GALECTIN THERAPEUTICS INC
Form 10-Q
August 14, 2018
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UNITED STATES

SECURITIES AND EXCHANGE COMMISSION

Washington, D.C. 20549

FORM 10-Q

Quarterly report pursuant to Section 13 or 15(d) of the Securities Exchange Act of 1934
For the quarterly period ended June 30, 2018

Transition report pursuant to Section 13 or 15(d) of the Securities Exchange Act of 1934
For the transition period from _____ to _____

Commission File No. 001-31791

GALECTIN THERAPEUTICS INC.

Nevada	**04-3562325**
(State or other jurisdiction	**(I.R.S. Employer**

of incorporation)	**Identification No.)**
4960 Peachtree Industrial Blvd.,	**30071**
Suite 240, Norcross, GA **(Address of Principal Executive Offices)**	**(Zip Code)**

(678) 620-3186

(Registrant s Telephone Number, Including Area Code)

Indicate by check mark whether the registrant (1) has filed all reports required to be filed by Section 13 or 15(d) of the Securities Exchange Act of 1934 during the preceding 12 months (or for such shorter period that the registrant was required to file such reports), and (2) has been subject to such filing requirements for the past 90 days. Yes No

Indicate by check mark whether the registrant has submitted electronically and posted on its corporate Web site, if any, every Interactive Data File required to be submitted and posted pursuant to Rule 405 of Regulation S-T (§232.05 of this chapter) during the preceding 12 months (or for such shorter period that the registrant was required to submit and post such files). Yes No

Indicate by check mark whether the registrant is a large accelerated filer, an accelerated filer, a non-accelerated filer or a smaller reporting company. See the definitions of large accelerated filer, accelerated filer, smaller reporting company and emerging growth company in Rule 12b-2 of the Exchange Act.

Large Accelerated Filer	Accelerated Filer
Non-Accelerated Filer (Do not check if a smaller reporting company)	Smaller reporting company
	Emerging growth company

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act.

Indicate by check mark whether the registrant is a shell company (as defined in Rule 12b-2 of the Exchange Act). Yes No

The number of shares outstanding of the registrant s common stock as of August 8, 2018 was 40,913,855.

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FOR THE QUARTER ENDED JUNE 30, 2018

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GALECTIN THERAPEUTICS INC.

CONDENSED CONSOLIDATED BALANCE SHEETS (UNAUDITED)

	June 30, 2018	December 31, 2017
	(in thousands)	
ASSETS		
Current assets:		
Cash and cash equivalents	$ 10,497	$ 3,053
Prepaid expenses and other current assets	541	766
Total current assets	11,038	3,819
Other	172	342
Total assets	$ 11,210	$ 4,161
LIABILITIES, REDEEMABLE CONVERTIBLE PREFERRED STOCK AND STOCKHOLDERS EQUITY (DEFICIT)		
Current liabilities:		
Accounts payable	$ 220	$ 608
Accrued expenses	1,148	2,292
Accrued dividends payable	67	68
Total current liabilities	1,435	2,968
Total liabilities	1,435	2,968
Commitments and contingencies (Note 9)		
Series C super dividend convertible preferred stock; 1,000 shares authorized, 176 shares issued and outstanding at June 30, 2018 and December 31, 2017, redemption value: $7,983,000, liquidation value: $1,760,000 at June 30, 2018	1,723	1,723
Stockholders equity (deficit):		
Undesignated stock, $0.01 par value; 20,000,000 shares authorized, 20,000,000 designated at June 30, 2018 and December 31, 2017, respectively		
Series A 12% convertible preferred stock; 1,742,500 shares authorized, 1,352,500 and 1,377,500 issued and outstanding at June 30, 2018 and December 31, 2017, respectively, liquidation value $1,352,500 at June 30, 2018	547	557
Series B-1 12% convertible preferred stock; 900,000 shares authorized, issued and outstanding at June 30, 2018 and December 31, 2017, liquidation value $1,800,000 at June 30, 2018	1,761	1,761
Series B-2 12% convertible preferred stock; 2,100,000 shares authorized, issued and outstanding at June 30, 2018 and December 31, 2017, liquidation value $4,200,000 at June 30, 2018	3,697	3,697
	1,224	1,224

Series B-3 8% convertible preferred stock; 2,508,000 shares authorized, 2,508,000 issued and outstanding at June 30, 2018 and December 31, 2017, liquidation value $2,508,000 at June 30, 2018		
Common stock, $0.001 par value; 100,000,000 and 50,000,000 shares authorized at June 30, 2018 and December 31, 2017, respectively, 40,628,183 and 35,789,388 issued and outstanding at June 30, 2018 and December 31, 2017, respectively	41	36
Additional paid-in capital	190,602	173,363
Retained deficit	(189,820)	(181,168)
Total stockholders equity (deficit)	8,052	(530)
Total liabilities, redeemable convertible preferred stock and stockholders equity(deficit)	$ 11,210	$ 4,161

See notes to unaudited condensed consolidated financial statements.

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GALECTIN THERAPEUTICS INC.

CONDENSED CONSOLIDATED STATEMENTS OF OPERATIONS (UNAUDITED)

	Three Months Ended June 30,		Six Months Ended June 30,	
	2018	2017	2018	2017
	(in thousands, except per share data)		(in thousands, except per share data)	
Operating expenses:				
Research and development	$ 1,476	$ 3,444	$ 3,774	$ 7,216
General and administrative	2,283	1,070	4,163	2,244
Total operating expenses	3,759	4,514	7,937	9,460
Total operating loss	(3,759)	(4,514)	(7,937)	(9,460)
Other income (expense):				
Interest income	4	6	8	15
Interest expense	(85)		(169)	
Total other income (expense)	(81)	6	(161)	15
Net loss	$ (3,840)	$ (4,508)	$ (8,098)	$ (9,445)
Preferred stock dividends	(268)	(301)	(553)	(573)
Net loss applicable to common stockholders	$ (4,108)	$ (4,809)	$ (8,651)	$ (10,018)
Net loss per common share basic and diluted	$ (0.11)	$ (0.14)	$ (0.23)	$ (0.29)
Weighted average common shares outstanding basic and diluted	38,227	34,692	37,755	34,312

See notes to unaudited condensed consolidated financial statements.

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GALECTIN THERAPEUTICS INC.

CONDENSED CONSOLIDATED STATEMENTS OF CASH FLOWS (UNAUDITED)

	Six Months Ended June 30,	
	2018	2017
	(in thousands)	
CASH FLOWS FROM OPERATING ACTIVITIES:		
Net loss	$ (8,098)	$ (9,445)
Adjustments to reconcile net loss to net cash used in operating activities:		
Depreciation and amortization		1
Stock-based compensation expense	2,630	599
Issuance of common stock for services	10	18
Non-cash interest expense	169	
Changes in operating assets and liabilities:		
Prepaid expenses and other assets	226	230
Accounts payable and accrued expenses	(1,532)	(268)
Net cash used in operating activities	(6,595)	(8,865)
CASH FLOWS FROM FINANCING ACTIVITIES:		
Net proceeds from issuance of common stock and warrants	14,039	2,630
Net cash provided by financing activities	14,039	2,630
NET (DECREASE) INCREASE IN CASH AND CASH EQUIVALENTS	7,444	(6,235)
CASH AND CASH EQUIVALENTS, BEGINNING OF PERIOD	3,053	15,362
CASH AND CASH EQUIVALENTS, END OF PERIOD	$ 10,497	$ 9,127
NONCASH FINANCING ACTIVITIES:		
Payment of preferred stock dividends in common stock	$ 553	$ 573

See notes to unaudited condensed consolidated financial statements.

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GALECTIN THERAPEUTICS INC.

NOTES TO UNAUDITED CONDENSED CONSOLIDATED FINANCIAL STATEMENTS

1. Basis of Presentation

Galectin Therapeutics Inc. (the Company) is a clinical stage biopharmaceutical company that is applying its leadership in galectin science and drug development to create new therapies for fibrotic disease, skin diseases and cancer. These candidates are based on the Company s targeting of galectin proteins which are key mediators of biologic and pathologic functions. These compounds also may have application for drugs to treat other diseases and chronic health conditions.

The unaudited condensed consolidated financial statements as reported in this Quarterly Report on Form 10-Q reflect all adjustments which are, in the opinion of management, necessary to present fairly the financial position of the Company as of June 30, 2018 and the results of its operations for the three and six months ended June 30, 2018 and 2017 and its cash flows for the six months ended June 30, 2018 and 2017. All adjustments made to the interim financial statements include all those of a normal and recurring nature. Amounts presented in the condensed consolidated balance sheet as of December 31, 2017 are derived from the Company s audited consolidated financial statements as of that date, but do not include all of the information and footnotes required by accounting principles generally accepted in the United States of America for complete financial statements. The Company considers events or transactions that occur after the balance sheet date but before the financial statements are issued to provide additional evidence relative to certain estimates or to identify matters that require additional disclosure. Subsequent events have been evaluated through the date these financial statements are available to be issued. The results for interim periods are not necessarily indicative of results that may be expected for any other interim period or for the full year. The unaudited condensed consolidated financial statements of the Company should be read in conjunction with its Annual Report on Form 10-K for the year ended December 31, 2017.

The Company has operated at a loss since its inception and has had no significant revenues. The Company anticipates that losses will continue for the foreseeable future. At June 30, 2018, the Company had $10.5 million of unrestricted cash and cash equivalents available to fund future operations. The Company believes there is sufficient cash, including availability of the line of credit (discussed in Note 3 below), to fund currently planned operations at least through June 30, 2019. The Company s ability to fund operations after its current cash resources are exhausted depends on its ability to obtain additional financing or achieve profitable operations, as to which no assurances can be given. Accordingly, based on the forecasts and estimates underlying the Company s current operating plan, the financial statements do not currently include any adjustments that might be necessary if the Company is unable to continue as a going concern.

The Company was founded in July 2000, was incorporated in the State of Nevada in January 2001 under the name Pro-Pharmaceuticals, Inc., and changed its name to Galectin Therapeutics Inc. on May 26, 2011.

2. Accrued Expenses

Accrued expenses consist of the following:

	June 30, 2018	December 31, 2017

	(in thousands)	
Legal and accounting fees	$ 77	$ 74
Accrued compensation	692	790
Accrued research and development costs and other	379	1,428
Total	$ 1,148	$ 2,292

3. Line of Credit

On December 19, 2017, the Company entered into a $10 million Line of Credit arrangement with Richard E. Uihlein, a director and shareholder who has an approximate 7% ownership interest in the Company on a fully-diluted basis at December 31, 2017. Borrowings may be made by the Company through December 31, 2018. Borrowings bear interest at the Applicable Federal Rate for short term loans published by the Internal Revenue Service (1.51% on December 19, 2017). All borrowings and interest are due on December 31, 2019 but may be prepaid without penalty. In connection with the Line of Credit agreement, the Company issued to Mr. Uihlein warrants to purchase 1 million shares of the Company s common stock for $5 per share. Half of the warrants vested at closing of the Line of Credit and the other half vest ratably with borrowings under the agreement. There have been no borrowings under the Line of Credit through June 30, 2018.

The fair value of the 500,000 warrants vested at closing was $696,000 at the date of issuance based on the following assumptions: an expected life of 7 years, volatility of 98%, risk free interest rate of 2.05% and zero dividends. The fair value of the vested warrants was recorded in other current assets and other assets (non-current) as a deferred financing cost and will be amortized

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on a straight-line basis from December 19, 2017 through December 31, 2019. Amortization for the three and six months ended June 30, 2018 of $85,000 and $169,000, respectively, was recorded as interest expense. The fair value of warrants that vest in the future based on borrowings will be computed when those borrowings occur and amortized over the remaining period through December 31, 2019.

4. Stock-Based Compensation

Following is the stock-based compensation expense related to common stock options, common stock, restricted common stock and common stock warrants:

	Three Months Ended June 30,		Six Months Ended June 30,	
	2018	2017	2018	2017
Research and development	$ 627	$ 132	$ 1,161	$ 301
General and administrative	816	137	1,469	298
Total stock-based compensation expense	$ 1,443	$ 269	$ 2,630	$ 599

The following table summarizes the stock option activity in the Company s equity incentive plans, including non-plan grants to Company executives, from December 31, 2017 through June 30, 2018:

	Shares	Weighted Average Exercise Price
Outstanding, December 31, 2017	5,155,263	$ 4.11
Granted	1,001,875	5.02
Exercised	(1,829,525)	1.79
Options forfeited/cancelled	(33,334)	6.24
Outstanding, June 30, 2018	4,294,279	$ 5.30

As of June 30, 2018, there was $1,819,000 of unrecognized compensation related to 1,140,318 unvested options, which is expected to be recognized over a weighted average period of approximately 0.5 years. The weighted-average grant date fair value for options granted during the three months ended June 30, 2018 was $3.98. The Company granted 460,000 stock options In January 2018, of which 115,000 options vested upon grant with the remaining 345,000 options vesting over the remainder of 2018. Approximately $526,000 of non-cash, stock-based compensation expense was recorded during the three months ended March 31, 2018 related to the options granted in January 2018 that vested upon the grant date. Additionally, Company granted 506,875 stock options during May 2018, of which 115,000 options vested upon grant with the remaining 391,875 options vesting over the remainder of 2018. Approximately $433,000 of non-cash, stock-based compensation expense was recorded during the three months ended June 30, 2018 related to the options granted in May 2018 that vested upon the grant date.

The fair value of all other options granted is determined using the Black-Scholes option-pricing model. The following weighted average assumptions were used:

	Six Months Ended June 30, 2018	Six Months Ended June 30, 2017
Risk-free interest rate	2.47%	
Expected life of the options	5.7 years	
Expected volatility of the underlying stock	104%	
Expected dividend rate	0%	

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5. Common Stock Warrants

The following table summarizes the common stock warrant activity from December 31, 2017 through June 30, 2018:

	Shares	Weighted Average Exercise Price
Outstanding, December 31, 2017	13,229,778	$ 3.35
Granted	267	5.15
Exercised	(2,414,709)	2.50
Forfeited/cancelled		
Outstanding, June 30, 2018	10,815,336	$ 3.53

6. Fair Value of Financial Instruments

At times, the Company has certain financial assets and liabilities recorded at fair value. Fair values determined by Level 1 inputs utilize observable data such as quoted prices in active markets. Fair values determined by Level 2 inputs utilize data points other than quoted prices in active markets that are observable either directly or indirectly. Fair values determined by Level 3 inputs utilize unobservable data points in which there is little or no market data, which require the reporting entity to develop its own assumptions. The carrying amounts reflected in the consolidated balance sheets for cash equivalents, accounts payable and accrued expenses approximate their carrying value due to their short-term nature. There were no level 2 or level 3 assets or liabilities at June 30, 2018 or December 31, 2017.

7. Loss Per Share

Basic net loss per common share is computed by dividing the net loss available to common stockholders by the weighted average number of common shares outstanding during the period. Diluted net loss per common share is computed by dividing the net loss available to common stockholders by the weighted average number of common shares and other potential common shares then outstanding. Potential common shares consist of common shares issuable upon the assumed exercise of in-the-money stock options and warrants and potential common shares related to the conversion of the preferred stock. The computation of diluted net loss per share does not assume the issuance of common shares that have an anti-dilutive effect on net loss per share.

Dilutive shares which could exist pursuant to the exercise of outstanding stock instruments and which were not included in the calculation because their affect would have been anti-dilutive are as follows:

	June 30, 2018 (shares)	June 30, 2017 (shares)
Warrants to purchase shares of common stock	10,815,336	12,249,189
Options to purchase shares of common stock	4,294,279	4,656,888
Shares of common stock issuable upon conversion of preferred stock	4,308,115	4,312,282
	19,417,730	21,218,359

8. Common Stock

2014 At Market Issuance of Common Stock

On March 30, 2014, the Company entered into an At Market Issuance Sales Agreement (the 2014 At Market Agreement) with a sales agent under which the Company may issue and sell shares of its common stock having an aggregate offering price of up to $30.0 million from time to time through the sales agent. Sales of the Company s common stock through the sales agent, if any, will be made by any method that is deemed an at the market offering as defined by the U.S. Securities and Exchange Commission. The Company will pay to the sales agent a commission rate equal to 3.0% of the gross proceeds from the sale of any shares of common stock sold through the sales agent under the 2014 At Market Agreement. During the three months ended March 31, 2017, the Company issued 1,496,797 shares of common stock for net proceeds of approximately $1,945,000 under the 2014 At Market Agreement. The 2014 At Market Agreement expired in March 2017.

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2017 At Market Issuance of Common Stock

On May 19, 2017, the Company entered into an At Market Issuance Sales Agreement (the 2017 At Market Agreement) with a sales agent under which the Company may issue and sell shares of its common stock having an aggregate offering price of up to $30.0 million from time to time through the sales agent. Sales of the Company s common stock through the sales agent, if any, will be made by any method that is deemed an at the market offering as defined by the U.S. Securities and Exchange Commission. The Company will pay to the sales agent a commission rate equal to 3.0% of the gross proceeds from the sale of any shares of common stock sold through the sales agent under the 2017 At Market Agreement. During June 2018, the Company issued 618,614 shares of common stock for net proceeds of approximately $5,241,000 under the 2017 At Market Agreement.

2017 Private Placement

On February 28, 2017, the Company closed a transaction with five individual investors through a private placement of common stock and warrants. In total, the Company issued 102,368 shares of common stock for proceeds of $200,000. The Company also issued, to the five investors, warrants to purchase 76,776 shares of common stock at $5.00 per share. The warrants have an expiration date of February 28, 2024. The exercise price of each warrant is adjustable in the event of a stock split or stock combination, capital reorganization, merger or similar event. The warrants were valued at approximately $101,000 as of the date of issuance, using the closing price of $1.86, a life of 7 years, a volatility of 97% and a risk-free interest rate of 1.92%. Based upon the Company s analysis of the criteria contained in ASC Topic 815-40, Derivatives and Hedging Contracts in Entity s Own Equity the Company has determined that warrants issued in connection with this financing transaction were not derivative liabilities and therefore, were recorded as additional paid-in capital.

Other

In 2018, the Company entered an agreement with a vendor whereby the Company will issue common stock to vendor in lieu of paying in cash in amount up to $100,000 for the year. For the six months ended June 30, 2018, the Company has issued 2,655 shares of common stock and 267 warrants to purchase shares of common stock pursuant to this agreement and the value of such shares has been recorded as research and development expense.

For the six months ended June 30, 2018, the Company has issued a total of 106,082 shares of common stock for dividends on Series A, Series B and Series C Preferred Stock.

9. Commitments and Contingencies

The Company records accruals for such contingencies to the extent that the Company concludes that their occurrence is probable, and the related damages are estimable. There are no other significant pending legal proceedings.

10. Galectin Sciences LLC

In January 2014, we created Galectin Sciences, LLC (the LLC or Investee), a collaborative joint venture co-owned by SBH Sciences, Inc. (SBH), to research and develop small organic molecule inhibitors of galectin-3 for oral administration. The LLC was initially capitalized with a $400,000 cash investment to fund future research and development activities, which was provided by the Company, and specific in-process research and development (IPR&D) contributed by SBH. The estimated fair value of the IPR&D contributed by SBH, on the date of contribution, was $400,000. Initially, the Company and SBH each had a 50% equity ownership interest in the LLC, with neither party having control over the LLC. Accordingly, from inception through the fourth quarter of 2014, the

Company accounted for its investment in the LLC using the equity method of accounting. Under the equity method of accounting, the Company s investment was initially recorded at cost with subsequent adjustments to the carrying value to recognize additional investments in or distributions from the Investee, as well as the Company s share of the Investee s earnings, losses and/or changes in capital. The estimated fair value of the IPR&D contributed to the LLC was immediately expensed upon contribution as there was no alternative future use available at the point of contribution. The operating agreement provides that if either party does not desire to contribute its equal share of funding required after the initial capitalization, then the other party, providing all of the funding, will have its ownership share increased in proportion to the total amount contributed from inception. In the fourth quarter of 2014, after the LLC had expended the $400,000 in cash, SBH decided not to contribute its share of the funding required. Since then, the Company has contributed a total of $1,723,000, including $60,000 for the three months ended June 30, 2018, for expenses of the LLC. Since the end of 2014, SBH has contributed $123,000 for expenses in the LLC. As of June 30, 2018, the Company s ownership percentage in the LLC was 80.2%. The Company accounts for the interest in the LLC as a consolidated, less than wholly owned subsidiary. Because the LLC s equity is immaterial, the value of the non-controlling interest is also deemed to be immaterial.

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Item 2. Management s Discussion and Analysis of Financial Condition and Results of Operations

In addition to historical information, the following Management s Discussion and Analysis of Financial Condition and Results of Operations contains forward-looking statements as defined under Section 21E of the Securities Exchange Act of 1934, as amended, and is subject to the safe harbor created therein for forward-looking statements. Such statements include, but are not limited to, statements concerning our anticipated operating results, research and development, clinical trials, regulatory proceedings, and financial resources, and can be identified by use of words such as, for example, anticipate, estimate, expect, project, intend, plan, believe and would, should, statements, other than statements of historical facts, included herein that address activities, events, or developments that the Company expects or anticipates will or may occur in the future, are forward-looking statements, including statements regarding: plans and expectations regarding clinical trials; plans and expectations regarding regulatory approvals; our strategy and expectations for clinical development and commercialization of our products; potential strategic partnerships; expectations regarding the effectiveness of our products; plans for research and development and related costs; statements about accounting assumptions and estimates; expectations regarding liquidity and the sufficiency of cash to fund currently planned operations through at least June 30, 2019; our commitments and contingencies; and our market risk exposure. Forward-looking statements are based on current expectations, estimates and projections about the industry and markets in which Galectin Therapeutics operates, and management s beliefs and assumptions. These statements are not guarantees of future performance and involve certain known and unknown risks and uncertainties that could cause actual results to differ materially from those expressed or implied by such statements. Such risks and uncertainties are related to and include, without limitation,

our early stage of development,

we have incurred significant operating losses since our inception and cannot assure you that we will generate revenue or profit,

our dependence on additional outside capital,

we may be unable to enter into strategic partnerships for the development, commercialization, manufacturing and distribution of our proposed product candidates,

uncertainties related to any litigation,

uncertainties related to our technology and clinical trials, including expected dates of availability of clinical data,

we may be unable to demonstrate the efficacy and safety of our developmental product candidates in human trials,

we may be unable to improve upon, protect and/or enforce our intellectual property,

we are subject to extensive and costly regulation by the U.S. Food and Drug Administration (FDA) and by foreign regulatory authorities, which must approve our product candidates in development and could restrict the sales and marketing and pricing of such products,

competition and stock price volatility in the biotechnology industry,

limited trading volume for our stock, concentration of ownership of our stock, and other risks detailed herein and from time to time in our SEC reports, and

The following discussion should be read in conjunction with the accompanying consolidated financial statements and notes thereto of Galectin Therapeutics appearing elsewhere herein.

Overview

We are a clinical stage biopharmaceutical company engaged in drug research and development to create new therapies for fibrotic disease, severe skin disease, and cancer. Our drug candidates are based on our method of targeting galectin proteins, which are key mediators of biologic and pathologic functions. We use naturally occurring, readily-available plant products as starting material in manufacturing processes to create proprietary complex carbohydrates with specific molecular weights and other pharmaceutical properties. These complex carbohydrate molecules are appropriately formulated into acceptable pharmaceutical formulations. Using these unique carbohydrate-based candidate compounds that largely bind and inhibit galectin proteins, particularly galectin-3, we are undertaking the focused pursuit of therapies for indications where galectins have a demonstrated role in the pathogenesis of a given disease. We focus on diseases with serious, life-threatening consequences to patients and those where current treatment options are limited. Our strategy is to establish and implement clinical development programs that add value to our business in the shortest period of time possible and to seek strategic partners when a program becomes advanced and requires significant additional resources.

Our lead galectin-3 inhibitor is GR-MD-02, which has been demonstrated in preclinical models to reverse liver fibrosis and cirrhosis. GR-MD-02 has the potential to treat many diseases due to galectin-3 s involvement in multiple key biological pathways such as immune cell function and immunity, cell differentiation, cell growth, and apoptosis (cell death). Galectin Therapeutics Inc. is using this inhibitor to treat advanced liver fibrosis and liver cirrhosis in NASH (non-alcoholic steatohepatitis) patients. We have

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completed two Phase 1 clinical studies, a Phase 2 clinical study in NASH patients with advanced fibrosis (NASH-FX) and a second Phase 2B clinical trial in NASH patients with well compensated cirrhosis. We announced, in December 2017 top line results from our Phase 2b study in NASH patients with cirrhosis (NASH-CX). NASH cirrhosis is a progressive disease, currently not treatable and ultimately may result in liver failure that has poor prognosis and no effective, approved medical therapies other than liver transplant. Galectin-3 expression is highly increased in the liver of patients with liver fibrosis and liver cirrhosis. We believe that our galectin-3 inhibitor, by reducing galectin-3 at the cellular level, ultimately showing a strong anti-fibrotic potential may provide a novel treatment for various forms of liver fibrosis.

We endeavor to leverage our scientific and product development expertise as well as established relationships with outside sources to achieve cost-effective and efficient drug development. These outside sources, amongst others, provide us with expertise in preclinical models, pharmaceutical development, toxicology, clinical trial operations, pharmaceutical manufacturing, sophisticated physical and chemical characterization, and commercial development. We also have established several collaborative scientific discovery programs with leading experts in carbohydrate chemistry and characterization. These discovery programs are generally aimed at the targeted development of new carbohydrate molecules that bind galectin proteins and offer alternative options to larger market segments in our primary disease indications. We also have established through Galectin Sciences LLC, a discovery program aimed at the targeted development of small molecules (generally, non-carbohydrate) that bind galectin proteins and may afford options for alternative means of drug delivery (e.g., oral) and as a result expand the potential uses of our galectin-3 inhibitor compounds. We are also pursuing a development pathway to clinical enhancement and commercialization for our lead compounds in immuno-oncology for cancer therapy and severe skin disease including moderate to severe plaque psoriasis and severe atopic dermatitis. However, our clinical development efforts are focused on both liver fibrosis and fatty liver disease as represented by a Phase 2 clinical trial in NASH-cirrhosis which reported top line data in December 2017. All of our proposed products are presently in development, including pre-clinical and clinical trials.

Our Drug Development Programs

Galectins are a class of proteins that are made by many cells in the body, but predominantly in cells of the immune system. As a group, these proteins are able to bind to sugar molecules that are part of other proteins, glycoproteins, in and on the cells of our body. Galectin proteins act as a kind of molecular glue, bringing together molecules that have sugars on them. Galectin proteins, in particular galectin-3, are known to be markedly increased in a number of important diseases including inflammatory diseases, scarring of organs (e.g. liver, lung, kidney, and heart) and cancers of many kinds. The increase in galectin protein promotes the disease and is detrimental to the patient. Published data substantiating the importance of galectin-3 in the fibrotic process arises from gene knockout experiments in animal studies. Mice genetically altered to eliminate the galectin-3 gene, and thus unable to produce galectin-3, are incapable of developing liver fibrosis in response to toxic insult to the liver and in fatty liver disease as well as development of fibrosis in other tissues.

We have one new proprietary chemical entity (NCE) in development, GR-MD-02, which has shown promise in preclinical and early clinical studies in treatment of fibrosis, severe skin disease, and in cancer therapy. Currently we are focusing on development of GR-MD-02 intended to be used in the treatment of liver fibrosis associated with fatty liver disease (NASH) and more specifically in NASH cirrhosis. We have also leveraged our relationships with well-known investigators to demonstrate clinical effects of GR-MD-02 in treating moderate to severe plaque psoriasis, severe atopic dermatitis, and in cancer therapy in combination with immune-system modifying agent(s). GR-MD-02 is a proprietary, patented compound derived from natural, readily available, plant-based starting materials, which, following chemical processing, exhibits the properties of binding to and inhibiting galectin-3 proteins. A second NCE, GM-CT-01 is a proprietary, patented compound that is made from a completely different starting source plant material

and also binds and inhibits galectin proteins.

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Our product pipeline is shown below:

Indication	Drug	Status
Fibrosis		
NASH with Advanced Fibrosis: NASH-CX trial and NASH-FX trial	GR-MD-02	IND submitted January 2013. Results from the Phase 1 clinical trial were reported in 2014, with final results reported in January 2015. End of Phase 1 meeting held with FDA in 2014. Two Phase 2 clinical trials were designed. The NASH FX trial was designed for patients with advanced fibrosis but not cirrhosis. The NASH FX trial top line data was reported in September 2016 The NASH CX trial was designed for patients with well compensated cirrhosis. The NASH CX trial top line data was reported in December 2017 and additional results derived from the study were presented subsequently.
Indication	**Drug**	**Status**
Lung Fibrosis	GR-MD-02	In pre-clinical development
Kidney Fibrosis	GR-MD-02	In pre-clinical development
Cardiac and Vascular Fibrosis	GR-MD-02	In pre-clinical development
	GM-CT-01	Not in active development
Cancer Immunotherapy		
Melanoma, Head, Neck Squamous Cell Carcinoma (HNSCC)	GR-MD-02	Investigator IND submitted in December 2013. Phase 1B study in process. A second Phase 1B study began in Q-1 2016. Investigator IND for that study submitted in September 2015. Early data was reported in February 2017 and studies with the 3rd cohort are ongoing.
Psoriasis		
Moderate to Severe Plaque Psoriasis Severe Atopic Dermatitis	GR-MD-02	IND submitted March 2015. A phase 2a trial in moderate to severe plaque psoriasis patients began in January 2016. Interim data on the first four patients were positive and were reported in May 2016. Further positive data was reported in September 2016. Investigator initiated IND submitted for treatment of three patients with severe atopic dermatitis, with positive preliminary data presented in February 2017.

Fibrosis. GR-MD-02 is our lead product candidate for treatment of fibrotic disease. Our preclinical data show that GR-MD-02 has a significant therapeutic effect on liver fibrosis as shown in several relevant animal models. In addition, in NASH animal models, GR-MD-02 has been shown to reduce liver fat, inflammation, and ballooning degeneration or death of liver cells. Therefore, we chose GR-MD-02 as the lead candidate in a development program targeted initially at fibrotic liver disease associated with non-alcoholic steatohepatitis (NASH, or fatty liver disease). In January 2013, an Investigational New Drug (IND) was submitted to the U.S. Food and Drug Administration (FDA) with the goal of initiating a Phase 1 study in patients with NASH and advanced liver fibrosis to evaluate the human safety of GR-MD-02 and pharmacodynamics biomarkers of disease. On March 1, 2013, the FDA indicated we could proceed with a US Phase 1 clinical trial for GR-MD-02 with a development program aimed at obtaining support for a proposed indication of GR-MD-02 for treatment of NASH with advanced fibrosis. The Phase 1 trial was completed

and demonstrated that GR-MD-02 up to 8 mg/kg, i.v. was safe and well tolerated. The human pharmacokinetic data defined a drug dose for use in the planned Phase 2 trials based on extrapolation from efficacy data in NASH animal models of liver fibrosis and/or cirrhosis. Additionally, there was evidence of a pharmacodynamic effect of GR-MD-02 at the 8 mg/kg dose with a decrease in alpha 2 macroglobulin, a serum marker of fibrotic activity, and a reduction in liver stiffness as determined by FibroScan®. An End of Phase 1 Meeting was held with FDA which, amongst other items, provided guidance on the primary endpoint for the Phase 2 clinical trial, the NASH-CX trial.

Additionally, an open label drug-drug interaction study was completed in healthy volunteers during the second quarter of 2015 with GR-MD-02, and it showed that with 8 mg/kg dose of GR-MD-02 and 2 mg/kg dose of midazolam there was no drug-drug interaction and no serious adverse events or drug-related adverse events were observed. This study was required by the FDA, and the primary objective was to determine if single or multiple intravenous (IV) doses of GR-MD-02 affect the pharmacokinetics (PK) of midazolam. The secondary objective was to assess the safety and tolerability of GR-MD-02 when administered concomitantly with midazolam. The lack of a drug interaction in this study enabled the Company to expand the number of patients eligible for its Phase 2 clinical trial. In addition, should GR-MD-02 be approved for marketing, the success of this study supports a broader patient population for the drug label.

Our Phase 2 program in fibrotic disease consists of two separate human clinical trials. The primary clinical trial is the Phase 2b NASH-CX study for one year for patients with NASH with well compensated cirrhosis, which began enrolling in June, 2015. This study is the primary focus of our program and is a randomized, placebo-controlled, double-blind, parallel-group Phase 2b trial to evaluate the safety and efficacy of GR-MD-02 for treatment of liver fibrosis and resultant portal hypertension in NASH patients with well compensated cirrhosis. A smaller, exploratory NASH-FX trial was conducted to explore potential use of various non-invasive imaging techniques in NASH patients with advanced fibrosis but not cirrhosis.

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NASH-FX Trial: The NASH-FX trial, a Phase 2a pilot trial NASH-FX for patients with NASH advanced fibrosis that explored use of three non-invasive imaging technologies, is now complete. It was a short, single site, four-month trial in 30 NASH patients with advanced fibrosis, but not cirrhosis, randomized 1:1 to either 9 bi-weekly doses of 8 mg/kg of GR-MD-02 or placebo. The trial did not meet its primary biomarker endpoint as measured using multi-parametric magnetic resonance imaging (LiverMultiScan(R), Perspectum Diagnostics). The trial also did not meet secondary endpoints that measure liver stiffness as a surrogate for fibrosis using, magnetic resonance-elastography and FibroScan® score. We, and many experts in the field, now believe that a four-month treatment period may not be sufficient to show efficacy results in established liver fibrosis. This small study was not powered for the secondary endpoints and thus, not surprisingly did not meet the secondary endpoints. In the trial, GR-MD-02 was found to be safe and well tolerated among the patient population with no serious adverse events. Although there was no apparent improvement in the three non-invasive tests for assessment of liver fibrosis in the four-month NASH-FX trial, the principal investigator of the NASH-FX trial has stated that the inhibition of galectin-3 with GR-MD-02 remains promising for the treatment of NASH fibrosis. Of note is that GR-MD-02 has demonstrated an improved clinical effect in moderate-to-severe psoriasis, suggesting the compound has activity in humans in an immune-mediated inflammatory human disease that can occur in association with NASH. We believe our drug candidate provides a promising new approach for the therapy of fibrotic diseases, and liver fibrosis in particular. Fibrosis is the formation of excess connective tissue (collagen and other proteins plus cellular elements such as myofibroblasts) in response to damage, inflammation or repair. When the fibrotic tissue becomes confluent, it obliterates the cellular architecture, leading to scarring and dysfunction of the underlying organ. Given galectin-3 s broad biological functionality, it has been demonstrated to be involved in cancer, inflammation and fibrosis, heart disease, and renal disease. We have further demonstrated the broad applicability of the actions of our galectin-3 inhibitor s biological effect in ameliorating fibrosis involving lung, kidney, blood vessels, and cardiac tissues in a wide variety of animal models.

NASH-CX Trial: The NASH-CX trial was a larger well-designed multi-center clinical trial which explored use of GR-MD-02 for the treatment of liver fibrosis and resultant portal hypertension in patients with well-compensated NASH cirrhosis. Enrollment in this trial was completed in September, 2016, and a total of 162 patients at 36 sites in the United States were randomized to receive either 2 mg/kg of GR-MD-02, 8 mg/kg of GR-MD-02 or placebo, with approximately 54 patients in each group. The primary endpoint is a reduction in change in hepatic venous pressure gradient (HVPG). Patients received an infusion every other week for one year, total of 26 infusions, and were evaluated to determine the change in HVPG as compared with placebo. HVPG was also correlated with secondary endpoints of fibrosis on liver biopsy as well as with measurement of liver stiffness (FibroScan(R)) and assessment of liver metabolism (^{13}C-methacetin breath test, Exalenz), which are non-invasive measures of the liver that may be used in future studies. Top line data readout was reported in December 2017 demonstrating positive efficacy data and safety and clinically meaningful results in the NASH patients with well compensated cirrhosis without esophageal varices (stage 1 cirrhosis).

In the total patient population, the primary endpoint HVPG showed a trend toward benefit with GR-MD-02 treatment, but the difference from placebo was not statistically significant. The mean change in HVPG of placebo from baseline to week 54 was 0.3 mm Hg. The mean change in HVPG from baseline was -0.37 and -0.42 for the 2 mg/kg dose and 8 mg/kg dose of GR-MD-02, respectively.

Further analysis showed that the drug effect was significantly dependent on dose varices in the total group of patients ($p<0.02$). In those NASH cirrhosis patients without varices at baseline (about 50% of the total population), there was a statistically significant effect of the 2 mg/kg dose of GR-MD-02 on the absolute change in HVPG (-1.08 mm Hg, $p<0.01$). The effect of the 8 mg/kg dose of GR-MD-02 on absolute or percent change in HVPG from baseline to week 54 was not significant. The population of patients without varices at baseline were further subdivided into those with mild portal hypertension (HVPG greater or equal to 6 mm Hg and less than 10 mm Hg). In patients with mild portal hypertension (MPH), both doses of GR-MD-02 demonstrated a statistically significant effect on change in HVPG. The

mean change in HVPG in the MPH group were +1.8 mm Hg for placebo and -0.3 and -0.4 mm Hg in the 2 mg/kg and 8 mg/kg dose groups, respectively. In patients with clinically significant portal hypertension (HVPG greater than 10 Mm Hg) with no varices at baseline, there was a statistically significant effect of 2 mg/kg of GR-MD-02 on the change in HVPG.

A responder analysis was performed on those patients without varices at baseline. Analysis was performed looking at two groups: those with an equal to or greater than 2 mm Hg decrease in HVPG from baseline or those with an equal to or greater than 2 mm Hg and greater than or equal to 20% decrease in HVPG from baseline. In both cases, the change observed in the GR-MD-02 2 mg/kg group was statistically significant ($p<0.01$) while that of the 8 mg/kg group was not.

Additional *ad hoc* analysis examining the PK-PD correlation between human data and the mouse NASH model showed that the apparent lack of a dose response in the 8 mg/kg dose group (GR8) may be due to fact that a large number of patients in the GR8 group had very high levels of GR-MD-02. When the GR8 group patients were divided into groups with serum drug levels greater than 12,000 ug*hr/mL and those below 12,000 ug*hr/mL, there was a statistically significant difference in the change in HVPG in the GR8

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group with lower serum levels of GR-MD-02 of approximately -8%. Thus, the apparent less pronounced effects of GR8 may be explained, at least in part, by the variable pharmacokinetics observed in the patients which received GR8. This mirrors the effects seen in the NASH mouse model experiments in which the anti-inflammatory effects of GR-MD-02 were significantly reduced at doses between 60 mg/kg and 120 mg/kg.

In terms of cirrhosis complications over the 54-week treatment period, in patients without varices there were statistically significantly fewer new varices that developed in the treatment groups vs placebo. We believe this may represent a useful measure of clinical outcome.

The major conclusions, to date from the NASH-CX trial results are that: i) GR-MD-02 had a statistically significant and clinically meaningful effect in improving HVPG vs placebo in patients with NASH cirrhosis who did not have esophageal varices at baseline; this effect was seen regardless of the patient s baseline portal hypertension. Furthermore, we believe that patients with esophageal varices may have masked benefits in the total patient population; ii) there was an important drug effect of GR-MD-02 in the total patient population on liver biopsy with a statistically significant improvement in hepatocyte ballooning (ie cell death); (iii) there was a statistically significant reduction (p=0.02) in the development of new esophageal varices in drug-treated patients compared to placebo; we believe that this is a clinically relevant endpoint related to patient outcomes; (iv) while there was a drug effect in both the 2 mg/kg and 8 mg/kg dosage groups on liver biopsy and in the mild portal hypertension group, there was a consistently greater and statistically significant effect of the 2 mg/kg dose of GR-MD-02; (v) the apparent lack of a dose response in the GR8 group may be explained by variable pharmacokinetics in the high dose group, and this suggests use of an additional and intermediate dose between 2 and 8 mg/Kg in subsequent trials; (vi) GR-MD-02 appears to be safe and well tolerated in this one year clinical trial; and (vii) we believe this is the first large, randomized clinical trial of any drug to demonstrate a clinically meaningful improvement in portal hypertension or liver biopsy in patients with compensated NASH cirrhosis without esophageal varices.

The Company met with FDA on May 08, 2018. Following which it announced that it is proceeding with plans for a Phase 3 clinical trial program with its galectin-3 inhibitor GR-MD-02 in NASH cirrhosis, incorporating advice and guidance obtained in the meeting with the FDA.

The target population of the Phase 3 clinical trial will be patients with NASH cirrhosis without esophageal varices. The primary endpoint will be chosen from two endpoints that the FDA agreed may be acceptable: The change in hepatic venous pressure gradient (HVPG), which is a measure of liver blood pressure, or the progression to esophageal varices. Both primary endpoints may be considered surrogate endpoints for clinical outcomes in the target population with NASH cirrhosis.

The basis for advancing to Phase 3 is the positive effects of GR-MD-02 on HVPG and the possible prevention or postponement of development of esophageal varices observed in the post-hoc analysis of the Phase 2 NASH-CX trial, which we believe is the first large, randomized clinical trial of any drug to demonstrate a clinically meaningful improvement in these patients. The potential choice between two primary endpoints for Phase 3 trials provides enhanced flexibility in designing the strongest trial to replicate the efficacy demonstrated in the Phase 2 NASH-CX trial. However, there can be no assurance that a larger trial will replicate the results of the NASH-CX trial. The choice of which primary endpoint to use is a complex and important decision for the design of the Phase 3 trial and may require additional input from external advisors and the FDA. Additionally, the clinical trial design discussed with the FDA provides for interim analysis which may provide confirmation of Phase 2 results and enhanced confidence for the ultimate results of the Phase 3 trial.

As disclosed in May 2018, the FDA has not granted breakthrough designation at this time based on the Agency s current assessment that additional confirmatory data are needed to identify the level of change in HVPG that is

reasonably likely to predict clinical outcomes. Although we disagree with FDA s decision not to grant Breakthrough Therapy designation, we understand their position because while the NASH-CX trial missed on the primary endpoint in the total population, the additional analysis of NASH-CX trial is, to our knowledge, the first randomized clinical trial of any drug to demonstrate a clinically meaningful improvement in HVPG in NASH cirrhosis patients. While Breakthrough Designation was not granted at this time, the program continues to benefit from Fast Track designation which provides many of the same advantages as Breakthrough Therapy designation, including potential for accelerated approval and priority review.

Details of the Phase 3 clinical trial design, including projected timings and costs, will be announced once the planning phase has been completed. These planning activities, include amongst other items and activities, completion of a detailed final clinical trial protocol that the company believes meets regulatory expectations, and estimates for pricing and timing are derived from a Request for Proposal process involving Clinical Research Organizations. Various other activities, amongst others include, manufacturing of clinical supplies in support of a Phase 3 program and certain antecedent advanced reproductive toxicology studies as defined by FDA in accordance with guidelines of the International Committee on Harmonization, assessing the need for foreign regulatory filings to permit inclusion of additional clinical sites into the Phase 3 trial program to accelerate enrollment of patients and meet ICH expectations on minimum patient exposure requirements. The focus and goal of the therapeutic program is to stop the progression of and reverse the fibrosis in the liver and, thereby improve liver function and prevent the development of complications of fibrosis/cirrhosis and liver-related mortality in patients. The results of the NASH-CX trial suggest that, subject to confirmation in additional or later stage clinical trials, this goal is achievable in a significant portion of the NASH cirrhosis patient population i.e. those NASH cirrhosis patients without esophageal varices.

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Cancer Immunotherapy. We believe there is potential for galectin inhibition to play a key role in the burgeoning area of cancer immunotherapy. For example, there have been several recent approvals of drugs that enhance a patient s immune system to fight cancer. It is our goal to use a galectin inhibitor to enhance the immune system function to fight cancer in a way that complements other approaches to this type of therapy. This hypothesis is supported by the fact that galectin-3 is expressed at high levels in multiple types of tumors, adds to the malignant nature of the tumors, and protects the tumors from immune system attack. Our drug candidates provide a promising new therapeutic approach to enhance the activity of the immune system against cancer cells. Preclinical studies have indicated that GR-MD-02 enhances the immune response to cancer cells, increased tumor shrinkage and enhanced survival in immune competent mice with prostate, breast, melanoma and sarcoma cancers when combined with one of the immune checkpoint inhibitors, anti-CTLA-4 or anti-PD-1, and with the immune cell activator anti-OX40. These preclinical data led to the filing of two Investigator-sponsored INDs and the initiation of studies of GR-MD-02 in combination with Yervoy® (ipilimumab) and KEYTRUDA (pembrolizumab) in Phase 1B studies of patients with metastatic melanoma. The KEYTRUDA trial has also been expanded to include patients with non-small cell lung cancer and head and neck squamous cell carcinoma. These studies are being conducted under the sponsorship of Providence Portland Medical Center s Earle A. Chiles Research Institute (EACRI).

Data on this combination immunotherapy program was presented on February 7, 2017 at the 9th GTCBio Immunotherapeutics & Immunomonitoring Conference in San Diego, CA by Dr. William L. Redmond, Providence Cancer Center. Preclinical results in mouse models of multiple types of cancers showed important anti-tumor and increased survival effects of combining GR-MD-02 with different types of immune modulators, providing a case for progressing studies into human patients with cancer. Seven patients were treated in the GR-MD-02 in combination with Yervoy trial, with no safety concerns in these low dose cohorts. Due to changes in the standard of care for metastatic melanoma (i.e., approval of anti-PD-1), recruitment has been slowed significantly in this trial. Promising results were reported in the Phase 1b trial combining GR-MD-02 with pembrolizumab (KEYTRUDA). Cohort 1 was completed (n=6, 5 with melanoma, one head and neck) with one partial response and one mixed response in 5 melanoma patients. There was a rapid and marked tumor response after 3 doses of combined GR-MD-02 and pembrolizumab in the one partial response patient who had failed high-dose IL-2 and oncolytic virus + ipilimumab. The study is ongoing and progression to further development will be based on response rate as compared to historical response rates to pembrolizumab alone. Results from third cohort of patients is expected in Summer, 2018.

Severe skin diseases. During our Phase 1 NASH fibrosis trial with GR-MD-02, a clinical effect on plaque psoriasis was observed in a NASH patient who also had this disease. This patient had marked improvement in her psoriasis, with improvement beginning after the third infusion. She reported that her psoriasis was completely gone and her skin was normal after the fourth infusion. Her skin remained normal for 17 months after the final infusion of study drug. The patient is convinced that the improvement in her psoriasis is related to the study drug.

This serendipitous finding, combined with galectin-3 protein being markedly upregulated in the capillary epithelia (small blood vessels) of the psoriatic dermis (plaque lesions), led to a phase 2a trial in patients with moderate to severe plaque psoriasis. GR-MD-02 inhibition of galectin-3 may attenuate capillary changes in the psoriatic dermis and inflammatory recruitment, perhaps explaining the improvements observed in the NASH fibrosis trial patient. In this open-label, unblinded trial (no placebo, all patients knowingly receive active drug), 5 patients with moderate to severe plaque psoriasis were administered GR-MD-02 every two weeks for 24 weeks. In May 2016, we reported positive results on the first four patients after 12 weeks of therapy. Based on these results, we modified the trial to include 24 weeks of therapy. In August 2016, we reported on four patients after 24 weeks of therapy and one patient after 12 weeks of therapy. The four patients who received 24 weeks of therapy experienced an average of 48% improvement in their plaque psoriasis. At this time, the average response in all five patients remains at 50% with one patient having an 82% improvement. However, there are existing drugs on the market in this disease that produce 75% and higher improvements in 60-90% of patients. While we are encouraged that this study has demonstrated clinically

meaningful results in a human disease with GR-MD-02, the next steps would entail a controlled, does-ranging clinical trial which we do not expect to conduct absent a strategic partnership.

We believe the mechanism of action for GR-MD-02 is based upon interaction with, and inhibition of, galectin proteins, particularly galectin-3, which are expressed at high levels in certain pathological states including inflammation, fibrosis and cancer. While GR-MD-02 is capable of binding to multiple galectin proteins, we believe that it has the greatest affinity for galectin-3, the most prominent galectin implicated in pathological processes. Blocking galectin in cancer and liver fibrosis has specific salutary effects on the disease process, as discussed below.

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Results of Operations

Three and Six Months Ended June 30, 2018 Compared to Three and Six Months Ended June 30, 2017

Research and Development Expense.

	Three Months Ended June 30,		Six Months Ended June 30,		2018 as Compared to 2017 Three Months		2018 as Compared to 2017 Six Months	
	2018	2017	2018	2017	$ Change	% Change	$ Change	% Change
	(In thousands, except %)							
Research and development	$ 1,476	$ 3,444	$ 3,774	$ 7,216	$ (1,968)	(57%)	$ (3,442)	(48%)

We generally categorize research and development expenses as either direct external expenses, comprised of amounts paid to third party vendors for services, or all other research and development expenses, comprised of employee payroll and general overhead allocable to research and development. We consider a clinical program to have begun upon acceptance by the FDA, or similar agency outside of the United States, to commence a clinical trial in humans, at which time we begin tracking expenditures by the product candidate. Clinical program expenses comprise payments to vendors related to preparation for, and conduct of, all phases of the clinical trial, including costs for drug manufacture, patient dosing and monitoring, data collection and management, oversight of the trials and reports of results. Pre-clinical expenses comprise all research and development amounts incurred before human trials begin, including payments to vendors for services related to product experiments and discovery, toxicology, pharmacology, metabolism and efficacy studies, as well as manufacturing process development for a drug candidate.

Our research and development expenses were as follows:

	Three Months Ended June 30,		Six Months Ended June 30,	
	2018	2017	2018	2017
	(in thousands)			
Direct external expenses:				
Clinical programs	$ 331	$ 2,865	$ 1,617	$ 6,027
Pre-clinical activities	50	35	116	66
All other research and development expenses	1,095	544	2,041	1,123
	$ 1,476	$ 3,444	$ 3,774	$ 7,216

Clinical programs expenses decreased primarily due to costs related to our Phase 2 clinical trials winding down late in 2017. Other research and development expense increased primarily due to increases in non-cash stock-based compensation expense of approximately $495,000 and $863,000 in the three and six months ended June 30, 2018 compared to the same periods in 2017.

Both the time required and costs we may incur in order to commercialize a drug candidate that would result in material net cash inflow are subject to numerous variables, and therefore we are unable at this stage of our development to forecast useful estimates. Variables that make estimates difficult include the number of clinical trials we may undertake, the number of patients needed to participate in the clinical trial, patient recruitment uncertainties,

trial results as to the safety and efficacy of our product, and uncertainties as to the regulatory agency response to our trial data prior to receipt of marketing approval. Moreover, the FDA or other regulatory agencies may suspend clinical trials if we or an agency believes patients in the trial are subject to unacceptable risks or find deficiencies in the conduct of the clinical trial. Delays or rejections may also occur if governmental regulation or policy changes during our clinical trials or in the course of review of our clinical data. Due to these uncertainties, accurate and meaningful estimates of the ultimate cost to bring a product to market, the timing of costs and completion of our program and the period during which material net cash inflows will commence are unavailable at this time.

General and Administrative Expense.

					2018 as Compared to 2017			
	Three Months Ended June 30,		Six Months Ended June 30,		Three Months		Six Months	
	2018	2017	2018	2017	$ Change	% Change	$ Change	% Change
	(In thousands, except %)							
General and administrative	$ 2,283	$ 1,070	$ 4,163	$ 2,244	$ 1,213	113%	$ 1,919	86%

General and administrative expenses consist primarily of salaries including stock-based compensation, legal and accounting fees, insurance, investor relations, business development and other office related expenses. The primary reasons for the increase in general and administrative expenses for the three-months ended June 30, 2018 as compared to the same period in 2017 is due to an

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increase non-cash, stock-based compensation expense of $679,000 and as increase in business development/investor relations expenses of approximately $191,000. The primary reasons for the increase in general and administrative expenses for the six-months ended June 30, 2018 as compared to the same period in 2017 is due to an increase non-cash, stock-based compensation expense of $1,168,000 and as increase in business development/investor relations expenses of approximately $333,000.

Liquidity and Capital Resources

Since our inception on July 10, 2000, we have financed our operations from proceeds of public and private offerings of debt and equity. As of June 30, 2018, we raised a net total of $146.1 million from these offerings. We have operated at a loss since our inception and have had no significant revenues. We anticipate that losses will continue for the foreseeable future. At June 30, 2018, the Company had $10.5 million of unrestricted cash and cash equivalents available to fund future operations. The Company believes there is sufficient cash, including availability of the line of credit, to fund currently planned operations at least through June 30, 2019. Our ability to fund operations after our current cash resources are exhausted depends on our ability to obtain additional financing or achieve profitable operations, as to which no assurances can be given. Accordingly, based on the forecasts and estimates underlying our current operating plan, the financial statements do not currently include any adjustments that might be necessary if we are unable to continue as a going concern.

Net cash used in operations decreased by $2,270,000 to $6,595,000 for the six months ended June 30, 2018, as compared to $8,865,000 for the six months ended June 30, 2017. Cash operating expenses decreased principally due to the winding down of Phase 2 research and development activities related to our clinical trial activity with GR-MD-02.

Net cash provided by financing activities the six months ended June 30, 2018, of $14,039,000 represents proceeds from the exercise of common stock warrants and options and sale of common stock. Net cash provided by financing activities the three months ended June 30, 2017, of $2,630,000 represents net proceeds from the sale of common stock and warrants.

Other.

We have engaged outside vendors for certain services associated with our clinical trials. These services are generally available from several providers and, accordingly, our arrangements are typically cancellable on 30 days notice.

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Off-Balance Sheet Arrangements

We have not created, and are not a party to, any special-purpose or off-balance sheet entities for the purpose of raising capital, incurring debt or operating parts of our business that are not consolidated into our financial statements. We do not have any arrangements or relationships with entities that are not consolidated into our financial statements that are reasonably likely to materially affect our liquidity or the availability of capital resources.

Application of Critical Accounting Policies and Estimates

The preparation of condensed consolidated financial statements requires us to make estimates and judgments that affect the reported amounts of assets, liabilities, expenses, and related disclosure of contingent assets and liabilities. On an ongoing basis, we evaluate our estimates, including those related to intangible assets, accrued expenses, stock-based compensation, contingencies and litigation. We base our estimates on historical experience, terms of existing contracts, our observance of trends in the industry, information available from other outside sources and on various other factors that we believe to be appropriate under the circumstances. Actual results may differ from these estimates under different assumptions or conditions.

Critical accounting policies are those policies that affect our more significant judgments and estimates used in preparation of our consolidated financial statements. We believe our critical accounting policies include our policies regarding stock-based compensation, accrued expenses and income taxes. For a more detailed discussion of our critical accounting policies, please refer to our 2017 Annual Report on Form 10-K.

Item 3. Quantitative and Qualitative Disclosures about Market Risk

Market risk represents the risk of loss that may impact our financial position, operating results or cash flows due to changes in the U.S. interest rates. The primary objective of our investment activities is to preserve cash until it is required to fund operations. To minimize risk, we maintain our portfolio of cash and cash equivalents in operating bank accounts and money market funds. Since our investments are short-term in duration, we believe that we are not subject to any material market risk exposure.

Item 4. Controls and Procedures

Our management, with the participation of the Chief Executive Officer and Chief Financial Officer, evaluated the effectiveness of our disclosure controls and procedures (as defined in Rule 13a-15(e) promulgated under the Securities Exchange Act of 1934) and concluded that, as of June 30, 2018, our disclosure controls and procedures were effective at a reasonable assurance level. During the quarter ended June 30, 2018, no change in our internal control over financial reporting has materially affected, or is reasonably likely to materially affect, our internal control over financial reporting.

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PART II OTHER INFORMATION

Item 1. Legal Proceedings

None except as discussed in Note 9 to our condensed consolidated financial statements included in this report.

Item 1A. Risk Factors

The information set forth in this report should be read in conjunction with the risk factors set forth in Item 1A, Risk Factors, of Part I of our Annual Report on Form 10-K for the year ended December 31, 2017, which could materially impact our business, financial condition or future results.

Item 2. Unregistered Sales of Equity Securities and Use of Proceeds

None

Item 3. Defaults Upon Senior Securities

None

Item 4. Mine Safety Disclosures

Not Applicable

Item 5. Other Information

Not Applicable

Item 6. Exhibits

Exhibit Number	Description of Document	Note Reference
31.1*	Certification Pursuant to Rule 13a-14(a) of the Securities Exchange Act of 1934	
31.2*	Certification Pursuant to Rule 13a-14(a) of the Securities Exchange Act of 1934	
32.1**	Certification Pursuant to 18 U.S.C. Section 1350, as Adopted Pursuant to Section 906 of the Sarbanes-Oxley Act of 2002	
32.2**	Certification Pursuant to 18 U.S.C. Section 1350, as Adopted Pursuant to Section 906 of the Sarbanes-Oxley Act of 2002	
101.INS	XBRL Instance Document*	
101.SCH	XBRL Taxonomy Extension Schema Document*	
101.CAL	XBRL Taxonomy Calculation Linkbase Document*	

101.DEF	XBRL Taxonomy Extension Definition Linkbase Document*
101.LAB	XBRL Taxonomy Label Linkbase Document*
101.PRE	XBRL Taxonomy Presentation Linkbase Document*

* Filed herewith.
** Furnished herewith and not filed for purposes of Section 18 of the Securities Exchange Act of 1934, as amended.

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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, thereunto duly authorized, on August 14, 2018.

GALECTIN THERAPEUTICS INC.

By: /s/ Harold H. Shlevin, Ph.D.
Name: Harold H. Shlevin, Ph. D
Title: Chief Executive Officer and President
(principal executive officer)

/s/ Jack W. Callicutt
Name: Jack W. Callicutt
Title: Chief Financial Officer

(principal financial and accounting officer)