements for transparency and provide information adequate for an appropriate analysis of investment value and risk. As the shares previously traded on the AMEX, we anticipate continued GALT share price appreciation and potential future listing on the Nasdaq or AMEX.

FIGURE 10: GALECTIN THERAPEUTICS, INC. – QUARTERLY INCOME STATEMENT

Income Statement ($MMs) Fiscal Year Ends December 31	2009A	1QA	2QA	3QA	4QA	2010A	1QA	2QA	3QE	4QE	2011E
Product Revenues	**$ 0.0**	**$ 0.0**	**$ 0.0**	**$ 0.0**	**$ 0.0**	**$ 0.0**	**$ 0.0**	**$ 0.0**	**$ 0.0**	**$ 0.0**	**$ 0.0**
DAVANAT (Colombia)	-	-	-	-	-	-	-	-	-	-	-
GM-CT-01 (DAVANAT, U.S.)	-	-	-	-	-	-	-	-	-	-	-
Total Revenue	**0.0**	**-**	**-**	**-**	**-**	**0.0**	**-**	**-**	**-**	**-**	**0.0**
Licensing, Royalties and Other Revenue	-	-	-	-	-	-	-	-	-	-	-
Cost of Goods Sold	-	-	-	-	-	-	-	-	-	-	-
Gross Profit	**0.0**	**-**	**-**	**-**	**-**	**0.0**	**-**	**-**	**-**	**-**	**0.0**
R&D Expenses	1.1	0.1	0.2	0.3	0.4	1.1	0.7	1.3	1.3	1.5	4.9
G&A Expenses	5.0	0.9	1.1	0.9	0.9	3.8	1.3	1.7	1.7	1.8	6.5
Total Operating Expenses	**6.1**	**1.0**	**1.4**	**1.2**	**1.3**	**4.9**	**2.0**	**3.0**	**3.0**	**3.3**	**11.3**
Income (Loss) from Operations	**(6.1)**	**(1.0)**	**(1.4)**	**(1.2)**	**(1.3)**	**(4.9)**	**(2.0)**	**(3.0)**	**(3.0)**	**(3.3)**	**(11.3)**
Interest Income	0.0	-	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Change in Fair Value of Warrant Liability	(1.4)	(1.1)	(0.3)	0.1	0.1	(1.2)	(0.4)	(0.1)	0.0	0.4	(0.1)
Other Income (Expense), net	(1.4)	(1.1)	(0.3)	0.1	0.6	(0.7)	(0.4)	(0.1)	0.0	0.4	(0.1)
Net Income (Loss), B4 Taxes	**(7.5)**	**(2.1)**	**(1.7)**	**(1.1)**	**(0.7)**	**(5.6)**	**(2.4)**	**(3.1)**	**(3.0)**	**(2.9)**	**(11.4)**
Preferred Stock Dividends and Accretion Costs	(2.0)	(0.7)	(0.8)	(0.8)	(0.8)	(3.1)	(0.3)	(0.8)	0.8	0.8	0.5
Income Tax Expense (Benefit)	-	-	-	-	-	-	-	-	-	-	-
Net Income (Loss)	**(9.4)**	**(2.8)**	**(2.5)**	**(1.9)**	**(1.5)**	**(8.7)**	**(2.7)**	**(3.9)**	**(2.2)**	**(2.1)**	**(11.0)**
EPS, Fully Diluted	**($0.20)**	**($0.06)**	**($0.05)**	**($0.03)**	**($0.02)**	**($0.15)**	**($0.04)**	**($0.06)**	**($0.03)**	**($0.03)**	**($0.15)**
Wt. Avg. Shares Outstanding, Diluted	48.3	49.9	53.9	58.8	61.8	56.3	66.9	69.5	72.3	77.0	71.4

All figures in millions except, per share numbers
Source: Company Reports, DJSI Research.

FIGURE 11: GALECTIN THERAPEUTICS, INC. – ANNUAL INCOME STATEMENT

Income Statement ($MMs) Fiscal Year Ends December 31	2009A	2010A	2011E	2012E	2013E	2014E	2015E
Product Revenues	**$ 0.0**	**$ 0.0**	**$ 0.0**	**$ 4.1**	**$ 7.0**	**$ 48.9**	**$ 232.2**
DAVANAT (Colombia)	-	-	-	4.1	7.0	48.9	146.7
GM-CT-01 (DAVANAT, U.S.)	-	-	-	-	-	-	85.5
Total Revenue	**0.0**	**0.0**	**0.0**	**14.1**	**12.0**	**58.9**	**257.2**
Licensing, Royalties and Other Revenue	-	-	-	10.0	5.0	10.0	25.0
Cost of Goods Sold	-	-	-	1.0	1.7	12.2	53.8
Gross Profit	**0.0**	**0.0**	**0.0**	**13.0**	**10.2**	**46.7**	**203.4**
R&D Expenses	1.1	1.1	4.9	7.1	13.4	22.1	30.5
G&A Expenses	5.0	3.8	6.5	9.5	12.0	18.9	26.7
Total Operating Expenses	**6.1**	**4.9**	**11.3**	**16.5**	**25.5**	**41.0**	**57.2**
Income (Loss) from Operations	**(6.1)**	**(4.9)**	**(11.3)**	**(3.5)**	**(15.2)**	**5.6**	**146.2**
Interest Income	0.0	0.0	0.0	0.2	0.6	1.1	3.2
Change in Fair Value of Warrant Liability	(1.4)	(1.2)	(0.1)	(1.6)	(1.6)	(3.2)	(6.4)
Other Income (Expense), net	(1.4)	(0.7)	(0.1)	(0.1)	(0.1)	(2.1)	(3.2)
Net Income (Loss), B4 Taxes	**(7.5)**	**(5.6)**	**(11.4)**	**(3.6)**	**(15.3)**	**3.5**	**143.0**
Preferred Stock Dividends and Accretion Costs	(2.0)	(3.1)	0.5	3.2	3.2	3.2	3.2
Income Tax Expense (Benefit)	-	-	-	7.0	6.0	29.5	85.9
Net Income (Loss)	**(9.4)**	**(8.7)**	**(11.0)**	**(7.4)**	**(18.1)**	**(22.7)**	**60.4**
EPS, Fully Diluted	**($0.20)**	**($0.15)**	**($0.15)**	**($0.09)**	**($0.18)**	**($0.21)**	**$0.53**
Wt. Avg. Shares Outstanding, Diluted	48.3	56.3	71.4	85.1	101.5	109.6	113.1

All figures in millions except, per share numbers
Source: Company Reports, DJSI Research.

IMPORTANT DISCLOSURES:

Price Chart

Price target and ratings changes over the past 3 years:
Initiated – September 5, 2011 – Buy – Target $4

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	Company Coverage		Investment Banking	
Ratings Distribution	# of Companies	% of Total	# of Companies	% of Totals
Buy	26	84%	8	31%
Neutral	4	13%	3	75%
Sell	1	3%	0	0%
Total	32	100%	11	35%

Information about valuation methods and risks can be found in the "VALUATION" and "RISKS" sections of this report.