AXONYX INC
Form 10-Q
August 08, 2006

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 June 30, 2006

or

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

Commission file number: 000-25571

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

Nevada	**86-0883978**
(State or Other Jurisdiction of incorporation or organization)	(IRS Employer Id. No.)

500 Seventh Avenue, 10th Floor,
New York, New York 10018
(Address of Principal Executive Offices)

Registrant s telephone number, including area code: **(212) 645-7704**

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

Indicate by check mark whether the registrant is a large accelerated filer, an accelerated filer or a non-accelerated filer:

Large accelerated filer o Accelerated filer x Non-accelerated filer o

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

As of August 7, 2006, there were 53,680,721 shares of the registrant s $.001 par value Common Stock outstanding.

AXONYX INC.

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PART I. FINANCIAL INFORMATION

ITEM 1. FINANCIAL STATEMENTS

AXONYX INC.
Condensed Consolidated Balance Sheets

	June 30, 2006	December 31, 2005
	(unaudited)	
ASSETS		
Current assets:		
Cash and cash equivalents	**$ 1,880,000**	$ 1,638,000
Investments	**48,850,000**	56,700,000
Other current assets	**340,000**	614,000
Total current assets	**51,070,000**	58,952,000
Property, plant and equipment, net	**42,000**	49,000
Investment in OXIS	**4,336,000**	4,917,000
Security deposits	**24,000**	124,000
	$ 55,472,000	64,042,000
LIABILITIES		
Current liabilities:		
Accounts payable	**$ 2,130,000**	$ 4,147,000
Accrued expenses	**2,316,000**	1,512,000
Total liabilities	**4,446,000**	5,659,000
STOCKHOLDERS EQUITY		
Preferred stock - $.001 par value, 15,000,000 shares authorized; none issued		
Common Stock - $.001 par value, 150,000,000 authorized, 53,680,721 shares issued and outstanding	**54,000**	54,000
Additional paid-in capital	**150,294,000**	149,466,000
Unearned compensation - stock options		(15,000)
Accumulated deficit	**(99,322,000)**	(91,122,000)
Total stockholders equity	**51,026,000**	58,383,000
Total liabilities and stockholders equity	**$ 55,472,000**	$ 64,042,000

See notes to the condensed consolidated financial statements

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AXONYX INC.
Condensed Consolidated Statements of Operations
(unaudited)

	Three months ended June 30,		Six months ended June 30,	
	2006	**2005**	**2006**	**2005**
Product sales	$	$	$	$ 403,000
Cost of product sales				210,000
				193,000
Costs and expenses:				
Research and development	**2,958,000**	7,351,000	**5,619,000**	16,673,000
General and administrative	**1,683,000**	1,213,000	**3,553,000**	2,876,000
	4,641,000	8,564,000	**9,172,000**	19,549,000
Loss from operations	**(4,641,000)**	(8,564,000)	**(9,172,000)**	(19,356,000)
Other income (expense)				
Interest income	**625,000**	558,000	**1,332,000**	1,130,000
Foreign exchange	**(32,000)**	(56,000)	**(23,000)**	(81,000)
Gain (loss) on issuance of subsidiary stock	**46,000**	11,000	**78,000**	(320,000)
Equity in loss of OXIS	**(181,000)**	(128,000)	**(415,000)**	(147,000)
Interest expense				(2,000)
Net loss before outside interest in subsidiary	**$ (4,183,000)**	(8,179,000)	**(8,200,000)**	(18,776,000)
Outside interest in loss of subsidiary				164,000
Net loss	**$ (4,183,000)**	$ (8,179,000)	**$ (8,200,000)**	$ (18,612,000)
Net loss per common share	**$ (.08)**	$ (0.15)	**$ (0.15)**	$ (0.35)
Weighted average shares basic and diluted	**53,681,000**	53,666,000	**53,681,000**	53,661,000

See notes to the condensed consolidated financial statements

AXONYX INC.

Condensed Consolidated Statements of Changes in Stockholders Equity

	Common Stock					
	Number of Shares	**Amount**	**Additional Paid-in Capital**	**Unearned Compensation Stock Options**	**Accumulated Deficit**	**Total Stockholders Equity**
Balance - December 31, 2005	53,680,721	$ 54,000	$ 149,466,000	$ (15,000)	$ (91,122,000)	$ 58,383,000
Issuance of common stock options and warrants for consulting services			18,000			18,000
Issuance of common stock options to employees and directors			810,000			810,000
Amortization				15,000		15,000
Net loss					(8,200,000)	(8,200,000)
Balance June 30, 2006 *(unaudited)*	53,680,721	$ 54,000	$ 150,294,000	$	$ (99,322,000)	$ 51,026,000

See notes to the condensed consolidated financial statements

AXONYX INC.
Condensed Consolidated Statements of Cash Flows
(unaudited)

	Six Months Ended June 30,	
	2006	**2005**
Cash flows from operating activities:		
Net loss	**$ (8,200,000)**	$ (18,612,000)
Adjustments to reconcile net loss to cash used in operating activities:		
Depreciation and amortization	**251,000**	293,000
Compensation related to options and warrants issued for services	**843,000**	265,000
Minority interest in net loss of OXIS		(164,000)
(Gain) Loss on issuance of subsidiary stock	**(78,000)**	320,000
Equity in loss of OXIS	**415,000**	147,000
Changes in:		
Accounts receivable		(105,000)
Inventories		(1,000)
Other current assets	**274,000**	(229,000)
Security deposits and other assets	**100,000**	1,000
Accounts payable	**(2,017,000)**	(1,682,000)
Accrued expenses	**816,000**	(886,000)
Accrued stock based compensation	**(12,000)**	(344,000)
Net cash used in operating activities	**(7,608,000)**	(17,633,000)
Cash flows from investing activities:		
Purchase of equipment		(1,000)
Additions to patents		(48,000)
Reduction in cash due to deconsolidation of OXIS		(4,907,000)
Purchases of investments	**(14,400,000)**	(41,500,000)
Proceeds from sales and maturities of investments	**22,250,000**	57,200,000
Net cash provided by investing activities	**7,850,000**	10,744,000
Cash flows from financing activities:		
Net proceeds from issuance of common stock and warrants		20,000
Collection of stock subscription receivable - OXIS		2,250,000
Net proceeds from exercise of common stock options in OXIS		33,000
Net cash provided by financing activities		2,303,000
Net increase (decrease) in cash and cash equivalents	**242,000**	(4,586,000)
Cash and cash equivalents at beginning of period	**1,638,000**	10,091,000

Cash and cash equivalents at end of period	**$ 1,880,000**	$ 5,505,000
Supplemental cash flow disclosures		
Interest paid		$ 2,000
Supplemental disclosure of non-cash financing activity:		
Minority interest in subsidiary equity transactions		$ 22,000

See notes to the condensed consolidated financial statements

Notes to Condensed Consolidated Financial Statements

(1) Recent Events

On June 8, 2006, Axonyx Inc. (the Company) announced that it had entered into a definitive merger agreement with TorreyPines Therapeutics, Inc., a private company. The merger would create a NASDAQ listed biopharmaceutical company that discovers and develops treatments for central nervous system (CNS) disorders. The resulting company will be named TorreyPines Therapeutics, Inc. and be headquartered in San Diego, California.

Under terms of the agreement, unanimously approved by both boards of directors, Axonyx would issue, and TorreyPines stockholders would receive, in a tax-free transaction, shares of Axonyx common stock such that TorreyPines stockholders would own approximately 58% of the combined company and Axonyx stockholders would own approximately 42%. These relative percentages will be adjusted if either party out-licenses one or more product candidates prior to closing. In addition, TorreyPines preferred shareholders will receive warrants to purchase combined company shares that, if fully exercised at closing, would give TorreyPines shareholders approximately 62% of the combined company. The transaction is subject to Axonyx maintaining certain minimum cash levels, as well as certain other customary conditions, including obtaining approval of each company s stockholders. The transaction is expected to close during the fourth quarter of this year. Upon closing, the company is expected to trade on the NASDAQ Stock Market for which the company has reserved the symbol TPTX.

On July 25, 2006 the Company filed a Registration Statement on Form S-4 with the Securities and Exchange Commission (SEC) which details the required proxy disclosures in connection with the merger.

The Company s common stock is listed on the NASDAQ Capital Market (formerly known as the NASDAQ SmallCap Market). In order to maintain a listing, the Company must meet minimum financial and other requirements. If the Company is unable to comply with NASDAQ s listing standards, they may determine to delist the common stock from the NASDAQ Capital Market. On August 2, 2006, the Company received notice from NASDAQ stating that it was out of compliance with bid price requirements because the closing bid price for its common stock was below $1.00 per share for 30 consecutive business days. The Company would have 180 days to regain compliance with bid price requirements. To regain compliance the closing bid price for the Company s common stock must be a minimum of $1.00 per share for at least 10 consecutive business days. If NASDAQ made a determination to delist the Company s common stock, the delisting procedure would involve a process beginning with NASDAQ s notification and would include a hearing and the possibility of appeal. There is no assurance that at the end of this process the Company s common stock would continue to be listed on the NASDAQ Capital Market. If the Company s common stock is delisted for any reason, it could reduce the value of the Company s common stock and its liquidity, and could potentially adversely impact the Company s proposed merger with TorreyPines Therapeutics Inc. Delisting could also adversely affect the Company s ability to obtain financing for the continuation of its operations or to its common stock in acquisitions. Delisting could result in the loss of confidence by suppliers, customers and employees.

(2) Financial Statement Presentation

The unaudited condensed consolidated financial statements of the Company herein have been prepared pursuant to the rules and regulations of the SEC and, in the opinion of management, reflect all adjustments necessary to present fairly the financial position at June 30, 2006, and the results of operations and cash flows for the quarterly and six month periods presented. Certain information and footnote disclosure normally included in financial statements, prepared in accordance with accounting principles generally accepted in the United States of America, have been condensed or omitted pursuant to such rules and regulations. However, management believes that the disclosures are adequate to make the information presented not misleading. These financial statements and notes thereto should be read in conjunction with the financial statements and the notes thereto for the year ended December 31, 2005, included in the Company s Form 10-K filing. The results for the interim periods are not necessarily indicative of the results for the full fiscal year.

The consolidated financial statements include the accounts of Axonyx Europe, B.V., a wholly owned subsidiary organized in Holland.

(3) Investment in OXIS International, Inc.

The Company consolidated the financial statements of OXIS International Inc. from the date of acquisition of 52% of the outstanding voting stock of OXIS (January 15, 2004) through February 28, 2005, when it no longer controlled the board of OXIS or directed its day-to-day management activities. The Company s ownership in OXIS was reduced from 52% to 34% on December 31, 2004 as a result of a third party financing. However, as the Company continued to control the board of OXIS and continued to direct the day-to-day management activities consolidation of OXIS continued through February 28, 2005. Beginning March 1, 2005, the Company accounts for its investment in OXIS under the equity method of accounting, as prescribed by Accounting Principals Board Opinion No. 18 The Equity Method of Accounting for Investments in Common Stock .

The Company owns approximately 14 million common shares of OXIS International Inc. with a carrying value at June 30, 2006 of $4,336,000. OXIS is traded on the bulletin board (OXIS.OB) with relatively little trading volume. At June 30, 2006, the market value of these shares was $0.34 per share ($4,760,000). Axonyx currently owns 32% of OXIS.

(4) Investments

The Company invests in auction-rate securities (ARS) that are held as investments available for sale. Auction rate securities are instruments with long-term underlying maturities, but for which an auction is conducted periodically, as specified, to reset the interest rate and allow investors to buy or sell the instruments. Because auctions generally occur more often than annually, and because the Company holds these instruments in order to meet short-term liquidity needs, the auction rate securities are classified as short-term investments in the Condensed Consolidated Balance Sheet. Consistent with our other securities that are classified as available-for-sale, the Condensed Consolidated Statement of Cash Flows reflects the gross amount of the purchases of auction rate securities and the proceeds from sales of auction rate securities.

(5) Stock-based Compensation

In 2000, the Board of Directors and the stockholders of the Company approved a Stock Option Plan (2000 Plan) which, as amended, provides for the granting of options to purchase up to 2,000,000 shares of common stock and pursuant to which officers, directors, advisors and consultants are eligible to receive incentive and/or non-statutory stock options. Incentive stock options granted under the 2000 Plan are exercisable for a period of up to 10 years from date of grant at an exercise price which is not less than the fair value on date of grant, except that the exercise period of options granted to a stockholder owning more than 10% of the outstanding stock may not exceed five years and their exercise price may not be less than 110% of the fair value of the common stock at date of grant. Vesting of 2000 Plan options varies from fully vested at the date of grant to multiple year apportionment of vesting as determined by the Board of Directors. Pursuant to the 2000 stock option plan as amended, 750,000 options were added to the share reserve effective January 1, 2003 and January 1, 2004. On March 30, 2004, the Company amended the 2000 Plan to increase the aggregate number of shares from 3,500,000 to 7,500,000. Stockholder approval for the increase was received in June 2004.

Commencing January 1, 2006 the Company adopted Statement of Financial Accounting Standard No. 123R, Share Based Payment (SFAS 123R), which requires all share-based payments, including grants of stock options, to be recognized in the statement of operations as an operating expense, based on fair values.

Prior to adopting SFAS 123R, the Company accounted for stock-based employee compensation under Accounting Principles Board Opinion No. 25, Accounting for Stock Issued to Employees . The Company has applied the modified prospective method in adopting SFAS 123R. Accordingly, periods prior to adoption have not been restated.

The following table illustrates the effect on net loss and loss per share if the fair value based method had been applied to all awards for the three and six months ended June 30, 2005 when APB opinion No. 25 was followed.

	Three Months Ended June 30, 2005	Six Months Ended June 30, 2005
Reported net loss attributable to common stockholders	$ (8,179,000)	$ (18,612,000)
Stock-based employee compensation included in net loss	52,000	84,000
Stock-based employee compensation determined under the fair value based method	(735,000)	(1,310,000)
Pro forma net loss	$ (8,862,000)	$ (19,838,000)
Loss per common share attributable to common stockholders (basic and diluted):		
As reported	$ (0.15)	$ (0.35)
Pro forma	$ (0.17)	$ (0.37)

As of June 30, 2006, there was $1,560,000 of total unrecognized compensation cost related to nonvested share-based compensation arrangements granted under existing stock option plans. This cost is expected to be recognized over a weighted-average period of 1.1 years. The total measurement fair value of shares vested during the six months ended June 30, 2006 was $832,000.

The Company uses the Black-Scholes option pricing model to determine the weighted average fair value of options. The fair value of options at date of grant and the assumptions utilized to determine are indicated in the following table:

	Six Months Ended June 30 ,	
	2006	2005
Weighted average fair value at date of grant for options granted during the period	$ 1.00	$ 1.21
Risk-free interest rates	4.57%	3.77% - 3.85%
Expected option life in years	10	10
Expected stock price volatility	.93	.97
Expected dividend yield	-0-	-0-

Under SFAS 123R forfeitures are estimated at the time of valuation and reduce expense ratably over the vesting period. This estimate is adjusted periodically based on the extent to which actual forfeitures differ, or are expected to differ, from the previous estimate. Under SFAS 123 and APB 25, the Company elected to account for forfeitures when awards were actually forfeited, at which time all previous pro-forma expense was reversed to reduce pro- forma expense for the period. As of June 30, 2006, the Company anticipates all outstanding options will vest.

The following summarizes the activity of the Company s stock options for the six months ended June 30, 2006.

	Shares	Weighted Average Exercise Price	Weighted Average Remaining Contractual Term (Years)	Aggregate Intrinsic Value
Outstanding at December 31, 2005	**5,321,000**	**$ 3.97**		
Granted	**75,000**	**1.13**		
Exercised				
Canceled or expired	**(241,000)**	**4.18**		
Outstanding at June 30, 2006	**5,155,000**	**$ 3.92**	5.77	$ **44,120**
Exercisable at June 30, 2006	**4,269,000**	**$ 4.13**	5.16	$ **44,120**

The following summarizes the activity of the Company s non-vested stock options for the six months ended June 30, 2006.

	Shares	Weighted Average Fair Value
Nonvested at December 31, 2005	1,034,000	$ 2.88
Granted	75,000	1.13
Vested	(223,000)	2.01
Nonvested at June 30, 2006	886,000	2.95

(5) Operating Segments

Beginning March 1, 2005, the OXIS segment reflects the Company s share of OXIS losses under the equity method. Prior to that date, the Company was organized into two reportable segments: Axonyx and OXIS.

The following table presents information about the Company s two operating segments for the quarter and six months ended June 30, 2005:

	Axonyx Inc.	OXIS Int l Inc.	Total
Quarter ended June 30, 2005			
Segment Loss	$ (8,050,000)	$ (129,000)	$ (8,179,000)
Six months ended June 30, 2005			
Revenue including minority interest		$ 403,000	$ 403,000
Segment loss	$ (18,286,000)	$ (326,000)	$ (18,612,000)

(6) Developments with SERONO International SA

Under a License Agreement with Applied Research Systems ARS Holding N.V. (ARS), a wholly owned subsidiary of Serono International, S.A. (Serono) effective September 15, 2000, the Company granted to ARS a sublicense of our patent rights and know-how regarding the development and marketing of the Amyloid Inhibitory Peptide (AIPs) and the Prion Inhibitory Peptide (PIPs) technology which had been licensed to us under a Research and License Agreement with New York University. The Company is negotiating a re-acquisition of those rights from ARS and an option to license, on a non-exclusive basis, certain Serono patents, technology and know-how related to AIPs and PIPs. If the Company exercises this option and acquires the license, it would be obligated to pay to Serono an upfront payment and under certain circumstances additional milestone payments and royalties would be due.

(7) Legal Proceedings

Several lawsuits were filed against the Company in February 2005 in the U.S. District Court for the Southern District of New York asserting claims under Sections 10(b) and 20(a) of the Securities Exchange Act of 1934 and Rule 10b-5 thereunder on behalf of a class of purchasers of the Company s common stock during the period from June 26, 2003, through and including February 4, 2005 (the Class Period). Dr. M. Hausman (a director and former

CEO of Axonyx), and Dr. G. Bruinsma (Axonyx CEO) were also named as defendants in the lawsuits. On April 10, 2006, following consolidation of the actions into a single class action lawsuit, the lead plaintiffs filed a consolidated amended complaint. The Company s motion to dismiss the consolidated amended complaint was filed on May 26, 2006 and will be submitted to the Court for a decision following the parties filing of their legal briefs.

The class action plaintiffs allege generally that Axonyx s Phase III Phenserine development program was subject to alleged errors of design and execution which resulted in the failure of the first Phase III Phenserine trial to show efficacy. Plaintiffs allege the defendants failure to disclose the alleged defects resulted in the artificial inflation of the price of the Company s shares during the Class Period.

There is also a shareholder derivative suit pending in New York Supreme Court (New York County) against current and former directors and officers of Axonyx. The named defendants are Marvin S. Hausman, Gosse B. Bruinsma, S. Colin Neill, Louis G. Cornacchia, Steven H. Ferris, Gerald J. Vlak, Ralph Snyderman and Michael A. Griffith. Defendants are alleged to have breached their duties to the Company and misused inside information regarding clinical trials of Phenserine. This action has been stayed pending further developments in the federal class action.

The complaints seek unspecified damages. The Company believes the complaints are without merit and intends to defend these lawsuits vigorously. However, the Company cannot make assurances that it will prevail in these actions, and, if the outcome is unfavorable to Axonyx, its reputation, operations and share price could be adversely affected.

(8) New Accounting Pronouncement

In June 2006, the Financial Accounting Standards Board (FASB) issued FASB Interpretation No. 48, Accounting for Uncertainty in Income Taxes an interpretation of FASB Statement No. 109 (FIN 48), which prescribes a recognition threshold and measurement attribute for the 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 de-recognition, classification, interest and penalties, accounting in interim periods, disclosure and transition. FIN 48 is effective for fiscal years beginning after December 15, 2006. The Company does not expect the adoption of FIN 48 to have a material impact on our financial reporting, and the Company is currently evaluating the impact, if any, the adoption of FIN 48 will have on our disclosure requirements.

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

This Form 10-Q contains forward-looking statements, as defined in the Private Securities Litigation Reform Act of 1995 that are based on current expectations, estimates and projections. Statements that are not historical facts, including statements about our beliefs and expectations, are forward-looking statements. These statements involve potential risks and uncertainties; therefore, actual results may differ materially. You are cautioned not to place undue reliance on these forward-looking statements, which speak only as of the date on which they were made. We do not undertake any obligation to update any forward-looking statements, whether as a result of new information, future events or otherwise.

Specifically, with respect to our drug candidates Phenserine, Posiphen and Bisnorcymserine, Axonyx cannot assure that: any preclinical studies or clinical trials, whether ongoing or conducted in the future, will prove successful, and if successful, that the results can be replicated; safety and efficacy profiles of any of its drug candidates will be established, or if established, will remain the same, be better or worse in future clinical trials, if any; pre-clinical results related to cognition and the regulation of beta-APP will be substantiated by ongoing or future clinical trials, if any, or that any of its drug candidates will be able to improve the signs or symptoms of their respective clinical indication or slow the progression of Alzheimer s disease; any of its drug candidates will support an NDA filing, will be approved by the FDA or its equivalent, or if approved, will prove competitive in the market; or that Axonyx will have or obtain the necessary financing to support its drug development programs. Axonyx cannot assure that it will be successful with regard to identifying a (sub-) licensing partner for any of its compounds, or that any such partner will successfully develop or commercialize any of such compounds. Axonyx undertakes no obligation to publicly release the result of any revisions to such forward-looking statements that may be made to reflect events or circumstances after the date hereof or to reflect the occurrence of unanticipated events.

We refer you to our report on form 10-K for the year ended December 31, 2005 filed with the SEC, where these risks and others are more fully described.

We do not undertake to discuss matters relating to our ongoing clinical trials or our regulatory strategies beyond those which have already been made public or discussed herein.

Recent Events

On June 8, 2006, the Company announced that it had entered into a definitive merger agreement with TorreyPines Therapeutics, Inc., a private company. The merger would create a NASDAQ listed biopharmaceutical company that discovers and develops treatments for central nervous system (CNS) disorders. The resulting company will be named TorreyPines Therapeutics, Inc. and be headquartered in San Diego, California.

Under terms of the agreement, unanimously approved by both boards of directors, Axonyx would issue, and TorreyPines stockholders would receive, in a tax-free transaction, shares of Axonyx common stock such that TorreyPines stockholders would own approximately 58% of the combined company and Axonyx stockholders would own approximately 42%. These relative percentages will be adjusted if either party out-licenses one or more product candidates prior to closing. In addition, TorreyPines preferred shareholders will receive warrants to purchase combined company shares that, if fully exercised at closing, would give TorreyPines shareholders approximately 62% of the combined company. The transaction is subject to Axonyx maintaining certain minimum cash levels, as well as certain other customary conditions, including obtaining approval of each company s stockholders. The transaction is expected to close during the fourth quarter of this year. Upon closing, the company is expected to trade on the NASDAQ Stock Market for which the company has reserved the symbol TPTX.

On July 25, 2006 the Company filed a Registration Statement on Form S-4 with the SEC which details the required format proxy disclosure in connection with the merger.

Our common stock is listed on the NASDAQ Capital Market (formerly known as the NASDAQ SmallCap Market). In order to maintain our listing, we must meet minimum financial and other requirements. If we are unable to comply with NASDAQ s listing standards, may determine to delist our common stock from the NASDAQ Capital Market. On August 2, 2006, we received notice from NASDAQ stating that we were out of compliance with bid price requirements because the closing bid price for our common stock was below $1.00 per share for 30 consecutive business days. We would have 180 days to regain compliance with bid price requirements. To regain compliance the closing bid price for our common stock must be a minimum of $1.00 per share for at least 10 consecutive business days. If NASDAQ made a determination to delist our common stock, the delisting procedure would involve a process beginning with NASDAQ s notification and would include a hearing and the possibility of appeal. There is no assurance that at the end of this process our common stock would continue to be listed on the NASDAQ Capital Market. If our common stock is delisted for any reason, it could reduce the value of our common stock and its liquidity, and could potentially adversely impact our proposed merger with TorreyPines Therapeutics Inc. Delisting could also adversely affect our ability to obtain financing for the continuation of our operations or to use our common stock in acquisitions. Delisting could result in the loss of confidence by suppliers, customers and employees.

Overview of our Company

We are a biopharmaceutical company, specializing in central nervous system (CNS) neurodegenerative diseases, engaged in the business of acquiring patent rights to clinical stage compounds, compounds with strong proof of concept data and compounds ready for proof of concept validation with convincing scientific rationale, or potentially another company with similar rights. We further develop and add value to these compounds and then seek to out-license or partner them when we believe it business prudent. We have acquired patent rights to three main classes of therapeutic compounds designed for the treatment of Alzheimer s disease (AD), Mild Cognitive Impairment, and related diseases. We have acquired patent rights to a class of potential therapeutic compounds designed for the treatment of prion related diseases, which are degenerative diseases of the brain that are thought to be caused by an infectious form of a protein called a prion. Prions, unlike viruses, bacteria and fungi, have no DNA and consist only of protein. Such diseases include Creutzfeldt Jakob Disease, new variant in humans, Bovine Spongiform Encephalopathy (BSE or Mad Cow Disease) in cows, and Scrapies disease in sheep. We have licensed

these patent rights from New York University. We also have co-ownership rights to patent applications regarding the therapeutic compound named Posiphen designed for the treatment of AD progression and Bisnorcymserine (BNC) in development for the treatment of severe AD.

Our mission is to be a leading biopharmaceutical company that develops products and technologies to treat central nervous system disorders. Our initial business strategy has been focused primarily on three compounds in development for AD. These are:

Phenserine A symptomatic and disease progression treatment of mild to moderate AD

Posiphen A disease progression treatment for AD

Bisnorcymserine (BNC) A symptomatic treatment of severe AD

Our current business strategy includes identifying and seeking to in-license potential compounds or partner with companies to expand our product development portfolio. Partnering may take the form of a business combination in which we merge with, acquire, or are acquired by, another company. See Recent Events, above.

Phenserine is an inhibitor of acetylcholinesterase for the potential treatment of mild to moderate AD. Acetylcholinesterase is an enzyme active in the nerve synapse that degrades the neurotransmitter acetylcholine in the brain and other tissues of the body. Acetylcholinesterase inhibitors are drugs designed to selectively inhibit acetylcholinesterase. Acetylcholine is a chemical substance that sends signals between nerve cells, called neurotransmission, and is therefore called a neurotransmitter. Neurotransmitters are secreted by neurons, or nerve cells, into the space between neurons called the synapse. Acetylcholine is a primary neurotransmitter in the brain, and is associated with memory and cognition. Inhibition of its breakdown in AD patients has been shown to improve memory and cognition.

Posiphen is a compound that appears to decrease the formation of the beta amyloid precursor protein (beta-APP) and amyloid with potential applications in the treatment of AD progression. Posiphen is the positive isomer of Phenserine. As such, it appears to affect the messenger RNA of beta-APP as well as inhibit beta secretase whereby levels of neurotoxic beta amyloid, in preclinical animal models, are reduced.

Bisnorcymserine is a butyrylcholinesterase inhibitor. Butyrylcholinesterase is found in high concentration in the plaques taken from individuals who have died from AD. Butyrylcholinesterase is an enzyme that is normally found widely in the body and butyrylcholine appears to play a relatively increasingly important role in advancing AD. Inhibition of the enzyme may prove valuable in the treatment of severe AD.

The Phenserine Development Program

Our most advanced compound, Phenserine, selectively inhibits acetylcholinesterase, the enzyme primarily responsible for degrading acetylcholine at the synaptic gap between neurons, thus increasing the availability of this neurotransmitter. Phenserine has been shown to be a potent and selective inhibitor of this enzyme in the rat brain and increases memory and learning over a wide therapeutic dosage range in aged rats without causing toxic side effects. The compound readily enters the brain, has minimal activity in other organs outside the brain, and has a long duration of action. In pre-clinical studies, Phenserine was shown to have a brain to blood ratio of 10:1. Increasing the concentration of the active drug agent in the brain versus the rest of the body potentially maximizes the effects of the drug while potentially reducing peripherally mediated side effects.

Phenserine also has been shown to have the ability to inhibit the formation of the beta-amyloid precursor protein (beta-APP), a large protein that is the source of the neurotoxic peptide , beta amyloid. By inhibiting the formation of beta-APP, Phenserine can decrease the presence of the soluble beta amyloid protein that is potentially deposited in the brain as amyloid plaques, apparently causing eventual neuronal cell death. These studies were conducted at laboratories at the National Institute of Aging (NIA) in human neuroblastoma cell cultures and *in vivo* in rodents. Studies in human neuroblastoma cell lines showed that the compound reduces the formation of beta-amyloid peptide. Neuroblastoma cell cultures are a type of cell derived from the human brain that can be grown in containers in the lab (*in vitro*) where they are able to reproduce and carry out many activities as if they were residing in the brain, including the synthesis and secretion of proteins such as the beta-amyloid protein which, in the human brain, can form plaques. A neuroblastoma cell culture is used to study brain cell function in a simple *in vitro* system, which allows testing of the ability of drugs to prevent the formation of the beta-amyloid precursor protein and

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secretion of beta amyloid. Additional animal studies using the transgenic mouse have confirmed these findings. The transgenic mouse is a bio-engineered animal that mimics hallmark pathologic changes that occur in the human AD brain. These results suggest that Phenserine may have the ability to slow the progression of AD in addition to providing symptomatic relief for the cognitive changes.

In December 1999, we initiated Phase I human clinical trials for Phenserine utilizing healthy elderly patients at a U.S. research center. These Phase I safety and tolerance trials involving both single and multiple ascending doses were successfully completed in September 2000.

In October 2001, we completed a Phase II proof-of-concept double-blind placebo-controlled clinical trial with Phenserine in AD patients. This Phase II proof-of-concept trial was designed to determine the drug s safety and possibly a trend toward efficacy in patients exhibiting mild to moderate AD. The trial included 72 patients, with 48 patients receiving two daily doses of Phenserine 10mg and 24 patients received a placebo. The safety results from the trial substantiated Phase I results indicating that the drug is safe and well tolerated. Although the trial was not of the duration necessary and did not include the number of patients required to detect statistically significant clinical improvement in efficacy, nevertheless certain memory tests showed statistically significant results while other tests showed a trend towards statistical significance.

To date, we have conducted the following Phase III clinical trials with Phenserine: AX-CL-06/06e, AX-CL-09, AX-CL-010, as well as a Phase IIb trial, AX-CL-06a.

Protocol AX-CL-06 was a double-blind, placebo controlled trial initiated in June 2003 comparing the efficacy and tolerability of Phenserine 10mg or 15mg twice daily doses with twice daily placebo in patients who met the diagnostic criteria for probable mild to moderate AD. Two different regimens, 10mg twice daily and 15mg twice daily, were compared with placebo in this trial. The randomization was 1:2:2 for placebo: 10mg twice daily: 15mg twice daily respectively. Patients randomized to active treatment were started on a 5mg twice daily regimen for the first month of treatment. This was increased to 10mg twice daily for the second month of treatment. The dose was then increased to 15mg twice daily during the third through sixth month for those patients randomized to the highest dose regimen. Once a patient reached his or her target dose, it was maintained for a total treatment duration of 26 weeks. Patients who could not tolerate their target dose were discontinued. Discontinued patients were not replaced. A total of 384 patients were enrolled in the study. Of these, 377 received treatment. The remaining 7 never received drug treatment so they were excluded from the data analyses.

The primary efficacy variables were the Alzheimer s Disease Assessment Scale-cognitive subscale (ADAS-Cog) and Clinical Interview Based Impression of Change (CIBIC+). The Phenserine groups showed consistently greater improvement in ADAS-Cog and CIBIC+ scores than the placebo group although the differences did not achieve statistical significance.

Protocol AX-CL-06a was a double-blind placebo controlled study of the effect of Phenserine 10- or 15mg twice daily on cerebrospinal and plasma amyloid peptides from baseline and, at 26 weeks, initiated in June 2003. Both doses of Phenserine tended to lower the plasma levels of beta-amyloid peptides more than placebo, while beta- amyloid levels in the CSF declined in those patients treated with placebo. This decline of amyloid levels in the CSF of untreated AD patients is consistent with historical and epidemiological data. None of the differences achieved statistical significance.

Protocol AX-CL-06e was an open-label extension to studies AX-CL-06 and AX-CL-06a that allowed all patients who had successfully completed either trial to continue on Phenserine 15mg twice daily dose for up to an additional six months. This extension was to gather additional safety data on Phenserine treatment.

Protocol AX-Cl-09/010, initiated in the second half of 2004, was originally initiated as two identical 26-week placebo controlled trials of 450 AD patients each. During the implementation of the studies, results of Protocol AX-CL-06 became available. The results of this earlier study showed a numerical benefit of Phenserine treatment relative to placebo but failed to achieve statistical significance. Based on these results, enrollment in the two ongoing studies was halted at 255 patients in total, and the primary endpoint analysis was shortened to 12 weeks. Because the individual curtailed studies were underpowered, their data were combined and analyzed as a single trial. This was a randomized, multinational, multicenter placebo-controlled parallel-group study. Because the study was curtailed, many patients did not reach the originally scheduled 26-week end of treatment. However, all patients were allowed to complete at least 12 weeks of therapy. Patients were screened within 21 days of entry and randomly assigned to receive 10 or 15 mg of Phenserine twice daily or placebo. A titration schedule was used so that patients

randomized to active treatment received 5mg twice daily for the first 4 weeks of the study followed by 10mg twice daily for 4 weeks. Patients randomized to 15mg twice daily received this dose starting in the ninth week. Treatment at the assigned doses was continued for up to 26 weeks. At the 12-week visit, patients randomized to 10mg twice daily had received this dose for approximately 8 weeks. Patients randomized to receive 15mg twice daily had received this dose for approximately 4 weeks.

Although the study did not achieve statistical significance in its primary endpoints, a subgroup of patients, who received Phenserine 15mg twice daily, demonstrated a statistically significant benefit over placebo as measured by the ADAS-cog, when treated for more than 12 weeks. Additionally, this subgroup showed a positive trend towards improvement in the CIBIC+ test, which approached statistical significance. There were no unexpected safety or tolerability concerns associated with Phenserine treatment.

We have comprehensive data sets on Phenserine having completed extensive manufacturing scale-up, preclinical studies and taken the drug into three Phase III clinical trials for mild to moderate AD. The Company has determined that it will not commit further resources to these Phase III trials, and is seeking to identify strategic partners that are able and willing to commit the necessary financial resources to Phenserine s further development and marketing approval.

On January 4, 2006, we announced that we had granted to Daewoong Pharmaceutical Company Ltd. (Daewoong) an exclusive license for the use of Phenserine in the South Korean market. Under the terms of the agreement Daewoong, at its own costs, undertakes to pursue the product development and regulatory work necessary for a New Drug Application (NDA) (or its equivalent) in South Korea with respect to Phenserine for the treatment of AD. The financial terms of the deal include royalty payments to us based on sales of Phenserine by Daewoong in the South Korean market.

On January 31, 2006, we announced that three presentations of data on our drug development candidate, Phenserine, and one presentation of data on its drug development candidate, Posiphen , would be made at the 9th International Geneva/Springfield Symposium on Advances in Alzheimer Therapy in Geneva, Switzerland, being held April 19-22, 2006. Phenserine has been in development by the Company for the treatment of mild to moderate Alzheimer s disease (AD) and Posiphen is currently in clinical development for the treatment of AD progression.

On February 14, 2006, we reported a statistically significant reduction compared to baseline in the plasma levels of beta-amyloid 1-42 (Aß-42) in healthy human subjects treated with Phenserine for 35 days in a previously conducted Phase I study.

On April 19, 2006 we reported an analysis of results suggesting stabilization of total brain volume and brain parenchymal fraction of mild-to-moderate Alzheimer s disease (AD) patients treated with Phenserine 10mg or 15mg twice daily (BID) for 26-weeks. The data was presented in its entirety as a poster in Geneva on Thursday, April 20, 2006 at the 9th International Geneva/Springfield Symposium on Advances in Alzheimer Therapy.

On April 20, 2006 we reported on data showing an increase in brain glucose metabolism and reduction of brain amyloid levels in the memory and cognition areas in brains of mild-to-moderate Alzheimer s disease (AD) patients treated with Phenserine 15mg twice daily (BID) for 13-weeks. The data was included in an oral presentation by Prof. Dr. Agneta Nordberg, MD, PhD of the Karolinska Institute, Stockholm, Sweden, on Friday, April 21st, 2006 at the 9th International Geneva/Springfield Symposium on Advances in Alzheimer Therapy.

The Posiphen Development Program

Posiphen is the positive isomer of Phenserine. It appears to decrease the formation of beta-amyloid (Aß) with potential application in the treatment of AD progression. The build-up of beta-amyloid (Aß) is generally believed to be causative of the dementia of Alzheimer s disease and its progression. Posiphen s mechanism of action is potentially through RNA translational inhibition and possibly beta secretase inhibition. Posiphen has been shown to lower beta amyloid precursor protein (beta-APP) and beta-amyloid levels in pre-clinical studies. The primary mechanism of action results in a dose dependent reduction of beta-amyloid, which may result in slowing AD progression. The initial pre-clinical side effect rates potentially allow for higher clinical doses. On August 1, 2005 we announced that the US Food and Drug Administration (FDA) approved our investigational new drug (IND) application allowing Phase I clinical testing of Posiphen . The first Phase I single ascending dose clinical study commenced in August 2005 and evaluated the safety of Posiphen in healthy volunteers.

In January 2006, we completed a single ascending dose Phase I trial with Posiphen . This double-blind, placebo controlled study of Posiphen in healthy men and women sought to establish well tolerated doses. Posiphen appears to be well tolerated at single doses up to and including 80mg. Blood levels of Posiphen associated with this study were higher than those associated with beneficial effects on beta-amyloid metabolism in animal models. The build-up of beta-amyloid (Aß) is generally believed to be causative of the dementia of AD. No serious adverse events were reported at any dose level. We initiated a Phase I multiple ascending dose study in the first quarter of 2006.

On April 24, 2006, we announced the results of an independent research study showing that Posiphen increased the ability of transplanted human neuronal stem cells (HNSC) to differentiate into neurons in APP transgenic mice, a model of Alzheimer s disease (AD) in humans.

On May 15, 2006 we announced the completion of a multiple dose Phase I study with Posiphen in clinical development for the treatment of Alzheimer s disease progression.

This double-blind, placebo-controlled multiple ascending dose safety and pharmacokinetic study of Posiphen in healthy volunteers sought to establish well tolerated doses. The initial review of the clinical adverse event data appears to be generally consistent with the results of the earlier single ascending dose Phase I study that suggested that the mean Posiphen blood levels associated with well tolerated doses in humans are higher than those associated with potentially beneficial effects on beta-amyloid metabolism in animal models. No serious adverse events were reported at any dose level in this second Phase I study.

This multiple ascending-dose study examined the effects of Posiphen 20, 40 and 60mg given four times daily, for a period of 7, 7 and 10 days respectively. On the first and last day of each dosing period one single dose of Posiphen was given. Each dose period was completed and evaluated for safety and tolerance before the next higher dose level was initiated. Each cohort was composed of a different set of 16 subjects, comprised of 12 who received Posiphen and 4 who received placebo, with equal numbers of males and females in each.

The necessary detailed safety, pharmacokinetic and pharmacodynamic analyses are ongoing. Based on this favorable clinical outcome, we are evaluating plans regarding the further clinical development steps for Posiphen.

The Bisnorcymserine Development Program

Our butyrylcholinesterase inhibitor compounds are designed to selectively inhibit butyrylcholinesterase, an enzyme similar to acetylcholinesterase. Normally these two enzymes coexist throughout the body, with acetylcholinesterase predominating in degrading acetylcholine. In the brain of AD patients, as acetylcholinesterase levels gradually fall, there appears to be a concomitant increase in butyrylcholinesterase levels in specific nerve pathways within the cortex and the hippocampus, areas associated with AD. Like acetylcholinesterase, butyrylcholinesterase degrades acetylcholine at the synaptic gap between neurons, decreasing the availability of this key neurotransmitter. This enzyme was identified as a target for inhibition in AD as it also terminates the action of the neurotransmitter acetylcholine in specific nerve pathways in regions of the brain associated with AD and is found in high concentration in amyloid plaques in the brains of AD patients. Our butyrylcholinesterase inhibitor compounds act to counter butyrylcholinesterase, thus enhancing the availability of acetylcholine, potentially improving memory and cognition. Inhibition of butyrylcholinesterase may also reduce any increased toxicity of beta-amyloid caused by the presence of butyrylcholinesterase in amyloid plaques.

Several butyrylcholinesterase inhibitor drug candidates, including Bisnorcymserine, have been studied extensively in pre-clinical studies and have been found to have many of the characteristics desirable for use in AD. Like Phenserine, these compounds have a dual mechanism of action in that, in addition to inhibiting the butyrylcholinesterase enzyme, they also inhibit the formation of beta-APP in cell culture, and in rats. These pre-clinical findings indicate that these butyrylcholinesterase inhibitor compounds may have an important role in preventing the formation of amyloid plaques in AD, in addition to its inhibition of butyrylcholinesterase. The compounds readily enter the brain, they have a long duration of action and are highly active in improving memory and learning in the aged rat. Currently it appears that Bisnorcymserine has several advantages over the other

compounds in pre-clinical results. Bisnorcymserine appears to be the most potent butyrylcholinesterase inhibitor in our patent portfolio. It has a 100-fold selectivity over acetylcholinesterase. Behavioral work shows it to improve memory in rodent models, and it reduces beta-APP in tissue cultures. Bisnorcymserine has three potential uses: (1) as an inhibitor of butyrylcholinesterase, (2) as an inhibitor of the production of beta-APP, thus inhibiting the formation of amyloid plaques, and (3) as an early diagnostic marker.

Bisnorcymserine (BNC) is a highly selective butyrylcholinesterase inhibitor. Butyrylcholinesterase is found in high concentration in the plaques taken from individuals who have died from AD. Butyrylcholinesterase appears to have an increasing role with advancing AD and its primary mechanism of action results in a dose dependent reduction of acetylcholine. The initial pre-clinical side effect rate potentially allows higher clinical doses. A secondary mechanism of action is associated with dose dependent reductions of beta APP and amyloid beta. BNC, the lead compound from our butyrylcholinesterase family, is currently in full pre-IND development and we plan an IND submission in third quarter 2006 followed by the potential to initiate Phase I clinical trials thereafter. A recently published article in the Proceedings of the National Academy of Science describes the underlying mechanism, in vitro and cognition results in animal models.

RESULTS OF OPERATIONS

Revenues

The Company had no revenue for the quarters ended June 30, 2006 and 2005. The Company had no revenue for the six months ended June 30, 2006 and $403,000 in revenue for the six months ended June 30, 2005. Revenue in 2005 was derived exclusively from the sale of research assays and fine chemicals at OXIS. The reduction in revenue in 2006 from prior year levels results from the fact that OXIS operations are no longer being consolidated with our results effective March 1, 2005 as discussed in Note 3 to the condensed consolidated financial statements.

Costs of Sales

The Company s costs of sales were entirely related to its subsidiary, OXIS. The percentage of cost of sales for the six months ended June 30, 2005 was 52%.

Research and Development

Research and development expenses were $2,958,000 and $7,351,000 for the quarters ended June 30, 2006 and 2005, respectively. Research and development costs declined by $4,393,000 from the prior year s quarter ended June 30, 2005. This reduction reflects a decline in Phenserine program expenditures of $5,282,000 due to the completion/curtailment of the Phenserine trials in late 2005. This reduction is offset, in part, by increased expenditures of $429,000 in the Posiphen program, $377,000 in the Bisnorcymserine program and $252,000 in non cash employee option charges incurred for related to the adoption of FASB 123(R) as discussed in note 5 in the notes to consolidated financial statements. In 2006 Posiphen is in clinical Phase I studies and Bisnorcymserine was in pre-clinical development towards filing an investigational new drug application (IND). Additional research and development expense reductions include research project costs, insurance and salary expenses.

Research and development expenses were $5,619,000 and $16,673,000 for the six months ended June 30, 2006 and 2005, respectively. Research and development costs declined by $11,054,000 from the six months ended June 30, 2005. This reduction reflects a decline in Phenserine program expenditures of $12,328,000 due to the completion/curtailment of the Phenserine trials in late 2005. This reduction is offset, in part, by increased expenditures of $519,000 in the Posiphen program and $521,000 in the Bisnorcymserine program. Additional research and development expense reductions include travel costs, consultants and salary expenses.

Sales, General and Administrative

Sales, general and administrative expenses were $1,683,000 and $1,213,000 for the quarters ended June 30, 2006 and 2005, respectively. Sales, general and administrative expenses increased $470,000 over the prior year s quarter ended June 30, 2005. This increase is attributed to a $436,000 increase in professional fees primarily resulting from costs related to the merger agreement discussed in note 1, Recent Events and a $125,000 increase in non-cash charges related to employee stock option grants related to the adoption of FASB 123(R) as discussed in note 5 in the

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notes to consolidated financial statements. These increases are offset in part by an $86,000 reduction in investor relations costs.

Sales, general and administrative expenses were $3,553,000 and $2,876,000 for the six months ended June 30, 2006 and 2005, respectively. Sales, general and administrative expenses increased $677,000 from the six months ended June 30, 2005. This increase is attributed to a $400,000 increase in patent acquisition costs, a $440,000 increase in non-cash charges related to stock option grants to consultants and employees and a $201,000 increase in professional fees primarily resulting from costs related to the merger agreement discussed in note 1, Recent Events. These increases are offset in part by a $116,000 reduction in investor relations costs and a $332,000 reduction in OXIS expenses which are no longer consolidated with our results effective March 1, 2005 as discussed in Note 3 to the condensed consolidated financial statements.

Other Income (Expense)

Interest income was $625,000 and $558,000 for the quarters ending June 30, 2006 and 2005, respectively. Interest income was $1,332,000 and $1,130,000 for the six months ended June 30, 2006 and 2005, respectively. Interest income reflects the decline in cash and investment balances offset by a rise in short term interest rates.

Foreign exchange losses for the quarters ended June 30, 2006 and 2005 were $32,000 and $56,000, respectively. Foreign exchange losses of $23,000 and $81,000 were incurred for the six months ended June 30, 2006 and 2005, respectively. The decline in foreign exchange losses reflects more stable exchange rates and a decline in the volume of Euro denominated transactions which reflects the completion of Phenserine trials occurring in Europe in late 2005.

Gain on issuance of subsidiary stock was $78,000 net for the six months ended June 30, 2006. This gain on issuance of subsidiary stock results from common stock equity transactions in OXIS.

Equity in loss of OXIS of $415,000 reflects the Company s proportional share of OXIS losses for the six months ended June 30, 2006 under the equity method of accounting.

Net Loss

The Company experienced net losses of $4,183,000 ($0.08 per share-basic and diluted) and $8,179,000 ($0.15 per share-basic and diluted) for the quarters ended June 30, 2006 and 2005, respectively. The Company experienced net losses of $8,200,000 ($0.15 per share basic and diluted) and $18,612,000 ($0.35 basic and diluted) for the six months ended June 30, 2006 and 2005, respectively. The decline in loss primarily reflects reduced expenditures in 2006 due to the completion/curtailment of the Phenserine development program in late 2005.

LIQUIDITY AND CAPITAL RESOURCES

As of June 30, 2006 we had $50,730,000 in cash, cash equivalents and investments, and $46,624,000 in working capital. We do not have any available lines of credit. Since inception we have financed our operations from private placements of equity securities, the exercise of common stock purchase warrants, license fees, interest income and loans from a shareholder.

Net cash used in operating activities for the six months ended June 30, 2006 was $7,608,000 resulting from a net loss of $8,200,000 and a decline of $2,017,000 in accounts payable. These declines are offset in part by non-cash expenses of $251,000 in depreciation and amortization, $843,000 in compensation related to options and warrants issued for services, $415,000 in equity in loss of OXIS and $274,000 in other current assets.

Net cash provided by investing activities for the six month ended June 30, 2006 resulted from investment sales and maturities in excess of investment purchases.

We plan to finance our needs principally from the following:

- our existing capital resources and interest earned on that capital; and
- future private placement financing or other equity financings.

We believe that we have sufficient capital resources to finance our plan of operation at least through June 30, 2007. However, as this is a forward-looking statement, and there may be changes that could consume available resources significantly

before such time including the impact of our proposed merger with TorreyPines Therapeutics

Inc. (see Recent Events above). Our long term capital requirements and the adequacy of our available funds will depend on many factors, including the effects of the proposed merger with TorreyPines Therapeutics Inc., eventual contract costs of undertaking large later stage clinical trials with any of our compounds under development, the potential cost of acquiring or developing compounds that we may license in, regulatory delays, patent costs for filing, prosecuting, maintaining and defending our patent rights, and defending our current class action securities litigation, among others.

We may be periodically seeking potential equity financing, sub-licensing and other collaborative arrangements that may generate additional capital for us in order to support our research and development activities. We cannot assure you that we will generate sufficient additional capital or revenues, if any, to fund our operations beyond June 30, 2007, that any future equity financings will be successful, or that other potential financings through bank borrowings, debt or equity offerings, or otherwise, will be available on acceptable terms or at all.

As described in Recent Events above, and elsewhere in our SEC filings, we have entered into a definitive merger agreement with TorreyPines Therapeutics Inc. The completion of this merger is subject to shareholder approval and is expected to close during the fourth quarter of 2006. Our business plan, portfolio prioritization and liquidity needs will all be reassessed upon completion of the merger.

Critical Accounting Policies and Estimates

This discussion and analysis of our financial condition and results of operations are based on our financial statements that have been prepared under generally accepted accounting principles (GAAP) of the United States of America. The preparation of financial statements in conformity with accounting principles generally accepted in the United States of America requires our management to make estimates and assumptions that affect the reported amounts of assets and liabilities and the disclosure of contingent assets and liabilities at the date of the financial statements and the reported amounts of revenue and expenses during the reporting period. Actual results could materially differ from those estimates. We have disclosed all significant accounting policies in note B to the financial statements included in our annual report on Form 10-K for the year ended December 31, 2005. Our critical accounting policies are:

Principles of consolidation: Our consolidated financial statements include the accounts of Axonyx Europe, B.V., a wholly owned subsidiary organized in The Netherlands.

Revenue recognition: We defer recognition of revenue from fees received in advance unless they represent the culmination of a separate earnings process. Such deferred fees are recognized as revenue over the term of the arrangement as they are earned, in accordance with the agreement. License fees represent the culmination of a separate earnings process if they are sold separately without obligating us to perform research and development activities or other services. Right to license fees is recognized over the term of the arrangement. Nonrefundable, non-creditable license fees that represent the culmination of a separate earnings process are recognized upon execution of the license agreement. Revenue from the achievement of milestone events stipulated in the agreements will be recognized when the milestone is achieved. Royalties will be recognized as revenue when the amounts earned become fixed and determinable.

Research, development costs: Research and development costs are expensed as incurred.

Stock-based compensation: Commencing January 1, 2006 we adopted Statement of Financial Accounting Standard No. 123R, Share Based Payment (SFAS 123R), which requires all share-based payments, including grants of stock options, to be recognized in the statement of operations as an operating expense, based on fair values on grant date. Prior to adopting SFS 123R, we accounted for stock-based employee compensation under Accounting Principles Board Opinion No. 25, Accounting for Stock Issued to Employees . We have applied the modified prospective method in adopting SFAS 123R. Accordingly, periods prior to adoption have not been restated.

Accounting for Investment in OXIS: Beginning March 1, 2005, the Company accounts for its investment in OXIS under the equity method of accounting, as prescribed by Accounting Principals Board Opinion No. 18 The Equity Method of Accounting for Investments in Common Stock . (See Note 3 to Condensed Consolidated Financial Statements)

Item 3. Quantitative and Qualitative Disclosures about Market Risk

We have foreign currency accounts that are exposed to currency exchange risk. These foreign currency accounts have been utilized to fund the operations of our wholly owned subsidiary, Axonyx Europe, based in The Netherlands. We had a net foreign exchange losses of $23,000 and $81,000 for the six months ended June, 2006 and 2005, respectively. If the foreign currency rates were to fluctuate by 10% from rates at June 30, 2006, the effect on our financial statements would not be material. However, there can be no assurance there will not be a material impact in the future. During 2003, we adopted a policy to limit the purchase of foreign currencies to the amounts necessary to cover firm contractual commitments in foreign currencies for the forward six months. However, as long as we continue to fund our foreign operations and activities, we will be exposed to some currency exchange risks. The majority of our ongoing clinical trials are/were being conducted in Europe.

We consider our investments in money market accounts and time deposits as cash and cash equivalents that have original maturities of three months or less at inception. The carrying values of these investments approximate fair value because of the nature of these instruments and accounts. We classify our investments in auction rate securities as short-term investments. The carrying value of these securities approximates fair value because of the liquidity they are traded as short term investments due to the interest reset feature. Therefore, changes in the market s interest rates do not affect the value of the investments as recorded by us.

We do not enter into or trade derivatives or other financial instruments or conduct any hedging activities.

Item 4. Controls and Procedures

Evaluation of Disclosure Controls and Procedures

The Company s management, under the supervision and with the participation of the Company s Chief Executive Officer and Chief Financial Officer, has reviewed and evaluated the effectiveness of the Company s disclosure controls and procedures as of June 30, 2006. Disclosure controls and procedures are defined in the Securities Exchange Act as controls and other procedures of the Company designed to ensure that information required to be disclosed by the Company in the reports that it files or submits under the Securities Exchange Act is recorded, processed, summarized and reported, within the time periods specified in the SEC s rules and forms and include controls and procedures designed to ensure that information required to be disclosed by the Company in the reports that it files or submits to the SEC is accumulated and communicated to the Company s management, including the CEO and CFO, to allow timely decisions regarding required disclosure. Based on its review and evaluation, the Company s management has concluded that the Company s disclosure controls and procedures were effective as of June 30, 2006.

Changes in Internal Controls

There was no change in our internal control over financial reporting (as defined in Rules 13a-15(f) and 15d-15(f) under the Securities Exchange Act of 1934, as amended) identified in connection with the evaluation of our internal control performed during our last fiscal quarter that has materially affected, or is reasonably likely to materially affect, our internal control over financial reporting.

PART II OTHER INFORMATION

Item 1. Legal Proceedings

Several lawsuits were filed against the Company in February 2005 in the U.S. District Court for the Southern District of New York asserting claims under Sections 10(b) and 20(a) of the Securities Exchange Act of 1934 and Rule 10b-5 thereunder on behalf of a class of purchasers of the Company s common stock during the period from June 26, 2003, through and including February 4, 2005 (the Class Period). Dr. M. Hausman (a director and former CEO of Axonyx), and Dr. G. Bruinsma (Axonyx CEO) were also named as defendants in the lawsuits. On April 10, 2006, following consolidation of the actions into a single class action lawsuit, the lead plaintiffs filed a consolidated amended complaint. The Company s motion to dismiss the consolidated amended complaint was filed on May 26, 2006 and will be submitted to the Court for a decision following the parties filing of their legal briefs.

The class action plaintiffs allege generally that Axonyx s Phase III Phenserine development program was subject to alleged errors of design and execution which resulted in the failure of the first Phase III Phenserine trial to show efficacy. Plaintiffs allege the defendants failure to disclose the alleged defects resulted in the artificial inflation of the price of the Company s shares during the Class Period.

There is also a shareholder derivative suit pending in New York Supreme Court (New York County) against current and former directors and officers of Axonyx. The named defendants are Marvin S. Hausman, Gosse B. Bruinsma, S. Colin Neill, Louis G. Cornacchia, Steven H. Ferris, Gerald J. Vlak, Ralph Snyderman and Michael A. Griffith. Defendants are alleged to have breached their duties to the Company and misused inside information regarding clinical trials of Phenserine. This action has been stayed pending further developments in the federal class action.

The complaints seek unspecified damages. The Company believes the complaints are without merit and intends to defend these lawsuits vigorously. However, the Company cannot make assurances that it will prevail in these actions, and, if the outcome is unfavorable to Axonyx, its reputation, operations and share price could be adversely affected.

Item 6. Exhibits.

Number	Exhibits
2.1	Agreement and Plan of Merger and Reorganization, dated as of June 7, 2006, between Axonyx Inc. and TorreyPines Therapeutics, Inc. (incorporated by reference to the correspondingly-numbered exhibit to the registrant s Current Report on Form 8-K filed June 12, 2006)
2.2	Form of Voting Agreement, dated as of June 7, 2006, between Axonyx Inc. and certain stockholders of TorreyPines Therapeutics, Inc. (incorporated by reference to the correspondingly-numbered exhibit to the registrant s Current Report on Form 8-K filed June 12, 2006)
2.3	Form of Voting Agreement, dated as of June 7, 2006, between TorreyPines Therapeutics, Inc. and certain stockholders of Axonyx Inc. (incorporated by reference to the correspondingly-numbered exhibit to the registrant s Current Report on Form 8-K filed June 12, 2006)
4.1	Rights Agreement Amendment, dated as of June 7, 2006, to the Rights Agreement, dated as of May 13, 2005, between Axonyx Inc. and The Nevada Agency and Trust Company (incorporated by reference to the correspondingly-numbered exhibit to the registrant s Current Report on Form 8-K filed June 12, 2006)
31.1	Certification of Chief Executive Officer pursuant to Exchange Act Rules 13a-14(a) and 15d-14 (a), adopted pursuant to Section 302 of the Sarbanes-Oxley Act of 2002.
31.2	Certification of Chief Financial Officer pursuant to Exchange Act Rules 13a-14(a) and 15d-14 (a), adopted pursuant to Section 302 of the Sarbanes-Oxley Act of 2002.
32.1	Certification of Chief Executive Officer pursuant to 18 U.S.C Section 1350, adopted pursuant to Section 906 of the Sarbanes-Oxley Act of 2002.
32.2	Certification of Chief Financial Officer pursuant to 18 U.S.C Section 1350, adopted pursuant to Section 906 of the Sarbanes-Oxley Act of 2002.

SIGNATURES

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

Dated August 8, 2006.

AXONYX INC.

By: /s/ Gosse B. Bruinsma, M.D.

Gosse B. Bruinsma, M.D.
President and Chief Executive Officer

By: /s/ S. Colin Neill

S. Colin Neill
Chief Financial Officer,
Secretary and Treasurer
(Principal Financial and Accounting Officer)