

**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**

Date of Report (Date of earliest event reported): May 11, 2015

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

Nevada
(State or Other Jurisdiction
of Incorporation)

001-31791
(Commission
File Number)

04-3562325
(IRS Employer
Identification No.)

**4960 PEACHTREE INDUSTRIAL BOULEVARD, Ste 240
NORCROSS, GA 30071**
(Address of principal executive office) (zip code)

Registrant's telephone number, including area code: (678) 620-3186

N/A
(Former name or former address, if changed since last report)

- 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))

SECTION 7 – REGULATION FD

Item 7.01 Regulation FD Disclosure.

On May 11, 2015, Galectin Therapeutics Inc. posted a corporate presentation on its website that contains, among other information, a summary of development of GR-MD-02 for Non-Alcoholic Steatohepatitis (NASH) With Advanced Fibrosis and Cirrhosis, which presentation is attached as Exhibit 99.1.

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.

SECTION 9 – FINANCIAL STATEMENTS AND EXHIBITS

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits.

<u>Exhibit Number</u>	<u>Description</u>
99.1	Corporate presentation

SIGNATURES

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

Galectin Therapeutics Inc.

Date: May 11, 2015

By: /s/ Jack W. Callicutt
Jack W. Callicutt
Chief Financial Officer

- 3 -



Corporate Presentation

May 11, 2015

NASDAQ: GALT
www.galectintherapeutics.com

© 2015 Galectin Therapeutics Inc.

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. These statements include those regarding potential therapeutic benefits of our drugs, expectations, plans and timelines related to our clinical trials, potential partnering opportunities and estimated spending for 2015. Factors that could cause our actual performance to differ materially from those discussed in the forward-looking statements include, among others, our trials may not lead to positive outcomes or regulatory approval. We may experience delays in our trials, which could include enrollment delays. Future phases or future clinical studies may not begin or produce positive results in a timely fashion, if at all, and could prove time consuming and costly. Plans regarding development, approval and marketing of any of our drugs are subject to change at any time based on the changing needs of our company as determined by management and regulatory agencies. Strategies and spending projections may change. We may be unsuccessful in developing partnerships with other companies or obtaining capital that would allow us to further develop and/or fund any studies or trials. We are currently the subject of litigation, which may impact our human and capital resources. To date, we have incurred operating losses since our inception, and our future success may be impacted by our ability to manage costs and finance our continuing operations. For a discussion of additional factors impacting our business, see our Annual Report on Form 10-K for the year ended December 31, 2014, and our subsequent filings with the SEC. 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.

Organ Fibrosis

- 45% of U.S. deaths estimated to be associated with fibrotic disease¹
- Lead indication is liver fibrosis/cirrhosis due to fatty liver disease (75% of all liver disease in U.S.)²
- Potentially applicable to other fibrotic diseases based on pre-clinical studies
- Phase 1 clinical trial completed
- Phase 2 trials in two NASH indications to start Q2 2015

Cancer Immunotherapy

- Focus on combination immunotherapy, one of the most prominent approaches to cancer therapy
- Lead indication is advanced melanoma, but technology applicable to other cancers
- Phase 1b trial in combination with Yervoy³ in progress
- Phase 1b trial in combination with KEYTRUDA⁴ expected to start Q3 2015

¹Wynn, TA. Nat Rev Immunol. 2004;4:583-594. doi:10.1038/nri1412

²Younossi, et al. Clin. Gastro. Hepatol. 2011;9:524-530

³Registered trademark, Bristol Myers Squibb

⁴Registered trademark, Merck Sharp Dome

Experienced Leadership Team

James Czirr, Executive Chairman	<ul style="list-style-type: none"> • Manager and general partner of 10X Fund, L.P., Co-Founder, Pro-Pharmaceuticals, CEO, Minerva Biotechnologies Corporation
Peter G. Traber, M.D. President, CEO, CMO	<ul style="list-style-type: none"> • Over 30 years experience in biomedicine and pharmaceutical industries in research and development, clinical medicine and business development. • GlaxoSmithKline (CMO), Un of Pennsylvania (CEO, Chief of GI, Chairman of Medicine), Baylor College of Medicine (CEO)
Harold H. Shlevin, Ph.D. COO & Corporate Secretary	<ul style="list-style-type: none"> • Over 32 years of senior experience in the development and commercialization of pharmaceuticals and business development including mergers and acquisitions. • Solvay Pharmaceuticals (CEO), CIBA Vision Ophthalmics (n/k/a Novartis Vision) (SVP & co-founder), Tilkah Therapeutics (Founder, CEO)
Jack W. Callicutt CFO	<ul style="list-style-type: none"> • Over 24 years in accounting and finance with life science and technology companies with significant experience in negotiating and closing financing transactions. • CFO Reach Health, CFO of Vystar Corporation, CFO Corautus Genetics, Deloitte
Eliezer Zomer, Ph.D. Pharmaceutical Development	<ul style="list-style-type: none"> • Over 30 years experience in biotechnology engineering and regulatory in pharmaceuticals and diagnostics. • Koor Biotechnologies, Charm Sciences, Glycogenesis , HU Medical School (Jerusalem), Harvard University
J. Rex Horton Executive Director, Regulatory Affairs and Quality Assurance	<ul style="list-style-type: none"> • Over 24 years of experience working in the biotech and life sciences industries, regulatory affairs and manufacturing. • Director Regulatory Affairs at Solvay Pharmaceuticals and Chelsea Therapeutics, Georgia Institute of Technology.

Galectin-3 Protein Function

- Binds to galactose residues in glycoproteins and promotes interactions between these proteins
- High expression in immune cells (macrophages)
- Modulates cell signaling and immune cell function

Role in Disease

- Gal-3 is increased in inflammation and fibrogenesis
- Genetic modification in mice that eliminates gal-3 prevents fibrosis in liver, lung, kidney and heart
- The majority of cancers express high levels of gal-3, which promotes tumor and inhibits immune response

Lead Drug Candidate GR-MD-02

- A complex carbohydrate with terminal galactose residues that binds to gal-3 and disrupts function, particularly affecting immune/repair function in macrophages
- Efficacy in preclinical models of fibrotic disease and cancer immunotherapy with encouraging early human results
- Existing patent coverage through 2031 with 2 composition and 4 method patents issued

Lead Indication in Organ Fibrosis

ADVANCED FIBROSIS AND CIRRHOSIS DUE TO NASH (NON-ALCOHOLIC STEATOHEPATITIS)

Estimated prevalence of NASH in U.S. adults^{1, 2} > 28 million

**U.S. Prevalence in
Asymptomatic Adults
(% of population studied)²**

Obesity

45%

Diabetes

17%

Fatty Liver

46%

NASH

12%

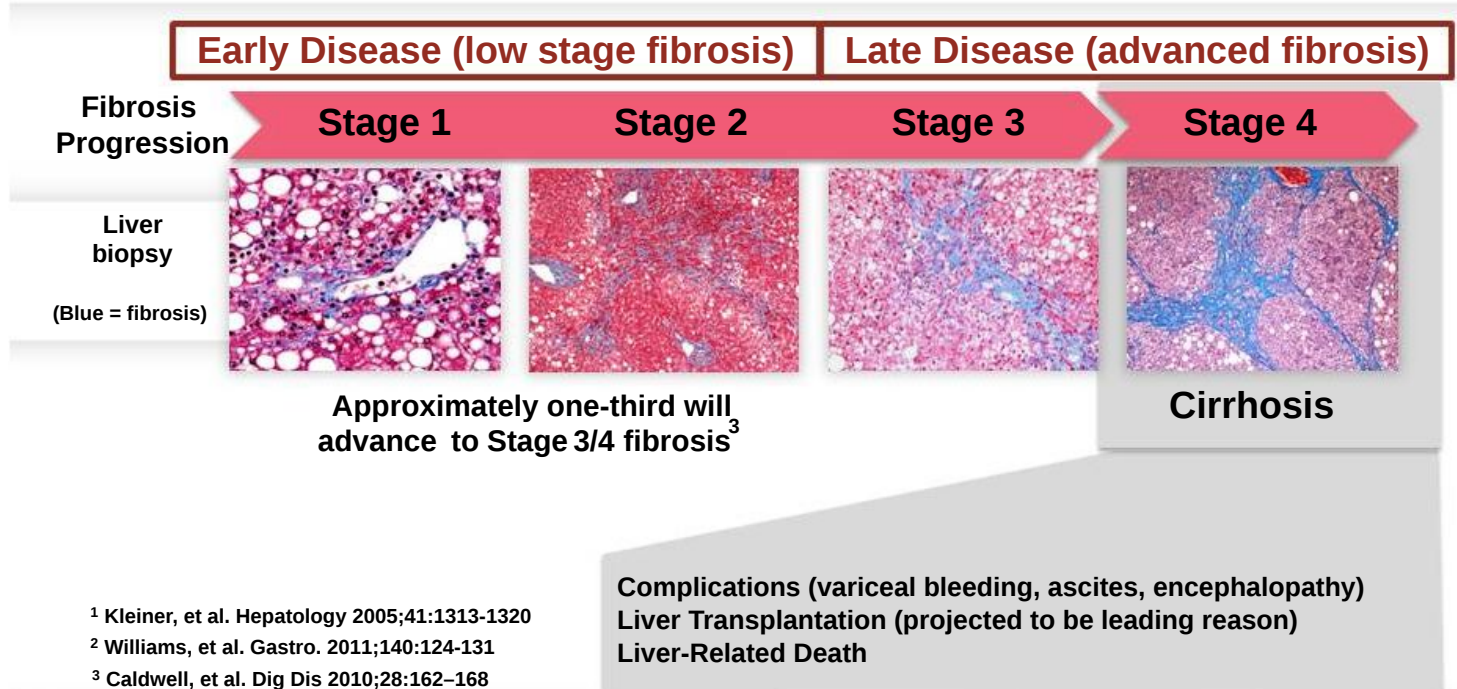
¹ Based on July 2013 US census data for people >20 years old

² Prospective evaluation of NAFLD and NASH prevalence (Williams, et al. Gastro. 2011;140:124-131)

Note: population recruited for this study were between ages 18 and 70

End-Stage Fibrosis (Cirrhosis) Is When Patients With NASH Experience Symptoms And Complications

Estimated US prevalence of advanced fibrosis^{1,2}: ~ 6 million
Estimated US prevalence of cirrhosis¹: ~ 1-2 million



¹ Kleiner, et al. Hepatology 2005;41:1313-1320

² Williams, et al. Gastro. 2011;140:124-131

³ Caldwell, et al. Dig Dis 2010;28:162-168

* Multiple other companies in discovery and Phase 1

Early Disease (low stage fibrosis)

Late Disease (advanced fibrosis)

Stage 1

Stage 2

Stage 3

Stage 4

- Obeticholic Acid (Intercept)
- GFT505 (GenFit)
- Liraglutide (Novo-Nordisk)
- Aramchol (Galmed)
- Cysteamine (Raptor)
- Cenicriviroc (Tobira)
- Emricasan (Conatus)

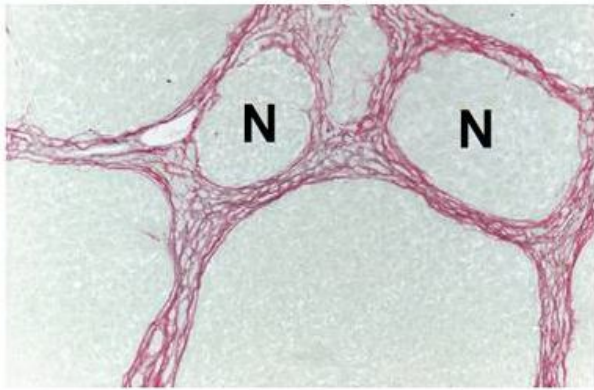
- Simtuzumab (Gilead)
- Emricasan (Conatus)
- GR-MD-02 (Galectin)

- Regulatory endpoints for late stage disease better defined and closer to clinical outcomes including complications of cirrhosis, liver transplant, and death
- Ultimately, combination therapies may be employed with agents that are tailored to different disease stages

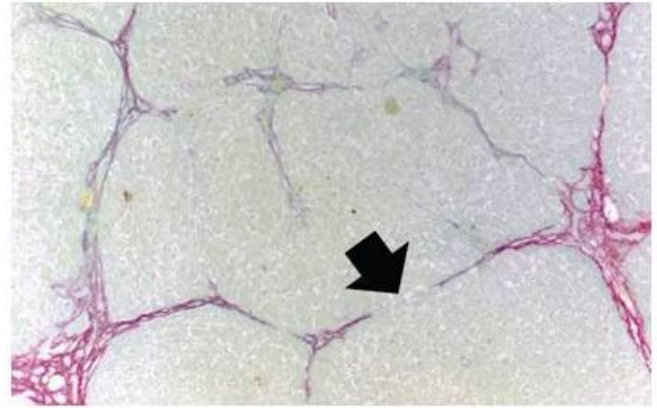
- **Mouse model of NASH**
 - Reduces inflammation, fat, and cell death
 - Prevents as well as reverses fibrosis
 - Targets macrophages and reduces galectin-3
 - Peer reviewed publication:
 - Traber PG and Zomer E. Therapy of Experimental NASH and Fibrosis with Galectin Inhibitors. PLOS ONE 2013;8:e83481
- **Rat model of liver fibrosis/cirrhosis**
 - Reduces inflammation and cell death
 - Reverses fibrosis and cirrhosis
 - Reduces portal hypertension associated with cirrhosis
 - Targets macrophages and reduces galectin-3
 - Peer reviewed publication:
 - Traber PG, Chou H, Zomer E, Hong F, Klyosov A Fiel M-I, Friedman, SL. Therapy of Regression of fibrosis and reversal of cirrhosis in rats by galectin inhibitors in thioacetamide-induced liver disease. PLOS ONE 2013;8:e75361.

Toxin induced cirrhosis in rats; toxin continued during GR-MD-02 treatment

Vehicle-Treated



**GR-MD-02-Treated
(4 weekly doses)**



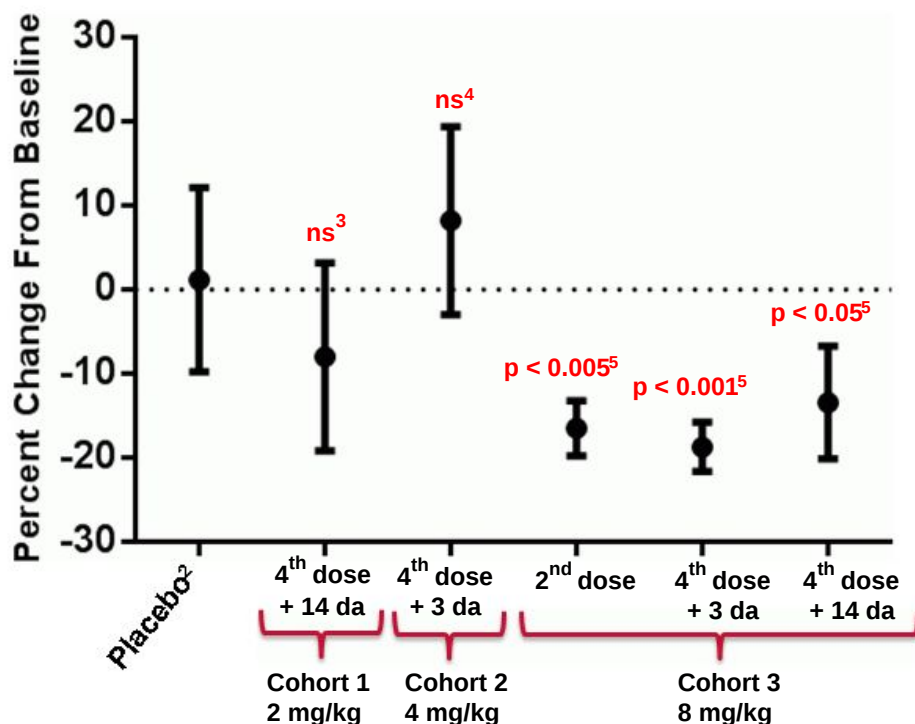
Reduces portal pressure which correlates with primary endpoint in planned cirrhosis trial

*Traber PG, Chou H, Zomer E, Hong F, Klyosov A Fiel M-I, Friedman, SL. Therapy of Regression of fibrosis and reversal of cirrhosis in rats by galectin inhibitors in thioacetamide-induced liver disease. PLOS ONE 2013;8:e75361.

- Multiple dose escalation, double-blind, placebo controlled trial in NASH patients with advanced fibrosis
- GR-MD-02 up to 8 mg/kg IV was safe and well tolerated
- 8 mg/kg IV is in the upper range of the targeted therapeutic window for drug administration
- The combined results of a reduction in serum alpha-2 macroglobulin and a reduction in liver stiffness as assessed by FibroScan® suggests that GR-MD-02 at the highest dose tested may have an effect on liver fibrosis
- Results provide strong foundation for entering Phase 2

Highly Significant Reduction In Alpha-2 Macroglobulin Serum Levels Seen At The 8 mg/kg Dose

A reduction in serum alpha-2-macroglobulin accounted for the reduction in FibroTest®

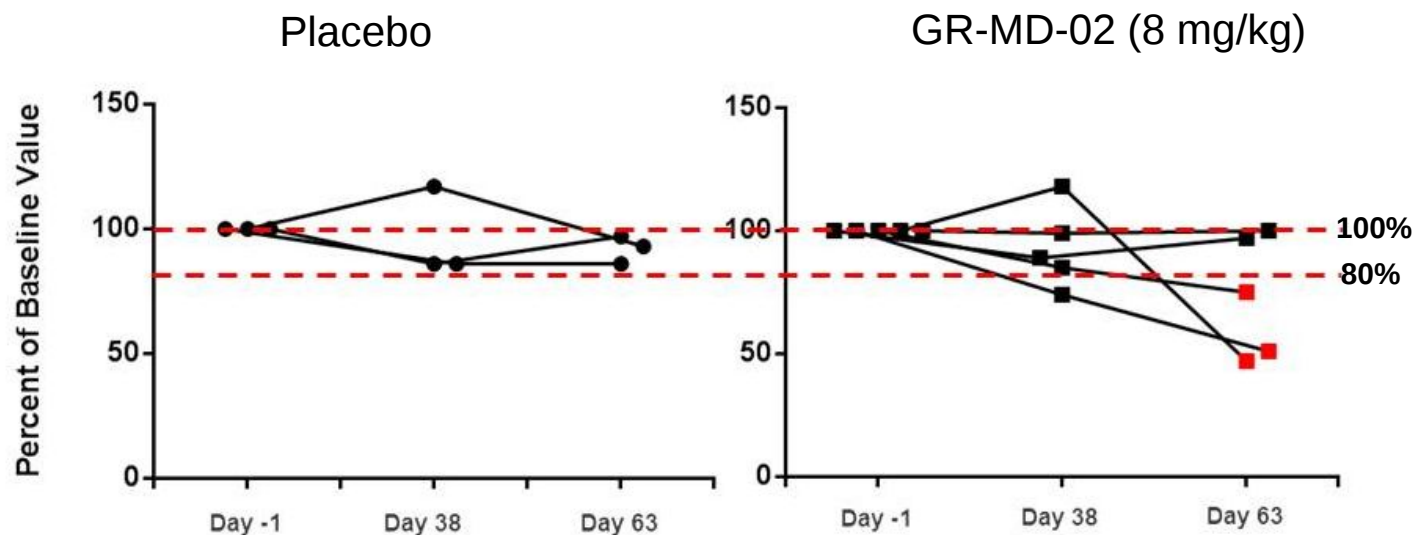


Legend and Notes:

1. Mean \pm standard deviation
2. Placebo values were combined for all three cohorts because there were no differences (n=19 separate data points)
3. Not significant versus placebo, two-sided t-test (n=6)
4. Not significant versus placebo, two-sided t-test (n=7)
5. Significant for three groups versus placebo, ANOVA with Dunnett's test for multiple comparisons (n=7)

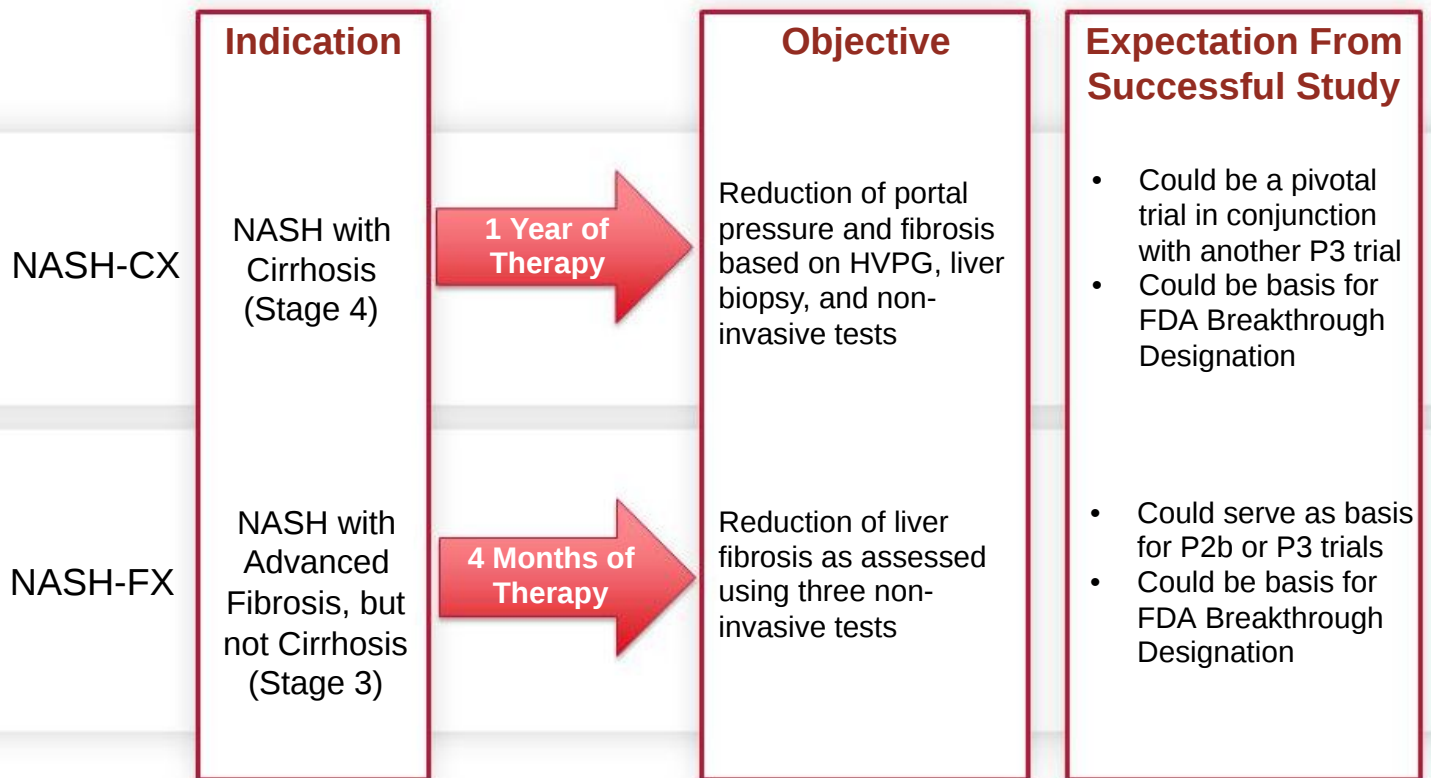
Evidence Of Reduced FibroScan® Scores In Cohort 3 Patients Treated With GR-MD-02

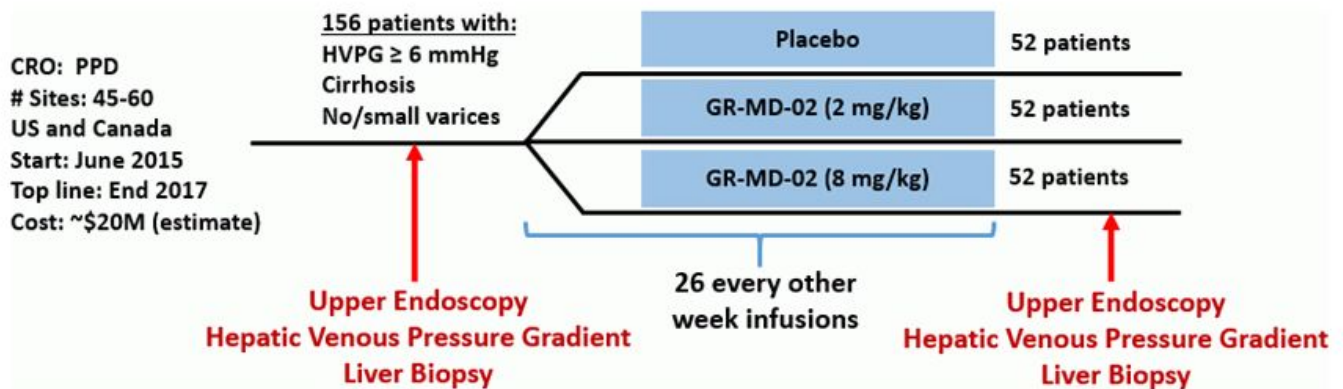
3 of 5 patients treated with GR-MD-02 had reduction in liver stiffness to below 80% of baseline values (red squares)*



*In cohort 3 there were technically adequate scans at baseline, Day 38 and Day 63 in 5 patients administered GR-MD-02 and 3 patients administered placebo. Five patients in cohort 3 were not available for FibroScan® analysis (3 placebo and 2 active) because of unavailability of the instrument at the site (1 placebo and 1 active), unavailability of the appropriate instrument probe (1 active), a technically inadequate baseline scan (1 placebo), and the Day 63 scan not being performed (1 placebo).

Phase 2 NASH Clinical Development Program Includes Trials In Two Separate Indications In NASH With Advanced Fibrosis

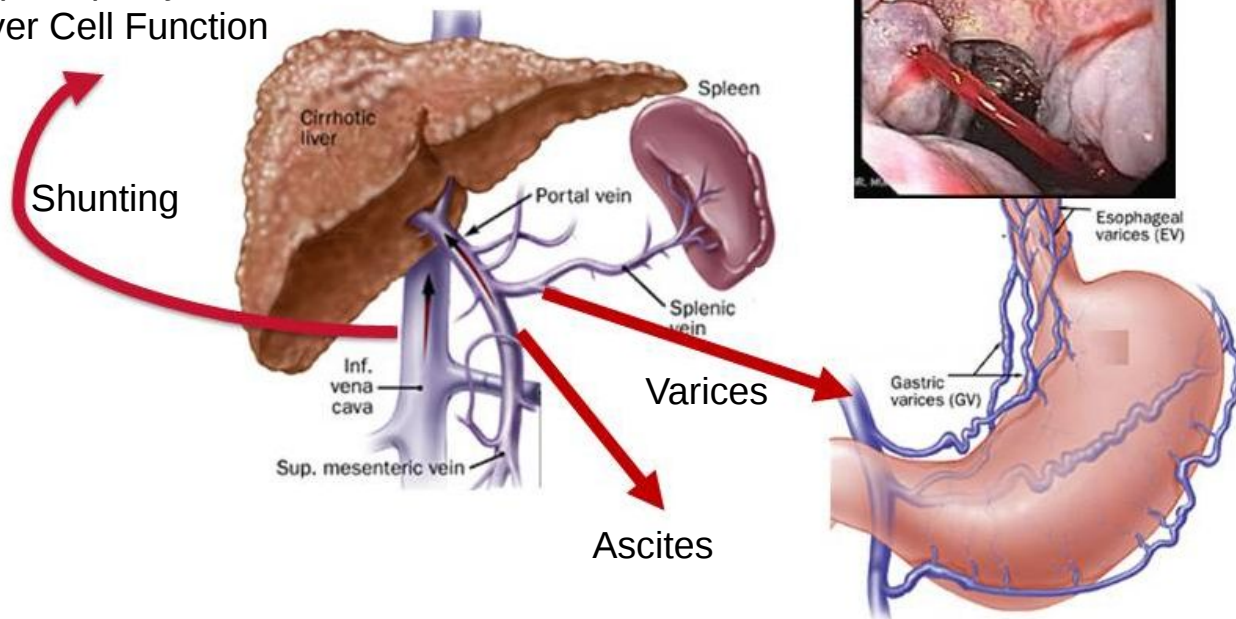




- **Primary Endpoint:**
 - Reduction of hepatic venous pressure gradient (HVPG) as a measure of portal pressure compared to placebo at 1 year of treatment
- **Secondary Endpoints:**
 - At least one stage change in histopathological fibrosis stage
 - Liver collagen on liver biopsy (digital morphometric analysis)
 - Liver stiffness as determined by FibroScan® Score
 - Metabolic capacity of the liver as determined by ^{13}C methacetin breath test
 - Progression of cirrhosis as determined by complications

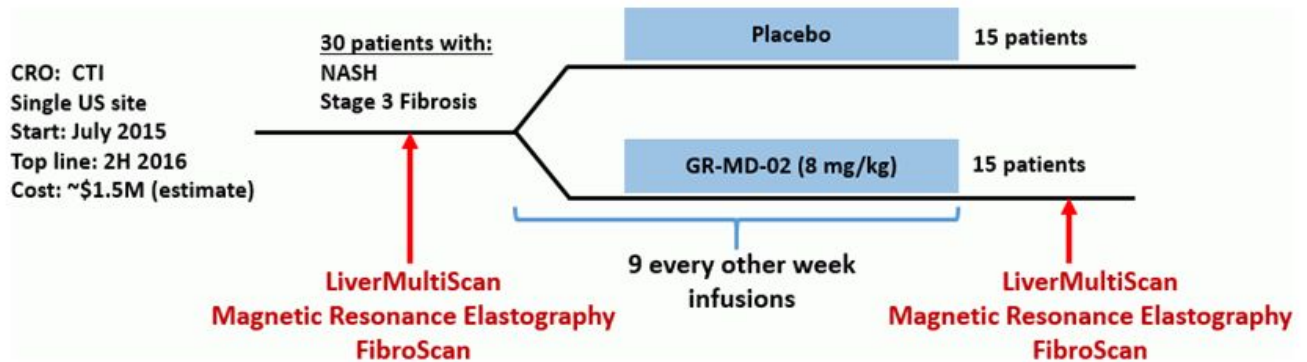
Complications of Portal Hypertension in Cirrhosis (Portal Hypertension = High Blood Pressure in Portal Vein)

Encephalopathy
↓ Liver Cell Function



The Goal is to:

↓ **Fibrosis** → ↓ **Portal Pressure** → ↓ **Complications** → ↓ **Morbidity and Mortality**



Primary Endpoint:

- Difference between placebo and GR-MD-02 in the baseline adjusted mean change in liver fibrosis as measured by corrected T1 (cT1) mapping as determined from LiverMultiScan (LMS), a multi-parametric MRI protocol.

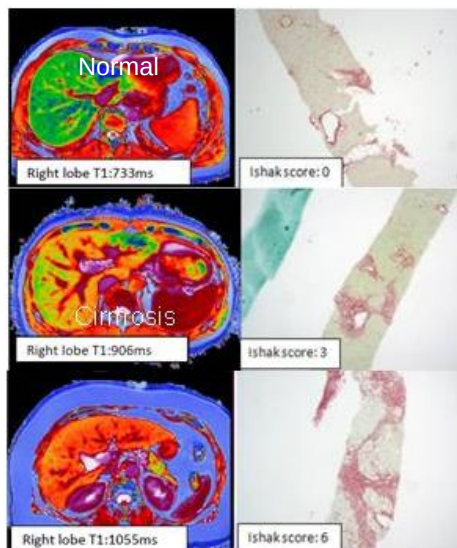
Secondary Endpoints:

- To determine the baseline-adjusted change in magnetic resonance elastography (as measured in kPa)
- To determine the baseline-adjusted change in FibroScan® Score (as measured in kPa)

<https://clinicaltrials.gov/ct2/show/NCT02421094?term=GR-MD-02&rank=3>

Non-Invasive Assessments of Liver Fibrosis in NASH-FX trial

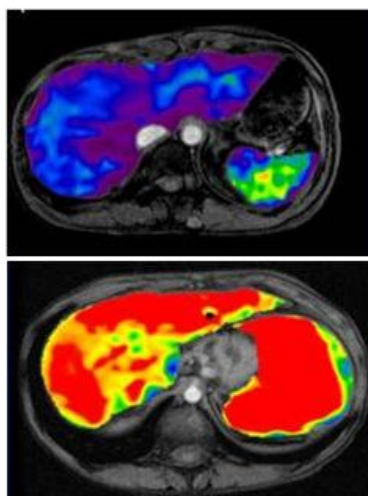
LiverMultiScan*



Whole liver assessment of fibrosis using multi-parametric magnetic resonance imaging

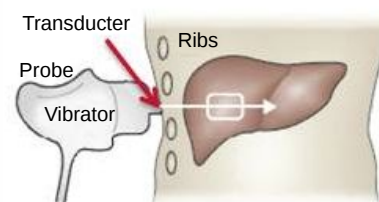
*Perspectum Diagnostics™

MR-Elastography



Whole liver assessment of stiffness using magnetic resonance imaging with mechanical pulse

Transient Elastography (FibroScan™)



Regional assessment of liver for tissue stiffness (FDA approved)

Echosense™

- Galectin-3 a well-validated target in pre-clinical studies
- GR-MD-02 binds gal-3 and is targeted to macrophages
- In pre-clinical models GR-MD-02 has multiple effects
 - Reduces inflammation, fat and ballooning hepatocytes in NASH
 - Reduces and reverses liver fibrosis and cirrhosis
 - Reduces portal pressure, an endpoint of NASH-CX trial
- In NASH patients with advanced fibrosis, GR-MD-02 is safe, well tolerated and therapeutic doses have been defined
- GR-MD-02 is well suited to target NASH with advanced fibrosis and cirrhosis, an area with less competition than early NASH
- Phase 2 clinical trial program address different patient populations
 - NASH-CX trial in cirrhosis with top line results end of 2017
 - NASH-FX trial in stage 3 fibrosis with top line results 2H 2016

Lead Indication in Cancer Immunotherapy

ADVANCED MELANOMA

Potential Sites Of Action For Galectin Inhibition In Tumor Immunology

Immunotherapies

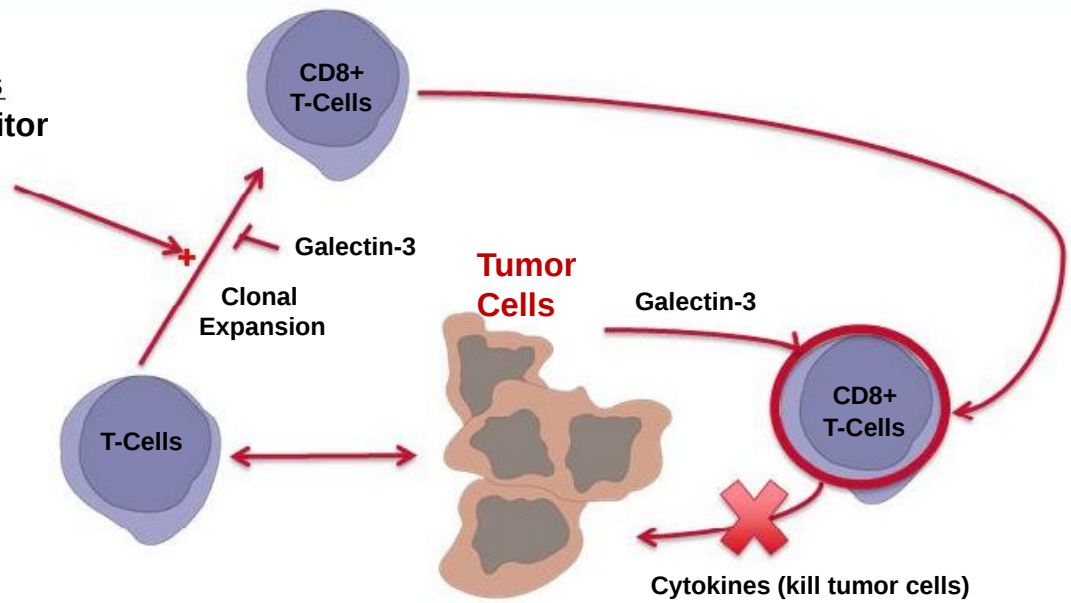
Checkpoint Inhibitor

Blockage:

anti-CTLA4

anti-PD1

Tumor Vaccines



Potential for galectin inhibitors to enhance anti-tumor immune response

Potential for galectin inhibitors to enhance anti-tumor activity of T-cells by blocking "Galectin Effect"

Focus on Immunotherapy

- Immunotherapy is a major breakthrough in cancer therapeutics
- Galectin-3 has an important role in reducing the ability of immune system to fight cancer
- GR-MD-02 is efficacious on tumors in combination with other immunotherapies in animal models

Advanced Melanoma as Initial Indication

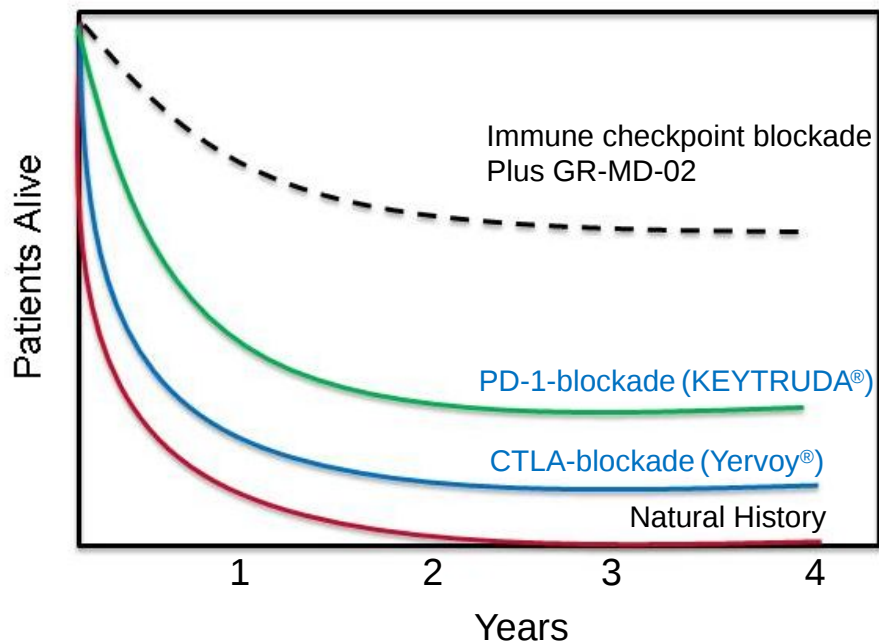
- In U.S. 76,000 new diagnoses and 9,100 deaths*
- Even with newly approved drugs, a substantial unmet medical need remains

Critical Collaboration Established

- Robert W. Franz Cancer Research Center, Earle A. Chiles Research Institute (EACRI) Providence-Portland Medical Center, Portland Oregon
- Demonstrated clinical trial expertise in melanoma and tumor immunology basic science research
- Ability to conduct clinical trials and assist in funding

*Siegel, et al. CA Cancer J Clin 2012;62:10

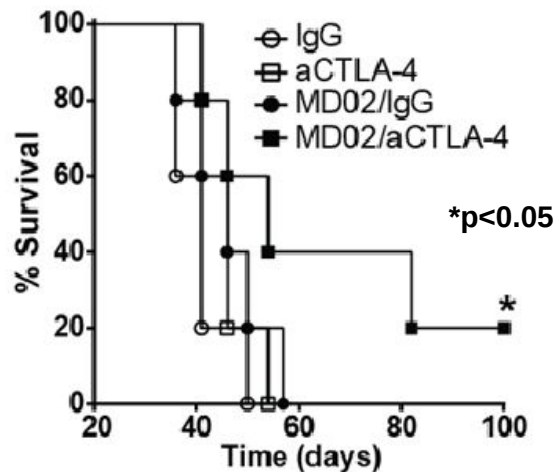
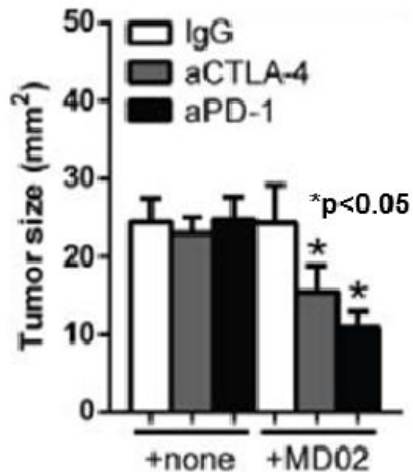
GR-MD-02 May Be A Complementary Therapy To Enhance Efficacy Of Immune Checkpoint Blockade Therapies



Note: these are illustrative curves not representative of actual data; redrawn from figure of the American Association for Cancer Research, 2013

Checkpoint Inhibitors Plus GR-MD-02 Boosts Anti-Tumor Immunity, Reduces Tumor Size And Increases Survival In Mouse Cancer Models

These data are on TC-1 prostate cancer cells (also effective in breast cancer, melanoma, and sarcoma)



aCTLA-4 = anti-CTLA-4 mAb [ipilimumab in humans (Yervoy[®], BMS)]

aPD-1 = anti-PD-1 mAb [pembrolizumab in humans (KEYTRUDA[®]) Merck]

Unpublished data 2013: Stefanie N. Linch, Melissa J. Kasiewicz, Peter G. Traber, and William L. Redmond, Galectin Therapeutics and Earle A. Chiles Research Institute (EACRI), Portland Oregon

Patients	Advanced melanoma with indication for Yervoy® treatment
Design	Three patients per cohort (+3 if serious adverse events) with 10 patients treated with maximum dose achieved
Dose	GR-MD-02 starting at dose of 1 mg/kg and escalating following each cohort to 8 mg/kg followed by standard dose of Yervoy®
Primary Endpoint	Determine a safe dose of GR-MD-02 used in combination with the approved dose of Yervoy®
Secondary Endpoints	<ul style="list-style-type: none"> • Measure the response rate as assessed by ir-RECIST criteria • Assess the biological activity by measuring: <ul style="list-style-type: none"> - CD4+ T cells with a memory phenotype - CD8+ T cells with effector phenotype - Melanoma-specific T cells using autologous and/or HLA-matched tumor - Examine composition of the tumor immune infiltrate from tumor biopsies • Assess quality of life during therapy using the FACT-M questionnaire.
Trial Site	Providence-Portland Medical Center (Dr. Brendan Curti)
Status	Cohort 1 completed—no dose limiting toxicity Cohort 2 underway (2 patients enrolled)

<http://clinicaltrials.gov/ct2/show/NCT02117362?term=GR-MD-02&rank=1>

Patients	<ul style="list-style-type: none"> • Patients who have had melanoma progression after Yervoy® and/or BRAF targeted therapy in melanomas with a BRAF mutation • Patients who have had melanoma progression after Keytruda® monotherapy
Design	Three patients per cohort (+3 if adverse events) with 10 patients treated with maximum dose achieved
Dose	GR-MD-02 IV starting at dose of 1 mg/kg and escalating following each cohort to 8 mg/kg followed by standard dose of Keytruda®
Primary Endpoint	Determine a safe dose of GR-MD-02 used in combination with the approved dose of Keytruda®
Secondary Endpoints	<ul style="list-style-type: none"> • Measure the response rate as assessed by ir-RECIST criteria • Assess the biological activity by measuring: <ul style="list-style-type: none"> -CD4+ T cells with a memory phenotype -CD8+ T cells with an effector phenotype -Melanoma-specific T cells using autologous and/or HLA-matched tumor -Examine composition of the tumor immune infiltrate from tumor biopsies • Assess quality of life during therapy using the FACT-M questionnaire
Trial Site	Providence-Portland Medical Center (Dr. Brendan Curti)
Status	Plan to initiate study July 2015

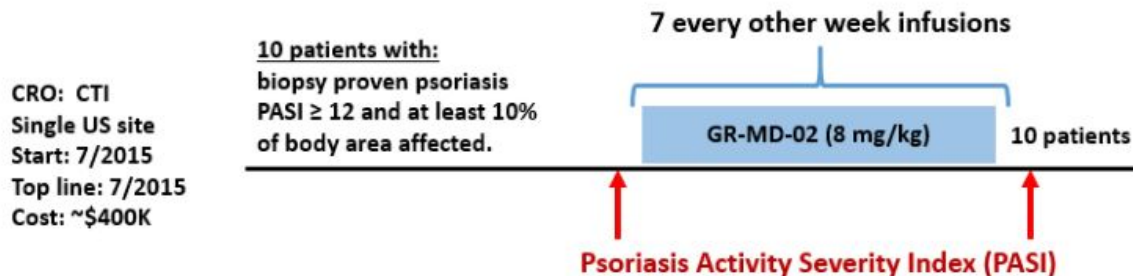
- Collaboration with investigative group at Providence Portland Medical Center (PPMC) who have significant expertise in basic tumor immunology and translational clinical trials
- Pre-clinical studies demonstrate in multiple cancers that GR-MD-02 augments the anti-tumor effects of monoclonal antibody checkpoint inhibitors
- Initial target is advanced melanoma, but also applicable to other cancer types
- Two Phase 1b clinical trials with GR-MD-02 in combination with Yervoy (ongoing) and KEYTRUDA (planned), funded by PPMC
- Advanced immune response markers being used to evaluate drug effect in addition to tumor response

Other Potential Indications in Inflammatory Disease

MODERATE TO SEVERE PLAQUE PSORIASIS

Exploratory Phase 2a Trial In Moderate to Severe Plaque Psoriasis

- Patient in Phase 1 trial had apparent remission of severe psoriasis while receiving GR-MD-02
- Galectin-3 is increased in skin lesions of patients with psoriasis



Primary Endpoint:

- Evaluate the number of patients who have 75% improvement in Psoriasis Activity Severity Index (PASI-75) following 12 weeks of therapy with GR-MD-02

Secondary Endpoints:

- PASI-50 and PASI-100 scores following 12 weeks of therapy with GR-MD-02
- Determine the durability of response to therapy in responders over a one year period following the end of therapy
- Determine whether there is any change in disease status of patients who also have psoriatic arthritis

<https://clinicaltrials.gov/ct2/show/NCT02407041?term=GR-MD-02&rank=4>

Clinical Focus		Stage of Development				
Drug	Indication	Discovery	Preclinical	Phase 1	Phase 2	Phase 3
Fibrosis (monotherapy)						
GR-MD-02	NASH cirrhosis				Q2 2015	
	NASH advanced fibrosis				Q2 2015	
	Lung, Kidney, Cardiovascular fibrosis					
Cancer Immunotherapy (combination therapy)						
GR-MD-02 + Yervoy	Melanoma					
GR-MD-02 + Keytruda	Melanoma				Q2 2015	
Plaque Psoriasis						
GR-MD-02	Moderate-severe				7/2015	
Galectin-3 Inhibitors						
Discovery program to identify subcutaneous and oral forms of carbohydrates and oral small molecules						

Advanced Liver Fibrosis/Cirrhosis

Study	Indication	Endpoints	Start	Data Reporting
GT-026 "NASH-CX"	NASH with cirrhosis	Portal pressure (HVPG); liver biopsy	June 2015	End 2017
GT-028 "NASH-FX"	NASH with advanced fibrosis	Multi-parametric MRI Comparisons include MRE and FibroScan®	July 2015	2H 2016

Advanced Melanoma

Study	Indication	Endpoints	Start	Data Reporting
Phase 1b: Yervoy®	Advanced melanoma	Safety ir-RECIST Immune markers	Underway	Dose Group 1: complete Dose Group 2: initiated, two patients enrolled
Phase 1b: KEYTRUDA®	Advanced melanoma	Safety ir-RECIST Immune markers	July 2015	TBD

Psoriasis

Study	Indication	Endpoints	Start	Data Reporting
Phase 2a: GT-030	Moderate to severe plaque psoriasis	Psoriasis Activity Severity Index (PASI)	July 2015	July 2016