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

FORM 10-Q

**[X] QUARTERLY REPORT PURSUANT TO SECTION 13 OR 15(d) OF THE SECURITIES
EXCHANGE ACT OF 1934**

For the quarterly period ended December 31, 2008

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 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 January 31, 2009, there were 26,049,846 shares of the registrant's Common Stock, par value \$0.0001 per share, outstanding.

This quarterly report on Form 10-Q consists of 36 pages of which this is page 1. The Exhibits Index page immediately follows page 35.

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)

	December 31,	June 30,
	2008	2008
ASSETS		
Current assets:		
Cash and cash equivalents	\$ 12,917	\$ 12,260
Marketable securities	--	4,495
Prepaid expenses and other current assets	759	401
Total current assets	13,676	17,156
Property and equipment, net	537	688
Other assets	290	523
	<u>\$ 14,503</u>	<u>\$ 18,367</u>
LIABILITIES AND STOCKHOLDERS' EQUITY		
Current liabilities:		
Accounts payable	\$ 833	\$ 1,055
Accrued liabilities	1,069	796
Note payable to related party	4,500	--
Total current liabilities	6,402	1,851
Deferred rent	69	71
Total liabilities	6,471	1,922
Stockholders' equity:		
Common stock	3	3
Additional paid-in capital	358,856	355,883
Accumulated other comprehensive income	--	10
Deficit accumulated during development stage.....	(350,827)	(339,451)
Total stockholders' equity	8,032	16,445
	<u>\$ 14,503</u>	<u>\$ 18,367</u>

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		Six Months Ended		Period From
	December 31,		December 31,		Inception
	2008	2007	2008	2007	(April 19, 1991)
					through
					December 31,
					2008
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*	2,986	4,462	6,189	9,702	318,111
General and administrative*	1,874	1,756	5,313	3,823	81,454
Purchased in-process research and development	<u>--</u>	<u>--</u>	<u>--</u>	<u>--</u>	<u>6,647</u>
Total operating expenses	<u>4,860</u>	<u>6,218</u>	<u>11,502</u>	<u>13,525</u>	<u>406,212</u>
Loss from operations	(4,860)	(6,218)	(11,502)	(13,525)	(392,203)
Interest and other income, net	<u>26</u>	<u>358</u>	<u>126</u>	<u>835</u>	<u>41,376</u>
Net loss	<u>\$ (4,834)</u>	<u>\$ (5,860)</u>	<u>\$ (11,376)</u>	<u>\$ (12,690)</u>	<u>\$ (350,827)</u>
Basic and diluted net loss per share	<u>\$ (0.19)</u>	<u>\$ (0.23)</u>	<u>\$ (0.44)</u>	<u>\$ (0.49)</u>	
Shares used to compute basic and diluted net loss per share	<u>26,034</u>	<u>25,968</u>	<u>26,025</u>	<u>25,977</u>	

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

Research and development	\$ 173	\$ 247	\$ 352	\$ 555	\$ 6,328
General and administrative	461	352	2,099	721	8,646

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)

	Six Months Ended December 31,		Period From Inception (April 19, 1991) through December 31, 2008
	2008	2007	
Cash flows from operating activities:			
Net loss	\$ (11,376)	\$ (12,690)	\$ (350,827)
Adjustments to reconcile net loss to net cash used in operating activities:			
Depreciation and amortization	166	178	15,151
Amortization of premium/discount on marketable securities, net.....	(33)	(94)	(27)
Purchased in-process research and development	--	--	4,500
Share-based compensation expense	2,451	1,276	14,974
Loss (gain) on sale of marketable securities	(1)	(7)	50
Write-down of fixed assets	--	--	381
Changes in assets and liabilities:			
Prepaid expenses and other assets	(125)	33	(1,049)
Accounts payable	(222)	(386)	833
Accrued liabilities	273	(106)	1,069
Deferred rent	(2)	(3)	69
Net cash used in operating activities	<u>(8,869)</u>	<u>(11,799)</u>	<u>(314,876)</u>
Cash flows from investing activities:			
Purchase of property and equipment	(15)	(20)	(12,300)
Proceeds from sale of property and equipment	--	--	112
Purchases of marketable securities	(3,179)	--	(532,725)
Proceeds from maturities and sales of marketable securities	7,698	18,994	532,702
Net cash provided by (used in) investing activities	<u>4,504</u>	<u>18,974</u>	<u>(12,211)</u>
Cash flows from financing activities:			
Issuance of common stock, net of issuance costs	20	48	308,938
Exercise of stock options	2	--	6,433
Proceeds from notes payable	5,000	--	8,000
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>5,022</u>	<u>48</u>	<u>340,004</u>
Increase in cash and cash equivalents	657	7,223	12,917
Cash and cash equivalents at beginning of period	12,260	11,941	--
Cash and cash equivalents at end of period	<u>\$ 12,917</u>	<u>\$ 19,164</u>	<u>\$ 12,917</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 pharmaceutical company leveraging our small-molecule drug development expertise to build a pipeline in oncology and immune mediated diseases based on novel targets, pathways, and mechanisms. 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 product revenues until we receive the necessary regulatory and marketing approvals and launch one of our products, if at all.

Our strategy is to identify promising product candidates, to develop them in a rapid cost effective manner, and to seek development and for commercialization partners as appropriate to complement our internal efforts.

We have incurred significant operating losses since our inception in 1991, and as of December 31, 2008, had an accumulated deficit of approximately \$350.8 million. Based upon the current status of our product development and plans, we believe that our existing cash, cash equivalents and marketable securities will be adequate to satisfy our capital needs through at least the next six months. 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 future. Currently, we are actively seeking partnership collaborations for our product candidates. We also expect to 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 progress and success of preclinical studies and clinical trials of our product candidates; and
- the costs and timing of obtaining regulatory approvals.

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 5,866,066 and 4,987,264 shares of common stock were outstanding at December 31, 2008 and 2007, 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 components of share-based compensation recognized in the company's statements of operations for the three and six months ended December 31, 2008 and 2007 and since inception are as follows:

	Three Months Ended December 31,		Six Months Ended December 31,		Period From Inception (April 19, 1991) through December 31,
	2008	2007	2008	2007	2008
Research and development	\$ 173,000	\$ 247,000	\$ 352,000	\$ 555,000	\$ 6,328,000
General and administrative	461,000	352,000	2,099,000	721,000	8,646,000
Total share-based compensation	<u>\$ 634,000</u>	<u>\$ 599,000</u>	<u>\$ 2,451,000</u>	<u>\$ 1,276,000</u>	<u>\$ 14,974,000</u>

The following table summarizes the company's stock option activity for the six months ended December 31, 2008:

	Number Of Shares	Weighted Average Exercise Price
Balance at June 30, 2008	5,540,544	\$ 8.10
Options granted	663,328	1.71
Options exercised	(1,250)	0.86
Options forfeited	(336,556)	11.17
Balance at December 31, 2008	<u>5,866,066</u>	7.20

At December 31, 2008, 3,233,717 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 six months period ended December 31, 2008 and 2007 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 December 31, 2008.

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 losses were as follows:

	Three Months Ended December 31,		Six Months Ended December 31,	
	2008	2007	2008	2007
Net loss	\$ (4,834,000)	\$ (5,860,000)	\$ (11,376,000)	\$ (12,690,000)
Change in net unrealized losses on available-for-sale securities	8,000	30,000	(10,000)	27,000
Comprehensive loss	<u>\$ (4,826,000)</u>	<u>\$ (5,830,000)</u>	<u>\$ (11,386,000)</u>	<u>\$ (12,663,000)</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 for other 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 December 31, 2008:

	Fair Value as of December 31, 2008	Basis of Fair Value Measurements		
		Level 1	Level 2	Level 3
Money market funds	\$ 1,015,000	\$ 1,015,000	\$ -	\$ -
Corporate bonds	-	-	-	-
Government securities	6,099,000	-	6,099,000	-
Commercial paper	-	-	-	-
Total cash equivalents	<u>\$ 7,114,000</u>	<u>\$ 1,015,000</u>	<u>\$ 6,099,000</u>	<u>\$ -</u>

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

As of December 31, 2008	Cost Basis	Unrealized Gain	Unrealized Loss	Fair Value
Money market funds	\$ 1,015,000	\$ -	\$ -	\$ 1,015,000
Corporate bonds	-	-	-	-
Government securities	6,099,000	-	-	6,099,000
Commercial paper	-	-	-	-
	<u>7,114,000</u>	<u>-</u>	<u>-</u>	<u>7,114,000</u>
Less cash equivalents	<u>(7,114,000)</u>	<u>-</u>	<u>-</u>	<u>(7,114,000)</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 – 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 7 – Note Payable

On December 30, 2008, the company borrowed \$5,000,000 from Robert W. Duggan & Associates ("RWD"). Under the terms of the unsecured loan, the company is to pay RWD the principal sum of \$5,000,000 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 loan has 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 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 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.

The accretion of the discount will cause an increase in the carrying amount of indebtedness and charges to interest expense from December 31, 2008 to June 30, 2009 of \$500,000.

Note 8 – 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 December 31, 2008, 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 9 – Recent Accounting Pronouncements

In December 2007, the FASB issued SFAS No. 141 (revised 2007), *"Business Combinations"* (SFAS No. 141(R)), which replaces SFAS No. 141. SFAS No. 141(R) establishes principles and requirements for how an acquirer recognizes and measures in its financial statements the identifiable assets acquired, the liabilities assumed, any non-controlling interest in the acquiree and the goodwill acquired. SFAS No. 141(R) also establishes disclosure requirements which will enable users to evaluate the nature and financial effects of the business combination. SFAS No. 141(R) is effective for business combinations for which the acquisition date is on or after the beginning of the first annual reporting period beginning on or after December 15, 2008. The company is currently evaluating the impact of the adoption of SFAS No. 141(R) on its financial statements.

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.

Note 10 – Related Party Transaction

As discussed in Note 7 – Note Payable, on December 30, 2008, the company borrowed \$5,000,000 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 29% of the company's outstanding common stock.

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

Our mission is to design, develop, and bring into commercialization novel small-molecule therapies intended to treat serious disease and improve quality of life and duration of life for patients.

Pharmacyclics is an innovative development stage biopharmaceutical company with four drug candidates in clinical development and several late stage preclinical programs. Each of these proprietary drug candidates targets large unmet medical needs in cancer and autoimmune disease.

We are committed to high standards of ethics, scientific rigor, and efficiency as we move each of these programs to commercialization.

Our goal is to identify promising product candidates based on exceptional scientific and development expertise, develop them in a rapid, cost-effective manner, and to seek development and/or commercialization partners particularly for ex-US markets as appropriate to complement our internal efforts.

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 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 December 31, 2008, had an accumulated deficit of approximately \$350.8 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 generates 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, alone or with others, to successfully complete the development of our products under development, 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 clinical drug candidates under development and a preclinical drug candidate under development, and a lead compound undergoing preclinical optimization. This includes a histone deacetylase inhibitor (PCI- 24781) currently in Phase 1 clinical trials; an inhibitor of Factor VIIa (PCI-27483) soon to be in a Phase 1b clinical trial; an inhibitor of Bruton's tyrosine kinase (Btk) (PCI-32765) currently in a Phase 1 clinical trial targeting oncology applications; a Btk inhibitor in advanced preclinical testing (PCI-45261) 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) has completed two Phase 3 trials in patients with brain metastases from non-small-cell lung cancer (NSCLC) and other cancers and is now in a Phase 2 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:

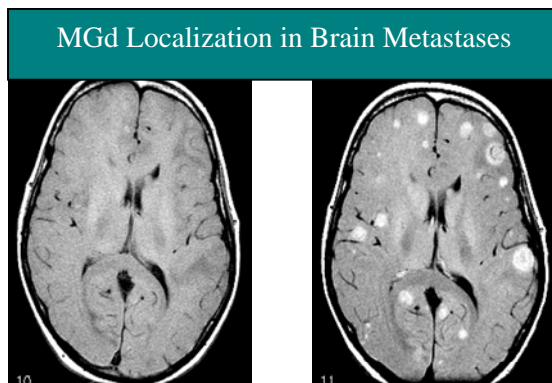
Product Candidate	Disease Indication	Development Status(1)
MGd		
With Radiation	Brain metastases from lung cancer	Phase 3 - complete
	Primary brain tumor ²	Phase 2 - enrolling
	Childhood brain tumors ²	Phase 2 - complete
	Brain metastases with stereotactic radiosurgery	Phase 2 - complete
PCI-24781 (HDAC Inhibitor)	Advanced solid tumors	Phase 1 - enrolling
	Recurrent B cell lymphomas	Phase 1 - enrolling
	Recurrent B cell lymphomas	Phase 2 - planned Q2 '09
	Sarcoma	Phase 1b/2a - planned Q2 '09
PCI-27483 volunteers) (Factor VIIa Inhibitor)	Cancer therapy	Phase 1 - complete (normal
		Phase 1b/2a -planned Q2 '09
PCI-32765 (B cell tyrosine kinase inhibitor)	Lymphomas	Phase 1 -- enrolling
PCI-45261 (B cell tyrosine kinase inhibitor)	Rheumatoid arthritis, Lupus, Mast cell disease	Preclinical
PCI-34051 (HDAC8 inhibitor)	Autoimmune and cancer	Preclinical

1. "Phase 1" means initial human clinical trials designed to establish the safety, dose tolerance and sometimes pharmacokinetics/pharmacodynamics of a compound. "Phase 2" means human clinical trials designed to establish safety, optimal dosage and preliminary activity of a compound. "Phase 3" means human clinical trials designed to lead to accumulation of data sufficient to support a new drug application, including substantial evidence of safety and efficacy.

2. Studies sponsored by the National Cancer Institute.

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 1 studies, one Phase 1/2 study, and one Phase 2 study, for evaluation which have and continue to provide valuable developmental insights and directions.



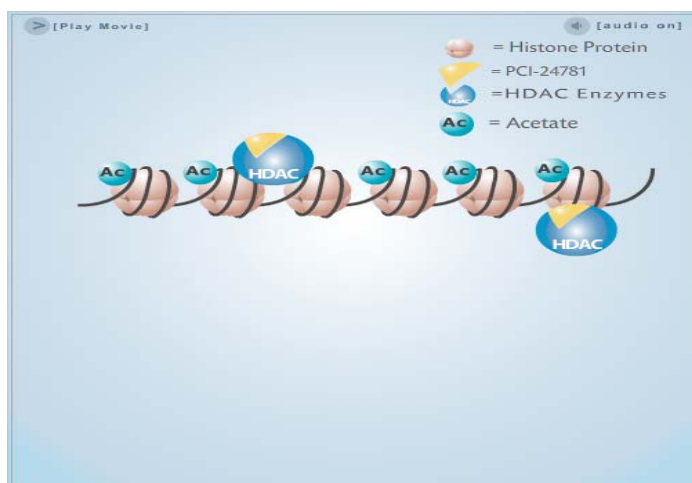
In the previously conducted pivotal SMART trial, (Study of Neurologic Progression with Motexafin Gadolinium and Radiation Therapy), patients given MGd in addition to whole brain radiation therapy (WBRT) had a median time to neurologic progression of 15.4 months, compared to 10.0 months for patients who received only WBRT ($p=0.12$, hazard ratio=0.78), a trend in favor of MGd. In North American patients ($N=348$), where WBRT was delivered more promptly, the median time to neurologic progression was increased from 8.8 months for patients treated with WBRT alone compared to 24.2 months for patients receiving WBRT plus MGd ($P=0.004$, hazard ratio=0.53). We subsequently concluded that prompt delivery of radiation therapy, as typically given in the U.S., is an important factor in treatment effect. In December 2006, we submitted a New Drug Application (NDA) to the Food and Drug Administration (FDA) for the use of MGd in combination with radiation therapy for the treatment of patients with brain metastases from NSCLC. In December 2007, we announced that the FDA determined that our NDA was non-approvable which will require us, among other things, to conduct additional clinical studies and submit that data before the FDA will approve MGd for marketing. We have concluded our review of MGd for the potential treatment of brain metastases from NSCLC and have decided not to pursue this indication.

Additionally, we are 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, and 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. Previously 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 2 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.

Pharmacyclis is also evaluating the use of MGd with stereotactic radiosurgery (SRS). Data presented in the 2007 Annual Meeting of the American Society of Clinical Oncology indicated that MGd may improve the efficacy of SRS by providing more accurate magnetic resonance imaging (MRI) treatment-planning and better defining the treatment field in patients with brain metastases from solid tumors. MGd allowed physicians to identify occult brain metastases in 24.4% of patients that were not previously detected with standard MRI contrast agents and were amenable to stereotactic radiosurgery. Currently Pharmacyclis is determining the potential for development of MGd with SRS.

Histone Deacetylase Inhibitor Program

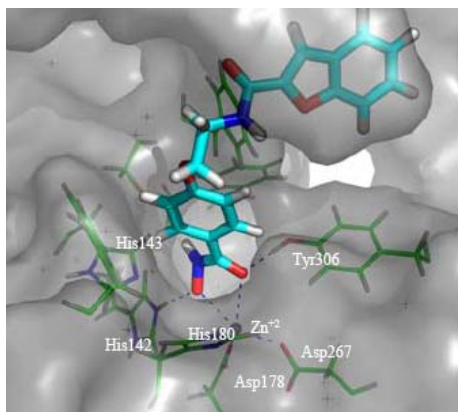
The human genome consists of a complex network of genes which are turned on or off depending on the needs of the cell. Cancer is characterized by genome-wide changes in gene expression within the tumor. These changes favor a tumor's ability to progress through the cell cycle, to avoid apoptosis, or to 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. Histone acetylation, DNA methylation, and other types of chromatin modifications constitute forms of epigenetic regulation that work coordinately to control gene expression in mammalian cells. A well studied histone modification is acetylation and deacetylation, modifications that are catalyzed by a family of enzymes known as histone acetyl transferases (or "HATs"), and histone deacetylases (or "HDACs"). HDAC is a family of eleven isoforms, denoted HDAC 1-11. Histone acetylation alters the transcriptional regulation of a subset of genes, including many tumor suppressors and genes involved in cell cycle control, cell division, and apoptosis. HDAC inhibitors lead to an increased acetylation of histone proteins and are able to restore a gene expression pattern in cancer cells that is more like that of a normal cell. HDAC inhibitors block cancer cell proliferation *in vitro*, and cancer cell growth arrest is observed *in vivo* at non-toxic concentrations.



PCI-24781 (Pan HDAC Inhibitor)

PCI-24781 is a novel, potent (low nM Ki), 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, platinum agents, and poly (ADP-ribose) polymerase inhibitors. 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 cancers. *In vivo* activity is correlated with drug exposure through a predictive measure of acetylation of histones and tubulin in circulating leukocytes and tumor samples.

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 1 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 1, 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, gall bladder, prostate, medullary thyroid and fibrosarcoma.

The second study by the oral route (PCYC-0403) is a Phase 1/2 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 1/2 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. 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. Some thrombocytopenia has been observed with PCYC-24781 but we believe it can be successfully managed through dose scheduling changes. No other grade 3 or higher adverse events have been observed to date.

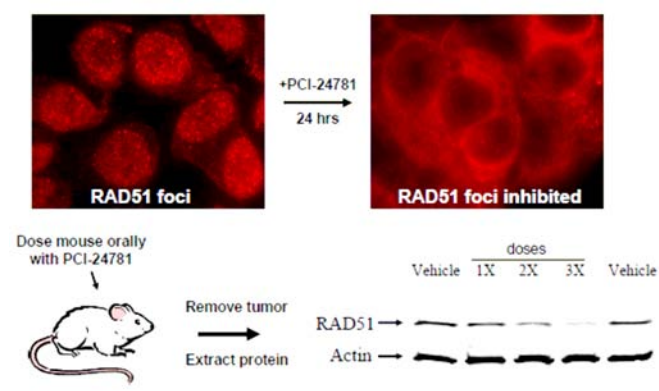
A third clinical study (Phase 2) will test PCI-24781 in combination with doxorubicin in patients with soft tissue sarcoma. This trial will be co-sponsored by investigators at Massachusetts General Hospital and Dana-Farber/Harvard Cancer Center, and is planned to begin in 2Q 2009.

Proprietary Predictive Assays

Following chemotherapy or radiation treatment, some patient's 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 may 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 essential for repair of double-strand breaks (DSB). It was demonstrated by Pharmacyclics that PCI-24781 effectively prevents DSB repair via the homologous recombination pathway by modulation of RAD51 (Adimoolam et al., Proc.Natl.Acad.Sci.U.S.A, v. 104, p. 19482-19487, 2007). 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 such as platinum agents, PARP inhibitors and doxorubicin (Adimoolam et al., Proc.Natl.Acad.Sci.U.S.A, v. 104, p. 19482-19487, 2007). We showed recently that RAD51 is overexpressed in a majority of human lymphoma samples and that pretreatment with PCI-24781 downregulates 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, we have initiated a Phase 1/2 trial of PCI-24781 in combination with doxorubicin for treating sarcoma with Dr. Edwin Choy at Massachusetts General Hospital and Dr. George Demitri at Dana-Farber Cancer Center. 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 neoplastic malignancies, including lymphoma, breast, lung and liver cancer.



Rad51 is a DNA repair gene that Pharmacocyclics scientists have discovered that predicts sensitivity to PCI-24781.

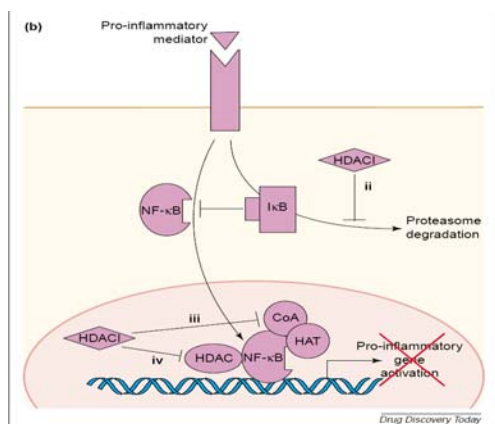
Top: PCI-24781 disrupts nuclear repair foci in colon cancer cells.

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 isoforms have been implicated in many other physiological processes and there is growing interest in using pan- and isoform-specific HDAC inhibitors in many disease areas including metabolic, neurological and immunological disorders as well as for treating bacterial and parasitic infections. For instance, in 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 and sepsis, diabetes and hemorrhagic shock. (Reviewed in Chipoy C. Drug Discov 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 is through the deacetylation of NF-κB subunit p65. This prevents binding to IκB and re-export from nucleus and results in broad inflammatory inhibition.

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

Patents

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

Competition

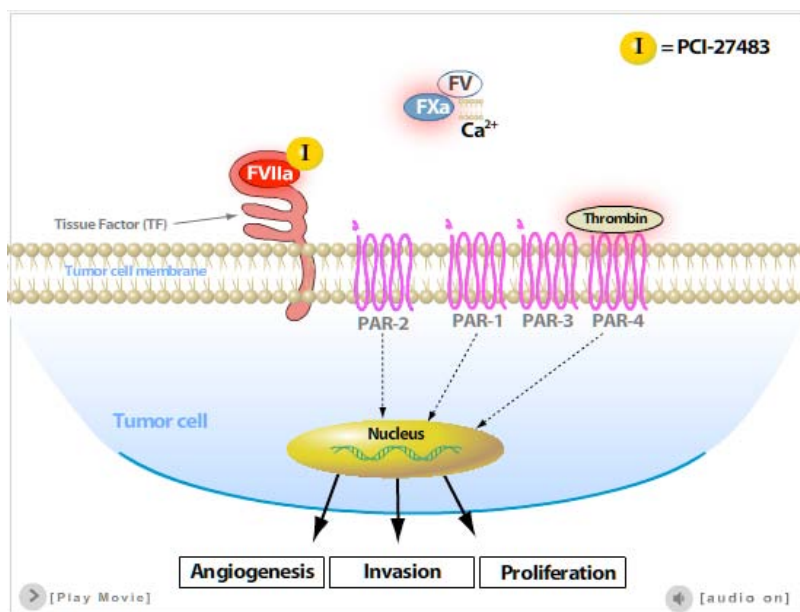
Merck's vorinostat (Zolinza[®]) has been approved by the FDA for cutaneous T-cell lymphoma patients who have progressive, persistent or recurrent disease on or following failure of two systemic therapies, making the oral drug the first in its class to reach the market. A number of structurally distinct HDAC inhibitors are currently in clinical trials including Novartis' LAQ-824 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, cancer, small-cell lung cancer, and melanoma, papillary thyroid carcinoma, and B- and T-cell lymphomas. Thrombocytopenia was identified as a dose-limiting toxicity for patients administered a number of these agents. Several of the competitors have reported cardiovascular toxicities such as Grade 3 QTc prolongation, arrhythmias and atrial fibrillation, in addition to fatigue, anorexia, infection, headache and nausea. To date, significant side effects, (other than reversible thrombocytopenia), have not been observed in clinical studies with PCI-24781 suggesting that PCI-24781 may offer a less toxic modality for the treatment of cancer than its competitors.

Partnering

Through its preclinical and Phase 1 and early Phase 2 development of PCI-24781, Pharmacyclics has built substantial product value by reducing product risk while identifying clinical activity. We believe that a drug in a Phase 2 program is an optimal value and risk/benefit inflection point in which to seek corporate partnerships for co-development. A strategic goal for Pharmacyclics is to retain US sales and marketing rights, while partnering ex-US territories, which management believes will return maximal value for shareholders. Partnering ex-US development and commercialization of PCI-24781 is an important goal for Pharmacyclics in 2009.

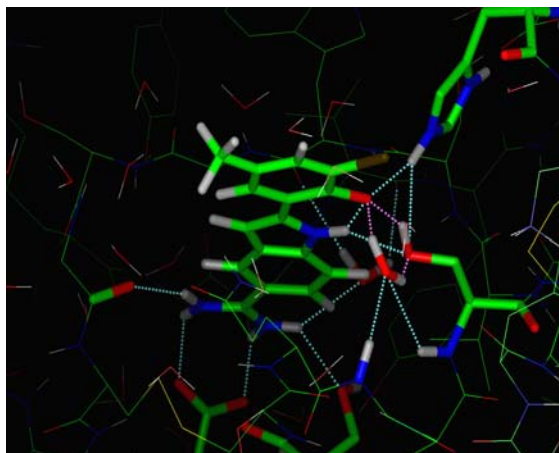
Factor VIIa Inhibitor Program

Factor VII is a serine protease that becomes activated (fVIIa) by binding to tissue factor (TF). 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, but is absent from vascular cells that come in contact with circulating fVII in the blood. Preclinical models of thrombosis 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, stomach or colon, overexpression of tissue factor is associated with an increased incidence of venous thromboembolism. Tissue factor overexpression also correlates with clinical stage and histological grade in a number of human tumor types (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. It is postulated that TF/fVIIa accelerates tumor progression through the PAR family of G-protein-couple receptors. Inhibition of TF/fVIIa activity can lead to inhibition of tumor growth, metastasis, and angiogenesis in tumors where TF is overexpressed. Inhibition of the TF/fVIIa complex may also down regulate VEGF expression, a critical molecule in angiogenesis (blood vessel formation to the tumor).



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 Factor VIIa when it is complexed with a protein called tissue factor (TF). PCI-27483 is an extremely potent inhibitor of factor VIIa (1.6 nM Ki). It is a selective inhibitor with between a 100 to 100,000-fold specificity towards other enzymes involved in hemostasis such as kallikrein-P, Factor XIa, Factor IXa, Thrombin and Factor Xa.

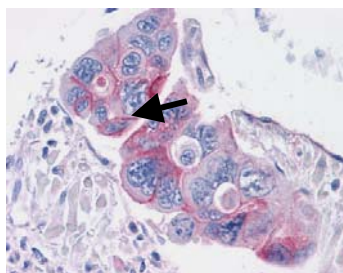


PCI-27483 was developed using rational drug design (Katz, B. A.; *et al. J. Mol. Biol.* **2001**, *307*, 1451-1486) against the target molecule Factor VIIa

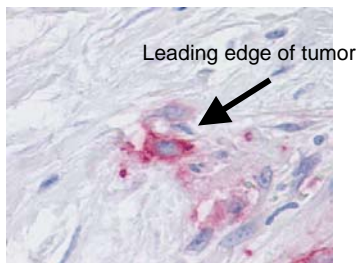
The antithrombotic effects of subcutaneous PCI-27483 administration were determined in a baboon model of arterial thrombosis. Increasing subcutaneous doses of PCI-27483 progressively inhibited platelet deposition and fibrin accumulation, with similar anti-coagulation effects as the low molecular weight heparin 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.

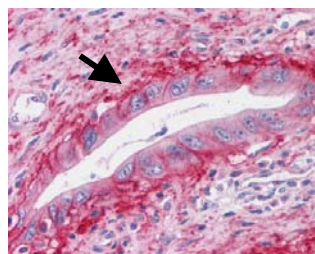
Clinical Program



Malignant Cells 40X



Malignant Cell at Pushing
Margin of Invasion 60X



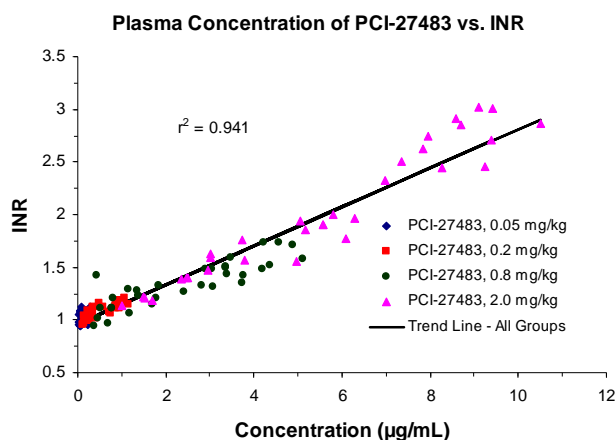
Malignant Cells and
Surrounding
Fibrocollagenous Matrix 40X

FVIIa was detected in 12/13 pancreatic carcinomas by immunohistochemical staining. Staining was detected in malignant cells while all normal epithelial 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 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 in normal 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 and is also associated with VEGF expression, increased microvessel density and angiogenesis, and clinical venous thromboembolism 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 1 testing of PCI-27483 in healthy volunteers. The primary objective of the ascending dose Phase 1 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 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 point of care blood test. A mean peak INR of 2.7 was achieved without adverse effects at the highest dose level administered. A peak INR response between 2 and 3 is being targeted for future clinical studies. INR responses correlated with plasma concentrations of PCI-27483 (see below). 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 heparins Lovenox (4.5 hours) and Fragmin (3 to 5 hours).



Measurements of INR (a measurement of coagulation potential) in patients dosed with PCI-27483 showing excellent predictability.

A multicenter Phase 1b/2a study is planned to begin in May 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

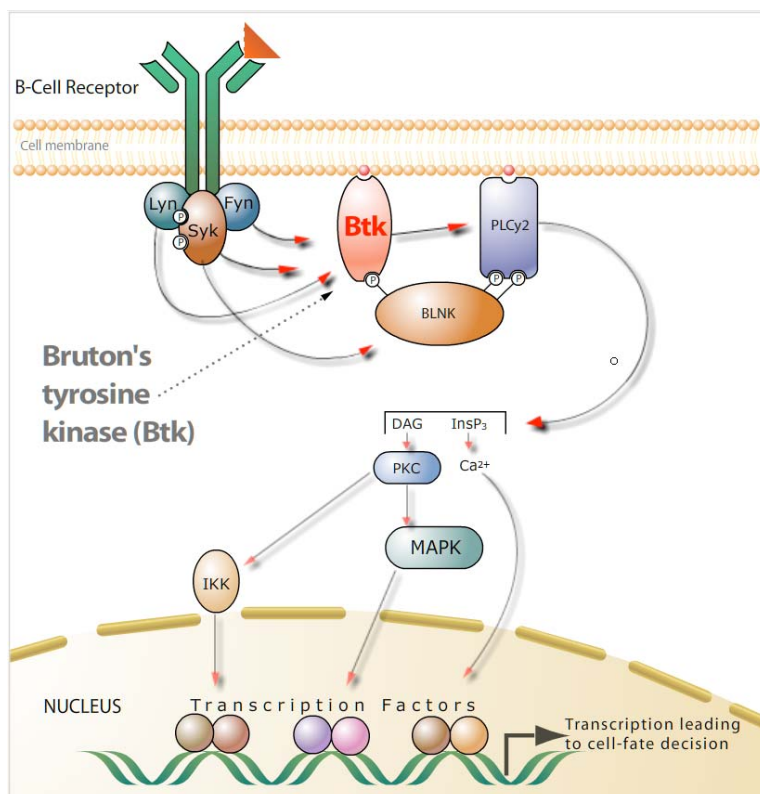
PCI-27483 (as a compound, in pharmaceutical compositions and in uses for treating a variety of diseases) is covered by US patent applications (issued and pending) and PCT national phase patent applications in 14 other jurisdictions, including Europe, Canada, Japan, China, India, South Korea, Australia 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 in ex-US territories following completion of the Phase 2 clinical trial. We believe this unique drug may be competitively positioned for a significant partnership following this clinical milestone if it proves to be successful.

Btk Inhibitors

Pharmacyclics is pioneering the development of orally bioavailable inhibitors of Bruton's tyrosine kinase (Btk), a non-receptor tyrosine kinase (signaling molecule) that is critically important in B and mast cell signaling. 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 result from mutations acquired during normal B-cell development leading to uncontrolled proliferation and B-cell malignancies. Thus the potential clinical indications for a Btk inhibitor are diverse and include non-oncology applications for treatment of rheumatoid arthritis (RA), systemic lupus erythematosus (SLE), and allergic diseases such as eosinophilic esophagitis. 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. Pharmacyclics has developed two programs of proprietary and chemically distinct inhibitors, producing one candidate optimized for oncology (PCI-32765) and currently in Phase 1 clinical trials; and one candidate optimized for autoimmune applications (PCI-45261) which is currently in advanced preclinical development with an anticipated IND in the first half of 2010.



BTK plays a critical role in signaling via B-cell receptor (BCR) signaling as well as FcγR, CD22 and CD19 receptor signaling. Btk inhibitors block B-cell activation and auto-antibody formation.

Genetic Validation of Inhibiting the Target in Humans

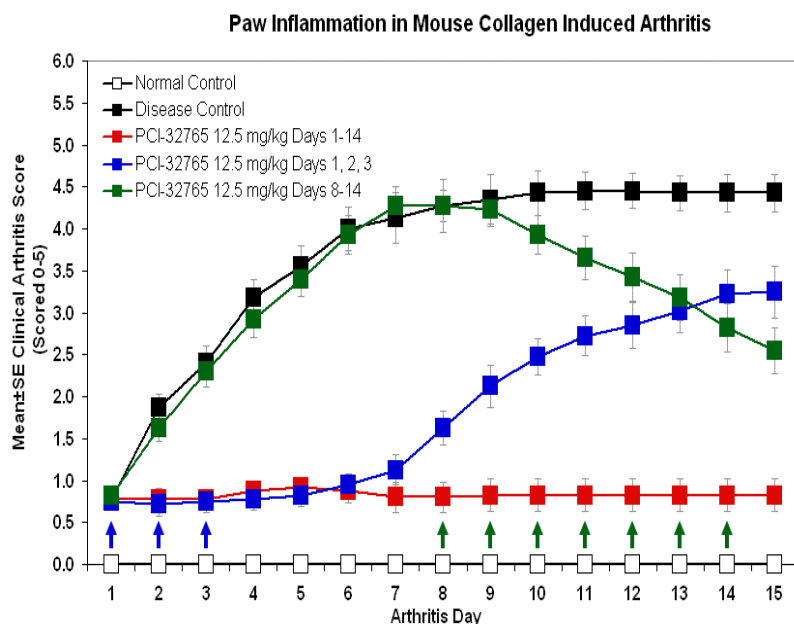
Unlike competing programs of inhibiting B-cell signaling such as with Syk inhibition, a human genetic mutation exists which helps to validate the science, safety and efficacy of a Btk inhibitor. Bruton's agammaglobulinemia is an X-linked disease (only male offspring being effected) occurring in approximately 1 in 250,000 males. In the absence of Btk, B lymphocytes do not differentiate or mature. Males with XLA have a total or almost total absence of B lymphocytes and plasma cells and very low levels of serum Ig. The major block occurs in the development of pro-B-cells to pre-B-cells and then to mature lymphocytes. Patients can have pre-B-cells in the marrow, but they have few, if any, functional (mature) B-cells in the peripheral blood and the lymphoid tissues. Without mature B lymphocytes, antibody-producing plasma cells are absent. Therefore Btk is absolutely necessary for the proliferation and the differentiation of B lymphocytes. Thus we believe that the B-cell selective function of Btk, as confirmed by the B-cell selective pathology in Bruton's agammaglobulinemia, indicates that Btk inhibition is better validated and will be more potent and have fewer side effects than inhibitors targeting other B-cell related kinases (e.g. Syk) in the B-cell signaling pathway. These findings are also observed in the mouse. A point mutation or knockout of Btk causes X-linked immunodeficiency (*xid*), with ~50% fewer conventional B2 B-cells, absent B1 B-cells, and reduced serum immunoglobulin (Ig) levels. In a transgenic mouse in which Btk is expressed at ~25% of wild-type levels, development of B2 B-cell is fully restored, but mature B-cells are still deficient in responding to BCR stimulation. Thus consistent with the human data, mature B-cells may be particularly dependent on Btk for activation.

PCI-32765 for Oncology

We have developed highly selective, small-molecule inhibitors of Btk using a proprietary scaffold and demonstrated oral efficacy in multiple preclinical models of autoimmune disease and lymphoma. Our inhibitors take advantage of a unique and proprietary mechanism to achieve potency and selectivity over other kinases. 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 is cytotoxic to a subset of lymphoma cell lines ($GI_{50} < 1 \mu M$) and *in vivo*, significantly inhibits xenograft tumor growth in the mouse. We have recently initiated a trial of PCI-32765 in spontaneous canine lymphoma in companion animals. Thus far, in six dogs treated with PCI-32765 we have observed two partial responses (by RECIST criteria).

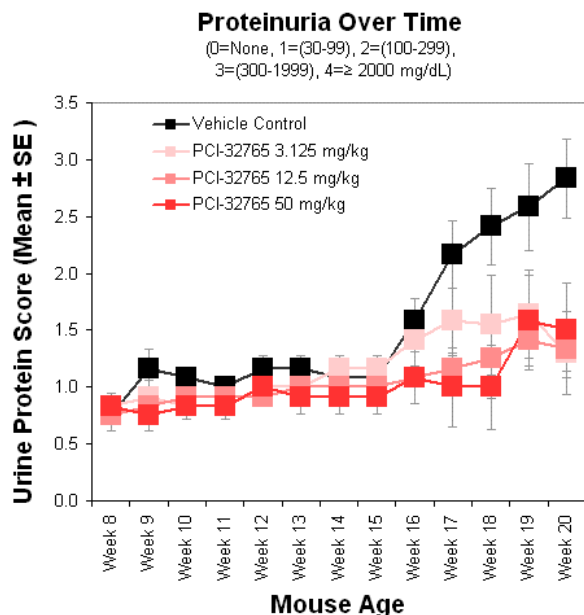
In animal models of rheumatoid arthritis, oral administration of PCI-32765 leads to the regression of established disease. *In vivo*, once daily oral dosing of PCI-32765 inhibited collagen induced arthritis (CIA) in the mouse ($ED_{50} = 4.55 \text{ mg/kg/day}$). In a scheduling study (below), PCI-32765 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 degranulation *in vitro* and inhibits the passive cutaneous anaphylaxis reaction *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 eosinophilic esophagitis as well as allergy.

PCI-32765 also significantly reduces disease severity in the mouse MRL/lpr lupus model, inhibiting proteinuria and anti-dsDNA antibody levels.



Effects of PCI-32765 in MRL/lpr female mice, a preclinical model of lupus. Drug was administered orally starting on week 8. Proteinuria (a measure of disease severity) is dramatically reduced in drug treated animals. Blood nitrogen and antibodies to dsDNA were also dramatically lowered (data not shown).

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 assay that can be used to monitor active-site occupancy of Btk by our inhibitors. We have developed a fluorescent probe that binds to the same site as PCI-32765 and PCI-45261 (see below) and this probe is used to measure Btk occupancy by the drug. 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 PBMC. 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 BCR signaling and mast cell activation *ex vivo* using samples from PCI-32765 treated patients.

A Phase 1 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.

Based on the pharmacodynamic and safety results in the early cohorts of the lymphoma trial, we may initiate a Phase 1 trial in rheumatoid arthritis with PCI-32765 as a proof-of-concept study for Btk inhibition autoimmune diseases.

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

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 were 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. PCI-45261 was selected in December 2008 as a clinical candidate. Btk inhibition by PCI-45261 is >2500-fold selective over the tyrosine kinases EGFR and JAK-3. We have confirmed that orally dosed PCI-45261 is highly efficacious in a murine model of collagen induced arthritis. Relatively low efficacious doses are predicted for humans based on interspecies scaling. IND-enabling studies are underway.

Data to date for PCI-32765 and PCI-45261 demonstrates improvements in signs of inflammation in rheumatoid arthritis models. However, based on the mechanism of action, we expect that this drug, PCI-45261, 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 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

Pharmacyclics will be seeking strategic pharma/biotech partner(s) to develop and commercialize PCI-32765 and PCI-45261 in Japan and Asia. We believe retaining commercialization rights for US and EU markets will add significant shareholder value.

HDAC8-specific inhibitor program: PCI-34051

Pharmacyclics' scientists have been in the forefront of research into HDAC isoform-specific inhibitors beginning with the cloning of the human HDAC8 isoform 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 isoform-specific inhibitor of a human HDAC (PCI-34051, HDAC8-specific inhibitor) in 2008 (Balasubramanian et al., Leukemia., v. 22, p. 1026-1034, 2008), and the first to discover a novel anti-inflammatory activity of an isoform-specific HDAC 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.

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 pan-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 isoforms, 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 phenotypes. 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 cytokines from PBMCs and monocytes (Balasubramanian et al., in preparation 2009). It is particularly effective at modulating 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) and cryopyrinopathies such as 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 PBMC 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.

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 cancer.* 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 autoimmune where we have established strength in preclinical and clinical development.
- *Utilize biomarkers and predictive assays wherever possible.* Targeting the right drug to the right patient and the right time with the right dose greatly expedites intelligent clinical development and reduces the time, cost and risk of clinical programs.
- *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 pipeline.* We reduce risk of failure by taking multiple 'shots on goal'.
- *Establishing strategic alliances.* We own the worldwide rights to our multiple product candidates. We intend to establish strategic alliances for the ex-US development and commercialization of our products at the opportune time.

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		Percent Change	Six Months Ended		Percent Change
	December 31,			December 31,		
	2008	2007		2008	2007	
Research and development expenses	\$ 2,986,000	\$ 4,462,000	-33%	\$ 6,189,000	\$ 9,702,000	-36%

The decrease of 33% or \$1,476,000 in research and development expenses for the three months ended December 31, 2008, as compared to the three months ended December 31, 2007, was primarily due to a decrease of \$695,000 in personnel costs due to lower headcount and a decrease of \$563,000 in drug manufacturing costs.

The decrease of 36% or \$3,513,000 in research and development expenses for the six months ended December 31, 2008, as compared to the six months ended December 31, 2007, was primarily due to a decrease of \$1,290,000 in personnel costs due to lower headcount and a decrease of \$1,235,000 in drug manufacturing costs and a decrease of \$659,000 in outside preclinical costs associated with our HDAC, Btk and Factor VIIa programs.

We expect research and development expenses to increase in the second half of fiscal 2009.

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 IA, "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 December 31,		Related R&D Expenses Six Months Ended December 31,	
				2008	2007	2008	2007
MGd	Cancer	Phase 3	Unknown	\$ 449,000	\$ 585,000	\$ 680,000	\$ 1,562,000
HDAC Inhibitors	Cancer	Phase 1	Unknown	515,000	933,000	1,025,000	1,842,000
Btk Inhibitors	Cancer	Phase 1	Unknown	659,000	966,000	1,545,000	1,561,000
Factor VIIa Inhibitor	Cancer	Phase 1b/2	Unknown	322,000	247,000	666,000	1,318,000
Total direct costs.....				1,945,000	2,731,000	3,916,000	6,283,000
Indirect costs.....				1,041,000	1,731,000	2,273,000	3,419,000
Total research and development expenses.....				\$ 2,986,000	\$ 4,462,000	\$ 6,189,000	\$ 9,702,000

General and Administrative

	Three Months Ended December 31,		Percent Change	Six Months Ended December 31,		
	2008	2007		2008	2007	
General and administrative expenses	\$ 1,874,000	\$ 1,756,000	7%	\$ 5,313,000	\$ 3,823,000	.

The increase of 7% or \$118,000 in general and administrative expenses for the three months ended December 31, 2008, as compared to the three months ended December 31, 2007 was primarily due to share-based compensation expense of \$400,000 and \$200,000 of severance expenses associated with a separation agreement entered into with the company's former CFO in September 2008 and an increase of \$190,000 in legal expenses, partially offset by lower personnel costs of \$325,000 due to lower headcount and a reduction of \$290,000 in non-severance related share-based compensation expense.

The increase of 39% or \$1,490,000 in general and administrative expenses for the six months ended December 31, 2008, as compared to the six months ended December 31, 2007, 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 \$495,000 due to lower headcount and a reduction of \$417,000 in non-severance related share-based compensation expense.

We expect general and administrative expenses to decrease in the second half of fiscal 2009.

Interest and Other, Net

	Three Months Ended December 31,		Percent Change	Six Months Ended December 31,		Percent Change
	2008	2007		2008	2007	
Interest and other, net	\$ 26,000	\$ 358,000	-93%	\$ 126,000	\$ 835,000	-85%

The decreases of 93% or \$332,000 in interest and other, net for the three months ended September 30, 2008, as compared to the three months ended December 31, 2007, and 85% or \$709,000 for the six months ended September 30, 2008, as compared to the six months ended December 31, 2007, were due to lower investment balances and lower average interest rates. Our cash equivalents consist primarily of fixed rate instruments.

We expect to record interest expense of approximately \$500,000 in the second half of fiscal 2009 associated with the accretion of the discount on the \$5,000,000 note payable discounted in Note 7 – “Note Payable.”

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 December 31, 2008, we had approximately \$12,917,000 in cash and cash equivalents. Net cash used in operating activities of \$8,869,000 and \$11,799,000 during the six months ended December 31, 2008 and 2007, respectively, resulted primarily from our net loss net of share-based compensation expenses.

Net cash provided by investing activities of \$4,504,000 in the six months ended December 31, 2008 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 \$18,974,000 in the six months ended December 31, 2007, consisted primarily of proceeds from maturities and sales of marketable securities.

Net cash provided by financing activities of \$5,022,000 in the six months ended December 31, 2008 was primarily due to proceeds from a \$5,000,000 loan. Net cash provided by financing activities of \$48,000 in the six months ended December 31, 2007, 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. See Note 7 for a description of the terms of this loan.

In November 2006, we completed a public offering of common stock and sold 4,830,000 shares of common stock at a price of \$4.75 per share for net proceeds of approximately \$21,300,000. 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 December 31, 2008 are as follows:

	Operating Lease Commitments	Note Payable
Remaining 6 months of fiscal 2009	\$ 456,000	\$ 5,000,000
Fiscal 2010.....	763,000	--
Fiscal 2011	644,000	--
Fiscal 2012.....	330,000	--
Total	<u>\$ 2,193,000</u>	5,000,000
Less unamortized discount.....		(500,000)
Total note payable.....		<u>\$ 4,500,000</u>

In April 2006, we 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 under the agreement, as amended, could total as much as \$104 million, although we currently cannot predict if or when any of the milestones will be achieved. In addition, Celera will also be entitled to royalty payments in the mid- to high single digits based on annual sales of any drugs commercialized from these programs.

Based upon the current status of our product development plans, we believe that our existing cash, cash equivalents and marketable securities will be adequate to satisfy our capital needs through at least the next six months. 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 help fund the development of our product candidates. We also expect to 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 December 31, 2008, all other investment securities are classified as available-for-sale and consequently are recorded on the balance sheet at fair value with

unrealized gains and losses reported as a component of accumulated other comprehensive income (loss) within stockholders' equity. 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.

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 amongst 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 December 31, 2008 would have declined by approximately \$0.

Item 4. Controls and Procedures

(a) *Evaluation of disclosure controls and procedures:* As required by Rule 13a-15 under the Securities Exchange Act of 1934, as of the end of the second 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 controls 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. We also expect to 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.

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 the next six months. 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 expects to receive a deficiency notice from NASDAQ. We do not expect such a notice to have an immediate effect on the company's NASDAQ listing.

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.

We have a history of operating losses and we expect to continue to have losses in the future.

We have incurred significant operating losses since our inception in 1991 and, as of December 31, 2008, had an accumulated deficit of approximately \$350.8 million. We expect to continue to incur substantial additional operating losses until such time, if ever, as the commercialization of our products generates sufficient revenues to cover our expenses. All of our product candidates are in the early stages of development and the commercialization of those products will not occur, if at all, for at least the next several years. Our achieving profitability depends upon our ability, alone or with others, to successfully complete the development of our product candidates, and to obtain required regulatory approvals and to successfully manufacture and market our proposed product. To date, we have not generated revenue from the commercial sale of our products.

If we lose or are unable to hire and retain qualified personnel, then we may not be able to develop our products or processes and obtain the required regulatory approvals.

We are highly dependent on qualified scientific and management personnel. We will need to expand and effectively manage our managerial, operational, financial, development and other resources in order to successfully pursue our research, development and commercialization efforts for our existing and future product candidates. Our success depends on our continued ability to attract, retain and motivate highly qualified management and personnel with pre-clinical and clinical experience. We will need to hire additional personnel as we continue to expand our research and development and partnering activities.

We face intense competition from other companies and research and academic institutions for qualified personnel. We may not be able to attract or retain qualified management and scientific personnel in the future due to the intense competition for

qualified personnel among biotechnology, pharmaceutical and other businesses, particularly in the San Francisco, California area. In September 2008, four members of our Board of Directors resigned and were replaced by four new members. At the same time of this change in our Board, our CEO and CFO resigned their positions. If we lose an additional executive officer, a manager of one of our programs, or a significant number of any of our staff or are unable to hire and retain qualified personnel, then our ability to develop and commercialize our products and processes, raise additional capital or implement our business strategy may be adversely affected or prevented. In particular, if we lose additional members of our senior management team, we may not be able to find suitable replacements in a timely fashion or at all and our business may be harmed as a result.

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

Not Applicable.

Item 3. Defaults Upon Senior Securities

Not Applicable.

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

On December 12, 2008, at the company's 2008 Annual Meeting of Stockholders, the following matters were submitted to and voted on by stockholders and were adopted:

- A. The election by the stockholders of Cynthia C. Bamdad, Ph.D., Robert W. Duggan, James L. Knighton, Minesh P. Mehta, M.D., Glenn C. Rice, Ph.D., and David D. Smith, Ph.D.

	Total Vote for Each Director	Total Vote Against Each Director	Total Abstain
Cynthia C. Bamdad, Ph.D.	21,143,716	2,182,256	256,100
Robert W. Duggan	22,559,735	769,475	252,862
James L. Knighton	21,151,855	2,175,065	255,152
Minesh P. Mehta, M.D.	21,172,789	2,160,032	249,251
Glenn C. Rice, Ph.D.	22,587,151	738,849	256,072
David D. Smith, Ph.D.	22,570,723	753,172	258,177

- B. The amendment of the company's Amended and Restated Certificate of Incorporation to increase the total number of authorized shares of the company's Common Stock from 49,000,000 to 100,000,000.

The results of the vote are as follows:

For	Against	Abstain
21,112,793	2,312,015	157,263

- C. The amendment of the company's 2004 Equity Incentive Award Plan (the "2004 Plan") in order to increase the total number of shares of Common Stock authorized for issuance over the term of the 2004 Plan by an additional 3,000,000 shares.

The results of the vote are as follows:

For	Against	Abstain
10,610,756	3,829,222	24,439

- D. The amendment and restatement of the Company's Employee Stock Purchase Plan (the "Purchase Plan") to (i) increase the maximum number of shares available for issuance under the Purchase Plan by an additional 300,000 shares; (ii) increase the maximum payroll deduction to twenty percent (20%); (iii) increase the maximum number of shares that may be purchased on any purchase date to 10,000 shares; (iv) remove the restriction on the number of times a participant may reduce his or her payroll deduction under the Purchase Plan; and (v) add an automatic reset feature that provides that the offering period in progress shall end immediately following the close of trading on a purchase date and a new offering period shall commence in the event that the fair market value of a share of the Company's common stock on any purchase date is lower than the fair market value of a share of the Company's common stock on the first day of the offering period in which the purchase date occurs.

The results of the vote are as follows:

For	Against	Abstain
13,781,606	661,631	21,180

- E. The ratification of the appointment of PricewaterhouseCoopers LLP as the company's independent registered public accounting firm for the fiscal year ending June 30, 2009.

The results of the vote are as follows:

For	Against	Abstain
23,346,690	162,098	73,283

Item 5. Other Information

Not Applicable.

Item 6. Exhibits

- 10.1 Loan Agreement entered into between the Company and Robert W. Duggan & Associates dated as of December 30, 2008.
- 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.

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: February 13, 2009

By: /s/ ROBERT W. DUGGAN

Robert W. Duggan

Chairman of the Board and Chief Executive Officer

Dated: February 13, 2009

By: /s/ RAINER M. ERDTMANN

Rainer M. Erdtmann

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

EXHIBITS INDEX

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