
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 March 31, 2009

or

☐ **TRANSITION REPORT PURSUANT TO SECTION 13 OR 15(d) OF THE SECURITIES
EXCHANGE ACT OF 1934**

For the transition period from _____ to _____

Commission File Number: 000-26658



PHARMACYCLICS, INC.

(Exact name of registrant as specified in its charter)

Delaware

(State or other jurisdiction of incorporation or organization)

94-3148201

(IRS Employer Identification Number)

995 E. Arques Avenue

Sunnyvale, California 94085-4521

(Address of principal executive offices including zip code)

(408) 774-0330

(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 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 (§232.405 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 definition of "large accelerated filer" and "accelerated filer" and "smaller reporting company" in Rule 12b-2 of the Exchange Act. (Check one):

Large accelerated filer ☐

Accelerated filer ☐

Non-accelerated filer ☐

Smaller reporting company ☒

(Do not check if a smaller reporting company)


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

As of May 5, 2009, there were 27,539,378 shares of the registrant's Common Stock, par value \$0.0001 per share, outstanding.

This quarterly report on Form 10-Q consists of 33 pages of which this is page 1. The Exhibits Index page immediately follows page 32.

PHARMACYCLICS, INC.
Form 10-Q
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PART I - FINANCIAL INFORMATION**Item 1. Financial Statements**

PHARMACYCLICS, INC.
(a development stage enterprise)
CONDENSED BALANCE SHEETS
(unaudited; in thousands)

	March 31,	June 30,
	2009	2008
ASSETS		
Current assets:		
Cash and cash equivalents	\$ 11,465	\$ 12,260
Marketable securities	--	4,495
Prepaid expenses and other current assets	633	401
Total current assets	12,098	17,156
Property and equipment, net	475	688
Other assets	290	523
	\$ 12,863	\$ 18,367
LIABILITIES AND STOCKHOLDERS' EQUITY		
Current liabilities:		
Accounts payable	\$ 2,811	\$ 1,055
Accrued liabilities	646	796
Note payable to related party	6,080	--
Total current liabilities	9,537	1,851
Deferred rent	68	71
Total liabilities	9,605	1,922
Stockholders' equity:		
Common stock	3	3
Additional paid-in capital	360,747	355,883
Accumulated other comprehensive income	--	10
Deficit accumulated during development stage.....	(357,492)	(339,451)
Total stockholders' equity	3,258	16,445
	\$ 12,863	\$ 18,367

The accompanying notes are an integral part of these condensed financial statements.

PHARMACYCLICS, INC.
(a development stage enterprise)
CONDENSED STATEMENTS OF OPERATIONS
(unaudited; in thousands, except per share data)

	Three Months Ended		Nine Months Ended		Period From
	March 31,		March 31,		Inception
	2009	2008	2009	2008	(April 19, 1991)
					through
					March 31,
					2009
Revenues:					
License and milestone revenues	\$ --	\$ --	\$ --	\$ --	\$ 7,855
Contract and grant revenues	--	--	--	--	6,154
Total revenues	<u>--</u>	<u>--</u>	<u>--</u>	<u>--</u>	<u>14,009</u>
Operating expenses:					
Research and development*	4,626	5,120	10,815	14,822	322,737
General and administrative*	1,789	2,100	7,102	5,923	83,243
Purchased in-process research and development	<u>--</u>	<u>--</u>	<u>--</u>	<u>--</u>	<u>6,647</u>
Total operating expenses	<u>6,415</u>	<u>7,220</u>	<u>17,917</u>	<u>20,745</u>	<u>412,627</u>
Loss from operations	(6,415)	(7,220)	(17,917)	(20,745)	(398,618)
Interest and other income, net	<u>(250)</u>	<u>240</u>	<u>(124)</u>	<u>1,075</u>	<u>41,126</u>
Net loss	<u>\$ (6,665)</u>	<u>\$ (6,980)</u>	<u>\$ (18,041)</u>	<u>\$ (19,670)</u>	<u>\$ (357,492)</u>
Basic and diluted net loss per share	<u>\$ (0.25)</u>	<u>\$ (0.27)</u>	<u>\$ (0.69)</u>	<u>\$ (0.76)</u>	
Shares used to compute basic and diluted net loss per share	<u>26,696</u>	<u>25,994</u>	<u>26,248</u>	<u>25,983</u>	

* Includes non-cash share-based compensation of the following:

Research and development	\$ 200	\$ 196	\$ 552	\$ 751	\$ 6,528
General and administrative	270	330	2,369	1,051	8,916

The accompanying notes are an integral part of these condensed financial statements.

PHARMACYCLICS, INC.
(a development stage enterprise)
CONDENSED STATEMENTS OF CASH FLOWS
(unaudited; in thousands)

	Nine Months Ended		Period From
	March 31,		Inception
	2009	2008	(April 19, 1991)
			through
			March 31,
			2009
Cash flows from operating activities:			
Net loss	\$ (18,041)	\$ (19,670)	\$ (357,492)
Adjustments to reconcile net loss to net cash used in operating activities:			
Depreciation and amortization	232	263	15,217
Amortization of premium/discount on marketable securities, net.....	(33)	(123)	(27)
Amortization of debt discount	250	--	250
Purchased in-process research and development	--	--	4,500
Share-based compensation expense	2,921	1,802	15,444
Loss (gain) on sale of marketable securities	(1)	(7)	50
Write-down (gain from sale) of fixed assets	(11)	--	370
Changes in assets and liabilities:			
Prepaid expenses and other assets	1	350	(923)
Accounts payable	1,756	168	2,811
Accrued liabilities	(150)	(435)	646
Deferred rent	(3)	(5)	68
Net cash used in operating activities	<u>(13,079)</u>	<u>(17,657)</u>	<u>(319,086)</u>
Cash flows from investing activities:			
Purchase of property and equipment	(19)	(47)	(12,304)
Proceeds from sale of property and equipment	11	--	123
Purchases of marketable securities	(3,179)	--	(532,725)
Proceeds from maturities and sales of marketable securities	7,698	21,994	532,702
Net cash provided by (used in) investing activities	<u>4,511</u>	<u>21,947</u>	<u>(12,204)</u>
Cash flows from financing activities:			
Issuance of common stock, net of issuance costs	1,371	48	310,289
Exercise of stock options	2	--	6,433
Proceeds from related party notes payable	6,400	--	9,400
Issuance of convertible preferred stock, net of issuance costs	--	--	20,514
Payments under capital lease obligations	--	--	(3,881)
Net cash provided by financing activities	<u>7,773</u>	<u>48</u>	<u>342,755</u>
(Decrease)/Increase in cash and cash equivalents	(795)	4,338	11,465
Cash and cash equivalents at beginning of period	12,260	11,941	--
Cash and cash equivalents at end of period	<u>\$ 11,465</u>	<u>\$ 16,279</u>	<u>\$ 11,465</u>

The accompanying notes are an integral part of these condensed financial statements.

PHARMACYCLICS, INC.
(a development stage enterprise)
NOTES TO CONDENSED FINANCIAL STATEMENTS

Note 1 – The Company and Summary of Significant Accounting Policies

Description of the Company

We are a clinical-stage biopharmaceutical company focused on developing and commercializing innovative small-molecule drugs for the treatment of immune mediated disease and cancer. Our purpose is to create a profitable company by generating income from products we develop, license and commercialize, either with one or several potential collaborators/partners or alone as may best forward the economic interest of our stakeholders. We endeavor to create novel, patentable, differentiated products that have the potential to significantly improve the standard of care in the markets we serve.

Presently, we have four product candidates in clinical development and two product candidates in pre-clinical development. It is our business strategy to establish collaborations with large pharmaceutical and biotechnology companies for the purpose of generating present and future income in exchange for adding to their product pipelines. In addition, we strive to generate collaborations that allow us to retain valuable territorial rights and simultaneously fast forward the clinical development and commercialization of our products.

We continue to evolve into a company focused on licensing and co-development activities. Most recently we signed a collaboration agreement for one of our key compounds allowing us to significantly expedite the development path outside of the U.S., while retaining U.S. rights and receiving upfront and milestone payments. This partnership is indicative of our strategy going forward.

To date, substantially all of our resources have been dedicated to the research and development of our products, and we have not generated any commercial revenues from the sale of our products. We do not expect to generate any commercial product revenues until we receive the necessary regulatory and marketing approvals and launch one of our products, if at all.

We have incurred significant operating losses since our inception in 1991, and as of March 31, 2009, had an accumulated deficit of approximately \$357.5 million. Based upon the current status of our product development plans and operational requirements, we believe that our existing cash, cash equivalents and marketable securities including the Servier upfront payment (see Note 13), will be adequate to satisfy our capital needs through at least March 31, 2010. We expect research and development expenses, as a result of on-going and future clinical trials, to consume a large portion of our existing cash resources. Changes in our research and development plans or other changes affecting our operating expenses may negatively impact actual future consumption of existing cash resources. Over time our operations may well require significant additional funding. In large part this will be due to our research and development expenses, preclinical and clinical costs. The amount of future funds needed may be impacted by the timing and structure of potential future corporate collaborations. If we do not identify collaboration partners concurrent with our expansion and development plans, we may raise additional funds through the public or private sale of securities, bank debt or otherwise. If we are unable to secure additional funds, whether through partnership collaborations or sale of securities, we will have to delay, reduce the scope of or discontinue one or more of our product development programs. Our actual capital requirements will depend on many factors, including the following:

- our ability to establish and the scope of any new partnership collaborations;
- the number, the size, the progress, the speed and the success of preclinical studies and clinical trials of our product candidates;
- the costs and timing of obtaining regulatory approvals; and
- the cost of product supply.

Our forecast of the period of time through which our financial resources will be adequate to support our operations is a forward-looking statement that involves risks and uncertainties, and actual results could vary materially. The factors described above will impact our future capital requirements and the adequacy of our available funds. We cannot be certain that additional funding will be available on terms attractive to us, or at all. Furthermore, any additional equity financing may be highly dilutive, or otherwise disadvantageous, to existing stockholders and debt financing, if available, may involve restrictive covenants. Collaborative arrangements, if necessary to raise additional funds, may require us to relinquish rights to certain of our technologies, products or marketing territories. Our failure to raise capital when needed and on acceptable terms, would require us to reduce our operating expenses and would limit our ability to respond to competitive pressures or unanticipated requirements, to develop our product candidates, and to continue operations, any of which would have a material adverse effect on our business, financial condition and results of operations.

Basis of Presentation

The accompanying interim condensed financial statements have been prepared by Pharmacyclics, Inc. (the “company” or “Pharmacyclics”), without audit, in accordance with the instructions to Form 10-Q and, therefore, do not necessarily include all information and footnotes necessary for a fair statement of its financial position, results of operations and cash flows in accordance with accounting principles generally accepted in the United States. The balance sheet at June 30, 2008 is derived from the audited balance sheet at that date which is not presented herein.

In the opinion of management, the unaudited financial information for the interim periods presented reflects all adjustments, which are only normal and recurring, necessary for a fair statement of results of operations, financial position and cash flows. These condensed financial statements should be read in conjunction with the financial statements included in the company’s Annual Report on Form 10-K for the fiscal year ended June 30, 2008. Operating results for interim periods are not necessarily indicative of operating results for an entire fiscal year.

The preparation of financial statements in conformity with accounting principles generally accepted in the United States requires management to make estimates and assumptions that affect the reported amounts and the disclosure of contingent amounts in the company’s financial statements and the accompanying notes. Actual results could differ from those estimates.

Note 2 - Basic and Diluted Net Loss Per Share

Basic net loss per share is computed using the weighted average number of common shares outstanding during the period. Diluted net loss per share is computed using the weighted average number of common and potential common shares outstanding during the period. Potential common shares consist of shares issuable upon the exercise of stock options (using the treasury stock method). Options to purchase 8,427,130 and 5,660,942 shares of common stock were outstanding at March 31, 2009 and 2008, respectively, but have been excluded from the computation of diluted net loss per share because their effect was anti-dilutive.

Note 3 - Share-Based Compensation

The company grants options to purchase its common stock pursuant to its 2004 Equity Incentive Award Plan. Options vest upon the passage of time or a combination of time and the achievement of certain performance obligations. The Compensation Committee of the Board of Directors will determine if the performance conditions have been met. Share-based compensation expense for the options with performance obligations will be recorded when the company believes that the vesting of these options is probable.

The components of share-based compensation recognized in the company’s statements of operations for the three and nine months ended March 31, 2009 and 2008 and since inception are as follows:

	Three Months Ended March 31,		Nine Months Ended March 31,		Period From Inception (April 19, 1991) through March 31,
	2009	2008	2009	2008	2009
Research and development	\$ 200,000	\$ 196,000	\$ 552,000	\$ 751,000	\$ 6,528,000
General and administrative	270,000	330,000	2,369,000	1,051,000	8,916,000
Total share-based compensation	<u>\$ 470,000</u>	<u>\$ 526,000</u>	<u>\$ 2,921,000</u>	<u>\$ 1,802,000</u>	<u>\$ 15,444,000</u>

The following table summarizes the company’s stock option activity for the nine months ended March 31, 2009:

	Number Of Shares	Weighted Average Exercise Price
Balance at June 30, 2008	5,540,544	\$ 8.10
Options granted	3,518,954	0.99
Options exercised	(1,250)	0.86
Options forfeited	(631,118)	9.39
Balance at March 31, 2009	<u>8,427,130</u>	5.04

At March 31, 2009, 672,653 shares were available for grant under the company's 2004 Equity Incentive Award Plan.

The company adopted an Employee Stock Purchase Plan (the "Purchase Plan") in August 1995. Qualified employees may elect to have a certain percentage of their salary withheld to purchase shares of the company's common stock under the Purchase Plan. The purchase price per share is equal to 85% of the fair market value of the stock on specified dates. Sales under the Purchase Plan in the nine month periods ended March 31, 2009 and 2008 were 18,207 and 26,301 shares of common stock at a price of \$1.08 and \$1.84, respectively. Shares available for future purchase under the Purchase Plan are 495,400 at March 31, 2009.

Note 4 - Comprehensive Loss

Comprehensive loss includes net loss and unrealized gains (losses) on marketable securities that are excluded from the results of operations.

The company's comprehensive loss was as follows:

	Three Months Ended March 31,		Nine Months Ended March 31,	
	2009	2008	2009	2008
Net loss	\$ (6,665,000)	\$ (6,980,000)	\$ (18,041,000)	\$ (19,670,000)
Change in net unrealized losses on available-for-sale securities	--	9,000	(10,000)	36,000
Comprehensive loss	<u>\$ (6,665,000)</u>	<u>\$ (6,971,000)</u>	<u>\$ (18,051,000)</u>	<u>\$ (19,634,000)</u>

Note 5 – Fair Value Measurements and Marketable Securities

In September 2006, the FASB issued Statement of Financial Accounting Standards No. 157, "*Fair Value Measures*" ("SFAS No. 157"). SFAS No. 157 defines fair value, establishes a framework for measuring fair value in GAAP, and expands disclosures about fair value measurements. In February 2008, the FASB issued FSP FAS 157-2 "Partial Deferral of the Effective Date of Statement 157" (FSP 157-2). FSP-2 delays the effective date of FAS 157 for non-financial assets and liabilities that are not measured or disclosed on a recurring basis to fiscal years beginning after November 15, 2008. The company adopted SFAS No. 157 on July 1, 2008. The company is currently in the process of evaluating the impact of adopting this pronouncement on its non-financial assets or liabilities.

SFAS 157 defines fair value as the exchange price that would be received for an asset or paid to transfer a liability (an exit price) in the principal or most advantageous market for the asset or liability in an orderly transaction between market participants on the measurement date. SFAS 157 also establishes a fair value hierarchy which requires an entity to maximize the use of observable inputs and minimize the use of unobservable inputs when measuring fair value. The standard describes three levels of inputs that may be used to measure fair value:

Level 1 - Quoted prices in active markets for identical assets or liabilities.

Level 2 - Observable inputs other than Level 1 prices such as quoted prices for similar assets or liabilities, quoted prices in markets that are not active, or other inputs that are observable or can be corroborated by observable market data for substantially the full term of the assets or liabilities. The Company's short-term investments primarily utilize broker quotes in markets with infrequent transactions for valuation of these securities.

Level 3 - Unobservable inputs that are supported by little or no market activity and that are significant to the fair value of the assets or liabilities.

The company utilizes the market approach to measure fair value for its financial assets and liabilities. The market approach uses prices and other relevant information generated by market transactions involving identical or comparable assets or liabilities.

The following table sets forth the company's financial assets (cash equivalents) as of March 31, 2009 and June 30, 2008:

	Fair Value as of March 31, 2009	Basis of Fair Value Measurements		
		Level 1	Level 2	Level 3
Money market funds	\$ 4,348,000	\$ 4,348,000	\$ -	\$ -
Corporate bonds	-	-	-	-
Government securities	7,117,000	-	7,117,000	-
Commercial paper	-	-	-	-
Total cash equivalents	<u>\$ 11,465,000</u>	<u>\$ 4,348,000</u>	<u>\$ 7,117,000</u>	<u>\$ -</u>

	Fair Value as of June 30, 2008	Basis of Fair Value Measurements		
		Level 1	Level 2	Level 3
Money market funds	\$ 1,038,000	\$ 1,038,000	\$ -	\$ -
Corporate bonds	3,001,000	-	3,001,000	-
Government securities	-	-	-	-
Commercial paper	12,134,000	-	12,134,000	-
Total cash equivalents and marketable securities	<u>\$ 16,173,000</u>	<u>\$ 1,038,000</u>	<u>\$ 15,135,000</u>	<u>\$ -</u>

The following is a summary of the company's available-for-sale securities at March 31, 2009 and June 30, 2008:

As of March 31, 2009	Cost Basis	Unrealized Gain	Unrealized Loss	Fair Value
Money market funds	\$ 4,348,000	\$ -	\$ -	\$ 4,348,000
Corporate bonds	-	-	-	-
Government securities	7,117,000	-	-	7,117,000
Commercial paper	-	-	-	-
	<u>11,465,000</u>	<u>-</u>	<u>-</u>	<u>11,465,000</u>
Less cash equivalents	<u>(11,465,000)</u>	<u>-</u>	<u>-</u>	<u>(11,465,000)</u>
Total marketable securities	<u>\$ -</u>	<u>\$ -</u>	<u>\$ -</u>	<u>\$ -</u>

As of June 30, 2008	Cost Basis	Unrealized Gain	Unrealized Loss	Fair Value
Money market funds	\$ 1,038,000	\$ -	\$ -	\$ 1,038,000
Corporate bonds	2,987,000	14,000	-	3,001,000
Commercial paper	12,138,000	-	(4,000)	12,134,000
	<u>16,163,000</u>	<u>14,000</u>	<u>(4,000)</u>	<u>16,173,000</u>
Less cash equivalents	<u>(11,678,000)</u>	<u>-</u>	<u>-</u>	<u>(11,678,000)</u>
Total marketable securities	<u>\$ 4,485,000</u>	<u>\$ 14,000</u>	<u>\$ (4,000)</u>	<u>\$ 4,495,000</u>

In February 2007, the FASB issued SFAS No. 159, *The Fair Value Option for Financial Assets and Financial Liabilities — Including an Amendment of FASB Statement No. 115*, which is effective for fiscal years beginning after November 15, 2007. This statement permits an entity to choose to measure many financial instruments and certain other items at fair value at specified election dates. Subsequent unrealized gains and losses on items for which the fair value option has been elected will be reported in earnings. The company elected to continue to measure related financial instruments and other items at their carrying amounts. Therefore, the adoption of SFAS No. 159 on July 1, 2008 did not have a material impact on the company's financial statements.

Note 6 – Amended Celera Agreement

The Celera agreement was amended twice during the most recent quarter. It was first changed on March 2, 2009. Pursuant to that amendment, the total future milestone payments to Celera were reduced to approximately \$97 million. Of that amount approximately 90% will become due upon regulatory approval and the achievement of specified net sales levels for the HDAC and Factor VII drug programs.

The amendment was again changed on March 30, 2009 to reflect changes to the payment timeline of certain payments to Celera and it also changed the obligations for the Company to pay royalties under certain conditions to Celera. In connection with this latest

third amendment, the Company paid Celera \$1 million in April 2009. This payment is creditable against amounts due Celera upon the company's sublicensing of one of the three programs and was recorded as research and development expense in the quarter ended March 31, 2009.

In April 2006 Pharmacyclics originally acquired multiple small molecule drug candidates for the treatment of cancer and other diseases from Celera Genomics, an Applera Corporation (now Celera Corporation) business. Future milestone payments to Celera are required for two of the three programs, HDAC and Factor VII; the BTK program will not require any milestone payments. Celera will be entitled to royalty payments in the mid-to high single digits, in percentage terms, based on annual sales of any drug commercialized from these programs.

Note 7 – Separation Agreements

In September 2008, Richard A. Miller, the company's President & CEO, and Leiv Lea, the company's Vice President, Finance and Administration and CFO resigned their positions and entered into separation agreements with the company. Under the separation agreements, Dr. Miller remained an employee until September 30, 2008 and Mr. Lea resigned his position as of October 31, 2008. Under the agreements, the company agreed to pay Dr. Miller and Mr. Lea one year of salary in severance payments, accelerate the vesting of all outstanding options, extend the exercise period of all outstanding options to three years after termination and provide healthcare benefits for twelve months following the termination of their employment. The company recorded severance expense of \$536,000 and share-based compensation expense of \$1,394,000 associated with the separation agreements in the quarter ended September 30, 2008. The company also recorded severance expense of \$600,000 including approximately \$200,000 relating to cash-based severance payments and share-based compensation expense of approximately \$400,000 in the quarter ended December 31, 2008 associated with Mr. Lea's separation agreement.

Note 8 – Related Party Notes Payable

On December 30, 2008, the company borrowed \$5,000,000 and on March 31, 2009 borrowed \$1,400,000 from Robert W. Duggan & Associates ("RWD"). Under the terms of the unsecured loans, the company is to pay RWD the principal sum of \$6,400,000 on the earlier of (i) July 1, 2010 or (ii) upon the closing of an equity offering or rights offering by the company. The loan bears interest as follows: (i) 1.36% from December 30, 2008 until March 31, 2009, (ii) the rate of interest in effect for such day as publicly announced from time to time by Citibank N.A. as its "prime rate" from April 1, 2009 until December 31, 2009 and (iii) the prime rate plus 2% from January 1, 2010 until the expiration of the loan. Interest is to be paid annually.

The principal amount of the loans have been discounted to fair value for balance sheet presentation such that the stated interest rate together with the accretion of the discount will reflect an estimate of the market interest rate during the term of the loan. As described in Note 5, SFAS No. 157 establishes a three-tier fair value hierarchy which prioritizes the inputs used in measuring the fair value of assets and liabilities. Due to the lack of reliable and objective observable data available to estimate the fair value of the \$5,000,000 loan, the value of the loan was determined using Level 3 inputs. These inputs included an estimate of the probable term of the loan. The loan matures at the earlier of an equity offering or a rights offering, meeting the criteria stated in the loan agreement, or eighteen months from its effective date. Due to already initiated corporate events and the current cash situation of the company, the probable term of the loan was estimated at 6.25 months.

With the term established, the company used various methods to estimate the fair market interest rate of the loan. The first and primary approach was a market risk approach, beginning with the risk-free rate on the effective date of the loan increased by the calculated estimates of the cost of each of the different premiums a lender would require for the various risks taken. These premiums included the non-marketability risk of the note, the risk of default, the cost of arrangement and the opportunity costs. Using this methodology, the company estimated the fair market interest rate to be 23%. The reasonableness of this rate was then further supported by comparison to the outcome of the Black Scholes pricing model calculated using the following assumptions:

- Strike price: \$5,000,000
- Expected term: 6.25 months
- Risk free interest rate: 0.28%
- Six month volatility: 139%

The calculation was performed using actual volatility for both the six-month and three-month periods prior to the loan effective date. The company's fair market rate estimate was further supported by market data estimating the interest rate on high yield bonds that the company believes would be of comparable quality to the loan.

As the terms of the \$1,400,000 loan are the same as the \$5,000,000 loan with the same expiration date, the company applied the same methodology to determine the fair value of this loan. Due to the lack of reliable and objective observable data available to estimate the fair value of the \$1,400,000 loan, the value of the loan was determined using Level 3 inputs. These inputs included first an estimate of the probable term of the loan. The loan matures at the earlier of an equity offering or a rights offering, meeting the criteria stated in the

loan agreement, or eighteen months from its effective date. Due to already initiated corporate events and the current cash situation of the company, the probable term of the loan was estimated at 3.25 months.

With the term established, the company used various methods to estimate the fair market interest rate of the loan. The first and primary approach was a market risk approach, beginning with the risk-free rate on the effective date of the loan increased by the calculated estimates of the cost of each of the different premiums a lender would require for the various risks taken. These premiums included the non-marketability risk of the note, the risk of default, the cost of arrangement and the opportunity costs. Using this methodology, the company estimated the fair market interest rate to be 23%. The reasonableness of this rate was then further supported by comparison to the outcome of the Black Scholes pricing model calculated using the following assumptions:

- Strike price: \$1,400,000
- Expected term: 3.25 months
- Risk free interest rate: 0.16%
- Three month volatility: 115%

The calculation was performed using actual volatility for the three month period prior to the loan effective date. The company's fair market rate estimate was further supported by market data estimating the interested rate on high yield bonds that the company believes would be of comparable quality to the loan.

The accretion of the remaining discount attributable to both of the RWD loans will cause an increase in the carrying amount of indebtedness and charges to interest expense from April 1, 2009 to June 30, 2009 of approximately \$300,000.

Note 9 – Income Taxes

We adopted the provisions of Financial Accounting Standards Board ("FASB") Interpretation No. 48, *Accounting for Uncertainty in Income Taxes-an interpretation of FASB Statement No. 109* ("FIN 48"), on January 1, 2007. FIN 48 clarifies the accounting for uncertainty in income taxes recognized in an enterprise's financial statements in accordance with SFAS No. 109, *Accounting for Income Taxes*, and prescribes a recognition threshold and measurement process for financial statement recognition and measurement of a tax position taken or expected to be taken in a tax return. FIN 48 also provides guidance on derecognition, classification, interest and penalties, accounting in interim periods, disclosure and transition. As of June 30, 2008, the company recorded a \$2.96 million reduction to deferred tax assets for unrecognized tax benefits, all of which were offset by a full valuation allowance. We may from time to time be assessed interest or penalties by major tax jurisdictions, although there have been no such assessments historically, with no material impact to our financial results. In the event we receive an assessment for interest and/or penalties, it would be classified in the financial statements as income tax expense. As of March 31, 2009, open tax years in major jurisdictions date back to 1991 due to the taxing authorities' ability to adjust operating loss carry forwards. The company does not anticipate a material change to its total amount of unrecognized tax benefits within the next 12 months.

Note 10 – Recent Accounting Pronouncements

In November 2007, the EITF issued a consensus, EITF 07-1, *Accounting for Collaboration Arrangements Related to the Development and Commercialization of Intellectual Property*, which is focused on how the parties to a collaborative agreement should account for costs incurred and revenue generated on sales to third parties, how sharing payments pursuant to a collaboration agreement should be presented in the income statement and certain related disclosure questions. EITF 07-1 is to be applied retrospectively for collaboration arrangements in fiscal years beginning after December 15, 2008. The company does not expect the adoption of EITF 007-1 to have a material impact on its results of operations or financial position.

In February 2008, the FASB issued FASB Staff Position No. FAS 157-2, "Effective Date of FASB Statement No. 157" ("FSP 157-2"), to partially defer FASB Statement No. 157, "Fair Value Measurements" ("SFAS 157"). FSP 157-2 defers the effective date of SFAS 157 for nonfinancial assets and nonfinancial liabilities, except those that are recognized or disclosed at fair value in the financial statements on a recurring basis (at least annually), to fiscal years, and interim periods within those fiscal years, beginning after November 15, 2008. The company adopted SFAS No. 157 for valuation and disclosures of its financial assets and liabilities in the first quarter of fiscal 2009 (see Note 5) and is currently evaluating the impact of adopting the provisions of FSP 157-2.

In July 2008, the company adopted EITF Issue No. 07-3, *Accounting for Nonrefundable Advance Payments for Goods or Services Received for Use in Future Research and Development Activities* ("EITF No. 07-3"). EITF No. 07-3 requires that nonrefundable advance payments for goods or services that will be used or rendered for future research and development activities be deferred and capitalized and recognized as an expense as the goods are delivered or the related services are performed. EITF No. 07-3 is effective, on a prospective basis, for fiscal years beginning after December 15, 2007. The adoption of EITF No. 07-3 did not had a material impact on the company's financial statements.

Note 11 – Related Party Transaction

As discussed in Note 8 – Note Payable, as of March 31, 2009, the company has borrowed \$6,400,000 from Robert W. Duggan & Associates (RWD) in the form of an unsecured loan. RWD is controlled by the company's Chairman of the Board and Chief Executive Officer, Robert W. Duggan. Mr. Duggan is the beneficial owner of approximately 27% of the company's outstanding common stock.

Note 12 – Unregistered Sale of Equity Securities

On February 19, 2009, the company sold approximately 1.5 million shares of common stock, at \$0.93 per share for an aggregate purchase price of approximately \$1.4 million. The purchasers of the shares were certain foreign and U.S. individuals and entities of which some are shareholders of Pacific Biopharma Group, Ltd. ("PBG"). Each investor acted individually and did not purchase shares for the account of PBG or any other affiliated company. Glenn Rice, our President and Chief Operating Officer, is a principal of PBG but did not participate in the transaction.

Note 13 – Subsequent Event

On April 16, 2009, the company entered into a collaboration agreement with Les Laboratoires Servier ("Servier"). Servier is the leading independent pharmaceutical company in France and the third in the world. Servier was founded in 1954, and is not related to Pharmacyclics. The Servier Group is established in 140 countries with its main therapeutic products used to treat diabetes, cardiovascular disease, CNS disorders, oncology and rheumatology. Over 25% of Servier's revenue is invested in Research & Development (R&D). Servier has 20,000 employees worldwide, including nearly 3,000 in R&D. Under the terms of the agreement, Servier acquired the exclusive right to develop and commercialize the Pan-HDAC inhibitor product worldwide except for the United States and will pay a royalty to Pharmacyclics on sales outside of the United States. Pharmacyclics will continue to own all rights within the United States. On May 11, 2009, Pharmacyclics received the upfront payment of \$10.45 million. Servier contractually withheld 5% of the upfront payment of \$11 million to pay for French withholding tax. Pharmacyclics is due to receive from Servier an additional guaranteed \$4 million for research collaboration over a 24 month period, paid in equal increments every 6 months with the initial payment due October 1, 2009. Servier will pay for all development costs outside the United States. In addition, Pharmacyclics has the right to receive approximately \$24.5 million upon the achievement of certain future milestones up to and including commercialization, as well as royalty payments based on regulatory approval and successful commercialization.

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

You should read the following discussion and analysis of our financial condition and results of operations together with our interim financial statements and the related notes appearing at the beginning of this report. The interim financial statements and this Management's Discussion and Analysis of Financial Condition and Results of Operations should be read in conjunction with the financial statements and notes thereto for the year ended June 30, 2008 and the related Management's Discussion and Analysis of Financial Condition and Results of Operations, both of which are contained in our Annual Report on Form 10-K filed with the Securities and Exchange Commission on September 5, 2008.

The following discussion contains forward-looking statements that involve risks and uncertainties. These statements relate to future events, such as our future clinical and product development, financial performance and regulatory review of our product candidates. Our actual results could differ materially from any future performance suggested in this report as a result of various factors, including those discussed in Part II, Item 1A, "Risk Factors", and elsewhere in this report, in the company's Annual Report on Form 10-K for the fiscal year ended June 30, 2008 and in our other Securities and Exchange Commission reports and filings. All forward-looking statements are based on information currently available to Pharmacyclics; and we assume no obligation to update such forward-looking statements. Stockholders are cautioned not to place undue reliance on such statements.

Overview

We are a clinical-stage biopharmaceutical company focused on developing and commercializing innovative small-molecule drugs for the treatment of immune mediated disease and cancer. Our purpose is to create a profitable company by generating income from products we develop, license and commercialize, either with one or several potential collaborators/partners or alone as may best forward the economic interest of our stakeholders. We endeavor to create novel, patentable, differentiated products that have the potential to significantly improve the standard of care in the markets we serve.

Presently, we have four product candidates in clinical development and two product candidates in pre-clinical development. It is our business strategy to establish collaborations with large pharmaceutical and biotechnology companies for the purpose of generating present and future income in exchange for adding to their product pipelines. In addition, we strive to generate collaborations that allow us to retain valuable territorial rights and simultaneously fast forward the clinical development and commercialization of our products.

It is our intention to identify product candidates based on exceptional scientific and development expertise, develop them in a rapid, cost-effective manner, and then seek development and/or commercialization partners. We are committed to high standards of ethics, scientific rigor, and operational efficiency as we move each of these programs to viable commercialization.

To date, substantially all of our resources have been dedicated to the research and development of our products, and we have not generated any commercial revenues from the sale of our products. We do not anticipate the generation of any product commercial revenues until we receive the necessary regulatory and marketing approvals to launch one of our products.

We have incurred significant operating losses since our inception in 1991, and as of March 31, 2009 have an accumulated deficit of approximately \$357.5 million. The process of developing and commercializing our products requires significant research and development, preclinical testing and clinical trials, manufacturing arrangements as well as regulatory and marketing approvals. These activities, together with our general and administrative expenses, are expected to result in significant operating losses until the commercialization of our products, or partner collaborations, generate sufficient revenues to cover our expenses. We expect that losses will fluctuate from quarter to quarter and that such fluctuations may be substantial. Our achieving profitability depends upon our ability, to successfully complete the development of our products, obtain required regulatory approvals and successfully manufacture and market our products.

Our Pipeline

Our pharmaceutical drug development candidates are synthetic small-molecules designed to target key biochemical pathways involved in human diseases with critical unmet needs. We currently have four proprietary drug candidates under clinical development and one drug candidate under preclinical development, and a lead compound undergoing preclinical optimization. This includes a histone deacetylase inhibitor (PCI-24781) about to enter a Phase II clinical trial; an inhibitor of Factor VIIa (PCI-27483) soon to be in a Phase II clinical trial; an inhibitor of Bruton's tyrosine kinase (Btk) (PCI-32765) currently in a Phase I clinical trial targeting oncology applications; a Btk inhibitor (PCI-45261 Series) in advanced preclinical lead optimization and testing targeting autoimmune and allergic indications; and an HDAC8 inhibitor lead (PCI-34051) that is currently being optimized for autoimmune and cancer indications. Motexafin gadolinium (MGd) is now in a Phase II trial being conducted by the National Cancer Institute (NCI) in patients with primary brain tumors.

Status of Products Under Development

The table below summarizes our product candidates and their stage of development:

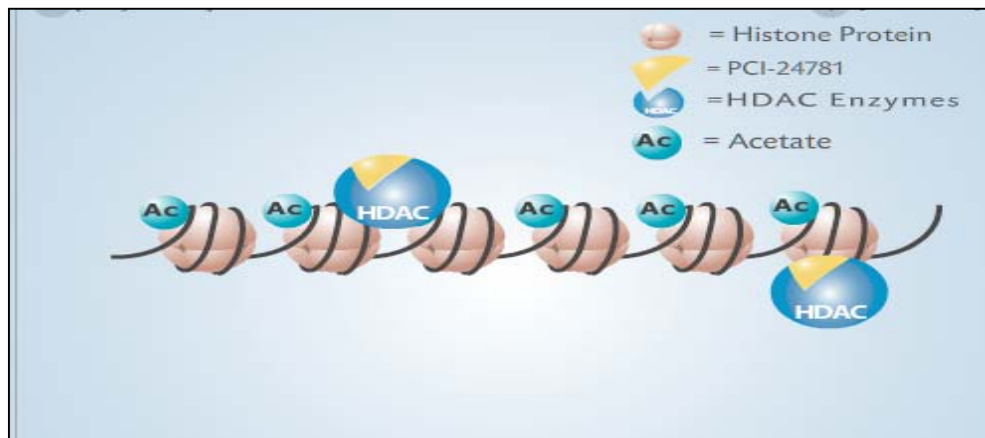
Product Candidate	Disease Indication	Development Status(1)
PCI-24781 HDAC Inhibitor	Advanced solid tumors Recurrent lymphomas Sarcoma	Phase I - enrolling Phase I/II - enrolling Phase I/II - planned second half 2009
PCI-27483 Factor VIIa Inhibitor	Cancer therapy	Phase I - complete Phase II - planned second half 2009
PCI-32765 B Cell Tyrosine Kinase Inhibitor	B-Cell Lymphomas	Phase I - enrolling
PCI-45261 Series B Cell Tyrosine Kinase Inhibitor	Autoimmune disease and Mast cell disease	Preclinical
PCI-34051 HDAC8 Inhibitor	Autoimmune and cancer	Preclinical
MGd	Primary brain tumor ² Childhood brain tumors ²	Phase II - enrolling Phase II - complete

1. "Phase I" means initial human clinical trials designed to establish the safety, dose tolerance, pharmacokinetics (i.e. absorption, metabolism, excretion), and pharmacodynamics (i.e. surrogate markers for efficacy) of a compound. "Phase II" means human clinical trials designed to establish safety, optimal dosage and preliminary activity of a compound in a patient population.

2. Studies sponsored by the National Cancer Institute.

Histone Deacetylase Inhibitor Program

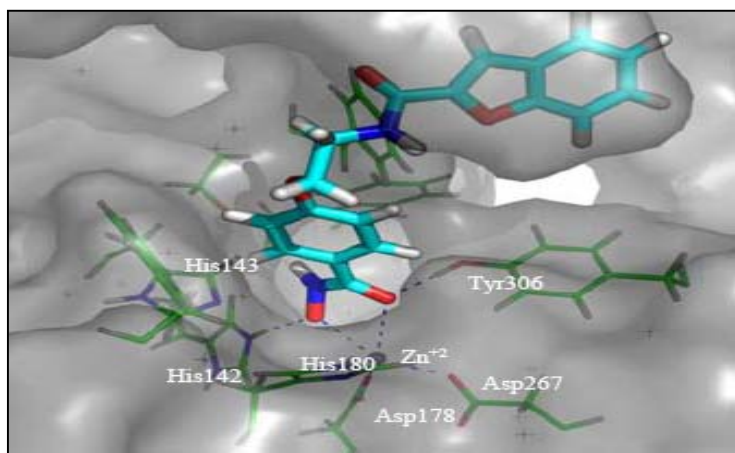
The human genome consists of a complex collection of genes which are turned on or off depending on the needs of the cell. Cancer is characterized by genome-wide changes in gene expression within the tumor. Turning off the expression of certain genes favors a tumor's ability to multiply, to avoid apoptosis (i.e. programmed cell death) or to become resistant to chemotherapy. One of the ways in which genes are turned on or off is by means of chemical modification of histone proteins. Histone proteins are structural components of chromosomes, and form a scaffold upon which DNA, the genetic material, is arranged, see image below. Histone acetylation (i.e. the addition of an acetate group to histones) alters the expression of genes involved in cell cycle control, cell division, and apoptosis. Histone deacetylation reverses histone acetylation by removing the acetyl groups. The process of histone deacetylation is controlled by a family of enzymes known as histone deacetylases (or "HDACs"). HDAC inhibitors prevent deacetylation, leading to an increase in histone acetylation and an increased expression of certain genes. This effect limits the tumor's ability to multiply, to avoid apoptosis or to become resistant to chemotherapy. HDAC inhibitors block cancer cell proliferation *in vitro* (i.e. in cultured cells) and cancer cell growth arrest is observed *in vivo* (i.e. in animals) at non-toxic concentrations.



PCI-24781 (Pan HDAC Inhibitor)

PCI-24781 is a novel, potent, small-molecule inhibitor of HDAC enzymes with anti-tumor activity *in vitro* and *in vivo* (Buggy *et al* Mol Cancer Ther 2006; 5 (5), p. 1309-1317). PCI-24781 treatment leads to synergistic efficacy in tumor cells in combination with DNA-damaging agents such as radiation and chemotherapy agents. The mechanism of the synergy may involve inhibition of DNA repair. PCI-24781 has activity against primary human tumors from patients with colon, ovarian, lung and many hematological (i.e. blood related) cancers.

We believe PCI-24781 has a half-life and potency superior to competitor drugs (e.g. Zolinza or LBH-589) that will allow us to achieve an ideal balance of efficacy with minimal toxicity.



Co-crystal of PCI-24781 chemical scaffold with HDAC showing optimized interactions with active site residues

Clinical Development -Oncology

Clinical development began with intravenous administration of PCI-24781 in an initial Phase I study, and has progressed to two clinical studies by the oral route in 2008, both of which are currently enrolling. The first study employing an oral capsule formulation (PCYC-0402) is a Phase I, ascending dose study in patients with solid tumors. This study is open and actively enrolling at four clinical centers: MD Anderson Cancer Center, Marin Oncology, The University of Chicago, and Sarah Cannon Cancer Center (www.clinicaltrials.gov). Single agent stable disease has been achieved in a number of solid tumor histologies including colon, tongue and ovarian carcinoma.

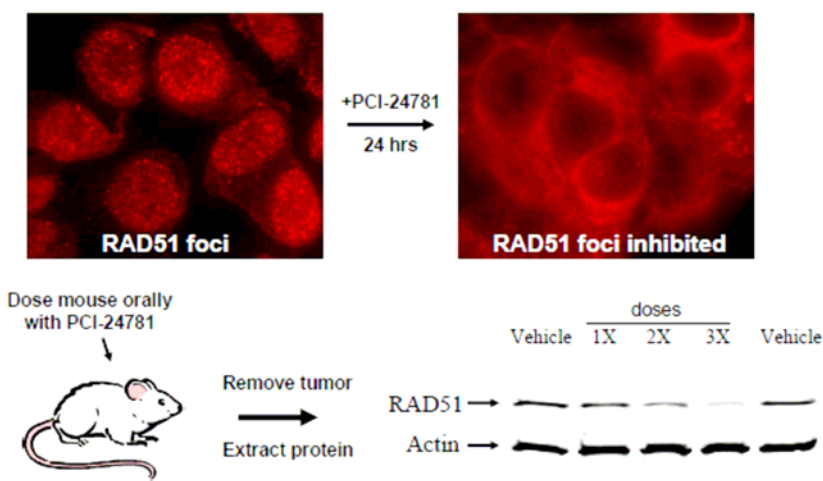
The second study by the oral route (PCYC-0403) is a Phase I/II trial in patients with lymphoma. The improved potency and pharmacokinetic aspects of PCI-24781 served as a basis for the ongoing proof of concept studies in Phase I/II in lymphoma. This trial is now open and actively enrolling at four centers: University of California, San Francisco, University of Nebraska, Northwestern University, and Washington University (St. Louis). Clinical responses have been recorded in this single agent clinical trial, with one partial response and six stable diseases to date in ten evaluated patients. Thrombocytopenia (reduced platelet count) is a reversible effect that has been observed with a number of HDAC inhibitors and is thought to be related to the pharmacologic mechanism of action. In the case of PCI-24781 we believe the thrombocytopenia can be successfully managed through dose scheduling changes. No other drug related serious adverse events have been observed to date.

A third clinical study, a Phase I/II, will test PCI-24781 in combination with doxorubicin in patients with soft tissue sarcoma. This trial will be co-sponsored by prominent investigators at Massachusetts General Hospital and Dana-Farber/Harvard Cancer Center, including Drs. George Demetri and Edwin Choy, and is planned to begin in second half of calendar 2009.

Proprietary Predictive Assays

Following chemotherapy or radiation treatment, some patients' tumors may turn on certain genes as a strategy by the tumor to adapt to the therapy and become resistant to cell death. One example of a genetic change that occurs in many cancers is the activation of the DNA repair gene RAD51. In response to treatment with DNA-damaging chemotherapy or radiation, tumors will often turn on DNA repair genes, such as RAD51, as an adaptive strategy to help the tumor repair the DNA damage done by these agents. In pre-clinical models, PCI-24781 was able to turn off RAD51 (and other DNA repair genes), effectively blocking the ability of the tumor to repair its damaged DNA, sensitizing the tumor to chemotherapy and radiation. PCYC has patented the predictive use of the biomarker RAD51 which was found by Pharmacyclics' scientists to potentially underlie resistance to therapy and may be used as a predictive measure of HDAC inhibitor activity that could be useful in the clinic. This research was published in the *Proceedings of the National Academy of Sciences* (Proc Natl Acad Sci U S A. 2007;104:19482-7. Epub 2007 Nov 27).

Thus PCI-24781 is effective at inhibiting repair of damaged DNA by downregulating RAD51, which is particularly essential for repair of double-strand breaks (DSB). It was demonstrated by Pharmacyclics that PCI-24781 effectively prevents DSB repair via one of the two major repair pathways, called the homologous recombination pathway, by modulation of RAD51. This allows PCI-24781 to synergize effectively with other agents that damage DNA, such as radiation (Banuelos et al., Clin Cancer Res., v. 13, p. 6816-6826, 2007) and chemotherapeutics i.e. doxorubicin (Adimoolam et al., Proc.Natl.Acad.Sci.U.S.A, v. 104, p. 19482-19487, 2007). We showed recently that RAD51 is over expressed in a majority of human lymphoma samples and that pretreatment with PCI-24781 down regulates RAD51 and potentiates cell killing by subsequent addition of doxorubicin (Balasubramanian et al., Blood (ASH 2007 Abstracts), v. 110, p. 1377.2007). One of our collaborators, Dr. Dina Lev at MD Anderson Cancer Center, has shown that PCI-24781 can also synergize with doxorubicin in sarcoma, both in cells and in animal models (Lopez et al., Clin Cancer Res., In Press. 2009). Accordingly, as mentioned above we plan to begin a Phase I/II trial of PCI-24781 in combination with doxorubicin for treating sarcoma with Dr. Edwin Choy at Massachusetts General Hospital and Dr. George Demetri at Dana-Farber Cancer Institute. These investigators are part of one of the leading consortiums in sarcoma in the world today. It is anticipated that clinical activity in this trial would pave the way to other indications for PCI-24781 in combination with doxorubicin, which is also used extensively in treatment of other cancers, including lymphoma, breast, lung, ovarian and liver cancer.

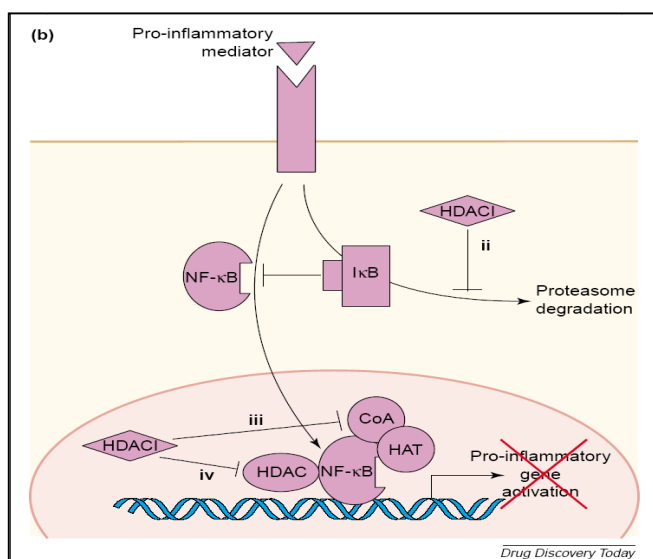


Rad51 is a DNA repair gene that Pharmacyclics scientists have discovered that predicts sensitivity to PCI-24781. Top: PCI-24781 disrupts nuclear repair foci in colon cancer cells. Bottom: PCI-24781 downregulates RAD51 in tumors grown in mice.

Market

Pan-HDAC inhibitors have the potential for broad anti-cancer indications in hematologic and solid malignancies when used in combination with numerous chemotherapeutic drugs and radiation.

Specific HDAC enzymes have been implicated in many other physiological processes and there is growing interest in using HDAC inhibitors in many disease areas including metabolic, neurological and immunological disorders as well as for treating bacterial and parasitic infections. For instance, in central nervous system (CNS) indications, HDAC inhibitors have shown activity in models of epilepsy and migraine headaches, dementia, Alzheimer's, Parkinson's and Huntington's disease (recently reviewed in Kazantsev & Thompson, Nat Rev Drug Discov. 2008 7(10):854-68; Steffan JS et al. Nature. 2001 Oct 18;413(6857):739-43). HDAC inhibitors have shown substantial activity in inflammatory models including rheumatoid arthritis, juvenile RA, multiple sclerosis, psoriasis, lupus, sepsis, diabetes and hemorrhagic shock (reviewed in Chipoy C. Drug Discovery Today. 2005 1;10(3):197-20; Gray SG, Dangond F. Epigenetics. 2006 Apr-Jun;1(2):67-75. Epub 2006 Mar 5; Susick L et al. J Cell Mol Med. 2009 epub Jan 28). Finally, HDAC inhibitors have shown substantial activity in antiviral, antibacterial and antiparasitic applications (Elaut G, et al. Curr Pharm Des. 2007;13(25):2584-620).



The anti-inflammatory effects of HDAC inhibitors can act in multiple ways. One way as shown here is through the inhibition of a major regulator of pro-inflammatory gene expression, the transcription factor NF-κB subunit p65.

Pharmacyclics is actively involved in exploring many of these non-oncology indications internally as well as with outstanding academic collaborators. Our internal programs include applications for RA, juvenile RA and dermatitis. Currently, Pharmacyclics is reviewing potential clinical options in these areas.

Patents

Key patent protection in US and international territories will extend beyond 2024 with the possibility of patent term extensions during development.

Competition

Merck's vorinostat (Zolinza®) has been approved by the FDA for cutaneous T-cell lymphoma patients who have progressive, persistent or recurrent disease on or following failure of two systemic therapies, making the oral drug the first in its class to reach the market. A number of structurally distinct HDAC inhibitors are currently in clinical trials including Novartis' LBH-589, the natural product depsipeptide (FK-228) from Gloucester, and the benzamide, SYND 275. HDAC inhibitors have exhibited clinical activity against a variety of human malignancies in initial clinical trials. For example, clinical improvements have been observed in patients with renal cell carcinoma, head and neck squamous carcinoma, mesothelioma, small-cell lung cancer, melanoma, papillary thyroid carcinoma and B- and T-cell lymphomas. Thrombocytopenia (a reduction in platelets, which are cells responsible for clotting blood) was identified as a dose-limiting toxicity for patients administered a number of these agents. Several of the competitors have reported cardiac toxicities such as Grade 3 QTc prolongation, arrhythmias and atrial fibrillation, in addition to fatigue, anorexia, infection, headache and nausea. Preliminary data suggests that PCI-24781 has not shown significant side effects, (other than reversible thrombocytopenia) in clinical studies suggesting that PCI-24781 may offer a less toxic modality for the treatment of cancer than its competitors.

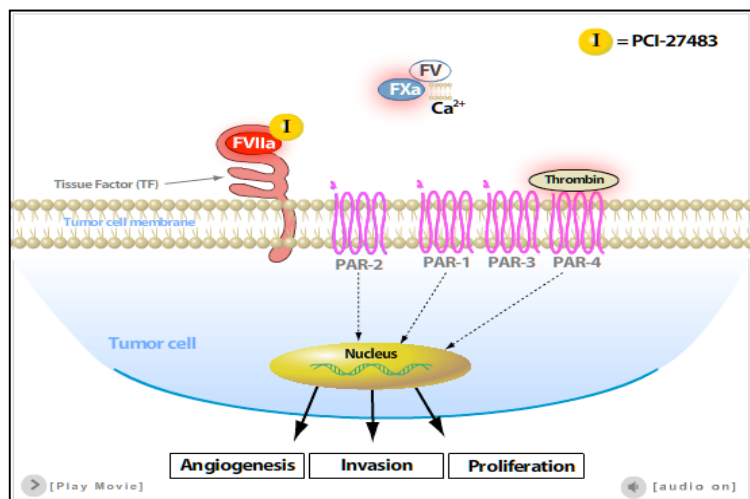
Partnering

On April 16, 2009, the company has entered into a collaboration agreement with Les Laboratoires Servier ("Servier") pursuant to which Pharmacyclics granted to Servier an exclusive license for its Pan-HDAC inhibitors, including PCI-24781, for territories throughout the world excluding the United States. Under the terms of the agreement, Servier acquired the exclusive right to develop and commercialize the Pan-HDAC inhibitor product worldwide except for the United States and will pay a royalty to Pharmacyclics on sales outside of the United States. Pharmacyclics will continue to own all rights within the United States (see Note 13) for further details).

Please see our website for additional information and further explanations.

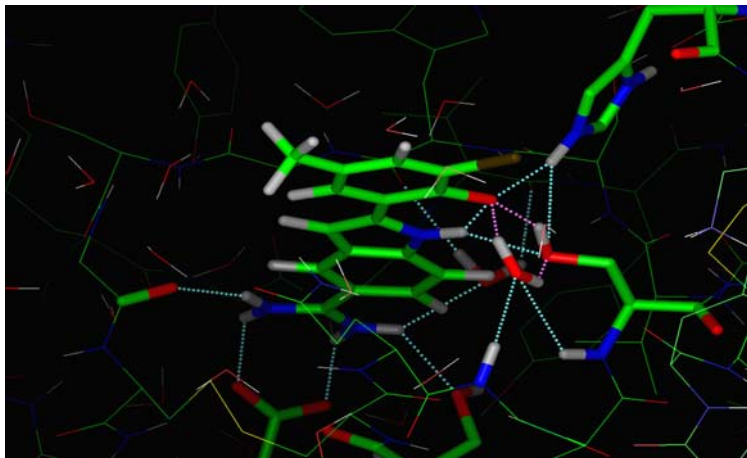
Factor VIIa Inhibitor Program

Factor VII is an enzyme that becomes activated (fVIIa) by binding to tissue factor (TF, a cell membrane protein). The fVIIa/TF complex triggers the extrinsic coagulation cascade that leads to the formation of a blood clot. Tissue factor is expressed in many cells such as fibroblasts and keratinocytes (i.e. skin cells), but is absent from vascular cells that come in contact with circulating fVII in the blood. Preclinical models of thrombosis (blood clots) in several species have indicated that a selective inhibitor of the Factor VIIa/Tissue Factor (fVIIa/TF) complex may have a greater therapeutic/safety index than inhibition of other coagulation factors. In many cancers, such as those arising from the pancreas, lung, stomach or colon, over expression of tissue factor is associated with an increased incidence in blood clots. Tissue factor over expression also correlates with a worsened prognosis for a number of human cancers (e.g. colorectal, pancreatic, glioblastoma, renal, etc.). Inhibitors of fVIIa/TF complexes have been shown to inhibit the growth of primary and metastatic tumors in mice.



PCI-27483

PCI-27483 is a highly optimized and first of its kind, small molecule inhibitor of Factor VIIa developed by Pharmacyclics' scientists. This drug selectively inhibits the active form of Factor VII (called Factor VIIa). PCI-27483 is an extremely potent inhibitor of coagulation Factor VII but does not inhibit other coagulation factors, such as Factor XIa, Factor IXa, Factor IIa (Thrombin) and Factor Xa.

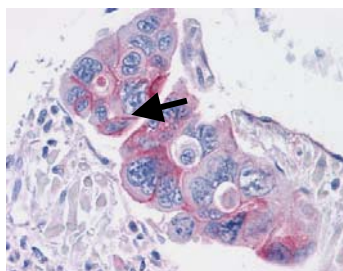


PCI-27483 was developed using rational drug design (Katz, B. A.; *et al. J. Mol. Biol.* **2001**, *307*, 1451-1486) against the target molecule Factor VIIa

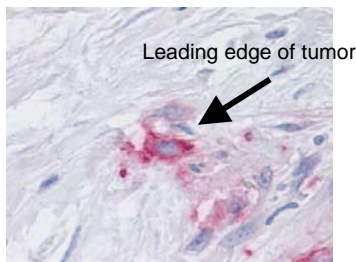
The antithrombotic effects of subcutaneously injected PCI-27483 were determined in a baboon model of arterial thrombosis. Increasing subcutaneous doses of PCI-27483 progressively has an antithrombotic effect similar to that of the low molecular weight heparin (i.e. anti coagulant) product, Lovenox.

In cancer, the Factor VIIa:TF complex triggers a host of physiologic processes that facilitate tumor angiogenesis, growth and invasion. Laboratory studies and animal models indicate that PCI-27483 blocks tumor growth, angiogenesis and metastases.

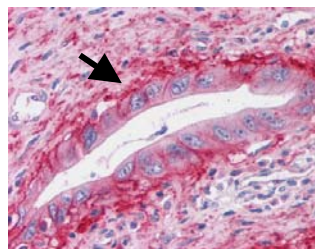
Clinical Program



Malignant Cells 40X



Malignant Cell at Pushing Margin of Invasion 60X



Malignant Cells and Surrounding Fibrocollagenous Matrix 40X

FVIIa was detected in 12/13 pancreatic carcinomas by staining techniques. Staining was detected in malignant cells while all normal cells were negative. Staining often detected at the leading edge of tumor invasion.

Pancreatic cancer is one of the significant causes of death from cancer in the US and Europe. Despite the improvements in the diagnosis and treatment of cancer, patients with locally advanced and/or metastatic pancreatic cancer have a median survival time of approximately 5 to 6 months. Gemcitabine is the most active drug in the treatment of advanced pancreatic cancer; however, the response rates of single agent gemcitabine are between 5% and 11% with a median survival time varying between 5.7 and 6.5 months. Cisplatin, a chemotherapy agent, with gemcitabine has been reported to yield response rates of 10–20% and 4–9 months of median survival times. Clearly, more effective therapy is needed.

TF expression has been observed in 89% of pancreatic cancers, but not within the typical pancreas. Pancreatic cancer patients with high TF expression have a venous thromboembolism rate of 26.3% compared with 4.5% in patients with low TF expression. (Korana et. al. Clin Cancer Res. 2007 May 15;13(10):2870-5). Indeed, thromboembolic complications are increasingly considered to be the leading cause of death in patients with cancer (Levine MN: Cancer Treat Rev 2002;28:145–149). Among 66,000 patients with cancer admitted to US medical centers from 1995 to 2002, patients with pancreatic cancer had the highest risk of thromboembolic complications (12.1% per hospitalization) (Khorana et. al. J Clinical Oncology 2006, 24: 484-490). TF expression occurs early in

pancreatic cancer, thus Pharmacyclics believes pancreatic cancer is an excellent focus for development of PCI-27483, which will have a dual mechanism of action of inhibiting tumor growth and thromboembolic events.

We have recently completed our initial Phase I testing of PCI-27483 in healthy volunteers. The primary objective of the ascending dose Phase I study was to assess the pharmacodynamic and pharmacokinetic profiles of PCI-27483 following a single, subcutaneous injection. In addition, the safety and tolerability of PCI-27483 was evaluated. The drug was well tolerated and no adverse event was observed at any dose level. The International Normalized Ratio (INR) of prothrombin time, a laboratory test for coagulation, was used to measure pharmacodynamic effect at dose levels of 0.05, 0.20, 0.80 and 2.0 mg/kg. Anticoagulation effects can be precisely and accurately measured a few hours following dosing with a simple blood test. A mean peak INR of 2.7 was achieved without adverse effects at the highest dose level administered. The target INR range for oral anti-coagulants i.e. Coumadin, is between 2 and 3. The half-life of PCI-27483 was 9 to 10 hours, which compares favorably to the single-dose half-life of the low molecular weight heparin Lovenox (4.5 hours) and Fragmin (3 to 5 hours).

A multicenter Phase II study is planned to begin second half of calendar 2009. The target patient population is locally advanced (non-metastasized) pancreatic cancer within 2 months of diagnosis either receiving or planned to receive gemcitabine therapy. The goals will be to; a) assess the safety of PCI-27483 at pharmacologically active dose levels; b) to assess potential survival benefit and c) obtain initial information of the effects on the incidence of thromboembolic events.

Market

Each year 230,000 individuals worldwide are diagnosed with pancreatic cancer (in the US more than 34,000 are diagnosed each year). The overall pancreatic cancer market is forecasted to double to \$1.2 billion in 2016. There are approximately 870,000 new cases of gastric cancer worldwide per year, with 670,000 deaths. Worldwide incidence of other cancers types that also have been shown to have high TF expression include: colon cancer (940,000 new cases per year); ovarian (190,000 new cases per year); breast (1.2 million new cases per year), and lung cancer (1.2 million new cases per year).

Patents

PCI-27483 (as a compound, in pharmaceutical compositions and in uses for treating a variety of diseases) is covered by US patent applications (issued and pending) and PCT national phase patent applications in 14 other jurisdictions, including Europe, Canada, Japan, China, India, South Korea, Australia and Brazil. The projected expiration of this coverage is through at least 2024 (without including patent term extensions in the various territories).

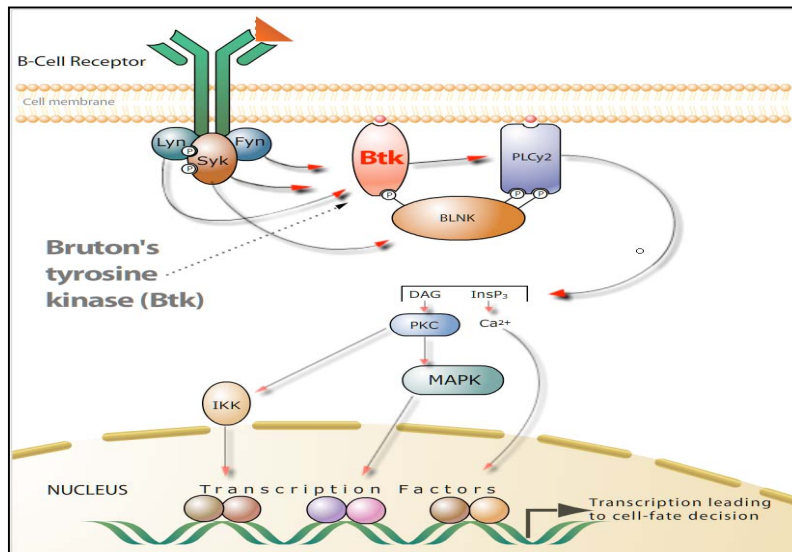
Partnering

Pharmacyclics will seek a partner to co-develop PCI-27483. We believe this unique drug may be competitively positioned for a significant partnership following the successful achievement of further clinical milestones.

Please see our website for additional information and further explanations.

Btk Inhibitors

Pharmacyclics is pioneering the development of orally bioavailable inhibitors of Bruton's tyrosine kinase (Btk), a signaling molecule that is critically important for the activity of B-cells (i.e. cells that lead to the productions of antibodies) and mast cell (i.e. a cell involved in allergic responses). When B-cells are overactive, the immune system produces inflammatory cells and antibodies that begin to attack the body's own tissue, leading to autoimmune diseases. Also, B-cell lymphomas and leukemias, which are common blood cancers, result from mutations acquired during normal B-cell development leading to uncontrolled B-cell proliferation and B-cell malignancies. Specific cancer indications include non-Hodgkin's lymphoma (NHL), chronic lymphocytic leukemia (CLL), and as a potential inhibitor of tumor stem cells (also known as Tumor Initiating Cells or TIC's) that have been identified in certain cancers. In addition, Btk inhibitors have potential for treatment of autoimmune diseases such as rheumatoid arthritis (RA), systemic lupus erythematosus (SLE), and allergic diseases such as eosinophilic esophagitis. Pharmacyclics has developed two programs of proprietary and chemically distinct inhibitors, producing one candidate optimized for oncology (PCI-32765) and currently in Phase I clinical trials; and one series of molecules (PCI-45261 Series) being optimized for autoimmune applications for an anticipated IND in the second half of 2010.



BTK plays a critical role in signaling via B-cell receptor (BCR) signaling. Btk inhibitors block B-cell activation and auto-antibody formation.

Genetic Validation of Inhibiting the Target in Humans

Unlike competing programs for inhibiting B-cell signaling such as with Syk inhibition, a human genetic mutation exists which helps to validate Btk as a drug target. Bruton's agammaglobulinemia (XLA) is an X-linked disease (only male offspring being effected) occurring in approximately 1 in 250,000 males, which disrupts the function of BTK. In the absence of Btk, B-cells do not come about or mature. Males with XLA have a total or almost total absence of B-cells and very low levels of circulating antibodies. Therefore, Btk is absolutely necessary for the proliferation and the differentiation of B-cells. A point mutation in mice also causes X-linked immunodeficiency (*xid*), with ~50% fewer conventional B2 B-cells, absent B1 B-cells, and reduced levels of antibodies.

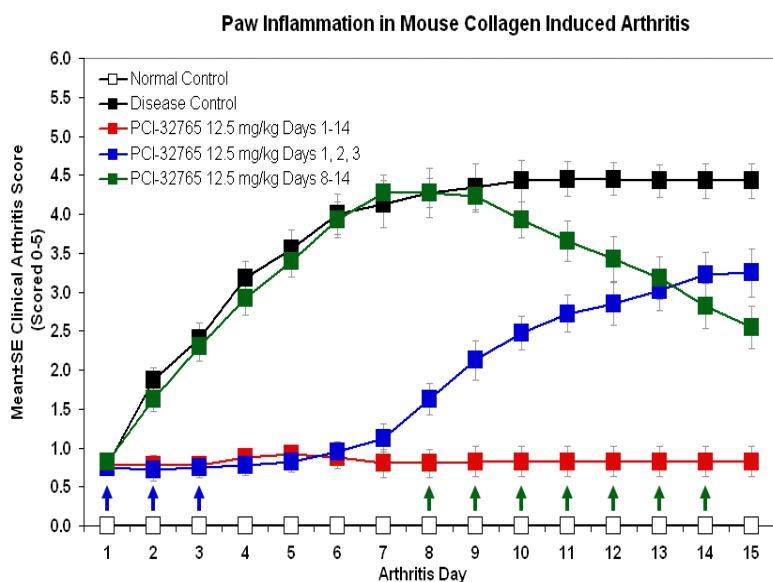
PCI-32765 for Oncology

We have developed highly selective, small-molecule inhibitors of Btk using a proprietary scaffold and demonstrated oral efficacy in preclinical models of lymphoma and spontaneous lymphoma in dogs. Our inhibitors take advantage of a unique and proprietary mechanism to achieve potency and selectivity over other kinases (i.e. signaling molecules). PCI-32765 inhibits purified Btk with an IC₅₀ of 0.46 nM. In *ex vivo* stimulation assays in whole blood, PCI-32765 inhibits human B-cell receptor activation (IC₅₀ ≈ 200 nM), while not affecting T-cell activation. We have also confirmed that PCI-32765 inhibits key phosphorylation events downstream of the B-cell receptor at similar concentrations. A one hour pulse of PCI-32765 is sufficient to inhibit B-cell activation for ~18 hours in cellular assays.

Our proprietary approach towards developing inhibitors makes PCI-32765 uniquely selective over closely related kinases. B-cell receptor signaling is implicated in the survival of B-cell derived Non-Hodgkin's lymphoma. Studies have shown that PCI-32765 inhibits the proliferation of B-cell lymphoma and leukemia cells. We have demonstrated that PCI-32765 kills a subset of lymphoma cell lines (GI₅₀ <1 μM). We have recently initiated a trial of PCI-32765 in spontaneous canine lymphoma in companion animals. Thus far, in five dogs, with monoclonal B-cell Lymphoma, we have observed two partial responses (by RECIST criteria) following treatment with PCI-32765.

PCI-32765 Preclinical Proof of Concept for Autoimmune diseases

In animal models of rheumatoid arthritis, oral administration of PCI-32765 leads to the regression of established disease. *In vivo*, once daily oral dosing of PCI-32765 inhibited collagen induced arthritis (CIA) in the mouse (ED₅₀ = 4.55 mg/kg/day). In a scheduling study (below), PCI-32765 rapidly regressed disease even when dosing was initiated at day 8, when inflammation was maximal. In addition, three days of PCI-32765 dosing resulted in inhibition of disease for six days, suggesting that intermittent dosing of PCI-32765 may result in sustained therapeutic effect. No PCI-32765-related weight loss was observed in the arthritis studies when dosed up to 200 mg/kg/day for 10 days. PCI-32765 also prevents the progression of anti-collagen induced arthritis at doses as low as 3 mg/kg.



In mouse models of collagen induced arthritis, orally administered PCI-32765 actually reversed disease. Shown are two dosing schedules for PCI-32765. In blue, animals were dosed for three days then the drug was withdrawn. The induction of the disease was delayed for four days. In green, the animals were allowed to develop the disease, and at day 8, the animals were dosed with PCI-32765. Within one day, the degree of disease severity was decreased.

PCI-32765 also prevents mast cell activation *in vitro* and inhibits mast cell-dependent anaphylaxis *in vivo*. Dual inhibition of mast cell and B-cell activation may explain the significant efficacy of PCI-32765 in animal models and may provide a treatment modality for a variety of allergic diseases including asthma and allergy.

Clinical Development of PCI-32765

A robust kilogram-scale GMP synthesis has been developed and API is available to support clinical studies. An optimized capsule formulation has been developed.

We have developed multiple pharmacodynamic assays to monitor inhibition of B-cells in peripheral blood including a proprietary fluorescent probe assay that can be used to monitor active-site occupancy of Btk by our inhibitors. We have confirmed that efficacy in our autoimmune models is correlated with doses that lead to Btk occupancy. In addition, we have adapted the probe assay so that it can be used to monitor Btk occupancy by PCI-32765 in human blood. This assay will be used to determine what dose levels of PCI-32765 lead to occupancy of Btk in clinical trials. In addition, we can measure inhibition of B-cell signaling and mast cell activation *ex vivo* using samples from PCI-32765 treated patients.

A Phase I trial in surface immunoglobulin positive B-cell lymphoma has begun at three clinical sites in the US. The objective of this study will be to determine the safety and tolerability of a 28-day oral dosing regimen and to evaluate effects on pharmacodynamic assays and tumor response.

Potential New Oral Disease Modifying Anti-Rheumatic Drug (DMARD) - PCI-45261 Series

Using the same chemical scaffold as PCI-32765, work was initiated on a second generation Btk inhibitor with the goal of optimizing for use in chronic disease. New chemical entities are being screened in a series of efficacy, pharmacokinetic, and safety assays designed to identify compounds that retained potent inhibition of Btk while exhibiting better selectivity and better pharmaceutical properties. Our current lead molecule, PCI-45261 Series was identified in December 2008. Btk inhibition by PCI-45261 Series is >2500-fold selective over the tyrosine kinases EGFR and JAK-3. We have confirmed that orally dosed PCI-45261 Series is highly efficacious in a mouse model of collagen induced arthritis. Relatively low efficacious doses are predicted for humans based on interspecies scaling. We are currently in the final stages of optimizing this series of molecules.

Data to date for PCI-32765 and PCI-45261 Series demonstrates improvements in signs of inflammation in rheumatoid arthritis models. Based on the mechanism of action, we expect that the optimized drug from this series will delay the progression of the disease and be classified as a DMARD (disease modifying anti-rheumatic drug).

Market Size

Pharmacyclics will generate proof-of-concept data in both lymphoma and RA indications. Pharmacyclics is not aware of any other competitors in clinical trials with other Btk inhibitors. The anti-B-cell biologics such as Rituxan® and Lymphostat B all have a distinction of massive B-cell depletion and lack of convenient oral dosing. The overall Non Hodgkin's Lymphoma market is

projected to increase from \$3.3 billion in 2007 to \$4.7 billion in 2017 (3.6% a year). It's expected that the sales of Rituxan® in the NHL market to increase from \$2.8 billion 2007 to \$3.2 billion in 2017.

The potential market size in autoimmune indications as an orally administered DMARD is even larger. Anti-TNF therapies such as Enbrel® and Humira® are T-cell specific with inconvenient subcutaneous injection dosing. Current aggregate market size of the anti-TNF therapies is \$5 billion. The clinical and regulatory strategy for both diseases is well defined. The market for rheumatoid arthritis (RA) therapies will show robust growth between 2009 and 2017; major market sales will nearly double to \$13.4 billion in 2017. We think the market opportunities in the other indications and potential future applications such as lupus are also quite large.

Patents

A variety of non-provisional PCT applications have been filed for methods, uses and composition of the lead and second generation compounds including PCI-32765 and for the Btk fluorescent probe (PD marker). For lead clinical candidate (PCI-32765), we expect global patent protection till at least December 2026 (without including pharmaceutical extensions).

Partnering

With further development progress Pharmacyclics will be seeking strategic pharma / biotech partner(s) to develop and commercialize PCI-32765 and PCI-45261 Series.

Please see our website for additional information and further explanations.

HDAC8-specific inhibitor program: PCI-34051

Pharmacyclics' scientists have been in the forefront of research into inhibitors for specific HDAC enzymes beginning with the cloning of the human HDAC8 in 2000 (Buggy et al., Biochem.J, v. 350 Pt 1, p. 199-205, 2000). Since then, we were the first to publish the crystal structure of a human HDAC (HDAC8) in 2004 (Somoza et al., Structure., v. 12, p. 1325-1334.2004), the first to publish the most selective inhibitor of human HDAC8 (PCI-34051) in 2008 (Balasubramanian et al., Leukemia., v. 22, p. 1026-1034, 2008), and the first to discover a novel anti-inflammatory activity of a HDAC8 inhibitor (Balasubramanian et al., in preparation 2009). This has led to a strong intellectual property position, with multiple patents on the gene, protein and a large selective inhibitor panel, and worldwide recognition of our efforts with seminar and poster presentations at major international conferences including the first HDAC inhibitors conference in 2007 and a subsequent one in 2008, as well as AACR and ASH conferences.

Using our unique knowledge of the crystal structure of HDAC8 complexed with multiple pan- and selective inhibitors, we have discovered a novel HDAC8 selective inhibitor, PCI-34051, which inhibits HDAC8 with a K_i of 10 nM with >200 fold selectivity over the other HDACs tested. With this very important tool compound, we have identified multiple clinical applications for this class of drugs.

T-cell lymphoma: PCI-34051 induces growth arrest and apoptosis in T-cell lymphomas and leukemias, but not in any other hematologic and most solid tumors (Balasubramanian et al., Leukemia., v. 22, p. 1026-1034.2008). Thus, it has the potential to offer an improved therapeutic index in these indications over non selective HDAC inhibitors such as vorinostat, which was approved for CTCL in 2006 but has been associated with multiple toxicities in the clinic.

Pediatric neuroblastoma: HDAC8, uniquely among all HDAC enzymes, is overexpressed in pediatric neuroblastoma tumors, and a high HDAC8 expression level is strongly associated with a poor prognosis (Oehme et al., Clin Cancer Res, v. 15, p. 91-99 2009). HDAC8-specific inhibitors including PCI-34051 induce growth inhibition and differentiation into non-tumor forms of neuroblastoma cells. Thus, HDAC8-specific inhibitors could prove valuable in treating this disease for which there is no curative therapy at present.

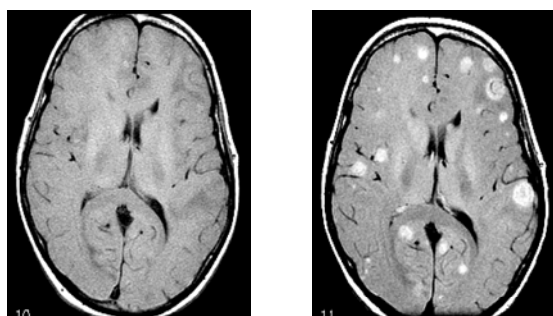
Inflammatory disease: We have discovered that PCI-34051 inhibits the secretion of many pro-inflammatory proteins from blood cells (Balasubramanian et al., in preparation 2009). It is particularly effective at modulating the proteins interleukin-1 beta (IL1b) and interleukin-18, both of which are associated with many autoimmune disorders. Anti-IL1b protein therapeutics have proven effective in treatment of RA and systemic juvenile RA (Pascual et al., J Exp.Med, v. 201, p. 1479-1486 2005), Adult-onset Still's Disease (Lequerre et al., Ann.Rheum.Dis., v. 67, p. 302-308, 2008), Familial Cold Syndrome and Muckle-Wells syndrome (Farasat et al., Arch.Dermatol., v. 144, p. 392-402, 2008). We have also shown that PCI-34051 is effective at reducing IL1b secretion from blood cells of patients with RA and psoriasis (Balasubramanian et al., in preparation, 2009). Thus, HDAC8-specific inhibitors offer a unique therapeutic modality in treatment of these autoimmune disorders.

Please see our website for additional information and further explanations.

Motexafin Gadolinium (MGd)

MGd is a radiation and chemotherapy sensitizing agent with a novel mechanism of action. MGd is designed to accumulate selectively in cancer cells. Once inside cancer cells, MGd in combination with radiation induces apoptosis (programmed cell death) by disrupting redox-dependent pathways. We believe MGd has the potential to be used for treating many types of cancer in combination with other treatments such as chemotherapy, targeted therapy or radiation therapy. MGd is also detectable by magnetic resonance imaging (MRI) and may allow for more precise tumor detection. The Division of Cancer Treatment and Diagnosis of the NCI, has sponsored eight Phase I studies, one Phase I/II study, and one Phase II study, for evaluation which have and continue to provide valuable developmental insights and directions.

MGd Localization in Brain Metastases



We are currently evaluating MGd in glioblastoma multiforme (GBM), wherein proof-of-efficacy relies on extending survival time. GBM is the most common primary brain tumor in adults accounting for 40% of primary central nervous system tumors. Radiation increases median survival by approximately 4 to 9 months, addition of temozolomide increases this to 14 months, but despite numerous studies of other potential therapies, the outcome of GBM has not changed beyond this. Previous collaborators, led by Dr. Judith Ford (Int. J. Rad. Oncol. Biol. Phys pp 1-8, 2007), showed that in a case matched analysis, patients treated with MGd (n=31) had a median survival of 16.1 months compared to the matched RTOG (Radiation Therapy Oncology Group) database patients with a median survival of 11.8 months. MGd is currently in a RTOG sponsored Phase II multi-center study in GBM in combination with radiation therapy and temozolomide (www.clinicaltrials.gov; 113 patients study). The principal investigator, Dr. David G. Brachman, is heading this study at the Barrow Neurological Institute at St. Joseph's Hospital in Phoenix, AZ, and anticipates accrual to be complete by June '09. Previous studies in malignant gliomas headed by Dr. William Shapiro from the Barrow Institute have shown that the combination of MGd and temozolomide has no additional overlapping toxicities when used in combination. MGd is also in a Children's Oncology Group (COG) sponsored Phase II study in children with pontine gliomas in combination with radiation therapy (www.clinicaltrials.gov; 60 patients). The principal investigator, Dr. Kristin A. Bradley is heading this multi-center study at the University of Wisconsin. The study has completed enrollment.

Pharmacyclics is also evaluating the use of MGd as an inhibitor of the DNA enzyme ribonucleotide reductase as shown by investigators at the Karolinska Institute (Hashemy, S.I., Ungerstedt, J.S., Avval, F.Z. Holmgren H J. Biol Chem 281:10961, 2006). Ribonucleotide reductase is a validated cancer target by itself and inhibition of this target with the use of hydroxyurea has shown activity in a wide variety of human tumors. However, hydroxyurea is relatively nonspecific and toxic. Recently independent investigators have shown that ribonucleotide reductase is critical to gemcitabine resistance (Bepler, G., Kusmartseva, I., Sharma, S., Gautam, A., Cantor, A., Sharma, A., Simon, G. J Clin Oncology 24:4731-4737, 2006). Gemcitabine is a widely used oncology chemotherapy drug. Pharmacyclics is currently exploring use of MGd in sensitizing tumors to gemcitabine.

Our Business Strategy

The key elements of our business strategy include:

- *Focusing on creating first in class and best in class drugs with validated molecular targets.* We are leveraging our expertise in chemistry and clinical development to create multiple novel drug candidates.
- *Focusing on proprietary drugs that address large markets of unmet medical need for the treatment of oncology and immune mediated diseases.* Although our versatile technology platform can be used to develop a wide range of

pharmaceutical agents, we have focused most of our initial efforts in oncology and immune mediated diseases where we have established strength in preclinical and clinical development.

- *Utilize biomarkers and predictive pharmacodynamic assays wherever possible.* Targeting the right drug to the right patient at the right time with the right dose has the potential to greatly expedite intelligent clinical development and reduce the time, cost and risk of clinical programs.
- *Provide major pharmaceutical companies access to validated drug candidates.* Major pharmaceutical companies have a need for promising drug candidates, which still may require large clinical trials. We focus on satisfying this need for novel, best in class or first in class drugs. A partnership with Pharmacyclics may provide these companies the opportunity to leverage the innovation and excellence of a creative, focused and experienced scientific team.
- *Establish strategic alliances and collaborations.* We own the worldwide rights to our multiple product candidates. At the opportune time in the clinical development path we intend to establish strategic alliances and collaborations for the development and commercialization of our products.
- *Leverage development with outsourcing.* We utilize outside vendors with expertise and capability in manufacturing and clinical development to more efficiently develop our multiple product candidates.
- *Create a large clinical pipeline.* We reduce risk of failure by taking multiple ‘shots on goal’.

We are subject to risks common to pharmaceutical companies developing products, including risks inherent in our research, development and commercialization efforts, preclinical testing, clinical trials, uncertainty of regulatory and marketing approvals, uncertainty of market acceptance of our products, history of and expectation of future operating losses, reliance on collaborative partners, enforcement of patent and proprietary rights, and the need for future capital. In order for a product to be commercialized, we must conduct preclinical tests and clinical trials, demonstrate efficacy and safety of our product candidates to the satisfaction of regulatory authorities, obtain marketing approval, enter into manufacturing, distribution and marketing arrangements, build a U.S. commercial oncology franchise, obtain market acceptance and, in many cases, obtain adequate coverage of and reimbursement for our products from government and private insurers. We cannot provide assurance that we will generate revenues or achieve and sustain profitability in the future.

Results of Operations

Research and Development

	Three Months Ended			Nine Months Ended		
	March 31,		Percent	March 31,		Percent
	2009	2008	Change	2009	2008	Change
Research and development expenses	\$ 4,626,000	\$ 5,120,000	-10%	\$ 10,815,000	\$ 14,822,000	-27%

The decrease of 10% or \$494,000 in research and development expenses for the three months ended March 31, 2009, as compared to the three months ended March 31, 2008, was primarily due to a decrease of \$437,000 in personnel costs due to lower headcount and a decrease of \$599,000 in outside preclinical costs and a decrease of \$141,000 in drug manufacturing costs associated with our HDAC, Btk and Factor VIIa programs, partially offset by an increase of \$1,000,000 in expense associated with the amendment of our agreement with Celera Corporation (see Note 6).

We expect research and development expenses to decline in the fourth quarter of fiscal 2009 as compared to the third quarter of fiscal 2009.

The decrease of 27% or \$4,007,000 in research and development expenses for the nine months ended March 31, 2009, as compared to the nine months ended March 31, 2008, was primarily due to a decrease of \$1,727,000 in personnel costs due to lower headcount and a decrease of \$1,376,000 in drug manufacturing costs and a decrease of \$1,258,000 in outside preclinical costs associated with our HDAC, Btk and Factor VIIa programs, partially offset by an increase of \$1,000,000 in expense associated with the amendment of our agreement with Celera Corporation (see Note 6).

Research and development costs are identified as either directly attributed to one of our research and development programs or as an indirect cost, with only direct costs being tracked by specific program. Direct costs consist of personnel costs directly associated with a program, preclinical study costs, clinical trial costs, and related clinical drug and device development and manufacturing costs, drug formulation costs, contract services and other research expenditures. Indirect costs consist of personnel costs not directly associated with a program, overhead and facility costs and other support service expenses. The following table summarizes our

principal product development initiatives, including the related stages of development for each product, the direct costs attributable to each product and total indirect costs for each respective period. The information in the column labeled “Estimated Completion of Phase” is our estimate of the timing of completion of the current in-process development phase. The actual timing of completion of those phases could differ materially from the estimates provided in the table. For a discussion of the risks and uncertainties associated with the timing and cost of completing a product development phase, see Part II, Item 1A, “Risk Factors.”

Prior to fiscal 1999, we did not track our research and development expenses by specific program and for this reason we cannot accurately estimate our total historical costs on a specific program basis. Direct costs by program and indirect costs are as follows:

Program	Description	Phase of Development	Estimated Completion of Phase	Related R&D Expenses Three Months Ended March 31,		Related R&D Expenses Nine Months Ended March 31,	
				2009	2008	2009	2008
HDAC Inhibitors	Cancer	Phase I/II	Unknown	\$ 1,920,000	\$ 1,122,000	\$ 2,945,000	\$ 2,964,000
Factor VIIa	Cancer	Phase I/II	Unknown	425,000	483,000	1,090,000	1,801,000
Btk Inhibitors	Cancer	Phase I	Unknown	786,000	950,000	2,332,000	2,511,000
MGd	Cancer	Phase II	Unknown	\$ 213,000	\$ 662,000	\$ 892,000	\$ 2,224,000
Total direct costs.....				3,344,000	3,217,000	7,259,000	9,500,000
Indirect costs.....				1,282,000	1,903,000	3,556,000	5,322,000
Total research and development expenses.....				<u>\$ 4,626,000</u>	<u>\$ 5,120,000</u>	<u>10,815,000</u>	<u>\$ 14,822,000</u>

General and Administrative

	Three Months Ended March 31,		Percent Change	Nine Months Ended March 31,		Percent Change
	2009	2008		2009	2008	
General and administrative expenses	\$ 1,789,000	\$ 2,100,000	-15%	\$ 7,102,000	\$ 5,923,000	20%

The decrease of 15% or \$311,000 in general and administrative expenses for the three months ended March 31, 2009, as compared to the three months ended March 31, 2008, was primarily due to a decrease in personnel costs due to lower headcount.

The increase of 20% or \$1,179,000 in general and administrative expenses for the nine months ended March 31, 2009, as compared to the nine months ended March 31, 2008, was primarily due to share-based compensation expense of \$1,795,000 and \$740,000 of severance expenses associated with separation agreements entered into with the company’s former CEO and CFO in September 2008, partially offset by lower personnel costs of \$861,000 due to lower headcount and a reduction of \$477,000 in non-severance related share-based compensation expense.

We expect general and administrative expenses in the fourth quarter of fiscal 2009 to be approximately the same as the third quarter of fiscal 2009.

Interest and Other Income, Net

	Three Months Ended			Percent Change	Nine Months Ended			Percent Change
	March 31,		March 31,					
	2009	2008	2009		2008			
Interest and other income, net	\$ (250,000)	\$ 240,000	-204%	\$ (124,000)	\$ 1,075,000	-112%		

The decrease of 204% or \$490,000 in interest and other income, net for the three months ended March 31, 2009, as compared to the three months ended March 31, 2008, was due to \$250,000 of amortized debt discount related to the Note Payable and a reduction of approximately \$230,000 in interest income due to lower average interest rates and lower investment balances.

The decrease of 112% or \$1,199,000 in interest and other income, net for the nine months ended March 31, 2009, as compared to the nine months ended March 31, 2008, was due to \$250,000 of amortized debt discount related to the Note Payable and a reduction of interest income due to lower average interest rates and lower investment balances.

Liquidity and Capital Resources

Our principal sources of working capital have been private and public equity financings and proceeds from collaborative research and development agreements, as well as interest income.

As of March 31, 2009, we had approximately \$11,465,000 in cash and cash equivalents. Net cash used in operating activities of \$13,079,000 and \$17,657,000 during the nine months ended March 31, 2009 and 2008, respectively, resulted primarily from our net loss, net of share-based compensation expenses, and an increase in accounts payable.

Net cash provided by investing activities of \$4,511,000 in the nine months ended March 31, 2009 consisted primarily of proceeds from maturities and sales of marketable securities, partially offset by purchases of marketable securities. Net cash provided by investing activities of \$21,947,000 in the nine months ended March 31, 2008, consisted primarily of proceeds from maturities and sales of marketable securities.

Net cash provided by financing activities of \$7,773,000 in the nine months ended March 31, 2009 was primarily due to proceeds from \$6,400,000 in loans and \$1,371,000 in proceeds from the sale of approximately 1.5 million shares of common stock. Net cash provided by financing activities of \$48,000 in the nine months ended March 31, 2008, was due to sales of stock under the company's employee stock purchase plan.

In December 2008, we borrowed \$5,000,000 from Robert W. Duggan & Associates. In March 2009, the loan amount was increased to \$6,400,000. See Note 8 for a description of the terms of this loan.

In April 2009, the company signed a collaboration agreement with Servier. On May 11, 2009, Pharmacyclics received the upfront payment of \$10,450,000. Servier contractually withheld 5% from the upfront payment of \$11,000,000 to pay for French withholding tax. Pharmacyclics is due to receive from Servier an additional guaranteed \$4,000,000 for research collaboration over a 24 month period (see Note 13).

In February 2009, the company sold approximately 1.5 million unregistered shares of common stock for an aggregate purchase price of approximately \$1,400,000.

In February 2007, we filed a registration statement on Form S-3 to offer and sell, from time to time, equity, debt securities and warrants in one or more offerings up to a total dollar amount of \$100 million. We may seek to raise funds through additional public offerings in the future but cannot guarantee that such efforts will be successful.

Our future contractual obligations at March 31, 2009 are as follows:

	Operating Lease Commitments	Note Payable
Remaining 3 months of fiscal 2009	\$ 224,000	\$ --
Fiscal 2010.....	763,000	6,400,000
Fiscal 2011	644,000	--
Fiscal 2012.....	330,000	--
Total	<u>\$ 1,961,000</u>	6,400,000
Less unamortized original issue discount.....		(320,000)
Total note payable.....		<u>\$ 6,080,000</u>

Based upon the current status of our product development plans, we believe that our existing cash, cash equivalents and marketable securities, including the Servier upfront payment, will be adequate to satisfy our capital needs through at least March 31, 2010. We expect research and development expenses, as a result of on-going and future clinical trials, to consume a large portion of our existing cash resources. Changes in our research and development plans or other changes affecting our operating expenses may affect actual future consumption of existing cash resources as well. In any event, we will need to raise substantial additional capital to fund our operations in the near future. Currently, we are seeking partnership collaborations to support the clinical development of our product candidates. We may also raise additional funds through the public or private sale of securities, bank debt or otherwise. If we are unable to secure additional funds, whether through partnership collaborations or sale of our securities, we will have to delay, reduce the scope of or discontinue one or more of our product development programs. Our actual capital requirements will depend on many factors, including the following:

- our ability to establish and the scope of any new collaborations;
- the progress and success of clinical trials of our product candidates; and
- the costs and timing of obtaining regulatory approvals.

Our estimate of the period of time through which our financial resources will be adequate to support our operations is a forward-looking statement that involves risks and uncertainties, and actual results could vary materially. The factors described above will impact our future capital requirements and the adequacy of our available funds. We cannot be certain that additional funding will be available on terms attractive to us, or at all. Furthermore, any additional equity financing may be highly dilutive, or otherwise disadvantageous, to existing stockholders and debt financing, if available, may involve restrictive covenants. Collaborative arrangements, if necessary to raise additional funds, may require us to relinquish rights to certain of our technologies, products or marketing territories. Our failure to raise capital when needed and on acceptable terms, would require us to reduce our operating expenses and would limit our ability to respond to competitive pressures or unanticipated requirements to develop our product candidates and to continue operations, any of which would have a material adverse effect on our business, financial condition and results of operations.

Off-Balance Sheet Arrangements

We do not have any off-balance sheet arrangements that have or are reasonably likely to have a current or future material effect on our financial condition, changes in our financial condition, revenues or expenses, results of operations, liquidity, capital expenditures or capital resources.

Critical Accounting Policies, Estimates and Judgments

This discussion and analysis of financial condition and results of operations is based upon our financial statements, which have been prepared in accordance with accounting principles generally accepted in the United States of America. The preparation of these financial statements requires us to make estimates and judgments that affect the reported amounts of assets, liabilities, revenues and expenses, and related disclosures. On an on-going basis, we evaluate our estimates, including those related to revenue recognition and clinical trial accruals. We base our estimates on historical experience and on various other factors that we believe are reasonable under the circumstances, the results of which form the basis for making judgments about the carrying values of assets and liabilities that are not readily apparent from other sources. Actual results, however, may differ significantly from these estimates under different assumptions or conditions and may adversely affect the financial statements.

We believe the following critical accounting policies reflect the more significant judgments and estimates used in the preparation of our financial statements and accompanying notes.

Revenue Recognition

Revenues are recognized when persuasive evidence of an arrangement exists, title has transferred or services have been rendered, the price is fixed and determinable and collectibility is reasonably assured. License revenue is typically recognized over the term of the arrangement and milestone revenue is recognized when earned as evidenced by achievement of the specified milestone and the absence of any on-going obligation. License, milestone, contract and grant revenues are not subject to repayment. Any amounts received in advance of performance are recorded as deferred revenues.

Cash Equivalents and Marketable Securities

We maintain investment portfolio holdings of various issuers, types and maturities. We consider all highly liquid investments purchased with original maturities of three months or less to be cash equivalents. At March 31, 2009, all of our investment securities are classified as available-for-sale as part of cash equivalents. Management assesses whether declines in the fair value of investment securities are other than temporary. If the decline in fair value is judged to be other than temporary, the cost basis of the individual security is written down to fair value and the amount of the write down is included in the statement of operations. In determining whether a decline is other than temporary, management considers the following factors:

- Length of the time and the extent to which the market value has been less than cost;
- The financial condition and near-term prospects of the issuer; and
- Our intent and ability to retain our investment in the issuer for a period of time sufficient to allow for any anticipated recovery in market value.

To date we have had no declines in fair value that have been identified as other than temporary.

Research and Development Expenses and Accruals

Research and development expenses include personnel and facility-related expenses, outside contracted services including clinical trial costs, manufacturing and process development costs, research costs and other consulting services. Research and development costs are expensed as incurred. In instances where we enter into agreements with third parties for clinical trials, manufacturing and process development, research and other consulting activities, costs are expensed upon the earlier of when non-refundable amounts are due or as services are performed. Amounts due under such arrangements may be either fixed fee or fee for service, and may include upfront payments, monthly payments, and payments upon the completion of milestones or receipt of deliverables.

Our cost accruals for clinical trials are based on estimates of the services received and efforts expended pursuant to contracts with numerous clinical trial centers and clinical research organizations. In the normal course of business we contract with third parties to perform various clinical trial activities in the on-going development of potential products. The financial terms of these agreements are subject to negotiation and vary from contract to contract and may result in uneven payment flows. Payments under the contracts depend on factors such as the achievement of certain events, the successful enrollment of patients, the completion of portions of the clinical trial or similar conditions. The objective of our accrual policy is to match the recording of expenses in our financial statements to the actual services received and efforts expended. As such, expense accruals related to clinical trials are recognized based on our estimate of the degree of completion of the event or events specified in the specific clinical study or trial contract.

Share-Based Compensation

We adopted SFAS 123R, *Share-Based Payments*, effective beginning July 1, 2005 using the modified prospective application transition method. The modified prospective application transition method requires that companies recognize compensation expense on new share-based payment awards and existing share-based payment awards that are modified, repurchased, or cancelled after the effective date. Additionally, compensation cost of the portion of awards of which the requisite service has not been rendered that are outstanding as of the July 1, 2005 has been expensed as the requisite service was rendered.

Options granted pursuant to our 2004 Equity Incentive Award Plan vest upon the passage of time or a combination of time and the achievement of certain performance obligations. The Compensation Committee of the Board of Directors will determine if the performance conditions have been met. Share-based compensation expense for the options with performance obligations will be recorded when management believes that the vesting of these options is probable.

The fair value of each stock option is estimated on the date of grant using the Black-Scholes valuation model. Expected volatility is based on historical volatility data of the company's stock. The expected term of stock options granted is based on historical data and represents the period of time that stock options are expected to be outstanding. The expected term is calculated for and applied to one group of stock options as the company does not expect substantially different exercise or post-vesting termination behavior among its employee population. The risk-free interest rate is based on a zero-coupon United States Treasury bond whose maturity period equals the expected term of the company's options.

Item 3. Quantitative and Qualitative Disclosures About Market Risk

Our exposure to interest rate risk relates primarily to our investment portfolio. Fixed rate securities may have their fair market value adversely impacted due to fluctuations in interest rates, while floating rate securities may produce less income than expected if interest rates fall. Due in part to these factors, our future investment income may fall short of expectations due to changes in interest rates or we may suffer losses in principal if forced to sell securities that have declined in market value due to changes in interest rates. The primary objective of our investment activities is to preserve principal while at the same time maximizing yields without significantly increasing risk. To achieve this objective, we invest in debt instruments of the U.S. Government and its agencies and high-quality corporate issuers, and, by policy, restrict our exposure to any single corporate issuer by imposing concentration limits. To minimize the exposure due to adverse shifts in interest rates, we maintain investments at an average maturity of generally less than two years. Assuming a hypothetical increase in interest rates of one percentage point, the fair value of our total investment portfolio as of March 31, 2009 would have declined by approximately \$5,000.

Item 4. Controls and Procedures

(a) *Evaluation of disclosure control and procedures:* As required by Rule 13a-15 under the Securities Exchange Act of 1934, as of the end of the third fiscal quarter of 2009, we carried out an evaluation of the effectiveness of the design and operation of our disclosure controls and procedures. This evaluation was carried out under the supervision and with the participation of our management, including our Chief Executive Officer and Vice President, Finance and Administration (Principal Accounting and Financial Officer). Based upon that evaluation, our Chief Executive Officer and Principal Accounting and Financial Officer concluded that our disclosure controls and procedures are adequate and effective to ensure that information required to be disclosed in our reports filed or submitted under the Exchange Act is recorded, processed, summarized, and reported, within the time periods specified in the Securities and Exchange Commission's rules and forms. Disclosure controls and procedures include, without limitation, controls and procedures designed to ensure that information required to be disclosed in our reports filed under the Exchange Act is accumulated and communicated to management, including our Chief Executive Officer and Principal Accounting and Financial Officer, as appropriate to allow timely decisions regarding required disclosure.

(b) *Changes in internal control over financial reporting:* There has been no change in our internal control over financial reporting during our most recent fiscal quarter that has materially affected, or is reasonably likely to materially affect, our internal controls over financial reporting.

PART II. OTHER INFORMATION

Item 1. Legal Proceedings

Not Applicable.

Item 1A. Risk Factors

In addition to the other information set forth in this report, you should carefully consider the factors discussed in Part I, "Item 1A. Risk Factors" in our Annual Report on Form 10-K for the fiscal year ended June 30, 2008, which have not materially changed other than as set forth below. Those risks, which could materially affect our business, financial condition or future results, are not the only risks we face. Additional risks and uncertainties not currently known to us or that we currently deem to be immaterial also may materially adversely affect our business, financial condition and/or operating results.

We will need substantial additional financing and we may have difficulty raising needed capital in the future.

We have expended and will continue to expend substantial funds to complete the research, development and clinical testing of our products. We are unable to entirely fund these efforts with our current financial resources. Currently, we are actively seeking partnership collaborations to help fund the development of our product candidates. On April 16, 2009, we entered into a partnership with Servier. On May 11, 2009, Pharmacyclics received the upfront payment of \$10.45 million. Pharmacyclics will also receive an additional guaranteed \$4 million for research collaboration over a 24 month period, paid in equal increments every 6 months with the

initial payment due October 1, 2009. We may also raise additional funds through the public or private sale of securities, bank debt or otherwise.

If we are unable to secure additional funds, whether through additional partnership collaborations or sale of our securities, we will have to delay, reduce the scope of or discontinue one or more of our product development programs. Based upon the current status of our product development plans, we believe that our cash, cash equivalents and marketable securities will be adequate to satisfy our capital needs through at least March 31, 2010. We may, however, choose to raise additional funds before then. Our actual capital requirements will depend on many factors, including:

- our ability to establish new partnership collaboration arrangements and the timing of such arrangements;
- continued progress of our research and development programs;
- our ability to establish and maintain collaborative arrangements with third parties;
- progress with preclinical studies and clinical trials;
- the time and costs involved in obtaining regulatory approval;
- the costs involved in preparing, filing, prosecuting, maintaining and enforcing patent claims;
- the amount and timing of capital equipment purchases; and
- competing technological and market developments.

In addition, our ability to raise additional capital may be dependent upon our stock being quoted on the NASDAQ Global Market. In the past, our stock price has fallen below the \$1.00 minimum bid price requirement for continued listing set forth in NASDAQ Marketplace Rule 4450(a)(5). While we have since regained compliance with Marketplace Rule 4450(a)(5), we can not assure you that our stock price will continue to remain above the required minimum bid price. If we do not remain in compliance with the \$1.00 minimum bid price requirement or any other NASDAQ listing requirement, our stock may be delisted by NASDAQ.

To maintain our listing on the NASDAQ Global Market, we are required, among other things, to either maintain stockholders' equity of at least \$10 million or a market value of at least \$50 million. We currently do not satisfy either of these requirements. The company received a deficiency notice from NASDAQ in February 2009. The company replied to the notice and received an extension until May 15, 2009 to regain compliance with either NASDAQ's stockholder equity or market value rules. While the company is exploring alternatives to regain compliance, there can be no assurance that it will be able to regain compliance by May 15, 2009. The company is also exploring the alternative of listing its shares on the NASDAQ Capital Market whose requirements the company believes it currently satisfies.

We also expect to raise any necessary additional funds through the public or private sale of securities, bank debt financings, collaborative arrangements with corporate partners or other sources that may be highly dilutive or otherwise disadvantageous, to existing stockholders or subject us to restrictive covenants. In addition, in the event that additional funds are obtained through arrangements with collaborative partners or other sources, such arrangements may require us to relinquish rights to some of our technologies, product candidates or products under development that we would otherwise seek to develop or commercialize ourselves. Additional funds may not be available on acceptable terms, if at all. Our failure to raise capital when needed and on acceptable terms would require us to reduce our operating expenses, delay or reduce the scope of or eliminate one or more of our research or development programs and would limit our ability to respond to competitive pressures or unanticipated requirements and to continue operations. Any one of the foregoing would have a material adverse effect on our business, financial condition and results of operations.

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

On February 19, 2009, the company sold approximately 1.5 million shares of common stock, at \$0.93 per share for an aggregate purchase price of approximately \$1.4 million. The purchasers of the shares were certain foreign and U.S. individuals and entities of which some are shareholders of Pacific Biopharma Group, Ltd. ("PBG"), a Cayman Islands company located in San Bruno California and Taizhou, Jiangsu Province, People's Republic of China. Each investor acted individually and did not purchase shares for the account of PBG or any other affiliated company. Glenn Rice, our President and Chief Operating Officer, is a principal of PBG but did not participate in the transaction.

The sale of the Shares as described above were deemed to be exempt from registration pursuant to Section 4(2) of the Securities Act of 1933, as amended, and Regulation S promulgated thereunder ("Regulation S"), as transactions by an issuer not involving a public offering. The recipients of the Shares represented their intention to acquire the securities for investment only and not with a view to or for sale in connection with any distribution thereof and appropriate legends were affixed to the share certificates and instruments issued in the sale of shares. All recipients either received adequate information about the Registrant or had adequate access, through their relationship with the Registrant, to information about the Registrant. In addition, the Non-U.S. investors also represented to the Registrant all appropriate representations required by Regulation S and the Registrant complied with all appropriate provisions of Regulation S.

The company intends to use the proceeds from the sale of the shares for working capital and general corporate purposes.

Item 3. Defaults Upon Senior Securities

Not Applicable.

Item 4. Submission of Matters to a Vote of Security Holders

Not Applicable.

Item 5. Other Information

Not Applicable.

Item 6. Exhibits

- 4.1 Form of Stock Purchase Agreement related to the February 19, 2009 sale of approximately 1.5 million shares of the Company's common stock to certain foreign and U.S. individuals and entities of which some are shareholders of Pacific Biopharma Group, Ltd.
- 10.1* Amendment No. 2 to Assignment Agreement by and between Pharmacyclics, Inc. and Celera Corporation dated March 2, 2009.
- 10.2* Amendment No. 3 to Assignment Agreement by and between Pharmacyclics, Inc. and Celera Corporation dated March 30, 2009.
- 10.3 Amendment No. 1 to Loan Agreement entered into between the Company and Robert W. Duggan & Associates dated as of March 31, 2009.
- 10.4 Offer letter dated February 2, 2009 by and between the Company and Glenn C. Rice, Ph.D.
- 10.5 Offer letter dated February 5, 2009 by and between the Company and Rainer M. Erdtmann.
- 10.6 Offer letter dated February 26, 2009 by and between the Company and Ahmed Hamdy, M.B.B.Ch.
- 31.1 Rule 13a-14(a)/15d-14(a) Certification of Principal Executive Officer.
- 31.2 Rule 13a-14(a)/15d-14(a) Certification of Principal Accounting and Financial Officer.
- 32.1 Section 1350 Certification of Principal Executive Officer and Principal Accounting and Financial Officer.

* Confidential treatment has been requested as to certain portions of this agreement.

Signatures

Pursuant to the requirements of the Securities Exchange Act of 1934, as amended (the "Exchange Act"), the registrant has duly caused this report to be signed on its behalf by the undersigned thereunto duly authorized.

Pharmacyclics, Inc.

(Registrant)

Dated: May 12, 2009

By: /s/ ROBERT W. DUGGAN

Robert W. Duggan

Chairman of the Board and Chief Executive Officer

Dated: May 12, 2009

By: /s/ RAINER M. ERDTMANN

Rainer M. Erdtmann

*Vice President, Finance and Administration (Principal
Accounting and Financial Officer) and Secretary*

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