UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
Washington, D.C. 20549

FORM 8-K

CURRENT REPORT

Pursuant to Section 13 or 15(d) of the
Securities Exchange Act of 1934

January 9, 2012
Date of Report (Date of earliest event reported)

GALECTIN THERAPEUTICS INC.
(Exact name of registrant as specified in its charter)

NEVADA	000-32877	04-3562325
(State or other jurisdiction of incorporation)	(Commission File Number)	(IRS Employer Identification No.)

7 WELLS AVENUE
NEWTON, MASSACHUSETTS
02459
(Address of principal executive offices) (Zip Code)

(617) 559-0033
(Registrant's telephone number, including area code)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions (*see* General Instruction A.2. below):

☐ Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)

☐ Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)

☐ Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))

☐ Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Item 7.01. Regulation FD Disclosure.

Corporate updated information is contained in the slide presentation attached as Exhibit 99.1 to this Current Report on Form 8-K (this "Report") on January 9, 2012.

The information in this Report is being furnished pursuant to this Item 7.01 and shall not be deemed "filed" for purposes of Section 18 of the Securities Exchange Act of 1934 (the "Exchange Act") or otherwise subject to the liabilities of that section, and it shall not be deemed incorporated by reference in any filing under the Securities Act of 1933 or under the Exchange Act, whether made before or after the date hereof, except as expressly set forth by specific reference in such filing to this Report.

Item 9.01. Financial Statements and Exhibits.

(d) Exhibits

99.1 Corporate update presentation slides - dated January 9, 2012.

SIGNATURES

Pursuant to the requirements of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

Galectin Therapeutics Inc.

By: /s/ Anthony D. Squeglia
Anthony D. Squeglia
Chief Financial Officer

Date: January 9, 2012

EXHIBIT INDEX

Exhibit No.:

99.1 Corporate update presentation slides, dated January 9, 2012.

Exhibit 99.1

Galectin Therapeutics

Company Overview

January 2012

OTC: GALT

Forward Looking Statements

This presentation contains, in addition to historical information, forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995. These statements relate to future events or future financial performance, and use words such as “may,” “estimate,” “could,” “expect” and others. They are based on our current expectations and are subject to factors and uncertainties which could cause actual results to differ materially from those described in the statements. Factors that could cause our actual performance to differ materially from those discussed in the forward-looking statements include, among others: incurrence of operating losses since our inception, uncertainty as to adequate financing of our operations, extensive and costly regulatory oversight that could restrict or prevent product commercialization, inability to achieve commercial product acceptance, inability to protect our intellectual property, dependence on strategic partnerships, product competition, and others stated in risk factors contained in our SEC filings. We cannot assure that we have identified all risks or that others may emerge which we do not anticipate. You should not place undue reliance on forward-looking statements. Although subsequent events may cause our views to change, we disclaim any obligation to update forward-looking statements.

Galectin Therapeutics Highlights

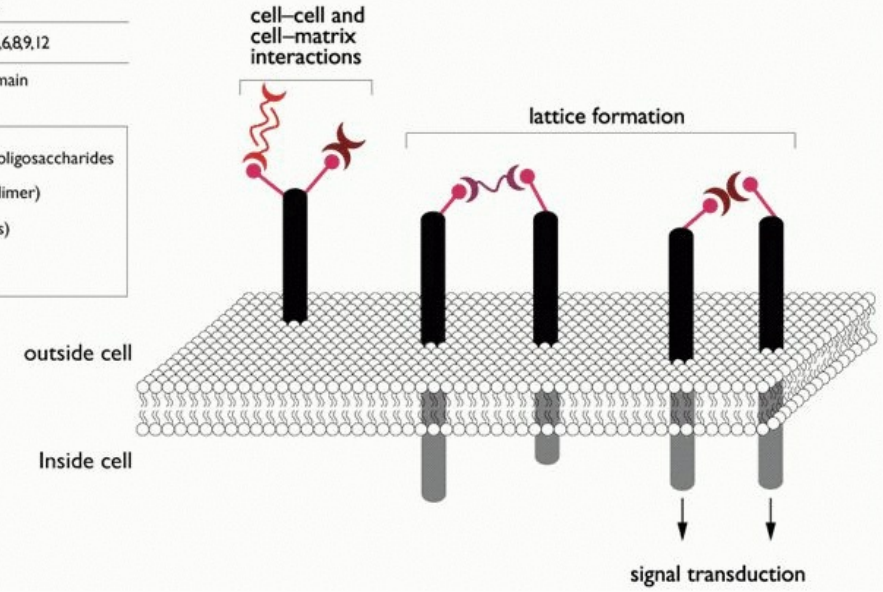
- Leader in galectin science
  - Pipeline of carbohydrate-based drug compounds that inhibit galectins
  - Focus on proven galectin activity in fibrosis and cancer
- Liver Fibrosis
  - Target validated in convincing pre-clinical data
  - Two indications: NASH and Post-transplantation fibrosis
  - Clinical trials expected to begin in end of 2012
- Cancer Therapy
  - Galectin inhibitor added to chemotherapy
  - Enhancement of immune function activates patient's own immune system to kill tumor cells
  - Clinical trial to begin January 2012

Galectin Proteins Are Normal Cell Proteins That Promote Interactions Between Glycoproteins

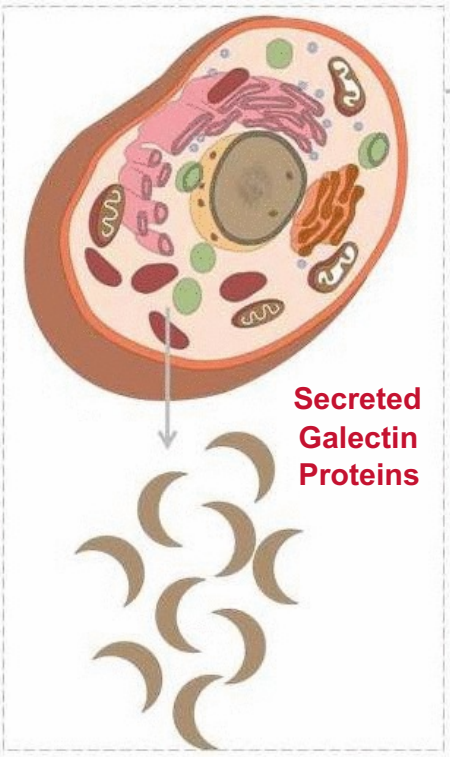
TYPE	STRUCTURE	GALECTIN
One-CRD		1,2,5,7,10,11,13,14,15
		3
Two-CRD		4,6,8,9,12

Carbohydrate **R**ecognition **D**omain

- Galactose-containing oligosaccharides
- One-CRD galectins (dimer)
- Galectins-3 (oligomers)
- Two-CRD galectins



Galectin Proteins Are Important In Disease Pathogenesis



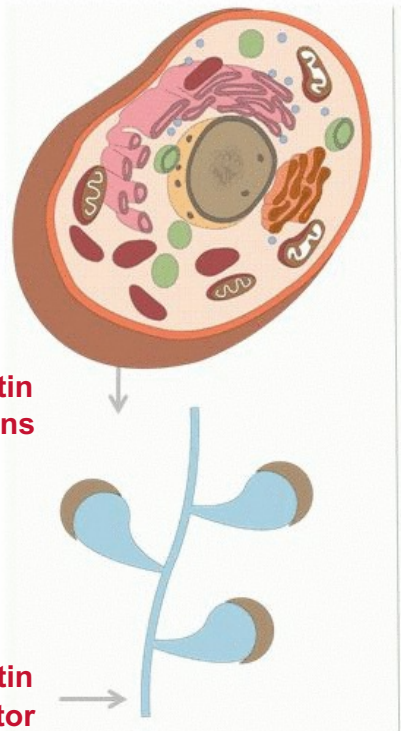
1. Bind to cell surface and matrix glycoproteins (galactose residues)
2. Modulate cell signaling
3. Promote cell-cell interactions
4. Promote cell-matrix interactions

Markedly Increased in:

1. Fibrosis
2. Cancer
3. Inflammation

PROMOTE PATHOLOGY

Our Galectin Inhibitors Are Novel Carbohydrate-Based Drug Compounds



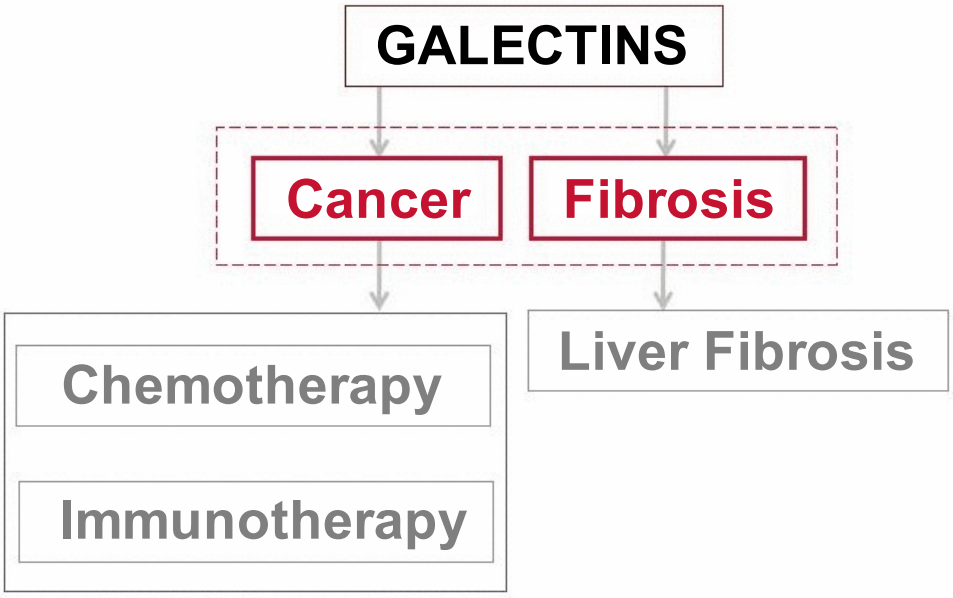
- Target secreted galectins and those associated with cell membrane
- Strong binding to multiple galectin proteins and multiple galectins per drug molecule
- High molecular weight allows long exposure to galectin containing compartment
- Low toxicity potential as a carbohydrate with no toxic metabolites
- Low manufacturing costs
- Strong patent protection with no licensing encumbrance
- Two major classes of compounds under development: GM-CT and GR-MD

Galectins Are Involved In The Pathogenesis Of Many Diseases

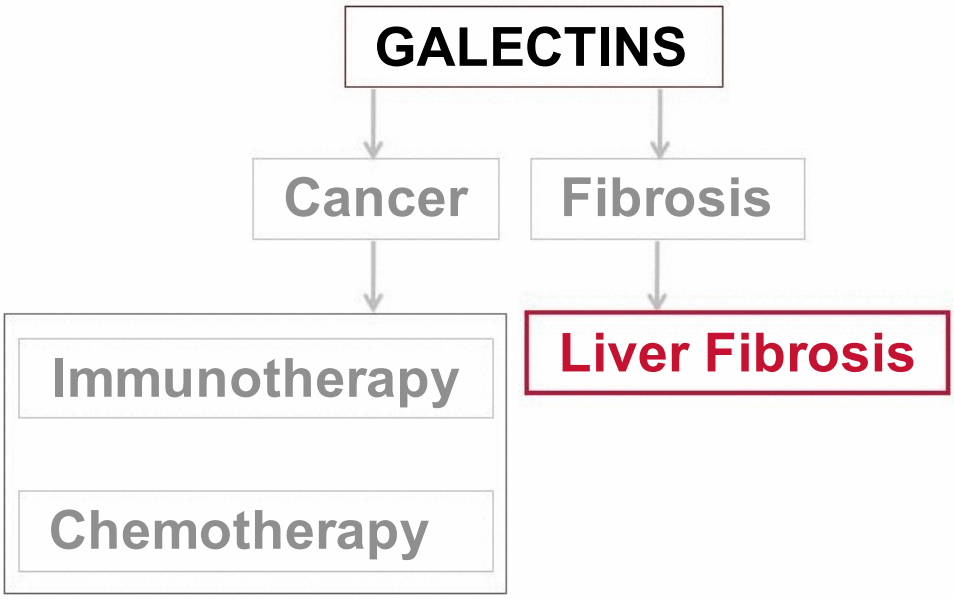
Galectins implicated in:

- **Fibrosis of organs**
- **Nearly all cancers**
- Heart failure
- Ischemic cardiovascular and cerebrovascular disease
- Arthritis
- Allergic disease
- Eczema and skin inflammation
- Inflammatory bowel disease
- Eye inflammation
- Inflammatory and autoimmune disorders
- Response to infections
- Kidney disease

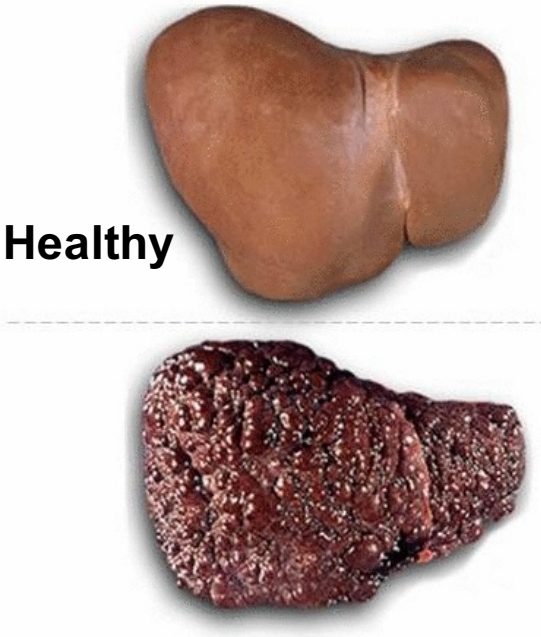
Disease Area Development Programs



Disease Area Development Programs



Multiple Diseases Lead To Liver Fibrosis And Cirrhosis With Serious Medical Consequences



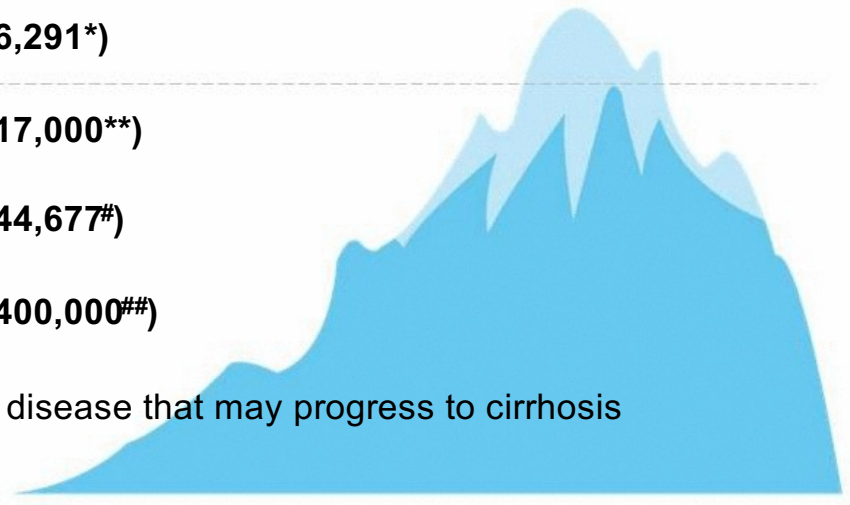
- Hepatitis C (57%)
- Alcoholic liver disease (24%)
- Non-alcoholic fatty liver (9.1%)
- Hepatitis B (4.4%)
- Miscellaneous (5.5%)

Source: Burden of liver disease in the United States: Summary of a workshop. American Association for the Study of Liver Disease, May 2001

Liver Cirrhosis Is A Major Problem In The United States

The ONLY current therapy is liver transplantation

Transplants	**(6,291*)**
Wait List	**(17,000**)**
Death	**(44,677#)**
Cirrhosis	**(400,000##)**



Millions of people with liver disease that may progress to cirrhosis

* Performed in US in 2010 (UNOS)

* * Prevalence in US 2010 (UNOS)

#Deaths in 1998 (AASLD Workshop, 2001)

##Prevalence in US 1976-1980 (NIDDK)

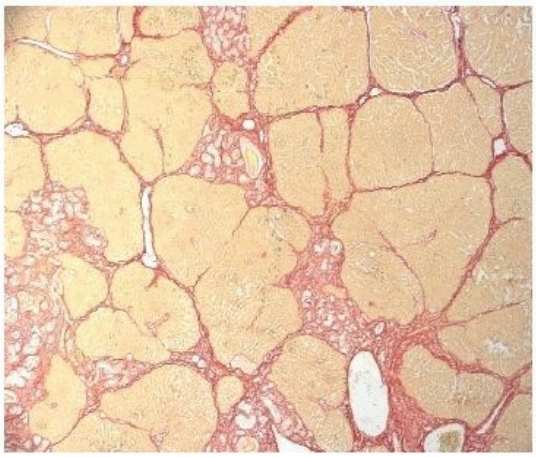
Galectin-3 Is A Critical Target For Therapy of Liver Fibrosis

Key Evidence:

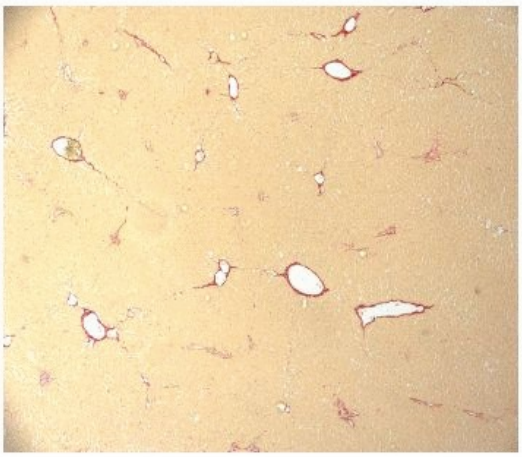
1. Galectin-3 is produced in large amounts by human fibrotic liver.
2. Galectin-3 is essential in mice for the development of liver fibrosis. Fibrosis due to toxin exposure or fatty liver does not occur in mice that lack the galectin-3 gene.
3. Galectin inhibitors block production of fibrogenic markers in the key human cell (stellate cells) responsible for liver fibrosis.
4. Galectin inhibitors reverse experimental fibrosis in rats induced by both fibrosis and fatty liver.

Galectin Inhibitors Effectively Treat Toxin-Induced Liver Fibrosis in Rats

Liver Fibrosis, induced by injection of chemical toxin for 8 weeks

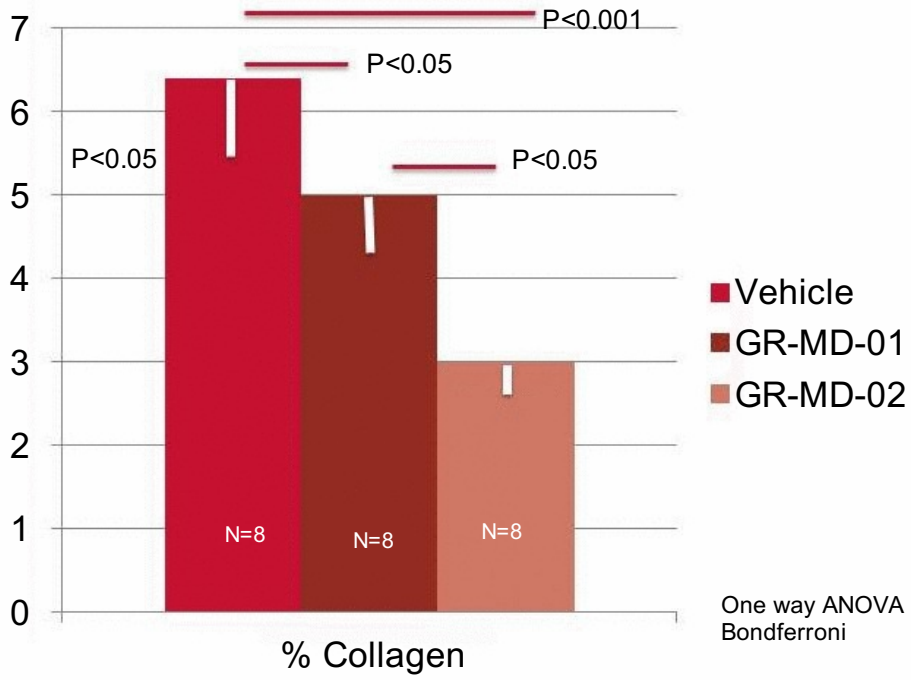


Regression of Fibrosis after 4 weeks of treatment with GR-MD-02

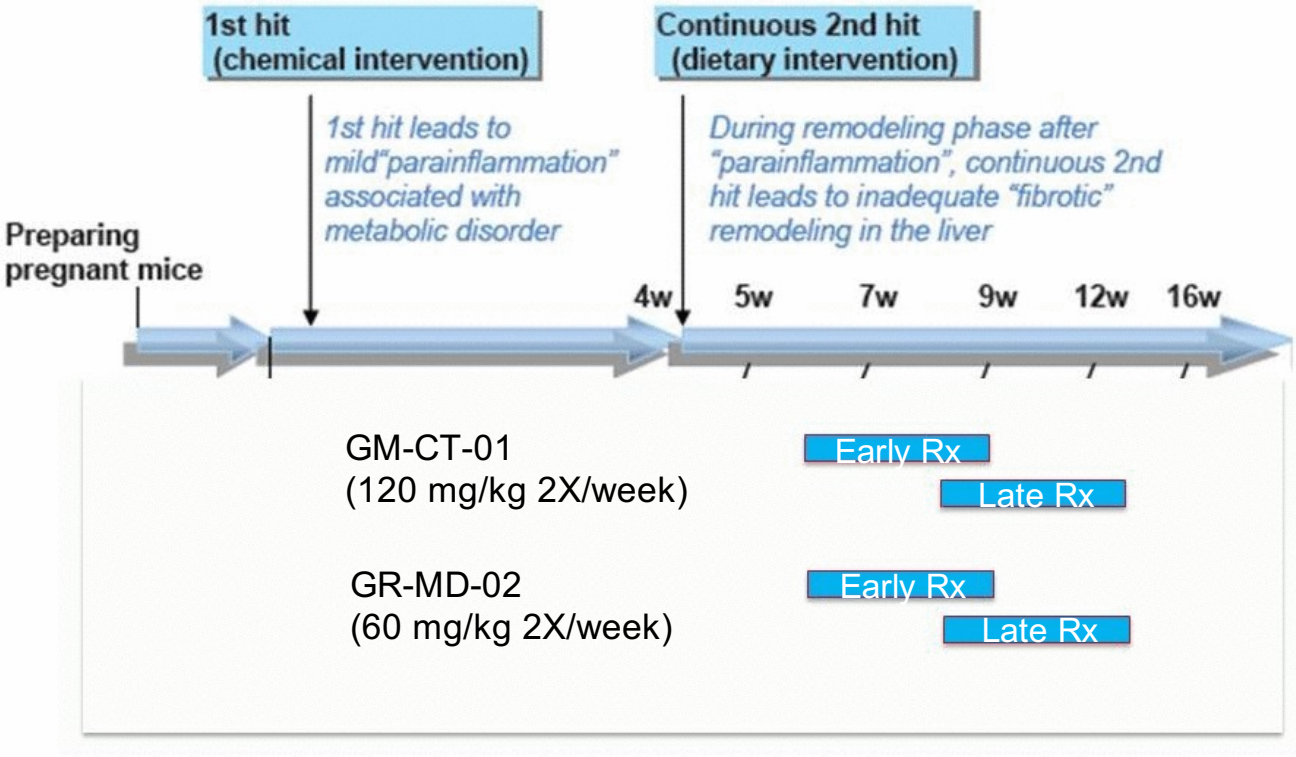


Study performed under contract by Dr. Ji-yao Wang of Fudan University, Shanghai, China

GR-MD-02 is Most Effective in Reducing Collagen Content in Fibrotic Rats



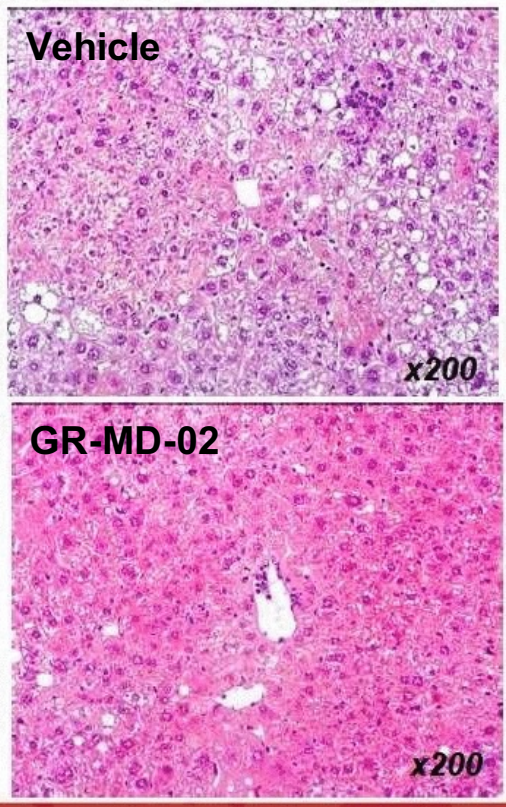
Mouse NASH Model (Non-Alcoholic Steatohepatitis, "Fatty Liver Disease")



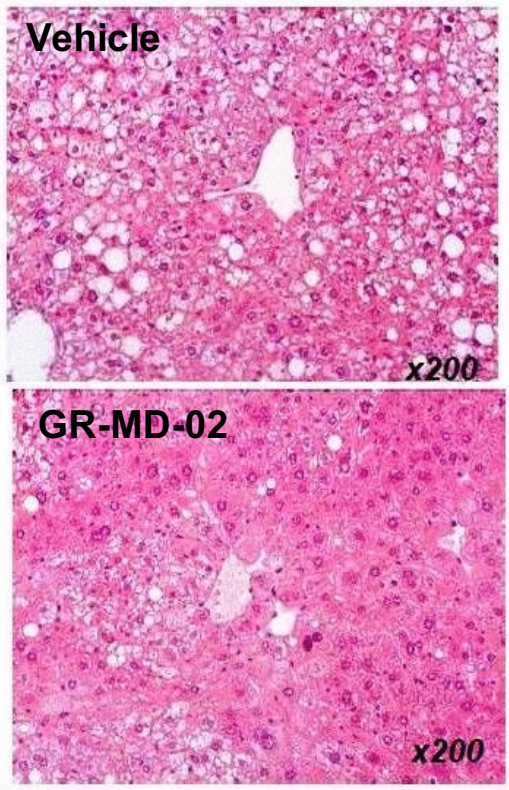
GR-MD-02 Markedly Improves NASH in a Mouse Model When it is Administered During Development of Disease and After Establishment of Disease

Galectin Therapeutics

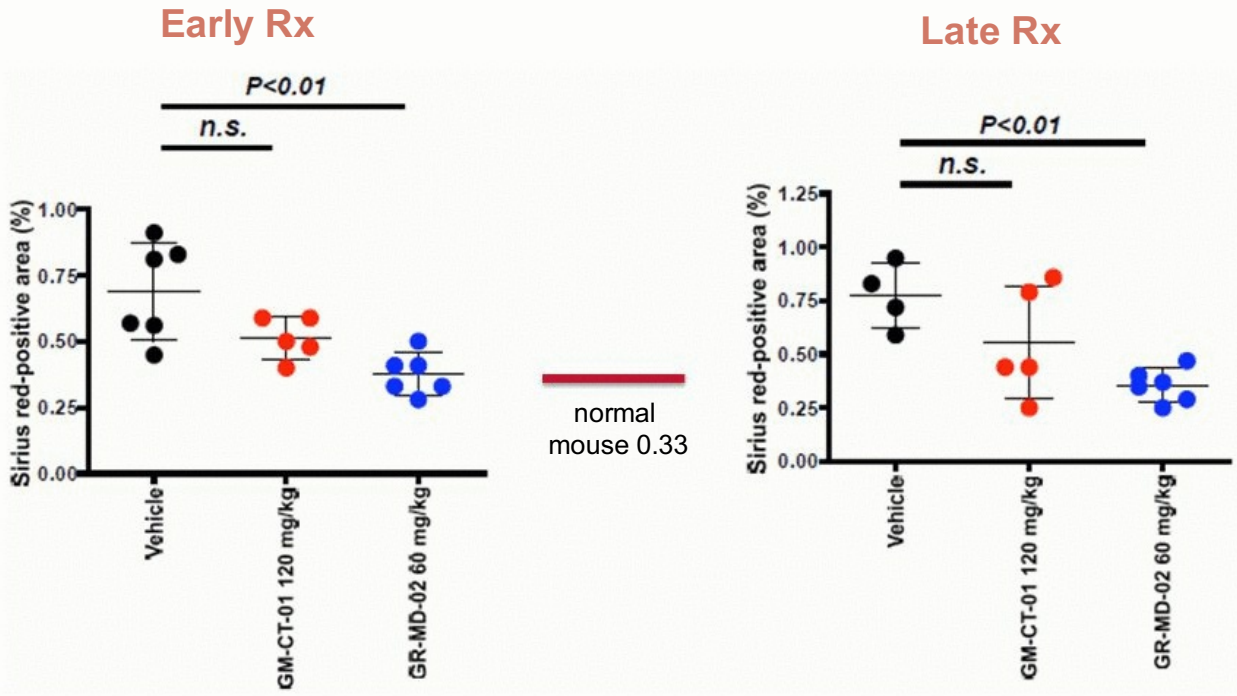
Early Rx



Late Rx



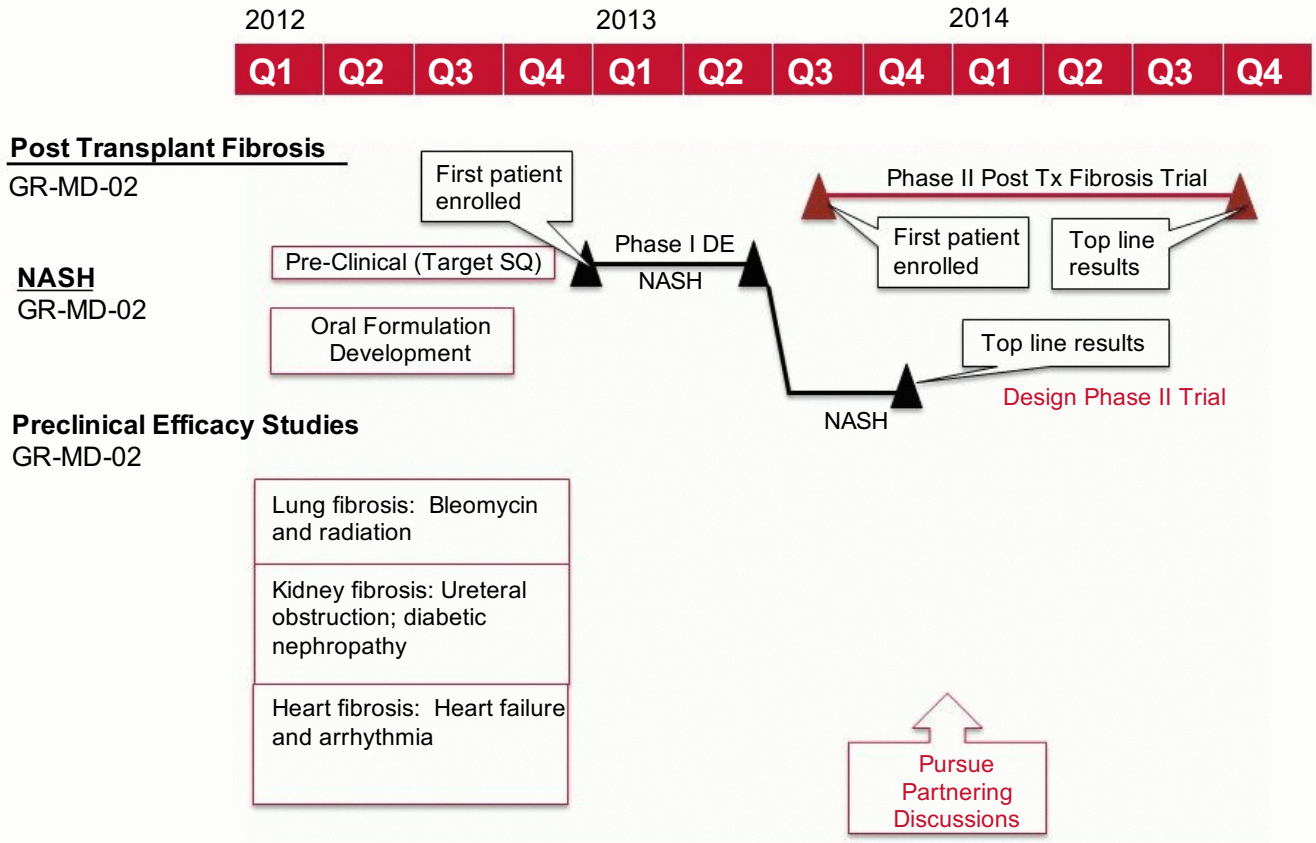
In a Mouse NASH Model GR-MD-02 Prevents and Completely Reverses Fibrosis



Development Program & Markets

- Pursue two indications for human proof of concept
  - NASH
  - Post-Transplant Hepatitis C Fibrosis
- NASH
  - NIH estimates 5% of US population has NASH
  - Projected to become the number one reason for liver transplant
  - Multiple candidates in development but none have multiple sites of action, effect on fibrosis, or safety profile
- Post-Transplant Hepatitis C Fibrosis
  - Much smaller population but high unmet medical need
  - Orphan disease status possible
- Expansion to other indications is possible
  - Other liver diseases such as hepatitis and alcoholic disease
  - Pulmonary, renal, and heart fibrosis

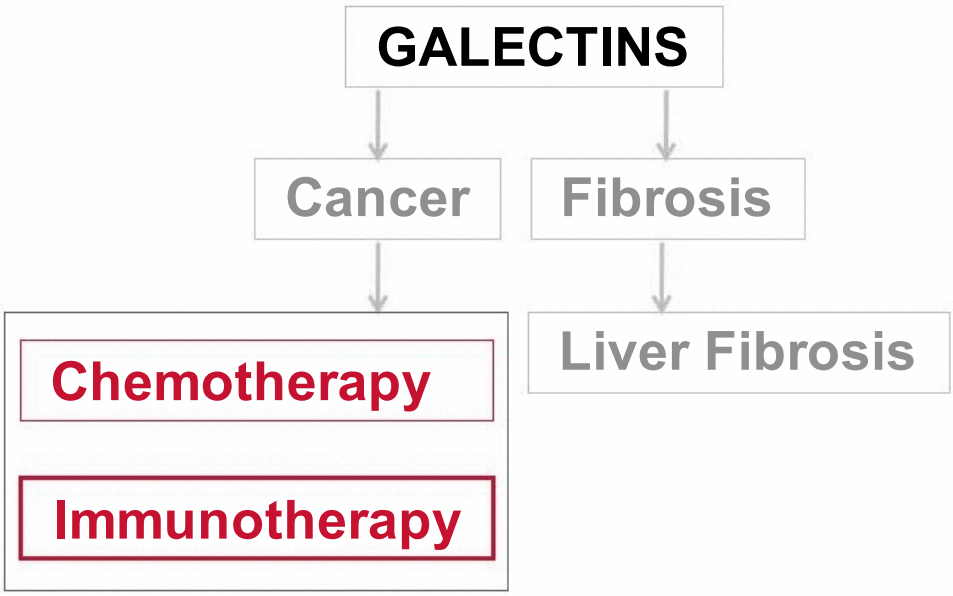
Fibrosis Development Program



Summary Of Development Program In Liver Fibrosis

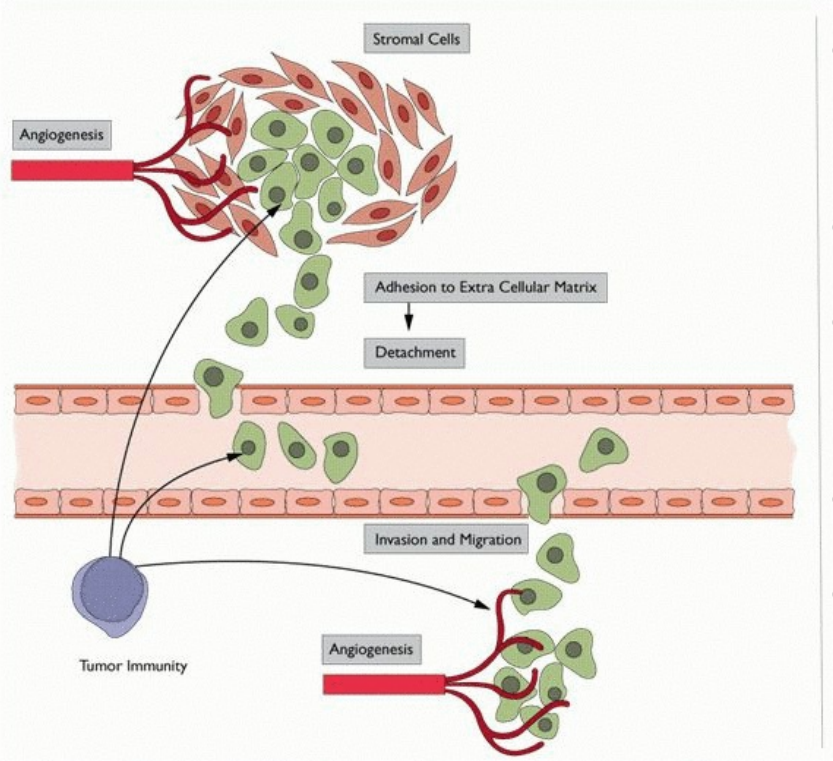
- Liver fibrosis represents a very large unmet medical need
- Galectin-3 protein is proven target
- Drugs reverse liver fibrosis in animals and show efficacy in human cell culture models of fibrosis
- Non toxic drugs with little likelihood of drug interactions
- Rapid clinical development pathways
- Evaluation of two clinical indications

Disease Area Development Programs



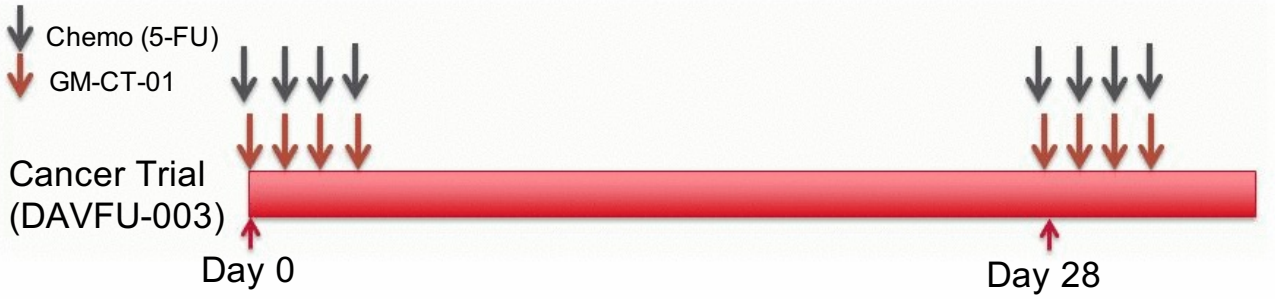
Roles Of Secreted Galectins In Cancer

The vast majority of cancers secrete large amounts of galectins



- **Tumor cell invasion:** extracellular matrix adhesion & detachment
- **Stromal cell function**
- **Metastasis:** cell invasion and migration
- **Angiogenesis**
- **Tumor immunity**

Clinical Trial Performed In Metastatic Colorectal Cancer (DAVFU-003)

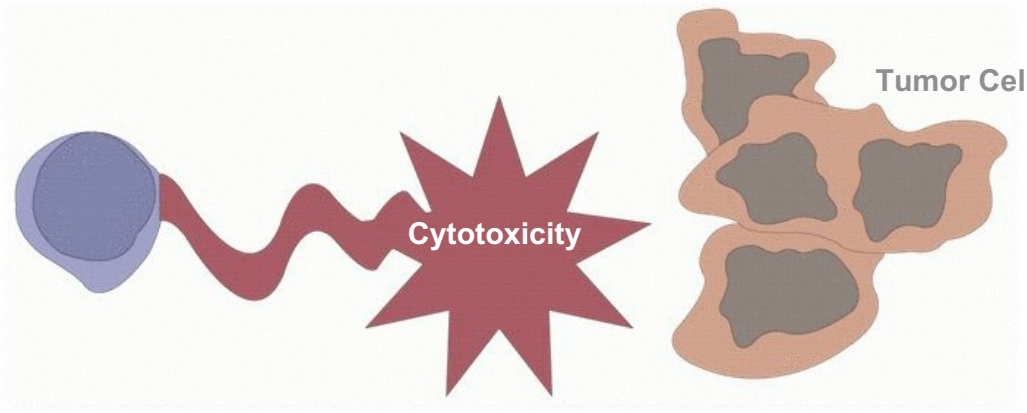


- Phase 2 trial of 5-FU plus GM-CT-01 in line 3/4 therapy of metastatic colorectal cancer showed 6.7 months median survival. In similar patients, Erbitux® had a 6.1 month survival compared to 4.6 months with no therapy
- Notably, serious adverse events were markedly lower in our studies with 5-FU/GM-CT-01 than in comparison to other studies using 5-FU

Development Approach In Colorectal Cancer

- Studies demonstrate potential utility of galectin inhibitors in combination with chemotherapy in cancer
- FDA has confirmed that preclinical and clinical data are adequate to proceed with large clinical trials
- We are deferring new clinical trials pending data from the tumor immunology clinical trial that may improve the design of future studies
- More rapid international registration is an approach that may provide revenue to support development programs and gain additional clinical experience with GM-CT-01

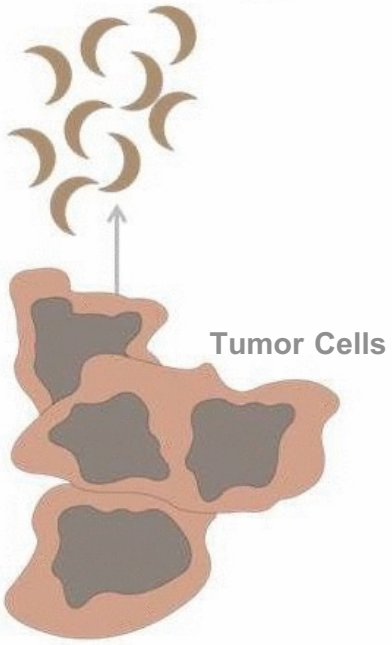
Blocking Galectins Enhances Tumor Killing By Immune System



There are tumor specific cytotoxic T-cells in patients with cancer that are capable of killing tumor cells

Experiments performed by Dr. Pierre van der Bruggen of the Ludwig Institute in Brussels, Belgium as a collaboration with Galectin Therapeutics

Blocking Galectins Enhances Tumor Killing By Immune System

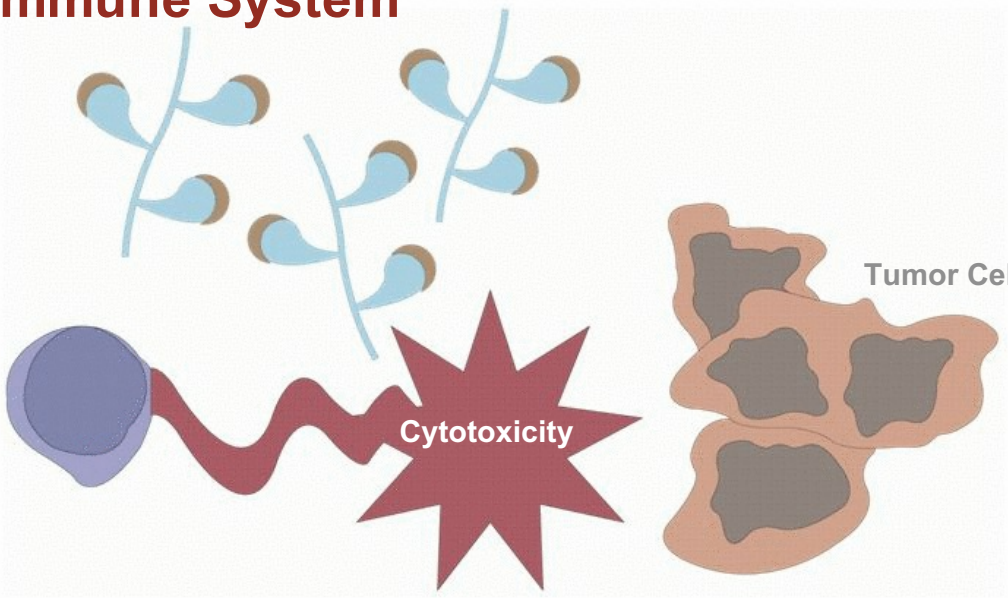


Tumor-Specific Cytotoxic T-Cell Lymphocytes

However, these T-cells lose the ability to kill tumor cells. A key reason is that Galectin-3 secreted by tumors bind to T-cells and turn off their ability to kill tumor cells

Experiments performed by Dr. Pierre van der Bruggen of the Ludwig Institute in Brussels, Belgium as a collaboration with Galectin Therapeutics

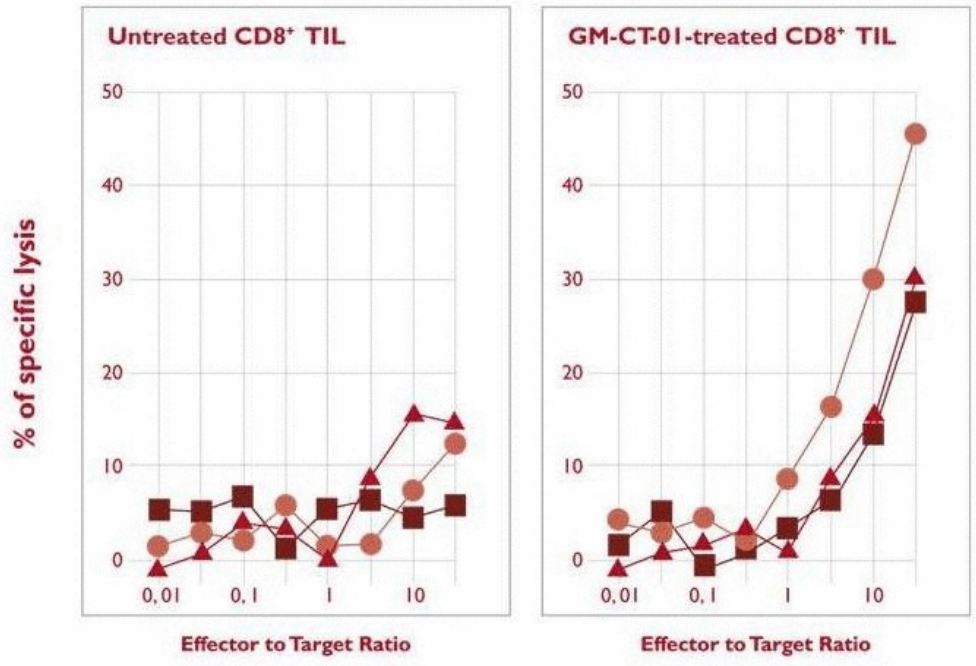
Blocking Galectins Enhances Tumor Killing By Immune System



Addition of our drugs, which inhibit galectins, restore the ability of T-cells to kill tumor cells

Experiments performed by Dr. Pierre van der Bruggen of the Ludwig Institute in Brussels, Belgium as a collaboration with Galectin Therapeutics

GM-CT-01 Restores Ability of Immune Cells to Kill Tumor Cells



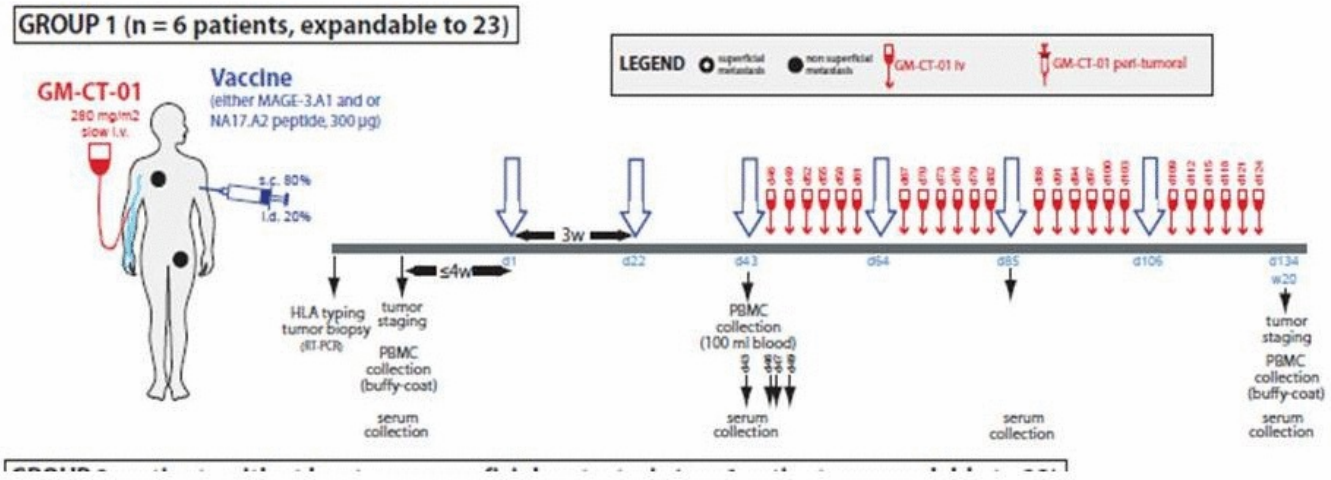
Effectors:
CD8+ T cells cultured in medium treated for 20h
Targets:
P815 cells loaded with anti-CD3+ cold K562

Effectors:
CD8+ T cells cultured in medium with GM-CT-01 for 20h
Targets:
P815 cells loaded with anti-CD3+ cold K562

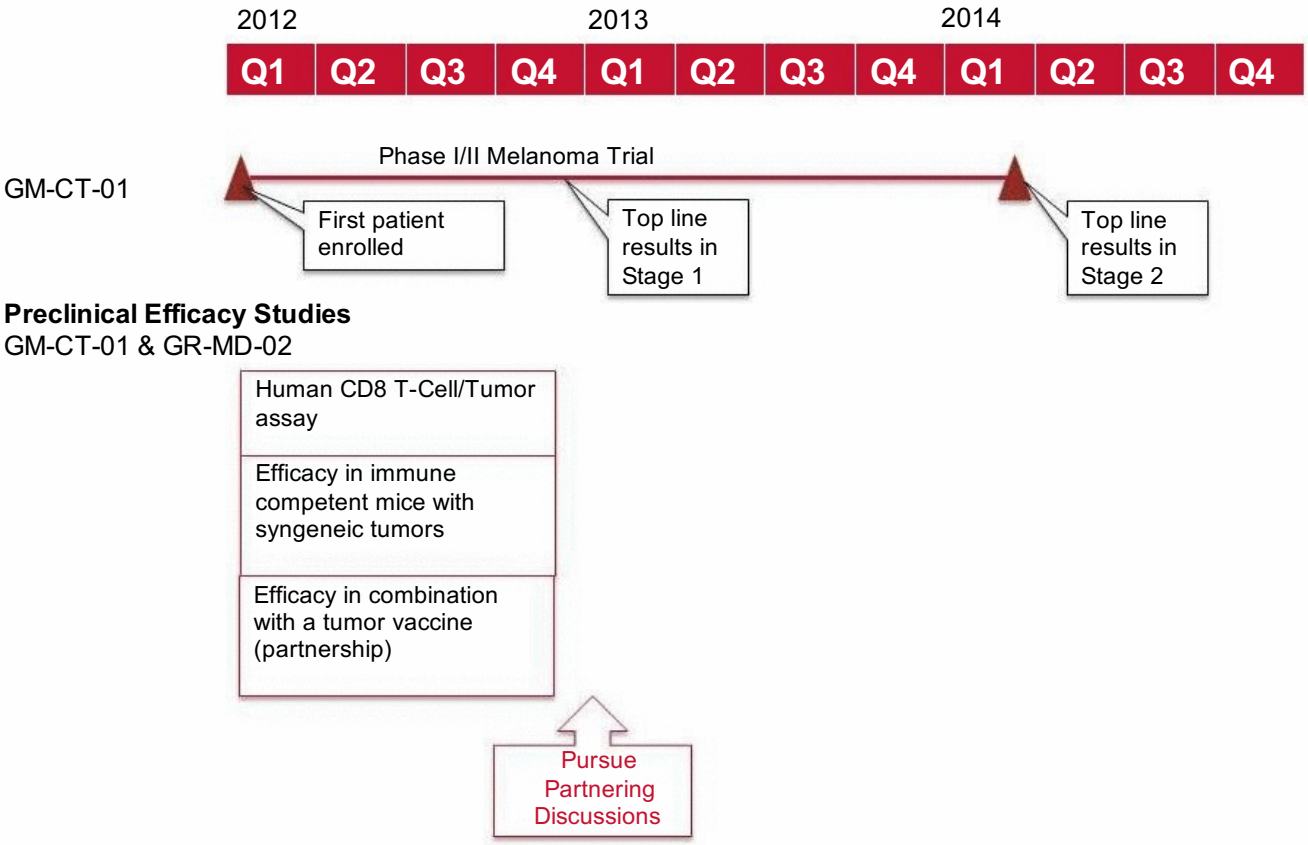
Melanoma Clinical Trial Design (I)

Phase I/II study of peptide vaccination associated with GM-CT-01, a galactomannan oligomer that inhibits galectin-3, in patients with advanced metastatic melanoma

IMPD approved by EMA



Tumor Immune Enhancement Development Program



Development Program In Cancer Immunotherapy

- Galectin proteins secreted by tumor cells are directly responsible for inhibiting the ability of immune cells to kill tumors
- GM-CT-01 restores the ability of immune cells to kill tumor cells
- Initial clinical trial for treatment of metastatic malignant melanoma
- Market for tumor vaccines is expected to grow to $7B by 2015
- Potential important therapy for many cancers

Pipeline

	Pre-Clinical	Phase 1	Phase 2	Phase 3	Registration Submitted
Chemotherapy					
Colorectal Cancer: GM-CT-01					
• International (Colombia)					
• United States					
Immune Enhancer					
Melanoma: GM-CT-01					
Liver Fibrosis					
Post Tx: GR-MD-02					
NASH: GR-MD-02					

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