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

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**FORM 10-Q**

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**Quarterly report pursuant to Section 13 or 15(d) of the Securities Exchange Act of 1934**

For the quarterly period ended June 30, 2017

**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

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**GALECTIN THERAPEUTICS INC.**

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Nevada  
(State or other jurisdiction  
of incorporation)

4960 Peachtree Industrial Blvd., Suite 240, Norcross, GA  
(Address of Principal Executive Offices)

04-3562325  
(I.R.S. Employer  
Identification No.)

30071  
(Zip Code)

(678) 620-3186  
(Registrant's Telephone Number, Including Area Code)

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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	<input type="checkbox"/>	Accelerated Filer	<input type="checkbox"/>
Non-Accelerated Filer	<input type="checkbox"/> (Do not check if a smaller reporting company)	Smaller reporting company	<input checked="" type="checkbox"/>
Emerging growth company	<input type="checkbox"/>		

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 7, 2017 was 35,051,705.

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FOR THE QUARTER ENDED JUNE 30, 2017**

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CONDENSED CONSOLIDATED BALANCE SHEETS (UNAUDITED)**

	June 30, 2017	December 31, 2016
	(in thousands)	
<b>ASSETS</b>		
Current assets:		
Cash and cash equivalents	\$ 9,127	\$ 15,362
Prepaid expenses and other current assets	202	432
Total current assets	<u>9,329</u>	<u>26,400</u>
Intangible assets, net	—	1
Total assets	<u>\$ 9,329</u>	<u>\$ 15,795</u>
<b>LIABILITIES, REDEEMABLE CONVERTIBLE PREFERRED STOCK AND STOCKHOLDERS' EQUITY</b>		
Current liabilities:		
Accounts payable	\$ 399	\$ 910
Accrued expenses	3,045	2,802
Accrued dividends payable	68	68
Total current liabilities	<u>3,512</u>	<u>3,780</u>
Total liabilities	<u>3,512</u>	<u>3,780</u>
Commitments and contingencies (Note 8)		
Series C super dividend convertible preferred stock; 1,000 shares authorized, 176 shares issued and outstanding at June 30, 2017 and December 31, 2016, redemption value: \$7,209,000, liquidation value: \$1,760,000 at June 30, 2017		
	1,723	1,723
Stockholders' equity:		
Undesignated stock, \$0.01 par value; 20,000,000 shares authorized, 20,000,000 and 14,001,000 designated at June 30, 2017 and December 31, 2016, respectively	—	—
Series A 12% convertible preferred stock; 1,742,500 shares authorized, 1,377,500 issued and outstanding at June 30, 2017 and December 31, 2016, liquidation value \$1,377,500 at June 30, 2017	557	557
Series B-1 12% convertible preferred stock; 900,000 shares authorized, issued and outstanding at June 30, 2017 and December 31, 2016, liquidation value \$1,800,000 at June 30, 2017	1,761	1,761
Series B-2 12% convertible preferred stock; 2,100,000 shares authorized, issued and outstanding at June 30, 2017 and December 31, 2016, liquidation value \$4,200,000 at June 30, 2017	3,697	3,697
Series B-3 8% convertible preferred stock; 2,508,000 shares authorized, 2,508,000 issued and outstanding at June 30, 2017 and December 31, 2016, liquidation value \$2,508,000 at June 30, 2017	1,224	1,224
Common stock, \$0.001 par value; 50,000,000 shares authorized at June 30, 2017 and December 31, 2016, 34,965,789 and 32,912,942 issued and outstanding at June 30, 2017 and December 31, 2016, respectively	35	33
Additional paid-in capital	170,539	166,721
Retained deficit	<u>(173,719)</u>	<u>(163,701)</u>
Total stockholders' equity	<u>4,094</u>	<u>10,292</u>
Total liabilities, redeemable convertible preferred stock and stockholders' equity	<u>\$ 9,329</u>	<u>\$ 15,795</u>

See notes to unaudited condensed consolidated financial statements.

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CONDENSED CONSOLIDATED STATEMENTS OF OPERATIONS (UNAUDITED)**

	Three Months Ended June 30,		Six Months Ended June 30,	
	2017	2016	2017	2016
	(in thousands, except per share data)		(in thousands, except per share data)	
Operating expenses:				
Research and development	\$ 3,444	\$ 4,226	\$ 7,216	\$ 8,603
General and administrative	1,070	1,305	2,244	3,742
Total operating expenses	4,514	5,531	9,460	12,345
Total operating loss	(4,514)	(5,531)	(9,460)	(12,345)
Other income (expense):				
Interest income	6	12	15	26
Total other income (expense)	6	12	15	26
Net loss	\$ (4,508)	\$ (5,519)	\$ (9,445)	\$ (12,319)
Preferred stock dividends	(301)	(250)	(573)	(403)
Preferred stock accretion	—	(58)	—	(115)
Net loss applicable to common stockholders	\$ (4,809)	\$ (5,827)	\$ (10,018)	\$ (12,837)
Net loss per common share — basic and diluted	\$ (0.14)	\$ (0.20)	\$ (0.29)	\$ (0.44)
Weighted average common shares outstanding — basic and diluted	34,692	29,023	34,312	29,001

See notes to unaudited condensed consolidated financial statements.

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CONDENSED CONSOLIDATED STATEMENTS OF CASH FLOWS (UNAUDITED)**

	<b>Six Months Ended</b>	
	<b>June 30,</b>	
	<b>2017</b>	<b>2016</b>
	<b>(in thousands)</b>	
<b>CASH FLOWS FROM OPERATING ACTIVITIES:</b>		
Net loss	\$ (9,445)	\$(12,319)
Adjustments to reconcile net loss to net cash used in operating activities:		
Depreciation and amortization	1	3
Stock-based compensation expense	599	1,625
Issuance of common stock for services	18	—
Changes in operating assets and liabilities:		
Prepaid expenses and other assets	230	315
Accounts payable and accrued expenses	(268)	2,276
Net cash used in operating activities	<u>(8,865)</u>	<u>(8,100)</u>
<b>CASH FLOWS FROM FINANCING ACTIVITIES:</b>		
Net proceeds from issuance of common stock and warrants	2,630	257
Net cash provided by financing activities	<u>2,630</u>	<u>—</u>
NET (DECREASE) INCREASE IN CASH AND CASH EQUIVALENTS	(6,235)	(7,843)
CASH AND CASH EQUIVALENTS, BEGINNING OF PERIOD	15,362	25,846
CASH AND CASH EQUIVALENTS, END OF PERIOD	<u>\$ 9,127</u>	<u>\$ 18,003</u>
<b>NONCASH FINANCING ACTIVITIES:</b>		
Payment of preferred stock dividends in common stock	\$ 573	\$ 403

See notes to unaudited condensed consolidated financial statements.

**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 function. 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, 2017 and the results of its operations for the three and six months ended June 30, 2017 and 2016 and its cash flows for the six months ended June 30, 2017 and 2016. 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, 2016 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 which 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, 2016.

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, 2017, the Company had \$9.1 million of unrestricted cash and cash equivalents available to fund future operations. The Company believes that with the cash on hand at June 30, 2017, there is sufficient cash to fund currently planned operations through January 2018. 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, 2017	December 31, 2016
	(in thousands)	
Legal and accounting fees	\$ 68	\$ 14
Accrued compensation	284	614
Accrued research and development costs and other	2,693	2,174
Total	<u>\$3,045</u>	<u>\$ 2,802</u>

**3. 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,	
	2017	2016	2017	2016
Research and development	\$ 132	\$ 179	\$ 301	\$ 433
General and administrative	137	223	298	1,192
Total stock-based compensation expense	<u>\$ 269</u>	<u>\$ 402</u>	<u>\$ 599</u>	<u>\$ 1,625</u>

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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, 2016 through June 30, 2017:

	Shares	Weighted Average Exercise Price
Outstanding, December 31, 2016	4,656,888	\$ 4.30
Granted	—	—
Exercised	—	—
Options forfeited/cancelled	—	—
Outstanding, June 30, 2017	<u>4,656,888</u>	\$ 4.30

As of June 30, 2017, there was \$595,000 of unrecognized compensation related to 1,158,214 unvested options, which is expected to be recognized over a weighted-average period of approximately 0.73 years. The weighted-average grant date fair value for options granted during the six months ended June 30, 2016 was \$1.05. The Company granted 277,500 stock options during the six months ended June 30, 2016, of which 69,375 options vested upon grant with the remaining 208,125 options vesting over 3 years. Approximately \$73,000 of non-cash, stock-based compensation expense was recorded during the six months ended June 30, 2016 related to the options granted during the quarter that were 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, 2017	Six Months Ended June 30, 2016
Risk-free interest rate	—	1.7%
Expected life of the options	—	6.0 years
Expected volatility of the underlying stock	—	94%
Expected dividend rate	—	0%

The following table summarizes the restricted stock grant activity in the Company's equity incentive plans from December 31, 2016 through June 30, 2017:

	Shares
Outstanding, December 31, 2016	754,605
Granted	—
Exercised	—
Options forfeited/cancelled	—
Outstanding, June 30, 2017	<u>754,605</u>

On March 12, 2015, the Company granted 81,352 shares of restricted stock to non-employee directors as a component of their compensation. A total of 77,784 shares were issued to seven directors representing non-cash compensation cost of \$280,000 which was recognized on a straight-line basis from the grant date through December 15, 2016, when the restricted shares vested in full. A total of 3,568 shares were issued to two directors, who were not nominated for reelection, representing non-cash compensation cost of \$12,845 that was recognized on a straight-line basis from the grant date through May 21, 2016, when the restricted shares vested in full.

#### 4. Common Stock Warrants

The following table summarizes the common stock warrant activity from December 31, 2016 through June 30, 2017:

	Shares	Weighted Average Exercise Price
Outstanding, December 31, 2016	13,488,296	\$ 3.44
Granted	78,054	5.00
Exercised	—	—
Forfeited/cancelled	(1,317,161)	5.63
Outstanding, June 30, 2017	<u>12,249,189</u>	\$ 3.22

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### 5. Fair Value of Financial Instruments

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, 2017 or December 31, 2016.

### 6. 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, 2017 (shares)	June 30, 2016 (shares)
Warrants to purchase shares of common stock	12,249,189	8,908,586
Options to purchase shares of common stock	4,656,888	3,499,638
Shares of common stock issuable upon conversion of preferred stock	4,312,282	2,522,936
Unvested shares of restricted common stock	—	337,935
	<u>21,218,359</u>	<u>15,269,095</u>

### 7. 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. In three months ended March 31, 2017, the Company issued 1,496,797 shares of common stock for net proceeds of approximately \$1,946,000 under the 2014 At Market Agreement.

#### *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. In three months ended June 30, 2017, the Company issued 198,170 shares of common stock for net proceeds of approximately \$484,000 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

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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 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 2017, 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. Through June 30, 2017, the Company has issued 12,781 shares of common stock pursuant to this agreement and the value of such shares has been recorded as research and development expense.

Through June 30, 2017, the Company has issued a total of 242,731 shares of common stock for dividends on Series A, Series B and Series C Preferred Stock for 2017.

## **8. Commitments and Contingencies**

### ***Shareholder Class Actions and Derivative Lawsuits***

On August 1 and 25, 2014, persons claiming to be Galectin shareholders filed putative shareholder derivative complaints in the Nevada District Court, seeking recovery on behalf of the Company against certain of the Company's directors and officers. On September 10, 2014, the Nevada District Court entered an order consolidating the two cases, relieving the defendants of any obligation to respond to the initial complaints, and providing that defendants may respond to a consolidated complaint to be filed by the plaintiffs. On January 5, 2015, the Nevada District Court granted Defendants' motion to transfer the consolidated putative derivative litigation to the United States District Court for the Northern District of Georgia (hereinafter referred to as the "Georgia Federal Derivative Action."). The plaintiffs filed a consolidated complaint on February 27, 2015. On April 6, 2015, the Company and defendants filed motions to dismiss the consolidated complaint. Rather than respond to those motions, the plaintiffs sought and obtained leave to file an amended complaint. Plaintiffs filed their amended complaint (the "Complaint") on May 26, 2015. The Complaint alleges that certain of the Company's directors and officers (the "Derivative Action Individual Defendants") breached their fiduciary duties to the Company's shareholders by causing or permitting the Company to make allegedly false and misleading public statements concerning the Company's financial and business prospects. The Complaint also alleges that the Derivative Action Individual Defendants violated the federal securities laws by allegedly making false or misleading statements of material fact in the Company's proxy filings, committed waste of corporate assets, were unjustly enriched, and that certain defendants breached their fiduciary duties through allegedly improper sales of Galectin stock. In addition, the Complaint alleges that the Derivative Action Individual Defendants and one of the Company's shareholders aided and abetted the alleged breaches of fiduciary duties. The Complaint seeks unspecified monetary damages on behalf of the Company, corporate governance reforms, disgorgement of profits, benefits and compensation by the defendants, costs, and attorneys' and experts' fees. The Company and defendants filed motions to dismiss the Complaint on July 8, 2015. On December 30, 2015, the United States District Court for the Northern District of Georgia dismissed the Georgia Federal Derivative Action with prejudice and entered a final judgment in favor of the defendants. Plaintiffs filed a notice of appeal seeking review of the dismissal order and final judgment. On July 7, 2016, the United States Court of Appeals for the Eleventh Circuit dismissed the appeal as the Plaintiffs failed to timely file their appeal brief. In September 2016, the Board received a demand letter from one of the plaintiffs in the Georgia Federal Derivative Action. The demand letter, among other things, requests that the Board investigate the conduct alleged in the Complaint and implement certain remedial measures purportedly designed to address the alleged conduct. It is expected that the Board will consider the demand letter in due course and in light of the related pending shareholder litigation described herein.

On August 29, 2014, another alleged Galectin shareholder filed a putative shareholder derivative complaint in state court in Las Vegas, Nevada, seeking recovery on behalf of the Company against the same Galectin directors and officers who are named as defendants in the derivative litigation pending in the Georgia Federal Derivative Action. The plaintiff in the Nevada action subsequently filed first and second amended complaints. The second amended complaint alleges claims for breach of fiduciary duties, unjust enrichment, and waste of corporate assets, based on allegations that are substantially similar to those asserted in the Georgia Federal Derivative Action (except that the Nevada action does not allege violations of the federal securities laws and does not assert any claim against the Galectin shareholder named as a defendant in the Georgia Federal Derivative Action), and seeks unspecified monetary damages on behalf of the Company, corporate governance reforms, disgorgement of profits, benefits and compensation by the defendants, costs, and attorneys' and experts' fees. The Company and defendants filed motions to dismiss the second amended complaint on April 22, 2015. On April 29, 2015, the plaintiffs in the Georgia Federal Derivative Action (the "Intervenor Plaintiffs") filed a motion to intervene in the Nevada action which, among other things, raised questions regarding the Nevada plaintiff's standing. Thereafter, the Nevada plaintiff filed a motion to join additional plaintiffs. At a hearing held on June 11, 2015, the Nevada court: (i) granted the Intervenor Plaintiffs' motion to intervene; (ii) directed the Intervenor Plaintiffs to file a complaint in intervention; (iii) directed the Nevada plaintiff to file a motion for leave to file a further amended complaint to add additional plaintiffs; (iv) stated

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that the defendants' motions to dismiss the second amended complaint were denied "at this point;" (v) ordered the Nevada action stayed until December 11, 2015; and (vi) directed the parties to submit a status report on December 11, 2015, updating the court on the progress and status of the Georgia Federal Derivative Action. On July 9, 2015, pursuant to the Nevada State Court's instruction, the Intervenor Plaintiffs filed a complaint-in-intervention in Nevada State Court, asserting similar claims to the ones they alleged in the Georgia Federal Derivative Action described above. On December 11, 2015, further to the Nevada State Court's instruction, the parties submitted status reports detailing the status of the Georgia Federal Derivative Action. On January 5, 2016, the Nevada State Court held a status conference during which the dismissal of the Georgia Federal Derivative Action was discussed. Subsequent to that conference, on January 19, 2016, the defendants filed a motion to dismiss the Nevada State Court litigation based on the dismissal of the similar Georgia Federal Derivative Action, among other grounds. Following full briefing and a hearing on March 3, 2016, the Nevada State Court granted dismissal of the Nevada State Court litigation. Notice of Entry of the Nevada State Court's order dismissing the Nevada State Court litigation was docketed on June 21, 2016. The Nevada plaintiff and Intervenor Plaintiffs ("Appellants") filed notices of appeal seeking review of the Nevada State Court's order and judgment dismissing the claims. The appeal is now fully briefed and awaiting a decision from the Nevada Supreme Court.

Estimating an amount or range of possible losses resulting from litigation proceedings is inherently difficult and requires an extensive degree of judgment, particularly where the matters involve indeterminate claims for monetary damages, are in the early stages of the proceedings, and are subject to appeal. In addition, because most legal proceedings are resolved over extended periods of time, potential losses are subject to change due to, among other things, new developments, changes in legal strategy, the outcome of intermediate procedural and substantive rulings and other parties' settlement posture and their evaluation of the strength or weakness of their case against us. For these reasons, we are currently unable to predict the ultimate timing or outcome of, or reasonably estimate the possible losses or a range of possible losses resulting from, the matters described above. Based on information currently available, the Company does not believe that any reasonably possible losses arising from currently pending legal matters will be material to the Company's results of operations or financial condition. However, in light of the inherent uncertainties involved in such matters, an adverse outcome in one or more of these matters could materially and adversely affect the Company's financial condition, results of operations or cash flows in any particular reporting period.

### ***Other Legal Proceedings***

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 pending legal proceedings except as noted above.

### **9. 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 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. As a result, the Company contributed the \$73,000 needed for the fourth quarter of 2014 expenses of the LLC. The Company contributed \$659,000 and \$687,000 for the LLC expenses in 2016 and 2015, respectively, and SBH contributed \$50,000 in 2016. The Company contributed \$45,000 and \$29,000 during the quarters ended March 31, 2017 and June 30, 2017, respectively. As of June 30, 2017, the Company's ownership percentage in the LLC was 81%. 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.

## 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,” “could” or “may.” All 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 December 31, 2017; 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, including shareholder class actions and derivative lawsuits filed,
- 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

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, certain severe skin diseases, 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

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using this inhibitor to treat advanced liver fibrosis and liver cirrhosis in NASH (non-alcoholic steatohepatitis) patients. We have completed two Phase 1 clinical studies, one Phase 2 clinical study in NASH patients with advanced fibrosis (NASH-FX) and have one ongoing Phase 2 clinical 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 extra-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 a discovery program aimed at the targeted development of small molecules (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 compounds. We are pursuing a development pathway to clinical enhancement and commercialization for our lead compounds in immune enhancement for cancer therapy and certain severe skin diseases including moderate to severe plaque psoriasis and severe atopic dermatitis. 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 will report 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 show that mice lacking 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.

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, certain severe skin diseases, 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 on 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. Previously in clinical development for cancer indications, this compound continues to be explored in preclinical studies.

Our product pipeline is shown below:

<b>Indication</b>	<b>Drug</b>	<b>Status</b>
<b>Fibrosis</b> 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 CX trial, was designed for patients with cirrhosis, and 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 and the NASH CX trial top line data is expected in December 2017.
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 and GM-CT-01	In pre-clinical development

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Indication	Drug	Status
<b>Cancer Immunotherapy</b>		
Melanoma	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 will continue.
<b>Psoriasis</b>		
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. Studies are now completed.

*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 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 and the human pharmacokinetic data defined a drug dose for use in the planned Phase 2 trials. 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.

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 U.S. Food and Drug Administration (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 Galectin 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 first clinical trial is the Phase 2b NASH-CX study for patients with NASH with 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 2 trial to evaluate the safety and efficacy of GR-MD-02 for the treatment of liver fibrosis and resultant portal hypertension in patients with 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 are receiving an infusion every other week for one year, total of 26 infusions, and will be evaluated to determine the change in HVPG as compared with placebo. HVPG will be correlated with secondary endpoints of fibrosis on liver biopsy as well as with measurement of liver stiffness (FibroScan<sup>(R)</sup>) and assessment of liver metabolism (<sup>13</sup>C-methacetin breath test, Exalenz), which are non-invasive measures of the liver that may be used in future studies. A late-breaking abstract on screening data results correlating clinically significant portal hypertension and methacetin breath test results was presented at The International Liver Congress™ in Amsterdam on April 20, 2017. The DSMB concluded its third and final review of the safety and conduct of the in-life phase of our NASH-CX trial and indicated its concurrence with continuation of the trial and the ‘clean’ safety profile. As of the time of their evaluation, therapy had been completed in 68% of subjects in the NASH-CX trial. As of June 30, 2017, 108 patients (71%) have completed all 52 weeks of infusions and 155 patients have completed 26 weeks of infusions in this Phase 2b clinical trial. Over 97% of the doses have already been administered. The dropout rate remains well below expectations, which may increase the power of the trial. We currently expect approximately 151 subjects will complete the trial

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by Fall 2017. This trial was designed, and is being conducted, with a primary endpoint that the U.S. Food and Drug Administration views may be a surrogate for outcomes for registration trials in this patient population. After completion of study endpoints and initial analysis of data, the Company remains on track to report top line data from the NASH-CX trial in early December 2017.

The second trial, our 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 shorter, 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<sup>®</sup>, 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. After analysis of the data, we do not believe that a four-month treatment period was likely long enough in the NASH population to show efficacy results. 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 and in moderate-to-severe atopic dermatitis patients, suggesting the compound has activity in an immune-mediated inflammatory human disease that can occur in association with NASH and in which galectin-3 is believed to play a role. 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 variety of animal models.

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.

*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<sup>®</sup> (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. This Phase 1b clinical trial that combines GR-MD-02 with pembrolizumab (KEYTRUDA<sup>®</sup>) continues to enroll patients and recently completed the second cohort, which used 4 mg/kg GR-MD-02. The third cohort, which will start 85 days following the final patient enrollment in the second cohort, will enroll 10 patients at a dose of 8 mg/kg GR-MD-02. There will likely be additional data reported in early 2018. The study is ongoing and progression to further development will be based on response rate as compared to historical response rates to pembrolizumab alone.

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### *Severe skin diseases.*

*Psoriasis.* 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. The results of this Phase 2a clinical trial was presented at the Maui Derm for Dermatologists meeting in March 2017.

*Atopic Dermatitis.* Atopic Dermatitis (AD) is a chronic pruritic (itching), immune-mediated, inflammatory skin disease that for some adult patients can be severe and debilitating. There is an important unmet medical need in adults with severe disease who are not adequately treated with topical medicines. Our findings in psoriasis led to an interest in exploring the potential utility of the compound in treatment of AD. A Phase 2a open label trial was initiated in 3 adult patients who were treated with GR-MD-02 at 8 mg/Kg every other week for 12 weeks, and dosage could be increased to 12 mg/Kg for weeks 12-24 if an incomplete response was observed. The response was objectively evaluated using two validated scores: the EASI (eczema area and severity index) and SCORAD (severity scoring of atopic dermatitis index). All three patients showed clinical responses as determined by reduction of EASI and SCORAD during the study, but no subject achieved a primary endpoint. Upon completion of the 24-week treatment phase subject 1 met both secondary endpoints of SCORAD-50 and EASI-50, and subject 2 met one secondary endpoint (EASI-50). Subject 3 approached a secondary endpoint with an EASI reduction of 49% at week 24. There were no drug related adverse events during this trial. These initial findings are believed to suggest a clinically relevant effect. Additionally, the 12 mg/Kg dosage was administered safely in this patient population.

We believe the mechanism of action for GR-MD-02 in moderate to severe skin diseases 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, skin diseases 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.

## **Results of Operations**

### **Three and Six Months Ended June 30, 2017 Compared to Three Months Ended June 30, 2016**

#### *Research and Development Expense.*

	Three Months Ended June 30,		Six Months Ended June 30,		2017 as Compared to 2016			
	2017	2016	2017	2016	Three Months		Six Months	
					\$ Change	% Change	\$ Change	% Change
Research and development	\$3,444	\$ 4,226	\$7,216	\$8,603	\$ (782)	(19%)	\$(1,387)	(16%)

(In thousands, except %)

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.

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We have two product candidates, GR-MD-02 and GM-CT-01; however only GR-MD-02 is in active development. We filed for an IND for GR-MD-02 in January 2013 and in February 2013 we entered into an agreement with CTI to conduct a Phase 1 clinical trial of GR-MD-02. In March 2013, the FDA indicated we could proceed with a Phase 1 human clinical trial of GR-MD-02, and we began enrolling patients in the third quarter of 2013. In January 2014, we completed the enrollment of the first cohort of patients in the Phase 1 trial with no serious adverse events being reported. We reported initial safety and tolerability results from the first cohort of patients on March 31, 2014. The second cohort of this Phase 1 trial began and enrollment was completed in April 2014. In July 2014, we reported the results from the second cohort of patients. Enrollment of the third cohort of Phase 1 began in July 2014 with interim results presented in November 2014 with the final report on cohort 3 presented in January 2015. The results of the Phase 1 study demonstrate that (i) GR-MD-02 was safe and well tolerated by patients with advanced NASH liver fibrosis after IV administration of four doses of 2 mg/kg, 4 mg/kg and 8mg/kg lean body weight, (ii) Pharmacokinetics revealed drug exposure in humans at the 8 mg/kg dose that was equivalent to the upper range of the targeted therapeutic dose determined from effective doses in NASH animal models, (iii) Disease Serum Marker Effect showed there was a statistically significant, dose-dependent reduction in FibroTest<sup>®</sup> scores due to a statistically significant reduction in alpha-2 macroglobulin (A2M) serum levels, and (iv) Liver Stiffness Effect, as measured by FibroScan<sup>®</sup> showed that there was a signal of reduced liver stiffness in patients receiving GR-MD-02. The reduction seen in A2M does *not* necessarily mean fibrosis got better in this short study, but does suggest changes in the fibrogenic process that might lead to an improvement in fibrosis with longer-term therapy. These Phase 1 results in NASH patients with advanced fibrosis provide a firm foundation for entry into a Phase 2 development program.

The Company held an “End of Phase 1 meeting” with the FDA and, amongst other things, received guidance on the primary endpoints for a Phase 2 trial. Our Phase 2 program in fibrotic disease consists of two separate human clinical trials. The first clinical trial is the Phase 2b NASH-CX study for patients with NASH with 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 2 trial to evaluate the safety and efficacy of GR-MD-02 for the treatment of liver fibrosis and resultant portal hypertension in patients with 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 are receiving an infusion every other week for one year, total of 26 infusions, and will be evaluated to determine the change in HVPG as compared with placebo. HVPG will be correlated with secondary endpoints of fibrosis on liver biopsy as well as with measurement of liver stiffness (FibroScan<sup>®</sup>) and assessment of liver metabolism (<sup>13</sup>C-methacetin breath test, Exalenz), which are non-invasive measures of the liver that may be used in future studies. Data readout is expected in early December 2017.

The second trial, our Phase 2a pilot trial NASH-FX for patients with NASH advanced fibrosis which explored use of three non-invasive imaging technologies, is now complete. It was a shorter, 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<sup>®</sup>, 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. After analysis of the data, we do not believe that a four-month treatment period was likely long enough in the NASH population to show efficacy results. 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 a 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, renal disease and stroke. We have further demonstrated the broad applicability of the actions of our galectin-3 inhibitor's biological effect in ameliorating fibrosis involving lung, kidney and cardiac tissues in a variety of animal models.

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.

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Additionally, during the Phase 1 clinical trial, there appeared to be a potential beneficial effect on at least one patient's moderate to severe psoriasis. 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), 4 patients with moderate to severe plaque psoriasis were administered GR-MD-02 every two weeks for 12 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. However, there are existing drugs on the market in this disease that produce 75% and higher improvements. While we are encouraged that this study has demonstrated clinically meaningful results in a human disease with GR-MD-02, we do not expect to conduct further trials in this area absent a strategic partnership.

An open label drug-drug interaction study was completed 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 enables Galectin 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.

Based on guidance from FDA and in furtherance of its understanding of the GR-MD-02 molecule, we continue to enhance its chemistry, manufacturing and control procedures on GR-MD-02 active pharmaceutical ingredient (API) as well as on the finished, sterile, pharmaceutical dosage form. Various state of the art and cutting-edge analytical technologies are being utilized, for example, to characterize and quantify the backbone vs. side-chain constituents and their quantitation, use of sophisticated linkage analysis with 2-D NMR to provide both qualitative and quantitative information on the proportion of oligomers, degree of methylation, as well as other monoclonal specific antibody techniques to map GR oligomer integrity and distribution. The Company has also characterized how the GR molecule behaves under conditions of forced degradation.

Our research and development expenses were as follows:

	Three Months Ended June 30,		Six Months Ended June 30,	
	2017	2016	2017	2016
	(in thousands)			
Direct external expenses:				
Clinical programs	\$2,865	\$ 3,366	\$6,027	\$6,772
Pre-clinical activities	35	269	66	574
All other research and development expenses	544	591	1,123	1,257
	<u>\$3,444</u>	<u>\$ 4,226</u>	<u>\$7,216</u>	<u>\$8,603</u>

Clinical programs expenses decreased primarily due to costs related to our Phase 2 clinical trials during the three and six months ended June 30, 2017 as compared to the same periods in 2016. As we continue our Phase 2 trials, we expect our clinical activities costs will increase although they may fluctuate from quarter to quarter as the trials progress. Clinical activities expenses primarily decreased due to reduced expenses associated with drug manufacturing and preparation. Pre-clinical activities decreased primarily because we have completed pre-clinical work directly related to our Phase 2 clinical trial program. Other research and development expense decreased in the six months ended June 30, 2017 compared to 2016 primarily due to a decrease in non-cash stock-based compensation expense of approximately \$135,000.

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.



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### **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, 2017, our disclosure controls and procedures were effective at a reasonable assurance level. During the quarter ended June 30, 2017, 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.

## **PART II — OTHER INFORMATION**

### **Item 1. Legal Proceedings**

None except as discussed in Note 8 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, 2016, 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**

On August 11, 2017, the Company entered into an Amendment to Employment Agreement with Jack Callicutt (the "Amendment"), amending the Employment Agreement, dated June 20, 2013, by and between the Company and Mr. Callicutt (the "Employment Agreement"). The Amendment was entered into to correct an error in the severance provision of the Employment Agreement. Pursuant to the Amendment, if Mr. Callicutt's employment with the Company is terminated by the Company "without cause," or by Mr. Callicutt for "good reason," (as such terms are defined in his agreement) he shall receive severance equal to: 3 months' base salary if such termination occurred within 12 months of July 1, 2013 (the "Commencement Date"); 6 months' base salary if such termination occurred between 12 and 18 months after the Commencement Date; or 9 months' base salary if such termination occurs after the date that is 18 months after the Commencement Date, plus, in each case, a portion of the performance bonus for the then-current year based on the number of days elapsed in the year. Prior to the Amendment, the Employment Agreement did not provide for any severance if Mr. Callicutt's employment with the Company was terminated by the Company "without cause," or by Mr. Callicutt for "good reason" after the date that was 24 months after the Commencement Date.

The foregoing description of the Amendment is not complete and is qualified in its entirety by the full text of the agreement, a copy of which is filed as Exhibit 10.1 to this report and incorporated herein by reference.

### **Item 6. Exhibits**

<u>Exhibit Number</u>	<u>Description of Document</u>	<u>Note Reference</u>
10.1*	Amendment to Employment Agreement	
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*	

\* Filed herewith.

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

**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, 2017.

GALECTIN THERAPEUTICS INC.

By: /s/ Peter G. Traber

Name: Peter G. Traber, M.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)

## AMENDMENT TO EMPLOYMENT AGREEMENT

THIS AMENDMENT TO EMPLOYMENT AGREEMENT (the "Agreement"), dated as of August 11, 2017 (the "Effective Date"), is entered into by and between **Jack W. Callicutt** (hereinafter "Executive") and **Galectin Therapeutics, Inc.**, a Nevada corporation (the "Company").

**WITNESSETH:**

**WHEREAS**, the parties herein entered into that certain Employment Agreement dated June 20, 2013 (the "Employment Agreement"), pursuant to which Executive agreed to be employed by the Company as its Chief Financial Officer on the terms and conditions as set forth therein;

**WHEREAS**, the Employment Agreement contains a certain severance payment provision in the event the Executive terminates the Employment Agreement for Good Reason or the Company terminates the Agreement without Cause (as such capitalized terms are defined in the Employment Agreement); and

**WHEREAS**, the Company and the Executive have identified an error in the Employment Agreement as relates to such severance provision and desire to correct it.

**NOW, THEREFORE**, in consideration of these premises, the promises hereinafter contained, and other good and valuable consideration, the receipt and sufficiency which are hereby acknowledged, the parties hereto agrees as follows:

1. Capitalized terms used but not otherwise defined herein shall have the meanings ascribed to them in the Employment Agreement.

2. Section 7(b) of the Employment Agreement is amended by deleting Section 7(b) in its entirety and the following is substituted therefore for all purposes whatsoever:

“(b) If either (i) Executive terminates this Agreement for Good Reason or (ii) the Company terminates this Agreement without Cause, then the Company shall pay to Executive (1) Executive’s Base Salary accrued through the date of termination, to the extent not theretofore paid, (2)(A) if such termination occurs within twelve (12) months after the Commencement Date, an amount equal to three (3) months of Executive’s Base Salary, or (B) if such termination occurs after the period specified in (A) above, but prior to the date that is eighteen (18) months after the Commencement Date, an amount equal to six (6) months of Executive’s Base Salary or (C) if such termination occurs after the period specified in (B) above, an amount equal to nine (9) months of Executive’s Base Salary, in any case payable within thirty (30) days after the date of such termination, (3) reimbursement of any unreimbursed expenses and (4) payment of a portion of the amount of the Performance Bonus equal to the maximum amount of the Performance Bonus multiplied by a fraction, (A) the numerator of which shall be the number of days

elapsed from the beginning of the calendar year in which such termination occurs and (B) the denominator of which shall be the total number of days in the calendar year in which such termination occurs (being 365 in a full year and 184 in 2013). In exchange for any such payments, Executive shall execute, within thirty (30) days following such termination, a full release of the Company and its affiliates from all obligations other than as set forth in this Section 7(b) or from any usual and customary indemnification obligations of the Company to Executive as an officer thereof, in form and substance acceptable to the Company in its sole discretion. Notwithstanding the foregoing, the Company shall not be obligated to make any payments pursuant to this Section 7(b) until it has received such release, fully executed by Executive. For avoidance of doubt, nonrenewal of this Agreement pursuant to Section 2 hereof shall not constitute a termination by the Company without Cause hereunder and shall not entitle Executive to receive any payments pursuant to this Section 7(b).”

3. Except as specifically amended by this Amendment, the Employment Agreement shall continue in full force and effect.

**IN WITNESS WHEREOF**, the parties have caused this Amendment to be duly executed as of the date first hereinabove set.

**Company:**

GALECTIN THERAPEUTICS, INC.

By: /s/ Peter G. Traber

Name: Peter G. Traber, CEO & CMO

**Executive:**

/s/ Jack W. Callicutt

Jack W. Callicutt

**Certification Pursuant to Rule 13a-14(a) of the Securities Exchange Act of 1934**

I, Peter G. Traber, certify that:

1. I have reviewed this quarterly report on Form 10-Q of Galectin Therapeutics Inc;
2. Based on my knowledge, this report does not contain any untrue statement of a material fact or omit to state a material fact necessary to make the statements made, in light of the circumstances under which such statements were made, not misleading with respect to the period covered by this report;
3. Based on my knowledge, the financial statements, and other financial information included in this report, fairly present in all material respects the financial condition, results of operations and cash flows of the registrant as of, and for, the periods presented in this report;
4. The registrant's other certifying officer and I are responsible for establishing and maintaining disclosure controls and procedures (as defined in Exchange Act Rules 13a-15(e) and 15d-15(e)) for the registrant and have:
  - (a) Designed such disclosure controls and procedures, or caused such disclosure controls and procedures to be designed under our supervision, to ensure that material information relating to the registrant, including its consolidated subsidiaries, is made known to us by others within those entities, particularly during the period in which this report is being prepared;
  - (b) Designed such internal control over financial reporting, or caused such internal control over financial reporting to be designed under our supervision, to provide reasonable assurance regarding the reliability of financial reporting and the preparation of financial statements for external purposes in accordance with generally accepted accounting principles;
  - (c) Evaluated the effectiveness of the registrant's disclosure controls and procedures and presented in this report our conclusions about the effectiveness of the disclosure controls and procedures, as of the end of the period covered by this report based on such evaluation; and
  - (d) Disclosed in this report any change in the registrant's internal control over financial reporting that occurred during the registrant's most recent fiscal quarter (the registrant's fourth fiscal quarter in the case of an annual report) that has materially affected, or is reasonably likely to materially affect, the registrant's internal control over financial reporting; and
5. The registrant's other certifying officer and I have disclosed, based on our most recent evaluation of internal control over financial reporting, to the registrant's auditors and the audit committee of the registrant's board of directors (or persons performing the equivalent functions):
  - (a) All significant deficiencies and material weaknesses in the design or operation of internal control over financial reporting which are reasonably likely to adversely affect the registrant's ability to record, process, summarize and report financial information; and
  - (b) Any fraud, whether or not material, that involves management or other employees who have a significant role in the registrant's internal control over financial reporting.

Date: August 14, 2017

/s/ Peter G. Traber

Name: Peter G. Traber, M.D.

Title: Chief Executive Officer and President  
(principal executive officer)

**Certification Pursuant to Rule 13a-14(a) of the Securities Exchange Act of 1934**

I, Jack W. Callicutt, certify that:

1. I have reviewed this quarterly report on Form 10-Q of Galectin Therapeutics Inc;
2. Based on my knowledge, this report does not contain any untrue statement of a material fact or omit to state a material fact necessary to make the statements made, in light of the circumstances under which such statements were made, not misleading with respect to the period covered by this report;
3. Based on my knowledge, the financial statements, and other financial information included in this report, fairly present in all material respects the financial condition, results of operations and cash flows of the registrant as of, and for, the periods presented in this report;
4. The registrant's other certifying officer and I are responsible for establishing and maintaining disclosure controls and procedures (as defined in Exchange Act Rules 13a-15(e) and 15d-15(e)) for the registrant and have:
  - (a) Designed such disclosure controls and procedures, or caused such disclosure controls and procedures to be designed under our supervision, to ensure that material information relating to the registrant, including its consolidated subsidiaries, is made known to us by others within those entities, particularly during the period in which this report is being prepared;
  - (b) Designed such internal control over financial reporting, or caused such internal control over financial reporting to be designed under our supervision, to provide reasonable assurance regarding the reliability of financial reporting and the preparation of financial statements for external purposes in accordance with generally accepted accounting principles;
  - (c) Evaluated the effectiveness of the registrant's disclosure controls and procedures and presented in this report our conclusions about the effectiveness of the disclosure controls and procedures, as of the end of the period covered by this report based on such evaluation; and
  - (d) Disclosed in this report any change in the registrant's internal control over financial reporting that occurred during the registrant's most recent fiscal quarter (the registrant's fourth fiscal quarter in the case of an annual report) that has materially affected, or is reasonably likely to materially affect, the registrant's internal control over financial reporting; and
5. The registrant's other certifying officer and I have disclosed, based on our most recent evaluation of internal control over financial reporting, to the registrant's auditors and the audit committee of the registrant's board of directors (or persons performing the equivalent functions):
  - (a) All significant deficiencies and material weaknesses in the design or operation of internal control over financial reporting which are reasonably likely to adversely affect the registrant's ability to record, process, summarize and report financial information; and
  - (b) Any fraud, whether or not material, that involves management or other employees who have a significant role in the registrant's internal control over financial reporting.

Date: August 14, 2017

/s/ Jack W. Callicutt  
Name: Jack W. Callicutt  
Title: Chief Financial Officer  
(principal financial and accounting officer)

**CERTIFICATION PURSUANT TO 18 U.S.C. SECTION 1350, AS ADOPTED  
PURSUANT TO SECTION 906 OF THE SARBANES-OXLEY ACT OF 2002**

In connection with the Quarterly Report of Galectin Therapeutics Inc. (the "Company") on Form 10-Q for the period ended June 30, 2017 as filed with the Securities and Exchange Commission on the date hereof (the "Report"), I, Peter G. Traber, Chief Executive Officer of the Company, certify, pursuant to 18 U.S.C. § 1350, as adopted pursuant to § 906 of the Sarbanes-Oxley Act of 2002, that, to my knowledge:

- (1) The Report fully complies with the requirements of Section 13(a) or 15(d) of the Securities Exchange Act of 1934; and
- (2) The information contained in the Report fairly presents, in all material respects, the financial condition and results of operations of the Company.

Date: August 14, 2017

/s/ Peter G. Traber

Name: Peter G. Traber, M.D.  
Title: Chief Executive Officer and President  
(principal executive officer)

A signed original of this written statement required by Section 906, or other document authenticating, acknowledging, or otherwise adopting the signature that appears in typed form within the electronic version of this written statement required by Section 906, has been provided to Galectin Therapeutics Inc. and will be retained by Galectin Therapeutics Inc. and furnished to the Securities and Exchange Commission or its staff upon request.

**CERTIFICATION PURSUANT TO 18 U.S.C. SECTION 1350, AS ADOPTED  
PURSUANT TO SECTION 906 OF THE SARBANES-OXLEY ACT OF 2002**

In connection with the Quarterly Report of Galectin Therapeutics Inc. (the "Company") on Form 10-Q for the period ended June 30, 2017 as filed with the Securities and Exchange Commission on the date hereof (the "Report"), I, Jack W. Callicutt, Chief Financial Officer of the Company, certify, pursuant to 18 U.S.C. § 1350, as adopted pursuant to § 906 of the Sarbanes-Oxley Act of 2002, that, to my knowledge:

- (1) The Report fully complies with the requirements of Section 13(a) or 15(d) of the Securities Exchange Act of 1934; and
- (2) The information contained in the Report fairly presents, in all material respects, the financial condition and results of operations of the Company.

Date: August 14, 2017

/s/ Jack W. Callicutt

Name: Jack W. Callicutt

Title: Chief Financial Officer

(principal financial and accounting officer)

A signed original of this written statement required by Section 906, or other document authenticating, acknowledging, or otherwise adopting the signature that appears in typed form within the electronic version of this written statement required by Section 906, has been provided to Galectin Therapeutics Inc. and will be retained by Galectin Therapeutics Inc. and furnished to the Securities and Exchange Commission or its staff upon request.